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 individuals' assessments of their subjective risks. This induces (for the relevant parameter range) a positive interaction between education and risk factors in the screening decision. Empirically, educated individuals are indeed more responsive to the presence of risk factors in their 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 likely most important socio-economic correlate of good health. This conclusion emerges from Grossman and Kaestner's (1997) detailed survey on the literature of health and education. There is however much less 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 test this hypothesis by analyzing the interaction of risk factors and education in the breast cancer screening decision.[2] We present a model of information acquisition and screening that allows us to relate the individual screening decisions to the risk factors present in the data. Our empirical analysis then exploits this structure in three complementary ways.

First, we document in reduced form 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. This finding is consistent with our interpretation.

Second, we propose and implement a test of the hypothesis that the process

[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.

of information acquisition about the individual's risk of cancer is constant in the population. This test requires estimating iso-screening manifolds in the space of observed characteristics (such as education, income, measures of health care access, region of residence, race, age, and or family structure). These manifolds are defined by equal propensities of screening. Under the H_0 the responsiveness of screening to risk factors is constant in the population along these iso-screening manifolds. We reject the H_0 that the process of information acquisition is constant in the population.

Third, we calibrate the parameters of our model to match a number of crucial moments in our data. We then use the calibrated model to ask whether the observed variation of screening behavior with risk factors and education corresponds to the predicted behavior given various assumptions on the relation between structural parameters and education. This approach allows us to directly examine competing hypotheses and helps us interpret our model of health behavior and its relation to the data. We leave a structural estimation of the parameters of this model to future research.

Our investigation into the allocative efficiency of education is structured as follows. Section 2 provides an (incomplete) review of the literature on health demands. Section 3 presents the model of health screening and develops a test of the notion that the individual signal variance is constant in the population. Section 3 also shows why individuals with lower individual signal variance will tend to be more responsive to risk factors then individuals. Unfortunately this result does not hold globably. There are parameter values for which the screening response increases in the signal variance. We therefore calibrate the model to the data to show that for reasonable parameter values the sensitivity of the screening decision with repect to cancer risk factors is decreasing in the signal variance. 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. This finding is again consistent with the notion that the signal variance declines with education. 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 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. The model by 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

The stochastic event that agents attempt to forecast and that correlates with the risk factors recorded in our data is of course the presence of breast cancer. Our task is to formulate a tractable relation between individuals' forecasts of this stochastic event and the information on risk factors present in the data.

We describe the risk of breast cancer using a latent ("true") state variable t which lives on the real line. This latent state variable describes breast cancer risk in the sense that if $t < 0$, then a woman develops breast cancer. It is normally distributed with $t \sim N(1, \sigma_t^2)$. To set the mean to 1 represents a normalization as long as the risk of breast cancer is less than 50% in the population.[6] In that

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

[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).

case we can fit any risk of breast cancer in the population 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 $s \tilde{} N\left(t, \sigma_s^2\right)$. 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

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

The NHIS records various risk factors for breast cancer. Following standard practice in the medical literature we aggregate these risk factors into a single index, 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. For now it is sufficient to note that we are able to construct this index based on the survey responses recorded in both the 2000 and 2005 cancer control module of the National Health and Interview Survey.

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 signal s_G the breast cancer risk for any

[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.

individual is therefore given by the cdf of this normal random variable evaluated at 0. Thus, conditional on σ_G^2 we can map the breast cancer risk associated with each reported GI for each woman to a unique s_G.

Below we calibrate 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 live for (a maximum of) two periods. After the initial period there is an option of screening for cancer. Individuals allocate consumption across periods and make their screening decision conditional on the information available to them at t=1. Agents thus maximize

$$u\left(c_1\right) + \beta E\left[u\left(c_2\right)|s,\theta\right] \tag{3}$$

subject to

$$c_1 + RE\left[c_2|s,\theta\right] + p_\theta * \theta \leq y + RE\left[y|s,\theta\right] \tag{4}$$

Here c_τ denotes consumption in period τ, y represents the (constant) income flow, R is the market discount factor, and β is the individuals discount factor. θ is a health good that can be purchased at price p_θ. Our empirical work identifies the breast cancer screening decision with this good, a discrete decision variable. We will therefore later restrict θ to be equal to an indicator variable. For the time being however it is convenient to think of θ as a continuous positive variable. To further simplify the problem let us set $\beta = R$. The Bernoulli utility functions have the usual properties. The control variables of this maximization problem are $\left(c_1, c_2, \theta\right)$.

Utility when dead is normalized to 0. Thus, $E\left[u\left(c_2\right)|s,\theta\right] = \pi\left(\theta, s\right) * u\left(c_2\right)$ and $E\left[c_2|s,\theta\right] = \pi\left(\theta, s\right) * c_2$. Here $\pi\left(\theta, s\right)$ is the survival probability for period 2 and c_2 is consumption in period 2 conditional on survival and conditional on the health investment θ. Assume that $\pi\left(\theta, s\right)$ is concave increasing in θ.

The above structure implicitely assume that there is a complete set of Arrow-Debreu Securities available or (equivalently) that there is a market for annuities contingent on the survival probabilities depending on individual information

and behavior. This assumption is made purely for simplicity and is, we believe, not central to our argument.

