Managing Genetic Tests, Surveillance, and Preventive Medicine Under a Public Health Insurance System

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Abstract

There is a prospect in the medium to long term future of substantial advancements in the understanding of the relationship between disease and genetics. We consider implications of an increase in information from genetic tests on predisposition to diseases from the perspective of managing health care provision under a public health insurance scheme. In particular, we consider how such information may potentially improve the targeting of medical surveillance to improve the chances of early detection of disease onset or preventive measures to reduce probability of onset. We show that social welfare can be enhanced only if careful attention is paid to coordinate changes in individual use of surveillance measures with genetic test results. The effects on the overall costs of health care are complex and, contrary to some expectations, improved targeting from the use of genetic information will not necessarily reduce overall healthcare costs.

Keywords: genetic tests; medical surveillance; public health insurance

JEL Codes: D8, I12, I18
1 Introduction

By all accounts in the scientific literature the potential benefits of the so-called Human Genome Project in providing enhanced prevention and treatment of disease are nothing short of revolutionary. A rough road map of the human genome has been available since 2003, having been undertaken in 1990. According to the NIH-sponsored web site genetests.org, there are currently over 1600 genetic tests used clinically.

Genomic science is now in something of a second phase of the Genomic Revolution in that current research involves not just the identification of so-called “disease genes” or, more appropriately, “disease alleles”, but also the understanding of how specific sequences of genes interact with each other as well as with environmental factors to affect onset and influence treatment of disease. According to the Nuffield Trust Genetics Scenario Project (2000), “The impact of the new genetics on existing health services in the United Kingdom has been compared to a tidal wave, a tsunami, sweeping all before it as it bursts upon the shore. A hyperbole perhaps; nevertheless the medicine that has been practised up to now, and the health service we have become familiar with, will undoubtedly be subject to enormous changes.” The project looks likely to lead to the real promise of vastly improved health care through genetic therapies, adoption of individualized drug treatments (pharmacogenomics) and generally improved understanding of disease. However, development of these advances have only just begun and will take substantial time to meet such bold expectations.

For the immediate and medium term future, information about differential predispositions towards many diseases such as breast and colon cancer can lead to improved use of existing medical technologies involving surveillance (e.g., mammograms and colonoscopies) as well as preventive measures (e.g., various prophylactic measures such as mastectomies or use of tamoxifen and other chemopreventive drugs). Some genetic tests continue to be quite expensive and so which tests to make available through health insurance plans, be they private or public, represents a challenge. Insurance or health care providers are concerned about the possibility of escalating costs due to the introduction of costly genetic tests that may also lead to increased treatment and preventive costs (e.g., see report by Miller, et al. (2002) funded by the Ontario Ministry of Health and Long Term Care) while others believe improved targeting of surveillance and preventive measures will substantially reduce health care costs. It is this aspect or phase of growth in genetic tests

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1 See Anderson, et al. (2006) for a cost-effectiveness comparison of various preventive strategies for women with BRCA1/2 mutations.

2 Caulfield, et al. (2008) summarize (and critique) these claims for the genetic field of nutrigenomics, noting (p. 47) that “The main themes in scientific journals were that nutrigenomics would lead to: improved dietary advice; the development of health-promoting supplements; preventive health strategies;
and related knowledge that we address here. In particular, we study the implications of improved genetic information about risk of disease in terms of the socially optimal management of surveillance and related health care strategies for public health insurance systems. The results of this exercise can be used to help determine guidelines to use in determining which genetic tests to offer. Some aspects of what we find could also be applied to a population covered (or partly covered) by private health insurance, although there are some important differences to consider.3

Many of the papers that model the effects of improved information about risk classification involve the private insurance market and exogenously specified (fixed) probabilities of loss/disease (e.g., Rothschild and Stiglitz, 1976; Wilson, 1977; Hoy, 1982, 1984; Crocker and Snow, 1985, 1986; Tabarrock, 1994; Hoel and Iversen, 2002; and Rees and Apps, 2006).4 Although our main model involves exogenously determined and differential probability of onset of disease, we allow for the possibility via surveillance of early or late detection of disease. For many diseases, early detection leads to improved treatment and outcomes. Information from genetic tests creates (or increases) differential assessment of risk of disease onset across individuals. Thus, although probability of onset may be fixed by genotype, choice of level of surveillance creates endogenous determination of detection being late or early (i.e., at least probabilistically). The benefit of a genetic test then arises from potential improvements in targeting of surveillance strategies for early detection of onset of disease. The important management issue is in determining the extent to which higher (lower) risks should increase (decrease) surveillance and then trying to encourage the appropriate responses from individuals. We show that a model of differential use of preventative medicine based on genotype is very similar and so determination of the value of genetic tests follows a similar pattern relating to improved targeting of such strategies.

It is well known that in the presence of health insurance, be it public or private, individuals face incentives that lead to actions that are not necessarily socially optimal; that is, due to standard moral hazard concerns individuals in our model may over-use or under-use medical surveillance or prevention.5 This implication of insurance will be exacerbated - or at least complicated - by the introduction of information about differential risk of disease and the reduction of health care costs. This last theme is particularly interesting because it is a common justification for supporting new technologies.”

3See Filipova and Hoy (2009a) for a description of the various potential uses to which this information can be applied, including pre-natal screening, genetic therapies, etc. Filipova and Hoy (2009b) investigate issues regarding the use and value of genetic testing that are specific to private health care systems.

4See Hoy (1989), Doherty and Posey (1998), and Hoel and Iversion (2002) for examples of models where self-protection (or prevention) can affect the probability of loss/disease differentially according to risk (geno-) type.

5Barros, Machado and Sanz-de-Galdeano (2008), for example, provide evidence that use of surveillance (blood and urine tests in their case) is higher for those covered by ‘extra’ health insurance.
onset. Under the assumption that individuals do not pay directly for surveillance or the medical cost of treating disease, we find under a broad range of scenarios that one group will tend to want to over-use surveillance while another group will tend towards under-use relative to socially optimal decisions. Perhaps counter-intuitively we find that it can be either those discovered to be high risk or low risk that end up under-utilizing surveillance or prevention with the other group over-utilizing. It may be reasonably straightforward on the one hand to ration publicly provided surveillance measures to correct for those who have an incentive to over-use, but less easy to encourage increased usage for under-users. Even the case of over-use may be difficult to control as we discuss in the paper.

The key feature of our model is that while genotype of a person is exogenously determined through inheritance, we assume either that the probability of early detection is determined endogenously by varying levels of surveillance or that the actual probability of disease is determined endogenously and differentially by genotype through varying levels of self-protection. Recognizing that the marginal effectiveness of either costly surveillance or prevention differs by genotype leads to a potential benefit from improved targeting of these medical interventions made possible by results of a relevant genetic test. We assume that the insurance system is "purely public" with all individuals assessed the same price for insurance. Further, we assume zero coinsurance payments for any medical costs whether these are associated with surveillance, prevention, medical treatment arising from onset of disease, or the cost of the genetic test. Of course, public health insurance systems can deviate from such policies and often do by charging patients for certain medical procedures including genetic tests. For example, the Ontario Hospital Insurance Plan does not cover the cost of the PSA test, a surveillance test for prostate cancer, but medical professionals do sometimes suggest its use. Introducing such instruments to a health insurance plan, be it public or private, can create incentive effects which can curtail the negative welfare implications arising from various moral hazard considerations. Although modeling such implications are beyond the scope of this paper, we do discuss such possibilities. Moreover, of course, public health insurance plans also generally cover only a subset of available genetic tests to be covered and one of the main points of our paper is to provide a model that is a useful input in determining just which tests to offer. Also, it is worth pointing out that private insurance plans tend to be more flexible with partial copayments for various medical interventions. However, under private insurance there is the worry about potentially negative implications of premium risk arising from genetic testing.6

In developing our model, we focus on the case of utilization of surveillance. In an initial

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6In many countries private health (and/or life) insurers are restricted from using genetic test results (or more broad sets of characteristics) in setting prices. Wynand, et al. (2007) have shown empirically that even with risk adjustments designed to provide incentives for private insurers to accept community rating, selection strategies for a set of European countries were nonetheless evident.
state of information no one holds any genetic test information and individuals are assumed homogeneous in all regards. Given the moral hazard implications of insurance, individuals may engage in either over- or under-use of surveillance, depending on whether their individually optimal choices are at a level such that, at the margin, increased surveillance leads to an increase or decrease in the financial cost of health care provision, respectively, where financial costs are absorbed by the insurance pool. We characterize how genetic testing can lead to changes in the pattern of over- and under-use of surveillance. As noted above, over-use is a problem that in principle can be resolved by rationing of the service but under-use is perhaps more problematic. It follows that such behavioral implications should be considered when determining the value of introducing a new genetic test to a health care system. Despite the potential of improved targeting of disease, these behavioral complications can lead to a negative welfare effect from the introduction of a costless genetic test. The implications and results for a model that highlights the use of prevention of disease are essentially analogous.

2 Model of Medical Surveillance with Genetic Testing

In this section we develop a simple model of medical surveillance and show how the privately optimal demand for surveillance (a) depends on the probability of onset of disease and (b) differs from the socially optimal level of surveillance due to the fact that individuals do not internalize the financial costs of any aspect of medical care, including surveillance and treatment of disease. We assume individuals are homogeneous in terms of their net income and basic preferences across all states of nature. Gross income of each individual is $y$ and each individual contributes an equal amount $\overline{TC}$ to account for the average or per capita cost of the health care system. To highlight the fact that there are no user payments required by the public health insurance plan and so individuals do not internalize the cost of their decisions about health care use, we use $\overline{TC}$ to denote this assessment when generating the individually optimal use of health care resources. Thus, net income for each individual is $y - \overline{TC}$.

We denote the perceived probability of disease by $\rho$ and, ex post, this probability depends on the results of a genetic test. Once an individual is afflicted by a disease, this can be detected either early or late. The probability of early detection, $p^{ED}(s)$ is a function of the level of medical surveillance $s \in [s, \overline{s}]$. Increasing surveillance will increase

\footnote{Of course in a more realistic model there would be differences between individual risks of disease and hence different optimal usage of surveillance based on risk differences revealed by characteristics other than genetic test results, such as family history or other available diagnostic tests. Our analysis demonstrates additional effects of risk differentiation arising from genetic test results. For an application along these lines in the context of private life insurance, see Hoy and Witt (2007).}
the probability that the disease is detected early rather than late, albeit at a decreasing rate \((p^{ED'}(s) > 0, p^{ED''}(s) < 0)\). Individuals’ welfare is made up of several components: 

\( u(\cdot) \) in the healthy state (i.e., conditional on no disease onset), 

\( w^{ED}(\cdot) \) in the disease state if disease is detected early, and 

\( w^{LD}(\cdot) \) in the disease state if disease is detected late.

Each utility index is assumed to be increasing and concave in net income \((u'(\cdot), w^{ED'}(\cdot), w^{LD'}(\cdot) > 0, u''(\cdot), w^{ED''}(\cdot), w^{LD''}(\cdot) \leq 0)\). We address the case of risk-neutrality not because we think it is a realistic assumption for a substantial fraction of the population but rather because it allows us to isolate the effect of risk aversion on our results. Further, we assume that an individual is better off in the disease state if the disease is detected early rather than late, \( w^{ED}(\cdot) > w^{LD}(\cdot) \); however, we allow for any ordering of first derivatives among the three utility indices.\(^8\) The particular ordering does not affect the qualitative nature of our results. Finally, there is a component that reflects a physiological (non-financial) cost of surveillance \( \Phi(s) \), which is assumed increasing and convex in the level of surveillance \((\Phi'(s) > 0, \Phi''(s) > 0)\). This gives us the expected utility function:

\[
EU(s) = (1 - \rho)u(y - TC) + \rho[p^{ED}(s)w^{ED}(y - TC) + (1 - p^{ED}(s))w^{LD}(y - TC)] - \Phi(s) \tag{1}
\]

A few remarks about the expected utility function generated from these assumptions, and its components, are in order. First, one can think of the utility indices for early and late detection of the disease as continuation utilities from a more elaborate model of choice that includes intertemporal expected utility defined over a lifetime. The presumption behind the assumption \( w^{ED}(\cdot) > w^{LD}(\cdot) \) is that individuals with a disease who are detected early will receive a treatment (or other medical attention) that is less invasive medically and so generates less disutility from treatment (e.g., a lumpectomy versus a mastectomy). Alternatively, early treatment leads to a better chance of cure and hence a higher expected value of future utility than if disease is detected late. Our results for the most part are independent of the relative ordering of marginal utilities in the healthy state compared to marginal utilities in either the early or late detection scenario of the disease state. Therefore, for the sake of simplicity we sometimes make the stricter assumption that \( w^{ED}(\cdot) = u(\cdot) - \kappa_E \) and \( w^{LD}(\cdot) = u(\cdot) - \kappa_L \), where \( \kappa_L \) and \( \kappa_E \) reflect a health-state dependent physiological (non-financial) cost, which is subtracted from utility in the case of disease, and which is larger in the case that the disease is detected late rather than early.

\(^8\)Most associated empirical studies suggest, roughly speaking, that marginal utility of income is lower in disease or illness states; e.g., see Viscusi and Evans (1990), Sloan, et al. (1998), and Finkelstein, Luttmer and Notowidigdo (2008). Tengstam (2007), however, finds the opposing result for the disability of having both legs paralyzed. The relationship probably depends on the particular disease or illness and so we don’t assume any particular ordering.

