# TOWARDS COMPUTERIZED UNDERWRITING - <br> A BIOLOGICAL AGE MODEL <br> Dr. K.S. Brown <br> Professor R.L. Brown <br> Department of Statistics University of Waterloo <br> Waterloo, Ontario <br> N2L 3Gl 

August 1982

ABSTRACT

This paper presents a theoretical basis for a computerized methodology for estimating biological age, a measure of time to death. The authors then discuss their attempts to reproduce the human underwriting of one life insurance company using a computerized methodology.

Competition within the life insurance industry seems to increase consistently with time. In order to achieve the lowest possible rates for their products, life insurance companies must pursue diligently all possible avenues for reducing expenses.

In the past thirty years, the advent and advancement of computer technology has assisted greatly in reducing costs in many insurance company operations.

In this paper, we will argue that it is now feasible to computerize much of the underwriting process, and with continuous monitoring of the computerized system, one should not have to sacrifice any significant level of underwriting accuracy.

This would have two immediate and obvious advantages. First, costs will be lowered significantly and, second, the time from application to issue of the policy will be shortened significantly.

In determining the premium to be paid for life insurance, or a life annuity, the actuary is concerned with time to death. This is clear from the mathematical formulation of the net single premium functions:

FOR LIFE INSURANCE: $\quad A_{x}=\int_{0}^{\infty} v^{t} t^{P} x_{x}{ }_{x+t} d t$
FOR LIFE ANMUTTIES: $\bar{a}_{x}=\int_{0}^{\infty} \bar{a} \bar{t} t^{p}{ }_{x}{ }_{x+t} d t$

It is also interesting to note that a good
approximation for these values can be determined as follows:

$$
\begin{align*}
& \bar{A}_{x} \simeq v^{e_{x}}  \tag{3}\\
& \bar{a}_{x} \simeq \bar{a}_{\dot{B}_{x}} \tag{4}
\end{align*}
$$

Hence, our concern is with time to death.

It may seem strange, therefore, that the single most important factor used to determine the size of these premiums is time since birth or chronological age. Of course, we realize that chronological age is an important
factor in predicting time to death, but it has been argued [Brown and Forbes, 1976] that a high correlation with chronological age is neither a necessary nor sufficient condition for an index which accurately measures time to death.

Instead, several biostatisticians have turned to a concept called biological age as a superior way to present, in a single parameter, the best estimate for time to death.

If a risk is assigned a biological age $x$, this means that the risk has a life expectancy, ${ }_{e}{ }_{x}$, which corresponds to the life expectancy of the average member of the defined group whose chronological age is $x$. That is, if you are assigned biological age 45 , this means you have the life expectancy of an average person whose chronological age is 45.

Therefore we can see that a biological age model is nothing more than a disguised statement of life expectancy. The reason for this method of statement will be explained later.

There have been several studies on the estimation of an index of biological age (see bibliography).

One study that has been used in the development of the biological age model is the Framingham Study of 1973 which was an epidemiological study of cardiovascular disease. The general framework of this study can be described as follows.

At the start of the study period, measurements on $k$ variables are taken on $n$ individuals deemed free from disease. After a period of $m$ years, the individuals are re-examined and the $n_{1}$ individuals who have developed the disease in the interim are noted. Based on these data, it is desired to estimate $P\left(x_{1}, x_{2}, \ldots, x_{k}\right)$, the probability that an individual with measurements $\left(x_{1}, x_{2}, \ldots x_{k}\right)$ will develop the disease in $m$ years. A model to estimate this probability, suggested by Cornfield (1962), has been used extensively in the analysis of such data:

$$
\begin{aligned}
& \qquad \text { If } P\left(x_{1}, \ldots x_{k}\right) \text { represents the probability of } \\
& \text { teveloping the disease given measurements }\left(x_{1}, \ldots, x_{k}\right) \\
& \text { then }
\end{aligned}
$$

$$
\begin{equation*}
P\left(x_{1}, \ldots, x_{k}\right)=\frac{p b_{1}\left(x_{1}, \ldots, x_{k}\right)}{f\left(x_{1}, \ldots, x_{k}\right)} \tag{5}
\end{equation*}
$$

and

$$
\begin{equation*}
1-P\left(x_{1}, \ldots, x_{k}\right)=\frac{(1-p) 6_{0}\left(x_{1}, \ldots, x_{k}\right)}{6\left(x_{1}, \ldots, x_{k}\right)} \tag{6}
\end{equation*}
$$

where $\delta_{0}\left(x_{1}, \ldots, x_{k}\right)$ and $\delta_{1}\left(x_{1}, \ldots, x_{k}\right)$ represent the distributions of $\left(x_{1}, \ldots, x_{k}\right)$ in the healthy and diseased populations respectively, $6\left(x_{1}, \ldots, x_{k}\right)$ represents the unconditional distribution, and $p$ represents the unconditional probability of developing the disease. Thus from equations (5) and (6)

$$
P\left(x_{1}, \ldots, x_{k}\right)=\left(1+\frac{(1-p) b_{0}\left(x_{1}, \ldots, x_{k}\right)}{p \sigma_{1}(x, \ldots, s)}\right)^{-1}
$$

