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HEALTH INSURANCE, GENETIC TESTING AND ADVERSE SELECTION

By R. D. MacMinn, P. L. Brockett and J. A. Raeburn

ABSTRACT

The implications of genetic testing information availability for society, medicine, employment, and individual privacy rights have generated much political debate, legislation and academic research. Part of this debate centres on the ethical and economic considerations resultant from this expanded knowledge, particularly for insurance practices. Within insurance economics, the possibility of adverse selection has been debated and the potential for a ban on an insurer's use of genetic testing has been studied with respect to whether or not such a ban might actually result in insurance market failure due to this adverse selection. Studies have examined the issue using expected loss cost (actuarial or 'fair') pricing models, and have not considered either equilibrium (supply and demand) price setting as is present in markets, or the potentially swamping effect of background health care risks facing the insured, having nothing to do with any particular genetic mutation. Here we construct a supply and demand function with both high and low risk individuals in the presence of background health care cost risks, and derive an equilibrium price and market composition to determine whether, if genetic information is allowed for individuals, but this same information is not shared with insurers: (1) is market failure inevitable? (it is not if the background risk is sufficiently high relative to potential genetic risk costs); (2) will equilibrium prices result in all low risk insured exiting the market? (not in the presence of significant background risk); and (3) how much would prices increase and market sales decrease if insurers do not have the same genetic information as the insured? (prices will increase, but not necessarily very much in the presence of background risk, and not as much as that previously estimated in the insurance literature).

KEYWORDS

Genetic Testing; Equilibrium Pricing; Background Risk; Adverse Selection

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Genetic testing has the potential to revolutionize medicine. But revolutions can have casualties.
Francis Collins,
Director of the Human Genome Project, *Newsweek*, 23 December 1996

All progress is precarious, and the solution of one problem brings us face to face with another problem.
Martin Luther King

1. Background on the Genome Project and the Political/Social Impetus for Restricted Information Distribution

After the initial planning process for the human genome project culminated in 1990 with the publication of a joint research plan, 'Understanding Our Genetic Inheritance: The U.S. Human Genome Project. The First Five Years FY 1991-1995' (NHGRI, 1990), what was supposed to be a 15-year project to map the human genome began. On 26 June 2000, however, the International Human Genome Sequencing Consortium announced the working draft of the human genome,[1] and on 15 February 2001 the complete human genome sequence was announced in the two leading scientific journals, *Nature* (NIH/DOE) (Lander *et al.*, 2001) and *Science* (Celera) (Venter *et al.*, 2001) almost five years ahead of schedule. On 30 May 2007 the first actual complete map of the genetic code of a particular individual was developed, that of James Watson, the discoverer of the three dimensional structure of DNA, i.e., see ABC (2007). While the successful completion of the project provides great hope for remarkable advances yet to come in the medical sciences, it also raises fears. In his 9 September 2000 remarks President Clinton said:

> "The fear of misuse of private genetic information is already very widespread in our nation. Americans are genuinely worried that their genetic information will not be kept secret, that this information will be used against them. As a result, they're often reluctant to take advantage of new breakthroughs in genetic testing — making a point I think we cannot make too often — if we do not protect the right to privacy, we may actually impede the reach of these breakthroughs in the lives of ordinary people, which would be a profound tragedy."[2]

Responding to a perceived need to protect people from abuses (or exploitation) of their genetic information, 44 of the 50 United States of America have legislation or moratoria prohibiting the use of genetic testing results in underwriting health insurance. On 21 May 2008 U.S. President G. W. Bush signed The Genetic Information Nondiscrimination Act, which prohibits employers and those selling health insurance from using genetic information to discriminate in employment, deny coverage, or charge higher premiums to healthy people. Currently there is a moratorium in effect on

[1] See http://videocast.nih.gov/ram/ihgsc062600.ram
[2] See http://www.genome.gov/10001356

insurers' use of genetic testing information in the United Kingdom, established originally in 2001 for five years, and recently extended until 2011.[3] Other countries in Europe ban its use with either a moratorium or legislation. The concern which prompted these bans and moratoria is widespread, because, if insurers or employers use genetic test results for profit, there is the potential to create an 'underclass' of uninsurable risks and another 'underclass' of unemployable risks, who find themselves in these positions because their genetic codes render them higher risk (and consequently more expensive to an employer or insurer) than is deemed acceptable in a competitive market. On the other hand, if insurers or employers are prohibited from using genetic testing results, then another problem is created. In this latter situation, the insurance and labour markets will be characterised by hidden information, as the individuals who possess the increased risk will have access to the genetic information through genetic tests, while the organisation which bears the risk (the insurer or employer) do not, and this lack of transparency also has the potential to create significant social costs. One such cost is due to adverse selection, wherein individuals behave differently, for example increase insurance amounts, etc., because they have this hidden information to the detriment of the risk bearer. The adverse selection problems created in insurance markets necessitate increased premia as the pool of insured risks becomes more heavily dominated by the poorer risks. It is still too early to measure the extent of the adverse selection problem which will occur if the moratoria on insurer access to genetic testing information in various countries and states become permanent. At its worst, the adverse selection problem can cause market failure, as, either consumers drop out of the market due to premiums spiraling upward, or insurers drop out of the market due to the escalating costs of providing the insurance and increased uncertainty in setting adequate premiums, or both. Should insurers have access to genetic test results? If not, then how costly will the adverse selection problem be? If so, then will it create groups which are uninsurable or unemployable? Should the state be an insurer of last resort or should 'assigned risk pools' be created and individuals allocated back to insurers in the 'standard' market, as is done in automobile insurance?

