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Estimating Adverse Selection Costs From Genetic Testing for Breast and Ovarian Cancer: The Case of Life Insurance

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Abstract

Genetic testing is a concern for insurers if they cannot use test results in underwriting. We model adverse selection in an insurance market with genetic testing for breast and ovarian cancer. Increased forces of mortality resulting from a family history of cancer or a positive test for a BRCA mutation are calculated. Using a Markov model, we estimate costs of adverse selection, assuming various testing and insurance purchase behaviors. Adverse selection should be controllable if companies apply strict underwriting rules, requesting cancer history and onset age for all first-degree relatives. If insurers fail to correctly identify the family history of the application and use it in pricing, adverse selection costs could become unbearable.

Genetic Testing and the Fear of Adverse Selection

Adverse selection can be defined as the process by which prospective policyholders may gain financial advantage through insurance purchase decisions based on risk characteristics known to them, but unknown and not revealed to the insurer. It is a source of concern for insurance companies because it could result in underpricing. Recent developments in the Human Genome Project, while offering medical prom-

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ise, have further increased insurer fears about this issue. Indeed, in many jurisdictions, insurers are not allowed to ask for results of genetic testing.

Several years ago, two gene mutations that affect the likelihood of developing breast and ovarian cancer were discovered. Commercial tests to detect the presence of these 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 life 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 life insurance. These two forces combine to increase the aggregate mortality of the insurance purchasers. 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 a decreasing number of policies issued, which 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 that would require that all test results provided to individuals also be made available to insurers. These insurers' request for a "level playing field" contrasts with efforts by consumer groups to increase the privacy protection of genetic information. Consumers are concerned that test information may find its way to employers or 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. While risk classification is unchallenged in some lines of business (smokers/nonsmokers in life insurance, distance from nearest fire station in homeowners' insurance), the use of genetic tests comes into conflict with social and moral concerns of society in the 1990s. These concerns have prompted legislators to regulate the use of genetic testing. Wisconsin was the first state to introduce a genetic testing law in 1992. Thirty-four states have now enacted laws prohibiting insurers of different types from using genetic information in their underwriting decisions. As of early 1998, more than 200 bills have been proposed in various state legislatures throughout the country that try to limit insurers' access to and use of genetic information (Jones, 1999). In March 1999, Sen. Olympia Snowe (R-ME) introduced a bill, backed by President Clinton and included in the GOP Patients' Bill of Rights, that would block insurers from denying coverage or setting premiums based on genetic information or family history (Government Relations Weekly Update, 1999).

Until very recently, the actuarial profession had not contributed much to the debate. This situation is changing as both the Institute and the Academy of Actuaries now have genetic testing task forces. The Academy Task Force published a brief on the issue in 1998 (American Academy of Actuaries, 1998). The January 1999 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 an overview article by Brockett, MacMinn, and Carter (1999), a perspective on legal issues by Hall (1999) and on regulation by Oakley (1999) and Jones (1999). Rothenberg (1999) considers the societal impact of genetic testing.

Pioneering actuarial research by Macdonald (1997, 1999) uses a Markov model to estimate the impact of adverse selection. Macdonald does not refer to any particular disease or genetic test. We consider here a life insurance market and the effects of breast and ovarian cancer on term insurance costs. We discuss consumer and insurer perspectives about genetic testing, specifically discrimination and adverse selection issues. We then restate the main results of Lemaire et al. (2000), who estimate the effects of family history and gene mutations on forces of mortality and on the costs of term life insurance. We present a continuous-time, discrete-state Markov model, adapting Macdonald's approach, to estimate adverse selection costs resulting from BRCA mutation testing and its subsequent effects on life insurance purchase. We conclude with life insurance underwriting recommendations.

Note that our conclusions are valid only for life insurance contracts. Life insurance underwriting is performed only once, renewal is automatically guaranteed without further evidence of insurability, and premiums are usually guaranteed for a long period. Health insurance would require another model, incorporating continual updates of the insured's health status and subsequent premium adjustments.

INFORMATION CONCERNS

Privacy and Discrimination

The extremely personal nature of genetic testing information has driven the movement toward regulation of its use. Employees fear that their employers could obtain this information without their permission and that higher-risk individuals may face job termination. In several industries, though, periodic genetic screening is administered for those employees exposed to gene-altering chemicals.

