PHILOSOPHICAL TRANSACTIONS OF THE ROYAL SOCIETY B BIOLOGICAL SCIENCES

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Phil. Trans. R. Soc. Lond. B 1997 **352**, 1067-1075
doi: 10.1098/rstb.1997.0087

How will improved forecasts of individual lifetimes affect underwriting?

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SUMMARY

The combined effects of underwriting and adverse selection among heterogeneous populations are considered, using a simple Markov model. I illustrate the possible extent of the costs of adverse selection; in all cases, above-average sums assured is the most significant factor.

1. UNDERWRITING AND ADVERSE SELECTION

(*a*) *Introduction*

Of all the questions raised by the prospect of genetic testing, few are more contentious than those concerning insurance. It would be easy to interpret this as a straightforward clash of cultures—on one side those concerned to remove discrimination, on the other the insurance industry which lives by discrimination—but that simple picture tends to obscure some of the issues.

One of these issues is the nature of the insurance industry as it is now, and as it might develop in future. The current position is this: (i) there is a huge life insurance industry, run on wholly voluntary lines and with no counterpart in the welfare state; and (ii) there are relatively small sickness, medical care and long-term care insurance industries, run on voluntary lines alongside massive, mostly compulsory provision within the welfare state.

Much of the heat which has been generated in the debate is because of the political climate, in which both major parties appear to support some level of replacement of state provision by private provision in (ii) above. This leads to fears that universal provision will be replaced by selective provision, with insurers able to exclude bad risks. This is, after all, the model of insurance underwriting which is well understood by the public. Genetic testing did not create these fears, but it has highlighted them.

We have two questions to address, and it causes some confusion if they are not regarded separately. One is the way in which a move from state to private provision is to be achieved, if at all, while retaining the universal coverage of the welfare state. The implicit question of whether such a switch should take place or not is the province of economists and politicians, there is nothing actuarial about it, and I am going to leave that question aside. Actuaries have a more legitimate interest in the means than the aims. The second question is the more immediate one of how genetic testing affects private life insurance, and that is one which the actuarial profession is better able to tackle.

It might be thought that answering the second question would help to answer the first question, but that is not necessarily so. Life insurance is much simpler than other forms of insurance, such as permanent health insurance (PHI). Solutions or compromises which might work for life insurance need not work even in the relatively small existing market for PHI, let alone in any larger market.

(*b*) *Underwriting the welfare state*

The first question can be put thus: can private insurance, including its model of underwriting, replace welfare provision, especially in unemployment, income support and health care? These questions have been convincingly addressed by economist Nicholas Barr (1993), the essence of whose analysis is as follows.

(i) The 'actuarial model' of private insurance is that in which premiums are related to individual risks.

(ii) For private insurance along actuarial lines to be feasible, five conditions must hold: the risk that any individual claims is independent of the risk that any other individual claims; the insured event must not be certain to occur, or there must be uncertainty as to its timing; the probability of claim must be known or estimable; the purchaser must not be able to conceal relevant information from the insurer; and the purchaser must not be able to manipulate the probability of claiming.

(iii) If the purchaser can conceal relevant information, *adverse selection* can occur. High-risk individuals will be more likely to buy insurance, prices will rise and fewer low-risk individuals will find it worthwhile to buy insurance.

(iv) If the purchaser can manipulate the probability of a claim, there is *moral hazard*. Moral hazard is not a large problem in life insurance, because the insured event is so undesirable, but it is a considerable problem in PHI business, which is one reason why life insurance practice cannot simply be extrapolated to other insurances.

Underwriting is part and parcel of the actuarial model; its role is to control adverse selection. It is by definition discriminatory. Some forms of discrimination just seem to have been accepted, for example charging

higher premiums to older lives. Others arouse fierce controversy, including race, sex and now genetic make-up; these clash head-on with our models of society. If society outlaws certain forms of discrimination in underwriting—and its right to do so is surely beyond dispute—there is *asymmetric information*; the actuarial model of insurance does not apply in its pure form, and it might be a sufficiently unstable pooling arrangement as to be unworkable. Barr (1993) set out alternative pooling arrangements to meet this case, generally relying on compulsory membership.

Asymmetric information violates the pure actuarial model but need not necessarily cause it to break down. The key issue is whether the information in question is related to an incentive to buy insurance. For example, if the Sex Discrimination Act were amended so that purchasers of life insurance did not have to state their sex, insurers would be deprived of a piece of information known to be very relevant, in statistical terms. However, it probably would not give a direct incentive to buy insurance, so there would be no adverse selection. There would be greater variability in the risk pool, but probably not enough to destroy the system. Wilkie (1997) and Le Grys (1997) have told us how much variability is in the risk pool ; it is quite a robust entity, as long as adverse selection can be controlled.