[3] The United Kingdom moratorium began on 1 November 2001. The moratorium does not ban the genetic test for Huntington's disease, as that test had previously been approved by the Government's Genetics and Insurance Committee. The moratorium has limits, so that policies exceeding the limits are exempt from the moratorium. However, U.K. insurers have agreed not to ask to see the results of any genetic test already performed on an individual, or to require that a genetic test be done when selling life insurance up to £500,000 or critical illness insurance up to £300,000. According to the Association of British Insurers, this ban covers an estimated 97% of policies sold. See http://www.doh.gov.uk/genetics/gaic/index.htm for more information. On 14 March 2005 it was announced that an agreement had been reached with the British insurers to extend the moratorium to 2011 (MNT, 2005).

2. Adverse Selection against Insurers with Genetic Testing: Review of the Literature

The literature on adverse selection in a genetic testing context is primarily concerned with the private and social value of the information derived from the testing (for example, Crocker & Snow, 1992; Doherty & Thistle, 1996; Doherty & Posey, 1998; and Hoy & Polborn, 2000).[4] These papers generate equilibria in economies characterised by symmetric and asymmetric information. The equilibria are primarily separating equilibria. The separation is difficult in practice for the insurance industry, and is not the point pursued here. The concern here is with the consequences for the market if the separation is prohibited due to moratoria or legislation banning the use of the genetic test findings. These prohibitions necessitate a pooling which might not otherwise exist and which exposes the market to the full magnitude of the adverse selection problem. There is another thread of the literature which is more closely related to ours (for example, MacDonald, 1999; Subramanian *et al.*, 1999; Lemaire *et al.*, 2000; and Armstrong *et al.*, 2003). This thread of the literature is more concerned with the measurement of cost due to adverse selection, and uses Markov models to formulate the problem and measure the cost. The Markov models have generated evidence on adverse selection costs. There is a price inelastic assumption in the current versions of these Markov models, however, that limits the extent of the adverse selection problem which can be revealed. Accordingly, the analysis here introduces a model in which the insurance demand and supply are endogenously determined, and so allow price elastic behaviour. We will suppose that the market is characterised by pooling equilibria due to moratoria. This will allow the full impact of the adverse selection problem to be investigated. The initial results show that if the expected cost differential between those testing positive versus negative for a genetic mutation is sufficiently large then the market may be characterised as a 'lemons' equilibrium, in which only the bad risks stay in the market; equivalently, this case corresponds to a severe adverse selection problem. For smaller expected cost differentials the market may be characterised by multiple equilibria, some of which are unstable. The context in which the discussion of this paper is framed is that of health insurance (the most important context in the U.S.A., where health insurance is primarily a voluntary employment-related employee benefit, as opposed to many European countries, wherein nationalised health insurance minimises the importance of adverse selection).

[4] An extensive literature exists on the adverse selection problem outside the context of genetic testing, however, for example Dionne & Doherty (1992). This, along with the more recent literature, neither gauge the extent of the adverse selection problem nor consider the costs and benefits of eliminating the adverse selection problem and moving to an equilibrium with transparent contracts.

The paper is structured as follows. Section 3 provides a hidden information model in which individuals test positive or negative for a genetic mutation. In Section 4 the individual demand functions are derived, and the insurer supply functions are in Section 5. Because of the adverse selection problem, the supply functions are not independent of demand. The character and magnitude of the adverse selection problem are considered. Section 6 provides some perspective on what remains to be done as a consequence of the potential adverse selection problems in health insurance markets.

3. Hidden Information Model

Suppose that each applicant for a health insurance policy has had a genetic test for pathological mutations in one of the genes BRCA1 and BRCA2, that the tests are accurate as well as reproducible and that they have been validated by family studies which demonstrate an abnormal mutation in people with early onset breast cancer and (mainly) the normal pattern in people without breast cancer. Such tests divide the 'at risk' population into two groups: those who test positive, and have an increased risk of contracting breast cancer; and those who test negative (i.e. are at lower risk). Alternatively, one might consider a population where a particular pattern of the ApoE gene (ApoE4) is associated with a risk of developing late onset Alzheimer's disease, which is ten times the risk of those with the other ApoE patterns (either ApoE2 or ApoE3). Those who have the ApoE4 pattern are at additional risk of Alzheimer's disease, compared with their negative (non-ApoE4) relatives. Both types of genetic test differentiate between high risk and low risk groups. Whether this is recognised by finding a mutation (e.g. in BRCA) or by finding a high risk genetic pattern (e.g. in ApoE) does not affect our calculations of the influence of symmetrical or asymmetrical information between insurers and their customers.

In a health insurance market characterised by symmetrical information, the insurers would be able to sell different policies at different prices based on the test results, possibly even expanding the market. Figure 1 illustrates the difference in risks for BRCA1 mutations in a subject with a family history of breast or ovarian cancer and in which a known pathological mutation has been identified in a parent. The risk of cancer is lower for those testing negative for BRCA1 or 2 and much higher for those testing positive. If such a genetic test has been performed on an applicant for insurance, then significant asymmetry of information occurs if test results are not shared. If shared, different policies could be priced and sold; in such cases the state could perhaps subsidise the increased premiums of those at greatest risk. The spectre of adverse selection is clear if information sharing is not required.

