Workplace interactions and fear of breast cancer: evidence from a dynamic natural experiment*

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Abstract. We study a dynamic natural experiment in which groups of women are randomly exposed to breast cancer via workplace social interactions, through colleagues who are diagnosed with the disease. We find that women in such (treatment) groups reduce their propensity to perform a screening mammography by almost 14 percentage points relative to other (control) women, on a basis of 72 percentage points, in the year in which the event occurs. This impact effect slowly diminishes in the following years. The negative average treatment effect we identify masks important heterogeneity: the effect is much stronger in case of exposure to severe (as opposed to milder) forms of breast cancer, is larger for younger and healthier women, and is much weaker and much less persistent for medical doctors and nurses. We interpret these results through the lens of a simple expected utility model and existing "mechanism experiments" from the medical literature. A plausible interpretation is the presence and prevalence of short–run anticipatory feelings leading to information aversion and thus to a reduced propensity to screen when breast cancer becomes more salient.

Keywords: breast cancer, social interactions, salience, information aversion.
JEL codes: I12, D03, Z13.

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1 Introduction

In this paper we study the dynamic effect of working with someone who was diagnosed with breast cancer on one's propensity to perform a screening mammography. That is, how does a woman's propensity to screen change over time after a colleague is diagnosed with breast cancer? As a benchmark—and assuming that preferences and technology are unaffected by the event in question—there is no effect to expect for a fully informed and rational decision–maker: if risks are independent and if a woman is fully informed about the underlying probability of developing the disease, then the fact that a colleague was diagnosed with breast cancer brings no new information. However, effects are possible if we depart from this benchmark, although in this case the theoretical sign of the change is uncertain. For example, suppose the proximate effect of knowing someone who is diagnosed with breast cancer is an increase in the perceived probability of developing the disease, as repeatedly suggested in the medical literature.[1] The net effect of such increased risk perception on the propensity to screen is not obvious because there are at least two underlying forces that work in opposite directions. First, an increased belief of developing the disease should induce a woman to screen more frequently in order to take timely action in case she is sick. Second, an increased perception that she may develop the disease may induce a woman to screen less frequently because of the fear of finding out that she is sick.[2]

Our research strategy is to resolve the question in a purely empirical way and then offer a simple model that helps rationalize our empirical findings. This strategy follows from the fact that although the model we will use has empirical content—which we connect directly to the empirical analysis—we would be unable to identify its structure.[3] We gained access to rich information about the universe of employees at a large medical organization in the United States, which we used to construct a unique panel data set spanning three years and containing demographic, professional, socioeconomic,

[1] For instance, Evans *et al.*, (1993), Helzlsouer *et al.* (1994), Hopwood (2000), and Montgomery *et al.* (2003) all document that this is the case.

[2] The latter is far from a remote possibility. For instance, Caryn Lerman *et al.* (1998) document that many women refuse to receive information on whether they have a genetic predisposition to breast cancer.

[3] This is because of the unobservability of the perceived probabilities of having breast cancer. In a different context, Lance Lochner (2007) uses repeated observations of the perceived probability of being arrested for committing specific crimes, and thus is able to identify the learning process triggered by indirect experiences of arrests. Unfortunately, we cannot do this for the problem at hand.

and high-quality health data for female employees at this organization. The data set also contains detailed information about physical location at the workplace, which allows us to construct reference groups in which social interactions plausibly occur on a daily basis.[4] Our baseline analysis considers the screening behavior of women in the age group 50 and older. Women in this group are the most at risk of developing breast cancer and are subject to unambiguous screening guidelines. We find a large, negative effect at impact. In the year during which a colleague was diagnosed with breast cancer, treated women become almost 14 percentage points less likely to perform a mammogram relative to control women (the baseline screening rate is about 72%). This initial effect vanishes over time, but at a slow pace: after two years about half the initial effect is still present. We find important heterogeneity behind this average dynamic treatment effect. First, it is much stronger for women who are exposed to more severe cases of breast cancer. Second, it is somewhat stronger for younger and healthier women. Third, when we isolate more knowledgeable employees such as medical doctors and nurses, we find that the treatment effect is much weaker than average and never statistically significant for this group. On the other hand, for all other employees the treatment effect is much more persistent than average: compared to an impact effect of about 13 percentage points, the propensity to perform a mammogram after 2 years is still 11 points below the baseline for those who are neither medical doctors nor nurses.

At the end of the paper we rationalize these results within an expected utility model that is augmented with an anticipatory component of preferences, in light of existing "mechanism experiments" from the medical literature. We argue that the available evidence supports one particular causal chain: experiencing breast cancer via workplace social interactions increases the salience of the disease and leads to a lower probability of screening in the short–run because of anticipatory feelings (fear of bad news) leading to information aversion. This interpretation is consistent with both the results of small-scale controlled experiments and real life experiences, as documented at the end of the paper.

The reason why with such a rich data set at hand we focus only on breast cancer is that this disease has clear prevention guidelines that allow us to unambiguously define "normal" screening behavior at an annual frequency. Because it is recommended (with virtually universal consensus in the medi-

[4] In this respect our study is close to the field experiment of Esther Duflo and Emmanuel Saez (2003), who find that providing information on retirement plans to randomly selected employees increases the enrollment rate of their initially uninformed co-workers as well.

cal community) for women to perform a screening mammography every year after age 50, we can look at the propensity to screen for breast cancer every year after this age as an indicator of the propensity to take "normal" preventive actions against the disease. This is much harder to do for other types of tumors, given the longitudinal limitation of our data. Consider, for instance, prostate cancer. Like breast cancer for women, in the United States this is the most common nonskin cancer affecting men, and the second most common cause of cancer–related death in this group. A recent statement of the US Preventive Services Task Force says that "the current evidence is insufficient to assess the balance of benefits and harms of prostate cancer screening in men younger than age 75 years", and "recommends against screening for prostate cancer in men age 75 years or older" (USPSTF, 2008). In this case it would be impossible to determine what "normal" screening behavior is. Or consider cervical cancer, where the guideline is that women should have a Pap test every three years. With three years of data and staggered screening it would be impossible, in our data set, to tell which women are complying with the guidelines and which are not.

The remainder of the paper is organized as follows. Section 2 describes the data. Section 3 illustrates the econometric design and discusses the identifying assumptions. Section 4 reports the results. Section 5 illustrates the theoretical framework we use to rationalize the results. Section 6 concludes.

2 Data set

Our data come from a large health care provider in the United States that has over 20,000 employees (70% of whom are women) at its main location. All employees are eligible to participate in a self-administered health plan run by the organization itself, and given the plan's comprehensive nature and the lack of comparable substitutes, participation is virtually universal. The data were gathered from a variety of different sources, and we used them to construct a three-year panel (2002, 2003, and 2004).

Demographic information like age, gender, marital status and family size were gathered from electronic administrative records maintained for all patients. We obtained information on occupation, whether an employee was full or part-time, salary class, job title, and job tenure from databases maintained by the Human Resources department. Information on physical location of each employee—at different levels of spatial aggregation—was obtained from the Information Technology department that maintains an employee telephone and internal address directory. Unfortunately, these

demographic and employment information were made available to us only for year 2004, so (apart from age) we need to assume they are constant between 2002 and 2004. Presumably, physical location at the workplace changed for only few workers over the three years horizon, and so the misclassification of reference groups (described in detail below) is most likely negligible. Information on health care utilization and costs were obtained from administrative billing records that are maintained for all patients. We used an electronic database that was developed to track patient visits to their primary care physician along with routine preventive care to create a variable that tracked whether an individual was up-to-date on breast cancer screening, as well as construct high quality indicators of health status. This information is available for 2002, 2003, and 2004. We measure an individual's health status using the classification scheme in Elixhauser *et al.* (1998) which essentially uses health utilization data to create a set of indicator variables revealing whether an individual has a history of medical claims for a certain health condition. We refer to the conditions identified by this classification scheme as "comorbidities".[5] We use indicators to also construct a synthetic health status variable labeled "comor". This variable is a simple count of the number of comorbidities. Finally, we used a high quality, locally maintained Tumor registry that keeps careful records of all patients who are diagnosed with a malignant tumor of any type at the organization, or who are diagnosed elsewhere but treated at the organization, to create a cancer diagnosis variable for all employees. This information is available for all years between 2000 and 2004, which allows us to construct longer breast cancer histories in the reference group. However, we do not observe women who died from breast cancer because they are removed from the administrative records. Presumably, these women are evenly distributed across treatment and control groups, and so are unlikely to be an important source of bias. The common element in all of these data was a patient ID variable that allowed us to link records. Data obtained from the Human Resources department were pre-linked with the patient ID before being made available to us in order to protect confidential personal information.

[5] While it is true that these conditions, because of the way we identify them, are known only if a medical claim for a patient appears in the organization's billing records at some point during the employee's tenure, they all correspond to serious, often chronic, health problems that do not allow a person to stay away from a doctor for very long. Because the individuals in our dataset are fully covered individuals with access to a premier health care facility, it is very unlikely that more than a tiny fraction of those without a condition in our final data set actually have it. Therefore, we are confident that the comorbidity indicators we construct provide high-quality health information at the individual level.

Summary statistics for the final sample are reported in Tables 1 and 2.

Table 1. Summary statistics: demographic and job information

Variable	Description	Mean	Std. Dev	Min	Max
age	Age	54.55	3.56	50	64
married	Married	0.71	0.45	0	1
divorced	Divorced	0.15	0.36	0	1
widowed	Widowed	0.01	0.09	0	1
separated	Separated	0.03	0.17	0	1
famsize	Family size	2.11	1.08	1	7
child	Has children below 18	0.09	0.29	0	1
hourly	Whether hourly pay	0.97	0.18	0	1
docnurse	Medical doctor or nurse	0.25	0.43	0	1
fte	% of full time employee	91.37	14.22	50	100
tenure	Job tenure, years	19.35	10.61	0	46
new	New employee (tenure=0)	0.01	0.11	0	1
salary	Salary range	0.76	0.52	–	–
t_chg	Charges for health services	7.92	13.8	0	204.98

Notes: All means refer to year 2004, except for "new" which is an average over the 2002–2004 period. Variable "docnurse" is constructed using information on job titles available in our data set: docnurse=1 means that an individual is either a medical doctor or a nurse; docnurse=0 means that an individual has a different job title (e.g., patient appointment coordinator, assistant, secretary, etc.). Notice that, due to data limitations, our definition of doctors may include some people with a doctorate in areas outside medicine. These cannot be separated from medical doctors. In addition, employees with medical or nursing degrees employed in non-clinical jobs are not classified as doctors or nurses. Both these categories of people are likely to be very small. Salary range is expressed as a % of the median. Neither absolute values nor the maximum and minimum can be reported, upon an explicit request of the organization to protect the confidentiality of this information. Charges for health services is expressed in thousands of 2004 US dollars. The age group is 50+.

