Estimating Adverse Selection Costs in a Market with Genetic Testing for Breast and Ovarian Cancer

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Abstract: Using data from the medical literature on age-specific and family-history specific incidence rates, we develop double-decrement models to evaluate the actuarial impact of a family history of breast cancer or ovarian cancer, and the impact of a positive test for the BRCA gene mutation. Increased forces of mortality are derived. It is found that females with some family histories of cancer and/or the presence of the BRCA mutation cannot be accepted at standard rates; depending on underwriting practice, most cases could be accepted at substandard rates. Then a Markov model is built to evaluate the likely effect of adverse selection resulting from women taking a genetic test without informing their insurer and consequently modifying their insurance purchase behavior. It is concluded that, at current testing rates, adverse selection should not be a major source of concern if companies apply strict underwriting rules, requesting cancer history and age at onset for all first degree relatives.

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1. Genetic Testing and the Fear of Adverse Selection

Adverse selection is a major source of concern for insurance companies because asymmetric information could result in huge underpricing. The recent developments in the Human Genome Project, while exciting from a biological standpoint, have further increased insurer fears about this issue. In many jurisdictions, insurers are not allowed to ask for results of genetic testing. Several years ago, the gene mutations which affect the likelihood of developing breast and ovarian cancer were discovered; commercial tests to detect the presence of these gene mutations are now available. Women who learn through genetic tests that they are at higher risk of death for breast or ovarian cancer may purchase more insurance, which to them looks inexpensive since it is priced at rates set for average risks. Women who learn they are at lower risk after a negative test may purchase less insurance. These two forces combine to increase the aggregate mortality of the purchasers of insurance. If insurers do not have access to the test results, they are unable to identify which women are at higher risk and which are not. They have to increase premiums for everyone, driving those at lower risk out of the pool. This creates a spiral of increasing prices and decreasing number of policies issued, that may threaten the financial solvency of the insurer.

The debate about insurer access to genetic screening information has industry representatives pointing to the risk of adverse selection. They advocate mandates which require that all test results provided to individuals also be made available to insurers. This insurers' request for a "level playing field" contrasts with opposite efforts by consumer groups to increase the privacy protection of genetic information. Consumers are concerned that test information may find its way to employers and result in employment and social discrimination. They fear that the use of genetic testing by insurers could result in the creation of a biological underclass of uninsurable individuals.

The issue is highly emotional and very political. Underlying conflicts in fundamental values have prompted legislators to regulate the use of genetic testing. Wisconsin was the first state to introduce a genetic testing law in 1992. Seventeen states now have enacted laws prohibiting insurers of different types from using genetic information in their underwriting decisions. As of early 1998, over 200 bills have been proposed in various state legislatures throughout the country that try, in one way or another, to limit insurers' access to and use of genetic information (Jones, 1998.)

Until very recently, the actuarial profession had not contributed much to the debate. This situation is changing as both the Institute of Actuaries and the Academy of Actuaries now have a genetic testing task force. The December 1998 issue of the North American Actuarial Journal is devoted entirely to the proceedings of the 1998 Bowles Symposium on genetic technology and underwriting. This issue contains a pioneering paper by Macdonald (1998), who uses a Markov model to estimate the impact of adverse selection. Macdonald does not refer to any particular disease or genetic test, and the parameters of his models are consequently estimated rather crudely. In this paper, we use Macdonald's seminal approach to quantify the impact of a family history of breast and/or ovarian cancer, and the impact of a positive test for the BRCA1 and BRCA2 gene mutations. In section 2, we restate the main results of a working paper (Lemaire et

al, 1999), that estimates the effect of family history and gene mutations on forces of mortality and on the costs of term insurance. In section 3, a Markov model is presented to evaluate the financial consequences of adverse selection. Section 4 concludes.

2. Increased Forces of Mortality in the Presence of a Family History of Breast or Ovarian Cancer, or in the Case of a BRCA Mutation.

This section summarizes recent work by Lemaire et al (1999), who provide an actuarial insight in the genetic testing debate by quantifying the impact of family history of breast cancer (BC), ovarian cancer (OC), and BRCA1/2 mutations on forces of mortality and on term life insurance costs.

The vast majority of BC and OC is the result of diet, lifestyle, environmental exposures, social interactions, and other factors, known and unknown. For instance, a late age at first childbirth and an early first menstruation slightly increase the likelihood of developing BC (Gail et al, 1989.) Women with more pregnancies, or longer use of oral contraceptives, or who underwent tubal litigation or hysterectomy, have a reduced probability to develop OC (Hartge et al, 1994.) However, some cancers are inherited. A small percentage of women (estimates range from one woman out of 833 to one out of 100) has a dominant mutated gene called BRCA1 or BRCA2 (Ford et al, 1995.) Women with a BRCA mutation are at extreme risk to develop BC or OC. Estimates of the probability to develop either of these cancers by age 70 are as high as 0.945 (Easton et al, 1995.)

