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Assurance maladie et tests génétiques

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**THÈSE DE DOCTORAT DE L'UNIVERSITÉ BOURGOGNE FRANCHE-COMTÉ
PRÉPARÉE À L'UNIVERSITÉ DE FRANCHE-COMTÉ**

École doctorale n°593 Droit, Gestion, Sciences Économiques et Politiques

Doctorat de Sciences Économiques

Par
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Assurance Maladie et Tests Génétiques

Thèse présentée et soutenue à Besançon, le 21 septembre 2020

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Titre: Assurance maladie et Tests génétiques

Mots clés: Aléa moral, Antisélection, Information génétique, Assurance, Médecine préventive, Médecine personnalisée

Résumé: Cette thèse apporte trois principales contributions. Le premier chapitre, un article paru en 2019 dans la Revue Française d'Économie n°2/vol XXXIV, propose une revue de la littérature sur les implications de la régulation des tests génétiques de prédisposition sur le marché de l'assurance santé. Nous montrons à travers la littérature que les formes actuelles de régulation réalisent un arbitrage entre la maximisation du bien-être social ex ante et l'incitation à la prévention. Cet arbitrage est conditionné par la façon dont l'acquisition de l'information affecte les comportements de prévention et de révélation des agents, la discrimination des risques par les assureurs ainsi que la nature des contrats. Le deuxième chapitre étudie théoriquement l'impact de la reclassification sur la prévention, la décision de faire le test ainsi que sur le bien-être social dans la réglementation d'obligation de divulgation. En particulier, nous montrons qu'en fonction du coût de l'effort de prévention et de l'intensité de la reclassification la valeur individuelle de l'information génétique avec reclassification peut être plus élevée que sans reclassification. De plus, nous montrons comment les préférences temporelles affectent la valeur individuelle de l'information génétique. Enfin, nous montrons que le bien-être social est plus élevé sans reclassification qu'avec reclassification. Le dernier chapitre étudie et caractérise des contrats incitatifs pouvant être mis en œuvre pour développer la médecine personnalisée avec des traitements très efficaces, dans un contexte d'aléa moral sur l'effort fourni par la firme pour améliorer l'efficacité du médicament. Nous considérons un modèle dans lequel l'autorité de santé a trois possibilités. Elle peut appliquer le même traitement (standard ou nouveau traitement) à l'ensemble de la population ou mettre en œuvre la médecine personnalisée, c'est-à-dire utiliser les informations génétiques pour proposer le traitement le plus adapté à chaque patient. Nous caractérisons d'abord le contrat de remboursement de médicaments d'une entreprise produisant un nouveau traitement avec un test génétique compagnon lorsque l'entreprise peut entreprendre un effort pour améliorer la qualité des médicaments. Ensuite, nous déterminons les conditions sous lesquelles la médecine personnalisée doit être mise en œuvre lorsque cet effort est observable et quand il ne l'est pas. Enfin, nous montrons comment la non observabilité de l'effort affecte la décision de l'autorité sanitaire de mettre en œuvre la médecine personnalisée avec des traitements hautement efficaces.

Title: Health Insurance and Genetic Information

Mots clés: Moral Hazard, Adverse Selection, Genetic Information, Insurance, Preventive Medicine, Personalized Medicine

Abstract: This thesis includes three main contributions. The first chapter, an article published in 2019 in the “Revue Française d’Économie n°2/vol XXXIV”, provides a literature review on the implications of genetic testing regulations on the health insurance market. We show through the literature that existing regulations make a trade-off between the maximization of ex-ante social welfare and incentive to undertake prevention actions. Indeed, this trade-off depends on the way information acquisition impacts prevention and revelation behaviors of agents, as well as of its impact on insurance contract. The second chapter studies theoretically how reclassification impacts testing and prevention decision as well as social welfare in the disclosure duty regulation. In particular, we show that the incentives of agents to take genetic with reclassification can be higher than without reclassification according to the effort cost. In addition, we show how time preferences affect the incentive to take genetic testing. Finally, we show that the social welfare is strictly higher without reclassification than with reclassification. The last chapter studies and characterizes contracts that can be implemented to develop personalized medicine with highly effective treatment in the context of moral hazard about firm effort to improve drug efficacy. It also studies how the non-observability of effort impacts the decision of a health authority to implement personalized medicine with highly effective treatments. We consider a model in which the health authority has three possibilities. It can apply either the same treatment (a standard or a new treatment) to the whole population or implement personalized medicine, i.e., use genetic information to offer the most suitable treatment to each patient. We first characterize the drug reimbursement contract of a firm producing a new treatment with a companion genetic test when the firm can undertake an effort to improve drug quality. Then, we determine the conditions under which personalized medicine should be implemented when this effort is observable and when it is not. Finally, we show how the unobservability of effort affects the conditions under which the health authority implements personalized medicine.



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*Refuser d'accepter l'hypothèse pour guide est
se condamner à prendre le hasard pour
maître.*

Gustave Le Bon (1841-1931)

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List of Abbreviations

ADN	Acide Désoxyribonucléique
BDD	Bardey and De Donder [2013]
DNA	Deoxyribonucleic Acid
FDA	Food and Drug Administration
GDP	Gross Domestic Product
OECD	Organisation for Economic Co-operation and Development
OMS	Organisation Mondiale de la Santé
PM	Personalized Medicine
QALY	Quality-Adjusted Life Year
US	United States

General Introduction

Insurance of financial risks related to a poor state of health represents a major economic and societal issue. Covering these risks allows to finance health care expenditures which represent a large share of GDP in OECD countries. For instance, in 2018, health expenditures represented about 11.3 % of GDP in France according to the OECD.

Traditionally, when economists study the health insurance market, they focus on determining the optimal insurance coverage of individuals and on demonstrating how asymmetric information impacts the supply and demand of health insurance as well as the social welfare. Basically, insurance market is a mechanism for transferring risk from risk-averse agents to insurers which are less risk-averse or even risk-neutral. If risks are not correlated and under the Law of Large Numbers, insurers are able to spread the risk among the insureds. Let us consider an individual subject to a negative event with a certain probability. Risk-averse individuals desire to purchase insurance to cover the risk of the negative event. The decision to purchase insurance will depend on the amount of the loss, the perceived probability of the negative event, the degree of risk aversion and the insurance premium. Thus, if premiums are actuarially fair, it is optimal that risk-averse individuals are fully insured (Mossin (1968)). A premium is actuarially fair if it reflects the expected cost of the negative events, i.e., the amount of loss times the probability of occurrence. Thus, one of

the benefits of insurance is to allow individuals to avoid variability in future consumption. Concerning the health insurance market, another important motive of individuals for purchasing an insurance policy is that it would allow them to access expensive treatments, which would be inaccessible in the absence of insurance (Nyman (1999)). However, other studies show that access to the health insurance market can lead individuals to overconsume health goods, which negatively impacts social welfare (Pauly (1968)).

In the presence of risks heterogeneity, when insurers can perfectly observe the risk of individuals and are allowed to discriminate according to risk, competition between insurers implies that insurance contracts are individually risk-adjusted premium. Risk segmentation is then the characteristics of all private health insurance market. In practice, the segmentation of risks on the market results in the exclusion of high-risk individuals facing prohibitive insurance premium. In most developed countries, insurers are prohibited from discriminating individuals according to their health risk in order to guarantee access to all individuals and due to the negative externalities of a poor health. Feldman and Dowd (2000) underline that risk segmentation may be efficient from an ex-post view, i.e., after consumers' risks are known. However, from the ex ante perspective, risk segmentation can be inefficient as it undermines long-term insurance contracts that would protect consumers against changes in lifetime risk. Nevertheless, Bardey (2003) shows that premium variation may be desirable on the health insurance market since it could give incentives to individuals to undertake prevention action to reduce the probability of the loss.

When only individuals have information about their risk while insurers cannot identify the risk type, insurance market is facing adverse selection problem. Indeed,

in the absence of risk-based pricing, or if premium is too far from individual risks, this can therefore lead to a market equilibrium where “high risks agents”, which contribute to increasing the level of the insurance premium, “push out low risk agents” who prefer not to purchase insurance policy. Economic theory shows that if insurers are unable to distinguish the level of risk of policyholders, there can be complete market failure (Rothschild and Stiglitz (1976)).

The economic theory characterizes a set of insurance contracts that can be implemented to face adverse selection problem on the health insurance market. First, Rothschild and Stiglitz (1976) have proposed a set of contracts according to which each individual chooses the contract that corresponds to his/her risk. This equilibrium exists if and only if the proportion of low risks in the population does not exceed a theoretical threshold. In this menu of contracts, the policy intended for low risks must have such a low level of coverage that it is not attractive to high risks. In this separating equilibrium market, low risks individuals obtain partial insurance coverage, while high risks are fully insured. As a result, the market separating equilibrium under asymmetric information is not efficient. Dahlby (1981) shows that, when a universal and compulsory partial insurance is introduced to the separating Rothschild and Stiglitz (1976) equilibrium, the resulting pair of contracts at market equilibrium Pareto-dominates the pure separating equilibrium. Indeed, introducing a compulsory partial insurance increases the coverage of low risk individuals while high risk types benefit from the cross-subsidization.

An alternative market solution to adverse selection is the equilibrium concept developed by Wilson (1977), Miyazaki (1977) and Spence (1978) in which low risk agents accept to subsidize high risk types in order to obtain a better but a partial

insurance coverage. As previously, the equilibrium market is inefficient since individuals obtain partial insurance coverage, impacting negatively the social welfare.

Another concern of economic analysis under imperfect information is to investigate how moral hazard (ex ante and ex post) impacts health insurance market. Ex ante moral hazard exists when insured behavior influences the probability of occurrence of damage and cannot be observed by insurers. Thus, the ex ante moral hazard results from the fact that insured have less incentive to undertake effort to reduce the probability of occurrence of the loss when they are fully insured. Ex post moral hazard refers to the situation in which an individual with insurance policy will have higher claim than an individual without insurance. In a moral hazard context, optimal contracts result from a trade-off between risk-sharing and financial incentives to undertake prevention. Indeed, insured individuals who benefit from full health coverage have no financial incentives for undertaking prevention action to reduce the risk since they are fully compensated in the event of a claim. In order to give financial incentives to individuals, insurers offer insurance contracts with incomplete coverage. As a result, the existence of moral hazard implies that full insurance coverage is not optimal (Pauly (1968)).

The development and the use of genetic testing, in particular predictive genetic testing, raise many questions and have many economic implications on the health system. According to Khan and Mittelman (2018), in 2018, more than 10 million people around the world have taken a genetic testing. A predictive genetic testing allows to assess the individual probability of developing a disease (genetic predisposition testing) or of being receptive to treatment (pharmacogenetics tests). Predictive genetic information has implications on prevention action, the choice of treatment,

as well as on the health insurance market and the pharmaceutical industry. The regulation of genetic predisposition testing is different from a country to another, going from strict regulation to the absence of regulation. There are four major types of health insurance market regulation of predictive genetic information: disclosure duty, consent law, laissez-faire and strict prohibition (see Hoy and Ruse (2005), Viswanathan et al. (2007)).

Theoretical economic studies have shown that the incentive to gather genetic information is stronger when the regulation limits the insurers' use of information in insurance pricing (Doherty and Thistle (1996); Doherty and Posey (1998) and Bardey et al. (2017)). However, the social welfare is higher when individuals must reveal to insurer the result of genetic testing (Barigozzi and Henriët (2011); Peter et al. (2017)). As information revelation leads to genetic discrimination, individuals have less incentives for performing genetic testing. The debate on the best way to limit genetic discrimination and opportunistic behavior with regard to genetic information is still topical and faces a dilemma.

Regarding pharmacogenetic tests, their utility stems from the fact that the efficacy of some drugs varies according to the patient's genetic marker. This represents a major medical and public health concern. Indeed, the ineffectiveness and side effects of drugs in a large number of patients generate substantial costs to the healthcare system. Pharmacogenetic tests allow to implement personalized medicine, i.e., to identify the most receptive patients in order to administer the best treatment to patients. Using a pharmacogenetic test to choose the best treatment to patient may generate direct and indirect effects. The direct effects include the possibility to better care patient by selecting the best treatment with the highest efficacy

and minimizing the side effects. The identification of individuals who are not receptive to certain drugs makes it possible to avoid administering treatments which can be expensive when the gains are relatively small. In addition, some individuals are exposed to side effects which represent tremendous costs for the health system. The implementation of personalized medicine should allow an individual to reduce his/her medical expenditures (since the test is inexpensive) and, consequently, his/her insurance premium. The indirect effects of personalized medicine cover both the reduction of uncertainty and a better perception of the efficacy of drugs that can lead to better therapeutic adherence (Garrison and Austin (2007)). For an individual, the fact that the drug is adapted to his pathology generates a positive psychological effect which can increase the placebo effect of the drug. The strategy of differentiated treatment clearly produces other non-monetary effects, in particular, confidence or increase in perception in the ability of drugs to produce the expected effects. A better perception of the effectiveness of drugs may contribute to a better therapeutic adherence leading to better results in terms of medical utility.

One of the major issues of personalized medicine is the risk of rising health care expenditures that could threaten the sustainability of health systems. In addition, personalized medicine could impact the pharmaceutical industry, which has significant R&D investments. The segmentation of the market may result in increasing the price of drugs due to the scale of production or even the modification of firms' strategies due to the complementarity between tests and treatments (Gabszewicz et al. (2001); Seror et al. (2006)).

Apart from clinical utility and validity of the test, ethical issues, and concerns about storage and confidentiality of genetic data, there are two main economic issues:

1) What are the implications of the different regulatory regimes on testing and prevention decisions and on the efficiency of the health insurance market?

2) How pricing and reimbursement of drugs will impact the incentives of pharmaceutical companies to develop companion test and treatment and what are the factors affecting the decision of the health authority and health providers to implement personalized medicine?

There are an extensive works on genetic testing in health insurance markets focusing on testing and prevention decisions and the welfare consequences of different regulatory regimes and insurance market organization (Doherty and Thistle (1996); Doherty and Posey (1998); Bardey et al. (2017); Barigozzi and Henriet (2011); Peter et al. (2017); Hoel and Iversen (2002)). There has been no consideration paid to how premium variation on the health insurance market affects testing and prevention decisions and social welfare. The theoretical literature investigating the economic implications of the development of personalized medicine is less abundant. Recently, the theoretical literature has investigated the economic implications of the development of personalized medicine. Research mostly focuses on how the degree of accuracy of the test affects the decision of the health authority to implement personalized medicine and on the factors impacting pricing policy of firms and the optimal outcome-based payment policy to encourage investment in post-approval companion test (Antonanzas et al. (2015), (2016), (2018)). One of the main questions is whether or not the implementation of personalized medicine will allow the pharmaceutical industry to develop drugs of higher quality. There is no paper dealing with this issue in the literature. To answer these questions, it is necessary to develop a normative research framework. This thesis deals with these issues and

offers a scientific approach. Two main methods are used in this thesis: literature review and microeconomic models.

This thesis includes three major contributions. Chapter 1 includes an article published in 2019 in the “Revue Française d’économie n°2/vol. XXXIV”, a literature review on the implications of genetic testing regulations on the health insurance market. We show through the literature that the choice of a regulation results from a trade-off between the maximization of ex-ante social welfare and incentive to undertake prevention actions. Indeed, this trade-off depends on the way information acquisition impacts prevention and revelation behaviors of agents, as well as of its impact on insurance contract.

In the first part of this chapter, we first analyze the utility derived from acquiring genetic information (in terms of prevention decision), and the economic implications of genetic testing on the health insurance market. We then examine how the different regulations affect the incentive of individuals to acquire genetic information. We underline that the utility of genetic predisposition testing, deriving from efficient prevention actions, can be offset by genetic discrimination, the psychological effect of being informed, as well as the level of accuracy of the test. In addition, the decision to take genetic testing depends on the cost and the efficiency of prevention action, the regulations in force and the type of the insurance contracts on the market. In particular, individuals take less often genetic testing in the disclosure duty than the other regulatory regimes.

In the second part, we study how the four main regulations affect the social welfare, considering different types of organization of the health insurance market. This comparative analysis of social welfare is carried out from an ex ante point of view, i.e. in

a context where all individuals are initially uninformed, and before the realization of risk. We show that the social welfare is higher when genetic information is revealed to insurers.

Finally, we study the original Tabarrock (1994) proposition, a market selling insurance against discrimination risk, to overcome genetic discrimination. We show that genetic insurance would both maximize social welfare and allows insureds to undertake optimal prevention actions. However, genetic insurance presents difficulties of implementation.

The second chapter provides a theoretical economic analysis of the implications of reclassification on testing and prevention decision as well as on social welfare in the disclosure duty regulation. We consider a health insurance market in which insureds are reclassified following the realization of the bad state of nature at renewal of contracts and may undertake primary prevention action. Compared to a single period model and a dynamic model without reclassification, we show that under some conditions individuals may prefer to acquire genetic information in the disclosure duty regulation when they are reclassified in the second period, even though there is no effective prevention action. The incentives of agents to take genetic testing with reclassification can be higher than without reclassification according to the intensity of reclassification and the ordering of the cost of effort thresholds. In addition, we show how time preferences affect the incentive to take genetic testing. Finally, we show that the social welfare is strictly higher without reclassification than with reclassification.

In the first part of this chapter, we study and compare with the one period model the incentives of individuals to take genetic testings in a two periods framework when there is no reclassification. This setting allows us to compare the incentives of indi-

viduals to take genetic testing depending on whether they value or not their second period expected utility. We show that the incentive to take genetic testing when individuals value their second period can be higher and depends on the technology of the test (its cost and its efficiency), the degree of risk aversion as well as the value assigned by the individual to the second period. In particular, if prevention efficiency is large, the incentive to take the test is higher the more the individual values their second period.

In the second part, we introduce the reclassification variable in the analysis. We study and compare the incentive of individuals to take genetic test with reclassification and without reclassification. We show that the incentive of individuals to take genetic test with reclassification can be higher and depends on the prevention efficiency, the cost of prevention, and the intensity of reclassification. In particular, when the cost of effort is low or high in such a way that individuals undertake the same prevention decision in the two models, the incentive to take the test with reclassification is higher than without reclassification.

Finally, we compare the social welfare achieved with and without reclassification. We show that the ex ante utility with reclassification is strictly lower than without reclassification even when agents take the same decision regarding the test and prevention action and independently of the impact of reclassification on prevention utility. Indeed, the social welfare with reclassification is always lower than without reclassification because of the loss of utility resulting from reclassification.

The third chapter, a research paper submitted for second review to the International Journal of Health Economics and Management (a work in collaboration with

professors François Maréchal and Florence Naegelen), contributes to the literature by analyzing the decision of a health authority to implement personalized medicine. The challenge of this chapter is to define incentive contracts allowing the development of personalized medicine with highly effective treatments, and to analyze the decision of an authority to set it up when the effort exerted by the firm to improve the effectiveness of the drug is not observable. We consider a model in which the health authority has three possibilities. It can apply either the same treatment (a standard or a new treatment) to the whole population or implement personalized medicine, i.e., use genetic information to offer the most suitable treatment to each patient. We first characterize the drug reimbursement contract of a firm producing a new treatment with a companion genetic test when the firm can undertake an effort to improve drug quality. Then, we determine the conditions under which personalized medicine should be implemented when this effort is observable and when it is not. Finally, we show how the unobservability of effort affects the conditions under which the health authority implements personalized medicine. In particular, our results reveal that personalized medicine will be implemented less often when effort is not observable than when it is, when the standard treatment is preferred without genetic information or when both the new treatment is preferred without genetic information and the price of this treatment is below a threshold.

Chapter 1

Tests génétiques prédictifs et marchés de l'assurance santé

1.1 Introduction

De nombreux tests génétiques apportent des informations sur l'état de santé actuel et/ou futur des individus. L'information issue de ces tests génétiques a de nombreuses implications, tant au niveau de la prévention et du choix des traitements, qu'au niveau de l'assurance maladie et de l'industrie pharmaceutique. Il existe principalement deux catégories de tests prédictifs : les tests de susceptibilité (ou de prédisposition) et les tests pharmacogénétiques ¹.

Les premiers apportent une information sur la probabilité de développer une maladie. Ainsi, les femmes porteuses de mutations sur les gènes BRCA1 et BRCA2 ont un risque plus élevé de développer un cancer du sein et de l'ovaire que les femmes non porteuses. La détection de ces mutations améliore l'information sur la probabilité de développer ce type de cancer pour une femme spécifique, probabilité qui sans cela serait égale au risque moyen de la population féminine. La deuxième catégorie de tests prédictifs, réalisés après l'apparition de la maladie, apporte une information sur la probabilité individuelle d'être réceptif à un traitement et permet donc de choisir les traitements les plus adaptés. Dans cet article, nous analysons les conséquences de l'utilisation (ou non) de l'information fournie par les tests génétiques de susceptibilité sur les comportements des individus, les marchés de l'assurance santé, et le surplus social.

Selon l'agence de la biomédecine, plus de 6 000 maladies sont liées à la mutation d'un ou de plusieurs gènes ². Les avancées médicales et technologiques ainsi que l'ouverture à la concurrence du marché ont entraîné une baisse considérable des coûts des tests génétiques. Actuellement, certaines firmes proposent des kits (in-

cluant une centaine de tests) pour environ 200 euros ³ Selon l'Institut national du cancer en France, le prix de l'analyse complète du génome humain, laquelle peut être réalisée en une quinzaine de jours, s'élève actuellement à près de 1 000 euros.

L'information issue des tests génétiques prédictifs a de nombreuses implications. Elle présente d'abord un intérêt au niveau de la prévention, permettant une meilleure gestion du risque maladie. Grâce à cette information, l'individu peut mieux ajuster ses actions de prévention. Un individu dont le risque de développer une maladie est au-dessus de la moyenne va ainsi pouvoir calibrer son effort de prévention en fonction de son risque individuel et non en fonction du risque moyen perçu, alors que réaliser cet effort peut être improductif pour une personne caractérisée par un risque faible. A titre d'illustration, à la suite d'un test génétique révélant une forte prédisposition au cancer du sein, l'actrice américaine Angelina Jolie a subi à titre préventif une double mastectomie, permettant ainsi de ramener ce risque à un niveau négligeable.

Si l'on se place maintenant du point de vue de l'assurance maladie, entreprendre un test génétique prédictif expose au risque de discrimination dans un système d'assurance maladie où la prime est calculée en fonction du risque individuel. Selon le principe de prime actuarielle, une compagnie d'assurance peut imposer à un individu une prime d'assurance prohibitive lorsque le résultat du test génétique se révèle positif. Par ailleurs, l'acquisition d'une information plus précise sur le risque individuel de santé peut avoir des conséquences négatives sur le marché de l'assurance. Cela peut notamment être le cas en présence de comportements stratégiques de la part des individus ayant acquis privativement l'information qu'ils sont à haut risque, dans le cas où cette information n'est pas connue de l'assureur⁴. L'information issue des tests génétiques de prédisposition peut ainsi être néfaste pour l'individu et

mettre à mal l'assurance maladie.

Au regard de ces enjeux, différentes modalités de régulations, visant à encadrer l'acquisition de l'information génétique et son utilisation par les assureurs, notamment celle issue des tests de prédisposition, ont vu le jour. La réglementation des tests génétiques a ainsi fait l'objet de traitements variables selon les pays, allant d'une réglementation stricte à l'absence de réglementation. Quatre types de régulation de l'information génétique peuvent être distingués : l'obligation de divulgation, le droit ou la règle de consentement, l'interdiction stricte, et le laissez-faire. Elles diffèrent par leur objectif fondamental, selon qu'elles cherchent à protéger les individus contre la discrimination génétique ou qu'elles visent plutôt à protéger le marché contre les comportements opportunistes des individus.

Il existe une littérature abondante sur la valeur de l'information génétique. Ces travaux s'intéressent à la nature des incitations à acquérir cette information, à son utilité pour l'individu ainsi qu'à l'évaluation de l'impact des différentes régulations sur le surplus social, selon différents types d'organisation du marché de l'assurance santé. De nombreuses études théoriques (Doherty et Thistle [1996] ; Doherty et Posey [1998] et Bardey *et al.* [2017]) ont montré que l'incitation à acquérir l'information génétique est plus forte lorsque la régulation limite la transmission de l'information. Au contraire, le surplus social est plus élevé lorsque la réglementation rend obligatoire la transmission de l'information génétique à l'assureur (Barigozzi et Henriët [2011] ; Peter *et al.* [2017]). Dans la mesure où la révélation de l'information conduit à la discrimination génétique, elle engendre en revanche un effet désincitatif sur la réalisation des tests. Le débat sur la meilleure façon de limiter la discrimination génétique et les comportements opportunistes vis-à-vis de l'information reste

toujours d'actualité et fait face à un dilemme. Nous montrerons ainsi que le choix d'une régulation résulte d'un arbitrage entre la maximisation du surplus social *ex ante* et l'incitation à la prévention.

Le reste de l'article s'organise de la manière suivante : nous étudions dans un premier temps la valeur individuelle de l'information génétique pour montrer que son utilité réside dans les actions de prévention qu'elle permet d'entreprendre. Nous montrons que la décision d'acquérir l'information dépend des préférences de l'individu et de la régulation du marché, mais que cette information peut être préjudiciable à l'individu, et est susceptible d'affecter la nature des contrats d'assurance sur le marché. Dans un second temps, nous étudions la valeur sociale de l'information génétique selon les différentes régulations et les différents types d'organisation du marché de l'assurance santé, puis nous discutons la proposition de Tabarrock [1994] d'instaurer une assurance génétique susceptible de contrecarrer les effets pervers de l'information, en soulignant les problèmes liés à sa mise en œuvre.

1.2 La valeur individuelle de l'information génétique

Nous analysons tout d'abord l'intérêt pour l'individu d'acquérir de l'information génétique (en termes d'actions de prévention rendues possibles), et les conséquences économiques des tests génétiques sur le marché de l'assurance santé. Nous examinons ensuite la façon dont les différentes régulations affectent l'incitation des individus à acquérir l'information.

1.2.1 L'effet préventif de l'information génétique

Les progrès réalisés en génétique médicale ont permis d'identifier à un stade pré-symptomatique les personnes susceptibles de développer certaines maladies, ce qui permet d'entreprendre des actions de prévention. Selon la classification établie par l'Organisation mondiale de la santé, deux types de prévention peuvent être effectués à ce stade⁵. La prévention primaire se réfère aux comportements individuels qui tendent à réduire les facteurs de risques liés au mode de vie et aux habitudes de consommation. La prévention secondaire a pour finalité de détecter de manière précoce certaines maladies dont la prise en charge permettrait un meilleur traitement et une réduction de la gravité de la maladie. En effet, la plupart des maladies génétiques sont multifactorielles. Leur apparition dépend de la présence d'une mutation génétique et du mode de vie de l'individu⁶. L'information issue des tests génétiques prédictifs est utile médicalement car elle permet à l'individu de modifier son comportement afin de mieux gérer les risques de santé. Par exemple, une femme peut choisir de réduire sa consommation de cigarettes afin de diminuer le risque de développer la forme la plus courante du cancer du sein. Afin de bien appréhender l'impact de l'acquisition de l'information génétique sur la prévention, il est donc important de comprendre la façon dont les individus décident d'entreprendre la prévention et la nature des bénéfices et des coûts associés.

Dans le cas d'une maladie génétique, une personne non informée (qui n'a donc pas réalisé de test de susceptibilité) évalue son risque en fonction de ses antécédents familiaux ou du risque moyen perçu dans la population⁷. En matière de prévention, deux cas peuvent alors se produire. Dans le premier, l'individu ne fait pas de prévention, alors que cela serait souhaitable. Dans le second, le choix de prévention peut

ne pas être optimal, au sens où l'individu ne fournit pas le niveau d'effort correct, en termes de réduction du risque, de coût de l'effort ou de bénéfice de cet effort. Le choix de prévention est efficient lorsque l'individu choisit l'intensité de l'effort de telle sorte que le coût additionnel soit égal au bénéfice marginal de la prévention.

Considérons une maladie génétique dont les actions de prévention ne sont pas efficaces pour une personne non porteuse d'une mutation. La prévention étant coûteuse, il n'est donc pas souhaitable de l'étendre à toutes les catégories de la population. Si le coût du test génétique est négligeable, un individu a intérêt à le faire afin de pouvoir fournir un effort de prévention adapté à son risque, ce qui lui permettra de réduire à la fois la probabilité de survenance et le montant du dommage. Par conséquent, la baisse de l'espérance mathématique du dommage implique une réduction de la prime d'assurance pour les assurés.

L'une des spécificités des tests génétiques est que l'acquisition de l'information génétique relève du choix de l'individu. Même si dans le contexte médical, le recours aux tests génétiques se fait sous le contrôle et l'autorisation du praticien, le patient a le droit d'accepter ou de refuser ce que le médecin préconise. Une personne rationnelle va alors décider de se renseigner sur son patrimoine génétique en comparant les coûts et bénéfices qui y sont associés.

En matière de prévention, l'analyse économique s'est essentiellement intéressée à la relation entre prévention et assurance. Ehrlich et Becker [1972] ont montré que les actions optimales de prévention primaire et secondaire dépendent de la probabilité de la perte et de la couverture d'assurance, notamment lorsque l'effort de prévention de l'assuré est parfaitement observable par l'assureur. Ainsi, en présence d'une tarification actuarielle, prévention secondaire et assurance de marché sont substituables

car les actions de prévention secondaire impliquent, comme l'assurance, un transfert de richesse entre les différents états de la nature. En revanche, la prévention primaire et l'assurance de marché peuvent être complémentaires. Cette complémentarité implique que les actions de prévention primaire croissent avec le niveau de couverture des assurés. En effet, la prévention primaire permet de réduire le prix relatif de l'assurance/prévention primaire, ce qui induit une augmentation de la demande d'assurance.

En revanche, lorsque les actions de prévention sont inobservables ou invérifiables, qu'elles ne peuvent donc pas être stipulées dans un contrat, hypothèse la plus vraisemblable, il en découle un problème d'aléa moral. Deux types d'aléa moral peuvent être observés, l'aléa moral *ex ante* et l'aléa moral *ex post*. Le premier résulte de l'incitation qu'a un assuré à ne pas fournir suffisamment d'effort afin de réduire la probabilité de survenance du dommage. Le second se réfère à la situation dans laquelle une personne ayant une couverture d'assurance dépense plus qu'une personne n'ayant pas de couverture. L'aléa moral *ex post* correspond donc au cas où une personne ne prend pas en compte l'effet de ses dépenses sur l'augmentation de la prime. Selon Henriët et Rochet [1998], l'aléa moral *ex post* devrait disparaître si l'on peut définir un traitement adapté à une pathologie et ensuite vérifier si la consommation est abusive.

En situation d'aléa moral, un dilemme apparaît entre le partage des risques et l'incitation à l'effort de prévention. Les assurés qui bénéficient d'une couverture de santé complète n'ont aucun intérêt à faire de la prévention pour réduire le risque puisqu'ils sont totalement indemnisés en cas de sinistre. Pour rétablir un niveau adéquat d'incitation, les compagnies d'assurance leur proposent des con-

trats d'assurance avec couverture incomplète (Shavell [1979] ; Arrow [1971] ; Pauly [1968])⁸. Ainsi, l'introduction de l'aléa moral modifie la relation assurance et prévention primaire qui ne deviennent plus complémentaires.

Au-delà des études théoriques, certains travaux empiriques étudient les comportements de prévention des individus et la relation entre les actions de prévention et l'assurance de marché. Ainsi, Courbage et Coulon [2004] mettent en évidence l'existence d'une complémentarité entre assurance et prévention primaire (en ce qui concerne la prévention des cancers du sein et du col de l'utérus et les pratiques d'activités physiques au Royaume-Uni). Dans le cadre d'une étude expérimentale, Pannequin *et al.* [2016] montrent que prévention secondaire (auto-assurance) et assurance sont substituables. Cependant, les individus choisissent un niveau de prévention secondaire non optimal en raison d'une faible élasticité-prix. En effet, le niveau d'effort de prévention secondaire fourni est significativement inférieur au niveau optimal, lorsque la prime par unité de couverture est supérieure ou égale au tarif actuariel. Au contraire, lorsque la prime par unité de couverture est inférieure au tarif actuariel, le niveau d'effort est supérieur à l'effort optimal.

L'incidence de l'acquisition de l'information génétique sur la prévention a également fait l'objet de travaux empiriques. Ces travaux ne permettent pas de conclure que l'acquisition de l'information amène les individus à s'engager dans des actions de prévention secondaire. Par exemple, aux États-Unis, il n'a été observé qu'une faible croissance du suivi mammographique pour les femmes non malades porteuses d'une mutation dans l'année ayant suivi l'annonce des résultats des tests (Peshkin *et al.* [2002]). L'acceptabilité théorique et réelle des mesures de prévention secondaire est essentielle, notamment relevant de la chirurgie prophylactique⁹. Empiriquement il

semble que la majorité des femmes néerlandaises choisissent la mastectomie prophylactique (Lodder *et al.* [2002] ; Meijers-Heijboer *et al.* [2000]), alors que les femmes américaines et françaises paraissent plus réticentes (Lerman *et al.* [2000] et [1996]). À cet égard, les préférences des individus et les spécificités des tests doivent être prises en compte afin de mieux évaluer les comportements des individus vis-à-vis des tests génétiques prédictifs et des méthodes de prévention.

En définitive, si l'information issue des tests génétiques présente de manière indiscutable une utilité médicale, l'acquisition de cette information est susceptible d'influencer les termes des contrats d'assurance proposés à l'individu. Nous allons voir dans la section suivante comment cette information peut être préjudiciable à un individu compte tenu de ses préférences vis-à-vis du risque, avant de montrer comment elle peut affecter le marché de l'assurance santé.

1.2.2 Les conséquences des tests génétiques pour le marché

L'acquisition de l'information génétique tend à générer deux types de phénomènes sur le marché de l'assurance santé : la discrimination génétique (dans un cadre statique) ou la reclassification des risques (dans un cadre dynamique) et l'antisélection¹⁰. La discrimination des risques provient de la capacité des assureurs à en tenir compte dans la définition des contrats d'assurance, ce qui entraîne des problèmes d'équité. Lorsqu'un individu entreprend un test génétique prédictif, il s'expose au risque de classification ou de reclassification. Ce risque correspond au fait de devoir payer une prime plus élevée lorsque le résultat d'un test génétique est positif. Toute régulation autorisant les assureurs à utiliser cette information dans la définition des contrats d'assurance conduit à une discrimination génétique. Les personnes dont le résultat

d'un test génétique se révèle positif se verraient infliger des primes élevées et pourraient être exclues du marché. Le second problème, l'antisélection, se produit dans le cas où cette information est détenue uniquement par les assurés, qui peuvent avoir des comportements stratégiques mettant à mal le marché de l'assurance.

1.2.2.1 Information génétique et discrimination des risques

En matière d'assurance, les comportements vis-à-vis du risque représentent un élément clé de la décision d'entreprendre les tests. Afin de comprendre les conséquences économiques des tests génétiques sur les marchés de l'assurance santé, en particulier les problèmes liés à la discrimination génétique, il est essentiel de définir la notion d'aversion à l'égard du risque ainsi que la valeur de l'information au sens de Hirshleifer [1971]. On considère généralement que les assurés font preuve d'aversion au risque lorsqu'ils préféreraient un paiement certain à une loterie dont l'espérance de gain est égale à ce paiement certain. De manière générale, la théorie économique considère que tout mécanisme permettant de révéler de l'information n'a aucune valeur pour l'individu qui est averse au risque, notamment lorsque cela lui permet de passer d'une situation où il perçoit le risque moyen à une situation où il connaît le niveau de risque individuel (Hirshleifer [1971])¹¹. Si l'on considère que les agents sont averses au risque et obtiennent un contrat d'assurance en fonction de leur statut informationnel ainsi que de leur risque de santé, alors un individu obtiendra une utilité espérée moindre lorsque l'information est acquise. Intuitivement, cela signifie qu'un individu préfère une connaissance moins précise sur son état de santé afin d'éviter d'être traité comme une personne à haut risque dans le cas d'un résultat positif lorsque la perte induite par la maladie est exogène. Puisqu'aucune couverture contre le risque de classification n'est offerte sur le marché, l'information est coûteuse

pour les personnes averses au risque.

Dans une perspective dynamique, la discrimination des risques expose l'individu à un risque de variation de prime d'assurance compte tenu de l'évolution du risque de santé dans le temps. Dans un contexte où il n'existe pas de marchés d'assurance pour se protéger contre le risque d'être reclassifié par son assureur, le transfert de risque des assurés averses au risque vers les assureurs supposés neutres au risque devient incomplet, ce qui génère en soi une inefficacité (Cochrane [1995]). Deux principaux types de contrats contre le risque de variation de la prime d'assurance ont été proposés dans la littérature. D'une part, Cochrane [1995] propose une assurance contre le risque de variation de prime. Ce contrat paye une indemnité couvrant la variation de la prime d'assurance consécutive à l'évolution du risque de santé. D'autre part, Pauly *et al.* [1995] ont montré comment les contrats d'assurance renouvelables à prix garantis peuvent assurer le risque de prime. Ces deux types de contrats d'assurance contre le risque de variation de prime lié à l'évolution du risque de santé dans le temps sont différents de l'assurance génétique proposée par Tabarrock [1994] que nous développons plus loin. En raison des contraintes informationnelles, les contrats d'assurance à long terme contre la variation de prime sont difficiles à mettre en place sur le marché. En effet, cela implique que l'assureur puisse observer parfaitement l'évolution du risque de l'individu afin de verser l'indemnité. Le contrat renouvelable à prix garanti suppose par ailleurs que l'individu ne change pas d'assureur pendant toute sa vie.

Une autre solution envisageable pour lutter contre le risque de variation de la prime est la mise en place d'une assurance publique permettant de mutualiser ces risques. *A priori*, dans un pays au sein duquel l'assurance santé revêt un carac-

tère obligatoire et où les cotisations ne dépendent pas des risques, le principe de non-sélection des risques semble cohérent puisque le système vise à opérer une redistribution entre les risques. En revanche, dans un tel système, les individus n'ont pas beaucoup d'intérêt à faire de la prévention. À cet égard, Bardey [2003] montre que le choix de la nature privée ou publique de l'assurance maladie dans le cadre de la gestion des risques longs en santé résulte d'un arbitrage entre deux types d'inefficacités : le risque de variation de prime et les comportements d'aléa moral. Si un système d'assurance maladie publique permet de mutualiser davantage le risque de variation de la prime, cela tend à accentuer les comportements d'aléa moral. En revanche, l'existence d'un risque de reclassification, spécifique aux marchés concurrentiels de l'assurance santé, amène les individus à internaliser les bénéfices de la prévention et diminue l'ampleur des comportements d'aléa moral *ex ante*. Ce dernier résultat permet de comprendre que la classification (ou la reclassification dans un cadre dynamique) des risques peut être souhaitable puisqu'elle permet de limiter l'inefficacité liée aux comportements d'aléa moral des assurés. En ce sens, le système français est intéressant puisqu'il combine à la fois une assurance universelle publique fournissant une couverture partielle à tous les assurés, permettant ainsi de mutualiser davantage une partie du risque de variation de prime, et une assurance complémentaire privée qui devrait limiter certains comportements liés au risque moral. Toutefois, pour les maladies considérées comme étant de longue durée par l'assurance maladie, les assurés bénéficient d'une couverture complète.

Par ailleurs, l'analyse économique a démontré que deux types d'inefficacités liés aux tests génétiques peuvent se produire dans un système mixte (combinant une assurance universelle obligatoire offrant une couverture incomplète et une assurance privée complémentaire facultative). En raison du taux de couverture unique

de l'assurance universelle les individus ne font pas les tests de prédisposition alors même que ces derniers seraient socialement souhaitables ou ils les font tandis qu'il ne serait pas socialement souhaitable de les faire (Hoel et Iversen [2002]). La première inefficacité se manifeste pour un taux élevé de prise en charge de l'assurance obligatoire, lorsque la régulation confère un caractère privé à l'information génétique. La deuxième inefficacité apparaît notamment lorsque l'assureur complémentaire ne peut pas disposer de cette information, et quand le taux de prise en charge de l'assurance obligatoire est faible.

En matière d'information génétique, deux éléments interviennent pour remettre en cause le résultat de Hirshleifer [1971] sur la valeur individuelle de l'information. Le premier facteur se rapporte aux possibilités de s'investir dans des actions de prévention afin de réduire le risque ou l'ampleur des conséquences de la maladie. Nous avons montré dans la section précédente que les informations issues des tests génétiques permettent aux personnes de modifier leur comportement et de calibrer leur effort de prévention en fonction de leur risque. Dans ce cas, l'acquisition de l'information a une valeur de décision, au sens où elle permet à l'individu d'en tirer un bénéfice médical associé à une meilleure gestion des risques. En conséquence, si le test permet de s'engager dans des actions de prévention, toute personne averse au risque décide d'acquérir l'information génétique lorsque l'utilité des gains est au moins aussi élevée que la désutilité des coûts. Ainsi, la décision d'entreprendre un test génétique dépend de l'efficacité et du coût de la technologie de la prévention. Les préférences comportementales des assurés constituent le second facteur conditionnant l'acquisition de l'information par le biais d'un test génétique. Il existe en effet des coûts et des bénéfices psychologiques à recevoir une information sur le risque de santé. Ainsi, Hoel *et al.* [2006] et Bardey *et al.* [2017] mettent l'accent sur le

fait que les individus ont des préférences comportementales différentes vis-à-vis de l'information génétique. Deux catégories d'individus peuvent être observées en pratique. D'une part, il y a ceux qui veulent savoir indépendamment des conséquences économiques, lesquels sont qualifiés dans la littérature d'individus exprimant une répulsion au hasard. D'autre part, il y a ceux qui préfèrent ne pas savoir, pour lesquels cette information aurait des coûts psychologiques, et qui sont donc plus heureux derrière le voile de l'ignorance. Ainsi, même si le test ne permet pas de s'engager dans des actions de prévention, un individu peut entreprendre un test génétique alors même que la valeur de cette information au sens de Hirshleifer est négative.

Par ailleurs, la plupart des tests génétiques n'apportent pas d'information précise et absolue. Ils peuvent entraîner des erreurs de type I (faux positif) et de type II (faux négatif). Par conséquent, lorsqu'une personne entreprend ces tests, elle obtient une information relative, c'est-à-dire un résultat positif avec une certaine probabilité et un résultat négatif avec la probabilité complémentaire. La théorie économique traite la question de l'information relative par le biais du concept d'aversion à l'ambiguïté. Ce concept ne se focalise pas tant sur le risque lui-même que sur l'incertitude relative à la distribution de ce risque. En conséquence, chaque résultat du test conduit à une révision des croyances chez l'individu. En ce sens, un test génétique correspond *ex ante* à une loterie entre une détérioration ou une amélioration des croyances. Lorsque le test n'est pas réalisé, l'individu averse à l'ambiguïté se soustrait à cette incertitude qui affecte négativement son utilité. C'est dans ce contexte que Hoy *et al.* [2014] ont démontré qu'une personne averse à l'ambiguïté ne fera pas le test, notamment lorsque la prévention est inefficace. Ce résultat découle du fait que la décision d'acquérir l'information génétique expose l'individu à une distribution de probabilité dont la variance est plus élevée alors que la moyenne reste identique.

1.2.2.2 Tests génétiques et antisélection

Nous avons vu dans la section précédente comment la discrimination génétique, résultant de la prise en compte de l'information issue des tests génétiques dans la définition des contrats, décourage cette acquisition d'information. Nous étudions à présent un autre phénomène, l'antisélection, qui apparaît lorsque l'information acquise à l'issue d'un test peut ne pas être révélée à l'assureur. Nous résumons dans un premier temps les principaux enseignements de la littérature sur l'antisélection, puis nous exposons les spécificités liées aux tests génétiques.

L'antisélection se produit sur le marché de l'assurance santé lorsque les individus détiennent une information sur leur niveau de risque qui n'est pas connue de l'assureur. Ce phénomène est amplifié lorsque les assureurs ne peuvent pas intégrer le résultat des tests génétiques dans le calcul de la prime d'assurance, alors que les individus peuvent décider d'acquérir cette information. Supposons qu'il existe deux catégories de risque dans la population : bas ou haut. Si la prime d'assurance reflète le niveau du risque moyen au sein de la population, la prime est trop onéreuse pour les individus qui savent qu'ils présentent le risque le plus faible. Dans un système où l'assurance santé n'est pas obligatoire, l'absence de tarification en fonction des risques individuels, ou tout du moins une tarification trop éloignée des risques individuels, peut donc aboutir à un équilibre où les "mauvais risques", qui contribuent à augmenter le niveau de la prime d'assurance, "chassent les bons" qui préfèrent ne plus s'assurer. Pour y remédier, les assureurs peuvent jouer sur deux variables - le prix et la quantité d'assurance. Deux principales notions d'équilibre sont proposées dans la littérature en présence d'antisélection. Rothschild et Stiglitz [1976] proposent

un concept d'équilibre séparable à condition que la proportion des individus à bas risque dans la population ne dépasse pas un seuil théorique. En supposant que tout entrant potentiel anticipe le retrait des contrats éventuellement déficitaires, Wilson [1977] propose un concept d'équilibre caractérisé par un "contrat mélangeant" souscrit à la fois par les bas risques et les hauts risques lorsque la proportion des bas risques est suffisamment élevée dans la population¹². Dans le modèle de Rothschild et Stiglitz [1976], les contrats mélangeants sont exclus puisque l'équilibre est atteint lorsqu'il n'existe pas de contrat hors équilibre qui, s'il était offert, générerait un profit.

L'équilibre mélangeant est défini par un contrat unique souscrit par les deux catégories de risque. Ce contrat offre le même niveau de remboursement à tous les individus et son prix est calculé en fonction du risque moyen dans la population. Le contrat mélangeant permet *ex ante* une subvention des bas risques vers les hauts risques et des individus bien portants vers les malades. Cet équilibre est obtenu sur les marchés d'assurance à condition que la redistribution effectuée par l'assurance ne soit pas trop importante¹³. En d'autres termes, les individus à bas risques acceptent de subventionner les hauts risques si ces derniers sont peu nombreux dans la population.

En revanche, lorsque la proportion des hauts risques est élevée, le menu de contrats séparateurs à la Rothschild-Stiglitz est proposé car un contrat mélangeant avec subventions croisées n'est plus tenable. Dans ce contrat d'équilibre, les individus s'auto-sélectionnent selon leur risque. Les hauts risques obtiennent une couverture complète pour une prime élevée ; les bas risques ont, quant à eux, accès à une couverture incomplète à bas coût (Rothschild et Stiglitz [1976]). Lorsque cet équilibre est atteint, les individus sont discriminés arbitrairement en fonction de leurs caractéristiques

généétiques et les bas risques ne peuvent pas obtenir une couverture complète.

Pour mieux saisir les enjeux des tests génétiques sur les marchés d'assurance santé en présence d'antisélection, il faut s'intéresser à la façon dont l'acquisition de l'information peut affecter la nature des contrats à l'équilibre du marché. En effet, les tests génétiques peuvent affecter le marché de l'assurance santé principalement par le biais de leur impact sur l'intensité de l'antisélection, c'est-à-dire en influençant la proportion des individus informés dans la population. Actuellement, comme les tests génétiques ne sont pas très répandus, la proportion des individus informés reste très faible (voir Hoy *et al.* [2014]). En outre, on n'observe pas en pratique des contrats séparateurs auto-sélectifs mais des contrats mélangeants dans lesquels les individus qui n'ont pas fait de tests subventionnent ceux qui ont acquis l'information et qui sont à haut risque, lorsque les individus à bas risque informés peuvent sortir de ce contrat ¹⁴. Ainsi, dans le cas où l'assurance santé n'est pas obligatoire, le développement des tests génétiques peut affecter à terme le marché de l'assurance santé par une hausse de la proportion des individus informés. Si la proportion des individus informés à haut risque devient importante et que les contrats mélangeants observés ne deviennent plus tenables en présence d'antisélection, cela pourrait engendrer des problèmes de discrimination, mais également une perte de surplus en raison de la couverture incomplète dont bénéficient les bas risques. Enfin, on observe que les phénomènes d'antisélection n'affectent que transitoirement les assureurs, et que les conséquences défavorables de l'antisélection sont répercutées uniquement sur les assurés, puisqu'elles conduisent à une couverture incomplète des assurés. Dans la section suivante, nous présentons les différentes régulations en vigueur avant d'analyser l'incitation individuelle à acquérir l'information dans chacune d'entre elles.

