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Genetic adverse selection: Evidence from long-term care insurance and Huntington disease

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ABSTRACT

Individual, personalized genetic information is increasingly available, leading to the possibility of greater adverse selection over time, particularly in individual-payer insurance markets. We use data on individuals at risk for Huntington disease (HD), a degenerative neurological disorder with significant effects on morbidity, to estimate adverse selection in long-term care insurance. We find strong evidence of adverse selection: individuals who carry the HD genetic mutation are up to 5 times as likely as the general population to own long-term care insurance. This finding is supported both by comparing individuals at risk for HD to those in the general population and by comparing across tested individuals in the HD-risk population with and without the HD mutation.

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1. Introduction

Personalized genetic information is increasingly available. Genes associated with increased risk of breast cancer, colon cancer, Parkinson disease and Alzheimer disease, among others, have been identified and testing for these genes is becoming much more common. Continued advances in technology and knowledge of the human genome are likely to bring even more sophisticated and precise testing, for these and other conditions.[1] This testing, in turn, is likely to increase the degree of private information that individuals have about their mortality and morbidity risks.[2] In this paper we explore the possible impact of this increased information on the markets for long-term care insurance.

There is a large literature in economics which estimates adverse selection in insurance markets.[3] Increasingly, the findings in this literature point to the importance of multiple dimensions of preference heterogeneity (Finkelstein and McGarry, 2006; Cohen and Einav, 2007; Cutler et al., 2008; Fang et al., 2008; Einav et al., 2009). Although private information about risk type can and does drive insurance purchases, this may be outweighed (or reinforced) by other dimensions of heterogeneity. For example, private risk information about mortality may be counteracted by the fact that people with a lower preference for risk also own more life insurance, and these people are, on average, *less* likely to die early (McCarthy and Mitchell, 2003). However, this heterogeneity may also work in the opposite direction – to reinforce adverse selection – for a product like annuities (Einav et al., 2009).

For long-term care insurance, which we consider in this paper, it appears that preference heterogeneity serves to offset adverse selection due to private information; studies typically find little or no correlation between ownership and risk realization(Finkelstein and McGarry, 2006; Cutler et al., 2008). However, while there is limited reason to expect changes in preference heterogeneity over time, increased availability of genetic testing has the potential to dramatically increase private information in this market. In principle, this information could change the overall correlation between ownership and payouts and have large effects on the viability of this market in the long-term, particularly as in light of legislation which limits insurer ability to observe individual genetic information (e.g. United States House of Representatives, 2007).

Evaluating whether we will see increased adverse selection as genetic information increases requires a setting in which (a) individuals have a large amount of private information, which the researcher can observe and (b) that this is information individuals would like to act on. This paper takes advantage of a setting in which both requirements are

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[1] For example, there has been significant recent work on genome wide association studies, which look at entire individual genomes to identify markers that are common in individuals with a given condition (e.g. Burton et al., 2007).

[2] Interestingly, it seems possible that much of this testing could take place outside of traditional clinical environments — for example, through companies like "23 and Me".

[3] See, for example, Finkelstein and McGarry, 2006; Cawley and Philipson, 1999; Finkelstein and Poterba, 2004; McCarthy and Mitchell, 2003; Chiappori and Salanie, 2000; Cutler et al., 2008; Smart, 2000; De Meza and Webb, 2001.

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satisfied, using a dataset on individuals at risk for Huntington disease (HD). We combine this with data on individuals without HD risk to estimate adverse selection in long-term care insurance.

HD is a degenerative neurological disorder caused by an inherited genetic mutation on chromosome 4 that affects roughly 1 in 10,000 individuals in Caucasian populations. Because of the inherited nature of the disease individuals have significant private information about their disease risk. Those with one parent with the disease know they have a 50% chance of developing it, and those who have taken a genetic test and carry the affected gene know they will develop the disease, assuming they do not die earlier from something else. A perfectly predictive genetic test for HD has been available since 1993. Further, the information is extremely relevant. Individuals who carry the HD genetic mutation begin to deteriorate neurologically (mentally and physically), typically between the ages of 30 and 50, and death follows an average of 20 years after onset. Individuals become increasingly disabled over this period (Walker, 2007). There is no cure for HD, and only limited treatment options. The long, likely expensive, disability period suggests that long-term care insurance would have significant value.

We focus on long-term care insurance in this paper for a number of reasons. First, we argue that it is a particularly clear case for exploring adverse selection, since the primary difference between individuals with and without the genetic mutation is the probability of needing long-term care. This is in contrast to life insurance, where individuals with the genetic mutation are more likely to die early but also experience very different income streams during their lifetime *as a result of the disease*. Second, most long-term care insurance is purchased through individual policies, enhancing the possibility for adverse selection heavily affecting the aggregate functioning of the market. Finally, although the long-term care insurance market is small relative to, for example, health insurance, it is still large in absolute terms – 1.2% of GDP in 2004 – and in the absence of private insurance coverage most expenditures are covered by the government, through Medicaid (Brown and Finkelstein, 2009).