Approximately one in nine women in the United States will develop BC in her lifetime; one in forty will die from the disease (American Cancer Society, 1992.) Probabilities to develop BC, as a function of age and family history, have been obtained by Claus et al. (1994). For instance, Table 1 indicates the predicted cumulative probability of BC for a woman who has a mother or sister affected, by age of onset of this first-degree relative. Onset is defined as the moment BC is diagnosed.

Table 1. Cumulative Probability of BC for a Woman who has One First-Degree Relative Affected with BC, by Age of Onset of the Affected Relative

AGE OF WOMAN	AGE OF ONSET IN AFFECTED RELATIVE					
	20-29	30-39	40-49	50-59	60-69	70-79
29	.007	.005	.003	.002	.002	.001
39	.025	.017	.012	.008	.006	.005
49	.062	.044	.032	.023	.018	.015
59	.116	.086	.64	.049	.040	.035
69	.171	.130	.101	.082	.070	.062
79	.211	.165	.132	.110	.096	.088

Survival probabilities exhibit exponential decay: the annual probability that a woman affected with BC will die from the disease is 0.036, irrespective of the time since diagnosis and age at onset (SEER Cancer Statistics Review, 1973-1995, and authors' calculations.)

OC is less prevalent, but deadlier: 1.8% of women will get the disease. The risk is multiplied by 5.4 in the presence of family history (Hartge et al, 1994). Survival rates are low, but improving. In 1973, only 59.9% of the women who developed OC survived the first year after diagnosis. The five-year survival rate was 36%, the 20-year rate 30.1%. In 1992, 78.3% of affected women survived their first year with OC (SEER Cancer Statistics Review, 1973-1995). Applying Taylor's separation method to OC survival rates (Taylor, 1977), the present-day five-year survival rate was estimated to be 50.9%, and the 20-year rate 36.3%.

Estimates of the *penetrance* (the percentage of those with the gene mutation who will develop BC) of BRCA1/2 vary from 56% to 85%, as there is a huge ethnic diversity in the sites of mutations. This wide range of estimates is typical of the medical cancer literature. We wish our estimates of increased forces of mortality and term premiums to be conservative from the insurer's perspective, i.e. our figures should err on the high side rather than on the low side. Therefore, an average penetrance of 65% was selected, as a conservative (Lowden, 1998). BRCA mutations not only increase the probability of developing BC, they also lead to earlier cancers. The Cancer and Steroid Hormone Study, 1980-82, estimated that the age at onset of BC for women without the mutation is normally distributed around a mean of 68.99 years and a standard deviation of 15.39. With BRCA mutations, the mean age at onset drops to 55.435, while the standard deviation is unaffected (Claus et al, 1994.)

Estimates of the likelihood to develop OC for a woman with a BRCA mutation vary widely, from 11% to 84%, depending on the type of mutation, the specific allele of BRCA1, and the population under study (Easton et al, 1995, Ford et al, 1994, Struewing et al, 1997). An average of 40% seems conservative.

Based on these medical estimates, an actuarial model was built to evaluate the increased death probability of a woman with a family history of BC or OC, or with a BRCA mutation, and the resulting increase in the net single premium of term insurance. First the survival probabilities for females given by the US Decennial Life Tables for 1989-91, published by the US Department of Health and Human Services, were fitted to a Makeham distribution. Then excess forces of mortality were calculated using a double-decrement model, and fitted with a quadratic function. Table 2 presents the μ-ratio, the ratio of the force of mortality with family history or a gene mutation to the baseline force of mortality, for a 30-year-old woman.

Table 2: μ-ratios with a family history of BC or OC, or with the BRCA mutation, for a 30-year-old woman (FDR = first-degree relative; SDR = second-degree relative; age at onset for BC: 20-29)

AGE	1 FDR-BC	1 SDR-BC	2 FDR-BC	1 FDR-OC	BRCA
31	1.0000	1.0000	1.0000	1.0302	1.0298
33	1.0345	1.0161	1.1051	1.1946	1.3543
35	1.0999	1.0465	1.3034	1.4011	1.8615
37	1.1822	1.0848	1.5518	1.5958	2.4323
39	1.2627	1.1225	1.7927	1.7350	2.9322
41	1.3385	1.1580	2.0159	1.7070	3.2351
43	1.3004	1.1391	1.9045	1.5812	2.9300
45	1.2976	1.1358	1.8999	1.6926	3.0133
47	1.3026	1.1362	1.9167	1.8143	3.1367
49	1.3174	1.1414	1.9586	1.9083	3.2691

