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NEW MATHEMATICAL LAWS OF SELECT AND ULTIMATE MORTALITY

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ABSTRACT

The objectives of this paper are (1) to show why scientific laws of mortality are preferable to the continued use of graduation techniques on mortality tables; (2) to develop a new theory leading to a new mathematical law of mortality that reduces to Gompertz's law as a first approximation, and to detail the biological justification for such laws; (3) to demonstrate an understanding of select mortality and produce a simple formula that is reasonably consistent with it; (4) to develop methods to test this and other similar formulas on various bodies of data to establish their validity and usefulness; (5) to discuss the meaning of the results produced above and provide some examples of the use of the formula for purposes other than graduation; and (6) to discuss extensions of the models.

The authors believe that the final formula is simple, easy to use, and considerably more useful than traditional graduation processes. The second-named author developed the models and wrote the sections dealing with their development and justification. The first-named author developed the techniques of regression analysis for testing and fitting the models and wrote the corresponding sections of this paper.

1. INTRODUCTION*

THE second-named author has been uncomfortable for many years with the use of graduation processes for smoothing mortality tables. Perhaps this is a result of his original training in the physical sciences, where the objective was always to discover the laws that govern events, and then to use those laws to predict the outcome of experiments. By contrast, graduation produces smooth data but avoids theory, and can give no hint of results where no data are available. Consider the following criticisms of graduation techniques:

1. By relying upon graduation techniques, we divorce ourselves from the biological sciences underlying this phase of actuarial work. This paper may

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be viewed as a step toward re-grounding actuarial science in the substructure of biology and gerontology.

2. The use of a graduated mortality table requires a religious level of faith. After disclaiming (by using a graduation technique) any understanding of what is actually going on, the graduator takes a leap of faith and uses the resulting table to predict what will occur in the future.
3. Using graduation techniques, one can never be sure whether significant data are being left out or "smoothed." An example of such data is the dip in mortality in the late twenties.
4. It is not possible to compare one graduated table with another simply, because such tables consist only of data points; they are not built on a small number of parameters having intrinsic meaning. Because of the construction methods used, each table is unique; there is no basis for comparison of the whole entities.
5. Because of the methods used to create a graduated table, there is no way to extend it to other ages or durations. Obtaining an estimate of mortality for ages beyond the central ages of groupings requires an extrapolation technique entirely foreign to the procedure originally used to create the table.
6. The lack of reference to a theory prevents us from improving our understanding of what is going on. If we are working from a theory, we observe deviations and thereafter improve the theory. If we are working with graduation techniques, we can only go on to the next graduation.

The advantages of working from a mathematical theory of mortality are the reverse of the above-stated criticisms:

1. We will have reestablished our relationship with the biological sciences, and particularly the recent developments in cellular biology and gerontology, and will be in a position to evaluate the effects on mortality statistics of changes in medical knowledge and treatment.
2. We can use the resulting table to predict the future levels of mortality because we believe that we understand most of the biological processes involved.
3. If systematic departures from our law appear in the data, we immediately become aware of them and make a positive decision as to necessary revisions in the law.
4. We should be able to compare two tables by comparing only a few parameters that we believe have some objective meaning.
5. A law should provide us with a built-in method of extrapolation beyond the range of the available data, or with other extensions of the usefulness of the available data. For example, a law of mortality might help us to estimate the effect on mortality differentials by age of the discrete changes in underwriting requirements as age advances.
6. We will be encouraged to improve our theories and our understanding of the underlying processes.

II. MATHEMATICAL LAWS OF MORTALITY, HUMAN VITALITY, AND GOMPERTZ'S LAW

An attempt to develop a law for select mortality would seem first to require some understanding of the work already done on laws of mortality that apply to unselected groups of lives, work that has preoccupied the actuarial profession over a large part of its history. We might begin by asking the question, what should be required for a mathematical relationship to be called a law of mortality? It seems to the authors that there are several generally accepted requirements:

1. The law should have been observed over a period of years and in different environments.
2. It should be usable for predictive purposes.
3. It should be consistent with other bodies of information, so that it is plausible from the point of view of such other disciplines and sciences as may be applicable.
4. There should be enough logical analysis behind it so that we can feel confident that we understand what influences would cause the crucial parameters to change. We should be able to judge when changes in the environment would necessitate changes in the parameters of the equation.

Laws of mortality can, and in special environments do, take on special forms. A law of mortality for birds in the wild seems to be that the rate of mortality is constant—in this environment, the accidental death factor apparently predominates. Mortality of soldiers is related more closely to the time spent in actual combat than to any other factor. Similarly, duration of exposure to radioactive emission seems to be an important factor in the total life span of experimental animals. These last two examples suggest the possible importance of a temporary deleterious environment. The law of mortality for chickens on an ocean cruise, where the same number can be expected to die every day, is a very special example of the environmental effect. The law of mortality for salmon—the rate becomes 1.0 after spawning—is another form that is consistent with our present knowledge of the biology involved.

These laws of mortality emphasize the importance of the effects of the environment. However, actuaries normally have been concerned with the changes in mortality rate associated with aging, sex, and race or nationality. The earliest law of mortality still in use is that of Gompertz. His paper [7] before the Royal Society in 1825 is still of interest. Unfortunately, his law was simply a statement of his observations; it seemed to be true for mortality tables then in existence.

Actuaries are familiar with the original Gompertz law, $\mu_x = Bc^x$, and also with Makeham's first modification, $\mu_x = Bc^x + A$. They are not

generally familiar with Makeham's second modification, $\mu_x = Bc^x + A + Hx$, or with the addition of the quadratic term $\mu_x = Bc_1^x c_2^{x^2}$, although the latter has been used at least once [5]. Can any of these pass the required criteria for a law of mortality?

Consider Gompertz's original insight—that mortality increases as an exponential function, with age as the power of some constant. How wide is the support for this observation? Figure 1, taken as are most of the

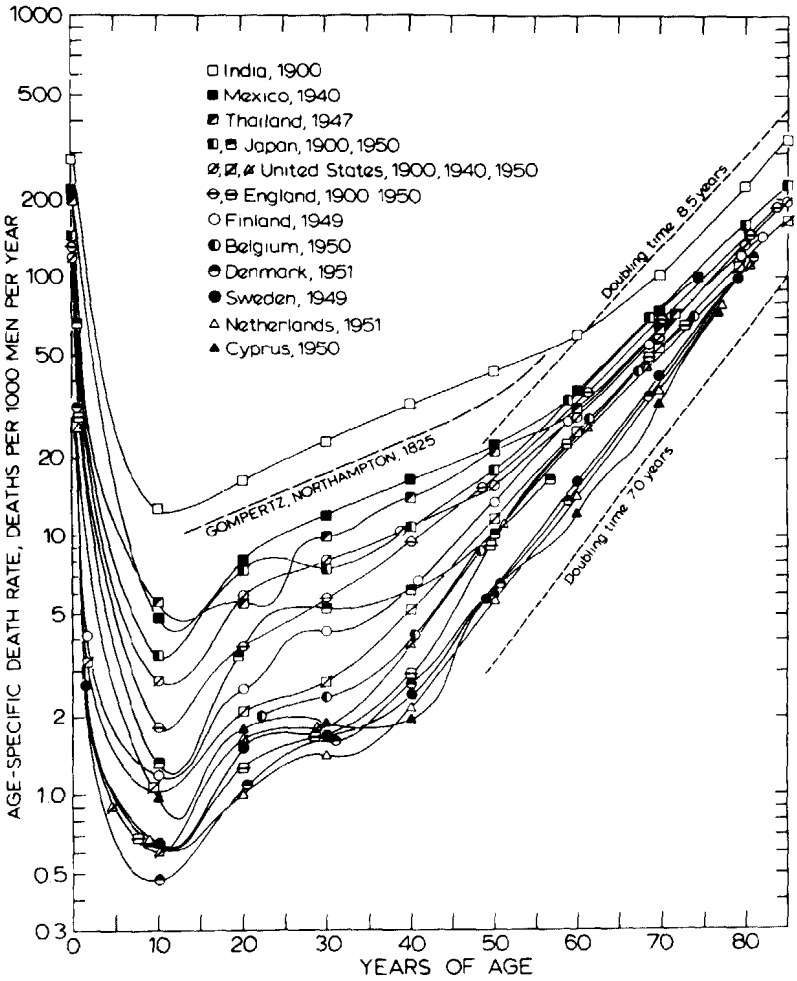


FIG. 1.—Age-specific death rates in various countries and years. (From Jones [10].) [From *Handbook of the Biology of Aging*, edited by Caleb E. Finch, Ph.D., and Leonard Hayflick, Ph.D. © 1976 by Litton Educational Publishing, Inc. Reprinted by permission of Van Nostrand Reinhold Company.]

illustrations in this paper from the *Handbook of the Biology of Aging* [6], gives graphic evidence of the widespread observations that follow this exponential increase. In every case, the mortality of males becomes a straight line on semilog paper after the age of maturity of the organism, about age 30. Figure 2 shows a series of mortality graphs for females in Sweden from 1751 to 1950. These graphs exhibit the same pattern. Variations in mortality, such as the dip in mortality in the late twenties attributable to reduced accident rates, or the relationship between female mortality and the period of childbearing, are not exceptions to the law of increasing mortality with age but rather modifications of it. Mortality at the younger ages currently seems related to accident rates and genetic weaknesses; these ages precede the period of effect of the

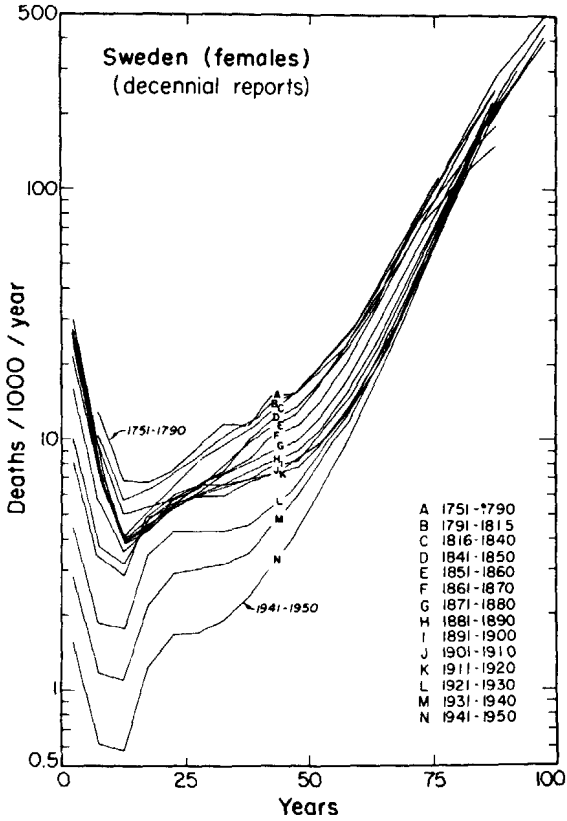


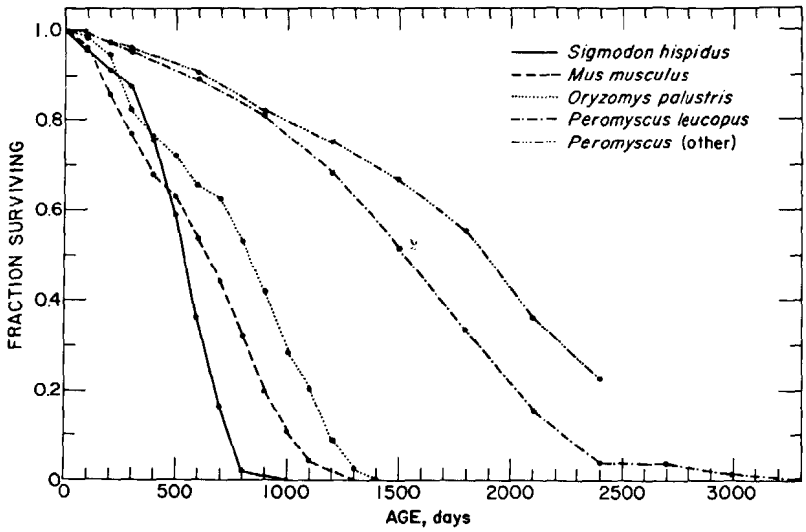
FIG. 2.—Age-specific death rates of Swedish females in various periods from 1751 to 1950. (From Jones [9].) [From *Handbook of the Biology of Aging*, edited by Caleb E. Finch, Ph.D., and Leonard Hayflick, Ph.D. © 1976 by Litton Educational Publishing, Inc. Reprinted by permission of Van Nostrand Reinhold Company.]

increase-with-age law. Landahl [13] developed a law of mortality based upon acquired immunity to disease, which was useful during the period in our history when infectious disease was an important cause of death among the young; however, this no longer seems to be an important factor. Figure 3 shows the mortality of various series of laboratory mice. Data (not shown) on the mortality of the common housefly provide further support for the general applicability of Gompertz's law, at least as a first approximation. Figures 4 and 5 show that the same basic pattern exists for specific causes of death in man and in rats.

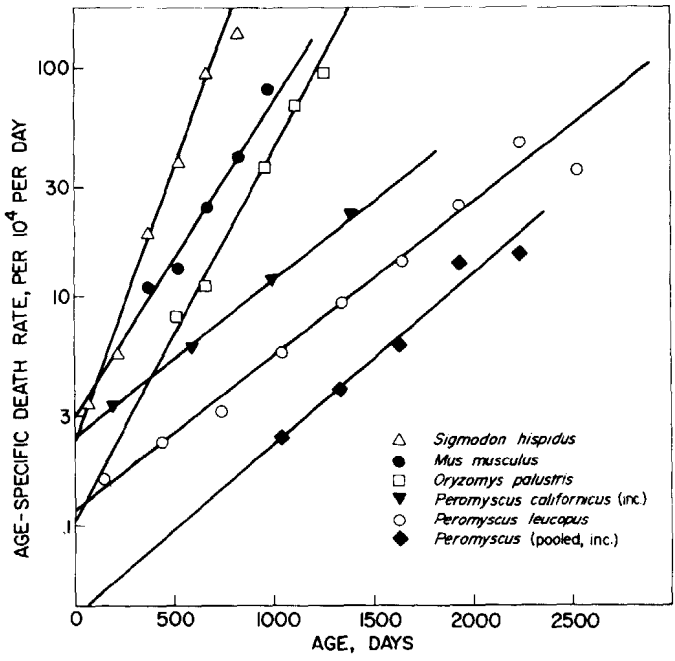
The above examples appear to provide adequate justification for the claim that Gompertz's law passes the first stated test—it has wide applicability, not only for humans but for other life forms. The second criterion was the use of the law for prediction. Since it has been used for this purpose in the construction of annuity tables over many years, and many companies have succeeded by putting their money on its correctness, this criterion would seem to be satisfied also. The crucial remaining criteria, and they are ones that do not seem to have been handled satisfactorily over the years, are those having to do with plausibility, consistency with other bodies of knowledge, and enough understanding so that modifications could be made when needed.

Before these problems are examined directly, we should first review what is known currently about the rate of deterioration in the various systems of the body. The surprising factor here is that the effectiveness of the various physical systems of the body does not decrease exponentially with age but rather decreases linearly. Perhaps the most crucial example of this linear trend is that of the actual ability of cells to reproduce themselves. Hayflick [6, p. 160] reports the results of a variety of studies on this subject. The evidence seems clear that a human cell can reproduce between forty and sixty times during its lifetime. More important, however, are the studies that show that the number of possible reproductions remaining for a given cell is reduced by 0.20 for each year of attained age of the donor. These figures apply only to normal cell populations, not malignancies, and the statistics have been subject to some criticism, but the general observation about the loss in function seems well supported.

Figure 6 shows the decline with age in various physiological functions of the human body. The general pattern of the loss of a constant percentage of the original value for each year of age seems obvious. Figures 7 and 8 show the decline in vital capacity and filtration rate of the kidneys for normal men and women. The linear decrease in physiological



(A)



(B)

FIG. 3.—Plots of survivorship (*A*) and of logarithm of rate of mortality or Gompertz function (*B*) versus age, for wild-type populations of five rodent species bred and reared in captivity (From Sacher and Staffeldt [22].) [From *Handbook of the Biology of Aging*, edited by Caleb E. Finch, Ph.D., and Leonard Hayflick, Ph.D. © 1976 by Litton Educational Publishing, Inc. Reprinted by permission of Van Nostrand Reinhold Company.]

functions seems as well established as the exponential increase in mortality with age.