Genetic testing is different, because it brings with it an incentive to buy insurance. However, that alone might not make actuarial pooling of risks unworkable. The pure model, as described by Barr (1993), is certainly polluted, but there are many other departures from it in practice anyway. Adverse selection has the potential to break the system, but in reality it depends on how expensive it is likely to be. A modest amount would surely be absorbed, although no doubt under protest, so it is important to have some idea of the possible costs. That is where the actuarial profession has a part to play.

In the remainder of this paper, I will describe some very crude experiments in assessing the impact of genetic testing on life insurance costs, assuming that underwriters are denied access to the results of any genetic tests. There are three (very strong) health warnings: (i) it is not possible at this stage to do any more than work out the consequences of some simple models which include some speculative assumptions; (ii) I ignore the possibility that broader restrictions might be placed on underwriting, as a result of changing attitudes towards discrimination, or otherwise; and (iii) the conclusions cannot be extrapolated to other forms of insurance, in particular alternatives to the welfare state, where anything but a compulsory scheme would seem, just now, to be a perilous experiment.

2. LIFE UNDERWRITING PRACTICE

(*a*) *The tools of underwriting*

The main source is Leigh (1990). Insurers obtain medical information by (i) asking questions on the proposal form; (ii) asking for a report from the applicant's doctor; or (iii) requiring the applicant to undergo a medical examination.

Table 1. *Typical medical underwriting limits* (*Leigh (1990)*; *updated by Leigh*, *personal communication*)

age next birthday	medical attendant's report (£)	medical examination (£)
up to 40	120000	300000
41–50	100000	200000
51–55	75000	125000
56–60	40000	75000
61–65	15000	25000
66–75	all	all

Table 2. *Typical medical underwriting of bronchitis* (*Leigh 1990*)

frequency	time off work	regular cough?	signs on MER	extra mortality
winter	few days	possibly	none	to 75%
often	4 weeks p.a.	yes	rhonchi	100–125%
all year	often	chronic	many	150% to decline

The last two are expensive, so are used sparingly—generally if the proposal form reveals a poor medical history, or is for a high sum assured. All insurers have *medical limits*, namely limits on the cover which can be obtained without moving up to the next level of underwriting; those in table 1 are typical.

Financial underwriting takes place also for high sums assured, to verify a reasonable need. Typically this might begin at £100000 for the cheapest forms of life cover, and £250000 for the more expensive savings contracts with life cover included (Leigh 1990).

Most life insurance medical underwriting is based on the *numerical rating* system. In this, each possible impairment is assumed to increase mortality by a percentage of the average. For example, table 2 shows a typical guide to underwriting bronchitis.

Some risks, for example hazardous occupations or pursuits, are less dependent on age, and so a level addition to the rates of mortality is appropriate. We will not consider risks of this type here.

The effect on the premium depends on the type of contract. Under a term assurance contract (pure life cover) the extra premium for the risk alone would be broadly in proportion to the extra mortality, although the increase in the overall premium would be somewhat smaller because of the expense loadings. Under an endowment assurance contract (substantially savings) the increase would be much smaller in relative terms.

(*b*) *Underwriting categories*

The numerical rating system is fairly rough, but it does give a guide to the expected mortality of those who fall into the following underwriting categories: (i) the *ordinary rates* (OR) class includes lives who can be

Table 3. *Percentage of applications accepted at ordinary rates by one large UK life insurance company*

	sales channel			
contracts	Independent Financial Adviser	tied agency	direct	total
term assurance	94.5	96.3	96.0	95.3
all business	93.9	96.8	96.1	95.9

offered life insurance without an extra premium; (ii) the *rated-up* class includes lives who can be offered life insurance subject to an extra premium; and (iii) the *declined* (or uninsurable) class includes lives who would not be offered life insurance, or whose application would be deferred.

The remarkable feature is the extent of the OR class. In the UK, it includes up to 97% of applications (Leigh 1990), elsewhere in Europe 95% or over (Chuffart 1996), and in the USA about 91% (Pokorski 1996). Table 3 shows the extent of the OR class in one large UK life insurer; notably, it is as high for term assurance as for the business as a whole. Leigh (1990) and Chuffart (1996) estimate the proportion of applications declined as about 1%, which is also about the level in the company described in table 3.

The extent to which applicants are self-selected, so that these proportions are overstated, is unknown; possibly the proportion of the whole population which would fall into the OR class is lower. Even so, the size of the OR class implies that (i) it contains lives of above average mortality (Leigh (1990) stated that it would typically extend to about 130% or 150% of the average mortality); (ii) there is a considerable amount of cross-subsidy; and (iii) life underwriters use much broader rating categories than e.g. motor insurers, and there is little or no 'cherry-picking'.

