TRANSACTIONS OF SOCIETY OF ACTUARIES
1967 VOL. 19 PT. 2 NO. 55

MORTALITY TRENDS AND PROJECTIONS

A. What light does recent thinking in biology and medicine throw on the prospects for greater longevity?
B. What can be inferred about the prospects of longevity in the United States and Canada from recent changes in population mortality in North America, western Europe, and Australia?
C. What factors affecting mortality should be considered in weighing future mortality trends?
D. What methods for projecting mortality rates have been used in (*a*) population forecasts, (*b*) social insurance projections, and (*c*) projections of annuitant mortality?

CHAIRMAN EDWARD A. LEW: In recent years we have not had an opportunity to take stock of our growing body of knowledge about mortality. This session is intended to take a hard look at our current understanding of the principal factors likely to influence the future course of mortality. It is fitting that we do so at this time since for more than a decade there have been little change in male death rates and only a small decline in female death rates, not only in the United States but in many parts of the Western world where conditions are not dissimilar to those in this country.

We could begin with the fact that the United States has for many years had a relatively poor longevity record for males in the age range from 45 to 64. This needs to be viewed first in relation to the recent leveling-out of male death rates and then in relation to the outlook for lower mortality stemming from discoveries in biophysics, bio-engineering, genetics, and research in medicine.

There is a great deal of wishful thinking about the increases in longevity that might be brought about by new scientific miracles. You have all seen statements that the span of human life will be extended to 120 or 150 years in the not-too-distant future. Such optimistic visions have been seen in an increasingly unfavorable urban environment characterized by growing air and water pollution, psychological stresses that lead to drug addiction, loss of a sense of community that is reflected in rising crime, and so on. The balance between the forces operating to raise mortality rates and those tending to lower them is by no means clear.

We will now try to explore these conflicting developments with our panel of experts.

For an introduction to the biological background of these problems,

we are fortunate to have with us a distinguished scientist, noted for his broad and imaginative views of the aging process. It is my privilege to present to you Dr. Bernard L. Strehler, currently professor of biology at the University of Southern California and for some years prior to that chief of the gerontology branch of the National Institutes of Health. Dr. Strehler is the author of numerous papers on aging, and I would heartily recommend to you his very readable book entitled *Time, Cells, and Aging*. He is currently endeavoring to organize an international gerontological quinquennium, which is intended to provide a systematic attack of the origins of senescence.

DR. BERNARD L. STREHLER: It is pleasant to return to Chicago, where I spent three years, from 1953 to 1956, as an assistant professor of biochemistry at the University of Chicago. It is more eventful, however, to speak to you, the professional descendants of a man who in 1825 produced a very important generalization about the nature of the force of mortality which has been of particular interest to those concerned with the biological origins of aging. I refer to Benjamin Gompertz, who, as all of you know, was an actuary. Gompertz was the first to formulate the law of mortality that relates the probability of death to attained age as $R_m = R_0 e^{at}$. Thus the probability of death for human beings doubles about every eight or nine years in the age range from 35 to 90.

The relationship between actuaries and biologists is a tenuous one. It is only occasionally that biologists resort to data produced by actuaries in order to test one or another hypothesis about senescence. I believe, nevertheless, that the phenomena which are of importance to you in determining premium rates for various kinds of insurance and annuity contracts can be understood more clearly only by reference to the underlying biological process of aging—the rate of which differs from one species to another.

It was in 1825 that Gompertz theorized about the rate of aging increasing exponentially with age. The Galapagos turtle, a species that can live several hundred years, was probably alive at that time. Recently through carbon dating, that is, by measuring the radioactivity in the carbon of their shells, Dr. W. Reichart found that such animals can survive well beyond 175 years of age. This would make them fairly young turtles at the time Gompertz discovered his law.

It may surprise you to learn that the Gompertz equation applies to the fruit fly, Drosophila, which has a mean life span of about thirty days, no less than to human beings, mice, and rats. Moreover, the internal clocks, that is, the factors which control the aging mechanism, are ap-

parently somewhat different for different species. The nature of the process of aging has, of course, most important implications for your work. Indeed, when I casually observed last evening that it might be possible to increase the average life expectancy by five or six years through relatively simple measures, one of the actuaries at the table was impelled to say, "That would be terrible. It would ruin the country if this happened too quickly, merely because of the effect it would have on pension plans."

I strongly suspect that each of us here would not be too unhappy if the male life expectancy were increased by five or six years, but someone's bull would, of course, be gored. I would like to explore with you some of the measures for achieving such an extension in life expectancy.

It is possible, of course, that we will find ways to patch and repair and replace and change and substitute, first, kidneys, then hearts, then—well, use your imagination! I would draw the line at the replacement of brain, because this could produce a person who might no longer have a sense of identity. This brings me to emphasize the motto of the Gerontological Society, "To add life to years, not just years to life." It also reminds me of the experience of a physician who was visiting an elderly patient and noted that the patient was in particularly fine physical condition.

"You know," the doctor said, "I'm making a study of the inheritance of longevity. Could you tell me how old your parents were when they died?"

The septuagenarian replied, "Well, my mother died at 97, but my father is 102 and is still alive."

"That's remarkable. Where is he?" the physician asked.

"He's over there in the Happy Valley rest home for the retired." The doctor responded, "I think I'll go over and look at him."

So over he went, but on arrival he found that the elderly gentleman was not around. He accordingly inquired about him and was told that he was away attending his father's wedding.

"How old is the father?" asked the surprised man of medicine. "137, I believe," was the answer.

"137! Don't you think it's silly for a man of 137 to get married again?"

"Well, yes," replied the attendant, "but he had to!"

Before getting down to the mechanisms which we believe are involved in aging, I would like to review with you the insights that we owe to Benjamin Gompertz.

Figure 1 represents a typical Gompertz plot of the log of the probability of death against age. The figures charted represent the mortality rate of white males in the United States population during 1939–41. I would call your attention to the middle segment of the curve, which is virtually a

straight line. This middle portion of the curve is of particular interest to biological scientists. When we encounter so clear-cut a linear relationship, we start looking for a plausible explanation.

The problem is to fix on a likely process that generates within a biological system an exponentially increasing probability of a reaction, in this case the death of an individual. We might first ask the question whether the common biological functions tend to decrease at an ex-

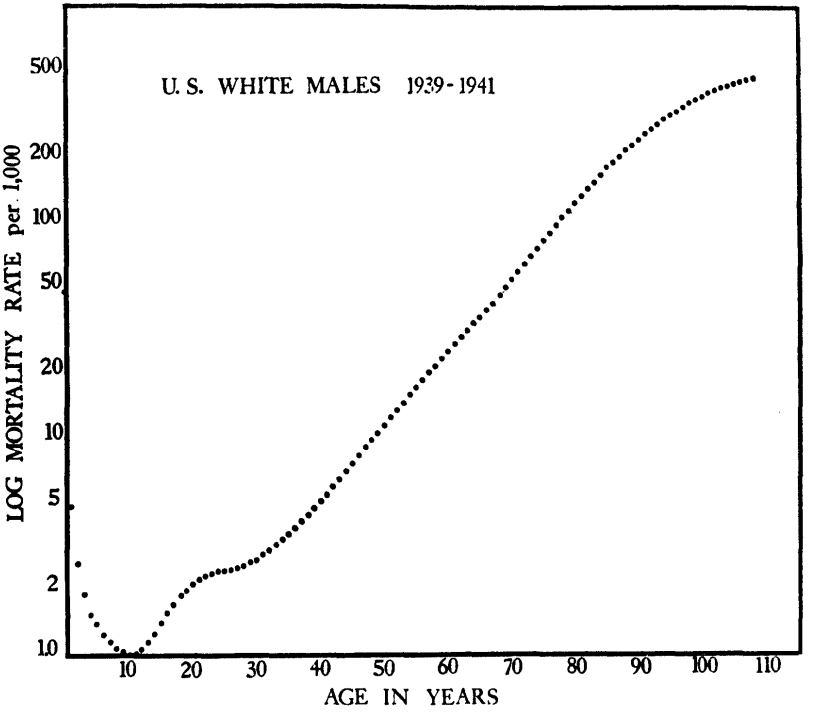

FIG. 1.—Log mortality rate per 1,000 v. age in years (United States white males, 1939–41).

ponentially accelerating rate. The answer is no! If we look at some physiological functions which have been studied intensively—for instance, the amount of physical work a man can do, the speed of nerve conduction, the amount of acid secreted by the gastric mucosa, the maximum rate at which one can breathe, the cardiac output, or the maximum kidney filtration rate—we find to our surprise (and delight?) that each year we lose only about 1 per cent of our youthful capability, such as we might have had, say, at age 30 (see Fig. 2). Yet by the time we have

lost about 40 per cent of a function, such as vital capacity, the probability of death will have increased almost a thousand-fold. This means that a 40 per cent decline in work capability is associated with a thousand-fold increase in the chance of dying. This surprising fact suggested an analogy to certain physicochemical laws that also involve the exponential function.

I would like to discuss this analogy briefly with you. It relates to the so-called Arrhenius relationship, which was formalized by Maxwell and

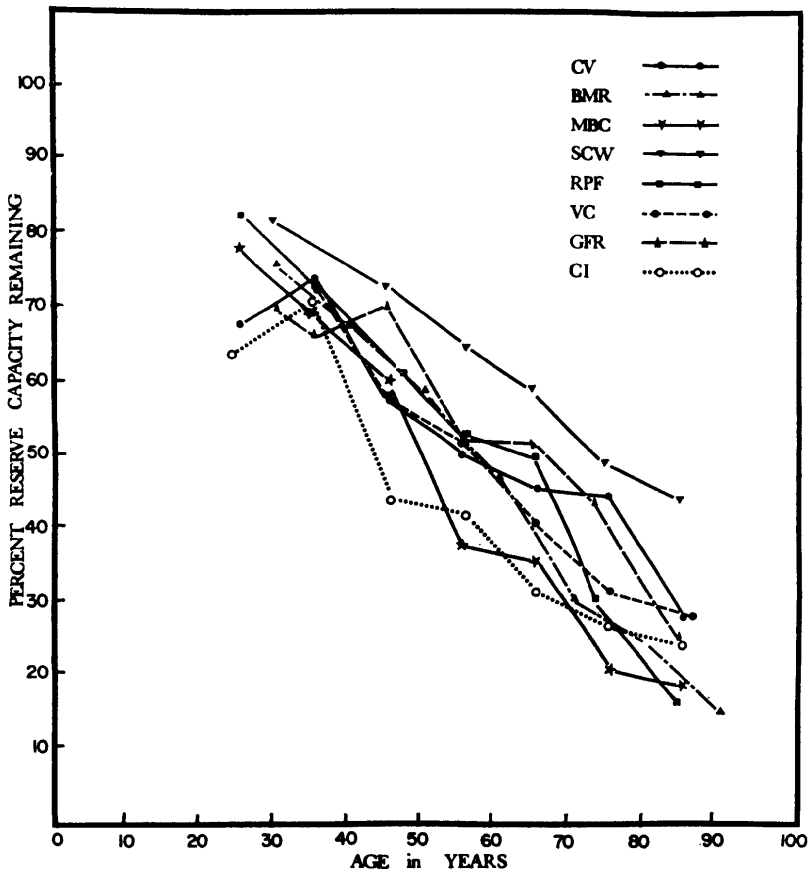

FIG. 2.—Per cent reserve capacity v. age. Designations are as follows:

CV = Conduction velocity	RPF = Renal plasma flow
BMR = Basic metabolic rate	VC = Vital capacity
MBC = Maximal breathing capacity	GFR = Glomerular filtration rate
SCW = Standard cell water	CI = Cardiac index

Boltzmann. Arrhenius postulated that a chemical reaction will take place only when existing bonds are broken. For this to occur, the bonds must be stretched, which will happen when there is an accidental accumulation of sufficient heat energy in a given molecule. Random collisions among gas molecules (or molecules in a liquid phase) will produce a certain proportion of molecules that have energy very much in excess of the average. Some molecules, of course, will have less than the average, but, at a given temperature, there will be a statistical distribution of energies determined by the Maxwell-Boltzmann equation $n/n_t = Ke - E/xT$. Of particular interest is the number of molecules that have an energy greater than that required to break potentially reactive bonds.

What is the relevance of this model to the Gompertz equation? Imagine a chemical reaction in which the bond strength becomes progressively weaker with the passage of time. In such a system an increasingly greater proportion of molecules will have an opportunity to react as time goes by. In fact, the rate of reaction (proportional to the number of molecules that acquire the minimum amount of energy necessary to break the bond) will increase exponentially with time (reflecting the exponential term in the Maxwell-Boltzmann equation). If we ask, "How many molecules have energy x, and how many have energy $(x - t_1)$, $(x - t_{11})$, and $(x - t_n)$?" we find that the smaller the energy, the larger will be the number of molecules that possess that energy, the number of molecules with lower energy increasing exponentially.

Obviously we can translate this model of declining activation energy to the decline in physiological functions that occurs with age. Bond strength is thus analogous to youthful vigor, and the challenges to existence are analogous to kinetic energy in the molecular model. Accordingly, if an individual's ability to resist challenges to existence decreases in a linear fashion, while the challenges are distributed exponentially (like the kinetic energy of molecules in a gas), then the linearly decreasing ability to withstand challenges will generate an exponential increase in the probability of death. This reasoning was developed independently by Dr. Albert Mildvan and me and by Dr. George Sacher of the Argonne National Laboratories in Chicago.

One of the interesting predictions from the Gompertz model is that the Gompertz slope (α) should be smaller but the Gompertz intercept (R_0) should be higher in an unfavorable environment. We predicted that the plot of log R_0 against α should display an inverse correlation and that the slope of this line should represent the reciprocal of the loss of function per year.

In order to test this prediction, we took the United Nations *Demograph-*

ic Year Book for 1955, looked up the age-specific mortality rates for different countries, and plotted on semilog paper the appropriate α's and R_0's, obtaining the results shown in Figure 3.

Each point in the chart represents the Gompertz constants for a particular country. You will note first that the curve produced is essentially a straight line. Although there is some scatter about the line, there is no suggestion of any significant curvature. Second, the slope of the line happens to be about 100, which indicates that the amount of function

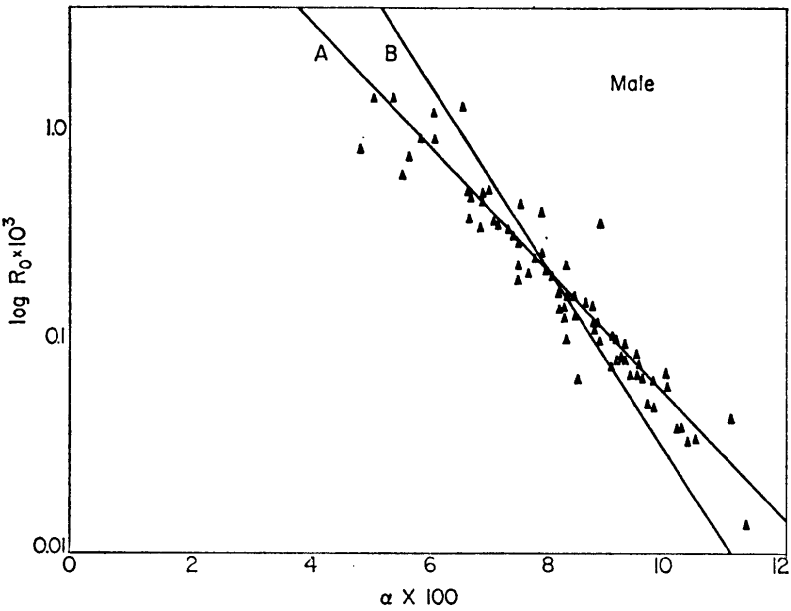

FIG. 3.—Log R_0 v. alpha

lost is about 1 per cent per year. This finding agrees with the results plotted in Figure 2, where the loss of function ranges from 0.5 to 1.5 per cent per year.

It is gratifying to be able to take mortality data from the United Nations *Year Book*, to deduce therefrom an index of the loss of function per year, and to find that the value agrees closely with the rate of functional loss as measured in laboratories. While these findings do not prove the reality of the model any more than a limited set of experiments could, they do suggest that there is some validity to the concepts elaborated.

At first glance it may be surprising that the less favorable the environment the longer it takes for the mortality rate to double. Such a con-

sequence could not be deduced from models which imply that environment kills by accumulated insults. It is, however, to be expected from models in which death results from random high-energy disruptive events.

By way of summarizing a great deal of existing information, I would say that it is not yet possible to explain fully the decline in physiological function with age, the real key to the aging process. It is clear that, if function did not change with time, the probability of dying would be a constant. Machines age, deteriorate, and get into trouble because they are not in what is called a "steady state." Aging systems do not replace and replenish completely the materials of which they are made at the same rate that these materials deteriorate. The following questions thus arise: What materials do decay? What mechanism underlies such decay? What is the genetic program that produces such deterioration in animals and human beings, and what distinguishes man from shorter-lived or longer-lived species?

A few observations are appropriate as an introduction to these questions. First, living things that continue to grow do not exhibit a clear-cut increase in age-specific mortality. A case in point is the plaice, a North Sea fish. The male of this species stops growing at about three years of age and shows a striking increase in age-specific mortality from this point on, whereas the female continues to grow and does not manifest a measurable increase in age-specific mortality. This suggested to an English physician by the name of Bidder that there might be a causal relationship between size limitation and the commencement of the aging process. Size limitation is adaptively useful; if we were twice as large as we are, we would have great difficulty in supporting our structure, assuming that our proportions were maintained. In order to be larger, we would have to invest more mass in bone and muscle and less in brains. On the other hand, if we were smaller and wanted to continue to enjoy the complexity of function which our nervous system provides, we might have to settle for a clumsy form with a disproportionately large head.

One of the key ideas in biology contributed in 1880 by the German biologist-essayist Weismann propounds a causal relationship between the process of cell specialization, the deceleration of growth, and the subsequent deterioration of highly evolved systems.

What kinds of deterioration develop in such systems and how can we try to explore this question? The systems approach to the failure of mechanical devices (whether macromachines or molecular machines) suggests an inventory of the possible mechanisms of deterioration. Such a list would include (1) hydrolysis, that is, the adding of water to important components, which results in their fragmentation; (2) denaturation of

proteins by heat and chemicals; (3) cross-linkage, the process by which different molecules in a system are accidentally bound to each other through interconnecting molecules, thus interfering with function (currently a popular theory); (4) damage to genetic material; and (5) loss with age in the ability of cells to translate specific code words in certain messages, particularly at the time when cells differentiate.

While other hypotheses have also been proposed, the real challenge, in my judgment, for the next decade at least is to sort out from the known or suspected sources of senescence those which can be validated by the application of a rigorous "systems" type of analysis. This approach has been extremely effective in business, in space programs, and in atomic research.

In this connection, you may recall that medieval philosophers had three dreams. One of them was the transmutation of elements, particularly that of the base metals into gold. This was achieved during the 1940's in the Manhattan Project. Another of the alchemists' dreams was to leave the earth and visit other celestial bodies. This dream will, I suggest, be fulfilled within the next few years, when human beings land on the moon. The third ancient aspiration was to prolong life by one or another kind of magic. We have not moved very far in this quest. Indeed, we do not even know whether this objective is possible and, if possible, whether it is desirable.

Regardless of the answer to the last question, I believe that one of the main achievements of biology during the next decade could be the unraveling and understanding of the aging process. A concrete proposal for achieving this objective has been submitted to the United States Congress in the form of an international gerontological quinquennium (ICQ). I will be glad to furnish details of this program to anyone who is interested.

In the time remaining to me I would like to explore with you two specific hypotheses related to the aging process. The first of these deals with substances called "age pigments." These substances accumulate in nondividing cells, such as those in the brain or heart, and may account for as much as 10 per cent of intracellular volume in an old individual.

Figure 4 illustrates these substances, with the bright spots being the lipofuscin age pigment. We believe that this age pigment is derived from deteriorating membranes—structures that contain both fat and protein materials. Membranes, I should add, act as barriers at the surface of cells and also make up important parts of cell interiors. Figure 5 shows the relationship between age and the amount of age pigment present in heart muscle.