The Health Insurance Portability and Accountability Act of 1996 bans characterizing an employee's specific genetic testing information as a pre-existing condition, for group health insurance, in the absence of a diagnosis for that particular disease. In the private insurance market, a similar consumer protection does not exist at the federal level. Several states have banned companies from demanding that applicants take a genetic test as a condition for insurance and prohibited them from requesting existing test results during the underwriting process. In several European countries, insurer associations have adopted a voluntary moratorium on the use of genetic test results, at least for insured benefits under a specified amount.

Asymmetric Information

Insurers question why they are not allowed access to genetic information. They argue that customers should be charged rates commensurate with their risk level. Insurers are allowed to consider an applicant's family history of disease in their underwriting. If family history, which is a proxy for genetic information, is allowed, why not the more precise genetic screening results, if available?

Insurers argue that in some cases genetic test information can improve individuals' chances of obtaining insurance. For instance, genetic screening for Huntington's disease could represent a mutually beneficial use of genetic information for consumers and insurers. Reporting a family history of this fatal neurological disorder renders an applicant uninsurable before the age of 40. For an individual with a family history

of Huntington's, survival past that age without symptoms implies that the mutation is exceedingly unlikely; insurance can then be sold. Insurers point out that if a genetic test proves that the genetic mutation is not present in a given individual, he could be insurable at a much younger age. Insurers argue that when genetic test information is excluded from underwriting, healthy risks, necessary for pooling and diversification, are unfairly shut out of the private insurance market.

Adverse Selection

Onset of the HIV epidemic and its effect on the life insurance underwriting process demonstrates how insurers react to potential adverse selection. Because of the huge mortality differential between affected and unaffected individuals, potentially catastrophic losses existed for those life insurance policies already in force, for which renewal was automatic without future evidence of insurability. In 1985, California passed legislation that banned insurers from using the HIV antibody test for new policies. Several jurisdictions, including Washington, D.C., followed that example and banned the use of blood testing for HIV in underwriting. Because of the high rate of AIDS in the District of Columbia, most life insurers pulled out of the region. Other insurers used proxy tests that resulted in a large number of false positives. This resulted in such an uproar by the public that all of the jurisdictions who had introduced testing restrictions rescinded them. After much debate and litigation, the insurance industry ended up winning these legislative battles, at the price of some important compromises (the use of sexual orientation in the underwriting process is now prohibited, for instance). Insurers adopted the following policy: to minimize the AIDS-risk exposure on new life insurance policies, applications were amended to ask whether the customer "had ever tested positive for AIDS." However, with this wording, HIV-positive individuals who had not developed AIDS could truthfully answer no to this question. Insurers had to change the wording to ask if the customer "had ever tested positive for antibodies to HIV." Blood tests are administered for large benefit amounts. HIV-positive and AIDS-afflicted individuals are usually uninsurable in the private insurance market, although one enterprising insurer has recently begun selling whole life insurance specifically to HIV-positive applicants (Koco, 1997).

Insurers responded to the threat of adverse selection from HIV infection with additional application questions and follow-up testing; regulatory restrictions currently prevent similar measures from being adopted for genetic testing. Before insurers become too fearful about potential adverse selection problems, they should consider the likely testing behavior of individuals, as these screening costs are non-negligible. Also, since the use of extensive family history of disease is allowable in underwriting, the amount of incremental information provided by specific genetic tests should be evaluated.

INCREASED FORCES OF MORTALITY

This section summarizes work by Lemaire et al. (2000), 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 ligation or hysterectomy, have a reduced probability of developing OC (Hartge et al., 1994). However, about 6 percent of breast and ovarian cancers are inherited (Claus et al., 1998). A small percentage of women (estimates range from 1 woman out of 833 to 2.3 percent in some ethnic groups) has a mutated dominant 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 of developing either of these cancers by age 70 are as high as .945, although this estimate was obtained from a selected group of women (Easton et al., 1995). Based on this high estimate, Engman and Pinkham (1996) estimate that the presence of a BRCA mutation in a 30-year-old woman may reduce her life expectancy by 9.3 years.

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 of developing 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 (FDR). Onset is defined as the moment BC is diagnosed. Survival probabilities exhibit exponential decay: the annual probability that a woman affected with BC will die from the disease is .036, irrespective of the time since diagnosis and age at onset (SEER Cancer Statistics Review, 1973-1995, and authors' calculations).

