

Optimal Expectations and Limited Medical Testing: Evidence from Huntington Disease[†]

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We use novel data to study genetic testing among individuals at risk for Huntington disease (HD), a hereditary disease with limited life expectancy. Although genetic testing is perfectly predictive and carries little economic cost, presymptomatic testing is rare. Testing rates increase with increases in ex ante risk of having HD. Untested individuals express optimistic beliefs about their health and make decisions (e.g., retirement) as if they do not have HD, even though individuals with confirmed HD behave differently. We suggest that these facts can be reconciled by an optimal expectations model (Brunnermeier and Parker 2005). (JEL D84, I12)

Huntington disease (HD) is a degenerative neurological disorder with onset around age 40, a life expectancy of around 60 and a healthy life expectancy of 10 years fewer than that. HD is caused by an inherited expansion in the Huntington gene. Individuals with one parent with HD have a 50 percent chance of inheriting the expanded copy of the gene and developing the disease. Since the early 1990s a genetic test has been available. This blood DNA test can provide at-risk individuals with certainty about whether they will develop HD. This test would appear to have significant value; a variety of life choices (childbearing, retirement, education, participation in clinical research) are likely to be affected by HD status. Although HD is a rare disease, genetic testing for other conditions is becoming increasingly common.

In this article we explore the decision to undergo genetic testing. Our first contribution is to document a number of facts about genetic risk, behavior, and genetic testing using a rich dataset of individuals at risk for HD. Our data cover 1,001 at-risk individuals who had chosen not to undergo genetic testing prior to enrollment into an observational study. Over a ten-year period we observe subsequent decisions by some research participants to pursue genetic testing for HD, yearly information on subjective and investigator-measured probability of carrying the HD expansion, and information on a variety of life events.

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We begin by documenting low rates of genetic testing in our sample: fewer than 10 percent of individuals pursue predictive testing during the study. This echoes what has been seen in other contexts (Kőszegi 2003; Lerman et al. 1996; Thornton 2008) and in other data on this population (Shoulson and Young 2011).

Although predictive testing rates are low overall, we find that the probability of undergoing genetic testing is increasing with ex ante risk of carrying the HD expansion. Individuals with higher objective probabilities of having HD (by virtue of emerging signs or symptoms) are more likely to pursue testing.¹ Further, testing appears to be commonly prompted by a *change* in symptoms which indicates increased likelihood of carrying the HD expansion. We show that testing *explicitly for confirmation* in this context is fairly common; as many as half of at-risk individuals will eventually be tested to “prove” what they know already from symptoms.

We next describe beliefs and behaviors among untested individuals. When asked about their chance of carrying the HD expansion, untested individuals report perceived probabilities which are much lower than their objective probabilities (determined by the investigator based on clinical signs). In many cases, the bias is extreme. For example, among untested individuals for whom a clinical investigator observes signs which represent “Certain Signs of HD (≥ 99 percent confidence),” the average perceived chance of having HD is 52 percent, and 11 percent of individuals in this group report believing there is *no* chance they carry the HD expansion.

Second, although behaviors (e.g., fertility, retirement choices) differ significantly for individuals who report certainty about either carrying or not carrying the affected gene, individuals who are uncertain almost always behave identically to those who are not carriers of the genetic expansion, rather than displaying intermediate behavior. For example, adjusting for demographics, retirement is more than twice as likely for individuals who report knowing they carry the HD expansion versus those who are certain they do not. However, retirement rates for individuals with intermediate probabilities are identical to those who are certain they do not carry the expansion. This remains true even when we focus on individuals whose symptoms indicate a 90 percent or greater chance of HD.

Other authors have noted that the combination of low testing rates with low testing costs is a challenge to a standard neoclassical model and suggested this behavior might be better modeled with a framework in which beliefs about the state impact utility directly (Kőszegi 2003; Caplin and Leahy 2004; Caplin and Leahy 2001). Our data suggest additional facts that such a model should accommodate.

In Section III we suggest that an optimal expectations model, based on Brunnermeier and Parker (2005), provides a parsimonious way to explain the patterns in the data.² Section IIIA describes the setup. There are three periods, a binary state (“sick” or “healthy”), and a binary action choice. Individuals are endowed with some probability that they carry the HD gene. At time 0, they can choose whether

¹ This objective probability is based on investigator evaluations done as part of the study. The results of these evaluations are not transmitted to the patient.

² We focus on a setup in which individuals make the choice about information seeking and actions on their own. This is related to a setting in which information can be conveyed by an agent who seeks to maximize utility of a principal (Kőszegi 2006). A major difference between our setup and the setup in Kőszegi (2006) is that individuals here get utility from not only instrumental outcomes but also from their health status per se. In addition, there are several other models which are close in spirit to Brunnermeier and Parker (2005). These include Yarovitz (2005) and Mayraz (2011). The Bénabou and Tirole (2002) model of self-confidence is also closely related, if slightly more distant.

to learn the true state, possibly for some real cost. An action is chosen at time 1, and then the true state is revealed at time 2. At time 1, individuals experience utility associated with their anticipation of future consumption; at time 2, actual consumption utility is delivered. Consumption utility is maximized when the action is correctly matched to the realized state.

The key feature of this model is that if individuals are untested they have the option to choose their beliefs about the probability of each state but are constrained to take actions consistent with those beliefs. Choosing an overly optimistic belief increases the time 1 anticipation utility but also increases the chance that the wrong action is chosen, with a time 2 utility cost. Overly optimistic beliefs may be optimal if the increase in anticipatory utility outweighs the decrease in consumption utility. If an individual chooses to test, he cannot “unlearn” the true state and, therefore, does not have the option to choose beliefs.

Section IIIB relates the model to the facts in Section II. Individuals in this model adopt overly optimistic beliefs in order to experience higher utility in the anticipation period. Having adopted such overly optimistic beliefs, individuals take overly optimistic actions, in accordance with these beliefs. These skewed choices of behavior naturally produce the result that testing is increasing in risk: as the objective risk increases and people continue to behave as if they do not carry the HD expansion, the utility loss from this behavior becomes larger and larger, increasing the incentive to test.

Once tested, individuals in this model can no longer manipulate their beliefs. This means that a significant “cost” of testing is the loss of the option to believe that one is healthy regardless of the true state; this cost may be so large that the value of testing is negative even ignoring any real costs. Even for those with a positive testing value, a very small real cost of testing may be sufficient to discourage testing. In an extension we show that it is possible to accommodate confirmatory testing in this model. A central feature of this model is that, although the preferences are non-standard, individuals are not making a mistake by not testing: information avoidance may be optimal.

In Section IV we return to evaluating the standard neoclassical framework. The combination of low testing rates with low testing costs seems like a threat to the standard model. However, in practice we observe many settings in which individuals are slow to take actions which benefit them (for example, poorly optimized retirement savings in Choi, Laibson, and Madrian 2011). Given this, it seems hasty to reject this framework if the *only* issue is the need for high real testing costs.

If we assume individuals place no weight on anticipation, the optimal expectations model collapses to the neoclassical framework. We derive the predictions of the model in the case without anticipatory utility. We argue the neoclassical model fails qualitatively in two concrete ways: it cannot accommodate skewed beliefs or confirmatory testing. In addition, generating skewed action choices and testing increasing in risk require assumptions on the parameters which, although plausible, do not accord with intuition or other survey data.

Overall, we argue the addition of these new facts more concretely rules out the neoclassical framework. In the online Appendix we describe two other models with anticipatory utility (Mayraz 2011; Kőszegi 2003) and relate them to our data.

Our primary contribution is to understanding the phenomenon of information avoidance. However, we argue this article also contributes to the medical literature.

The observation that medical and genetic testing rates are low has been made in a number of settings, and this literature is often concerned with how to encourage testing, and whether doing so is a good idea. This article provides a concrete answer to why individuals may avoid testing and a framework in which to think about policy.

I. Background and Data on Huntington Disease

A. Genetic Testing

Advances in the understanding and sequencing of the human genome have dramatically increased the number of diseases and conditions for which genetic testing is available. For many conditions, the information provided by such tests is limited. Although individuals with a particular genetic mutation may be more likely to develop the disease, the change in probabilities is small.

There are a few conditions for which more informative genetic tests are available. HD is one; other common examples are breast and ovarian cancer, colon cancer, and Alzheimer's disease. In most cases, use of these tests is low. HD is an extreme example, with testing rates under 10 percent (Shoulson and Young 2011). Studies of cancer marker testing among those with a family history suggest testing rates of around 60 percent for breast and ovarian cancer and 40 percent for colon cancer (Ropka et al. 2006; Lerman et al. 1999). Roberts et al. (2004) find only 25 percent of contacted individuals with a family history of Alzheimer's were interested in genetic testing.

Greater degree of family history has been found to be correlated with increased testing (Ropka et al. 2006). Higher education levels are also positively correlated with testing rates (Lerman et al. 1999; Roberts et al. 2004). Perhaps most interesting, uptake is typically quite a bit higher when individuals are asked about *hypothetical* testing (Ropka et al. 2006; Quaid and Morris 1993).