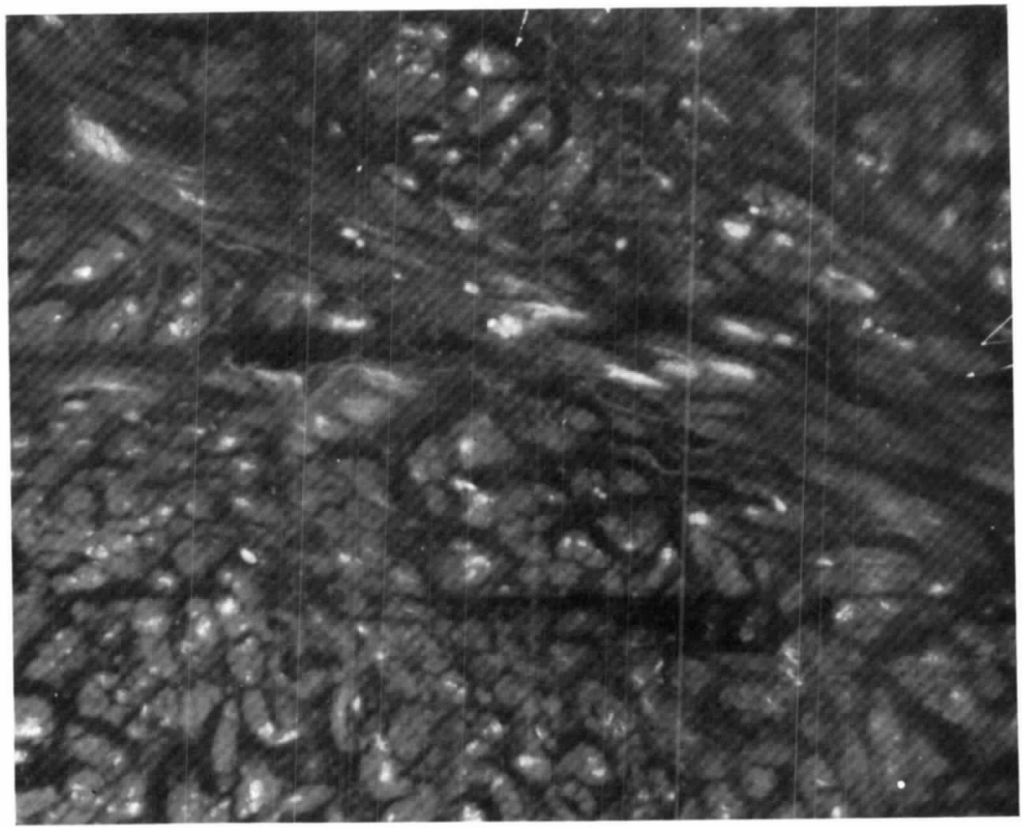

FIG. 4.—Black-and-white photograph of fluorescent age pigment

We do not know the extent to which deterioration in function accompanying the accumulation of these pigments is caused by them. The main reason for this gap in our knowledge is the scarcity of talented workers in the field; however, limited resources and lack of administrative support are also serious handicaps. A concerted effort on the part of those

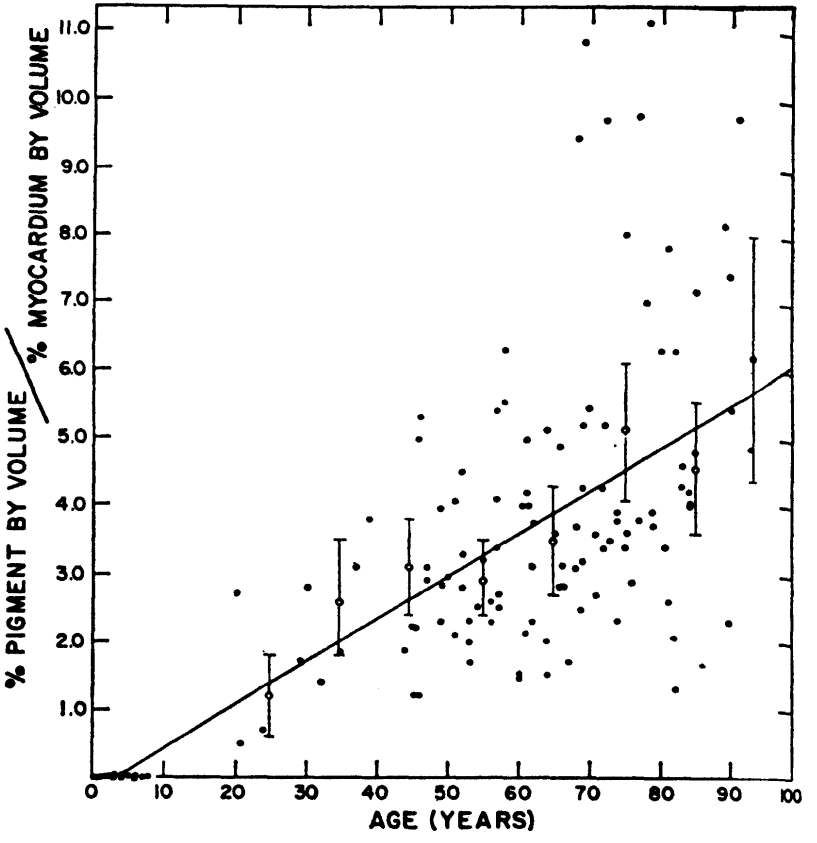

FIG. 5.—(Percent pigment by volume)/(per cent myocardium by volume) v. age. •, individual cases; o, decade averages $\pm 2\sigma$ (mean).

able to influence the allotment of human talent and material resources into this field will be necessary if real progress is to be made soon in our understanding of the phenomenon of aging.

Accumulation of age pigments takes place in cells which do not divide, that is, in cells of the heart, brain, skeletal muscle, prostate, and adrenals and in certain cells of the kidney. These pigments do not occur to any

appreciable extent in those types of cells which are continually being replenished. For instance, we generate a new layer of skin about every seven days or so, the lining of our intestines is replaced about every four days, and our blood is replenished about every 120 days. It is an ironical fact that those portions of the body which are weakest and, therefore, need replacement on a regular basis are not the important sites of aging. Surprisingly, the more solidly constructed components, such as the muscles, heart, and brain, which can last for more than a century, are those that ultimately fail and lead to senescence.

What is there about the cell which is not capable of replacing itself (the fixed postmitotic cell) that may give us a clue to its deterioration? Are there, for example, substances within such cells that are not being replaced or substances that are laid down at a particular stage of development only, perhaps even during early embryogeny? If such substances were indeed involved, it would follow that their deterioration could be one of the major sources of the deterioration of cells and organs.

A partial answer to this question is already available. Using radioactive tracer techniques, we first injected C^{14}-labeled acetate—a precursor of fatty substances—into mice at ten days of age. About two months later we injected acetate labeled with another isotope—tritium—into the same mice, and after several more months we chemically isolated from these animals various materials that had been made out of the acetate. We were able to tell from the ratio of the two isotopes in each material whether it was being regularly replenished, slowly replenished, or not replenished at all. This investigation demonstrated that there are substances within cells, particularly in the brain, the heart, and the muscle tissue, that are laid down early in life and are either not replaced later at all or are replaced only very slowly. It may very well be that the golden age pigments to which I referred earlier are the deteriorated remains of such nonreplenishing substances.

At this point, we need to ask what mechanism turns off late in life an animal's ability to make certain materials that it manufactured without difficulty at an earlier time? And how does this mechanism in a mouse differ from that in a man, keeping in mind that a mouse is mature in two to three months and old at two years, whereas man requires 40–50 times as long to run his course?

Ultimately, we have to go back to the genetic control mechanism which underlies differentiation and growth. By differentiation, I mean the process by means of which one cell type makes one group of products, while another cell type produces a different list of offspring. We are now seeking this answer in the details of the genetic code. The last decade has

been an epochal period in modern molecular genetics, because during this time it was demonstrated that the entire prescription for a human being (or a mouse or a fruitfly or a carrot) is written down in a language composed of just four letters. In other words, the directions for life are coded through the arrangement of just four letters in a sequence. Everything that is required to specify an individual is written in a long string of these four letters arranged in a particular order. The translation of the messages made up of these strings into the useful working parts (proteins) of which each living creature is composed was one of the most elegant puzzles to confront scientists. This puzzle has now been almost completely deciphered.

The mechanism of translation consists of several elements. First, a copy of a part of the total message (DNA) is made; this part is called M-RNA. Only certain portions of the total library are copied for each cell type, and different portions are copied for different kinds of cells. In the process of translation, three consecutive letters in the code signify a particular amino acid, the next three letters stand for another amino acid, and so on.

This translation process includes a submicroscopic machine, called a ribosome, which ratchets down three places at a time, reading one word after another, so that a chain (which follows the specifications of the original code) is manufactured and finally released as a completed protein. Such proteins are the central functional working elements of which all life is made.

In the translation process a group of somewhat smaller molecules (t-RNA's) are assembled along the message in a pattern determined by the M-RNA. These molecules at their free ends are specifically bound, in turn, to materials called amino acids. Thus the translation (production of protein) is completed by the clipping-together of the amino acids in a sequence determined ultimately by the sequence in the total message (DNA).

How is all this related to aging? Well, it needs first to be pointed out that the number of possible three-letter words is 64 but the number of building blocks in proteins is only 20. Therefore, each kind of building block (amino acid) can, on the average, be written in three different ways. Now, suppose that the ability to read certain of these words in the genetic code were lost as a cell differentiated. In such event, the messages involving "lost words" could no longer be decoded, or, in biological terms, the cell would not be able to make the proteins represented by the lost words.

During the last three years we tried to test this hypothesis. Specifical-

ly, we asked whether cells from different tissues differ in the groups of words that they can translate. Briefly, I can report that liver cells have an ability to read certain code words which immature red blood cells, called reticulocytes, cannot decode and that certain kidney cells are unable to read another word which reticulocytes and liver cells can. Accordingly, we think that we may be on the trail of reality in the particular model that we are testing. Our hypothesis states that, as differentiation occurs and the program for the development of a living organism is set into motion, there ensues a loss of ability to read certain genetic code words, which results in the loss of ability to translate messages using these words. When the ability to carry out specific protein syntheses is lost in subsequent stages of development, a gradual deterioration of the irreplaceable molecules occurs, evidenced by the accumulation of age pigments. Thus, we may grow old because we can no longer replace certain molecules when these deteriorate. Bodily functions are impaired, and the interaction between the altered psychological state of the individual and the challenges that he meets is such as to generate the Gompertz function and the actuarial statistics with which you are familiar.

CHAIRMAN LEW: After listening to Dr. Strehler, it is clear that much needs to be done to follow through on the recent insights into the process of aging. We have been remiss in not keeping up with the implications of recent developments in areas such as biophysics and genetics.

We must at the same time continue to follow mortality trends and to analyze their significance. I know of no one who has given more thought to this subject than our next speaker, Dr. Moriyama, who was for many years chief of the Mortality Analysis Section of the National Office of Vital Statistics. Currently he is director of the Office of Health Statistics Analysis. During the past few years he has given us a number of very informative papers on mortality trends which we would do well to study. His summary of the current mortality situation is a product of firsthand experience with extensive observational data and of seasoned and perceptive judgment in dealing with mortality statistics.

DR. IWAO M. MORIYAMA: When Mr. Lew asked me to examine the the mortality trends in the United States and to comment on what I see in them in terms of future mortality, it suggested to me that what is really needed is a reliable crystal ball. What will actually happen in the future is anyone's guess. Experience has shown that the projection of trends is not a pastime in which one can safely engage unless it is on a very short-run basis or is for a period far beyond the average expectancy of the pro-

fessional life of the investigator. Even in the latter case, there is always the danger of seeing current events prematurely catching up with the projections.

In 1961 it was reported[1] that the trend of the crude death rate had leveled off in the United States after a long period of rapid decline. Since the time of that report additional data have been accumulated which reinforce these previous findings. The crude death rate for the United States has now been more or less stationary during the past sixteen years—1950–66.

It may be seen from the trend of the crude death rates (Fig. 1) that the

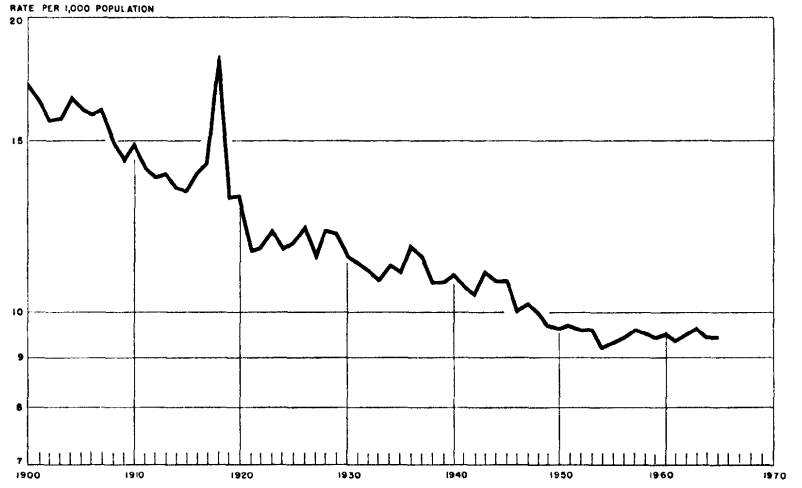

FIG. 1.—Crude death rates in the United States, 1900–1965

rate of decline has not been uniform over the entire period. Also, it is difficult to discern any well-defined series of linear trends. Part of the problem is the large annual variations in the crude death rate. Superimposed on it is the effect of the changing age composition of the population.

A large part of the variability in the crude death rate results from the frequent outbreaks of influenza. Collins and Lehmann[2] have described the influenza epidemics which occurred in twenty-six of the thirty-three

[1] I. M. Moriyama, *The Change in Mortality Trend in the United States* (United States Public Health Service Pub. No. 1,000, Series 3, No. 1 [Washington, D.C.: National Center for Health Statistics, 1961]).

[2] S. D. Collins and J. Lehmann, *Excess Deaths from Influenza and Pneumonia and from Important Chronic Diseases during Epidemic Periods, 1918–51* ("Public Health Monograph No. 10," Pub. No. 213 [Washington, D.C.: United States Public Health Service, 1953]).

major trends. The first of these is from 1900 to 1937. Then, beginning about 1938, there is an acceleration in the downward trend. Starting about 1954, a definite deceleration in the rate of decline is evident.

Both the crude death rate and the age-adjusted death rate are composite measures which are useful as over-all indexes of mortality. However, they are both weighted heavily by the population in the older ages. On the other hand, age-specific death rates are not subject to the same kind of problem and are in many ways more informative in tracing the course of mortality.

The death rates by age (Fig. 3) show a deceleration in the rate of decline for virtually every age group regardless of color and sex. This picture is now much more pronounced than it was five years ago. For certain age groups, notably those 85 and over and males 65–74, the movement of the death rates appears to be clearly upward. There is also a suggestion of an upturn in the death rates for nonwhite males in the age group 25–54.

It is pertinent to ask if these changes are real or apparent. If they are real, what is the underlying cause or causes of these changes? With regard to the first question, there seems to be little question that it is a real phenomenon. There is no evidence to suggest that it is an artifact of definition. registration completeness, errors in intercensal population estimates, or errors in the statement of age. Also, the consistency both spatially and demographically argues against its being an artifact.

With respect to the second question, our study suggests that the leveling-off of the death rates can be accounted for by the combination of two sets of factors. The first is the dramatic reduction in the death rates for diseases of infectious origin with the successive introduction and application of pneumonia serum therapy, the sulfonamides, and the antimicrobials. The accelerated decline started about 1938 and then lost its impetus in the 1950's. By that time, the mortality from the infectious diseases had reached a level at which it no longer contributed in a major way to total mortality. Even if the trend of the death rates for the infective and parasitic diseases, including pneumonia and influenza, had continued downward without interruption, this would not have accounted for all the leveling-off of the total death rate.

The long-term decline in mortality from the infectious diseases resulted in a major realignment of the principal causes of death, which uncovered a second set of factors. These factors involve the trends of mortality from the present numerically important causes of death, namely, the cardiovascular-renal diseases and the malignant neoplasms at all ages, congenital malformations through the childhood years, accidents and other violence from childhood through middle age, cirrhosis of the liver in

