

## **WELFARE EFFECTS OF BANNING GENETIC INFORMATION IN THE LIFE INSURANCE MARKET: THE CASE OF BRCA1/2 GENES**

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### **ABSTRACT**

We investigate whether regulations that ban insurance companies from access to individuals' genetic tests are likely to lead to substantial adverse selection costs for the specific example of the so-called breast cancer (BRCA1/2) genes. Using a data set including economic, demographic, and relevant family background information to simulate the market for 10-year term life insurance, we find generally only modest adverse selection costs associated with such a regulatory ban. However, for family background groups that are at high risk for carrying one of these genes, the efficiency cost of adverse selection may be significant should the test become widely adopted.

### **INTRODUCTION**

The debate on whether insurance companies should be allowed to use results of genetic tests for the purpose of assessing differential prices for health, life, or disability insurance has been very lively and increasingly relevant as genetic technology and lawmaking efforts are both progressing very rapidly.<sup>1</sup> The U.S. Senate in 2003 passed the so-called Genetic Information Nondiscrimination Act (GINA) by a unanimous

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<sup>1</sup> For example, the special issue of the *North American Actuarial Journal* (Vol. 3, No. 1, January 1999), which is devoted to articles on genetic technology and underwriting, reflects such an interest.

vote and the bill is waiting attention by the House. Although GINA restricts the use of genetic test results only for health insurance and employment purposes, many other countries extend restrictions to life and long-term disability insurance.<sup>2</sup>

Arguments in favor of restricting the use of genetic test results for rate-making purposes are generally based on concerns with equity and/or protection of privacy. Those who favor allowing insurance companies to use the information argue that not to do so would expose the market to serious adverse selection problems.<sup>3</sup> There is a limited empirical literature, mostly based on actuarial simulations that estimate possible price effects for insurance that would result from a ban on the use of genetic test results for rate-making purposes (e.g., Macdonald, 2003; Macdonald and Pritchard, 2000, 2001). Although this is one important component of a full analysis of the associated adverse selection costs of such regulation, this work can be usefully supplemented by standard economic welfare analysis. In this article we provide such an analysis for the specific case of information relating to breast cancer, that is, the potential for genetic screening for the so-called breast cancer susceptibility genes (BRCA1/2 genes). The advantage of a standard welfare analysis is that it is based on a rational choice (utility-based) model that allows both for price effects from related demand analysis and efficiency measurement of alternative information scenarios and regulations.

We adopt a standard economic model of the life insurance market, based on Abel (1986), Brugiavini (1993), Villeneuve (2000, 2003), and Hoy and Polborn (2000). Insurance companies are assumed to offer nonexclusive contracts and to price these contracts linearly. This contrasts with the models involving price–quantity competition, pioneered by Rothschild and Stiglitz (1976) and Wilson (1977). Those models are well suited to property-liability insurance where the size of the loss is clearly identifiable and so the amount of insurance provided in the contract acts as an effective (self-) selection mechanism for different risk types. However, the economic cost associated with the loss of life is not easily observable or objective. Moreover, individuals often change the amount of their life insurance holdings over time and through various heterogeneous products (e.g., whole life, group life, individual term insurance). Therefore, we develop a stylized model of the term life insurance market and simulate the effect of allowing consumers to hold private information (genetic test results) when deciding how much insurance to purchase.<sup>4</sup> We compare the implications of allowing consumers to hold this information as private to a *laissez faire* situation in which insurers do risk-rate premiums according to genetic test

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<sup>2</sup> For a discussion on the variety of international regulatory responses concerning genetic information and insurance, see Lemmens and Bahamin (1998), Lemmens (2000), and Knoppers, Godard, and Joly (2004). A recent editorial in *Nature Genetics* (May 2004) discusses the U.S. Genetic Information Nondiscrimination Act.

<sup>3</sup> See Kitcher (1996) for a good discussion of the equity arguments supporting prohibition of genetic information being used to risk-rate insurance. Also, see Hoy (1984), Hoy and Lambert (2000), and Bossert and Fleurbaey (2002). For a general discussion of efficiency concerns created by adverse selection in insurance see Crocker and Snow (1986). See Brockett and Tankersley (1997) for a discussion about regulation of genetic information in insurance markets.

<sup>4</sup> For empirical evidence on linearity of contracts and nonexclusivity of provision, see Cawley and Philipson (1999) and Pauly et al. (2003).

results. In the case of private information, higher-risk individuals purchase more insurance than do lower-risk individuals, hence driving the price higher than the population-weighted actuarially fair price. From an efficiency perspective this means higher-risk types overconsume while lower-risk types underconsume, hence generating adverse selection costs. We measure the adverse selection costs as a standard deadweight loss based on the compensating variation measure.

In the short-term future at least (say less than 5 years), it seems plausible that not many women will obtain genetic test results for the BRCA1/2 genes. One reason is cost. As Subramanian et al. (1999) note, the cost of the test is very high (\$2,400), and since the test is patented the cost may not fall significantly for some time. Therefore, we consider the implications for adverse selection if only 5 percent of women become informed (i.e., take the test), if 20 percent, and if 100 percent; the last of these covers the maximum adverse selection costs possible for this particular genetic test. We also break down our results by risk classes as defined by different family backgrounds for breast and ovarian cancer. Since higher-risk classes as defined by family background imply different likelihoods for possessing the gene, this is an important consideration. In particular, women with more first-degree relatives having had breast or ovarian cancer, and at a younger age, will presumably feel more at risk and hence be more likely to obtain the genetic test.<sup>5</sup> Moreover, these classes also have a higher proportion of women who have the relevant cancer genes. Therefore, not surprisingly, we find that adverse selection costs can be substantially higher for classes represented by higher-risk family backgrounds.

We also use a variety of assumptions about demand and risk preferences. We base our analysis of the demand for life insurance on the presumption that a family's objective is to maintain its per capita standard of living should an adult (income earning) member die.<sup>6</sup> In fact, we make explicit use of household equivalence scales to generate benchmark preferences for life insurance. However, we do assume that insurance purchasing is sensitive to price and so the above goal applies only if the individual considers the price to be actuarially fair. Within this framework, we allow for a variety of possible risk aversion levels. In this way we are able to provide extensive sensitivity analysis for our simulations. Our overall conclusion is that the size of adverse selection costs generated by a regulation prohibiting insurers from using genetic test results for the BRCA1/2 genes would probably be very modest in most circumstances. Thus, equity and privacy arguments that favor such regulation would not pale in comparison. However, for some higher-risk family background types, if women in sufficient numbers obtain genetic tests, then adverse selection costs from such regulation could be substantial. This points to the possibility that as genetic information in society grows, there may come a point when genetic privacy may not be desirable. Thus, short- to medium-term moratoria on the use of genetic test results by insurance companies may be a more desirable policy framework than strict regulation through legislation that may be difficult to change in the future. In the long run, however, as information that is available to individuals about their own genetic predispositions grows and becomes more easily obtainable, regulations prohibiting

<sup>5</sup> There is evidence that women at higher risk are more likely to obtain diagnostic testing (e.g., mammograms). See Picone, Sloan, and Taylor (2004) and Witt (2004).

<sup>6</sup> See, for example, Bernheim et al. (2003).

insurers from access to genetic information may lead to substantial adverse selection costs. In this latter scenario, alternatives to banning the use of genetic test results by insurers for risk-rating premiums, such as limited public life insurance provision for high-risk types, may be more socially desirable.

The article proceeds as follows. In the section on the "Theoretical Background" we describe the basic underlying life insurance model. The application for this model is described in the next section, along with a brief discussion of the data sources and methods used to select parameters. This is followed by a discussion on "Results" whereas some extended results are designed to provide sensitivity analysis in the following section. "Summary and Conclusions" are given in the final section.<sup>7</sup>

### THEORETICAL BACKGROUND

First we present the model we use to describe an individual's decision about how much life insurance to purchase.<sup>8</sup> We then show how market equilibrium is determined when there is a homogeneous group of consumers (i.e., individuals with the same probability of death), a scenario that applies under symmetric information conditions. This is followed by a discussion of equilibrium determination under conditions of asymmetric information (i.e., when the population is made up of individuals who have different probabilities of death and these are private information of consumers so that adverse selection occurs). In the following section we show how to apply these models to examine the welfare implications of regulatory adverse selection in the case of genetic testing for BRCA1/2 genes.

An individual's probability of death,  $p$ , depends on family history and is assumed known to the individual and the insurer. If the person has a genetic test for one of the BRCA1/2 genes, then the individual revises her assessment of this probability depending on the test result according to Bayes' law. The insurer may or may not have access to genetic test results as this depends on legislation as well as inherent observability of "personal" information. We consider both cases of genetic information being used by firms and not being used by firms, the latter situation resulting in adverse selection.