1.2.3 Différentes régulations de l'information génétique

Pour résoudre le problème de la discrimination génétique et de l'antisélection, les pays ont adopté différentes régulations qui ont des impacts variables sur la décision de faire le test.

Les régulations des marchés d'assurance santé actuellement en vigueur diffèrent selon qu'elles cherchent à éviter l'intensification des problèmes d'antisélection dont peut pâtir le marché de l'assurance santé, ou plutôt qu'elles visent à atténuer, voire éliminer, la discrimination des risques auxquels font face les assurés.

La littérature sur la régulation des marchés d'assurance santé en matière de tests génétiques permet de distinguer quatre types de régulation de l'information génétique : l'obligation de divulgation, le droit ou la règle de consentement, l'interdiction stricte, et le laissez-faire¹⁵. Le tableau n°1.1 décrit les règles en vigueur dans plusieurs pays. Ceux adoptant des lois strictes interdisant l'utilisation des tests génétiques dans la définition des contrats d'assurance santé visent à protéger les individus contre la discrimination génétique¹⁶. Par ailleurs, la réglementation concerne aussi les autotests proposés par des laboratoires agréés auxquels les individus peuvent avoir accès sans passer par un professionnel de santé. Cette possibilité offerte aux individus est susceptible de renforcer les problèmes d'asymétrie d'information.

Table 1.1: Régulation selon les pays

Pays	Disposition légale	Forme de régulation	Autotests
États-Unis	Genetic non-discrimination act (Gina) votée en 2008 ¹⁶ .	Interdiction formelle ou stricte	Oui
France	Loi n° 2011-814 du 7 juillet relative à la bioéthique.	Interdiction formelle	Non
Royaume-Uni	Absence de loi, moratoire signé par les compagnies d'assurance jusqu'en 2019.	Interdiction formelle	Oui
Allemagne	Genetics Diagnostics Act adopté en avril 2009.	Interdiction formelle	Non
Australie	Genetechnik law, 1994.	Laissez-faire	Oui
Suisse	Loi fédérale sur l'analyse génétique humain (LAGH) du 8 octobre 2004.	Règle de consentement	Oui
Nouvelle Zélande	Absence de loi spécifique interdisant son utilisation par les assureurs.	Obligation de divulgation	Oui

L'obligation de divulgation est une réglementation en vertu de laquelle les individus doivent révéler aux assureurs les résultats de tests génétiques réalisés antérieurement. Cependant, les assureurs ne peuvent exiger que les individus entreprennent des tests avant de souscrire un contrat d'assurance. De ce fait, la prime peut être ajustée en fonction du statut informationnel et du risque de santé de l'assuré. Cette régulation permet alors d'éliminer l'antisélection tout en laissant l'opportunité aux assurés d'échapper à la discrimination des risques s'ils ne font pas de test. Néanmoins, les assurés qui ne veulent pas être soumis à ce processus de discrimination doivent renoncer à l'information médicale véhiculée par les tests. Par ailleurs, il peut s'avérer difficile dans la pratique de faire respecter l'obligation de

révéler les résultats des tests génétiques et de vérifier l'information, compte tenu du secret médical auquel sont soumis les professionnels de santé.

La règle de consentement permet aux assurés de faire un test et d'en utiliser les résultats à leur convenance. Ils peuvent ainsi les garder secrets ou communiquer les résultats à leurs assureurs, lesquels sont alors autorisés à en tenir compte dans leur tarification. Cette régulation protège en effet les assurés du risque de discrimination d'un mauvais héritage génétique, alors que les assureurs sont exposés au problème de l'antisélection. Les personnes informées à bas risque ont une forte incitation à transmettre l'information pour bénéficier d'une prime actuariellement équitable, tandis que celles dont le résultat est positif n'ont aucun intérêt à le faire et ont donc intérêt à se faire passer pour non-informées. En d'autres termes, cette régulation restreint la discrimination des risques puisque les assurés sont protégés des conséquences négatives de cette discrimination, i.e. ils sont discriminés seulement quand cela leur est favorable (en informant l'assureur de leur faible risque santé). En revanche, cette régulation génère des problèmes d'antisélection, puisque le contrat offert aux individus qui n'auraient pas effectué de test peut aussi être souscrit par ceux dont le résultat du test est défavorable.

Le laissez-faire, qui correspond concrètement à une absence de régulation, permet aux assureurs d'exiger de la part de leurs assurés qu'ils réalisent un test génétique et qu'ils communiquent les résultats de ce test. Cette régulation, qui est peu appliquée, permet de protéger le marché des conséquences de l'antisélection mais la discrimination génétique est susceptible d'y atteindre son paroxysme.

Enfin, le régime d'interdiction stricte n'autorise pas les assureurs à prendre en compte l'information génétique dans la définition des contrats d'assurance. Les

assureurs ne sont pas autorisés à demander aux individus d'effectuer des tests génétiques et ne peuvent pas utiliser les résultats des tests effectués pour élaborer les contrats d'assurance. Cette régulation confère un caractère privé à l'information génétique et engendre des comportements stratégiques de la part des individus. À première vue, le risque de discrimination est totalement éliminé, mais les problèmes d'antisélection peuvent mettre à mal le fonctionnement des marchés d'assurance santé privés concurrentiels. Cette régulation peut en effet amener les assureurs à discriminer les risques pour faire face à l'antisélection, en définissant des classes de risque et ainsi cumuler à la fois les inconvénients de la discrimination et de l'antisélection pour les assurés.

Comme nous l'avons évoqué précédemment, en fonction de la proportion des individus informés à haut risque, différents types de contrat peuvent être proposés aux individus qui impactent négativement leur surplus. La plupart des pays interdisent aux assureurs d'utiliser l'information génétique pour la définition des contrats d'assurance. L'interdiction de discriminer est généralement justifiée sur la base des considérations éthiques et morales.

1.2.4 La valeur individuelle de l'information dans les différentes régulations

La valeur individuelle de l'information génétique se définit comme la différence entre l'utilité espérée obtenue par un assuré lorsque le test est réalisé et celle obtenue lorsque l'individu n'entreprend pas le test. Un individu décide d'entreprendre un test génétique à condition que son utilité espérée quand l'information est acquise soit au moins aussi élevée que celle qu'il en retire lorsque l'information n'est pas

acquise en considérant également la réglementation en vigueur et ses préférences.

Pour déterminer la valeur individuelle de l'information génétique dans les différentes réglementations nous considérons un marché d'assurance concurrentiel sur lequel les assureurs proposent des contrats d'assurance aux individus. Des individus averses au risque ont la possibilité d'acquérir l'information génétique et de la transmettre avant de souscrire un contrat d'assurance à l'assureur si la réglementation le permet, et ils peuvent éventuellement entreprendre des actions de prévention qui sont observables. On suppose que, si l'information n'est pas acquise, l'individu et l'assureur perçoivent un risque moyen¹⁷. L'assureur propose alors un contrat d'assurance dont la prime est calculée en fonction du risque moyen pondéré dans la population. Par ailleurs, si l'information est acquise, l'individu choisit un contrat d'assurance selon son risque.

Dans le régime d'obligation de divulgation, les individus peuvent obtenir un contrat d'assurance selon leur type et leur statut informationnel. L'assureur propose alors trois contrats d'assurance avec couverture complète en fonction du statut informationnel, du risque de l'assuré et du choix de prévention¹⁸. Lorsque les individus décident de ne pas faire le test génétique, ils choisissent le contrat qui leur est destiné, c'est-à-dire celui dont la prime reflète le risque moyen pondéré des individus non informés. En revanche, s'ils font le test, ils obtiennent pour une probabilité donnée le contrat avec couverture complète destiné aux hauts risques ou le contrat d'assurance complète adressé aux bas risques pour la probabilité complémentaire. En d'autres termes, ils obtiennent un contrat d'assurance complète dont la prime est ajustée en fonction de leur risque maladie et de leur choix de prévention. Les individus décident d'acquérir l'information à condition que l'utilité du gain de la prévention

soit au moins aussi élevée que les coûts qui comprennent la désutilité de l'effort et la perte d'utilité liée au risque de classification. Dans ce contexte, si le test permet d'entreprendre de la prévention secondaire observable par les assureurs, Barigozzi et Henriot [2011] ont montré que les individus font le test uniquement lorsque le risque de classification (i.e. le surcoût de prime lié à l'écart entre les probabilités de survie du risque) est faible et/ou l'utilité du gain de la prévention est élevée, pour des coûts de prévention ni trop élevés, ni trop faibles. Lorsque les individus ont la possibilité d'entreprendre des actions de prévention primaire observables ou non par les assureurs, Bardey et De Donder [2013] et Peter *et al.* [2017] ont mis en évidence des résultats similaires et montrent qu'ils entreprennent le test si les gains de la prévention sont élevés et pour des valeurs peu élevées et pas trop faibles du coût de l'effort. En particulier, les résultats de Bardey et De Donder [2013] révèlent que la valeur de l'information est une fonction non monotone du coût de la prévention. En effet, la valeur individuelle maximum est plus élevée avec aléa moral que sans aléa moral. Plus précisément, lorsque le coût de la prévention est faible, les individus réalisent des actes de prévention même lorsqu'ils n'ont pas fait de test génétique. En revanche, les choix de prévention ne sont pas optimaux. Dans ce cas, la valeur de l'information augmente avec le coût de l'effort de prévention qui peut être évité en cas de test négatif. En revanche, lorsque le coût de la prévention est tel que les individus réalisent des actes de prévention seulement en cas de test positif, la valeur individuelle de l'information diminue avec le coût de la prévention. Enfin, l'impact de l'aléa moral sur la valeur individuelle de l'information est ambigu. Ce résultat découle du fait que l'aléa moral affecte de manière différente l'utilité des individus selon leur risque, et, en outre, son impact varie selon que le coût de l'effort est faible ou intermédiaire.

Dans le régime de la règle de consentement, les personnes informées à bas risque ont une forte incitation à transmettre l'information pour bénéficier d'une prime actuariellement équitable, tandis qu'une personne dont le résultat est positif n'a aucun intérêt à le faire. L'assureur doit alors anticiper la décision des individus afin de proposer des contrats d'assurance. Si les assureurs anticipent que tous les individus vont entreprendre les tests et proposent deux contrats d'assurance pour les deux catégories de risque supposées, la valeur individuelle de l'information est positive. En effet, si les individus restent non informés, ils obtiennent le contrat destiné aux personnes à haut risque. Ainsi, en acquérant l'information, les individus à bas risque obtiennent un contrat d'assurance fournissant une utilité plus élevée et cela permet à tous les individus d'ajuster leur effort de prévention en fonction de leur risque. Par ailleurs, si les assureurs anticipent que tous les individus ne vont pas entreprendre les tests et proposent un contrat mélangeant dont la prime est fonction du risque moyen de la population et qui est souscrit par tous les assurés, les individus ont intérêt à acquérir l'information afin de calibrer leur effort de prévention en fonction de leur risque. Ainsi, à l'équilibre, tous les individus entreprennent les tests. Les assureurs anticipent alors parfaitement la décision des agents et proposent un contrat à bas risque à ceux ayant révélé un résultat favorable et un contrat dont la prime reflète un risque élevé à tous les autres. La valeur individuelle de l'information est positive, car tous les individus non informés ont intérêt à entreprendre le test.

Dans le régime d'interdiction stricte, pour déterminer le contrat qui sera proposé aux individus, on utilise le même raisonnement que celui de la régulation du droit de consentement à la seule différence que, lorsque l'information est acquise, l'assureur n'est pas autorisé à l'intégrer dans le calcul de la prime en raison de son caractère privé et de l'interdiction formelle qui s'impose. Par conséquent, l'assureur ne

peut pas distinguer les individus et l'antisélection se manifeste sur le marché. Pour y faire face, l'assureur propose alors soit des contrats révélateurs, soit un contrat mélangeant en fonction de la distribution des risques dans la population. Ainsi, les individus vont acquérir l'information quelle que soit l'anticipation des assureurs. La valeur individuelle de l'information est positive quel que soit le contrat préféré par un individu non informé dans le menu de contrats proposé. À l'équilibre, en raison de l'antisélection, les individus à bas risques ne peuvent pas obtenir un contrat d'assurance avec une prime actuarielle et couverture complète.

En conclusion, la valeur individuelle de l'information diffère selon la réglementation en vigueur. Celle-ci est plus élevée dans le régime du droit de consentement que dans celui d'obligation de divulgation et d'interdiction stricte. Peter *et al.*[2017] étendent ces résultats dans le cas où les individus peuvent entreprendre des actions de prévention primaire qui ne sont pas observables par l'assureur. En effet, dans le régime du droit de divulgation, les individus peuvent obtenir un contrat d'assurance selon leur type et leur statut informationnel. En revanche, si la régulation limite la transmission de l'information à l'assureur (règle de consentement et interdiction stricte), ce dernier n'est plus en mesure d'une part de proposer des contrats en fonction du statut informationnel et du risque de l'assuré et, d'autre part de s'assurer que chaque individu choisisse le contrat qui lui est destiné. Dans le régime de la règle de consentement, les assureurs proposent un contrat avec couverture complète correspondant au risque faible à ceux ayant révélé un résultat favorable et un contrat dont la prime reflète un risque élevé à tous les autres, alors que dans la régulation d'interdiction stricte, lorsqu'un menu de contrats révélateurs est proposé aux individus, les bas risques obtiennent une couverture partielle. Dans la régulation de la règle de consentement, les personnes non informées ne parviennent pas à obtenir

un contrat d'assurance avec une prime actuarielle satisfaisante alors qu'en faisant le test ils peuvent bénéficier d'un contrat d'assurance en fonction de leur risque. Contrairement au régime de la règle de consentement, les personnes dont le résultat du test se révèle négatif obtiennent une couverture incomplète dans le cadre de la réglementation d'interdiction stricte. Ainsi, l'incitation à faire les tests génétiques est plus élevée dans la réglementation de la règle de consentement. Barigozzi et Henriet [2011] ont mis en évidence des résultats similaires lorsque les individus peuvent entreprendre des actions de prévention secondaire qui sont observables par les assureurs. Cependant, lorsque la régulation limite la transmission de l'information (droit de consentement et interdiction stricte), Barigozzi et Henriet [2011] introduisent un mécanisme incitatif pour éviter que les individus informés se fassent passer pour les autres. Pour inciter les individus à choisir le contrat qui leur est destiné, selon ce mécanisme, les assureurs dégradent la situation de certains types (bas risques et/ou non informés selon la régulation) afin d'assurer une contrainte d'autosélection.

Par ailleurs, dans le régime de laissez-faire dans lequel l'assureur peut demander à l'individu de faire les tests et de révéler les résultats, plusieurs études théoriques, dont celle de Barigozzi et Henriet [2011], arrivent à la conclusion que tous les assureurs vont exiger des individus qu'ils entreprennent les tests génétiques avant la souscription du contrat. En effet, si le test n'est pas coûteux, la meilleure stratégie des assureurs est d'imposer aux individus d'entreprendre les tests afin de proposer des contrats en fonction du risque individuel. En conséquence, la valeur individuelle de l'information sera positive si la technologie de la prévention est efficace, sinon elle sera négative. Barigozzi et Henriet [2011] considèrent des assureurs en concurrence qui, s'ils ne disposent pas de cette information, ne peuvent pas offrir un contrat en

fonction du risque individuel. Pour éviter de faire face à l'antisélection, les assureurs imposent aux individus d'entreprendre les tests génétiques. En conséquence, il n'y a aucun intérêt à calculer la valeur individuelle de l'information puisque la décision ne résulte pas du choix de l'individu. Ce résultat théorique manque toutefois de pertinence empirique puisque dans la pratique les assureurs ne demandent pas systématiquement aux individus d'entreprendre les tests génétiques avant de souscrire un contrat d'assurance. Dans deux pays qui appliquent une politique de type laissez-faire, à savoir le Canada (jusqu'en 2015) et l'Australie, les assureurs n'obligent pas les individus à faire des tests génétiques avant la souscription des contrats. Dans la pratique, les assureurs demandent aux individus d'entreprendre des tests à condition qu'ils aient des antécédents familiaux. Contrairement aux prédictions théoriques, la proportion d'individus ayant fait les tests prédictifs reste très faible, même dans les pays dans lesquels la discrimination génétique est interdite. Par exemple, Meiser et Dunn [2000] ont constaté que, pour la maladie de Huntington, le pourcentage des individus à risque ayant fait le test variait de 10 % à 20 % dans certaines villes britanniques. Dans une étude empirique, Lerman *et al.*[1996] trouvent que près de 60 % de la population étudiée ayant des antécédents familiaux de cancer du sein et/ou de l'ovaire ont refusé d'entreprendre un test génétique pour le gène BRCA1. Levy *et al.* [2011] constatent que seulement 30 % des femmes atteintes d'un cancer du sein acceptent d'entreprendre un test génétique pour déterminer le traitement le plus approprié. Cette différence entre prédictions théoriques et faits empiriques peut s'expliquer notamment par la non-prise en compte de l'hétérogénéité des individus *ex ante* dans ces modèles théoriques. Par exemple, certains individus observent un signal imparfait via leurs antécédents familiaux qui est le plus souvent déterminant dans la décision d'acquérir l'information. Ainsi, on pourrait s'interroger sur la validité externe de cette hypothèse généralement retenue dans ces travaux selon laquelle

les individus sont tous identiques et non-informés *ex ante* , ce qui les conduit à prendre tous la même décision.

De nombreux modèles théoriques considèrent en effet des individus identiques *ex ante* . Par conséquent, ils sont censés prendre la même décision vis-à-vis des tests génétiques. Dans certains travaux, lorsque la régulation limite l'accès à l'information (règle de consentement, et interdiction stricte), on suppose que les assureurs dégradent la couverture des personnes qui n'ont pas réalisé de tests génétiques afin d'assurer une contrainte d'autosélection (Barigozzi et Henriët [2011], Doherty et Thistle [1996]). En faisant les tests, les individus peuvent obtenir un contrat d'assurance fournissant une utilité plus grande. Ainsi, ces études arrivent à la conclusion selon laquelle tous les individus vont entreprendre les tests dans de telles régulations. À cet égard, on peut noter qu'introduire une contrainte incitative visant à empêcher les individus à haut risque ayant acquis l'information à se faire passer pour "non informés" peut apparaître contradictoire puisque tous les individus font les tests et sont donc informés¹⁹. D'ailleurs, Doherty et Posey [1998] et Peter *et al.* [2017] ont démontré qu'entreprendre les tests génétiques constitue une stratégie dominante. Ainsi, la croyance des assureurs doit être cohérente avec la décision des individus. En conséquence, les articles qui reposent sur les équilibres séparateurs et l'introduction d'une telle contrainte d'autosélection sur les individus non informés pourraient considérer le cas où les individus sont différents initialement et donc vont prendre des décisions différentes²⁰. Il serait également possible de considérer une population dans laquelle certains individus connaissent leur type et d'autres non, cette information étant connaissance commune mais où les assureurs ne peuvent pas observer le statut informationnel et le niveau de risque de l'individu. Sous ces conditions, il devient cohérent d'introduire une contrainte d'incitation pour éviter que les hauts risques

ayant acquis l'information et/ou les non informés se fassent passer pour d'autres²¹.

Une des principales limites de la plupart des études est de considérer un seul type d'équilibre (l'équilibre séparateur à la Rothschild-Stiglitz) en présence d'antisélection. Or, d'autres types d'équilibre peuvent être considérés, lesquels ont d'ailleurs plus de pertinence empirique. À notre connaissance, seuls les travaux de Crainich [2017], Hoy *et al.* [2003] et Bardey *et al.* [2017] analysent la décision des individus d'effectuer ou non des tests génétiques et leurs comportements de prévention dans le cadre de contrats mélangeants en présence d'antisélection. Hoy *et al.* [2003] proposent une étude empirique sur le marché de l'assurance santé en France dans le cadre d'une application au cas des facteurs de risque génétique de cancer du sein. Ils mettent en évidence le fait que le marché de l'assurance semble caractérisé par un équilibre mélangeant en raison de l'incidence limitée des mutations génétiques associées à un risque plus élevé dans la population féminine générale. Crainich [2017] a analysé la décision individuelle vis-à-vis des tests génétiques et les choix des individus en matière de prévention secondaire lorsque celle-ci est observable par les assureurs et lorsque tous les individus sont non informés *ex ante*, ont les mêmes préférences et prennent donc tous la même décision vis-à-vis des tests génétiques. Trois types d'équilibre (le contrat séparateur à la Rothschild-Stiglitz, le contrat séparateur avec subvention croisée au sens de Miyazaki et Spence et le contrat mélangeant au sens de Wilson [1977]) sont considérés sur le marché d'assurance dans le cas du régime d'interdiction stricte, conférant un caractère privé à l'information. Cette analyse montre que les individus préfèrent ne pas acquérir l'information et donc qu'une régulation stricte ne permet pas d'exploiter efficacement les bénéfices des tests génétiques si le contrat mélangeant est offert. En effet, lorsque les individus entreprennent le test génétique, ils obtiennent un contrat mélangeant avec couverture incomplète,

alors qu'ils obtiennent sinon un contrat d'assurance avec couverture complète. Par conséquent, tous les individus préfèrent ne pas entreprendre le test et fournissent un effort en fonction du risque moyen perçu.

Bardey *et al.* [2017], quant à eux, étudient la décision individuelle relative à l'information génétique, en prenant en compte l'hétérogénéité des individus en termes de préférences comportementales vis-à-vis de l'information et pour un test génétique dont le coût est non nul et varie entre les individus (ce coût contient à la fois le coût financier du test et le coût psychologique exprimé en termes monétaire). Ils analysent aussi la préférence des individus quant à ces deux régulations (règle de consentement et obligation de divulgation) qui réalisent des arbitrages différents entre l'antisélection et la discrimination génétique, dans le cadre d'un contrat mélangeant quand les assureurs ne disposent pas d'information sur le type génétique des individus. La prime de ce contrat mélangeant reflète la composition du groupe qui contient des individus non informés et ceux qui prétendent ne pas avoir fait le test, alors qu'ils l'ont réalisé et obtenu un mauvais résultat. Bardey *et al.* [2017] adoptent à la fois une approche théorique et expérimentale permettant d'étudier la manière dont les choix et la décision des individus sont affectés par l'intensité de l'antisélection, laquelle est mesurée par la proportion des individus, parmi ceux qui souscrivent le contrat mélangeant, qui découvrent qu'ils sont à haut risque. Cette analyse montre que les individus décident plus souvent d'acquérir l'information dans la régulation de la règle de consentement. La proportion des individus préférant la régulation de la règle de consentement à celle de l'obligation de divulgation dépend de l'intensité de l'antisélection et varie en fonction du coût du test. Cette proportion est une fonction non monotone du coût du test, puisqu'elle augmente de manière intuitive lorsque le coût décroît et diminue pour des coûts ni trop faibles et ni trop élevés.

Ces enseignements, bien que basés sur une construction théorique assez solide, ne s'intéressent essentiellement qu'à la valeur de l'information sans prendre en compte les préférences sociales de l'individu. La théorie des préférences sociales a permis de modifier le modèle standard de la fonction d'utilité, prenant en compte notamment l'altruisme des individus, pour lui permettre de reproduire les comportements prosociaux observés dans les expériences comme dans les faits stylisés. Cependant, toute la littérature portant sur les conséquences des tests génétiques sur les marchés d'assurance santé repose sur un cadre théorique de rationalité individuelle stricte. Or, ce cadre ne permet pas d'expliquer ces comportements, en particulier dans le cadre des tests génétiques. En effet, certains individus décident d'acquérir l'information génétique et de transmettre cette information pour alimenter la recherche médicale et des banques de données génétiques afin de développer la médecine de précision permettant d'optimiser les traitements médicaux. Par conséquent, la décision d'entreprendre un test peut ne pas répondre uniquement à un intérêt individuel mais à satisfaire les préférences sociales de l'individu. Ainsi, l'introduction d'un paramètre permettant de prendre en compte ces préférences permettrait d'analyser plus finement la décision et les choix de l'individu vis-à-vis des tests génétiques.

Pour résumer, cette partie montre que l'utilité des tests génétiques de prédisposition qui réside dans les actions de prévention peut être contrebalancée par la discrimination génétique, les préférences comportementales de l'individu, ainsi que le degré de précision du test. En outre, la décision d'acquérir l'information dépend du coût et de la technologie des actions de prévention, de la réglementation en vigueur et de la nature des contrats d'assurance sur le marché. En particulier, les individus

ont toujours intérêt à acquérir l'information dans tous les régimes de réglementation, sauf dans le régime d'obligation de divulgation. La connaissance des comportements individuels vis-à-vis des tests génétiques va donc nous permettre, dans la partie suivante, d'analyser les conséquences des différentes régulations sur le surplus social.

1.3 La valeur sociale de l'information dans les différentes régulations

Dans la première partie nous avons montré que la stratégie de révélation de l'information génétique aux assureurs influence les contrats d'équilibre et donc la valeur individuelle de l'information. Nous avons également montré que l'existence des actions de prévention efficaces élargit l'ensemble des équilibres possibles dans les différentes régulations. Nous allons maintenant étudier comment les quatre principales régulations affectent le surplus social. Cette analyse comparative de surplus social est effectuée d'un point de vue *ex ante*, c'est-à-dire dans un contexte où tous les individus sont initialement non informés, et avant la réalisation du risque.

Nous présentons d'abord les résultats de la littérature théorique adoptant le cadre d'un marché d'assurance concurrentiel. Nous montrons comment les différentes régulations impactent le surplus social, du point de vue *ex ante*. Nous analysons ensuite les conséquences des différentes régulations sur le surplus social dans le cadre d'un marché d'assurance mixte, avant d'étudier les inefficacités liées à un système d'assurance publique universelle. Enfin, nous étudions la proposition de Tabarrock [1994] qui serait susceptible de se rapprocher de l'allocation socialement optimale, limitant à la fois les problèmes liés à la discrimination génétique et à l'antisélection. En particulier, nous montrons que l'assurance génétique permettrait à la fois de max-

imiser le surplus social et de faire des choix optimaux de prévention, mais qu'elle présente des difficultés de mise en oeuvre.

1.3.1 Le cas d'un marché concurrentiel

La question de l'efficacité des différentes régulations de l'information génétique nécessite de préciser ce qu'est l'efficacité. On utilise usuellement le concept de surplus social pour mesurer l'efficacité du marché. Si l'on considère des assureurs en concurrence réalisant des profits nuls à l'équilibre, le surplus social correspond donc au surplus des consommateurs.

En information symétrique et en présence d'hétérogénéité des risques, l'équilibre du marché est caractérisé par des contrats d'assurance dont la prime reflète exactement le niveau de risque de l'assuré. Cet équilibre est efficace dans un contexte où les individus sont déjà informés de leur niveau de risque, au sens où les assurés souscrivent un contrat d'assurance avec couverture complète (Mossin [1968]). Avec un critère d'efficacité *ex ante* /dynamique, dans un cadre où les individus sont initialement non informés, pour un niveau de risque et un montant du dommage exogènes, une couverture complète en contrepartie d'une prime actuarielle en fonction du risque individuel n'est pas efficace car cela implique déjà une discrimination des risques qui conduit au résultat de Hirshleifer. En revanche, l'introduction d'une asymétrie d'information entre assurés et assureurs modifie la nature des contrats à l'équilibre sur le marché. Lorsque l'assuré est mieux informé sur son risque que l'assureur, ce dernier n'est plus en capacité de distinguer les individus au regard du risque encouru. En raison de l'antisélection, nous avons vu précédemment que les assureurs peuvent proposer un contrat mélangeant ou un menu de contrats séparateurs en fonction de la distribution des risques dans la population, impactant négativement le surplus

des individus.

L'analyse précédente des différentes régulations du marché de l'assurance en matière d'information génétique a démontré que l'on aboutit à deux types d'équilibre sur les marchés d'assurance santé pour des préférences individuelles homogènes. Le premier équilibre est tel que tous les individus décident d'entreprendre le test, choisissent le contrat qui leur est destiné et fournissent un effort de prévention en fonction de leur risque individuel de santé²². Cet équilibre émerge systématiquement sur le marché de l'assurance santé dans les régulations de la règle de consentement, du laissez-faire et de l'interdiction stricte. Dans le régime d'interdiction stricte, on suppose que la seule façon pour les assureurs de faire face à l'antisélection est de proposer un menu de contrats à la Rothschild et Stiglitz [1976] dans lequel les individus à bas risque bénéficient d'une couverture incomplète²³. Par ailleurs, ce type de contrat d'équilibre peut émerger également sur le marché de l'assurance santé dans le régime d'obligation de divulgation pour un coût d'effort qui n'est ni trop élevé, ni trop faible, et pour une technologie de prévention suffisamment efficace pour compenser la perte d'utilité liée à la hausse de la prime d'assurance pour les individus à haut risque.

Le deuxième type d'équilibre est celui selon lequel tous les individus choisissent de rester non informés, bénéficient d'un contrat d'assurance complète dont la prime reflète le risque moyen de la population et entreprennent des actions de prévention en fonction de ce risque perçu. Ce type d'équilibre émerge sur le marché dans le régime d'obligation de divulgation lorsque le coût de l'effort est faible ou élevé et l'efficacité de l'effort de prévention est faible et/ou le risque de classification est élevé. Afin de mener cette analyse de surplus social, reprenons quelques éléments du modèle

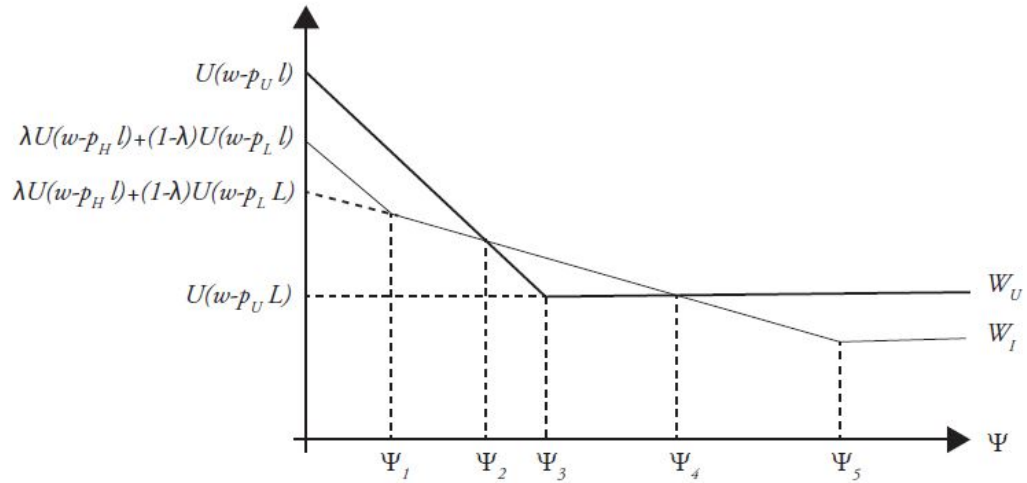
de Barigozzi et Henriët [2011] et leur graphique permettant de comparer le surplus social de ces deux équilibres en fonction du coût de l'effort de prévention secondaire observable par les assureurs.

Soit un marché d'assurance santé concurrentiel dans lequel les individus peuvent souscrire des contrats d'assurance et sont *ex ante* non informés. Le taux de couverture des contrats d'assurance est noté k , avec $0 \leq k \leq 1$. Les assureurs proposent d'abord des contrats, sauf dans le régime de type laissez-faire où ils peuvent demander aux individus d'entreprendre des tests génétiques avant de choisir un contrat. Puis, les individus décident de faire le test et d'en révéler, ou non, le résultat à l'assureur avant la souscription du contrat d'assurance. On considère deux catégories d'individus dans la population, une proportion à haut risque et l'autre à bas risque. En l'absence d'information génétique, on suppose que les individus et les assureurs perçoivent le risque moyen pondéré dans la population, considéré comme connaissance commune. Comme le test génétique fournit une information sur la probabilité individuelle de contracter une maladie, l'information transmise permet d'estimer si la personne possède un risque supérieur ou inférieur au risque moyen.

On suppose que les individus ont une dotation initiale w et des préférences caractérisées par une fonction d'utilité de type von Neumann-Morgenstern, $u(w)$, croissante et concave. Chacun fait face à une perte L s'il ne fournit pas d'effort et l si l'effort (dont le coût est Ψ) est fourni, avec $l < L < 0$. Les deux types de risque dans la population tels que les hauts risques sont associés à p_H et les bas risques sont associés à p_L , avec $0 < p_L < p_H < 1$. La proportion des assurés à haut risque dans la population est λ et celle des bas risques est $1 - \lambda$, avec $0 < \lambda < 1$. Le risque moyen perçu par un individu non informé est noté $p_U = \lambda p_H + (1 - \lambda)p_L$. Si les assureurs

proposent des contrats d'assurance avec couverture complète ($k = 1$) et prime actuarielle en fonction du statut informationnel et du risque encouru, l'utilité d'un individu non informé est $u(w - p_U l) - \Psi$ ou $u(w - p_U L)$ selon que l'effort est fourni ou non. Lorsqu'un individu entreprend le test, il obtient $(u(w - p_H L); u(w - p_H l) - \Psi)$ ou $u(w - p_L L); u(w - p_L l) - \Psi$, selon son risque et son effort de prévention.

La figure n°1.1 montre l'évolution du surplus social en fonction du coût de l'effort de prévention secondaire selon que l'information est acquise ou non. L'expression $W_U = u(w - p_U l) - \Psi$ correspond au surplus social lorsque les individus choisissent de ne pas entreprendre le test et exercent un effort de prévention, alors que $W_I = \lambda(u(w - p_H l) - \Psi) + (1 - \lambda)(u(w - p_L l) - \Psi)$ représente le surplus social qui est la somme pondérée de l'utilité individuelle lorsque le test est réalisé et l'effort fourni. On remarque sur le graphique que le surplus social W_U est au-dessus de W_I pour des coûts de l'effort peu élevés (inférieurs à Ψ_2 , le seuil audessous duquel l'utilité marginale de la prévention est inférieure à la désutilité marginale), ou élevés (supérieurs à Ψ_4 , la valeur au-dessus de laquelle la perte d'utilité liée au risque de classification dépasse l'utilité du gain net de la prévention).



Source : Barigozzi et Henriët [2011].

Figure 1.1: Le surplus social en fonction du coût de l'effort de prévention secondaire

Cependant, les choix de prévention des individus ne sont pas efficaces. Lorsque le coût de l'effort est peu élevé ou très grand, l'ignorance conduit à des choix de prévention inefficaces. Le surplus social W_I est en revanche plus élevé pour des coûts de prévention ni trop élevés et ni trop faibles (compris entre Ψ_2 et Ψ_4). Les actions de prévention sont alors efficaces.

Le tableau n°1.2 résume l'expression du surplus social dans les différentes régulations en fonction du coût de la prévention, lorsque celui-ci est tel qu'il est efficace pour une personne à haut risque de faire l'effort ($\Psi \in [\Psi_1; \Psi_5]$). La comparaison des différentes régulations permet d'observer le résultat suivant. Un système dans lequel l'information génétique peut être transmise domine en termes de surplus social toute régulation limitant sa révélation à l'assureur²⁴. En particulier, le régime d'obligation de divulgation domine faiblement tous les autres régimes puisque le test est entrepris uniquement lorsqu'il est socialement optimal. Peter *et al.* [2017] étendent ces résultats au cas où les individus peuvent entreprendre de la prévention

Table 1.2: Le surplus social sur un marché concurrentiel selon la régulation en fonction du coût de la prévention secondaire

Régulations	Coût de l'effort	Surplus social
Interdiction formelle ou stricte	$\Psi \in [\Psi_1; \Psi_5]$	$\lambda(u(w-p_H l) - \Psi) + (1-\lambda)[(1-p_L)u(w-kp_L L) + p_L \times u(w-kp_L L - (1-k)L)]$ avec $0 < k < 1$ le taux de couverture de l'assurance.
Règle de consentement et laissez-faire. Obligation de divulgation	Pour $\Psi \in [\Psi_1; \Psi_5]$ Pour $\Psi_2 \leq \Psi \leq \Psi_4$	$\lambda(u(w-p_H l) - \Psi) + (1-\lambda)(u(w-p_L L))$
Obligation de divulgation	$\Psi < \Psi_2$ ou $\Psi > \Psi_4$	$W_U = u(w-p_U)((1-\lambda)L + \lambda l) - \Psi$

primaire non observable par les assureurs. Dans le régime d'obligation de divulgation, les individus font le test lorsque le risque de classification est faible et/ou la technologie de la prévention est efficace et pour des valeurs intermédiaires du coût de l'effort. Toutefois, lorsque le test n'est pas réalisé, les choix de prévention ne sont pas optimaux.

Lorsqu'il n'est pas efficace d'acquérir l'information, le régime d'obligation de divulgation est le seul dans lequel l'individu n'entreprend pas les tests et obtient donc un contrat d'assurance avec couverture complète dont la prime reflète le risque moyen de la population. Par conséquent, le régime d'obligation de divulgation domine toutes les autres régulations. Lorsque le coût de l'effort est compris entre Ψ_2 et Ψ_4 , le surplus social est égal à W_I dans les régimes suivants : règle de consentement, obligation de divulgation et laissez-faire. Le surplus social dans le régime d'interdiction stricte est inférieur à celui des autres régulations, quel que soit le niveau du coût de l'effort, en raison de la couverture incomplète dont bénéficient les bas risques ($0 < k < 1$ correspond au taux de couverture des bas risques). Une régulation in-

terdisant strictement la révélation de l'information génétique impacte négativement le surplus social en raison de la distorsion sur la couverture dont bénéficient les bas risques. Peter *et al.* [2017] montrent que ces résultats peuvent être étendus lorsque le test est coûteux et pour des préférences comportementales différentes vis-à-vis de l'information.

Ces analyses permettent de tirer l'enseignement suivant. Aucune de ces quatre formes de régulation ne permet systématiquement aux individus de choisir l'intensité de l'effort de façon optimale et de maximiser en même temps le surplus social *ex ante* dans un système d'assurance concurrentiel. Lorsque le marché de l'assurance sélectionne les individus en fonction de leur risque, cela implique une perte de surplus social en raison de la nature de la fonction d'utilité des assurés. Les différentes régulations réalisent donc un arbitrage entre l'incitation à l'effort et la maximisation du surplus social *ex ante*.

Revenons à présent au contrat d'équilibre du marché concurrentiel dans le régime d'interdiction stricte. Dans ce régime, nous avons supposé que le marché est caractérisé par un équilibre séparateur à la Rothschild-Stiglitz, lequel n'est pas toujours garanti et observé dans la réalité. Lorsqu'il n'existe pas de solutions d'équilibre au sens de Rothschild-Stiglitz, nous avons vu que l'on peut recourir à d'autres concepts d'équilibres tels que l'équilibre mélangeant à la Wilson [1977] et l'équilibre séparateur avec subventions croisées au sens de Miyazaki [1977] et Spence [1978]. Ces deux concepts d'équilibre permettent de mieux rendre compte de l'efficacité des différentes régulations actuelles du marché car elles ont une pertinence empirique. Par exemple, Hoy *et al.* [2003] dans une étude empirique sur le cas des cancers du sein et Posey et Yavas [2007] dans le cadre d'une expérimentation ont

mis en évidence l'existence de subventions croisées sur le marché d'assurance santé en présence d'antisélection. En France, les compagnies d'assurance santé ne réalisent pas des profits nuls sur chaque contrat, mais utilisent une approche de subventions croisées, notamment les mutuelles et les complémentaires santé. *A priori*, l'équilibre séparateur à la Rothschild-Stiglitz n'est donc pas celui qui caractérise le marché de l'assurance santé. Si les études de Crainich [2017], Bardey *et al.* [2017] et Hoy *et al.* [2003] sont les seules jusqu'ici à considérer de tels équilibres en présence d'antisélection afin d'analyser la décision et les choix des individus, elles ne fournissent pas d'analyse comparative de l'ensemble des régulations observées en termes de surplus social. Quoi qu'il en soit, en présence d'antisélection, il y a toujours une perte de surplus social en raison de la baisse de la couverture globale d'assurance des individus.

D'après les conclusions de Bardey et De Donder [2017], lorsque les assureurs offrent un contrat mélangeant (dont la prime reflète la composition du groupe) à tous les individus qui n'ont pas révélé leur résultat, le régime de la règle de consentement serait *a priori* plus efficace puisqu'il permet d'éliminer la discrimination génétique en contrepartie d'un faible impact de l'antisélection. Ces conclusions sont susceptibles de changer si les tests génétiques deviennent très répandus. La section suivante examine les conséquences des tests génétiques sur le surplus social dans un système d'assurance maladie mixte, caractérisé par la combinaison d'une assurance maladie universelle obligatoire et d'une assurance complémentaire facultative.

1.3.2 Le cas d'un système d'assurance maladie mixte

Dans un système d'assurance maladie mixte, l'assurance universelle obligatoire n'a pas pour vocation de discriminer les individus en fonction de leur risque, ce que peut faire en revanche l'assurance complémentaire fournie par des assureurs privés. Ce système d'assurance maladie mixte équivaut au mode d'organisation de l'assurance maladie en France dans laquelle la Sécurité sociale, par le biais de l'assurance maladie, fournit une couverture de santé universelle incomplète à l'ensemble des assurés qui peuvent volontairement choisir une complémentaire santé²⁵.

Dans un système mixte, l'assurance universelle exerce deux effets opposés sur la décision de faire le test. Elle réduit, d'une part, le risque de classification car l'individu est discriminé uniquement sur la partie remboursée par l'assurance complémentaire. D'autre part, en raison de l'assurance universelle, une partie seulement des bénéfices de la prévention est internalisée par l'individu. Cela réduit donc l'intérêt individuel à faire de la prévention.

Hoel et Iversen [2002] étudient l'impact de l'interaction entre assurance universelle obligatoire et assurance complémentaire sur la valeur de l'information issue des tests génétiques dans le cadre d'une régulation stricte, conférant un caractère privé à l'information génétique, après avoir considéré le régime d'obligation de divulgation qui correspond au cas de l'information publique. Dans cette analyse, l'information a une valeur de décision car elle permet aux personnes ayant une prédisposition génétique de s'engager dans des actions de prévention primaire qui peuvent être (ou non) observables. Le surplus social est plus élevé dans le dernier régime que dans le premier, en raison des conséquences négatives de l'antisélection sur la couverture d'assurance des personnes à bas risque. En effet, lorsque les assureurs peuvent observer le statut informationnel, le résultat du test et les actions de prévention des

individus, ces derniers décident de faire le test lorsque le coût de la prévention et le risque de classification (liés à l'achat de l'assurance complémentaire) sont faibles ou lorsque l'efficacité de la prévention est élevée. En outre, en fonction du taux de couverture de l'assurance obligatoire, deux effets distincts se produisent. Un taux de remboursement élevé de l'assurance universelle obligatoire associé à une part d'assurance complémentaire faible amène certains individus à ne pas entreprendre les tests. Ce résultat découle du fait que seule une partie des bénéfices de la prévention est internalisée ce qui peut être insuffisant pour compenser le coût de l'effort lorsque le taux de prise en charge de la complémentaire santé est faible.

À l'inverse, un faible taux de remboursement de l'assurance universelle, associé à un taux élevé de la complémentaire santé, exerce un effet désincitatif sur la réalisation des tests. Il en résulte des choix de prévention inefficaces. En effet, lorsque l'individu réalise le test et que le taux de prise en charge de la complémentaire santé est élevé, il s'expose au risque de classification. L'individu préfère ne pas faire le test et souscrire une complémentaire santé avec une prime actuarielle basée sur le risque moyen de la population. Ce type d'inefficacité se produit davantage lorsque l'effort de prévention n'est pas observable par l'assureur.

Dans le cas où les assureurs ne peuvent pas observer le statut informationnel, le type et les actions de prévention des individus, un taux élevé de couverture de l'assurance complémentaire crée une forte incitation à entreprendre le test et les actions de prévention sont optimales lorsque le risque de classification est inférieur à un certain seuil. Il est à noter que Hoel et Iversen [2002] considèrent un taux de couverture de l'assurance maladie exogène. Par conséquent, leur modèle ne permet pas de caractériser la combinaison optimale des taux de couverture de l'assurance de base et de l'assurance complémentaire qui tire profit de l'information génétique de manière efficace.

En résumé, pour un taux de remboursement moyen de l'assurance universelle, une régulation autorisant la transmission de l'information à l'assureur domine toute régulation en limitant l'accès. Dans le régime d'interdiction stricte, puisque les individus à bas risque obtiennent une couverture incomplète lorsque l'équilibre du marché est caractérisé par un menu de contrats séparateurs à la Rothschild-Stiglitz, nous pouvons étudier l'évolution du surplus social en introduisant une assurance publique obligatoire. Dans ce contexte, l'analyse économique des marchés de l'assurance santé montre que l'introduction de l'assurance publique améliore le surplus des assurés au sens de Pareto par rapport au cas où il n'existe pas d'assurance universelle obligatoire (Dahlby [1981]). L'intuition de ce résultat est que les individus à haut risque bénéficient de la subvention croisée de l'assurance universelle obligatoire, alors que les bas risques bénéficient d'un taux de couverture plus élevé en raison de l'assouplissement du rationnement d'assurance, permettant une couverture globale plus élevée. Pour terminer l'analyse des conséquences des différentes régulations sur le surplus social, nous allons considérer le système d'assurance maladie universelle dans lequel les individus ne sont pas sélectionnés en fonction de leur risque.

1.3.3 Le cas d'une assurance maladie universelle

Dans un système d'assurance maladie universelle, la prime est indépendante du risque individuel. La décision individuelle d'acquérir l'information génétique n'expose donc pas l'individu au risque de classification. Une personne non informée a intérêt à acquérir l'information, pour un test non coûteux, afin de fournir un effort de prévention adapté à son risque. En revanche, cette acquisition d'information peut affecter les comportements d'aléa moral, qui impactent négativement le surplus social. En effet, en présence d'une assurance publique, les individus n'internalisant pas le coût

financier de la prévention secondaire peuvent effectuer des choix de prévention collectivement inefficaces. C'est dans ce contexte que Filipova-Neumann et Hoy [2014] étudient les conséquences de l'acquisition de l'information sur le surplus social et sur les choix de prévention secondaire, dans le cadre d'une assurance publique. Ils ne s'intéressent pas tant au processus d'acquisition de l'information qu'à son impact sur le surplus social et les comportements d'aléa moral, dans un contexte où les individus peuvent entreprendre un effort non observable permettant de détecter la maladie à un stade précoce. Cet effort permet de générer un bénéfice médical, mais ces actions de prévention sont coûteuses. Il s'agit de déterminer les conditions sous lesquelles l'acquisition de l'information génétique amène les individus à surinvestir (ou sous-investir) dans des actions de prévention secondaire, ce qui impacte négativement le surplus social. Selon leurs résultats, si le niveau d'effort individuel croît avec le niveau de risque, en revanche, le niveau d'effort socialement optimal ne croît pas forcément avec le niveau de risque. En particulier, lorsque le coût du traitement est plus élevé si la maladie est détectée à un stade précoce, tous les individus ont tendance à surinvestir dans des actions de prévention secondaire.

Par ailleurs, en raison de l'aléa moral relatif à l'intensité de l'effort de prévention secondaire, l'acquisition de l'information peut diminuer ou augmenter le surplus social. Son impact dépend de la nature de la fonction du coût global de prise en charge des assurés par rapport à la probabilité de tomber malade. Si ce coût croît avec la probabilité d'apparition de la maladie mais à un taux décroissant, alors l'introduction d'un test génétique améliore le surplus social. En revanche, si cette fonction est strictement convexe, le surplus social peut augmenter ou diminuer avec l'introduction d'un test génétique selon l'efficacité de l'effort de prévention secondaire des deux types de risque. L'intuition sous-jacente à ce résultat est la suivante. Un

test génétique correspond à un étalement à moyenne constante de la probabilité d'apparition de la maladie et donc, si la fonction de coût est convexe par rapport à cette probabilité, alors l'introduction d'un test génétique conduit à une augmentation du coût global de la prise en charge. Bien que l'acquisition de l'information amène les individus à fournir un effort en fonction de leur risque de santé, de ses bénéfices et de son coût du point de vue individuel, la non-intériorisation des coûts financiers de la prévention et du traitement en cas d'apparition de la maladie conduit à des choix de prévention socialement non optimaux. Par conséquent, si le coût croît plus vite que les bénéfices, cela affecte négativement le surplus social. Ainsi, le niveau d'effort de prévention secondaire de l'individu s'éloigne de l'optimum social. Pour se rapprocher du niveau socialement optimal, l'assureur peut limiter l'accès aux actions de prévention ou définir une politique incitative en fonction des instruments dont il dispose (subvention partielle, responsabilisation des individus etc.), en sachant que les agents n'internalisent pas le coût social.