It is seen that excess mortality can exceed 100% in some cases of family history of BC, and 225% for a woman with a BRCA mutation. While some females with a family history of cancer can be accepted at standard rates, others need to be quoted sub-standard rates. Depending on the underwriting policy of the company, females with the gene mutation can possibly be covered, at a rate incorporating a severe mortality surcharge. Note that the common assumption that a given disease simply multiplies forces of mortality by a multiplicative constant *(constant frailty hypothesis)* does not apply in the case of BC and OC. Table 3, extracted from Brackenridge and Elder (1998), shows that these mortality increases are comparable, or even higher, than increases resulting from common diseases.

Table 3. Mortality Ratios for Common Diseases

DISEASE	MEASUREMENT	μ-RATIO
Systolic blood pressure	158-167 (men)	2.06
Systolic blood pressure	178-187 (women)	2.78
Diabetes mellitus	Men	2.50
Build	40% overweight (women)	1.62
Build	60% overweight (men)	2.60
Epilepsy	All types	2.78
Alcoholism	5 drinks a day	3.00
Smoking	Average (men)	1.70
Smoking	40 cigarettes/day (men)	2.00
HIV	35-year-old male	50.00

3. Adverse Selection Costs

3.1. Markov Model

A continuous time, discrete state, Markov model is developed here to represent the actuarial environment of genetic screening. The model, shown in Figure 1, decomposes the natural history of an individual into a series of discrete states, that decompose term insurance purchasing and genetic testing decisions. At all times, every individual is assigned to one and only one state. Transitions from one state to another can occur at any time. At time 0, a woman may be in either State 1 or State 2. A woman in State 1 has not been tested from BRCA mutations and has no insurance. A woman in State 2 has not been tested, but has insurance. From State 1, seven future states are possible: She can remain untested and purchase insurance (State 2); she can test negative and buy insurance (State 4); she can test positive and buy insurance (State 5); she can test negative and remain uninsured (State 12); she can test positive and remain uninsured (State 13); she can die before getting tested or becoming insured (State 14); or she can remain untested and without insurance (State 1). Correspondingly, eleven future states are possible from State 2. Each state is represented by an ellipse in Figure 1. The possible transitions from one state to another are represented by arrows. Most transitions have cash flow implications. Transitions into States 2 and 4 through 11 imply insurance premium payments. We assumed that policies are purchased through net single premiums. Transitions from States 2 and 4 through 11 into State 14 imply insurance benefit payments.

One Markov model can be defined for every single age of the population under study at time 0, and for every possible family history. We shall consider three initial ages (30, 40, and 50) and four family histories (no BC or OC in the family; one FDR with OC; one FRD with BC, onset age 20 to 29; two FDR with BC, onset ages 20 to 29 for both), resulting in 12 subgroups or 12 different Markov models. At time zero, women are assumed to be unaffected by BC and OC.

We shall assume that the demand for insurance is inelastic to price. If this is not the case, the determination of the forces of transition would be very complex, as they are expected to change over time. If adverse selection occurs, more individuals at higher risk will purchase insurance, purchase insurance with larger benefit amounts, or hold onto that insurance for longer periods, increasing the financial exposure of insurance companies. That should drive insurance premiums up. In turn, this should decrease the transition probabilities into states that reflect insurance coverage (States 4 through 11), because higher costs will drive some women out of the market. To the extent that this increase in premiums preferentially drives out women who test negative or remain untested, which is what insurance companies fear, premiums will continue to rise in the spiral of adverse selection.

3.2. Thiele's Equations

Assume that the population is subdivided into subgroups, according to family history of OC or BC. Following the notation of Macdonald (1998), the (continuous time) force of transition at

time t from State j to State k, for subgroup i, is denoted $\mu_t^{i,j,k}$ (in the sequel superscript i will be omitted.)

For each State, we wish to calculate, under a variety of assumptions, the actuarial present value of future payments, incorporating mortality and interest. This expected value, called the benefit reserve, is a liability to the company. The insurer is indifferent between paying the benefit reserve and insuring the risk. As the reserves for the various States are dependent, their values can only be found by solving a set of differential equations, Thiele's equations for benefit reserves. One differential equation can be written for each State for which there is an outward transition. The equation for State *j*, is written:

$$\frac{d}{dt}V_t^{(1)j} = \delta_t V_t^{(1)j} - \sum_{k \neq j}(b_t^{jk} + V_t^{(1)k} - V_t^{(1)j})\mu_t^{jk}$$

where

$V_t^{(1)j}$ Benefit reserve for State *j* at time *t*

$V_t^{(1)k}$ Benefit reserve for State *k* at time *t*

δ_t Force of interest at time t

b_t^{jk} Payment due upon transition from State *j* to State *k*.