The economic resources available to families are measured in net present value (NPV) terms, although for ease of discussion we generally simply refer to income. If the individual (woman) in question dies and no life insurance were purchased, the income earned by surviving family members is amount  $K$ , where  $K$  is the NPV of the income stream of the other adult household member(s) (generally the father or stepfather). For single-parent families  $K = 0$  (strictly speaking).<sup>9</sup> If the individual lives, income consists of the income of that individual,  $Y$ , as well as amount  $K$ . Life insurance can be

<sup>7</sup> Details concerning the data and computational methods are quite complex and so are made available in a technical appendix ([www.uoguelph.ca/~mhoy/](http://www.uoguelph.ca/~mhoy/)). This appendix also includes substantial detail on our simulations not reported here as well as additional simulations for the purpose of sensitivity analysis.

<sup>8</sup> This model is based on Hoy and Polborn (2000).

<sup>9</sup> Other sources of income could of course contribute to  $K$  and it is unlikely that the children of a single parent who dies would be left with no resources. We have no person-specific information about such resources and so in our simulations we set  $K$  equal to 5 percent of  $Y$ .

purchased in amount  $L \geq 0$ , at price  $\lambda$ . If the individual purchases life insurance, then in the death state surviving family members have income  $K$  as well as income from the insurance claim, resulting in a net amount of  $K + (1 - \lambda)L$ . If the individual lives, income is  $Y + K - \lambda L$ ; that is, total income less the cost of life insurance purchased. Letting  $u(\cdot)$  and  $v(\cdot)$  represent von Neumann–Morgenstern elementary utility indices in the life and death states, respectively, with  $u'(\cdot), v'(\cdot) > 0$  and  $u''(\cdot), v''(\cdot) < 0$ , then the expected utility of the family as a function of insurance purchases is

$$EU(L) = (1 - p)u(K + Y - \lambda L) + pv(K + (1 - \lambda)L). \quad (1)$$

The family will maximize expected utility subject to  $L \geq 0$ , with first-order condition

$$-(1 - p)\lambda u'(K + Y - \lambda L) + p(1 - \lambda)v'(K + (1 - \lambda)L) \leq 0 \quad (2)$$

and second-order condition

$$(1 - p)\lambda^2 u''(K + Y - \lambda L) + p(1 - \lambda)^2 v''(K + (1 - \lambda)L) < 0. \quad (3)$$

The second-order condition is always satisfied since we assume  $u''(\cdot) < 0$  and  $v''(\cdot) < 0$ . For our simulations, we further assume families have constant relative risk aversion (CRRA) utility functions with degree of risk aversion  $\gamma$ ,

$$u(X) = \frac{X^{1-\gamma}}{1-\gamma} \quad \gamma > 0, \gamma \neq 1, \quad (4)$$

$$u(X) = \ln X \quad \gamma = 1. \quad (5)$$

Additionally, utility in the death state is a fraction of utility in the life state; that is,

$$v(x) = a \cdot u(x) \quad a < 1. \quad (6)$$

This is a simple way to parameterize the objective function in order to reflect the fact that when an adult family member dies the total income required to maintain per capita income levels for the remaining members of the family falls. Note that this implies that, at the same total family income, marginal utility of income is less in the death state than in the life state.<sup>10</sup> Hence, expected utility is

$$EU(L) = (1 - p) \left( \frac{(K + Y - \lambda L)^{1-\gamma}}{1-\gamma} \right) + pa \left( \frac{(K + (1 - \lambda)L)^{1-\gamma}}{1-\gamma} \right) \quad (7)$$

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in these cases, an admittedly conservative assumption. We also added 30 percent of  $Y$  to each family's income (in the death state) as a robustness test and this did not change the qualitative nature of our results.

<sup>10</sup> That is, with one less family member per capita income is higher in the death state for the same level of income that applies in the life state. Our method is consistent with the use of equivalence scales for standardizing income needs for families of different size, as described in the technical appendix available online.

with first-order condition

$$-(1-p)\lambda(K+Y-\lambda L)^{-\gamma} + pa(1-\lambda)(K+(1-\lambda)L)^{-\gamma} \leq 0 \quad (8)$$

and second-order condition

$$-(1-p)\lambda^2\gamma(K+Y-\lambda L)^{-\gamma-1} - pa(1-\lambda)^2\gamma(K+(1-\lambda)L)^{-\gamma-1} < 0. \quad (9)$$

From (8) it is straightforward to derive that the demand for insurance is the value of the following function:

$$L(a, p, K, Y, \lambda) = \frac{p^{1/\gamma}(1-\lambda)^{1/\gamma}a^{1/\gamma}(K+Y) - (1-p)^{1/\gamma}\lambda^{1/\gamma}K}{(1-p)^{1/\gamma}\lambda^{1/\gamma}(1-\lambda) + p^{1/\gamma}(1-\lambda)^{1/\gamma}a^{1/\gamma}\lambda}. \quad (10)$$

Note that if  $a = 1$  (i.e.,  $v(\cdot) = u(\cdot)$ ) and  $Y > 0$ , then at zero insurance purchases ( $L = 0$ ) the marginal utility of income is higher in the death state than in the life state (since  $u''(\cdot) < 0$ ),

$$u'(K+Y) < v'(K) = u'(K). \quad (11)$$

If this were the case, then at actuarially fair pricing (i.e.,  $\lambda = p$ ) the family would purchase an amount of insurance so that income in the two states is the same. However, if  $a < 1$  and insurance is priced at the actuarially fair rate, the optimal amount of insurance purchases, which equalizes marginal utility across the two states, implies less consumption in the death state than in the life state. Moreover, since  $u''(\cdot) < 0$ , the marginal utility in the life state is less the smaller  $Y$  is. Thus, if  $Y$  is sufficiently small relative to  $K$ , then given  $a < 1$ , it is possible that marginal utility in the death state will be less than that in the life state even if no insurance is purchased. In this case, it is optimal for the individual not to purchase any insurance even if it is offered at the actuarially fair rate. We will see in our simulations that this is an important possibility.

We assume that insurers are risk neutral, face zero administrative costs, and are in a competitive price environment. If insurers can observe the individual's probability of death,  $p$ , then equilibrium is simply characterized by a price of  $\lambda = p$  for each consumer and demand is determined by Equation (10). In this case of symmetric information, we can allow for individuals to have different values of the various parameters ( $a$ ,  $K$ , and  $Y$ ) with no change in the determination of equilibrium price.

Now, suppose we allow for heterogeneity of consumers in risk type ( $p$ ). We index risk type by  $r \in T$  where  $T = \{1, 2, \dots, t\}$ . Thus,  $p^r$  represents the probability of death for risk type  $r$  with  $p^1 \leq p^2 \leq \dots \leq p^t$ . Individuals also vary according to their inherent demand for life insurance, which depends on family composition and other considerations. We index demand type by  $d \in M$  where  $M = \{1, 2, \dots, m\}$ . Thus,  $a^d$  represents the taste parameter for demand type  $d$  with  $a^1 \leq a^2 \leq \dots \leq a^m$ . The equilibrium price when different risk types are pooled will be determined according to an interaction of these two dimensions of heterogeneity (i.e., risk type and demand type).

If insurers are privy to the same information about risk type as are consumers themselves, and are allowed legally to use this information, then the price charged will

simply be the actuarially fair rate; that is,  $\lambda = p^r$ . Under these symmetric information conditions, the demand for insurance again follows from Equation (10) but differentiated by risk and demand type. Thus, we can summarize this outcome in Equation (12),

$$L^{rd} = L(a^d, p^r, K, Y, \lambda); \quad \lambda = p^r. \quad (12)$$

Thus, the distribution of the parameter  $a^d$  within the population has no impact on the determination of price under symmetric information.

