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LONGEVITY AND GENETIC ENGINEERING

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MR. A. ANTHONY AUTIN, JR.: Our purpose today is to broaden the perspective of the practicing actuary by discussing the possible effects of genetic engineering on our policyholders and clients. To this end my role will be to describe genetic engineering and to discuss the part it may play in changes in longevity. My fellow panelist, Robert Collett, will discuss the effect of such changes on current mortality trends and will comment on how actuaries can anticipate and respond to such changes.

A logical question is: "How important is genetic engineering as a means of changing life patterns?" This is patently obvious when we learn that researchers have found over 1,000 diseases which are genetically based and expect to discover another 3,000 by the year 2000. A quick look at some of the diseases that are considered to be genetically related can amplify this for us. Researchers have included in this category, cancer, heart disease, stroke, arthritis, diabetes, senile dementia, auto-immune disease, cystic fibrosis, hemophilia, muscular dystrophy, sickle-cell anemia, leukemia, atherosclerosis and hypertension. These diseases can be the result of various genetic disorders such as failure of the cell to produce the correct type or quantity of enzyme or hormone, failure to divide on schedule or to repair normal wear and tear.

While there is a temptation to extrapolate broadly and esoterically from the results being achieved today in the field of genetic engineering, we shall attempt to look objectively at the current state of the art. A frequent statement by futurists is that consideration of the possible futures requires an ability to perceive the realities of the present. In an evolving, rapidly developing field such as genetics, achieving an accurate perception is a challenge.

Definition of Genetic Engineering

It is necessary that we define genetic engineering before we delve into this topic. We will lead into this definition by discussing genes and their role in the human organism. Briefly, genes are protein compounds which are combined from base chains on only 4 distinct nucleotides in a unique form for each person (other than identical twins) to form the now familiar DNA molecule. It is believed that nearly every cell in the normal human body contains the full complement of genes, some 50,000 in total, according to one estimate and 6,000,000 by another, identical to that found in the initial fertilized egg cell. The genes constitute, if you will, the blue-print or library of instructions required to develop, maintain and, as we shall see, possibly destroy the body.

As well known as DNA is today, it may only play a passive role in the cell. The leading role may belong to other elements which control the utilization of the genetic information contained in the DNA. Whether such elements are

the RNA message carriers of the cells or something else is not yet settled. Some researchers report that elements called histines act as covers to prevent certain genes from being accessed by the RNA. The controller of such histines could then be the true leaders of cell function.

Genetic engineering, as we will use it in this discussion, will mean the utilization of the available knowledge and understanding of the role of genes to change or maintain cellular functioning. The application of this knowledge may be within the genes themselves, outside of the cell nucleus where the genes are housed or even outside of the cell itself. This definition is broader than that used by some writers. Many of these use the term more restrictively to describe the field of recombinant DNA research wherein changes in genes are the desired result. For our purposes such changes will be considered along with engineering designed to improve or change cellular functioning without such changes.

Recent Emergence of Genetic Engineering

Genetic engineering is truly an emerging field. The modern start of the field was perhaps the double helix conceptualization of the DNA structure by Crick and Watson in 1956. Within 5 years the genetic code had been cracked. Within 11 years DNA had been synthesized and within 20 years a working gene had been synthesized out of shelf chemicals and implanted in a bacterial cell. Today, scientists have mapped over 200 of the approximately 50,000 genes existing in the 23 pairs of human chromosomes.

What then is the current state of the art in this field of genetic engineering? This can be judged by the following collection of results reported by researchers within the last 5 years:

1. In 1974, researchers used a virus to fuse different cells together.

2. In 1975, human and plant cells were fused and grown in combinations; leading to suggestions that plants could be made to produce needed human-type enzymes.

3. In 1976, cancer researchers treated patients, who had had lung cancer removed, with weakened tuberculosis organisms to stimulate the patient's own immune system against further lung cancer. Other researchers used antigens removed from cancer surfaces as direct coding messengers for the body's regular immune system.

4. In 1976, University of Alabama researchers reported that an anti-dental caries oral vaccine could be available within a few years. The vaccine used killed streptococcus mutans bacteria to trigger antibodies in the gastro-intestinal tract (the source of antibodies for salivary glands).

5. Specific genes known to be implicated in serious genetic disorders have been mapped (e.g. tay-sachs and lesch-nyhan syndromes).

6. Certain DNA rings called plasmids exist outside of chromosomes and specific enzymes have been isolated which split open the rings long enough to permit insertion of new genes.