μ_t^{jk} Force of transition from State *j* to State *k* at time *t*.

The interpretation of the differential equation is as follows: at all times, the reserve increases through interest accrual. Upon a transition from j to k, a benefit b_t^{jk} might be paid. In our case this would only happen for a transition into State 14, when a death benefit is to be paid to an insured individual. Switching from State j to State k also implies the release of the reserve for State j and acquiring the reserve for State k.

This set of differential equations can be solved backward recursively, using the boundary conditions $V_{t^*}^{(1)j} = 0$, where t^* is the ending time for the period under consideration. In other words, the insurance company, at time t^*, needs no longer to hold funds aside for this policy, because the policy term and the corresponding financial obligations have ended.

The benefit reserve is the expected value of future payments. Higher order moments can also be computed. Norberg (1995) has shown that $V_t^{(q)j}$ the moment of order q about the origin for State j, is the solution of the set of differential equations

$$\frac{d}{dt}V_t^{(q)j} = (q\delta_t + \mu_t^{j\cdot})V_t^{(q)j} - \sum_{k \neq j}\mu_t^{jk}\sum_{r=0}^{q}\binom{q}{r}(b_t^{jk})^r V_t^{(q-r)k}$$

where

$$\mu_t^{j\cdot} = \sum_{k \neq j} \mu_{jk}$$

Variances, coefficients of variations, and Pearson skewness coefficients can then easily be calculated from these moments.

We are interested in the *implicit cost of adverse selection,* the cost of adverse selection and genetic testing in the market. To calculate this, we first solve the differential equations, assuming no allowed use of genetic testing by insurers. Women flow through the system at the base case transition rates and experience mortality at rates based only on their family history, the information which the insurer uses for pricing purposes. This gives us the expected present value of costs in the "no genetic testing" case. We then solve the same differential equations, assuming genetic testing and allowing women to flow through the system at the same base case transition rates. However, the transition rates will now be mortality rates corresponding to the woman's BRCA status, if she is tested (baseline mortality if negative, BRCA mortality if positive,) and to her family history, if she remains untested. This gives us the expected present value of costs, in the genetic testing – full information case. The ratio of these two measures yields the implicit cost of adverse selection, the ratio of what the true risk is to what is claimed and charged.

$$Implicit\ Cost\ of\ Adverse\ Selection = \frac{EV(full\ information)}{EV(allowable\ information)}$$

3.3. Initial Behavioral Assumptions

Estimates are necessary for each force of transition. Transitions into State 14 (Dead) reflect mortality, which we estimate using the procedure outlined in section 2. These mortality rates differ by age, family history and BRCA status. The other transitions involve a combination of testing behavior, test results and insurance purchasing behavior. Reasonable base case estimates for these transitions will be assumed, erring on the side of cautiousness

The following behavioral transition intensities were selected for our benchmark calculations.

- *Rate of insurance purchase* μ_x^{12}. From industry figures (ACLI, 1997), this rate was estimated to be 5%.

- *Rate of genetic testing r.* Very few women get tested presently: only 250 women have been tested at the University of Pennsylvania since the test became available, late in 1996. The test is very expensive ($2,400) and not covered by medical insurance. The price of the test is not expected to decrease dramatically, as one laboratory owns the patent. To be on the conservative side, a rate of 5% was selected.

- *Force of interest.* Denoted as δ, we assume a force of interest of 5%.

- *Test results.* The probability p that a test result will be positive depends on the sub-group: a woman with two first-degree relatives affected by BC is much more likely to have the BRCA gene than a woman with no family history of the disease. The value of this probability is found by introducing the constraint that expected benefits need to be equal in the two cases: (1) no genetic testing; and (2) women get tested but their insurance purchase decisions are not affected. This results in p=0.005 for a woman with no family history, p=0.08 for a woman with one first-degree relative affected with OC, age at onset unknown. For a woman with one first-degree relative affected with BC, onset age 20-29, p=0.15; for a woman with two first-degree relatives affected with BC, both with onset age 20-29, p=0.40.

- *Insurance benefits.* The baseline amount of term insurance is assumed to be \$1. A woman buying "less insurance" always reduces her benefit amount from \$1 to \$0.50; for a woman buying "more insurance," we consider increased benefit amounts of \$2, \$4, or \$10. A woman lapsing her insurance policy ceases to be insured.