TABLE 1
Cumulative Probability of BC for a Woman Who Has One FDR Affected With BC, by Age at Onset of the Affected Relative

Age of	Age of Onset of Affected Relative					
Woman	20-29	30-39	40-49	50-59	60-69	70-79
29	0.007	0.005	0.003	0.002	0.002	0.001
39	0.025	0.017	0.012	0.008	0.006	0.005
49	0.062	0.044	0.032	0.023	0.018	0.015
59	0.116	0.086	0.064	0.049	0.040	0.035
69	0.171	0.130	0.101	0.082	0.070	0.062
79	0.211	0.165	0.132	0.110	0.096	0.088

OC is less prevalent, but deadlier: 1.79 percent 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 percent of the women who developed OC survived the first year after diagnosis. The five-year survival rate was 36 percent, and the 20-year rate was 30.1 percent. In 1992, 78.3 percent of affected women survived their first year with OC (SEER Cancer Statistics Review, 1973-1995).

Applying a forecasting separation technique (Taylor, 1977) from property-liability loss reserving to OC survival rates, the present-day five-year survival rate was estimated to be 50.9 percent, and the 20-year rate was 36.3 percent.

Estimates of the penetrance (the percentage of those with the gene mutation who will develop BC) of BRCA1/2 vary from 56 percent to 85 percent, depending on the selection bias in the population under study. This wide range of estimates is typical of the medical cancer literature. We wish our estimates of increased forces of mortality to be conservative from the insurer's perspective; i.e., our assumptions will tend to overstate somewhat the additional costs of life insurance. An average penetrance of 65 percent was selected as conservative (Lowden, 1998). BRCA mutations not only increase the probability of developing BC, but they also lead to earlier cancer development. 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 with a standard deviation of 15.39. With a BRCA mutation, the mean age at onset drops to 55.435, and 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 percent to 84 percent, depending on the type of mutation, the specific allele of BRCA1, and the population under study (Ford et al., 1994; Easton et al., 1995; and Struewing et al., 1997). An average of 40 percent seems conservative.

Based on these medical estimates, a double-decrement model was built to evaluate the increased force of mortality of a woman with a family history of BC or OC, or with a BRCA mutation. First, the survival probabilities for females given by the U.S. Decennial Life Tables for 1989-91, published by the U.S. Department of Health and Human Services, were fitted to a Makeham distribution. Then, excess forces of mortality were calculated 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, cancer-free at age 30. A second degree relative (SDR) is a grandmother or an aunt.

TABLE 2
μ-Ratios With a Family History of BC or OC, or with a BRCA Mutation, 30-Year-Old Woman, 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

Excess mortality can reach 100 percent in some cases of family history of BC, and 225 percent for a woman with a BRCA mutation. It seems to be common practice among insurers to accept at ordinary rates applicants with a force of mortality up to 150 percent of aggregate (Macdonald, 1999). Consequently, while some women 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, women with a 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 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 to, or even higher than, increases resulting from common risk factors.

TABLE 3
Mortality Ratios for Common Risk Factors

Risk Factor	Measurement	μ-ratio
High systolic blood pressure	158-167 (men)	2.06
High systolic blood pressure	178-187 (women)	2.78
Diabetes mellitus	Men	2.50
Build	40 percent overweight (women)	1.62
Build	60 percent 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

ADVERSE SELECTION COSTS

A 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 history of an individual into a series of discrete states (represented by ellipses), which analyze term insurance purchasing and genetic testing decisions. At all times, every individual is assigned to one and only one state. Transitions (represented by arrows) from one state to another can occur at any time. Forces of transition only depend on the state currently occupied and not on past history. At time $t = 0$, a woman may be in either State 1 or State 2. A woman in State 1 has not been tested for BRCA mutations and has no insurance. A woman in State 2 has not been tested, but has insurance. From State 1, six transitions are possible: a woman can remain untested and purchase insurance (State 2); she can test negative and buy insurance (State 4) or remain uninsured (State 12); she can test positive and buy insurance (State 5) or remain uninsured (State 13); she can die before getting tested or becoming insured (State 14). She can also remain in State 1. Correspondingly, ten future states are possible from State 2, including the possibility of lapsing the policy. Most transitions

