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The Effect of Tort Reform on Cancer Treatment and Patient Outcomes: Evidence from the US during the Late 20th Century

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I. INTRODUCTION

The Congressional Budget Office (2011) estimates that total expenditures on health care in the United States will rise from 17 percent of GDP in 2009 to nearly 50 percent of GDP by 2082. By itself, this increase might not be cause for concern assuming Americans are better off as a result. However, research has consistently shown that this is not the case. Many countries spend far less than the US on health care, yet they have significantly better health outcomes (WHO, 2000). Accordingly, reducing wasteful health care spending is not only desirable but also—to the extent that the current trajectory is unsustainable—necessary.

Many Americans, including two US Presidents, attribute much of the blame for spiraling health care costs on an increasingly dysfunctional medical malpractice liability system.[1] In an ideal world, tort law—the legal underpinning of medical malpractice—ought to provide a mixture of incentives and deterrents to doctors such that they keep medical errors at a safe, yet non-zero lower bound. Today's system, however, drives doctors to keep medical error rates at impossibly low levels by keeping the threat of punishment for a medical error exceedingly high. In response to this elevated liability risk, many doctors practice "defensive medicine." That is, they carry out unnecessary scans and procedures in order to reduce their own legal liability. While seemingly innocuous, the practice of defensive medicine costs Americans an astronomical $55.6 billion per year, i.e., roughly 2.4% of total annual health care spending in the US (NPR, 2010).

[1] During a 2005 speech, then President George W. Bush claimed the US was in the middle of "a medical liability crisis" (PBS, 2005). His subsequent description of the crisis was—and continues to be—the prevailing story, transcendent of party lines, as evidenced by President Obama's remarkably similar address to Congress in September 2009 (White House, 2009).

With such potential savings, it may come as little surprise that tort reform—broadly defined as any change in the tort law which results in reduced tort litigation or paid damages—has been so successful in recent years. Indeed, during the past three decades, over thirty states have passed some sort of tort reform (Hyman & Silver, 2006). Since then, various measures of physician liability have fallen, including average malpractice claims payouts and medical liability insurance premiums (Mello, 2006). But, is there any evidence to suggest that doctors have stopped practicing defensive medicine?

In truth, the answer to this final question remains quite illusive and, for all of its possible policy ramifications, remarkably understudied. Indeed, of the multiplicity of papers addressing tort reform, only two directly address its effect on defensive medicine using an empirically rigorous approach. In doing so, however, the authors find conflicting results. Whereas Kessler and McClellan (1996) find evidence that tort reform reduces defensive medicine, Currie and MacLeod (2008) find evidence that certain kinds of tort reform only exacerbate it.

We confirm that the Currie and MacLeod theory is more broadly applicable to cancer—a significant finding given how fundamental cancer is to the US health care system. Indeed, not only is cancer the second leading cause of death among Americans, but its treatment costs are also expected to climb to an exorbitant $158 billion by 2020 (Mariotto et. al., 2011). And yet, with ever increasing numbers of "delay-in or failure-to-diagnose" malpractice claims, cancer is acutely susceptible to defensive medicine. Studdert et. al. (2005) find that 24% of high-risk specialty doctors report detection of cancer as the chief impetus for their overuse of diagnostic imaging, specialist referrals, and invasive procedures.

Our work examines the effect of damage caps and other direct tort reforms on cancer-specific defensive medicine.[2] We adopt a series of state tort reforms, which occurred between 1980 and 2000, as natural experiments and compile state-level data from two sources, the Behavioral Risk Factor Surveillance System (BRFSS) and the Surveillance Epidemiology and End Results (SEER) Program, to capture information on the use of screening tests and therapeutic procedures as well as health outcomes. Then, we use a differences-in-differences strategy, accounting for both national trends and pre-existing differences among states, to estimate the effect of tort reform on cancer treatment intensity and cancer patient health outcomes.

In theory, if tort reform succeeds in reducing or eliminating defensive medicine, then our estimates should reflect that doctors in states with capped damage awards are cutting back on cancer treatment intensity at no health cost to patients. Since the liability risk associated with *not* carrying out the marginal test or procedure is lower, we should find that—all else equal—the number of tests and procedures in states with tort reform is falling relative to the number in states without. Moreover, since treatments motivated by defensive medicine have little to no intrinsic medical benefit, we should find that—all else equal—cancer patient outcomes are not changing in states with tort reform in relation to those without tort reform.

Our results are largely inconsistent with this model. We find that damage caps and other direct reforms have intensifying effects on test and procedure use. That is, doctors use *more* scans and procedures post-reform. Additionally, we find that cancer patients in states with reform have significantly *worse* health outcomes post-reform, e.g., their tumors are diagnosed at later stages, their survival time post-diagnosis falls, etc. Estimating models separately by age

[2] As defined by Kessler and McClellan (1996), direct tort reforms include any which, “truncate the upper tail of the distribution of awards, such as caps on damages and the abolition of punitive damages, and reforms that shift down the mean of the distribution, such as collateral-source rule reform and the abolition of mandatory pre-judgment interest.”

block and health insurance status, we show that these treatment effects intensify with greater physician discretion. For example, we show that tort reform has a smaller effect on mammography use among women above age forty because they are subject to more rigid screening guidelines. Finally, we make several modifications to our specification to test the robustness of our principal results and show that, with the exception of procedure use, they do not change our estimates substantially.

The paper proceeds as follows. First, we provide background information on the most recent series of tort reforms in the US and offer our argument as to why cancer serves as a good case study. Next, we review the literature, albeit sparse, which examines the effect of tort reform on treatment and health outcomes in other contexts. Then, once we describe our data and research strategy, we present our empirical results, threats to validity, and robustness checks. We conclude with a brief policy-oriented discussion of our findings and suggest future expansions of this research.

II. Background

A. What is tort reform?

As defined by Garner (1999), all tort reforms are not created equal. In other words, they can take on many forms ranging from caps on damages to mandates on periodic payments of compensatory awards. We focus on two aspects of tort law most frequently targeted by reform: the awarding of damages (both non-economic and punitive) and the collateral source rule.

Non-economic damages refer to compensatory awards to victims for pain and suffering, disfigurement, and/or loss of consortium.[3] Due to the nature of these losses, the size of non-economic damage awards varies massively, depending in large part upon the composition of the jury. In the courtroom, jurors are instructed only "to be fair and reasonable and to use their enlightened conscience to make the plaintiff feel whole" (Vidmar & Rice, 1993). Therefore, although awards typically increase with the severity of the injury, they are very inconsistent overall (Studdert *et. al.*, 2011). Juries may and often do award different damages to two different patients whose doctors have made the same negligent error.

Punitive damages, in contrast, are awarded only when there is a "preponderance of evidence that the defendant [the doctor] *deliberately* failed to avoid unnecessary injury" (Cohen, 2003). Although smaller in size and far less prevalent than non-economic damage awards, punitive damage awards often pose a more serious threat to the economic viability of doctors since many states forbid medical liability insurers from covering them (Hubbard, 1989). Moreover, punitive damage awards are frequently given not to the victim but to a third-party victims' compensation fund (NCPA, 2007). As such, they serve a purely punitive role, compounding any punishment delivered by state licensing committees or, in particularly flagrant cases, criminal courts (Rustad & Koenig, 1995).

The collateral source rule allows victims to recover damages from a doctor even if they may also receive them from elsewhere (Cohen, 2003). For example, if a patient suffers $50,000 worth of injuries as a result of a negligent physician and his or her insurance covers only $48,000, then

[3] Loss of consortium refers to, "reciprocal rights inherent in the marital relationship of husband and wife" (Garner, 1999).

he or she can ask the court for the full $50,000. Therefore, unless the victim has signed a subrogation agreement in advance, he or she can effectively receive double damages.[4]

By capping damages and reforming the collateral source rule, states are not trying to undercompensate victims but rather to decrease doctors' liability (Avraham et. al., 2009).[5] With respect to the latter, they have been very successful. Holtz-Eakin (2004) finds that damage caps have consistently decreased the size and distribution of awards, and Avraham et. al. (2007) show that direct reforms have led to significant reductions in the number of malpractice claims filed, i.e., roughly 5-13 percent. As these reductions carried over into the liability insurance market, doctors' insurance premiums also have fallen (Studdert *et. al.*, 2011). Aggregating these effects and controlling for as much observable variation as possible, one recent study published in the *Journal of Law, Medicine, and Ethics* estimates that doctors working in hospitals located in states with tort reform pay, on average, $1,300 less in malpractice costs per bed: a statistically significant and economically meaningful difference (Ellington *et. al.*, 2010).[6]

Despite this well-established effect on liability, tort reform still may not curb defensive medicine. To the extent that a doctor's insurance pays for the marginal award dollar, tort reforms that only affect the size of damage awards may result in no behavior change whatsoever. There, capping damages becomes an ineffective and dangerous way to lower malpractice costs because it does not "fix the problem" but rather mechanically pulls down awards, perhaps at the cost of injured patients' financial well-being (Nelson *et. al.*, 2007). In short, direct reform will only

[4] If the insurance company (or collateral source) has a right of subrogation against the victim, then—in our example—the victim must take $48,000 from his or her award and repay the insurance company (Cohen, 2003).
[5] Collateral source rule reform prohibits victims from receiving double damages. In our example, this would imply that the patient could only request $2,000 compensation from the court.
[6] To generate their "per bed" estimates, the authors add together all medical malpractice costs incurred by the doctors working in a hospital and then divide the resulting total by the number of beds in the hospital.

significantly decrease malpractice costs if it brings down awards *and* realigns the risk of legal liability for physicians. Only then will it deter the practice of defensive medicine.

In theory, if direct reforms are sufficiently targeted to lower physician liability, then there are two testable predictions. First, since the malpractice risk associated with *not* ordering an extra test or performing an extra procedure is lower, physicians ought to opt for less intensive treatments on the margin. Second, since medical interventions associated with defensive medicine have little to no medical merit, *not* doing the extra test or procedure will leave patients unaffected or perhaps leave them even healthier. In sum, not only are the direct costs associated with the marginal treatment and any resulting complication saved, but also patients are spared the health, opportunity, and psychic costs of having to go to the hospital to get unnecessary procedures.

Does evidence support this theory? Surprisingly, there is not yet a clear answer. As of today, researchers have only examined the effect of tort reform on two medical specialties: obstetrics (Currie & MacLeod, 2006) and cardiology (Kessler & McClellan, 1996), described below in the literature review. Our study expands the economics research on the impact of tort reform to the medical specialty of oncology.

B. Why cancer?

The treatment of cancer is central to the US health care system. Not only is it the second leading cause of death among Americans, accounting for nearly one out of every four deaths, but also its prevalence is far greater than even those two statistics suggest. As of January 2007, 11.7 million Americans were alive who had been diagnosed with the disease. Given the lifetime risk of cancer for men and women is 1:2 and 1:3, respectively, and some 1.6 million new cases are diagnosed each year, the number of Americans affected by cancer will only increase in the near future, especially with the aging of the US population (ACS, 2011).

Additionally, cancer is very expensive to diagnose, to treat, and to monitor. According to recent estimates, total direct expenditures for cancer were $103 billion in 2010, but researchers predict that expenditures will climb to at least $158 billion in the next eight years, excluding any indirect expenditure related to productivity losses and opportunity costs (Mariotto *et. al.*, 2011). Consequently, to the extent that cancer treatment is and will continue to be a major component of the US health care system costs moving forward, it is crucial that we fully understand the effects any proposed cost-cutting policy would have on it.

The treatment of cancer in and of itself provides an ideal setting for defensive medicine. Frequent medical scans and tests are crucial, not only for diagnosis but also for treatment and monitoring purposes. Everyone—at some point in his or her lifetime—will undergo a cancer screening, be it a mammogram or a pap smear or a rectal exam. Patients diagnosed with cancer may undergo hundreds of scans. Moreover, cancer, perhaps more than other specialties, offers physicians a great deal of discretion in determining a course of treatment given that no two cancers are identical. Whereas Patient A may receive two months of chemotherapy for her breast cancer, Patient B's doctor may instead decide to remove her tumor surgically.

Beyond all of that, however, cancer is a particularly interesting case study because—*a priori*—defensive medicine may be a *good* thing for patients. Typically, when people criticize defensive medicine, they refer to the patient who walked into the emergency room with a headache and walked out, hours later, having undergone a CT scan confirming that he or she had a headache. With cancer, however, not only would extra scans on marginal patients almost certainly result in earlier diagnoses of tumors but also extra surgeries and radiation would almost certainly result in extensions of survival time. In such a world, reducing defensive medicine would lead to *more* cancer deaths. This is a result that seriously questions the virtue of any tort reform, health care cost savings there may be.

Accordingly, if, at the end of the day, our primary goal is to determine whether tort reform affects defensive medicine and it is exceedingly difficult to identify defensive medicine on a case-by-case basis, then we must focus on medical conditions with enough scope for tort reform to have an effect. If our secondary goal is to evaluate tort reform as a policy tool, then it seems most compelling to look at prevalent yet expensive medical conditions. Cancer fulfills both these criteria.

III. Literature Review

As shown in Appendix 1, prior to 2000, twenty-five states enacted one of the aforementioned varieties of direct tort reform (Currie & MacLeod, 2008). Usually, this involved turning a law "on," or enacting new tort reform legislation; however, there were a few instances in which a law was turned "off," or ruled unconstitutional. Only eight out of the twenty-five states targeted medical malpractice, i.e., most intended only to update general tort statutes.