In order to make Gompertz's law plausible, we must explain how a linear decrease in physiological function leads to an exponential increase in mortality with age. There have been a number of recent attempts at such an explanation. The one that is probably most familiar to actuaries is that of David Brillinger [3]. Brillinger points out that death is the result of the failure of one of the systems of the body necessary for life and that the probability of death is the sum of the probabilities of failure

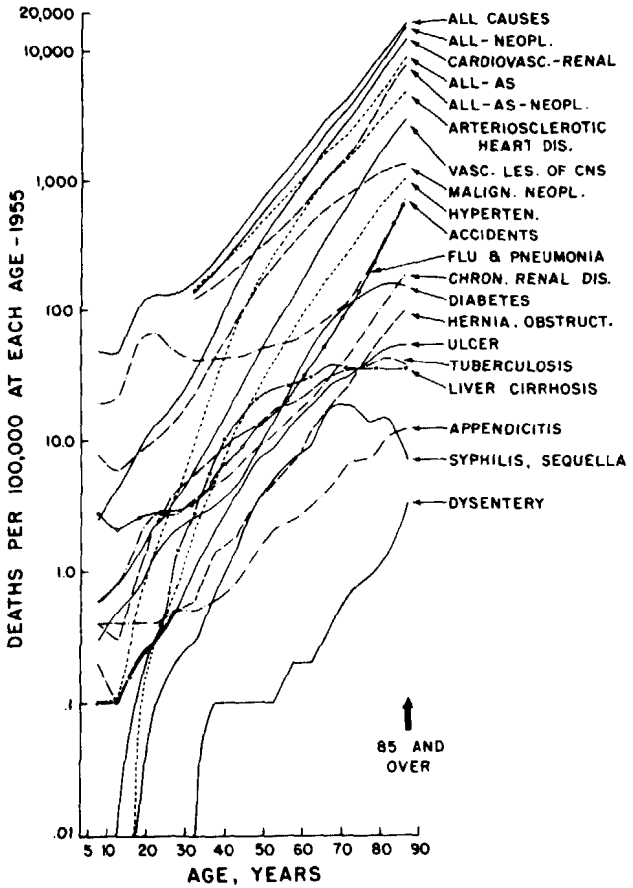


FIG. 4.—Age-specific death rates for major causes, in the United States, 1955. (From Kohn [12].) [From *Handbook of the Biology of Aging*, edited by Caleb E. Finch, Ph.D., and Leonard Hayflick, Ph.D. © 1976 by Litton Educational Publishing, Inc. Reprinted by permission of Van Nostrand Reinhold Company.]

of each of the individual systems. He then proposes a model in which each year the value of each of these systems is represented by a designated value. Then, using the extreme-value theory of Fisher and Trippet (later developed in a most practical fashion by Gumbel [8]), he develops the statistical laws that can represent the lowest values selected from the assumed identical distributions of vitality in each such system. This reasoning leads to the very general law

$$\mu_x = \Sigma H_i(x - B_i)^{c_i - 1} + \Sigma A_j/(b_j - x)^{c_j + 1} + \Sigma E_k d_k^x .$$

The authors of this paper have some difficulty in accepting the appropriateness of this model. Is aging represented simply as the successive results of choosing a value from a randomly distributed variable, with death the result of the lowest value being below some present level? The model would be equally applicable if none of the physiological variables discussed above were changing with age. The fact that they are changing with age argues against the applicability of a purely statistical model. An additional problem with this approach is that the law that finally results is too general. We do not observe the vast multitude of terms

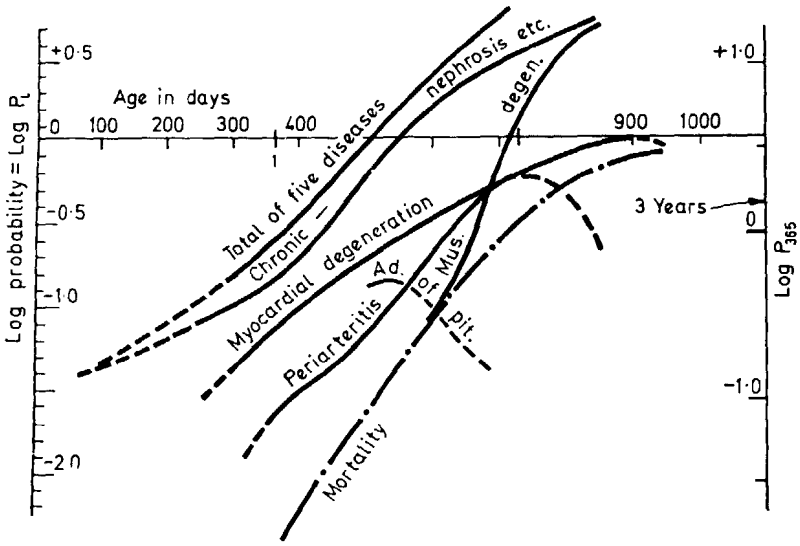


FIG. 5.—Age distribution at onset of major lesions, as compared with mortality from all causes, in aging rats. (From Simms [24].) [From *Handbook of the Biology of Aging*, edited by Caleb E. Finch, Ph.D., and Leonard Hayflick, Ph.D. © 1976 by Litton Educational Publishing, Inc. Reprint by permission of Van Nostrand Reinhold Company.]

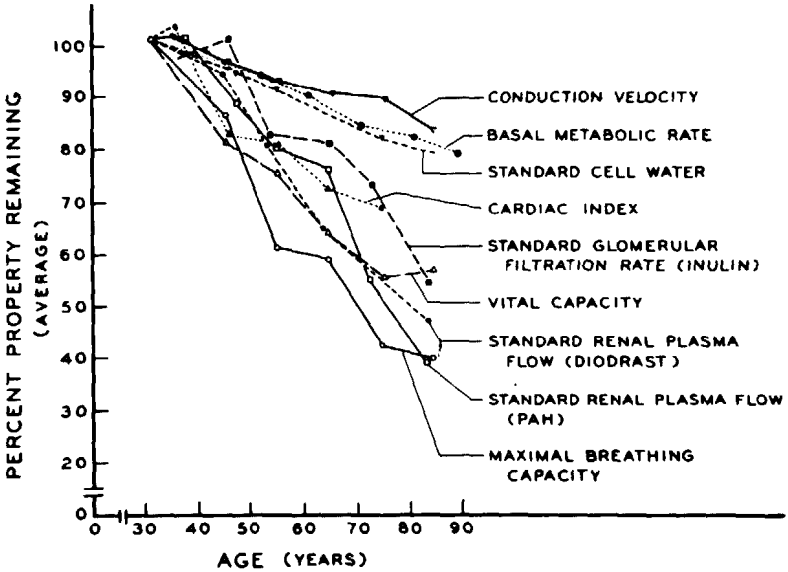


FIG. 6.—Decline with age in selected physiological functions in humans. (From Shock [23].) [From *Handbook of the Biology of Aging*, edited by Caleb E. Finch, Ph.D., and Leonard Hayflick, Ph.D. © 1976 by Litton Educational Publishing, Inc. Reprinted by permission of Van Nostrand Reinhold Company.]

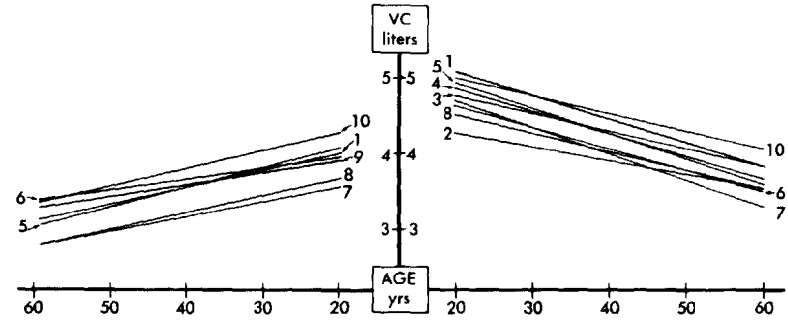


FIG. 7.—Changes in vital capacity with age in females (*left panel*) and males (*right panel*). (From Muiesan, Sorbini, and Grassi [17].) [From *Handbook of the Biology of Aging*, edited by Caleb E. Finch, Ph.D., and Leonard Hayflick, Ph.D. © 1976 by Litton Educational Publishing, Inc. Reprinted by permission of Van Nostrand Reinhold Company.]

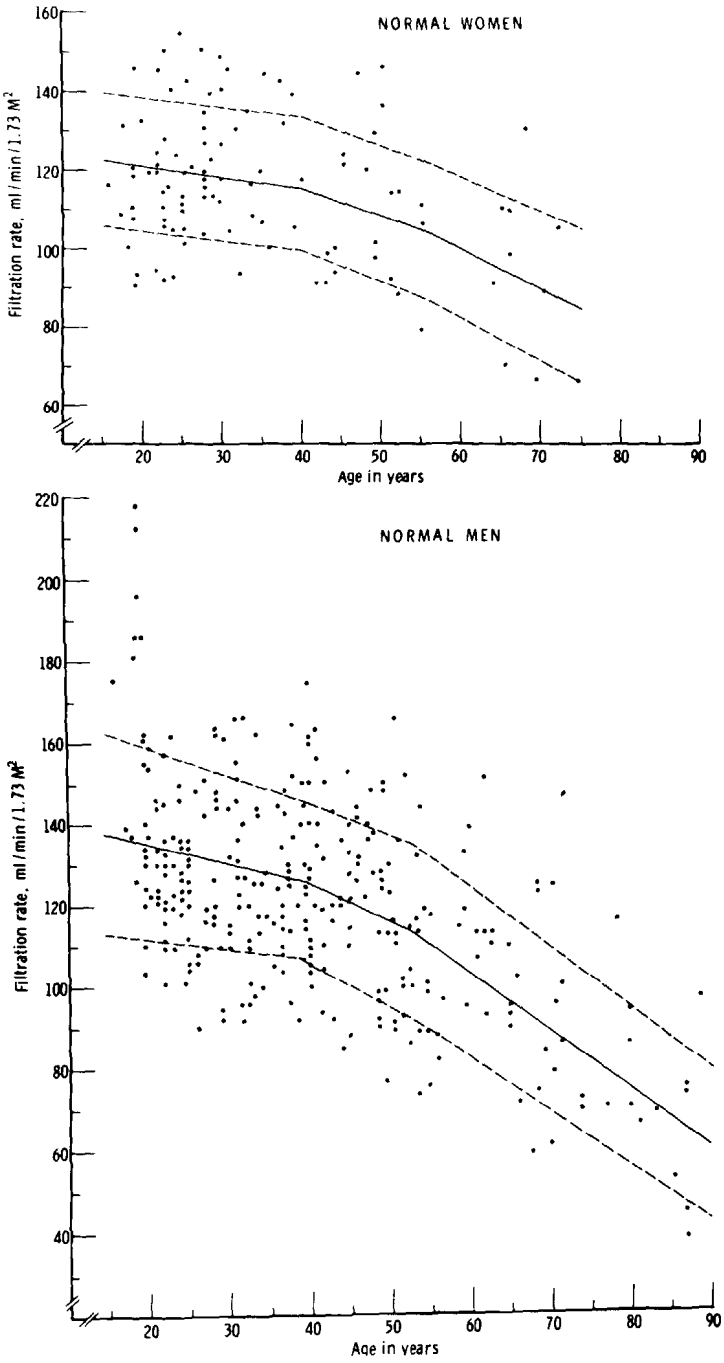


FIG. 8.—Relationship of age to filtration rate (inulin clearance) per 1.73 m² surface area in normal men and women. Solid line is the mean, by age, and dashed lines represent the limits of 1 standard deviation. (From Wesson [26].) [Wesson, *Physiology of the Human Kidney*, Grune & Stratton, New York, 1969, by permission of the publisher.]

made possible by this equation. While the approach is undoubtedly valid, and could be modified to consider the changing physiological parameters, it does not explain why one relatively simple pattern should dominate.

The development of Gumbel, using extreme-value theory, leads to Gompertz's law and provides the interesting additional insight that extreme age at death and average age at death are inversely related. Unfortunately, Gumbel begins with the assumption that the initial variate is of the exponential type. He has assumed the required form of Gompertz's law. This analysis by Gumbel should be sound once the basic riddle is solved.

Two other theories of mortality have been presented that are essentially biological in orientation. The Strehler-Mildvan theory [25] accepts the linear loss of vitality as a basic fact and develops Gompertz's law from the assumption that the stresses of life are distributed according to the Maxwell-Boltzmann distribution. However, since the form of the Maxwell-Boltzmann distribution is exponential, the Strehler-Mildvan theory cannot be regarded as an explanation of the rate of increase in mortality, despite the interesting results that follow from this approach.

The other modern biologically-oriented theory is that of Sacher-Trucco [21], which also accepts linear loss of function as a basic premise. The new assumption is that vitality (death resistance) at any age is distributed normally in the population. The mean of the distribution decreases linearly with age, but, as age increases, the proportion of the population falling below a minimum level of vitality, and therefore dying, increases at more than a linear rate. The theory is well developed, but Strehler has pointed out that the rate of loss of vitality required to fit the known increases in mortality is far greater than that actually observed. In addition, the theory is deficient for purposes of this discussion, since it does not answer the basic question of why an exponential increase should be observed at all. The Sacher-Trucco theory also introduces the exponential factor as the result of a specific additional assumption—that of the normal distribution of vitality. Such an assumption implies the existence of a small but real group with extraordinarily high vitality for their age. No such group seems to exist. Neither of these theories can rationalize the exponential form.

Each of the previously discussed theories can produce a rationale for Gompertz's law, but each of them must start with the assumption of the basic form. The Gumbel extreme-values-theory approach does not demand an exact exponential as a start, but it does require something of an exponential form. Similarly, the biologically oriented Strehler and

Sacher-Trucco theories depend upon the introduction of an exponential form.

III. A NEW THEORY OF ULTIMATE MORTALITY

In their paper "A Critique of Theories of Mortality," [15] Mildvan and Strehler list three specific criteria for a scientific theory of mortality:

1. The assumptions must not be qualitatively or quantitatively inconsistent with observation.
2. The theory must not make predictions that are qualitatively or quantitatively inconsistent with natural law or observation.
3. The number of assumptions should be kept to a minimum.

The observations that are listed as crucial in judging a theory are the following:

1. The law must be consistent with Gompertz's law over the large range of ages.
2. The rate of loss of physiological function for the system of the body is linear.
3. A large value of B in the Bc^x quantity of the Gompertz law is accompanied by a depressed value of c, and conversely—a relationship observed in many human populations.
4. The relation between continuous and intermittent exposure to radiation is such that continuous exposure to radiation increases the value of c in the Gompertz equation, while intermittent exposure increases the value of B but not the value of c.
5. The Gompertz function seems to apply to specific diseases as well as to total mortality.
6. Mortality seems to fall away from the Gompertz law at great ages.

Mildvan and Strehler conclude in their article that only the Strehler-Mildvan theory explains the first five observations. Sacher has elsewhere argued that the Sacher-Trucco theory is preferable, although Mildvan-Strehler's criticism is not answered. None of the reviewed theories explains the sixth observation.

The second-named author has never come across the following theory, which he believes is consistent with all six observations and involves much simpler assumptions than either the Strehler-Mildvan or the Sacher-Trucco theory. It produces the basic exponential form naturally and simply, as opposed to the two previously mentioned theories, which must specifically introduce it.

The theory is based upon the acceptance of the observation of linearity of loss of function of biological systems with age and the assumption that the essential nature of biological systems is redundancy. The observation of linearity seems well established in biological literature, and only some of the evidence in favor of it has been presented. Since

acceptance of it is also one of the Mildvan-Strehler criteria, it will be considered proved without further comment.

The second assumption of the theory, however, does require justification. Anyone who studies biological systems must be impressed by their basis of organization—they exhibit redundancy; that is, they contain backup systems in excess of those needed for functioning. If we consider the nervous system in particular, we are impressed with the parallelism of its operation. There are many connections between nerve fibers, and the basis for the connections is parallelism. A nerve fails to transmit a message only if all, or almost all, of the various cells fail. Similarly, other systems of the body fail only when all components fail.

If a system fails only when all components fail, and there are n components, each with a probability of failure f, then the probability of failure of the system is q, and

$$q_0 = f^n .$$

If, over a period of time, a number x of the components are eliminated, then the new probability of failure of the modified system is

$$q_x = f^{n-x} .$$

The ratio of q_x to q_0 is

$$\frac{q_x}{q_0} = \frac{f^{n-x}}{f^n} = \frac{1}{f^x} = \left(\frac{1}{f}\right)^x ;$$

thus,

$$q_x = q_0\left(\frac{1}{f}\right)^x = f^n\left(\frac{1}{f}\right)^x .$$

This obviously has the form of Gompertz's law, and the loss of x components corresponds to the linear loss of vitality of biological systems.

If there are two systems, the failure of either of which will cause death,

$$q_0 = f^n + f^n - f^n f^n = 2f^n - f^{2n}$$

and

$$q_x = f^{n-x} + f^{n-x} - f^{n-x}f^{n-x} = 2f^{n-x} - f^{2(n-x)} .$$

The ratio q_x/q_0 is no longer so simple. Instead, it forms a series:

$$A_1 f^{-x} + A_2 f^{-2x} .$$

The law of mortality is then

$$q_x = q_0(A_1 f^{-x} + A_2 f^{-2x}) ,$$

or

$$q_x = B_1\left(\frac{1}{f}\right)^x + B_2\left(\frac{1}{f}\right)^{2x} .$$

Similarly, if there are three or more subsystems, the failure of any of which causes death, the number of terms that enter into the various

constants increases, and the form is now

$$q_x = B_1\left(\frac{1}{f}\right)^x + B_2\left(\frac{1}{f}\right)^{2x} + B_3\left(\frac{1}{f}\right)^{3x}.$$

As a final illustration, consider three systems. The failure of the first two "crucial" systems will cause death, but that of the third "semi-crucial" system will create only a probability a of death. Now

$$\begin{aligned} q_0 &= f^n + f^n + af^n - f^{2n} - af^{2n} - af^{2n} + af^{3n} \\ &= (2 + a)f^n - (1 + 2a)f^{2n} + af^{3n}; \\ q_x &= (2 + a)f^{n-x} - (1 + 2a)^{2n-2x} + af^{3n-3x}; \\ \frac{q_x}{q_0} &= A_1\left(\frac{1}{f}\right)^x + A_2\left(\frac{1}{f}\right)^{2x} + A_3\left(\frac{1}{f}\right)^{3x}; \end{aligned}$$

and

$$q_x = B_1c^x + B_2c^{2x} + B_3c^{3x}.$$

While we know that there are a number of systems in the body, we cannot, given the current state of our knowledge, say what they are or how many there are. There may be a number whose failure causes only a fractional probability of death. There may be a master system, such as the immunological system or the basic ability of cells to divide. Nonetheless, the formula for q_x always develops the same basic form—a power series in c (or $1/f$).

