*B.A.J.* **9**, IV, 787-874 (2003)

# GENETICS AND INSURANCE — SOME SOCIAL POLICY ISSUES

By C. D. Daykin, D. A. Akers, A. S. Macdonald, T. McGleenan, D. Paul and P. J. Turvey

[Presented to the Institute of Actuaries, 24 February 2003, and to the Faculty of Actuaries, 17 March 2003]

ABSTRACT

Rapid developments in genetic science have been accompanied by confusion regarding the predictive power of DNA-based tests and in the impact of such tests on the insurance industry. The United Kingdom actuarial profession has begun to engage in the associated social policy issues and to try to throw some light on the issues through quantitative research and objective analysis. At the same time, many insurance industry actuaries have been involved in work on behalf of the insurance industry to develop a sound basis for permission to be sought to make use of the results of certain predictive genetic tests. This paper briefly outlines some of the history of the development of the debate in the U.K. and draws together some of the debates and discussions which have taken place within the Genetics Group of the Social Policy Board of the U.K. actuarial profession, as well as providing some pointers to the directions which the debate might take in the future, with some important potential consequences for the insurance industry and for actuaries.

KEYWORDS

Genetics; Insurance; Predictive Genetic Tests; Underwriting; Family History

CONTACT ADDRESS

C. D. Daykin, C.B., Hon. D.Sc., M.A., F.I.A., Hon. F.F.A., F.S.A., Government Actuary's Department, Finlaison House, 15-17 Furnival Street, London EC4A 1AB, U.K. Tel: +44(0)20-7211-2620; Fax: +44(0)20-7211-2650; E-mail: chris.daykin@gad.gov.uk

## 1. Introduction

1.1 Important advances in scientists' understanding of molecular genetics have already begun to have widespread repercussions. The project to map the human genome has progressed more quickly than many expected, and there seems little doubt that the pace of development will accelerate.

1.2 Increased knowledge of the role of genetic factors in particular illnesses will facilitate better treatment and lead to the development of new therapies. It could have major implications for the treatment of some diseases, and it is possible that it could lead to significant increases in life



https://doi.org/10.1017/S1357321700004360 Published online by Cambridge University Press

expectancy in due course. However, genetic epidemiology has progressed at a rather more stately pace than molecular genetics, and the development of sound research into the potential impact of these factors on insurance has lagged still further behind.

1.3 DNA tests may confirm suspicions, for example from family medical history, that someone has a particular genetic mutation. However, for the time being such knowledge may not unlock any cure, or even permit any worthwhile treatment. There is public concern about the potential for the use and misuse of genetic information, particularly by insurance companies and by employers. In part this may be exaggerated, because people expect genetic tests to yield rather more precise and definitely predictive information than is in fact likely to be the case, but a social policy perspective needs to address such concerns, which are widely held.

1.4 Genetic information is different from many other types of medical information, as changes to the genetic code are passed down through families. Some particular genetic mutations are relatively common in certain small ethnic and geographically restricted communities. Genetic information can be regarded as intensely personal property, since a person's genetic code might be regarded by some as the essence of individuality, notwithstanding that many other factors contribute to that person's identity.

1.5 Of direct relevance to the insurance industry is the commonly held and deep-seated fear that increased knowledge of any genetic mutations may render individuals uninsurable, and thus lead to the development of a significant genetic underclass. Also alarming to some is the idea that insurers might seek to identify a genetic 'superclass' of people with particularly positive genetic characteristics, and offer them preferential terms, with a consequent worsening in the terms for everyone else. Genetic information could, some people allege, come to be used like postcodes to provide a more detailed risk classification in life, critical illness, income protection and other forms of insurance, such as that which already exists in motor and property insurance. These fears are mostly founded on theoretical speculation and extrapolation, rather than on evidence. However, the fears are no less real by virtue of the lack of evidence.

1.6 Insurers, on the other hand, are worried about the practical and financial consequences of an increasing number of people knowing from genetic tests that they have a genetic mutation which increases their susceptibility to illness which is either likely to lead to premature death or to early claims on critical illness, income protection or long-term care insurance policies, in particular if the results of any such tests are not also available to the insurance companies (information asymmetry). Insurers see a risk that those who know that they have higher risk of morbidity or mortality will take advantage of this knowledge by applying for insurance they would otherwise not take out (or at higher levels than would otherwise have been the case). (Fischer & Berberich, 1999)

https://doi.org/10.1017/S1357321700004360 Published online by Cambridge University Press

1.7 It is important to emphasise that there are relatively few monogenic conditions, where a single gene mutation is known to bear a close relationship to a particular illness or condition and where a genetic test will provide a clear indication one way or the other as to whether someone is likely to develop the condition in adult life (there are some monogenic conditions which manifest themselves at birth or in childhood, but these are not generally of relevance from the perspective of insurance). Far more common is the situation where a genetic mutation indicates a greater susceptibility, but where a combination of common alleles and genetic mutations, together with various other factors, provokes the eventual outcome. In most cases relatively little is known yet about the dynamics of such conditions, and for the foreseeable future there may be little predictive, or even indicative, value in such genetic tests. In due course, increased levels of testing are likely to be linked to improvements in medication or treatment.

1.8 In April 1997 the Council of Europe adopted a Convention for the Protection of Human Rights and Dignity of the Human Being with regard to the Application of Biology and Medicine. This took a strong stance on genetic tests, reflected in Article 11, which prohibits any form of discrimination against a person on grounds of his or her genetic heritage, and Article 12, which states that genetic testing may only be carried out for purposes of health care or research. This convention has not yet been ratified by the United Kingdom, but it is indicative of a general level of concern about the use of genetic tests other than for therapeutic or research purposes.

1.9 Also indicative of these concerns is the fact that a number of European countries have imposed legislation to prevent insurers from obtaining, or making any use of, information about genetic test results in respect of applicants for insurance (Chuffart, 1996). This is also the case in the United States of America, where concerns are particularly acute in the area of personal medical expenses insurance, since for people of working age, and for their dependants, access to good quality health care is dependent on being able to have such insurance.

1.10 In the U.K. in 1997 the Human Genetics Advisory Commission (HGAC) issued their report: *The Implications of Genetic Testing for Insurance*. The HGAC was concerned about the potential use of genetic test information by insurers, but concluded that a permanent ban on the use of genetic tests in insurance would be inappropriate. Instead, they recommended that insurers should respect a moratorium, for the time being, on the use of genetic tests. They argued, moreover, that a requirement by insurers that prospective policyholders should disclose the results of particular genetic tests would only be acceptable when a quantifiable association had been established between a given pattern of test results and events which were 'actuarially relevant' for a specific insurance product.

https://doi.org/10.1017/S1357321700004360 Published online by Cambridge University Press

1.11 In November 1998 the U.K. Government's response to the HGAC report concurred that a permanent ban on the use of genetic tests by insurers would not be appropriate (Department of Trade and Industry, 1998). The Government welcomed the then recently published Code of Practice on Genetics of the Association of British Insurers (ABI), which committed insurers not to require genetic tests to be taken for insurance purposes, and imposed a limited moratorium on the use by insurers of pre-existing genetic test results, including banning members from using genetic test results to offer better than standard premiums. Such information was to be ignored by insurers for mortgage-related life insurance business for sums assured up to £100,000. The ABI made known a list of predictive genetic tests which it proposed that member insurers should be entitled to use for cases over £100,000 and those not related to mortgages. The ABI also committed that its member insurers would keep logs of cases underwritten with cognisance of a listed test result. In the event that a listed test was not subsequently approved, ABI insurers would backdate and make good any adverse underwriting decision. The Government agreed with the HGAC that an effective mechanism should be established to evaluate the reliability and actuarial evidence relating to the use of specific genetic tests by insurers. Following a period of consultation on how this mechanism could be put in place, the Government established the Genetics and Insurance Committee (GAIC) and the broad-ranging Human Genetics Commission (HGC).

1.12 GAIC was given the role of determining which genetic tests were suitable for use by the insurance industry, and for what specific types of contract. Submissions to GAIC from the insurance industry would be required to demonstrate both the clinical and the actuarial relevance of the particular test. How GAIC interpreted 'actuarial relevance' is discussed later on (see ¶4.1.3), but it is of note that neither HGAC nor the Government appears to have given any indication of what they intended or expected.

1.13 The first submission from the ABI to GAIC was a request to use the results of the genetic test for Huntington's disease in connection with applications for life insurance. This request was approved by GAIC in October 2000. A number of further submissions were made by the ABI at the end of December 2000. These included the use of Huntington's disease test for other lines of business (critical illness, income protection and long-term care insurance) and tests for the BRCA1 and BRCA2 breast and ovarian cancer susceptibility genes and the Presenilin-1 (PS1) and APP gene mutations in relation to early onset Alzheimer's disease. December 2000 was a significant deadline, because the ABI's own commitment had been made in such a way that it felt insurers could not reasonably continue to ask for the results of a test into 2001 if an application had not been submitted to GAIC. In the event, there was some belated criticism that the ABI code could, from the outset, have been interpreted as implying an assumption of approval until disapproved, or an assumption of approval because a

https://doi.org/10.1017/S1357321700004360 Published online by Cambridge University Press

submission had been made. In the wake of recommendations from the HGC (HGC, 2002) and from the House of Commons Select Committee on Science and Technology (HCSTC, 2001), and the Government's response thereto, GAIC (which had halted its deliberations in early 2001 pending the General Election) was reconstituted with a new chair and membership in mid 2002.

1.14 Following an extensive consultation process on its paper 'Whose Hands on your Genes?', HGC recommended that the moratorium on the use of genetic test results by insurers should be extended for a further three years, covering all life insurance policies up to sums assured of £500,000 (HGC, 2002). However, they proposed that the moratorium should not apply to negative test results, so that the application of a blanket moratorium should not inhibit the benefits of improved insurability which could derive from certain negative test results. The HGC was not convinced that the present system of self-regulation was working adequately, based on the ABI's Code of Practice, and called for legislation to enforce the moratorium.

1.15 The HGC also said that it wanted to examine further the use which is made by insurers of family medical history information. Information about conditions from which an individual's forebears or siblings have suffered (or died) can contain a significant element of genetic information. Insurance companies certainly do make use of some family medical history information as a proxy for genetic information. However, family medical history is not uniquely genetic in nature, as it may also contain elements relating to the area of residence, quality of living conditions, diet, exposure to infectious diseases, stress and other factors.

1.16 In response to the HGC's recommendations, the ABI negotiated with the Government a revised moratorium on the use of predictive genetic test results. For five years, from October 2001, U.K. insurers will continue not to require any genetic tests to be taken and will not expect to receive information about genetic test results in respect of applications for life insurance products with sums assured of less than £500,000, critical illness insurance with sums assured of less than £300,000, with corresponding annual amounts for income protection coverage.

1.17 In 1998 the U.K. actuarial profession launched an initiative to establish a broad-based discussion forum on genetics and insurance. With support from The Royal Society, the ABI, the Wellcome Trust, the Nuffield Foundation and the Consumers' Association, the U.K. Forum for Genetics and Insurance (UKFGI) was established in October 1999. It has a broad membership of individuals and corporate entities (professions, charities, research bodies, as well as insurance companies and trade bodies), and aims to promote dialogue on issues of genetics and insurance, to share relevant information, to perform an educative role and to foster and encourage relevant research.

https://doi.org/10.1017/S1357321700004360 Published online by Cambridge University Press

## 2. Some Philosophical Considerations

2.1 *Solidarity and Mutuality.*

2.1.1 Social insurance schemes operate according to principles of solidarity and equality. Individuals contribute a sum to the insurance pool which is not explicitly linked to their actual level of risk. In social insurance systems, any claim which is made on the insurance fund is met from the pool, and may be based on an entitlement arising from contributions (e.g. incapacity benefit in the U.K.) or may be related to the individual's level of need (e.g. income support in the U.K.). Even in the former case there may be a significant degree of redistribution implied, especially in systems with flat-rate benefits and earnings-related contributions. Social insurance programmes based on solidarity principles emerged in many developed nations in the 20th century, following pioneering developments in some countries in the 19th century. In many countries (although not particularly in the U.K.) these schemes are increasingly proving to be unaffordable, and are subject to major structural reforms, often involving the introduction of a greater role for complementary provision through private sector institutions or agencies.