Table 2. Summary statistics: health information

Variable	Description	Mean	Std. Dev	Min	Max
bcancer	Breast cancer	0.004	0.060	0	1
comor	Number of comorbidities	0.824	1.110	0	11
neuro	Neurological disorders	0.013	0.115	0	1
chrnlung	Chronic lung disease	0.052	0.221	0	1
dm	Diabetes	0.046	0.209	0	1
dmcx	Diabetes w/complications	0.014	0.117	0	1
hypothy	Hypothyroidism	0.087	0.282	0	1
renlfail	Renal failure	0.001	0.037	0	1
chf	Congestive heart failure	0.001	0.037	0	1
arythm	Arythmia	0.035	0.185	0	1
valve	Valvular disorders	0.025	0.155	0	1
pulmcirc	Pulmonary circular disorders	0.002	0.043	0	1
perivasc	Perivascular disorders	0.009	0.092	0	1
para	Parathyroid disorders	0.001	0.025	0	1
liver	Liver disorders	0.008	0.087	0	1
ulcer	Ulcers	0.002	0.044	0	1
tumor	Tumor	0.061	0.240	0	1
arth	Arthritis	0.021	0.145	0	1
coag	Coagulation	0.004	0.061	0	1
obese	Extreme obesity	0.088	0.283	0	1
wghtloss	Significant weightloss	0.006	0.075	0	1
lytes	Electrolyte disorders	0.013	0.114	0	1
bldloss	Blood loss	0.001	0.025	0	1
anemdef	Anemic Deficiency	0.045	0.207	0	1
alcohol	Alcoholism	0.005	0.069	0	1
psych	Psychoanalytic Disorders	0.059	0.235	0	1
depress	Depression	0.031	0.173	0	1
lymph	Lymphatic disorders	0.002	0.047	0	1
mets	Metastatic cancer	0.006	0.078	0	1
htn	Hypertension	0.231	0.422	0	1

Notes: All means refer to observations pooled over the 2002–2004 period. Cancer information comes from the Tumor registry maintained by the organization. Comorbidity indicators are constructed from individual administrative health records using the classification scheme of Elixhauser et al. (1998); see text for details. The age group is 50+.

The insurance plan for all female employees in the eligible age group (i.e., above 40) includes free annual mammograms. The facility for screening is located on campus and is well enough staffed so as not to keep patients waiting for an appointment. These women are reminded about their recommended annual mammogram, if they have not already had one, each time they visit their primary care physician. Their physician is provided a copy of their electronic medical record and this lists the last time they had a mammogram conducted. If they are due for a mammogram, the physician generally schedules an appointment during the visit (or asks a nurse to do so) unless the patient refuses an appointment. In this study we focus on the age group 50 and older (about 3,200 women, or about 25% of the total), for two reasons. First, mammograms are not universally recommended before 50 by the medical community in the US, and they may be perceived as a less effective screening tool among younger women; thus both the screening recommendation and the perception of "normal prevention" in our sample may be ambiguous in the age range 41–49.[6] Second, we lack screening information in the age range 41–49 for year 2002, and so including this group would make the panel severely unbalanced. We show later that our results do not depend on the exclusion of the available portion of information for the 41–49 age group.[7]

Tables 3a and 3b report annual screening rates in our sample, by age range, by health status (as summarized by the "comor" variable), and by job title (as summarized by the "docnurse" variable). That is, the three individual characteristics along which we find heterogeneous treatment effects. The screening rates are larger than corresponding national rates, as expected for a group of women employed in the health industry with full

[6] There is some controversy over when women should begin breast cancer screening. The American Cancer Society, American College of Radiology, American Medical Association, National Cancer Institute and the American College of Obstetrics and Gynecology recommend annual screening starting at age 40. The American College of Physicians recommends that women in the age range 40–49 make decisions about mammography together with their provider, based on the individual risk profile and the potential costs and benefits of performing a mammogram. In November 2009 the US Preventive Services Task Force advised women below 50 years old not to get routine mammograms, but to discuss the pros and cons with their provider and decide together when to start screening. This advice—whose rationale is the fact that mammograms may generate false alarms and unnecessary treatment—has generated a lot of debate in the US. Moss *et al.* (2006) perform a large randomized controlled trial in the UK and find that annual mammographies in the age range 39–48 do not lead to a significantly lower mortality rate relative to control women who did not screen regularly.

[7] Of course we use women of all ages to identify cases of breast cancer in the period 2000-2004— information that is available for all women in the sample.

health coverage. The tables show a downward trend in screening rates in all age and health groups, in line with the national trend in the period under investigation. In our sample, screening rates tend to increase with age, as one expects given that age is the most important risk factor for the development of breast cancer.[8] Table 3b refers to the age group 50 and older, and indicates that healthy women are much less likely to screen for breast cancer than unhealthy ones—the gap is almost 20 percentage points, a very large difference. A possible explanation is that unhealthy women see their physician more often and so—given the way reminders are provided at the organization—are more likely to be scheduled for a mammogram, possibly as part of medical check-ups related to comorbidities. Finally, medical doctors and nurses screen slightly less than other employees, although the difference is not statistically significant in any of the three years.

A crucial piece of information is the administrative data on the physical location of employees at the workplace. Because we construct reference groups based on such information, it is important to describe the "geography" of our data set. The organization is administratively divided into departments that are often subdivided into divisions. Smaller departments tend to be physically located in one place, as are most divisions. Larger departments and divisions may have multiple physical locations across campus. The campus consists of about 30 buildings which house the vast majority of employees. Most buildings are multi-storied; large departments will often occupy multiple contiguous floors. It is common for two or more divisions within a department or in different departments to occupy one floor. Each floor has a mail room where the institution's internal mail service regularly delivers campus and external mail. In large buildings, a floor may have more than one mail room, whereas in small buildings, different floors may share a mail room. Each employee is assigned a mail room code based on their main office location. This mail room code defines the smallest (on average) clusters of employees observable in our data set. Therefore, we take the mail room as our first definition of reference groups, labeled "Mail" groups: this particular dimension defines groups of people who are likely to interact closely on a daily basis—both because they are very likely to belong

[8] Based on estimates from the Surveillance, Epidemiology, and End Results registry (SEER), the NCI estimates that the probability of developing breast cancer over the next 30 years for a woman aged 40 is 7.53%. This number increases to 9.68% at age 50 and then declines slightly to 9.54% at age 60—see http://www.cancer.gov/cancertopics/pdq/screening/breast/healthprofessional/page3 The slight decline in the estimated 30 year rate may be partially due to a substantial increase in all cause mortality for women over the age of 70.

to the same department and division, and because they necessarily meet in hallways, share facilities, etc.

Table 3a. Screening rates by year and age range

Year	Age 41–49	Age 50+	Age 50–54	Age 55–59	Age 60+
2002	—	0.769	0.758	0.785	0.812
	—	n=2407	n=1512	n=757	n=138
2003	0.619	0.760	0.738	0.787	0.806
	n=4086	n=2835	n=1685	n=898	n=252
2004	0.528	0.646	0.621	0.694	0.636
	n=4194	n=3267	n=1828	n=1041	n=398

Notes: The table reports the average, by year and age range, of the individual screening indicator, which takes value 1 if a woman performed a mammogram in a given year and 0 otherwise; n is sample size. The increase in sample size reflects the aging process.

Table 3b. Screening rates by year, health status, and job title

Year	comor=0	comor>0	docnurse=1	docnurse=0
2002	0.680	0.865	0.747	0.776
	n=1255	n=1144	n=582	n=1825
2003	0.654	0.856	0.731	0.768
	n=1351	n=1475	n=685	n=2151
2004	0.541	0.762	0.640	0.648
	n=1716	n=1538	n=810	n=2457

Notes: The first two columns report the average, by year and health status, of the individual screening indicator for the age group 50+. Health status is summarized by variable "comor", which counts the number of comorbidities: comor=0 means that an individual has no breast cancer and no comorbidities (i.e., is healthy); comor>0 means that an individual has no breast cancer but at least one comorbidity (i.e., is unhealthy); The last two columns report the same average, by year and job title for the age group 50+. Job title is here summarized by the variable "docnurse" (see Notes to Table 1 for details). The increase in sample size reflects the aging process.

A concern is that given the relatively small dimension of mail room groups as well as the particular setting of our study, the Stabe-Unit-Treatment-Value assumption may be violated: people may well interact with other employees outside the group defined by mailbox locations, which would result in attenuation bias. Therefore, we use an alternative, larger definition of reference groups at the floor level, labeled "Floor" groups. On average, Floor groups are almost three times larger than Mail groups, hence they extend the possible range of social interactions and allow for broader patterns of information sharing. Table 4 summarizes the number and size (counting both men and women) of Mail and Floor groups.[9] As a comparison, Esther Duflo and Emmanuel Saez (2003) define reference groups as departments, whose average size in terms of staff is about 30 in their data.

Table 4. Reference groups

Group	Number	Mean size	Std. dev.
Mail	861	52.0	43.7
Floor	288	138.3	100.1

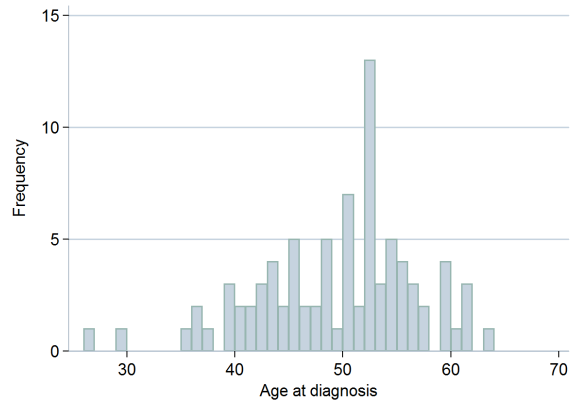
Notes: The Table reports the number and average size of the two alternative reference groups used in this study—reference groups defined by mailbox locations (Mail) and floors within buildings (Floor)—as well as the standard deviation of group size.

There are 10 women diagnosed with breast cancer in 2000, 21 in 2001, 14 in 2002, 20 in 2003, and 20 in 2004 in our data. These numbers correspond to an annual incidence ranging between 0.23% and 0.29%, in line with national estimates.[10] The age distribution of cancer diagnoses is plotted in Figure 1. Occurrences below age 40 are about 10% of the total, and those above 49 almost 60% of the total. These sample statistics and by and large in line with national estimates for the United States. These occurrences imply that about 5% of women in Mail groups and about 13% of women in Floor groups are treated—at different lags—every year between 2002 and 2004.

