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Genetic testing with primary prevention and moral hazard[1]

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Abstract

We develop a model where a genetic test reveals whether an individual has a low or high probability of developing a disease. A costly prevention effort allows high-risk agents to decrease this probability. Agents are not obliged to take the test, but must disclose its results to insurers, and taking the test is associated to a discrimination risk.

We study the individual decisions to take the test and to undertake the prevention effort as a function of the effort cost and of its efficiency. If effort is observable by insurers, agents undertake the test only if the effort cost is neither too large nor too low. If the effort cost is not observable by insurers, moral hazard increases the value of the test if the effort cost is low. We offer several policy recommendations, from the optimal breadth of the tests to policies to do away with the discrimination risk.

JEL Codes: D82, I18.

Keywords: discrimination risk, informational value of test, personalized medecine.

1 Introduction

"Increasing the focus on prevention in our communities will help improve America's health, quality of life and prosperity. For example, seven out of 10 deaths among Americans each year are from chronic diseases (such as cancer and heart disease), and almost one out of every two adults has at least one chronic illness, many of which are preventable". This statement by the US Centers for Disease Control and Prevention (CDC) reveals the crucial and increasing role played by prevention for health care systems worldwide.

An important characteristic of prevention actions is that, while many individuals may undertake prevention and thus incur its costs, the health and financial benefits generally only accrue to the individuals who are at risk of developing the disease or injury. There is evidence that, for many important health risks, individuals differ significantly in how efficient prevention is for them. For instance, the same CDC write that "Several genetic disorders are associated with increased risk of premature heart attacks. A relatively common disorder is familial hypercholesterolemia, which causes high levels of "bad" cholesterol (low density lipoprotein, or LDL cholesterol) beginning at birth. One out of 500 people in the United States may inherit this condition. Early detection of this disorder can help reduce the burden of heart disease in the person with hypercholesterolemia as well as in their family members."

This in turn means that sizeable welfare gains would be reaped if it were possible to identify individual characteristics associated to a larger efficiency of prevention. One way to uncover those characteristics is through genetic tests. The main thesis of Collins (2010) (as well as other books, such as Davies [2010]) is precisely that genetic testing is ever more reliable and allows not only to be better informed about individual health risks, but also to use this information to individually tailor prevention. Collins insists that improvement in the assessment of the risk of occurrence of a disease very often allows the individual to take preventive action in order to prevent this disease from occurring. "There are many diseases such as cystic fibrosis or PKU, for which a particular biochemical or DNA test result makes a very strong prediction about the likelihood of illness, and interventions are available" (page 802). There is actually a whole range of such prevention activities: "institution of drug therapies; (...) special diets; (...) surgery or other options" (page 815). As Collins writes quoting a patient "I know early in my life something I am substantially predisposed to. I now have the opportunity to adjust my life to reduce those odds" (page 1070).

The objective of our paper is to try and assess the impact of offering a (genetic) test to individuals on both the private health insurance market and on the welfare of individuals. More precisely, we aim at understanding under what circumstances such a test would be voluntarily taken by individuals, what the consequences of the availability of testing would be on the extent to which individuals undertake prevention efforts, and

whether such a test would increase individual welfare. The simple model we develop to answer these questions has the main ingredients of Collins's story. Agents differ in their risk to develop a disease, with two types (L and H) corresponding to two levels of risk in the general population: a fraction λ has the high probability p_H of developing the disease while the remainder has the low probability $p_L < p_H$. People are born uninformed about their individual risk level, but can undertake a (genetic or otherwise[1]) test in order to assess (without any error) whether they are of a low or high type (in the former case, we talk about a negative test, *versus* a positive test in the latter case). After the testing phase, agents decide whether to undertake a prevention effort, at a cost, in order to decrease the probability of occurrence of the disease. That is, we model primary prevention (as opposed to secondary prevention, which does not affect the probability that the disease occurs, but decreases its severity).[2] Collins (2010) provides many examples of both primary and secondary prevention ("discoveries are providing powerful new insights into both treatment and prevention", page 1084).

We assume that prevention is efficient at reducing the risk of illness only if the individual's test is positive (*i.e.*, if he is of a high type). One can give several examples of tests/illnesses with such features, ranging from prophylactic mastectomy in case of mutated BRCA1 gene, to "intense medical surveillance and removal of polyps (that) can be lifesaving for those at high risk" of colon cancer (page 1853). One reason why prevention effort may be efficient only if an individual has a high type is that "it is a combination of the genes that you have inherited and the environment that you live in that determines the outcome. Hence the common saying, "genes load the gun, and environment pulls the trigger" (page 1098). For instance, "Participants in the lifestyle intervention group reduced their risk of developing full-blown diabetes by 58 percent." (page 1313). For macular degeneration, "it became clear that almost 80 percent of the risk could be inferred from a combination of (...) two genetic risk factors, combined with just two environmental risk factors (smoking and obesity)" (page 1169). Another reason why effort may be efficient for high risk only is that it has to combine several approaches, including drug therapies: "In many instances, dietary modification turns out to be insufficient (...) Thus drugs in the class known as statins have become the most widely prescribed in the developed world" (page 1313).

We assume perfect competition between profit-maximizing insurers, who observe whether individuals have taken the test, and the result of the test. On the other hand, insurers cannot force individuals to undertake the test, and/or the prevention effort. This corresponds to the situation labeled "disclosure duty" by Barigozzi and Henriet (2011), and to the legal environment in New Zealand and the United Kingdom. Observe

[1] Alternatively, the "test" could be an exploration of family history, which Collins (2010, page 1084) indeed dubs a "free genetic test".

[2] We thus do not cover the illnesses that are entirely driven by genetic conditions and/or for which there is no known prevention effort (such as, for instance, Huntington desease).

that this regulation is simply an extension to genetic testing of the generic regulation for insurance existing in most countries : when buying insurance (health or otherwise), buyers have to disclose the risk factors that they are aware of. For instance, health insurers may ask questions about the family history of the patient, and patients who lie or fail to disclose important elements may be penalized or even see their insurance contract made void.

We also assume, in line with existing conditions, that genetic insurance (*i.e.*, insurance against the risk of a positive test) is not available in the market. Taking the test then corresponds to a lottery, since it means (under disclosure duty and with separating insurance contracts) that the agent ends up with probability λ with the contract designed for high types, and with probability $1-\lambda$ with the contract designed for the low type, rather than with the contract designed for uninformed agents and based on the average risk $\lambda p_H + (1-\lambda)p_L$. In other words, taking the test means supporting a discrimination risk. We already know from previous literature (Hirshleifer, 1971) that, in a classical von Neumann-Morgenstein expected utility framework, risk averse agents will not undertake the test in the simple setting where the test does not allow to better calibrate prevention efforts.

We then add the possibility for the individuals to exert some primary prevention effort in order to decrease their probability of bad health from p_H to the lower p_H^1.[3] The availability of a prevention strategy should give stronger incentives to undertake the test. Whether individuals make the prevention effort and thus decrease their risk is also of interest to the insurers. An open question is whether this prevention effort is observable by insurers. Prevention is easily observable when it takes the form of surgery, or even drug therapy. It is much more difficult to observe if it consists of lifestyle changes such as dietary modifications or exercise. We thus cover the two cases, treating first the situation where the prevention is observable, and then the case where it is not observable by insurers. Throughout our analysis, we stress two dimensions of the prevention effort: its **cost** for the agent, and its **effectiveness**, *i.e.* the amount by which it reduces the risk of someone whose test is positive.[4]

We first study the benchmark situation where the effort is observable, verifiable and contractible by the insurers. Even in this simple situation, our results reveal that the value of information given by the test has an interesting relationship with the cost of the preventive actions. More precisely, we first point out that the genetic test generates a valuable information only for intermediate levels of the prevention cost. When the prevention effort cost is low, even uninformed people (who do not take the test) make

[3] See Barigozzi and Henriet (2011) for a comparison of legal environments in a setting with observable secondary prevention.

[4] We assume that the prevention effort is useless for an agent of type L. This assumption is made to simplify the analysis – our results would carry through qualitatively to the case where prevention is effective for type L as well, provided that the risk decreases more for type H than for type L.

the prevention effort, although it is efficient only with probability λ. In such a case, the genetic test precisely allows to forego the effort (and its cost) if the test is revealed negative. The value of the test, defined as the difference in *ex ante* utility between taking the test or not, is then increasing with the effort cost, and may become positive if both the cost and efficiency of effort are not too low. For intermediate values of the effort cost, agents undertake the prevention effort only if they have a positive test.[5] The test then allows them to undertake the prevention effort, and the value of the test is decreasing in the effort cost. This value if positive provided that the effort cost is not too large. Finally, when the effort cost is large, even high type agents do not undertake the effort, and the value of the test is always negative since the only impact of taking the test is to expose agents to the discrimination risk. As is intuitive, the value of the test increases with the efficiency of the prevention effort.

We then turn to the case where effort is not observable by insurers. Full insurance would induce agents not to provide any effort: we are facing a moral hazard problem, solved by insurers by under-providing insurance.[6] A naïve intuition would suggest that this under-provision, by reducing the utility level with effort (compared to the perfect information case) is detrimental to the value of the test, whose only *raison d'être* is to provide information allowing to calibrate the prevention effort to one's own circumstances. We show that this intuition does not hold in general. More precisely, this intuition is correct for the middle range of values of the effort cost, where the effort is undertaken only in the case of a (positive) test. But it does not hold when the effort cost is low enough that prevention is undertaken both if uninformed or if tested positive. In that case, we show that the value of the test is actually larger with than without moral hazard, because moral hazard degrades more the utility when the test is not taken (and effort is undertaken) than when it is taken (and effort undertaken only in the case of a positive test). Roughly, this is true because insurers have to ration coverage more to uninformed types than to high types in order to induce them to undertake the effort.

Comparing further the cases with and without moral hazard, we obtain two main results. First, for a given efficiency level of prevention, the interval of (intermediate) values of the effort cost which are inducing agents to take the test (*i.e.*, for which the value of the test is positive) moves to the left as we introduce moral hazard considerations. That is, quite counter intuitively, there exist combinations of effort cost and efficiency such that the genetic test is undertaken if and only if effort is not observable

[5] This corresponds to the following two observations by Collins (2010): "Information about an elevated genetic risk may cause people to take actions they otherwise would have ignored" (page 1313), and "She was aware that she was following diet and exercise routines that she probably should have adhered to anyway, but she found the additional genetic information helpful in inducing a greater sense of urgency to make these changes" (page 1461).

[6] For instance, Dave and Kaestner (2006) "find evidence that obtaining health insurance reduces prevention and increases unhealthy behaviors among elderly men."

by insurers! Second, we find occurrences where the test is undertaken for lower values of the efficiency of effort when this effort is unobservable than when it is observed by insurers. Both results are due to the fact that the value of the test is larger with than without moral hazard when the effort cost is sufficiently low that even uninformed agents undertake the prevention effort.

Finally, we assess the impact of the various ingredients of our model on *ex ante* (expected) utility or welfare. We start from the situation where there is no insurance, no genetic test and no prevention effort available, and we measure the impact on welfare of allowing each of these three ingredients as a function of the prevention effort cost. We also show that moral hazard is always detrimental to both the prevention effort decision and ex ante utility of agents. Observe that, in the light of the results presented above, this is not a foregone conclusion. For certain combinations of prevention effort cost and efficiency, the introduction of moral hazard considerations changes the testing and effort decisions of agents. At first sight, such a change could then be beneficial to the prevention decision and generate a larger welfare for agents if moral hazard were to induce agents to test while uninformed agents do not undertake the effort. We show that this situation never happens because, for moral hazard to induce the test, the effort cost need to be low enough that uninformed agents do undertake prevention.

The welfare analysis allows us to make several policy recommendations. First, targeted genetic tests (tailored for a specific disease) are to be encouraged rather than a single, all encompassing test, since the value of the information may be positive for certain health risks and negative for others. Second, our analysis provides no ground for policies that would indiscriminately increase the prevention efforts by all agents (for certain configurations of prevention efficiency and costs, there is already too much effort at equilibrium). Third, since the presence of a discrimination risk explains why there is less than optimal testing in our model, it is tempting to recommend that governments do away with this risk. We study three possible ways to proceed and we show that two of them actually decrease welfare! We recommend the creation of genetic insurance that would be made mandatory in order to take the genetic tests. We do not recommend two alternative policies: the strict prohibition of the use of genetic information by insurers (which creates an adverse selection problem if agents are aware of their genetic risk when buying health insurance), and requiring proof of health insurance when taking the tests (since it would blunt incentives to test and to make the effort when socially optimal). Finally, although moral hazard may have some welfare enhancing properties (when it decreases a prevention level that is socially too large), its overall impact on welfare is always negative. We then call for the enlargement of the "disclosure duty" approach to the prevention efforts, as well.

Related literature

This paper is part of a growing literature dealing with genetic testing and the value of information. An implication of the seminal paper by Hirshleifer (1971) is that, if health risk is exogenously determined (*i.e.*, there is no prevention effort available), the value of the information brought by the test is negative, because individuals are faced with a discrimination risk. Doherty and Thistle (1996) have further shown that the private value of information is non-negative only if insurers cannot observe consumers' information status or if consumers can conceal their informational status.[7] Several papers have extended this analysis to settings with prevention efforts.[8] As pointed out in Ehrlich and Becker (1972), preventive actions can be primary or secondary.

Secondary prevention (or self-insurance) is analyzed in Barigozzi and Henriet (2011) and Crainich (2011). Barigozzi and Henriet (2011) compare several regulatory approaches used in practice, from *laissez-faire* to the prohibition of tests. They show that policyholders are better off under a "disclosure duty" regulation, which is the one we study in this paper and where policyholders cannot been forced by insurers to undertake the test, but are obliged to disclose its results when known. The superiority of this regulation method is mainly due to the fact that it does not create any adverse selection problem for the insurers, while allowing to use the information provided by the test to self insure against the damage.[9] Crainich (2011) points out that the consequences of regulating the insurers' access to genetic information crucially depend on the nature of the equilibrium in the health insurance market – whether pooling or separating. Crainich (2011) also analyzes conditions to ensure that the genetic insurance market suggested by Tabarrok (1994) induces the optimal level of secondary prevention. We come back to this important point in section 6.

Primary prevention is considered in Doherthy and Posey (1998) and Hoel and Iversen (2002). Both papers assume that policyholders are not required to inform insurers about their test results and thus focus on the interplay between risk discrimination and adverse selection. Our framework is closer to Hoel and Iversen (2002). We share the assumption that only high risk people can reduce their health risk thanks to primary prevention actions, but we differ when they assume that uninformed policyholders never undertake preventions while we explore all cases in our paper. Also, Hoel and Iversen (2002) allow for both compulsory and voluntary (supplementary) health insurance.

The main difference between this paper and all the articles which introduce prevention (primary or secondary) is that we assume that primary prevention (especially

[7] Rees and Apps (2006) study how redistributional policies can counteract the discrimination risk in order to induce all buyers to supply their genetic information to the insurers.

[8] Another way to make testing more agreeable to individuals is to introduce a "repulsion from chance" component to their utility, as in Hoel *et al.* (2006).

