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## Abstract

While it is the ongoing growth in healthcare spending that has been making headlines, improving and maintaining healthcare quality is a critical goal of healthcare policy. In this dissertation I answer three questions relating to healthcare quality: does physician-hospital financial integration improves healthcare quality; how physician-hospital financial integration improves healthcare quality – in particular does it alter referral patterns, and does it affect EMR usage in physician offices; and whether medical malpractice suits are the results of poor healthcare quality (as opposed to random events).

Physician-hospital financial integration rates have surged over the past few years. The Medicare fee rules permit integrated physicians to charge higher prices for office visits. Meanwhile, industry stakeholders claim that integration may promote care coordination and quality, each of which may in turn improve patient health. Economic theory provides mixed evidence about the consequences of physician-hospital financial integration. I examine the effect of physician-hospital financial integration on health outcomes and spending using patient-year level Medicare data, and physician-year level integration data. I exploit the granularity of this data to estimate the effect of integration on health outcomes more precisely than previous studies have done. I address selection on both patient and physician unobservables by using an instrumental variables model with physician fixed effects. This allows me to identify the causal effects of physician-hospital financial integration. I find that having an integrated primary care physician (PCP) does not significantly affect average mortality risk, but does reduce the risk of less severe adverse health outcomes attributable to conditions that are treatable in primary care settings. I also show that attending an integrated PCP does not increase health care spending. I find that poor patients die more when their PCP is acquired by a hospital but wealthy patients die less.

Physician-hospital financial integration could impact patient health via several mecha-

nisms. I test for changes to referral patterns, EMR installation, evidence of improved care coordination between specialists, and evidence that higher prices reduce access to care. My results indicate that installation of Electronic Medical Records (EMRs) may be playing a role in the beneficial effects of physician-hospital financial integration. Meanwhile, neither inter-specialty coordination, the probability of being referred, nor referral concentration appears to be playing a role. I also show that the higher prices associated with integrated PCPs do not reduce access to primary care. My inpatient referral results support the hypothesis that physicians are redirecting their patients to their hospital-owner after they are acquired. However, the Cardiology referral results do not appear to support this hypothesis. I test whether PCPs who are acquired by hospitals redirect their wealthy patients to their hospital-owner and their poor patients away from their hospital-owner. Although my point estimates appear to support this hypothesis, the standards errors are too large to confirm it.

Advocates of malpractice reform often argue that most malpractice claims are unrelated to the quality of the care provided. In a coauthored chapter, Bernard Black, Zenon Zabinski and I study the connection between hospital adverse events and malpractice claim rates in the two states with public data sets on medical malpractice claim rates: Florida and Texas. We use Patient Safety Indicators (PSIs), developed by the Agency for Healthcare Research and Quality, to measure rates for 17 types of adverse events. Hospitals with high rates for one PSI usually have high rates for other PSIs. We find a strong association between PSI rates and malpractice claim rates with extensive control variables and hospital fixed effects (in Florida) or county fixed effects (in Texas). Our results, if causal, provide evidence that malpractice claims leading to payouts are not random events. Instead, hospitals that improve patient safety can reduce malpractice payouts.



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# Chapter 1

## The effect of physician-hospital financial integration on health outcomes and spending

### 1.1 Introduction

Historically, the US medical system was quite fragmented, with few physicians being formally linked to hospitals. Most physicians operated within their own business entity, known as a “physician practice.” In recent years, hospitals have purchased many physician practices and employed the physicians. When a hospital buys a physician practice this is known as “physician-hospital financial integration.”

Under the Medicare fee schedule, hospital-owned physician practices can be classified as hospital outpatient departments and are therefore allowed to charge patients a facility fee. This leads to patients paying higher prices for routine visits (Neprash et al., 2015). However, industry stakeholders claim that physician-hospital financial integration has benefits. These

purported benefits include better coordination between primary care physicians<sup>1</sup> (PCPs) and hospitals, and improved treatment at the primary care level (Burns et al., 2013), both of which may in turn reduce hospital admissions and emergency department visits.

Some elements of economic theory lend support to the claim that physician-hospital integration could improve patient health. For instance, financial integration may promote joint investments in care coordination by internalizing externalities. Furthermore, shifting the “business aspects” of a physician practice to a hospital may allow physicians to specialize in patient care, which could result in higher quality primary care. Meanwhile, other aspects of economic theory suggest reasons why physician-hospital financial integration could negatively impact patient outcomes. First, higher fees for physician office visits may deter patients from receiving preventative care, leading to worse patient health. Second, employing physicians will attenuate their incentives to provide high quality care because the financial benefits of patient loyalty will no longer accrue solely to the physician. Whether physician-hospital financial integration ultimately improves patient health outcomes is an empirical question.

Physician-hospital financial integration may lead to lower health care spending if it reduces utilization sufficiently, in spite of the higher prices associated with integrated physicians. Consequently, the net impact of physician-hospital financial integration on total health care spending must also be determined empirically.

Evidence on the impact of physician-hospital financial integration on patient outcomes is lacking. There have been numerous studies documenting *correlations* between physician-hospital financial integration and health outcomes (e.g. Madison, 2004; McWilliams et al., 2013). The studies providing the strongest evidence about the impact of physician-hospital financial integration on health outcomes have data limitations that prevent their results from being conclusive (Cuellar & Gertler, 2006; Carlin et al., 2015). Existing empirical evidence

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<sup>1</sup>Primary care physicians are doctors who are usually patients’ first and more frequent point of contact with the health care system. They are usually generalists, in that they do not focus on particular diseases or bodily systems. They typically do not have direct contact with patients during hospital stays.



indicates that physician-hospital financial integration increases health care spending (e.g. Baker et al., 2014; Capps et al., 2015; Neprash et al., 2015).

In this paper I use reduced form causal inference methods to estimate the effect of physician-hospital financial integration (henceforth abbreviated to integration) on health outcomes and health care spending. My health outcome variables are indicators for death, an unplanned hospital admission, and an appropriate emergency department visit. My explanatory variable of interest is whether a patient’s primary care physician (PCP) is integrated with a hospital. I define a patient’s PCP as their most frequently attended physician for outpatient evaluation and management services. My sample is patients who have chronic conditions for which proper treatment in a primary care setting should reduce the need for hospital care. Such conditions are known as “ambulatory care sensitive chronic conditions” (ACSCCs).

I use beneficiary-year level data from the Medicare Fee-For-Service<sup>2</sup> (FFS) 5% sample to determine which beneficiaries have ambulatory care sensitive chronic conditions, each beneficiary’s health outcomes and health care spending for the year, and the identity of each beneficiary’s primary care physician in each year. I use physician survey data from the company SK&A to ascertain which physicians are integrated in each year.

I regress each outcome measure on an indicator for whether the patient’s PCP is integrated with a hospital. I use an extensive set of control variables. An ordinary-least-squares estimate of the coefficient on the integration status of the patient’s PCP would be biased due to selection on unobserved patient and physician characteristics. I use a physician fixed effects model with instrumental variables to address these two sources of bias. I have two instruments each of which relies on a different type of patient behavior. My first instrument

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<sup>2</sup>Under Medicare Fee-For-Service insurance, also known as traditional Medicare, Medicare pays providers a fee for each service the provider performs. This is in contrast to Medicare Advantage plans where Medicare pays a private insurer an amount based on the number of patients enrolled in that insurer’s Medicare Advantage plans and the private insurer selectively contracts with providers.

for the current integration status of the beneficiary's current PCP is the current integration status of the beneficiary's original PCP. To execute regressions with this instrument I exclude beneficiaries whose original PCP was already integrated when the beneficiary first chose them. Identification relies on people's tendency to keep the same primary care provider over time. My second instrument for the integration status of the beneficiary's PCP is the average integration status of PCP's serving the patient's ZIP code, excluding the patient themselves from the average. Identification relies on patients aversion to travel for primary care. The area average instrument allows me to include patients who initially chose an integrated PCP in the sample, but has less variation than the original PCP instrument.

I show that attending a PCP whose practice is owned by a hospital reduces unplanned admissions and appropriate emergency department visits due to conditions that are treatable in primary care settings. I find that attending an integrated PCP does not have a significant impact on average mortality rates. In contrast to existing literature, I find that integration does not increase total health care spending. I believe this can be attributed to several factors including the public sector setting, and the subsample of patients that are the focus of my study.

Economic theory suggests a number of mechanisms via which physician-hospital financial integration *may* change the delivery of care. There is no evidence about which of these mechanisms are actually operating in practice. Several mechanisms predict heterogeneous effects of integration over particular patient characteristics. I test these predictions in my data. I find that the benefits of integration are no larger and may even be smaller for patients whose underlying characteristics predict that they will see many specialists. This finding implies that improved care coordination between specialists is not driving the health improvements that result from physician-hospital financial integration, which suggests that improved primary care quality or better discharge management may be playing a role. I show that integration induced reductions in unplanned admissions and emergency department vis-

its attributable to primary care treatable conditions are unrelated to ZIP code income. This suggests that poor patients are still able to access primary care in spite of the higher costs associated with integrated physicians. However, I find suggestive evidence that integration induces higher mortality rates among the poor and lower mortality rates among the wealthy, which suggests that integration with a hospital may induce physicians to selectively refer patients to high or low quality hospitals based on the patient's income.

This paper makes several contributions to the literature. First, I use better data than previous studies on the effect of physician-hospital integration on health outcomes; my data is more representative than one study and more granular than the other studies. Second, I account for both patient and physician selection, something previous studies of the effect of physician-hospital integration on health outcomes did not do. Third, I examine heterogeneous effects over patient characteristics and in doing so provide some evidence about the mechanisms via which integration may be affecting outcomes.

The paper proceeds as follows: In section 1.2, I discuss previous empirical papers on the impact of integration on health outcomes and define ambulatory care sensitive chronic conditions. I outline my data sources, the construction of my variables, and the distributions of key variables in section 1.3. In section 1.4, I outline my estimation strategy in detail. I present and discuss my main results in section 1.5. In section 1.6, I outline the mechanisms via which physician-hospital financial integration may affect health outcomes. I use these mechanisms to predict heterogeneity in the effect of integration over patient characteristics. I then test these predictions in the data. In section 1.7, I examine the timing of integration's effects. In section 1.8, I show that health system management or ownership of physician practices does not have the same effect as hospital ownership of physician practices. I perform numerous robustness checks of my main results in section 1.9. I discuss the main points of my paper in section 1.10 and conclude in section 1.11.

## 1.2 Background

### 1.2.1 Previous literature on the impact of physician-hospital financial integration

Little attention has been devoted to the impact of physician-hospital financial integration on patient outcomes. Existing studies about the effect of integration on patient outcomes do not account for one or more important sources of bias. Furthermore, many studies have data limitations or limited scope. The literature examining the effect of integration on health care spending is much more extensive. However, many studies of the effect of integration on health care spending only have hospital data, and hence are not able to capture outpatient spending. Studies with individual level or outpatient data focus on privately insured patients.

Most studies investigating the impact of physician-hospital financial integration on patient outcomes do not account for the possibility that physician practices that are acquired may be better or worse than those that are not acquired (e.g. Madison, 2004; McWilliams et al., 2013). Consequently, the estimates of the effect of integration on health outcomes in these studies may be biased. Two studies take selective acquisition of physicians into account, Cuellar & Gertler (2006); and Carlin et al. (2015). A third study, Baker et al. (2014), also accounts for selective acquisition of physicians. Technically, Baker et al. (2014) investigates the effect of integration on hospital utilization, not on health outcomes. However, one of my health outcome measures is related to hospital utilization.

Cuellar & Gertler (2006) find no significant impact of integration on patient outcomes for patients insured under Fee-For-Service contracts. However, they do not account for Fee-For-Service patients choosing their providers. Patients may possess unobserved characteristics that both make the patients more inclined to choose an integrated hospital, and tend to result in poor health outcomes (e.g. forgetful, poorer than average underlying health). Consequently, ignoring patient selection may mask a beneficial effect of integration on patient

health outcomes. Cuellar & Gertler (2006) only have inpatient data. Anticipated benefits of integrated care include better health reducing the need for hospital care. Unfortunately, their limited data prevents them from examining whether this occurs. They also have very coarse measures of integration: indicators for the type of organization the hospital belongs to (fully integrated hospital organization, closed physician hospital organization, open physician hospital organization, or independent practice association). The coarseness of the integration measures may be responsible for them finding few significant results on health outcomes for Fee-For-Service patients. Patients with chronic conditions interact more frequently with the health care system than the average patient benefits. Consequently, it seems likely that patients with chronic conditions benefit more from care coordination than the average patient. Cuellar & Gertler (2006) pool these patients with other patients that likely benefit little from care coordination. This pooling could contribute to them finding no significant effect of integration on patient health outcomes.

Carlin et al. (2015) investigate the effect of three clinic acquisitions by two hospital-owned integrated delivery systems. The findings from these few acquisitions may not be generalizable and are sometimes in conflict with one another. Carlin et al.'s (2015) data comes from a private insurer and covers a combination of privately insured and Medicaid Managed Care patients. Economic theory predicts that the effect of integration may differ between patients covered by Fee-For-Service and capitated insurance plans. Carlin et al. (2015) do not provide separate estimates for each type of insurance plan, nor do they include interaction terms that would allow the identification of a difference in the effect of an acquisition on people covered under different types of insurance plans. Like Cuellar & Gertler (2006), they do not account for patient selection of physicians, in spite of most patients in their sample having insurance plans that permit the patients to choose providers.

Baker et al. (2014) uses county-year level data and county fixed effects. They find weak evidence that increasing the share of integrated hospitals in the county decreases utilization.

However, they are unable to exclude the possibility that integration is increasing in response to declining hospital volumes. They are also unable to exclude the explanation that patients may begin traveling across county lines for hospital care in response to the increasing level of integration in their county.

This paper makes several improvements over existing literature on the effect of physician-hospital integration on health outcomes. First, I have better data; it is more granular than Cuellar & Gertler’s (2006) and Baker et al.’s (2014) data, and has broader coverage than Carlin et al.’s (2015) case study. Second, I account for patient selection in addition to physician selection. Third, I examine heterogeneity in the effect of physician-hospital financial integration over patient characteristics, something none of these three previous studies have done. I also use the heterogeneous effects of integration to help explain which mechanisms are causing the health effects of integration.

### **1.2.2 Ambulatory care sensitive chronic conditions**

Chronic conditions are diseases that are ongoing in time, in contrast to acute conditions, which are short lived. Chronic conditions can lead to acute episodes. For example asthma is a chronic condition whereas an asthma attack is acute. “Ambulatory care sensitive conditions” also known as “primary care sensitive conditions” are diseases for which good outpatient care can reduce the probability that a patient will need hospital care (Billings et al., 1993; Agency for Healthcare Research and Quality, 2013).

The Agency for Healthcare Research and Quality (AHRQ) has developed a set of “Prevention Quality Indices” (PQIs). The PQIs measure admissions or health care complications due to ambulatory care sensitive conditions and are designed to be proxies for quality of, and access to, community based care (Agency for Healthcare Research and Quality, 2013). The PQIs were developed following an extensive literature search (Davies et al., 2001) and are based on a variety of studies that link lack of primary care access to increased admissions

due to particular conditions (e.g. Bindman et al., 1995; Weissman et al., 1992).

Following the AHRQ PQI for chronic conditions (PQI 92), I include asthma, chronic obstructive pulmonary disease (COPD), diabetes, heart failure, and hypertension as ambulatory care sensitive chronic conditions (ACSCCs) in my study (Agency for Healthcare Quality and Research, 2015). Unlike the current version of the AHRQ chronic conditions PQI (version 5.0), I do not include angina. The AHRQ recently announced that they will be removing angina from their PQIs in version 6.0 due in part to concerns that physicians have been coding admissions due to angina under coronary artery disease (the underlying condition) in response to the publication of the PQIs (Agency for Healthcare Research and Quality, 2016).

### 1.3 Data sources and variable construction

To test the relationship between PCP-hospital integration and patient outcomes, I need data that allows me to construct several variables. First, I require data on patient outcomes. Second, I need to determine the identity of each patient's PCP. Third, I must ascertain whether the PCP is integrated with a hospital. Finally, I need data on patients' health conditions, patients' demographic characteristics, and the characteristics of the patients' communities.

My data on patient outcomes, the identity of each patient's PCP, and patient level control variables come from the 5% sample of Medicare claims data. I construct an indicator of whether each physician is integrated with a hospital using data from a physician survey conducted by the company SK&A. My community level control variables are at the ZIP code level and are based on data from the Internal Revenue Service, the National Bureau of Economic Research, and the American Hospital Association survey.

### 1.3.1 Medicare 5% sample

My patient level data is constructed from Medicare claims data. I have a 5% sample of Medicare Fee-For-Service<sup>2</sup> patients. I have all claims for these patients in outpatient facilities, inpatient facilities, skilled nursing facilities, and physician office settings over 2004-2013.<sup>3</sup> Patients have encrypted identifiers that allow me to track them across health care settings, and over time. The data contain patient demographic information, patient ZIP codes, patient death date (if applicable), diagnosis codes, procedure codes, provider identifiers, and spending amounts. The data do not include claims for home-health-care, pharmaceuticals used outside hospitals, or medical equipment.

I limit my sample to patient-years where the patient was enrolled in Medicare Parts A and B for the whole year, since I can only see hospital claims when a patient is covered by Medicare Part A, and I can only see outpatient claims when a patient is covered by Medicare Part B. I also limit the sample to patient-years where the patient was 68 or over at the beginning of the year, since I need one to three years of data to identify chronic conditions from the claims data<sup>4</sup>, and people who are eligible for Medicare when they are under 65 often are substantially sicker than most of the Medicare population.

### 1.3.2 Identifying chronic conditions

I identify which beneficiaries have chronic conditions using the Chronic Conditions Data Warehouse (CCW) algorithm (2014) as best as I am able.<sup>5</sup> The CCW algorithms use diagnosis and procedure codes from the previous one to three years of claims data to determine whether a patient has the chronic condition. Depending on the condition and the data file

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<sup>3</sup>The formal names of the files I use are the Medicare Beneficiary Summary File, the Outpatient Research Identifiable File, the MedPAR Research Identifiable File, and Carrier Research Identifiable File.

<sup>4</sup>The number of claims required varies by condition and setting.

<sup>5</sup>Since I do not have access to the Home Health or the Durable Medical Equipment Research Identifiable Files, I am unable to perfectly replicate the CCW 2014 algorithms.



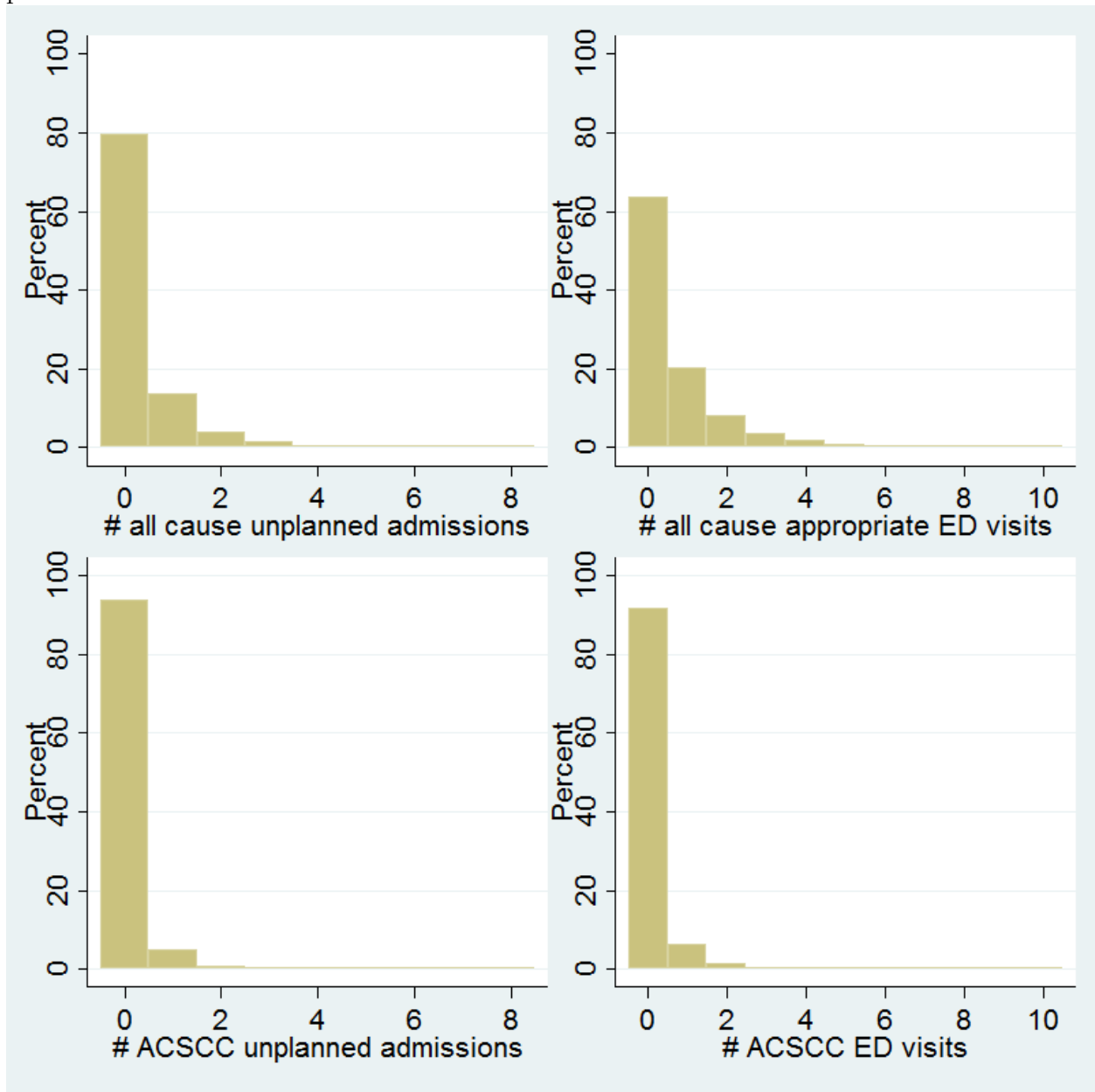
the algorithm may require multiple instances of a diagnosis or procedure for the condition, to classify the beneficiary as having that condition.

### 1.3.3 Health outcome variables

My indicators of poor health outcomes are death, unplanned hospital admissions, and appropriate emergency department (ED) visits. I use two versions of these indicators where possible. The first version of the indicators includes all cause poor health outcomes. The second version of the indicators includes poor health outcomes that can be directly attributed to one of the AHRQ ambulatory care sensitive chronic conditions (ACSCCs) that the patient was identified as having based on previous years' claims. I am unable to construct an indicator for deaths due to ACSCCs because the Medicare data does not contain cause of death. All cause measures of health outcomes provide noisy indications of the primary care experience, since they capture events that primary care could not possibly affect such as emergency department visits due to car accidents. The AHRQ ACSCC health outcome measures are less noisy indicators of the primary care experience. However, they underestimate negative health outcomes attributable to primary care, since the AHRQ ACSCC list is not an exhaustive list of ACSCCs. Although I have counts of unplanned admissions and appropriate ED visits, I focus on indicator variables, since most of the variation is on the extensive margin (see Figure 1.1).

I treat an admission or ED visit as “due” to an ACSCC if the primary diagnosis code is either directly for one of the patient's ACSCCs, or is a common consequence of one of the patient's ACSCCs. For instance, common complications of hypertension include heart attacks, heart failure, strokes, and kidney disease (James et al., 2013) so if a patient with hypertension is admitted with a primary diagnosis of hypertension, a new heart attack, a new stroke, new heart failure, or new kidney disease, I count this as an admission due to hypertension. For asthma and COPD I follow AHRQ's PQI algorithm in considering some

Figure 1.1: Distributions of counts of unplanned admissions and counts of emergency department visits



All counts  $>8$  for the admission variables and all counts  $>10$  for the ED visit variables have been censored in accordance with Medicare rules which prohibit the display of cell sizes with  $\leq 10$  beneficiaries.

non-primary diagnosis codes. For a full outline of the criteria for an admission, or ED visit being counted as “due to” each chronic condition refer to Table A.1 in the Appendix.

Admissions via the ED are almost certainly due to an adverse health outcome, whereas admissions not via the ED may be influenced by the opinion or incentives of the PCP. Since I want to capture the effect of integration on health outcomes, as opposed to utilization, I focus on admissions via an ED or an urgent care clinic.<sup>6</sup> I refer to these as “unplanned admissions.” In section 1.9.1 I show that the results are not driven by the choice to focus on unplanned admissions.

Visits to an ED may be driven by a genuine need for ED services, or by difficulty getting an appointment with a primary care provider. To best capture health outcomes, I focus on appropriate use of the ED. Billings et al. (2000) developed an algorithm for identifying appropriate use of EDs. They had a panel of doctors examine patient medical records from visits to a New York hospital. They classify an ED visit as appropriate if either the patient was admitted to the hospital, or the patient needed facilities that are available at EDs but not in primary care settings. They produced tables that summarize, for each diagnosis code, the percentage of patients that actually needed an ED<sup>7</sup>. Like Billings et al. (2000) I treat all ED visits resulting in an admission as appropriate. As such, unplanned admissions represent a subset of appropriate ED visits. I classify the remaining ED visits as inappropriate if all diagnosis codes from the visit had a 0% chance of being appropriate use of the ED according to the Billings data. Virtually all of the diagnosis codes indicating a poor health outcome due to one of the AHRQ ACSCCs constitute appropriate use of the ED according to the Billings study. Consequently, I do not distinguish between appropriate and total use of the ED for ED visits due to ACSCCs.

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<sup>6</sup>In theory urgent care clinics visits may be substituted for certain types of ED visits. Among my sample however, the use of urgent care clinics is extremely low relative to the use of EDs (the correlation between an indicator of admissions via an ED and an indicator of admissions via the ED or on the same day as an urgent care visit is 0.9996).

<sup>7</sup><http://wagner.nyu.edu/faculty/billings/nyued-background>

The average values of the outcome variables in the full sample are given in Table 1.1 column 1.

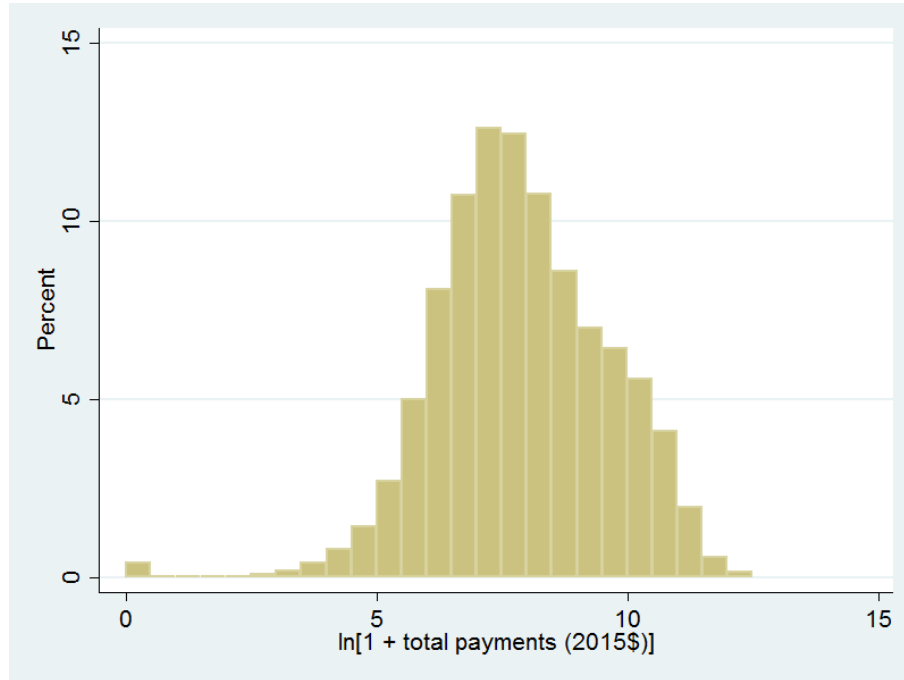
Table 1.1: Average outcome variables in the full and restricted samples

Sample	Full	Original PCP not initially integrated
100 x 1[all cause unplanned admission]	19.567 (39.672)	19.632 (39.721)
100 x 1[ACSCC unplanned admission]	6.016 (23.779)	6.071 (23.879)
100 x 1[all cause appropriate ED visit]	35.623 (47.889)	35.495 (47.850)
100 x 1[ACSCC (appropriate) ED visit]	8.206 (27.446)	8.229 (27.480)
100 x 1[all cause death]	3.555 (18.517)	3.618 (18.675)
ln[1 + total spending 2015\$]	8.308 (1.415)	8.315 (1.416)

Figures in brackets are standard deviations.

### 1.3.4 Spending outcome variable

The spending outcome variable is the logarithm of 1 plus total spending (in 2015 dollars) on the beneficiary's health care. To determine spending I add total payments by Medicare, total payments by primary payers other than Medicare, the beneficiary's deductible liability, and beneficiary's coinsurance liability. This total spending measure includes all inpatient services, outpatient services, physician services, and various other services such as testing captured in the Carrier file but excludes home-health-care, pharmaceuticals administered outside a hospital, and durable medical equipment, since I was unable to obtain these Medicare claim files. The distribution of the total spending variable is shown in Figure 1.2. The average value is given in Table 1.1, column 1 in the final row. The average value corresponds to \$4,055.

Figure 1.2: Distributions of  $\ln[1 + \text{total payments (2015\$)}]$ 

All values of the variable  $>12.5$  have been censored to 12.5 in accordance with Medicare rules which prohibit the display of cell sizes with  $\leq 10$  beneficiaries.

### 1.3.5 Physician integration data

The company SK&A conducts surveys of all office based physician sites in the United States. Each physician office location is contacted approximately once every six months and the data is archived in December of each year. SK&A data is the most reliable publicly available source of data that both records which physicians are owned by hospitals, and includes physician identifiers. The SK&A data also contains a site ID (that can be used to link all physicians practicing at the same site), and the number of physicians practicing at that site.

Due to the substantial expense of the SK&A data, I first used the Medicare data to determine which specialties were most likely to serve as primary care physicians to patients with one or more of the AHRQ ACSCCs. Most of the patients in my sample use Family Practitioners, General Practitioners, or Internal Medicine specialists as primary care physicians



### 1.3.6 Primary care physician integration variable

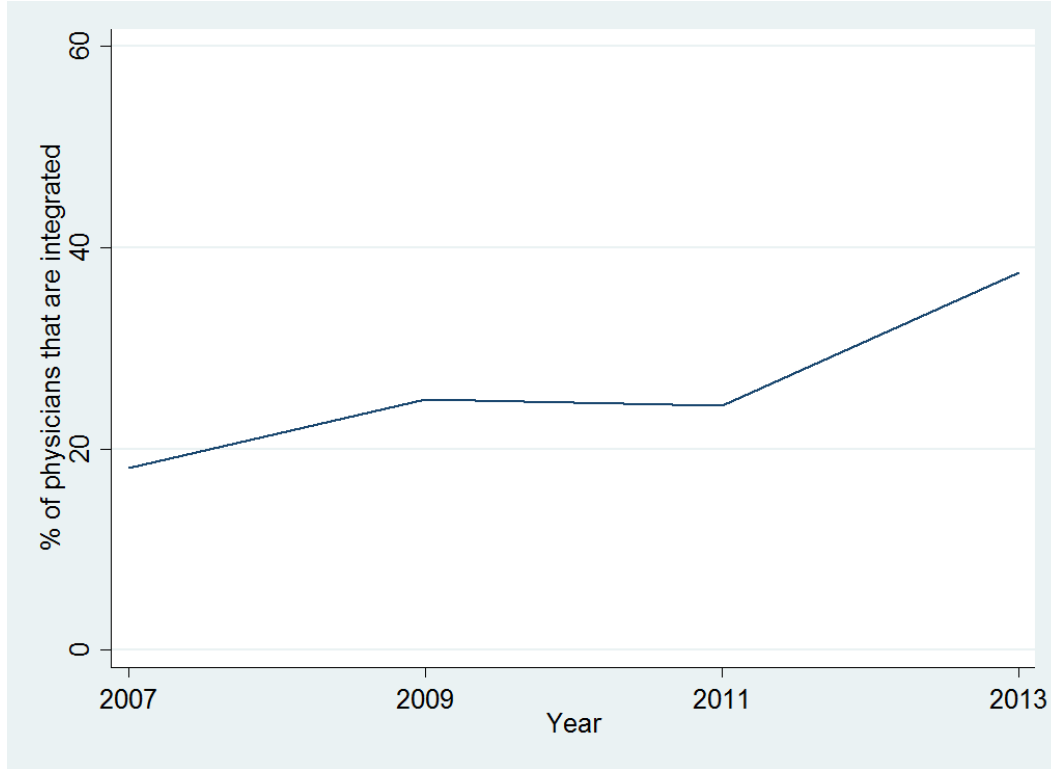
My explanatory variable of interest is an indicator of whether the beneficiary's primary care physician (PCP) is integrated with a hospital. I designate the outpatient physician most frequently attended by a beneficiary in a given year for evaluation and management services as the beneficiary's PCP for that year. I treat beneficiaries with two or less outpatient evaluation and management claims with a particular physician as not having a PCP, and hence exclude them from the sample. (In section 1.9.6 I examine the sensitivity of my results to this threshold.) I use the SK&A data to determine whether the PCP is integrated with a hospital.

The SK&A data I have are as at December in 2007, 2009, 2011, and 2013. However, they were collected over the period from approximately May to December of those years. Consequently, I treat the values of the SK&A variables as at December of year  $x$  as the values of the SK&A variables for during year  $x$ .

Figure 1.4 focuses on physicians who are in the SK&A data in all years. The percentage of these physicians that are owned by a hospital increases between 2007 and 2009, decreases slightly from 2009 to 2011, and increases between 2011 and 2013. This general pattern is similar across all states in the data. Physicians rarely separate from hospitals once integrated. Less than 3% of physicians in the sample ever become independent having been previously integrated and this affects c. 2% of beneficiaries in the sample.

A limitation of using the SK&A data to identify which sites are integrated is that the survey response specifies either the name of the hospital that owns the site or is blank. When the response is blank it is not clear whether the site is independent or whether the survey question was not answered. To combat this I assume that a site, once integrated, remains integrated: that the integration indicator is weakly increasing. 90% of sites have weakly increasing integration indicators based purely on the data. Among those that do not, some

Figure 1.4: Percentage of physicians in the sample in all years that are integrated



list a particular hospital as their owner in one year have a blank in the following year of data and then list that same hospital as their owner in the year of data after that. Consequently, I believe the assumption of weakly increasing site integration status is reasonable. (I later show that my results are robust to dropping this assumption - see section 1.9.4) Though I assume that sites have weakly increasing integration status, I do not make any such assumption about physicians as they may choose to move sites when their site integrates.

I interpolate the values of the integration variable for even years of data where possible. Let  $x$  be an even year. If the value of the integration indicator for a particular physician is the same in years  $x - 1$  and  $x + 1$  then I set the value of the integration indicator in year  $x$  to this value. If the integration indicator takes different values in years  $x - 1$  and  $x + 1$  I treat the integration indicator for this physician as missing in year  $x$ . (In section 1.9.4 I



show that limiting my analysis to odd years somewhat weakens my results but the signs and orders of magnitude of the estimated effects are unchanged).

Some physicians practice at multiple sites. Conflicting integration status between sites is rare. When integration status conflicts between multiple sites attended by the same physician I drop the physician from the sample.

### 1.3.7 Control variables

I use age, sex, and race indicators from the Medicare data. I also use indicators for chronic conditions from the Chronic Conditions Data Warehouse algorithm.<sup>10</sup> Since people with multiple chronic conditions generally have worse health outcomes than people with few chronic conditions (Friedman et al., 2009), I include a count of chronic conditions in addition to the individual condition indicators.

The financial resources available to beneficiaries may be important for their health outcomes, and may influence their tendency to choose an integrated or independent physician. I do not have individual income data so I include two proxies for income. First, since people of similar means are often geographically clustered, I use the logarithm of average household income (in thousands of 2015 dollars) in the beneficiary's 5-digit ZIP code. I obtain ZIP code-year level income data from Internal Revenue Service (2014) for 2006-2013. My other proxy for income is an indicator for Medicaid covering part of the beneficiary's Medicare premium. Medicaid covers part of the beneficiary's premium when the beneficiary's income is less than 100% of the federal poverty level (Centers for Medicare and Medicaid Services, 2016). As one would expect,  $\ln(\text{ZIP code income})$  is negatively correlated with the indicator for Medicaid covering part of the Medicare premium. The correlation coefficient is -0.14.

Since chronic conditions are generally identified based on the previous two years of Medi-

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<sup>10</sup>I do not include Alzheimers Disease as it is also included in the condition "Alzheimer's Disease and Related Disorders or Senile Dementia"

care claims, it is possible that coding practices of prior PCPs could influence whether a patient is included in the sample. For instance, an overzealous coder may code a diagnosis of diabetes when in fact the patient was only tested to see if they had diabetes. The chronic condition algorithms often require multiple occurrences of diagnoses before a patient is counted as having the condition; however, requiring multiple occurrences of a diagnosis code may not entirely address the issues caused by over-coding. If over-coding is more prevalent among physicians who have a high propensity to integrate, patients with integrating PCPs may have been incorrectly coded as having a chronic conditions that made them eligible for the sample. Hence these patients would be healthier than they appear in the data. One would expect overzealous coders to have more diagnosis codes recorded per claim. I find the average number of diagnoses on each claim for each physician-year in the 5% Medicare sample. Then for each patient in my sample, take the maximum of this value from their PCPs over the previous 2 years. I include this variable as a control in the regressions.

Where possible I include physician or physician practice characteristics. I include the logarithm of the total number of physicians at the physician's site, since this is highly related to the physician's propensity to integrate. The total number of physicians at each physician's site comes directly from an SK&A survey question in odd years. In even years, if the physician practiced at the same site in the two adjacent odd years, I take the average of the number of physicians at the site in these two years. If the physician did not practice at the same site in the two adjacent odd years, I treat the number of physicians at the site as missing. I also include the logarithm of the total number of 5% sample Medicare patients the physician sees during the year.

## 1.4 Estimation strategy

My data is at the beneficiary-year level. I focus on beneficiaries who have chronic conditions that, if treated properly in a primary care setting, should not require treatment in a hospital. These conditions are called ambulatory care sensitive chronic conditions (ACSCCs). My health outcomes are indicators for death, unplanned hospital admissions, and appropriate emergency department visits. My cost outcome variable is the natural logarithm of total payments in 2015 dollars. My main dependent variable is an indicator for whether a beneficiary's primary care physician (PCP) is integrated with a hospital. Both patient and physician selection on unobservables are likely. To address selection I use an instrumental variables model with physician fixed effects.

### 1.4.1 Selection on physician characteristics

Some physicians may be more willing to be employees than others, and some physician practices may be more appealing acquisition targets for hospitals. Generalists and Cardiologists who were acquired during my sample period (2007-2013), have different observables in 2006 and 2007 to those that were not acquired over the sample period. The details of this comparison are in Appendix A.3.

Being a Cardiologist substantially increases the likelihood of integrating between 2008 and 2013 relative to a generalist. This is likely a consequence of a change to Cardiology reimbursement practices in 2010.

Sites with more physicians are more likely to integrate. This may reflect a preference for autonomy among physicians at solo or small practices. It may also reflect hospitals trading off the transaction costs of negotiating a merger, with the benefit of the merger. The benefit of a physician-hospital merger for a hospital is heavily influenced by the referral potential of the physician practice, which is related to the practice size.

Practicing in a hospital service area with a more beds per capita seems to be correlated with being acquired by a hospital. This greater probability of hospital acquisition in capacity slack areas aligns with the theory that vertical integration is a response to input insecurity, or in this particular case that hospitals acquire physician practices to ensure themselves a supply of patients.

Being further from a hospital is negatively correlated with being acquired by a hospital. Practicing in a higher income area is positively correlated with hospital integration. Population density is negatively correlated with integration.

In spite of the differences on observables between integrating and non-integrating physicians, observables explain little of the variation in integration as evidenced by the low psuedo R-square value in Table A.2 and there is substantial overlap in the distributions of these observable characteristics. These factors could be due to unobserved physician characteristics or due to some randomness in which physicians are acquired. Nevertheless, there is substantial room for unobserved physician characteristics to play a role in which physicians integrate.

### 1.4.2 Physician fixed effects

Some unobserved physician characteristics may impact both the physician's propensity to be acquired and their patients' health outcomes. Of particular concern is physician skill. High skilled physicians may benefit more from operating independently, meanwhile hospitals may be disinterested in acquiring the practices of low skilled physicians. To address physician unobservables I include physician fixed effects. Since I am highly concerned that the physician unobservables are correlated with the regressor of interest, a random effects model is not appropriate.

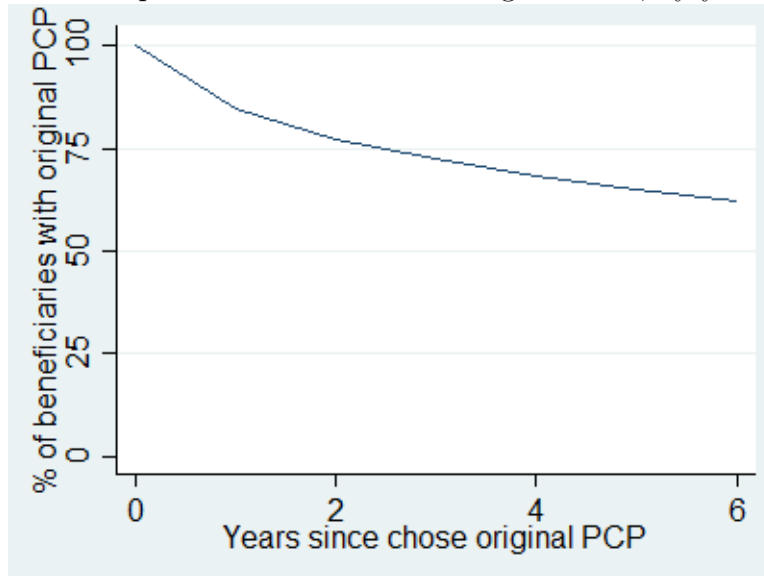
### 1.4.3 Selection on patient characteristics

Fee-For-Service (FFS) patients are able to choose their PCP and may choose an integrated PCP due characteristics that also cause the patients to have poor health outcomes. For instance, suppose that disorganized patients prefer to have someone else coordinate their care for them. This would cause them to select integrated PCPs at a higher rate than other patients. Meanwhile, disorganized patients may be prone to forgetting to take their prescribed medication causing them to have worse health outcomes. In this scenario, attending an integrated PCP would be correlated with poor health outcomes due to patients attending integrated PCPs being on average more disorganized. However, since we cannot observe disorganization, it would look like integrated PCPs are bad for patient health. To address patient selection I use an instrumental variables approach. I use two instruments in alternative specifications: an original PCP instrument, and an area average instrument.

### 1.4.4 Original PCP instrument

I focus on patients who choose an independent physician the first year the patient is in the sample. I refer to the PCP the patient first chose as the patient's "original PCP." The elderly have a particularly strong aversion to changing physicians (Robinson, 1997, p. 17). This leads to substantial inertia in physician choice among Medicare patients (see Figure 1.5). I exploit this inertia by instrumenting for the *current* integration status of the patient's *current* PCP, with the *current* integration status of the patient's *original* PCP, following Capps et al. (2015). Patients' initial PCP choice is likely influenced by observable physician characteristics so I add the original observable characteristics of the original PCP to my set of control variables, including whether the original PCP is a Cardiologist.

Figure 1.5: Fraction of patients still with their original PCP, by years in the sample



The sample is Medicare Fee-For-Service beneficiaries over 68 with at least one ACSCC whose original PCP is independent when the patient chose them.

#### 1.4.4.1 Intuition for identification

In summary, I am focusing on patients who initially choose an independent PCP. I assume that beneficiaries cannot tell if the physician will integrate in the future. Beneficiaries will largely remain with their originally chosen PCP due to a high degree of inertia in physician choice. Identification of the effect of integration on beneficiary outcomes is driven by the integration of the initially chosen PCP.

We can think of this in the framework of a randomized controlled trial where there are several treatment groups, each of which is treated at a different time. The treatment is attending an integrated PCP. Patients are assigned to a treatment group based on when their original PCP integrates. Patients whose original PCP never integrates are assigned to the control group. However, some subjects may attrit from their group, both before and after the treatment occurs. Attrition may be related to the subjects unobservable characteristics. I instrument for the actual treatment - attending an integrated PCP, with intention to treat

- having an original PCP that is currently integrated.

My estimation strategy is not quite analogous to a randomized controlled trial because the average values of patient's observable characteristics differ between the control and the various treatment groups. There is however, substantial overlap in the groups the distributions of patient characteristics. I include observable patient characteristics as control variables in the regressions.

#### 1.4.4.2 Formal statement of model, identification, and assumptions

Formally, my model can be stated as follows. Let  $i$ ,  $t$ , and  $k$  index patients, years, and physicians respectively. Let  $\tau$  be the first year that patient  $i$  is in the data. Patient  $i$  chooses a PCP in period  $t$  i.e.  $k = k(i, t)$ .  $v_{kt}$  takes a value of 1 if  $k$  is integrated in  $t$  and a value of 0 if  $k$  is not integrated in  $t$ .  $y_{ikt}$  is an outcome variable for patient  $i$  in year  $t$  who has chosen physician  $k$  as their PCP in  $t$ . Patients have both observable and unobservable characteristics, any of which may be time varying. Let  $\mathbf{X}_{it}$  be a vector of  $i$ 's observable characteristics in year  $t$  and  $\xi_{it}$  be the effect of  $i$ 's unobservable characteristics on  $y_{ikt}$ . Physicians have observable time varying characteristics  $\mathbf{W}_{kt}$  but their only unobservable characteristics that influence their patients' outcome are fixed over time. The effect of these time consistent PCP characteristics is represented by  $\delta_k$ . The effect of unobserved time varying variables is captured by the year fixed effects,  $\alpha_t$ . Finally, the patient's outcome is determined in part by a iid shock that is normally distributed about zero,  $u_{ikt}$ . The structural equation is 1.1. The instrument for  $v_{k(i,t)t}$  is  $v_{k(i,\tau)t}$ .

$$y_{ikt} = \beta v_{k(i,t)t} + \gamma \cdot \mathbf{X}_{it} + \omega \cdot \mathbf{W}_{k(i,t)t} + \theta \cdot \mathbf{W}_{k(i,\tau)\tau} + \delta_{k(i,t)} + \xi_{it} + \alpha_t + u_{ikt} \quad (1.1)$$

Since there is no obvious ordering of beneficiary-years within a physician, I use the within transformation to eliminate the effect of physician unobservables. In standard panel data

the within transformation requires strong conditional exogeneity i.e. that

$$E[v_{(i,\tau)s}\xi_{it}|\mathbf{X}_{it}, \mathbf{X}_{is}, \mathbf{W}_{k(i,t)t}, \mathbf{W}_{k(i,s)s}, \mathbf{W}_{k(i,\tau)\tau}, \tau, s, t] = 0 \quad \forall s, t \quad (1.2)$$

(Cameron & Trivedi, 2005, p. 758). Since I have within panel variation across beneficiaries as well as time, I technically need  $E[v_{(i,\tau)s}\xi_{jt}|\text{observables}] = 0 \quad \forall s, t, i, j$ . However, in practice there is no reason to expect that a patient's unobservables would be related to a characteristic of someone else's PCP.

Summary of assumptions:

1. Unobserved physician characteristics and their effect on health outcomes do not vary over time.
2. Any changes in unplanned admission rates, appropriate ED visit rates, death rates, and total spending due to external factors (e.g. policy changes) can be captured by year fixed effects.
3. The effect of physician-hospital integration does not depend on the timing of integration.
4. Strong exogeneity of the instrument conditional on observables: the current integration status of the original PCP is unrelated to past, current, and future patient unobservables conditional on observables.

For conditional strong exogeneity to hold in the context of my model, I must make the following assumptions.

- (i) Patients cannot observe whether physicians will integrate in the future.
- (ii) Patients cannot see any physician characteristics, beyond what is captured in the data, that would cause patient unobservables in period 0 to cluster by physician integration



propensity.

- (iii) Patients cannot see any physician characteristics, beyond what is captured in the data, that would cause patient unobservables in period 0 to cluster by the timing of future integration.
- (iv) Unobserved characteristics of the original PCP only impact future patient health via next period observables i.e.  $\delta_{k(i,0)}$  only affects  $X_{it}$ , not  $\xi_{it}$ .
- (v) Physicians do not choose to integrate nor are chosen as acquisition targets by hospitals on the basis of their patients' unobservables.

Assumptions 4i-4iii imply  $\delta_{k(i,0)}$  is not related to  $\xi_{i0}$ . Adding assumption 4iv extends this to  $\delta_{k(i,t)}$  is not related to  $\xi_{it}$  for all  $t \geq 0$ . A lack of correlation between  $\delta_{k(i,0)}$  and  $\xi_{it}$  is important because  $\delta_{k(i,0)}$  in part determines whether a physician later integrates.

There are a few circumstances that would violate each of these assumptions. For instance, Assumption 1 would be violated if physician skill accumulates over time. There is substantial evidence that physician skill remains relatively fixed. Furthermore, the time period of my study is quite short (7 years), which makes this assumption more likely to hold. I expect physician effort to change in response to integration but I consider this to be part of the effect of integration.