2.1.2 Those who make provision for their life and health risks using private commercial insurance encounter a very different system, generally based on the principle of mutuality. The workings of private commercial insurance are, of course, very familiar to actuaries. It suffices here to recall that each person should pay an insurance premium which is commensurate with his or her actual or perceived level of risk. The higher the risk brought by the proposer the higher the premium, and there is no assessment either of ability to pay or of the adequacy of benefit entitlement in relation to need (unless the amount being purchased appears unreasonably high for their circumstances).

2.1.3 A feature of public policy arguments about genetics and insurance in the U.K. has been confusion between solidarity and mutuality (Wilkie, 1997; McGleenan, 2001). To an extent, many commentators have not troubled to question whether mutuality systems can have elements of solidarity imposed on them indiscriminately. Meanwhile, some people in the insurance industry seem not to have realised that public policy was bound to continue to press, whether reasonably or unreasonably, for greater solidarity. Indeed, there have been many conflicting signals. For example, the flexible pricing of motor insurance risks has been almost unchecked in the U.K., and it might be thought therefore that the principles of a mutuality-based insurance system are reasonably well understood by the public. The flexible pricing of motor insurance premiums is risk-dependent. The need, for reasons of equity, to pay a high premium for an expensive vehicle in an area with a heightened risk of criminal damage or theft is an accepted part of this mass market.

https://doi.org/10.1017/S1357321700004360 Published online by Cambridge University Press

2.2 *Moral Hazard*

2.2.1 The fact that individuals have chosen to take out insurance policies can alter their behaviour patterns in a range of ways. At a relatively benign level, having insurance cover in place can cause individuals to become less risk averse. If insurance provides protection, albeit financial, against a particular risk, then policyholders may exercise less caution in the face of that risk than they might otherwise have done. Studies in the motor insurance field suggest that insurance can weaken the incentive for loss prevention and contribute to an increase in accident frequency (Cummins & Tennyson, 1996). The situation is somewhat different in respect of insurance on human life and health, but the presence of insurance coverage can still affect behaviour, particularly, perhaps, with regard to income protection insurance or personal medical expenses insurance.

2.2.2 Another form of moral hazard arises when policyholders misrepresent information when applying for insurance or when making a claim, or simply withhold information. The increased costs incurred by insurance companies as a result of this are usually borne by other policyholders. Advances in genetic testing may increase the potential for moral hazard. If some individuals are in possession of information about their genetic risk status and that information may not be asked for by the insurer to which application is made for an insurance policy, then the insurer may be exposed to a higher than anticipated number of claims.

2.3 *Concepts of Fairness in Insurance*

2.3.1 In so far as the private insurance market requires the classification of individuals into different risk groups, it involves a degree of discrimination. However, until relatively recently the actuarial practice of risk classification seems to have been generally regarded as an acceptable, or at least a not too invidious, form of discrimination. Even in the latter half of the twentieth century, when issues of anti-discrimination came to the fore and various codes were developed, specific exceptions were often granted for what was described as the 'fair' discrimination practised by insurers, provided that this was based on adequate actuarial or statistical information, although such derogations did not go unchallenged altogether.

2.3.2 The public policy debate on the appropriateness of using genetic information in the underwriting of insurance contracts has been characterised by a clash between different interpretations of what constitutes fair treatment. On the one hand, the insurance industry has relied upon the practice of what is described in the economics literature as 'actuarial fairness', although actuaries and insurers would perceive it in terms of 'actuarial equivalence of risk'. This concept relies on the insurer measuring the risk that an individual brings to the insurance fund and charging a premium which matches that risk as closely as possible. To some extent this concept is in conflict with the principles of risk pooling. The compromise is

https://doi.org/10.1017/S1357321700004360 Published online by Cambridge University Press

that relatively broad risk categories are established, with actuarial fairness or equivalence applied as the principle within each category. The categories by which risks are classified are, however, determined by a mixture of custom and practice and social acceptability, and may well change over time. For example, classification by smoker/non-smoker status would not have been considered acceptable 30 years ago, but it is now almost universal for life insurance products. Insurers would argue that, within the broad bands necessary in modern mass markets, the system conforms to the principles of what can be described as actuarially fair.

2.3.3 On the other hand, it is argued that it is morally unfair to consider certain personal characteristics in the pricing of insurance, notwithstanding the fact that they may be relevant to the individual's level of risk. One objection against risk-rating based on personal characteristics is that these are often outside our personal control. This argument has a strong following in relation to genetic information. For example, since genetic information is immutable or inherited, it is argued that it is unfair to penalise affected individuals with higher premiums when they can do nothing to alter their genes. A similar argument could, perhaps, be applied in respect of classification by age and sex, which have nevertheless been accepted for many years as the most obvious and acceptable rating factors, although discrimination by sex is regarded by some as unacceptable. There are also non-genetic conditions which an insurer would expect to take into account in underwriting and which are outside the individual's personal control, such as raised blood pressure.

2.3.4 A different type of objection to insurance underwriting behaviour arises where particular personal characteristics are also associated with historical patterns of discrimination. It is for this reason that some information, which might be relevant from an actuarial perspective, is not used in the process of risk classification. For example, in some circumstances race might provide actuarially relevant information about mortality or sickness, etc. However, strong anti-discrimination norms tend to ensure that, in most countries, this information is not used by insurers when setting premiums. Whereas, in the U.K. disability discrimination legislation permits insurers nonetheless to discriminate, subject to strict controls, the U.K.'s race discrimination legislation allows no such exemption. No exemption was ever sought by insurers, and the Continuous Mortality Investigation of the U.K. actuarial profession (CMI), for example, has never been in a position to investigate insured population mortality rates subdivided by race, since data are not collected in a form that would enable such an investigation to be carried out.

2.3.5 It is clear, therefore, that a requirement simply that information should be actuarially relevant is not sufficient for society to consider that it is appropriate that the information should be taken into account. Nor is it the case that an insurer will use all actuarially relevant information, even when

https://doi.org/10.1017/S1357321700004360 Published online by Cambridge University Press

there are no restrictions. An insurance market segment may operate on 'actuarial equivalence' with a highly detailed risk classification, but it could work equally well from an actuarial point of view if a less detailed classification were adopted, provided that there is a stable mix of risks within the class. This sort of variety exists between market segments, and any particular segment may also see change in this attribute over time.

2.4 *Adverse Selection.*

2.4.1 Adverse selection is a term used in the insurance industry to describe situations where those seeking insurance cover have more information about their true level of risk than the insurer, and use this to their advantage by purchasing more insurance than they otherwise would have done (or by purchasing insurance when they otherwise might not have done so at all). This could be the result of perfectly rational purchasing decisions by prospective policyholders, rather than any attempt to defraud, but the result, from the insurer's point of view, is that claim costs are greater than might be expected for the risk group, and the cost of insurance will tend to be forced up for other policyholders.

2.4.2 There can be little doubt that any advance in predictive medicine is likely to increase the potential for what the insurance industry will see as adverse selection for some insurance products, such as life and critical illness insurance. Genetic information, where it is actuarially relevant, is little different from other forms of predictive healthcare information. As the accuracy and predictive power of genetic tests increases, individuals may, because of information that they acquire through genetic diagnosis, alter their insurance purchasing behaviour, either knowingly or unwittingly. Whether this happens on any appreciable scale will depend on the accuracy of predictive genetic tests, the cost and availability of the tests, the prevalence and actuarial significance of the genetic diseases and the regulatory environment which persists at the time.

2.4.3 There is little empirical evidence anywhere on the incidence of adverse selection, although there was some evidence of its potential impact when, for a period in the U.K., life insurance business in connection with mortgages was offered without any underwriting questions asked. If a highly accurate genetic test were to become available which provided predictive information about the likelihood of contracting a common and serious illness before retirement age, this would be extremely relevant from a term life insurance perspective. Of course, it will also be extremely relevant whether the availability of such test results is associated with the emergence of improved treatment possibilities or a clearer understanding of lifestyle or dietary changes which could offset the increased level of risk.

2.4.4 Currently, there are very few actuarially relevant genetic tests which could provide predictive information of this quality. Even if such tests do emerge, they are only likely to have an impact in terms of adverse selection if

https://doi.org/10.1017/S1357321700004360 Published online by Cambridge University Press

traditional underwriting practices are disrupted. In particular, the level of exposure to adverse selection will be increased if individual policyholders are not required to reveal their true risk status, derived from genetic tests or other predictive means, before entering into an insurance contract. This is the case in the U.K., where the industry has agreed a moratorium on the use of information derived from molecular genetic tests (ABI, 1999). Similar restrictions have been adopted in other countries (Chuffart, 1996; McGleenan, 2001).

2.4.5 Adverse selection is, in itself, a feature of any insurance market, and pricing implicitly takes it into account. However, significant changes in the extent of adverse selection may cause unexpected changes in insurance companies' experience. The impact of adverse selective behaviour is contingent upon the financial strength of the company affected and the regulatory safeguards which are available to prevent or punish any actions which might be deemed to be fraudulent or against the public interest.

2.4.6 There is, in practice, considerable disagreement about whether or not advances in genetic technology will ever be accompanied by adverse selection effects sufficient to cause significant financial problems to insurance companies.

## 3. Actuarial Research into Genetics and Insurance

### 3.1 *Questions to be Answered*

3.1.1 Actuarial research into genetics and insurance was stimulated in 1996, with the joint meeting of the Faculty and Institute of Actuaries and the Royal Society of London (Le Grys, 1997; Macdonald, 1997; Ross, 1997; Wilkie, 1997). Public discussion between the insurance industry and other interested parties (including the House of Commons Science and Technology Committee) had revealed only irreconcilable points of view: to most insurers it seemed an obvious point of principle that applicants had to disclose all relevant information; to others, it was equally obvious that people should not be penalised because of their genes. The surprising fact is that financial implications of genetics were discussed so much with hardly a shred of quantitative evidence either way. The aim of the bulk of actuarial research in this area has been to begin to supply that evidence.

3.1.2 Genetic information could have financial implications for individuals or for insurers:

(a) Where insurers may use genetic information in underwriting, then people with adverse family medical histories or genetic test results could be charged extra premiums. How large might these be, and would they lead to the creation of a 'genetic underclass'?

(b) Where insurers may not use genetic information, then adverse selection may result, leading to increased premiums for everybody. These increases give a useful measure of the 'cost' of adverse selection; how great might they be?

https://doi.org/10.1017/S1357321700004360 Published online by Cambridge University Press

3.1.3 At first the focus was entirely on genetic test results, defined narrowly in terms of direct examination of DNA or chromosomes, but it was hard to understand the logic of regarding as different tests for gene products — such as ultrasound scans for kidney cysts in adult polycystic kidney disease (APKD) — or family histories of Mendelian disorders (see Zimmern (2001) for a discussion of what 'genetic information' might mean). The Human Genetics Commission (HGC, 2000) made it clear that it took a broad view of genetic information. In assessing possible costs of adverse selection, it is necessary, therefore, to consider the various forms that a ban or moratorium could take, disallowing all genetic test results, or adverse test results only, or family medical history as well.

3.1.4 Even if a ban or moratorium remains in place on using genetic information for premium rating, in the U.K. and in other countries around the world, there would still be a need for actuarial research into the impact of genetic information on insurance, in order to understand what risks are being accepted into the insurance pool.

### 3.2 *Actuarial Models*

3.2.1 Broadly speaking, the genetic disorders that may be relevant for insurance fall into two groups, for which different modelling approaches may be needed:

(a) Dominant single-gene disorders result from a mutation in just one gene, which then encodes a harmful variation of the gene product, or one that is unable to perform an essential function (such as repairing damage to DNA that, unchecked, might lead to cancer). Inheriting just one copy of the mutation from either parent exposes the carrier to the risk of disease. Disorders of interest to insurers, which are disorders in which onset is delayed to adult ages, are rare. Therefore, the probability of both parents carrying mutations in the same gene, or of one parent carrying two mutations, is negligible, and so the probability that a child of a carrier will also be a carrier is about 50%. Knowledge of these disorders is not new, they are precisely those that have long been observed to 'run in families', and they have been studied for a long time on the basis of family medical history.