Now suppose insurers cannot charge separate prices from individuals of different risk types and must, instead, charge all individuals the same price  $\lambda$ . Let  $i = 1, \dots, N$  index all individuals, regardless of risk type or demand type. Each individual is distinguished by her risk type  $r$  and demand type  $d$ . Let  $p_i$  be the probability of death and  $a_i$  be the demand type parameter for individual  $i$ . Thus, for each  $i$  it follows that  $p_i = p^r$  for some  $r \in T$  and  $a_i = a^d$  for some  $d \in M$ . We suppress the parameters  $K_i$ ,  $Y_i$ , and  $a_i$ , and denote the demand for life insurance by individual  $i$  as  $L^i(p_i, \lambda)$ . Thus, the total revenue from selling insurance to the entire group of individuals, where of course in some situations demand is zero, is  $TR(\lambda) = \sum_i \lambda L^i(p_i, \lambda)$  while the expected total cost is  $ETC(\lambda) = \sum_i p_i L^i(p_i, \lambda)$ . Under conditions of perfect competition equilibrium price is determined by the zero expected profit condition,  $\Pi(\lambda) = TR(\lambda) - ETC(\lambda) = 0$ , and so

$$\lambda_e = \frac{\sum_i p_i L^i(p_i, \lambda_e)}{\sum_i L^i(p_i, \lambda_e)}. \quad (13)$$

We are ensured that an equilibrium price  $\lambda_e$  exists provided at least one person of the highest risk type will demand a positive amount of insurance if charged the actuarially fair rate for that risk type (i.e., if  $\lambda = p^t$ ). The reason for this is that if  $\lambda = p^1$ , then the expected profit,  $\Pi(\lambda)$ , will be negative since the firm just breaks even on sales to the lowest risk-type individuals and all other contracts sold generate expected losses. If  $\lambda = p^t$  then the profit will be greater or equal to zero since the firm breaks even on sales to the individuals of the highest risk type and, if anyone of lower risk type purchases insurance, then the insurer will earn positive expected profits on those contracts. Since all component functions of  $\Pi(\lambda)$  are continuous in  $\lambda$ , then so is  $\Pi(\lambda)$  continuous. Thus, the function  $\Pi(\lambda)$  must equal zero for at least one value of  $\lambda$ . The lowest such value is then the equilibrium price  $\lambda_e$ .<sup>11</sup>

## APPLICATION

Our application is a simulation of the market for 10-year term life insurance for Canadian women in the age group of 35 to 39 years. We use data from the 1999 Canadian National Population Health Survey (NPHS), Health Component Data Set, which provides both detailed health information, including family background of

<sup>11</sup> If there is more than one value of  $\lambda$  for which  $\Pi(\lambda) = 0$ , then the lowest such value is the equilibrium price since any higher price would be undercut by some firm(s). A formal proof would be essentially the same as that in Section 2 of Hoy and Polborn (2000).

breast cancer, as well as relevant economic and demographic information that affects demand for life insurance. The survey does not include information about life insurance purchases, and this is simulated using a standard expected utility model. Information on risk factors (family background) is used in the program *CancerGene* to determine both the probabilities of getting cancer and the probability that if the woman were to obtain a genetic test for one of the BRCA1/2 genes that she would test positive.

Here we provide a sketch of how this information was utilized in generating simulations of the insurance market in both scenarios of symmetric and asymmetric information regarding genetic test results. A more detailed explanation of all of the computations and assumptions involved in the simulations is provided in the appendix ([www.uoguelph.ca/~mhoy/](http://www.uoguelph.ca/~mhoy/)). The variables needed to describe the insurance market are  $\gamma, p_i, Y_i, K_i, \alpha_i, q_i, p_i^{\text{neg}}, p_i^{\text{pos}}$ , where person-specific information is indicated by subscript  $i$ . Although the risk preference parameter,  $\gamma$ , would realistically vary across individuals, we assume it is the same for all. Not only would it be a substantial complication to depart from this assumption, but we have no person-specific information about this risk parameter. We do, however, vary this parameter in our robustness tests and so can demonstrate how our results depend on this parameter.

First we establish a 10-year death probability from all causes (i.e., the probability that a woman in the age group of 35 to 39 years will die within the next 10 years) as a benchmark by using the CDC Life Tables (Arias, 2002). We then deduct from this value the probability that a woman would die of breast cancer over this period (i.e., not conditioned on family background).<sup>12</sup> This is our benchmark 10-year probability of death due to all causes except breast cancer. Then, using the *CancerGene* program and the family background information from the NPHS data set mentioned above, we create 13 risk categories according to family background. The death probabilities are denoted simply by  $p_j, j = 1, 2, \dots, 13$ ; that is, each person  $i$  belongs to one of these risk groups.

If individuals obtain genetic test results, then their probability of dying from breast cancer, of course, depends on both family background and whether the person tested positive or negative. Adding these differential probabilities of death from breast cancer to the probability of death from all other causes leads to probabilities of death specific to each risk group  $j$ , denoted  $p_j^{\text{pos}}$  and  $p_j^{\text{neg}}$ , respectively. Summary data describing the 13 risk groups and the relevant probabilities for those who test positive and negative, should test results be obtained, are given in Table 1. Also included is the probability that a woman from a given risk group will test positive ( $q$ ) for one of the BRCA1/2 genes and the number of individuals in each group. (Note: We suppress subscripts  $i$  or  $j$  whenever it will not lead to confusion.)

The groups are ordered, roughly speaking, by increasing risk level. Women in Groups 1 and 2 have no family history of breast cancer. However, women in Group 1 have a sister who has not been affected by breast or ovarian cancer while women in Group 2 have no sister at all and so have a slightly higher risk level due to the absence of any

<sup>12</sup> This probability subtracted from the general population is taken from Ford et al. (1998), and the 10-year survival rate for women getting breast cancer is taken from the SEER Cancer Registry (see Ries et al., 2004), based on all women under the age of 50 years.

**TABLE 1**  
Mortality Data on Family Background Risk Groups

| Group | $p$      | $p^{\text{neg}}$ | $p^{\text{pos}}$ | $q$   | No. of Individuals<br>per Group |
|-------|----------|------------------|------------------|-------|---------------------------------|
| 1     | 0.017340 | 0.017309         | 0.048413         | 0.001 | 491                             |
| 2     | 0.017344 | 0.017313         | 0.048417         | 0.001 | 157                             |
| 3     | 0.017476 | 0.017269         | 0.086038         | 0.003 | 4                               |
| 4     | 0.017483 | 0.017208         | 0.086037         | 0.004 | 21                              |
| 5     | 0.017494 | 0.017287         | 0.085975         | 0.003 | 2                               |
| 6     | 0.017500 | 0.017294         | 0.085982         | 0.003 | 5                               |
| 7     | 0.017584 | 0.017240         | 0.086009         | 0.005 | 1                               |
| 8     | 0.017608 | 0.017195         | 0.086005         | 0.006 | 2                               |
| 9     | 0.017769 | 0.017219         | 0.086018         | 0.008 | 8                               |
| 10    | 0.017814 | 0.017194         | 0.086017         | 0.009 | 1                               |
| 11    | 0.018088 | 0.017193         | 0.086000         | 0.013 | 6                               |
| 12    | 0.020125 | 0.017165         | 0.086007         | 0.043 | 1                               |
| 13    | 0.021480 | 0.016994         | 0.086010         | 0.065 | 1                               |

*Notes:* Group refers to family background risk group,  $p$  is the relevant group's average probability of death,  $p^{\text{neg}}$  and  $p^{\text{pos}}$  are death probabilities conditional on testing negative and positive for one of the BRCA genes, while  $q$  is the probability of an individual from the relevant risk group testing positive for one of the BRCA genes.

"good news" about family history. Women in Groups 3 to 11 have one first-degree relative affected by either breast or ovarian cancer, while those in Groups 12 and 13 have multiple family members who have experienced breast or ovarian cancer. Within Groups 3 to 13 family background risk is differentiated by age at onset of cancer for family members, whether death occurred, etc. Not surprisingly, these family background characteristics imply different likelihoods that women from these groups would test positive for the breast cancer gene (from 0.001 in group 1 to 0.009 in group 10 and up to 0.065 in group 13). Also, the penetrance (i.e., the likelihood that a given mutation of a gene will actually result in disease) of the BRCA1/2 gene varies according to its mutation type, and this is correlated with family history.<sup>13</sup> Thus, the ratio  $p^{\text{pos}}/p^{\text{neg}}$  also varies across family type, increasing with overall family risk level, and in particular, is much smaller for the first two risk groups. That is, a woman with no family history of breast cancer not only is less likely to have one of the breast cancer genes, but if she does it is more likely to be one with a relatively low degree of penetrance and hence will have a lower  $p^{\text{pos}}$  value.

Incomes of the household and the woman in particular are available from the data set, although in group form. This information is used to generate the important economic variables  $Y_i$  and  $K_i$ . Details on how grouped information was handled are in

<sup>13</sup> The so-called breast cancer gene is relatively complex and with over 100 known mutations. Different versions of a mutation imply different likelihood and severity of onset of cancer ("high penetrance mutations" vs. "low penetrance mutations"). Therefore, the likelihood and type of "genetic" breast cancer is correlated with the type of detailed family history available and the program *CancerGene* makes use of this information.

the appendix. The functional form of the utility function is that of constant relative risk aversion, as mentioned earlier. We used three different values for the degree of relative risk aversion, namely, 0.5, 1, and 3. We believe this reflects a reasonable range as suggested by the empirical and experimental literature on risk taking under uncertainty (see Blake, 1996).

Finally, we use a benchmarking exercise to determine the parameter  $a$  for the death state utility function,  $v(x) = a \cdot u(x)$ . The purpose of this exercise is to find a utility function so that the "average consumer" of insurance buys that amount of insurance, when offered at the actuarially fair rate, so that the household's standard of living remains unchanged should she die and her income be lost to the family. If insurance is priced higher than the actuarially fair rate, which happens in the presence of adverse selection for individuals who test negative for the BRCA1/2 genes, then demand is less.