\(^9\)The physiological cost of surveillance may include discomfort/pain, a psychological component, time taken to have the procedure, and possible side effects (e.g., a certain fraction of colonoscopies result in damage - knicking - to the colon).
\((\kappa_L > \kappa_E)\). With this specification\(^{10}\), the marginal utility of income is independent of the health state or early/late detection distinction. Thus, for this more specific assumption on utility indices we get

\[
EU(s) = u(y - \overline{TC}) - \rho[p^{ED}(s)\kappa_E + (1 - p^{ED}(s))\kappa_L] - \Phi(s)
\] (2)

Using this latter version of the utility function we see that the marginal utility of income is simply \(u'(y - \overline{TC})\). When generating the socially optimal allocation of surveillance the implications on the overall cost of providing health care will of course be relevant. Thus, the effective marginal cost of an additional unit of health care is dependent on the level of health care costs according to the factor \(u'(y - \overline{TC})\). In the case of the less restrictive version of the expected utility function, equation (1), it is a more complicated expression and so the following transformation is useful. Define \(\Omega(y - \overline{TC})\) to be the utility of income function:

\[
\Omega(y - \overline{TC}) = (1 - \rho)u(y - \overline{TC}) + \rho[p^{ED}(s)w^{ED}(y - \overline{TC}) + (1 - p^{ED}(s))w^{LD}(y - \overline{TC})]
\] (3)

The result is that \(u(\cdot)\) and \(\Omega(\cdot)\) have similar properties; \(u'(\cdot) > 0\) and \(u''(\cdot)\), \(\Omega''(\cdot) \leq 0\). Thus, as we will see later, when using the more general case of the expected utility function we replace the term \(u'(\cdot)\) with \(\Omega'(\cdot)\) and \(u''(\cdot)\) with \(\Omega''(\cdot)\) in first and second order conditions. This explains why for the most part our results are independent of the ranking of the marginal utilities of income across the different health states. Therefore, for the main body of the text we adopt the more restrictive form of the utility function and simply note the differences generated at appropriate points. We investigate more fully the implications of this assumption in the Appendix.

We now turn our attention to the financial cost of providing health care. In the case that an individual incurs the disease, we assume that the assigned treatment will generate financial costs which are lower when the disease is detected early rather than late \((C^{DL} > C^{DE})\).\(^{11}\) This accounts for the fact that usually the severity of a disease grows when it is detected late, which in turn limits the choice of alternative treatment measures to those which are more aggressive and most expensive. There are also direct financial costs of surveillance \(C(s)\), which are assumed increasing and convex in the level of surveillance \(C'(s) > 0, C''(s) > 0\).\(^{12}\) We think of increased surveillance mostly as further, more invasive

\(^{10}\) This separation of utility into an income component and a health component is similar to Kifman (2001). Strohmenger and Wambach (2000) also use a state contingent utility function in an adverse selection model.

\(^{11}\) This assumption is not likely true for all diseases. Early detection of HIV that inevitably leads to full blown AIDS is probably more costly to treat since ‘end costs’ are the same while drug treatment through the pre-AIDS period is an additional cost. The qualitative nature of our results does not depend on this assumption.

\(^{12}\) Generally we may allow \(C''(s) = 0\) and still satisfy conditions for an interior optimum. Linearity of \(C(s)\) may reflect more frequent (repeated) applications of a given monitoring technology.
and expensive technologies being applied, rather than simply repeated occurrences of a given technology. Overall, the per capita expected cost of providing health care will depend on the level of surveillance through its direct effect $C(s)$ as well as its indirect effect through the probability of early versus late detection (i.e.; on $p^{ED}(s)$). Thus, the per capita health care costs are given by

$$TC(s) = \rho[\rho^{ED}(s)C^{DE} + (1 - \rho^{ED}(s))C^{DL}] + C(s)$$

(4)

Again, as for the utilities used in the disease state, one can think of the financial costs $C^{DE}$ and $C^{DL}$ as the expected value of the continuation costs of treatment over an individual’s remaining lifetime conditional on incurring the disease and having detection occur early or late, respectively.

We begin our analysis of health care choice with the presumption that it is the individual who (freely) chooses his level of surveillance. Due to the public insurance system with no user fees, individuals do not take into account their use of surveillance $s$ on the financial cost of the health care system (i.e., $\frac{\partial TC}{\partial s} = 0$). Homogeneity of preferences implies that each individual’s optimal choice of surveillance, $\hat{s}$, will be the same and so each individual’s contribution to health care cost, $\overline{TC}$ will equal $TC(\hat{s})$ - the equilibrium per capita health care cost. Thus, we use the same symbol $\hat{s}$ to denote both the individual’s privately optimal level of surveillance and the equilibrium level.

In order to compare the situation before a genetic test (GT) is conducted with the situation after a genetic test, we denote the initially perceived probability of disease as $\rho^0$, which also is assumed equal to the true population average probability of disease. A genetic test classifies individuals (possibly imperfectly) into two risk groups: those who test positive (negative) have, on average, a probability of disease $\rho^H (\rho^L)$, where $\rho^L < \rho^0 < \rho^H$. The proportion of individuals who test negative is denoted by $\eta_L$ while the proportion who test positive is $\eta_H = (1 - \eta_L)$. Thus, before a GT, every individual perceives the probability of disease to be $\rho^0$, while after a GT the fraction $\eta_L (\eta_H)$ perceive the probability of disease to be $\rho^L (\rho^H)$. For simplicity we will presume that the fractions testing positive and negative are fixed and that a more precise test is associated with lower rates of both false positives and negatives in a symmetric (or rather fixed) fashion as noted below. Since the population average probability of disease $\rho^0$ is constant, we have

$$\rho^0 = \eta_L \cdot (\rho^0 - \frac{\varepsilon}{\eta_L}) + \eta_H \cdot (\rho^0 + \frac{\varepsilon}{\eta_H}),$$

(5)

with $\varepsilon > 0$ describing the ‘degree of accuracy’ of the information. In developing the intuition about the value of GTs, it turns out to be convenient to consider the effect of a marginal increase of information, i.e., an increase of $\varepsilon$. Thus, a more precise test implies
the probabilities of disease for tested negatives and tested positives approach the true probabilities of the disease of low and high risk types (denote them by $\rho_L$ and $\rho_H$), which need not even be known. Note that $\rho^0 = \eta_L\rho_L^L + \eta_H\rho_H^H$ holds irrespective of the precision of the test.

### 2.1 Privately optimal demand for surveillance

In what follows we present the results for the special case of the more restrictive utility function of equation (2). However, we indicate what would differ if one uses the more general utility function of equation (1).

Individuals choose a level of surveillance to maximize their expected utility. As noted earlier, from a private perspective $\frac{\partial EY}{\partial \rho} = 0$. Denote by $\hat{s}$ the privately optimal choice of surveillance for a given set of parameters, which is the solution to the first order condition

$$FOC: \rho \cdot p^{ED}(\hat{s}) \cdot [\kappa_L - \kappa_E] - \Phi'(\hat{s}) = 0$$

(6)

which equates the marginal personal (non-financial) benefit of surveillance, i.e. the utility gain resulting from early detection, $\kappa_L - \kappa_E$, multiplied by the probability of disease, $\rho$, as well as by the marginal increase in the probability of early detection, $p^{ED}(\hat{s})$, to the marginal physiological cost of surveillance, $\Phi'(\hat{s})$.

For the second order condition $(SOC)$ one gets that

$$\rho \cdot p^{ED''}(\hat{s}) \cdot [\kappa_L - \kappa_E] - \Phi''(\hat{s}) < 0, \forall s.$$  

(7)

The optimal level of surveillance depends on the probability of disease $\rho$. Specifically, there will be a critical level for the probability of disease, below which individuals will choose the smallest possible level of surveillance ($\hat{s} = \underline{s}$), which of course could be zero. Denote by $\underline{\rho}$ this critical level for the probability of disease; i.e., for which $\underline{\rho} \cdot p^{ED}(\hat{s}(\underline{\rho})) \cdot [\kappa_L - \kappa_E] - \Phi'(\hat{s}(\underline{\rho})) = 0$ holds, where $\hat{s}(\underline{\rho}) = \underline{s}$. Thus, for any $\rho \leq \underline{\rho}$ the individual will choose $\hat{s} = \underline{s}$. This critical level for the probability is $\rho = 0$, if $\Phi'(\underline{s}) = 0$. A similar treatment may apply for some maximal probability $\overline{s}$ and associated $\overline{\rho}$. In what follows we focus only on the range of probabilities for which $\underline{s} < \hat{s} < \overline{s}$ (i.e., we ignore corner solutions).

Applying the implicit function theorem to the FOC yields

$$\frac{d\hat{s}}{d\rho} = -\frac{p^{ED'}(s)[\kappa_L - \kappa_E]}{\rho \cdot p^{ED''}(s) \cdot [\kappa_L - \kappa_E] - \Phi''(s)} > 0$$

(8)

and

$$\frac{d^2\hat{s}}{d\rho^2} = -\frac{p^{ED''}(s) \cdot [\kappa_L - \kappa_E] - \Phi''(s) \frac{d\kappa_L}{d\rho}}{\rho \cdot p^{ED''}(s) \cdot [\kappa_L - \kappa_E] - \Phi''(s)^2}$$

(9)
A sufficient condition for $\frac{d^2\hat{s}}{dp^2} < 0$ (concavity of the $\hat{s}(\rho)$ schedule) is that $\Phi'''(s) \geq 0$ and $p^{EDm}(s) \leq 0$, the case depicted in Figure 1.

The implication of a genetic test on the demand for surveillance is clear. Tested positives, for which the probability of disease will be larger than before the test, will demand more surveillance and tested negatives, for which the reverse holds, will demand less surveillance. It can also be seen that the demand for surveillance is larger the higher is the marginal productivity of surveillance given the disease, i.e. the more sensitive is the probability for early detection to the level of surveillance, and the larger are the savings of the physiological costs of disease when it is detected early, i.e., $[\kappa_L - \kappa_E]$. For the more general utility function given in equation (1) one need simply replace $[\kappa_L - \kappa_E]$ by the term $[w^{ED}(y - TC) - w^{LD}(y - TC)]$ in all of the expressions above. Since both $[\kappa_L - \kappa_E]$ and $[w^{ED}(y - TC) - w^{LD}(y - TC)]$ are positive, the sign conditions are not affected. Note also that the demand for surveillance is smaller the faster the personal cost of surveillance increases (the larger is $\Phi'(\hat{s})$).

It is worth demonstrating some properties of the more restrictive function $EU(s)$ in relation to the optimal choice of $s$, ($\hat{s}$), as well as the effect of a change in the probability of disease. Due to the separability of income and health effects from surveillance, altering
the cost of insurance simply creates a family of vertically parallel $EU(s)$-curves, with higher curves of course relating to lower payment for health care costs (insurance). This is illustrated in Figure 2 for a pair of values of health care costs (insurance premiums), with $\overline{TC}_1 < \overline{TC}_2$. For the more general utility function, if $[w^{ED}(y - TC) - w^{LD}(y - TC)]$ is falling in income (i.e., marginal utility of income is higher in the disease state under late detection than under early detection), then the curve for $EU(s)$ given $\overline{TC}_1$ would lie above that given $\overline{TC}_2$ but not vertically and would achieve its maximum at a smaller value of $s$ and vice versa if the marginal utility of income is lower in the case of late detection than early detection. An increase in $\rho$ obviously reduces the level of expected utility conditional on any given level of $s$ and we have shown that the privately optimal level of $s$ will increase with an increase in $\rho$ for either specification of the utility function. Thus, we have the configuration for $\rho_1 < \rho_2$, but $\overline{TC}$ constant, of the two curves $EU(s; \rho_1)$ and $EU(s; \rho_2)$ shown in Figure 3.

It is useful also to consider how the value of $s$ that minimizes the per capita financial cost of providing health care, which we will refer to as $\tilde{s}$, changes as $\rho$ changes. A similar comparative statics exercise as for $\tilde{s}$ leads to the conclusion that an increase in probability of disease, $\rho$, leads to an increase in $\tilde{s}$, as illustrated in Figure 4. Thus, since individuals
Figure 3: Shift of expected utility due to increase of probability of disease
Figure 4: Shift of per capita cost of health care due to increase of probability of disease

do not internalize the financial implications of their choice of surveillance ($\hat{s}$), it follows that if $\hat{s}$ is less than $\tilde{s}$, as illustrated in Figure 5, a marginal increase in $s$ beyond $\hat{s}$ by all individuals in the insurance pool will, due to the envelope theorem, have no effect on each individual’s expected utility ($EU$) based on the net effects of physiological health benefits and costs, which is all the consumer takes into account. An increase in $s$ would, however, reduce per capita cost of health insurance for all in the pool, hence increasing everyone’s welfare. This is a straightforward moral hazard problem. Thus, in this case the welfare maximizing choice of surveillance, $s^*$, will be greater than the privately optimal choice. The contrary implication would be the case if $\tilde{s}$ were less than $\hat{s}$. The implications of these patterns are developed more fully later in the paper.

Individuals will change their demand for surveillance after a genetic test in a manner that reflects their improved information about their risk of disease. This suggests some potential ‘production efficiency’ through better targeting of surveillance. Formally, the decision whether to accept a genetic test is based on whether the ex ante expected utility, i.e., before knowing whether one will be tested positive ($\rho = \rho^H$) or negative ($\rho = \rho^L$), exceeds the expected utility without a genetic test, where the average probability of disease is $\rho^0$. Furthermore, since individuals do not internalize the financial costs of their actions,
Figure 5: Socially optimal level of surveillance
the availability of a genetic test and the resulting changes in individuals behaviors, once
they become better informed about their risk types, creates externalities to the insurance
pool. We deal with these issues in the following subsections.