Kessler and McClellan's (1996) seminal study on defensive medicine used this series of reform as a multi-year natural experiment. Focusing on elderly Medicare beneficiaries who had been receiving treatment for serious heart conditions, the authors used the direct and indirect tort reform in the 1970s and 1980s as an identifiable source of variation in physician liability pressure for their differences-in-differences empirical strategy. In accordance with the two-pronged mechanism underlying defensive medicine, they examined the effect of tort reform not only on hospital expenditures—they had access to Medicare claims panel data—but also on patient outcomes, taking into account patient demographic characteristics, state legal and political characteristics, as well as state, time, and state-time fixed effects.

Their findings were not only significant but also robust. Although neither direct nor indirect reforms seemed to affect patient outcomes, their estimates suggested that direct reforms to tort law cut hospital expenditures by five to nine percent, representing a savings of nearly $450 million per year in 1996.[7] Indeed, their findings strictly accord with the underlying theory of tort reform. That is, once the risk of legal liability declined, physicians performed fewer tests and procedures, patients' health outcomes were unaffected, and costs fell.

Twelve years later, Currie and MacLeod (2008) expanded the analysis of the impact of tort reform to obstetrics using the reforms of the 1980s and 1990s as their source of exogenous variation. Instead of classifying reforms as direct and indirect, the authors treated the four most prevalent reforms—caps on non-economic damages, caps on punitive damages, collateral source rule reform, and reform of the joint and several liability rule—separately such that they could parse out the full effect of each. Then, using birth data from the National Vital Statistics System (NVSS) and standard panel data methods, they estimated the effect of each type of tort reform on procedure use and the health outcomes of mothers and their babies, controlling for personal characteristics, year fixed effects, state-time fixed effects, and county fixed effects.

Their findings were equally as significant and robust as those of Kessler and McClellan (1996) but had vastly different implications for the impact of tort reform. Specifically, while they found that joint-and-several liability did reduce C-sections and complications, driving down costs and improving patient health outcomes, caps on non-economic and punitive damages did just the opposite. Specifically, as caps fell, physicians performed *more* procedures, causing

[7] This is equivalent to roughly $658 million in 2012 dollars.

worse patient outcomes and *higher* overall costs. Although inconsistent with tort reform theory, Currie and MacLeod posit that their second result is easily explained if two assumptions on physician conduct are made.

They first assume that we are in a world where tort reform lowers physician liability in the event of an error. From there, they posit that the error rate will always increase with tort reform. Consider it a substitution effect. With tort reform, the cost of making a mistake falls, so physicians—perhaps unwittingly—make more mistakes. Then, they assume that every physician has a point at which they are indifferent between doing a procedure and not doing a procedure, doing a test and not doing a test, etc. This condition is a function of two separate variables: current liability law and their patient's condition. While more straightforward patients will undergo the procedure, the more complicated patients will not.

With these assumptions in place, parsing out the effect of capping damages is straightforward. A damage cap will decrease liability and lower the indifference condition as long as the risk of making a medical error is greater upon going forward with the procedure. Simply put, more patients fall into the "straightforward" category where it is utility maximizing for the physician to perform the marginal procedure (or test). If it is riskier *not* to do the procedure, a damage cap will instead raise the indifference condition and lead to fewer procedures overall.

The next section explains the data we will use to test the applicability of these predictions to the context of cancer care.

IV. DATA

Prevailing theory suggests that tort reform may affect not only physician choice of screening tests and medical procedures but also patient health outcomes. We use two separate data sources for our analysis to test for an effect of tort reform on treatment intensity and health separately.

In our study, data on the prevalence and the frequency of medical testing come from the Behavioral Risk Factor Surveillance System (BRFSS). Created in 1984, the BRFSS is a nationwide telephone health survey administered yearly by the Centers for Disease Control (CDC) to capture trends in risk behaviors associated with the leading causes of death in the US. The unit of observation is an individual within a given state at a particular month (and year). Since the majority of the tort reforms we use in our analysis occur in the late 1980s through 1990s, we restrict our sample to a thirteen-year period, January 1987 to December 2000 such that the final data set includes 1,484,740 observations.

Our chosen subset of the BRFSS includes a ranked indicator of general health as well as information on four medical screening tests—mammograms, pap smears, rectal, and proctoscopic exams.[8] As shown in Table 1, respondents provide information not only on their lifetime screening history but also on their screening use over the past year. The means listed in the first four rows of Table 1 (the "have you ever had" series), where rows 1 and 2 exclude men, show the commonplace nature of each of these scans. That is, a large fraction of the population reports having had at least one of these screening exams in their lifetime. Even so, if tort reform succeeds in decreasing defensive medicine, past-year scanning use should nonetheless fall in states *with* reform relative to those *without* it.

[8] From 1993 onwards, respondents were asked to rank their health on a five-point scale—from excellent (1) to poor (5). We have converted the variable into a "good health" dummy which equals 1 if the respondent reported having good, very good, or excellent health.

It is important to keep in mind, however, that these data may not be entirely representative of medical testing practices throughout the 1987-2000 period. During the CDC's initial implementation of the BRFSS, a number of the medical testing variables dropped in and out of the survey. Of greatest consequence, the data on rectal and proctoscopic exams span only 1988 to 1996 and 1988 to 1995, respectively. Furthermore, as may be expected in a health survey, many respondents—nearly 5% on average—refused to answer or could not recall their medical testing history.[9] Therefore, the effective number of observations varies significantly depending upon the choice of dependent variable. To the extent that there is overlap between when the scanning variable is missing and when the bulk of the variation in tort reform occurs, it limits our ability to use this methodology to examine the effect of tort reform on the use of that particular scan.

The BRFSS also includes a host of demographic controls and proxies for health insurance status. For the purposes of our analysis, we focus on six—age, sex, race or ethnicity, marital status, educational attainment, and current health insurance status—and control for the first five in all of our models. As illustrated in Table 1, dummy variables are used in place of many of the original categorical variables using the convention wherein its value equals 1 if the variable is true and 0 otherwise. Taken altogether, the summary statistics suggest that the typical observation within our sample is a middle-aged, married white female in good health with some form of health insurance and at least a high school diploma.[10]

[9] Both types of non-responses are classified as missing in the subsequent analysis.

[10] Throughout our analysis, we use the CDC final sampling weights (*_finalwt*) to mitigate the BRFSS's inherent non-coverage error. Previous research has shown that—with these weights—the BRFSS data is generally representative and provides comparable information to other national health-oriented surveys including the National Health Interview Survey (NHIS). See Nelson et. al. (2001), Nelson et. al. (2003), and Arday et. al. (1997). Accordingly, our

Data on the remaining pathway of tort reform's presumed effect—procedure choice and cancer patient outcomes—come from the Surveillance Epidemiology and End Results (SEER) Program. Organized by the National Cancer Institute, the SEER program is effectively a multi-state tumor registry covering thirteen distinct regions of the US, i.e., approximately 28% of the US population.[11] As shown in Table 2, the registry draws the bulk of its cases from hospital inpatient records but—as will prove critical later on—it also collects information from autopsies and death certificates, e.g., tumors which went undiagnosed until death.

For our analysis, we use the so-called SEER 13 database, which incorporates all tumors—both malignant and in situ—diagnosed between January 1973 and December 2008. The unit of observation, therefore, is not an individual but rather a tumor diagnosed within a given state and at a particular month (and year). As was the case with the BRFSS, due to the availability of tort law reforms, we again restrict our sample to only those tumors diagnosed between January 1983 and December 2000. In total, then, the final sample includes 2,514,489 tumors representing 1,776,803 cancer patients.[12]

The data provide comprehensive information on each tumor—from its physical characteristics (position, cellular composition, size, etc.) to any prescribed first course of treatment. Given that tort reform ought not have any effect on the physiology of a tumor but rather its probability of being diagnosed and perhaps the intensity of any prescribed treatment regimen post-diagnosis, we focus on three treatment intensity indicators: incidence of surgical

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methodology will yield externally valid estimates as long as there are no systematic differences across groups of doctors in how they respond to tort reform.

[11] These regions include Atlanta, Connecticut, Detroit, Hawaii, Iowa, New Mexico, San Francisco-Oakland, Seattle-Puget Sound, Utah, Los Angeles, San Jose-Monterey, Rural Georgia, and Alaska.

[12] Over 700,000 cancer patients appear in the SEER 13 database multiple times (up to 16) upon diagnosis of additional tumors.

intervention, incidence of radiation therapy, and stage upon diagnosis. Recoded into dummy variables, the former two simply indicate whether a patient has had surgery or radiation therapy as part of his or her first course of treatment. The staging variable, in contrast, collapses assorted information on the extent of the disease at diagnosis into a single ordinal variable, which takes on values of 0 (*in situ*), 1 (*localized*), 2 (*regional),* and 4 (*distant*).[13] Again, if tort reform reduces defensive medicine, then a positive "shift" in stage at diagnosis should occur. That is, because doctors are less worried about their own liability, they should carry out fewer diagnostic scans and—as a result—catch fewer tumors at stage zero and more tumors at stage four. We recode the staging variable into two dummies, stage 0 and stage 4, to test this hypothesis directly.

The SEER database also contains two measures of patient health outcomes. The first, survival time, measures the time (in years) from the initial diagnosis of cancer to death or to the registry's cutoff date of December 2008, whichever comes first.[14] The other is a dummy variable that indicates whether or not a cancer patient died as a direct result of his or her tumor.[15] To the extent that these two measures are complementary—that is, living longer is associated with a decreased probability of dying of cancer, we expect to find oppositely signed effects. More specifically, if tort reform succeeds in reducing defensive medicine, then cancer patients should live for *more* time post-diagnosis and be *less* likely to die.

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[13] Within the SEER program's data dictionary, this variable is named SEER historical stage A. As defined by SEER, a tumor which is (i) *in situ* refers to a noninvasive tumor which has not penetrated the basement membrane nor extended beyond the epithelial tissue, (ii) *localized* refers to an invasive tumor which is confined entirely to the organ of origin, (iii) *regional* refers to a tumor which has extended beyond the limits of the organ of origin and into surrounding organs, and (iv) *distant* refers to a tumor that has spread to parts of the body remote from the primary tumor.

[14] By default, tumors added to the registry from death certificates and autopsies have patient survival times of 0 years. Given this is not an accurate measure of these patients' true survival time, we have recoded all such cases to missing.

[15] Here, the omitted category includes both cancer patients who are still alive and cancer patients who died of a cause unrelated to cancer, e.g., a car accident.

Despite the rich tumor-specific data provided by the SEER program, the database lacks much of the information on individual characteristics of BRFSS. Although we have and therefore can control for a patient's race/ethnicity, sex, and age at diagnosis, we do not have access to any measures of educational attainment, marital status, or health insurance status. Therefore, if—in running our regressions with the BRFSS data—we find that these characteristics vary systematically across states with and without tort reform, then our estimates of the effect of tort reform on cancer-specific outcomes may only reflect these same underlying differences within the sample. This brings us to a discussion of our empirical strategy.

V. METHODS

Our empirical strategy exploits variation in the timing of states' reform of their tort laws to determine the effect of these laws on cancer treatment and cancer patient outcomes. In particular, we focus our attention on the effect of lowering damage caps and other so-called direct reforms.

The bulk of our analysis relies upon a standard differences-in-differences approach. The base model is specified as follows:

$$(1) \qquad OUTCOME_{ist} = a + b_1 TORT_{st} + b_2 XVAR_i + \gamma_s + \delta_t + e_{ist},$$

where OUTCOME represents a screening test or procedure or health outcome; TORT is a dummy which indicates whether a tort reform is "on," or active; XVAR is a vector of individual characteristics; γ and δ refer to state and year fixed effects, respectively; and e is a random error term.

As exemplified by the work of Curie and MacLeod (2006), many specifications of TORT are possible. Given the motivation of our study, we consider only two. We begin by estimating the aforementioned model by allowing TORT to equal 1 only if there is reform of a damage cap, and then we re-estimate the model allowing for the existence of *any* sort of direct reform.[16, 17] In both instances, we define TORT at an annual level, e.g., if Arkansas lowered its cap on punitive damages in August 1997, then—if the individual is a resident of Arkansas—TORT equals 1 on or after 1997 and 0 beforehand.[18]

The coefficient b_1 on TORT may be positive or negative. With a screening test or procedure outcome as the dependent variable, a negative coefficient would suggest that tort reform is, in fact, reducing defensive medicine: doctors are carrying out fewer tests and procedures. A positive coefficient, in contrast, would suggest the opposite. That is, it would suggest that doctors are responding to tort reform by further increasing treatment intensity—a result indicative of an excessive procedure rate pre-reform in the theory developed by Currie and MacLeod (2006). The same dichotomous reasoning applies to a patient outcome as the dependent variable. Specifically, a "positive" (or zero) coefficient would imply that patients are doing just as well off post reform in accordance with the traditional defensive medicine theory while a "negative" coefficient would imply that patients are doing worse off post reform, perhaps as a result of the excessive treatment effect documented by Currie and MacLeod (2006).[19]

[16] Here, we again refer to the classification of direct reform by Kessler and McClellan (1996).
[17] As discussed in the background section, the tort reforms we include in our analysis are not homogenous; therefore, our current specification captures the effect of an "average" tort reform.
[18] We also repeat our analysis by defining TORT at a monthly level and find no significant difference in our results.
[19] Here, we use "positive" and "negative" figuratively to represent positive and negative health outcomes, respectively. Therefore, a positive coefficient on "death by cancer" would have a negative sign, and a positive coefficient on "survival time" would have positive sign.