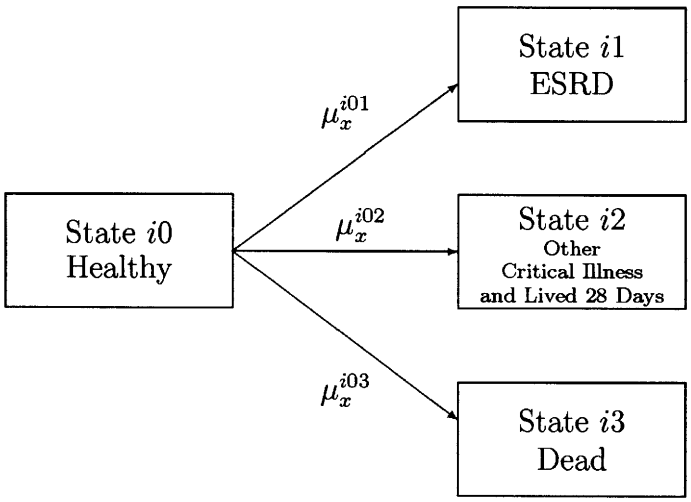
(b) Multifactorial disorders result from combinations of variants of many genes ('polymorphisms') together with environment and lifestyle. Polymorphisms in each individual gene do not necessarily confer a large increase in risk, so they might be common in the population; for the same reason it might be extremely difficult to measure the contributions of such polymorphisms to the risk of disease. The genetic component of common diseases, such as heart disease and many cancers, is probably multifactorial in nature; we all have some multifactorial 'disorders'.

https://doi.org/10.1017/S1357321700004360 Published online by Cambridge University Press

We have omitted other genetic disorders — recessive, X-linked, chromosomal and somatic — as these have not yet been the subject of actuarial models, but the approach we describe below should be equally applicable.

3.2.2 To a first approximation, dominant single-gene disorders divide the population into two distinct genotypes; mutation carriers and non-carriers. If the disorder has no cause except the gene mutation, non-carriers are not at risk at all. The discrete nature of the resulting risk groups is well-suited to the use of multiple-state models. As an example, consider APKD and critical illness insurance. Figure 1, based on Gutiérrez & Macdonald (2001), shows a model in which a healthy person can progress to end-stage renal disease (kidney failure) because of APKD, which will result in a critical illness insurance claim; or can claim for any other reason, including end-stage renal disease not resulting from APKD; or can die. There are two sub-populations (mutation non-carriers and carriers) labelled $i = 1$ and $i = 2$ respectively, and the difference between them is in the intensities of onset of end-stage renal disease. $\mu_x^{101} = 0$ because APKD is entirely genetic, while $\mu_x^{201}$ has to be estimated from epidemiological studies.

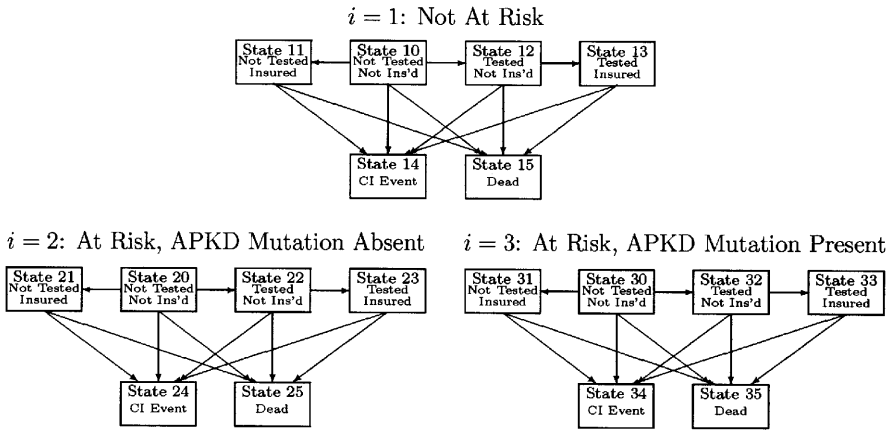
3.2.3 When examined in more detail, it might not be enough to treat all mutation carriers as alike. Some disorders may be caused by mutations in any of several genes (APKD, breast/ovarian cancer and early onset




Source: Gutiérrez & Macdonald (2001)

Figure 1. A model for APKD and critical illness insurance, in the $i$th of several subpopulations representing genotype

https://doi.org/10.1017/S1357321700004360 Published online by Cambridge University Press




Source: Gutiérrez & Macdonald (2001)

Figure 2. A Markov model of critical illness insurance allowing for family history of APKD and genetic testing

Alzheimer's disease, for example); this is easily dealt with by defining separate sub-populations for carriers of mutations in each gene, usually ignoring multiple mutations because of their rarity. More serious, however, is heterogeneity of mutations of a single gene (Weatherall, 2000). BRCA1, a breast cancer gene, has over a thousand known mutations; Presenilin-1 (PS1), an early onset Alzheimer's disease gene, has over seventy. Little is known yet about the different risks conferred by different mutations, and, in fact, most epidemiological studies to date assume homogeneity, but as we learn more it might be necessary to distinguish between mutations.

3.2.4 To estimate the costs of adverse selection, we must extend the models to the purchase of insurance. An example of the simplest such model is shown in Figure 2, also from Gutiérrez & Macdonald (2001). Here there are three sub-populations:

(a) persons with no family medical history, not at risk of APKD ($i = 1$);
(b) persons at risk of APKD because they have a family medical history, but who do not, in fact, have a mutation ($i = 2$); and
(c) persons at risk of APKD because they have a family medical history, and who do have a mutation ($i = 3$).

3.2.5 The members of the $i$th sub-population start in state $i0$, in which they have not had a genetic test, nor have they bought insurance. From there they can simply buy insurance (move to state $i1$) or have a genetic test (move to state $i2$), and then perhaps buy insurance (state $i3$). At any time

https://doi.org/10.1017/S1357321700004360 Published online by Cambridge University Press

they can die or suffer an event which triggers a critical illness insurance claim. This model captures all the features we need:

(a) The size of the insurance market is determined by the rate at which insurance is bought, which can depend on age if that information is available.
(b) The incidence of genetic testing (screening in the whole population, or testing of at-risk persons only) is represented by the rate of genetic testing in each sub-population.
(c) The mutation frequency is represented by the proportions in each sub-population. For example, APKD mutations occur in about 1 per 1,000 of the population, so at younger ages 0.1% would be at-risk carriers, 0.1% at-risk non-carriers and 99.8% not at risk. At older ages the mutation frequencies among healthy persons can be found by solving the Kolmogorov forward equations for the occupancy probabilities.
(d) The behaviour of 'adverse selectors' — both the probability that they buy insurance and the amount that they buy — is represented.
(e) Each state in the model can be assigned to the appropriate underwriting class, depending on what information the insurer is allowed to use, and appropriate premiums can be calculated within each class.
(f) The model can easily be extended to allow for mutations in different genes, or other events such as lapsing insurance or buying more insurance (Pritchard, 1997; Subramanian *et al.,* 2000, for example).
(g) Variations of the model can handle life, critical illness, income protection or long-term care insurance, or annuities (Tan, 1997, for example). Critical illness insurance is the easiest to model, since we need only rates of onset of the disorder. To model life insurance we need survival rates after onset, which often depend on duration as well as on age. There is little or no useful genetic epidemiology relating to disability levels as opposed to survival and death.

3.2.6 The obvious difficulty is in choosing the intensities, especially those relating to behaviour. Research on attitudes to risk would be very helpful here. It is clear that, for rates of onset and progression of a disorder, we depend entirely on results from genetic epidemiology, since actuaries are unlikely to be able to collect genetic data directly; we comment on that in Section 3.4.

3.2.7 The basic tools for handling multiple-state models are differential equations: Kolmogorov's forward equations for occupancy probabilities; and Thiele's equations for prospective reserves (Hoem, 1988). By solving Thiele's equations with suitable choices of premium rates and benefits, the expected losses with and without adverse selection are found, and the excess costs arising because of adverse selection are translated into an increased rate of premium. Both Kolmogorov's and Thiele's equations are systems of linear ordinary differential equations, which can be solved

https://doi.org/10.1017/S1357321700004360 Published online by Cambridge University Press

numerically. Sometimes, in simple cases (such as the model in Figure 1, for example) approximations that can be handled by a spreadsheet are available, but linear ordinary differential equations should, in any case, be tractable.

3.2.8 Multifactorial disorders present a more continuous spectrum of risk, and multiple-state models will offer, at best, a discrete approximation of the population risk. However, the study of multifactorial disorders is much less advanced than that of single-gene disorders, and it may be some time before there are any usable risk estimates. To date actuarial models have furnished only broad conclusions based on 'top-down' models of multifactorial disorders as an entire class (see Section 3.3).

### 3.3 *Top-Down or Bottom-Up?*

3.3.1 Multiple-state models have been applied to genetic problems in two ways:

(a) In what may be called a 'top-down' approach, extremely adverse assumptions are made about the risk associated with some mutation or class of mutations, the incidence of genetic testing and the extent of adverse selection. If it can then be shown that the cost of adverse selection is modest, that can be given its due weight in setting policy on the use of genetic information. In Macdonald (1997, 1999) the entire class of multifactorial disorders was considered, and in Macdonald (2001) the entire class of single-gene disorders was treated similarly. The great advantage of a 'top-down' model is that we do not need detailed epidemiology of individual disorders, just enough information to be sure that our assumptions are extreme. The disadvantage is that they are limited to seeking 'null results', in which extreme assumptions have trivial consequences. If they suggest that adverse selection could bring significant costs, we have to turn to a 'bottom-up' approach.

(b) In a 'bottom-up' approach, we model each genetic disorder as well as the available epidemiology permits, and obtain a total cost of adverse selection by aggregating the individual costs. Clearly this is much more demanding than a 'top-down' approach, but in the long run it is the only convincing approach. Its chief drawback at the moment is that genetic epidemiology is scarce, even in respect of some major disorders. The first 'bottom-up' models were those of Lemaire *et al.* (2000) and Subramanian *et al.* (2000), dealing with breast/ovarian cancer and life insurance, and Smith (1998), dealing with Huntington's disease and life insurance. Others are Macdonald & Pritchard (2000, 2001) and Warren *et al.* (1999) on Alzheimer's disease and long-term care, Macdonald, Waters & Wekwete (2003a, 2003b) on breast/ovarian cancer and critical illness insurance, and Gutiérrez & Macdonald (2001) on APKD and critical illness insurance.

https://doi.org/10.1017/S1357321700004360 Published online by Cambridge University Press

3.4 *Genetic Epidemiology*

3.4.1 The key to useful actuarial models of individual disorders is genetic epidemiology. The necessary data may include:

(a) rates of onset of the disorder, by genotype;
(b) rates of progression of the disorder, including mortality; and
(c) population frequencies of each relevant mutation (note that these are necessary only for modelling the cost of adverse selection or for estimating premiums based on family medical history information).

3.4.2 The (apparently) simpler single-gene disorders, such as Huntington's disease, suggest a simple model for an epidemiological study. Identify a population of mutation carriers, observe when they suffer onset of the disorder, and carry out a classical survival analysis of the results. Implicit in this scheme, however, are some assumptions that hold rather rarely, since Huntington's disease is not typical of all single-gene disorders. In particular:

(a) Identifying mutation carriers is typically difficult, especially because the prevalence of genetic testing tends to be low if there is no effective treatment for the disorder.
(b) If the disorder has common causes other than the mutation being considered, onset does not identify mutation carriers. A similar problem arises if mutations in one of several genes can cause the disorder. Mutation carriers may also be missed if, as is most common, penetrance is less than 100% (meaning that some mutation carriers will never get the disorder).
(c) If the penetrances of different mutations in a single gene are variable, the more severe variants are more likely to come to the attention of researchers, as are large families with unusually large numbers of affected members. This leads to 'ascertainment bias', overstating the risks associated with a mutation and understating mutation frequencies.