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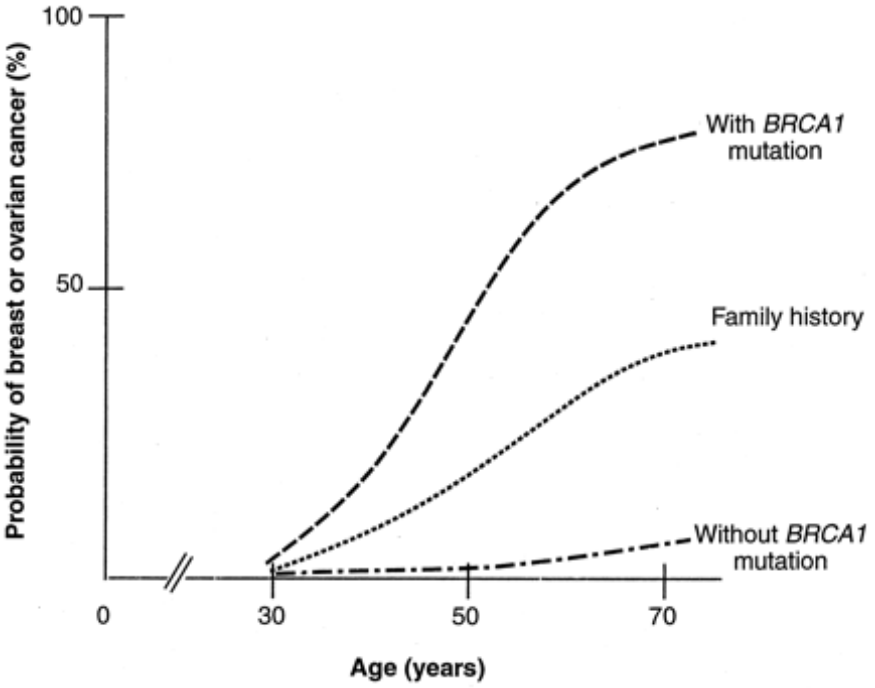

Figure 1. Statistical likelihood of breast or ovarian cancer (reproduced with permission from AAAS, Ponder, 1997)

In health insurance markets characterised by asymmetric or hidden information, insurers are not able to sell different policies at different prices based on the test results. Indeed, countries in North America and Europe either have regulations prohibiting insurance companies from asking or from using the results of genetic tests or moratoria requiring essentially the same forbearance. If an insurance market is characterised by hidden information due to regulations or moratoria, then a classic adverse selection problem may exist in the market. If the premium on the health insurance policy is set at an actuarially fair level, then those testing negative have the incentive to reduce their coverage or exit the market; that, in turn, changes the characteristics of the insured pool, and the premium must be raised to cover expected losses; that exacerbates the incentive for those testing negative to further reduce coverage, and so increases the actuarially fair premium, etc. In the limit, only those testing positive may remain in the market if the premium is still economically feasible for them.

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4. Demand

Consider the behaviour of individuals seeking health insurance coverage in a voluntary market. The demand is a behavioural function which indicates the maximum number of contracts which individuals are willing to buy at each possible premium. We suppose that the individuals seeking coverage are characterised by constant absolute risk aversion and suffer losses which have a Poisson distribution during the period covered by the premium. This assumption is developed by assuming that the claim arrivals follow a Poisson process, which corresponds to a 'random arrival' of claims, stationarity over time, and for which the number of claims arriving over distinct subintervals of time are independent variables (a characterisation of the Poisson process). For any fixed time period (like the policy period), it follows that the claim amount during the period has a Poisson distribution.

The Poisson probability model is used for several reasons. First and foremost, it allows us to endogenously derive a demand and supply function, and hence to determine prices in a market equilibrium setting, so that the effects of adverse selection on demand, supply, and prices can be determined in a market-based consumer economic model. Second, for health care insurance where individuals can experience multiple bouts of heath care costs during the policy period, it allows for the examination of the relative influence of the background health hazard versus the losses due to health concerns related to the disease on the impact of adverse selection on prices and demand. In a longer-term life insurance context (as opposed to health insurance), the Poisson model is already used to model the cost dynamics of a portfolio of risks (the collective risk model). While the focus of this paper is more conceptually aligned with the shorter-term health insurance market, where multiple events can occur within a period, it is also applicable to the life insurance model. The Poisson model is flexible, as it can arise, conceptually, as a limit of binomial models or can itself be subject to a central limit for Gaussian approximations. A further advantage of the Poisson distribution in our context is that it allows the explicit formulaic presentation of supply and demand so that general conclusions can be drawn (as opposed to simply having numerical solutions to equilibrium equations). Finally, this choice highlights the link between expected utility and demand and the consequent effect of risk aversion on equilibrium derived prices. We are able also to see from the explicit modelling that multiple equilibrium prices are possible, which might be hard to ascertain from numerical simulations which would be necessary if another distribution for claims were assumed.

We assume that there are two types of individuals: those with a genetic mutation, e.g. BRCA1, or a high risk genetic profile, e.g. ApoE4; and those without this mutation or profile. The Poisson distribution of claims for an individual of type j, Λ_j, can be decomposed into two parts $\Lambda_j = \Gamma + \Delta_j$, where

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Γ denotes the (random) losses due to health concerns unrelated to the mutation, with expectation γ, and Δ_j represents the random losses due to health concerns related to the disease whose incidence is affected by the mutation. The expected value of the claims for an individual of type i is λ_j. One may conceptualise the expected cost of the treatment directly related to the disease whose incidence is governed by the genetic mutation under investigation δ_i, as $\delta_j = \pi_i \delta$, where δ is the expected cost of the treatment directly related to the disease under examination and π_i is the probability that an individual of type j contracts the disease under study. Both those with the mutation and those without the mutation also experience losses due to background health risks Γ (accidents, other health costs, etc.), but the likelihood of contracting the genetically expressed disease differs between the two types. Table 2 in Hirschhorn *et al.* (2002) gives a list of 166 diseases associated with genetic abnormalities and their relative frequencies in the population.