These low rates of genetic testing are consistent with low use of general medical tests. Kőszegi (2003) summarizes a number of papers on reluctance to visit a doctor among individuals who suspect they have cancer. More generally, screening for cancer (mammography, colonoscopy) are far from universal (DeSantis et al. 2011; Cummings and Cooper 2011). Data on HIV testing in Africa suggests only about 10 percent of the population there has ever undergone an HIV test, despite very high prevalence rates (Matovu and Makumbi 2007). Relative to this existing work, we have much richer data. Our hope is that this advantage will allow us to shed light on the reasons for limited testing in these other settings.

B. Huntington Disease Background³

Huntington disease (HD) is a degenerative neurological disorder that clinically affects an estimated 30,000 individuals in the United States. Individuals with the disease typically begin to manifest symptoms in early middle age (30–50). Symptoms include involuntary movement, impaired cognition and psychiatric

³In this section we provide only a brief overview of Huntington disease; for a fuller clinical discussion, please see Shoulson and Young (2011).

disturbances. Individuals will need increasing levels of supportive and institutional care for many years. Death follows approximately 20 years after onset. A test for the HD genetic expansion was developed in 1993. Since everyone with the expansion will eventually develop HD, this test is perfectly predictive.

HD is a genetic disorder due to an excessive expansion in the Huntington gene on chromosome 4. The disease is inherited in an autosomal dominant manner: individuals who have a parent with HD have a 50 percent chance of having inherited the genetic expansion. The fact that HD has such clear and strong genetic predisposition means individuals are generally aware of their family history and genetic risk.⁴

Although the risk of HD at birth for someone with an affected parent is 50 percent, as they age individuals should update their probability either up or down. The progression of HD is slow but steady, and timing of onset varies.⁵ Early symptoms are not a perfect signal of HD. As symptoms develop, individuals should update their probability of carrying the expansion, generating variation in probability above 50 percent. On the other hand, as individuals age without symptoms, especially moving through middle age, it becomes progressively less likely that they carry the expansion.

There is no cure for HD. Nevertheless, there may be some private gains to knowing that one carries the genetic mutation. One clear gain is the ability to make better life decisions. This includes the choices we analyze below. In other work (Oster, Shoulson, and Dorsey forthcoming) we also show differences in human capital and health investments across these groups and differences in insurance ownership (Oster et al. 2010). There is also at least one treatment which is available to address the motor symptoms of HD, which could be prescribed if the disease is acknowledged.

C. Data Description

PHAROS (Prospective Huntington At Risk Observational Study) was a prospective, observational study of individuals at risk for HD conducted by the Huntington Study Group (Huntington Study Group PHAROS Investigators 2006). The study began in 1999 and included 1,001 individuals at roughly 40 study sites in the United States and Canada. Individuals in the PHAROS study were interviewed at recruitment and then approximately every nine months afterward. Prospective clinical evaluation in the PHAROS study concluded in 2010. PHAROS enrolled individuals who were (at the time of enrollment) at risk for HD. They had one parent (or first-degree relative) with HD but had not pursued genetic testing.

Sample Selection: Participants in PHAROS are not a random sample of individuals at risk for HD. Of particular concern is the fact that participants had to be untested at the time of enrollment into the study. Since our goal is, in large part, to

⁴It is, of course, possible that people may not know of their risk until they are older, since their parents' age of onset may be late or their parents may die of something other than HD before onset. In the case of our sample, knowledge of risk is required for enrollment in the study, so everyone in our setting knows they are at 50 percent risk.

⁵Timing of onset has an inverse relationship with the extent of the cytosine-adenine-guanine (CAG) expansion. The greater the expansion, the earlier the onset. Tested individuals would learn their CAG expansion count, which provides information on expected age of onset.

draw conclusions about why people avoid testing, it would be a concern if this selection procedure drew in people with a much lower demand for testing than the average person. Empirically, this doesn't seem to be the case: testing rates in our sample are around 5 percent, similar to other data (Shoulson and Young 2011). A related concern is that the reason for testing avoidance in this population is different than in the general HD population. Our sample by definition excludes the youngest testers; if the behavior and beliefs of these individuals is very different, we may overstate the importance of our explanation.

Mechanically, this is unlikely to be a major concern simply because testing is unusual. Given that, over a whole lifetime, only 5 to 10 percent of individuals undergo predictive testing, the share of people who test at young ages is even smaller than that. Any conclusions we draw from those who are not tested while young will apply to the majority of the population. In addition, if we limit our sample to individuals who enter the study before the age of 45 (the younger half of the sample) we see virtually identical results (available from the authors). The fact that these results look very similar to our overall population suggests that if we could include younger testers it would not change the conclusions.

There is also a concern that the type of individual who chooses to participate in a research study of this type may be more aware of his HD risk and more thoughtful about his testing choices than the general HD population. This would seem to work against our findings that these individuals hold incorrect beliefs about their risk. In addition, to the extent that we are hoping to draw conclusions about testing in other populations of this type (for example, those with a family history of cancer) the behavior of this group may be an accurate reflection of those individuals most "at risk" for testing.

Variables Used: Participant visits during PHAROS contained two segments. First, individuals responded to a set of questionnaires, which collected information on demographics, life events and HD-specific behaviors and beliefs. Second, there was a neurological exam performed by the investigator. We use data from both segments.

Investigator Evaluation of HD Status.—Individuals in PHAROS were given a series of clinical tests at each visit. These tests were designed to evaluate whether the individual was developing HD, and they include tests of motor and ocular performance, gait and involuntary movements such as chorea. Based on this test, individuals were given a motor score which could range from 0 (no signs of HD) through 154 (maximum signs of HD). In addition, at the end of the exam the investigators, who remained unaware of gene carrier status, make a composite judgment of confidence on a scale from 0 to 4. A 0 indicates "normal (no abnormalities)," a 1 indicates "nonspecific motor abnormalities (less than 50 percent confidence of having HD)," a 2 indicates "motor abnormalities which may be a symptom of HD (50–89 percent confidence of having HD)," a 3 indicates "motor abnormalities that are likely signs of HD (90–98 percent confidence)" and a 4 indicates "motor abnormalities that are unequivocal signs of HD (≥ 99 percent confidence of having HD)." We should note that given the construction of this sample, individuals who have no signs of HD at all (and are sufficiently young) are still at about 50 percent risk, since they have a parent with the HD expansion and could have inherited the expansion. Any clinical

confidence score greater than zero indicates a higher likelihood than the nominal 50 percent risk.

The other source of variation in probability comes from age. As individuals age without signs or symptoms, they become less likely to carry the HD expansion. This generates variation in the probability below 50 percent: at birth, the probability is 50 percent, and as people age without signs or symptoms, the probability drops. At-risk individuals who do not develop signs of HD by their late 60s and beyond are unlikely to carry the expansion.

Perceived Probability of HD.—Individual subjective probability of carrying the HD expansion is based on the following question: “On a scale of 0 to 100, today, how likely do you think it is that you carry the genetic mutation that causes HD? 0 = absolutely certain that you do not have the gene mutation that causes HD and 100 = absolutely certain that you do have the gene mutation that causes HD.” Summary statistics for the motor score and perceived probability appear in panel A of Table 1.

HD Testing and Gene Status.—Information on testing is drawn from a question, asked at each visit, about whether the individual has undergone HD testing since his last visit. The share of individuals who choose to be tested is summarized in panel A of Table 1. In addition to exploring testing as an outcome, we use these data to pin down optimal behavior for individuals who are certain that they do or do not carry the HD mutation.⁶ Using the testing data for this latter purpose requires knowing individual test results. While everyone in the study consented to independent research analysis of a blood DNA sample as part of the study, these individual identifiable research results are never made available to anyone, either research participants or investigators. However, for individuals who chose to be tested outside the study we can infer their test result by using information from the investigator assessment or from their subjective probabilities (after testing, a large share of people report either 0 percent or 100 percent chance of carrying the HD expansion). The inference procedure is described in more detail in Oster et al. (2010).

Life Events.—Information on life events is drawn from a questionnaire entitled the “Life Experience Survey” which was administered (for most participants) five or six times over the 7–10 year study. This questionnaire listed a number of life events and asked the individual about whether he had experienced each event in the last year.⁷ We use data on a subset of events which reflect choices. These are: marriage, pregnancy (either self or partner), divorce, getting a new job, reporting a major change in finances (including reports of borrowing), change in church activities, change in recreation, and retirement.

These data do not cover all life experiences in which we might be interested. In addition, the survey did not probe in more depth about exactly *what* is implied

⁶ A fundamental concern here is that those individuals who are tested behave differently than those who are not. This is worth keeping in mind, although in practice we will find the behavior among those who say they do not carry the HD expansion is very similar to those with intermediate risk suggesting, perhaps, that this is of limited concern.

⁷ Details on these questions are included as part of the online data Appendix.