7. Some scientists have recently applied for patents for processes using new kinds of bacteria that can create biochemical factories producing antibiotics and antibodies.

8. In May, 1978, Toledo, Ohio researchers isolated genes which control hypertension in rats.

9. In October, 1978, synthetic genes were inserted into E Coli, a harmless bacterium commonly found in the human intestinal tract, resulting in the production of human insulin.

To a large extent, genetic engineering is yet an experimental science and, as such, not one ready for widespread application in the field of medicine. Yet many see the potential for near-term usable results. As an example, the 1977 edition of "Current Medical Diagnosis and Treatment" contains a chapter on medical genetics which begins with the following statement: "The rapid advance of the science of genetics in recent years has so many applications to clinical medicine, that a knowledge of basic genetic principles is now a necessity for diagnostic purposes."

Extending Life Expectancies

Let us look at some of the results in genetic engineering which would tend to extend life expectancies within the 100-120 year maximum life span most feel is the current upper limit of life. Clearly the current life expectancies at birth of around 70 years for males and 75 for females leave considerable room for increases within such a limit.

Earlier, we listed a number of diseases that are considered genetically related. Of these, the major causes of death for ages over 35 are the cardiovascular and related diseases. They represent 30% of overall mortality rates in the 35-44 age range, 55% in the 65-74 range and 65% in the 85 and over range. One form of genetic research underway now could significantly reduce mortality from these causes.

The heart, like most muscles in the body, consists of post-mitotic cells, cells which have ceased dividing. In the way of contrast, skin cells continue dividing throughout life. This distinction results in the well-known permanent damage to hearts due to infarctions. Since the heart cell's nucleus contains the necessary blueprint to make new cells, it is theoretically possible, say some researchers, to teach or reteach the heart cells how to divide again and to replace the damaged cells. One form of the research involves the introduction of cancer cells into the heart in an attempt to transfer the cancer cell's dividing ability to the heart cells. For obvious reasons, this research is proceeding very carefully and has only reached the test-tube or "in vitro" stage of experimenting for human cells.

The second major cause of death averaging 20-25% of mortality rates for ages between 35 and 85 is cancer. Cancers represent cells that have lost the ability to stop dividing or perhaps are somehow prevented from getting the message that they should stop dividing. Some scientists suggest that cancer cells are relatively common in the human body but are normally controlled by the body's very effective immune system.

One attack on cancer is based on the theory that the thymus gland's efficiency decreases with age. The hormone thymosin is the trigger that causes the bone marrow to produce T cells. The latter are part of the body's attack mechanism on foreign cells. Researchers have found that bovine (cow) thymosin can be effectively used to replace the diminished supply of this hormone and can perhaps help reduce the incidence of cancer. This is an example of an immunological approach which led some experts to the prediction that cancer will be cured by the year 2000. Parenthetically, last week's newspapers, here in the New Orleans area, carried an announcement of successful cancer treatment based on the use of cancer antigens. As noted earlier in this presentation, these are surface elements on cancer cells which have been isolated and used to give the immunological forces in the body a clear identification of the invading cancer cells. The announcement reported the opinion of one researcher that an effective immunization technique against many forms of cancer was now within reach.

Over the 1968-76 period, overall mortality rates in the U.S. improved by 13% for white males and 17% for females. In the same period of time, mortality rates from cancer increased 4% for males and did not change at all for females. Obviously a cure in the next 21 years would represent a significant discontinuity in current trends.

While not as dramatic in potential, genetic research is underway in areas which could impact favorably on diabetes, hypertension and infectious diseases. Since such diseases may tend to produce many deaths classified as heart attacks, the true impact may be larger than one would infer from observing the death rates from these diseases.

Although faced by substantial technical challenges, another area of potential is cloning of humans. One area of applications is in the area of transplanted organs. Normal bodies search out and destroy any foreign tissue which enters them. Tissues from a body's clone would theoretically be indistinguishable to the body's defense mechanism from those in the original body. The specific use of a person's own clones as sources of non-rejectable replacements for disease ravaged parts is then clear. But other uses of clones, for example as a source of young T and B cells to aid an aging body's normal supply of these essential immunological components has been suggested.

So far, we have talked about the potential for increase in life expectancy as a result of progress in genetic engineering against disease. But, many feel that the elimination of the current major causes of death would not mean we will live forever.

