

**PREDICTIVE GENETIC INFORMATION AND  
PRIVATE COMMERCIAL INSURANCE  
WORKSHOP, ROYAL SOCIETY OF MEDICINE,  
LONDON, FEBRUARY 22ND 2001**

- 10.45 - 11.00**                      **Welcome and Introduction**
- 11.00 - 11.30**                      **Genetics Scoping Presentation.  
Professor Nick Hastie,  
MRC Human Genetics Unit, Edinburgh**
- 11.30 - 11.45.**                      **Questions and Clarification on Genetics  
Presentation.**
- 11.45 - 12.15**                      **Insurance Scoping Presentation:  
Dr Tony McGleenan.**
- 12.30 - 1.00**                                      **Lunch**
- Afternoon Workshops**
- 1.00 - 1.30 Part One: The Social Good Question.**
- 1.30 - 2.00 Part Two: The Genetic Information Question.**
- 2.00 - 2.30 Part Three: The Prohibited Information Question.**
- Part Four: The Commercial Freedom Question**
- 3.00 - 3.30**                      **Part Five: The Resource Question**
- 3.30 - 4.00 Part Six: Implications for Policy.**
- 4.00pm**                      **Workshop Close.**

## **Genetics Scoping Presentation - Professor Nick Hastie MRC Human Genetics Unit, Edinburgh**

I will use a few conditions to:

- Illustrate the limits of genetic prediction
- Raise various issues
- Look at the changing predictability and different complexity of genetic disorders using 3 examples.

These are:-

- Huntington's Disease
- Bowel Cancer
- Alzheimer's Disease.

What goes wrong at the molecular level? This is the key to genetic prediction. I had assumed that although we would have more understanding of diseases in the next 10-20 years, treatments would be 30-40 years away. However, some remarkable studies using animal models have given hope that treatment may be not such a distant possibility.

### **Huntington's Disease**

Huntington's disease (HD) is just about 100% penetrant so that everyone who carries the mutation, gets it. It is easy to test. Currently, there are no treatments.

It is a disease of the brain with the atrophy of some regions due to cell death. The clinical features include psychiatric and movement problems. After the onset of the first symptoms, it is approximately fifteen years to death, due to brain stem failure. There is some variability in the disease and not all symptoms appear in each case. Studying genetics teaches us so much about the disease. The disease arises through an increased number of CAG repeat sequences in the coding region of a gene that encodes a protein called Huntingtin. These CAG triplets encode a polyglutamine stretch of variable length in the protein.

HD is apparent if the patient has 36 CAG repeats or more. In the normal population, there are from 8 to 35 CAGs repeats. Greater than 36 CAG repetitions is diagnostic of HD. The Huntingtin protein with expanded polyglutamine stretches forms aggregates which lead to cell death. We still do not entirely understand the process.

The more repeats the patient has the earlier the onset of the symptoms, for example, someone who has 90 repetitions will develop the disease in their 40's, whereas with a smaller number of repetitions the onset is later in life. These abnormal proteins are expressed in all tissues but only cause damage in the brain.

Mouse models have been used to help understanding of the disease process and to develop possible treatments. Researchers have expressed amplified polyglutamine stretches in mice and this leads to cell death in the brain. The type of cell death observed is programmed cell death or apoptosis which requires the activation of certain proteins called caspases. Inhibitors of caspases (usually antibiotics) have been given to these mice and the amount of cell death in the brain and mortality is decreased; thus raising the possibility of a strategy for treatment in humans.

## **Colorectal (Bowel) Cancer**

This is mainly thought to be sporadic and not inherited, but it is more genetic than we think. Twin studies have shown that 42% of prostate cancer, 35% of colorectal cancer and 27% of breast cancer occur in part due to the inheritance of genetic risk factors which are likely to work together with environmental and lifestyle factors.

We don't know the genetic risk factors for bowel cancer; it is looked at as one of the risk factors along with the age and sex of the patient, their lifestyle and their diet, inflammatory bowel disease and the use of hormone replacement therapy.

There are several stages to bowel cancer and there are particular somatic genetic changes as the cancer progresses. For example, p53 mutation is nearly always found as a relatively late event in tumorigenesis, whereas inactivation of the APC gene (see below) is always a very early event.

Studies of the few rare inherited cancer syndromes tell us much about the mechanisms underlying the more common sporadic forms. The two major forms of inherited bowel cancer are hereditary non-polyposis colorectal cancer (HNPCC) which accounts for 2-5% of all bowel cancer and familial adenomatous polyposis (FAP) which accounts for about 0.2% of all bowel cancer.