The first order conditions of this problem are:

$$u'(c_1) = \lambda \tag{5}$$

$$u'(c_2) = \lambda \tag{6}$$

$$\beta \frac{u(c_2)}{\lambda} = \frac{p_\theta}{\pi'(\theta, s)} \tag{7}$$

in addition to the budget constraint. λ denotes the Lagrange multiplier - the value (in utils) of an additional dollar wealth. The left hand side of equation (7) delivers an expression commonly known as the Value of a Statistical life – the dollar value of an increase in the survival probability. The RHS represents the dollar costs of increasing the survival probability through the purchase of health goods.

Specialize the survival probability as $\pi(\theta, s) =$

$\pi * (1 - (\Pr(t < 0|s) * \Pr(death|t < 0, \theta)))$. Mortality risk is modeled as a competing risk - the probability of survival is multiplicative in the probability of surviving absent breast cancer (π) and the probability of not dieing due to breast cancer. The probability of dieing of breast cancer in turn depends on the probability of having breast cancer $\Pr(t < 0|s)$ times the mortality risk due to breast cancer conditional on the treatment θ : $\Pr(death|t < 0, \theta)$. Thus, we assume that the consumption of health goods (i.e. screening) affects the probability of survival conditional on being afflicted by breast cancer because earlier diagnosis improves the survival prognosis. This is the generally offered rationale for cancer screening. Cancer screening differs from other preventive health goods in that it essentially is a diagnostic tool - it does not affect the probability of contracting the disease but rather improves the prognosis through earlier diagnosis.

We further specify the effect of breast cancer screening on mortality risk to be proportional to the mortality risk m associated with breast cancer:

$$\pi(\theta, s) = \pi * \left(1 - \Pr(t < 0|s) * m * \widetilde{\theta}\right)$$

where $\widetilde{\theta} \in [0, 1)$ represents the proportional reduction of breast cancer mortality risk associated with screening. The assumption that breast cancer screen-

ing leads to a proportional decline in breast cancer mortality across different risk groups defined for instance by age is common in the medical literature.

Assume furthermore that breast cancer risk is small (it is) and that screening costs for breast cancer are likewise small (they are relative to life-time wealth). Then, we can approximate the dichotomous decision of screening using (7) to read:

$$\theta = 1 \Leftrightarrow \beta \frac{u(y)}{u'(y)} > \frac{p_\theta}{\Pr(t<0|s)\, m\left(1-\widetilde{\theta}\right)}$$

or alternatively

$$\theta = 1 \Leftrightarrow \Pr(t<0|s) > \frac{1}{VSL} \frac{p_\theta}{m\left(1-\widetilde{\theta}\right)} \tag{8}$$

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.

To simplify expressions let us denote the RHS in equation (8) as $\xi = \frac{1}{VSL} \frac{p_\theta}{m\left(1-\widetilde{\theta}\right)}$. It measures the cost of a unit reduction in mortality relative to the value of a unit reduction in mortality conditional on having breast cancer for sure.

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 thus identical to the hypothesis that σ_s^2 declines with individuals' education. Therefore we will pay particular attention to the role of σ_s^2 in what follows.

Consider inequality (8). 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: $\theta = 1 \Leftrightarrow s \leq c\left(\sigma_s^2, \xi\right)$. 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 $V(t|s)$.

3.3.1 Screening Response to latent state t

Our data contains only a signal on the latent state t. However, the variation of screening with the signal is derived from the variation of screening with the latent state t. It will be useful to discuss this response of screening to the latent state t first.

The probability of screening for an individual with latent state t is equal to the conditional[8] probability of observing a signal s below the cut-off $c\left(\sigma_s^2, \xi\right)$. Since the signal is distributed $N\left(t, \sigma_s^2\right)$ we have:

$$\Pr\left(\theta = 1|t, \sigma_s^2, \xi\right) = \Phi\left(\frac{c\left(\sigma_s^2, \xi\right) - t}{\sigma_s}\right) \tag{9}$$

Here $\Phi(.)$ represents the standard normal cdf. Let $\phi(.)$ represent the corresponding pdf. The change in the screening propensity with t is then

$$\frac{\partial \Pr\left(\theta = 1|t, \sigma_s^2, \xi\right)}{\partial t} = -\phi\left(\frac{c\left(\sigma_s^2, \xi\right) - t}{\sigma_s}\right) * \frac{1}{\sigma_s} \tag{10}$$

The responsiveness of the propensity to screen to variation in the latent state therefore depends on σ_s^2 in various ways. An increase in t will be perceived more accurately by individuals as the reliability increases (and σ_s declines). This is indicated by the fraction $\frac{1}{\sigma_s}$ that multiplies the pdf in equation (10). Individuals respond in their assessment of the posterior state to variation in the true, unobserved, latent state. This response is quantitative larger if the signal reliability increases. However, the presence of $\phi\left(\frac{c\left(\sigma_s^2, \xi\right) - t}{\sigma_s}\right)$ in equation (10) complicates matters. This term captures the fact that depending on the overall screening probability the share of individuals who are indifferent to screening varies non-monotonically with σ_s. This monotonicity of $\phi\left(\frac{c\left(\sigma_s^2, \xi\right) - t}{\sigma_s}\right)$ renders it impossible to derive global results for the variation of the screening responsiveness to t with σ_s.