2.2 Individual behavioral effects of genetic tests

To analyze the change in expected utility (welfare) associated with a genetic test from an
individual’s perspective denote the maximized value of expected utility (the individual’s
value function) by \( v = EU(\hat{s}) \), which we can write as a function of the probability of
disease, i.e., \( v(\rho) = EU(\hat{s}(\rho)) \). This value function takes into account any changes in
financial costs of providing health care associated with the implications of how surveillance
decisions adapt to changes in the probability of disease (i.e., through the function \( \hat{s}(\rho) \)).

Without a genetic test individuals have expected utility value of \( v(\rho^0) = EU(\hat{s}(\rho^0)) \).
After a genetic test individuals adjust their optimal surveillance decisions in line with
the outcome of the test and so those who test negative end up with expected utility
\( v(\rho_L) = EU(\hat{s}(\rho_L)) \) while those who test positive end up with expected utility
\( v(\rho_H) = EU(\hat{s}(\rho_H)) \). From an ex ante perspective (i.e., before results of a genetic test are known),
the expected utility (in equilibrium) from taking a genetic test with information value \( \varepsilon \)
is \( EU^A(\varepsilon) = \eta_L v(\rho_L) + \eta_H v(\rho_H) \). Since \( \rho^0 = \eta_L \rho_L + \eta_H \rho_H \), it follows that a genetic test
represents a mean preserving spread in the probabilities of disease onset. Thus, global
convexity of the value function \( v(\rho) \) implies that ex ante expected welfare from a genetic
test is larger than the initial expected welfare (utility), while the reverse is implied by
global concavity.

It is important to realize at this point that global concavity, which means a costless
genetic test to which everyone submits leads to a reduction in each individual’s well-being,
does not mean that such a test would be refused. In fact, as we explain below, individuals
in our model will always accept a costless genetic test and react accordingly (i.e., according
to \( \hat{s}(\rho) \)) even if, given that everyone in the insurance pool has the same incentive, the end
result is that everyone is worse off. The reason is that individuals don’t internalize the
financial cost implications of their own behavioral changes and nor can they control the
behavioral changes of others resulting from the improved information. In other words,
ignoring this financial cost effect, over which the individual has no control, the genetic
test always appears ex ante to have (private) value.

For the first derivative of the value function we have
\[
\frac{dv(\rho)}{d\rho} = \frac{\partial EU}{\partial \hat{s}} \frac{d\hat{s}}{d\rho} + \frac{\partial EU}{\partial \rho}
\]
(10)

First consider a marginal increase of information at the point of no information, i.e. at
\( \varepsilon = 0 \). Applying the envelope theorem we see that the first term equals zero. Although
the individual (correctly) does not perceive that his choice of \( s \) affects his per capita cost of health care insurance, the fact that everyone adjusts \( s \) accordingly means this cost is affected nonetheless. We write \( TC_e(\rho) = TC(\hat{s}(\rho), \rho) \) to reflect the equilibrium per capita cost of insurance given perceived probability of disease \( \rho \). Thus, the second term in the above equation is the partial derivative of \( EU(s) \), taken from equation (2) but with \( TC_e(\rho) \) replacing \( \overline{TC} \), with respect to \( \rho \) and so, for the (perhaps expected) case of \( \frac{dTC_e}{d\rho} > 0 \) we have

\[
\frac{dv(\rho)}{d\rho} = u'(y - TC_e(\rho))(-\frac{dTC_e}{d\rho}) - [p^{ED}(\hat{s}(\rho))\kappa_E + (1 - p^{ED}(\hat{s}(\rho)))\kappa_L] < 0. \tag{11}
\]

The first term reflects the financial consequences of the probability of disease \( \rho \) changing while the second term reflects the perceived health consequences of learning that one faces an updated probability of disease onset. As noted above, although an individual doesn’t take into account the (negligible) effect of any change in his own choice of \( s \) on the per capita cost of providing health care arising in the insurance pool, the individual is affected in equilibrium by the fact that all those assigned to a different risk class with associated value of \( \rho \) do create financial implications regarding this insurance cost. This cost is determined by the privately optimal choices individuals make according to the schedule \( \hat{s}(\rho) \). It is important to note here that it is possible that an increase in \( \rho \), which induces an increase in \( s \), could lead to a decrease in \( TC_e \), hence leading to the perhaps counterintuitive result that \( \frac{dTC_e}{d\rho} < 0 \). This possibility is illustrated by Figure 6 below. Moreover, a similar result applies even if the choice of \( s \) being considered is that which maximizes social welfare since both \( EU(s) \) and \( TC(s) \) come into play. This possibility will become evident later in the paper.

Therefore, we have

\[
TC_e(\rho) = \rho[p^{ED}(\hat{s}(\rho))C^{DE} + (1 - p^{ED}(\hat{s}(\rho)))C^{DL}] + C(\hat{s}(\rho)) \tag{12}
\]

which implies

\[
\frac{dTC_e}{d\rho} = p^{ED}(\hat{s}(\rho))C^{DE} + (1 - p^{ED}(\hat{s}(\rho)))C^{DL} - \rho[p^{ED}(\hat{s}(\rho))[C^{DL} - C^{DE}]\frac{d\hat{s}}{d\rho} + \frac{dC(\hat{s}(\rho))}{d\hat{s}} \cdot \frac{d\hat{s}}{d\rho} \tag{13}
\]

Note that our use of \( \hat{s} \) to denote both the individual’s optimal choice of surveillance as well as the equilibrium level is something of an abuse of notation. To be explicit, consider the following more literal way of modeling this relationship. The total (financial) costs of medical care, per capita, is a function of each individual’s surveillance level \( s_i \), \( i = 1, 2, ..., n \). We would then have \( TC_e(s_1, \hat{s}_2, ..., \hat{s}_n; \rho) \) with each \( \hat{s}_i \) being a function of \( \rho \) and with the final argument in \( TC_e \) reflecting the direct effect of an increase in the
Figure 6: Change of equilibrium per capita cost of health care due to increase of the probability of disease
probability of disease, \( \rho \) (i.e., the effect if \( \rho \) were to change with no effect on any of the \( s_i \) choices. Thus,

\[
\frac{dTC_e}{d\rho} = \frac{\partial TC_e}{\partial \rho} \bigg|_{\hat{s}_j \text{ constant}} + \left\{ \frac{\partial TC_e}{\partial \hat{s}_i} \frac{d\hat{s}_i}{d\rho} + \sum_{j=1,(\neq i)}^{n} \frac{\partial TC_e}{\partial \hat{s}_j} \frac{d\hat{s}_j}{d\rho} \right\}
\]  

(14)

Individual \( i \) treats \( \frac{\partial TC_e}{\partial \hat{s}_i} \) as effectively zero, which it is, but the last (summation) term on the RHS reflects the total sum of the effect of all (others') choices regarding surveillance on cost of provision by the insurer. These are not under control by any individual but do of course affect \( TC_e \) and so can’t be treated as zero. Given that agents are homogeneous in their choices (\( \hat{s}_j = \hat{s}(\rho), \forall j = 1, ..., n \)), we adopt a short form \( \frac{\partial TC_e}{\partial \hat{s}} \frac{\partial \hat{s}}{\partial \rho} \) to represent this overall effect of individuals altering their surveillance level on the per capita cost of provision and sometimes write \( TC_e(\hat{s}(\rho), \rho) \) to reflect indirect and direct effects, respectively, of a change in \( \rho \) on the per capita cost of providing health care. Thus, the first line of equation (14) reflects the (direct effect of) added cost of treating patients due to the higher incidence of disease (i.e., higher \( \rho \) but holding \( \hat{s} \) fixed). The two terms in the second line reflect the impact on health care costs due to the effect of a change in \( \rho \) on the equilibrium level of surveillance, \( \hat{s} \). The first of these two terms is negative, representing a reduction in health care costs since better targeting means a shift from lower to higher risk types being engaged in surveillance which leads to more individuals avoiding the more costly late detection outcome. The second of these terms is positive, representing the (direct) cost of providing a higher level of surveillance. Thus, as noted above, overall the total derivative \( \frac{dTC_e}{d\rho} \) may be negative.\(^{13}\) Therefore, there is a perhaps counterintuitive possibility that after individuals take genetic tests, those determined to be low risk types could end up creating higher costs for the health insurance plan while the high risk types create lower costs. If \( TC_e(\rho) \) is strictly concave then a GT, which is a mean preserving spread (wrt the population) of the risk of disease onset, leads to a lower per capita financial cost of providing health care, and vice versa if it is strictly convex. Thus, the curvature of \( TC_e(\rho) \) also figures into the determination of whether a GT will enhance welfare. We now turn our attention to explicitly consider the welfare implications of a genetic test.

2.3 Social welfare implications of genetic tests

Suppose a genetic test is costless. Standard results from information theory lead one to expect that from an ex ante position individuals can never become worse off as a result of taking a genetic test. We know that a (fair) lottery over probabilities has a neutral effect on expected utility if an individual doesn’t change his behavior. This follows from the property that expected utility is linear in probabilities. However, if the individual would

\(^{13}\)In fact, without further restrictions, we cannot sign either \( \frac{\partial TC_e}{\partial \rho} \) or \( \frac{\partial^2 TC_e}{\partial \rho^2} \).
choose an alternative (optimal) decision for at least one outcome of the lottery, then the information has positive value. In our model individuals do adjust their privately optimal behavior conditional of either signal (i.e., whether the genetic test indicates they are low or high risk). This suggests that the signal has a positive private value and so the individual will accept the GT. However, in determining the social value of such a costless test, there is an external effect to be considered in our particular application. If all members of the insurance pool take a GT and adjust their surveillance activities accordingly, a single individual cannot avoid the implications on the average cost of health care provision by not taking the test himself. Hence, even though each change in behavior is privately optimal and would otherwise increase expected utility, the genetic test leads to adjustments in surveillance which could increase the average cost of provision and possibly even reduce welfare. This will indeed be possible if the equilibrium cost function is convex (i.e., if $TC''_\rho > 0$). Thus, it is possible that all individuals will voluntarily submit to a GT even though it ultimately makes everyone worse off.

To see more formally why individuals always voluntarily choose to have a GT even when it may lead to lower welfare for all consider the following argument. An individual correctly perceives his financial contribution to the cost of the health care system to be independent both of whether he has taken a test or, if he has, it is independent of his test result. Thus, the individual compares choosing $s$ without information about $\rho$ (so perceiving $\rho = \rho^0$) to choosing $s$ conditional on taking a test and discovering $\rho$ takes on a value of $\rho^H$ with probability $\eta_H$ or a value of $\rho^L$ with probability $\eta_L$ with $\rho^0 = \eta_H \rho^H + \eta_L \rho^L$. The individual’s decision is based only on that part of expected utility associated with physiological health effects, including the disutility cost of surveillance, and so the individuals’ effective objective function is

\[
EU^A = -\rho[(1 - \rho^{ED}(s)) \kappa_L + \rho^{ED}(s) \kappa_E] - \Phi(s) \tag{15}
\]

Artificially fixing the value of $s$ at its optimal value if $\rho = \rho^0$, say $s = \hat{s}_0$, taking a GT is equivalent to submitting to a mean preserving spread in the probability $\rho$. $EU^A$ is linear in $\rho$ and so conditional on not re-optimizing on $s$ conditional on revelation of $\rho^L$ or $\rho^H$, the individual views such a lottery as neutral. However, the individual can increase conditional expected utility in each case by re-optimizing on $s$ and so the individual, in ignoring any possible financial costs to the insurance pool, chooses to have the GT. Now

---


15 From equation (11) we can derive $v''(\rho)$ which includes the term $\rho^{ED}(\hat{s})|\kappa_L - \kappa_E|$ which is positive. However there are also terms involving derivatives of $TC_e(\rho)$, which cannot be signed. Hence, the function $v(\rho)$ isn’t necessarily convex.

16 One can also show that if we derive the private value function, $\hat{v}(\rho)$ for the individual based on equation (15) and disregarding the non-internalized effect of $\hat{s}$ on $TC_e$, this restricted value function
we show formally that the availability of the GT may lead to him, and everyone else, becoming worse off.

To determine a useful expression that isolates the various aspects of the effect of genetic test information on social welfare, we consider an initial value of $\varepsilon > 0$ as the increase in $\varepsilon$ affects expected utility and costs.\(^{17}\) This way we can address the effects of an increasingly informative signal rather than one that offers a little information starting from none. Moreover, the results can be applied to a scenario in which there is initially some information available about differential health risks in the population, say from family background information, where this is followed by an improved signal of health type that results from a genetic test.