FAP is rare with a prevalence of 1/13,000. It arises through mutation in the APC gene. Mutations in APC are 100% penetrant. Importantly it is possible to identify individuals at risk in families prior to disease onset. Endoscopy can be used to identify polyps before they become malignant and the affected region of the colon removed by surgery. This gives protection against onset of the disease.

HNPCC is an autosomal dominant condition and the penetrance is incomplete. Individuals carrying mutations inherit predisposition to bowel cancer but may also

develop uterine, ovarian or gastric cancers. Unlike the situation with FAP there are no polyps that may act as a diagnostic warning signal. HNPCC arises through mutations in genes encoding proteins involved in DNA mismatch repair. There is a complex of proteins which are required to repair damage in DNA. Individuals with mutations in these repair proteins cannot repair the damage and accumulate mutations in genes, for example the APC gene itself.

This is much more complicated than FAP as genes encoding at least 5 mismatch repair proteins may be mutated in different HNPCC kindreds. However, 2 of these genes predominate. Hence mutations in the LMSH1 gene account for 52% of all HNPCC cases and mutations in the LMSH2 gene account for 45% of all cases.

As well as locus heterogeneity there is also allelic heterogeneity in HNPCC. Hence many different sites in the gene can be mutated in the different families. In 10-20% of cases mutations have not yet been detected. We need to improve the technology to detect and identify the mutations.

HNPCC is not completely penetrant with men having an 80% lifetime risk of bowel cancer and females only a 30% risk. However females have a 40% risk of developing uterine cancer and 9% risk of developing ovarian cancer.

Mutations in HNPCC genes are more likely to predispose to cancer at a younger age. For example, 30% of individuals with bowel cancer under the age of 30 will have inherited mutations in one of these mismatch repair genes. 20% of individuals under 45 with bowel cancer will have inherited mutations in mismatch repair genes.

The number of people who are destined to develop colorectal cancer in the UK population amounts to approximately 480,000, with 15,000 due to high penetrance mutations of the MMR gene, or rarely other single genes such as APC.

The vital aspect about bowel cancer is that the disease can be treated if it can be detected early. Surgical removal of early lesions is associated with a 95% chance of long term survival. However the disease is normally detected at a much later stage so there is reduced survival. Having genetic knowledge increases the possibility of identifying it in the early stages, and thus increasing the survival rate.

## **Alzheimer's Disease**

Alzheimer's disease (AD) is a common form of dementia, characterised by aggregates of protein called plaques which accumulate around neurones in the brain. Through some unexplained mechanism this leads to neuronal cell death. These plaques consist of a small 42 amino acid protein called beta amyloid.

Whereas the majority of the disease is sporadic or multifactorial, involving genetic and environmental risk factors, there are rare inherited forms of early onset AD that have shed a great deal of light on the mechanisms underlying pathology.

The beta amyloid peptide is an abnormal product of the so-called amyloid precursor protein that normally resides at the surface of nerve cell membranes. For a long time it was unclear whether these beta amyloid plaques played a causal role in pathogenesis or whether they were innocent bystanders of the disease. Genetic studies have provided unequivocal evidence that beta amyloid plays a causal role. In some rare forms of early onset Alzheimer's disease the amyloid precursor gene itself is mutated. These mutations lead to an increase in the levels of beta amyloid. In two other slightly more common inherited forms of AD, the mutant proteins are involved in processing the beta amyloid.

AD is (at present) an unusual common disease in that a major genetic risk factor in the population has been identified. The apoE protein has been known for many years as a protein that carries cholesterol. There are various common polymorphic forms of apoE in the population. Individuals carrying one of these forms, apoE4, have a 10% chance of developing Alzheimer's disease. Individuals who are homozygous for apoE4 have a 50% chance of developing the disease by the age of 80. 30% of the population carry one apoE4 allele. However another allele, apoE2, appears to protect against the disease. Again it seems that apoE4 indirectly increases the levels of beta amyloid suggesting that the mechanisms involved in common AD are the same as those in the rare familial forms.

These findings have raised the distinct possibility of new rational therapies for the disease. For example, drugs have been identified that inhibit the enzymes that produce the beta amyloid peptide. These will soon be in clinical trial. However the most exciting recent development concerns studies with mice that overexpress the beta amyloid protein and develop pathology similar to that seen in humans. These mice also have reduced ability to perform certain cognitive tasks such as learning how to negotiate a water maze. Remarkably when these mice are vaccinated with the beta amyloid protein have reduced pathology and are much better at carrying out these simple learning tasks.

