"LAWS" OF MORTALITY FROM THE BIOLOGICAL POINT OF VIEW.

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For some years one of my principal interests has been to study, in collaboration with my colleagues, E. M. Newbold, W. W. C. Topley and J. Wilson, the epidemiological phenomena observable in communities of mice exposed to risk of infection. In this work we have often found it convenient to summarise part of our results under the form of what we call a Life Table. We treat the entrants to our communities as live-births and determine the rate of mortality prevalent in the community over a certain epoch for each day of life, in the familiar life-table form. The result has a formal resemblance to a life table, but, from the nature of the case, it can have little or no bearing upon the course of events in a normal community. Naturally, however, one desired to set up some normal standard of mortality for the animal species used and, having obtained some scanty data, one was led to speculate further upon the biology of the so-called "laws" which have been from time to time proposed to describe the course of mortality in man. In this paper, I have brought together the imperfect results of such study as I have been able to make. Their practical value, from the point of view of the description of human mortality, is, it need hardly be said, negligible, while as a contribution to the history of "laws" of mortality what is omitted is perhaps as important as what is discussed. I have, however, felt justified in printing this essay in the hope that it might be accepted as a tribute *in piam memoriam* of my friend and colleague John Brownlee. The title of one of Brownlee's papers—"The Biology of a Life Table"—was the inspiration of much of his life-work. He approached the problem with an erudition, both biological and mathematical, to which I have no pretensions and, had his power of exposition been equal to his natural sagacity and learning, there would have been small need of any other writer.

The Concept of a Life Table.

A Life Table may be looked at from so many angles and may be constructed in so many ways that it is easy to fall into confusion as to what can and what cannot be learned from it. Let us begin with that ideal form which those of us whose duty it is to lecture, first describe to students. We assume n individuals all born on the same day and all exposed from the cradle to the grave to unchanging external conditions and we suppose them all to be under observation until the last survivor is dead; we have, to use the German term, a complete Order of Dying-out. Let it now be supposed that we really

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had observed n individuals under such conditions and that n is large and the sample typical of its "universe"; we shall, perhaps, wish to *graduate* the empirical series of l_x's[1] (a complete series for all values of x from 0 to that value of x for which $l_x = 0$), that is to say, we shall wish to replace the observed values by values which, while not differing materially from the observed values, have some advantage over them. The *advantage* in the last sentence might be one of several kinds. It might be, for instance, that some hypothesis adopted on, let us say, biological grounds as to the intrinsic connection between age and mortality could be clothed in an arithmetically manageable form and would give us values not substantially different from the observations. *Pro tanto*, such a result would be a justification of our biological hypothesis. Or, again, it might be that some assumed relation between the measures of age and of mortality, although without biological justification, would take an arithmetical form enabling us to determine readily some practically useful results dependent upon the probability of survivorship, such as the values of annuities on lives. Finally we might content ourselves with a method which without biological affinities or indirect arithmetical usefulness still expressed the functional relation between age and mortality with mathematical neatness and precision.

As a matter of history, no human life table closely approximating to the ideal has ever been used. The first life table made, that of John Graunt, of course diverged widely from it and his method of graduation (Graunt, Chap. XI) —for the pioneer with respect to the conception of an Order of Dying-out was actually the pioneer of graduation—is uncertain (Greenwood, 1928). Halley's Breslau Table was better than Graunt's but, like his, a population life table not an Order of Dying-out. The biological interest of a Life Table, in Brownlee's sense, was hardly thought of two hundred years ago, but the *practical* uses to which the method of that table could be put very soon commanded attention. Hence the importance of a graduation or "law" which could facilitate the monetary calculations to be based upon the Life Table, in days when arithmometers were not used. That need was the motive of the first useful hypothesis of graduation, Abraham de Moivre's. It is common form to speak of de Moivre's hypothesis, that the decrements of l_x are constant, with a certain condescension and to be told, for instance, that "de Moivre's hypothesis is only a rough approximation to the truth." By "truth" is here meant the form of l_x columns of tables constructed two or more generations after de Moivre was dead. Let us recur to de Moivre's own account of the hypothesis. He first notes that Halley's Breslau Table was based upon but a few years' experience and so was "not so entirely to be depended upon as to make it the foundation of a fixed and unalterable valuation of annuities on lives." He then pointed out that, in Halley's table, "the decrements of life, for considerable intervals of time, were in arithmetical progression; for in-

[1] For the sake of any readers who may be unfamiliar with the notation, a short note is appended to the paper.

stance, out of 646 persons of 12 years of age, there remain 640 after one year, 634 after two years, 628, 622, 616, 610, 604, 598, 592, 586, after 3, 4, 5, 6, 7, 8, 9, 10 years respectively, the common difference of those numbers being 6. Examining afterwards other cases, I found that the decrements of life for several years were still in arithmetical progression; which may be observed from the age of 54, to the age of 71, where the difference for 17 years together is constantly 10." That is to say, de Moivre knew that, even for Halley's table, the l_x's did not uniformly decrease in a single arithmetical progression. He therefore contemplated composing a table of the values of annuities "by keeping close to the tables of observation; which would have been done with ease, by taking, in the whole extent of life, several intervals whether equal or unequal." But, "before I undertook the task, I tried what would be the result of supposing those decrements uniform from the age of twelve; being satisfied that the excesses arising on one side, would be nearly compensated by the defects on the other; then comparing my calculations with that of *Dr Halley*, I found the conclusion so very little different, that I thought it superfluous to join together several different rules, in order to compose a single one." With respect to the assumption involved in his calculations that the extreme limit of life is 86 years, he remarked, firstly, that Halley's observations ceased at 84; secondly, that Graunt's table gave no survivor after 86—"this was deduced from the observations of several years both in the city and the country, at a time when the city being less populous, there was a greater facility of coming at the truth, than at present"; thirdly, that some Swiss tables of observation also gave the limit of life as 86. "As for what is alleged, that by some observations of late years, it appears, that life is carried to 90, 95, and even to 100 years; I am no more moved by it, than by the examples of *Parr*, or *Jenkins*, the first of which lived 152 years, and the other 167. To this may be added, that the age of purchasing annuities for life seldom exceeds 70, at which term *Dr Halley* ends his tables of the valuation of lives." These quotations are from the preface to de Moivre's work. In the text (p. 2 of third edition) he again says explicitly: "I will not say that the decrements of life are precisely in that proportion (*i.e.* in arithmetical progression), still, comparing that hypothesis with the table of *Dr Halley*, from the observations made at *Breslau*, they will be found to be exceedingly approaching." In other words, de Moivre's position was that the hypothesis while not exactly describing the scanty observations at his command, only diverged from them to an extent which did not materially affect the value of the functions he desired to calculate.

The distinguished actuary W. M. Makeham used language we might all adopt when he wrote: "Although subsequent observations have shown that de Moivre's attempt in the discovery of the law of mortality was made in the wrong direction, yet the idea was a happy and ingenious one, and the hypothesis by no means deserves the contumelious terms in which the late Mr Morgan permitted himself to speak of it. De Moivre, we may be quite

sure, was as fully alive as Mr Morgan could be to the shortcomings of his hypothesis; but he was also equally alive (which the other was not) to the defects of the tables formed with the means of observation then existing" (*J. Inst. Actuar.* XIII, 347). In the century between his work and that of Gompertz no contribution was made to the subject which, whether from the point of view of biological plausibility or of practical utility, was a real improvement upon de Moivre's "law"[1].

Benjamin Gompertz (1779–1865) the son of an Amsterdam diamond merchant of the Jewish race, and, like Boole, one of the small number of first rate mathematicians who were wholly self-taught, takes rank with such men as Woolhouse, Sprague and George Francis Hardy among the great masters of actuarial science. Augustus de Morgan, one of the first to recognise the significance of his work, said that "Had the law (*i.e.* Gompertz's) been propounded in the days of Newton, *vitality* would have been made a thing of, like *attraction*" (Adler, p. 13) and was his doughty and witty champion against attempts to deprive him of a rightful priority[2]. De Morgan, too, seems to have been the first to demonstrate the convenient property of Uniform Seniority to which Gompertz's method and its first modification by W. M. Makeham largely owed their usefulness in actuarial practice[3].

This is not the place, nor am I the person, to write a detailed account of the reception of Gompertz's method and Makeham's modification by the actuarial profession. I think, however, that the following is a not unfair summary of what happened. The attractiveness of the fundamental idea, its ease of algebraical expression, the fact that tables graduated by its help did not materially distort the observations, and the simplification of calculations involving annuities on joint lives which a table graduated by the Makeham-Gompertz hypothesis introduced, gave the Makeham-Gompertz "law" a position from which no other "law" (such for instance as those of Thiele and Wittstein) has shown the least likelihood of ousting it[4]. That, in our own time,

[1] While I do not fall behind Dr Singer in my respect for the memory of de Moivre, Dr Singer's statement that de Moivre's hypothesis "was under discussion for a century, but is now accepted" (Singer, p. 167) is, I think, benevolent rather than accurate. It may be added that Mr King's account of the history of the subject on pp. 68–70 of the second edition of Institute of Actuaries' *Text Book* (Part 2) does complete justice to de Moivre's position.