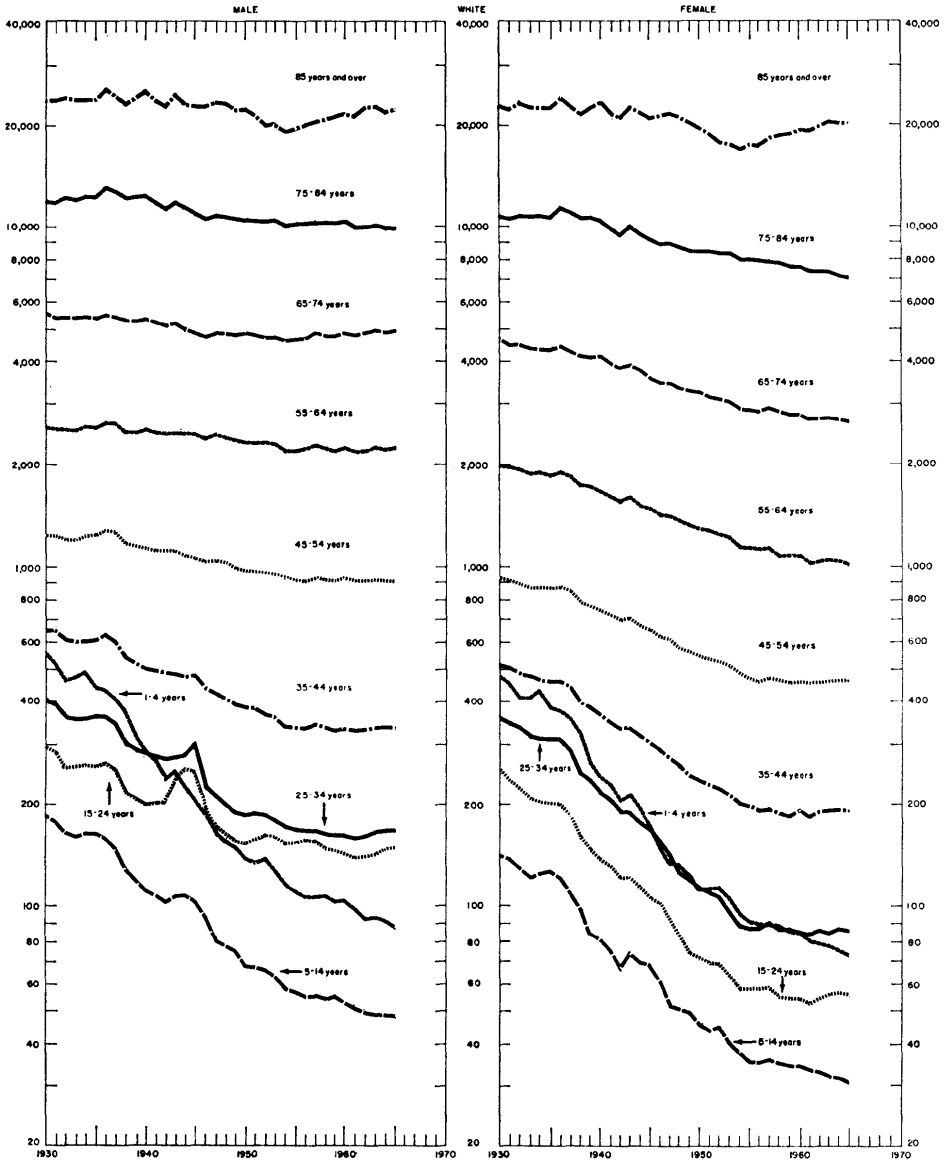

FIG. 3.—Death rates per 100,000 population in the United States, 1930–65

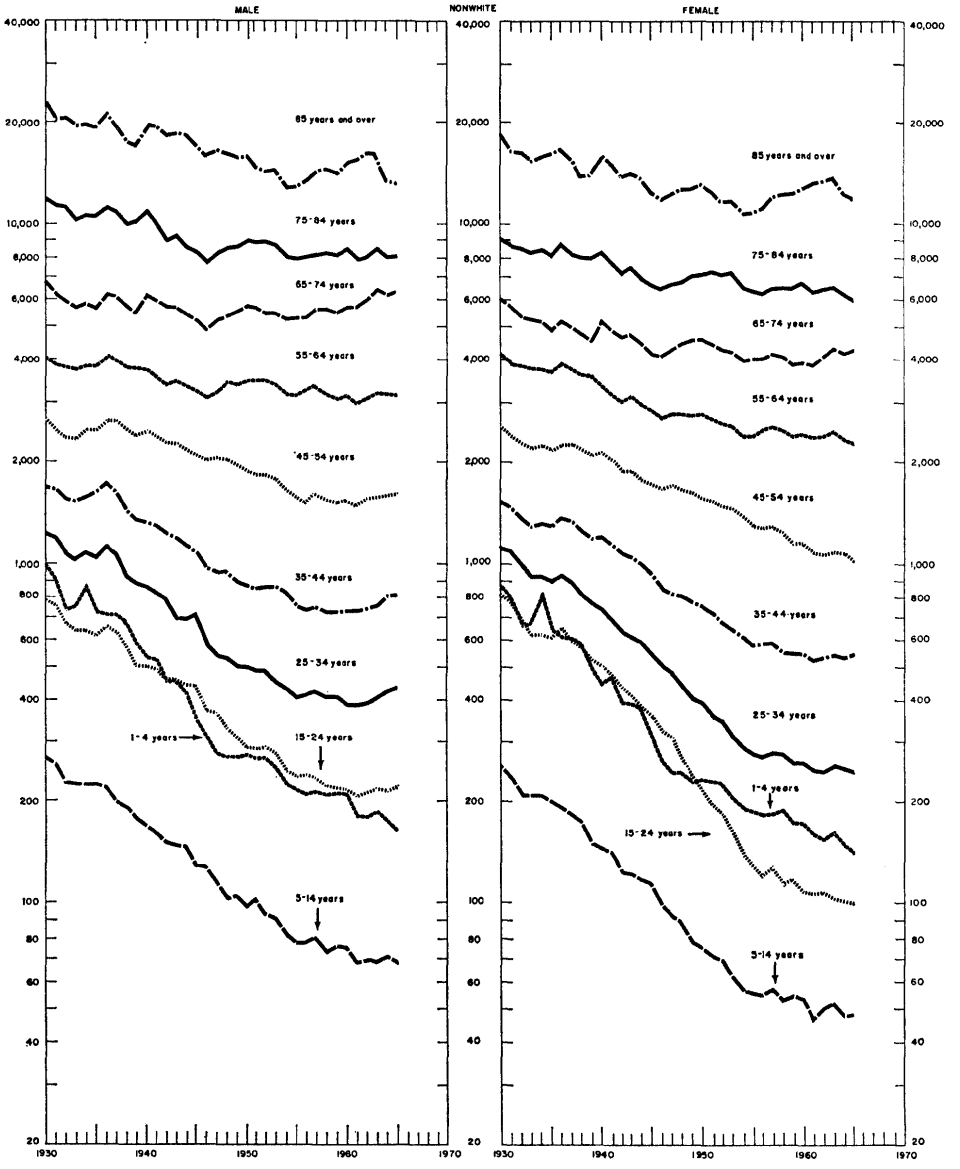

FIG. 3.—*Continued*

middle age, and diabetes mellitus from middle age into old age. Also, new problems are emerging. The dramatic upward trend of the chronic bronchopulmonary disease mortality from middle age onward seems particularly significant. The combined effect of these various trends is to slow down the rate of decline of the total death rate.

Further reductions in total mortality in the United States are possible, but substantial decreases are now getting more and more difficult to achieve. For example, even the complete elimination of deaths from all infective and parasitic diseases, including influenza and pneumonia, will not result in the reduction of total mortality of much more than 4 per cent. The complete control of accidental deaths will lower the total death rate by about 6 per cent. Cancer contributes more to total mortality—about 16 per cent. Except for the cardiovascular-renal diseases, and possibly malignant neoplasms, even the complete prevention of deaths from certain causes of death, such as infective diseases, diabetes, and accidents, will have relatively little impact on total mortality. Therefore, unless there is a major breakthrough in the control of chronic noninfective diseases, such as the cardiovascular-renal diseases and cancer, large reductions in mortality cannot be expected in the future. This was the conclusion reached on the basis of data for the years 1930–60. The experience of the past six years gives no cause to alter this conclusion. In fact, the stagnation of the death rate, which has now been evident for the past fifteen or sixteen years, strengthens the conclusion concerning the future course of mortality in the United States. With an aging population, even an upturn in mortality trend is possible.

A notable factor to consider in the study of mortality trends for the total population is that it is obviously impossible for the death rate to decline indefinitely. At some point in time, the mortality rate must level off as it reaches the irreducible minimum. What the biological irreducible minimum is for the United States population is difficult to say. However, it is obvious that the death rates by age and sex, even among the whites in the United States, are still far above the low levels established in the Scandinavian countries and in the Netherlands. For example, if the minimum death rates by age and sex observed in the Scandinavian countries and in the Netherlands in 1959 or 1960 were applied to the United States population in 1960, the resulting death rate would be more than 20 per cent lower than the recorded death rate in the United States in 1960.

Additional gains should be possible, but, as mentioned before, it will be more and more difficult to make large advances in the prevention of mortality. This problem may be illustrated in another way. It may be estimated that, in order to increase by five years the life expectancy at birth of the United States population as of 1960, it will be necessary to

reduce total mortality under age 55 by 80 per cent. The same results can be achieved by reducing mortality for the population aged 55 and over by about 50 per cent. It is obvious that similar estimated gains may be made by varying combinations of reduced mortality under and over age 55.

The life-expectancy figures raise the question of increased longevity of the United States population. There has been a significant increase in the proportion of total deaths occurring in the age group 85 and over. In 1951, 8.2 per cent of all deaths were of those 85 and over. In 1965, the corresponding percentage was 11.9, which suggests increasing survival into the older age groups. However, this change did not affect the median age at death for the age group 85 and over, which remained between 87 and 88 during this period.

The official mortality tabulations of the so-called centenarians show that the number of those whose ages are reported to be over 100 increased from about fifteen hundred in 1953 to twenty-three hundred in 1965. Those reported as 110 or more numbered between seventy and eighty-two during the years 1953–63. In 1964 this number dropped to fifty and then in 1965 to thirty-three. The oldest age reported during period 1953–65 was 136. Those tabulated as centenarians represent ages reported on death certificates which, however, are not verified statements or documented in any way. It is not possible to conclude from these data that there has been an increase in the life span. According to Myers[4] there is no real or convincing evidence from other data, such as those of Civil War veterans or social security beneficiaries, of survival up to an advanced age as high as 110.

Returning to the question of projections, let us note that Jenkins and Lew[5] in 1949, in what has been termed a "monumental masterpiece," included the opinions of a number of eminent physicians, demographers, and actuaries regarding the future course of mortality from various diseases. These experts were uniformly optimistic about the effect of new medical discoveries on future mortality. In view of the intensified attacks on medical problems in the postwar years, there was every reason to view with optimism the possibilities of a future decline in death rates.

In 1952 Dorn[6] estimated the annual mortality rate for the United

[4] R. J. Myers, "Validity of Centenarian Data in the 1960 Census," *Demography*, III (1966), 470–76.

[5] W. A. Jenkins and E. A. Lew, "A New Mortality Basis for Annuities," *Transactions of Society of Actuaries*, I, No. 1 (November, 1949), 369–498.

[6] H. F. Dorn, "Prospects of Further Decline in Mortality Rates," *Human Biology*, XXIV, No. 4 (December, 1952), 235–61.

States for 1960 and 1970 by age, sex, and color on the basis of data for the years 1920–48. In making his projections, Dorn assumed that

> unlike the past, the major factor bringing about large decreases in mortality in the future will be advances in medicine and surgery. This does not mean that the factors operating to bring about lower mortality rates in the past will not continue to be effective in the future. It is believed, however, that the future decreases in death rates due to these factors cannot equal those of the past over comparable periods of time; consequently, the decrease in mortality rates will tend to become smaller and smaller unless other factors begin to operate.

Dorn's projections of mortality for 1960 for the white population 5–39 years of age were extremely good. In the age group 40 and over, the projected rates were generally lower than the recorded rates, but the correspondence between the two sets of rates was fairly good. The agreement between the projected and the observed rates for the nonwhites was not nearly as good.

The projections for 1970 show that the estimated rates for 1970 are already far below the rates recorded for 1965. Dorn did not anticipate the large change in the rate of decline in the death rate which took place in the 1950's. There was no way in which he could have taken into account the future course of mortality in the United States.

By the same token, there is no way now of anticipating what the mortality situation will be in the future. For the short run there is no reason to expect much change in the death rate. Over a longer period of time, much will depend upon the future discovery of new methods of prevention of morbidity and mortality from the cardiovascular-renal and other chronic diseases. The medical progress anticipated by the experts who expressed their opinions on long-term mortality decreases seventeen or eighteen years ago is not thus far reflected by the death rates.

The comments up to this point have been limited to the mortality trend in the United States. As we examine trends for other countries, it seems significant that the United States is not alone in experiencing a change in the trend of the crude death rate. The death rates for the Netherlands, Sweden, Australia, Denmark, Canada, and England and Wales have definitely leveled off. In some of these countries, namely, the Netherlands, Australia, Sweden, and Denmark, the rates appear to be increasing.

These changes in mortality trends for the developed countries suggest that the developing countries might well experience a rapid decline in mortality as a result of the reduction of death rates for the various infective and parasitic diseases. (Certainly, the technical means are now available to make this possible.) However, after a period of rapid decline in the

death rate, the mortality trends for the developing countries will probably level off in the same manner as the death rates for the developed countries.

CHAIRMAN LEW: For an evaluation of the salient factors that affect mortality, I turned to my good friend Mortimer Spiegelman, who is preparing a revised edition of the Society of Actuaries' textbook on demography. As chairman of the Committee on Vital and Health Statistics Monographs of the American Public Health Association, he is also directing the completion of a series of publications recording some of the most authoritative research in these fields. In this capacity he has at his disposal much unique information on mortality. He has graciously offered to present some of it to us.

MR. MORTIMER SPIEGELMAN: These comments will be confined to some recent results in the study of the characteristics and trends of population mortality in the United States.[1] With but one exception, which will be noted, the characteristics described relate to the period 1959–61; trend data cover the years from 1950 to 1965.

Errors in population mortality.—The actuary, who is acquainted with the features of his insurance and annuitant data that bear upon mortality, is also aware of the classical weaknesses of misstatement of age, underenumeration, and underregistration that affect population mortality. Intensive investigations in connection with the 1960 census of population shed further light upon these weaknesses.

The extent of variation produced by investigators in the degree of undercount of total population in the 1960 census is not small. Thus, on the basis of investigation using several techniques, the net undercount for the 1960 census was estimated to range from 1.6 to 3.0 per cent for white males, 1.1 to 1.7 per cent for white females, 4.2 to 12.2 per cent for nonwhite males, and 3.4 to 8.8 per cent for nonwhite females; the estimates on the high side were considered as the more likely.[2] According to the same source, the net undercount of nonwhite males at ages 15–44 was almost 20 per cent.

An insight into the quality of age data in death statistics was made pos-

[1] Except for the socioeconomic data presented here, all of the following data on mortality characteristics are taken from an extensive set of tabulations made expressly for a series of vital and health statistics monographs being prepared by the American Public Health Association with support from the United States Public Health Service (Grant CH-00075).

[2] J. S. Siegel and M. Zelnik, "An Evaluation of Coverage in the 1960 Census of Population by Techniques of Demographic Analysis and by Composite Methods," in American Statistical Association, *Proceedings of the Social Statistics Section*, 196, p. 716.

sible by a comparison of the age stated on a sample of death certificates for the period May–August, 1960, with the statements of age for the same persons in the census taken as of April 1, 1960. When the data are combined into ten-year age groups, the deviations in statements of age on the two sources were relatively small in the case of the white population, the maximum ranging from 4 to almost 5 per cent at ages 25–44, where death

TABLE 1

PER CENT DIFFERENCE IN STATEMENT OF AGE AND OF MARITAL STATUS ON DEATH CERTIFICATES AND IN A CENSUS ENUMERATION FOR THE SAME PERSONS*

	MALE		FEMALE	
	White	Nonwhite	White	Nonwhite
Age group:				
1–4	− 0.6	− 1.3	− 0.9	− 1.7
5–14	0.2	0.4	− 1.5	− 2.3
15–24	0.2	− 2.9	− 1.8	− 4.8
25–34	− 4.0	− 3.2	− 4.9	− 2.4
35–44	− 4.1	− 1.9	− 4.0	− 1.1
45–54	− 1.2	6.2	− 1.3	13.3
55–64	0.2	13.6	− 0.6	23.6
65–74	− 0.3	2.1	− 2.8	− 4.5
75–84	1.2	−15.1	2.5	−17.2
85–99	1.9	−23.3	2.8	−21.5
Marital status, 15–99:				
Single	− 3.3	− 1.9	− 1.8	− 5.5
Married	− 1.2	− 2.6	− 2.3	− 3.4
Widowed	3.2	2.1	4.9	2.7
Divorced	20.3	8.7	23.9	19.8

* Sample of certificates of deaths during May–August, 1960, matched with returns in census of April 1, 1960. Per cent based upon census count. A negative per cent means that a correction amounts to increasing the number of deaths.

SOURCE.—R. D. Grove, "Vital Statistics for the Negro, Puerto Rican, and Mexican Populations: Present Quality and Plans for Improvement" (presented at Conference on Social Statistics and the City, Center for Urban Studies of the Massachusetts Institute of Technology and Harvard University, Washington, June 22–23, 1967), and unpublished data from the Division of Vital Statistics, National Center for Health Statistics.

rates are rather low. On the other hand, for the nonwhite population, the deviations amount to more than 20 per cent for both sexes at ages 85–99 and for females at ages 55–64. Though of lesser magnitude, they are appreciable for nonwhites of both sexes at ages 75–84, for males at ages 55–64, and for females at ages 45–54. Although the statement of age for the same person may be in error on both the death certificate and in the census, the deviations are such as to indicate a considerable understatement of mortality at the very high ages for nonwhites.

The picture presented by these findings is that population mortality

rates according to age are particularly unreliable for nonwhite males at all ages after childhood and for nonwhite females after age 45. For this reason, and also because of limitations of time, the rest of this discussion will be confined to mortality of the white population.

Age and sex.—Among white males the age-adjusted death rates for all ages reached a low point in 1954 and since then have been practically stationary; the influenza outbreaks of 1957, 1960, and 1963 brought small elevations. For white females the corresponding death rates declined from 1950 to 1961 and have since changed little.

No evidence of a recent leveling since 1950 is evident in the death rates at ages under 15; each year up through 1965 established an improvement over the year before, with the result that both males and females benefited by a reduction of somewhat over 30 per cent from 1950 to 1965. The situation was by no means as favorable for the next-older age group, 15–24. Although the general trend in death rates was initially downward for each sex, a low point was reached in 1961, with a marked rise since then. This age group was hardly affected by the influenza outbreaks of 1957, 1960, and 1963.

A course very similar to that for ages 15–24 is found for the two broad age brackets 25–44 and 45–64. Here, also, each sex showed a tendency toward improved mortality to a low point in 1961 and a subsequent rise. Whereas white males and white females were similar in their mortality trends at ages under 65, an entirely different picture emerges at ages 65 and over. Among white males at ages 65–74 and again at ages 75 and over, the death rates remained at practically the same level from 1950 to 1965. White females, on the other hand, continued to record mortality reductions at ages 65–74 and at ages 75 and over.

Geographic variations.—The range of variation of death rates within the United States is wide. Thus, for white males at all ages during 1959–61, the variation in age-adjusted rates was from a low of 795.7 per 100,000 in North Dakota to a high of 1,082.8 in Nevada—a 36 per cent margin. A more penetrating insight into geographic variation in mortality shows that the pattern is by no means uniform when age is taken into account in addition to sex. For white persons under age 15, the best records were made in Hawaii, Delaware, and Connecticut, and the poorest records were made in New Mexico and the District of Columbia. However, at ages 15–64, North Dakota and Nebraska emerge among the best states, and Nevada and the District of Columbia are the poorest. At ages 65 and over, Florida makes the most favorable showing, most likely because of its attraction for the aged who are healthy and wealthy enough to migrate there.

TABLE 2

DEATH RATES PER 100,000 FROM ALL CAUSES FOR WHITE PERSONS BY SEX AND AGE, UNITED STATES, 1950–65

Year	All Ages*	Under 15*	15–24	25–44*	45–64*	65–74	75 and Over*
	Males						
1965	909.3	201.2	148.7	245.5	1,438.6	4,929.5	11,667.6
1964	903.9	206.3	146.9	246.2	1,431.0	4,891.2	11,512.1
1963†	920.5	212.3	141.8	243.6	1,449.3	4,982.1	11,947.8
1962†	902.9	214.2	139.2	240.0	1,425.3	4,839.3	11,705.9
1961	891.0	216.5	138.4	235.8	1,412.1	4,740.3	11,522.6
1960	917.7	228.0	143.7	241.5	1,456.3	4,848.4	11,879.7
1959	902.1	231.6	145.5	239.8	1,433.7	4,731.1	11,570.8
1958	912.4	233.6	145.9	242.8	1,441.7	4,788.2	11,796.5
1957	922.9	236.4	156.0	247.3	1,461.1	4,863.7	11,748.8
1956	907.5	238.7	157.5	243.2	1,429.5	4,726.7	11,648.1
1955	905.0	243.2	156.1	246.4	1,422.9	4,698.7	11,560.4
1954	897.2	250.6	152.3	247.7	1,425.5	4,645.8	11,198.3
1953	939.3	267.8	163.3	262.5	1,495.3	4,771.5	11,763.6
1952	944.4	278.3	166.1	270.5	1,507.7	4,751.2	11,667.6
1951	956.5	283.3	158.5	277.8	1,511.0	4,827.2	11,955.0
1950	963.1	289.1	152.4	275.7	1,519.5	4,864.9	12,125.7
	Females						
1965	531.7	149.4	56.0	136.1	690.5	2,644.3	8,879.1
1964	534.7	154.4	56.7	136.3	691.5	2,670.4	8,888.1
1963†	546.2	158.3	56.3	135.5	697.7	2,729.0	9,221.6
1962†	541.7	159.9	54.3	134.8	691.7	2,707.9	9,116.3
1961	536.6	161.7	52.4	131.3	686.6	2,700.4	8,979.4
1960	555.0	168.6	54.9	134.0	710.1	2,779.3	9,322.3
1959	551.9	171.2	54.6	132.7	704.8	2,775.1	9,222.3
1958	564.7	176.7	54.7	133.8	719.2	2,851.6	9,448.3
1957	574.5	176.9	59.1	139.5	735.1	2,915.6	9,497.3
1956	567.6	177.9	58.5	135.7	722.0	2,882.3	9,414.3
1955	572.8	180.5	59.2	139.3	729.8	2,911.4	9,432.7
1954	574.2	187.4	57.6	141.9	749.9	2,918.8	9,174.4
1953	606.8	198.3	63.2	151.4	792.3	3,075.5	9,671.9
1952	619.1	212.1	69.4	160.2	810.6	3,100.3	9,685.8
1951	632.6	212.4	69.5	165.5	833.8	3,151.8	9,919.6
1950	645.0	216.9	71.5	169.6	849.3	3,242.8	10,026.8

* Age-adjusted on the basis of the age distribution of the total population of the United States in the census of April 1, 1940.

† United States, excluding New Jersey.

SOURCE.—Unpublished data from the Division of Vital Statistics, National Center for Health Statistics.