The reader must determine whether consideration of the organization of organic systems as parallel structures is a reasonable assumption consistent with observation, the first criterion of Mildvan and Strehler. We can, however, easily conform with their other crucial observations:

1. The Gompertz law is the first term of the expansion and, therefore, the first approximation of the stated theory.
2. Linear rate of loss is taken as a basic assumption.
3. If the number of crucial systems is increased or if the value of a in semi-crucial systems is increased (an unfavorable environment), the higher-order terms become more important, and an attempt to fit the simple Gompertz law will develop a lower value of c, the slope variable. A higher value of B in the term Bc^x will correspond to a lower value of c. Tests of the law with two terms does not seem to support any significant variation in the value of c in different environments.
4. Exposure to radiation would cause a more rapid loss of the redundancy of the systems during the period of exposure. This, according to the model, would mean a change in the slope for continuous exposure, but only a change in the level for intermittent exposure.
5. Not only is the argument consistent with the observation that Gompertz's

law applies to specific diseases; in fact, the power-series form must follow if separate causes of mortality follow Gompertz's law.

6. The natural development of powers of c^x provides an automatic reduction in the rate of mortality rate increases at higher ages. The addition of only the second-order term radically improves the fit. Further tests will be published at some future time.

Only two further comments seem necessary. First, the theory is consistent with the addition of the constant Makeham term. It is only necessary to assume a small probability of exposure to an environmental hazard that always overwhelms all systems. Second, the formulas can be easily solved by linear regression techniques. Of course, there must be a number of tests for values of c to find the value that provides the best fit. Since c falls between 1.06 and 1.1, a limited number of trials, at most, may be necessary.

IV. SELECT MORTALITY

The following is an attempt to describe, very carefully and precisely, the development of select mortality rates over a period of years after selection. As such, it follows in the line of work of Levinson [14], Ziock [27], and Berger [2]. Unlike these authors, the present writers will attempt to develop the ideas without the use of mathematical notation until the computational model has been developed. (An earlier version of the paper used tensor notation for this section; however, since no one seemed to understand it, the authors decided that the most useful system of notation was probably words. The mathematically sophisticated reader can supply either matrix or tensor notation as suits his background.)

Underwriters contend that normally they are capable of distinguishing about fifteen separate mortality classes at any age. Let us assume that, with sufficient information, twenty different classes can be delineated. Unlike Levinson's classes, these classes are operational distinctions. We will assume that all lapses and changes in class occur once a year.

The selection process establishes that certain of these classes are eligible for standard insurance; about 90 percent of lives applying for insurance fall into these classes. There is a mortality rate for each of the twenty mortality classes for each year of attained age. The observed select mortality rate is the product of the rates for each select class times the number of lives in the class divided by the total number of lives in all select classes.

Lapses also occur. In principle, it would be possible to establish separate lapse rates for each mortality class. In practice, we cannot develop such tables; nevertheless, we know that if the lapse rates are

higher in the lower mortality rate classes, the average mortality of the group will increase. Specifically, the ultimate group can show more rapidly increasing mortality (a higher Gompertz c) if these favored groups have high lapse rates. If an observed group of lives has unusually high lapse rates, it probably is safe to assume that a degeneration of the group is taking place.

Considering only deaths and lapses, we would end each year with the same classes we had at the beginning of the year, although the relative proportions may have been changed by lapses. There is a third modification of the group—class transformation. The various hazards and environmental influences transform each class. In fact, each class is transformed into each of the twenty classes. The vast majority of the members of any class are transformed into the same class, but there is some proportion that goes into each of the other classes.

In principle, in the absence of cost constraints, these transformations actually could be observed. The membership in each class at the beginning of the second year contains representatives from all classes existing at the beginning of the previous year. One peculiarity of the transformation process is that it always changes the ultimate group into itself. This corresponds mathematically to the fixed point of a stochastic matrix. This characteristic of the transformation process also means that any initial distribution of classes is transformed gradually into the distribution of the ultimate or unselected group.

In the second and later years, the same processes take place. The aggregate mortality rate of the originally standard group is now affected by the presence of other than standard class members, even though the set of mortality rates for each class depends only on attained age. Lapses again can have the effect of changing the relative proportions in the various classes, and the transformation process once again moves the composition closer to that of the ultimate group.

The key ideas of this exposition are intended to be the following:

1. The characteristics of the transformation process.
2. The possible effects of lapses perhaps causing degeneration in the class composition even to mortality levels worse than in the ultimate group.
3. The fact that in this formulation every class composition, transformation, and mortality and lapse rate is, in principle, observable. There are no hypothetical unobservable groups or rates.

V. COMPUTATIONAL MODEL

We know that a body of ultimate lives usually follows Gompertz's law reasonably closely over the range of ages that are relevant for insurance

purposes and for which select mortality data are also available. We know that the select-to-select transition is very high—a continuous select body of lives exists. What pattern will continuously select life mortality follow? A good candidate for this pattern would seem to be the Gompertz formula. The arguments in favor of this particular pattern seem plausible.

Who is in the select group, and how do the members of this group differ from the general body of lives? Only in that they can, year after year, pass a medical examination. What does this mean? Only that they have not developed the specific impairments that an examination would demonstrate: (1) they have no high blood pressure; (2) they have no heart murmurs; (3) they have no history of heart attacks; (4) they have no history of cancer; and (5) they have no history of the various other conditions listed on the nonmedical portion of the application.

What we are really saying is that this group is immune to a variety of perceptible degenerations in the body. Other imperceptible losses do take place—immunity to disease is not perpetual youth. But the signs of these losses of vitality are not apparent to the underwriter. Many of the conditions to which the select group is immune follow the same slope as general mortality (see Fig. 4). If all diseases had the same rate of increase with age, we would expect only the coefficient B in a select version of Gompertz's law to be different from that in the ultimate version. However, some conditions do not follow the same slope, and some only start to appear or assume the general mortality slope after a specific age. For these reasons, the idea of a different exponentiated value seems consistent with the medical information presented in previous sections. We also must be aware of existing mortality data that show increasing percentage differences between ultimate and select mortality. This increasing percentage difference implies a lower c for select lives.

Even if Gompertz's law applied exactly to select lives, we would not expect perfect statistical obedience. Mortality statistics are subject to chance fluctuations. More important, even for a single company, the data are heterogeneous. Underwriting criteria change at discrete age intervals. The 40-year-old is selected according to different tests and criteria than the 39-year-old. The amount of insurance plays a varying role according to age. Underwriters change—the underwriter's frame of mind and the underwriting standards used can depend on the amount of pressure for new business at year-end. The underlying nature of the mortality involved is not constant. Styles in food and cigarette smoking, for example, change over the years. Incidence of cancer of the lung in men may seem to vary by age in the general population when all that is really happening is that the attitude toward cigarette smoking has

changed over the years and the effects of smoking are only now being exhibited.

Although we would not really expect one select mortality curve to fit Gompertz's law exactly, in some cases the variations from it may give additional insight into the underwriting and epidemiological processes involved. If we can accept the idea that mortality for an ultimate group of lives follows Gompertz's law and that the mortality of a group that remains continuously select also will follow Gompertz's law, with a different set of parameters, then the problem of developing a mathematical law that describes the progression of mortality rates over the select period is reduced to the problem of describing how mortality rates for an initially select group move from the select Gompertz curve to the ultimate Gompertz curve. The solution to this problem for this paper is presented in a conceptual form as formula (IA), the initial algorithm, which appears below.

The argument for the initial algorithm is as follows. If we begin with a group of select lives some will have died at the end of the first year in accordance with Gompertz's law for newly selected lives. Of the remaining lives, a certain proportion s will remain select and the remainder $(1 - s)$ will become ultimate. The mortality rate for the combined group in the second year then will be $sq_{[x+1]} + (1 - s)q_{x+1}$. In the next year the ultimate group will stay ultimate (by definition of an ultimate group), and the survivors of the select group can again be split into a portion that continues as first-year select and a portion that goes into the ultimate group. If the proportion of select lives that remains select is the same as it was in the previous year and if the difference between the select and ultimate rates of mortality is small compared to the value of s (which can be demonstrated), then the mortality rate for the next year for the combined group will be $s^2q_{[x+2]} + (1 - s^2)q_{x+2}$. Following the same argument for successive years leads immediately to formula (IA), which is

$$\mu_{[x]+t} = s^t\mu_{[x+t]} + (1 - s^t)\mu_{x+t}\,. \tag{IA}$$

We have already argued for the use of Gompertz's law for both select and ultimate mortality. Hence

$$\mu_{[x]+t} = s^tB_sc_s^{x+t} + (1 - s^t)B_uc_u^{x+t}\,. \tag{1}$$

The additional approximation involves replacing expression (1) by the geometric average:

$$\mu_{[x]+t} = (B_sc_s^{x+t})^{s^t}(B_uc_u^{x+t})^{1-s^t}\,. \tag{2}$$

It can be shown that equations (1) and (2) are approximately equivalent (see Appendix I). The use of equation (2) implies that $\mu_{[x]+t}$ follows Gompertz's law for all values of t. We will write equation (2) as follows:

$$\mu_{[x]+t} = Bc^{x+t}(B_3c_3^{x+t})^{s^t}, \tag{3}$$

where $B = B_u$, $c = c_u$, $B_3 = B_s/B_u$, and $c_3 = c_s/c_u$, and refer to this as Model III or Vanderhoof's law. This law actually involves two separate assumptions. The first is that both select and ultimate mortality may be represented by Gompertz curves. The second is the specific way mortality goes from one curve to another. Other laws of select mortality, referred to as Model I and Model II, respectively, are given by

$$\mu_{[x]+t} = B_1r_1^tc_1^{x+t}, \tag{4}$$

$$\mu_{[x]+t} = Bc^{x+t}(B_2c_2^{x+t})^{1/(t+1)}. \tag{5}$$

These three models will be considered in this paper.

In Section VI we will compare characteristics of these three models. In Section VII we will discuss the estimation of the parameters of these three models from data on crude mortality rates. The remaining sections deal with the application of these methods to obtaining estimated mortality models using the data of (*a*) Society of Actuaries 1965–70 Basic Tables, (*b*) Society of Actuaries 1955–60 Basic Tables, and (*c*) Equitable male experience, 1965–70. The usefulness of these three models will be discussed and the fit to the real data assessed.

VI. CHARACTERISTICS OF THE MODELS FOR SELECT MORTALITY

The first-named author believes that, in order to compare the three models of select mortality introduced in Section V, it is useful to represent the force of mortality as $\mu_{[x-t]+t}$. The resulting expressions yield the force of mortality corresponding to attained age x and duration t. From equations (4), (5), and (3), respectively, Models I, II, and III can be expressed as follows:

$$\text{I.} \quad \mu_{[x-t]+t} = B_1r^tc_1^x; \tag{6}$$

$$\text{II.} \quad \mu_{[x-t]+t} = Bc^x(B_2c_2^x)^{1/(t+1)}; \tag{7}$$

$$\text{III.} \quad \mu_{[x-t]+t} = Bc^x(B_3c_3^x)^{s^t}, \quad 0 < s < 1. \tag{8}$$

Any select mortality law should have the following characteristics: (1) $\mu_{[x-t]+t}$ should be an increasing function of t, and (2) the limiting value

of $\mu_{[x-t]+t}$ as t becomes arbitrarily large should be the ultimate Gompertz mortality curve, that is,

$$\lim_{t\to\infty} \mu_{[x-t]+t} = \mu_x = Bc^x .$$

Models I, II, and III satisfy characteristic 1, provided that $r > 1$, $B_2c_2^x < 1$, and $B_3c_3^x < 1$ for all attained ages x. Models II and III satisfy characteristic 2. Although Model I does not satisfy characteristic 2, we will consider it as a first approximation to the building of a select mortality model.

All three models can be represented as Gompertz curves, for fixed duration t, as follows:

$$\mu_{[x-t]+t} = B(t)[c(t)]^x .$$

Model I assumes $c(t) = \text{constant} = c_1$, and $B(t) = B_1r^t$; Model II assumes that $c(t) = cc_2^{1/(t+1)}$ and $B(t) = BB_2^{1/(t+1)}$; Model III assumes that $B(t) = BB_3^{s^t}$ and $c(t) = cc_3^{s^t}$. In Model I, the simplest case, it is assumed that $B(t)$ rises exponentially with duration. In Model III, if both B_3 and c_3 are less than 1, both $B(t)$ and $c(t)$ decrease with duration. If $B_3 > 1$ and $c_3 < 1$, then $B(t)$ increases with duration and $c(t)$ decreases with duration. The case in which B_3 and c_3 both exceed 1 is impossible because this would violate the inequality $B_3c_3^x < 1$. The remaining case, $B_3 < 1$ and $c_3 > 1$, is possible but did not occur in our empirical work. Model II exhibits the same trends as Model III in this regard if we consider the parameters B_2 and c_2.

Equation (2) implies that, under Model III,

$$\mu_{[x-t]+t} = \mu_{[x]}^{s^t}\mu_x^{1-s^t} .$$

Thus, the force of mortality is a weighted geometric average of the ultimate force of mortality and the select force of mortality at duration 0. Model II has the same property, since, from equation (7),

$$\mu_{[x-t]+t} = \mu_{[x]}^{1/(t+1)}\mu_x^{t/(t+1)} .$$

A useful measure of comparison of the three models of select mortality is given by

$$Q(t) = \frac{1}{\mu_{[x-t]+t}} \frac{\partial \mu_{[x-t]+t}}{\partial t} .$$

$Q(t)$ measures the rate of increase of the force of mortality as duration increases for a fixed attained age, where the rate of increase is expressed as a proportion of the force of mortality. It can be shown from equations (6), (7), and (8) that, for Models I, II, and III, respectively,

$$\text{I.} \quad Q(t) = \ln r ;$$

$$\text{II.} \quad Q(t) = -\frac{\ln (B_2 c_2^x)}{(t+1)^2} ;$$

$$\text{III.} \quad Q(t) = s^t \ln (B_3 c_3^x)(\ln s) .$$

Since $r > 1$, $B_2 c_2^x < 1$, $B_3 c_3^x < 1$, and $0 < s < 1$ for all x, $Q(t) > 0$ for all three models. For Model I the proportional increase in the force of mortality as duration increases for fixed attained age is constant for all durations. For Model II the proportional increase is a decreasing hyperbolic function of t, whereas for Model III the proportional increase decreases exponentially with duration. Empirically, we will discover that the value of s in Model III is in the range (0.7, 0.8). If we compare Model II with Model III in terms of the rate of increase in the force of mortality as duration increases, the conclusion is that the rate of increase decreases more sharply under Model II. This effect is depicted in Figure 9.

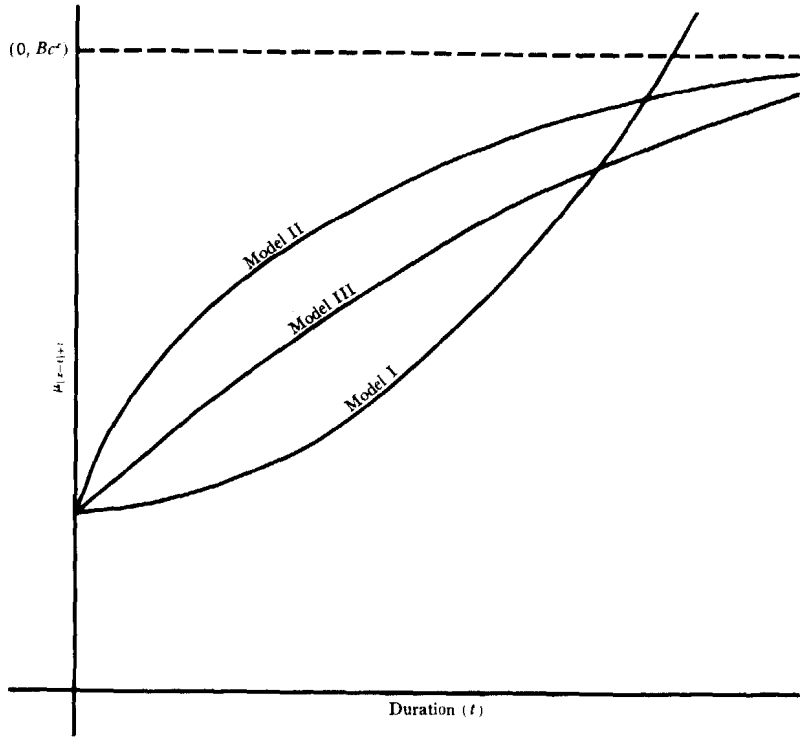


FIG. 9.—Graph of the select force of mortality as a function of duration (t) for fixed attained age (x) for Models I, II, and III.

For Models II and III, the transition to the Gompertz ultimate mortality curve is smooth. For Model I, $Q(t)$ = constant. Hence, the select mortality model does not tend to the ultimate Gompertz curve for increasing durations. The practical utility of these three models for describing the behavior of select and ultimate mortality will be tested empirically by using data on crude mortality rates.