- *Insurance purchase probabilities.* Insurance decisions are assumed to occur shortly after the test result is provided. Benchmark probabilities were selected as follows:

° If uninsured and test positive:	P(buy insurance) = 0.25
	P(not buy) = 0.75
° If uninsured and test negative:	P(buy insurance) = 0.03
	P(not buy) = 0.97
° If insured and test positive:	P(more insurance) = 0.27
	P(same insurance) = 0.70
	P(less insurance) = 0.02
	P(lapse policy) = 0.01
° If insured and test negative:	P(more insurance) = 0.02
	P(same insurance) = 0.75
	P(less insurance) = 0.18
	P(lapse policy) = 0.05

Transition rates are then obtained by multiplying the appropriate rates and probabilities. For instance, $\mu_x^{I,12} = r*(1-p)*0.97$.

3.4. Costs of Adverse Selection, by Family History

The model was run, using the initial behavioral assumptions, for the four family histories under consideration.

Table 4. Implicit costs of adverse selection for a Woman with No Family History of BC or OC, Insured at Onset.

		TERM			
Age	Increased benefit	5	10	15	20
30	2	1.0006	1.0021	1.0039	1.0054
	4	1.0009	1.0030	1.0056	1.0077
	10	1.0016	1.0057	1.0106	1.0145
40	2	1.0008	1.0026	1.0042	1.0046
	4	1.0012	1.0037	1.0060	1.0066
	10	1.0023	1.0070	1.0112	1.0123
50	2	1.0004	1.0013	1.0020	1.0021
	4	1.0006	1.0019	1.0029	1.0030
	10	1.0012	1.0035	1.0054	1.0056

Adverse selection only has a minuscule impact for a woman who has no family history of BC or OC. This is due to the fact that the probability of having the gene, for a woman with this particular family history, is only 0.005. Insurance companies should not be overly concerned with restrictions of the use of genetic testing information for women with no family history.

Table 5. Implicit costs of adverse selection for a Woman with One First-Degree Relative Affected with OC, Age at Onset: Unknown, Insured at Onset.

		TERM			
Age	Increased benefit	5	10	15	20
30	2	1.0023	1.0107	1.0194	1.0237
	4	1.0057	1.0226	1.0409	1.0524
	10	1.0158	1.0563	1.0999	1.1299
40	2	1.0027	1.0115	1.0186	1.0182
	4	1.0073	1.0256	1.0409	1.0425
	10	1.0208	1.0655	1.1022	1.1079
50	2	0.9995	1.0002	1.0011	1.0018
	4	1.0019	1.0072	1.0119	1.0131
	10	1.0089	1.0271	1.0415	1.0435

For a woman with one first-degree relative with OC, potential adverse selection costs are non-negligible, as they exceed 10% in some cases. We reach a similar conclusion as Macdonald (1998): the main costs of adverse selection do not result from the lapsing behavior of the women who test negative; it results from the women who select very high benefit levels following a positive test.

The longer the duration of the policy, the higher the adverse selection costs. Obviously, longer terms give women more opportunities to get tested and make insurance-related decisions, and more time to develop OC or BC and die.

Adverse selection costs are always higher for an initial age of 40. Women who are cancer-free at the age of 30 are unlikely to develop BC or OC before the age of 50, and even more unlikely to die from the disease during that 20-year period. Women who are 50 at time zero are more prone to develop cancer than women who are 10 years younger, but it is also much more probable that they die from other causes, so that the adverse selection cost, which is a ratio, is lower.

Table 6. Implicit costs of adverse selection for a Woman with One First-Degree Relative Affected with BC, Age at Onset: 20-29, Insured at Onset.

		TERM			
Age	Increased benefit	5	10	15	20
30	2	1.0065	1.0177	1.0294	1.0386
	4	1.0126	1.0369	1.0626	1.0825
	10	1.0300	1.0893	1.1495	1.1935
40	2	1.0144	1.0380	1.0544	1.0523
	4	1.0233	1.0632	1.0925	1.0928
	10	1.0489	1.1322	1.1924	1.1949
50	2	1.0079	1.0197	1.0271	1.0250
	4	1.0128	1.0331	1.0468	1.0449
	10	1.0269	1.0698	1.0985	1.0952

Table 7. Implicit costs of adverse selection for a Woman with Two First-Degree Relatives Affected with BC, Ages at Onset: 20-29, Insured at Onset

		TERM			
Age	Increased benefit	5	10	15	20
30	2	1.0089	1.0168	1.0237	1.0294
	4	1.0186	1.0421	1.0631	1.0787
	10	1.0445	1.1034	1.1510	1.1819
40	2	1.0259	1.0578	1.0749	1.0667
	4	1.0419	1.0956	1.1264	1.1183
	10	1.0848	1.1873	1.2413	1.2269
50	2	1.0143	1.0296	1.0365	1.0307
	4	1.0234	1.0510	1.0652	1.0584
	10	1.0479	1.1030	1.1298	1.1171

Adverse selection costs are the highest in the last two cases, reaching 20% in some cases. There is a substantial probability that the gene mutation is present in the family in the two cases; consequently the result of the test provides a lot of information.