TABLE 3

DEATH RATES PER 100,000 FOR ALL CAUSES BY COLOR AND SEX, AGE-ADJUSTED* FOR ALL AGES AND AGES UNDER 15, UNITED STATES, EACH GEOGRAPHIC DIVISION AND STATE, 1959–61

AREA	ALL AGES				AGES UNDER 15			
	White		Nonwhite		White		Nonwhite	
	Male	Female	Male	Female	Male	Female	Male	Female
United States	906.3	549.7	1,185.4	878.5	226.7	167.9	414.6	326.7
New England	920.3	569.4	1,127.8	803.8	216.5	158.7	365.0	268.7
Connecticut	875.5	551.7	1,173.6	832.6	198.1	144.6	388.7	280.5
Maine	950.6	579.2	1,220.5	703.6	264.3	194.2	249.0†	122.2†
Massachusetts	930.7	574.8	1,081.9	790.5	210.7	153.1	373.0	260.2
New Hampshire	947.6	560.7	1,521.6	478.3	236.1	176.2	136.2†	38.5†
Rhode Island	915.3	582.3	1,247.9	831.8†	211.0	161.6	273.1†	336.1†
Vermont	948.3	563.0	575.2†	730.9†	256.2	187.1	.....	.....
Middle Atlantic	946.0	604.6	1,232.4	872.1	212.3	158.8	427.0	328.6
New Jersey	929.7	595.3	1,210.8	891.4	204.9	156.4	439.3	341.5
New York	940.0	603.2	1,243.3	850.8	213.4	158.9	435.4	326.0
Pennsylvania	963.9	612.2	1,215.8	893.9	214.5	160.0	404.2	324.8
East North Central	908.1	563.2	1,152.1	872.2	218.7	161.8	365.5	291.0
Illinois	935.0	573.6	1,226.6	917.2	218.6	160.4	380.0	313.0
Indiana	903.5	552.3	1,175.9	880.5	223.9	165.8	385.2	281.5
Michigan	908.0	562.8	1,026.6	815.5	221.0	161.6	335.4	259.9
Ohio	906.6	569.0	1,164.9	860.9	215.9	161.4	364.2	288.0
Wisconsin	854.1	537.2	1,025.7	803.1	214.6	162.2	374.0	311.2
West North Central	839.1	502.6	1,227.7	909.8	218.5	158.6	408.0	332.3
Iowa	832.7	496.7	1,050.5	903.1	209.9	158.5	346.7†	348.5†
Kansas	813.9	484.8	1,024.0	808.3	222.5	157.3	330.2	237.0
Minnesota	823.7	501.5	1,098.5	753.5	217.8	156.8	302.9†	252.9†
Missouri	883.8	525.7	1,266.0	923.9	216.1	160.7	406.0	342.7
Nebraska	812.0	483.7	1,363.2	929.0	218.4	156.9	448.8†	321.8†
North Dakota	795.7	494.9	1,352.1†	1,074.1†	230.4	167.5	614.3†	379.2†
South Dakota	838.3	486.7	1,544.0	1,197.4	245.2	155.0	704.3	595.2
South Atlantic	915.7	525.2	1,323.9	957.7	238.2	172.7	450.7	351.3
Delaware	923.0	573.2	1,364.7	996.3	195.8	141.2	508.3†	337.9†
District of Columbia	1,046.7	583.2	1,304.0	910.2	301.1	241.2	394.2	308.6
Florida	842.5	468.3	1,252.7	934.7	251.9	169.1	468.6	367.3
Georgia	941.3	511.1	1,365.6	984.7	240.9	165.0	451.7	349.6
Maryland	968.8	590.1	1,319.8	964.9	217.5	163.0	440.9	336.1
North Carolina	929.5	518.5	1,318.1	927.9	229.1	175.0	471.1	360.7
South Carolina	1,003.1	552.6	1,441.9	1,030.4	235.8	176.4	463.8	369.3
Virginia	924.5	539.5	1,305.6	961.3	238.2	172.6	425.8	336.9
West Virginia	943.5	574.4	1,211.2	862.0	257.6	199.2	317.0	285.4
East South Central	900.8	532.4	1,214.2	933.2	243.9	193.9	443.8	355.7
Alabama	917.9	518.9	1,240.2	957.7	230.7	187.8	415.2	340.9
Kentucky	902.6	553.7	1,319.7	965.7	251.8	200.4	428.0	316.4
Mississippi	895.4	510.8	1,147.8	880.8	241.9	188.4	498.0	398.6
Tennessee	889.8	531.2	1,211.1	950.0	247.3	194.3	402.9	318.9
West South Central	877.2	494.7	1,118.4	841.5	246.4	184.8	410.4	330.4
Arkansas	848.3	477.2	1,040.9	813.2	233.4	173.7	367.3	299.1
Louisiana	960.9	533.5	1,174.4	891.0	221.7	162.5	438.4	354.6
Oklahoma	869.2	484.3	1,054.6	795.0	231.2	172.3	413.0	305.7
Texas	865.2	491.5	1,110.2	817.3	258.1	195.0	398.3	323.2
Mountain	898.0	529.7	1,095.2	776.4	262.6	191.9	502.7	406.9
Arizona	958.2	520.7	1,118.3	790.5	281.9	208.4	571.2	430.5
Colorado	860.0	526.1	988.4	652.3	264.5	190.1	379.2	277.6†
Idaho	846.6	501.4	1,108.5†	782.8†	244.1	175.8	425.6†	260.9†
Montana	947.0	544.3	1,276.8	1,099.9	245.4	171.4	489.8†	519.4
Nevada	1,082.8	595.3	1,109.1	990.7†	283.1	226.1	496.6†	367.6†
New Mexico	884.2	556.9	1,021.2	656.4	307.6	224.3	495.5	422.8
Utah	823.4	509.3	1,182.8	713.1†	201.1	160.8	429.8†	418.9†
Wyoming	911.0	523.7	1,139.2†	1,178.9†	288.2	181.9	325.1†	440.2†
Pacific	887.4	524.0	871.5	624.7	225.3	165.7	313.4	238.8
Alaska	931.6	549.2	1,019.6	894.2	261.7	171.0	821.4	579.1
California	888.5	525.8	900.1	644.0	223.3	166.4	299.9	234.2
Hawaii	991.8	638.2	701.7	503.9	175.8	150.0†	234.5	176.5
Oregon	883.1	512.4	1,146.1	761.6	242.3	172.8	356.8†	316.7†
Washington	882.3	517.7	1,056.5	689.1	226.9	159.0	435.0	269.6

* Computed by the direct method, using as the standard population the age distribution of the total population of the United States as enumerated in 1940.

† Age-adjusted rate computed from age-specific rates where more than one-half of the rates were based on frequencies of less than 20 deaths.

TABLE 4

DEATH RATES PER 100,000 FOR ALL CAUSES BY COLOR AND SEX, AGE-ADJUSTED* FOR AGES 15–64 AND AGES 65 AND OVER, UNITED STATES, EACH GEOGRAPHIC DIVISION AND STATE, 1959–61

AREA	AGES 15–64				AGES 65 AND OVER			
	White		Nonwhite		White		Nonwhite	
	Male	Female	Male	Female	Male	Female	Male	Female
United States	562.0	277.5	920.6	671.6	6,814.4	4,652.1	6,636.2	4,952.7
New England	553.4	284.0	780.6	540.9	7,141.2	4,908.8	7,368.7	5,373.7
Connecticut	513.2	270.0	808.4	542.2	6,954.4	4,841.0	7,674.3	5,738.8
Maine	575.1	292.3	988.4	485.3	7,192.9	4,838.9	7,079.7†	4,999.9†
Massachusetts	567.8	290.7	745.8	541.2	7,171.1	4,941.4	7,015.5	5,207.7
New Hampshire	570.3	273.7	951.2	520.7	7,300.0	4,820.3	12,258.1†	1,664.2†
Rhode Island	544.2	285.1	825.9	541.0	7,179.9	5,074.9	9,007.3	5,535.0
Vermont	567.4	275.8	595.7	523.9	7,265.6	4,792.4	2,474.6†	5,460.6†
Middle Atlantic	571.7	303.8	944.8	633.3	7,349.6	5,225.3	7,036.1	5,233.5
New Jersey	555.2	300.9	914.7	646.5	7,303.2	5,126.8	6,975.1	5,337.2
New York	566.7	301.7	972.0	609.6	7,307.6	5,224.9	6,894.0	5,167.9
Pennsylvania	588.7	309.2	908.6	662.4	7,434.0	5,278.2	7,237.4	5,275.9
East North Central	548.8	281.8	855.1	648.4	7,001.0	4,828.6	6,980.4	5,222.6
Illinois	570.7	292.3	927.1	681.8	7,176.1	4,881.1	7,299.2	5,466.8
Indiana	549.8	274.5	860.8	668.6	6,904.4	4,726.9	7,199.4	5,176.8
Michigan	538.6	282.1	748.6	611.7	7,092.5	4,820.7	6,317.9	4,873.2
Ohio	549.6	284.4	871.1	639.0	6,981.2	4,888.8	7,013.0	5,161.6
Wisconsin	509.9	257.8	721.7	525.3	6,614.6	4,686.4	6,431.0	5,363.6
West North Central	508.8	246.0	935.1	678.5	6,392.3	4,311.3	7,134.0	5,320.4
Iowa	502.0	241.0	644.4	572.8	6,398.0	4,275.5	7,661.1	6,215.2
Kansas	495.5	238.2	707.3	582.5	6,141.9	4,133.7	6,709.3	5,142.1
Minnesota	490.6	244.6	795.1	493.2	6,350.3	4,315.9	7,023.5	5,172.2
Missouri	548.4	262.1	989.7	707.4	6,659.8	4,480.7	7,157.4	5,201.2
Nebraska	482.0	233.1	1,068.8	723.9	6,263.6	4,170.4	7,633.3	5,188.0
North Dakota	468.3	231.7	936.4	778.6	6,117.4	4,308.5	8,182.7	6,552.4
South Dakota	515.0	237.1	1,269.4	768.2	6,221.2	4,181.4	7,343.8	7,666.7
South Atlantic	601.7	265.5	1,099.6	783.0	6,514.4	4,395.8	6,746.6	4,911.6
Delaware	560.1	285.5	1,031.3	712.9	7,189.9	5,012.6	7,810.5	6,220.6
District of Columbia	708.2	322.4	982.1	637.6	7,137.9	4,426.6	7,830.6	5,820.0
Florida	582.1	256.6	1,075.1	815.0	5,590.6	3,667.2	5,884.7	4,198.9
Georgia	612.9	255.6	1,197.1	871.0	6,767.5	4,316.9	6,381.9	4,436.6
Maryland	590.1	295.2	995.7	707.1	7,480.7	5,083.5	7,754.9	5,826.9
North Carolina	611.8	243.9	1,069.8	704.9	6,648.8	4,504.4	6,883.5	5,218.6
South Carolina	648.9	278.0	1,350.4	954.1	7,330.3	4,657.8	5,927.6	4,205.9
Virginia	577.7	258.6	1,010.8	730.7	6,881.3	4,673.7	7,452.9	5,537.0
West Virginia	623.0	290.8	947.6	632.3	6,637.4	4,766.3	7,101.4	5,253.7
East South Central	579.9	257.4	939.3	713.7	6,493.2	4,504.6	6,763.8	5,226.7
Alabama	597.4	249.3	1,008.4	774.9	6,616.8	4,409.5	6,561.0	5,029.7
Kentucky	587.7	274.0	1,028.5	737.3	6,412.3	4,626.5	7,474.5	5,610.4
Mississippi	582.9	242.1	828.5	629.2	6,391.2	4,360.7	6,698.0	5,145.7
Tennessee	558.0	254.6	947.3	726.4	6,538.0	4,513.3	6,788.9	5,480.5
West South Central	555.1	248.1	866.9	651.8	6,385.6	4,079.6	6,207.2	4,596.2
Arkansas	551.6	232.7	789.2	638.6	6,046.2	4,017.4	6,005.9	4,428.3
Louisiana	629.2	263.6	884.0	667.8	6,961.4	4,573.0	6,752.3	5,071.7
Oklahoma	553.9	250.8	811.4	582.8	6,336.1	3,946.3	5,818.1	4,693.3
Texas	537.1	246.4	890.2	658.3	6,347.2	4,012.8	5,900.2	4,204.1
Mountain	576.6	281.3	876.3	564.9	6,417.0	4,234.0	5,438.1	4,230.1
Arizona	644.5	291.4	967.0	583.3	6,549.4	3,942.5	4,623.1	4,167.2
Colorado	539.4	275.1	681.7	449.8	6,224.4	4,250.3	6,264.7	4,035.7
Idaho	521.1	259.4	862.5	615.3	6,285.4	4,097.9	6,051.3	4,356.5†
Montana	617.5	288.0	1,006.1	868.0	6,787.7	4,455.8	6,845.0	5,528.0
Nevada	720.7	343.3	884.5	769.1	7,606.5	4,450.6	5,581.5	5,471.7†
New Mexico	571.7	299.1	814.7	438.8	6,098.8	4,336.0	4,996.0	3,673.7
Utah	512.4	256.2	891.5	479.5	6,191.0	4,300.1	6,831.8	4,111.5†
Wyoming	578.7	267.4	982.3	1,052.9	6,491.8	4,321.5	5,675.6†	5,131.9†
Pacific	557.1	279.5	587.1	409.8	6,592.5	4,264.5	5,739.5	4,171.7
Alaska	565.3	257.9	715.7	597.9	7,023.0	4,828.3	4,766.0	4,991.9
California	560.0	283.7	624.0	435.3	6,587.0	4,246.8	5,839.6	4,217.4
Hawaii	603.2	355.8	402.4	286.5	7,838.7	5,231.0	5,385.7	3,862.6
Oregon	553.9	264.5	965.8	496.5	6,498.8	4,218.5	5,823.9	5,024.4
Washington	545.2	264.0	750.9	486.2	6,629.9	4,351.7	6,366.7	4,239.7

* Computed by the direct method, using as the standard population the age distribution of the total population of the United States as enumerated in 1940.

† Age-adjusted rate computed from age-specific rates where more than one-half of the rates were based on frequencies of less than 20 deaths.

TABLE 5

LOW- AND HIGH-RANKING STATES ACCORDING TO AGE-ADJUSTED DEATH RATES* FROM ALL CAUSES BY COLOR AND SEX FOR ALL AGES, UNDER 15, 15–64, AND 65 AND OVER, UNITED STATES, 1959–61

AGE	LOW			HIGH		
	Lowest	Second Low	Third Low	Highest	Second High	Third High
	White Males					
All ages	North Dakota	Nebraska	Kansas	Nevada	District of Columbia	South Carolina
Under 15	Hawaii	Delaware	Connecticut	New Mexico	District of Columbia	Wyoming
15–64	North Dakota	Nebraska	Minnesota	Nevada	District of Columbia	South Carolina
65 and over	Florida	Arkansas	New Mexico	Hawaii	Nevada	Maryland
	White Females					
All ages	Florida	Arkansas	Nebraska	Hawaii	Pennsylvania	New York
Under 15	Delaware	Connecticut	Hawaii	District of Columbia	Nevada	New Mexico
15–64	North Dakota	Arkansas	Nebraska	Hawaii	Nevada	District of Columbia
65 and over	Florida	Arizona	Oklahoma	Pennsylvania	Hawaii	New York
	Nonwhite Males†					
All ages	Kansas	Wisconsin	Michigan	South Dakota	South Carolina	Georgia
Under 15	Minnesota	West Virginia	Kansas	South Dakota	North Dakota	Delaware
15–64	Iowa	Kansas	Wisconsin	South Carolina	South Dakota	Georgia
65 and over	Oklahoma	Florida	Texas	North Dakota	District of Columbia	Delaware
	Nonwhite Females†					
All ages	Minnesota	Oklahoma	Wisconsin	South Dakota	North Dakota	South Carolina
Under 15	Kansas	Minnesota	Michigan	South Dakota	Mississippi	North Dakota
15–64	Minnesota	Wisconsin	Iowa	South Carolina	Georgia	Florida
65 and over	Florida	Texas	South Carolina	South Dakota	North Dakota	Delaware

* Age-adjusted on the basis of the age distribution of the total population of the United States in the census of April 1, 1940.

† Excluding New England, Mountain, and Pacific Coast states.

The extent of variation in death rates among the states has been reduced, along with the general decline in mortality for the country as a whole. In other words, the rates of improvement for the states with the initially poorer records have been faster than those with the better initial records. A ready summary of the situation is provided by the standard deviations and coefficients of variation of the age-adjusted death rates for all ages among the states in 1940, 1949–51, and 1959–61. For both males and females, without distinction by color, there was an appreciable reduction in the extent of variation from 1940 to 1949–51, a period of marked reduction in mortality. However, in the decade from 1949–51 to 1959–61, a

TABLE 6

STANDARD DEVIATION AND COEFFICIENT OF VARIATION* OF DEATH RATES PER 1,000† AT ALL AGES FOR EACH STATE‡ AND THE DISTRICT OF COLUMBIA, 1940, 1949–51, AND 1959–61

PERIOD	STANDARD DEVIATION		COEFFICIENT OF VARIATION	
	Male	Female	Male	Female
1940.....	1.63	1.15	.135	.125
1949–51.....	0.87	0.65	.088	.095
1959–61.....	0.77	0.55	.082	.095

* Standard deviation divided by arithmetic mean of rates.

† Age-adjusted on the basis of the age distribution of the total population of the United States in the census of April 1, 1940.

‡ Excludes Alaska and Hawaii.

period of rather slow mortality reduction, the extent of variation in death rates among the states narrowed by only a small margin.

Cities, suburbs, and rural areas.—An insight into mortality for these three types of residence is indicated by data for Standard Metropolitan Statistical Areas containing a central city, such counties without a central city, and all other counties. The definitions for these aggregates of population are fixed by the Bureau of the Budget.

For the country as a whole the age-adjusted death rates for all ages are highest for the metropolitan counties containing a central city. In the case of white males of all ages death rates are generally lowest in metropolitan counties without a central city; these are largely suburban in character. However, the death rates for white females in metropolitan counties without a central city tend to be somewhat greater than those for nonmetropolitan counties (largely rural in character).