If $f_{0}$ and $f_{1}$ are assumed to be multivariate normal with the same variance-covariance matrix $\sum$ and means $u_{0}$ and $u_{1}$ respectively, then

$$
\begin{equation*}
P\left(x_{1}, \ldots, x_{k}\right)=\left\{1+\exp \left(-\alpha-\sum_{i=1}^{k} \beta_{i}^{x} i\right)\right\}^{-1} \tag{8}
\end{equation*}
$$

where

$$
\begin{gather*}
a=-\frac{1}{2}\left(\mu_{1}-u_{0}\right) \cdot \sum^{-1}\left(\mu_{0}+\mu_{1}\right)-\log \{(1-p) / p\},  \tag{9a}\\
\left(f_{1}, B_{2}, \ldots, B_{k}\right)=\left(\mu_{1}-\mu_{0}\right)^{\prime} \Sigma^{-1} \tag{9b}
\end{gather*}
$$



Fig. 1 Graph of the bgistic function used to estuate the tisk of developing cardionascular disease. i.e. the probatiluy $P$ is given by
$P=\{1+\exp (-L R)\}^{-1}$. where $L R$ is the logit of risk.

When the unknown parameters are replaced by their estimates, the resulting estimates of $\alpha$ and $B_{i}$ are

$$
\begin{align*}
& \hat{\mathrm{c}}=-\frac{1}{2} \sum_{i=1}^{k} \sum_{j=1}^{k}\left(\bar{X}_{1 j}-\bar{X}_{0 j}\right) S_{i j}^{-1}\left(\bar{X}_{1 i}+\bar{X}_{0 i}\right)-\log \left(n_{0} / n_{1}\right)  \tag{10a}\\
& \hat{e}_{i}=\sum_{j=1}^{k}\left(x_{1 j}-x_{0 j} \mid S_{i j}^{-1}\right. \tag{10b}
\end{align*}
$$

These may be recognized as the estimated linear discriminant function coefficients with $\alpha=c-\log \left(n_{0} / n_{1}\right)$ (see, for example, Rao 1968). Thus, under the assumptions of multivariate normality with equal variance-covariance structure in both populations, the model and estimates of the parameters are well defined. Of course, the imposed structure is rarely justified since the measured variables will include binary type
data (e.g. positive family history of heart disease) and markedly non-normal data (e.g. number of cigarettes smoked per day). However, the form of the model (see Fig.l) is intuitively a reasonable one for estimating risk, since it ranges between zero and one and increases rapidly over the middle portion of the range. Hence, the model attempts to find the linear function of $\left(x_{1}, \ldots, x_{k}\right)$ which places the healthy individuals at the 'zero' end of the curve and the diseased individuals at the 'one' end.

> Table 1. Probability per 1000 of developing cardiovascular disease ${ }^{A}$ in next 8 years by specified characteristics

The characteristics are as shown together with glucose intolerance (GI) absent and left ventricular hypertrophy by ECG (LVH-ECG) negative. The probabilities are given for levels of serum cholesterol (SC) in mg/ 100 ml and systolic blood pressure (SBP) in $m \mathrm{mg}$ ( 130 Pa )
(From the Framingham Study 1973)

| SC | Probability ${ }^{B}$ per 1000 for 45 year old male -smoker |  |  |  |  |  |  |  |  |  |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | $S B P=105$ | 120 | 135 | 150 | 165 | 180 | 195 | 105 | 120 | 135 | 150 | 165 | 180 | 195 |
| 185 | 22 | 27 | 35 | 43 | 54 | 68 | 84 | 38 | 47 | 59 | 73 | 91 | 112 | 138 |
| 210 | 28 | 35 | 43 | 54 | 68 | 84 | 104 | 47 | 59 | 73 | 91 | 113 | 138 | 169 |
| 235 | 35 | 44 | 54 | 68 | 84 | 104 | 129 | 59 | 74 | 91 | 113 | 139 | 169 | 205 |
| 260 | 44 | 55 | 68 | 85 | 105 | 129 | 158 | 74 | 92 | 113 | 139 | 170 | 206 | 247 |
| 285 | 55 | 68 | 85 | 105 | 129 | 158 | 192 | 92 | 113 | 139 | 170 | 206 | 247 | 293 |
| 310 | 68 | 85 | 105 | 130 | 158 | 192 | 232 | 114 | 140 | 170 | 206 | 248 | 294 | 345 |
| 335 | 85 | 105 | 130 | 159 | 193 | 232 | 277 | 140 | 171 | 207 | 248 | 295 | 346 | 401 |