[8] Conditional on the true state t.

Let us now turn to the variation in the screening response with the Gail Index.

3.3.2 Screening Response to Gail Index

Observing the Gail Index is equivalent to observing a signal s_G on the latent state variable t with variance σ_G^2. From eq (2) we have the posterior distribution of t conditional on observing the signal s_G. Denote this distribution as $\kappa(t|s_G)$. By assumption the signal noise $s_G - t$ is independent of all other disturbances. For each t, the propensity of screening is given by inequality (8) . Thus, conditional on s_G we have:

$$\begin{aligned} \Pr\left(\theta = 1|\sigma_s^2, \xi, s_G\right) &= \int \kappa\left(t|s_G\right) * \Pr\left(\theta = 1|\sigma_s^2, \xi, t\right) dt \qquad (11) \\ = \quad & \int \kappa\left(t|s_G\right) * \Phi\left(\frac{c\left(\sigma_s^2, \xi\right) - t}{\sigma_s}\right) dt \end{aligned}$$

We are seeking an expression for the response of screening to variation in s_G. A useful expression for this response can be obtained by acknowledging that a unit-variation in s_G is equivalent to a shift of the entire posterior distribution $\kappa(t|s_G)$ by $\frac{\sigma_t^2}{\sigma_t^2+\sigma_G^2}$. Thus, we can express the variation in the propensity to screen by holding the distribution of the prediction error $\widetilde{\kappa}\left(t - E[t|s_G)\right)$ [9] constant and integrate over the changing propensity in screening with a shift of the latent variable t by $\frac{\sigma_t^2}{\sigma_t^2+\sigma_G^2}$. The distribution $\widetilde{\kappa}\left(t - E[t|s_G)\right)$ is constant with respect to s_G due to the normal structure of this problem. Thus we can write the response of screening to the gail signal is:

$$\begin{aligned} & \frac{\partial \Pr\left(\theta = 1|\sigma_s^2, \xi, s_G\right)}{\partial s_G} \qquad (12) \\ = \quad & -\frac{\sigma_t^2}{\sigma_t^2 + \sigma_G^2} \frac{1}{\sigma_s} \int \widetilde{\kappa}\left(t - E[t|s_G)\right) * \phi\left(\frac{c\left(\sigma_s^2, \xi\right) - t}{\sigma_s}\right) dt \end{aligned}$$

It is difficult to obtain global solutions for the variation of $\frac{\partial \Pr\left(\theta=1|\sigma_s^2,\xi,s_G\right)}{\partial s_G}$ with σ_s for the same reason that it is difficult to get global solutions for the variation of $\frac{\partial \Pr\left(\theta=1|t,\sigma_s^2,\xi\right)}{\partial t}$ with σ_s. Clearly the multiplication by $\frac{1}{\sigma_s}$ tends in-

[9] This distribution of the prediction error is independent of the observed signal s_G.

crease in the responsiveness of screening behavior as the signal variance declines. This is due to the fact that individuals are more responsive in updating their posterior expectations of t to variation in the signal s if σ_s declines. However, the population 'at risk' is determined by $\phi\left(\frac{c\left(\sigma_s^2,\xi\right)-t}{\sigma_s}\right)$ whose variation with σ_s is not well understood (by us) yet.

The fact that we currently do not have global solutions for the responsiveness of screening behavior with respect to σ_s leads us to examine the behavior of the screening response numerically. In particular, we calibrate the main parameters in equation (12) to match crucial features of the data and then examine the comparative statics with respect to these same parameters.

Before turning towards the calibration exercise however, we want to briefly discuss a test of the hypothesis that the individual signal variance σ_s is constant in the population. This test is implied by the equations (11) and (12) .

3.4 Test for constant σ_s in the population?

We are testing the H_0 that σ_s is constant in the population. Under the H_0 the RHS of equation (11) is increasing in the cut-off $c\left(\sigma_s^2,\xi\right)$. Equation (11) therefore defines, conditional on s_G, a one-to-one mapping between the $c\left(\sigma_s^2,\xi\right)$ and the propensity to screen. We can invert this mapping to obtain $c\left(\sigma_s^2,\xi\right) = h\left(p,\sigma_s^2,s_G\right)$.

Substituting $h\left(p,\sigma_s^2,s_G\right)$ in (12) delivers

$$\begin{aligned}\frac{\partial \Pr\left(\theta=1|\sigma_s^2,\xi,s_G\right)}{\partial s_G} &= -\frac{1}{\sigma_s} * \widetilde{f}\left(s_G,\sigma_s^2,h\left(p,\sigma_s^2,s_G\right);\sigma_t^2,\sigma_G^2\right) \quad (13)\\ &= -\frac{1}{\sigma_s} * f\left(s_G,\sigma_s^2,p;\sigma_t^2,\sigma_G^2\right)\end{aligned}$$

This expression contains next to σ_s only the parameters σ_t^2 and σ_G^2 and the propensity p. Thus, under the H_0 we have that the responsiveness to screen should be independent of individual characteristics conditional on the overall propensity to screen and the observed gail signal. This can be tested in the data by first estimating the propensity to screen based on a vector of individual characteristics and then examine the responsiveness in the propensity to screen to the Gail Index for values of the characteristics vector that generate the same propensity to screen.