3.4.3 See Macdonald (2003) for a more detailed discussion of these problems. Ascertainment bias makes it difficult to know what risks to assume in respect of an insured population, which may not be typical of the population as a whole or of any population selected for a genetic study. On the one hand, it is plausible that an applicant who has detailed knowledge of his or her own genetic risk must be a member of those groups studied by geneticists, precisely because they have acquired that knowledge. On the other hand, if it becomes more common for people to have genetic information, prospective population-based studies may be necessary in future.

3.4.4 It is important to realise that genetic epidemiology yields results years or even decades after the disease-causing genes have been discovered in

https://doi.org/10.1017/S1357321700004360 Published online by Cambridge University Press

the laboratory. This sometimes leads to confusion in the media about the consequences of the discoveries. Since we are now just at the stage of identifying genes, it should be no surprise that epidemiology is sparse, at least compared with the demanding requirements of actuarial models. Moreover, most studies address medical questions, and they follow the reporting conventions of medical statistics. Some specific problems are:

(a) Study populations are often small, so only a few figures are reported (median survival times, lifetime penetrances and so on).
(b) When age-related probabilities are given, they are usually in the form of graphs, often very small.
(c) Sometimes researchers are very helpful in supplying missing or background information; sometimes they are not.

3.4.5 It may be possible, in some circumstances, to estimate rates of onset from data in the medical literature. This can be the case if it is usual to publish pedigrees when reporting the discovery of novel mutations in a particular gene. In this way, Gui & Macdonald (2002) estimated rates of onset of early onset Alzheimer's disease associated with Presenilin-1 (PS1) mutations, but this opportunity was quite unusual.

### 3.5 *Examples of Conclusions*

Here we list briefly some of the conclusions reached in actuarial research to date. We refer readers to the original sources for full details.

(a) Multifactorial disorders are unlikely to be of much significance for life insurance. Even under quite extreme assumptions, premium increases caused by adverse selection are unlikely to be significant (Macdonald, 1997, 1999). The more serious aspect of adverse selection is any tendency to take out unusually large amounts of insurance (Macdonald, 1997, 1999; Subramanian *et al.*, 2000).
(b) In a large life insurance market, even a ban on using genetic test results for severe single-gene disorders would be unlikely to lead to significant adverse selection costs, provided excessive sums assured were controlled. A ban on using family medical histories of Mendelian disorders would lead to modest premium increases just because persons at risk would be charged standard premiums; adverse selection in addition would not have a serious effect (Macdonald, 2001).
(c) A ban on using family medical histories of breast/ovarian cancer would be much more significant than a ban on using the results of tests for the mutations in the BRCA1 and BRCA2 genes (Subramanian *et al.*, 2000).
(d) Carriers of BRCA1 mutations, and nearly all carriers of BRCA2 mutations, would face extremely high extra premiums for critical illness insurance. However, because mutations in these genes account for only about 5% of breast cancer, ratings for family medical history are very variable, depending on age, policy term and details of the affected and

https://doi.org/10.1017/S1357321700004360 Published online by Cambridge University Press

unaffected relatives. Significant adverse selection costs could arise in small critical illness insurance markets (Macdonald, Waters & Wekwete, 2003a, 2003b).

(e) Persons at risk of Huntington's disease may be offered life insurance, for certain ages and over certain terms, at lower cost than previously thought possible (Smith, 1998).

(f) Persons known to carry a mutation leading to APKD (on the basis of an ultrasound scan) would be charged very high extra premiums for critical illness insurance (of the order of +400%), and would, effectively, be uninsurable except possibly at high ages and for short terms. However, such mutations are rare enough that adverse selection costs from this cause alone would be negligible under a moratorium on genetic test results. A ban on using family medical histories would lead to higher standard rates of premium, just because persons at higher risk were now admitted to the standard rates risk pool, even if adverse selection were absent. This model suggested that such premium increases could reach about 1% in a small critical illness insurance market, which seems quite high for just one single-gene disorder with a population frequency of about 1 per 1,000. However, it would be necessary to complete studies of other single-gene disorders before reaching conclusions about the potential for premium increases for this reason or because of further adverse selection. (Gutiérrez & Macdonald, 2001).

(g) Based on published pedigrees, the penetrance of mutations in the Presenilin-1 (PS1) genes (to onset of early onset Alzheimer's disease) exceeds 50% by the late 40s (Gui & Macdonald, 2002).

## 4. The Insurance Industry Response to the GAIC Process

### 4.1 *The GAIC Process*

4.1.1 As mentioned earlier, it was proposed at the end of 1998 in the U.K. that insurers should use genetic test results for underwriting purposes only if those tests had been approved by the Genetics and Insurance Committee (GAIC). That committee prescribed a series of questions to which detailed answers were required as a basis of submission for approval. A separate report was required for each test and for each type of insurance, combining the input from geneticists and actuaries to demonstrate that the test met the GAIC evaluation criteria. The ABI took the lead in developing submissions to GAIC. Since there was little or no peer-reviewed research which could be used directly in support of these submissions, teams of actuaries from insurance and reinsurance companies were assembled to develop the actuarial and statistical arguments in support of the submissions. It is not clear whether the detailed submissions which they produced will be made public, although a full actuarial critique of the first submission — on

https://doi.org/10.1017/S1357321700004360 Published online by Cambridge University Press

the use of predictive tests for Huntington's disease in relation to life insurance applications — is in the public domain (Wilkie, 2000). In this section we describe briefly the work that was done by actuaries, working on behalf of the ABI, in preparing the submissions; the underlying content is not the work of the authors of the current paper.

4.1.2 Each submission contained sections that covered the accuracy and reliability of the test in a clinical setting. These included a detailed description of the genetic test itself, the genetic basis and the clinical impact of the medical condition(s) to which the test relates, and specific reference to the factors which influence the associated morbidity and life expectancy.

4.1.3 The actuarial sections of the submission required evidence to demonstrate the 'actuarial relevance' of the genetic test to the type of insurance covered by the application. 'Actuarial relevance' was not formally defined, and the appropriateness of this term was challenged by the actuarial profession when the GAIC consulted on the application requirements (the profession suggested that 'actuarial significance' might be a more appropriate term, since almost any information could be held to be relevant, but the issue of interest to GAIC was whether it was significant for underwriting and pricing purposes). The intention was to require demonstration that the results of the test would be robust and would have clear implications for actuarial assessment of the proper premium rate to charge.

4.1.4 The evaluation criterion set by the GAIC at that time was that the additional risk for at least one relevant age/term combination should be at least 50% in the case of life insurance and 25% in the case of critical illness, income protection or long-term care insurance. The aim of the ABI exercise was to determine whether the test was, in principle, relevant for taking underwriting decisions, but not to produce results which would necessarily be appropriate for producing guidance on quantifying the extra risk when making those decisions. No guidance was provided by GAIC as to the degree of technical precision required in this analysis, and there was no requirement to recognise the prevalence of the mutation in the population or the possible commercial impact of a ban on the use of the predictive test results.

4.1.5 In fact, the GAIC process did not address at all how insurers might use predictive genetic test results to define underwriting guidelines and set risk premiums, should they be permitted to do so. This could be seen as a weakness in the GAIC process compared to the standards of relevance of information required under the Sex Discrimination Act or the Disability Discrimination Act. The intention in setting the 50% and 25% values seems to have been to prohibit insurers from using test results which were not particularly significant anyway.

4.1.6 There was no precedent for the GAIC process for approval of the use of test results anywhere in the world. Neither had the industry previously

https://doi.org/10.1017/S1357321700004360 Published online by Cambridge University Press

been required to publish papers or statistical evidence to justify its use of other medical evidence in its underwriting. The recognised approach had been for reinsurance companies to carry out analyses of increased risk arising from a wide range of medical conditions. These were used by the reinsurers to compile guidelines for their underwriting manuals, but these guidelines are practical commercial material, and it has not been industry practice to publish any of these guidelines, or the basis for them, or to subject them to public or professional review. It is unusual for direct writers of insurance to do any such work of their own.

4.1.7 The ABI made submissions on behalf of the industry for five of the tests from the original set of ten, which it had identified provisionally as relevant and reliable for insurance underwriting. These were for Huntington's disease, breast cancer (BRCA1 and BRCA2) and early onset Alzheimer's disease (APP and Presenilin-1 (PS1)). The others were not submitted as part of the first batch for various reasons (these were the tests for familial adenomatous polyposis, hereditary motor and sensory neuropathy, myotonic dystrophy, multiple endocrine neoplasia and the Presenilin-2 (PS2) test for early onset Alzheimer's disease). The ABI genetics adviser compiled the content of the genetics sections and provided numerous references for the research material needed to support the actuarial content. The actuarial resource was provided by a number of reinsurers and direct-writing insurers, and no attempt was made to obtain academic input. The work was carried out by small 'virtual' teams, working concurrently to a very tight deadline.

4.1.8 There were no statistics available which would indicate directly the probability of a person with an adverse genetic test result of any kind making a claim under a particular type of insurance policy, since no such research had been carried out. It was necessary to use a combination of the most relevant published data from medical and epidemiological research, together with expert opinion. Areas of uncertainty within the results had to be dealt with by setting the assumptions with a margin in whichever direction would reduce the calculated additional risk, i.e. in favour of the client.

4.1.9 There was little formally published actuarial material available describing any recognised methodology for this type of work, which had not before been required for any formal approval process. There was a slight exception in the case of some relevant modelling work which had already been carried out by a reinsurer on the mortality experience of asymptomatic individuals who had tested positive for the Huntington's disease mutation. It was not to the depth of peer-reviewed academic papers, but it had been made public within the industry. This work (Smith, 1998) was used as the foundation for the ABI submission to the GAIC on Huntington's disease and life insurance, and was developed further for the other types of insurance; submissions for other tests then followed a similar approach.

https://doi.org/10.1017/S1357321700004360 Published online by Cambridge University Press

4.2 *The Modelling Process*

4.2.1 The general approach was:

(a) to construct a model for calculating the probability of a claim by an asymptomatic individual with an adverse test result;
(b) to calculate the probability of a claim based on a standard insured population; and
(c) to calculate the level percentage extra mortality/morbidity which a standard individual would have to suffer over the term of the policy in order for the probability of a claim to equal the probability of a claim for an asymptomatic person with an adverse test result. In the case of income protection and long-term care insurance, the additional risk was determined via the additional cost rather than the additional probability of claim, as it was necessary to introduce the concept of the length of claim.

4.2.2 The actuarial requirement for modelling the experience was to have values for age-dependent incidence rates and age and duration-based rates of transition to the various possible states, some of which give rise to the events relevant to insurance; these should be based on large populations. Unfortunately, not only were there no sources providing the insurance data required, as indicated earlier, but the published research papers did not provide any detailed data. Their results were presented in various ways, according to the question which they were attempting to answer, but the data tended to be in the form of mean ages of onset or death and sometimes mean durations from onset to death, with some standard deviations provided; and the volume of data analysed was, in some cases, quite small.

4.2.3 The results were to be used for a purpose which differed from that for which the studies had been set up, so care was needed to ensure that it could be used without inadvertently introducing any bias or ambiguity, paying particular attention to the selection of the lives to be studied. For example, a retrospective study of those who had been identified as carrying the mutation only after they had developed the medical condition would not reflect the penetrance of the disease in mutation-carriers. If these results were used just as reported, the additional risk would be overstated.

4.2.4 The nature of the model varied slightly between applications, not only according to the type of insurance, but also as a result of the differences in the progress of the various diseases and the nature of the research data available. The research was based on onset, diagnosis and death rather than insurance claim criteria, so where there were no useful data it was necessary to rely on expert opinion, for example at what point a claim for income protection insurance would be triggered and for how long it would be paid. Table 1 shows the nature of the assumptions made.