In a 1976 address before the national symposium on Suggested State Legislation for the Elderly, noted British Gerontologist and author, Alex Comfort, had the following to say on this topic: "Advanced societies are now approaching the practical limits of public health in prolonging life. While the expectation of life at birth has increased steadily over the last century, the expectation at 65 years has changed little, if at all, having risen by only 2 years since 1901. It is computed that the total cure of the 3 leading causes of natural death in the U.S. (cardiovascular, cerebrovascular, and malignant diseases) while greatly beneficial to those who contract them young, would only increase the mean expectation of life at age 65 by 2.5 years."

Extending Life Spans

We have previously noted a 100 to 120 year maximum life span. While not a precise limit, there does appear to be a point by which everyone dies. This ultimate cause of death is not known to us; but actuaries might call it the omega factor. Here we enter the field of gerontology and a brief review of the major theories of aging is in order. One such theory is called the "Error Hypothesis". This hypothesis suggests that cell operations are imperfect and that errors occasionally result. While normal cell operations correct many errors and repair any damage, some go undetected or uncorrected. Eventually, the accumulative errors mount up to a level which results in a nonreversible trend in cellular disfunction ending with death. Reducing errors or improving the reparative capacity of the cells would tend to counter this effect and prolong life.

In the "Free Radical Hypothesis", an electrically imbalanced particle attaches itself to other cell molecules when it should not. This interferes with the cell functioning and could lead to death. Researchers have found elements called anti-oxidants which tend to reduce the level of free-radicals in the body. Vitamin E is one such element.

Another theory is the "Cross-Linkage Theory". Here, cross-links between protein molecules interfere with their regular operations. For example, RNA, the messenger which carries coded information from the DNA nucleus to the operational part of the cell, may be prevented from accessing the DNA due to cross-linkage.

In the "Auto-Immune Theory", the body's defense mechanism (B-cells, T-cells and microphages) begins to attack its own normal cells rather than just foreign cells. It literally kills itself. Here research suggests that the use of thymosin, restricted diets or removal of the spleen may be effective.

The "Brain-Clock Hypothesis" proposed by the Gerontologist, Denckla, states that the brain signals the pituitary to release a blocking hormone (DECO). This hormone prevents the cells from using thyroxine secreted by the thyroid. The result is a number of critical imbalances in cellular functioning and death. Prevention of this hormone release could prevent aging.

Finally, there is the "Cellular Aging Clock Theory" by Leonard Hayflick of Stanford, who demonstrated that human cells will divide only about 50 times in vitro. He suggests that the cell is programmed to this limit and could theoretically be taught to re-program itself for a higher limit. One researcher has found that vitamin E would double the Hayflick limit of human cells in vitro. Another found that cells that were deep-frozen had their limits suspended during their hibernation. Upon unfreezing, they again began to divide but would still be restricted by the original limit.

The balance of support for these theories tends to move with each new discovery. However, at the present time, the concensus lies with the clock theories of Denckla and Hayflick. Some suggest that both theories are accurate but one is primary and the other is a failsafe (back-up) mechanism. Whichever theory turns out to be the correct omega factor, it is generally agreed that some break-through in the aging process itself will be necessary to extend the life span beyond 120 years.

Some Restraints on Genetic Engineering

We should be aware that there is a growing countermovement against some forms of genetic engineering, in particular the recombinant DNA research. It began in 1974 when leading researchers declared a voluntary moratorium on several types of experiments judged to be conceivably risky. Their concern was over the creation of, and possible escape into the environment of, new forms of bacteria for which humans had no effective defense. In 1976, the National Institute of Health released guidelines for such experiments. Many are not satisfied with the guidelines. Others object to any genetic tampering on the basis of religious or moral beliefs. At a minimum, some type of regulation of genetic engineering can be expected, at least in this country.

Parenthetically, an August, 1978 article in the Journal of the American Medical Association reported on the difficulties some research labs have had in obtaining liability insurance covering recombinant DNA research. The author noted that most liability policies did not contain exclusion clauses for accidents from DNA experiments. He indicated that this picture is rapidly changing as carriers become more knowledgeable.

Summary

Summarizing, the field of genetic engineering, as we have broadly defined it, does appear to have the potential for significant impact on longevity. The timing and extent of the impact is not clear.

Some predictions by experts include the following:

1. 1974 Rand Corporation survey of 82 gerontologists predicted the addition of 20 years to life spans by 1991.

2. 1975 Third Survey of Technological Breakthrough and Widespread Applications of Significant Technical Developments indicates that by the year 2000 we would see --
   - Drugs to prevent and cure cancer
   - Chemical control over hereditary characteristics
   - Chemical control over the aging process

3. Vance Packard, in his recent book, "The People Shapers" suggests that direct genetic engineering in humans before 1984 is unlikely, but that by the year 2000, control of single gene caused defects would be seen.

