

*Report to  
CIA Research Committee*

**Genetic Testing Model:  
If Underwriters Had  
No Access to Known Results**

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## Memorandum

To: CIA Research Committee

From: Robert C. W. (Bob) Howard, FCIA, FSA

Date: July 10, 2014

Subject: **Report: Engagement to model the impact of underwriters of individual life insurance having no access to the results of genetic tests**

The Research Committee engaged me to construct a model to assess the impact on companies and the public if underwriters were prohibited from accessing the results of genetic tests known to applicants. I was to consider individual life insurance only. The genetic assumptions were to be provided by a committee of doctors and underwriters assembled by the Canadian Life and Health Insurance Association (CLHIA), but otherwise my modelling was to be independent of the CLHIA and member companies.

The Research Committee appointed a research project management team (RPMT) chaired by Bernard Naumann and including Alison Begley, Robert L. Brown, Greg Cerar, and Paul Fryer, all Fellows of the Canadian Institute of Actuaries. My work was supervised by the RPMT. Both method and assumptions were discussed with RPMT at length.

This document is a report on my work: a description of the model method and assumptions, my observations from the modelling, and my conclusions about the impact on the insurance companies and the insuring public of Canada.

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## 1 EXECUTIVE SUMMARY

Consideration is being given in some legislative bodies in Canada to prohibiting life insurance companies from accessing the results of genetic tests for the purpose of underwriting a potential insured. Such an action would create an imbalance of information between the applicant and the insurance company. This report describes a model that explores the impact of the prohibition on insurance company results and on the premiums paid by Canadians. The model considers 13 conditions that are known to be associated with a genetic marker and for which reasonable estimates of their effect on mortality are known. The model simulates the purchase of insurance, premiums, expenses, and death claims separately for each condition.

The key assumptions, other than those related to mortality under the 13 conditions, are what proportion of those who test positive will seek to buy life insurance and how much will they buy. The baseline assumption in the model is that 75% of those who test positive will apply for as much as they can get, and the rest will not seek additional insurance. The greater the publicity surrounding the prohibition, the higher will be the proportion buying insurance.

I conclude that the impact on insurance companies will be substantial. The valuation strain (pricing loss) for the industry from those who test positive in a single year (based on the assumptions) would be about 12% of the total death claims for the year. The impact on consumers is likely to be even greater. As a result of the prohibition the average mortality rates are likely to increase by about 35% for males, and 60% for females in the age range 20–60; there would be a concomitant increase in term insurance premium rates.

It is important to note that the results are highly dependent on the assumptions, particularly the amount of life insurance sold to those who test positive, whether through each purchasing a larger amount or a higher proportion seeking insurance.

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### **3 INTRODUCTION**

In our society, genetic tests are becoming increasingly affordable and accessible. This is good for the public because people are able to determine whether they are prone to certain serious conditions. Knowing that they have a high probability of an illness often improves the outcomes thanks to closer monitoring and lifestyle changes before the illness becomes manifest. Of course, the presence of a particular gene does not, in most cases, indicate that the person has the disease currently; there remains uncertainty about if and when the disease will emerge.

For many of the more serious conditions, those who test positive for the gene or genes associated with the condition will recognize that their life expectancy is markedly impaired compared to the rest of the population. It would be logical for them to want to acquire additional insurance, particularly if it can be had at a favourable price.

Some European countries have enacted legislation to make the results of genetic tests inaccessible to underwriters of insurance. Consideration has been given in some Canadian legislative bodies to doing the same. If insurance underwriters are not permitted to know the results of genetic tests that are known to the applicants, then many of those who test positive will be able to acquire life insurance at the same price as those who test negative.

The purpose of my model is to explore the actuarial implications of an enforced imbalance of genetic information (the applicant may know it, but the underwriter may not) and to determine whether there is likely to be a material impact on the individual life insurance market in Canada.

### **4 MODEL SPECIFICATIONS**

#### **4.1 Overview**

My model simulates the purchase of life insurance policies in one year by those who test positive for any of a number of genetic markers and follows the policies for many years. The totals for these policies are compared to the total death claims for the industry to estimate the overall financial impact on companies (cost model). The totals for these policies are also compared to the totals for a mortality study done by the Canadian Institute of Actuaries (CIA) to determine the impact on observed mortality rates over a narrower range of ages (experience model).

