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Take-up for genetic tests and ambiguity

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Abstract Under the expected utility hypothesis a costless genetic test has, at worst, zero private value. This happens if it does not affect optimal decisions. If the genetic test facilitates better decision-making for at least one possible test outcome, then it has positive private value. This theoretical result seems to contradict the fact that empirically observed take-up rates for genetic tests are surprisingly low. We demonstrate that if individuals display ambiguity aversion, a costless genetic test that does not affect optimal decisions is never taken. Furthermore, there is a trade-off between aversion against uncertainty of test results and utility gains from better decision-making if optimal decisions depend on the level of information. The reason is that, from an ex-ante view, a genetic test introduces uncertainty of probabilities which diminishes the value of information to an ambiguity-averse decision-maker. Ambiguity aversion regarding test results thus provides an explanation for low take-up rates for genetic tests.

Keywords Non-expected utility · Ambiguity · Genetic tests · Value of information

JEL Classifications D81 · D83 · I12

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1 Introduction

Genetic tests have the potential to provide information about one's health and mortality prospects. Such information can in turn be used by individuals to improve their life planning decisions, including whether to have children, better choosing an optimal savings plan, or deciding on what occupational training to acquire. If insurers are not allowed access to the information, then one can also improve (opportunistically) one's insurance purchase decisions. Moreover, genomic science has advanced to the stage of being able to identify some so-called "disease genes" or, more appropriately, "disease alleles" which improve our understanding of how specific sequences of genes interact with each other and with environmental factors to affect the onset and influence the treatment of diseases (e.g., see Filipova and Hoy (2014) for a discussion on how such information provides private value with respect to decision-making about surveillance and prevention for disease). Despite these apparent benefits from genetic information, the take-up rates for existing genetic tests seem surprisingly low.[1]

There are some potential negative implications of genetic tests. If insurers are allowed access to such information, as for example are life insurance providers in the United States and Canada, then bad test results can shrink one's market opportunities or lead to uninsurability.[2] Concern over this possibility, often referred to as genetic discrimination, has led many countries to restrict insurers' access to such information.[3] Take-up rates for genetic tests are surprisingly low even in countries which restrict the use of genetic information by insurers and employers.

[1] For example, in various clinical testing environments, when anonymous genetic tests for Huntington's disease were offered at zero cost, Meiser and Dunn (2000) found that the percentage at risk who requested testing varied from 9% to 20% in various centers in UK cities and Vancouver. Lerman et al. (1996) report that almost 60% of their subjects with a family history of breast and/or ovarian cancer declined to receive a costless genetic test for the BRCA1 gene. Levy et al. (2011) find that only 30% of newly diagnosed women with early onset breast cancer choose to receive a genetic test to determine appropriate treatment options. For further discussion, see also Babul et al. (1993) and Quaid and Morris (1993).

[2] This general phenomenon has been termed premium risk. See Tabarrok (1994), Doherty and Thistle (1996) and Strohmenger and Wambach (2000). For a review of the implications of genetic testing for insurance, see Hoy and Ruse (2005).

[3] Worldwide, the reaction to the use of genetic testing for life, private health, and long term disability insurance purposes varies from legislation or total moratoria banning any use of genetic test results by insurers to a status quo approach letting the industry regulate itself. In most of Western Europe the ban is almost total, falling in line with the UNESCO Declaration on Human Genetic Data 2003. In Belgium, insurers are prohibited from even accepting favorable genetic test results provided voluntarily by consumers. In the United Kingdom and the Netherlands, companies can ask for genetic test results only for large policies (those exceeding £500,000 in Britain and the equivalent of $150,000 U.S. (approx.) in the Netherlands). Australia, New Zealand and Canada are among those who allow the status quo to remain (i.e., no explicit restrictions on insurers' access to genetic test results), relying on existing privacy laws and the insurance industry's self-regulation. The United States is a particular case in that, in the absence of socialized medical insurance, the issue involves both the health and the life insurance industries. As well, the regulations vary from state to state. Federally, the Genetic Information Nondiscrimination Act (GINA) passed in May of 2008 addresses the use of genetic testing in health insurance (and is likely to be superseded by the introduction of so-called Obamacare). Only 14 states have introduced some laws to govern the use of genetic testing in life insurance and these laws make restrictions rather than outright bans.

All of the above discussion has been cast, implicitly, within the context of expected utility theory (EUT). If test results do not shrink one's market opportunities (or more generally, one's choice set), then the expected utility generated by a (costless) genetic test must be at least as great as not taking the test. Moreover, if one's optimal decision is changed in light of at least one of the possible test results, then the test has positive expected value. The empirical evidence quoted above is, therefore, surprising as it relates to information that is provided costlessly and anonymously.

As noted by Gollier (2001), in non-expected utility models agents might dislike information. In this paper, we consider the possibility that the behavioral model of ambiguity aversion, as formulated by Klibanoff et al. (2005)—KMM hereafter—can offer an explanation for the observed low take-up rates of genetic tests. This approach is suitable due to the fact that taking a genetic test can be interpreted as a situation where an individual faces second-order probabilities. According to the beliefs of the individual, which may reflect the prevalence of the disease in the population or be based on family medical history, a genetic test with a given test technology leads to a positive test result for a genetic mutation with a certain probability and to a negative test result with the complementary probability. Now each test result itself leads to a specific change in the beliefs of the individual, as individuals receiving "bad" news necessarily revise their priors towards being high risk, whereas people receiving "good" news from a test revise their priors in the other direction. In this sense, a genetic test corresponds ex-ante to a lottery between a deterioration and an improvement in beliefs. By not taking the test, however, the individual avoids this uncertainty and is free to stick to her individual priors over the disease risk. Let us give an example.

A mutation of the genes BRCA1 and BRCA2 is known to be associated with a higher risk for breast or ovarian cancer (see Thompson et al. 2002). If women do not take a test for whether they have such a mutation, they recognize that they might or might not have it. Hence, the risk for breast or ovarian cancer may be viewed as a compound lottery in the first place and the woman is likely to think of her risk exposure in terms of priors over the risk type she may be, especially given the vast amount of information that has been disseminated regarding the role of the BRCA1 and BRCA2 mutations. Someone who chooses not to take a genetic test presumably bases her priors over these probabilities on the relevant subpopulation based on family medical history. Individuals can receive counsel on such likelihoods from medical professionals.[4] If, however, she takes a test, she could either obtain a positive or negative result. The perceived probability of testing positive depends on two factors, individual priors and test technology (or accuracy). If a woman now finds out that she carries a mutation of the respective genes, she will, of course adjust her priors to reflect the new information. In the extreme case where test technology is such that there are no false positives, she will even believe that she is a high-risk type with

[4] See, for example, Table 1, p. 531, in Hoy and Witt (2007) who report that, based on women in the age group 35 to 39, a woman with no first degree relatives has a probability of carrying one of the BRCA genes of 0.001 while if her mother and a sister had ovarian cancer before the age of 50, then the probability rises to 0.065. The relevant (unconditional) probabilities for incurring breast cancer over the next ten years for these two cases are (approximately) 0.013 and 0.30, respectively.

certainty. Naturally, in the case of a negative test result she will think of her breast and ovarian cancer risk more in terms of her being a low risk. Again, she believes being a low risk with certainty if the test has a rate of false negatives of zero. Before taking the test, however, there is still the uncertainty of test results to be resolved. Lastly, the value of genetic information also depends on the opportunities at hand given a specific test result. Since the 1970s strong medical progress has been made regarding therapies available for breast cancer especially when detected early (see Goldhirsch et al. 2007). Hence, individuals would probably adjust their surveillance behavior once they test positive. It becomes apparent that the interaction of decision-making value created by new information and the attitude towards changes in priors together determine the attractiveness of genetic information.