The vector XVAR controls for individual characteristics.[20] These controls include AGE and AGE^2 to account for a non-linear age-effect as well as dummy variables for race (BLACK, HISPANIC, and OTHERRACE), educational attainment (HIGH SCHOOL, SOMECOLLEGE, COLLEGEPLUS), and marital status (MARRIED). In regressions not limited to females (i.e., those without mammogram or pap smear as a dependent variable), a dummy variable for sex (FEMALE) is also included.[21]

Fixed effects, in contrast, control for national trends and pre-existing differences between states. Our year fixed effects allow there to be state invariant differences in treatment protocols and health statuses across years. For example, they will control for the rapid increase in screening mammography for breast cancer that began in the mid-1980s (Ernster *et. al.*, 1996). Our state fixed effects allow there to be time invariant differences in treatment protocols and health statuses across states. They will, among other things, control for the fact that doctors in Texas provide more intensive care to their Medicare patients than do doctors in Oregon (Skinner & Wennberg, 2000).

Together, these three sets of controls—individual, state, and time—will provide us with causal estimates of the effect of tort reform as long as there are no time variant yet unobservable differences across states. In other words, even if elderly individuals en masse were moving from states without tort reform into states with tort reform and were dragging down average health outcomes in reform states in the process, our estimates of the effect of tort reform on patient outcomes would still be valid because we control for age. However, if doctors in Texas began to

[20] Including these controls should, among other things, increase the precision of our estimates because of their importance in affecting health behaviors and outcomes. See Ippolito (2003) and Cheng (2009).

[21] As noted in the data section, the vector of individual controls is more limited in regressions run with SEER data.

increase their treatment intensity even further relative to doctors in Oregon causing legislators in Texas to enact tort reform, then our estimates would be biased.

This type of policy endogeneity bias is an acute concern for the validity of our results given the well-documented pressure that was on state legislators to put an end to the medical liability crisis of the 1980s (Thorpe, 2004). The endogeneity can work in two directions. If, as in our Texas example, states with reform were on an increasingly higher trajectory of treatment intensity (or outcomes) relative to states without reform, then our estimate of the effect of tort reform might suggest that it increased treatment intensity even if it actually decreased it. On the other hand, if states with reform were on an increasingly lower trajectory of treatment intensity, and policymakers in that state passed tort reform in hopes of increasing procedure use, our estimates of the effect of tort reform would suggest that it decreased treatment intensity even if it actually increased it.

We test for this type of policy endogeneity bias by running regressions re-estimating (1) but allowing for one and two year leads and lags of TORT.[22] Given that doctors have no incentive to change their behavior before a state enacts a new tort law, both lead coefficients should be close to zero if states with and without tort reform were on the same trajectory in the two years prior to reform. If we find something different, then we have reason to suspect that policy endogeneity may be present—a finding that will temper the interpretation of any results.

As a more general test of parallel trends in health across states, we also run two placebo regressions, re-estimating (1) but replacing the dependent variable with two different proxies for health: height and seatbelt use. Since states' decisions to enact tort reform should not have had any direct effect on people's heights or their use of seatbelts, the coefficient on TORT in both

[22] We also repeat our analysis combining the one and two year leads and lags into a single pre and post lead and lag term and find no significant difference in any of the results.

regressions should equal zero as long as there are no differential state time trends that are correlated with our health proxies. The validity of our results only becomes questionable if we find a statistically significant coefficient on TORT. For example, it is possible that people living in states with imminent reform were becoming increasingly health risk averse such that they were not only wearing their seatbelts more frequently but also forcing their doctors into carrying out additional diagnostic scans. In such a scenario, our estimate of the effect of tort reform on treatment intensity would be spuriously positive.

We use a different empirical strategy—the Cox Proportional-Hazards model—to re-estimate the effect of tort reform on cancer patient survival time post-diagnosis as a final robustness check. The base model is specified as follows:

(2) $$h_i\ (t) = h_0\ (t) \exp\ (b_1 TORT_{st} + b_2 XVAR_i + REFORM_s + \delta_t + e_{ist})$$

where TORT is a dummy which indicates whether a tort reform is “on,” or active; XVAR is a vector of individual characteristics; REFORM is a dummy which equals 1 if the state *ever* had tort reform; δ refers to year fixed effects; and e is a random error term. The individual characteristics and year fixed effects serve the same purpose as before in our differences-in-differences strategy. Here, REFORM provides a more inflexible state fixed effect insomuch as it accounts for unobservable time invariant differences between states with and without tort reform.

We will use the Hazard model to estimate the probability that a cancer patient dies at time *t* conditional on being alive at time *t-1*. Unlike our linear probability model, the Hazard model does not assume a linear (or any other parametric) relationship between time at risk and dying of cancer. Moreover, it allows for (right) censoring whereas our ordinary least squares model does not.

Our survival data from SEER is very likely subject to both right and left censoring. Given that the SEER 13 database only includes information up to December 2008, we do not know the survival outcomes of patients who were alive at that cutoff. This, by definition, is right censoring. Left censoring, in contrast, involves us not knowing when cancer patients first developed their tumors, i.e., when they first became at risk of dying of cancer. Using date of diagnosis as a proxy for this time, while convenient for our differences-in-differences specification, is inappropriate since tort reform—through its effect on scanning prevalence—may systematically affect when tumors are diagnosed and thereby bias our coefficients towards finding a "positive" or "negative" effect of tort reform on survival when there may not be any.[23] As such, within our specification, we assume that all patients—regardless of whether they were in a state with tort reform or not—first became at risk of dying of cancer in January 1983 (the date of diagnosis of the first observation in the registry).[24]

As before, if tort reform reduces defensive medicine, then we would expect to find that it has no negative effect on health outcomes. In terms of the Hazard, this would entail a coefficient on b_1 that is less than or equal to one. A coefficient greater than one would suggest that tort reform is making patients worse off—a finding consistent with the Currie and MacLeod (2006) theory. With these considerations in mind, we now turn to our results.

[23] See Footnote *9*.

[24] Although an imperfect solution to our left censoring problem, this assumption should not affect the validity of our estimates as long as people in states with tort reform are not disproportionally represented within the early stages of the registry. An inspection of the data suggests this is not the case.

VI. EMPIRICAL RESULTS

A. Determinants of medical screening frequency

Tables 3a and 3b present the results of a series of regressions with lifetime testing variables for mammograms, pap smears, digital rectal exams, and proctoscopic exams as their dependent variables. Regressions with mammograms and pap smears as their dependent variable exclude men. Each column represents a single regression. Column 1 includes only observable individual controls, column 2 adds year fixed effects, and column 3 further adds state fixed effects. Each regression includes population weights and standard errors clustered at the state level.

In general, we find these variables are all highly correlated with the probability of an individual having had a particular screening test. As expected, as individuals get older, they are more likely to have undergone a scan; however, the marginal probability declines once they reach middle age, i.e., around age forty.[25] With the exception of black women undergoing mammograms, racial and ethnic minorities are—on average—less likely to have had any scan; although, this effect often becomes insignificance upon the addition of state and year fixed effects. Perhaps the most robust finding is that individuals with at least a high school diploma are significantly more likely to have had any screening procedure, particularly digital rectal and proctoscopic exams.[26]

One estimate in the mammogram panel of Table 3a runs contrary to expectations. Specifically, we find that women with some form of health insurance—be it publicly or privately

[25] We re-estimate the model using age fixed effects rather than continuous measures of age and age^2 and then plot the intercepts (relative to 18 year olds). Again, we find that screenings max out around middle age, except perhaps for proctoscopic exams which peak at age 70. See Appendix 2.
[26] Here, the omitted category includes high school dropouts.

sourced—are about 20 percentage points *less* likely to have had a mammogram in their lifetime than those without insurance, which represents a 35% decrease.[27] Since most states require health insurance companies to fully reimburse their subscribers' mammography costs, we expected to find a positive coefficient, which we do find for all other screening tests (NCI, 2010). Nevertheless, this estimate is subject to three sets of controls: individual, state, and year. Running a univariate regression of lifetime mammogram use and health insurance status yields the expected positive and highly statistically significant coefficient of 0.220, which implies that people with health insurance are 22 percentage points more likely to have had a mammogram in their lifetime than those without insurance.

The fact that—adding in state *and* year fixed effects—turns this and other coefficients statistically indistinguishable from zero underlies their importance and joint statistical significance as controls. In other words, our results suggest that national trends and preexisting time invariant differences across states account for a lot of the variability in scanning intensity between 1983 and 2000.

B. Impact of tort reform on medical screenings and procedures

Tables 4a-4d present the results of our most basic BRFSS specification, wherein the dependent variables are indicators of whether the individual received a specific screening test in the past year. Again, men are excluded from all regressions with mammogram or pap smear as their dependent variable, and each of the columns represents a separate regression. Columns 1-3 display the effect of a *cap* being turned "on" (i.e., damage awards have been capped), and columns 4-6 display the effect of a *direct reform* being turned "on" (i.e., either damage awards have been capped or the collateral source rule has been reformed or both). Within each panel, the

[27] 56.9% of women within our sample reported that they had undergone a mammography in their lifetime.

first column (columns 1 and 4) represents a single-variable regression of OUTCOME on TORT; the second column (columns 2 and 5) incorporates state- and year- fixed effects; and the third column (columns 3 and 6) adds the aforementioned vector of individual controls, resulting in a final specification identical to equation (1). Just as before, each regression includes population weights and standard errors clustered at the state level.

Broadly speaking, our results are consistent with the findings of Currie and MacLeod (2006). That is, women living in states with caps are being treated at a higher intensity than are women living in states without. Specifically, we find that women in the treatment group are 1-8 percentage points more likely to have had a mammogram and 3-12 percentage points more likely to have had a pap smear in the past year. Taking into account the full-sample probability of having had either test, these estimates amount to substantial 5-7% increases in the probability of past-year screening.

The estimates for rectal and proctoscopic exam usage, however, seem to suggest a different effect. Although none of the coefficients are ever statistically different from zero, the point estimates are consistently negative. This implies that—with respect to rectal and proctoscopic exams—physicians may have *decreased* treatment intensity in response to lower liability pressures. Returning to the Currie and MacLeod (2006) theoretical framework, this finding is consistent with a pre-reform environment where the use of rectal and proctoscopic exams was not excessive and so, in response to a damage cap (or collateral source rule reform), physicians merely cut back.

Overall, these results appear robust to changes in specification. That is, adding in controls, we find no difference in the direction and only modest percentage point differences in the magnitude of our coefficients of interest. These findings intimate that tort reform was in no way correlated with national trends, preexisting time invariant differences across states, or personal characteristics such that controlling for these factors does not result in a paring down of the coefficient shown in the simple regression framework.

The previous results examine the impact of tort reform on screening tests. As shown by both Kessler and McClellan (1996) and Currie and MacLeod (2006), tort reform may also affect the choice of treatment. We examine treatment intensity indicators in the cancer population with our analysis of the SEER data.

Table 5 again presents the results of a regression much like equation (1). Because of data limitations, we now only consider the effect of direct reform and include far fewer exogenous covariates as controls although state and year fixed effects remain. Since cancer patients may appear more than once within the SEER registry and cancer treatment varies across tumors, we cluster standard errors on two variables: individual and cancer type.

Here, our results are somewhat contradictory. On the one hand, as shown in column 1, we find that the probability of having surgery post-reform falls by a highly statistically significant 1.78 percentage points, which, given that 65.5% of tumors listed in the registry are treated with surgery, represents a modest 2.7% decrease. On the other hand, in the next column, the probability of radiation as a first course of therapy seems to *increase*. Although the coefficient itself is not statistically different from zero, the magnitude of the effect is on the same scale as that of surgery once average radiation rates are taken into account. Going back to the Currie and MacLeod (2006) theory, these results imply that surgery rates pre-reform were not excessive and radiation rates were.

The final regressions in columns 3 and 4 of Table 5 offer a retroactive look at the effect of tort reform on cancer-specific diagnostic treatment intensity, wherein the dependent variables are dummy variables of the stage at diagnosis. Our stage zero dummy (column 3) refers to a tumor diagnosed at its earliest possible stage whereas our stage four dummy (column 4) refers to a tumor diagnosed at its latest possible stage, i.e., after it has metastasized.[28]

If tort reform does cause doctors to carry out more scans like our results from the BRFSS suggest, then, we might expect that they would diagnose more tumors at earlier stages. Nevertheless, our estimates from Table 5 suggest the opposite. Specifically, the coefficient in column 3 indicates that direct reform decreases the probability of a tumor being diagnosed at stage zero by 0.1 percentage points: a relatively small 1.4% decrease. In contrast, the coefficient in column 4 indicates that tort reform increases the probability of a tumor being diagnosed at stage 4 by 1.94 percentage points: an increase of nearly 9%. This last finding—in conjunction with the fact that tumors diagnosed at later stages contribute most to cancer deaths—suggests that there may be serious health consequences associated with this shift in treatment due to tort reform (Clinton, 2006). As such, we now move to examine its effect on health outcomes.

C. Impact of tort reform on health outcomes

Tables 6 and 7 present analogous results to those in tables 4a-4d and 5, respectively. Here, however, we insert proxies for patient health outcomes. As shown by the coefficient on TORT in columns 3 and 6 of Table 6, we find little evidence of an effect of damage caps or the collateral source rule on the general public's overall health. Although columns 1 and 4 do suggest that people living in states with active tort reform are significantly healthier than people living in states without tort reform, adding in state and year fixed effects moves the coefficient back to zero. Nevertheless, the lack of an effect of tort reform here may not be particularly surprising since the overall health of the general population may be relatively unaffected by cancer-specific

[28] See Footnote 13 for a formal definition of the stages.

screening and procedure intensity. As such, in Table 7, we turn to cancer patient specific health outcomes.

Table 7 presents these results. The regression in column 1 has as its dependent variable a continuous measure of survival time (in years), and the regression in column 2 has as its dependent variable a dummy indicative of a cancer patient dying of cancer. To mitigate the right censoring issue, we limit both regressions to patients who had died by the SEER registry's December 2008 cutoff. We include only those individuals who died of cancer in the survival time regressions.