Although the same pattern will be observed at ages 15–64, it is quite different for the ages outside that range. Thus, at ages under 15 the ad-

TABLE 7

DEATH RATES PER 100,000 FOR ALL CAUSES FOR WHITE PERSONS BY SEX, AGE-ADJUSTED* FOR ALL AGES, UNDER 15, 15–64, AND 65 AND OVER, FOR METROPOLITAN AND NONMETROPOLITAN COUNTIES, UNITED STATES, 1959–61

SEX AND GEOGRAPHIC DIVISION	METROPOLITAN COUNTIES			NON-METROPOLITAN COUNTIES	METROPOLITAN COUNTIES			NON-METROPOLITAN COUNTIES
	Total	With Central City	Without Central City		Total	With Central City	Without Central City	
	All Ages				Ages under 15			
Males:								
United States	923.0	938.0	858.9	882.5	217.2	221.7	201.1	242.7
New England	919.4	925.3	861.2	920.6	208.7	209.3	203.1	240.3
Middle Atlantic	946.4	977.0	870.6	942.3	208.0	215.5	193.8	228.7
East North Central	931.8	943.2	862.3	871.0	213.1	214.5	206.3	228.8
West North Central	893.6	917.3	812.7	809.9	211.7	220.7	185.7	223.7
South Atlantic	907.2	929.8	855.5	925.4	226.6	238.4	203.5	250.5
East South Central	936.0	930.6	977.0	888.5	219.2	215.4	250.1	258.4
West South Central	905.9	912.1	848.6	860.5	236.6	239.5	205.7	259.4
Mountain	897.8	911.8	789.1	900.2	245.9	249.9	221.7	278.8
Pacific	892.3	902.1	828.6	874.9	219.5	221.3	209.9	246.1
Females:								
United States	562.3	567.4	541.1	528.2	161.7	165.8	147.1	178.6
New England	572.8	577.0	531.5	556.6	153.4	155.1	137.1	175.1
Middle Atlantic	607.7	624.8	564.9	591.9	156.2	163.7	142.0	168.8
East North Central	574.9	579.6	546.0	543.1	160.4	162.0	153.0	164.2
West North Central	521.9	527.3	505.5	490.2	155.9	164.2	132.7	160.7
South Atlantic	522.5	526.6	518.8	527.8	163.5	169.1	152.3	182.6
East South Central	533.7	526.9	586.2	532.2	172.3	167.2	211.3	206.5
West South Central	509.0	512.1	477.9	482.5	179.4	181.2	158.6	191.8
Mountain	526.2	531.9	487.0	532.9	184.1	188.5	157.3	199.4
Pacific	527.3	530.8	503.6	510.2	160.2	163.1	144.6	185.9
	Ages 15–64				Ages 65 and Over			
Males:								
United States	565.9	581.4	502.1	555.4	7,053.9	7,102.3	6,811.6	6,474.0
New England	555.0	561.0	498.0	546.2	7,141.2	7,165.2	6,878.4	7,130.1
Middle Atlantic	570.0	598.6	502.4	576.9	7,388.6	7,523.5	7,006.1	7,183.7
East North Central	559.5	569.2	503.3	528.1	7,261.3	7,326.2	6,829.5	6,628.1
West North Central	541.3	564.8	467.3	489.2	6,889.8	6,968.8	6,539.0	6,141.7
South Atlantic	586.1	618.5	503.4	619.0	6,587.6	6,552.5	6,740.2	6,439.1
East South Central	592.3	588.7	621.5	575.5	6,973.5	6,945.0	7,168.7	6,304.5
West South Central	565.3	568.6	532.3	550.2	6,739.5	6,786.0	6,344.2	6,143.1
Mountain	579.4	594.2	472.7	576.7	6,446.8	6,489.1	6,009.8	6,388.6
Pacific	556.2	566.0	494.5	562.1	6,694.0	6,732.7	6,412.1	6,283.4
Females:								
United States	286.3	291.2	265.9	261.2	4,771.3	4,782.1	4,718.1	4,461.4
New England	287.2	290.6	255.1	271.7	4,945.6	4,967.2	4,722.0	4,784.4
Middle Atlantic	305.0	316.6	276.6	298.2	5,268.0	5,375.0	4,977.8	5,059.1
East North Central	289.5	293.0	268.5	267.0	4,928.3	4,955.7	4,741.4	4,673.4
West North Central	259.1	264.9	238.9	236.9	4,472.9	4,464.0	4,518.8	4,213.5
South Atlantic	269.5	277.7	250.0	261.0	4,350.2	4,308.4	4,531.5	4,443.0
East South Central	263.1	259.4	292.6	254.4	4,545.6	4,501.2	4,875.5	4,485.2
West South Central	258.6	260.7	237.0	237.8	4,203.9	4,221.2	4,040.0	3,978.6
Mountain	284.0	291.0	234.8	278.6	4,184.7	4,182.9	4,199.9	4,280.9
Pacific	282.2	284.5	267.6	268.7	4,306.1	4,324.0	4,162.4	4,097.2

* Computed by the direct method, using as the standard population the age distribution of the total population of the United States as enumerated in 1940.

vantage still lies with the metropolitan counties not containing a central city, but for each sex the death rates are highest in the nonmetropolitan counties. On the other hand, at ages 65 and over the advantage passes to the nonmetropolitan counties for the country as a whole.

Socioeconomic differentials.—The socioeconomic data discussed here are from a large-scale study at the University of Chicago in which deaths during May–August, 1960, were matched with reports for the same individuals in the census of 1960.[3] Up to the point of the Chicago study the

[3] E. M. Kitagawa and P. M. Hauser, "Methods Used in a Current Study of Social and Economic Differentials in Mortality," *Emerging Techniques in Population Research* (New York: Milbank Memorial Fund, 1963), p. 230.

TABLE 8

MORTALITY DIFFERENTIALS BY YEARS OF SCHOOL COMPLETED AND FAMILY INCOME FOR THE WHITE POPULATION 25 YEARS OF AGE AND OVER, BY SEX AND AGE, UNITED STATES, MAY–AUGUST, 1960

	MALES			FEMALES		
	25 and Over	25–64	65 and Over	25 and Over	25–64	65 and Over
	Years of School					
Total	100%	100%	100%	100%	100%	100%
Under 5 years	101	115	102	126	160	117
Elementary:						
5–7 years	104	115	100	108	118	104
8 years	101	106	100	105	108	103
High school:						
1–3 years	102	103	98	88	91	94
4 years	98	91		92	87	
College:						
1 or more years	89	77	100	73	81	70
	Family Income					
Total	100%	100%	100%	100%	100%	100%
Under $2,000	113	156	109	105	123	96
$ 2,000–$3,999	103	119	100	103	110	98
4,000– 5,999	97	100	92	100	101	105
6,000– 7,999	92	87		102	96	
8,000– 9,999	100	93	97	95	92	100
10,000 or more	89	84		92	87	

SOURCE.—E. M. Kitagawa and P. M. Hauser, "Education and Income Differentials in Mortality, United States, 1960," *Proceedings of the 11th Pacific Science Congress, Symposium No. 1* (Tokyo; August, 1966).

socioeconomic stratification of a population for the study of mortality differentials was usually based upon occupational groupings of the family head. In the United States at least, the individual frequently shifts occupations over a working lifetime, so the mortality of an occupational group at the older ages may be influenced by shifts into and out of the group in earlier years. On the other hand, the level of educational attainment is fixed for most persons by age 25.

The Chicago study shows that both males and females at ages 25–64 recorded a decrease in death rates with a rise in the level of educational attainment; this was more evident for females. At ages 65 and over, however, the relationships is greatly modified, particularly among males. Mortality is also inversely correlated with family income at ages 25–64, much more for males than for females. The quality of this relationship is affected by the reductions of income often accompanying illness before death. At ages 65 and over there is no apparent relationship between mortality and family income.

Nativity.—After account is taken of the difference in the age distribution between the white native-born and the white foreign-born by age-adjusted death rates, the mortality of the latter is the higher by only 2 per cent in the case of males and by 13 per cent for females. At ages under 15 the death rates for the foreign-born are about two-thirds those of the native-born. This probably reflects a selective influence, since the young foreign-born were presumably healthy enough to migrate and to pass physical requirements for admission. At ages 15–64 ,the death rates for the two categories are not much different. However, at ages 65 and over the mortality of the foreign-born is the higher by 11 per cent for males and by 23 per cent for females. These comparisons between the native-born and the foreign-born may be affected by differentials in their socioeconomic and geographic distributions.

Cause of death.—It is possible to comment in great detail upon trends in mortality according to cause of death if variations by age, sex, and color are taken into account. However, for sake of brevity, the present discussion will be restricted to age-adjusted death rates for all ages for the leading causes.

Foremost among the broad categories of causes of death are the major cardiovascular-renal diseases. During 1961–65 these accounted for somewhat over half of the total deaths for white persons of each sex. The proportions were practically identical for 1950–54, an indication that the trend for these diseases was about the same as that for all causes of death.

Outstanding among the major cardiovascular-renal diseases are

TABLE 9

DEATH RATES PER 100,000* FROM ALL CAUSES FOR BROAD AGE GROUPS AND AT ALL AGES FROM SELECTED CAUSES FOR WHITE PERSONS BY SEX, SEPARATELY FOR THE NATIVE-BORN AND THE FOREIGN-BORN, UNITED STATES, 1959–61

CAUSE OF DEATH	NATIVE		FOREIGN		RATIO: FOREIGN TO NATIVE		RATIO: MALE TO FEMALE	
	Male	Female	Male	Female	Male	Female	Native	Foreign
All causes:								
All ages	903.5	538.4	919.8	608.7	1.02	1.13	1.68	1.51
Under 15	234.0	173.5	160.2	114.3	0.68	0.66	1.35	1.40
15–64	555.1	269.5	530.9	290.2	0.96	1.08	2.06	1.83
65 and over	6,815.4	4,546.5	7,563.7	5,583.0	1.11	1.23	1.50	1.35
All ages:								
Major cardiovascular-renal	490.8	281.8	498.8	342.1	1.02	1.21	1.74	1.46
Vascular lesions	82.8	69.0	76.2	71.9	0.92	1.04	1.20	1.06
Arteriosclerosis	299.4	129.6	320.1	177.0	1.07	1.37	2.31	1.81
Malignant neoplasms	95.2	86.7	104.5	94.5	1.10	1.09	1.10	1.11
Digestive	43.9	32.3	66.3	44.9	1.51	1.39	1.36	1.48
Respiratory	34.4	5.3	38.0	6.3	1.10	1.19	6.49	6.03
Breast	0.2	22.3	0.2†	22.3	1.00	1.00	0.01	0.01
Cervix	.....	7.4	.....	5.9	.....	0.80	.....	.....
Female genitourinary	.....	13.2	.....	14.2	.....	1.08	.....	.....
Prostate	12.9	.....	12.1	.....	0.94	.....	.....	.....
Diabetes	11.0	12.3	12.5	19.0	1.14	1.54	0.89	0.66
Diseases of the digestive system	37.6	21.5	41.5	24.2	1.10	1.13	1.75	1.71
Influenza and pneumonia‡	26.9	16.6	28.7	19.2	1.07	1.16	1.62	1.49
Tuberculosis	6.5	2.1	7.2	2.7	1.11	1.29	3.10	2.67
Suicide	17.2	5.0	16.6	6.0	0.97	1.20	3.44	2.77
Accidents:								
Total	69.4	25.0	72.9	26.7	1.05	1.07	2.78	2.73
Motor vehicle	33.8	11.0	32.9	11.4	0.97	1.04	3.07	2.89
Other	35.6	14.0	40.0	15.3	1.12	1.09	2.54	2.61

* Age-adjusted on the basis of the age distribution of the total population of the United States in the census of April 1, 1940.

† Age-adjusted rate based on age-specific rates more than half of which were based upon frequencies of less than 20 deaths.

‡ Excepting pneumonia of the newborn.

SOURCE.—Special tabulations in the National Center for Health Statistics for the series of vital and health statistics monographs by the American Public Health Association.

TABLE 10

DEATH RATES PER 100,000 AT ALL AGES* FROM SELECTED CAUSES FOR WHITE PERSONS BY SEX, UNITED STATES, 1950–65

YEAR	MAJOR CARDIOVASCULAR-RENAL DISEASES						MALIGNANT NEOPLASMS
	Total	Diseases of Cardiovascular System				Chronic Nephritis, etc.	
		Total	Vascular Lesions	Diseases of Heart	General Arteriosclerosis		
	Males						
1965....	479.3	474.5	73.8	368.6	13.4	4.8	148.0
1964....	479.2	474.1	74.6	367.7	13.1	5.0	145.6
1963†...	490.5	485.2	77.6	375.7	13.8	5.2	143.8
1962†...	484.5	479.1	77.4	370.3	13.7	5.5	142.0
1961....	480.5	474.9	77.3	367.3	13.7	5.7	142.0
1960....	493.2	487.3	80.3	375.4	14.7	5.9	141.6
1959....	487.3	481.2	80.9	369.4	14.4	6.0	139.4
1958....	495.1	488.2	82.9	373.7	14.9	6.9	138.2
1957....	496.8	489.2	84.4	375.6	14.8	7.6	139.9
1956....	488.8	481.1	82.1	370.2	14.7	7.8	139.1
1955....	487.0	478.6	82.7	367.4	15.6	8.4	137.4
1954....	480.4	471.3	82.0	362.2	14.8	9.0	136.0
1953....	501.5	491.3	85.6	376.5	16.7	10.2	135.5
1952....	501.9	490.4	86.4	374.7	16.8	11.5	133.7
1951....	508.3	495.7	87.3	378.0	17.9	12.6	131.1
1950....	514.1	499.9	87.0	381.1	18.4	14.2	130.9
	Females						
1965....	272.9	269.8	62.1	185.9	10.9	3.0	108.1
1964....	276.1	272.9	63.3	187.9	11.1	3.3	107.6
1963†...	283.7	280.3	65.9	192.3	11.4	3.4	107.3
1962†...	283.3	279.8	66.5	191.6	11.5	3.5	107.4
1961....	281.6	277.8	66.3	190.5	11.2	3.7	108.5
1960....	291.5	287.6	68.7	197.1	11.7	3.9	109.5
1959....	292.6	288.5	70.2	196.6	11.7	4.1	109.4
1958....	301.6	296.9	72.5	202.4	11.9	4.8	110.6
1957....	305.3	299.8	73.4	205.8	11.9	5.5	112.6
1956....	301.9	296.2	72.3	203.5	11.9	5.7	113.2
1955....	303.8	297.8	73.2	204.0	12.4	6.0	114.3
1954....	303.2	296.4	73.5	202.6	12.1	6.9	115.1
1953....	321.5	313.8	77.7	214.1	13.4	7.7	116.4
1952....	325.1	316.7	78.4	215.7	13.9	8.4	117.6
1951....	349.8	339.7	83.9	218.9	16.5	10.0	119.1
1950....	339.0	328.0	79.7	223.6	14.3†	11.0	119.4

* Age-adjusted on the basis of the age distribution of the total population of the United States in the census of April 1, 1940.

† United States, excluding New Jersey.

‡ Age-adjusted rate based on age-specific rates more than half of which were based upon frequencies of less than 20 deaths.

SOURCE.—Unpublished data from the Division of Vital Statistics, National Center for Health Statistics.

TABLE 11

DEATH RATES PER 100,000 AT ALL AGES* FROM SELECTED CAUSES FOR WHITE PERSONS BY SEX, UNITED STATES, 1950–65

YEAR	ACCIDENTS			INFLUENZA AND PNEUMONIA†	DIABETES MELLITUS	CIRRHOSIS OF THE LIVER	TUBERCULOSIS, ALL FORMS
	Total	Motor Vehicle	Other				
	Males						
1965	75.4	39.4	36.0	26.9	11.9	15.6	4.5
1964	73.4	38.1	35.3	25.9	11.8	15.0	4.8
1963‡	72.4	36.7	35.6	30.7	11.9	14.7	5.4
1962‡	70.7	34.9	35.7	26.5	11.8	14.6	5.6
1961	68.5	33.4	35.2	24.8	11.4	14.2	6.1
1960	70.6	34.0	36.5	31.0	11.6	14.4	6.8
1959	71.2	34.5	36.6	25.8	11.0	13.7	7.3
1958	71.1	34.3	36.7	27.7	11.1	13.7	8.0
1957	76.1	36.8	39.2	30.5	10.9	14.4	8.9
1956	77.8	38.5	39.4	23.5	10.9	13.4	9.3
1955	77.7	37.8	40.0	22.3	10.9	12.9	10.0
1954	75.9	35.4	40.6	21.2	10.9	12.8	11.1
1953	81.8	38.0	43.8	27.5	11.2	13.0	13.2
1952	83.2	38.4	44.8	24.8	11.5	13.0	16.6
1951	84.5	37.8	46.7	26.6	11.2	12.3	21.1
1950	81.0	35.9	45.0	27.1	11.3	11.6	23.3
	Females						
1965	27.7	13.7	14.0	16.1	12.9	7.6	1.3
1964	27.3	13.6	13.7	15.7	12.9	7.3	1.4
1963‡	26.7	12.6	14.1	19.0	13.3	7.2	1.6
1962‡	26.2	12.0	14.2	16.7	13.1	6.8	1.8
1961	24.5	10.8	13.6	15.5	13.3	6.7	1.9
1960	25.4	11.1	14.4	19.0	13.7	6.6	2.2
1959	25.1	11.0	14.1	16.2	13.5	6.4	2.4
1958	25.1	10.8	14.3	17.2	13.6	6.3	2.6
1957	27.0	11.2	15.8	19.3	14.3	6.6	2.9
1956	27.4	11.5	15.8	15.1	14.0	6.5	3.2
1955	27.6	11.4	16.2	14.9	14.1	6.1	3.6
1954	27.1	10.6	16.6	13.9	14.5	6.0	4.1
1953	29.1	11.5	17.6	18.2	15.6	6.4	5.0
1952	30.6	11.6	19.0	16.9	15.8	6.2	6.8
1951	32.0	11.2	20.8	19.7	16.6	6.2	9.2
1950	30.6	10.6	20.0	18.9	16.4	5.8	10.2

* Age-adjusted on the basis of the age distribution of the total population of the United States in the census of April 1, 1960.

† Excepting pneumonia of the newborn.

‡ United States, excluding New Jersey.

SOURCE.—Unpublished data from the Division of Vital Statistics, National Center for Health Statistics.

TABLE 12

DEATH RATES PER 100,000 AT ALL AGES* FROM SELECTED CAUSES FOR NONWHITE PERSONS BY SEX, UNITED STATES, 1950–65

YEAR	MAJOR CARDIOVASCULAR-RENAL DISEASES						MALIGNANT NEOPLASMS
	Total	Diseases of Cardiovascular System				Chronic Nephritis, etc.	
		Total	Vascular Lesions	Diseases of Heart	General Arteriosclerosis		
	Males						
1965	569.8	555.8	134.2	376.1	13.6	14.0	173.3
1964	565.3	550.8	131.1	375.2	13.6	14.5	170.1
1963†	584.7	569.6	135.7	387.0	14.7	15.2	168.7
1962†	569.4	554.7	133.4	376.4	15.0	14.7	159.1
1961	548.3	533.5	127.5	362.7	14.2	14.8	157.9
1960	564.0	547.8	135.2	368.3	15.5	16.1	154.8
1959	562.2	545.4	134.8	365.9	15.2	16.8	152.5
1958	582.1	563.4	141.2	377.1	16.0	18.7	146.4
1957	589.7	569.8	140.2	387.2	15.2	20.0	149.2
1956	568.2	547.2	134.3	372.7	14.8	21.0	145.0
1955	567.3	546.7	136.2	369.2	16.2	20.5	138.7
1954	567.4	542.7	136.0	367.0	15.5	24.7	140.6
1953	611.2	584.6	141.7	398.3	17.7	26.6	133.2
1952	621.4	588.6	143.9	402.8	17.1	32.9	130.7
1951	619.9	586.0	142.6	400.5	17.1	33.9	125.3
1950	633.7	595.4	144.0	407.5	18.0‡	38.3	125.8
	Females						
1965	440.8	430.1	125.5	269.2	11.3	10.7	125.2
1964	450.2	438.5	125.8	276.4	11.8	11.6	124.0
1963†	466.8	455.3	131.7	286.9	12.4	11.5	124.4
1962†	463.2	451.2	132.2	283 0	12.1	12.0	124.7
1961	455.0	443.4	130.2	278.0	11.5	11.7	124.3
1960	467.1	453.8	134.4	283.3	12.3	13.2	125.0
1959	465.4	451.2	136.5	279.2	12.3	14.2	121.3
1958	492.4	476.2	139.4	297.5	13.7	16.3	125.3
1957	503.6	485.2	142.2	307.6	13.1	18.4	125.2
1956	490.8	471.8	139.1	298.7	12.5	19.0	127.8
1955	486.1	466.5	139.3	293.0	12.5	19.6	124.7
1954	489.4	469.0	142.0	292.1	12.6	20.4	125.9
1953	520.9	496.2	145.7	314.6	13.5	24.7	125.1
1952	538.6	509.6	149.9	323.0	13.1	29.1	126.8
1951	550.2	518.6	152.1	329.5	13.1	31.6	126.4
1950	570.8	534.7	153.4	342.9	13.7‡	36.1	131.0

* Age-adjusted on the basis of the age distribution of the total population of the United States in the census of April 1, 1940.

† United States, excluding New Jersey.

‡ Age-adjusted rate based on age-specific rates more than half of which were based upon frequencies of less than 20 deaths.

SOURCE.—Unpublished data from the Division of Vital Statistics, National Center for Health Statistics.