A In the Framingham definition, cardiovascular disease is considered to have developed if there is a definite manifestation of coronary heart disease, intermittent claudication, congestive heart failure or cerebrovascular accident in the absence of a previous manifestation of any of these diseases or rheumatic heart disease.
${ }^{B}$ Probabilities are estimated using the model

$$
P\left(x_{1}, \ldots, x_{8}\right)=\left\{1+\exp \left(-\hat{o}-\sum_{i=1}^{8} \hat{B}_{i} x_{i}\right)\right\}^{-1}
$$

where $\alpha=-19.7709560$ and

| $i$ | $\hat{\xi}_{i}$ | $x_{i}$ | $i$ | $\hat{\beta}_{i}$ | $x_{i}$ |  |
| ---: | :---: | :---: | :---: | :---: | :--- | :--- |
| 1 | 0.3743307 | age | 5 | 0.5583013 | cigarettes (0 nonsmoker; 1smoker) |  |
| 2 | -0.0021165 | (age) $\times($ age $)$ | 6 | 1.0529656 | LVH-ECG | (0 none; 1 present) |
| 3 | 0.0258102 | SC | 7 | 0.6020336 | GI | (0 absent; 1 present) |
| 4 | 0.0156953 | SPB | 8 | -0.0003619 | SC xage |  |

Estimates of $\alpha$ and $\left(\beta_{1}, \ldots, \beta_{k}\right)$ when assumptions of normality are not made (but the form of the model is assumed) may be obtained by maximizing the likelihood function

$$
L\left\{a, P_{1}, \ldots, B_{k} \mid=\prod_{j \in \|_{0}}^{\left\{1-P_{j}\left(x_{1}, \ldots, x_{k}\right)\right\} \prod_{j \in U_{1}} P_{j}\left(x_{1}, \ldots, x_{k}\right), ~\left(x_{j}\right)}\right.
$$

for $\alpha$ and $\left(B_{1}, \ldots, \beta_{k}\right)$, where $U_{0}$ and $U_{1}$ represent the sets of individuals in the healthy and diseased populations respectively and $P_{j}\left(x_{j}, \ldots, x_{k}\right)$ represents equation (8) evaluated at the values of $\left(x_{1}, \ldots, x_{k}\right)$ observed for the $j$ th individual. The maximum likelihood estimates often do not differ significantly from the linear discriminant coefficients and the latter are
often used because their calculation does not involve iteration.

In such problems, there are often a large number of independent variables available for entry into a model. It is a common problem to attempt to determine an "optimal" subset of these variables; that is a relatively small number of the independent variables which may predict risk nearly as well as the entire set.

The importance of any variable, say the $\ell$ th (e.g. systolic blood pressure), for predicting risk may be investigated by maximizing $L_{1}\left(\alpha, \beta_{1}, \beta_{2}, \ldots, \beta_{l}=0, \ldots \beta_{k}\right)$ The ratio $-2 \log \left\{L_{1}(\tilde{\alpha}, \tilde{B}) / L(\hat{\alpha}, \hat{B})\right\}$, where $(\tilde{\sim}, \tilde{B})$ is the vector of estimates under the second model and $(\hat{\alpha}, \hat{\beta})$ is the vector of estimates under the full model, is asymptotically $\chi_{1}^{2}$ under the hypothesis $\beta_{\ell}=0$ and large values of this quantity indicate evidence against the hypothesis that the $\ell$ th variable is unimportant in predicting the development of the disease, after adjusting for other variables.

These types of tests form the basis for stepwise procedures for model building. That is, variables can be entered into models one at a time, at each step the variable entered is the one which increases the likelihood function
the most, given the previous variables in the model. The procedure stops when no variable, not in the model, increases the likelihood appreciably. Such procedures can be combined with variable elimination procedures which remove variables one at a time until the removal of any variable in the model would decrease the likelihood appreciably. These stepwise procedures have been extensively used to try to determine "optimal" subsets of independent variables; however, they have recently come into considerable criticism (e.g. Hocking(1976), Lawless and Singhal (1978)) . With the development of high speed computers and numercial methods it is possible to very quickly screen a great many of the models and determine which models might be suitable "candidates" for consideration for an optimal model. The programe SMOD as described in Lawless and Singhal (1978) does this model screening, and was used in the analysis described later.

One advantage of developing adequate models of risk, other than the ability to test quantitatively for significant predictor variables, is that the model may be used to prepare convenient summary tables such as is presented in Table l. Such tables allow a physician to inform the patient of his personally estimated risk of developing disease and also enable him to determine which factors are elevating this risk. Further, when combined with a table such as Table 2 the physician is
able to present the individual's risk relative to individuals experiencing "average" (or perhaps, with some modification to the table, "ideal") risk.