The inclusion of two risks allows the effect of the relative severity of the expected losses due to the genetic mutation to be examined in the context of background risk. Without background risk, there would be no motivation for those without the genetic abnormality to purchase health insurance. With background risk, lower risk individuals who are risk averse may be motivated to stay in the risk pools, provided that the expected premium costs for the aggregate pool are not too high due to the inclusion of the high risk individuals. See Figure 4 versus Figure 5. The constant absolute risk aversion assumption does eliminate any income effect, but does allow the derivation of an elastic demand function, and the utility and distribution assumptions allow a simple derivation of the demand functions for those testing positive and negative. The following notation is used in the development of the demand:

W	wealth *now*;
j	$j = 1$ for no mutation and $j = 2$ for mutation;
Λ_j	random loss per type j risk: $\Lambda_j = \Gamma + \Delta_j$, where Γ = losses unrelated to the mutation, and Δ_j = losses due to health concerns related to the mutation;
n_j	proportion of risk $j = 1, 2$ insured;[5]
p	insurance premium for full coverage;
m_j	number of risks of type $j = 1, 2$;
r	interest rate in the financial market;
W_j	random wealth *then*, i.e., $W_j \equiv (w - pn_j)(1 + r) - (1 - n_j)\Lambda_j$;
a	measure of absolute risk aversion; and
$-e^{-aW_i}$	constant absolute risk aversion utility function.

[5] This is one minus the retained coinsurance proportion. As noted by Arrow (1963), the use of coinsurance contracts can arise naturally when adverse selection and moral hazard problems are present, such as in health insurance contracts, since this provides an incentive for the insured to mitigate losses, and can decrease the demand for insurance.

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Each risk type j has Λ_j losses, each of size one dollar, where Λ_j is Poisson (λ_j). The individual selects the proportion of full coverage to buy n_j, in order to maximise the expected utility of wealth *then*. This expected utility is:

$$\begin{aligned} \mathrm{E}[-e^{-aW_j}] &= -\mathrm{E}[e^{-a((w-pn_j)(1+r)-(1-n_j)\Lambda_j)}] \\ &= -e^{-a(w-pn_j)(1+r)}\mathrm{E}[e^{(1-n_j)\Lambda_j}] \\ &= e^{-a(w-pn_j)(1+r)}M_{\Lambda_j}(a(1-n_j)) \end{aligned} \tag{1}$$

where M_{Λ_j} is the moment generating function for Λ_j. Since the moment generating function for a Poisson random variable is:

$$M_{\Lambda_j}(t) = \exp\{\lambda_j(e^t - 1)\} \tag{2}$$

it follows that:

$$M_{\Lambda_j}(a(1-n_j)) = e^{\lambda_j(e^{a(1-n_j)}-1)}.$$

Thus, (1) may be equivalently expressed as:

$$\begin{aligned} E[-e^{-aW_j}] &= -e^{-a(w-pn_j)(1+r)}M_{\Lambda_j}(a(1-n_j)) \\ &= -e^{-a(w-pn_j)(1+r)+\lambda_j(e^{a(1-n_j)}-1)}. \end{aligned} \tag{3}$$

Observe that maximising the expected utility in (3) is equivalent to minimising the expression in equation (4) with respect to n_j:

$$-a(w-pn_j)(1+r) + \lambda_j(e^{a(1-n_j)} - 1). \tag{4}$$

The first order condition for an interior minimum is:

$$ap(1+r) - a\lambda_j\, e^{a(1-n_j)} = 0. \tag{5}$$

Equation (5) may be equivalently stated as follows:

$$p(1+r) = \lambda_j\, e^{a(1-n_j)} \Leftrightarrow e^{a(1-n_j)} = \frac{p(1+r)}{\lambda_j} \Leftrightarrow a(1-n_j) = \ln\left(\frac{p(1+r)}{\lambda_j}\right). \tag{6}$$

The insurance demand of an individual risk j is bounded between zero and one, and so the demand is of the following form:

$$n_j = \min\left\{1, \max\left\{0, 1 - \frac{\ln\left(\frac{p(1+r)}{\lambda_j}\right)}{a}\right\}\right\}. \tag{7}$$

Suppose that $j = 1$ indicates the individual who tests negative for the genetic

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mutation and let the parameters be $a = 0.2$,[6] $\lambda_1 = \$1{,}000$, and $r = 0.05$, where these parameters represent the measure of risk aversion, the expected loss and the interest rate, respectively. Then the demand function for the individual testing negative is depicted as $n_1(p)$ in Figure 2. If $j = 2$ indicates the individual who tests positive for the genetic mutation and that individual has an expected loss of $\lambda_2 = \$10{,}000$, then the demand for the individual testing positive is given by $n_2(p)$ in Figure 2.[7] It may be noted that all risk types demand full coverage at all premia below the present value of the expected loss and that the demand for coverage drops off significantly at higher premia. From (7) it is clear that the rate at which the individuals reduce coverage does depend on how risk averse they are.[8] In Figure 2[9] the premium is represented on the horizontal axis while the proportion of full coverage is measured along the vertical axis.

Aggregating demand across all type j risks results in the aggregate demand for this risk type $d_j(p)$, i.e.:

$$\begin{aligned} d_p(p) &= m_j n_j \\ &= m_j \min\left\{1, \max\left\{0, 1 - \frac{\ln\left(\frac{p(1+r)}{\lambda_j}\right)}{a}\right\}\right\}. \end{aligned} \tag{8}$$

[6] The measure of absolute risk aversion is an estimate from Ventura & Eisenhauer (2003). Also see Halek & Eisenhauer (2001).

[7] This scenario corresponds roughly to the parameters in Figure 1, where there is a probability of approximately $\pi_1 = 0.05$ for a person who tests negative for BRCA1 contracting breast cancer before age 70, and a probability of approximately $\pi_2 = 0.75$ for a person who tests positive for BRCA1 contracting breast cancer before age 70. The ratio of expected costs of $10 = \lambda_2/\lambda_1 = (\gamma + \pi_2\delta)/(\gamma + \pi_1\delta)$ occurs when the expected cost of cancer treatment δ is 36 times more than the expected cost of background health care cost γ. For shorter duration contracts or lower ages, the ratio π_2/π_1 can be as much as 50, in which case the ratio of $10 = \lambda_2/\lambda_1$ occurs when the ratio $\delta/\gamma = 0.225/\pi_1$, which, for $\pi_1 = 0.0125$, is approximately 18.