[9] Hoy and Polborn (2000) and Strohmenger and Wambach (2000) also study the impact of genetic tests on the health insurance market in the presence of adverse selection.

when it consists of lifestyle improvements such as exercising or eating healthy food) is not observable by insurers, which gives rise to a moral hazard problem solve by providing partial insurance coverage.[10]

2 Setting and notation

The economy is composed of a unitary mass of individuals. Each individual may be sick with some probability, with sickness modeled as the occurrence of a monetary damage of amount d. Individuals belong to one of two groups according to their risk: a fraction λ of individuals are of type H and have a high probability, p_H^0, of incurring the damage (with $0 < \lambda < 1$), while the remaining fraction $1 - \lambda$ is of type L and has a lower probability, p_L (with $0 < p_L < p_H^0 < 1$). Therefore, the average risk in the society is given by $p_U^0 = \lambda p_H^0 + (1 - \lambda) p_L$.

Individuals are not aware of the group they belong to (*i.e.*, of their risk level) unless they take a genetic test.[11] The test is assumed to be costless and perfect, in the sense that it tells the individual who takes it with certainty whether he is of type L or H.[12] After having taken this test or not, individuals choose whether to exert some prevention effort. Unlike Barigozzi and Henriet (2011), we consider primary prevention –*i.e.*, an effort which decreases the probability that the damage occurs, but does not decrease the amount of the damage. For simplicity reason, we assume that the prevention decision is binary and that the effort cost (normalized to zero if no effort is undertaken) c is measured in utility terms rather than in money. The assumption of a utility cost fits better the behavior modification type of prevention effort, which is also the type of prevention that is the most difficult to observe for insurers. We further assume that prevention has no effect for a low risk individual, while it decreases the risk of a high risk individual to p_H^1, with $p_L \leq p_H^1 < p_H^0$. We capture the prevention efficiency through Δ with $\Delta = p_H^0 - p_H^1$. The parameter Δ can take any value between zero (prevention has no impact on risk, $p_H^1 = p_H^0$) and $\bar{\Delta} = p_H^0 - p_L$ (prevention decreases the risk of a type H agent to the level of a low risk agent, $p_H^1 = p_L$). The two characteristics of the prevention technology, its cost c and efficiency Δ, will play an important role in our analysis.

[10] A recent exception is the paper by Filipova and Hoy (2009), which focuses on surveillance and more precisely on the moral hazard risk of over-consumption of surveillance when financial costs are absorbed by the insurance pool. Also, they concentrate on the consequences of information on prevention, while we endogenize both the prevention and testing decisions.

[11] To shorten the text, we sometimes write that an individual is of type U when he is uninformed about his own type and thus believes that he has type H with probability λ and type L with probability $1 - \lambda$.

[12] The cost of genetic tests decreases exponentially and is believed to cross the $1,000 mark within a few years. See Collins(2011) and Davies (2010).

We now come to the description of the insurance market. We assume that there is a competitive fringe of profit-maximizing insurers. Insurers offer contracts that are composed of a premium π to be paid before the risk realization, and of an indemnity (net of the premium) I paid to the individual once and if the risk has materialized. Contracts can of course be conditioned upon what the insurers observe. Contracts are offered and bought after the individuals have obtained information from the test (provided they chose to take it), but before they exert any prevention effort.

The timing of the model consists in four sequential stages: (1) insurers offer contracts, (2) agents decide whether to take the test or not, (3) they choose one insurance contract (or remain uninsured), and (4) they then exert or not some prevention effort.

In the rest of the paper, we compute and compare the equilibrium allocations depending upon what is observed by the insurers. Section 3 studies the simplest scenario, where the insurers observe both whether an individual has taken the test or not, the result of the test, and whether the individual exerts a prevention effort or not. Effort is both observable, verifiable and contractible, so that insurers are allowed to condition the contract they offer on both the test result (when one is taken) and the prevention effort. Section 4 assumes that effort is not observable or contractible, so that insurers face a moral hazard problem. Section 5 studies the impact of introducing moral hazard on testing and prevention. Section 6 investigates the welfare characteristics of the equilibrium and discusses the role of the discrimination risk and how to move closer to the first best allocation. Section 7 concludes and presents policy recommendations.

3 Perfect information

In this section, insurers can observe all relevant information. This allows them to condition the contracts they offer on whether a test has been taken, its results and whether effort is provided or not. We then start by describing the contracts offered by the insurers, and we then move to the individuals' decisions of whether to test and to make a prevention effort.

3.1 Contracts offered by the insurers

With perfect information, insurance contracts can be conditioned on both the intrinsic risk of the individual (low, high or average if the individual has not taken the test) and on whether the individual exerts effort. By assumption, prevention has costs but no benefit when the individual is revealed by the test to be of a low type, so that the contracts offered to type L agents entail no prevention effort. Competition forces insurers to offer actuarially fair contracts, so that individuals prefer full insurance at these actuarially fair terms. Insurers may then offer at most five contracts.

One contract is destined to the low type agents (*i.e.*, those who have taken a test whose result has been negative, and who thus exert no effort): the premium is denoted by π_L and the indemnity (net of the premium) by I_L. The zero-profitability constraint together with full coverage impose that

$$\begin{aligned} \pi_L &= p_L d, \\ I_L &= (1-p_L)d. \end{aligned}$$

The expected utility of a low type agent buying this contract is then given by

$$\begin{aligned} U_L &= (1-p_L)v(y-\pi_L) + p_L v(y-d+I_L) \\ &= v(y-p_L d) \equiv v(c_L), \end{aligned}$$

where $v(.)$ is a classical von Neumann Morgenstein utility function ($v'(.) > 0$, $v''(.) < 0$) with y the individual's exogenous income. We then denote by c_L the consumption level of a low type agent.

The second contract will be sold to the high type agent who is *not* exerting any effort. The same analysis as above results in

$$\begin{aligned} \pi_H^0 &= p_H^0 d, \\ I_H^0 &= (1-p_H^0)d, \end{aligned}$$

and in an individual's utility of

$$\begin{aligned} U_H^0 &= (1-p_H^0)v(y-\pi_H^0) + p_H^0 v(y-d+I_H^0) \\ &= v(y-p_H^0 d) \equiv v(c_H^0), \end{aligned}$$

where the superscript 0 indicates that the agent makes no effort.

The third contract is aimed at the high type agent who *is* exerting effort. We then obtain that

$$\begin{aligned} \pi_H^1 &= p_H^1 d, \\ I_H^1 &= (1-p_H^1)d, \end{aligned}$$

with a resulting individual utility of

$$\begin{aligned} U_H^1 &= (1-p_H^1)v(y-\pi_H^1) + p_H^1 v(y-d+I_H^1) - c \\ &= v(y-p_H^1 d) - c \equiv v(c_H^1) - c, \end{aligned}$$

where the superscript 1 indicates that the agent makes a prevention effort. Observe that the two differences between U_H^1 and U_H^0 are the lower risk (recall that $p_H^1 \leq p_H^0$) and the utility cost of effort c.

Insurers also devise contracts to be sold to agents who are *not* taking the test and not exerting any effort. The risk level of these agents is given by

$$p_U^0 = \lambda p_H^0 + (1-\lambda)p_L,$$

so that they are offered a contract with

$$\begin{aligned} \pi_U^0 &= p_U^0 d, \\ I_U^0 &= (1-p_U^0)d, \end{aligned}$$

which results in an individual's utility level of

$$\begin{aligned} U_U^0 &= (1-p_U^0)v(y-\pi_U^0) + p_U^0 v(y-d+I_U^0) \\ &= v(y-p_U^0 d) \equiv v(c_U^0). \end{aligned}$$

Finally, the fifth contract is devised for the agent who is *not* taking the test but *is* exerting effort. The risk of this agent is given by

$$p_U^1 = \lambda p_H^1 + (1-\lambda)p_L,$$

so that he is offered a contract with

$$\begin{aligned} \pi_U^1 &= p_U^1 d, \\ I_U^1 &= (1-p_U^1)d, \end{aligned}$$

and a corresponding individual's utility of

$$\begin{aligned} U_U^1 &= (1-p_U^1)v(y-\pi_U^1) + p_U^1 v(y-d+I_U^1) - c \\ &= v(y-p_U^1 d) - c \equiv v(c_U^1) - c. \end{aligned}$$

We now turn to the contract chosen by the agent, *i.e.* whether they take the test and perform some prevention. Agents first choose whether to test, observe the result and then decide whether to exert effort. We then proceed backwards, solving first for the prevention decision before looking at the testing decision.

3.2 The choice of prevention

We first look at agents who have taken the test in the first stage of the game. These agents know with certainty (since the test is always correct) whether they are of type L (negative test) or H (positive test). Agents of type L have no incentive to perform the effort and so buy the contract (π_L, I_L) giving them a utility level of U_L.[13] Agents

[13] It is straightforward that agents prefer to be fully insured at an actuarially fair rate rather than not buying any contract and shouldering their risk alone (Mossin 1968).

of type H have the choice between two contracts (with and without effort) and choose the contract they prefer by comparing the utility level attained under the two contracts. Then, they buy the contract with effort provided that

$$\begin{aligned} & U_H^1 > U_H^0 \\ \Leftrightarrow \quad & v(c_H^1) - c > v(c_H^0) \\ \Leftrightarrow \quad & c < c_{max} \equiv v(c_H^1) - v(c_H^0). \end{aligned} \tag{1}$$

Not surprisingly, this condition imposes an upperbound on the cost of effort. Observe that, if this condition is satisfied, then no insurance firm will propose the no-effort contract (π_H^0, I_H^0) at equilibrium. If one firm were to do so, then another firm would propose the effort contract $(\pi_H^1, I_H^1 - \varepsilon)$ with ε small, would attract the patronage of all H type, and would make a strictly positive profit.

We now look at agents who have decided not to take the test. These agents do not know their true type, but only that they are of average type U. They choose the contract specifying effort offered by insurers to type U if it gives them a higher utility level than the same contract without prevention–*i.e.* if

$$\begin{aligned} & U_U^1 > U_U^0, \\ \Leftrightarrow \quad & v(c_U^1) - c > v(c_U^0) \\ \Leftrightarrow \quad & c < c_{min} \equiv v(c_U^1) - v(c_U^0). \end{aligned} \tag{2}$$

We can apply the same reasoning as above to show that, if it is individually optimal for an individual who has not taken the test to make a protection effort (resp., not to make an effort), then only the corresponding contract (π_U^1, I_U^1) (resp., the contract (π_U^0, I_U^0)) will be offered at equilibrium by private firms to this individual.

There are two reasons why $c_{min} < c_{max}$ if $\Delta > 0$. First, as the cost of effort does not depend on the type, it is always effective if type H, but not always effective if type U. Second, due to the higher actuarial premium paid by policyholders of type H, they are characterized by a lower consumption, so that their marginal utility is higher. They thus gain more than average type U from the lower premium made possible by the prevention effort.

Finally, it is easy to see that, if condition (1) is not satisfied, then no agent chooses to exert effort at equilibrium, and our model boils down to a special case of Hoel *et al.* (2002).

We then have the following result:[14]

Result 1 *Depending on the cost of prevention c, we are in one of the following three cases:*

[14] To simplify notation, we write c_{min} and c_{max} rather than $c_{min}(\Delta)$ and $c_{max}(\Delta)$.

a) $c < c_{min}$: all individuals who have chosen not to take the test buy a contract prescribing prevention effort, as well as agents who have taken a test and discovered that they belong to the high risk type.

b) $c_{min} < c < c_{max}$: only individuals who have taken the test and who are of a high risk type do buy a contract prescribing prevention.

c) $c > c_{max}$: no one buys a contract with prevention.

We now move to the first stage of the model, and assess under what circumstances individuals choose to make the test.

3.3 To test or not to test

The first stage decision of an individual –*i.e.* whether taking the test is worth its while, depends on the value of c, since it determines under what circumstances an individual makes a prevention effort. We cover in turn the three cases covered by Result 1: $c > c_{max}$ (so that effort is never undertaken), $c < c_{min}$ (so that effort is always undertaken, except if a test is taken and results in a low type), and finally $c_{min} < c < c_{max}$ (where the effort is undertaken only in the case of a positive test).

In all cases, we define as the value of the test, denoted by $\Psi(c, \Delta)$, the difference between the utility the agent gets with and without taking the test (anticipating in both cases the contract he will buy and whether he will make the prevention effort). Recall that the individual takes the test if and only if this value is positive.

3.3.1 No one undertakes prevention: $c \geq c_{max}$

Result 2 *When $c \geq c_{max}$, $\Psi(c, \Delta) < 0$, $\forall (c, \Delta)$ so that the test is not taken.*

Proof. In that case, we have

$$\begin{aligned} \Psi(c, \Delta) &= \lambda U_H^0 + (1 - \lambda) U_L - U_U^0 \\ &= \lambda v(y - p_H^0 d) + (1 - \lambda) v(y - p_L d) - v(y - p_U^0 d) < 0, \end{aligned}$$

so that individuals do not test. ■

This is the well known (Hirshleifer, 1971) result of the negative value of a genetic test whose results are observable and contractible but which does not allow the individual to use the information to mitigate his risk. The intuition is that taking the test is like buying a lottery, with a good outcome with probability $1 - \lambda$ and a bad outcome with probability λ. On the other hand, by not taking the test, the individual obtains a certain payoff (since he is perfectly insured) at an actuarially fair rate. If the individual is risk averse *i.e.* exhibits a concave utility function $v(.)$ (in the expected utility framework),

he prefers the sure and actuarially fair payoff to the lottery. We call this drawback of the test the discrimination risk, in line with Barigozzi and Henriet (2011).

Observe that Ψ is independent of both the cost and effectiveness of prevention, as long as the cost c is larger than the threshold c_{max}. We then have that $\Psi(c,\Delta) \equiv \Psi_0 < 0$ for $c > c_{max}$.

We now move to the case where effort is undertaken even when the test is not taken.

3.3.2 Uninformed types undertake prevention: $c \leq c_{min}$

The value of the test is given by

$$\begin{aligned}\Psi(c,\Delta) &= \lambda U_H^1 + (1-\lambda)U_L - U_U^1 \\ &= \lambda(v(y-p_H^1 d)-c) + (1-\lambda)v(y-p_L d) - \big(v(y-p_U^1 d)-c\big) \\ &= (1-\lambda)c - \big[v(y-p_U^1 d) - \big(\lambda v(y-p_H^1 d) + (1-\lambda)v(y-p_L d)\big)\big]. \quad (3)\end{aligned}$$

The first term in (3) measures the gain from the test, which allows to forgo the prevention effort cost c if the test proves negative (*i.e.*, with probability $1-\lambda$) while the terms between brackets represent the drawback from taking the test (moving from a certain payoff to a lottery with the same average payoff, since effort is undertaken even if the test is not taken, but pays off only if the agent has a high risk).

We are now in a position to state the following result:[15]

Result 3 *When $c \leq c_{min}$, the value of the test, $\Psi(c,\Delta)$, is positive provided that the prevention effort's cost c and efficiency Δ are large enough. Formally,*
a) there exists a unique value of Δ, denoted by $\tilde{\Delta}$, such that $0 < \tilde{\Delta} < \bar{\Delta}$ and $\Psi(c_{min},\tilde{\Delta}) = 0$;
b) for all $\Delta \geq \tilde{\Delta}$, there exists a unique value of c, denoted by $\tilde{c}_1(\Delta)$, such that $0 \leq \tilde{c}_1(\Delta) \leq c_{min}$ and $\Psi(\tilde{c}_1(\Delta),\Delta) = 0$;
c) $\Psi(c,\Delta) > 0$ for all $\Delta > \tilde{\Delta}$ and $\tilde{c}_1(\Delta) < c < c_{min}$;
d) for all $\Delta \geq \tilde{\Delta}$, $\tilde{c}_1(\Delta)$ decreases with Δ;
e) $\tilde{c}_1(\bar{\Delta}) = 0$.