[9] There is a third, larger, definition of reference groups that we could use, namely entire buildings. However, the average dimension of reference groups in this case would be very large (about 1,200) and would very likely result in severe misclassification of treated and control individuals.

[10] The 230,480 new cases of breast cancer estimated by the National Cancer Institute in the US for 2011 are equivalent to an incidence of 0.31% of women above age 40, which were estimated to be roughly 75 million in the US in 2010.

Figure 1. Age distribution of breast cancer diagnoses, 2000–2004



Notes: The histogram represents the frequency distribution of breast cancer diagnosis, by age, pooling all female employees at the organization in the 2000-2004 period.

3 Econometrics

The outcome of interest is whether a woman performs a mammogram in a given year or not. We denote the screening indicator by $Y_t = 0, 1$. That is, a woman may screen during year t ($Y_t = 1$) or not ($Y_t = 0$). We are interested in the causal effect of breast cancer history in one's reference group on the individual propensity to screen. More precisely, belonging in year t to a group g where someone was diagnosed with breast cancer during that year is a binary event denoted $T_g^t = 0, 1$. That is, a member of group g may be diagnosed with breast cancer during year t ($T_g^t = 1$) or not ($T_g^t = 0$). We denote by $\mathbf{T}_g^t = \{T_g^k\}_{k=0}^t$ the history of such events in group g up to time t. In order to avoid misclassification, each T_g^t is then adjusted for the exact dates (i.e., day and month of year t) when breast cancer was diagnosed in a group and when women in that group screened, in a way to be illustrated in detail below. The conditional probability of screening (a structural object derived formally in Section 5) is equal to the conditional expectation of the binary screening indicator:

$$\Pr(Y_t = 1 | \mathbf{T}_g^t, \mathbf{X}) = \mathbb{E}[Y_t | \mathbf{T}_g^t, \mathbf{X}], \tag{1}$$

where $\mathbf{X}$ are the conditioning variables. The goal of the empirical analysis is to identify the dynamic average treatment effect of experiencing breast

cancer in the group of co-workers (i.e., the effect of treatment history $\mathbf{T}_g^t$) on the average propensity to screen, $\Pr(Y_t = 1|\mathbf{T}_g^t, \mathbf{X})$.

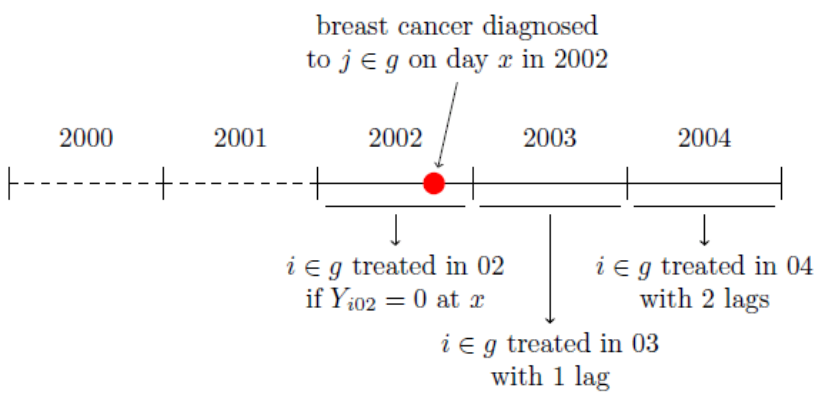
3.1 Treatment and control groups

A possible definition of treatment in a given year is the event that a co-worker was diagnosed with breast cancer during that year. This definition raises two issues. First, one may wonder whether there is any treatment at all. In fact we do not know whether a woman who develops breast cancer shares such information with her colleagues or not. We claim that it is very likely that co-workers soon become aware of the sick woman's condition. The reason is that a woman diagnosed with breast cancer typically leaves the workplace for some time, either to complete therapy or because of a decreased desire or ability to work, as documented by Michael Hassett *et al.* (2009). Cathy Bradley *et al.* (2005) have analyzed the effects of a new breast cancer diagnosis on labor supply of women who were employed before the diagnosis. They find that six months after the diagnosis, sick women are much less likely to be employed (about 20 percentage points relative to the control group) and if they are employed their weekly hours of work are 12% to 28% depressed relative to the control group, depending on the stage of the malignancy. It is very unlikely that such substantial changes in labor supply and prolonged absences go unnoticed by co-workers, even in the absence of word-of-mouth. This is particularly unlikely for severe cases of breast cancer: we will later exploit information on SEER grade (a measure of the severity of cancer) to show how the treatment effect varies with the severity of cases.

Second, because our data is at the annual frequency, this definition of treatment would result in attenuation bias, because we would incorrectly classify as "treated" women who could not respond to the emergence of breast cancer in their group. To see why, notice that according to this definition if a woman performed a mammogram in year t the day before a co-worker was diagnosed with breast cancer then this person is classified as treated, even if she was not treated when she decided to screen in year t. To fix this shortcoming we exploit high frequency information in the data set. Specifically, we know the exact calendar dates on which women in our sample performed mammograms, as well as the exact calendar dates on which sick women were diagnosed with breast cancer. We use this information to refine the definition of treatment, as follows: a woman i in group $g(i)$ is classified as treated in year t if a colleague $j \in g(i)$ of any age was diagnosed with breast cancer during year t *and* if in that year i was not up-to-date with

breast screening the day that j was diagnosed with the malignancy. In this case $T^t_{g(i)} = 1$. If, instead, she was up to date that day then she is not used in estimation that year. The control group, therefore, are all women belonging to reference groups where no one was diagnosed with breast cancer. For these women $T^t_{g(i)} = 0$. Lagged treatment is defined in a similar way and denoted $T^{t-k}_{g(i)}$, where k is the lag. Given the longitudinal limitations of our data set we restrict up front to treatment histories of the type $\mathbf{T}^t_{g(i)} = (T^t_{g(i)}, T^{t-1}_{g(i)}, T^{t-2}_{g(i)})$. To illustrate, consider the timeline in Figure 2. Suppose that $j \in g$ received the bad news on April 1st, 2002 ("day x", in the figure) If $i \in g$ had not performed a mammogram by that date, then she is classified as treated in 2002. Furthermore, she is classified as treated with 1 and 2 lags in 2003 and 2004, respectively. If $i \in g$ had performed a mammogram on, say, February 1st, 2002 then $i \in g$ is not used for estimation in 2002. However, in this case $i \in g$ is still classified as treated with 1 and 2 lags in 2003 and 2004, respectively, and used for estimation in those years.[11]

Figure 2. Timeline.



[11] We can perform only a partial adjustment for exact calendar dates in years 2002 and 2003. The reason is that although we observe all dates of breast cancer diagnosis, we only observe the date a woman last screened as of December 31st, 2004, not the complete screening history. Therefore, in 2002 and 2003 we cannot adjust the treatment indicator for those women who perform a mammogram every year. These are about 1/3 of the total. We will show later that the adjustment does not affect the pattern we identify, and that the impossibility to fully adjust for years 2002 and 2003 is very unlikely to do so.

3.2 Identification

For any individual i we observe one of eight counterfactual outcomes at each point in time:

$$Y_{it} = Y_{it}(\mathcal{T}_0, \mathcal{T}_1, \mathcal{T}_2) \quad \text{if} \;\; T^t_{g(i),0} = \mathcal{T}_0 \;\; \text{and} \; T^t_{g(i),1} = \mathcal{T}_1 \;\; \text{and} \; T^t_{g(i),2} = \mathcal{T}_2, \quad (2)$$

where $(\mathcal{T}_0, \mathcal{T}_1, \mathcal{T}_2) \in \{0,1\} \times \{0,1\} \times \{0,1\}$. The maintained identifying assumption is that the treatment is strictly exogenous conditional on individual and group characteristics:

$$\mathbf{T}_{g(i)t} \perp (Y_{it}(\mathcal{T}_0, \mathcal{T}_1, \mathcal{T}_2)) | \mathbf{x}_{it}, \mathbf{x}_{g(i)t}, \theta_i, \quad (3)$$

where $\mathbf{x}_{it}$ and $\mathbf{x}_{g(i)t}$ are time-varying individual and group covariates, respectively, and θ_i are individual fixed effects, respectively. A look at Figure 2 reveals that this conditional independence assumption is actually twofold. The first part requires that conditional on individual and group characteristics, a cancer diagnosis in one's reference group at any point in time is as good as randomly assigned (i.e., is independent of potential outcomes in any period).[12] This part follows easily from the fact that cancer arises randomly in one of two otherwise identical groups—the conditioning variables ensuring that such two groups are "identical". The second part requires that being not up-to-date *by the time a colleague is diagnosed* with breast cancer is also random with respect to potential outcomes. This part is more problematic simply because the two potential outcomes are being up-to-date or not *at the end of the year*. A testable, necessary condition for this second part to hold is that the distributions of screening dates for women without colleagues diagnosed with breast cancer and women who will have such a colleague at some future point during the year are not statistically different from each other. We show below why this is a necessary condition, and we test it in our data.

Under assumption (3) women belonging to groups where no one is diagnosed with cancer in a given year are a valid counterfactual for women belonging to groups where someone was and who were not up-to-date the day their co-worker was diagnosed. We assume constant treatment effects and we estimate the following linear probability model:

$$Y_{it} = \alpha \mathbf{t} + \beta \mathbf{x}_{it} + \gamma \mathbf{x}_{g(i)t} + \tau_0 T^t_{g(i)} + \tau_1 T^{t-1}_{g(i)} + \tau_2 T^{t-2}_{g(i)} + \theta_i + \varepsilon_{igt}, \quad (4)$$

[12] Of course this is not true for women who are diagnosed with breast cancer, who we exclude from the analysis after using them to classify other women as treated or not.

where $\mathbf{t}$ is a vector of year dummies, τ_0 is the contemporaneous treatment effect, τ_1 and τ_2 the lagged effects at 1 and 2 years distance, respectively, and ε_{igt} is the error term.

In order to corroborate the identifying assumptions, we first provide standard evidence about the statistical equivalence of treatment and control groups with respect to observable covariates. Tables 5 and 6 report the characteristics of treated and control women in Mail and Floor groups, respectively, for each of the three years, as well as the size of the reference group across treatment status. These characteristics tend to be well-balanced across the two groups. If in a given year we reject the hypothesis of equality of means for some variable, the same hypothesis for the same variable is either not rejected or the sign of the difference is reversed in another year. The test, of course, is more stringent for Floor groups, which are larger.