My model simulates individual life insurance only. There would likely be little impact on group insurance, other than perhaps for large amount optional insurance. There is

typically no underwriting other than ensuring that the member is actively at work. The amount of insurance is determined by salary or other objective factors. The same people would be covered and for the same amounts regardless of whether there is genetic testing. It is doubtful that there would be any impact on premium rates. The concern is only for those types of insurance for which the individual has discretion on whether to buy and how much to buy.

A similar study could be done for individual health insurance, especially critical illness insurance. It is likely that the impact on individual critical illness insurance would be much greater than for individual life insurance, but my model does not explore the issue at all.

## **4.2 Assumptions about Genetic Markers**

The assumptions related to the genetic markers were provided by a committee of medical doctors and chief underwriters drawn by the Canadian Life and Health Insurance Association (CLHIA) from its member companies. Their work (especially for prevalence, penetrance, and rating, as described below) has been reviewed by a Canadian geneticist and by the leading geneticists of two global reinsurance companies. I have had further discussions with two of those doctors. I am not qualified to make these assumptions myself, and I have relied on but do not take responsibility for the assumptions set out below in table 1 as the consensus of those doctors. (This is a disclosure in accordance with paragraph 1610.02 of the Standards of Practice. It does not imply any objection to the assumptions.) From my discussion with the doctors and my knowledge of their expertise, I am comfortable using their work.

The references supporting the assumptions are shown in appendix 3, table 4.

### *4.2.1 Conditions Included*

There are over 5,000 genes that have been identified as relating to illnesses, and more are being discovered daily. In some cases, a single gene is associated with a disease; in other cases it is a combination of two or more genes. Only a few have been studied in sufficient detail that they could now be used effectively in underwriting. Table 1 shows the 13 conditions that were chosen for inclusion in my model. They are listed below with the abbreviations that appear in table 1 shown in parentheses.

1. Breast cancer (BRCA1 or 2);
2. Hypertrophic cardiomyopathy (HCM);
3. Dilated cardiomyopathy (DCM);
4. Arrhythmogenic right ventricular cardiomyopathy (ARVC);
5. Long QT syndrome (Long QT) ;
6. Brugada syndrome (Brugada);
7. Huntington's disease (Huntington);
8. Polycystic kidney disease (PKD);
9. Myotonic dystrophy (DM1 or 2);
10. Alzheimer's disease early onset – autosomal dominance (AEO);
11. Hereditary nonpolyposis colorectal cancer (HNPCC);
12. Marfan's syndrome (Marfan); and
13. Catecholaminergic polymorphic ventricular tachycardia (CPVT).

#### 4.2.2 *Prevalence*

The prevalence of the genetic marker in the Canadian population is expressed as 1 per n.

#### 4.2.3 *Penetrance*

Penetrance is defined as the probability that those with a particular gene will ultimately develop the disease. Not all with the genetic marker will develop the disease. Some will die first of other causes. Studies of penetrance are of limited duration; some may develop the disease at a higher age. Penetrance is expressed as a percentage of those who have the gene. The complement of penetrance is assumed to have standard mortality. It is important to note the division into these two groups, penetrance and its complement (referred to as substandard and standard), is an artifact of the model. An individual would not know in which group he or she belonged until symptoms appeared indicating membership in the substandard group.

The penetrance for BRCA1 or 2 is unique. It is believed that the probability of developing cancer is about 50% in females and zero in males. The probability used is 25%, and all of them are assumed to be female.

#### 4.2.4 *Rating*

Those with the disease will exhibit higher mortality. That higher mortality is expressed either as a percentage of standard or as a number of additional deaths per year. The rating is assumed to continue for life with one exception. Long QT is assumed to have excess mortality only until age 40 and be standard thereafter.

#### 4.2.5 *Predicted*

Some of those with the gene will be identified by the underwriting process from family history or early symptoms of the disease even if the results of the genetic test are not disclosed. This is expressed as a percentage. Thus if “predicted” is shown as 25%, it is assumed that 25% of those who test positive will be identified by the underwriter and rated, and 75% will obtain insurance at standard rates. Two chief underwriters and four doctors of Canadian insurance companies were involved in determining this predicted factor based on their knowledge and experience. For simplicity, the factors were chosen in 25% intervals.

