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Genetic Testing with Primary Prevention and Moral Hazard¹

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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. Testing is not mandatory, but agents have to reveal their test results to the insurers, facing a discrimination risk. A costly prevention effort allows agents with a genetic predisposition to decrease their probability to develop the disease. 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 small. 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 medicine.

1 Introduction

There is evidence that, for many important health risks, individuals differ significantly in how much they would benefit from prevention. Sizeable welfare gains would be reaped by identifying the individual characteristics associated with a larger efficiency of prevention. One way to uncover those characteristics is through genetic tests. The main thesis of “personalized medicine” (see Collins [2010] and Davies [2010] among others) is precisely that genetic testing provides cheap and reliable information on health risks that can be used to individually tailor prevention. “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” (p. 802).¹ There is actually a whole range of such prevention activities: “institution of drug therapies; (...) special diets; (...) surgery or other options” (p. 815).

The objective of our paper is to assess the impact of a (genetic) test on both the private health insurance market and on welfare. More precisely, we aim at understanding under what circumstances such a test would be voluntarily taken, 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 welfare.

We develop a model where a fraction of agents have a higher probability of developing a disease than the rest of society. People are born uninformed about their individual probability level, but can undertake a (genetic or other²) test in order to assess (without any error) whether they have a low or high disease probability (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 costly (primary) prevention effort in order to decrease the probability of occurrence of the disease. We assume that prevention reduces the probability of illness only for individuals whose genes predispose them to the disease. 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 (p. 1853). One reason why prevention effort may be efficient only if an individual has a positive test 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” (p. 1098). For instance, 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)” (p. 1169).

¹All quotations in the Introduction are taken from Collins(2010), the page numbers refer to the kindle edition.

²Alternatively, the “test” could be an exploration of family history, which Collins (2010, p. 1084) indeed dubs a “free genetic test”.

We study perfect competition between profit-maximizing health insurers who cannot force individuals to undertake the test, and/or the prevention effort, but who do observe the tests results. 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. 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 means abandoning the sure pay-off associated with the pooling insurance contract (based on average probability) developed for uninformed agents for an actuarially fair lottery between the (separating) contracts devised for agents with low and high probabilities. Risk-averse agents dislike this so-called discrimination risk and would never undertake a test that would not allow them to better adapt their prevention effort to their individual circumstances.

The availability of a prevention strategy tailored for high risk agents gives stronger incentives to undertake the test. Whether such individuals make the prevention effort and thus decrease their disease probability 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 then cover the two cases in the paper. 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 disease probability of someone who is genetically predisposed to the disease.

We first study the benchmark situation where the effort is observable, verifiable and contractible by the insurers. Our main result is that agents undertake the test only for intermediate values of the cost of prevention effort. When this cost is low, uninformed people make the prevention effort, so that a (negative) genetic test allows insurees to forego the (cost of the) prevention effort. 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 obtain a positive test. The test value is then *decreasing* in the effort cost. Finally, when the effort cost is high, even a positive test does not induce agents to undertake prevention, so that the discrimination risk translates into a negative test value. As is intuitive, the value of the test increases with the efficiency of the prevention effort, whatever its cost.

We then turn to the case where effort is not observable. Insurers face a moral hazard problem that they solve by offering partial coverage, since full coverage induces agents not to provide any effort.³ A naïve intuition would suggest that this under-provision,

³For instance, Dave and Kaestner (2006) “find evidence that obtaining health insurance reduces

by decreasing the utility level with prevention, also reduces the value of the test. This is indeed the case for intermediate values of the effort cost, when the effort is undertaken only in the case of a (positive) test. But this intuition is misleading when the effort cost is low enough that prevention is undertaken by uninformed agents. In that case, the value of the test is actually larger with than without moral hazard, because moral hazard decreases utility more when the test is not taken (and effort is provided) than when it is taken (and effort is provided only in the case of a positive test). The driving force behind this result is that insurers have to ration coverage more to uninformed agents than to those with a positive test in order to induce them to make the effort. Comparing further the cases with and without moral hazard, we also 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.

Finally, we assess the impact of the various assumptions of our model on welfare and provide several policy recommendations regarding the desirability of targeted genetic tests, of policies to increase prevention by all, of different ways to eliminate the discrimination risk associated to testing, and of enlarging the disclosure duty to the prevention effort.

Related literature

The seminal paper by Hirshleifer (1971) establishes the negative value of the information brought by a test when health risk is exogenous and when individuals face a discrimination risk. The information value may become positive if insurers do not observe the consumers' information status (Doherty and Thistle, 1996), if agents exhibit a "repulsion from chance" (Hoel *et al.*, 2006) or if insurance embodies redistribution (Rees and Apps, 2006).

Several papers have added prevention choice to this setting. 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 Henriët (2011) and Crainich (2011). Barigozzi and Henriët (2011) compare several regulatory approaches used in practice, from *laissez-faire* to the prohibition of tests. They show that policyholders are better off under the "disclosure duty" regulation studied in our paper, because this regulation does not create an adverse selection problem while allowing insurees to use the information provided by the test to self-insure against the damage.⁴ 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 provides conditions ensuring that

prevention and increases unhealthy behaviors among elderly men."

⁴Hoy and Polborn (2000) and Strohmeier and Wambach (2000) also study the impact of genetic tests on the health insurance market in the presence of adverse selection.

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 Doherty 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 disease probability thanks to primary prevention actions, but we differ when they assume that uninformed policyholders never undertake prevention. Also, Hoel and Iversen (2002) considers both compulsory and voluntary (supplementary) health insurance.

The main difference between our paper and the articles above is that we assume that primary prevention (especially 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 solved by providing partial insurance coverage, and thus affects the value of the genetic test.⁵

2 Setting and notation

The economy is composed of a unitary mass of individuals. Each individual develops a disease with some probability, with sickness modeled as the occurrence of a monetary damage of amount d . 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^0 (with $0 < p_L^0 < p_H^0 < 1$). Therefore, the average probability of illness in society is given by $p_U^0 = \lambda p_H^0 + (1 - \lambda)p_L^0$.

Individuals are uninformed of their type unless they take a genetic test. The test is assumed to be costless and perfect, revealing with certainty the individual true type, L or H .⁶ Individuals choose whether to test and also whether to exert some primary prevention effort. We assume that the prevention decision is binary and that the effort cost (normalized to zero if no effort is undertaken) ϕ is measured in utility terms. The assumption of a utility (rather than monetary) cost fits better behavior modification efforts (the type of prevention that is the most difficult to observe for insurers) and is innocuous in our binary setting.⁷ We further assume that prevention has no effect

⁵A recent exception is the paper by Filipova and Hoy (2009), which focuses on the moral hazard risk of over-consumption of surveillance when financial costs are absorbed by the insurance pool. They concentrate on the consequences of information on prevention, while we endogenize both the prevention and testing decisions.

