We can now add that the proved existence of the antinucleons has very strongly corroborated this possibility, although we also know that the symmetry between electric charges breaks down for weak interactions. As far as astronomical means are concerned, a verification seems impossible in principle, because they depend on electromagnetic phenomena, which are invariant under charge conjugation. It is, however, interesting that the recent important discoveries about beta decay and the neutrino now give a method for looking for antimatter which, while still impossible in practice, is sound in principle, being based on weak interactions which are *not* invariant under charge conjugation. This method, if it could be executed, would solve unambiguously the question of the existence of antiworlds. If we observe a star and from its astronomical characteristics can decide that most of its energy comes from a known cycle, as for example the carbon cycle, which is domi-

nated by beta decays, we can see whether the antineutrinos coming from it are or are not of the same kind as the antineutrinos coming from a pile or from our sun by performing an inverse beta-decay experiment. If it should turn out that they are neutrinos —different from those coming from the sun—then the star is of antimatter (*14, 15*).

References and Notes

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14. As in many investigations in high-energy physics in recent times, this experiment is the result of a large cooperative effort. The credit for the success is shared by many individuals and even by a machine, which was obviously necessary to produce particles above the threshold for nucleon pair production. Since it is impossible to mention all the numerous contributors, I shall limit myself to a few. Oreste Piccioni helped materially in the early planning of the experiment, especially by suggesting the use of magnetic quadrupole lenses. Edward J. Lofgren most ably directed the operation of the Bevatron. Herbert M. Steiner supplied invaluable help during the whole experiment. Tom J. Ypsilantis, our colleague and coauthor, also worked with us all the time. Above all, however, our coauthor and comrade of 20 years of work, Clyde Wiegand, was indispensable and deserves a major part of the credit for the success of our investigation.
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General Theory of Mortality and Aging

A stochastic model relates observations on aging, physiologic decline, mortality, and radiation.

Bernard L. Strehler and Albert S. Mildvan

Although statistics on human mortality furnish one of the most extensive and reliable collections of biological data, general theories to account for the quantitative relationships between age and death rate have not been completely satisfactory. The essential observations which must be taken into account in any general theory of mortality are as follows:

1) The death rate at any age (of

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most adult human populations and of many populations of animals) may be expressed as the sum of an age-dependent term (Gompertz, *1*) and an age-independent term (Makeham, *2*)—that is, $R = A + R_0 e^{\alpha t}$).

2) In certain environments the Makeham term (A) predominates (for example, wild birds, *3*), while in most human populations (between ages 35 to 85) and in certain animal populations, the Gompertz term predominates (see Fig. 1). The Gompertzian period is followed by a gradual reduction in

the rate of increase of the mortality rate (*4*).

3) The rate of decrease of most physiological functions of human beings is between 0.5 and 1.3 percent per year after age 30, and is fit as well by a straight line as by any other simple mathematical function (*5, 6*) (see Fig. 2).

4) Death rates due to certain important specific causes (for example, cancer, tuberculosis, and heart disease) increase exponentially with age similarly to the total death rate (*7, 8*).

5) Continuous exposure of experimental animals to high-energy radiation tends to increase the Gompertz slope (α) by an amount proportional to dose rate, whereas exposure to a single dose of ionizing radiation does not appreciably affect α, but does increase $\ln R_0$ proportionally to dose (*9–11*).

Among the recent attempts to postulate mechanisms underlying the Gompertz function or the other generalities given above, or both, are the theories of Jones (*8*), Failla (*12*), Sacher (*13*), Yockey (*14*), and others (*15, 16*). Each theory has certain attractive features but either fails to account for all of the above observations, possesses internal inconsistencies, or makes incorrect predictions. A detailed critique of these and other theories is in press (*17*).

Statement of Theory

The present theory (*18*) can be summarized as follows:

Postulate 1. An organism consists of a number of subsystems, each of which has a certain maximum ability to restore initial conditions after a challenge (that is, a change in condition due to internal or external energy fluctuations). Death occurs when the rate at which an organism does work to restore the original state is less than that demanded to overcome the effects of a given challenge.

Postulate 2. The magnitudes of challenges (or, more appropriately, the responses required to overcome these challenges) are distributed energetically like a Maxwell-Boltzmann distribution of energies among molecules (*19*). When these postulates are combined with the Gompertz equation, a number of consequences follow (*20*).

In this article, these predictions and consequences of the theory are enumerated and tested against observation wherever possible. The theory appears to relate simply and satisfactorily the Gompertz function and physiological capacity measurements versus age, as well as a number of other demographic observations. It makes several predictions which subsequent analyses of mortality and physiological data have substantiated. We have been unsuccessful in showing any major inconsistencies between this theory and observation.

The following definitions are used:

1) *Aging.* The inherent process(es) whereby organisms exhibit a gradual change in their physical, chemical, or physiological properties after reproductive maturity. These changes result in a gradual increase in the probability of death in the organisms' normal environment.

2) *Gompertz equation.*

$$R = R_0 e^{\alpha t} \qquad (1)$$

This is a kinetic equation describing the probability of death of many populations as a function of age. R is the rate at any time t, R_0 is the hypothetical rate at zero time, and α is the Gompertz coefficient, which describes the rate of increase of the exponential term.

Makeham's modification of Gompertz' equation,

$$R = A + R_0 e^{\alpha t}, \qquad (2)$$

applies to aging populations which are also characterized by high age-independent death rates. In Eq. (2), A is a measure of the age-independent death rate.

3) *Vitality.* Vitality is defined as the capacity of an individual organism to stay alive, as measured by an appropriately weighted average of the maximum rate of work output (power output) less the basal power output of all of the functional modalities contributing to survival in the normal environment. This weighted mean of an individual's vitality at a given age is designated as V^t. The hypothetical value of V^t at $t = 0$ is V_0^t. Similarly, V and V_0 represent the averages of the above quantities for a population of uniform age. This concept of vitality is similar to one set forth by Medawar (*21*).