4.2.5 The basic structure was a traditional multiple decrement table with

https://doi.org/10.1017/S1357321700004360 Published online by Cambridge University Press

Table 1. Modelling assumptions for progression from onset to insurance claim

| | Huntington's disease | Early onset Alzheimer's disease | Breast cancer |
|---|---|---|---|
| Onset | Normal distribution | Normal distribution | Based on age-related penetrance data |
| Death for life claim | Normal distribution following onset | Fixed duration from onset | Fixed % p.a. compound |
| Critical illness claim | Total permanent disability: expert opinion for fixed duration from onset | Diagnosis | Diagnosis |
| Income protection claim | Expert opinion for incidence; fixed period for length of claim | Expert opinion for incidence; fixed period for length of claim | Expert opinion for incidence; fixed period for length of claim |
| Long-term care claim | Expert opinion for incidence; very high mortality rates once in payment | Expert opinion for incidence; very high mortality rates once in payment | Not submitted |

annual decrements and with some simplifications for convenience, i.e. ignoring some types of decrement where this would lighten the results for additional risk. Greater sophistication was not thought to be necessary, given the aim of the investigation and the wide margin by which the results passed the required threshold. Later this proved to be a point on which opinions differed.

4.2.6 In the case of Huntington's disease and early onset Alzheimer's disease, the onset of the disease was assumed to follow a normal distribution over the age ranges under consideration, and this was shown to be a reasonable fit to the data. In the case of breast cancer, the rates of onset were derived from the age-related penetrance data in the research.

4.2.7 For life insurance, the post-onset mortality was modelled differently for each of the three conditions, based on the typical pattern of progress of each condition. For critical illness insurance the diagnosis was assumed to trigger a claim immediately in the case of breast cancer and early onset Alzheimer's disease, but Huntington's disease sufferers were assumed to become eligible only when they became totally and permanently disabled from carrying out their own occupations, a criterion which is not addressed in the research. For this, and for income protection and long-term care insurance, it was necessary to rely on expert opinion to judge at what point a claim would be triggered and for how long it would be paid. For income protection insurance, the length of a claim was assumed to be a fixed number

https://doi.org/10.1017/S1357321700004360 Published online by Cambridge University Press

of years, but varying according to the condition. A deferred period of 26 weeks was used as the central assumption of the type of policy. For long-term care insurance claims in payment a very high rate of mortality was assumed.

4.2.8 The methodology required standard tables for basic insurance experience, which are not available for all types of insurance. Calculation of the probability of a death claim for a standard life was based on published actuarial tables for mortality under U.K. life insurance contracts (AM92/AF92 from CMI Report Number 17 (CMI, 1999)), but in the case of Huntington's disease a Dutch mortality table was used, as the research which was used as the main source had been carried out in the Netherlands.

4.2.9 For income protection insurance claims, incidence rates published in CMI Report Number 12 (CMI, 1991) were used, adjusted as indicated in CMI Report Number 18 (CMI, 2000).

4.2.10 For critical illness insurance, there is not yet a published standard actuarial table, so the assumption of standard insured experience was based on Dinani *et al.* (2000), which uses population data as one key source. When pricing critical illness insurance, it is usual to make some allowance for the expected favourable differential between the experience of insured lives and that of the general population, but this was not incorporated here; the precise adjustment to make would be a matter of judgement, and to assume a harsher level for standard experience will tend to understate the results for additional risk.

4.2.11 There is no published standard actuarial table for long-term care insurance either; the source used was Dullaway & Elliot (1998), which uses population data adjusted in line with insurance definitions of disability.

4.2.12 Some of the formulae used in the submissions are reproduced below. These are not the work of the authors of this paper:

(a) Calculation of the additional mortality risk was as follows in the case of Huntington's disease:

While unaffected, the lives are assumed to have normal mortality $q_x$.

Once affected, the lives are assumed to have an increased mortality rate which is independent of age $q_t^a$.

The substandard survival factor ${}_nP_x^{ssd}$ is the probability that someone aged $x$ with the abnormal gene will survive to age $x+n$, calculated from the model based on research data.

Similarly, the survival factor for a normal life ${}_nP_x$ is the probability that someone aged $x$ will survive to age $x+n$. This can be calculated as:

$$ {}_nP_x = \prod_{t=0}^{n-1}(1-q_{x+t}) = (1-q_x)*(1-q_{x+1})*\ldots*(1-q_{x+n-1}). $$

So, if ${}_nP_x(\alpha_{x,n})$ is the survival factor for a life with average extra

https://doi.org/10.1017/S1357321700004360 Published online by Cambridge University Press

mortality of $\alpha_{x,n}$ from age $x$ until $x+n$, then the required average extra mortality risk $\alpha_{x,n}$ over the next $n$ years for someone with the defective gene and aged $x$ is calculated iteratively by equating the calculated values of ${}_nP_x^{ssd}$ to:

$$ {}_nP_x(\alpha_{x,n}) = \prod_{t=0}^{n-1}[1-(1+\alpha_{x,n})q_{x+t}]. $$

The calculations for critical illness insurance were similar.

(b) For income protection insurance, the calculation of additional morbidity risk was calculated as follows:
${}_nM_x^h$ is the expected cost of claims for a pre-symptomatic individual with the abnormal gene from age $x$ until age $x+n$, calculated from the model based on research data.
${}_nM_x$ is the expected cost of claims for a standard life from age $x$ until age $x+n$.
This is calculated as:

$$ {}_nM_x = \sum_{t=0}^{n-1} v^{t+1/2} * (l_{x+t}/l_x) * i_{x+t} * a_{x+t+1/2} $$

where:
$i_{x+t}$ is the income protection insurance inception rate for a life aged $x+t$;
$l_{x+t}$ is the number of lives in force at age $x+t$;
$a_{x+t+1/2}$ is the present value of the incapacity benefit, at the time of onset of incapacity, payable annually in advance following the deferred period until the claim ceases; and
$v$ is the discount factor based on an interest rate of 5% p.a.

So, ${}_nM_x(\alpha_{x,n})$, the expected cost of claims for a life with average additional morbidity of $\alpha_{x,n}$ from age $x$ until age $x+n$ is calculated as follows:

$$ {}_nM_x(\alpha_{x,n}) = \sum_{t=0}^{n-1} v^{t+1/2} * (l_{x+t}/l_x) * i_{x+t} * (1+\alpha_{x,n}) * a_{x+t+1/2}. $$

The required value of $\alpha_{x,n}$ was calculated iteratively by equating the calculated values of ${}_nM_x^h$ to ${}_nM_x(\alpha_{x,n})$.

The calculations for long-term care insurance were similar.

https://doi.org/10.1017/S1357321700004360 Published online by Cambridge University Press

4.3 *Results*

4.3.1 In all of the 18 submissions made, the results included values well in excess of the threshold required by GAIC at the time, even with many margins included, which tended to reduce the measure of extra risk. In general, the results for life insurance and critical illness insurance showed extremely high additional risk over a range of combinations of age and term, in many cases showing the risk to be increased by factors of between two and ten. For income protection insurance the results were not quite so dramatic. The modelling assumptions and simplifications may have diluted the true results for the additional risk, but the length of claim assumed was generally quite short, owing to the nature of the illnesses, so the additional cost may well be lower than for the lump sum benefits. For long-term care insurance the results exceeded the threshold significantly in many cases.

Table 2. Level percentage extra mortality for asymptomatic individual tested positive for Huntington's disease mutation; male with affected father

| | Term (years) | | |
|---|---|---|---|
| Age | 10 | 20 | 30 |
| 20 | 254 | 980 | 1,297 |
| 30 | 379 | 832 | 809 |
| 40 | 205 | 402 | 375 |
| 50 | 67 | 145 | Not calculated |

4.3.2 When presenting the results, it was necessary to identify the potential shortcomings of the model and the data used. These included the paucity of data in the research; the use of non-U.K. data; the choice of lives for the studies (focusing on families with very strong family medical histories); and no standard published tables for critical illness insurance experience. Sensitivity tests were carried out on many of the assumptions made, such as the mean and variance of the normal distributions used and the post-onset mortality rates.

4.3.3 GAIC required the submissions to be reviewed by relevant experts, but did not appear to demand that the work should be carried out to the full peer-reviewed standards required for publication in scientific journals. For the initial Huntington's disease life insurance submission, the actuarial section was reviewed by Professor David Wilkie (Wilkie, 2000). His thorough approach to the task of review identified many weaknesses in the detail of the work from an academic point of view, covering both the general method applied and also some of the specific aspects relating purely to Huntington's disease. These included the use of the normal distribution and the assumption that intensities of death depended only on duration since onset and not also on age. In Wilkie's opinion Smith (1998) was, in principle, on the right lines, although not up to peer-reviewed standards. Wilkie commented that,

https://doi.org/10.1017/S1357321700004360 Published online by Cambridge University Press

without doing any detailed work, it was obvious that the GAIC criterion would be met; when the work was revised by him the results were not very different from those in the submission, and in his opinion the conclusion was the same.

4.3.4 In October 2000 GAIC approved the use of test results for Huntington's disease in the context of life insurance applications. The other submissions were still awaiting assessment at the time GAIC was temporarily disbanded in 2001. It is likely that the content will need to be revisited and modified before being considered by the new GAIC, but at the time of writing GAIC had not yet made known its requirements.

4.3.5 It seems likely that GAIC, even had it not been re-formed, would have had to develop further its criteria for approval. Huntington's disease had arguably been a uniquely simple case. Later approvals would undoubtedly have been more complex. With BRCA1 and BRCA2, for example, should the actuarial models consider the impact of prophylactic mastectomy and oophorectomy?

4.3.6 In public statements, the U.K. actuarial profession has been generally supportive of the GAIC process, as it points to the need for proper research and analysis rather than jumping to conclusions. Approval is a necessary condition for insurance use; opinion may vary on whether it is sufficient. The flexibility of the GAIC process, allied to adaptable industry self-regulation agreed by government, might generally be seen as preferable to the rigidity of legislation at this early stage in the development of understanding the interaction between genetics and insurance.

4.3.7 In recent years, public opinion, in general, has shifted towards expectations of greater transparency and a requirement for stronger evidence relating to matters that affect individuals. The insurance industry will need to be responsive to these demands, and future practice in the field of underwriting will no doubt have to recognise this. There may well be a need for a greater number of actuaries to get involved in this work in the future, and for relevant research to be carried out at an appropriately rigorous level.

## 5. The Moratorium and Beyond

### 5.1 *Introduction*

5.1.1 The first moratorium by the insurance industry on the use of predictive genetic test results was introduced in the autumn of 1998 for a period of three years. When it came up for review in 2001, it was in the wake of a critical report from the House of Commons Select Committee on Science and Technology (HCSTC, 2001), and a report from the HGC which recommended that the Government should introduce legislation to enforce a continuation of the moratorium for a further three years (HGC, 2002).

https://doi.org/10.1017/S1357321700004360 Published online by Cambridge University Press

5.1.2 In the event, the insurance industry negotiated a new five-year moratorium with the Government (see ¶1.16) and the Government decided to continue with the GAIC process, albeit with a majority of new members and a somewhat broader remit. The re-formed GAIC met for the first time in September 2002, and now includes two actuaries — Professor David Wilkie and David Paul (one of the authors). The September meeting initiated a review of the GAIC criteria, responding to requests to do so by the Government and by the HGC. While there is a review under way the previous ABI applications are effectively stood down, and so different applications may be brought back, in a different order.

5.1.3 The developments over the past three years, and the many debates and discussions which have taken place, in the HGC, at the meetings of the UKFGI and elsewhere, place the issues now in a somewhat different context from hitherto. In this section we analyse some of the arguments which are made and explore where this might take us in terms of a reconciliation between the insurance industry instincts and a coherent social policy perspective.

