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 the implications of increased information from genetic tests about predispostion 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 (or prevention) activities to improve the chances of early detection of disease onset. We show that the moral hazard implications inherent in surveillance and prevention decisions that are chosen to be privately rather than socially optimal may be exacerbated by increased information about person-specific predisposition to disease.

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1 Introduction

It is fair to say that genomic science is now in its second phase since current research involves not only 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 and with environmental factors to affect the onset and influence the treatment of diseases. Claims in the scientific literature and the media suggest that advancements made in genetic information will lead to significant improvements in the effectiveness of prevention and treatment of disease. A rough road map of the human genome has been available since 2003 and currently, according to the NIH-sponsored web site genetests.org, there are over 1600 genetic tests used clinically. With the prospect of the so-called $1000 genome close to reality (see Davies, 2010), whole genome sequencing may soon become the norm for developed countries. The information that can be gleaned from an individual's whole genome has the potential to revolutionize the practice of medicine with population wide genome sequencing forming the basis of so-called P4 medicine (i.e., medicine that is Predictive, Preventive, Personalized and Participatory). Although the future of P4 medicine has many proponents, not least of whom is Leroy Hood through his P4 Medicine Institute (p4mi.org), there is some controversy over the pace of its progress.[1]

Once the relationships between specific genes, environment, and diseases are better understood, harnessing this information to create improved health outcomes in a cost effective manner requires a good understanding of how individuals will behave in the context of such individualized informational change. We provide insight into this debate by focusing on how individuals' incentives for use of surveillance (monitoring) technologies, such as colonoscopies or mammograms, change in the presence of risk-type specific information about the likelihood of onset of disease. It has been debated in the literature whether population wide screening for diseases such as colon cancer or breast cancer is cost effective and whether monitoring should be restricted to those at higher risk as identified, for example, by family history. As genetic tests become more wide ranging and less costly, there is the potential of substantial improvements to the targeting of surveillance techniques such as colonoscopies with the potential of improved overall health outcomes in a more cost effective manner. However, we show that the usual moral hazard problems associated with insurance coverage may interact with improved knowledge of individual risks in a way that could blunt the potential for such improvements. Through the use of simple models, we develop a series of results which characterize the possible outcomes that could develop as more genetic information becomes available.

Many genetic tests continue to be expensive and so choosing which tests to make

[1] As noted by Roukos (2008), "although personalized medicine and oncology in clinical practice is still a dream, some isolated first steps have been taken."

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 adoption of more genetic tests (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 ultimately reduce health care costs.[2] It is this aspect or phase of growth in genetic testing 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 develop guidelines to use in determining which genetic tests to offer within the coverage of the public health system. 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.

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).[3] Although our 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 probablistically). The possible benefit of a genetic test in this context 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. [4]

[2] See, for example, Caulfield, et al. (2008) for a critical evaluation of such claims.

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

[4] See Filipova-Neumann and Hoy (2009) for a model describing the implications of genetic testing for differential prevention strategies as well as a discussion of the implications for private health insurance plans.

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. In our context, we presume that individuals do not pay for the financial costs of surveillance or treatment, should onset of disease occur. The result is that individuals may either over-use or under-use medical surveillance or prevention. The moral hazard problems due to insurance are complicated by the introduction of information about differential risk of disease onset. We characterize how genetic testing can lead to changes in the pattern of over- and under-use of surveillance. We find, under a broad range of scenarios, that at least one group (i.e., the average, high or low risk types) will tend to want to over-use surveillance relative to the socially optimal decision. The relative extent to which over-use (or under-use when it occurs) of surveillance reduces social welfare can vary across the groups in counter-intuitive ways. Overall efficiency may fall as improved knowledge about risk type interacts with the standard moral hazard implications of insurance leading to a reduction in social welfare.

In the following section, we introduce a simple model of surveillance, which is also referred to as screening or monitoring. The basic model describes the decision for intensity of monitoring taken by the individual and compares that to the socially optimal decision. In section 3, we present our results regarding the implications of introducing genetic tests and then provide a discussion, conclusion, and suggestions for further research in the final section 4.

2 Model of Medical Surveillance

The role of surveillance is to increase the likelihood of early detection of disease. One key aspect of the model is the relationship between the intensity of surveillance and its effectiveness at early detection and also its financial cost. In the context of screening for colon cancer, one can think of the use of FOBT - fecal occult blood test - as a low level and low cost approach to screening; FSIG - flexible sigmoidoscopy - as an intermediate level and intermediate cost approach; and CSCPY - standard colonoscopy - as a higher intensity and higher cost method of screening. The relative unit costs of these approaches, quoted in U.S. Congress report OTA-BP-H-146 (1995) are \$10, \$80, and \$285 respectively while the {sensitivity, specificity} in regards to detection of cancer are {40%, 90%}, {90%, 98%}, and {90%, 100%}, respectively. FOBT is not very effective at detecting polyps (sensitivity of only 10%) compared to colonoscopy (sensitivity of 90%). One can then think of an intensity of surveillance as a mixture of the various techniques that one can apply with varying frequency starting at a particular age (e.g., FOBT once yearly with CSCPY once every five years starting at age 50). We describe the relationship between the

intensity of surveillance and the probability of early detection of disease by the function $p^{ED}(s)$, with $p^{ED\prime}(s) > 0$ and $p^{ED\prime\prime}(s) < 0$; that is, the probability of early detection of disease increases (at a decreasing rate) with the intensity (and/or frequency) of surveillance as measured by s. The financial cost to the health care system of providing an individual with level of surveillance s is $C(s)$, which we assume is increasing and convex in the level of surveillance; i.e., $C'(s) > 0$, $C''(s) > 0$.[5]

The financial cost of treatment, for those who eventually have onset of disease, depends on whether the disease is detected early or late. In our model we have in mind all future lifetime medical costs conditional on stage of detection of disease. We refer to these as C^{DE} and C^{DL} for cases of early and late detection, respectively. The cost may be higher in either one or other detection stage depending on the disease and depending on the choice of treatment. For simplicity we only model two stages while in many cases - especially cancer - progression of disease is modeled for several stages. Importantly, whether C^{DE} is lower or higher than C^{DL} is not always monotonic in the various possible stages of disease. For example, in U.S. Congress report OTA-BP-H-146 (1995), lifetime cost of treating colon cancer if detected early is estimated at $35,000 and $45,000 if detected late. Brown, et al. (2002), on the other hand, break up possible detection stages into five; in situ (cancer cells restricted to polyps) and cancer stages I through IV.[6] They find that lifetime cancer-related costs are lower if detection occurs in situ or stage I compared to later stages of cancer while they are higher for cancers detected in stage II than any other stage, including stage IV. The reason for this last outcome is that long-term costs for stage II cases involve additional continuing care costs due to longer survival time. In general, if sufficiently early detection leads to a sufficiently higher rate of cure than does later detection, then it is possible for C^{DL} to exceed C^{DE}. It is common for the opposite to hold and so in our analysis we allow for both $C^{DL} > C^{DE}$ (e.g., for early detection being in situ or stage I in our example of colon cancer) and $C^{DL} < C^{DE}$ (e.g., for early detection being stage II and late detection being stage IV).