⁶With a slight abuse of terminology, we denote an uninformed agent as having type U .

⁷This would not be true if the prevention choice were continuous: a monetary cost would then generate income effects (with the well known complications detailed in Dionne and Eeckhoudt [1985])

for a low probability individual, while it decreases the disease probability of type H individuals to p_H^1 , with $p_H^0 > p_H^1 \geq p_L^1 = p_L^0 = p_L$, where p_j^i denotes the disease probability of an agent of type $j \in \{L, H, U\}$ who is (resp., is not) making a prevention effort, denoted by $i = 1$ (resp., $i = 0$). We capture the prevention efficiency through $\Delta = p_H^0 - p_H^1$. The parameter Δ can take any value between zero (prevention has no impact, $p_H^1 = p_H^0$) and $\bar{\Delta} = p_H^0 - p_L$ (prevention decreases the probability of a type H agent to the level of a low probability agent, $p_H^1 = p_L$). The two characteristics of the prevention technology, its cost ϕ and efficiency Δ , will play an important role in our analysis.

The timing of the model consists in five sequential stages: (1) a competitive fringe of insurers offer insurance contracts, (2) agents decide whether to take the test or not, and observe its result, (3) they choose one insurance contract (4) they exert or not some prevention effort and (5) the damage occurs or not, and the corresponding payoffs are realized.

We compute and compare the equilibrium allocations depending upon what is observed by the insurers. Section 3 studies the case where the prevention effort is observable and contractible by insurers. Section 4 assumes that effort is not observable/contractible, so that insurers face a moral hazard problem. Section 5 compares the results obtained in the two previous sections to shed light on 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 start by describing the contracts offered by the insurers, and then move to the individuals' decisions of whether to test and to make a prevention effort.

3.1 Contracts offered by the insurers

Competition forces insurers to offer actuarially fair contracts, so that individuals favor full insurance. The insurance contract devised for an agent of type $j \in \{L, H, U\}$ as a function of his effort decision ($i \in \{0, 1\}$) is denoted by (π_j^i, I_j^i) and characterized by a

that are assumed away in our formulation.

premium in case of health,

$$\pi_j^i = p_j^i d,$$

and an indemnity (net of the premium) in case of sickness,

$$I_j^i = (1 - p_j^i) d.$$

The expected utility of an agent buying the contract devised for his type j and effort decision i is

$$\begin{aligned} U_j^i &= (1 - p_j^i)v(y - \pi_j^i) + p_j^i v(y - d + I_j^i) - \phi^i \\ &= v(y - p_j^i d) - \phi^i \equiv v(c_j^i) - \phi^i, \end{aligned}$$

where $v(\cdot)$ is a classical von Neumann Morgenstein utility function ($v'(\cdot) > 0$, $v''(\cdot) < 0$) with y the individual's exogenous income, c_j^i his consumption level, and where $\phi^1 = \phi$ while $\phi^0 = 0$.

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 only contracts offered to type L agents entail no prevention effort. Insurers may then offer at most five contracts.

We now turn to the choice of a contract by the agent, *i.e.* whether they take the test and perform some prevention.

3.2 Prevention and Health Insurance Decisions

We proceed backwards and first look at the prevention decision of agents who have taken the test. These agents know with certainty 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^0, I_L^0) giving them a utility level of U_L^0 . Agents of type H have the choice between two contracts (with and without effort) and buy the contract with effort provided that

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

This condition imposes an upperbound on the cost of effort. Observe that, if this condition is satisfied, then no insurance firm proposes the contract (π_H^0, I_H^0) at equilibrium. If one firm were to do so, then another firm would propose the contract $(\pi_H^1, I_H^1 - \varepsilon)$ with ε small, would attract all type H agents, and would make a strictly positive profit.

We now look at agents who have not taken 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 if

$$\begin{aligned} U_U^1 &> U_U^0, \\ \Leftrightarrow \phi < \phi_{min} &\equiv v(c_U^1) - v(c_U^0). \end{aligned}$$

The same reasoning as above shows 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 $\phi_{min} < \phi_{max}$ if $\Delta > 0$. First, effort is always effective if type H but not always if type U , with the same cost in both cases. Second, type H individuals pay a higher premium than type U agents, so that their marginal utility is higher. Type H thus gain more than average type U from the lower premium made possible by the prevention effort. 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:

Result 1 *The prevention decision of agents depends on the effort cost ϕ as follows:*

- a) $\phi < \phi_{min}$: *uninformed individuals and agents with a positive test undertake effort,*
- b) $\phi_{min} < \phi < \phi_{max}$: *only agents with a positive test undertake effort,*
- c) $\phi > \phi_{max}$: *no one undertakes effort.*

We proceed backward and solve the testing decision of agents.

3.3 To test or not to test

Whether taking the test is worth its while depends on the value of ϕ , which determines under what circumstances an individual makes a prevention effort. We cover in turn the three cases covered by Result 1. In all cases, we define as the value of the test, denoted by $\Psi(\phi, \Delta)$, 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). An individual takes the test if and only if its value is positive.

3.3.1 No one undertakes prevention: $\phi \geq \phi_{max}$

Result 2 *When $\phi \geq \phi_{max}$, $\Psi(\phi, \Delta) \equiv \Psi_0 < 0$, $\forall(\phi, \Delta)$ so that the test is not taken.*

This is the well known (Hirshleifer, 1971) result of the negative value of a genetic test, whose results are observable and contractible, in the absence of prevention. 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 . By not taking the test, the

individual obtains a sure payoff (since he is perfectly insured) at an actuarially fair rate. Any risk averse agent hence prefers the sure and actuarially fair payoff to the lottery. This drawback of the test is called the discrimination risk. Observe that Ψ is independent of both the cost and effectiveness of prevention, as long as the cost ϕ is larger than the threshold ϕ_{max} .

We now move to the polar case where effort is undertaken even when individuals do not test.

3.3.2 Types U and H undertake prevention: $\phi \leq \phi_{min}$

The value of the test is given by

$$\begin{aligned}\Psi(\phi, \Delta) &= \lambda U_H^1 + (1 - \lambda)U_L^0 - U_U^1 \\ &= (1 - \lambda)\phi - [v(y - p_U^1 d) - (\lambda v(y - p_H^1 d) + (1 - \lambda)v(y - p_L d))].\end{aligned}\quad (2)$$

The first term in (2) measures the gain from the test, which allows agents to forgo the prevention effort cost ϕ if the test proves negative (*i.e.*, with probability $1 - \lambda$) while the terms between brackets represent the drawback from taking the test, *i.e.* the discrimination risk.