This test has the advantage that it does not require calibration or estimation

of $\left(\sigma_t^2, \sigma_G^2, \xi, \sigma_s^2\right)$. However, it does not allow estimating the relation between σ_s and individual characteristics if H_0 is rejected. If σ_s varies with individual characteristics, then the responsive to screen will vary with these characteristics in a way that can not be easily described due to the presence of σ_s^2 in the function f in equation (13) .

3.5 Calibrating the population parameters

As argued above, we are not able to produce global results (holding for the entire parameter space) for how the response of screening to the presence of risk varies with σ_s. We therefore rely on computational comparative statics with respect to σ_s to develop the argument. We will also examine the possibility that the responsiveness to the Gail signal varies with ξ, a parameter that is surely not invariant to education.

We are interested in examining the empirical relationships which arise when σ_s^2 and ξ are not constant in the population and in particular the consequence of having these vary systematically with education. To assess these relationships we pursue the following strategy: First, we assume that σ_s and ξ are constant parameters in the population and calibrate their values together with (σ_G, σ_t) - the variances of the Gail signal and the latent variable t.

Using this calibrated version of the model allows us to examine how screening would vary with education if (σ_s, ξ) are assumed to depend on education. In particular, we can examine our main hypothesis - that education lowers the signal variance σ_s and we can examine the hypothesis that education lowers the costs of screening and/or increases the Value of a Statistical Life. (see Murphy and Topel 2005). Thus, we will use the calibrate version of the model to perform comparative statics with respect to σ_s and ξ. In particular, we show that as σ_s increases the response to the Gail signal declines. And, we show furthermore that the responsiveness to the Gail Signal also declines as ξ decreases. Thus, the observed patterns in the data can not be explained by increased willingness to pay for mortality reductions with increasing education.

Thus we need to calibrate the four parameters $(\sigma_t^2, \sigma_G^2, \sigma_s^2, \xi)$ to four moments in the data.

First, we match the probability of cancer in the population by choice of σ_t^2. Then, we choose σ_G^2 to match the distribution of the Gail index observed in the population. Our last two parameters σ_s^2 and ξ are then chosen to match the average population screening rates and the average variation in those screening

rates as the Gail Index varies.

Recall that t the true state of the decision maker is distributed $N(1, \sigma_t^2)$ in the population. Also recall that a person will develop breast cancer if and only if their value of t is less than 0, and that this realization is independent of all choice variables (screening for breast cancer reduces mortality conditional on breast cancer, but does not reduce the probability of developing breast cancer.) Therefore the prior variance of t must fit the unconditional probability of a woman developing breast cancer in the population. The National Cancer Institute[10] estimates the average ten year risk of breast cancer for an American woman to be approximately 1/40. Setting:

$$\Phi\left(-\frac{1}{\sigma_t}\right) = 0.025 \Rightarrow \sigma_t \approx 0.5 \tag{14}$$

Next, recall that the Gail score measures the relative risk of developing breast cancer based on all the information commonly evaluated by doctors when attempting to estimate a patients risk of developing breast cancer. The Gail signal is interpreted as the signal s_G that gives rise to this Gail score. The Gail signal is distributed around the true state t of each person, with variance σ_G^2. We will calibrate σ_G^2 by fitting it to the distribution of Gail scores in the population. This can be done by requiring the agent who receives the $x'th$ percentile Gail signal to have a posterior risk of breast cancer equal to the x'th percentile in the observed distribution of the Gail Score.

The prior distribution of Gail signals in the population is $N(1, \sigma_t^2 + \sigma_G^2)$. Hence, the xth percentile signal (denote s_x) must satisfy two conditions. First, the cdf of the prior distribution of Gail signals must correspond to the xth percentile of the prior distribution of Gail signals in the population:

$$\Phi\left(\frac{s_x - 1}{\sqrt{\sigma_t^2 + \sigma_G^2}}\right) = x \tag{15}$$

where s_x is the Gail signal the xth percentile person sees. Then we can define P_x as the implied risk of breast cancer for the xth percentile of the Gail Score distribution. Second, it must be that after observing signal s_x, the posterior probability of the person developing breast cancer is the same as that implied by the Gail score P_x. Using equation 2 we can write this as:

[10] see: http:/www.cancer.gov/downloads/stt/CAFF06Prob.pdf

$$\Phi\left(\frac{-\left(1+\phi_G\left(s_x-1\right)\right)}{\sqrt{\frac{\sigma_t^2\sigma_G^2}{\sigma_t^2+\sigma_G^2}}}\right)=P_x \tag{16}$$

National cancer statistics suggest that $25th$ and $75th$ percentile of the Gail Index correspond to a ten-year risk of breast cancer of 0.0268 and 0.00913, respectively. Solving equations 15 and 16 for these two values implies values for σ_G of 0.82 and 1.41, respectively. For simplicity then, we adopt an approximate value of 1 for σ_G.

Finally, we wish to calibrate the remaining two parameters of the model, σ_s^2, and ξ. These two parameters can be fit using the observed percentage of the population that screens for breast cancer and the average responsiveness of screening to the Gail signal.