As noted previously, survival time post-diagnosis and probability of death by cancer—our two proxies of cancer patient health outcomes—are inherently linked. If we are living longer post-diagnosis, then our probability of dying of cancer at any given moment is lower. Based on our previous finding that tort reform leads doctors to diagnose tumors at later stages, we might expect a positive coefficient on TORT when the dependent variable is death by cancer and a negative coefficient on TORT when the dependent variable is survival time. Indeed, this is exactly what we find.

Although statistically insignificant from zero, the point estimate on TORT in the regression with survival time in column 1 not only has the expected negative sign but also is extremely large in magnitude. It implies that cancer patients diagnosed in states with direct tort reform die, on average, a half year earlier than cancer patients in states without reform, all else equal. This finding is corroborated by the positive coefficient in column 2. Of all cancer patients who died prior to December 2008, those who were diagnosed in states with direct tort reform were 1.32 percentage points more likely to die of cancer: an substantial increase of nearly 4%.

D. Hazard survival analysis

As discussed in the methods section, our survival data from SEER is subject to both right and left censoring. To the extent that this censoring is correlated with tort reform, our estimates of tort reform's effects on cancer patient outcomes using our differences-in-differences specification may be biased. For example, if tort reform leads doctors to diagnose tumors at later stages, as our preliminary findings strongly suggest, then any estimate of the effect of tort reform on cancer mortality will necessarily be biased upwards due to this left censoring. We use a Cox Proportional Hazards model, estimating equation (2), to evaluate this possibility.

Table 8 presents our results.[29] Column 1 omits controls; column 2 includes year fixed effects; column 3 adds a state fixed effect; and column 4 adds age, sex, and race individual controls. The variable of greatest interest—diagnosed after reform—is a dummy which receives a value of 1 if a cancer patient was diagnosed in a state which had enacted direct tort reform and 0 otherwise.

Consistent with our differences-in-differences estimates, we find that tort reform is associated with an upward shift of in the Hazard function. In other words, our results suggest that tort reform lowers cancer patients' survival times post-diagnosis. This finding is robust to the inclusion of controls—the coefficient retains its magnitude and high statistical significance throughout each of our four specifications. Moreover, the magnitude of the effect is consistent with that of our linear probability model. Whereas before we estimated that tort reform increased the probability of cancer death by 4 percentage points, here we find a range of estimates between 2 and 6 percentage points. In sum, this consistency lends credence to the validity of our baseline model in spite of its inability to account for censoring.

[29] We display coefficients instead of hazard ratios.

E. Heterogeneous effects: the role of discretion

Another potential flaw with our baseline model is that it fails to account for the fact that physicians may not have a lot of discretion in deciding whether or not to carry out the screening tests we use as dependent variables. Since the US Preventive Services Task Force (2012) annually publishes guidelines on how often an individual of a given age, race, and sex should have a mammogram, pap smear, rectal exam, and proctoscopic exam, there may be little scope for physicians to push for any more of these scans without strong medical evidence that they are needed, even if the doctors themselves are inclined to do so post-reform. Since we do not have access to other testing variables, isolating instances where doctors have the most discretion to carry out these tests may be the key to identifying whether or not our findings underestimate the effect of tort reform on scanning intensity.

As shown in Tables 3a and 3b, individuals are increasingly more likely to have had any scan (in their lifetime) the older they get. But, as illustrated graphically in Appendix 1, whereas women experience a surge in the probability of their getting a mammogram upon turning forty, they experience no corresponding surge at that age in the probability of their getting a pap smear. This is broadly consistent with national guidelines which recommend that women start getting mammograms once every two years after age 50 and pap smears once every three years after age 21 (USPSTF, 2012). Accordingly, separating individuals into below-40 and 40-and-over age groups may be the simplest and most natural way of creating a proxy for physician discretion. In other words, if we re-run our baseline regression—equation (1)—separately for the two age groups, we would expect to find a coefficient on TORT which is larger and more positive for the younger group with mammogram in the past year as the dependent variable and coefficients which are about the same across the two groups with pap smear in the past year as the dependent variable since doctors have greater discretion to carry out additional mammograms among younger women.

Indeed, panels A and B of Table 9 support this theory. We find that direct tort reform increases the probability of having had a mammogram in the past year by 2.38 percentage points (4%) for women under 40 and by only 0.80 percentage points (1%) for women 40 and above. We find that tort reform increases the probability of having had a pap smear by 1.63 percentage points (2%) for women under 40 and 1.99 percentage points (3%) for women 40 and above. Once again, as implied by our earlier estimates, we find that tort reform increased scanning intensity. But, these age-block results show that tort reform's effect on mammogram use is stronger in the under 40 population, where we suspect that doctors have more treatment discretion. This last finding suggests that we may have underestimated the effect of tort reform on scanning intensity.

F. Controlling for health insurance status

Researchers have shown repeatedly that an individual's use of health care services is intrinsically tied to his or her health insurance status.[30] We confirm this with our first series of regressions in Table 3a and 3b. As such, parsing out effects of tort reform for the insured and uninsured separately may help to eliminate some of the ambiguity of our findings.

Previous studies have shown that having insurance—be it publically, privately, or employer provided—can contribute to a patient's insensitivity to the marginal dollar spent on health care. Using the Rand Health Insurance Experiment (HIE), Manning et. al. (1987) found that patients who had to pay more out-of-pocket used fewer health care resources, e.g., fewer face-to-face

[30] Card et. al. (2008) used a regression discontinuity design to show that newly eligible Medicare recipients experienced higher treatment intensity and larger reductions in mortality relative to those elderly individuals just ineligible for Medicare. See also Hurd & McGarry (1995).

visits with doctors, fewer outpatient expenses, etc. We would expect therefore that insured individuals would be more likely to along with doctor's attempts to increase treatment intensity since they do not bear the full out-of-pocket cost of this increased treatment.

To test this hypothesis, we again re-estimate equation (1), but this time, we include a dummy variable for insurance status and an additional interaction term between insurance status and TORT. If, as we have shown, tort reform increases scanning intensity, then we would expect to find not only a positive coefficient on the health insurance dummy reflecting the increased access to health care scans for the insured but also a positive coefficient on the interaction term and the uninteracted TORT term. Thus, for any increase in treatment intensity associated with a cap being turned "on," we should find a correspondingly larger effect for the insured. We run these regressions using only the BRFSS data as the SEER data contains no information on insurance status.

Tables 10a and 10b present the results of the aforementioned series of regressions. We find that the interaction term always has the *opposite* sign as TORT, regardless of scan. Thus, instead of compounding any effect of tort reform, having insurance actually offsets it. For example, uninsured women are significantly *more* likely to have had a mammogram when a cap is turned "on" in their state of residence while their insured counterparts are significantly *less* likely. That is, the sum of the coefficients on TORT and the interaction term, -0.046, is statistically different from zero at the 1% significance level.

This robust finding suggests that insurance companies are providing some friction against physicians' attempts to increase treatment intensity post-reform. Perhaps, they apply this pressure on the patients themselves, limiting them to only one mammogram or pap smear per year such that they must bear the full cost burden of subsequent tests if they choose go through with them.

G. Threats to validity

The most acute threat to the validity of our study is whether or not the treatment and control groups—states with and without tort reform, respectively—are moving along the same trend line or, at the very least, a parallel trend line with respect to treatment and health outcomes in the pre-reform period. This is known as the "parallel trends" assumption. By including individual controls and state and year fixed effects in our specifications, our estimates will be valid as long as there are no state-specific time trends related to health.

As a first attempt at evaluating whether our model meets this assumption, we run two placebo regressions, re-estimating equation (1) but using height (in inches) and a dummy for seatbelt use as dependent variables. Given what we know about tort reforms, it seems unlikely that they would have any direct effect on an individual's height or his or her decision to wear a seatbelt. We would therefore expect the coefficient on TORT to equal zero as long as our specification was unaffected by any state-specific time trends correlated with either of these proxies for health. Any non-zero coefficient would suggest a violation of the parallel trends assumption and bring into question the validity of our estimates elsewhere in our work.

Table 11 presents the results of these placebo regressions. The estimates of the effect of both damage caps and direct reforms on height and seatbelt are never statistically significant and always small in magnitude. Specifically, living in a state that caps damage awards is associated with a 0.005-inch reduction in height and a 3.45 percentage point increase in the probability of wearing a seatbelt. Relative to average height and seatbelt use rates in our sample, these represent very modest 0.007% decreases and a 4% increases, respectively. Our failure to detect any spurious treatment effect suggests that the parallel trends assumption is appropriate here. Nevertheless, in the event that these placebo regressions did not identify a state-specific trend only because the underlying trend variable was uncorrelated with both our health proxies, we perform another test.

As discussed previously, it seems likely that state legislators may have enacted tort reform in the 1980s and 1990s in reaction to an alarming upward trend in medical malpractice fees. To the extent that these fees rising were reflective of an unobserved state-specific trend in defensive medicine, our estimate of the effect of tort reform could be biased towards finding doctors further increasing treatment intensity. If instead these rising fees are reflective of a state-specific trend in negative defensive medicine, our estimate of the effect of tort reform could be biased towards finding doctors further decreasing treatment intensity.[31]

To test for this type of policy endogeneity, we re-estimate equation (1) allowing TORT to take on a non-linear outcome trend rather than limiting it to a simple on-and-off structure. We include one and two year leads and one, two, and three or more year lags of TORT, where each term is a dummy which receives a value of one if it is that many years before or after the reform was enacted. Again, if policy endogeneity were not present, we would expect each lead term to approach zero. If it were present, then we would expect lead terms to be significantly different from zero and increasing in magnitude—be it in a positive or negative direction.

As shown in Table 12a and graphically in Appendix 3, we find little evidence of policy endogeneity in scanning intensity and general health. As expected, none of the coefficients on any of the lead terms are statistically different from zero: a result robust not only to the choice of dependent variable but also, given the similar findings in Table 12b, to the specification of TORT. We estimate coefficients of equal signs and similar magnitudes whether we look at the

[31] Negative defensive medicine occurs when doctors underprovide their services out of liability concerns.

effect of damage caps or direct reforms more generally. Moreover, the graphs of mammogram and pap smear use in Appendix 3 illustrate a discrete break in the trendline either in the year of reform or one year after reform, indicating that, although tort reform led to increased numbers of scans, there was somewhat of a delay. This would be consistent with doctors' only slowly internalizing their lowered liability.

When we consider only cancer patients, we do find evidence of policy endogeneity. As shown in column one of Table 13, every lead coefficient in a regression with a dummy dependent variable of surgery use is not only negative but also statistically different from zero. This result suggests that tumors diagnosed in states with an active tort law or a soon-to-be active tort law were 4-6 percentage points less likely to be surgically operated on. Scaled appropriately, this represents a 9% difference.

Closer inspection of the graphed lead and lag coefficients from this regression in Appendix 4 suggests that a policy endogeneity story relating to negative defensive medicine may explain our non-zero lead coefficients. That is, prior to reform, doctors in states with reform were carrying out relatively fewer surgical procedures, and—as a result—policymakers intervened and enacted tort reform in hopes of bringing the surgical rate back in line with other states. The slight upward trend of the line upon enactment of the damage cap suggests that our previously estimated negative effect of a damage cap on the probability of surgical intervention from Table 5 was dwarfing the true positive effect of the damage cap. That is, with endogeneity present, the negative effect we estimated might have only reflected the fact that states with reform have doctors who are less willing to advocate surgery. In reality, tort reform led doctors to increase procedure intensity.

Taken altogether, the results from these two tests indicate that assuming parallel trends in screening and health outcomes is appropriate. Therefore, we may interpret our estimates of the effect of tort reform on these areas as causal. Based on the likelihood of policy endogeneity with respect to surgical use, however, we may *not* interpret our estimates as causal. Therefore, although our differences-in-differences estimates suggest that tort reform resulted in a decrease in procedure intensity, graphical inspection of leads and lags of the reform implies that the true effect of tort reform was to increase procedure intensity.

VII. DISCUSSION

With this paper, we seek to understand the effect of direct tort reform on doctors' treatment decisions and patients' health outcomes within the field of cancer. We use cancer as a case study not only because of its prominence within the US health care system but also because of its reliance on discretion in treatment.

If tort reform were to have an effect on defensive medicine, then it would have to work through reducing or, at the very least, realigning the liability pressures on doctors. Our reading of the literature suggests that the direct tort reforms enacted by states in the 1980s and 1990s were very successful in doing this. Not only did they lower malpractice claims payouts and insurance premiums, but they also cut down on the number of medical malpractice claims brought to court. As such, it would seem reasonable to expect that these direct reforms would have resulted in less defensive medicine since defensive medicine, at its core, is spurred on by doctors' fears of their own liability.

Our results indicate, however, that states' direct reform of their tort laws during this period did *not* result in the reduction or elimination of doctors' defensive practices. If anything, we find evidence which suggests that they became even *more* defensive in how they practiced medicine.

On the one hand, we find that doctors in states with tort reform started carrying out more cancer screenings once caps were enacted and—although not a statistically significant finding—it appears as though they may have even pushed for more intensive treatments. This screening effect is robust to several specifications. Re-estimating our same baseline model but accommodating differential effects by age and health insurance status, we discover that our baseline estimates may even underestimate the true magnitude of the effect of tort reform on cancer screening intensity since the tests that we use in our models—mammograms, pap smears, rectal exams, and proctoscopic exams—do not offer doctors as much discretion as other, less prevalent cancer screening tests might.

