Education and Allocative Efficiency. Evidence from Breast Cancer Screening.

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Abstract

We study the role of education in promoting allocative efficiency in health care decisions using the breast cancer screening decision. We model the screening decision of individuals facing heterogeneous risks of developing breast cancer. Education is assumed to improve the accuracy of individuals' assessments of their risks. This induces a positive interaction between education and individual risk factors in the screening decision. Empirically, educated individuals are indeed more responsive to the presence of risk factors in their breast cancer screening decision. They also are more likely to adjust their subjective risk assessments to account for the presence of risk factors. This study therefore provides support for a role of education in promoting allocative efficiency in health care decisions.

1 Introduction

Education is the most important socio-economic correlate of good health (see eg. Grossman and Kaestner (1997)) There is however no consensus on what gives rise to the correlation between schooling and health. In fact, there is not even consensus on the causality of schooling for health. For public policy however it is crucial to establish both this causality and the channels through which education might affect health.[1]

Our paper tests whether schooling improves allocative efficiency in health decision making. Schooling is said to improve allocative efficiency if schooling improves the acquisition and processing of health information and consequently leads to a more efficient allocation of resources in producing health. We present a model of the cancer screening decision and use it to derive a novel test of the allocative efficiency of education hypothesis. This test examines the interaction between education and risk factors in the breast cancer screening decision.[2] In our model the accuracy of individuals information about breast cancer risk plays a crucial role We show individuals with more accurate assessments of their individual risk are more response to breast cancer risk factors present in the data.

Empirically we show that more educated individuals are more responsive to the presence of risk factors in their cancer screening behavior. This is consistent with the hypothesis that education increases allocative efficiency – more educated individuals are able to assess their risk with greater accuracy and therefore respond to the presence of risk factors. We also examine how self-reported cancer risk assessments respond to the presence of risk factors. More educated individuals are more likely to take objective risk factors into account when determining their individual cancer risk. We then implement a series of placebo tests using cancer screening for other types of cancers. The reason is

[1] Auster et al. (1969) for instance argue that it might be preferable to improve health by promoting schooling rather than by increasing outlays on public education.

[2] There are a number of advantages for studying health care demands using the breast cancer screening decision. Not least is the fact that the National Health and Interview Survey (NHIS) has made data on screening decisions, risk factors, and other variables related to breast cancer available in both 2000 and 2005. Furthermore, there is substantial medical knowledge about risk factors related to breast cancer. In particular, there is a well established model relating individual characteristics to breast cancer risk, the Gail model. We implement the Gail model using the NHIS 2000 and 2005 to provide an index of breast cancer risk. A further advantage of using the breast cancer screening decision is that it is a decision faced by asymptomatic individuals, thus avoiding problems of selection bias inherent in samples of individuals diagnosed for various diseases.

that we want to rule out spurious correlations arising through unmodeled interactions between education and determinants of breast cancer risk in health demand. We find that the decision to screen for colorectal cancer as well as cervical cancer does not display a similar interaction between breast cancer risk factors and education. Together this set of results supports the notion that education increase allocative efficiency in health decision making.

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Our investigation into the allocative efficiency of education is structured as follows. Section 2 reviews the literature on health demands. Section 3 presents the model of health screening and shows that individuals with lower individual signal variance will tend to be more responsive to risk factors. In Section 3 we also derive our test of the allocative efficiency hypothesis: this involves examining the interaction of education and the index of individual risk factors in the latent index governing the screening decision. Section 4 presents the data for our analysis. This data is analyzed in Section 5. We show that individual cancer risk and education interact positively in the cancer screening decision and that this result is robust to various alternative specifications. We also show that individual risk assessments are more responsive to risk factors for more educated individuals. Both of these findings support the notion that individuals with more education are better informed about their individual risk and that they act on this information when making screening decisions. We consider evidence on alternative explanations. Section 6 concludes.

2 Literature Review

The literature on the demand for health is vast. One strand of the literature directly models the willingness to pay for mortality reducations. This literature builds on the hedonic model (Rosen 1974) to provide estimates of the willingness to pay for mortality reductions. These estimates can then be related to and interpreted within standard life-cycle models of earnings and consumption. Murphy and Topel (2005) and the survey by Kip Viscusi (1993) provide good starting points to learn about this approach to the demand for health.

An alternative, complementary approach to this literature is based on the capital theoretic model by Grossman (1972a and b). This model illustrates the main linkages between human and health capital investments. Grossman endogenizes health by modeling it as a capital stock subject to investments. Health investments are the outcome of a production process taking both medical expenditures and time as its inputs. Education is often assumed to affect the efficiency with which medical expenditures and time are mapped into new units of the health capital stock. Grossman therefore distinguishes between medical expenditures and health. Health capital is valuable because it produces "healthy" time and because it enters the utility function directly. "Healthy time" can be sold in the market to generate earnings, or used in home production of both the stock of health and other commodities. If we specialize the model such that health does not enter the utility function of individuals directly, then we speak of the investment model.[3] The stock of health capital depreciates over time and the rate of depreciation increases with age. Eventually the stock of health declines below a minimum level and the individual dies.

Grossman's model generates various interactions between education and health, many of which would on their own generate a positive relation between education and health. We refer the interested reader to Grossman (2000) and Grossman and Kaestner (1997) as valuable surveys of the literature on the link between schooling and health and here simply list the most important linkages. First, education and health are both outcome of an investment decision. Thus, variables such as interest or discount rates are driving both relations and will

[3] The model where Health only affects the utility function is called the (pure) consumption model. The introduction of health as an argument in the utility function complicates the analysis and as Grossman (2000) states, "the investment model rather than the consumption model generates powerful predictions from simple analysis and innocuous assumptions". We therefore limit the discussion initially to the investment model and will refer to the consumption model only in passing.

plausible generate positive correlation in health and education. Second, health and education investments are complements since a longer life horizon extends the period during which education generates benefits to individuals. And, conversely, education as an investment defers earnings and thus raises the benefits from investing into health. Third, positive income effects of education directly raise the marginal rate of substitution of consumption for mortality reduction. Fourth, health investments are produced using individual effort and time as well as medical inputs. Education might increase the efficiency of time in making health investments and thus lower the effective price of health (see Michael, 1972; Michael and Becker, 1972). Grossman (1972b), Wagstaff (1986) and Erbsland (1995) test for productive efficiency within the context of Grossman's health capital model and find some, albeit far from conclusive evidence for the hypothesis that schooling raises productive efficiency.