The overall per capita cost of providing health care to an individual who experiences onset of disease with probability ρ is, therefore, the cost of surveillance plus the expected cost of treatment which depends on whether the disease is detected early or late. Thus, the per capita health care costs are given by

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

It follows that $TC'(s) = \rho \cdot p^{ED\prime}(s)(C^{DE} - C^{DL}) + C'(s) > 0$ if $C^{DL} < C^{DE}$. Increasing the

[5] 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.

[6] Lansdorp-Vogelaar, et al. (2009) suggest that as chemotherapy costs increase for advanced colorectal cancer, screening can become cost effective in terms of overall costs of cancer treatment.

level of surveillance in this case increases per capita health care costs due to both the direct cost of more surveillance and the fact that increasing the probability of early detection leads to an increase in expected treatment costs for those who incur disease. If $C^{DE} < C^{DL}$, an increase in surveillance leads to opposing effects; the increase in surveillance costs may be more than compensated by the savings in lifetime cost of treatment since early detection leads to lower lifetime treatment costs. In this latter case, it is of course possible that $TC(s)$ may not be monontonic (i.e., it could be U-shaped). We allow for all of these possibilities.

We now turn to the issue of individual behaviour. Individuals choose a level of surveillance to maximize their utility. 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 TC to account for the average or per capita cost of the health care system. To highlight the fact that, by assumption, 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 per capita cost when determining the privately optimal use of health care resources. Net income for each individual is $y - \overline{TC}$.

Individual utility or welfare is made up of several components: $u(\cdot)$ in the healthy state (with $u'(\cdot) > 0$, $u''(\cdot) < 0$), $w^{ED}(\cdot) = u(\cdot) - \kappa_E$ in the disease state if it is detected early, and $w^{LD}(\cdot) = u(\cdot) - \kappa_L$ in the disease state if it detected late. The terms κ_L and κ_E reflect a health-state dependent physiological (non-financial) cost, which is subtracted from utility in the case of disease. This amount is assumed to be larger in the case that the disease is detected late rather than early ($\kappa_L > \kappa_E$). With this specification, [7] the marginal utility of income is independent of the health state or early/late detection distinction. Finally, there is a component that reflects a physiological (non-financial) cost of surveillance $\Phi(s)$. This 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).[8] We assume $\Phi(s)$ is increasing and convex in s ($\Phi'(s) > 0, \Phi''(s) > 0$). This gives us the expected utility function:

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

[7] This separation of utility into an income component and a health component is similar to Kiffman (2001). Strohmenger and Wambach (2000) also use a state contingent utility function in an adverse selection model.

[8] In the US Congress report OTA-BP-H-146 they note that the rate of perforation of a colon in colonosopies (CSPCY) is 0.1%. This creates an obvious personal cost and would also add to the financial cost $C(s)$ as they estimate a lifetime cost of treating a perforated colon at $35,000.

If one uses the more general specification of expected utility of:

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

then one can allow for differing marginal utilities of income in the various disease states relative to the healthy state. As long as one assumes that $w^{ED\prime}(\cdot)$, $w^{LD\prime}(\cdot) > 0$, $w^{ED\prime\prime}(\cdot)$, $w^{LD\prime\prime}(\cdot) \leq 0$, and $w^{ED}(\cdot) > w^{LD}(\cdot)$, there are no qualitative differences in our results and so we restrict our attention to the simpler specification in this paper.[9] In Filipova-Neumann and Hoy (2009) we fully work out the relevant conditions for the more general specification of expected utility.

We assume the individual (freely) chooses her level of surveillance. As noted above, in a 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., $\frac{\partial \overline{TC}}{\partial s} = 0$). Homogeneity of preferences implies that each individual's optimal choice of surveillance, $\widehat{s}$, will be the same (conditional on ρ) and so each individual's contribution to health care cost, $\overline{TC}$ will equal $TC(\widehat{s})$ - the equilibrium per capita health cost. Thus, we use the same symbol $\widehat{s}$ to denote both the individual's privately optimal level of surveillance and the equilibrium level of surveillance; which is characterized by the first order condition

$$FOC\ \{\widehat{s}\} : \rho \cdot p^{ED\prime}(\hat{s}) \cdot [\kappa_L - \kappa_E] - \Phi'(\hat{s}) = 0 \quad (4)$$

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, ρ, as well as by the marginal increase in the probability of early detection, $p^{ED\prime}(\widehat{s})$, to the marginal physiological cost of surveillance, $\Phi'(\widehat{s})$.

Our curvature assumptions, along with , $\kappa_L > \kappa_E$, ensure satisfaction of the second order condition:

$$SOC\ \{\widehat{s}\} : \rho \cdot p^{ED\prime\prime}(\hat{s}) \cdot [\kappa_L - \kappa_E] - \Phi''(\hat{s}) < 0, \forall s. \quad (5)$$

To reflect the fact that the privately optimal level of surveillance depends on the probability of disease ρ we write $\widehat{s}(\rho)$.[10]

Applying the implicit function theorem to the FOC yields

$$\frac{d\hat{s}}{d\rho} = -\frac{p^{ED\prime}(s)[\kappa_L - \kappa_E]}{\rho \cdot p^{ED\prime\prime}(s) \cdot [\kappa_L - \kappa_E] - \Phi''(s)} > 0 \quad (6)$$

[9] 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 presumably depends on the particular disease.

[10] One could define lower and upper bounds on ρ which would imply upper and lower bounds on $\widehat{s}$. For the sake of simplicity, we restrict our attention to interior optima for $\widehat{s}$.

and so the implication of a genetic test on the demand for surveillance is clear. Tested positives, for whom the probability of disease will be larger than before the test, will demand more surveillance and tested negatives, for whom 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., $[\kappa_L - \kappa_E]$.