Given that the family history questions refer to genetic disorders, one might think that the underwriting process would be very successful in identifying those who might carry a gene of concern. However, because the questions are limited to parents and siblings and because it is common to have only one or no siblings, there is often no sign of the disorder although the gene is present.

#### 4.2.6 *Tested*

This is the average age at testing. Typically the age range over which the testing is most often done is 10–15 years in length, but, to simplify the model, all are assumed to be tested at the average age.

#### 4.2.7 *Male*

This is the proportion who are male. Breast cancer is assumed to apply to females only. All other conditions are equally distributed by gender.

#### 4.2.8 Standard

This is the assumed number of years following testing for which the mortality of those who test positive is taken as standard, even if it is assumed that the disease will eventually emerge. Some have “standard” as 0; in that case it is assumed that higher mortality is applicable immediately from testing.

#### 4.2.9 Grading

This is the number of years over which mortality is assumed to increase from standard to the full rating. The grading is linear over this period. For example, if “grading” is 5, then mortality in the first year of the grading is assumed to be 10% of the rating, 30% in the second, 50% in the third, 70% in the fourth, 90% in the fifth, and 100% thereafter.

Condition	Prevalence	Penetrance	Rating	Predicted	Tested	Male	Standard	Grading
BRCA1 or 2	500	25%	200%	50%	30	0%	0	15
HTCM	500	69%	0.01	50%	25	50%	0	0
DCM	2700	75%	0.04	25%	35	50%	0	10
ARVCM	1250	75%	0.023	25%	25	50%	0	0
Long QT	3000	50%	0.001	25%	20	50%	0	0
Brugada	2000	75%	0.015	25%	30	50%	0	0
Huntington	20000	90%	1000%	50%	25	50%	5	10
PKD	1000	100%	500%	75%	30	50%	20	15
DM1 or 2	8000	75%	500%	50%	25	50%	15	10
ADEO	2427	100%	1000%	50%	30	50%	15	10
HNPCC	500	50%	300%	50%	30	50%	0	15
Marfan	5000	50%	500%	50%	20	50%	0	0
CPVT	10000	75%	1000%	25%	20	50%	0	5

### 4.3 Other Assumptions

#### 4.3.1 Population

The population is assumed to be 35 million.

#### 4.3.2 Testing Rate

It is assumed that those in the population who have one of the genes are tested at a uniform rate of 1/30 per year; thus, all will be tested over a generation. It is expected that there will be some event that precipitates the call for testing, such as a family member being diagnosed with the condition. Then siblings, children, and probably cousins and others would be encouraged to be tested to determine whether they also have a propensity for the condition.

#### 4.3.3 Seeking Insurance

Those who test positive will likely learn very soon, if they did not already know, that life insurance companies are prohibited from asking for the results of the genetic test, and that therefore they can obtain life insurance at a much more favourable price than most

others in the population. Most are likely to buy life insurance for any of a variety of reasons:

1. They have dependents whom they wish to protect.
2. They have no immediate need for life insurance, but they buy now in anticipation of future need, knowing that they will not be able to buy once symptoms emerge.
3. They see the price as low enough to constitute a good investment which will benefit their heirs.
4. They know that the insurance policy can be sold to a viatical company, by disclosing the results of the positive test for a significant amount of cash.

The proportion seeking insurance would certainly not be 100%, but it could be close. It would tend to increase as the prohibition gains publicity, particularly among interest groups supporting those with the diseases related to the positive tests. The assumption used is 75%.

#### 4.3.4 *Declined*

It is assumed that 5% of applicants are declined for reasons unrelated to the conditions under study. It is typical for an insurance company to decline 8–10% of applicants, but most of those are at much higher ages than the assumed age at testing.

#### 4.3.5 *Mortality*

Standard mortality is based on the CIA 97–04 table for non-smokers, age nearest birthday. The table is multiplied by factors taken from the most recently published study of the CIA, 72.7% for male select, 71.8% for female select, 80.1% for male ultimate, and 87.7% for female ultimate. To these mortality rates four years (2010 to 2014) of mortality improvement are applied using the CIA Committee on Life Insurance Financial Reporting scale. No future mortality improvement assumption is assumed.