Following the definition given by Camerer and Weber (1992), "ambiguity is uncertainty about probability created by missing information that is relevant and could be known." If individuals consider taking a genetic test, they realize that information regarding disease alleles is missing, but can be obtained, possibly imperfectly, from the test. If they fear ambiguity, the uncertainty of test results might be detrimental in terms of welfare. While a person choosing not to submit to a genetic test also recognizes the potential to be a high- or low-risk type, by choosing not to find out, there is no ex-ante prospect that she will experience the resolution of this uncertainty. In other words, a person submitting to a genetic test "lives through" the ambiguity while a person who remains ignorant does not. Ellsberg (1961) was the first to demonstrate that people might fear uncertainty about probabilities, a phenomenon which has been coined ambiguity aversion. The prevalence of ambiguity aversion has been documented in laboratory experiments (see Einhorn and Hogarth 1986; Chow and Sarin 2001), in market set-ups with educated individuals (see Sarin and Weber 1993), and in surveys of business owners and managers (see Viscusi and Chesson 1999; Chesson and Viscusi 2003). This paper demonstrates that ambiguity aversion over genetic test results may provide a simple and straightforward explanation of empirically observed low take-up rates for genetic tests.

Prior models of information transmission in doctor-patient relations choose different avenues through which to incorporate negative attitudes towards "bad news". In the context of HIV testing, Caplin and Eliaz (2003) introduce an anxiety cost function that captures both the anxiety an agent experiences when diagnosed with HIV for sure and how this relates to the accuracy of tests. They employ these preferences to design a mechanism that encourages testing and slows down the transmission of disease. Kőszegi (2003) models patients' fears as arising from expectations about future health conditions by formulating a utility function of beliefs about physical outcomes. This can explain why agents avoid visiting doctors or obtaining readily available information. Also Caplin and Leahy (2004) incorporate patients' anxiety into a model of information revelation by policy makers. In their models, individuals are heterogeneous regarding the source of anxiety: For some individuals anxiety results from uncertainty about future conditions, whereas for others the extent of certainty of specific future health states constitutes anxiety. The problem of information revelation between two parties where the information has decision-making value, but potentially adverse emotional consequences for one party and the other reacts strategically to those fears, is studied in a general set-up by Kőszegi (2006).

Oster et al. (2013) study the decision to undergo a genetic test for Huntington's disease and find low take-up rates of 10%. They demonstrate that the behavior of tested and untested individuals is consistent with a model of optimal expectations. Schweizer and Szech (2012) study attitudes towards genetic testing in a framework of anticipatory utility and derive implications for test design. The models so far incorporate fear of information in rather ad hoc manners by modeling preferences in various ways that reflect this anxiety. We demonstrate that the information structure implied by a genetic test in combination with ambiguity-averse preferences alone are sufficient to generate low take-up rates and to diminish the decision-making value of genetic information.

We suggest that health information derived from genetic tests is different from other diagnostic or predictive information obtained through traditional medical testing such as determining one's blood sugar level. Even though both types of tests could imply, for example, a revised and higher perceived predisposition towards future onset of type 2 diabetes, a genetic test is viewed as having broader and more profound implications for future lives of individuals. The extent to which this is an appropriate distinction in how these different types of information are perceived by individuals may indeed vary across genetic and other medical tests and we would not claim that all other types of medical testing are immune to the effects of ambiguity aversion. However, much attention has been given to the special character of genetic tests—a phenomenon which has been termed genetic essentialism.[5]

Given the likely future of genetic research and technological developments, it is important to understand the surprising reticence of people to opt for potentially useful genetic tests. With the prospect of the so-called $1000 genome close to reality (see Davies 2010), whole genome sequencing may soon become the norm for developed countries. Even now, according to the web site of the Centers for Disease Control and Prevention, there are over 3000 diseases for which genetic tests have been developed and about 2000 are in use in clinical settings.[6] The information that can be gleaned from an individual's whole genome has the potential to revolutionize the practice of medicine with population wide genome sequencing forming the basis of so-called P4 medicine (i.e., medicine that is Predictive, Preventive, Personalized and Participatory).[7] However, we must understand how individuals assess the value of this information if it is indeed to provide substantial benefits to society.

2 A simple model

In this section we develop a simple model to analyze the decision to undertake a genetic test. We assume that individuals are homogeneous with respect to preferences

[5] See, for example, Nelkin and Lindee (1995), Wolpe (1997), Nordgren and Juengst (2009), and Durnin et al. (2012) for discussion on the "perceived special importance" of genetic information.

[6] See http://www.cdc.gov/genomics/gtesting/, accessed February 27, 2014.

[7] The potential for P4 medicine to improve life outcomes has many proponents, not least of whom is Leroy Hood through his P4 Medicine Institute (p4mi.org). Even more modest use of genetic information should provide substantial benefits (see Filipova and Hoy 2014).

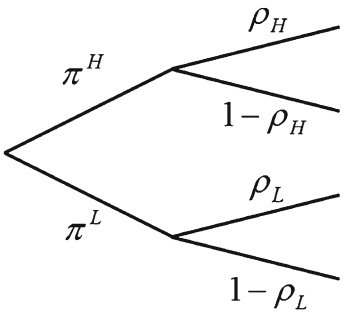

Fig. 1 Information structure before taking a test

across states of nature. Individuals in our model face the risk of becoming ill. In the good state of nature, which we denote by "h" for "healthy", individuals derive utility u_h from consumption.[8] In the bad state of nature, which we denote by "s" for "sick", individuals derive utility u_s from consumption. In this sense, preferences over outcomes are state-dependent and it is natural to assume that $u_h(c) \geq u_s(c)$, i.e., consumption utility when healthy is larger than when sick.[9]

The sick state of the world occurs with probability ρ, and the healthy state of the world with probability $1 - \rho$. Individuals differ in the probability of onset of disease; i.e., we distinguish high-risk and low-risk individuals. This reflects the fact that decision-makers are heterogeneous with respect to their genetic make-up and therefore there are those who suffer from a genetic mutation which entails a higher likelihood ρ^H of becoming ill, and those who do not and who therefore enjoy a lower likelihood ρ^L of becoming ill, $\rho^L < \rho^H$. The probabilities of illness might be objective or subjective. Individuals do not know ex-ante whether they have a genetic mutation and hence form beliefs about their likelihood of being either type. Let π^H denote their belief that they belong to the high-risk group and $\pi^L = 1 - \pi^H$ their belief of belonging to the low-risk group. Figure 1 illustrates the information structure in the absence of information from a genetic test. Again, individuals might differ in their prior beliefs for instance due to different information regarding family medical history. As an example, for the BRCA genes discussed above, the relevant parameter values are $\{\pi^H = 0.001, \pi^L = 0.999, \rho^H = 0.14, \rho^L = 0.0125, \rho = 0.013\}$ for a woman with no family history of breast cancer, and $\{\pi^H = 0.065, \pi^L = 0.935, \rho^H = 0.295, \rho^L = 0.0111, \rho = 0.03\}$ for a woman if her mother and a sister had ovarian cancer before the age of 50 (see Hoy and Witt 2007).