There are two main drivers behind this result. First, the value of the test increases with the cost of prevention effort, c: although this may seem counter-intuitive, it is due to the fact that the test allows to forgo making the effort when its results are negative. Second, the value of the test also increases with the prevention efficiency Δ: although the expected monetary gain associated to a lower risk after prevention is the same whether the test is taken or not, the marginal utility of money is larger when taking

[15] Most proofs are relegated to the Appendix. Throughout the paper, when varying Δ, we keep p_H^0 fixed and we decrease p_H^1. In other words, we replace p_H^1 by $p_H^0 - \Delta$.

the test, since the gain occurs when the individual pays the large premium associated to being of type H rather than the average premium when the test is not taken.

Hence, when the efficiency of prevention is low, the value of the test remains negative for all values of $c \leq c_{min}$: any gain from taking the test (in terms of foregone cost of effort if the test results are negative) is too low to compensate for the discrimination risk entailed by the test. When Δ is large enough, Ψ becomes positive provided that the effort cost is large enough. Formally, we identify both a threshold $\tilde{\Delta}$ on effort efficiency and on cost, $\tilde{c}_1$, above which the value of the test is positive. The threshold cost decreases with prevention efficiency: the value of the test increases with Δ, so that it remains positive for lower values of c as Δ increases. When Δ reaches $\bar{\Delta}$, the value of the test is positive for all values of $c \leq c_{min}$.

We then move to the intermediate case, where effort is undertaken if and only if the policyholder is of type H.

3.3.3 Only informed types undertake prevention: $c_{min} < c < c_{max}$

In such a case, the value of the test for a policyholder is given by

$$\begin{aligned} \Psi(c, \Delta) &= \lambda U_H^1 + (1-\lambda) U_L - U_U^0, \\ &= \lambda(v(y - p_H^1 d) - c) + (1-\lambda) v(y - p_L d) - v(y - p_U^0 d). \end{aligned} \tag{4}$$

In this case, taking the test allows to make the prevention effort in case the test is positive. Equation (4) then shows that the discrimination risk associated to testing is mitigated by the lower premium, thanks to prevention, when the test is positive. It is then easy to see that the value of the test increases with prevention efficiency Δ, but decreases with the cost of effort c. This latter result is in stark contrast with the one obtained when even uninformed types undertake prevention, where taking the test allowed **not** to make the prevention effort in case of a negative result.

We then obtain the following result.

Result 4 *When $c_{min} \leq c < c_{max}$, the value of the test is positive provided that the prevention efficiency Δ is large while the effort cost c is small. Formally,*
a) for all $\Delta \geq \tilde{\Delta}$ (as defined in Result 3), there exists a unique value of c, denoted by $\tilde{c}_2(\Delta)$, such that $c_{min} \leq \tilde{c}_2(\Delta) < c_{max}$ and $\Psi(\tilde{c}_2(\Delta), \Delta) = 0$;
b) $\Psi(c, \Delta) > 0$ for all $\Delta > \tilde{\Delta}$ and $c_{min} < c < \tilde{c}_2(\Delta)$;
c) for all $\Delta \geq \tilde{\Delta}$, $\tilde{c}_2(\Delta)$ increases with Δ;
d) $\tilde{c}_1(\tilde{\Delta}) = \tilde{c}_2(\tilde{\Delta}) = c_{min}$ and $c_{min} < \tilde{c}_2(\bar{\Delta}) < c_{max}$.

The value of the test is positive provided that prevention is cost effective (same threshold $\tilde{\Delta}$ as in Result 3) and that the cost of effort is low enough. As effectiveness

increases, the threshold cost $\tilde{c}_2$ below which the value of the test is positive increases, so that the test is undertaken for larger values of c.

Figure 1 provides a graphical illustration of the value of the test as a function of prevention cost for four different values of the prevention efficiency. Throughout the paper, graphical illustrations are based on the following assumptions: $v(c) = \sqrt{c}$, $y = 5$, $d = 3$, $\lambda = 0.3$, $p_L = 0.1$, $p_H^0 = 0.6$, so that $\bar{\Delta} = 0.5$.

Insert Figure 1 around here

3.4 Testing and effort at equilibrium

We now summarize our results so far in the following proposition.

Proposition 1 *a) If the efficiency of prevention is low enough ($\Delta \leq \tilde{\Delta}$), the test is never chosen, whatever the prevention cost.*

b) If the efficiency of prevention is large enough ($\Delta > \tilde{\Delta}$), the test is chosen only if the prevention cost takes intermediate values: $\tilde{c}_1(\Delta) \leq c \leq \tilde{c}_2(\Delta)$.

c) The set of values of the prevention cost compatible with agents taking the test increases with the prevention efficiency.

We already know from Hirshleifer (1971) that the value of the test for agents is negative in the absence of prevention effort. Prevention may increase the value of the test, because the test determines whether prevention has a benefit or not. Hence, a large enough efficiency of prevention is a necessary condition for the test to be taken, as shown in part a) of Proposition 1. Part b) is less intuitive. Recall that if the prevention cost is low ($c < c_{min}$), prevention is undertaken in the absence of test. The gain from taking the test is then that it allows not to do a prevention effort if the test is negative. The test then allows to save the prevention cost c (with probability $1 - \lambda$). If the prevention cost is too low, then this gain from taking the test is dominated by the discrimination risk such that the value of the test remains negative. If the prevention cost is larger ($c_{min} < c < c_{max}$), agents undertake prevention only if they obtain a positive test. Taking the test is then a necessary condition to make the prevention effort, and the gain from the test decreases with the cost of prevention. If this cost is too large, the value of the test remains also negative.

The following proposition states when prevention is undertaken as a function of its cost and efficiency.

Proposition 2 *a) If the efficiency of prevention is low enough ($\Delta \leq \tilde{\Delta}$), then all agents undertake prevention if its cost is low enough ($c < c_{min}$) while no one undertakes prevention otherwise (if $c > c_{min}$).*

b) If the efficiency of prevention is large enough ($\Delta > \tilde{\Delta}$), then everyone undertakes prevention if its cost is low enough ($c < \tilde{c}_1(\Delta)$), only people of type H undertake prevention if its cost is intermediate ($\tilde{c}_1(\Delta) \leq c \leq \tilde{c}_2(\Delta)$) while no one makes a prevention effort otherwise (i.e., if $c > \tilde{c}_2(\Delta)$).

We illustrate the results of Propositions 1 and 2 on Figure 2, which depicts the thresholds $\tilde{c}_1$ (in yellow), c_{min} (in blue), $\tilde{c}_2$ (in green) and $c_{\max}$ (in purple) as functions of Δ. With this numerical example, the value of $\tilde{\Delta}$ is 4%. The area between the curves $\tilde{c}_2(\Delta)$ and $\tilde{c}_1(\Delta)$ represents the combinations of prevention cost and efficiency for which agents take the test, and where they make an effort only if this test is positive. Outside of this region, no individual takes the test. Combinations of (c, Δ) located below the c_{min} and $\tilde{c}_1(\Delta)$ curves are such that everyone makes the prevention effort, while combinations above the c_{min} and $\tilde{c}_2(\Delta)$ curve are such that no prevention effort is made.

Insert Figure 2 around here

We now move to the case where both the test and its results are observable and contractible, but where the prevention effort is not.

4 Unobserved prevention effort

In that case, we have a moral hazard problem, since the desired prevention effort has to be induced by the insurer by adequately crafting the insurance contracts. We proceed as in section 3 and we first study the contracts proposed by the insurers before moving to the choice of prevention effort and of testing by the agents.

4.1 Contracts offered by the insurers

First, observe that contracts without prevention effort are unchanged, compared to the previous section. These are the contracts offered to low-type (for whom making a prevention effort is not worthwhile), (π_L, I_L), to the high type (π_H^0, I_H^0) and the average type (π_U^0, I_U^0) who need not be induced to make an effort.

Look now at the contract offered to a high type who the insurer would like to induce to make an effort, which we denote by (π_H^1, I_H^1). For a type H individual to make

an effort, it must be the case that the following incentive compatibility (IC hereafter) constraint holds:

$$(1-p_H^1)v(b_H^1)+p_H^1v(d_H^1)-c\geq(1-p_H^0)v(b_H^1)+p_H^0v(d_H^1), \tag{5}$$

where b_H^1 and d_H^1 denote the consumption level of a type H individual buying the (π_H^1, I_H^1) contract in case they are lucky and in case the damage occurs–*i.e.*,

$$\begin{aligned} b_H^1 &= y-\pi_H^1, \\ d_H^1 &= y-d+I_H^1. \end{aligned}$$

The IC constraint (5) states that the individual, when buying the contract (π_H^1, I_H^1), is at least as well off making an effort (the LHS of (5)) than pretending to make one (the RHS of (5)). It is straightforward to see that such a result cannot be attained if the individual is provided with full coverage, since in that case consumption levels are equalized across states of the world ($b_H^1 = d_H^1$), and the individual never makes an effort. As pointed out by Shavell (1979), in such a case, the only way for the insurer to induce effort making is then to restrict the coverage offered to the individual (the competition between insurers ensures that the contracts remain actuarially fair). We denote the contracts as

$$\begin{aligned} \pi_H^1 &= \alpha_H p_H^1 d, \\ I_H^1 &= \alpha_H(1-p_H^1)d, \end{aligned}$$

where α_H is the (maximum) coverage rate offered to individuals of type H in order to induce them to make an effort. The value of α_H is implicitly obtained by solving the IC constraint (5) with equality. Restated in terms of c, we then obtain that

$$c=\Delta(v(b_H^1)-v(d_H^1)). \tag{6}$$

The IC constraint (6) equalizes, on its LHS, the cost of effort with its benefit on the RHS, given the contract offered to a high type pretending to undertake prevention. This benefit is the product of the efficiency of the prevention effort, Δ, with the utility gap between the two states of the world (sick or healthy) when making the effort. We have that $b_H^1 > d_H^1$: the insured is better off if the damage does not occur, which gives him the exact incentive needed to support the prevention effort cost c.

Observe that the same argument as in the previous section explains why the insurers offer either the contract (π_H^1, I_H^1) or the contract (π_H^0, I_H^0) to individuals of type H, depending upon which of the two contracts gives more utility to these buyers. In other words, competition among insurers ensures that only the utility-maximizing contract (given the observability constraints) is offered to types H.

Insurers face a similar problem with the individuals who have not taken the test. The effort-inducing contract offered to them is (π_U^1, I_U^1) with

$$\begin{aligned} \pi_U^1 &= \alpha_U p_U^1 d, \\ I_U^1 &= \alpha_U (1 - p_U^1) d, \end{aligned}$$

and with α_U satisfying the following incentive compatibility constraint

$$c = \lambda \Delta (v(b_U^1) - v(d_U^1)), \tag{7}$$

with

$$\begin{aligned} b_U^1 &= y - \pi_U^1, \\ d_U^1 &= y - d + I_U^1, \end{aligned}$$

and $b_U^1 > d_U^1$.

We obtain the following useful lemma.

Lemma 1 *a)* $\alpha_U < \alpha_H < 1$.
b) α_H *and* α_U *are decreasing in c. There exists a maximum value of c, denoted by* $\bar{c}_H$ *(respectively,* $\bar{c}_U$*) such that effort by type H (resp., U) may be induced only if* $c \leq \bar{c}_H$ *(resp.,* $c \leq \bar{c}_U$*). Moreover,* $\bar{c}_U < \bar{c}_H$.

Observe that there are two effects at play, both pushing towards a larger coverage rate for type H than for type U. First, the expected effectiveness of the prevention effort is larger for type H than for an average type, since for the latter there is a probability $1 - \lambda$ that his effort is actually worthless. Second, the utility gap between the good and bad states of the world is larger for type H than for type U for a given coverage level, because the insurance premium is larger for H than for U. Both effects explain why it is necessary to underprovide insurance by a smaller amount for a type H than for an average type in order to induce them to undertake the costly prevention effort.

The amount of coverage offered to type $i \in \{H, U\}$ equalizes his cost and benefit of prevention effort, with the latter equal to the product of the efficiency of prevention, Δ, by the utility difference between good and bad states of the world when making the effort (see equations (6) and (7)). As the cost of effort increases, it is necessary to increase this utility difference, and hence to reduce the coverage α_i offered to an individual of type i. At the limit, this coverage tends toward zero, determining the maximum value of the effort cost, $\bar{c}_i$, compatible with inducing prevention effort for

type i. Intuitively, this maximum prevention cost $\bar{c}_i$ is lower for type U (when effort works with probability λ) than for type H.[16]

Figure 3 illustrates Result 1 for our numerical example.

Insert Figure 3 around here

We now move to the prevention choice of agents.

4.2 The choice of prevention

An individual of type H chooses the contract inducing effort (with the expected utility denoted by U_H^{1MH}) rather than the other one proposed to his type if

$$\begin{aligned} & U_H^{1MH} > U_H^0 \\ \Leftrightarrow \quad & (1-p_H^1)v(b_H^1) + p_H^1 v(d_H^1) - c > v(c_H^0) \\ \Leftrightarrow \quad & c < c_{max}^{MH} \equiv (1-p_H^1)v(b_H^1) + p_H^1 v(d_H^1) - v(c_H^0). \end{aligned} \tag{8}$$

Likewise, the condition under which it is optimal for an individual who has not taken the test to exert some prevention effort is

$$\begin{aligned} & U_U^{1MH} > U_U^0, \\ \Leftrightarrow \quad & (1-p_U^1)v(b_U^1) + p_U^1 v(d_U^1) - c > v(c_U^0) \\ \Leftrightarrow \quad & c < c_{min}^{MH} \equiv (1-p_U^1)v(b_U^1) + p_U^1 v(d_U^1) - v(c_U^0). \end{aligned} \tag{9}$$

The following result parallels Result 1.

Result 5 *Uninformed agents undertake the effort provided that* $c \leq c_{min}^{MH} < \min[c_{min}, \bar{c}_L]$, *while type H agents exert the prevention effort provided that* $c \leq c_{max}^{MH} < \min[c_{max}, \bar{c}_H]$.

The maximum values of the prevention cost inducing (uninformed or type H) agents to make a prevention effort decrease when this effort is not observable by the insurers. The intuition for this result rests on the observation that contracts intended for effort-making agents are actuarially fair both with and without moral hazard, and differ only in the lower coverage rates offered with moral hazard. It is well known (Mossin, 1968) that

[16] As for the benefit of prevention, it need not increase with prevention efficiency, because a larger value of Δ decreases the utility gap between states of the world for a given coverage level (both consumption levels b_i^1 and d_i^1 increase by the same amount with Δ, but the marginal utility is larger in the bad state of the world –*i.e.*, with d_i^1). The non monotonic relationship between the prevention efficiency Δ and the level of coverage α in *ex ante* moral hazard model has already been pointed out in Bardey and Lesur (2005).

agents prefer full coverage when offered actuarially fair terms. It then results that the introduction of moral hazard degrades the utility obtained with the contract intended for effort-making agents, decreasing the maximum values of the effort cost compatible with making the effort.

We now move to the value of the genetic test.

4.3 To test or not to test

The value of the test depends upon whether effort is undertaken at equilibrium –*i.e.*, of how c compares with c_{min}^{MH} and $c_{\max}^{MH}$. As in the previous section, we consider three cases according to the value taken by the prevention cost.

4.3.1 No one undertakes prevention: $c \geq c_{max}^{MH}$

It is easy to see that

Result 6 *For $c \geq c_{max}^{MH}$, $\Psi^{MH}(c,\Delta) = \Psi(c,\Delta) = \Psi_0 < 0$, $\forall (c,\Delta)$ so that the test is not taken.*

Result 2 extends to the case with moral hazard, which is intuitive since we are back to the case where no prevention effort is undertaken, so that there is complete insurance at full coverage.