3.5. Introducing Fraud.

The above tables assumed that women reported their family history truthfully. Now let us consider the impact of fraud. Assume a woman with 2 FDR with BC reports no family history, and the insurer fails to detect this fraud.

Table 8. Implicit costs of adverse selection for a Woman with Two First-Degree Relatives Affected with BC, Ages at Onset: 20-29, Insured at Onset, Who Claims no Family History of BC or OC.

		TERM			
Age	Increased benefit	5	10	15	20
30	2	1.2356	1.5149	1.7253	1.8490
	4	1.2813	1.6364	1.9312	2.1263
	10	1.4170	1.9933	2.5300	2.9260
40	2	1.2536	1.5176	1.6803	1.6947
	4	1.3076	1.6565	1.8963	1.9461
	10	1.4678	2.0639	2.5243	2.6703
50	2	1.1469	1.2909	1.3704	1.3669
	4	1.1878	1.3865	1.5138	1.5339
	10	1.3090	1.6670	1.9305	2.0151

Adverse selection costs are huge in this case. It is of crucial importance for insurance carriers to request detailed family information (including age at onset) during the underwriting process, and to investigate the applicant's statements vigorously.

3.6. Very Conservative assumptions.

The benchmark behavioral hypotheses specified in 3.3. assumed a high degree of inertia. Most women did not change their insurance purchase behavior following the results of the test. We now assume major behavioral changes, decreasing the probability of keeping the same amount of insurance by 50% and adding 50% to each probability which affects the adverse selection.

° If insured and test positive: P(more insurance) = 0.77
P(same insurance) = 0.20

° If insured and test negative: P(less insurance) = 0.67
P(same insurance) = 0.25

Table 9 shows adverse selection costs under this set of conservative assumptions, for a woman with one FDR with BC. As expected, the adverse selection costs are sizable.

Table 9. Implicit costs of adverse selection for a Woman with One First-Degree Relative Affected with BC, Age at Onset: 20-29, Insured at Onset.

		TERM			
Age	Increased Benefit	5	10	15	20
30	2	1.0151	1.0466	1.0810	1.1082
	4	1.0324	1.0998	1.1700	1.2226
	10	1.0786	1.2269	1.3642	1.4539
40	2	1.0256	1.0722	1.1091	1.1135
	4	1.0510	1.1419	1.2114	1.2193
	10	1.1184	1.3082	1.4340	1.4329
50	2	1.0141	1.0380	1.0556	1.0551
	4	1.0280	1.0751	1.1084	1.1071
	10	1.0650	1.1636	1.2238	1.2127

3.7. Women uninsured at time zero.

Finally, adverse selection costs are estimated for a woman uninsured at time zero (i.e., in State 1 at t=0). As shown in table 10, costs are higher from State 1 than from State 2. This conclusion was also reached by Macdonald (1998): the population currently uninsured may lead to higher costs than the insured population.

Table 10. Implicit costs of adverse selection for a Woman with One First-Degree Relative Affected with BC, Age at Onset: 20-29, Uninsured at Onset.

		TERM			
Age	Increased Benefit	5	10	15	20
30	2	1.0194	1.0372	1.0515	1.0606
	4	1.0413	1.0800	1.1108	1.1299
	10	1.0982	1.1880	1.2558	1.2942
40	2	1.0339	1.0620	1.0756	1.0664
	4	1.0654	1.1174	1.1426	1.1283
	10	1.1475	1.2568	1.3061	1.2753
50	2	1.0187	1.0327	1.0385	1.0324
	4	1.0360	1.0623	1.0735	1.0630
	10	1.0812	1.1370	1.1587	1.1358

3.8. Coefficient of variation; skewness coefficient

All preceding results were obtained comparing mean costs under a variety of assumptions. Table 11 shows standard deviations, coefficients of variation, and skewness coefficients, in one particular case. An increased benefit of 10 was selected to maximize the increase of the higher moments. Table 11 shows that the impact of genetic testing on second and third moments is quite limited.

Table 11. Mean, standard deviation, coefficient of variation, and skewness coefficient of the present value of claims, for a Woman with 1 First-Degree Relative affected with BC, Age at onset 20-29, Insured at Onset. Increased benefit = 10.