[2] See particularly the discussion initiated by Dr Morgan in the 9th volume of the *Journal of the Institute of Actuaries* (*Assurance Magazine*). I cannot resist quoting one sentence of de Morgan's rejoinder to Edmonds, "the process of bringing Dr Price into the paper is one which has been repeated many and many a time. When *A* is charged with dealing unfairly with the writings of *B*, he tries to prove—sometimes he does prove—that he has dealt just as unfairly with *C*." The importance of Gompertz's work was not at once realised. Thomas Young, an admirable Crichton, who *ought* to have perceived its significance, in a paper published in the *Phil. Trans.* for 1826 (Part 3, pp. 251–303) made a colourless reference to Gompertz's "reduction and interpolation" of Morgan's data and proposed a method of graduation which seems to have neither theoretical nor practical recommendations.

[3] de Morgan, *J.I.A.* **8**, 1860, 181.

[4] Cf. the 4th and 5th of Hardy's lectures on *The Theory of the Construction of Tables of Mortality*. London, 1909.

the hypothesis is of less *practical* importance than it was a generation ago is not because a better "law" has been discovered, but because improvements in the semi-skilled work of computation and the introduction of mechanical aids have reduced the demand for formulae or "laws" which may serve as labour-saving devices. What may be called the physiological interest of a Life Table, the sort of interest which would attach to the analysis of a *real* Order of Dying-out under uniform conditions, does not attach to the useful statistical fictions which we call Population Life Tables which tell us how a human generation *would* die out were it exposed from the cradle to the grave to rates of mortality to which no generation has been exposed, or is likely to be exposed. This criticism does not, no doubt, apply with the same force to tables based upon office experience, particularly that of assured lives of a social class and within a range of age with respect to which secular changes of mortality are slight. But the limitation implied and the further consideration that, to an actuary, a life table is not a subject for curious speculation but a working tool, are sufficient to explain why, in actuarial circles, interest in biological "laws" of mortality is lukewarm. But there are some lines of research in which such speculations have interest. With the ultimate object of learning how to describe, to formulate the "laws" of epidemic mortality, Topley and his colleagues exposed animal communities to special risks of death. The experience has covered the lives of many thousands of individual animals (mice) and we have expressed the results both in secular terms and also in the form of "real" life tables, actual Orders of Dying-out (subject to a limitation to be mentioned). Here we have the evolution of mortality with age under abnormal environmental conditions; one naturally asks what would be the development under more favourable conditions and whether it could be effectively described by some biologically plausible "law." Data for the study of the Order of Dying-out of animals living under "normal" conditions are far from numerous. For the animal interesting me, the mouse, I have at command two rather scanty series. Raymond Pearl and his associates—whose work on the biology of dying is of first rate importance—have provided and analysed more extensive data for the fruit fly *Drosophila melanogaster*. These "normal" series will be the, rather exiguous, basis of fact for what follows.

First, however, we must consider with some care the physiological implications of Gompertz's hypothesis and try to see how far it is worthy of the praise it received, that it "is based upon a physiological principle of high probability" (Adler, 13).

Gompertz's original enunciation of his principle in the memoir of 1825 (p. 517) has been reprinted so often that it is sure to be familiar to any reader of this paper. The paper he presented to the International Statistical Congress of 1860[1] (reprinted on pp. 329–344, *J. Inst. Actuar.* XVI, 1872) contains his

[1] It occupies pp. 454–462 of the "Programme and Report of the Proceedings etc." of the Congress, printed in 1860.

definitive opinions[1] and is less well-known. In this paper the author discusses a generalisation which amounts to replacing kg^{cx}[1] by a product of four terms, two of which are of the familiar form[2], the object being to graduate the whole life table from birth to extreme old age, which the original expression failed to do. With these modifications I am not concerned in this paper, because, for the animals and the range of age with which I have to deal, the force of mortality is increasing—the complication of points of inflection does not arise. Hence I am only interested in Gompertz's physiological interpretation of his increasing geometrical factor, the q^x of the 1860 paper (now usually written c^x). He writes: "I shall first pay attention to the force q^x, which, as q is above unity, will increase with x, and not decrease, as the density of the air in the receiver of an air-pump, by x equal strokes of the piston, but in indirect proportion, which I mention because the indefinite mode of expression I by some chance used in the article of my paper in the *Philosophical Transactions* of 1825, in the comparison I made of that force to the density of air in the receiver which remained after x strokes, may seem to express that q^x is in direct proportion to the reduced density, which is impossible when x increases, if q be greater than 1; and I observe that in the other forces, such as $A\lambda e.\, e^x$, where e is less than 1, the force might be expressed properly by stating it to be directly as the diminished density in the receiver by the strokes of the piston. This force q^x is a force to destroy life, or, in other words, a force diminishing the effect of the forces which preserve life. But though I thought the comparison an apt one to relieve the mind in search of a reason why the tables introduced the expression q^x, I had no wish to bind the theory of mortality to having sprung from such a cause, for there are a variety of actions which may arithmetically lead to the same numbers." Gompertz then repeats and illustrates his air pump analogy. This I need not quote, for, as he says, it is only an analogy and possibly nowadays Brownlee's chemical comparison is more enlightening. Brownlee (p. 43) observed that the Gompertz formula "implies that the substances or capacities on which life depends decay according to the law of the unimolecular reaction, that is, that the amounts present at the end of equal intervals of time can be represented by the terms of a geometrical progression."

The reader may have perceived that there is a possibility of confusion between individual and average, or statistical, formulae. Thus, he might urge,

[1] Makeham wrote of this paper: "this paper, which I believe is very little known, was drawn up by Mr Gompertz for the International Statistical Congress, during the preparation of his more elaborate contribution to the Royal Society in June, 1861. It contains much interesting matter, not included in the last mentioned paper—relating more especially to the physiological basis of the author's theory of mortality. The omission to incorporate this matter in his later paper arose doubtless from a fear of infringing the rule of the Royal Society against the admission to its "Transactions" of anything previously published. That it did not arise from any wish on the author's part to suppress any opinions expressed in the accompanying paper, is sufficiently proved by the fact that my copy was presented to me by Mr Gompertz in July, 1861, and, therefore, after his paper had been read before the Royal Society" (*J. Inst. Actuar.* **16**, 1872, 344).

[2] See Appendix.

while the concept of the individual life running down in terms of, say, a particular unimolecular change, is plausible—even if too beautifully simple to be likely to be true—Gompertz is not dealing with individuals but aggregates. Thus if the law be true of the individual, then should not the force of mortality for the aggregate of N individuals be represented not by Bc^x but rather by

$$\frac{1}{N}\sum_{i=1}^{i=N}(B_i c_i^x)?$$

As a matter of fact such a modification was actually introduced and sometimes figures in the literature under the name of Lazarus (Blaschke, p. 161). That Gompertz himself was quite alive to the difficulty appears from the following: "And to show that the formula does not require all persons of a birth to live to the same age whatever the causes may be on which the formula depends, suppose, instead of considering the terms ke^x, $k'xe'^x$, nq^x etc. as they stand in the formula, we take a function Ap^x on the supposition that it belongs to the formula, as by substitution of the elements it could be made to express either, as, if p be greater than 1, it might stand for q; if p be less than 1, it might stand for e or for e'; and supposing the different individuals of a birth were called 1, 2, 3, 4, etc., and that their portions referable to the term Ap^x were $A_1p_1^x$, $A_2p_2^x$, $A_3p_3^x$, etc., all different or not from each other, which supposition supposes only that Ap^x can for all values of x be

$$= A_1p_1^x + A_2p_2^x + A_3p_3^x \text{ etc.},$$

and then it would appear that the formula does not require all individuals of the same birth to live to the same age. Now, if p_1, p_2, p_3, etc., were all equal to each other, the supposition would only require A to be equal to

$$A_1 + A_2 + A_3 + A_4 \text{ etc.},$$

without requiring A_1, A_2, A_3 to be equal to each other; and if p_1, p_2, p_3 differed so little from each other that, though they were different, some would be but a small matter greater than some approximate value p, and some a small matter less, in such sort that p might be approximately taken for them, it would follow that Ap^x might be taken for an approximate result of the effect on the whole number of persons of which L_0 consists, though owing to the effect which is various on the individuals." Although Gompertz is *in part* addressing himself to an unreal objection (for I do not see why anybody should require his hypothesis to involve so simple a particular case as that of the equality of all individual life spans) it does take account of a difficulty. We determine from the data the *mean* force of mortality experienced by a group of N persons, we find that this mean value increases geometrically with age, is this consistent with the *individual* hypothesis? In other words is it probable that

$$\frac{1}{N}\sum_{i=1}^{i=N}B_i c_i^x$$

will be *approximately* equal to $\bar{B}\bar{c}^x$, where $\bar{B}$ and $\bar{c}$ are mean values?

Writing the former expression

$$\frac{1}{N}\sum_{i=1}^{i=N}(\bar{B}+e_i)(\bar{c}+\eta_i)^x,$$

where $\bar{B}$ and $\bar{c}$ are the means and e_i and η_i deviations from the means we see that it is equal to

$$\bar{B}\bar{c}^x+\bar{B}\bar{c}^x\left(\frac{x(x-1)}{2!}\frac{c\mu_2}{\bar{c}^2}+\frac{x(x-1)(x-2)}{3!\,\bar{c}^3}\,c\mu_3+\ldots\ldots\right)$$
$$+\bar{c}^x\left(\frac{x}{\bar{c}}\bar{p}_{11}+\frac{x(x-1)}{2!\,\bar{c}^2}\bar{p}_{12}+\ldots\ldots\right)$$

where the μ's are moment coefficients of c, and p's product moment coefficients of B and c.