[8]The consumption good might consist of several attributes. The analysis below does not require us to model this explicitly.

[9]The question of how changes in health status affect marginal utility of consumption is still not completely resolved. Viscusi and Evans (1990) use job injuries to infer that marginal utility drops due to decreased health, whereas Evans and Viscusi (1991) find that decreased health corresponds to a drop in income and does not affect the structural form of consumption utility. Important determinants of whether changes in health increase or decrease marginal utility of consumption seem to be the severity and the permanence of the health condition under consideration.

type / result	H	L
p	p_{pH}	p_{pL}
n	p_{nH}	p_{nL}
	1	1

Fig. 2 Test technology

Now consider that a genetic test is available that classifies individuals into high and low risks. We assume test technology to be exogenous. However, a test might not classify individuals perfectly meaning that the rate of false positives and false negatives might be strictly positive. The scientific reliability of a genetic test may be affected by a number of factors including sample contamination, incorrect laboratory testing procedures, mislabeling, misreporting, and transcription errors.[10] In the following we denote by p_{ij} the proportion of type j individuals ($j \in \{H, L\}$) who receive a positive or negative test result ($i \in \{p, n\}$). Hence, p_{pH}, which is referred to as the sensitivity of the test, is the proportion of correctly classified high-risk individuals who possess the genetic mutation; p_{nL}, which is referred to as the specificity of the test, is the proportion of correctly classified low-risk individuals who do not possess the genetic mutation; p_{pL} is the rate of false positives (i.e., the proportion of low-risk individuals who erroneously test positive); and p_{nH} is the fraction of false negatives (i.e., the proportion of high-risk individuals who erroneously test negative). Naturally, $p_{pH} + p_{nH} = 1$ and $p_{pL} + p_{nL} = 1$, which means that individuals of each type must test either positive or negative. Figure 2 summarizes the features of test technology.

Next, we are interested in how individuals perceive ex-ante the information structure induced by a genetic test. First, they should realize that they could test positive or negative. The perceived probability of receiving a specific test result depends, however, on the priors over $\{\rho^L, \rho^H\}$. More precisely, the perceived probability of testing positive is given by $\lambda^p \equiv \pi^H p_{pH} + \pi^L p_{pL}$, whereas the perceived probability of testing negative is $\lambda^n \equiv \pi^H p_{nH} + \pi^L p_{nL}$. Second, once a specific test result has been obtained the beliefs will be subject to updating. We use Bayes' rule to infer the new set of beliefs after testing. If individuals test positive, the perceived probability of belonging to the high-risk group should change to $\frac{\pi^H p_{pH}}{\lambda^p}$, which we denote by p^{Hp}. Thereby, the numerator represents the probability of being a high-risk type and receiving a positive test result and the denominator represents the probability of receiving a positive test result. Note that the new prior for ρ^H is larger than π^H if

[10] See "Essentially Yours: The Protection of Human Genetic Information in Australia" (ALRC Report 96), available at http://www.alrc.gov.au/publications/10-genetic-testing/reliability-genetic-testing, accessed February 25, 2014.

and only if $p_{pH} > p_{pL}$, i.e., if and only if test technology is such that high-risk types have a higher likelihood of receiving a positive test result than low-risk types. If this condition does not hold, then the test has zero information value. This is a minimum requirement on the discriminatory power of the test and can therefore be assumed to hold. Furthermore, if individuals test positive, the perceived probability of belonging to the low-risk group should change to $\frac{\pi^L p_{pL}}{\lambda^p} \equiv p^{Lp}$ which is strictly less than π^L. Looking at the distribution functions over $\{\rho^L, \rho^H\}$ it is easy to see that receiving a positive test result corresponds to a first-order stochastic improvement in the priors, i.e., it is now less likely that one is a low-risk individual. If individuals test negative, there will be a first-order stochastic deterioration in beliefs, i.e., it is now more likely that one is a low-risk individual. Conditional on receiving a negative result for the genetic test, the perceived probabilities of belonging to the high-risk group and to the low-risk group are given by $\frac{\pi^H p_{nH}}{\lambda^n} \equiv p^{Hn}$ and $\frac{\pi^L p_{nL}}{\lambda^n} \equiv p^{Ln}$, respectively. Hence, a positive or negative test result does not change the expected utility of either type, but the beliefs that one belongs to a specific risk group. The new information structure induced by the genetic test is illustrated in Fig. 3.

Comparing the information structure induced by a genetic test to the information structure without a test we see that a genetic test introduces another stage, the stage of possible test results. On each branch the (ex-ante perception of) ambiguity is changed by shifting priors towards the better scenario in case of a negative test result or towards the worse scenario in case of a positive test result. In this way, a genetic test introduces ambiguity through the uncertainty of test results. This is due to the fact that from an ex-ante perspective the individual does not know whether she will test positive or negative and hence a genetic test can be viewed as a lottery between an improvement in beliefs or a deterioration of beliefs. Although a person who does not submit to a genetic test may also be a high- or low-risk type (i.e., she may or may not have the disease gene), since this person does not have to face the chance of finding out whether her prospects are better or worse than she initially thinks, we treat this person as not experiencing ambiguity over test results. So a person can choose to stick with her (known) perceived distribution of illness types because she does

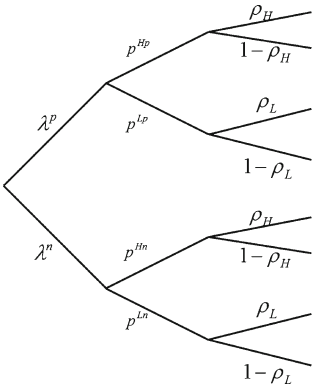

Fig. 3 Information structure induced by a genetic test

not want that perception to be challenged by a genetic test. Otherwise, if she submits to the test, she must experience the anticipation of receiving either good or bad news. Therefore, a genetic test represents a mean-preserving spread in ambiguous beliefs as defined by Snow (2010, p. 137). For the case of unproductive information accompanied by a change in the *general informativeness* of a test (see our Definition 1 in Section 3.2), our results represent a direct application of the theory and results developed by Snow (2010). Details are provided in the following sections.

In this paper we want to distinguish between individuals who are not affected by such uncertainty in the space of probabilities and those who care about uncertainty of probabilities. To operationalize this notion we follow Snow (2010) and apply the by now standard model (KMM) developed by Klibanoff et al. (2005).[11] Here, individuals form second-order probabilities to judge the likelihood of competing probabilistic scenarios. Given the information structure explained in Fig. 3, we incorporate individuals' fear of uncertain test results by utilizing λ^p and λ^n as second-order probabilities. Employing the Savage axioms in expected utility theory, these second-order or subjective probabilities would be treated as any other probabilities in a compound lottery and appear linearly in the expected utility formulation. The KMM formulation of ambiguity, on the other hand, treats these second-order probabilities differently with decision-makers forming a ϕ-weighted average about their prospects from a positive or negative test result. ϕ is typically assumed to be increasing because scenarios providing larger expected utility are better. Since individuals are uncertain about the probability of being on one or the other of these branches of the compound lottery, a mean-preserving spread in the expected utility values on these branches reduces utility for an individual who is averse to ambiguity. Thus, concavity of the ϕ-functional would reflect ambiguity aversion regarding uncertain test results, a linear ϕ represents ambiguity neutrality, and convexity of ϕ indicates ambiguity loving preferences.