On the other hand, we also find evidence that cancer patients in states with tort reform have much worse health outcomes post-reform. Not only do we find that their tumors are significantly more likely to be diagnosed at stage four, but we also find that they are significantly more likely to die as a result of their cancer at any given time. Much like our testing result, this health outcome effect is also robust to several specifications, including survival analysis using a Hazard model.

In the introduction to our paper, we offered two testable predictions for determining whether or not direct tort reforms would cut back on defensive medicine. One, we would find that physicians opted for less intensive treatments on the margin, and two, we would find that cancer patients' health outcomes would be unaffected. Given that our estimates imply that cancer treatment intensity increased and cancer patient health outcomes decreased post-reform, our work appears to offer compelling evidence that direct tort reform neither eliminates nor reduces defensive medicine in cancer treatment.

In sum, despite the complicated nature of this analysis, its ultimate policy prescription is very simple. At least in the field of cancer, the costs of direct tort reform—both in terms of added defensive medicine and loss of life—appear to outweigh the benefits. Therefore, policymakers should be wary of advocating direct reform as a safe cost-saving measure.

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IX. Tables

Table 1: Summary Statistics for Key Dependent Variables with Demographic Controls

	1988	1994	2000	Min	Max
Outcome variables					
Have you ever had a:					
-mammogram [W]	0.409	0.543	0.625	0	1
-pap smear [W]	0.951	0.932	0.943	0	1
-rectal exam	0.726	0.734	-	0	1
-proctoscopic exam	0.278	0.297	-	0	1
In the last year, have you had a:					
-mammogram [W]	0.653	0.612	0.694	0	1
-pap smear [W]	0.705	0.689	0.707	0	1
-rectal exam	0.571	0.578	-	0	1
-proctoscopic exam	0.390	0.341	-	0	1
Are you currently in good health?	-	0.582	0.55	0	1
Background variables					
Female	0.523	0.520	0.519	0	1
Age	43.4	43.9	45.4	18	99
Black	0.087	0.091	0.101	0	1
Hispanic	0.082	0.078	0.120	0	1
Other race	0.028	0.032	0.036	0	1
Married	0.612	0.602	0.586	0	1
High school diploma	0.339	0.325	0.310	0	1
Some College	0.269	0.264	0.273	0	1
College or more	0.218	0.252	0.282	0	1
Do you have health insurance?	-	0.862	0.860	0	1
Employed	0.628	0.626	0.642	0	1
Height (inches)	67.0	67.1	67.1	24	101
Do you have wear a seatbelt?	0.612	0.907	-	0	1
Total	55,283	104,414	177,773		

Source: Behavioral Risk Factor Surveillance System, 1987-2000. The data have been weighted using CDC sampling weights. The [W] implies that the variable is relevant to women only. The "-" implies that the variable was not included in the BRFSS survey during that year. The increasing number of observations reflect the fact that, throughout the 1980s and 1990s, the BRFSS was being introduced in additional states.

Table 2: Summary Statistics for Key Dependent Variables with Demographic Controls

	1983	1991	2000	Min	Max
Outcome variables					
Did you have any surgical procedure to remove and/or to destroy tissue of the primary site in the first course of treatment?	0.689	*0.662*	*0.642*	*0*	*1*
Did you have any radiation therapy as part of the first course of treatment?	0.249	0.255	0.287	0	1
Did you have multiple tumors?	0.346	0.472	0.502	0	1
Stage Zero	0.117	0.148	0.080	0	1
Stage Four	0.213	0.236	0.212	0	1
Survival time (years) if you eventually died of cancer	2.835	2.614	1.565	0	26
Probability of dying of cancer	0.437	0.353	0.293	0	1
Background variables					
Female	0.522	0.507	0.493	0	1
Age	63.479	63.641	65.136	0	116
Age at diagnosis	62.989	63.155	64.639	0	115
Black	0.084	0.085	0.093	0	1
Hispanic	0.001	0.019	0.037	0	1
Source of Information					
Hospital inpatient	97.1%	93.9%	93.5%		
Radiation treatment center	0.0%	0.0%	0.0%		
Laboratory	0.6%	2.7%	2.3%		
Physician's Office	0.9%	2.2%	3.1%		
Nursing home or hospice	0.1%	0.0%	0.0%		
Autopsy	0.7%	0.4%	0.2%		
Death certificate	0.7%	0.8%	1.0%		
Total	89,800	123,207	182,264		

Source: Surveillance Epidemiology and End Results (SEER) Program, 1983-2000. These data have *not* been weighted. The underlying sample for the calculation of survival time includes only cancer patients who died of cancer prior to January 2009; the probability of dying of cancer includes all cancer patients who died prior to January 2009. All other calculations include the full sample within the registry.

Table 3a: Empirical Evidence of Determinants of Mammogram and Pap Smear Usage among Women in 1987-2000

	Have you *ever* had a mammogram?			Have you *ever* had a pap smear?		
	(1)	(2)	(3)	(4)	(5)	(6)
AVG of Y		0.569			0.937	
Age	0.0545***	0.0587***	0.0591***	0.0109***	0.0108***	0.0107***
	(0.000736)	(0.000797)	(0.00101)	(0.00370)	(0.00348)	(0.00347)
Age^2	-0.000417***	-0.000442***	-0.000443***	-9.48e-05**	-9.42e-05**	-9.31e-05**
	(7.20e-06)	(1.36e-05)	(1.63e-05)	(4.26e-05)	(4.12e-05)	(4.13e-05)
Black	-0.0698**	0.00134	0.00645	0.0634***	0.0618***	0.0579***
	(0.0274)	(0.00657)	(0.00889)	(0.0178)	(0.0207)	(0.0193)
Hispanic	-0.0510***	0.00973	0.0224	0.0577	0.0566	0.0570
	(0.00514)	(0.0228)	(0.0201)	(0.0447)	(0.0477)	(0.0502)
Other race	-0.0353***	-0.00387	-0.00304	-0.00469	-0.00489	-0.00425
	(0.0105)	(0.00682)	(0.00647)	(0.0151)	(0.0158)	(0.0152)
Married	-0.145***	-0.110***	-0.106***	0.0737***	0.0729***	0.0714***
	(0.0535)	(0.0347)	(0.0360)	(0.0133)	(0.0146)	(0.0148)
High school diploma	0.406*	0.409*	0.410*	0.408*	0.408*	0.410*
	(0.224)	(0.220)	(0.220)	(0.229)	(0.230)	(0.230)
Some college	0.251	0.219	0.218	0.375*	0.376*	0.377*
	(0.159)	(0.139)	(0.140)	(0.195)	(0.194)	(0.195)
College or more	0.261*	0.307*	0.313*	0.340*	0.340*	0.342*
	(0.152)	(0.168)	(0.166)	(0.178)	(0.180)	(0.182)
Health insurance	-0.237**	-0.198*	-0.197	0.101***	0.0999***	0.101***
	(0.105)	(0.116)	(0.120)	(0.00493)	(0.00383)	(0.00366)
Constant	-0.833***	-1.007***	-1.112***	0.204*	0.208	0.227*
	(0.0263)	(0.0525)	(0.0579)	(0.116)	(0.125)	(0.123)
Year fixed effects		X	X		X	X
State fixed effects			X			X
Observations	702,322	702,322	702,322	699,471	699,471	699,471
R-squared	0.425	0.454	0.456	0.367	0.368	0.370

Source: BRFSS, 1987-2000. Women are excluded from all regressions. The dependent variable is a dummy variable of a lifetime treatment indicator. Robust standard errors, clustered by state, are in parentheses. Results are estimated using OLS. "X" indicates the inclusion of fixed effects. The number of observations varies across dependent variable because of survey non-response. Statistical significance is denoted as follows: *** p<0.01, ** p<0.05, * p<0.10.

Table 3b: Empirical Evidence of Determinants of Rectal and Proctoscopic Exam Usage in 1987-2000

	Have you *ever* had a rectal exam?			Have you *ever* had a proctoscopic exam?		
	(1)	(2)	(3)	(4)	(5)	(6)
AVG of Y	0.701			0.279		
Age	0.0223***	0.0292***	0.0294***	0.0152***	0.0212***	0.0212***
	(0.00256)	(0.000813)	(0.000851)	(0.00285)	(0.00298)	(0.00298)
Age^2	-0.000155***	-0.000209***	-0.000211***	-5.42e-05**	-0.000101***	-0.000102***
	(1.86e-05)	(8.19e-06)	(7.96e-06)	(2.48e-05)	(2.61e-05)	(2.61e-05)
Female	-0.0389***	-0.0391***	-0.0385***	-0.0581***	-0.0588***	-0.0586***
	(0.0106)	(0.0105)	(0.0104)	(0.00654)	(0.00646)	(0.00644)
Black	-0.0291***	-0.0286***	-0.0210***	-0.00474	-0.00227	-0.00336
	(0.00876)	(0.00847)	(0.00605)	(0.00788)	(0.00821)	(0.00885)
Hispanic	-0.0373**	-0.0472***	-0.0603***	0.00618	-0.00378	-0.00644
	(0.0157)	(0.0149)	(0.0140)	(0.0167)	(0.0125)	(0.0142)
Other race	-0.0495***	-0.0430**	-0.0491**	-0.0127	-0.00473	-0.00848
	(0.0169)	(0.0166)	(0.0186)	(0.0205)	(0.0200)	(0.0208)
Married	0.0192***	0.0201***	0.0236***	0.0140***	0.0148***	0.0151***
	(0.00452)	(0.00396)	(0.00343)	(0.00270)	(0.00261)	(0.00257)
High school diploma	0.0390***	0.0371***	0.0349***	0.0311***	0.0278***	0.0266***
	(0.00810)	(0.00690)	(0.00607)	(0.00607)	(0.00663)	(0.00605)
Some college	0.0828***	0.0801***	0.0719***	0.0601***	0.0554***	0.0522***
	(0.00893)	(0.00797)	(0.00743)	(0.00848)	(0.00952)	(0.00880)
College or more	0.113***	0.109***	0.101***	0.0897***	0.0839***	0.0812***
	(0.0113)	(0.00953)	(0.00866)	(0.00873)	(0.0112)	(0.0106)
Health insurance	0.122***	0.122***	0.124***	0.0485***	0.0481***	0.0487***
	(0.00830)	(0.00864)	(0.00583)	(0.00943)	(0.00874)	(0.00891)
Year fixed effects		X	X		X	X
State fixed effects			X			X
Observations	145,596	145,596	145,596	135,646	135,646	135,646
R-squared	0.058	0.064	0.082	0.087	0.092	0.096

Source: BRFSS, 1987-2000. These regressions include both men and women. The dependent variable is a dummy variable of a lifetime treatment indicator. Robust standard errors, clustered by state, are in parentheses. Results are estimated using OLS. "X" indicates the inclusion of fixed effects. The number of observations varies across dependent variable because of survey non-response. Statistical significance is denoted as follows: *** p<0.01, ** p<0.05, * p<0.10.

Table 4a: Mammogram Use in 1-Year Period by Women

Avg. of Y = 0.639	*Damage Caps*			*Direct Reform*		
	(1)	(2)	(3)	(4)	(5)	(6)
Reform is "on"	0.0795***	0.0203*	0.00854	0.0748***	0.0167**	0.0117*
	(0.0190)	(0.0110)	(0.00756)	(0.0195)	(0.00823)	(0.00625)
Year fixed effects		X	X		X	X
State fixed effects		X	X		X	X
Demographic controls			X			X
Observations	470,170	470,170	470,170	470,170	470,170	470,170
R-squared	0.007	0.013	0.121	0.006	0.013	0.121

Source: BRFSS, 1987-2000. These regressions include only women. The dependent variable is a dummy variable which equals 1 if a woman underwent a mammogram in the past year and 0 otherwise. Robust standard errors, clustered by state, are in parentheses. Results are estimated using OLS. "X" indicates the inclusion of a given type of control. Columns 1-3 estimate the effect of a cap on damages awards, and columns 4-6 estimate the effect of a direct reform. Statistical significance is denoted as follows: *** p<0.01, ** p<0.05, * p<0.10.

Table 4b: Pap Smear Use in 1-Year Period by Women

Avg. of Y = 0.685	*Damage Caps*			*Direct Reform*		
	(1)	(2)	(3)	(4)	(5)	(6)
Reform is "on"	0.119***	0.0501***	0.0307***	0.110***	0.0250**	0.0203**
	(0.0233)	(0.0154)	(0.00880)	(0.0247)	(0.0101)	(0.00899)
Year fixed effects		X	X		X	X
State fixed effects		X	X		X	X
Demographic controls			X			X
Observations	704,465	704,465	704,465	704,465	704,465	704,465
R-squared	0.017	0.027	0.184	0.015	0.027	0.184

Source: BRFSS, 1987-2000. These regressions include only women. The dependent variable is a dummy variable which equals 1 if a woman underwent a pap smear in the past year and 0 otherwise. Robust standard errors, clustered by state, are in parentheses. Results are estimated using OLS. "X" indicates the inclusion of a given type of control. Columns 1-3 estimate the effect of a cap on damages awards, and columns 4-6 estimate the effect of a direct reform. Statistical significance is denoted as follows: *** p<0.01, ** p<0.05, * p<0.10.