We then obtain

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

The value of the test increases both with the cost of prevention effort (since a negative test allows the testee *not* to make the effort) and with its efficiency (a larger Δ moves p_H^1 closer to p_L and reduces the discrimination risk). When the efficiency of prevention is low, the value of the test remains negative for all values of $\phi \leq \phi_{min}$: the gain from taking the test is too low to compensate agents for the attached discrimination risk. When Δ is high enough, Ψ becomes positive provided that the effort cost is large enough. Formally, we identify thresholds on effort efficiency, $\tilde{\Delta}$, and cost, $\tilde{\phi}_1$, 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 ϕ as Δ increases. When Δ reaches $\bar{\Delta}$, the value of the test is positive for all values of $\phi \leq \phi_{min}$.

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

3.3.3 Only type H agents undertake prevention: $\phi_{min} < \phi < \phi_{max}$

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

$$\begin{aligned}\Psi(\phi, \Delta) &= \lambda U_H^1 + (1 - \lambda)U_L^0 - U_U^0, \\ &= \lambda(v(y - p_H^1 d) - \phi) + (1 - \lambda)v(y - p_L d) - v(y - p_U^0 d).\end{aligned}\quad (3)$$

Taking the test results in the agents making the prevention effort whenever their test is positive. Equation (3) shows that the discrimination risk associated with testing is mitigated by the lower premium, thanks to prevention, when the test is positive. The value of the test increases with prevention efficiency Δ , but decreases with the cost of effort ϕ . This latter result is in stark contrast with the one obtained when even uninformed types undertake prevention.

Result 4 *When $\phi_{min} \leq \phi < \phi_{max}$, the value of the test is positive provided that the prevention efficiency Δ is large while the effort cost ϕ is small. Formally,*

- a) *for all $\Delta \geq \tilde{\Delta}$ (as defined in Result 3), there exists a unique value of ϕ , denoted by $\tilde{\phi}_2(\Delta)$, such that $\phi_{min} \leq \tilde{\phi}_2(\Delta) < \phi_{max}$ and $\Psi(\tilde{\phi}_2(\Delta), \Delta) = 0$;*
- b) *$\Psi(\phi, \Delta) > 0$ for all $\Delta > \tilde{\Delta}$ and $\phi_{min} < \phi < \tilde{\phi}_2(\Delta)$;*
- c) *for all $\Delta \geq \tilde{\Delta}$, $\tilde{\phi}_2(\Delta)$ increases with Δ ;*
- d) *$\tilde{\phi}_1(\tilde{\Delta}) = \tilde{\phi}_2(\tilde{\Delta}) = \phi_{min}$ and $\phi_{min} < \tilde{\phi}_2(\bar{\Delta}) < \phi_{max}$.*

The value of the test is positive provided that prevention is sufficiently effective (same threshold $\tilde{\Delta}$ as in Result 3) and that the cost of effort is not too large. As effectiveness increases, the threshold cost $\tilde{\phi}_2$ below which the value of the test is positive increases, so that the test is undertaken for larger values of ϕ .

To summarize, when effort is observable and contractible by insurers, the genetic test is undertaken at equilibrium provided that the prevention efficiency is large enough ($\Delta > \tilde{\Delta}$) while its cost takes intermediate values ($\tilde{\phi}_1(\Delta) < \phi < \tilde{\phi}_2(\Delta)$). The interval of effort costs compatible with test taking enlarges as prevention efficiency increases. Figure 1 provides a graphical illustration of the value of the test as a function of the 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

Figure 2 depicts the thresholds $\tilde{\phi}_1$, ϕ_{min} , $\tilde{\phi}_2$ and ϕ_{max} as functions of Δ . With this numerical example, the value of $\tilde{\Delta}$ is 0.04. The area between the curves $\tilde{\phi}_2(\Delta)$ and $\tilde{\phi}_1(\Delta)$ 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 (ϕ, Δ) located below the ϕ_{min} and $\tilde{\phi}_1(\Delta)$ curves are such that everyone makes the prevention effort, while combinations above the ϕ_{min} and $\tilde{\phi}_2(\Delta)$ curve are such that no prevention effort is made by anyone.

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

Insurers now face a moral hazard problem, since the prevention effort has to be induced 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

Contracts without prevention effort, (π_j^0, I_j^0) , $j \in \{L, H, U\}$, are unchanged, compared to the previous section. We then consider the contract (π_j^1, I_j^1) offered to a type $j = \{H, U\}$ who the insurer would like to induce to make an effort. For such an individual to make an effort, the following incentive compatibility (IC hereafter) constraint must be satisfied:

$$(1 - p_j^1)v(b_j^1) + p_j^1v(d_j^1) - \phi \geq (1 - p_j^0)v(b_j^1) + p_j^0v(d_j^1), \quad (4)$$

where b_j^1 and d_j^1 denote the consumption level of a type $j = \{H, U\}$ buying the (π_j^1, I_j^1) contract in case they are lucky and in case the damage occurs, respectively—*i.e.*, $b_j^1 = y - \pi_j^1$ and $d_j^1 = y - d + I_j^1$.

The IC constraint (4) states that the individual, when buying the contract (π_j^1, I_j^1) , is at least as well off making an effort (the LHS of (4)) than pretending to make one (the RHS of (4)). This IC constraint is incompatible with the provision of full coverage, since in that case consumption levels are equalized across states of the world ($b_j^1 = d_j^1$). As

pointed out by Shavell (1979), the only way for the insurer to induce effort making is to restrict the coverage offered to the individual (the competition between insurers ensures that the contracts remain actuarially fair). We rewrite the contracts as $\pi_j^1 = \alpha_j p_j^1 d$ and $I_j^1 = \alpha_j (1 - p_j^1) d$, where α_j is the (maximum) coverage rate offered to individuals of type $j = \{H, U\}$ in order to induce them to make an effort. The value of α_j is implicitly obtained by solving the IC constraint (4) with equality, and we obtain

$$\phi = \Delta(v(b_H^1) - v(d_H^1)), \quad (5)$$

for $j = H$ and

$$\phi = \lambda \Delta(v(b_U^1) - v(d_U^1)), \quad (6)$$

for $j = U$. The IC constraint equalizes the cost and benefit of effort making when buying the contract (π_j^1, I_j^1) , the latter being the product of the efficiency of the prevention effort (Δ and $\lambda \Delta$ in the case of types H and U , respectively) and the utility gap between the two states of the world (sick or healthy). We have that $b_j^1 > d_j^1$: the insured is better off if the damage does not occur, which gives him the exact incentive needed to support the prevention effort cost ϕ .

We obtain the following useful lemma.