Related to productive efficiency is the concept of allocative efficiency (see the review by Kenkel (2000)). This is the notion that education enables individuals to process information more efficiently and promotes better use of limited resources in producing health. We test for allocative efficiency in this paper.

Our paper builds on, and extends, the literature on preventive health care. A number of studies link schooling to increased preventive activities. Leigh (1990) finds that schooling raises the propensity of using seat belts, while Kenkel (1991a, 1991b) finds that schooling leads to healthier choices regarding smoking, drinking, and exercise. Interestingly he also finds that health knowledge leads to healthier behaviors and that schooling and health knowledge correlate positively.[4] This provides some support for the hypothesis that schooling increases allocative efficiency in health decisions. Kenkel (1994) also finds that schooling increases the propensity of women to engage in cervival and breast cancer screening - a result that we confirm in our data. Finally, Mullahy (1999) examines the propensity for influenza immunizations and likewise finds that schooling increases the propensity to receive such shots.

The simple observation that more educated individuals are healthier or engage in healthier activities does not settle the issue in favor of the allocative efficiency hypothesis. It is clear that schooling and health behaviors might be

[4] A number of researchers find that health knowledge about preventive care and life cycle choices is imperfectly distributed in the population. Viscusi (1998) for instance reports that most people overestimate mortality risks due to smoking. A different type of misperception is a disconnect between perceived health risks in the population and subjective health risks of individuals. Schoenbaum (1997) documents such a disconnect for heavy smokers.

correlated for various reasons, even if schooling itself has no impact on either productive or allocative efficiency in health decisions. The positive correlation between health knowledge and schooling observed in Kenkel (1991a, 1991b) therefore remains so far among the most convincing evidence in favor of an allocative efficiency role of schooling. Other evidence in factor of the allocative efficiency hypothesis is provided by De Walque (2004a). He examines smoking behavior between 1940 and 2000. In the decades following the Second World War a consensus started to form on the harmful health consequences of smoking. De Walque observes that prior to the 1950s more educated individuals were more likely to smoke than less educated, while this gradient has since then reversed.[5] Today more educated individuals are much less likely to smoke than are the less educated. This is consistent with educated individuals being more alert to new information, however it is also consistent with some of the other hypotheses linking health demand and education.

We propose to provide additional evidence by examining directly how the decision of allocating health inputs varies with schooling and whether more educated individuals are more likely to allocate scarce resources to activities with the highest expect health benefits.

3 Model

3.1 The information model

In our model, people as well as the econometrician have information about their likelihood of developing breast cancer. Neither party can predict cancer perfectly, but both make inferences based on the presence of personal risk factors, some of which are genetic and some of which are behavioral. Our model links individuals' forecasts of developing breast cancer and the information on risk factors present in the data.

We model the likelihood of developing breast cancer by a latent ("true") state variable t which lives on the real line. As modeled, this latent state variable perfectly describes breast cancer risk in the sense that a person will develop breast cancer if and only if their state t is less than 0. There is uncertainty about whether any individual will develop breast cancer though; this uncertainty is

[5] Interestingly, De Walque (2004b) also documents a similar pattern in sexual behaviors in Uganda during beginning and subseqent to the HIV Epidemic.

modelled by a distribution of beliefs about the true state t. In our model t is normally distributed as $N(1, \sigma_t^2)$, implicitly assuming that the uncertainty of whether a person will develop breast cancer derives from the sum of a number of smaller unknowns. To set the mean to 1 is simply a normalization as long as the risk of breast cancer is less than 50% in the population.[6] This implies that we can fit any population-wide risk of breast cancer by appropriate choice of σ_t^2.

Agents in this economy draw a signal s on the true state. This signal is normally distributed around the true state t such that the signal error $\varepsilon_s\tilde{}N\left(0, \sigma_s^2\right)$. Individuals update expectations using Bayesian rules. Thus, agents posterior distribution of the state variable is:

$$t|s\tilde{}N\left(1+\phi\left(s-1\right), \frac{\sigma_t^2\sigma_s^2}{\sigma_t^2+\sigma_s^2}\right) \tag{1}$$

with

$$\phi = \frac{\sigma_t^2}{\sigma_s^2+\sigma_t^2}$$

Agents update their beliefs more (ϕ is larger) if the signal variance is low relative to the variance of the prior. And, the posterior variance declines in the signal variance.

The NHIS records various risk factors for breast cancer. Following standard practice in the medical literature we aggregate these risk factors into a single number, the Gail Index. The Gail Index is based on evidence from several large scale breast cancer and screening effectiveness studies and generally accepted in the medical literature (see Gail et al. 1989, Gail et al. 1999) as the appropriate measure of relative breast cancer risk across individuals.[7] In Section 4 we will describe this index in more detail; we are able to construct this index based on the survey responses recorded in both the 2000 and 2005 cancer control module

[6] The ten-year risk of breast cancer varies between 0.1 and 15% in the population. A woman of age 50 with no additional risk factors has a ten year risk of developing breast cancer of about 2% (see Gail, 1999).

[7] The fact that the Gail Index is widely accepted as the appropriate model of breast cancer risk is indeed the reason the various risk factors that are required to compute the Gail Index were elicited by the 2000 and 2005 Cancer Control Modules of the National Health and Interview Survey. These are age, number of direct relatives with a diagnosis of breast cancer, age at first menstruation, parity and age at first birth, and race.

More recent models of breast cancer risk seek to refine the Gail index using additional risk factors. The risk factors entering the Gail Index are however generally accepted as crucial for determining relative breast cancer risk.

of the NHIS.