[8] The price elasticity of demand for risk j is:

$$\varepsilon_j = -\frac{\frac{dn_j}{n_j}}{\frac{dp_j}{p_j}} = \frac{1}{a - \ln\left(\frac{p(1+r)}{\lambda_j}\right)}.$$

At the fair premium for risk j, $p = \lambda_j/(1+r)$ and the elasticity is $1/a$.

[9] This figure may be viewed at http://www.livemath.com/lmstorage/files/1181781523669/indivi1.html. It does require a LiveMath plug-in which should, with permission, be automatically installed. The figure animates the changes in individual demands for changes in risk aversion. The risk aversion and other parameters may be changed to see the impact on demand.

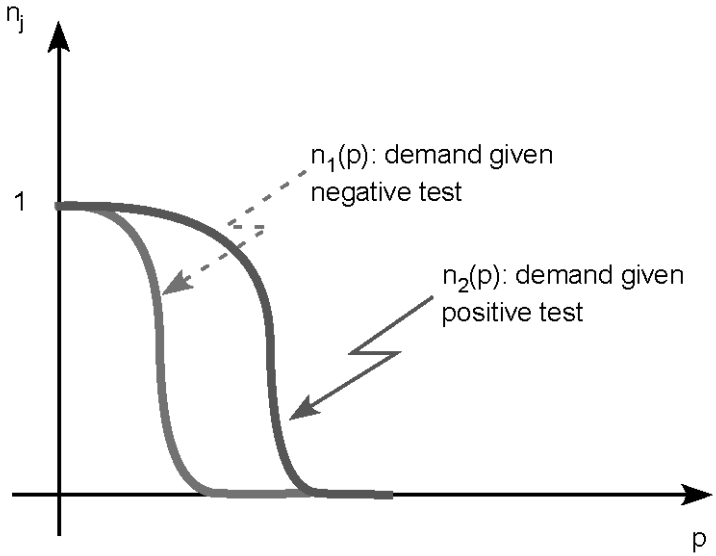

Figure 2. Individual demand by risk type as a proportion of full coverage

The aggregate demand for each risk type is shown in Figure 3, where again $d_2(p)$ indicates those testing positive (having the mutation) and $d_1(p)$ indicates those testing negative for the genetic mutation. The population is assumed to be 100,000, with one in 50 testing positive for the mutation.[10] The other parameters are the same as previously assumed, $\lambda_1 = \$1{,}000$, $r = 0.05$, and $\lambda_2 = \$10{,}000$. Each demand becomes less elastic as the measure of absolute risk aversion increases. The aggregate demands by risk type are shown in Figure 3.[11]

Finally, aggregating across all risks yields the market demand $d(p)$ is:

[10] The incidence of BRCA1 or BRCA2 mutation in the Ashkenazi Jewish population is over one in 50, i.e., see Struewing *et al.* (1997), as is the incidence of the ApoE epsilon 4 mutation associated with Alzheimer's and other diseases (estimated to be 0.23 in Hirschhorn *et al.*, 2002).

[11] This figure may be viewed at http://www.livemath.com/lmstorage/files/1181781523669/aggreg1.html. The figure animates the changes in aggregate demands for changes in risk aversion. The risk aversion and other parameters may be changed to see the impact on aggregate demand.

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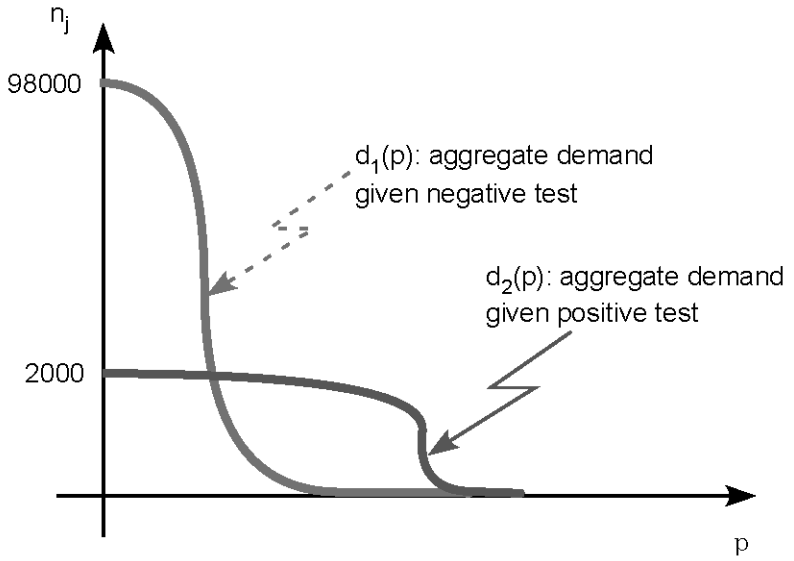

Figure 3. Aggregate demand by risk type
(the total population is 100,000, with one in 50 testing positive)

$$
\begin{aligned}
d(p) &= d_1(p) + d_2(p) \\
&= m_1 \min\left\{1, \max\left\{0, 1 - \frac{\ln\left(\frac{p(1+r)}{\lambda_1}\right)}{a}\right\}\right\} \\
&\quad + m_2 \min\left\{1, \max\left\{0, 1 - \frac{\ln\left(\frac{p(1+r)}{\lambda_2}\right)}{a}\right\}\right\}. \qquad (9)
\end{aligned}
$$

5. Supply

In a transparent economy (an economy with no hidden information), the insurers would be able to separate risks and sell insurance contracts designed for each risk type. The supply would be derived independent of demand. This is because the characteristics of the pool to be covered would be known to the insurer. Hence, the premium could be calculated and the quantity