It is assumed that those who test positive for the gene can be divided into two groups based on penetrance: those who develop the disease (the penetrance percentage, referred to as substandard) and those who do not (one minus the penetrance percentage, referred to as standard). It is important to draw this distinction because all who test positive will be motivated to buy insurance, but the extra mortality applies only to substandard. Of course, in reality one cannot know which group an individual will fall into, and not all in the standard group will exhibit standard mortality.

The extra mortality, when the rating is expressed as a multiple greater than 1, is obtained by multiplying the ultimate mortality rate from the table at the appropriate attained age by (rating – 1). The ultimate is used because the extra mortality concerns a condition that is not caught in underwriting and because the data for extra mortality are based on population studies rather than insured lives.

When the rating is a number less than 1, it is taken as a flat addition to standard mortality in all years.

However, the extra mortality may be less than implied by the two preceding paragraphs. See sections 4.2.8 and 4.2.9.

#### 4.3.6 *Lapse*

The lapse rate for all years is 0.5% for the substandard lives and 3% for standard lives. The lapse rate is higher for standard lives because they are more likely to abandon their insurance after no evidence of the disease for many years. It might be better to assume the lapse rate for standard starts out at 0.5% and gradually increases because individuals do not know initially whether they are in the standard or substandard group, but the flat assumption was used to simplify the model.

Because the extra mortality for Long QT syndrome is assumed to apply only before age 50, all are expected to lapse at age 50.

#### 4.3.7 *Conversion*

The representative policy (see below) is assumed to be convertible term to 65. Some of those who survive to age 65 will convert to a permanent policy at that time. For substandard the conversion rate is 100% for Alzheimer's because the individual will expect the condition to emerge soon if it has not already. For other conditions of the substandard group, the conversion rate is 75% because most conditions have an onset much earlier than age 65 and some of those who survive that long may think that they have overcome the disease. For the standard group the conversion rate for Alzheimer's is 50% because it is expected that none of these will have exhibited any symptoms of the disease. For other conditions of the standard group, it is assumed that none will convert.

#### 4.3.8 *Amount of Insurance*

It is expected, based on the bills currently being considered in legislatures, that the underwriters will have access to the results of genetic tests for amounts of insurance in excess of \$1 million. Therefore it is assumed that all who apply for insurance will seek \$1 million of insurance less the amount already in force, which is assumed to be \$100,000 on average. Thus, the assumed average size is \$900,000. Most of those who are tested are assumed to be relatively young, and it is likely that few of them have yet purchased individual insurance.

It is also possible that the threshold for allowing access to the results of genetic tests could be set much lower than \$1 million; the results shown below include two scenarios: the one described above because it is consistent with proposed legislation, and one with a much lower limit. The second scenario assumes a threshold of \$100,000. In that case, it is assumed that 20% of those who test positive already have at least \$100,000 of insurance and cannot apply for more.

#### 4.3.9 *Representative Policy*

It is assumed that all will purchase convertible term to 65, and if converting, term to 100 at age 65. I obtained approximate premium rates from term4sale.ca for a \$1 million policy. Policies are assumed to have annual premium frequency for simplicity.

#### 4.3.10 *Expenses*

It is assumed that the expense of underwriting, issue, and maintenance is \$1,500, all deducted at issue; that premium tax is 2.3%; and that commission and related marketing expenses are 120% of first-year premium for T65, 150% of first-year premium for T100, and 3% of renewal premium.

#### 4.3.11 Interest

The interest rate in all years is assumed to be 4%.

#### 4.3.12 Note for Valuation and Pricing

For valuation purposes, the Appointed Actuary is obligated by the Standards of Practice (paragraphs 1730.18–.23 and 2350.05 in particular) to take into account potential anti-selection, and pricing actuaries, although not obligated, are prudent to do so as well. It will be many years before there is any certainty around how many who have a certain gene are tested each year, how many of those who test positive will buy insurance, and how much insurance they will buy. In the meantime, if laws are passed to restrict access to the results of genetic tests, actuaries may need to assume rates of mortality that would result from the assumptions described above.

### 4.4 Method

#### 4.4.1 Simulation

The model simulates purchases of life insurance in one year by those who test positive for each condition. The number of policies purchased for a condition is the population multiplied by the prevalence, multiplied by the proportion tested each year, multiplied by the proportion not declined, multiplied by 1 less the proportion predicted in underwriting. This number of policies is divided into four groups based on the assumed penetrance and gender: male substandard, male standard, female substandard, and female standard. Each is assumed to buy a policy for the average amount of \$900,000 at the average age for testing.