The Gail Index is a measure of relative risk - a woman with a Gail Index of 3 is deemed 3 times more likely to develop breast cancer than a woman with a Gail Index of 1. To map this into our latent state structure we presume that the Gail Index is derived from an underlying signal s_G which itself is distributed $s_G \tilde{} N\left(t, \sigma_G^2\right)$. The posterior distribution of the latent variable t is then:

$$t|s_G \tilde{} N\left(1 + \phi_G\left(s_G - 1\right), \frac{\sigma_t^2 \sigma_G^2}{\sigma_t^2 + \sigma_G^2}\right) \tag{2}$$

with

$$\phi_G = \frac{\sigma_t^2}{\sigma_G^2 + \sigma_t^2}$$

Conditional on the "Gail signal" s_G, the breast cancer risk for any individual is given by the CDF of the random variable in equation 2, evaluated at 0. For any σ_G^2 this risk is monotonic in s_G, therefore we can invert the breast cancer risk associated with any Gail score back into a unique s_G.

Below we show how we can identify the parameters $\left(\sigma_G^2, \sigma_s^2, \sigma_t^2\right)$ using the observed breast cancer risk, the distribution of the Gail Index and the variation in screening rates with the Gail Index. For this purpose we first need to link the information structure to the individual decision problem.

3.2 The individual decision problem

We will start by describing an agent's decision problem conditional on her subjective risk assessment. Individuals are willing to trade off income and mortality risks. The willingness to pay for a small reduction in mortality is known as the Value of a Statititistical Life (VSL). An individual will forego an opportunity to reduce her mortality by a small amount if the price of this investment falls short of the Value of a Statistical Life.[8]

For our analysis this means concretely that an individual i will choose to

[8] The Value of a Statistical Life(VSL) is a standard concept in Health economics. It describes the trade-off between consumption and mortality risk and is typically estimated using hedonic techniques applied to employment risk. Murphy and Topel (2006) provide a model of the VSL that links the willingness-to-pay to life-cycle consumption and income profiles as well as the mortality risks faced by individuals. Their model relies on the availability of actuarially fair annuities and time-additive preference. One can model the VSL in other settings. Our analysis does not require taking a stand on the determinants of the VSL as long as one can define such a willingness-to-pay for mortality reductions.

screen if the following inequality holds:

$$\frac{p}{VSL_i * \Pi_i} \leq \Phi_{AgPt}\left(0; s_i, \sigma_s^2\right) \tag{3}$$

where $\Phi_{AgPt}(\cdot)$ is the posterior of the agent as given in expression (1), p is the cost of screening, and Π is the percent reduction in cancer mortality due to early diagnosis conditional on having cancer. The individual will screen if (i) she perceives her likelihood of having breast cancer to be high (ii) the costs of screening are low relative to the mortality gains available from screening and (iii) the Value of a Statistical Life is high – that is the marginal rate of substitution of consumption for survival is high. Let us denote the LHS in expression (3) as ξ_i.

3.3 The response of screening to the latent state t and the Gail signal s_G.

We are now in a position to consider how the screening probability is related to the individual signal s and the Gail signal s_G. This signal s_G is not directly observed by agents. Instead, the screening decision will respond to s_G only because both s and s_G are measures on the underlying latent state t. How tight the link between s_G and s is depends on the signal variances σ_G^2 and σ_s^2. It is the latter that captures the idea that individuals are imperfectly informed. In our model the hypothesis that education improves decision making is identical to the hypothesis that σ_s^2 declines with individuals' education. We test for allocative efficiency by testing for a negative association between σ_s^2 and education.

Consider inequality (3). The left hand side is strictly declining in s, which implies that a woman's screening decision can be described by a cut-off for the signal s: $\theta = 1 \Leftrightarrow s \leq \widetilde{c}\left(\sigma_s^2, \xi\right)$. This cut-off depends on σ_s^2, because σ_s^2 affects the responsiveness of the posterior mean $E[t|s]$ to s, but also because it affects the posterior variance $V(t|s)$. In addition, this cut-off will increase as individuals' willingness to pay for mortality reductions ξ increases.

We can thus express the decision to screen as

$$\theta_i = 1 \Leftrightarrow \varepsilon_s \leq \widetilde{c}\left(\sigma_s^2, \xi\right) - t_i \tag{4}$$

Unfortunately, the true state variable for health t_i is not observed. Given the structure of the information model above we have however $s_G\tilde{\ }N\left(t, \sigma_G^2\right)$ and thus $t_i = s_G - \varepsilon_G$ where ε_G is the error for the Gail signal. This error is independent of the signal error ε_s (we will explore the implications of relaxing this assumption in the next section). Inserting into equation (4) delivers:

$$\theta_i = 1 \Leftrightarrow \varepsilon_s - \varepsilon_G \leq \widetilde{c}\left(\sigma_s^2, \xi\right) - s_G \tag{5}$$

$$\Leftrightarrow u \leq c\left(\sigma_s^2, \xi\right) - \frac{1}{\sqrt{\sigma_s^2 + \sigma_G^2}} s_G \tag{6}$$

where the disturbance u is $N(0,1)$ and $c\left(\sigma_s^2, \xi\right) = \frac{1}{\sqrt{\sigma_s^2+\sigma_G^2}} \widetilde{c}\left(\sigma_s^2, \xi\right)$.

Equation (5) sets up a probit on variables affecting $c\left(\sigma_s^2, \xi\right)$ as well as s_G. The coefficient on s_G identifies $\frac{-1}{\sqrt{\sigma_s^2+\sigma_G^2}}$. It is this probit that provides the empirical specification that we will implement below. In particular, we will use the information on individual risk factors available in the data to test the hypothesis that the coefficient on s_G declines in education.

Equation (5) refers to the index function in the probit specification. Our model therefore leads us to test for allocative efficiency using the index function itself rather than marginal effects. This in fact has real economic content. It is possible that more educated individuals are more responsive in updating their risk assessments due to the presence of risk factors, and at the same time the probability of screening varies less with risk for educated than less educated individuals. This case can arise if the overall screening rates for educated are substantially greater than for the less educated – in that case it is possible that there is simply no 'room' to react for the more educated. Our theory is clear that even in this case we can test for allocative efficiency and it requires examining the probit index, rather than the marginal effects.

The next section generalizes our results to the case when individuals signals and the gail signals are correlated. The basic result and the specification of the empirical test are unchanged.