Table 5. Characteristics of treated and control groups, by year: Mail groups

	2002			**2003**			**2004**		
Variable	treat.	cont.	p-val	treat.	cont.	p-val	treat.	cont.	p-val
group size	87.5	48.2	0.00	111.9	45.2	0.00	55.5	49.2	0.14
age	53.6	53.9	0.35	54.1	54.2	0.77	55.0	54.5	0.18
married	0.71	0.70	0.90	0.72	0.71	0.80	0.62	0.72	0.05
divorced	0.17	0.16	0.79	0.17	0.16	0.58	0.20	0.15	0.13
widowed	0.00	0.01	0.30	0.02	0.01	0.11	0.02	0.01	0.22
separated	0.03	0.03	0.89	0.02	0.03	0.36	0.02	0.03	0.62
famsize	2.13	1.98	0.14	2.11	2.04	0.38	1.94	2.16	0.11
child	0.04	0.06	0.42	0.10	0.08	0.19	0.12	0.09	0.32
hourly	0.95	0.96	0.42	0.98	0.96	0.22	0.99	0.96	0.18
docnurse	0.32	0.24	0.09	0.22	0.24	0.52	0.17	0.25	0.08
fte	87.3	91.2	0.01	92.7	91.2	0.15	92.7	91.3	0.34
tenure	22.3	19.7	0.02	17.1	19.8	0.00	18.7	19.4	0.54
new	0.01	0.02	0.62	0.02	0.01	0.71	0.02	0.01	0.26
salary	0.87	0.76	0.04	0.67	0.78	0.01	0.69	0.77	0.16
t_chg	10.1	8.1	0.19	11.7	7.7	0.00	7.6	7.9	0.84
comor	0.76	0.79	0.77	1.06	0.89	0.05	1.09	0.79	0.01

Notes: The Table compares, for each year, the means of the observable characteristics of treated and control women in the age range 50+, when reference groups are defined by mailbox locations (Mail); the p-value refers to the test of the hypothesis that the difference between the two means is zero.

Table 6. Characteristics of treated and control groups, by year: Floor groups

	2002			**2003**			**2004**		
Variable	treat.	cont.	p-val	treat.	cont.	p-val	treat.	cont.	p-val
group size	220.5	126.6	0.00	234.9	123.2	0.00	253.5	122.1	0.00
age	53.7	53.9	0.46	54.0	54.2	0.14	54.6	54.5	0.81
married	0.71	0.70	0.57	0.71	0.71	0.80	0.70	0.71	0.49
divorced	0.14	0.16	0.35	0.16	0.15	0.61	0.16	0.15	0.52
widowed	0.01	0.01	0.56	0.01	0.01	0.21	0.01	0.01	0.21
separated	0.03	0.03	0.66	0.03	0.03	0.93	0.02	0.03	0.49
famsize	2.01	1.98	0.20	2.08	2.04	0.41	2.12	2.11	0.84
child	0.05	0.06	0.31	0.09	0.08	0.27	0.10	0.09	0.44
hourly	0.95	0.96	0.34	0.97	0.96	0.75	0.97	0.96	0.45
docnurse	0.42	0.22	0.00	0.24	0.24	0.82	0.15	0.27	0.00
fte	87.9	91.4	0.00	92.5	91.1	0.07	93.4	91.1	0.00
tenure	22.2	19.5	0.00	17.8	19.9	0.00	18.0	19.6	0.01
new	0.01	0.02	0.19	0.02	0.01	0.13	0.02	0.01	0.03
salary	0.92	0.74	0.00	0.73	0.77	0.19	0.71	0.77	0.02
t_chg	8.8	8.1	0.49	9.3	7.8	0.06	7.4	7.9	0.48
comor	0.77	0.79	0.74	1.02	0.88	0.05	0.84	0.79	0.46

Notes: The Table compares, for each year, the observable characteristics of treated and control women in the age range 50+, when reference groups are defined by floors within buildings (Floor); the p-value refers to the test of the hypothesis that the difference between the two means is zero.

The only systematic difference pertains to group size: treated women always belong to larger groups. This has an obvious explanation: treatment is defined by belonging to a group where a woman was diagnosed with breast cancer, and the larger the group the higher the probability of finding a sick woman in that group. In the interest of space we do not report the analog of Tables 5 and 6 for the comorbidity indicators summarized in Table 2: these are also well-balanced across treatment and control groups.

Next, we provide evidence supporting the second part of the identifying assumption. That is, being not up-to-date, in a given year, by the time a colleague is diagnosed with breast cancer during that year is also as good as randomly assigned. The derivation of a testable hypothesis requires a little extra notation. Think of time as being partitioned in years but flowing continuously within years, as in Figure 2. Let δ be the first instant of year t, $\delta + 1$ the first instant of year $t + 1$, and so on. Define the following two binary random variables,

$$
\begin{aligned}
Y_t &= \mathbb{I}[\text{screened between } \delta \text{ and } \delta+1], \\
d_t &= \mathbb{I}[\text{colleague diagnosed with cancer between } \delta \text{ and } \delta+1],
\end{aligned}
$$

where $\mathbb{I}$ denotes the indicator function, and two auxiliary random variables: σ, which denotes the instant a woman last screened, and x, which denotes the instant a colleague was last diagnosed with cancer. It is understood that if a woman never screened or if a colleague was never diagnosed with breast cancer, then $\sigma = -\infty$ and $x = -\infty$, respectively. Let P be a probability function. It follows from these definitions that

$$
\begin{aligned}
\Pr(Y_t = 1) &= P(\delta \leq \sigma < \delta + 1), \\
\Pr(Y_t = 1 | d_t = 1) &= P(\delta \leq \sigma < \delta + 1 | \delta \leq x < \delta + 1), \\
\Pr(Y_t = 1 | d_t = 0) &= P(\delta \leq \sigma < \delta + 1 | x < \delta).
\end{aligned}
$$

The relation between $\Pr(Y_t = 1|d_t = 1)$ and $\Pr(Y_t = 1|d_t = 0)$ is left unrestricted, because this is the treatment effect of interest. That is:

$$
P(\delta \leq \sigma < \delta + 1 | d_t = 1) \gtreqless P(\delta \leq \sigma < \delta + 1 | d_t = 0),
$$

which can also be written as:

$$
P(\delta \leq \sigma < \delta + 1 | \delta \leq x < \delta + 1) \gtreqless P(\delta \leq \sigma < \delta + 1 | x < \delta). \tag{5}
$$

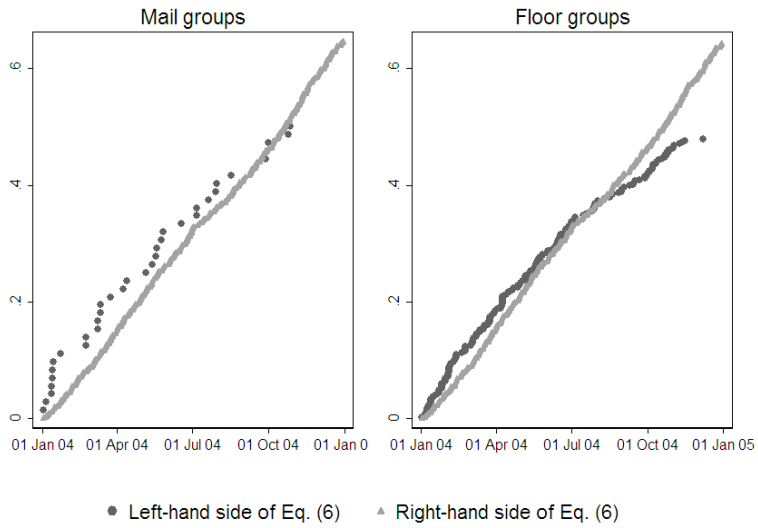
A necessary condition for the second part of the identifying assumption to hold is that for any instant τ such that $\delta \leq \tau < \delta + 1$, the following holds:

$$
P(\delta \leq \sigma \leq \tau | \tau \leq x < \delta + 1) = P(\delta \leq \sigma \leq \tau | x < \delta). \tag{6}
$$

The right-hand side is simply the cumulative probability—at each date of a given year—of screening in groups where no woman was diagnosed with breast cancer during that year. The left-hand side, instead, is the cumulative probability—at each date of a given year—of screening before a co-worker is diagnosed with breast cancer, in those groups where breast cancer will emerge during the remaining portion of that year. Condition (6) does not restrict the relation in (5), and can be tested. Figure 3 illustrates the empirical analogs of these two cumulative probabilities in our sample for year 2004. The two cumulative probabilities look quite similar. If we restrict to values below 0.5—the largest value taken by the RHS of (6) in the data—then a formal Kolmogorov-Smirnov test cannot reject the hypothesis

that the two distributions are equal. That is, the difference between the two is not statistically significant, with an exact p-value of essentially 1, both at the Mail and Floor group level.

Figure 3. Timing of screening



Notes: The picture shows the empirical analogs of the two sides of equation (6) for Mail and Floor groups. The analogs of the RHS reported in the picture are the cumulative frequency of screening in Mail and Floor groups where no woman was diagnosed with breast cancer during 2004. The analogs of the left-hand side are the cumulative frequency of screening before a co-worker is diagnosed with breast cancer, in Mail and Floor groups where breast cancer will emerge during the remaining portion of 2004.

4 Results

We begin inference with a nonparametric analysis of screening rates by treatment status for both Mail and Floor groups, reported in Table 7. The p-values refer to tests of the null hypothesis that the difference between the screening rates of treated and control individuals is zero. The table shows that the contemporaneous effect ("At time t") of the treatment is negative, except in 2003. However, the 2003 difference is not statistically significant

at conventional significance levels. As for the lagged effect ("At time $t-1$" and "At time $t-2$"), this is generally also negative, except in 2004, although lower in magnitude and statistically insignificant.

Table 7. Screening rates by year and treatment status.