The extra mortality for the substandard group continues to be standard for the number of years assumed, then it grades linearly to the full extra over the number of years assumed.

Each group is followed as a cohort of lives in a deterministic simulation to the end of the mortality table, or to age 50 for Long QT. The simulation notes, for each group and each condition and for each duration, the number of lives and amount of insurance in force, the number and amount of death claims, and the premium and expense for the year.

#### 4.4.2 Cost Sub-model

The cost sub-model estimates the impact on mortality cost due to a strengthening of reserves for the excess mortality over standard. The mortality cost is the present value of the cash outflows described in section 4.4.1.

The present value is calculated by

$$\bar{A}_x - P_x \ddot{a}_x$$

where  $P_x$  is net of expenses and premium taxes. Note that the present value will be positive for substandard, but it will be offset by a negative amount for standard because of the low lapse rate.

The total cost is compared to the total individual death claims for 2012, estimated by the CLHIA at \$3.5 billion for the life insurance industry in Canada. Note that the model compares a present value (of the additional claim costs) to recent cash claims from normal operations. The ratio between the two gives a good estimate of the long-term

impact of preventing underwriters from having access to genetic tests. The impact on cash claims will be much less initially.

It is valid to criticize the model for comparing a valuation strain on sale against a recent total of claims. That comparison is correct only in a steady state such that the total claims are the same each year. In fact, claims in a year have shown a slow upward trend for many years. Hence the number from the cost sub-model is a little on the high side. Nonetheless, comparing to recent total claims is helpful for understanding the scale of the impact. If the strain were small compared to recent claims, then one could conclude that the impact of a prohibition would be small.

#### 4.4.3 *Experience Sub-model*

The purpose of the experience sub-model is to estimate the change in the CIA mortality study that would occur if these policies were added. The CIA mortality study is important and is used as a significant factor by many companies in determining their mortality assumption for pricing. If mortality rates were to increase, or were expected to increase, because many substandard lives were being rated as standard, those increases would soon be reflected in general premium rates.

The model is built on two simulations. The first notes the attained age for exposure and death for each policy issued to those who test positive for one of the genetic markers as described in section 4.4.1.

The second is based on data from the CIA mortality study. The sales for each sex and age are the exposures for the first policy year in the study, for ages 18 and up. Because the study was for 2010–2011, the amounts are increased by 5% to allow for inflation. Because not all companies contribute to the CIA mortality study, the counts and amounts are further increased by 20%. All are treated as non-smokers; there is no distinction by smoking, preferred class, or policy type. The mortality rates are the same as those described above for standard. The lapse rates were inferred by determining the decrease in the in-force from the 2009–2010 policy year in the CIA study to the in-force for 2010–2011 at the same issue age but one duration later, after backing off standard mortality. Because of low exposure, the lapse rates for durations 31–40 were set to the simple average of the raw rates for 31–34.

The exposure and deaths for the two simulations were summarized for attained ages of 20 to 60 with duration not more than 40. These ages and durations are important for the purchase of insurance; those who test positive will typically be found more at the younger end of the range. The impact of the prohibition was measured as the increase in the A/E ratio (actually the simulated death claims divided by the expected death claims) for the sum of the two simulations over that for the normal simulation alone.

## 5 RESULTS OF MODEL RUNS

The cost model shows the present value of claim costs from those who tested positive in the year to be 12% of total claims. Table 2 shows the cost for each condition. Although the costs were calculated with the precision shown, the assumptions are uncertain enough that the cost should not be considered accurate to more than one significant digit.

<b>Table 2. Costs associated with each condition</b>	
<b>Condition</b>	<b>Cost</b>
BRCA1 or 2	5,363,834
HTCM	89,187,658
DCM	56,493,774
ARVCM	111,141,682
Long QT	1,315,716
Brugada	49,166,827
Huntington	2,571,615
PKD	24,030,962
DM1 or 2	3,694,493
ADEO	30,029,655
HNPCC	23,480,469
Marfan	3,133,402
CPVT	5,845,864
<b>Total</b>	<b>405,455,952</b>

The experience model finds that overall mortality experience for attained ages 20–60 would go up by 36% male and 58% female. The increase for females is much larger because the current in-force is significantly smaller for females than for males. (The increase over males and females combined is 44%. The unisex increase will be used in sensitivity testing, but the sex-distinct impact seems to be of more practical significance.)