TABLE 13

DEATH RATES PER 100,000 AT ALL AGES* FROM SELECTED CAUSES FOR NONWHITE PERSONS BY SEX, UNITED STATES, 1950–65

YEAR	ACCIDENTS			INFLUENZA AND PNEUMONIA†	DIABETES MELLITUS	CIRRHOSIS OF THE LIVER	TUBERCULOSIS, ALL FORMS
	Total	Motor Vehicle	Other				
	Males						
1965....	109.3	46.4	63.0	52.3	18.1	23.3	16.0
1964....	105.2	44.2	61.0	51.2	17.6	19.6	17.0
1963‡...	103.4	42.7	60.8	66.6	16.6	17.1	18.8
1962‡...	100.5	40.3	60.2	52.1	16.1	16.8	19.2
1961....	95.1	38.0	57.1	49.6	14.9	15.9	19.9
1960....	101.1	39.5	61.6	68.0	16.1	14.9	21.4
1959....	100.2	40.5	59.7	53.6	14.1	13.7	22.6
1958....	100.2	40.4	59.7	61.4	13.0	12.5	26.5
1957....	106.5	43.5	63.0	65.0	12.5	13.4	27.6
1956....	109.7	47.9	61.7	49.1	11.7	11.8	29.5
1955....	106.5	45.1	61.5	50.8	11.2	10.3	32.1
1954....	104.6	42.6	62.0	49.1	11.3	9.8	35.8
1953....	116.2	47.8	68.4	68.6	11.3	10.0	45.8
1952....	118.2	46.9	71.4	60.6	11.5	10.6	59.6
1951....	114.1	45.2	68.8	62.3	11.8	9.9	73.6
1950....	107.1	41.2	66.0	63.4	11.8	9.0	81.8
	Females						
1965....	35.8	13.7	22.1	32.3	28.6	13.4	6.4
1964....	34.9	12.5	22.5	31.4	29.0	11.3	6.7
1963‡...	36.1	11.8	24.3	45.1	29.1	11.0	7.3
1962‡...	35.4	11.4	24.0	35.4	27.1	11.0	8.1
1961....	33.9	11.0	23.0	30.5	26.7	9.9	8.5
1960....	36.1	10.6	25.5	43.3	26.8	9.1	9.3
1959....	34.6	10.8	23.8	35.2	24.2	9.1	10.9
1958....	35.8	10.2	25.6	39.0	24.3	7.4	11.9
1957....	38.4	12.5	25.9	46.4	23.6	8.4	12.8
1956....	37.3	12.5	24.8	34.6	22.2	7.8	14.8
1955....	38.5	12.6	25.8	33.9	21.6	6.8	16.5
1954....	37.8	11.8	26.0	33.8	21.2	6.5	19.1
1953....	39.8	12.3	27.5	49.1	22.9	6.3	23.9
1952....	41.6	12.4	29.2	44.0	21.5	5.7	32.7
1951....	41.0	12.5	28.6	47.2	21.3	6.3	42.8
1950....	38.8	11.1	27.7	50.6	22.6	5.9§	53.2

* Age-adjusted on the basis of the age distribution of the total population of the United States in the census of April 1, 1940.

† Excepting pneumonia of the newborn.

‡ United States, excluding New Jersey.

§ Age-adjusted rate based on age-specific rates more than half of which were based upon frequencies of less than 20 deaths.

SOURCE.—Unpublished data from the Division of Vital Statistics, National Center for Health Statistics.

diseases of the heart.[4] For these the adjusted death rate for white males at all ages has hovered around a level of 370 per 100,000 since 1952. On the other hand, females give evidence of recent improvement since 1960. Mortality from vascular lesions affecting the central nervous system declined markedly. Some of the decline may reflect better diagnosis, with an increasing allocation of deaths to better-recognized conditions. Another possibility is that the introduction of drugs for the control of elevated blood pressure may have reduced the incidence of cerebral hemorrhage. Better diagnosis may also account for the gradual decline in the death rates for general arteriosclerosis and for the sharp drop in the case of chronic and unspecified nephritis and other renal sclerosis.

The trend of death rates from cancer (malignant neoplasms) for males was consistently upward from 1950 to 1965, with but occasional minor dips, the increase amounting to 13 per cent. Contrary to this record for males, white females recorded a decrease of 9.5 per cent over the same interval. During this period, the death rate from cancer of the digestive system and peritoneum, the leading site of cancer deaths, declined among the white population. However, cancer of the respiratory system rose rapidly for both males and females; in recent years, this site, which consists principally of cancer of the lung, accounted for about one-sixth of all cancer deaths. Among males mortality from cancer of the respiratory system rose at all ages over 25, while among females the rise was concentrated at ages 25–64.

Accidents rank next to the major cardiovascular-renal diseases and cancer as a cause of death in the United States. Motor vehicle fatalities, the leading cause of accidental death, reached a low point in 1961 among males but rose rapidly in the few following years to 1965. In that year, the rate for motor vehicle accidents among white males was the highest within the period from 1950. White females also experienced a rapid rise in the motor vehicle accident rate in recent years. Among males the death rate from this type of accident is generally highest at ages 15–24, but the same level is closely approached at ages 65 and over. However, for females the rate is generally lowest at ages 15–24.

The death rates from influenza and pneumonia (except pneumonia of the newborn) show peaks in 1953, 1957, 1960, and 1963—years of influenza epidemics—but otherwise no trend is evident for the period as a whole. The widespread use of antibiotics and sulfa drugs has had a significant role in bringing mortality from pneumonia to its present low level.

[4] A. J. Klebba, *Mortality Trends in the United States, 1954–63* (Series 20, No. 2 [Washington, D.C.: National Center for Health Statistics, June, 1966]).

Within the period from 1950 to 1965, the death rates from diabetes mellitus remained stationary for white males and showed a slight improvement for white females.

Most spectacular is the reduction in mortality from tuberculosis since 1950. For white males the death rate in 1965 was only one-fifth of that in 1950; for females the ratio was only one-eighth. An important factor in this trend is the introduction of new chemotherapeutic agents for the control of tuberculosis in the early years of the period under study.

Mortality from the major chronic respiratory diseases other than tuberculosis and cancer of the lung and bronchi has increased rapidly during the survey period. Most significant in this trend is the marked rise for emphysema, but some of this may represent a shift in diagnosis and reporting practice away from other chronic respiratory diseases, such as asthma, bronchitis, and bronchiectasis.

Generation changes.—A picture of the recent progression in mortality from one age group to the next is obtained by comparing the death rate for an age group in 1949–51 with its rate when it becomes ten years older in 1959–61. Since the generation remains the same, the change reflects purely temporal influence, both environmental and what may be called the "aging process." For white females the per cent changes in generation or cohort mortality tend upward with advance in age to a peak at ages 75–84. However, progression of change in generation mortality for white males is different from that for white females. Thus the death rate for males at ages 15–24 in 1959–61, which reflects a high incidence of fatal accidents, was more than double that for the same generation when they were ten years younger, one decade earlier; for white females the corresponding rise was by a little over one-sixth. In distinction from white females, the approach of mid-life by white males brought with it a marked rise in generation mortality. Thus, for the generation of males that became ages 45–54 in 1959–61, the death rate increased by 138 per cent over what it experienced one decade earlier; for those 55–64 in 1959–61 the rise was by 122 per cent. Females of these ages recorded an increase of 93 per cent. For older generations of males the death rates doubled in passing from 1949–51 to 1959–61. Some preliminary findings of a study now under way show that this picture of change in generation mortality of males in mid-life may be a recent development in the United States and may be emerging in some other countries.

Mortality cross-over.—Comparisons of pairs of mortality curves for different populations often show that they cross about mid-life or shortly thereafter. The question arises why the population with the advantage in mortality at the younger ages loses it at the higher ages. No ready ex-

planation is evident for the crossing of mortality curves, nor may one appear until hypotheses are offered and tested. One such hypothesis is that the population with the better medical care will not only have greater control of the infectious diseases but will also have the benefit of a lengthening survival of its chronically ill.[5] If it is assumed that the incidence of chronic illness is subject to little control,[6] the lengthening survival will

[5] M. Spiegelman's discussion of R. J. Myers and F. Bayo, "Mortality of Workers Entitled to Old-Age Benefits under OASDI," *Transactions of the Society of Actuaries*, XVII (1965), 432.

[6] Actually, some chronic illness consequent to certain infections is controlled by prevention of the infections.

TABLE 14

DEATH RATES BY AGE, SEX, AND COLOR FOR THE UNITED STATES, 1949–51 AND 1959–61, AND THEIR PER CENT OF CHANGES SPECIFIC FOR AGE AND SPECIFIC FOR COHORT

Age Group	Death Rate per 100,000		Per Cent Change for:		Death Rate per 100,000		Per Cent Change for:	
	1949–51	1959–61	Age	Cohort	1949–51	1959–61	Age	Cohort
	White Males				White Females			
5–14....	69.9	52.9	−24.3	.....	46.1	34.3	−25.6	.....
15–24....	152.8	143.3	− 6.2	105.0	71.8	54.3	−24.4	17.8
25–34....	186.4	161.3	−13.5	5.6	113.9	85.5	−24.9	19.1
35–44....	384.9	329.1	−14.5	76.6	236.5	187.4	−20.8	64.5
45–54....	990.3	917.6	− 7.3	138.4	551.2	456.6	−17.2	93.1
55–64....	2,313.8	2,200.0	− 4.9	122.2	1,299.5	1,063.1	−18.2	92.9
65–74....	4,858.2	4,764.7	− 1.9	105.9	3,243.9	2,755.4	−15.1	112.0
75–84....	10,533.3	10,181.7	− 3.3	109.6	8,489.2	7,619.7	−10.2	134.9
85 and over...	22,074.9	21,611.3	− 2.1	105.2	19,770.2	19,271.9	− 2.5	127.0
	Nonwhite Males				Nonwhite Females			
5–14....	101.4	73.5	−27.5	.....	74.8	51.6	−31.0	.....
15–24....	291.6	213.5	−26.8	110.6	217.1	109.2	−49.7	46.0
25–34....	492.5	387.9	−21.2	33.0	386.0	252.2	−34.7	16.2
35–44....	866.5	724.1	−16.4	47.0	752.0	538.4	−28.4	39.5
45–54....	1,874.0	1,508.6	−19.5	74.1	1,568.1	1,122.1	−28.4	49.2
55–64....	3,655.1	3,073.3	−15.9	64.0	3,012.6	2,360.4	−21.7	50.5
65–74....	5,210.3	5,537.2	6.3	51.5	3,987.1	3,907.1	− 2.0	29.7
75–84....	8,858.4	8,408.6	− 5.1	61.4	7,151.9	6,626.2	− 7.4	66.2
85 and over...	15,820.3	14,776.3	− 6.6	66.8	13,101.3	12,776.6	− 2.5	78.6

result in increasing the proportions of chronically ill at the older ages.[7] Since the chronically ill have a higher mortality than those not so afflicted, a situation is created which may account for the higher mortality at the older ages in the population with the better medical care.

In view of the rapid expansion of medical care in the United States since 1950, the same hypothesis may be considered in seeking an explanation for the lack of mortality reduction at ages 65 and over for white males and the slackening improvement for white females. In fact, it is possible that advances in medical care of the chronically ill benefiting all stages of life may eventually produce a reversal of mortality at the very high ages.

No attempt has yet been made to reduce the hypothesis to a form suited for testing. Whatever the form may be, it will necessarily include such morbidity elements as variations in incidence of disease and of survival after the onset of chronic illness. It is a complicated task.

Mortality projections.—Mortality projections are made for many purposes, and the approaches taken toward them will be influenced accordingly.[8] Consider, for example, the attitudes that may be taken toward the reliability of mortality projections. The vital statistician may be asked to estimate mortality as a standard of accomplishment, assuming widespread use of the best in current medical knowledge, including patterns of medical care and public health administration. He would naturally look for the best mortality experiences currently available and the circumstances that produced them, compare trends, and then set down a list of assumptions as a guide to arrive at his projections. The vital statistician may or may not propose a range of variation for his projections to indicate the extent of his uncertainty. On the other hand, the demographer concerned with population projections makes use of survival rates. Since these are complementary to mortality rates which are relatively small, they are not very sensitive to a moderate range of variation in the projected mortality. Accordingly, the demographer will generally use only one set of projected survival rates, in place of a range, for population projections.[9] This is not the case with the actuary in social security who

[7] M. S. Goldstein, "Theory of the Survival of the Unfit," *Journal of the National Medical Association*, XLVII (July, 1955), 223; H. V. Muhsam, "Mode of Life and Longevity in Israel," *Jewish Journal of Sociology*, VIII (June, 1966), 39; and G. A. Sacher, "The Gompertz Transformation in the Study of the Injury-Mortality Relationship: Application to Late Radiation Effects and Ageing," in *Radiation and Ageing*, ed. P. J. Lindop and G. A. Sacher (London: Taylor & Francis, Ltd., 1966).

[8] This section is elaborated in *Introduction to Demography*, the 1955 edition published by the Society of Actuaries and the 1968 edition to be published by the Harvard University Press.

[9] Bureau of the Census, *Projections of the Population of the United States, by Age, Sex, and Color to 1990, with Extensions of Total Population to 2015* ("Current Population

makes use of population projections for his cost estimates.[10] In order to express the extent of his uncertainty in these estimates, his population projections will contain a range of variation; each of the elements entering into this population projection, including mortality, will have a range of variation.

The actuary estimating costs for annuity or pension plans may also take into account variations in mortality projections, but he will necessarily adopt only one set for his final purpose. He differs, further, in one important respect from the vital statistician, the demographer, and the actuary in social security. Whereas they are concerned solely with population mortality, he is required to consider particular bodies of annuitants or pensioners, each with its own characteristics. Even when these particular experiences contain a basis for projecting mortality, they will be influenced by the factors affecting population mortality; for that reason alone they require study of both their past and probable trends.

CHAIRMAN LEW: We now come to the more important applications of mortality trends in insurance. The first example is that of the use of mortality projections in connection with social security benefits. Over the years Bob Myers has made this field his own.

DR. ROBERT J. MYERS: My remarks will be directed primarily to the topic of projecting mortality rates for the purpose of population forecasts and social insurance projections. I shall, however, deal briefly with the near-future mortality prospects for persons aged 65 and over.

There is a very direct interrelationship, in the projection of mortality rates, between population forecasts and social insurance projections insofar as the work of the Social Security Administration is concerned. From the very inception of the social security program our cost estimates have been founded on population projections for the country as a whole, so that, to make cost estimates, we have had first to make population projections.

The cost estimates prepared in 1934 for the Committee on Economic Security, which made the studies underlying the original Social Security Act, were based on a single population projection that assumed constant

---

Reports," Series P-25, No. 359, February 20, 1967). See also earlier reports cited in this source.

[10] F. Bayo, *United States Population Projections for OASDHI Cost Estimates* ("Actuarial Study No. 62" [Washington, D.C.: Office of the Actuary, Social Security Administration, December, 1966]).

age-specific mortality rates into the future at the level of the 1920–29 experience. Cost estimates prepared shortly after the enactment of the Social Security Act took into account, as an alternative, mortality that decreased in the future in accordance with the medium assumptions set forth by the National Resources Committee in its recently released study.

In subsequent years the cost estimates for the social security system were based on population projections that used low- and high-mortality-assumption trends—in both cases with declines in mortality being assumed. At first, the various alternative assumptions of the National Resources Committee and its successor, the National Resources Planning Board, were used. Later the actuarial staff of the Social Security Administration, with the co-operation and counsel of an eminent group of actuaries and demographers, developed its own assumptions with regard to the future trend of mortality to be used in the underlying population projections.

In population projections presented in 1952 ("Actuarial Study No. 33"), future mortality rates were developed by mathematical-projection methods based on the reductions that had occurred in 1940–48. The high-mortality assumptions were determined from the corresponding low-mortality assumptions by assuming that the latter rates for 1975 would be the same as the former rates for the year 2000 (assumed to be the ultimate condition, with level rates thereafter).

In population projections presented in 1957 ("Actuarial Study No. 46"), the projection of future mortality rates was accomplished in an entirely different manner. The low mortality and high mortality rates were developed independently. In each instance, mortality rates for the year 2000 (thereafter assumed level) were derived by projecting current rates subdivided into ten broad groups of causes of death. For each such cause assumed percentages of reduction were applied (separately by sex and, generally, by quinquennial age groups) to obtain the rate for the year 2000.

In our latest population projections, presented at the end of 1966 ("Actuarial Study No. 62"), the same general procedure was followed, except that it was decided that sufficient evidence of likely future trends was not available to establish independent estimates for low-mortality and high-mortality assumptions. Instead, the low mortality rates were developed in the same general manner as those in the previous projection, and the high mortality rates were derived by assuming that the improvement in mortality over the period would be half of that assumed for the low mortality rates. The assumed declines in mortality for the low mortality rates were reduced compared with the previous projection, in light of

the experience that had developed since the previous projections were prepared. As a matter of fact, the current projections are significantly less optimistic (i.e., less decline in mortality rates than in the previous ones was assumed) at almost all ages.

It was, in part, for this reason that the over-all cost estimates for the OASDI system that were prepared on the basis of the new population projections showed a lower cost (relative to taxable payroll) than the previous cost estimates and thus indicated a relatively favorable actuarial balance for the OASDI system. Digressing for a moment, we may point out that this favorable actuarial status has been used to finance a substantial proportion of the benefit increases in the legislation that the Administration recommended to Congress and that the Congress is now acting upon.

An interesting sidelight in our mortality projections has been the treatment of the well-known differential between male and female mortality. In the early projections it was assumed that—in logic—this gap, which had been widening in the past, would be reduced over the long-range future. Over the years, since the first projections were made, the experience has been just the reverse—the gap has been broadening. As a result, in our current projections we are assuming a slightly greater future improvement in mortality for females than for males, which implies the assumption of a continuation of a widening of the mortality gap between the two sexes.

Finally, I should like to discuss the aspect of mortality trends for persons aged 65 and over. Over the long-run past there had been slow but significant decreases in mortality rates for this age group, but during the last decade there has been a virtual leveling-off. A widely held theory, which has considerable logic and even some statistical proof, is that, when mortality improves greatly at the younger ages, this will result in many weakened or inferior lives attaining the older ages, so that mortality rates at that stage will be much less likely to decline.

As evidence of this, there is cited the fact that mortality rates for persons aged 65 and over in economically poor countries are frequently reported at about the same level as those in the economically well-developed countries with high standards of living and health care. This same situation also prevails in the United States between the white and nonwhite populations, for whom there is little difference in the reported mortality rates at the older ages despite significant differentials at the younger ages. Another indication is presented in our "Actuarial Study No. 60," in which reasonably accurate data for OASDI beneficiaries seem to indicate that,

at the very oldest ages, the differential between male and female mortality narrows and perhaps even becomes nonexistent or reverses.

Nonetheless, an opposite viewpoint seems to have considerable logic—that, at least in many instances, the elimination of certain causes of mortality at the younger ages does not mean that an impaired life survives but rather that a healthy life completely avoids a certain cause of death and, with various medical advances, is likely to have lower mortality at the older ages than would have been the case in the past. The truth of the situation perhaps lies somewhere between the two extremes—that some of the improvement in mortality at the younger ages results in more impaired lives surviving to the older ages, while other such improvements have no effect on the mortality status of the survivors (or else even have a favorable effect).

Some supporters of the Medicare program, in urging its adoption, asserted that it would lead to better health care for the aged, many of whom were not being adequately cared for, and, as a result, that it would result in decreasing mortality for this age category. For a theoretical standpoint, it is possible that a group can be furnished better medical care and yet their mortality may be only little affected. For example, the medical care might result in better health while living, with less pain and suffering, but perhaps with little or no extension of life.

It is as yet too early to draw any sound conclusions on this matter. Nonetheless, the Health Commissioner of New York City is reported to have stated recently that he believes that one of the reasons for the decline in the nation-wide crude death rate in the first half of 1967 was the increase in the use of medical facilities because of the Medicare and Medicaid programs. Actually, the crude death rate for all ages combined decreased by about 4 per cent in the first half of 1967 compared with the corresponding period of 1966. Although this is statistically quite significant, it is not really a large absolute change and is quite possibly due to the absence in 1967 of any sizable influenza or respiratory disease epidemic.

I have computed the death rates for persons aged 65 and over (divided into ages 65–74, 75–84, and 85 and over) for the first twelve months of operation of the Medicare program compared with similar periods in the preceding five years. For each of these age groups, the rate in the first year of Medicare is somewhat lower than that for the previous year. However, for ages 65–74 the latest rate is at about the same level as that in a number of the preceding years, so that no real downward trend is evident. On the other hand, for the two oldest age groups there appears to be a slow downward trend in the mortality rate for the six-year period con-

sidered, which is more pronounced for ages 75–84. Therefore, it may be said that the decrease in the Medicare year for these groups was more or less a continuation of recent trends. Only for the oldest group, 85 and over, was there a really sharp drop in the latest year (about 8 per cent), but this may have been due to an accidental fluctuation for this relatively small group. Further, it may be noted that over the last decade there has been some evidence of an increase in mortality for this category, despite the slightly decreasing trend that had occurred in the last few years. In other words, any decrease that is now occurring may have the effect of only coming back to a level that had prevailed a number of years ago.