Brackenridge (1962) said, 'The object of underwriting should be to accept as large a proportion of cases as possible at ordinary rates of premium, leaving only a small percentage of sub-standard lives to be rated according to the risk of the particular impairment present. Very few cases should be declined outright.' This flies in the face of economic theory, which says that good risks should be able to obtain cheaper cover; low prices and high barriers are one way to cream off profitable business. The traditional ethos, exemplified by Brackenridge, comes from an older, perhaps paternalistic, approach; it might not survive in a more ruthless market. If it does not, very wide differences in premiums might arise, whether or not genetic testing becomes an issue.

(*c*) *Genetic disorders*

The following brief list is based on Chuffart (1996).

(i) Under *monogenic* disorders, a modification in a specific gene leads to a specific clinical outcome, such as Huntington's chorea. Some such conditions are already disclosed to underwriters on the basis of family history.

(ii) If genetic material is missing from, or added to, a chromosome, the disorder is *chromosomal*; Down's syndrome is an example. Symptoms are usually present from an early age.

(iii) *Multifactorial* disorders cover combinations of defects, which, with environmental factors, indicate increased risk of death by many of the commonest causes. Genetic testing might reveal these long before any symptoms are present. The additional risk is likely to be variable but rather less than in the case of monogenic disorders; Chuffart suggests 10–20%.

(iv) *Somatic* genetic disorders are those which arise from changes to genetic material after birth.

(*d*) *The insurance risk*

Barr's second condition, for insurance to be feasible, was that the insured event should not be certain. Just as important to a life insurer, however, is that the *time* of death should not be certain. For this reason, monogenic and multifactorial disorders are likely to have quite different implications for insurers.

(i) A monogenic disorder not only pinpoints the most likely cause of death, but also bounds the age at death within a much narrower range (with high probability). It is the latter, more than the former, that represents the increased risk under a life insurance policy.

(ii) A multifactorial disorder might indicate a likely cause of death, but what matters is whether it indicates a much advanced *time* of death. Absolute certainty as to the cause of death would be no problem (to an insurer) as long as the future lifetime was not too different from the usual. In the case of the most common causes, this is quite plausible. I should point out that this is one feature of life insurance which might not be reflected in other forms of insurance.

In other respects too, the risks which multifactorial disorders present are much less clear. They might appear quite different to sufferers, their medical attendants and insurers. Anyone told that they have a 10% or 20% extra risk of dying, every year, should be concerned; however, only at the margins of an underwriting class would this matter. Furthermore, among younger lives these disorders might be present among healthy and unhealthy alike, for example because of lifestyle, and the overall mortality of a sufferer need not be above average. We suggest that the impact on life insurance costs of genetic testing, and restrictions on underwriting, might take two forms: (i) a change in the insuring habits of lives who would currently be acceptable at ordinary rates, or with a modest extra premium; and (ii) the addition to the insurance pool of a small proportion of lives currently not acceptable for insurance. We will attempt to model these separately.

3. A MODEL OF ADVERSE SELECTION

(*a*) *A Markov model*

Three main elements govern adverse selection in life insurance: (i) the rate at which people buy life insurance. If this is high, the impact of a small

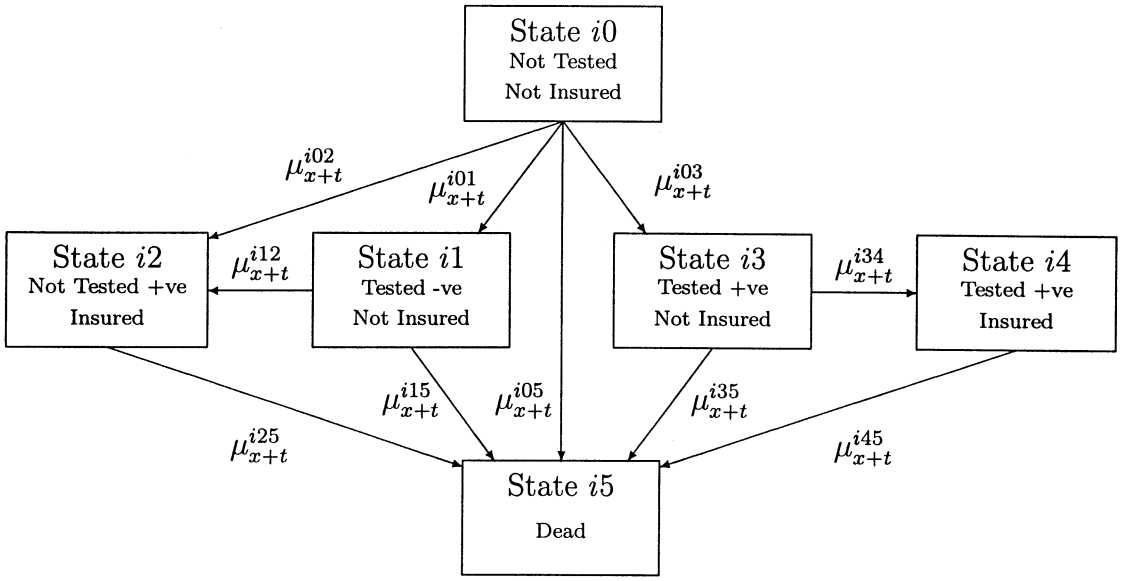

Figure 1. A Markov model for the ith of M subgroups.

number of 'adverse selectors' is relatively smaller. In the limit, if everyone had adequate coverage, provision would be compulsory in all but name; (ii) the extent to which people with a known risk factor, who are potential 'adverse selectors' , are more likely to buy insurance; and (iii) the extent to which 'adverse selectors' also insure their lives for higher amounts.

