and a fourth is considering the adoption of this procedure; in two other instances a state board is given the responsibility for patent administration; patent policies and problems in thirteen other educational institutions are under the jurisdiction of faculty committees and/or governing boards. Twelve schools have not as yet organized or have not felt the need of formulating a definite patent policy. Thus about two thirds of the institutions investigated have a definite patent policy, and these include the institutions which receive the greatest support for research from industry. There is apparently no difference in the actual practice of state-supported and privately endowed institutions. Nearly as many have patent matters administered through research foundations, or the Research Corporation of New York, as through institutional governing boards.

Those universities and colleges which have definite patent policies are of the opinion that both the social and economic welfare of the public are being enhanced by their methods of handling patents and of encouraging creative activity of their staff members. Unexploitative commercialization of patents is of definite value to the public. Earnings from patents are used to reward the inventor and to support research.

The procedure of utilizing the personnel and equipment of educational institutions in cooperative research with industry is sometimes looked upon with doubt and disapproval. Colleges and universities often are prejudiced against cooperative research because of the legal and administrative problems involved, precedent and policy of governing boards and moral and ethical principles. On the other hand, industry is often skeptical of cooperative research on the grounds that educational institutions are too unbusinesslike and too theoretical in their research activities. The initiative in bridging the gap between the two groups can be taken by colleges and universities in formulating and administering patent policies designed to remove the difficulties and misunderstandings retarding cooperation between industry and educational institutions.

The fact that the income accruing to educational institutions from patents is insignificant indicates that the incentive for discovery and invention in colleges and universities is not financial reward but is involved in the fair and equitable recognition of creative genius and of aid given by industry to research. Scientific research is the main concern of higher education with reward to the institution and inventor as a secondary consideration. Inventive ability is placed on the same level as good teaching and good relationship with the public, and the creation of new scientific knowledge is recognized and rewarded. Industries cooperating with educational institutions find that a considerable number of colleges and universities through a sound patent policy are in a position to protect the findings of the research laboratory, while encouraging creative talent and reward for accomplishment.

PHYSIOLOGICAL ALTERATIONS AS THE CAUSE OF SENILE DEBILITY AND SENILE MORTALITY[1]

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SENESCENCE is characterized by two outstanding manifestations increasing debility and increasing death rate. Of the two, debility seems to cause the greater concern to the average individual. The data discussed in this paper indicate that both these manifestations of senescence may result from the same physiological alterations.

When the logarithm of the probability of death (log P_t) is plotted against the age of the individual (t) a straight line curve after the age of 35 is obtained. This may be expressed by the equation:

$$P_t = P_o\, e^{kt} \qquad (1)$$

where k is a positive constant and P_o is the (extrapolated) probability of death at the time of birth. This is another way of expressing the law of Gompertz,[2] which was published in 1825. For total deaths in 1936 from all causes k is equal to 0.078, which means that the probability of death increases 8.1 per cent. each year in a manner analogous to the accumulation of compound interest.

Most of the diseases[3] (namely, groups A and B in

[1] This investigation has been aided by grants from the Josiah Macy, Jr. Foundation and the John and Mary Markle Foundation.

[2] B. Gompertz, *Phil. Trans. Roy. Soc. London*, 1825. For a discussion of attempts to interpret the biological significance of this and other equations, see Bernstein, *Symp. Quart. Biol.*, 2: 209, 1934. See also A. Putter, *Naturwissenschaften*, 8: 201, 1920, and K. Kupfmuller, *ibid.*, 9: 25, 1921. An equation for "loss of vitality" was used by S. Brody (*Jour. Gen. Physiol.*, 6: 245, 1923). "Vitality" was the reciprocal of "Mortality," so that k was negative. This was applied to egg-laying and to healing of wounds.

[3] E. B. Nathan (*Trans. of the Faculty of Actuaries*, 10: 45, 1924) stated that diseases can be classified according to whether or not they follow Gompertz's law or Makeham's modification of that law. Our data, from a different country and decade, are in general agreement with Nathan's in that the following diseases do not obey equation (1): accidents, tuberculosis, infancy, childbirth, congenital malformations and chronic alcoholism. The reasons are obvious, except for tuberculosis and alcoholism.