Let $\overline{TC}_A$ be the average (financial) cost of providing health care services to the insurance pool under community rating. If individuals obtain GTs, then this becomes

$$\overline{TC} = \eta L \overline{TC}_L + \eta H \overline{TC}_H$$

where $\overline{TC}_L$ is the per capita cost of health care for those of type $L$ and $\overline{TC}_H$ is per capita cost of health care for those of type $H$. That is, all individuals pay the per capita cost of health care provision based on the weighted average of the cost of provision for each risk type. Recall that choice of surveillance level depends on perceived probability of disease ($\rho_L$ and $\rho_H$ for low and high risk types respectively). The value function then becomes

$$v(\rho) = EU_A$$

below. We need to account for equilibrium values for $\overline{TC}_L$ and $\overline{TC}_H$ which we will write as $\overline{TC}_L$ and $\overline{TC}_H$ and let $\overline{TC}_e$ represent the equilibrium average (or actual) cost assessed to each person. So, we have

$$EU_A = \eta L \{u(y - \overline{TC}_e) - \rho_L[(1 - p^{ED}(s^L))\kappa_L + p^{ED}(s^L)\kappa_E] - \Phi(s^L)\} + \eta H \{u(y - \overline{TC}_e) - \rho_H[(1 - p^{ED}(s^H))\kappa_L + p^{ED}(s^H)\kappa_E] - \Phi(s^H)\}$$  (16)

remembering that individuals treat $\overline{TC}_e$ as unaffected by their choice of surveillance, but of course cost does end up depending on the choices that individuals make (see relevant equation (13) for this effect). For convenience, we drop the $\hat{}$ notation on the variables $s^L$ and $s^H$, break the above expression into its component parts, $EU_A = \eta_L EU^L + \eta_H EU^H$, and separately write out $dEU_i/d\varepsilon$ for $i = L, H$.

$$dEU_A = \eta L dEU^L + \eta_H dEU^H$$  (17)

\(^{17}\)The above arguments apply to each type in the starting position of this exercise, and so each type would accept a better signal - i.e., an initial genetic test or a more informative genetic test.
with
\[
\frac{dEU^L}{d\varepsilon} = u'(y - TC_e^A) \cdot \left( \frac{dTC_e^A}{d\varepsilon} \right) - \frac{d\rho^L}{d\varepsilon} \left[ p^{ED}(s^L) \kappa_E + (1 - p^{ED}(s^L)) \kappa_L \right] - \rho^L \left[ \frac{dp^{ED}}{ds^L} \frac{ds^L}{d\rho^L} \cdot (\kappa_L - \kappa_E) \right] - \frac{d\Phi(s^L)}{ds^L} \frac{ds^L}{d\rho^L} \frac{d\rho^L}{d\varepsilon}
\] (18)

and
\[
\frac{dEU^H}{d\varepsilon} = u'(y - TC_e^A) \cdot \left( \frac{dTC_e^A}{d\varepsilon} \right) - \frac{d\rho^H}{d\varepsilon} \left[ p^{ED}(s^H) \kappa_E + (1 - p^{ED}(s^H)) \kappa_L \right] - \rho^H \left[ \frac{dp^{ED}}{ds^H} \frac{ds^H}{d\rho^H} \cdot (\kappa_L - \kappa_E) \right] - \frac{d\Phi(s^H)}{ds^H} \frac{ds^H}{d\rho^H} \frac{d\rho^H}{d\varepsilon}
\] (19)

Note the following (to be substituted into the above two equations):
\[
\frac{d\rho^L}{d\varepsilon} = -\frac{1}{\eta_L}, \quad \frac{d\rho^H}{d\varepsilon} = \frac{1}{\eta_H}
\] (20)

and
\[
\frac{dTC_e^A}{d\varepsilon} = \eta_L \cdot \frac{dTC_e^L}{d\rho^L} \frac{d\rho^L}{d\varepsilon} + \eta_H \cdot \frac{dTC_e^H}{d\rho^H} \frac{d\rho^H}{d\varepsilon}
\] (21)

Using the above, we can write:
\[
\frac{dEU^A}{d\varepsilon} = u'(y - TC_e^A) \cdot \left[ \frac{dTC_e^L}{d\rho^L} - \frac{dTC_e^H}{d\rho^H} \right]
+ \left[ p^{ED}(s^H) - p^{ED}(s^L) \right] \cdot (\kappa_L - \kappa_E)
- \rho^L \frac{dp^{ED}}{ds^L} \frac{ds^L}{d\rho^L} (\kappa_L - \kappa_E) + \frac{d\Phi(s^L)}{ds^L} \frac{ds^L}{d\rho^L}
+ \rho^H \frac{dp^{ED}}{ds^H} \frac{ds^H}{d\rho^H} (\kappa_L - \kappa_E) - \frac{d\Phi(s^H)}{ds^H} \frac{ds^H}{d\rho^H}
\] (22)

The first line in the above equation represents the financial implications of the genetic test through the effects of changes to the cost of health care provision to the two risk types through the equilibrium cost functions. That is, people assigned to different risk classes choose different levels of surveillance and face different probabilities of disease and so there are financial implications as measured by the term \( \left[ \frac{dTC_e^L}{d\rho^L} - \frac{dTC_e^H}{d\rho^H} \right] \). If the cost function is linear in \( \rho \) this term disappears since this would imply that the \( \text{average} \) per capita cost of providing health care is unaffected by a mean preserving spread in disease probabilities. If \( TC_e(\rho) \) is strictly convex, then \( \frac{dTC_e^L}{d\rho^L} < \frac{dTC_e^H}{d\rho^H} \) (due to \( \rho^L < \rho^H \)) and the expected cost of health care provision in the presence of information from genetic testing will rise and so the first term in equation (22) will be negative, and vice versa if \( TC_e(\rho) \) is strictly concave.

\(^{18}\text{Recall that it is possible even that } \frac{dTC_e^L}{d\rho^L} < 0 \text{. Let us assume here that it is positive, although it isn’t important to do so.}\)
The expected disutility from onset of disease is greater if it is detected late than early and high risk types adopt a higher level of surveillance (i.e., $s^H > s^L$ and so $p^ED(s^H) > p^ED(s^L)$). So line 2 reflects the efficiency gain of information from a genetic test in the use of surveillance that arises from better targeting (i.e., more people who are high risk rather than low risk now realize this). The efficiency effect is higher the greater the difference in the probabilities of early detection for the two types, $[p^ED(s^H) - p^ED(s^L)]$, and the greater the utility benefits of early detection, $[\kappa_L - \kappa_E]$. Lines 3 and 4 each represent the marginal effect on the decisions regarding $s$ of $L$-types and $H$-types, respectively (i.e., their privately optimal decisions). The envelope theorem applies and each of these terms is zero. To see that is the case, rearrange lines 3 and 4 as:

$$\frac{d\hat{s}^L}{dp^L} \cdot \left[ \rho^L \frac{dp^ED}{ds^L} (\kappa_L - \kappa_E) - \frac{d\Phi}{ds^L} \right]$$  \hspace{1cm} (23)

$$\frac{d\hat{s}^H}{dp^H} \cdot \left[ \rho^H \frac{dp^ED}{ds^H} (\kappa_L - \kappa_E) - \frac{d\Phi}{ds^H} \right]$$  \hspace{1cm} (24)

Thus, we have the following proposition.

**Proposition 1.** Under community rating individuals will always voluntarily submit to a (costless) genetic test. However, due to noninternalized cost implications, the resulting welfare implications may be positive or negative.

1. If $\frac{dTCA_s}{d\rho^L} \leq 0$ individuals’ expected welfare will (unambiguously) increase as a result of introducing GTs.

2. If $\frac{dTCA_s}{d\rho^L} > 0$ there is a negative effect on individuals’ expected welfare due to a resulting increase in the per capita cost of health care provision. Individuals will experience an increase (decrease) in expected welfare if

$$[p^ED(s^H) - p^ED(s^L)] \cdot [\kappa_L - \kappa_E] > (>) - u'(y - TCA_i) \cdot \left[ \frac{dTCL^L}{dp^L} - \frac{dTCH^H}{dp^H} \right]$$  \hspace{1cm} (25)

### 2.4 Socially optimal levels of surveillance under genetic testing

The socially optimal level of surveillance, which would be determined by a social planner, will generally deviate from the private choice - a standard moral hazard effect from insurance coverage which can hinder or even dominate the potential efficiency benefits of better information about risk of disease. To better understand these complications, we demonstrate the difference between the privately optimal choice of surveillance, $\hat{s}$, the choice of surveillance that would minimize the financial cost of health care provision, $\tilde{s}$, and the socially optimal choice of surveillance, $s^*$, conditional on a given perceived probability of disease, $\rho$. 

21
We have assumed that $p^{ED}(s)$ is concave while $\Phi(s)$ and $C(s)$ are convex, which together imply that $EU(s)$ is concave in $s$ while $TC(s)$ is convex in $s$. For simplicity, let us assume in all cases strict concavity/convexity as the case may be. Further, assume interior optima [i.e., $\hat{s}, \tilde{s} \in (\underline{s}, \overline{s})$ which will imply $s^* \in [\underline{s}, \overline{s}]$]. We can characterize three sets of comparisons based on how the privately optimal choice deviates from the socially optimal choice, $s^*$. We refer to these cases as (1) under-utilization ($\hat{s} < s^*$), (2) appropriate utilization ($\tilde{s} = s^*$), and (3) over-utilization ($\hat{s} > s^*$). As will become apparent below, it follows that in these three cases we also have (1) $\hat{s} < s^* < \tilde{s}$, (2) $\hat{s} = \tilde{s} = s^*$, and (3) $\hat{s} > s^* > \tilde{s}$, respectively.

First consider the case of under-utilization (Figure 7), which occurs if $\hat{s} < \tilde{s}$. At the privately optimal level ($s = \hat{s}$) the envelope theorem applies and so a small increase in $s$ has no first-order effect on utility. However, given $s < \tilde{s}$, it follows also that an increase (i.e., at least a small increase) in $s$ by all (homogeneous) individuals would reduce the cost of health care. Thus, at the social optimum, the marginal reduction in $EU$ from an increase in $s$ from changes to physiological health effects is just counter-balanced by the marginal reduction in the per capita financial cost of health care, as illustrated in Figure 7 where $u'()$converts units apporpirately.

![Figure 7: Under-utilization of surveillance](image-url)
The case of appropriate utilization ($\hat{s} = s^*$) occurs (see Figure 8) where the two curves $TC(s)$ and $EU(s)$ attain their minimum and maximum values, respectively, at the same value of $s$. This is not a generic result but is a useful benchmark case for demarcating possibilities as we see below. Finally, the case of over-utilization occurs if the value of $s$ at which $TC(s)$ is minimized is less than that at which $EU(s)$ is maximized (see Figure 9).

Earlier we showed that an increase in $\rho$ leads to an increase in $\hat{s}$. A similar (simple) comparative static analysis demonstrates that an increase in $\rho$ also leads to an increase in $\tilde{s}$. So an increase in the probability of disease shifts the $EU(s)$ curve downwards and its maximum point ‘to the right’, while the $TC(s)$ curve is shifted upwards and its minimum point is also shifted ‘to the right’. The effect on the socially optimal point $s^*$, however, is determined by how the slopes of the two curves change at a value(s) intermediate between $\hat{s}$ and $\tilde{s}$. It turns out that $s^*$ may either increase or decrease when $\rho$ increases, a relationship which we explore formally below. But first we describe the mathematical derivation of the marginal conditions characterizing the socially optimal level of surveillance. This involves a social planner choosing a level $s = s^*$ to maximize $EU(s)$ but, unlike individuals private decision making behavior, the planner takes into account the effect of surveillance on total
Figure 9: Over-utilization of surveillance
health care costs, \( \frac{\partial TC}{\partial s} \). The first order condition, which we denote by \( FOC^* \) is

\[
u'(y-TC(s^*)) \cdot [\rho \cdot p^{ED}(s^*)(C^{DL}-C^{DE})-C'(s^*)] + \rho \cdot p^{ED}(s^*) \cdot (\kappa_L-\kappa_E) - \Phi'(s^*) = 0 \tag{26}
\]

For the second order condition, \( SOC^* \), we get

\[
u''(y - TC(s^*)) \left( -\left. \frac{\partial TC}{\partial s} \right|_{s^*} \right)^2 + u'(y - TC(s^*)) \left[ \rho \cdot p^{ED''}(s^*) (C^{DL}-C^{DE}) - C''(s^*) \right] + \rho \cdot p^{ED''(s^*)} \cdot (\kappa_L-\kappa_E) - \Phi''(s^*) < 0 \tag{27}
\]

where

\[
\left. \frac{\partial TC}{\partial s} \right|_{s^*} = -\rho \cdot p^{ED}(s^*) (C^{DL}-C^{DE}) + C'(s^*). \tag{28}
\]

In order to see the relationship between the probability of disease and the socially optimal level of surveillance, we apply the implicit function theorem to equation (26) and obtain

\[
\frac{ds^*}{d\rho} = -\left\{ \frac{-u''(y - TC^*) \cdot \left( \left. \frac{\partial TC^*}{\partial \rho} \right|_{s^*} \right) \cdot \left( -\left. \frac{\partial TC}{\partial s} \right|_{s^*} \right) + p^{ED}(s^*) [u'(y - TC^*) (C^{DL}-C^{DE}) + (\kappa_L-\kappa_E)]}{SOC^*|_{s=s^*}} \right\} \tag{29}
\]

where

\[
\left. \frac{\partial TC^*}{\partial \rho} \right|_{s^*} = p^{ED}(s^*)C^{DE} + (1 - p^{ED}(s^*))C^{DL}, \tag{30}
\]

and \( SOC^*|_{s=s^*} \) is the expression in equation (27). Note that in describing the planner’s problem we need to recognize that the interpretation of cost as a function of \( \rho \) is different from that in the private optimization problem where \( \rho \) induces choice \( \hat{s}(\rho) \) and so leads to \( TC_e(\rho) = TC(\hat{s}(\rho), \rho) \), the resulting cost in equilibrium. For the social optimum, the planner facing disease probability \( \rho \) is of course not constrained to choose the privately optimal value of surveillance but can pick whatever value of \( s \) that maximizes the value of individual welfare (i.e., taking into account financial cost implications). This implies some schedule or relationship \( s^*(\rho) \) and hence some resulting total cost \( TC^*(\rho) = TC(s^*(\rho), \rho) \), recognizing that this function \( s^*(\rho) \) is not a constraint but rather an implication of the planner’s optimization decision. Comparing these schedules \( \hat{s}(\rho) \) and \( s^*(\rho) \), as well as \( TC_e(\rho) \) and \( TC^*(\rho) \), is of interest in considering policy implications of genetic testing as developed below.