Whether the above types of changes would influence our work as actuaries is the subject of our next speaker, Robert Collett.

MR. ROBERT L. COLLETT:

I. WHAT HAS BEEN HAPPENING (WHERE ARE WE COMING FROM)?

I will begin my presentation with remarks about what appears to have been happening to life expectancies during the last 75 or so years. During that time frame, the United States life expectancy at birth has increased from 49 years to approximately 72 years (about 69 or 70 years for males and about

76 years for females). My first table, Table A, shows changes in life expectancies at various ages for white males and white females. The figures are taken from population tables.

TABLE A

LIFE EXPECTANCIES

(Population Data)

Age	In 1900	In 1975
White Males		
0	48	69
45	24	28
75	7	9
White Females		
0	50	76
45	26	35
75	7	11

Life expectancy measured from birth has been extended by more than 20 years in this century. At age 75, it has been extended only about two to four years.

The latest population information I have reviewed suggests that there have been important reductions in mortaltiy rates since the late 1960's also. As Tony indicated earlier, sources show a reduction in overall male mortality from 1968 to 1976 of approximately 13%. The female reduction during the same period has been about 17%.

Information from the Metropolitan Life Insurance Company on standard Ordinary lives show that during the period of 1969 through 1976, male mortality rates decreased by some 23% and female rates by about 16%. So, there have been significant improvements in this century and in the last decade. One reason is the virtual elimination of many infectious diseases as causes of death, through the development and application of penicillin and other wonder drugs. Our increased affluence undoubtedly has been a factor, since it allows us to afford better nutrition and medical care.

II. TRENDS IN MORTALITY RATES BY CAUSE OF DEATH

Let us consider a couple of particularly important causes of death: cancer and cardiovascular problems. Table B shows changes in cancer mortality rates during the 1960's and into the 1970's for the general population and for insured lives:

TABLE B

CHANGES IN CANCER DEATH RATES

(Population Data)

	Male	Female
1960 to 1967	+6%	-2%
1968 to 1976	+5	0

(Insured Data)

	Male	Female
1962 to 1968	+1%	+5%
1969 to 1976	-11	-3

There are some differences between the population figures and the Metropolitan Life's insured experience. The insured trend is the more favorable one. Not shown in Table B, but apparent if one looks at results by age group, is the fact that greater strides have been made in reducing younger age mortality rates, with lesser strides at the older ages.

The next table, Table C, is a comparison of a similar type for cardiovascular problems. Incidentally, when I use the term "cardiovascular," I mean to include heart and artery problems of all types, including cerebrovascular problems.

TABLE C

CHANGES IN CARDIOVASCULAR DEATH RATES

(Population Data)

	Male	Female
1960 to 1967	-5%	-10%
1968 to 1976	-18	-23

(Insured Data)

1962 to 1968	-6%	+9%
1969 to 1976	-29	-24

Here the pattern clearly is one of improving mortality rates. Further, there are improvements in every age group with the exception of the youngest age category, where the data is not conclusive. Increases in rates at younger ages may reflect important decreases in other causes of death for infants, or they may reflect more exact diagnoses of causes of death in infants.

We appear to have made some significant strides in dealing with heart disease in the last decade. In fact, during the 1970's, mortality from most causes, even accidents, improved. An extrapolation of the general trends of the 1970's if valid, shows much reduced rates of death within two generations.

III. WHAT IS LEFT TO BE ACCOMPLISHED?

Table D below shows the five principal causes of death in 1970 and it shows ranks for these causes at earlier points in time.

TABLE D

LEADING CAUSES OF DEATH

	RANK		
	1970	1940	1900
Heart Disease	1	1	4
Cancer	2	2	8
Cerebral Vascular	3	3	5
Accidents	4	6	7
Influenza and Pneumonia	5	5	1

The first three of these causes would seem to be where the greatest opportunities lie for improvements on account of genetic engineering. It is not apparent to me how accident rates can be significantly impacted by genetic engineering. Influenza and pneumonia may be adequately controlled at the younger ages by existing wonder drugs. Perhaps at the older ages influenza and pneumonia rates are subject to being affected by genetic engineering. Deaths from pneumonia and influenza at the older ages may be the result of other basic causes which perhaps could be treated through genetic engineering.