Notice in Table 1 that for most risk groups there are very few observations. Thus, for each risk group we simulate the insurance market based on socio-economic information of all 700 observations available rather than use only the individuals in each group. As long as socio-economic information is not correlated with risk factors (i.e., family background of the woman with respect to breast cancer incidence), this process is not of concern. To check on this we did separately simulate the market for each of the risk categories 1, 2, and 4 using only the observations from their respective groups rather than the entire set of observations. We obtained qualitatively similar results.

Below we describe each of the four sets of simulations we do, which we refer to as Modules 1 through 4. Each module refers to a different information scenario regarding whether individuals have taken genetic tests and whether insurers are banned from using genetic test results in rate making. In each module we compute the efficiency gain from insurance by comparing the per capita compensating variation (CV) that results from the opportunity to purchase insurance in the given information scenario. That is, we compute the value of CV that equates the utility an individual would receive with no insurance (and no genetic test information) to expected utility if insurance is available (conditional on a given information scenario) with income in both states of the world reduced by amount CV. Note that insurance availability always leads to a higher expected utility level and so for all of our modules, or information scenarios, the per capita CV will be positive. It is the difference in the levels of the per capita CV, however, that we use to compare the relative efficiency of the market in the different modules. In particular, this allows us to determine a measure of inefficiency due to adverse selection for the case in which insurers are banned from using genetic test results for rate making.

Another measure that is often reported as a measure of the impact of adverse selection is the amount by which the price of insurance rises, relative to the average probability of death (also the population weighted actuarially fair price), due to adverse selection. We also report this value for each risk group (i.e.,  $\lambda_e^j/p_j$  for each group  $j$  where  $\lambda_e^j$  is the equilibrium price under adverse selection).<sup>14</sup>

<sup>14</sup> Note that equilibrium price  $\lambda_e^j$  is as defined in Equation (13) where  $j$  refers to the subpopulation of risk group  $j$ . Note also that  $\lambda_e^j/p_j = 1$  if there is no adverse selection.

We now describe more formally the modules or information scenarios. In all cases insurance companies are assumed to observe and be allowed to use family background information in their pricing behavior. Thus, the only difference between modules involves genetic tests.

**Module 1:** *Individuals have no genetic test information.*

In this case the price of insurance is actuarially fair according to family background, that is,  $\lambda = p_j, j = 1, 2, \dots, 13$ . There are 13 risk groups, hence 13 different prices. This case provides an interesting benchmark with which to compare our other results.

**Module 2:** *All individuals obtain genetic tests and insurance companies are allowed to use this information in setting prices (i.e., in addition to family background information).*

Again, from individuals' perspectives the price of insurance is actuarially fair. However, within each of the family background groups,  $j = 1, 2, \dots, 13$ , there are now two prices. One price applies to those who test positive for having one of the BRCA1/2 genes, in which case  $\lambda = p_j^{\text{pos}}$ , while a second price applies to those who test negative (i.e.,  $\lambda = p_j^{\text{neg}}$ ). The fraction of individuals who test positive,  $q_j$ , depends on family background and so we write  $q_j$  to represent this fraction for individuals from risk (family background) group  $j, j = 1, 2, \dots, 13$ . Fraction  $1 - q_j$  tests negative. Thus, this scenario reflects a situation in which each individual knows whether she has one of the breast cancer genes BRCA1/2 and insurers are also privy to this information.

Since information is symmetric in both Modules 1 and 2 there is no efficiency loss due to adverse selection in either case. Suppose in the context of Module 2 we refer to the situation before test results are known as the *ex ante* position and the situation after test results are known as the interim position. The interim position reflects the state of information after test results are revealed to both insureds and insurers but before the state of the world—life or death—is realized. We refer to the situation after the state of the world is realized as the *ex post* position. From an *ex ante* perspective, an individual views her expected utility as generated by a lottery in which she faces price  $\lambda = p_j^{\text{pos}}$  with probability  $q_j$  and price  $\lambda = p_j^{\text{neg}}$  with probability  $1 - q_j$ , where the price an individual faces in Module 1 is simply the expected price from this lottery (i.e.,  $p_j = q_j \cdot p_j^{\text{pos}} + (1 - q_j) \cdot p_j^{\text{neg}}$ ). It turns out, as explained more formally below, that this lottery over prices, often referred to as premium risk, is undesirable. Thus, from an *ex ante* perspective the insurance opportunity in Module 2 is less valuable than in Module 1. For this reason we find that the CV measures in Module 2 are uniformly less than for Module 1, although the “size” of the efficiency loss due to premium risk turns out not to be very large.

To formally substantiate the claim made in the above paragraph, we introduce the following notation. Let  $l(p)$  represent demand for life insurance under symmetric information: that is,  $\lambda = p$  and so the optimal value of  $L$  is such that  $L \equiv l(p)$ . Symmetric information implies that this notation is appropriate for both Modules 1 and 2, except, of course, under different prices (i.e., different  $ps$ ). In Module 1 the value function for (*ex ante*) expected utility is,

$$\Omega(p_j) = (1 - p_j)u(K + Y - p_j l(p_j)) + p_j v(K + (1 - p_j)l(p_j)). \quad (14)$$

In Module 2 individuals face price  $\lambda_j = p_j^{\text{pos}}$  with probability  $q_j$  and price  $\lambda_j = p_j^{\text{neg}}$  with probability  $(1 - q_j)$ . Thus, in this scenario the value function for the interim expected utility in each of these scenarios, respectively, is

$$\Omega(p_j^{\text{pos}}) = (1 - p_j^{\text{pos}})u(K + Y - p_j^{\text{pos}}l(p_j^{\text{pos}})) + p_j^{\text{pos}}v(K + (1 - p_j^{\text{pos}})l(p_j^{\text{pos}})), \quad (15)$$

$$\Omega(p_j^{\text{neg}}) = (1 - p_j^{\text{neg}})u(K + Y - p_j^{\text{neg}}l(p_j^{\text{neg}})) + p_j^{\text{neg}}v(K + (1 - p_j^{\text{neg}})l(p_j^{\text{neg}})), \quad (16)$$

while *ex ante* expected utility in Module 2 is

$$q_j\Omega(p_j^{\text{pos}}) + (1 - q_j)\Omega(p_j^{\text{neg}}). \quad (17)$$

Thus, since  $p_j = q_j \cdot p_j^{\text{pos}} + (1 - q_j) \cdot p_j^{\text{neg}}$  we have  $\Omega(p_j) > q_j\Omega(p_j^{\text{pos}}) + (1 - q_j)\Omega(p_j^{\text{neg}})$  if the function  $\Omega(\cdot)$  is strictly concave (i.e.,  $\Omega''(\cdot) < 0$ ). The proof of this result is provided in the appendix. It is directly derived from a result in a somewhat different context in Hoy and Polborn (2000).

**Module 3:** In this module all (or some) individuals obtain genetic tests and insurance companies are **not** allowed to use this information in setting prices. There are three variations:

- 3.1. 5 percent of individuals obtain a genetic test.
- 3.2. 20 percent of individuals obtain a genetic test.
- 3.3. 100 percent of individuals obtain a genetic test.

Scenario 3.3 represents the maximum possible effect of private information on adverse selection costs created by genetic tests and a ban on their use for rate-making purposes. Scenarios 3.1 and 3.2 are more realistic scenarios for the near-term future since, as noted earlier, a small proportion of the population takes such tests, in part presumably due to the high financial cost.

The source of inefficiency in the life insurance market under asymmetric information arises from the fact that each person faces the same price. A person who tests negative will want to reduce her purchases of life insurance (underinsure) while a person who tests positive will want to increase her purchases (overinsure). The channel by which this efficiency cost is transmitted is the price system. With “better” risks buying less insurance and “worse” risks buying more insurance, the price that persists will be higher than the population-weighted death probability (i.e.,  $\lambda_e > p$ ). The price  $\lambda_e$ , which is referred to as the average clientele risk, is determined according to specific applications of Equation (13). First consider the scenario when everyone gets tested (Module 3.3). Computing the value of  $\lambda_e$  means finding the minimum value of  $\lambda$  that satisfies the equality of the expected total revenue (ETR) and the expected total cost (ETC) for each of the 13 risk groups ( $j = 1, 2, \dots, 13$ ). We will denote this value by  $\lambda_e^j$ . Thus, let  $n_j$  represent the number of individuals in risk group  $j$  and index these individuals by index  $i$ , ( $i = 1, 2, \dots, n_j$ ). Let  $q_j$  be the probability that any individual

in risk group  $j$  is determined by the genetic test to be carrying one of the BRCA1/2 genes. Then we have,<sup>15</sup>

$$ETR(\lambda_e^j) = \lambda_e^j \cdot \sum_{i=1}^{n_j} [q_j \cdot L^i(p_j^{\text{pos}}, \lambda_e^j) + (1 - q_j) \cdot L^i(p_j^{\text{neg}}, \lambda_e^j)], \quad (18)$$

$$ETC(\lambda_e^j) = \sum_{i=1}^{n_j} [q_j \cdot p_j^{\text{pos}} \cdot L^i(p_j^{\text{pos}}, \lambda_e^j) + (1 - q_j) \cdot p_j^{\text{neg}} \cdot L^i(p_j^{\text{neg}}, \lambda_e^j)]. \quad (19)$$

As discussed in the section on “Theoretical Background” we know some such value  $\lambda_e^j \in (p_j^{\text{neg}}, p_j^{\text{pos}}]$  exists.