Assumption 2 would be violated if changes in external factors are location specific.

Assumption 3 simply assumes that  $\beta$  does not need a time subscript. This assumption would be violated if the physicians most suited to integration are the first to integrate. If this assumption is violated  $\beta$  will still reflect the average effect of integration for all integrators in the sample.

If physicians pre-announce their intention to integrate in a set number of years, Assumption 4i would fail to hold. Anecdotally, it is quite common for patients not to realize that

their physician has integrated with a hospital until they get a bill including a facility fee. Even the physician themselves presumably does not know that they will integrate many years in advance.

To examine the validity of Assumption 4ii I regress each outcome variable on all the control variables and an indicator of whether the originally chosen physician ever integrates, using the first year each beneficiary is in the data only. I include ZIP code fixed effects to capture area differences. (In the main regressions the physician fixed effects will largely capture the area differences.) The results are in Table 1.2. These suggest that the patients who choose physicians that eventually integrate tend to be a little sicker, conditional on observables. This will bias my regressions toward finding that integration is bad for patients. In spite of this, my regressions in fact find that integration is good for patients.

Table 1.2: Testing if patient unobservables are clustered by future physician integration type

Outcome variable	Coefficient on 1(PCP eventually integrates)
100 x 1[all cause unplanned admission]	-0.116 (0.399)
100 x 1[ACSCC unplanned admission]	0.430* (0.253)
100 x 1[all cause appropriate ED visit]	0.984** (0.476)
100 x 1[ACSCC (appropriate) ED visit]	0.631** (0.294)
100 x 1[all cause death]	-0.094 (0.188)
ln[1 + total spending 2015\$]	0.0081 (0.0141)

The main sample is Medicare beneficiaries over 68 with at least one ACSCC, who were covered by both Parts A and B during the portion of the year they were alive, and whose original PCP was independent when the patient first chose them. I include only the first year each beneficiary is in the main sample. Each cell represents the coefficient on 1(PCP later integrates) from a separate regression. The standard errors are shown in brackets. The left hand column specifies the outcome variables. SEs are clustered on the current PCP. \*\*\* significant at the 1% level, \*\* significant at the 5% level, \* significant at the 10% level. All regressions include a constant. All regressions include ln(PCP's number of Medicare patients), ln(number of physicians at PCP's site), 1(PCP is a Cardiologist), ln(PCP's number of Medicare patients), PCP's average diagnoses per claim, ln[adjusted gross income per household in ZIP code (2015 \$'000)], ln(miles to nearest hospital), 1(Medicare paid part of premium), age, 1(female), race indicators, count of chronic conditions from the the Medicare Chronic Conditions Data Warehouse list, indicators for each chronic condition from the Medicare Chronic Conditions Data Warehouse list, average diagnoses per claim for PCPs from previous 2 years, and year dummies.

Assumption 4iv is the most controversial assumption of this estimation strategy. The year in which a patient first enters the sample may be related to their health characteristics so I include dummy variables for the first year the patient enters the dataset. Suppose that assumption 4iv fails i.e. that future integrators cause better patient health even before the original physician integrates and that this is not fully captured in the observables. This would tend to bias my results toward finding a beneficial effect of physician-hospital integration. Suppose instead that future integrators cause worse patient health, which is not fully captured by observables. This would bias my results toward finding that physician-hospital

integration is detrimental for patient health.

For Assumption 4v to fail there would have to be an unobserved patient characteristic that could drive a physician to want to integrate or make a physician an appealing acquisition target. Patient Medicaid eligibility, ZIP code average income, patient demographic characteristics, and patient health are observed so this leaves little scope for such a variable to exist. A physician drawing an unusually low income set of patients relative to the area may be a less attractive acquisition target. However, the unusual low income would have to be high enough to not impact Medicaid eligibility. It is not entirely implausible that a physician specializing in low income patients in a high cost of living area could get patients that are low income relative to the city but not low enough income to qualify for Medicaid. However, this is unlikely to be a common occurrence, especially as none of New York, California, the District of Columbia, or Massachusetts is in the data.

#### 1.4.5 Area average instrument

The area average instrument is the average integration status of PCPs serving the beneficiaries ZIP code, excluding the beneficiary themselves. I consider a physician to be serving the beneficiary  $i$ 's ZIP code if one of the other sample beneficiary's in beneficiary  $i$ 's ZIP code uses this physician as their PCP. I exclude beneficiary  $i$  from the average to avoid mechanical correlation of the instrument with the beneficiary's unobservables. Formally this instrument can be expressed as

$$z_{ikt} = \sum_{j \neq i} v_{k(j,t)t}$$

where  $t$  is the current time period, and  $k = k(i, t)$  is beneficiary  $i$ 's PCP in period  $t$ . Since the originally chosen PCP and the year in which the beneficiary first enters the sample is not of great relevance in this specification, I no longer include the control variables for the original characteristics of the patient's original PCP or the dummies for the first year the

beneficiary was in the sample. I do keep the current characteristics of the current PCP.

The first three assumptions are the same as for the original PCP instrument. I also still require conditional strong exogeneity of the instrument to use the within transform. In this context conditional strong exogeneity means that the average integration status of PCPs serving the patient's ZIP is uncorrelated with past, current, and future unobserved patient characteristics conditional on observables. The area average instrument also requires the stochastic monotonicity assumption, that is an increased proportion of integrated PCPs serving the region increases the probability of any individual living in the region choosing an integrated PCP. Validity of the area average instrument requires that integration of PCPs in a beneficiary's ZIP code only affects the outcomes of the patient via the integration status of their own PCP.

Since my identifying variation is at the ZIP code level but I am using physician fixed effects, I cluster standard errors at the Primary Care Service Area (PCSA). PCSAs are defined by the Dartmouth Atlas of Healthcare project. PCSAs are clusters of ZIP codes where most people living in the cluster seek primary care from within the cluster.

The “only though” assumption could plausibly be violated for the all cause health outcome variables if integrated physicians are better at preventing communicable diseases. However, this is unlikely to be a first order concern.

To violate stochastic monotonicity there would need to be perverse people in the population who are happy to attend an integrated PCP if there are only a few around but not if there are many. Although one sees this sort of behavior with luxury goods it is unlikely to apply to the selection of an integrated PCP.

Conditional strong exogeneity will be violated if people cluster geographically on the unobservables that are important for health outcomes, beyond what can be accounted for by income and distance to the nearest general acute care hospital.

Strong exogeneity could also be violated for the admissions and emergency department

visit outcomes if hospitals decide to acquire surrounding physicians due to low utilization. Since emergency departments are once of the least profitable hospital departments, I do not expect this to be an issue for the emergency department outcomes. For the admission variables, I expect that unplanned admissions by Medicare FFS patients due to ACSCCs should make up a small enough fraction of total admissions that low admissions of this type should not drive hospital financial decisions. For the all cause admission variable it may perhaps be somewhat of a concern. In section 1.9.2 I show that my results are not greatly affected by weakening the exogeneity assumption from conditional strong exogeneity to conditional weak exogeneity.

#### **1.4.6 Comparison of the two estimation strategies**

Instrumental variables strategies identify local average treatment effects. An instrument using a different source of variation or that includes a different set of people in the sample could find different results. The original PCP instrument estimation strategy relies on inertia in PCP choice, whereas the area average instrument estimation strategy relies on patients' aversion to travel to see a PCP. The original PCP instrument estimation strategy requires me to exclude people whose original choice of PCP was already integrated when the physician chose them, whereas the area average instrument allows me to use the entire sample. The original PCP instrument requires a slightly controversial assumption (4iv). However, the original PCP instrument uses a more granular source of variation (patient-year level variation) than the area average instrument, which uses ZIP-year level variation. I later show that the original PCP instrument has a much stronger first stage than the area average instrument (refer to Panel B column 4 of Tables 1.3 and 1.4). The stronger first stage and more granular source of identifying variation lead to the coefficient estimates from the original PCP instrument regressions having much smaller confidence intervals than the coefficient estimates from the area average instrument regressions.

### 1.4.7 Why not a non-linear model for the health outcome regressions?

In the health outcomes regressions a linear functional form is not truly appropriate, since the outcomes are binary variables. However, including fixed effects for the tens of thousands of physicians in a non-linear model would be unwise due to the incidental parameters problem. It is also likely that a non-linear model with this many dummy variables would cause computational issues, as maximum likelihood estimation often does not converge in such models. Additionally, non-linear instrumental variables estimators suffer from problems. All the afore mentioned issues combine to make a linear instrumental variables with physician fixed effects estimator a more sensible alternative.

## 1.5 Results

Table 1.3, Panel A shows the coefficients of 1(PCP is integrated) in 24 separate regressions. Each row contains a different outcome variable and each column contains a different model (OLS, year fixed effects, PCP and year fixed effects, and instrumental variables with PCP and year fixed effects). The average values of the outcome variables in the sample used in these regressions are given in Table 1.1, column 2. The coefficients for the regressions with  $\ln(1 + \text{total payments } \$2015)$  as the outcome variables can be interpreted as follows: a coefficient of  $\beta$  indicates that changing from an independent to integrated physician is associated with an approximately  $100\beta\%$  change in total payments in 2015 dollars at the mean of the transformed total payments variable. The average value of the total spending variable in the restricted sample corresponds to \$4,084.

Table 1.3: Coefficients from regressions of each outcome on an indicator of the PCP's practice being owned by a hospital

Outcome variable	Specification			
	OLS	Year FE	PCP & year FE	Orig PCP instr, PCP & year FE
<i>Panel A: Coefficients of 1(PCP is integrated)</i>				
100 x 1[all cause unplanned admission]	0.725** (0.306)	1.619*** (0.320)	-0.331 (0.480)	0.194 (0.671)
100 x 1[ACSCC unplanned admission]	0.435** (0.187)	0.647*** (0.195)	-0.307 (0.295)	-0.745* (0.411)
100 x 1[all cause appropriate ED visit]	3.473*** (0.377)	3.586*** (0.394)	0.486 (0.573)	0.339 (0.794)
100 x 1[ACSCC (appropriate) ED visit]	1.080*** (0.226)	1.125*** (0.237)	-0.082 (0.341)	-0.802* (0.473)
100 x 1[all cause death]	-0.135 (0.146)	-0.102 (0.153)	0.056 (0.230)	0.081 (0.320)
ln[1 + total spending 2015\$]	0.0379*** (0.0106)	0.0501*** (0.0112)	-0.0073 (0.0160)	-0.0116 (0.0222)
No. beneficiary-years	357,648	357,648	357,648	342,275
No. clusters (physicians)	23,705	23,705	23,705	23,016
<i>Panel B: First stage statistics</i>				
Coefficient on instrument				0.689***
SE				(0.007)
F-stat				10,103

Each cell represents the coefficient on 1(PCP is integrated) from a separate regression. The standard errors are shown in brackets and are clustered on the current PCP. The sample is Medicare Fee-For-Service beneficiaries over 68 with at least one ACSCC, who were covered by both Parts A and B during the portion of the year they were alive, and whose original PCP was independent when the patient chose them. \*\*\* significant at the 1% level, \*\* significant at the 5% level, \* significant at the 10% level. All regressions include ln(PCP's number of Medicare patients), ln(number of physicians at the PCP's site), 1(original PCP is a Cardiologist), ln(original PCP's original number of Medicare patients), original PCP's original average diagnoses per claim, ln[adjusted gross income per household in ZIP code (2015 \$'000)], ln(miles to nearest hospital), 1(Medicaid paid part of the Medicare premium), age, 1(female), race indicators, a count of chronic conditions from the CMS chronic conditions data warehouse list, an indicator for each chronic condition from the CMS chronic conditions data warehouse list, average diagnoses per claim for PCPs from previous 2 years, dummies for the first year the patient is in the sample, and a constant. Regressions without PCP fixed effects also include 1(PCP is a Cardiologist).



Table 1.4: Coefficients from regressions of each outcome on an indicator of the PCP's practice being owned by a hospital

Outcome variable	Specification			
	OLS	Year FE	PCP & year FE	Area ave instr, PCP & year FE
<i>Panel A: Coefficients of 1(PCP is integrated)</i>				
100 x 1[all cause unplanned admission]	0.333* (0.183)	0.563*** (0.183)	-0.277 (0.394)	-4.934** (2.433)
100 x 1[ACSCC unplanned admission]	0.167* (0.0959)	0.230** (0.0968)	-0.208 (0.241)	-2.362 (1.506)
100 x 1[all cause appropriate ED visit]	1.811*** (0.248)	1.767*** (0.250)	0.452 (0.437)	2.119 (2.885)
100 x 1[ACSCC (appropriate) ED visit]	0.475*** (0.115)	0.497*** (0.116)	-0.002 (0.279)	-2.323 (1.724)
100 x 1[all cause death]	-0.190*** (0.0691)	-0.219*** (0.0687)	-0.116 (0.194)	-1.127 (1.149)
ln[1 + total spending 2015\$]	-0.0036 (0.0078)	0.0018 (0.0079)	-0.0059 (0.0133)	-0.141* (0.0831)
No. beneficiary-years	444,507	444,507	444,507	438,735
No. physicians	27,415	27,415	27,415	27,313
No. clusters (PCSAs)	1,611	1,611	1,611	1,603
<i>Panel B: First stage statistics</i>				
Coefficient on instrument				0.177***
SE				(0.010)
F-stat				330

Each cell represents the coefficient on 1(PCP is integrated) from a separate regression. The standard errors are shown in brackets and are clustered on the Primary Care Service Area (PCSA). The sample is Medicare Fee-For-Service beneficiaries over 68 with at least one ACSCC, who were covered by both Parts A and B during the portion of the year they were alive. \*\*\* significant at the 1% level, \*\* significant at the 5% level, \* significant at the 10% level. All regressions include ln(PCP's number of Medicare patients), ln(number of physicians at the PCP's site), ln[adjusted gross income per household in ZIP code (2015 \$'000)], ln(miles to nearest hospital), 1(Medicaid paid part of Medicare premium), age, 1(female), race indicators, a count of chronic conditions from the CMS chronic conditions data warehouse list, an indicator for each chronic condition from the CMS chronic conditions data warehouse list, average diagnoses per claim for PCPs from previous 2 years, and a constant. Regressions without PCP fixed effects also include 1(PCP is a Cardiologist).

The OLS results on the subsample of beneficiaries who originally chose an independent PCP (see Table 1.3, column 1) indicate that attending an integrated PCP is positively correlated with the probability of an unplanned admission, the probability of an appropriate emergency department visit, and total health care spending. There appears to be no statistically significant correlation between attending an integrated PCP and mortality risk.

Adding year fixed effects increases all coefficients (refer to Table 1.3, column 2). Adding current PCP fixed effects reduces the estimated coefficients of the integration indicator for all outcomes excluding mortality (see Table 1.3, column 3). The change in the coefficients for the non-death outcomes is consistent with either low skilled PCPs being more likely to integrate, or PCPs who eventually integrate attracting sicker patients initially. None of the coefficients are significant in the specification with PCP and year fixed effects.

In my preferred specification, where I address both patient and physician selection on unobservables and include year fixed effects, the estimated coefficients on the integration indicator are lower than in any other specification, except for the mortality and all cause unplanned admission outcomes (see Table 1.3, column 4). The reduction in the coefficients relative to the physician and year fixed effects specification without the instrument, suggests that sicker patients tend to chose integrated PCPs. I find that unplanned admissions due to AHRQ ACSCCs are significantly reduced by integrated PCPs, as are emergency department (ED) visits due to AHRQ ACSCCs. I find no significant effect on the all cause versions of these variables, which may be attributable to non ambulatory care sensitive conditions introducing noise into these outcome variables. I find no significant change in total health care spending due to attending an integrated PCP. The significant coefficients for health outcomes correspond to a 12% decrease in the risk of an ACSCC unplanned admission and a 10% decrease in the risk of an ACSCC ED visit. The coefficient for total spending corresponds to a decrease of \$47 on a base of \$4,084, which is not statistically significant.

Table 1.4 shows the results using the full sample and the area average instrument. For the

admissions and ED visit outcomes, the OLS results on the full sample document correlations of the same direction but smaller magnitude than the OLS results on the sample of patients that initially chose an independent physician (refer to Table 1.4, column 1). Unlike in the restricted sample, the OLS results on the full sample find no correlation between attending an integrated PCP and total health care spending, and the OLS results on the full sample suggest a significantly negative correlation between attending an integrated PCP and death. One interpretation of these and earlier results is that although the patients who chose an integrating physician do tend to be sicker, the patients who chose an integrating physician in their first year in the sample are less sick than the patients who chose an independent physician in their first year in the sample but later end up with an integrated PCP. This could be explained if pre-existing integration in 2007 was highly geographically clustered, so most people in those regions initially attended an integrated PCP regardless of their health, but people who switch to an integrated physician as it becomes available in their region are sicker than those that stay with their non-integrated physician.

Adding year fixed effects reinforces the positive correlation between attending an integrated PCP and the unplanned admission outcomes (see Table 1.4, column 2). The year fixed effects do not have a substantial impact on the estimated coefficients for the appropriate ED visit outcomes. The negative correlation between attending an integrated PCP and death is slightly greater with year fixed effects. The coefficient for total spending flips sign when year fixed effects are added but it remains extremely small and not significantly different from zero. Similarly to in the restricted sample, adding PCP fixed effects reduces the estimated coefficients for all outcomes (refer to Table 1.4, column 3).

Instrumenting for the integration status of the patient's PCP with the average integration status of PCP's serving the patient's ZIP code results in lower coefficients for all outcomes except all cause appropriate ED visits (refer to Table 1.4, column 4). The specification that uses the area average instrument and physician and year fixed effects suggest that attending

an integrated PCP reduces the risk of all cause unplanned admissions and total healthcare spending. The estimated coefficients on unplanned admissions due to an AHRQ ACSCC, and ACSCC ED visits are both negative but are not significant. The health outcome coefficients correspond to reducing the risk of an unplanned hospital admission by 25%, reducing the risk of an unplanned admission due to an ACSCC by 39%, and reducing the risk of an ACSCC ED visit by 28%. The coefficient on the total spending outcome variable corresponds to a reduction of \$533 on a base of \$4,055.

Since the base rate of all cause unplanned admissions is by definition higher than the base rate of AHRQ ACSCC unplanned admissions, it seems logical that the estimated effect in levels on all cause unplanned admissions would be weakly larger than the effect on AHRQ ACSCC. Using the area instrument, this is what I find for unplanned admissions. However, the original PCP instrument results do not have this feature, nor do the appropriate ED visit coefficients.

The precision of the original PCP instrument results is much greater than the precision of the area instrument results; standard errors of the coefficient on the integration indicator are >3.5 times higher in the area average instrument specifications than in the original PCP instrument specifications. The discrepancy in precision occurs because identifying variation in the original PCP instrument regressions is at the patient-year level within a physician whereas identifying variation in the area average instrument regressions is only at the ZIP-year level within physician. In other words identifying variation in the area average instrument regressions only comes from variation over time, and from some patients coming from different ZIP codes to other patients seeing the same physician. Due to the superior precision of the original PCP instrument results, I consider the original PCP instrument results to be my primary results and the area average instrument results to be a check on the primary results.

The original PCP results indicate that attending an integrated PCP results in fewer

unplanned ACSCC admissions, and fewer ACSCC ED visits. The coefficients in the area average instrument results for these outcomes are also negative, and in fact larger in magnitude than the original PCP instrument estimates. However, the much larger standard errors lead to the area average instrument coefficients not being classified as significantly different from zero. In spite of the significance issue, I conclude that the area average instrument results reinforce my original PCP results for these outcomes. Although the original PCP instrument results found no significant effect of integration on total spending, the area average instrument results find a significant reduction in total spending in spite of the larger standard errors. Combined, these two results reveal that integration does not increase health care spending

The effect sizes found with using the area average instrument and physician fixed effects are larger than the effect sizes found using the original PCP instrument. This may reflect two factors. First, the area average instrument results include people who originally chose an integrated PCP whereas the original PCP results exclude these people. The larger coefficient magnitudes in the original PCP instrument specifications relative to the area average instrument specifications, are consistent with the explanation that people who stand to benefit the most from integration chose an integrated PCP in the first year they were in the sample. Second, the differences between specifications in the estimated effect of integration on ACSCC unplanned admissions, the estimated effect on all cause appropriate ED visits, and the estimated effect on ACSCC ED visits, could reflect some upward bias in the original PCP estimates as a result of the initial patient unobservable differences shown in Table 1.2. However, this is unlikely to be the case for the all cause unplanned admission outcome since there were no significant unexplained differences in pre-treatment unplanned all cause admission rates. Recall that all cause unplanned admissions regressions were the most likely to be affected by reverse causality concerns in the area average instrument specifications. This could explain some portion of the discrepancy between the coefficients in the area av-

erage and original PCP specifications. However, as I explained above, I would expect the estimated effect on all cause unplanned admissions to be weakly larger than the estimated effect of AHRQ ACSCC unplanned admissions so I would not expect reverse causality to explain more than about half the difference between the coefficient for the unplanned all cause admission outcome in the area average specification and the coefficient for the unplanned all cause admission outcome in the original PCP specification.

Throughout the specifications, the relationship between death and whether the patient's PCP is integrated appears to differ from the relationship between the other health outcome indicators and whether the patient's PCP is integrated. Furthermore, the estimated effect of integration on average mortality is not significant in either of the specifications that address both patient and physician selection (see Tables 1.3 and 1.4, column 4). This is consistent with the PCP primarily preventing less severe health outcomes, at least in the short term. Death may be more reflective of the experience the patient has in a hospital if admitted, or may be the result of an accumulation of health care experiences.

## **1.6 Mechanisms via which integration affects health outcomes and spending**

There are several mechanisms via which hospital-physician financial integration may affect health outcomes and spending including improved care coordination, better outpatient care quality, and higher prices. Several of these mechanisms imply that the effect of integration will be heterogeneous over patient characteristics.

### 1.6.1 Higher prices

Under Medicare’s payment rules, many services incur higher fees when they are performed at a hospital outpatient facility than when they are performed at a physician office.<sup>11</sup> Medicare rules also permit hospitals to count the physician practices owned by the hospital as hospital outpatient departments, subject to a few unrestrictive conditions. Consequently, integration results in higher prices for many primary care services. Higher prices for primary care services directly raise spending on primary care. Additionally, higher costs of primary care could reduce primary care utilization and treatment compliance, which would detract from patient health. Assuming utility is concave in wealth, one would generally expect poorer people to suffer disproportionately from the health pitfalls of price increases in primary care, relative to their wealthier counterparts.

Supplemental insurance<sup>12</sup> could mitigate this effect, since it insulates beneficiaries from the higher prices of care. In addition to being associated with low income earners, Medicaid acts as supplemental insurance for dual eligibles.<sup>13</sup> Medicaid covers Medicare premiums, copayments, and coinsurance for the poorest people (<100% of the federal poverty level) (Centers for Medicare and Medicaid Services, 2016). Wealthier people may also have supplemental insurance via a MediGap plan.

### 1.6.2 Care coordination

Fragmented care can lead to poor health outcomes due to specialist’s treatment plans interacting poorly. Fragmented care can also waste resources if tests must be repeated due to a lack of information flow between settings. Primary care physicians (PCPs) often lack

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<sup>11</sup>This is slated to change.

<sup>12</sup>Supplemental insurance pays after the primary insurance plan. Supplemental insurance contributes to the out-of-pocket costs beneficiaries would incur if their primary insurance plan was their only insurance plan.

<sup>13</sup>Dual eligibles are people who are eligible for both Medicare and Medicaid.

information about care provided to patients in other settings (Smith et al., 2005; Schoen et al., 2009; Mehrotra et al., 2011; Jones et al., 2014).

Electronic Medical Records (EMRs) have been touted as a solution to care coordination issues. However, EMR take-up is not straightforward. First, EMRs are easily shared within a system but are not across systems. Consequently, the benefit to a physician practice of choosing a particular system is dependent on the systems chosen by the hospitals their patients attend, and the benefit to a hospital of using a particular EMR system depends on the systems chosen by referring physicians. Under joint ownership these externalities will be internalized. Second, EMRs require a large up-front investment. This can pose challenges, particularly for smaller physician practices. Hospitals, with their much larger capital stock, may be better positioned to make such large investments. Furthermore, their size may allow them to negotiate better rates with EMR providers. The large up-front investment, and the fact that the benefits accrue over a long time period, makes investment by a hospital in a physician practice risky if there is some possibility that the physician-hospital linkage will break down. Similarly, it makes investment by a physician practice in a system that aligns with a particular hospital risky. Co-ownership of a hospital and a PCP practice should ensure that they share patients in future periods. Additionally, ownership of a physician practice by a hospital should give the hospital control over any investment they make in the physicians' facilities. Lammers (2013) identifies a positive relationship between the probability that a hospital uses health IT and the probability of the hospital using an integrated salary model, using variation in state laws that prevent hospitals from employing physicians. In my companion paper (Wagner, 2016) I show that physician-hospital integration appears to result in physician practices installing EMRs.

A less modern but potentially effective solution to cross-setting care coordination is physician phone calls. Surveys of PCPs indicate that some PCPs confer only with specialists that treat more than a few of their patients (O'Malley et al., 2009, p. 7). This suggests that



there may be some economies of scale present when PCPs and specialists have more shared patients. Interviews reported in Jones et al. (2014) regarding communication between PCPs and hospitalists provide some reasons why this may be the case, such as difficulty reaching each other on the phone due to busy schedules. For entities with many shared patients, it may make sense to schedule time for such conversations. Physicians may be provided with incentives to refer their patients to either the hospital that owns the practice or to specialist physicians at other practices owned by the hospital. Furthermore, when physician practices are acquired by a nearby hospital, this should ensure alignment of insurance plan acceptance between the physician practice and the hospital owner. Both these factors would act to increase the concentration of referrals. Referral concentration would allow physicians to take advantage of the apparent economies of scale in cross-setting communication activities and increase the number of patients whose care is discussed with their other physicians. I directly test the effect of physician-hospital integration on referral patterns in Wagner (2016) .

The benefits of improved care coordination between different physicians are likely larger for patients who see physicians from many different specialties. However, the number of types of specialists a patient sees may be influenced by whether their PCP is integrated. I estimate the expected number of specialist types a patient will see based on the patient's characteristics, their ZIP code characteristics, and year fixed effects using a negative binomial model. If integration improves care coordination between specialists or between PCP and specialists, I anticipate that health outcome improvements from integration should be increasing in the expected number of specialists.

### **1.6.3 Better outpatient care quality**

Employing physicians alters their incentives. The alteration to physician incentives will depend on the employment contract. Consequently, the expected impact on patient outcomes is unclear. Physicians as entrepreneurs stand to capture the full financial benefit of activ-

ities in their practice, unlike physicians who are employees. Entrepreneur physicians may therefore exert more effort to provide high quality care to maintain patient loyalty. However, with less financial motivation to see many patients, employed physicians may spend more time with each patient, which may result in higher quality care. Hospitals are well aware of the pitfalls of employment contracts for physician motivation so they often add performance clauses into physician contracts.

Physician incentives may also affect referrals and therefore the quality of care received. Integrated physicians no longer benefit as much from keeping small procedures in house, which may lead them to refer patients elsewhere for small procedures once the physician integrates with a hospital. This may be beneficial for patient health if the patient is referred to a specialist in the procedure. However, referrals from primary care offices to specialists may result in a higher price.

Outpatient care quality does not provide unambiguous predictions for heterogeneity in the effect of integration over patient characteristics. However, if the heterogeneity predictions prove false, better outpatient care quality may be the explanation for the improvement in health outcomes that I found in the main results.

#### **1.6.4 Estimation strategy for testing heterogeneity predictions**

To test for heterogeneity in the effect of attending an integrated PCP on health outcomes and costs, I rerun the physician fixed-effects instrumental variables regressions, including both the dimension of heterogeneity and the interaction between the dimension of heterogeneity and the indicator for the PCP being integrated. I then select a series of evenly spaced values across the support of the heterogeneity variable. For  $\ln(\text{ZIP code income})$  I use  $\{3.2, 3.4, 3.6, 3.8, 4.0, 4.2, 4.4, 4.6, 4.8\}$ . For the expected number of physician specialties I use integer values between 3 and 9. Finally, I use bootstrapping to estimate the ratio of the predicted impact of integration to the average outcome value at each level of the heterogeneity variable.

To determine the base rate of the outcome at each level of  $\ln(\text{ZIP code income})$  I use the average value of the outcome for patients within 0.1 of the target level. To determine the base rate of the outcome at each level of the expected specialty count I use the average value of the outcome for patients within 0.5 of the target level.

## 1.6.5 Heterogeneity results

### 1.6.5.1 Heterogeneity over ZIP code income

The effect of integration on unplanned admissions, appropriate emergency department visits, and total spending appears to be relatively flat over the ZIP code income distribution (see Figures 1.6 to 1.9, and 1.11). This suggests that less wealthy patients are not being deterred from receiving preventative care by the higher prices associated with integrated PCPs. It is possible that this reflects a high rate of supplemental insurance, which tends to decrease price sensitivity of patients.

The death graph (see Figure 1.10) slopes downward. It appears that the poorest patients experience increases in mortality risk when their PCP integrates but the wealthiest patients experience decreases in mortality risk when their PCP integrates. Deaths are likely influenced by hospital care as well as primary care. Hence the slope could be induced by the quality of hospital care received when patients are admitted. Baker et al. (2016) show that Medicare beneficiaries are more likely to choose the hospital that owns their physician's practice. If the hospitals that acquire physician practices in low income areas tend to be of low quality relative to other hospitals in the area or the hospitals that acquire physician practices in high income areas tend to be of high quality relative to other hospitals in the area, this could induce a downward slope in the effect of integration on death without affecting the less severe health outcomes. An alternative explanation is that integrated physicians could be referring their poor patients to low quality hospitals and their wealthy patients to high

quality hospitals. Several factors could motivate this type of selective referral. First, high quality hospitals might cost more than low quality hospitals. Physicians may think they are helping poor patients by referring them to a hospital where they can afford the out-of-pocket payments. However, if this were occurring I would expect the total spending effect to be decreasing in ZIP code income, which it is not. Second, hospitals may direct their affiliated physicians to refer them the more profitable and less risky patients. There may be an expectation that poor patients are more likely to default on their out-of-pocket liabilities. Alternatively, poor patients may have unobserved factors that make them riskier hospital patients. If the hospitals that tend to acquire physician practices are high quality hospitals, selective referral of poor patients to other hospitals could explain integration increasing death rates for poor patients but not wealthy patients.

Figure 1.6: Heterogeneity in effect of integration on all cause unplanned admissions over ZIP code income

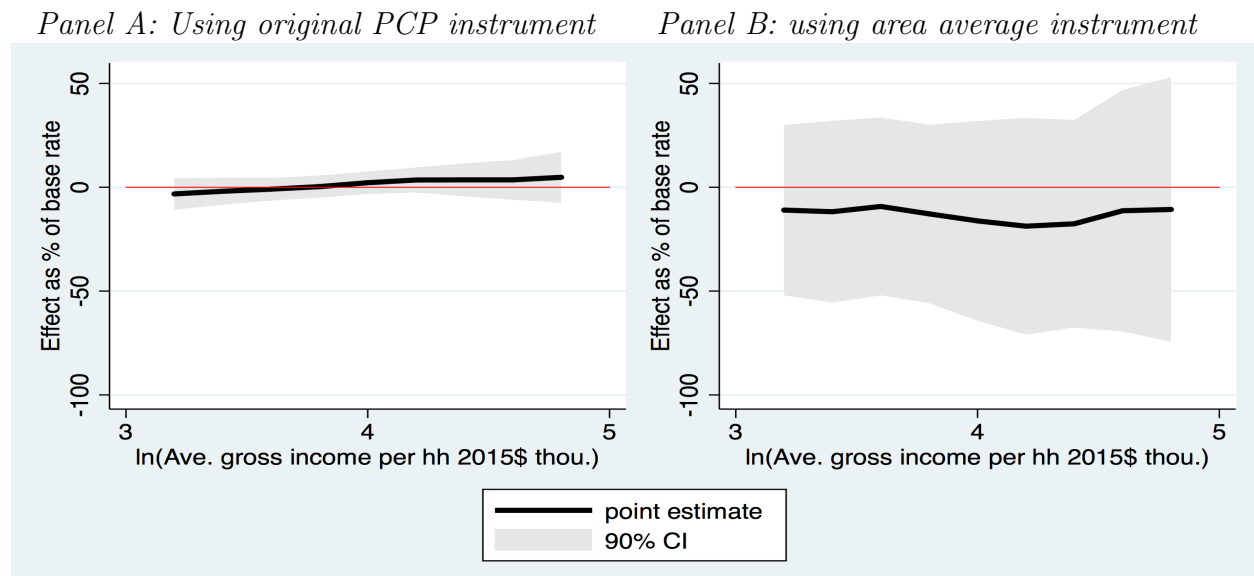


Figure 1.7: Heterogeneity in effect of integration on ACSCC unplanned admissions over ZIP code income

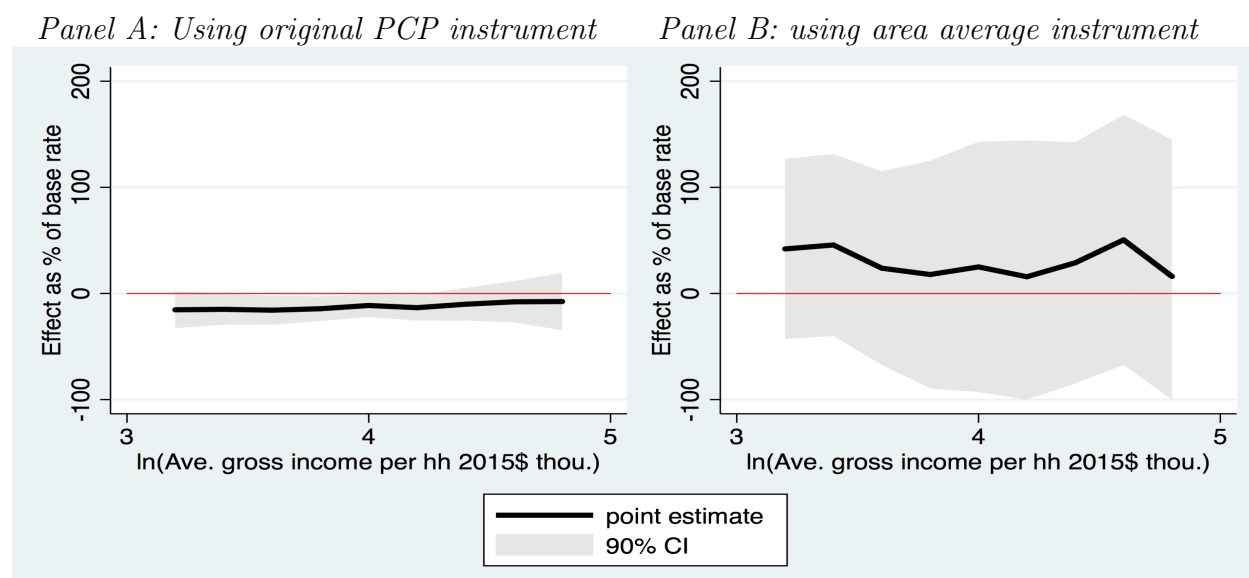


Figure 1.8: Heterogeneity in effect of integration on all cause appropriate ED visits over ZIP code income

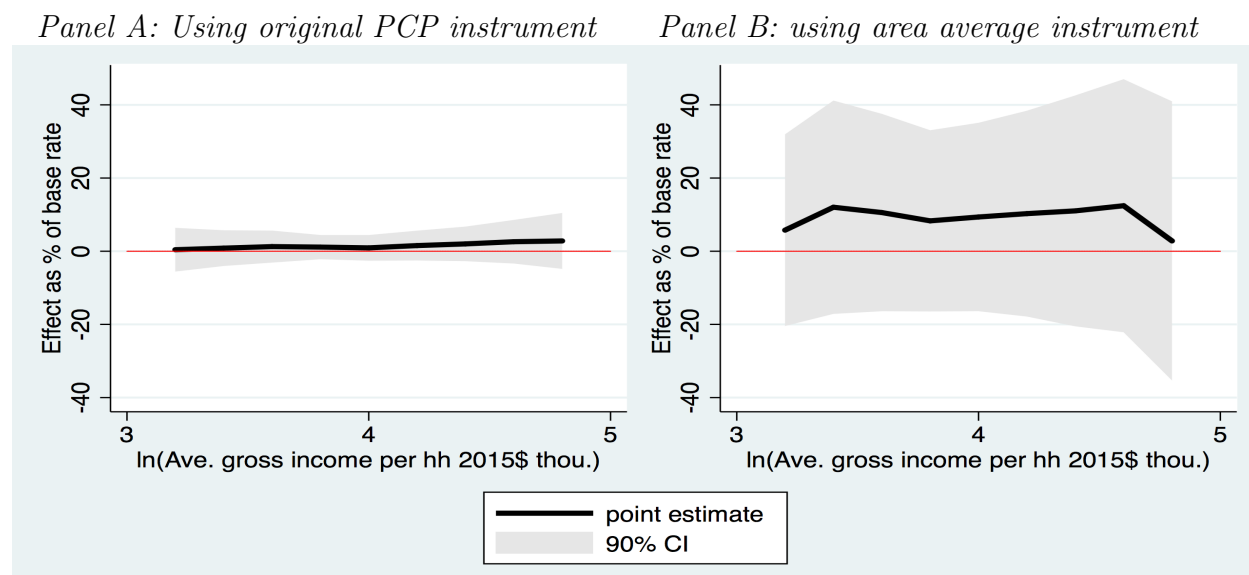


Figure 1.9: Heterogeneity in effect of integration on ACSCC ED visits over ZIP code income

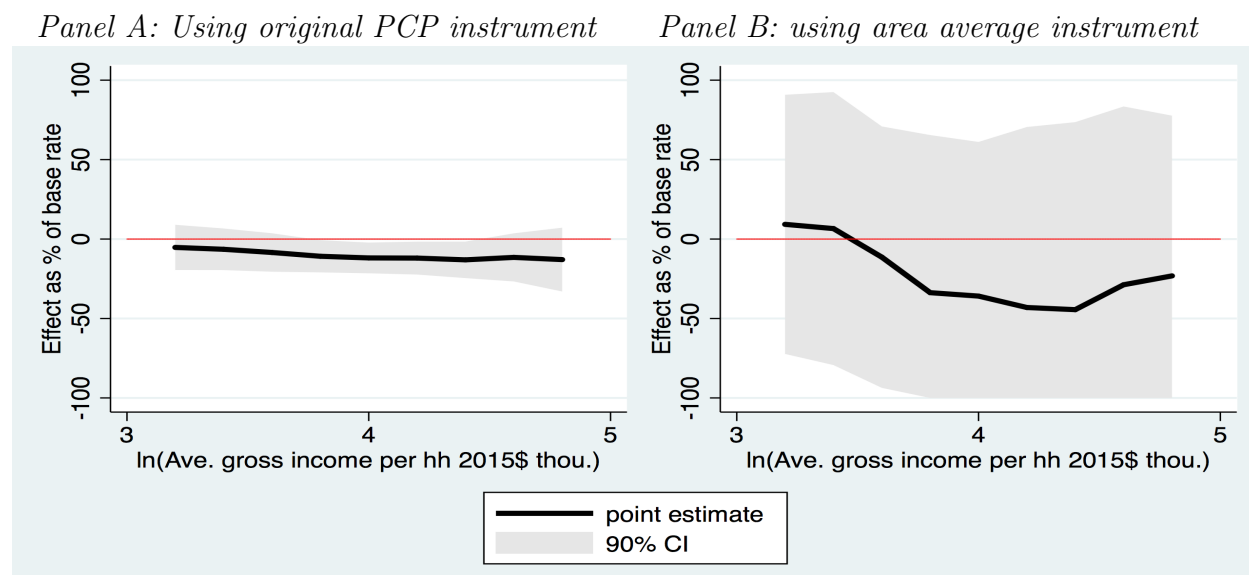


Figure 1.10: Heterogeneity in effect of integration on death over ZIP code income

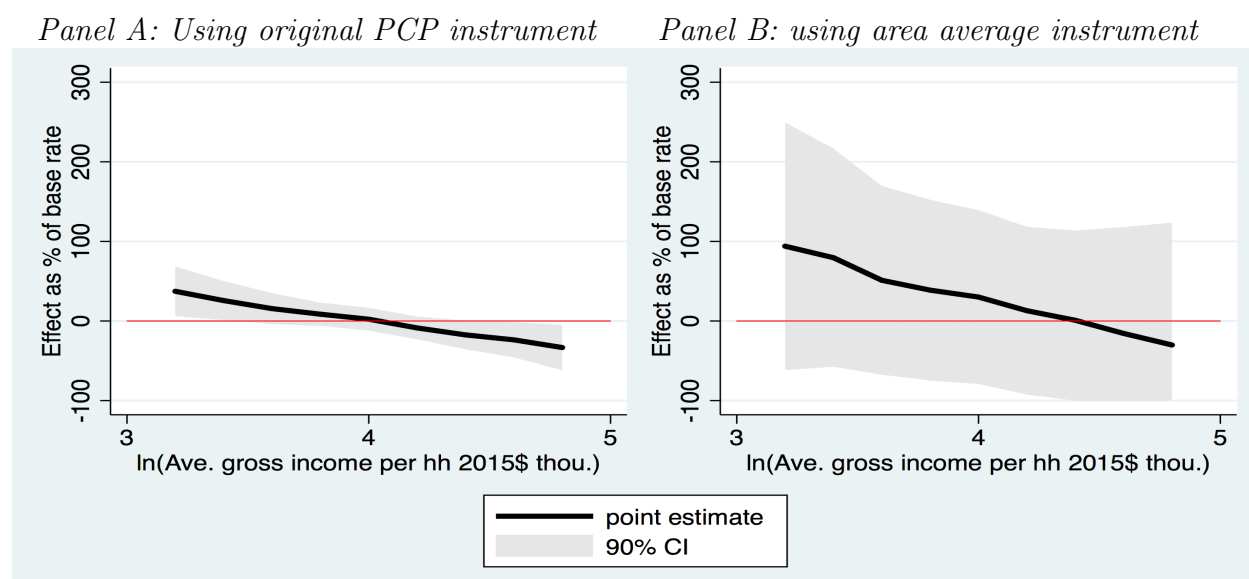
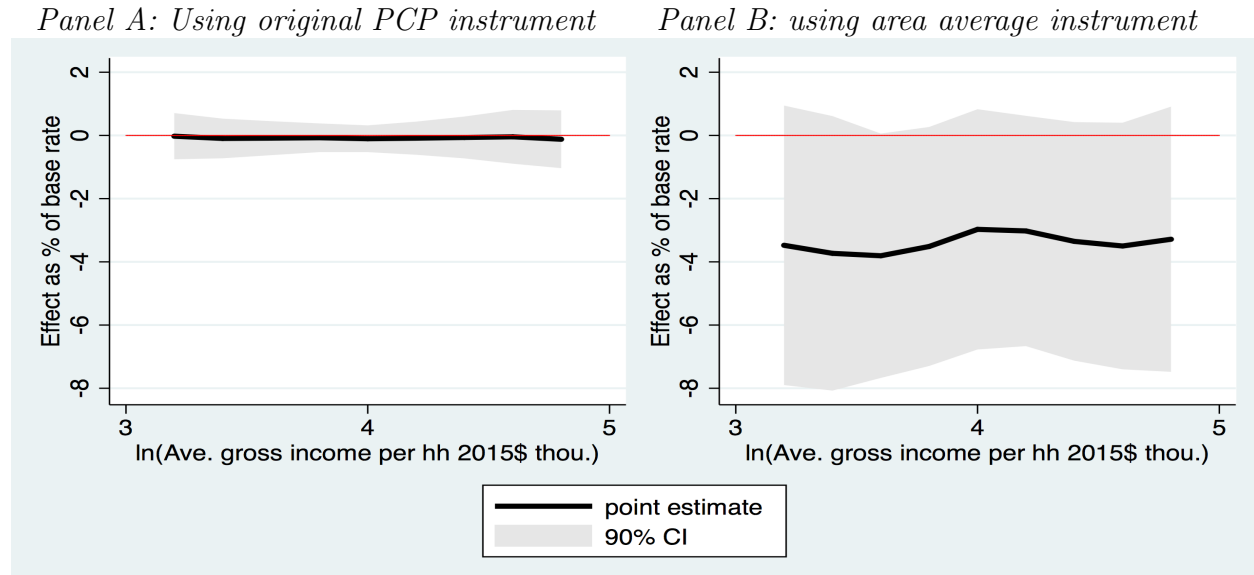


Figure 1.11: Heterogeneity in effect of integration on total health care spending over ZIP code income



#### 1.6.5.2 Heterogeneity over expected number of specialists

Across most outcomes I cannot reject that the effect of integration on the outcome is non-decreasing in the number of specialist types the patient is expected to need based on their underlying characteristics. The exception is that the original PCP instrument results indicate that integration may reduce all cause appropriate ED visits more for patients who are expected to see many specialists than for patients who are expected to see few specialists. The area average instrument results would seem to refute this. The balance of the evidence is not in favor of PCP-to-specialist or specialist-to-specialist care coordination explaining the benefits of physician-hospital financial integration.

One caveat to this is that the expected number of specialties probably does a good job of capturing a patient's need for physician-to-physician care coordination, but it may not do a good job at picking up a patient's need for hospital-to-physician coordination. A patient's need for hospital to physician coordination is especially strong when they are discharged.

My unplanned admissions indicators may be picking up some readmissions at the beginning of the year from initial admissions that occurred the previous year. Consequently, part of the reduction in unplanned admissions risk found in my main results could be attributable to readmission prevention via physician-hospital care coordination.

Another caveat is that for several outcomes, the effect of integration does increase in levels as the expected number of specialty types rises. The ratio declines because the base rate of the outcome grows faster than the effect size. It is possible that reducing an adverse outcome rate from 30 percentage points to 27 percentage points is harder than reducing an adverse outcome rate from 3.0 percentage points to 2.7 percentage points even though each of these represent a 10% reduction in the adverse outcome risk relative to the base rate of the outcome.

In ongoing work, (Wagner, 2016) I directly investigate whether physicians and hospitals are implementing changes that give them the opportunity to better coordinate care, both between different types of outpatient physicians, and between hospitals and physicians.