I have described 3 conditions of varying genetic complexity, illustrating the variation in the ability to make clear-cut predictions about outcome. Also, in all 3 cases, therapies may appear in the not too distant future.

## **Genetics & Insurance Seminar Presentation**

### **Dr Tony McGleenan**

This scoping presentation was intended to brief workshop participants on some of the key components of contemporary insurance with reference to life, critical

illness and long term care insurance products.

## **Conditions for Private Insurance**

In order for a private commercial insurance market to function the following five conditions have been suggested as prerequisites. These are:

- Individual independent risks
- Uncertainty about the outcome (or date)
- Susceptible to actuarial analysis
- Symmetrical information
- Absence of moral hazard

To what extent will predictive genetic information affect these components? Not all of these conditions are satisfied in every insurance market. However, it is legitimate to ask in the light of predictive genetic information what compromises have been made, what regulatory measures taken and what actions will follow if these conditions are not satisfied?

## **Symmetrical Information**

In most contracts the assumption is that both parties are privy to the same information. With insurance contracts there is often dissonance between the parties. Insurance requires an alteration of the normal law of contract. In insurance, the standard contractual basis of *Caveat Emptor* (Buyer Beware) is replaced by *Uberimmae Fides* (Utmost Good Faith). How does predictive health care information impact on this relationship? If individuals have information about their healthcare status which they do not share with the insurer then there is a real risk of asymmetrical information arising.

The classic principle of *uberimmae fides* was set out in the case of *Carter v Boehm* 'Insurance is a contract upon speculation. The special facts upon which the contingent chance is to be computed, lie most commonly in the knowledge of the insured only: the underwriter trusts to this representation and proceeds upon the confidence that he does not keep back any circumstance in his knowledge.'

## **Adverse Selection**

One of the major concerns of insurers is adverse selection. This is the idea that individuals will act differently in the possession of asymmetrical information in their relations with insurance contracts. Adverse selection arises when a disproportionate number of bad risks enter the insurance market at standard premium rates. The good risks will then face premium rises and may leave the market. This leaves the risk pool with an increased number of bad risks with

rising premiums. In insurance theory this can lead to an adverse selection spiral which can destroy a market.

As Pokorski points out individuals can have a strong financial incentive to engage in adverse selection. *'If life expectancy is much shorter than anticipated then purchasing life insurance at standard rates is the world's best financial investment.'* (Pokorski, R.) An investment of £15/month translates into £100,000 in life insurance. In certain circumstances, the insured can play the market to significant advantage.

There are a number of key factors which affect adverse selection, these include:

- the rate of purchase of insurance
- the rate at which bad risks purchase insurance
- the sum assured values purchased by bad risks
- the maturity of the market (LTC less than 1%)
- the mitigating effects of regulation
- the availability of genetic tests (cost and predictive value)

## **Adverse Selection and BRCA1/2**

The possibilities of adverse selection arising as a consequence of genetic testing can be illustrated by considering the following scenario based on BRCA1 testing. Women with BRCA mutation are at extreme risk of developing BC or OC. BRCA mutations are responsible for 80% of all inherited BC and 5% of all BC. Since 1996, a genetic test had been available for detecting these mutations.<sup>1</sup> Woman at high risk may purchase more life insurance whilst woman at low risk will purchase less. Thus the aggregate mortality of the risk pool increases and the health profile worsens. If there is informational symmetry the insurers can price premiums accurately, conversely, they must increase premiums if information is withheld. Increased premiums will lead to the selective lapsing of low risks thus diminishing the pool. Those low risks who remain may seek to insure for below average sums. High risks however may seek to insure for above average sums (e.g.: 2-3 times as much). The corollary of adverse selection is a spiraling market which must eventually implode.

## **Moral Hazard**

Moral Hazard occurs when individuals behave differently in the presence of insurance than in the absence of insurance. For example; they may exercise less caution, may seek healthcare intervention earlier or require a higher quality

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<sup>1</sup> Lemaire, J., Subramaniam, K *et al.* "Pricing term assurance in the presence of a family history of breast or ovarian cancer" **4** *North American Actuarial Journal* (2000).

of care. The rational choice for any individual is overutilisation of resources.<sup>2</sup>

In the United States, for example, workers compensation costs as per cent of the payroll were 1.96% in 1980 and rose to 2.36% in 1990. In the same period, workplace fatalities fell by 40%.<sup>3</sup> If safety is increasing, moral hazard explains the higher incidence of insurance cases. For example, most workplace injuries these days are soft tissue rather than bone injuries. The Monday morning phenomena is common – a worker might sprain an ankle playing sport during the weekend and then report it during Monday, assigning it as a work place injury.