The structure for price determination in Scenarios 3.1 and 3.2 is essentially the same as that of 3.3. In these cases only  $x$  percent will know their true genetic status ( $x = 5$  percent and 20 percent, respectively), hence only  $x \cdot q$  percent will actually know that they are a high-risk type. The smaller the fraction of the group holding private information about risk type, the smaller is the expected impact of adverse selection.

**Module 4:** *In this module, information is again asymmetric, as individuals can obtain a genetic test and insurance companies cannot ask for the results. We consider the specific case in which 20 percent of the individuals take a genetic test. Although insurance companies cannot ask for test results, we assume that those who test negative may present their test result to the insurer in order to obtain an actuarially fair (lower) insurance premium.*

This module reflects the possibility, allowed by some regulatory regimes, for insurers to accept negative test results and price accordingly but not to allow insurers access to positive test results. Thus, those who test negative, and who make up 20 percent  $\cdot (1 - q)$  of the population within a risk group, will receive price  $\lambda = p_j^{\text{neg}}$ . Those who test positive will be pooled with those who remain uninformed. Insurance companies offering insurance to those in this pooled group face a group composed of a fraction of 20 percent  $\cdot q$  who are informed that they have death probability  $p_j^{\text{pos}}$  while the uninformed (composed of 80 percent of the population within the overall group) will have a death probability  $p_j$ . The extent of adverse selection for this pooled group is in fact worse than in Module 3.2 when 20 percent of the people get tested because in this module nobody who tests negative will be in this group (i.e., only uninformed individuals and those who test positive form this pool).

For each of the modules we compute the compensating variation (CV) of the insurance opportunity implicitly by equating expected utility with no insurance,  $EU_{\text{no}}$ , to expected utility with insurance but with an amount of income taken away (CV) in both states of the world. Thus, we have for all modules,<sup>16</sup>

$$EU_{\text{no}}^j = (1 - p_j)u(K + Y) + p_j v(K). \quad (20)$$

The precise method of determining CV varies across the modules since individuals hold different information sets. In Module 1 individuals within each risk group face the same probability of death and so CV is determined according to

<sup>15</sup> As earlier, we suppress the variables  $a_i, Y_i, K_i$ , in  $L^i(\cdot, \cdot)$

<sup>16</sup> We suppress individual subscript  $i$  whenever doing so does not lead to confusion.

$$EU^j(CV^j) = (1 - p_j)u(K + Y - \lambda L - CV^j) + p_j v(K + (1 - \lambda)L - CV^j) = EU_{no}^j, \quad (21)$$

where  $L$  is the optimal amount of insurance demanded. In Modules 2 and 3 we determine separate values for the  $CV$  for those who test positive and for those who test negative or, in the cases of 3.1 and 3.2, including those who do not test at all. A similar process is applied in Module 4 noting that those who test negative are not pooled with any others and they receive price  $\lambda = p_j^{\text{neg}}$ . In each module we compute the per capita  $CV$  value and use this as our relative measure of efficiency. Note that the per capita  $CV$  in each module is based on the same benchmark value of expected utility with no insurance. This is important since if one were not to do this one would be led to rather odd conclusions. For example, if suddenly the probability of death were to become zero for all individuals, an admittedly unrealistic scenario, the willingness to pay for insurance would become zero. But this, of course, does not mean people become worse off.<sup>17</sup>

It is important to realize that our model focuses only on the issue of insurance market effects of genetic testing. For example, we do not explicitly account for the impact of the cost of the test to the insured. From a calculation perspective, doing so would be quite straightforward since the cost of the test would be deducted from wealth in both states of the world, and so the  $CV$  value would simply be reduced by this amount under all regulatory scenarios. However, our article is forward looking and we do not know the future cost of the test. Moreover, this cost would be the same whether or not there is a regulation that prohibits insurers from using genetic test results in rate making. We also do not attempt to model the value of genetic test results in choosing strategies to improve one's health. All of these factors would be very interesting to consider but represent potential future research.

## RESULTS

In this section our results are based on the elementary utility function  $u(x) = \ln(x)$ . In the section on "Sensitivity Analysis" we discuss the results obtained by using the other utility functions mentioned earlier. Full detail for the results mentioned here, as well as for some additional cases, are available in the technical appendix.

The average amount of insurance purchased, and the participation rate, does not vary much across risk groups in Module 1. In each of the risk categories 70.3 percent of women purchase insurance and, among those who participate in the market, the average demand for insurance varies between 390,400 and 390,900 across the risk groups, which implies an average replacement rate of 45 percent (i.e., women who purchase insurance on average buy an amount that would replace 45 percent of their income for their family's use should they die).

In Module 2 individuals continue to face actuarially fair prices, although, of course, those who test positive face a higher price reflecting their correspondingly higher probability of death. Since only 0.1 percent of this risk group would test positive, the probability of death changes very little for those who test negative relative to the pooled probability from Module 1. Not surprisingly, those who test negative have

<sup>17</sup> We also computed the equivalent variation for the insurance market opportunity. Although we do not report these values here, we obtained qualitatively similar results.

**TABLE 2**

Per Capita CV for Each Risk Group for Modules 1 and 2

| Group | Per Capita CV<br>Module 1 | Per Capita CV<br>Module 2 |
|-------|---------------------------|---------------------------|
| 1     | 4,017.3                   | 4,016.8                   |
| 2     | 4,018.3                   | 4,017.6                   |
| 3     | 4,048.1                   | 4,047.5                   |
| 4     | 4,050.4                   | 4,046.8                   |
| 5     | 4,053.4                   | 4,052.8                   |
| 6     | 4,054.4                   | 4,052.6                   |
| 7     | 4,073.1                   | 4,072.8                   |
| 8     | 4,078.3                   | 4,077.3                   |
| 9     | 4,114.7                   | 4,109.6                   |
| 10    | 4,124.9                   | 4,122.6                   |
| 11    | 4,186.9                   | 4,184.5                   |
| 12    | 4,646.1                   | 4,633.1                   |
| 13    | 4,950.1                   | 4,932.4                   |

very similar demand behavior as they would in Module 1. Those who test positive demonstrate a slight rise in the amount of insurance, varying from 394,200 to 398,900 across risk groups, for an average replacement rate of 45.5 percent. Thus, although a state contingent utility function allows for the amount of insurance to vary as the probability of death varies, we see that under risk-type-specific actuarially fair pricing demand behavior is quite stable for the range of variations in  $p$  that is relevant to our scenarios.

In Table 2 we provide the per capita compensating variation values of the insurance market for each risk group for both modules. As noted in the section on "Theoretical Background," individuals face premium risk in Module 2 and so some loss in per capita CV is expected. However, since  $q_j$  is very small, it is not too surprising that the extent that premium risk reduces welfare as measured by the CV value is small. Notice that, roughly speaking,  $q$  increases through the risk Categories 1 to 13, as does the reduction in CV associated with moving to the scenario of Module 2. In other words, the efficiency loss due to premium risk is increasing in  $q$ .

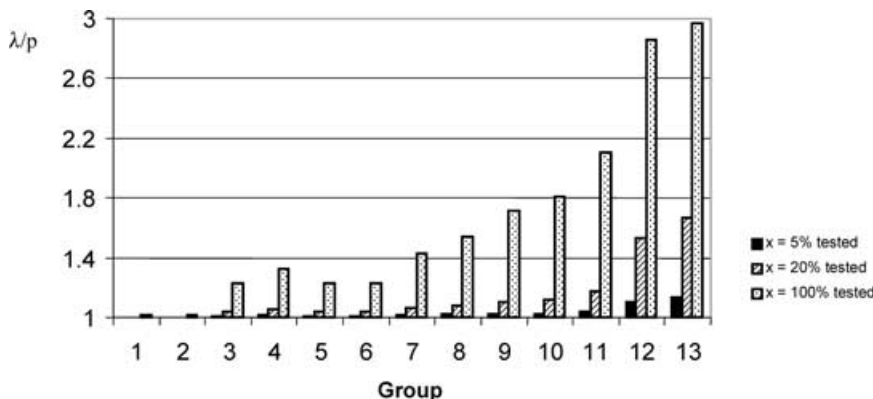
In Module 3 insurers are not allowed to use results of genetic tests that are known by insureds and so adverse selection occurs. Table 3 shows the ratio of the (pooled) equilibrium price to the weighted average of the death probability (and hence the *population*-weighted actuarially fair price) for each risk group (i.e., as defined by family background). This information is also illustrated graphically in Figure 1. In Risk Groups 1 and 2 the ratio  $p^{\text{pos}}/p^{\text{neg}}$  is substantially lower than for the other groups. Moreover, the fraction of individuals who would test positive is only 0.001. For these two categories ( $j = 1, 2$ ) the impact of adverse selection is to increase the price to a level not more than 1.5 percent above the population-weighted actuarially fair price (or probability of death). The price increases more for Groups 3–13, rising to a substantial price increase (relative to the average price in Modules 1 and 2) of a factor of almost 3 (for the case of 100 percent of people tested). Not surprisingly, the fewer people who hold private information, the lesser the extent of adverse selection.