### 5.2 *Insurers' Lines of Reasoning*

5.2.1 Insurers would like to be able to use genetic tests. Their underlying reasons seem to be:

(a) *status quo:* the practical convenience of traditional underwriting freedoms not being eroded; undermining the right to underwrite in this area could prove to be the thin end of a wedge;
(b) *protection against options:* the fear of individual applicants taking a financially advantaged position at the expense of an insurer in the light of information asymmetry;
(c) *damage to competitiveness:* the fear that a single insurer would be put at a financial disadvantage (against its competitors) if it were burdened with a disproportionate share of higher risks;
(d) *ideological:* an ideological stance that private insurance should be allowed to match price to risk in an unfettered way (and that it is not the role of private markets to administer a cross-subsidy imposed on them);
(e) *fairness:* giving special treatment to people at risk of genetic conditions would be unfair to other prospective policyholders who have the same level of extra risk, but arising from a non-genetic impairment or because they are, say, 20 years older;
(f) *operability:* the concern that a market sector could be rendered inoperable if higher risk customers were able to secure cover which pushed up the 'standard' premium to such an extent that low and medium risk customers abstained from purchasing policies; and
(g) *future uncertainty:* whilst it may be possible to ignore genetic information at the moment, it is too early to say how important this may become for the insurance industry in the future, and it is wise to take a cautious approach at this stage.

https://doi.org/10.1017/S1357321700004360 Published online by Cambridge University Press

5.2.2 However, these proved not to be very easy arguments for insurers to advance. It is natural for public sentiment to be swayed towards the individual (and against large financial institutions). It can be argued that small numbers of applicants for insurance with a few specific rare monogenic conditions do not realistically represent any serious threat to an insurer's financial position. Clearly, from a public policy viewpoint there is a strong appeal in insisting that disadvantaged lives should benefit from what can be labelled 'equal' treatment.

5.2.3 In fact, during the 1997-2001 period, insurers were less and less successful in deploying arguments in favour of their right to use genetic tests. During this period no large group of the population emerged with predictive genetic test results that conclusively set them apart. Without such large groups to point to in support of the arguments, it became more difficult to say either that markets were being rendered inoperable or that insurers were being required to operate an unreasonable cross-subsidy.

5.2.4 Ultimately, the insurers' argument of needing to have access to predictive genetic test results to protect themselves against the exercise of an option by policyholders (the second reason in ¶5.2.1) was the only successful defence which allowed insurers to retain the use of tests under the 2001 moratorium — but only for very large sums assured (see ¶1.16). Even in this area there has been much scepticism from outside the industry as to whether adverse selection is really likely to occur on any significant scale in practice.

5.3 *The GAIC Process 1998-2001*

5.3.1 In retrospect, it may seem odd that ideological and operability reasons were not advanced more by insurers during the debate. However, this largely came about because of the manner in which the GAIC process shaped so much of the debate over this period. The creation of GAIC, and the process it subsequently created and administered, seemed initially to offer insurers in the U.K. a means by which they could demonstrate that they could use test evidence quite reasonably, subject to tight controls, but on a par with other inputs to conventional underwriting. U.K. insurers' energies, therefore, became directed towards quite substantial projects of statistical work intended to demonstrate that tests, whose approval was sought, conclusively indicated uplifts to rates of mortality and morbidity that exceeded certain arbitrary low hurdles set by GAIC (see Section 4).

5.3.2 In the event, the GAIC process did not prove to advance matters as insurers might have initially expected or hoped. Only one test has so far been approved, but with some attaching critical commentary concerning the quality of the submission. GAIC was suspended, pending re-establishment, before it had had a chance to consider the other applications which the ABI had submitted before the original deadline of end 2000. Submissions proved difficult and time-consuming to compile, particularly in view of the lack of

https://doi.org/10.1017/S1357321700004360 Published online by Cambridge University Press

suitable epidemiological data, and fewer were completed and submitted than was originally expected. It became clear that GAIC expectations had not been well understood, and, if test submissions were to pass scrutiny equivalent in rigour to that for work published in academic journals, then it could be difficult for any tests to be approved (the Huntington's disease test was possibly an exception in this regard) and the timescale for achieving approval would certainly be quite a long one.

5.3.3 It is interesting to speculate what would have happened had the moratorium not been introduced. Possibly some approvals of tests might have been withheld by GAIC — ironically there could have been, in the event, a *de facto* moratorium (with the single exception being the Huntington's disease test), without any government action.

5.3.4 It is probably fair to acknowledge that insurers, and indeed some of the actuaries involved, may have misjudged the GAIC process. This misjudgement was exacerbated by the ABI guidelines allowing insurers to continue using some tests on the assumption that they would, in due course, be approved. This tended to stiffen resistance to the industry line, since the public perception was that genetic tests should not be used unless and until they had been approved for use by the GAIC.

5.4 *The Moratorium 2001-2006*

5.4.1 It is worth remembering that prior to the moratorium a few insurers had not sought to use adverse predictive genetic test results at all — whether or not they were within their rights so to do. This made it an even more complex argument — how could some insurers say that it was necessary for them? The different stances can possibly be explained between:

— those who felt that the principles must be defended even when practical dangers were minimal, in order to be in a strong position as and when future problems developed; and
— those who believed that the principles could be better defended if real dangers emerged and were evident.

The different stances were also in part due to different products and pricing and to the different markets within which individual insurers operate.

5.4.2 The moratorium is configured in a way that offers flexibility, in that:

(a) GAIC has been re-formed rather than dismantled. The implications of this are that a body remains whose terms of reference are to balance the interests of the public and of insurers in this sphere.
(b) A route remains by which insurers can protect themselves for high sums assured (although only the Huntington's disease test for life insurance is approved at this stage).
(c) The test approval mechanism is extant, and insurers may still propose tests where they feel their businesses are singly or collectively under threat.

https://doi.org/10.1017/S1357321700004360 Published online by Cambridge University Press

5.5 *Underwriting Practices in General*

5.5.1 It looks increasingly likely that, with or without genetic tests, insurers will be called upon more and more in the coming years to justify traditional underwriting procedures, in line with the general trend in society towards higher expectations of openness, transparency and accountability, and the pressure for evidence to support decisions (Goford, 2003)

5.5.2 Such justification will undoubtedly prove difficult if the method and standard of proof is similar to that which prevailed in the pre-2001 GAIC process. In reality, life insurance rating procedures are characterised by:

(a) approximation and rough estimates which an insurer hopes will overall match its income to its claims, given a fairly broad-brush approach to categorising risk pools;
(b) direct writers' use of reinsurers' rating manuals; these manuals in turn, however, are based on a mixture of scientific evidence and underwriters' and Chief Medical Officers' judgement, and certainly do not purport to demonstrate the rigour required for publication in a peer-reviewed academic journal; and
(c) professional judgement on the part of underwriters' and insurers' medical officers, applied to individual cases in an attempt to be as fair as possible.

5.5.3 Possibly insurers need to marshall a different set of arguments around the underwriting topic:

(a) additional rating mechanisms need to be presented as a means of extending insurance cover to customers who would otherwise be unable to gain cover at 'standard rates' or indeed at all (it is worth restating, perhaps, that life insurers are private trading legal entities and are, in the final analysis, not obliged to transact any particular contract with any individual customer at a given time — of course, this ideal, from the perspective of the industry, is in practice constrained by legislation, regulatory approvals and by public attitudes, for example to ban discrimination, or to prevent 'red-lining');
(b) additional rating mechanisms operate in a competitive market, so the customer is protected — not by rules imposed on the rating mechanism — but by an insurer's fear of being outbid by a competitor; and
(c) competitive forces in effect act to produce the lowest possible premiums, even for higher risks — insurers possibly need to work harder to dispel the notion that there is some malevolent force at work.

5.5.4 The GAIC process, pre-2001, purported to demonstrate that genetic test evidence was sufficiently predictive to be used as an adjunct to conventional underwriting. Yet the paradox is that many aspects of conventional underwriting do not themselves have such a 'scientific' basis,

https://doi.org/10.1017/S1357321700004360 Published online by Cambridge University Press

and, indeed, were not designed in that way, although they may need to move more in that direction in future. It also needs to be borne in mind that insurers are taking on risks for the long-term future. Statistical evidence from the past may be a guide, but it is only that. Insurers have to take risks and accept uncertainty, and it should be recognised that the underwriting process has to reflect such realities.

5.6 *U.K. Insurance after the Moratorium — beyond 2006*

5.6.1 One practical way forward for insurers, in relation to the use that they can make of genetic tests, would perhaps be to require:

— not a demonstration of statistical significance (which in effect is a much more rigorous test than applies to most traditional underwriting); but,
— instead, to require a financial demonstration that insurers are financially exposed to the aggregate risk that single applicants, or groups of applicants, will take advantage of an insurer's vulnerability resulting from an asymmetry of information about the applicant's medical condition.

5.6.2 Insurers might then, for example, paradoxically be unable to produce such a financial demonstration of 'vulnerability' to justify requesting knowledge of the results of a Huntington's disease test in respect of a life insurance applicant. Insurers might not be judged 'vulnerable', since, in this case, they could protect themselves, to some extent at least, by asking family medical history questions. On the other hand, insurers would possibly be able to produce a convincing financial demonstration of their 'vulnerability' if it were proposed that they should not be allowed to know either family medical history or a genetic test result in connection with Huntington's disease.

5.6.3 A financial demonstration of insurers' vulnerability would seem to offer a way of avoiding insurers embarking on specious statistical exercises for which data may not be available for another ten or more years. Requiring such a 'vulnerability' demonstration might also be an effective way of distinguishing between the current high profile monogenic tests and the sorts of multiple-gene pattern analyses which some believe will, in the future, prove to have more importance, given that they could affect a significant proportion of insurance applicants and involve many common and serious conditions.

5.6.4 It may be argued by some that the results of monogenic tests should not be required by insurers — on the plain evidence that insurers have been able to operate quite satisfactorily during the moratorium; but some insurers would wish to argue that their operations are, in fact, becoming severely strained in certain products in certain customer segments and the true effects of the moratorium may take many years to emerge. The 'vulnerability' demonstration would allow the merits of the two stances to be tested.

https://doi.org/10.1017/S1357321700004360 Published online by Cambridge University Press

5.6.5 The U.K. actuarial profession's submission to the HGC 'Whose Hands on Your Genes?' consultation set out some ideas about 'vulnerability'. Subsequently, the HGC drew on these ideas and supported their development (HGC, 2002).

## 6. Looking Forward

### 6.1 *Future Developments*

6.1.1 In this section we consider possible developments in three areas, namely:

— the medical understanding of genes and their impact on the individual;
— the implications of this knowledge for areas where actuaries are asked to advise; and
— the formulation of public policy and law in the light of these developments.

### 6.2 *Genetic Understanding — Monogenic Disorders*

6.2.1 Although much has been achieved in understanding monogenic disorders such as Huntington's disease, there is still a great deal to be discovered relating to currently unidentified genes, and how variations in abnormal genes affect the patient's prognosis. Increased understanding will lead to development of therapies for some, if not many, of those affected.

6.2.2 Our understanding of diseases such as diabetes will be improved by recognising that it is actually a collection of different diseases arising from different genetic abnormalities; treatment is likely to be more effective when it reflects the underlying genetic cause in each patient.

6.2.3 Where links between genes and disease have been established, we will begin to understand better the genes' impact on mortality and health. Accuracy in predicting the course of the disease will, however, be limited by the continuing development of treatments (genetic or traditional), which themselves modify the prognosis for an individual, not to mention the fact that outcomes will always be subject to a significant degree of statistical variation.

### 6.3 *Genetic Understanding — Multi-Factorial Disorders*

Advances may also be made in the understanding of multi-factorial diseases where the prognosis for an individual depends on more than one genetic and/or environmental factor, including lifestyle. It is impossible to predict how much progress will be made in understanding this process, but currently it seems unlikely that significant insights will be achieved in the next five years.

https://doi.org/10.1017/S1357321700004360 Published online by Cambridge University Press

6.4 *Genetic Understanding — Behaviour*

Research has so far concentrated on genetic factors which influence the development of a disease. A new field is the link between genes and behaviour, where a recent paper (Caspi *et al.*, 2002) has shown a correlation between genes and anti-social behaviour. We can only speculate whether statistically reliable correlations will ever be found between genes and, say, claim rates under motor insurance.

6.5 *Economics of Genetic Testing*

Testing will become quicker, cheaper and easier. As therapies are identified for more and more genetic conditions, the day may come when mass screening for a range of conditions becomes cost-effective, raising the possibility that every individual could have access to a significant amount of information about their genetic make-up. Quite apart from the insurance implications, this could have profound implications for society as a whole.