Figure 1.12: Heterogeneity in effect of integration on all cause unplanned admissions over expected specialty type count

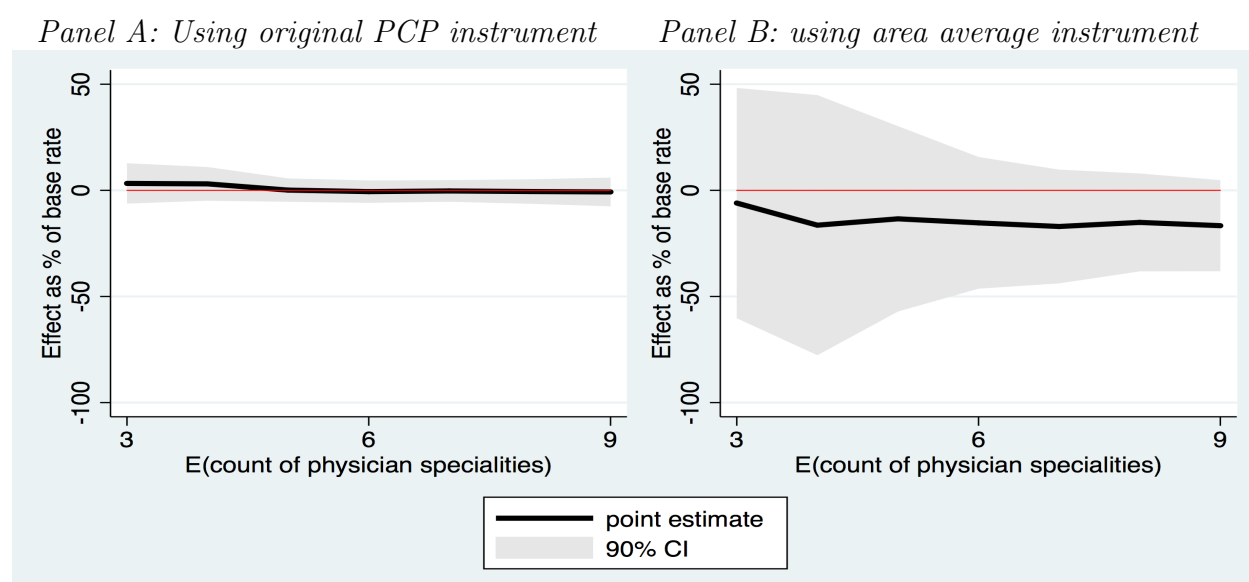




Figure 1.13: Heterogeneity in effect of integration on ACSCC unplanned admissions over expected specialty type count

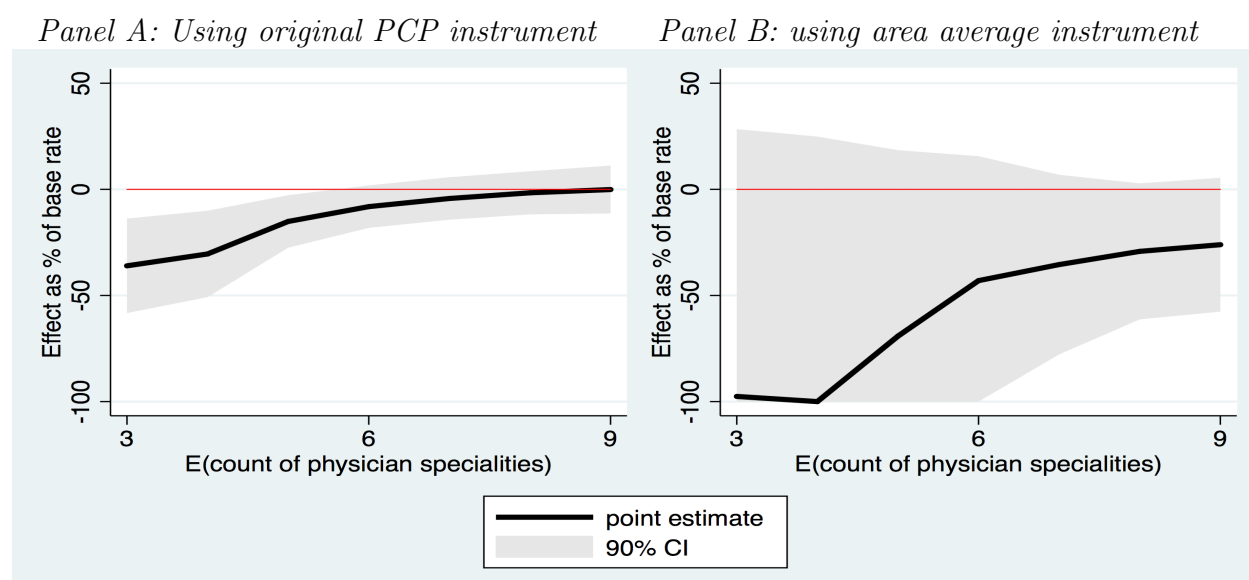


Figure 1.14: Heterogeneity in effect of integration on all cause appropriate ED visits over expected specialty type count

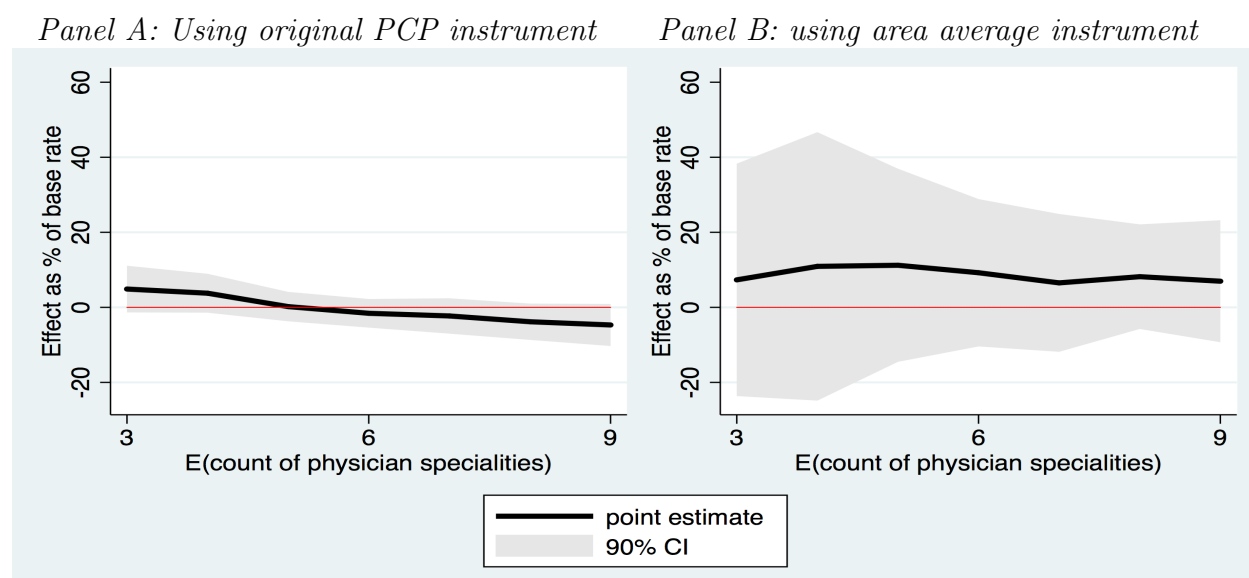


Figure 1.15: Heterogeneity in effect of integration on ACSCC ED visits over expected specialty type count

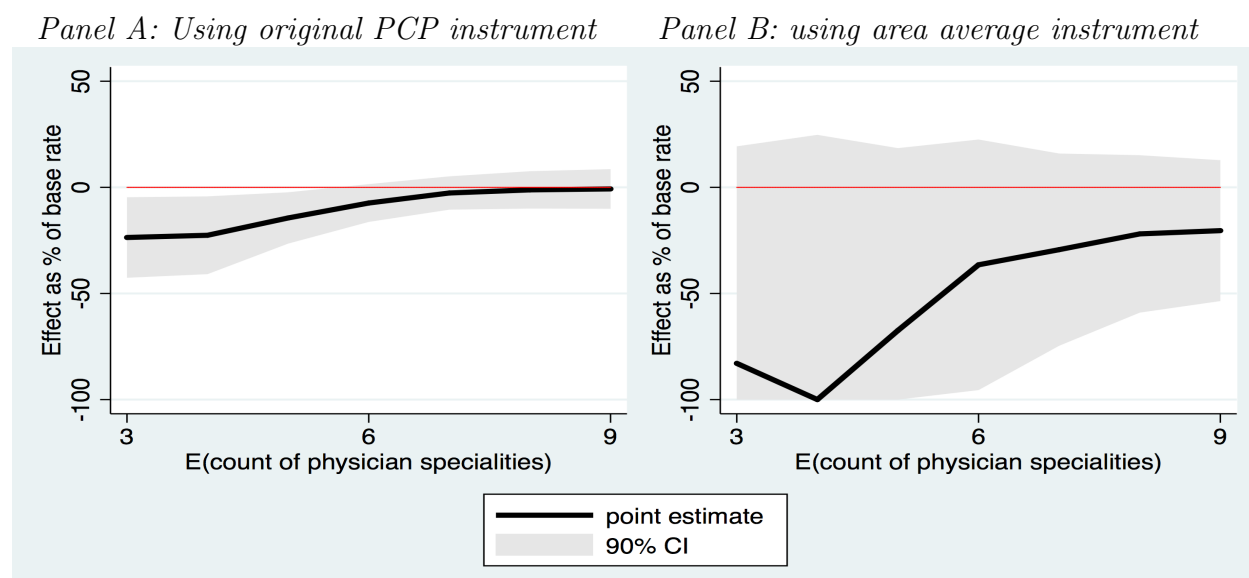


Figure 1.16: Heterogeneity in effect of integration on death over expected specialty type count

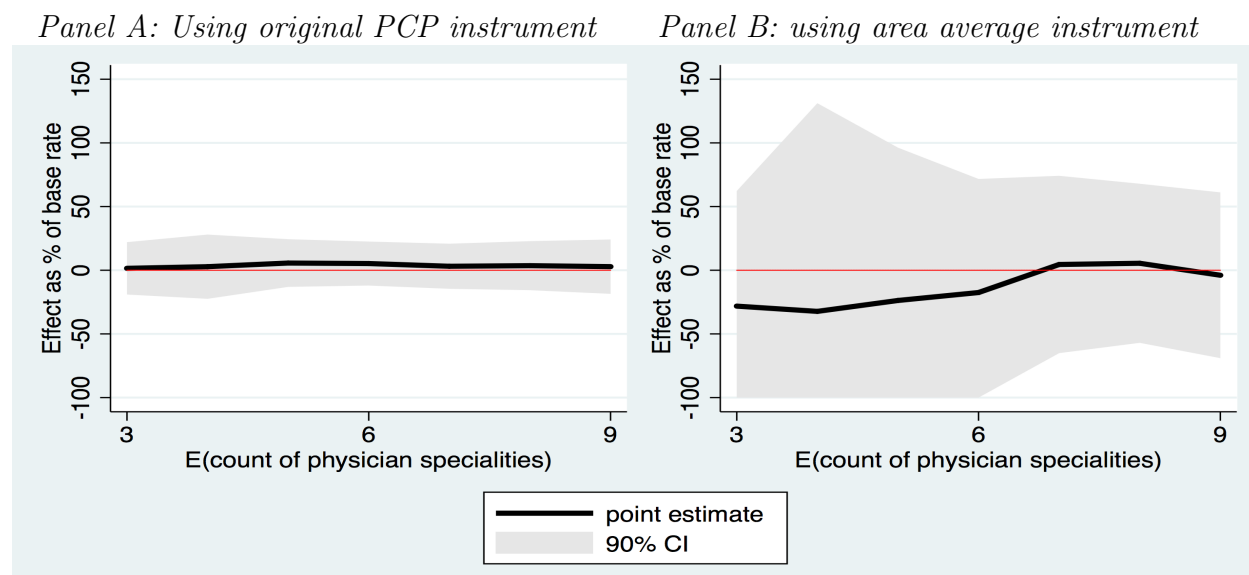
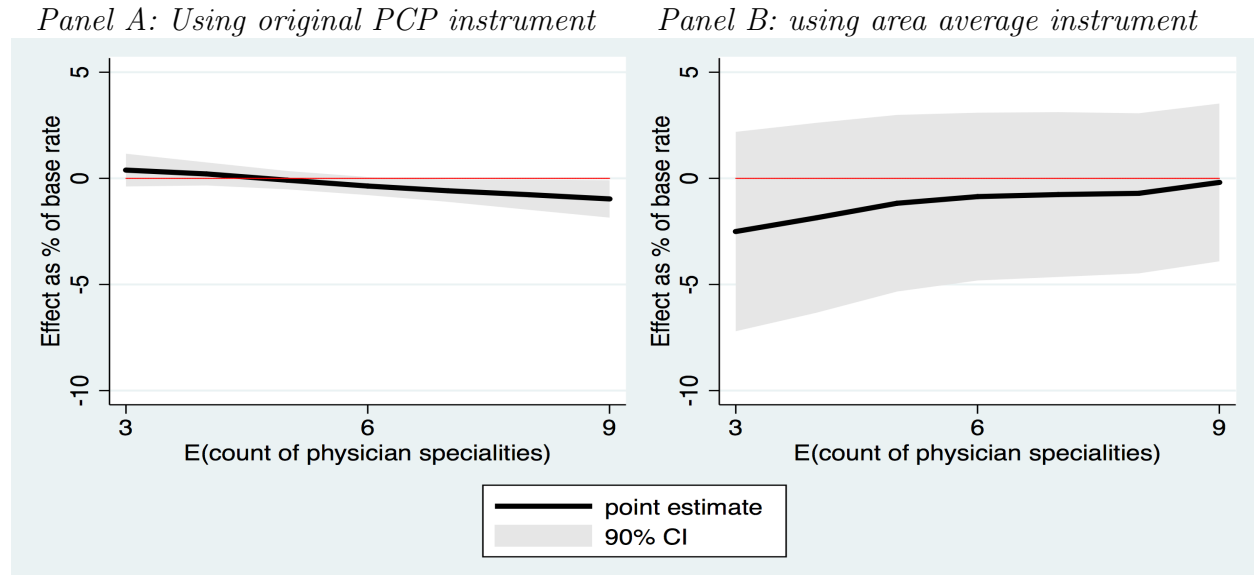


Figure 1.17: Heterogeneity in effect of integration on total health care spending over expected specialty type count



## 1.7 Timing of effect

A newly integrated physician practice may take some time to change their technology and physician behaviors. To examine how the effect of integration develops over time, I replace  $1(\text{PCP is integrated})$  with 3 indicator variables:  $1(\text{PCP has been integrated for 1-2 years})$ ,  $1(\text{PCP has been integrated for 3-4 years})$  and  $1(\text{PCP has been integrated for } \geq 5 \text{ years})$ . I instrument for these with analogues of the original PCP instrument -  $1(\text{original PCP has been integrated for 1-2 years})$ ,  $1(\text{original PCP has been integrated for 3-4 years})$ , and  $1(\text{original PCP has been integrated for } \geq 5 \text{ years})$ . In a separate specification I use analogues of the area average instrument - the fraction of PCPs serving the ZIP code who have been integrated for 1-2, 3-4, and  $\geq 5$  years.

Since the original PCP instrument sample excludes beneficiaries whose original PCP was integrated in the first year, there are no beneficiary years in the sample for whom the original PCP has been integrated  $>5$  years. In fact it turns out that very few people entered the

data in 2007 and initially chose a PCP that integrates in 2013. Of those that did fewer still are in the data in 2013 due to a combination of deaths, adopting Medicare Advantage, dropping either Part A or Part B coverage, or simply having too fewer claims to be classified as having a PCP. Consequently in the original PCP instrument regressions I cannot identify the coefficient on  $1(\text{PCP has been integrated for } \geq 5 \text{ years})$ . The area average instrument regressions do not suffer from this problem.

*A priori* I expected to find that the longer a PCP had been integrated, the more integration would affect health outcomes. This prediction is roughly borne out in the area average instrument point estimates (refer to Table 1.5). It is also the case under the original PCP instrument specification, for the two outcomes that showed a significant response to integration in the main results. However, the point estimates from the other original PCP instrument regressions do not conform to this prediction. The coefficients for each time since integration indicator are not statistically distinguishable from each other.

Splitting the integrated physician-years by the time since integration, reduces the statistical power of the test for an integrated PCP having a non-zero effect on the outcome variable. Furthermore, the number of observations in these regressions is lower than in the main regressions. There are two reasons for the lower number of observations in the time since integration regressions. First, I exclude the 2% of observations who had a physician who separated from a hospital after integrating. Second, it is not possible to identify the years since integration for some physicians. The standard errors are increasing in the number of years since integration. This is because only physicians that integrate early in the sample contribute directly to the identification of the coefficient on indicators for integrating many years ago, but all physicians that eventually integrate contribute directly to the identification of the coefficient on  $1(\text{PCP integrated 1-2 years})$ .

Table 1.5: Effect by time since integration for health indicators

	Yrs since integrated	Instrument			
		Original PCP		Area average	
		Coeff.	(SE)	Coeff.	(SE)
100 x 1[all cause unplanned admission]	1 to 2	0.538	(0.670)	-0.547	(1.544)
	3 to 4	0.666	(1.347)	-4.333	(2.938)
	$\geq 5$	-	-	-5.821	(3.912)
100 x 1[ACSCC unplanned admission]	1 to 2	-0.451	(0.410)	-1.357	(0.935)
	3 to 4	-0.625	(0.825)	-3.174*	(1.662)
	$\geq 5$	-	-	-1.709	(2.288)
100 x 1[all cause appropriate ED visit]	1 to 2	0.456	(0.785)	2.097	(1.818)
	3 to 4	0.121	(1.561)	-0.129	(3.583)
	$\geq 5$	-	-	-0.394	(4.299)
100 x 1[ACSCC (appropriate) ED visit]	1 to 2	-0.652	(0.469)	-1.479	(1.081)
	3 to 4	-1.060	(0.962)	-2.924	(1.984)
	$\geq 5$	-	-	-1.976	(2.603)
100 x 1[all cause death]	1 to 2	0.245	(0.318)	-0.900	(0.701)
	3 to 4	-0.022	(0.618)	-1.159	(1.221)
	$\geq 5$	-	-	-3.227*	(1.773)
ln[1 + total spending 2015\$]	1 to 2	-0.0079	(0.0223)	-0.0550	(0.0536)
	3 to 4	0.0170	(0.0434)	-0.0533	(0.0982)
	$\geq 5$	-	-	-0.0785	(0.133)
No. beneficiary-years		330,626		369,540	
No. physicians		19,670		21,746	
No. PCSAs		n/a		1,537	

## 1.8 Different forms of physician integration

The focus of this paper is physician-hospital financial integration via hospital ownership of physician practices. Other forms of physician integration include health management system (HMS) ownership of physician practices, and HMS management of physician practices. The SK&A physician survey asks physician practices which HMS owns or manages their practice if any. The question is framed in such a way that I can not separately identify practices that are owned rather than just managed by a HMS. I therefore have three types of physician integration I can identify: (1) the practice is owned by a hospital, (2) the practice is owned

or managed by a HMS, and (3) the practice is owned by a hospital, owned by a HMS, or managed by a HMS.

Table 1.6 displays the results of regressions using each of these physician integration measures. Unlike hospital ownership, HMS ownership or management does not have a significant impact on unplanned admissions due to ACSCCs or on ED visits due to ACSCCs.

Table 1.6: Effects of attending an integrated PCP using different forms of integration

Outcome	Ownership definition		
	Hospital owns	HMS owns, or HMS manages	Hospital owns, HMS owns, or HMS manages
<i>Panel A: Using original PCP instrument and within transform</i>			
100 x 1[all cause unplanned admission]	0.194 (0.671)	0.194 (0.616)	0.054 (0.604)
100 x 1[ACSCC unplanned admission]	-0.745* (0.411)	-0.067 (0.380)	-0.534 (0.376)
100 x 1[all cause appropriate ED visit]	0.339 (0.794)	0.209 (0.725)	0.233 (0.717)
100 x 1[ACSCC (appropriate) ED visit]	-0.802* (0.473)	-0.181 (0.443)	-0.582 (0.436)
100 x 1[all cause death]	0.0810 (0.320)	0.423 (0.296)	0.209 (0.294)
ln[1 + total spending 2015\$]	-0.0116 (0.0222)	-0.0274 (0.0203)	-0.0294 (0.0201)
No. beneficiary-years	342,275	334,768	291,055
No. physicians	23,016	21,974	19,798
<i>Panel B: Using area average instrument and within transform</i>			
100 x 1[all cause unplanned admission]	-4.934** (2.433)	-2.800* (1.599)	-2.081 (1.926)
100 x 1[ACSCC unplanned admission]	-2.362 (1.506)	-0.859 (0.943)	-0.941 (1.174)
100 x 1[all cause appropriate ED visit]	2.119 (2.885)	-2.014 (1.886)	0.968 (2.291)
100 x 1[ACSCC (appropriate) ED visit]	-2.323 (1.724)	-0.832 (1.097)	-0.389 (1.362)
100 x 1[all cause death]	-1.127 (1.149)	0.141 (0.683)	-0.316 (0.874)
ln[1 + total spending 2015\$]	-0.141* (0.0831)	-0.0780 (0.0546)	-0.0762 (0.0687)
No. beneficiary-years	438,735	444,239	437,677
No. physicians	27,313	27,474	27,390
No. PCSAs	1,603	1,596	1,602

## 1.9 Robustness checks

### 1.9.1 Using all admissions and ED visits rather than unplanned admissions and appropriate ED visits

For the main results I used unplanned admissions (admissions via the ED) rather than all admissions because I wanted to focus on changes in health outcomes rather than changes in utilization. One concern that may arise with using unplanned admissions rather than total admissions, is that patients' propensity to attend an ED prior to an admission may depend on the integration status of their PCP. Suppose a patient has an adverse health event and attends their PCP, who decides the patient must be admitted to hospital. If the PCP's practice is integrated with a hospital the PCP may be able to arrange for the patient to be admitted to the hospital directly, which would not count as an unplanned admission. Meanwhile if the PCP's practice is not owned by a hospital the PCP may be unable to arrange an admission and may instead tell the patient to go to an ED, from where the patient's admission would be counted as unplanned. This would bias the coefficient on  $1(\text{PCP is integrated})$  downwards. To address this concern, I repeat the main regressions using all admissions rather than only unplanned admissions. The results are shown in columns 2 and 4 of Table 1.7, alongside the results from the main regressions (columns 1 and 3). Rather than weakening the results as the above scenario predicts, using all admissions in place of unplanned admissions makes the results stronger. This suggests that the main results are not driven by a difference in how the patients of integrated and independent physicians are admitted to hospital.



Table 1.7: Using all admissions rather than unplanned admissions

Instrument	Original PCP	Original PCP	Area average	Area average
Types of admissions	Unplanned	All	Unplanned	All
100 x 1[all cause admission]	0.194 (0.671)	0.0822 (0.728)	-4.934** (2.433)	-6.375** (2.551)
100 x 1[ACSCC admission]	-0.745* (0.411)	-0.795* (0.435)	-2.362 (1.506)	-2.713* (1.632)
No. beneficiary-years	342,275	342,275	438,735	438,735
No. physicians	23,016	23,016	27,313	27,313
No. PCSAs	n/a	n/a	1,603	1,603

I also test the robustness of the results to using all ED visits rather than only appropriate ED visits. This is only relevant for the all cause ED visit indicator since due to the lack of difference between 1(appropriate ED visit due to an ACSCC) and 1(ED visit due to an ACSCC) I used the 1(ED visit due to an ACSCC) outcome in the main results. Table 1.8 demonstrates that using all ED visits rather than only appropriate ED visits does not affect the qualitative conclusion that physician-hospital integration does not have a significant effect on all cause ED visits, only ACSCC ED visits.

Table 1.8: Using all ED visits rather than only appropriate ED visits

Instrument	Original PCP	Original PCP	Area average	Area average
Types of ED visits	Appropriate	All	Appropriate	All
100 x 1[all cause ED visit]	0.339 (0.794)	0.749 (0.826)	2.119 (2.885)	-0.388 (3.018)
No. beneficiary-years	342,275	342,275	438,735	438,735
No. physicians	23,016	23,016	27,313	27,313
No. PCSAs	n/a	n/a	1,603	1,603

### 1.9.2 Weakening the strong exogeneity assumption in the area average instrument regressions

If I weaken the exogeneity assumption from strong conditional exogeneity to weak conditional exogeneity the threat that reverse causality poses to the all cause admission results is reduced.

Weak exogeneity requires only that  $E[z_{is}\xi_{it}|\text{observables}] = 0$  for all  $t$  and for all  $s \leq t$  (Cameron & Trivedi, 2005, p. 758). In other words, weak exogeneity does not preclude integration responding to past unexpectedly low admission rates. The cost and time required for a merger mean that lower than expected admissions in a given year are unlikely to induce acquisitions in that same year. With weak exogeneity the remaining reverse causality threat to identification is that changes in admission rates are correlated over time. In other words the remaining concern is that past declines in admissions both motivate hospitals to acquire physician practices, and are related to current declines in admissions.

Since I am using patient-year data with PCP fixed effects, the weak exogeneity assumption requires a two step transform. First I take the average of all variables within each PCP-year. Then I take the difference of the PCP-year averages. Since I only have integration variation between odd years (because I only purchased odd years of data), I take second differences rather than first differences. I weight each PCP-year level observation using the average number of sample patients they had over years  $t$  and  $t - 2$ .

Formally, the structural equation is equation 1.3. Let  $\mathcal{I}_{kt}$  be the set of patients whose period  $t$  PCP is  $k$ . Define the operators  $\overline{(\cdot)}_{kt} = \frac{1}{\#(\mathcal{I}_{kt})} \sum_{i \in \mathcal{I}_{kt}} (\cdot)_{it}$  and  $\Delta_2(\cdot)_{kt} = (\cdot)_{kt} - (\cdot)_{k,t-2}$ . The transformed equation is equation 1.4. The transformed instrument is  $\Delta_2 \overline{z_{kt}}$ .  $\overline{z_{kt}}$  can be interpreted as the average propensity of  $k$ 's patients to choose an integrated physician in year  $t$  based only on their location.

$$y_{ikt} = \beta v_{k(i,t)t} + \gamma \cdot \mathbf{X}_{it} + \omega \cdot \mathbf{W}_{k(i,t)t} + \delta_{k(i,t)} + \xi_{it} + \alpha_t + u_{ikt} \quad (1.3)$$

$$\Delta_2 \overline{y_{kt}} = \beta \Delta_2 \overline{v_{kt}} + \gamma \cdot \Delta_2 \overline{\mathbf{X}_{kt}} + \omega \cdot \Delta_2 \overline{\mathbf{W}_{kt}} + \Delta_2 \overline{\alpha_{kt}} + \Delta_2 \overline{\xi_{kt}} + \Delta_2 \overline{u_{kt}} \quad (1.4)$$

There is an efficiency cost to using weak rather than strong exogeneity. The efficiency cost comes from ignoring the within PCP-year variation and from the exclusion of PCPs who are only in one year of the data.

The coefficients generally retain the same sign using the second differences of PCP-year averages transform. The exception is for the all cause appropriate ED visit outcome where the sign becomes negative under the second differences of physician averages transform. The coefficient for the all cause unplanned admission outcome is more negative in the regression that assumes only weak conditional exogeneity than in the regression that assumes strong conditional exogeneity, which suggests the concern about hospitals buying physician practices in response to lower than expected admissions is not biasing the coefficient estimate downwards in the strong exogeneity regressions. However, the standard error on the weak conditional exogeneity estimator is more than 85% larger than the standard error on the strong conditional exogeneity estimator as a result of the efficiency loss. Due to the much larger standard error, the coefficient in the all cause unplanned admission weak exogeneity regression is not classified as significantly different from zero. The estimated reduction in total spending due to integration is larger in the second differences of PCP-year averages transform regression than in the within transform regression, though the difference is not statistically significant. In spite of the larger standard error of the second difference of PCP-year averages transform coefficient, the coefficient remains significantly negative. The estimated response of unplanned ACSCC admissions and ACSCC ED visits to integration remains negative and not statistically significant, which suggests that the area average instrument results lend support to the original PCP instrument results, even when the strong conditional exogeneity assumption is weakened to weak conditional exogeneity.

Table 1.9: Weakening the strong exogeneity assumption in the area average instrument regressions

Transform	Within	2nd difference in
		PCP-year ave
Sample	Full	PCP in $\geq 1$ year
<i>Panel A: Coefficients of 1(PCP is integrated)</i>		
100 x 1[all cause unplanned admission]	-4.934** (2.433)	-6.241 (4.543)
100 x 1[ACSCC unplanned admission]	-2.362 (1.506)	-1.206 (2.873)
100 x 1[all cause appropriate ED visit]	2.119 (2.885)	-2.210 (5.481)
100 x 1[ACSCC (appropriate) ED visit]	-2.323 (1.724)	-2.794 (3.276)
100 x 1[all cause death]	-1.127 (1.149)	-0.574 (2.275)
ln[1 + total spending 2015\$]	-0.141* (0.0831)	-0.254* (0.152)
No. beneficiary-years	438,735	263,415
No. physicians	27,313	18,883
No. PCSAs	1,603	1,482
<i>Panel B: First stage statistics</i>		
Coefficient on instrument in 1st stage	0.177***	0.307***
SE on instrument in 1st stage	(0.010)	(0.019)
F-stat for including instrument in 1st stage	330	380

### 1.9.3 Placebo test

As a placebo test for the “due to ACSCC” health outcomes regressions I use admissions and ED visits due to injury or poisoning unrelated to medical care as health outcome measures. Of the ICD-9 codes relating to injury and poisoning I exclude those for poisoning by drugs, those for medicinal and biological agents, and those for complications of medical care not elsewhere classified. This leaves me with ICD-9 codes 800-959, and 980-995. The base rate for injury and poisoning ED visits is higher than the base rate for ED visits due to ACSCC

(10.4% versus 8.2%), which if anything should make it easier to find results for regressions with injury or poisoning ED visits. Unfortunately, the base rate of unplanned admissions for injury and poisoning is too low to be a good comparator for unplanned ACSCC admissions (2.2% versus 6.0%). However, I present the results here for completeness. The results of the placebo test are shown in Table 1.10. Reassuringly, there is no significant relationship between attending an integrated PCP and unplanned admissions or ED visits due to non-medical related injury or poisoning.

Table 1.10: Effect of 1(PCP practice is owned by hospital) on placebo outcomes

Outcome	Instrument	
	Original PCP	Area average
1(unplanned injury or poisoning admission)	-0.147 (0.250)	0.779 (0.822)
1(ED visit due to injury or poisoning)	0.038 (0.539)	2.759 (1.903)
No. beneficiary years	342,275	438,735
No. physicians	23,016	27,313
No. PCSAs	n/a	1,595

#### 1.9.4 Reducing assumptions on the integration variable

My above results are based on assuming that sites once integrated, remain integrated, and that a physician that is integrated (independent) in two adjacent odd years is also integrated (independent) in the intervening even year. In Table 1.11 I show the results of the regressions when one or both of these assumptions is removed. The main results are restated in column 1. The results are qualitatively unchanged as these assumptions are removed with a few exceptions. In the original PCP instrument specifications, excluding the even years of data reduces the magnitude of the estimated effect of integration on unplanned admissions and ED visits due to ACSCCs and these coefficients become statistically insignificant. In the area average instrument specifications, excluding even years of data results in the estimated

effect of integration on unplanned ACSCC admissions being classified as significant. Finally, in the area average instrument regressions, dropping the weakly increasing integration status of sites assumption causes the coefficient for the total spending variable to be classified as insignificant; however, this does not change my conclusion that integration does not increase total spending on health care.

Table 1.11: Changing assumptions on the integration variable

Sites have weakly incr. integration status	Yes	Yes	No	No
Interpolate integration variable in even years	Yes	No	Yes	No
<i>Panel A: Using original PCP instrument and within transform</i>				
100 x 1[all cause unplanned admission]	0.194 (0.671)	0.375 (0.686)	-0.024 (0.636)	0.084 (0.653)
100 x 1[ACSCC unplanned admission]	-0.745* (0.411)	-0.589 (0.426)	-0.755* (0.386)	-0.676* (0.400)
100 x 1[all cause appropriate ED visit]	0.339 (0.794)	0.597 (0.815)	0.049 (0.759)	0.201 (0.786)
100 x 1[ACSCC (appropriate) ED visit]	-0.802* (0.473)	-0.455 (0.486)	-0.872* (0.445)	-0.617 (0.459)
100 x 1[all cause death]	0.0810 (0.320)	0.0476 (0.330)	0.262 (0.306)	0.279 (0.317)
ln[1 + total spending 2015\$]	-0.0116 (0.0222)	-0.0097 (0.0226)	-0.0070 (0.0212)	-0.0098 (0.0217)
No beneficiary-years	342,275	211,888	348,645	217,174
No physicians	23,016	22,160	23,767	23,013
<i>Panel B: Using area average instrument and within transform</i>				
100 x 1[all cause unplanned admission]	-4.934** (2.433)	-4.344* (2.300)	-3.387** (1.600)	-2.902* (1.532)
100 x 1[ACSCC unplanned admission]	-2.362 (1.506)	-2.640* (1.360)	-1.522 (0.984)	-1.742* (0.938)
100 x 1[all cause appropriate ED visit]	2.119 (2.885)	1.611 (2.584)	0.858 (1.843)	1.128 (1.771)
100 x 1[ACSCC (appropriate) ED visit]	-2.323 (1.724)	-2.024 (1.516)	-1.203 (1.107)	-1.106 (1.042)
100 x 1[all cause death]	-1.127 (1.149)	-1.285 (1.010)	-0.831 (0.742)	-0.727 (0.708)
ln[1 + total spending 2015\$]	-0.141* (0.0831)	-0.161** (0.0766)	-0.0876 (0.0538)	-0.0955* (0.0502)
No. beneficiary-years	438,735	270,042	433,563	270,531
No. physicians	27,313	26,611	27,325	26,660
No. PCSAs	1,603	1,604	1,598	1,598

### 1.9.5 Excluding Cardiologists

Cardiologists have a higher probability of integrating during the sample period and are particularly likely to integrate in 2013 rather than 2009. Furthermore people who chose a Cardiologist as a PCP may be sicker than people who choose a generalist as a PCP. Table 1.12 has the main results in columns 1 and 3 and the results excluding Cardiologists in columns 2 and 4. Excluding Cardiologists does not have a substantial impact on the health outcome results but does reduce the coefficient and raise the standard error in the area average instrument regression for the total spending outcome, which results in the coefficient being classified as not significantly different from zero. The qualitative conclusion that attending an integrated PCP does not increase health care spending remains unaffected by this change.

Table 1.12: Excluding Cardiologists

Instrument	Original PCP	Original PCP	Area average	Area average
Include Cardiologists	Yes	No	Yes	No
100 x 1[all cause unplanned admission]	0.194 (0.671)	-0.0427 (0.627)	-4.934** (2.433)	-4.714* (2.475)
100 x 1[ACSCC unplanned admission]	-0.745* (0.411)	-0.683* (0.380)	-2.362 (1.506)	-2.097 (1.461)
100 x 1[all cause appropriate ED visit]	0.339 (0.794)	0.454 (0.753)	2.119 (2.885)	2.035 (2.931)
100 x 1[ACSCC (appropriate) ED visit]	-0.802* (0.473)	-0.840* (0.436)	-2.323 (1.724)	-1.860 (1.680)
100 x 1[all cause death]	0.0810 (0.320)	0.0718 (0.297)	-1.127 (1.149)	-1.771 (1.149)
ln[1 + total spending 2015\$]	-0.0116 (0.0222)	-0.0117 (0.0213)	-0.141* (0.0831)	-0.128 (0.0864)
No. beneficiary-years	342,275	305,295	438,735	402,461
No. physicians	23,016	19,714	27,313	24,209
No. PCSAs	n/a	n/a	1,603	1,568



### 1.9.6 Alternative definitions of PCP

In my main regressions I required that a physician have at least three claims with a patient to be treated as the patient's PCP. If the physician has less than three claims with the beneficiary during the year, I treated the beneficiary as not having a PCP and excluded them from the sample. I chose this threshold because it ensured a high proportion of beneficiaries had a unique physician that accounted for their maximum number of claims without resulting in too many beneficiaries being dropped from the sample. I adjust this threshold both up and down and rerun the regressions.

Aside from the death outcome, the standard errors of the estimates are increasing in the minimum number of claims to count as a PCP across both instruments (see Table 1.13). This is likely driven by the reduction in the sample size. Using either instrument, the signs of the estimated coefficients generally do not change.

The significance of the estimated effect of integration on ACSCC unplanned admissions and ACSCC ED visits is sensitive to changing the threshold. In the original PCP instrument specifications, the coefficient for the ACSCC unplanned admissions outcome is only classified as significant when the threshold is three, and the coefficient for the ACSCC ED visit outcome is classified as significant when the threshold is one or three. When the threshold is four, the area average instrument coefficients for the ACSCC unplanned admissions and ACSCC ED visit outcomes become significant. Using the area average instrument, the effect of integration on all cause unplanned admissions remains negative and significant, as the claim threshold is raised but not as it is lowered. Regardless of the threshold and instrument used, the finding that total spending is not increased by integration persists.

Table 1.13: Sensitivity of results to changing the minimum number of claims required to count the most frequently attended outpatient physician as a PCP

Outcome	Minimum claims to count as a PCP				
	1	2	3	4	5
<i>Panel A: Using original PCP instrument and within transform</i>					
100 x 1[all cause unplanned admission]	0.410 (0.605)	0.395 (0.630)	0.194 (0.671)	0.390 (0.726)	-0.046 (0.806)
100 x 1[ACSCC unplanned admission]	-0.611 (0.378)	-0.542 (0.391)	-0.745* (0.411)	-0.447 (0.445)	-0.375 (0.500)
100 x 1[all cause appropriate ED visit]	0.218 (0.729)	0.354 (0.756)	0.339 (0.794)	0.835 (0.859)	0.656 (0.946)
100 x 1[ACSCC (appropriate) ED visit]	-0.820* (0.436)	-0.734 (0.449)	-0.802* (0.473)	-0.435 (0.511)	-0.321 (0.576)
100 x 1[all cause death]	0.143 (0.324)	0.127 (0.319)	0.0810 (0.320)	0.0461 (0.331)	0.015 (0.358)
ln[1 + total spending 2015\$]	-0.0085 (0.0216)	-0.0108 (0.0220)	-0.0116 (0.0222)	-0.0007 (0.0232)	-0.0022 (0.0245)
No. beneficiary-years	392,010	374,072	342,275	304,764	261,803
No. physicians	24,408	23,847	23,016	22,047	20,837
<i>Panel B: Using area average instrument and within transform</i>					
100 x 1[all cause unplanned admission]	-1.656 (2.157)	-2.352 (2.288)	-4.934** (2.433)	-6.241** (2.748)	-5.567* (3.144)
100 x 1[ACSCC unplanned admission]	-1.815 (1.308)	-1.751 (1.357)	-2.362 (1.506)	-2.803* (1.623)	-1.890 (1.855)
100 x 1[all cause appropriate ED visit]	3.607 (2.584)	3.300 (2.752)	2.119 (2.885)	0.786 (3.161)	0.786 (3.651)
100 x 1[ACSCC (appropriate) ED visit]	-1.799 (1.502)	-1.551 (1.575)	-2.323 (1.724)	-3.434* (1.871)	-2.045 (2.142)
100 x 1[all cause death]	-0.620 (1.147)	-1.083 (1.104)	-1.127 (1.149)	-1.847 (1.224)	-0.872 (1.351)
ln[1 + total spending 2015\$]	-0.0756 (0.0806)	-0.0847 (0.0816)	-0.141* (0.0831)	-0.191** (0.0919)	-0.185* (0.101)
No. beneficiary-years	508,502	483,354	438,735	386,965	329,071
No. physicians	28,756	28,172	27,313	26,224	24,934
No. PCSAs	1,637	1,614	1,603	1,580	1,560

## 1.10 Discussion

### 1.10.1 Summary of findings

My results on health outcomes suggest that there are health benefits to primary care physician (PCP) hospital integration for some people. Those health outcome benefits extend to preventing unplanned hospital admissions and emergency department (ED) visits due to ambulatory care sensitive chronic conditions (ACSCCs) but not to reducing mortality. I find that total spending on health care does not increase when a patient's PCP integrates, even though integrated PCPs charge higher prices.

I provide evidence that physicians who integrate are either less skilled or on average attract sicker patients than physicians who do not integrate. I also find that patients with better health are more likely to choose a new PCP when their PCP integrates. These findings confirm the necessity of accounting for both patient and physician selection on unobservables when examining the effect of physician-hospital financial integration on health outcomes. No prior studies about the effect of physician-hospital financial integration on health outcomes have accounted for both patient and physician selection on unobservables.

I show that patients in low income ZIP codes are receiving similar benefits to patients in high income ZIP codes, in terms of preventing unplanned admissions and appropriate emergency department visits. However, I also show suggestive evidence that patients in low income areas experience mortality risk increases from attending integrated PCPs whereas patients in high income areas experience mortality risk reductions from attending integrated PCPs. These findings are consistent with integrated PCPs providing better primary care. These findings are also consistent with either integration driving physicians to refer their poor patients to systematically worse hospitals than their wealthy patients, or poor quality hospitals acquiring physician practices in poor neighborhoods and high quality hospitals acquiring physician practices in wealthy neighborhoods.

I find a non-decreasing relationship between the effect of attending an integrated PCP (relative to the base outcome rate) and the number of specialists a patient is expected to need based on their underlying conditions. This suggests that physician-hospital financial integration has little impact on physician to physician care coordination, which leaves two mechanisms that could explain my finding that integration improves health outcomes: (1) improved care quality at the primary care level; (2) better hospital to physician coordination post discharge.

### 1.10.2 Comparison to the literature

Like Cuellar & Gertler (2006), I do not find significant effects of attending an integrated provider on mortality. I do however, find significant health benefits to attending an integrated provider for less severe health outcomes, which Cuellar & Gertler (2006) were not able to investigate due to their limited data. My result that integration decreases unplanned hospital admissions aligns with Baker et al.'s (2014) finding of decreased utilization in areas with a higher concentration of integrated hospitals. I am able to address concerns such as patient selection and reverse causality, which Baker et al. (2014) is not. I am able to draw some conclusions about the mechanisms via which physician-hospital integration may be influencing health outcomes, something that previous papers did not address.

Several papers other than this one have examined the impact of physician-hospital financial integration on total health care spending (e.g Capps et al., 2015; Baker et al., 2014). In contrast to this paper, prior papers find that physician-hospital integration increases total spending on health care for Fee-For-Service patients. Several factors may contribute to the difference between my results and the results of previous papers: my use of Medicare rather than privately insured patients, and my focus on patients with ACSCCs.

This paper focuses on Medicare beneficiaries whereas the other papers focus on the beneficiaries of private insurance plans. Under the Medicare system, fees are set by governmental

decree but prices for the privately insured are set via a bargaining process between insurers and providers. Intuitively, joint physician-hospital entities should have more bargaining power than any of the entities alone, which may result in higher prices for both physician and hospital services. Gal-Or (1999) uses a simple theoretical model to show that when physicians and hospitals merge, they are able to bargain for higher prices in the more competitive market under all circumstances, and higher prices in the less competitive market under some circumstances.<sup>14</sup>

This paper focuses on patients with ACSCCs. Patients without ACSCCs will still face higher primary care costs when their PCP integrates, in spite of potentially having no reduction in hospital utilization. Hence patients without ACSCCs may experience net increases in health care spending, unlike patients with ACSCCs.

### 1.10.3 Out of sample validity

Beneficiaries in the Medicare charges data are Fee-For-Service patients. The financial incentives for providing services and coordinating care differ under Fee-For-Service (FFS) arrangements and capitated contracts. Consequently, conclusions of this study should not be extrapolated to Medicare Advantage patients.

Some Medicare FFS beneficiaries may have access to medical care that is not paid for by Medicare and is therefore not recorded in the Medicare charges data. As a result, patients who receive most of their care via a source other than Medicare may not be classified as having an ACSCC when they in fact do. Consequently, these patients would be excluded from my sample. This is most likely to occur among beneficiaries who are high priority in the Department of Veterans Affairs system or who have comprehensive (as opposed to

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<sup>14</sup>Another theoretical paper that models the impact of physician-hospital mergers on prices, Eggleston et al. (2004), makes different predictions. However, Gal-Or (1999) considers a change from zero physician-hospital affiliations to one affiliation and Eggleston et al. (2004) considers a change from zero physician-hospital affiliations to every hospital being affiliated with every physician. The data on physician-hospital integration indicate we are closer to Gal-Or's case.

supplemental) private insurance. My results may not be valid in these populations.

My results should be considered an upper bound on the benefits of physician-hospital integration for two reasons. First, my sample includes only Medicare beneficiaries with at least one ACSCC. This includes about 70% of Medicare FFS patients who are 68 or over. Beneficiaries without any ACSCCs would likely benefit less from physician-hospital integration as their primary care experience is unlikely to deter unplanned hospital admissions or emergency department visits. The effects I find on health outcomes should either be applied only to patients with ACSCCs, or can be thought of as an upper bound on the magnitude of the effect of physician-hospital integration on health. Physicians do not integrate with hospitals at random. It is likely that the physicians who integrated were those best suited to physician-hospital integration. Similarly, beneficiaries do not choose their physician at random. I expect that beneficiaries who chose integrated PCPs are those who stood to benefit the most from attending an integrated PCP. This also supports thinking of my results as an upper bound on the benefits of physician-hospital financial integration. Consequently, my results should absolutely not be extrapolated to predict what would happen if all physicians were forced to integrate or if all FFS patients were obliged to attend integrated physicians.

#### **1.10.4 Other caveats**

My patient health outcome measures are limited to deaths, unplanned hospital admissions, and appropriate emergency department visits, so I may be missing milder negative health outcomes. However, I likely capture the most costly poor health outcomes and my poor health outcomes measures are an improvement over previous papers about the effect of integration on health outcomes, which relied only on mortality, and in hospital events (Cuellar & Gertler, 2006).

While my results are largely robust to the perturbations of definitions and assumptions explored in the robustness section of the paper (section 1.9), the results on health outcomes

are somewhat sensitive to the minimum number of claims required to count a physician as a PCP.

Some patients with alternative health care or alternative health insurance may receive enough care via Medicare to be included in the sample. For patients with alternative health care or alternative health insurance, the Medicare data will miss any hospital admissions and emergency department visits not claimed at least in part from Medicare. In such an event, the Medicare data will underestimate negative health outcomes. For people with supplemental insurance, Medicare covers the bulk of hospital or emergency department fees. For people with comprehensive private insurance in addition to Medicare FFS insurance, Medicare covers out-of-pocket payments. Hence underestimation of negative health outcomes due to alternative health insurance should only affect people who have comprehensive private insurance plans with no coinsurance or copays. For people who have access to care via the Department of Veterans Affairs however, underestimation of negative health outcomes may be a larger problem. Using the area average instrument, if beneficiaries covered also by the Department of Veterans Affairs are disproportionately in ZIP codes served by integrated physicians, this will bias the coefficient downward (towards finding an effect) in the admissions and ED visit regressions. For Department of Veterans Affairs care to bias the results downward in the original PCP instrument regressions, beneficiaries with alternative insurance would have to disproportionately choose future integrators. Similarly to the area average instrument regressions this could occur if beneficiaries with alternative insurance tend to be located in areas where integration increases more.

### 1.10.5 How to reconcile my results with hospital incentives for integrating

At face value, my results suggest that hospitals may be weakly harming their revenue stream by acquiring physician practices, which makes it surprising that they would choose to integrate. There are several reasons why this is an overly simplistic conclusion. First, my results focus on people insured by Medicare. As I mentioned above, there is evidence in the literature that physician-hospital financial integration enables hospitals to charge higher prices to private insurance companies due to enhanced bargaining power. It may be that hospitals find this increase in bargaining power more valuable than the loss of some Medicare patient admissions. Second, I found suggestive evidence that integrated physicians systematically refer their poor patients to worse hospitals than their wealthy patients. My results on total spending suppose that all out-of-pocket liabilities incurred by patients are eventually paid. It is possible that hospital acquisitions of physician practices allow the acquiring hospital to redirect patients who are at risk of defaulting on their out-of-pocket liability. Finally, there may be a competitive aspect to why hospitals choose to buy physician practices. Physician-hospital integration may reduce the total number of hospital admissions but it may also redirect the most profitable patients to particular hospitals. Hospital management may be concerned that if they do not buy physician practices their competitors will buy them instead, leading to the most profitable patients being redirected to their competitors.

## 1.11 Conclusion

I use patient-year level Medicare data along with physician survey data to estimate the effect of physician-hospital financial integration on health outcomes and health care spending. My health outcome measures are indicators for death, unplanned hospital admissions, and appropriate emergency department visits. I use an instrumental variables regression model



with physician fixed effects. My first instrument for the indicator of whether the patient's *current* primary care physician (PCP) is currently integrated, is an indicator of whether the patient's *original* PCP is currently integrated. This instrument is only valid in the subsample of patients whose original PCP was not integrated when the patient first chose them. Identification is driven by the timing of the integration of the original PCP and relies on inertia in patient choice of physician. My second instrument is the average integration status of PCPs serving each patient's ZIP code. It is valid in the entire sample but is not as strong as the first instrument. Identification in regressions using the second instrument is driven by ZIP codes where the fraction of integrated PCPs increases over time and relies on people's tendency to obtain primary care close to home.

I demonstrate that attending a PCP that is owned by a hospital improves health for patients with ambulatory care sensitive chronic conditions enough to reduce hospital admissions and emergency department visits due to these conditions but not enough to reduce death. In contrast to prior studies I find that the net effect on total spending is not positive. These results hold with either instrument and are robust to many other perturbations of the estimation strategy. I find that reductions in unplanned admissions and emergency department visits induced by integration extend to both poor and wealthy areas. However, I find suggestive evidence that integration increases mortality risk in poor areas and decreases mortality risk in wealthy areas. The relationship I find between the expected number of physician specialties and the effect of integration suggests that physician to physician care coordination improvements do not play a large role in the health improvements that accrue from PCP-hospital integration. Alternative explanations include integration improving primary care quality, and integration enhancing discharge coordination.

Whereas the literature found largely negative effects of physician-hospital financial integration in the form of cost increases, my results show that some patients – elderly patients with chronic conditions that are treatable in primary care settings – benefit from physician-

hospital financial integration. Together these findings suggest that physician-hospital financial integration benefits some patients to the detriment of others. Regulators may wish to consider potential distributional effects when evaluating physician-hospital mergers.

## Chapter 2

# The impact of physician-hospital financial integration on the referral patterns of primary care physicians

### 2.1 Introduction

In Chapter 1 I found that physician-hospital financial integration improved health outcomes among Medicare patients with chronic conditions. The exact mechanisms by which a financial arrangement impacts patient health outcomes is not clear. There are several channels via which physician-hospital financial integration is theorized to impact patient health outcomes including changes in referral patterns, improved care coordination, better outpatient care quality, and the installation of electronic medical records (EMRs). In this chapter I extend on the results of three recent papers (Baker et al., 2016; Carlin et al., 2016; Walden, 2016) by examining heterogeneity in the impact of physician-hospital integration on referral patterns. I also make improvements to the estimate of the effect of physician-hospital financial integration on referral volume.

I use data on Medicare FFS patients and survey data on physician integration status from the company SK&A to test whether PCP-hospital integration results in changes in referral patterns. Throughout the paper I use a number of different reduced form methods though I primarily rely on linear models with fixed effects and an instrument.

I test the effect of PCP-hospital integration on the number of referrals from the PCP per patient-year using reduced form regression methods. I include PCP and year fixed effects and an extensive set of control variables in my regression. I instrument for the integration status of each beneficiaries PCP with the integration status of the original PCP the beneficiary picked when they first entered the data. I exclude beneficiaries who chose an integrated PCP the first year they entered the data. This instrument addresses the concern that the PCPs patient mix may change in unobservable dimensions due to the PCP being acquired by a hospital. I find no evidence that PCPs change their volume of referrals per patient-year in response to being acquired by a hospital.