**TABLE 3**  
Price Effects of Adverse Selection From Module 3

| Group | $\lambda/p$ (5% Tested) | $\lambda/p$ (20% Tested) | $\lambda/p$ (100% Tested) |
|-------|-------------------------|--------------------------|---------------------------|
| 1     | 1.001096                | 1.002826                 | 1.015513                  |
| 2     | 1.001096                | 1.002825                 | 1.015510                  |
| 3     | 1.011044                | 1.039654                 | 1.228484                  |
| 4     | 1.012870                | 1.052908                 | 1.327460                  |
| 5     | 1.011032                | 1.039657                 | 1.228249                  |
| 6     | 1.011086                | 1.039657                 | 1.228228                  |
| 7     | 1.014559                | 1.065742                 | 1.429708                  |
| 8     | 1.021978                | 1.078771                 | 1.538788                  |
| 9     | 1.025325                | 1.104114                 | 1.717541                  |
| 10    | 1.026945                | 1.116762                 | 1.807230                  |
| 11    | 1.038976                | 1.177189                 | 2.105987                  |
| 12    | 1.101366                | 1.528695                 | 2.860394                  |
| 13    | 1.135661                | 1.666390                 | 2.966983                  |

Note:  $\lambda/p$  is the ratio of demand-weighted price to population-weighted actuarially fair price.

**FIGURE 1**  
Price Effects for Module 3



Not surprisingly, looking at average insurance purchases by lower and higher risk types within these risk groups displays an expected pattern with average demand by lower risk types (i.e., those testing negative) falling compared to Modules 1 and 2 for the higher risk groups. The participation rate for those who test negative for the BRCA1/2 genes in Risk Group 13 falls from 70.3 percent in Modules 1 and 2 to 21.3 percent in the presence of adverse selection when 100 percent of people get tested, to 27.6 percent when 20 percent get tested, and to 42.3 percent when 5 percent get tested. There is a much smaller impact, however, for lower risk groups where both the fraction of people who are high-risk genotypes is smaller and the difference in death probabilities between genotypes is lower. Moreover, for those who test negative

**TABLE 4**  
Ranges of Demand Effects for Module 3

| Module             | Test Status | Average Replacement Rate |          | Percentage With Positive Demand |          |
|--------------------|-------------|--------------------------|----------|---------------------------------|----------|
|                    |             | Group 1                  | Group 13 | Group 1                         | Group 13 |
| 3.3<br>(100% test) | Negative    | 0.450                    | 0.124    | 66.6                            | 21.3     |
|                    | Positive    | 4.120                    | 0.920    | 100                             | 95.3     |
| 3.2<br>(20% test)  | Uninformed  | 0.448                    | 0.282    | 69.7                            | 32.6     |
|                    | Negative    | 0.445                    | 0.223    | 69.7                            | 27.6     |
|                    | Positive    | 4.201                    | 3.401    | 100                             | 100      |
| 3.1<br>(5% test)   | Uninformed  | 0.450                    | 0.346    | 69.7                            | 63.1     |
|                    | Negative    | 0.444                    | 0.291    | 69.7                            | 42.3     |
|                    | Positive    | 4.212                    | 6.061    | 100                             | 100      |

and continue to purchase insurance, their replacement rate falls by as much as from 45 percent to 12.4 percent. These results are summarized in Table 4.<sup>18</sup>

Note that, conversely, those who test positive have a revised probability of death that implies insurance is priced at less than the actuarially fair price. As a consequence, these higher risk types within each risk category purchase more insurance than they would have under risk-type-specific actuarially fair pricing. Their replacement rates range from 0.92 to 7.2, implying substantial degrees of overinsurance in some cases.<sup>19</sup> Of course, even those who test positive for one of the BRCA genes are not insensitive to price, and so even though the ratio  $p^{\text{pos}}/p^{\text{neg}}$  is very large for risk groups 12 and 13, the fact that participation in the insurance market by those who test negative is very low leads to a relatively high price and so a lower degree of overinsurance by those who test positive.

In Figure 2 we illustrate the per capita CV computations for each risk group associated with the insurance market opportunity when adverse selection is present.<sup>20</sup> Not surprisingly, we find a reduction in per capita CV in comparison to that in Module 2 in which insurers are allowed to risk-rate according to genetic test results and this reduction is greater the higher the fraction of the population that becomes informed. The loss in market efficiency is, roughly speaking, greater for those cases where adverse selection has a greater effect on price.

For the case of 100 percent tested, the price increase due to adverse selection for Risk Groups 1 and 2 is about 1.5 percent while the loss of efficiency (relative to Module 2) in

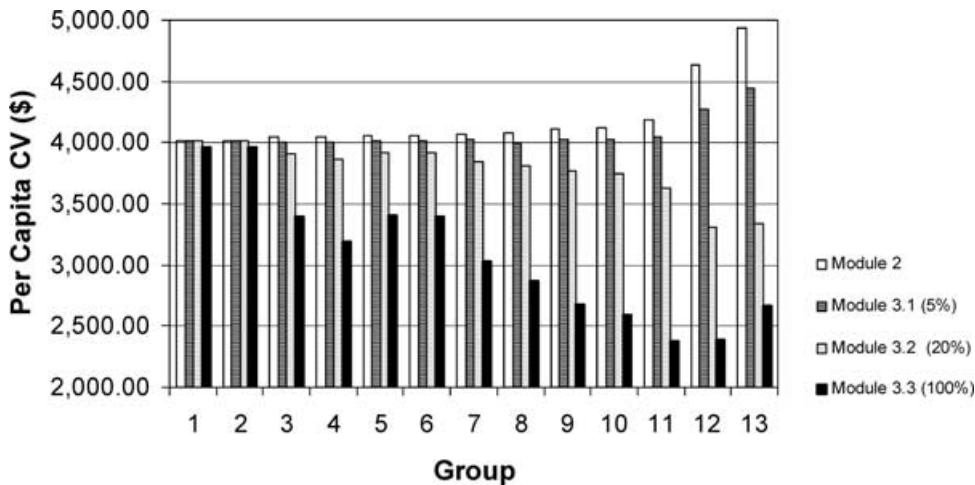
<sup>18</sup> Fully detailed results on the effect of adverse selection on quantity of insurance demanded for the scenario of Module 3.3 (100 percent get tested) are provided in the appendix.

<sup>19</sup> A replacement rate of 7 implies approximately \$3 million (CAD) coverage. In the United Kingdom there are some genetic tests for which insurers may require disclosure of any genetic test results for anyone demanding more than 500,000 pounds coverage, which is approximately \$1 million (CAD). Such limits clearly can limit the degree of adverse selection. However, only the Netherlands and the United Kingdom have adopted such restrictions.

<sup>20</sup> For detailed computations see the appendix.

**FIGURE 2**

Per Capita CV for Modules 2 and 3



terms of reduction in CV is modest at approximately 1.3 percent. The loss of efficiency, however, rises to as much as 43 percent for Risk Group 11. This comparison across risk groups demonstrates that one needs to consider separately different risk groups according to family background when measuring efficiency effects arising from a ban on insurers using genetic test results for risk-rating purposes.