3.4 Correlation between private and public information

Note that so far, we have assumed that the agent's signal s and the Gail signal s_G are uncorrelated signals drawn around the true state t. This allowed us to simplify the individual's decision problem significantly, but may seem unrealistic especially given that the information summarized by the Gail signal is known to agents (often the Gail measure is constructed from self-reported information about a patient). This suggest that the agent's decision may be correlated with the part of the error in the Gail score. To relax this assumption we can augment our standard model to allow agents in this economy make decision based on two signals, a private signal s_p on the true state t, and a noisy draw on the Gail signal s_G.

For simplicity assume the signal s_1 is normally distributed around t with signal error $\varepsilon_1\tilde{}N\left(0,\sigma_1^2\right)$, and the second is distributed around s_G with signal error $\varepsilon_2\tilde{}N\left(0,\sigma_2^2\right)$ Intuitively, agents have both private information on their health states and also have (possibly imperfect) access to what their doctors (and the econometrician) know. These signals would naturally average to the composite signal s we modeled before, but allow for correlation between s and s_G. More concretely, we can solve for the weighted sum of private signal s_1 and the noisy public signals $s_G+\varepsilon_2$ which would make observing these or the signal s,equivalent under Bayes rule. This gives us:

$$s = \alpha * s_1 + \beta * (s_G + \varepsilon_2) \tag{7}$$

$$= t + \alpha\varepsilon_1 + \beta\left(\varepsilon_G + \varepsilon_2\right) \tag{8}$$

$$\text{where } \alpha = \frac{\sigma_2^2+\sigma_G^2}{\sigma_1^2+\sigma_2^2+\sigma_G^2} \quad \text{and} \quad \beta = \frac{\sigma_1^2}{\sigma_1^2+\sigma_2^2+\sigma_G^2}$$

and where s is distributed as:

$$s\tilde{}N\left(t,\alpha^2\sigma_1^2+\beta^2\left(\sigma_G^2+\sigma_2^2\right)\right) \tag{9}$$

That is, that we can re-write our previous variance σ_s^2 as a weighted sum of three separate variances σ_1^2, σ_G^2, and σ_2^2. The individual's signal error ε_s can be written as the weighted sum of the Gail error ε_G and two private error terms, ε_1 and ε_2. The point is that we can model the individual's information as derived from two signals - one independent of the s_G (conditional on t) and one as a signal on s_G. Equivalently we can model the individuals information as a single signal on t which correlates with the gail signal and has the variance given in equation (9) . The individual's decision can be rewritten using equations (8) and (4) as:

$$\theta_i = 1 \Leftrightarrow \alpha * \varepsilon_1 + \beta * \varepsilon_2 - (1-\beta) * \varepsilon_G \leq \widetilde{c}\left(\sigma_s^2, \xi\right) - s_G \tag{10}$$

$$\Leftrightarrow \quad u \leq c\left(\sigma_s^2, \xi\right) - \frac{1}{\sqrt{\alpha^2\sigma_p^2 + \beta^2\sigma_{ed}^2 + (1+\beta^2)\sigma_G^2}} s_G \tag{11}$$

$$= \quad c\left(\sigma_s^2, \xi\right) - \frac{1}{\sqrt{\sigma_s^2 + \sigma_G^2}} s_G \tag{12}$$

Therefore the specification estimated here is exactly the same as previously, but takes on a new interpretation. If we find that σ_s^2 declines with education, then this can be due because individuals have better information about the gail signal or because they have better information about risks that are not modeled by the Gail Index. Empirically both the model with correlated and with uncorrelated signals map into the same specification.

3.4.1 Interaction of education and screening responsive to the Gail Score

Examining the empirical specification (5) we see that education may affect the decision to screen in several ways. Education would affect the cutoff screening value c through the value of a statistical life ξ, as well as through σ_s^2. Note though, that if the education of the individual is not correlated with the informativeness of their Gail signal (if education is independent of σ_G^2) then any relationship between education and the responsiveness of screening to the Gail score must be driven by education's effect on either the accuracy of their private health knowledge, or the degree to which they incorporate publicly known medical information. So, for example, if all significant risk factors for breast cancer are captured by the Gail score, then $\alpha^2\sigma_p^2$ will be close to zero, and any load on the interaction between the Gail signal and education will be due to the effect of education on σ_{ed}^2.

However, it is of course possible that spurious correlation results in biased estimates. That is, it is possible that the Gail signal interacts with education in $c\left(\sigma_s^2, \xi\right)$ in ways not captured by our model. For instance, it is possible that the term $\xi = \frac{p}{VSL_i * \Pi_i}$ depends on both education and on the variables that go into the construction of the Gail signal. To address this concern we exploit that fact that ξ will also affect other cancer screening decisions in the same direction as it impacts the breast cancer screening decision. This suggests examining the interaction between the Gail signal and education in screening decisions unrelated to breast cancer risk. If our findings for breast cancer risk are due to spurious correlation between ξ and s_G, then we expect similar effects to arise when examining the decision to screen for colorectal cancer or cervical cancer. We implement these placebo tests in Section (4.3) below.

4 Data

The National Health and Interview Survey (NHIS) is an annual household survey of the civilian, non-institutionalized population of the US. The NHIS records demographic and socio-economic data as well as data on health behaviors, health status, and access to health care. In selected years additional modules are administered as part of the NHIS. The 2000 and 2005 Surveys include a cancer control module. Our analysis uses these two waves.

In both years about 40,000 families with a total of 100,000 family members were interviewed. In each household one adult (the "sample adult") and one child (the ”sample child”) are asked a more detailed set of questions. In 2000 (2005) there were 32,374 (31,428) Sample Adults. We are limiting ourselves to non-hispanic sample adult females. Only women aged 30 and older were asked questions relating to breast cancer screening. This leaves us with 11,764 (11,726) women aged 30-85 in 2000 (2005). Dropping individuals with invalid answers about education, whether they ever had cancer, and on whether they have had a mammogram removes 75, 6, and 271 (2005: 125/13/871) observations respectively. A further 335 (369) women report having had breast cancer and are likewise dropped. In order to construct the Gail Index we require the age of onset of menstruation, information on whether a woman has ever given a live birth and at what age, and also the number of direct family members (parents, siblings, and children) who have ever developed breast cancer. Insufficient or incoherent responses for these variables removes another 698 (680) individuals. We thus retain 10,379 (9,668) women in the appropriate age range.