TABLE 1—SUMMARY STATISTICS

	Mean	SD	Observations
<i>Panel A. HD status variables</i>			
Motor score (0–100)	3.76	6.45	7,426
Doctor evaluation of risk (0–4 scale)	0.668	1.04	7,449
Perceived chance of HD mutation (untested)	42.7%	25.0%	3,413
Tested (0/1)	5.7%	23.2%	7,576
Tested positive (0/1) (if test status inferred)	57.3%	49.5%	386
<i>Panel B. Life experience summary statistics</i>			
Pregnant (self or partner), under 40	10.9%	31.1%	1,011
Get married (if unmarried)	6.9%	25.3%	973
Get divorced (if married)	1.7%	13.0%	3,106
New job	14.0%	34.7%	3,842
Retire	1.4%	12.0%	3,864
Major financial change	38.5%	48.7%	4,110
Change in church attendance	8.2%	27.4%	4,045
Change in recreation activities	18.7%	39.0%	4,048
<i>Panel C. Demographic summary statistics</i>			
Age	41.8	7.3	1,001
Male (0/1)	31.2%	46.3%	1,001
Education (years)	14.9	2.6	999

Notes: This table shows simple summary statistics from the PHAROS data. In panels A and B an observation is an individual-year, since perceived probability and doctor score can both vary across visits for a given individual, and actions are chosen in multiple years. In panel C there is just one observation per individual. Two individuals did not respond to the survey asking about education. Oster et al. (2010) detail the methodology for inferring test results for tested individuals.

by the behavior. In some cases, like “Made a Major Financial Change,” it is not entirely clear what happened (in others, like pregnancy, it is). Despite these drawbacks, we believe that these data are informative about behavior. Summary statistics, reporting the share of individuals engaging in each behavior, are reported in panel B of Table 1.

Demographics.—We will also use data on basic demographics—gender, age, and education. These are summarized in panel C of Table 1.

II. Descriptive Analysis: Testing, Risk, Beliefs, and Behavior

A. HD Testing Rates and Testing Costs

We begin with the most basic fact about HD testing: it is uncommon. In the ten years that the PHAROS study ran, about 7 percent of individuals with uncertain HD status chose to take an HD test. Testing is even more limited, about 5 percent, when we focus on people who test prior to observing *any* signs or symptoms of HD. We might expect testing rates in this population to be especially low, given that a requirement for enrollment is that individuals are untested. However, the levels are very similar to what is seen in the HD population overall (Myers 2004).

Laboratory costs for an HD test are on the order of \$200–\$300. The actual financial costs may be higher, perhaps twice that, once they include consulting a neurologist and genetic counselor before testing, which most testing centers require. This testing

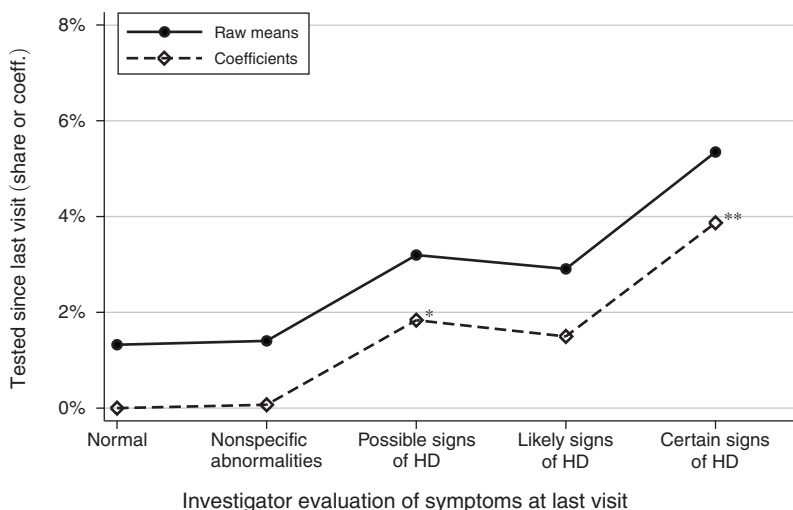


FIGURE 1. TESTING BEHAVIOR AND INVESTIGATOR EVALUATION OF RISK

Notes: This figure shows the relationship between testing and investigator evaluation of risk. The x -axis shows the investigator evaluation of HD status at the last visit. “Possible” signs of HD indicate an estimated 50–89 percent probability of having HD; “Likely” signs indicate 90–98 percent chance; “Certain” signs indicate ≥ 99 percent chance. The data is limited to individuals who were not tested at the time the evaluation was done (i.e., those at risk of testing). We report the chance of testing before the next visit. The solid line is the raw mean chance of testing. The dashed line is the coefficient from a regression adjusting for education, age, and gender. * significantly different from “Normal” at 5 percent level; ** significantly different from “Normal” at 1 percent level.

would be covered by insurance, although in a large share of cases individuals report paying out of pocket for testing, likely to retain the option to keep their test results private (Oster et al. 2008).

B. Testing and Pretesting Risk

Although testing is low in general, testing rates vary with individual ex ante risk of finding they carry the HD expansion. Figure 1 shows the probability of testing before the next PHAROS visit graphed against investigator diagnostic confidence score at the last visit. The solid line shows the raw means—the share of individuals testing. The dotted line shows coefficients adjusted for demographic controls (listed in the table notes). With either approach, we see an increasing pattern: the highest probability of testing is among individuals with an investigator score of 4. It is perhaps puzzling that people who should be nearly certain they carry the expansion nevertheless choose to test. However, as we will see below, in practice many of these individuals report believing their probability is lower, implying they believe there is still information to learn.

Figure 2 shows this result in changes. We graph the chance of testing between visits against the change in investigator score in the two visits leading up to that. This answers the question of whether changes in symptoms prompt testing. Again, this slopes up: individuals tend to test when new information points towards an increasing chance of carrying the expansion.

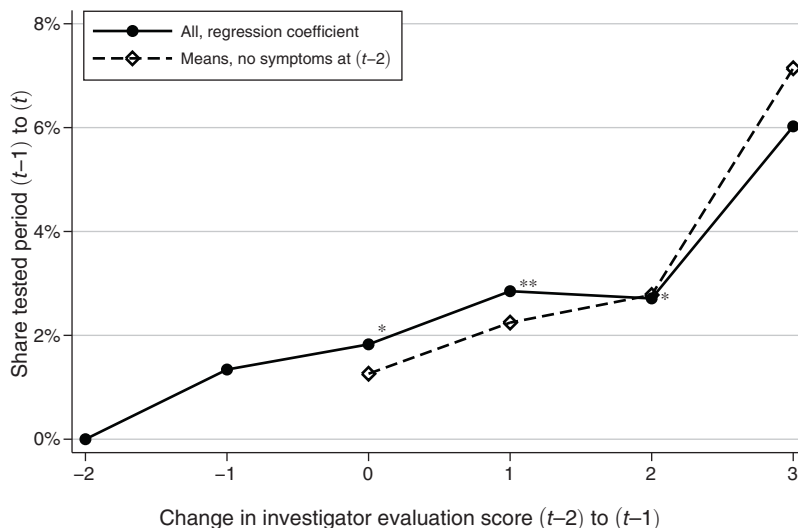


FIGURE 2. TESTING BEHAVIOR AND CHANGE IN RISK

Notes: This figure shows the relationship between testing and change in investigator evaluation of risk. The x-axis shows the change in risk between visits ($t-2$) and ($t-1$), and the y-axis represents the chance of testing between visits ($t-1$) and t . The dashed line shows raw means for individuals with an investigator report of “Normal” at baseline (i.e., at $t-2$). The solid line shows coefficients from a regression which combines all individuals and controls linearly for baseline score at $t-2$. Coefficients are relative to individuals with a change of -2 (i.e., they have significantly fewer symptoms at time $t-1$ relative to time $t-2$). The data are limited to individuals who were not tested at visit $t-1$. *significantly different from change of -2 at 10 percent level; **significantly different from change of -2 at 5 percent level.

Finally, we explore age variation in testing. As individuals age without developing symptoms, their (objective) updated probability of carrying the HD expansion declines. This allows us to look at whether testing becomes more common as people become more sure that they *do not* carry the expansion. Figure 3 shows the chance of testing by the next visit by age group (labels indicate updated HD risk). Testing probability is not systematically varying with age. Reductions in risk below 50 percent do not prompt testing.

It is worth noting that these results are especially surprising in this case because there is no cure for HD. In settings with a good treatment, it might not be surprising to find that higher risk individuals are more likely to get tested. Here, that mechanism is not active, and yet we still see this pattern.

Even at the highest risk levels, testing is still somewhat uncommon. However, testing to confirm HD status once it is known is much more frequent. Of the people in our data with acknowledged symptoms of HD, 30 percent have undergone HD testing. In another dataset (the COHORT study), confirmatory testing is more common: among individuals who notice symptoms without having been tested, 75 percent of them choose to have a confirmatory genetic test.