Lemma 1 a) $\alpha_U < \alpha_H < 1$.

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

Two effects push 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 type U , since for the latter there is a probability $1 - \lambda$ that the 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 . Also, as the cost of effort increases, insurers have to increase this utility gap, 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, $\bar{\phi}_i$, compatible with inducing prevention effort for type i . Intuitively, this maximum prevention cost $\bar{\phi}_i$ is lower for type U (when effort works with probability λ) than for type H .⁸

⁸The benefit of prevention (the RHS of (5) and (6)) 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. The non monotonic relationship between the prevention efficiency Δ and the level of coverage α in *ex ante* moral hazard models has been pointed out in Bardey and Lesur (2005).

Figure 3 illustrates Lemma 1 for our numerical example.

Insert Figure 3 around here

We now proceed backward, starting with the prevention choice of agents.

4.2 The choice of prevention

An individual of type H chooses the contract inducing effort (with the corresponding expected utility level denoted by U_H^{1MH}) rather than (π_H^0, I_H^0) if⁹

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

Likewise, the condition under which it is optimal for an uninformed individual to exert a prevention effort is

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

The following result parallels Result 1.

Result 5 *Uninformed agents undertake the effort provided that $\phi \leq \phi_{min}^{MH} < \min[\phi_{min}, \bar{\phi}_L]$, while type H agents do so provided that $\phi \leq \phi_{max}^{MH} < \min[\phi_{max}, \bar{\phi}_H]$.*

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. Introducing moral hazard then degrades the utility obtained by effort-making agents, decreasing the maximum values of the effort cost compatible with exercising prevention.

Moving backward again, we study the incentive to take the genetic test.

4.3 To test or not to test

The value of the test depends on whether effort is undertaken at equilibrium *-i.e.*, on how ϕ compares with ϕ_{min}^{MH} and ϕ_{max}^{MH} . In the case where $\phi \geq \phi_{max}^{MH}$, we obtain that $\Psi^{MH}(\phi, \Delta) = \Psi(\phi, \Delta) = \Psi_0 < 0$ (since we are back to the case where no prevention effort is undertaken), so that the test is not taken. We now consider the other two possibilities.

⁹Recall that, at equilibrium, competition among insurers ensures that only the utility-maximizing contract (given the observability constraints) is offered to types $j = \{H, U\}$.

4.3.1 Types U and H undertake prevention: $\phi \leq \phi_{min}^{MH}$

Individuals take the test if

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

We first present the following lemma.

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

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

As was already the case when effort was observable (see (2)), raising the cost of effort increases the value of the test by the probability that the test is negative (and the effort useless), $1 - \lambda$. With moral hazard, insurers moreover react to a larger cost of effort by decreasing the coverage rates offered to both types U and H agents, thereby decreasing the utility levels they both attain. It is unclear in general how the *difference* of utility levels between types U and H varies with these lower coverage rates, because those types differ both in coverage ($\alpha_U < \alpha_H$) and in probability ($p_H^1 \geq p_U^1$). When $\Delta \rightarrow \bar{\Delta}$, the probabilities 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, $d_U^1 < d_H^1 < c_H^1 < c_U^1 < b_H^1 < b_U^1$), 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.

We then obtain the following result.

Result 6 *When $\phi \leq \phi_{min}^{MH}$, the value of the test is positive provided that the prevention efficiency Δ and the effort cost ϕ are large enough. Formally, assume that Δ is large enough. We then have that*

$$\begin{aligned} a) \text{ there exists a (unique) value of } \phi, \text{ denoted by } \tilde{\phi}_1^{MH}(\Delta), \text{ such that } \tilde{\phi}_1^{MH}(\Delta) < \phi_{min}^{MH} \\ \text{and } \Psi^{MH}(\tilde{\phi}_1^{MH}(\Delta), \Delta) = 0. \text{ Moreover, } \tilde{\phi}_1^{MH}(\bar{\Delta}) = 0; \\ b) \Psi^{MH}(\phi, \Delta) < 0 \text{ for } \phi < \tilde{\phi}_1^{MH}(\Delta) \text{ and } \Psi^{MH}(\phi, \Delta) > 0 \text{ for } \phi > \tilde{\phi}_1^{MH}(\Delta). \end{aligned}$$

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 $\phi \leq \phi_{min}^{MH}$ and $\Delta \rightarrow \bar{\Delta}$, so that we can identify a threshold effort cost level above (respectively, below) which agents do (resp., do not) undertake the test.¹⁰ Observe that Result 6 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 small enough values of ϕ . This weaker statement is due to the fact that, with moral hazard, the value of the test may not always increase with Δ , because the utility of an uninformed 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.

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

4.3.2 Only type H agents undertake prevention: $\phi_{min}^{MH} < \phi < \phi_{max}^{MH}$

The value of the test in that case is

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

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

Lemma 3 For $\phi_{min}^{MH} < \phi < \phi_{max}^{MH}$, we have that

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

With intermediate values of ϕ , prevention efficiency affects the value of the test only through its impact on the utility level attained by type H agents. This impact is twofold. The direct impact of a larger Δ lowers both the disease probability and the premium, for a given coverage level α_H , and thus increases type H utility. The indirect impact is ambiguous, since recall from footnote 8 that Δ may either decrease or increase α_H . We show in the proof of Lemma 3 that, even if α_H decreases with Δ , the direct impact is larger than the indirect one, so that the value of the test always increases with Δ when $\phi_{min}^{MH} < \phi < \phi_{max}^{MH}$. 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 for a given insurance contract, while the indirect impact of ϕ on the insurance contract

¹⁰We will compare the threshold costs with and without moral hazard in section 5.

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 ϕ and Δ on the value of the test is the same as without moral hazard. 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 ϕ and Δ on the value of the test ambiguous in general.

We then obtain the following result.

Result 7 *When $\phi_{min}^{MH} \leq \phi < \phi_{max}^{MH}$, the value of the test is positive provided that the prevention efficiency Δ is large while the effort cost ϕ is small. Formally, assume that Δ is large enough. We then have that*

- a) *there exists a unique value of ϕ , denoted by $\tilde{\phi}_2^{MH}(\Delta)$, such that $\phi_{min}^{MH} \leq \tilde{\phi}_2^{MH}(\Delta) < \phi_{max}^{MH}$ and $\Psi^{MH}(\tilde{\phi}_2^{MH}(\Delta), \Delta) = 0$. Moreover, $\phi_{min}^{MH} < \tilde{\phi}_2^{MH}(\bar{\Delta}) < \phi_{max}^{MH}$;*
- b) *$\Psi^{MH}(\phi, \Delta) > 0$ for $\phi_{min}^{MH} < \phi < \tilde{\phi}_2^{MH}(\Delta)$;*
- c) *$\tilde{\phi}_2^{MH}(\Delta)$ increases with Δ .*

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.