4) *Maxwell-Boltzmann distribution* (*19*).

$$n/n_t = k(E/RT)^{\frac{1}{2}} e^{-E/RT} \qquad (3)$$

when $E \geq RT$.

For $E \gg RT$, the equation can be simplified to

$$n/n_t \doteq K e^{-E/RT}, \qquad (4)$$

Where E is the energy of a state of a molecular system, T is the absolute temperature, R is the gas constant, n is the number of molecules with energy equal to or greater than E, and n_t is the total number of molecules.

Equations 3 and 4 describe the distribution of challenges to molecular bonds. Analogous equations derived from the postulates of the theory described in this article appear below (Eqs. 7 and 8), in which the terms E and RT are supplanted by the analogous expressions V and ϵD, respectively. D is a measure of the relative deleteriousness of an environment, whereas ϵ is a constant chosen so that the quotient $V/\epsilon D$ is unitless. In addition, ϵD is a measure of the average demand for energy expenditure over and above the basal level.
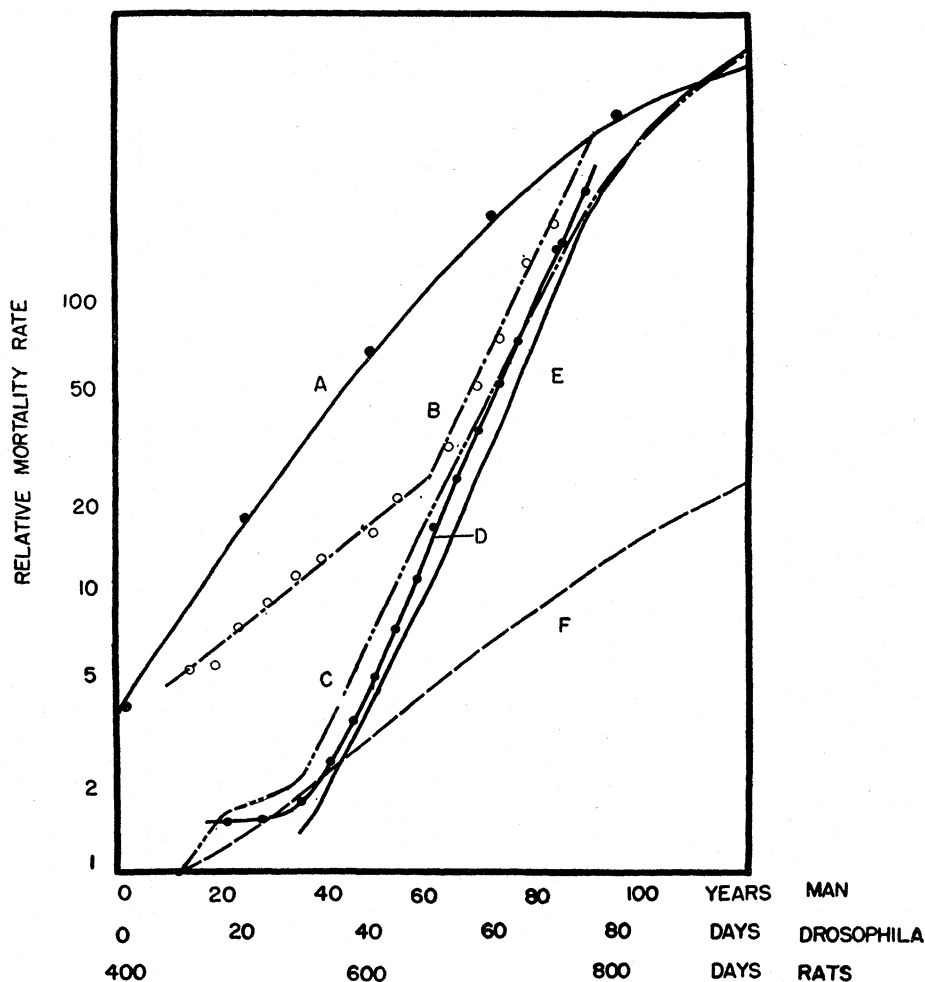

Fig. 1. Gompertz plots of mortality rates for various species. *A*, male rats (*26*); *B*, human male (Egypt 1947) (*22*); *C*, human male (U.S., white—North Central Division, 1949–51) (*4*); *D*, human male (Sweden, 1953) (*22*); *E*, human female (U.S., white—North Central Division, 1949–51) (*4*); *F*, *Drosophila melanogaster* (*29*).

5) *Attrition coefficient*. The attrition coefficient B is the fractional loss each year of original vitality V_0.

$$B = b + f(D), \qquad (5)$$

where b is the attrition coefficient due to normal aging and $f(D)$ is the attrition coefficient due to environmental factors (see also Medawar, *21*).

Our theory may be stated mathematically as follows:

Postulate 1.

$$R = CX, \qquad (6)$$

where R is the mortality rate, C is the total number of challenges per unit time, and X is the fraction of challenges equal to or greater than V.

Postulate 2.

$$X = k'(V/\epsilon D)^{\frac{1}{2}}e^{-V/\epsilon D} \qquad (7)$$

or

$$X \doteq K'e^{-V/\epsilon D} \qquad (8)$$

Substituting Eq. 7 in Eq. 6 and using Eq. 1 (that is, a Gompertz function), we obtain

$$R = R_0 e^{\alpha t} = Ck'(V/\epsilon D)^{\frac{1}{2}}e^{-V/\epsilon D}$$
$$= k(V/\epsilon D)^{\frac{1}{2}}e^{-V/\epsilon D}. \qquad (9)$$

Similarly, substituting Eq. 8 in Eq. 6 and using Eq. 1, we obtain

$$R = R_0 e^{\alpha t} \doteq Ke^{-V/\epsilon D}. \qquad (10)$$

Equations 9 and 10 are the basic equations of our theory. All subsequent relationships are derived from them.