Dans un système d'assurance maladie universelle où la prime est déconnectée du risque individuel de l'assuré, les individus considèrent le montant de la prime comme exogène. Supposons que la prévention soit efficace uniquement pour les individus dont le résultat est positif. En analysant l'impact de l'information génétique sur les choix de prévention dans un système d'assurance universelle, les résultats de Hoel et Iversen [2002] amènent à considérer que l'une des façons d'inciter les assurés à faire de la prévention est d'en compenser le coût. En effet, lorsque la prime d'assurance est calculée en fonction du risque moyen, la décision de faire le test n'expose pas l'individu au risque de classification. Cependant, la réduction de la prime qui devrait résulter de son effort de prévention ne peut être mise en oeuvre. Si le coût de l'effort n'est pas compensé et s'il n'y a aucune incitation, l'individu n'a aucun intérêt à en-

treprendre des actions de prévention et *in fine* à faire le test. Dans la pratique, il y a plusieurs limites à la mise en place d'une telle compensation. D'abord, les problèmes liés à l'introduction d'une compensation monétaire du coût de l'effort proviennent fondamentalement de la non-observabilité des actions de prévention. Ensuite, le coût de la prévention peut varier d'un individu à un autre. Ce coût a deux composantes. La première correspond au coût des examens médicaux dont l'individu peut bénéficier dans le cadre du dépistage. Dans la pratique, cette composante ne varie quasiment pas entre les agents et ces actes sont remboursés. La deuxième est relative au coût personnel lié aux activités de prévention telles que les exercices physiques, les régimes alimentaires et le temps consacré. C'est cette deuxième composante, variable d'un individu à l'autre, qui peut poser des problèmes de mise en oeuvre dans le cadre d'une compensation monétaire, d'autant plus que ce coût personnel de l'effort n'est pas toujours observable. Il devient alors difficile d'en avoir une évaluation parfaite et objective. Si le gouvernement décide de mettre en place une politique de compensation monétaire dont la valeur correspond au coût moyen de l'effort de prévention, tous les individus dont le coût est supérieur au coût moyen n'entreprennent pas les actions de prévention.

Dans un système d'assurance concurrentiel ou mixte, lorsque l'individu fait le test, il s'expose au risque de faire face à une prime élevée, notamment lorsque l'assureur peut disposer de cette information. En conséquence, un individu averse au risque est moins incité à faire le test en raison du risque de classification et donc les choix de prévention peuvent être sousoptimaux. En outre, il en résulte une perte du surplus social liée à la discrimination des risques. Dans la pratique, l'une des façons de lutter contre la discrimination génétique est d'adopter des lois strictes visant à interdire aux assureurs d'en tenir compte dans le calcul de la prime. Or,

une régulation stricte se fait au détriment du surplus social. Pour pallier ces problèmes, Tabarrock [1994] propose d'instituer une assurance génétique. Nous allons voir comment cette assurance est susceptible de réduire à la fois les conséquences de la discrimination génétique et de l'antisélection.

1.4 Les fondements de l'assurance génétique

Dans un système d'assurance privé concurrentiel, pour éviter la discrimination génétique, la solution la plus couramment adoptée par les pouvoirs publics est d'interdire aux assureurs d'utiliser cette information dans la définition des contrats. Or, une régulation stricte de l'information génétique engendre des comportements vis-à-vis de l'acquisition et de la transmission de l'information génétique qui pourraient, à terme, impacter négativement le surplus social. Une façon alternative de traiter la classification des risques est de constater que le problème vient de l'absence d'un marché explicite de l'assurance génétique. Selon Tabarrock [1994], lorsque l'information génétique a une valeur de décision, la solution consiste à créer un marché permettant de couvrir le risque d'être porteur d'un gène muté.

L'assurance génétique devrait alors être obligatoire avant de faire le test génétique. Cette assurance obligatoire serait souscrite par l'individu avant de réaliser le test et prendrait en charge l'accroissement de prime en cas de résultat défavorable du test. Par exemple, un gouvernement pourrait adopter des lois rendant illégale la réalisation des tests génétiques prédictifs par les individus s'ils ne détiennent pas d'assurance génétique. Ainsi, avant de souscrire un contrat d'assurance santé, toute personne non informée qui souhaite faire le test doit prendre une assurance génétique. L'assureur verse alors une indemnité à la personne seulement dans le cas où le résultat se révèle positif. La valeur optimale de l'indemnité de l'assurance génétique

est donnée par la différence entre le montant des dépenses engagées pour les deux types dans la population. Ces dépenses incluent à la fois le coût des traitements et le coût de la prévention. Dans le cas de prime actuarielle, le montant de la prime s'obtient en multipliant le montant de l'indemnité par la probabilité d'être à haut risque. L'assurance génétique peut être vendue sur des marchés privés concurrentiels comme les autres types d'assurance. Elle peut être aussi une assurance publique exclusive. Si l'assurance génétique est proposée à un niveau de prime actuarielle, tout individu averse au risque a intérêt à souscrire une assurance génétique car il obtient une utilité plus élevée.

Concrètement, l'assurance génétique ne peut pas être mise en place si la régulation confère un caractère privé à l'information. Lorsque l'individu fait le test, le résultat doit être révélé à l'assureur afin que ce dernier puisse verser l'indemnité prévue. Avec l'assurance génétique, une personne non informée choisira de faire le test, de révéler le résultat à l'assureur et de fournir l'effort de prévention puisque cela lui permet de maximiser son utilité. La mise en place de l'assurance génétique permettrait de limiter, à la fois, les conséquences de la classification des risques et les problèmes de l'antisélection. Dans une optique d'efficacité, l'assurance génétique doit se limiter aux tests utiles médicalement afin d'éviter le recours excessif aux tests prédictifs. Un test est utile médicalement lorsqu'il est possible de mieux se prémunir contre le développement ou d'atténuer l'ampleur de la maladie, et de la traiter. On observe dans la pratique que les firmes proposent un test global permettant de diagnostiquer le risque d'une centaine de maladies génétiques. Par conséquent, la valeur d'un test global peut être négative, bien que certains tests puissent avoir une valeur positive. Conformément aux recommandations de Bardey et De Donder [2013], il serait préférable de restreindre les tests génétiques à ceux dont la valeur individuelle

est positive.

Pour concrétiser l'assurance génétique, la plupart des travaux théoriques (Hoel et Iversen [2002] ; Bardey et De Donder [2013] ; Barigozzi et Henriet [2011]) supposent que tous les individus sont non informés *ex ante*. Avant l'achat d'une couverture de santé, cette assurance serait souscrite par l'individu avant de réaliser le test et prendrait en charge l'accroissement de la prime en cas de résultat défavorable du test. Dès lors qu'une personne ne perçoit que le risque moyen, elle a intérêt à souscrire un contrat d'assurance génétique avant de faire le test. Dans la pratique, certains individus détiennent une information (éventuellement imparfaite) sur leur risque de développer certaines maladies. Il peut exister en effet deux catégories d'individus *a priori* informés. Certaines personnes reçoivent un signal sur leur niveau de risque *via* les antécédents familiaux et d'autres personnes ont déjà entrepris des tests génétiques. Dans le premier cas, une personne peut être incitée à faire le test (par conséquent à souscrire une assurance génétique) afin d'éliminer toute incertitude. En revanche, les individus ayant déjà entrepris les tests génétiques ne pourront pas bénéficier de l'assurance génétique permettant de prendre en charge la hausse de la prime.

Si l'assurance génétique est *a priori* une alternative efficace pour lutter contre la discrimination génétique et les conséquences des comportements stratégiques résultant de l'asymétrie d'information, aucun pays n'a mis en place une telle assurance. À l'image de l'assurance contre la variation de prime, le développement de l'assurance génétique se heurte aux problèmes de mise en oeuvre d'ordre informationnel. En effet, il est difficile dans la pratique de garantir qu'une personne n'a pas déjà réalisé un ou plusieurs tests génétiques avant de souscrire une telle assurance. L'une des

principales difficultés à faire respecter ce principe est liée au fait que les individus ont la possibilité de réaliser des autotests, accessibles en ligne.

1.5 Conclusion

Cet article s'est intéressé aux implications économiques de la diffusion et de la régulation des tests génétiques prédictifs sur les marchés de l'assurance santé. Si l'utilité médicale de certains tests génétiques, qui réside dans l'amélioration de la prise en charge et de la gestion des risques de santé des patients, est largement reconnue, il n'y a pas de consensus sur les conditions dans lesquelles cette information peut être intégrée au sein des contrats d'assurance santé, comme en témoigne la multiplicité des lois au niveau international. Les différentes régulations observées répondent chacune à au moins un des objectifs suivants. Il s'agit, d'une part, d'éviter que le marché d'assurance ne pâtisse des conséquences de l'antisélection et, d'autre part, de protéger les assurés des assureurs qui pourraient imposer des primes prohibitives. Lorsque l'information a une valeur de décision, les résultats montrent qu'un régime dans lequel l'information peut être transmise à l'assureur domine strictement celui de l'interdiction stricte. En d'autres termes, le surplus social obtenu par les individus est plus élevé lorsque l'information génétique est transmise. Toutefois, le test génétique fournissant de l'information sur le risque maladie, l'utilisation d'une telle information permet de classer les types de risque. Pour cette raison, les individus ne sont donc pas incités à faire le test. Plus précisément, ils décident d'acquérir l'information lorsque le risque de classification est faible et l'utilité du gain net de la prévention suffisamment élevée.

Par ailleurs, nous avons montré que le régime d'obligation de divulgation domine tous les autres régimes de réglementation du point de vue du surplus social. Cependant, pour des coûts peu ou très élevés de l'effort de prévention, le surplus social est plus élevé lorsque le test n'est pas réalisé. En revanche, dans ce cas les choix de prévention ne sont pas optimaux. Par conséquent, en fonction du coût de l'effort de prévention, le régulateur doit choisir la régulation optimale du marché en arbitrant entre l'accroissement du surplus social *ex ante* et l'encouragement des individus à entreprendre des actions de prévention de façon optimale. Notons toutefois que ces résultats sont déterminés dans le cas où la seule façon pour les assureurs de faire face à l'antisélection est de proposer des contrats révélateurs. Or, dans la pratique, on n'observe pas toujours sur les marchés de l'assurance santé des contrats séparateurs à la Rothschild-Stiglitz, et en raison de l'impact psychologique négatif de l'information génétique, certains individus renoncent à acquérir l'information.

En outre, nous avons mis en évidence les limites des différentes régulations en vigueur, lesquelles ne permettent pas simultanément de maximiser le surplus social *ex ante* et d'encourager la prévention. La proposition de Tabarrock [1994] aurait donc pu constituer une alternative attractive permettant de limiter la discrimination génétique sans diminuer le surplus social, voire même l'augmenter. Cette proposition correspondant à la mise en place d'une assurance génétique obligatoire pour toute personne souhaitant entreprendre un test génétique, n'a cependant été mise en place dans aucun pays. Or, il serait possible d'obtenir une allocation de premier rang avec l'introduction de l'assurance génétique permettant d'indemniser les individus face à la hausse de la prime d'assurance. Le fait que les individus aient la possibilité d'entreprendre des tests avant la souscription d'une telle assurance compromet toutefois sa mise en place effective.

Enfin, nous avons montré que les tests génétiques de prédisposition peuvent être utiles pour l'individu à travers son impact sur la prévention. L'acquisition de l'information semble induire des choix de prévention de manière efficace à condition que le marché puisse séparer les bas risques des hauts risques. Toutefois, l'impact espéré sur la prévention est nuancé par de nombreux éléments tels que le risque de classification, l'impact psychologique, la perception du risque par les individus ainsi que l'incertitude liée au manque de précision des tests. Par ailleurs, d'autres questions se posent, parmi lesquelles, l'implication des préférences sociales des individus ainsi que l'incidence de l'évolution de leur risque dans le temps sur leurs choix et comportements. Il est essentiel de prendre en compte ces différents aspects dans toute analyse économique des tests génétiques sur les marchés de l'assurance santé pour mieux rendre compte de l'efficacité des différentes régulations.

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Notes de fin de chapitre

Notes

1. Les termes pharmacogénétique et pharmacogénomique sont souvent utilisés indistinctement. Selon un rapport de l'Académie nationale de médecine de 2009 (<http://www.academie-medecine.fr/publication100036098/>) la pharmacogénomique se définit comme l'utilisation de la connaissance du génome dans la recherche de nouveaux médicaments.
2. Voir <https://www.genetique-medicale.fr/la-genetique-l-essentiel/genetique-etmaladies-quels-liens/article/qu-est-cequ-une-maladie-genetique> pour plus de détails.
3. Voir <https://www.23andme.com/en-gb/health/> pour plus d'information.
4. Ce problème d'asymétrie d'information peut être le résultat d'une régulation interdisant aux assureurs de prendre en compte les résultats des tests génétiques dans le calcul de la prime d'assurance, notamment lorsque les individus ont la possibilité de faire des autotests (i.e. tests directement accessibles à tous sans passer par un professionnel de santé).
5. Ces deux types de prévention définis par l'OMS peuvent être complétés par une prévention sélective et ciblée (pour reprendre la classification de Gordon [1993]), rendue possible par la réalisation des tests génétiques. Ainsi, les actions de prévention primaire et secondaire doivent être ciblées pour les maladies génétiques.
6. L'épigénétique est la discipline de la biologie qui s'intéresse à la manière dont les gènes sont utilisés par les cellules et à la façon dont l'environnement modifie leur expression pour une séquence d'ADN donnée.
7. La perception du risque est l'un des facteurs sociocognitifs clés de la prédiction des comportements en santé. Des enquêtes montrent en effet que le risque de santé perçu par les individus peut différer considérablement du risque objectif (Slovic [2000] ; Andersson et Lundborg [2007]). Etner et Jeleva [2013] ont étudié l'impact de la perception des risques et de l'acquisition de l'information issue des tests génétiques de diagnostic sur les décisions des individus en matière de prévention primaire et tertiaire. Cette dernière se définit comme les actions de prévention permettant de limiter les risques de rechute ou d'aggravation après un traitement. Dans leur analyse, les personnes sont supposées être pessimistes puisqu'elles surévaluent le risque de la maladie. Ainsi, leur analyse prouve que l'individu choisit de faire le test s'il n'est pas trop pessimiste. L'acquisition de l'information accroît l'intensité de l'effort de prévention primaire mais peut détériorer le surplus des personnes qui surévaluent le risque.
8. Selon Bardey et Lesur [2005], si l'on considère que les individus prennent en compte l'impact de la détérioration de leur état de santé sur leur utilité, l'effet de l'aléa moral *ex ante* en matière d'assurance santé est limité, au sens où l'individu va entreprendre de la prévention même s'il bénéficie d'une couverture de santé quasiment complète, notamment pour les maladies graves.

9. L'objectif de cette chirurgie est d'intervenir avant l'apparition de la tumeur proprement dite. En pratique, il s'agit de retirer des éléments anormaux (organes, ou cellules) bénins, mais dont on sait que le risque d'évolution vers un cancer est important.
10. Voir Bardey et De Donder [2017] pour une synthèse de l'arbitrage entre discrimination des risques et antisélection, exacerbées par les tests génétiques sur les marchés de l'assurance santé.
11. Chiappori [2006] s'intéresse à l'ampleur de l'effet Hirshleifer en proposant une estimation empirique du degré d'aversion au risque des individus. Selon ses résultats, en raison de l'importance du coût des traitements de certaines maladies, la perte de surplus *ex ante* résultant de l'effet Hirshleifer peut être importante.
12. Un autre concept d'équilibre peut être considéré, celui identifié par Miyazaki [1977] et adapté au marché d'assurance par Spence [1978], qui modifie la nature de la contrainte de profit des assureurs. Dans ce cas, l'assureur propose un menu de contrats séparateurs dans lequel les bas risques subventionnent partiellement les hauts risques afin de bénéficier d'une couverture plus élevée. Crainich [2017] est la seule étude qui considère ce type d'équilibre en présence d'antisélection dans le cas des tests génétiques.
13. Dans un système d'assurance sociale, si les cotisations sont fonction des revenus, cela permet une redistribution de richesse entre les classes de risque en cas de corrélation négative entre morbidité et productivité (voir Rochet [1991] et Cremer et Pestieau [1996]).
14. Hoy *et al.*[2003] montrent que, sur la base d'une application du cas des facteurs génétiques liés au cancer du sein, l'équilibre mélangeant est plus susceptible d'apparaître sur le marché d'assurance santé en raison de l'incidence limitée des mutations géniques associées à un risque plus élevé. Voir également les travaux de Browne [1992] et Posey et Yavas [2007] concernant les contrats d'assurance observés sur les marchés.
15. Pour plus d'information sur les dispositions légales se rapportant à chacune de ces régulations, voir Otlowski *et al.*[2012] ; Joly *et al.*[2010] ; Hoy et Ruse [2005] ; Lemmens *et al.*[1998 et 2008] ; Viswanathan *et al.*[2007] ; Emmens *et al.*[2004] ; Munich Reinsurance Company [2004] ; European Commission [2004] ; Rafiq *et al.*[2015]. Peter *et al.* [2017] analysent une quatrième régulation intitulée code de conduite plutôt que le laissez-faire, appliquée en Afrique du Sud, dans laquelle l'assuré doit révéler son statut informationnel mais n'est pas tenu de révéler le résultat des tests réalisés.
16. Il est à noter que deux États américains (Mississippi et Washington) n'ont pas adopté cette loi fédérale interdisant la discrimination génétique sur le marché de l'assurance maladie (voir lien suivant : <https://www.genome.gov/about-genomics/policy-issues/Genetic-Discrimination>).
17. Crocker et Snow [1992] montrent que, si les agents n'ont initialement aucune information privée, le surplus social est plus élevé si tout le monde décide de ne pas acquérir l'information, et qu'il existe un contrat mélangeant unique. En revanche, la valeur sociale de l'information peut être positive ou négative en raison de l'observation d'un signal imparfait sur son risque. Les résultats de Crocker et Snow impliquent que, si les assureurs peuvent observer ou non le statut informationnel des individus et si les consommateurs n'ont aucune information privée préalable, alors la valeur privée de l'information est négative et les individus n'ont aucun intérêt à entreprendre les tests. Selon ces résultats, la valeur privée provient de l'observation *a priori* d'un signal imparfait. Par ailleurs, Doherty et Thistle [1996] prouvent que la valeur positive de l'information provient fondamentalement de la non-observabilité du statut informationnel des individus par les assureurs

et non de l'observation d'un signal imparfait par les individus sur leur risque.

18. On suppose qu'il n'y a pas de problème d'aléa moral. Bardey et De Donder [2013] proposent une analyse du régime de réglementation d'obligation de divulgation dans un contexte d'aléa moral sur les actions de prévention primaire (deux caractéristiques des actions de prévention sont prises en compte : leur coût et leur efficacité). Si l'acquisition de l'information permet de faire des choix de prévention optimaux, en revanche, les assurés bénéficient alors d'une couverture partielle. En conséquence, l'utilité sociale *ex ante* est plus faible avec l'aléa moral que sans l'aléa moral lorsque l'effort est fourni. L'impact de l'aléa moral sur la valeur individuelle du test est indéterminé puisque la non-observabilité des actions de prévention primaire affecte de manière non uniforme l'utilité des différents types de risque.
19. Nous remercions un rapporteur anonyme de la revue de nous avoir suggéré cette remarque.
20. Une telle hypothèse peut être justifiée en raison du fait que certains individus éprouvent de l'anxiété et de la peur à l'idée de faire des tests génétiques, dont l'intensité peut varier entre les individus (voir les travaux de Codori et Brandt [1994] ; Marteau et Croyle [1998] ; Codori *et al.* [1994] ; Caplin et Eliaz [2003] ; Caplin et Leahy [2004]).
21. En supposant que certaines personnes sont déjà informées de leur niveau de risque *ex ante*, Doherty et Thistle [1996] ont démontré que la valeur individuelle de l'information est positive à condition que l'assureur ne puisse observer le statut informationnel et le résultat du test *ex ante*.
22. On suppose que les actions de prévention sont observables et contractualisables.
23. Une hypothèse souvent retenue dans la littérature, mais qui n'a pas forcément de pertinence empirique. Certains travaux (comme Bardey *et al.*[2017] ; Hoy *et al.*[2003] et Crainich [2017]) considèrent d'autres types d'équilibre sur le marché en présence de l'antisélection.
24. Contrairement au marché de l'assurance santé, une régulation stricte de l'information génétique semble avoir un impact positif sur le surplus social en matière d'assurance-décès (Hoy et Polborn [2000] ; Polborn, Hoy et Sadanand [2006] ; Hoy et Witt [2007] ; et Viswanathan *et al.*[2007]). Les résultats d'une régulation stricte de l'information génétique sur le marché de l'assurance-décès diffèrent de ceux obtenus en matière d'assurance santé en raison de ses caractéristiques et de son mode de fonctionnement. En effet, le marché de l'assurance-décès est caractérisé par la non-exclusivité des contrats, puisqu'un assureur ne peut pas observer le montant de l'assurance achetée par un assuré, ce dernier ayant la possibilité de souscrire des contrats auprès de plusieurs assureurs. Par conséquent, il n'est pas possible de proposer des contrats prix/quantité en asymétrie d'information. Les assureurs proposent alors une prime uniforme pour tous les contrats. Le prix des contrats dans ce cas est proportionnel à la demande d'assurance.
25. Notons que pour les maladies de longue durée, les individus bénéficient d'une couverture complète par la Sécurité sociale.

Chapter 2

Genetic testing policies, health insurance market and reclassification

2.1 Introduction

In the era of predictive medicine, management of diseases is evolving into a more personal and individualized approach, as more data are available regarding clinical and genetic aspects. Predictive medicine involves assessing the risks of developing a disease through genetic testing. The revelation of this information could generate genetic discrimination on a private insurance market. In addition, from a long term perspective, individuals are exposed to premium variation when insurers can observe additional information after signing the contract. For example, the health conditions of individuals evolve over time. When this information is observed by insurers, they will take it into account at renewal of contract.

The epigenetic that is interested in modifications that are not coded by the DNA sequence shows us that the activity of the genes also depends on our lifestyle which can facilitate or prevent their expression. Thus, an individual who does not carry a genetic mutation can see the activity of his/her genes evolve because of his/her way of life. This phenomenon makes it possible to understand the evolution of an individual's health risk, which is expected to change in life. Therefore, on private health insurance markets, carrier and non carrier individuals of genetic mutation are exposed to reclassification at renewal of contract when insurers can observe additional information after signing the contract. If we consider that individuals who got sick in the previous period become riskier in the next period, they are then exposed to reclassification risk in a private health insurance market. Reclassification risk is defined as the premium variation individuals are exposed following the realization of the bad state of nature in the previous period.

Two main market solutions have been proposed in the literature to overcome

the problem of premium risk. In the first approach, Pauly et al. (1995) propose a market mechanism taking into account the risk of premium variation over time, in which insurers offer insurance contracts in similar terms to those defined at the time of the conclusion of the first contract. This is called in the literature renewable contracts with guaranteed premium. The implementation of these contracts requires a non-renegotiable bilateral commitment to ensure the proper functioning of the market in which the individual pays a surcharge in the previous period guaranteeing the insurance premium. However, such a constraint would limit the possibilities for policyholders to change insurance company and thus reduce insurers' incentive for efficiency. These insurance contracts exist in Australia and in Germany, in particular with regard to private insurance markets. The second approach, developed by Cochrane (1995), consists in proposing an ex ante insurance contract separately at an actuarial premium in the first period, which would pay an indemnity to policyholders whose risk has increased to compensate for the increase in the premium at the beginning of the next period. Unlike Pauly's proposal, individuals can change insurers in the second period. Moreover, the implementation of these contracts requires perfect observability of the level of risk of each insured to avoid problems of collusion between insured and insurers or opportunistic behavior of insurers. This type of contract does not exist in any country.¹

Beside the two market solutions, any health insurance system in which premium is independent of individual health risk aims to protect against premium variation. This solution involves an ex ante redistribution between the different risk group.

¹Bardey (2003) shows that, in the presence of moral hazard, it is not desirable to fully share the risk of premium variation. Kifmann (2001) characterizes a system of contract that pushes insurers to contract with high quality care providers in a managed care insurance system in which the insurer can imperfectly identify the type of individuals in the second period and can decide on the quality of care.

The insurance premium is higher than the expected loss of low risk individuals. The acceptability of this approach depends on the distribution of types in the population, the degree of risk aversion of individuals, and the ability of the system to keep everyone in the system. In particular, if health insurance is not mandatory, low-risk individuals may not have an interest to buy insurance.

In practice, in most countries health insurance markets offer short-term contracts to cover the financial risk of the disease over a year. Insureds are then unable to insure against the change of their risk over time.² Therefore, when the insurer has new information indicating that the risk associated with an individual has changed, and that he can terminate the contract, he will inevitably be encouraged to do so, or to adjust the insurance premium. This leads, particularly in the United States, some individuals suffering from a serious affection to be offered prohibitive premiums, or even are unable to insure their risk. In most developing countries, insurers exclude some pre-existing health conditions from health insurance coverage or are allowed to take them into account at renewal of contract. The fact that sick people face high health insurance premiums was an important argument for an additional regulation on health insurance market during the 2009-2010 health reform debate. In 2010, a major health reform has been adopted in US in order to guarantee access to more people on the health insurance market. According to this reform, insurance companies are prohibited from risk-adjusting premiums in the individual market. This can be considered as a regulatory way to remove reclassification risk. However, only 36 states at this time apply the Obama Care Act.³ Pashchenko and Porapakarm (2015) provide an empirical analysis of the welfare costs of reclassification risk in the

²Geoffard (2000) points out that insurers fail to insure this risk, especially because of unpredictable health expenditure and show that for the complementary health insurance in France a pooling contract would be more efficient.

³For further information, see the following link : <https://www.advisory.com/daily-briefing/resources/primers/medicaidmap>

US health insurance market. They measure welfare gains from introducing explicit insurance against this risk in the form of guaranteed renewable health insurance contracts. Their result shows that the average welfare gains from having access to guaranteed renewable contracts exceed 4 % of the annual consumption. In this paper, we consider a private competitive insurance market where there is no market solution to protect individuals from premium variation.

One of the benefits of genetic information is that it allows individuals to undertake or to adapt prevention actions according to their risk. Two types of prevention actions can be undertaken, primary and secondary preventions. Primary preventions are the activities to reduce the risk of sickness, while secondary prevention intends to reduce the extent of the disease. However, for some genetic diseases (for example the Huntington disease), there is no effective prevention action.

Regulations of genetic testing vary across countries.⁴ There are four types of regulations (*laissez-faire*, disclosure duty, consent law and strict prohibition) aiming to protect individuals from genetic discrimination or to avoid adverse selection on the market. The existing literature on the private value of genetic information shows that the acquisition of information allows individual to choose the optimal level of effort to reduce the probability of occurrence of the risk and the amount of the loss, when there is effective prevention action.⁵ Theoretical predictions reveal that, when there is no effective prevention action, individuals prefer to stay uninformed

⁴See Hoy and Ruse (2005), Viswanathan *et al.* (2007) and Otlowski *et al.* (2012) for a discussion of the different regulations.

⁵See Bardey and de Donder (2013) for the impact of the non observability of primary prevention action on the private value of the test; and see Crainich (2017) for the impact of the nature of the market equilibrium on secondary prevention action, when insurers cannot use genetic information to discriminate insureds.

in the disclosure duty regulation, whereas they will always undertake genetic test before signing the contract in the other regulations (Barigozzi and Henriet (2011)); Peter et al. (2017); Bardey and de Donder (2013)). In addition, a disclosure duty regulation strictly dominates strict prohibition and weakly dominates the others in terms of social welfare. In these papers, they consider a single period framework allowing to take into account the impact of genetic discrimination and prevention actions on individual decision. Thus, none of these works takes into account the risk of premium variation in their analysis. This paper tries to fill this gap. We consider a dynamic insurance market allowing to capture the impact of premium variation on the individual incentive to acquire genetic information. In addition, this framework allows to account for the effect of time preferences on agent decision to take genetic testing. Time preference is defined as the current value agents placed on returns in the future captured by the discount rates. The higher the time preference, the higher the value of the discount rate.⁶ There are an extensive works showing that time preferences play a major role in health behavior and on demand of medical tests (Chapman (2005) ; Chapman (2003) ; Andersen *et al.* (2013) ; Picone *et al.* (2004)).⁷

We follow Bardey (2003) by analyzing a private health insurance market in a two-period setting.⁸ Bardey (2003) analyzes how the risk of premium variation affects primary prevention actions when effort is not observable and determine the condition under which it is optimal to pool premium risk on the health insurance market. In particular, he shows that the desirability to pool premium risk depends on the level of the discount rate and the magnitude of the risk of premium variation.

⁶See Frederick *et al.* (2002) for a critical review on time discounting and time preference.

⁷See Lawless *et al.* (2013) for a review on the interplay between time preferences and health behaviour.

⁸Menegatti (2009) and Courbage and Rey (2012) analyze prevention decision in two periods setting but study respectively the impact of prudence and the impact of background risk on primary prevention activities in a non-insurance context.

As in Bardey (2003) and Bardey and De Donder (2013), we consider that there is effective prevention action to reduce the probability of loss. Unlike them, we consider only prevention action that is perfectly observable. In fact, some insurance companies propose insurance contracts with a prevention program whose tracking can be made via connected objects.

To the best of our knowledge, no work has addressed the issue of the implications of genetic testing policies on the health insurance market in a dynamic context. To do so, we add a second period to the Bardey and De Donder (2013) model with the possibility of reclassification, in the case where primary prevention action is perfectly observable. Bardey and De Donder (2013) focus on the impact of the technology (the cost and the efficiency) and the non-observability of prevention action on the decision to undertake genetic test as well as on social welfare in the disclosure duty regulation. In our paper, we analyze the individual incentives to acquire genetic information and provide a welfare analysis of the disclosure duty regulation in a dynamic context. We analyze a duty to disclose regime under which individuals must provide the results of any genetic tests to insurers. This latter cannot impose individuals to take the test, but can adjust the premium according to the individual informational status and risk. Our goal is to analyze how time preferences and the risk of premium variation in a dynamic insurance market affects the social welfare and the decision of individuals to take genetic testing before buying insurance contract. The results show that insureds may have incentive to acquire genetic information in disclosure duty regulation, even though there is no effective prevention action. The incentive to take genetic testing with reclassification is higher than without reclassification when the cost of effort is low or high, and differs according to the intensity of reclassification. The impact of time preferences on the incentive to gather

genetic tests depends on the technology of prevention action. Finally, we show that the social welfare is higher without reclassification than with reclassification.

The paper proceeds as follows. Section 2.2 outlines the model setup. Section 2.3 studies the insurance contracts, the prevention actions and the incentive of agents to take the test when they are not reclassified. In section 2.4, we describe insurance contracts, and analyze how reclassification impacts prevention decision and the incentives of individuals to gather genetic information. Section 2.5 provides a welfare comparison assessing the effect of reclassification on the ex ante utility of individuals. In section 2.6, we provide some concluding remarks.

2.2 Outline of the model

We adapt the model of Bardey and De Donder (2013) (hereafter BDD) to introduce the possibility of reclassification in a second period. We consider a population of individuals living two periods, facing the risk of being sick with health care expenditure $d > 0$ in each period. Individuals preferences are characterized by the same utility function v for the two periods, with $v(\cdot)$ increasing ($v'(\cdot) > 0$), concave ($v''(\cdot) < 0$) in wealth and $v'''(\cdot) > 0$. We denote by y the safe wealth in period 1 and period 2. Individuals buy insurance contracts to cover the loss d in each period on a private and competitive insurance market. We consider that as in Bardey [2003] the discount rate of individual utility of the second period is defined by β , with $\beta > 0$.

The population is made up of two types of individuals. A proportion λ of agents have a genetic background predisposing them to develop the disease (type- H) with a probability p_H^0 , while the complementary proportion $(1 - \lambda)$ has a lower probability to get sick (type- L), denoted by p_L , with $0 < p_L < p_H^0 < 1$ and $0 < \lambda < 1$. The type H or L of an agent can only be revealed by a genetic test. The test is assumed to be

costless and perfect, revealing with certainty the individual true type. When agents do not take the test (hereafter, type- U), we assume that they estimate their risk by the average probability to get sick in the population defined by $p_U^0 = \lambda p_H^0 + (1 - \lambda)p_L$, with $p_L < p_U^0 < p_H^0$.

Individuals are initially uninformed. In the first period, they decide to take or not the test. Then they decide to undertake or not prevention action and buy insurance contract. Prevention activities can take the form as special diet and sporting activities (to avoid obesity) or to stop smoking in order to reduce the risk to develop some cancers. We assume that prevention action is perfectly observable and costly. Some prevention actions can be tracking via connected objects. Others, such as smoking status, can be observed via a blood test in order to detect the presence of nicotine. The prevention action is binary, with $e \in \{0, 1\}$. The effort cost is normalized to zero if no effort is undertaken ($\phi(0) = 0$) and such that $\phi(1) = \phi$. It is measured in utility terms.⁹

We further assume as in BDD that effort is effective for only type- H agents, reducing their probability to get sick from p_H^0 to p_H^1 , while it is ineffective for type- L agents, i.e., $p_L^1 = p_L^0 = p_L$ with $0 < p_L < p_H^1 < p_H^0 < 1$. We define the prevention efficiency by Δ , with $\Delta = p_H^0 - p_H^1$. Δ can take any value in $[0, \bar{\Delta}]$, with $\bar{\Delta} = p_H^0 - p_L$. When $\Delta = \bar{\Delta}$, effort reduces the probability of a type- H agent from p_H^0 to the level of a type- L agent p_L , while when $\Delta = 0$, prevention has no impact. When a type- U agent undertakes effort, the probability to get sick is given by : $p_U^1 = \lambda p_H^1 + (1 - \lambda)p_L$, with $p_L < p_U^1 < p_H^1$ and $p_U^1 < p_U^0$. When type- U agents undertake or not effort, uninformed types L and H agents inherit from the prevention decision.

⁹Bardey (2003) considers that all individuals have the same probability of loss in the first period. Prevention action reduces the probability of loss only in the first period. A person who has suffered a loss in the first period is considered as high risk in the second period. Second period loss probabilities are exogenous.

In the second period, when agents are reclassified, the insurance contracts offered depend on the realization or not of the bad state of nature. We assume that individuals who got sick in the first period, regardless of whether they take the genetic test or not, will have a higher risk in the second period. In order to simplify, we assume that high-risk and low-risk agents who got sick in the first period have the same risk in the second period and obtain the same contract. The second period probability to get sick for an individual who got sick in the first period is exogenous. This probability is defined by p_h , with $p_H^0 < p_h < 1$.

When there is possibility of reclassification, we consider the following timing of events (see Figure 2.1).

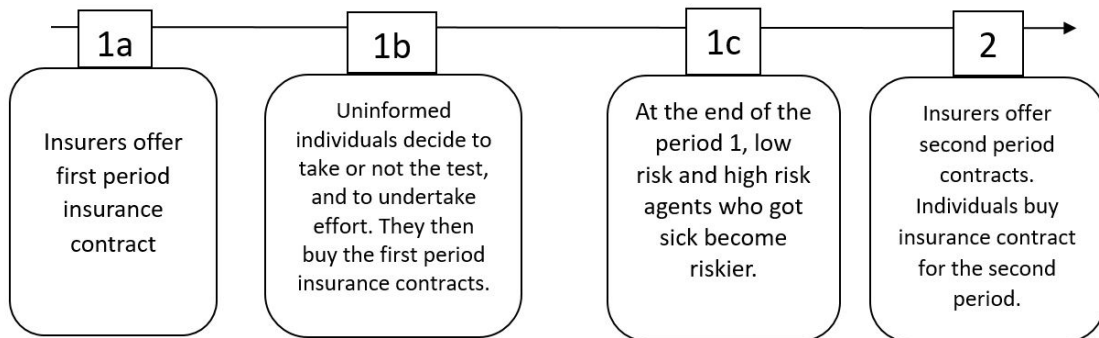


Figure 2.1: The timing-sequence of events in the model with reclassification

In the first period, competitive insurers offer insurance contracts. Individuals are uninformed, decide to take or not a genetic test, to undertake prevention action and buy insurance contract. Insurers observe the realization of the states of nature (i.e., if agents got sick or not) at the end of the first period. We consider two cases concerning the second period:

- In the model without reclassification, in the second period, insurers renew the insurance contract at same premium for all agents.

- In the model with reclassification, insurers renew the insurance contract at same premium for agents who did not get sick in the first period, while they propose a unique insurance contract with a higher premium to agents who got sick in the first period.

Figures 2.2 and 2.3 describe the different probabilities of loss when agents are informed and when they are reclassified or not. Figure 2.4 depicts the probabilities of loss when agents are uninformed and when they are reclassified. We first study as benchmark the model where individuals are not reclassified. Afterwards, we introduce reclassification in our model. The model is resolved through backward induction.

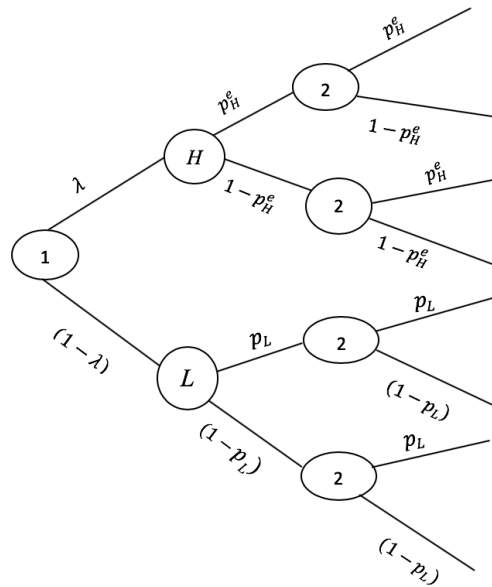


Figure 2.2: Probabilities of informed agents to get sick without reclassification

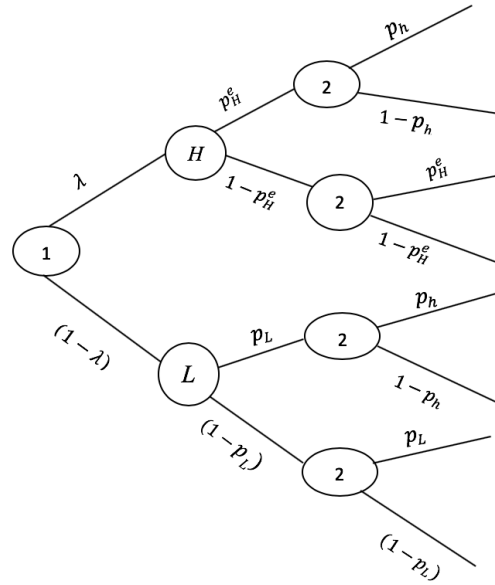


Figure 2.3: Probabilities of informed agents to get sick with reclassification

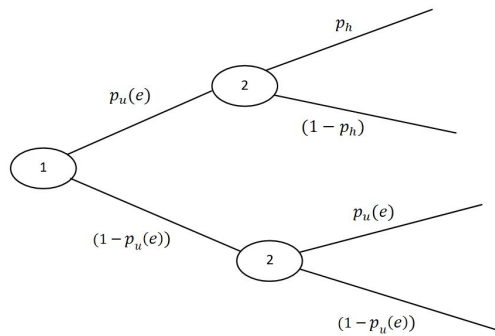


Figure 2.4: Probabilities of uninformed agents to get sick with reclassification

2.3 Dynamic model without reclassification

In this framework, insureds are not reclassified at renewal of contracts. They then have the same insurance contract in the second period independently if they are sick or not in the first period. We start by describing the contracts offered by insurers before moving to the individuals' choice of prevention action and insurance contract. Afterwards, we study the decision of individuals to perform genetic testing. We also provide two numerical examples.

2.3.1 Insurance contracts

Insurance contracts are characterized by the first period and second period premiums P_1 and P_2 , and the first period and second period indemnities I_1 and I_2 that depend on whether individuals make the test and undertake prevention effort. We determine insurance contracts from an interim perspective, that is, after the agent has taken his decision and made this information public which is the case in the disclosure duty regulation.

The optimal contract for each type derives from the maximization of the intertemporal expected utility of the agent with respect to the premium and the indemnity subject to the non negativity constraint profit of risk neutral insurance company.

For a type- H agent, the optimal contract is the solution of the following program:

$$\begin{aligned}
 \max_{P_1, P_2, I_1, I_2} U_H^e &= (1 - p_H^e)v(y - P_1) + p_H^e v(y - P_1 - d + I_1) - \phi(e) + \\
 &\beta [(1 - p_H^e)((1 - p_H^e)v(y - P_2) + p_H^e v(y - P_2 - d + I_2)) + \\
 &\quad p_H^e((1 - p_H^e)v(y - P_2) + p_H^e v(y - P_2 - d + I_2))] \\
 \text{subject to: } &P_1 \geq p_H^e I_1; P_2 \geq p_H^e I_2.
 \end{aligned} \tag{2.1}$$

The first line of this expression is the first period expected utility of a type- H agent. Lines 2 and 3 reflect the second period expected utility of the agent, weighted by the

discount rate factor β . Condition (2.1) ensures that the profit of insurers for each insurance contract is non negative.

As competition forces insurers to offer actuarial premium to insureds, we have $P_1 = p_H^e I_1$, $P_2 = p_H^e I_2$. Risk-averse agents buy full insurance at a fair premium $I_1 = d$ and $I_2 = d$. The intertemporal expected utility of a type- H agent, denoted by U_H^e , becomes

$$U_H^e = v(y - p_H^e d) - \phi(e) + \beta [v(y - p_H^e d)] = (1 + \beta)v(C_H^e) - \phi(e),$$

denoting by $C_H^e = y - p_H^e d$, the consumption level of a type- H agent according to effort, with $C_H^1 > C_H^0$.

For a type- L agent, the optimal contract is the solution of the following program

$$\begin{aligned} \max_{P_1, P_2, I_1, I_2} U_L &= (1 - p_L)v(y - P_1) + p_L v(y - P_1 - d + I_1) \\ &+ \beta [p_L((1 - p_L)v(y - P_2) + p_L v(y - P_2 - d + I_2)) \\ &+ (1 - p_L)((1 - p_L)v(y - P_2) + p_L v(y - P_2 - d + I_2))] \end{aligned}$$

$$\text{subject to : } P_1 \geq p_L I_1; P_2 \geq p_L I_2.$$

For the same reason as previously, the optimal contract provides full insurance: $I_1 = d$ and $I_2 = d$ at a fair premium $P_1 = p_L I_1$ and $P_2 = p_L I_2$.

The intertemporal expected utility of a type- L agent buying full insurance is defined by:

$$U_L = v(y - p_L d) + \beta [v(y - p_L d)] = (1 + \beta)v(C_L),$$

where C_L is the consumption level of a type- L agent, with $C_L = y - p_L d$.

For a type- U agent, the optimal contract is the solution of the following program:

$$\max_{P_1, P_2, I_1, I_2} U_U^e = (1 - p_U^e)v(y - P_1) + p_U^e v(y - P_1 - d + I_1) - \phi(e) +$$

$$\beta[(1 - p_U^e)((1 - p_U^e)v(y - P_2) + p_U^e v(y - P_2 - d + I_2)) + p_U^e((1 - p_U^e)v(y - P_2) + p_U^e v(y - P_2 - d + I_2))]$$

$$\text{subject to: } P_1 \geq p_U^e I_1; P_2 \geq p_U^e I_2$$

Again, competitive insurers propose actuarially fair premium to policyholders. We have $P_1 = p_U^e I_1$ and $P_2 = p_U^e I_2$. As a result, risk-averse agents buy insurance contract with complete coverage : $I_1 = d$ and $I_2 = d$.

The intertemporal expected utility of an uninformed individual with a contract providing complete coverage becomes:

$$U_U^e = v(y - p_U^e d) - \phi(e) + \beta [v(y - p_U^e d)] = (1 + \beta)v(C_U^e) - \phi(e),$$

where the consumption level of a type- U agent according to effort is defined by C_U^e .

We have $C_L > C_U^1 > C_H^1 > C_U^0 > C_H^0$, and $v(C_L) > v(C_U^1) > v(C_H^1) > v(C_U^0) > v(C_H^0)$ since $v'(\cdot) > 0$.

We have $C_L > C_U^1 > C_H^1$; $C_H^1 > C_H^0$ and $C_U^0 > C_H^0$, and $v(C_L) > v(C_U^1) > v(C_H^1)$; $v(C_H^1) > v(C_H^0)$ and $v(C_U^0) > v(C_H^0)$ since $v'(\cdot) > 0$.

We also have $U_L > U_U^0 > U_H^0$, and $U_L > U_H^1 > U_U^1$. Finally, we have $v'(C_H^0) > v'(C_H^1) > v'(C_U^1) > v'(C_L)$ and $v'(C_U^0) > v'(C_U^1)$ since $v''(\cdot) < 0$.

We now move to the choice of prevention decision of individuals.

2.3.2 Prevention and health insurance decisions

Type- H agents undertake effort if $U_H^1 > U_H^0$, i.e., if

$$v(C_H^1) - \phi + \beta [p_H^1 v(C_H^1) + (1 - p_H^1)v(C_H^1)] > v(C_H^0) + \beta [p_H^0 v(C_H^0) + (1 - p_H^0)v(C_H^0)]$$

$$\iff \phi < (1 + \beta) [v(C_H^1) - v(C_H^0)] \equiv (1 + \beta)\phi_{\max},$$

ϕ_{\max} and ϕ_{\min} correspond to the same threshold effort cost of BDD.

Type- U agents undertake effort if $U_U^1 > U_U^0$, i.e., if

$$v(C_U^1) - \phi + \beta [p_U^1 v(C_U^1) + (1 - p_U^1)v(C_U^1)] > v(C_U^0) + \beta [p_U^0 v(C_U^0) + (1 - p_U^0)v(C_U^0)]$$

$$\iff \phi < (1 + \beta) [v(C_U^1) - v(C_U^0)] \equiv (1 + \beta)\phi_{\min}.$$

Result 1 sums up these findings.

Result 1.

- (i) *Type- H and type- U agents undertake effort more often in the dynamic context than in the static context of BDD.*
- (ii) *In the dynamic context, type- H agents undertake effort more often than type- U agents.*

Proof.

- (i) Straightforward since $\beta > 0$, $v(C_H^1) - v(C_H^0) > 0$, and $v(C_U^1) - v(C_U^0) > 0$.
- (ii) As $\phi_{\max} > \phi_{\min}$ (see BDD) and $\beta > 0$, we have $(1 + \beta)\phi_{\max} > (1 + \beta)\phi_{\min}$.

□

Thus, adding a second period expands the threshold effort cost compatible to prevention action for both types of agents.

We now analyze the incentive of individuals to take the genetic test when they are not reclassified.

2.3.3 The private value of the test

The private value of the test is defined as the difference between the expected utility obtained by an insured when the test is performed and the expected utility when

the individual does not take the test. Superscript (e,e) reflects respectively the prevention effort of type- H and type- U agents.¹⁰

An individual decides to take a genetic testing provided that his/her expected utility when he/she is informed is higher than his/her utility when staying uninformed.

We first study the case where no individual undertakes prevention action, that is when $\phi \geq (1 + \beta)\phi_{max}$.

2.3.3.1 No one undertakes prevention action

When $\phi \geq (1 + \beta)\phi_{max}$, the private value of the test is defined as follows

$$\begin{aligned}\psi_{NR}^{0,0}(\phi, \Delta) &= \lambda U_H^0 + (1 - \lambda)U_L - U_U^0 \\ &= (1 + \beta) [\lambda v(C_H^0) + (1 - \lambda)v(C_L) - v(C_U^0)] < 0.\end{aligned}\quad (2.2)$$

Result 2. When $\phi \geq (1 + \beta)\phi_{max}$, $\psi_{NR}^{0,0}(\phi, \Delta)$ is

- (i) strictly negative for all ϕ and Δ ,
- (ii) decreasing in β and smaller in a dynamic model without reclassification than in a static model (i.e., $\beta=0$),
- (iii) increasing in p_L and decreasing in p_H^0 .