C. Perception of Risk

We turn now to beliefs and actions among individuals who remain untested. In our data, we observe both self-reports about the probability of carrying the HD

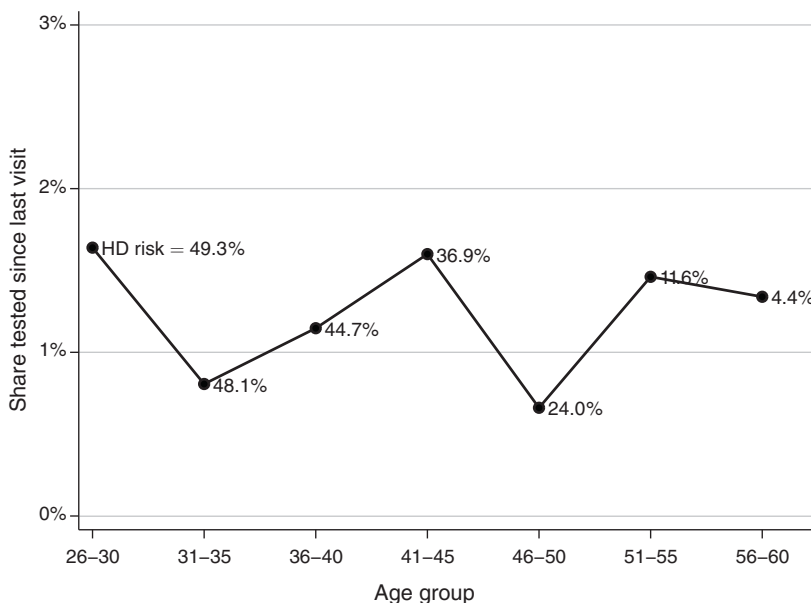


FIGURE 3. TESTING BEHAVIOR BY AGE, INDIVIDUALS WITH NO SYMPTOMS

Notes: This figure shows the relationship between testing and age among asymptomatic individuals. The labels indicate the remaining risk of carrying the HD mutation for the average individual in that age group with no symptoms. Posterior probabilities are calculated for someone with 44 CAG repeats (the average in a comparable sample) based on the formula given in Langbehn et al. (2004).

expansion and investigator evaluation of motor signs of HD. These signs are informative, but not unequivocal. More signs make the diagnosis more certain. Based on data which include motor signs of HD and actual gene status, we calculate the posterior probability of carrying the HD expansion by level of motor signs.⁸ Figure 4 shows the actual posterior chance of carrying the HD expansion and individual self-perception. In addition, we graph the share of untested individuals at each level of motor signs who report there is *no* chance they carry the HD expansion.

Based on this figure, it is clear individuals are overly optimistic. Among those with limited symptoms, the average reported risk is about 40 percent, only slightly lower than the 50 percent objective risk. However, individuals update only very minimally with increasing symptoms. As the objective chance of carrying the HD expansion increases to 100 percent, the average subjective probability moves from 40 percent to just over 50 percent. The interquartile range typically does not include the true value. Moreover, some individuals persist in reporting there is no chance

⁸This is calculated using Bayes's rule based on a dataset in which motor evaluation is done for individuals with HD and control individuals without any HD risk. We adjust the posterior based on the chance of noticing symptoms at each symptom level among individuals who know they carry the HD mutation. That is: a motor score of 1 is actually reasonably informative about whether the individual has HD, but even among individuals who know they carry the mutation only a small share of them notice motor symptoms at this point. When we calculate the posterior in Figure 4 we therefore report: $(Pr_{notice})(posterior_{motor=x}) + (1 - Pr_{notice})(0.5)$. Note that if we showed the true posterior, unadjusted, individuals would be even more overly optimistic.

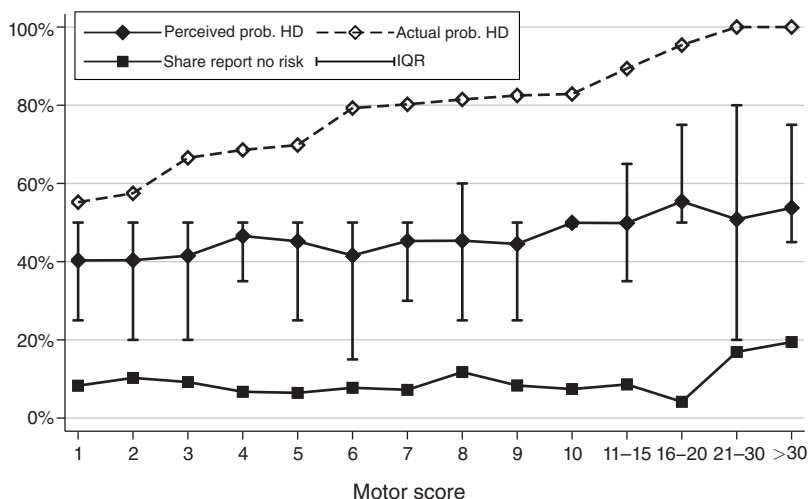


FIGURE 4. PERCEIVED AND ACTUAL RISK OF HD, BY MOTOR SCORE

Notes: This figure shows actual risk of HD by motor score (the dashed line) and perceived risk of HD (upper solid line) by motor score. The bottom line displays the share of individuals at each motor score reporting no risk of HD. The IQR (twenty-fifth to seventy-fifth percentile) for perceived chance is also displayed. Actual risk is based on accurate Bayesian updating based on motor score combined with information on the chance of noticing HD at each symptom level given known genotype. Details are in footnote 8.

that they carry the HD expansion, even when they have significant symptoms.⁹ Regressing self-perception on actual risk with simple demographic controls yields a coefficient of around 0.09, much less than the value of 1 that we would expect if self-perceptions and objective assessments were synchronous.¹⁰

D. Risk and Behavior

Finally, we consider behaviors undertaken by individuals with varying objective or subjective probabilities of carrying the HD expansion. Table 2 compares behaviors for those who report being certain they carry the HD expansion and those who report being certain they do not carry the expansion. Column 1 shows means, and column 2 shows regression coefficients adjusted for age, gender, and education.¹¹

⁹One concern with this is that a large share of people are defaulting to 50 percent, and if we ignored individuals with a report of 50 percent we would see something different. This is not the case; leaving these individuals out the perceived risk in the lowest groups is around 37 percent and in the highest is around 52 percent, very similar to what we observe when including all the data.

¹⁰HD has mental as well as physical symptoms, so one possibility is that this apparent “bias” is simply due to confusion. However, the lack of updating of risk appears even among individuals with fairly low motor scores who are unlikely to be so impaired that they are unable to process the question. In addition, there is little reason to think this confusion would bias consistently downward.

¹¹The sample of people who are sure they do carry the HD expansion includes individuals who have been tested and know they carry the HD expansion but do not have symptoms, as well as those who are certain they have the expansion due to symptom development. Given this, one concern is that behavior might be different since these individuals are actually sick and cannot engage in certain behaviors. In practice, this does not seem to impact our results: controlling for the degree of motor symptoms observed makes no difference.

TABLE 2—BEHAVIOR AMONG INDIVIDUALS WITH CERTAIN HD STATUS

	Difference in mean probability	Coefficient adjusted for controls
	With HD mutation – without HD mutation (1)	With HD mutation relative to without mutation (2)
Pregnant (self or partner), under 40	0.136**	0.318*
Get married (if unmarried)	0.032	0.022
Get divorced (if married)	0.045**	0.046**
New job	0.013	0.013
Retire	0.055**	0.051**
Major financial change	0.163***	0.184***
Change in church attendance	0.036	0.047
Change in recreation activities	0.115**	0.115**

Notes: This table shows, for each action, the relative probability of taking the action in the last year for individuals who are certain they do or do not carry the HD mutation. Column 1 shows the basic difference in means (significance from *t*-tests); column 2 shows the coefficients from a regression adjusting for simple controls (gender, education, and age). In the case of pregnancy we also adjust for number of children and pregnancy last year.

*** Significant at the 1 percent level.

** Significant at the 5 percent level.

* Significant at the 10 percent level.

These groups do not differ on every action, but there are large significant differences in behavior for five of the eight items. Divorce is more likely for individuals who know they carry the HD expansion. Individuals who know they carry the HD expansion are more likely to get pregnant, more likely to retire, and much more likely to report major financial changes and changes in recreational activities.

Although this is not the focus of the paper, we note that for the most part these patterns are what we would expect based on a life cycle model, especially retirement, financial changes, and changes in recreation. The direction of the differences for pregnancy is surprising. It may be that the knowledge of a shortened lifespan advances forward optimal fertility timing. It is also worth noting that although this impact is large and statistically significant, the fertility results rely on a very small sample and should be taken with caution.

If we take the behavior of those individuals who are certain about their status as reflecting full-information choices, we can then ask where the behavior of uncertain individuals lies relative to these points. Of course, it is meaningful to ask this only about the subset of actions which differ in Table 2.¹² Graphical evidence on the behavior among uncertain individuals can be seen in Figure 5. This figure shows coefficients, adjusted for demographic controls, measuring differences across groups. In each case we show the coefficients for uncertain individuals and those who know they carry the HD expansion relative to those who are certain they do not carry the expansion. In all cases, we see evidence that behavior differs for the two extreme groups (as in Table 2) but find that the behavior of those individuals who remain untested mimics that of those who know they do not carry the HD expansion.