TABLE 1*

GROUP A. DISEASES WHICH OBEY EQUATION (1) WITH A LOW VALUE OF k

Cause of death	k
Infectious diseases (except T.B.)	0.044
Digestive diseases	0.048
Lobar pneumonia	0.046
Diseases of the nervous system (not including cerebral hemorrhage, embolism or hemiplegia)	0.046
Respiratory diseases (except T.B. and pneumonia)	0.053
Goiter, pellagra, arthritis, etc.	0.053
Diseases of skin and bones	0.046
Average	**0.048**

GROUP B. DISEASES WHICH OBEY EQUATION (1) WITH A HIGH VALUE OF k. THE VALUES IN PARENTHESES ARE LESS CONSTANT THAN THE OTHERS

Cause of death	k
Cerebral hemorrhage	0.104
Chronic nephritis	0.101
Cerebral embolism and thrombosis	0.106
Hemiplegia and unspecified paralysis	0.108
Chronic myocarditis and myocardial degeneration	0.120
Arteriosclerosis	0.145
Miscellaneous and unspecified heart disease	0.095
Chronic endocarditis	(0.085)
Coronary arteries	(0.106)
Angina pectoris	(0.106)
Average	**0.108**

GROUP C. CAUSES OF DEATH WHICH INCREASE WITH AGE BUT HAVE CHANGING VALUES OF k

	Cause of death	k in middle age	k in old age
Decreasing k	Cancer	0.13	0
	Diabetes mellitus	0.14	−0.04
Increasing k	Bronchopneumonia	0.06	0.11
	Trauma from falling (not mechanical)	0.06	0.13
	Genito-urinary diseases except chronic nephritis and prostate	0.04	0.08

* These values of k were calculated from the mortality data in the United States in 1936 (Bureau of Census, Special Reports, Vol. 5, No. 41, page 303). The causes of death are those of the International List.

Table 1) have death rates which increase with age according to this equation after the age of 30. These may be divided into two groups: Diseases in group A have a value of k equal to about 0.048. The death probability from any of these diseases increases about 5 per cent. each year. The other group (B) consists of diseases having a much higher value of k, namely 0.105, which means that the death probability from each disease in this group increases about 11 per cent. each year. Although this classification was made entirely on the basis of the k values (slopes of the curves), it will be seen that group B is composed essentially of vascular diseases. These cause over 50 per cent. of the deaths after the age of 30.

Groups A and B are distinct from each other with no gradation between,[4] although there are some irregular diseases (such as cancer, diabetes, bronchopneumonia, etc.) which increase with age but have a variable k. The few remaining causes of death[3] are not correlated with senescence.

In Table 1 it will be noted that although the diseases in group A are widely diversified, nevertheless, their lethalities increase at nearly identical rates (k values). This suggests that the lethality of each of these diseases is dependent upon a common physiological state or function which changes with age. This function, designated by the letter Q, may be defined by the equation:

$$Q_t = Q_o e^{k_Q t} \tag{2}$$

where Q_t is the value of Q at age t, and k_Q (the constant for the group A diseases) is equal to 0.048, corresponding to a 5 per cent. increase in the value of Q each year.

The change in Q may be a single physiological alteration, or it may be the resultant of several alterations. There are indications (from "case mortality" data) that the change in Q affects mortality by increasing the death rate when disease is present, rather than by increasing the tendency to become diseased. This is an important point which needs thorough investigation. A study of the physiology of death should help to clarify the problem.

If we assume[5] that the change in the Q function affects deaths from diseases in group B as well as group A, then the faster increase in death probability of the vascular diseases may be ascribed to the alteration of a second physiological function (or composite function) R, according to the equations:

$$Q_t R_t = Q_o R_o e^{(k_Q + k_R)t} = Q_o R_o e^{0.108t} \tag{3}$$

$$R_t = R_o e^{k_R t} = R_o e^{0.06t} \tag{4}$$

The nature of this R function is also unknown, although it may very well be some property of the vascular system such as the arterial distensibility or capillary permeability. It has been suggested that the faster change of the vascular diseases may be due merely to a greater action of the Q function. This would merely alter Q_o but not k. However, it is possible that R is a property which gives the Q function a greater opportunity to exert its lethal action.

Objection might be raised to the use of the U. S. Mortality Statistics on the ground that many of the diagnoses of the cause of death may be wrong; first, because of the presence of several pathological conditions in a single individual; second, because the

[4] Except for diseases of the blood-forming organs ($k = 0.062$).