Predictions and Tests of the Theory

Linear loss of vitality with age.
1) Derivation: Taking logarithms and solving Eq. 10 for V, we obtain

$$V = \epsilon D \ln (K/R_0) - \epsilon D\alpha t \qquad (11)$$

$$= \epsilon D \ln (K/R_0)\left[1 - \frac{\alpha t}{\ln (K/R_0)}\right]. \qquad (12)$$

When $t = 0$,

$$V = V_0 = \epsilon D \ln (K/R_0). \qquad (13)$$

Since α, K, and R_0 are constant, we can define the term containing them as B (the attrition coefficient); that is,

$$B \equiv \frac{\alpha}{\ln (K/R_0)}, \qquad (14)$$

and thus

$$V = V_0(1 - Bt). \qquad (15)$$

2) Prediction: From Eq. 15 it follows that vitality should be lost at a constant rate.

3) Test: This prediction is in agreement with observation 3 in the introduction (see Fig. 2 also).

Inverse relationship between R_0 and α.

1) Derivation: Rearranging Eq. 14, we obtain

$$\ln R_0 - \ln K = -(\alpha/B) \qquad (16)$$

2) Prediction: Contrary to the intuitive notions relating the values of R_0 and α, the theory predicts that a high initial mortality rate (presumably characteristic of an inhospitable environment) should be associated with a low rate of increase (α) of mortality rate. In its most simple form the theory predicts that a straight line will be obtained if $\ln R_0$ is plotted against α, whose slope is $-1/B$ and whose intercept is $\ln K$ (if B is a constant).

3) Test: The extent to which natural human populations approximate this relationship is illustrated in Figs. 3 and 4, in which the individual points represent the values of R_0 and α derived for various countries from the United Nations *Demographic Yearbook* for 1955 (*22, 23*).

Figures 3 and 4 clearly indicate that both B (which measures the fractional loss of vitality per year) and K (which, according to this formulation, measures the total number of challenges per unit time regardless of their magnitudes) are essentially constant. The constancy of K is a necessary consequence of postulate 1 (and Eqs. 6 to 10). The present observation is thus consistent with this assumption. More surprising is the fact that B appears to be nearly constant
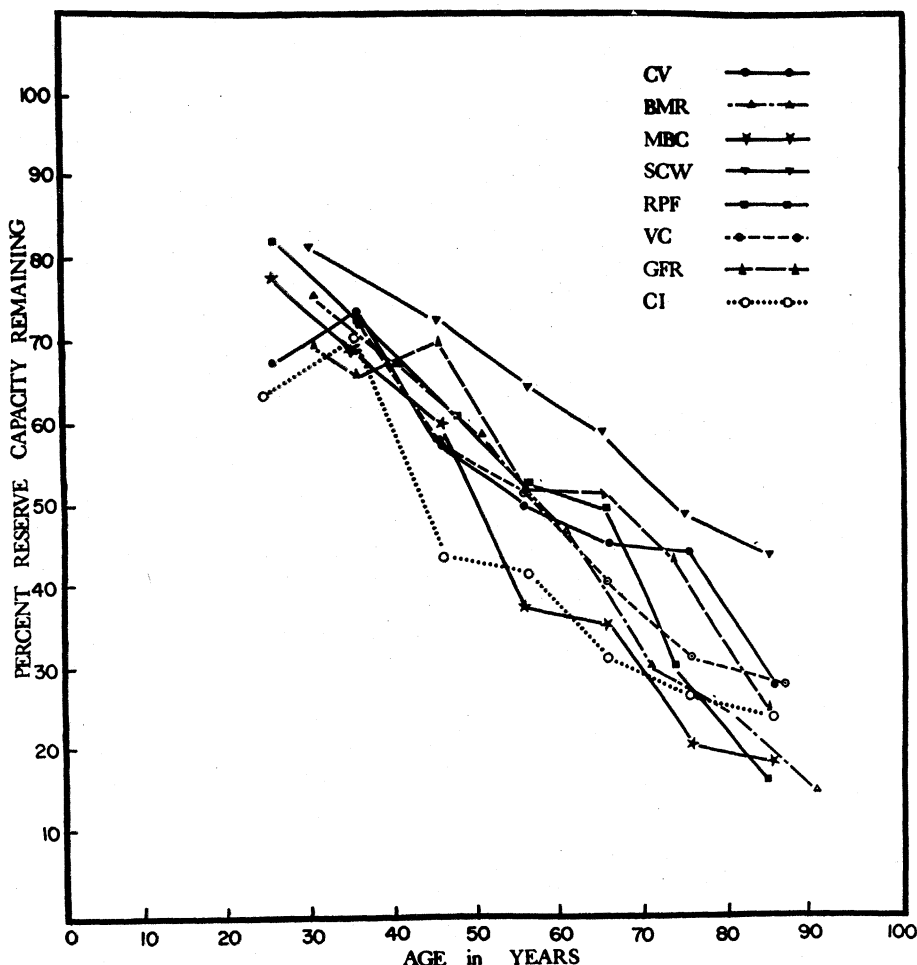

Fig. 2. Percent of reserve capacity of a number of physiologic functions remaining at various ages in human males (calculated from the data of Shock *et al.*, *5, 6, 30*). The decade average values for each physiologic function were divided by the calculated initial reserve value for that function. This initial reserve for each physiologic function was measured by the difference between the value of the function extrapolated to age 0 and a lower limiting value of the function. This lower limiting value, in turn, was assumed to be equal to the lowest measured individual value. Independent techniques of estimating this lower limit gave comparable results. (See *17* for a more detailed treatment.) *CV*, nerve conduction velocity; *BMR*, basal metabolic rate; *MBC*, maximal breathing capacity; *SCW*, standard cell water; *RPF*, standard renal plasma flow (Diodrast); *VC*, vital capacity; *GFR*, standard glomerular filtration rate (inulin); *CI*, cardiac index.