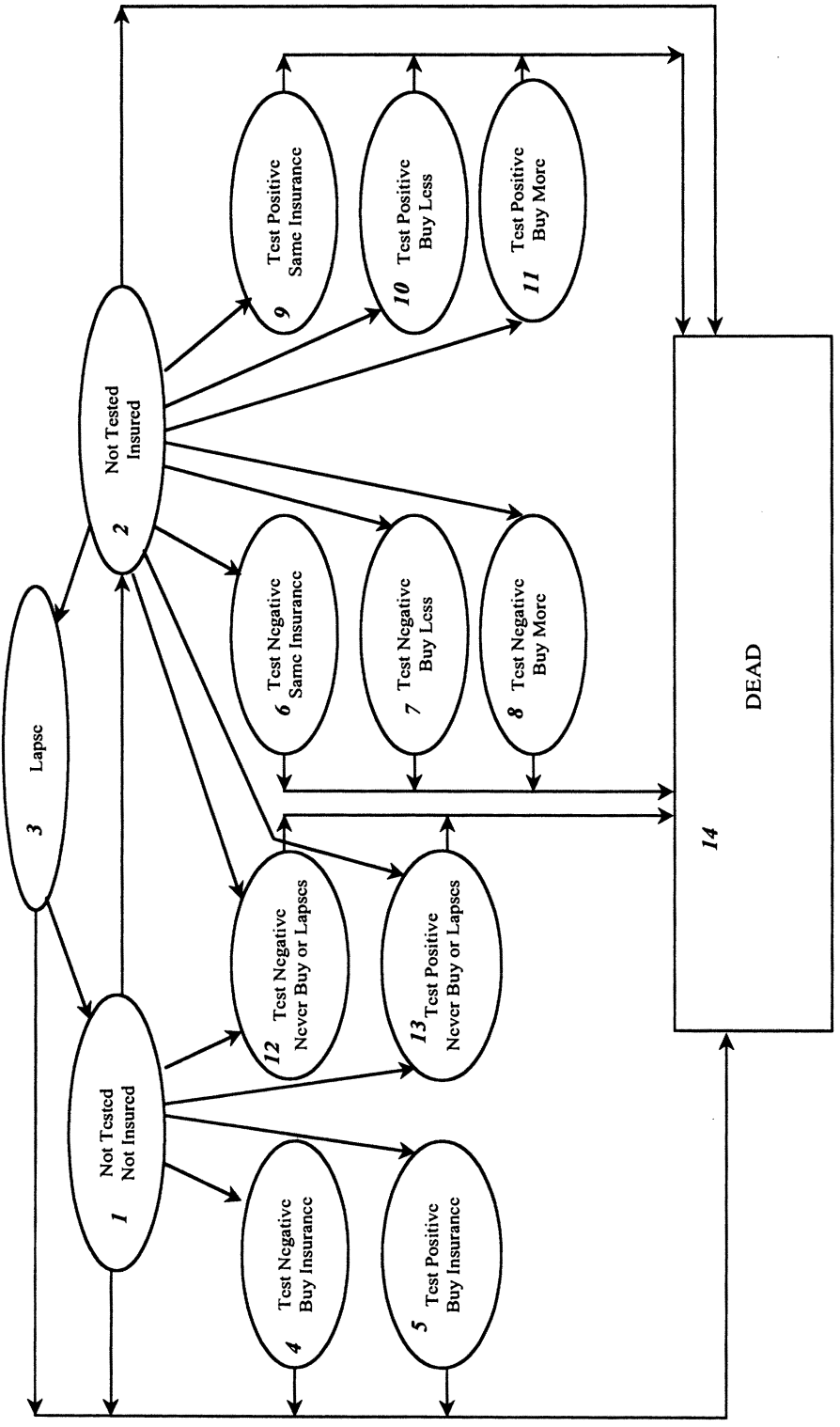


FIGURE 1
Markov Model: Heritable Breast Cancer and Adverse Selection

have cash flow implications. Transitions into States 2 and 4 through 11 imply insurance premium payments. Transitions from States 2 and 4 through 11 into State 14 imply insurance benefit lump sum payments. We assume that policies are purchased through net single premiums and that the demand for insurance is inelastic to price. Only the applicant has full information about her state.

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

Thiele's Equations

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 insurance benefits incorporating mortality and interest. This expected value, called the benefit reserve, is a liability to the company. If we assume that insurers are risk-neutral and bear no transaction costs, at all times 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 that generalizes Thiele's equation for benefit reserves (see, for instance, Gerber, 1995). One differential equation can be written for each state for which there is an outward transition.