To represent these, we use the Markov (multiple state) model of figure 1. It models the history of a single life, who is assumed to start at age x in the originating state, and to move between states as shown by the arrows, with probabilities governed by the *transition intensities*, μ^{i01}_{x+t}, μ^{i02}_{x+t} and so on. Specifically, a life in state ij, at time age $x+t$, moves to state ik, during the next dt, with probability $\mu^{ijk}_{x+t}\,dt$, provided dt is small. We express the intensities using a time unit of one year. The main features of the model follow:

(i) In the originating state, a life is not insured and has not had any genetic test. From there, a life can die, obtain insurance without taking a genetic test, or have a genetic test with a positive (meaning that a disorder is present) or a negative result.

(ii) The rate of movement from the originating state and the 'tested but negative' state into the insured state models the 'normal' level of insurance against which adverse selection is measured.

(iii) The rates of movement from the originating state into the two tested states model the extent of genetic testing, and their difference models the likelihood of a genetic disorder being present.

(iv) The rate of movement from the 'tested and positive' state into the corresponding insured state models the incentive of potential 'adverse selectors' to insure themselves.

The behaviour of insurance companies is also captured by this model. For instance, if we disallow movements from the originating state directly into the insured state, we model the effect of all applicants being required to take a genetic test. If we disallow transitions from the 'tested and positive' state into the insured state, we model the declinature of these lives. However, the aim of this work is to measure adverse selection assuming that insurers do *not* behave in this way.

(*b*) *The model of heterogeneity*

To represent different levels of underlying mortality, we suppose that the population is divided into M subgroups, labelled $i = 1, 2, \ldots, M$, within each of which everyone suffers the same mortality as a proportion of the average. In each subgroup, we suppose that insurance behaviour is represented by a model like that in figure 1, with different transition intensities. We suppose we know, or can estimate, the proportion of the population in each subgroup.

For a baseline (average) force of mortality we use a Gompertz formula:

$$\mu_{x+t} = 0.00002072\mathrm{e}^{0.103571(x+t)}, \qquad (1)$$

chosen so that ${}_{30}p_{30}$ and ${}_{60}p_{30}$ are the same as those of the 'AM80 ultimate mortality table', based on male assured lives during 1979–82.

The baseline 'force of insurance' (that is, transition intensity from uninsured into insured states) is harder to calibrate. In 1994, 2865000 new life insurance policies were sold in the UK, with premiums amounting to £1407 million. Term assurance business comprised 25% of these policies and 15% of these premiums. Given that most insurance is bought by economically active people with dependents, a constant intensity of 0.05 is on the low side (it means that an uninsured life has about a one in 20 chance of buying insurance during the next year). This is purely a nominal figure against which the *relative* prevalence of genetic testing can be measured.

(*c*) *Insurance payments*

We introduce the following insurance payments. (i) As long as a life aged $x+t$ is present in a given state ij (that is, in the jth state of the ith subgroup), a premium is payable *to* the insurer, at rate b^{ij}_t (not necessarily

Table 4. *Baseline model—expected present value of £1 death benefit: baseline mortality*

state at outset	age 30 10 years	age 30 20 years	age 30 30 years	age 40 10 years	age 40 20 years	age 50 10 years
insured	0.00611	0.01638	0.03322	0.01708	0.04507	0.04722
uninsured	0.00141	0.00688	0.01894	0.00392	0.01881	0.01078

constant). (ii) On a transition from state ij to state ik within the ith subgroup, a benefit of b^{ijk} (which does not depend on age $x+t$) is paid *by* the insurer. For simplicity, we suppose that the normal sum assured is £1. (iii) $b_t^{ij} = b^{ijk} = 0$ unless state ij is one of the insured states.

We suppose that premiums are multiples of the baseline force of mortality; a life granted ordinary rates will pay premiums at a rate of μ_{x+t} per unit sum assured at age $x+t$; a life rated at +100% will pay premiums at a rate $2\,\mu_{x+t}$, and so on. This scheme of premium payment is actuarially 'fair', meaning that the expected present values of benefit outgo and premium income are equal, but it is not the only such scheme.