In any event, it will be most interesting and important over the next few years to study the developing mortality experience for persons aged 65 and over.

CHAIRMAN LEW: In no area of the actuary's responsibilities is a full understanding of mortality downtrends more important than in the determination of rates for annuity contracts. The Teachers Insurance and Annuity Association of America has for a long time devoted a great deal of attention to mortality trends among annuitants, and in recent years this problem has been in Bob Duncan's capable hands. Bob will now tell us how these issues have been approached in his company.

MR. ROBERT M. DUNCAN: In the broad field of mortality trends under discussion here this afternoon, I'm going to limit myself primarily to annuity mortality under contracts issued by life insurance companies in the United States and Canada, and, within this grouping, I am going to concentrate for the most part on individual rather than group annuities. You will easily see my particular interest in this area if I mention in passing that my company has had for many years a tradition of reporting the dollar amount of its insurance and annuity policy reserves as greater than the face amount of its life insurance in force—and the company is not a writer of group annuities.

At the outset, there is one very important point which I would like to stress, because it affects the approach that we, as actuaries, can never forget as we go about developing methods of mortality projections. This has to do with the inevitable assumptions and judgments which must be cranked into the process before we can arrive at a set of figures with dollar signs on them. In making these assumptions and in developing our methods, we must keep in mind the purpose of the mortality projections. Because of the secular trend of mortality, therefore, in the case of annuity mortality it is obviously necessary for us to be more conservative than for

insurance or population mortality projections or, for that matter, for social security coverage over a long span of years, because of the different arrangements available to meet the costs of paying the eventual benefits. Along the same line, too, the problem is presented to us of what provisions should be made as between deferred annuities and immediate annuities. A deferred annuity issued to a man aged 25 calls for quite a bit more prudence on our part than an immediate annuity now issued to a man at age 65.

What sort of base do we have to build on? Time does not permit a detailed accounting of the various mortality experiences showing the lengthening of annuitant lifetimes, faster during some periods than others, such as the last ten or fifteen years when the change has been small or of no practical significance for many types of annuities. But was it not Santayana who once said that those who have not learned the lessons of history will be forced to relive it? So perhaps a quick summary of the important annuity mortality developments over the last twenty years will be useful in bringing this all together. I will try not to bore you with a lot of figures. These are all published in our literature and have been ably organized by our hard-working Committees on Mortality and Morbidity.

Of course, the prime data for this specialized field are our own *Reports of Mortality and Morbidity Experience*, but we must also look to the other comparable experiences already mentioned by the previous speakers. I have listed here four major points which arise from the 1948–63 annuity mortality experience and related data.

1. Mortality rates among annuitants, insured lives, and the general population have been fairly level in general for the last ten to fifteen years. However, male nonrefund annuity mortality (by income) continued to decline in the financially important age range of 60–79 and declined markedly by income at ages 60–69 in the last five-year period studied. Also, the intercompany group annuity mortality experience for retirements on or after the normal retirement date shows small improvements in mortality, by amount of income, for both male and female over the entire fifteen-year period ending in 1965.

2. We now know that selection at issue of immediate annuities has more effect than had been observed in earlier studies. This was generally thought, 20 years ago, to extend for only two to three years. Based on this significant 1948–63 experience, selection on nonrefund annuities persisted for three to four years at ages 60–69, for at least five years at ages 70–79, and even longer over age 80. This appears to have more effect than the assumptions that have usually been made about improvements in mortality at ages over 75 and does not appear to have much effect at ages under 65.

3. The difference in annuitant life expectancies between males and females continues to widen. This effect is also shown in general population studies of

white males and white females. Looking at the two most important causes of death, which are, of course, cancer and cardiovascular-renal diseases, we find a rise in cancer deaths for males and little change in male cardiovascular-renal deaths. There has been little change in female cancer death rates, and cardiovascular-renal rates have been declining for females. Recent group annuity mortality experience also shows female death rates continuing to improve while the male rates were fairly level. The same phenomenon shows up in the experience of several categories in the reports on self-administered retirement plans.

4. Differences in mortality on different types of annuities have been narrowing. In fact, the differences between nonrefund and refund annuities on female lives have been quite slight in recent years.

What do we take from all this as we consider what projections we should make with regard to the future course of annuity mortality? Certainly we are going to take into account our new-found knowledge on the four points that I have just mentioned; but there are many other variables, many other questions, some clues, and very few answers.

First, let us consider the other variables and questions. Among the important factors that should be mentioned as affecting future mortality are nutrition; occupation; retirement-age patterns; urbanization; housing; evolution of marriage and family-unit patterns; education; advances in medical knowledge; availability of medical services through Medicare and Medicaid, as well as through private means; and the general economic standard of living. Obviously, there is also an interrelation among many of these factors, to say nothing about other factors which I have probably neglected to mention. We could spend a great deal of time discussing some of the items on this list, but we do not have the time, except for a few brief observations about one or two of them.

Let us give some thought to medical science. Just what is the potential from the single factor of advances in medical science? For all practical purposes, we are now discussing only cancer and cardiovascular-renal causes of death as having any real significance among medical causes, since our good friends the doctors have taken care of almost everything else along this line. Consider the effects of a 15 per cent reduction in death rates for each of these two causes on annuity values for a man aged 65. I will have to admit that these are pretty healthy reductions but certainly not what you would term ridiculous assumptions. Very approximately, then, for cancer this amounts to an age setback of almost a third of a year, or $1\frac{1}{2}$ per cent of the single premium; for cardiovascular-renal diseases, the age setback is about $1\frac{1}{2}$ years, or 6 per cent of the single premium. In the annuity business, these are important percentages. A few weeks ago I heard Dr. Wesley Hall, who is chairman of the board of the American

Medical Association, predict substantial reductions in the near future for deaths from both cancer and cardiovascular-renal causes. I was fascinated to hear what is going on in the field of internal cancer surgery by way of pinpoint accuracy and advances in surgical safety through the use of laser beams. With regard to developments in prevention, cancer immunization by cell-transfer techniques has already been proved with animals and the prognosis is good that the technique can be perfected for humans. As to cardiovascular-renal diseases, we are much further along with artificial-heart and heart-component developments than we were a few years ago, and other organ transplants are on the way, according to Dr. Hall.

What of the effects of Medicare and Medicaid? It is perhaps too soon to judge, but we know that older Americans have received about 15 per cent more inpatient hospital service since Medicare started, and we do know that the nation's death rates dropped far below normal in each of the first twenty weeks of 1967. I'm not saying that there is necessarily a cause-and-effect relationship in this case, but it is nevertheless something to think about. On the other side we see an upward trend of respiratory diseases, probably due to our increased urbanization and air pollution.

My own view is that prudence indicates that some allowance should be made for increased longevity, in spite of the experience of the last ten or fifteen years. We have had periods in the past when mortality has leveled out for a time, only to break out eventually on the down side and again start the recurring pattern of lengthening life expectancies. Perhaps something like Scale B, which produces smaller and smaller allowances to about age 90, is the most likely pattern. Certainly, in many periods of mortality improvement this sort of progression by advance in age has been quite typical and would appear to be consistent with the Gompertzian theories of the general wearing-down of the human mechanism. One way of looking at this is to consider the last fifteen years as an interruption or plateau in the long historical trend of the advancements of knowledge, the improvements in standard of living, and the attendant advances in longevity.

Finally, let me say a few words on actuarial methods that have been employed in building provisions for mortality trends into annuity rates. Although each actuary must decide on the constants in a scale of improvement to be employed as most appropriate to the particular population with which he is working and the type of benefits to be made available, it is hard to see how we can improve very much on the basic principle used by Ed Lew and Wilmer Jenkins in their development of scales for the a-1949 Table. This principle is that the rates of improvement in annuity mortality depend solely on attained age and the calendar year

of exposure and is now considered superior to the older "generation" methods which based projections principally by year of birth. The approach used in the *a*-1949 Scales allows easily for consistent treatment of deferred and immediate annuities. There are other practical advantages, particularly for those of us in the less-than-giant size operations. By using a standard table—such as *a*-1949 Scale B—which has been widely recognized and continues to be highly valuable as a representative pattern in many situations, it is possible by the judicious use of age setbacks and standard improvement scales starting at earlier or later calendar years to adjust to many different benefit patterns or to other mortality projection considerations in establishing the mortality basis of our premium rates.

C. M. Sternhell made the job easier for us when he used the same principle to develop special commutation columns for use in projecting mortality improvement in the calculation of approximate annuity values. (See *TSA*, Vol. II; see also Vol. XIII for a paper that he wrote with C. H. Page on certain modifications of *a*-1949 and Scale B, particularly at the high ages.)

J. E. Hoskins (*TSA*, Vol. IV) showed that very good results can be obtained in deriving *a*-1949 Scale B annuity values by merely reducing male ages by 0.075 years and females by 0.06 years for each year by which the year of birth exceeds 1875.

In projecting mortality and mortality trends, some allowance for fluctuation is usually made and can take several forms. I have seen the following used, either singly or in combinations:

1. Percentage of premium.
2. Standard deviation of annuity value.
3. Difference in annuity values on different tables from different periods of exposure.
4. Difference in annuity values calculated from the basic mortality table and then from graded percentages of the death rates in the basic mortality table.

I am sure that there are others, but the main point here is to build in some allowance for fluctuation. We have come a long way in our knowledge of annuity mortality—and this is my final point—but we cannot project mortality rates with close accuracy and it is only good sense to make provision for this.

CHAIRMAN LEW: Before throwing the meeting open for discussion, I would like to call on a prominent Norwegian actuary, Dr. Tønnes Ore. Dr. Ore is the director of a statistical office of Norwegian life insurance companies and has given much thought to mortality trends.

DR. TØNNES K. ORE: I am very glad to have the opportunity to attend this meeting and to be with my American colleagues on their home grounds. My office in Oslo is an actuarial-statistical one and serves all Norwegian life insurance companies. Our principal jobs include statistical studies of mortality among insured lives and annuitants, construction of new mortality tables, calculation of new premium rates, and so on. Each year we also make valuations for several companies. The organization is similar to that of a consulting office, but it is owned and sponsored by the companies. The chief actuaries of the companies decide what they want the office to do.

Before I left Oslo, I made a comparison between the mortality rates in Norway and those of the white population in the United States. The comparison revealed that the rates of mortality in the United States are considerably higher than those in Norway in the age range from 10 to 75. American death rates are from 20 to 80 per cent higher than the Norwegian.

An actuary needs to ask what the reasons are for the higher mortality in the United States. Since your country enjoys high standards of medical service and since medical science is quite advanced here, the reasons for higher mortality rates must be sought elsewhere. There are a number of facts which a visitor cannot avoid noticing when he travels—many more heavy smokers, a great deal of stress, and, last but not least, more pronounced obesity than in any other country that I have seen.

The impact of obesity on longevity is well known. One of the best pieces of evidence on the close correlation between the amount of food consumed and mortality rates is to be found in the Norwegian experience during the German occupation. The food rations were very small, and the consumption of fat was microscopic. We nevertheless experienced a considerable drop in mortality rates (except for war deaths) at the middle and advanced ages. I would not recommend such rations over a long period of time, but our experience leads to the conclusion that the human body runs better and healthier with a smaller amount of food than we customarily like to take.

Judging by the papers just read, it would appear that the mortality trends during the past ten to fifteen years show many similarities for our two countries but also some noticeable differences. The recent trends in Norwegian mortality may briefly be summarized as follows:

1. At the younger ages, mortality is still declining in both sexes—though less so than in earlier periods.
2. A tendency for male mortality to rise at the middle and older ages has been observed since about 1950. Since then, mortality rates among men at ages over 35 have risen by 10 to 20 per cent.

3. Mortality rates among women have continued to decrease in the middle-age groups but at a slower rate than before; mortality rates among older women have more or less stopped declining.

The picture is no longer as clear-cut as it was twenty to thirty years ago when mortality rates were declining continuously in both sexes at most ages. The recent experience is confusing, considering all the sanitary and other measures introduced by authorities in the interest of public health, as well as the continuous progress of medical science and research.

A closer examination shows that the adverse trends are due chiefly to three distinct causes of death—road traffic accidents, lung cancer, and coronary heart disease (myocardial infarction). From this we can fairly draw the conclusion that the change in the trend of mortality rates is due mainly to human factors—ways of living and human behavior. This represents a great challenge to our health authorities and to the life insurance business.

Mortality statistics throughout the world have given a lucid picture of the large gains in health and longevity during the past fifty years, resulting from progress in medical science, sanitary regulations, and increasingly higher living standards. Attention now needs to be paid to the impact of human behavior on health and longevity and to how human behavior might be influenced for the better. We need more information and education, and the psychological approach to influencing human behavior is very important.

With regard to projections of future mortality, I have no confidence in forecasting based on extrapolation from the mortality experience in recent years because of the heterogeneous trends in the current situation. In my country, prognoses based on statistical or technical considerations would be of theoretical interest only. I believe that the problem should be approached through studies of the trend of the more important causes of death. Due regard should also be paid to several other factors: the results to be expected from medical research, further improvements in health measures, better traffic control, and so forth. The outlook is likely to vary from one age group to another, and I find the forecasting problem to be very complex at this time.

MR. HENRY BRIGHT: I have a question for Dr. Moriyama. He said that, if cancer were eliminated as a cause of death, the total death rate would decrease by a certain percentage. He said 16 per cent.

I wanted to ask whether this 16 per cent, or whatever percentage it is, was derived by applying the residual rates of death to the population distribution that is being worked with. The point that I am making is that, if you eliminate a specific cause of death, you inevitably shift your

population distribution, and eventually, of course, the total death rate must return to the original level, because distributionally the total death rate is going to be a constant percentage.

I have an additional thought on the same subject. It seems that a more meaningful statistic for measuring the effects of the elimination of a particular cause of death would be to develop a mature population using the rates of death under consideration and then to eliminate a particular cause of death, develop a new stable age distribution, and compare the age-specific rates resulting.

DR. MORIYAMA: In making those estimates I was trying to give a broad indication of what could happen. I was merely using proportionate mortalities, just a rough measure of the possible consequences of, say, the elimination of cancer mortality.

DR. THOMAS N. E. GREVILLE: Since I had something to do with producing those figures, maybe I can clarify this a little. These proportionate mortality rates were applied to a life-table population. What we calculated was the life expectancy at birth in the life table, with the assumption that these reductions in mortality would occur.

MR. L. JEFFERSON STULCE: The so-called trend of mortality is undoubtedly the sum of many trends. Different segments of the population experience different levels of mortality, and the mortality trend for one segment may be quite different from that for another. The total population can be grouped and subgrouped in countless ways, and some will be more meaningful and significant than others. To this novice demographer, it would seem that there is implicit in any carefully refined projection of population mortality trends at least the following steps:

1. Identify major groupings of the population which have distinct mortality levels (that is, mortality differing significantly from average).
2. Obtain a realistic measure of the group's characteristic mortality (separate by age, sex, etc.).
3. Estimate and project future changes in mortality for each groups.
4. Project the population growth within each of the particular population groupings.
5. Combine Step 3 and Step 4 results so as to develop a projection for the total population which reflects shifting from one subpopulation group to another.

We do not know nearly enough about significant subpopulation groupings—either about their identity or about their distinct mortality trends. We do not know as much as we ought to know, nor as much as we could have known by now, had we exercised all the curiosity, the initiative, and

the resourcefulness that we would like to claim for our profession and for our industry.

With but a few exceptions, we have come to look increasingly to the Public Health Service, the National Institutes of Health, and the universities (and other research groups) to gather data and to study, publish, and interpret mortality (and morbidity) statistics for population groupings. We are indebted to these organizations for mortality studies indicating mortality and morbidity differences according to such things as state of residence, educational level, marital status, ethnic origin, diet characteristics, use of tranquilizers and other such things, and, last but not least, the statistical relationship between excessive mortality and a factor that I will not mention, just yet, by name. Let us call it Factor SS.

There have been numerous studies, quite convincing to me, dealing with Factor SS. For several years the issues have seemed about as controversial as the round-earth versus the flat-earth question. And yet, controversial they remain.

Several years ago a "matched life" study was undertaken which would eliminate or minimize various biases while shedding new light on Factor SS. In this study each participant was classified according to at least a dozen or more individual characteristics deemed to have some bearing on mortality. Individual lives were paired off, by computer, in such a way that each pair of men was identical in each of these important characteristics except for Factor SS, where one was positive and the other negative. Each participant who could not be exactly matched with an opposite Factor SS was eliminated from the study. The final results showed, among other things, that more than twice as many deaths occurred among the matched lives where Factor SS was positive than among the equal number where Factor SS was negative. This study was among those reported in detail in the May, 1964, issue of the *Journal of the National Cancer Institute.*

Another study reported on in the same article showed perhaps even more astounding results. For the details I would refer you to Table 7, on page 1172, of the *Journal.* The following results are shown in Table 7 (all death rates were age-standardized):

1. The college graduate has appreciably better mortality than persons with less than eight years of schooling, *but* college graduates with Factor SS positive had appreciably *worse* mortality than persons with Factor SS negative who had less than eight years of schooling.
2. Married men had appreciably better mortality than single and divorced men. However, married men with Factor SS positive had worse mortality than the latter group with Factor SS negative.
3. Negro males had substantially higher mortality than white males. However,

white males with Factor SS positive had higher mortality than Negro males with Factor SS negative.

So it went, all down the list—family history, selected occupations, exercise habits, use of tranquilizers and other common medicines, extent of nervous tension, consumption of fried foods, and so forth. This study showed interesting statistical relationships between each of these and the resulting mortality levels—and in each instance the relationship was exactly as you would expect. However, this statistical relationship was *reversed* in each instance where persons associated with Factor SS positive were selected from the "favorable" group and compared with persons with Factor SS negative in the "unfavorable" group. Would you agree, then, that Factor SS positive is associated with excessive mortality and therefore merits consideration in making mortality projections?

Suppose that Factor SS were a controllable factor—unlike family history and certain physical impairments. Suppose that it was the eating of cranberries that led each year to 300,000 needless deaths. Would we in our projection of mortality trends consider the possibility of an escalation of the cranberry crave in the general population? Would we regard the possibility of a loss in cranberry popularity as a factor to consider in projecting mortality trends? And how quickly would our industry seize on this opportunity to make a public-spirited announcement—to exercise its powerful persuasion and influence—to steer people away from cranberries.

Our industry and our profession have not exhibited enough concern about Factor SS. Today we have listened to this distinguished panel of experts discourse for two hours and twenty-two minutes about factors influencing mortality, mortality trends, making mortality projections, and so on, and their erudite and scholarly discussion has interested and impressed us all. Yet not one mention has been made of Factor SS. One gentleman listed perhaps fifteen separate factors which would influence mortality in the future, and still others were mentioned by other speakers, but not a single mention was made of Factor SS.

It seems that Factor SS is a highly delicate subject, likely to give offense, and not fit for polite society. Well, you may as well brace yourselves, because I am going to come right out and say it anyway. By SS I mean "Smoke Screen"—the smoke screen that has been surrounding and obscuring the cigarette hazard. "Factor SS positive" refers, of course, to those who *do* smoke cigarettes. The smoke screens emitted by the tobacco companies have served to keep the public confused and undecided for a number of years. However, with cigarettes now labeled as a

major health hazard by the Public Health Service and the American Cancer Society, some people are beginning to ask why actuaries and insurance statisticians have exhibited a relative lack of interest in this subject.

Consider now the bystander's bewilderment at our industry's apparent indifference to the disturbing statistics published in the late fifties and the early sixties and even after the Surgeon General's report. Picture a huge industry, with business operations mathematically precise, management skills meticulously honed for scientific orientation, management policies directed to the public interest, and corporate goals aimed altruistically toward enriching the lives of the 200 million people of this country, providing them security and promising hope that they might achieve their highest aspirations for their families, without regard to life's perils. In considering the response of these honored old institutions, add in a gigantic financial stake in the health and longevity of the nation's people. What would you consider an appropriate response from these good companies to the challenge posed by the cigarette hazard, which is affecting their highest purposes and aims, costing millions of dollars annually in terms of excessive mortality claims, and creating through wasted human resources a vast loss to the nation?