IV. IMPACT OF GENETIC ENGINEERING ON ACTUARIAL FUNCTIONS

A. Improvements in Life Expectancies Without Extensions in the Span of Life

Let us consider the impact on some familiar actuarial functions of major advancements in the cardiovascular and cancer areas. For my own convenience, I have chosen to use as my base table the 1968 U.S. White Population Table. This is a table which I had readily available in convenient form with breakdowns by cause of death. I will confine my figures to male values for age 65 in order to keep the volume of numbers down. Incidentally, in this table, the life span ends at age 107. In the 1968 table with no modifications, some familiar actuarial values are as shown below in Table E.

TABLE E

1968 U.S. WHITE MALES

ACTUARIAL VALUES

Age 65

	Values At 3%	6%
e_x	12 Years	
$\ddot{a}_x$	11	9
1000 A_x	$691	$506
1000 P_x	$65.19	$57.91

What could happen if a particular cause of death were to be eliminated entirely? Before providing some arithmetical answers, let me restate some facts which probably are obvious. As rates from one cause of death go down, rates from others should be expected to increase. When cause "A" is eliminated, the opportunities to die from cause "B" are increased. Also, keep in mind the simple-minded nature of some of my calculations. In deleting one, while keeping other causes of death, I am assuming that they are totally independent. Many causes are not totally independent, and some are closely interrelated.

What would we find if cardiovascular diseases were to be eliminated entirely? Some possible results are shown in the following table. Table F shows percentage changes in the values of the previous table, in the event such elimination should occur.

TABLE F

% CHANGES IF CARDIOVASCULAR DEATHS ELIMINATED

Age 65

	3%	6%
e_x	+60%	
$\ddot{a}_x$	+41	+32%
1000 A_x	-19	-30
1000 P_x	-42	-48

Some researchers have expressed disappointment that life expectancy changes are not more spectacular when these actuarial exercises are carried out, especially since the likely near term occurence will be a reduction in the impact of these diseases, rather than their complete elimination. I disagree.

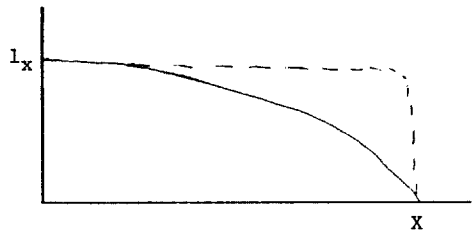
It is not shown, but the result of this exercise at age 35 is a 26% increase in the life expectancy. To me that is exciting, since it translates into 10 additional years of expected life. The 60% addition shown above for age 65 translates into eight more years.

In like manner we can test the impact of the elimination of cancer as a cause of death. The results in this case are much less dramatic. Cancer's elimination offers less promise than the elimination of heart disease. As a limiting case (but one which continues to respect the apparently existing limit on the span of life, represented by age 107 by our population table), suppose that all causes of death other than accidents were to be eliminated through genetic engineering and other efforts. In such case, we have expected ages of death near or above age 90 right from the time of birth. This kind of situation is sometimes referred to as "squaring the mortality curve." Chart G shows what is meant by that term.

CHART G

"SQUARING" OF

MORTALITY CURVE



_____ Today

-------- Tomorrow?

B. EXTENDING THE SPAN OF LIFE

Tony described how genetic engineering efforts are not limited to trying to eliminate major causes of death. Even with the elimination of the major causes of death, we have observed that there still seems to be a maximum limit on the number of years humans can live. Tony described how gerontologists are attempting to find the genetic keys to aging, in order to learn how to modify them. They seek to find the time clocks within us, which cause our bodies to cease functioning, although they do not know where these clocks are, or if, indeed, there really are such clocks. If the clocks were to be located and manipulated, conceivably the span of life could be extended to age 200 or even to age 250.

With a greatly expanded span of life, many different scenarios are possible. I will illustrate a couple of them. Suppose that we were able to stabilize mortality rates at the age 55 level, so that they would not rise above that level until some extremely advanced age, such as age 250. Table H shows what would happen to our actuarial values.

TABLE H

% CHANGES IF RATES ABOVE AGE 55

SAME AS AGE 55

	Age 65 3%	6%
e_x	+374%	
$\ddot{a}_x$	+111	+60%
1000 A_x	-50	-59
1000 P_x	-76	-74

For this case, the results are quite dramatic! Even for age zero, life expectancy is increased by about 50%. Life expectancy is increased by nearly 375% at age 65. It could be noted that the impact on monetary values is not quite as spectacular, due to the impact of discounting.