If, as would in practice happen, B is a positive proper fraction, the second corrective term is likely to be the more important. It seems probable that Gompertz's surmise that the mean values would give a sufficiently close approximation is correct. For instance, Blaschke (p. 161) quotes the Gompertz constants for four different life tables and these, expressed as forces of mortality, are as follows: $0{\cdot}0077 \times 1{\cdot}0261^x$, $0{\cdot}0067 \times 1{\cdot}0164^x$, $0{\cdot}0047 \times 1{\cdot}0307^x$ and $0{\cdot}0039 \times 1{\cdot}0282^x$. The greatest force of mortality is almost double the smallest for $x = 20$. Now suppose a population constituted of equal groups of individuals the characteristics of each group being the four pairs of values of B and c, how would the real values of the forces of mortality for $x = 10$ and $x = 20$ differ from those reached by taking the mean values of B and c? The mean values of $B = 0{\cdot}00575$ and $c = 1{\cdot}02535$. The values of $0{\cdot}00575 \times 1{\cdot}02535^x$ for $x = 10$ and $x = 20$ are 0·007385 and 0·009486. By direct calculation the true values would be 0·007339 and 0·009393. Turning to the expansion, we see that all the terms except the first in each bracket will not affect the first four decimal places. We find $\frac{c\mu_2}{\bar{c}^2} = 0{\cdot}0000279$ and $\bar{p}_{11} = -0{\cdot}0000044825$. Hence for $x = 10$, we must add to 0·007385,

$$0{\cdot}007385 \times 4{\cdot}5 \times 0{\cdot}0000279 = 0{\cdot}00000927186$$

and must subtract

$$1{\cdot}2844 \times 9{\cdot}752767 \times 0{\cdot}0000044825 = 0{\cdot}0000548539.$$

We have

$$0{\cdot}007385 - 0{\cdot}0000548539 + 0{\cdot}00000927186 = 0{\cdot}0073394,$$

which is correct to the number of places required. The corresponding corrections for $x = 20$, viz. $-$ 0·00014425 and + 0·000050285, give for the force of mortality 0·009392, again correct. This is, of course, a mere arithmetical illustration, but it suggests that *if* the individual biological law were true, the average results—which are all we can possibly arrive at, since the dispersion of the *individual* c's and B's of the hypothesis and their frequency function, are not open to investigation—ought not to give a bad representation of the facts. It might be suggested that we should surmount the difficulty by fitting *ab initio* a sum of terms. As mentioned above, Lazarus proposed

to do this. In Blaschke's words: "As Makeham's table only correctly reproduces the mortality from 29–90 it was generalised by Lazarus who took into consideration the forces of mortality relative to the individual causes of death. If for n causes of death we have the forces of mortality

$$a_1 + b_1 q_1^x,\ a_2 + b_2 q_2^x \ \ldots\ a_n + b_n q_n^x$$

we reach $$\phi(x) = a_1 + a_2 + \ldots a_n + b_1 q_1^x + b_2 q_2^x + \ldots b_n q_n^x.$$

Hence $$l_x = c s^x k_1^{q_1^x} k_2^{q_2^x} \ldots k_n^{q_n^x}.$$

The application of this generalised form has led to the result that in all cases $l_x = c.s^x.k_1^{q_1^x}.k_2^{q_2^x}$ is sufficient." (Blaschke, p. 161.)

This passage does not do historical justice to either Gompertz or Makeham, both of whom added generalising terms to the formulae which go under their names. The reason why their extended formulae are forgotten is that the modifications deprived the "laws" of the practical advantages they possessed for actual computation. There is also, I think, a logical objection to identifying Gompertz's individual resisting powers with the mortalities assigned to different "diseases." It seems to me that the *formal* application of Gompertz's individual hypothesis to an aggregate requires us to assume that his B and c are continuous variables and that the force of mortality at age x is:

$$\frac{\iint \phi(c, B)\, B c^x\, dc\, dB}{\iint \phi(c, B)\, dc\, dB}.$$

This, however, is *merely* formal.

CONSTITUTIONAL AND ENVIRONMENTAL FACTORS OF MORTALITY.

Gompertz conceived the force of mortality as compounded of two factors, one independent of constitution *and* age, the other, in the mathematical sense, a direct function of age, its *scale* being fixed by the peculiarity of the individual make-up. He did not himself (in 1825) introduce into the "law" the former factor, that step was taken by Makeham, and the Makeham-Gompertz expression is the first acceptable formulation of what the "law" of mortality would be *if* the whole chance of death were compounded of two factors independent one of the other. Were that separation credible, that is to say, if the Order of Dying-out of any animals, really depended upon two independent factors, one of which was wholly independent of the physiological constitution of the individuals composing the population, the Makeham-Gompertz formula would be the ideal biometer—as Farr would have said—of the public health officer. Confronted with two Orders of Dying-out, say for Manchester and the Eastern Counties Rural Districts, then if the advantage of the rural districts were purely environmental (environmental in the man in the street's sense of preventable or removable without resort to eugenic action) he should find that the forces of mortality of the two life tables differed only in the A term. As a matter of statistical fact, this is not what one finds. Some years ago, stimulated by a paper of a German physiologist, Pütter, who thought he could make such a distinction (Pütter did not use the Gompertz-Makeham formula

but a slightly different and perhaps less convenient one), I Makehamised (Greenwood, 1924) a large number of life tables in order to see whether, for instance, the improvement of adult mortality at different epochs, that is under a (presumably) improving environment, were mainly due to a change of A[1]. Up to a point, the results were not inconsistent with the hypothesis, viz., on the whole, the A term did change much more than the term in Bc^x when one considered, for instance, the series of Swedish National Life Tables. But the theory is destroyed by a few awkward facts. Thus Trachtenberg found that the graduation of the English sectional table for the Rural Districts (1911–12) required an A term half as large again as that for London. It would require a bolder speculator than I am to believe that rural environment is 50 per cent. worse than that of London, and that the more favourable mortality of the country is due to the superior constitutional qualities of the exposed to risk—although Brownlee has used phrases respecting physiological age which might bear that interpretation. I conclude that this easy separation of the environmental and constitutional is far too good to be true and that we must try to be a little clearer as to what we mean by these words.

I do not propose to epitomise here the definitions of physiological constitution which have been proposed in the course of the two thousand years during which the subject has been discussed. Some account of the Greek doctrine will be found in my paper on Galen (Greenwood, 1921), while Günther (pp. 9 *et seq.*) and Pearl (pp. 97 *et seq.*) describe adequately the more intelligible of modern hypotheses. Whether we speak of a characteristic pattern, an individually characteristic labile chemical substance, or whatever mental picture we please, it is not (to me) possible to separate completely in the terms of a "law" of mortality, the constitutional from the environmental. Gompertz thought of the rate of decay of a something characteristic of the individual. Suppose one has a specific chemical substance undergoing change, the result will be affected by temperature, which in the present sense is a purely environmental factor. So it would be a reasonable hypothesis to argue that the original Gompertz "law" has already its environmental factor in the B term, that innately identical individuals brought under different environments should die out by Gompertz's law, the c's to be identical but the B's different. Statistically this is as close to (or as far from) the facts as the surmise that in the Makeham-Gompertz "law" A measures environment and Bc^x constitution. I have found, for instance (*vide infra*), that two Orders of Dying-out of mice kept under different environments were both well graduated (well, that is, having regard to the probable errors involved) by the simple Gompertz term. The c's differed very little, some 4 per cent., the B's enormously. But the whole discussion is trivial, for it virtually assumes that environmental mortality rates will be independent of age. This can seldom be literally true. If, for instance, an Order of Dying-out could be constructed in the following way, we should realise the condition. Of the survivors at age x, p per cent.

[1] G. F. Hardy and H. J. Rothery made a similar trial (*J. Inst. Actuar.* **27**, 1888, 179).

are taken out *by lot* and destroyed, where p is constant for all values of x. Under those circumstances we should bring into the expression for $\log l_x$ a term linear in x and the larger the p and the smaller the rate of mortality from other causes the more nearly would $\log l_x$ approximate to a straight line. On that assumption the separation by formula would succeed. But (with certain exceptions to be noted later) this is not the way environment operates. Suppose for instance that the *whole* of the risks associated with occupations of males or with the bearing of children in females were wholly independent of individual constitution; still, in the expression of the force of mortality, they would not be independent of x. If, of course, we admit a relation between constitutional make-up and liability to succumb to environmental assaults, the importance of the correlation with x is increased, because, on, *e.g.*, Gompertz's hypothesis, the proportion of individuals with small c's (*i.e.* great resisting powers) who survive to age x will be larger than the proportion of survivors with large c's.

I conclude that, certainly for human mortality, probably for all mortality, it is unlikely that environmental factors would be covered by a term in the expression of the force of mortality which is independent of x. That is perhaps a pragmatic justification of the course which, I suggested, seemed illogical of leaving Gompertz's ground and considering the forces of mortality of separate "causes." It might of course be that the functional relation between the force of mortality due to a cause of death such as tuberculosis, which has a maximum at a finite value of x, and x is really constitutional; but it is not *a priori* impossible that variations of exposure independent of constitution are the cause. That is the case for such a "law" as Thiele's. But, in practice, we could not thus separate the environmental from the constitutional, because the most wholehearted believer in the exposure doctrine of mortality from tuberculosis would shy from the proposition that the constitutional factor is entirely negligible. I now pass to the actual data.

