A GENETIC APPROACH TO THE MODELLING OF SICKNESS RATES, WITH APPLICATION TO LIFE INSURANCE RISK CLASSIFICATION

by

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A genetic approach to the modelling of sickness rates, with application to life insurance risk classification.

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Abstract

A model is developed from genetic principles which enables sickness rates to be constructed which are dependent upon genotype, environment and age. It is shown how these rates can be easily incorporated into a simple 3-state Markov model of policyholder progression, from which life insurance costs can be estimated. Hypothetical examples are shown which demonstrate the use of the approach, and illustrate the potential for anti-selection from multifactorial genetic testing for late-onset disease. The inclusion of the specific disease link between the genetic cause and mortality is considered to be important when assessing the potential of genetic testing in risk classification. The examples show that genetic factors are of little importance in risk classification if there are more significant environmental determinants of risk. However, where genetic effects are dominant, and the genetic-environment interaction in the determination of risk are insignificant or unambiguous, then risk classification by genetic factors for such diseases may become important as genetic testing becomes more widespread and cheaply available in the future.

1. *Introduction*

1.1 *Background and overview*

The implications of genetic testing for life and health insurance is a subject of considerable current controversy and debate. This paper makes no attempt to take positions in this debate, but attempts to inform by showing how both genetic and actuarial modelling can be usefully combined in the assessment of the possible future need for risk classification by genetic factors.

Many of the current issues were discussed at a joint meeting of the Royal Society of London with the Institute and Faculty of Actuaries in the UK, held in 1996 (see The Royal Society, 1997). A particular paper by MacDonald (1997) uses a Markov model for the combined processes of insurance buying, underwriting and policyholder progression, in order to assess potential anti-selection from genetic testing in life insurance. The results suggest that the potential cost of anti-selection is likely to be low except in cases where such selection is associated with atypically large sums assured.

MacDonald's model assumed a direct link between the genetic determinants of risk and mortality. In the present paper it is argued that the special nature of genetic risk determination, which is through its effect on the propensity to develop disease, should be incorporated explicitly in the modelling process. This, it is proposed, should be done through the specific inclusion of a diseased (or 'sick') state in the model, relating explicitly to the particular disease from which lives of the genetic types under consideration are considered to be at risk. This allows mortality from other causes to be independent of the

genetic type, which is a more realistic assumption. The model is described in section 2 of the paper.

An attempt is then made in section 3 to show how genetic theory can be used to develop a model for sickness rates. These rates can then be used in the assumed model of policyholder progression, incorporating the specific sick state. In sections 4 and 5 of the paper the model is applied on the basis of some illustrative assumptions and the conclusions are discussed in section 6.

1.2 *Risk classification in life insurance*

There are essentially two extreme views on the approach which insurers should adopt for risk classification. At one extreme, proponents of 'risk pooling' would advocate the classification of risk only where the risk (and costs) of anti-selection would otherwise be too great. Proponents of 'actuarial equity' would propose risk classification by all factors which could reliably be established as producing a risk differential between individuals, thereby charging cheaper premiums to low risk lives but higher premiums to the higher risks (the basis of so-called 'preferred lives' pricing: see Werth, 1996.) These views reflect the issues of 'moral' versus 'actuarial' fairness (Pokorski, 1997).

Whichever approach may be considered the more justifiable, there has to be a minimal degree of risk classification which must be performed in order to avoid extreme costs of anti-selection, and this is essentially the risk-poolers' philosophy. I will adopt this philosophy as the basis for the development of new risk classifications by genetic factors, not because it is an approach that I necessarily support, but because it has appeal as the natural minimalist assumption.

Risk classification in life insurance is concerned with identifying factors which have major influence on claim frequencies, through the underwriting process. Hence with most life insurance the risk is one of early claim, caused by early death. In this context any death before the age of 60 or even 70 can be considered as 'early' for insurance purposes, and it is therefore the common causes of death at these ages which are of interest to the life insurance underwriter. While there are a few monogenic late onset killer diseases, such as Huntington's disease, the majority of diseases causing death at these ages (such as heart disease and cancer) have a multifactorial genetic basis. I would suggest that the rarity of the monogenic lethal disorders means that the financial effect of any anti-selection, while potentially severe in an occasional individual case, will have little impact on insurer solvency. Should adverse selection occur with respect to the common multifactorial causes of early death, however, then there could be a more significant risk to the company's financial stability. It is therefore these causes of death, and their multifactorial determination, which will be the focus of our attention in this paper.