(*d*) *The approach used*

Choose a life, at random from the population, aged x, and observe this life for n years. We do *not* know to which of the M subgroups the life belongs, but we do observe transitions into tested or insured states, and death; that is, at any age $x+t$ with $0 \leqslant t \leqslant n$, we know which state in figure 1 a life occupies, but not the value of i. Any set of transitions, and the times at which they are made, comprise a lifetime which we suppose to be a random drawing ω from the set of all possible lifetimes Ω. Knowing the life history, we also know the amounts and times of all insurance payments, and we can compute their present value at age x, denoted (ω). Finally, if we know the probabilities: $p_i = P$[life is in the ith subgroup], where $(1 \leqslant i \leqslant M)$, we can compute any moments of $L(\omega)$ (Norberg 1995); in particular, the expected present value of future payments, $\mathrm{E}[L(\omega)]$.

For illustration, we consider starting ages of $x = 30$, 40 and 50, and terminal ages $x+n = 40$, 50 and 60. We assume that the force of interest is a constant, $\delta = 0.05$.

(*e*) *Baseline expected costs*

Before considering adverse selection, take the simplest case of a single, homogeneous population, and no genetic testing. That is, $M = 1$ and $p_1 = 1$. Table 4 shows the expected present value of a death benefit of £1, payable on death while insured. The first line shows the costs assuming the life to be insured at outset; this is the expected present value which an actuary would use to compute a premium for an applicant. The second line shows the costs assuming the life to be uninsured at outset. These are much lower, because: (i) a life might die before taking out insurance; or (ii) a life might never take out insurance.

We concentrate on lives uninsured at outset, since adverse selection takes place against a background of 'normal' insurance activity. In the next sections, we consider the simplest possible heterogeneous models; those with two subgroups, $M = 2$. We will calculate the expected present value of the insurer's loss, under various assumptions about adverse selection, and we express this as a percentage of the expected present value of the benefit payment in the absence of adverse selection.

4. A MODEL OF THE ORDINARY RATES CLASS

(*a*) *The model of heterogeneity*

Let $M = 2$. We suppose that there is a 'low mortality' subgroup, group 1, suffering 75% of average mortality; and a 'high mortality' subgroup, group 2, suffering 125% of average mortality. Bearing in mind the composition of the OR class, this is not extreme. These assumptions are arbitrary but that does not impair the generality of the conclusions; the additional costs of adverse selection are roughly in proportion to the mean extra mortality in the 'high mortality' subgroup(s), which here is +25%.

If there is no adverse selection, the expected present values of benefit payments, or losses allowing for premium payments, are very close to those in the model with $M = 1$. For simplicity, we suppose that insurance behaviour in group 1 is unaffected by genetic testing. This might overstate costs, since selection within group 1 would be beneficial.

(*b*) *Genetic testing*

The rate of genetic testing in group 2 is represented by $\mu_{x+t}^{201} + \mu_{x+t}^{203}$, and the chance that a test should be positive by $\mu_{x+t}^{203}/(\mu_{x+t}^{201} + \mu_{x+t}^{203})$. We contrast two possibilities: (i) a relatively low incidence of genetic testing, $\mu_{x+t}^{201} + \mu_{x+t}^{203} = 0.05$, so that genetic testing is no more common than buying insurance; or (ii) a much higher level of genetic testing, $\mu_{x+t}^{201} + \mu_{x+t}^{203} = 0.25$, so that most of the population is tested within a few years.

In each case, we suppose that the chance that a test is positive is 20%. The chances that a life in group 2 should be tested and found positive over a 30 year period are then just under 10% and just over 15%, respectively; according to some estimates of the incidence of genetic disorders these are quite high (Chuffart 1996).

(*c*) *Adverse selection*

We model adverse selection by varying two more components in group 2: (i) the rate of transfer to the insured state from the 'tested and positive' state, μ_{x+t}^{234}; and (ii) the sums assured taken out by 'adverse selectors', b^{245}.

Table 5. *Ordinary rates class: mean present value of loss, all sums assured £1, as a percentage of baseline costs*

level of testing	μ^{234}_{x+t}	age 30 10 years %	age 30 20 years %	age 30 30 years %	age 40 10 years %	age 40 20 years %	age 50 10 years %
low	0.25	0.7	0.7	0.4	0.8	0.5	0.6
	0.50	1.4	1.0	0.5	1.0	0.7	0.9
	1.00	1.4	1.0	0.6	1.3	0.9	1.2
high	0.25	2.9	1.9	1.1	2.6	1.7	2.3
	0.50	3.6	2.3	1.2	3.6	2.1	3.5
	1.00	4.3	2.5	1.3	4.3	2.3	4.3