It is helpful to rewrite the first-order condition in two ways:

\[
FOC^*a : \rho \cdot p^{ED}(s^*) \cdot (\kappa_L-\kappa_E) - \Phi'(s^*) = -u'(y - TC(s^*)) \left[ \rho \cdot p^{ED}(s^*) (C^{DL}-C^{DE}) - C'(s^*) \right]
\]
and

\[ FOC^* b : \rho \cdot p^{ED}(s^*) \cdot [(\kappa_L - \kappa_E) + u'(y - TC(s^*)) (C^{DL} - C^{DE})] = \Phi'(s^*) + u'(y - TC(s^*)) C'(s^*) \]

The first equation above (\( FOC^* a \)) illustrates that the socially optimal level of surveillance equates the net marginal health benefits for individuals (i.e., \( EU'(s^*) \)) ignoring any effect on financial cost implications for health care) to the net marginal (financial) cost of providing health care. Notice that for the under-utilization case both these values are negative (see Figure 7) while for the over-utilization case both these values are positive (see Figure 9).

The second equation (\( FOC^* b \)) reinterprets the socially optimal level of surveillance as that which equates the gross marginal benefit of surveillance to the gross marginal cost (i.e., for combined health and financial aspects). The gross marginal benefit is made up of that part due to physiological health benefits from early detection \( (\rho \cdot p^{ED}(s^*) \cdot [\kappa_L - \kappa_E]) \) plus that part due to financial savings from early detection. The gross marginal cost is made up of that part associated with physiological aspects of surveillance plus that part due to direct financial cost of providing surveillance. The ‘financial parts’ are multiplied by the scale or conversion factor \( u'(y - TC(s^*)) \) to reflect the marginal utility value of dollar costs.\(^{19}\) So, holding \( s \) fixed, an increase in \( \rho \) increases both the marginal health benefits and financial benefits of surveillance (\( LHS \) of \( FOC^* b \)) while leaving the marginal cost terms (\( RHS \) of \( FOC^* b \)) unchanged. Thus, with \( u''(\cdot) = 0 \) an increase in \( \rho \) increases the marginal net benefit of surveillance which implies a corresponding increase in \( s^* \).

Now, consider the case of \( u''(\cdot) < 0 \). Since an increase in \( \rho \) increases \( TC \), the scale factor \( (u'(y - TC)) \) on financial terms in \( FOC^* a \) (\( RHS \)) increases. So if \( \frac{\partial TC}{\partial s}|_{s^*} < 0 \) an increase in \( \rho \) enhances the effective net marginal financial benefit of surveillance (i.e., since an increase in \( s \) at \( s^* \) reduces this cost). Since an increase in \( \rho \) also (always) enhances the marginal net (physiological) health benefit of surveillance, it follows that under risk aversion and \( \frac{\partial TC}{\partial s}|_{s^*} < 0 \) we also have that \( \frac{\partial s^*}{\partial \rho} > 0 \). This is always so for the case of under-utilization of surveillance. However, if \( \frac{\partial TC}{\partial s}|_{s^*} > 0 \), the fact that an increase in \( \rho \) leads to higher \( TC \) and hence higher marginal utility of net income means the effective marginal financial cost of surveillance rises due to an increase in \( \rho \) (i.e., since an increase in \( s \) at \( s^* \) increases this cost in this case). Whether or not \( s^* \) will rise or fall due to an increase in \( \rho \) under these conditions depends on whether the increase in the marginal financial cost is greater or smaller than the associated increase in the marginal net health benefit of surveillance. We can summarize this result in the following proposition and corollary:

**Proposition 2.** For the relationship between the probability of disease and the socially optimal level of surveillance we have that, under risk-aversion, i.e., \( u''(\cdot) < 0 \):\(^{19}\)

19Under risk neutrality, this conversion factor is unchanged by any change in \( \rho \) and hence any change in the level of \( TC \).
1. \( TC'(s^*) = C'(s^*) - \rho \cdot p^{ED}(s^*) (C^{DL} - C^{DE}) \leq 0 \) is a sufficient condition for \( ds^*_{d\rho} > 0 \).

2. If \( TC'(s^*) = C'(s^*) - \rho \cdot p^{ED}(s^*) (C^{DL} - C^{DE}) > 0 \), then \( \frac{ds^*}{d\rho} \geq 0 \iff TC''(s^*) \leq \alpha \), where

\[
\alpha = \frac{p^{ED}(s^*)[u'(y - TC^*)(C^{DL} - C^{DE}) + (\kappa_L - \kappa_E)]}{-u''(y - TC^*)[p^{ED}(s^*)C^{DE} + (1 - p^{ED}(s^*))C^{DL}]}
\]  
(32)

\[
= \frac{p^{ED}(s^*)[(C^{DL} - C^{DE}) + \frac{(\kappa_L - \kappa_E)}{u'(y - TC^*)}]}{A(y - TC^*)[p^{ED}(s^*)C^{DE} + (1 - p^{ED}(s^*))C^{DL}] > 0}
\]  
(33)

and \( A(y - TC^*) = \frac{-u''(y - TC^*)}{u'(y - TC^*)} \).

**Corollary** In the case of under-utilization we have \( ds^*_{d\rho} > 0 \) for \( u''(\bullet) \leq 0 \) while in the case of over-utilization \( ds^*_{d\rho} > 0 \) for \( u''(\bullet) = 0 \) but the sign for \( ds^*_{d\rho} \) is indeterminate if \( u''(\bullet) < 0 \).

The intuition for the above results is more fully developed below. In the previous subsection it was shown that, when only the non-financial benefits and costs of surveillance are taken into consideration (i.e., for the privately optimal decision), the level of surveillance increases as the probability of disease increases. The reason was that a higher probability of disease raises the marginal non-financial benefit of surveillance. Here, in addition, we have to consider the financial benefits and costs of surveillance. With risk-neutrality, the marginal utility of income, and hence the willingness to pay for surveillance, is constant. This means that, with a higher probability of disease, there is an increased benefit from reducing treatment costs through surveillance (due to higher rates of early detection) even if total costs rise in surveillance. Along with the higher non-financial marginal benefit, this implies that the optimal level of surveillance increases in \( \rho \), the result specified in the Corollary to Proposition 2.

Under risk aversion, however, marginal utility decreases in net income and hence the willingness to pay for surveillance falls if \( TC \) rises. First assume that, at the initially optimal level of surveillance, the marginal savings of treatment costs exceed the marginal surveillance costs, and so \( TC'(s^*) < 0 \). Under this condition a marginal increase of surveillance will increase net income and thus increase the willingness to pay for more surveillance. A higher probability of disease will, other things equal, increase both the financial and non-financial marginal benefit of surveillance. The increase of the marginal benefits of surveillance, together with the higher willingness to pay for surveillance, lead unambiguously to an increase of the optimal level of surveillance.

However, in the case with the marginal financial costs of surveillance \( C'(s^*) \) exceeding the marginal financial savings \( \rho \cdot p^{ED}(s^*) (C^{DL} - C^{DE}) \), so that \( TC'(s^*) > 0 \), all other
things equal, an increase in the level of surveillance reduces net income and thus decreases the willingness to pay for more surveillance. The income effect of more surveillance, which is negative, opposes the favorable financial and non-financial effect of more surveillance being applied as the probability of disease becomes larger. If this negative (indirect) income effect of surveillance is strong enough, i.e. if health care costs increase sufficiently fast in the level of surveillance, an increase of the probability of disease can even lead to a decrease of the optimal level of surveillance.\textsuperscript{20}

Observing the above expressions, a (counter-intuitive) negative relationship between the probability of disease and the socially optimal level of surveillance is more likely to result: (1) the smaller are the marginal savings of financial treatment costs and non-financial psychological costs when the disease is detected early rather than late (i.e., the smaller are \((C^{DL} - C^{DE})\) and \((\kappa_L - \kappa_E)\)), and the less effective is \(s\) (i.e., the stronger is the curvature of \(p^ED(s)\)), (2) the larger are the marginal financial costs of surveillance \(C'(s^*)\) (i.e., the faster the costs of surveillance increase in the level of surveillance), (3) the higher are the increased treatment costs in general \((\frac{\partial TC}{\partial \rho})\), and (4) the higher is the degree of absolute risk aversion (i.e., the more quickly the willingness to pay for health care falls as net income falls).\textsuperscript{21}

We now develop some results concerning the pattern of over versus under-utilization of surveillance for different information scenarios (i.e., concerning the perceived probability of disease both in the absence and presence of genetic tests). It is certainly possible that both in the absence of a person having a genetic test \((\rho = \rho^D)\) and for both risk types conditional on a genetic test \((\rho = \rho^L\) or \(\rho^H\) as the case may be), either all individuals under-utilize or all individuals overutilize surveillance. It seems unlikely, however, that the extent to which this would be the case would be uniform over these different information sets. Below we show conditions under which one group may overutilize while another group underutilizes surveillance. Thus, a single policy for all individuals developed to try to rectify this problem, say rationing or taxing (through coinsurance payments) of surveillance in the over-utilization case or encouraging additional surveillance by some means in the under-utilization case, would not be appropriate. We address this exercise using what one may consider the perhaps more plausible case of moderate marginal surveillance costs such that, when the probability of disease increases, the socially optimal level of surveillance will increase (Assumption 1 below).

**Assumption 1:** \(TC'(s^*) < \alpha\) and hence \(\frac{ds^*}{d\rho} > 0\) for all relevant values for \(\rho\).

\textsuperscript{20}Note that this is an indirect income effect since utility from income is assumed separable from activity associated with any change in health status. Upon relaxing this separability assumption, the income effect becomes more complicated to analyze as illustrated in the Appendix, but it can still be negative.

\textsuperscript{21}Notice that \(A(.)\) appears in the denominator of \(\alpha\) and so the higher is \(A(.)\), that is, the higher is the degree of curvature of \(u(.)\), the more quickly marginal utility of income rises as \(TC\) rises.
Our second assumption is not intended to be a realistic expectation concerning an original scenario in the absence of a genetic test, but rather simply reflects a useful hypothetical starting point to allow us to develop an understanding of scenarios in which the schedules $s^*(\rho)$ and $\tilde{s}(\rho)$ may intersect, giving rise to the possibility that, conditional on having a GT, one risk type over-utilizes surveillance while the other type under-utilizes surveillance.

**Assumption 2:** Before a genetic test is conducted, the private demand for and the socially optimal level of surveillance coincide, that is $\tilde{s}(\rho_0) = s^*(\rho_0)$. Thus, for $\rho_0$ it holds that $TC'(s^*(\rho_0)) = 0$, and hence, $C'_{DL-C^D} = \varphi'(s^*(\rho_0))$.

First we develop the intuition and some graphs to understand two different patterns in how $\bar{s}$, $s^*$, and $\tilde{s}$ change as $\rho$ changes. Recall that, at a given value of $\rho$ (probability of disease), if the per capita financial cost of health care is falling in $s$ at the privately optimal level of surveillance ($s = \bar{s}$), then the consumer is under-utilizing $s$ and the cost-minimizing level of $s$ exceeds the privately optimal level as well as the socially optimal level of surveillance (i.e., $\bar{s} > s^* > \tilde{s}$ - see Figure 7). Therefore, if starting from a position $(\rho = \rho_0)$ in which these three levels of surveillance are equal it is the case that use of surveillance is, at the margin, relatively ineffective in reducing the financial savings from early detection while being relatively costly to provide, then an increase in $\rho$ will induce a relatively small increase in $\bar{s}$. This enhances the likelihood that the corresponding increase in the privately optimal choice of surveillance ($\tilde{s}$) will be larger than the increase in $\bar{s}$, leaving one in a position of $\bar{s} < s^* < \tilde{s}$ for $\rho > \rho_0$ and vice versa for $\rho < \rho_0$.

In other words, the schedule $\tilde{s}(\rho)$ will be flatter than the schedule $\bar{s}(\rho)$, which in turn implies it is also flatter than the schedule $s^*(\rho)$. This possibility is illustrated in Figure 10. The corresponding case for surveillance being relatively effective in reducing financial savings from early detection while being relatively inexpensive to provide - at the margin - is illustrated in Figure 11. The formal requirements for these two cases are described below.\(^{22}\)

**Case 1: Surveillance is relatively ineffective and costly at the margin** (In this scenario we assume that $p^{EDr}(s)$ and $p^{EDh}(s)$ are small enough for all $s$; and $C'(s)$ and $C''(s)$ are large enough for all $s$, such that the following inequality holds). Suppose that the marginal financial costs of surveillance increase in the probability of disease:

$$
\frac{d}{d\rho} \left( \frac{\partial TC}{\partial s} \right) = \frac{\partial^2 TC}{\partial s \partial \rho} + \frac{\partial^2 TC}{\partial s^2} ds > 0 \text{ for all } \rho, \quad (34)
$$

where

$$
\frac{\partial^2 TC}{\partial s \partial \rho} = -p^{EDh}(s)(C^{DL} - C^{DE}) < 0, \quad (35)
$$

\(^{22}\)If there are bounds on the feasible values for $s$, then there can be horizontal portions at the “ends” of some/all of the functions $\tilde{s}(\rho)$, $s^*(\rho)$, and $\bar{s}(\rho)$. 