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supplied determined by the characteristics of the pool, which would be known and determined without reference to demand. This leads to the actuarial or supply side pricing, which is independent of market supply/demand considerations and focuses on insurer needed prices for profitability. This is why (in part) insurers use classification and underwriting to segregate their market for separate pricing within segmented pools. On the other hand, in an economy characterised by hidden information, the supply is not independent of demand. With hidden information the pool is composed of all risk types, but the low risks exit the pool more rapidly than the high risks as the premium is increased, and so the premium determines the demand, which, in turn, determines how many of each type will be in the pool (the pool characteristics). This determines the expected losses and hence the premiums, which, in turn, determine the demands by the different risk types, which, in turn, determine the characteristics (composition) of the risk pool and supply, etc. Thus, the premium, demand, pool composition and supply are all interrelated.

Consider the insurance firms operating in this insurance market characterised by the two risk types. Consider the following additional notation:

S insurer surplus *now*;
$\theta(p)$ proportion of contracts purchased by type one risks or equivalently negative testers;
L the random loss per exposure unit (which depends on the composition of the pool which depends on the premium);
N the number of standard exposures which the insurer chooses to cover, i.e., number of contracts sold now; and
Π the random firm payoff *then* $\Pi = (pN + S)(1 + r) - NL(p)$.

The random loss of the insurance company is a linear combination of Poisson random variables corresponding to the two risk types, and the proportion of the demand from each risk type determines the combination coefficients. The proportion of the demand from those testing negative, i.e. type one risk, is:

$$\theta(p) = \frac{d_1(p)}{d_1(p) + d_2(p)}. \tag{10}$$

The random loss per exposure unit is then:

$$L(p) = \theta(p)\Lambda_1 + (1 - \theta(p))\Lambda_2. \tag{11}$$

The loss per insured in (11) shows how the proportion of each risk type changes, and hence the characteristics of the insured pool changes as the premium changes. As the premium increases, the better risks, that is those

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testing negative, have an incentive to exit partially or completely from the insured pool, and so the characteristics of the pool will change. The expected loss per insured in the pool at the premium p is:

$$\begin{aligned} \mathrm{E}L(p) &= \theta(p)\mathrm{E}\Lambda_1 + (1-\theta(p))\mathrm{E}\Lambda_2 \\ &= \theta(p)\lambda_1 + (1-\theta(p))\lambda_2. \end{aligned} \tag{12}$$

The insurer will be modelled as a risk neutral agent in the economy. The risk neutral assumption is often made for simplicity. However, finance theory also gives us an additional reason to use this assumption for a publicly traded insurance firm. As individual investors, themselves, can diversify away idiosyncratic or firm specific risk in the stock market by holding a well diversified portfolio, there is no reason why they should pay extra (a risk premium) for the insurer to bear this risk. While this is an approximate argument for publicly traded firms, in a competitive market, other insurers' pricing structures must also reflect this very low risk premium level or lose the business in pricing competition. Hence, risk neutrality is the most innocuous assumption from the perspective of the adverse selection cost.

The risk neutral insurer selects an operating decision, that is N, to maximise the expected payoff. The expected payoff for the company is then:[12]

$$\mathrm{E}\Pi = (pN + S)(1+r) - N\mathrm{E}L(p) \tag{13}$$

and the first order condition for expected profit maximisation is

$$\frac{d}{dN}\mathrm{E}\Pi = p(1+r) - \mathrm{E}L = 0 \tag{14}$$

which happens when:

$$p = \frac{\mathrm{E}L(p)}{1+r}. \tag{15}$$

Equation (15) says that the premium on the next contract must equal the present value of the expected loss on that contract. The premia which equal the present value of the expected unit loss are shown in Figures 4 and 5, and they represent the types of possible economic equilibria in the

[12] We may incorporate fixed and variable costs as well as the target profit level in the insurer profit equation by including these as a constant and into $L(p)$. The resulting equation differs only slightly from (14) and the qualitative results are the same, so this particular complication is ignored in the sequel in the interests of parsimony.

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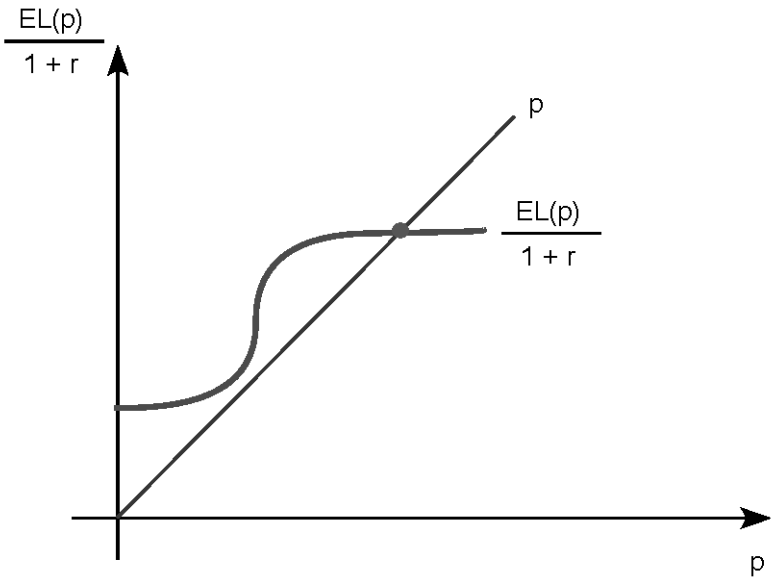

Figure 4. The lemons equilibrium
(the 45° line denotes the premium and the expected loss curve denotes the present value of the expected loss per exposure unit; here the expected loss for high risk (positive testing) individuals is ten times the expected loss for low risk (negative testing) individuals, $\lambda_1 = 1{,}000$, and $\lambda_2 = 10{,}000$)

market. Figure 4[13] represents the situation with but a single equilibrium in the market, and in which adverse selection leads to all lower risk individuals dropping out of the risk pool, and only high risk individuals remaining, that is the so-called 'lemons equilibrium'.[14] Figure 5 represents the situation in which multiple equilibria exist, one of which is the 'all high risk' or lemons risk pool, and the other two equilibria represent situations wherein both high and low risk individuals choose to remain in the risk pool.