First, approximately 80% of the population chooses to screen for breast cancer using a mammogram. Given any value of σ_s^2 we know the prior distribution of private signals in the population. Also, given any signal s, σ_s^2, and ξ, we can solve for the decision maker's posterior distribution of their state t, and compute their expected utilities from screening and not screening. This allows us to solve for the agent's decision rule, which is the cutoff signal $c(\sigma_s^2,\xi)$, above which they forgo a mammogram, and below which they choose to screen.

The prior distribution of agent's signals are distributed $N(1,\sigma_t^2+\sigma_s^2)$, and therefore fitting the overall screening rate of the population is equivalent to solving:

$$\Phi\left(\frac{c(\sigma_s^2,\xi)-1}{\sqrt{\sigma_t^2+\sigma_s^2}}\right)=0.8 \tag{17}$$

Second, in a simple OLS framework, breast cancer screening appears to increase approximately 9 percentage points for every extra point on the Gail score. Further, conditional on the Gail signal of a person the signal s will be distributed $s\tilde{}N\left(1+\phi_G\left(s_G-1\right),\frac{\sigma_t^2\sigma_G^2}{\sigma_t^2+\sigma_G^2}+\sigma_s^2\right)$, and the proportion who will screen is given by:

$$\Pr(\theta=1|s_G,\sigma_s,\xi)=\Phi\left(\frac{c(\sigma_s^2,\xi)-\left(1+\phi_G\left(s_G-1\right)\right)}{\sqrt{\frac{\sigma_t^2\sigma_G^2}{\sigma_t^2+\sigma_G^2}+\sigma_s^2}}\right) \tag{18}$$

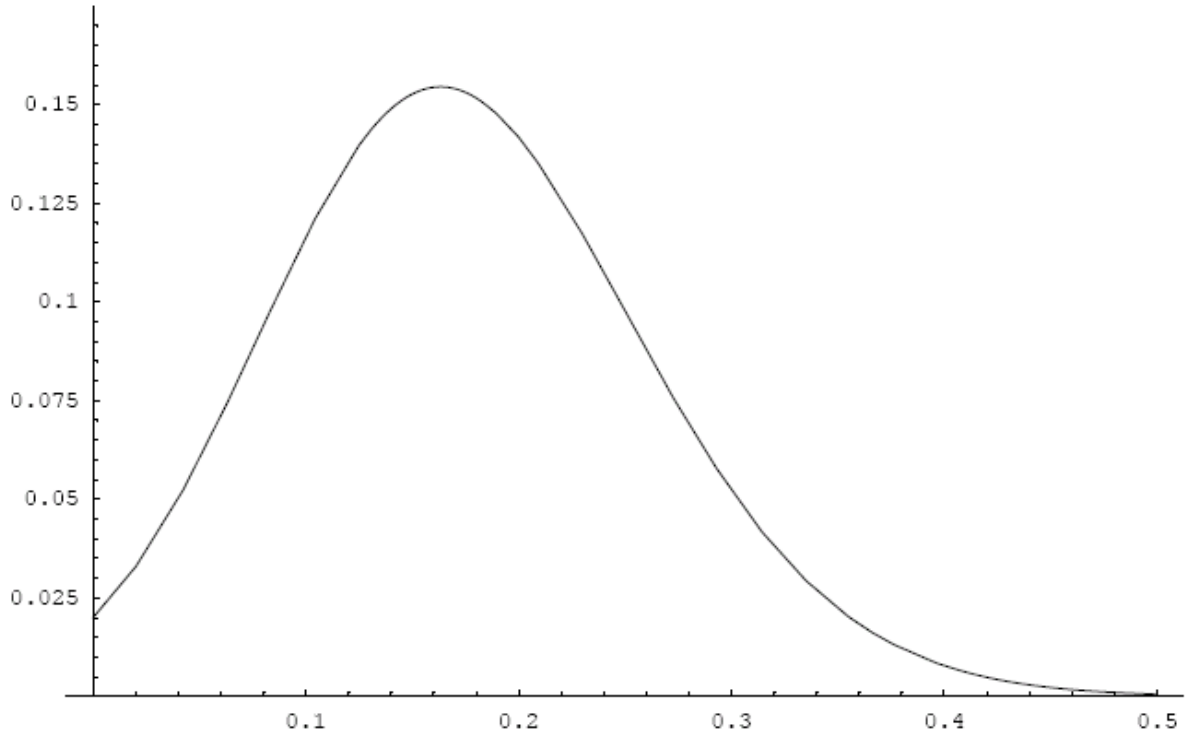
This last expression shows that the probability of screening is a function of the Gail signal s_G as well as the two parameters we want to calibrate. The probability of screening for any particular value of s_G given in equation (16)corresponds closely to the concept in equation (15) . Both essentially pin down the average screening rate. We therefore choose not to calibrate to this probability (15) directly. Instead, we calibrate the screening response to variation in the Gail score at a Gail score =1 (corresponding to the average Gail in the population).

Thus, we evaluate the derivative of eq (16) with respect to s_G at the value of s_G corresponding to a Gail score of 1 and then match this derivative as well as equation (15) by choice of σ_s and ξ.