29
Figure 10: Surveillance ineffective and costly at the margin
Figure 11: Surveillance effective and inexpensive at the margin
\[
\frac{\partial^2 TC}{\partial s^2} = C''(s) - \rho p^{ED''}(s)(C^{DL} - C^{DE}) > 0,
\]  
and \(\frac{ds}{d\rho} > 0\) by assumption. The indirect effect of the probability of disease on the marginal costs of surveillance \(\left(\frac{\partial^2 TC}{\partial s^2} \frac{ds}{d\rho}\right)\), which is positive, is stronger than the direct effect \(\left(\frac{\partial^2 TC}{\partial s^2}\right)\), which is negative. For this relationship to hold, the costs of surveillance and the probability of early detection must be strongly curved in the level of surveillance \((C''(s), |p^{ED''}(s)| >> 0 \ \forall s)\).

Together with assumption 2, this implies that \(TC''(s^*(\rho)) < 0\) for small values of \(\rho\), including \(\rho^L\), and \(TC''(s^*(\rho)) > 0\) for high values of \(\rho\), including \(\rho^H\). As was shown above, this is equivalent to a situation, where \(\frac{C'(s^L*)}{C^{DL} - C^{DE}} < \frac{\Phi'(s^L*)}{\kappa_L - \kappa_E}\) and \(\frac{C'(s^H*)}{C^{DL} - C^{DE}} > \frac{\Phi'(s^H*)}{\kappa_L - \kappa_E}\), and \(\tilde{s}^H > s^H\) and \(\tilde{s}^L < s^L\) hold respectively (high risks overinvest in surveillance, low risks under-invest in surveillance).

**Case 2: Surveillance is effective and inexpensive at the margin.** Suppose that the marginal costs of surveillance decrease in the probability of disease, \(\frac{d}{d\rho} \left(\frac{\partial TC}{\partial s}\right) = \frac{\partial^2 TC}{\partial s^2} + \frac{\partial^2 TC}{\partial s^2} \frac{ds}{d\rho} < 0\) for all \(\rho\). This implies that the direct effect of the probability of disease on the marginal costs of surveillance, which is negative, must exceed the positive indirect effect. This relationship is more likely to hold if the costs of surveillance and the probability of early detection are “almost linear” in the level of surveillance \((C''(s), p^{ED''}(s) \approx 0 \ \forall s)\). Together with assumption 2 this implies that \(TC''(s^*(\rho)) > 0\) for small values of \(\rho\), including \(\rho^L\), and \(TC''(s^*(\rho)) < 0\) for high values of \(\rho\), including \(\rho^H\). In this case, the financial marginal cost-benefit ratio is small for high probabilities of disease and large for low probabilities of disease, \(\frac{C'(s^L*)}{C^{DL} - C^{DE}} > \frac{\Phi'(s^L*)}{\kappa_L - \kappa_E}\) and \(\frac{C'(s^H*)}{C^{DL} - C^{DE}} < \frac{\Phi'(s^H*)}{\kappa_L - \kappa_E}\), and \(\tilde{s}^H < s^H\) and \(\tilde{s}^L > s^L\) (high risks under-invest in surveillance, low risks overinvest in surveillance).

The above analysis explains the relationship between the (financial) cost minimizing, privately optimal, and socially optimal levels of surveillance as a function of some known or perceived probability of disease. Clearly there is a tension in that the socially optimal level of surveillance may be either lower or higher than the privately optimal choice of individuals when insurance plans allow any desired level of surveillance by consumers and this cost is borne by the insurance pool. In the case of over-utilization, this source of inefficiency can be avoided if the insurance provider can ration or tax (through coinsurance payments) the use of surveillance. Solutions to under-utilization perhaps create a more subtle problem. One could create financial incentives for consumers to increase their utilization of surveillance by taxing (through coinsurance) the financial cost of treating disease since this is greater for late detection than for early detection. Of course, this

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23The case of \(C''(s) \approx 0\) includes the situation of greater surveillance being achieved by simply increasing the frequency of use of a single monitoring technology.

24Byrne and Thompson (2001) consider economic incentives to correct for under-screening or suboptimal prevention in the context of myopia (i.e., improper discounting).
creates a conflict with the risk reducing property of full insurance and/or the notion of solidarity of public health insurance. This aspect applies to surveillance levels as well in the ex post scenario of genetic testing given the different utilization rates implied for the two risk types. Moreover, our results demonstrate that the relationship between over or under-utilization of surveillance and the variation in the probability of disease across genotypes is not a simplistic one. The coinsurance rate or even the same sign of an implicit tax (subsidy) on surveillance would typically have to vary depending on the probability of disease (risk type) and such flexible instruments may not be politically feasible. Moreover, the issue becomes significantly more complex if one were to allow for other sources of heterogeneity across individuals (e.g., differences in the function $\Phi(s)$ or the value of $[\kappa_L - \kappa_E]$ across individuals). We leave these questions for future research although raise them in the discussion section.

Thus, only if the socially optimal levels of $s$ can be obtained (i.e., $\hat{s}^L = s^{L*}$ and $\hat{s}^H = s^{H*}$) by way of some instruments that do not create any loss of utility due to the introduction of risk-bearing costs, can it be assumed that a costless genetic test will always improve welfare (at least weakly) in the case of a mandatory public health insurance plan with community rating. This follows simply because the social planner always has the option of maintaining the same level of surveillance as before genetic tests are taken for both risk types. This would imply the same expected financial costs and health benefits/costs due to the information. Thus, any alterations in the risk-type specific level of surveillance that could improve average welfare, if possible, would be undertaken by the social planner (or by the insurance provider).

### 3 A model of prevention

In this section we outline the changes to the model for surveillance required to represent the decision to take preventive measures that are both financially costly from the insurance provider’s perspective and physiologically costly from the individual’s perspective. An example would be the use of prophylactic mastectomy or chemoprevention (e.g., tamoxifen) sometimes taken by individuals who are sufficiently highly predisposed to breast cancer. The model is very similar to that for surveillance but of course the interpretation is quite different. Moreover, the structure imposed on the model is different and has different motivation. We keep the basic framework of the model in the previous section except that this time we are interested in how the level of prevention changes the probability of getting the disease at all.

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25 See, for example, Anderson, et al. (2006) for a discussion of possible preventive strategies suggested for women with a BRCA1 or BRCA2 mutation.
Prevention may be either observable by the health care provider (surgery, therapy) or unobservable (lifestyle). We focus on observable medical preventions, which we denote by $m \in [m_-, m_+]$. The probability of disease is a function of the level of prevention $\rho(m)$, with $\rho'(m) < 0, \rho''(m) > 0$.

We regard the level of surveillance as fixed and summarize the financial cost of treatment, which is assigned in case of disease, as $C_D$, and the non-financial, physiological cost of disease as $\kappa$. Note that in terms of the model on surveillance, we could set $C_D = p^{ED}_E C^{DE} + (1 - p^{ED}) C^{DL}$ and $\kappa = p^{ED}_E \kappa_E + (1 - p^{ED}) \kappa_L$ and include a second decision, that of surveillance represented by $s$ in the previous model. Alternatively, one can see that consideration of only one or the other decision variable $m$ or $s$ allows for recognizing the similarity in the two models through the relationships $m \equiv s$ with $\rho'(m) \kappa \equiv \rho'[p^{ED}_E(s) \kappa_E + (1 - p^{ED}(s)) \kappa_L] = \rho[-p^{ED}_E(s) \kappa_L + \kappa_E + \kappa_L]$ in which case properties of the function $\rho(m)$ play a similar role to properties of the function $p^{ED}(s)$.

A similar relationship can be drawn between the functions $TC(s)$ and $TC(m)$. Thus, the expected utility of individuals with probability of disease $\rho(m)$ is

$$EU(m) = u(y - TC(m)) - \rho(m) \cdot \kappa - \Phi(m)$$

where

$$TC(m) = \rho(m) \cdot C^D + C(m)$$

are the financial health care costs which are incurred per capita to the health care system. $C(m)$ is the financial cost of prevention, where $C'(m) > 0, C''(m) > 0$. $\Phi(m)$, with $\Phi'(m) > 0, \Phi''(m) > 0$, is the non-financial (physiological) cost of prevention incurred by the individual.

Before a GT is conducted, the perceived probability of disease for a given level of prevention $m$ is the same from the viewpoint of all individuals and the health care provider, and we denote it by $\rho^0(m)$. As before, a genetic test classifies individuals into two risk groups - low and high - which have probabilities of disease $\rho^L(m)$ and $\rho^H(m)$ respectively. Thus, for the same level of prevention $m$, the population average probability of disease can be written as $\rho^0(m) = \eta_L \rho^L(m) + \eta_H \rho^H(m)$, where $\eta_L$ ($\eta_H$) is the proportion of tested negatives (positives), and $\rho^H(m) > \rho^0(m) > \rho^L(m)$.

Let the probabilities of disease for low risks and high risks be

$$\rho^L(m) = \rho^0(m) - \frac{e \cdot \varepsilon(m)}{\eta_L} \quad (37)$$

$$\rho^H(m) = \rho^0(m) + \frac{e \cdot \varepsilon(m)}{\eta_H} \quad (38)$$

where $e$ is the precision of the test, and $\varepsilon(m) > 0$ reflects the difference in the effect of the level of prevention on the probabilities of disease for the two risk types.
Further, we assume that \( \varepsilon(m) \) is such that the second order conditions hold for those tested positive or negative. Specifically, we assume that, after a GT is conducted, both for tested positives and tested negatives the probability of disease is decreasing and convex in the level of prevention, i.e. \( \rho^H(m) < 0, \rho^{H''}(m) > 0 \) and \( \rho^L(m) < 0, \rho^{L''}(m) > 0 \) for all \( m \). With the above specifications this is equivalent to

\[
\rho^H(m) = \rho^0(m) + \frac{e \cdot \varepsilon'(m)}{\eta_H} < 0, \quad (39)
\]

\[
\rho^{H''}(m) = \rho^{0''}(m) + \frac{e \cdot \varepsilon''(m)}{\eta_H} > 0.
\]

and

\[
\rho^L(m) = \rho^0(m) - \frac{e \cdot \varepsilon'(m)}{\eta_L} < 0, \quad (40)
\]

\[
\rho^{L''}(m) = \rho^{0''}(m) - \frac{e \cdot \varepsilon''(m)}{\eta_L} > 0.
\]

The difference between the probabilities of disease of those tested positive and negative for the same level of prevention \( m \), is

\[
\rho^H(m) - \rho^L(m) = \frac{e \cdot \varepsilon(m)}{\eta_L \eta_H}.
\]

For \( e = 1 \) (perfect precision of the test) those probabilities of disease can be regarded as the true probabilities of disease of high risks and low risks, while for \( e < 1 \) (imperfect precision) they are simply the average probabilities of disease for tested positives and tested negatives.

Notice that we make no assumptions about the sign of \( \varepsilon'(m) \) or of \( \varepsilon''(m) \); that is, we allow for the difference between high and low risk types to become either wider or narrower with increased level of self prevention \( m \). We do, however, maintain the requirement that for any value of \( m \), \( \rho^H(m) > \rho^L(m) \) and that \( \rho^{H''}(m), \rho^{L''}(m) > 0 \).\(^{26}\) An increase in the precision of the test \( e \) is used to reflect both a smaller rate of false negatives and a smaller rate of false positives. Thus, for a given population, the average probability of

\(^{26}\)This is similar to the characterization of the difference between safety technologies in Hoy (1989). There is no restriction on the sign of \( \varepsilon''(m) \), but its absolute size cannot be such that one of \( \rho^H(m), \rho^L(m) \) is concave.
disease before a genetic test is conducted is $\rho^0(m)$, a bigger difference between the observed probabilities of disease, or a more precise test in general (i.e. with both a lower rate of false positives *and* false negatives) is reflected by a larger $e$.

From an individual’s perspective the level of prevention has no effect on health care costs $\frac{\partial TC(m)}{\partial m} = 0$. The private choice of prevention $\hat{m}$ is found as the solution to

$$FOC : -\rho'(\hat{m}) \cdot \kappa = \Phi'(\hat{m})$$

where

$$SOC : -\rho''(\hat{m}) \cdot \kappa - \Phi''(\hat{m}) < 0.$$ 

For the socially optimal level of prevention $m^*$, for which the effect on health care costs is taken into account, it holds that

$$FOC^* : u'(y - TC(m^*)) \cdot (-\frac{\partial TC(m)}{\partial m}|_{m=m^*} - \rho'(m^*) \cdot \kappa - \Phi'(m^*) = 0$$

where

$$\frac{\partial TC(m)}{\partial m}|_{m=m^*} = \rho'(m^*) \cdot C^D + C'(m^*).$$

For the second order condition we get

$$SOC^* : u''(y - TC(m^*)) (-\frac{\partial TC(m)}{\partial m}|_{m=m^*})^2 + u'(y - TC(m^*)) (-\frac{\partial^2 TC(m)}{\partial m^2}|_{m=m^*}) - \rho''(m^*) \cdot \kappa - \Phi''(m^*) < 0,$$

where

$$\frac{\partial^2 TC(m)}{\partial m^2}|_{m=m^*} = \rho''(m) \cdot C^D + C''(m) > 0$$

for all $m$.