Table 6. *Ordinary rates class: mean present value of loss, higher sums assured, as a percentage of baseline costs*

level of testing	μ^{234}_{x+t}	b^{245}	age 30 10 years %	age 30 20 years %	age 30 30 years %	age 40 10 years %	age 40 20 years %	age 50 10 years %
low	0.25	2	2.1	2.2	1.8	1.8	2.0	1.7
	0.25	4	4.3	5.1	4.8	4.1	4.8	4.0
	1.00	2	3.6	2.8	2.2	3.3	2.6	3.1
	1.00	4	7.1	6.4	5.5	6.9	6.2	6.7
high	0.25	2	6.4	5.4	4.1	6.4	5.2	6.0
	0.25	4	13.6	12.7	10.4	13.8	12.4	13.6
	1.00	2	10.0	6.7	4.7	10.2	6.5	9.9
	1.00	4	21.4	15.1	11.4	21.7	14.9	21.4

Table 5 shows, with 'low' and 'high' incidence of genetic testing as above, the expected losses per life resulting from $\mu^{234}_{x+t} = 0.25$, 0.5 or 1.0. The corresponding probabilities that a life in the 'tested and positive' state will buy insurance during any year are 0.22, 0.39 and 0.63, respectively; this represents extreme adverse selection. The losses are expressed as a proportion of the expected present value of the benefit costs in the absence of adverse selection. None is high enough to be troublesome; the highest (about 4%) result from considering short terms of insurance, while 'adverse selectors' would possibly prefer longer terms, including more likely ages at death.

Next, we suppose that lives in group 2 who have tested positive apply for sums assured twice or four times as high as other lives. Table 6 shows the expected losses as proportions of the baseline costs. Only in the most extreme cases, and over short periods, do they exceed 20% of the baseline costs. The longer the period considered, the greater the numbers who buy insurance anyway, and the larger the pool which bears the cost of adverse selection.

Tables 5 and 6 suggest that adverse selection with average sums assured might not, by itself, have a large effect on the OR class; the most costly aspect is likely to be higher than average sums assured. This highlights the importance of limiting the sums assured which might be obtained without disclosure of known genetic information.

The higher figures in table 6 require the following, possibly unlikely, circumstances to hold. (i) The incidence of genetic testing is so high that almost all of the population will be tested within a few years. (ii) One in five of all lives tested in group 2, test positive. (iii) Almost all lives who test positive take out insurance of more than the average amount within a few years of being tested (and, by implication, can afford to do so). (iv) Genetic testing leads to no medicinal benefits or improvements in lifestyle. (v) There is no change in the insurance behaviour in group 1, although some of these lives might also test positive.

The technical losses above are greater than they would be under level premium contracts, because no policy reserves are held. Also, taking the insurance market as a whole, the 'normal' rate at which young lives buy insurance is probably higher than the rate of 0.05 which we assumed, independent of age.

5. A MODEL OF AN EXTENDED ORDINARY RATES CLASS

(*a*) *The model of heterogeneity*

We now assume that group 1 represents the *current* OR class, and has mortality 100% of the baseline, with no adverse selection. We suppose that the mortality in group 2 is 200% of the baseline. The Continuous Mortality Investigation Bureau (CMIR **14** 1995) found that mortality under rated-up assurances varied widely, from less than expected to over 200% of expected. For calculating baseline costs, we suppose that lives in group 2 would currently pay premiums $2\mu_{x+t}$ per unit sum assured.

The insurance behaviour of lives in group 2 can only be guessed. If we suppose that they are just as likely to insure as lives in group 1 ($\mu^{102}_{x+t} = \mu^{202}_{x+t} = 0.05$), we have a basis for testing other possibilities.

The proportion of applications currently accepted at ordinary rates would suggest $p_1 = 0.95$ or thereabouts, but this might be higher than in the population as a

Table 7. *Whole population: expected present value of £1 death benefit, life uninsured at outset, no adverse selection*

p_1	age 30 10 years	age 30 20 years	age 30 30 years	age 40 10 years	age 40 20 years	age 50 10 years
0.95	0.00147	0.00721	0.01980	0.00411	0.01967	0.01128
0.90	0.00154	0.00755	0.02067	0.00431	0.02053	0.01178
0.85	0.00161	0.00788	0.02153	0.00450	0.02138	0.01228

Table 8. *Whole population: mean present value of loss with ordinary rates granted to lives in group 2, with and without adverse selection, but same sums assured, as a percentage of baseline costs*

adverse selection	p_1	age 30 10 years %	age 30 20 years %	age 30 30 years %	age 40 10 years %	age 40 20 years %	age 50 10 years %
no	0.95	4.8	4.7	4.6	4.6	4.6	4.6
	0.90	9.1	9.0	8.8	9.1	8.8	8.8
	0.85	13.0	12.9	12.6	12.9	12.6	12.7
yes	0.95	13.6	9.6	7.5	13.6	9.4	13.5
	0.90	26.6	18.4	14.4	26.2	18.1	25.7
	0.85	37.9	26.4	21.1	37.6	26.0	37.1

Table 9. *Whole population: mean present value of loss, with adverse selection, $p_1 = 0.95$, as a percentage of baseline costs*