Proposition 1 *Individuals take the test if the efficiency of prevention is large enough and the prevention cost takes intermediate values: $\tilde{\phi}_1^{MH}(\Delta) \leq \phi \leq \tilde{\phi}_2^{MH}(\Delta)$. Moreover, the threshold $\tilde{\phi}_2^{MH}(\Delta)$ increases with Δ .*

The main difference with results obtained without moral hazard is due to the fact that, as we have underlined in section 4.3.1, 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 cost also. This also prevents us from assessing how the lowest prevention cost compatible with taking the test varies with 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 high enough, and that prevention costs take intermediate values.

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

The same caveats apply as for Proposition 1, 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 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 ϕ . This complements nicely our analytical finding of Lemma 2 that the slope of Ψ^{MH} is larger than $1 - \lambda$ when $\Delta \rightarrow \bar{\Delta}$. The curve Ψ^{MH} is then (as proved in Lemma 3) decreasing in ϕ until it reaches Ψ_0 for $\phi > \phi_{max}^{MH}$. Finally, a striking characteristic of Figure 4 is that $\Psi^{MH}(\phi_{min}^{MH}, \Delta)$ is increasing in Δ : although a larger prevention efficiency does not increase the value of the test for all values of ϕ 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, ϕ . We assume that Δ is close to $\bar{\Delta}$, and that $\phi_{min}^{MH} < \phi_{min} < \phi_{max}^{MH} < \phi_{max}$ (the case where

$\phi_{min}^{MH} < \phi_{max}^{MH} < \phi_{min} < \phi_{max}$ can be treated similarly and does not bring any new insight, so we leave it to the reader).

We then obtain the following proposition.

- Proposition 3** *Assume that Δ is large enough (close but not equal to $\bar{\Delta}$). Then*
- (a) *there exists a threshold $\phi_{min}^{MH} < \phi < \phi_{min}$ 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 $\tilde{\phi}_1^{MH}(\Delta) < \phi < \min[\tilde{\phi}_1(\Delta), \tilde{\phi}_2^{MH}(\Delta)]$, the value of the test is positive with moral hazard but negative without: agents take the test if and only if there is moral hazard;*
 - (c) *for $\max[\tilde{\phi}_1(\Delta), \tilde{\phi}_2^{MH}(\Delta)] < \phi < \tilde{\phi}_2(\Delta)$, the value of the test is 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 with moral hazard than without:*

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

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 disease probability. 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 direct 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. 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 when the test is 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 when Δ is large enough (see Result 6). Similarly, part (c) establishes that, for higher values of the

effort cost (such that the value of the test is lower with than without moral hazard), agents undertake the test at equilibrium if and only if there is no moral hazard.

Finally, part (d) shows that the maximum value of the test is larger with moral hazard, when prevention efficiency is close to its maximum, because the reduced insurance coverage generated by moral hazard considerations hurts more uninformed than type H agents.

With our numerical example, Proposition 3 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 moral hazard on the amount of prevention effort at equilibrium.

Proposition 4 *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 (ϕ, 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 $\phi_{min}^{MH} < \phi_{min}$ and that $\phi_{max}^{MH} < \phi_{max}$. We now show that the same result holds if (ϕ, Δ) is such that the introduction of moral hazard changes the testing decision. Proposition 3 has shown that two situations may occur. The first one happens when (ϕ, Δ) is such that the test is taken if and only if there is moral hazard. This case materializes when the effort cost is low enough ($\phi < \tilde{\phi}_1(\Delta) < \phi_{min}$) 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 induces low type agents not to exert the effort. We then obtain that introducing moral hazard decreases by $1 - \lambda$ the fraction of the population exerting the prevention effort at equilibrium. A similar phenomenon appears when the effort cost is high enough that agents take the test if and only if there is no moral hazard. The cost is high enough ($\phi > \tilde{\phi}_2^{MH}(\Delta) > \phi_{min}^{MH}$) that, with moral hazard, agents remain uninformed and do not exert effort while, without moral hazard, agents take the test and thus exert effort if they are of type H . 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 .

Proposition 3 (a) proves that 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 and when Δ is large enough. This suggests that taking the test may be compatible with lower values of the prevention efficiency with than without moral hazard. 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 0.034) with than without moral hazard (where $\tilde{\Delta} = 0.04$). We then have that

Proposition 5 *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 then add sequentially the availability of prevention and of testing in order to measure their separate 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 ϕ , for a given value of Δ , under various scenarios.

Insert Figure 6

When prevention is not available, 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 ϕ . This utility level corresponds to the horizontal line on Figure 6. We then introduce the possibility to exert observable effort but assume that the genetic test is not available. In that case, agents are uninformed about their individual probability and exert effort if and only if the effort cost is lower than the threshold ϕ_{min} (see Result 1). Their *ex ante* utility is given by $v(c_U^1) - \phi$ for $\phi < \phi_{min}$, and $v(c_U^0)$ for $\phi \geq \phi_{min}$. 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 with observable effort. 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 large enough that the test is worth taking for certain values of ϕ . We know from Results 3 and 4 that the test is taken only if the effort cost ϕ is comprised between $\tilde{\phi}_1$ and $\tilde{\phi}_2$. For $\phi < \tilde{\phi}_1$, agents remain uninformed and exert effort, so that their utility remains $v(c_U^1) - \phi$, while if $\phi > \tilde{\phi}_2$ they also remain uninformed but do not exert effort, with a utility level of $v(c_U^0)$. For ϕ in between $\tilde{\phi}_1$ and $\tilde{\phi}_2$, agents test and their *ex ante* utility is $\lambda(v(c_H^1) - \phi) + (1 - \lambda)v(c_L^0)$, which decreases with ϕ 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 ϕ . 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 as a benchmark.¹¹ 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 (*ex ante* identical) individuals.¹² The test gives information that can be acted upon to reduce the health risk and is then prescribed to everyone. High type agents are told 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 $v(c_U^1) - \lambda\phi$ 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 obtain *ex ante* a utility level of $v(c_U^0)$ based on the higher average probability p_U^0 . So, the first-best solution entails effort for all agents of type H if and only if

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

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

¹¹The 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.

¹²We assume that the effort cost, being a utility cost, is not ensurable.

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 $\phi < \tilde{\phi}_1$) or to too little of it (if $\tilde{\phi}_2 < \phi < \phi_{min}/\lambda$). Second, even when the prevention decisions are first-best optimal (when $\phi_1 < \phi < \phi_2$), 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 available 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 decentralize the first-best allocation. 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 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.¹³ 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

¹³A similar mechanism (although in a different context) can be found in Cochrane (1995).