A comparison of the first order conditions for the the private choice of prevention and the socially optimal level shows that

**Lemma 1:** $m^* \leq \hat{m} \iff \frac{\partial TC(m)}{\partial m}|_{m=m^*} = \rho'(m^*) \cdot C^D + C'(m^*) \geq 0.$

As we did for the model of surveillance, one can derive similar relationships between the privately optimal level of prevention, $\hat{m}$, the cost minimizing level of prevention, $\tilde{m}$, and the socially optimal level of prevention, $m^*$, as well as between the pattern of over and under use of prevention for different information sets (i.e., knowledge of $\rho^0(m)$, $\rho^H(m)$, and $\rho^L(m)$). The only added complication from a modeling perspective is that the difference in genetic types for the model of surveillance represents a straightforward difference in probability of disease, with $\rho^H > \rho^L$, while to model genome specific prevention we need a difference in functions, $\rho^H(m) > \rho^L(m)$ where the relationship between $\rho^H(m)$ and $\rho^L(m)$ is important and may vary across multifactorial genetic diseases.\(^{27}\)

\(^{27}\)Multifactorial genetic diseases are those for which both genes and environment (including possibly medications, surgery, etc.) interact to generate a probability of disease or degree of severity of disease.
possibility of over and under prevention is possible as well as the possibility of a rich set of patterns of over and under prevention being generated by genetic testing. Thus, attention should be paid to correct for such inefficiencies due to moral hazard considerations. Again, the instruments required may seem horizontally inequitable in that the optimal policy may imply that one group face a tax (through coinsurance payments) on preventive activities while another group may face a subsidy.

4 Discussion

In this section we present some discussion intended to place the results of the model into the real world context of health care decision making. The points of discussion are grouped into two areas. These relate to (1) the model of decision making by individuals and how that relates in a more realistic context to the role of the health care system and professional advice; (2) expansion of the relationship between genetic test results and various medical strategies, including surveillance as modeled in this paper, that may follow.

4.1 Individual decision making and the role of the health care system

The model of decision making at the individual level in the context of the type of medical information involved here may admittedly be considered ultra-rational. It is even difficult in many cases for health care professionals to assess probabilities of alternative outcomes of genetic tests and surveillance procedures. However, there is some evidence that individuals do respond to information in a manner that reflects rational processing of probabilistic information in an expected utility framework. Of particular relevance to this paper, are studies on the demand for surveillance measures that find evidence suggesting that increased risk of certain diseases does lead individuals to increase their level of surveillance. For example, in a study on the demand for three measures for early detection of breast and cervical cancer, Picone, et al. (2004) find that information on increased risk of these diseases leads to an increase in individuals' choice of level of surveillance. Witt (2007) finds that women who are at a higher risk of breast cancer as suggested by family history are more likely to demand a mammogram. Nonetheless, one should be very cautious in accepting results such as those in this paper without reservations. The attention paid to the importance of bundling genetic counseling with genetic services when tests are provided by the health care system is well placed. It is important to explain to individuals the relative odds of disease onset associated with alternative genetic tests and their results as well as both the value of health benefits and costs arising from any resulting follow-up strategies such as increased surveillance and/or preventive measures. Recent commentary/research about direct-to-consumer genetic testing services offered through
the internet (e.g., see www.23andme.com) raises concerns about the ability of consumers to process and rationally respond to such information if it is not provided by the health care system itself. Our model of rational individual decision making through the use of the expected utility hypothesis is probably a more accurate description of behavior under the presumption of substantial help from the health care professionals involved in explaining information and consequences of various medical strategies of our model.

Counselling services from health care professionals that may help individuals understand the outcomes of tests and strategies, however, do not correct for the wedge between privately and socially optimal decisions resulting in the difference between individual demand for surveillance, $\hat{s}(\rho)$, and the socially optimal level, $s^*(\rho)$. For example, suppose many of those who perfectly understand the extent of the increased risk of a disease resulting from a positive genetic test wish to increase surveillance beyond what is socially optimal (see Figure 9). This is perfectly rational individual decision making. Our model demonstrates that in some cases the health care system must respond to such pressures by denying the full demand for increased surveillance if a social optimum is to be achieved. Moreover, we have shown that this may in fact be the case even when those with negative genetic test results, or those who haven’t been tested, do not need to have their private demands for surveillance rationed. Doctors are often modeled as gatekeepers of medical services but such practices can sometimes be difficult to carry out when patients’ desires are in conflict with the socially optimal provision of services. In other instances, individuals under-use surveillance - possibly only after a genetic test is received (see Figure 10) - and the socially optimal level of surveillance can only be achieved by encouraging an individual to submit to a higher level of surveillance than the individual wishes. This can also present a challenge to “doctors as gatekeepers”. Doctor-patient trust can be eroded by such conflicts.

Another important feature of our model that departs from reality is that the desire or willingness to accept a genetic test is taken in isolation of any implications other than health care. From such a narrowly described expected utility model of decision making, since expected utility is linear in probabilities, a GT would never have negative private value in our model. Moreover, individuals would place positive value on such an opportunity to acquire such information if any behavioral change whatsoever would be implied, conditional on at least one of the possible results of the genetic test.\(^{28}\) It is widely documented, however, that even when individuals have access to free genetic tests and free counseling, many do not accept such offers. For example, Meiser and Dunn (2000) note that the percentage at risk for Huntington disease (HD) who requested testing varied from 9% to 20% in various centers in UK cities and Vancouver despite the offer being at no

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\(^{28}\)For a detailed explanation of these implications see Hirshleifer and Riley (1992), pp. 179-185.
cost. One reason for such a low rate of demand for the genetic test in the case of HD may be that there is little value in the test from the perspective of health care decisions since no effective treatment (or relevant surveillance) for the disease exists. On the other hand, some aspects of life planning conditional on whether one has an HD allele presumably could be improved upon (i.e., outside of the health care framework) and so the test might be presumed to carry a positive value in the context of a broader interpretation of agents behaving according to the expected utility hypothesis. There are, however, many implications of genetic test results that are external to the sort of decision making considerations mentioned above. There is a substantial literature about how individuals perceive possible genetic test results affecting their lives in manners that would suggest personal consequences, such as possible social stigmatization, reduced access to employment or life insurance opportunities, possibly negative impact on family members or relationships, that lie outside of a model focusing on optimal health care strategies. This has lead many to emphasize the idea that individuals should not be pressured to obtain genetic tests; a concept referred to as the “right not to know.”

### 4.2 On the use of surveillance and other medical strategies

In many cases there is substantial debate in the medical community about the effectiveness of certain surveillance tests and related medical procedures, which in turn would impact on the value of a particular genetic test. An important factor for evaluation of surveillance tests, and hence the genetic tests that imply changes in surveillance levels conditional on GT results, are the associated rates of false positives (for both sets of tests) and the consequences of increased surveillance or preventive measures on any misclassified individuals. The PSA test for prostate cancer with a false positive rate exceeding 50% is such an example. In modeling the informativeness of the signal that arises from a genetic test, we have avoided studying the distinction between false negatives and false positives (i.e., the distinction between sensitivity versus specificity of the test). For many diseases the type of surveillance or preventive measures that one would recommend or adopt will depend on the relative size of these two characteristics of the test. Summarizing the informativeness or accuracy of the test using a single parameter ($\varepsilon$ - in conjunction with

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29 See also Babul et al. (1993) and Quaid and Morris (1993).
30 See Hoy and Ruse (2005) for a broad discussion of insurance market and other implications that can reduce the value of genetic tests.
31 See Lemmens, et al. (2008) for a discussion.
32 See [http://www.cancer.gov/cancertopics/factsheet/Detection/PSA#r3](http://www.cancer.gov/cancertopics/factsheet/Detection/PSA#r3). The American Cancer Society has questioned its value and suggest decisions on this test need to be made according to presonal preference and by informed individuals (see related story and links at [http://www.cnn.com/2010/HEALTH/03/03/prostate.screening/](http://www.cnn.com/2010/HEALTH/03/03/prostate.screening/)).
$\eta_L$ and $\eta_H$ fixed) as we have done in this paper makes both the derivation of results and underlying intuition more straightforward. However, we have implicitly and artificially fixed the relationship between the rates of false positives and negatives. Perhaps even more importantly, we have modeled the result of the surveillance technology as determining a relationship between intensity of the surveillance strategy and presence of a disease also with no explicit consideration of false positives and negatives of the surveillance technology.

Introducing richer sets of parameters to represent informativeness for both the genetic tests and the surveillance (or preventive) technologies would lead to a much greater degree of complexity in our model and loss of intuition. However, one can consider these implications informally in terms of their effect on the utility values for early versus late detection (i.e., $w^{ED}(\cdot)$ and $w^{LD}(\cdot)$). Suppose an individual tests positive for the predisposition gene, which may be a false positive, and also is determined by surveillance activities/tests to have the disease in early stages. For the sake of this discussion suppose late stage disease is always detected with 100% accuracy. If the test suggesting early detection is prone to a high rate of false positives, then it may well be prudent to take only a very mild invasive medical strategy to deal with the increased possibility of disease existing. A higher rate of false positives from the genetic test would exacerbate this problem as more individuals who do not have the high predisposition to disease will undergo surveillance, thus increasing the overall rate of people without the disease having a signal of early detection.

Some insights into the above issues can be obtained by thinking of the person who has ultimately received a positive indication from surveillance for having the disease (detected early) to be facing the probability pair $\rho_{pos}$ and $(1 - \rho_{pos})$ of actually having or not having the disease, respectively, while those not receiving such an indication face probability pair $\rho_{neg}$ and $(1 - \rho_{neg})$, with $\rho_{pos} > \rho_{neg}$. Then suppose using these probabilities one chooses the best practice treatment or set of medical strategies, which might simply involve watchful waiting if $(1 - \rho_{pos})$ is sufficiently high, for those who received the positive indication of early detection of the disease. Then $w^{ED}(\cdot)$ can be treated as the expected utility from such a path of interventions (i.e., for those receiving a positive test result for early detection from surveillance) while $w^{LD}(\cdot)$ will reflect expected utility for those who do not receive an indication of early onset of disease (accepting the fact that some of these people received false negatives in part due to a lower rate of surveillance).\footnote{Of course, some people who didn’t receive a positive test from whatever level of surveillance they adopted end up better off than some in the early detection category. The important feature would be that, conditional on receiving an early indication of disease, expected value of future utility $w^{ED}(\cdot)$ is higher as a result of following the best practice medical strategy than if this signal is ignored.}
those who have a negative initial indication of disease. Thus, a higher rate of either false
negatives or false positives will imply a smaller difference between \( w^{ED}() \) and \( w^{LD}() \)
and hence a lower value to genetic testing and smaller difference in surveillance rates for
those who have a positive GT result versus those with a negative GT result. However, if
best practice is followed conditional on test results and knowledge of false positives and
false negatives, then it still follows that the difference \( w^{ED}() - w^{LD}() \) will be positive
and the same sorts of results as we have found would be relevant. Moreover, the value
of a genetic test would be a decreasing function of both measures of informativeness (i.e.,
both for false positives and negatives) for both the genetic test itself and the surveillance
technology. Modeling the difference between what is individually optimal versus socially
optimal would be done in an analogous manner with similar qualitative results.

Similar concerns would be introduced by the false positive/negative distinctions con-
cerning the informativeness of tests for the case of prevention. For example, if the false
positive rate were relatively high for a genetic test for colon cancer predisposition, then the
value of certain chemopreventive or surgical strategies to prevent development of cancer
would be lower. However, treatments with less serious side effects for those improperly
classified may well improve future expected utility. In either the surveillance or prevention
case, if the risk of treating those who don’t actually have the disease or who are not in fact
so highly predisposed to the disease due to inaccuracy of the genetic test is sufficiently ad-
verse, then no action following a positive genetic test might represent best practice. This
would imply a zero value for the genetic test from the perspective of immediate medical
decisions.

We have ignored heterogeneity of individuals in our model. Family history can often be
used to create different subpopulations facing different likelihoods of receiving a positive
genetic test result. This specific feature is not difficult to include in our analysis as it
simply implies the relevant parameters vary across such subgroups as would the value
of a GT. However, it may also be difficult for individuals to understand the relationship
between family background and the way in which a genetic test relates to these, especially
if false positives and negatives are in play. So genetic counseling also needs to take such
factors into account. Another, and perhaps even more problematic, type of heterogeneity
is the likelihood of different personal preferences over the physiological benefits and costs
of surveillance or prevention. Some people may simply have a higher disutility from certain
surveillance procedures. This makes it difficult to decide, for example, which individuals
should receive higher surveillance levels and which lower surveillance levels in order to
obtain a social welfare optimum. Such information about preferences would be private
information and this represents a serious challenge to rationing decisions (of surveillance)
and, more generally, to so-called one-size-fits-all health insurance which tends to be a
natural feature (at least formally) of public health insurance plans. But this feature is not unique to our problem.

5 Conclusions

In this paper we have considered the implications of improved information about risk type from genetic tests on two aspects of behavior in health care: (1) surveillance (monitoring) to improve the chances of early detection of disease onset and (2) preventive actions to reduce the probability of onset of disease. We have developed models to provide some intuition into the question of how both private demand (individual's desire) and the socially optimal levels for these health care strategies may change as a result of the acquisition of information from genetic tests. This is modeled in the setting of a pure public health insurance program; that is, a health care system which is compulsory with 100% coverage of included medical procedures and with no user fees. Information from genetic tests has the potential to improve the targeting of surveillance or preventive health care strategies, and hence improve expected welfare (health outcomes) for individuals. However, moral hazard considerations inherent with full coverage insurance can imply that improved information leads to a reduction in social welfare through the reactions in the health care system.