16

regardless of the environment. B was defined in Eq. 5 as being made up of a constant term, b, and an environmental term, $f(D)$. It thus appears that B is dominated by b, or in other words that the rate of loss of vitality during the aging process is largely independent of the environment.

Prediction of the value of the attrition coefficient (B).

Method 1. From the relation of R_0 and α for various countries. If one uses the data of Fig. 3 and Eq. 16, the value of B derived from the over-all slope is about 1.46 percent of V_0 per year. The value of B for the eight highest α countries is about 1.27 percent of V_0 per year, which suggests the possibility of slight curvature. The corresponding range of K values is 0.05 to 0.15 per year. Since real mortality rates approach 0.6 per year in extreme old age, it is clear that the former value is too low and that the real value of K is greater than 0.6.

Method 2. From the relation of R_0 and α, assigning a value of K $(K = 1)$ and using Eq. 18. Values for B averaging 0.0097 and ranging from 0.0086 to 0.0111 were obtained. The values for individual countries are listed in Table 1.

Method 3. From the terminal curvature of mortality rate curves. From Eq. 9, which more closely gives the rate to be expected when the reserve capacity (V) approaches the average demand (ϵD), and using Eq. 15, we obtain

$$R = k \ [(V_0/\epsilon D) \ (1 - Bt)^{\frac{1}{2}} e^{-V_0/\epsilon D} \times \\ e^{\ (V_0/\epsilon D)Bt}, \quad (17)$$

and when $t = 0$

$$R_0 = k \ (V_0/\epsilon D) \ ^{\frac{1}{2}} e^{-V_0/\epsilon D}, \quad (18)$$

Substituting Eq. 15 in Eq. 10, we obtain

$$R = K e^{-V_0/\epsilon D} \ e^{\ (V_0/\epsilon D)Bt} \\ = R_0 e^{\alpha t}. \quad (19)$$

Thus

$$\alpha = (V_0/\epsilon D)B$$

and

$$\alpha/B = V_0/\epsilon D. \quad (20)$$

Substituting α/B for $V_0/\epsilon D$ in Eq. 18, we obtain

$$R/R_0 = (1 - Bt)^{\frac{1}{2}} \ e^{\ \alpha t}. \quad (21)$$

Squaring and solving for B, we obtain

$$B = \frac{1 - (R/R_0 e^{\alpha t})^2}{t}. \quad (22)$$
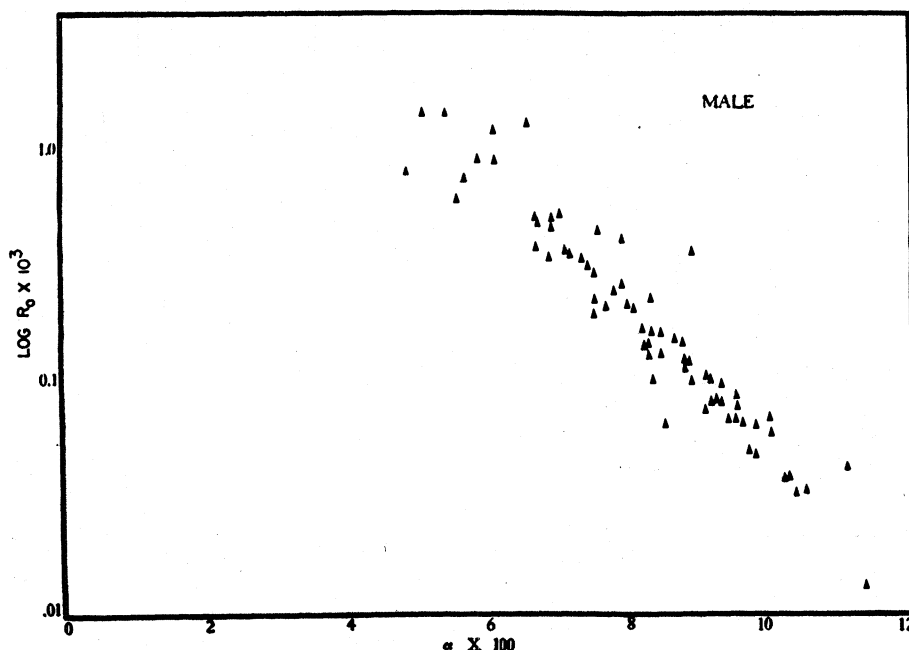
Test: The values of B calculated

Fig. 3. Gompertz slope (α) versus logarithm of extrapolated hypothetical mortality rate at age 0 (ln R_0) for males of all countries for which adequate data are available (*22*). Each country's mortality rate was plotted individually on semi-log paper. A straight line was drawn between points from 35 to 85 (or 50 to 70, if large departures from linearity occurred), and the slopes and intercepts were measured or calculated. Only a few countries, whose Gompertz plots exhibited great irregularities, were excluded.

from methods 1 and 2 range from 0.92 to 1.37 percent per year, which is in reasonable agreement with the observed rate of loss of human physiologic functional capacities (0.5 to 1.3 percent per year). (See observation 3.) (For a detailed analysis, see Mildvan and Strehler, *17*.)