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

$$\phi < v(c_U^1) - v(c_U^0) = \phi_{min}.$$

The expected welfare of agents is then $v(c_U^1) - \phi$ if $\phi < \phi_{min}$ and $v(c_U^0)$ if $\phi \geq \phi_{min}$. 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 ϕ_{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 undertake the prevention effort.

We now turn to Figure 7, which depicts the impact of the unobservability of the prevention effort when the testing technology is available (but entails a discrimination risk). Lemma 4 in the Appendix shows that moral hazard reduces the value of the two cost thresholds between which policyholders take the test ($\tilde{\phi}_i^{MH} < \tilde{\phi}_i$, $i = 1, 2$). Moreover, the *ex ante* utility is lower with moral hazard when effort is undertaken, 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 probability 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 analysis? 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 agents with a predisposition to the disease to perform effort, with a low prevention cost there is actually too much prevention (all undertake the effort) while with a high 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 discrimination risk that is associated with 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 the discrimination risk disappear, only one decentralizes 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 agents to make the effort only when 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 efforts 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.¹⁴

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 discussion and projections about decoding the whole genome of individual human beings, in order to screen for as many potential disease risks 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.

¹⁴We acknowledge that the disclosure duty requirement (whether applied to the test results or the prevention effort) faces the problem of false statements. This problem is usually attenuated by the use of stiff penalties (up to the voiding of the contract) when a damage occurs and the insurer discovers the false statements (assuming they are verifiable *ex post* at a cost).

References

- [1] Bardey D. and R. Lesur, 2005, "Optimal health insurance contract: Is a deductible useful?," *Economics Letters*, vol. 87(3), p. 313-317.
- [2] Barigozzi F. and D. Henriët, 2011, "Genetic information: comparing alternative regulatory approaches when prevention matters", *Journal of Public Economic Theory*, 13(1), p. 23-46.
- [3] Cochrane J.H, 1995, "Time-Consistent Health Insurance," *Journal of Political Economy*, vol. 103(3), p445-73.
- [4] Collins F., 2010, *The Language of Life: DNA and the Revolution in Personalized Medicine*, kindle edition, HarperCollins Publishers.
- [5] Crainich D., 2011, "Self-insurance with genetic testing tools", Working paper Lem.
- [6] Dave D. and R. Kaestner, 2006, "Health Insurance and ex ante Moral Hazard: Evidence from Medicare", NBER Working Paper 12764.
- [7] Davies K., 2010, *The \$1,000 genome*, Free Press.
- [8] Dionne G. and L. Eeckhoudt, 1985, "Self-Insurance, Self-Protection an Increased Risk Aversion", *Economics Letters*, 17, p39-42.
- [9] Doherty N. and L. Posey, 1998, "On the value of a checkup: adverse selection, moral hazard and the value of information", *The Journal of Risk and Insurance*, 65(2), p. 189-211.
- [10] Doherty N. and P. Thistle, 1996, "Adverse selection with endogenous information in insurance market", *Journal of Public Economics*, 63, p. 83-102.
- [11] Ehrlich I. and G. Becker 1972, "Market insurance, self-insurance and self-protection", *Journal of Political Economy*, 80, p. 623-648.
- [12] Filipova L and M. Hoy, 2009, "Impact of genetic testing on surveillance and prevention", Working paper University of Guelph.
- [13] Hirshleifer J., 1971, "The Private and Social Value of Information and the Reward to Incentive Activity", *The American Economic Review*, 61, p. 561-74.
- [14] Hoel M. and T. Iversen, 2002, "Genetic testing when there is a mix of compulsory and voluntary health insurance" *Journal of Health Economics*, 21(2), p. 253-270.

- [15] Hoel M. and Iversen T, Nilssen T. and Vislie J., 2006, "Genetic testing in competitive insurance markets with repulsion from chance: A welfare analysis", *Journal of Health Economics*, 25, p. 847-860.
- [16] Hoy M., 1989, "The value of screening mechanisms under alternative insurance possibilities", *Journal of Public Economics*, 39, p. 177-206.
- [17] Hoy M. and Polborn M., 2000, "The value of genetic information in the life insurance market", *Journal of Public Economics*, 78, p. 235-252.
- [18] Rees R. and P. Apps, 2006, "Genetic testing, income distribution and insurance markets", *Les Annales d'Economie et de Statistique*, 83-84, p. 295-325.
- [19] Shavell S., 1979, "On moral hazard and insurance," *Quarterly Journal of Economics*, vol 93, No 4, p. 541-562.
- [20] Strohmenger R. and A. Wambach, 2000, "Adverse selection and categorical discrimination in the health insurance markets: the effects of genetic tests", *Journal of Health Economics*, 19(2), p. 197-218.
- [21] Tabarrok, A., 1994, "Genetic testing: an economic and contractarian analysis", *Journal of Health Economics*, 13, p. 75-91.

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 $\phi_{min} = \phi_{max} = 0$ if $\Delta = 0$, and that $\Psi(0, 0) < 0$. We also know that $\partial\phi_{min}/\partial\Delta = \lambda dv'(c_U^1) > 0$ (so that $\phi_{min} > 0$ if $\Delta > 0$) which, together with $\partial\Psi(\phi, \Delta)/\partial\phi > 0$ and $\partial\Psi(\phi, \Delta)/\partial\Delta > 0$, implies that

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

Finally, we know that $\Psi(0, \bar{\Delta}) = 0$ and that $\phi_{min} > 0$ when $\Delta = \bar{\Delta}$, which imply that $\Psi(\phi_{min}, \bar{\Delta}) > 0$. The continuity of $\Psi(\phi, \Delta)$ in Δ together with the fact that $\Psi(\phi, \Delta)$ is strictly increasing with Δ for any ϕ implies, by the intermediate value theorem, that there exists a unique value $0 < \Delta < \bar{\Delta}$, denoted by $\tilde{\Delta}$, such that $\Psi(\phi_{min}, \tilde{\Delta}) = 0$.

b) By the same reasoning as above, we know that $\Psi(\phi_{min}, \Delta) > 0$ for all $\Delta > \tilde{\Delta}$. The fact that $\partial\Psi(\phi, \Delta)/\partial\phi > 0$ 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 ϕ , denoted by $\tilde{\phi}_1(\Delta)$, such that $0 \leq \tilde{\phi}_1(\Delta) \leq \phi_{min}$ and $\Psi(\tilde{\phi}_1(\Delta), \Delta) = 0$;