1.3 *Multifactorial genetic determination*

The premise for the experiments in this paper is that it will become possible to identify an individual's genetic predisposition to major late onset killer diseases, particularly heart disease and cancer, through genetic testing of the genes at various *loci* known to affect the probability of developing these diseases. (A locus is the location or position of a particular gene in the genetic material of an individual.) Such diseases are

examples of multifactorial genetic traits, which generally have the following characteristics:

(i) *Alleles* of genes at more than one locus, and often many loci, may be involved. (An allele is a variation of the gene at any given locus).

(ii) The different genes affecting the trait often do so to differing degrees. Hence there would often be a relatively small number of genes having a relatively large effect on the trait, while there may be many genes with individually minor effects. For the purpose of estimating the risk of disease, for example, the minor (or modifying) genes can probably be ignored.

(iii) Most *genotypes* for a multifactorial trait will lead to an increased or decreased probability of showing the particular trait; only rarely would the probability be made certain. (The genotype of an individual is the set of alleles at the loci of interest in respect of that individual.) The presence of a particular allele at a particular major locus can lead to the latter effect. In this case the situation essentially becomes monogenic in nature, as the expression of the genes at the remaining loci becomes irrelevant in the manifestation of the trait.

Hence a multifactorial trait is affected by alleles usually at many loci, whoses individual influence can vary from total (in which case the trait is behaving as if monogenically determined), to the very minor (modifying in a small way the effects of the major genes involved in the trait).

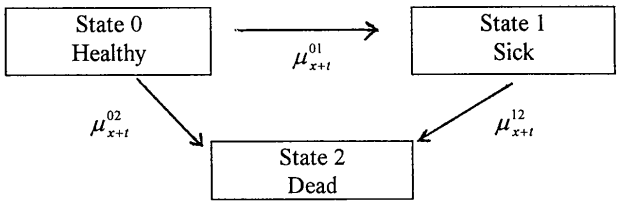
The different alleles which may be present at the same locus in different individuals are the results of mutations (ie alterations) of the DNA code at this position which have occurred in earlier generations. Only non-lethal alleles can persist in the population, as a mutation which causes death before the individual has had time to reproduce will not be passed on to the next generation. Hence the alleles which are found persisting in the population can, by definition, only affect mortality (a) not at all, (b) to a degree or (c) after the age of reproduction.

It is this last category of mutation which is largely important for insurance purposes, as by definition these mutations will lead individuals to have different genetic predispositions to late onset diseases. It would also be expected that there could be considerable variety of alleles at individual loci responsible for these effects, as they would not be subject to removal by natural selection. The likelihood of finding any effective risk classifications through genetic testing is therefore already reduced by this observation: the greater the variety of genes at individual loci which affect any predisposition to disease, the harder it will be to assess the risk accurately. The difficulty is further exacerbated by the interaction between genes that can occur when they are expressed in the same individual (this is known as *epistasis*: see Hedrick, 1985). Hence for example the presence of two different alleles each known to affect the trait in isolation could possibly produce double or half (or any other other multiple) of their combined individual effect when present together in a single individual.

2. *The model of policyholder progression*

It is clear from the foregoing that any genotype which may affect mortality will do so through the manfestation of disease. It would therefore seem logical to adopt the 3-state Markov (multiple-state) model shown in Figure 1 for the purpose of modelling the evolution of a population of insured lives.

Figure 1. The 3-state Markov model of policyholder progression



This models the history of a life assumed to start as a healthy policyholder at age x, with transitions between the three states as shown by the arrows. The annual forces of transition between states i and j at an exact moment t are given by μ^{ij}_{x+t}, and these govern the rates of continuous transfer between states. The main features of the model are as follows.

(i) No recovery is allowed from the sick state. Hence only those diseases which are terminal are included in the model. This nevertheless deals adequately with the types of disease of interest in this investigation, and the inclusion of transitions from sick to healthy would be an unnecessary complication of the model.