One question for underwriters is how to deal with this type of misrepresentation? One long standing practice has been to subject all proposals for certain types of insurance to the scrutiny of underwriters.

## Underwriting

The task of the underwriter is to attempt to accurately match risk to premium. There is currently political pressure to restrict underwriting **based on genetic information. This pressure can be explained in part by a confusion between the concepts of actuarial fairness and moral fairness. Actuarial fairness requires equity between risks in the charging of premiums. Moral fairness is a much broader concept which may require, for example, equity between individuals in the distribution of goods. Those who argue that underwriters should be restricted in the practices tend to conflate the concepts of moral and actuarial fairness.**

There are however legitimate concerns about underwriting which include those of:

- Accuracy
- Controllability
- Suspect variables
- Privacy

Underwriting involves a process of discrimination in the value-neutral sense. This is reflected in the fact that many anti-discrimination laws contain exceptions for “fair discrimination” by insurers. The areas listed above may lead to objections about “fair” discriminatory underwriting practices.

## Accuracy concerns

Actuarial fairness requires matching risk to premium. Risk classification can

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<sup>2</sup> Dowd, B.E. “The logic of moral hazard: A game theoretic illustration” **49** *Journal of Risk and Insurance*, (1982).

<sup>3</sup> Butler, R *et al.* “Increasing claims for soft tissue injuries in workers’ compensation: Cost shifting and moral hazard.” **13** *Journal of Risk and Uncertainty* pp.73-87 (1996).

never be completely accurate. The risk factors used in classification are selected on the grounds of utility, efficiency and availability.

Take the pricing of motor insurance as proxy for risk. Higher premiums are imposed for younger drivers. There is internal unfairness as all younger drivers will be put in the same category regardless of their safety record. If you take ten drivers all with an identical risk classification, you can't further subdivide.

## **Accuracy and BRCA1&2**

In the context of genetic information we can return to the example of BRCA1/2. If we assume that 30% of Ashkenazi Jewish women will develop hereditary forms of breast cancer related to BRCA1 and BRAC2. The risk could be classified by genetically testing all woman in the category to accurately define the risk. An increased premium could be charges to all women of Ashkenazi Jewish origin. In such an instance religion is used as a proxy for the genetic test result. There will inevitably be inaccuracies in these classifications but there are also cost **savings to be gained from** not using the genetic test. It is possible, therefore, that the use of genetic test information can actually lead to a reduction in unfairness.

## **Limiting Factors in Genetic Testing**

Genetic testing is not currently widespread. Nor is it likely to be widely available in the near future. There are a number of factors which continue to inhibit the increased clinical and diagnostic uses of genetic tests. These include:

- The cost of the genetic test
- The accuracy of the genetic test
- The predictive power of the genetic test
- The approach of competitors

## **Controllability**

Those who are opposed to the use of genetic tests in insurance classification often argue that they are unfair because they involve risk rating based on characteristics over which the individual has no control. It is suggested that these risk rating variables are outside the control of the proposer thus denying individuals the opportunity to exercise individual control to effect a change in risk categorisation. Why is this particularly unfair in relation to genetic information? Is genotype controllable? Is susceptibility to multifactorial disease controllable? It has been pointed out that many widely accepted risk rating variables are, in part, uncontrollable. The example was given of flood insurance.

## **Suspect Variables**

A further strong objection to underwriting practices is that they sometimes utilise what are called suspect variables. This applies to the use of risk factors which are:

- objectionable on symbolic grounds (race?)
- insufficiently probative (unreliability of individual genetic tests and potential for false responses)
- used to disadvantage but not advantage
- used to perpetuate external (non-insurance) disadvantage.

Thus, for example, if a religious affiliation were a reliable proxy for an increased susceptibility for a genetic disease then it is possible that insurers would use this to rate risk. However, if **that religious affiliation had been** the source of historical disadvantage then this would be a suspect variable.