I test the effect of PCP-hospital integration on where PCPs refer patients, conditional on a referral being made. Observations are referral occasions interacted with all hospitals/ Cardiologists in the choice set (depending on whether I am considering the effect on inpatient referrals or the effect on Cardiology referrals). I use PCP-hospital or PCP-Cardiologist, and year fixed effects. My main regressors of interest are an indicator for the PCP being owned by a particular hospital,  $h$  (or for the PCP sharing a hospital owner with Cardiologist  $h$ ) and an indicator for the PCP being owned by a hospital other than  $h$  (or for the PCP being owned by a hospital but not the same hospital that owns Cardiologist  $h$ ). I instrument for these variables using the original PCP – however, the instrumentation does not drive my results. I find suggestive evidence that hospital acquisition is affecting the hospital PCPs refer their patients to. However I do not find evidence that sharing a hospital-owner with a Cardiologist affects the probability to directing referral to that Cardiologist. My inpatient referral results are also suggestive (albeit not statistically significant) of PCPs redirecting

their wealthy patients towards their hospital-owner, and their poor patients away from their hospital owner.

The rest of this chapter proceeds as follows. Section 2.2 discusses why integration might or might not change referral patterns, and previous literature on this topic. Section 2.3 presents the hypotheses I test. Section 2.4 describes my data and the variables I construct from it. Section 2.5 outlines my estimation strategy for testing each hypothesis. Section 2.6 presents my results. Section 2.7 discusses how my results fit into the literature and some caveats of my results. In Section 2.8 I conclude this chapter.

## **2.2 Background**

### **2.2.1 Total volume of referrals and shifting of referrals to the hospital-owner**

One of the purported motivations for hospital acquisitions of primary care physician practices is that hospitals hope to capture referrals to specialists operating out of their hospitals (Kocher & Sahni, 2011). When hospitals purchase physician practices they may provide incentives for physicians to refer to the hospital or to affiliated providers. Though explicit payments are banned under the Stark Act, the organization may be able to provide implicit pressure or may invest in procedures that make it simpler to refer to the hospital owner, or to specialists also owned by the hospital owner. Furthermore, as hospitals typically bargain with insurers on behalf of their employees (Burns et al., 2013), physician-hospital financial integration ought to lead to synchronization in insurance coverage between physicians and the hospital owner. This may also act to increase referrals from hospital-owned physicians to their hospital-owner, and from hospital-owned primary care physicians (PCPs) to hospital-owned specialists.

Although redirection of referrals has the potential to be beneficial or detrimental for patients via quality and cost channels of greater concern is the potential to physicians to change their referral volume. Over-referrals or under-referrals could both be detrimental to patients. A physician that is an entrepreneur stands to claim 100% of their earnings whereas an employed physician will be able to capture a smaller fraction of their earnings. A greater concern about earnings could cause independent physicians either to under-refer, relative to what would most benefit their patients, or to over-refer. There is a direct financial benefit to independent physicians from keeping procedures in house where possible – this suggests that physician-hospital financial integration may increase the total volume of referrals. However, if patients tend to request referrals and physicians are concerned about patient loyalty and its impact on their long-term revenues, independent physicians may experience greater pressure to over-refer. Although physician employment contracts are increasingly written so that physician pay is conditional on the amount of revenue the physician generates, hospitals may be able to account for revenue generated to the hospital via referrals in this metric in spite of the Stark Act. On balance I expect independent physicians face stronger financial incentives to keep procedures in house where possible, whereas integrated physicians face stronger financial incentives to make referrals. Physician contracts where reimbursement rates are skewed toward direct revenue generated (i.e. volume of patients treated) will tend to mitigate the expected increase in referrals due to physicians becoming employed, whereas physician contracts where reimbursement rates are skewed towards referrals generated will tend to reenforce the increase in referrals due to physician-hospital integration.

Another factor that may influence how responsive referrals are to physician-hospital integration is physicians' intrinsic motivation both to act in the best interests of their patients and to preserve their reputation. Most physicians claim to be acting in the best interests of their patients and there is evidence that physicians respond to intrinsic motivation (e.g. Kolstad, 2013). However, various studies have demonstrated the responsiveness of physicians

to financial incentives both in the laboratory (e.g. Hennig-Schmidt et al., 2011) and in the real world (Clemens & Gottlieb, 2014, e.g.). Nevertheless, physicians' intrinsic motivation can be expected partly attenuate the effect of physician-hospital integration on changes in referral patterns.

Carlin et al. (2016) did a case-study of hospital-ownership of physician practices. They study the effect of three physician practice acquisitions by two hospitals. The chief disadvantage of this paper is it uses very few mergers for identification so the results may not be generalizable.

Baker et al. (2016) conduct a cross-sectional analysis of the effect of hospital ownership of physicians on referral patterns. They use 2009 data on Medicare FFS patients and the SK&A data. They show that patients at hospital owned physician practices are more likely to attend the hospital owner than if the patients were at an independent physician practice. However, due to the cross-sectional nature of their study they are unable to rule out that hospitals may favor acquiring physicians that would refer to them often, which would bias their results toward finding an effect. Burns et al. (2013) provides anecdotal evidence that physicians that refer to a hospital often are in fact more likely to be acquired than physicians who do not often refer to a hospital. Moreover, using additional data from 2010 and adding a lead term to the model, Baker et al. (2016) show that their results about the effect of physician-hospital integration on total referrals to the acquiring hospital are in fact upwardly biased.

Walden (2016) studies the joint effect of hospitals acquisitions of PCP practices and multi-specialty physician practices acquisition of PCP practices. Using an event study framework, she finds that these types of acquisitions do not affect the total volume of referrals from PCP practices. She shows that the acquisition of a physician practice by a hospital does not result in a higher total volume of referrals. However, she also shows that acquisition of a PCP practice results in a shift of referrals to specialists affiliated with the new owner

of the practice. Walden uses aggregate Medicare datasets to construct both her ownership and her referral measures. She defines a specialist visit as a referral from a particular PCP if the patients sees the specialists within 30 days of that PCP, which she determines from the “Physician Shared Patient Patterns” datasets. She defines a physician as an employee of a hospital or multi-speciality physician practice if the physician bills under the hospital/practices tax identification number (TIN) more often than any other TIN. This information comes from the Medicare Provider Practice and Specialty files. One drawback of relying entirely on TINs to determine employment is that a physician may bill under a TIN when they operate out of a hospital even if they are not owned by that hospital. Nevertheless, using TINs should give some indication of integration if not precisely employment.

### **2.2.2 Implications of changes in referral patterns for cost and quality of care**

Hospital employment of previously independent physicians changes the incentives of the physicians in a number of ways, some of which could explain the finding in Chapter 1 that physician hospital integration improves health outcomes. As discussed above, physician-hospital financial integration may increase the total volume of referrals to specialists. Specialists generally charge more than generalists so additional referrals may act to increase the cost of care. However, specialists may be better at performing the procedures, which may improve patient outcomes.

Directing more referrals to the hospital owner and hospital owned specialists may have implications for both the cost and the quality of care patients receive at hospitals and specialists. Baker et al. (2016) also found that patients are more likely to choose a high cost, low quality hospital if their physician practice is owned by that hospital (both than they would be otherwise and relative to a low cost, high quality hospital). They showed evidence



that this second finding is less likely to be upwardly biased.

In Chapter 1 I find that hospital ownership of physician practices improves patient outcomes for Medicare patients with primary care sensitive chronic conditions. There are a number of potential explanations for why this finding may be compatible with Baker’s finding. Firstly, the finding that hospital ownership of physician practices improves patient outcomes for Medicare patients with primary care sensitive chronic conditions may be a consequence of better care in the primary care setting, which may be unrelated to the quality of the hospital care. Secondly, an additional consequence of more patients being sent to the hospital owner, or specialists affiliated with the hospital is that it will likely result in higher concentration of referrals. Surveys of primary care physicians (PCPs) indicate that some PCPs confer only with specialists that treat more than a few of their patients (O’Malley et al., 2009, p. 7). This suggests that there may be some economies of scale present when PCPs and specialists have more shared patients. Interviews reported in Jones et al. (2014) regarding communication between PCPs and hospitalists provide some reasons why this may be the case, such as difficulty reaching each other on the phone due to busy schedules. For entities with many shared patients, it may make sense to schedule time for such conversations. Referral concentration would allow physicians to take advantage of the apparent economies of scale in cross-setting communication activities and increase the number of patients whose care is discussed with their other physicians. This could lead to better patient outcomes even when patients are being referred to hospitals or specialists that have lower quality metrics.

### **2.2.3 Heterogeneity in changes in referral patterns**

In Chapter 1, I found suggestive evidence that PCPs may be directing their potentially less profitable patients away from their hospital owner. This suggests there may be heterogeneity over patient characteristics in changes to referral patterns induced by physician-hospital

integration. None of the existing literature has examined heterogeneity over patient characteristics in the effect of integration on referral patterns.

#### **2.2.4 Contributions of this paper to the literature**

In this paper I contribute to the literature on the impact of physician-hospital integration on referral patterns in a number of ways. First, I examine whether the changes in referral patterns induced by physician-hospital integration differ by patient characteristics, which no prior study has done.

Second, I am able to obtain a better estimate of the effect of PCP-hospital integration on the total volume of referrals and referrals to the hospital owner than did previous studies. Relative to Baker et al. (2016) I am able to include physician fixed effects, which will eliminate the upward bias in the estimated effect of physician-hospital integration on total referrals their paper suffers from. Relative to Walden (2016) I am able to focus on the effect of hospital ownership rather than the joint effect of hospital or multi-specialty practice ownership. Relative to Carlin et al. (2016) I am able to draw on a much larger number of acquisition events, which makes my results more generalizable.

Third, I use a different measure of integration that may be more closely correlated with employment than Walden (2016).

Forth, I directly examine the correlation between physician-hospital integration and referral concentration, which may help explain why Baker et al. (2016) finds that physician-hospital integration results in patients being referred to lower quality hospitals yet I find (refer to Chapter 1) that physician-hospital integration results in better health outcomes for patients with primary care sensitive chronic conditions.

## 2.3 Hypotheses

*Hypothesis 2.1 (propensity to refer)* Hospital acquisition of a primary care physician (PCP) practice increases the number of referrals each acquired PCP makes per patient, holding fixed patient characteristics.

*Hypothesis 2.2 (redirection to owner)* Physician hospital integration induces acquired PCPs to make a higher proportion of referrals to their acquirer (or to specialists at practices owned by their acquirer) conditional on making a referral.

*Hypothesis 2.3 (heterogeneity in redirection)* Physician hospital integration induces acquired PCPs to redirect their poor patients away from their acquirer but their wealthy patients toward their acquirer.

*Hypothesis 2.4 (concentration)* Physician hospital integration is associated with referrals becoming more concentrated.

## 2.4 Data and variables

To test the above hypotheses I need data where I can identify primary care physicians (PCPs), see referrals of the PCPs patients, and determine whether the PCP's practice is owned by a hospital. For testing hypotheses 2.2 and 2.3 I must be able to determine which hospital owns which physician practice. I need data on a proxy for patient income or wealth to test hypothesis 2.3. Finally, I need data on patient, physician/ physician-practice, and hospital characteristics to use as control variables. My five main sources of data are Medicare claims data, physician survey data from the company SK&A, American Hospital Association (AHA) survey data, ZIP code level income data from the Internal Revenue Service (IRS), and ZIP code geographic coordinates from the U.S. Census Bureau.

### **2.4.1 Medicare 5% sample**

My source of patient level data is the 5% sample of Medicare claims data for fee-for-service (FFS) beneficiaries over 1999-2013. I have the Medicare Beneficiary Summary Files; and the Carrier, Outpatient, and MedPAR Research Identifiable Files (RIFs). Patients have encrypted identifiers that allow me to track them across health care settings, and over time. The Medicare Beneficiary Summary Files contain patient demographic information and the patients' ZIP codes. The Outpatient, MedPAR, and Carrier Files contain patient diagnoses and procedures. The Carrier Files include physician claims (even for procedures performed in a hospital), whereas the Outpatient and MedPAR files include hospital claims. For physician claims both the referring physician and the procedure-performing physician are recorded where applicable. The hospital claims data include the hospital identifier and information on whether or not the patient used the emergency department. Both the physician and hospital claims data include procedure dates.

I limit my sample to beneficiary-years where the beneficiary was enrolled in Medicare Parts A and B for the whole year, since I can only see inpatient claims when a patient is covered by Medicare Part A, and I can only see outpatient claims when a patient is covered by Medicare Part B. I also limit the sample to patient-years where the patient was over 65 at the beginning of the year, since people who are eligible for Medicare when they are under 65 often are substantially sicker than most of the Medicare population.

### **2.4.2 SK&A physician survey data**

I obtain physician level data from the company SK&A. SK&A conducts surveys of approximately all office based physicians in the United States. Each physician office is contacted once every six months and the data is archived at the end of each year.

SK&A data is the most reliable publicly available source of data that records which

physicians are owned by hospitals or health systems and includes physician identifiers. It also includes the name of the hospital/ health system owners; a site ID (that can be used to link all physicians practicing at the same site); the number of physicians practicing at that site, and average daily patient volume at that site.

Due to the substantial expense of the SK&A data, I first checked the Medicare data to find out which specialties were most likely to serve as primary care physicians (PCPs) to patients in my sample. Most of the sample patients use Family Practitioners, General Practitioners, or Internal Medicine Specialists as primary care physicians (PCPs). However, numerous patients also use Cardiologists as primary care physicians. Consequently, I purchased data covering Family Practitioners, General Practitioners, Internal Medicine Specialists, Cardiologists, and Geriatricians. I limited the sample of physicians further by geography to cut down on the expense: I included Wisconsin, West Virginia, South Dakota, Oregon, Oklahoma, North Carolina, New Mexico, Montana, Missouri, Mississippi, Minnesota, Michigan, Maine, Indiana, Idaho, and Hawaii. Figure 1.3 shows the geographic distribution of my SK&A data.

### **2.4.3 Identifying primary care physicians**

I assign each beneficiary-year to the physician the beneficiary attends most frequently in an non-inpatient setting for evaluation and management services. I call this physician the beneficiaries “primary care physician” (PCP) for the year if the beneficiary has at least three claims with that physician during the year. If a beneficiary has less than three claims with any single physician in a given year, I consider the beneficiary to have no PCP that year, and drop the beneficiary-year from the sample.

### 2.4.4 Identifying referrals in the Medicare data

I use two separate methods to identify two types of referrals in the Medicare data: referrals from PCPs to specialists, and referrals from PCPs to inpatient hospital settings. I do not consider referrals to outpatient hospital settings because independent physician practices are often reclassified as outpatient clinics when they are acquired by a hospital.

To identify referrals from PCPs to specialists I exploit the referring physician and procedure-performing physician variables in the Medicare Carrier Files. Specifically, I treat a claim in the Carrier File for year  $t$  as a referral of beneficiary  $i$  to specialist  $h$  by PCP  $j$  if all of the below conditions hold:

1.  $i$  is the beneficiary on the claim
2.  $i$ 's PCP, (enumerated by  $j$ ) is listed as the referring physician on the claim
3. physician  $h$  is listed as one of the physicians performing a procedure on the claim
4.  $h$ 's recorded speciality in the Medicare data is not a generalist (Family/ General Practice, Internal Medicine, Gerontology), or a speciality that PCPs do not typically refer to directly (Anesthesiology, Critical Care). If the PCP ( $j$ ) is a Cardiologist<sup>1</sup>,  $h$ 's recorded speciality in the Medicare data is not Cardiology.
5. beneficiary  $i$  does not have a prior claim with specialist  $h$  during year  $t$

To identify “referrals” from PCPs to inpatient hospital settings I combine information on the timing of PCP visits from the Carrier Files with information on the timing of inpatient hospital visits from the MedPAR Files. In most instances PCP referrals are to specialists

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<sup>1</sup>Recall that according to my PCP definition any specialty can theoretically serve as a PCP but generalists and Cardiologists were the most common specialties to actually act as PCPs in the Medicare data. As a result I only purchased data on physician characteristics (including integration status) for generalists and Cardiologists from SK&A and hence PCPs from other specialties (and their associated beneficiary-years) are dropped from the sample.

rather than the hospitals they operate from. Think of this variable as a proxy for the count of referrals to specialists operating out of a particular hospital. I count a (PCP visit)-(inpatient hospital admission) pair as a referral of beneficiary  $i$  from PCP  $j$  during year  $t$  if the following conditions hold:

1. beneficiary  $i$  visited hospital  $h$  within 0-30 days of a visit to their PCP,  $j$
2. the PCP visit occurred in year  $t$
3. the beneficiary did not arrive at the hospital via the emergency department
4. patient  $i$  had not previously visited hospital  $h$  during year  $t$  (or January of year  $t + 1$ )
5. patient  $i$  did not visit another hospital between the PCP visit and the visit to hospital  $h$

### 2.4.5 Measures of propensity to refer

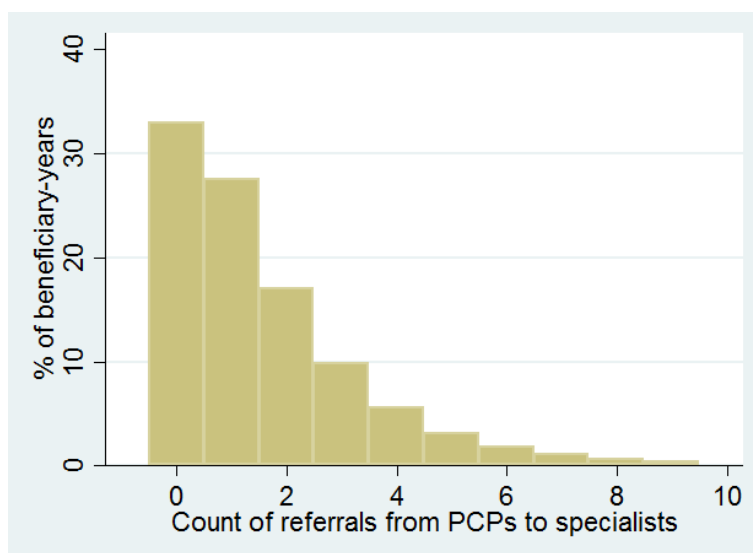
To test hypothesis 2.1 (propensity to refer) I define a PCP's "propensity to make referrals" as the number of referrals for each patient who uses the physician as their PCP. An alternative definition for propensity to refer would be the probability of a patient's visit to the PCP resulting in a referral. However, it is possible that due to the higher cost of attending an integrated physician, patients of integrated PCP could wait until they are sicker before seeing a physician. Such waiting may lead to the "correct" level of referrals per visit being higher after integration, which would bias the estimated effect of integration on PCP's propensity to refer upwards.

I construct three measures of a PCP's propensity to refer: a count of each patient's referrals to all specialists, a count of each patient's referrals to Cardiologists, and a count of each patient's referrals to hospital inpatient settings. Each of these has certain advantages and disadvantages. First, the count of referrals to specialists has the greatest amount of

variation. The distribution of the inpatient referral count variable is close to binary with 92.69% of beneficiary-years having zero inpatient referrals, 6.24% of beneficiary-years having one inpatient referral, and 0.87% having two inpatient referrals. The Cardiologist referral count measure has the next most variation (see Figure 2.2) and the specialist referral count measure has the greatest amount of variation of the three (see Figure 2.1). Second, the count of referrals to all specialists and the count of referrals to inpatient hospital settings are both defined for both generalist and Cardiologist PCPs, whereas the count of referrals to Cardiologists is only defined for generalist PCPs. Third, inpatient visits have the disadvantage of being indirect proxies for actual PCP referrals. PCPs generally do not refer patients to inpatient hospital departments. Rather they may refer them to an outpatient specialist who then refers them to the inpatient department. Fourth, the specialist and Cardiologist referral counts have the advantage of capturing referrals where the patient was not able to see the patient within 30 days – something not unheard of in capacity constrained specialties or for non-urgent visits – but the specialist referral measure relies on the procedure performing physician coding in the referring physician. The inpatient referral count misses referrals that do not occur rapidly. However, it has the advantage of capturing referrals for which the referred to physician does not record a referring NPI. Another small advantage of the inpatient referral count is that the referrals are able to be attributed to the year in which they were made, whereas the specialist and Cardiologist referral counts are obliged to attribute referrals to the year the resulting specialist visit occurred because the referring-physician variable has no associated date. For the propensity to refer hypothesis (2.1) the count of referrals to specialists is the best dependent variable.

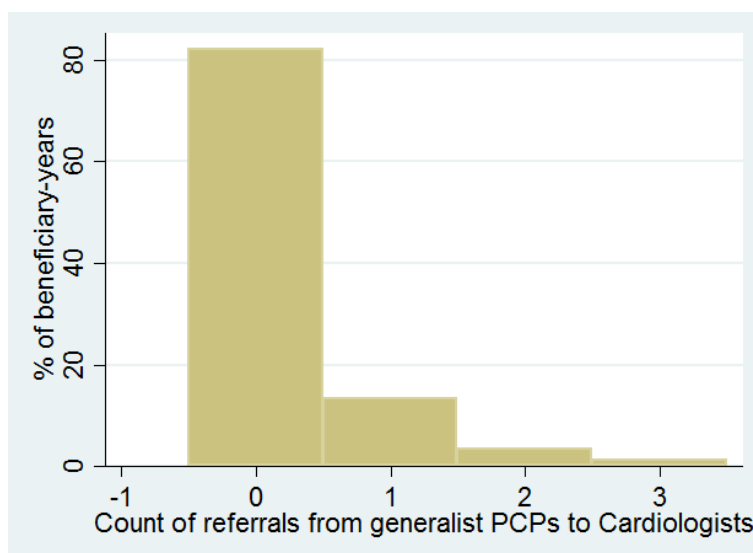


Figure 2.1: Distribution of count of referrals from PCPs to specialists per beneficiary-year



The top 1% of counts are excluded due to Medicare's censoring rules. PCPs are mostly generalists but may also be Cardiologists but if the PCP is a Cardiologist referrals to Cardiologists are excluded from the count of referrals to specialists.

Figure 2.2: Distribution of count of referrals from generalist PCPs to Cardiologists per beneficiary-year



The top 1% of counts are excluded due to Medicare's censoring rules.

### 2.4.6 Dependent variable for hypotheses 2.2 (redirection to owner) and 2.3 (heterogeneity in redirection)

The principal advantage of using inpatient referrals and Cardiologist referrals over all specialist referrals, is that I can determine whether the referrals are to inpatient departments/Cardiologists, linked to the PCP practice by shared ownership by a hospital. This feature of inpatient and Cardiologist referrals allows me to test hypotheses 2.2 (redirection to owner) and 2.3 (heterogeneity in redirection).

For the referrals to inpatient hospital settings tests of hypotheses 2.2 (redirection to owner) and 2.3 (heterogeneity in redirection), my data consists of all occasions on which a beneficiary was referred to an inpatient hospital setting by their PCP (according to the definition of PCP to inpatient hospital referrals I defined above), interacted with all hospitals in their choice set (approximated by the set of hospitals within 100 miles – distances are determined as described in Section 2.4.11<sup>2</sup>). A 100 mile boundary is on the larger end of what is usually utilized in the literature for a hospital choice set. However, I examine the sensitivity of my results to excluding PCP-hospital pairs with no variation in the outcome variable, which excludes PCP-hospital pairs that never share a referral. My dependent variable is a binary indicator for whether a particular PCP-visit results in a referral to a particular hospital,  $h$ .

For the referrals to Cardiologists tests of hypotheses 2.2 (redirection to owner) and 2.3 (heterogeneity in redirection), my data consists of all occasions on which a beneficiary was

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<sup>2</sup>Technically I am using the set of hospitals that are within 100 miles both of the PCP site and the beneficiary's home. Since the patient's choice of PCP is likely endogenous, it would be preferable to use only the distance from the beneficiary's home to the hospitals when forming the hospital-choice-set. However, it is far more computationally intensive to construct the choice set in this way and several factors mitigate my concerns about this affecting the main results. First, patients rarely travel far to their PCP so the distance from a beneficiary's PCP's practice to each hospital should be highly related to the distance from the beneficiary's home to each hospital. Second, >95% of referrals to hospitals within 100 miles of the beneficiary's PCP's practice are to hospitals <55 miles away from the beneficiary's home (see Figure B.1). Finally, in some of my regressions, I drop PCP-hospital pairs with no shared referrals from the sample.

referred to a Cardiologist by their PCP, interacted with all Cardiologists in their choice set (approximated by all Cardiologists within 100 miles – distances are determined as described in Section 2.4.11). My dependent variable is a binary indicator for whether the referral was made to a particular Cardiologist  $h$ .

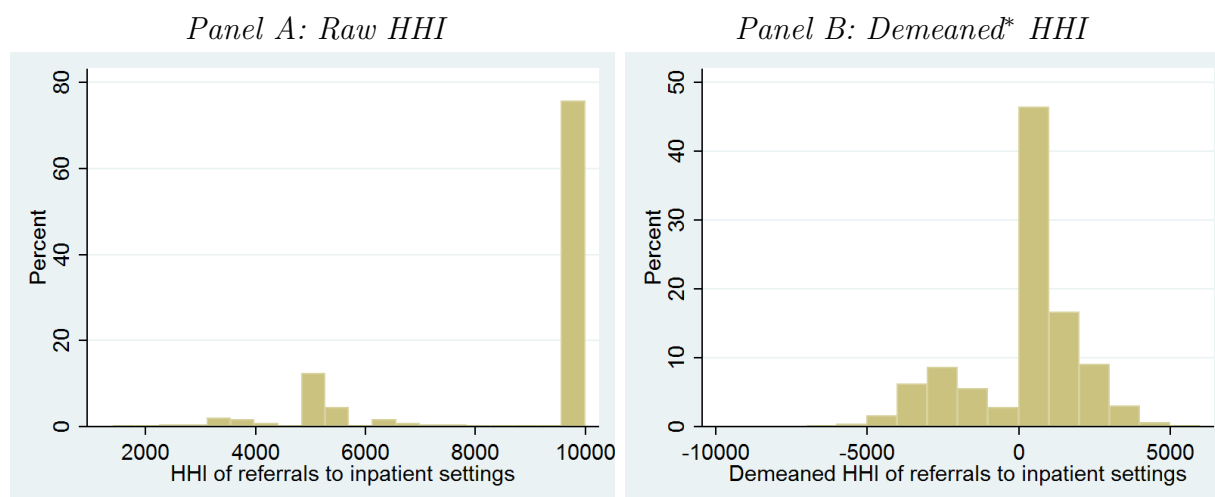
### 2.4.7 Referral concentration measures

To test hypothesis 2.4 I require measures of referral concentration. I construct a measure of referral concentration separately for inpatient referrals and for each specialist type. Since uncommon specialties will not have much variation in referral concentration, I limit the outpatient specialty types to specialties that account for  $\geq 2\%$  of the referrals: Cardiology, Dermatology, Diagnostic Radiology, Gastroenterology, General Surgery, Gynecology, Hematology, Nephrology, Neurology, Oncology, Ophthalmology, Orthopedic Surgery, Otolaryngology, Pathology, Physical Medicine and Rehabilitation, Psychiatry, Pulmonary Disease, and Urology. Let  $s_{jkt}^K$  be the percentage of PCP  $j$ 's type  $K$  referrals that were to specialist  $k$  during year  $t$ . I use the Herfindahl-Hirschman Index (HHI) formula to aggregate these shares into a concentration measure (see equation 2.1).  $C_{jt}^K$  is the concentration index for PCP  $j$ 's referrals to provider type  $K$  during year  $t$ . Since I use percentage shares, the theoretically possible values for  $C_{jt}^K$  are in the range  $(0, 10000]$ .

$$C_{jt}^K = \sum_{k \in K} (s_{jkt}^K)^2 \quad (2.1)$$

The distribution of the inpatient concentration variable is left skewed and closer to binary than normal with substantial clumping around 5,000, which is equivalent to splitting referrals evenly between two hospitals, and 10,000, which represents sending all referrals to a single hospital (see Figure 2.3 Panel A). The demeaned version of the variable, which is what the PCP fixed effects regression uses, is closer to normal but with substantial clumping at zero

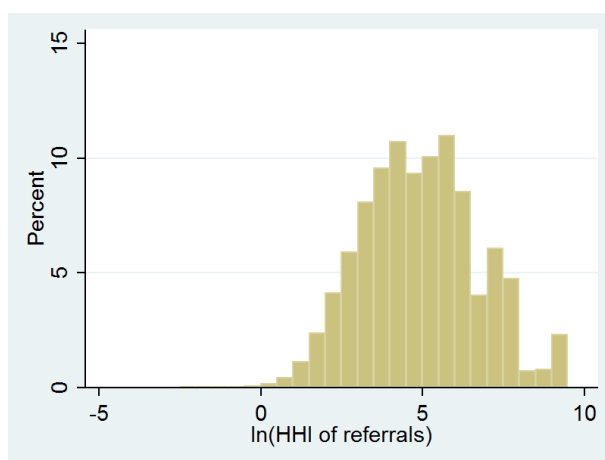
Figure 2.3: Distribution of HHI of inpatient referrals



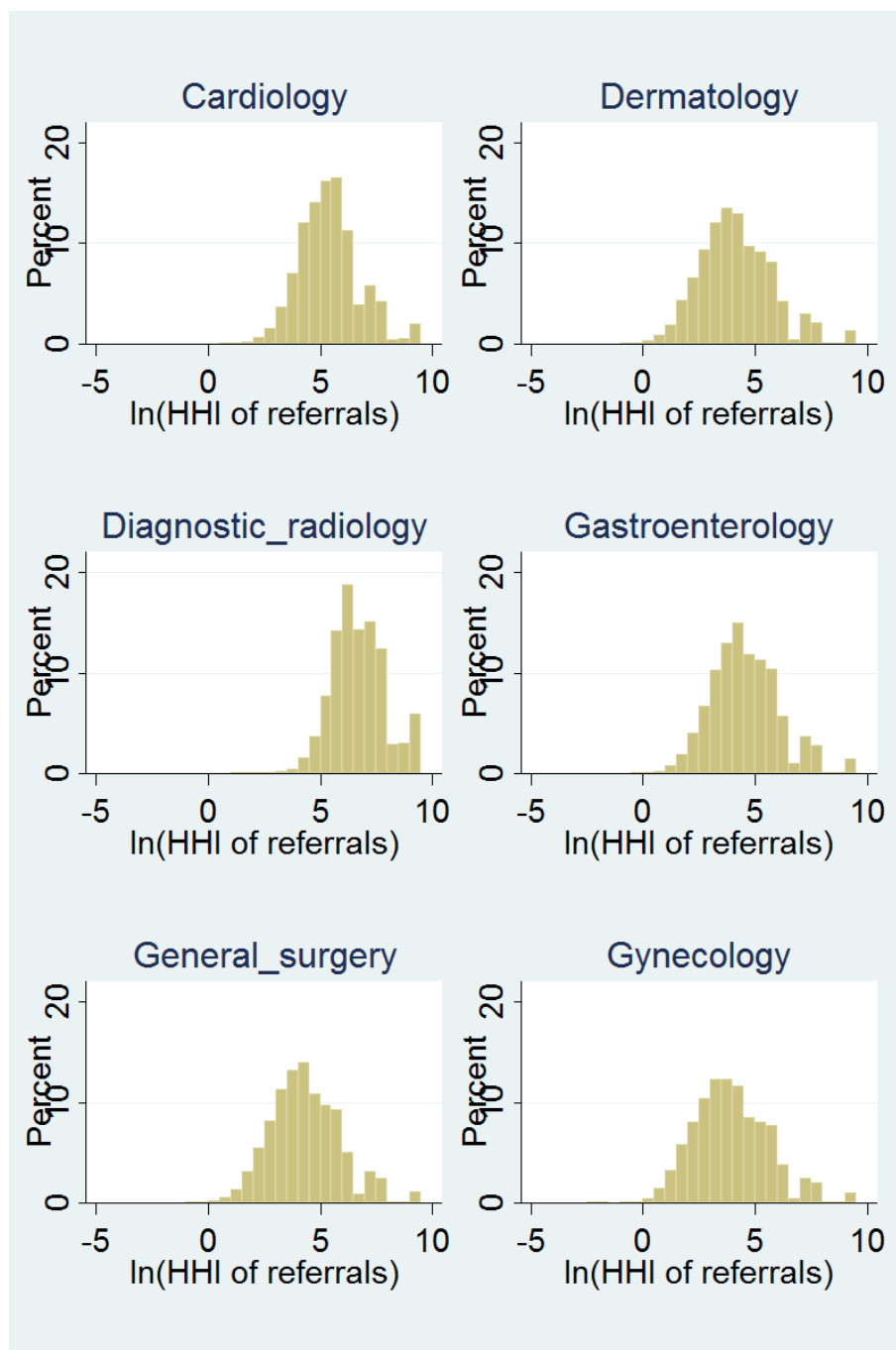
Each observation is a PCP-year. \*demeaning is within a PCP.

(see Figure 2.3 Panel B).

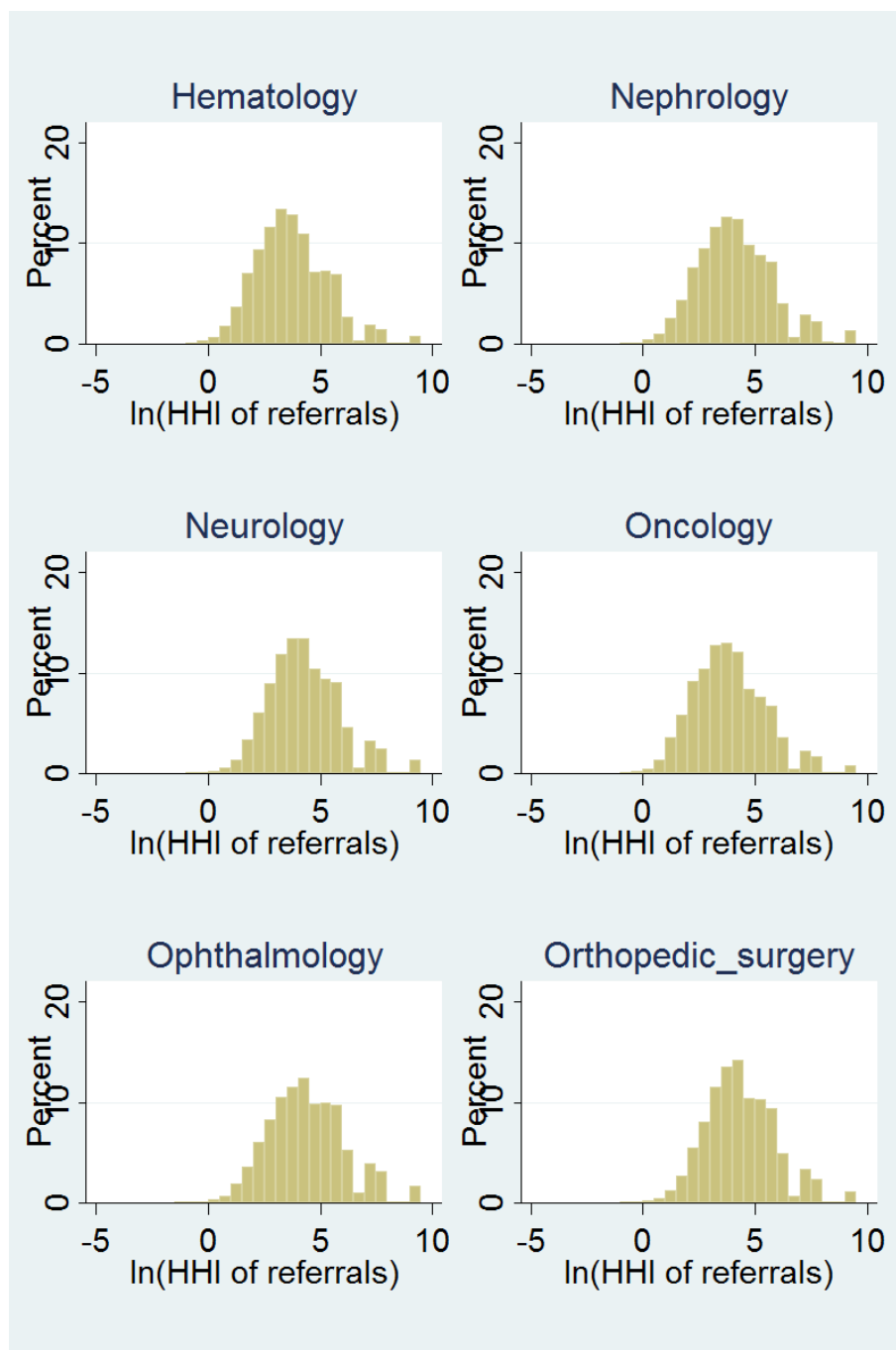
The distributions of the specialist concentration variables are right skewed but their logarithms are close to normally distributed (see Figures 2.4 to 2.7). Hence, for testing hypothesis 2.4 among specialist I use the natural logarithm of the concentration measure as the outcome variable in a regression, rather than the raw concentration measure.

Figure 2.4: Distribution of  $\ln(\text{HHI of all specialist referrals})$ 

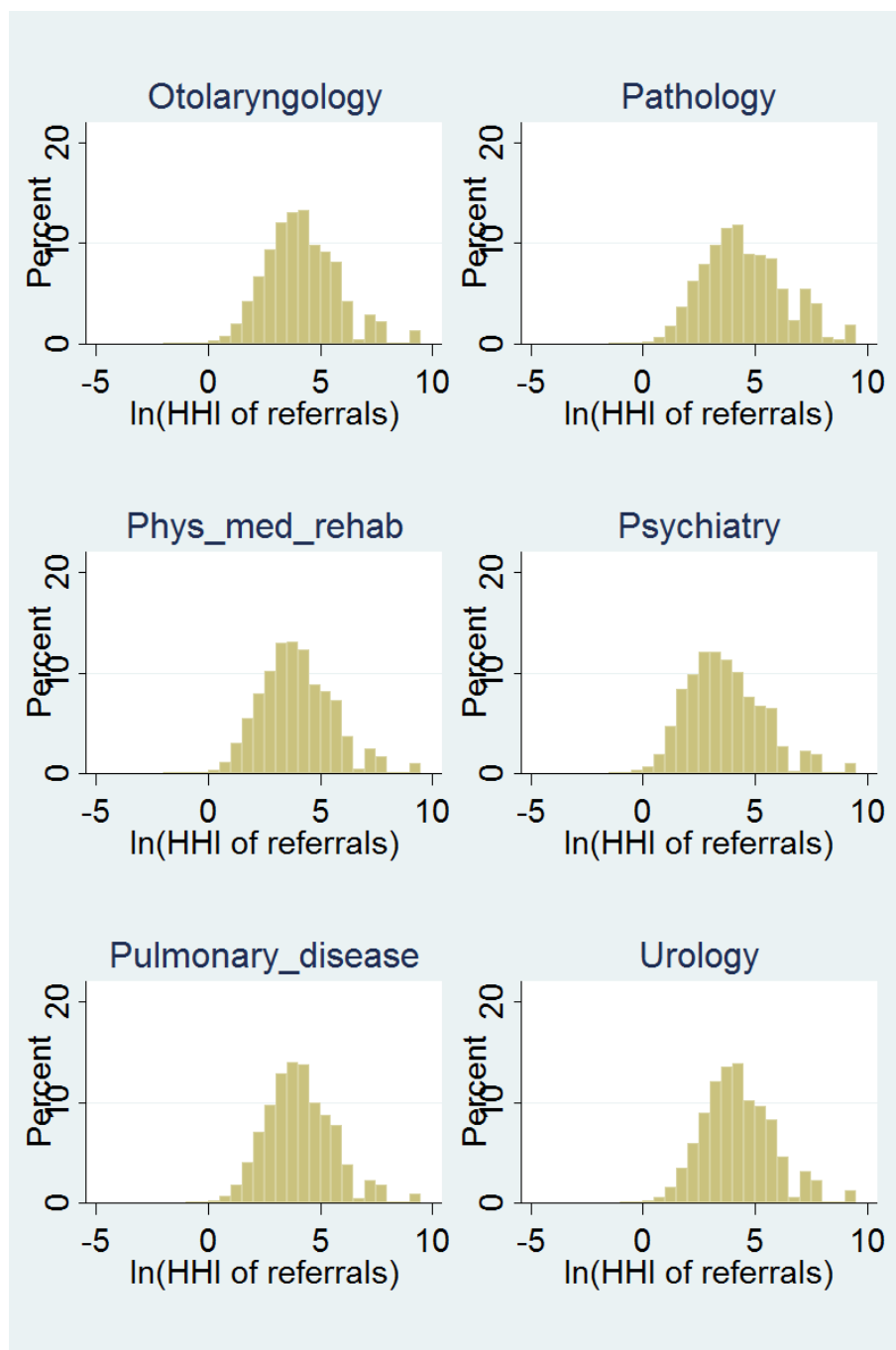
Each observation is a PCP-specialty-year. Sample excludes PCPs that are Cardiologists.

Figure 2.5: Distribution of  $\ln(\text{HHI of specialist referrals})$  - specialty set 1

Each observation is a PCP-year. Sample excludes PCPs that are Cardiologists.

Figure 2.6: Distribution of  $\ln(\text{HHI of specialist referrals})$  - specialty set 2

Each observation is a PCP-year. Sample excludes PCPs that are Cardiologists.

Figure 2.7: Distribution of  $\ln(\text{HHI of specialist referrals})$  - specialty set 3

Each observation is a PCP-year. Sample excludes PCPs that are Cardiologists.

### 2.4.8 Determining which hospital owns which physician practice

To determine the hospital-owner for each physician practice (where applicable) I use data from several sources: the hospital name (in both the SK&A data and the Medicare data), the relative location of the physician practice and the hospital (distances are determined as described in Section 2.4.11), and information on whether the hospital reports jointly with another hospital or under an alternative name from the American Hospital Directory website (<https://www.ahd.com/search.php>). When physician practices list a hospital as their owner in year  $t$  and no hospital-owner in year  $t + 1$  I assume that the hospital-owner in year  $t + 1$  is the same as the hospital-owner in year  $t$ . Note that this assumption applies to *physician practices* not the individual physicians operating therein. I map the practice-years to physician-years using the physician-year to practice mapping I developed in Chapter 1. The method for matching physicians to hospital-owners is described in further detail in Appendix B.2. About 15% of physician-years are affiliated with more than one physician practice. When one of the practices is integrated with a hospital and another is not integrated with any hospital (this affects  $\sim 2\%$  of physician-years), I drop the physician-year and its affiliated patients from the sample. When all the practices are integrated but not all with the same hospital (affects just 0.15% of physician-years) I keep the physician-year in the sample and it simply has more than one hospital in its choice set for which the shared ownership indicator is one. At the end of this process I have a list of all physicians and all the hospitals in their choice set each year, along with an indicator that is one if the hospital in the choice set owns the physician's practice during the year.

I construct a number of other indicators using this physician-hospital-year level data. I construct an indicator for whether each physician is owned by any hospital, which varies at the physician-year level. I also construct an indicator for whether each hospital owns any PCP practices, which varies at the hospital-year level.<sup>3</sup>

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<sup>3</sup>The count of how many PCPs are owned by each hospital in a given year is highly right-skewed and has



I also use the physician-year to hospital-owner mapping to construct indicators for whether each generalist-Cardiologist pair shares a hospital-owner (varies at the generalist-Cardiologist-year level), whether each generalist shares a hospital-owner with one or more Cardiologists (varies at the generalist-year level), and whether each Cardiologist shares a hospital-owner with one or more generalists (varies at the Cardiologist-year level).

### **2.4.9 ZIP code level income data**

I use ZIP code level income data from Internal Revenue Service (2014) from 2006-2013. ZIP codes with low populations are masked in the data. In other ZIP codes I calculate average household income based on total adjusted gross income in the ZIP code divided by the number of returns. One of the censoring rules that the IRS follows drops returns that represent 75% of the total value of returns in their ZIP code and income class. This should alleviate concerns about high outliers biasing averages upward. I merge this data into the Medicare data using the beneficiary ZIP codes listed in the Medicare Beneficiary Summary File.

### **2.4.10 American Hospital Association survey data**

I have data from the American Hospital Association (AHA) survey in even years from 2006 to 2014. I use this data to determine hospital location (latitude and longitude) and hospital bed count. The hospital location I use to determine the hospital choice set and to construct a control variable – distance to the hospital from the beneficiary’s ZIP code centroid. I use both distance to the hospital and the logarithm of the hospital bed count as control variables when testing the hypotheses about where patients are referred to (hypotheses 2.2 and 2.3). Many other interesting hospital characteristics are not time variable so I do not include them

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a lot of variation on the extensive margin – 56.18% of hospital-years do not own any PCPs.

as control variables due to having physician-hospital fixed effects in the regressions for these hypotheses.

### 2.4.11 Determining locations and distances

ZIP codes are postal codes that do not have publicly available locational data. The U.S. Census Bureau develops what they call “ZIP Code Tabulation Areas” (ZCTAs) by sticking together census blocks which have the same modal ZIP codes ({U.S. Census Bureau}, 2015). Since most census blocks are quite small – there were 11,078,297 of them in the 2010 census<sup>4</sup> – the ZCTAs approximate the locations of ZIP codes. I use the ZCTA centroid latitudes and longitudes from the 2010 Census Gazetteer File to approximate the location of each beneficiary’s home (in conjunction with the beneficiary’s five-digit ZIP code from the Medicare data) and the location of each physician practice (in conjunction with the practice ZIP codes in the SK&A data). Recall that I have hospital latitudes and longitudes from the AHA survey. To determine distances between beneficiary-(physician practice) pairs and beneficiary-hospital pairs, I use the Stata command “geodist”, which calls Vincenty’s (1975) equations.

### 2.4.12 Constructing additional control variables from the Medicare data

The Medicare claims data contain information that allows me to construct a number of beneficiary and beneficiary-year level control variables. I get age, sex, and race indicators directly from the Medicare data.

I construct indicators for chronic conditions from the claims data using the Chronic Conditions Data Warehouse (CCDW) algorithm.<sup>5</sup> I also include a count of chronic conditions,

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<sup>4</sup><https://www.census.gov/geo/maps-data/data/tallies/tractblock.html>

<sup>5</sup>I do not include Alzheimers Disease as it is also included in the condition “Alzheimer’s Disease and

since people with multiple chronic conditions are likely to require more referrals. The CCDW algorithm detects chronic conditions by considering claims from previous years (usually the 2 previous years). Hence, if physicians that code less (more) intensively also tend to integrate the chronic conditions indicators and count will underestimate (overestimate) how sick a patient is and hence how appropriate a referral would be for that patient, which could bias the results. To address this I include a measure of how intensively the beneficiary's PCPs from the previous 2 years were at coding claims as a control variable ("the previous coding intensity variable"). I take the average diagnoses per claim for each physician-year in the data and then my measure of previous PCP coding intensity is the maximum of the average diagnoses per claim for the beneficiary's PCP in year  $t - 1$  and in year  $t - 2$ .

I construct a binary variable that takes a value of one if Medicaid covered part of the beneficiary's Medicare premium and zero otherwise. This is effectively an indicator for the beneficiary being very poor (having income  $<100\%$  of the federal poverty line (Centers for Medicare and Medicaid Services, 2016)). However, it is not a pure income indicator since Medicaid covers the 20% coinsurance that Medicare patients typically have.

I also construct a few variables that capture physician characteristics from the Medicare data. I determine physician specialities based on the specialty they bill under most frequently in the Medicare data. I construct the logarithm of the total number of 5% sample Medicare patients the physician sees during the year from the Medicare data.

### **2.4.13 Constructing additional control variables from the SK&A data**

I get the number of physicians at each practice from the SK&A data. Since I only have SK&A survey data in odd years, for even years I interpolate the number of physicians at the

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Related Disorders or Senile Dementia." I also exclude Endometrial Cancer since the percentage of patients with Endometrial Cancer is extremely small.

site. If the physician practiced at the same site in the two adjacent odd years, I take the average of the number of physicians at the site in these two years. If the physician did not practice at the same site in the two adjacent odd years, I treat the number of physicians at the site as missing.

I also use the SK&A data to construct indicators for whether each generalist-Cardiologist pair shares a site, whether each generalist shares a site with one or more Cardiologists, and whether each Cardiologist shares a site with one or more generalists.

## 2.5 Estimation strategy

### 2.5.1 Testing hypothesis 2.1 (propensity to refer)

I run beneficiary-year level regressions with year fixed effects but cluster standard errors at the PCP level. My outcome variables are the counts of referrals to all specialists, referrals to Cardiologists, and referrals to hospital inpatient settings, as outlined in Section 2.4.5. My explanatory variable of interest is whether the patient's PCP is integrated with a hospital. However, this variable is likely endogenous due to two factors: (1) Some physicians may be more willing to be employees than others, and some physician practices may be more appealing acquisition targets for hospitals. This could be correlated with the physician's tendency to refer patients to specialists and/ or hospitals. (2) The patient composition of a PCP could change when the physician integrates, which could affect the likelihood that the physician refers their patient onwards.

If PCPs that tend to integrate with hospitals also tend to refer more patients to specialists and hospitals this will bias the estimated effect of integration on referrals upward. To help address this I include observable physician characteristics as control variables where possible. I include an indicator for the PCP being a Cardiologist (except in the referrals to Cardiologists regressions where Cardiologist PCPs are excluded from the sample). I include

the logarithm of the total number of 5% sample Medicare patients the PCP sees during the year, and the logarithm of the total number of physicians at the PCP's practice.