THE "LAW" OF MORTALITY OF NORMAL MICE.

In the course of the joint work of Newbold, Topley, Wilson and the present writer several life tables (nine so far) have been constructed showing the Order of Dying-out of mice exposed to risk of death by infection. The work has involved the use of many thousands of mice and although the form of the calculations has been that of an Order of Dying-out, it does not relate to the Order of Dying-out from birth but from the date of entry into our herds, an unknown age, which might perhaps, on the average, be three or four months. But within the range of life open to our inspection, three things were obvious. First, that the actual average life span was very short, in no case much more than 60 days. Second, that the probability of dying, q_x, increased to an early maximum. Third, that when the maximum was passed there was no regular increase with age. These findings suggested that here the conditions were such that the factor of ordinary mortality which is of primary importance,

viz. that due to the increasing lability of the organism itself with increasing age was, relatively to the external factor, quite insignificant. To take a rough analogy, if a "life table" were constructed in terms of days at the front, starting a card for each soldier as he entered the trenches, it is quite certain that the influence of increasing natural age would be inappreciable upon the graph of q_x where x stood for "trench age" in days or weeks. We could study the effects of experience at the front with complete disregard of natural age. It seemed to us probable that this would be true for our epidemiological work. But there was this difficulty, that we *knew* nothing, and by inquiry could learn very little, about the way mice died when *not* exposed to heavy risk of fatal infection. It was *possible* that a proper mortality table for mice might differ very much in form from a human mortality table. *A priori* we did not anticipate a fundamental difference, having regard to Pearl's work on *Drosophila* which, biologically, is even more remote from man than is the mouse. Pearl—to whose work I shall return—found that the distribution with x of l_x for normal *Drosophila*, on certain plausible assumptions was closely similar to that of a human life table. To test the matter for mice two series of data were available. The first I owe to the kindness of Dr Leonard Hill, F.R.S., Director of the Department of Applied Physiology in the National Institute of Medical Research, the second to the kindness of Dr J. A. Murray, F.R.S., Director of the Imperial Cancer Research Fund.

Dr Hill carried out a research into the effects of various diets upon the mortality and fertility of mice (see Hill, A. B., *J. Hygiene*, 1925, XXIV, p. 232). The diets used were adequate and the mice used bred and reared at Hampstead under the charge of Miss Brad. The environment of these mice appears to have been very favourable; no epidemic broke out amongst them and the neonatal mortality was extraordinarily low. Of 269 mice born (the numbers are too small to admit of separate statistical treatment of the sexes) only one died within 60 days of birth and the average duration of life was 636·5 days. That the record is an exhaustive enumeration of all viable foetuses, that there was absolutely no cannibalism on the part of parents (a habit which experienced breeders affirm to be always found in a proportion of mice) cannot be proved; Miss Brad is, however, sure that losses in this way must have been, if there were any at all, very few.

We have here a real Order of Dying-out of a small but well-observed population. In the second column of Table I are shown the actual survivors at intervals of 50 days, in the third the Makeham-Gompertz and in the fourth the simple Gompertz graduations; the constants were obtained by the method of summation (4 consecutive sums of 213 days were formed). The equation for the Makeham-Gompertz graduation was:

$$\log l_x = 2{\cdot}43078 + 0{\cdot}0000136x - 0{\cdot}0039009\,(1{\cdot}006645)^x,$$

that is an "absurd" value for the linear term—and for the Gompertz graduation,

$$\log l_x = 2{\cdot}43222 - 0{\cdot}0039009\,(1{\cdot}006645)^x.$$

Table I. *Hill's Normal Mice.*

Ag (in days)	Observed l_x	Makeham-Gompertz graduations l_x	Gompertz graduations l_x
0	269	267·2	268·1
50	269	266·7	267·2
100	265	265·8	265·9
150	263	264·4	264·1
200	259	262·3	261·6
250	258	259·3	258·1
300	254	254·9	253·4
350	249	248·8	247·0
400	244	240·5	238·3
450	237	229·1	226·7
500	223	214·1	211·5
550	193	194·7	192·0
600	162	170·4	167·8
650	140	141·5	139·1
700	105	109·1	107·1
750	76	76·0	74·4
800	51	45·8	44·9
850	22	22·7	22·2

The two graduations differ very little and, by mere inspection, seem good. We have for χ^2 respectively 1·987 and 2·483. Were it proper to apply the usual "Goodness of Fit" test, these values would imply P's little less than unity. But that test is inappropriate. A more satisfactory plan is to compare the deviations in the observed q_x's, which are the really fundamental variates, with their respective standard deviations of sampling. This comparison is effected in Table II. If we seek to make a summary comparison, we may, *assuming* a "normal" distribution of errors, and ignoring the fact that the reduction to unit standard deviation has not allowed for the "weights" of the determinations of the individual standard deviations, proceed as follows. The mean absolute deviation of a Normal Curve with unit standard deviation is $\sqrt{\frac{2}{\pi}} = 0{\cdot}798$. For a sample of size 16 this will be subject to a standard error of 0·141. The actually observed mean absolute deviations are 1·09 for the Makeham-Gompertz and 0·99 for the Gompertz graduation. Either might have arisen without difficulty in sampling a "universe" characterised by $0{\cdot}798 \pm 0{\cdot}141$. The experience is a small one, but, one may suppose, the result would have given pleasure to Gompertz or to Brownlee. Dr Murray lent me the individual cards of female mice bred in connection with a study of the inheritance of cancer some years ago. For my purpose, the cards of 619 females were available, but the Order of Dying-out has been followed only from age 182 days because it appeared that the records of the first six months of life were not exhaustive. Table III sets out the data at intervals of 50 days. Remembering that the origin is 182 days, comparison with the Hill experience shows that the mortality is heavier. The mean after life time at age 182 days was for the Hill experience 466·8 days, for the Murray experience only 278·6 days (but still, of course, immensely longer than in any of our epidemic series). These data were graduated by Gompertz's "law" with the result shown. The

Table II. *Hill's Normal Mice.*

Makeham-Gompertz.

Age x	Observed $\pm$s.d. of observed q_x	Calculated $\bar{q}_x$	$q_x - \bar{q}_x$	$\frac{q_x - \bar{q}_x}{\sigma_{q_x}}$
0	0·0149 ±0·0044	0·0052	0·0097	2·20
100	0·0075 ±0·0045	0·0053	0·0022	0·49
150	0·0152 ±0·0055	0·0079	0·0073	1·32
200	0·0039 ±0·0067	0·0116	−0·0077	−1·15
250	0·0155 ±0·0080	0·0168	−0·0013	−0·16
300	0·0197 ±0·0096	0·0238	−0·0041	−0·43
350	0·0201 ±0·0114	0·0337	−0·0136	−1·19
400	0·0287 ±0·0136	0·0472	−0·0185	−1·36
450	0·0591 ±0·0161	0·0656	−0·0065	−0·40
500	0·1345 ±0·0192	0·0908	0·0437	2·28
550	0·1606 ±0·0238	0·1246	0·0360	1·51
600	0·1358 ±0·0295	0·1697	−0·0339	−1·15
650	0·2500 ±0·0355	0·2287	0·0213	0·60
700	0·2762 ±0·0449	0·3040	−0·0278	−0·62
750	0·3289 ±0·0561	0·3964	−0·0675	−1·20
800	0·5686 ±0·0700	0·5055	0·0631	0·90
Absolute mean				1·06
Gompertz.				
0	0·0149 ±0·0056	0·0084	0·0065	1·16
100	0·0075 ±0·0050	0·0068	0·0007	0·14
150	0·0152 ±0·0060	0·0095	0·0057	0·95
200	0·0039 ±0·0071	0·0132	−0·0093	−1·31
250	0·0155 ±0·0083	0·0183	−0·0028	−0·34
300	0·0197 ±0·0099	0·0254	−0·0057	−0·58
350	0·0201 ±0·0117	0·0352	−0·0151	−1·29
400	0·0287 ±0·0138	0·0487	−0·0200	−1·45
450	0·0591 ±0·0163	0·0671	−0·0080	−0·49
500	0·1345 ±0·0194	0·0922	0·0423	2·18
550	0·1606 ±0·0239	0·1260	0·0346	1·45
600	0·1358 ±0·0296	0·1711	−0·0353	−1·21
650	0·2500 ±0·0356	0·2299	0·0201	0·56
700	0·2762 ±0·0449	0·3049	−0·0287	−0·64
750	0·3289 ±0·0561	0·3975	−0·0686	−1·22
800	0·5686 ±0·0700	0·5061	0·0625	0·89
Absolute mean				0·9912

Expected value of the absolute mean by the normal curve = 0·798 ±0·141.

Frequencies of $\frac{q_x - \bar{q}_x}{\sigma_{q_x}}$.

Range	Expected by normal curve	Observed: Makeham-Gompertz graduations	Observed: Gompertz graduations
Over 4	0·001	—	—
3 to 4	0·042	—	—
2 to 3	0·685	2	1
1 to 2	4·349	7	7
0 to 1	10·923	7	8
	16·000	16	16

equation for $\log l_x$ is $3{\cdot}20453 - 0{\cdot}417749\ (1{\cdot}021669)^x$, wherein the unit of x is 10 days. The testing of concordance, by the method above suggested is shown in Table IV. Taking the summary comparison, it will be seen that the average mean absolute error differs but little from its expectation, the latter, since n is 13 instead of 16, being subject to a rather larger standard error, viz. $\pm$ 0·156, than before. Again, we can hardly be dissatisfied with the graduation.