The equation for State j is written (1)

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

where

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

δ_t = Force of interest at time t, and

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

Usually, Thiele's equation includes a positive premium rate term. This term is not present in equation (1) since policies are purchased with net single premiums. 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. This would happen only for a transition into State 14, when a death benefit is to be paid to a beneficiary. We assume no withdrawal benefit is paid for a transition from State 2 to State 3, the lapse state.[1] Switching from State j to State k also implies the release of the reserve for State j and acquiring the reserve for State k. The amount between parentheses is called the net sum at risk.

[1] We did evaluate the effect on adverse selection costs when including maximum withdrawal benefits (the full return of unearned premium) and found the differences to be very small.

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. At time t^*, the company no longer needs to hold funds aside for this policy, because the policy term and the corresponding financial obligation have ended. We solve the set of differential equations using a mathematical programming package.

The benefit reserve is the expected value of future payments. Higher order moments may be needed, for instance, to calculate risk-based capital margins. 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

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

where

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

Standard deviations, coefficients of variations, and Pearson skewness coefficients can then be calculated from these moments.

We are interested in the *cost of adverse selection* attributable to the availability of testing, under conditions where women have access to genetic test results, but insurers either have no access or are prohibited from using that information in underwriting. 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 baseline transition rates and experience mortality at rates based only on their family history, the information that the insurer uses for pricing purposes. This first solution gives us the expected present value of benefits in the "no use of genetic testing" case. We then solve the same equations, assuming genetic testing use is allowed. Women flow from States 1 to 13 at the same baseline transition rates. Net single premiums are paid in the same way. However, the transition rates into State 14 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 solution provides the expected present value of benefits in the full information case. The ratio of the two measures yields the cost multiplier of adverse selection, the ratio of what the true risk is to what is claimed and charged.

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

Baseline Assumptions

Estimates are necessary for each force of transition. Transitions into State 14 reflect mortality rates that differ by age, family history, and BRCA status. The other transitions involve a combination of testing behavior, test results, and insurance purchasing behavior. The following forces were selected for our baseline calculations.

- *Behavior before testing.* From industry figures (American Council for Life Insurance, 1998), we estimate the rate of insurance purchase $\mu_x^{1,2}$ at 5 percent, the lapse rate $\mu_x^{2,3}$ at 5 percent, and the rate of re-entry $\mu_x^{3,1}$ into State 1 at 25 percent.

- *Rate of genetic testing r.* Very few women get tested presently: only 180 women have been tested at the University of Pennsylvania since the test became available, late in 1996. The test is very expensive ($2,400), due to the fact that the BRCA1 gene has several hundred known mutations, some of which observed in a single family. The cost is not expected to decrease dramatically soon, since one laboratory owns the patent. The testing rate may depend on family history; however, a uniform rate enables us to compare adverse selection costs across family histories. Therefore, we select a testing rate of 5 percent. This rate may be considered too high for women with no family history of BC or OC and too low for women with two FDRs with BC, but for a population of women, this rate is conservative.
- *Force of interest.* We assume a constant force of interest of 5 percent.
- *Test results.* The probability p that a test result will be positive depends on individual characteristics: a woman with two FDRs affected by BC is much more likely to have the BRCA gene mutation than a woman with no family history of the disease. The value of p is found by introducing the constraint that expected benefits need to be equal in two cases: (1) there is no genetic testing, and (2) women get tested but their insurance purchase decisions are not affected. These constraints yield the following probabilities of a positive test result.

$p = .005$	no family history
$p = .080$	one FDR affected with OC, onset age unknown
$p = .150$	one FDR affected with BC, onset age 20-29
$p = .400$	two FDRs affected with BC, both with onset age 20-29

- *Changes in insurance benefits.* We assume the baseline amount of term insurance to be $1. A woman buying "less insurance" always reduces her benefit amount from $1 to $0.50; a woman buying "more insurance," increases her benefit amount to $2, $4, or $10, varied in a sensitivity analysis.
- *Insurance purchase probabilities.* Insurance decisions are assumed to occur shortly after the test result is provided. Baseline probabilities were selected as follows:
  - If uninsured and test positive: P(buy insurance) = .25; P(not buy) = .75
  - If uninsured and test negative: P(buy insurance) = .05; P(not buy) = .95
  - If insured and test positive: P(more insurance) = .27; P(same insurance) = .70; P(less insurance) = .02; P(lapse policy) = .01

- If insured and test negative: P(more insurance) = .02
  P(same insurance) = .75
  P(less insurance) = .18
  P(lapse policy) = .05

Transition rates are then obtained by multiplying the appropriate rates and probabilities. For instance, $\mu_x^{1,12} = 0.95r(1-p)$.