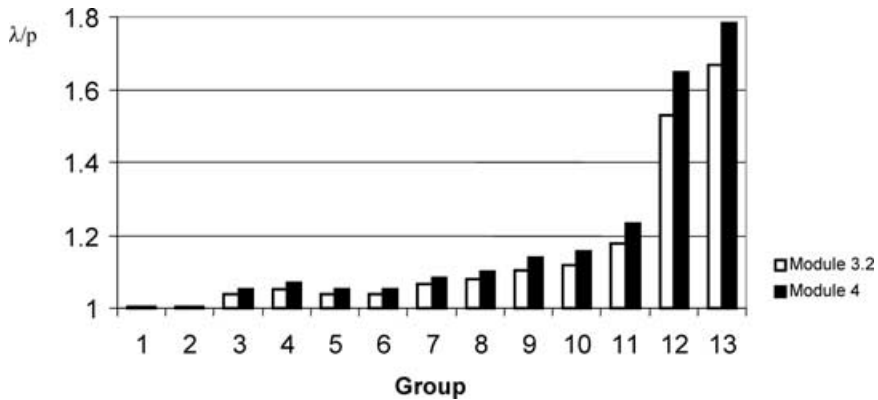
In Module 4 we reconsider the effect of a regulation that (i) allows individuals who have had a negative test result to present that result to an insurer and to receive a corresponding actuarially fair price but (ii) prohibits insurers from asking those with positive test results to disclose them. We assume 20 percent of the population becomes tested, which compares to the case of Module 3.2. Thus, only those who test negative will declare their test results and, as noted above, these people receive price  $\lambda = p_j^{\text{neg}}$ . Those who test positive keep this information private and are pooled with the uninformed consumers, thus creating an adverse selection scenario, which will be more intense than in the similar situation in Module 3.2, as described earlier. The comparison of the extent of adverse selection in this case and with that of Module 3.2 is illustrated in Figure 3 (see appendix for detailed calculations).

Thus, adverse selection conditions are in a sense worsened by allowing people who test negative to have their test results used to reduce their insurance price and this is relatively more important for higher risk groups. Although we do not present the results here, not surprisingly people in these adverse selection scenarios purchase less insurance coverage. However, those who do test negative and receive price  $\lambda = p_j^{\text{neg}}$  avoid adverse selection altogether and are clearly made better off by the opportunity provided by a regulation that allows people the freedom to present negative test results to the insurer.

### SENSITIVITY ANALYSIS

A more detailed description of results from various changes in our assumptions is available in the appendix. There are many assumptions that deserve attention from

**FIGURE 3**  
Price Effects of Module 4



the point of view of sensitivity analysis. Perhaps chief among these is the accuracy of the probabilities used to determine likelihoods of getting cancer for women of different family backgrounds and whether they have one of the BRCA1/2 genes. Also important is the determination of the 10-year survival rate for women diagnosed with cancer and the fraction of women, conditional on family background, who are assumed to have one of the BRCA1/2 genes. However, simply by the fact that we have a wide variety of such probabilities across our family backgrounds provides a good sense of how sensitive the operation of our life insurance model is to these factors in the direction of an increasing degree of genetic risk.

Another reason to consider a wide range of possible probability estimates (and ratios) in simulation exercises such as ours is that individuals' subjective perception of probabilities can vary from objective, data-based values. This is evident from the observation that, in some contexts, alternative choice models, such as prospect theory, cumulative prospect theory, and rank-dependent utility models display significantly better predictive ability over the standard expected utility model. Our trials with higher probabilities can reflect choice behavior, and hence adverse selection effects, in such instances where individuals behave according to probabilities that are upward biased when objective probabilities are relatively small.<sup>21</sup> We also chose a ratio  $p^{\text{pos}}/p^{\text{neg}}$  that is half the value of that in risk group 1. The result was, as expected, a reduction in the effect of adverse selection, with  $\lambda/p$  values consistently lower than for Risk Group 1 in all scenarios.

As  $q$ , the fraction of individuals in the risk group (as determined by family background) to have one of the BRCA1/2 genes varies across these groups, we can see significant sensitivity as to how the impact of adverse selection is realized in the insurance market. This is also true for the relative size of the probabilities of death for those with and without one of the BRCA1/2 genes (i.e., the ratio  $p^{\text{pos}}/p^{\text{neg}}$ ). The

<sup>21</sup> See, for example, the work of Quiggin (1982), Tversky and Kahneman (1992), and Wu and Gonzalez (1996). Also, see an application of using weighted probabilities in an adverse selection problem in insurance by Ryan and Vaithianathan (2003).

insurance market behavior under adverse selection is also very sensitive to the proportion of individuals in a risk group who obtain information from genetic testing. In fact, identifying these sensitivities is, we believe, the main contribution of this article.

For our presentation in the “Results section”, we assumed the particular elementary utility function representing CRRA preferences with degree of risk aversion of one (i.e., the logarithmic utility function). We also used constant relative risk aversion utility functions with degrees  $1/2$  (i.e., less risk averse) and  $3$  (i.e., more risk averse).<sup>22</sup> In terms of insurance market behavior under symmetric information a lower degree of risk aversion leads to a lower demand for insurance and a lower value, in terms of CV, for the insurance market opportunity as one would expect. Comparison for the numerical results across these cases is provided in the appendix. Most interesting is that the higher is the degree of risk aversion, the lesser is the extent of adverse selection as measured by the price effect (i.e., the value of  $(\lambda/p)$ ). When an individual faces an actuarially fair price for life insurance the “optimal rule” that determines insurance demand is to purchase that amount of insurance that equates marginal utility of income across states of the world (i.e., the life and death states). This is a straightforward application of the fundamental theorem of risk-bearing. However, when price varies from the actuarially fair rate, the individual adjusts her demand for insurance accordingly and so a wedge between the marginal utilities of income in these two states is created. In an adverse selection scenario, the low risk types consider the price to be excessive relative to their risk and so “underinsure” while the high risks do the opposite. This is the force that drives the price above the population-weighted average probability of death and so generates adverse selection costs. However, the higher the individual’s degree of risk aversion the smaller is the wedge that an individual is willing to accept. This means a dampening effect on both overinsurance and underinsurance. Hence, a higher degree of risk aversion will reduce demand by high risks and increase demand by low risks. Both of these effects will reduce the impact of adverse selection on the resulting price of insurance (i.e., the fact that greater risk aversion leads to less flight from insurance by low risk types and less overinsurance by high risk types). Our sensitivity results provided in the appendix demonstrate this intuition.

## SUMMARY AND CONCLUSIONS

In this article we have developed simulations of the market for 10-year term life insurance targeted at women aged 35 to 39 years under various information and regulatory scenarios concerning genetic testing based on the BRCA1/2 genes. We first generate benchmark results (Module 1) based on a model of rate-making in which genetic test results are not available to either insured or insurers but insurers have access to and use relevant family background to establish risk groups. In particular, we compute the compensating variation (CV) measure of the opportunity to insure in this environment for all women in the model. We then introduce information, available to

<sup>22</sup> Using values of the risk aversion parameter that lie outside the “usual range” suggested by the literature is an important exercise in this context. Both the theoretical and empirical literature recognizes the possible difference in risk aversion preferences for group (family in our case) decision makers compared to that for individual decision makers. See Wilson (1968) and Shupp and Williams (2006).

both insureds and insurers, about genetic test results for the BRCA1/2 genes for all individuals (Module 2). Under the assumption that insurers are allowed to use this information to risk-rate insurance policies, this results in separate premiums for those who test positive and those who test negative within each risk group, as determined by family background. The per capita CV is computed for this scenario as well and turns out to be slightly lower than that for Module 1. This result reflects the fact that in Module 2 individuals face premium risk in comparison to the single premium charged in Module 1.

In Module 3 we simulate the insurance market for cases in which 100 percent, 20 percent, and 5 percent of consumers have genetic tests for the BRCA1/2 genes given that insurers are banned by regulation (or mutual agreement) not to use this information to risk-rate premiums. The resulting impact of adverse selection is captured in a number of ways. For each family background risk group we consider (i) the effect on demand for life insurance according to whether individuals test positive or negative for one of the genes, (ii) the overall effect on the price of insurance in relation to the population-weighted actuarially fair price of insurance, and (iii) the effect on the per capita CV value of the insurance market opportunity. We find that for each of these criteria, the impact of adverse selection varies substantially across family background risk groups. This is because, roughly speaking, women with a stronger history of family members having had breast or ovarian cancer are more likely to test positive for one of the BRCA1/2 genes and so this exacerbates the impact of adverse selection on the market. Our analysis demonstrates the importance of considering separately the various risk groups as defined by family background when estimating or simulating the impact of a regulation banning insurers from using genetic test results. Also, not surprisingly, the extent to which adverse selection affects the market increases for scenarios in which a larger fraction of the population obtains genetic tests.

We also considered (in Module 4) the effects of a regulation that allows those who obtain genetic tests and test negative to provide their results to the insurance company in order to obtain a "discount." Those who test positive are allowed to keep this information private and so get pooled with uninformed individuals. The result of this scenario was a modest increase in the degree of inefficiency due to adverse selection in comparison with the scenario in which insurers are not allowed to use any test results, whether positive or negative, for risk-rating purposes. Of course, the option in such a regulation does improve welfare of those who test negative.