When we do this, we estimate a value of σ_s that is approximately 0.27 and a value of ξ corresponding to a VSL of \$x, a screening costs of 200, and a increase of the 5 year survival rate from cancer from 0.8 to 0.84 (of course since ξ is a function mapping the costs of screening, the mortality benefits, and the VSL into the real line, there exists a 3-dimensional manifold that can generate the same screening behavior) Note that this suggests that the agent's private signal as to their cancer risk has approximately the same variance as their prior beliefs, and is roughly three times as accurate as the Gail score.

3.6 Numerical comparative statics

The above calibration allows us to consider how screening response varies with the individual signal variance σ_s and the parameter ξ describing the Marginal rate of substitution between consumption and survival. It also provides the basis for a more thorough structural exploration of our model. For the time being however, we limit ourselves to examining how the responsiveness of screening to the Gail Index varies with the signal variance σ_s and with ξ. The former is important since we the allocative efficiency hypothesis of education in this model is captured by a negative variation of σ_s with education. This implies that more educated individuals are better informed about their risk and therefore are better placed to make efficiency screening decisions. The numerical comparative statics with respect to ξ are important since education is generally assumed to increase the marginal rate of substitution of consumption for survival. It is therefore of interest to know whether the observed interactions between education and risk factors in screening might be generate simply by the direct effect of education on this willingness to pay for health, without any

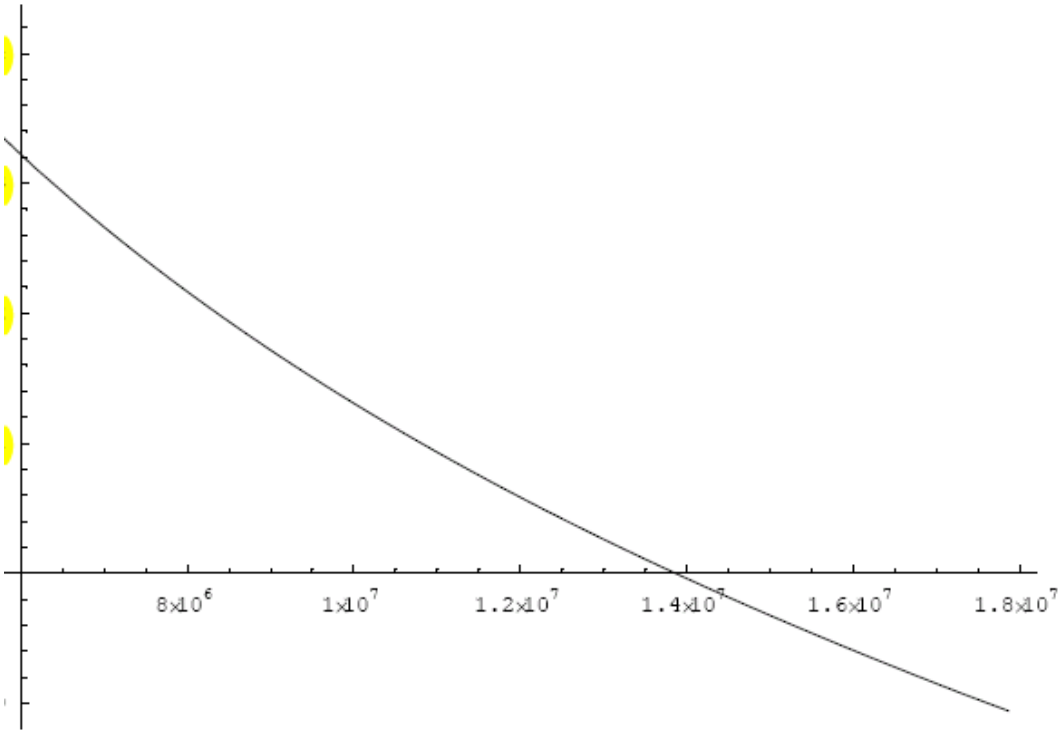


need to rely on allocative efficiency to generate the observed relation.

Figure 1 shows how the responsiveness of screening to the Gail Score varies with σ_s around the calibrated values of the parameter vector $(\sigma_s, \sigma_G, \sigma_t, \xi)$ and around a Gail score of 1.

Figure 1: The responsiveness of screening to variation of risk factors as a function of σ_s

First, we observe that the relation to σ_s is indeed not monotone. For very low values of σ_s the screening response declines with the signal variance . The reason is simple. For a Gail Score of 1 the $P(t < 0|s_G) = 0.025$. As σ_s goes to 0 individuals become almost perfectly informed about their risk of cancer. This implies that in the limit only those who actually have t<0 will get screened. And, consequently, the responsiveness to the Gail score tends to equal the change in the proportion with t<0 as the Gail score increases. Since the Gail score is a relative risk, the screening responsiveness to variation in the Gail score therefore



has to decline to 0.025. The calibrated value of σ_s is however well on the 'other' side of this hump, implying that variation in the signal variance around σ_s will tend to lead to inverse variation in the screening responsiveness. This reflects the higher response in assessed risk to variation in the Gail Score by individuals with lower σ_s.