We now consider the case where even uninformed types make the prevention effort.

4.3.2 Uninformed types undertake prevention: $c \leq c_{min}^{MH}$

Individuals decide to take the test if

$$\begin{aligned} & \Psi^{MH}(c,\Delta) = \lambda U_H^{1MH} + (1-\lambda)U_L - U_U^{1MH} > 0 \\ \Leftrightarrow \quad & \lambda\left[(1-p_H^1)v(b_H^1) + p_H^1 v(d_H^1) - c\right] + (1-\lambda)v(c_L) - \left[(1-p_U^1)v(b_U^1) + p_U^1 v(d_U^1) - c\right] > 0. \end{aligned}$$

We first discuss the following lemma.

Lemma 2 *When $c \leq c_{min}^{MH}$, we have that*

$$\begin{aligned} & a)\ \frac{\partial \Psi^{MH}(c,\Delta)}{\partial c} > 1-\lambda \text{ if } \Delta \to \bar{\Delta}, \\ & b)\ \Psi^{MH}(0,\Delta) = \Psi(0,\Delta) \text{ for all } \Delta. \end{aligned}$$

As in the situation without moral hazard, taking the test allows to save on the cost of effort in case the test is negative –*i.e.*, with probability $1-\lambda$. Additionally, with moral hazard, insurers decrease their coverage to keep the incentive for policy holders

faced with a larger cost of effort to exert this prevention effort. This decreases both the utility of individuals who receive (with probability λ) a positive test and of those who do not take the test (and undertake prevention in all cases). The sign of the difference between these two affects is in general ambiguous, because types U and H differ both in coverage ($\alpha_U < \alpha_H$) and in risk ($p_H^1 \geq p_U^1$). When $\Delta \to \bar{\Delta}$, the risks of both types converge when they undertake prevention, while the coverage rate remains lower for type U than for type H (because prevention is effective only with probability λ for type U). We then obtain that a larger effort cost degrades more the utility of type U than of type H, because there is a larger utility gap between states of the world for type U (formally, $d_U^1 < d_H^1 < c_U^1 = c_H^1 < b_H^1 < b_U^1$), who then suffers more at the margin from the decrease in coverage rate. This in turn increases the value of the test, compared to the case where prevention is observable. Part b) of Lemma 2 is straightforward since the unobservability by insurers of the prevention effort does not matter when this effort is costless.

Finally, observe that the value of the test may not always increase in prevention efficiency, because the utility of an uniformed type may increase more with Δ than that of a type H, due to the partial and endogenous coverage offered by insurers to both types in order to induce them to make the prevention effort.

We then obtain the following result.

Result 7 *When $c \leq c_{min}^{MH}$, the value of the test is positive provided that the prevention efficiency Δ and the effort cost c are large enough. Formally, assume that Δ is large enough. We then have that*
a) there exists a (unique) value of c, denoted by $\tilde{c}_1^{MH}(\Delta)$, such that $\tilde{c}_1^{MH}(\Delta) < c_{min}^{MH}$ and $\Psi^{MH}(\tilde{c}_1^{MH}(\Delta), \Delta) = 0$. Moreover, $\tilde{c}_1^{MH}(\bar{\Delta}) = 0$;
b) $\Psi^{MH}(c, \Delta) < 0$ for $c < \tilde{c}_1^{MH}(\Delta)$ and $\Psi^{MH}(c, \Delta) > 0$ for $c > \tilde{c}_1^{MH}(\Delta)$.

This result is similar to the one obtained without moral hazard (Result 3): Lemma 2 implies that the value of the test is larger with than without moral hazard when $c \leq c_{min}^{MH}$ and $\Delta \to \bar{\Delta}$, so that we can identify a threshold effort cost level above (respectively, below) which agents do (resp., do not) undertake the test.[17] Observe that Result 7 concentrates on large values of Δ while Result 3 is stronger and shows the existence of a threshold value of Δ above which the value of the test is positive for low enough values of c. The fact that, unlike in the perfect information case, the value of the test may not always increase with Δ explains this weaker statement.

We now turn to the case where effort is undertaken if and only if the policyholder's type is high.

[17] We will compare the threshold costs with and without moral hazard in section 5.

4.3.3 Only informed types undertake prevention: $c_{min}^{MH} < c < c_{\max}^{MH}$

The value of the test for a policyholder is here given by

$$\begin{aligned}\Psi^{MH}(c,\Delta) &= \lambda U_H^{1MH} + (1-\lambda)U_L - U_U^0 \\ &= \lambda\left[(1-p_H^1)v(b_H^1) + p_H^1 v(d_H^1) - c\right] + (1-\lambda)v(c_L) - v(c_U^0).\end{aligned}$$

The next lemma states how prevention cost and efficiency affect the value of the test:

Lemma 3 *For $c_{min}^{MH} < c < c_{max}^{MH}$, we have that*

$$\begin{aligned} a)\ \frac{\partial \Psi^{MH}(c,\Delta)}{\partial \Delta} &> 0, \\ b)\ \frac{\partial \Psi^{MH}(c,\Delta)}{\partial c} &< 0. \end{aligned}$$

With intermediate values of c, the prevention efficiency Δ affects the value of the test only through its impact on the utility level attained by agents who obtain a positive test (and thus make the prevention effort). This impact is twofold. The direct impact of a larger efficiency Δ lowers both the risk and premium, for a given coverage level α_i, $i = \{U, H\}$ and thus increases the utility of this individual. The indirect impact of Δ takes place through variations in the coverage rate. If the coverage rate α_i increases with Δ, this indirect impact reinforces the direct one. We show in the proof that, even if the coverage rate decreases with Δ, the direct impact is larger than the indirect one, so that the value of the test always increases with Δ when $c_{min}^{MH} < c < c_{\max}^{MH}$. The impact of a higher prevention cost on the value of the test works similarly: the direct impact decreases the utility of the individual with a positive test (who makes the prevention effort) for a given insurance contract, while the indirect impact of c on the insurance contract is to decrease the coverage rate α_H proposed by the insurer (see Lemma 1), further damaging the utility of this individual and thus the value of the test.

Observe that the sign of the impact of c and Δ on the value of the test is the same as without moral hazard: the moral hazard effects, through variations in the coverage rate α_H, either reinforce the direct impact on the value of the test, in the case of c, or are swamped by the direct effect, in the case of Δ. This is in stark contrast with the previous section, where the fact that moral hazard affects the insurance contracts offered to both types H and U (since they both undertake prevention and are offered insurance contracts with partial coverage) renders the sign of the impact of c and Δ on the value of the test ambiguous in general.

We then obtain the following result.

Result 8 *When $c_{min}^{MH} \leq c < c_{max}^{MH}$, the value of the test is positive provided that the prevention efficiency Δ is large while the effort cost c is small. Formally, assume that Δ is large enough. We then have that*
a) there exists a unique value of c, denoted by $\tilde{c}_2^{MH}(\Delta)$, such that $c_{min}^{MH} \leq \tilde{c}_2^{MH}(\Delta) < c_{max}^{MH}$ and $\Psi^{MH}(\tilde{c}_2^{MH}(\Delta), \Delta) = 0$. Moreover, $c_{min}^{MH} < \tilde{c}_2^{MH}(\bar{\Delta}) < c_{max}^{MH}$;
b) $\Psi^{MH}(c, \Delta) > 0$ for $c_{min}^{MH} < c < \tilde{c}_2^{MH}(\Delta)$;
c) $\tilde{c}_2^{MH}(\Delta)$ increases with Δ.

This result is also similar to the one obtained without moral hazard (Result 4), with the same caveat as explained after Result 7, due to the ambiguity of the impact of prevention efficiency on the value of the test when only type H makes the prevention effort.

We now take stock of what we have learned when prevention is not observable, and we compare our results with the perfect information case.

5 The impact of introducing moral hazard on testing and prevention

We first summarize our results with unobservable prevention effort in the following propositions. They follow closely Propositions 1 and 2 obtained in the absence of moral hazard.

Proposition 3 *A sufficient condition for the test to be taken is that the efficiency of prevention is large enough and that the prevention cost takes intermediate values: $\tilde{c}_1^{MH}(\Delta) \leq c \leq \tilde{c}_2^{MH}(\Delta)$. Moreover, the threshold $\tilde{c}_2^{MH}(\Delta)$ increases with Δ.*

The main difference with Proposition 1 is due to the fact that, as we have underlined in section 4.3.2, the value of the test need not always be increasing in the efficiency of prevention when the cost of prevention is low enough that even uninformed types take the test. This prevents us from determining a specific prevention efficiency threshold above which individuals take the test for specific values of prevention also. This also prevents us from assessing how the lowest prevention cost compatible with taking the test varies with the prevention efficiency. Except for these caveats, the main gist of our results is not affected by the introduction of moral hazard: the test is undertaken provided that the prevention efficiency is large enough, and that prevention costs take intermediate values.

The following proposition states when preventions is undertaken as a function of its cost and efficiency and parallels Proposition 2.

Proposition 4 *a) If the efficiency of prevention is large enough, then everyone undertakes prevention if its cost is low enough ($c < \tilde{c}_1^{MH}(\Delta)$), only people of type H undertake prevention if its cost is intermediate ($\tilde{c}_1^{MH}(\Delta) \leq c \leq \tilde{c}_2^{MH}(\Delta)$) while no one makes a prevention effort otherwise (i.e., if $c > \tilde{c}_2^{MH}(\Delta)$).*
b) If the efficiency of prevention is low enough that $\Psi^{MH}(c, \Delta) < 0 \ \forall c$, then all agents undertake prevention if its cost is low enough ($c < c_{min}^{MH}$) while no one undertakes prevention otherwise (if $c > c_{min}^{MH}$).

The same caveats apply for Proposition 4 as for Proposition 3, compared to the situation where prevention is observable.

Figure 4 provides a graphical illustration of the value of the test as a function of prevention cost for five different values of the prevention efficiency. It is based on the same assumptions as those used to depict Figures 1 to 3, and is the equivalent, with moral hazard, of Figure 1.

Insert Figure 4 around here

Each curve on Figure 4 shows the value of the test as a function of prevention cost for a given value of prevention efficiency. All curves have the same shape, so we start by focusing on any curve –*i.e.*, on any given efficiency Δ. We observe that Ψ^{MH} is first increasing and convex in c. This complements nicely our analytical finding of Lemma 2 that the slope of Ψ^{MH} is larger than $1 - \lambda$ when $\Delta \to \bar{\Delta}$. The curve Ψ^{MH} is then (as proved in Lemma 3) decreasing in c until it reaches Ψ_0 for $c > c_{max}^{MH}$.[18] Finally, a striking characteristic of Figure 4 is that $\Psi^{MH}(c_{min}^{MH}, \Delta)$ is increasing in Δ: although a larger prevention efficiency does not always increase the value of the test for all values of c such that even untested types undertake effort, the maximum value of the test is indeed increasing with Δ in our numerical example.

We now look at the impact of the unobservability of the prevention effort. We first assume that Δ is fixed, and look at how the testing and prevention decisions are affected by moral hazard as a function of the cost of prevention effort, c. We assume that Δ is close to $\bar{\Delta}$, and that $c_{min}^{MH} < c_{min} < c_{max}^{MH} < c_{max}$ (the case where $c_{min}^{MH} < c_{max}^{MH} < c_{min} < c_{max}$ can be treated similarly and does not bring any new insight, so we leave it to the reader).

We then obtain the following proposition.

[18] A close examination of Figure 4 reveals that Ψ^{MH} is indeed not always increasing in Δ when $c < c_{min}^{MH}$, as suggested in section 4.3.2: we obtain that Ψ^{MH} increases with Δ for low values of c, and then decreases with Δ for larger values of $c < c_{min}^{MH}$.

Proposition 5 *Assume that Δ is large enough (close but not equal to $\bar{\Delta}$). Then*
(a) there exists a threshold $c_{min}^{MH} < \hat{c} < c_{min}$ such that the value of the test is larger (resp., lower) with than without moral hazard for all prevention costs below (resp., above) this threshold;
(b) for $\tilde{c}_1^{MH}(\Delta) < c < \min[\tilde{c}_1(\Delta), \tilde{c}_2^{MH}(\Delta)]$, the value of the test if positive with moral hazard but negative without: agents take the test if and only if there is moral hazard;
(c) for $\max[\tilde{c}_1(\Delta), \tilde{c}_2^{MH}(\Delta)] < c < \tilde{c}_2(\Delta)$, the value of the test if positive without moral hazard but negative with: agents take the test if and only if there is no moral hazard;
(d) the maximum value of the test is higher under moral hazard than without:

$$\Psi^{MH}(c_{min}^{MH}, \Delta) > \Psi(c_{min}, \Delta).$$

We give the intuition for this proposition, starting with part (a). Recall that the value of the test is defined as the difference between the expected utility of taking the test and of remaining uninformed about one's own risk. We know that the value of the test is larger with than without moral hazard when the effort cost is so low that even uninformed agents undertake the prevention effort (a direction consequence of Lemma 2). The reason is that, moral hazard damages more the utility of the uninformed type than that of type H, through a lower coverage.[19] By contrast, the value of the test is lower with than without moral hazard when only type H undertakes the prevention effort (*i.e.*, for intermediate values of the prevention cost). In that case, uninformed and low type agents receive the same contract (and thus utility level) with and without moral hazard. The contract offered to type H with moral hazard is degraded compared to the situation without moral hazard because of the partial coverage offered, hence lowering the value of the test. Since the value of the test is continuous in prevention cost whether prevention is observable or not, the intermediate value theorem implies that there exists a cost threshold below (resp., above) which the value of the test is larger (resp., lower) with than without moral hazard.

Part (b) shows that, for some values of the prevention cost low enough that even uninformed agents undertake prevention, the value of the test is positive if and only if prevention is **not** observable. Recall that the value of the test is negative for very low values of the prevention cost (since the discrimination risk trumps the gain from foregoing the cheap prevention effort if tested positive), whether prevention is observable or not. The result then obtains directly from the observation that the value of the test increases faster with effort cost with than without moral hazard (thanks to the increase in coverage rate of type H) when Δ is large enough. Similarly, part (c) establishes that, for larger values of the effort cost (such that the value of the test is lower with than

[19] As we explain after Lemma 2, this is true for Δ large enough that the main difference between these two types of agents is the coverage rate they buy, rather than their riskiness.

without moral hazard), agents undertake the test at equilibrium if and only if there is no moral hazard.

Finally, we give the intuition for part (d). In both cases (with and without moral hazard), the value of the test is measured for the prevention cost that renders uninformed agents indifferent between making the effort or not (anticipating the contract they obtain in each case). They also obtain the same contract in case of a negative test, or if they remain uninformed and do not exert any prevention. We then obtain that the value of the test is larger with moral hazard if the difference in utility levels between uninformed and type H agents is larger with moral hazard than without. We show that it is the case when prevention efficiency is close to its maximum, because, while the risks of the two types of agents converge in that case, the lower coverage offered by insurers to uninformed types (as opposed to type H) when prevention is not observable is especially detrimental to them.

With our numerical example, Proposition 5 holds for all values of Δ, as illustrated in Figure 5 for the case where $\Delta = 0.1 < \bar{\Delta} = 0.5$.

Insert Figure 5 around here

We now endogenize the decision to take the test and study the impact of introducing moral hazard on the amount of prevention effort at equilibrium.