Among the socio-economic variables used in the analysis are education, a categorical variable on family income (as ratio to poverty line), the size of the MSA the woman resides in, and various variables describing the health care coverage (medicare, private, etc...). We also use answers on whether the woman had ever undergone a mammogram and as an alternative independent variable the number of mammograms the woman has received during the last 6 years. This variable allows us to examine how the intensity of cancer screening varies across individuals. We are also analyzing responses to questions concerning womens' subjective cancer risk. In 2000 we have a categorical variable (low, medium, high) describing the subjective overall cancer risk and in 2005 we have a similar variable describing the subjective breast cancer risk. Finally, we are using a variable that indicates whether a woman has been counselled by her physician to receive a mammogram. Unfortunately, this question was only adminstered to those women in 2000 who have not been screened previously. Thus, we use this variable only in 2005.

Table 1 presents summary statistics of the variables used in the analysis. The distribution of socio-economic variables is typical for the population of women aged 30-85. Screening rates are fairly high and increasing. In 2000 about 73 percent of all women has ever received a mammogram. In 2005 this number is 75%, The Gail Index is our measure of the medical information on risk factor available. It is a constructed variable based on whether women have ever given birth, their age at first birth, their age at first menstruation, the parity, their family history of cancer, their race and age. It measures the relative risk of breast cancer relative to a woman without an individual history of unusual screens, wihout a family history of breast cancer, with menarche after age 13 and with a first birth before age 20. In addition the past frequency of positive breast cancer screens is included in the construction of the Gail Index. Our main dependent variable refers to whether the women has every received a cancer screen. We can therefore calculate the Gail Index without the measures refering to the frequency of (positive) biopsies. Our measure of the Gail index is therefore an 'abridged' Gail index. We will refer to it as the Gail index from here on out. Table 2 summarizes the Gail model that allows us to construct the Gail index. The information about breast cancer risk summarized by the Gail model goes beyond a list of risk factors and also includes the size of the impact of these risk factors and the interaction with other risk factors.

The average Gail Index in the population is slightly greater than 1. It increases between 2000 and 2005. This is driven by the observed decline in age at menarche, number of individuals with directfamily members that had breast cancer and the increase in age at first birth. There is quite a bit of variation in the Gail Index in the population. The standard deviation is about 0.8 in both years.

5 Empirical Specifications and Results.

5.1 Responsiveness of Screening to Risk Factors

Equation (5) leads us to estimate a probit model as the basic empirical specification. The latent index is additively separable in the Gail signal and the cut-off $c\left(\sigma_s^2, \xi\right)$. The cut-off depends on the determinants of health demand that enter ξ as well as the signal variance σ_s^2. However, the functional form is unknown. We specify this cut-off as a function of various variables describing the costs of screening and variables that presumably affect the willingness-to-pay for health, including education. The hypothesis we are considering is that the signal variance σ_s^2 declines with education, indicating that more educated individuals have better information about their breast cancer risk. We include an interacted function of the index of risk (the Gail Index) and education interactions and test the null hypothesis that the interaction is positive. Note that equation (5) implies that we have to test for this interaction in the latent index, not the actual screening probability itself.

The main specificaiton we consider therefore takes the following form:

$$\theta = 1 \Leftrightarrow \beta_x' x + \beta_G * GI + \beta_s * S + \beta_{GxS} * S * GI + \varepsilon > 0 \tag{13}$$

where the disturbance is normal. We estimate this specification using a probit function where the dependent variable is an indicator for whether a woman has ever received a screen. The latent variable specification allows for various controls x, schooling S, the gail index GI and, crucially, the interaction $S * GI$. A positive estimate of β_{GxS} indicates that more educated individuals are more responsive to the presence of risk factors in their screening decision. The data however contains more information. In particular, it contains information on the number of screens received during the last 6 years. In order to incorporate this information on screening intensity we also consider a tobit where the dependent variable θ_6 indicates the number of mammograms a woman has received during the course of the last 6 years. The specification of the latent variable of this Tobit model is the same as that in eq. (13).

Table 2 Panel 1 presents the parameter estimates for both specifications obtained on the 2000 data. In columns 1 and 6 (our baseline) the control set includes a set of dummies for the income variable.[9] The baseline specification

[9] The income variable is a categorical variable that reports family income as a multiple of

also includes a full set of dummies for age and race. In order to provide an indication of the magnitude of events we report marginal effects for the probit specifications in table 3.

Table 2 and 3.here

At the mean of schooling (~13 years of schooling) we observe that both education and elevated breast cancer risk raises the screening propensity. Neither of these findings is informative on the question whether schooling improves health decision making. The estimated interaction between education and the Gail Index represents the first empirical finding directly related to this question. We find, and this finding is robust across most specifications we are considering, that more educated individuals respond more to the presence of risk factors than less educated individuals. The interaction term reported in column 1 is positive and highly significant. The effect on the propensity to screen (the marginal effect) reported in table 3, column 1 is not directly relevant for our purpose but gives an indication for the size of the effect. An increase in the Gail Index by one unit (doubling the relative risk of breast cancer) raises the probability of screening by about 3 percentage points more among the college educated than among woman with only a high school degree. This interaction is not negligble given the relative high overall rate of screening in the population (~73%). The findings are similar when we consider the intensity of screening using the Tobit specification (column 6).

The remaining columns consider alternative specifications. Columns 2 and 7 relax the functional form by controlling for a full set of education dummies and a quadratic for the Gail Index. All specifications include full sets of family income dummies.[10] In columns 3 and 8 we introduce interactions between the family income variables and the Gail Index. The results indicate that increasing incomes likewise raise the responsiveness to the presence of risk factors. At the same time the point estimate of the interaction between education and the Gail Index weakens somewhat, but remain marginally significant. It is not possible to reject that the estimated interaction in column 3 is the same as the coefficient in column 1.

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the poverty line. The highest category represents incomes 5 times above the poverty line. There is a sizable number of invalid answers for this question and we include a dummy for missing answers.

[10] Family income is measured as a categorical variable relative to the poverty line.