(ii) Lives which are sick from any cause other than that (or those) included in the explicit sick state of the model are included among the 'healthy' lives. The mortality from healthy to dead therefore includes mortality from all causes (including illnesses) which are not included in the mortality of lives from the sick state of the model.

The model should be useful in calculating the expected costs arising from death claims arising from a specific, non-reversible, disease or diseases. These costs can be put in the context of the total costs of claims arising from all causes, and different assumptions can be made in respect of each type of claim, which would seem appropriate to reflect the specific relationship required to exist between the genetic risk and mortality via the particular disease which the genetic risk affects.

However the model cannot be used for the measurement of health insurance risks without further modification. This is because, while it could be used to estimate the cost of claims arising while sick from the specific disease or diseases, costs of claims while sick from other causes are not allowed for. This would overstate the importance of the risk

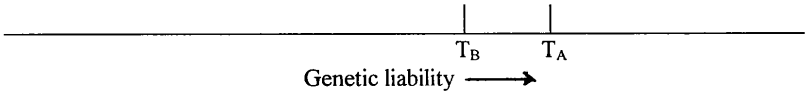
from the genetically determined cause, and could significantly distort the modelled effect of any anti-selection.

3. *A genetic model of sickness rates*

It is well known in biology that observed characteristics are affected both by inherent (genetic) factors and by the environment. In the present study the observed characteristic (which can be referred to as the *phenotype* of the individual) is the manifestation of the specified disease at a particular point in time (that is, either the life does or does not have the disease at that point).

Hedrick (1985) describes how, for a given environment, an individual may be expected to develop a particular disease at some future stage if his or her 'genetic liability' exceeds some threshold level specific for that environment. The genetic liability is a quantification of the aggregate risk (the propensity to develop the disease) resulting from the combined effects of the specific alleles present at all the relevant loci in that individual. Figure 2 shows the idea for two different environments, A and B.

Figure 2. Disease thresholds for different environments



Hence in Figure 1 no individual will develop the disease if their genetic liability is less than T_B; individuals in environment B will develop the disease if their genetic liability exceeds T_B; and so on.

The problem with this approach is that (a) it does not identify *when* the disease will occur, only whether it will occur at all, and (b) it is a deterministic model. To deal with these two problems the following modification is proposed.

First we refine the definition of the phenotype to be:

$P_{x+t,j} = 1$; if a healthy life j at age $x+t$ becomes sick during the year of age $[x+t, x+t+1]$

$P_{x+t,j} = 0$; otherwise.

We now introduce the concept of *phenotypic liability*. This is the actual propensity for a healthy life to develop the disease during one year (say), which will be defined by the following model:

$$L_{x+t,j} = G_{x+t,j} + E_{x+t,j} + I_{x+t,j} + R_{x+t,j} \qquad (1)$$

where $G_{x+t,j}$ = the genetic liability

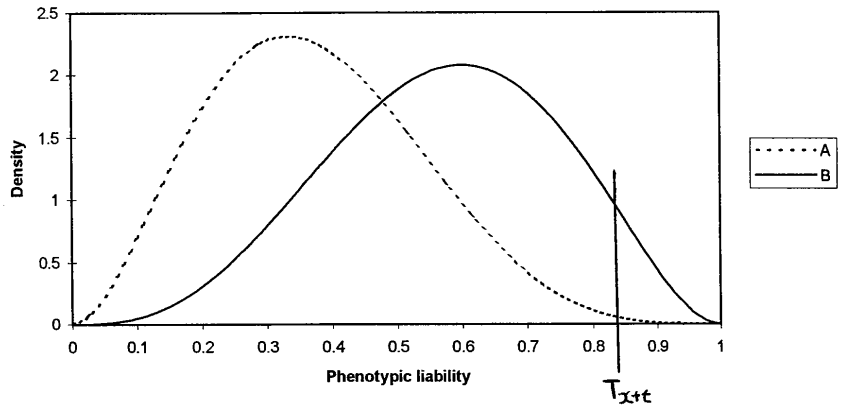
$E_{x+t,j}$ = an adjustment due to the environment

$I_{x+t,j}$ = an adjustment due to the interaction between the environment and the genotype

$R_{x+t,j}$ = a random quantity

for a life j aged $x+t$ at the start of the year. Hence for a given environment and for a given genetic liability, $L_{x+t,j}$ will take the form of a probability distribution. Two examples are shown in Figure 3.