[13] This figure may be viewed at http://www.livemath.com/lmstorage/files/1181781523669/equili1.html. The figure animates the changes in the expected present value of losses for changes in risk aversion. The risk aversion and other parameters may be changed to see the impact on the equilibria.

[14] This nomenclature comes from Akerlof's work in economics; see Akerlof (1970). Here the high or bad risks (the 'lemons') in the market drive out the low risks in equilibrium with asymmetric information.

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In Figure 4 the premium is represented along the horizontal axis, while the present value of the expected loss per contract and the premium are represented along the vertical axis. The intersection is the solution to equation (13). The same population, utility function, loss and financial parameters are assumed here. From the present value of the expected losses curve we note that, when the premium is low, both risk types are in the pool and the expected loss (average claims) is flat, while the premium increases. When the premium rises above the expected discounted loss for the low risk group, however, the low risk individuals start dropping out, causing the composition $\theta(p)$, and hence the average loss of the remaining risk pool, to increase, until, eventually, only the high risk individuals are left in the pool and the expected loss for the pool again stabilises. The one fixed point occurs where the marginal benefit equals the marginal cost, c.f. equation (15), and this represents a stable equilibrium, since the marginal cost is greater than the marginal benefit to the left of the equilibrium and less than the marginal benefit to the right. The equilibrium premium becomes the present value of the expected loss per exposure unit, i.e. \$9,523.81, an increase of 747% from the premium (\$1,123.81) which would exist in the market with no testing and no adverse selection effects, and a constriction in market coverage of 98%. This is the result when the background risk is not very large relative to the impact of the genetic risk; equivalently, it is the case in which the genetic risk swamps the background risk.

In Figure 5 the risk aversion coefficient for the consumer is still assumed to be $a = 0.2$, but the difference in the expected loss is reduced from a ten-fold increase to a three-fold increase. Thus, the low and high risks have expected losses of \$1,000 and \$3,000, respectively, that is that the background risk constitutes a larger part of the high risk group's total loss exposure. This altered assumption leads to the equilibria portrayed in Figure 5. As in Figure 4, the qualitative characteristics of the expected unit loss curve remain similar. However, now due to the closer expected loss costs for the high and low risk individuals, there are multiple intersections (equilibria). The extreme intersections represent equilibria which are stable, while the middle intersection represents an unstable equilibrium. Consider the first intersection; to the left of that equilibrium the expected loss per contract is greater than the expected revenue per contract, and so an increased premium is required to elicit more contracts. Similarly, to the right of that intersection the expected loss per contract is less than the revenue per contract, and so competition generates a decrease in the premium. Similarly, to the left of the middle equilibrium the marginal revenue exceeds the marginal contract cost, and so competition decreases the premium; to the right of the middle equilibrium, the marginal contract cost exceeds the marginal revenue, and so competition increases the premium. The two effects make the middle equilibrium unstable. Note also that the stable leftmost equilibrium (with a premium of \$1,003.65) has both low and high risk individuals in the market ($d_l(1{,}003.65) = 72{,}308$, and

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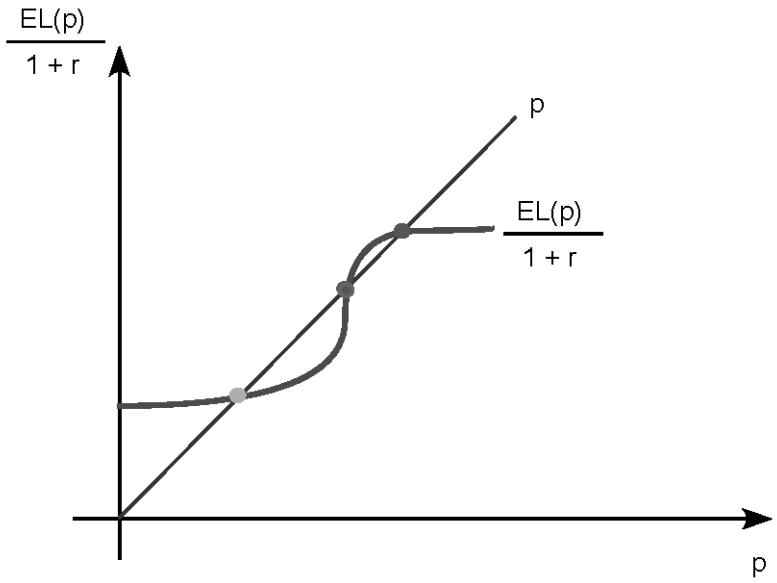

Figure 5. Multiple equilibria
(the 45° line denotes the premium and the expected loss curve denotes the present value of the expected loss per exposure unit. Here the expected loss for high risk (positive testing) individuals is 3 times the expected loss for low risk (negative testing) individuals $\lambda_1 = 1{,}000$, and $\lambda_2 = 3{,}000$)