However, PCPs may have other characteristics that influence both their tendency to make referrals and their tendency to make referrals that I cannot see in the data. To address time-invariant unobserved physician characteristics I add PCP fixed effects to the model (when I do so the indicator for the PCP being a Cardiologist drops from the model). I must assume that there are no important time-varying unobserved physician characteristics. The PCP fixed effects model requires the assumption that in the absence of integration, changes in the amount of referrals (conditional on observed patient and physician characteristics) would follow the same trend between the integrating and non-integrating physicians. It would be an issue for identification if physician practices change their referral patterns to make them more attractive to potential hospital buyers. However, this would tend to bias the estimated effect of integration of referrals toward zero. It would also be an issue if the integration status of a particular PCP affects the tendency of other PCPs to make referrals.

To address changes about the patient composition of a PCP changing when they integrate I first include detailed beneficiary and beneficiary-year level control variables: the variables described in Section 2.4.12, the natural logarithm of average household income in the beneficiary-year's 5-digit ZIP code (see Section 2.4.9), and the natural logarithm of the distance (in miles) from beneficiary's home to the nearest hospital (see Section 2.4.11).

It is still possible that the composition of patient unobservables could introduce bias to the estimated effect of PCP-hospital integration on referrals from PCPs. For instance suppose that patients who have particularly complicated health in ways not captured by the observed patient characteristics both prefer integrated physicians and require more specialist care, this would bias the estimated effect of integration on referrals upwards even in the models with physician and year fixed effects. To address this I instrument for the current integration status of the beneficiary's current PCP with the current integration status of

the beneficiary's original PCP, as I did in Chapter 1. To address the fact that patients can see PCPs initial characteristics when they first choose them I also add the original characteristics of the original PCP to the set of control variables. Validity of the instrument requires excluding beneficiaries who initially chose an integrated physician. The strength of this instrument relies on patient inertia in PCP choice, which is extremely strong in the elderly population (Robinson, 1997, p. 17). In Chapter 1 I showed that after 6 years more than 60% of the Medicare FFS patients  $\geq 68$  who initially chose an independent physician were still with their original physician (refer to Figure 1.5).

The credibility of the model described above relies on Assumptions 1-5.

1. Unobserved physician characteristics and their effect on referral rates do not vary over time.
2. Any changes in referral rates attributable to changes in external factors (such as policy changes) are captured by the year fixed effects.
3. The effect of hospital-ownership on PCP propensity to refer does not depend on the year of integration.
4. The current integration status of the original PCP is unrelated to past, current, and future patient unobservables conditional on observables (i.e. strong exogeneity of the instrument).
5. The integration status of one physician does not affect the referrals of other physicians.

Assumption 4 follows from a number of other assumptions:

- (i) Initial patient unobservables are not clustered on physician unobservables
- (ii) Physicians do not integrate on the basis of their patients' unobservables

- (iii) Unobserved characteristics of the original PCP only impact future appropriateness of referrals via next period observables

Assumption 1 would be violated if physicians' propensity to refer changes over time within a physician. For instance if newly trained physicians are highly inclined to refer patients onward but stop doing so as they become more confident. Assumption 2 could be violated if there was some state specific policy shock that made physicians more or less likely to refer patients onward. For instance if a damage cap for medical malpractice was implemented in a particular state during the sample period, physicians in that state might reduce cautionary referrals. If Assumption 3 is violated, the estimated effect of integration on referral counts from my model will still reflect the average effect of integration over all integrating PCPs in the sample. If the integration status of a particular PCP,  $j$  causes surrounding unintegrated PCPs to reduce their referrals, a violation of Assumption 5, this lowers referrals in the control group relative to the treatment group, which would lead to an overestimate of how much PCP-hospital integration increases referrals from the PCP. This could occur in a situation where the specialists or hospitals are capacity constrained so an increase in referrals from PCP  $j$  would increase wait times at the specialists or hospitals, which could cause non-integrated PCPs to perform more procedures in-house. I do not expect this to be a large problem since PCP-hospital integration occurs more frequently in capacity slack areas. If the integration status of a particular PCP,  $j$  causes surrounding unintegrated PCPs to increase their referrals, this would lead to an underestimate of how much PCP-hospital integration increases referrals from the PCP. This could occur if PCPs believe their patients will choose a new PCP if they are not referred often.

Formally, my model can be described by equation 2.5.1. Let  $i$  and  $t$  enumerate beneficiaries and years respectively. Each beneficiary-year,  $it$ , is associated with a single PCP,  $j = \mathcal{J}(i, t)$ . Let  $r_{it}$  be the count of referrals by PCP  $j$  for beneficiary  $i$  in year  $t$ .  $v_{jt}$  is an indicator that is one if PCP  $j$  is integrated with a hospital in year  $t$  and zero otherwise.

$\mathbf{x}_{it}$  is a vector of observed beneficiary characteristics.  $\mathbf{w}_{jt}$  is a vector of observed physician/physician practice characteristics. Let  $\tau$  be the first year beneficiary  $i$  appears in the data, and  $q = \mathcal{J}(i, \tau)$  be the beneficiary's physician the first year the beneficiary was in the data.  $\phi_t$  and  $\delta_j$  are the year and PCP fixed effects respectively.  $\xi_{it}$  is the effect of the unobserved patient characteristics.  $\phi_t$ ,  $\delta_j$ , and  $\xi_{it}$  may all be correlated with  $v_{jt}$ .  $e_{it}$  is a random shock that is not correlated with  $v_{jt}$ .  $e_{it}$  is distributed iid  $N(0,1)$ .

$$r_{it} = \alpha + \beta v_{jt} + \gamma \mathbf{x}_{it} + \eta \mathbf{w}_{jt} + \theta \mathbf{w}_{q\tau} + \phi_t + \delta_j + \eta_{it} + e_{it} \quad (2.2)$$

The instrument can be written as  $z_{it}=v_{qt}$ . Assumption 4 corresponds to  $\text{corr}(v_{qt}, \xi_{is}) = 0$  for all  $q, t, i, s$ . The test of hypothesis 2.1 technically corresponds to a test of  $\beta > 0$  though I test instead  $\beta \neq 0$  to allow for the possibility that integration in fact causes PCPs to decrease the amount of referrals they make.

Similarly to the previous chapter, the distributions of the outcome variables suggest a non-linear model may be more appropriate than a non-linear model, since the distribution of the outcome variables suggest that  $e_{it}$  is unlikely to be normally distributed in reality. However, the small number of beneficiary-years within each PCP<sup>6</sup> makes the incidental parameters problem a concern in models with PCP fixed effects. Furthermore, the small number of beneficiary-years within each PCP creates computational difficulties – particularly in models with both fixed effects and an instrument. I show evidence in Appendix B Section B.3 that my use of linear models is unlikely to be driving my conclusions.

As a preliminary step for testing hypothesis 2.3, I also examine whether the effect of attending an integrated PCP on referral counts varies by beneficiary income. To do so I introduce an interaction between the indicator for the PCP being owned by a hospital, and the income proxy.

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<sup>6</sup>For instance for the hospital referral count variable, among the PCPs that have variation in their outcome variable, the mean number of beneficiary-years within the PCP is 20.



### 2.5.1.1 Leads and lags regressions

To check for pre-trends and to determine whether the effect of integration on propensity to refer grows or disperses as time passes after the acquisition, I replace the indicator for the patient's PCP being integrated with a hospital in year  $t$ ,  $v_{jt}$ , with a set of indicators that capture the number of years since the PCP first integrated. I use negative numbers to denote years prior to integration. I group years since integration into pairs to retain statistical power. Let  $v_{jt}^{\{a,b\}}$  be an indicator that takes a value of one if PCP  $j$  was first integrated in year  $t - a$  or in year  $t - b$ . My leads and lags regression equation is

$$r_{it} = \alpha + \beta \cdot \begin{pmatrix} v_{jt}^{\{-6,-5\}} \\ v_{jt}^{\{-4,-3\}} \\ v_{jt}^{\{-2,-1\}} \\ v_{jt}^{\{2,3\}} \\ v_{jt}^{\{4\}} \end{pmatrix} + \gamma \mathbf{x}_{it} + \eta \mathbf{w}_{jt} + \theta \mathbf{w}_{q\tau} + \phi_t + \delta_j + \eta_{it} + e_{it}. \quad (2.3)$$

I instrument for the set of integration variables from the current PCP with the set of integration variables from the PCP the beneficiary chose when they first entered the data.

Since PCPs who are independent in 2013 may never integrate I exclude patients with PCPs who remain unintegrated in 2013 from the leads and lags regression. It is also impossible to tell how many years since a PCP first integrated if the PCP is integrated the first year they enter the dataset so I also exclude beneficiaries with a PCP that is integrated the first year the PCP entered the dataset – this exclusion results in a relatively small incremental number of beneficiaries being excluded from the sample because most beneficiary-years where the PCP has first entered the dataset are also the first year the beneficiary has entered the dataset and beneficiaries who initially chose an integrated PCP are already excluded from the dataset. Due to the presence of PCP fixed effects in equation 2.5.1 identification of the

effect of integration in the main referral propensity regression is driven by the subsample that will be used in the leads and lags regression. However, the additional PCPs included in the main regression do improve the precision of the estimated coefficients of the control variables.

## 2.5.2 Testing hypothesis 2.2 (redirection to owner)

### 2.5.2.1 Testing using inpatient referrals

I am able to test this hypothesis 2.2 using the inpatient and Cardiologist referrals but not the pooled specialists referrals, since I do not have data on hospital ownership of specialist practices, other than for Cardiologists.

For the inpatient referrals regressions, each observation consists of a (PCP-visit)-hospital pair. Each PCP-visit is affiliated with a single beneficiary-year,  $it$ , but there may be multiple PCP-visits for each beneficiary-year. I index the PCP visits by  $it\ell$  where  $\ell$  distinguishes between multiple visits within a beneficiary-year  $it$ . I enumerate hospitals with  $h$ .

I model the probability of a PCP-visit ( $it\ell$ ) resulting in a referral to hospital  $h$  conditional on the PCP-visit resulting in a referral to any hospital, as a function of the whether the PCP ( $j$ ) is owned by hospital  $h$ ; whether the PCP ( $j$ ) is owned by a hospital other than  $h$ ; whether hospital  $h$  owns any PCP practices; and an interaction between the latter two terms (refer to Section 2.4.8 for information on how I construct these variables); beneficiary ( $i$ ) and beneficiary-year characteristics as described in Section 2.4.12; the natural logarithm of average household income in the beneficiary-year's 5-digit ZIP code (see Section 2.4.9); the distance from beneficiary  $i$ 's home to hospital  $h$  (see Section 2.4.11), the logarithm of the number of beds hospital  $h$  has, and time fixed effects. I include PCP-hospital fixed effects to account for persistence in PCP preferences.

I assume that beneficiaries make the decision about which PCP to see but that the PCP

chooses the hospital on the beneficiaries' behalf. This allows me to treat the indicator for the hospital owning one or more PCPs as exogenous. Since a patient may select a PCP based on the PCP's affiliations and their own unobserved characteristics, I instrument for the PCP integration variables, with their analogues for beneficiary  $i$ 's originally chosen PCP. Using the originally chosen PCP for the instruments necessitates dropping patients who originally chose an integrated PCP from the sample.

Ideally, in this situation I would use a conditional logit model. Unfortunately, the small number of observations within a panel (PCP-hospital) means the incidental parameters problem is likely to be a problem. Furthermore, the size of the dataset, the number of fixed-effects, and the requirement that the standard errors be bootstrapped in a non-linear model with an instrument, makes estimating a non-linear version of my model computationally infeasible with the equipment I have available. Due to all these factors, I remain with a linear model.

Formally my model can be written as shown in equation 2.4, where  $R_{i\ell ht}$  is a binary value that takes a value of one if beneficiary  $i$ 's PCP visit  $\ell$  during year  $t$  results in a referral to hospital  $h$ , and a value of zero otherwise.  $\lambda_{ht}$  is an indicator for hospital  $h$  owning one or more PCP practices in year  $t$ ,  $d_{ih}$  is the distance between the beneficiary's ZIP code centroid and hospital  $h$ , and  $b_{it}$  is the natural logarithm of the number of beds at hospital  $h$  in year  $t$ .  $\delta_{jh}$  is the PCP-hospital fixed effect term. All other notation is the same as outlined above. Note that  $V_{jht}\lambda_{ht}$  is perfectly correlated with  $V_{jht}$ , since if  $V_{jht} = 1$ , hospital  $h$  necessarily owns at least one PCP. I instrument for the PCP integration variables,  $V_{jht}$ ,  $v_{j-ht}$ , and  $v_{j-ht}\lambda_h$ , with their analogues for beneficiary  $i$ 's originally chosen PCP –  $V_{qht}$ ,  $v_{q-ht}$ , and  $v_{q-ht}\lambda_h$ .

$$\begin{aligned}
 R_{i\ell ht} = & \alpha + \beta_V V_{jht} + \beta_v v_{j-ht} + \beta_\lambda \lambda_{ht} + \beta_{v\lambda} v_{j-ht} \lambda_{ht} \\
 & + \boldsymbol{\gamma} \mathbf{x}_{it} + \boldsymbol{\eta} \mathbf{w}_{jt} + \boldsymbol{\theta} \mathbf{w}_{q\tau} + \psi d_{ih} + \omega b_{it} + \phi_t + \delta_{jh} + \xi_{it} + e_{it\ell h} \quad (2.4)
 \end{aligned}$$

There are five types of PCP-hospital combinations:  $j$  owned by  $h$ ;  $j$  owned by a hospital,

hospital  $h$  does not own PCPs;  $j$  owned by a hospital, hospital  $h$  owns PCPs but not  $j$ ;  $j$  is not owned by a hospital and  $h$  does not own PCPs;  $j$  is not owned by a hospital but  $h$  owns PCPs.

$\beta_V$  tells us how much more likely a PCP is to refer to a particular hospital after they are integrated with it, relative to if the hospital had instead purchased a different PCP.  $\beta_v$  tells us how much more likely a PCP,  $j$ , is to refer to a particular hospital that owns no PCPs after PCP  $j$  is purchased by a hospital, relative to if PCP  $j$  had not been purchased.  $\beta_v + \beta_{\lambda v}$  tells us how much more likely a PCP,  $j$ , is to refer to a particular hospital,  $h$ , that owns other PCPs after PCP  $j$  is purchased by a hospital other than  $h$ , relative to if PCP had not been purchased. The correspondence between Hypothesis 2.2 and  $\beta_V > 0$  is straightforward. Hypothesis 2.2 also corresponds to  $\beta_v$  and  $\beta_v + \beta_{\lambda v} < 0$ .  $\beta_\lambda$  tells us how much more likely an unintegrated PCP is to refer to a hospital that owns other PCPs, relative to a hospital that owns no PCPs.

### 2.5.2.2 Testing using Cardiology referrals

For the referrals to Cardiologists regressions, observations consist of occasions where the beneficiary's PCP referred them to a Cardiologist, interacted with all Cardiologists within the beneficiary's choice set as outlined in Section 2.4.6. The sample excludes any PCPs who are not generalists, and all their affiliated beneficiary-years. Each observation is enumerated by  $it\ell h$  where  $i$  is the beneficiary,  $t$  is the year,  $\ell$  distinguishes between multiple referral occasions within a single beneficiary-year, and  $h$  is a Cardiologist.

A complication that arises in referrals to Cardiologists but not to inpatient settings is that generalist PCPs may share a site with one or more Cardiologists (affects PCP-years corresponding to 4% of observations). PCPs that share a site with a Cardiologist likely refer to them frequently both before and after the site integrates, and PCPs and Cardiologists

at the same site will generally integrate simultaneously with the same hospital<sup>7</sup>. As such, I expect that including PCPs who share a site with a Cardiologist in the sample will bias the estimated effect of integrating with a particular hospital on referrals to Cardiologists integrated with that hospital downward (toward zero). I hence exclude PCPs who share a site with a Cardiologist (in addition to PCPs who are Cardiologists) from the sample.

My model for referrals for PCPs to Cardiologists is similar to my model for inpatient referrals described above. I model the probability of a PCP-visit ( $it\ell$ ) resulting in a referral to Cardiologist  $h$  conditional on the PCP-visit resulting in a referral to any Cardiologist ( $R_{i\ell ht}$ ), as a function of the whether the PCP  $j$  and hospital  $h$  have the same hospital-owner ( $V_{jht}$ ); whether PCP  $j$  is owned by a hospital but Cardiologist  $h$  either independent or owned by a different hospital ( $v_{j-ht}$ ); whether Cardiologist  $h$  is owned by any hospital ( $\lambda_h$ ); whether PCP  $j$  and Cardiologist  $h$  are both owned by hospitals but not the same one ( $v_{j-ht}\lambda_h$ ); beneficiary ( $i$ ) and beneficiary-year characteristics as described in Section 2.4.12; the natural logarithm of average household income in the beneficiary-year's 5-digit ZIP code (see Section 2.4.9); the distance from beneficiary  $i$ 's home to the closest of Cardiologist  $h$ 's physician practices ( $d_{iht}$ ); year fixed effects; and PCP-Cardiologist fixed effects. Similarly, to the inpatient referral model, I assume that beneficiaries make the decision about which PCP to see but that the PCP chooses the Cardiologist on the beneficiaries' behalf, and treat the integration status of the Cardiologist as exogenous. I instrument for the PCP integration variables,  $V_{jht}$ ,  $v_{j-ht}$ , and  $v_{j-ht}\lambda_h$ , with their analogues for beneficiary  $i$ 's originally chosen PCP –  $V_{qht}$ ,  $v_{q-ht}$ , and  $v_{q-ht}\lambda_h$ . The formal definition of my model for which Cardiologists PCP's refer their patients to is the same as equation 2.4, excluding the term for the log of hospital beds ( $\omega b_{it}$ ). Similarly to the inpatient referral regressions, I would ideally use a logit regression but computational concerns and the incidental parameters problem lead me to approximate the situation with a linear model.

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<sup>7</sup>the exception being when either the PCP or the Cardiologist leaves the site

### 2.5.2.3 Testing in a model analogous to Baker et al. (2016)

One of the contributions of my paper is addressing the upward bias in Baker et al.'s (2016) estimate of the effect of a hospital owning a patient's physician on the probability of referring to that hospital conditional on a referral being made. This bias is attributable to hospitals' tendency to acquire physicians that already refer to them often. To identify how much of the difference between Baker et al.'s (2016) estimate and my estimate is attributable to my using a different model to Baker, as opposed to different samples of the Medicare and SK&A data and somewhat different scenarios, I replicate the key features of Baker et al.'s (2016) model using my data sample and scenarios.

***Essential differences between Baker et al.'s data and my data.*** Both Baker et al. (2016) and I rely primarily on the Medicare FFS patient data, the SK&A outpatient physician survey data, and the American Hospital Association (AHA) survey data. However, we have different subsamples of the Medicare and SK&A data. Baker et al. (2016) has an extremely large cross-sectional data set whereas I have panel data that covers a smaller set of patients and hospitals. Baker et al. (2016) use the full sample of Medicare FFS hospital inpatient data and the 20% sample of the "Carrier file" (which contains the physician services) in 2009 only, whereas I use the 5% sample of these datasets over 2007-2013. Baker et al. (2016) have the entire SK&A outpatient physician survey dataset for 2009 whereas I have the SK&A outpatient physician survey dataset for generalists and Cardiologists in selected states (see Figure 1.3 for the specific states) in odd years from 2007 to 2013. Both Baker et al. (2016) and I limit the sample to patients over 65. However, I do not limit my sample to urban residents as Baker et al. does.

***Comparison of Baker et al.'s model and my preferred model.*** My model is linear with PCP-hospital fixed effects (PCP fixed effects interacted with hospital fixed effects) and an indicator that captures whether the physician is integrated with a hospital

other than the hospital in the current observation, whereas Baker et al.'s (2016) model is a conditional logit, or effectively a logit model with “referral occasion” fixed effects. The difference in the choice of fixed effects reflects that Baker is treating each referral occasion as a separate decision, whereas I am treating each referral occasion within a PCP as a repeated observations on the PCP's decisions. I interact the PCP fixed effects with hospital fixed effects to capture the unexplained component of a PCP's preference for a particular hospital. It is the interaction of the PCP fixed effects with the hospital fixed effects that removes the bias due to hospitals' tendency to acquire physicians that already refer to them often. Baker et al. (2016) did not have the option of PCP-hospital fixed effects, since they only had one year of data (2009) and hence no variation in PCP-hospital relationships within a PCP-hospital pair.

An additional consequence of Baker et al. (2016) having only have one year of data is even with just PCP fixed effects (rather than PCP-hospital fixed effects), they would not be able to identify the coefficient on indicator for a physician being integrated with a hospital other than the hospital in the current observation, as within a PCP, it is perfectly correlated (negatively) with the indicator for a physician being integrated with the hospital in the current observation. The referral occasion fixed effects also prevent the inclusion of this variable. Furthermore referral occasion fixed effects also prevent Baker et al. (2016) from identifying the effect of any patient characteristics in their model.

***Differences between Baker et al.'s scenario and my scenarios.*** My definition of a patient's PCP most closely resembles Baker et al.'s (2016)'s second definition of “a patient's physician” – the most frequently attended physician in the carrier file during the 90 days prior to the patient's hospital admission. My PCP definition is the most frequently attended physician for outpatient evaluation and management services during the year that includes the admission/ Cardiologist visit. However, because my sample is limited to PCPs that are generalists or Cardiologists Baker et al.'s (2016)'s third definition of “a patient's

physician” – the most frequently attended *generalist* physician in the carrier file during the 90 days prior to the patient’s hospital admission should also be similar to my PCP definition.

Baker et al.’s (2016)’s outcome variable, an indicator for an admission to a particular hospital within 90 days of a visit to the patient’s physician is closest to my inpatient referral indicator, which captures admissions to a particular hospital excluding those via the emergency department that occur within 30 days of visit to the patient’s PCP.

As mentioned above Baker et al. (2016) treat each referral occasion as an independent decision whereas I treat referral occasions as repeated observations on physician decisions.

Finally, I treat all hospitals within 100 miles as in the choice set, whereas Baker et al. (2016) treats all hospitals within 35 miles *and* all teaching hospitals within 100 miles as in the choice set. In part I use the 100 mile radius for all hospitals because I keep the patients in rural areas in my data set.

***My strategy for replicating the model from Baker et al.*** I use my PCP definition and a variation of my admission indicator that is closer to Baker’s definition. This variation on my admissions indicator allows 90 days to pass between the PCP visit and the admission rather than 30 days. I adopt Baker et al.’s base model specification (see equation 1 of Baker et al. (2016)) as closely as I am able. Unfortunately, I do not have the hospital cost data or the hospital quality data that Baker has so my hospital characteristics are limited to hospital size, an indicator for being a for-profit hospital, an indicator for being a teaching hospital, and the distance from the patient’s ZIP code to the hospital. My dependent variable of interest is an indicator for the patient’s PCP being integrated with the hospital in the current observation. I include an indicator for the hospital owning any PCPs, rather than Baker et al.’s indicator for the hospital owning any physicians. I also interact the indicator for the hospital owning any PCPs with an indicator for the PCP being owned by any hospital. I use a conditional logit model where the data is grouped by referral occasion and cluster the standard errors at the 3 digit patient ZIP code level.



I also present several models that are hybrid's of Baker et al.'s model and my preferred specification. In the first hybrid model I replace the 90 day admissions indicator with the 30 day admissions indicator I use in the rest of the paper as the inpatient referral proxy. In the second hybrid model I cluster standard errors at the PCP level, rather than at the 3-digit ZIP code level. In the third hybrid model I replace the referral occasion fixed effects with PCP-hospital fixed effects. As a result of using these fixed effects some of the hospital characteristics control variables drop from the model, since they do not vary within PCP-hospital. In the final hybrid model I replace Baker's control variables with my control variables.

#### **2.5.2.4 Leads and lags regressions for inpatient referrals redirection**

In almost all cases once a PCP integrates with a hospital, they remain integrated with that hospital. Additionally, extremely few PCP's are integrated with more than one hospital at any given time in spite of many physicians operating from multiple sites. Consequently the time profile of how a PCPs referral locations change in response to being acquired by a hospital can be captured with two items: a vector of indicators that capture the number of years until/ since the PCP integrates/ integrated with *any* hospital, and an indicator that takes a value of one if the PCP *ever* integrates with the hospital in the current observation. I limit my sample to observations where the PCP is not integrated with a hospital when they first enter the data but they integrate over the course of the study period. The vector that captures when the PCP integrates with any hospital will capture the time profile of referrals for PCP-hospital pairs that never integrate, whereas the interaction between this vector and the indicator for the PCP-hospital pair ever integrating will capture the time profile of referrals for PCP-hospital pairs that do integrate. One of the years since integration categories must be excluded. In addition, since the indicator for the PCP-hospital pair ever integrating does not vary within PCP-hospital pair. Therefore in the model with PCP-

hospital fixed effects, the interaction between the indicator for the PCP-hospital pair ever integrating and one of the years since integration indicators must also be excluded. In other words I cannot separately identify effect of the PCP-hospital pair eventually integrating given that the PCP will integrate with some hospital in  $x$  years, from the effect of the PCP-hospital pair never integrating given that the PCP will integrate with some hospital in  $x$  years, where  $x$  is the excluded category. Similarly to the main “referral direction” regressions, I treat the PCP and PCP-hospital integration variables as endogenous but the hospital owns PCPs indicator as exogenous (conditional on observables). My set of instruments is the current values of the PCP and PCP-hospital integration variables for the physician the beneficiary chose as their PCP the first year the beneficiary entered the dataset.

Formally my model can be expressed as follows. Let  $A_{ht}$  be an indicator that takes a value of one if PCP  $j$  is *ever* integrated with hospital  $h$  during the study period. Let  $\mathcal{I}_{jt}$  be a vector of indicators that capture how long PCP  $j$  has been integrated with *any* hospital as at year  $t$ , as shown in equation 2.5.

$$\mathcal{I}_{jt} = \begin{pmatrix} v_{jt}^{\{-6,-5\}} \\ v_{jt}^{\{-4,-3\}} \\ v_{jt}^{\{-2,-1\}} \\ v_{jt}^{\{0,1\}} \\ v_{jt}^{\{2,3\}} \\ v_{jt}^{\{4\}} \end{pmatrix} \quad (2.5)$$

Let  $\widehat{\mathcal{I}}_{jt}$  denote the vector  $\mathcal{I}_{jt}$  with the 1st element removed. The analogue of equation 2.4

that captures pre- and post-trends is hence equation 2.6.

$$\begin{aligned}
 R_{ilht} = & \beta_{\mathcal{I}} \cdot \widehat{\mathcal{I}}_{jt} + \beta_{\mathcal{I}A} \cdot \widehat{\mathcal{I}}_{jt} A_{jh} + \beta_{\lambda} \lambda_{ht} + \beta_{\mathcal{I}\lambda} \cdot \mathcal{I}_{jt} \lambda_{ht} + \beta_{\mathcal{I}A\lambda} \cdot \mathcal{I}_{jt} A_{jh} \lambda_{ht} \\
 & + \gamma \mathbf{x}_{it} + \eta \mathbf{w}_{jt} + \theta \mathbf{w}_{q\tau} + \psi d_{ih} + \omega b_{it} + \phi_t + \delta_{jh} + \xi_{it} + e_{itlh} \quad (2.6)
 \end{aligned}$$

I instrument for the set of endogenous variables  $\{ \mathcal{I}_{jt}, \mathcal{I}_{jt} A_{jh}, \mathcal{I}_{jt} \lambda_{ht}, \mathcal{I}_{jt} A_{jh} \lambda_{ht} \}$  with the set of exogenous variables  $\{ \mathcal{I}_{qt}, \mathcal{I}_{qt} A_{qh}, \mathcal{I}_{qt} \lambda_{ht}, \mathcal{I}_{qt} A_{qh} \lambda_{ht} \}$  where  $q$  denotes the PCP the beneficiary chose the first year the beneficiary entered the data.

$\beta_{\mathcal{I}}$  captures how referrals to the non-PCP-owning hospitals the PCP never integrates with, respond to integration over time (in terms of years until/ since integration).  $\beta_{\mathcal{I}} + \beta_{\mathcal{I}\lambda}$  captures how referrals to the PCP-owning hospitals the PCP never integrates with, respond to integration over time.  $\beta_{\mathcal{I}} + \beta_{\mathcal{I}A}$  captures how referrals to the hospital the PCP does integrate with, respond to the years until/ since integration if the hospital is independent prior to purchasing the PCP.  $\beta_{\mathcal{I}} + \beta_{\mathcal{I}A} + \beta_{\mathcal{I}\lambda} + \beta_{\mathcal{I}A\lambda}$  captures how referrals to the hospital the PCP does integrate with, respond to the years until/ since integration if the hospital currently owns PCPs.

### 2.5.2.5 Leads and lags regressions for Cardiology referrals redirection

Unlike the PCP-hospital relationships, PCP-Cardiology relationships are not usually characterized by a single change within a PCP. Instead the typical situation is that a PCP gains a shared hospital-owner relationship with a Cardiologist in one year (either when the PCP's practice is purchased by a hospital or when the hospital that already owns the PCP's practice first acquires a practice with a Cardiologist) and then the PCP gains further shared hospital-owner relationships with other Cardiologists as the hospital purchases more physician practices with Cardiologists. To accommodate this I replace the indicator  $A_{jh}$  from equation 2.6 with a vector of indicators of the number of years (0-1, 2-3, or 4-5) between the

PCP sharing a hospital-owner with any Cardiologist and the PCP sharing a hospital-owner with the Cardiologist in the current observation. I denote this vector with  $\mathbf{A}_{jh}$ . If the PCP never shares a hospital-owner with the Cardiologist in the current observation all elements of  $\mathbf{A}_{jh}$  take values of zero. The formal expression of my Cardiology referral location model with leads and lags is hence equation 2.7, where  $\mathcal{I}_{jt}$  has dimensions  $6 \times 1$ ,  $\widehat{\mathcal{I}}_{jt}$  has dimensions  $5 \times 1$ ,  $\mathbf{A}_{jh}$  has dimensions  $1 \times 3$ ,  $\beta_{\mathcal{I}\mathbf{A}}$  has dimensions  $5 \times 3$ ,  $\beta_{\mathcal{I}\mathbf{A}\lambda}$  has dimensions  $6 \times 3$ , and  $\mathbf{a} \cdot \mathbf{b}$  is defined to be the sum of the products of the corresponding elements of matrices  $a$  and  $b$  i.e.  $\mathbf{a} \cdot \mathbf{b} = \sum_{i,j} a_{ij}b_{ij}$ .

2.6.

$$\begin{aligned} R_{ilht} = & \beta_{\mathcal{I}} \cdot \widehat{\mathcal{I}}_{jt} + \beta_{\mathcal{I}\mathbf{A}} \cdot (\widehat{\mathcal{I}}_{jt} \times \mathbf{A}_{jh}) + \beta_{\lambda} \lambda_{ht} + \beta_{\mathcal{I}\lambda} \cdot \mathcal{I}_{jt} \lambda_{ht} \\ & + \beta_{\mathcal{I}\mathbf{A}\lambda} \cdot (\mathcal{I}_{jt} \times \mathbf{A}_{jh}) \lambda_{ht} + \gamma \mathbf{x}_{it} + \eta \mathbf{w}_{jt} + \theta \mathbf{w}_{q\tau} + \psi d_{ih} + \phi_t + \delta_{jh} + \xi_{it} + e_{itlh} \quad (2.7) \end{aligned}$$

Theoretically I could instrument for the set of endogenous variables  $\{ \mathcal{I}_{jt}, \mathcal{I}_{jt} \times \mathbf{A}_{jh}, \mathcal{I}_{jt} \lambda_{ht}, \mathcal{I}_{jt} \times \mathbf{A}_{jh} \lambda_{ht} \}$  with the set of exogenous variables  $\{ \mathcal{I}_{qt}, \mathcal{I}_{qt} \times \mathbf{A}_{qh}, \mathcal{I}_{qt} \lambda_{ht}, \mathcal{I}_{qt} \times \mathbf{A}_{qh} \lambda_{ht} \}$  where  $q$  denotes the PCP the beneficiary chose the first year the beneficiary entered the data. However, splitting up integration by timing leads to some of the instruments being weak. This results in such large standard errors that the confidence interval is useless (effectively I can say the effect of integration is between -1 and 1, which is a necessary condition of the outcome being a probability). Consequently, I will present only the results of the fixed effects estimation with the sample limited to eliminate beneficiaries who initially chose an integrated PCP.

### 2.5.3 Testing hypothesis 2.3: heterogeneity of impact on referrals

To test whether physicians are redirecting patients with poor capacity to pay away from their hospital owner, I introduce interactions between the income proxy and the ownership

indicators in the referrals regression described in Section 2.5.2.1. In other words for the inpatient heterogeneity regression I estimate

$$\begin{aligned}
 R_{iht} = & \alpha + \beta_V V_{jht} + \beta_v v_{j-ht} + \beta_\lambda \lambda_{ht} + \beta_{v\lambda} v_{j-ht} \lambda_{ht} + \beta_{VI} V_{jht} I_{it} + \beta_{vI} v_{j-ht} I_{it} \\
 & + \beta_{\lambda I} \lambda_{ht} I_{it} + \beta_{v\lambda I} v_{j-ht} \lambda_{ht} I_{it} + \gamma \mathbf{x}_{it} + \eta \mathbf{w}_{jt} + \theta \mathbf{w}_{q\tau} + \psi d_{ih} + \omega b_{it} + \phi_t + \delta_{jh} + \xi_{it} + e_{itlh}
 \end{aligned} \tag{2.8}$$

where  $I_{it}$  is the natural logarithm of the average household income (in 2015 \$) in beneficiary  $i$ 's ZIP code in year  $t$ .  $I_{it}$  is also one of the elements of  $\mathbf{x}_{it}$ . I include interactions between the income proxy and the PCP ownership indicators as additional instruments. The regression for testing hypothesis 2.3 with the Cardiology referrals is the same as described in equation 2.8 except the term  $\omega b_{it}$  is excluded, and the definitions of the variables are adapted to the Cardiology referral scenario as described above.

#### 2.5.4 Testing hypothesis 2.4: concentration of referrals

I conduct separate regressions for hospital and specialist referrals. In the hospital regressions each observation is a PCP-year and I regress the log of my hospital referral concentration measure,  $C_{jt}^{\text{hospitals}}$ , on an indicator for the PCP being integrated with a hospital, in a model with PCP and year fixed effects. I cluster standard errors at the PCP level. Since, I am merely trying to identify whether physician-hospital integration is associated with an increase in referral concentration (see hypothesis 2.4), as opposed to whether it causes a change in referral concentration, I do not include control variables. This can be expressed as shown in equation 2.9, where  $v_{jt}$  takes a value of one if PCP  $j$  is integrated with a hospital in year  $t$ ,  $\delta_j^{\text{hospitals}}$  is a PCP fixed effect,  $\phi_t^{\text{hospitals}}$  is a year fixed effect,  $e_{jt}^{\text{hospitals}}$  is a random error term,

and  $\alpha^{hospitals}$  and  $\beta^{hospitals}$ ) are parameters of the inpatient referrals concentration model.

$$C_{jt}^{hospitals} = \alpha^{hospitals} + \beta^{hospitals} v_{jt} + \phi_t^{hospitals} + \delta_j^{hospitals} + e_{jt}^{hospitals} \quad (2.9)$$

In the overall specialist regressions each observation is a PCP-specialty-year, and I exclude PCPs that are Cardiologists from the sample. Due to the shape of the distribution of the specialist referral concentration measure,  $C_{jt}^K$ , I regress its logarithm on an indicator for the PCP being integrated with a hospital, in a model with PCP-specialist and year fixed effects. I cluster standard errors at the PCP level. The regression equation can be written as

$$\ln [C_{jt}^K] = \alpha^{specialists} + \beta^{specialists} v_{jt} + \phi_t^{specialists} + \delta_j^K + e_{jt}^K \quad (2.10)$$

where  $\delta_j^K$  is the (PCP  $j$ )-(speciality  $K$ ) fixed effect,  $\phi_t^{specialists}$  are year fixed effects, and  $\alpha^{specialists}$  and  $\beta^{specialists}$  are parameters of the pooled specialist referrals concentration model.

I also conduct individual specialty regressions where each observation is a PCP-year. Similarly, I regress the log of my specialist referral concentration measure on an indicator for the PCP being integrated with a hospital and include PCP and year fixed effects. Again, I cluster standard errors at the PCP level. The regression equation for the individual specialty regressions is

$$\ln [C_{jt}^K] = \alpha^K + \beta^K v_{jt} + \phi_t^K + \delta_j^K + e_{jt}^K \quad (2.11)$$

where  $\alpha^K$  and  $\beta^K$  are specific to specialty  $K$ .

The test of hypotheses 2.4 corresponds to testing  $\beta^{hospitals} > 0$ ,  $\beta^{specialists} > 0$ , and  $\beta^K > 0$ .

For the coefficient on PCP integration to provide a valid test for whether PCP integration is associated with an increase in referral concentration beyond what would occur in the absence of the PCP integrating, I must assume that any referral shifting by PCPs who are

acquired by hospitals, does not affect referral location for PCPs who are not acquired by hospitals. Of particular concern would be capacity constraints. If acquired PCPs switch their referrals to their capacity constrained hospital-owner, then surrounding PCPs may be obliged to shift referrals away from the hospital that acquired their competitor. Such a situation would cause some of the integration associated referral concentration increases to load on the year fixed effects, thus underestimating the relationship between PCP integration and PCP referral concentration. Since PCP-hospital integration is more prevalent in capacity slack areas (see Appendix A.3), I do not expect this assumption to be violated in practice.

## 2.6 Results

### 2.6.1 Propensity to make referrals

In Table 2.1 I report the coefficients of interest from the propensity to make referrals regressions. Columns (1) and (2) show the results for referrals from PCPs to specialists, columns (3) and (4) show the results for referrals from generalist PCPs to Cardiologists, and columns (5) and (6) show the results for referrals from PCPs to inpatient hospital settings. Note that the number of observations is less for the generalist PCP to Cardiologist referrals because I exclude PCPs who are Cardiologists from the sample.

Table 2.1: Influence of integration on PCP propensity to make referrals

	(1)	(2)	(3)	(4)	(5)	(6)
	Specialists		Cardiologists		Inpatient hospital	
	OLS	Linear FE	OLS	Linear FE	OLS	Linear FE
<i>Panel A: No instrument</i>						
Coeff. on PCP integration status	0.099***	0.029	0.0037	-0.0042	-0.0023	-0.0018
(Standard error)	(0.017)	(0.019)	(0.0052)	(0.0077)	(0.0018)	(0.0031)
No. beneficiary-years	553,170	553,170	509,902	509,902	553,170	553,170
<i>Panel B: Reduced form</i>						
Coeff. on original PCP integration	0.043*	0.002	0.0138	0.0002	-0.0050*	-0.0038
(Standard error)	(0.023)	(0.020)	(0.0094)	(0.0082)	(0.0027)	(0.0033)
No. beneficiary-years	435,796	435,796	400,396	400,396	435,796	435,796
<i>Panel C: First stage</i>						
Coeff. on original PCP integration	0.810***	0.668***	0.836***	0.702***	0.810***	0.668***
(Standard error)	(0.005)	(0.006)	(0.005)	(0.007)	(0.0050)	(0.0064)
[F-statistic for excluding instrument]	[25,966]	[10,971]	[29,624]	[11,689]	[25,966]	[10,971]
No. beneficiary-years	435,796	435,796	400,396	400,396	435,796	435,796
<i>Panel D: Instrumental variables</i>						
Coeff. on PCP integration status	0.052*	0.003	0.0165	0.0003	-0.0061*	-0.0058
(Standard error)	(0.028)	(0.030)	(0.0112)	(0.0117)	(0.0034)	(0.0049)
No. beneficiary-years	435,796	435,796	400,396	400,396	435,796	435,796

I find a positive significant correlation between a PCP being integrated with a hospital and the amount of specialist referrals the physician makes per patient-year (panel A column 1). This positive relationship is not being driven by referrals to Cardiologists (panel A column 3). There is also no relationship apparent between whether a PCP is owned by a hospital and whether the PCP refers to hospital inpatient settings. The relationship between PCP integration status and referrals to specialists disappears when I account for physician fixed effects. This suggests that some PCPs have unobserved characteristics that make them more inclined to make referrals to specialists than other PCP, or have unobserved characteristics that attract patients that tend to require more referrals to specialists.

In panel C of Table 2.1, I demonstrate that the first stage is very strong. The usual



threshold for testing the correlation aspect of the validity of an instrument is an F-statistic of about 10. Across all outcome variables, and over both the OLS and linear fixed effects models this threshold is vastly exceeded.

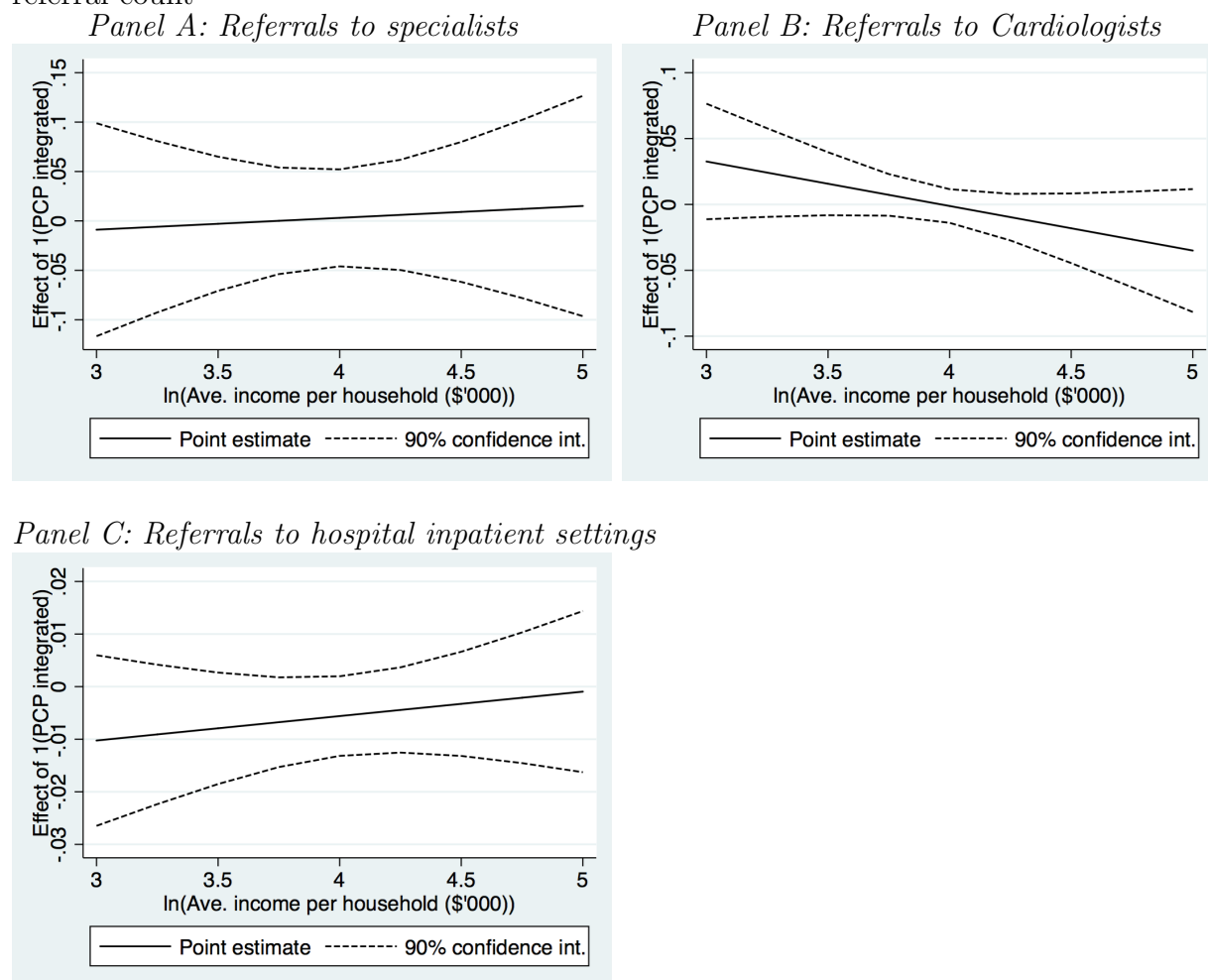
In panel D of Table 2.1, I present the results where I account for unobserved patient characteristics by instrumenting for the integration status of each patient's PCP with the current integration status of that patient's original PCP. For referrals from PCPs to specialists and referrals from PCPs to inpatient hospital settings, the instrument reduces the point estimate of the effect of PCP integration on referrals in the models with and without physician fixed effects. This suggests that integrated physicians attract patients that are more in need of specialist/ inpatient referrals (or possibly patients that request more specialist/ inpatient referrals) beyond what is captured in the observable patient characteristics. In the referrals to specialists model without physician fixed effects there remains a statistically significant positive relationship between physician integration status and the count of referrals to specialists. However, including both the instrument and the physician fixed effects (which is my preferred specification) results in a relatively precise zero for the estimated effect of physician integration on propensity to refer to specialists. In the referrals to hospital inpatient settings model without physician fixed effects adding the instrument results in a marginally significant negative relationship between physician integration status and the count of referrals to inpatient hospital settings. The model with both the instrument and the physician fixed effects results in a conclusion of no causal relationship between PCP-hospital integration and referrals to hospital inpatient settings.

I show evidence in Appendix B.3 that the signs, approximate magnitudes, and significance of these results on the relationship between PCP integration status and referrals to hospitals, are generally not driven by the choice to use a linear model. The one point of difference is that in the Poisson model with physician fixed effects but no instrument, the coefficient on PCP integration status is marginally significant in the referrals to specialists regressions.

The conclusion from the Poisson analogue of the preferred specification is the same as the preferred specification in the linear model presented here.

Figure 2.8 shows the results of the model with physician fixed effects and an instrument where an interaction term was introduced between 1(PCP integrated) and  $\ln(\text{average household income})$ . At the 90% level, I find no evidence that the effect of attending an integrated PCP on referrals to specialists differs from zero for patients in any portion of the income distribution.

Figure 2.8: Variation over income in the effect of the beneficiary's PCP being integrated on referral count



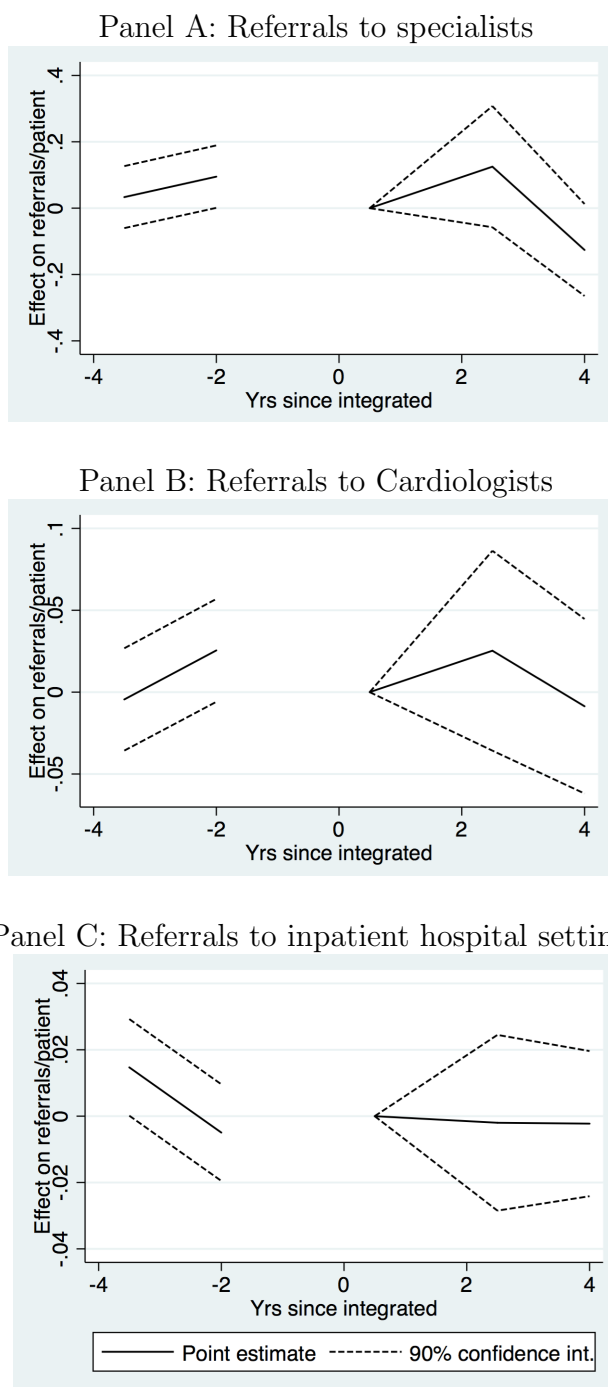
All graphs are based on the regression with both the instrument and the physician fixed effects.