These are all arguments used to restrict underwriting. Age and sex are uncontrollable factors but insurers are allowed to take these into consideration. In the USA, race is also used as a riskrating factor (undifferentiated in the UK) but this factor blurs the boundaries of fairness/unfairness and shades into controversy. Fairness is finally a subjective decision and consideration must be given to what is *fair* and *unfair* discrimination.

## **Restrict Underwriting**

If the argument that we should restrict underwriting is accepted by policymakers. Then questions such as why? Restrict what? How? Cost? Who pays? must be addressed. In the UK the ratio of OR: IP refused is 95:4:1. There are two consequences of widespread genetic testing. In the case of monogenic diseases such as HD, unaffected individuals could be admitted, or in the case of multifactorial disorders, premiums may be increased for those affected. A small number of individuals with monogenic disorder will be included in the OR class which may lead to adverse selection or conversely, adverse selection may be mitigated. A larger number of individual lives with multifactorial disorders may be included in the OR class, but increased mortality of these individuals may be marginal.

## **Insurance as Social Good**

One of the issues to be explored further in the afternoon workshops is the question of the relationship between insurance and social goods. Insurance is perceived in a number of different ways, as:

- a consumer commodity
- a pragmatic means of securing a capital sum

- a means of externalising the risk of a calamitous event
- a socially important good *per se*.

Pressure for restrictions on underwriting is increased by the fact that insurance is sometimes seen as a vital prerequisite for certain social goods. For example, it is perceived as an access point for housing. These arguments may have more rhetorical force than they deserve in societies where such social goods are also provided by the state.

Long Term Care (LTC) insurance raises an interesting questions about its role as a social good. 6.8 million people are engaged as informal carers in the UK. The number of people needing some form of LTC in 1995 was 6.5 million. The number projected to need some form of ITC in 2031 is 8.8 million. The Age Dependency Ratio will rise from 30% today to 50% in 2035. Is LTC a social good? Is long term care *insurance* a social good? If it is, then there is greater leverage for restricting underwriting. There is also a greater case for state provision of that social good. This is something which only the Scottish Executive seem to have accepted,

## **How do we restrict underwriting?**

Are laws needed to restrict the use, disclosure or acquisition of genetic information? It is problematic that insurance already uses non-molecular predictive information in underwriting. Drawing a conceptual difference between these practices and the use of similar information derived by molecular means is very difficult. How are we to *define* genetic information?

US jurisprudence regarding genetic information has evolved over the last 15 years. In its first phase, information about single disease, such as for example, sickle cell disease could not be admitted. A second generation of laws required the informed consent of a proband before any genetic information could be used. Autonomy issues about consent are problematic - informed consent is a difficult concept. The third generation of genetic privacy laws in the United States restrict the acquisition of genetic information. If you can't *get* the information in the first place then you can't *use* it. Is there is any conceptual difference between the practices and the use of similar information derived by molecular means?

In summary, the following questions are, I believe, central to the debate on genetics and insurance. These will form the basis for much of the afternoon discussion.

1. What type of social good, if any, is related to healthcare, life, LTC or critical illness insurance?
2. If the insurance product correlates with a social good is it so necessary that it justifies the disruption of actuarial principals to support provision? What is

the justification? Is it that genetic information is different? If so, how is it different?

3. If a case can be made for setting aside actuarial principles, for which particular insurance products, should this be done?
4. What will the costs be for setting aside conventional actuarial principals in relation to these products?
5. Who should bear this cost? The insurers/shareholders, policy-holders or taxpayer?
6. What are the downstream consequences of this cost-shifting?

## 1. The Social Good Question

This Part of the Seminar examined the question of what sort of good, social or otherwise, life insurance, critical illness insurance and long term care insurance are perceived to be in the United Kingdom. Are these products to be considered as primary social goods? If so, is the current balance of provision for such goods through private commercial insurance and state provision an adequate or appropriate model?