Figure 2 examines the variation of the screening response with ξ.[11]

Figure 2: The variation of the responsiveness of screening to risk factors as a function of income

Clearly, any positive association of the willingness to pay for mortality reductions will imply that the responsiveness of screening to risk factors declines with education rather than increases. The reason for this is that as incomes increase overall screening rates increases, reducing the number of individuals

[11] The variation in ξ is generated by variation in incomes.

who are indifferent between screening and not screening. These marginal individuals are however those that are potentially responsive to variation in the risk factors. Therefore, increasing incomes and increasing willingness to pay for mortality reductions can not explain any positive interaction between risk factors and education in the screening decision.

We draw two conclusions from this (preliminary) exploration of the model around the calibrated values. First, if we implement the allocative efficiency hypothesis by assuming that the individual signal variance is indeed declining with education, then we can test the allocative efficiency hypothesis by examining the interaction between education and observed risk factors in the screening decision. The prediction from the allocative efficiency hypothesis is that education and observed risk factors should indeed interact in a positive way in predicted screening equations. We will test this hypothesis below. Second, any observed positive interaction between education and risk factors can not be explained by an increase in the willingness-to-pay for mortality reductions. Thus, any positive effect of education on the willingness-to-pay can not serve as an explanation for the positive interaction between education and risk factors documented below.

We will now turn to the empirical analysis, beginning with the description of the data.

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 woman 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 gail index 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. 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 thus do not need to rely on the frequency of positive breast cancer screens in constructing the Gail Index.

5 Empirical Specifications and Results.

5.1 Reduced Form Analysis

We start the empirical analysis by directly examining how education and risk factors are related to screening behavior. For this purpose we consider two specifications. First, we estimate a probit on whether an individual has ever undergone cancer screening:

$$\theta = 1 \Leftrightarrow \beta'_x x + \beta_G * GI + \beta_s * S + \beta_{GxS} * S * GI + \varepsilon > 0 \tag{19}$$

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. Our second specification allows for an intensive margin in the screening decision. The variable θ_6 indicates the number of mammograms a woman has received during the course of the last 6 years. We analyze θ_6 in a standard Tobit regression to account for the fact that the dependent variable is left censored at 0. The specification of the latent variable of this Tobit model is the same as that in eq. (19) .

Table 2 Panel 1 presents the parameter estimates for both specifications obtained using the 2000 data. In column 1 and 2 (our baseline) the control set includes a set of dummies for the income variable.[12] The baseline specification also includes a full set of dummies for age and race. Reported coefficients for probits correspond to marginal effects throughout this paper. At the mean of schooling (~13 years of schooling) we observe that both education and a higher risk of breast cancer raises the screening propensity. Neither of these findings is informative on the question whether schooling improves health decision making. The coefficient estimate relating to the 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 indeed respond more to the presence of risk factors than less educated individuals. An increase in the Gail Index by one unit (doubling the relative risk of breast cancer) raises the probability of screening by

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

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 2). The interaction between the Gail Index and Education is positive, small, but not neglible. Columns 3-8 consider alternative specifications. Columns 3 and 4 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.[13] In columns 5 and 6 we also 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 significant. The difference to the estimates in column 1 and 2 is not statistically signficant. 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 5 and 6 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 7 and 8 control for health care coverage. We find that the results are robust to all of these variations in functional form and control sets.

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 2). This is robust to relaxing the functional form (columns 3 and 4). Women with higher family incomes are likewise more sensitive to the presence of risk factors (columns 5 and 6), 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 7 and 8). Finally, the 2005 survey also asked all women whether a doctor had recommended them to receive a mammogram within the last 12 month. Controlling

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

for this direct measure of the role of health care intermediary does also not substantially affect the results (columns 9 and 10).

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. That model allowed for an allocative role of education in health care decisions by allowing the signal noise to depend inversely on education. 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.

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 3, Panel 1) and the 2005 (Table 3, Panel 2) data.

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.

5.2 Testing for variation of σ_s in the population.

In Section 3.4 we proposed a test of the null hypothesis that σ_s is constant in the population by examining the partial derivative of screening with respect to risk factors along iso-screening manifolds. This section implements this procedure. First, we estimate the propensity for screening based on flexible estimating equations. This allows us to identify the iso-screening manifolds within the support of the covariates that are defined by equal screening rates. We can then examine the variation of the screening response within these manifolds with covariates such as education income, age, and health care coverage.

To determine the isoscreening manifolds we estimate the propensity to screen as a function of controls x, the signal individual Gail Index GI, and education e. We then evaluate the partial derivative of the propensity to screen with respect to the Gail Index at various points of the estimated manifold.