¹² Since the choices are binary, it is difficult to understand what “intermediate” behavior would be. The simplest way to envision this is to imagine that learning they carry the HD expansion prompts 20 percent of people to get pregnant. Intermediate behavior would suggest that a 50 percent risk would push 10 percent of people into pregnancy.

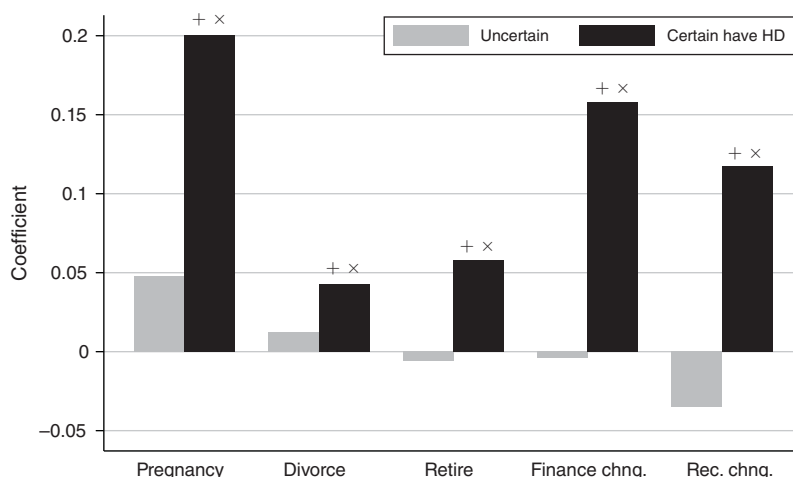


FIGURE 5. BEHAVIOR CHOICE RELATIVE TO INDIVIDUALS WITHOUT HD EXPANSION

Notes: This figure shows differences in behavior relative to tested individuals who report they do not carry the HD mutation. Uncertain individuals are those who are untested and report intermediate probabilities. Those who carry the mutation, know they carry it either through testing or through early symptoms. Regression adjusts for age, education, and gender. The pregnancy regression restricts to people under 40 and controls for existing children and pregnancy last year. + indicates significantly different from those without the HD mutation at 5 percent level; × indicates significantly different from uncertain individuals at 5 percent level.

Table 3 shows further regression evidence in which untested individuals are differentiated based on their symptom level. This gives us some sense of whether individuals are at least more likely to engage in intermediate behaviors as their objective risk increases. This table indicates that actions among untested individuals are strongly skewed toward the expansion-negative optimal action. For pregnancy, divorce, financial changes and recreation there are no significant differences in behavior even up to the highest risk group. Individuals with motor scores above 11 have a greater than 90 percent chance of carrying the HD expansion (see Figure 4) and yet seem to behave no differently from those who are certain they do not carry the expansion. In the case of retirement there is some evidence of intermediate behavior in this highest risk group, but nothing in the groups with lower motor scores. When we aggregate (column 6), we find no evidence of changes in behavior even among the group with the highest motor scores.

III. Theory: Optimal Expectations

Low medical testing rates in settings where the information impacts decision making and the financial costs of testing are small seem to be a challenge to a standard neoclassical model of behavior (e.g., Kőszegi 2003; Caplin and Leahy 2004). This has led to the suggestion that models of this behavior should incorporate some form of anticipatory utility (Caplin and Leahy 2001), wherein individuals care about their expectations about the future in addition to their present consumption. The descriptive evidence in Section II presents several other related facts which such a model would ideally accommodate.

TABLE 3—BEHAVIOR BY HD SYMPTOM LEVELS

Dependent variable Sample	Pregnant (self or partner) Age < 40 (1)	Get divorced Married (2)	Retire All (3)	Major finance change All (4)	Change in recreation All (5)	All behaviors All (6)
Uncertain, motor = 0	0.062 (0.059)	0.008 (0.014)	−0.007 (0.011)	0.002 (0.044)	−0.048 (0.035)	−0.006 (0.016)
Uncertain, motor 1–3	0.015 (0.060)	0.017 (0.014)	−0.007 (0.011)	0.003 (0.045)	−0.047 (0.035)	−0.005 (0.16)
Uncertain, motor 4–6	0.077 (0.064)	0.019 (0.015)	−0.010 (0.012)	−0.017 (0.047)	0.002 (0.038)	0.002 (0.017)
Uncertain, motor 7–10	−0.008 (0.070)	0.010 (0.016)	−0.006 (0.013)	−0.032 (0.052)	−0.011 (0.041)	−0.008 (0.019)
Uncertain, motor > 11	0.060 (0.093)	0.007 (0.019)	0.032** (0.015)	0.012 (0.059)	0.010 (0.047)	0.019 (0.024)
Certain carry mutation	0.200** (0.097)	0.043** (0.018)	0.058*** (0.014)	0.158*** (0.056)	0.117*** (0.044)	0.102*** (0.023)
General controls	Yes	Yes	Yes	Yes	Yes	Yes
Prev. children controls	Yes	No	No	No	No	No
Observations	625	2,788	3,462	3,677	3,624	14,486

Notes: This table reports estimates of differences in behavior for individuals with varying risk of HD. The omitted category is individuals who are tested and know they do not carry the HD mutation. Uncertain individuals are those who report an intermediate probability of carrying the mutation; we differentiate them by their doctor-assigned motor score, which ranges from 0 (no symptoms) up to 100 (which would indicate extremely advanced HD). The true updated probability of HD for each motor score group can be seen in Figure 4. The certain group contains those individuals who report being sure they carry the HD mutation. General controls are: fixed effects for ten-year age group, gender, years of education. Column 1 also controls for the number of existing children and pregnancy last year. The final column estimates the impact on all behaviors together, with the data stacked so the observation is an individual-behavior. In this case we control for dummies for each behavior and cluster the standard errors by individual. Standard errors in parentheses.

*** Significant at the 1 percent level.

** Significant at the 5 percent level.

* Significant at the 10 percent level.

In this section we outline an optimal expectations model, based on Brunnermeier and Parker (2005), which we argue provides a parsimonious explanation for both low testing rates and the facts described in Section II. There are two key underpinnings of the model. First, individuals experience anticipatory utility. Second, as long as they are uncertain about the future state, individuals can hold beliefs about the state which differ from the true probabilities. We introduce the possibility of testing and learning the true state before the action is chosen.

It is perhaps important to note that although the language used in this model indicates that individuals “choose” their beliefs, this need not be a description of the psychological process by which these beliefs occur. Individuals may “choose” beliefs, for example, by ignoring signs which would contradict their beliefs (as in Dawson, Gilovich, and Regan 2002).

A. Setup

There is a binary state $s \in \{0, 1\}$ where $s = 1$ indicates the individual has the gene or disease (in this case, carries the HD expansion) and $s = 0$ indicates he does not. We refer to these states as “sick” and “healthy.” Individuals have some

exogenous $p = E(s)$. This p is not affected by behavior; there is nothing an individual can do to impact his probability of developing the disease. This is consistent with the HD setting.

At time 0, individuals choose whether or not to learn the true state through testing. This testing has a real cost, denoted C . At time 1, individuals choose a binary action $a \in \{0, 1\}$ and experience (discounted) utility associated with their expectation of time 2 consumption. Ex post individual consumption utility is maximized when action is matched to state. At time 2, the true state is revealed, and individuals receive consumption utility.

The key assumption in this model is that if individuals do not choose to learn the true state they are able to adopt beliefs about the probability of each state at time 1. These chosen beliefs may differ from the true probability p . Actions are picked at time 1 based on these chosen beliefs only. Denote the chosen belief as π and utility given action a and realized state s as $u(a, s)$. Assume anticipation utility is down-weighted by a factor $\delta \in [0, 1]$.

Formally, individuals choose $\pi \in [0, 1]$ to maximize

$$U(\pi|p) = \delta E(u(\hat{a}, s)|\pi) + E(u(\hat{a}, s)|p),$$

where $\hat{a}(\pi) = \operatorname{argmax}_a E[u(a, s)|\pi]$. With the binary state we can write expected utility at time 2 as $E[u(\hat{a}, s)|p] = pu(\hat{a}, 1) + (1 - p)u(\hat{a}, 0)$, and similarly for π in the anticipation period.

If individuals learn the true state at time 0 they are no longer free to choose beliefs. However, they will adopt the ex post optimal action, so $a = s$, and two-period utility is simply given by $(1 + \delta)[pu(1, 1) + (1 - p)u(0, 0)]$.

We define the following parameter values: $u(0, 1) = -\Omega$, $u(1, 1) = 0$, $u(1, 0) = 1 - \Phi$, and $u(0, 0) = 1$. Being healthy and taking the state-matched action has a value of 1; being sick and taking the state-matched action has a value of 0. Taking the wrong action in either case leads to a loss of utility. This loss is Φ if the state is “healthy” and Ω if the state is “sick.” In the simplest, symmetric case, $\Omega = \Phi$. We assume that $\Phi, \Omega < 1$, implying that individuals value health more than they value the correct action.

B. Results

Beliefs and actions are chosen at time 1. Lemma 1 below describes action choice given beliefs.

LEMMA 1: $\hat{a}(\pi) = 0$ if $\pi \leq \frac{\Phi}{\Phi + \Omega}$ and $\hat{a}(\pi) = 1$ if $\pi > \frac{\Phi}{\Phi + \Omega}$.