We now compare the privately optimal level of surveillance ($\widehat{s}(\rho)$) with the socially optimal level ($s^*(\rho)$). It is useful first to consider how the value of s that minimizes the per capita financial cost of providing health care, which we will refer to as $\widetilde{s}$, changes as ρ changes. In the case where $C^{DE} > C^{DL}$, any increase in s leads to an increase in $TC(s)$ and so, in this case, $\widetilde{s} = 0$ for any value of ρ (i.e., since $TC(s)$ is monotonically increasing) and it follows that $\frac{d\widetilde{s}}{d\rho} = 0$. For the case where $C^{DL} > C^{DE}$, it follows that as long as the cost of surveillance does not rise "too fast" as s rises (at low levels of s), then $TC(s)$ will be U-shaped or at least decreasing over some range of s.[11] In this case, a similar comparative statics exercise as for $\widehat{s}$ leads to the conclusion that an increase in probability of disease, ρ, leads to an increase in $\widetilde{s}$; i.e.,

$$\frac{d\widetilde{s}}{d\rho} = -\frac{p^{ED\prime}(\widetilde{s})[C^{DE} - C^{DL}]}{\rho \cdot p^{ED\prime\prime}(\widetilde{s}) \cdot [C^{DE} - C^{DL}] + C''(\widetilde{s})} > 0 \tag{7}$$

To characterize the socially optimal choice of surveillance, s^*, requires that we account for the effect of choice of surveillance, s, on the cost of health care. Hence, we replace $\overline{TC}$ in equation (3) with the actual cost as a function of s (i.e., $TC(s)$ given in equation (1)). This leads to the following first-order condition that characterizes s^*.

$$FOC\ \{s^*\} : u'(y - TC(s^*)) \cdot [-\frac{\partial TC}{\partial s}|_{s^*}] + \rho \cdot p^{ED\prime}(s^*) \cdot (\kappa_L - \kappa_E) - \Phi'(s^*) = 0 \tag{8}$$

The second order condition requires:

$$u''(y - TC(s^*)) \left(-\frac{\partial TC}{\partial s}|_{s^*}\right)^2 + u'(y - TC(s^*))[\rho \cdot p^{ED\prime\prime}(s^*)(C^{DL} - C^{DE}) - C''(s^*)] \tag{9}$$

$$+\rho \cdot p^{ED\prime\prime}(s^*) \cdot (\kappa_L - \kappa_E) - \Phi''(s^*) < 0$$

where

$$\frac{\partial TC}{\partial s}|_{s^*} = -\rho \cdot p^{ED\prime}(s^*)(C^{DL} - C^{DE}) + C'(s^*). \tag{10}$$

Rewriting equation (8) as

$$\rho \cdot p^{ED\prime}(s^*) \cdot (\kappa_L - \kappa_E) - \Phi'(s^*) = u'(y - TC(s^*)) \cdot [\frac{\partial TC}{\partial s}|_{s^*}] \tag{11}$$

[11] A sufficient condition for this to be the case is $\rho \cdot p^{ED\prime}(s)(C^{DL} - C^{DE}) > C'(s)$ at $s = 0$.

and comparing to equation (4) makes it clear that s^* is smaller (greater) than $\widehat{s}$ if $\frac{\partial TC}{\partial s}|_{\widehat{s}}$ is greater than (less than) zero. In the case that $\frac{\partial TC}{\partial s} > 0$ we have the usual moral hazard effect that individuals over-utilize a health care service - in this case surveillance - since they do not account for its financial cost. However, recall that if $C^{DL} > C^{DE}$, then it is possible that an increase in surveillance (at level $s = \widehat{s}$) could lower the overall cost of health care since the increase in early detection reduces the cost of treatment for those who incur the disease. This possibility of under-utilization of surveillance is illustrated in Figure 1. Note that the condition $C^{DL} > C^{DE}$ is required for, but does not guarantee, such a result. If the minimum value of a U-shaped $TC(s)$ curve occurs at a value $s = \widetilde{s}$ which is less than $\widehat{s}$, then over-utilitzation occurs as illustrated in Figure 2.

Insert Figure 1 here

Insert Figure 2 here

We collect these results in the following proposition.

Proposition 1. *The socially optimal use of surveillance (s^*) is greater or less than the privately optimal use of surveillance ($\widehat{s}$) according to the following scenarios.*

1. ***Over-utilitzation of surveillance****: If $\frac{dTC(s)}{ds} > 0$ at $s = \widehat{s}$, then $s^* < \widehat{s}$. A sufficient condition for this scenario is $C^{DE} > C^{DL}$. More generally, this relationship will hold whenever $\widetilde{s} < \widehat{s}$ (i.e., the cost-minimizing level of surveillance is less than the privately optimal level).*

2. ***Under-utilization of surveillance****: If $TC(s)$ is U-shaped, then $s^* > \widehat{s}$ whenever $\widetilde{s} > \widehat{s}$ (i.e., the cost-minimizing level of surveillance is greater than the privately optimal level). A necessary condition for this scenario is $C^{DE} < C^{DL}$.*

A genetic test leads to the revelation for some individuals that the probability of disease is greater than previously thought, while others realize it is smaller than they previously thought. It is, therefore, of interest to understand how each of the critical values $\widehat{s}$, $\widetilde{s}$, and s^* vary as the probability of disease (ρ) varies. We have already established that (i) $\frac{d\widehat{s}}{d\rho} > 0$, (ii) $\frac{d\widetilde{s}}{d\rho} = 0$ when $C^{DE} > C^{DL}$ (since $\widetilde{s} = 0$ for any value of ρ in this case), and (iii) $\frac{d\widetilde{s}}{d\rho} > 0$ when $TC(s)$ is U-shaped. In this last scenario, which requires that $C^{DL} > C^{DE}$ (as a necessary condition), there is the possibility of a counter-intuitive result that the health care costs for those who experience an increase in the perceived (and actual for that matter) probability of disease will impose lower costs on the health care system than those who discover they have a lower probability of disease than previously thought. The intuition is simple. Those who realize they have a higher probability of disease increase their surveillance intensity and so increase their chances of having the disease detected early. If the impact of this increase in surveillance is sufficiently great, in conjunction

with $C^{DL} - C^{DE}$ being sufficiently large, the overall health care cost for the higher risk group may be lower than it was before they realized their higher risk status.[12] This result demonstrates that the impact of genetic tests may not be what one expects.