Table 4c: Digital Rectal Exam Use in 1-Year Period

Avg. of Y = 0.574	*Damage Caps*			*Direct Reform*		
	(1)	(2)	(3)	(4)	(5)	(6)
Reform is "on"	-0.0228	-0.00689	-0.00663	-0.0208	-0.00212	-0.00158
	(0.0253)	(0.0215)	(0.0208)	(0.0190)	(0.0215)	(0.0208)
Year fixed effects		X	X		X	X
State fixed effects		X	X		X	X
Demographic controls			X			X
Observations	119,977	119,977	119,977	119,977	119,977	119,977
R-squared	0.000	0.008	0.024	0.000	0.008	0.024

Source: BRFSS, 1987-2000. These regressions include both men and women. The dependent variable is a dummy variable which equals 1 if an individual underwent a digital rectal exam in the past year and 0 otherwise. Robust standard errors, clustered by state, are in parentheses. Results are estimated using OLS. "X" indicates the inclusion of a given type of control. Columns 1-3 estimate the effect of a cap on damages awards, and columns 4-6 estimate the effect of a direct reform. There are relatively fewer observations in this panel because the BRFSS survey included this variable for a shorter period of time, 1988-1996. Statistical significance is denoted as follows: *** p<0.01, ** p<0.05, * p<0.10.

Table 4d: Proctoscopic Exam Use in 1-Year Period

Avg. of Y = 0.366	*Damage Caps*			*Direct Reform*		
	(1)	(2)	(3)	(5)	(6)	(7)
Reform is "on"	-0.0306	-0.0130	-0.0149	-0.0218	-0.00143	-0.00374
	(0.0268)	(0.0275)	(0.0236)	(0.0202)	(0.0263)	(0.0224)
Year fixed effects		X	X		X	X
State fixed effects		X	X		X	X
Demographic controls			X			X
Observations	46,864	46,864	46,864	46,864	46,864	46,864
R-squared	0.000	0.015	0.034	0.000	0.015	0.034

Source: BRFSS, 1987-2000. These regressions include both men and women. The dependent variable is a dummy variable which equals 1 if an individual underwent a proctoscopic exam in the past year and 0 otherwise. Robust standard errors, clustered by state, are in parentheses. Results are estimated using OLS. "X" indicates the inclusion of a given type of control. Columns 1-3 estimate the effect of a cap on damages awards, and columns 4-6 estimate the effect of a direct reform. There are relatively fewer observations in this panel because the BRFSS survey included this variable for a shorter period of time, 1988-1995. Statistical significance is denoted as follows: *** p<0.01, ** p<0.05, * p<0.10.

Table 5: Prevalence of Surgery, Radiation, and the Stage at Diagnosis

	SURGERY (1)	RADIATION (2)	STAGE 0 (3)	STAGE 4 (4)
Avg. of Y	0.655	0.262	0.109	0.222
Direct reform is "on"	-0.0178***	0.00417	-0.00157	0.0194***
	(0.00678)	(0.00757)	(0.00701)	(0.00717)
Year fixed effects	X	X	X	X
State fixed effects	X	X	X	X
Demographic controls	X	X	X	X
Observations	2,279,108	2,341,872	1,824,864	1,824,864
R-squared	0.852	0.790	0.830	0.866

Source: SEER, 1983-2000. These regressions include all tumors diagnosed between January 1983 and December 2000. The dependent variables are dummy variables which equal 1 if the tumor: required surgery (column 1), required radiation (column 2), was diagnosed at stage zero (column 3), or was diagnosed at stage four (column 4). All columns estimate the effect of a direct reform. Robust standard errors, clustered by individual and type of cancer, are in parentheses. Results are estimated using OLS. "X" indicates the inclusion of a given type of control. The number of observations varies across dependent variables because of non-response. Statistical significance is denoted as follows: *** p<0.01, ** p<0.05, * p<0.10.

Table 6: Good Health

Avg. of Y = 0.594	*Damage Caps*			*Direct Reform*		
	(1)	(2)	(3)	(4)	(5)	(6)
Reform is "on"	0.287***	0.00472	-0.00364	0.242***	0.00517	0.00124
	(0.0655)	(0.00424)	(0.00732)	(0.0780)	(0.00440)	(0.00525)
Year fixed effects		X	X		X	X
State fixed effects		X	X		X	X
Demographic controls			X			X
Observations	1,484,740	1,484,740	1,474,182	1,484,740	1,484,740	1,474,182
R-squared	0.072	0.647	0.678	0.053	0.647	0.678

Source: BRFSS, 1987-2000. These regressions include both men and women. The dependent variable is a dummy variable which equals 1 if an individual reports they are in good, very good, or excellent health. Robust standard errors, clustered by state, are in parentheses. Results are estimated using OLS. "X" indicates the inclusion of a given type of control. Columns 1-3 estimate the effect of a cap on damages awards, and columns 4-6 estimate the effect of a direct reform. Statistical significance is denoted as follows: *** p<0.01, ** p<0.05, * p<0.10.

Table 7: Survival Time Post-Diagnosis and Probability of Death by Cancer

	Survival time (1)	Death by cancer (2)
Avg. of Y	2.323	0.353
Direct reform is "on"	-0.568	0.0132**
	(0.601)	(0.00621)
Year fixed effects	X	X
State fixed effects	X	X
Demographic controls	X	X
Observations	858,496	2,411,755
R-squared	0.935	0.809

Source: SEER, 1983-2000. These regressions include only cancer patients who died before January 2009. Of these, column 1 includes only those cancer patients who died of cancer. The dependent variable in column 1 is a continuous measure (in years) of a cancer patient's survival time post diagnosis whereas the dependent variable in column 2 is a dummy variable which equals 1 if the patient died of cancer and 0 otherwise. Both columns estimate the effect of a direct reform. Robust standard errors, clustered by individual and type of cancer, are in parentheses. Results are estimated using OLS. "X" indicates the inclusion of a given type of control. The number of observations varies across dependent variables because of non-response. Statistical significance is denoted as follows: *** p<0.01, ** p<0.05, * p<0.10.

Table 8: Survival Analysis with Cox Proportional Hazards Model

VARIABLES	(1)	(2)	(3)	(4)
Diagnosed after reform	0.0611***	0.0252***	0.0190***	0.0198***
	(0.00207)	(0.00307)	(0.00307)	(0.00307)
Year fixed effects		X	X	X
State fixed "effect"			X	X
Demographic controls				X
Observations	1,757,185	1,757,185	1,757,185	1,757,185

Source: SEER, 1983-2000. Coefficients are shown instead of exponentiated coefficients. "X" indicates the inclusion of a given type of control. The state fixed effect is included to control for time invariant differences between states with and without tort reform. The demographic control variables include age at diagnosis, sex, and race (black of Hispanic). All columns estimate the effect of a direct reform. Statistical significance is denoted as follows: *** p<0.01, ** p<0.05, * p<0.10.

Table 9: Mammogram and Pap Smear Use in 1-Year Period by Age Block

Panel A: Under 40

VARIABLES	(1) Mammogram	(2) Pap smear
Avg. of Y	0.577	0.803
Direct reform is "on"	0.0238	0.0163**
	(0.0205)	(0.00714)
Observations	86,168	287,924
R-squared	0.350	0.127

Panel B: 40 and over

VARIABLES	(1) Mammogram	(2) Pap smear
Avg. of Y	0.653	0.602
Direct reform is "on"	0.00880	0.0199
	(0.00610)	(0.0126)
Observations	379,793	411,169
R-squared	0.093	0.103

Source: BRFSS, 1987-2000. These regressions include only women. The dependent variable is a dummy variable which equals 1 if a woman underwent a mammogram (column 1) or a pap smear (column 2) in the past year and 0 otherwise. Robust standard errors, clustered by state, are in parentheses. Results are estimated using OLS. Both regressions include a full set of individual controls, state fixed effects, and year fixed effects. Statistical significance is denoted as follows: *** p<0.01, ** p<0.05, * p<0.10.

Table 10a: Past-year Screening Use and General Health Outcomes Controlling for Health Insurance

VARIABLES	(1) Mammogram	(2) Pap smear	(3) Rectal exam	(4) Proctoscopic exam	(5) Good health
Cap is "on"	0.535***	0.246***	-0.0464	0.0324	0.0340
	(0.109)	(0.0479)	(0.0364)	(0.0951)	(0.0342)
Has insurance * cap is "on"	-0.581***	-0.245***	0.0701***	-0.0325	-0.0430
	(0.110)	(0.0507)	(0.0234)	(0.0764)	(0.0466)
Has insurance	0.167***	0.178***	0.140***	0.0322**	0.0666***
	(0.0208)	(0.0184)	(0.00858)	(0.0153)	(0.00633)
Observations	412,374	647,398	101,896	39,902	1,220,796
R-squared	0.182	0.200	0.028	0.031	0.321

Source: BRFSS, 1987-2000. Columns 1 and 2 include only women. Columns 3-5 include both men and women. The dependent variable in columns 1-4 is a dummy variable which equals 1 if an individual has had a particular scan in the past year and 0 otherwise. The dependent variable in column 5 is a dummy variable which equals 1 if the individual reported being in good, very good, or excellent health and 0 otherwise. All regressions measure the effect of a cap on damage awards. Robust standard errors, clustered by state, are in parentheses. Results are estimated using OLS. All regressions include a full set of individual controls, state fixed effects, and year fixed effects. Statistical significance is denoted as follows: *** p<0.01, ** p<0.05, * p<0.10.

Table 10b: Past-year Screening Use and General Health Outcomes Controlling for Health Insurance

VARIABLES	(1) Mammogram	(2) Pap smear	(3) Rectal exam	(4) Proctoscopic exam	(5) Good health
Direct reform is “on”	0.528***	0.233***	0.0168	0.0465	0.0431
	(0.0990)	(0.0429)	(0.0317)	(0.0536)	(0.0407)
Has insurance * direct reform is “on”	-0.560***	-0.236***	0.0139	-0.0343	-0.0411
	(0.105)	(0.0468)	(0.0257)	(0.0375)	(0.0454)
Has insurance	0.168***	0.178***	0.142***	0.0342**	0.0667***
	(0.0213)	(0.0186)	(0.00872)	(0.0160)	(0.00644)
Observations	412,374	647,398	101,896	39,902	1,220,796
R-squared	0.180	0.199	0.028	0.031	0.321

Robust standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1

Source: BRFSS, 1987-2000. Columns 1 and 2 include only women. Columns 3-5 include both men and women. The dependent variable is a dummy variable which equals 1 if an individual has had a particular scan in the past year and 0 otherwise. All regressions measure the effect of direct tort reform. Robust standard errors, clustered by state, are in parentheses. Results are estimated using OLS. All regressions include a full set of individual controls, state fixed effects, and year fixed effects. Statistical significance is denoted as follows: *** p<0.01, ** p<0.05, * p<0.10.

Table 11: Placebo Regressions

	Damage Caps		*Direct Reform*	
	HEIGHT (1)	SEATBELT USE (2)	HEIGHT (3)	SEATBELT USE (4)
Avg. of Y	66.893	0.842	66.893	0.842
Reform is “on”	-0.00541	0.0345	0.00904	0.00743
	(0.0165)	(0.0413)	(0.0135)	(0.0221)
Year fixed effects	X	X	X	X
State fixed effects	X	X	X	X
Demographic controls	X	X	X	X
Observations	1,462,705	72,286	1,462,705	72,286
R-squared	0.048	0.065	0.048	0.064

Source: BRFSS, 1987-2000. These regressions include men and women. In columns 1 and 3, the dependent variable is a measure of an individual’s height in inches. In columns 2 and 4, the dependent variable is a dummy variable which equals 1 if an individual typically wears his or her seatbelt in the car and 0 otherwise. Columns 1 and 2 examine the effect of a cap on damage awards while columns 3 and 4 examine the effect of direct reform. Robust standard errors, clustered by state, are in parentheses. Results are estimated using OLS. All regressions include a full set of individual controls, state fixed effects, and year fixed effects. Statistical significance is denoted as follows: *** p<0.01, ** p<0.05, * p<0.10.

Table 12a: Test of Legislative Endogeneity within Past-Year Scan Incidence and Good Health

VARIABLES	(1) Mammogram	(2) Pap smear	(5) Good health
2-Year Lead	-0.0239	-0.00626	-0.000746
	(0.0165)	(0.00890)	(0.0144)
1-Year Lead	-0.0119	0.00720	-0.00883
	(0.0151)	(0.0132)	(0.0129)
Cap is "on"	-0.0289	0.0129	-0.0125
	(0.0289)	(0.0227)	(0.0113)
1-Year Lag	0.00654	0.0298	-0.00722
	(0.0126)	(0.0194)	(0.00899)
2-Year Lag	0.0140	0.0204	-0.0117
	(0.0122)	(0.0136)	(0.0115)
3+ Year Lag	0.00735	0.0584***	-0.0269
	(0.0287)	(0.0173)	(0.0183)
Observations	465,144	697,974	1,471,893
R-squared	0.121	0.178	0.400

Source: BRFSS, 1987-2000. Columns 1 and 2 exclude men. In columns 1 and 2, the dependent variable is a dummy variable which equals 1 if the individual has had a mammogram or pap smear in the past year and 0 otherwise. The dependent variable in column 3 is a dummy variable which equals 1 if the individual reported being in good, very good, or excellent health and 0 otherwise. Here, the effect of a cap on damage awards is estimated using one and two year leads and one, two, and three or more year lags. The omitted category includes individuals living in states which are three or more years away from enacting tort reform. Robust standard errors, clustered by state, are in parentheses. Results are estimated using OLS. All regressions include a full set of individual controls, state fixed effects, and year fixed effects. Statistical significance is denoted as follows: *** p<0.01, ** p<0.05, * p<0.10.