PROOF:

Actions are chosen in this model based only on the period 1 anticipation utility.

The individual will choose $a = 0$ if and only if

$$\pi u(0, 1) + (1 - \pi)u(0, 0) \geq \pi u(1, 1) + (1 - \pi)u(1, 0)$$

$$\pi \leq \frac{\Phi}{\Phi + \Omega}.$$

Note that under the symmetric assumption that $\Phi = \Omega$, this cutoff value is $\pi = 0.5$.

Choice of Beliefs and Resulting Actions.—We begin by deriving the implications of this model for the choice of beliefs and resulting actions.

PROPOSITION 1 (Choice of Beliefs): *Individuals will always choose beliefs such that $\pi \leq p$.*

PROOF:

Lemma 1 describes the choice of actions given the choice of beliefs. Given that result, utility is given by

$$U = \begin{cases} \delta(1 - \pi) + (1 - p) - (\delta\pi + p)\Omega & \text{if } \pi \leq \frac{\Phi}{\Phi + \Omega} \\ (\delta(1 - \pi) + (1 - p))(1 - \Phi) & \text{if } \pi > \frac{\Phi}{\Phi + \Omega}. \end{cases}$$

We have assumed that $\Phi, \Omega < 1$, so the agent will only ever choose either $\pi = 0$ or $\pi = \frac{\Phi}{\Phi + \Omega}$. As long as the cutoff point at which individuals switch to belief $\pi = \frac{\Phi}{\Phi + \Omega}$ is above $p = \frac{\Phi}{\Phi + \Omega}$, we then have the result that $\pi < p$. Individuals will choose $\pi = 0$ if the following inequality holds:

$$\delta + (1 - p) - p\Omega \geq \left(\delta \left(\frac{\Omega}{\Phi + \Omega} \right) + (1 - p) \right) (1 - \Phi)$$

$$p^* \leq \frac{\Phi}{\Phi + \Omega} + \frac{\delta\Phi(1 + \Omega)}{(\Phi + \Omega)^2}.$$

This implies they are choosing a value of $\pi = 0$ for $p \leq p^*$, with $p^* = \frac{\Phi}{\Phi + \Omega} + \frac{\delta\Phi(1 + \Omega)}{(\Phi + \Omega)^2} > \frac{\Phi}{\Phi + \Omega}$.

We note that in the case where $\Phi = \Omega$, the π cutoff is 0.5, so the proposition indicates that the actor in this model chooses $\pi = 0$ up to $p = 0.5 + \frac{\delta(1 + \Phi)}{4\Phi}$ and $\pi = 0.5$ for values of p above that. Proposition 2 summarizes action choices.

PROPOSITION 2 (Choice of Action if Untested): *Action $a = 0$ will be chosen for values of $p \leq p^*$, and action $a = 1$ will be chosen for values of $p > p^*$.*

PROOF:

This follows directly from Lemma 1 and Proposition 1. Individuals will choose belief $\pi = 0$ up to a value of $p^* = \frac{\Phi}{\Phi + \Omega} + \frac{\delta\Phi(1 + \Omega)}{(\Phi + \Omega)^2}$ and $\pi = \frac{\Phi}{\Phi + \Omega}$ for larger p . By Lemma 1, the agent chooses $a = 0$ in the first case and $a = 1$ in the latter case.

Proposition 2 shows that individuals will take action $a = 0$ for some values of $p > 0.5$ as long as Ω is not much larger than Φ . In the simple case where $\Phi = \Omega$ we

will see actions $a = 0$ for at least some values of $p > 0.5$, since $p^* > 0.5$. If $\Phi > \Omega$, this result is reinforced. It is only in cases where the cost to taking the wrong action if the true state is sick is much larger than if the true state is healthy that we might not see skewed actions.

The intuition behind the skewed action result is fairly straightforward. Skewed action choices are delivered by individuals' desire to "pretend" they do not have the disease. When individuals choose an overly optimistic belief, they benefit from experiencing positive anticipation: when they think about the future they experience anticipation of the ideal utility state, in which they are healthy and have taken the correct action. This overly optimistic belief has costs, however, since ex post actors experience a loss in consumption utility from having likely taken the wrong action. To the extent that the anticipation gain outweighs the realized loss later, it will be optimal to adopt an overly optimistic belief.

Testing and Risk.—When evaluating the value of testing, individuals compare the utility delivered when tested to the utility delivered by their optimal choice while untested. The latter is described above. The utility if tested is given below:

$$U_{test} = (1 + \delta)(pu(1,1) + (1 - p)u(0,0)) - C = (1 + \delta)(1 - p) - C,$$

where C is the cost of testing. The value of testing (V_{test}) is the difference between this testing utility and the utility delivered if untested.

Given the beliefs and action choices described, Proposition 3 describes testing behavior.

PROPOSITION 3 (Testing Behavior): *Define p^* as in Proposition 1. There are two cases, corresponding to a high and low anticipation value.*

Low Value of Anticipation: $\delta < \Omega$. *The following statements hold:*

- (i) *For values of $p \leq p^*$, the value of testing is positive if and only if $p(\Omega - \delta) > C$.*
- (ii) *For values of $p > p^*$, the value of testing is positive if and only if $\frac{\delta\Phi(1 + \Omega)}{\Phi + \Omega} - p(\delta + \Omega) + \Phi > C$.*

High Value of Anticipation: $\delta \geq \Omega$. *The value of testing is negative and decreasing in p at all values of p .*

PROOF:

If $p \leq p^*$, individuals take action $a = 0$. If $p > p^*$ they take action $a = 1$. The value of testing for each range is given below.

$$V_{test} = p(\Omega - \delta) - C \quad \text{if } p \leq p^*$$

$$V_{test} = \frac{\delta\Phi(1 + \Omega)}{\Phi + \Omega} - p(\delta + \Omega) + \Phi - C \quad \text{if } p > p^*.$$

Low Value of Anticipation: $\delta < \Omega$. For $p \leq p^*$ and $a = 0$, the value of testing is increasing in p (since $\Omega - \delta > 0$), meaning it is maximized at p^* . For values of $p > p^*$ and action $a = 1$, the value of testing is decreasing in p (since $-(\delta + \Omega) < 0$), meaning it is maximized at the lowest value of p , namely p^* . The implications about value of testing come directly out of the testing values given above. Note that these conditions imply that value of testing is maximized at p^* .

High Value of Anticipation: $\delta \geq \Omega$. For $p \leq p^*$ and $a = 0$, V_{test} is decreasing in p , since $-(\delta + \Omega) < 0$. However, it is always negative: individuals with this set of parameter values will never choose to test. We note that at p^* the value of testing is the same in the $a = 0$ and $a = 1$ cases. This is because p^* is defined such that at that value the utility from the two actions is the same. The utility in the tested case is also the same, so the total value of testing is the same for action $a = 0$ and $a = 1$ at p^* . For values of $p > p^*$ the value of testing is decreasing, and since it is negative at p^* , it is always negative.

The high value of anticipation case in Proposition 3 does not generate any variation in testing behavior, since individuals with this set of parameter values will never test. Any variation in testing with p will therefore be driven by individuals with low value of anticipation. These individuals may or may not choose to test. Their value of testing will be highest at p^* , defined as in Proposition 1. If anticipation is important, it is possible that even this maximum value may be very small.

We can illustrate this result graphically. For simplicity, we focus on the base case of symmetric losses: $\Phi = \Omega$. Consider first the impact of testing on time 1 anticipatory utility only. This impact is the difference between the anticipatory utility *with* testing, which is $\delta[(1 - p)]$, and the anticipatory utility *without* testing, which is δ for values of $p < p^*$ and $0.5\delta(1 - \Phi)$ for values of $p \geq p^*$. These two utilities, and their difference, are graphed against p (for benchmark values of Φ and δ) in Figure 6, panel A. Up to p^* , utility without testing is constant (since people are just acting as if they are healthy and experiencing anticipation associated with that state), and utility with testing is decreasing in p . The difference $(-\delta p)$ is therefore also decreasing in p .

The second element is the impact of testing on time 2 consumption utility. This is the difference between realized utility with testing, which is $(1 - p)$, and realized utility without testing, which is $-p\Phi + (1 - p)$ if $p < p^*$ and $(1 - p)(1 - \Phi)$ if $p \geq p^*$. These utilities are graphed against p in Figure 6, panel B. Utility both with and without testing is decreasing in p , but the utility without testing is decreasing faster. The time 2 difference in utilities below p^* is $p\Phi$, which is increasing in p up to p^* .