The relationship between the socially efficient level of surveillance and the probability of disease is more complicated to understand. From the first-order condition for s^*, we see 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.[13] It is helpful to rewrite this first-order condition as:

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

FOC^*b indicates that the socially optimal level of surveillance equates the marginal nonfinancial health benefits plus financial benefits of surveillance to the marginal nonfinancial plus financial costs of surveillance. Consider first the case of risk neutrality (i.e., $u'(y - TC)$ a constant). If $C^{DL} - C^{DE} > 0$, it follows that an increase in ρ leads to an increase in both the marginal financial and nonfinancial benefits of surveillance (LHS of FOC^*b), while the marginal cost of surveillance remains the same (RHS of FOC^*b). It follows that an increase in ρ will lead to an increase in s^*. If $C^{DL} < C^{DE}$, then an increase in ρ would again increase the marginal nonfinancial benefit of surveillance but would decrease the marginal financial benefit. On balance, for an interior optimum, the first effect must dominate the second and so there would be a net increase in the overall marginal benefit of surveillance as a result of an increase in ρ.[14] An increase in ρ again leads to no change in the marginal cost of surveillance (RHS of FOC^*b) and so again, it follows that an increase in ρ implies an increase in s^*. However, if $u''(y - TC) < 0$, an increase in ρ leads to an increase in $TC(s^*)$ and so an increase in $u'(y - TC)$. This implies that in this latter case, the RHS of FOC^*b rises and the LHS falls (since a higher value of $u'(y - TC(s^*))$ is multiplied by $C^{DL} - C^{DE}$, which is negative).[15] Therefore, in this scenario, an increase in ρ may not lead to an increase in s^* due to the fact that the increased financial cost of surveillance caused by the increase in ρ has greater impact due to the associated increase in marginal utility of income. We have the following proposition and corollary:

[12] Of course the opposite could hold for those who discover they are a lower risk than previously thought.

[13] The "financial part" is multiplied by the scale or conversion factor $u'(y - TC(s^*))$ to reflect the marginal utility value of dollar costs. Under risk neutrality, this conversion factor is unchanged by any change in ρ and hence any change in the level of TC.

[14] For an interior optimum it must be that $(\kappa_L - \kappa_E) > u'(y - TC(s^*))(C^{DE} - C^{DL})$ when $C^{DE} > C^{DL}$ (since RHS of FOC^*b is positive).

[15] In the former case, $C^{DL} - C^{DE} > 0$, the increase in $u'(y - TC)$ that is associated with an increse in ρ implies an even larger increase in the marginal benefit of surveillance and so this reinforces the effect that s^* increases as a result of an increase in ρ.

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.,* $u''(\bullet) < 0$*:*

1. $TC'(s^*) = C'(s^*) - \rho \cdot p^{ED\prime}(s^*)(C^{DL} - C^{DE}) \leq 0$ *is a sufficient condition for* $\frac{ds^*}{d\rho} > 0$.

2. If $TC'(s^*) = C'(s^*) - \rho \cdot p^{ED\prime}(s^*)(C^{DL} - C^{DE}) > 0$, *then*
$\frac{ds^*}{d\rho} \gtreqless 0 \Leftrightarrow TC'(s^*) \lesseqgtr \alpha$,
where

$$\alpha = \frac{p^{ED\prime}(s^*)[u'(y - TC^*)(C^{DL} - C^{DE}) + (\kappa_L - \kappa_E)]}{-u''(y - TC^*)[p^{ED}(s^*)C^{DE} + (1 - p^{ED}(s^*))C^{DL}]} \tag{12}$$

$$= \frac{p^{ED\prime}(s^*)[(C^{DL} - C^{DE}) + \frac{(\kappa_L - \kappa_E)}{u'(y - TC^*)}]}{A(y - TC^*)[p^{ED}(s^*)C^{DE} + (1 - p^{ED}(s^*))C^{DL}]} > 0 \tag{13}$$

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

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

3 Welfare Implications of Genetic Testing

We now develop the welfare implications of introducing information gained from a genetic test. 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 ρ^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 ρ^H (ρ^L), where $\rho^L < \rho^0 < \rho^H$. The proportion of individuals who test negative is denoted by η_L while the proportion who test positive is $\eta_H = (1 - \eta_L)$. 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 false negatives as described below. Since the population average probability of disease is ρ^0, we have

$$\rho^0 = \eta_L \cdot \underbrace{(\rho^0 - \frac{\varepsilon}{\eta_L})}_{\rho^L} + \eta_H \cdot \underbrace{(\rho^0 + \frac{\varepsilon}{\eta_H})}_{\rho^H}, \tag{14}$$

with $\varepsilon > 0$ describing the 'degree of accuracy' of the information. An increase in ε implies both greater sensitivity and specificity (i.e., lower rates of false positives and false negatives from the GT). 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 (ε). A more

precise test implies the probabilities of disease for tested negatives and tested positives become closer to the true probabilities of disease for low and high risk types (denote them by ρ_T^L and ρ_T^H), which need not even be known. Note that $\rho^0 = \eta_L\rho^L + \eta_H\rho^H$ holds irrespective of the precision of the test.[16]

From our result that $\frac{d\widehat{s}(\rho)}{d\rho} > 0$, the implication of a genetic test on the demand for surveillance is clear. Tested positives, for whom the probability of disease is perceived to be higher than before the test, will demand more surveillance and tested negatives, for whom the reverse holds, will demand less surveillance. It may seem natural to expect genetic tests to lead to better targeting of surveillance and hence higher social welfare. However, since individuals do not internalize the financial cost of either medical surveillance or treatment of disease (should it occur), it is not clear that the resulting changes in surveillance will lead to a welfare improvement since the degree to which each risk types alters her surveillance level may not be in accordance with what is socially efficient even if the "direction of change" is consistent. To determine conditions under which a GT leads to a higher or lower level of social welfare, we develop the value function $v(\rho) = EU(\hat{s}(\rho))$ which determines the individual's utility conditional on the probability of disease, ρ, and hence her privately optimal choice of surveillance, $\hat{s}(\rho)$. Although the individual ignores the implications of the financial cost of her health care decisions in making her choice of surveillance, the value function $v(\rho)$ does take into account any such changes. Since a GT is essentially a mean preserving spread in probabilities, the curvature of $v(\rho)$ will determine whether a GT leads to an increase in welfare (if $v(\rho)$ is convex in ρ) or a decrease in welfare (if $v(\rho)$ is concave in ρ). We show below that the outcome depends (in part) on the shape of the equilibrium cost function $TC_e(\rho)$, which in turn is determined by the function $\hat{s}(\rho)$.