Proof.

- (i) Straightforward from Jensen's inequality.
- (ii) Idem.
- (iii) We have $\frac{\partial \psi_{NR}^{0,0}(\phi, \Delta)}{\partial p_L} = d(1 - \lambda)(1 + \beta)(v'(C_U^0) - v'(C_L)) > 0$ and $\frac{\partial \psi_{NR}^{0,0}(\phi, \Delta)}{\partial p_H^0} = -d\lambda(1 + \beta)(v'(C_H^0) - v'(C_U^0)) < 0$.

¹⁰type- L agent effort is omitted because when he/she is informed $e = 0$.

□

The expression inside the brackets of (2.2) measures the negative impact of genetic discrimination and is strictly negative according to Jensen's inequality. Therefore, as $\beta > 0$, $\psi_{NR}^{0,0}(\phi, \Delta) < 0$. Thus, the private value of information is negative in a dynamic model without reclassification when information does not create opportunity to invest in efficient prevention actions. Individuals will never undertake a genetic test when there is no effective prevention action. This result corresponds to the Hirshleifer's [1971] result according to which the private value of information is negative when information does not have decision making value. The intuition is that when agents undertake the test, they are exposed to discrimination risk. Whenever there is no effective prevention action, agents never undertake the test and obtain the sure payoff, as in a one-period model (with $\beta=0$). In a dynamic model, agents accumulate the negative effect of the first and second period of taking a genetic testing. Therefore, the private value of the test is decreasing in β and smaller in a dynamic model without reclassification than in a static model.

The loss of utility resulting from genetic discrimination is higher the higher the discrimination risk (the gap between individual probabilities of getting sick), i.e. when p_H^0 is high and p_L is low. As a result, the private value of information is decreasing with discrimination risk.

We now consider the case where type- H and type- U agents undertake prevention.

2.3.3.2 The two types (H, U) undertake prevention

When $\phi \leq (1 + \beta)\phi_{min}$, the private value of information is defined by

$$\begin{aligned} \psi_{NR}^{1,1}(\phi, \Delta) &= \lambda U_H^1 + (1 - \lambda)U_L - U_U^1 \\ &= (1 + \beta) [\lambda v(C_H^1) + (1 - \lambda)v(C_L) - v(C_U^1)] + (1 - \lambda)\phi \end{aligned} \quad (2.3)$$

The term $(1 - \lambda)\phi$ measures the prevention cost saved by informed low risk individuals, while the term inside the brackets of expression (2.3), measuring the loss of utility due to genetic discrimination, is negative. Then we obtain the following result¹¹

Result 3. *When $\phi \leq (1 + \beta)\phi_{min}$, the sign of the private value of the test is undetermined. It is positive if the prevention cost saved by informed low risk individuals is higher than the loss of utility due to genetic discrimination. Moreover,*

- (i) $\psi_{NR}^{1,1}(\phi, \Delta)$ is increasing in ϕ ;
- (ii) there exists a unique value of Δ , denoted by $\tilde{\Delta}$, such that $0 < \tilde{\Delta} < \bar{\Delta}$ and $\psi^{1,1}((1 + \beta)\phi_{min}(\tilde{\Delta}), \tilde{\Delta}) = 0$;
- (iii) for all $\Delta > \tilde{\Delta}$, there exists a unique value of ϕ , denoted by $\tilde{\phi}_{1NR}(\Delta)$, such that $\psi_{NR}^{1,1}(\tilde{\phi}_{1NR}(\Delta), \Delta) = 0$ and $0 < \tilde{\phi}_{1NR}(\Delta) < (1 + \beta)\phi_{min}$;
- (iv) $\psi_{NR}^{1,1}(\phi, \Delta)$ is decreasing in β ;
- (v) the private value of the test is lower (resp., higher) in a dynamic model than a static model for all $\phi < \phi_{S=D}^1(\Delta)$ (resp., $\phi_{S=D}^1(\Delta) < \phi < (1 + \beta)\phi_{min}(\Delta)$);
- (vi) $\psi_{NR}^{1,1}(\phi, \Delta)$ is positive for all $\Delta > \tilde{\Delta}$ and $\tilde{\phi}_{1NR}(\Delta) < \phi \leq (1 + \beta)\phi_{min}$;
- (vii) $\psi_{NR}^{1,1}(\phi, \Delta)$ is increasing in p_L and decreasing in p_H .

The private value of the test increases with the cost of prevention effort, ϕ , because the test allows informed type- L agents to avoid the prevention cost, that is ineffective for them. The private value of the test is positive if prevention cost saved by informed type- L individuals is higher than the utility loss resulting from genetic

¹¹See the appendix 1 for a proof.

discrimination weighted by the factor $(1 + \beta)$. Thus, individuals are more likely to undertake the test if they value lowly their second period utility.

The private value of the test is more likely positive for low values of p_H^1 , for values of p_L close to p_H^1 and when individuals are not too risk-averse. As a result, agents are likely to take the test when the discrimination risk is low, i.e., when p_L is close to p_H^1 .

We can formally identify the value of ϕ , denoted by $\tilde{\phi}_{1NR}(\Delta)$, above which the private value of the test is positive in the dynamic model when individuals are not reclassified. To do so, let us set $\psi_{NR}^{1,1}(\phi, \Delta) = 0$, to obtain

$$\tilde{\phi}_{1NR}(\Delta) = \frac{(1 + \beta)[v(C_U^1) - (\lambda v(C_H^1) + (1 - \lambda)v(C_L))]}{1 - \lambda}.$$

$\tilde{\phi}_{1NR}(\Delta)$ is increasing in β since $v(C_U^1) - (\lambda v(C_H^1) + (1 - \lambda)v(C_L)) > 0$ from Jensen's inequality. Therefore, the value of ϕ above which the private value of the test is positive is higher in a dynamic model when individuals are not reclassified than a static model.

In order to focus on the impact of reclassification in our model, in the following of the paper, we assume that $\Delta > \tilde{\Delta}$. We now consider the case where only informed type- H individuals undertake prevention action.

2.3.3.3 Only type- H agents undertake effort

When $(1 + \beta)\phi_{min} < \phi < (1 + \beta)\phi_{max}$, the private value of the test is defined as follows

$$\begin{aligned} \psi_{NR}^{1,0}(\phi, \Delta) &= \lambda U_H^1 + (1 - \lambda)U_L - U_U^0 \\ &= (1 + \beta) [\lambda v(C_H^1) + (1 - \lambda)v(C_L) - v(C_U^0)] - \lambda\phi. \end{aligned} \quad (2.4)$$

The sign of the private value of the test is undetermined. It depends on the value of ϕ . Indeed, the terms inside the brackets of equation (2.4), measuring the

difference between the utility of prevention action for informed high risk agent and the negative impact of genetic discrimination, is strictly positive since $\Delta > \tilde{\Delta}$. The term $\lambda\phi$ measures the prevention cost of informed high risk individuals. We have the following Result¹²

Result 4. *When $(1 + \beta)\phi_{min} < \phi < (1 + \beta)\phi_{max}$, the private value of test can be positive or negative according to the value of ϕ . It is positive if the utility of prevention action is higher than the loss of utility generated by genetic discrimination, and when the prevention cost is small. Moreover,*

- (i) $\psi_{NR}^{1,0}(\phi, \Delta)$ is decreasing in ϕ ;
- (ii) for all $\Delta > \tilde{\Delta}$, there exists a unique value of ϕ , denoted by $\tilde{\phi}_{2NR}(\Delta)$, such that $\psi_{NR}^{1,0}(\tilde{\phi}_{2NR}(\Delta), \Delta) = 0$ and $(1 + \beta)\phi_{min}(\Delta) < \tilde{\phi}_{2NR}(\Delta) < (1 + \beta)\phi_{max}(\Delta)$.
- (iii) $\psi_{NR}^{1,0}(\phi, \Delta)$ is increasing in β ;
- (iv) the private value of the test is higher (resp., lower) in a dynamic model without reclassification than a static model for all $(1 + \beta)\phi_{min}(\Delta) < \phi < \phi_{S=D}^2(\Delta)$ (resp., $\phi > \phi_{S=D}^2(\Delta)$);
- (v) $\psi_{NR}^{1,0}(\phi, \Delta)$ is positive for all $\Delta > \tilde{\Delta}$ and $(1 + \beta)\phi_{min}(\Delta) < \phi < \tilde{\phi}_{2NR}(\Delta)$.
- (vi) $\psi_{NR}^{1,0}(\phi, \Delta)$ is increasing in p_L and p_H^0 , and decreasing in p_H^1 .

The private value of the test is more likely positive when prevention action is highly effective (see Result 3) and that is not too costly, for high values of β , and when the loss of utility resulting from genetic discrimination is low (see item *vi*) of Result 4). When the technology of prevention is sufficiently efficient to offset the negative impact of genetic discrimination and when agents value their second period,

¹²See the appendix 2 for a proof.

individuals may take the test. When the private value of the test is positive, it is strictly higher in the dynamic model without reclassification than a static model since it is increasing in β . We formally identify the prevention cost $\tilde{\phi}_{2NR}(\Delta)$ below which the private value of the test is positive. We note that individuals take the test for higher values of effort cost in the dynamic model without reclassification than a static model.

In summary, when agents are not reclassified and prevention effort is observable, agents take the test at equilibrium provided that the prevention efficiency is large enough, while its cost takes intermediate values and for high value of β when the effort cost is such that only type- H agents undertake prevention action.

2.3.3.4 Numerical examples

We provide two numerical examples illustrating the private value of the test according to ϕ when $(1 + \beta)\phi_{min}(\Delta) > \phi_{max}(\Delta)$ and when $(1 + \beta)\phi_{min}(\Delta) < \phi_{max}(\Delta)$. We can state the condition on β ensuring that $(1 + \beta)\phi_{min}(\Delta) > \phi_{max}(\Delta)$.

$$(1 + \beta)\phi_{min}(\Delta) > \phi_{max}(\Delta) \text{ provided that } \beta > \frac{\phi_{max}(\Delta) - \phi_{min}(\Delta)}{\phi_{min}(\Delta)}.$$

As a first numerical example, let us use the relevant parameters¹³ from the BRCA genes for a woman if her mother or her sister had ovarian cancer before the age of 50, i.e., $\lambda = 0.065$, $p_H^0 = 0.295$, $p_L = 0.0111$. We also consider that $\alpha = 0.5$, $p_H^1 = 0.2$, $y = 2500$, $d = 400$, and $\beta = 0.8$. Given these parameters, a comparison between static and dynamic contexts is depicted by Figure 2.5. In this Figure $(1 + \beta)\phi_{min}(\Delta) < \phi_{max}(\Delta)$, when $\beta = 0.8$, we have $(1 + \beta)\phi_{min} = 0.045$, $(1 + \beta)\phi_{max} = 0.7$, $\tilde{\phi}_{1NR} = 0.00068$, and $\tilde{\phi}_{2NR} = 0.68$. When $\beta = 0$, we have $\phi_{min} = 0.025$, $\phi_{max} = 0.39$, $\tilde{\phi}_{1S} = 0.00038$, and $\tilde{\phi}_{2S} = 0.38$.

¹³See Hoy et al. (2014) and Hoy and Witt (2007).

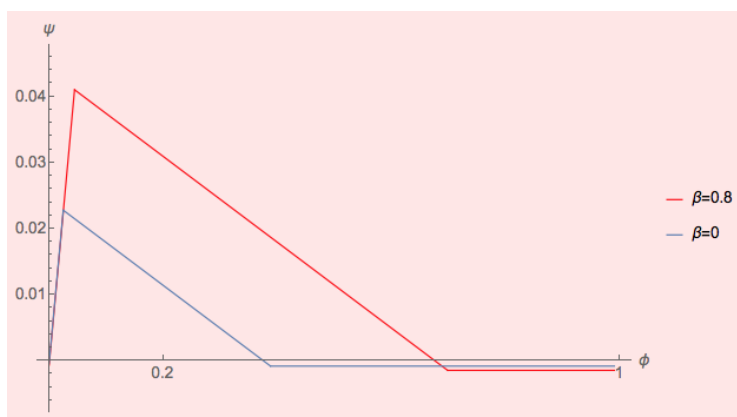


Figure 2.5: Comparison between static and dynamic contexts with BRCA

A zoom on Figure 2.5, in order to consider the low values of ϕ , is depicted by Figure 2.6. This Figure shows that the red curve is well below the blue curve for all $\phi < \phi_{S=D}^1(\Delta)$.

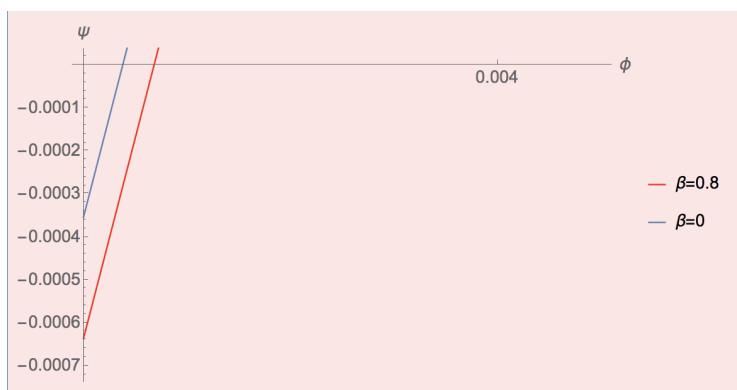


Figure 2.6: Zoom of Figure 2.5 for low values of ϕ

As a second numerical example (see Figure 2.7) where $(1 + \beta)\phi_{\min} > \phi_{\max}$, let us now consider $\lambda = 0.95$, $p_H^0 = 0.9$, $p_H^1 = 0.8$, $p_L = 0.001$, still with $y = 2500$, $\alpha = 0.5$, $d = 400$, and $\beta = 0.8$. In this case, we have $(1 + \beta)\phi_{\min} = 0.73$, $(1 + \beta)\phi_{\max} = 0.77$, $\tilde{\phi}_{1NR} = 0.2$, and $\tilde{\phi}_{2NR} = 0.76$. When $\beta = 0$, we have $\phi_{\min} = 0.42$, $\phi_{\max} = 0.43$, $\tilde{\phi}_{1S} = 0.11$, and $\tilde{\phi}_{2S} = 0.42$.

We now move to the dynamic model where individuals who got sick in the first

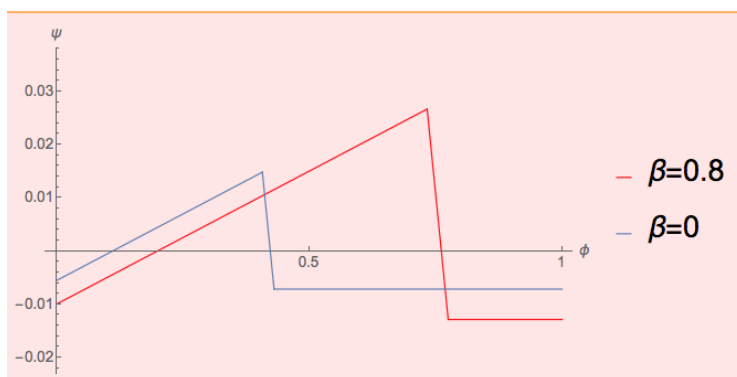


Figure 2.7: The private value of the test according to ϕ in a static versus a dynamic context

period are reclassified in the second.

2.4 Dynamic model with reclassification

We now introduce the possibility of reclassification in our model. We first determine the insurance contracts of agents and show how reclassification impacts prevention decision of agents. We then analyze the impact of reclassification on testing decision and provide some numerical examples.

2.4.1 Insurance contracts

As previously, insurance contracts are determined from an interim perspective. The optimal contract for each agent's type in each period derives from the maximization of his intertemporal expected utility with respect to the premium and the indemnity, subject to the non negativity constraint profit of a risk-neutral insurance company on a competitive market.

For a type- H agent, the optimal contract is the solution of

$$\max_{P_1, P_2, P_{2h}, I_1, I_2, I_{2h}} (1 - p_H^e)v(y - P_1) + p_H^e v(y - P_1 - d + I_1) - \phi(e) + \beta [(1 - p_H^e)((1 - p_H^e)v(y - P_2) + p_H^e v(y - P_2 - d + I_2) +$$

$$p_H^e((1 - p_h)v(y - P_{2h}) + p_hv(y - P_{2h} - d + I_{2h}))$$

$$\text{subject to: } P_1 \geq p_H^e I_1; P_2 \geq p_H^e I_2; P_{2h} \geq p_h I_{2h} \quad (2.5)$$

The first line of this expression is the first period expected utility of a type- H agent. The second and third line correspond to the sum of the second period expected utility of a type- H agent who did not get sick in the first period and of a type- H agent who got sick in the first period, weighted by the discount rate factor β . Conditions (2.5) ensure non negative profit for insurers in each insurance contract.

Competition forces insurers to offer actuarial premium to insureds. So, we have $P_1 = p_H^e I_1$, $P_2 = p_H^e I_2$ and $P_{2h} = p_h I_{2h}$. Risk-averse agents buy full insurance at a fair premium $I_1 = d$, $I_2 = d$ and $I_{2h} = d$. The intertemporal expected utility of a type- H agent denoted by, U_H^e , becomes

$$U_H^e = v(y - p_H^e d) - \phi(e) + \beta [p_H^e v(y - p_h d) + (1 - p_H^e)v(y - p_H^e d)].$$

This can be written as

$$U_H^e = v(C_H^e) - \phi(e) + \beta [p_H^e v(C_h) + (1 - p_H^e)v(C_H^e)],$$

where C_h is the second period consumption level of an agent who got sick in the first period, with $C_h < C_H^0$.

For a type- L agent, the optimal contract is the solution of

$$\max_{P_1, P_2, P_{2h}, I_1, I_2, I_{2h}} U_L = (1 - p_L)v(y - P_1) + p_L v(y - P_1 - d + I_1)$$

$$+ \beta [p_L((1 - p_h)v(y - P_{2h}) + p_hv(y - P_{2h} - d + I_{2h}))$$

$$+ (1 - p_L)((1 - p_L)v(y - P_2) + p_L v(y - P_2 - d + I_2))]$$

$$\text{subject to: } P_1 \geq p_L I_1; P_2 \geq p_L I_2; P_{2h} \geq p_h I_{2h}. \quad (2.6)$$

For the same reason as previously, risk-averse agents buy full insurance $I_1 = d$, $I_2 = d$ and $I_{2h} = d$ at a fair premium, with $P_1 = p_L I_1$, $P_2 = p_L I_2$ and $P_{2h} = p_L I_{2h}$. The intertemporal expected utility of a type- L agent buying full insurance is defined by

$$\begin{aligned} U_L &= v(y - p_L d) + \beta [p_L v(y - p_h d) + (1 - p_L) v(y - p_L d)] \\ &= v(C_L) + \beta [p_L v(C_h) + (1 - p_L) v(C_L)]. \end{aligned}$$

For a type- U agent, the optimal contract is the solution of

$$\max_{P_1, P_2, P_{2h}, I_1, I_2, I_{2h}} U_U^e = (1 - p_U^e) v(y - P_1) + p_U^e v(y - P_1 - d + I_1) - \phi(e) +$$

$$\begin{aligned} &\beta [(1 - p_U^e)((1 - p_U^e) v(y - P_2) + p_U^e v(y - P_2 - d + I_2) + \\ &p_U^e ((1 - p_h) v(y - P_{2h}) + p_h v(y - P_{2h} - d + I_{2h})))] \end{aligned}$$

$$\text{subject to: } P_1 \geq p_U^e I_1; P_2 \geq p_U^e I_2; P_{2h} \geq p_h I_{2h}. \quad (2.7)$$

As previously, competitive insurers propose actuarial premium to insureds. We have $P_1 = p_U^e I_1$, $P_2 = p_U^e I_2$ and $P_{2h} = p_h I_{2h}$. Risk-averse agents buy full insurance $I_1 = d$, $I_2 = d$ and $I_{2h} = d$ at a fair premium. The intertemporal expected utility of a type- U individual with a complete insurance contract becomes

$$\begin{aligned} U_U^e &= v(y - p_U^e d) - \phi(e) + \beta [p_U^e v(y - p_h d) + (1 - p_U^e) v(y - p_U^e)] \\ &= v(C_U^e) - \phi(e) + \beta [p_U^e v(C_h) + (1 - p_U^e) v(C_U^e)], \end{aligned}$$

with $C_h < C_H^0$.

Given our assumptions, We have $U_L > U_U^0 > U_H^0$ and $U_L > U_U^1 > U_H^1$. We also have $v'(C_h) > v'(C_H^0)$ since $v''(\cdot) < 0$.

We now focus on the choice of prevention decision of individuals.

2.4.2 Prevention and health insurance decisions

Type- H agents undertake effort if $U_H^1 > U_H^0$, i.e., if

$$v(C_H^1) - \phi + \beta [p_H^1 v(C_h) + (1 - p_H^1)v(C_H^1)] > v(C_H^0) + \beta [p_H^0 v(C_h) + (1 - p_H^0)v(C_H^0)]$$

$$\iff \phi < \phi_H = (1 + \beta)\phi_{\max} + \beta x_H,$$

with $x_H = (p_H^1 - p_H^0)v(C_h) - p_H^1 v(C_H^1) + p_H^0 v(C_H^0)$. The term βx_H captures the impact of reclassification on the choice of prevention effort of type- H agents.

Type- U agents undertake effort if $U_U^1 > U_U^0$, i.e., if

$$v(C_U^1) - \phi + \beta [p_U^1 v(C_h) + (1 - p_U^1)v(C_U^1)] > v(C_U^0) + \beta [p_U^0 v(C_h) + (1 - p_U^0)v(C_U^0)]$$

$$\iff \phi < \phi_U = (1 + \beta)\phi_{\min} + \beta x_U,$$

with $x_U = (p_U^1 - p_U^0)v(C_h) - p_U^1 v(C_U^1) + p_U^0 v(C_U^0)$. The term βx_U captures the impact of reclassification on the choice of prevention effort of type- U agents.

In comparison with the situation without reclassification, we obtain more cases. So, it is useful to interpret the terms x_H and x_U in order to understand the impact of reclassification on prevention decision. x_H corresponds to the difference between the second period expected utility of the prevention effort when one is reclassified and when not for informed type- H individuals who got sick in the first period. In the same way, x_U measures the difference between the second period expected utility of the prevention effort when one is reclassified and the second period expected utility of the prevention effort when one is not reclassified for agents who did not take the test and got sick in the first period.

Thus, if x_H and x_U are positive, then the expected utility of prevention effort with reclassification is higher than without reclassification. As a result, individuals undertake prevention effort for larger prevention costs in the model with reclassification, i.e. $\phi_H > (1 + \beta)\phi_{\max}$ and $\phi_U > (1 + \beta)\phi_{\min}$. We notice that x_H and x_U

are more likely positive when the intensity of reclassification is high. However, if x_H and x_U are negative, then the expected utility of the effort in the model without reclassification is higher than with reclassification. In this case, we conclude that reclassification reduces the cost threshold compatible with effort, i.e., $\phi_H < (1 + \beta)\phi_{\max}$ and $\phi_U < (1 + \beta)\phi_{\min}$.

From the previous definition, $\phi_H - \phi_U = (1 + \beta)(\phi_{\max} - \phi_{\min}) + \beta(x_H - x_U)$, with $\phi_{\max} > \phi_{\min}$. Without reclassification, $\phi_H - \phi_U = (1 + \beta)(\phi_{\max} - \phi_{\min}) > 0$. Let us now analyze the comparison of ϕ_H and ϕ_U when reclassification occurs. Three cases can be considered

- (i) If $x_U - x_H < 0$, then reclassification increases the difference in prevention between type- H and type- U agents (compared with the case without reclassification) and $\phi_H > \phi_U$.
- (ii) If $0 < (x_U - x_H) < \frac{(1+\beta)}{\beta}(\phi_{\max} - \phi_{\min})$, then reclassification decreases the difference in prevention between H and U but we still have $\phi_H > \phi_U$.
- (iii) If $(x_U - x_H) > \frac{(1+\beta)}{\beta}(\phi_{\max} - \phi_{\min})$, then reclassification decreases the difference in prevention between H and U and we have $\phi_H < \phi_U$.

We obtain the following result concerning the prevention decision.

Result 5. *The prevention decision of agents depends on the effort cost as follows:*

- a) *when ϕ is high ($\phi \geq \max(\phi_H, \phi_U)$), no agent undertakes effort;*
- b) *when ϕ is low ($\phi \leq \min(\phi_H, \phi_U)$), the two types (H, U) undertake effort;*
- c) *when $\phi_H > \phi_U$ and for intermediate values of ϕ (i.e., for $\phi_U < \phi < \phi_H$), only informed high risk agents undertake prevention actions;*

d) when $\phi_H < \phi_U$ and for intermediate values of ϕ (i.e., for $\phi_H < \phi < \phi_U$), only uninformed individuals undertake effort.

The likelihood of these different cases depends on the value of the parameters whose impact is analyzed in the following subsections.

2.4.2.1 Impact of β on prevention

Let us now analyze the impact of β on the prevention effort. Adding and subtracting $\beta(1 - p_H^1)v(C_H^0)$ to ϕ_H , and rearranging the expression, we obtain

$$\phi_H = \phi_{max} + \beta[(p_H^0 - p_H^1)(v(C_H^0) - v(C_h)) + (1 - p_H^1)(v(C_H^1) - v(C_H^0))],$$

with $(p_H^0 - p_H^1)(v(C_H^0) - v(C_h)) + (1 - p_H^1)(v(C_H^1) - v(C_H^0)) > 0$.

In order to study how β affects ϕ_U , we add and subtract $\beta(1 - p_U^1)v(C_U^0)$ to ϕ_U .

After rearranging the expression, we obtain

$$\phi_U = \phi_{min} + \beta[\lambda(p_H^0 - p_H^1)(v(C_U^0) - v(C_h)) + (1 - p_U^1)(v(C_U^1) - v(C_U^0))],$$

with $\lambda(p_H^0 - p_H^1)(v(C_U^0) - v(C_h)) + (1 - p_U^1)(v(C_U^1) - v(C_U^0)) > 0$.

Then, the following result can be highlighted:

Result 6.

- (i) ϕ_H (resp., ϕ_U) can be higher or lower than $(1 + \beta)\phi_{max}$ (resp., $(1 + \beta)\phi_{min}$);
- (ii) ϕ_H and ϕ_U are increasing in β ;
- (iii) type-H and type-U agents undertake effort more often in the dynamic model with reclassification than in a static model.

Proof of Result 6:

- (i) Straightforward since x_H and x_U can be positive or negative.

- (ii) Straightforward since the coefficient of β in ϕ_H and ϕ_U is positive.
- (iii) Straightforward given *ii*) and since $\beta > 0$.

□

We notice that if effort has a higher impact on the utility of agents with reclassification than without reclassification, then the more individuals value the second period, the more they will be ready to undertake effort for high levels of the cost of this effort.

2.4.2.2 Impact of p_h on prevention

The lowest possible level of reclassification is $p_h = p_H^0$. This level of reclassification affects neither a Type- L agent, whose effort is supposed to be ineffective, nor a Type- H agent who does not make effort. It however affects both a Type- H agent who makes an effort and a Type- U agent, whatever its effort. Then, we have the following result¹⁴

Result 7.

- (i) x_H and x_U are increasing in p_h and the increase of x_H is higher than x_U in p_h ;
- (ii) x_U is higher than x_H when $p_h = p_H^0$;
- (iii) If x_U is higher than x_H when $p_h = 1$, then x_U is higher than $x_H \forall p_h \in [p_H^0, 1]$;
- (iv) If x_U is lower than x_H when $p_h = 1$, then there exists a unique value p_h^* such that x_U is higher than x_H for $p_h \in [p_H^0, p_h^*]$ and x_U is lower than x_H for $p_h \in [p_h^*, 1]$;

¹⁴See the appendix 3 for a proof.

(v) $x_U - x_H$ decreases in p_h at an increasing rate and the decrease of $x_U - x_H$ in p_h is lower for higher value of λ .

The direction of change of x_U and x_H with respect to p_h deserves some comments. We know that the second period expected utility of the prevention effort when individuals are not reclassified is independent of p_h , while the second period expected utility of prevention effort when one is reclassified is increasing in p_h . Therefore, x_U and x_H are necessarily increasing in p_h since the second period expected utility of the prevention effort when individuals are reclassified is larger for higher values of p_h .

2.4.2.3 Impact of p_L on prevention

Let us analyze the impact of p_L on x_H and x_U . The level of p_L affects the expected utility of prevention effort of only type- U agents. Then, we have the following result¹⁵

Result 8.

- (i) x_H is independent of p_L and x_U is decreasing in p_L . So, $x_U - x_H$ is decreasing in p_L ;
- (ii) $x_U - x_H$ is positive when p_L tends to zero;
- (iii) If $x_U - x_H$ is positive when $p_L = p_H^1$, then x_U is higher than $x_H \forall p_L \in [0, p_H^1]$.
- (iv) If $x_U - x_H$ is negative when $p_L = p_H^1$, then there exists a unique value p_L^* such that x_U is higher than x_H for $p_L \in [0, p_L^*]$ and x_U is lower than x_H for $p_L \in [p_L^*, p_H^1]$.

The term x_U is decreasing in p_L since p_L impacts negatively the second period expected utility of prevention effort when uninformed individuals are not reclassified, while the expected utility of prevention effort when uninformed individuals are

¹⁵See the appendix 4 for a proof.

reclassified is independent of p_L . p_L has no impact on x_H because the expected utility of prevention effort of informed high risk individuals is independent of p_L . Therefore, the term $x_U - x_H$ is more likely positive when p_L tends to zero.¹⁶

From Result 7 and Result 8, the case $x_U - x_H < 0$ and $\phi_H > \phi_U$ is more likely for high values of p_h and/or p_L , while the case $(x_U - x_H) > \frac{(1+\beta)}{\beta}(\phi_{\max} - \phi_{\min})$ and $\phi_H > \phi_U$ is more likely for relatively low values of β . Moreover, the case $(x_U - x_H) > \frac{(1+\beta)}{\beta}(\phi_{\max} - \phi_{\min})$ implying that $\phi_H < \phi_U$ is more likely for relatively high values of β . This case is also more likely when p_h is low. Indeed, we know that ϕ_{\max} and ϕ_{\min} do not depend on p_h and $\frac{\partial x_U - x_H}{\partial p_h} < 0$.

2.4.2.4 Effect of Δ on prevention decision

Let us consider that $p_H^1 = p_H^0 - \Delta$. Recall that $\phi_H = (1 + \beta)\phi_{\max} + \beta x_H$ and $\phi_U = (1 + \beta)\phi_{\min} + \beta x_U$.

We can compute $\frac{d\phi_{\max}}{d\Delta} = dv'(C_H^1)$ and $\frac{d\phi_{\min}}{d\Delta} = d\lambda v'(C_U^1)$. Thus, $\frac{d\phi_{\max}}{d\Delta} > \frac{d\phi_{\min}}{d\Delta} > 0$.

Then

$$\begin{aligned} \frac{d\phi_H}{d\Delta} &= (1 + \beta) \frac{d\phi_{\max}}{d\Delta} + \beta \frac{dx_H}{d\Delta} \\ &= dv'(C_H^1) [1 + \beta(1 - p_H^1)] + \beta [v(C_H^1) - v(C_h)] > 0 \end{aligned}$$

and

$$\begin{aligned} \frac{d\phi_U}{d\Delta} &= (1 + \beta) \frac{d\phi_{\min}}{d\Delta} + \beta \frac{dx_U}{d\Delta} \\ &= \lambda \left[dv'(C_U^1) [1 + \beta(1 - p_U^1)] + \beta [v(C_U^1) - v(C_h)] \right] > 0 \end{aligned}$$

So, ϕ_H and ϕ_U are increasing in Δ . When $\Delta = 0$, we have $p_H^1 = p_H^0$, $p_U^1 = p_U^0$, and thus $\phi_H = \phi_U$. Then, when $\Delta > 0$, we have $\phi_H > (<) \phi_U$ if $\frac{d\phi_H}{d\Delta} > (<) \frac{d\phi_U}{d\Delta}$. Thus, the more the efficiency of the effort, the more individual undertakes prevention for

¹⁶The impact of p_L on $\phi_U - \phi_H$ is undetermined.

higher effort costs.

2.4.2.5 Comparison of the thresholds of the cost of effort

When comparing the thresholds of the cost of effort of the static model, the dynamic with and without reclassification, we identify 25 possible cases given the conditions on the thresholds:

$$\phi_{min} < \phi_{max} ; \phi_{min} < (1 + \beta)\phi_{min}; \phi_{min} < \phi_U; (1 + \beta)\phi_{min} < (1 + \beta)\phi_{max}; \phi_{max} < (1 + \beta)\phi_{max}; \text{ and } \phi_{max} < \phi_H.$$

We have 3 possible cases when comparing the thresholds to undertake prevention in the dynamic model with reclassification and in a static model.

1. $\phi_{min} < \phi_{max} < \phi_U < \phi_H$;
2. $\phi_{min} < \phi_{max} < \phi_H < \phi_U$;
3. $\phi_{min} < \phi_U < \phi_{max} < \phi_H$.

We also identify 12 possible cases when comparing the thresholds to undertake prevention in the dynamic model without reclassification and with reclassification. We do not consider all the possible cases because it does not bring any new insights to the analysis.

In the next section, we analyze the decision of individuals to take the test.

2.4.3 The private value of the test

In this two-period model with reclassification, we denote the value of the test by $\psi^{e,e}$. We analyze the different cases of prevention action described in Result 5, considering firstly the case where no one undertakes effort, i.e., when $\phi \geq \max(\phi_H, \phi_U)$.

2.4.3.1 No prevention action

When $\phi \geq \max(\phi_H, \phi_U)$, the private value of information is defined by

$$\begin{aligned}\psi^{0,0} &= \lambda U_H^0 + (1 - \lambda)U_L - U_U^0 \\ &= \lambda v(C_H^0) + (1 - \lambda)v(C_L) - v(C_U^0) + \\ &\quad \beta[(\lambda p_H^0 + (1 - \lambda)p_L)v(C_h) + \lambda(1 - p_H^0)v(C_H^0) + \\ &\quad (1 - \lambda)(1 - p_L)v(C_L) - p_U^0 v(C_h) - (1 - p_U^0)v(C_U^0)].\end{aligned}$$

After replacing p_U^0 of the second term in the fourth line and simplifying the expression, we obtain

$$\psi^{0,0} = (1 + \beta)[\lambda v(C_H^0) + (1 - \lambda)v(C_L) - v(C_U^0)] + \beta\alpha_0, \quad (2.8)$$

with $\alpha_0 = p_U^0 v(C_U^0) - \lambda p_H^0 v(C_H^0) - (1 - \lambda)p_L v(C_L)$.

The term $\beta\alpha_0$ measures the impact of reclassification on the private value of the test. The (negative) term inside the brackets of equation (2.8) measures the impact of genetic discrimination. Then, we have the following proposition:¹⁷

Proposition 1. *When $\phi \geq \max(\phi_H, \phi_U)$, then*

- (i) *the private value of the test is independent of ϕ and p_h ,*
- (ii) *reclassification impacts positively the private value of the test. The private value of the test with reclassification is higher than without reclassification when agents do not undertake effort both in the model with and without reclassification,*
- (iii) *when the cost of effort is such that types H and U or type-H agents undertake prevention effort in the model without reclassification, the private value of the test with reclassification can be higher or lower than without reclassification,*

¹⁷See the appendix 5 for a proof.

- (iv) *the private value of the test can be increasing or decreasing in β according to the parameters value,*
- (v) *the private value of the test can be positive or negative according to the parameters value,*
- (vi) *the impact of reclassification on the private value of the test is decreasing in p_L and increasing in p_H^0 .*

The intuition of the positive impact of reclassification on the private value of the test is as follows. When individuals are not reclassified, the second period expected utility of uninformed individuals who got sick in the first period weighted by their probability to get sick, $p_U^0 v(C_U^0)$, is higher than the second period expected utility of informed individuals who got sick in the first period weighted by their probability to obtain a test result and to get sick, $\lambda p_H^0 v(C_H^0) + (1 - \lambda) p_L v(C_L)$. However, with reclassification, the second period expected utility of agents when they are sick in the first period does not change whether they are informed or not, since they obtain $v(C_h)$ in the second period. As a result, the private value of the test is higher with reclassification than without reclassification when agents do not undertake prevention effort both in the two models. This positive impact of reclassification on the private value of the test is higher for high values of p_H^0 and low values of p_L . In other words, this positive impact is higher the higher the discrimination risk, i.e., when $p_H^0 - p_L$ is high.

Furthermore, when agents undertake prevention effort in the model without reclassification, the private value of the test with reclassification can be higher or lower than without reclassification. Indeed, the private value of the test with reclassification is higher than without reclassification if the effect of reclassification dominates the effect of prevention effort. Finally, unlike a dynamic model without reclassifica-

tion, the private value of the test can be increasing in β and positive in a dynamic model with reclassification when individuals do not undertake prevention action. It is an interesting result that the private value of the test can be positive. The intuition is that when agents do not undertake effort, the first period sure payoff is strictly higher than the first period lottery for risk-averse individuals. When agents got sick in the first period, they will have the same insurance contract in the second period regardless of whether they perform genetic testing or not. Informed low risk and high risk agents will keep their first period insurance contract respectively with the probability $(1 - \lambda)(1 - p_L)$ and $\lambda(1 - p_H^0)$, while uninformed individuals will keep the same insurance contract with the probability $(1 - p_U^0)$, with $(1 - p_U^0) < 1$ and $(1 - p_U^0) = (1 - \lambda)(1 - p_L) + \lambda(1 - p_H^0)$. Thus, individuals compare their second period expected utility when they would not get sick in the first period, making a trade off between the loss of utility of the second period resulting from genetic discrimination and the positive effect of reclassification α_0 . So, the second period expected utility of informed agents who did not get sick in the first period is higher than the second period expected payoff of uninformed agents who did not get sick in the previous period if the positive effect of reclassification dominates the second period Hirschleifer effect. Therefore, individuals may undertake the test if the second period expected utility of the test is higher than its costs and if they value sufficiently their second period relatively to the first period. This is more likely to happen if agents are not too risk-averse (See proof in appendix 6).

We now move to the case where informed high risk individuals and uninformed agents undertake prevention actions, i.e., when $\phi \leq \min(\phi_H, \phi_U)$.

2.4.3.2 Types H and U agents undertake effort

When $\phi \leq \min(\phi_H, \phi_U)$, the private value of the test is given by

$$\begin{aligned}\psi^{1,1} = & \lambda(v(C_H^1) - \phi) + (1 - \lambda)v(C_L) - (v(C_U^1) - \phi) + \\ & \beta[(\lambda p_H^1 + (1 - \lambda)p_L)v(C_h) + \lambda(1 - p_H^1)v(C_H^1) + \\ & (1 - \lambda)(1 - p_L)v(C_L) - p_U^1v(C_h) - (1 - p_U^1)v(C_U^1)].\end{aligned}$$

Proceeding as previously to simplify the expression, we obtain:

$$\psi^{1,1} = (1 + \beta)[\lambda v(C_H^1) + (1 - \lambda)v(C_L) - v(C_U^1)] + \beta\alpha_1 + (1 - \lambda)\phi,$$

with $\alpha_1 = p_U^1v(C_U^1) - \lambda p_H^1v(C_H^1) - (1 - \lambda)p_Lv(C_L)$.

The term $\beta\alpha_1$ captures the impact of reclassification on the private value of the test when types H and U agents undertake prevention effort. The term inside the bracket measures the negative impact of genetic discrimination. The last term measures the utility of the test, that is the prevention cost saved by informed low risk agents for which prevention is ineffective.

We then have the following proposition ¹⁸:

Proposition 2. *When $\phi \leq \min(\phi_H, \phi_U)$,*

- (i) the private value of test is increasing in ϕ and independent of p_h ,*
- (ii) reclassification impacts positively the private value of the test,*
- (iii) the private value of the test with reclassification can be higher or lower than without reclassification according to the ordering of the cost of effort thresholds,*
- (iv) the private value of the test can be increasing or decreasing in β according to the parameters value,*

¹⁸See proof in appendix 7.

- (v) *the private value of the test can be positive or negative according to the parameters value,*
- (vi) *the impact of reclassification on the private value of the test is decreasing in p_L and increasing in p_H^1 ,*
- (vii) *when $\psi^{1,1}(\min(\phi_H, \phi_U)) > 0$ and $\psi^{1,1}(\phi = 0) < 0$, there exists a unique value ϕ , denoted by $\tilde{\phi}_{1R}$, such that $\psi^{1,1}(\tilde{\phi}_{1R}) = 0$ and $0 < \tilde{\phi}_{1R} < \min(\phi_H, \phi_U)$, with $\tilde{\phi}_{1R} < \tilde{\phi}_{1NR}$.*

Thus, when the cost of effort is such that the two types undertake effort, the private value of the test is more likely positive if the cost of effort is relatively high (but lower than $\min(\phi_H, \phi_U)$), the loss of utility resulting from genetic discrimination is low and the positive effect of reclassification is high. As precedently and for the same arguments, the second period expected utility of informed agents who did not get sick in the first period is higher than their second period expected utility of uninformed agents that are not sick in the first period provided that the positive effect of reclassification dominates the second period negative effect of genetic discrimination, i.e., the Hirschleifer effect. The private value of the test is always higher in a dynamic model with reclassification than without reclassification when the two types H and U agents undertake effort in the two models and when only type- H agents undertake effort in the model without reclassification. The impact of reclassification on the private value of the test is higher for high value of p_H^1 and for low value of p_L . In other words, the positive effect of reclassification on the private value of the test is greater the higher the discrimination risk. The intuition on the positive impact of reclassification on the private value of the test is similar to the case where no one undertakes prevention effort.

We note that $\tilde{\phi}_{1R} < \tilde{\phi}_{1NR}$ since α_1 is strictly positive. Therefore, when $\tilde{\phi}_{1R}$ exists,

reclassification leads individuals to take the test for lower values of the cost of effort compared to the case where there is no reclassification.

We now analyze the incentives to gather genetic information when only informed high risk individuals undertake effort.

2.4.3.3 Only type- H agents undertake effort

Only type- H undertakes effort when $\max(\phi_H, \phi_U) = \phi_H$ and $\phi_U < \phi < \phi_H$. In this case, the private value of the test is given by:

$$\psi^{1,0} = \lambda U_H^1 + (1 - \lambda)U_L - U_U^0, \text{ i.e.,}$$

$$\begin{aligned} \psi^{1,0} = & \lambda[v(C_H^1) - \phi] + (1 - \lambda)v(C_L) - v(C_U^0) + \\ & \beta[(\lambda p_H^1 + (1 - \lambda)p_L)v(C_h) + \lambda(1 - p_H^1)v(C_H^1) + \\ & (1 - \lambda)(1 - p_L)v(C_L) - p_U^0v(C_h) - (1 - p_U^0)v(C_U^0)]. \end{aligned}$$

Replacing p_U^0 of the second term in the third line and rearranging the expression, we obtain :

$$\psi^{1,0} = (1 + \beta)[\lambda v(C_H^1) + (1 - \lambda)v(C_L) - v(C_U^0)] + \beta\alpha_2 - \lambda\phi, \quad (2.9)$$

with $\alpha_2 = p_U^0v(C_U^0) - \lambda p_H^1v(C_H^1) - (1 - \lambda)p_Lv(C_L) - \lambda(p_H^0 - p_H^1)v(C_h)$.

The term $\beta\alpha_2$ reflects how reclassification affects the private value of the test.

The expression inside the brackets in equation (2.9) can be positive or negative, and its sign depends on the prevention efficiency and the loss of utility generated by genetic discrimination. Given that we assume that $\Delta > \tilde{\Delta}$ in the dynamic model with reclassification, the expression inside the bracket is strictly positive. The last term measures the cost of effort for informed high risk individuals. We have the following proposition¹⁹ :

¹⁹See proof in appendix 8.

Proposition 3. When $\max(\phi_H, \phi_U) = \phi_H$ and $\phi_U < \phi < \phi_H$,

- (i) the private value of the test is increasing in ϕ and p_H^1 , and decreasing in p_h and p_H^0 ,
- (ii) the impact of reclassification on the private value of the test is undetermined, increasing in p_h and decreasing in p_L ,
- (iii) the private value of the test can be decreasing or increasing in β according to the parameters value,
- (iv) the sign of the private value of the test can be positive or negative according to the parameters value,
- (v) when $\psi^{1,1}(\phi_U) > 0$ and $\psi^{0,0}(\phi_H) < 0$, there exists a unique value ϕ , denoted by $\tilde{\phi}_{2R}$, such that $\psi^{1,0}(\tilde{\phi}_{2R}) = 0$ and $\phi_U < \tilde{\phi}_{2R} < \phi_H$,
- (vi) if the effect of reclassification is positive (i.e., $\alpha_2 > 0$), then the private value of the test with reclassification is higher than without reclassification when the cost of effort is such that types U and H or type- H agents undertake effort without reclassification.

The effect of reclassification on the private value of the test is more likely positive for high values of p_h and low values of p_L . It is useful to interpret the terms of α_2 in order to understand the impact of reclassification on the private value of the test. Indeed, α_2 is the difference between the second period expected utility of an uninformed individual who did not get sick in the first period and the second period expected utility of informed high risk and low risk individuals who did not get sick in the previous period when there is no reclassification and when agents do not undertake effort (α_0), plus the difference between the second period expected

utility of prevention effort of informed high risk individuals who got sick in the first period when they are reclassified and not. We know from Proposition 1 that $\alpha_0 > 0$. However, the second period expected utility of prevention effort of informed high risk individuals who got sick in the first period without reclassification can be higher or lower than with reclassification (x_H). As a result, reclassification may impact positively or negatively the private value of the test.

The private value of the test is more likely positive when prevention efficiency is large, the cost of effort is not large, the loss of utility resulting from genetic discrimination is low and for high values of p_h . In the first period, the expected utility of informed agents can be higher or lower than their expected utility when they stay informed. Indeed, agents make a trade off between the utility of the test (prevention utility of effort) and its cost (the effort cost for type- H agent and the loss of utility resulting from genetic discrimination).

In the second period, the expected utility of informed high risk and low risk individuals who did not get sick in the previous period can be higher or lower than the expected utility of uninformed individuals who did not get sick in the first period. Indeed, in the second period agents make a trade-off between the positive effect of reclassification (α_0), the prevention utility of effort ($\lambda(\phi_{max} + x_H)$), and the negative effect of genetic discrimination. When the positive effects dominate the negative impact of genetic discrimination, the private value of the test is greater the higher agents value their second period.

We formally identify, when there exists, the prevention cost $\tilde{\phi}_{2R}$, below which the private value of the test is positive. We set $\psi^{1,0}$ to zero. We have then:

$$\tilde{\phi}_{2R} = \frac{(1 + \beta)[\lambda v(C_H^1) + (1 - \lambda)v(C_L) - v(C_U^0)] + \beta\alpha_2}{\lambda}$$

In fact, $\tilde{\phi}_{2R}$ can be higher or smaller than $\tilde{\phi}_{2NR}$ since α_2 can be positive or nega-

tive. Therefore, reclassification can reduce or expand the effort cost below which individuals take the test and undertake effort if the test result is positive, compared to the dynamic model without reclassification. Thus, when the intensity of reclassification is high, agents perform the test for higher values of ϕ with reclassification than without reclassification.

We now analyze the case where only type- U agents undertake prevention action.