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Table III.

x (days)	Murray's mice (observed)	Murray's mice (Gompertz graduations)	Hill's mice (taking 0 of Murray's experience = day 182 of Hill's experience and making the l_{182} of Hill = 619)
0	619	612·1	619
50	553	548·9	610
100	494	486·3	600
150	421	424·9	588
200	353	365·7	581
250	308	309·4	565
300	259	256·9	538
350	213	208·9	473
400	172	166·0	410
450	133	128·3	348
500	95	96·5	278
550	62	70·2	195
600	36	49·3	139
650	23	33·2	71

Table IV. *Murray's Mice.*

Age x	Observed ±S.D. of observed q_x	Calculated $\bar{q}_x$	$q_x - \bar{q}_x$	$\frac{q_x - \bar{q}_x}{\sigma_{q_x}}$
0	0·1066 ±0·0122	0·1033	0·0033	0·27
50	0·1067 ±0·0135	0·1140	−0·0073	−0·54
100	0·1478 ±0·0150	0·1263	0·0215	1·43
150	0·1615 ±0·0169	0·1393	0·0222	1·31
200	0·1275 ±0·0192	0·1540	−0·0265	−1·38
250	0·1591 ±0·0214	0·1697	−0·0106	−0·50
300	0·1776 ±0·0242	0·1868	−0·0092	−0·38
350	0·1925 ±0·0277	0·2054	−0·0129	−0·47
400	0·2267 ±0·0320	0·2271	−0·0004	−0·01
450	0·2857 ±0·0374	0·2479	0·0378	1·01
500	0·3474 ±0·0457	0·2725	0·0749	1·64
550	0·4194 ±0·0581	0·2977	0·1217	2·09
600	0·3611 ±0·0782	0·3266	0·0345	0·44
Absolute				0·8823

Expected value of the absolute mean by the normal curve = 0·798 ±0·156.

Frequencies of $\frac{q_x - \bar{q}_x}{\sigma_{q_x}}$.

Range	Expected by normal curve	Observed
Over 4	0·001	—
3 to 4	0·034	—
2 to 3	0·556	1
1 to 2	3·533	5
0 to 1	8·875	7
	12·999	13

Unless we suppose that different samples of mice differ greatly in innate biological quality—as perhaps they may—it would appear that the Murray mice did not live out their lives under such favourable conditions as those reared for Dr Hill. The c's of the two simple Gompertz graduations are not widely different—less than 2 per cent.—but the $\log g$ terms are far apart. I may, however, remark that it was not found possible to graduate the Murray data on the assumption that in Makeham's $A + Bc^x$, the second term was that of the Hill experience. To test the matter, approximate values of the force of mortality at 50 day intervals were computed from the Murray data and equated to $A + Bc^x$ where Bc^x was taken as known. The resultant gradua-

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tion was quite unsatisfactory. For reasons given already, it was not probable that this method would succeed.

Does it follow from these results that the Gompertz formula *more* effectively represents the law of mortality in mice than in men? To answer that question, at least in a way that is satisfactory to me, requires an experiment. After all, our data are ludicrously sparse; we have only a few hundreds of individuals while in the human experiences which, it is agreed, cannot be graduated by a Gompertz formula, the basal data ran to tens or hundreds of thousands. In the former case when the observed value is, for instance, 51 and the computed value 44·9, the contribution to, say, χ^2, is trifling although the *percentage* discrepancy is 13·5. A graduation giving precisely the same relative agreement as shown in the Hill case, but based upon a hundred times as many observations would have given us a χ^2 not of 2·486 but of 248·6. All we can fairly say is that, considering the size of the experience, the Gompertz graduation is an excellent one. Let us then see if a human experience, reduced to the same scale, obeys Gompertz's rule as effectively.

I made the following experiment.

To bring the mice to the same time scale as the human experience, we note that for the mice q_x is a minimum at the beginning of life while for men (*English Life*, No. 9) the minimum is at age 11. If we take the expectation of life at 11 as 55 years (the mean of the values for males and females) then as the mean expectation for mice is 636·5 days, the 55 human years are equivalent to 636·5 mice days. Or we might take the interval which elapses in the two cases before the survivors are reduced to 1 per cent. of the entrants (Pearl took 0·1 per cent. in his larger data). This requires us to equate (approximately) 1010 mouse days to 81 human years. By the former 1 mouse day would correspond to 0·086 human years, by the latter to 0·081 human years. Quinquennial intervals of a human table would correspond to from 58 to 62 mouse days. The next table (Table V) gives in the second column the actual survivors of the mouse experience at intervals of 62 days, and in the third the l_x's of

Table V.

Interval n (62 days for mice, 5 years for men)	Hill's mice l_n	English Life Table, No. 9 (males)	E.L.T. Gompertz graduations
0	269	269	265·7
1	268	266	264·2
2	264	262	262·0
3	261	257	259·0
4	258	252	254·8
5	253	246	249·0
6	247	238	241·0
7	240	229	230·3
8	225	217	215·9
9	184	201	197·2
10	152	180	173·6
11	118	152	145·0
12	80	117	112·5
13	49	77	78·7
14	17	41	47·6
15	7	15	23·4

English Life Table, No. 9 (Males) at quinquennial intervals from l_{11}, each entry having been multiplied by 269 and divided by the tabular value of l_{11}. This column is therefore in magnitude and time-scale roughly comparable with the second column. Let us now graduate by Gompertz's hypothesis. Fitting from three sums of five (*i.e.* using all the data but the last entry) we reach $\log l_x = 2{\cdot}430637 - 0{\cdot}00619036 \times 1{\cdot}409123^x$. The graduated values are shown in the fourth column. $\chi^2 = 5{\cdot}3105$ for this graduation. The test used before is applied in Table VI. The deviations of q_x are almost identically

Table VI. *English Life Table, No. 9 (Gompertz Graduations).*

Observed ±S.D. of observed q_x	Calculated $\bar{q}_x$	$q_x - \bar{q}_x$	$\frac{q_x - \bar{q}_x}{\sigma_{q_x}}$
0·0112 ±0·0045	0·0056	0·0056	1·24
0·0150 ±0·0056	0·0083	0·0067	1·20
0·0191 ±0·0066	0·0115	0·0076	1·15
0·0195 ±0·0079	0·0162	0·0033	0·42
0·0238 ±0·0094	0·0228	0·0010	0·11
0·0325 ±0·0112	0·0321	0·0004	0·04
0·0378 ±0·0134	0·0444	−0·0066	−0·49
0·0524 ±0·0160	0·0625	−0·0101	−0·63
0·0737 ±0·0191	0·0866	−0·0129	−0·68
0·1045 ±0·0229	0·1197	−0·0152	−0·66
0·1556 ±0·0276	0·1647	−0·0091	−0·33
0·2303 ±0·0338	0·2241	0·0062	0·18
0·3419 ±0·0424	0·3004	0·0415	0·98
0·4675 ±0·0557	0·3952	0·0723	1·30
0·6341 ±0·0781	0·5084	0·1257	1·61
Absolute mean			0·7347

Expected value of the absolute mean by the normal curve = 0·798 ±0·146.

Frequencies of $\frac{q_x - \bar{q}_x}{\sigma_{q_x}}$.

Range	Expected by normal curve	Observed
Over 4	0·001	—
3 to 4	0·040	—
2 to 3	0·642	—
1 to 2	4·077	5
0 to 1	10·241	10
	15·001	15

distributed in accordance with "expectation" and the mean absolute deviation is 0·73, sensibly the same as its expectation. If *only* an experience of this order of magnitude had been available, it would have been fair to say that the Gompertz formula described the "law" of human mortality over the range from age 11 to age 86. The answer, therefore, to my question is, that we have *no* sound reason for thinking that the force of mortality in mice increases with age more nearly geometrically than the force of mortality in men. On the contrary, it is probable that if our statistical experience of mice were as great as that of men, we should reach the same conclusion with respect to the applicability of the "law" as actuaries have reached for human experience. To some readers this demonstration will be superfluous, but there may be a few who will benefit by it; it is not very difficult to be impressed by agreement between observation and hypothesis and to forget the *scale* of our observations.

Some years ago Mr Elderton, speaking of the χ^2 test, said: "I have found, in applying this test, that when the numbers dealt with are very large, the probability is often small, even though the curve appears to fit the statistics very closely. The explanation is that the statistics with which we deal in practice nearly always contain a certain amount of extraneous matter, and the heterogeneity is concealed in a small experience by the roughness of the data. The increase in the number of cases observed removes the roughness, but the heterogeneity remains. The meaning, from the curve-fitting point of view, is that the experience is really made up of more than one frequency-curve; but a certain curve, approximating to the one calculated, predominates" (Elderton, p. 142). If this reasoning were just, we might suppose that for both mice and men one element of mortality is really expressed by Gompertz's "law" and agree with de Morgan that Gompertz made an important discovery and with Brownlee that a geometrical law is a partial explanation of the facts of animal life.