At first glance it might seem worrisome that the interaction between income and the Gail is likewise positive. However, the large majority of income is generated through earnings (past or present) and therefore income can be interpreted as an alternative measure of human capital. Thus, the results in columns 3 and 8 are consistent with the interpretation that education as well as other forms of human capital increase the systematic component of screening decisions – they raise the responsiveness of women to the presence of risk factors. Columns 4 and 9 control for health care coverage. We find that the results are robust to all of these variations in functional form and control sets. The results in columns 5 and 10 utilize a variable reflecting the subjective risk assessment of individuals and are presented here for future reference.

Table 2, Panel 2 presents the results for the 2005 data. These are similar to the ones obtained for 2000. More educated women are more responsive to the presence of risk factors (columns 1 and 7). This is robust to relaxing the functional form (columns 2 and 8). Women with higher family incomes are likewise more sensitive to the presence of risk factors (columns 3 and 9), and this lowers the education-Gail interaction in the probit specification. Nevertheless, the education-Gail interaction when accounting for the intensity of screening remains strongly positive. In 2005 we have more detailed information on the type of health coverage. This allows us to include sets of dummies for not only the presences of health coverage, but also for the type of health care coverage (medicare, private insurance, etc...). Again, our coefficient of interest on the education-Gail index is not sensitive to including these controls (columns 4 and 10). The 2005 survey also asked all women whether a doctor had recommended them to receive a mammogram within the last 12 month. Controlling for this direct measure of the role of health care intermediary does also not substantially affect the results (columns 5 and 11). Again, we will interpret the results controlling for self-assessed cancer risk (columns 6 and 12) at a later point.

Overall this first examination of the data therefore suggests that education increases the responsiveness to the presence of breast cancer risk factors as measured by the Gail Index. This is consistent with the notion that education improves the health care decisions made by women and the model of the screening decision outlined in Section 3. This first examination of the data however also reveals that women with higher incomes do likewise respond more to the presence of risk factors. We presume that family incomes are at least partially reflective of womens human capital. Given this interpretation the data supports the hypothesis that human capital overall, not just education, serves to increase the responsiveness to the presence of risk factors.

5.2 Subjective Risk Assessments and Education

The 2000 and 2005 data also allow us to directly examine the role education plays in forming subjective cancer risk assessments. In 2000 women responded on a 3 point scale to the question whether they believed themselves to be at high, medium, or low risk of developing cancer overall. In 2005 they answered the question whether they believed their risk of breast cancer to be higher than, equal to, or lower than the average risk of breast cancer. We examine directly the relation between education and risk assessment of individuals. In particular, if women with higher education do more accurately predict their individual cancer risk, then the relation between risk factors as measured by the Gail Index and education should also be stronger among more educated individual. The results of estimating ordered probits for the risk assessment confirm this hypothesis for both the 2000 (Table 4, Panel 1) and the 2005 (Table 4, Panel 2) data.

Table 4 here

Therefore both the individual screening behavior decisions and the reported risk assessment data suggest that education indeed increases the responsiveness of individuals to the presence of risk factors. Education does appear to raise the ability of individuals to arrive at more acurate assessments of their own breast cancer risk. And, this more accurate subjective risk assessment translates into screening behavior that is responsive to the presence of risk factors. The reduced form results presented here therefore are consistent with the notion that education indeed improves allocative health decision making.

5.3 Placebo Test

We next present results from a placebo test using other forms of cancer screening. To understand the rationale behind examining other cancer screening behavior, consider again equation (5) and also the components of the gail index presented in Section @. The Gail Index is constructed using a variety of behavioral variables as well as the incidence of breast cancer in the family. It is not possible to rule out that these risk factors are related to education or the willingness to pay for mortality reductions ξ in unknown ways. This implies that the cut-off c might be a complicated function of education and either the variables entering the Gail Index or variables correlated with these. Even if we do not have any reason to expect a positive interaction between education and the Gail Index, we can not rule it out.

However, the cut-off $c(\sigma_s, \xi)$ in equation (5) represents a measure of health demand that will affect other preventive health care measures in a similar manner. For instance, the willingness-to-pay for mortality reductions ξ might be a function of education and one of the variables determining the gail index. In this case, a similar decision rule for any other cancer screening method will likewise deliver a cut-off depending on ξ. And, we would expect the same relation between education and the determinant of the Gail Index to affect not just Breast Cancer Screening, but also other cancer screening methods.

We consider colorectal examinations as well as home fecal occult blood stool tests (hfob) to screen for colon cancer as well as the Papanicolaou test (commonly known as pap smear) for cervical cancer. Results for these three screening methods and both survey years are presented in Table 5, panels 1 and 2. For both years we find, as expected, that more educated individuals are more likely to screen. More importantly for our purpose however is that for both years we find that the interaction between education and the Gail Index is consistently a statistical zero and switches signs repeatedly across screening procedures, years, and specifications. We do not find any evidence that highly educated individuals *and* high breast cancer risk (Gail Index) are more likely to screen for cancer overall. It is only for breast cancer screenign that we find this interaction between the Gail Index and education.

6 Conclusion

In this paper we examine whether education increases allocative efficiency using the rich data on Breast cancer screening decisions available in the NHIS 2000 and 2005. We develop a model of information that links individual screening decisions based on imperfect signals about individual risk to medically appropriate measures or risk. The latter are interpreted as independent measures of individual risk of developing breast cancer. Given this model we show that the response of screening to risk factors is increasing if individuals are better informed about their risk.

We then relate screening to education and risk factors in the data. We find that individuals with higher individual risk are more likely to screen for breast cancer. More importantly for evaluating the allocative efficiency hypothesis: individuals with more education are more responsive to the presence of risk factors in their screening behavior. Our interpretation of this finding is that education increases the precision of individuals subjective risk assessments. This interpretation is supported by the finding that education indeed also leads to a greater responsiveness of reported individual cancer risk assessments. And, breast cancer risk factors do not interact with education in screening choices related to other types of cancer. This is evidence against an interaction between education and the willingness-to-pay for health that might generate the observed patterns.

Overall we conclude that the data on Breast Cancer Screening from the NHIS is broadly supportive of the hypothesis that education increases allocative efficiency in health care decisions.