Proposition 6 *Introducing moral hazard considerations (weakly) decreases the fraction of the population exerting the prevention effort.*

To prove this proposition, observe first that, for values of (c, Δ) such that the testing decision is not affected by moral hazard, the fraction of the population exerting the prevention effort either remains constant or decreases. This is a straightforward consequence of the fact (see Result 5) that $c_{min}^{MH} < c_{min}$ and that $c_{max}^{MH} < c_{max}$. We now show that the same result holds if (c, Δ) is such that the introduction of moral hazard changes the testing decision. Proposition 5 has shown that two situations may occur. The first one happens when (c, Δ) are such that the test is taken if and only if there is moral hazard. This case materializes when the effort cost is low enough ($c < \tilde{c}_1(\Delta) < c_{min}$) that, without moral hazard, all individuals choose to remain uninformed and to undertake the prevention effort. The decision to take the test when moral hazard exists then induces low type agents not to exert the effort, decreasing the prevention effort (since $c < \tilde{c}_2^{MH}(\Delta) < c_{max}^{MH}$). We then obtain that introducing moral hazard decreases the fraction of the population exerting the prevention effort at equilibrium by a factor $1 - \lambda$. A similar phenomenon appears when the effort cost is large enough that

agents take the test if and only if there is no moral hazard. The cost is large enough ($c > \tilde{c}_2^{MH}(\Delta) > c_{min}^{MH}$) that, with moral hazard, agents remain uninformed and do not exert effort while, without moral hazard, agents take the test and exert effort if they are of type H (since $c < \tilde{c}_2(\Delta) < c_{max}$). Hence, moral hazard also decreases prevention effort from a fraction λ of the population to zero.

The analysis we have performed up to now in this section looks at the impact of introducing moral hazard for a given value of Δ. We now look at how this impact varies as a function of Δ. The value of the test is larger with than without moral hazard when the prevention cost is low enough that uninformed agents undertake the effort. This suggests that making the test may be compatible with lower values of the prevention efficiency with than without moral hazard. Unfortunately, the larger value of the test with moral hazard can only be proven for large values of Δ. Resorting to numerical simulations, we obtain that the minimum value of Δ above which there exists an interval of prevention cost values compatible with taking the test is lower (at 3.4%) with than without moral hazard (where $\tilde{\Delta} = 4\%$). We then have that

Proposition 7 *Introducing moral hazard considerations may induce individuals to undertake the genetic test for lower values of the prevention efficiency Δ.*

Up to now, we have concentrated on the value of the test, and on the testing and prevention decisions of agents. We now look at their welfare level.

6 Welfare analysis

In this section, we investigate the impact of the availability of (observable or not) prevention effort, testing and insurance on the *ex ante* welfare of agents. We then contrast these results with the first best allocation, and we discuss three ways to do away with the discrimination risk that is at the root of the non optimality of the equilibrium allocation studied here.

We start from the simplest case where prevention is not available, and we then add sequentially the availability of prevention and of testing in order to measure their individual impact on welfare. We illustrate our results with the help of Figures 6 and 7, which depict welfare (*ex ante* utility) as a function of the prevention cost c, for a given value of Δ, under various scenarios.

Insert Figure 6

We start from the simplest situation where prevention is not available. In that case, whether the test is available or not plays no role: policyholders do not take the

test since it has only drawbacks, namely the discrimination risk. The *ex ante* utility level is then $v(c_U^0)$ which is of course independent of c. This utility level corresponds to the horizontal line on Figure 6.[20] We then introduce the possibility to exert effort but assume that the genetic test is not available. In that case, agents are uninformed about their individual risk and exert effort if and only if the effort cost is lower than the threshold c_{min} (see Result 1). Their *ex ante* utility is given by $v(c_U^1) - c$ for $c < c_{min}$, and $v(c_U^0)$ for $c \geq c_{min}$. We represent this utility level on Figure 6. The vertical distance between this utility level and the horizontal line (denoted by A on Figure 6) represents the *ex ante* utility gain from the prevention technology. It obviously decreases linearly (at a rate of one) with the cost of effort.

The next step consists in introducing the testing technology, assuming that the prevention effort is observable and the prevention efficiency Δ is large enough that the test is worth taking for certain values of c. We know from Result 3 that the test is taken only if the effort cost c is comprised between $\tilde{c}_1$ and $\tilde{c}_2$. For $c < \tilde{c}_1$, agents remain uninformed and exert effort, so that their utility remains $v(c_U^1) - c$, while if $c > \tilde{c}_2$ they also remain uninformed but do not exert effort, with a utility level of $v(c_U^0)$. For c in between $\tilde{c}_1$ and $\tilde{c}_2$, agents test and their *ex ante* utility is $\lambda(v(c_H^1) - c) + (1 - \lambda)v(c_L)$, which decreases with c at a rate of λ since the test enables those who, with probability λ, are of a high type to make the prevention effort at a cost c. Figure 6 depicts the value of the test as a function of the cost of prevention (vertical distance labeled B). It is composed of the gain from the targeted effort, minus the discrimination risk.

Before turning to the impact of moral hazard, we study the first best allocation in order to look for ways to improve upon the equilibrium allocation studied in this paper.[21]

The first best allocation maximizes the expected welfare of the (*ex ante* identical) individuals. Given risk aversion, the first best allocation should perfectly ensure against both the risk of being of type H (or discrimination risk) and the health risk, and should thus give the same (*ex post*) consumption to all individuals.[22] The test gives information that can be acted upon to reduce the health risk and is then prescribed to everyone. High type agents are all prescribed to do the prevention effort provided that its cost is not too large. From an *ex ante* perspective, if effort is prescribed for types H, the average probability to incur the damage in the economy equals p_U^1 and the individuals' expected utility is $v(c_U^1) - \lambda c$ because of the probability λ of being of type H and of having to do the effort. If type H agents are told not to make the effort, all agents

[20] This level is larger than the expected *ex ante* utility in case no insurance exists, which is given by $p_U^0 v(y - d) + (1 - p_U^0)v(y)$.

[21] The comparison between first best and equilibrium allocations under various assumptions is more easily made assuming away moral hazard. Moreover, the introduction of moral hazard would not change significantly the arguments made here.

[22] We assume that the effort cost, being a utility cost, is not ensurable.

obtain *ex ante* a utility level of $v(c_U^0)$ based on the higher average risk p_U^0. So, the first best solution entails effort for all agents of type H if and only if

$$\begin{aligned} & v(c_U^1) - \lambda c \geq v(c_U^0) \\ \Leftrightarrow \quad & c < \frac{v(c_U^1) - v(c_U^0)}{\lambda} = \frac{c_{min}}{\lambda}. \end{aligned}$$

The welfare level attainable under the first best allocation is represented on Figure 6. It corresponds to $v(c_U^1) - \lambda c$ if $c < c_{min}/\lambda$ and to $v(c_U^0)$ otherwise. Its slope with respect to c equals the probability of having to make the effort, which is λ if the effort cost is low enough, and zero otherwise.

The vertical distance C on Figure 6 represents the utility difference between expected welfare levels attained at the first-best and at the equilibrium allocation studied in this paper. The discrimination risk explains this difference, through two channels. First, the discrimination risk may bias the prevention decision of agents away from the first best level, leading to too much prevention (if $c < \tilde{c}_1$) or to too little of it (if $\tilde{c}_2 < c < c_{min}/\lambda$). Second, even when the prevention decisions are first best optimal (when $\tilde{c}_1 < c < \tilde{c}_2$), the discrimination risk by itself entails a decrease in the *ex ante* utility. It is then very tempting to infer as policy recommendation that the discrimination risk should be banned in order to move us closer to the first best allocation. It is important to remain cautious in this area, since there are different ways for a planner to do away with the discrimination risk, and since these different ways have very different welfare implications.

By far the best way to remove discrimination is to create a market selling insurance against the discrimination risk. Testing would then be allowed only after having shown proof of subscription to this "genetic insurance". In other words, it would be illegal to perform the genetic test without first purchasing this insurance. Tabarrok (1994) has shown that creating this insurance market would allow to decentralize the first best allocation.[23] To the best of our knowledge, no country has adopted such a policy, and no such insurance exists.

Another, much more travelled route to get rid of the discrimination risk consists in prohibiting insurers from asking the test results and from using this information. This corresponds to the "strict prohibition" regulation studied by Barigozzi and Henriet (2011) and implemented in Austria, Belgium, Denmark, France, Germany, Israel, Italy, Norway and the US. Note that, in that case, nothing prevents individuals from taking the test before buying insurance contracts, as assumed in our model. Even though insurers are prohibited from asking the test results, nothing prevents them from proposing menus of contracts that will be self selected by agents according to their (private) information

[23] Crainich (2011) analyzes conditions to ensure that the genetic insurance market suggested by Tabarrok (1994) induces the optimal level of secondary prevention.

about their genetic risk. In other words, strict prohibition introduces adverse selection into the insurance market, and Barigozzi and Henriet (2011) show that this results into strict prohibition being weakly dominated by the disclosure duty approach!

There is a third way to get rid of the discrimination risk, which is less demanding than the first one, since it does not entail the creation of a new insurance product covering this risk. As with Tabarrok (1994), agents would have to show proof of insurance before taking a test, but the insurance concerned is classical health insurance, rather than the (empirically non available) genetic insurance.[24] In other words, agents would have to take the test (if they wish to) after having bought health insurance, and not before. This would prevent insurers from distorting coverage rates in order to extract from agents their private information regarding their type, since this private information would not exist at the stage where agents buy health insurance contracts. Competition among insurers would then drive premia to their actuarially fair levels: insurers would offer a contract with the sure consumption level of c_U^0 if the agent performs no prevention, and of c_U^1 otherwise. Agents would decide about the prevention effort after having tested (or not), as in the sequence studied above, and would then perform prevention provided that its cost is low enough, and more precisely, that[25]

$$c < v(c_U^1) - v(c_U^0) = c_{min}.$$

The expected welfare of agents is then $v(c_U^1) - c$ if $c < c_{min}$ and $v(c_U^0)$ if $c \geq c_{min}$. This corresponds to the utility when the test is not available while effort is, and is thus weakly dominated by the disclosure duty situation studied in the rest of the paper. The intuition is that the provision of a pooling insurance contract interferes with the prevention decision, leading to too much prevention if the effort cost is lower than c_{min}, and to too little for larger values of this cost.

This comparison of three ways to get rid of discrimination risk shows that the only way to proceed to increase welfare consists in creating a new product, namely genetic insurance, while making it mandatory for those who wish to take genetic tests. The other ways to get rid of discrimination risk end up being detrimental for *ex ante* welfare, either because of adverse selection by insurers, or because the pooling of health insurance interferes with the incentives to do the prevention effort.

We now turn to Figure 7, which depicts the impact of making the prevention effort unobservable when the testing technology is available (but entails a discrimination risk). Lemma 4 in the Appendix shows that moral hazard reduces the two cost thresholds below which, respectively, policyholders exert effort if uninformed about their own risk

[24] A similar mechanism (although in a different context) can be found in Cochrane (1995).

[25] Observe that agents are indifferent about testing or not, since the pay off they obtain (either $v(c_U^1)-c$ or $v(c_U^0)$) depends only upon effort and not upon their type.

level ($c_{min}^{MH} < c_{min}$) and if they know their type to be H ($c_{max}^{MH} < c_{max}$). Moreover, the *ex ante* utility is lower with moral hazard, even when the testing decision is the same than without moral hazard, because of the lower coverage implied by the unobservability of the prevention effort. Figure 7 represents this welfare loss of moral hazard as the vertical distance D between the two curves.

Insert Figure 7

7 Conclusion

We have studied the situation where a costless genetic test perfectly informs an individual about his risk of developing a specific disease in the future. This information allows the individual to better inform his decision to undertake a costly prevention effort, which reduces his probability of incurring the health damage in the case the genetic test is positive. The drawback of the genetic test is that its results are used by insurers to price their insurance policies, so that agents undertaking the test are faced with a discrimination risk. We first show that, when the prevention effort is observable, the pros of the test are larger than its cons when the prevention efficiency is large while its cost is neither too low nor too high. We then obtain that, when effort is not observable by insurers, the private value of the genetic test is not always increasing with the efficiency of prevention. Also, and contrary to the intuition, the value of the test may actually be larger when effort is not observable, so that the test may be taken for lower values of the prevention efficiency than when prevention is observable.

What policy implications can we derive from this paper? Even when effort is observable, there is too little testing since people choose to test only for intermediate values of the prevention effort cost, while the first best allocation calls for testing for a larger set of values of this cost. The equilibrium prevention level can be too small or too large: while optimality calls for only type H to perform effort, with a low prevention cost there is actually too much prevention (all undertake the effort) while with a large prevention cost there is too little of it (no one exerts the prevention effort). This model then does not provide ground to recommend policies that would result in a general increase in prevention efforts by all. Pushing for more testing would not be advisable either, because of the utility cost (the discrimination risk) that is associated to taking the test.

Since this discrimination risk is at the root of the inefficiencies exhibited by the equilibrium allocation (both because it decreases directly the utility of agents and because it biases their testing and prevention decisions away from the socially optimal levels), the main recommendation is to get rid of this risk. We have shown that, out of three

ways to proceed to make discrimination risk disappear, only one allows to decentralize the first best allocation: completing the insurance markets by creating a "genetic insurance" against the risk of a positive test, and making this insurance mandatory in order to test. The other two procedures studied actually result in a worse *ex ante* welfare level than the equilibrium allocation studied here: the "strict prohibition" regulation introduces adverse selection into the problem, while requiring that agents buy health (as opposed to genetic) insurance before testing defeats the purpose of the test because it suppresses the agents' incentive to tailor their prevention decision to the test result. Our main recommendation is then to combat discrimination risk by making genetic insurance mandatory, together with implementing the disclosure duty regulation on the testing decision and results.

Moral hazard considerations further reduce *ex ante* welfare. This is true even though moral hazard may actually induce agents to take the test, for certain configurations of the effort cost and efficiency parameters for which the test would not be taken without moral hazard. Also, this happens even though taking the test allows to make the effort only if it is socially worthwhile. The reason is that moral hazard, by decreasing the coverage rate offered to those insurers want to induce to exert the prevention effort, reduces more the utility of uninformed than of informed types. So, even if moral hazard may have beneficial effects on both the testing and prevention decisions, its net impact on welfare is always negative. This calls for policy measures that would make prevention effort more easily observable by insurers. One prominent such measure would consist in enlarging the scope of disclosure duty to prevention decisions: insurees could not be obliged to perform such an effort, but would be required to disclose truthfully whether they have stopped smoking or perform physical exercise regularly. In other words, one conclusion of our work is that disclosure duty should be embraced not only for genetic tests, but also for the prevention activities whose desirability they inform.

Another policy recommendation concerns the breadth of the tests, measured by the number of health problems a genetic test shows light on. There is a lot of talk and projections about decoding the whole genome of individual human beings, allowing to screen for as many potential risk issues as possible in a single, global test. As long as discrimination risks persist, such a global test has a lower value than the sum of narrower tests aiming at a single health issue at a time. Even if the value of the global test is positive, it may include information on specific diseases for which the configuration of prevention cost and effectiveness is such that agents would prefer not to be informed about these specific risks. At the limit, the value of a global test may be negative, even though the value of several of its components is positive. We then advocate the issue of targeted rather than all encompassing tests, allowing the individuals to choose the tests whose value is positive.