As another scenario for a greatly expanded span of life, consider a case where science succeeds in the future in reducing mortality rates to levels equal to levels now applicable to a person half as old. Put another way, let us presume a new mortality table containing twice as many ages, wherein the rate for a person age 70 is what we use today as applicable to a 35 year old, and so forth. Table I shows results.

TABLE I

% CHANGES IF DEATH RATES AT AGE X

EQUAL TO THOSE CURRENTLY AGE X/2

	Age 65 3%	6%
e_x	+386%	
$\ddot{a}_x$	+155	87%
1000 A_x	-69	-85
1000 P_x	-87	-92

This scenario might represent what we would see with a slowing of the rate of cell division breakthrough, in the vein Tony mentioned. The assumption produces the most extreme results of those examined today. Note the 92% reduction in the age 65 whole life net premium.

I am sure that none of these scenarios will prove to be exactly what will happen in the future. However, they do lead to some interesting results. Life expectancies are increased as specific causes of death are controlled, but the most striking increases occur when we postulate extended spans of life along with reduced mortality.

IV. IMPLICATIONS

If expectancies for a person age 65 might be doubled within our lifetime, or quadrupled in the next 100 years, we obviously have some real problems for nearly all types of annuities and life-settlement options. Should such changes occur suddenly, annuity companies might require liens in order to survive. Social Security would desperately need upward retirement age revisions. These seem to be coming anyway.

We have some problems on the life insurance side, as well. We would appear to be on the verge of making lots of money. In fact, though, mutual companies probably would deal with such a situation through greatly increased dividends. Perhaps non-participating companies would find it necessary to make unilateral "gifts" in order to head off wholesale lapsing or government intervention. Of course, mortality changes may come in gradual steps, so that the transition could be much more orderly than I have implied.

Some of the problems can be exaggerated, of course. On the pricing side, if one visualizes gross annual premiums based on high rates of assumed investment earnings, and with expenses and persistency included in the picture, one would see that it makes much less difference what happens to life expectancies.

What should companies do in anticipation of the possibilities of such changes? We will need products which will accommodate a long life. Probably such products will need to be able to deal with multiple partners or serial marriages over that long life. It seems to me that products surely will need built-in mechanisms to deal with inflation. It further seems that there will be many different stages of need over this long life. Products which lack flexibility or convertibility may become obsolete over time.

Long life expectancies may call for long-term term insurance or many-times-renewable term insurance. With a squaring of the mortality curve, so-called life expectancy term could become quite cheap and run to some fairly advanced ages. Single premium life insurance could be offered at much lower rates than at present.

We might have an opportunity to sell insurance policies which cover only specific external causes. I would think that accidental death insurance would be a very popular and important coverage. If the suicide risk increases (and I think it could), it may be that products which permanently exclude suicide could be significantly cheaper than those which cover the hazard. It also seems that we might have to be more concerned in our underwriting with the likely exposure of the individual to external hazards. In other words, a chemical worker might require a rate for expectancy term which would be five or 10 times the rate paid by someone not envisioned as ever being exposed to comparable hazards.

Speaking of underwriting, Tony has pointed out to me that if the genes hold the keys to the timing of natural death for an individual, then as we learn more about how to read that data, we may be able to read it for underwriting purposes. Perhaps a tissue sample taken from a prospect might show how much life he (as an individual) has left. This adds a whole new meaning to the term "life expectancy".

As for annuity products, maybe we will want to push term annuities to protect ourselves against unanticipated life extensions. Annuities certain or perpetuities might find favor because they do not contain the longevity risk. Also, they sound more attractive than life annuities while not really costing much more under several of the scenarios.

If life is extended, it is reasonable to expect that the period of healthy productive life will also be lengthened. Therefore, there could be very little need for an annuity with payments beginning at age 65. The continuing need in the annuity area would be for a short annuity to begin near the end of the healthy portion of the span of life.

MR. AUTIN: At the start of the session, I indicated that our purpose was to broaden the perspective of the practicing actuary by discussing the possible effects of genetic engineering on our policyholders and our clients. We definitely do not want to leave you with the impression that genetic engineering is the only element with which you should be concerned. It is only one of several. As actuaries, you need to be aware of some of the significant trends that are occurring in our society and developments either at the sociological, economic or physical level which may have a significant impact on our policyholders, the means by which we reach those policyholders and distribute our products, and perhaps their interest in buying our products. The implications of some of the scenarios that we have been drawing here are rather difficult to pin down, and at this point we would like to open the floor for possible comments. Maybe some of you haven't heard the term "genetic engineering" before you came in today. To put you a little at ease, those of us on the panel here really hadn't heard it much before you!