Although the individual (correctly) does not perceive that his choice of s affects his per capita cost of health care, the fact that everyone adjusts s to any change in ρ means this cost is affected nonetheless. We write $TC_e(\rho)$ to reflect the equilibrium per capita cost of health care given probability of disease ρ, where

$$TC_e(\rho) = \rho[p^{ED}(\widehat{s}(\rho))C^{DE} + (1 - p^{ED}(\widehat{s}(\rho)))C^{DL}] + C(\widehat{s}(\rho)) \tag{15}$$

which implies

$$\begin{aligned}\frac{dTC_e}{d\rho} &= p^{ED}(\widehat{s}(\rho))C^{DE} + (1 - p^{ED}(\widehat{s}(\rho)))C^{DL} \\ &\quad -\rho[p^{ED\prime}(\widehat{s}(\rho))[C^{DL} - C^{DE}]]\frac{d\widehat{s}}{d\rho} + \frac{dC(\widehat{s}(\rho))}{d\widehat{s}} \cdot \frac{d\widehat{s}}{d\rho}\end{aligned} \tag{16}$$

[16] ε has a maximum value, indicating perfect information about true risk type. This value satisfies both $\overline{\varepsilon} = \eta_L(\rho^0 - \rho_L^T)$ and $\overline{\varepsilon} = \eta_H(\rho_H^T - \rho^0)$ since $\rho^0 = \eta_L\rho_L^T + \eta_H\rho_H^T$ and $\eta_L + \eta_H = 1$. This formulation allows us to characterize the amount of information using one, rather than two, parameters.

Note that our use of $\widehat{s}$ to denote both the individual's optimal choice of surveillance as well as the equilibrium level is something of an abuse of notation. But this is justified by the fact that all individuals with the same ρ choose the same level of surveillance. We sometimes write the equilibrium cost function as $TC_e(\widehat{s}(\rho), \rho)$ to reflect indirect and direct effects, respectively, of a change in ρ on the per capita cost of providing health care. Thus, the first line of equation (16) reflects the (direct effect of) added cost of treating patients due to the higher incidence of disease (i.e., higher ρ but holding $\widehat{s}$ fixed). The two terms in the second line reflect the impact on health care costs due to the effect of a change in ρ on the equilibrium (privately optimal) level of surveillance, $\widehat{s}$. The first of these two terms is positive (negative) if $C^{DE} > (<)\ C^{DL}$. The second of these terms is positive, representing the (direct) cost of providing a higher level of surveillance. However, what is important in terms of the welfare implications of a GT is the sign of the second, rather than the first, derivative of this function.

If $TC_e(\rho)$ is linear in ρ, then a mean preserving spread in ρ will leave average health care costs unchanged. Since in a public health care system each individual pays the average per capita cost, the introduction of the GT has no effect on health care costs in this scenario. However, since a change in ρ affects the level of surveillance for individuals who discover themselves to be an either higher or lower risk type than previously thought, there is no reason to expect $TC_e(\rho)$ to be linear. The expression for the second derivative of $TC_e(\rho)$ is messy, containing terms that are both positive and negative. Even making an assumption about the relative size of C^{DE} versus C^{DL} will not lead to a definite sign for $TC_e''(\rho)$. Overall it seems plausible that in some cases (i.e., for some diseases) one can expect that GTs will lead to an increase in overall health care costs due to the reaction of individuals in their choices of surveillance intensity while in other cases overall health care costs will fall.

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 $v(\rho^H) = EU(\hat{s}(\rho^H))$. Both ρ^L and ρ^H depend on ε (the precision of the test). Thus, 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 ε is $EU^A(\varepsilon) = \eta_L v(\rho^L) + \eta_H v(\rho^H)$.

Consider first the private value of a GT. 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.[17] We know that a (fair) lottery over probabilities

[17] Of course, if taking a GT has an effect on one's market opportunities such as for life insurance (see Hoy, 2006) or employement, one may be better off to not take the GT as long as one can demonstrate

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 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 (surveillance) conditional on either outcome of the 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.[18] However, in determining the *social* value of such a costless test, there is an external effect to be considered in that changes in surveillance intensity by members of the insurance pool who take a GT affects the overall cost (to everyone) of health care. Since any single individual cannot avoid these implications, it is privately optimal to accept a GT and change her level of surveillance accordingly.[19]

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 $\varepsilon > 0$ and see how an increase in ε affects expected utility and costs. Let $\overline{TC}^A$ be the average (financial) cost of providing health care services to the insurance pool under the public health insurance system. If individuals obtain GTs, then this becomes

$$\overline{TC}^A = \eta_L \overline{TC}^L + \eta_H \overline{TC}^H$$

where $\overline{TC}^t$ is the per capita cost of health care created by a type t individual, $t = L, H$. Since 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, the expected value function then becomes $EU^A(\varepsilon)$ below, where TC_e^L and TC_e^H represent the equilibrium average (or actual) cost generated by a person of type $t = L, H$, respectively.

$$\begin{aligned} EU^A(\varepsilon) &= \eta_L\{u(y - TC_e^A) - \rho^L[(1 - p^{ED}(s^L))\kappa_L + p^{ED}(s^L)\kappa_E] - \Phi(s^L)\} \\ &\quad + \eta_H\{u(y - TC_e^A) - \rho^H[(1 - p^{ED}(s^H))\kappa_L + p^{ED}(s^H)\kappa_E] - \Phi(s^H)\} \end{aligned} \quad (17)$$

For convenience, we drop the $\widehat{\ }$ notation on the variables s^L and s^H, giving:

$$\frac{dEU^A}{d\varepsilon} = \eta_L \frac{dEU^L}{d\varepsilon} + \eta_H \frac{dEU^H}{d\varepsilon} \quad (18)$$

one's decision.

[18] See Hirshleifer and Riley (1992, pp. 170-185) for details of this argument.