Figure 3. Example distributions of the phenotypic liability, L



In this formulation, the threshold T_{x+t} for the disease manifestation at age $x+t$ is fixed in relation to the liability, while the location and shape of the distribution are assumed to vary between individuals due to their genetic and environmental attributes. For those whose phenotypic liability L exceeds T, then the disease will develop during the year, but will not develop otherwise. ie:

$P_{x+t,j} = 1$ if $L_{x+t,j} \geq T_{x+t}$;

and $P_{x+t,j} = 0$ if $L_{x+t,j} < T_{x+t}$.

The probability of becoming sick during the year is therefore given by the area under the density curve to the right of T. ie:

$$\Pr(P_{x+t,j} = 1) = 1 - F(T_{x+t}) \qquad (2)$$

where $F(L_{x+t,j})$ is the distribution function of $L_{x+t,j}$.

Hence in the example in Figure 3, life B has a higher probability of becoming sick than life A (because they have different genetic liabilities and/or they are subject to different environments).

In terms of the 3-state model defined in section 2, the rate of sickness obtained from the above genetic model is equivalent to:

$$\Pr(P_{x+t,j} = 1) = \int_0^1 {}_r p^{00}_{x+t} \cdot \mu^{01}_{x+t+r} \, dr$$

where $${}_r p^{00}_y = \exp[-\int_0^r (\mu^{01}_{y+s} + \mu^{02}_{y+s}) ds]$$

which is the dependent rate of sickness among the healthy lives over the year of age $[x+t, x+t+1]$. We will denote this rate by s_{x+t} for convenience.

4. *Illustration of the use of the model: methodology*

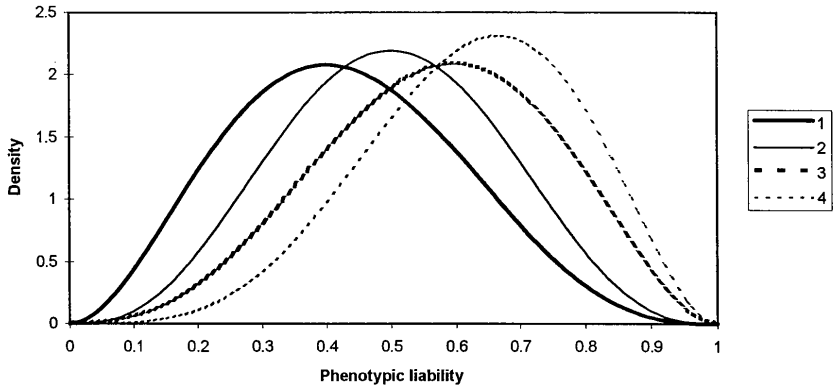
4.1 *Experience assumptions*

It is assumed that all contracts are issued at exact age $x = 40$ and terminate at exact age 75.

For simplicity it will be assumed that individuals can have just two different genetic liabilities with respect to a particular disease, and that there are two different environments. It should be recognised that this is not the same as assuming that there are just two different *genes* affecting the liability: it means that the combined effects of *all* the various genes affecting the trait always leads to one of just two values for the liability. This is not quite as heroic an assumption as it first sounds: in practice a bimodal continuous distribution of genetic liabilities would probably approximate quite well to the assumption made here. The assumption of a bimodal environmental factor is also reasonable: the case of smoker versus non-smoker comes readily to mind.

In any given experiment there will therefore be four different genotype-environment combinations, referred to as *risk types* and denoted by E*i*G*j* ($i,j = 1,2$). Each of these four risk types is assumed to have a different phenotypic liability distribution, such as shown, for example, in Figure 4.

Figure 4. Example phenotypic liability distributions



The sickness rate for each risk type is obtained using expression (2) by assuming a particular threshold T_{40+t} for each particular age $40+t$. Assuming a scale for L_{x+t} of ($0 < L_{x+t} < 1$), we chose:

$$T_{40+t} = 1 - .004t; \qquad 0 \le t \le 35 \tag{3}$$

Hence T_{40+t} reduces by equal steps from 1.0 at age 40 to 0.86 by age 75. This scale was chosen so that, in conjunction with the assumed density functions (described below), the resulting sickness rates seemed credible in relation to the assumed mortality basis.