3 Take-up under the expected utility hypothesis

3.1 Unproductive information

Having developed the outline of the model in the previous section we analyze the decision to undertake a genetic test from the perspective of an expected utility maximizer who is not affected by the potential presence of ambiguity and is, in this sense, ambiguity-neutral. We first analyze a scenario where decisions do not depend upon the information acquired and call information in this case unproductive, as it does not affect the actions taken by the decision-maker. Let individual endowment be state-dependent, i.e., the net present value generated by human capital might be different in the sick state than in the healthy state. We denote by c_h consumption in the healthy

[11] In the literature there is a variety of competing models to incorporate ambiguity-averse preferences, e.g., Gilboa and Schmeidler (1989) and Schmeidler (1989). We use the Klibanoff et al. (2005) specification due to its analytical appeal but briefly address robustness of our results to other specifications in the conclusion.

state and by c_s consumption in the sick state of nature.[12] Therefore, if individuals are uninformed their expected utility will be given by

$$\pi^H EU(\rho^H) + \pi^L EU(\rho^L), \tag{1}$$

where $EU(\rho^i)$ denotes expected utility when being type i, i.e., $EU(\rho^i) = \rho^i u_s(c_s) + (1 - \rho^i)u_h(c_h)$. If now individuals consider taking a genetic test, the ex-ante information structure is given by Fig. 3, i.e., individuals face a perceived chance of λ^p of receiving bad news and a chance of λ^n of receiving good news and conditional on test results the priors shift as described above. Hence, from an ex-ante perspective the expected utility from taking the test is

$$\lambda^p \left(p^{Hp} EU(\rho^H) + p^{Lp} EU(\rho^L) \right) + \lambda^n \left(p^{Hn} EU(\rho^H) + p^{Ln} EU(\rho^L) \right).$$

Since expected utility is linear in probabilities and a genetic test simply represents a mean-preserving spread in the probability of being either type H or L, this resolves to (1) and is therefore identical to the expected utility of not taking the test.

Note that this holds irrespective of the test technology. This is due to the fact that the upward and downward shift in the priors induced by positive and negative test results are such that the average belief of belonging to a specific risk type are unaltered, formally $\lambda^p p^{Hp} + \lambda^n p^{Hn} = \pi^H$ and likewise for π^L. This is because high specificity of the test is "bad" in case of a positive test result, as your belief of belonging to the high-risk group has to be increased, whereas it is "good" in case of a negative test result, because the chance of being a high-risk type that has simply been misclassified is very low, so your belief of belonging to the high-risk group drops. Those two effects are just offsetting each other and the same holds true for the sensitivity of the test. We collect the above results in the following proposition.

Proposition 1 *Under EUT the value of unproductive genetic information is zero, independent of sensitivity and specificity of the test.*

On that account, at least there is no reason to see low take-up rates on average. One could, however, argue that in reality at least a small cost for taking a genetic test seems plausible which would in this set-up deter testing and would be consistent with low take-up rates. This might apply to diseases like Huntington's disease where there is basically no cure available and little can be done in medical terms once the information is obtained. We believe, however, that even when medical treatments are irrelevant other non-medical choices like savings decisions, occupational decisions or family planning will depend on the information which will be dealt with in the next subsection.

3.2 Productive information

Let us now consider a situation where individuals are not completely helpless regarding the information received from a genetic test, but can take actions conditional

[12] As said before, the consumption good might be multidimensional.

upon the information acquired to increase their expected utility. In this sense information is productive, as it affects decisions taken by the individual. Examples would be diseases where prevention matters or where individuals are likely to adjust their behavior in terms of surveillance to the information obtained from a genetic test. In addition, as mentioned in the introduction, in countries where insurance companies are denied access to genetic information we should likely see opportunistic behavior in terms of insurance purchasing based upon information obtained from testing. We incorporate this into the model by assuming that X denotes the set of potential life plans available to the individual and that $x \in X$ denotes a particular life plan. Individuals will choose the life plan which maximizes their expected utility given the information available. Therefore, we denote by $EU(\rho^i, x)$ the expected utility of risk type i if life plan x is selected.[13]

If no genetic test is undertaken and the individual evaluates her situation according to the initial priors, then a life plan will be chosen according to

$$\max_{x \in X} \left\{ \pi^H EU(\rho^H, x) + \pi^L EU(\rho^L, x) \right\}.$$

Let x^u denote the optimal choice of an uninformed individual (i.e., the value of x that satisfies the preceding maximization problem). If the individual, however, contemplates whether to undertake a genetic test or not, she will be well aware of the fact that the information received from the test is likely to impose the necessity (or option) of adjusting her chosen life plan. Technically, the choice of $x \in X$ will be conditional on the information acquired from the test, i.e., the individual solves

$$\max_{x \in X} \left\{ p^{Hi} EU(\rho^H, x) + p^{Li} EU(\rho^L, x) \right\},$$

when receiving test result $i \in \{p, n\}$ yielding optimal decision x^i.

Hence, ex-ante utility from undertaking a genetic test will be given by

$$\lambda^p \left(p^{Hp} EU(\rho^H, x^p) + p^{Lp} EU(\rho^L, x^p) \right) + \lambda^n \left(p^{Hn} EU(\rho^H, x^n) + p^{Ln} EU(\rho^L, x^n) \right). \tag{2}$$

From the definition of an optimal choice it is clear that individuals are weakly better off if they decide to take action x^p rather than x^u when testing positive and taking x^n rather than x^u when testing negative. The inequality is strict for situations where a life plan conditional on the information acquired from a genetic test is utility-enhancing, e.g., prevention and/or surveillance are effective. In general, the optimal decision needs to change only for the case of one of the test results in order that the information

[13] This modeling is very flexible, as it is comprised of decisions that affect the consumption vector and/or the illness probabilities. Hence, any sort of prevention or surveillance decision is captured within our model, as are savings or insurance decisions.

has positive value ex-ante and increases expected utility. Under such conditions, this demonstrates that the ex-ante expected utility from a genetic test exceeds

$$\begin{aligned}&\lambda^p\left(p^{Hp}EU(\rho^H,x^u)+p^{Lp}EU(\rho^L,x^u)\right)\\&\quad+\lambda^n\left(p^{Hn}EU(\rho^H,x^u)+p^{Ln}EU(\rho^L,x^u)\right)\\&=\pi^HEU(\rho^H,x^u)+\pi^LEU(\rho^L,x^u).\end{aligned}$$

It becomes evident that as soon as information obtained from a genetic test facilitates better decision-making, the value of costless genetic testing should be strictly positive meaning that a rational decision-maker would always take a costless genetic test.[14] Note that this does not depend on test technology, and therefore level of accuracy of the test, and hence information, even if it might not be accurate in all cases, is still valuable to an expected utility maximizer. This is in sharp contrast to low take-up rates.[15]

Let us now turn to the question how changes in test technology affect the value of genetic information in the EUT case. As explained in the Appendix, looking at the rate of false negatives or looking at the fraction of high risks who test positive (i.e., sensitivity of the test) is equivalent. Let the parameters δ and ϵ represent (marginal) changes to the sensitivity and specificity of the test (respectively). Let V be shorthand for (2) and $\overline{p}_{pH} = p_{pH} + \delta$. Then, utilizing the marginal effects derived in the Appendix, we obtain[16]

$$\frac{dV}{d\delta}=\pi^H\Big[EU(\rho^H,\overline{x}^p)-EU(\rho^H,\overline{x}^n)\Big]+\left.\frac{\partial V}{\partial\delta}\right|_{\overline{p}_{pH}\text{ fixed}}.$$

[14]The weak positivity of the value of information was already noted by Savage (1954), as "the person is free to ignore the observation. That obvious fact is the theory's expression of the commonplace that knowledge is not disadvantageous." Gollier (2001) provides a comprehensive summary of the theory of the value of information under the expected utility hypothesis. It should also be acknowledged that in the context of health decisions, people often appear to choose non-optimal behaviors and so improved information about disease likelihood may not lead to improved decision-making for reasons outside of our model.