VII. ESTIMATION OF PARAMETERS BY WEIGHTED LEAST SQUARES

The data to which these mortality laws will be fitted are the crude rates $\hat{q}_{[x-t]+t}$. In order to estimate the parameters of the three select mortality models discussed in the previous section, we must relate the true mortality rates $q_{[x-t]+t}$ under these three models. Theorems 1, 2, and 3 state the results for Models I, II, and III, respectively. Theorem 4 states the relationship between the true ultimate mortality rates q_x and the parameters of the ultimate Gompertz model. These theorems are proved in Appendix II.

THEOREM 1. *For Model I given by equation* (6),

$$Y_{xt} = \ln [\operatorname{colog} (1 - q_{[x-t]+t})] \doteqdot \alpha_0 + \alpha_1 x + \alpha_2 t , \tag{9}$$

where

$$\alpha_0 = \ln (B_1) + \ln (rc_1 - 1) - \ln (\ln rc_1) ,$$

$$\alpha_1 = \ln c ,$$

$$\alpha_2 = \ln r ,$$

and

$$q_{[x-t]+t} = 1 - \exp\left[- \frac{B_1 r^t c_1 (rc_1 - 1)}{\ln rc_1} \right] .$$

THEOREM 2. *For Model II given by equation* (7),

$$Y_{xt} = \ln [\operatorname{colog} (1 - q_{[x-t]+t})]$$

$$\doteqdot \alpha_0 + \alpha_1 (x + 0.5) + \alpha_2 \left(\frac{x + 0.5}{t + 0.5} \right) + \alpha_3 \left(\frac{1}{t + 0.5} \right) , \tag{10}$$

where

$$\alpha_0 = \ln B , \quad \alpha_1 = \ln c , \quad \alpha_2 = \ln c_2 , \quad \alpha_3 = \ln B_2 ,$$

and

$$q_{[x-t]+t} = 1 - \exp [-Bc^{x+0.5} (B_2 c_2^{x+0.5})^{1/(t+0.5)}] .$$

THEOREM 3. *For Model III given by equation (8),*

$$Y_{xt} = \ln [\operatorname{colog} (1 - q_{[x-t]+t})] \doteq \alpha_0 + \alpha_1(x + 0.5) + \alpha_2 s^{t+0.5}(x + 0.5) + \alpha_3 s^{t+0.5}, \tag{11}$$

where

$$\alpha_0 = \ln B, \quad \alpha_1 = \ln c, \quad \alpha_2 = \ln c_3, \quad \alpha_3 = \ln B_3,$$

and

$$q_{[x-t]+t} = 1 - \exp [-Bc^{x+0.5}(B_3 c_3^{x+0.5})^{s^{t+0.5}}].$$

THEOREM 4. *Consider the ultimate Gompertz curve*

$$\mu_x = Bc^x.$$

Then

$$Y_x = \ln [\operatorname{colog} (1 - q_x)] \doteq \alpha_0 + \alpha_1 x, \tag{12}$$

where

$$\alpha_0 = \ln B + \ln (c - 1) - \ln (\ln c),$$

$$\alpha_1 = \ln c,$$

and

$$q_x = 1 - \exp\left[-\frac{B(c - 1)c^x}{\ln c}\right].$$

Suppose we have available crude mortality rates that are defined as $\hat{q}_{[x-t]+t}$ and $\hat{q}_x$ for the select and ultimate rates, respectively, for which the true rates of the underlying mortality table are $q_{[x-t]+t}$ and q_x, respectively. Under Models I, II, and III, the true rates are expressed as functions of x and t by equations (9), (10), and (11), respectively. In order to estimate the parameters in these select models, we can use linear least squares in the following manner: first transform the select crude mortality rates by the following equation:

$$\hat{Y}_{xt} = \ln [\operatorname{colog} (1 - \hat{q}_{[x-t]+t})]; \tag{13}$$

then choose the parameters to minimize

$$\mathrm{SS} = \Sigma_t \Sigma_x (\hat{Y}_{xt} - Y_{xt})^2,$$

where Y_{xt} is given by equations (9), (10), and (11) for Models I, II, and III, respectively.

By transforming from the crude mortality rates to the Y_{xt} variable, we have linearized the relationship for Models I and II. In Model III the relationship is also linear if s is assumed known. Consequently, the methods of linear least squares in multiple linear regression as discussed by Draper and Smith [4] can be used to estimate the parameters for the various mortality models.

One of the assumptions of multiple linear regression is that the errors have equal variances. The implication is that the variance of $\hat{Y}_{xt}$ for a given age-duration class is constant. This assumption is not valid. It is proved in Appendix III that

$$\text{Var}\,(\hat{Y}_{xt}) \doteq \frac{1}{\theta_{xt}}, \tag{14}$$

where θ_{xt} is the number of deaths in the attained-age–duration class of the mortality study upon which the crude mortality rates are based. To correct for the inequality of the variances, a weighted-least-squares approach is adopted in which the parameters are estimated by minimizing

$$\text{WSS} = \Sigma_x \Sigma_t \theta_{xt}(Y_{xt} - \hat{Y}_{xt})^2 . \tag{15}$$

We will use the method of weighted least squares throughout this paper. The general method of weighted least squares is discussed in Appendix IV. In order to carry out the computations of Appendix IV, the methodology contained in a computer package known as statistical analysis system (SAS) [1] was used on the data under discussion in this paper. This method will also be used to estimate B and c for the ultimate Gompertz curve. In this case, we will minimize

$$\text{WSS} = \Sigma_x (\hat{Y}_x - Y_x)^2\theta_x ,$$

where Y_x is given by equation (12), θ_x is the number of ultimate deaths corresponding to attained age x, and

$$\hat{Y}_x = \ln [\text{colog}\,(1 - \hat{q}_x)] . \tag{16}$$

VIII. APPLICATIONS OF THE MODELS TO EMPIRICAL DATA

In Sections IX, X, and XI we present the results of fitting Models I, III, and II, respectively, using the method of weighted least squares discussed in the previous section. We will use five sets of select and ultimate crude mortality rates:

1. Equitable Life Assurance Society's male experience for 1965–70.
2. Male experience intercompany study for the 1965–70 Basic Tables [19].
3. Female experience intercompany study for the 1965–70 Basic Tables.
4. Male experience intercompany study for the 1955–60 Basic Tables [18].
5. Female experience intercompany study for the 1955–60 Basic Tables.

The data on select crude mortality rates are based on amount of claims and amount of exposure. The select rates pertain to starting-age groups (20–24, 24–29, . . . , 65–69, over 70) and fifteen groups of policy years. In order to use these data for estimating the parameters in the various mortality laws discussed in this paper, the following assumptions were made:

a) The attained age x and duration t for a given crude mortality rate were assigned values $x = m + p - 1$ and $t = p - 1$, where m is the midpoint of the starting-age interval and p is the policy year.

b) Crude mortality rates corresponding to attained ages under 30 were not considered, thus avoiding the characteristic dip in mortality rates that is not consistent with Gompertz's law.

c) Crude mortality rates in the over 70 starting-age group were not considered.

d) Average face amounts of policies and average death claims were assumed not to vary significantly over the various age-duration cells considered.

The last assumption is required because the values of the crude mortality rates are based on amount of claims rather than number. To use weighted least squares, we require the number of claims θ_{xt} as opposed to amount. In order to use amount-related data, we must assume that the crude mortality rates based on amount are close to the crude mortality rates based on number and that the amount of death claims is proportional to the number of death claims in the age-duration cells.

IX. APPLICATIONS OF MODEL I

To determine the estimates of B_1, r, and c_1 in Model I as expressed by equation (6), we first use weighted least squares to estimate α_0, α_1, and α_2 of equation (9). This procedure was carried out on the five data sets described in the preceding section. The next step is to solve the equations in Theorem 1 for B_1, r, and c in terms of α_0, α_1, and α_2. This yields

$$c_1 = \exp \alpha_1 , \qquad r = \exp \alpha_2 ,$$

$$B_1 = \frac{(\alpha_1 + \alpha_2) \exp \alpha_0}{\exp (\alpha_1 + \alpha_2) - 1} .$$

Thus, B_1, r, and c_1 can be estimated.

Table 1 shows the estimated values of these parameters along with

TABLE 1

ESTIMATES OF PARAMETERS OF MODEL I

PARAMETER	DATA SET				
	1 Equitable Male 1965–70	2 Combined Male 1965–70	3 Combined Female 1965–70	4 Combined Male 1955–60	5 Combined Female 1955–60
$B_1 \times 10^5$	3.3262	3.9024	6.8879	3.9508	3.3901
r	1.0323	1.0456	1.0486	1.0451	1.0414
c		1.0917	1.0701	1.0929	1.0856
$100R^{2*}$	95.6%	98.4%	87.8%	98.4%	84.2%

* $100R^2$ = percentage of explained variation.

the usual measure of fit $100R^2$, where R^2 is the proportion of explained variation, defined as

$$R^2 = 1 - \frac{\Sigma_t \Sigma_x (Y_{xt} - \hat{Y}_{xt})^2 \theta_{xt}}{\Sigma_t \Sigma_x (Y_{xt} - \bar{Y})^2 \theta_{xt}},$$

and

$$\bar{Y} = \frac{\Sigma_t \Sigma_x Y_{xt} \theta_{xt}}{\Sigma_t \Sigma_x \theta_{xt}}.$$

Tests of significance on the individual parameters α_0, α_1, and α_2 (using the t-test for the significance of regression coefficients) indicate that α_0, α_1, and α_2 are all nonzero (at the 5 percent significance level). This implies that both r and c are greater than 1.

From the results of Section VI, the parameter defined by $\alpha_2 = \ln r$ can be interpreted as $Q(r)$, the proportional rate of increase in the force of mortality as duration increases for fixed attained age. Model I assumes that this rate of increase is constant for all durations and attained ages. Since this assumption is not expected to hold in general, we can interpret $\alpha_2 = \ln r$ as the average proportional rate of appreciation in the force of mortality. This parameter is useful for comparing the average effect of selection in different mortality tables. For Equitable male data, this average rate of appreciation is 3.18 percent, whereas it is 4.56 percent for combined male experience (1965–70).

It is interesting to note that the values of B_1, r, and c_1 remain relatively stable for the two sets of combined male experience data (sets 2 and 4 of Table 1). This is not true for the combined female experience data. Model I fits male experience data better than female experience data, as is evidenced by the higher R^2 for male experience data.

The disadvantage of Model I is that we cannot obtain the estimates of the Gompertz ultimate curve to which the select mortality model tends as the duration increases. Models II and III do allow the estimation of the ultimate curve and are discussed in the next two sections.

X. APPLICATIONS OF MODEL III: THE VANDERHOOF MODEL

To determine the estimates of B, c, B_3, c_3, and s in Model III, we cannot use linear least squares because Y_{xt}, given by formula (11), cannot be converted to a linear combination of α_1 and known functions of x and t. On the other hand, if s is known, we can express Y_{xt} as

$$Y_{xt} = \alpha_0 + \alpha_1 Z_1 + \alpha_2 Z_2 + \alpha_3 Z_3,$$

where

$$Z_1 = x + 0.5, \quad Z_2 = s^{t+0.5}(x + 0.5), \quad Z_3 = s^{t+0.5}.$$

To estimate the parameters, we first guess at an initial starting value for s and use the method of weighted least squares to estimate α_0, α_1,

α_2, and α_3 above. An iterative approach, discussed in chapter 10 of Draper and Smith [4], is then used to obtain better estimates of s, α_0, α_1, α_2, and α_3. The iterations are continued until the reduction in the weighted sum of squares is insignificant.

This process is available on SAS [1]. Initial values in the range $0.5 < s < 0.8$ were used, and estimates of s, α_0, α_1, α_2, and α_3 were obtained. The estimates of B, c, B_3, and c_3 can be obtained from the results of Theorem 3 as

$$B = \exp \alpha_0 , \quad c = \exp \alpha_1 , \quad B_3 = \exp \alpha_3 , \quad c_3 = \exp \alpha_2 .$$

Table 2 shows the values of the estimated parameters for the five data sets under consideration. The values of B and c correspond to the ulti-

TABLE 2

ESTIMATES OF PARAMETERS OF MODEL III

PARAMETER	DATA SET				
	1 Equitable Male 1965–70	2 Combined Male 1965–70	3 Combined Female 1965–70	4 Combined Male 1955–60	5 Combined Female 1955–60
$B \times 10^5$	3.4922	5.2902	10.9861	6.0969	5.0809
c	.....	1.0966	1.0716	1.0953	1.0889
B_3	2.9515	1.2793	0.54682	0.64532	0.85167
c_3	0.96577	0.97775	0.99611	0.99260	0.98622
s	0.77136	0.76989	0.76127	0.78276	0.80948
$100R^2$	96.4%	99.1%	87.5%	98.9%	86.2%

mate Gompertz parameters, whereas the values of B_3 and c_3 represent the multiplicative corrections to adjust for the selection effect. If we compare the values of R^2 in Tables 1 and 2, it is evident that the fit of the data to Model III is an improved one, except for female mortality rates from the 1965–70 data.

For attained age x, the value of $B_3 c_3^x$ represents the ratio of the force of mortality at $t = 0$ to the ultimate force of mortality. If $c_3 = 1$, this ratio is independent of attained age. The values of c_3 are all less than 1, which implies that the effects of selection increase with attained age. The smaller the value of c_3, the greater is the decrease in selection effect for fixed B_3. The smaller the value of B_3, the greater the overall selection effect. Table 3 tabulates the values of $B_3 c_3^x$ for various attained ages and for the data sets under consideration.

For this model to be correct, the factor $B_3 c_3^x$ should be less than 1 for

all attained ages x. This holds true except when $x \leq 32$ in data set 1 (Equitable 1965–70 male data). Upon examination of the crude mortality rates, it is evident that this is a sampling fluctuation for the following reasons: (1) in starting-age group 30–34, the mortality rates decrease as the policy year $(t + 1)$ increases; (2) for these data, the selection effect was minimal in the lower age groups but more significant in the higher age groups; and (3) the model is valid only for attained ages in excess of 30.

TABLE 3

ESTIMATED RATIO OF FORCE OF MORTALITY AT DURATION 0 TO ULTIMATE FORCE OF MORTALITY (MODEL III)

ATTAINED AGE	DATA SET				
	1 Equitable Male 1965–70	2 Combined Male 1965–70	3 Combined Female 1965–70	4 Combined Male 1955–60	5 Combined Female 1955–60
30	1.038	0.651	0.486	0.516	0.562
35	0.872	0.582	0.477	0.497	0.524
40	0.733	0.520	0.467	0.479	0.489
45	0.616	0.465	0.459	0.462	0.456
50	0.517	0.415	0.450	0.445	0.425
55	0.435	0.371	0.441	0.429	0.397
60	0.365	0.332	0.433	0.413	0.370
65	0.307	0.296	0.424	0.398	0.345
70	0.258	0.265	0.416	0.384	0.332

For data set 1 (Equitable male data) the effects of selection vary substantially as a function of attained age. For data set 3 (1965–70 female combined data) the effects of selection are fairly constant for all attained ages. The other data sets fall between these two extremes.

XI. APPLICATIONS OF MODEL II

To determine the estimates of B, c, B_2, and c_2 in Model II, weighted linear least squares has been used, as discussed in Appendix IV. The output will yield estimates of α_0, α_1, α_2, and α_3 of equation (10). Then, by using the results of Theorem 2, the parameters in Model II can be estimated as

$$B = \exp \alpha_0 , \quad c = \exp \alpha_1 , \quad B_2 = \exp \alpha_3 , \quad c_2 = \exp \alpha_2 .$$

The estimated parameters are tabulated in Table 4. The values of R^2 are all higher for Model II than for Model I, although the differences are not substantial. The values of B and c correspond to the ultimate Gom-

pertz parameters and compare favorably with the corresponding values in Table 4 for Model III.

XII. COMPARISONS OF FIT TO MODELS I, II, AND III FOR SELECT MORTALITY

For ease of comparison, Table 5 shows the values of $100R^2$ for the five data sets and Models I, II, and III. From this table, we can see that in terms of the value of the percentage of explained variation, the Vanderhoof model (Model III) fits the male data best, whereas Model II fits the female data best. Since R^2 is a decreasing function of the weighted sum of squares, then, for a given data set, this latter measure of fit takes on its smallest value for Model III in the case of the male data and for Model II in the case of the female data.

Of course, the differences in the R^2 values are minimal. Models I, II, and III contain three, four, and five parameters, respectively, so that it

TABLE 4

ESTIMATES OF PARAMETERS OF MODEL II

PARAMETER	DATA SET				
	1 Equitable Male 1965–70	2 Combined Male 1965–70	3 Combined Female 1965–70	4 Combined Male 1955–60	5 Combined Female 1955–60
$B\times10^6$	3.1394	4.8442	9.9028	5.2945	4.4060
c	.....	1.0995	1.0749	1.0988	1.0926
B_2	6.9076	1.9187	0.76881	0.88982	1.90931
c_2	0.94123	0.95853	0.97782	0.97583	0.95568
$100R^2$	96.3%	99.0%	87.9%	98.8%	87.4%

TABLE 5

COMPARISON OF $100R^2$ VALUES

DATA SET	MODEL		
	I	II	III
1. Equitable male, 1965–70	95.6%	96.3%	96.4%
2. Combined male, 1965–70	98.4	99.0	99.1
3. Combined female, 1965–70	87.8	87.9	87.5
4. Combined male, 1955–60	98.4	98.8	98.9
5. Combined female, 1955–60	84.2	87.4	86.2

may be argued that the differences are not really substantial. A true picture of the situation can be obtained by comparing the residuals, which are the differences between the estimated and the observed mortality rates. It was found that Model III seemed to exhibit the most random pattern in the residuals. It was also found that the pattern was more random in the case of the Equitable male data and less random in the case of the combined male (1965–70) data. Since these crude rates apply over the same period of study, it is useful to compare the crude and estimated mortality rates over these two data sets.