The form of distributiion for the phenotypic liability seemed to be most suitably defined by the Beta distribution, having density:

$$f(L) = \frac{\Gamma(\alpha+\beta)}{\Gamma(\alpha)\Gamma(\beta)} L^{\alpha-1}(1-L)^{\beta-1} \qquad 0 < L < 1$$

The standard Beta distributions assumed for the four phenotypic types were as shown in Table 1.

Table 1. The assumed phenotypic distribution

Distribution	α	β
1	3	4
2	4	4
3	4	3
4	5	3

These distributions are shown in ascending order of the size of the sickness rate obtained at any given age, with (1) producing the lowest rates. The resulting distributions are the ones actually shown in Figure 4, from which the relative differences between them can be appreciated.

The effect of these four different distributions, combined with the assumption for T_{40+t} in expression (3), give rise to the sickness rates s_{x+t} shown in Table 2.

Table 2. Assumed sickness rates (specimen ages)

	Distribution (see Table 1)			
Age	(1)	(2)	(3)	(4)
40	0	0	0	0
45	0	0	.00015	.00026
50	.00004	.00008	.00117	.00198
55	.00018	.00039	.00376	.00629
60	.00054	.00118	.00851	.01401
65	.00127	.00273	.01585	.02569
70	.00254	.00537	.02610	.04164
75	.00455	.00943	.03945	.06197

The mortality of healthy lives was assumed to be equal to approximately 70% of the A1967/70 select mortality table, a standard table based upon the UK life offices' assured lives experience over the period 1967-1970. Note that the choice of table is immaterial for the validity of the study, as it is assumed to be the same in all experiments and for all risk types. These rates are shown in Table 3 and the ratio of sickness rate to mortality rate are are also shown for comparison.

Table 3. Mortality rates of healthy lives
and sickness rate as a percentage of the mortality rate at each age

		Distribution (see Table 1)			
Age	Mortality	(1)	(2)	(3)	(4)
40	.00071	0	0	0	0
45	.00185	0.13%	0.28%	8.25%	14.22%
50	.00337	1.07%	2.41%	34.70%	58.92%
55	.00594	2.97%	6.60%	63.43%	106.05%
60	.01015	5.31%	11.59%	83.88%	138.10%
65	.01690	7.52%	16.15%	93.81%	152.05%
70	.02750	9.25%	19.53%	94.91%	151.44%
75	.04380	10.38%	21.54%	90.08%	141.49%

The progression of sickness rates with age from this model begins with very low rates of sickness at young ages followed by a rapid increase with increasing age. For the

two highest bases (distributions (3) and (4)) sickness rates become approximately stable as a proportion of the assumed mortality, while the ratios continue to increase with age for distributions (1) and (2).

This pattern might be reasonable for diseases whose presence would be screened for by the underwriting process at the issue of the contract, which would lead to very low rates of sickness immediately following entry. The increase in the incidence rate of new cases of the disease rises with age, typical of late onset disorders whose incidence will also be affected by the cumulative effect of environment over the lifetime (such as from smoking).

The use of this genetic model gives an interesting insight into the reason why sickness rates might progress differently with age than do mortality rates. However, while the form of the model might be appropriate, the parameters (defined here by expression (3) and in Table 1) are arbitrary. The form of the model is, however, completely flexible, because any particular sickness rate can be obtained from any assumed liability curve by choosing the appropriate vaue for T_{x+t}. The model therefore adds nothing where only one risk type is involved: indeed it could not be parameterised as there would be an infinite number of models which could generate the same sickness rates. The key to the practical usefulness of the model depends upon whether the *same* function for T_{x+t} can model effectively the rates of sickness of *different* risk types by assuming different phenotypic liability distributions, ie as done in this paper. This is clearly an area for further investigation.

4.2 *Miscellaneous assumptions*

The values of μ^{12}_{x+t} (the mortality of lives in the sick state) are assumed to be constant at all ages. For the standard basis a value of $\mu^{12}_{x+t} = 0.5$ was assumed (which implies a life expectation of sick lives of 2 years), which seems reasonably appropriate for at least some of the types of disease being considered.