The conclusions reached with respect to mice hold for Pearl's flies. Pearl's largest series consisted of 1407 male and 1415 female flies and he graduated the experiences (separately) by means of the formula

$$\log l_x = e^{dx}(a + bx + cx^2 + dx^3).$$

Table VII compares the actual values of l_x, at intervals of 6 days, with the sums of Pearl's graduations, a simple Gompertz and a Makeham-Gompertz graduation of the data (sexes not distinguished). Here again the Makeham-

Table VII. *Pearl's Normal Drosophila (both Sexes).*

x (days)	Observed l_x	Gompertz-Makeham graduations l_x	Gompertz graduations l_x	Pearl's graduations (sum of males and females)
1	2822	2824	2839	2822
7	2781	2792	2792	2798
13	2753	2740	2726	2733
19	2693	2657	2630	2635
25	2522	2534	2496	2500
31	2337	2357	2310	2328
37	2109	2114	2061	2106
43	1763	1796	1742	1820
49	1438	1409	1559	1463
55	1070	983	943	1046
61	510	576	551	624
67	272	262	249	278
73	111	82	77	80

Gompertz graduation requires an absurd (positive) value of s. The values of χ^2 are 37·49 for Pearl's graduations, 45·06 for the Gompertz graduations and 27·94 for the Makeham-Gompertz graduation. The relative deviations of the q_x values in all three cases are also improbably large, having mean values of 3·34, 2·99 and 3·10 (see Table VIII). There is little to choose between these results, none is a good fit by the usual criterion and each would have been an excellent fit, by that criterion, had the data been one-tenth as extensive, *i.e.* on the scale of the Hill experience.

Table VIII. *Pearl's Flies.*

Gompertz-Makeham graduations.

Age x	Observed $q_x \pm$ S.D.	Calculated $\bar{q}_x$	$q_x - \bar{q}_x$	$\frac{q-\bar{q}}{\sigma_{q_x}}$
1	0·0145 ±0·0020	0·0113	0·0032	1·60
7	0·0101 ±0·0026	0·0186	−0·0075	−2·88
13	0·0218 ±0·0033	0·0303	−0·0085	−2·58
19	0·0635 ±0·0040	0·0463	0·0172	4·30
25	0·0734 ±0·0051	0·0698	0·0036	0·71
31	0·0976 ±0·0063	0·1031	−0·0055	−0·87
37	0·1641 ±0·0078	0·1504	0·0137	1·76
43	0·1843 ±0·0098	0·2154	−0·0311	−3·17
49	0·2559 ±0·0121	0·3023	−0·0464	−3·83
55	0·5233 ±0·0151	0·4140	0·1093	7·24
61	0·4667 ±0·0221	0·5451	−0·0784	−3·55
67	0·5919 ±0·0281	0·6870	−0·0951	−3·38
Absolute mean				2·989

Gompertz graduations.

Age x	Observed $q_x \pm$ S.D.	Calculated $\bar{q}_x$	$q_x - \bar{q}_x$	$\frac{q_x-\bar{q}_x}{\sigma_{q_x}}$
1	0·0145 ±0·0024	0·0163	−0·0018	−0·75
7	0·0101 ±0·0029	0·0239	−0·0138	−4·76
13	0·0218 ±0·0035	0·0350	−0·0132	−3·77
19	0·0635 ±0·0042	0·0512	0·0123	2·93
25	0·0734 ±0·0052	0·0745	−0·0011	−0·21
31	0·0976 ±0·0064	0·1079	−0·0103	−1·61
37	0·1641 ±0·0079	0·1548	0·0093	1·18
43	0·1843 ±0·0099	0·2195	−0·0352	−3·56
49	0·2559 ±0·0122	0·3060	−0·0501	−4·11
55	0·5233 ±0·0151	0·4163	0·1070	7·09
61	0·4667 ±0·0220	0·5478	−0·0811	−3·69
67	0·5919 ±0·0281	0·6896	−0·0977	−3·48
Absolute mean				3·095

Pearl's graduations.

Age x	Observed $q_x \pm$ S.D.	Calculated $\bar{q}_x$	$q_x - \bar{q}_x$	$\frac{q_x-\bar{q}_x}{\sigma_{q_x}}$
1	0·0145 ±0·0017	0·0085	0·0060	3·53
7	0·0101 ±0·0029	0·0232	−0·0131	−4·52
13	0·0218 ±0·0035	0·0358	−0·0140	−4·00
19	0·0635 ±0·0042	0·0512	0·0123	2·93
25	0·0734 ±0·0050	0·0688	0·0046	0·92
31	0·0976 ±0·0061	0·0954	0·0022	0·36
37	0·1641 ±0·0075	0·1358	0·0283	3·77
43	0·1843 ±0·0095	0·1962	−0·0119	−1·25
49	0·2559 ±0·0119	0·2850	−0·0291	−2·45
55	0·5233 ±0·0150	0·4034	0·1199	7·99
61	0·4667 ±0·0220	0·5545	−0·0878	−3·99
67	0·5919 ±0·0275	0·7122	−0·1203	−4·37
Absolute mean				3·34

Expected value of the absolute mean by the normal curve = 0·798 ±0·163.

Frequencies of $\frac{q_x - \bar{q}_x}{\sigma_{q_x}}$.

Range	Expected by normal curve	Observed: Makeham-Gompertz graduations	Observed: Gompertz graduations	Observed: Pearl's graduations
Over 4	0·001	2	3	3
3 to 4	0·032	4	4	4
2 to 3	0·514	2	1	2
1 to 2	3·262	2	2	1
0 to 1	8·192	2	2	2
	12·001	12	12	12

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If it were true that the Gompertz "law" expressed some important element of biological truth, would its distortion when adequate data, from a numerical point of view, are at command depend upon the fact, (*a*) that its functional expression is imperfect, or (*b*) upon the blurring effect of the "environmental" factors which it cannot be supposed adequately to express? I surmise that both explanations are true. So far as the improvement of the mathematical expression of the "law" is concerned, there is little reason to suppose that we shall make any more progress by statistical experiment upon the data of *human* mortality. The data I have for mice are too scanty to justify further manipulation, for, so far as they are concerned, the Gompertz formula is adequate and we could not *prove* that any other imagined expression was better. Pearl disposed of more extensive material and in the next section I discuss some of his conclusions.

Professor Pearl's Results.

Pearl remarked that the forms of the function $\log l_x$ which have been observed lie between two straight lines drawn from that point on the axis of $\log l_x$ which corresponds to $x = 0$ (the arbitrary radix of the survivorship table). These two straight lines imply that the force of mortality is constant; the upper (Pearl's rectangular type) is the $\log l_x$ line if the force of mortality is zero within the range of observation, so that no deaths occur; the lower when the force of mortality is a constant greater than unity. It will be noticed that the first form emerges from Gompertz's formula when c approximates to 0 and the second form when c approximates to 1. For, by Gompertz's formula, $\log l_x = k - Bc^x/\log c$, and $\log l_0$ is an arbitrary constant, say N, so that $k = N + B/\log c$, or $\log l_x = N + B(1 - c^x)/\log c$ which is N when $c = 0$ and $N - Bx$ when c tends to 1. Above the diagonal would fall all the cases of c greater than 1. For instance, if 1000 entrants decreased logarithmically so that at the end of 80 years there were only one survivor, the logarithms (common) of the survivors after 10, 40 and 60 years would be 2·625, 1·500 and 0·750. If the population obeyed Gompertz's law with $c = 1{\cdot}1$ and the other constants so chosen that after 80 years there was one survivor, the $\log l_x$ for $x = 10$ would be 2·998, for $x = 40$, 2·935 and for $x = 60$, 2·555, the graph—which would, of course, be convex upwards—forming a bow with the diagonal as its string, attached at $x = 0$ and $x = 80$.

The domain below[1] and to the left of the diagonal would be occupied by types which Pearl notes are theoretically possible but which have not yet been realised in practice, viz. when the rate of mortality decreases with age. This could be represented by a Gompertz formula with c less than one (if c

[1] Of course the convex (upwards) and concave curves are respectively above and below the "diagonal" only when the assumption is made that the curves intersect the "diagonal" at some finite distance to the right of the origin, an assumption made by Pearl in reducing his tables to a common measure (see above, pp. 282–283, where I have virtually adopted Pearl's method of reduction). Naturally by a suitable choice of constants a convex curve might lie wholly below and to the left and a concave curve wholly above and to the right of any given "diagonal."

is less than one, $\log l_x = k + Mc^x$, where M is a positive quantity) and, in the imagined case of an expression giving one of a thousand surviving to age 80, the bow would be below and to the left of the diagonal string, the curve being concave upwards. If we took $c = 0{\cdot}9$, the values of $\log l_x$ for $x = 10$, 40 and 60, would be 1·046, 0·044 and 0·005. Of course in the three imagined cases, with increasing x, the proportion of survivors diminishes indefinitely in the diagonal and upper domain cases and in the lower domain case is asymptotic to a constant value. Pearl finds that the closest approximations to the rectangular type yet observed are those of a human life table and of that of the normal wild type of *Drosophila*, while that of the much shorter lived vestigial type approximates more closely to the diagonal curve for $\log l_x$. He is here referring to the comparison of the transformed graphs (see chart, Pearl, p. 43), where the fly curves are expressed in a time unit which makes all the l_x's the same for $x = 0$ and $x = k$, where k is different for different species. The vestigial *Drosophila* were reared under precisely the same environmental conditions as the longer lived wild type. Pearl next deals with the survivorship curves of flies reared under different conditions and shows first of all that there is an optimum density of population, from the point of view of survivorship. "The survivorship distributions of the extremely high densities approximate closely to the 'straight diagonal line' type of life curve which has been discussed in Chap. III, and shown to be characteristic of vestigial flies" (Pearl, p. 59). Finally, Pearl compares the Order of Dying-out of flies completely deprived of nourishment and finds, once more expressing the results by the method of transformation, that the $\log l_x$ curve of the starved flies differs little in form from that of the fed flies which, we have already seen, is closely similar to a human $\log l_x$ curve (Pearl, p. 107). It was also found that, under conditions of starvation, variations of density of population did not affect the form of $\log l_x$ nor was there any significant difference between normal wild and vestigial flies. He remarks: "this result I take to mean the following things:

"1. That the *inherent* vitality of an individual fly is not, in fact, altered by the environmental circumstances in which its life is lived. It is, on the contrary, of the nature of a constant for the individual, in the sense that the morphology of a leg, for example, is constant for the individual.