2.4.3.4 Only type- U agents undertake effort

This case is specific to reclassification. It corresponds to the case where reclassification leads type- U agents to undertake prevention effort more often than type- H agents. When $\max(\phi_H, \phi_U) = \phi_U$ and $\phi_H < \phi < \phi_U$, the private value of the test is defined as follows :

$$\begin{aligned}\psi^{0,1} &= \lambda U_H^0 + (1 - \lambda)U_L - U_U^1, \\ \psi^{0,1} &= \lambda v(C_H^0) + (1 - \lambda)v(C_L) - [v(C_U^1) - \phi] + \\ &\quad \beta[(\lambda p_H^0 + (1 - \lambda)p_L)v(C_h) + \lambda(1 - p_H^0)v(C_H^0) + \\ &\quad (1 - \lambda)(1 - p_L)v(C_L) - p_U^1 v(C_h) - (1 - p_U^1)v(C_U^1)].\end{aligned}$$

After replacing p_U^1 of the second term in the third line and simplifying the expression, we obtain :

$$\psi^{0,1} = (1 + \beta)[\lambda v(C_H^0) + (1 - \lambda)v(C_L) - v(C_U^1)] + \beta\alpha_3 + \phi, \quad (2.10)$$

with $\alpha_3 = p_U^1 v(C_U^1) - \lambda p_H^0 v(C_H^0) - (1 - \lambda)p_L v(C_L) + \lambda(p_H^0 - p_H^1)v(C_h)$.

The term $\beta\alpha_3$ captures the impact of reclassification on the private value of the test. The expression inside the brackets in equation (2.10) is strictly negative, and represents the negative impact of genetic discrimination and the utility of prevention action for uninformed individuals. The last term measures the cost of prevention

effort for uninformed individuals.

We have the following proposition ²⁰:

Proposition 4. *When $\max(\phi_H, \phi_U) = \phi_U$ and $\phi_H < \phi < \phi_U$,*

- (i) the private value of the test is increasing in ϕ and p_H^1 , decreasing in p_h and p_H^0 ,*
- (ii) the impact of reclassification on the private value of the test is decreasing in p_L and p_h , and is more likely positive if the intensity of reclassification is low,*
- (iii) the private value of the test can be increasing or decreasing in β according to the parameters value,*
- (iv) the sign of the private value of the test can be positive or negative according to the parameters value,*
- (v) when $\psi^{1,1}(\phi_H) < 0$ and $\psi^{0,0}(\phi_U) > 0$, there exists a unique value ϕ , denoted by $\tilde{\phi}_{3R}$, such that $\psi^{0,1}(\tilde{\phi}_{3R}) = 0$ and $\phi_H < \tilde{\phi}_{3R} < \phi_U$,*
- (vi) the private value of the test with reclassification can be higher or lower than without reclassification according to the ordering of the thresholds.*

When the cost of effort is such that only uninformed agents undertake effort, the impact of reclassification on the private value of the test is undetermined and depends on the prevention actions of agents in the model without reclassification. When no agent undertakes effort in the model without reclassification, the private value of the test with reclassification is higher than without reclassification if $\alpha_3 > 0$. However, when types H and U agents undertake effort in the context without reclassification, the private value of the test with reclassification is lower than without

²⁰See proof in appendix 9.

reclassification if $\alpha_3 < 0$. The private value of the test with reclassification is more likely positive for high values of the cost of effort (i.e., for ϕ close to ϕ_U) and p_H^1 (i.e., low prevention efficiency) and for low values of p_h and p_H^0 . In the first period, individuals make a trade off between the loss of utility resulting from genetic discrimination, the utility and the cost of prevention effort of uninformed agents. In the second period, agents make a trade off between the loss utility resulting from genetic discrimination, the positive impact of reclassification (α_0), and the prevention utility of effort for uninformed agents. It should be noted that this case is singular since reclassification leads uninformed individuals to undertake effort more often than informed high risk individuals. Compared to a dynamic model without reclassification, individuals may perform the test in the dynamic model with reclassification while type- H agents do not undertake prevention action but agents would undertake effort if they stay uninformed.

We formally identify, when there exists, the prevention cost, $\tilde{\phi}_{3R}$, above which the private value of the test is positive. To do so, we set $\psi^{0,1}$ equal to zero. We obtain :

$$\tilde{\phi}_{3R} = (1 + \beta)[v(C_U^1) - \lambda v(C_H^0) - (1 - \lambda)v(C_L)] - \beta\alpha_3.$$

We note that $\tilde{\phi}_{3R}$ is higher and increasing with β when α_3 is negative since $v(C_U^1) - \lambda v(C_H^0) - (1 - \lambda)v(C_L) > 0$. $\tilde{\phi}_{3R}$ can be higher or lower than $\tilde{\phi}_{1NR}$ and $\tilde{\phi}_{2NR}$. Therefore, reclassification can lead individuals to take the test for lower or higher values of the effort cost relatively to the case without reclassification. Furthermore, we note that $\tilde{\phi}_{1R}$ does not exist when $\tilde{\phi}_{3R}$ exists since the private value of the test is always increasing in ϕ when $\phi_U > \phi_H$ and when agents undertake effort.

In summary, we notice that the private value of the test is not strictly negative in the disclosure duty regulation in a two periods model with reclassification, regardless

of whether there is efficient prevention action. We also note that the private value of the test can be strictly positive or negative for all ϕ (See proof in appendix 10). Finally, we notice in particular that the private value of the test is strictly higher in the dynamic model with reclassification than without reclassification when the effort cost is such that no individual undertakes prevention or both types U and H agents undertake prevention action in the two models.

2.4.3.5 Comparison of thresholds to take the test

In the static model, let us denote $\tilde{\phi}_{1S}$, the value of ϕ above which the private value of the test is positive and $\tilde{\phi}_{2S}$ the value of ϕ below which the private value of the test is positive. Thus, agents perform the test in a static model for all $\tilde{\phi}_{1S} < \phi < \tilde{\phi}_{2S}$.

We have the following conditions on the effort costs thresholds to take the test $\tilde{\phi}_{1S} < \tilde{\phi}_{2S}$; $\tilde{\phi}_{1NR} < \tilde{\phi}_{2NR}$; $\tilde{\phi}_{1S} < \tilde{\phi}_{1NR}$ and $\tilde{\phi}_{2S} < \tilde{\phi}_{2NR}$.

There are two possible cases when comparing the thresholds to take the test in the dynamic model without reclassification and in a static model.

We obtain :

1. $0 < \tilde{\phi}_{1S} < \tilde{\phi}_{1NR} < \tilde{\phi}_{2S} < \tilde{\phi}_{2NR}$;
2. $0 < \tilde{\phi}_{1S} < \tilde{\phi}_{2S} < \tilde{\phi}_{1NR} < \tilde{\phi}_{2NR}$.

We know from Propositions 1 – 4 that $\tilde{\phi}_{1R} < \tilde{\phi}_{2R}$; $\tilde{\phi}_{1R} < \tilde{\phi}_{1NR}$; $\tilde{\phi}_{2R}$ and $\tilde{\phi}_{3R}$ can be higher or lower than $\tilde{\phi}_{2NR}$ and $\tilde{\phi}_{1NR}$. Moreover, we know that if $\tilde{\phi}_{1R}$ exists, then $\tilde{\phi}_{3R}$ does not exist since the private value of the test is strictly increasing in ϕ in this case. When $\tilde{\phi}_{1R}$, $\tilde{\phi}_{2R}$ or $\tilde{\phi}_{3R}$ exist, there are 6 possible cases when comparing the effort cost thresholds to take the test in the dynamic model without reclassification and with reclassification. We obtain the following cases :

1. $0 < \tilde{\phi}_{1R} < \tilde{\phi}_{1NR} < \tilde{\phi}_{2R} < \tilde{\phi}_{2NR}$;

$$2. 0 < \tilde{\phi}_{1R} < \tilde{\phi}_{1NR} < \tilde{\phi}_{2NR} < \tilde{\phi}_{2R};$$

$$3. 0 < \tilde{\phi}_{1R} < \tilde{\phi}_{2R} < \tilde{\phi}_{1NR} < \tilde{\phi}_{2NR};$$

$$4. 0 < \tilde{\phi}_{1NR} < \tilde{\phi}_{3R} < \tilde{\phi}_{2NR};$$

$$5. 0 < \tilde{\phi}_{1NR} < \tilde{\phi}_{2NR} < \tilde{\phi}_{3R};$$

$$6. 0 < \tilde{\phi}_{3R} < \tilde{\phi}_{1NR} < \tilde{\phi}_{2NR}.$$

When individuals always take or never take the test in the model with reclassification, i.e., $\tilde{\phi}_{1R}$, $\tilde{\phi}_{2R}$ or $\tilde{\phi}_{3R}$ do not exist, we obtain the following ordering of thresholds with and without reclassification: $0 < \tilde{\phi}_{1NR} < \tilde{\phi}_{2NR}$.

When individuals take the test with reclassification for all $\phi > \tilde{\phi}_{1R}$, i.e., $\tilde{\phi}_{2R}$ or $\tilde{\phi}_{3R}$ does not exist, we obtain the following ordering of the thresholds with and without reclassification :

$$0 < \tilde{\phi}_{1R} < \tilde{\phi}_{1NR} < \tilde{\phi}_{2NR}.$$

Finally, when $\tilde{\phi}_{1R}$ does not exist but $\tilde{\phi}_{2R}$ does exist, we have the following cases :

$$1. 0 < \tilde{\phi}_{1NR} < \tilde{\phi}_{2R} < \tilde{\phi}_{2NR};$$

$$2. 0 < \tilde{\phi}_{1NR} < \tilde{\phi}_{2NR} < \tilde{\phi}_{2R};$$

$$3. 0 < \tilde{\phi}_{2R} < \tilde{\phi}_{1NR} < \tilde{\phi}_{2NR};$$

When comparing the thresholds to take the test in the dynamic model without reclassification, with reclassification and in a static model, when $\tilde{\phi}_{1R}$, $\tilde{\phi}_{2R}$ or $\tilde{\phi}_{3R}$ exist, we identify 20 possible cases given the conditions on the thresholds. As previously, it is not useful to analyze all possible cases because this does not bring new

insights to the intuition.

2.4.4 Graphical analysis

We provide four graphical illustrations. In the first three numerical examples, we use the same parameters value of BRCA genes as in the numerical example without reclassification of figure 2.5. In the last one, we use the same parameters value as in figure 2.7 in order to illustrate the case where type- U agents undertake effort more often than type- H agents in the model with reclassification.

2.4.4.1 Case where $p_h = 0.6$, $x_H > x_U \Rightarrow \phi_H > \phi_U$ and $\phi_H > (1 + \beta)\phi_{\max}$

Figure 2.8 illustrates a numerical example when the private value of the test is strictly positive in the model with reclassification for all ϕ , and compares the private value of the test in the model with and without reclassification as well as in a static model. In Figure 2.8, with reclassification (and when $p_h = 0.6$), we have $\phi_U = 0.056$, $\phi_H = 0.73$. Without reclassification, recall that $(1 + \beta)\phi_{\min} = 0.045$, $(1 + \beta)\phi_{\max} = 0.7$, $\tilde{\phi}_{1NR} = 0.00068$, and $\tilde{\phi}_{2NR} = 0.68$.

We notice that the incentive to take genetic testing with reclassification is strictly higher than without reclassification and a static model.

2.4.4.2 Case where $p_h = 0.4$, $x_H < x_U$, $\phi_H > \phi_U$ and $\phi_H < (1 + \beta)\phi_{\max}$

Figure 2.9 illustrates a numerical example where the private value of the test is strictly positive for all ϕ in the dynamic model with reclassification ($p_h = 0.4$) with $\phi_H < (1 + \beta)\phi_{\max}$, and depicts the private value of the test according to ϕ in a static model as well as with reclassification and without reclassification. We have $\phi_U = 0.052$ and $\phi_H = 0.67$.

Without reclassification, recall that $(1 + \beta)\phi_{\min} = 0.045$, $(1 + \beta)\phi_{\max} = 0.7$,

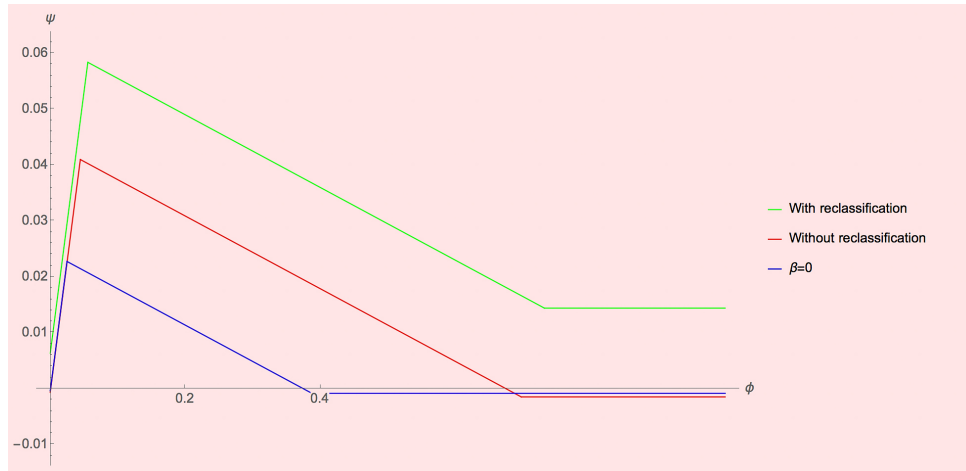


Figure 2.8: The private value of the test according to ϕ , with $p_h = 0.6$

$\tilde{\phi}_{1NR} = 0.00068$, and $\tilde{\phi}_{2NR} = 0.68$. As precedently, the incentive to perform genetic testing with reclassification is strictly higher than without reclassification and a static model.

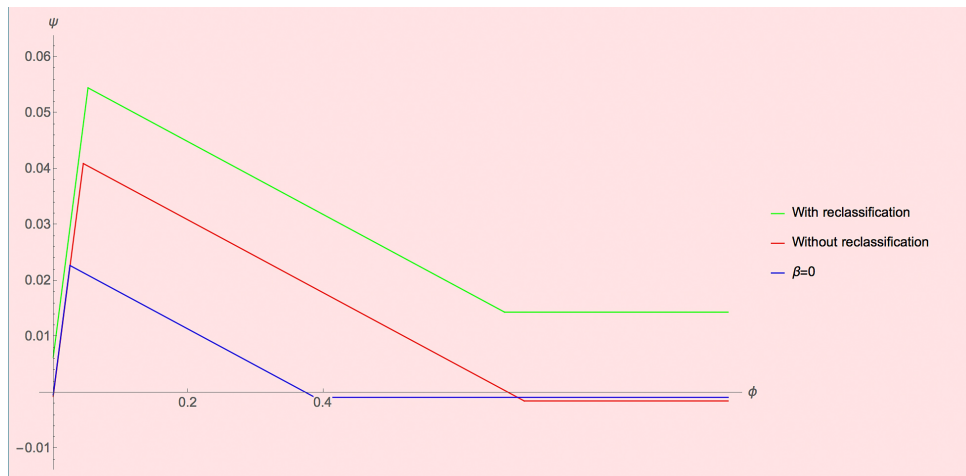


Figure 2.9: The private value of the test according to ϕ , with $p_h = 0.4$

2.4.4.3 Case where the private value of the test with reclassification can be positive or negative according to ϕ

We provide a graphical illustration where the private value of the test with reclassification is positive for intermediate values of ϕ , and compare the private value of the test with reclassification and without reclassification. In figure 2.10, we use the same values of the parameters as in figure 2.8 except $\beta = 0.02$. With reclassification, we have $\phi_U = 0.02553$, $\phi_H = 0.39637$, $\tilde{\phi}_{1R} = 0.00020$, $\tilde{\phi}_{2NR} = 0.38984$. Without reclassification, we have $(1 + \beta)\phi_{\min} = 0.02525$, $(1 + \beta)\phi_{\max} = 0.39551$, $\tilde{\phi}_{1NR} = 0.00039$, and $\tilde{\phi}_{2NR} = 0.38287$.

In this case, the incentive to take genetic testing with reclassification is strictly higher than without reclassification. The set of values of ϕ for which the private value of the test with reclassification is positive is larger than without reclassification.

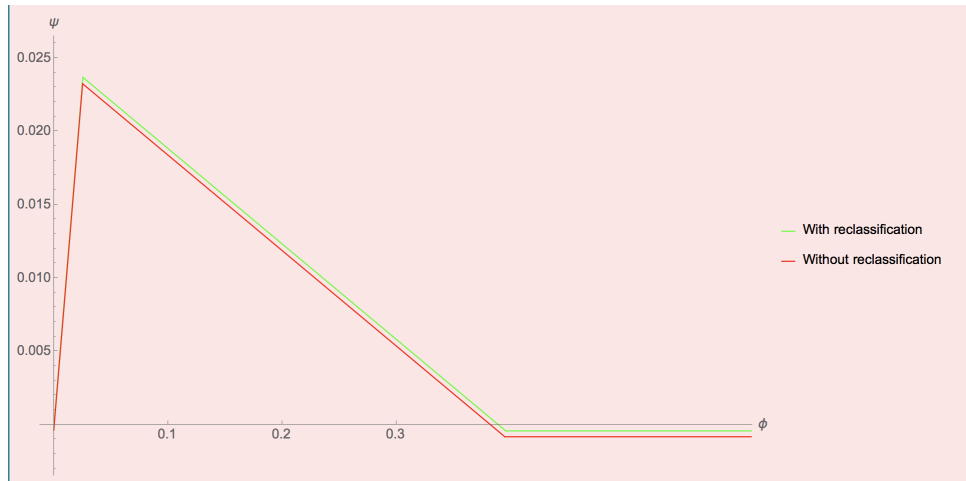


Figure 2.10: The private value of the test with versus without reclassification according to ϕ

2.4.4.4 Case $x_H < x_U$ and $\phi_H < \phi_U$

If we take the values of the parameters of figure 2.5, there is no value of p_h such that $\phi_H < \phi_U$. In order to illustrate this case, we consider the same values of the parameters as in figure 2.7, with $p_h = 0.92$. Figure 2.11 illustrates a numerical example where the private value of the test is strictly positive for all ϕ in the dynamic model with reclassification, and depicts the private value of the test with reclassification versus without reclassification according to ϕ , with $\phi_U > \phi_H$. We have $\phi_U = 0.50205$ and $\phi_H = 0.50151$.

The incentive of agents to take the test with reclassification is strictly positive and higher than without reclassification for all ϕ .

We now move to the welfare analysis in order to analyze the impact of reclassification on the ex ante utility of agents.

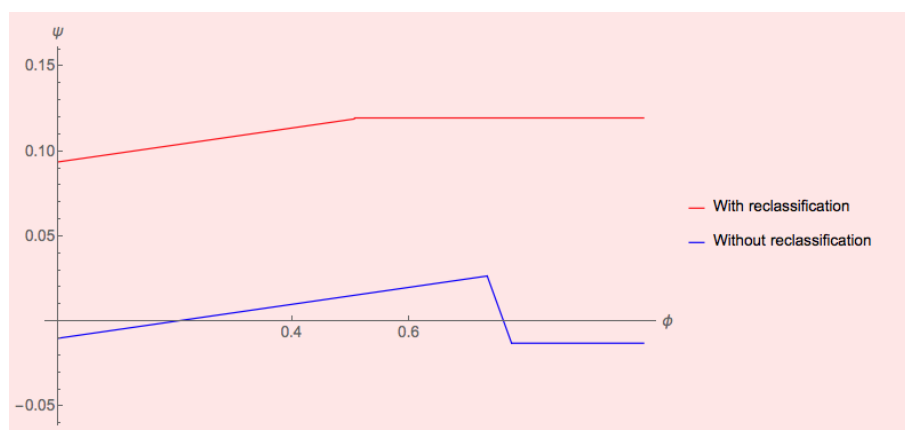


Figure 2.11: The private value of the test according to ϕ , with $\phi_H < \phi_U$

2.5 Welfare analysis

We provide in this section a welfare analysis in order to assess the impact of reclassification on the ex ante social welfare. The ex ante social welfare is defined as the ex ante utility of agents. We first compare the social welfare achieved in the dynamic model without reclassification to a static model. Afterwards, we compare the dynamic model with reclassification to the dynamic model without reclassification.

2.5.1 Static vs dynamic without reclassification

There are two possible cases when comparing the effort cost thresholds to take the test in the dynamic model without reclassification and a static model. The two cases are:

1. $0 < \tilde{\phi}_{1S} < \tilde{\phi}_{1NR} < \tilde{\phi}_{2S} < \tilde{\phi}_{2NR}$;
2. $0 < \tilde{\phi}_{1S} < \tilde{\phi}_{2S} < \tilde{\phi}_{1NR} < \tilde{\phi}_{2NR}$.

In the first case, we have $\tilde{\phi}_{1NR} < \tilde{\phi}_{2S}$, while $\tilde{\phi}_{2S} < \tilde{\phi}_{1NR}$ in the second case. We can determine the condition on β which ensures that $\tilde{\phi}_{1NR} < \tilde{\phi}_{2S}$.

We have

$$\tilde{\phi}_{1NR} = \frac{(1 + \beta)[v(C_U^1) - (\lambda v(C_H^1) + (1 - \lambda)v(C_L))] }{1 - \lambda} = (1 + \beta)\tilde{\phi}_{1S}.$$

Thus, $\tilde{\phi}_{1NR} < \tilde{\phi}_{2S}$ provided that

$$\beta < \frac{\tilde{\phi}_{2S} - \tilde{\phi}_{1S}}{\tilde{\phi}_{1S}}.$$

We know that agents do not take the test but undertake effort in the dynamic model without reclassification (resp., in the static model) for all $0 < \phi < \tilde{\phi}_{1NR}$ (resp., $0 < \phi < \tilde{\phi}_{1S}$). Agents take the test and undertake effort if they are of type- H in the dynamic model without reclassification (resp., in the static model) for all

$\tilde{\phi}_{1NR} < \phi < \tilde{\phi}_{2NR}$ (resp., $\tilde{\phi}_{1S} < \phi < \tilde{\phi}_{2S}$). When $\phi > \tilde{\phi}_{2NR}$ (resp., $\phi > \tilde{\phi}_{2S}$) agents do not take the test and do not undertake effort in the dynamic model without reclassification (resp., in the static model).

For all $0 < \phi < \tilde{\phi}_{1S}$, individuals do not take the test and undertake prevention action in the two models regardless of the ordering of the threshold effort costs.

In the static model, the social welfare is defined as follows :

$$\text{SWSU}(1) = v(C_U^1) - \phi.$$

The social welfare in the dynamic model without reclassification is given by :

$$\text{SWNRU}(1) = (1 + \beta)v(C_U^1) - \phi.$$

We conclude that the social welfare in the dynamic model without reclassification is higher than a static model since β is positive.

For all $\phi > \tilde{\phi}_{2NR}$, individuals do not take the test and do not undertake prevention in the two models regardless of the ordering of the thresholds. In the static model, the ex ante social welfare is defined as follows :

$$\text{SWSU}(0) = v(C_U^0).$$

The social welfare in the dynamic model without reclassification is given by :

$$\text{SWNRU}(0) = (1 + \beta)v(C_U^0).$$

We conclude that the social welfare in the dynamic model without reclassification is higher than a static model since $\beta > 0$.

For all $\phi \in]\tilde{\phi}_{1S}, \tilde{\phi}_{1NR}[$ in case 1 or for all $\phi \in]\tilde{\phi}_{1S}, \tilde{\phi}_{2S}[$ in case 2, individuals take the test only in the static model, and undertake prevention action in the two models. The ex ante social welfare in the static model is defined as follows :

$$\text{SWSI}(1) = \lambda v(C_H^1) + (1 - \lambda)v(C_L) - \lambda\phi.$$

The social welfare in the dynamic model without reclassification is given by :

$$\text{SWNRU}(1) = (1 + \beta)v(C_U^1) - \phi.$$

$\text{SWNRU}(1) > \text{SWSI}(1)$ provided that

$$(1 + \beta)v(C_U^1) - \phi > \lambda v(C_H^1) + (1 - \lambda)v(C_L) - \lambda\phi.$$

We know from Result 3 that

$$(1 + \beta)[\lambda v(C_H^1) + (1 - \lambda)v(C_L) - v(C_U^1)] + (1 - \lambda)\phi < 0 \text{ for all } 0 < \phi < \tilde{\phi}_{1NR}.$$

It implies that

$$\lambda v(C_H^1) + (1 - \lambda)v(C_L) - (1 + \beta)v(C_U^1) + (1 - \lambda)\phi < 0 \text{ for all } 0 < \phi < \tilde{\phi}_{1NR}.$$

We conclude that the social welfare in the dynamic model without reclassification is higher than the static model.

In the first case, when $\tilde{\phi}_{1NR} < \phi < \tilde{\phi}_{2S}$, this is the only interval where individuals undertake the test and perform prevention in the two models. The ex ante utility in the static model is defined as follows :

$$\text{SWSI}(1) = \lambda v(C_H^1) + (1 - \lambda)v(C_L) - \lambda\phi.$$

The ex ante social welfare in the dynamic model without reclassification is given by :

$$\text{SWNRI}(1) = (1 + \beta)[\lambda v(C_H^1) + (1 - \lambda)v(C_L)] - \lambda\phi.$$

The ex ante social welfare is higher in the dynamic model without reclassification than a static since $\beta > 0$.

In the second case, the decision for all $\tilde{\phi}_{2S} < \phi < \tilde{\phi}_{1NR}$ is specific because it is the only interval where individuals do not perform the test in the two models, but undertake effort only in the dynamic model. In the static model, the ex ante social welfare is defined as follows :

$$\text{SWSU}(0) = v(C_U^0).$$

The social welfare in the dynamic model without reclassification is given by :

$$\text{SWNRU}(1) = (1 + \beta)v(C_U^1) - \phi.$$

We know that for all $\phi \in [\tilde{\phi}_{2S}, \tilde{\phi}_{1NR}]$,

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

It implies that

$$(1 + \beta)(v(C_U^1) - v(C_U^0)) - \phi > 0.$$

We show that $\text{SWNRU}(1) - \text{SWSU}(0) > 0$.

$$\text{SWNRU}(1) - \text{SWSU}(0) = (1 + \beta)v(C_U^1) - v(C_U^0) - \phi > 0,$$

since

$$(1 + \beta)v(C_U^1) - v(C_U^0) - \phi > (1 + \beta)(v(C_U^1) - v(C_U^0)) - \phi > 0.$$

We conclude that the social welfare in the dynamic model without reclassification is higher than a static model.

Finally, for all $\phi \in]\tilde{\phi}_{2S}, \tilde{\phi}_{2NR}[$ in case 1 or $\phi \in]\tilde{\phi}_{1NR}, \tilde{\phi}_{2NR}[$ in case 2, individuals take the test and undertake effort in the dynamic model while they do not take the

test and do not undertake prevention action in the static model.

In the static model, the ex ante social welfare is defined as follows :

$$\text{SWSU}(0) = v(C_U^0).$$

The ex ante social welfare in the dynamic model without reclassification is given by :

$$\text{SWNRI}(1) = (1 + \beta)[\lambda v(C_H^1) + (1 - \lambda)v(C_L)] - \lambda\phi.$$

$\text{SWNRI}(1) > \text{SWSU}(0)$ provided that

$$(1 + \beta)[\lambda v(C_H^1) + (1 - \lambda)v(C_L)] - \lambda\phi > v(C_U^0).$$

We know from Result 4 that

$$(1 + \beta)[\lambda v(C_H^1) + (1 - \lambda)v(C_L) - v(C_U^0)] - \lambda\phi > 0 \text{ for all } (1 + \beta)\phi_{min} < \phi < \tilde{\phi}_{2NR}.$$

It implies that

$$(1 + \beta)[\lambda v(C_H^1) + (1 - \lambda)v(C_L)] - v(C_U^0) - \lambda\phi > (1 + \beta)[\lambda v(C_H^1) + (1 - \lambda)v(C_L) - v(C_U^0)] - \lambda\phi > 0 \text{ for all } \phi < \tilde{\phi}_{2NR}.$$

We conclude that the ex ante social welfare in the dynamic model is higher than a static model.

We summarize our results in the following proposition.

Proposition 5. *The social welfare of agents in the dynamic model without reclassification is higher than a static model regardless the ordering of the thresholds to take the test.*

When agents take the same decision regarding the test and prevention effort, the ex ante utility of agents without reclassification is higher than a static model since

$\beta > 0$. The more agents value their second period, the more the ex ante utility without reclassification is high. When agents take different decision regarding the test and prevention effort, the ex ante utility of agents without reclassification is higher than a static model since they take the test if the utility of the test is higher than its cost.

Social welfare in lines	0 to $\tilde{\phi}_{1S}$	$\tilde{\phi}_{1S}$ to $\tilde{\phi}_{1NR}$	$\tilde{\phi}_{1NR}$ to $\tilde{\phi}_{2S}$	$\tilde{\phi}_{2S}$ to $\tilde{\phi}_{2NR}$	$\phi > \tilde{\phi}_{2NR}$
Static	SWSU(1)	SWSI(1)		SWSU(0)	
Dynamic without R	SWNRU(1)		SWNRI(1)		SWNRU(0)

Table 2.1: Ex ante utility according to ϕ in case 1

Social welfare in lines	0 to $\tilde{\phi}_{1S}$	$\tilde{\phi}_{1S}$ to $\tilde{\phi}_{2S}$	$\tilde{\phi}_{2S}$ to $\tilde{\phi}_{1NR}$	$\tilde{\phi}_{1NR}$ to $\tilde{\phi}_{2NR}$	$\phi > \tilde{\phi}_{2NR}$
Static	SWSU(1)	SWSI(1)	SWSU(0)		
Dynamic without R	SWNRU(1)			SWNRI(1)	SWNRU(0)

Table 2.2: Ex ante utility according to ϕ in case 2

Tables 2.1 and 2.2 summarize the ex ante utility achieved in the two cases in the two models according to the cost of effort. Figure 2.12 illustrates a numerical example of case 1. For this graphical illustration, we use the same values of the parameters of figure 2.5, with $\tilde{\phi}_{1S} = 0.00038$, $\tilde{\phi}_{2S} = 0.38$, $\tilde{\phi}_{1NR} = 0.00068$ and $\tilde{\phi}_{2NR} = 0.68$. For this numerical example, the value of β above which $\tilde{\phi}_{1NR} > \tilde{\phi}_{2S}$ is equal to 999, which is an unrealistic value. Figure 2.13 illustrates a numerical example of case 2, i.e., when $\tilde{\phi}_{1NR} > \tilde{\phi}_{2S}$. We consider the same values of the parameters of figure 2.12 except $\beta = 1052$. For this numerical example, $\tilde{\phi}_{1S} = 0.00038$, $\tilde{\phi}_{2S} = 0.38$, $\tilde{\phi}_{1NR} = 0.40014$ and $\tilde{\phi}_{2NR} = 400.14$. We note that the curve representing the ex ante utility according to effort cost in the dynamic model without reclassification is always above that of the static model in the two figures.

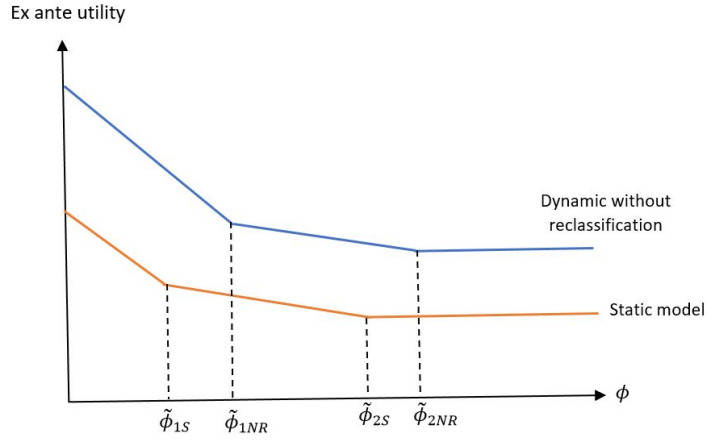


Figure 2.12: The ex ante utility according to ϕ without reclassification and in the static model of case 1

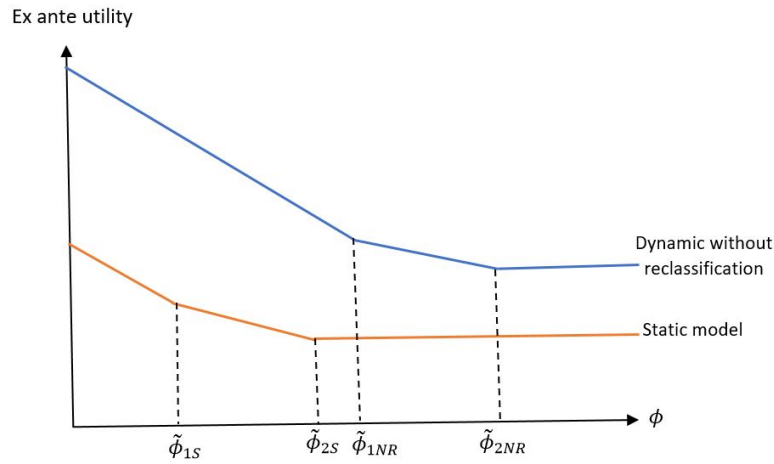


Figure 2.13: The ex ante utility according to ϕ without reclassification and in the static model of case 2

2.5.2 Dynamic model with vs without reclassification

We now turn to the impact of reclassification on the ex ante social welfare. Propositions 1–4 show that reclassification may lead individuals to always or never take the test for all ϕ (case of the non existence of ϕ for which the private value of the test is null). In addition, reclassification may lead agents to take the test for lower values of the cost of effort than without reclassification, particularly when the cost of effort

is such that types H and U agents undertake effort (i.e., $\tilde{\phi}_{1R} < \tilde{\phi}_{1NR}$). Furthermore, when the cost of the effort is such that type-H or type-U agent undertakes effort, the value of the effort cost below which the private value of the test with reclassification is positive may be lower or higher than without reclassification.

The ex ante utility with reclassification is always lower than without reclassification even when agents take the same decision regarding the test and prevention action, and regardless of the ordering of the thresholds to take the test because of the loss of utility generated by reclassification in the second period. The intuition is as follows. The ex ante utility of agents with reclassification is always lower than without reclassification when they take the same decision regarding the test and prevention effort because of the loss of utility resulting from reclassification in the second period. When agents do not take the test in the model without reclassification, while they take the test in the model with reclassification, they got a lower ex ante utility with reclassification because of the negative effect of genetic discrimination and reclassification on their utility. When the test is taken in the model without reclassification, but agents stay uninformed in the model with reclassification, the ex ante utility without reclassification is higher than with reclassification because agents take the test in the model without reclassification when the utility of the test is higher than its costs. Finally, reclassification will impact negatively the ex ante utility of agents even if the utility of prevention effort with reclassification is high (i.e., for high value of p_h) because the loss of utility resulting from reclassification is higher.

Let us compare the ex ante utility of agents with and without reclassification, especially when in the model with reclassification individuals take the test for all ϕ

and when ϕ_H is higher than ϕ_U and $(1 + \beta)\phi_{max}$. We then obtain the following effort cost ranking $0 < \tilde{\phi}_{1NR} < \tilde{\phi}_{2NR} < \phi_H$ since the private value of the test is always positive in the dynamic model with reclassification.

For all $0 < \phi < \tilde{\phi}_{1NR}$, agents undertake prevention effort and do not take the test in the model without reclassification, while agents take the test and type- H agents undertake prevention effort in the dynamic model with reclassification. When there is no reclassification, the ex ante utility is given by

$$\text{SWNRU}(1) = (1 + \beta)v(C_U^1) - \phi.$$

The social welfare is then decreasing in ϕ , with slope -1 . The ex ante utility of agents with reclassification is defined as follows :

$$\text{SWRI}(1) = (1 + \beta)[\lambda v(C_H^1) + (1 - \lambda)v(C_L)] - \lambda\phi.$$

The ex ante utility is decreasing in ϕ , with slope $-\lambda$.

We show that $\text{SWRI}(1) - \text{SWNRU}(1) < 0$:

$$\begin{aligned} \text{SWRI}(1) - \text{SWNRU}(1) &= (1 + \beta)[\lambda v(C_H^1) + (1 - \lambda)v(C_L) - v(C_U^1)] + (1 - \lambda)\phi \\ &\quad - \beta \times [\lambda p_H^1(v(C_H^1) - v(C_h)) + (1 - \lambda)p_L(v(C_L) - v(C_h))]. \end{aligned}$$

We know from item *vi*) of Result 3 that

$$(1 + \beta)[\lambda v(C_H^1) + (1 - \lambda)v(C_L) - v(C_U^1)] + (1 - \lambda)\phi < 0 \text{ for all } \phi < \tilde{\phi}_{1NR}.$$

It implies that $\text{SWRI}(1) - \text{SWNRU}(1) < 0$ since $v(C_H^1) > v(C_h)$ and $v(C_L) > v(C_h)$.

When $\tilde{\phi}_{1NR} < \phi < \tilde{\phi}_{2NR}$, agents take the test and undertake prevention effort in the dynamic model with and without reclassification. The ex ante utility of agents without reclassification is decreasing in ϕ and is given by :

$$\text{SWNRI}(1) = (1 + \beta)[\lambda v(C_H^1) + (1 - \lambda)v(C_L)] - \lambda\phi.$$

The ex ante utility of agents with reclassification is decreasing in ϕ and is defined as follows :

$$\begin{aligned} \text{SWRI}(1) &= (1 + \beta)[\lambda v(C_H^1) + (1 - \lambda)v(C_L)] - \lambda\phi - \\ &\quad \beta \times [\lambda p_H^1(v(C_H^1) - v(C_h)) + (1 - \lambda)p_L(v(C_L) - v(C_h))]. \end{aligned}$$

We show that $\text{SWRI}(1) - \text{SWNRI}(1) < 0$.

$$\text{SWRI}(1) - \text{SWNRI}(1) = -\beta \times [\lambda p_H^1(v(C_H^1) - v(C_h)) + (1 - \lambda)p_L(v(C_L) - v(C_h))] < 0$$

since $v(C_H^1) > v(C_h)$ and $v(C_L) > v(C_h)$.

For all $\tilde{\phi}_{2NR} < \phi < \phi_H$, agents do not take the test and do not undertake prevention effort without reclassification, while they take the test and undertake effort with reclassification.

The ex ante utility of agents without reclassification is defined by

$$\text{SWNRU}(0) = (1 + \beta)v(C_U^0).$$

The ex ante utility of agents with reclassification is given by

$$\begin{aligned} \text{SWRI}(1) &= (1 + \beta)[\lambda v(C_H^1) + (1 - \lambda)v(C_L)] - \lambda\phi - \\ &\quad \beta \times [\lambda p_H^1(v(C_H^1) - v(C_h)) + (1 - \lambda)p_L(v(C_L) - v(C_h))]. \end{aligned}$$

We show that $\text{SWRI}(1) - \text{SWNRU}(0) < 0$.

$$\begin{aligned} \text{SWRI}(1) - \text{SWNRU}(0) &= (1 + \beta)[\lambda v(C_H^1) + (1 - \lambda)v(C_L) - v(C_U^0)] - \lambda\phi - \\ &\quad \beta \times [\lambda p_H^1(v(C_H^1) - v(C_h)) + (1 - \lambda)p_L(v(C_L) - v(C_h))]. \end{aligned}$$

We know from item v) of Result 4 that

$$(1 + \beta)[\lambda v(C_H^1) + (1 - \lambda)v(C_L) - v(C_U^0)] - \lambda\phi < 0 \text{ for all } \phi > \tilde{\phi}_{2NR}.$$

It implies that $SWRI(1) - SWNRU(0) < 0$.

Finally, when $\phi > \phi_H$, agents do not take the test and do not undertake prevention effort in the dynamic model without reclassification, while they take the test and do not undertake effort with reclassification. The ex ante utility of agents without reclassification is defined as follows :

$$SWNRU(0) = (1 + \beta)v(C_U^0).$$

The ex ante utility of agents with reclassification is given by :

$$\begin{aligned} SWRI(0) &= (1 + \beta)[\lambda v(C_H^0) + (1 - \lambda)v(C_L)] - \\ &\beta \times [\lambda p_H^0(v(C_H^0) - v(C_h)) + (1 - \lambda)p_L(v(C_L) - v(C_h))]. \end{aligned}$$

We show that $SWRI(0) - SWNRU(0) < 0$.

$$\begin{aligned} SWRI(0) - SWNRU(0) &= (1 + \beta)[\lambda v(C_H^0) + (1 - \lambda)v(C_L) - v(C_U^0)] - \\ &\beta \times [\lambda p_H^0(v(C_H^0) - v(C_h)) + (1 - \lambda)p_L(v(C_L) - v(C_h))]. \end{aligned}$$

We know that $\lambda v(C_H^0) + (1 - \lambda)v(C_L) - v(C_U^0) < 0$ according to Jensen's inequality.

It implies that $SWRI(0) - SWNRU(0) < 0$.

Table 2.3 summarizes the ex ante utility of agents according to effort cost in the two models with and without reclassification, when agents always take the test in the dynamic model with reclassification.

Social welfare in lines	0 to $\tilde{\phi}_{1NR}$	$\tilde{\phi}_{1NR}$ to $\tilde{\phi}_{2NR}$	$\tilde{\phi}_{2NR}$ to ϕ_H	$\phi > \phi_H$
Without reclassification	SWNRU(1)	SWNRI(1)	SWNRU(0)	
With reclassification	SWRI(1)			SWRI(0)

Table 2.3: Ex ante utility of agents according to effort cost with and without reclassification, when the private value of the test with reclassification is positive for all ϕ .

Figure 2.14 represents the ex ante utility of agents as a function of the effort cost in the two models, especially when in the model with reclassification individuals take the test for all ϕ and when ϕ_H is higher than ϕ_U and $(1 + \beta)\phi_{max}$. We take the same values of the parameters of figure 7 for this numerical example, with $\phi_H = 0.73$, $\tilde{\phi}_{1NR} = 0.00068$, and $\tilde{\phi}_{2NR} = 0.68$. For the curve representing the

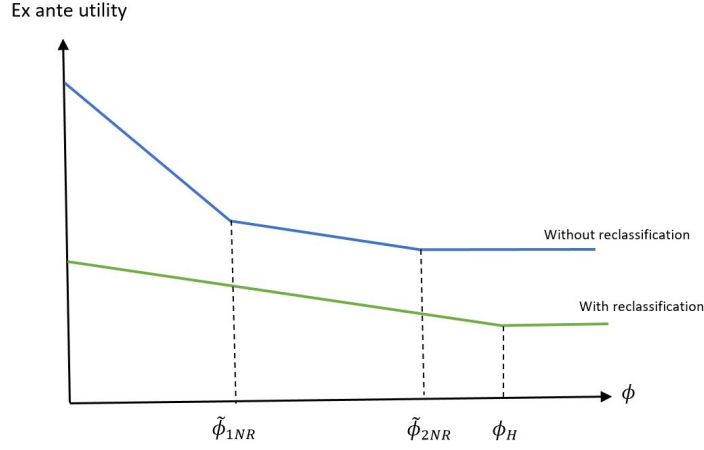


Figure 2.14: The ex ante utility according to ϕ with reclassification and without reclassification when agents always take the test with reclassification.

ex ante utility of agents with reclassification, we show only the value of ϕ_H because the private value of the test is always positive. In order to plot the curve representing the ex ante utility in the dynamic model without reclassification, we only need to show the effort cost affecting the decision regarding to the genetic testing. The vertical distance between the two curves in figure 2.14 measures the welfare loss resulting from reclassification.

We now compare the ex ante utility of agents when the private value of the test can be positive or negative in the two models, and also when $\tilde{\phi}_{1R}$ and $\tilde{\phi}_{2NR}$ exist, with $\phi_H > \phi_U$ and $\tilde{\phi}_{2R} > \tilde{\phi}_{2NR}$. We obtain the following ordering of the thresholds:

$$0 < \tilde{\phi}_{1R} < \tilde{\phi}_{1NR} < \tilde{\phi}_{2NR} < \tilde{\phi}_{2R}.$$

For all $0 < \phi < \tilde{\phi}_{1R}$, agents do not take the test but undertake prevention effort in the model without and with reclassification. The ex ante utility of agents without reclassification is defined by

$$\text{SWNRU}(1) = (1 + \beta)v(C_U^1) - \phi.$$

The ex ante utility of agents with reclassification is defined as follows :

$$\text{SWRU}(1) = (1 + \beta)v(C_U^1) - \phi - \beta \times [p_U^1(v(C_U^1) - v(C_h))].$$

We show that $\text{SWRU}(1) - \text{SWNRU}(1) < 0$.

$$\text{SWRU}(1) - \text{SWNRU}(1) = -\beta \times [p_U^1(v(C_U^1) - v(C_h))] < 0$$

since $v(C_U^1) > v(C_h)$.

When $\tilde{\phi}_{1R} < \phi < \tilde{\phi}_{1NR}$, agents take the test and type- H agents undertake prevention effort in the model with reclassification, while they do not take the test but undertake prevention effort in the model without reclassification. The ex ante utility of agents without reclassification is given by

$$\text{SWNRU}(1) = (1 + \beta)v(C_U^1) - \phi.$$

The ex ante utility of agents with reclassification is defined as follows :

$$\begin{aligned} \text{SWRI}(1) &= (1 + \beta)[\lambda v(C_H^1) + (1 - \lambda)v(C_L)] - \lambda\phi - \\ &\quad \beta \times [\lambda p_H^1(v(C_H^1) - v(C_h)) + (1 - \lambda)p_L(v(C_L) - v(C_h))]. \end{aligned}$$

We study the sign of $\text{SWRI}(1) - \text{SWNRU}(1)$ and show that is negative.

$$\begin{aligned} \text{SWRI}(1) - \text{SWNRU}(1) &= (1 + \beta)[\lambda v(C_H^1) + (1 - \lambda)v(C_L) - v(C_U^1)] + (1 - \lambda)\phi \\ &\quad - \beta \times [\lambda p_H^1(v(C_H^1) - v(C_h)) + (1 - \lambda)p_L(v(C_L) - v(C_h))]. \end{aligned}$$

We know from item *vi*) of Result 3 that

$$(1 + \beta)[\lambda v(C_H^1) + (1 - \lambda)v(C_L) - v(C_U^1)] + (1 - \lambda)\phi < 0 \text{ for all } \phi < \tilde{\phi}_{1NR}.$$

It implies that $SWRI(1) - SWNRU(1) < 0$.

For all $\tilde{\phi}_{1NR} < \phi < \tilde{\phi}_{2NR}$, agents take the test and undertake prevention effort in the dynamic model with and without reclassification. The ex ante utility of agents without reclassification is decreasing in ϕ and is given by

$$SWNRI(1) = (1 + \beta)[\lambda v(C_H^1) + (1 - \lambda)v(C_L)] - \lambda\phi.$$

The ex ante utility of agents with reclassification is defined as follows :

$$\begin{aligned} SWRI(1) &= (1 + \beta)[\lambda v(C_H^1) + (1 - \lambda)v(C_L)] - \lambda\phi - \\ &\quad \beta \times [\lambda p_H^1(v(C_H^1) - v(C_h)) + (1 - \lambda)p_L(v(C_L) - v(C_h))]. \end{aligned}$$

We show that $SWRI(1) - SWNRI(1) < 0$.

$$SWRI(1) - SWNRI(1) = -\beta \times [\lambda p_H^1(v(C_H^1) - v(C_h)) + (1 - \lambda)p_L(v(C_L) - v(C_h))] < 0.$$

When $\tilde{\phi}_{2NR} < \phi < \tilde{\phi}_{2R}$, agents take the test and undertake prevention effort in the model with reclassification, while they do not take the test and do not undertake prevention effort without reclassification. The ex ante utility of agents without reclassification is defined by

$$SWNRU(0) = (1 + \beta)v(C_U^0).$$

The ex ante utility of agents with reclassification is given by :

$$\begin{aligned} SWRI(1) &= (1 + \beta)[\lambda v(C_H^1) + (1 - \lambda)v(C_L)] - \lambda\phi \\ &\quad - \beta \times [\lambda p_H^1(v(C_H^1) - v(C_h)) + (1 - \lambda)p_L(v(C_L) - v(C_h))]. \end{aligned}$$

We show that $SWRI(1) - SWNRU(0) < 0$.

$$\begin{aligned} SWRI(1) - SWNRU(0) &= (1 + \beta)[\lambda v(C_H^1) + (1 - \lambda)v(C_L) - v(C_U^0)] - \lambda\phi \\ &\quad - \beta \times [\lambda p_H^1(v(C_H^1) - v(C_h)) + (1 - \lambda)p_L(v(C_L) - v(C_h))]. \end{aligned}$$

We know from item $v)$ of Result 4 that

$$(1 + \beta)[\lambda v(C_H^1) + (1 - \lambda)v(C_L) - v(C_U^0)] - \lambda\phi < 0 \text{ for all } \phi > \tilde{\phi}_{2NR}.$$

We conclude that $SWRI(1) < SWNRU(0)$.