	TERM→	5		10		15		20	
		No GT	GT	No GT	GT	No GT	GT	No GT	GT
AGE									
30	Mean	.003764	.003877	.008102	.008826	.013263	.015246	.019427	.023187
	S.D.	.070639	.076955	.109946	.131328	.142733	.180096	.170492	.220747
	C.V.	18.7661	19.8490	13.5695	14.8797	10.7612	11.8121	8.7759	9.5204
	Skewness	53.1344	58.0937	39.0313	38.8954	29.4323	27.0411	23.0061	20.1617
40	Mean	.007868	.008252	.017191	.019462	.028389	.033851	.041578	.049684
	S.D.	.102027	.115431	.159824	.200794	.207970	.272270	.247757	.321376
	C.V.	12.9679	13.9875	9.2972	10.3170	7.3258	8.0431	5.9588	6.4684
	Skewness	36.8666	41.0981	26.9017	26.3122	20.2025	18.2458	15.8148	14.1743
50	Mean	.018122	.018609	.039056	.041727	.063555	.069814	.091825	.100566
	S.D.	.154149	.165657	.238732	.273345	.307314	.360431	.361884	.420125
	C.V.	8.5061	8.9019	6.1204	6.5508	4.8354	5.1627	3.9410	4.1776
	Skewness	24.3324	26.2882	17.9645	18.0224	13.6399	13.0250	10.8068	10.2973

(No GT = use of genetic tests results not allowed)

4. Conclusions

All preceding results have been obtained under conservative assumptions. Also our calculations assume that genetic testing leads to no medical benefits in the form of improved risk reduction. There is some hope that women found to carry BRCA mutations can reduce their risk of BC mortality by increased mammogram surveillance, prophylactic mastectomy, or chemoprevention with tamoxifen. Therefore we believe that the figures in all tables of section are cautious upper bounds of adverse selection costs. The main conclusion is that these costs are certainly manageable for the insurance industry. Only in a few cases (20-year term, family history of BC with early age at onset) does the cost exceed 10%. The average cost in a portfolio is likely to be way below 10%. So the excess cost due to adverse selection is likely to be compensated by the overall decrease in mortality rates (a factor not introduced in our calculations) that stands at 0.5% per year these days.

This conclusion only holds if companies apply very tight underwriting standards. In the application process, prospective insureds need to provide the detailed family history of all their first-degree relatives, with ages at onset of any cancer. Applicants' statements need to be carefully checked by underwriters. If companies fail to correctly identify the family history of the applicant, adverse selection costs could become unbearable.

References

ACLI (1997). Life Insurance Fact Book

American Cancer Society (1992). Cancer facts and figures. Atlanta.

R.D.C. Brackenridge and W. J. Elder (1998). Medical Selection of Life Risks. Macmillan Reference Ltd.

Claus, E., Risch, N., and Thompson, W.D. (1994). Autosomal Dominant Inheritance of Early-Onset Breast Cancer. *Cancer*, 73, 643-651.

Easton, D., Ford F., Bishop, T., and the Breast Cancer Linkage Consortium (1995). Breast and Ovarian Cancer Incidence in BRCA1-Mutation Carriers. *American J. of Human Genetics*, 56, 265-271.

Ford, D., Easton, D., Bishop, D. *et al* (1994). Risks of cancers in BRCA1 mutation carriers. *Lancet*, 343, 692-695.

Ford, D., Easton, D., and Peto, J. (1995). Estimates of the Gene Frequency of BRCA1 and Its Contribution to Breast and Ovarian Cancer Incidence. *American J. of Human Genetics*, 57, 1457-1462.

Gail, M., Brinton, L., Byar, D., *et al* (1989). Projecting Individualized Probabilities of Developing Breast Cancer for White Females Who Are Being Examined Annually. *J. of the National Cancer Institute*, 81, 1879-1886.

Hartge, P., Whittemore, A., Intyre, J., McGowan, L., Cramer, D., and the Collaborative Ovarian Cancer Group (1994). Rates and Risks of Ovarian Cancer in Subgroups of White Women in the United States. *Obstetrics and Gybecology,* 84, 760-764.

Jones, C. (1998). The Current State of Genetic Testing: An Insurance Industry Perspective on the Rush to Legislate. North American Actuarial Journal, 2.

Lemaire, J., Subramanian, K., Armstrong, K., and Asch, D. (1999). Pricing Term Insurance in the Presence of a Family History of Breast or Ovarian Cancer. Submitted.

Lowden, J.A. (1998). The Current State of Genetic Testing in Life Insurance, Genetic Testing: Implications for Insurance Symposium Proceedings, The Actuarial Foundation, 19-28.

MacDonald, A.S. (1998). Modeling the Impact of Genetics on Insurance. North American Actuarial Journal, 2.