The total value of testing (ignoring the real cost) combines these two utilities. This is graphed in Figure 6, panel C, along with (for reference) the value of Φ . Because we have assumed that $\delta < \Phi$, the time 2 consumption utility dominates, and we observe that, overall, the value of testing is increasing in risk. However, because of the incorporation of the anticipatory utility, this testing value is much lower than it would be if we considered only the consumption utility. With this addition, even a very small real cost of testing could push people, especially those with low values of p , to not test. Effectively, a large portion of the cost of testing is the loss of the anticipatory utility.¹³

¹³ This section focuses on a binary action choice. This matches the data we observe but certainly doesn't cover all possible actions. In online Appendix A we develop a case of the model with continuous actions and quadratic

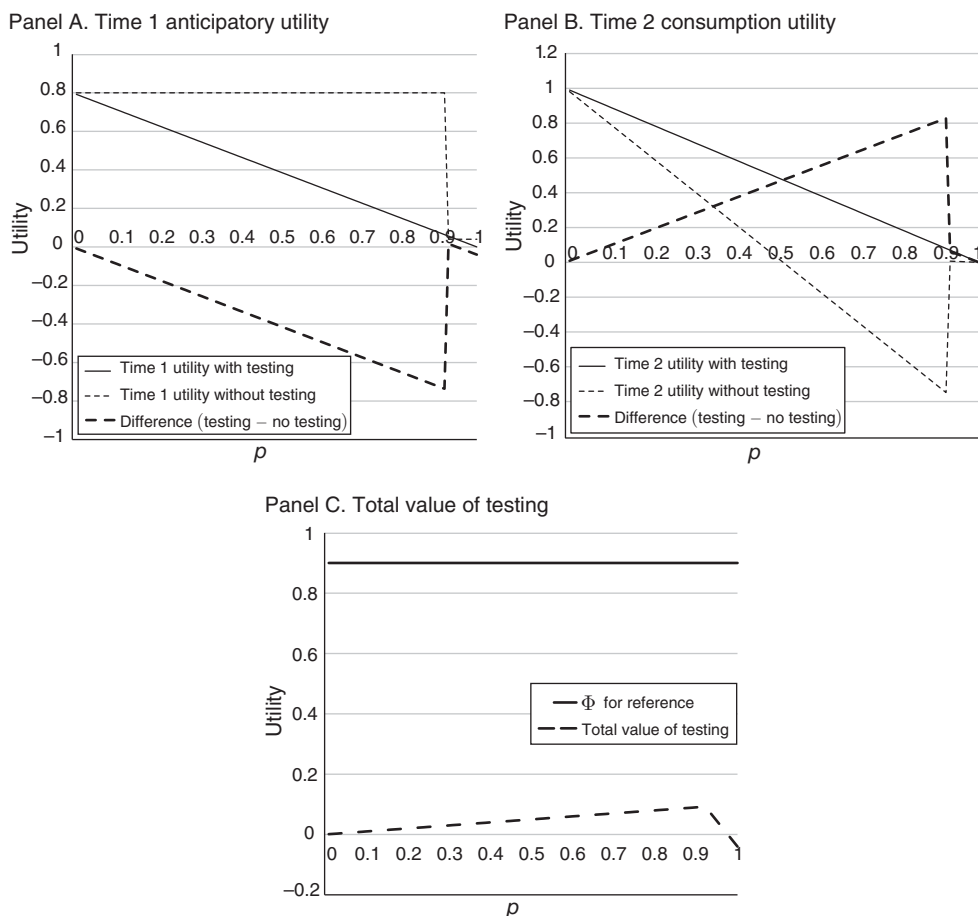


FIGURE 6. OPTIMAL EXPECTATIONS MODEL, GRAPHICAL INTUITION

Notes: These figures give intuition for the optimal expectation result on testing. Panel A shows the utility in the anticipatory period with and without testing. Panel B shows utility in the consumption period, with and without testing. In both cases the thicker dotted line shows the difference between the two utilities. Panel C shows the sum of the utilities; this figure also includes (for reference) a line showing the value of $\Phi = 0.9$. These graphs are made assuming symmetric losses from the wrong action ($\Phi = \Omega$) and assuming that $\delta < \Phi$. Under the chosen values of δ and Φ we have $p^* = 0.92$.

Extension: Confirmatory Testing.—We consider now a simple extension to the model: adding the possibility of confirmatory testing. We add to the setup the assumption that if the individual does turn out to have HD, there will be an incentive to undergo confirmatory testing, for example, to have “proof” for disability or other claims. Assume the value of this confirmation is Ψ . A reasonable assumption would appear to be that $\Psi < \Omega$. That is, if you turn out to carry the HD expansion, the value to taking the correct action at all times leading up to confirmation is higher than the value of the confirmation.

losses. We show the results are extremely similar in that case. More generally, the original version of the model developed in Brunnermeier and Parker (2005) delivers overly optimistic beliefs and skewed actions for a general choice of a . The result that testing is increasing in risk may hold in the general case, although it will not hold under all possible functional forms (details about the conditions under which this will hold are available from the authors).

Importantly, we are considering here an individual who believes that $p = 1$. This individual has no option to choose beliefs which differ from the true p , so that “cost” of testing is eliminated. Propositions 1 and 2 are identical with this modification. The key follow-up question is under what conditions will individuals test for confirmation but *not* engage in predictive testing. Are there parameter values such that people will avoid testing for all $p < 1$ and yet still be willing to test once they are sure they have the gene? The condition is summarized in Proposition 4.

PROPOSITION 4 (Confirmatory and Informative Testing): *Assume p^* is given as in Proposition 1. Individuals will engage in confirmatory but not predictive testing if $\Psi > C$ and one of the two following conditions holds:*

$$\delta \geq \Omega$$

$$\delta < \Omega \text{ and } C > \frac{(\Omega - \delta)(\Phi(\Phi + \Omega) + \delta\Phi(1 + \Omega))}{\Omega(\Phi + \Omega) - \delta\Phi(1 + \Omega)}.$$

PROOF:

Confirmatory without predictive testing requires that individuals experience all values of p up to $p = 1$ without testing, but they do want to test once there is no anticipation loss. The condition for wanting to test later is simply $\Psi > C$. For any $p < p^*$ the condition for preferring “test later” to “test now” is $C > \frac{p(\Omega - \delta)}{(1 - p)}$.¹⁴ If $\delta > \Omega$ then the right-hand side is negative and any positive value of C will satisfy this.

If $\delta < \Omega$ we note that the value of testing is highest at p^* . If the cost C exceeds the value at p^* , this will hold. Simplified, the condition for this is given above. Note that combined with the condition for ever wanting to test, this implies that Ψ is also greater than that expression.

There are parameter values under which individuals will engage in confirmatory but not predictive testing. Put simply: with confirmatory testing the real costs are the same, and the benefits are lower. However, when testing for confirmation individuals do not experience the cost associated with having to face the truth. If this cost is important enough, it may be that confirmatory testing is a good idea and predictive testing is not.

It is worth noting that this represents a departure from the optimal expectations framework, and the result here requires the assumption that there is some value to confirmation. Without this, this behavior cannot be explained by this model (although, as we note below, the neoclassical version of the model cannot explain confirmatory testing even *with* this assumption).

IV. Neoclassical Case

The evidence above suggests that both qualitatively and quantitatively, the optimal expectations model can fit the patterns we observe in the data. What we do not

¹⁴ This is derived by comparing the total utility from testing now (and making the correct choice) and testing later (and therefore possibly making the wrong choice, but nevertheless getting Ψ from testing later).

answer above is whether we could do as well, or almost as well, with the neoclassical, no-anticipatory-utility version of the model *if* we were willing to assume a higher cost of testing. That is, we can ask whether the only thing ruling out the neoclassical model is the need for a high cost of testing. If that is the case, the conclusion that people want to avoid information seems hasty; perhaps our impression of the cost is skewed.

To begin, Proposition 5 below summarizes the results of the model under the assumption of no anticipation.

PROPOSITION 5: *Assume that $\delta = 0$. Then:*

- (i) *Beliefs are accurate ($\pi = p$).*
- (ii) *Action $a = 0$ is taken as long as $p < p^*$ where $p^* = \frac{\Phi}{\Phi + \Omega}$. Note $p^* > 0.5$ if and only if $\Phi > \Omega$.*
- (iii) *The value of testing is increasing in p for values of $p < p^*$ and decreasing in p for values of $p \geq p^*$. This value is positive if $p < p^*$ and $p\Omega > C$ or if $p \geq p^*$ and $\Phi - p\Omega > C$.*
- (iv) *Confirmatory testing will occur without predictive testing if and only if $\Psi > \Phi$.*

PROOF:

These conditions follow directly from Propositions 1–4, with the assumption in each case that $\delta = 0$.

A. Evidence on Neoclassical Model

Beliefs: The neoclassical case does not accommodate the overly optimistic self-reported beliefs that we observe in the data. There is simply no sense in which individuals can hold beliefs which are different from the truth.

Confirmatory Testing: Recall the assumption that $\Psi < \Omega$. The neoclassical case allows for confirmatory testing only if $\Psi > \Phi$. Further, note that this model generates skewed actions only if $\Phi > \Omega$, so observing confirmatory testing would require $\Psi > \Omega$, which is in violation of the assumption.

Actions and Testing: Skewed action choices and the claim that testing is increasing in risk are both delivered in the model by a $p^* > 0.5$. As stated above, this will occur only if $\Phi > \Omega$, implying that it is much worse to take the wrong action if the true state turns out be “healthy” than if it turns out to be “sick.” To deliver the actual patterns in the data this asymmetry needs to be quite large: in order to have skewed actions up to $p = 0.9$, as we observe in the data, it must be the case that $\Phi \geq 9\Omega$.