We have also calculated B values using Eq. 22 and ages over 100 years (*4*). Although the values calculated range from 0.003 to 0.009 per year,
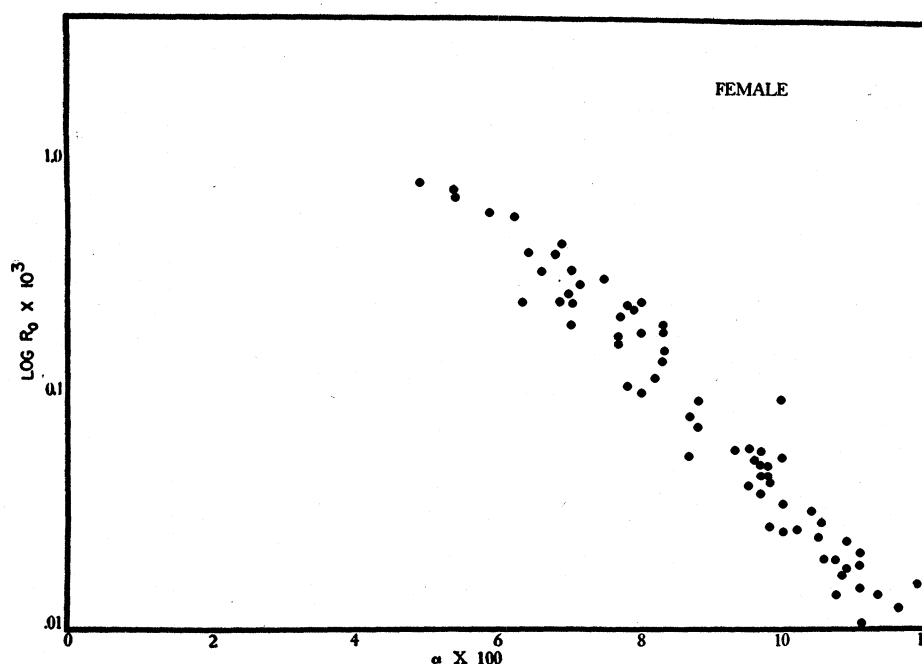

Fig. 4. Gompertz slope (α) versus logarithm of extrapolated hypothetical mortality rate at age 0 (ln R_0) for females of all countries for which adequate data are available (*22*). See legend to Fig. 3 for technique of calculation.

these results are, in all likelihood, for·tuitous, since the shape and magnitude of curvature depart from the predicted shape and magnitude by considerable amounts.

Therefore, Eq. 22 cannot be applied to human populations because variability among individuals and subpopulations, combined with inherent inaccuracies in the calculated and reported age specific rates, at advanced ages, will produce unpredictable departures from the ideal. It is hoped that an independent test can be developed on some experimental and more homogeneous population of organisms.

Prediction of quantitative relationship between average demand and reserve vitality of young individuals. In an "ideal" environment an organism would need to expend energy· at only the basal rate required for maintenance in the absence of challenges. In real environments, there are demands over and above this basal level which we here define as ϵD, in which D measures the relative deleteriousness of the

milieu. In the present analogy V_0 represents the amount by which the maximum rate of energy expenditure of a young person exceeds the basal rate of energy dissipation (appropriately weighted). Particularly, it should be kept in mind that V and V_0 (vitality) include only the reserve capacity of an organism to do work in overcoming challenges to its existence. It does not include the work it must do to maintain itself in the absence of challenge. The latter, appropriately weighted, is the basal work rate.

$V_0/\epsilon D$ is then the ratio of maximum vitality (V_0) to the average demand (ϵD) in excess of the basal work rate, a ratio which the theory has enabled us to estimate, even though we cannot make estimates of the value of V_0 and ϵD independently. Using Eq. 20, $\alpha/B = V_0/\epsilon D$, we can calculate that this ratio for various national environments ranges from about 7.0 to about 11.0 in the worst and best environments, respectively (see Table 1).

Test: For the three functions which

we were able to test by analysis of values in the published literature, the ratio $V_0/\epsilon D$ ranges from 7.9 to 32 (see Table 2). The cardiac index and heat output calculations are in good agreement with the values of $V_0/\epsilon D$ calculated from mortality figures; the ventilation rate figures are not.

Relative deleteriousness (D values) of various national environments. From Eq. 20 one can also estimate the relative D values of various countries. The values thus obtained are illustrated in Table 1, in which the most favorable present national environment is assigned a value of 100. Note that the countries with the most extreme values differ from each other only by about a factor of 1.5 in the average stress magnitudes (ϵD). Unfortunately we have not been able to estimate relative deleteriousness by independent objective means.

Prediction of relationship between Gompertz slope, R_0, and radiation exposure. We make the assumption that the total irreparable damage (d) done by radiation exposure is proportional to the total radiation dose (r_t). That is,

$$d = \beta r_t = \beta \rho t, \qquad (23)$$

where ρ is the dose rate. The corollary assumption is made that this damage is reflected in a corresponding loss of vitality. Thus,

$$V = V_0(1 - Bt - \beta r_t) \qquad (24)$$

or

$$V = V_0[1 - (B + \beta\rho)t] \qquad (25)$$

for continuous exposure from $t = 0$. Substituting Eq. 24 and Eq. 25 into Eq. 10 of our theory, we obtain

$$R = Ke^{-(V_0/\epsilon D)(1 - Bt - \beta r_t)} \qquad (26)$$

and

$$R = Ke^{-(V_0/\epsilon D)(1 - Br_t)}e^{(V_0/\epsilon D)Bt} \qquad (27)$$

for a single dose and

$$R = Ke^{-V_0/\epsilon D}e^{(V_0/\epsilon D)(B + \beta\rho)t} \qquad (28)$$

for continuous exposure from $t = 0$.

From these relationships (Eqs. 27 and 28),

$$R_0 = Ke^{-(V_0/\epsilon D)(1 - Br_t)} \qquad (29)$$

for a single dose and

$$\alpha = (V/\epsilon D)(B + \beta\rho)$$

for continuous exposure from $t = 0$.

In other words, the theory (combined with the subsidiary assumptions above) predicts that the extrapolated ln R_0 for a single dose will increase proportion-

Table 1. R_0, α, and derived quantities for human males of various countries.