μ_{x+t}^{234}	b^{245}	age 30 10 years %	age 30 20 years %	age 30 30 years %	age 40 10 years %	age 40 20 years %	age 50 10 years %
0.25	1	1.4	1.4	1.2	1.5	1.3	1.4
0.25	2	2.7	2.6	2.3	2.9	2.6	2.7
0.25	4	5.4	5.4	4.6	5.6	5.3	5.6
0.50	1	1.9	1.5	1.2	1.9	1.4	1.8
0.50	2	4.1	3.1	2.4	3.6	2.9	3.6
0.50	4	7.5	6.1	4.5	7.5	5.9	7.3
1.00	1	1.9	1.5	1.2	2.0	1.4	2.0
1.00	2	4.1	3.2	2.5	4.4	3.1	4.3
1.00	4	8.8	6.4	5.0	8.5	6.2	8.4

whole. Table 7 shows the expected present values of benefit payments, assuming that $p_1 = 0.95$, 0.90 or 0.85, for a life uninsured at outset. Again, we take these as our baseline costs.

Table 8 shows the mean losses which would arise if *all* lives in group 2 were granted ordinary rates, with and without some adverse selection; under the latter, all rates of transition into insured states in group 2 are 0.25, but without increased sums assured. (This table has nothing to do with genetic testing *per se*, but indicates the effect of more severe restrictions on underwriting generally. There might be good financial reasons to maintain as broad an OR class as possible.)

(*b*) *Genetic testing in group 2*

We suppose that genetic tests in group 2 identify lives with monogenic disorders and, possibly, severe multifactorial disorders. About 1% of lives born have a monogenic disorder (Chuffart 1996); not all will live to the insuring ages, but we suppose that, overall, about 1% have disorders of financial significance. The larger p_1 is, the higher should be the incidence of positive tests in group 2. For example, based on 1% of the whole population, with $p_1 = 0.95$ we might get up to one in five tests positive, with $p_1 = 0.9$ up to one in ten, and so on. Then we find that the costs of adverse selection due to genetic knowledge alone is almost independent of p_1; in what follows we suppose $p_1 = 0.95$, $\mu_{x+t}^{201} = 0.2$ and $\mu_{x+t}^{203} = 0.05$. We ignore the possibility that underwriting restrictions will extend to non-genetic information.

(*c*) *Adverse selection*

We model adverse selection by assuming that (i) $\mu_{x+t}^{234} = 0.25$, 0.5 or 1.0; (ii) sums assured are one, two or four times the average; and, (iii) lives who have tested positive are charged ordinary rates. Other lives are rated-up as usual. Table 9 shows the mean losses, as proportions of the baseline costs. As before, an increased tendency to seek insurance after a positive test result does not, by itself, make much difference; the costly element is above-average sums assured.

6. A MODEL OF SEVERE LATE-ONSET DISORDERS

Under some disorders, symptoms appear relatively late in life, so mortality is only higher at older ages. This differs from the 'proportional hazards' assumption used above. Suppose, for illustration, that group 1

Table 10. *Late-onset disorders: mean present value of loss, $p_1 = 0.995$, as a percentage of baseline costs*

b^{245}	age 30 30 years %	age 40 20 years %	age 50 10 years %
1	5.1	8.4	14.1
2	9.4	15.5	25.4
4	18.1	29.5	48.2

contains 99.5% of the population, with the baseline mortality, and group 2 contains 0.5% of the population, whose mortality is the same as the baseline up to age 50, but 20 times the baseline over age 50. This possibly overstates the proportion of sufferers of monogenic disorders who will reach the insuring ages. For these lives, ${}_{10}q_{50} = 0.725$, so they have relatively little chance of survival much beyond 60. Table 10 shows the mean losses, as proportions of baseline costs with group 2 uninsurable, assuming that: (i) $\mu^{201}_{x+t} = 0$ and $\mu^{203}_{x+t} = 0.25$ (a high level of genetic testing); (ii) $\mu^{234}_{x+t} = 1.0$ (very high adverse selection); and (iii) $b^{245} = 1$, 2 or 4 (higher sums assured).

There are only losses if the period considered includes ages over 50, but these losses are considerable.

7. A COMBINED MODEL

Finally, we look briefly at a model with three subgroups. Based on the proportions given by Le Grys (1997), we have 94% of the population with 81.2% of average mortality, 5% with 206% of the average and 1% with 490% of the average. To compute baseline costs we assume that the first group is charged ordinary rates, the second is rated up and the third is declined.

In this model it might be reasonable to suppose that genetic testing with positive results is very prevalent in the second and third groups, we set $\mu^{201}_{x+t} = \mu^{203}_{x+t} = \mu^{301}_{x+t} = \mu^{303}_{x+t} = 0.1$; and we suppose that adverse selection is at a high level, $\mu^{234}_{x+t} = \mu^{334}_{x+t} = 1.0$. Table 11 shows the expected losses with sums assured of one, two or four times the average among the 'adverse selectors'.