Tables 6 and 7 exhibit the estimated-to-crude ratios of the Model III

TABLE 6

RATIO OF ESTIMATED TO CRUDE MORTALITY RATES FOR MODEL III AND EQUITABLE MALE DATA

t	AGE AT ISSUE									
	22	27	32	37	42	47	52	57	62	67
0	.....	.....	0.811	1.682	1.294	0.976	0.791	1.002	1.248	0.913
1	.....	.....	1.178	0.953	1.186	1.053	1.074	0.939	1.664	0.205
2	.....	.....	0.890	0.866	0.736	1.304	1.051	1.234	0.600	1.256
3	.....	0.914	1.289	1.390	1.115	0.818	1.077	0.909	0.827	1.032
4	.....	1.017	1.203	0.770	0.970	1.016	1.217	1.085	1.611	0.777
5	.....	1.140	0.890	1.137	1.014	0.948	0.950	1.056	0.709	1.709
6	.....	0.438	1.264	1.074	1.128	1.231	1.100	1.017	0.793	0.690
7	.....	0.851	0.881	0.987	1.006	0.864	1.016	1.036	1.180	1.806
8	0.721	1.044	0.972	0.992	0.754	1.247	1.332	0.888	1.765	1.221
9	0.873	1.309	1.107	1.047	1.041	1.239	0.982	0.867	1.854	0.605
10	0.913	0.891	0.877	0.925	1.073	1.188	1.010	0.984	1.524	1.021
11	1.547	1.323	0.828	0.923	1.025	1.005	0.977	1.144	1.387	1.317
12	1.806	1.119	1.091	1.035	1.035	0.934	1.076	1.261	1.032	0.539
13	1.039	1.090	1.202	0.986	0.934	1.099	0.877	0.548	0.772	1.005
14	1.171	1.043	1.021	1.073	1.065	1.219	1.110	1.321	1.156	0.939

TABLE 7

RATIO OF ESTIMATED TO CRUDE MORTALITY RATES FOR MODEL III AND COMBINED MALE EXPERIENCE, 1965–70

t	AGE AT ISSUE									
	22	27	32	37	42	47	52	57	62	67
0	.....	.....	1.004	1.184	1.108	1.026	1.117	1.038	0.822	0.815
1	.....	.....	0.793	1.073	0.937	1.068	0.938	1.055	1.464	1.537
2	.....	.....	0.896	1.071	0.872	0.946	1.109	0.845	1.100	1.177
3	.....	1.137	1.028	0.976	0.869	0.868	0.957	0.995	0.808	0.836
4	.....	1.074	1.097	1.024	1.056	0.995	1.027	1.005	1.265	0.735
5	.....	1.124	1.289	0.996	0.976	0.878	0.927	1.080	1.213	1.201
6	.....	0.989	1.104	1.092	0.940	1.082	1.128	1.207	0.969	0.900
7	.....	1.116	1.105	1.017	0.947	0.993	0.914	1.190	1.196	1.370
8	1.012	1.134	1.068	1.018	0.946	1.015	1.073	1.173	1.339	1.310
9	1.081	1.207	1.092	0.995	0.958	0.978	0.959	1.038	1.062	1.088
10	1.017	1.064	1.014	0.991	0.969	1.000	0.980	1.144	1.030	1.174
11	1.002	1.160	0.988	0.984	1.003	0.989	0.899	1.160	0.991	1.196
12	1.135	1.187	1.020	0.978	1.001	0.907	0.951	1.072	1.202	1.282
13	1.003	1.065	1.072	0.937	0.918	0.920	0.941	0.964	1.251	1.079
14	1.072	1.078	1.030	0.982	0.898	0.892	0.910	1.082	1.057	1.796

mortality rates for these two data sets. The estimated mortality rates were calculated by using the formula in Theorem 3. A ratio in excess of 1 implies overestimation, whereas a ratio less than 1 implies underestimation. Upon examination of Tables 6 and 7, several trends are evident:

1. In the case of the Equitable data, there is no systematic underestimation or overestimation as we increase t for fixed starting ages or as we increase the starting ages for fixed t. The ratios deviate from 1 both positively and negatively in a random fashion.
2. For the combined male experience, there appears to be more overestimation in the lower starting ages (22–32) and more underestimation for starting ages in the range 37–52 when $t \geq 7$. Neither trend, however, appears to be overly significant.
3. The deviation from 1 in the ratios for the combined male experience seems to be less than for the Equitable experience. This is an expected result, since $100R^2 = 99.1$ percent for the former and 96.4 percent for the latter.

It is also important to point out that Model III, the Vanderhoof model, was based on a theoretical argument. Model II was presented as an alternative to Model III, which has the same properties in that the limiting mortality law is that of Gompertz (see Fig. 9). Model I is an alternative simple model that fits surprisingly well. The authors believe that a model that is to be adopted should have some theoretical justification as well as empirical validation. The Vanderhoof model seems to satisfy these two requirements. It seems to perform better for male data than for female data.

XIII. FIT OF THE ULTIMATE MORTALITY DATA

Using the ultimate crude mortality rates available for the five data sets discussed in Section VIII, we fit Gompertz's law using the method of weighted least squares. To carry this out, we minimize

$$\Sigma\,(\hat{Y}_x - \alpha_0 - \alpha_1 x)^2\theta_x = \text{WSS}\,,$$

where $\hat{Y}_x$ is given by equation (16). This process yields estimates of α_0 and α_1. To estimate B and c, we solve the two equations in Theorem 4 for B and c in terms of α_0 and α_1. Thus

$$B = \frac{\alpha_1 \exp \alpha_0}{\exp \alpha_0 - 1}\,, \qquad c = \exp \alpha_1\,.$$

Table 8 tabulates the values of B, c, and R^2 for the five data sets under comparison. Also tabulated are the corresponding parameters of the ultimate curve as estimated from the select Model III (previously shown in Table 2).

We do not expect these two ultimate curves to be the same, because the ultimate data are based on medical and nonmedical issues whereas the estimated ultimate curve from the select data is based only on medical issues. Table 9 shows the ratio of the ultimate force of mortality based on the Gompertz crude mortality rates to the ultimate force of mortality estimated from the select curve. The Equitable data on males

TABLE 8

PARAMETERS IN GOMPERTZ ULTIMATE MORTALITY LAW

PARAMETER	DATA SET				
	1 Equitable Male 1965–70	2 Combined Male 1965–70	3 Combined Female 1965–70	4 Combined Male 1955–60	5 Combined Female 1955–60
	Ultimate Parameters Estimated Using Crude Ultimate Mortality Rates				
$B \times 10^5$	5.1507	6.3129	3.1961	7.8473	2.2221
c	.....	1.0959	1.0994	1.0932	1.106
$100R^2$	99.6%	99.8%	95.4%	99.4%	96.7%
	Ultimate Parameters Estimated from Select Model III				
$B \times 10^5$	3.4922	5.2902	10.9861	6.0969	5.0809
c	.....	1.0966	1.0716	1.0953	1.0886

TABLE 9

RATIO OF ULTIMATE FORCE OF MORTALITY DERIVED FROM CRUDE ULTIMATE RATES TO ULTIMATE FORCE OF MORTALITY DERIVED FROM SELECT RATES

ATTAINED AGE x	DATA SET			
	2 Combined Male 1965–70	3 Combined Female 1965–70	4 Combined Male 1955–60	5 Combined Female 1955–60
30	1.171	0.617	1.215	0.704
40	1.163	0.810	1.192	0.825
50	1.156	1.047	1.169	0.966
60	1.148	1.353	1.147	1.132
70	1.141	1.747	1.125	1.327

were not used because the ultimate curve was based on male and female experience combined, and it was not possible to separate the corresponding data.

As can be seen from Table 9, these ratios are always greater than 1 for male mortality. However, for female mortality, the ratios are less than 1 for lower attained ages, rising to a level greater than 1 for higher attained ages. For male data the ratios decrease with age; the opposite is true for female data. We believe that the ratios are consistent with the underlying data, since female mortality in both select and ultimate periods exhibits variations from the expected pattern of mortality increasing with age and duration.

XIV. CONCLUSION AND DISCUSSION

In this paper we have presented theoretical arguments in favor of Gompertz's law as the first approximation of a more complete law of mortality and have then developed from logical and biological arguments the Vanderhoof model of select mortality.

This model was tested by using several bodies of data, and comparisons were made of the fit of this model with that of two other similar models. While all models had high R^2, only our preferred model seemed to have a random distribution of residuals. This model also seems to provide the most useful information about the various bodies of data. The value of s, which relates to the rapidity of wearing off of selection, seems stable over the bodies of data considered. Values of this variable for other data would be interesting. The parameter c represents the ultimate Gompertz curve implied by the select data. The values of c for males are very close to the values found by fitting the available ultimate data for men. The values of B for men seem consistent with the knowledge that the select and ultimate data sets are not homogeneous. Tests of the ultimate data set of medically issued risks would be interesting. Tests of the forms for substandard lives would be interesting, and the implication of the value of s about the rate of change from standard to substandard might be informative. The model's implied ultimate values for women were not satisfactory.

The acid test of this model should be a comparison of the fits with the actual graduations produced by the Committees on Mortality and Morbidity of the Society of Actuaries; a comparison of R^2 values is shown in the accompanying table. As would be expected, the Society of Actuaries' graduation has higher values of R^2. However, the Society of Actuaries' graduation uses more parameters and provides for smoothness only between successive durations for the same issue age. There is no

VALUES OF $100R^2$

	Data Set			
	Data Set 2 Combined Male 1965–70	Data Set 3 Combined Female 1965–70	Data Set 4 Combined Male 1955–60	Data Set 5 Combined Female 1955–60
Society of Actuaries	99.4%	88.7%	99.6%	89.3%
Model III (Vanderhoof)	99.1	87.5	98.9	86.2

inherent smoothness between attained ages. Our preferred model has perfect smoothness between ages and durations. The crude data have an R^2 of 100. To achieve smoothness by duration for the 1965–70 male data, the Society of Actuaries' graduation gives up 0.60 percent of the R^2. To achieve the additional smoothness by age, our model requires an additional loss of only 0.30 percent of the R^2. This argument does not apply to the 1955–60 data but does apply much more forcefully to the female sets. Our preferred model provides an additional dimension of smoothness as compared with traditional graduation techniques, with very little loss of fit to the underlying data.

The value of this formula to the practical actuary should be obvious:

1. A practicing actuary may wish to work with a mortality table that has the same ultimate mortality rates as the most recent Society data, but where the relationship between the ultimate rates and the first-year select rates is that of his own company. Alternatively, he might wish to accept his own company's data for the ultimate rates but use Society data to implant either first-year select rates or the relationship between select and ultimate rates. The formula makes the preparation of such "mosaic" tables simple.
2. Safety margins can be introduced easily.
3. The common procedure of using a constant percentage coefficient for modifications in a table is justified.
4. The effects of withdrawal rates on mortality probably can be observed through the values of s and c where a low and high value, respectively, imply mortality affected by high lapses.
5. Regression analysis and least-squares techniques are very easy to apply to company data.

A possible extension of the Vanderhoof model to take into account irregularities in mortality with attained age is the final algorithm of the approach:

$$\mu_{[x]+t} = K^{f(x+t)} B c^{x+t} (B_3 c_3{}^{x+t})^{s^t}, \qquad \text{(FA)}$$

where $f(x + t)$ is some numerically determined function of attained age from outside data. The addition of this term would, it is hoped, take into account such irregularities as the dip in mortality in the late twenties for males and the development of a local maximum or point of inflection in the female mortality curve, reflecting, in the former case, accident rates for males and, in the latter, fertility rates for females.

APPENDIX I

GEOMETRIC AND ARITHMETIC AVERAGES

In this appendix we prove that equation (1) is approximately equivalent to equation (2). Consider equation (2):

$$\begin{aligned}\mu_{[x]+t} &= (B_s c_s^{x+t})^{s^t}(B_u c_u^{x+t})^{1-s^t} \\ &= f_1^{s^t} f_2^{1-s^t} \\ &= f_2\left(1 + \frac{f_1}{f_2} - 1\right)^{s^t} \\ &= f_2\left[1 + s^t\left(\frac{f_1}{f_2} - 1\right) + \frac{s^t(s^t - 1)}{2}\left(\frac{f_1}{f_2} - 1\right)^2\right] + \cdots .\end{aligned}$$

Now

$$\frac{f_1}{f_2} = \frac{\text{Select mortality rate}}{\text{Ultimate mortality rate}} < 1 .$$

Thus

$$\begin{aligned}\mu_{[x]+t} &\doteq f_2\left[1 + s^t\left(\frac{f_1}{f_2} - 1\right)\right] \\ &= s^t f_1 + (1 - s^t) f_2 ,\end{aligned}$$

or

$$\mu_{[x]+t} = s^t(B_s c_s^{x+t}) + (B_u c_u^{x+t})(1 - s^t) ,$$

which is equation (1).

APPENDIX II

RELATIONSHIP BETWEEN MORTALITY RATES AND THE FORCE OF MORTALITY

In Section VII we stated four theorems concerning the relationship between $q_{[x-t]+t}$ and $\mu_{[x-t]+t}$. In this appendix, we prove these results.

Proof of Theorem 1. The relationship between the force of mortality and the mortality rate is given by Jordan [11] as

$$-\ln (1 - q_{[x-t]+t}) = \int_0^1 \mu_{[x-t]+t+z} dz .$$

From equation (6) we have

$$\operatorname{colog}(1 - q_{[x-t]+t}) = \int_0^1 B_1 r^{t+z} c_1^{x+z} dz$$

$$= B_1 r^t c_1^x \int_0^1 (rc_1)^z dz .$$

Thus

$$\operatorname{colog}(1 - q_{[x-t]+t}) = \frac{B_1 r^t c_1^x (rc_1 - 1)}{\ln rc_1} .$$

If we take natural logarithms of both sides of the above equation, equation (9) follows, which was to be proved.

Proof of Theorems 2 and 3. To prove Theorems 2 and 3, we use the approximate result

$$\operatorname{colog}(1 - q_{[x-t]+t}) = \mu_{[x-t]+t+0.5}$$

as given by Jordan. From equations (7) and (8), we obtain, for Models II and III, respectively,

$$\operatorname{colog}(1 - q_{[x-t]+t}) = Bc^{x+0.5}(B_2 c_2^{x+0.5})^{1/(t+1.5)} ,$$

$$\operatorname{colog}(1 - q_{[x-t]+t}) = Bc^{x+0.5}(B_3 c_3^{x+0.5})^{s^{t+0.5}} .$$

If we take logarithms of both sides of the above two equations, we obtain equations (10) and (11), respectively.

Proof of Theorem 4. Theorem 4 is proved by Jordan [11, chap. 1]. An alternate proof is to consider Theorem 1 in the specific case where $r = 1$, $c_1 = c$, and $B_1 = B$. Equation (9) reduces to equation (12).

APPENDIX III

VARIANCE OF THE COLOG OF THE SELECT SURVIVAL RATE

The justification for weighted least squares is equation (14) for select rates. In this section, we prove this result.

THEOREM 5. *Let* $\hat{Y}_{xt}$ *and* $\hat{Y}_x$ *be defined by*

$$\hat{Y}_{xt} = \ln[\operatorname{colog}(1 - \hat{q}_{[x-t]+t})] ,$$

$$\hat{Y}_x = \ln[\operatorname{colog}(1 - \hat{q}_x)] ,$$

where

$$\hat{q}_x = \theta_x / E_x , \qquad \hat{q}_{[x-t]+t} = \theta_{xt} / E_{xt} ;$$

θ_x *and* E_x *are, respectively, the number of deaths and the number of exposed at attained age* x *for ultimate experience, and* θ_{xt} *and* E_{xt} *are*

the number of deaths and the number of exposed at attained age x and duration t for select experience. Then

$$V[\hat{Y}_{xt}] = 1/\theta_{xt}\,, \qquad V[\hat{Y}_x] = 1/\theta_x\,.$$

Proof of Theorem 5. Following Rao [20], we have

$$V[f(X)] \doteq [f'(\mu)]^2 V(X)\,,$$

where X is a random variable with mean μ. Thus

$$V(\hat{Y}_{xt}) \doteq \left[\frac{1}{\text{colog}\,(1-q)}\,\frac{1}{(1-q)}\right]^2 V(\hat{q})\,,$$

where $\hat{q} = \hat{q}_{[x-t]+t}$. If we treat $\hat{q}E_{xt} = \theta_{xt}$ as a binomial variate, we obtain

$$V(\hat{q}) = q(1-q)/E_{xt}\,,$$

where $q = E(\hat{q})$ is the true mortality rate. Hence

$$V(\hat{Y}_{xt}) = \frac{q(1-q)}{E_{xt}(1-q)^2[\text{colog}\,(1-q)]^2}\,.$$

Making use of the approximations

$$\text{colog}\,(1-q) \doteq q\,, \qquad qE_{xt} \doteq \theta_{xt}\,, \qquad 1-q \doteq 1\,,$$

we have

$$V[\hat{Y}_{xt}] \doteq \frac{1}{\theta_{xt}(1-q)} = \frac{1}{\theta_{xt}}\,.$$

The second equation follows similarly.