6.6 *A Thought Experiment — what if Access to Genetic Information were Unrestricted?*

6.6.1 Although somewhat remote from reality, it could be a useful thought experiment to consider how companies and individuals might behave in a business environment in which there was full access to genetic knowledge, and no restrictions on its use. This may help us understand the significance of genetic information, and inform the development of public policy where the consequences of unfettered freedom are considered unacceptable.

6.6.2 *Insurance buying practices*

An individual with a family medical history which suggested the possibility of a genetic problem, and who had had a genetic test which confirmed a genetic abnormality, and understood the implications, could modify his behaviour — e.g. by anti-selection in insurance. Insurers would combat anti-selection by seeking access to the same information.

6.6.3 An insurer, alerted by an adverse family medical history declared on a proposal form, might have a commercial incentive to request a genetic test if a reliable cost-effective test existed, comparable with the position for AIDS testing.

6.6.4 With cheap tests, the day might come when individuals with no adverse family history could request a genetic profile, just as many healthy individuals have regular medical check-ups. Whilst this would not change the mortality/morbidity of the typical individual, it would sub-divide the larger group into categories ranging from super-select to substandard, with a corresponding effect on the cost of insurance.

6.6.5 If genetic tests could save more in claims than they cost to administer, it could be in the insurer's financial interest to commission a test for applicants with no adverse family medical history, as they currently do

https://doi.org/10.1017/S1357321700004360 Published online by Cambridge University Press

for medical symptoms such as blood pressure. They might also aim to identify a class of preferred lives for which they could gain commercial advantage by reducing premiums.

6.6.6 *Impact on the individual*

If an insurer wished to, and were allowed to, commission a genetic test, it might lead an individual to confront self-knowledge which he or she would prefer not to have, especially if there was nothing that could be done to modify the risk. Conversely, as the possibility of therapy grows, it could be argued that, on balance, the individual would gain, as a result of being alerted to the need to seek treatment. For blood pressure testing, the latter argument holds sway, whereas received opinion is against genetic testing; but the analogy is very similar, and attitudes might converge as effective therapies are developed.

6.6.7 An individual with an adverse family medical history could be (and some already are) faced with a difficult choice as to whether to take the relevant genetic test, whether as a result of his or her own initiative or because of a request from an insurer.

6.6.8 Untested, he or she would not be able to plan for the future, and would expect to be offered worse terms for insurance than an individual with no family medical history. Insurers would assume that the latent condition would not be treated until it manifested, and that the proposer would not benefit from any treatment which might have been given in the asymptomatic phase of the disease.

6.6.9 Tested, and found clear, he or she could plan for a normal future, and obtain insurance terms without any loading for the extra risk relating to family medical history. Tested, and found positive, he or she could plan for a future likely to involve the disease, including seeking therapy which might modify, or even prevent, the progress of the disease, but would expect to be offered appropriately poorer terms for insurance, allowing for any therapy past or planned.

6.6.10 An individual with no adverse family medical history, who was considering whether to request a test, would also have to face the pros and cons of the new-found self-knowledge which he or she would acquire. To make an informed choice between testing or not would not be easy. Geneticists could offer counselling on the personal issues, but would not be qualified to advise on the insurance issues.

6.6.11 *Impact on insurers*

Continuing the hypothetical thought experiment, if insurers had full access to available genetic information and were permitted to utilise it, they could set the terms of contracts in ways that they thought would make the business profitable, subject to the pressures of the marketplace. Without such access, or with restrictions on the use of genetic information, the financial impact on

https://doi.org/10.1017/S1357321700004360 Published online by Cambridge University Press

insurers would depend upon the extent of individual medical knowledge at the time, and the degree to which applicants used this knowledge to select against the insurer, including the size and type of policies purchased. In the worst case, if a large group of proposers at extra risk on genetic grounds concentrated their policies on a small insurer and were able to obtain ordinary rates, this could lead to financial instability or even insolvency.

6.6.12 *Group insurance*

Similar considerations apply in the field of group insurance, including defined benefit pension schemes. An enterprise might, at least theoretically, be able to gain a competitive edge if it could identify and recruit workers who were expected to be less susceptible to illness during their working life, and, from the perspective of financing defined benefit pension commitments, those who were not likely to enjoy exceptional longevity; but extending this argument suggests that, in a well-informed free market, employees whose genetic susceptibilities would reduce expected pension costs might argue for other forms of remuneration, such as a compensatory salary premium.

6.6.13 Similarly, an employer would naturally prefer to avoid recruiting those who were likely to take more time off work through sickness, but such employees might be willing to negotiate non-standard contracts of employment rather than remain unemployed.

6.7 *An Alternative Future?*

6.7.1 Much of the thought experiment of Section 6.6 would seem familiar to anyone who has followed the debate on genetics and insurance, as it implicitly attributes a high predictive value to much of the genetic information that might become accessible in future; but it is not at all certain that this will be the case, at least in terms of the outcomes that concern insurers, namely the development of disease. It is worth emphasising that the discovery by molecular geneticists of a genetic contribution to a disease might be completely precise in its own terms, but the implications for the development of the disease during the human lifespan might be very difficult to quantify and to disentangle from confounding factors.

6.7.2 Increasingly, some geneticists are beginning to criticise themselves (as a group) for overstating what genetics will lead to and how soon, while their own research is more and more leading to the conclusion that what were thought to be simple genetic disorders are not, including most of the monogenic disorders. There could be a lot of legitimate doubt about the scenario in which people routinely get a genetic read-out, and understand the implications, and that these are then of much relevance to insurers. An alternative scenario is that in (say) ten years' time, a few significant breakthroughs in (say) the neurological genetic disorders will show how genetic information can improve lives and reduce or remove problems about insurance. Meanwhile, few, if any, other categories of genetic information

https://doi.org/10.1017/S1357321700004360 Published online by Cambridge University Press

lead to reliable predictions of abnormal risk much worse than those associated with familiar, non-genetic risk factors. Adverse selection then becoming a remote possibility, insurers' use of genetic information becomes an issue of competition rather than of solvency, and fears about genetic information will dissipate.

6.8 *Public Policy Formation — Education*

6.8.1 Unfortunately, there is a significant lack of public understanding on all matters genetic — whether GM food or cloning or testing. The mechanics of insurance are also not at all well understood, and when the two topics are linked in the same story, all too frequently the result is heat and no light.

6.8.2 We welcome the U.K. Forum for Genetics and Insurance, which is bringing together the experts in different fields and encouraging informed dialogue. There is an important ongoing task of educating ourselves, as actuaries, and of educating other commentators and opinion-formers.

6.9 *Freedom or Control*

6.9.1 A key issue is whether, and to what extent, society should restrict the freedom of insurance companies and individuals to negotiate freely.

6.9.2 One route would be to require equality of information, so that both parties negotiated in full knowledge of the facts (c.f. annual percentage rates of charge — APRs — in consumer credit); another would be to outlaw certain outcomes as socially unacceptable. We do not yet have all the facts needed to inform the public debate on issues of this kind, and there is a key role here for the actuarial profession. This debate itself is influenced by the existence of GAIC; unless this is itself open for review, policy options will be restricted.

6.9.3 Alternatively, if aspects of genetics and insurance are to be controlled, which should be restricted and how? The current régime, in the few cases where rating is allowed on genetic grounds, requires that the ratings be justified by an independent body (GAIC); are we clear why genetic impairments are singled out for close attention when, for example, ratings for hypertension are unfettered?

6.9.4 This leads to a larger question of the freedom of insurers to underwrite, and the extent to which the premiums they charge should be subject to external control. Where certain aspects of social policy might be held back by lack of access to insurance, we should note a difference between freedom to underwrite in order to protect the insurance pool from adverse selection, and freedom to underwrite simply to allow insurers to compete in a free market, and therefore to fragment existing and working risk pools at will. For example, there was no suggestion that life insurers' risk pools 20 years ago were threatened by an influx of smokers that had to be controlled by charging them higher premiums; the latter course of action was taken purely for commercial advantage.

https://doi.org/10.1017/S1357321700004360 Published online by Cambridge University Press

6.10 *Voluntary Arrangements*

6.10.1 As in other spheres of human endeavour, public policy may reflect voluntary agreements, entered into by an industry, which mitigate the financial impact on those with problem gene mutations. The ABI code prohibits the use of information derived from predictive genetic testing in most cases, which appears to provide a manageable solution for the present.

6.10.2 The question is whether an arrangement of this kind is sustainable as genetic knowledge grows; for example, it produces no useful statistics.

6.10.3 The five-year period of the moratorium provides a breathing space to examine the robustness of a permanent ban on using genetic tests, and for other options to be explored. The profession can do much to inform the debate, which is being led by the HGC.

6.11 *Pooling*

6.11.1 One option which should be investigated is some kind of pooling arrangement which enables policyholders to pay premiums which are lower than those which would be charged at arm's-length, with the cost met by the non-impaired through an organised and well-understood mechanism. From the applicant's perspective, the key access issues include:

— Eligibility for subsidised premium rates, such as genetic condition, age, size of policy and product; should this be limited to 'basic needs' (however defined), or should the pool be broader?
— The level of subsidy; should all the extra cost be borne by the pool, or should the policyholder bear part of it?
— Purchasing insurance; how would this be arranged in a world where marketing of insurance is typically highly regulated?
— How should costs be shared between the industry and the taxpayer?

Costs would depend upon the scope of the scheme, and in some scenarios these could be material. No scheme should be launched until these are well understood.

6.11.2 There is a range of options for the mechanics of such a scheme, ranging from a reinsurance pool to a new company. The overhead costs of any of these would not be trivial, and would need careful analysis. An important benefit of a pool could be the ability to collect detailed statistics on the experience of the lives involved, which could be of assistance to future medical researchers.

## 7. Conclusions

7.1 The rapid increase in understanding of the human genome can be expected to bring considerable benefits, in terms of better appreciation of the causes of many medical conditions, which in turn can be expected to lead to

https://doi.org/10.1017/S1357321700004360 Published online by Cambridge University Press

improved opportunities for effective treatment and management of these conditions. Probably the greatest impact that this will have on the insurance industry and on pensions and other financial services will be felt in increased life expectancy and changing patterns of morbidity. It may also mean that people will live much longer with conditions which have, up to now, been considered as likely to lead to relatively early death.

7.2 One way and another, it is likely that these developments will change the map of human mortality and morbidity experience, and that there could be significant changes in the pricing considerations for many insurance products. Failure to appreciate these changes in time could have serious financial consequences for the insurance industry.

7.3 However, with the current state of knowledge there is little that can be said about these prospects, beyond voicing our belief that they could be significant. The current debate on insurance and genetics has focused on a much narrower point, namely the right or otherwise of insurers to have access to the results of predictive genetic tests and the consequences if they do not have such access. The particular medical conditions for which genetic test results are today genuinely predictive are those caused by mutations in single genes (monogenic conditions), and they are all fairly rare in the general population. Even for some of these it is possible that the genetic test information will prove to be much less predictive than many imagine. It is, therefore, perhaps all the more strange that so much heat has been generated over this topic — on all sides. There is little doubt that the real concerns which have motivated the insurance industry's response to this issue relate to the possible long-term implications of genetic tests and the potential erosion of their ability to underwrite freely. They might be even more concerned if the trend towards restricting the use which insurers can make of genetic test results were to be extended to other genetically related information, such as family medical history, since this is used as a marker for potential risk for many of the more common serious conditions, such as coronary heart disease and cancers.

7.4 Perhaps a greater challenge overall for the life insurance industry is that the attention which public policy entities are focusing on it, in relation to the use of genetic information, will lead to a general questioning of the processes used to underwrite in respect of all types of medical condition. Gone are the days when applicants had a deferential attitude towards highly respected financial institutions. In these days of openness, transparency and accountability there will be pressure for insurers to develop and to demonstrate the scientific basis for all of their underwriting policies and decisions and to disclose much more to prospective policyholders on how they are viewed by the underwriting process, especially when they are rated up or refused cover.