MR. DAVID E. MORRISON: I was wondering if there has been anything published on the impact of this extension of life on the economy? While longevity will be increased, quite likely the cost of medical care will be higher, for example. Also, there will be many more people potentially in the work force, and increased numbers of births. This will definitely have an impact on the whole economic support mechanism.

MR. AUTIN: I have not come across any material in the actuarial literature which will describe the impact of genetic engineering and even begin to address your question. I think you can begin to see however, just from general reasoning, some of the possible impacts. You mentioned that we would have more people in the work force; we may, as a first step have many people who are going to be retired for a lot longer. I think that this will lead to what you suggest, that people will stay in the work force much longer. That poses some significant challenges to society to find meaningful work for them. One of the most useful introductions to the subject is in the book by Vance Packard, that I mentioned, The People Shapers. And there are other books written by science editors. Among these "Prolongevity" by Albert Rosenfelt and "No more dying; the conquest of Aging and the extension of Human Life" by Joel Kurtzman and Phillip Gordon. I will be happy

to put together a biography and send it to anyone who would be interested.

MR. RICHARD W. GARNER: Please comment on the future that you see for the mortality differential by sex from a genetic standpoint?

MR. AUTIN: Throughout the first 70 years of this century the mortality differentials by sex have been widening rather then decreasing. I have seen nothing in the genetic literature that would give me any clue on whether or not that gap will be decreasing. Many of you have seen articles indicating that females entering the work force will now be subjected to, the same kinds of environment that males have been in for years, suggesting that it is environment that is the cause of the difference in mortality rates, not some basic genetic difference between the sexes. But, I haven't seen anybody really demonstrate that this is true.

MR. COLLETT: Above age 75, the death rates from heart disease are very close for the two sexes; the cancer rates of death, as a whole are higher for males, cerebral-vascular accidents are higher for females. There could be different results, therefore, depending on when the breakthrough comes and for which disease. On Dave Morrison's question, a couple of the things I have read suggest that if the length of life is added to greatly we would run into difficulties with accumulation of wealth. One writer was very concerned, if people had 125 years instead of 55 to accumulate money, the distribution of wealth in the economy would be even less widely dispersed. He would advocate some limitation on the accumulation of wealth to control that problem. Another stated that the only thing young people had going for them was youth, and if old people were given more youth or could retain their youthful health and appearance longer, young people would truly be at a disadvantage in society.

MR. STEVE ROCK: We've been talking about extending longevity through genetic engineering. We must also consider the possibility of inhibitive longevity through accidents in genetic engineering, through mistakes in genetic research or perhaps some radiation accidents, like the Harrisburg incident we just had. Is the Society going to investigate the effects such incidents have on mortality?

MR. AUTIN: Harrisburg might be categorized as an unfortuitous example of genetic engineering. Another example of a negative scenario could be envisioned in relation to the current high cost of reparative medical technology. We are, increasingly, finding it possible to sustain life for almost an indefinite period of time but at a high level of cost. Society is showing some tendency to view such cost as a severe economic disadvantage. In light of this, one might understand a tendency to quickly adopt any genetic engineering advances which promise to prevent disease but, if there are long term negative effects of that kind of genetic engineering which do not show up for several years, there could be a significant impact on the life insurance business. This would be the case if actuaries, after observing the initial improvements in mortality, had built expected gains into their guaranteed prices. I expect that society will adopt fairly quickly any genetic engineering advances which would avoid disease or prevent disease. But if there are any long term negative effects of that kind of genetic engineering which don't show up immediately, but are delayed for several years, there will be a significant impact on the life insurance business. Actuaries will reflect improved mortality, improved control of disease in their pricing, but will not have been able to observe the tail end of the mortality curve. That's

obviously somewhat of a frightening scenario.

You asked if the Society was involved in this kind of investigation? To my knowledge there is no work being done in this area, other than the occasional work by speakers such as myself or Bob. I think what we are suggesting here is that the members of the actuarial profession need to consider genetic engineering as just one of the factors that we take into consideration as we design products. If we consider the possibility of having a life insurance policy inforce for 200 years with long term guarantees, I'm not sure the design and features we build into our products today will necessarily be appropriate.