For years, with controversy raging this way and that, with many policyholders confused by conflicting claims, and with some policyholders' lives perhaps in the balance, what information have the insurers of the nation's health communicated to its policyholders? Knowing that our silence might be misunderstood by many (and to their own detriment), what public stand have we taken? How has this response affected our public image? A few companies, it is true, have quietly called attention to the hazards of cigarette smoking, and more recently several companies have begun to offer lower rates to nonsmokers. These companies are taking a clear-cut public stand, conveying a message to the public, and helping to resolve some of the doubt and confusion in their customers' minds.

Perhaps by now you have the impression that I think one of the answers to question 111 is cigarette smoking. That is true. But how can we include the excess deaths due to cigarette smoking in our mortality projections without evaluating future social acceptance of smoking and other outside influences bearing on the spread of this habit? We do have the opportunity to influence the acceptance of smoking to a degree and thus to help lower the mortality of our policyholders.

It seems altogether possible that the leveling of mortality in recent years is the purely accidental result of very different trends among major

subgroups of the population. We may continue with leveling mortality for the total population, or conceivably even end up with worsening mortality—if there is a shifting in the distribution of our population from nonsmokers to smokers, from one-pack-a-day to two-pack-a-day men, and so on. Even with gradually improving mortality within each subgroup, we could end up with level or worsening mortality over all, if there is a shifting in the distribution of our population into more unfavorable categories.

Such a development might lead to a reversal in the trend of female mortality, assuming that smoking continues to be socially "correct." Among women a disturbing transition in smoking usage is under way. To develop this point a bit, I would refer you to Monograph 19, published in January, 1966, by the National Cancer Institute of the Public Health Service.

As a preliminary step, look at page 133, which shows mortality ratios according to the number of cigarettes smoked per day (using nonsmokers' death rates as standard). These mortality ratios increase as the number of cigarettes increase. For illustration the ratios for ages 45–54 are as follows: for males this ratio increases from 184 to 226 to 241 to 276 per cent for 1–9 cigarettes, 10–19 cigarettes, 20–39 cigarettes, and 40 or more cigarettes; for women these ratios increase from 95 to 122 to 154 to 196 per cent.

Page 134 shows mortality ratios according to whether the subject regarded his degree of inhalation as none, slight, moderate, or deep. Using ages 45–54 again, these ratios are 205, 214, 242, and 254 per cent for males; for females these ratios increase from 101 to 121 to 130 to 178 per cent.

Page 135 shows mortality ratios according to the age at which the subject started smoking cigarettes. The age categories start at age 30 or older and move progressively down to "below age 15," and the resulting mortality ratios increase consistently as starting age reduces. Using attained ages 45–54 again as an illustration, this page shows the ratios for males to be 140, 181, 213, 249, and 301 per cent; for females these mortality ratios are 110, 129, 130, and 138 per cent. The last figure shown is for starting ages 15–19, since the females in this study at attained ages 45–54 who started smoking before age 15 produced less than ten expected deaths.

Page 182 shows the distribution of females covered by attained age and also according to the ages at which they started cigarette smoking. Among other things, it shows that females now aged 55 and over most often did not start to smoke until they had passed age 30. It is, of course, above age 55 where we have the most deaths, and also most deaths that

are linked with cigarette smoking. Looking at the attained-age groups below age 49, however, we find in each instance that the largest number of women started to smoke between ages 15 and 19. This suggests that when this younger group of women moves into the higher attained-age groupings, where most excess deaths occur, they will then have an average of perhaps forty or more years of cigarette smoking, whereas most women now in their late fifties and early sixties have smoked only twenty or twenty-five years.

Pages 178 and 180 show somewhat similarly that younger women today smoke appreciably more cigarettes, and they also inhale more than older women. Consequently, the women who will be maturing in age during the next twenty years would be expected to increasingly surpass today's mature women in each of these three unfavorable characteristics—duration of smoking, number of cigarettes, and degree of inhalation.

MR. SPIEGELMAN: The previous speaker's comments are important. I want to emphasize, however, that several actuaries have been active in studies of cancer mortality, especially with reference to the problem of cigarette smoking.

Our chairman, Mr. Edward Lew, has for many years been a statistical consultant and member of the Statistical Committee of the American Cancer Society. He has had a hand in some of these studies which have profoundly influenced our thinking on the issues to which Mr. Stulce referred.

In the review section of the *Transactions* of the Society, I have had a number of reviews and digests of investigations dealing with smoking, cancer, and related matters. In fact, in a recent issue of *The Actuary*, you will find a review of the latest study of smoking and cancer made by the Public Health Service. Instead of taking a back seat in the thinking on this question, I believe that we can actually boast of the part we have played.

Finally, I am pleased to report that several years ago I had the privilege of serving as consultant to the Advisory Committee on Smoking and Health of the Surgeon General of the Public Health Service. Actuaries should continue to take part in mortality studies of this kind and we need not be at all ashamed of our past activities in this area.

MR. WILBUR M. BOLTON: In partial answer to Mr. Stulce, I recall that four years ago in Atlantic City there was a session attended by a large number of actuaries, at which several speakers talked about smoking and death rates.

I have been told of two basic reasons why large companies do not seem

to be interested in issuing special policies to nonsmokers. The first reason is the underwriting argument. If an applicant states that he has not smoked for the last two years, how can you tell whether he is telling the truth? The answer to this presumably is whether he has cigarette stains on his fingers. At any rate, the company I was with until last year endeavored to underwrite the smoking hazard on this basis. The second reason is that at this time a company issuing a special nonsmoker's policy would be offering a contract that would be of interest only to a small proportion of the total male population. I believe that actuaries are fully aware of the effects of smoking on over-all mortality, on cancer mortality, and on cardiovascular-renal mortality.

MR. HILLARY J. FISHER: The dramatic decline in United States population death rates in all age groups, which began in the late 1930's, was followed by a leveling-off of death rates during the middle and late 1950's and, more recently and for some age-sex-color groups, by an upturn in death rates.[1] Several influences which account for this change in trend may be distinguished:

1. The 1938–53 decline was the result basically of the development and application of serum therapy, chemotherapy, and vaccines for the treatment and prevention of infectious diseases. Death rates in 1955 for a number of these diseases were less than 10 per cent of the death rates in 1940. Although rates continued to drop by as much as 5 per cent or more a year, the numerical decreases in death rates since 1955 have been much smaller fractions of the aggregate death rates for all causes combined.

2. As a result, the trend of aggregate death rates by age group in recent years has been governed largely by the trends in the death rates for the various chronic diseases, such as cardiovascular-renal diseases and malignant neoplasms in the older adult population; for malignant neoplasms and accidents, homicide, and suicide in the younger population; and for congenital malformations among children.[2]

While death rates for some important chronic diseases have fallen steadily, death rates for others have remained stationary or tended to creep upward. For still others, particularly the chronic bronchopulmonary diseases, death rates have been rising dramatically. Deaths of younger and middle-aged adults from accidents and other violence, on the whole, have been increasing and probably still are increasing rather rapidly. For any particular age-color-sex group, then, the net trend in the death rate currently is likely to be stationary or increasing rather than decreasing.

[1] Iwao M. Moriyama, *The Change in Mortality Trend in the United States* (Public Health Service Publication No. 1,000, Series 3, No. 1 [March, 1964]), pp. 1, 5, 7.

[2] *Op. cit.*, p. 38.

3. A third factor is significant, although it actually is a corollary of the first two influences. This factor is the effect of influenza-pneumonia epidemics on aggregate mortality. It is well recognized that epidemics and localized outbreaks of influenza are accompanied by excess deaths from all causes, which are at least five or ten times the excess deaths attributed to influenza alone. The death rate for pneumonia, especially, rises far above the average during a "flu" epidemic.

Other causes for which excess mortality is highly correlated with influenza epidemics are the chronic bronchopulmonary diseases and the cardiovascular diseases. As the severity and, possibly, the frequency of influenza epidemics have declined as the result of more effective treatment and more widespread vaccination, there has been a corresponding decline in secondary excess deaths from other causes. Since the severity of influenza epidemics has been reduced to a relatively low level because of increased immunity resulting from exposure and/or vaccination, as evidenced by the decline in mortality from influenza itself to a very small fraction of its former rate, influenza should have little effect on the future trends in mortality for the chronic diseases, although appearance of a new strain of virus may temporarily increase aggregate mortality rates.

What *is* the present trend of mortality? To shorten the discussion of this question, I shall consider only adults from about 25 to 64 years of age. In the period 1955–60 death rates for the population in this age range were slowly decreasing, except those for white males over 45, which were slowly increasing.[3] We can make a rough, educated guess at the current rates of change by grouping causes of death into those with decreasing and those with increasing rates. (A more accurate estimate might be made by considering each cause separately.) If the average rates of change for recent years are applied to the latest available mortality rates, separately for those causes with decreasing rates and those causes with increasing rates, one possible aggregate rate of change can be estimated. (Other estimates can be obtained by assuming that the rates of decrease or of increase will be various multiples of the recent averages.)

Assuming that the average percentage decrease or increase in mortality rate in recent years, for each age-sex-color group, continues for each cause of death, the indication is that death rates for white men are increasing about 1 per cent or so per year at ages 25–44 but are about stationary at ages 45–64; that death rates for white women are stationary at ages 25–34, increasing 1 per cent to 2 per cent per year at ages 35–54, but decreasing about 1 per cent a year at ages 55–64; that death rates for nonwhite men are increasing 2 to 3 per cent per year at ages 25–54 but decreasing at higher ages; and that death rates for nonwhite women are

[3] *Op. cit.*, p. 10.

decreasing at least 1 per cent a year at ages 25–44 and 55–64 but are stationary at ages 45–54.

What causes of death are the principal determinants of the current mortality trend? First, we might consider which causes show the strongest upward trends in each sex-color-age group.

In the younger age groups—except nonwhite women—accidental and other violent death rates are increasing the fastest. Motor vehicle accidents rank first or second, measured by increases in death rates, for white men and women from 25–44 and for nonwhite men aged 25–34; they are high, too, for older whites and for some older nonwhites. Nonmotor vehicle accidents rank first, measured by increases in death rates, for nonwhite men aged 25–34, and they rank high for some older nonwhite men. Suicide ranks first for white women 35–44 and is an important cause, measured by numerical increase in rate, for other white women up through age 54 and for white and nonwhite men 25–34. Homicide death rates are increasing rapidly for all nonwhite men and for younger white men.

For older adults and for nonwhites aged 35–44, increases in death rates are largest for malignant neoplasms of the respiratory system and breast or for certain other chronic diseases—emphysema, bronchitis, arteriosclerotic (coronary) heart disease, miscellaneous vascular diseases (aortic aneurysm, phlebitis, Buerger's disease, etc.), cirrhosis of the liver, or diabetes mellitus.

Table 1 lists by age-color-sex group the causes recently and currently responsible for appreciable increases in death rates (number of "x's" indicates relative rapidity of increase). From the table it is clear that the major problems of increasing mortality concern respiratory cancer, emphysema (among whites), coronaries, cirrhosis of the liver, and motor vehicle accidents. Of only slightly less importance are homicide, suicide, diabetes mellitus, and the group of vascular diseases that includes aneurysm, phlebitis, and Buerger's disease. For certain groups, increases in breast cancer mortality, in bronchitis mortality, and in nonmotor vehicle accidental death rates are important.

While the number of causes of death exhibiting substantial decreases in mortality rates is small, the size of the decreases for some causes is surprisingly large. By far the largest contributor—or group of contributors—to improvement in mortality at the adult ages has been the cardiovascular-renal group, excluding arteriosclerotic (coronary) heart disease and the miscellaneous vascular diseases mentioned before. Included are vascular lesions affecting the central nervous system (strokes), hypertensive heart disease and other hypertensive disease, generalized arterio-

sclerosis, rheumatic fever and chronic rheumatic heart disease,[4] endocarditis, and chronic nephritis and renal sclerosis. Even coronary death rates improved for whites in some age groups. These cardiovascular-renal diseases currently account for one-fourth to one-third of the total decreases at the younger ages and for 75 per cent or more of the total decreases at the older ages. Evidently drugs for the amelioration of hypertension have been a major factor contributing to these decreases. If so, then the rates of decrease should be slowing down. Some new breakthrough will be needed to insure continued progress on this front.

The only cancer mortality rates that seem to be decreasing appreciably are the rates for malignant neoplasms of the digestive system and of the genital organs. The decreases for digestive system cancer, which have been of significant benefit to whites over 45 and to nonwhites over 55, are largely attributable to decreases in stomach cancer mortality, since the trend for other sites generally is mixed or, in several cases, upward. Women of all ages will gain from further substantial decreases in death rates for cancer of the reproductive organs, provided widespread periodical examinations are promoted to insure early detection.

Little mortality gain can be expected for the infectious and parasitic diseases. With tuberculosis mortality rates decreasing 5 to 10 per cent a year for all groups in the 25–64 age range, the gains in deaths per 100,000 are rapidly becoming smaller and smaller. In the immediate future, however, decreases in tuberculosis mortality rates will contribute appreciably to total mortality gains for older white men and for all nonwhites. The largest decreases for nonwhites aged 25–34 probably will be due to improvement in the tuberculosis death rate.

Maternal mortality rates for white women are now so low—1 to 3 per 100,000—that further improvement will have only a very slight effect on their aggregate mortality rate. For nonwhite women, however, the death rate is higher and appreciable numerical reductions in death rates undoubtedly will be made in the next few years.

In analyzing the mortality trend, those causes should not be overlooked for which the trend is level, or nearly so, or indeterminate. Pneumonia, which is the cause of about 3 per cent of all deaths, shows a wide cyclical variation in deaths per thousand, but the basic pneumonia death rate, after several decades of decline, seems to have been slowly increasing during the last decade. The death rate for stomach ulcer is neither improving nor worsening, nor are the death rates, in general, for cancer of the pharynx, cancer of the urinary organs, or leukemia and aleukemia.

[4] Some of the apparent decrease for this category resulted from a change in coding classification rules in 1963.

TABLE 1

Age	Malignant Neoplasm of Respiratory System	Malignant Neoplasm of Breast	Bronchitis	Emphysema	Arteriosclerotic Heart Disease	Miscellaneous Vascular Diseases	Cirrhosis of Liver	Diabetes Mellitus	Motor Vehicle Accident	Nonmotor Vehicle Accident	Suicide	Homicide
White Males												
25–34									XXX		XX	XX
35–44	XX				XX		X	X	XXX			
45–54	XXX			XX		X	XX		XX			
55–64	XXX		X	XXX	XX	XX	XX		X			
White Females												
25–34									XXX	X	XX	
35–44	XX	X			X	X	X		XXX	X	XXX	
45–54	XXX	XXX		X	XXX	X	XX		XX		XX	
55–64	XXX	X	X	XX		X	XXX		XX	X		
Nonwhite Males												
25–34					X	X	XX		XXX	XXX	X	XX
35–44	XX				XXX		XXX	XX	XX	X		XXX
45–54	XXX				XXX	X	XX			X		X
55–64	XXX			XX	XXX	X	XX	X				
Nonwhite Females												
25–34						XXX	XXX					
35–44	X				X	XX	XXX		X			
45–54	X	XXX		X	X	XXX	XX	XXX				
55–64	XX				XXX	X		XXX	X			

There have been no significant changes recently in the nonmotor vehicle accidental death rates at ages 25–64 for white males or for nonwhite females.

This discussion would not be complete without mention of the fact that there has been a substantial increase during the past few years, for practically every age-color-sex group, in the number of deaths recorded under the category "Senility and Ill-defined Conditions." At the younger- and middle-adult ages, such deaths presumably would be due mostly to ill-defined conditions. The recorded increase may be due to a greater percentage of deaths with causes not accurately diagnosed because of the shortage of medical attendants. On the other hand, it may indicate an increased desire on the part of more attendants to make a careful and accurate diagnosis when possible. Either assumption seems to be supported by the large increases in deaths of nonwhites assigned to this category. Also, because of the frequent difficulty in distinguishing between the underlying cause and a contributing cause, the former may be stated in terms too general for specific classification—as, for example, "CVR disease," or "chronic respiratory disease." Causes which bear a social stigma sometimes may not be truthfully recorded; if so, some decreases in recorded death rates may not be as large as they seem.[5]

MR. LEROY V. BOTKIN: I have seen statistics showing that there are much higher mortality rates for people who drink, too. I see from observation that there seems to be a high correlation between those who smoke and those who drink. I wonder if there have been any results of studies made of nonsmoking drinkers and smoking drinkers, and smokers and nonsmokers. I have no axe to grind, because I am guilty of both.

CHAIRMAN LEW: In the years to come, we will need to have a great deal more fundamental understanding about mortality rates. Much of it can come only from biologists. I am therefore going to ask Dr. Strehler again to comment on what he has heard here and to indicate what in his judgment are some of the possibilities for greater longevity.

DR. STREHLER: Some of you may know of Hardin Jones's analysis of the relationship between age-specific death rates and smoking. You may recall that, if we plot the log of the probability of death against age, we get a Gompertz-type curve for virtually all populations; for smokers the curve is merely advanced in time so as to approximately double the

[5] See Metropolitan Life Insurance Company, "On the Confidential Reporting of Causes of Death," *Statistical Bulletin* (June, 1948).

normal mortality rate. In effect, the average cigarette smoker can be considered by actuaries as an individual who is about seven years older than his chronological age. In like fashion, exposure to ionizing radiation (in doses of 300 or 400 roentgens total exposure) also increases age-specific death rate by a factor of about 2.

In this connection I would like to point out another fact, which is not widely appreciated. It is that, if we were to eliminate all deaths due to heart diseases and cancer, we would simply move down the Gompertz curve by approximately ten years. This is somewhat more of a shift in the opposite direction than is accomplished by the average cigarette smoker.

Finally, we might ask, "What is the possibility that we could change the slope of the Gompertz curve?" The answer depends, of course, upon the mechanism of aging. Nevertheless, I would like to point out to you that there are at least two experimental situations in which the Gompertz slope of experimental animals has been effectively reduced.

The first of these was the famous McKay experiment, in which the life expectancy of rats was doubled by drastically underfeeding them for a substantial portion of the animals' lifetime. This experiment suggests that there is a relationship between the total amount of metabolism which takes place and the wear and tear on the machine; this is perfectly consistent with the well-known fact that obese individuals have a lower life expectancy than do slim persons.

The second type of experiment is one which may have more profound and immediate implications for actuarial projections. Loeb and Northrup showed in 1912 that the rate of aging of cold-blooded animals decreased by a factor of 2 for about every 7 or 8° C. that the temperature is lowered. In other words, fruit flies, trout, rotifers, and campanularia all experienced increased life expectancy when their body temperatures were lowered. We now know that this applies to vertebrates, our close relatives, as well as to a variety of invertebrates. Apparently what happens is that all metabolic functions, including aging, are slowed down when body temperature is reduced. Are these studies germane to man?

There are pharmacological materials that can reset the internal thermostat of experimental animals, such as mice, to a lower level. Normally, the small thermostat in the human brain tells our body to turn up the heat when temperature drops; we then begin to shiver and thereby increase our metabolism. Conversely, if body temperature rises above the thermostat setting, we tend to lose heat, for instance, by sweating. Because of this sensitive internal device, we are automatically programed to age at a certain rate.

I would therefore pose the following question: If human beings react essentially in the same way as simpler animals, what would be the effect of setting the thermostat in the human brain down by 2° C. (to 35° C.)? My answer is that we could add twenty to twenty-five years to life expectancy. This is more than one would expect from the discovery of cures for all the major diseases.

In my judgment, there are startling implications for you in the results of research relating to aging. Depending on the outcome of such research, the results could be very disturbing for those of you who are primarily in the annuity business. On the other hand, they would be very cheering to the life insurance business. Since I am interested in understanding the problem, I would like to appeal to the insurance people to support the research that needs to be carried on.