The type of contract assumed is a 35 year term assurance for a level sum assured of £1, issued to the life aged 40.

In calculating expected present values of the benefits an effective rate of interest of 5% per annum is assumed. The expected present value of the benefits is equivalently the single premium (or the expected present value of the annual premiums, as appropriate) that would be payable in order to meet the policy claim costs.

Expenses and all other outgo, including taxation and profits, are ignored.

4.3 *The assumed relationships between genotype and environment in the model*

The environmental and genetic components of the phenotypic liability L_j (expression (1)) are rarely additive. If they were, then the interaction term I_j would be zero. There is much evidence in biology, however, which shows that the phenotypic advantage of one genotype over another can be altered and even reversed in different environments. The classic case in humans is that of sycle-cell anaemia (see Hedrick, 1985). The presence of the sycle-cell gene provides resitance to malaria, and hence gives survival advantage over normal genotypes in environments where malaria is prevalent; where malaria is absent the gene leads to a form of anaemia which reduces the survival of

the carrier compared with normal genotypes. From the evidence available it would seem that I_j is of considerable importance in any genetic-environment model.

The possible interaction effects between the two environmental types and the two genetic types in our experiment are, of course, infinite. In order to restrict the number of variations to a manageable level, we will assume that each risk type E*i*G*j* has the pattern of sickness rates defined by one of the four assumed phenotypic distributions defined in Table 1. Possible allocations of the risk types to the four distributions are shown in Table 4, and an interpretation of each allocation (which will be referred to as models M1 - M5) follows.

Table 4. Example allocations of the four risk types among the four distributions.

	Distribution			
Model	1	2	3	4
M1	E1G1	E1G2	E2G1	E2G2
M2	E1G1	E2G1	E1G2	E2G2
M3	E1G1	E1G2	E2G2	E2G1
M4	E1G1	E2G1	E2G2	E1G2
M5	E1G1	E2G2	E2G1	E1G2

M1 This model has E2 > E1 and G2 > G1 (ie the sickness rates in E2 are greater than those for E1, and the sickness rates for G2 are greater than those for G1, all else being equal). The environmental effect is stronger than the genetic effect on sickness (ie the difference in sickness rates between environments for the same genetic type is greater than the difference in sickness rates between genetic types for the same environment).

M2 Again E2 > E1 and G2 > G1. However in this model the genetic effect on sickness is stronger than the environmental effect.

M3 Here G2 > G1 in environment E1, but G1 > G2 in environment E2, which is therefore an example of a genetic-environment interaction effect. However the environmental component has the strongest overall effect on sickness rates.

M4 This is the same as M3 except that the genetic effect is dominant.

M5 Again an interaction is present, but with environmental and genetic effects equally dominant.

While there are 20 other possible allocations, they can all be grouped according to their nature into one of the five models M1 - M5 described above. (Specifically there are 8 M5 combinations, and 4 combinations for each of M1 - M4). Hence we can study the efect of all 24 different arrangements by considering the five models listed in Table 4.

5. *Illustration of the use of the model: results.*

5.1 *Calculation of expected present values, standard basis*

Expected present values of the death benefits for each risk type under each model are shown in Table 5.

Table 5. Expected present values of death costs on standard assumptions

	Risk type			
Model	E1G1	E1G2	E2G1	E2G2
M1	.1126	.1172	.1560	.1806
M2	.1126	.1560	.1172	.1806
M3	.1126	.1172	.1806	.1560
M4	.1126	.1806	.1172	.1560
M5	.1126	.1806	.1560	.1172

The results show that our assumptions lead to relatively little difference in expected claim cost between the two groups of lowest risk, while larger differences occur between groups expressing liability distributions 2, 3 and 4.