Country	Year	$R_0 \times 10^3$	α	B^* ($K=1$)	$\dfrac{\alpha}{B}$ ($K=1$)	Relative D value; ($D_{Norway}=100$)
Alaska	1950	0.247	0.0774	0.00934	8.3	130
Algeria (European)	1948	.335	.0735	.00935	7.9	136
Algeria (Moslem)	1948	.820	.0612	.00864	7.1	152
Argentina	1947	.364	.0724	.00908	8.0	134
Australia	1953	.057	.0948	.00970	9.8	110
Austria	1953	.0450	.1010	.01016	9.9	109
Belgium	1953	.0620	.0943	.00974	9.7	111
Brazil	1950	.640	.0671	.00914	7.4	146
Canada	1953	.0370	.0986	.00970	10.1	106
Costa Rica	1950	.0780	.0935	.00990	9.4	114
Chile	1952	.205	.0818	.00975	8.4	128
Denmark	1954	.0340	.102	.0110	9.2	116
Egypt	1947	.150	.0865	.00977	8.8	122
El Salvador	1950	.172	.0788	.00912	8.6	125
Finland	1953	.133	.0866	.00971	8.9	120
France	1953	.0910	.0917	.00986	9.3	115
Germany	1937	.0900	.0896	.00963	9.3	115
Ireland	1953	.0780	.0953	.0101	9.4	114
Italy	1951	.030	.103	.0093	10.7	100
Japan	1953	.062	.0963	.00994	9.7	111
Mexico	1950	.310	.0731	.00908	8.1	133
Netherlands	1953	.038	.0980	.00965	10.2	106
New Zealand	1954	.0318	.1030	.00997	10.3	104
North Ireland	1954	.0560	.0950	.00971	9.8	110
Norway	1953	.0245	.104	.00970	10.7	100
Portugal	1954	.0540	.100	.0102	9.8	110
Sweden	1953	.037	.0987	.00970	10.2	106
Switzerland	1953	.069	.0929	.0097	9.6	112
Trinidad and Tobago	1953	.022	.119	.0111	10.7	100
United States	1953	.200	.0783	.0092	8.5	126
Venezuela	1950	.376	.0684	.00863	7.9	135
West Germany	1953	.062	.0943	.00973	9.7	111

* Statistical constants related to B when $K = 1$: number, 32; mean, 0.00966; standard deviation, 0.000469; standard deviation/mean, 0.0485; standard error of mean, 0.0000828.

Table 2. Calculation of reserve ratio $V_0 / \epsilon D$ for several physiological functions.

Function	Units	Basal value	Av. value	Maximal value (av.)	$\dfrac{\text{Max. basal}}{\text{av. basal}} = \dfrac{V_0}{\epsilon D}$	References
Cardiac index	lit. /M² min	4.3	4.6–5.0*	9.8	7.9–18	(31, 32)
Ventilation rate	lit./min	4.9	9.15†	68.5–140.5	15–32	(30, 33)
Heat output	kcal/hr	65	104–146‡	600	6.6–13.7	(31)

* Average cardiac index calculated on the basis of 2500 to 3500 kcal per day according to the observed functional relation between cardiac index and heat output (33). † Average ventilation rate calculated on the basis of 2900 kcal/day (33) and of an assumed respiratory quotient of 0.9 ‡ Average heat output calculated on the basis of 2500 to 3500 kcal/day.

ally to the dose and that the Gompertz slope (α) will be increased proportionally to the dose rate for continuous exposure.

Test: This is in agreement with observation 5 (see Fig. 5).

Discussion

The present theory of mortality is based on the kinetics of death of populations and entails the tacit assumption of approximate equivalence among the various individual members of a population. However, it is clear that considerable variability does exist among individuals with respect to their physiological and physical properties and maximum (5, 6) performance capabilities, and we shall therefore here consider the potential effects of individual variability on the mortality behavior of populations in terms of the present theory.

Variability among individuals might be expected to arise from difference either in their genetic constitution or in their environment. Each of these factors contributes to the several terms in the equations.

Thus, V_0' (for an individual) is the resultant of the interaction of genetic make-up and the environmental factors contributing to maturation. Similarly, B, which represents the fractional loss of vitality per year, is made up of an intrinsic (presumably genetically determined) term and an environmental term.

Variability in V_0' and B' within a population is capable of producing curvature in the Gompertz plot. When ϵD is assumed to be a constant, it is clear (from Eq. 20) that subpopulations will have identical α's only when the product $V_0 B$ is a constant. Thus, a distribution of values of either V_0' or B', when $V_0' B'$ is not constant, will result in a curve with a gradually increasing slope (α) (since the sum of several increasing exponentials with different α's al-

ways yields a curve whose slope increases with time).

On the other hand, there may be an opposing effect of variability in either B' or V_0' (assuming either that BV_0 is constant or is not constant). This tendency of the average value of α in a heterogeneous population to decrease at great ages is a consequence of the fact that those individuals are most likely to die who have the smallest V at any age. The death of such low-vitality individuals tends to leave the average vitality of the remainder at a higher value than it would have if the weaker individuals had not been removed.

Since the death rate of a total population is determined by the average V, the Gompertz slope will approach the value of the lower mortality rate subpopulation. This may yield a gradual decrease in α. These two opposing effects are illustrated in Fig. 6.

In addition, it can be shown from Eq. 17 that one would expect a gradually decreasing value of α with advancing age.

Since real populations exhibit remarkably linear Gompertz kinetics up to great ages, we must assume either that variability is not great compared to ϵD or that the subpopulations are made up of such mixtures of the above three possibilities that opposing effects tend to cancel each other out. Thus, individual variability is not inconsistent with the present theory. The extent to which individual variability contributes to the shape of mortality curves cannot now be evaluated.

Simms (16) and Jones (8) have pointed out that a number of individual causes of death also exhibit Gompertz kinetics. Jones has emphasized the approximate similarity of slopes of various cause-specific mortality curves. We have plotted and examined the cause-specific death rates for the United States and have found some, although not general, confirmation of this observation. Of the four major causes of death (heart disease, cerebrovascular accidents, neoplasms, and accidents), the second and third have almost identical slopes and intercepts, whereas the principal cause of death (heart disease) has a lower intercept but greater slope. The sum of these curves is not a good straight line.