APPENDIX IV

METHOD OF WEIGHTED LINEAR LEAST SQUARES

The method of weighted least squares involves minimizing equation (15). We can rewrite this equation as

$$\text{WSS} = \sum_{i=1}^{n} W_i(Y_i - \hat{Y}_i)^2\,, \tag{17}$$

where the W_i's are the weights, or in this case, the number of deaths in attained-age–duration class i; $\hat{Y}_i$ is given by equation (13) for the same attained-age–duration class; and $Y_i = \ln[\text{colog}\,(1-q_i)]$. Here q_i is the true mortality rate in attained-age–duration class i. Equation (15) can be further rewritten as

$$\text{WSS} = \sum_{i=1}^{n} (Y_i' - \hat{Y}_i')^2\,,$$

where

$$Y_i' = W_i^{1/2} Y_i , \qquad \hat{Y}_i' = W_i^{1/2} \hat{Y}_i .$$

For Models I and II, and for Model III in the case where s is a known constant, Y_i' can be expressed as

$$Y_i' = \alpha_0 Z_{0i} + \alpha_1 Z_{1i} + \alpha_2 Z_{2i} + \alpha_3 Z_{3i} .$$

Consequently, to estimate α_0, α_1, α_2, and α_3, linear least squares can be used. The steps are as follows:

1. Transform $\hat{q}_i$ to $\hat{Y}_i = \ln [\text{colog } (1 - \hat{q}_i)]$.
2. Transform $\hat{Y}_i$ to $\hat{Y}_i' = W_i^{1/2} \hat{Y}_i$.
3. Determine Z_{0i}, Z_{1i}, Z_{2i}, and Z_{3i} for the model of interest according to equations (9), (10), or (11) for Models I, II, and III, respectively. For example, for Model II,

$$Z_{0i} = W_i^{1/2} , \qquad Z_{1i} = W_i^{1/2}(x + 0.5) ,$$

$$Z_{2i} = W_i^{1/2}(x + 0.5)/(t + 0.5) , \qquad Z_{3i} = W_i^{1/2}/(t + 0.5) .$$

4. Perform linear least squares through the origin, using $\hat{Y}_i'$ as the dependent variable and Z_{0i}, Z_{1i}, Z_{2i}, and Z_{3i} as the independent variables.

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DISCUSSION OF PRECEDING PAPER

WILBUR M. BOLTON:

The authors are to be congratulated on their attempt to formulate an ultimate mathematical model to represent the varieties of mortality rates in different populations. This discussion will record the results of an attempt to fit a Makeham model to part of the Commissioners 1980 Standard Ordinary Mortality Tables, and to the unloaded basic tables from which these tables were developed by the Special Committee to Recommend New Mortality Tables for Valuation.

The first step was to solve for a set of values of μ_x by Jordan's formula (1.21), that is,

$$\mu_x \doteq \frac{8(l_{x-1} - l_{x+1}) - (l_{x-2} - l_{x+2})}{12l_x}.$$

Since the basic data from which these tables were constructed were divided into quinquennial attained-age groups, it seemed appropriate to use only data at the assumed central attained ages of these groups. In this manner, interpolated rates are retained at the pivotal ages, and the effect of the choice of graduating function actually used would be minimized.

Also, since the hypothesis was that a geometric curve could be passed through the data points, it seemed appropriate to employ as a measure of fit not only the customary deviation between the "observed" values and graduated values but also a measure of "relative" fit:

$$\text{Relative deviation} = \frac{\text{Observed } \mu_x - \text{ Graduated } \mu_x}{\text{Observed } \mu_x}.$$

Using this measure of "relative" fit, a deviation of 1 death per 1,000 at an age where the observed value is 40 deaths per 1,000 would have no greater significance than a deviation of 0.1 death per 1,000 at an age where the observed value is 4 deaths per 1,000. Such a test seems intuitively appropriate where the underlying function is expected to follow an exponential form.

For the range of 50 attained ages illustrated, a best fit was determined for the value of A such that, in the equation

$$\ln (\mu_x - A) = \ln B + x \ln c,$$

the coefficient of determination,

$$R^2 = \left[\Sigma x_i \ln(\mu_x - A) - \frac{\Sigma x_i}{n} \Sigma \ln(\mu_x - A)\right]^2 \times \left\{\left[\Sigma x_i^2 - \frac{(\Sigma x_i)^2}{n}\right]\left[\Sigma [\ln(\mu_x - A)]^2 - \frac{[\Sigma \ln(\mu_x - A)]^2}{n}\right]\right\}^{-1},$$

would be maximized.

These graduations were done by an exponential-curve-fitting program on the HP-25. Table 1 shows the result for a limited range of ages (attained ages 45–94) for male lives, and Table 2 shows corresponding results for female lives. Review of these results showed that the fit for female lives was less than half as good as the fit for male lives for this range of ages. The average relative deviation for females was 4.06 percent, compared with 1.76 percent for males.

In an attempt to determine whether the problem with obtaining a fit was an artifact of the type of loading formula used in developing the loaded table, a similar graduation was applied to the underlying basic tables. (See Exhibit 3 in the report of the Special Committee.) The results are shown in Tables 3 and 4 for males and females, respectively.

TABLE 1

MAKEHAM GRADUATION OF COMMISSIONERS 1980 STANDARD ORDINARY MALE MORTALITY TABLE, AGES 45–94

Age (1)	$1{,}000q_x$ (2)	Observed $1{,}000\mu_x$ (3)	Makeham $1{,}000\mu_x$ (4)	Observed minus Graduated [(3)−(4)] (5)	Relative Deviation [100(5)/(3)] (6)
47.....	5.32	5.129	5.070	0.059	1.15%
52.....	7.96	7.646	7.766	−0.120	−1.57
57.....	12.49	12.038	12.044	−0.006	−0.05
62.....	19.19	18.501	18.835	−0.334	−1.81
67.....	30.44	29.550	29.615	−0.065	−0.22
72.....	47.65	46.437	46.724	−0.287	−0.62
77.....	77.12	76.649	73.882	2.767	3.61
82.....	117.25	118.967	116.988	1.979	1.66
87.....	179.55	189.662	185.409	4.253	2.24
92.....	253.45	280.967	294.010	−13.043	−4.64
Total	.....	785.546	790.343	−4.797	−0.25%

Makeham constants:

$A = 0.00048$; $B \times 10^5 = 5.96645+$; $c = 1.0968+$; $100R^2 = 99.971$;
$\Sigma|\text{relative deviations}| = 17.57\%$; average relative deviation = 1.76%;
number of sign changes = 3.

TABLE 2

MAKEHAM GRADUATION OF COMMISSIONERS 1980 STANDARD ORDINARY FEMALE MORTALITY TABLE, AGES 45–94

Age (1)	$1{,}000q_x$ (2)	Observed $1{,}000\mu_x$ (3)	Makeham $1{,}000\mu_x$ (4)	Observed minus Graduated [(3)−(4)] (5)	Relative Deviation [100(5)/(3)] (6)
47.....	4.05	3.929	4.024	−0.095	−2.42%
52.....	5.70	5.512	5.231	0.281	5.10
57.....	8.03	7.834	7.330	0.504	6.43
62.....	10.96	10.567	10.982	−0.415	−3.93
67.....	17.43	16.856	17.332	−0.476	−2.82
72.....	26.87	25.787	28.374	−2.587	−10.03
77.....	48.04	46.512	47.578	−1.066	−2.29
82.....	82.40	80.980	80.976	0.004	0.00
87.....	143.32	146.358	139.055	7.303	4.99
92.....	228.81	246.489	240.059	6.430	2.61
Total	.....	590.824	580.941	9.883	−2.36%

Makeham constants:

$A = 0.00239$; $B \times 10^5 = 0.89985+$; $c = 1.1170+$; $100R^2 = 99.840$;
$\Sigma|\text{relative deviations}| = 40.62\%$; average relative deviation = 4.06%;
number of sign changes = 3.

Review of these tables shows that the male basic table for the age range 40–89 could be graduated fairly well using Makeham, with an average relative deviation of 1.15 percent and a nice alternation of the signs of deviation of graduated values from observed values. Adding data for younger and/or older ages causes substantial deterioration in the fit of graduated to observed rates.

However, the female basic table for the same age range develops Makehamized values that underpredict deaths consistently at attained ages 45–59, overpredict deaths substantially at attained ages 60–79, and underpredict again at ages 80 and over. The average relative deviation exceeds 7 percent, compared with 1.15 percent for male lives. Again, adding data for younger ages makes the fit poorer.

The same features of wide underprediction in the fifties and overprediction in the 60–79 age range for females result whether the chosen pivotal values are the ten from the age range 42–87 or the ten from 47–92. It would seem that proponents of the mathematical-law approach to graduation need an explanation or a mechanism to account for this hump and dip for female attained ages 45–79 in comparison with the "best fit" Makeham curve.

TABLE 3

MAKEHAM GRADUATION OF 1970–75 BASIC MALE TABLE, AGES 40–89
(FIRST 5 POLICY YEARS EXCLUDED)

Age (1)	$1{,}000q_x$ (2)	Observed $1{,}000\mu_x$ (3)	Makeham $1{,}000\mu_x$ (4)	Observed minus Graduated [(3)−(4)] (5)	Relative Deviation [100(5)/(3)] (6)
42.....	2.36	2.253	2.244	0.009	0.40%
47.....	3.84	3.673	3.652	0.021	0.57
52.....	6.08	5.798	5.922	−0.124	−2.14
57.....	10.06	9.644	9.581	0.063	0.65
62.....	15.95	15.313	15.476	−0.163	−1.06
67.....	26.01	25.147	24.976	0.171	0.68
72.....	41.38	40.121	40.285	−0.164	−0.41
77.....	68.00	67.221	64.955	2.266	3.37
82.....	103.61	104.354	104.711	−0.357	−0.34
87.....	158.63	165.743	168.776	−3.033	−1.83
Total	.....	439.267	440.578	−1.311	−0.11%

Makeham constants:

$A = -0.00006$; $B \times 10^5 = 4.18532$; $c = 1.1001+$; $100R^2 = 99.988$;
$\Sigma|\text{relative deviations}| = 11.45\%$; average relative deviation = 1.15%;
number of sign changes = 7.

TABLE 4

MAKEHAM GRADUATION OF 1970–75 BASIC FEMALE TABLE, AGES 40–89
(FIRST 5 POLICY YEARS EXCLUDED)

Age (1)	$1{,}000q_x$ (2)	Observed $1{,}000\mu_x$ (3)	Makeham $1{,}000\mu_x$ (4)	Observed minus Graduated [(3)−(4)] (5)	Relative Deviation [100(5)/(3)] (6)
42.....	1.81	1.716	1.798	−0.082	−4.78%
47.....	2.77	2.672	2.471	0.201	7.52
52.....	4.11	3.951	3.614	0.337	8.53
57.....	6.01	5.851	5.551	0.300	5.13
62.....	8.33	7.986	8.836	−0.850	−10.64
67.....	13.88	13.367	14.408	−1.041	−7.79
72.....	21.89	20.874	23.858	−2.984	−14.30
77.....	40.72	39.175	39.885	−0.710	−1.81
82.....	71.11	69.358	67.065	2.293	3.31
87.....	125.07	126.468	113.161	13.307	10.52
Total	.....	291.418	280.647	10.771	−4.31%

Makeham constants:

$A = 0.00083$; $B \times 10^5 = 1.14469$; $c = 1.1114+$; $100R^2 = 99.609$;
$\Sigma|\text{relative deviations}| = 74.33\%$; average relative deviation = 7.43%;
number of sign changes = 3.

A conjecture may be made that this experience will not follow a true Makeham curve because, even though the first five policy years are excluded, it contains some "select" lives in policy years 6–15. It seems to me that this conjecture would be more applicable to male insured lives than to female insured lives in this age range; further, review of Exhibits 5 and 6 of the report of the Special Committee shows that female insured mortality is relatively closer to female population mortality than is the case for the corresponding male experience.

It is interesting that in Tables 2, 4, and 5 of the paper the authors also show a significantly lower value of $100R^2$ for the female experience than for the male experience, indicating a poorer fit of the actual and graduated tables for females.

It also seems peculiar that, in the tables in this discussion, the derived values of A, the constant to allow for random or accidental death in the Makeham formula, are much smaller for males than for females in both the "loaded" recommended valuation table and the basic table. This is contrary to our a priori knowledge that the accidental death rate for males is consistently higher than for females in modern studies.

Perhaps the authors can offer a verbal interpretation for the negative value of A resulting from attempting to Makehamize the 40–89 age range of the 1970–75 Basic Male Table. I don't have one.

My conclusion from the work underlying this discussion is that the Special Committee to Recommend New Mortality Tables for Valuation was eminently correct in not attempting to force either the new basic tables or the new valuation tables into a Makeham mold. Distortion of the results, particularly in regard to females, would have been unacceptable. However, if the authors can hypothesize sex- and age-dependent factors that will account for the major observed twists and turns in adult mortality, future generations of actuaries will be greatly in their debt.

Certainly the authors should be commended for reminding us that the search for a mathematical law of mortality should not be totally abandoned by our profession simply because no one up to now has succeeded in developing one that fits all major features of the data.

K. S. BROWN:

The authors are to be commended for their statement that models of mortality should be based on reasonable biological assumptions and should not be just mathematical functions in which the parameters have no biological interpretation.

However, after a well-organized review of the relationships between

the linear decline of physiological function with age and the exponential increase in mortality rates with age, the authors propose a model that is neither biologically plausible nor mathematically correct.

The authors assume that there exists a biological system of n components and the probability that any component in such a system fails is f. (Although this is not stated, it probably is assumed that f is the probability that a component fails in a one-year period.) Given these assumptions, $q_0 = f^n$ (i.e., components fail independently, and if all components fail between ages 0 and 1, then the individual dies between those ages).

Under what assumptions would f be independent of age? Is it not more reasonable to assume that f would be a function of age, with a greater chance of failure the older the component, or have the authors assumed that failures are the result of random events?

Second, if more than one system becomes involved, why is it assumed that each system has the same number (n) of components, and that each component has the same probability (f) of failure?

Finally, if $q_x = f^{n-x}$ as the authors claim (but see below), then $q_n = 1$, and n is automatically determined to be in excess of 100. What is the evidence for a number of systems, each of which has more than 100 levels of redundancy?

Mathematically, if f is assumed to be the probability of failure of any component in a one-year period, then the probability that any component fails between ages x and $x + 1$ is

$$(1 - f)^x f ,$$

and the probability that any component does not fail before age x (i.e., survives to age x) is

$$\sum_{t=x}^{\infty} (1 - f)^t f = (1 - f)^x = S^*(x) .$$

Now the system fails when all components fail, and hence the probability of survival of the *system* to age x is

$$S(x) = 1 - [1 - (1 - f)^x]^n .$$

Hence

$$q_x = \frac{S(x) - S(x + 1)}{S(x)}$$

$$= \frac{[1 - (1 - f)^{x+1}]^n - [1 - (1 - f)^x]^n}{1 - [1 - (1 - f)^x]^n} \neq f^{n-x} .$$

The authors apparently have confused age x with the number of components (x) that have failed. A simple model for which the expected number of component failures by age x equals x has

$$n(1 - S^*(x)) = x\,,$$

or

$$S^*(x) = 1 - x/n\,,$$

and, thus,

$$q_x^* = \frac{S^*(x) - S^*(x+1)}{S^*(x)} = \frac{1}{n-x}\,,$$

which is not constant (i.e., f) as the authors claim.

There are a number of other models that have been postulated since Sacher-Trucco and Strehler-Mildvan. Some of these (e.g., Forbes-Sprott [3]) postulate random "hit" mechanisms and do not directly assume the linear decline in physiological function. Another model (Brown-Forbes [1, 2]) does assume the linear decline and has been fitted successfully to mortality data for the major causes of death. The successful fitting of mortality data is a more essential criterion than merely demonstrating that the mortality curves produced follow Gompertz's law.

In fitting by weighted least squares, the authors must obtain approximations for the variances of the transformed rates that involve assumptions that are not always consistent (e.g., $1 - q \simeq 1$, but $\operatorname{colog}(1 - q) \simeq q$). Have they checked the fits that would be obtained by directly estimating the parameters using maximum-likelihood techniques? With current high-speed computers, three- or four-parameter models can often be fitted quickly even when first or second derivatives of the likelihood function cannot be found easily.

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MICHAEL COHEN:

The authors are to be congratulated on presenting a very thought-provoking, well-researched (with one exception mentioned below), and well-written paper. If its only effect is to wean actuaries from fidelity to summation-formula methods of graduation, it will have served its purpose, but it should have a much more profound influence on actuarial thinking.

However, I was surprised to note an absence of reference to any work undertaken in the United Kingdom other than Gompertz's pioneering study. This is particularly surprising in view of British actuaries' aban-

donment of summation methods (which, significantly, the second-named author identifies with "graduation" in the Introduction) in favor of graduation by mathematical formula, and their attempts, stretching back fifty years and more, to find explanations for terms in the Gompertz and Makeham formulas, as well as adaptations of the Pearson family of curves ("logistics") to the graduation of insurance, annuity, and population data.

Two papers particularly worthy of mention are "On Some Experiments in the Graduation of Mortality Statistics" (*JIA*, LXIII, 12) by Perks, submitted to the Institute of Actuaries in 1931, and "An Exploration into Patterns of Mortality" (*JIA*, XCV, 243) by Redington, submitted in 1969.