Table 2. Appraised age of individual
with specified characteristics

The appraised or risk age is the age of the average Framingham Study (1973) male with the equivalent risk of developing cardiovascular disease in the next 8 years by the same characteristics as specified in Table 1 (GI absent, LVH-ECG negative)
[From Brown and Forbes 1976 ]

| SC | Appraised age ${ }^{\text {A }}$ of 45 year old male |  |  |  |  |  |  |  |  |  |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | Non-smoker |  |  |  |  |  |  | Smoker |  |  |  |  |  |  |
|  | SBP $=105$ | 120 | 135 | 150 | 165 | 180 | 195 | 105 | 120 | 135 | 150 | 165 | 180 | 195 |
| 185 | 36 | 37 | 39 | 40 | 42 | 44 | 46 | 39 | 41 | 43 | 45 | 47 | 50 | 53 |
| 210 | 37 | 39 | 40 | 42 | 44 | 46 | 49 | 41 | 43 | 45 | 47 | 50 | 53 | 56 |
| 235 | 39 | 40 | 42 | 44 | 46 | 49 | 52 | 43 | 45 | 47 | 50 | 53 | 56 | 63 |
| 260 | 40 | 42 | 44 | 46 | 49 | 52 | 55 | 45 | 47 | 50 | 53 | 56 | 63 | 71 |
| 285 | 42 | 44 | 46 | 49 | 52 | 55 | 60 | 47 | 50 | 53 | 56 | 63 | 71 | 74 |
| 310 | 44 | 46 | 49 | 52 | 55 | 60 | 70 | 50 | 53 | 56 | 63 | 71 | 74 | 75 |
| 335 | 46 | 49 | 52 | 55 | 60 | 70 | 73 | 53 | 57 | 63 | 71 | 74 | 75 | 77 |

A
Calculations were based on the following average risks (see Section 28, the Framingham Study)

| Age | 35 | 40 | 45 | 50 | 55 | 60 | 65 | 70 |
| :--- | ---: | ---: | ---: | ---: | ---: | ---: | ---: | ---: |
| Risk/1000 | 18 | 41 | 75 | 115 | 159 | 193 | 212 | 229 |

and risks of approximately $0 / 1000$ and $1000 / 1000$ at ages 0 and 105 respectively. The appraised age was estimated by an average quadratic logistic interpolation procedure; results $>70$ were obtained by interpolation between the arbitrary values given above, and should be treated with caution.

Table 2 is based on Robbins and Hall's (1970) approach to preventive medicine, entitled 'health hazard appraisal'. In this system, an individual's chances of dying from a number of diseases are computed, based on his physiological measurements, lifestyle etc. These are combined, and the composite risk is compared with the risk of death experienced by an"average"member of the population. The individual's appraised age, the age of the average individual with same risk of death, is obtained in this way together with recommendations aimed at reducing the appraised age. Since most persons appreciate the consequences of being, say, 5 years older than they are chronologically, the message is presented more effectively than if the same information were expressed as a probability. In this way, individuals appraised as being 'older' than their chronological age may be encouraged to reduce factors which are elevating risk, and those appraised as 'younger' may have their positive lifestyles reinforced.

A similar "health hazard appraisal" model is used by Health and Welfare Canada in their "Evalu-vie" programme. The input to the computer program are the coded answers to the following questions:


| FCT: | PACTOR | ABBREV. | CODE | DLSCRİTION |
| :---: | :---: | :---: | :---: | :---: |
| 7 | MILES <br> TLR <br> YINR | MILES |  | Enter miles driven per year an'/ or miles ars an auto passinnicr |
| 8 | SEAT BELT USE | SEAT BELT | $\begin{aligned} & 1 \\ & 2 \\ & 3 \\ & 4 \end{aligned}$ | Worn less than los of the time <br> Worn 10-248 of the time <br> Worn 25-748 of the time <br> Worn 75-100\% of the time |
| 9 | HISTORY OF BhCTERIAL PIVEUMONIA | PNEUMONIA | $\begin{aligned} & 1 \\ & 2 \end{aligned}$ | Has had <br> Has not had |
| 10 | ```BLOOD PRESSU SYSTOLIC (ir unsure cnter l20)``` | ```RE BP: SYSTOLIC``` |  | Enter systolic blood pressure in mm |
| 11 | ELOOD <br> PPESSURE <br> DIASTOLIC <br> (if unsure <br> enter 80) | BP: DIASTOLIC |  | Enter diastolic blood pressure in mm |
| 12 | ```BLOOD CHOLESTEROL (if unsure use 2)``` | CHOLESTEROL | $\begin{aligned} & 1 \\ & 2 \\ & 3 \end{aligned}$ | $\begin{array}{ccc} \text { Cholesterol Level } & 280+ \\ " & " & 220-279 \\ " & " & 219 \text { and belo.v } \end{array}$ |
| ] | DIAISETIC | HIABETES | $\begin{aligned} & 1 \\ & 2 \\ & 3 \end{aligned}$ | ```Diabetic Diabetic (Controlled) Not Diabetic``` |
| 14 | HEIGHT | HEIGHT |  | Enter Height in inches with shor. (Without shoes: <br> ADD 1 inch for Males 2 inches for Ferales) |
| 15 | WEIGHT | WEIGHT |  | Enter weight in pounds (In indoor clothing and shoes) |
| 16 | FRAME | FRAME | $\begin{aligned} & 1 \\ & 2 \\ & 3 \end{aligned}$ | Small <br> Medium <br> Large |