MR. HAROLD D. SINGH: Isn't it possible that the issue of genetic engineering may not ever apply to someone my age or older? Perhaps only in newborns will it have any significant effect. Therefore, the life policies we sell today may be quite realistic, in spite of advances in genetic engineering. Is that correct?

MR. AUTIN: The question is, really: "Will genetic engineering be something that can perhaps initially only be practical for future generations?" Can we avoid only genetic defects in the egg cells or the sperm cells in the current population, affecting future generations? Or, can we apply genetic engineering to people who are already around who already have genetic defects? The information that I have indicates that we're more likely to see initially the use of genetic engineering to have a positive effect in reducing future causes of disease or perhaps early mortality, through its effect on people yet to be born. However, many genetic engineers and many in the medical profession, do see by the end of this century, a mere 20 years away, the ability to cure through genetic engineering, diseases of the existing population, cancer being one, heart disease possibly being another. I'm basically an optimist, and since I happen to be in my early forties, in 20 years I'll be in my early sixties, and so I certainly am hopeful!

MR. FRANK V. BROLL, JR.: My question relates to Mr. Collett's comment about a tissue sample being used to determine a person's life expectancy. Does that not invalidate the pooling of risk concept we have in insurance today? Would insurance still exist if that type of information were available?

MR. COLLETT: Yes, it would invalidate some pooling concepts. But it wouldn't eliminate entirely the insurance risk. I've referred several times to external unpredictable things which would still be insurable.

MR. AUTIN: Today we are able to classify individuals based on existing impairments. I really don't see very much of a change. In what I have read the Hatlic limit suggests that people will die at about 100 or 125. Knowing the specific upper limit for an individual may not invalidate the pooling of risk concept. This would probably be the case unless genetic engineering, or other advances eliminated the many causes of death which come into play now keeping us from reaching the Hatlic limit.

UNIDENTIFIED: Of all the potential cataclysmic impacts on the economy from all this, it seems to me that there are several that are particularly worth attention. One is the energy shortage. Increased population means more energy demands and more pressure to solve that problem. Another problem of more immediate noticeability is that with increased population, but an increasing number of people continuing to retire in their 50's and 60's, we will have a rapidly falling percentage of the population providing the support for the entire population. In the long run economic forces will probably attract people into the labor force, as a result of inflation, but, these may be the formerly retired who may not be the best prepared to come back. Thus it appears necessary to think about extending the retirement age rather than lowering it. How we program into that I think is an extremely interesting question, since it could involve many stresses on the economy in getting there.

MR. AUTIN: As I mentioned earlier, genetic engineering is only one of the factors with which we need to concern ourselves. Despite the fact that man may one day have the ability to prolong life and eliminate disease, we must remember that there are always alternative uses for the same resources with which he would do that. In addition, we have not addressed in this particular agenda the different perceptions or values of the population which might result from genetic engineering changes. Perhaps one small example of that is if the probability of dying gets so small that perhaps, for all practical purposes, people would die from accidents alone, then maybe people would not be interested in buying life insurance at all. My view is who would buy life insurance when they did not perceive a need for it and who would sell it when they did.

On the subject of energy and environment, I think that, long before genetic engineering has an effect, the economy will have to adjust to the increase in population, the decreased or the limited supply of food, energy and other natural resources. I'm again optimistic that society will find ways to answer those questions long before some of the additional problems resulting from genetic engineering advances are put on the table.

MR. COLLETT: One mathematician's calculation suggests that, if cancer were eliminated as a cause of death in 1980, the population in the year 2050 would be 3% larger in the United States; if cardiovascular problems were eliminated it would be 12% larger. On the subject of the effects of a larger population on consumption of energy and agricultural produce, one article I saw suggests that genetisists have an answer for that. They've shown that smaller people would consume less food and could live in smaller houses and use less energy. So there's something we could do there too!

MR. AUTIN: There's an answer for every problem and I think that's part of the message that we're trying to deliver this afternoon. Some of the actions being taken in society today in the name of progress have some built-in problems in them. That doesn't mean that we shouldn't proceed with those actions. It merely means we should begin to think of the consequences of these actions; are they good, are they bad; what can we do to minimize the problems that we as a society feel are problems? I'm a member on the Committee on Futurism of the Society, and one of the basic premises of futurists is that there is no single future which we can all sit down and predict. There are merely alternative futures available to us, and there is much that we can choose from.

In closing, this limerick will test to see if you were absorbing what we were trying to communicate:

There once was an actuary progressive,
whose conservative genes were excessive.
He met a virus reagent
Which had escaped from an agent;
Now his conservative genes are recessive.