[19] From equation (11) we can derive $v''(\rho)$ which includes the term $p^{ED\prime}(\widehat{s})[\kappa_L - \kappa_E]\frac{d\widehat{s}}{d\rho} > 0$, which reflects the private value of the test. However there are also terms involving derivatives of $TC^e(\rho)$, which cannot be signed. Hence, the function $v(\rho)$ isn't necessarily convex.

with

$$\begin{aligned}\frac{dEU^t}{d\varepsilon} &= u'(y-TC_e^A)\cdot\left(-\frac{dTC_e^A}{d\varepsilon}\right)-\frac{d\rho^t}{d\varepsilon}[p^{ED}(s^t)\kappa_E+(1-p^{ED}(s^t))\kappa_L] \\ &\quad -\rho^t\left[-\frac{dp^{ED}}{ds^t}\frac{ds^t}{d\rho^t}\frac{d\rho^t}{d\varepsilon}\cdot(\kappa_L-\kappa_E)\right]-\frac{d\Phi(s^t)}{ds^t}\frac{ds^t}{d\rho^t}\frac{d\rho^t}{d\varepsilon} \qquad (19)\end{aligned}$$

for $t=L,H$. Upon substituting $\frac{d\rho^L}{d\epsilon}=-\frac{1}{\eta_L}$, $\frac{d\rho^H}{d\varepsilon}=\frac{1}{\eta_H}$, we obtain

$$\frac{dTC_e^A}{d\varepsilon}=\eta_L\cdot\frac{dTC_e^L}{d\rho^L}\cdot\frac{d\rho^L}{d\varepsilon}+\eta_H\cdot\frac{dTC_e^H}{d\rho^H}\cdot\frac{d\rho^H}{d\varepsilon} \qquad (20)$$

and this gives:

$$\begin{aligned}\frac{dEU^A}{d\varepsilon} &= u'(y-TC_e^A)\cdot\left[\frac{dTC_e^L}{d\rho^L}-\frac{dTC_e^H}{d\rho^H}\right] \\ &\quad +[p^{ED}(s^H)-p^{ED}(s^L)]\cdot[\kappa_L-\kappa_E] \\ &\quad -\rho^L\frac{dp^{ED}}{ds^L}\frac{ds^L}{d\rho^L}(\kappa_L-\kappa_E)+\frac{d\Phi}{ds^L}\frac{ds^L}{d\rho^L} \\ &\quad +\rho^H\frac{dp^{ED}}{ds^H}\frac{ds^H}{d\rho^H}(\kappa_L-\kappa_E)-\frac{d\Phi}{ds^H}\frac{ds^H}{d\rho^H} \qquad (21)\end{aligned}$$

The first line in the above equation represents the financial implications of the genetic test. People assigned to different risk classes choose different levels of surveillance and face different probabilities of disease and so financial implications are measured by the term $\left[\frac{dTC_e^L}{d\rho^L}-\frac{dTC_e^H}{d\rho^H}\right]$.[20] If the cost function is linear in ρ this term disappears since this would imply that the *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 $\frac{dTC^L}{d\rho^L}<\frac{dTC^H}{d\rho^H}$ (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.

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 and so adopt higher intensity of surveillance). This efficiency effect is higher the greater is 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

[20] Recall that it is possible even that $\frac{dTC^e}{d\rho}<0$. Let us assume here that it is positive, although it isn't important to do so.

theorem applies and each of these terms is zero. To see that is the case, rearrange lines 3 and 4 as:

$$-\frac{d\widehat{s}^L}{d\rho^L}\cdot\left[\rho^L\frac{dp^{ED}}{d\widehat{s}^L}(\kappa_L-\kappa_E)-\frac{d\Phi}{d\widehat{s}^L}\right] \tag{22}$$

$$\frac{d\widehat{s}^H}{d\rho^H}\cdot\left[\rho^H\frac{dp^{ED}}{d\widehat{s}^H}(\kappa_L-\kappa_E)-\frac{d\Phi}{d\widehat{s}^H}\right] \tag{23}$$

Thus, we have the following proposition.

Proposition 3. *Under public insurance (no differential pricing and full coverage of costs), 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 $\frac{d^2TC_e}{d\rho^2}\leq 0$ *individuals' expected welfare will (unambiguously) increase as a result of introducing GTs.*

2. If $\frac{d^2TC_e}{d\rho^2}>0$ *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*

$$[p^{ED}(s^H)-p^{ED}(s^L)]\cdot[\kappa_L-\kappa_E]>(<)\ -u'(y-TC_e^A)\cdot\left[\frac{dTC_e^L}{d\rho^L}-\frac{dTC_e^H}{d\rho^H}\right] \tag{24}$$

4 Conclusions and Discussion

We have provided a model that characterizes moral hazard implications resulting from an individual's choice of surveillance/monitoring for disease (e.g., use of colonoscopies, FOBT, and other techniques for detecting colon cancer). We have shown that, although a higher predisposition to a disease will always lead to an increase in the privately optimal level of surveillance chosen by individuals, this is not necessarily the case for the socially optimal level. If the continuing lifetime costs of treatment for those incurring disease are higher under early detection than under late detection, which is a common phenomenon, then individuals will over-utilitze surveillance relative to what would be socially efficient. However, in some cases early detection of disease leads to a cure or preemption of disease (e.g., detection and removal of polyps through colonoscopy) and this is not as costly compared to the lifetime costs of treating the disease conditional on late detection. An example would be detecting colon cancer in situ or at stage 1 rather than at a later stage. In such a case, under-utilitzation of surveillance may occur. Given all of these possibilities, it is clear that, in the context of genetic testing, careful attention must be paid to the problem of which individuals should be encouraged to either decrease or increase their level of surveillance. This relationship will depend on the specifics of the disease, including; (i)

perceived (personal) health benefits of early versus late treatment of disease; (ii) relative financial costs of treatment under early versus late detection; (iii) personal (including psychological) and financial costs of the monitoring technologies available; (iv) relative effectiveness of these technologies at detecting disease early and how this effectiveness varies with intensity of surveillance across risk types.[21]

The main contribution of our paper is to develop a method to analyze the welfare implications of a genetic test (or other diagnostic test) that creates improved information about person-specific risk-type (i.e., predisposition to disease onset). We show how such information interacts with the moral hazard phenomenon regarding choice of surveillance intensity in a way that may lead to a reduction in social welfare even for a costless genetic test. Whether welfare will be enhanced or reduced depends crucially on the curvature of the equilibrium cost function. This function describes how the per capita overall cost of health care provision depends on the relationship between a person's perceived probability of onset of disease and her privately optimal choice of surveillance (in conjunction with other health care parameters). If this function is concave in the probability of disease onset (ρ), then the introduction of a genetic test will be welfare improving. However, if this function is strictly convex then welfare *may* fall. The intuition underlying this result is that a genetic test is essentially a mean preserving spread in the population wide perceived probability of onset of disease and so, if the cost function is convex in this probability, then the introduction of improved information leads to an increase in the overall cost of providing health care. Although the improved information leads to individuals choosing surveillance levels that reflect improved targeting from the perspective of their personal health benefits, individuals ignore the financial cost implications of both their decision to obtain a genetic test and their surveillance choices. Therefore, if health care delivery costs increase sufficiently, these advantages to improved information may be insufficient to result in a welfare improvement.