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8 Appendix

8.1 Proof of Result 3

a) First note that $c_H^1 - c_H^0 = (p_H^0 - p_H^1)d$ while $c_U^1 - c_U^0 = \lambda(p_H^0 - p_H^1)d$, so that $c_{min} = c_{max} = 0$ if $\Delta = 0$, and that $\Psi(0,0) < 0$. We also know that $\partial c_{min}/\partial\Delta = \lambda dv'(c_U^1) > 0$ (so that $c_{min} > 0$ if $\Delta > 0$) which, together with $\partial\Psi(c\,,\Delta)/\partial c > 0$ and $\partial\Psi(c,\Delta)/\partial\Delta > 0$, implies that

$$\frac{d\Psi(c_{min},\Delta)}{d\Delta} = \frac{\partial\Psi(c_{min},\Delta)}{\partial c}\frac{\partial c_{min}}{\partial\Delta} + \frac{\partial\Psi(c_{min},\Delta)}{\partial\Delta} > 0.$$

Finally, we know that $\Psi(0,\bar{\Delta}) = 0$ and that $c_{min} > 0$ when $\Delta = \bar{\Delta}$, which imply that $\Psi(c_{min},\bar{\Delta}) > 0$. The continuity of $\Psi(c,\Delta)$ in Δ together with the fact that $\Psi(c,\Delta)$ is strictly increasing with Δ for any c implies, by the intermediate value theorem, that there exists a unique value $0 < \Delta < \bar{\Delta}$, denoted by $\tilde{\Delta}$, such that $\Psi(c_{min},\tilde{\Delta}) = 0$.
b) By the same reasoning as above, we know that $\Psi(c_{min},\Delta) > 0$ for all $\Delta > \tilde{\Delta}$. The fact that $\partial\Psi(c\,,\Delta)/\partial c > 0$ and that $\Psi(0,\Delta) \leq 0$ for all $\Delta > \tilde{\Delta}$ imply, by the intermediate value theorem, that there exists a unique value of c, denoted by $\tilde{c}_1(\Delta)$, such that $0 \leq \tilde{c}_1(\Delta) \leq c_{min}$ and $\Psi(\tilde{c}_1(\Delta),\Delta) = 0$;

c) Straightforward since $\partial\Psi(c\,,\Delta)/\partial c > 0$.
d) We have by definition that $\Psi(\tilde{c}_1(\Delta),\Delta) = 0$ so that

$$\frac{d\Psi(\tilde{c}_1(\Delta),\Delta)}{d\Delta} = \frac{\partial\Psi(\tilde{c}_1(\Delta),\Delta)}{\partial\tilde{c}_1(\Delta)}\frac{\partial\tilde{c}_1(\Delta)}{\partial\Delta} + \frac{\partial\Psi(\tilde{c}_1(\Delta),\Delta)}{\partial\Delta} = 0.$$

Our claim then results from the fact that $\partial\Psi(c\,,\Delta)/\partial c > 0$ and that $\partial\Psi(c\,,\Delta)/\partial\Delta > 0$ for all c and Δ.
e) Straightforward since $\Psi(0,\bar{\Delta}) = 0$.

□

8.2 Proof of Result 4

a) First, part a) of the proof of Result 3 has shown that $\Psi(c_{min},\Delta) > 0$ for all $\Delta > \tilde{\Delta}$. Second, Result 2 has shown that $\Psi(c_{max},\Delta) < 0$ for all Δ. The fact that $\partial\Psi(c\,,\Delta)/\partial c = -\lambda < 0$ then implies, by the intermediate value theorem, that there exists a unique value of c, denoted by $\tilde{c}_2(\Delta)$, such that $c_{min} \leq \tilde{c}_2(\Delta) < c_{max}$ and $\Psi(\tilde{c}_2(\Delta),\Delta) = 0$;
b) Straightforward since $\partial\Psi(c\,,\Delta)/\partial c = -\lambda < 0$.
c) We have by definition that $\Psi(\tilde{c}_2(\Delta),\Delta) = 0$ so that

$$\frac{d\Psi(\tilde{c}_2(\Delta),\Delta)}{d\Delta} = \frac{\partial\Psi(\tilde{c}_2(\Delta),\Delta)}{\partial c}\frac{\partial\tilde{c}_2(\Delta)}{\partial\Delta} + \frac{\partial\Psi(\tilde{c}_2(\Delta),\Delta)}{\partial\Delta} = 0.$$

Our claim then results from the fact that $\partial\Psi(c\,,\Delta)/\partial c < 0$ and that $\partial\Psi(c\,,\Delta)/\partial\Delta = \lambda dv'(c_H^1) > 0$ for all c and Δ.
d) The fact that $\tilde{c}_1(\tilde{\Delta}) = \tilde{c}_2(\tilde{\Delta}) = c_{min}$ comes from the definitions of $\tilde{\Delta}$, $\tilde{c}_1(\Delta)$ and $\tilde{c}_2(\Delta)$. The fact that $c_{min} < \tilde{c}_1(\bar{\Delta}) < c_{max}$ comes from the observation that $\Psi(c_{min},\bar{\Delta}) > 0$ while $\Psi(c_{max},\bar{\Delta}) < 0$.

□

8.3 Proof of Lemma 1

a) α_H and α_U are respectively implicitly determined by

$$c = (p_H^0 - p_H^1)(v(b_H^1) - v(d_H^1))$$

and,

$$c = \lambda(p_H^0 - p_H^1)(v(b_U^1) - v(d_U^1)).$$

It is worth noticing that $\alpha_U = \alpha_H$ in the special case $\lambda = 1$ (since $p_U^1 = p_H^1$). Then, let us consider the following function

$$F(\alpha_U,\lambda) = \lambda(p_H^0 - p_H^1)(v(b_U^1) - v(d_U^1)) - c.$$

The implicit function theorem gives

$$\begin{aligned}\frac{d\alpha_U}{d\lambda} &= -\frac{\partial F(\alpha_U,\lambda)/\partial\lambda}{\partial F(\alpha_U,\lambda)/\partial\alpha_U} \\ &= \frac{\left(p_H^0-p_H^1\right)\left[v(b_U^1)-v(d_U^1)+\lambda\alpha_U\left(p_H^1-p_L\right)d\left(v'(d_U^1)-v'(b_U^1)\right)\right]}{\lambda\Delta\left(p_U^1v'(b_U^1)+(1-p_U^1)v'(d_U^1)\right)}>0.\end{aligned}$$

b) The implicit function theorem implies:

$$\frac{\partial\alpha_H}{\partial c}=-\frac{1}{\Delta d\left[p_H^1v'(b_H^1)+\left(1-p_H^1\right)v'(d_H^1)\right]}<0.$$

The coverage rate α_H attains the minimum value of zero when

$$c=\bar{c}_H=\Delta(v(y)-v(y-d)).$$

We proceed similarly to prove that α_U is decreasing in c, and that the minimum value of $\alpha_U=0$ is reached when

$$c=\bar{c}_U=\lambda\Delta(v(y)-v(y-d)),$$

so that $\bar{c}_U<\bar{c}_H$.

□

8.4 Proof of Result 5

We have respectively

$$\begin{aligned}c_{min}^{MH}-c_{min} &= (1-p_U^1)v(b_U^1)+p_U^1v(d_U^1)-v(c_U^0)-\left(v(c_U^1)-v(c_U^0)\right) \\ &= (1-p_U^1)v(b_U^1)+p_U^1v(d_U^1)-v(c_U^1)<0\end{aligned}$$

and

$$\begin{aligned}c_{max}^{MH}-c_{max} &= (1-p_H^1)v(b_H^1)+p_H^1v(d_H^1)-v(c_H^0)-\left(v(c_H^1)-v(c_H^0)\right) \\ &= (1-p_H^1)v(b_H^1)+p_H^1v(d_H^1)-v(c_H^1)<0.\end{aligned}$$

Also, When $c=\bar{c}_H$, we have $\alpha_H=0$ so that the agent is not insured at all (and is indifferent between making the prevention effort or not). His utility is then lower than what he gets under U_H^0, where he is fully insured at an actuarially fair price (without effort). Since U_H^{1MH} is decreasing in c (because of both the direct effect of a higher c and the indirect impact through the decrease in coverage rate) while U_H^0 is not affected by c, we have that $c_{max}^{MH}<\bar{c}_H$. The proof that $c_{min}^{MH}<\bar{c}_L$ is obtained in a similar way.

□

8.5 Proof of Lemma 2

a) We have that

$$\begin{aligned}\frac{\partial \Psi^{MH}(c,\Delta)}{\partial c} &= 1-\lambda \\ &\quad +\lambda\left[(1-p_H^1)p_H^1 d\frac{\partial \alpha_H}{\partial c}\left[v'(d_H^1)-v'(b_H^1)\right]\right] \\ &\quad -\left[p_U^1(1-p_U^1)d\frac{\partial \alpha_U}{\partial c}\left[v'(d_U^1)-v'(b_U^1)\right]\right].\end{aligned}$$

We then have that

$$\frac{\partial \Psi^{MH}(c,\Delta)}{\partial c} \geq \frac{\partial \Psi(c,\Delta)}{\partial c} = 1-\lambda$$

if and only if

$$\lambda\left[\frac{(1-p_H^1)p_H^1\left[v'(d_H^1)-v'(b_H^1)\right]}{\left[p_H^1 v'(b_H^1)+(1-p_H^1)v'(d_H^1)\right]}\right] \leq \left[\frac{p_U^1(1-p_U^1)\left[v'(d_U^1)-v'(b_U^1)\right]}{\lambda\left[p_U^1 v'(b_U^1)+(1-p_U^1)v'(d_U^1)\right]}\right].$$

If $\Delta \to \bar{\Delta}$, this condition simplifies to

$$\lambda^2\left[\frac{v'(d_H^1)-v'(b_H^1)}{p_L v'(b_H^1)+(1-p_L)v'(d_H^1)}\right] \leq \left[\frac{v'(d_U^1)-v'(b_U^1)}{p_L v'(b_U^1)+(1-p_L)v'(d_U^1)}\right].$$

A sufficient condition is

$$\begin{aligned}&\left[p_L v'(b_U^1)+(1-p_L)v'(d_U^1)\right]\left[v'(d_H^1)-v'(b_H^1)\right] \leq \left[p_L v'(b_H^1)+(1-p_L)v'(d_H^1)\right]\left[v'(d_U^1)-v'(b_U^1)\right], \\ \iff\ & v'(d_U^1)v'(b_H^1) \geq v'(b_U^1)v'(d_H^1),\end{aligned}$$

which is true since $d_U^1 < d_H^1$ for $\Delta \to \bar{\Delta}$.

The proof of part b) of the lemma is straightforward.

□

8.6 Proof of Result 7

a) Start by assuming that $\Delta = \bar{\Delta}$. We know from Lemma 2 b) that

$$\Psi^{MH}(0,\bar{\Delta}) = \Psi(0,\bar{\Delta}) = 0.$$

Part a) of Lemma 2 shows that

$$\frac{\partial \Psi^{MH}(c,\bar{\Delta})}{\partial c} \geq \frac{\Psi(c,\bar{\Delta})}{\partial c} = 1-\lambda \text{ for all } c < c_{min}^{MH},$$

which implies that

$$\Psi^{MH}(c, \bar{\Delta}) > 0 \text{ for all } c < c_{min}^{MH}.$$

We then have that $\tilde{c}_1^{MH}(\bar{\Delta}) = 0$.

Assume now that $\Delta < \bar{\Delta}$ while remaining close enough. Observe that, by continuity of Ψ and Ψ^{MH} in c, we have that

$$\frac{\partial \Psi^{MH}(c, \Delta)}{\partial c} \geq \frac{\Psi(c, \Delta)}{\partial c} = 1 - \lambda \text{ for all } c < c_{min}^{MH} \text{ and } \Delta \to \bar{\Delta}.$$

We know from Lemma 2 b) that

$$\Psi^{MH}(0, \Delta) = \Psi(0, \Delta) < 0.$$

Since $\Psi^{MH}(c, \Delta)$ is continuous in c, we have that

$$\Psi^{MH}(c_{min}^{MH}, \Delta) > 0.$$

By the intermediate value theorem, there exists a unique value of c, denoted by $\tilde{c}_1^{MH}(\Delta)$, such that $\tilde{c}_1^{MH}(\Delta) < c_{min}^{MH}$ and $\Psi^{MH}(\tilde{c}_1^{MH}(\Delta), \Delta) = 0$.

b) The proof is straightforward by definition of $\tilde{c}_1^{MH}(\Delta)$ and by the intermediate value theorem.

□

8.7 Proof of Lemma 3

a) Observe that, for $c_{min}^{MH} < c < c_{max}^{MH}$, we have

$$\Psi^{MH}(c, \Delta) = \lambda \left(c_{max}^{MH} - c + v(c_H^0) \right) + (1 - \lambda) v(c_L) - v(c_U^0),$$

so that

$$\frac{\partial \Psi^{MH}(c, \Delta)}{\partial \Delta} = \lambda \frac{\partial c_{max}^{MH}}{\partial \Delta}.$$

The derivative of c_{max}^{MH} with respect to Δ is

$$\begin{aligned} \frac{\partial c_{max}^{MH}}{\partial \Delta} &= v(b_H^1) - v(d_H^1) + \alpha_H d \left[\left(1 - p_H^1\right) v'(b_H^1) + p_H^1 v'(d_H^1) \right] \\ &\quad + d \frac{\partial \alpha_H}{\partial \Delta} \left(1 - p_H^1\right) p_H^1 \left(v'(d_H^1) - v'(b_H^1) \right) \end{aligned}$$

If $\partial\alpha_H/\partial\Delta > 0$, it is clear that $\partial c_{max}^{MH}/\partial\Delta > 0$. On the contrary, if $\partial\alpha_H/\partial\Delta < 0$, the sign is *a priori* ambiguous. We have

$$\begin{aligned}\frac{\partial c_{max}^{MH}}{\partial\Delta} &= v(b_H^1) - v(d_H^1) + \alpha_H d v'(b_H^1) \\ &\quad + \alpha_H d p_H^1 \left[v'(d_H^1) - v'(b_H^1)\right] + d\frac{\partial\alpha_H}{\partial\Delta}\left(1-p_H^1\right)p_H^1\left(v'(d_H^1) - v'(b_H^1)\right).\end{aligned}$$

A sufficient condition to have $\partial c_{max}^{MH}/\partial\Delta > 0$ is then

$$\alpha_H + \frac{\partial\alpha_H}{\partial\Delta}\left(1-p_H^1\right) \geq 0.$$

Using the implicit function on the equation defining α_H, we obtain

$$\frac{d\alpha_H}{d\Delta} = \frac{v(b_H^1) - v(d_H^1) + \Delta\alpha_H d\left[v'(b_H^1) - v'(d_H^1)\right]}{\Delta d\left[p_H^1 v'(b_H^1) + \left(1-p_H^1\right)v'(d_H^1)\right]},$$

whose denominator is always positive. Therefore, the previous sufficient condition becomes

$$\begin{aligned}&\alpha_H \geq -\left(\frac{v(b_H^1) - v(d_H^1) + \Delta\alpha_H d\left[v'(b_H^1) - v'(d_H^1)\right]}{\Delta d\left[p_H^1 v'(b_H^1) + \left(1-p_H^1\right)v'(d_H^1)\right]}\right)\left(1-p_H^1\right) \\ \Leftrightarrow\quad &\Delta d\alpha_H\left[p_H^1 v'(b_H^1) + \left(1-p_H^1\right)v'(d_H^1) + \left[v'(b_H^1) - v'(d_H^1)\right]\left(1-p_H^1\right)\right] \geq -\left(v(b_H^1) - v(d_H^1)\right)\left(1-p_H^1\right) \\ \Leftrightarrow\quad &\Delta d\alpha_H v'(b_H^1) \geq -\left(v(b_H^1) - v(d_H^1)\right)\left(1-p_H^1\right),\end{aligned}$$

which is always true since the RHS is negative.