Finally, when $\phi > \tilde{\phi}_{2R}$, agents do not take the test and do not undertake prevention effort in the dynamic model with and without reclassification. The ex ante utility of agents without reclassification is defined as follows :

$$SWNRU(0) = (1 + \beta)v(C_U^0).$$

The ex ante utility of agents with reclassification is given by :

$$SWRU(0) = (1 + \beta)v(C_U^0) - \beta p_U^0(v(C_U^0) - v(C_h)).$$

We show that $SWRU(0) - SWNRU(0) < 0$.

$$SWRU(0) - SWNRU(0) = -\beta \times p_U^0(v(C_U^0) - v(C_h)).$$

We conclude that $SWRU(0) < SWNRU(0)$ since $v(C_U^0) > v(C_h)$.

Table 2.4 summarizes the ex ante utility of agents according to the cost of effort in the two models with and without reclassification, when the private value of the test can be positive or negative in both of them.

Social welfare in lines	0 to $\tilde{\phi}_{1R}$	$\tilde{\phi}_{1R}$ to $\tilde{\phi}_{1NR}$	$\tilde{\phi}_{1NR}$ to $\tilde{\phi}_{2NR}$	$\tilde{\phi}_{2NR}$ to $\tilde{\phi}_{2R}$	$\phi > \tilde{\phi}_{2R}$
Without reclassification	SWNRU(1)		SWNRI(1)	SWNRU(0)	
With reclassification	SWRU(1)	SWRI(1)			SWRU(0)

Table 2.4: The ex ante utility of agents according to the cost of effort with and without reclassification, when the private value of the test can be positive or negative.

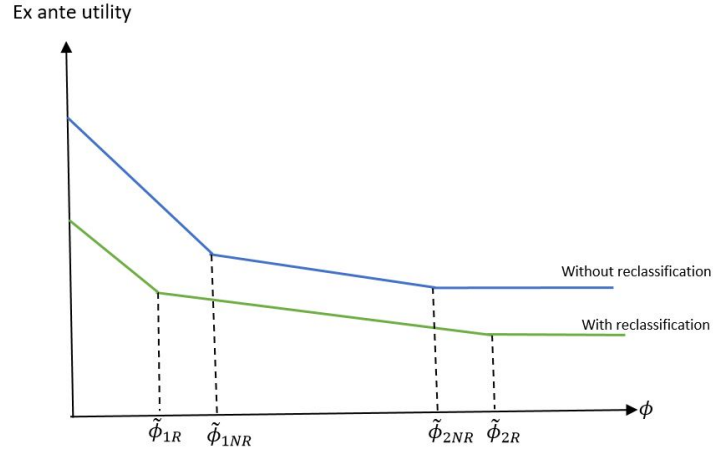


Figure 2.15: The ex ante utility as a function of ϕ with reclassification and without reclassification when the private value of the test can be positive or negative in the two models

As another numerical example, figure 2.15 depicts the ex ante utility of agents with and without reclassification according to ϕ , when the private value of the test can be positive or negative in the two models, and where ϕ_H is higher than ϕ_U and $(1 + \beta)\phi_{\max}$. We take the same values of the parameters of figure 2.14 except $\beta = 0.0200$. For this numerical example, $\tilde{\phi}_{1NR} = 0.00038$, $\tilde{\phi}_{2NR} = 0.38287$, $\tilde{\phi}_{1R} = 0.00020$ and $\tilde{\phi}_{2R} = 0.38984$. The vertical distance between the two curves in figure 2.15 measures the welfare loss resulting from reclassification.

2.6 Conclusion

The purpose of this paper was to analyze the impact of reclassification on the incentive of individuals to perform genetic testing as well as on social welfare in the

disclosure duty regulation. We also analyzed the effect of time preferences on individuals' decision regarding genetic testing and prevention effort to reduce the probability of occurrence of the loss. The utility of the test is to allow individuals to undertake prevention action according to their risk and prevention efficiency. The drawback of the test is that its results are used by insurers to price their insurance policies, so that agents face discrimination risk. In addition, the introduction of the second period allows to take account for the premium variation in the second period. We first show that the incentive of individuals to acquire genetic information differs in the disclosure duty whether individuals value or not their second period and are reclassified or not. In particular, the incentive of agents to take genetic testing with reclassification is higher than without reclassification when the cost of effort is such that agents take the same prevention decision in the two models and when the intensity of reclassification is high. In addition, we show that the social welfare without reclassification is higher than with reclassification.

This analysis makes it possible to understand agents decision if the genetic test was made after signing the first period insurance contract. For example, if we assume that insurers would offer a unique insurance contract in the first period whose premium reflects the average risk of the population. In this context, undertaking a genetic test would allow agents to adjust their prevention effort, when there is effective prevention, according to their type without affecting the first period market equilibrium. The decision of agents to perform the test will then impact the second period market equilibrium. If we consider that insurers will use the information observed at the end of the first period and the result of the genetic test when it is revealed, the incentive of agents to take the test would be different.

Furthermore, our model for assessing the incentives of agents to take genetic test in a

two periods framework can be adapted to a model where agents value differently the second period. Therefore, individuals would take different decisions regarding the test according to their time preferences. Finally, it would be interesting to analyze the effect of reclassification on prevention and testing decision when the amount of the loss is uncertain and when agents can undertake secondary prevention actions. These issues are left for future research.

Chapter 3

Implementation of personalized
medicine in a context of moral
hazard and uncertainty about
treatment efficacy

3.1 Introduction

The completion of the human genome project in 2003 to identify the whole human gene sequence and the potential application of predictive genetic information in medical decisions have raised much interest and are promising in terms of improving patients' health. One of the main applications of predictive genetic information in medical decision-making is the ability to identify patients who may be more responsive and/or exposed to the side effects of certain drugs before they are administered. This new medical approach is referred to as personalized medicine (hereafter PM), which uses a biomarker for selecting optimal therapies based on a patient's genetic information. It requires new innovative treatments which may be very expensive and may expose patients to serious side effects in the absence of genetic testing.

Oncology is one main field of application of PM. For example, there are diagnostic tests based on genetics (Oncotype DX and MammaPrint) that distinguish women with breast cancer in terms of risks categories for which there are specific treatments. Breast-cancer sufferers with an overexpression of HER2 may receive chemotherapy plus herceptin (trastuzumab) instead of chemotherapy alone, thus reducing the recurrence tumors (Hornberger et al. (2005)). There are also several drugs available to treat colorectal cancer whose effectiveness differs among patients and depends on the genetic mutation involved in the development of the disease (Lièvre et al. (2006)).

A growing number of drugs are now labelled with pharmacogenetic information. The U.S. Food and Drug Administration (FDA) listed more than 200 FDA-approved drugs with pharmacogenomic information in their labelling in many therapeutic areas. However, pharmacogenetic tests are not yet widely available and used in daily practice. For example, the National Institutes of Health (in their Genetic Testing

Registry) identify 45 pharmacogenetic tests for which insurance coverage is available and included in medical practices. In the US and France, about 30 pharmacogenetic tests are available and used to choose the optimal treatment for patients.

From a health benefit perspective, the implementation of PM will have a positive impact. One of the benefits for the pharmaceutical industry is to induce more efficient clinical trials. Then pharmaceutical companies will have to develop new treatments associated with genetic tests to identify the more responsive patients. As a result, market opportunities will be smaller. Moreover, when current pricing mechanisms are inflexible, the price of a drug on the market cannot be adjusted according to the value created by PM. Therefore, when a drug enters or is already on the market, a firm has no interest in introducing a genetic test to stratify patients.¹ Indeed, market segmentation could have a negative impact on the profit of drug producers, particularly because of economies of scale. As a result, firms may want to set higher prices to maintain their level of profit or because of scale of production problems.

From the health authority perspective, the implementation of PM will allow a better allocation of resources by reducing medical expenses related to side effects and ineffective drugs and providing better care for patients. However, it will imply higher prices for new treatments to guarantee a profit for pharmaceutical firms. Thus, depending on the distribution of patients in the population, it may be too costly for the health authority to implement PM.

The literature on PM has focused on different topics. A lot of papers consider cost benefit analysis (See Hatz et al. (2014) and included references). That research estimates the incremental costs and benefits of each particular treatment using cost

¹See Scott Morton and Seabright (2013) for a study on the incentives for pharmaceutical firms to invest in biomarkers that identify most responsive patients.

per QALY (quality-adjusted life year) to measure health benefits.² More general papers, often in medical journals, consider the challenges of PM in terms of industrial organization, pricing, reimbursement strategy, and drug quality (Danzon and Towse (2002); Faulkner et al.(2012); Trusheim and Berndt (2012); Miller et al. (2011); Goetz et al. (2018); Hendricks-Sturup and Lu (2019); Knowles et al. (2017)). Recently, the theoretical literature has investigated the economic implications of the development of PM. Antoñanzas et al. (2015) analyze the decision to implement PM in a context where the tests imperfectly identify patients while Antoñanzas et al. (2016) analyze the drug pricing policy of a firm in a context of PM when prevalence rates of disease conditions may vary among hospitals and are private information. Antoñanzas et al. (2018) characterize the optimal outcome-based payment policy that a health authority should offer a pharmaceutical firm when the firm can choose to invest or not in a post-approval diagnostic test. Our paper contributes to the literature by analyzing the decision of a health authority to implement PM with high effectiveness in a context of both non observability of the firm's effort and variability as to health benefits. We consider that the health authority has three possibilities. It may either recommend applying the same treatment (a standard or a new treatment) to the whole population or recommend implementing PM, i.e., use genetic information to apply the most suitable treatment to each patient. Our results show that the unobservability of effort impacts the contract offered and the decision of the payer when the standard treatment is preferred without genetic information or when the new treatment is preferred without genetic information and when the price of the new treatment is below a threshold. In these cases, PM will be less often implemented when effort is not observable than when it is observable. When the

²Some papers highlight their limits and methodological issues, particularly those performing cost benefit analysis (Postma et al. (2011); Annemans et al. (2013)).

new treatment is preferred without genetic information and when the price of the new treatment exceeds the threshold, the unobservability of effort does not impact the implementation of PM.

The remainder of this paper is organized as follows. Before developing our model, we consider the general problem of incentives to develop a treatment and/or a companion test in Section 3.2. Section 3.3 describes the model, i.e., the different treatments and corresponding expected health benefits for the population. In Section 3.4, we determine the optimal reimbursement contracts when effort is either observable or not. Section 3.5 establishes the conditions under which PM will be implemented by the health authority in both cases. Some conclusions are drawn in the final section.

3.2 Developing treatments and companion tests

The implementation of PM must improve the resource allocation by reducing medical expenses related to side effects and ineffective drugs and provide better care for patients. However, following Garrison and Austin (2007), the distribution of the economic value created by this implementation depends on many factors among which - the way the companion genetic test and the medical treatment (medicine) are priced and reimbursed - whether intellectual property exists on both products or only on treatment, and - the timing of the launch of the genetic test innovation. The development cost of a companion diagnostic may be high. Many new pharmacogenomics-genetic diagnostics are more costly than historical and routine tests, often ranging from the low hundreds to the thousands for a few emerging multimer tests (e.g., OncotypeDX, AlloMap, ChemoFX, and MammaPrint).³ These potentially high costs in R&D investment raise the question of the incentives for

³Cf. Faulkner et al. (2012).

firms to either develop a treatment, a diagnostic test or a combination of both. Several scenarios are possible and result in different appropriation of the economic value by the stakeholders. For treatments and diagnostic tests, prices may be cost or value-based. Historically, in a lot of countries, reimbursement of treatments have been cost or value-based while cost-based for tests. The diagnostic test may be developed ex ante or ex post. Uncertainties concerning these scenarios and regarding clinical and economic evidence requirements require test and treatment manufacturers to consider research methods and commercial implications of biomarkers at every stage of the pharmaceutical life cycle. Moreover, some test value may be difficult to assess when the test is not associated to a treatment.

From an industrial organization point of view, the development of PM relates to the case of complementary innovations.⁴ As Cournot (1838) showed, a single monopolist of several complements sets a lower price than several monopolists controlling separately each one of the goods. The intuition is that a single firm internalizes the complementarity by considering the positive effect of a decrease of one price on the demand of the other products. Moreover, the joined pricing of complements benefits to both consumers and firms. Thus, when it enhances the value of treatment for patients, developing drug and diagnostic as a link product is an optimal strategy from an industrial organization point of view. The codevelopment of companion diagnostics and therapeutic products is also recommended by health authorities for efficacy and evaluation purposes.⁵ The FDA seeks to facilitate innovations in PM by providing sponsors with a set of principles that may be helpful for effective codevelopment and in fulfilling FDA's applicable regulatory requirements.⁶

⁴See for example Belleflamme and Peitz (2015) for developments on complementary innovations in Industrial Organization.

⁵Cf. e.g. Jørgensen (2019) who recalls that the FDA has, for years, recognized that the codevelopment of companion diagnostics and therapeutic products is critical to the advancement of PM and that both products have to be available at the same time.

⁶See Principles for Codevelopment of an In Vitro Companion Diagnostic Device with a Thera-

When there is no integration, the coordination of the decisions in order to protect innovation may take several forms, such as the formation of patent pool or of cross-licencing. However, this form of coordination is quite complex and may be unstable due to the existence of some asymmetries among the stakeholders (pharmaceutical industry and pharmacogenomics-genetics diagnostics manufacturers).

In the health sector, as the products are of great social utility, this intellectual protection is completed by a price regulation. According to Danzon and Towse (2002), drug price inflexibility gives manufacturers little commercial incentives to invest in diagnostic testing. Therefore, innovative medicine and associated diagnostic device imply a global development and financing. As argued by Garrison and Towse (2017), pricing and reimbursement should be considered globally, value-based, and flexible in order to create stronger incentives for the development of PM. Our model contributes to the understanding of these incentives.

3.3 The model

We consider a therapeutic market for prescription drugs in which there are two firms: firm S produces a standard treatment, S , and firm N has developed a new treatment, that can be applied with or without the companion genetic test to identify patients for whom it is suitable. To simplify, we consider a cost equal to zero for the companion test. The implementation of PM allows each patient to receive the treatment most likely to provide the greatest health benefit. We analyze the decision of a health authority to implement PM with payments that ensure that firm N has an incentive to use genetic information (i.e., to use a companion test) and produce with high effectiveness. One of the central assumptions of previous

peutic Product. Draft Guidance for Industry and Food and Drug Administration Staff. Document issued on: July 15, 2016.

theoretical studies of PM is that a patient eligible for treatment will enjoy for certain a constant health benefit when this treatment is administered (Antoñanzas et al. (2015) and (2016)). However, even though a patient eligible for a new drug can enjoy greater expected health benefits than with the standard treatment, there is still intra individual variability (Towse and Garisson (2013), O'Donnell (2013), Faulkner et al. (2012), Antoñanzas et al. (2018)). Our analysis considers this variability by assuming that the benefit of any treatment is a random variable, and that the probability of a patient deriving great health benefits depends on the suitability and the effectiveness of the drug.

Departing from Antoñanzas et al. (2018), we assume that this probability (which represents drug effectiveness or quality) can be influenced by the firm's level of effort to improve it. As usual in the incentive theory literature, this effort can be understood as a quality investment, prior to bringing the treatment to the market, to improve treatment efficacy. As argued by Lam (2003), if randomized controlled trials are the "gold standard" to demonstrate the clinical utility of a drug, in the context of PM, the types of study design and/or the quality of study data for evidence of clinical validity and utility have to be rethought. As a companion diagnostic test is intended for use with a drug to produce the optimal efficacy and safety, this makes it difficult to distinguish the clinical utility of the test that is different from that of the drug or the drug-test combination. In contrast to evidence based practice, the emphasis and value of pharmacogenomics are more geared towards incremental advantages in efficacy and safety for the outliers (the poor metabolizers, the ultrarapid metabolizers, the nonresponders, or those susceptible to develop adverse drug reactions) over traditional therapy or standard dosing regimen. Effort can be defined in this paper as the research of these incremental advantages in efficacy and safety in clinical practices. As an example, as elimination of tolbutamide is known to be

50% and 84% slower in carriers of CYP2C9*2 and CYP2C9*3 variants, respectively, than in homozygous carriers of CYP2C9*1 (cf. Kirchheiner et al. (2002)), evaluating tolbutamide efficacy can be done after implementation of these dosage reductions. Given the definition of effort, it seems realistic to assume that this effort cannot be directly observable by the health authority. Thus, moral hazard will be a key issue when designing the optimal reimbursement contract. As a consequence, when deciding to implement PM, the health authority needs to design the optimal policy ensuring that the firm accepts the contract and is induced to exert effort to improve drug quality.

3.3.1 Population and treatments

The health authority's unit cost, for treatment S , is P_S . When producing and selling treatment N without a companion genetic test, firm N will serve the whole market if treatment N is recommended to all patients rather than treatment S . However, the application of a new innovative treatment must be cost effective for the health authority in order to apply it to all patients without genetic information. Like Barros (2011), we suppose that firm N enjoys market exclusivity for its product guaranteed by patent protection. If treatment N is proposed on the market without a companion genetic test, i.e., to serve the whole population, the unit cost of treatment N is P_N , with $P_N > P_S$. We assume that P_N and P_S (in the absence of PM) are exogenous. As considered by Antoñanzas et al. (2018), the manufacturer might have been authorized to sell the drug for such a price, fixed by the health authorities through a negotiation process.

The size of the population is normalized to one. When a patient is administered a treatment, he can get either high monetary equivalent health benefits (\bar{b}) or low benefits (\underline{b}), with $\bar{b} > \underline{b} > 0$. Without loss of generality, we assume that the health

benefit of a patient is costlessly and perfectly observable. We consider two types of patients for a given pathology in the population, differing with respect to their expected health benefits from a particular therapy, due to their gene expression. A proportion λ (with $0 \leq \lambda \leq 1$) of patients is suitable for treatment N whereas a proportion $(1 - \lambda)$ is suitable for treatment S . This means that each part of the population obtains the high benefit \bar{b} with a higher probability when treated with the suitable treatment. This probability represents the treatment effectiveness. It depends both on patient's type and drug quality. When PM is implemented, the genetic test reveals whether a patient belongs to the group λ and, in this case, treatment N is applied only to that group. The proportion of patients with a high benefit \bar{b} depends on the effort e undertaken by firm N to improve the drug's effectiveness, with $e \in \{0, 1\}$. $e = 0$ means that firm N exerts no specific effort while $e = 1$ corresponds to a high effort.

When treatment S is applied to the whole population, the part $(1 - \lambda)$ of the population gets a benefit \bar{b} with a probability β (and \underline{b} with the complementary probability $(1 - \beta)$) whereas the part λ of the population obtains a high benefit \bar{b} with a lower probability γ (and \underline{b} with the complementary probability $(1 - \gamma)$). When treatment N is applied, the part λ of the population obtains \bar{b} (resp. \underline{b}) with a probability $\pi(e)$ (resp. $1 - \pi(e)$), increasing with e . If PM is applied, the part $(1 - \lambda)$ of the population receives treatment S and then obtains \bar{b} with the probability β . However, if treatment N is applied without genetic testing, the part $(1 - \lambda)$ of the population obtains \bar{b} (resp. \underline{b}) with the probability $\theta(e)$ (resp. $1 - \theta(e)$), with $\theta(e) < \pi(e)$ and $\theta(e)$ decreasing with e . Under this assumption, exerting effort (for example, to find the optimal dose or formula) improves the treatment's effectiveness for the group λ , while it is detrimental to the other group.⁷

⁷For example, Tardif et al. (2016) analyze the effects of dalcetrapid, a cardiovascular disease-

Given our assumptions, the probabilities are ranked as follows: $\gamma < \beta$,⁸ $\theta(e) < \pi(e)$, $\gamma < \pi(e)$, and $\theta(e) < \beta$ for all e . Hence, the probability of getting great health benefits is higher when the patient is administered the treatment suitable for his disease than the probability of receiving large health benefits when treatment is not adapted, independently of whether effort is exerted or not. In the following, when effort is exerted, drug effectiveness, $\pi(e)$, is denoted as π_1 , while drug effectiveness is denoted as π_0 when it is not, with $0 < \pi_0 < \pi_1 < 1$. Exerting an effort is costly to the firm. The effort cost is denoted $\psi(e)$. It is such that $\psi(0) = 0$ and $\psi(1) = \psi$. The effort may not be directly observable by the health authority.

We consider that the health authority is risk-neutral. Its objective is to maximize the net expected health benefits of patients. To do so, it has to decide whether to implement PM or not. Without the test, the decision to introduce treatment N and apply it to all patients, when a treatment S is already on the market, depends on the comparison between net expected health benefits of patients. The health authority decides to implement PM if the net expected health benefit of patients is higher than the benefit when a treatment, either N or S , is applied to all patients. When PM is implemented, drug prescription is related to genetic characteristics of patients revealed by the test. We suppose that the companion genetic test is perfect, i.e., there are no false positives or false negatives. This assumption seems realistic insofar as the specificity and the sensitivity of most genetic tests are quite high.

fighting agent, that depend on the genetic characteristics of individuals. They show that the benefits and side effects of this drug depend on whether patients present an AA or a GG genotype. Dalcetrapid seems to be more beneficial for patients with an AA genotype, whereas it may increase the risk of future cardiovascular events in patients with the GG variant of the ADCY9 gene.

⁸Empirical studies (Bertucci et al. (2000) and (2006)) show that the probability of patients belonging to the group λ deriving a large health benefit from the new treatment may be higher than the probability of patients amenable to the standard treatment receiving a large health benefit when they are administered their best treatment.

3.3.2 Expected health benefits

From the health authority perspective, the major concern about the choice of a treatment is the medical issue. However, the health authority must effect a trade-off between the expected health outcomes and the cost of each treatment. According to our previous assumptions, Figures 3.1 and 3.2 depict respectively the health benefits of the patients (with associated probabilities) when treatment S or treatment N are applied to the whole population, i.e., when PM is not implemented. Figure 3.3 depicts the health benefits when PM is applied.

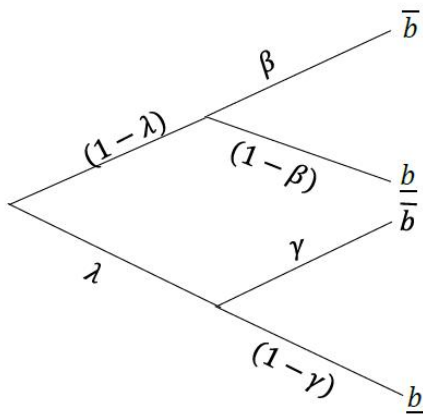


Figure 3.1: Health benefits with treatment S

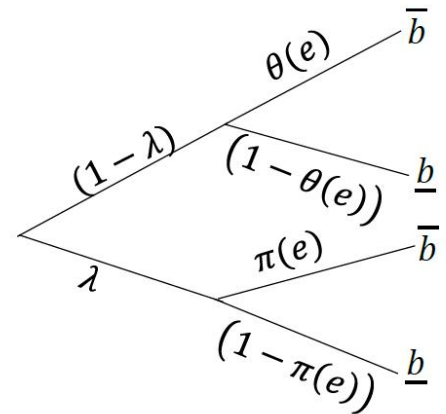


Figure 3.2: Health benefits with treatment N

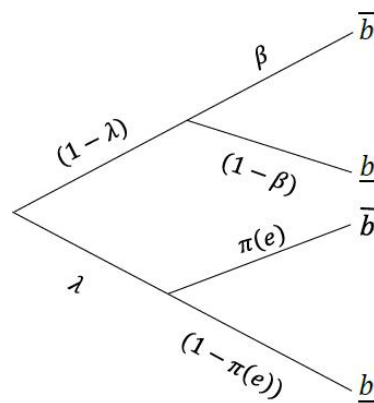


Figure 3.3: Health benefits with PM

When treatment S is applied to all patients, the net expected health benefit is

$$H^S = \lambda(\gamma \bar{b} + (1 - \gamma) \underline{b}) + (1 - \lambda) (\beta \bar{b} + (1 - \beta) \underline{b}) - P_S \quad (3.1)$$

When treatment N is administered to all patients without genetic information, firm N may undertake effort to improve drug quality. However, it has no interest in exerting any effort (since effort is costly and does not influence P_N). Therefore, when $e = 0$, the expected health benefit is given by

$$H^N = \lambda [\pi_0 \bar{b} + (1 - \pi_0) \underline{b}] + (1 - \lambda) [\theta_0 \bar{b} + (1 - \theta_0) \underline{b}] - P_N. \quad (3.2)$$

The treatment N will be administered to all patients if $H^N \geq H^S$. Given (3.1) and (3.2), we obtain

$$\Delta b[\lambda(\pi_0 - \gamma) - (1 - \lambda)(\beta - \theta_0)] \geq P_N - P_S \quad (3.3)$$

with $\Delta b = (\bar{b} - \underline{b})$. According to (3.3), the incremental expected benefit of patients in group λ minus the expected health losses of the other group must be at least equal to the price differential. Then, treatment N is administered to all patients, in the absence of PM, if

$$\lambda \geq \lambda_0 = \frac{(\beta - \theta_0)\Delta b + P_N - P_S}{(\pi_0 + \beta - \gamma - \theta_0)\Delta b}. \quad (3.4)$$

If $P_N - P_S \geq \Delta b(\pi_0 - \gamma)$, i.e., if the price differential between treatments N and S is higher than the incremental expected benefit of patients in group λ , treatment N is never applied. On the contrary, it is applied if $\lambda > \lambda_0$, with $0 < \lambda_0 < 1$.

When PM is implemented, drug administration is based on the test result and a patient's expected health benefit depends on whether he belongs to group λ or not as well as whether effort is exerted or not. Patients belonging to group λ receive treatment N while the others receive treatment S . Thus, when the health authority offers a fixed price $P(e)$, the net expected health benefit is

$$H^{PM/e} = \lambda [\pi(e)\bar{b} + (1 - \pi(e))\underline{b} - P(e)] + (1 - \lambda) [\beta \bar{b} + (1 - \beta) \underline{b} - P_S] \quad (3.5)$$

3.4 Optimal reimbursement contracts to implement PM

In our setting, the health authority has to decide whether to implement PM or not.

When $\lambda \geq \lambda_0$ and when PM is not implemented, treatment N is administered to all patients and firm N gets a profit, $\Pi = P_N$. Firm N 's alternative is to put on the market a companion genetic test to identify patients amenable to treatment N . However, when the price P_N of the drug is inflexible, PM implies fewer patients, and therefore, all else being equal, a reduction in gross sales. Namely, firm N 's profit would become, $\Pi = \lambda P_N - \psi(e) < P_N$. As a result, firm N has no interest in putting treatment N on the market with a companion genetic test since it makes a higher profit when treatment N is applied without genetic information. If the health authority wants to implement PM, it must offer firm N a contract that provides an expected profit at least equal to the firm's reservation profit, i.e., P_N .

When $\lambda < \lambda_0$ and when firm N does not implement PM, it does not serve the market and makes zero profit. Therefore, in order to encourage the development of PM and the production of a high-performance drug, the health authority has to propose a drug reimbursement contract that ensures non negative profits for firm N .

In the following, we derive the optimal reimbursement contract the health authority must propose to firm N in order to encourage the development of PM and the production of a high performance drug, successively when effort is observable or when it is not.

3.4.1 Optimal contract with observable effort

When the health authority observes the level of effort, when it can impose it on firm N , and when it decides to implement PM with a high-performance drug, the optimal payment contract must satisfy firm N 's participation constraint. Let us first

consider the case when treatment N is preferred and then the case when treatment S is preferred.

When $\lambda \geq \lambda_0$, the health authority maximizes the net expected health benefit subject to the firm's participation constraint, which is defined by

$$\lambda P(e) - \psi(e) \geq P_N$$

As (3.5) is decreasing in $P(e)$, the participation constraint is binding and we obtain $P(e) = \frac{P_N + \psi(e)}{\lambda}$. When $e = 1$ is imposed,⁹ $P_1 = \frac{P_N + \psi}{\lambda}$ and when $e = 0$ is imposed, $P_0 = \frac{P_N}{\lambda}$.

Comparing (3.5) when $P(e)$ is replaced with P_1 or P_0 , i.e., when effort is induced or not, we show that it is optimal for the health authority to impose effort if $\lambda \Delta b \Delta \pi \geq \psi$, with $\Delta \pi = \pi_1 - \pi_0$. Thus, the choice of the level of effort results from a trade-off between increasing the expected health benefit for patients by increasing the drug's effectiveness from π_0 to π_1 for the part λ of the population and increasing medical expenses by ψ to improve the drug's effectiveness.

When $\lambda < \lambda_0$, as firm N does not serve the market, its unit price is equal to zero and its participation constraint becomes

$$\lambda P(e) - \psi(e) \geq 0$$

Then, the optimal payments are respectively, $P_1 = \frac{\psi}{\lambda}$, when the health authority decides to impose the high level of effort and, $P_0 = 0$, when $e = 0$ is imposed. As in the previous case, we can show that it is optimal to impose effort on the firm if $\lambda \Delta b \Delta \pi \geq \psi$. Henceforth, we assume that this condition is satisfied.

3.4.2 Optimal contract when effort is unobservable

In this section, we consider that firm N 's effort level to improve drug quality is not observable by the health authority. Depending on the reimbursement contract pro-

⁹In the following, $P(1)$ will be denoted as P_1 and $P(0)$ as P_0 .

posed by the health authority, firm N chooses to exert effort or not. Let us consider that the health authority offers firm N a payment depending of the observed health benefit. When the health authority proposes an ex post reimbursement contract $\{\bar{P}, \underline{P}\}$ based on observable health benefit and wishes to induce the firm to exert effort, the expected net health benefits are

$$\tilde{H}^{PM/1} = \lambda[\pi_1(\bar{b} - \bar{P}) + (1 - \pi_1)(\underline{b} - \underline{P})] + (1 - \lambda)[\beta \bar{b} + (1 - \beta)\underline{b} - P_S].$$

Let us now derive the optimal reimbursement contract in the situation where treatment N and treatment S would be applied respectively without PM.

3.4.2.1 Treatment N is applied without PM

Let us first consider that $\lambda \geq \lambda_0$. When firm N 's effort to improve treatment effectiveness is unobservable and when this firm's reservation profit is equal to P_N , the optimal reimbursement contract to induce effort is the solution of the following program

$$\max_{\bar{P}, \underline{P}} \tilde{H}^{PM/1}$$

$$\text{subject to } \lambda[\pi_1 \bar{P} + (1 - \pi_1) \underline{P}] - \psi \geq P_N \quad (3.6)$$

$$\bar{P}, \underline{P} \geq 0 \quad (3.7)$$

$$\lambda[\pi_1 \bar{P} + (1 - \pi_1) \underline{P}] - \psi \geq \lambda[\pi_0 \bar{P} + (1 - \pi_0) \underline{P}] \quad (3.8)$$

Condition (3.6) is firm N 's participation constraint which ensures that it makes a higher profit with PM than without. Condition (3.7) guarantees that unit prices are positive. Condition (3.8) is the incentive compatibility constraint ensuring that firm N 's expected profit when effort is provided is at least equal to its expected profit when not. The optimal ex post reimbursement contract depends on the value of P_N and is presented in the following proposition:

Proposition 6. *When $\lambda \geq \lambda_0$ and when effort to improve treatment effectiveness is unobservable, the optimal payment contract is such that*

- $\bar{P} = \frac{P_N \Delta\pi + (1-\pi_0)\psi}{\lambda \Delta\pi}$ and $\underline{P} = \frac{P_N \Delta\pi - \pi_0 \psi}{\lambda \Delta\pi}$ if $P_N \geq \frac{\pi_0 \psi}{\Delta\pi}$
- $\bar{P} = \frac{\psi}{\lambda \Delta\pi}$ and $\underline{P} = 0$ if $P_N < \frac{\pi_0 \psi}{\Delta\pi}$

Proof of Proposition 6

Two cases can be considered. In the first case, the incentive compatibility constraint and the participation constraint are binding and condition (7) is satisfied. This occurs when $\underline{P} = \frac{P_N \Delta\pi - \pi_0 \psi}{\lambda \Delta\pi}$ is positive, i.e., when $P_N \geq \frac{\pi_0 \psi}{\Delta\pi}$. In the second case, when $P_N < \frac{\pi_0 \psi}{\Delta\pi}$, these two constraints cannot simultaneously be binding. Then, $\underline{P} = 0$ and $\bar{P} = \max(\frac{P_N + \psi}{\lambda \pi_1}, \frac{\psi}{\lambda \Delta\pi}) = \frac{\psi}{\lambda \Delta\pi}$. \square

The intuition of this result is as follows. The binding incentive compatibility constraint implies that $\bar{P} - \underline{P} = \frac{\psi}{\lambda \Delta\pi}$ and the expected payment with the high level of effort must be at least equal to $\frac{P_N + \psi}{\lambda}$. When $P_N \geq \frac{\pi_0 \psi}{\Delta\pi}$, the required minimum expected payment and the gap between payments to accept the contract and to exert effort can be obtained with positive payments and firm N enjoys no rent. When $P_N < \frac{\pi_0 \psi}{\Delta\pi}$, as the health authority cannot punish the firm in the bad state of nature, the optimal contract implies that the health authority makes no payment in the bad state of nature and a positive payment, $\bar{P} = \frac{\psi}{\lambda \Delta\pi}$, when the health benefit is high. The expected payment, $\lambda \pi_1 \frac{\psi}{\lambda \Delta\pi}$, is equal to the cost of the effort ψ plus $\frac{\pi_0 \psi}{\Delta\pi}$. It guarantees a rent $\frac{\pi_0 \psi}{\Delta\pi} - P_N > 0$ to firm N .

Let us now derive the contract offered by the health authority when effort is not induced. The health authority maximizes the net expected health benefit with respect to $\{\bar{P}, \underline{P}\}$ and subject to both (3.7) and the participation constraint of firm N

$$\lambda [\pi_0 \bar{P} + (1 - \pi_0) \underline{P}] \geq P_N$$

At the optimum, the participation constraint of this firm is binding and the solution is any positive pair of payments (\bar{P}, \underline{P}) which satisfies this participation constraint and gives the firm a higher (or equal) payment when it does not exert effort than when it does. In this case, the health authority can make an identical payment in both states of nature for any value of P_N . The payment satisfying both constraints is P_N/λ .

When $P_N < \frac{\pi_0 \psi}{\Delta \pi}$, it is efficient for the health authority to induce effort whenever

$$\tilde{H}^{PM/1} = \lambda \left[\pi_1 \left(\bar{b} - \frac{\psi}{\lambda \Delta \pi} \right) + (1 - \pi_1) \underline{b} \right] + (1 - \lambda) [\beta \bar{b} + (1 - \beta) \underline{b} - P_S]$$

is higher than

$$H^{PM/0} = \lambda \left[\pi_0 \bar{b} + (1 - \pi_0) \underline{b} - \frac{P_N}{\lambda} \right] + (1 - \lambda) [\beta \bar{b} + (1 - \beta) \underline{b} - P_S].$$

Hence, we can show that $\tilde{H}^{PM/1} \geq H^{PM/0}$ if

$$\lambda \Delta \pi \Delta b \geq \frac{\pi_1 \psi - P_N \Delta \pi}{\Delta \pi} = \psi + \frac{\pi_0 \psi}{\Delta \pi} - P_N.$$

The right-hand member of this inequality is equal to the disutility of effort plus the rent.

When $P_N \geq \frac{\pi_0 \psi}{\Delta \pi}$, it is efficient to induce effort if $\lambda \Delta \pi \Delta b \geq \psi$ and the high effort is induced exactly as in the perfect information case. Thus, the unobservability of effort has no impact on the health authority's decision to stimulate firm N to exert effort to improve drug effectiveness. Otherwise (when $P_N < \frac{\pi_0 \psi}{\Delta \pi}$), firm N obtains a rent and inducing effort is more costly for the health authority. Hence, the latter must make a trade-off between inducing effort and giving up an ex ante rent to the firm.

3.4.2.2 Treatment S is applied without PM

Let us now consider that $\lambda < \lambda_0$, i.e., treatment S is applied without PM and firm N does not serve the market. If the health authority wants to implement PM, it has to

propose a payment contract ensuring that firm N receives a non negative payment. When the health authority wishes to induce effort, the program is the same as in the previous case except that the participation constraint is now defined as

$$\lambda[\pi_1\bar{P} + (1 - \pi_1)\underline{P}] - \psi \geq 0$$

We then have the following proposition:

Proposition 7. *When $\lambda < \lambda_0$ and effort is not observable, the optimal reimbursement contract on drug performance inducing the firm to make an effort to improve drug efficacy is defined as $\bar{P} = \frac{\psi}{\lambda \Delta\pi}$ and $\underline{P} = 0$.*

Proof of Proposition 7

This case corresponds to the previous case with $P_N = 0$. Then P_N is “low” and there is a conflict between the non negativity constraint on payments and the participation constraint. As previously, the optimal contract is defined by $\bar{P} = \frac{\psi}{\lambda \Delta\pi}$ and $\underline{P} = 0$. This contract implies no payment for low health benefit, the incentive compatibility constraint is binding, and the firm enjoys a strictly positive rent, equal to $\frac{\pi_0 \psi}{\Delta\pi}$, as in the previous case. \square

When the health authority does not want to induce effort, condition (3.7) and the following participation constraint

$$\lambda [\pi_0\bar{P} + (1 - \pi_0)\underline{P}] \geq 0 \tag{3.9}$$

matter, as well as the incentive constraint implying that the firm gets a higher (or equal) payment when it does not exert effort than when it does. The optimal contract inducing no effort is determined when condition (3.9) is binding with a payment independent of the health benefit. Then, payments are given by $\bar{P} = \underline{P} = 0$. Firm N receives no payments in both states of nature.

Comparing the net expected health benefits of patients when effort is induced or not reveals that it is efficient to induce effort if $\lambda \Delta\pi \Delta b \geq \frac{\pi_1 \psi}{\Delta\pi} > \psi$. When the firm reservation profit equals zero, the expected cost of inducing effort is always higher when effort is not observable than when it is. Table 3.1 provides a summary of the optimal contracts in the different cases depending on whether effort is observable or not and depending on the treatment applied in the absence of PM.

Table 3.1: Optimal contracts

	Optimal contract when effort is observable	Optimal contract when effort is not observable
$\lambda \geq \lambda_0$ If $P_N \Delta\pi - \pi_0 \psi \geq 0$	$P_1 = \frac{P_N + \psi}{\lambda}$	$(\bar{P} = \frac{P_N \Delta\pi + (1 - \pi_0) \psi}{\lambda \Delta\pi}; \underline{P} = \frac{P_N \Delta\pi - \pi_0 \psi}{\lambda \Delta\pi})$
$\lambda \geq \lambda_0$ If $P_N \Delta\pi - \pi_0 \psi < 0$	$P_1 = \frac{P_N + \psi}{\lambda}$	$(\bar{P} = \frac{\psi}{\lambda \Delta\pi}; \underline{P} = 0)$
$\lambda < \lambda_0$ The standard treatment is preferred ex ante	$P_1 = \frac{\psi}{\lambda}$	$(\bar{P} = \frac{\psi}{\lambda \Delta\pi}; \underline{P} = 0)$

3.5 Health authority's decision

In this section, we analyze the decision of the health authority to implement PM, which depends on the comparison between the net expected health benefit with PM and without PM.

3.5.1 Implementation of PM when effort is observable

Assume that $\lambda \geq \lambda_0$, i.e., treatment N is applied without genetic information. The net expected health benefit when PM is implemented is

$$H^{PM/1} = \lambda [\pi_1 \bar{b} + (1 - \pi_1) \underline{b} - P_1] + (1 - \lambda) [\beta \bar{b} + (1 - \beta) \underline{b} - P_S] \quad (3.10)$$

with $P_1 = \frac{P_N + \psi}{\lambda}$. When PM is not implemented, the net expected health benefit, H^N , is defined by (3.2). Therefore, PM should be implemented if $H^{PM/1} > H^N$, i.e., if

$$\lambda \Delta\pi \Delta b + (1 - \lambda)(\beta - \theta_0) \Delta b \geq \psi + (1 - \lambda) P_S \quad (3.11)$$

According to this condition, expected health gains (derived from effort and health gain of patients most responsive to treatment S) must be higher or equal to the cost of effort plus the cost of treating patients responsive to treatment S . P_N does not play a role in the decision of the health authority, as it is paid in both cases. Thus, only the cost of effort and the cost of administering treatment S to patients eligible for it matter.

Assume now that $\lambda < \lambda_0$, i.e., treatment S is applied without genetic information. The net expected health benefit when PM is implemented is equal to $H^{PM/1}$, with $P_1 = \frac{\psi}{\lambda}$. According to (3.1), when PM is not implemented, the net expected health benefit is equal to H^S . Therefore, PM should be implemented if

$$\lambda[(\pi_1 - \gamma)\Delta b] \geq \psi - \lambda P_S$$

Expected health gains (derived from treating patients belonging to the group λ with the suitable treatment with high effort) must be higher or equal to the cost of effort minus the cost of treating patients responsive to treatment N with treatment S , as treatment N is costless to the health authority. Thus, only the cost of effort and the gain of not administering treatment S to patients not eligible to it matter.

3.5.2 Implementation of PM when effort is unobservable

We now study the impact of the unobservability of effort on the health authority's decision to implement PM and to encourage the production of high performance treatment.

Assume first that $\lambda \geq \lambda_0$. Two cases must be considered

- $P_N \geq \frac{\pi_0 \psi}{\Delta \pi}$. The net health expected benefit with PM is defined by

$$\tilde{H}^{PM/1} = \lambda[\pi_1(\bar{b} - \bar{P}) + (1 - \pi_1)(\underline{b} - \underline{P})] + (1 - \lambda)[\beta\bar{b} + (1 - \beta)\underline{b} - P_S] \quad (3.12)$$

with $\bar{P} = \frac{P_N \Delta \pi + (1 - \pi_0) \psi}{\lambda \Delta \pi}$ and $\underline{P} = \frac{P_N \Delta \pi - \pi_0 \psi}{\lambda \Delta \pi}$. Without PM, the net health expected benefit is H^N . Given (3.2) and (3.12), $\tilde{H}^{PM/1} \geq H^N$ if

$$\Delta b(\lambda \Delta \pi + (1 - \lambda)(\beta - \theta_0)) \geq \psi + (1 - \lambda) P_S \quad (3.13)$$

which is the same condition as (3.11).

- $P_N < \frac{\pi_0 \psi}{\Delta \pi}$. The net expected health benefit with PM is defined by $\tilde{H}^{PM/1}$ with $\bar{P} = \frac{\psi}{\lambda \Delta \pi}$ and $\underline{P} = 0$. Without PM, the net health expected benefit is H^N . So, PM should be implemented if

$$\Delta b(\lambda \Delta \pi + (1 - \lambda)(\beta - \theta_0)) \geq \frac{\pi_1 \psi}{\Delta \pi} - P_N + (1 - \lambda) P_S \quad (3.14)$$

$\frac{\pi_1 \psi}{\Delta \pi} - P_N$ is the expected cost of inducing treatment effectiveness for the part of the population suitable for treatment N . It is equal to the disutility of effort ψ plus the rent $\frac{\pi_0 \psi}{\Delta \pi} - P_N$. $(1 - \lambda) P_S$ is the cost of treatment S .

Assume now that $\lambda < \lambda_0$. Without genetic information, treatment S is applied to the whole population and the net expected health benefit is H^S . The net expected health benefit with PM is defined by $\tilde{H}^{PM/1}$ with $\bar{P} = \frac{\psi}{\lambda \Delta \pi}$ and $\underline{P} = 0$. So, the health authority decides to implement PM and to induce effort if

$$\lambda [(\pi_1 - \gamma) \Delta b] \geq \frac{\pi_1 \psi}{\Delta \pi} - \lambda P_S. \quad (3.15)$$

Our results are summarized in the following proposition:

Proposition 8.

- If $\lambda \geq \lambda_0$ and $P_N \geq \frac{\pi_0 \psi}{\Delta\pi}$, PM is implemented with the highly effective drug whether the effort is observed or not if

$$\lambda \Delta\pi \Delta b + (1 - \lambda)(\beta - \theta_0) \Delta b \geq \psi + (1 - \lambda) P_S \quad (3.16)$$

- If $\lambda \geq \lambda_0$ and $P_N < \frac{\pi_0 \psi}{\Delta\pi}$, then
 - When effort is unobservable, the health authority implements PM and induces effort if $\lambda \Delta\pi \Delta b + (1 - \lambda)(\beta - \theta_0) \Delta b \geq \frac{\pi_1 \psi}{\Delta\pi} - P_N + (1 - \lambda)P_S$.
 - When effort is observable, PM is implemented if

$$\lambda \Delta\pi \Delta b + (1 - \lambda)(\beta - \theta_0) \Delta b \geq \psi + (1 - \lambda) P_S \quad (3.17)$$

- If $\lambda < \lambda_0$.
 - When effort is unobservable, PM is implemented if $\lambda [(\pi_1 - \gamma) \Delta b] \geq \frac{\pi_1 \psi}{\Delta\pi} - \lambda P_S$.
 - When effort is observable, PM is implemented if $\lambda [(\pi_1 - \gamma) \Delta b] \geq \psi - \lambda P_S$.

Proposition 8 calls for some comments. When treatment N is applied without genetic information and when $P_N \geq \frac{\pi_0 \psi}{\Delta\pi}$, the unobservability of effort has no impact on the health authority's decision to implement PM. It follows that the health authority offers a contract and chooses to implement PM as it would do when effort is perfectly observable and can be imposed. Hence, the unobservability of effort does not lead to economic inefficiency when using a payment contract based on ex post drug efficacy.

Corollary 1. *When treatment S , or treatment N when $P_N < \frac{\pi_0 \psi}{\Delta\pi}$, are applied without PM, PM will be implemented less often when effort is unobservable.*

Proof of Corollary 1.

- When $\lambda \geq \lambda_0$ and $P_N < \frac{\pi_0 \psi}{\Delta\pi}$, $\frac{\pi_1 \psi}{\Delta\pi} - P_N + (1 - \lambda)P_S = \psi + \frac{\pi_0 \psi}{\Delta\pi} - P_N + (1 - \lambda)P_S > \psi + (1 - \lambda)P_S$
- When $\lambda < \lambda_0$, $\frac{\pi_1 \psi}{\Delta\pi} - \lambda P_S = \psi + \frac{\pi_0 \psi}{\Delta\pi} - \lambda P_S > \psi - \lambda P_S$. \square

When effort is not observable, our results reveal that the unobservability of effort impacts the contract offered and the decision of the health authority when treatment S is preferred without genetic information or when treatment N is preferred without genetic information with $P_N < \frac{\pi_0 \psi}{\Delta\pi}$. In these cases, PM will be implemented less often when effort is not observable than when it is. When treatment N is preferred without genetic information with $P_N > \frac{\pi_0 \psi}{\Delta\pi}$, the unobservability of effort does not impact the implementation of PM.

3.6 Conclusion

In this paper, we have analyzed the decision of a health authority to implement PM when a firm has the possibility of putting a new treatment on the market with or without a companion genetic test. In particular, we have considered a model where the efficacy of this new innovative treatment varies between and within patients and where the firm can exert an effort, that may not be observable, to improve treatment quality. After characterizing the optimal reimbursement contract to offer to this firm, we have stated the conditions under which the health authority will decide to implement PM when the firm's effort is observable and when it is not observable. Our results reveal that PM will be implemented less often when effort is not observable than when it is, when the standard treatment is preferred without genetic information or when both the new treatment is preferred without genetic

information and the price of this treatment is below a threshold. However, when the new treatment is preferred without genetic information and when the price of the new treatment exceeds the threshold, the unobservability of effort does not impact the health authority's decision.

Our results are obtained under some simplifying assumptions. First, we have focused on the health authority's decisions to implement PM with a highly effective drug when tests classify patients perfectly. Our theoretical framework can also be used to analyze the decision-making process when tests categorize patients imperfectly. Taking this characteristic of genetic tests into account would negatively impact the expected health benefits of patients and so would probably affect the health authority's decision. Other extensions of this paper could consider the case where the marginal cost of producing the test is positive and/or the case where the firm developing the new treatment also produces the standard one.