### 2.6.1.1 Leads and lags

Figure 2.9 Panel A shows the results of the instrumental variables with PCP fixed effects regressions, of the propensity of a PCP to refer to a specialist in the years prior to and after the PCP integrates with a hospital. Panels B and C show the analogous results for referrals to Cardiologists and referrals to inpatient hospital settings. Recall that the regression specification included indicators for the PCP being integrated for -4 to -3 years, -2 years, 2 to 3 years, and 4 years. 0 to 1 years is the excluded category. There are no observations for the PCP being integrated for -1 years, since I treat integration status as unknown in the year between when the data reported that the physician was independent and when the data reported that the physician is integrated. At the midpoint of the range specified by each indicator variable, I graph the estimated coefficient of that indicator variable and its 90% confidence interval.

All Panels in Figure 2.9 demonstrate no significant pre-integration trends, no significant difference between the last year prior to integration (as we would expect based on the main results) and the first two years in which the PCP is integrated, and no significant post-integration trends.

Figure 2.9: PCPs' propensity to refer by years since the PCP integrated



## 2.6.2 Directing referrals

### 2.6.2.1 Inpatient referrals

In Table 2.2 I present the results of the location of inpatient referrals regressions. Hypotheses 2.2 predicts that the coefficient on “PCP and hospital are integrated with each other” will be positive (i.e. integrating physicians will redirect referrals to their hospital-owner).

Table 2.2: Effect of affiliations on inpatient hospital referrals - large choice set

Regression number Model	(1)	(2)	(3)	(4)	(5)	(6)
	OLS		PCP- hospital FE		IV	IV with PCP-hospital FE
Sample	Full	Restricted	Full	Restricted	Restricted	Restricted
PCP and hospital are integrated ( $\beta_V$ )	0.517*** (0.0115)	0.496*** (0.0107)	0.0054 (0.0177)	-0.0021 (0.0179)	0.547*** (0.0125)	0.0270 (0.0275)
PCP is integrated but with someone else ( $\beta_v$ )	-0.0004 (0.0004)	0.0001 (0.0004)	0.0004 (0.0004)	0.0003 (0.0004)	-0.00002 (0.0005)	0.0002 (0.0006)
hospital owns 1 or more PCPs ( $\beta_\lambda$ )	0.0326*** (0.0004)	0.0324*** (0.0004)	0.0071*** (0.0003)	0.0071*** (0.0003)	0.0327*** (0.0004)	0.0072*** (0.0004)
(PCP is integrated but with someone else) X (hospital owns 1 or more PCPs) ( $\beta_{v\lambda}$ )	-0.0204*** (0.0008)	-0.0202*** (0.0007)	-0.0017*** (0.0007)	-0.0013* (0.0007)	-0.0235*** (0.0008)	-0.0024** (0.0009)
$\beta_v + \beta_{v\lambda}$	-0.0208*** (0.0007)	-0.0201*** (0.0007)	-0.0014* (0.0007)	-0.0010 (0.0008)	-0.0235*** (0.0008)	-0.0022* (0.0012)

Each column contains coefficients and standard errors from a single regression. Standard errors are in parentheses. \*\*\* significant at the 1% level, \*\* significant at the 5% level, \* significant at the 10% level. All regressions also included year fixed effects and the control variables outlined in the estimation strategy section (see Section 2.5.2.1). The restricted sample excludes beneficiaries whose initial choice of PCP is already either integrated when the beneficiary chose them. The full sample contains 12,440 clusters (PCPs); 490,240 panels (PCP-hospital pairs); and 1,461,938 observations (an average of 3 per panel). The restricted sample contains 12,089 clusters; 477,663 panels; and 1,403,666 observations (an average of 3 per panel).

Columns (1) and (3) of Table 2.2 use the full sample of referral occasions  $\times$  hospitals in the choice set. All other columns exclude beneficiaries who initially chose a PCP that is at a practice that is owned by a hospital or has Cardiologists working there at the time the

beneficiary first chose the PCP. Comparing the OLS results from columns (1) and (2) to each other and the PCP-Cardiologist FE results in columns (3) and (4) to each other, it appears that restricting the sample in this way does not impact the coefficient estimates dramatically. In the models with no physician fixed-effects, the coefficients on “PCP and hospital are integrated with each other” are unrealistically large, suggesting that having a PCP that is integrated with a particular hospital increases the probability of referring patients to that hospital inpatient department by approximately 50 percentage points, whereas the probability for being referred to a random hospital in the choice set is just 2.5%. Adding PCP-hospital fixed effects reduces this figure dramatically and eliminates the statistical significance of the coefficient estimate for “PCP and hospital are integrated with each other”. This suggests that hospitals are buying the practices of PCPs that are highly likely to send their patients to the hospital irrespective of whether the hospital owns their practice. Adding the instrument in the physician fixed effects model does not substantially change the results.

Focusing on the preferred specification (column 6), I find that when a PCP integrates with a hospital there is no statistical evidence to support that they make more referrals to that hospital. However, the coefficient estimate,  $\beta_V$  represents a doubling of the probability of being referred to a particular hospital in the choice set, so the lack of statistically significant results for the probability of redirecting referrals to the hospital owner could be attributable to a lack of statistical power. I also find that when a PCP integrates with a hospital, they do appear to change their propensity to refer to “independent hospitals” (hospitals that do not own PCPs) ( $\beta_v \approx 0$ ) but they do reduce their referrals to other “integrated hospitals” (hospitals that own PCPs) ( $\beta_v + \beta_v \lambda < 0$ ). A possible explanation for the disparity in where integrating physicians reduce their referrals is that the hospitals that remain independent differ from the hospitals that integrate in other ways. For instance, if the tertiary hospitals are more likely to remain independent, they may also be the only hospitals that can properly address complicated cases, so they get a steady stream of referrals from all PCPs regardless

of whether the PCPs are acquired or not. The lack of statistical power in the test of  $\beta_V = 0$  relative to the tests of  $\beta_v = 0$  and  $\beta_v + \beta_{v\lambda}$  is not surprising. The standard deviation of the indicator 1(PCP  $j$  and hospital  $h$  are integrated) is just 0.060, whereas the standard deviations of 1(PCP  $j$  is owned by a hospital but not  $h$ ) and 1(PCP  $j$  is owned by a hospital but not  $h$ ) $\times$ 1(hospital  $h$  owns one or more PCPs) are 0.340 and 0.274 respectively. These results in combination provide suggestive evidence that integrated PCPs may be redirecting referrals away from other integrated hospitals to their hospital owner. I also find that a hospital that switches from not owning PCPs to owning PCPs becomes more appealing to independent physicians ( $\beta_\lambda > 0$ ). This may reflect improvements to the hospital that are made simultaneously with acquiring the other PCPs.

I reestimate my model using only panels where there is variation in the outcome variable. This corresponds to the sample which a conditional logit regression would use. In practice, one could imagine that many of the hospitals classified as in the choice set using 100 mile radius definition are not truly considered a potential choice by the PCP, which is not unsurprising (though clearly some PCPs do refer patients this far – see Figure B.1). With this redefined choice set, and excluding PCPs who only ever refer to a single hospital, the average probability a referral being to a specific hospital within the choice set is much higher at 35% (as opposed to 2.5% using the previous definition of the choice set). This does reduce the coefficients in the OLS model and serves to make the coefficients in the preferred model look more realistic, since they make up a smaller fraction of the base rate (see Table 2.3).

Table 2.3: Effect of affiliations on inpatient hospital referrals - small choice set and excluding PCPs who only ever refer to a single hospital

Regression number Model	(1) OLS	(2) PCP- hospital FE	(3) IV	(4) IV with PCP-hospital FE
PCP and hospital are integrated ( $\beta_V$ )	0.125*** (0.0136)	-0.0005 (0.0267)	0.144*** (0.0153)	0.0390 (0.0413)
PCP is integrated but with someone else ( $\beta_v$ )	0.0188 (0.0151)	-0.0694** (0.0330)	0.0294* (0.0174)	-0.0951** (0.0405)
hospital owns 1 or more PCPs ( $\beta_\lambda$ )	0.317*** (0.0117)	0.303*** (0.0197)	0.319*** (0.0119)	0.302*** (0.0199)
(PCP is integrated but with someone else) X (hospital owns 1 or more PCPs) ( $\beta_{v\lambda}$ )	-0.0398** (0.0171)	0.0602* (0.0339)	-0.0583*** (0.0194)	0.0581 (0.0406)
$\beta_v + \beta_{v\lambda}$	-0.0210** (0.0087)	-0.0092 (0.0130)	-0.0289*** (0.0099)	-0.0370* (0.0201)

Each column contains coefficients and standard errors from a single regression. Standard errors are in parentheses. \*\*\* significant at the 1% level, \*\* significant at the 5% level, \* significant at the 10% level. All regressions also included year fixed effects and the control variables outlined in the estimation strategy section (see Section 2.5.2.1). The sample excludes beneficiaries whose initial choice of PCP is already either integrated or sharing a site with a Cardiologist when the beneficiary chose them. The sample contains 4,784 clusters; 10,775 panels; and 65,877 observations (an average of 6 per panel).

The general picture of the results is however fairly similar to in Table 2.2. The coefficients in the OLS model are of the same sign, similar magnitudes, and similar levels of statistical significance. Adding PCP-hospital fixed effects eliminates these apparent effects. Again the estimated coefficient on 1(PCP  $j$  is owned by hospital  $h$ ) is not statistically significant. The point estimate of the coefficient now represents c.11% of the base rate, which still suggests the test on this coefficient may have moderately low statistical power but not as abysmal as in the previous regression. This point estimate corresponds to the PCP raising the probability of a referral to hospital  $h$  from the mean of 35% to 39% when the PCP integrates with that hospital. The major change from the results in Table 2.2 is that the PCP integrating with a hospital now appears to lower the probability of it making referrals to independent hospitals



(see Table 2.3, column 4,  $\beta_v < 0$ ) not just other integrated hospitals (see Table 2.3, column 4,  $\beta_v + \beta_{v\lambda} < 0$ ).

Overall the referrals to inpatients suggest that hospitals tend to buy the practices of PCPs that refer to them frequently even prior to the ownership relationship. There is also some evidence that PCPs may shift their inpatient referrals in response to being acquired by a hospital.

### 2.6.2.2 Cardiology referrals

In Table 2.4 I present the results of the location of Cardiology referral regressions. Columns (1) and (3) use the “full” sample, which is referral occasions  $\times$  Cardiologists in the choice set, excluding the PCPs who share a site with a Cardiologist, or are a Cardiologist themselves. All other columns exclude beneficiaries who initially chose a PCP that is at a practice that is owned by a hospital. Comparing the OLS results from columns (1) and (2) to each other and the PCP-Cardiologist FE results in columns (3) and (4) to each other, it appears that restricting the sample in this way does not impact most of the coefficients of interest dramatically. None of the coefficients change in sign or have a dramatic shift in their level of significance. The magnitudes of the coefficients of interests are similarly unaffected in most cases. The most notable exception is the coefficient on 1(PCP and Cardiologist are integrated with the same hospital) in the model with PCP-Cardiologist fixed effects (columns 3 and 4). This coefficient drops by about 40% when the sample is restricted. However, it is not significantly different from zero (at the 10% level) either before or after the sample restriction.

For all of the variables of interest, introducing PCP-Cardiologist fixed effects (comparing columns 2 and 4) reduces the magnitudes of the point estimates. The estimated effect of an independent PCP integrating with a hospital that owns a Cardiologist on the probability of referring that Cardiologist ( $\beta_v$ ) declines dramatically from about a 3.5 percentage point effect

Table 2.4: Effect of affiliations on Cardiology referrals - large choice set

Regression number Model	(1)	(2)	(3)	(4)	(5)	(6)
	OLS		PCP- Cardiologist FE		IV	IV with PCP-Cardio FE
Sample	Full	Restricted	Full	Restricted	Restricted	Restricted
PCP and Cardiologist are int. with the same hospital ( $\beta_V$ )	3.343*** (0.188)	3.450*** (0.194)	0.222 (0.140)	0.129 (0.145)	3.502*** (0.229)	0.113 (0.192)
PCP is int. but not with same hospital as Cardio. ( $\beta_v$ )	0.043*** (0.012)	0.031** (0.012)	-0.016** (0.008)	-0.014* (0.008)	-0.215*** (0.021)	-0.027 (0.020)
Cardiologist is integrated with a hospital ( $\beta_\lambda$ )	-0.281*** (0.008)	-0.281*** (0.008)	-0.031*** (0.009)	-0.033*** (0.009)	-0.305*** (0.009)	-0.037*** (0.010)
PCP and Cardio. both int. but with different hosp. ( $\beta_{v\lambda}$ )	-0.067*** (0.015)	-0.045*** (0.016)	0.007 (0.011)	0.006 (0.013)	0.132*** (0.026)	0.035 (0.025)
$\beta_v + \beta_{v\lambda}$	-0.007 (0.014)	-0.014 (0.015)	-0.010 (0.011)	-0.008 (0.011)	-0.083*** (0.025)	0.008 (0.022)

Each column contains coefficients and standard errors from a single regression. Standard errors are in parentheses. **All coefficients and standard errors have been multiplied by 100 to ease interpretation.** \*\*\* significant at the 1% level, \*\* significant at the 5% level, \* significant at the 10% level. All regressions also included year fixed effects and the control variables outlined in the estimation strategy section (see Section 2.5.2.2). Both full and restricted samples exclude beneficiaries whose PCP shares a site with a Cardiologist. The restricted sample excludes beneficiaries whose initial choice of PCP is already integrated when the beneficiary chose them. The full sample contains 14,153 clusters (PCPs); 3,116,175 panels (PCP-Cardiologist pairs); and 22,206,950 observations (an average of 7 per panel). The restricted sample contains 13,651 clusters; 2,996,983 panels; and 20,838,660 observations (an average of 7 per panel).

to a 0.1 percentage point effect. This decline demonstrates that hospitals are more likely to acquire PCPs that are already referring to their Cardiologists prior to the acquisition.

Instrumenting for the PCP ownership variables (see Table 2.4, column 6) does not have a substantial impact on the coefficients of interest. Though the estimated effect of an independent PCP integrating on referrals to Cardiologists who are not integrated with hospitals switches from being considered significant at the 10% level to not significant at the 10% level. Column 6 is the preferred specification.

In the restricted sample the mean probability of the PCP referring to any specific Cardiologist (given that a referral will be made to some Cardiologist in the choice set) is 0.380 percentage points. Hypothesis 2.2 predicts the coefficient on 1(PCP and Cardiologist are

owned by the same hospital) will be positive (i.e.  $\beta_V > 0$ ). In the preferred specification the sign of the point estimate aligns with this prediction. However, the estimated coefficient is not significant at the 10% level so I cannot conclusions that integration is driving PCPs to refer to Cardiologists owned by their hospital-owner more often. The point estimate for the coefficient on 1(PCP and Cardiologist are owned by the same hospital) in the preferred specification (column 6) corresponds to an increase of 0.113 percentage points. Hence (ignoring the statistical insignificance of the estimate) the coefficient on 1(PCP  $j$  shares a practice with a Cardiologist but not  $h$ ) indicates that when an independent PCP is purchased by a hospital the probability that they refer to a particular Cardiologist owned by that hospital will increase by c.30%.

In the preferred specification  $\beta_v$  indicates the predicted effect of a PCP integrating with a Cardiologist-owning hospital on the probability of making a referral to a particular independent Cardiologist.  $\beta_v + \beta_{v\lambda}$  gives the predicted effect of a PCP integrating with a Cardiologist-owning hospital on the probability of making a referral to a particular Cardiologist that is integrated with a different hospital to the PCP. Hypothesis 2.2 also predicts at least one of  $\beta_v$  and  $\beta_v + \beta_{v\lambda}$  will be negative. Neither coefficient is statistically significant in the preferred specification.

As in the inpatient referral regressions I also repeat the restricted sample regressions with the observations that a conditional logit regression would utilize – the subset of panels that have variation in the outcome variable. These results are shown in Table 2.5. The signs of all estimated coefficients in the preferred specification remain under the “conditional logit sample” (Table 2.5 column 4) and the original restricted sample (Table 2.4 column 6). However the statistical significance of some of the estimated coefficients changes between samples. Additionally, the sign on the sum of the coefficients  $\beta_v$  and  $\beta_{v\lambda}$ , which represents the estimated effect of a PCP integrating with a Cardiology-owning PCP on their probability that the PCP refers to a Cardiologist owned by a hospital other than their owner, changes

but the total remains statistically insignificant. Unlike the preferred specification presented here (Table 2.4 column 6), the results of the preferred specification in the conditional logit sample (Table 2.5 column 4), show slight evidence that PCPs are redirecting Cardiology referrals to Cardiologists that share their owner when the are acquired by a hospital in the form of  $\beta_v$  being statistically significantly negative at the 10% level.  $\beta_V$  and  $(\beta_v + \beta_{v\lambda})$ , while of the predicted signs, are not significantly different from zero. The overall picture is one of no strong evidence to support the hypothesis that PCPs are redirecting Cardiology referrals to Cardiologists that share their owner when the are acquired by a hospital.

Table 2.5: Effect of affiliations on Cardiology referrals - small choice set and excluding PCPs who only ever refer to a single Cardiologist

Regression number Model	(1) OLS	(2) PCP- Cardiologist FE	(3) IV	(4) IV with PCP-Cardio FE
PCP and Cardiologist are integrated with the same hospital ( $\beta_V$ )	5.020*** (0.724)	1.167** (0.515)	3.889*** (0.839)	0.912 (0.630)
PCP is integrated but not with the same hospital as the Cardiologist ( $\beta_v$ )	1.692*** (0.582)	-0.245 (0.199)	0.467 (0.929)	-1.527* (0.908)
Cardiologist is integrated with a hospital ( $\beta_\lambda$ )	0.411 (0.318)	-0.972*** (0.295)	0.611* (0.359)	-1.188*** (0.349)
PCP and Cardiologist are both integrated but with different hospitals ( $\beta_{v\lambda}$ )	-0.625 (0.890)	-0.738 (0.592)	-1.466 (1.321)	0.538 (1.352)
$\beta_v + \beta_{v\lambda}$	1.065 (1.065)	-0.983* (0.563)	-1.003 (1.176)	-0.992 (1.226)

Each column contains coefficients and standard errors from a single regression. Standard errors are in parentheses. **All coefficients and standard errors have been multiplied by 100 to ease interpretation.** \*\*\* significant at the 1% level, \*\* significant at the 5% level, \* significant at the 10% level. All regressions also included year fixed effects and the control variables outlined in the estimation strategy section (see Section 2.5.2.2). The sample excludes beneficiaries whose PCP shares a site with a Cardiologist, and beneficiaries whose initial choice of PCP is already integrated when the beneficiary chose them. The sample excludes beneficiaries whose initial choice of PCP is already either integrated or sharing a site with a Cardiologist when the beneficiary chose them. The sample contains 9,360 clusters (PCPs); 40,677 panels (PCP-Cardiologist pairs); and 626,366 observations (an average of 15 per panel).

In both samples, the preferred specification indicates a strong negative relationship between a Cardiologist integrating with a hospital and the probability that independent PCPs

refer to that Cardiologist. ( $\beta_\lambda$  from Table 2.4 column 6 and Table 2.5 column 4). This may reflect independent PCPs protecting their patients from the higher costs associated with integrated Cardiologists.

### 2.6.2.3 Comparison to Baker

In Table 2.6 model 1 I show my approximate replication of Baker et al.'s (2016) base model. In Table 2.6 model 7 I show the results of my preferred specification for inpatient hospital referrals (these closely resemble Baker et al.'s definition of referrals) in the set of observations that a conditional logit version of my preferred specification would use (this corresponds to the small choice set regressions discussed above).<sup>8</sup> Models 2 to 6 are hybrid models with lower numbers more closely resembling the Baker-replication of model 1 and higher numbers more closely resembling my preferred specification in column 7. Panel A describes each model and Panel B contains the coefficients and standard errors of the ownership indicators from each model. I do not present marginal effects for the logit models, since they can only be estimated under the unrealistic assumption that all fixed effects are zero.

Comparing model 1 to model 2 in Table 2.6, you can see that switching from the 90 day admission referral definition to the 30 day admission referral definition does not substantively change the results. Clustering on PCP rather than 3 digit ZIP code somewhat reduces the standard error of the coefficient estimates of 1(PCP-hospital integrated) and 1(Hospital owns PCPs) but raises the standard error on the interaction term (model 2 versus model 3). A critical change is evident between models 3 and 4, where I switch the referral occasion fixed effects for PCP-hospital fixed effects. The estimated effect of a PCP integrating with

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<sup>8</sup>The coefficients on 1(PCP integrated with any hospital) and 1(PCP integrated with any hospital) $\times$ 1(Hospital owns PCPs) in model 7 do not correspond directly to the coefficient estimates in Table 2.5. I have combined coefficient estimates where appropriate to match the ownership indicators in Baker's model. The coefficient on 1(PCP integrated with any hospital) in model 7 is equal to  $\beta_v + \beta_V$  from Table 2.5 column 4. Similarly the coefficient on 1(PCP integrated with any hospital) $\times$ 1(Hospital owns PCPs) in model 7 is equal to  $\beta_{v\lambda} + \beta_V$  from Table 2.5 column 4.

a hospital on the PCP's probability of referring to that hospital conditional on making a referral drops dramatically. This is because the PCP-hospital fixed effects capture PCPs' preference for referring to a particular hospital regardless of whether a hospital owns their practice or not. Due to hospitals' tendency to acquire PCPs that already refer to them frequently, the PCP-hospital fixed effects eliminate the substantial upward bias in the estimated effect of hospital-ownership of PCP practices on referral direction present in the Baker style models. Switching from referral occasion fixed effects to PCP-hospital fixed effects results in most of the hospital characteristics being eliminated from the model, as they are not time varying. Switching from referral occasion fixed effects to PCP-hospital fixed effects also has the potential cost of introducing bias due to patient or case specific characteristics that the PCP may see that are not present in the data. Model 5 repeats the regression from model 4 but includes the control variables I use in my preferred specification, which include an extensive set of patient characteristics. Introducing these control variables has very little impact on the estimated coefficients on the PCP-hospital relationship indicators. Model 6 is a linear model with fixed effects whereas model 5 is a logit model with fixed effects. Since marginal effects can only be estimated in logit models with fixed effects under the unrealistic assumption that all fixed effects are zero, I have presented only the coefficient estimates, which is why the coefficient estimates for models 5 and 6 are so different. Similarly to in model 5 the estimated coefficient on 1(PCP-hospital are integrated) is not significantly different from zero in model 6. The coefficient estimates on 1(Hospital owns PCPs) are both significantly positive at the 1% level. The estimated coefficients on the interaction term are both positive but not significant in models 5 and 6. The estimates of the coefficient on 1(PCP int. any hospital) are both negative but in the linear model (6) the estimate appears to be significantly different from zero at the 10% level, unlike in the logit model. The estimate in model 5 corresponds to PCPs who are owned by any hospital reducing their odds of referring to a particular hospital by 18% whereas the estimate in model 6 corresponds to

PCPs who are owned by any hospital reducing their odds of referring to a particular hospital by 20% relative to the average probability of referring to a particular hospital (i.e. from 35% to 28%). Adding the instrument to the linear model does not affect the conclusions about 1(PCP-hospital are integrated) or 1(Hospital owns PCPs) but does switch which of 1(PCP int. any hospital) and the interaction term appear to be significant at the 10% level.

Table 2.6: Effect of affiliations on inpatient hospital referrals - comparison to Baker

<i>Model number</i>	(1)	(2)	(3)	(4)	(5)	(6)	(7)
<i>Panel A: Model description</i>							
Functional form	Logit	Logit	Logit	Logit	Logit	Linear	Linear
Fixed effects	Referral occasion	Referral occasion	Referral occasion	PCP-hospital	PCP-hospital	PCP-hospital	PCP-hospital
Controls	Baker	Baker	Baker	Baker	Wagner	Wagner	Wagner
Max. days between PCP visit & admission to count as referral	90	30	30	30	30	30	30
Instrument	No	No	No	No	No	No	Yes
Level of standard error clustering	ZIP3 <sup>^</sup>	ZIP3 <sup>^</sup>	PCP	PCP	PCP	PCP	PCP
<i>Panel B: Coefficient estimates and standard errors</i>							
1(PCP-hospital integrated)	1.842*** (0.0716)	2.005*** (0.0763)	2.005*** (0.0569)	0.144 (0.163)	0.146 (0.163)	-0.000466 (0.0267)	0.0390 (0.0413)
1(Hospital owns PCPs)	22.20*** (0.116)	21.58*** (0.122)	21.58*** (0.0750)	19.43*** (0.0802)	19.30*** (0.0750)	0.303*** (0.0197)	0.302*** (0.0199)
1(PCP int. any hospital)				-0.224 (0.321)	-0.198 (0.252)	-0.0699* (0.0389)	-0.0561 (0.0509)
1(PCP int. any hospital) × 1(Hospital owns PCPs)	0.248 (0.178)	0.441 (0.273)	0.441 (0.331)	0.0949 (0.258)	0.0889 (0.246)	0.0597 (0.0408)	0.0971* (0.0545)
Number of observations	1,655,491	1,319,204	1,319,204	69,464	69,464	65,877	65,877

<sup>^</sup>3 digit ZIP code. \*\*\* significant at the 1% level, \*\* significant at the 5% level, \* significant at the 10% level.

### 2.6.2.4 Leads and lags

Figure 2.10 shows the effect of time since integration on the probability that a PCP makes a referral to the hospital they eventually integrate with conditional on the PCP making a referral to some hospital. The graph shows no evidence of pre-integration trends. The point estimate is suggestive of the proportion of referrals being redirected to the hospital-owner increases as the years since integration increases. However, the standard errors are too large for this trend to be statistically significant.

Figure 2.10: Effect of integrating with a hospital on probability of referring to the inpatient department of that hospital by years since integration

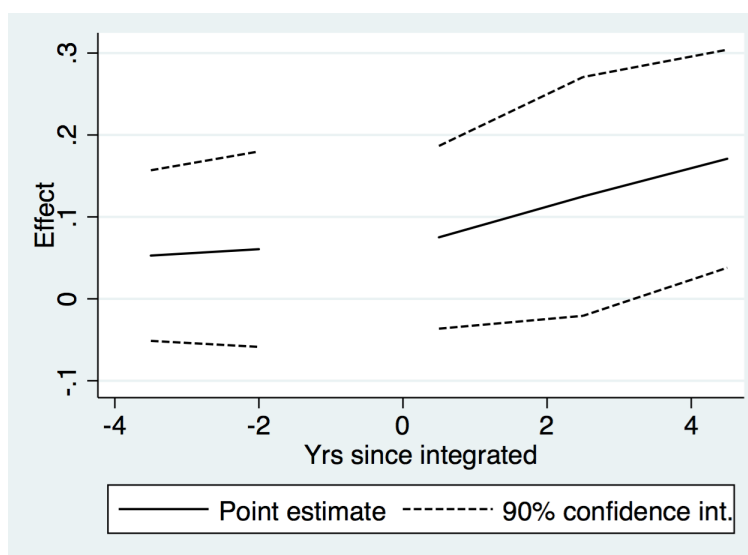


Figure 2.11 shows the effect of time since integration on the probability that a PCP makes a referral to a Cardiologist that they eventually share a hospital-owner with, conditional on the PCP making a referral to some Cardiologist. The time since integration refers to the time since the PCP integrates with any hospital. The graph has three panels, each representing PCP-Cardiologist pairs with a different number of years between the PCP sharing a hospital-owner with *any* Cardiologist, and the PCP sharing a hospital-owner with the Cardiologist



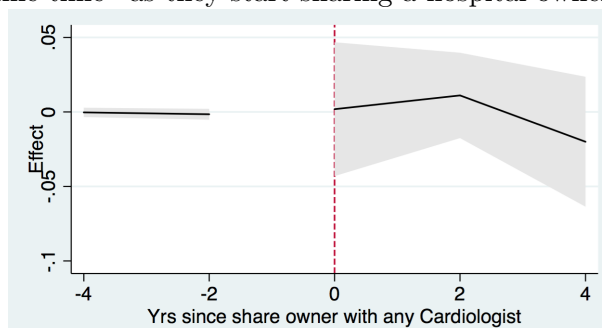
in the current observation. None of the panels suggest significant pre-integration or post-integration trends.

#### **2.6.2.5 Summary of directing referrals results**

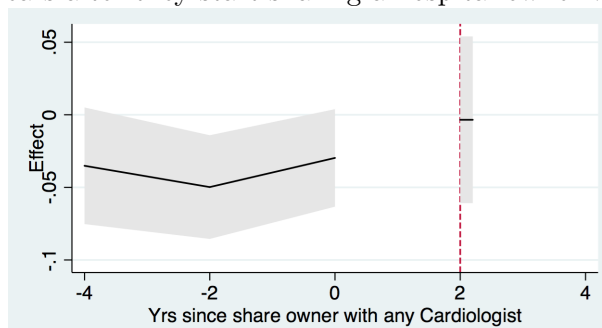
My overall conclusion from this Section is that I have at best weak evidence to support the hypothesis that physicians shift their referrals toward their hospital-owner. However, the leads and lags regressions for inpatient hospital referrals are suggestive of a delayed response to integration in referral shifting. My results provide no evidence to support the hypothesis that physicians shift their referrals towards Cardiologists that have the same hospital owner. The difference between my estimated effect of PCP-hospital integration on the probability of referring to the acquiring hospital and Baker et al.'s (2016) estimated effect can be explained by their estimated effect being upwardly biased due to not accounting for inherent physician preferences to refer to a particular hospital being correlated with the likelihood that a physician's practice is acquired by that hospital.

Figure 2.11: Effect of integrating with a hospital that owns Cardiologists on probability of referring to a particular one of those Cardiologists by years since integration

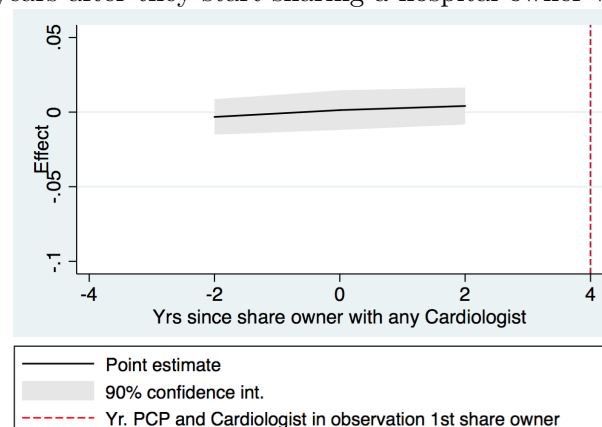
Panel A: PCPs that start sharing a hospital-owner with the Cardiologist in the current observation at the same time\* as they start sharing a hospital-owner with any Cardiologist



Panel B: PCPs that start sharing a hospital-owner with the Cardiologist in the current observation two\* years after they start sharing a hospital-owner with any Cardiologist



Panel C: PCPs that start sharing a hospital-owner with the Cardiologist in the current observation four\* years after they start sharing a hospital-owner with any Cardiologist



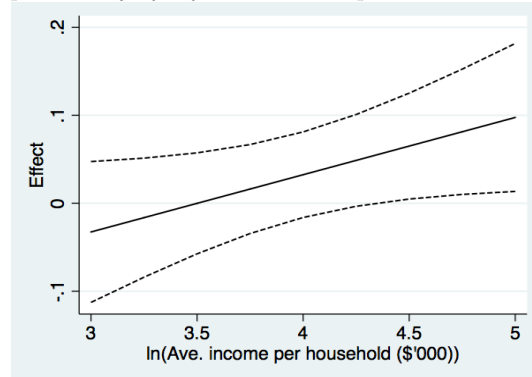
\* There are very few observations for which this difference is not an even number due to the integration variable being constructed from data that is available every other year. The few odd numbered differences result from hospitals that open or close during the period of the study. Odd numbered differences are grouped into the graph with the preceding even numbered difference.

### 2.6.3 Heterogeneity of directing referrals

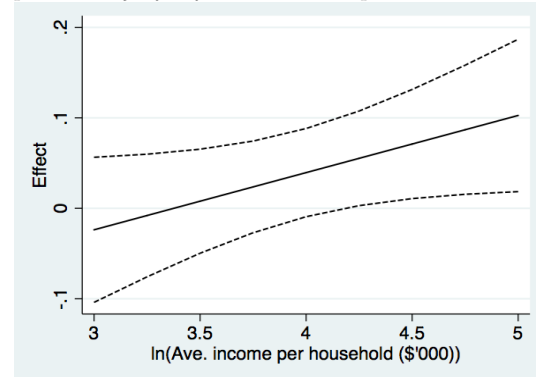
Figure 2.12 shows heterogeneity over beneficiary ZIP code average income, in the estimated effect of whether a PCP is integrated with the hospital of interest and whether a PCP is integrated with another hospital on where the PCP directs his inpatient referrals. The point estimates from Panels A and B suggest that PCPs may be redirecting their poor patients away from their hospital-owner and their wealthy patients toward their hospital-owner after they integrate. However, the confidence intervals are too wide to truly draw this conclusion. PCPs who are acquired by a hospital appear to shift some of their referrals of wealthy patients away from non-PCP-owning hospitals and some of their referrals of poor patients towards non-PCP-owning hospitals (see 2.12 Panel C). PCPs who are acquired by a hospital do not appear to condition vary their change in referrals to other PCP-owning hospitals based on patient income (see 2.12 Panel D). Taken together these results suggest that we should not rule out physician-hospital integration resulting in physicians redirecting their wealthier patients towards their hospital owner and their poorer patients away from their hospital-owner. However, the results are not strong enough to conclude that PCPs' referral response to integration varies by income.

Figure 2.12: Heterogeneity over beneficiary income\* in the effect of hospital-ownership on where PCPs direct their inpatient referrals

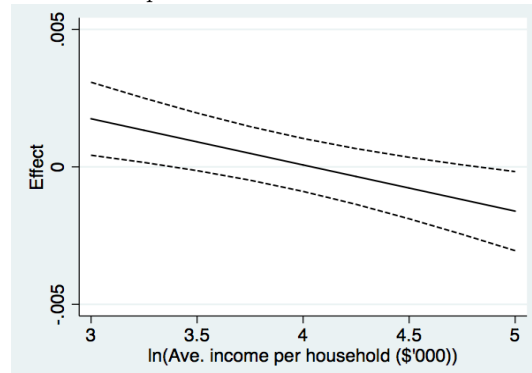
*Panel A: Unintegrated PCP integrating with a hospital that already owns PCPs, effect on probability of refer to that hospital*



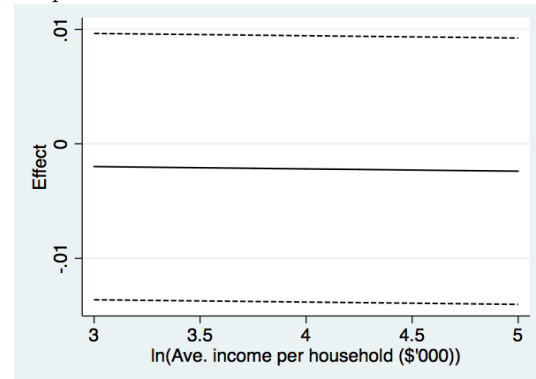
*Panel B: Unintegrated PCP integrating with a hospital that does not own PCPs, effect on probability of refer to that hospital*



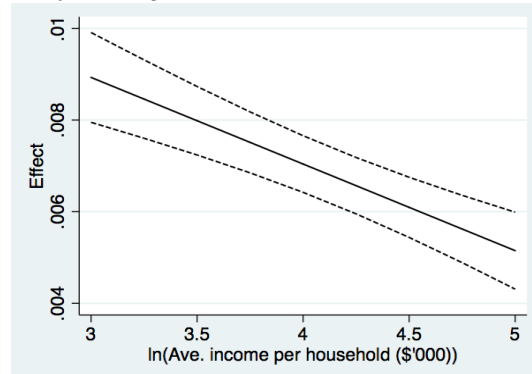
*Panel C: Unint. PCP integrating with a hospital, effect on the probability of refer to another hospital that does not own PCPs*



*Panel D: Unint. PCP integrating with a hospital, effect on the probability refer to another hospital that does own PCPs*



*Panel E: Hospital that owns no PCPs buying a PCP, effect on the probability of being referred to by unintegrated PCPs*

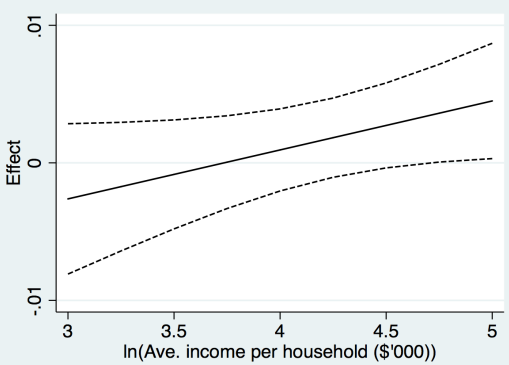


Solid line is the point estimate. Dotted lines are the boundaries of the 90% confidence interval. \*Proxied by average income per household in the beneficiary's 5-digit ZIP code. Panel A effect is calculated as  $\beta_V + \beta_{VI} * I_{it}$ . Panel B effect is  $\beta_V + \beta_\lambda + (\beta_{VI} + \beta_{\lambda I}) * I_{it}$ . Panel C effect is  $\beta_v + \beta_{vI} * I_{it}$ . Panel D effect is  $\beta_v + \beta_{v\lambda} + (\beta_{vI} + \beta_{v\lambda I}) * I_{it}$ . Panel E effect is  $\beta_\lambda + \beta_{\lambda I} I_{it}$ .

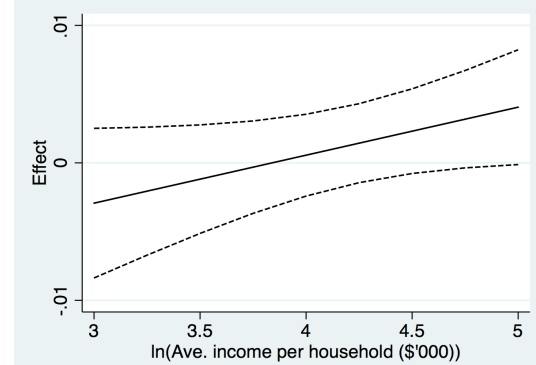
Figure 2.13 shows heterogeneity over beneficiary ZIP code average income, in the estimated effect of several integration variables on where PCPs direct their Cardiology referrals. Similarly to the inpatient referrals, the point estimates in Panels A and B indicate that when a PCP integrates they begin directing their wealthy patients to Cardiologists who share a hospital-owner with the PCP but also begin directing their poor patients away from Cardiologists who share a hospital-owner with the PCP. However, the standard errors are too large for this apparent difference to be statistically significant, even at the 90% level. In contrast to what one would expect, the point estimate of the effect of a PCP integrating on their probability of referring to an independent Cardiologist exhibits a slight upward slope (see 2.13 Panel C), though again the slope is not statistically different from zero at the 10% level. Panel D demonstrates that PCPs who are acquired by a hospital do not appear change their probability of referring to hospital-owned Cardiologists at any level of patient income.

Figure 2.13: Heterogeneity over beneficiary income\* in the effect of hospital-ownership and shared practices on where PCPs direct their Cardiology referrals

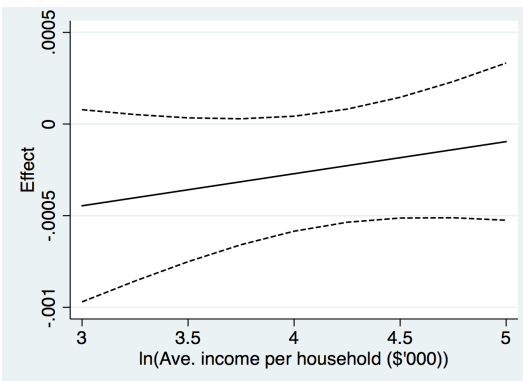
*Panel A: PCP integrating with a hospital that is also owns Cardiologists, effect on probability refer to hospital-owned Cardiologist*



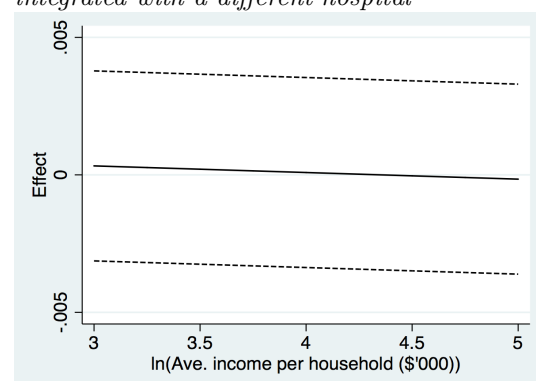
*Panel B: PCP integrating with a hospital that does not own Cardiologists, effect on probability refer to hospital-owned Cardiologist*



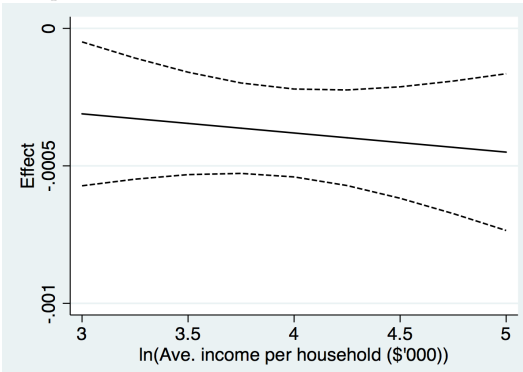
*Panel C: PCP integrating with a hospital, effect on probability refer to independent Cardiologist*



*Panel D: PCP integrating with a hospital, effect on probability refer to Cardiologist that is integrated with a different hospital*



*Panel E: Cardiologist integrating with a hospital, effect on probability of a referral from an independent PCP*



Solid line is the point estimate. Dotted lines are the boundaries of the 90% confidence interval. \*Proxied by average income per household in the beneficiary's 5-digit ZIP code. Panel A effect is calculated as  $\beta_V + \beta_{VI} * I_{it}$ . Panel B effect is  $\beta_V + \beta_\lambda + (\beta_{VI} + \beta_{\lambda I}) * I_{it}$ . Panel C effect is  $\beta_v + \beta_{vI} * I_{it}$ . Panel D effect is  $\beta_v + \beta_{v\lambda} + (\beta_{vI} + \beta_{v\lambda I}) * I_{it}$ . Panel E effect is  $\beta_\lambda + \beta_{\lambda I} I_{it}$ .

### 2.6.4 Concentration of referrals

The point estimate for the association between a PCP integrating with a hospital and the concentration of referrals to inpatient hospital settings in a model with PCP and year fixed effects is 124 with a standard error of 80 (t-statistic=1.56). This point increase corresponds to a 1.4% increase in concentration relative to the base rate but is not statistically significant, even at the 10% level. Consequently, I conclude that a PCP integrating with a hospital is not associated with an increase in the concentration of referrals to inpatient settings.

Table 2.7 shows the association between a generalist PCP integrating with a hospital and the concentration of their referrals to various types of specialists. Column 1 lists the specialties, column two gives the association between the PCP being integrated with a hospital and the log of the referral concentration measure, partialling out year and PCP fixed effects (or in the all speciality regression partialling out year and PCP-specialty fixed effects), column 3 gives the standard error of this association, column 4 displays the exponential of the mean of the logarithm of the referral concentration measure column, column 5 shows the change in the referral concentration measure predicted by the coefficient, column 6 gives the number of observations in the sample (PCP-specialty-years for the all specialties regression and PCP-years for the other regressions), and column 7 gives the number of PCPs in the sample. These sample numbers exclude observations for PCP's (or PCP-specialties for the all specialty regression) with only one observation, since these observations do not help with identification in the fixed effects model. The all specialties regression does not find an association between a PCP integrating and changing the concentration of his referrals. At first glance it would seem that from among the eighteen specialties one has a significant association at the 5% level and 3 more have a significant association at the 10% level. These apparent associations run in opposite directions. However, with eighteen repetitions one significant result at the 5% or better level and two at the 10% or better level could easily

be coincidences. Given the sparseness of significant results, the opposite directions of the apparently statistically significant relationships, and the null result in the all specialties regression, I am inclined to conclude that there is no evidence to support a the hypothesis that PCP-hospital integration is associated with an increase in concentration among referrals to specialists.

## 2.7 Discussion

In spite of using a very different integration measure to Walden (2016) – I define a physician as integrated if the physician’s practice is listed as owned by a hospital in the SK&A physician survey, whereas Walden (2016) defines a physician as integrated if the physician is owned by a hospital or part of a multi-specialty physician practice, as determined by the physician billing under the hospital/ multi-specialty practice’s tax identification number – my finding that hospital ownership of PCP practices does not cause the physicians at he practice to make more referrals to specialists aligns with what Walden (2016) found. I confirm that this finding holds for referrals to Cardiologists specifically. I also find that there is no relationship between a PCP being integrated with a hospital and the likelihood that their patients are “referred” to a hospital inpatient setting (using being admitted to a hospital within 30 days of a PCP visit without going via the emergency department as a proxy for referrals to hospital inpatient settings). Unlike the results on specialist and Cardiologist referrals, it is possible that the lack of significant results for inpatient referrals may be due to low statistical power in this test resulting from the much lower number of observations relative to the other types of referrals.

One drawback of the propensity to refer to specialist and the propensity to refer to Cardiologist results is that a positive relationship between a PCP being integrated and referrals could be masked if if the physicians in the hospital could be becoming worse at



Table 2.7: Association between physician integration and concentration of referrals to specialists

Specialty	coefficient	(std. err.)	exp(mean ln(HHI))	change to HHI	obs.	PCPs
All	-0.020	(0.018)	135	-3	398197	20596
Cardiology	-0.028	(0.029)	202	-6	62283	14998
Dermatology	-0.090	(0.077)	52	-4	12498	4066
Diagnostic radiology	0.003	(0.020)	768	2	95492	19775
Gastroenterology	-0.027	(0.041)	84	-2	34214	9719
General surgery	0.056	(0.054)	64	4	26261	8125
Gynecology	0.259*	(0.151)	40	12	4568	1709
Hematology	-0.202	(0.190)	32	-6	2395	992
Nephrology	-0.024	(0.098)	53	-1	7880	2737
Neurology	-0.097*	(0.051)	61	-6	25123	7748
Oncology	-0.085	(0.126)	37	-3	6376	2368
Ophthalmology	0.175**	(0.087)	74	14	11314	3496
Orthopedic surgery	-0.029	(0.047)	67	-2	29250	8912
Otolaryngology	0.008	(0.063)	51	0	18519	6171
Pathology	-0.070	(0.065)	80	-5	15146	4992
Physical medicine and rehabilitation	-0.057	(0.120)	40	-2	6433	2383
Psychiatry	0.163	(0.263)	32	6	1375	535
Pulmonary disease	-0.127*	(0.068)	48	-6	16337	5372
Urology	-0.048	(0.054)	60	-3	22733	6881

The all specialty regression includes PCP-specialty and year fixed effects. All other regressions include PCP and year fixed effects. Only generalist PCPs are included in the sample. \*\*\* significant at the 1% level, \*\* significant at the 5% level, \* significant at the 10% level.

recording the referring physician on the hospital claim as the hospital acquires more PCP practices. However, this seems unlikely.

I find slight evidence in favor of a relationship between a PCP practice being owned by a hospital and how likely they are to refer to that hospital conditional on making a referral. My results are consistent with the shifting of referrals not being immediate. My results indicate a much smaller shift of referrals due to PCP-hospital integration than do Baker et al.'s (2016) results. Unlike Baker et al. (2016) I am able to include PCP-hospital (or PCP-Cardiologist) fixed effects to separate out how much of the shift in referrals to the hospital-owner is driven by the ownership relationship as opposed to a physician's inherent preference for a particular hospital. I find no evidence to support the hypothesis that PCPs shift their referrals towards Cardiologists that have the same hospital owner.

These conclusions rely on the assumption that referrals of acquired PCPs would follow the same trend as the referrals of unacquired PCPs if the acquired PCPs had not been acquired. A situation that could bias my results toward zero would be if PCP practices direct more referrals to hospitals in advance of the acquisition. The assumptions underlying the validity of the instrument are not critical as the conclusions of this paper would largely be the same if I used the models with fixed effects only, rather than the models with both fixed effects and the instrument.

My heterogeneity results are suggestive of hospital acquisition of PCPs causing the PCPs to redirect their poor patients away from their hospital-owner and redirect their wealthy patients toward their hospital-owner. However, they are not statistically significant. This prediction that PCPs would differentially redirect referrals based on patient income was motivated by several findings I made in Chapter 1: Poor patients experience similar reductions in unplanned hospital admissions when their PCP integrates with a hospital to wealthy patients. Meanwhile poor patients have a higher death rate and wealthy patients have a lower death rate after their PCP integrates with a hospital. I theorized that this

could be attributable to PCPs directing unprofitable or high-risk patients away from their hospital-owner and profitable or low-risk patients toward their hospital owner. I also find a suggestion of similar income based redirection of results in the inpatient referrals regressions. However, the inpatient referral results on their own are too weak to confirm this hypothesis alone.

In Section 2.6.4 I concluded that PCPs integrating with hospitals is not associated with an increase in the concentration of referrals to specialists. This is consistent with the result that PCPs are not much more likely to refer their patients to their hospital owner.