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

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

Our claim then results from the fact that $\partial\Psi(\phi, \Delta)/\partial\phi > 0$ and that $\partial\Psi(\phi, \Delta)/\partial\Delta > 0$ for all ϕ 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 $\Psi(\phi_{min}, \Delta) > 0$ for all $\Delta > \tilde{\Delta}$. Second, Result 2 has shown that $\Psi(\phi_{max}, \Delta) < 0$ for all Δ . The fact that $\partial\Psi(\phi, \Delta)/\partial\phi = -\lambda < 0$ then implies, by the intermediate value theorem, that there exists a unique value of ϕ , denoted by $\tilde{\phi}_2(\Delta)$, such that $\phi_{min} \leq \tilde{\phi}_2(\Delta) < \phi_{max}$ and $\Psi(\tilde{\phi}_2(\Delta), \Delta) = 0$;
b) Straightforward since $\partial\Psi(\phi, \Delta)/\partial\phi = -\lambda < 0$.
c) We have by definition that $\Psi(\phi_2(\Delta), \Delta) = 0$ so that

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

Our claim then results from the fact that $\partial\Psi(\phi, \Delta)/\partial\phi < 0$ and that $\partial\Psi(\phi, \Delta)/\partial\Delta = \lambda dv'(c_H^1) > 0$ for all ϕ and Δ .

- d) The fact that $\tilde{\phi}_1(\tilde{\Delta}) = \tilde{\phi}_2(\tilde{\Delta}) = \phi_{min}$ comes from the definitions of $\tilde{\Delta}$, $\tilde{\phi}_1(\Delta)$ and $\tilde{\phi}_2(\Delta)$. The fact that $\phi_{min} < \tilde{\phi}_1(\tilde{\Delta}) < \phi_{max}$ comes from the observation that $\Psi(\phi_{min}, \tilde{\Delta}) > 0$ while $\Psi(\phi_{max}, \tilde{\Delta}) < 0$.

□

8.3 Proof of Lemma 1

- a) α_H and α_U are respectively implicitly determined by

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

and,

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

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

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

The implicit function theorem gives

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

b) The implicit function theorem implies:

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

The coverage rate α_H attains the minimum value of zero when

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

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

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

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

□

8.4 Proof of Result 5

We have respectively

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

and

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

Also, When $\phi = \bar{\phi}_H$, 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 ϕ (because of both the direct effect of a higher ϕ and the indirect impact through the decrease in coverage rate) while U_H^0 is not affected by ϕ , we have that $\phi_{max}^{MH} < \bar{\phi}_H$. The proof that $\phi_{min}^{MH} < \bar{\phi}_L$ is obtained in a similar way.

□

8.5 Proof of Lemma 2

a) We have that

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

We then have that

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

if and only if

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

If $\Delta \rightarrow \bar{\Delta}$, 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

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

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

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

□

8.6 Proof of Result 6

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

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

which implies that

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

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

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

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

We know from Lemma 2 b) that

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

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

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

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

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

□

8.7 Proof of Lemma 3

a) Observe that, for $\phi_{min}^{MH} < \phi < \phi_{max}^{MH}$, we have

$$\Psi^{MH}(\phi, \Delta) = \lambda(\phi_{max}^{MH} - \phi + v(c_H^0)) + (1 - \lambda)v(c_L^0) - v(c_U^0),$$

so that

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

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

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

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

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

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

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

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

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

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

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

which is always true since the RHS is negative.

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

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

□

8.8 Proof of Result 7

a) Recall that, for $\phi_{min}^{MH} < \phi < \phi_{max}^{MH}$, we have

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

We have established at the beginning of section 4.3 that $\Psi^{MH}(\phi_{max}^{MH}, \bar{\Delta}) < 0$. We now prove that $\Psi^{MH}(\phi_{min}^{MH}, \bar{\Delta}) > 0$. We have

$$\begin{aligned} \Psi^{MH}(\phi_{min}^{MH}, \bar{\Delta}) &= (1 - \lambda) [v(c_L^0) - v(c_U^0)] \\ &\quad + \lambda [p_L [v(d_H^1) - v(d_U^1)] + (1 - p_L) [v(b_H^1) - v(b_U^1)]] . \end{aligned} \quad (7)$$

Note that the first term of (7) is positive. Moreover, using the mean value theorem, we obtain that

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

with $\varpi \in [d_H^1, d_U^1]$ and $\nu \in [b_H^1, b_U^1]$. Therefore, the second term of (7) becomes

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

As $\alpha_H > \alpha_U$ (Lemma 1) and $\varpi < \nu$, then the concavity of $v(\cdot)$ implies that $\Psi^{MH}(\phi_{min}, \bar{\Delta}) < 0$.

Moreover, Lemma 3 has shown that $\partial\Psi^{MH}(\phi, \Delta)/\partial\phi < 0$ for $\phi_{min}^{MH} < \phi < \phi_{max}^{MH}$. As $\Psi^{MH}(\phi, \bar{\Delta})$ is continuous in ϕ , the intermediate value theorem implies that there exist $\tilde{\phi}_2^{MH}(\bar{\Delta}) \in]\phi_{min}^{MH}, \phi_{max}^{MH}[$ such that $\Psi^{MH}(\tilde{\phi}_2(\bar{\Delta}), \bar{\Delta}) = 0$. By continuity of $\Psi^{MH}(\phi, \Delta)$ in Δ , this threshold $\tilde{\phi}_2^{MH}(\Delta)$ also exists for value of Δ close enough to $\bar{\Delta}$. Observe that, when it exists, $\tilde{\phi}_2^{MH}(\Delta) < \phi_{max}^{MH}$ since $\Psi^{MH}(\phi_{max}^{MH}, \Delta) < 0$ for all Δ . From now on, we consider only values of Δ large enough that $\tilde{\phi}_2^{MH}(\Delta)$ exists.

b) The claim is straightforward since $\partial\Psi^{MH}(\phi, \Delta)/\partial\phi < 0$ by Lemma 3.

c) We have by definition that $\Psi^{MH}(\tilde{\phi}_2^{MH}(\Delta), \Delta) = 0$ so that

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

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

□

8.9 Proof of Proposition 3

We first prove the following two lemmatas.

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

Proof. Result 3 has shown that $\tilde{\phi}_1(\Delta) < \phi_{min}$ exists if $\Delta \geq \tilde{\Delta}$ (defined as $\Psi^{MH}(\phi_{min}, \tilde{\Delta}) = 0$), with $\Psi(\phi, \Delta) < 0$ for $\phi < \tilde{\phi}_1(\Delta)$. Obviously, $\Delta \rightarrow \bar{\Delta} > \tilde{\Delta}$, so that $\tilde{\phi}_1(\Delta)$ exists. Similarly, Result 6 shows that $\tilde{\phi}_1^{MH}(\Delta)$ exists for $\Delta \rightarrow \bar{\Delta}$, with $\Psi^{MH}(\tilde{\phi}_1^{MH}(\Delta), \Delta) = 0$. We then have that $\Psi^{MH}(\phi, \Delta) > \Psi(\phi, \Delta)$ for $\phi < \phi_{min}^{MH}$ (a consequence of Lemma 2) implies that $\tilde{\phi}_1^{MH}(\Delta) < \tilde{\phi}_1(\Delta)$.