For both sub-models, the results shown are forward-looking in the sense that the impact on cash flows will be fairly small initially, probably not even noticeable for a couple of years. Gradually the impact will become observable in traditional mortality studies. The impact will be observed much earlier in financial statements because valuation actuaries are required to take into account expected future experience. Pricing actuaries are wise to respond even earlier than valuation actuaries.

I ran a second scenario in which the threshold for prohibiting access to the results of genetic tests is \$100,000 of insurance. I assume that 25% of those who test positive either choose not to buy insurance or already have more than the threshold. I obtained another set of premium rates based on the purchase of \$100,000 of insurance. Other assumptions are unchanged. In this scenario the cost of claims goes up by only 1.8% compared to 12% in the main scenario. The experience model shows an increase of 3% for males and 8% for females.

It is important to note that because I am modelling a situation that has not happened in Canada and because the medical information is still emerging, the assumptions are not precise, and hence, the results cannot be taken as precise. Nonetheless, it is clear that the impact of a prohibition, as modelled here, is likely to be large, and may result in a very significant increase in term insurance premium rates.

## 6 CONCLUSIONS

The impact from not allowing underwriters to have access to the results of genetic tests known to the insurance applicant, at a threshold of \$1 million, is substantial; in my opinion it is much more than insurance companies could be expected to absorb without a response. That response is likely to be a very substantial increase in premium rates for term insurance. The rates for traditional permanent insurance would also rise but to a lesser extent.

As more genetic tests emerge for serious illnesses, it is likely that the impact will continue to grow.

On the other hand, if the threshold is set as low as \$100,000, the impact would be small enough that, in my opinion, the response from insurance companies would be limited and gradual. The impact is in the range of two to three years of mortality improvement, and because it would emerge over several years, it is unlikely that it would be observed against the general background of changes in mortality over time.

The implications for Canada are not necessarily exportable to other countries. Canadians buy mostly guaranteed products and a lot of term insurance (about 70% by face amount). If life insurance products were largely adjustable, any change in premium rates might be more gradual, as experience emerged. If, as is the case in some countries, life insurance is most often bought to cover a mortgage, there may be less impact because the amount of insurance is controlled by the balance of the mortgage.

## 7 LIMITATIONS

My assignment was to construct a simple model that would be understandable to most actuaries. Due to my keeping the model simple, the results may be less representative of reality than would be the case for a more robust model. Nonetheless I believe that although the magnitude of the results might vary, the conclusions would not change materially.

My comments on the limitations come under a number of headings: enhancements which might tend to increase the impact, enhancements which might tend to decrease the impact, enhancements with uncertain impact, and sensitivities of the results to assumptions with the current method. The enhancements suggested would move the model toward what might be considered a higher level of accuracy, but few of the enhancements could be considered cost-justified in the sense of having a large enough impact on the quantitative results that different qualitative conclusions would be reached.

### 7.1 Enhancements Increasing Impact

1. The assumptions for the genetic markers agreed to by the doctors were generally chosen to be at the lower end of any given ranges where such choices had to be made. Using neutral estimates would produce a larger impact.
2. As this model considers 13 out of thousands of genetic markers, more could be added. However, the list was chosen based on mortality impact and prevalence of information. The model considers only life insurance. The impact for critical illness insurance, and in some cases long-term care insurance, could be much larger. There could be an impact on disability income insurance as well.

## 7.2 Enhancements Decreasing Impact

The denominator for the ratios in the cost sub-model is from data two years old. An estimate of the values for 2014 is likely to be a little larger, and hence the ratios would be a little less.

## 7.3 Enhancements with Uncertain Impact

Rather than assuming all are tested at the same age, a range of ages might be used.

## 7.4 Sensitivities

Table 3 shows the impact on the cost model and the experience model from changing any one of a variety of assumptions. For this purpose, the male and female results of the experience model are combined into a unisex number. The results for the base case are shown on the line “Base assumptions”; these are the same as before but shown with more precision.

The last four scenarios are shown only for the cost sub-model because they are not relevant to the experience sub-model.