"2. That the difference between normal wild type flies and vestigial flies in respect of duration of life, which under normal conditions of feeding (that is when it is the expression of the total vitality implicit in the normal $A + B$ physiological economy) follows the Mendelian laws of inheritance, is *not* dependent upon a fundamental difference in *inherent* vitality. This difference, on the contrary, appears merely to be due to the fact that under the environmental conditions represented by the standard fly husbandry of the laboratory (the A of our schema) vestigial flies were not able to bring their inherent vitality to so complete expression in duration of life as were the wild type flies under the same conditions" (Pearl, p. 127). Part of Pearl's argument turns upon the congruence or want of congruence of the functions $\log l_x$ for different

species when reduced to a common scale. Algebraically, Pearl's method of reduction is this. We have two functions of x, $f_1(x)$ and $f_2(x)$, such that $f_1(0) = f_2(0)$ while for all finite values of x greater than 0, $f_1(x)$ is greater than $f_2(x)$ and both functions decrease with x. A value of x, say a, is chosen and the value of x is found for which $f_2(x) = f_1(a)$. Suppose this value is b, where of course b is less than a, then $f_2(x)$ is replaced by $f_2\left(\frac{a}{b} \cdot x\right)$. In the particular case of the two functions being Gompertz functions which differed only in respect of the c term, *i.e.* $A - Bc_1^x$ and $A - Bc_2^x$ where c_2 is greater than c_1, the transforming factor would be $\log c_1/\log c_2$ so that the transformed function would coincide with its standard. If

$$f_1(x) = A_1 - B_1c_1^x \text{ and } f_2(x) = A_2 - B_2c_2^x$$

with the condition $A_1 - B_1 = A_2 - B_2$. Then the substitution in $f_2(x)$ of:

$$\frac{\log c_1}{\log c_2} \cdot \frac{\log\left[1 + \frac{A_1 - B_1}{B_2}(1 - \lambda)\right]}{\log\left[1 + \frac{A_1 - B_1}{B_1}(1 - \lambda)\right]} x$$

for x will fulfil the conditions, where $\lambda(A_1 - B_1)$ is the value of $f_1(x)$ to which $f_2(x)$ is to be equated. Here $0 < \lambda < 1$ while $B_2 \neq B_1$.

Obviously here—and indeed generally—the transforming factor is a function of λ and the question arises as to what should be the criterion determining it. Pearl has taken $\lambda = 0{\cdot}001$[1] and the second and third columns of Table IX compare the values of l_x for normal male and "vestigial" male

Table IX.

Centiles of life span	Line 107 males	Vestigial males $\lambda = 0{\cdot}001$	Vestigial males $\lambda = 0{\cdot}5$	Centiles of life span	Line 107 males	Vestigial males $\lambda = 0{\cdot}001$	Vestigial males $\lambda = 0{\cdot}5$
0	1000	1000	1000	55	564	94	588
5	997	989	999	60	466	65	529
10	988	938	989	65	363	44	471
15	973	851	971	70	258	30	365
20	952	738	943	75	167	21	317
25	924	613	908	80	94	14	274
30	890	486	866	85	45	9	235
35	847	371	817	90	17	6	200
40	794	274	763	95	5	3	170
45	730	196	707	100	1	1	144
50	654	137	648				

Drosophila so standardised. The third column gives the figures obtained when the point of agreement is the median, viz. $\lambda = 0{\cdot}5$. So far as the graphs of the results (whether of l_x or $\log l_x$) are concerned, the greater λ the more nearly

[1] I am not quite clear what precise value was taken to reach Table XXI of Pearl's book. It appears from Tables XVII and XIX that wild males are reduced to 0·001 times the entrants in 81 days and vestigial males in 42 days but the survivorship table of vestigial males at intervals of 42/81 days, or (on the centile basis) wild males at intervals of 0·81 days and vestigial males at intervals of 0·42 days do not precisely reproduce Table XIX. The difference is not, however, important.

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the graphs will resemble one another because agreement is imposed where the values of y are large. In dealing with empirical data limited in extent, there is a disadvantage in making λ small, because the probable error of the number of survivors is large; we have a very poor approximation to the mathematical expectation of the value of x which makes $l_x/l_0 = 0{\cdot}001$.

Of course if the function it is proposed to transform either really is or approximates at all closely to a linear function of x (as when the force of mortality is sensibly constant for all values of x), all that happens is that its slope is changed. If the curve to which it was to be standardised decreased very little over a considerable range of x, by a suitable choice of λ the standardised curve would, over that range, lie very close to the standard of reduction, *i.e.* if λ be so chosen as to make the straight line intersect the curve at the point where the latter's rate of mortality began to increase sensibly. Thereafter, the standard curve would lie wholly below the standardised straight line. These considerations, although tending to show that the method of reduction to equivalent life spans is by no means free from difficulties, do not, I think, affect any of the conclusions drawn by Pearl. Thus, in the last imagined case, the increasing divergence between the standard and the transformed straight line would be a perfectly cogent proof of diversity between the "laws" or mortality of the two species of samples, provided the equivalent spans were so chosen that the observations available for values of x after the value for which the ordinates were equal were not too scanty.

In Table X I give some results of the method of standardisation in mice. A comparison is made of the survivorships of the normal mice (Murray's sample) and the mice which formed the subject of Experiment 2 of Greenwood and Topley's 1925 memoir. The full details of survivorship of these latter mice will be found in the memoir (Greenwood and Topley, 1925, pp. 75 *et seq.*). The number of mice under observation from the time of entry into the infected herd until death was 2354, and the mean after life time (expectation of life) at entry was 21·48 days. As already stated, Murray's mice numbered 619 and the mean after life time at the age of 182 days was 278·58 days. In Table X, second column, we have the survivorship of the normal mice at intervals of 20 days and in the third column that of the mice exposed to *pasteurellosis*. In the fourth column we have the transformed values when agreement is imposed at the medians, the fifth column contains the results when the standardisation is based upon equality when l_x is 100 and the sixth column contains the results when l_x is 10 in each experience. The first standardisation is of the form mentioned in the last paragraph, quite close agreement is imposed for a considerable distance, until interval 23, and thence an increasing and enormous divergence is obvious. The other transformations produce agreement only in the vicinity of the arbitrary points of accord. It will be noticed that, if the data for mice under experiment had numbered 235 instead of 2354, one might easily have been impressed by the closeness of agreement between the normal and transformed experimental series over

Table X. (20 *days interval for Normal Mice.*)

Equivalent intervals	Normal mice l_n (Murray)	Mice of Experiment 2			
		Observed	$\lambda=0{\cdot}5$	$\lambda=0{\cdot}1$	$\lambda=0{\cdot}01$
0	1000	1000	1000	1000	1000
1	958	293	977	946	870
2	910	138	943	858	685
3	885	85	905	756	548
4	834	46	847	668	435
5	798	28	794	586	325
6	748	19	742	528	250
7	704	10	693	468	210
8	648	4	652	402	180
9	596	3	609	347	162
10	570	2	571	299	146
11	536	1	541	261	136
12	504	0	511	230	126
13	481	—	480	212	116
14	454	—	447	197	106
15	418	—	412	181	95
16	389	—	379	170	86
17	363	—	351	162	76
18	334	—	325	152	71
19	302	—	301	146	62
20	278	—	280	140	58
21	267	—	261	134	50
22	231	—	243	129	42
23	210	—	229	124	41
24	179	—	219	118	38
25	153	—	211	114	36
26	128	—	203	109	32
27	111	—	194	102	28
28	87	—	185	96	26
29	71	—	178	91	26
30	58	—	172	87	25
31	52	—	168	81	22
32	42	—	163	77	19
33	34	—	160	73	17
34	29	—	154	71	14
35	24	—	149	68	13
36	16	—	146	61	12
37	13	—	144	60	11
38	8	—	141	56	10
39	3	—	137	52	8

that part of the range for which the observations were numerous and have been inclined to explain away the subsequent discrepancy on the ground of paucity of observations. Even when the data of Pearl are in question, I am not quite sure that Pearl's graph of the log l_x's really justifies his remark that "From these life table curves several results of interest emerge. In the first place it is evident that the distribution of mortality in the different parts of the biologically equivalent life span is substantially identical quantitatively in an inbred strain of *Drosophila* (line 107) and in human beings of the present time" (Pearl, p. 44). As my own reduction of normal mice to the human scale (see Table V *supra*) diverged sensibly from the "equivalent" human values, even when allowance was made for errors of sampling, I at first thought that, possibly owing to less adequate stability of environmental conditions, my reduction was less successful than Pearl's. An experiment, the results of which are set out in Table XI, leads me to doubt this. From Pearl's tables it appeared that for male (normal) *Drosophila* 1000 entrants had diminished to