5.2 *Modelling the take-up of insurance*

MacDonald (1997) allows for the rate of take-up of life insurance by people with different risk characteristics explicitly in his Markov model. Here a simpler approach will be taken, bearing in mind the assumptions to be made are highly problematic, as MacDonald admits. It will be assumed that companies will be able to price their contracts on the basis of environmental but not genetic evidence (in other words any genetic test information known to applicants is not divulged to the insurer, leading to the potential for anti-selection to occur). We define no anti-selection to be the assumption that all risk types are present in equal proportions in the insuring population, so that the (single) premiums charged for the various risk types, based purely on environmental risk characteristics, are calculated as:

$$P(E1) = P(E1G1) = P(E1G2) = 0.5\ [\ V(E1G1) + V(E1G2)\ ]$$

and $$P(E2) = P(E2G1) = P(E2G2) = 0.5\ [\ V(E2G1) + V(E2G2)\ ]$$

where P( ) is the premium for risk type ( ), and V( ) is the expected present value of the benefits for risk type ( ) from Table 5.

The premiums charged under this basis are shown in Table 6.

Table 6. Premiums charged for each risk type.

Model	Risk type E1	E2
M1	.1149	.1683
M2	.1343	.1489
M3	.1149	.1683
M4	.1466	.1366
M5	.1466	.1366

The absolute value of the proportionate deviations between the premium charged, and the risk borne, are calculated according to (4) and are shown in Table 7.

$$D(EiGj) = \left| \frac{P(EiGj) - P(Ei)}{P(Ei)} \right| \qquad (4)$$

Table 7. Absolute proportionate deviations between cost and premium

Model	Risk type D(E1G*j*)	D(E2G*j*)
M1	.020	.073
M2	.162	.213
M3	.020	.073
M4	.232	.142
M5	.232	.142

We now make the assumption that genetic information is known to the insurance applicant and, furthermore, the applicant can accurately assess the relative risk that this information implies. The extent to which this assertion is valid is critical, of course, to the degree of anti-selection that might occur. The assertion made here will almost certainly lead to a considerable overstatement of the cost of anti-selction obtained. Hence these results should be considered as presenting very much a worst case scenario for anti-selection involving these complex and mostly multifactorially determined risks.

Retruning to our example, the larger the absolute size of the proportionate deviation, the more likely it would seem for anti-selection to occur. Anti-selection will be caused by a greater proportion of lives in the higher risk group taking out insurance than those in the low risk group, at the average premium for the given environment shown in Table 6. We will adopt the crude weightings listed in Table 8.

Table 8. Proportions of high and low risk groups (in respect of risks in a given environment) taking out insurance

Proportionate deviation	High risk weight	Low risk weight
Less than 5%	0.50	0.50
5% and less than 15%	0.75	0.25
15% and above	1.00	0

The cost of anti-selection can then be calculated, for a particular model, by:

$$C(EiGj) = \frac{\sum_{i=1}^{2}\sum_{j=1}^{2} W(EiGj) \times V(EiGj)}{0.5\left[P(E1) + P(E2)\right]} - 1$$

where W(E*i*G*j*) is the weighting from Table 6 applied to risk type E*i*G*j*. Applying these weights results in the anti-selection costs shown in Table 9.

Table 9. Costs of anti-selection, standard basis.

Model	Anti-selection cost
M1	2.2%
M2	18.9%
M3	2.2%
M4	15.4%
M5	15.4%

Two observation can be made from Table 9: where environmental determinants of risk are dominant to genetic determinants (models M1 and M3), then the premium is largely sensitive to the risk and the cost of anti-selection is minimal. Where genetic effects are dominant then the cost of anti-selection is considerably higher, there being a slightly higher cost where ther genetic and environmental effects operate additively (M2) than when interaction effects are present (M4 and M5).

5.3 *Varying the assumptions*

The costs of anti-selection were recalculated according to the following variations.

V1 Assuming lower rates of sickness as defined by the parameters of the Beta distributions shown in Table 10.

Table 10. Parameters of the Beta distributions used for the reduced sickness rate models

Distribution	α	β
1	3	5
2	3	4
3	4	4
4	4	3

It should be noted that it appeared unrealistic to examine the case where higher sickness rates were assumed, as the sickness rates of the highest risk type in the standard basis was already as high as could be realistically envisaged. The effect of the reduction in rates was (very approximately) of the order of halving the standard sickness rates at each age.

V2 Assuming lower or higher mortality of sick lives.

Lower basis: $\mu^{12} = 0.25$
Higher basis: $\mu^{12} = 1.0$

The results of these variations in terms of their anti-selection costs are shown in Table 11.