Mail groups

	At time t (current)			At time $t-1$ (lag 1)			At time $t-2$ (lag 2)		
t	treat.	cont.	p-val	treat.	cont.	p-val.	treat.	cont.	p-val.
2002	0.674	0.773	0.025	0.729	0.772	0.242	0.770	0.769	0.978
	(0.048)	(0.009)		(0.037)	(0.009)		(0.054)	(0.009)	
	n=95	n=2311		n=144	n=2263		n=61	n=2346	
2003	0.778	0.757	0.513	0.735	0.761	0.521	0.730	0.761	0.363
	(0.029)	(0.008)		(0.042)	(0.008)		(0.035)	(0.008)	
	n=198	n=2627		n=113	n=2722		n=159	n=2677	
2004	0.480	0.647	0.001	0.700	0.645	0.500	0.656	0.646	0.799
	(0.051)	(0.009)		(0.031)	(0.009)		(0.040)	(0.008)	
	n=98	n=3132		n=220	n=3032		n=131	n=3135	

Floor groups

	At time t (current)			At time $t-1$ (lag 1)			At time $t-2$ (lag 2)		
t	treat.	cont.	p-val.	treat.	cont.	p-val.	treat.	cont.	p-val.
2002	0.733	0.773	0.136	0.736	0.775	0.097	0.774	0.769	0.870
	(0.029)	(0.009)		(0.022)	(0.009)		(0.032)	(0.009)	
	n=273	n=2128		n=387	n=2020		n=177	n=2230	
2003	0.785	0.753	0.164	0.752	0.763	0.666	0.725	0.769	0.066
	(0.021)	(0.009)		(0.024)	(0.009)		(0.021)	(0.009)	
	n=396	n=2417		n=335	n=2493		n=444	n=2392	
2004	0.507	0.643	0.000	0.643	0.708	0.007	0.650	0.648	0.920
	(0.024)	(0.009)		(0.021)	(0.009)		(0.024)	(0.009)	
	n=424	n=2655		n=449	n=2788		n=383	n=2874	

Notes: The two tables report the average, by year and lag of treatment, of the individual screening indicator (which takes value 1 if a woman performed a mammogram in a given year and 0 otherwise) of treated and control women in the age range 50 and older, when reference groups are defined by mailbox locations (Mail) and floors within buildings (Floor), respectively; n is sample size; standard deviations are given in parentheses; the p-value refers to the test of the hypothesis that the difference between the two means is different from zero.

Next, we estimate equation (4) on the three-year panel using the fixed-effects estimator and clustering standard errors at the individual level. All specifications (except those using "comor" as a regressor) include the time-varying comorbidity indicators summarized in Table 2, both at the individual level and at the group level—i.e., $\mathbf{x}_{it}$ and $\mathbf{x}_{g(i)t}$ in equation (4). The baseline results are reported in Table 8. Columns 1 and 2 show a negative and significant response of the propensity to screen to the treatment in the current year (bcancergroup$_t$): women whose colleague was diagnosed with breast cancer in a given year become amost 14 percentage points less likely to screen during that year when groups are defined by campus mail code and about 8 points when groups are defined by a floor within a building. This difference across the two alternative reference groups suggests that there is no attenuation bias when using the smaller Mail groups. On the contrary, the weaker effect in the larger group (where physical and social distance from the woman diagnosed with cancer is, on average, higher) suggests that Mail groups are a better measure of reference groups in our data. The coefficients on the lagged treatment indicators (bcancergroup$_{t-1}$ and bcancergroup$_{t-2}$) are also negative, and indicate that the effect of the treatment has some persistence: after 2 years treated women are still about 7 (in Mail groups) and 5 (in Floor groups) percentage points less likely to screen than women in the control group. Columns 3 and 4 of Table 8 report results from a "placebo" specification: the fact that breast cancer will emerge in a group in the following year (bcancergroup$_{t+1}$) does not help predicting the screening propensity in the current year, as one would expect.

Table 9 shows the results we obtain from these regressions without correcting the definition of treatment for the exact calendar dates of screening and breast cancer diagnosis, as described in Section 3.1. The table shows that the pattern we identify is not an artifact of the adjustment. The fact that coefficients are smaller than in Table 8 (down to statistical insignificance at the Floor level) indicates that the adjustment is doing precisely what it is supposed to do, namely remove the attenuation bias resulting from classifying as treated women who are actually not treated because they could not respond in a given year to the emergence of breast cancer in the reference group that same year. Attenuation bias, of course, is more severe at the Floor level: these groups are larger and so more control women are misclassified as treated if one does not adjust for calendar dates.

Table 8. Results: baseline

	1	2	3	4
	up2date$_t$	up2date$_t$	up2date$_t$	up2date$_t$
bcancergroup$_t$	−0.138**	−0.083**		
	(0.030)	(0.019)		
bcancergroup$_{t-1}$	−0.081**	−0.048*		
	(0.026)	(0.021)		
bcancergroup$_{t-2}$	−0.073*	−0.051*		
	(0.031)	(0.022)		
bcancergroup$_{t+1}$			0.028	−0.005
			(0.022)	(0.015)
Constant	0.722**	0.718**	0.710**	0.712**
	(0.020)	(0.030)	(0.019)	(0.030)
Reference Group	Mail	Floor	Mail	Floor
Age	50+	50+	50+	50+
Fixed effects	Yes	Yes	Yes	Yes
Individual controls	Yes	Yes	Yes	Yes
Group controls	Yes	Yes	Yes	Yes
Year dummies	Yes	Yes	Yes	Yes
Observations	8,099	7,931	8,147	8,147
Individuals	3,221	3,203	3,226	3,226

Notes: Linear probability models, fixed-effects estimator. In all models the dependent variable, up2date$_t$, is the individual screening indicator at time $t = 1, 2, 3$, which takes value 1 if a woman performed a mammogram year t and 0 otherwise; bcancergroup$_t$ is the current-year treatment indicator (whether someone in the group was diagnosed with breast cancer); bcancergroup$_{t-1}$ and bcancergroup$_{t-2}$ are the first and second lags, respectively, of the treatment indicator (whether someone in the group was diagnosed with breast cancer at time $t-1$ and $t-2$, respectively); bcancergroup$_{t+1}$ is the next-year treatment indicator (placebo); reference groups are defined by either mailbox locations (Mail) or floors within buildings (Floor); robust standard errors clustered at the individual level are given in parentheses; * significant at 5%; ** significant at 1%.

Table 9. Results: baseline, no calendar dates adjustment

	1	2	3	4
	up2date$_t$	up2date$_t$	up2date$_t$	up2date$_t$
bcancergroup$_t$	−0.070*	−0.006		
	(0.029)	(0.019)		
bcancergroup$_{t-1}$	−0.066*	−0.028		
	(0.026)	(0.020)		
bcancergroup$_{t-2}$	−0.055	−0.045*		
	(0.031)	(0.021)		
bcancergroup$_{t+1}$			0.029	−0.005
			(0.022)	(0.015)
Constant	0.718**	0.715**	0.710**	0.712**
	(0.019)	(0.030)	(0.019)	(0.030)
Reference Group	Mail	Floor	Mail	Floor
Age	50+	50+	50+	50+
Fixed effects	Yes	Yes	Yes	Yes
Individual controls	Yes	Yes	Yes	Yes
Group controls	Yes	Yes	Yes	Yes
Year dummies	Yes	Yes	Yes	Yes
Observations	8,147	8,147	8,147	8,147
Individuals	3,226	3,226	3,226	3,226

Notes: Linear probability models, fixed-effects estimator. In all models the dependent variable, up2date$_t$, is the individual screening indicator at time $t = 1, 2, 3$, which takes value 1 if a woman performed a mammogram year t and 0 otherwise; bcancergroup$_t$ is the current-year treatment indicator (whether someone in the group was diagnosed with breast cancer); bcancergroup$_{t-1}$ and bcancergroup$_{t-2}$ are the first and second lags, respectively, of the treatment indicator (whether someone in the group was diagnosed with breast cancer at time $t-1$ and $t-2$, respectively); bcancergroup$_{t+1}$ is the next-year treatment indicator (placebo); reference groups are defined by either mailbox locations (Mail) or floors within buildings (Floor). Contrary to Table 8, in this table variable bcancergroup$_t$ is not adjusted for calendar dates of screening and breast cancer diagnosis (see Section 3.1 for details about such correction); robust standard errors clustered at the individual level are given in parentheses. * significant at 5%; ** significant at 1%.

Table 10 replicates the baseline regression after including women in the age group 41–49. The pattern is insensitive to excluding or including this group. However, for the reasons outlined in Section 2, we believe the most reliable results are those based on the sample of women age 50 and older.[13]

Table 10. Results: baseline, including age group 41–49

	1	2	3	4
	up2date$_t$	up2date$_t$	up2date$_t$	up2date$_t$
bcancergroup$_t$	−0.140**	−0.125**		
	(0.025)	(0.017)		
bcancergroup$_{t-1}$	−0.054*	−0.037*		
	(0.023)	(0.018)		
bcancergroup$_{t-2}$	−0.054*	−0.031		
	(0.026)	(0.018)		
bcancergroup$_{t+1}$			−0.008	−0.012
			(0.019)	(0.013)
Constant	0.667**	0.664**	0.662**	0.660**
	(0.017)	(0.026)	(0.017)	(0.026)
Reference Group	Mail	Floor	Mail	Floor
Age	41+	41+	41+	41+
Fixed effects	Yes	Yes	Yes	Yes
Individual controls	Yes	Yes	Yes	Yes
Group controls	Yes	Yes	Yes	Yes
Year dummies	Yes	Yes	Yes	Yes
Observations	15,876	15,522	16,007	16,007
Individuals	7,345	7,322	7,352	7,352

Notes: Linear probability models, fixed-effects estimator. The dependent variable and the regressors are defined as in Tables 8 and 9; robust standard errors clustered at the individual level are given in parentheses. * significant at 5%; ** significant at 1%.

[13] Notice that the possible residual attenuation bias in the lagged effect following from the partial adjustment for calendar dates in 2002 and 2003 is most likely negligible. A comparison of Table 8 and Table 9 reveals that not correcting for calendar dates in 2002 and 2003 would lead to lagged coefficients of 0.66 and 0.55 instead of 0.81 and 0.73 after the correction. It's unlikely that correcting the treatment indicator for the 1/3 of women for whom we cannot perform the correction would generate sensibly larger lagged coefficients.

We investigated possible heterogeneous responses underlying this average treatment effect, and we found noteworthy heterogeneity along four dimensions: severity of breast cancer, age, health, and job title. In particular, we found differences deriving from: (*i*) exposure to cases of breast cancer of different severity; (*ii*) age at the time of treatment; (*iii*) health status at the time of treatment; (*iv*) being a doctor or nurse rather than an employee with a different job title. Such heterogeneity is illustrated next.