Several conclusions can be drawn from the above. First, although the death rates due to specific disease are not constant fractions, throughout life, of the total death rate, their slopes and rates are so adjusted that their sums approximate straight lines. This is apparently true because of the inverse re-
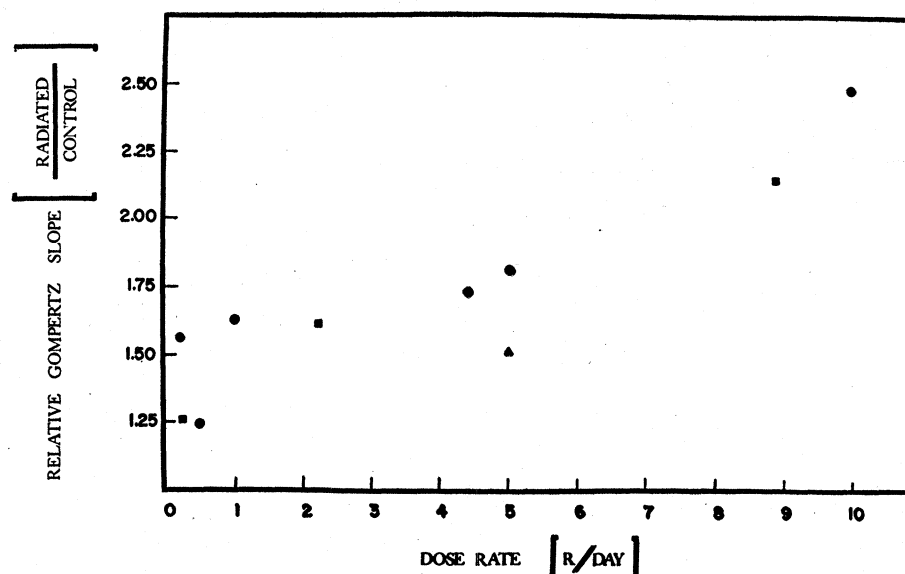

Fig. 5. Effect on Gompertz slope of chronic irradiation exposure of rats, after original data from: (circle) Furth et al. (10); (square) Dowdy et al. (34); (triangle) Brues and Sacher (35). For a more detailed discussion, see Berlin and DiMaggio (28).

lationship between R_0 and α of cause-specific rates; this relationship tends to compensate for the curvature otherwise to be expected.

It appears that the V_0's and B's for important cause-specific death rates may have been subjected to evolutionary selection in such a manner that total deaths due to the three major causes are similar near the close of the normal reproductive lifetime. As Williams (24) has pointed out, it is to be expected that there would be a strong selection pressure against any genetically dependent single cause of death.

Finally, it appears possible that at least some of the increased slope of the mortality rate curve in "good" environments is related to the predominant contribution of heart disease in these countries.

The fact that the present theory predicts the observed relationships between dosage, α, and R_0 is a valid test of the theory. It is not, however, a test of the theory that radiation accelerates aging. Radiation, like any other noxious agent that produces permanent damage to organisms or cells, would be expected to contribute to mortality as observed. But radiation damage may affect systems adversely that normally might be relatively unaffected by time, and vice versa (25–28). A test of the theory of the equivalence of time and radiation awaits a detailed description of the intimate mechanisms and details of the changes produced by each process.

It is of interest that the theory predicts that the maximum lifetime attainable in a homogeneous population will be approximately $1/B = 103$ years for the calculated $B = 0.0097$. Compare this figure with the maximum ages reliably reported for human beings—110 to 115 years.

Summary and Conclusions

1) A theory of the kinetics of death is presented which is based upon the experimentally determined Gompertz function and the two following postulates: (i) The distribution of stress magnitudes is a Maxwell-Boltzmann distribution; (ii) an organism dies when stress magnitude exceeds the organism's maximum ability to compensate therefor.

2) The theory predicts a zero-order loss of function versus age. This is borne out in human males by independent observation.

3) The theory permits several independent calculations of the value of B, the percent of loss per year of physiologic function. The calculated values range from 0.9 to 1.4 percent per year and agree closely with the observed rates in human males.

4) The theory predicts an inverse linear relationship between Gompertz slope and $\ln R_0$ (intercept), which is closely confirmed by observation.

5) The theory predicts that the mean ratio of maximum reserve capacity to average demand lies between 7 and 11. Independent physiologic measurement data are in reasonable agreement with these values.
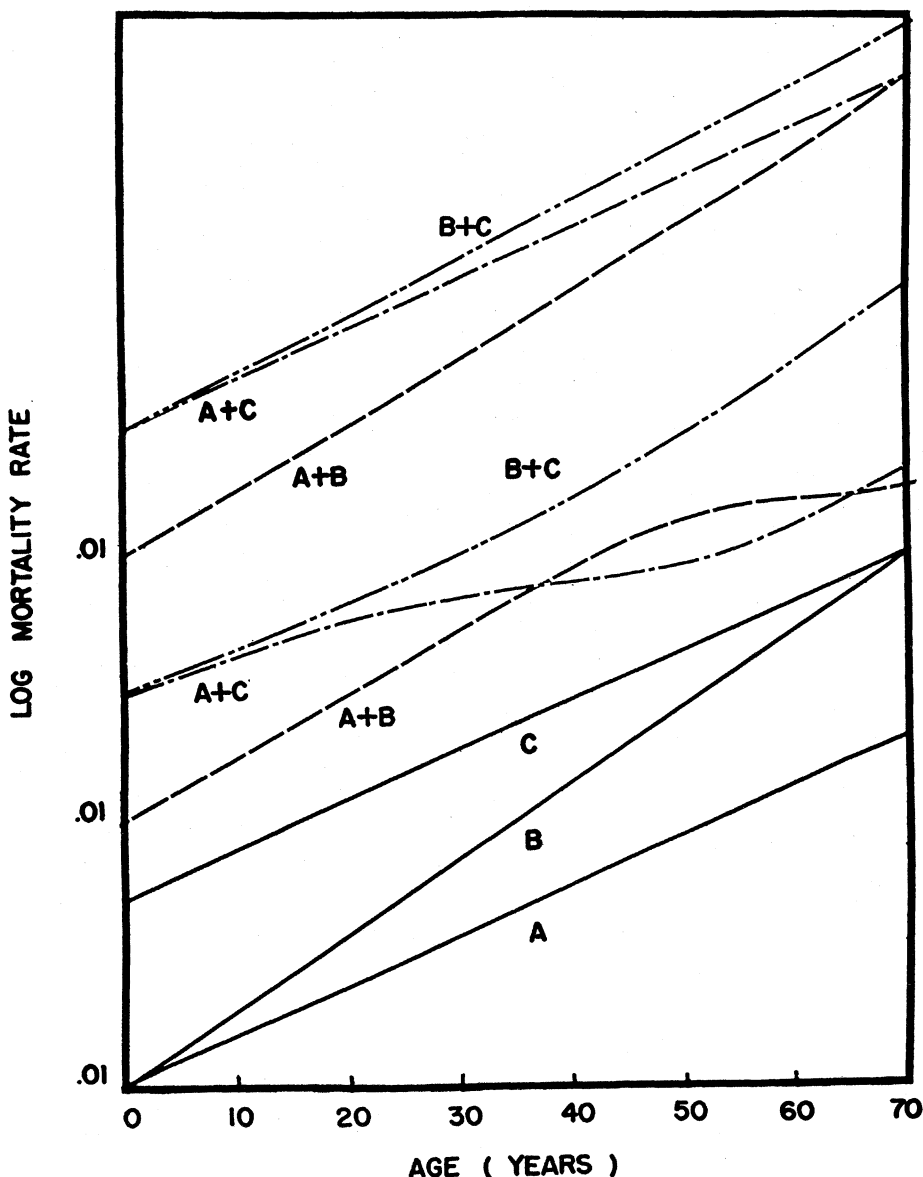