Assumption	Cost		Experience	
	Total	Increase	Total	Increase
Base assumptions	11.6%	n/a	43.8%	n/a
Insurance from 900K to 1M	12.9%	1.3%	48.4%	4.6%
Buyers from 100% to 80%	12.4%	0.8%	46.5%	2.8%
Testing from 1/30 to 1/40	8.7%	-2.9%	33.3%	-10.5%
Declined from 5% to 10%	11.0%	-0.6%	41.6%	-2.2%
Substd lapse rate from 0.5% to 0	12.9%	1.3%	47.6%	3.8%
Std lapse rate from 3% to 0.5%	11.7%	0.1%	43.9%	0.1%
Interest rate from 4% to 5%	9.6%	-2.0%		
Expenses from 1500 to 1000	11.5%	-0.1%		
Commission increased by 10%	11.7%	0.1%		
Premium rates increased by 10%	11.3%	-0.3%		

There is very strong sensitivity to assumptions that influence the amount of insurance bought: the average size of purchase, the proportion buying, the rate at which testing is done, and the proportion declined for other reasons. The sensitivity for the substandard lapse rate is also fairly strong, but there is very little sensitivity to the standard lapse rate.

Note that interest, expense, commission, and premium have an impact on the cost sub-model only. These factors are not relevant for the experience sub-model.

My testing indicates that the results are linear in most of the assumptions. That is, if the change in assumption were double the amount shown in table 3, then the change in the result would also be double. One caution: linearity may not hold over a very wide range of values.

**APPENDIX 1. REVIEW OF PAPER**

The CIA Report Project Management Team actively reviewed my assumptions and method as I built the model, made suggestions, and ultimately gave its approval.

The calculations were reviewed by another actuary by doing comparable cost calculations in GGY's Axis. The results were judged sufficiently close to mine to be acceptable.

## APPENDIX 2. PAPER BY MACDONALD AND YU

The paper [\*The Impact of Genetic Information on the Insurance Industry: Conclusions from the 'Bottom-Up' Modelling Programme\*](#) (Macdonald, A. S., and F. Yu, 2011, *ASTIN Bulletin*, 41: 343–376) has frequently been cited to give evidence that the impact of prohibiting the use of the results of genetic tests would be minimal on insurance companies. Because the conclusions of my model seem on the surface to be substantially at variance with Macdonald and Yu, some comment is required.

Macdonald and Yu's paper was intended to set a benchmark more than determine an absolute level. As more genes are taken into account and as the anti-selection grows, the results may be adjusted essentially linearly. In a sense my work is similar; I too set a benchmark relative to the assumptions mentioned in this report. As our knowledge expands to include more genes and as we develop a better understanding of the degree of anti-selection, my results can be adjusted essentially linearly.

Because Macdonald and Yu use a multi-state Markov model, it is difficult to compare most of the assumptions between models. For conditions that are in both, the assumptions seem to be in the same ballpark.

Macdonald and Yu include only six conditions compared to the 13 in my model. They include BRCA1 or 2, Huntington's, PKD, DM1 or 2, ADEO, and HNPCC. My work shows that these conditions account for only 22% of the total cost from the 13 conditions. The conditions present in mine but missing in the Macdonald/Yu model are primarily heart related, most of which are assumed to cause a large increase in mortality.

Macdonald and Yu assume that the policy purchased is of average size compared to more than double the average size in my model. Hence, the impact in my model is more than double their impact for this factor.

The two differences mentioned above account for my cost estimate being more than 12 times that of Macdonald and Yu. There are a number of other differences in assumptions and method; for most of these Macdonald and Yu appear to have made a choice that would yield a lower result than mine. They estimate the costs as under 1% of premium in most circumstances, while my estimate of the present value of the cost is 12% of death claims in a steady state.

I believe that Macdonald and Yu's model and my model are in substantial agreement. If either were to rerun his model using the assumptions of the other, our results would not be dramatically different. I conclude that it is vital to keep updating the list of genes covered by the model because the cost will increase as the number of genes useful in underwriting increases.

### APPENDIX 3. REFERENCES FOR GENETIC ASSUMPTIONS

Table 4 below contains references to documents supporting many of the genetic assumptions. These references were provided by the doctors who developed the assumptions.