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Table XI. *Wild Male Drosophila and English Life Table, No. 9 (Males) reduced to equivalence.*

	E.L.T. No. 9	*Drosophila*		E.L.T. No. 9	*Drosophila*
0	1407	1407	8	1082	929
1	1392	1400	9	970	775
2	1368	1380	10	816	599
3	1340	1348	11	621	418
4	1309	1303	12	397	252
5	1270	1241	13	193	124
6	1222	1161	14	63	46
7	1162	1058	15	13	13

9 after 75 days' exposure. Taking the survivors at age 11 of English Life Table, No. 9 (males) as the basis it appears that these entrants will have decreased to 0·9 per cent. of their strength at age 92·477 years. Hence 81·477 human years are biologically equivalent to 75 *Drosophila* days ($\lambda = 0{\cdot}009$). That is if we interpolate the human table at intervals of 5·432 years we may compare the values with those of the *Drosophila* table at intervals of 5 days. If we then take l_0 as 1407 (the number of male *Drosophila* entrants) we have values comparable, on the hypothesis, and of the right scale. These form the second and third columns of Table XI. Clearly the agreement of the two series is not close. Calculating the q_x's of the standard series and of the Pearl observations and comparing the differences between observed and calculated q_x's with the standard error of the latter as before, one can satisfy oneself that the discrepancies are unlikely to have arisen as errors of sampling (see

Table XII.

Age x	*Drosophila* $q_x \pm$S.D.	English Life Table, No. 9, $\bar{q}_x$	$q_x - \bar{q}_x$	$\frac{q_x - \bar{q}_x}{\sigma_{q_x}}$
0	0·0050 ± 0·0027	0·0107	−0·0057	−2·11
1	0·0143 ± 0·0035	0·0172	−0·0029	−0·83
2	0·0232 ± 0·0038	0·0205	0·0027	0·71
3	0·0334 ± 0·0041	0·0231	0·0103	2·51
4	0·0476 ± 0·0047	0·0298	0·0178	3·79
5	0·0645 ± 0·0054	0·0378	0·0267	4·94
6	0·0887 ± 0·0063	0·0491	0·0396	6·29
7	0·1219 ± 0·0078	0·0688	0·0531	6·81
8	0·1658 ± 0·0100	0·1035	0·0623	6·23
9	0·2271 ± 0·0131	0·1588	0·0683	5·21
10	0·3022 ± 0·0174	0·2390	0·0632	3·63
11	0·3971 ± 0·0235	0·3607	0·0364	1·55
12	0·5079 ± 0·0315	0·5139	−0·0060	−0·19
13	0·6290 ± 0·0421	0·6736	−0·0446	−1·06
14	0·7174 ± 0·0597	0·7937	−0·0763	−1·28
Absolute mean				3·14

Expected value of the absolute mean by the normal curve = 0·798 ± 0·146.

Frequencies of $\frac{q_x - \bar{q}_x}{\sigma_{q_x}}$.

Range	Expected by normal curve	Observed
Over 4	0·001	5
3 to 4	0·040	2
2 to 3	0·642	2
1 to 2	4·077	3
0 to 1	10·241	3
	15·001	15

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Table XII). It is, of course, but one experiment, a different value of λ might have given a better result[1], but I see no reason to believe that any such transformation would render the functions substantially identical, if by that phrase we mean render the transformed function such that the values might easily have arisen in random sampling within a universe typified by the standard function.

After all, our function *is* rather hampered by conditions. The orginal function *must* decrease, or at least not increase, with x and the transformed function must agree with the standard at two values of x. Take the logarithm of such a function, plot on a small scale, and much real discrepancy is compatible with graphical similarity.

Pearl, of course, does not base his case merely upon this, as I think, slender evidence. His beautiful experiment upon the effects of pure starvation, showing a congruence of the log l_x curves of wild and vestigial flies, is not dependent upon the method of translation. That experiment illustrates well the operation of a factor which, in one sense, is environmental, but in the sense of this paper is not environmental at all but purely constitutional.

Concluding Observations.

With respect to the logical basis and applicability of "laws" of mortality to the phenomena of human life, it appears that nothing of real importance has been added to the work of Gompertz and that his "physiological" hypothesis, involving a geometrical rate of increase with age of the force of mortality, has not been improved upon. Owing to the impossibility of following out entrants at *any* age until death under conditions such that no factor of mortality save those inherent in the physiological make-up shall intervene, the validity of the hypothesis for human mortality cannot be adequately tested. Modifications of the original formula having the intention of taking account of these physiologically extraneous factors, such as Makeham's first modification, tacitly assume an improbable state of affairs, such as an environmental factor of mortality independent of age; the justification of these formulae is purely pragmatic. Given two Orders of Dying-out one of which is adequately and the other inadequately graduated by the textbook Makeham-Gompertz "law," no conclusion as to biological or environmental difference can safely be drawn. In particular we cannot separate "nurture" and "nature" into the two factors of the formula. That, upon human data, the validity of physiological hypotheses can ever be satisfactorily tested, is unlikely. The least unpromising material would be afforded by the Order of Dying-out of a select community, such as the "administrative" class of the home civil service. The faint practical interest of such an inquiry for those in a position to have access to data holds out small hope that it will ever be pursued.

[1] If $f_1(x)$ and $f_2(x)$ are *known* functions of x, we can determine the value of μ for which

$$S \frac{[f_1(x) - f_2(\mu x)]^2}{f_1(x)}$$

is a minimum.

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Passing to the "laws" of mortality describing the survivorship of other animals than man, we find that we are in the infancy of the subject. I believe it is literally correct to say that Raymond Pearl and—in a limited sense—my colleagues and I are pioneers. The conclusions that can be drawn from these first researches are scanty. So far as appears, having regard to the scale of the observations, I see no reason to think that any more complex formulation of a physiological "law" would describe the observed facts better than Gompertz's century-old simple formula. I also find no proof of essential diversity in Order of Dying-out between mice and flies. So far as the one practically important result embedded in this discursive paper is concerned, viz. a test of the truth or falsehood of the hypothesis that in the analysis of the epidemic data physiological age influence is negligible, I think I have demonstrated the influence to be really negligible. That finding of course simplifies our treatment of the epidemic problem.

The general biological problem is, however, left in an unsatisfactory state, unless Pearl's brilliant researches, as I hope may happen, stimulate others to imitate him. It seems, indeed, strange that it is only with respect to a few hundreds of mice and a few thousands of flies that Orders of Dying-out have been studied. I must suppose that the information is really not available with respect to blood stock in horses. I hope that, in time, my colleagues and myself may accumulate sufficient data for normal mice to extend the present tentative conclusions, but we have other work to do. I fear that to collect and analyse such data is too simple and unexciting a task to attract young "researchers," while when one has reached the age at which the grass-hopper becomes a burden, even the relatively exiguous life-span of the mouse is a consideration, so that veterans independent of Research Fellowships will not attempt it.

Whether, in our existing state of ignorance of herd physiology and biology, many deductions from "crucial" experiments upon laboratory animals are not as futile as the speculative philology and etymology of the days of Horne-Tooke, is an interesting question[1].

APPENDIX.

Note on Terminology.

It is usual to represent the number surviving to the beginning of the xth time-unit by l_x, and the ratio l_{x+1} to l_x by p_x; $1 - p_x$ is represented by q_x. The differential coefficient of the logarithm of l_x with respect to x taken negatively, viz. $-\frac{dl_x}{dx}.1/l_x$, is called *the Force of Mortality* and represented by the symbol μ_x.

If this is an integrable function of x, when its value is known, we know $\log l_x$ and therefore l_x. All investigations of "laws" of mortality, since Gom-

[1] I am indebted to my colleague Miss Newbold for valuable criticism and verification and to Mrs Wallace and Mr Martin for laborious computations.

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pertz's time, have started from some assumption as to the form of μ_x and all of them are included in the expression:

$$\mu_x = A + \underset{i}{S}\,[B_i \phi_i(x)] + \underset{j}{S}\,[c_j e^{d_j \phi_j(x)}].$$

Gompertz's first (1825) "law" made $A = 0$, $\underset{i}{S}\,[B\phi_i(x)] = 0$, $j = 1$, $\phi_j(x) = x$.

Makeham's first (1860) modification of this made $A \neq 0$.

Makeham's second modification (1889) made $\underset{i}{S}\,[B_i \phi_i(x)] = Bx$.

Other modifications have been either putting $i \neq 1$ and $\phi_i(x)$ a polynomial in x, or $j \neq 1$ and $\phi_j(x)$ a polynomial in x.

If, as in Thiele's form, it is desired to bring into the "law" a symmetrical exponential function, such as the "normal" function, one gets over the difficulty that the indefinite integral of the normal function cannot be evaluated, by noting the fact that, by definition:—

$$\log(l_x/l_0) = \int_x^0 \mu_x \, dx,$$

so that

$$\log(l_x/l_{x+1}) = -\log p_x = \int_x^{x+1} \mu_x \, dx.$$

It may be added that in all English textbooks what I have termed e^{dx} is written c^x and that when one passes from the logarithm of l_x to l_x the letters used for the constants in, *e.g.*, Gompertz's first "law" are k, g and c, *i.e.* $l_x = kg^{c^x}$.

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