8. DISCUSSION

(*a*) *Conclusions*

The figures above are illustrative only, but they suggest the following tentative conclusion. If life insurance companies refrain from using (or are forbidden to use) the results of any genetic test in underwriting, additional mortality costs are likely to arise. However, if adverse selection does not extend to untypically large sums assured the magnitude of these costs is greatly reduced; large sums assured is the costliest aspect of adverse selection. The range of figures suggests that 10% would then be a more realistic order of magnitude for the additional costs than would, say, 100%.

In case society should decide that genetic information should not be used by underwriters, it would seem reasonable to impose an upper limit on the sums assured that can be obtained in this way. The patterns of age- and term-related costs suggests that an age-related upper limit would be reasonable (as in table 1).

(*b*) *Pricing strategies*

In order to price long-term insurance, it is necessary to estimate not only the probability of a claim, but the distribution of the time of a claim, and in the case of PHI, the duration of a claim. The more uncertain these are, the more difficult it is to set premiums at a level which will attract purchasers without threatening the solvency of the insurer. Simply increasing premiums to cope with the cost of adverse selection would therefore be difficult.

There is less of a problem if prices can be set retrospectively, allowing for experience. This is the essence of the with-profits system, over 200 years old. A premium is charged which exceeds any reasonable estimate of the insurance costs, thus protecting solvency. As time passes, profits are earned from the funds built up in excess of the claims experience, and these profits are distributed to the policyholders in the form of bonuses. Thus equity is preserved even though the insurer might be unable to estimate the relevant probabilities in advance, so that Barr's third condition fails.

In the UK, term assurance business has traditionally been priced very competitively, on a non-profit basis, which does rely on good estimates of future mortality. In other European countries, life cover has been considerably more expensive. Bennet *et al.* (1984) found that premiums in Denmark were 15–127% more than their UK equivalents, and in (West) Germany 90–373% more. In these countries, all business has been with-profits, but in others which do allow non-profit term assurances, premiums are still considerably higher than in the UK. The problems faced by UK life insurers, therefore, stem in part from their traditional practices, and will seem less troublesome in other countries whose practices are different. It cannot be said that they are insoluble.

A second form of retrospective pricing is found in unit-linked business, where mortality costs are charged for by deallocating units, according to a scale of charges which can usually be altered if the experience justifies doing so.

Table 11. *Three subgroups: mean present value of loss, as a percentage of baseline costs*

b^{245}	age 30 10 years %	age 30 20 years %	age 30 30 years %	age 40 10 years %	age 40 20 years %	age 50 10 years %
1	10.7	7.7	5.8	10.3	7.3	9.7
2	19.7	15.8	12.0	20.9	14.9	19.7
4	42.6	31.9	24.5	41.8	30.1	38.5

If premiums are not increased, any extra costs will simply reduce the profits available for bonuses and dividends. (This buffer is not available to a unit-linked office, however.) This is a reasonable option if the costs are small; otherwise, it results in one group of policyholders, whose main aim is savings, subsidizing another group whose aim is protection. Leaving aside the equity of such an arrangement, it is likely, if at all significant, to make life insurance a less competitive investment medium, and most insurers would probably prefer to cost life cover explicitly.

The final option would be to pull out of the market altogether, or to decide not to enter a new market. For the reasons given above, this seems unlikely in the case of life insurance, but it might not be unrealistic in other cases.

REFERENCES

Barr, N. 1993 *The economics of the welfare state*, 2nd edn. London: Weidenfeld & Nicolson.

Bennet, I. R., Barclay, K. J., Blakeley, A. G., Crayton, F. A., Darvell, J. N., Gilmour, I., McGregor, R. J. W. & Stenlake, R. W. 1984 Life assurance in four European countries (with discussion). *Trans. Faculty Actuaries* **39**, 170–250.

Brackenridge, R. D. C. 1962 *The medical aspects of life assurance*. London: Staples Press.

Chuffart, A. 1996 *Genetics and life insurance in Europe*. Zurich: Swiss Re.

Continuous Mortality Investigation Bureau (CMIB) 1995 The mortality of impaired assured lives, 1983–90. *Continuous Mortality Investigation Report* **14**, 105–120.

Le Grys, J. 1997 Actuarial considerations on genetic testing. *Phil. Trans. R. Soc. Lond.* B **352**, 1057–1061.

Leigh, T. S. 1990 Underwriting—a dying art? (with discussion) *J. Institute Actuaries* **117**, 443–531.

Norberg, R. 1995 Differential equations for moments of present values in life insurance. *Insurance: Maths & Economics* **17**, 171–180.

Pokorski, R. J. 1996 Use of genetic tests to predict and diagnose cancer: an insurance perspective. *J. Tumor Marker Oncology* **11**, 33–44.

Wilkie, D. 1997 Mutuality and solidarity: assessing risks and sharing losses. *Phil. Trans. R. Soc. Lond.* B **352**, 1039–1044.