| $\cdots \because$ |  | ABBELV. | CODE | DNSMRIPT10.i |
| :---: | :---: | :---: | :---: | :---: |
| 21 | FAMILY HISTORY OF ISCHEMIC | FH/HEART | 1 | Both parcnts died before 60 of Ischemic Heart Disease |
|  | HEART <br> DISEASE |  | 2 | One parent died tefore 6.0 of Ischemic lleart Disease |
|  |  |  | 3 | One or both parents died before 60 of cause other than ischemic: Heart Disease OR still alive below 60 |
|  |  |  | 4 | None of the above |
| 22 | FAMILY <br> HISTORY OF DIABETES | FH/DIAB |  | FAMILY HISTORY: mother, <br> father, sister, brother, chilc |
|  |  |  | $\begin{aligned} & 1 \\ & 2 \end{aligned}$ | Yes No |
| 23 | FAMILY <br> HISTORY OF SUICIDE | FH/SUICD | $\frac{1}{2}$ | Yes <br> No |
| 24 | LMPHYSEMA <br> AND/OR <br> ERONCHITIS | EMPHYSEMA | $\begin{aligned} & 1 \\ & 2 \end{aligned}$ | Has Emphysema and/or Bronchiti: Has no signs or symptons of Emphysema and/or Bronchitis |
| 26 | RECTAL POLYP | POLYP | $\begin{aligned} & 1 \\ & 2 \end{aligned}$ | Has had Has not had |
| 27 | $\begin{aligned} & \text { PROCTO- } \\ & \text { SIGMOIDOS- } \\ & \text { COPY } \end{aligned}$ | PROCTO | $\begin{aligned} & 1 \\ & 2 \end{aligned}$ | Has annually <br> Does not have annually |
| 29 | RECTAL <br> BLEEDING | RCTBLOOD | 1 | Has had undiagnosed rectal bleeding in the past year Has not had undiagnosed rectal bleeding in the past year |
| 30 | CHRONIC PHEUMATIC HEART | RH: FEVER | 1. | Rheumatic Heart Murmur, no Chemoprobhylaxis Rneumatic Heart Murmur, on Chemoprophylaxis |
|  | DISEASE |  | 3 4 | History of Rheumatic Fever but no Murmur, no Chemodrophylaxis History of Rheumatic Fever but no Heart Murmur, on Chemoprophy Iaxis |
|  |  |  | 5 | No history of Rheumatic Fever dru no Fireumatic Heart Mumur |
|  |  |  |  |  |


| $\because$ \% | HACTOK | ABBRE:V. | CODE | טESCRTMFION |
| :---: | :---: | :---: | :---: | :---: |
| 31 | SIGNS OR SYMP'ROMS OF CHRONIC RHLUMATIC LiEART DISEASE | RH: S/O/S | $\begin{aligned} & 1 \\ & 2 \end{aligned}$ | No <br> Yes |
| 32 | ULCERATIVE COLITIS | ULCERCOL | 1 <br> 2 <br> 3 | Has had Ulcerative Colitis 10 years or more <br> Has had Ulcerative Colitis <br> less than 10 years <br> Has no symptoms of Ulcerative: Colitis |

* ${ }^{T H E}$ FOLLOWING FACTORS ARE FOR FEMALES ONLY:

| に「』 | FACTOK | ABBREV． | CODE | DFSGRIPTION |
| :---: | :---: | :---: | :---: | :---: |
| 33 | FACTORE 33－38 ARE TO＿BE CODED BY＿FEMMLES＿ONLY： |  |  |  |
|  | VAGINAL <br> BLEEDING | VAGBLOOD | 1 | Has had undiagnosed vaginal bleeding in past year |
|  |  |  | 2 | Has not had undiagnosed vaginal bleeding in past yea： |
| 34 | AGE AT <br> MARRIAGE <br> OR ONSE＇T <br> OF <br> JNTERCOURSE | AGE／MAR | $\begin{aligned} & 1 \\ & 2 \\ & 3 \end{aligned}$ | Teenage 20-25 <br> Over 25 or never |
| 35 | IAPSMEAR | PAPSMEAR | $\begin{aligned} & 1 \\ & 2 \\ & 3 \\ & 4 \end{aligned}$ | Has not had <br> Negative within 5 years <br> Negative within 1 year 3 Negative within 5 years |
| 36 | $\begin{aligned} & \text { ECONOMIC } \\ & \text { AI:D } \\ & \text { SCCIAL } \\ & \text { STATUS } \end{aligned}$ | SOCIO／EC | $\begin{aligned} & 1 \\ & 2 \\ & 3 \end{aligned}$ | Low <br> Average High |
| 37 | JEWISH | JEWISH | $\begin{aligned} & 1 \\ & 2 \end{aligned}$ | No <br> Yes |
| 38 | EREMILY <br> HISTORY <br> 1）$\Gamma$ BREAST <br> CANCER | EH／BREAST | 1 2 | Mother or sister had Breast Cancer <br> Mother or sister had Brcasit Cancer but patient examines breasts regularly and has periodic examination by phys！ |
|  |  |  | 3 | Neither mother nor sister had Breast Cancer |
|  |  |  | 4 | Neither had Breast Cancer but patient examines breasts regularly and has periodic examination by physician |