Cost of Adverse Selection, by Family History

The model was run, using the baseline behavioral assumptions, for the four family histories under consideration. Table 4 shows adverse selection costs for a woman with no family history. Adverse selection invokes only a small cost in this case because the probability of having the mutation for a woman with no family history of BC or OC is only .005. Insurance companies should not be concerned with restrictions of the use of genetic testing information for women with no family history.

TABLE 4
Costs of Adverse Selection for a Woman With No Family History of BC or OC, Insured at Time $t = 0$

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

Table 5 presents adverse selection costs for a woman with one FDR with OC. Costs rise as a function of the selected increased benefit of women who get tested and exceed 10 percent in some cases. We reach a similar conclusion as Macdonald (1999): the most expensive aspect of adverse selection results from the women who select high benefit levels following a positive test. The lapsing behavior of the women who test negative has much less of an impact. The most costly part of adverse selection is higher insured benefits. This result provides some support to regulations restricting the benefit amounts that can be obtained without having to disclose genetic test results.

TABLE 5
Costs of Adverse Selection for a Woman With One FDR Affected With OC, Age at Onset Unknown, Insured at Time $t = 0$

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

For the most part, adverse selection costs increase with duration, because longer terms give women more opportunities to get tested and increase their insured benefits. Occasionally, for some 40- and 50-year-old women, the costs for a 15-year period exceed the costs for a 20-year period. Recall that these are ratios of expected losses. As these women age, they become more vulnerable to other causes of mortality, thus increasing both numerator and denominator and somewhat decreasing overall adverse selection costs.

The relationship between age and the cost of adverse selection is not monotonic. Of the three initial ages, adverse selection costs are always higher for a woman age 40. Women who are cancer-free at the age of 30 are relatively unlikely to develop BC or OC before the age of 50, and even less likely to die from the disease during that 20-year period. Women who are 50 are more prone to develop cancer, but also more prone to die from other causes, so that the adverse selection cost, which is a ratio, is lower.

As shown in Tables 6 and 7, adverse selection costs are the highest when one or two FDRs are affected with early BC, reaching 20 percent in some cases. There is a substantial probability that the gene mutation is present in the family when family history is strong; consequently, the test has high expected information content beyond what is known from family history alone. Again, the major determinant of adverse selection costs is large insured benefits.

TABLE 6
Costs of Adverse Selection for a Woman With One FDR Affected With BC, Age at Onset: 20-29, Insured at Time $t = 0$

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

TABLE 7
Costs of Adverse Selection for a Woman With Two FDRs Affected with BC, Age at Onset: 20-29, Insured at Time $t = 0$

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

Figure 2 explores the sensitivity of our results to alternate behavioral assumptions. The baseline hypotheses assumed a high degree of inertia: more than 70 percent of all women do not change their insurance purchase behavior following the results of the test. Figure 2 presents adverse selection costs for a 40-year-old woman with 2 FDRs with BC and a term of 20 years. The x-axis is the probability to increase the benefit to ten following a positive test result. The y-axis is the probability to reduce benefits after a negative result. Costs could exceed 40 percent if women systemati-

cally change their insurance purchase behavior when learning the test result. We notice diminishing marginal adverse selection cost increases as the probability of buying more insurance after a positive test result increases.