We can frame the required difference in terms of timing. For example, we observe in the data that individuals who carry the HD expansion choose to retire earlier than those who do not. The data we observe would be generated, therefore, if people felt

it was much worse to retire too early than to retire too late. In principle, there is nothing that rules this out, and in some cases (retirement, for example) this assumption may seem reasonable.

We attempt to move slightly beyond introspection on these questions with a simple survey, run through Amazon's Mechanical Turk marketplace. We asked 300 individuals from the general population about the timing of marriage, childbearing, and retirement. For simplicity, we looked for direction rather than intensity of preference. Individuals were asked to imagine their optimal age for childbearing or retirement. We then asked them whether, if their optimal age was not possible, they would prefer to undertake the action too early relative to their optimal age or too late.

The data does not support the view that losses are asymmetric. On both outcomes, individuals are fairly evenly split between preferring to undertake the action too early versus too late: 57 percent on childbearing and 50 percent on retirement. Of course, there are many reasons why this exercise falls short of proving or disproving asymmetry in losses. Further work, ideally with the HD population and with better unidentified choices, would need to be done to draw conclusions.

Impact of Testing Costs: In the neoclassical case, avoidance of testing is driven only by cost. If the cost of testing were zero, everyone would test. Changes in the cost of testing should therefore have a large impact on testing behavior. This need not be true in the case with anticipation.

There are two things in the data which call into question the prediction of responsiveness to testing costs. The first comes from the comparison between Canada and the United States. Real costs of testing in Canada are likely to be lower still than those in the United States; with a national health care and disability plan, there is no concern about loss or denial of insurance with testing. This both removes one cost of testing *and* makes it less likely people will feel they need to pay out of pocket to keep their test results anonymous (Oster et al. 2008). Despite this lower cost, predictive testing rates are only slightly higher in Canada: in our data, about 7 percent versus 5 percent in the United States. Of course, this argument comes with the general caveats about cross-country data: there may well be other reasons why the countries are similar, even if cost of testing makes a difference.

The second piece of evidence comes from reported reasons for avoiding testing. At enrollment into the PHAROS study individuals are asked why they have not undergone genetic testing. They are provided with a list of possible reasons and asked to indicate the importance of each reason. One of the reasons given is, "The financial costs of testing are too high" and another is "The testing process takes a long time." Only 20 percent of individuals report that financial costs are a "Somewhat" or "Extremely" important reason to avoid testing, and only 8 percent of individuals report that time costs are an important reason. In contrast, 60 percent of people say that a preference for living with uncertainty is an important reason for not testing.

Together, this evidence suggests that the addition of these new facts, in particular the evidence on overly optimistic beliefs, more comprehensively rejects the neoclassical model. Even if we allowed for a large cost of testing, a model without anticipation still fails to fit the data.

B. *Alternative Nonneoclassical Models*

The optimal expectations model is not the only nonneoclassical candidate to explain these facts. In online Appendix B we describe two other models. We begin with a model of wishful thinking (Mayraz 2011). We find this model produces implications very similar to the optimal expectations case and in this sense is a plausible alternative. Little in our data distinguishes these two models; the one feature leading us to favor optimal expectations is that the Mayraz (2011) model requires higher real costs of testing to explain low testing rates and has difficulty accommodating confirmatory testing. Second, we describe a model with anticipatory utility and information-averse preferences (Kőszegi 2003). This model fails to match the bias in reported beliefs and requires the same asymmetry in utility losses that is necessary to explain behavior in the neoclassical case. We therefore argue the data more strongly reject this alternative.

A third class of models to consider are those with some type of procrastination or present-biased preferences. We could imagine individuals understand the value of information and yet avoid testing because it takes time and requires some action on their part. This would echo work on the stickiness of defaults and models of hyperbolic discounting (Laibson 1997; Choi et al. 2002; Choi, Laibson, and Madrian 2011). This setup has many of the same pitfalls as the neoclassical case. There is no scope for biased beliefs or skewed actions in a simple version; it would simply provide a way to explain why testing costs matter overmuch relative to the standard setup.

V. Discussion and Conclusion

The central puzzle with which we began this paper is low rates of medical testing. The analysis of HD data here demonstrates several additional stylized facts about testing and behavior. We argue that these facts are well explained by an optimal expectations model (Brunnermeier and Parker 2005). The primary reason for testing avoidance in this model can be summarized, colloquially, as not wanting to live with the anticipation of future ill health. This intuition aligns closely with a literature in psychology in which individuals facing bad news look for reasons to avoid believing it (see, for example, Dawson, Gilovich, and Regan 2002).

At the start of the paper we noted that the low testing rates in this setting are similar to what we see in other settings, such as HIV testing and genetic tests for cancer markers, which are of more policy relevance. In both of these settings the “real” costs of testing appear to play a limited role (Lerman et al. 1996; Thornton 2008). Thornton (2008), for example, finds that 20 percent of individuals in her study in Malawi avoid getting their HIV test results even though they would be *paid* to get them. Although our analysis has focused on the HD case, to the extent we would favor this model for explaining low testing rates more generally, it seems reasonable to ask whether there are behavior patterns in these other settings consistent with the facts we saw in Section II.

There is virtually no data in either case which discusses beliefs; even if there were, objective risk is difficult to define. There is similarly little or no information about actions like retirement or pregnancy among individuals with varying test results. What we *can* observe is information on follow-up health behaviors for individuals with varying risk. Several papers have looked at patterns of cancer screening after

testing for the BRCA gene. Individuals testing for this are told either they do not have the mutation (normal cancer risk) or they do (very high lifetime cancer risk). Untested individuals with a family history have an intermediate risk, similar to the HD case. Foster et al. (2007) analyze data of this type and find that cancer screening increases after a positive test result but is unchanged after a negative test result. This would imply that those with an intermediate risk behave like those with a negative result. Schwartz et al. (2003) find a similar result for ovary removal after BRCA testing; the rates of prophylactic ovary removal are similar for individuals with a negative test and those who are untested but are much higher for those with a positive genetic test (even though these individuals still do not have cancer, just an increased risk).

For HIV, the behaviors of interest tend to be risky sexual behavior and condom use. Both in Africa (Thornton 2008) and in data from the United States (Weinhardt et al. 1999) researchers find that individuals with a positive HIV test decrease their risky behavior after the test. However, those who have a negative test result do not change behavior relative to the pretest period. This again implies something similar to what we see in HD: differences in behavior between the tested-positive group and everyone else, but no difference between those who are untested and those who test negative.

When we turn to testing and risk, we have a natural measure of risk in the BRCA case: extent of family history. The more relatives have had breast or ovarian cancer, the higher the chance of the BRCA gene.¹⁵ In a number of studies of testing behavior it would appear that testing is increasing with risk according to this measure (Lerman et al. 1996; Meijers-Heijboer et al. 2000; Baer et al. 2010). Meijers-Heijboer et al. (2000) explicitly separates individuals at 25 percent risk from those at 50 percent risk and shows higher testing rates among the 50 percent risk group. The data on HIV is less helpful here, since studies tend to focus on self-perceived risk and testing, rather than objective risk, and risk of either sort is associated with high-risk sexual behavior (which may impact testing for other reasons).

This evidence is far from conclusive. The data are less rich than in the HD case, and the presence of reasonable treatments muddles the discussion of testing in particular. There is a sense in which the chance of actually realizing the bad state (p) is dependent on testing. Nevertheless, the fact that we see at least some evidence of similar patterns is suggestive of the idea that this model plays a role in driving low testing rates in these contexts, as well.

We can also ask what this model implies about policy. In some cases (i.e., HIV), low testing rates are a policy concern. The model allows us to think about how they might be increased. It is important to note that in the optimal expectations world, the individual choice to avoid testing is privately optimal. Individuals are not making a mistake, nor do they lack information: they are avoiding testing because they prefer to consume happiness in the anticipation period. Given this fact, we should be wary of inadvertent revelation of information about genetic status since it may make individuals worse off.

Higher testing rates may be socially optimal even if not privately in cases where there are externalities (through contagion, for example). The optimal expectations model suggests that actions like lowering the real cost of testing, or making it more

¹⁵ This is because the more relatives with cancer the more likely it is genetic, as opposed to random occurrences.

accessible, may have limited impacts on testing behavior. In contrast, increased testing might be achieved by making more salient the changes in behavior which could be undertaken if testing occurred.

A final note: the key assumption in the optimal expectations framework is that, once tested, individuals are no longer able to change their beliefs. This seems like an appropriate assumption in cases like HD (or HIV) where the test is fully accurate. A different assumption may be appropriate in cases where the test is not fully accurate. In this case we could imagine that the test restricts belief manipulation to some extent, but not completely. It is possible this would make the test more attractive. In a world in which actions are modified more continuously, this could have positive impacts because people may modify their behavior somewhat. In principle, this could mean there is some value to making a test that is less accurate.

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