[5] This assumption is partly warranted by the fact that if the increasing death rate in group B were due to the alteration of a single function this alteration would reach such enormous values that it would probably have been already discovered. It would increase 650 fold between the ages of 30 and 90, whereas the Q and R functions increase 18 and 32 fold, respectively, during this period. Although the vascular deaths might be controlled by two or more alterations not including the Q function, this seems improbable for two reasons: First, if the Q function controls deaths from such a variety of unrelated causes as are found in group A, it would be strange if the vascular diseases were unaffected by it. Second, a progressive change which obeys an exponential equation such as equation (2) is rather unusual and, although the evidence indicates there are at least two such changes, the probability of three or more simultaneous exponential changes is small.

terminal disease may not indicate the underlying disease; and third, the underlying disease may in itself result from some pathological condition. Thus an individual with arteriosclerosis might develop nephritis and die with terminal pneumonia. We have taken this into account and feel that these inaccuracies do not alter our general conclusions. Whatever the mistakes in diagnosis may be (within group A or group B) the values of k will not be affected provided the same mistakes are made at all ages. The consistency within each group indicates that this is the case and that cross-diagnosis between the groups does not play a large role (except after the age of 80).

Senile debility is not as easily measured as the death rate is. However, a composite measure of both physical and mental debility is given by the data on male unemployment. We admit that this is only a crude measure of debility, but feel that most of the complicating influences affect the value of P_o rather than the value or constancy of k. It was found that in 1930 the probability of male unemployment followed equation (1) after the age of 50, with k equal to 0.115 (and approximated this equation from 40 years up). The data in 1920 are consistent with the 1930 findings. These values are for unemployment from all causes. Data on "Unemployment Class C" (admittedly unable to work) in six northeastern states also follow equation (1) but with a lower value of k (0.032). The unwillingness of older people to admit disability may make this value less reliable. Nevertheless, the agreement with equation (1) is significant.

Thus there is a statistical correlation between senile debility and senile death rate. This suggests that the progressive debility in old age is caused by an alteration of the same (Q and R) functions which control the death rate.

This brief report is preliminary to a more detailed treatment of the data which will be submitted to another journal. The author hopes that these findings may help define the problem of senescence, and that the equations may serve as a tool in determining whether or not observed changes with age are primarily correlated with senescence. This is determined as follows: The logarithms of the observed values are plotted against numerical age. If the change is correlated with senescence, a straight line curve will be found in middle life and old age. The slope of the line gives the value of k which can be compared with $k_Q = 0.048$ and $k_R = 0.06$ (on a natural logarithm basis). Experimental prolongation of life of animals may be considered to affect senescence only if there is a decrease in the value of k for known causes of death of mature adults, rather than a reduction in deaths through a lowering of the value of P_o for certain diseases. The author feels that superficial characteristics, such as the condition of the hair and skin, are unsatisfactory criteria of senility. More knowledge concerning the physiology of death and the physiology of arteries is needed in order to clarify the mechanism of senescence.

These findings also show that for the most part no disease can be called a "senile disease" more than another in the same group. The high death rate in old age is the result of changes which make us succumb more readily to all diseases, although the change is faster for the vascular diseases. Nearly four deaths out of five after the age of 30 are due, not to a greater prevalence of disease, but rather to the change in the Q and R functions which increases the death rate from the same diseases which affect young people.

The theory that senescence results from a random accumulation of degenerative changes is not supported by these findings. A random accumulation should follow a linear equation rather than the exponential equations (2) to (4). It is perhaps correct to say that there is an accumulation of degenerative changes, but that the process follows a definite mechanism such that the rate of change at any age depends upon the amount of accumulated change. Why this mechanism should be followed remains to be determined.

As was mentioned above, there seems to be more popular concern about senile debility than about senile death rate. Should we be correct in our assumption that these are both caused by the same physiological alterations, then it is to be expected that any mitigation of these alterations will prolong the vigor of youth as well as delay death in old age. This would mean a prolonged middle life with a relatively smaller portion of life spent in dependency.

OBITUARY

LETA S. HOLLINGWORTH

DR. LETA S. HOLLINGWORTH, member of the staff of Teachers College, Columbia University, since 1916 and a world-renowned authority on the psychology and education of exceptional children, died on November 27, in the Columbia-Presbyterian Medical Center in New York City. She was the wife of Dr. H. L. Hollingworth, professor of psychology in Barnard College, Columbia University.

Professor Leta S. Hollingworth was born in Chadron, Nebraska, the daughter of John G. and Margaret D. Stetter. She received her B.A. degree from the University of Nebraska in 1906, in the same class with her husband. Together they were honored for distinguished contributions to science and education by their alma mater, which conferred on them the degree of doctor of laws in June, 1938.

After her graduation Mrs. Hollingworth taught