[15]Still, people argue that obtaining a bad test result might affect an individual's expected utility by deteriorating the choice set, i.e., $X^H \subset X^0$. This happens for instance when insurance companies are allowed to use genetic information for rate-making purposes. Then the rates for health and/or life insurance coverage will be dramatically higher for tested individuals with an unfavorable result and, in this sense, their market opportunities are worsened. If this is the case, one cannot simply ignore the information as actions taken when uninformed might no longer be available. However, as outlined in the introduction, we observe low take-up rates even in countries where the use of genetic information by insurance companies and employers is banned and therefore we do not focus on this aspect.

[16]The first component can be obtained easiest by first simplifying (2) and then taking the derivative. Alternatively, one can first take derivatives to get

$$\begin{aligned}&\pi^H\left(\overline{p}^{Hp}EU(\rho^H,\overline{x}^p)+\overline{p}^{Lp}EU(\rho^L,\overline{x}^p)\right)-\pi^H\left(\overline{p}^{Hn}EU(\rho^H,\overline{x}^n)+\overline{p}^{Ln}EU(\rho^L,\overline{x}^n)\right)\\&\quad+\overline{\lambda}^p\frac{\pi^H\pi^Lp_{pL}}{(\overline{\lambda}^p)^2}\Big[EU(\rho^H,\overline{x}^p)-EU(\rho^L,\overline{x}^p)\Big]-\overline{\lambda}^n\frac{\pi^H\pi^Lp_{nL}}{(\overline{\lambda}^n)^2}\Big[EU(\rho^H,\overline{x}^n)-EU(\rho^L,\overline{x}^n)\Big],\end{aligned}$$

which simplifies to the first summand.

The first summand contains the effect of increased sensitivity on the probabilities of testing positive and negative and on the priors conditional on a given test result. It is non-negative as long as $\overline{p}_{pH} \geq \overline{p}_{nH}$ due to the fact that there is more weight on $EU(\rho^H, x)$ and hence the optimal decision will be closer to the decision maximizing $EU(\rho^H, x)$. The second summand reflects the effect of higher sensitivity on decision-making.[17] Note that

$$\pi^H \overline{p}_{pH} EU(\rho^H, \overline{x}^p) + \pi^L p_{pL} EU(\rho^L, \overline{x}^p) \geq \pi^H \overline{p}_{pH} EU(\rho^H, x^p) + \pi^L p_{pL} EU(\rho^L, x^p),$$

due to the optimality of $\overline{x}^p$. This can be rearranged to

$$\pi^H \overline{p}_{pH} \left[EU(\rho^H, \overline{x}^p) - EU(\rho^H, x^p) \right] + \pi^L p_{pL} \left[EU(\rho^L, \overline{x}^p) - EU(\rho^L, x^p) \right] \geq 0.$$

The same holds true for the case of a negative test result and therefore the second effect is positive as well. Particularly, $\frac{dV}{d\delta}\big|_{\delta=0} \geq 0$, so higher sensitivity leads to more appropriate decision-making and this is what improves expected utility in the case of illness which is more likely to be indicated by the test. A similar argument holds for the specificity of the test.

Sensitivity and specificity of genetic tests is one way of measuring accuracy. However, tests will hardly be comparable in terms of one isolated parameter only. Therefore, we define another method for comparing genetic tests which will prove to be useful when looking at the value of genetic information under ambiguity aversion.

Definition 1 A test is generally more informative than another test if the perceived probability of testing positive or negative is unchanged, but beliefs are shifted more.

It is easy to see that a generally more informative test represents a mean-preserving spread in the subjective probability distribution over disease probabilities. A change in one of either the sensitivity or specificity of the test would alter the perceived probability of testing either positive or negative and so these are distinct definitions of what one means by a change in the accuracy or informativeness of a test.[18]

To formalize this notion of generally more informative tests, we can employ directional derivatives. We assume V to be a function of sensitivity and specificity, i.e., $V = V(p_{pH}, p_{nL})$. Then, the value of genetic information for a generally more informative test can be determined by looking at the directional derivative along the vector $\nu \equiv (\pi^L, \pi^H)$ in the (p_{pH}, p_{nL})-plane. Applying this directional

[17] In the following analysis, we will use the operator $\frac{\partial}{\partial \delta}$ to describe the decision-making value due to marginal increases in sensitivity and the operator $\frac{\partial}{\partial \epsilon}$ for the decision-making value due to marginal increases in specificity.

[18] An example for a combined improvement of sensitivity and specificity is given by the move from linkage analysis to the identification of the DNA sequence associated with a disease gene (see Farrer et al. 1988; Meiser and Dunn 2000). Initial genetic testing for HD used linkage analysis, which resulted in a sensitivity of approximately 90 to 95% and a specificity of 90% or more. This is due to the fact that linkage analysis is based on the presence of markers and therefore not 100% accurate. Modern direct detection methods typically provide sensitivity and specificity of nearly 100%. Also note that in the context of multifactorial genetic diseases tests are continually refined because there is ongoing discovery of interacting genes.

derivative approach yields that the probabilities of testing positive and negative stay constant; i.e.,

$$\partial_{\mathrm{v}}\lambda^{p} = \partial_{\mathrm{v}}\lambda^{n} = 0.$$

It also follows that an individual receiving a positive (negative) test result believes "more strongly" to be an H-type (L-type); i.e.,

$$\partial_{\mathrm{v}} p^{Hp} = \frac{\pi^{H}\pi^{L}}{\lambda^{p}} > 0,\ \partial_{\mathrm{v}} p^{Lp} = -\frac{\pi^{H}\pi^{L}}{\lambda^{p}} < 0,$$

and

$$\partial_{\mathrm{v}} p^{Ln} = \frac{\pi^{H}\pi^{L}}{\lambda^{n}} > 0,\ \partial_{\mathrm{v}} p^{Hn} = -\frac{\pi^{H}\pi^{L}}{\lambda^{n}} < 0.$$

For the value of information this yields

$$\begin{aligned}\partial_{\mathrm{v}} V &= \pi^{L}\frac{dV}{dp_{pH}} + \pi^{H}\frac{dV}{dp_{nL}} = \\ &= \pi^{H}\pi^{L}\left[EU(\rho^{H}, x^{p}) - EU(\rho^{L}, x^{p}) + EU(\rho^{L}, x^{n}) - EU(\rho^{H}, x^{n})\right] \\ &\quad + \pi^{L}\frac{\partial V}{\partial \delta} + \pi^{H}\frac{\partial V}{\partial \epsilon},\end{aligned}$$

which is, of course, still positive. Generally more informative tests increase the value of information. Let us summarize the above discussion in the following proposition.

Proposition 2 *Under EUT the value of productive genetic information is positive. It is larger for tests with higher sensitivity or specificity and is also larger for generally more informative tests.*

In summary, expected utility maximizers should be indifferent about obtaining unproductive genetic information or not, but always demand productive genetic information when it is costless. They benefit from increased accuracy of the test, because the decision-making value is higher for more accurate tests. Hence, in light of expected utility theory, low take-up rates are not to be expected.