Gene	Prevalence of gene mutation (1 in X)	Prevalence web reference	Penetrance of clinical expression given mutation positive	Penetrance web reference	Mortality impact (%)	Mortality impact (\$/K)	Mortality web reference
BRCA1 or 2	500		25%		200%		
Hypertrophic cardiomyopathy 1%/yr	500	<a href="http://www.ncbi.nlm.nih.gov/books/NBK1768/">http://www.ncbi.nlm.nih.gov/books/NBK1768/</a>	69%	<a href="http://www.ncbi.nlm.nih.gov/pubmed/9219008">http://www.ncbi.nlm.nih.gov/pubmed/9219008</a>		1% per year	
Dilated cardiomyopathy 4%/yr	2700	<a href="http://www.ncbi.nlm.nih.gov/books/NBK1309/">http://www.ncbi.nlm.nih.gov/books/NBK1309/</a>	75%	<a href="http://www.ncbi.nlm.nih.gov/pubmed/10400009">http://www.ncbi.nlm.nih.gov/pubmed/10400009</a>		4% / year	<a href="http://www.patient.co.uk/doctor/Dilated-Cardiomyopathies.htm#ref-2">http://www.patient.co.uk/doctor/Dilated-Cardiomyopathies.htm#ref-2</a>
Arrhythmogenic right ventricular cardiomyopathy 2.3%/yr	1250	<a href="http://www.ncbi.nlm.nih.gov/books/NBK1131/">http://www.ncbi.nlm.nih.gov/books/NBK1131/</a>	75%	<a href="http://www.ncbi.nlm.nih.gov/books/NBK1131/">http://www.ncbi.nlm.nih.gov/books/NBK1131/</a>		2.3% annually	
Long QT 500% mortality up to age 40, 100% thereafter	3000	<a href="http://www.ncbi.nlm.nih.gov/books/NBK1129/">http://www.ncbi.nlm.nih.gov/books/NBK1129/</a>	50%	<a href="http://www.ncbi.nlm.nih.gov/books/NBK1129/">http://www.ncbi.nlm.nih.gov/books/NBK1129/</a> <a href="http://www.ncbi.nlm.nih.gov/pubmed/9927399">http://www.ncbi.nlm.nih.gov/pubmed/9927399</a>		See comment in section 4.3.6	<a href="http://www.medscape.com/viewarticle/429964_2">http://www.medscape.com/viewarticle/429964_2</a>
Brugada 1.5%/yr	2000		75%			1.5% per year	<a href="http://onlinelibrary.wiley.com/doi/10.1038/npg.els.0003634/abstract">http://onlinelibrary.wiley.com/doi/10.1038/npg.els.0003634/abstract</a>

Huntington's	20000	<a href="http://www.ncbi.nlm.nih.gov/books/NBK1305/">http://www.ncbi.nlm.nih.gov/books/NBK1305/</a>	90%	<a href="http://www.ncbi.nlm.nih.gov/books/NBK1305/">http://www.ncbi.nlm.nih.gov/books/NBK1305/</a>	1000%		
Polycystic kidney disease	1000		100%		500%		
Myotonic dystrophy	8000	<a href="http://omim.org/entry/160900">http://omim.org/entry/160900</a>	75%	<a href="http://www.ncbi.nlm.nih.gov/books/NBK1165/">http://www.ncbi.nlm.nih.gov/books/NBK1165/</a>	500%		<a href="http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1767476/">http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1767476/</a>
Alzheimer's – autosomal dominance (100% penetrance bucket)	2427	<a href="http://omim.org/entry/104300">http://omim.org/entry/104300</a>	100%		1000%		
Colorectal cancer (HNPCC)	500	<a href="http://www.ncbi.nlm.nih.gov/books/NBK1211/">http://www.ncbi.nlm.nih.gov/books/NBK1211/</a>	50%	<a href="http://www.ncbi.nlm.nih.gov/books/NBK1211/">http://www.ncbi.nlm.nih.gov/books/NBK1211/</a>	300%		
Marfan	5000		50%		500%		
Catecholaminergic polymorphic ventricular tachycardia	10000	<a href="http://www.ncbi.nlm.nih.gov/books/NBK1289/">http://www.ncbi.nlm.nih.gov/books/NBK1289/</a>	75%	<a href="http://omim.org/entry/604772">http://omim.org/entry/604772</a>	1000%		<a href="http://cardiovascres.oxfordjournals.org/content/67/3/379.full">http://cardiovascres.oxfordjournals.org/content/67/3/379.full</a>