We first exploit information on the severity of each case of breast cancer. Our data contains information on breast cancer grade along the Surveillance, Epidemiology, and End Results (SEER) four-grade scale. Simply put, a grade on the SEER 1–4 scale (more precisely, the ICD-O-3 grade code) is a measure of the speed at which breast cancer cells are growing, from low (1) to high (4). We classify grades 1–2 as mild cases (these are 1/3 of all cases in our data) and grades 3–4 as severe cases (these are the remaining 2/3 in our data). The latter may be thought of as providing the "real" treatment, or a stronger treatment: contrary to mild cases, severe cases are very unlikely to go unnoticed, because they have more visible consequences and news about them is likely to spread faster within the reference group. We re-estimated the model separately for the two cases. The results are reported in Table 11. Here the treatment is defined, alternatively, as belonging to a group where someone was diagnosed with a grade 1–2 breast cancer (columns 1 and 2) or a grade 3–4 cancer (columns 3 and 4). This table shows that at the Mail group level the impact treatment effect when exposed to severe cases of breast cancer is almost twice as large as the treatment effect when exposed to milder cases: the point estimates of the contemporaneous effect are −18 and −10.7 percentage points, respectively. In the following year, though, the magnitude of the residual effect is similar, although not always statistically significant. At the Floor group level, instead, the effect when exposed to grade 1–2 cancer is not significantly different from zero. This heterogeneity has a simple interpretation: because breast cancer status is private information, it is possible that only severe cases become known to co-workers because they have more visible effects—such as longer absences from work, aesthetic effects of chemotherapy, etc. Along this line of interpretation, it is interesting to note that mild cases have lagged effects similar to those of severe cases. In Mail groups the magnitude is about 7 percentage points after one year. Even mild cases are likely to become known after a year, when information had sufficient time to spread or the sick woman's condition worsened.

We next investigate heterogeneous responses in age by estimating the model separately for two different age ranges: 50–54 (56% of the total) and 55+ (44%). The results are reported in Table 12. This table shows that at

least the contemporaneous response of women between 50 and 54 years of age is larger (in absolute value) than the response in the age range 55 and older. That the response is larger for younger women is also suggested by a comparison between Tables 8 and 10: when women in the age range 41–49 are included the contemporaneous response is slightly larger than when they are excluded. This different behavioral response is consistent with what is known about the evolution of noncognitive traits along the life cycle. For instance, Daniel Read and N.L. Read (2004) find that in a health decision problem — When to face a hypothetical flu? — the discount factor increases monotonically with age; Brent Roberts, Kate Walton, and Wolfgang Viechtbauer (2006) show that noncognitive traits such as emotional stability and conscientiousness—which may have a bearing on the problem we study—increase monotonically with age; finally, John Ameriks, Andrew Caplin, John Lehay, and Tom Tyler (2007) find that younger individuals have more self-control problems than older ones.

We then use the "comor" variable (total number of comorbidities) described in Section 2 as a synthetic indicator (relative to the single comorbidity indicators) of a woman's overall health status: a woman with zero comorbidities (that is, no conditions) can be considered "healthy". We estimate the model separately for women with zero comorbidities (51% of the total) and with at least one comorbidity (49%). In this regression, of course, we cannot include comorbidity indicators as additional regressors. The results are reported in Table 12. As this table shows, the negative effect is somewhat stronger and more persistent for healthy women (comor=0), especially at the Mail group level. A possible explanation is that women with health conditions see doctors more often. As a consequence, they may be more accustomed, or more strongly recommended by doctors, to perform clinical tests. This includes reminders to perform mammograms—as illustrated in Section 2— which is consistent with the higher screening rate reported in Table 3b.

Finally, we explore the role of one's profession within the organization. A group of obvious interest is the group of medical doctors and nurses. This group, by training and experience, should be more knowledgeable than average about health issues. In particular, it should be more aware of the underlying cancer risk and of the benefits (and costs, as well) of screening and so may be expected to respond more rationally to indirect experiences of breast cancer. Table 14 shows that the impact treatment effect for this group is substantially smaller than average, and actually never statistically significant—although lack of significance may just reflect the reduced sample size. The dynamic effect is also quite different. The effect for those who

are neither doctors nor nurses is very persistent: within the time horizon we can exploit (i.e., 3 years) the impact effect declines by no more than 3 percentage points. Therefore, it seems that medical doctors and nurses are indeed behaving more rationally in our data set.

Table 11. Results: severity of cancer

	1	2	3	4
	up2date$_t$	up2date$_t$	up2date$_t$	up2date$_t$
bcancergroup$_t$	−0.107*	−0.022	−0.180**	−0.091**
	(0.036)	(0.024)	(0.047)	(0.026)
bcancergroup$_{t-1}$	−0.082*	−0.029	−0.063	−0.033
	(0.032)	(0.026)	(0.044)	(0.027)
bcancergroup$_{t-2}$	−0.078	−0.019	−0.054	−0.046
	(0.046)	(0.035)	(0.042)	(0.026)
Constant	0.714**	0.710**	0.720**	0.714**
	(0.031)	(0.020)	(0.020)	(0.030)
Cancer grade	1–2	1–2	3–4	3–4
Reference Group	Mail	Floor	Mail	Floor
Age	50+	50+	50+	50+
Fixed effects	Yes	Yes	Yes	Yes
Individual controls	Yes	Yes	Yes	Yes
Group controls	Yes	Yes	Yes	Yes
Year dummies	Yes	Yes	Yes	Yes
Observations	8,099	7,931	8,099	7,931
Individuals	3,221	3,203	3,221	3,203

Notes: Linear probability models, fixed-effects estimator. In all models the dependent variable, up2date$_t$, is the individual screening indicator at time $t = 1, 2, 3$, which takes value 1 if a woman performed a mammogram year t and 0 otherwise; bcancergroup$_t$ is the current-year treatment indicator (whether someone in the group was diagnosed with breast cancer); bcancergroup$_{t-1}$ and bcancergroup$_{t-2}$ are the first and second lags, respectively, of the treatment indicator (whether someone in the group was diagnosed with breast cancer at time $t-1$ and $t-2$, respectively); cancer grade is measured on the SEER 1–4 scale, see text for details; reference groups are defined by either mailbox locations (Mail) or floors within buildings (Floor); robust standard errors clustered at the individual level are given in parentheses; * significant at 5%; ** significant at 1%.

Table 12. Results: age 50–54 vs. 55+

	1	2	3	4
	up2date$_t$	up2date$_t$	up2date$_t$	up2date$_t$
bcancergroup$_t$	−0.161**	−0.094**	−0.122**	−0.037
	(0.042)	(0.026)	(0.049)	(0.030)
bcancergroup$_{t-1}$	−0.078*	−0.036	−0.073	−0.060
	(0.038)	(0.029)	(0.049)	(0.035)
bcancergroup$_{t-2}$	−0.073	−0.070*	−0.048	−0.025
	(0.044)	(0.032)	(0.049)	(0.033)
Constant	0.730**	0.732**	0.735**	0.740**
	(0.029)	(0.031)	(0.031)	(0.034)
Reference Group	Mail	Floor	Mail	Floor
Age	50-54	50-54	55+	55+
Fixed effects	Yes	Yes	Yes	Yes
Individual controls	Yes	Yes	Yes	Yes
Group controls	Yes	Yes	Yes	Yes
Year dummies	Yes	Yes	Yes	Yes
Observations	4,762	4,674	3,337	3,356
Individuals	2,295	2,277	1,417	1,418

Notes: Linear probability models, fixed-effects estimator. In all models the dependent variable, up2date$_t$, is the individual screening indicator at time $t = 1, 2, 3$, which takes value 1 if a woman performed a mammogram year t and 0 otherwise; bcancergroup$_t$ is the current-year treatment indicator (whether someone in the group was diagnosed with breast cancer); bcancergroup$_{t-1}$ and bcancergroup$_{t-2}$ are the first and second lags, respectively, of the treatment indicator (whether someone in the group was diagnosed with breast cancer at time $t-1$ and $t-2$, respectively); reference groups are defined by either mailbox locations (Mail) or floors within buildings (Floor); robust standard errors clustered at the individual level are given in parentheses; * significant at 5%; ** significant at 1%.

Table 13. Results: no conditions vs. at least one condition

	1	2	3	4
	up2date$_t$	up2date$_t$	up2date$_t$	up2date$_t$
bcancergroup$_t$	−0.133**	−0.079**	−0.097*	−0.072**
	(0.046)	(0.029)	(0.040)	(0.026)
bcancergroup$_{t-1}$	−0.084	−0.037	−0.034	−0.066*
	(0.044)	(0.035)	(0.033)	(0.026)
bcancergroup$_{t-2}$	−0.073	−0.042	−0.045	−0.032
	(0.048)	(0.036)	(0.038)	(0.029)
Constant	0.694**	0.690**	0.836**	0.852**
	(0.034)	(0.036)	(0.030)	(0.032)
Comorbidities	0	0	1 or more	1 or more
Reference Group	Mail	Floor	Mail	Floor
Age	50+	50+	50+	50+
Fixed effects	Yes	Yes	Yes	Yes
Individual controls	No	No	No	No
Group controls	Yes	Yes	Yes	Yes
Year dummies	Yes	Yes	Yes	Yes
Observations	4,285	4,213	4,129	4,033
Individuals	2,205	2,188	2,056	2,036

Notes: Linear probability models, fixed-effects estimator. In all models the dependent variable, up2date$_t$, is the individual screening indicator at time $t = 1, 2, 3$, which takes value 1 if a woman performed a mammogram year t and 0 otherwise; bcancergroup$_t$ is the current-year treatment indicator (whether someone in the group was diagnosed with breast cancer); bcancergroup$_{t-1}$ and bcancergroup$_{t-2}$ are the first and second lags, respectively, of the treatment indicator (whether someone in the group was diagnosed with breast cancer at time $t-1$ and $t-2$, respectively); reference groups are defined by either mailbox locations (Mail) or floors within buildings (Floor); robust standard errors clustered at the individual level are given in parentheses; * significant at 5%; ** significant at 1%.