We start by estimating a probit on a latent index function $y\left(x, GI, e\right)$ depending on a N_x-dimensional vector of individual characteristics x, education e, and Gail index GI. The control vector x includes a cubic in age, a cubic in family income as a multiple of the poverty line, indicators if family income is unknown or larger than 5 times the poverty line, an dummy variable to determine whether a woman has health insurance coverage, education, and the Gail Index. We furthermore divide the support of the Gail Index into 10 categories with equal numbers of observations and include dummies for these to allow for non-linear effects of the Gail Index. Finally we interact the Gail Index with the cubic in age, the specification for income, and education. Thus, for any given estimated propensity p of screening and Gail Index GI we have:

$$y(x, GI, e) = \Phi^{-1}\left(p\right) \tag{20}$$

Equation (20) maps (p, GI) into an $N_x + 1$ dimensional surface along which the screening propensity is constant. Denote this surface $m\left(p, GI\right)$. We estimate this surface and then numerically examine the partial derivative for the screening propensity for various points along $m\left(p, GI\right)$:

$$\frac{\partial p}{\partial GI} = \phi\left(p\right) * \frac{\partial y\left(x, GI, e\right)}{\partial GI} \tag{21}$$

Table 5 examines the partial derivative (21) for 10 points in the control-space ("types") along the manifold with p=0.9 and a $GI = 1$. The standard errors are

boot-strapped with 1,000 replications. The first stage estimation results of the manifold are of no particular interest here and therefore not presented here.[14] The types 1-8 in table 4 are chosen to cover the entire income space for women aged 50. Type 9 examines the variation in the responsiveness with age for a woman with the modal family income, and type 10 the variation with health insurance for a woman of age 50 with modal family income. Conditional on GI the education variable enters linearly in the index function which ensures that for any choice of controls x there is a unique value for education that sets p=0.9. We thus solve for the value of education that, given type and GI=1, ensures that p=0.9. We then evaluate numerically the partial derivative of the screening propensity with respect to GI for each type. This partial derivative varies substantially across types. The relation is non-monotonic with income. The highest responsiveness to the GI is observed for types 9 and 10 with income=3 and high levels of education. The standard errors in table 4 however are large.

Table 6 presents the difference in the partial derivatives (21) across types 1-10. Each cell represents the estimated difference of the responsiveness between the row and column type and reports the p-value of the (one-sided) hypothesis that this difference is greater than (or smaller than depending on the sign) 0. Looking across this table we observe that there are indeed many p-values that are significant at the 10 or 5% level, indicating that the responsiveness to the Gail Index is indeed not constant along the manifold. Within our model such differences in the responsiveness of screening are inconsistent with constant signal variances across individuals. We therefore reject the hypothesis that σ_s is constant across individuals.

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 for parameters consistent with basic features of the data the response of screening to risk factors is increasing if individuals are better informed about their risk.

[14] These results are available upon request from the authors.

We also show that the response to income is predicted to decline in incomes. The response is also predicted to be lower if individuals have better access to the health care system, interpreted as having lower costs of screening.

We then relate screening to education and risk factors in the data. We find that individuals with higher individual risk screen more. 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 in the NHIS.

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 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	0.050						
	[0.003]	[0.021]*						
Gail Index	-0.074	-0.416	-0.017	0.058	-0.102	-0.464	-0.115	-0.714
	[0.032]*	[0.187]*	[0.042]	[0.233]	[0.036]**	[0.210]*	[0.035]**	[0.206]**
School*Gail Index	0.008	0.056	0.007	0.047	0.005	0.035	0.005	0.046
	[0.003]**	[0.014]**	[0.003]**	[0.015]**	[0.003]~	[0.016]*	[0.003]~	[0.015]**
Gail Index ^2			-0.008	-0.062				
			[0.004]*	[0.020]**				
Income*Gail					0.009	0.038	0.010	0.053
					[0.003]**	[0.015]*	[0.003]**	[0.015]**
(Income>5)*Gail					0.141	0.467	0.151	0.610
					[0.033]**	[0.164]**	[0.032]**	[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	-0.026								
	[0.003]	[0.023]								
Gail Index	-0.052	-0.571	-0.006	-0.188	-0.068	-0.522	-0.085	-0.611	-0.064	-0.467
	[0.035]	[0.195]**	[0.047]	[0.246]	[0.037]	[0.210]*	[0.036]*	[0.210]**	[0.032]*	[0.206]*
School*Gail Index	0.008	0.066	0.006	0.057	0.002	0.061	0.003	0.061	0.002	0.055
	[0.003]**	[0.015]**	[0.003]*	[0.015]**	[0.003]	[0.016]**	[0.003]	[0.016]**	[0.003]	[0.016]**
Gail^2			-0.005	-0.042						
			[0.004]	[0.017]*						
Income*Gail					0.013	0.002	0.015	0.011	0.010	-0.001
					[0.003]**	[0.015]	[0.003]**	[0.015]	[0.002]**	[0.015]
(Income>5)*Gail					0.154	0.082	0.169	0.170	0.111	0.112
					[0.029]**	[0.165]	[0.029]**	[0.165]	[0.025]**	[0.161]
Doctor recommend									0.234	1.538
									[0.009]**	[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	0.115	-0.042
	[0.082]	[0.106]	[0.093]
School*Gail Index	0.020	0.018	0.019
	[0.006]**	[0.006]**	[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	0.273	-0.152	-0.153
	[0.079]	[0.102]**	[0.085]	[0.089]
School*Gail Index	0.034	0.028	0.032	0.032
	[0.006]**	[0.006]**	[0.007]**	[0.007]**
$(\text{Gail Index})^2$		-0.045		
		[0.007]**		
Income*Gail			0.009	0.010
			[0.006]	[0.006]
(Income>5)*Gail			0.089	0.107
			[0.065]	[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*	*Educa-tion*	*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.