FIGURE 2
Sensitivity Analysis: 40-Year-Old Woman, 2 FDRs With BC

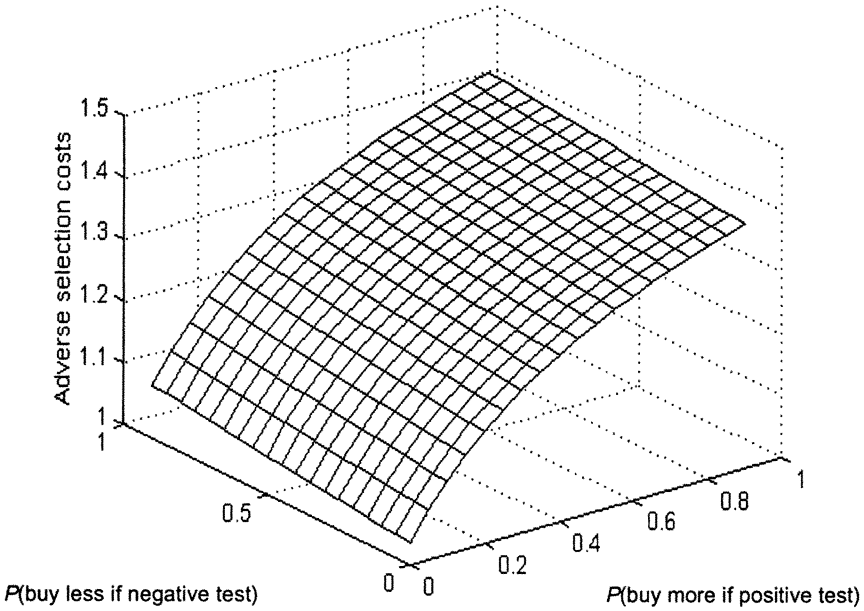


Table 8 shows standard deviations, coefficients of variation, and Pearson skewness coefficients, given one specific family history. An increased benefit of ten was selected to maximize the increase of these higher moments. Coefficients of variation and skewness measures are extremely large for term insurance, especially for short policy durations: moments are dominated by the large, but infrequent, death benefits. When genetic testing results are not allowed in underwriting, all women are priced according to their family history. When testing results are allowed, women are priced using three different mortality functions, which introduces greater variability. As a result, standard deviations always increase. Coefficients of variation and skewness, since they are ratios of increasing moments, sometimes increase and sometimes decrease. The overall effect of permitting the use of test results is moderate; it may trigger slightly higher risk-based capital margins.

One major advantage of a Markov model approach is that we can also study behavior from other states, specifically the population uninsured at time $t = 0$. Table 9 shows adverse selection costs for a woman with one FDR with BC, in State 1 at time $t = 0$. Costs are higher for a cohort of uninsured women because the adverse selection effects impact a much smaller benefit reserve.

TABLE 8
Mean, Standard Deviation, Coefficient of Variation, and Skewness Coefficient of the Present Value of Benefits for a Woman With One FDR Affected with BC, Age at Onset 20-29, Insured at Time $t = 0$, Increased Benefit = 10

	Term (in years)→	10		20	
Age		No GT	GT	No GT	GT
30	Mean	0.0081	0.0088	0.0194	0.0232
	Standard Deviation	0.1099	0.1313	0.1705	0.2207
	Coefficient of Variation	13.5695	14.8797	8.7759	9.5204
	Skewness Coefficient	39.0313	38.8954	23.0061	20.1617
40	Mean	0.0172	0.0195	0.0416	0.0497
	Standard Deviation	0.1598	0.2008	0.2478	0.3214
	Coefficient of Variation	9.2972	10.3170	5.9588	6.4684
	Skewness Coefficient	26.9017	26.3122	15.8148	14.1743
50	Mean	0.0391	0.0417	0.0918	0.1006
	Standard Deviation	0.2387	0.2733	0.3619	0.4201
	Coefficient of Variation	6.1204	6.5508	3.9410	4.1776
	Skewness Coefficient	17.9645	18.0224	10.8068	10.2973

(No GT = insurers cannot use genetic test results in underwriting)

TABLE 9
Costs of Adverse Selection for a Woman With One FDR Affected With BC, Age at Onset: 20-29, Uninsured at Time $t = 0$

		Term (in years)			
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

Finally, we consider the financial consequences when insurers do not incorporate the correct family history information in the pricing process. This could result from either a ban on the use of family history in underwriting or from policyholder fraud. Our previous results assumed that women report their family history truthfully and that insurers are allowed to use family history in underwriting. Now assume a woman with 2 FDRs with BC reports no family history of BC or OC, and the insurer fails to detect this fraud. Table 10 reveals huge adverse selection costs as compared to Table 7, in which an insurer correctly underwrites a woman with this family history. It is of crucial importance for insurers to request detailed family information (including age at onset) during the underwriting process and to investigate the applicant's statements vigorously.