Table 14. Results: medical doctors and nurses vs. the rest

	1	2	3	4
	up2date$_t$	up2date$_t$	up2date$_t$	up2date$_t$
bcancergroup$_t$	−0.072	−0.059	−0.139**	−0.085**
	(0.065)	(0.038)	(0.034)	(0.022)
bcancergroup$_{t-1}$	−0.036	−0.052	−0.098**	−0.058*
	(0.051)	(0.036)	(0.032)	(0.025)
bcancergroup$_{t-2}$	−0.083	−0.036	−0.108**	−0.053*
	(0.069)	(0.045)	(0.036)	(0.025)
Constant	0.743**	0.737**	0.703**	0.707**
	(0.046)	(0.050)	(0.021)	(0.022)
Job title	MD/nurse	MD/nurse	other	other
Reference Group	Mail	Floor	Mail	Floor
Age	50+	50+	50+	50+
Fixed effects	Yes	Yes	Yes	Yes
Individual controls	Yes	Yes	Yes	Yes
Group controls	Yes	Yes	Yes	Yes
Year dummies	Yes	Yes	Yes	Yes
Observations	2,068	2,029	6,346	6,217
Individuals	809	806	2,451	2,437

Notes: Linear probability models, fixed-effects estimator. In all models the dependent variable, up2date$_t$, is the individual screening indicator at time $t = 1, 2, 3$, which takes value 1 if a woman performed a mammogram year t and 0 otherwise; bcancergroup$_t$ is the current-year treatment indicator (whether someone in the group was diagnosed with breast cancer); bcancergroup$_{t-1}$ and bcancergroup$_{t-2}$ are the first and second lags, respectively, of the treatment indicator (whether someone in the group was diagnosed with breast cancer at time $t-1$ and $t-2$, respectively); In the Job title row, "MD/nurse" indicates the group of medical doctors and nurses and "other" indicates the group of those who are neither medical doctors nor nurses—see Notes to Table 1 for details; reference groups are defined by either mailbox locations (Mail) or floors within buildings (Floor); robust standard errors clustered at the individual level are given in parentheses; * significant at 5%; ** significant at 1%.

5 Rationalization

Finally, we lay down a model to rationalize our results. The model consists of a simple expected utility framework along the lines of Gabriel Picone *et al.* (2004) and Julia Witt (2008). We consider a dynamic setting and a noninfectious disease which may lead to death. Time is discrete and, in what follows, time and age are indistinguishable. An individual's health capital at time t is denoted h_t. Because the fundamental difference is between being alive or not, we assume this stock can take only two values:

$$h_t = \begin{cases} h > 0 & \text{if individual is alive at time } t, \\ 0 & \text{if individual is dead at time } t. \end{cases} \tag{7}$$

The stock of health capital yields a benefits flow at a constant rate r at the beginning of each period. We denote by s_t the individual's sickness status at time t, a binary variable:

$$s_t = \begin{cases} 1 & \text{if individual is sick at time } t, \\ 0 & \text{if individual is healthy at time } t. \end{cases} \tag{8}$$

The (subjective) probability of being sick at time t is denoted p_t. A screening test is available in every period at a cost $c > 0$, measured in utility units. The test reveals the unknown sickness status without error.[14] That is, the uncertainty about one's sickness status can be resolved by screening. The screening indicator is denoted $Y_t = 0, 1$. After screening the probability of being sick is updated as follows:

$$p_t = \begin{cases} 1 & \text{if } Y_t = 1 \text{ and } s_t = 1, \\ 0 & \text{if } Y_t = 1 \text{ and } s_t = 0. \end{cases} \tag{9}$$

If the individual screens ($Y_t = 1$) and tests positive ($s_t = 1$) then she receives medical care at no cost. Medical care allows health capital to be maintained at level $h > 0$ with probability π. However, with probability $1 - \pi$ the intervention fails and health capital falls to 0. If the individual

[14] We ignore the possibility of test errors of type I and II (false negative and false positive, respectively) so to keep the model as simple as possible. It is straightforward to extend the model to allow for type I and II errors, but such errors would play no substantial role in the interpretation of the results (an extended version of the model including such extra parameters is available from the authors upon request). However, they may be important in practice. For instance, the possibility of false positives implies the possibility of unnecessary treatment—in which case screening per se can have a negative impact on health.

screens ($Y_t = 1$) and tests negative ($s_t = 1$) or does not screen ($Y_t = 0$), then no medical care is received. However, if no medical care is received when the individual is sick then her health capital drops to 0 with probability 1. That is, the dynamics of health capital is given by:

$$h_{t+1} = \begin{cases} h_t & \text{if } s_t = 0 \text{ or, w/prob. } \pi,\ s_t = 1 \text{ and } Y_t = 1; \\ 0 & \text{if } h_t = 0 \text{ or } s_t = 1 \text{ and } Y_t = 0 \text{ or, w/prob. } 1-\pi,\ s_t = 1 \text{ and } Y_t = 1 \end{cases} \tag{10}$$

This extreme formulation captures in a straightforward way the idea that screening for breast cancer does not add anything to a woman's health, but can save her life if breast cancer is present and is detected early enough. Death occurs at an endogenous death τ:

$$\tau = \min\{t : h_t = 0\},$$

and period utility is given by:

$$U_t = \begin{cases} rh - cY_t + a(p_t) + \varepsilon(Y_t) & \text{if } t < \tau \\ 0 & \text{if } t \geq \tau \end{cases}$$

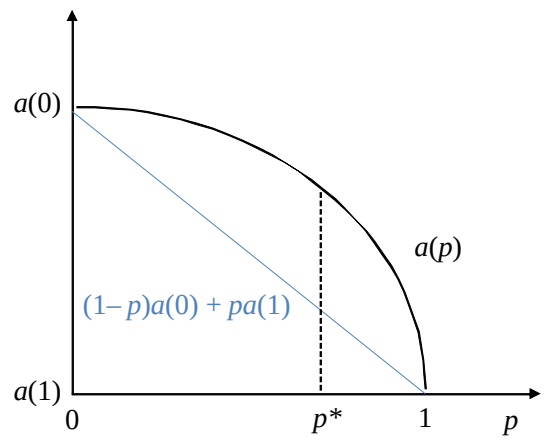
where r is rate at which health capital yields its benefit, $\varepsilon(Y_t)$ represents unobserved taste variation, and $a(p_t)$ is a decreasing and concave anticipatory component of utility, to be described below. In order to ensure that in this model death is the worst possible outcome, we assume that the support of $\varepsilon(Y_t)$ is finite and that $U_t(Y_t) > 0$ when $t < \tau$.

Function $a(p_t)$ captures the dependence of preferences on beliefs about having the disease, and is known as anticipatory utility.[15] Simply put, this component allows for utility from thinking today (worrying, in this case) about what could happen in the future. Since having breast cancer is an uncertain event until a woman screens, anticipatory utility is utility from beliefs. In particular, the person has lower anticipatory utility the higher the belief that she is sick because this means negative events such as dying are more likely. This has implications for the propensity to collect information about the likelihood of such events: by screening, one may find that she is sick and thus experience the worst possible anticipatory feeling, $a(1)$. Notice that this is on top of the conventional forward–looking, intertemporal utility effect, which is defined over outcomes (utility of health capital h_t, in this case). Concavity of $a(.)$ means information aversion: when $a(p) >$

[15] See, among others, George Loewenstein (1987), Andrew Caplin and John Leahy (2001), Andrew Caplin and Kfir Eliaz (2003), and Botond Kőszegi (2003; 2010).

$pa(1)+(1-p)a(0)$, for any $p \in (0,1)$, the person prefers to remain uncertain about her true health state rather than learning it. Figure 4 illustrates; p^* denotes the point at which the difference between $a(p)$ and $pa(1)+(1-p)a(0)$ reaches the maximum.

Figure 4. Anticipatory utility and information aversion.



The individual takes the initial health stock $h_0 = h$ as given, discounts the future by factor $\beta \in (0,1)$ and chooses a screening plan to maximize the expected discounted present value of lifetime utility subject to the dynamics of health capital (10) and to the endogenous death date τ. The Bellman equation is:

$$V(h_t) = \max_{Y_t}\{rh - cY_t + a(p_t) + \varepsilon(Y_t) + \beta\mathbb{E}V(h_{t+1})\},$$

where the expectation is taken with respect to the perceived probability of being sick, p_t. Notice that $\mathbb{E}V_{t+1}(0) = 0$. This simple binary choice problem is solved by comparing the payoff from not screening and screening at time t. These are, respectively:

$$\begin{aligned} V(h_t|Y_t &= 0) = rh + a(p_t) + \varepsilon(0) + \beta(p_t\mathbb{E}V_{t+1}(0) + (1-p_t)\mathbb{E}V_{t+1}(h)) \\ V(h_t|Y_t &= 1) = rh + (1-p_t)a(0) + p_t a(1) - c + \varepsilon(1) \\ &\quad +\beta(p_t(\pi\mathbb{E}V_{t+1}(h) + (1-\pi)\mathbb{E}V_{t+1}(0)) + (1-p_t)\mathbb{E}V_{t+1}(h)) \end{aligned}$$

The probability that the person screens at time t, given the perceived probability of having the disease and the cost of screening, is equal to the probability that $V(h_t|Y_t = 1) \geq V(h_t|Y_t = 0)$, or

$$\Pr(Y_t = 1|p_t, \pi, c, C_t) = F[\underbrace{\beta p_t \pi \mathbb{E} V_{t+1}(h) - c}_{\text{net expected benefit}} + \underbrace{(1 - p_t)a(0) + p_t a(1) - a(p_t)}_{\text{information aversion component}}], \tag{11}$$

where F denotes the probability function of $\varepsilon(0) - \varepsilon(1)$. This probability is affected by two main forces: the net expected benefit (the larger this is the higher the probability of screening) and information aversion (the less information averse the individual is—i.e., the closer to linear $a(.)$ is—the higher the probability of screening).

Equation (11) is the theoretical counterpart of the empirical probability in (1). If we assume that preferences (i.e., F, β, V, and a), technology (i.e., c and π) and own health (i.e., h) do not change when a colleague is diagnosed with breast cancer, then in the model the only way such event may alter screening behavior is by altering the perceived probability of being sick. That is, it must be that

$$p_t = p(\mathbf{T}_g^t), \tag{12}$$

where $\mathbf{T}_g^t = \{T_g^k\}_{k=0}^t$ is the history of such events in group g up to time t, as illustrated in Section 3.[16] Equation (12) offers a convenient way of thinking about salience effects. Roughly speaking, increased salience means that something is "more present" to an individual. From an operational viewpoint, in a model with uncertainty about that "something", this is equivalent to a higher probability of that "something" being actually there, thus inducing a behavioral response.[17] That is, we can think of p_t as being increasing in the treatment, in the following sense:

[16] Another possibility is that the model is misspecified, and that the treatment affects aspects of the decision problem different from the perceived probability of having the disease. For instance, suppose that women diagnosed with breast cancer used to screen regularly: the fact that despite a regular screening habit they were diagnosed with the disease may lead their colleagues to think that screening is not useful, and thus to screen less. In the model above this corresponds to a lower perceived π conditional on screening. However, this story is inconsistent with what we see in our data: women diagnosed with breast cancer had screening habits identical to those who were not.