b) The derivative of $\Psi^{MH}(c,\Delta)$ with respect to c is

$$\begin{aligned}\frac{\partial\Psi^{MH}(c,\Delta)}{\partial c} &= \lambda\left(-1 - \left(1-p_H^1\right)v'(b_H^1)\frac{\partial\alpha_H}{\partial c}p_H^1 d + p_H^1 v'(d_H^1)\frac{\partial\alpha_H}{\partial c}\left(1-p_H^1\right)d\right) \\ &= \lambda\left(p_H^1\left(1-p_H^1\right)d\frac{\partial\alpha_H}{\partial c}\left[v'(d_H^1) - v'(b_H^1)\right] - 1\right) < 0.\end{aligned}$$

□

8.8 Proof of Result 8

a) Recall that, for $c_{min}^{MH} < c < c_{max}^{MH}$, we have

$$\Psi^{MH}(c,\bar{\Delta}) = (1-\lambda)v(c_L) + \lambda\left[(1-p_L)v(b_H^1) + p_L v(d_H^1) - c\right] - v(c_U^0).$$

Result 6 has shown that $\Psi^{MH}(c_{max}^{MH}, \bar{\Delta}) < 0$. We now prove that $\Psi^{MH}(c_{min}^{MH}, \bar{\Delta}) > 0$.We have

$$\begin{aligned}\Psi^{MH}(c_{min}^{MH}, \bar{\Delta}) &= (1-\lambda)\left[v(c_L) - v(c_U^0)\right] \\ &\quad +\lambda\left[p_L\left[v(d_H^1) - v(d_U^1)\right] + (1-p_L)\left[v(b_H^1) - v(b_U^1)\right]\right].\end{aligned} \tag{10}$$

Note that the first term of (10) is positive. Moreover, using the mean value theorem, we obtain that

$$\begin{aligned}v(d_H^1) - v(d_U^1) &= v'(\varpi)d(1-p_L)\left(\alpha_H - \alpha_U\right), \\ v(b_H^1) - v(b_U^1) &= v'(\nu)dp_L\left(\alpha_U - \alpha_H\right),\end{aligned}$$

with $\varpi \in \left[d_H^1, d_U^1\right]$ and $\nu \in \left[b_H^1, b_U^1\right]$. Therefore, the second term of (10) becomes

$$\begin{aligned}&p_L\left[v(d_H^1) - v(d_U^1)\right] + (1-p_L)\left[v(b_H^1) - v(b_U^1)\right] \\ =\ &d(1-p_L)p_L\left(\alpha_H - \alpha_U\right)\left[v'(\varpi) - v'(\nu)\right].\end{aligned}$$

As $\alpha_H > \alpha_U$ (Lemma 1) and $\varpi < \nu$, then the concavity of $v(.)$ implies that $\Psi^{MH}(c_{min}, \bar{\Delta}) < 0$.
Moreover, Lemma 3 has shown that $\partial\Psi^{MH}(c,\Delta)/\partial c < 0$ for $c_{min}^{MH} < c < c_{max}^{MH}$. As $\Psi^{MH}(c, \bar{\Delta})$ is continuous in c, the intermediate value theorem implies that there exist $\tilde{c}_2^{MH}(\bar{\Delta}) \in]c_{min}^{MH}, c_{max}^{MH}[$ such that $\Psi^{MH}(\tilde{c}(\bar{\Delta}), \bar{\Delta}) = 0$. By continuity of $\Psi^{MH}(c,\Delta)$ in Δ, this threshold $\tilde{c}_2^{MH}(\Delta)$ also exists for value of Δ close enough to $\bar{\Delta}$. Observe that, when it exists, $\tilde{c}_2^{MH}(\Delta) < c_{max}^{MH}$ since $\Psi^{MH}(c_{max}^{MH}, \Delta) < 0$ for all Δ. From now on, we consider only values of Δ large enough that $\tilde{c}_2^{MH}(\Delta)$ exists.
b) The claim is straightforward since $\partial\Psi^{MH}(c\,,\Delta)/\partial c < 0$ by Lemma 3.
c) We have by definition that $\Psi^{MH}(\tilde{c}_2^{MH}(\Delta), \Delta) = 0$ so that

$$\frac{d\Psi^{MH}(\tilde{c}_2^{MH}(\Delta), \Delta)}{d\Delta} = \frac{\partial\Psi^{MH}(\tilde{c}_2^{MH}(\Delta), \Delta)}{\partial c}\frac{\partial\tilde{c}_2^{MH}(\Delta)}{\partial\Delta} + \frac{\partial\Psi^{MH}(\tilde{c}_2^{MH}(\Delta), \Delta)}{\partial\Delta} = 0.$$

Our claim then results from the fact that $\partial\Psi^{MH}(c\,,\Delta)/\partial c < 0$ and that $\partial\Psi^{MH}(c\,,\Delta)/\partial\Delta > 0$ for all c and Δ.

□

8.9 Proof of Proposition 5

We first prove the following two lemmatas.

Lemma 4 *We have (a)* $\tilde{c}_1^{MH}(\Delta) < \tilde{c}_1(\Delta)$ *and (b)* $\tilde{c}_2^{MH}(\Delta) < \tilde{c}_2(\Delta)$ *when* $\Delta \to \bar{\Delta}$.

Proof. Result 3 has shown that $\tilde{c}_1(\Delta) < c_{min}$ exists if $\Delta \geq \tilde{\Delta}$ (defined as $\Psi^{MH}(c_{min}, \tilde{\Delta}) = 0$), with $\Psi(c, \Delta) < 0$ for $c < \tilde{c}_1(\Delta)$. Obviously, $\Delta \to \bar{\Delta} > \tilde{\Delta}$, so that $\tilde{c}_1(\Delta)$ exists. Similarly, Result 7 shows that $\tilde{c}_1^{MH}(\Delta)$ exists for $\Delta \to \bar{\Delta}$, with $\Psi^{MH}(\tilde{c}_1^{MH}(\Delta), \Delta) = 0$. We then have that $\Psi^{MH}(c, \Delta) > \Psi(c, \Delta)$ for $c < c_{min}^{MH}$ (a consequence of Lemma 2) implies that $\tilde{c}_1^{MH}(\Delta) < \tilde{c}_1(\Delta)$.
Result 4 has shown that $c_{min} < \tilde{c}_2(\Delta) < c_{max}$ exists if $\Delta \geq \tilde{\Delta}$, with $\Psi(c, \Delta) > 0$ for $\tilde{c}_1(\Delta) < c < \tilde{c}_2(\Delta)$. Similarly, Result 8 shows that $\tilde{c}_2^{MH}(\Delta) < c_{max}^{MH}$ exists for $\Delta \to \bar{\Delta}$, with $\Psi^{MH}(c, \Delta) > 0$ for $\tilde{c}_1^{MH}(\Delta) < c < \tilde{c}_2^{MH}(\Delta)$. We then have that $\Psi^{MH}(c, \Delta) < \Psi(c, \Delta)$ for any Δ when $c_{min}^{MH} < c_{min} < c < c_{max}^{MH}$ (Lemma 5) implies that $\tilde{c}_2^{MH}(\Delta) < \tilde{c}_2(\Delta)$. ■

Lemma 5 *$\Psi^{MH}(c, \Delta) < \Psi(c, \Delta)$ for any Δ when $c_{min}^{MH} < c_{min} < c < c_{max}^{MH}$.*

Proof. Recall that, when $c_{min} < c < c_{max}^{MH}$, we have

$$\begin{aligned} \Psi^{MH}(c, \Delta) &= \lambda \left[(1 - p_H^1) v(b_H^1) + p_H^1 v(d_H^1) - c\right] + (1 - \lambda) v(c_L) - v(c_U^0), \\ \Psi(c, \Delta) &= \lambda \left[v(c_H^1) - 1\right] + (1 - \lambda) v(c_L) - v(c_U^0), \end{aligned}$$

hence we obtain

$$\Psi(c, \Delta) - \Psi^{MH}(c, \Delta) = \lambda \left[v(c_H^1) - (1 - p_H^1) v(b_H^1) - p_H^1 v(d_H^1)\right] > 0.$$

■

We now prove Proposition 5

Proof. (a) Recall that, when Δ is close enough to $\bar{\Delta}$, we have that $\Psi^{MH}(0, \Delta) = \Psi(0, \Delta)$ and that $\Psi^{MH}(c, \Delta) > \Psi(c, \Delta)$ for $c < c_{min}^{MH}$ (see Lemma 2 for both), which implies that $\Psi^{MH}(c_{min}^{MH}, \Delta) > \Psi(c_{min}^{MH}, \Delta)$. Lemma 5 shows that $\Psi^{MH}(c, \Delta) < \Psi(c, \Delta)$ for $c_{min}^{MH} < c_{min} < c < c_{max}^{MH}$. By continuity of $\Psi^{MH}(c, \Delta)$ and $\Psi(c, \Delta)$ in c, the fact that $\partial \Psi^{MH}(c, \Delta)/\partial c < 0$ for $c_{min}^{MH} < c < c_{min}$ (see Lemma 3) and the intermediate value theorem, we then have that there exists a unique value of c, denoted by $\hat{c}$, with $c_{min}^{MH} < \hat{c} < c_{min}$, and such that $\Psi^{MH}(c, \Delta) > \Psi(c, \Delta)$ for $c < \hat{c}$, $\Psi^{MH}(\hat{c}, \Delta) = \Psi(\hat{c}, \Delta)$ and $\Psi^{MH}(c, \Delta) < \Psi(c, \Delta)$ for $\hat{c} < c < c_{max}$. As for the latter inequality, observe that $\Psi^{MH}(c, \Delta) = \Psi_0 < \Psi(c, \Delta)$ for $c_{max}^{MH} \leq c < c_{max}$, while $\Psi^{MH}(c, \Delta) = \Psi(c, \Delta) = \Psi_0$ for $c \geq c_{max}$.
(b) The proof of Lemma 4 shows that $\tilde{c}_1(\Delta)$, $\tilde{c}_1^{MH}(\Delta)$ and $\tilde{c}_2^{MH}(\Delta)$ exist when $\Delta \to \bar{\Delta}$. The claim follows from the observation that $\Psi^{MH}(c, \Delta) > 0$ for $\tilde{c}_1^{MH}(\Delta) < c < \tilde{c}_2^{MH}(\Delta)$ (Results 7 and 8) while $\Psi(c, \Delta) < 0$ for for $c < \tilde{c}_1(\Delta)$ (Result 3).
(c) The proof of Lemma 4 shows that $\tilde{c}_1(\Delta)$, $\tilde{c}_2(\Delta)$ and $\tilde{c}_2^{MH}(\Delta)$ exist when $\Delta \to \bar{\Delta}$. The claim follows from the observation that $\Psi^{MH}(c, \Delta) < 0$ for $c > \tilde{c}_2^{MH}(\Delta)$ (Result 3) while $\Psi(c, \Delta) > 0$ for $\tilde{c}_1(\Delta) < c < \tilde{c}_2(\Delta)$ (Result 1 (b)).

(d) We know that $\Psi(c,\Delta)$ is maximized at $c = c_{min}$, and we now show that from $\Psi^{MH}(c^{MH}_{min},\Delta) > \Psi(c_{min},\Delta)$. We have that

$$\begin{aligned}\Psi^{MH}(c^{MH}_{min},\Delta) &= \lambda\left[(1-p^1_H)v(b^1_H)+p^1_H v(d^1_H)-(1-p^1_U)v(b^1_U)-p^1_U v(d^1_U)\right]+(1-\lambda)\left[v(c_L)-v(c^0_U)\right],\\ \Psi(c_{min},\Delta) &= \lambda\left[v(c^1_H)-v(c^1_U)\right]+(1-\lambda)\left[v(c_L)-v(c^0_U)\right],\end{aligned}$$

so that

$$\begin{aligned}&\Psi^{MH}(c^{MH}_{min},\Delta) > \Psi(c_{min},\Delta)\\ \Leftrightarrow\quad & v(c^1_U)-\left[(1-p^1_U)v(b^1_U)-p^1_U v(d^1_U)\right] > v(c^1_H)-\left[(1-p^1_H)v(b^1_H)+p^1_H v(d^1_H)\right].\end{aligned}$$

If we assume that $\Delta = \bar{\Delta}$, the latter inequality becomes

$$\begin{aligned}(1-p_L)\left[v(b^1_U)-v(b^1_H)\right] &< p_L\left[v(d^1_H)-v(d^1_U)\right]\\ (1-p_L)\left[b^1_U-b^1_H\right]v'(\alpha) &< p_L\left[d^1_H-d^1_U\right]v'(\beta),\end{aligned}\tag{11}$$

with $\alpha > \beta$. Using

$$\begin{aligned}b^1_U-b^1_H &= (\alpha_H-\alpha_U)p_L d,\\ b^1_U-b^1_H &= (\alpha_H-\alpha_U)p_L d,\end{aligned}$$

the inequality (11) becomes

$$v'(\alpha) < v'(\beta),$$

which is true.

By continuity of $\Psi^{MH}(c^{MH}_{min},\Delta)$ and of $\Psi(c_{min},\Delta)$ in Δ, we obtain that $\Psi^{MH}(c^{MH}_{min},\Delta) > \Psi(c_{min},\Delta)$ for $\Delta \to \bar{\Delta}$.