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Table 1 Summary Statistics

	NHIS 2000		*NHIS 2005*	
Screening Variables				
Ever Had?	0.727		0.755	
# within 6 Years (if >0)	3.81		4.05	
	(2.02)		(2.41)	
Demographic Variables				
White	0.82		0.82	
Black	0.18		0.18	
Age	52.80		53.54	
	(15.68)		(15.28)	
Socio-Economic Variables				
Years of Schooling	13.21		13.50	
	(2.65)		(2.60)	
MSA – Size				
	non-MSA	22.90%	na	
	<250K	9.34%		
	250-500K	12.02%		
	500K-1M	11.83%		
	1-2.5M	23.65%		
	2.5-5M	12.11%		
	>5M	8.14%		
Family Income[1]				
	Aver < 5	2.40	2.40	
		(1.30)	(1.27)	
	% >5	20.67%	21.62%	
	not available	22.20%	20.77%	
Health Care Coverage				
Not Covered	9.98%		8.99%	
Breast Cancer Risk				
Gail Index[2]	1.03		1.09	
	(0.78)		(0.82)	
Parity>0	0.81		0.81	
Age at first Birth (if parity>0)	23.01		23.24	
	(4.99)		(5.14)	
Age at first Menstruation	12.83		12.76	
	(1.77)		(1.70)	
# of direct female relatives with breast cancer				
0	90.11%		88.86%	
1	9.09%		10.16%	
>1	0.80%		0.98%	
Doctor recommended screening[3]	na		53.41%	
Subjective Risk Assessment[4]				
	Low	52.18%	Less likely	34.57%
	Medium	29.49%	About as likely	48.09%
	High	11.47%	More likely	11.64%
	na	6.86%	na	5.71%
Observations	10,379		9,668	

1 Family Income is reported relative to poverty line with 13 categories between 0 and 5. For the summary statistics I assign the mid point to each interval. There is no separate distinction for family incomes above 5 times the poverty line. The percentages in this category as well as those with invalid responses are reported. The analysis uses the income variable as a categorical variable throughout, including invalid responses as a separate category.
2 The Gail Index is a constructed variable using the age at menstruation, age, family cancer history variables, parity, and the age at first birth.
3 (within last 12 months). In 2000 this question was only asked of women who were never screened.
4 In 2000 the subjective risk assessment variable refers to asked whether general subjective risk of cancer was low, medium, or high. In 2005 the question referred specifically to Breast Cancer Specific Risk and asked whether likelihood of developing cancer relative to average women.

Table 2 Relative risks from the Gail model. Reproduced from Gail et al. (1999)

Risk factor category		Relative risk factor
A. Age at menarche, years		
>13		1
12-13		1.2
<12		1.21
B. Age at first live birth, years	# of first-degree relatives with breast cancer	
<20	0	1
	1	1
	>1	6.8
20-24	0	1.24
	1	2.68
	>1	5.78
25-29 or nulliparous	0	1.55
	1	2.76
	>1	4.91
>29	0	1.93
	1	2.83
	>1	4.17
C. # of breast biopsies		
Age at counseling: <50		
0		1
1		1.27
>1		1.62
Age at counseling, 50+		
0		1
1		1.27
>1		1.62
D. Atypical hyperplasia		
No biopsies		1
At least one biopsy, no atypical hyperplasia		0.93
Atypical hyperplasia in at least one biopsy		1.82

To compute overall relative risk multiply four component relative risk from categories A,B,C,D

Table 2 Panel 1: Responsiveness of Screening Behavior to Education – 2000

	(1) Ever?	(2) # in 6 yrs	(3) Ever?	(4) # in 6 yrs	(5) Ever?	(6) # in 6 yrs	(7) Ever?	(8) # in 6 yrs
Years of Schooling	0.006 [0.003]	0.050 [0.021]*						
Gail Index	-0.074 [0.032]*	-0.416 [0.187]*	-0.017 [0.042]	0.058 [0.233]	-0.102 [0.036]**	-0.464 [0.210]*	-0.115 [0.035]**	-0.714 [0.206]**
School*Gail Index	0.008 [0.003]**	0.056 [0.014]**	0.007 [0.003]**	0.047 [0.015]**	0.005 [0.003]~	0.035 [0.016]*	0.005 [0.003]~	0.046 [0.015]**
Gail Index ^2			-0.008 [0.004]*	-0.062 [0.020]**				
Income*Gail					0.009 [0.003]**	0.038 [0.015]*	0.010 [0.003]**	0.053 [0.015]**
(Income>5)*Gail					0.141 [0.033]**	0.467 [0.164]**	0.151 [0.032]**	0.610 [0.163]**
Obs	10,379	10,234	10,379	10,234	10,379	10,234	10,379	10,234

~ significant at 10% * significant at 5% ** significant at 1%
Odd columns report marginal effects from probit on whether a woman ever received a mammogram.
Even columns report coefficient estimates of tobit model on number of mammograms in last 6 years.
Column 1-2: Baseline with dummies for income, race, and age.
Column 3-4: + Education dummies, quadratic gail
Column 5-6: + income*gail interaction
Column 7-8: + health insurance

Table 2 Panel 2: Responsiveness of Screening Behavior to Education – 2005

	(1) Ever?	(2) # in 6 yrs	(3) Ever?	(4) # in 6 yrs	(5) Ever?	(6) # in 6 yrs	(7) Ever?	(8) # in 6 yrs	(9) Ever?	(10) # in 6 yrs
Years of Schooling	0.002 [0.003]	-0.026 [0.023]								
Gail Index	-0.052 [0.035]	-0.571 [0.195]**	-0.006 [0.047]	-0.188 [0.246]	-0.068 [0.037]	-0.522 [0.210]*	-0.085 [0.036]*	-0.611 [0.210]**	-0.064 [0.032]*	-0.467 [0.206]*
School*Gail Index	0.008 [0.003]**	0.066 [0.015]**	0.006 [0.003]*	0.057 [0.015]**	0.002 [0.003]	0.061 [0.016]**	0.003 [0.003]	0.061 [0.016]**	0.002 [0.003]	0.055 [0.016]**
Gail^2			-0.005 [0.004]	-0.042 [0.017]*						
Income*Gail					0.013 [0.003]**	0.002 [0.015]	0.015 [0.003]**	0.011 [0.015]	0.010 [0.002]**	-0.001 [0.015]
(Income>5)* Gail					0.154 [0.029]**	0.082 [0.165]	0.169 [0.029]**	0.170 [0.165]	0.111 [0.025]**	0.112 [0.161]
Doctor recommend									0.234 [0.009]**	1.538 [0.062]**
Obs	9,701	7,264	9,700	7,264	9,700	7,264	9,700	7,264	8,887	6,952