Thus, a biological age index may be seen as a function of a set of observable quantities, each of which makes an important contribution to the prediction of age at death, years until death or some other measure stratifying the population by risk subgroups.

$$
\text { A biological age index, } y \text {, may be written then }
$$

in the form

$$
y=f(x, V, I, \Delta)
$$

where $y$ represents, for example, age at death, years until death, reduction in optimal lifespan, an individual's appraised or risk age etc; $x$ is the chronological age; $V$ represents the additional contribution (i.e. adjusted for chronological age and other variables in the function) of the absolute level of a set of variables, such as blood pressure, cigarette smoking habits, family history, etc.; I represents the additional contribution of the interaction of variables in $V$ with other variables in $V$ and with chronological age; and $\Delta$ represents the additional contribution of past changes in any of the variables in $V$, and also of changes in variables that are not in $V$ but are important because a change in them implies an elevation or reduction of $y$. The variables in the sets $V$ and $\Delta$ and all constants entering into this function have to be determined from a study of a reasonably large number of individuals followed longitudinally.

Late in 1981, the authors approached the Mutual Life of Canada, a large mutual insurance company located in Waterloo, to see if we might test the biological age model by attempting to computerize their underwriting process.

Mutual Life presently issues about 80,000 individual life insurance policies a year. Approximately 75\% of these policies are issued non-medical. Even for these policies, the cost of underwriting and issue averages close to $\$ 100$ an application and the time needed for underwriting averages around five days.

The Mutual Life allowed us access to their adult non-medical cases. We were sent coded data which included the following information:

- beneficiary relationship
- insurance amount
- the underwriters decision (issue standard, issue rated, or decline)
- and if the decision was other than standard what other information was requested by the underwriter.

We were also provided with the answers given to the non-medical application form as shown on the next page.


DECLARATION: I declare the above answers and staiements are full. comptete and true and shalf form part of the evidence of insurabitly in respect of my applicalion for insurance fot for reinslatement of or change in my present insurance) in The Mutual Lite Assurance Company of Canada
AUTHORIZATION: I authorize any physician or practinoner who has observed me for diagnosis or treatment, and any hospital, clinic or other medical or medically elated tacility where I have been a patien! and any insurance compary. The Medica! iniormation Buteau or other organization, institution of person, Ihat has any records or knowiedge ot me or my health. Io give full particulars thereot incuding any phor medical history to the Mutual Lite Assuramce Company ot Canada, or its reinsurers. A photosiat of this authorization shailibe as valid as the

| Shoned at |  |
| :--- | :--- |
| Whtness |  |
| $3-2-80$ |  |
| Stgnature of |  |
| Lifa insured |  |

COMPLETE THIS SECTION FOR PARAMEDICAL AND MEDICAL EXAMINATIONS


## ADDITIONAL REPORT BY MEDICAL EXAMINER





## ADDITIONAL REMARKS

| Soned at | Dato | , 0 | ${ }_{\text {A M }}^{\text {M }}$ |
| :---: | :---: | :---: | :---: |

## Ekgnature el Examiner

Our first and most serious problem was that there existed little or no matching between the non-medical questions asked by Mutual Life and the questions used in the biological age models now in existence. Hence, it was impossible for us to feed the Mutual Life data into any of the pre-packaged biological age models.

Instead, it was decided that we would access two sets of underwriting data. Each set would consist of approximately eight hundred applications. Using the first set of applications (which, in fact, totalled 824) we would build a model that could discriminate between those applicants that were rated or rejected from those applicants who were issued standard without further information. Using the model developed on this first set of applications, we would then run it on the second set of applications (which, in fact, totalled 829) to see if our model could correctly separate out those applicants that could safely be issued insurance at standard rates with no further underwriting, from those who could not.

Using the first 824 apps, then, we built a model of the type described earlier (e.g. see equation 8) where, in this instance, $P\left(x_{1}, \ldots, x_{k}\right)$ is the probability that an individual with variables $x_{1}, \ldots, x_{k}$ corresponding to information from the applicatịon, will be judged non-standard or rejected.

Then

$$
P\left(x_{1}, \ldots, x_{k}\right)=\frac{1}{1+e^{-\alpha-L E_{i} x_{i}}}
$$

where $\left\{x_{1}, \ldots, x_{k} \mid\right.$ represent responses to a set of $K$ items chosen from the questionnaire, and the c's represent the weights attached to the items.