7.5 These are fundamental questions for the future of the life insurance industry, and ones which the actuarial profession would do well to address well in advance of them becoming serious issues.

https://doi.org/10.1017/S1357321700004360 Published online by Cambridge University Press

## References


ABI (1999). *Genetic testing: ABI code of practice (revised August 1999)*. Association of British Insurers, London.

Caspi, A., McClay, J., Moffitt, T.E. *et al.* (2002). Role of genotype in the cycle of violence in maltreated children. *Science*, **297**, 851-4.

Chuffart, A. (1996). Genetics: the implications for life. Paper presented at the LIA Convention, 1996.

CMI (1991). *Continuous Mortality Investigation Reports Number 12*. Institute of Actuaries and Faculty of Actuaries.

CMI (1999). *Continuous Mortality Investigation Reports Number 17*. Institute of Actuaries and Faculty of Actuaries.

CMI (2000). *Continuous Mortality Investigation Reports Number 18*. Institute of Actuaries and Faculty of Actuaries.

Cummins, J.D. & Tennyson S. (1996). Moral hazard in insurance claiming: evidence from automobile insurance. *Journal of Risk and Uncertainty*, **12**, 29-51.

Department of Trade and Industry (1998). Government response to the Human Genetic Advisory Commission's Report on *The implications of genetic testing for insurance*. DTI.

Dinani, A., Grimshaw, D., Robjohns, N. *et al.* (2000). A critical review: report of the critical illness healthcare study group. Presented to the Staple Inn Actuarial Society, London, 14 March 2000.

Dullaway, D. & Elliott, S. (1998). Long-term care insurance: a guide to product design and pricing. Presented to the Staple Inn Actuarial Society, London, 10 March 1998.

Fischer, E.-P. & Berberich, K. (1999). *Impact of modern genetics on insurance*. Publications of the Cologne Re, No. 42.

Goford, J. (2003). Thinking and behaviour. Presidential address to the Institute of Actuaries. *British Actuarial Journal*, **9**, 1-33 .

Gui, E.H. & Macdonald, A.S. (2002). A Nelson-Aalen estimate of the incidence rates of early-onset Alzheimer's disease associated with the Presenilin-1 gene. *ASTIN Bulletin*, **32**, 1-42.

Gutiérrez, M.C. & Macdonald, A.S. (2001). Adult polycystic kidney disease and critical illness insurance. Genetics and Insurance Research Centre, Heriot-Watt University: Research Report No. 01/4.

Hoem, J.M. (1988). The versatility of the Markov chain as a tool in the mathematics of life insurance. *Transactions of the 23rd International Congress of Actuaries, Helsinki* **S**, 171-202.

HCSTC (2001). *House of Commons Science and Technology Committee, Fifth report: Genetics and insurance*. HC174. T.S.O., London.

HGAC (1997). *The implications of genetic testing for insurance*. Human Genetics Advisory Commission, London.

HGC (2000). Whose hands on your genes? Consultation paper. Human Genetics Commission, November 2000.

HGC (2002). *Inside information: balancing interests in the use of personal genetic data*. Human Genetics Commission, May 2002.

Le Grys, D.J. (1997). Actuarial considerations on genetic testing. *Philosophical Transactions of the Royal Society* B, **352**, 1057-1061, and (with discussion) *British Actuarial Journal*, **3**, 997-1008 and 1044-1058.

Lemaire, J., Subramanian, K., Armstrong, K. & Asch, D.A. (2000). Pricing term insurance in the presence of a family history of breast or ovarian cancer. *North American Actuarial Journal*, **4**, 75-87.

Macdonald, A.S. (1997). How will improved forecasts of individual lifetimes affect underwriting? *Philosophical Transactions of the Royal Society* B, **352**, 1067-1075, and (with discussion) *British Actuarial Journal*, **3**, 1009-1025 and 1044-1058.


https://doi.org/10.1017/S1357321700004360 Published online by Cambridge University Press

MACDONALD, A.S. (1999). Modeling the impact of genetics on insurance. *North American Actuarial Journal*, **3:1**, 83-101.

MACDONALD, A.S. (2001). Moratoria on the use of genetic tests and family history for mortgage-related life insurance. Genetics and Insurance Research Centre, Heriot-Watt University: Research Report No. 01/3.

MACDONALD, A.S. (2003). Genetics and insurance: what have we learned so far? *Scandinavian Actuarial Journal*, 2003, 324-348.

MACDONALD, A.S. & PRITCHARD, D.J. (2000). A mathematical model of Alzheimer's disease and the APOE gene. *ASTIN Bulletin*, **30**, 69-110.

MACDONALD, A.S. & PRITCHARD, D.J. (2001). Genetics, Alzheimer's disease and long-term care insurance. *North American Actuarial Journal*, **5:2**, 54-78.

MACDONALD, A.S., WATERS, H.R. & WEKWETE, C.T. (2003a). The genetics of breast and ovarian cancer I: a model of family history. *Scandinavian Actuarial Journal*, 2003, 1-27.

MACDONALD, A.S., WATERS, H.R. & WEKWETE, C.T. (2003b). The genetics of breast and ovarian cancer II: a model of critical illness insurance. *Scandinavian Actuarial Journal*, 2003, 28-50.

MCGLEENAN, T. (2001). *Insurance and genetic information*. Association of British Insurers.

PASTERNAK, J.J. (1999). *An introduction to human molecular genetics*. Fitzgerald Science Press, Bethesda, Maryland.

PRITCHARD, D.J. (1997). Life assurance: financial implications of a change in insuring behaviour resulting from individuals' increased knowledge of their genetic predispositions. M.Sc. dissertation, Heriot-Watt University, Edinburgh.

ROSS, T. (1997). The likely financial effect on individuals, industry and commerce of the use of genetic information. *Philosophical Transactions of the Royal Society* B, **352**, 1103-1106, and (with discussion) *British Actuarial Journal*, **3**, 1027-1034 and 1044-1058.

SMITH, C. (1998). *Huntington's chorea: a mathematical model for life insurance*. Swiss Re Publications, Zurich.

STRACHAN, T. & READ, A.P. (1999). *Human molecular genetics, second edition*. BIOS Scientific Publishers, Oxford.

SUBRAMANIAN, K., LEMAIRE, J., HERSHEY, J.C., PAULY, M.V., ARMSTRONG, K. & ASCH, D.A. (2000). Estimating adverse selection costs from genetic testing for breast and ovarian cancer: The case of life insurance. *Journal of Risk and Insurance*, **66**, 531-550.

TAN, K.W. (1997). The financial impact of genetic testing on annuities. M.Sc. dissertation, Heriot-Watt University, Edinburgh.

WARREN, V., BRETT, P., MACDONALD, A.S., PLUMB, R.H. & READ, A.P. (1999). Genetic tests and future need for long-term care in the U.K.: report of a Work Group of the Continuing Care Conference Genetic Tests and Long-Term Care Study Group. Continuing Care Conference, London.

WEATHERALL, D.J. (2000). Science, medicine and the future. Single gene disorders or complex traits: lessons from the thalassaemias and other monogenic diseases. *British Medical Journal*, **321**, 1117-1120.

WILKIE, A.D. (1997). Mutuality and solidarity: assessing risks and sharing losses. *Philosophical Transactions of the Royal Society* B, **352**, 1039-1044, and (with discussion) *British Actuarial Journal*, **3**, 985-996 and 1044-1058.

WILKIE, A.D. (2000). Peer review of application to GAIC for approval of the use of the predictive genetic test for Huntington's disease in respect of life insurance applications.

ZIMMERN, R. (2001). What is genetic information? *Genetics Law Monitor*, **1:5**, 9-13.

https://doi.org/10.1017/S1357321700004360 Published online by Cambridge University Press

## GLOSSARY OF TERMS

This glossary is intended to be a short guide to the relevant subset of the terminology of human genetics. For definitive treatments of this subject, see Pasternak (1999) or Strachan & Read (1999).

| | |
|---|---|
| ABI | Association of British Insurers |
| actuarial fairness | When a proposer for an insurance contract is allocated to a risk group on the basis of the risk that he or she brings to the insurance pool and is charged a premium which matches that risk as closely as possible |
| actuarial relevance | Where the result of a genetic test is significant, from the point of view of an actuary, in determining the individual's risk propensity for a particular insurance product — and hence whether the test is relevant for taking underwriting decisions or determining non-standard premium rates |
| APKD | adult polycystic kidney disease |
| adverse selection | When an individual, having a better knowledge of his or her own level of risk than they need to disclose to the insurer, chooses to apply for insurance when they would not otherwise have done so, or to apply for a greater amount of insurance than they would have applied for in the absence of such information about their own risk |
| allele | There are often two or more alternative forms of a gene, each version being called an allele, any or all of which may be mutated or altered in some way |
| chromosome | A DNA molecule containing many genes joined together; an organism's genetic information is stored in one or more chromosomes |
| CMI | Continuous Mortality Investigation of the U.K. actuarial profession |

https://doi.org/10.1017/S1357321700004360 Published online by Cambridge University Press

| | |
|---|---|
| critical illness insurance | Insurance which pays a sum assured when the insured person is diagnosed as having one of a number of specified conditions or undergoes a surgical procedure specified in the policy |
| DNA | Deoxyribonucleic acid; the chemical substance in chromosomes and genes in which genetic information is coded |
| DNA test | A chemical test involving examination of the constitution of a gene or chromosome |
| family medical history | Information about the illnesses suffered by parents or other close relatives, and, in particular, where applicable, the cause of their deaths, usually in the context of disclosures required by an insurer of a proposer for insurance, in order to inform the underwriting process |
| gene | The biological unit of heredity; a sequence of DNA which codes for one protein or other molecule |
| genetic test | A test to detect the presence or absence of, or change in, a particular gene or chromosome |
| genetic code | A mapping of the genes of a particular organism |
| GAIC | Genetics and Insurance Committee |
| HCSTC | House of Commons Science and Technology Committee |
| HGAC | Human Genetics Advisory Commission |
| HGC | Human Genetics Commission |
| income protection insurance | Insurance which pays a monthly income while an individual is unable to work through illness, accident or injury; the income payments continue until either the individual is fit to return to work or reaches the age of retirement |

https://doi.org/10.1017/S1357321700004360 Published online by Cambridge University Press

| | |
|---|---|
| information asymmetry | The situation which arises when one party to an insurance contract has more information relative to the risk propensity than does the other party; this typically arises when the proposed does not disclose all material information to the insurer |
| life insurance | Insurance payable on the survival of humans for particular periods or on death within certain periods, including whole of life insurance, endowment insurance and temporary life insurance (also known as term insurance or term life insurance) |
| long-term care insurance | Insurance which pays all or a proportion of the costs of any long-term nursing care in the event of a disability which meets criteria specified in the policy |
| monogenic condition | A hereditary disorder caused by a mutation in a single gene |
| moral hazard | This occurs when individuals behave differently in the presence of insurance: *ex ante* moral hazard can occur when a proposer does not reveal all relevant information prior to the conclusion of an insurance contract; *ex post* moral hazard can occur, for example, when an insured person manipulates the level of loss after the occurrence of an insured event |
| multifactorial genetic disease | A genetic disorder resulting from the combined action of more than one gene, or from the combination of genetic and environmental factors |
| mutation | The change in a gene or chromosome that causes a disorder or the inherited susceptibility to a disorder or the ability to pass on such susceptibility to one's heirs |
| penetrance | The penetrance of a genetic mutation refers to the proportion of people with that genetic mutation who develop the disorder. This is usually expressed as penetrance by a particular age |

https://doi.org/10.1017/S1357321700004360 Published online by Cambridge University Press

| | |
|---|---|
| personal medical expenses insurance | Insurance against the costs incurred as a result of medical consultation, treatment, operations and hospitalisation |
| polymorphism | A genetic locus or region of a chromosome at which there are two or more variants that are reasonably common in a population (for example, the genes that confer blue or brown eyes) |
| predictive genetic testing | Genetic testing which is used to determine susceptibility to genetic disease; commonly used to provide an estimate of an individual's risk exposure to a particular multifactorial or polygenic disorder |
| therapeutic uses | Uses which could be relevant for the medical treatment or alleviation of a condition |

https://doi.org/10.1017/S1357321700004360 Published online by Cambridge University Press