General Conclusion

The objective of this thesis was to analyze how the different regulations of genetic testing impact the health insurance market, as well as the implications of the development of personalized medicine on the health system. The main lessons learned from this thesis are as follows. According to theoretical predictions, when individuals have homogeneous preferences, they always perform genetic testing in all the regulations, except in the disclosure duty regulation. In this latter, the decision to take the test depends on its cost and utility. Thus, individuals take the test in the disclosure duty regime when the utility of the test is higher than its costs. In practice, many individuals forgo genetic testing for different reasons, even in countries prohibiting its use by insurers. The most common regulation, the strict prohibition, is the one that would be less efficient in terms of social welfare. Thus, by protecting individuals from any possibility of discrimination, the regulator makes a choice impacting negatively the social welfare. The heterogeneity in the selection of a regulation of genetic predisposition testing on the health insurance market illustrates that there is no consensus on the type of trade-off realized by the different countries. We believe that more theoretical studies are needed to bring insights about individual decision regarding genetic testing in non-insurance context.

Despite the arguments in favor of the disclosure duty regulation where insurers are able to discriminate the insureds according to their health risk, we have shown

that the regulator must make a trade-off between the maximization of ex ante social welfare and incentive to undertake prevention action. Indeed, the regulation which forces individual to reveal information to the insurer removes adverse selection problem on the market but suffers from the problem of incentive to take the test and then to undertake optimal prevention action because of genetic discrimination.

We have also demonstrated that the incentive to take a genetic testing in the disclosure duty regulation also depends on the valuation of the second period utility and the intensity of the variability of insurance premium over time. In particular, when an individual values his/her second period, we have shown that the incentive to take a genetic testing is higher with reclassification than without reclassification, especially when the effort cost is such that individuals take the same prevention decision. Reclassification may lead individuals to take genetic testing for which there is no efficient prevention action allowing a better management of disease risk. Moreover, the social welfare is lower when individuals are reclassified and discriminated according to genetic test result.

As a policy recommendation for genetic predisposition testing, our suggestion for governments aiming at protecting “bad genetic risks” from discrimination is to not impose strict regulation of genetic information. Health authorities should instead opt for a disclosure duty. They also must provide a specific public program, or expand the existing ones, offering insurance coverage for the high risks and premium variation. Another solution that policy makers can implement is to create the conditions for the missing market for “genetic insurance”, as proposed by Tabarrok (1994) and for premium variation, as suggested by Cochrane (1995).

In the last chapter, we have studied the incentives issues for developing person-

alized medicine with highly effective drug. Our analysis shows that such incentives could be implemented with a payment system based on ex post performance of drug. We believe that health authority could use such instrument to encourage the production of highly effective differentiated treatment. Health authorities in most OECD countries already use outcome-based reimbursement policy to encourage pharmaceutical firms to undertake research and development activities for developing new innovative treatments. The contribution of this thesis is to show how some parameters affect the structure of outcome-based reimbursement contract as well as the decision of the health authority to encourage the development of personalized medicine with highly effective treatments. In the health care system, the decision to adopt the strategy of personalized medicine depends on health care providers which may have or not incentive to do so. Even when personalized medicine technology is available, physicians may under-use it. As a result, the issue of effective adoption of personalized medicine also depends on how physicians' payment schemes affect their incentives to use personalized medicine.

This thesis is mainly based on theoretical economic modeling in order to analyze the implications of the different regulations on testing and prevention decisions as well as on the efficiency of the insurance market and the incentives issues for developing personalized medicine. Future works must mobilize empirical tools to test theoretical predictions. More empirical evidences (on real or experimental data) are needed to shed light on policy makers, in line with the works of Bardey et al. (2019), Mimra et al. (2019) and Peyron et al. (2018). Also, it would be interesting to empirically investigate how the decision of physicians to use personalized medicine is affected when they face incentives to provide a particular treatment, in the line with the works of Bardey et al. (2018) and Howard et al. (2017).

Appendix

1 Proof of Result 3

- (i) Straightforward since $\frac{\partial \psi_{NR}^{1,1}(\phi, \Delta)}{\partial \phi} = (1 - \lambda) > 0$, with $0 < \lambda < 1$.
- (ii) This proof is the same as that of BDD. We have simply adapted it to the dynamic case.

When $\Delta = 0$ (that is $p_H^0 = p_H^1$), we have $(1 + \beta)\phi_{min} = (1 + \beta)\phi_{max} = 0$. We know, from Result 2, that

$$\psi_{NR}^{0,0}(0, 0) = (1 + \beta) [\lambda v(C_H^0) + (1 - \lambda)v(C_L) - v(C_U^0)] < 0.$$

We also know that $\frac{\partial((1+\beta)\phi_{min}(\Delta))}{\partial \Delta} = (1 + \beta)\lambda dv'(C_U^1) > 0$ implying that

$$(1 + \beta)\phi_{min}(\Delta) > 0 \text{ if } \Delta > 0.$$

We have $\frac{\partial \psi_{NR}^{1,1}(\phi, \Delta)}{\partial \phi} = (1 - \lambda) > 0$ and $\frac{\partial \psi_{NR}^{1,1}(\phi, \Delta)}{\partial \Delta} = (1 + \beta)d\lambda(v'(C_H^1) - v'(C_U^1)) > 0$.

We then have:

$$\begin{aligned} \frac{d\psi_{NR}^{1,1}((1+\beta)\phi_{min}(\Delta), \Delta)}{d\Delta} &= \frac{\partial \psi_{NR}^{1,1}((1 + \beta)\phi_{min}(\Delta), \Delta)}{\partial \phi} \frac{\partial((1 + \beta)\phi_{min}(\Delta))}{\partial \Delta} \\ &+ \frac{\partial \psi_{NR}^{1,1}((1 + \beta)\phi_{min}(\Delta), \Delta)}{\partial \Delta} > 0. \end{aligned}$$

In addition, we have $\psi_{NR}^{1,1}(0, \bar{\Delta}) = (1 + \beta)[\lambda v(C_L) + (1 - \lambda)v(C_L) - v(C_L)] = 0$ and $(1 + \beta)\phi_{min} > 0$ when $\Delta = \bar{\Delta}$. Therefore, we have $\psi_{NR}^{1,1}((1 + \beta)\phi_{min}, \bar{\Delta}) > 0$.

The fact that $\psi_{NR}^{1,1}(\phi, \Delta)$ is continuous and strictly increasing in Δ for any ϕ implies, by the intermediate value theorem, that there exists a unique $0 < \Delta < \bar{\Delta}$, denoted by $\tilde{\Delta}$, such that $\psi_{NR}^{1,1}((1 + \beta)\phi_{min}(\tilde{\Delta}), \tilde{\Delta}) = 0$. Note that $\tilde{\Delta}$ is the same in the dynamic model as that of the static model.

- (iii) If $\Delta > \tilde{\Delta}$, we know that $\psi_{NR}^{1,1}((1 + \beta)\phi_{min}(\Delta), \Delta) > 0$. We also know that $\psi_{NR}^{1,1}(0, \Delta) < 0$. Given that $\frac{\partial \psi_{NR}^{1,1}(\phi, \Delta)}{\partial \phi} = (1 - \lambda) > 0$, it implies, by the intermediate value theorem, that there exists a unique value of ϕ , denoted by $\tilde{\phi}_{1NR}(\Delta)$, such that $\psi_{NR}^{1,1}(\tilde{\phi}_{1NR}(\Delta), \Delta) = 0$ and $0 < \tilde{\phi}_{1NR}(\Delta) < (1 + \beta)\phi_{min}(\Delta)$.
- (iv) Let us denote by \hat{H}_1 the expression $\lambda v(C_H^1) + (1 - \lambda)v(C_L) - v(C_U^1)$ inside the brackets of (2.3), representing the Hirschleifer effect. We know from Jensen's inequality that \hat{H}_1 is negative. Therefore, $\psi_{NR}^{1,1}(\phi, \Delta)$ is decreasing in β .
- (v) We know from item *i*) that the private value of the test in a dynamic model without reclassification is increasing with ϕ for all $\phi \leq (1 + \beta)\phi_{min}(\Delta)$, while it is increasing in a static model for all $\phi \leq \phi_{min}(\Delta)$. If $\phi = 0$, $\psi_{NR}^{1,1}(0, \Delta) < 0$ and is smaller than the private value of the test in a static model since $\psi_{NR}^{1,1}(\phi, \Delta)$ is decreasing in β .

Then we have for all $\phi \leq \phi_{min}(\Delta)$,

$$\psi_{NR}^{1,1}(\phi, \Delta) < \lambda v(C_H^1) + (1 - \lambda)v(C_L) - v(C_U^1) + (1 - \lambda)\phi = \psi_S^{1,1}(\phi, \Delta),$$

with $\psi_S^{1,1}(\phi, \Delta)$ the private value of the test in a static model.

BDD have shown that the max of the curve in the static model, denoted by $\psi_S^{1,1}(\phi_{min}(\Delta), \Delta)$, is positive if $\Delta > \tilde{\Delta}$.

As $\psi_{NR}^{1,1}((1 + \beta)\phi_{min}(\Delta), \Delta) = (1 + \beta)\psi_S^{1,1}(\phi_{min}(\Delta), \Delta)$, the maximum of the curve with reclassification, $\psi_{NR}^{1,1}((1 + \beta)\phi_{min}(\Delta), \Delta)$ is higher than the maxi-

mum of the curve of the static model. Thus, the maximum of the curve with reclassification is also positive.

For all $\phi_{min} < \phi \leq (1 + \beta)\phi_{min}(\Delta)$, $\psi_{NR}^{1,1}(\phi, \Delta)$ is increasing in ϕ , while $\psi_S^{1,0}(\phi, \Delta)$ is decreasing with ϕ . We also know that $\psi_S^{0,0}(\phi, \Delta) < 0$ and is constant for all $\phi \geq \phi_{max}(\Delta)$.

Note that $(1 + \beta)\phi_{min}(\Delta)$ can be higher or smaller than $\phi_{max}(\Delta)$.

$(1 + \beta)\phi_{min}(\Delta) > \phi_{max}(\Delta)$ provided that $\beta > \frac{\phi_{max}(\Delta) - \phi_{min}(\Delta)}{\phi_{min}(\Delta)}$.

Therefore, $\psi_{NR}^{1,1}(\phi, \Delta)$ and $\psi_S^{1,0}(\phi, \Delta)$ cross each other between $\phi_{min}(\Delta)$ and $\min(\phi_{max}(\Delta), (1 + \beta)\phi_{min}(\Delta))$ or $\psi_{NR}^{1,1}(\phi, \Delta)$ and $\psi_S^{0,0}(\phi, \Delta)$ cross each other between $\phi_{max}(\Delta)$ and $(1 + \beta)\phi_{min}(\Delta)$.

Let us denote $\phi_{S=D}^1(\Delta)$ the value of ϕ such that

$$\psi_{NR}^{1,1}(\phi_{S=D}^1(\Delta), \Delta) = \psi_S^{1,0}(\phi_{S=D}^1(\Delta), \Delta),$$

with $\phi_{min}(\Delta) < \phi_{S=D}^1(\Delta) < \min(\phi_{max}(\Delta), (1 + \beta)\phi_{min}(\Delta))$.

We then have $\psi_{NR}^{1,1}(\phi, \Delta) > \psi_S^{1,0}(\phi, \Delta)$ for all $\phi_{S=D}^1(\Delta) < \phi \leq (1 + \beta)\phi_{min}(\Delta)$, while $\psi_{NR}^{1,1}(\phi, \Delta)$ is lower than the private value of the test in a static model for all $\phi < \phi_{S=D}^1(\Delta)$. $\phi_{S=D}^1(\Delta)$ is defined as follows :

When $\phi_{min}(\Delta) < \phi_{S=D}^1(\Delta) < \min(\phi_{max}(\Delta), (1 + \beta)\phi_{min}(\Delta))$,

$$\phi_{S=D}^1(\Delta) = v(C_U^1) - v(C_U^0) + \beta[v(C_U^1) - \lambda v(C_H^1) - (1 - \lambda)v(C_L)].$$

However, if $\phi_{max}(\Delta) < \phi_{S=D}^1(\Delta) < (1 + \beta)\phi_{min}(\Delta)$, then

$$\phi_{S=D}^1(\Delta) = \frac{\phi_{min} - \lambda\phi_{max} + \beta[v(C_U^1) - \lambda v(C_H^1) - (1 - \lambda)v(C_L)]}{1 - \lambda}.$$

(vi) Straightforward given items *ii)* and *iii)* and since $\frac{\partial \psi_{NR}^{1,1}(\phi, \Delta)}{\partial \phi} > 0$.

(vii) We have $\frac{\partial \psi_{NR}^{1,1}(\phi, \Delta)}{\partial p_L} = d(1 - \lambda)(1 + \beta)(v'(C_U^1) - v'(C_L)) > 0$.

We have $\frac{\partial \psi_{NR}^{1,1}(\phi, \Delta)}{\partial p_H} = -d\lambda(1 + \beta)(v'(C_H^1) - v'(C_U^1)) < 0$.

□

2 Proof of Result 4

(i) Straightforward since

$$\frac{\partial \psi_{NR}^{1,0}(\phi, \Delta)}{\partial \phi} = -\lambda < 0.$$

(ii) We know from item *ii)* of Result 3 that $\psi_{NR}^{1,1}((1 + \beta)\phi_{min}(\Delta), \Delta)$ is strictly positive for all $\Delta > \tilde{\Delta}$, and from Result 2 that $\psi_{NR}^{0,0}((1 + \beta)\phi_{max}(\Delta), \Delta) < 0$ for all Δ . The fact that $\frac{\partial \psi_{NR}^{1,0}(\phi, \Delta)}{\partial \phi} = -\lambda < 0$ then implies, by the intermediate value theorem, that there exists a unique value of ϕ , denoted by $\tilde{\phi}_{2NR}(\Delta)$, such that $(1 + \beta)\phi_{min}(\Delta) < \tilde{\phi}_{2NR}(\Delta) < (1 + \beta)\phi_{max}(\Delta)$, $\psi_{NR}^{1,0}(\tilde{\phi}_{2NR}(\Delta), \Delta) = 0$, and

$$\tilde{\phi}_{2NR}(\Delta) = \frac{(1 + \beta)[\lambda v(C_H^1) + (1 - \lambda)v(C_L) - v(C_U^0)]}{\lambda}.$$

(iii) $\psi_{NR}^{1,0}(\phi, \Delta)$ is increasing in β if $\lambda v(C_H^1) + (1 - \lambda)v(C_L) - v(C_U^0) > 0$. We know from part *ii)* of Result 4 that

$$\psi_{NR}^{1,0}(\phi, \Delta) = (1 + \beta)[\lambda v(C_H^1) + (1 - \lambda)v(C_L) - v(C_U^0)] - \lambda\phi > 0$$

for all $0 < (1 + \beta)\phi_{min} < \phi < \tilde{\phi}_{2NR}(\Delta)$. We then have

$$(1 + \beta)[\lambda v(C_H^1) + (1 - \lambda)v(C_L) - v(C_U^0)] > \lambda\phi > 0.$$

We conclude that

$$\lambda v(C_H^1) + (1 - \lambda)v(C_L) - v(C_U^0) > 0 \text{ for all } \Delta > \tilde{\Delta}.$$

As a result, $\psi_{NR}^{1,0}(\phi, \Delta)$ is increasing in β .

(iv) We know from Result 3 that $\psi_{NR}^{1,1}((1 + \beta)\phi_{min}, \Delta) > 0$ for all $\Delta > \tilde{\Delta}$, and $\psi_{NR}^{1,1}((1 + \beta)\phi_{min}, \Delta)$ is higher in a dynamic model without reclassification than the private value of the test in a static model.

We also know from Result 4 that $\psi_{NR}^{1,0}(\phi, \Delta)$ is decreasing in ϕ , while the private value of the test in a static model ($\psi_S^{0,0}(\phi, \Delta)$) is negative and independent of ϕ for all $\phi \geq \max(\phi_{max}, (1 + \beta)\phi_{min})$.

In addition, we have shown in Result 2 that if $\phi \geq (1 + \beta)\phi_{max}$, then $\psi_{NR}^{0,0}(\phi, \Delta)$ is negative and lower than the private value of the test in a static model. Therefore, $\psi_{NR}^{1,0}(\phi, \Delta)$ and $\psi_S^{0,0}(\phi, \Delta)$ cross each other between $\max(\phi_{max}, (1 + \beta)\phi_{min})$ and $(1 + \beta)\phi_{max}$.

Let us denote $\phi_{S=D}^2(\Delta)$, the value of ϕ such that the private value of the test is equivalent in a static model and a dynamic model without reclassification, with $\max(\phi_{max}, (1 + \beta)\phi_{min}) < \phi_{S=D}^2(\Delta) < (1 + \beta)\phi_{max}$.

When $\Delta > \tilde{\Delta}$, we then conclude that $\psi_{NR}^{1,0}(\phi, \Delta)$ is higher (resp., lower) in the dynamic model without reclassification than a static model for all $(1 + \beta)\phi_{min} < \phi < \phi_{S=D}^2$ (resp., $\phi > \phi_{S=D}^2$).

$\phi_{S=D}^2$ is defined as follows :

$$\phi_{S=D}^2 = \frac{\lambda(v(C_H^1) - v(C_H^0)) + \beta \times [\lambda v(C_H^1) + (1 - \lambda)v(C_L) - v(C_U^0)]}{\lambda}.$$

(v) Straightforward given item *iv*) and since $\frac{\partial \psi_{NR}^{1,0}(\phi, \Delta)}{\partial \phi} = -\lambda < 0$.

(vi) We have $\frac{\partial \psi_{NR}^{1,0}(\phi, \Delta)}{\partial p_L} = d(1 - \lambda)(1 + \beta)(v'(C_U^0) - v'(C_L)) > 0$.

We have $\frac{\partial \psi_{NR}^{1,0}(\phi, \Delta)}{\partial p_H} = -d\lambda(1 + \beta)v'(C_H^1) < 0$.

We have $\frac{\partial \psi_{NR}^{1,0}(\phi, \Delta)}{\partial p_H^0} = d\lambda(1 + \beta)v'(C_U^0) > 0$.

□

3 Proof of Result 7

(i) We have

$$\frac{\partial x_H}{\partial p_h} = d(p_H^0 - p_H^1)v'(C_h) > 0 \text{ since } p_H^0 > p_H^1 \text{ and } v'(C_h) > 0.$$

$$\frac{\partial x_U}{\partial p_h} = d\lambda(p_H^0 - p_H^1)v'(C_h) > 0.$$

Thus, $\frac{\partial x_H}{\partial p_h} > \frac{\partial x_U}{\partial p_h}$ since $\lambda < 1$.

(ii) The proof involves three steps.

(a) We first show that $x_U - x_H > 0$ at $p_h = p_H^0$ when $\lambda = 0$.

When $\lambda = 0$, let us evaluate $x_U - x_H$ at $p_h = p_H^0$. We have obviously $x_U = 0$, $x_H = p_H^1(v(C_H^0) - v(C_H^1)) < 0$. So, we have $x_U - x_H = p_H^1(v(C_H^1) - v(C_H^0)) > 0$.

(b) Secondly, when $\lambda = 1$, we have obviously $x_U - x_H = 0$ (all agents are of type H).

(c) Then, we show that $x_U - x_H$ is concave in λ .

We have

$$\begin{aligned} \frac{\partial(x_U - x_H)}{\partial \lambda} &= (p_H^1 - p_H^0)v(C_h) - (p_H^1 - p_L)v(C_U^1) + (p_H^0 - p_L)v(C_U^0) \\ &\quad + d * p_U^1(p_H^1 - p_L)v'(C_U^1) - d * p_U^0(p_H^0 - p_L)v'(C_U^0) \end{aligned}$$

and

$$\begin{aligned} \frac{\partial^2(x_U - x_H)}{\partial \lambda^2} &= d[(p_H^1 - p_L)^2(2v'(C_U^1) - dp_U^1 v''(C_U^1)) \\ &\quad - (p_H^0 - p_L)^2(2v'(C_U^0) - dp_U^0 v''(C_U^0))] \end{aligned}$$

Since $v'''(\cdot) > 0$, we have

$$\frac{\partial^2(x_U - x_H)}{\partial \lambda^2} < 0.$$

So, $\forall p_h$, the function $x_U - x_H$ is concave in λ and vanishes at $\lambda = 1$. When $p_h = p_H^0$, we know that the function $x_U - x_H$ is also positive at $\lambda = 0$. So, we can conclude that $x_U > x_H$ at $p_h = p_H^0$.

(iii) Straightforward given (i) and (ii).

(iv) Straightforward given (i) and (ii).

$$(v) \quad \frac{\partial^2(x_U - x_H)}{\partial p_h^2} = d^2(p_H^0 - p_H^1)v''(C_h)(1 - \lambda) < 0.$$

$$\frac{\partial^2(x_U - x_H)}{\partial p_h \partial \lambda} = d(p_H^0 - p_H^1)v'(C_h) > 0.$$

□

4 Proof of Result 8

(i) We can compute

$$\frac{\partial x_U}{\partial p_L} = (1 - \lambda) [v(C_U^0) - v(C_U^1) - d(p_U^0 v'(C_U^0) - p_U^1 v'(C_U^1))]$$

We have $C_U^0 < C_U^1$. So, $v(C_U^0) < v(C_U^1)$ since $v'(\cdot) > 0$ and we have $v'(C_U^0) > v'(C_U^1)$ since $v''(\cdot) < 0$. Since $p_U^0 > p_U^1$, We also have $p_U^0 v'(C_U^0) - p_U^1 v'(C_U^1) > 0$.

Therefore we conclude that $\frac{\partial x_U}{\partial p_L} < 0$. Since $\frac{\partial x_H}{\partial p_L} = 0$, it follows that $\frac{\partial(x_U - x_H)}{\partial p_L} < 0$.

(ii) At $p_L = 0$, we can compute

$$\begin{aligned} x_U - x_H|_{p_L=0} &= (\lambda - 1)(p_H^1 - p_H^0)v(C_h) - \lambda p_H^1(y - d\lambda p_H^1) \\ &\quad + \lambda p_H^0(y - d\lambda p_H^0) + p_H^1 v(C_H^1) - p_H^0 v(C_H^0). \end{aligned}$$

If $\lambda = 0$, $x_U - x_H|_{p_L=0} = -(p_H^1 - p_H^0)v(C_h) + p_H^1 v(C_H^1) - p_H^0 v(C_H^0)$.

This last expression is increasing in p_h , and at $p_h = p_H^0$, it equals

$p_H^1 (v(C_H^1) - v(C_H^0))$, which is positive. So, this implies that $x_U - x_H|_{p_L=0} > 0$

when $\lambda = 0$. This result, combined with $x_U - x_H|_{p_L=0} = 0$ when $\lambda = 1$ and

the fact that $x_U - x_H$ is concave in λ , shows that $x_U - x_H|_{p_L=0} > 0$.

(iii) Straightforward given (i) and (ii).

(iv) Straightforward given (i) and (ii).

□

5 Proof of Proposition 1

(i) Straightforward since $\psi^{0,0}$ is independent of ϕ and p_h .

(ii) We first compute α_0 for $\lambda = 0$ and $\lambda = 1$, and then study the concavity of α_0 .

When $\lambda = 0$, $\alpha_0 = p_L v(C_L) - p_L v(C_L) = 0$. When $\lambda = 1$, $\alpha_0 = p_H^0 v(C_H^0) - p_H^0 v(C_H^0) = 0$.

Moreover

$$\frac{\partial \alpha_0}{\partial \lambda} = p_L v(C_L) - p_H^0 v(C_H^0) + (p_H^0 - p_L)v(C_U^0) - d(p_H^0 - p_L)p_U^0 v'(C_U^0),$$

and

$$\frac{\partial^2 \alpha_0}{\partial^2 \lambda} = -2d(p_H^0 - p_L)^2 v'(C_U^0) + d^2(p_H^0 - p_L)^2 p_U^0 v''(C_U^0) < 0,$$

Therefore, α_0 is concave in λ . The fact that $\alpha_0 = 0$ for $\lambda = 0$ and $\lambda = 1$ and is concave with λ implies that $\alpha_0 > 0$ for $0 < \lambda < 1$.

If $\phi \geq \text{Max}((1 + \beta)\phi_{max}, \text{max}(\phi_H, \phi_U))$, we have $\psi^{0,0} = \psi_{NR}^{0,0} + \beta\alpha_0$, with $\beta\alpha_0 > 0$. Therefore, $\psi^{0,0} > \psi_{NR}^{0,0}$.

(iii) When $(1 + \beta)\phi_{min} \leq \text{max}(\phi_H, \phi_U) \leq \phi < (1 + \beta)\phi_{max}$, only type- H agents undertake effort in the model without reclassification, while agents do not undertake effort in the model with reclassification. In this case, the private value of the test without reclassification, $\psi_{NR}^{1,0}$, is given by : $\psi_{NR}^{1,0} = (1 + \beta)[\lambda v(C_H^1 + (1 - \lambda)v(C_L) - v(C_U^0))] - \lambda\phi$.

We compute $\psi^{0,0} - \psi_{NR}^{1,0}$ and study its sign.

$$\psi^{0,0} - \psi_{NR}^{1,0} = \beta\alpha_0 - \lambda[(1 + \beta)\phi_{max} - \phi].$$

We know that $\phi < (1 + \beta)\phi_{max}$. Therefore, $\psi^{0,0} > \psi_{NR}^{1,0}$ provided that $\beta\alpha_0 > \lambda[(1 + \beta)\phi_{max} - \phi]$.

When $\text{max}(\phi_H, \phi_U) \leq \phi < (1 + \beta)\phi_{min}$, types H and U agents undertake effort in the model without reclassification, while agents do not undertake effort in the model with reclassification. The private value of the test without reclassification $\psi_{NR}^{1,1}$ is given by : $\psi_{NR}^{1,1} = (1 + \beta)[\lambda v(C_H^1 + (1 - \lambda)v(C_L) - v(C_U^1))] + (1 - \lambda)\phi$.

We compute $\psi^{0,0} - \psi_{NR}^{1,1}$ and study its sign.

$$\psi^{0,0} - \psi_{NR}^{1,1} = \beta\alpha_0 + (1 + \beta)\phi_{min} - \phi - \lambda[(1 + \beta)\phi_{max} - \phi].$$

We know that $\phi < (1 + \beta)\phi_{min} < (1 + \beta)\phi_{max}$. We conclude that $\psi^{0,0} > \psi_{NR}^{1,1}$ provided that $\beta\alpha_0 + (1 + \beta)\phi_{min} - \phi > \lambda[(1 + \beta)\phi_{max} - \phi]$.

(iv) We study the sign of the coefficient of β , denoted by A , in $\psi^{0,0}$.

Rearranging $\psi^{0,0}$, we obtain $\psi^{0,0} = \lambda v(C_H^0) + (1 - \lambda)v(C_L) - v(C_U^0) + \beta A$,
with

$$\begin{aligned} A &= \lambda(1 - p_H^0)v(C_H^0) + (1 - \lambda)(1 - p_L)v(C_L) - (1 - p_U^0)v(C_U^0), \\ &= \lambda v(C_H^0) + (1 - \lambda)v(C_L) - v(C_U^0) + p_U^0 v(C_U^0) - \lambda p_H^0 v(C_H^0) - \\ &\quad (1 - \lambda)p_L v(C_L) \end{aligned}$$

Let us denote \hat{H} , the Hirshleifer effect, with $\hat{H} = \lambda v(C_H^0) + (1 - \lambda)v(C_L) - v(C_U^0) < 0$. It becomes $A = \hat{H} + \alpha_0$.

Thus, in the second period agents make a trade off between the negative effect of genetic discrimination, commonly called in the literature Hirshleifer effect \hat{H} and the positive effect of reclassification α_0 . $A > 0$ and then $\psi^{0,0}$ is increasing in β when the positive effect of reclassification $\alpha_0 > \hat{H}$. This is more likely to happen if individuals are not too risk-averse.

(v) We study the sign of $\psi^{0,0} = C + \beta \times A$, with

$$C = \lambda v(C_H^0) + (1 - \lambda)v(C_L) - v(C_U^0)$$

and

$$A = \lambda(1 - p_H^0)v(C_H^0) + (1 - \lambda)(1 - p_L)v(C_L) - (1 - p_U^0)v(C_U^0).$$

According to Jensen's inequality, C is strictly negative while A can be positive or negative from item *iv*).

We conclude that :

If $A < 0$, then $\psi^{0,0} < 0$.

When $A > 0$, $\psi^{0,0}$ can be positive or negative.

If $A > 0$, then $\psi^{0,0} > 0$ provided that $\beta > \frac{-C}{A}$.

If $A > 0$, then $\psi^{0,0} < 0$ if and only if $0 < \beta < \frac{-C}{A}$.

(vi) We have

$$\frac{\partial \alpha_0}{\partial p_L} = -(1 - \lambda)(v(C_L) - v(C_U^0)) - d(1 - \lambda)[p_U^0 v'(C_U^0) - p_L v'(C_L)].$$

We know that $C_L > C_U^0$. So, $v(C_L) > v(C_U^0)$ since $v'(\cdot) > 0$. We also have $v'(C_U^0) > v'(C_L) > 0$ since $v''(\cdot) < 0$. Since $p_U^0 > p_L$, we can conclude that $\frac{\partial \alpha_0}{\partial p_L} < 0$.

We compute $\frac{\partial \alpha_0}{\partial p_H^0}$.

$$\frac{\partial \alpha_0}{\partial p_H^0} = \lambda(v(C_U^0) - v(C_H^0)) + \lambda d(p_H^0 v'(C_H^0) - p_U^0 v'(C_U^0)).$$

We have $C_U^0 > C_H^0$. So, $v(C_U^0) > v(C_H^0)$ since $v'(\cdot) > 0$.

We know that $p_H^0 > p_U^0$. We also have $v'(C_H^0) > v'(C_U^0)$ since $v''(\cdot) < 0$.

Therefore, we can conclude that $\frac{\partial \alpha_0}{\partial p_H^0} > 0$.

□

6 Impact of risk aversion on the decision to take the test with reclassification

We study how the degree of risk aversion impacts the decision to take the test when no agent undertakes effort.

We recall that $\psi^{0,0} = \lambda v(C_H^0) + (1 - \lambda)v(C_L) - v(C_U^0) + \beta A$,

with $A = \lambda(1 - p_H^0)v(C_H^0) + (1 - \lambda)(1 - p_L)v(C_L) - (1 - p_U^0)v(C_U^0)$.

We recall that $\hat{H} = \lambda v(C_H^0) + (1 - \lambda)v(C_L) - v(C_U^0) < 0$, representing the negative impact of genetic discrimination, commonly called in the literature the Hirshleifer effect, that is higher the higher the degree of risk aversion.

It becomes : $\psi^{0,0} = \hat{H} + \beta \times A$.

We then study how the degree of risk aversion affects the sign of A .

Dividing A by $(1 - p_U^0)$, we obtain :

$$\begin{aligned}\frac{A}{(1 - p_U^0)} &= \frac{\lambda(1 - p_H^0)}{(1 - p_U^0)}v(C_H^0) + \frac{(1 - \lambda)(1 - p_L)}{(1 - p_U^0)}v(C_L) - v(C_U^0) \\ &= \mu v(C_H^0) + (1 - \mu)v(C_L) - v(C_U^0),\end{aligned}$$

with $\frac{\lambda(1 - p_H^0)}{(1 - p_U^0)} = \mu < \lambda$ that is equal to λ when $\lambda = 0$ and $\lambda = 1$ and $\frac{(1 - \lambda)(1 - p_L)}{(1 - p_U^0)} = (1 - \mu) > (1 - \lambda)$.

We know that according to Jensen's inequality if $v'(\cdot)$ is concave,

$$v(C_U^0) > \lambda v(C_H^0) + (1 - \lambda)v(C_L)$$

However, if $v'(\cdot)$ is linear, corresponding to risk neutral agent, we have :

$$v(C_U^0) = \lambda v(C_H^0) + (1 - \lambda)v(C_L)$$

So if the agent is risk neutral, as we increase the weight of $v(C_L)$ but decrease the weight of $v(C_H^0)$, with $v(C_L) > v(C_H^0)$. we obtain :

$$\frac{\lambda(1 - p_H^0)}{(1 - p_U^0)}v(C_H^0) + \frac{(1 - \lambda)(1 - p_L)}{(1 - p_U^0)}v(C_L) - v(C_U^0) > 0$$

This inequality should be satisfied if $v(\cdot)$ is not too concave. We conclude that $\psi^{0,0}$ is likely positive if agents are lowly risk-averse.

7 Proof of Proposition 2

(i) $\frac{\partial \psi^{1,1}}{\partial \phi} = (1 - \lambda) > 0$.

As $\psi^{1,1}$ is independent of p_h , $\frac{\partial \psi^{1,1}}{\partial p_h} = 0$

(ii) We show that $\alpha_1 > 0$. To do so, we first evaluate *it* for $\lambda = 0$ and $\lambda = 1$, and then study its concavity.

When $\lambda = 0$, $\alpha_1 = p_L v(C_L) - p_L v(C_L) = 0$. When $\lambda = 1$, $\alpha_1 = p_H^1 v(C_H^1) -$

$$p_H^1 v(C_H^1) = 0.$$

Moreover

$$\frac{\partial \alpha_1}{\partial \lambda} = p_L v(C_L) - p_H^1 v(C_H^1) + (p_H^1 - p_L) v(C_U^1) - d(p_H^1 - p_L) p_U^1 v'(C_U^1),$$

and

$$\frac{\partial^2 \alpha_1}{\partial \lambda^2} = -2d(p_H^1 - p_L)^2 v'(C_U^1) + d^2(p_H^1 - p_L)^2 p_U^1 v''(C_U^1) < 0.$$

Therefore, α_1 is concave in λ .

The fact that $\alpha_1 = 0$ for $\lambda = 0$ and $\lambda = 1$ and is concave with λ implies that $\alpha_1 > 0$ for $0 < \lambda < 1$.

- (iii) When $\phi \leq \text{Min}((1 + \beta)\phi_{min}, \min(\phi_H, \phi_U))$, types U and H agents undertake effort both in the model with and without reclassification. We have
- $$\psi^{1,1} = \psi_{NR}^{1,1}(\phi, \Delta) + \beta\alpha_1.$$

Then $\psi^{1,1} - \psi_{NR}^{1,1}(\phi, \Delta) = \beta\alpha_1 > 0$.

When $(1 + \beta)\phi_{min} < \phi \leq \min(\phi_H, \phi_U) < (1 + \beta)\phi_{max}$, only type- H agents undertake effort in the dynamic model without reclassification. The private value of the test without reclassification is given by :

$$\psi_{NR}^{1,0} = (1 + \beta)[\lambda v(C_H^1) + (1 - \lambda)v(C_L) - v(C_U^0)] - \lambda\phi.$$

We compute $\psi^{1,1} - \psi_{NR}^{1,0}$.

$$\psi^{1,1} - \psi_{NR}^{1,0} = -(1 + \beta)\phi_{min} + \phi + \beta\alpha_1.$$

We know that $\phi > (1 + \beta)\phi_{min}$. It implies that $\phi > (1 + \beta)\phi_{min} - \beta\alpha_1$ since $\beta\alpha_1 > 0$. We conclude that $\psi^{1,1} > \psi_{NR}^{1,0}$.

When $(1 + \beta)\phi_{max} < \phi \leq \min(\phi_H, \phi_U)$, no agents undertake prevention effort in the dynamic model without reclassification. The private value of the test without reclassification, $\psi_{NR}^{0,0}$, is defined as follows :

$$\psi_{NR}^{0,0} = (1 + \beta)[\lambda v(C_H^0) + (1 - \lambda)v(C_L) - v(C_U^0)].$$

We compute $\psi^{1,1} - \psi_{NR}^{0,0}$.

$$\psi^{1,1} - \psi_{NR}^{0,0} = (1 + \beta)[\lambda\phi_{max} - \phi_{min}] + \beta\alpha_1 + (1 - \lambda)\phi.$$

We conclude that $\psi^{1,1} > \psi_{NR}^{0,0}$ if $\lambda\phi_{max} > \phi_{min}$ or provided that

$$(1 - \lambda)\phi + \beta\alpha_1 > (1 + \beta)[\phi_{min} - \lambda\phi_{max}]$$

when $\lambda\phi_{max} < \phi_{min}$.

(iv) Rearranging $\psi^{1,1}$, we obtain $\psi^{1,1} = \lambda v(C_H^1) + (1 - \lambda)v(C_L) - v(C_U^1) + \beta A_1 + (1 - \lambda)\phi$,

with A_1 the coefficient of β in $\psi^{1,1}$ such that $A_1 = \lambda(1 - p_H^1)v(C_H^1) + (1 - \lambda)(1 - p_L)v(C_L) - (1 - p_U^1)v(C_U^1)$.

Rearranging A_1 , we obtain :

$$A_1 = \lambda v(C_H^1) + (1 - \lambda)v(C_L) - v(C_U^1) + p_U^1 v(C_U^1) - \lambda p_H^1 v(C_H^1) - (1 - \lambda)p_L v(C_L).$$

We can rewrite A_1 as follows : $A_1 = \hat{H}_1 + \alpha_1$.

Thus, as in the case where agents do not undertake effort, in the second period agents make a trade-off between the negative Hirschleifer effect \hat{H}_1 and the positive effect of reclassification α_1 . $A_1 > 0$ and then $\psi^{1,1}$ is increasing in β provided that $\alpha_1 > \hat{H}_1$. Otherwise, $\psi^{1,1}$ is decreasing in β .

(v) We study the sign of $\psi^{1,1}$.

Let $C_1 = \lambda v(C_H^1) + (1 - \lambda)v(C_L) - v(C_U^1) + (1 - \lambda)\phi$ and $A_1 = \lambda(1 - p_H^1)v(C_H^1) + (1 - \lambda)(1 - p_L)v(C_L) - (1 - p_U^1)v(C_U^1)$.

We can define $\psi^{1,1}$ as follows : $\psi^{1,1} = C_1 + \beta A_1$.

C_1 can be positive or negative and depends on ϕ while we know that A_1 can be positive or negative from iii).

We then have:

If C_1 and $A_1 < 0$, then $\psi_{DD}^{1,1} < 0$.

If C_1 and $A_1 > 0$, then $\psi_{DD}^{1,1} > 0$.

If $C_1 > 0$ and $A_1 < 0$ (resp. $A_1 > 0$ and $C_1 < 0$), then $\psi_{DD}^{1,1} > 0$ provided that $\beta < \frac{-C_1}{A_1}$ (resp. $\beta > \frac{-C_1}{A_1}$).

If $C_1 > 0$ and $A_1 < 0$ (resp. $A_1 > 0$ and $C_1 < 0$), then $\psi_{DD}^{1,1} < 0$ provided that $\beta > \frac{-C_1}{A_1}$ (resp. $\beta < \frac{-C_1}{A_1}$).

(vi) We have

$$\frac{\partial \alpha_1}{\partial p_L} = -(1 - \lambda)(v(C_L) - v(C_U^1)) - d(1 - \lambda)[p_U^1 v'(C_U^1) - p_L v'(C_L)].$$

We have $C_L > C_U^1$. So, $v(C_L) > v(C_U^1)$ since $v'(\cdot) > 0$. We also have $v'(C_U^1) > v'(C_L) > 0$ since $v''(\cdot) < 0$. We also know that $p_U^1 > p_L$. We finally conclude that $\frac{\partial \alpha_1}{\partial p_L} < 0$.

We can compute

$$\frac{\partial \alpha_1}{\partial p_H^1} = \lambda(v(C_U^1) - v(C_H^1)) + d\lambda[p_H^1 v'(C_H^1) - p_U^1 v'(C_U^1)].$$

We have $C_U^1 > C_H^1$. $v(C_U^1) > v(C_H^1)$ since $v'(\cdot) > 0$.

We know that $p_H^1 > p_U^1$. We also have $v'(C_H^1) > v'(C_U^1)$ since $v''(\cdot) < 0$.

Therefore, we can conclude that $\frac{\partial \alpha_1}{\partial p_H^1} > 0$.

(vii) We first show that $\psi^{1,1}(\phi = 0)$ and $\psi^{1,1}(\min(\phi_H, \phi_U))$ can be positive or negative. We have $\psi^{1,1}(\phi = 0) = (1 + \beta)[\lambda v(C_H^1) + (1 - \lambda)v(C_L) - v(C_U^1)] + \beta \alpha_1$, with $\lambda v(C_H^1) + (1 - \lambda)v(C_L) - v(C_U^1) < 0$ according to Jensen's inequality. Thus, $\psi^{1,1}(\phi = 0) > 0$ provided that $\beta \alpha_1 > (1 + \beta)[\lambda v(C_H^1) + (1 - \lambda)v(C_L) - v(C_U^1)]$.

If $\min(\phi_H, \phi_U) = \phi_U$, $\psi^{1,1}(\min(\phi_H, \phi_U)) = \psi^{1,1}(\phi_U)$.

We have $\psi^{1,1}(\phi_U) = (1 + \beta)[\lambda v(C_H^1) + (1 - \lambda)v(C_L) - v(C_U^1)] + \beta \alpha_1 + (1 - \lambda)\phi_U$,

with $\phi_U = (1 + \beta)\phi_{min} + \beta x_U$.

We can rewrite $\psi^{1,1}(\phi_U)$ as follows:

$$\psi^{1,1}(\phi_U) = \psi_{NR}^{1,1}((1 + \beta)\phi_{min}) + \beta\alpha_1 + (1 - \lambda)\beta x_U.$$

We know from Result 3 that $\psi_{NR}^{1,1}((1 + \beta)\phi_{min}) > 0$ for all $\Delta > \tilde{\Delta}$. We also know that x_U can be positive or negative. We conclude that $\psi^{1,1}(\phi_U) > 0$ if $x_U > 0$ or $x_U < 0$ and $|x_U| < \psi_{NR}^{1,1}((1 + \beta)\phi_{min}) + \beta\alpha_1$. If $x_U < 0$ and $|x_U| > \psi_{NR}^{1,1}((1 + \beta)\phi_{min}) + \beta\alpha_1$, then $\psi^{1,1}(\phi_U) < 0$.

If $\min(\phi_H, \phi_U) = \phi_H$, $\psi^{1,1}(\min(\phi_H, \phi_U)) = \psi^{1,1}(\phi_H)$. We have : $\psi^{1,1}(\phi_H) = (1 + \beta)[\lambda v(C_H^1) + (1 - \lambda)v(C_L) - v(C_U^1)] + \beta\alpha_1 + (1 - \lambda)\phi_H$,

with $\phi_H = (1 + \beta)\phi_{max} + \beta x_H$.

We can rewrite $\psi^{1,1}(\phi_H)$ as follows:

$$\psi^{1,1}(\phi_H) = \psi_{NR}^{1,1}((1 + \beta)\phi_{max}) + \beta\alpha_1 + (1 - \lambda)\beta x_H,$$

with $\psi_{NR}^{1,1}((1 + \beta)\phi_{max}) > 0$ for all $\Delta > \tilde{\Delta}$, and x_H that can be positive or negative. We conclude that $\psi^{1,1}(\phi_H) > 0$ if $x_H > 0$ or $x_H < 0$ and $|x_H| < \psi_{NR}^{1,1}((1 + \beta)\phi_{max}) + \beta\alpha_1$. If $x_H < 0$ and $|x_H| > \psi_{NR}^{1,1}((1 + \beta)\phi_{max}) + \beta\alpha_1$, then $\psi^{1,1}(\phi_H) < 0$.

As $\frac{\partial \psi^{1,1}}{\partial \phi} = (1 - \lambda) > 0$, $\psi^{1,1}$ is strictly increasing and continuous in ϕ . This implies, by the intermediate value theorem, that there exists a unique value of ϕ , denoted by $\tilde{\phi}_{1R}$, such that $\psi^{1,1}(\tilde{\phi}_{1R}) = 0$ and $0 < \tilde{\phi}_{1R} < \min(\phi_H, \phi_U)$ provided that $\psi^{1,1}(\min(\phi_H, \phi_U)) > 0$ and $\psi^{1,1}(0) < 0$.

When $\Delta > \tilde{\Delta}$, we know from Result 3 that there exists a unique positive value of ϕ , denoted by $\tilde{\phi}_{1NR}(\Delta)$, such that $\psi_{NR}^{1,1}(\tilde{\phi}_{1NR}(\Delta), \Delta) = 0$.

Thus $(1 + \beta)[\lambda v(C_H^1) + (1 - \lambda)v(C_L) - v(C_U^1)] + (1 - \lambda)\tilde{\phi}_{1NR}(\Delta) = 0$.

We compute $\psi^{1,1}$ for $\phi = \tilde{\phi}_{1NR}(\Delta)$.

$$\begin{aligned}\psi^{1,1}(\tilde{\phi}_{1NR}(\Delta)) &= (1+\beta)[\lambda v(C_H^1) + (1-\lambda)v(C_L) - v(C_U^1)] + \beta\alpha_1 + (1-\lambda)\tilde{\phi}_{1NR}(\Delta), \\ &= \psi_S^{1,1}(\tilde{\phi}_{1NR}(\Delta), \Delta) + \beta\alpha_1, \text{ with } \psi_S^{1,1}(\tilde{\phi}_{1NR}(\Delta)) = 0.\end{aligned}$$

Then, we have $\psi^{1,1}(\tilde{\phi}_{1NR}(\Delta)) = \beta\alpha_1 > 0$, since $\beta > 0$ and $\alpha_1 > 0$.

As $\psi^{1,1}$ is increasing with ϕ , the value ϕ for which $\psi^{1,1}(\phi, \Delta) = 0$, denoted by $\tilde{\phi}_{1R}$, is strictly lower than $\tilde{\phi}_{1NR}(\Delta)$.

$\psi^{1,1}(\tilde{\phi}_{1R}) = 0$ implies that

$$\tilde{\phi}_{1R} = \frac{(1+\beta)[v(C_U^1) - \lambda v(C_H^1) - (1-\lambda)v(C_L)] - \beta\alpha_1}{1-\lambda}.$$

□

8 Proof of Proposition 3

(i) Straightforward since $\frac{\partial \psi^{1,0}}{\partial \phi} = -\lambda < 0$.

$$\frac{\partial \psi^{1,0}}{\partial p_h} = \beta \lambda d(p_H^0 - p_H^1) v'(C_h) > 0.$$

$$\frac{\partial \psi^{1,0}}{\partial p_H^0} = d\lambda v'(C_U^0) + \beta[\lambda(v(C_U^0) - v(C_h)) + d\lambda(1 - p_U^0)v'(C_U^0)] > 0.$$

$$\frac{\partial \psi^{1,0}}{\partial p_H^1} = -d\lambda v'(C_H^1) + \beta[-\lambda(v(C_H^1) - v(C_h)) - d\lambda(1 - p_H^1)v'(C_H^1)] < 0.$$

(ii) We compute $\frac{\partial \alpha_2}{\partial p_h}$.

$$\frac{\partial \alpha_2}{\partial p_h} = \lambda d(p_H^0 - p_H^1) v'(C_h) > 0.$$

We compute $\frac{\partial \alpha_2}{\partial p_L}$.

$$\frac{\partial \alpha_2}{\partial p_L} = -(1 - \lambda)(v(C_L) - v(C_U^0)) - d(1 - \lambda)[p_U^0 v'(C_U^0) - p_L v'(C_L)] < 0.$$

We now study the sign of α_2 .

We add and subtract $\lambda p_H^0 v(C_H^0)$ to α_2 , we obtain :

$$\alpha_2 = p_U^0 v(C_U^0) - \lambda p_H^0 v(C_H^0) - (1 - \lambda)p_L v(C_L) + \lambda[(p_H^1 - p_H^0)v(C_h) + p_H^0 v(C_H^0) - p_H^1 v(C_H^1)].$$

We know from proposition 1 that $\alpha_0 = p_U^0 v(C_U^0) - \lambda p_H^0 v(C_H^0) - (1 - \lambda)p_L v(C_L) > 0$. We also know that $x_H = (p_H^1 - p_H^0)v(C_h) + p_H^0 v(C_H^0) - p_H^1 v(C_H^1)$ which can be positive or negative.

It becomes : $\alpha_2 = \alpha_0 + \lambda x_H$. We conclude that if $x_H > 0$, then $\alpha_2 > 0$. If $x_H < 0$, then α_2 can be positive or negative.

(iii) We study the sign of the coefficient of β , denoted by A_2 , in $\psi^{1,0}$.