Redington's paper, while far from "scientific" (a fact he admits himself), has an interesting explanation of why mortality falls off at older ages. He recasts the familiar Gompertz relationship, $\mu_x = Bc^x$, into $\mu_x = c^{x-z}$, where $\mu_z = c^0 = 1$, z being the age at which the annual force of mortality is unity. He then considers that the population consists of a mixture of "pure strains" each having its own values of c and z. Plotting μ_x against x on semilog paper gives a straight line for such a "pure strain," but obviously produces a curve bending toward the x-axis for mixed z's and c's. In other words, the survivors at older ages are there not just because of the stochastic process, but because they have a greater resistance to death, thereby lightening the observed mortality.

In his paper Redington says, "If posterity passes this way, it will drive a paved road where I have used a footpath." Tenenbein and Vanderhoof undoubtedly have used that paved road. It is a pity that they did not spot the footpath on the other side of the ocean, not to mention the variety of pathways that have been used by British actuaries over the past half-century and more.

STUART KLUGMAN:

Very little has been written on the construction of select mortality tables. The authors have provided a clever yet simple approach to this problem, and they are to be commended for attacking the problem from both a physiological and a statistical viewpoint. My comments relate only to their approach for constructing the ultimate portion of the table. They cover four areas: the authors' reasons for rejecting other graduation methods, the sacrific of accuracy for simplicity, the interpretation of R^2, and the use of the terms θ_{xt} as weights.

In Section I, six criticisms of commonly used graduation techniques are listed. While some are valid, I must disagree with items 2, 3, and 6.

Several methods use an understanding of the mortality process. Our belief that q_x is a smooth function is incorporated into all these methods. The Bayesian approach allows our knowledge of mortality rates to be directly incorporated. Items 3 and 6 refer to the observation of deviations. A residual analysis can accompany any graduation; however, it may be more difficult to use the results to alter a biologically based model than to adjust a prior opinion.

A fundamental concern is the balance between the gains from simplification and the loss of accuracy. How much error can we tolerate to receive the benefits of a mathematical model? There is little doubt that Gompertz's law holds for numerous examples for ages 30–70 [2]. Are the errors at other ages insignificant, particularly with respect to financial values? How does one decide? Further research into this area may be very useful.

My third comment relates to the statistical analysis. All of the graduations produce extremely large values of R^2. Even the unsatisfactory Model I produces values above 0.87. As an experiment, I gave crude values of $Y_x = \ln[-\ln(1 - q_x)]$ and weights θ_x for ages 30–70 from the 1965–70 ultimate combined male study to Professor Johannes Ledolter of the University of Iowa's Department of Statistics. Among linear models, he selected $Y_x = \beta_0 + \beta_1 x + \beta_2 x^2$ as providing the best representation. This was done despite the fact that R^2 was equal to 0.99745 for the first-degree model. Two factors led him to select the quadratic function. First, the residuals for the straight-line fit did not appear to be random. They were positive for ages 30–34, negative for 35–49, positive for 56–61, and mixed elsewhere. In all, there were 13 sign changes, significantly fewer than the 20 expected. Second, when fitting the quadratic term, the significance test for β_2 yielded $t = -3.5$ ($p = 0.0013$). Even so, the value of R^2 was raised only to 0.99806. I believe there are two reasons for this. First, the high value of R^2 results from the steep slope of the line dominating the local fluctuations. Second, the weights place little emphasis on ages 30–40, where most of the curvature was observed. The above comments lead me to return to the question of parsimony raised above.

Finally, I must comment on assumption d in Section VIII. When a unit other than lives is used for counting exposures and deaths, $\text{Var}[\hat{Y}] \doteq \beta/n\alpha^2$, where n is the number of lives, α is the average number of dollars per life, and β is the average square of the number of dollars [1]. In the authors' notation, $\theta_{xt} = n_{xt}\alpha_{xt}$ and therefore $\text{Var}[\hat{Y}_{xt}] \doteq \beta_{xt}/\theta_{xt}\alpha_{xt}$. Thus, θ_{xt} is an appropriate weight only if β_{xt}/α_{xt} does not vary significantly over the various age-duration cells considered.

None of the above comments is designed to detract from the authors' contribution to the construction of select mortality tables. I also look forward to their work describing the second-order version of their model.

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ARTHUR LEVENGLICK:

Messrs. Tenenbein and Vanderhoof have made an ingenious attempt to provide a strong theoretical foundation for mortality patterns that continually recur in humans and other life forms. I would like to comment briefly on two areas of their work.

The authors' model relates Gompertz-type mortality patterns to a model based upon death defined in terms of the failure of certain systems (currently not identifiable). Their approach entails a number of strict assumptions.

1. The systems are mutually independent.
2. The systems have identical numbers of critical components.
3. All components in each system have identical probabilities of failure.
4. The failure rate of components is linear with time.

Although it is possible to develop a theory with weaker assumptions, the theory would be more complicated and less closely related to the Gompertz law.

The authors' analysis of select mortality confused me somewhat. In Section IV, the authors assumed that each of the mortality classes delineated at issue was transformed during the select period into each of the other classes. It would appear that during this initial period no "select" mortality class could be transformed into the "ultimate" class, a term apparently reserved to denote those lives selected a long time ago. In Section V, however, the authors defined the select group as those individuals who could pass a medical examination each year. This definition would imply that the terms "select" and "ultimate" have nothing to do with the time elapsed since the initial underwriting process but rather involve the current state of health of the individuals. For example, an individual with high blood pressure who has just been underwritten and issued a (rated) policy would be considered an "ultimate" life rather than a member of a select substandard mortality class. I think that the algorithm presented in the paper is inconsistent with the generally accepted usage of the terms "select" and "ultimate."

COURTLAND C. SMITH, JR.:

Messrs. Tenenbein and Vanderhoof are to be complimented for an imaginative and challenging paper.

Essentially this paper seems to say that since Gompertz's law, $\mu_x = Bc^x$, assumes ongoing degeneration in physiological processes with advance in age, it should be applicable to a group of continuously healthy lives qualifying for new life policies as well as to a group of lives in average health. In life industry parlance, the healthy lives would be zeroth-duration or first-policy-year standard select risks, and the lives in average health would be standard ultimate risks. If the ultimate lives remain ultimate while the select lives tend to become ultimate in increasing proportion with advance in duration, then we can express the mortality of a cohort at duration t as the weighted mean of zeroth-duration select mortality rates and ultimate mortality rates. Finally, if Gompertz's law is truly universal, then it should apply equally well to each duration t.

Symbolically, for attained age x, select duration t, and select proportion s, where $0 < s < 1$, equations (IA) and (1) of Section V may be written

$$\mu_{[x-t]+t} = s^t\mu_{[x]} + (1 - s^t)\mu_x = s^tB_sc_s^x + (1 - s^t)Bc^x ,$$

or

$$\mu_{[x-t]+t} = \mu_x + s^t(\mu_{[x]} - \mu_x) ,$$

where the last term is obviously negative. Here $\mu_{[x-t]+t}$ is a weighted arithmetic mean of the select and ultimate μ's. Since Gompertz's law itself depicts a geometric curve, a weighted geometric mean may constitute an appropriate approximation. Accordingly, Model III, or Vanderhoof's law, is proposed in equations (2) and (8) as

$$\mu_{[x-t]+t} = (\mu_{[x]})^{s^t}(\mu_x)^{1-s^t} = (B_sc_s^x)^{s^t}(Bc^x)^{1-s^t} ,$$

or

$$\mu_{[x-t]+t} = Bc^x\left[\left(\frac{B_s}{B}\right)\left(\frac{c_s}{c}\right)^x\right]^{s^t} = B_4c_4^x ,$$

where $B_4 = B(B_s/B)^{s^t}$ and $c_4 = c(c_s/c)^{s^t}$. Thus, tth-duration mortality also follows a Gompertz curve.

The resulting curves for ages 30 and over give a reasonably good fit to some recent North American select and ultimate tables for males but not to those for females. In the concluding paragraphs, the authors comment wistfully that the inclusion of an additional factor in equation (8) perhaps may be sufficient "to take into account such irregularities as the dip in mortality in the late twenties for males and the development of a local maximum or point of inflection" for females.

It is both interesting and instructive to compare this approach with

that outlined in a recent paper in the *Journal of the Institute of Actuaries*, entitled "The Age Pattern of Mortality," by L. Heligman and J. H. Pollard (CVII, Part I [1980], 49–80). Heligman and Pollard suggest a mathematical expression or "law of mortality" for graduating postwar Australian national mortality data, of the form

$$q_x/p_x = B_1^{(x+J)^M} + B_2 \exp\left[-E(\ln x - \ln F)^2\right] + Bc^x .$$

(See eq. [1] of their paper.)

The first term is intended to represent the exponential drop in mortality during the early years following the trauma of birth as the child "adapts to its new environment and gains immunity from diseases of the outside world." The second term, which is lognormal in form, reflects the "accident hump" for males and the combined accident and maternal-mortality hump for females. The third term, "the well-known Gompertz exponential, reflects the near geometric rise in mortality at the adult ages, and is considered to represent the ageing or deterioration of the body, i.e., senescent mortality."

Together the three components give a surprisingly good fit to recent Australian population data. However, the fit is not completely satisfactory, and two modifications of the basic curve are proposed. One modification improves the fit for Australian males, where the curvature of ln (q_x/p_x) is concave downward or flattened at the older ages. A second modification fits better for females, where the curvature is concave upward at the older ages.

The Heligman/Pollard concluding paragraphs include a conjecture that a more general "law of mortality" might be expressed in the form

$$q_x/p_x = \sum_{i=1}^{n} B_i \exp\left\{-E_i[f_i(x) - F_i]^{G_i}\right\} .$$

Normally the papers in the *JIA* pay only limited attention to long-term selection. Nevertheless, one response to the Tenenbein-Vanderhoof wistful comment may well be to propose a "law" of mortality of the Heligman-Pollard variety, namely,

$$\mu_{[x-t]+t} = B_1^{(x+J)^M} + B_2 \exp\left[-E\,(\ln x - \ln F)^2 + Bc^x + s^t(B_s c_s^x - B_c^x)\right] ,$$

where the last term is negative. Alternatively, if we use Vanderhoof's law, we have

$$\mu_{[x-t]+t} = B_1^{(x+J)^M} + B_2 \exp\left[-E\,(\ln x - \ln F)^2 + B_4 c_4^x\right] ,$$

where B_4 and c_4 are as defined earlier.

All this suggests the following comments and questions to me:

1. Tenenbein and Vanderhoof seem to consider mathematical expressions or "laws" to be completely antithetical to graduation techniques. Why must they be completely antithetical? Can we not graduate statistical data using mathematical curves in the Heligman-Pollard manner?
2. Even if mathematical expressions and graduation techniques should indeed be antithetical in part, cannot the ends of science still be satisfied by either means? Cannot either means serve to describe observed statistical phenomena?
3. At our company we have found the Vanderhoof "law"—Model III—very useful in approximating the intercompany 1965–70 Basic Select and Ultimate Table. On a computer, it is easier to calculate an array of mortality rates from a few parameters than to load all the rates as input. Even so, may it not be misleading to give real credence to the notion that select mortality in duration t is actually a weighted mean of zeroth-duration select mortality and ultimate mortality? Do we really have a combination of zeroth-duration select risks and ultimate risks at each renewal duration, or do we rather have a continuum of risks in various states of health?
4. Under current reentry select and ultimate term policies, many companies attempt to reselect risks at a renewal duration. Can they really do this successfully given the limitations of the underwriting process and the realities of the marketplace?
5. North American reinsurers often experience higher mortality ratios in the first policy year than in years 2–5. My query would be: Is there an adjustment that could be made to the general Heligman-Pollard expression to reflect this antiselective force in reinsurance experience?

PAUL THOMSON:

The authors of this fine paper are to be commended for a valuable addition to actuarial science. With Vanderhoof's law, a whole select and ultimate table above age 30 can be expressed compactly in a formula and a few constants, thus at once saving computer storage space and solving graduation and interpolation problems. Formerly, the chief reason for fitting Gompertz or Makeham curves was to permit the use of uniform seniority tables for joint life functions, but modern computers have obviated this need. In this work the authors have shown convincingly that the Gompertz model does after all essentially represent the underlying trends of mortality rates.

The authors mention the problem of mortality falling away from the Gompertz law at high ages, and they note that addition of only the second-order term in the power series of c^x improves the fit. It occurred to me that since Vanderhoof's law is a compound Gompertz formula with an element dependent on duration, it might be made to represent the falling-away phenomenon with duration t measured from a transitional

age T. Up to age T, ratios of consecutive μ_x's are fairly constant, with the unexplained decline occurring thereafter. To test this application, I used the following derivation, which is similar to the authors' but with slightly different symbols to avoid confusion, starting with their geometrical average formula (2):

$$\mu_x = (B_a c_a^x)^{h^t} (B_b c_b^x)^{1-h^t} ,$$

where B_a and c_a are determined from ages up to T, $t = x - T$, and B_b and c_b are to be found. Also, h replaces the authors' s; although it also lies between 0 and 1, it represents the rapidity of transition between curves in the opposite direction from the select to ultimate process. As in the paper, the above can be expressed as

$$\mu_x = B_b c_b^x (B_c c_c^x)^{h^t} ,$$

where $B_c = B_a/B_b$ and $c_c = c_a/c_b$. The authors' Theorem 4 with linear regression was used to obtain B_a and c_a. For $x \geq T + 1$ the following was used to find B_b and c_b:

$$\ln [\text{colog } (1 - q_x)] = \ln B_b + (x + 0.5) \ln c_b + h^{t+0.5}[\ln (B_a c_a^{x+0.5}) - \ln B_b - (x + 0.5) \ln c_b] ,$$

whence

$$Y_{xt} = \frac{\ln [\text{colog } (1 - q_x)] - h^{t+0.5} \ln (B_a c_a^{x+0.5})}{1 - h^{t+0.5}} = \alpha_0 + (\alpha + 0.5)\alpha_1 ,$$

where $\alpha_0 = \ln B_b$ and $\alpha_t = \ln c_b$. This involves guessing at a value of h, and hence requires only simple linear regression and trying other values of h if necessary, or finding an optimal value as the authors did, having the necessary computing facilities.

The mortality rates I used are those shown in Table 13 of Francisco Bayo's paper "Mortality of the Aged" (*TSA*, Vol. XXIV), and the resulting parameters found are shown in Table 1 of this discussion, with the resulting ratios of rates in Table 2. Age 86 seemed appropriate for T for all cases except for white females, where it is 95, perhaps because of a quirk in the data. Quinquennial ages were used for the range up to T and triennial ages above, except that individual ages were used for white females aged 96–100. Unweighted least squares were used, since numbers of deaths were not available.

The ratios of rates in Table 2 indicate that the fit may be satisfactory in each case, although there is obviously some bending of the original rates both below and above age T. A value of 0.90 for h seemed to give reasonable results, both in terms of R_b^2 and trends of ratios of consecutive

μ's without further trials, except for white males, where 0.94 reproduced the trend better with a small reduction in R_b^2. In testing the continuity of rates around the junction at age T, the white female group shows a sharp drop in ratios of consecutive μ's; however, this is a reflection of the same phenomenon in the original data.

Considering where this application of Vanderhoof's law leads for ages above 101, it can be seen that since h^t approaches zero as t increases, eventually $\mu_x \simeq B_b c_b^x$ at extremely high ages. Whether this will prove realistic can be verified only by using far more data than are yet available;

TABLE 1

ESTIMATES OF PARAMETERS
DATA SET (FROM BAYO'S TABLE 13)

PARAMETER	FEMALE		MALE	
	Nonwhite	White	Nonwhite	White
$B_a \times 10^4$	0.9654	0.1537	3.8964	1.7360
c_a	1.08638	1.11009	1.07234	1.08320
$100R_a^2$	99.7%	99.9%	99.8%	99.98%
T	86	95	86	86
h	0.90	0.90	0.90	0.94
$B_b \times 10^4$	19.327	2.241	221.556	64.839
c_b	1.04798	1.07271	1.02323	1.04148
$100R_b^2$	99.4%	96.7%	94.4%	97.4%
$B_c \times 10^4$	499.508	685.855	175.865	267.740
c_c	1.03664	1.03485	1.04799	1.04006

TABLE 2

RATIO OF ESTIMATED MORTALITY RATES TO DATA MORTALITY RATES

AGE	FEMALE		MALE	
	Nonwhite	White	Nonwhite	White
66	1.042	1.031	1.031	0.999
71	0.973	1.004	0.975	1.001
76	0.968	0.997	0.981	1.009
81	0.984	0.966	0.994	1.006
86	1.034	0.955	1.020	0.993
89	1.006	0.975	1.008	1.006
92	0.987	1.023	0.984	0.993
95	1.001	1.042	0.991	0.989
98	1.001	0.995	1.013	0.992
101	1.004	1.006*	1.001	1.016

* Ratio at age 100.

meanwhile, there may be some utility in using this approach in extending mortality rates of annuitants and others toward assigned tabular closing-out ages.

FRANK A. WECK:

The authors set themselves the task of discovering and then formulating the basic laws that govern mortality. I believe they not only have succeeded in offering new and useful mathematical expressions for the mortality curve but also have developed a theory of aging that opens new avenues for research into the nature of mortality itself.