Table 12b: Test of Legislative Endogeneity within Past-Year Scan Incidence and Good Health

VARIABLES	(1) Mammogram	(2) Pap smear	(5) Good health
2-Year Lead	-0.0234	-0.00536	-0.00241
	(0.0159)	(0.00858)	(0.0140)
1-Year Lead	-0.00988	0.00941	-0.00977
	(0.0141)	(0.0127)	(0.0126)
Direct reform is "on"	-0.0259	0.0123	-0.0130
	(0.0275)	(0.0225)	(0.0112)
1-Year Lag	0.00768	0.0301	-0.00851
	(0.0119)	(0.0190)	(0.00918)
2-Year Lag	0.0132	0.0240*	-0.0119
	(0.0113)	(0.0133)	(0.0110)
3+ Year Lag	0.00687	0.0573***	-0.0234
	(0.0267)	(0.0170)	(0.0183)
Observations	465,144	697,974	1,471,893
R-squared	0.121	0.178	0.400

Source: BRFSS, 1987-2000. Columns 1 and 2 exclude men. In columns 1 and 2, the dependent variable is a dummy variable which equals 1 if the individual has had a mammogram or pap smear in the past year and 0 otherwise. The dependent variable in column 3 is a dummy variable which equals 1 if the individual reported being in good, very good, or excellent health and 0 otherwise. Here, the effect of a direct reform is estimated using one and two year leads and one, two, and three or more year lags. The omitted category includes individuals living in states which are three or more years away from enacting tort reform. Robust standard errors, clustered by state, are in parentheses. Results are estimated using OLS. All regressions include a full set of individual controls, state fixed effects, and year fixed effects. Statistical significance is denoted as follows: *** p<0.01, ** p<0.05, * p<0.10.

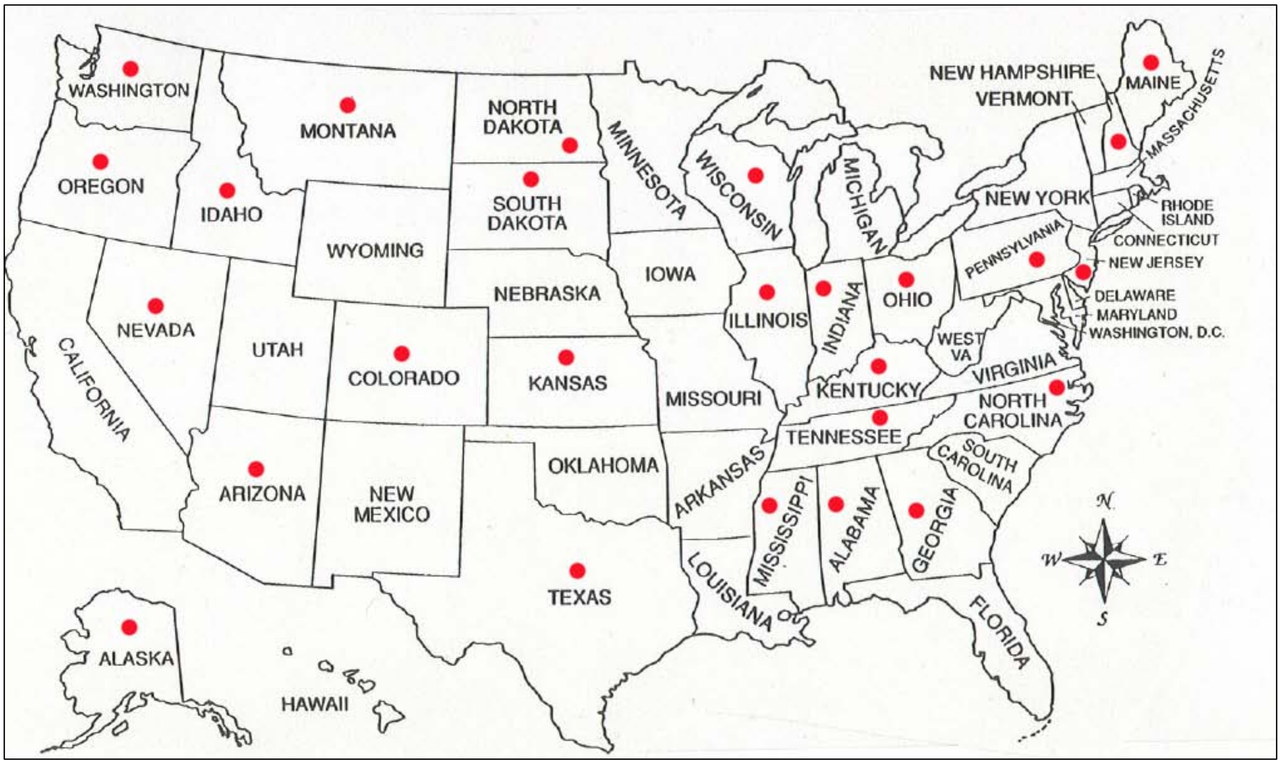
Table 13: Test of Legislative Endogeneity within Cancer-specific Treatments and Health Outcomes

VARIABLES	(1) Surgery	(2) Radiation	(3) Stage 0	(4) Stage 4	(5) Survival time	(6) Death by cancer
2-Year Lead	-0.0439*	0.0122	-0.0191	-0.0105	0.209	-0.0356
	(0.0249)	(0.0273)	(0.0259)	(0.0289)	(1.578)	(0.0257)
1-Year Lead	-0.0589**	0.0286	-0.0152	0.0169	0.650	-0.0349
	(0.0260)	(0.0290)	(0.0271)	(0.0295)	(1.790)	(0.0265)
Direct reform is "on"	-0.0442*	0.0240	-0.00939	0.00988	0.212	-0.0425
	(0.0258)	(0.0289)	(0.0272)	(0.0289)	(1.896)	(0.0262)
1-Year Lag	-0.0445*	0.00576	-0.00510	0.00425	-0.414	-0.0325
	(0.0254)	(0.0287)	(0.0271)	(0.0285)	(1.994)	(0.0257)
2-Year Lag	-0.0379	-0.00211	-0.0172	0.00517	-1.107	-0.0302
	(0.0254)	(0.0283)	(0.0263)	(0.0283)	(1.974)	(0.0255)
3+ Year Lag	-0.0448**	0.00480	-0.0187	0.0202	-1.167	-0.0282
	(0.0218)	(0.0245)	(0.0225)	(0.0244)	(1.936)	(0.0219)
Observations	2,279,108	2,341,872	1,824,864	1,824,864	858,496	2,411,755
R-squared	0.852	0.790	0.830	0.866	0.935	0.809

Source: SEER, 1983-2000. The regressions in columns 1-4 include all tumors diagnosed between January 1983 and December 2000. The regressions in columns 5 and 6 include only those cancer patients who died prior to January 2009. In columns 1-4, the dependent variables are dummy variables which equal 1 if the tumor: required surgery (column 1), required radiation (column 2), was diagnosed at stage zero (column 3), or was diagnosed at stage four (column 4). In column 5, the dependent variable is a continuous measure (in years) of a cancer patient's survival time post diagnosis. In column 6, the dependent variable is a dummy variable which equals 1 if the patient died of cancer and 0 otherwise. All columns estimate the effect of a direct reform. Robust standard errors, clustered by individual and type of cancer, are in parentheses. Results are estimated using OLS. "X" indicates the inclusion of a given type of control. The number of observations varies across dependent variables because of non-response. Statistical significance is denoted as follows: *** p<0.01, ** p<0.05, * p<0.10.

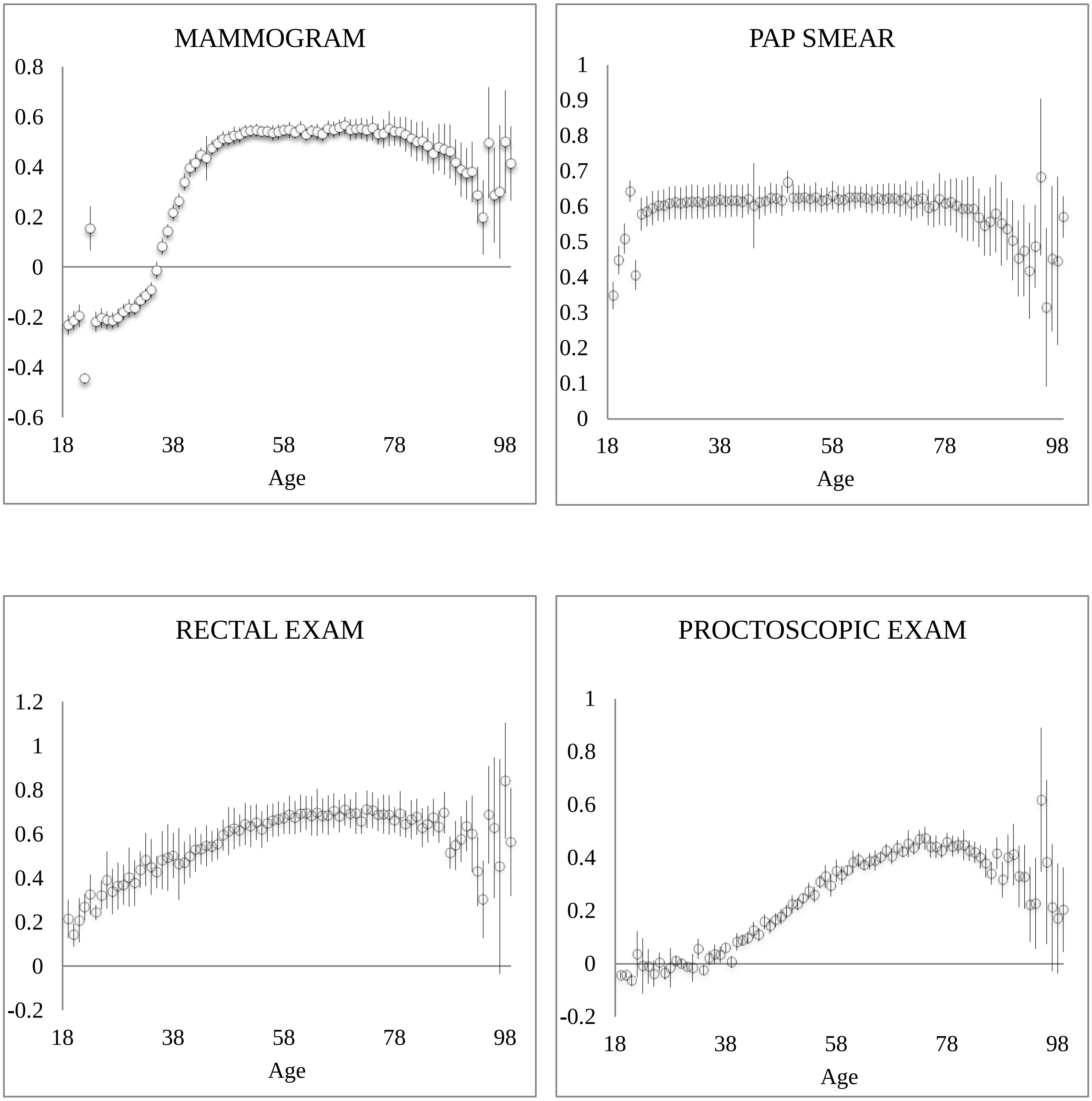
X. APPENDIX

Appendix 1: Tort Reform in the 1980s and 1990s

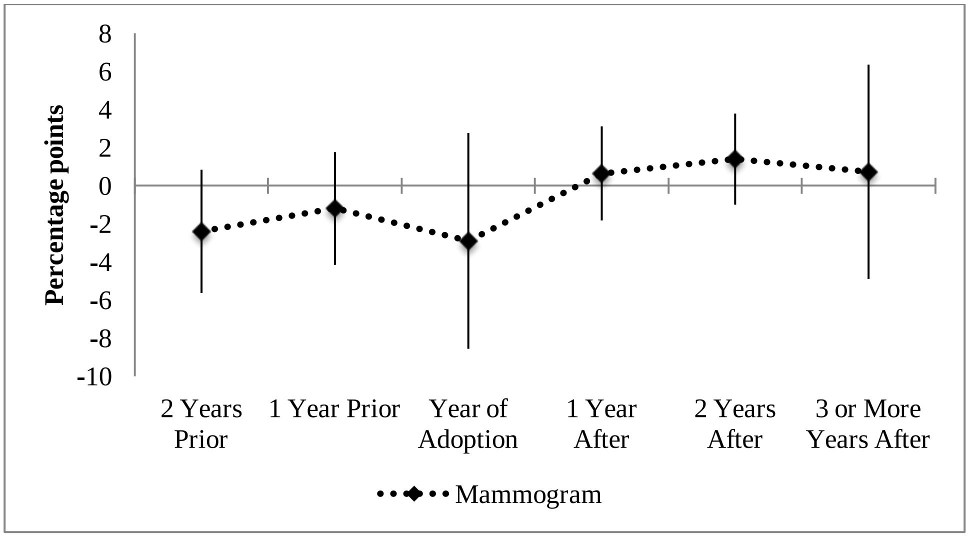


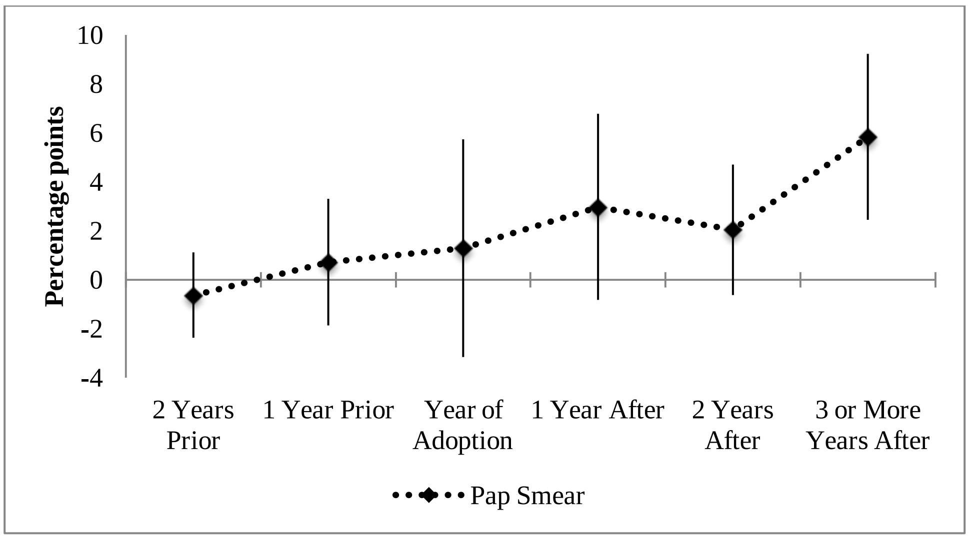
Source: Currie & MacLeod (2008). Dots indicate the location of a state where there was direct tort reform during the 1980s or the 1990s.