We test for the presence of adverse selection in long-term care insurance using data from a prospective cohort study (PHAROS) of approximately 1000 individuals at risk for HD. At enrollment into this sample, individuals had one parent who had HD, had not undergone genetic testing and were asymptomatic, so their chance of carrying the genetic mutation is approximately 50%.[4] Participants have been re-surveyed approximately every 9 months from the time of enrollment (1999 or 2000) to the present, and over this period approximately 10% have pursued testing for the genetic mutation.

We perform several tests using these data. First, we compare insurance ownership among the population at risk for HD to individuals in the general population, drawn from the Health and Retirement Survey (HRS). Second, for a subset of the tested individuals we are able to observe their genetic status[5] and identify individuals who know they have the genetic mutation (100% risk) and those that know they do not have the mutation (0% risk). We compare each of these two groups to the untested individuals with 50% risk. Finally, we can compare individuals who have been tested and know they carry the genetic mutation to those who have been tested and know they do not. This last analysis is our cleanest test. However, the sample size in this final test is small: we have only 71 tested individuals for whom we observe genetic status versus roughly 900 at-risk individuals who choose not to be tested. For this reason, the comparison of untested individuals with the general population in the HRS provides an important part of the analysis, as it reinforces the findings with a larger sample size and more statistically robust claim.

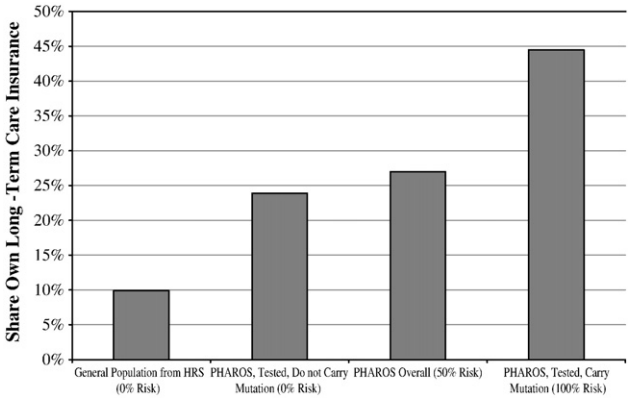

Fig. 1. Long-term care ownership, by HD risk. Notes: this shows a simple comparison of mean levels of ownership for individuals in the HRS, and those in PHAROS, by risk status.

Using these data, we find evidence of significant adverse selection. In the general population (the HRS), 10% of individuals own long-term care insurance. In the PHAROS population, 27% of untested individuals own long-term care insurance (this proportion is extremely similar to the overall proportion in the PHAROS population, since most individuals are untested). Among those who pursued genetic testing and know they carry the mutation responsible for HD, ownership is close to 50%.d1.51.5[6] The rate of long-term care insurance ownership among those individuals who know they are at 100% risk for developing HD is 20 to 30 percentage points higher than those who also pursued genetic test and know they do not carry the genetic mutation for HD. This primary result of the paper can be seen simply in Fig. 1.

An important issue in interpreting these results is whether individuals in the HD population are comparable to those in the HRS, other than their HD risk. The primary concern is that individuals in the HRS are, on average, older than individuals in the HD population. We attempt to address this by including controls for demographics and find that the effects of HD are not driven by demographic differences. In addition, comparing individuals within the HD at-risk population provides an extremely well identified test: these individuals are, in expectation, identical *ex ante* — nature "randomizes" which of them receive the affected copy of the gene. Finally, because long-term care insurance ownership tends to be higher among older individuals, the older population in the HRS suggest that, if anything, our results are an underestimate of adverse selection in long-term care.[7]

We add to a small existing literature on insurance purchases and genetic risk, which so far has had somewhat mixed results with small sample sizes (Armstrong et al., 2003; Aktan-Collan et al., 2001; Zick et al., 2000, 2005; Taylor et al., 2009).In the most closely related of these papers, Zick et al. (2005) show increases in long-term care purchases as a result of an intervention informing individuals about an increased risk for Alzheimer disease. The analysis here provides sharper evidence on this question, because HD has a clearer genetic risk, our sample is much larger and the population contains individuals at widely different levels of risk.

Our results may be particularly policy relevant in light of the recent health care legislation (United States Senate, 2010). The health care bill

[4] The fact that we observe only individuals who have not been tested at enrollment introduces possible selection issues, which are discussed in more detail in the context of the results.

[5] We infer individual test results based on subsequent disease development, or subsequent responses to questions about their self-perception of carrying the genetic mutation; this is described in more detail in the data section.

[6] Throughout the paper we will sometimes refer to individuals who are tested and know they carry the genetic mutation as having a "positive" test result.

[7] In an appendix we show estimates based on an alternative dataset (the Consumer Expenditure Survey), which is less well suited for many reasons, but does have a population with a closer age match. If anything, the results are stronger with this comparison group.

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