We do not think that our study should on its own be considered sufficiently comprehensive as to provide an answer to the question of whether life insurers should have access to insurance buyers' genetic test results. There are many aspects of genetic privacy besides the efficiency effects resulting from adverse selection. However, considering the efficiency effect of adverse selection should be a component of a general policy discussion concerning genetic privacy. Our simulation exercises suggest that at least for some family background types, if a sufficiently large fraction of women were to become informed about whether they have one of the BRCA1/2 genes, the efficiency effects may be quite substantial. If one were to consider the possibility of insurance buyers being informed about a wide range of genetic test results that have a significant impact on mortality, then this concern would be reinforced. On the other hand, if the fraction of informed individuals is quite small our results suggest that

the efficiency effects may well be quite low. Legislation that altogether bans the use of genetic test results for rate-making by life insurance companies could be difficult to reverse. Thus, we suggest that it may well be preferable to establish reasonably short-term moratoria (e.g., 5 years) banning insurers from using information from genetic tests with a review of the situation at the end of the 5-year term. In this way if the possibility of significant adverse selection costs from genetic privacy should eventually arise over time, then the terms of the moratorium could be reconsidered and alternative means of dealing with the equity issues revolving around the so-called genetic underclass could be considered.

## REFERENCES

- Abel, A., 1986, Capital Accumulation and Uncertain Lifetimes with Adverse Selection, *Econometrica*, 54: 1079-1097.
- Arias, E., 2002, *United States Life Tables 2000, National Vital Statistics Reports*, Volume 51, Number 3, (Hyattsville, MD: National Center for Health Statistics).
- Bernheim, B. D., L. Forni, J. Gokhale, and L. J. Kotlikoff, 2003, The Mismatch Between Life Insurance Holdings and Financial Vulnerabilities: Evidence from the Health and Retirement Study, *American Economic Review*, 93: 354-365.
- Blake, D., 1996, Efficiency, Risk Aversion and Portfolio Insurance: An Analysis of Financial Asset Portfolios Held by Investors in the United Kingdom, *The Economic Journal*, 106: 1175-1192.
- Bossert, W., and M. Fleurbaey, 2002, Equitable Insurance Premium Schemes, *Social Choice and Welfare*, 19: 113-125.
- Brockett, P. L., and E. S. Tankersley, 1997, The Genetics Revolution, Economics, Ethics and Insurance, *Journal of Business Ethics*, 16: 1661-1676.
- Brugiavini, A., 1993, Uncertainty Resolution and the Timing of Annuity Purchases, *Journal of Public Economics*, 50: 31-62.
- Cawley, J., and T. Philipson, 1999, An Empirical Examination of Information Barriers to Trade in Insurance, *American Economic Review*, 89(4): 827-846.
- Crocker, K. J., and A. Snow, 1986, The Efficiency Effects of Categorical Discrimination in the Insurance Industry, *The Journal of Political Economy*, 94: 321-344.
- Ford, D., D. F. Easton, M. Stratton, S. Narod, D. Goldgar, P. Devilee, D. T. Bishop, B. Weber, G. Lenoir, J. Chang-Claude, H. Sobol, M. D. Teare, J. Struewing, A. Arason, S. Scherneck, J. Peto, T. R. Rebbeck, P. Tonin, S. Neuhausen, R. Barkardottir, J. Eyfjord, H. Lynch, B. A. Ponder, S. A. Gayther, J. M. Birch, A. Lindblom, D. Stoppa-Lyonnet, Y.-J. Bignon, A. Borg, U. Hamann, N. Haites, R. J. Scott, C. M. Maugard, H. Vasen, S. Seitz, L. A. Cannon-Albright, A. Schofield, M. Zelada-Hedman, and the Breast Cancer Linkage Consortium, 1998, Genetic Heterogeneity and Penetrance Analysis of the BRCA1 and BRCA2 Genes in Breast Cancer Families, *American Journal of Human Genetics*, 62(3): 676-689.
- Hoy, M., 1984, The Impact of Imperfectly Categorizing Risk on Income Inequality and Social Welfare, *Canadian Journal of Economics*, 17(3): 557-568.
- Hoy, M., and P. J. Lambert, 2000, Genetic Screening and Price Discrimination in Insurance Markets, *The Geneva Papers on Risk and Insurance Theory*, 25: 103-130.

- Hoy, M., and M. Polborn, 2000, The Value of Genetic Information in the Life Insurance Market, *Journal of Public Economics*, 78: 235-252.
- Kitcher, P., 1996, *The Lives to Come: The Genetic Revolution and Human Possibilities* (New York: Simon and Schuster).
- Knoppers, B., B. Godard, and Y. Joly, 2004, Life Insurance and Genetics: A Comparative, International Overview, in: M. Rothstein, ed., *Genetics and Life Insurance: Medical Underwriting and Social Policy* (Cambridge, MA, London: MIT Press), 173-194.
- Lemmens, T., 2000, Selective Justice, Genetic Discrimination, and Insurance: Should We Single Out Genes in Our Laws?, *McGill Law Journal*, 45(2): 347-412.
- Lemmens, T., and P. Bahamin, 1998, Genetics in Life, Disability and Additional Health Insurance in Canada: A Comparative Legal and Ethical Analysis, in: B. Knoppers, ed., *Socio-Ethical Issues in Human Genetics* (Cowansville, PQ: Yvon Blais), 108-275.
- Macdonald, A., 2003, Genetics and Insurance: What Have We Learned So Far?, *Scandinavian Actuarial Journal*, 4: 324-348.
- Macdonald, A., and D. Pritchard, 2000, A Mathematical Model of Alzheimer's Disease and the APOE Gene, *Astin Bulletin*, 30(1): 69-110.
- Macdonald, A., and D. Pritchard, 2001, Genetics, Alzheimer's Disease, and Long-Term Care Insurance, *North American Actuarial Journal*, 5(2): 54-78.
- North American Actuarial Journal*, 1999, *Feature Articles on Genetic Technology and Underwriting: The 1998 Bowles Symposium*, volume 3, no. 1.
- Pauly, M. V., K. H. Withers, K. Subramanian-Viswana, J. Lemaire, and J. C. Hershey, 2003, *Price Elasticity of Demand for Term Life Insurance and Adverse Selection*, NBER Working paper no. 9925.
- Picone, G., F. Sloan, and D. Taylor Jr., 2004, Effects of Risk and Time Preference and Expected Longevity on Demand for Medical Tests, *The Journal of Risk and Uncertainty*, 28(1): 39-53.
- Quiggin, J., 1982, A Theory of Anticipated Utility, *Journal of Economic Behavior and Organization*, 3: 323-343.
- Ries, L. A. G., M. P. Eisner, C. L. Kosary, B. F. Hankey, B. A. Miller, L. Clegg, A. Mariotto, E. J. Feuer, and B. K. Edwards, (eds.), 2004, *SEER Cancer Statistics Review, 1975-2001* (Bethesda, MD: National Cancer Institute).
- Rothschild, M., and M. Stiglitz, 1976, Equilibrium in Competitive Insurance Markets: An Essay on the Economics of Imperfect Information, *Quarterly Journal of Economics*, 90(4): 630-649.
- Ryan, M. J., and R. Vaithianathan, 2003, Adverse Selection and Insurance Contracting: A Rank-Dependent Utility Analysis, *Contributions to Theoretical Economics (B.E. Journals in Theoretical Economics)*, 3(1): 1-18.
- Shupp, R. S., and A. W. Williams, 2006, *Risk Preference Differentials of Small Groups and Individuals*, unpublished mimeo, Department of Economics, Indiana University, p. 42.
- Subramanian, K., J. Lemaire, J. C. Hershey, M. Pauly, K. Armstrong, and D. A. Asch, 1999, Estimating Adverse Selection Costs from Genetic Testing for Breast

- and Ovarian Cancer: The Case of Life Insurance, *The Journal of Risk and Insurance*, 66(4): 531-550.
- Tversky, A., and D. Kahneman, 1992, Advances in Prospect Theory: Cumulative Representation of Uncertainty, *Journal of Risk and Uncertainty*, 5: 297-323.
- Villeneuve, B., 2000, Life Insurance, in: G. Dionne, ed., *Handbook of Insurance* (Dordrecht: Kluwer Academic Publishers), pp. 901-931.
- Villeneuve, B., 2003, Mandatory Pensions and the Intensity of Adverse Selection in Life Insurance Markets, *Journal of Risk and Insurance*, 70: 527-548.
- Wilson, C., 1977, A Model of Insurance with Incomplete Information, *Journal of Economic Theory*, 16: 167-207.
- Wilson, R., 1968, The Theory of Syndicates, *Econometrica*, 36(1): 119-132.
- Witt, J., 2004, *The Effect of Information in the Utilization of Preventive Health Care Strategies: An Application to Breast Cancer*, Unpublished mimeo, Melbourne Institute of Applied Economic and Social Research, The University of Melbourne, Australia.
- Wu, G., and R. Gonzalez, 1996, Curvature of the Probability Weighting Function, *Management Science*, 42: 1676-1690.

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