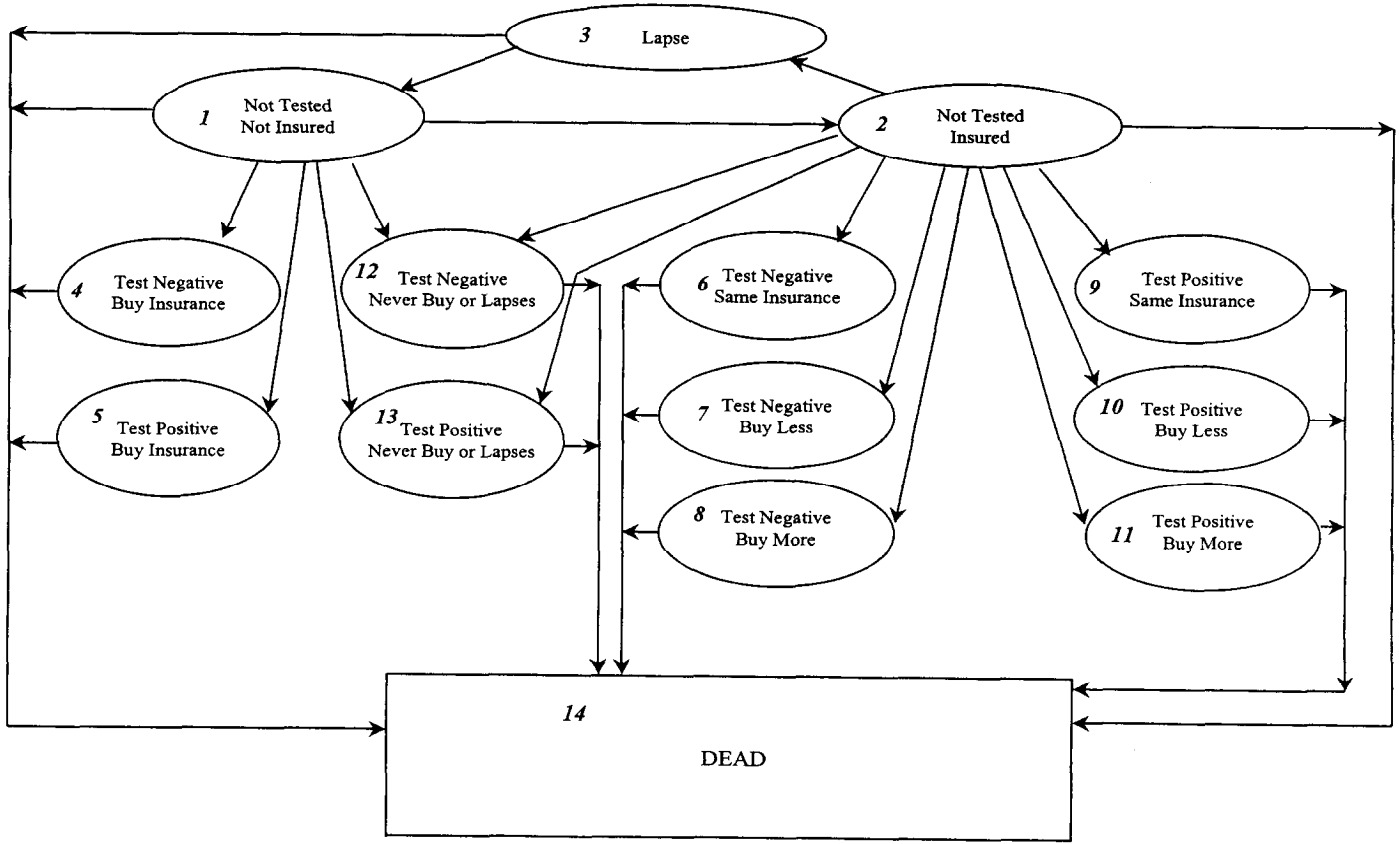
Norberg, R. (1995). Differential equations for moments of present values in life insurance. Insurance: Mathematics and Economics, 17, 171-180.

SEER Cancer Statistics Review 1973–1995, National Cancer Institute, National Institutes of Health.

Struewing, J., Hartge, P., Wacholder, S. *et al* (1997). The risk of cancer associated with specific mutations of BRCA1 and BRCA2 among Ashkenazi Jews. *New England J. of Medicine,* 336, 1401-1408.

Taylor, G. (1977). Separation of inflation and other effects from the distribution of non life insurance claim delays. *ASTIN Bulletin,* 9, 219-230.

MARKOV MODEL: HERITABLE BREAST CANCER AND ADVERSE SELECTION



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