Standard errors in brackets, * significant at 5%, ** significant at 1%
Odd columns report marginal effects from probit on whether a woman ever received a mammogram.
Even columns report coefficient estimates of tobit model on number of mammograms in last 6 years.
Column 1-2: Baseline with dummies for income, age, ethnicity and race.
Column 3-4: + Education dummies, quadratic gail
Column 5-6: + income*gail interaction
Column 7-8: + health coverage and various type of health insurance
Column 9-10: + doctor recommendation

Table 3 Panel 1: Cancer Risk Assessment and Education - 2000

	(1)	(2)	(3)
	risk of getting cancer in the future		
Years of Schooling	-0.041 [0.009]**		
Gail Index	0.022 [0.082]	0.115 [0.106]	-0.042 [0.093]
School*Gail Index	0.020 [0.006]**	0.018 [0.006]**	0.019 [0.007]**
$(\text{Gail Index})^2$		-0.011 [0.009]	
Income*Gail			0.011 [0.007]
(Income>5)*Gail			0.056 [0.072]
Observations	9667	9667	9667

Standard errors in brackets
* significant at 5%; ** significant at 1%
Report estimates from ordered probit regressions on individual cancer risk assessment: low, medium, high
controlling for ethnicity, age-dummies, ratio of income to poverty (dummies)
Columns use (2), (3) use dummies for education.

Table 3 Panel 2: Cancer Risk Assessment and Education - 2005

	(1)	(2)	(3)	(4)
	Greater Risk of Breast Cancer in Future			
Years of Schooling	-0.050 [0.009]**			
Gail Index	-0.093 [0.079]	0.273 [0.102]**	-0.152 [0.085]	-0.153 [0.089]
School*Gail Index	0.034 [0.006]**	0.028 [0.006]**	0.032 [0.007]**	0.032 [0.007]**
$(\text{Gail Index})^2$		-0.045 [0.007]**		
Income*Gail			0.009 [0.006]	0.010 [0.006]
(Income>5)*Gail			0.089 [0.065]	0.107 [0.068]
Doctor recommend				0.150 [0.027]**
Observations	9147	9147	9147	8406

Standard errors in brackets
* significant at 5%; ** significant at 1%
Report estimates from ordered probit regressions on individual cancer risk assessment: low, medium, high
controlling for ethnicity, age-dummies, ratio of income to poverty (dummies)
Columns (2)-(4) use dummies for education.

Table 4: Did doc recommend mam within last 12 months? (2005)

	doctor_rec
gail1	-0.039
	[0.035]
educgail1	0.003
	[0.003]
rat_gail	0.003
	[0.002]
rich_gail	0.008
	[0.027]
Constant	0.124
	[0.223]
Observations	8,888
R-squared	0.13

Only in 2005 do we have the variable: Did doctor recommend mammogram within the last 12 months for the entire sample. In 2000 this variable was only collected for woman who did not receive a mammogram.

Table 5: Partial Derivative with respect to Gail along NHIS 2000 Isoscreening manifold (P=0.9)

Type	*Screen Prop.*	*Gail Index*	*Age*	*Income*	*Coverage*	*Education*	*dScreen/ dGail*
(1)	0.9	1	50	0.5	Yes	15.20 (2.73)	4.64% (3.29)
(2)	0.9	1	50	1	Yes	16.41 (2.35)	6.13% (3.18)
(3)	0.9	1	50	2	Yes	15.08 (2.14)	8.53% (3.14)
(4)	0.9	1	50	3	Yes	11.20 (2.28)	9.26% (3.08)
(5)	0.9	1	50	4	Yes	7.68 (2.83)	7.22% (3.47)
(6)	0.9	1	50	5	Yes	7.44 (4.33)	1.32% (4.86)
(7)	0.9	1	50	>5	Yes	5.82 (3.19)	6.95% (3.93)
(8)	0.9	1	50	N/A	Yes	10.66 (2.31)	4.94% (3.07)
(9)	0.9	1	45	3	Yes	19.08 (2.46)	16.76% (3.90)
(10)	0.9	1	50	3	No	25.37 (4.07)	13.02% (4.82)

Table 6 Difference in Partial Derivative to Gail across Types[1]

	(1)	*(2)*	*(3)*	*(4)*	*(5)*	*(6)*	*(7)*	*(8)*	*(9)*	*(10)*
(1)	-	1.49% (.839)	3.89% (.949)	4.61% (.948)	2.58% (.817)	-3.32% (.778)	2.31% (.754)	0.30% (.578)	12.12% (.999)	8.38% (.988)
(2)		-	2.40% (.975)	3.12% (.909)	1.09% (.684)	-4.81% (.877)	0.82% (.616)	-1.19% (.312)	10.63% (.999)	6.88% (.978)
(3)			-	0.72% (.708)	-1.31% (.654)	-7.21% (.957)	-1.58% (.661)	-3.59% (.972)	8.23% (.999)	4.48% (.890)
(4)				-	-2.04% (.927)	-7.94% (.960)	-2.31% (.832)	-4.31% (.993)	7.51% (.999)	3.76% (.805)
(5)					-	-5.90% (.944)	-0.27% (.663)	-2.28% (.865)	9.54% (.999)	5.80% (.877)
(6)						-	5.63% (.930)	3.62% (.824)	15.44% (.999)	11.70% (.970)
(7)							-	-2.00% (.822)	9.81% (.994)	6.07% (.845)
(8)								-	11.82% (.999)	8.07% (.966)
(9)									-	-3.75% (.884)
(10)										-

1 Presented are the differences (column minus row) in the partial derivative across types (same as in table 4) together with the (1-p-value) for the one-sided test of diff=0.