Rearranging $\psi^{1,0}$, we obtain $\psi^{1,0} = \lambda v(C_H^1) + (1 - \lambda)v(C_L) - v(C_U^0) + \beta A_2$, with

$$A_2 = \lambda(1 - p_H^1)v(C_H^1) + (1 - \lambda)(1 - p_L)v(C_L) - (1 - p_U^0)v(C_U^0) - \lambda(p_H^0 - p_H^1)v(C_h).$$

We can rewrite A_2 as follows:

$$\begin{aligned} A_2 &= \lambda v(C_H^1) + (1 - \lambda)v(C_L) - v(C_U^0) + p_U^0 v(C_U^0) - \lambda p_H^1 v(C_H^1) - \\ &\quad (1 - \lambda)p_L v(C_L) + \lambda(p_H^1 - p_H^0)v(C_h). \end{aligned}$$

We add and subtract $\lambda v(C_H^0)$ and $\lambda p_H^0 v(C_H^0)$ to A_2 .

We obtain :

$$\begin{aligned} A_2 &= \lambda v(C_H^0) + (1 - \lambda)v(C_L) - v(C_U^0) + p_U^0 v(C_U^0) - \lambda p_H^0 v(C_H^0) - \\ &\quad (1 - \lambda)p_L v(C_L) + \lambda[(p_H^1 - p_H^0)v(C_h) - p_H^1 v(C_H^1) + p_H^0 v(C_H^0)] + \\ &\quad \lambda(v(C_H^1) - v(C_H^0)). \end{aligned}$$

We know that $\hat{H} = \lambda v(C_H^0) + (1 - \lambda)v(C_L) - v(C_U^0) < 0$,

$$\alpha_0 = p_U^0 v(C_U^0) - \lambda p_H^0 v(C_H^0) - (1 - \lambda)p_L v(C_L) > 0,$$

$$x_H = (p_H^1 - p_H^0)v(C_h) - p_H^1 v(C_H^1) + p_H^0 v(C_H^0) \text{ and } \phi_{max} = v(C_H^1) - v(C_H^0) > 0.$$

We know from Result 6 that x_H can be positive or negative.

$$\text{We have then : } A_2 = \hat{H} + \alpha_0 + \lambda x_H + \lambda \phi_{max}.$$

We conclude that, when $x_H > 0$ (i.e, if the intensity of reclssification is high),

A_2 is positive provided that $\alpha_0 + \lambda x_H + \lambda \phi_{max} > -\hat{H}$. Otherwise, A_2 is negative.

When $x_H < 0$, A_2 is positive provided that $\alpha_0 + \lambda \phi_{max} > -\hat{H} - \lambda x_H$.

(iv) In order to study the sign of $\psi^{1,0}$, we can rewrite it as follows :

$$\psi^{1,0} = C_2 + \beta A_2,$$

with $C_2 = \lambda v(C_H^1) + (1 - \lambda)v(C_L) - v(C_U^0) - \lambda \phi$ and

$$A_2 = \lambda(1 - p_H^1)v(C_H^1) + (1 - \lambda)(1 - p_L)v(C_L) - (1 - p_U^0)v(C_U^0) - \lambda(p_H^0 - p_H^1)v(C_h).$$

We have $\beta > 0$, A_2 and C_2 can be positive or negative.

If C_2 and $A_2 < 0$, then $\psi_{DD}^{1,0} < 0$.

If C_2 and $A_2 > 0$, then $\psi_{DD}^{1,0} > 0$.

If $C_2 > 0$ and $A_2 < 0$ (resp. $A_2 > 0$ and $C_2 < 0$), then $\psi_{DD}^{1,0} > 0$ provided

$$\text{that } \beta < \frac{-C_2}{A_2} \text{ (resp. } \beta > \frac{-C_2}{A_2} \text{)}.$$

If $C_2 > 0$ and $A_2 < 0$ (resp. $A_2 > 0$ and $C_2 < 0$), then $\psi_{DD}^{1,0} < 0$ provided

$$\text{that } \beta > \frac{-C_2}{A_2} \text{ (resp. } \beta < \frac{-C_2}{A_2} \text{)}.$$

(v) We know from item *vii*) of Proposition 2 that $\psi^{1,1}(\phi_U)$ can be positive or

negative. We have $\psi^{0,0}(\phi_H) = (1 + \beta)[\lambda v(C_H^0) + (1 - \lambda)v(C_L) - v(C_U^0)] + \beta \alpha_0 - \lambda \phi_H$. We can rewrite $\psi^{0,0}(\phi_H)$ as follows: $\psi^{0,0}(\phi_H) = (1 + \beta)\hat{H} + \beta \alpha_0 - \lambda \phi_H$.

Thus, $\psi^{0,0}(\phi_H) > 0$ provided that $\beta \alpha_0 > -(1 + \beta)\hat{H} + \lambda \phi_H$. If $\beta \alpha_0 < -(1 + \beta)\hat{H} + \lambda \phi_H$, then $\psi^{0,0}(\phi_H) < 0$.

Moreover, we have $\frac{\partial \psi^{1,0}}{\partial \phi} = -\lambda < 0$, implying that $\psi^{1,0}$ is strictly decreasing and continuous in ϕ . This implies, by the intermediate value theorem, that there exists a unique value of ϕ , denoted by $\tilde{\phi}_{2R}$, such that $\psi^{1,0}(\tilde{\phi}_{2R}) = 0$ and $\phi_U < \tilde{\phi}_{2R} < \phi_H$ provided that $\psi^{1,1}(\phi_U) > 0$ and $\psi^{0,0}(\phi_H) < 0$.

(vi) For all $(1 + \beta)\phi_{min} \leq \phi_U < \phi < \phi_H \leq (1 + \beta)\phi_{max}$, only type- H agents undertake effort both in the model with and without reclassification. We have : $\psi^{1,0} = \psi_{NR}^{1,0} + \beta\alpha_2$. item *ii*) shows that α_2 can be positive or negative.

We conclude that $\psi^{1,0} > \psi_{NR}^{1,0}$ provided that $\alpha_2 > 0$.

We now compare $\psi^{1,0}$ to $\psi_{NR}^{1,1}$, i.e., for all $\phi_U < \phi < \phi_H \leq (1 + \beta)\phi_{min}$.

We have : $\psi^{1,0} - \psi_{NR}^{1,1} = (1 + \beta)\phi_{min} - \phi + \beta\alpha_2$. We know that $\phi < (1 + \beta)\phi_{min}$.

It implies that $(1 + \beta)\phi_{min} - \phi > 0$. We conclude that $\psi^{1,0} > \psi_{NR}^{1,1}$ when $\alpha_2 > 0$.

If $\alpha_2 < 0$, then $\psi^{1,0} > \psi_{NR}^{1,1}$ provided that $(1 + \beta)\phi_{min} - \phi > -\beta\alpha_2$.

We now compare $\psi^{1,0}$ to $\psi_{NR}^{0,0}$, i.e., for all $(1 + \beta)\phi_{max} \leq \phi_U < \phi < \phi_H$.

We compute $\psi^{1,0} - \psi_{NR}^{0,0}$.

$$\psi^{1,0} - \psi_{NR}^{0,0} = \lambda[(1 + \beta)\phi_{max} - \phi] + \beta\alpha_2.$$

We know that $\phi > (1 + \beta)\phi_{max}$, i.e., $(1 + \beta)\phi_{max} - \phi < 0$. Therefore, $\psi^{1,0} > \psi_{NR}^{0,0}$ provided that $\alpha_2 > 0$ and

$$\alpha_2 > \frac{\lambda(\phi - (1 + \beta)\phi_{max})}{\beta}.$$

□

9 Proof of Proposition 4

(i) Straightforward since $\frac{\partial \psi^{0,1}}{\partial \phi} = 1 > 0$.

We compute $\frac{\partial \psi^{0,1}}{\partial p_H^1}$.

$$\frac{\partial \psi^{0,1}}{\partial p_H^1} = d\lambda v'(C_U^1) + \beta d\lambda v'(C_U^1)(1 - p_U^1) + \beta\lambda(v(C_U^1) - v(C_h)) > 0.$$

We compute $\frac{\partial \psi^{0,1}}{\partial p_h}$.

$$\frac{\partial \psi^{0,1}}{\partial p_h} = -\beta\lambda d(p_H^0 - p_H^1)v'(C_h) > 0.$$

We compute $\frac{\partial \psi^{0,1}}{\partial p_H^0}$.

$$\frac{\partial \psi^{0,1}}{\partial p_H^0} = -d\lambda v'(C_U^0) - \beta d\lambda(1 - p_H^0)v'(C_H^0) - \beta\lambda(v(C_H^0) - v(C_h)) < 0.$$

- (ii) Straightforward since $\frac{\partial \alpha_3}{\partial p_L} = -(1 - \lambda)[v(C_L) - v(C_U^1)] - d(1 - \lambda)[p_U^1 v'(C_U^1) - p_L v'(C_L)] < 0$.

We compute $\frac{\partial \alpha_3}{\partial p_h}$. We have

$$\frac{\partial \alpha_3}{\partial p_h} = -\lambda d(p_H^0 - p_H^1)v'(C_h) < 0.$$

We now study the sign of α_3 .

Adding and subtracting $p_U^0 v(C_U^0)$, and rearranging α_3 , we obtain :

$$\alpha_3 = p_U^0 v(C_U^0) - \lambda p_H^0 v(C_H^0) - (1 - \lambda)p_L v(C_L) - [(p_U^1 - p_U^0)v(C_h) - p_U^1 v(C_U^1) + p_U^0 v(C_U^0)].$$

We know that $\alpha_0 = p_U^0 v(C_U^0) - \lambda p_H^0 v(C_H^0) - (1 - \lambda)p_L v(C_L) > 0$. We also know from Result 6 that $x_U = (p_U^1 - p_U^0)v(C_h) - p_U^1 v(C_U^1) + p_U^0 v(C_U^0)$, that can be positive or negative. It becomes : $\alpha_3 = \alpha_0 - x_U$. Therefore, if $x_U < 0$ (i.e. if the intensity of reclassification is low), then $\alpha_3 > 0$. If $x_U > 0$, then α_3 can be positive or negative. It is positive if $\alpha_0 > x_U$. Otherwise, α_3 is negative.

- (iii) We study the sign of the coefficient of β , denoted by A_3 , in $\psi^{0,1}$.

Rearranging $\psi^{0,1}$, we obtain :

$\psi^{0,1} = \lambda v(C_H^0) + (1 - \lambda)v(C_L) - v(C_U^1) + \phi + \beta A_3$, with

$$A_3 = \lambda(1 - p_H^0)v(C_H^0) + (1 - \lambda)(1 - p_L)v(C_L) - (1 - p_U^1)v(C_U^1) + \lambda(p_H^0 - p_H^1)v(C_h).$$

We can rewrite A_3 as follows:

$$A_3 = \lambda v(C_H^0) + (1 - \lambda)v(C_L) - v(C_U^1) + p_U^1 v(C_U^1) - \lambda p_H^0 v(C_H^0) - (1 - \lambda)p_L v(C_L) - (p_U^1 - p_U^0)v(C_h).$$

We add and subtract $\lambda v(C_U^0)$ and $\lambda p_U^0 v(C_U^0)$ to A_3 .

We obtain :

$$A_3 = \lambda v(C_H^0) + (1 - \lambda)v(C_L) - v(C_U^0) + p_U^0 v(C_U^0) - \lambda p_H^0 v(C_H^0) - (1 - \lambda)p_L v(C_L) - [(p_U^1 - p_U^0)v(C_h) - p_U^1 v(C_U^1) + p_U^0 v(C_U^0)] - (v(C_U^1) - v(C_U^0)).$$

We know that $\hat{H} = \lambda v(C_H^0) + (1 - \lambda)v(C_L) - v(C_U^0) < 0$, $\alpha_0 = p_U^0 v(C_U^0) - \lambda p_H^0 v(C_H^0) - (1 - \lambda)p_L v(C_L) > 0$, $x_U = (p_U^1 - p_U^0)v(C_h) - p_U^1 v(C_U^1) + p_U^0 v(C_U^0)$ and $\phi_{min} = v(C_U^1) - v(C_U^0) > 0$.

We have then : $A_3 = \hat{H} + \alpha_0 - x_U - \phi_{min}$.

We conclude that, when $x_U < 0$, A_3 is positive provided that $\alpha_0 - x_U > \phi_{min} - \hat{H}$. Otherwise, A_3 is negative. When $x_U > 0$, A_3 is positive provided that $\alpha_0 > \phi_{min} - \hat{H} + x_U$.

(iv) In order to study the sign of $\psi^{0,1}$, we can rewrite it as follows :

$$\psi^{0,1} = C_3 + \beta A_3,$$

with $C_3 = \lambda v(C_H^0) + (1 - \lambda)v(C_L) - v(C_U^1) + \phi$ and

$$A_3 = \lambda(1 - p_H^0)v(C_H^0) + (1 - \lambda)(1 - p_L)v(C_L) - (1 - p_U^1)v(C_U^1) + \lambda(p_H^0 - p_H^1)v(C_h).$$

We have $\beta > 0$, C_3 and A_3 can be positive or negative.

If C_3 and $A_3 < 0$, then $\psi_{DD}^{0,1} < 0$.

If C_3 and $A_3 > 0$, then $\psi_{DD}^{0,1} > 0$.

If $C_3 > 0$ and $A_3 < 0$ (resp. $A_3 > 0$ and $C_3 < 0$), then $\psi_{DD}^{0,1} > 0$ provided
that $\beta < \frac{-C_3}{A_3}$ (resp. $\beta > \frac{-C_3}{A_3}$).

If $C_3 > 0$ and $A_3 < 0$ (resp. $A_3 > 0$ and $C_3 < 0$), then $\psi_{DD}^{0,1} < 0$ provided
that $\beta > \frac{-C_3}{A_3}$ (resp. $\beta < \frac{-C_3}{A_3}$).

(v) We know from item *vii*) of Proposition 2 that $\psi^{1,1}(\phi_H)$ can be positive or negative. We have $\psi^{0,0}(\phi_U) = (1+\beta)[\lambda v(C_H^0) + (1-\lambda)v(C_L) - v(C_U^0)] + \beta\alpha_0 + \phi_U$. We can rewrite $\psi^{0,0}(\phi_U)$ as follows: $\psi^{0,0}(\phi_U) = (1+\beta)\hat{H} + \beta\alpha_0 + \phi_U$. Thus, $\psi^{0,0}(\phi_U) > 0$ provided that $\beta\alpha_0 + \phi_U > -(1+\beta)\hat{H}$. If $\beta\alpha_0 + \phi_H < -(1+\beta)\hat{H}$, then $\psi^{0,0}(\phi_U) < 0$.

We have shown that $\frac{\partial\psi^{0,1}}{\partial\phi} = 1 > 0$. The fact that $\psi^{0,1}$ is strictly increasing and continuous in ϕ then implies, by the intermediate value theorem, that there exists a unique value ϕ , denoted by $\tilde{\phi}_{3R}$, such that $\psi^{0,1}(\tilde{\phi}_{3R}) = 0$ and $\phi_H < \tilde{\phi}_{3R} < \phi_U$ provided that $\psi^{1,1}(\phi_H) < 0$ and $\psi^{0,0}(\phi_U) > 0$.

(vi) When $(1+\beta)\phi_{max} < \phi_H < \phi < \phi_U$, only type- U agents undertake effort in the model with reclassification while no agent undertakes prevention effort in the model without reclassification.

We compute $\psi^{0,1} - \psi_{NR}^{0,0}$.

$$\psi^{0,1} - \psi_{NR}^{0,0} = \phi - (1+\beta)\phi_{min} + \beta\alpha_3.$$

We know that $\phi > (1+\beta)\phi_{max} > (1+\beta)\phi_{min}$. It implies that $\phi - (1+\beta)\phi_{min} > 0$. We also know from item *ii*) that α_3 can be positive or negative. We conclude that $\psi^{0,1} > \psi_{NR}^{0,0}$ if $\alpha_3 > 0$. If $\alpha_3 < 0$, then $\psi^{0,1}$ can be higher or lower than $\psi_{NR}^{0,0}$.

We now compare $\psi^{0,1}$ to $\psi_{NR}^{1,1}$, i.e., for all $\phi_H < \phi < \phi_U < (1+\beta)\phi_{min}$. We compute $\psi^{0,1} - \psi_{NR}^{1,1}$.

$$\psi^{0,1} - \psi_{NR}^{1,1} = -\lambda[(1+\beta)\phi_{max} - \phi] + \beta\alpha_3.$$

We know that $\phi < (1 + \beta)\phi_{min} < (1 + \beta)\phi_{max}$. It implies that $(1 + \beta)\phi_{max} - \phi > 0$. Therefore, if $\alpha_3 < 0$, then $\psi^{0,1} < \psi_{NR}^{1,1}$. If $\alpha_3 > 0$, then $\psi^{0,1}$ can be higher or lower than $\psi_{NR}^{1,1}$.

Finally, we compare $\psi^{0,1}$ to $\psi_{NR}^{1,0}$, i.e., for all $(1 + \beta)\phi_{min} < \phi_H < \phi < \phi_U < (1 + \beta)\phi_{max}$.

We compute $\psi^{0,1} - \psi_{NR}^{1,0}$.

$$\psi^{0,1} - \psi_{NR}^{1,0} = -\lambda[(1 + \beta)\phi_{max} - \phi] - [(1 + \beta)\phi_{min} - \phi] + \beta\alpha_3.$$

We know that $\phi > (1 + \beta)\phi_{min}$. Therefore, $(1 + \beta)\phi_{min} - \phi < 0$. We also know that $\phi < (1 + \beta)\phi_{max}$. It implies that $(1 + \beta)\phi_{max} - \phi > 0$. We conclude that $\psi^{0,1} > \psi_{NR}^{1,0}$ provided that

$$\phi - (1 + \beta)\phi_{min} + \beta\alpha_3 > \lambda[(1 + \beta)\phi_{max} - \phi].$$

Thus, $\psi^{0,1}$ can be higher or lower than $\psi_{NR}^{1,0}$ for positive or negative values of α_3 . However, $\psi^{0,1}$ is more likely higher than $\psi_{NR}^{1,0}$ if $\alpha_3 > 0$.

□

10 Conditions under which the private value of the test is always positive or negative

We first determine the conditions under which the private value of the test is strictly positive or negative when $\phi_U > \phi_H$.

If $\phi_U > \phi_H$, the private value of the test is always positive provided that the minimum of the curve, denoted by $\psi^{1,1}(\phi = 0)$, is positive since the private value of the test is increasing in ϕ for all $0 < \phi < \phi_U$ and constant for all $\phi \geq \phi_U$.

We compute $\psi^{1,1}(\phi = 0)$.

$$\psi^{1,1}(\phi = 0) = \lambda v(C_H^1) + (1 - \lambda)v(C_L) - v(C_U^1) + \beta \times A_1, \text{ with}$$

$$A_1 = \lambda(1 - p_H^1)v(C_H^1) + (1 - \lambda)(1 - p_L)v(C_L) - (1 - p_U^1)v(C_U^1).$$

We know from Jensen's inequality that $\lambda v(C_H^1) + (1 - \lambda)v(C_L) - v(C_U^1) < 0$. We also know from item *iv*) of Proposition 2 that the coefficient of β , denoted by A_1 , can be positive or negative.

As a result, $\psi^{1,1}(\phi = 0) > 0$ provided that $A_1 > 0$ and

$$\beta > \frac{v(C_U^1) - \lambda v(C_H^1) - (1 - \lambda)v(C_L)}{A_1}.$$

If $\phi_U > \phi_H$, the private value of the test is always negative provided that the private value of the test when no agents undertake effort, $\psi^{0,0}$, is negative. We recall that $\psi^{0,0}$ is given by :

$$\psi^{0,0} = C + \beta \times A,$$

with $C = \lambda v(C_H^0) + (1 - \lambda)v(C_L) - v(C_U^0) < 0$ according to Jensen's inequality; and $A = \lambda(1 - p_H^0)v(C_H^0) + (1 - \lambda)(1 - p_L)v(C_L) - (1 - p_U^0)v(C_U^0)$ that can be positive or negative according to item *iv*) of proposition 1.

Therefore, $\psi^{0,0} < 0$ provided that $A < 0$ or $A > 0$ and $\beta < \frac{-C}{A}$.

We now move to the case where $\phi_H > \phi_U$. When $\phi_H > \phi_U$, the private value of the test is always negative provided that the maximum of the curve, $\psi^{1,1}(\phi_U)$, is negative. We compute $\psi^{1,1}(\phi_U)$.

$$\psi^{1,1}(\phi_U) = (1 + \beta)[\lambda v(C_H^1) + (1 - \lambda)v(C_L) - v(C_U^1)] + \beta\alpha_1 + (1 - \lambda)\phi_U.$$

We know that $\phi_U = (1 + \beta)\phi_{min} + \beta x_U$. We can rewrite $\psi^{1,1}(\phi_U)$ as follows :

$$\begin{aligned}\psi^{1,1}(\phi_U) &= (1 + \beta)[\lambda v(C_H^1) + (1 - \lambda)v(C_L) - v(C_U^1)] + (1 - \lambda)(1 + \beta)\phi_{min} \\ &\quad + \beta\alpha_1 + (1 - \lambda)\beta x_U.\end{aligned}$$

We know from item *viii*) of Result 3 that

$$\begin{aligned}\psi_{NR}^{1,1}((1 + \beta)\phi_{min}) &= (1 + \beta)[\lambda v(C_H^1) + (1 - \lambda)v(C_L) - v(C_U^1)] + \\ &\quad (1 - \lambda)(1 + \beta)\phi_{min} > 0 \text{ for all } \Delta > \tilde{\Delta}.\end{aligned}$$

We have then : $\psi^{1,1}(\phi_U) = \psi_{NR}^{1,1}((1 + \beta)\phi_{min}) + \beta\alpha_1 + (1 - \lambda)\beta x_U$, with $\alpha_1 > 0$ and x_U that can be positive or negative. We conclude that $\psi^{1,1}(\phi_U) < 0$ provided $x_U < 0$ and

$$|x_U| > \frac{\psi_{NR}^{1,1}((1 + \beta)\phi_{min}) + \beta\alpha_1}{(1 - \lambda)\beta}.$$

We now determine the conditions under which the private value of the test is always positive when $\phi_H > \phi_U$.

The private value of the test is always positive provided that $\min(\psi^{1,1}(\phi = 0), \psi^{0,0}) > 0$. As previously, $\psi^{1,1}(\phi = 0) > 0$ provided that $A_1 > 0$ and $\beta > \frac{v(C_U^1) - \lambda v(C_H^1) - (1 - \lambda)v(C_L)}{A_1}$. $\psi^{0,0} > 0$ provided that $A > 0$ and $\beta > \frac{-C}{A}$.

Bibliography

- [1] Andersen S., Harrison G.W., Lau, M.I. and Rutström, E.E. (2013). Discounting Behaviour and the Magnitude Effect: Evidence from a Field Experiment in Denmark, *Economica*, 80(320), pp. 670 - 697.
- [2] Andersson, H. and Lundborg, P. (2007). Perception of Own Death Risk. An analysis of Road-Traffic and Overall Mortality Risks, *Journal of Risk and Uncertainty*, 34(1), pp. 67-84.
- [3] Annemans, L., Redekop, K. and Payne, K. (2013). Current methodological issues in the economic assessment of personalized medicine, *Value Health*, 16(6), pp. 20-26.
- [4] Antoñanzas, F., Juárez-Castellá, C.A. and Rodríguez-Ibeas, R. (2016). Implementing personalized medicine with asymmetric information on prevalence rates, *Health Economics Review*, 6(1), pp. 1-8.
- [5] Antoñanzas, F., Juárez-Castellá, C.A. and Rodríguez-Ibeas, R. (2015). Some economics on personalized and predictive medicine, *European Journal of Health Economics*, 16(9), pp. 985-994.
- [6] Antoñanzas F., Rodríguez-Ibeas, R. and Juárez-Castellá, C.A. (2018). Personalized medicine and pay for performance: Should pharmaceutical firms be fully penalized when treatment fails?, *PharmacoEconomics*, 36(7), pp. 733-743.

- [7] Arrow, K.J. (1971). Insurance, Risk and Resource Allocation, in *Essays in the Theory of Risk-Bearing*, pp. 134-143.
- [8] Bardey, D. (2003). Gestion des risques longs et comportements de risque moral sur les marchés d'assurance maladie : la première inefficacité peut-elle éliminer la seconde ? *Revue d'économie politique*, 113, pp. 323-334.
- [9] Bardey, D. and De Donder, P. (2019). Welfare Impacts of Genetic Testing in Health Insurance Markets: Will Cross-Subsidies Survive?, *Working paper*.
- [10] Bardey, D. and De Donder, P. (2017). Médecine personnalisée, tests génétiques et assurance santé : une tension exacerbée entre antisélection et discrimination des risques, *Revue d'économie financière*, 113, pp. 323-334.
- [11] Bardey, D. and De Donder, P. (2013). Genetic Testing with Primary Prevention and Moral Hazard, *Journal of Health Economics*, 32(5), pp. 768-779.
- [12] Bardey, D., De Donder, P. and Mantilla, C. (2019). How Is the Trade-off between Adverse Selection and Discrimination Risk Affected by Genetic Testing ? Theory and Experiment, *Journal of Health Economics*, 68 :102223.
- [13] Bardey, D. and Lesur, R. (2005). Optimal Health Insurance Contract and *ex ante* Moral Hazard : Is a Deductible Useful ?, *Economics Letters*, 87, pp. 313-317.
- [14] Bardey, D., Nzalé, S. K. and Ventelou, B. (2018). Physicians incentives to adopt personalized medicine: Experimental evidence, *TSE Working Paper*, n° 18-968.
- [15] Barigozzi, F. and Henriët, D. (2011). Genetic information : Comparing Alternative Regulatory Approaches when Prevention Matters, *Journal of Public Economic Theory*, 13 (1), pp. 23-46.

- [16] Barros, P.P. (2011). The simple economics of risk sharing agreements between the NHS and the pharmaceutical industry, *Health Economics*, 20, pp. 461-470.
- [17] Belleflamme, P. and Peitz, M. (2015). Industrial organization: markets and strategies. Cambridge University Press.
- [18] Berberich, K. and Fischer, E.P. (1999). Impact of Modern Genetics on Life Insurance, Cologne Re Publication, 42.
- [19] Bertucci, F., Finetti, P., Cervera, N., Maraninchi, D., Viens, P. and Birnbaum, D. (2006). Gene expression profiling and clinical outcome in breast cancer, *Omic*s, 10(4), pp. 429-443.
- [20] Bertucci, F., Houlgatte, R., Benziane, A., Granjeaud, S., Adélaïde, J., Tagett, R., Loriod, B., Jacquemier, J., Viens, P., Jordan, B., Birnbaum, D. and Nguyen, C. (2000). Gene expression profiling of primary breast carcinomas using arrays of candidate genes, *Human Molecular Genetics*, 9(20), pp. 2981-2991.
- [21] Browne, M. (1992). Evidence of Adverse Selection in the Individual Health Insurance Market, *Journal of Risk and Insurance*, 59(1), pp. 13-33.
- [22] Caplin, A. and Eliaz, K. (2003). Aids policy and Psychology : A Mechanism-Design approach, *Rand J. Econ.* 34 (4), pp. 631-646.
- [23] Caplin, A. and Leahy, J. (2004). The Supply of Information by a Concerned Expert, *Economic Journal*, 114 (497), pp. 487-505.
- [24] Chapman, G.B. (2005). Short-term Cost for Long-term Benefit : Time Preference and Cancer Control, *Health Psychology*, 24(4), pp. S41 - S48.
- [25] Chapman G. B. (2003). Time discounting of health outcomes, *in Time and Decision : Economic and Psychological Perspectives on Intertemporal Choice*.

- Edited by : Lowenstein GA, Read D, Baumestier RF. Russell Sage Foundation, New York, New York ; pp. 395 - 418.
- [26] Chiappori, P-A. (2006). The Welfare of Predictive Medicine, in Handbook : Competitive Failures in Insurances Markets : Theory and Policy Implications, MIT Press, Cambridge, pp. 55-78.
- [27] Cochrane, J. (1995). Time-consistent Health Insurance, *Journal of Political Economy*, 103, pp. 445-473.
- [28] Codori, A.M. and Brandt, J. (1994). Psychological Costs and Benefits of Predictive Testing for Huntington's Disease, *American Journal of Medical Genetics*, 54, pp. 174-184.
- [29] Codori, A.M., Hanson, R. and Brandt, J. (1994). Self-selection in Predictive Testing for Huntington's Disease, *American Journal of Medical Genetics*, 54, pp. 167-173.
- [30] Courbage, C. and De Coulon, A. (2004). Prevention and Private Health Insurance in the UK, *The Geneva Papers on Risk and Insurance*, 29(4), pp. 719-727.
- [31] Courbage, C. and Rey, B. (2012). Optimal prevention and other risks in a two-period model, *Mathematical Social Sciences*, 63, pp. 213 - 21.
- [32] Cournot, A. A. (1838). Recherches sur les principes mathématiques de la théorie des richesses (Researches into the Mathematical Principles of the Theory of Wealth), NT Bacon. Initially published in French.
- [33] Crainich, D. (2017). Self-Insurance with Genetic Testing Tools, *Journal of Risk and Insurance*, 84(1), pp. 73-94.

- [34] Cremer, H. and Pestieau, P. (1996). Redistributive Taxation and Social Insurance, *International Tax and Public Finance*, 3, pp. 281-295.
- [35] Crocker, K.J. and Snow, A. (1992). The Social Value of Hidden Information in Adverse Selection Economies, *Journal of Public Economics*, 48(3), pp. 317-347.
- [36] Dahlby, B. (1981). Adverse Selection and Pareto Improvements through Compulsory Insurance, *Public Choice*, 37, pp. 547-558.
- [37] Danzon, P. and Towse, A. (2003). Differential pricing for pharmaceuticals: Reconciling access, R&D and patents, *International Journal of Healthcare Finance and Economics*, 3, pp. 183-205.
- [38] Danzon, P. and Towse, A. (2002). The economics of gene therapy and pharmacogenetics, *Value in Health*, 5(1), pp. 5-13.
- [39] Danzon, P., Towse, A. and Mestre-Ferrandiz, J. (2015). Value-based differential pricing: Efficient prices for drugs in a global context, *Health Economics*, 24, pp. 294-301.
- [40] Doherty, N.A. and Posey, L. (1998). On the Value of a Checkup: Adverse Selection, Moral Hazard and the Value of Information, *The Journal of Risk and Insurance*, 65(2), pp. 189-211.
- [41] Doherty, N.A. and Thistle, P.D. (1996). Adverse Selection with Endogenous Information in Insurance Markets, *Journal of Public Economics*, 63(1), pp. 83-102.
- [42] Ehrlich, I. and Becker, G. (1972). Market Insurance, Self-Insurance and Self-Protection, *Journal of Political Economy*, 80(4), pp. 623-648.

- [43] Etner, J. and Jeleva, M. (2013). Risk Perception, Prevention and Diagnostic Tests, *Health Economics*, 22(2), pp. 144-56.
- [44] European Commission, (2004). 25 Recommendations on the Ethical, Legal, and Social Implications of Genetic Testing, Brussels.
- [45] Faulkner, E., Annemans, L., Garrison, L., Helfand, M., Holtorf, A-P., Hornberger, J., Hughes, D., et al. (2012). Challenges in the development and reimbursement of personalized medicine-payer and manufacturer perspectives and implications for health economics and outcomes research: A report of the ISPOR Personalized Medicine Special Interest Group, *Value in Health*, 15(8), pp. 1162-1171.
- [46] Feldman, R. and Dowd, B. (2000). Risk Segmentation : Goal or Problem ?, *Journal of Health Economics*, 19(4), pp. 499-512.
- [47] Feldman, S. and Zaller, J. (1992). The Political Culture of Ambivalence : Ideological Responses to the Welfare State, *American Journal of Political Science*, 36(1), pp. 268-307.
- [48] Filipova-Neumann, L. and Hoy, M. (2014) : Managing Genetic Tests, Surveillance, and Preventive Medicine under a Public Health Insurance System, *Journal of Health Economics*, 34(1), pp. 31-41.
- [49] Frederick S, Loewenstein G. and O'Donoghue, T. (2002). Time Discounting and Time Preference : A Critical Review, *Journal of Economic Literature*, 40 (2), pp. 351 - 401.
- [50] Gabszewicz, J., Sonnac, N. and Wauthy, X. (2001). On price competition with complementary goods, *Economics Letters*, 70(3), pp. 431-437.

- [51] Garrison, L. P. and Austin, M. F. (2007). The economics of personalized medicine: a model of incentives for value creation and capture. *Drug information journal: DIJ/Drug Information Association*, 41(4), pp. 501-509.
- [52] Garrison, L.P. and Towse, A. (2017). Value-based pricing and reimbursement in personalized healthcare: Introduction to the basic health economics, *Journal of Personalized Medicine*, 7(3), pp. 1-10.
- [53] Garrison, L.P. and Towse, A. (2014). Economics of personalized medicine: Pricing and reimbursement policies as a potential barrier to development and adoption, Economics of, in: Anthony J. Culyer, *Encyclopedia of Health Economics*, Elsevier, 2, pp. 484-490.
- [54] Geoffard, P. Y. (2000). Assurance maladie : la gestion des risques longs, *Revue d'Économie Politique*, 4, pp. 457 - 482.
- [55] Goetz, L. H. and Schork, N. J. (2018). Personalized medicine: motivation, challenges, and progress, *Fertility and sterility*, 109(6), pp. 952-963.
- [56] Gordon, R. S. Jr. (1983). An Operational Classification of Disease Prevention, *Public Health Reports*, 98(2), pp. 107-109.
- [57] Hatz, M.H.M., Schremser, K. and Rogowski, W.K. (2014). Is individualized medicine more cost-effective? A systematic review, *PharmacoEconomics*, 32(5), pp. 443-455.
- [58] Hendricks-Sturup, R. M. and Lu, C. Y. (2019). Understanding implementation challenges to genetic testing for familial hypercholesterolemia in the United States, *Journal of personalized medicine*, 9(1), p. 9.

- [59] Henriët, D. and Rochet, J-C. (1998). Les justifications de l'intervention publique dans les systèmes de santé, *Économie publique*, 2.
- [60] Hermalin, B. E. and Katz, M.L. (2006) : Privacy, Property Rights and Efficiency: The Economics of Privacy as Secrecy, *Quantitative Marketing and Economics*, 4(3), pp. 209-239.
- [61] Hirshleifer, J. (1971). The Private and Social Value of Information and the Reward to Incentive Activity, *The American Economic Review*, 61(4), pp. 561-574.
- [62] Hoel, M. and Iversen, T. (2002). Genetic Testing when there is a Mix of Compulsory and Voluntary Health Insurance, *Journal of Health Economics*, 21(2), pp. 253-270.
- [63] Hoel, M., 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(5), pp. 847-860.
- [64] Hornberger, J., Cosler, L.E. and Lyman, G.H. (2005). Economic analysis of targeting chemotherapy using a 21-gene RT-PCR assay in lymphnode-negative, estrogen-receptor-positive, early-stage breast cancer, *American Journal of Managed Care*, 11, pp. 313-324.
- [65] Howard, D.H., Hockenberry, J. and David, G. (2017). Personalized medicine when physicians induce demand, in: *Economic Dimensions of Personalized and Precision Medicine*, University of Chicago Press, pp. 217-235.
- [66] Hoy, M., Orsi, F., Eisinger, F. and Moatti, J. P. (2003). The Impact of Genetic Testing on Insurance, *The Geneva Papers on Risk and Insurance-Issues and Practice*, 28(2), pp. 203-221.

- [67] Hoy, M., Peter, R. and Richter, A. (2014) : Take-up for Genetic Tests and Ambiguity, *Journal of Risk and Uncertainty*, 48(2), pp. 111-133.
- [68] Hoy, M. and Polborn, M. (2000). The Value of Genetic Information in the Life Insurance Market, *Journal of Public Economics*, 78(3), pp. 235-252.
- [69] Hoy, M. and Ruse, M. (2005). Regulating Genetic Information in Insurance Markets, *Risk Management and Insurance Review*, 8(2), pp. 211-237.
- [70] Hoy, M. and Witt, J. (2007). Welfare Effects of Banning Genetic Information in the Life Insurance Markets : The Case of BRCA1/2 gens, *Journal of Risk & Insurance*, 74(3), pp. 523-546.
- [71] Joly, Y., Braker, M. and Le Huyhn, M. (2010). Genetic Discrimination in Private Insurance : Global Perspectives, *New Genetics and Society*, 29 (4), pp. 351-368.
- [72] Jørgensen, J. T. (2019). Regulatory Requirements for Companion Diagnostics and Drug-Diagnostic Codevelopment in the United States. In *Companion and Complementary Diagnostics*, pp. 307-318, Academic Press.
- [73] Khan, R. and Mittelman, D. (2018). Consumer Genomics Will Change Your Life, Whether You Get Tested or Not, *Genome Biology*, 19(1):120.
- [74] Kifmann, M. (2001). Premium Risk and Managed Care, *The journal of Risk and Uncertainty*, 22 (3), pp. 277 - 293.
- [75] Kirchheiner, J., Bauer, S., Meineke, I., et al. (2002). Impact of CYP2C9 and CYP2C19 polymorphisms on tolbutamide kinetics and the insulin and glucose response in healthy volunteers, *Pharmacogenetics*, 12(2), pp. 101-109.

- [76] Knoppers, B., Godard, B. and Joly, Y. (2004). Life Insurance and Genetics : A Comparative, International Overview, in M. Rothstein éd., *Genetics and Life Insurance, Medical Underwriting and Social Policy*, Cambridge, MA : MIT Press.
- [77] Knowles, L., Luth, W. and Bubela, T. (2017). Paving the road to personalized medicine: recommendations on regulatory, intellectual property and reimbursement challenges, *Journal of Law and the Biosciences*, 4(3), pp. 453-506.
- [78] Lam, Y.W. (2013). Scientific challenges and implementation barriers to translation of pharmacogenomics in clinical practice, *ISRN pharmacology*, Article ID 641089, 17 pages.
- [79] Lawless, L., Drichoutis, A. C and Nayga Jr, R. M. (2013). Time preferences and health behaviour: a review, *Agricultural and Food economics*, pp. 1-17.
- [80] Lemmens, T. and Bahamin, P. (1998) : Genetics in Life, Disability and Additional Health Insurance in Canada : A Comparative Legal and Ethical Analysis, in B. Knoppers ed., *Socio-Ethical in Human Genetics*, Cowansville, Les Editions Yvon Blais Inc.
- [81] Lemmens, T., Luther, L. and Hoy, M. (2008). Genetic Information Access, a Legal Perspective : A Duty to Know or a Right not to Know, and a Duty or Option to Warm ? in *Encyclopedia of Life Sciences*, John Wiley & Sons, pp. 1-5.
- [82] Lerman, C., Hughes, C., Croyle, R.T., Main, D., Durham, C., Snyder, C., Bonney, A., Lynch, J.F., Narod, S.A., and Lynch, H.T. (2000). Prophylactic Surgery Decisions and Surveillance Practices One Year Following BRCA1/2 Testing, *Preventive Medicine*, 31(1), pp. 75-80.

- [83] Lerman, C., Narod, S., Schulman, K., Hughes, C., Gomez-Caminero, A., Bonney, G., Gold, K., Trock, B., Main, D., Lynch, J. *et al.*(1996). BRCA1 Testing in Families with Hereditary Breast-Ovarian Cancer, *The Journal of the American Medical Association*, 275(24), pp. 1885-1892.
- [84] Levy, D., Byfield, S., Comstock, C., Garber, J., Syngal, S., Crown, W., and Shields, A. (2011). Underutilization of BRCA1/2 Testing to Guide Breast Cancer Treatment: Black and Hispanic Women Particularly at Risk, *Genetics in Medicine*, 13(4), pp. 349-355.
- [85] Lièvre A., Bachet, J.B., Le Corre, D., et al. (2006). KRAS mutation status is predictive of response to cetuximab therapy in colorectal cancer, *Cancer Research*, 66(8), pp. 3992-3995.
- [86] Lodder, L.N., Frets, P.G., Trijsburg, R.W., Meijers-Heijboer, E.J., Klijn, J.G., Seynaeve, C., van Geel, A.N., Tilanus, M.M., Bartels, C.C., Verhoog, L.C., Brekelmans, C.T., Burger, C.W. and Niermeijer, M.F. (2002). One Year Follow-up of Women Opting for Presymptomatic Testing for BRCA1 and BRCA2: Emotional Impact of the Test Outcome and Decisions on Risk Management (Surveillance or Prophylactic Surgery), *Breast Cancer Research and Treatment*, 73(2), pp. 97-112.
- [87] Marteau, T.M. and Croyle, R.T. (1998). The New Genetics : Psychological Responses to Genetic Testing, *British Medical Journal*, 316, pp. 693-696.
- [88] Meijers-Heijboer, E.J., Verhoog, L.C., Brekelmans, C.T., Seynaeve, C., Tilanus-Linthorst, M.M., Wagner, A., Dukel, L., Devilee, P., van den Ouweland, A.M., van Geel, A.N. and Klijn, J.G. (2000). Presymptomatic DNA Testing and Pro-

- phylactic Surgery in Families with a BRCA1 or BRCA2 Mutation, *The Lancet*, 355(9220), pp. 15-20.
- [89] Meiser, B. and Dunn, S. (2000). Psychological Impact of Genetic Testing for Huntington's Disease : An Update of the Literature, *Journal of Neurology, Neurosurgery and Psychiatry*, 69(5), pp. 574-578.
- [90] Menegatti, M. (2009). Optimal Prevention and Prudence in a Two-period Model, *Mathematical Social Sciences*, 58, pp. 393 - 397.
- [91] Miller I., Ashton-Chess, J., Spolders, H., Fert, V., Ferrara, J., et al. (2011). Market access challenges in the EU for high medical value diagnostic tests, *Personalized Medicine*, 8(2), pp. 137-148.
- [92] Mimra, W., Nemitz, J. and Waibel, C. (2019). Voluntary Pooling of Genetic Risk: A Health Insurance Experiment, *Journal of Economic Behavior and Organization*, <https://doi.org/10.1016/j.jebo.2019.04.001>.
- [93] Miyazaki, H. (1977). The Rat Race and Internal Labour Market, *Bell Journal of Economics*, 8 : pp. 394-418.
- [94] Mossin, J. (1968). Aspects of Rationale Insurance Purchasing, *Journal of Political Economy*, 76, pp. 553-568.
- [95] Munich Reinsurance Company (2004). Genetic Testing and Insurance - A Global View, 2ème éd.
- [96] Nyman, J. A. (1999). The Value of Health Insurance: the access motive, *Journal of Health Insurance*, 18(2), pp. 141-152.

- [97] O'Donnell, J.C. (2013). Personalized medicine and the role of health economics and outcomes research: Issues, applications, emerging trends, and future research, *Value Health*, 16(6), pp. 1-3.
- [98] Otlowski, M., Taylor, S. and Bombard, Y. (2012). Genetic Discrimination: International Perspectives, *Annual Review of Genomics and Human Genetics*, vol. 13, pp. 433-454.
- [99] Pannequin, F., Corcos, A. and Montmarquette, C. (2016). Behavioral Foundations of the Substitutability between Insurance and Self-Insurance: An Experimental Study, Cirano working papers.
- [100] Pashchenko, S. and Porapakkarm, P. (2015). Welfare Costs of Reclassification Risk in the Health Insurance Market, *Journal of Macroeconomics*, 45, pp. 21-44.
- [101] Pauly, M.V. (1968). The Economics of Moral Hazard: Comment, *American Economic Review*, 58(3), pp. 531-536.
- [102] Pauly, M.V., Kunreuther, H. and Hirth, R. (1995). Guaranteed Renewability in Insurance, *Journal of Risk and Uncertainty*, 10(2), pp. 143-156.
- [103] Peshkin, B.N., Schwartz, M.D., Isaacs, C., Hughes, C., Main, D. and Lerman, C. (2002). Utilization of Breast Cancer Screening in a Clinically Based Sample of Women after BRCA1/2 Testing, *Cancer Epidemiology, Biomarkers and Prevention*, 11(10), pp. 1115-1118.
- [104] Peter, R., Richter, A. and Thistle, P. (2017). Endogenous Information, Adverse Selection, and Prevention : Implications for Genetic Testing Policy, *Journal of Health Economics*, 55, pp. 95-107.

- [105] Peyron, C., Pélissier, A. and Béjean, S. (2018). Preference heterogeneity with respect to next-generation sequencing : A discrete choice experiment among parents of children with rare genetic diseases, *Social Science and Medicine*, 214, pp. 125-132.
- [106] Picone G., Sloan, F. and Taylor, D. (2004). Effects of Risk and Time Preference and Expected Longevity on Demand for Medical Tests, *Journal of Risk and Uncertainty*, 28(1), pp. 39 - 53.
- [107] Polborn, M.K., Hoy, M. and Sadanand, A. (2006). Advantageous Effects of Regulatory Adverse Selection in the Life Insurance Market, *The Economic Journal*, 116(508), pp. 327-354.
- [108] Posey, L. L. and Yavas, A. (2007). Screening Equilibria in Experimental Markets, *Geneva Risk and Insurance Review*, 32, pp. 147-167.
- [109] Postma, M.J., Boersma, C., Vandijck, D., Vegter, S., Le, H.H. and Annemans, L. (2011). Health technology assessments in personalized medicine: illustrations for cost-effectiveness analysis, *Expert Review of Pharmacoeconomics and Outcomes Research*, 11(4), pp. 367-369.
- [110] Rafiq, M., Ianuale, C., Ricciardi, W. and Boccia, S. (2015). Direct-to-Consumer Genetic Testing: A Systematic Review of European Guidelines, Recommendations, and Position Statements, *Genetic Testing and Molecular Biomarkers*, 19(10), pp. 1-13.
- [111] Rochet, J.-C. (1991). Incentives, Redistribution and Social Insurance, *The Geneva Papers of Risk and Insurance*, 16, pp. 143-165.

- [112] Rothschild, M. and Stiglitz, J. (1976). Equilibrium in Competitive Insurance Markets : An Essay on the Economics of Imperfect Information, *Quarterly Journal of Economics*, 90(4), pp. 629-649.
- [113] Scott Morton, F. and Seabright, P. (2013). Research into biomarkers: how does drug procurement affect the design of clinical trials? *Health Management, Policy and Innovation*, 1(3), pp. 1-15.
- [114] Seror, V., Elasri, K. and Avenel, E. (2007). Premières applications de la pharmacogénomique en oncologie, *Revue d'économie industrielle*, 120(4), pp. 1773-0198.
- [115] Shavell, S. (1979). On Moral Hazard and Insurance, *Quarterly Journal of Economics*, 93 (4), pp. 541-562.
- [116] Slovic, P. (2000). The Perception of Risk, Earthscan Publications Ltd. : London and Sterling.
- [117] Spence, M. (1978). Product Differentiation and Performance in Insurance Markets, *Journal of Public Economics*, 10, pp. 427-447.
- [118] Tabarrok, A. (1994). Genetic Testing: An Economic and Contractarian Analysis, *Journal of Health Economics*, 13(1), pp. 167-207.
- [119] Tardif, J.C., Rhainds, D., Brodeur, M., Feroz Zada, Y., Fouodjio, R., Provost, S., Boulé, M., Alem, S., Grégoire, J.C., L'Allier, P.L., Ibrahim, R., Guertin, M.C., Mongrain, I., Olsson, A.G., Schwartz, G.G., Rhéaume, E. and Dubé, M.P. (2016). Genotype-dependent effects of dalcetrapib on cholesterol efflux and inflammation: Concordance with clinical outcomes, *Circulation: Cardiovascular Genetics*, 9(4), pp. 340-348.

- [120] Trusheim, M.R. and Berndt, E.R. (2012). Economic challenges and possible policy actions to advance stratified medicine, *Personalized Medicine*, 9(4), pp. 413-427.
- [121] Viswanathan, K.S., Lemaire, J., Withers, K., Armstrong, K., Baumritter, A., Hershey, J.C., Pauly, M.V. and Asch, D.A. (2007). Adverse Selection in Term Life Insurance Purchasing Due to the BRCA1/2 Genetic Test and Elastic Demand, *Journal of Risk & Insurance*, 74(1), pp. 65-86.
- [122] Wilson, C. (1977). A Model of Insurance Markets with Incomplete Information, *Journal of Economic Theory*, 16(2), pp. 167-207.