Result 4 has shown that $\phi_{min} < \tilde{\phi}_2(\Delta) < \phi_{max}$ exists if $\Delta \geq \tilde{\Delta}$, with $\Psi(\phi, \Delta) > 0$ for $\tilde{\phi}_1(\Delta) < \phi < \tilde{\phi}_2(\Delta)$. Similarly, Result 7 shows that $\phi_2^{MH}(\Delta) < \phi_{max}^{MH}$ exists for $\Delta \rightarrow \bar{\Delta}$, with $\Psi^{MH}(\phi, \Delta) > 0$ for $\tilde{\phi}_1^{MH}(\Delta) < \phi < \tilde{\phi}_2^{MH}(\Delta)$. We then have that $\Psi^{MH}(\phi, \Delta) < \Psi(\phi, \Delta)$ for any Δ when $\phi_{min}^{MH} < \phi_{min} < \phi < \phi_{max}^{MH}$ (Lemma 5) implies that $\tilde{\phi}_2^{MH}(\Delta) < \tilde{\phi}_2(\Delta)$. ■

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

Proof. Recall that, when $\phi_{min} < \phi < \phi_{max}^{MH}$, we have

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

hence we obtain

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

■

We now prove Proposition 3

Proof. (a) Recall that, when Δ is close enough to $\bar{\Delta}$, we have that $\Psi^{MH}(0, \Delta) = \Psi(0, \Delta)$ and that $\Psi^{MH}(\phi, \Delta) > \Psi(\phi, \Delta)$ for $\phi < \phi_{min}^{MH}$ (see Lemma 2 for both), which implies that $\Psi^{MH}(\phi_{min}^{MH}, \Delta) > \Psi(\phi_{min}^{MH}, \Delta)$. Lemma 5 shows that $\Psi^{MH}(\phi, \Delta) < \Psi(\phi, \Delta)$ for $\phi_{min}^{MH} < \phi_{min} < \phi < \phi_{max}^{MH}$. By continuity of $\Psi^{MH}(\phi, \Delta)$ and $\Psi(\phi, \Delta)$ in ϕ , the fact that $\partial\Psi^{MH}(\phi, \Delta)/\partial\phi < 0$ for $\phi_{min}^{MH} < \phi < \phi_{min}$ (see Lemma 3) and the intermediate value theorem, we then have that there exists a unique value of ϕ , denoted by $\hat{\phi}$, with $\phi_{min}^{MH} < \hat{\phi} < \phi_{min}$, and such that $\Psi^{MH}(\phi, \Delta) > \Psi(\phi, \Delta)$ for $\phi < \hat{\phi}$, $\Psi^{MH}(\hat{\phi}, \Delta) = \Psi(\hat{\phi}, \Delta)$ and $\Psi^{MH}(\phi, \Delta) < \Psi(\phi, \Delta)$ for $\hat{\phi} < \phi < \phi_{max}$. As for the latter inequality, observe that $\Psi^{MH}(\phi, \Delta) = \Psi_0 < \Psi(\phi, \Delta)$ for $\phi_{max}^{MH} \leq \phi < \phi_{max}$, while $\Psi^{MH}(\phi, \Delta) = \Psi(\phi, \Delta) = \Psi_0$ for $\phi \geq \phi_{max}$.

(b) The proof of Lemma 4 shows that $\tilde{\phi}_1(\Delta)$, $\tilde{\phi}_1^{MH}(\Delta)$ and $\tilde{\phi}_2^{MH}(\Delta)$ exist when $\Delta \rightarrow \bar{\Delta}$. The claim follows from the observation that $\Psi^{MH}(\phi, \Delta) > 0$ for $\tilde{\phi}_1^{MH}(\Delta) < \phi < \tilde{\phi}_2^{MH}(\Delta)$ (Results 6 and 7) while $\Psi(\phi, \Delta) < 0$ for $\phi < \tilde{\phi}_1(\Delta)$ (Result 3).

- (c) The proof of Lemma 4 shows that $\tilde{\phi}_1(\Delta)$, $\tilde{\phi}_2(\Delta)$ and $\tilde{\phi}_2^{MH}(\Delta)$ exist when $\Delta \rightarrow \bar{\Delta}$. The claim follows from the observation that $\Psi^{MH}(\phi, \Delta) < 0$ for $\phi > \tilde{\phi}_2^{MH}(\Delta)$ (Proposition 1) while $\Psi(\phi, \Delta) > 0$ for $\tilde{\phi}_1(\Delta) < \phi < \tilde{\phi}_2(\Delta)$ (Results 3 and 4).
- (d) Recall that

$$\begin{aligned}\Psi^{MH}(\phi_{min}^{MH}, \Delta) &= \lambda [(1 - p_H^1)v(b_H^1) + p_H^1v(d_H^1) - (1 - p_U^1)v(b_U^1) - p_U^1v(d_U^1)] + (1 - \lambda) [v(c_L^0) - v(c_U^0)], \\ \Psi(\phi_{min}, \Delta) &= \lambda [v(c_H^1) - v(c_U^1)] + (1 - \lambda) [v(c_L^0) - v(c_U^0)],\end{aligned}$$

so that

$$\begin{aligned}\Psi^{MH}(\phi_{min}^{MH}, \Delta) &> \Psi(\phi_{min}, \Delta) \\ \Leftrightarrow v(c_U^1) - [(1 - p_U^1)v(b_U^1) - p_U^1v(d_U^1)] &> v(c_H^1) - [(1 - p_H^1)v(b_H^1) + p_H^1v(d_H^1)].\end{aligned}$$

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

$$\begin{aligned}(1 - p_L) [v(b_U^1) - v(b_H^1)] &< p_L [v(d_H^1) - v(d_U^1)] \\ \Leftrightarrow (1 - p_L) [b_U^1 - b_H^1] v'(\alpha) &< p_L [d_H^1 - d_U^1] v'(\beta), \quad (8)\end{aligned}$$

with $\alpha > \beta$. Using

$$\begin{aligned}b_U^1 - b_H^1 &= (\alpha_H - \alpha_U)p_L d, \\ b_U^1 - b_H^1 &= (\alpha_H - \alpha_U)p_L d,\end{aligned}$$

the inequality (8) becomes

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

which is true.

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

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