## 2.8 Conclusion

I use data on Medicare FFS patients and survey data on physician integration status from the company SK&A to test whether PCP-hospital integration results in changes in referral patterns. I use a number of different reduced form methods though I primarily rely on linear models with fixed effects and an instrument. I find no evidence that PCPs change their volume of referrals per patient-year in response to being acquired by a hospital. I do not find some evidence that suggests hospital acquisition of PCP practices may change the hospital to which the acquired PCPs refer. This does not hold for referrals to Cardiologists owned by the same hospital. I demonstrate that the difference between my results and the previous leading study on this topic (Baker et al., 2016) is primarily attributable to my addressing PCPs underlying preferences for particular hospitals.

## Chapter 3

# The association between hospital acquisition of primary care practices and the installation of electronic medical records in primary care practices

### 3.1 Introduction

My main result from Chapter 1 was that physician-hospital financial integration improved health outcomes among Medicare patients with chronic conditions. The installation of Electronic Medical Records (EMRs) in physician offices is one potential mechanism via which the financial relationship could be influencing patient health outcomes. Empirical evidence on the impact of physician-hospital integration on EMR installation is limited to a cross-sectional study with variation at the state level and little way to address omitted variable

bias. The primary contribution of this paper is to provide much more robust evidence on the impact of physician-hospital financial integration on EMR installation.

I rely primarily on physician survey data from the company SK&A collected in 2009, 2011, and 2013. My hypothesis is that...

*Hypothesis 3.1* hospitals that acquire physician practices will install EMR systems in those physician practices.

I use a variety of reduced form models to test this hypothesis, including linear models with year and physician-office fixed effects, and logit models with year and ZIP code fixed effects. I find strong evidence that hospitals install EMRs in physician practices when they acquire them.

In the next section of this chapter I give some background information about EMRs and discuss the prior paper on the association between physician-hospital integration and EMR use. In section 3.3 I describe my data and variables. In section 3.4 I outline my estimation strategy. I display my results in Section 3.5. In section 3.6 I discuss both how my results from this chapter compare to the previous paper on this topic, and how the results of this chapter relate to my results from the previous two chapters. I conclude this chapter in section 3.7.

## 3.2 Background

Primary care physicians (PCPs) often lack information about care provided to patients in other settings (Smith et al., 2005; Schoen et al., 2009; Mehrotra et al., 2011; Jones et al., 2014). If patients must repeat tests because their PCP does not have their results, this wastes time and money. Furthermore, if patients do not fully recall all their medications (not uncommon among elderly people with many health conditions) they risk unforeseen medication interactions. These issues arise from a lack of care coordination.

Electronic Medical Records (EMRs) have been touted as a solution to care coordination issues. However, EMR take-up is not straightforward. Firstly, EMRs are easily shared within a system but are not across systems. Consequently, the benefit to a physician practice of choosing a particular system is dependent on the systems chosen by the hospitals their patients attend, and the benefit to a hospital of using a particular EMR system depends on the systems chosen by referring physicians. Under joint ownership these externalities will be internalized. Secondly, EMRs require a large up-front investment. This can pose challenges, particularly for smaller physician practices.

Hospitals, with their much larger capital stock may be better positioned to make such large investments. Furthermore, their size may allow them to negotiate better rates with EMR providers. The large up-front investment, and the fact that the benefits accrue over a long time period, makes investment by a hospital in a physicians' office risky if there is some possibility that the physician-hospital linkage will break down. Similarly, it makes investment by a physicians' office in a system that aligns with a particular hospital risky. Co-ownership of a hospital and a PCP practice should ensure that the insurance plans covered are accepted at the hospital and PCP practice will be the same in the future. This should ensure that they share patients in future periods. Additionally, ownership of a physician practice by a hospital should give the hospital control over any investment they make in the physicians' facilities. Lammers (2013) identifies a positive relationship between the probability that a hospital uses health IT and the probability of the hospital using an integrated salary model, using variation in state laws that prevent hospitals from employing physicians. However, as the laws on hospital employment of physicians do not change over the period of his study, he is unable to rule out that some unobserved state characteristics could make health IT less appealing in states that also happen to ban hospital employment of physicians.

In this paper I investigate the impact of hospital-ownership on the probability that a physician practice installs an EMR. What I do here is distinct from Lammers (2013) in a

number of ways. Principally, I investigate the impact of hospital employment of physicians on the installation of EMRs *at the physicians' offices*, whereas Lammers (2013) investigates the impact of hospital employment of physicians on installation of EMRs *at the hospital*. Second, I am able to address time invariant unobservables due to having a panel data design.

### 3.3 Data and variables

To examine the impact of physician-hospital integration on EMR installation at physician practices, I rely on the SK&A data and Medicare data described in Chapter 2. There are a few features of the data that are particularly relevant to the EMR question. The survey question about whether the physician practice had an EMR system was asked only in 2009, 2011, and 2013. In 2011 and 2013 the survey also contains data on actual use of EMRs - whether EMRs were used for patient notes, whether they were used for viewing labs and X-rays, and whether they were used for prescriptions. However, these 3 indicators are highly correlated both with each other (correlation coefficients  $> 0.91$ ), and the indicator for whether the practice had an EMR system (correlation coefficients  $> 0.84$ ). Due to the EMR use indicators being highly correlated with the having an EMR indicator, and the EMR use indicators being available in less years, I conduct the study with the having an EMR indicator only. Occasionally in the data it appears that a practice has an EMR one year and no EMR the following year. Due to the high fixed costs of installing EMRs I assume that this reflects a data error and drop the practice from the dataset.<sup>1</sup>

I define each physician practice using the site identifier in the SK&A data. These site identifiers are based on street addresses.<sup>2</sup> Hence, when outpatient physician practice simul-

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<sup>1</sup>The results are robust to not doing this.

<sup>2</sup>Occasionally, the street address associated with a site identifier changes. I consider these to be likely data errors if the ZIP code changes. I ignore changes to the rest of the street address as most of those are due to discrepancies in the spelling of street names. I treat the most commonly occurring ZIP as correct and remove the site-years with the anomalous ZIP codes from the dataset.

taneously is acquired by a hospital and gets an EMR in the SK&A data, this reflects the installation of an EMR at the physician practice rather than the physicians relocating to the hospital, which already has an EMR.

### 3.3.1 Control variables

In several specifications I include an extensive set of control variables derived from the SK&A data, Medicare data, the American Hospital Association (AHA) Survey, and the Internal Revenue Service ZIP code level data. From the SK&A data I have the number of physicians at the practice site. Due to the highly skewed nature of the distribution of this variable I include indicators for the number of physicians at the practice being equal to 2, 3-5, 6-10, 11-20,  $\geq 21$ , rather than including the variable linearly. I also include miles from the practice's ZIP code's centroid to the nearest general-acute-care hospital. I construct this variable using hospital locations (latitude and longitude) from the American Hospital Association (AHA) survey, using practice ZIP codes from the SK&A data, approximating latitudes and longitudes of ZIP code centroids with ZCTA centroids from the 2010 U.S. Census Gazetteer file, and using the Stata command `geodist`, which calls Vincenty's (1975) equations. I also include the average adjusted gross income per household in the practice's ZIP code (2015 \$ thousands). The ZIP code income variable comes from the IRS ZIP code income data (Internal Revenue Service, 2014). Assuming physician practices would like to synchronize their patient records with nearby hospitals, practices that only has one hospital nearby will have an obvious choice of EMR system whereas practices with multiple hospitals nearby may have to choose which hospital to synchronize with. To account for this I include an indicator for the practice's HSA containing a single hospital. To some degree this variable may also pick up how urban the HSA is as urban regions are more likely to have multiple hospitals in the HSA.

The rest of the control variables are based either entirely or in part on the Medicare



FFS 5% sample described in the previous Section. To map site identifiers to the Medicare data I first identify all physicians-years who are affiliated with a single practice identifier in the SK&A data (this represents c.85% of physician-years in the SK&A data). I use these physicians to create a map from NPI-years to practice identifiers, which I use to add practice identifiers to the Medicare data. I estimate the fraction of each practice's daily patients who are Medicare FFS patients. To do so I determine the number of Medicare FFS patients in the 5% sample visiting a practice in any given day of the year and average this over the year. I multiply this by 20 and then divide by the average total number of patients per day visiting the practice according to the SK&A data. The Medicare data captures only patient visits to generalists and Cardiologists with single-practice affiliations due to the NPI-year to site identifier mapping being based only on these types of physicians. Meanwhile the SK&A data should capture patient visits to all specialties and multi-practice affiliated physicians. Consequently, my estimated fraction of each practice's daily patients who are Medicare FFS patients will on average be an underestimate. I also include an indicator for whether the site contains only generalists and Cardiologists (as opposed to physicians from other specialities). I construct this indicator by comparing the number of physicians at the site as reported in the SK&A survey to the number of physicians at the site in the subsample of the SK&A data that I purchased, which contains only generalists and Cardiologists.

Finally, I construct claim-weighted average<sup>3</sup> values of characteristics for the site's Medicare FFS patients who are  $\geq 65$ . The patient characteristics are age; indicators for the patient having each of the chronic conditions listed in the CMS chronic conditions warehouse (asthma; diabetes; chronic obstructive pulmonary disease; heart failure; hypertension; hyperlipidemia; ischemic heart disease; atrial fibrillation; kidney disease; Alzheimers, dementia, and other related disorders; acquired hypothyroidism; anemia; depression; hip fracture;

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<sup>3</sup>The weight assigned to each patient is equal to the number of claims they have with the practice's single-practice-affiliated generalists and Cardiologists over the year, divided by the total number of claims the single-practice-affiliated generalists and Cardiologists at that practice have over the year.

osteoporosis; stroke; acute myocardial infarction; benign prostatic hyperplasia; cataract; glaucoma; arthritis; breast cancer; colorectal cancer; prostate cancer; and lung cancer; endometrial cancer); a count of the number of chronic conditions the patient has; and indicators for the patient being Medicaid eligible, female, black, Hispanic, Asian or Pacific Islander, or other non-white.

### 3.4 Estimation strategy

To test whether physician-hospital integration affects the probability of a physician practice having an EMR installed, I use practice level regressions but cluster the standard errors at the Hospital Service Area (HSA) level. I regress an indicator of the practice having electronic medical records (EMRs) on an indicator of the practice being owned by a hospital.

I begin with the simple linear model and gradually add control variables, practice or area fixed effects, and non-linearity. The simple linear model with control variables is

$$y_{kt} = \alpha + \beta v_{kt} + \gamma \mathbf{x}_{kt} + \phi_t + e_{kt} \quad (3.1)$$

where  $y_{kt}$  is an indicator that is 1 if physician practice  $k$  has an EMR in year  $t$  and 0 otherwise, and  $v_{kt}$  is an indicator that is 1 if the physician practice is integrated with a hospital and 0 otherwise,  $\phi_t$  are year fixed effects, and  $\mathbf{x}_{kt}$  are the control variables outlined in Section 3.3.1. It is possible that an unobserved variable such as physician preference for technology could both drive physician practices to install EMRs and cause them to be more likely to affiliate with hospitals (since hospitals tend to be more technology heavy than physician practices). Consequently the estimates of  $\beta$  in this model could be upwardly biased. To help address this I introduce site fixed effects,  $\delta_k$  resulting in the model

$$y_{kt} = \alpha + \beta v_{kt} + \gamma \mathbf{x}_{kt} + \phi_t + \delta_k + \varepsilon_{kt} \quad (3.2)$$

An unobserved physician preference for technology would only remain a problem if it is time varying differently between physicians at integrating and never-integrating sites. The site fixed effects specification cannot rule out that hospitals are buying physician practices because the physician practice installed an EMR. However, this seems highly unlikely due to the difficulty of sharing information between different EMR systems. Even with the site fixed effects I cannot rule out that PCPs may be approaching hospitals to acquire their PCP practice because they want an EMR installed. Consequently, my fixed effects results should not be extrapolated to physicians that did not integrate.

The simple linear model assumes  $e_{it}$  follows a normal distribution and the linear model with fixed effects assumes  $\varepsilon_{kt}$  follows a normal distribution, which is not quite appropriate given that the outcome variable is binary. Given the binary outcomes, the logit model is more appropriate than the linear model. The logit model with control variables and time fixed effects can be described as follows: suppose the site achieves net value  $\pi_{kt}$  from installing an EMR, this value follows the function described in equation 3.3,  $e_{kt}$  is distributed according to a type I extreme value distribution, and that installation of an EMR is determine according to equation 3.4.

$$\pi_{kt} = \alpha + \beta v_{kt} + \gamma \mathbf{x}_{kt} + \phi_t + e_{kt} \quad (3.3)$$

$$y_{kt} = \begin{cases} 1 & \text{if } \pi_{kt} \geq \pi^* \\ 0 & \text{if } \pi_{kt} < \pi^* \end{cases} \quad (3.4)$$

Similarly to in the linear model, I would like to include site fixed effects. This would lead to me replacing equation 3.3 with equation 3.5, where  $\varepsilon_{kt}$  is assumed to follow a type I extreme value distribution. However, since I have at most three years of data for each site my estimate of  $\beta$  will be biased due to the incidental parameters problem (even in the linear model). In the non-linear model there is the additional problem of computational feasibility. To address these issues I follow Bester & Hansen's (2016) approach of estimating

the model using “group effects”. This approach involves selecting a “group” in which you believe the individual level fixed effects are correlated, then using group effects in place of individual fixed effects in the estimation. I use the ZIP code of the site as the grouping variable. Estimating a non-linear group effects model trades off bias due to the incidental parameters problem and bias due to misspecification of the functional form of the model. The group effects model estimates the system of equations defined by 3.6 and 3.4, where  $z$  enumerates the ZIP code in which the physician practice is located.

$$\pi_{kt} = \alpha + \beta v_{kt} + \gamma \mathbf{x}_{kt} + \phi_t + \delta_k + \varepsilon_{kt} \quad (3.5)$$

$$\pi_{kzt} = \alpha + \beta v_{kt} + \gamma \mathbf{x}_{kt} + \phi_t + \delta_z + \varepsilon_{kt} \quad (3.6)$$

A caveat of the results found with these models is that even in the fixed effects models with control variables I rely on changes in *observed* patient characteristics to capture influences the changing patient composition may have on the choice to install an EMR. As noted elsewhere in this dissertation, an integrated physician practice may attract patients that are different on their unobserved characteristics than the patients attracted to non-integrated physician practices.

### 3.4.1 Leads and lags regressions

It is possible physician-hospital integration’s effect on EMR installation may not be instant. To test for both a delayed response and pre-trends I replace the term  $\beta v_{kt}$  in the regression equations outlined above with a set of indicators for the number of years until a practice integrates and the number of years since a practice has integrated. I exclude physician practices that were already integrated in the first year they appear in the data (usually 2007) from the sample, since it is not possible to tell how long these practices have been integrated for. Similarly, I exclude physician practices that are still independent in their

last year in the data (usually 2013) from the sample, since not possible to tell when, if ever, these practices will integrate. This subsample of physicians is the same set of physicians that identify the effect of integration in the main EMR regressions with practice fixed effects.

### 3.5 Results

Table 3.1: Marginal effect of physician practice being owned by a hospital on the probability of the practice having an EMR

<i>Panel A: Linear models</i>				
fixed effects	year	year	year & site	year & site
controls	N	Y	N	Y
marginal effect	0.142***	0.078***	0.046***	0.043***
S.E.	(0.011)	(0.011)	(0.011)	(0.013)
<i>Panel B: Logit models</i>				
fixed effects	year	year	year & ZIP	year & ZIP
controls	N	Y	N	Y
marginal effect	0.146***	0.084***	0.114***	0.059***
S.E.	(0.012)	(0.012)	(0.009)	(0.016)

All models include a constant. Standard errors are clustered at the Hospital Service Area (HSA) level. \*\*\* significant at the 1% level. Control variables include: site size indicators (site size = 2, 3-5, 6-10, 11-20,  $\geq 21$ ), miles from site to the nearest general-acute-care hospital, average adjusted gross income in the site's ZIP code (2015 \$ thousands), estimated % of site's daily patients who are Medicare FFS patients, an indicator for the site only containing generalists and/ or Cardiologists, a 1 year lag of an indicator for the HSA containing a single hospital, and average values of characteristics for the site's Medicare FFS patients who are  $\geq 65$ . The patient characteristics are age; a count of chronic conditions; indicators for patients being Medicaid eligible, female, black, Hispanic, Asian or Pacific Islander, or other non-white; and indicators for the patient having each of the chronic conditions listed in the CMS chronic conditions warehouse.

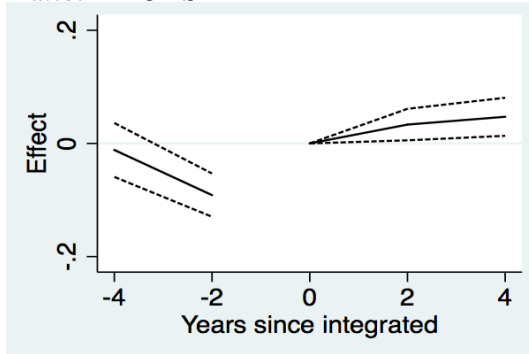
Table 3.1 shows the estimated marginal effect of a physician practice being vertically integrated on the probability of having an EMR system, resulting from all models described above. Across all specifications of the model there is a consistent positive and significant (at the 1% level) relationship between a site being owned by a hospital and the site using

EMRs.

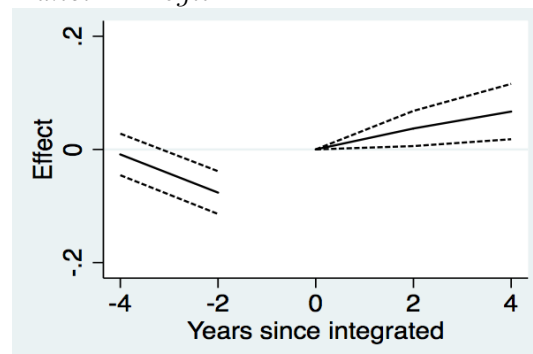
### 3.5.1 Leads and lags

Figure 3.1: Marginal effect of years since the physician practice was acquired by a hospital on the probability of the practice having an EMR - regressions without fixed effects

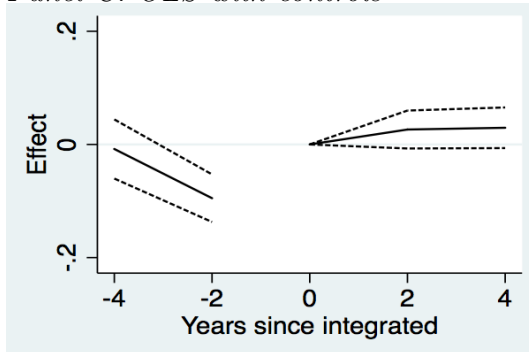
Panel A: OLS



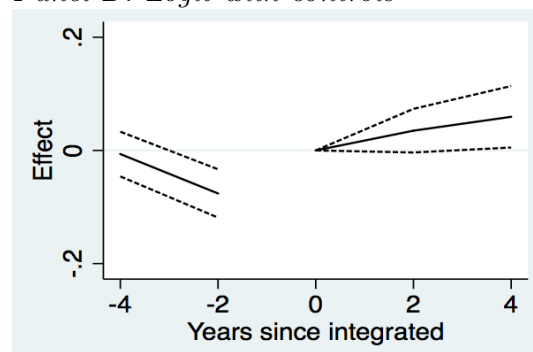
Panel B: Logit



Panel C: OLS with controls



Panel D: Logit with controls

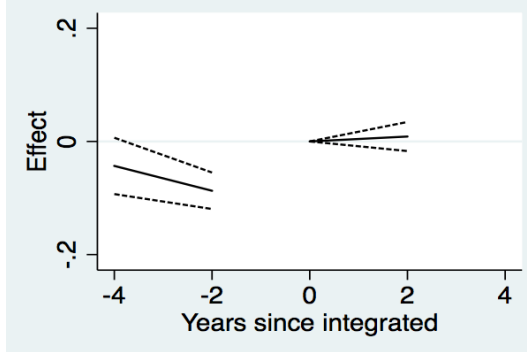


The solid black line is the point estimate. The dotted black lines enclose the 95% confidence interval. All regressions included year fixed effects and a constant.

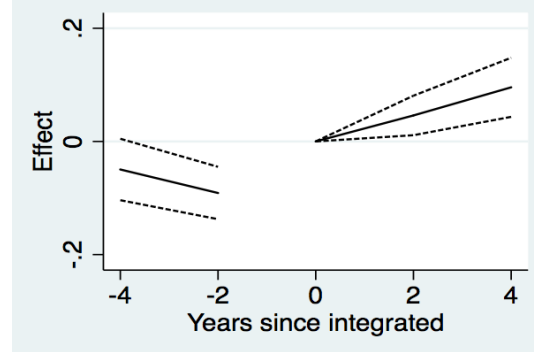
Figures 3.1 and 3.2 display the pre- and post-integration trends in EMR installation in eight different model specifications. The horizontal axis shows the number of years since the physician practice integrated. Year zero is the first year the practice is recorded as being owned by a hospital in the SK&A physician survey. Negative years since integration indicate the amount of time before the physician practice will be acquired. The vertical axis shows the estimated effect of the practice being acquired  $x$ -years ago.

Figure 3.2: Marginal effect of years since the physician practice was acquired by a hospital on the probability of the practice having an EMR - regressions with fixed effects

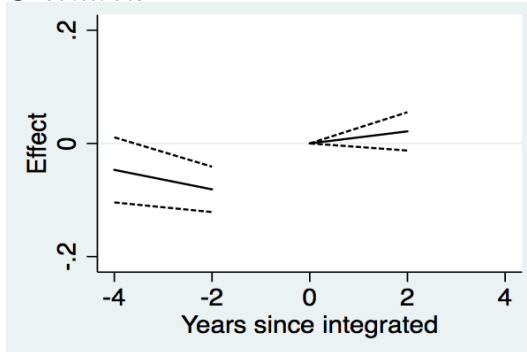
*Panel A: Linear with practice fixed effects*



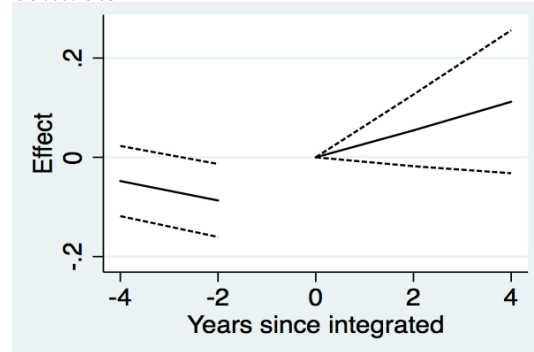
*Panel B: Logit with ZIP fixed effects*



*Panel C: Linear w. practice fixed effects & controls*



*Panel D: Logit with ZIP fixed effects & controls*



The solid black line is the point estimate. The dotted black lines enclose the 95% confidence interval. All regressions included year fixed effects and a constant.

All models exhibit evidence of a downward pre-acquisition trend in EMR uptake (see Figure 3.1 and Figure 3.2). This is likely a consequence of the expected probability of having an EMR increasing over time, which is captured by the year fixed effects. As expected from my main results, all models demonstrate an increase in EMR uptake in the first year they appear as acquired in the survey. Post-acquisition, four of the eight models (Figure 3.1 Panels A, B, and D; and Figures 3.2 Panel B) show a statistically significant upward trend. Two of the remaining four models show an upward trend in their point estimate that is not significant at the 5% level. I conclude that most of the effect of integration occurs in the first year of integration in my data. However, some mergers may involve a delayed response.

The apparent immediacy of EMR installation resulting from hospital acquisition of physician practices could be reflecting the relative coarseness of my timing data. Since I only have integration data every other year, it is possible that the physicians may have integrated as much as two years previously.

### 3.6 Discussion

I find strong empirical evidence to support the hypothesis that physician-practices are installing EMRs when they are acquired by hospitals. Technically I cannot rule out that they may be spontaneously installing the EMRs at the same time as the practice is acquired by a hospital and not due to the practice being acquired by a hospital. However, I have strong theoretical underpinnings for believing this to be unlikely (see Section 3.2). My finding that physician-practice acquisitions are associated with the installation of EMRs roughly aligns with the finding of Lammers (2013) but my estimation strategy eliminates many of the concerns about omitted variable bias in Lammers (2013) that arise from his reliance on cross-sectional variation in state policies. The reason I claim only rough alignment with Lammers' (2013) results is that I find physician-hospital integration results in the installation of EMRs at the physician practices, whereas Lammers (2013) shows a relationship between physician-hospital integration and installation of EMRs at the hospitals.

Combining the findings of this Chapter, with the findings from Chapter 2 (on the impact of integration on referral patterns, and the findings from heterogeneity section in Chapter 1 (see Section 1.6), I conclude that likely candidates for driving the relationship between physician-hospital integration and improved patient outcomes (in terms of reductions in unplanned hospital admissions): installation of EMRs, and better outpatient care quality. I find that physician-physician coordination is not a likely explanation (though this does not rule out PCP-hospital coordination), nor is more referrals. I also find that the heterogeneity



in the effect of physician-hospital integration on death rates across patient income, may be explained by physicians directing poor patients away from their hospital-owner and wealthy patients towards their hospital-owner after the acquisition.

## 3.7 Conclusion

I use data on Medicare FFS patients and survey data on physician integration status from the company SK&A to test whether PCP-hospital integration results in the installation of EMRs at physician offices. I find evidence that hospitals install EMRs in physician practices when they acquire them. This result is robust across several linear and logit specifications with PCP fixed effects and ZIP code fixed effects respectively.

In conjunction with Chapters 1 and 2, this chapter suggests that drivers of the positive effect of physician-hospital integration on average health outcomes may include better outpatient care quality and the installation of EMRs in physician practices, but likely exclude physician-physician coordination or more care by specialists. My results are consistent (albeit weakly) with physicians directing their poor patients away from their hospital-owner and wealthy patients towards their hospital-owner, which may explain my finding in Chapter 1 that poor patients have higher death rates after their PCP is acquired by a hospital and wealthy patients have lower death rates after their PCP is acquired, in spite of poor patients not losing access to primary care when their PCP integrates.

## Chapter 4

# The association between Patient Safety Indicators and medical malpractice risk: Evidence from Florida and Texas

This chapter was coauthored with Bernard S. Black<sup>1</sup> and Zenon Zabinski<sup>2</sup>. It is also published in the American Journal of Health Economics vol. 3, issue 2, spring 2017. The copywrite agreement allowing inclusion in this dissertation is in Appendix C.4. As per this agreement the authors request any citations refer to the AJHE publication.

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## 4.1 Introduction

A central goal of tort liability is to deter risky or negligent behavior by imposing liability on the “acting” party for harm to an injured party. This creates incentives for the acting party to take precautions to prevent injury. However, if tort suits are unrelated, or only weakly related, to actual injury or negligence, tort liability may impose costs on acting parties without creating appropriate incentives to change behavior.

In the United States, medical patients who believe they have been the victims of negligent medical care may attempt to recover damages by bringing medical malpractice lawsuits against health-care providers. The threat of suit may deter negligence by providers. However, advocates of caps on noneconomic damages and other reforms that would discourage malpractice suits argue that the medical malpractice liability system in the United States is largely a “lawsuit lottery” in which many claims are filed in cases with no negligence and juries often award damages in cases with no negligence (American Medical Association 2012). At the same time, a high proportion of medical injuries with apparent negligence do not lead to malpractice claims (Baker 2005a). If malpractice suits are weakly related to negligent care, the risk of being sued will not create incentives for hospitals to invest in care quality. Conversely, a strong association between adverse patient safety events and medical malpractice claim rates suggests that hospitals can reduce malpractice claims by making the investments, often in people and training rather than capital equipment, needed to reduce adverse events.

We study here the association between rates of adverse patient safety events and rates for paid medical malpractice claims (below, simply “claims” or “malpractice claims”), using data from Florida and Texas, the only states with publicly available data on these claims. In Florida, we find evidence, with hospital fixed effects and extensive covariates, that adverse event rates predict malpractice claim rates. Our point estimates suggest hospitals can

meaningfully reduce malpractice claims by investing in patient safety. An improvement from one standard deviation above to one standard deviation below the expected adverse event rate predicts a 32 percent drop in paid malpractice claims. In Texas, we have only county-level data on malpractice claim rates, but obtain similar point estimates, using county fixed effects.

The prior literature examining the relationship between patient safety and medical malpractice claim rates is limited. The principal study is Greenberg et al. (2010), who study the relationship between adverse events and malpractice claim rates in California. They find that county-level variation in adverse event rates predicts variation in malpractice claim rates.<sup>3</sup> This study is subject to several limitations, however. They lack hospital-level data on malpractice claims, so cannot control for hospital-level confounders. Their data cover only a subset of insurers from one state, and their measure of adverse events is a raw sum of events, which gives dominant weight to high frequency (often low severity) events. They also do not link specific types of adverse events to specific types of malpractice claims. We address these shortfalls, using hospital-level data from Florida.

Our study is also related to ex-post studies of whether the medical malpractice lawsuits that are brought appear to involve actual negligence. These studies generally find that many, but far from all, malpractice claims involve probable medical error. The leading study is Studdert et al. (2006); see also the review of earlier studies by Baker (2005b). We also contribute to a broader literature studying the factors that predict malpractice claims. For physicians, one malpractice claim predicts future claims (Bovjberg and Petronis 1994; Sloan et al. 1989). A personal style that leads to patient complaints also increases malpractice risk (Hickson et al. 2002).

We complement the malpractice claim data with data on adverse patient safety events,

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<sup>3</sup>See also Grunebaum, Chervenak, and Skupski (2011), who report on the effort by a major academic center to reduce adverse childbirth events, and the resulting drop in malpractice claims.

using Florida and Texas “state inpatient data sets” which cover all hospital discharges from each state. We use these inpatient data sets to measure rates for 17 Patient Safety Indicators (PSIs). The PSIs were developed by the Agency for Healthcare Research and Quality (AHRQ) and provide measures of patient safety by capturing often-avoidable adverse events, such as wound or bloodstream infections, or sponges left in the body during surgery.

Our underlying causal model is the following: (1) the PSIs, especially when pooled, are a proxy for overall patient safety; (2) poor patient safety practices cause medical errors captured by the PSIs, and also likely proxy for other unobserved causes of medical errors; and (3) some medical errors lead to malpractice claims. Although we lack an external shock to PSI rates, our results are likely to be causal. Reverse causation is not a plausible explanation, because higher malpractice risk should, if anything, induce hospitals to pay more attention to patient safety. This should reduce PSI rates. Thus, any reverse causation should bias our estimates downward. For causal inference, the more serious concern is omitted variable bias. However, we have a strong empirical specification, especially in Florida where we can use hospital fixed effects (hospital FE). We combine the hospital FE with extensive patient-level covariates that should control for any changes in patient mix over time. Patient mix and hospital characteristics should also be reasonably stable over time, so unobserved patient and hospital characteristics should be largely captured by the hospital FE.

To determine whether PSI rates predict malpractice claim rates, we adopt a two-step approach. First, we construct measures of residual claim rates and PSI rates, controlling for an array of hospital-level covariates. These residual measures capture whether a hospital has more adverse events or malpractice claims than one would expect, based on the covariates. The residual PSI measures are positively correlated, suggesting that, especially when pooled, they are a reasonable proxy for (unobserved) overall “patient safety.”

In the second step, we use panel data methods to assess whether the residual PSI measure predicts the residual malpractice claims measure. We find a strong positive association

between residual PSI rates and residual malpractice claim rates. This suggests that paid malpractice claims are nonrandom events, and that hospitals can reduce their paid claim rates by improving patient safety. For Florida, the coefficient on our pooled PSI measure is 0.141. If our results are causal, this implies that a one standard deviation reduction in PSI rates would decrease paid malpractice claims by about 16 percent.

We also document substantial variation in PSI rates at the hospital level. For example, among the 106 large Florida hospitals in our data set (>10,000 discharges in 2010), annual total PSI rates range from 55 to 390 per 10,000 discharges ( $\sigma = 53$ ). This wide variation in PSI rates implies that much lower PSI rates are achievable at reasonable cost, since some hospitals are achieving them.

This chapter proceeds as follows. Section 4.2 provides background on medical malpractice litigation and patient safety in the United States. Section 4.3 describes our data sources, and Section 4.4 details our empirical methodology. Section 4.5 presents our main, hospital-level results from Florida on the association between PSI rates and malpractice claim rates. Section 4.6 shows that we obtain similar results in Texas, with county-level malpractice claim data. Section 4.7 discusses our findings and their implications, and Section 4.8 concludes. Appendix C contains additional material relating to this chapter.

## 4.2 Background

### 4.2.1 Medical Malpractice Litigation

In the United States and many other countries, patients may bring lawsuits against health-care providers alleging injury due to negligent care. If liability is found, the claimant may recover economic damages, noneconomic damages, and punitive damages. Economic damages are composed of monetary losses due to the injury including medical expenses and lost earnings. Non-economic damages are non-monetary harm, such as pain and suffering. Punitive

damages are available in theory, but are rarely awarded in practice, and even more rarely paid (Hyman et al. 2007).

Reform of the US medical malpractice system has long been a contested public policy issue (American Medical Association 2012). The principal arguments made by the American Medical Association and other reform advocates include claims that liability fears lead providers to practice “defensive medicine,” driving up health-care costs (e.g., Kessler and McClellan 1996, 2002; Sloan and Shadle 2009; Paik, Black, and Hyman 2017); and claims that liability fears lead physicians to locate in lower-risk jurisdictions (e.g., Matsa 2007; Helland and Seabury 2015; Paik, Black, and Hyman 2016).

Of more direct relevance to this study, reform advocates also claim that many malpractice claims are frivolous and that many cases with no actual medical error lead to payouts (see literature discussion above). An important policy question is whether hospitals can reduce malpractice risk by making investments that reduce the incidence of medical error.

### 4.2.2 Patient safety

Patient safety has been a focus for the health-care industry in the United States since the publication of the seminal report by the Institute of Medicine, *To Err Is Human: Building a Safer Health System* (Kohn, Corrigan, and Donaldson 2000), which brought to public attention the high rate of medical errors in the United States and the resulting death toll. Since the publication of *To Err Is Human*, increased attention has been paid to measuring patient safety, including identifying sources of medical error, public reporting of patient safety outcomes, and a variety of both voluntary and government-mandated initiatives to reduce the frequency of adverse events.

The Agency for Healthcare Research and Quality (AHRQ) developed the Patient Safety Indicators (PSIs) as measures of patient safety that can be calculated using standard hospital inpatient data sets. Hospital inpatient data sets are available in many states, including

Florida and Texas. The PSIs are designed to measure often avoidable adverse events in general acute-care hospitals. They were developed by researchers at Stanford University; University of California, Davis; and University of California, San Francisco, under an AHRQ-commissioned project. The development process involved several stages. The researchers began by identifying over 200 potential indicators, assessed their validity, developed a much smaller number of proposed indicators, and conducted a review of the proposed indicators by clinical panels.<sup>4</sup> These measures are widely used to assess patient safety in hospitals; to evaluate the effectiveness of patient safety initiatives; and to study the determinants of patient safety. For example, the Leapfrog Group uses PSIs as part of its annual survey of safety and quality in US hospitals (Leapfrog Group 2015).

Table 4.1 lists and describes the set of PSIs defined by AHRQ, many of which are severe events, including death. For example, PSI-2 is death in the hospital, for patients with conditions for which in-hospital death is rare. PSI-3 is pressure ulcer, usually due to failure of hospital staff to turn an immobile patient often enough. PSI-4 is death from serious but treatable complications that generally involve medical error to begin with. PSI-5 is leaving a foreign object, usually a sponge, in the body during surgery. PSI-6 is collapsed lung suffered in the hospital. And so on.

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<sup>4</sup>The PSIs are described at [http://www.qualityindicators.ahrq.gov/Modules/psi\\_resources.aspx](http://www.qualityindicators.ahrq.gov/Modules/psi_resources.aspx); see also Encinosa and Bernard (2005).



Table 4.1: Patient safety indicators (PSI) descriptions

<b>PSI</b>	<b>Short description</b>	<b>Fuller description</b>
2	Death in Low-mortality DRGs	In-hospital death for patients with a Diagnosis Related Group (DRG) with less than 0.5% mortality rate. Excludes cases of trauma, cancer, or immunocompromised states.
3	Pressure Ulcer	High severity pressure ulcers in a patient who stays in hospital for 5 or more days. Excludes patients with who were admitted due to skin disease, subcutaneous tissue disease, breast disease; patients who are paralyzed, have spina bifida or anoxic brain damage; and cases relating to pregnancy or childbirth.
4	Death of Surgical Inpatients with Serious Treatable Complications	Serious treatable complications include pulmonary embolism or deep vein thrombosis (see PSI-12 description); pneumonia; sepsis (see PSI-13 description); shock or cardiac arrest; and gastrointestinal hemorrhage or acute ulcer.
5	Foreign Body Left during Procedure	Occurs when a foreign object, such as a sponge used during surgery, is not removed from the patient's body after the procedure.
6	Iatrogenic Pneumothorax	Collapsed lung due to a medical procedure. Excludes patients with diagnosis of chest trauma or fluid around the lungs or who received a thoracic procedure, a lung biopsy, a cardiac procedure, or diaphragmatic surgery repair.
7	Central Venous Catheter-related Bloodstream Infection	Also known as Central line-associated bloodstream infections or CLABSI. Occurs when a central line, inserted into a major vein, becomes infected, leading to a bloodstream infection. Excludes patients with cancer or in immunocompromised states.
8	Postoperative Hip Fracture	Occurs when a patient suffers a hip fracture during a hospital stay after a surgical procedure. Excludes patients with a hip fracture as their primary diagnoses or with musculoskeletal or connective tissue diseases.
9	Postoperative Hemorrhage or Hematoma	Bleeding or bruising after a surgical procedure.
10	Postoperative Physiologic and Metabolic Derangement	Includes a number of diabetes-related complications and acute kidney failure. Excludes patients with non-elective surgeries.
11	Postoperative Respiratory Failure	Occurs when the respiratory system is unable to deliver oxygen to the bloodstream or remove carbon dioxide. Excludes patients with non-elective surgeries.
12	Postoperative Pulmonary Embolism or Deep Vein Thrombosis	Pulmonary embolism is a blockage in a lung artery. Deep vein thrombosis is a blood clot that develops in a vein deep in the body.

13	Postoperative Sepsis	Sepsis after a surgical procedure. Sepsis is a severe immune response to a bacterial infection, which causes decreased blood flow, potentially leading to organ failure or shock. Excludes patients with non-elective surgeries.
14	Postoperative Wound Dehiscence	Occurs when a wound reopens after surgery. Cases-at-risk are limited to surgeries performed on the abdomen or pelvis.
15	Accidental Puncture or Laceration	An accidental cut, puncture, perforation, or laceration during a medical or surgical procedure. Excludes spine surgeries.
16	Transfusion Reaction	Occurs when the patient has a reaction against the blood received in a blood transfusion. Such a reaction can be fatal.
17	Birth Trauma – Injury to Neonate	Includes a number of severe injuries to a newborn, including hemorrhage or injury to the spine or skeleton. Excludes preterm newborns weighing less than 2,000 grams, infants with brittle bone disease, and infants with brachial plexus injuries.
18	Obstetric Trauma – Vaginal Delivery with Instrument	Injuries to the mother during vaginal deliveries with assistance of an instrument.*
19	Obstetric Trauma – Vaginal Delivery without Instrument	Injuries to the mother during vaginal deliveries without assistance of an instrument.*

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Sources: AHRQ PSI definitions (AHRQ 2011); MedlinePlus at nih.gov; Medscape.com. PSIs -3, -6, and -8 to -15 include only patients aged 18+. PSIs -2, -4, -5, -7 and -16 include both patients aged 18+ and patients who are pregnant, giving birth, or have recently given birth.

\* There is no PSI for injury to the mother during a Cesarean section. These injuries were formerly in PSI-20, but AHRQ later removed this PSI.

For each PSI, AHRQ provides specific criteria for identifying PSI events and determining which patients are at risk for that PSI. These criteria include admission type and source, patient age, ICD-9-CM diagnosis and procedure codes, time between procedure date and adverse event date, Diagnosis Related Group (DRG), Major Diagnostic Category (MDC), length of stay, and patient discharge type. For instance, cases at risk for postoperative sepsis (PSI-13) include all elective surgical discharges (based on DRG and ICD-9-CM codes) of patients 18 and older, excluding discharges where the patient was admitted with sepsis or infection and patients who are immunocompromised or have specified cancers. The count

of PSI-13 events is the subset of these cases at risk with an ICD-9-CM code of sepsis in a secondary diagnosis field (AHRQ 2011).

In this study we interpret PSI events not as event-specific precursors to malpractice claims, but instead as proxies for overall hospital patient safety. Some PSI events could lead to paid malpractice claims, but so could many other adverse outcomes that are not PSIs. The PSIs are imperfect proxies for overall safety in a number of ways. First, the PSIs are based on billing records, rather than full clinical records. The imperfect fit between PSIs and actual adverse events will introduce noise into the PSI measures. This noise may not be random, and thus might bias our estimates, if reporting practices vary across hospitals. However, hospital FE should capture much of this nonrandom variation. Second, the PSIs measure a subset of adverse events. They were designed to (1) capture adverse events that are usually preventable; and (2) have relatively high specificity (the fraction of PSIs which in fact reflect adverse events), at the cost of reduced sensitivity (the fraction of adverse events identified).

A number of studies assess how well the PSIs perform as patient safety measures. We review here some representative studies. Classen et al. (2011) compare PSI rates to a broader set of adverse events identified from clinical records. They find that the PSI measures have specificity of 98.5 percent, but sensitivity of only 8.5 percent. Thus, the PSIs miss many adverse events, but rarely provide false positives.<sup>5</sup> Zhan and Miller (2003) report that patients with PSI events had longer hospital stays and higher mortality, compared with matched patients without PSI events.<sup>6</sup> Singer et al. (2009) report an association between PSI rates and a survey-based measure of the patient safety climate.

In contrast, Isaac and Jha (2008) study four PSIs (PSI-2, death in low-mortality DRGs;

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<sup>5</sup>Other similar studies include Romano et al. (2009), studying PSIs 10 through 14; Utter et al. (2009), studying PSI-15; White et al. (2009), studying PSI-12, and a series of studies of different PSIs in VA hospitals, Rosen and Itani (2011).

<sup>6</sup>Rivard et al. (2008) find similar results for VA patients as do Raleigh et al. (2008) for the United Kingdom.

PSI-3, pressure ulcer; PSI-4, death of surgical inpatients with serious treatable complications; and PSI-7, central venous catheter-related bloodstream infection) for Medicare patients in 2003. They compare PSI rates with (1) risk-adjusted mortality rates; (2) “process measures” of care quality from the HHS Hospital Compare project; and (3) US News hospital rankings. Lower PSI-4 rates predict lower risk-adjusted mortality, but they find mixed results for the other PSIs and the other quality measures. However, there is reason to question their quality measures. The HHS measures are poor predictors of outcomes (e.g., Nicholas et al. 2010). The US News rankings largely measure reputation. Whether they capture patient safety or other aspects of overall “quality” is unknown. Also, a measure that pools a number of PSIs could predict overall safety, even if some individual measures do not.

These studies all assess whether the PSIs have good “construct validity” in two senses. First, is the PSI construct a good predictor of the underlying medical event: Do the patients who are coded as having PSIs actually have the underlying condition? Second, do the PSIs predict outcomes: Do patients who are coded as having PSIs realize worse outcomes, on average, than similar patients who do not suffer PSIs? Our overall sense of this research is that the PSIs, especially if pooled, are likely to provide a reasonable, if noisy, proxy for overall patient safety.

There is extensive research, much of it associated with the Dartmouth Atlas of Health Care project, on local variation in health-care intensity, and the limited association between treatment intensity and outcomes (e.g., Fisher et al. 2003; Baicker and Chandra 2004). Only one study examines local variation in patient safety. Thornlow and Stukenborg (2006) study five PSIs (death in low-mortality DRGs, death of a surgical inpatient with serious treatable complications, central-line-associated bloodstream infection, post-operative hemorrhage or hematoma, and postoperative respiratory failure). They find that hospital ownership (for profit, nonprofit, or government), location (urban versus rural), and teaching status are weak predictors of PSI rates.

## 4.3 Data

To study the association between medical malpractice claim rates and patient safety, we need measures of both. Our measures of malpractice claim rates come from public data sets produced by the Florida Office of Insurance Regulation and the Texas Department of Insurance. These data sets include only closed, paid claims. We lack data on unpaid claims, and learn about claims only when they are closed. We use the claims data sets to estimate paid claims by injury year. We use hospital inpatient data sets from Florida and Texas to compute PSI rates.

### 4.3.1 Medical malpractice claims data

#### 4.3.1.1 Florida office of insurance regulation data

Our primary source for malpractice claims data is Florida. The Florida Office of Insurance Regulation provides data on closed, paid medical malpractice claims to researchers for a nominal charge on request (to [PublicRecords@floir.com](mailto:PublicRecords@floir.com)). We use the “current” Florida data set, which includes claims due to injuries between 1994 and 2014.<sup>7</sup> This data set covers claims against both physicians and hospitals. It includes injury setting (e.g., hospital inpatient facility, hospital outpatient facility, physician’s office, patient’s home) and hospital identifiers, if applicable. Since PSIs are measured in hospitals, we keep only medical malpractice claims where the injury was in a hospital inpatient setting. Moreover, PSIs are intended for use in general acute-care (GAC) hospitals, so we limit the sample to these hospitals. Our final sample includes 219 hospitals, which account for 95 percent of discharges in our inpatient data set.<sup>8</sup>

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<sup>7</sup>Florida medical malpractice data are also available for claim closing years from 1975 to 1993 (with some records extending to 1H 1999) in a different data format. We do not use the earlier data in this study. We discard claims with zero payout since Florida ceased to require reporting of zero payout claims in 1997.

<sup>8</sup>We exclude 6 hospitals without names (following advice from the Florida Agency for Health Care Administration, the inpatient data provider). We then identify and exclude 69 non-GAC hospitals in several steps.

#### 4.3.1.2 Texas department of insurance data

Our second source for malpractice claims data is Texas. The Texas Department of Insurance (TDI) data provide data on five lines of commercial personal liability insurance, including medical malpractice (TDI 2014), for paid claims closed over 1988-2012.<sup>9</sup> The data set includes county of injury, but, unlike Florida, does not include hospital identifiers, so we conduct our Texas analysis at the county level.

#### 4.3.1.3 Time consistency

We discuss Florida here, and discuss Texas in Appendix C. Figure 4.1 shows the number of closed, paid medical malpractice claims in Florida by injury year. There is a drop in paid claims beginning in 2004. There are two main reasons for this drop. First, the data are right-censored – they contain only closed claims, so claims for injuries that occurred in more recent years but did not close by the end of 2014 are excluded. Second, in 2003, Florida adopted a cap on noneconomic damages in medical malpractice cases, which substantially reduced claim rates (Paik, Black, and Hyman 2013).

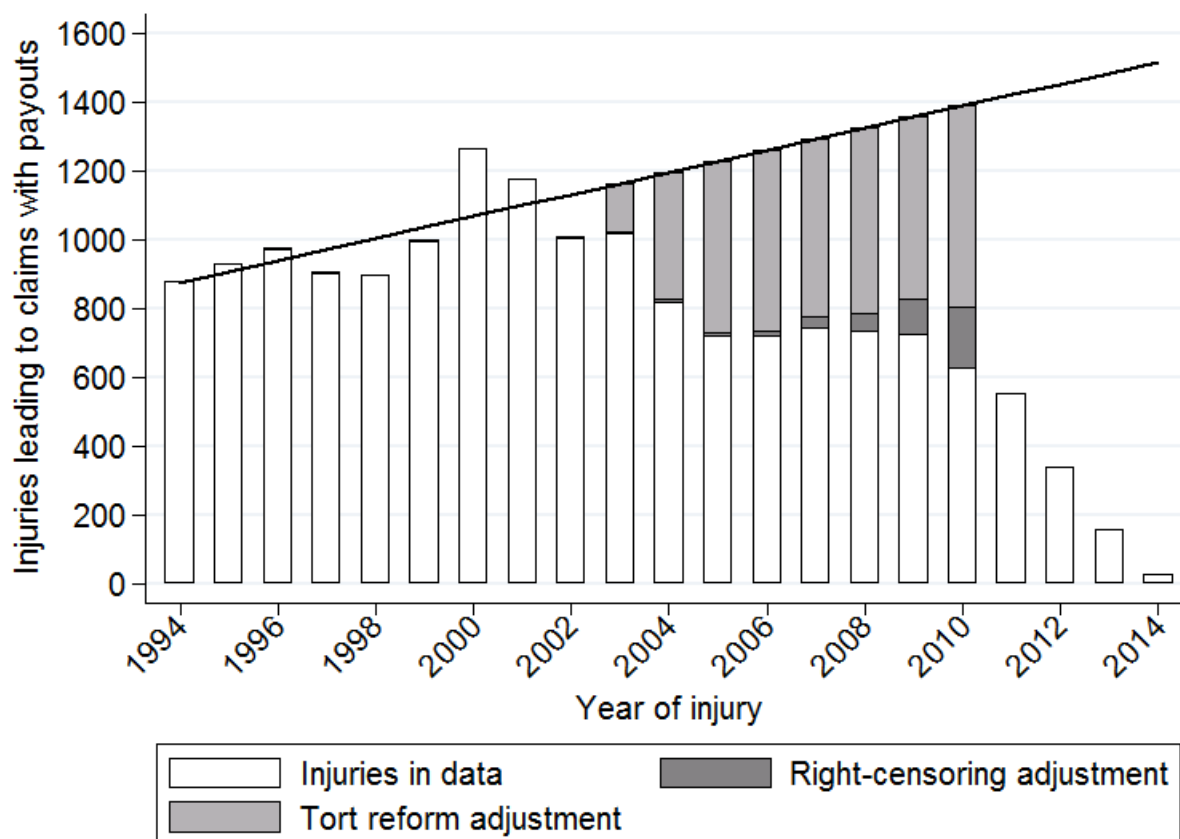
To make our measure of malpractice activity more time-consistent, we adjust for these two effects, but as we show below, we obtain very similar results without these adjustments.