4 Ambiguity aversion and genetic information

4.1 Unproductive information

Let us now turn to a situation where the individual might be negatively affected by the uncertainty of test results. As outlined before we will utilize ambiguity preferences to incorporate this sensation and compare the information structures with and without a genetic test. Remember that Fig. 3 was obtained from Fig. 1 by applying Bayes' rule to the priors over types to reflect the inflow of new information from test results. Viscusi and Magat (1992) document that in ambiguous situations where learning takes place, individuals might depart from Bayesian updating and display so-called ambiguous belief aversion. Gilboa and Schmeidler (1993) and Pires (2002) study different updating rules and give axiomatizations of the rule that requires the updating

of all priors by Bayes' rule. To achieve comparability across decision-makers, we assume that under EUT and KMM priors will be updated according to Bayes' rule and hence abstract from heterogeneity regarding the updating rule and focus on the evaluation of the resulting information structure.

Consider the situation where there is no possibility to react to the information acquired. We now include the possibility of ambiguity preferences, captured by the ambiguity functional ϕ as described above. Again consider an individual who is not tested and does not consider taking a genetic test. Hence, uncertainty of test results does not matter for this individual and her welfare will be given by

$$\phi\left(\pi^H EU(\rho^H) + \pi^L EU(\rho^L)\right).$$

In utility terms this corresponds to $\phi^{-1}\left(\phi\left(\pi^H EU(\rho^H) + \pi^L EU(\rho^L)\right)\right)$, but we will rather work on the ϕ-scale which preserves rankings due to the fact that ϕ is increasing.

If individuals consider taking a genetic test, they face the information structure as described in Fig. 3. Therefore, the uncertainty of the test result matters for ambiguity-averse agents. Ex-ante expected welfare from taking the test is given by

$$\begin{aligned}
&\lambda^p \phi\left(p^{Hp} EU(\rho^H) + p^{Lp} EU(\rho^L)\right) + \lambda^n \phi\left(p^{Hn} EU(\rho^H) + p^{Ln} EU(\rho^L)\right) \\
&\quad < \phi\left(\lambda^p\left(p^{Hp} EU(\rho^H) + p^{Lp} EU(\rho^L)\right) + \lambda^n\left(p^{Hn} EU(\rho^H) + p^{Ln} EU(\rho^L)\right)\right) \\
&\quad = \phi\left(\pi^H EU(\rho^H) + \pi^L EU(\rho^L)\right),
\end{aligned}$$

due to concavity of the ambiguity function ϕ. This reflects the fact that ambiguity-averse decision-makers dislike mean-preserving spreads in the space of probabilities. Therefore, from an ex-ante perspective, ambiguity aversion discourages genetic testing. Furthermore, this effect is more pronounced with stronger ambiguity aversion, i.e., for more ambiguity-averse decision-makers the value of unproductive genetic information is more negative. This is due to the fact that the spread in priors is increasingly painful. In this sense, ambiguity-aversion represents one avenue to explain low take-up for genetic tests.

Let us again study the effect of test technology on the value of genetic information. Let $V^i \equiv p^{Hi} EU(\rho^H) + p^{Li} EU(\rho^L),\ i \in \{p, n\}$, then the ex-ante value of testing is $V \equiv \lambda^p \phi(V^p) + \lambda^n \phi(V^n)$. As above we study marginal variations in sensitivity. It holds that

$$\begin{aligned}
\frac{dV}{dp_{pH}} &= \pi^H\left(\phi(V^p) - \phi(V^n)\right) + \pi^H \pi^L\left[EU(\rho^H) - EU(\rho^L)\right] \\
&\quad \times \left(\frac{1 - p_{nL}}{\lambda^p}\phi'(V^p) - \frac{p_{nL}}{\lambda^n}\phi'(V^n)\right).
\end{aligned}$$

The first summand is negative due to the fact that the situation with a positive test result is worse than with a negative one. This reflects the fact that with increased sensitivity the overall perceived probability of testing positive increases which is undesirable. The second summand describes the effect of increased sensitivity on how the priors over risk types will be adjusted. In case of a positive test result, it

becomes harder to believe that you are low risk due to high sensitivity of the test. This is bad news and hence this part of the expression is negative. However, with a negative result you should increase your belief in being low risk which is good news (i.e., good news that is even better news if we test negative compared to the less sensitive test) and makes this part of the expression positive. The overall effect is indeterminate and also depends on the effect on the marginal evaluation of the two outcomes. Note that for linear ϕ we are back in the EUT-case and the marginal effect of sensitivity is zero, as the value of unproductive genetic information is uniformly zero. A similar reasoning holds for the specificity of the test.

Here, the concept of greater general informativeness (Definition 1) proves to be useful. As before, we can look at the directional derivative of the value of genetic information into the direction of $\mathrm{v} = (\pi^L, \pi^H)$ in the (p_{pH}, p_{Ln})-plane. It is given by

$$\begin{aligned}
\partial_{\mathrm{v}} V &= \pi^L \frac{dV}{dp_{pH}} + \pi^H \frac{dV}{dp_{nL}} \\
&= \pi^H \pi^L \left(\phi(V^p) - \phi(V^n)\right) + \pi^H \pi^L \left(\phi(V^n) - \phi(V^p)\right) \\
&\quad + \pi^H \pi^L \left[EU(\rho^H) - EU(\rho^L)\right] \left(\pi^L \frac{1 - p_{nL}}{\lambda^p} \phi'(V^p) - \pi^L \frac{p_{nL}}{\lambda^n} \phi'(V^n)\right) \\
&\quad + \pi^H \pi^L \left[EU(\rho^H) - EU(\rho^L)\right] \left(\pi^H \frac{p_{pH}}{\lambda^p} \phi'(V^p) - \pi^H \frac{1 - p_{pH}}{\lambda^n} \phi'(V^n)\right) \\
&= \pi^H \pi^L \left[EU(\rho^H) - EU(\rho^L)\right] \left(\phi'(V^p) - \phi'(V^n)\right),
\end{aligned}$$

which is negative due to the fact that utility for high risks is smaller than for low risks and that prospects when testing positive are worse than when testing negative and ϕ is concave. In this sense the value of unproductive genetic information is more negative for generally more informative tests as the first-order stochastic shifts in the beliefs induced by the test results are stronger. This is detrimental to individuals with KMM-preferences. The results are summarized in the following proposition.

Proposition 3 *With ambiguity aversion, the value of unproductive genetic information is negative. The effects of increased sensitivity or specificity of the test are indeterminate, but for generally more informative tests the value becomes more negative.*

It is worth pointing out that the last part of this proposition represents a direct application of Theorems 1 and 2 of Snow (2010, p. 138). This follows because a genetic test that is generally more informative than another represents a mean-preserving spread in the subjective probability distribution over probabilities of disease consistent with the set-up of Snow (2010). In our model, the case of unproductive information relates to situations in which there is no incentive to change one's decision as a result of a genetic test result, while in Snow (2010) individuals choose decisions in advance of any information received and so information in his model cannot be productive in the sense we use here. However, in Snow (2010), an individual's action or decision can depend on the degree of ambiguity and degree

of ambiguity aversion. The innovations in our paper stem from the decision-making context, the comparison of the case of unproductive and productive information in the context of genetic tests, and the separate treatment of the dimensions of informational accuracy described by the sensitivity and specificity (or equivalently the false negatives and false positives) of genetic tests.