Table 11. Anti-selection costs under different sickness and mortality assumptions

Model	Lower mortality	Higher mortality	Lower sickness
M1	1.9%	2.3%	4.0%
M2	12.8%	20.4%	8.9%
M3	1.9%	2.3%	4.0%
M4	13.2%	20.4%	9.6%
M5	13.2%	20.4%	9.6%

At appears that anti-selection reduces slightly with a reduction in the mortality of sick lives. A decrease in sickness rates has a more dramatic effect on the reduction in anti-selection. [It should be noted that the slight increase in anti-selection costs for M1 and M3 in this case results from a switch in weights according to the very crude weighting system adopted.] The overall pattern of differences between the environment dominant models (M1 and M3) and the genetic-dominant models (M2, M3 and M5) remain when the assumptions are varied; however the relatively minor difference between M2 and M4/M5 does not seem to behave consistently, at least under the assumptions made, and therefore cannot be taken to be a general feature.

6. *Discussion*

We have shown how a model for sickness rates can be developed from a genetic viewpoint. The approach taken allows for specific interaction of genetic and environmental components of the phenotypic liability (the propensity to become sick). Assumptions for the distributions of phenotypic liability, in conjunction with an assumed function for T_{x+t}, can be used to generate sickness rates. These can be incorporated into multiple state models of policyholder progression involving healthy, sick and dead states, for the purpose of estimating insurance costs. Issues relating to the usefulness of the sickness rate model have already been discussed at the end of section 4.1.

The three state model of policyholder progression used here is a useful extension to the Markov model approach used by MacDonald (1997), in that it allows for the inclusion of a cause of death from a specified disease or group of related diseases. This is important for assessing the risk from genetic factors as genetic test information will usually relate to the risk of developing specific diseases only, and will often not affect the mortality risk from other causes. However it is pointed out that the model is not satisfactory from the view of assessing health insurance risks, as the claim costs from other causes of sickness are excluded from the model. A revised multiple state model, with a second sick state (for all other causes of sickness) should be introduced for this purpose. This is certainly an area for further investigation, especially due to the extra uncertainty involved in assessing risk under health insurance contracts.

It must be noted that no empirical data have been used in this work, hence the absolute results presented are purely illustrative. The results obtained for the levels of anti-selection are, however, of the same order of magnitude as those shown by MacDonald (1997), and also by Brett (1997), and so may be considered to be reasonably realistic.

Despite the hypothetical nature of the assumptions, an important message emerges regarding the implication of the genetic-environment relationship for the expected cost of anti-selection. Where differences between environmental factors have greater effect on risk than the differences in genetic factors, then insurance rating by environmental factors will prevent any significant anti-selection risk. However where genetic factors are more influential than the environment in the determination of risk, then the possibility of anti-selection becomes more significant.

The importance of identifying the genetic-environment interaction in the determination of risk cannot be underestimated. In this paper all variations of the genetic-environment relationship were assumed to be correctly interpreted by the insurance applicants. In practice, assuming genetic test results are provided to the insurer, the insurer should be in a better position than the applicant to read the genetic information correctly and to translate this into the appropriate risk assessment, in the context of possibly interacting environmental factors. Correct reading by the insurer of the combined effect on risk from the genotype and the environment must be made if appropriate risk classification of multifactorial traits is to be considered as a realistic proposition.

Whether risk classification using genetic information relating to the major late-onset diseases becomes a practical possibility remains to be seen. This paper demonstrates that, even in the unlikely scenario where insurance applicants have perfect genetic

knowledge *and* perfect understanding of how this translates into risk, the cost of anti-selection does not seem to be extreme. In the much more likely scenario in which there is imperfect knowledge, and almost certainly imperfect understanding particularly of the genetic-environment interaction in determining risk, then the effects of anti-selection are likely to be minimal. The only scenarios where multifactorial genetic testing may lead to a significant risk of anti-selection, and hence could be important for risk classification, is where:

(a) risk is much more heaviliy affected by genetic rather than by environmental factors; and

(b) there is no significant interaction effect between the genotype and the environment in the determination of risk, so that the effect of the genotype on risk is likely to be correctly interpreted by both applicants and insurers.

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