TABLE 10

Costs of Adverse Selection for a Woman With Two FDRs With BC, Ages at Onset: 20-29, Insured at Time $t = 0$, Priced at No Family History of BC or OC

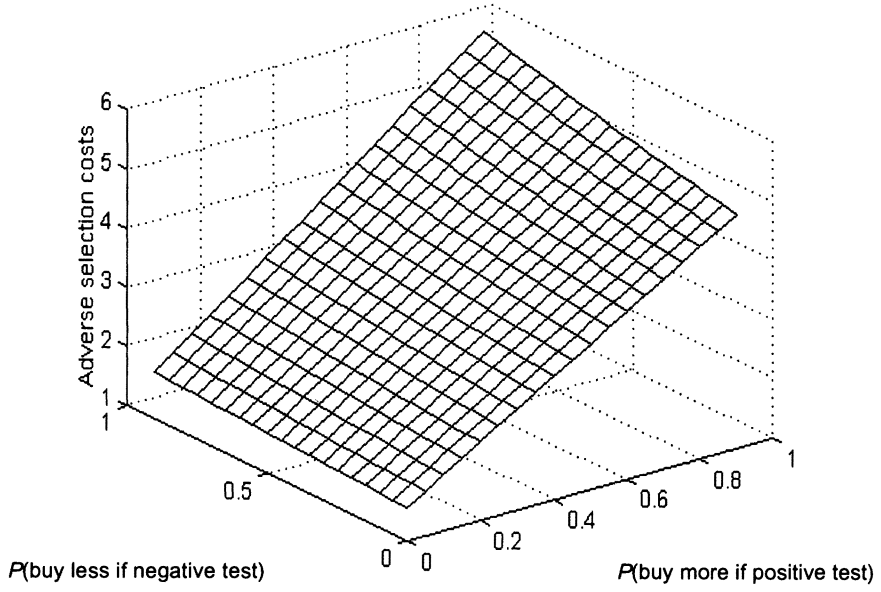
		Term (in years)			
Age	Increased Benefit	5	10	15	20
	$2	1.2356	1.5149	1.7253	1.8490
30	4	1.2813	1.6364	1.9312	2.1263
	10	1.4170	1.9933	2.5300	2.9260
	$2	1.2536	1.5176	1.6803	1.6947
40	4	1.3076	1.6565	1.8963	1.9461
	10	1.4678	2.0639	2.5243	2.6703
	$2	1.1469	1.2909	1.3704	1.3669
50	4	1.1878	1.3865	1.5138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

Figure 3 tests the sensitivity of the behavioral assumptions in the fraud case; we find adverse selection costs exceeding 200 percent for most behavioral hypotheses and approaching 600 percent in extreme cases.

CONCLUSIONS

Should the insurance industry oppose any ban on the use of genetic testing results in underwriting, to avoid adverse selection? Should legislators enforce some degree of subsidization among policyholders, in order not to penalize people because of their bad luck in the genetic lottery, a situation for which they are not responsible? The answer to these questions may depend on the cost of adverse selection to the insurance industry and on the likelihood that it may threaten the solvency of some insurers. In this article, we have attempted to provide some actuarial insight in the debate by quantifying the impact of adverse selection.

FIGURE 3
Sensitivity Analysis: 40-Year-Old Woman, Fraudulent Reporting of Family History



All our 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 hope that women found to carry BRCA mutations can reduce their risk of BC mortality by increased surveillance through higher frequency of mammograms, prophylactic mastectomy, or chemoprevention with tamoxifen. Schrag (1998) estimates that the gain in life expectancy for a 30-year-old woman with the BRCA mutation who chooses to undergo prophylactic mastectomy and oophorectomy could be as high as 5.3 years. Therefore, we believe that all figures are cautious upper bounds of adverse selection costs.

As Tables 4 to 7 illustrate, it is only in a few cases (20-year term, family history of BC with early age at onset, large benefit amounts) that the adverse selection cost exceeds ten percent. The problem resulting from very large benefit amounts could be alleviated if insurers were allowed to use genetic test results for the underwriting of such policies, in return for a ban on the use of tests for policies with a reasonable amount. The Association of British Insurers enforces such a rule, by which disclosure of test results is not required if the sum insured does not exceed £100,000 and if the policy is being purchased in connection with a mortgage. (This also reduces adverse selection since lenders in the U.K. usually require mortgage protection insurance).

Under our approach, the average adverse selection cost in a portfolio is expected to be way below ten percent. So, this cost is likely to be compensated by the overall long-term trend of decrease in mortality rates (a factor not introduced in our calculations) that stands currently around 0.5 percent per year. Therefore, we believe that adverse selection is a problem that insurers can control.

This conclusion holds only if companies apply very tight underwriting standards. In the application process, prospective insureds need to provide a 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, Table 10 shows that adverse selection costs could become unbearable.

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