- This discussion needed to be informed by a definition of “social good”. The definition was advanced of (i) primary social goods (elements essential for basic human dignity) (ii) secondary social goods (elements required to enhance quality of life beyond a minimum standard and (iii) commodities (items which could not be considered to be socially essential).
- The State provides a minimum level of support in terms of primary social goods. There is some dispute as to whether this should be seen as a baseline or a safety net. An overriding questions is whether or not the current level of state provision for primary social goods is adequate. If the state considers something to be **essential** it will have either provided it or made private sector provision mandatory. Whilst views on what constitutes social goods may evolve changes should be coherent, not piecemeal. Provision of “secondary” social goods (i.e. those which are desirable, not essential) is normally by encouragement or facilitation not legislation.
- In the context of genetic illness, those who are affected suffer compounded disadvantage in relation to social goods. Although individuals genetically at risk may currently be asymptomatic themselves a family history of ill health may have depleted their resources and capacity for self reliance. This may have been an operative factor for a number of generations in a particular family. This may reflect on whether the minimum level of state sustenance for primary social goods is adequate in these specific cases. These historical and intergenerational factors may be of significance in deciding whether specific policy dispensations ought to be created for certain categories of genetic disease.
- Market behavior may give some indication of the social prioritisation of insurance products. Insurance is not universally purchased. The market for

long term care insurance is extremely small. Even the largest market, life insurance, is composed in large part of individual households which have purchased term assurance in conjunction with a house purchase or those who have cover as a result of an occupational scheme. Given that few people voluntarily seek out life insurance and other related products it is difficult to argue that it is a *primary* social good.

- A number of suggestions were made as to what were social goods including such things as choice and personal responsibility. Limiting the freedom of the market, it was suggested, would also limit the opportunity to avail of these social goods.
- It was suggested that if it is decided that the provision of certain insurance products is seen as being inextricably linked to the acquisition of a social good then the state can intervene in a variety of ways. It was suggested that state regulation is not the only alternative available. It could be possible to stop short of legislation and to attempt to influence purchasing behavior in other ways. The example cited to support this was the introduction of stakeholder pensions, although some feel it to be stretching the definition of “social good” beyond tangible items into areas of personal choices and responsibility.
- To the extent that life insurance can be seen as necessary to support house purchases and housing is agreed as a social good then life insurance can be argued to be a social good in that context.

## **2. The Genetic Information Question.**

This Part examined the question of whether genetic information should be considered as special or different from other forms of predictive healthcare information. Should the debate on the use of genetic information in insurance be confined to an analysis of the use of molecular diagnostic information? If the debate is to be so confined what are the downstream consequences of approaching the problem in this way?

- It was suggested that the public perception was that genetic information was in some way different from other healthcare information. While it may be difficult to draw a clear conceptual distinction the perception remains. It was noted that misunderstandings and misperceptions often dictate modes of behavior in market environments. It was also pointed out that the public perception of genetic information could also be altered through an educative process. Given that society has determined that insurers must certain groups (men/women, disabled people) fairly in that premiums relate to risks brought to the pool, it would seem ultimately to be the case that genetic risk should fall into the same category.
- It was noted that genetic information may in fact feel different to an individual who is presented with the outcome of a genetic diagnostic test. While medically speaking, or indeed, actuarially speaking there may be no

difference, the ethical, emotional and familial implications made the information “feel” different.

- It was also noted that the existence of GAIC and the operation of a “licensing” system for genetic tests was an implicit recognition that DNA was different. It was argued that it would be difficult to row back from that now and formulate a position based on the (logically defensible) claim that DNA information was no different from any other predictive or diagnostic information.
- If a case is being made (or sustained) that DNA is different than this can be supported by two points. Firstly, genetic information has an intergenerational and cross-familial impact. Secondly, genetic information should be treated differently because (unusually but not uniquely) private individual disadvantage (uninsurability, higher premiums) is incurred as a direct consequence of public sector investment in genetic technology, notably the human genome project. This work has been funded by the public and will benefit many individuals in society. Those with an adverse genetic test result will fall into a small category of “losers”. Should they not derive some protection for a misfortune which has been indirectly funded by the public purse?
- Genetic information is different because of accuracy. This can have different impacts. Accuracy is a good thing from a population point of view. Given current technology many genetic tests for Mendelian disorders are not particularly accurate either in the sense that they reveal the presence or absence of a mutation or that they are accurately predictive (e.g. BRCA1 or 2). Family history information may also add to the predictive power of the genetic test result. It is better from a public health perspective to have clear and accurate information about population health. It is also better from an actuarial point of view to have clear and accurate information about the aggregate mortality of the insured population. However, from an individual perspective this accuracy can have grave detrimental consequences. It was noted that for increased mortality of +500% no insurance coverage would be offered.
- The inaccuracy and public acceptability of family history information was commented upon. Family history information is based on subjective reporting of historical information. It can often be extremely inaccurate. Family history is seen as an acceptable means of underwriting. Genetic information can actually provide a more accurate underwriting tool and reduce mis-rating.
- It was noted that almost all disease has a genetic component. This is obviously more influential in the small (and atypical) single gene disorders such as Huntington’s Disease. It was noted that the number of single gene disorders was likely to be small. The suggestion was made that an arrangement be made to “look after” individuals falling into this category since the numbers involved was likely to be small. It seems very likely that predictive genetic testing for common disorders will prove to be biologically implausible. In future genetic and other factors may provide predictive

phenotypic markers for future diseases but the extent to which this may prove to be so is debatable.