4.2 Productive information

Let us now assume again that individuals can make better decisions if they acquire information and can better design their life plan conditional on the information about illness propensities. As before, let $x^i \in X$ be the choice of an individual who is uninformed, has obtained a positive test result or a negative test result, respectively, $i \in \{u, p, n\}$. Due to the fact that ϕ is increasing, the $x^i \in X$ also maximizes ϕ of the maximand. Notice that x will be implemented after the resolution of ambiguity, i.e., after information from a genetic test has been obtained. As noted above, this is one important respect in which we differ from Snow (2010) who assumes that an action is taken before the ambiguity is resolved. In a situation of genetic testing it seems, however, appropriate to incorporate actions that depend on the individual's level of information. This is motivated by the fact that individuals are likely to evaluate their opportunities when tested positive ("What can I do in case of bad news?") before actually deciding to take a test and this will, of course, affect how they value the genetic information contained in the test. We apply the ϕ-functional to formalize the notion that individuals face uncertainty about their actual test result ex-ante. With this in mind, expected welfare from taking a test is given by

$$\begin{aligned}&\lambda^p \phi\left(p^{Hp} EU\left(\rho^H, x^p\right) + p^{Lp} EU\left(\rho^L, x^p\right)\right)\\ &\quad + \lambda^n \phi\left(p^{Hn} EU\left(\rho^H, x^n\right) + p^{Ln} EU\left(\rho^L, x^n\right)\right).\end{aligned}$$

As before this exceeds

$$\begin{aligned}&\lambda^p \phi\left(p^{Hp} EU\left(\rho^H, x^u\right) + p^{Lp} EU\left(\rho^L, x^u\right)\right)\\ &\quad + \lambda^n \phi\left(p^{Hn} EU\left(\rho^H, x^u\right) + p^{Ln} EU\left(\rho^L, x^u\right)\right),\end{aligned}$$

due to the optimality of x^p and x^n. However, the last expression is smaller than the individual's welfare without taking a test due to concavity of ϕ. In other words, an ambiguity-averse individual may not wish to submit to a genetic test even if information is potentially productive. Still, if an ambiguity-averse person does obtain a genetic test, she is of course better off if she adjusts her behavior in an optimal fashion according to the test result that she receives.

In this situation, one cannot unambiguously conclude whether the informational value from testing is positive or negative. On the one hand, the presence of ambiguity aversion makes genetic testing unattractive due to the uncertainty of test results. Having a lottery between a deterioration of beliefs and an improvement of beliefs is detrimental in terms of ex-ante expected welfare for an ambiguity-averse decision-maker. However, information facilitates better decision-making and if at least one possibility promises a strictly larger expected utility than in the no-information case,

this is what makes genetic testing potentially attractive. Therefore, in the most general case of ambiguity aversion and endogenous action that affects individual well-being and depends on the information at hand, we conclude that the interaction of the set of actions X, their effect in terms of expected utility of the agent, and the presence of ambiguity aversion together determine whether the informational value from genetic testing is positive or negative.

The effect of increased sensitivity or increased specificity consists now of four components. As before, $V^i = p^{Hi} EU(\rho^H, x^i) + p^{Li} EU(\rho^L, x^i),\ i \in \{p, n\}$, and the ex-ante value of testing is $V = \lambda^p \phi(V^p) + \lambda^n \phi(V^n)$. Then,

$$\begin{aligned}\frac{dV}{dp_{pH}} &= \pi^H \left(\phi(V^p) - \phi(V^n)\right) + \pi^H \pi^L \frac{1 - p_{nL}}{\lambda^p} \phi'(V^p) \\ &\quad \times \left[EU\left(\rho^H, x^p\right) - EU\left(\rho^L, x^p\right)\right] \\ &\quad + \pi^H \pi^L \frac{p_{nL}}{\lambda^n} \phi'(V^n) \left[EU\left(\rho^L, x^n\right) - EU\left(\rho^H, x^n\right)\right] + \frac{\partial V}{\partial \delta}.\end{aligned}$$

The first summand is negative, as positive test results are "bad news". The second and third describe how priors are adjusted due to increased sensitivity of the test. The second term is negative and the third one is positive. Lastly, higher sensitivity implies a larger decision-making value of genetic information, i.e., the last summand is positive again. This can be shown analogously as in Section 3.2. The overall effect is indeterminate. However, we can again employ the concept of generally more informative tests as defined above. After some algebra, one obtains

$$\begin{aligned}\partial_v V &= \pi^H \pi^L \Bigg(\phi'(V^p) \underbrace{\left[EU(\rho^H, x^p) - EU\left(\rho^L, x^p\right)\right]}_{<0} \\ &\qquad + \phi'(V^n) \underbrace{\left[EU\left(\rho^L, x^n\right) - EU\left(\rho^H, x^n\right)\right]}_{>0} \Bigg) \\ &\quad + \pi^L \frac{\partial V}{\partial \delta} + \pi^H \frac{\partial V}{\partial \epsilon}.\end{aligned}$$

Now, $V^p < V^n$ and therefore $\phi'(V^p) > \phi'(V^n)$, as a positive test result always implies that your beliefs shift more towards being high risk. However,

$$EU(\rho^H, x^p) - EU\left(\rho^H, x^n\right) + EU\left(\rho^L, x^n\right) - EU\left(\rho^L, x^p\right) > 0,$$

as x^p is the optimal decision when more weight is placed on the high-risk utility, whereas x^n is the optimal decision when more weight is placed on the low-risk utility. Hence, the first summand is indeterminate. Note, however, that under sufficient ambiguity aversion, so with ϕ sufficiently concave, the first summand will be negative as the spread in the probabilities is increasingly painful and not outweighed by the gains through better decision-making.

When evaluated at $(p_{pH}, p_{nL}) = (0.5, 0.5)$ the first summand is zero due to the fact that for uninformative tests $p^{Hp} = p^{Hn} = \pi^H$ and $p^{Lp} = p^{Ln} = \pi^L$, and hence $x^p = x^n$ and $V^p = V^n$. In this case, $\partial_v V$ is non-negative implying that agents

always want to obtain genetic tests that contain "a little bit" of information. This is due to the fact that ambiguity aversion as modeled within the KMM framework is a second-order phenomenon, so for small levels of ambiguity individuals behave as if ambiguity-neutral.

Proposition 4 *With ambiguity aversion, the value of productive genetic information is indeterminate. It reflects the trade-off between aversion against ambiguity and better informed decision-making. The effects of increased sensitivity or specificity of the test are indeterminate. Generally more informative tests trade-off increased ambiguity against more suitable decision-making and are valued less with sufficiently strong ambiguity aversion. Tests containing a little bit of information are always taken.*

In this sense, ambiguity aversion represents one rationale to explain low take-up rates of genetic tests.

5 Conclusion

Even when the private cost for genetic tests is zero, take-up rates are often very low. Given the fact that an expected utility maximizing decision-maker attaches, at worst, zero private value to genetic information (i.e., when it is unproductive in that no possible result has any effect on her optimal decision) and positive private value otherwise, this is surprising. In most instances, even if there are no preventive measures available against the disease, it is likely that people would adjust their life plans according to some results derived from a genetic test (e.g., in terms of savings decisions, occupational training, whether to have children etc.). In countries where insurers are denied access to information received from genetic testing, people are also likely to opportunistically adjust their insurance consumption. Hence, genetic information should be productive and therefore low take-up rates may indeed seem very surprising.