Our model for assessing the benefits of genetic testing in the context of "improved" surveillance decisions can be adapted to a model for prevention.[22] Instead of individuals choosing a level of intensity of surveillance, which affects the probability that a disease which has already been incurred will be detected early, consider a scenario in which individuals choose a level of preventive care. The higher the level of preventive care, the greater

[21] See Filipova-Neumann and Hoy (2009) for greater detail on these issues, including a characterization of when higher risk types are likely to over-utilitze surviellance while lower risk types under-utilitze, and vice versa.

[22] Such a model is essentially isomorphic to the one presented in this paper. One simply replaces the risk-type specific probability of disease and the function describing probability of early detction based on surveillance level ($p^{ED}(s)$) with a function that desribes how the *probability of disease* onset depends on risk type and level of prevention. For details see Filipova-Neumann and Hoy (2009).

the likelihood that there will be no onset of disease. There are a multitude of diseases for which genetic tests can lead to potential improvements in choice of level of prevention (i.e., so-called multifactorial genetic diseases). For example, a woman who discovers she has the BRCA1 gene may choose to have prophylactic surgery that can significantly reduce the probability of later onset of breast or ovarian cancer. There is a similar moral hazard phenomenon associated with preventive activity and so over-utilization and under-utilization are possibilities. Similar analyses follow in that the way decisions of individuals regarding privately optimal levels of preventive care change as a result of information from genetic tests and this may lead to either an increase or decrease in social welfare.

Our model could also be adapted to scenarios in which relevant health care strategies involve both surveillance and prevention activities. Suppose an individual is diagnosed with (relatively) early stage breast cancer and this occurs at an early age. A genetic test may reveal whether this person has one of the so-called breast cancer genes (BRCA1/2). If the test is positive, then the individual may choose an aggressive treatment for the disease (e.g., double mastectomy) while if the test is negative the individual may choose a less aggressive treatment (lumpectomy). The result of the genetic test may also affect the intensity of surveillance going forward in the individuals life in order to detect early any reoccurrence of the disease. The continuing lifetime costs depend on both the person's genetic type and the treatment option where (current) treatment behaves also as a preventive measure against reoccurrence.

A major challenge in organizing health care is to decide which programs of surveillance and prevention are worthwhile. Cohen, et al. (2008) point out that there is often excessive optimism about the ability of preventive health measures and technologies (including screening programs) to reduce health care costs. This raises questions as to which measures and technologies are reasonable investments. Our paper offers a methodology to aid in determining how specific preventive medicine programs may be improved through the use of genetic tests in conjunction with targeted screening directives. As they note (Cohen, et al., 2008, p. 661), for example, "the efficiency of cancer screening can depend heavily on both the frequency of the screening and the level of cancer risk in the screened population." Suppose, in the context of our model, that the existing level of screening that occurs for an entire population with probability of disease ρ^0 (i.e., without genetic testing) represents over-utilitization. A genetic test will identify those at lower risk (ρ^L), who are predicted by the model to reduce surveillance, which in this scenario is likely to improve welfare. Those who are discovered to be high risk (ρ^H) will increase their intensity of surveillance and since targeting surveillance to more risk prone individuals is often more efficient, this *may* lead to an improvement in welfare for this group as well. However, as our model points out, whether or not these changes will represent an overall improvement in welfare

is not straightforward even though the "direction of change" seems to be appropriate for both risk groups. Our model provides a starting point for conceptualizing a cost-benefit approach that includes behavioural responses to assess the value of the introduction of any particular genetic test.

We also offer directions for future research. Our model presumes individuals are ultra-rational in that they are expected utility maximizers who understand well the various probabilities in the model (i.e., probability of onset of disease and probability of early detection at various levels of intensity of surveillance). Applied studies need to consider how individuals interpret risks such as genetic predispositions to disease (e.g., see O'Doherty and Suthers, 2007) and how, conditional on genetic risk type, they understand how different intensities of prevention and surveillance affect the probability of onset of disease or early detection. Non-EU or behavioural models of decision making also deserve attention in the context we have described in this paper. It should be recognized that counselling services from health care professionals may help individuals understand the outcomes of tests and strategies but that in itself does not correct for the wedge between privately and socially optimal decisions. 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. This can be a perfectly rational individual decision. Our model demonstrates that, in such a case, the health care system should, in principle, respond to such pressures by denying the full demand for increased surveillance. 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 - 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. Methods for dealing with these problems are beyond the scope of this paper but certainly worthy of study.

We have ignored many potential sources of 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 genetic test (e.g., see Hoy and Witt (2007) for such an approach in a different context). A more problematic type of heterogeneity is the range of different personal preferences one would expect over the physiological benefits and costs of surveillance or prevention. For example, some people may simply have a higher disutility from certain surveillance procedures. This makes it difficult to determine, for example,

which individuals should receive higher surveillance levels and which lower surveillance levels in order to obtain a social optimum. Such information about preferences is intrinsically private and so this represents a serious challenge to so-called one-size-fits-all health insurance/provision. But this feature, admittedly, is not unique to our problem.

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 directly provide some guidance for such private schemes. Private insurance regimes, however, are often more open to user fees and co-payments. These features 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 co-payments and user fees would fall differentially across (genetic) risk types, use of such instruments would create a phenomenon akin to premium risk. It is also worth integrating models that highlight the risk premium problem or adverse selection costs associated with genetic testing in related markets such as life insurance. These models demonstrate such information may be welfare reducing (see, for example, Hoy and Polborn (2000), Hoy and Ruse (2005) and Hoy (2006)) and such effects can be compared to the potential welfare improvements associated with improved health care decisions.