- Comment was made on the predictive power of other non-genetic information. Cholesterol, smoking, diet and social class can all be of predictive value in underwriting decisions. The public at large may be resistant to the use of DNA information in underwriting decisions but they do not reject the use of other modes of predictive information.
- The numbers of individuals involved with genetic disorders was quite small (it was claimed). The question of whether this picture would alter in the future was examined. If there are only six or so monogenic disorders then this cost may be sustainable. However, new predictive methods may lead to a different scenario. The geneticists opinion seemed to be that there were not many more genes to be discovered and that while new techniques may increase the scope for predictive testing this would still take place in the area of multifactorial disease.
- It was also noted that genetic testing was not currently a widespread phenomenon. One of the reasons for this is that there is currently, in most cases, no therapeutic option available. If however, there was a therapeutic option following on from a negative diagnostic tests this would have two consequences for actuarial practice. The number of tested individuals in the population would increase, the information would have to be disclosed and a clearer picture of mortality would be obtained. The therapy would have an impact on the overall mortality. Provided actuarial consideration were taken of the second point as well as the first the net detriment may be limited significantly.
- General comment was passed on public understand and misunderstanding of both genetics and insurance. In terms of insurance the concepts of discrimination and differentiation are generally misunderstood. In relation to genetics a perceptions has developed, perhaps through exposure of forensic techniques, that DNA information equates with certainty. The public have therefore developed a deterministic view of genetic information which causes difficulties.

### **3. The Prohibited Information Question.**

This Part of the session explored the potential consequences for the insurance industry and the consumer of ignoring (or being prohibited from using) various forms of predictive information. In particular, what would the implications be of ignoring predictive information derived from DNA analysis, information derived from family history and information derived from other predictive diagnostic methods.

- The 95:4:1 ratio was pointed out in support of the argument that the majority of proposers are insured at ordinary rates. There are significant costs to the insurer in attempting to impose sophisticated underwriting tools.

- Prohibiting the use of genetic information was undesirable because it reinforced a public perception that genetic information was toxic information that must be kept secret or else negative consequences would flow. This perception was bad from a public health perspective as it would militate against openness and the sharing of genetic information. It is important to be clear that there is no requirement by insurance companies to be tested as part of the process of seeking insurance cover. Nor is there any intention to introduce this requirement.
- Questions were asked about the extent to which the principle of *uberimmae fides* was adhered to? Are the rules on the disclosure of information rigidly enforced at the proposal stage.
- It was repeatedly noted that if banning of underwriting was being considered this must be seen on the context of particular insurance products. Different issues apply in relation to long term care and critical illness insurance than apply in relation to income protection life insurance. It is also important to consider the size of the insurance pool and the maturity of the market. The size of the sums assured is also a significant factor.
- It was suggested that there is no need to prohibit the use of genetic information because ultimately everything is insurable provided an appropriate premium is paid. In reality, however, cut-off points based on mortality increases will be imposed.
- The relationship between different insurance products should be explored. If currently asymptomatic but genetically at risk individuals “bundled” their financial service products with the one provider then difficulties in relation to life insurance, for example, would be offset by the altered demands on the pension funds. In relation to long term care insurance it was suggested that this could be “bundled” with (whole) life assurance policies. This would enable the LTC pool to grow to a size where it would be able to withstand a degree of adverse selection. It was claimed that people with genetic disease are not a “cost” in a global sense because they tend to have a reduced lifespan thereby reducing their impact on pension funds, for example. This would not apply to money purchase schemes, where each member has an individual “pot”.
- It was suggested that the single gene disorders would generate costs which were absorbable by the insurance industry providing the sum insured was capped. The real difficulty lay with multi-factorial disorders where the genetic component was only one of a number of factors and where the distribution in the overall population was of a significantly higher order.
- It was observed that banning underwriting was not a cost-free option. The cost would have to be borne by either the shareholders, the policyholders or the state (taxpayer). If the increased cost burden were imposed on policyholders then this would lead to more individuals not entering the pool. It was claimed that there seems to be no independent evidence that having the use of the genetic test results currently under consideration by GAIC would lead to increased costs. Further research is needed to clarify this.