We demonstrate that if individuals display ambiguity aversion over test results, then one can rationalize this observation within the context of rational decision-making. In cases where genetic information is unproductive, an ambiguity-averse decision-maker will never take a costless genetic test. This follows because, from an ex-ante perspective, the test generates a lottery between a first-order stochastic improvement of beliefs and a first-order stochastic deterioration of beliefs. Even if information is productive, the uncertainty of test results attached to genetic testing is unfavorable for the individual and might dominate utility gains from better informed decisions. This downside is more pronounced with stronger ambiguity aversion and with more precise tests (conditional on equivalent productiveness of a test). Paradoxically, the more accurate the test the more negatively is individual ex-ante valuation of the test affected by the ambiguity which is in line with empirical observations. In a similar spirit, individuals will always take a genetic test containing "a little bit" of information as in this case the utility-enhancing informed decision-making component prevails.

In this paper, we model ambiguity attitudes utilizing the smooth model developed by Klibanoff et al. (2005). However, the basic intuition carries over to other decision-making models under ambiguity. Under maxmin expected utility as proposed by Gilboa and Schmeidler (1989) individuals fear the ambiguity introduced by a test due to the fact that in case of a positive test result they increase their belief in being high risk. This is again detrimental to gains in expected utility from better decision-making. Under α maxmin expected utility as proposed by Ghirardato et al. (2004) sufficient pessimism again creates the result that the ex-ante uncertainty of test results can discourage testing. In all cases, the basic intuition remains unchanged: Taking a genetic test corresponds to a lottery between a first-order stochastic improvement of beliefs and a first-order stochastic deterioration of beliefs from an ex-ante perspective, and so introduces ambiguity. Under ambiguity aversion decision-makers exhibit a preference for sticking to their known perceived distribution over risk types and do not want it to be challenged by a genetic test.

The conclusions to draw from that are at least twofold. If we believe that genetic information is likely to improve or even to revolutionize the practice of medicine, one must understand how individuals assess the value of genetic information. Genetic information is useless if it is simply not obtained by individuals due to behavioral reasons. Second, improving on accuracy of tests alone is not sufficient or might even be counter-productive due to the trade-off between ambiguity aversion and the utility gains from better decision-making. With more accurate tests, the ambiguity of taking a test is more pronounced and decreases consumer ex-ante valuation of taking a test. Hence, if there is little to do with the information acquired, i.e., utility conditional on any given test result cannot be increased by much, the fear of ambiguity predominates and the test will not be taken. In this respect it is also important that individuals do not underestimate the productivity of genetic information. One consideration for policy is to offer genetic counseling which informs potential test-takers about the productivity of genetic information and may reduce the sense of ambiguity that can arise from lack of knowledge of the process of genetics. If one believes that ambiguity aversion is a psychological tendency that inhibits "good" decision-making and that expected utility calculations represent the appropriate "normative" framework for assessing individual (and for that matter societal) well-being, then other sorts of policy intervention would be in order. In the real world of (financially) costly genetic tests, an obvious intervention would be to subsidize genetic tests. This may tip the balance of individuals' perceived (overall) costs of genetic testing—including disutility from aversion to ambiguity created by the tests—and perceived benefits of improved ex-post decision-making based on better information about risk of disease.

We believe our paper is, of course, not the last word on explaining why people seem to be surprisingly reticent to obtain genetic tests. There is an extensive literature from clinical researchers and a nascent economic literature directed at understanding individual attitudes and proclivities in regard to taking genetic tests. However, at the present time there is little structure in the former literature and few results in the latter literature. We hope that our modeling efforts in this paper will provide some impetus to develop a more systematic approach to this important research area.

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Appendix: Changes in predictive power of the test

The predictive power of a genetic test is improved by either an increase in the sensitivity (p_{pH}) or the specificity (p_{nL}) of the test; that is, the greater rate at which "correct classifications" are made. This is equivalent to reductions in the false negatives or false positives of the test, respectively. If we write the rate of false negatives and positives as ε_{fn}, and ε_{fp}, respectively, then it follows that $p_{pH} = 1 - \varepsilon_{fn}$, $p_{nH} = \varepsilon_{fn}$, $p_{pL} = \varepsilon_{fp}$, $p_{nL} = 1 - \varepsilon_{fp}$. By substituting $p_{nH} = 1 - p_{pH}$ and $p_{pL} = 1 - p_{nL}$, we obtain the following:

$$\lambda^p = \pi^H p_{pH} + \pi^L (1 - p_{nL}),$$
$$\lambda^n = \pi^H (1 - p_{pH}) + \pi^L p_{nL}.$$

We will use the following results to derive our propositions.

$$\frac{\partial \lambda^p}{\partial p_{pH}} = \pi^H, \ \frac{\partial \lambda^p}{\partial p_{nL}} = -\pi^L, \ \frac{\partial \lambda^n}{\partial p_{pH}} = -\pi^H, \ \frac{\partial \lambda^n}{\partial p_{nL}} = \pi^L.$$

Using the above, and the formulae for p^{ji} (repeated below), we get the following derivatives.

$$p^{Hp} = \frac{\pi^H p_{pH}}{\lambda^p}, \ p^{Lp} = \frac{\pi^L p_{pL}}{\lambda^p}, \ p^{Hn} = \frac{\pi^H p_{nH}}{\lambda^n}, \ p^{Ln} = \frac{\pi^L p_{nL}}{\lambda^n}.$$

So, for example, writing $p^{Hp} = \pi^H p_{pH} (\lambda^p)^{-1}$, we have

$$\begin{aligned} \frac{\partial p^{Hp}}{\partial p_{pH}} &= \pi^H (\lambda^p)^{-1} + \pi^H p_{pH} (-1)(\lambda^p)^{-2} \pi^H \\ &= \frac{\pi^H}{\lambda^p} - \frac{(\pi^H)^2 p_{pH}}{(\lambda^p)^2} = \frac{\pi^H (\lambda^p - \pi^H p_{pH})}{(\lambda^p)^2} = \frac{\pi^H \pi^L (1 - p_{nL})}{(\lambda^p)^2}. \end{aligned}$$

The remaining relevant derivatives are:

$$\begin{aligned}
\frac{\partial p^{Hp}}{\partial p_{nL}} &= \frac{\pi^H \pi^L p_{pH}}{(\lambda^p)^2},\\
\frac{\partial p^{Lp}}{\partial p_{pH}} &= -\frac{\pi^H \pi^L (1-p_{nL})}{(\lambda^p)^2},\\
\frac{\partial p^{Lp}}{\partial p_{nL}} &= -\frac{\pi^H \pi^L p_{pH}}{(\lambda^p)^2},\\
\frac{\partial p^{Ln}}{\partial p_{nL}} &= \frac{\pi^H \pi^L (1-p_{pH})}{(\lambda^n)^2},\\
\frac{\partial p^{Ln}}{\partial p_{pH}} &= \frac{\pi^H \pi^L (p_{nL})}{(\lambda^n)^2},\\
\frac{\partial p^{Hn}}{\partial p_{nL}} &= -\frac{\pi^H \pi^L (1-p_{pH})}{(\lambda^n)^2},\\
\frac{\partial p^{Hn}}{\partial p_{pH}} &= -\frac{\pi^H \pi^L p_{nL}}{(\lambda^n)^2}.
\end{aligned}$$

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