Fig. 6. Summation of mortality rates of various pairs of Gompertzian subpopulations. Three homogenous Gompertzian subpopulations are depicted as A, B, and C at the bottom of the figure. The mortality rates (deaths per year per individuals alive at the beginning of the year) varied as follows between ages 0 and 70: curve A, 0.01 to 0.2; curve B, 0.01 to 1.0; curve C, 0.05 to 1.0. The behavior of the sum of various combinations of A, B, and C, as calculated by two different methods, is also shown: At the top of the figure the average rates are calculated without taking into account the selective loss of individuals from the subpopulation having the higher mortality rate. Note that the slope of $A + C$ is identical to the slope of A or C, whereas the $A + B$ and $B + C$ curves show very slight concavity upwards. By contrast, the more real situation depicted in the curves in the middle of the figure shows the effect of the selective elimination of individuals (by death) from one of the subpopulations on their combined mortality curve. Note particularly that the curve for $A + B$ is highly reminiscent of the behavior of many human and animal populations (particularly at great ages). On the other hand, $B + C$ yields a curve which displays considerable concavity upwards, whereas $A + C$ gives a curve with an inflection part way up and with approximately equal slopes before and after.

20

6) The theory predicts the observed effects of prolonged or "instantaneous" exposure of experimental animals to ionizing radiation.

7) The relative deleteriousness (D) of various national environments can be calculated. They have been found to differ by approximately 50 percent. We have been unable to make an independent test of these relative values.

8) Despite the fact that it is derived for a homogeneous population the theory is shown to be not inconsistent with individual variability within a population.

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20. The present theory is expressed in terms of energy fluctuations arising internally or externally. This dimensional system is chosen because: (i) death undoubtedly cannot occur without the occurrence of a change in structure or physiological state, which change is the result of energy expenditure; (ii) death will occur if the capacity of the system to restore conditions necessary for life is exceeded; (iii) this restoration of original conditions requires the expenditure of energy of a certain kind at a certain minimum rate. Thus, an organism lives or dies according to whether its maximum power output in the challenged modality is sufficient to overcome the disruptive influence of the challenge. It is also clear that any parameter exhibiting an inverse logarithmic relationship between magnitude and frequency (coupled with a linearly decreasing resistance thereto) would be sufficient to generate the observed mortality behavior. Such a functional relationship would result from the square of any variable that exhibits a Gaussian distribution—for example, molecular velocity. Thus, the agreement between prediction and observation does not "prove" the validity of the energy analogy. However, attempts to postulate and measure underlying variables other than energy or its equivalent have thus far not been successful. For the above reasons we have emphasized the energy analogy as being the most appropriate one at present.
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Roy Chapman Andrews, Explorer

Some sixty years ago Roy Chapman Andrews, the son of a rural druggist in southern Wisconsin, chanced upon *A Handbook of North American Birds* by Frank M. Chapman of the American Museum of Natural History. The book, and the author of that book, were to influence most profoundly the life of Roy Andrews, just as decades later the writings of Andrews were to cast their spell over the lives of many boys entering into manhood. Young Andrews had a burning desire to meet Chapman, and eventually he did meet him, in the summer of 1906.

Andrews had at that time just completed his undergraduate career at Beloit College, and he faced the world with restless energy. To him, a young man fond of the outdoors and a veteran of numerous camping trips in his native state, the American Museum of Natural History and Frank Chapman were the two poles of a magnet that attracted him with irresistable force to New York. It is an oft-repeated tale, how he approached the director of the Museum and asked for employment, how he was told that there were no staff openings available, and how he took a menial job as general attendant and handyman in the department of preparation, where he mixed clay and scrubbed floors.

His enthusiasm for his work and for the museum were immediately apparent, and he very quickly advanced within the institution. In 1908 he went to British Columbia to make field studies of whales, an endeavor that set the pattern of his life for the next 8 years, which he spent in active and diverse field work on Pacific whales. As a result of these studies he published two monographs and several short papers on the Cetacea, and these were in essence the totality of his research publications.

It became apparent to him early in his career that research was not his major interest; rather, he developed an overwhelming desire to carry on field work and exploration. This was in part the result of his youthful camping days