■

Figure 1 : Value of test as a function of c for several values of Δ

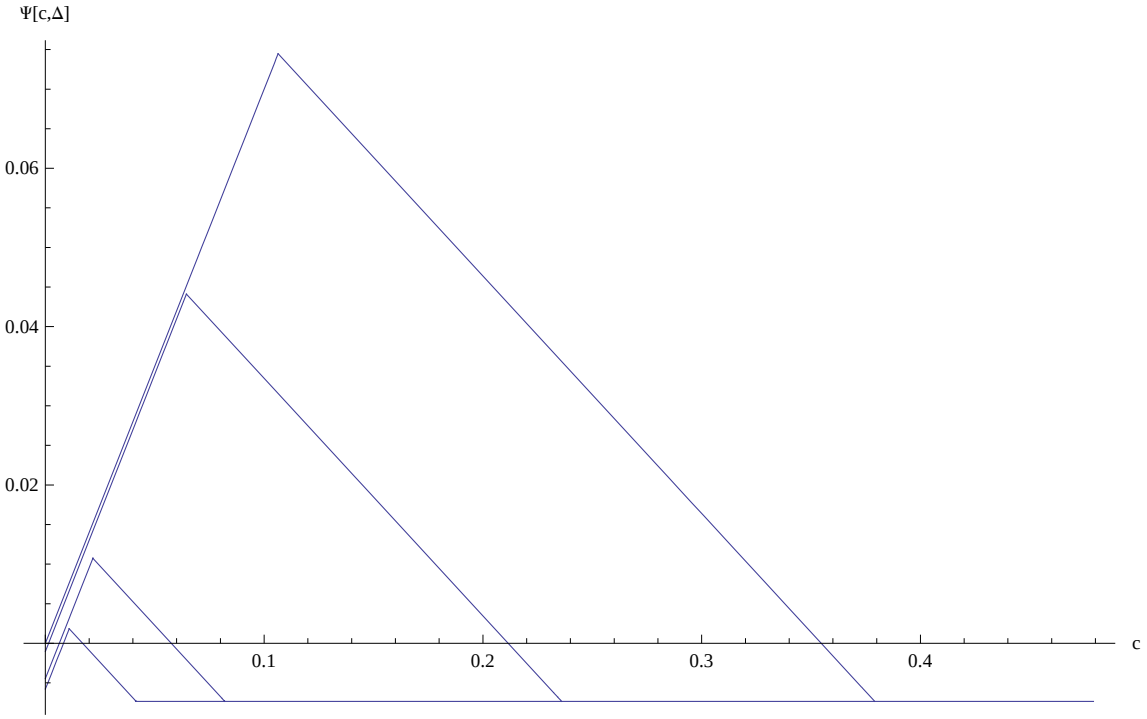

Figure 2 : Value of c~1 (yellow), cmin (blue), c~2 (green) and cmax (purple) as a function of Δ

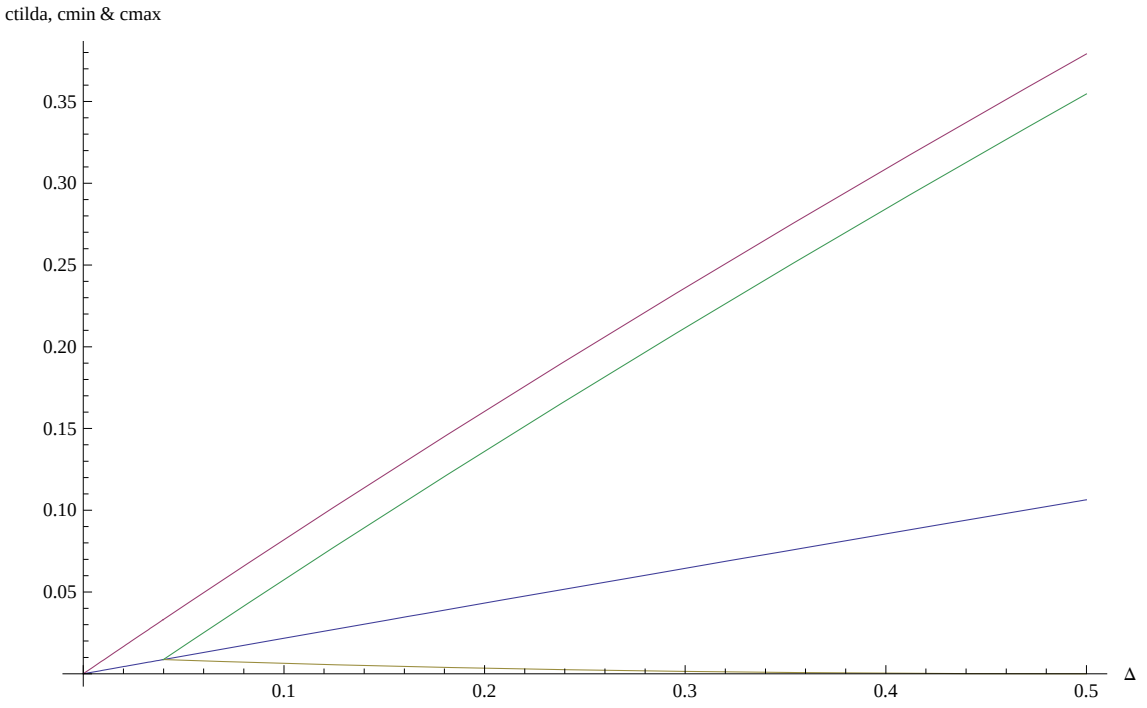

Figure 3 : αH and αU (αU < αH) as a function of c

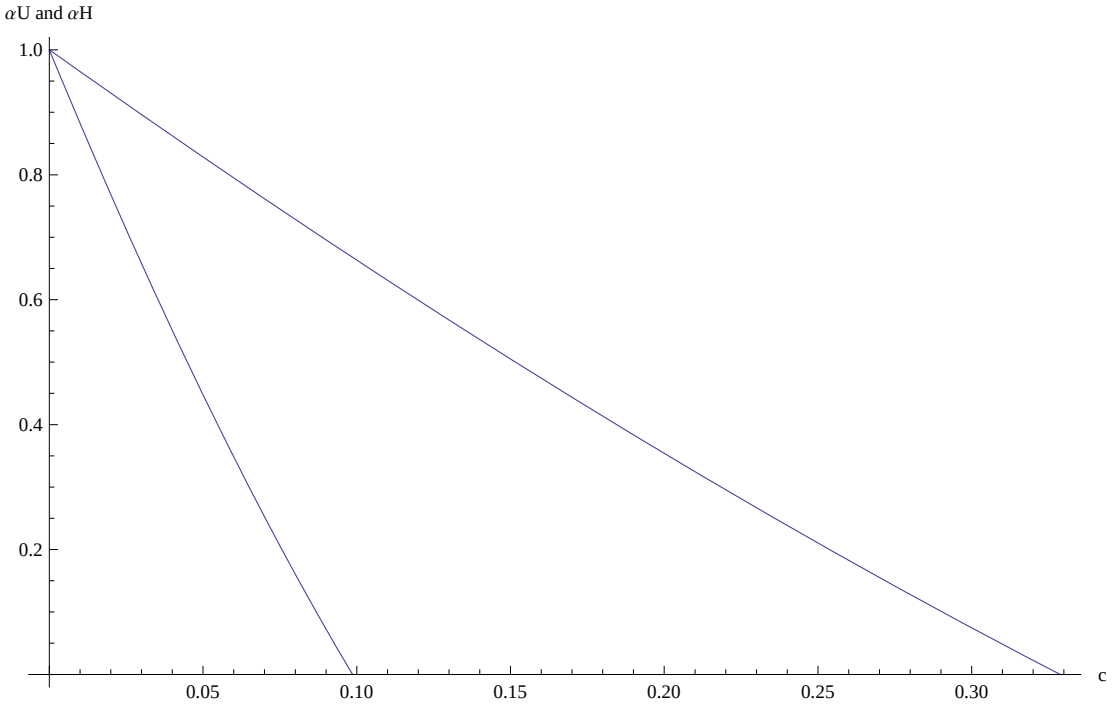

Figure 4 : Value of test with moral hazard as a function of c for several values of Δ

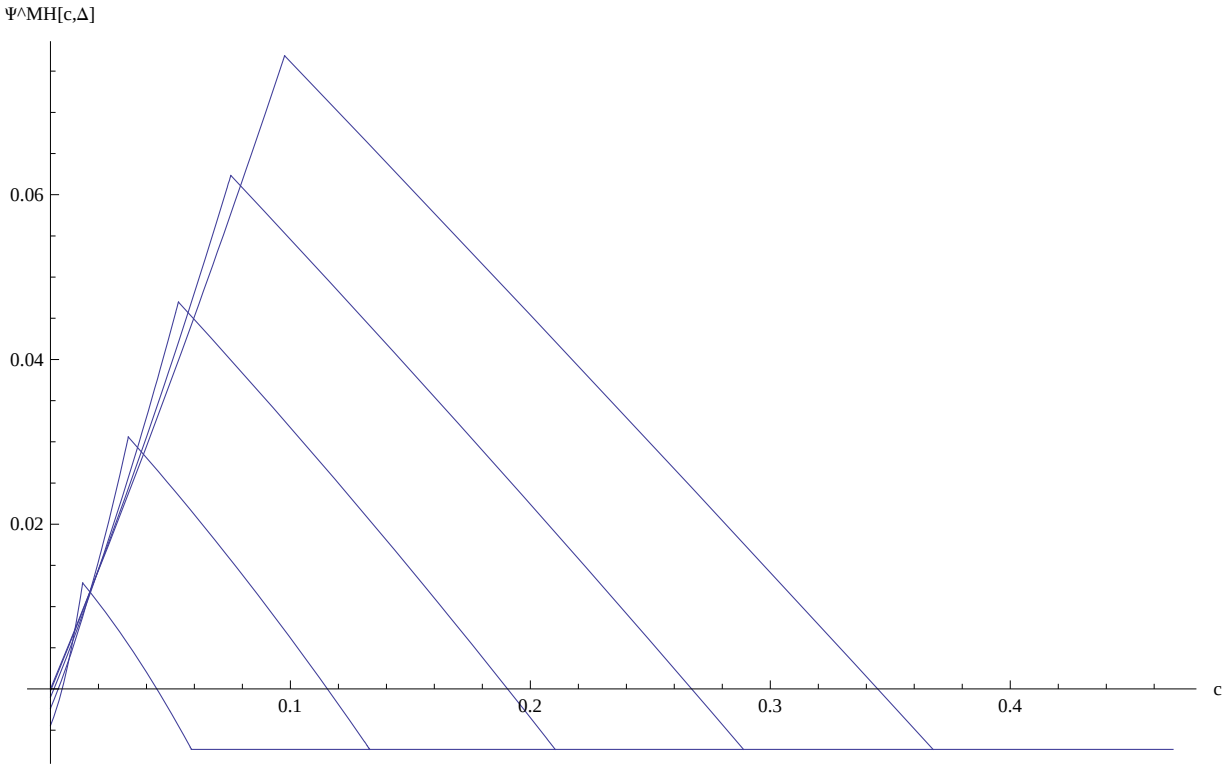

Figure 5 : Comparison of value of test, with (right) and without MH (left), as a function of c for Δ = 0.1

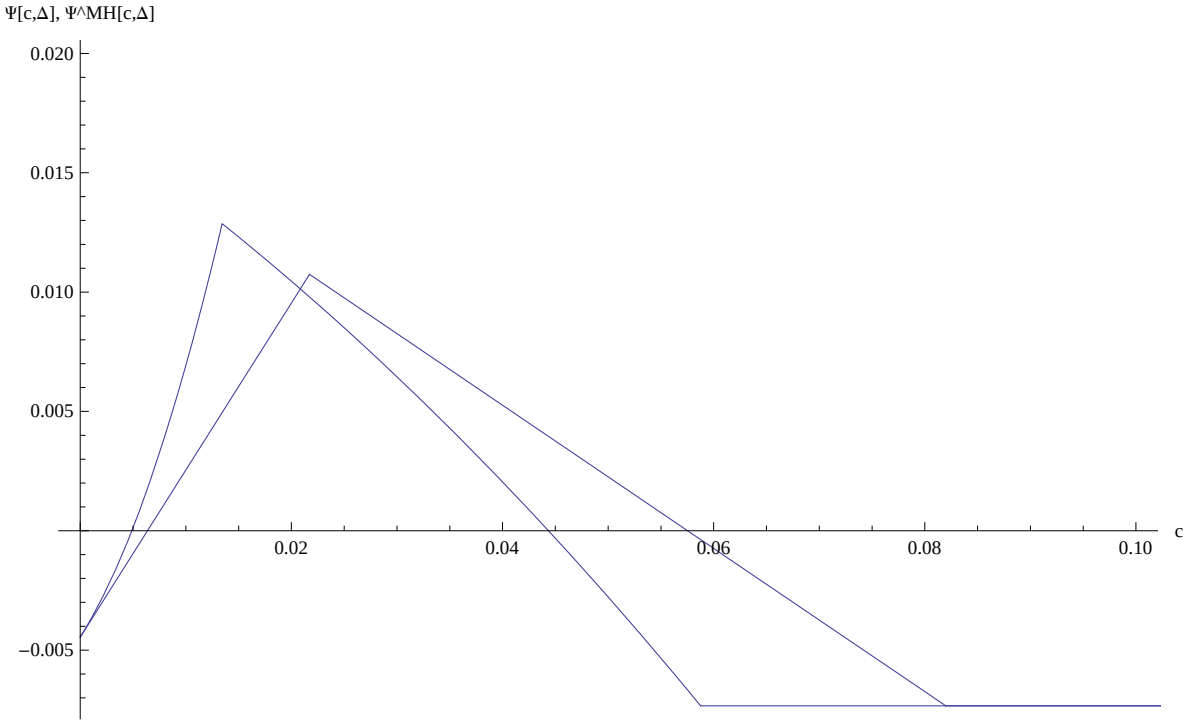

Figure 6 : Ex-ante utility as a function of effort cost

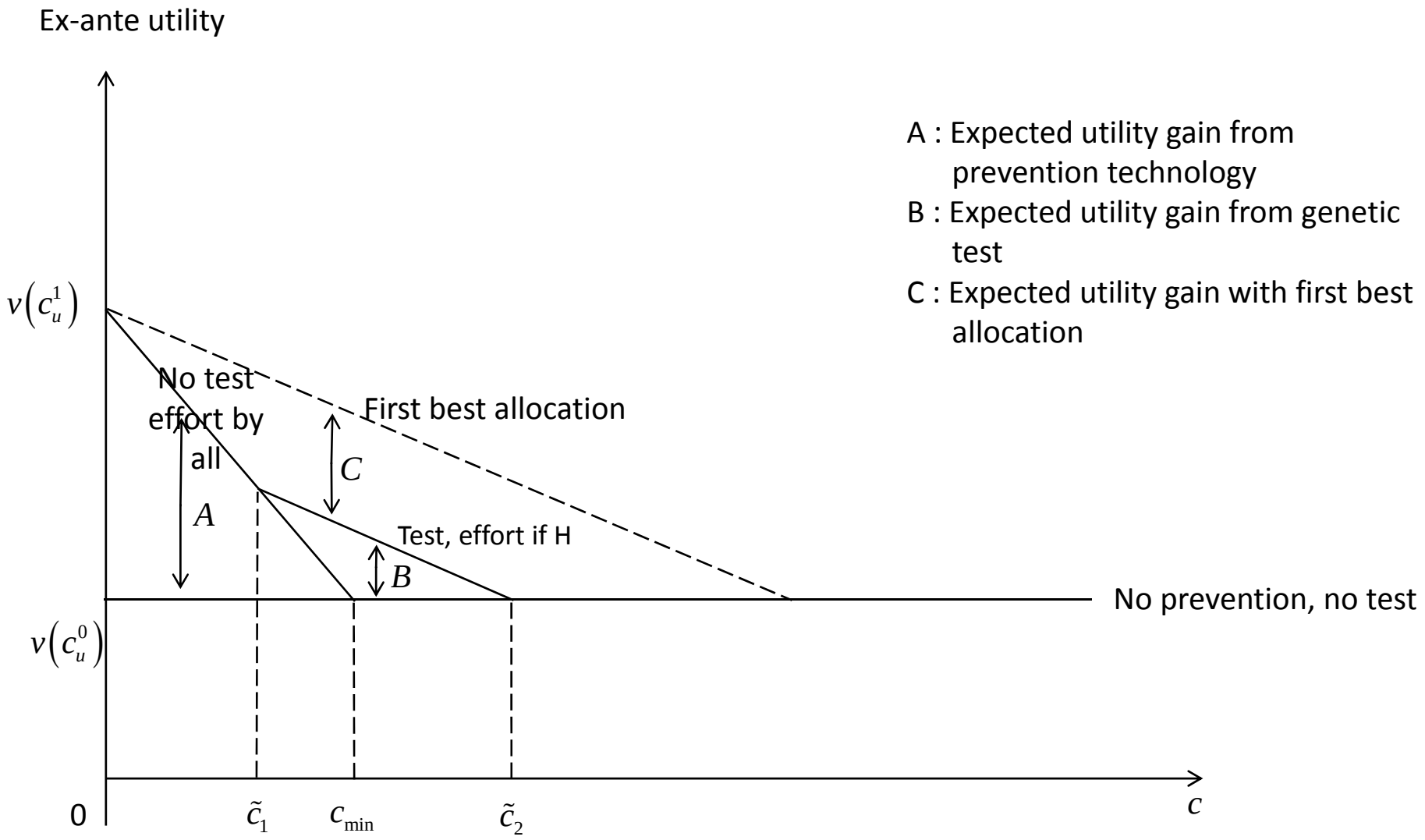

Figure 7 : Ex-ante utility with and without moral hazard