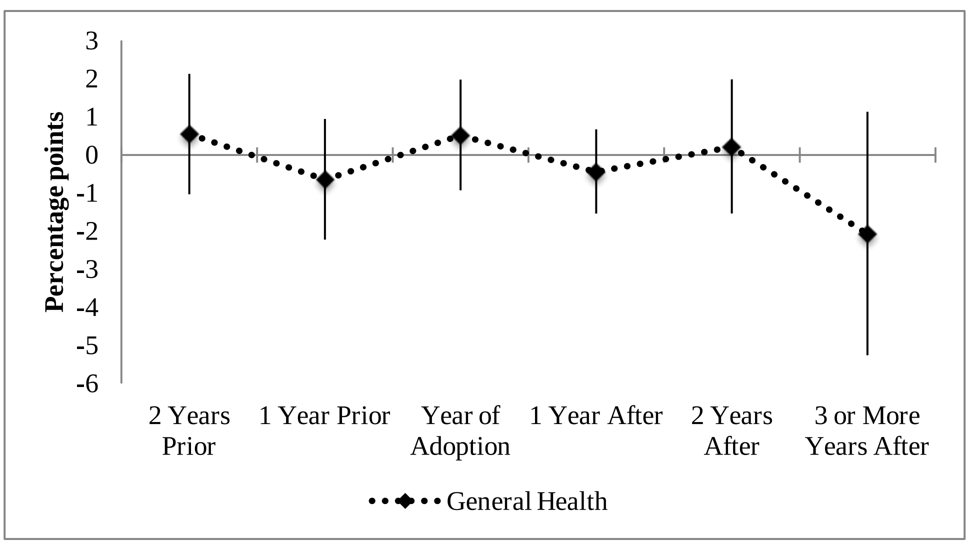
Appendix 2: Graphical representation of the effect of age on the probability of lifetime testing



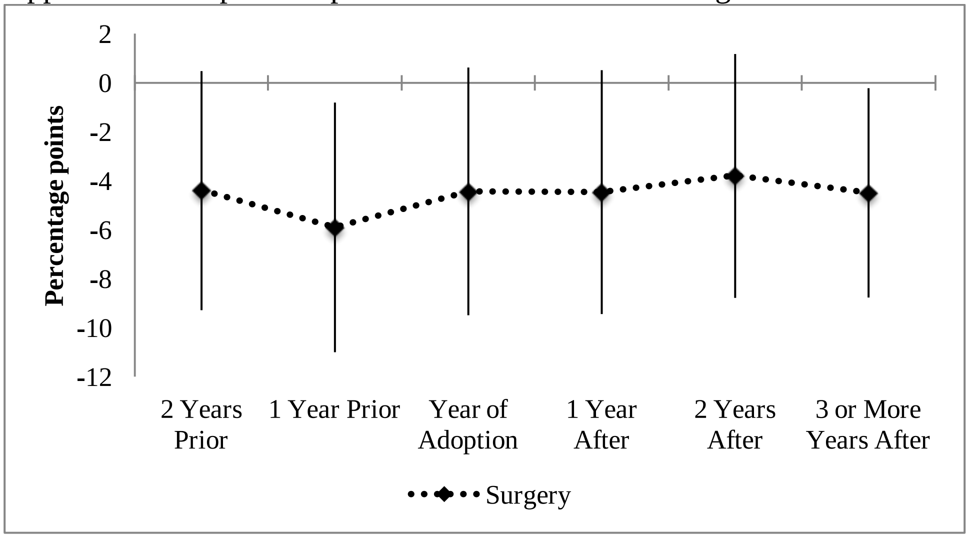
Appendix 3: Graphical representation of the lead and lag coefficients from Table 12a

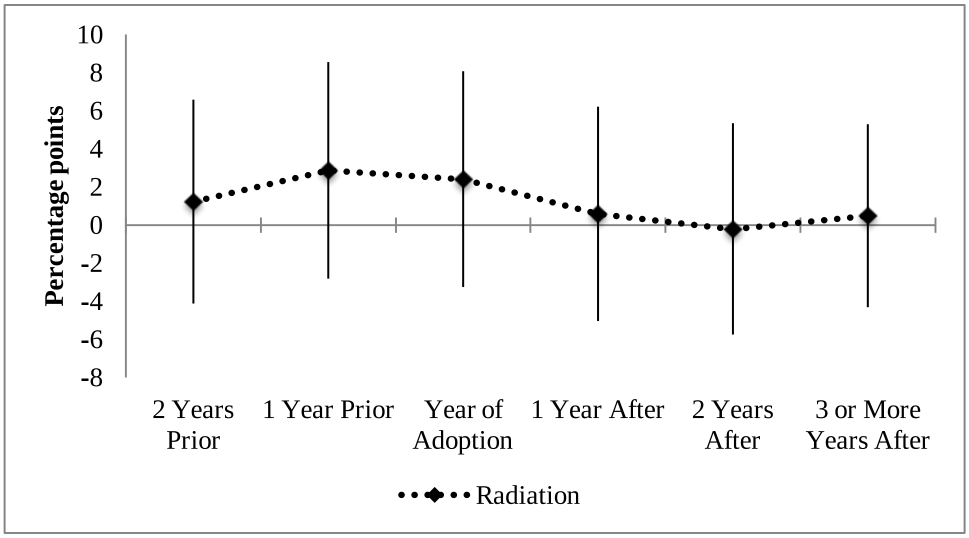


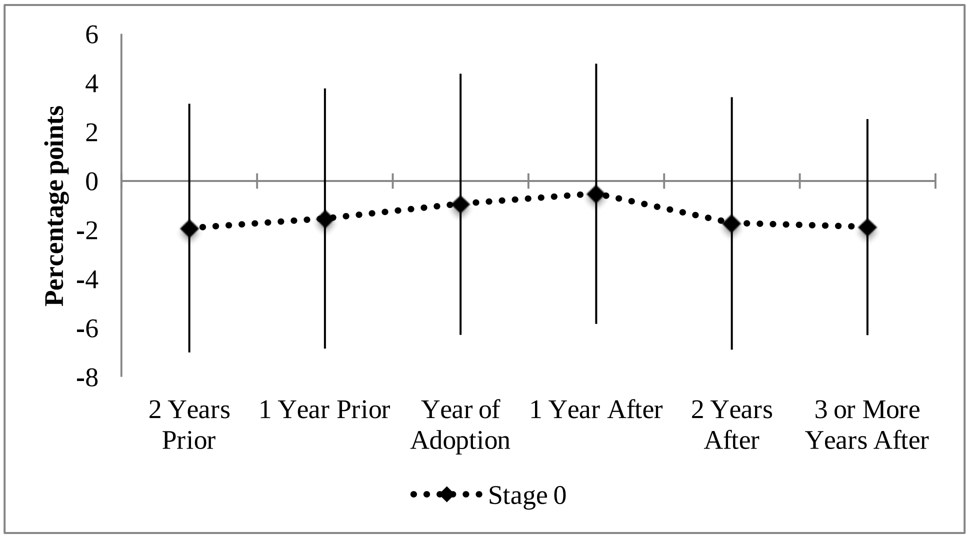


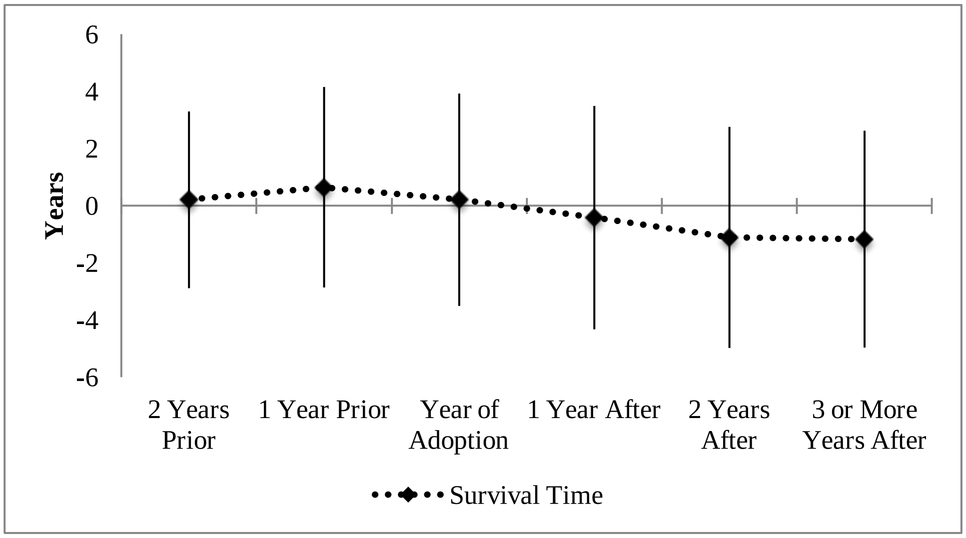


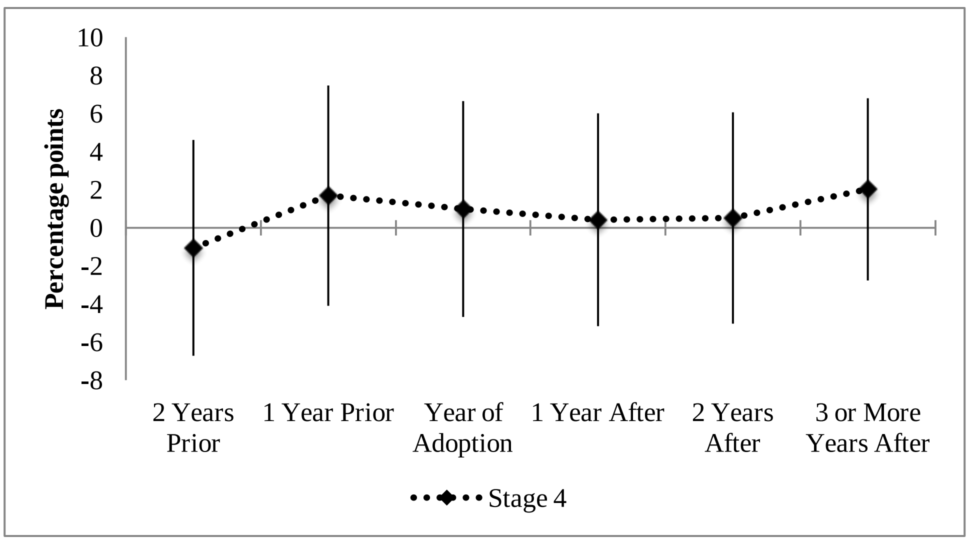
Appendix 4: Graphical representation of the lead and lag coefficients from Table 13

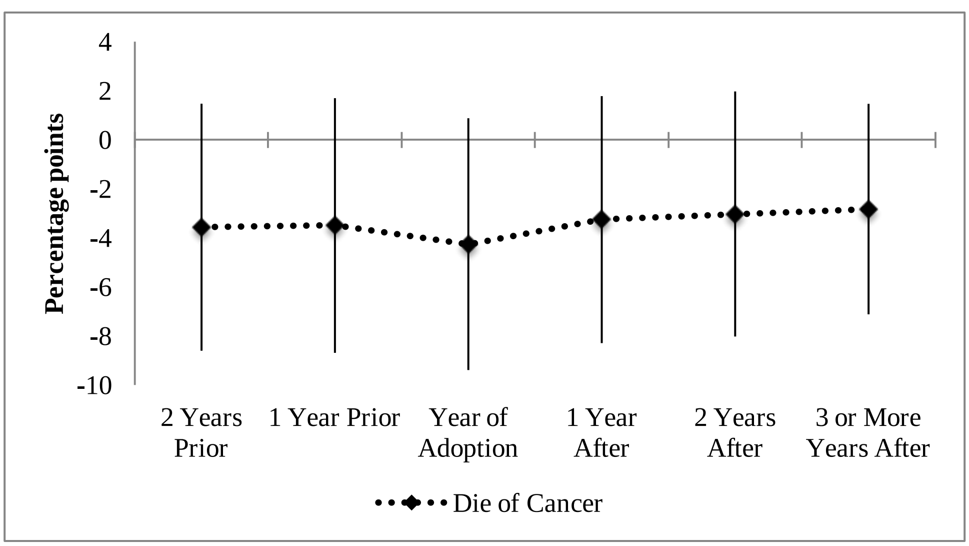







6
4
2
0
-2
-4
-6
Years
2 Years Prior
1 Year Prior
Year of Adoption
1 Year After
2 Years After
3 or More Years After
Survival Time


10
8
6
4
2
0
-2
-4
-6
-8
Percentage points
2 Years Prior
1 Year Prior
Year of Adoption
1 Year After
2 Years After
3 or More Years After
Stage 4


4
2
0
-2
-4
-6
-8
-10
Percentage points
2 Years Prior
1 Year Prior
Year of Adoption
1 Year After
2 Years After
3 or More Years After
Die of Cancer