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Appendix (for use of referees only)

Note regarding Proposition 2: The first part follows directly from the first order condition for s^* and the discussion in the paragraph preceding the statement of the proposition.

For part 2, note that total differentiation of the first-order condition for s^* gives:

$$\frac{ds^*}{d\rho} = -\frac{\left\{\begin{array}{c} -u''(y-TC^*)\cdot\left(\frac{\partial TC^*}{\partial \rho}|_{s^*}\right)\cdot\left(-\frac{\partial TC}{\partial s}|_{s^*}\right) \\ +\,p^{ED\prime}(s^*)[u'(y-TC^*)(C^{DL}-C^{DE})+(\kappa_L-\kappa_E)] \end{array}\right\}}{SOC^*|_{s=s^*}} \tag{25}$$

where

$$\frac{\partial TC^*}{\partial \rho}|_{s^*} = p^{ED}(s^*)C^{DE} + (1-p^{ED}(s^*))C^{DL} > 0, \tag{26}$$

$$-\frac{\partial TC}{\partial s}|_{s^*} = \rho\cdot p^{ED\prime}(s^*)(C^{DL}-C^{DE}) - C'(s^*), \tag{27}$$

and $SOC^*|_{s=s^*}$is the expression in equation (9). Part 2 of the proposition then follows from the above expression upon making the substitution noted for α.

Note regarding shape of cost function: The expression for the second derivative of $TC_e(\rho)$ is given below.

$$\frac{d^2TC_e}{d\rho^2} = p^{ED\prime}(\widehat{s}(\rho))\frac{d\widehat{s}}{d\rho}[C^{DE}-C^{DL}] + \rho[p^{ED\prime\prime}(\widehat{s}(\rho))[C^{DE}-C^{DL}]]\left[\frac{d\widehat{s}}{d\rho}\right]^2 \tag{28}$$

$$+[p^{ED\prime}(\widehat{s}(\rho))[C^{DE}-C^{DL}]]\frac{d\widehat{s}}{d\rho} + \rho[p^{ED\prime}(\widehat{s}(\rho))[C^{DE}-C^{DL}]]\frac{d^2\widehat{s}}{d\rho^2} \tag{29}$$

$$+\frac{d^2C(\widehat{s}(\rho))}{d\widehat{s}^2}\cdot\left[\frac{d\widehat{s}}{d\rho}\right]^2 + \frac{dC(\widehat{s}(\rho))}{d\widehat{s}}\cdot\frac{d^2\widehat{s}}{d\rho^2} \tag{30}$$

Of the six terms that make up the expression for $\frac{d^2TC_e}{d\rho^2}$, only the fifth term can be signed (as positive) given our assumptions. If $C^{DE} > C^{DL}$, then three more terms can be signed (the first and third being positive, the second being negative). The other terms (fourth and sixth) depend on the sign of $\frac{d^2\widehat{s}}{d\rho^2}$ and there is no clear intuition why the function $\widehat{s}(\rho)$ should be expected to be concave or convex.

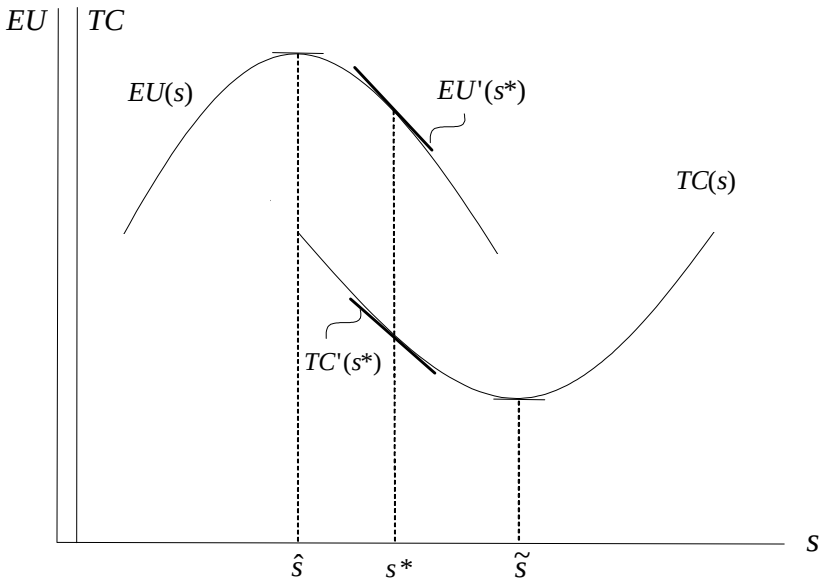

Figure 1: Underutilization of Surveillance

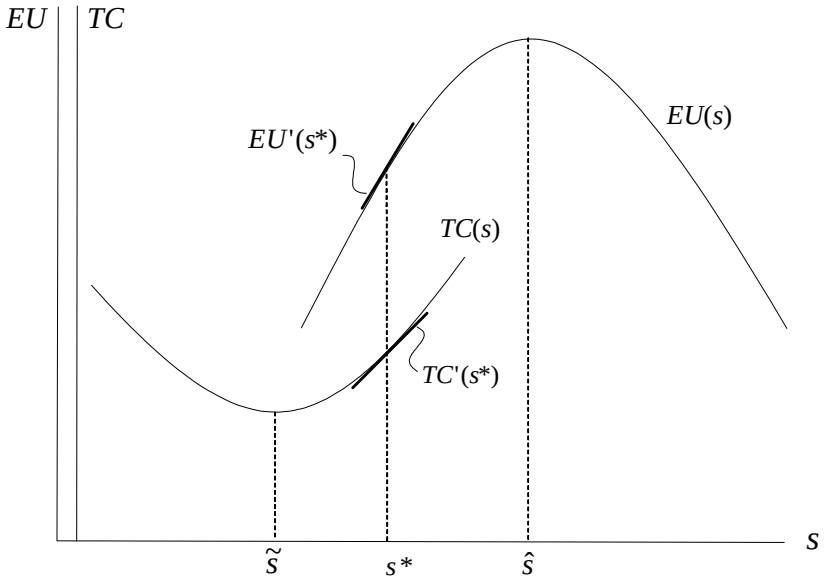

Figure 2: Overutilization of Surveillance