[17] These "salience effects" may be important in many contexts. Recent experimental work in economics shows that this is the case even for prices: Raj Chetty, Adam Looney and Kory Kroft (2009) show that making a consumption tax more salient by posting tax-inclusive prices in a grocery store reduces purchases; Amy Finkelstein (2009) shows that

$$
\begin{aligned}
p(1, \mathbf{T}_g^{t-1}) &\geq p(0, \mathbf{T}_g^{t-1}), \\
p(T_g^t, 1, \mathbf{T}_g^{t-2}) &\geq p(T_g^t, 0, \mathbf{T}_g^{t-2}), \\
p(T_g^t, T_g^{t-1}, 1, \mathbf{T}_g^{t-3}) &\geq p(T_g^t, T_g^{t-1}, 0, \mathbf{T}_g^{t-3}), \\
... &\geq ...
\end{aligned} \tag{13}
$$

The available evidence from the medical literature is consistent with this set of inequalities. Montgomery *et al.* (2003) find that having a friend diagnosed with breast cancer increases the perceived risk of the disease. Similarly, Kathy Helzlsouer *et al.* (1994) find that experiencing cancer through family and friends is significantly associated with the perceived risk of cancer in general. Penelope Hopwood (2000) and both Evans *et al.* (1993) and Caryn Lerman *et al.* (1995) report that experience of breast cancer in the family leads to substantial overestimation of the probability of developing the disease, despite the fact that less than 5% of breast cancer is hereditary (Beth Newman *et al.*, 1988). Constance Drossaert *et al.* (2002) asked a group of women in the age range 50–69 to indicate to what extent the hypothetical situation of hearing that an acquaintance has got breast cancer would cause nervousness or anxiety: 10.8% said they would become 'very' nervous or anxious; 23.7% 'rather'; 38.9% 'a little'; only 26.6% reported 'not at all'.[18]

Such reactions are all examples of salience effects: experiencing breast cancer via social interactions or family interactions makes the disease more salient and induces a higher perceived probability of developing the disease.[19]

making a toll less salient by introducing an electronic toll collection system reduces the elasticity of driving with respect to the toll.

[18] We are grateful to Dr. Drossaert for sharing this unpublished tabulation with us.

[19] We can characterize in the same way the dependence of p_t on $\mathbf{T}_g^t$ by assuming that the learning process is Bayesian. Suppose a woman observes that a co-worker is diagnosed with breast cancer. Consider first the case in which this woman has the correct prior and knows the statistical process governing the disease. Then the new evidence is uninformative, and the posterior is equal to the prior. Suppose instead this woman has incorrect subjective prior probabilities of developing breast cancer. If the prior is downward biased relative to the objective probability then observing that a colleague is diagnosed with the disease leads to a higher posterior probability—the new piece of information goes against the prior. If, instead, the prior is upward biased then Bayesian updating cannot lead to a downward revision of the probability of having breast cancer—the new piece of information is confirming the prior. The opposite happens after observing the opposite signal. That is, that no one in the group was diagnosed with breast cancer in a certain period. In this case low priors remain low and high priors become smaller. Eventually this dynamics converges to the objective probability. Now consider the case in which given the prior

The ensuing behavioral response can be computed by differentiating (11) with respect to p_t and noticing that V_{t+1} is independent of p_t. A marginal increase in the perceived probability of being sick induces a change in the probability of screening equal to:

$$[\underbrace{\beta V_{t+1}(h)}_{\text{standard effect, } >0} + \underbrace{a(1) - a(0) - a'(p_t)}_{\text{anticipatory effect, } \lesseqgtr 0}]f(.), \tag{14}$$

where $f(.)$ is the density associated with F, evaluated at the baseline in (11). This equation makes clear that there are two effects at work. The first effect is standard: when the perceived probability of being sick increases, the probability of screening increases because a timely diagnosis has a positive value, and so information becomes more valuable. The second effect is anticipatory, and is of uncertain sign: it is negative for sufficiently low values of the perceived probability and positive otherwise. In Figure 4 the threshold is p^*.

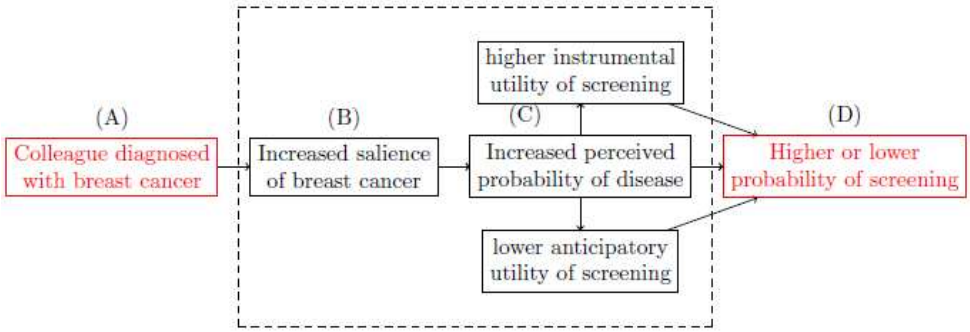
The causal chain implied by this model is illustrated in Figure 5. When a co-worker is diagnosed with breast cancer the salience of the disease increases via workplace social interactions: (A)→(B). Such increased salience leads to a higher perceived probability of developing the disease: (B)→(C). This may lead to a higher or lower propensity to screen, depending on whether the higher instrumental value of screening offsets the possible lower anticipatory value or not: (C)→(D).

Our empirical analysis identifies the (A)→(D) effect. The specific mechanisms in–between cannot be tested with our data. That is, we can neither identify the role of social interactions, nor the effect on salience, nor the effect on the perceived probability of developing breast cancer. However, we can look at independent "mechanism experiments" (in the jargon of Jens

a woman does not know the rate at which breast cancer occurs and believes that the binary signal (breast cancer emerges in the group or not) is generated by a random draw without replacement. In this case it follows from the "law of small numbers" (Tversky and Kahneman, 1971; Rabin, 2002) that a woman will over-infer from a small sample. In this case, too, observing breast cancer in the group will lead to a higher perceived probability of having the disease. Finally, consider the case (Rabin, 2002) in which a woman believes that the binary signal she observes is generated by a random draw without replacement (i.e., she believes in the "law of small numbers", as above) but *does* know the rate at which signals are drawn. Then she expects the next signal to be negatively correlated with the one just observed. In other words, a woman observing that a colleague was diagnosed with breast cancer in the group would think that it is unlikely that someone else in the group has the disease. This would lead to a lower perceived probability of having breast cancer, which is inconsistent with the evidence from the medical literature.

Ludwig, Jeffrey Kling, and Sendhil Mullainathan, 2011) to support the assumptions of our model. First, the aforementioned research by Constance Drossaert *et al.* (2002) provides evidence in favor of the (A)→(B) step. Evidence in favor of an (A)→(C) effect is provided by the four papers mentioned above: Evans *et al.*, (1993), Helzlsouer *et al.* (1994), Hopwood (2000), and Montgomery *et al.* (2003). These papers suggest that experiencing breast cancer through a person that is close in the social space increases the perceived probability of developing the disease. Finally, the (B)→(D) effect is identified by a controlled experiment performed by Jamie Arndt *et al.* (2007). These authors' experiment effectively replicates in a laboratory a static version of the dynamic natural experiment we study. Specifically, they increase the salience of breast cancer by inducing a group of treated (young) women to think hard about the disease through a questionnaire on "life attitude", and find that the treatment causes a significant drop in the intention to conduct breast exams in the future. They interpret such avoidance intention as a defence in front of the activation of death-related thoughts. Given that, empirically, the (A)→(D) effect is negative while the (A)→(C) effect is positive, a model with anticipatory feelings and information aversion is a parsimonious way of rationalizing the evidence we have produced in this paper.

Figure 4. Causal chain.



Our results—notably the finding that the impact effect is large and then vanishes in time as the distressing event becomes more and more distant—are also consistent with anecdotal evidence of short-run emotional reaction ("fear of cancer") described by women who experienced breast cancer through colleagues or family members. For instance, Sara Austin offers on

Women's Health on msnbc.com, a touching account of her temporary fear of screening (because of the fear that the disease could materialize) after her mother died of breast cancer. The following passage is particularly revealing: "It has taken me nearly two years from the time my doctor recommended a mammogram for me to actually get one. I delayed making an appointment for more than a year, the prescription sitting in a stack of junk mail. Now my reprieve is over [...]. I am well insured, well informed [...] and well aware of the lifesaving difference early detection can make. The only thing standing in my way has been a mix of scary emotions".[20]

6 Conclusions

In this paper we have exploited a natural experiment occurring repeatedly in time in a unique panel data set of employees at a large medical organization in the United States to study the reaction of women in the age range 50 and older to the event that a colleague is diagnosed with breast cancer. It is plausible that such events soon become known—through social interactions—to co-workers physically close in the workplace. Presumably, such knowledge makes breast cancer more salient and may lead to changes in the propensity to screen. We have estimated a dynamic treatment effect model and we have found a negative contemporaneous effect. This effect has persistence and slowly decays over time. We have found important heterogeneity behind this average treatment effect. Our results are consistent with theories of information aversion. Our large-scale dynamic natural experiment confirms the findings of smaller laboratory experiments in which salience of breast cancer is artificially manipulated, and is consistent with a large medical and economic literature on risk perception and noncognitive traits. We are well aware of the fact that the external validity of our study is limited: women in our sample work in the heath industry, have full health coverage, and most of them are well educated. Despite this limitation, our results point to a potentially important and overlooked source of screening avoidance, and suggest that (*i*) information campaigns that make a noninfectious disease so salient as to induce death-related thoughts may have effects that are the opposite of the intended ones—i.e., they may induce some women to screen *less*; (*ii*) the apparent under-use of mammographies in the United States[21]

[20] "When cancer-screening fears are all too real. 1 in 5 women does not want to know if she has the disease, survey shows." January 19, 2009

[21] This question receives recurrent attention by the media. See, for instance "Mammograms in Decline", *The New York Times*, May 15, 2007.

may be partly due to fear of being diagnosed with breast cancer; in this case, as suggested by Andrew Caplin and Kfir Eliaz (2003) for AIDS, it may be optimal to decrease the informativeness (to patients) of screening tests when the test is positive, because this would mitigate the fear of bad news and thus increase the propensity to screen; (*iii*) in order to avoid the rate of regular clinical breast screening to fall below the socially efficient level, one should pay particular attention to women in social environments in which someone was diagnosed with breast cancer; (*iv*) the effect of information aversion in health matters may be more relevant when salience is stronger and may be stronger for relatively young and healthy individuals. More knowledgeable individuals (such as medical doctors and nurses in our study) seem less subject to such an effect. For other individuals, the effect is highly persistent. We believe these issues are important and deserve further investigation in future research.

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