The data on which the model was built consisted of information, of the type described earlier, on 824 applications representing approximately one month's adult non-medical applications. Of these 824 applications, 10 were issued nonstandard, and 17 were rejected. For the purposes of these analyses, these latter 27 policies were grouped into one category, called the non standard group hereafter. Further, the analysis emphasized only the health related information from the questionnaire.

Initially, simple summaries of the data
comparing the responses of the standard group and the nonstandard group on each of the medical based questions from the application were conducted. These analyses indicated certain questions for which the response pattern was different in the two groups. Secondly, as an overall measure of the status of the applicant, a single variable, the number of
"no" answers to certain questions* was created. Finally, guided by the results of these preliminary analyses, the model screening programme (SMOD; see earlier description) was uscd to identify subsets of the questions, the responses to which would separate the two groups almost as well as the responses to the large number of original questions. This latter procedure had to be carried out in steps. At each stage, different combinations of items from the questionnaire were entered into a full model and those items which did not appear to make any significant contribution to the prediction of the non-standard cases were deleted.

Finally, two models emerged as models which seemed to be able to discriminate between standards and nonstandards nearly as well as the full set of independent variables.

The variables in these models were:

[^0]MODEL 1

Response to Question 8A Response to question 9B The number of "no" answers

Age

MODEL 2

Alcohol use
Cigarette smoking
Response to question 8A
The number of "no" answers Age

Both models seemed to predict equally well, however there were some anomalies in the models in that responding "yes" to the questions on alcohol use, cigarette smoking and question 8 A increased the estimated probability of being judged standard. (Those patterns were also apparent in the original data, i.e. a higher proportion of standard cases drank, and a higher proportion smoked.)

With the maximum likelihood estimates of the coefficients in the model, it was possible to estimate, for each subject, his or her chance of being judged standard. By choosing a cut-off level and declaring standard anyone with an estimated probability of being standard which exceeded that value, it was possible to assign each individual to one of the four cells in the summary table below.

Table 3. Results of classification based on the model

MODEL
CLASSIFICATION


Thus, in the table, $n_{00}+n_{l l}$ cases are correctly classified, while $n_{10}+n_{01}$ are incorrectly classified. By varying the cut-off level it is possible to increase or decrease the number predicted in the standard class. For the purposes of this illustration, the cut-off level $\frac{795}{824}=.9672$ was chosen as this produced small values of $n_{01}$ with reasonably small values of $n_{10}$. In practice, one could choose a cut-off level which reduced the size of $n_{01}$ at the expense of increasing $n_{10}$ by assessing the costs of misclassifying a standard as ₹ non-standard against the costs of the other misclassification (i.e. the extra cost of underwriting 'standard' cases vs. missing a substandard case). Alternatively, one could choose to underwrite, by hand, the $x \%$ of cases with the lowest estimated probabilities of being standard.

When applied to the original data set, using the cut-off level .9672, the two models were able to discriminate as shown below.

Table 4. Results of classification for Models 1 and 2

MODEL 1

ACTUAL RATING

|  | Standard | Non-standard |  |
| :--- | :---: | ---: | ---: |
| Standard | 668 | 2 | 670 |
| Non-standard | 129 | 25 | 154 |
| 797 | 27 | 824 |  |

Thus, using model $1,15.9 \%\left(\frac{131}{824}\right)$ of the cases were misclassified but most importantly, only two non-standard cases were misclassified as standard. Increasing the cut-off to . 975 resulted in only one non-standard case being misclassified as standard, however 151 standard cases were classfied as nonstandard. This one non-standard case could not be correctly classified with a cut-off level as high as .99*. Model 2 did not fare quite so well, misclassifying fewer cases overall, but

[^1]missing 4 out of 27 non-standard cases (3 out of 27 using a level of .975).

It is well known that regression models often predict considerably better for the data set on which they were built than on other similar data sets; but in testing the models developed earlier on the next 829 applications coded, (819 standard, 10 non-standard or rejected) as indicated in Table 5 , both models were able to correctly identify all 10 non-standard cases!! Model l, using a cut-off point of .9672, misclassified 139 standard cases as non-standard, while model 2 , with a cut-off level of .9672 did marginally better, misclassifying lī5 standard cases.

## Table 5. Results of Classifications <br> based on models 1 and 2 <br> for the second data set

MODEL 1

ACTLAL RATING
Standard Non-standard

| Standard | 680 | 0 | 680 |
| :--- | :---: | :---: | :---: |
| Non-standard | 139 | 10 | 149 |
|  | 819 | 10 | 829 |

MODEL 2

ACTUAL RATING
Standard Non-standard

| Standard | 684 | 0 | 684 |
| :--- | :---: | :---: | :---: |
| Non-standard | 135 | 10 | 145 |
|  | 819 | 10 | 829 |

Thus, model 1 would have declared 680 applications as standard, leaving only 149 (18\%) to be underwritten in the usual way. This could have resulted in a potential saving of about $80 \%$ of the cost of underwriting, balanced against the additional expense of approximately $\$ .62$, the cost of computer time to use the model to classify the 829 applications.