The channel through which improved information from genetic tests can lead to a reduction in social welfare arises from individual incentives to choose levels of surveillance (or prevention) that are privately optimal but that ignore the financial cost implications for the insurance pool. In the absence of risk type specific information individuals may well choose to under-utilize or over-utilize surveillance or prevention activities from a social welfare perspective. Individuals will wish to use more (less) of these activities if their risk of disease is determined to be higher (lower) as the result of a genetic test and this improves their privately determined level of well-being. However, the average per capita financial cost of providing health care may either increase or decrease as a result of these behavioral changes and these financial implications are not internalized by the individuals. If this cost decreases, then the result of a costless genetic test will be an increase in social welfare. If this cost increases, then one must weigh the relative degree of inefficient use of medical resources before and after the introduction of the improved information against the change in expected health benefits to determine whether welfare will rise or fall. A reduction in social welfare is possible and we have outlined some of the factors that need to be considered to determine which outcome will be the case. Moreover, for a costly genetic test, our methodology can help in calculating the gross benefits and costs from the information in order to determine whether the genetic test is a worthwhile
addition to covered medical procedures for the public health insurance plan.

Our model also aids in identifying scenarios in which individual demands (desires) for surveillance leads to over-utilization versus under-utilization and for which risk (geno-) type. Thus, to the extent that the health care system can control the usage through rationing or promotion of surveillance, our model aids in this determination. We have acknowledged in the paper, however, that it may be difficult for a health care system that uses doctors as gatekeepers to deal with these conflicts in personal versus societal goals.\textsuperscript{34} Thus, user fees and selection of what tests and procedures to include and promote in a health care system may help to reconcile private incentives and social goals if these measures are politically acceptable in a public health care regime.

The information from a genetic test generates a mean preserving spread in the perceived probability of disease onset. We have identified as a key issue in determining the social value of a specific genetic test the way in which behavioral changes adapt to such information to lead to an equilibrium cost function that is either convex or concave in an individual’s perceived probability of disease onset, $\rho$ (or function $\rho(m)$ in the model of prevention). If the cost function is strictly convex in $\rho$, then the genetic test will create an increase in the average cost of health care provision, and vice versa if it is strictly concave. We have shown that which of these results obtains depends on the rate at which increased surveillance increases the probability of early detection of disease as well as how quickly the cost of providing improved surveillance rises. However, the overall determination of the curvature of this equilibrium cost function also depends on behavioral responses to surveillance resulting from changes in one’s perceived risk of disease, as developed through the function $\hat{s}(\rho)$. Thus, contrary to some expectations, it is not necessarily the case that improved information from genetic tests will lead to cost savings from “better targeted” use of existing medical technologies, although of course it may.

Many countries with substantial coverage through private health insurance plans have prohibitions on risk-rating of premiums as well as mandatory coverage of certain items. Thus, our models and results can provide some guidance for private health insurance scenarios as well. However, private insurance regimes tend to be more open to user fees and copayments. These offer additional instruments for influencing private choices of surveillance or prevention and so may offer some interesting avenues for future research. However, since the burden of copayments and user fees would fall differentially according to risk type, as determined by genetic test results, use of such instruments would create a phenomenon akin to premium risk that would reduce their value through increased risk-bearing costs on individuals (see Hoy and Polborn (2000) and Hoy and Ruse (2005)). Of

\textsuperscript{34}The possibility of using taxes and/or subsidies to elicit efficient levels of screening and prevention is investigated in a different context in Byrne and Thompson (2001).
course, community rating requirements can blunt or remove this cost. This presumably is the intent of the so-called GINA bill (Genetic Information Nondiscrimination Act) in the USA that President Bush signed the into law on May 21, 2008, after about 10 years in the making. This bill prohibits insurance companies from using genetic test results as a means of rate-making for health insurance as well as excludes firms from using such information for employment decisions. However, risk selection issues may substitute for risk-rating and create a different sort of problem.\(^{35}\)

Several directions for future research include, among others, (1) allowing for heterogeneous preferences regarding the health benefits and costs of surveillance or prevention activities, (2) explicitly modeling false negative/positive characteristics for both genetic tests and surveillance techniques, (3) allowing for a model of preventive activity that involves only personal costs - such as dietary change - and which may or may not be private information, (4) explicitly introducing second-best instruments such as coinsurance on either surveillance (prevention) costs or treatment costs, (5) considering models of private insurance and introducing asymmetric information regarding whether a genetic test has been taken, (6) consideration of the types or characteristics in the development of new types of surveillance technologies as a result of increased genetic information availability,\(^{36}\) (7) including additional social costs related to morbidity and mortality that are external to the health care system and are not internalized by individuals.

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\(^{35}\)As noted in Wagstaff et al. (1999, p. 269), “It is well known that during the last decade or so, there has been a shift in many OECD countries away from public sources of finance (for health care) to private sources.” If insurance continues to fall under a community rating regulation, however, the risk premium issues identified here would vanish although risk selection issues might arise.

\(^{36}\)See Bhattacharya and Packalen (2008) for a model relating individual’s inefficient adoption of prevention with that of induced technological innovation.
References


Lemmens, T., Luther, L, and Hoy, M.(2008), “Genetic Information Access, a Legal Perspective: A Duty to Know or a Right Not to Know, and a Duty or Option to Warn?,” Encyclopedia of Life Sciences (ELS), John Wiley & Sons, LTD: Chichester. DOI: 10.1002/9780470015902.a0005188


In this appendix we derive the alterations necessary to accommodate the possibility that marginal utility of income is dependent on the health state (i.e.; for those results for which we haven’t already done so). The general form of the utility function which allows for such state dependence is

\[ EU(s) = (1-\rho)u(y-TC)+\rho[p^{ED}(s)w^{ED}(y-TC)+(1-p^{ED}(s))w^{LD}(y-TC)]-\Phi(s) \] (41)

We assume that \( u(\cdot) > w^{ED}(\cdot) > w^{LD}(\cdot) \) but allow any ordering over the marginal utilities for the component parts (i.e.; \( u'(\cdot), w^{ED'}(\cdot), \) and \( w^{LD'}(\cdot) \)). In the main text, many results are based on the simplification of identical marginal utility of income across all possible health states, with \( w^{ED}(\cdot) = u(\cdot) - \kappa_E \) and \( w^{LD}(\cdot) = u(\cdot) - \kappa_L \), where \( \kappa_L \) and \( \kappa_E \) reflect a health-state dependent physiological (non-financial) disutility cost, \( \kappa_L > \kappa_E \). Thus, for this more specific assumption on utility indices we get

\[ EU(s) = u(y-TC) - \rho[p^{ED}(s)\kappa_E + (1-p^{ED}(s))\kappa_L] - \Phi(s) \] (42)

As noted in the main text, if we define \( \Omega(y-TC) \), which is a weighted utility of income function, as

\[ \Omega(y-TC; \rho) = (1-\rho)u(y-TC)+\rho[p^{ED}(s)w^{ED}(y-TC)+(1-p^{ED}(s))w^{LD}(y-TC)] \] (43)

then many results simply require (roughly speaking) replacing \( u(\cdot) \) with \( \Omega(\cdot) \), \( u'(\cdot) \) with \( \Omega'(\cdot) \), both \( > 0 \); and \( u''(\cdot) \) with \( \Omega''(\cdot) \), both \( \leq 0 \). Since it is natural to assume that the derivatives (up to second) of these two functions have the same signs this means results are easily converted to accommodate the more general form of the utility function. In the case of deriving results in equations (8) and (9), as noted in the main text, one must simply use the more general form for the utility difference between early and late detection, \( w^{ED}(y-TC_e^A) - w^{LD}(y-TC_e^A) \) rather than the more specific one of \( \kappa_L - \kappa_E \). Since both expressions are positive valued there is no qualitative change in the results.

Following through with the appropriate substitutions equation (22), which underlies Proposition 1, becomes:

\[
\frac{dEU_A}{d\varepsilon} = \left[ \eta_L \Omega'_L(y-TC_e^A) + \eta_H \Omega'_H(y-TC_e^A) \right] \cdot \left[ \frac{dT^{CL}_e}{d\rho} - \frac{dT^{CH}_e}{d\rho} \right] \\
+ \left[ p^{ED}(s^H) - p^{ED}(s^L) \right] \cdot [w^{ED}(y-TC_e^A) - w^{LD}(y-TC_e^A)] \\
- \rho_L \frac{dp^{ED}}{ds^L} \frac{ds^L}{d\rho} [w^{ED}(y-TC_e^A) - w^{LD}(y-TC_e^A)] + \frac{d\Phi}{ds^L} \frac{ds^L}{d\rho} \\
+ \rho_H \frac{dp^{ED}}{ds^H} \frac{ds^H}{d\rho} [w^{ED}(y-TC_e^A) - w^{LD}(y-TC_e^A)] - \frac{d\Phi}{ds^H} \frac{ds^H}{d\rho} \] (44)
The first line in the above equation again represents the financial implications of the genetic test through the effects of changes to the cost of health care provision to the two risk types through the equilibrium cost functions. The only difference is that the term \[ \frac{dT_{CL}}{dp^L} - \frac{dT_{CH}}{dp^H} \] is multiplied by the weighted marginal utility of income rather than simply \( u'(y - T_{C_e}) \). Line 2 again reflects the efficiency gain of information from a genetic test in the use of surveillance with \( \kappa_L - \kappa_E \) replaced by \( [w^{ED}(y - T_{C_e}) - w^{LD}(y - T_{C_e})] \).

And again lines 3 and 4 are both zero due to the envelope theorem. Thus, the revised Proposition 1 is isomorphic to the previous one given these new utility index replacements.

Although qualitatively the same results also follow regarding the socially optimal levels of surveillance under genetic testing when using the more general utility function, there is a more substantial change introduced into the second order condition. This follows because the socially optimal rule accounts for the fact that altering the surveillance level affects the per capita cost of delivering health services and this has differential impacts on the marginal utility of income across the various health states, including the states reflecting early versus late detection conditional on the onset of disease.

So, the first order condition, \( FOC^* \), becomes

\[
\Omega'(y - T_C(s^*)) \left( -\frac{\partial T_C}{\partial s}|_{s^*} + \rho \cdot p^{ED'}(s^*) \cdot [w^{ED}(y - T_{C^*}) - w^{LD}(y - T_{C^*})] - \Phi'(s^*) \right) = 0
\]

(45)

For the second order condition, \( SOC^* \), we get

\[
\Omega''(y - T_C(s^*)) \left( -\frac{\partial T_C}{\partial s}|_{s^*} \right)^2 + \Omega'(y - T_C(s^*)) \left[ \rho \cdot p^{ED''}(s^*) (C^{DL} - C^{DE}) - C''(s^*) \right] \\
+ \rho \cdot p^{ED'}(s^*) \cdot [w^{ED}(y - T_{C^*}) - w^{LD}(y - T_{C^*})] - \Phi''(s^*)
\]

(46)

\[+\rho \cdot p^{ED'}(s^*) \cdot [w^{ED}(y - T_{C^*}) - w^{LD}(y - T_{C^*})] \left( -\frac{\partial T_C}{\partial s}|_{s^*} \right) < 0
\]

(47)

The terms in the \( FOC^* \) here are essentially identical to those in the text except for the replacement of the more general utility indices. No change in sign or interpretation of components is different. This is also the case here for the first three terms of the \( SOC^* \). However, there is an additional term that arises because of the possibility that the marginal utility of income for the disease state may differ for early versus late detection. The sign of this final term depends on whether marginal utility of income is higher under early or late detection. This seems likely to be ambiguous in that it depends on the particular disease under investigation. However, this is not of critical importance since the second-order condition will be satisfied if an interior optimum applies whereas corner solutions, which have rather straightforward interpretations, must be considered separately from a
mathematical perspective regardless of which form of the utility function is used.

That part of the comparative statics for the socially optimal rule $s^*(\rho)$ that is derived from the first-order condition (i.e.; the numerator of $\frac{ds^*}{d\rho}$) is also similar to that in the main text, given appropriate substitutions for the more general utility function, except for the fact that a change in $\rho$ that affects the per capita cost of health care has an extra effect through the difference in marginal utilities of income in the two states of early versus late detection of disease. This is shown in the final term of equation (48) below. Whether this final term contributes to the numerator being positive or negative depends both on the sign of $\frac{\partial TC^*}{d\rho}$ and which of the detection states has higher marginal utility of income. Neither of these two terms has unambiguous sign and so the implication of ambiguity of the sign of $\frac{ds^*}{d\rho}$ continues to hold. The second order condition, although of course different, is still summarized accordingly and will be negative for an interior optimum. Therefore, we have

$$\frac{ds^*}{d\rho} = \frac{-\Omega'(y - TC^*) \cdot \left(\frac{\partial TC^*}{d\rho}\right) \cdot \left(-\frac{\partial TC^*}{ds^*}\right)}{-p_{ED}^E(s^*)[\Omega(y - TC^*)(C^{DL} - C^{DE}) + w_{ED}^E(y - TC^*) - w_{LD}^D(y - TC^*)] + \rho p_{ED}^E(s^*)[w_{ED}^E(y - TC^*) - w_{LD}^D(y - TC^*)] \left(-\frac{\partial TC^*}{d\rho}\right) \cdot SOC^*|_{s=s^*}}$$

(48)

Thus, the changes to the results that follow from adoption of the more general utility function do not have significant importance, although it is useful to know this given the relevance per se of the issue concerning how marginal utility of income may vary across different health states. Only minor deviations need to be made to the conditions of Proposition 2. They are, however, sufficiently messy and so we do not reproduce them here.