$d_2(1{,}003.65) = 2{,}000$, $\theta(1{,}003.65) = 0.97308$ proportion low risk), and hence one can observe that, for situations in which the background risk (which is shared by those testing negative) constitutes a sizable proportion of the total risk faced by those testing positive, there is no market failure in the sense that both high and low risk individuals remain in the market. There is, however, an adverse selection effect, as prices increase from a premium of \$940.48, for the baseline situation in which all 100,000 individuals are untested, to \$1,003.65, when testing is allowed, information is not shared, and premiums are endogenously determined. This represents an increase of 1.3% due to adverse selection. Simultaneously, the number covered by insurance decreases to 74,308 people from the original market of 100,000 people, a decrease of 25.7% due to adverse selection effects (good risks dropping out of the market). The corresponding values for the two other equilibria are a premium of \$1,112.71, an increase of 12.3% and a decrease in market sales of 76.2% due to adverse selection, and finally, the lemons

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solution, with a premium of $2,857.14, representing an increased premium of 188%, and a decrease in market coverage (number insured) of 98%.[15]

6. Concluding Remarks

The issue of adverse selection is often brought up by insurers when addressing the potential negative economic consequences of proposed laws, banning or severely restricting their ability to use information related to genetic information, while allowing individual insured risks to have this same information.[16] Authors using actuarial pricing (expected losses) have examined this issue, and found that there can be private economic value to the individual insured risks of this information asymmetry, while the public value of this information suppression has been variable, depending upon the assumptions of the particular model, some finding public value to allow such asymmetries to exist, while others only find a negative value (Crocker & Snow, 1992; Doherty & Thistle, 1996; Hoy & Polborn, 2000). Of course public policy is often set using public value rather than private value, so this uncertainty does not give much guidance to the legislators or to public policy decision makers. Moreover, actual prices in the marketplace are set by the economic dynamics of supply and demand endogenously, so, while expected loss cost models may give some insights into the consequences of restricting genetic information distribution to insurers, the reality in a supply-demand driven market place may be different, a factor which should also be considered by policymakers in weighing the consequences of bans or restrictions.

The results in this paper may be distinguished from other results in the

[15] In this model the market is assumed to be competitive, and so the insurer responds to market prices by choosing how much it will write at the given price. If a monopolist insurer was assumed, then the insurer could set prices to maximise profit in (13) subject to market demands and resulting market composition at the chosen price level; the resulting price would be between the first two equilibria, $p = \$1{,}057.50$, a 6.8% increase in price and a decrease of 50.8% in market coverage due to adverse selection.

[16] Because insurers have not been forced to ignore valuable information in the past, there have been few (if any) actual example of market failure in insurance due to adverse selection caused by information asymmetries resulting in price increase spirals and ultimately market failure. In a regulated insurance market, however, the insurer may have limited ability to change premiums in response to adverse selection in a timely manner, and hence, in the long run, may voluntarily choose instead to withdraw from a market or severely reduce writing if information asymmetries are forced upon it. Anticipation of losses and the time lags needed to respond with price increases in a regulated market, along with the costs of doing so; together with the anticipated market effect of increased prices, can result in voluntary curtailment action by the insurer. Such was the situation in Washington D.C. when insurers were disallowed from testing for AIDS before issuing life insurance. While this could arguably be posed as an example of adverse selection causing market failure, in fact, the failure was due to insurers' voluntary withdrawal of insurance because of *anticipated* adverse selection rather than the *effect* of adverse selection itself.

literature in two respects. First, the emphasis has been placed on generating demand and supply functions from first principles, so that the market equilibrium could be demonstrated; this process has made it clear that supply is not independent of demand when the adverse selection problem exists, and it has revealed the existence of more than one equilibrium. The existence of more than one equilibrium is important, because it introduces a different measure of the adverse selection cost for each equilibrium. Second, the impact of adverse selection is determined in much of the literature by supposing that the individual fully insures if the premium is less that the cash equivalent of the risk or, equivalently, the expected loss plus the Pratt risk premium (see Pratt, 1964). This analysis allows the individual to purchase any amount of insurance between full and none, and that decision is expressed in the individual demand functions by risk type. The demands are then aggregated across individuals and risk types, and this aggregation allows us to determine the composition of the pool of insured risks. Finally, the adverse selection costs are determined by comparing the fair premium with each equilibrium premium; of course the equilibrium premium is determined by that quantity of insurance at which demand equals supply. Additionally, while Markov models can be viewed as 'long term', due to the limiting stationary distribution for the Markov model, it can take time for this limiting distribution to be achieved. Similarly, economic equilibrium pricing is a notion which is assumed to occur over time (it takes time for equilibrium to be achieved) so it can also be considered as a limiting case, given the information scenario presented to the consumer.

This paper takes an endogenous economic approach, which considers prices set by the market according to supply and demand (rather than just expected value pricing). Such prices respond dynamically to changes in the composition of the risk pool, with increased pricing resulting in decreased demand by lower risk insured individuals. This leads to an insurance buying risk pool with a higher percentage of high risk insured, resulting in a higher expected loss cost to the insurer. This, in turn, leads to a higher price for the product (and reduced supply at a given price), resulting in a responding demand change for the product. We show, however, that if there is sufficient background health care risk needed to be covered by both high and low risk individuals (e.g., other illnesses, accidents, flu, operations, cancers, heart diseases, etc.), and that if the genetic mutation under examination is sufficiently rare that the ratio of the expected total loss costs for the high and low risk groups from all causes is not too large (regardless of the ratio of costs for the particular cause governed by the mutation), then the market will not fail, an equilibrium price will be endogenously set with both high risk and low risk individuals participating in the market, and the insurance company will continue to be profitable. By examining the ultimate composition of the insured pool (the proportion left in the equilibrium priced pool who are high risk and buy, and those who are low risk and buy, and those in both groups

who do not buy) we can derive Markov transition probabilities for states in Markov models endogenously rather than exogenously, c.f. Subramanian *et al.* (1999) p541. Thus, this model can serve as a starting point for a generalisation of a Markov model wherein the transition probabilities are a function involving utility-based economic decision making of the consumers and are endogenously determined in an equilibrium setting.

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