## CONCLUSION

As pointed out earlier, the Mutual life issues close to 80,000 individual life insurance policies a year. $75 \%$ of these are issued non-medical with an underwritina and issue cost of around $\$ 100$ each and delay time of close to five days.

Only $3.5 \%$ of the non-medical applications are rate or declined, on average.

We have shown, through the use of a fairly crude model, that a computer can be programmed to produce results very close to those determined by human underwriters. This was done using input data that was not designed for computerization nor was it based on any preconceived model such as the biological age model.

We feel that a great deal of the underwriting that is being done in an expensive and relatively slow manner today could be computerized. This would require using a machine-readable application form and we might wish to use input more suited to the existing biological age models, although we have shown that this may not be essential.

One of the exciting frospects of a computerized underwriting model is that it can continuously reassess itself! For example, every time a policyholder dies, the computer can retrieve the application form for the policyholder and determine what information was provided that might have predicted that early (normal, or late) death. In this manner, the computer can statistically reanalyze the weights that have been given to each input parameter and continuously improve its own programme!

Once one has faith in this computerized model, one can have a much more refined pricing stratification than exists today. No longer will $75-85 \%$ of all cases be priced based on sex and biological age only. Rather, the computer will determine the "time to death" in its program and set the proper premium level accordingly.

This same methodology, applied to life annuities, would go far to answering the feminists clamouring for unisex annuity mortality tables since one would no longer be pricing based only on sex and chronological age. Rather, one would be pricing based on an objective statistical prediction as to "time to death".

One can visualize a day, in the not-too-distant future, 'vhere an agent will enter the client's home with his portable computer terminal. After using the terminal as a sales aid (e.g. to show cost comparisons and investment attributes) the agent will then ask the client the usual application questions. The answers to these questions will be entered into the computer through the portable terminal and, in a matter of seconds, the computer will tell the agent whether his client has been accepted, rejected, or whether further information is required. (Note -- the term rated will no longer be used).

If the client is accepted, the computer will
produce the price level for the policy required based on the statistical analysis of time-to-death. Then, if the client is in agreement, the portable terminal will print out a policy and the process will be complete!

The authors would like to thank the Mutual Life of Canada and, in particular, Barry J. Triller (New Business Executive) and Wilhemina Gould (New Business Analyst) for the assistance with this project. Without their help, this research could not have been completed. We would also like to acknowledge the assistance of Evan Kelly who was responsible for the computer analysis underlying this project.

## BIBLIOGRAPHY

Borkan, G. A. (1980). Assessment of Biological Age Using a Profile of Physical Parameters, Journal of Gerontology Vol. 35, No. 2, pp. 177-184.

Brown, K. S. (1977). Mathematical Contributions to the Study of Risk Factors in Cardiovascular Disease. II., Math Scientist, 2, lll-125.

Brown, K. S., Forbes, W. F. (1976) Concerning the Estimation of Biological Age, Journal of Gerontology, Vol. 22, pp. 428-437.

Brown, K. S., Nabert, W. (1977). Evaluation of the Existing Method for Calculating Health Hazard Appraisal Age. Drug Directorate, Health Protection Branch, Health and Welfare Canada.

Comfort, Alex. (1956). The Biology of Senescence. Churchill Livingstone.

Comfort, Alex. (1969). Test-battery to Measure Aging - Rate in Man, The Lancet, $\mathrm{O}=\mathrm{l} . \mathrm{6}, \mathrm{pp}$. 1411-1414.

Hershey, Daniel. (1980). A New Age-Scale for Humans. Lexington Books.

Hocking, R.R. (1976). The Analysis and Selection of Variables in Linear Regression. Biometrics, 32, pp. 1-50.

Lawless, J.F., Singhal, K. (1978). Efficient Screening of Nonnormal Regression Models. Biometrics, 39, pp. 318-327.

Palmore, E., Jeffers, F. C. (1971). Prediction of Life Span. Heath Lexington Books.

Rao, C. R. (1968). Linear Statistical Inference and Its Applications. Wiley, New York.

Robins, L, Hall, J.R. (1970). How to Practice Prospective Medicine. Public Health Service.

Rockstein, M. (1974). Theoretical Aspects of Aging. Academic Press.
Ross, C. L. (1971). Predicting Longevity. Heath Lexington Books.
Shurtleff, Dewey. (1974). An Epidemiological Investigation of Cardiovascular Disease. U.S. Government.


[^0]:    * 

    Questions: $3,4 \mathrm{~A}, 4 \mathrm{~B}, 4 \mathrm{D}, 4 \mathrm{E}, 4 \mathrm{~F}, 4 \mathrm{G}, 4 \mathrm{I}, 4 \mathrm{~J}, 5(\mathrm{all})$, 6 (all), 7, 8A, 8B, 9A, 9B, 9C, 11, were considered in arriving at this index.

[^1]:    * $₹$ urther investigation revealed that this case was rated nonstandard on the basis of special information on the applicant's arthritis, gout and rheumatism obtained from X-ray examination.