- There was a difficulty with ring-fencing any prohibition. There was little consensus even among existing legal norms as to the appropriate definition of genetic information which should be used in situations where insurers were to be prohibited from using “genetic information” in underwriting.
- Banning the use of genetic information could also have a negative effect in terms of overall actuarial fairness as this information can be used for population purposes rather than in individual rating.

#### **4. The Commercial Freedom Question**

This Part examined the issues which might arise when an insurer, having complied with the various regulatory mechanisms and having utilised objective scientific data, forms the view that a particular insurance product is not commercially viable because of the threat posed by predictive information. Should such a commercial insurer be subject to any duty to provide such a product? If so, what implications could flow from this?

- It was suggested that the implication that insurers should have a duty to insure should be inverted. The suggestion was that it should be compulsory for the public to take responsibility and insure themselves. In a compulsory model the low risk individuals will cross subsidise the high risks.
- In a compulsory model the question was whether there was added value in having such a system administered by the private insurance sector rather than the state. It was suggested that competition and efficiency would be gains from the private sector approach. It may also foster a greater sense of responsibility.
- It was indicated that in compulsory markets risk rating of premiums was offset by the option to insure for a lesser degree of cover. In a compulsory market for, say, long term care, it may not be possible or desirable to opt for “third party” cover.
- A compulsory market may not be attractive for the insurance industry. Such a model would require strong regulation with real impact. There may be a political disincentive here as a government may be reluctant to align itself too closely with a market model which might ultimately fail. The example of stakeholder pensions was cited in support of this point.
- It was suggested that there is a degree of public acceptance of fraud against the insurance company. This was perceived as a form of “victimless crime”. Why? Perhaps because it is seen as small individual against massive corporation. However, the same power asymmetry applies to activities such as shoplifting which do not enjoy (apparently) the same level of tacit social acceptability.
- Issues of fairness and equity were raised. If the insurance industry decided to absorb the costs of making a special arrangement for single gene disorders, how can this be justified to other proposers who might become symptomatically identical but not as a consequence of a single gene disorders? This may well be inequitable. One response to this is that a

certain amount of inequity and unfairness is probably inevitable. If the unfairness is as a consequence of political as opposed to actuarial decision-making then that is not a matter for the insurance industry alone. It was suggested that a goal for public policy in this area must be to minimise the amount of inequity.

## **5. The Resource Question**

This Part develops the issues raised in Part Five and examines the scenario where an insurance company is placed under a duty to provide a particular insurance product notwithstanding an adverse commercial analysis. What compensatory mechanisms should be made available to such an organisation - state subsidies, increased premiums, cross-subsidisation etc.

- It was suggested that the overall cost of the single gene disorders could be borne by the insurance industry so long as the over included was to be capped. Life expectancy was still increasing therefore the overall size of insurance pools was also increasing.
- In response to the argument that compelling insurers to insure without full underwriting imposed a social cost on commercial companies it was stated that this occurred in other contexts. For example, disability discrimination laws have imposed costs on private business by requiring the modification of buildings. In such a situation the impact on an individual company of becoming a “target” for high risk customers would have to be considered. In the event of such an occurrence measures to redistribute risks might need to be considered.
- It was suggested that the impact of genetics was not important enough to justify state regulation. A distinction was made between the actuarial and political impact of genetics. Actuarially the case for treating genetic information differently was not convincing. The political importance of genetic information was another matter.
- Questions were asked about the actual behavior of the public in relation to genetic testing. Was it the case that take-up of genetic testing was low because of concerns about insurance? This question ought to be addressed with empirical analysis of public behavior. Such research could be conducted “back to back” with research as to how insurance companies decided on appropriate loadings on premiums in the light of genetic test information or family history.
- In the United Kingdom it was suggested that the life insurance industry was extremely competitive. The margins operated by the companies were relatively small. Extra costs could not be easily absorbed and would have to be passed on in some form. This could be contrasted with the situation elsewhere in Europe where price competition is less evident, where large premiums are charged and where pricing is done in retrospect. In the United Kingdom once the contract price is agreed it is not reviewed during the term.

- In the life insurance market it was noted that in real terms premiums have been reducing for almost 20 years. It was suggested that this indicated that the policyholder's were prepared to pay more for their insurance because in their previous experiences they had, in fact, paid more.