To explain the nature of the mortality curve, the authors have linked two hypotheses: (*a*) the linearity of loss of function of biological systems with advancing age and (*b*) the redundancy of biological systems. Physiological and biological studies lend support to each of these hypotheses, at least as a first approximation. It was brilliant insight on the part of the authors to see the possibility of combining these two hypotheses to derive mortality laws.

In the case of utlimate mortality, especially for the middle and older ages, the arguments in favor of the authors' approach seem to me to be quite persuasive. At the infantile ages, the complex of forces probably precludes quantification into a simple enveloping mathematical formula. During childhood and at the younger adult ages, external forces (and childbearing, in the case of females) have significant impacts on mortality. It probably is best, as the authors propose, to treat the effects of these impacts by means of modifications superimposed on the formulas applicable to the older ages.

In the case of select mortality, it seems to me that several questions remain to be resolved. Some of these questions flow from the purpose to be served by a select mortality table, while others involve the theory relating select to ultimate mortality.

For underwriting purposes, a cohort of lives (policies) should be viewed as select to the end of the mortality table. Whether or not such select mortality merges with some ultimate table is irrelevant. The degree of selection, that is, the difference between select and ultimate mortality, usually is determined from experience tabulated by age at entry and duration. Some allowance may be made for changes in underwriting standards during the period of experience. Adjustments are made less commonly for changes in mortality levels by calendar year of experience. Rarely are mortality levels projected into the future to reflect current trends or the impact of changing life-styles and medical progress. Yet allowance for the effect of each of these factors is important if past experience is to be used as a guide for underwriting.

Some of the apparent anomalies and unexplained differences between select and ultimate mortality, which have been evidenced in various mortality studies, may be the consequence of giving too little weight to changes in underwriting processes and standards and to variations by calendar year of experience. The authors' approach commends itself as a natural point of departure from which to make allowance for the latter. It would be interesting to pursue such an investigation, if suitable data were to become available. Also of interest would be an indication by the authors of how their formulas could be used to recognize various assumptions regarding future mortality.

It has always been difficult to develop a satisfactory transition from select to ultimate mortality. This has been due not only to the lack of suitable experience data but also to the absence of any generally accepted theoretical concept. Here again, the authors have filled the breach by proposing an appealing theoretical foundation for relating select to ultimate mortality.

The authors consider that at time of issue all lives can be grouped into a number of distinguishable classes with expected mortality levels ranging, in order, from low to high mortality. Following this approach, select lives at issue are considered to be those that are included in the classes with expected mortality at or below the maximum for standard insurance. Each year after issue there is assumed to be a "transformation" of classes. At the beginning of the second year (and at the beginning of each subsequent year) the membership of each class, which now includes nonstandard as well as standard lives, is assumed to include representatives from all the classes existing at the beginning of the previous year. As a consequence of this assumption, ultimate mortality will contain representatives from all classes, and any initial distribution of select classes will be "transformed gradually into the distribution of the ultimate or unselected group."

I think it is difficult to know how far it is stretching facts to assume that members from each of the select classes can end up in each of the ultimate classes. There must be some classes eligible for standard insurance whose members never can have expected mortality as low as the expected mortality of the best classes. An example might be family history; another might be history of radiation exposure. I doubt whether data exist that could be used to provide quantitative tests of this aspect of the authors' theory. Perhaps the underwriters could evaluate the significance of this matter, but I consider it a theoretical caveat of only minor significance. What the authors have done, I think, is to strike a good balance between theoretical and practical considerations, which leads to some useful formulas.

I have some other minor reservations in regard to the formulas that the authors have developed as models for select mortality. This is not to say that their models are unreasonable; rather, their basic assumptions provide a point of departure from which a multitude of formulas could be constructed. The tests of their three models do not seem to me to be conclusive, if only because the raw data are flawed by the lack of adjustment for calendar year of experience. Even if the raw data are deemed to provide a suitable test, the variation in the magnitude of the several constants in the models leaves one in doubt as to which model, if any, deserves the title of "law."

Moreover, there is no reason to require that select mortality merge with ultimate mortality, except for practical convenience. While Gompertz's law can be defended as a logical approximation to the ultimate mortality curve, it may well be that a model for select mortality that embraces all durations would provide an even better expression for what is termed ultimate mortality. One merit of the authors' models is that they can be applied to reach the limiting age of the mortality table.

No mathematical formula can be more than an imperfect expression of a mortality "law." Graduation can, I believe, improve adherence to the underlying mortality law. By smoothing the raw data, graduation bridges the categories by age, duration, and so on, and thereby minimizes fluctuations due to small numbers. Moreover, graduation does not merely smooth; it can reflect broad trends as well.

While the authors rightly disavow graduation as the best means of arriving at a meaningful mortality curve, their formulas do produce graduated results. In fact, they point out that their preferred model "provides an additional dimension of smoothness as compared with traditional graduation techniques." I would suggest that one test of the authors' formulas would be to graduate the departures of the raw mortality rates from those produced by their formulas. Any broad and consistent divergence would seem to call for further investigation.

Much of the paper is devoted to computational models and procedures. Formulas are presented that deal with force of mortality, mortality rates, and q_x. Some of the computational complications could be avoided by the device of working with what may be termed "the mean annual mortality rate." The mean annual mortality rate over an interval of age, t, may be symbolized by ${}_t\mu_x$, where

$$ {}_t\mu_x = \frac{1}{t} \int_0^t \mu_{x+h} dh \, , $$

t (which may be fractional) being expressed in years.

For research along the lines on which the authors have embarked, the major advantages in working with ${}_t\mu_x$ include the following.

1. Exponential expressions for μ_x retain an exponential form in ${}_t\mu_x$.

2. The rate ${}_1\mu_x$ may be translated exactly into q_x (and vice versa) by using the formulas

$$q_x = 1 - e^{-{}_1\mu_x}, \qquad {}_1\mu_x = -\log(1 - q_x),$$

or may be very closely approximated by using the formulas

$$q_x \doteq \frac{{}_1\mu_x}{1 + \frac{1}{2}({}_1\mu_x)}, \qquad {}_1\mu_x \doteq \frac{q_x}{1 - \frac{1}{2}q_x}.$$

3. For component causes of mortality, for example, causes $r_1, r_2, \ldots$, such as the authors have considered in connection with biological systems, the following equalities hold:

$$\mu_x = \mu_x^{(r_1)} + \mu_x^{(r_2)} + \ldots.$$

Correspondingly,

$${}_t\mu_x = {}_t\mu_x^{(r_1)} + {}_t\mu_x^{(r_2)} + \ldots$$

and

$$q_x^{(r_1)} = 1 - \exp\left(-{}_1\mu_x^{(r_1)}\right),$$

and similarly for $r_2, \ldots$. The other formulas in paragraph 2 above also apply to individual causes.

4. The quantity ${}_t\mu_x$ may be derived directly from raw data by using the formula

$$\text{Crude } {}_t\mu_x = \frac{\text{Observed deaths}}{\text{Exposure (with deaths treated the same as any other increment or decrement, that is, exposed until time of death)}},$$

and exactly the same formula, with the same denominator, applies to any component cause (e.g., r_1) of mortality.

For a more extensive treatment of the foregoing formulas, reference may be made to F. A. Weck, "The Mortality Rate and Its Derivation from Actual Experience," *RAIA*, XXXVI (1947), 23–54.

(AUTHORS' REVIEW OF DISCUSSION)

AARON TENENBEIN AND IRWIN T. VANDERHOOF:

The authors would like to thank all the discussants for their comments, many of which were very detailed and thought-provoking. We appreciate the time the discussants spent on their very thorough reading of the paper. We will respond to the comments in alphabetical order.

Wilbur M. Bolton

Mr. Bolton's discussion of the attempts to fit the Makeham curve to the Commissioners 1980 Standard Ordinary Mortality Tables is very interesting. His measure of relative deviation is essentially the same measure of fit we used in Tables 6 and 7 of our paper. His query on the negative value of A can be resolved by setting A equal to zero; this results in a Gompertz curve. In fact, the value of A is very close to zero. This issue is an important one in curve-fitting because the value of A should be constrained to be greater than or equal to zero. The fact that A became zero may be an indication that the accidental death rate does not have a substantial relative effect for these data in the over-40 age range.

K. S. Brown

Dr. Brown's comments involve three basic themes: (1) the biological assumptions are inconsistent; (2) the variances of the transformed rates involve inconsistent approximations; and (3) the use of maximum-likelihood estimation may produce better fits. We will respond to each of these comments.

1. Dr. Brown's first point is that the value of f should vary over the life span. This is an unnecessary assumption, for two reasons. First, the form of the proposed model is such that the reduction of redundancy in living systems can be represented by a reduction in the number of components. It is not necessary for this model to represent the same reduction in redundancy twice.

Second, the assumption of identical distributions of components for different systems is obviously the simplest assumption that can be made. Another more complex assumption could be made, provided the justification for such an assumption existed. In general, the assumption of identical distributions and rates of failure of components is what we would expect from consideration of the evolutionary process. If there were a system that was weaker than any other, evolutionary forces eventually would weed it out. While there is necessarily a considerable range of redundancy of systems between individuals, consistent weaknesses are eliminated by evolution. The assumption used in the paper is, then, not only the simplest but also, generally, the most plausible.

Dr. Brown interprets the paper as saying that, if a component fails in one period, it has left the system. This is not our argument. Our argument is that components leave the system only through age, not through failure in an earlier period. This is consistent with the common observation that damaged parts of the body are repaired or replaced. Dr. Brown

then implies that the authors are confused because they have followed this argument rather than the argument that he presents. If we had used the assumptions he used, which we did not, we would have reached his conclusion, which we did not.

2. We used the approximations $1 - q \doteq 1$ and $\operatorname{colog}(1 - q) \doteq q$. Dr. Brown seems to imply that these two approximations are inconsistent because $\operatorname{colog}(1 - q) \doteq \operatorname{colog} 1 = 0$.

If this is what is being implied, it is erroneous. The impact of these approximations is the *relative error* that results from their use, because in a weighted least-squares procedure the *relative value* of these weights becomes important. The relative error in the first of these approximations is

$$\frac{1 - q - 1}{1 - q} \doteq q ,$$

or approximately $100q$ percent. The relative error in the second approximation is

$$\frac{\operatorname{colog}(1 - q) - q}{\operatorname{colog}(1 - q)} \doteq \frac{q}{2} ,$$

or approximately $100(q/2)$ percent. Both of these relative errors are satisfactorily low. The relative error in the third approximation (which we did not use) is

$$\frac{\operatorname{colog}(1 - q) - 0}{\operatorname{colog}(1 - q)} \doteq 1 .$$

This is a 100 percent relative error which, of course, is not satisfactory.

3. We did not use maximum-likelihood estimation because the data on mortality rates are based on amount and not on number, which makes it difficult to specify the likelihood function. If this difficulty could be overcome, either with other data or with approximate maximum-likelihood techniques, it would be useful to compare maximum-likelihood techniques with weighted least-squares methods. The approximations for the variances are consistent as explained under comment 2.

Michael Cohen

Mr. Cohen's comments relate to our omission of the work of British actuaries on graduation by mathematical formula. The main thrust of our paper was the fitting of select mortality rates, which are not used in the United Kingdom to the extent they are used here. To our knowledge, British actuaries have made no attempt to form select mortality models because they do not deal with this kind of data. As a result, we did not make reference to this literature. However, Mr. Cohen's comments are justified and we apologize for this omission. Another article of interest is

the Heligman and Pollard paper mentioned by Mr. Smith in his discussion.

Stuart Klugman

Dr. Klugman's comment on the use of residual analysis, rather than R^2, to determine fit is a very important point in any regression analysis. We discussed this issue in Section XII and presented results of a residual analysis in Tables 6 and 7 of our paper. His comment on the relative constancy of β_{xt}/α_{xt} over the various age-duration cells is correct. This ratio can be expressed as $\mu_{xt} + \sigma^2_{xt}/\mu_{xt}$, where μ_{xt} and σ_{xt} represent the mean and standard deviation of the face amounts in each age-duration cell. The assumption of relative constancy implies that the mean face amount does not vary appreciably from cell to cell and the standard deviation of face amounts is negligible.

Arthur Levenglick

As Mr. Levenglick points out, a number of strong assumptions are required to arrive precisely at Gompertz's law. We believe, however, that using weaker assumptions leads to a power-series version of Gompertz's law, which may be necessary in any case if we argue that each cause of death should follow Gompertz's law and that an individual may die of only one cause.

Mr. Levenglick also points out some inconsistency in terminology. We blushingly agree that consistent use of the term "standard" to represent lives that can now pass underwriting, and "select" to denote lives that have passed underwriting at some time in the past, would have been better.

Courtland C. Smith, Jr.

We appreciate the comments from Mr. Smith and believe, along with him, that the additional factors utilized by Heligman and Pollard may provide an answer to the problem of the peculiar bumps in the mortality curve at ages under thirty for males and at ages under fifty for females. We are currently investigating the possible uses of this factor and other similar factors to improve the fit of the equations. We have experienced some difficulty thus far. We would like to point out, however, that, even though the proposed formula does not fit the female data as well as we would like, the fit is very nearly as good as that of the published graduated tables. The evidence would seem to be, therefore, that the real pattern has not been caught either by us or by the graduators. We will report when our present efforts are completed.

Our response to Mr. Smith's numbered comments is as follows:

1 and 2. Mr. Smith's argument concerning graduation is a strong one, perhaps unduly so. Certainly he, and anyone else, has the right to use graduation techniques and represent the results as being just as scientific as those of others. It seems to us, however, that the use of graduation techniques implies a lack of understanding of the underlying phenomena. The faith expressed in this paper is that all phenomena eventually can be understood. As far as we can tell, this was the first attempt to develop a mathematical form for select mortality. The simplicity of the arguments that lead to the mathematical form must lead to the conclusion that the same, or a better, result could have been achieved by very many others—if they had believed that it was possible. The paper then can be looked upon as a demonstration that phenomena can be understood, and when they are understood graduation techniques can be discarded. Until they are understood, of course, we have to use the less satisfying technique.

3. Mr. Smith raises the problem of the actual composition of the groups at various durations. We have developed the argument that leads to the formulas but better answers Mr. Smith's point. It involves assuming that, at the end of one year, a certain number of lives become impaired and we can associate with them a number of unimpaired lives, so that this new collective exhibits ultimate mortality. This group should continue to exhibit ultimate mortality in the future, and, eventually, substantially all the original standard lives are associated with such impaired lives to form the total ultimate group. As was mentioned in the paper, the characteristics of an ultimate group are such that the distribution of lives within underwriting classes must be stable after it is formed. Only moderately strong assumptions are necessary to develop the argument completely, but the argument itself is long and complex and was, therefore, discarded in favor of the simpler intuitive argument presented. Certainly, as Mr. Smith points out, the final ultimate group must be composed of lives in all categories, since many of them can obtain standard insurance.

4. The financial viability of policies providing the option of reselection has not yet been demonstrated. We believe that there is a theoretical set of rates that could apply, but the rates we have seen do not always seem adequate.

5. In view of our remarks concerning Mr. Smith's comments under 1 and 2, we would have to argue that the experience of reinsurers for the years mentioned can be modeled, but we cannot now present an answer because we have not done it.

Paul Thomson

Mr. Thomson's comments on the use of the model to allow for bending of rates at higher ages are very interesting. The paper by Heligman and Pollard, which was mentioned in Mr. Smith's discussion of our paper, attempts to do the same thing.

Frank A. Weck

Mr. Weck brings up a number of interesting points. We believe that a large part of the observed differences between select mortality at very high durations and ultimate mortality is a result of their being drawn from different bodies of experience. We have not published the fitting of the proposed law of select mortality to nonmedical select data. In addition, the published ultimate data are reasonably consistent with the theory that they are a combination of these two separate bodies of data. Since the published ultimate data do include both medical and nonmedical policies, we believe that this is a reasonable explanation for the failure of the published select medical data to converge to the published ultimate table.

On the question of the assertion that a life in any class can end up in any other class, Mr. Weck misses a small point, but one that was intentionally introduced. The classes are always the classes into which an individual is put, or would be put, by an underwriter. Underwriters can make mistakes and are sometimes provided with incorrect data. An individual classified in one way because of a reported heart attack can move into the best class if the attending physician's report was in error. We know of precisely such a case. The argument was not put in this form just to catch the unwary reader, however. The reason for the insistence on underwriter classes rather than true status was so that the composition of groups was always, at least in principle, discoverable. We have avoided requiring at any stage knowledge of the true status of individuals. We developed the arguments, content with what could be actually perceived.

His questions about the various models and the use of the word "law" are appropriate. However, the third model has been tested by using a very wide variety of data going back to the last century. For every body of data developed since the American experience table, the parameter that represents the pattern of the development of select to ultimate mortality has shown most reasonable stability. Stability of this crucial parameter seems a real basis for arguing that there is something more than a chance relationship—that is, that there is a law. In addition, it is well known that the second-named author considers false modesty

such a grievous sin that he will go to any length, and take any risk, to avoid it.

We do not agree with Mr. Weck's next point. We believe that there is no reasonable basis for accepting a discontinuity between select and ultimate experience. We believe that the two must converge, unless they are representative of different pools of experience.

His last point, in connection with the fit of the formulas, is an interesting one, and one that we have considered. The simple result, which follows from the inspection of the residuals, is that there are peculiarities in the first several years. We believe that this is evidence of antiselection. The random pattern of the residuals, along with very high levels of R^2, precludes the existence of large persistent deviations from the fitted formula.