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First, we check hospital service type in the American Hospital Association (AHA) survey data, and exclude hospitals that are not “general medical and surgical.” For 10 hospitals whose service category changes over time, we use the most common designation. Second, for 27 hospitals that are not in the AHA data, we parse all versions of the hospital name in the inpatient and medical malpractice data sets for character strings that indicate non-GAC status, and classify a hospital as non-GAC if any version of its name contains one or more of the strings, “behavioral,” “psychiatric,” “long term,” “rehab,” “addict,” “recovery center,” “residential,” “child,” or “specialty.” For the 10 remaining hospitals, we conduct a manual Internet search to determine the hospital type.

<sup>9</sup>Our criteria for defining a medical malpractice claim follows Paik et al. (2012): a claim must satisfy at least two of three criteria: (1) payment under medical professional liability insurance; (2) physician or hospital defendant; (3) injuries caused by “complications or misadventures of medical or surgical care.” We have cause of injury only for claims with payouts over \$25,000 (nominal). We require claims with smaller payouts to meet criteria (1) and (2). The TDI data includes claim-level data only for claims with payouts over \$10,000 (nominal). For time consistency, we retain only claims with payouts of at least \$10,000 in 1988 dollars (the first data year). The threshold for reporting individual claims rises from \$10,000 to \$25,000 for claims closed after September 1, 2009. The data set excludes Veterans Administration hospitals, self-insured hospitals, including the University of Texas hospital system, and physicians employed by these hospitals.

Figure 4.1: Injuries leading to paid medical malpractice claims in Florida by year of injury



Notes: Number of closed paid medical malpractice claims included in Florida data set, by injury year. Hollow bars show actual data. Drop in number of claims beginning with 2004 reflects two main factors: (1) the flow of new claims dropped after Florida adopted tort reforms, effective for suits filed after September 1, 2003; and (2) some claims resulting from injuries in those years are not yet closed (right-censoring of the closed claims data set). Adjustments for these factors are shown in light and dark shaded bars, respectively. See text for estimation procedure. Regression line shows estimated number of paid claims without tort reform or censoring, based on linear trend using data from 1994 to 2002: No. of claims =  $876.4 + 32.1 (\text{year} - 1994)$  [ $t = 2.32$ ].

To correct for right-censoring, due to some claims not having closed by the final data year, we use all claims in the state to estimate the probability  $1/p_{close,\ell}$ , that a medical malpractice claim closes by the end of the year that is  $\ell$  years after the date of injury. We then scale the number of claims in each hospital-year with injury year  $t$  by  $1/p_{close,(T-t)}$ , where  $T$  is the last year with claims data. This yields the number of claims we ultimately expect for year  $t$ , once all claims have closed. Since later years are more affected by censoring, we drop the last three years of claims data. Because of the limited span of the data on PSIs, we also drop claims with injuries before 1999 or after 2010. The dark shaded bars in Figure 4.1 show the additional paid claims we expect once all claims have closed.

To adjust for the effect of tort reform, we regress the number of claims, adjusted for right-censoring, on year during the pre-reform period. We then use the regression coefficient to predict the statewide number of claims that would be expected without reform in 2003 and later years. For these years, we multiply right-censoring-adjusted claims in each hospital-year by the statewide ratio of (expected claims without reform)/(observed claims), to obtain an estimated number of claims without reform. Figure 4.1 shows the linear trend line, and the additional claims that would be expected, in 2003 and later years, without tort reform, in light shaded bars. Our regression specifications include year dummies, which control for deviations from the assumed linear trend in claim rates. References below to number of malpractice claims are after these two adjustments.

The bottom panel of Table 4.2 provides summary statistics for Florida medical malpractice claims. The sample includes 219 distinct Florida hospitals and 2,484 hospital-years over 1999–2010. These hospitals experience 9,743 raw claims and 13,558 adjusted claims.



Table 4.2: Summary statistics for medical malpractice claims and PSI events in Florida

	Total events	Cases at risk	Rate per 10,000 cases at risk	Hospital level rates		
				Mean	Std. dev.	Min Max
PSI 2: Death in Low-mortality DRGs	2,304	5,696,239	4.04	0.93	1.31	0 11
PSI 3: Pressure Ulcer	173,269	7,480,457	231.63	69.75	64.57	0 498
PSI 4: Death of Surgical Inpatients with Serious Treatable Complications	27,231	196,417	1386.39	10.96	13.56	0 112
PSI 5: Foreign Body Left during Procedure	1,908	24,464,728	0.78	0.77	1.27	0 9
PSI 6: Iatrogenic Pneumothorax	11,501	20,375,598	5.64	4.63	5.74	0 41
PSI 7: Central Venous Catheter-related Bloodstream Infection	28,378	16,398,883	17.3	11.42	14.44	0 136
PSI 8: Postoperative Hip Fracture	1,080	4,136,173	2.61	0.43	0.87	0 7
PSI 9: Postoperative Hemorrhage or Hematoma	14,801	6,200,382	23.87	5.96	8.38	0 75
PSI 10: Postoperative Physiologic and Metabolic Derangement	2,583	3,172,316	8.14	1.04	2.13	0 40
PSI 11: Postoperative Respiratory Failure	28,245	2,508,147	112.61	11.37	17.54	0 189
PSI 12: Postoperative Pulmonary Embolism or Deep Vein Thrombosis	62,361	6,212,002	100.39	25.11	36.24	0 506
PSI 13: Postoperative Sepsis	10,781	783,945	137.52	4.34	6.92	0 108
PSI 14: Postoperative Wound Dehiscence	1,955	1,128,156	17.33	0.79	1.23	0 8
PSI 15: Accidental Puncture or Laceration	58,207	21,241,890	27.4	23.43	33.25	0 292
PSI 17: Birth Trauma - Injury to Neonate	5,097	2,433,563	20.94	2.05	5.32	0 71
PSI 18: Obstetric Trauma - Vaginal Delivery with Instrument	16,137	117,435	1,374.12	6.5	13.68	0 206
PSI 19: Obstetric Trauma - Vaginal Delivery without Instrument	39,592	1,535,639	257.82	15.94	28.47	0 319
Total PSI events	485,430	124.1 mil.	39.12	195.42	193.57	0 1444
Hospital Discharges	28.5 mil.			11,458	9,761	3 55,978
Medical malpractice claims	9,743			3.92	5.3	0 54
Adjusted medical malpractice claims/hospital-year	13558			5.46	7.12	0 54

Table shows, for 1999-2010, total statewide PSI events, cases at risk for each PSI, and PSI rate per 10,000 cases at risk; and hospital-level mean, standard deviation, minimum, and maximum annual PSI rates. Hospital-level data is based on 2,484 hospital-years with positive discharges (219 distinct hospitals). Bottom rows show: hospital discharges, medical malpractice claims, and “adjusted medical malpractice claims,” using adjustments described in text to remove estimated effects of right-censoring of claims and Florida’s 2003 medical malpractice reform.

### 4.3.2 Patient safety data

To detect PSI events, we apply AHRQ’s definitions to the state inpatient data sets from Florida and Texas.<sup>10</sup> The Agency for Health Care Administration’s inpatient data set file covers all inpatient discharges in Florida. It is available for 1988–2013. This file contains a unique identifier for each discharge, the year and quarter of the discharge, hospital identifiers, patient demographic characteristics, and ICD-9-CM diagnosis and procedure codes. We merge the inpatient data to hospital-level medical malpractice data using the hospital IDs in both data sets.

AHRQ regularly updates the PSI definitions to reflect changes in the ICD-9-CM diagnosis and procedure codes. New ICD-9-CM codes are often more specific than older codes. Unfortunately, switching to newer and often-narrower codes, as AHRQ does, produces time-inconsistent measures of PSI rates. We therefore modify the AHRQ definitions of PSIs to generate measures that are closer to being time-consistent over our study period. All PSI counts use our modified definitions. We have time-consistent definitions over 1999 to 2010, and therefore limit the inpatient data to this period. Our data include roughly 29 million discharges between 1999 and 2010.

We use 17 PSIs (all but PSI-16, which is too infrequent to be usable), which we describe in Table 1, and list in Table 2 along with summary statistics on the number of PSI events, the number of cases at risk for each PSI, and the PSI rate (per 10,000 cases at risk). There are roughly 500,000 total PSI events in Florida. PSI rates exhibit substantial variation across hospitals, suggesting that hospitals can improve their PSI rates at manageable cost, because some hospitals are achieving lower rates.

Figure 4.2 provides evidence on this variation. We compute annual rates per 1,000 cases at risk for each PSI for each hospital during the sample period, normalize the PSI-specific rates, and sum these normalized rates for each hospital to compute an overall pooled hospital

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<sup>10</sup>For details on the Texas inpatient data set, see Appendix, C.3.2.

PSI rate:

$$\text{PooledPSIRate}_{it} = \sum_{j \in \text{PSI}} \text{norm}(\text{PSIjrate}_{it}) \quad (4.1)$$

$\text{PSIjrate}_{it}$  is the number of PSI- $j$  events divided by the number of cases at risk for PSI- $j$  at hospital  $i$  in year  $t$ ;  $\text{norm}()$  is a function that converts its argument to mean = 0 and standard deviation = 1 over all hospital-years. To generate Figure 4.2, we limit the sample to hospital-years with cases at risk for all of the PSIs, divide hospitals into quintiles based on average number of discharges over our sample period, and show box and whisker plots of time-averaged rates for each hospital  $[\text{mean}_t(\text{PooledPSIRate}_{it})]$ .

Larger hospitals tend to have higher PSI rates. This could reflect major “tertiary” hospitals tending to have sicker patients, who need more complex treatment or are more fragile, leading to more adverse events.<sup>11</sup> However, there is also substantial scatter in PSI rates, both within and across quintiles. For example, the top whisker in each quintile is well above the 75th percentile in most other quintiles.<sup>12</sup> Variance decomposition analysis indicates that 86 percent of the variance in PSI rates occurs within discharge quintiles; only 14 percent is across quintiles.<sup>13</sup> There is also sufficient variation in PSI rates within hospitals across time, to make feasible our core hospital FE analysis.

### 4.3.3 Covariates

Patient mix should be reasonably stable, and hence largely captured by our hospital FEs. To address remaining variation in patient mix, we include patient demographic characteristics and comorbidity counts as covariates in our regressions. We use the inpatient data to

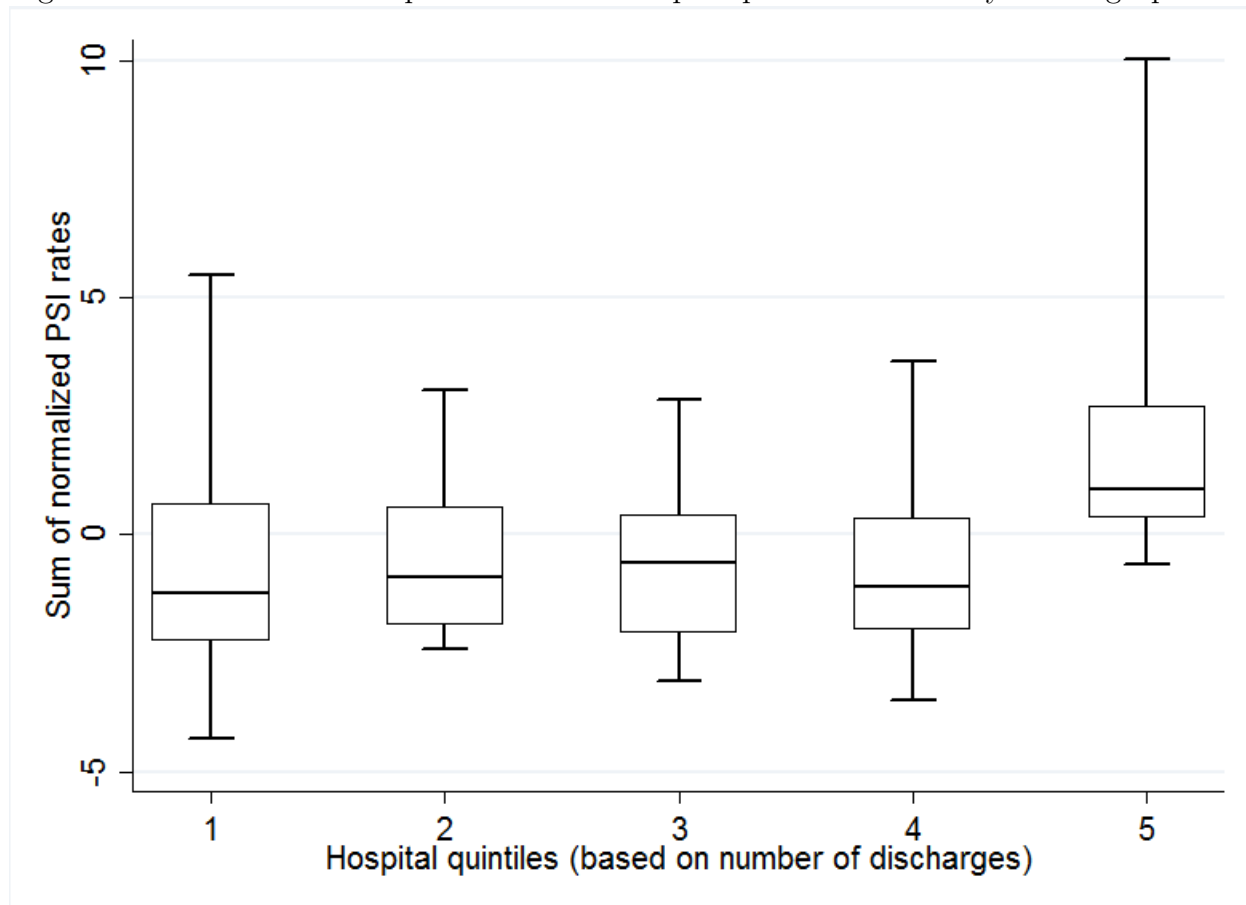
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<sup>11</sup>Case-mix differs substantially between rural and urban hospitals (VanBibber, Zuckerman, and Finlayson 2006).

<sup>12</sup>Two caveats for the outliers in the box and whiskers plots. First, “outlier” hospitals with low PSI rates could either be doing a good job of preventing adverse events, or a poor job of documenting adverse events. Also, some hospitals, especially smaller ones, may specialize in lower-risk services, leading to lower PSI rates.

<sup>13</sup>We decompose the variance of pooled hospital PSI rates  $V_{tot} = V_{disch} + V_{err}$ , where  $V_{err}$  is the variance of the residual from an OLS regression of the pooled, time-averaged hospital PSI rates on discharge quintile dummies. The within-quintiles fraction of the total variance is  $V_{err}/V_{tot}$ .

Figure 4.2: Box and whiskers plots for Florida hospital pooled PSI rates by discharge quintiles



Notes: Box and whiskers plots of hospital pooled PSI rates for hospital size quintiles, based on mean discharges over 1999–2010, for 133 hospitals (1,330 hospital-years) with cases at risk for each PSI. Pooled PSI rate is mean (across years) for hospital  $k$  of  $\sum_{j \in \text{PSI}} \{\text{norm}(\text{PSI}_j \text{ rate per 1,000 cases at risk})\}_{kt}$ , with PSI rates normalized to mean = 0 and standard deviation = 1. Boxes give 25th, 50th, and 75th percentiles; whiskers give 2.5th and 97.5th percentiles.

construct these covariates. The demographic variables we use are the fraction of patients, discharged from each hospital, who are female, white, Hispanic, aged 0-4 years, the excluded category 5-19 years, 20-34 years, 35-49 years, 50-64 years, 65-84 years, and  $\geq 85$ .

We also control for patient health using each of the comorbidities that enter the widely used Charlson comorbidity index (Charlson et al. 1987; Quan et al. 2005). The Charlson comorbidity index is based on 17 broad diagnostic categories that predict patient mortality: myocardial infarction; congestive heart failure; peripheral vascular disease; cerebrovascular disease; dementia; chronic pulmonary disease; rheumatic disease; peptic ulcer disease; mild liver disease; moderate or severe liver disease; diabetes without chronic complication; diabetes with chronic complication; renal disease; hemiplegia or paraplegia; any malignancy, including lymphoma and leukemia, except malignant neoplasm of skin; metastatic solid tumor; and AIDS/HIV.<sup>14</sup> We include each as a separate covariate.

To address the concern that larger tertiary hospitals may have sicker patients in ways not captured by the Charlson comorbidities, we include the natural logarithm of total discharges as a covariate.

## 4.4 Empirical strategy

To investigate the relationship between medical malpractice claim rates and PSI rates, we adopt a two-step approach. In the first step, we estimate residual PSI rates and residual medical malpractice claim rates. For PSIs, we estimate the difference between observed PSI events in each hospital-year and the *expected* number of events, given location (hospital for Florida, county for Texas) and patient characteristics, and use this difference to compute a residual PSI rate. We adopt a similar approach for medical malpractice claims. In the

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<sup>14</sup>The mapping from the broad Charlson categories to specific ICD-9-CM diagnoses in inpatient data sets changes over time as ICD-9-CM changes; different researchers have also developed somewhat different mappings. We used Stata's "Charlson" command to identify cases with each of these comorbidities.

second step, we apply standard panel data methods, in which we regress the residual medical malpractice claim rate on the residual PSI rate and assess the association between the two. We explain below why we choose this approach rather than directly regressing medical malpractice claim rates on PSI rates and covariates.

#### 4.4.1 Residual patient safety and medical malpractice measures

To compute the residual PSI measures, we first estimate expected PSI events based on the number of inpatient cases at risk for each PSI in each hospital-year, and the covariates discussed above. If hospital  $i$  has more (fewer) PSI events of type  $j$  than expected, given its size and case mix, it will have a positive (negative) residual for PSI- $j$ . Specifically, for each PSI- $j$ , we estimate the following, using ordinary least squares (OLS):

$$\ln(1 + \text{PSI}_{jit}) = \alpha + \beta_{\text{CAR}j} \ln(1 + \text{CAR}_{jit}) + \beta_D \ln(\text{Discharges}_{it}) + \beta_X \mathbf{X}_i + \left[ \sum_{M \in \text{Charlson}} \beta_{\text{Ch}M} \ln(1 + \text{Charlson}M_{it}) \right] + \text{PSI}j\text{Res}_{it} \quad (4.2)$$

where  $\text{PSI}_{jit}$  is the number of events for PSI type  $j$ , in hospital  $i$  and year  $t$ ,  $\text{CAR}_{jit}$  is the number of cases at risk for PSI- $j$ ,  $\text{Discharges}_{it}$  is the number of hospital discharges,  $\text{Charlson}M_{it}$  is the number of cases with Charlson comorbidity  $M$ , the  $\mathbf{X}_i$  are patient demographic variables (aggregated to the hospital level), and  $\text{PSI}j\text{Res}_{it}$  is the regression residual. We add one in the logarithms in order not to lose county-years with zero PSI events, cases at risk, or Charlson comorbidities. By construction,  $\text{PSI}j\text{Res}_{it}$  is uncorrelated with each of the predictors. We present the first-stage results in Appendix C.C.2, Table C.1.

We normalize  $\text{PSI}j\text{Res}_{it}$  for each PSI to mean zero and standard deviation one to make regression coefficients comparable across PSI types, given very different base frequencies for different PSIs. Let  $\text{norm}(X)$  be the normalized version of variable  $X$ . Our “PSI- $j$  measure”

is then  $\text{norm}(\text{PSI}_j \text{Res}_{it})$ .

We also construct a pooled measure of adverse events by summing the individual PSI measures. As noted above, we interpret the PSIs, not as indicating specific events that lead to medical malpractice claims, but instead as proxies for overall patient safety. Our hypothesis is that lower overall safety predicts higher medical malpractice claim rates. Given this use of the PSIs, it seems reasonable to weight each measure equally in constructing the pooled measure.<sup>15</sup> Our “pooled PSI measure” is the re-normalized sum of the PSI- $j$  measures:

$$\text{PooledPSIMeasure}_{it} = \text{norm} \left\{ \sum_{j \in \text{PSI}} \text{norm}(\text{PSI}_j \text{Res}_{it}) \right\} \quad (4.3)$$

We use a similar approach to measure residual medical malpractice risk. Our measure is the number of closed paid claims in each hospital-year, relative to the number we would expect given the number of cases at risk for each PSI and the same covariates that we use to predict PSIs. We use a flexible specification in which the number of cases at risk for each PSI can separately predict expected medical malpractice claims:

$$\begin{aligned} \ln(1 + \text{MedMal}_{it}) = & \alpha + \left[ \sum_{j \in \text{PSI}} \beta_{\text{CAR}_j} \ln(1 + \text{CAR}_{j_{it}}) \right] + \beta_D \ln(\text{Discharges}_{it}) + \\ & \beta_X \mathbf{X}_i + \left[ \sum_{M \in \text{Charlson}} \beta_{\text{Ch}_M} \ln(1 + \text{Charlson}_{M_{it}}) \right] + \text{MedMalRes}_{it} \end{aligned} \quad (4.4)$$

Here  $\text{MedMal}_{it}$  is the number of medical malpractice claims paid by hospital  $i$  for injuries that occurred in year  $t$ . Other variables are defined above. A positive (negative) residual indicates that there were more (fewer) medical malpractice claims than expected. One can see the number of discharges as the primary measure of medical activity that generates medical mal-

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<sup>15</sup>As a robustness check, instead of summing the normalized PSI measures, which gives equal weight to each PSI, we use principal component analysis, which weights the individual PSI measures so that the first principal component explains the greatest possible amount of the variance in the individual measures, and use this principal component as a pooled patient safety measure, with similar results. We discuss the principal component analysis below.

practice risk, while PSI cases at risk and our covariates control for time variation in patient mix. We normalize medical malpractice risk measure to mean zero and standard deviation one across all sample years so our “medical malpractice measure” is  $\text{norm}(\text{MedMalRes}_{it})$ . We provide the first-stage regressions in Appendix C.C.2, Table C.2.

As discussed above, we treat the individual PSIs as imperfect proxies for overall hospital safety. Table 4.3 lists hospital-year-level correlations among the individual PSI measures. Most correlations are positive and statistically significant. A high rate for one PSI predicts high rates for other PSIs, including PSIs that are not directly related and occur in different parts of a hospital.

These positive correlations provide evidence that the PSIs include a common core, which we term “overall patient safety.” One way to confirm this common core is to apply principal component analysis. The loadings on all the PSIs for the first principal component are positive and similar in magnitude, ranging from 0.165 to 0.332 for all PSIs except PSI-3 (loading = 0.075). The second and higher principal components have no obvious interpretation. The positive correlations also suggest that a pooled measure that combines the PSIs will provide a better proxy for overall patient safety than any individual PSI.

#### 4.4.2 Regression Specifications

We test whether adverse events predict medical malpractice claim rates using three panel data specifications: pooled OLS, hospital random effects (RE), and hospital fixed effects (FE). We rely principally on the hospital FE specification, but obtain similar results with all three approaches. Our pooled OLS regression specification is

$$\text{norm}(\text{MedMalRes}_{it}) = \alpha + \delta_t + \gamma \text{PooledPSIMeasure}_{it} + \varepsilon_{it} \quad (4.5)$$



Table 4.3: Correlations among individual PSI measures in Florida hospitals

PSI	2	3	4	5	6	7	8	9	10	11	12	13	14	15	17	18
3	0.099**	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
4	0.179**	0.062**	.	.	.	.	.	.	.	.	.	.	.	.	.	.
5	0.102**	0.068**	0.184**	.	.	.	.	.	.	.	.	.	.	.	.	.
6	0.143**	0.099**	0.270**	0.207**	.	.	.	.	.	.	.	.	.	.	.	.
7	0.143**	0.316**	0.177**	0.167**	0.244**	.	.	.	.	.	.	.	.	.	.	.
8	0.115**	0.064**	0.155**	0.132**	0.197**	0.110**	.	.	.	.	.	.	.	.	.	.
9	0.095**	0.090**	0.278**	0.264**	0.337**	0.267**	0.184**	.	.	.	.	.	.	.	.	.
10	0.126**	0.096**	0.293**	0.219**	0.226**	0.213**	0.164**	0.280**	.	.	.	.	.	.	.	.
11	0.124**	0.132**	0.352**	0.215**	0.244**	0.226**	0.123**	0.285**	0.405**	.	.	.	.	.	.	.
12	0.105**	0.140**	0.202**	0.223**	0.243**	0.303**	0.148**	0.375**	0.210**	0.305**	.	.	.	.	.	.
13	0.086**	0.097**	0.303**	0.222**	0.275**	0.260**	0.111**	0.292**	0.380**	0.523**	0.285**	.	.	.	.	.
14	0.094**	0.081**	0.155**	0.147**	0.171**	0.102**	0.114**	0.219**	0.179**	0.168**	0.172**	0.143**	.	.	.	.
15	0.091**	0.091**	0.199**	0.244**	0.338**	0.241**	0.195**	0.447**	0.214**	0.178**	0.303**	0.212**	0.178**	.	.	.
17	0.130**	0.059**	0.168**	0.145**	0.224**	0.175**	0.143**	0.218**	0.183**	0.149**	0.093**	0.177**	0.152**	0.184**	.	.
18	0.056**	0.055**	0.098**	0.124**	0.116**	0.162**	0.095**	0.134**	0.142**	0.080**	0.090**	0.096**	0.127**	0.158**	0.292**	.
19	0.081**	0.029	0.127**	0.130**	0.158**	0.174**	0.133**	0.176**	0.168**	0.131**	0.177**	0.129**	0.109**	0.135**	0.355**	0.589**

Correlations among individual PSI measures. PSI measures are defined in the text. Hospitals are indexed by  $i$ , PSIs by  $j$ , Charlson comorbidities by  $m$ , years by  $t$ . Sample is 2,484 hospital-year observations over 1999-2010 with positive discharges, for 219 hospitals. Significance level: \* 5%, \*\* 1%.

Here  $\text{norm}(\text{MedMalRes}_{it})$  is our medical malpractice measure and  $\text{PooledPSIMeasure}_{it}$  is the pooled PSI measure, both described above;  $\delta_t$  are year dummies and  $\varepsilon_{it}$  is the error. The parameter of principal interest is  $\gamma$ , which will be positive if PSI rates predict medical malpractice claim rates.

The pooled OLS specification will be biased if unobserved hospital-level factors correlate with both medical malpractice activity and PSI rates. We partly address this possibility by controlling for hospital size, measured by  $\ln(\text{Discharges})$ , and other covariates, when we estimate residual PSI and malpractice claim rates. We further address the risk of bias by adding hospital effects, using RE and FE specifications. We estimate the following model, where  $u_i$  is the hospital effect

$$\text{norm}(\text{MedMalRes}_{it}) = \alpha + \delta_t + \gamma \text{PooledPSIMeasure}_{it} + u_i + e_{it} \quad (4.6)$$

The pooled OLS and RE models make a “strict exogeneity” assumption; one form of this assumption is that the hospital effects are independent of other regressors in all time periods,  $\text{Cov}(u_i, x_{i,t}) = 0 \forall t$ . The FE model is consistent even if the hospital effects are correlated with other regressors, but has weaker power to detect an association between PSI rates and medical malpractice claims rates because it uses only within-hospital variation over time. In all regressions, we cluster standard errors on hospital to address potential within-hospital correlation of the errors.

Our two-stage approach has several advantages over a one-stage regression, with claims (or claim rates) as the dependent variable, PSIs (or PSI rates) as independent variables, and patient and hospital characteristics as covariates. First, PSIs vary greatly in frequency (see Table 4.2). The two-stage approach lets us give the same weight to variation in a high-frequency PSI such as pressure ulcer (PSI-3), as to a low frequency but more serious PSI, such as foreign object left in body during surgical procedure (PSI-5). There is no obvious

way to give equal weight to variation in each PSI in a one-stage regression. Second, our approach lets the impact of covariates vary across PSIs. A one-stage regression would not readily allow this.<sup>16</sup> Note that if we use a single PSI to measure patient safety, one- and two-stage regressions will give the same results in pooled OLS and FE specifications (this is the Frisch-Waugh-Lovell Theorem; e.g., Hansen (2015; Theorem 3.15.1)).

### 4.4.3 Childbirth regression specifications

For the most part, we cannot link specific types of PSIs to similar types of medical malpractice claims. Childbirth is a partial exception, which allows us to test whether variation in the childbirth-specific PSIs predicts variation in childbirth-related malpractice claims. The Florida data do not allow us to further separate mother claims from baby claims.<sup>17</sup> We identify 1,695 birth-related claims over 1994–2014.

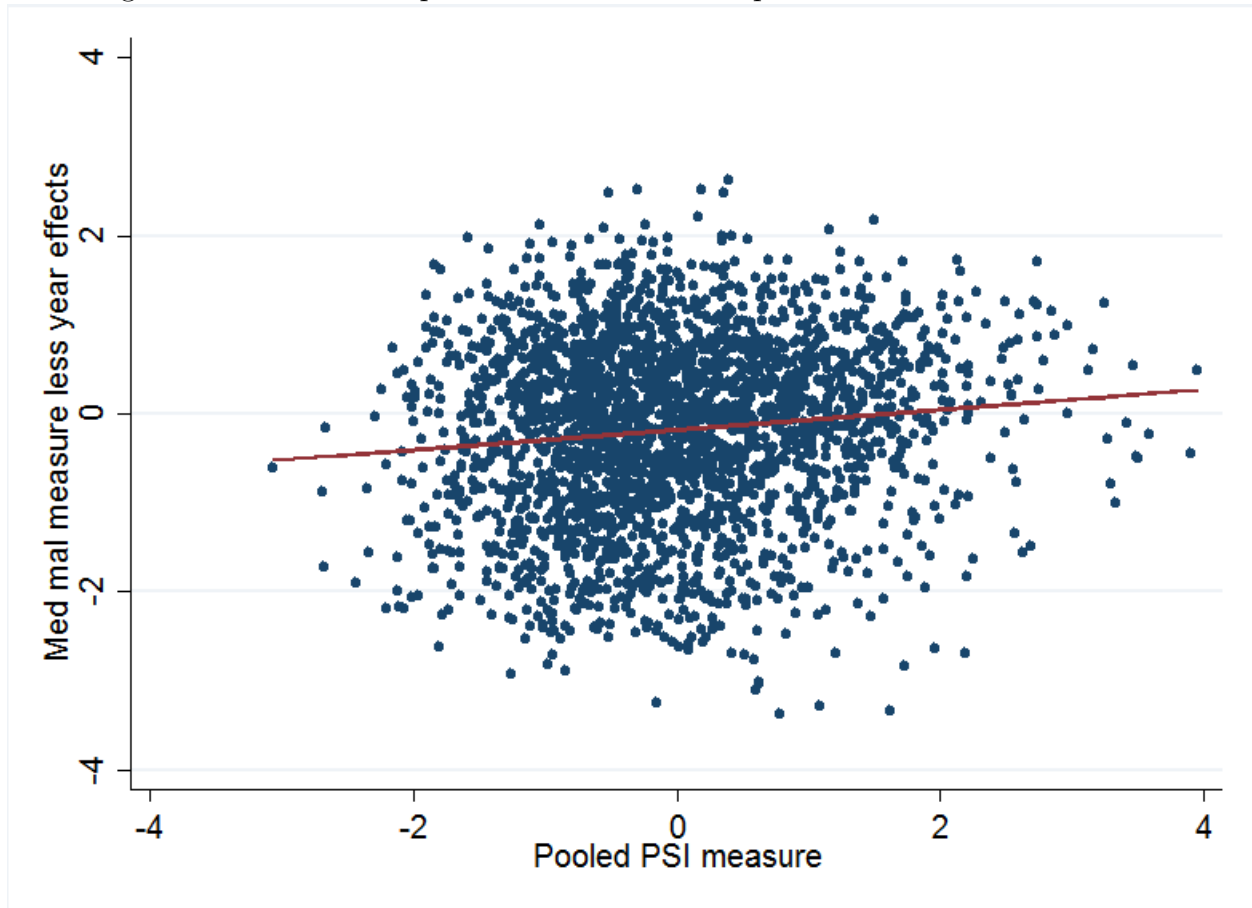
We test whether a residual measure of childbirth claims (constructed similarly to our overall malpractice claims measure) is predicted by (1) the PSI measures for each of PSI-17 (birth injury to neonate), PSI-18 (injury to mother for vaginal delivery with instrument), and PSI-19 (injury to mother for vaginal delivery without instrument); and (2) a pooled measure for all three birth PSIs. We construct the pooled birth PSI measure in the same way as the overall pooled PSI measure except that we sum only over the measures for PSIs 17–19.

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<sup>16</sup>We have too few hospital-year observations (2,484) to make feasible a one-stage model in which we interact each covariate with each of the PSI dummies.

<sup>17</sup>To find birth-related claims, we begin with free text fields in the database for final diagnosis, misdiagnosis, cause of injury, and principal injury. We parse the text for terms such as “neonate” and “labor.” The full list of terms was the following: neonate, newborn, new born, infant, baby, birth, stillborn, still born, fetus, fetal, delivery, C-section, cesarean section, labor, NICU, utero.

Figure 4.3: Medical malpractice measure versus pooled PSI measure in Florida



Notes: Scatter plots and regression lines for medical malpractice measure versus pooled PSI measure, after partialling out year dummies. Medical malpractice and pooled PSI measures are defined in the text. Sample is 2,484 hospital-years over 1999–2010 with positive discharges. Linear trend:  $y = -0.180 + 0.113x$  ( $t = 3.33$ ), with standard errors clustered on hospital.

## 4.5 Results for Florida

### 4.5.1 Results with pooled PSI measure

We find a strong, positive relationship between the pooled PSI measure and the malpractice measure (coefficient = 0.113,  $t = 3.33$ ) in a graphical analysis (see Figure 4.3).

We also find a strong positive relationship between the pooled PSI measure and the malpractice claims measure in regression analyses (see Table 4.4). This relationship is consistent across pooled OLS, hospital random-effects, and hospital fixed-effects specifications.

Table 4.4: Regressions of medical malpractice measure on pooled PSI measure: Florida

Dependent variable	Medical malpractice measure		
Sample period	1999-2010	1999-2009	1999-2010
Use adjusted no. of claims	Y	Y	N
<i>Panel A: Pooled OLS</i>			
Pooled PSI measure coefficient	0.113***	0.108***	0.124***
(t-statistic)	(3.33)	(3.04)	(3.52)
$R^2$	0.0200	0.0200	0.0269
<i>Panel B: Hospital random effects</i>			
Pooled PSI measure coefficient	0.129***	0.119***	0.149***
(t-statistic)	(4.37)	(4.12)	(4.95)
Random effects $\lambda$	0.549	0.539	0.562
Between $R^2$	0.0211	0.0200	0.0199
Within $R^2$	0.0200	0.0203	0.0291
Overall $R^2$	0.0199	0.0199	0.0266
<i>Panel C: Hospital fixed effects</i>			
Pooled PSI measure coefficient	0.141***	0.129***	0.168***
(t-statistic)	(3.63)	(3.40)	(4.31)
Within $R^2$	0.0200	0.0204	0.0293
CRE t-statistic	-0.48	-0.31	-0.78

Pooled OLS, hospital random effects (RE) and hospital fixed effects (FE) regressions for Florida over indicated periods of medical malpractice measure on pooled PSI measure, year dummies, and constant term. Medical malpractice and pooled PSI measures are defined in the text. In columns 1 and 2, the number of claims is adjusted to remove estimated impact of medical malpractice reform and still-open claims; see text for details. Last row shows t-statistics from correlated random effects test for difference between RE and FE coefficients. Sample over 1999-2010 is 2,484 hospital-year observations of 219 hospitals. t-statistics, with standard errors clustered on hospital, in parentheses. Significance level: \* 10%, \*\* 5%, \*\*\* 1%.

Using the full sample period, 1999–2010, we find a coefficient of 0.141 ( $t = 3.63$ ) in the hospital FE specification (see Table 4.4, panel C, column 1). A correlated random-effects (CRE) test for equality of coefficients between RE and FE (Wooldridge 2013, §14.3) produces a t-statistic of only 0.48. This suggests that hospital effects are not important, and that the statistically stronger RE results (coefficient = 0.129;  $t = 4.37$ ) are likely to be reliable.<sup>18</sup>

<sup>18</sup>The CRE model adds the time mean of the pooled PSI measure to the RE model in equation 4.6. The t-statistic for this variable tests whether RE and FE coefficients are different. The CRE test has several advantages over the more familiar Hausman test: (1) one can use clustered standard errors; (2) one can

We present robustness checks in Table 4.4, columns 2 and 3. In column 2, we drop 2010, the last year of the sample period. This year has the noisiest measure of malpractice claims because a small fraction of claims with injuries in 2010 closed by the end of our data period, so 2010 has the fewest number of closed claims. In column 3, we use the raw number of paid malpractice claims to construct the malpractice measure, rather than the adjusted number of claims. This makes the claim rate less time-consistent, but year dummies should largely capture the effect of the adjustments, and the adjustments could introduce noise. The FE coefficient on the pooled PSI measure rises to 0.168 ( $t = 4.31$ ).

We can use the coefficients from Table 4.4 to estimate the effect of increased safety (proxied by fewer PSI events) on the malpractice claim rates – in effect, an elasticity of paid claims with regard to PSI events. Appendix C.1 provides details on how we compute this elasticity. Our hospital FE results, from Table 4.4, panel C, column 1, imply that a one standard deviation reduction in each PSI rate would decrease paid medical malpractice claims by 16.2 percent. Thus, if our results are causal, achievable improvements in patient safety could significantly reduce the malpractice claim risk that hospitals face, in addition to their direct benefits for patients.

### 4.5.2 Results with individual PSI measures

As a further robustness check, which can address the concern that our results are driven by how we pool the PSIs, we repeat the regressions presented in Table 4.4 using each of the individual PSI measures, one at a time, instead of the pooled PSI measure (see Table 4.5). Across specifications, almost all of the coefficients are positive. All 17 PSI measures take positive coefficients with pooled OLS, and 16 of the 17 take positive coefficients with hospital RE or hospital FE. None of the negative coefficients are significantly different from

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test for different FE and RE coefficients both for a specific variable of interest and for multiple regression coefficients together (the Hausman test applies only to all variables together); (3) in our experience in other studies, the Hausman test often fails to run.

zero. Of the positive coefficients, eight are statistically significant with pooled OLS (at the 5 percent level or better), six are significant with hospital RE, and four are significant with hospital FE.

Table 4.5: Regressions of malpractice measure on individual PSI measures: Florida

Dependent variable Regression model	Medical malpractice measure		
	OLS	Hosp. RE	Hosp. FE
PSI 2: Death in Low-mortality DRGs	0.110*** (4.48)	0.052** (2.52)	0.029 (1.39)
PSI 3: Pressure Ulcer	0.019 (0.78)	0.022 (1.01)	0.027 (1.11)
PSI 4: Death of Surgical Inpatients with Serious Treatable Complications	0.079*** (2.75)	0.058** (2.40)	0.048* (1.83)
PSI 5: Foreign Body Left during Procedure	0.038 (1.49)	0.023 (1.19)	0.017 (0.85)
PSI 6: Iatrogenic Pneumothorax	0.106*** (3.84)	0.097*** (4.08)	0.091*** (3.42)
PSI 7: Central Venous Catheter-related Bloodstream Infection	0.080*** (2.70)	0.080*** (3.32)	0.079*** (3.03)
PSI 8: Postoperative Hip Fracture	0.064*** (2.94)	0.022 (1.10)	0.005 (0.26)
PSI 9: Postoperative Hemorrhage or Hematoma	0.01 (0.34)	0.009 (0.32)	0.006 (0.19)
PSI 10: Postoperative Physiologic and Metabolic Derangement	0.064** (2.04)	0.063** (2.43)	0.062** (2.21)
PSI 11: Postoperative Respiratory Failure	0.038 (1.21)	0.033 (1.31)	0.028 (0.98)
PSI 12: Postoperative Pulmonary Embolism or Deep Vein Thrombosis	0.042 (1.35)	0.044* (1.77)	0.046 (1.62)
PSI 13: Postoperative Sepsis	0.019 (0.65)	-0.019 (-0.76)	-0.043 (-1.53)
PSI 14: Postoperative Wound Dehiscence	0.037 (1.54)	0.022 (1.08)	0.017 (0.77)
PSI 15: Accidental Puncture or Laceration	0.024 (0.73)	0.023 (0.84)	0.023 (0.77)
PSI 17: Birth Trauma - Injury to Neonate	0.069** (2.12)	0.048* (1.83)	0.037 (1.28)
PSI 18: Obstetric Trauma - Vaginal Delivery with Instrument	0.051 (1.55)	0.049* (1.90)	0.053* (1.95)
PSI 19: Obstetric Trauma - Vaginal Delivery without Instrument	0.073** (2.03)	0.070** (2.03)	0.069** (1.98)

Each cell is from a separate regression of the medical malpractice measure on the indicated PSI measure. Medical malpractice and PSI measures are defined in the text. Sample is 2,484 hospital-years (219 hospitals) over 1999-2010 with positive discharges. t-statistics, with standard errors clustered on hospital, in parentheses. Significance level: \* 10%, \*\* 5%, \*\*\* 1%.



These results further support an association between overall patient safety and malpractice risk, with the individual PSIs serving as imperfect proxies for patient safety. The consistent results across individual PSIs imply that the results for the pooled PSI measure, presented in Table 4.4, are not sensitive to how we construct the pooled measure.

The coefficients for individual PSIs are generally lower in magnitude than for the pooled PSI measure. This is expected, since individual PSI measures are noisier proxies for overall patient safety, and measurement error in an independent variable biases coefficients toward zero (e.g., Wooldridge 2013, ch. 9).

### 4.5.3 Results with alternative PSI pooling methods

We further confirm that our results for the pooled PSI measure are not sensitive to how we construct this measure, by using two alternative pooling approaches. First, we include all 17 individual PSI measures in a single regression, and sum the coefficients on these measures. The sum of the coefficients on the individual PSI measures is positive and statistically significant across specifications 0.266 ( $t = 3.80$ ) for pooled OLS, 0.274 ( $z = 4.43$ ) with hospital RE, and 0.301 ( $t = 3.74$ ) with hospital FE.

Second, we conduct principal components analysis, in which we replace the pooled PSI measure with the normalized first principal component of the individual PSI measures. The coefficients on the first principal component are close to those reported in Table 4.4. For example, the hospital FE coefficient is 0.137 ( $t = 3.46$ ), versus the 0.141 coefficient reported in Table 4.4.

### 4.5.4 Birth claims

For malpractice claims related to childbirth, we are able to assess whether the birth-specific PSIs predict birth-related claim rates. We find that both the three individual birth-related

PSIs and a pooled PSI measure that combines all three of these PSIs take positive coefficients across pooled OLS, hospital RE, and hospital FE specifications (see Table 4.6). All coefficients are statistically significant with pooled OLS and hospital RE. With hospital FE, the coefficients for PSI-19 and for the pooled birth PSIs (17–19) are statistically significant. The coefficient on the pooled birth PSI measure with hospital FE is 0.110 ( $t = 2.72$ ); this is comparable in magnitude to the 0.141 coefficient on the pooled PSI measure in Table 4.4, which includes all PSIs and all claims.

Table 4.6: Childbirth PSIs and childbirth injury claims: Florida

Dependent variable Regression model	Medical malpractice measure		
	OLS	Hosp. RE	Hosp. FE
PSI 17: Birth Trauma - Injury to Neonate	0.105*** (2.72)	0.080** (2.36)	0.051 (1.43)
PSI 18: Obstetric Trauma - Vaginal Delivery with Instrument	0.090** (2.46)	0.073** (2.46)	0.058* (1.89)
PSI 19: Obstetric Trauma - Vaginal Delivery without Instrument	0.129*** (3.30)	0.114*** (3.32)	0.093** (2.46)
Pooled PSIs 17-19	0.139*** (3.38)	0.127*** (3.52)	0.110*** (2.72)

Each cell is from a separate regression of birth (mother or baby) medical malpractice measure on the indicated PSI measures. Medical malpractice and PSI measures are defined in the text. Column (2) uses hospital RE; column (3) uses hospital FE. t-statistics, with standard errors clustered on hospital, in parentheses. Sample is same as in Table 4.4. Significance level: \* 10%, \*\* 5%, \*\*\* 1%.

## 4.6 Results for Texas

The Florida data provide hospital-level data on malpractice claims, and also let us link the rates of birth claims to birth-specific PSIs. But relying only on Florida data creates the risk that our results may be driven by something specific to Florida. We therefore conduct a similar analysis using malpractice claims data from Texas, the only other state with publicly available data. In Texas, we have claims data only at the county level. Hence the strongest available design relies on county FE; we cannot use the hospital FE that were available in

Florida. Our estimation strategy is otherwise similar to Florida.<sup>19</sup> We present limited results for Texas below, and additional results, comparable to those for Florida, in Appendix C.3.

#### 4.6.1 Overall Texas results

Our Texas results are statistically strong, and the coefficients on the pooled PSI measure are quite similar to the Florida results. For example, the coefficient on the pooled PSI measure in Texas with county FE is 0.130 ( $t = 2.76$ ; see Table 4.7), compared with a coefficient of 0.141 in Florida with hospital FE. As in Florida, results are similar if we drop the last sample year, and are stronger if we use the raw rather than the adjusted number of claims. The county FE results imply that a one standard deviation reduction in each PSI rate (with standard deviations measured at the hospital-year level) predicts a 16.8 percent drop in paid malpractice claims.

The pooled OLS and county RE coefficients are larger than the county FE coefficients (unlike Florida, where all coefficients were similar), and the CRE test mildly rejects equality of coefficients ( $t = 1.84$ ). The differences in estimates across methods could reflect the cruder nature of the county-level Texas analysis, versus the hospital-level Florida analysis.

#### 4.6.2 Birth claims

In Texas, we can identify “newborn” claims, based on age at the time of injury less than one month; most but not all of these will involve birth injury. We cannot identify mother claims due to injury during childbirth. We find that the PSI-17 measure (PSI-17 is birth injury to neonate) predicts a measure of residual newborn malpractice claims (see Appendix C.3.5,

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<sup>19</sup>In Texas, unlike Florida, we cannot identify which malpractice claims were due to injuries suffered while a hospital inpatient. Thus, our malpractice risk measure is based on all paid claims in a county-year. In Texas, we base demographic characteristics on county characteristics, obtained from the Census Bureau (see <http://www.tsl.state.tx.us/ref/abouttx/census.html>), rather than on the characteristics of patients in each hospital. Our covariates are per capita income and the fractions of the population: living in rural areas, over age 62, white, and Hispanic.

Table 4.7: Regressions of medical malpractice measure on pooled PSI measure: Texas

Dependent variable	Medical malpractice measure		
Sample period	1999-2009	1999-2008	1999-2009
Use adjusted no. of claims	Y	Y	N
<i>Panel A: Pooled OLS</i>			
Pooled PSI measure coefficient	0.225***	0.238***	0.266***
(t-statistic)	(5.71)	(5.69)	(6.44)
$R^2$	0.0598	0.0602	0.1026
<i>Panel B: Hospital random effects</i>			
Pooled PSI measure coefficient			
(t-statistic)	(5.81)	(5.64)	(6.73)
Random effects $\lambda$	0.198	0.211	0.357
Between $R^2$	0.2182	0.2255	0.2226
Within $R^2$	0.0198	0.0142	0.0602
Overall $R^2$	0.0598	0.0602	0.1022
<i>Panel C: Hospital fixed effects</i>			
Pooled PSI measure coefficient			
(t-statistic)	0.130***	0.141***	0.159***
	(2.76)	(2.66)	(3.48)
Within $R^2$	0.0210	0.0147	0.0626
CRE t-statistic	1.84*	1.96*	1.94*

Pooled OLS, county random effects (RE) and county fixed effects (FE) regressions for Texas over indicated periods of medical malpractice measure on pooled PSI measure, year dummies, and constant term. Medical malpractice measure and PSI measures are defined in the text. In regressions (1)-(2), the number of claims is adjusted to remove estimated impact of medical malpractice reform and still-open claims; see text for details. The last row shows t-statistics from correlated random effects test for difference between RE and FE coefficients. The sample over 1999-2009 is 1,205 county-year observations of 139 counties. t-statistics, with standard errors clustered on hospital, in parentheses. Significance level: \* 10%, \*\* 5%, \*\*\* 1%.

Table C.7, for the regression results). The coefficients on the PSI-17 measure are positive across pooled OLS, county RE, and county FE specifications, are statistically significant for the pooled OLS and county RE specifications, and are positive and marginally significant (coefficient = 0.072;  $t = 1.73$ ) with county FE.

## 4.7 Discussion

### 4.7.1 Overview

We find a strong positive association in Florida between adverse patient safety events in hospitals and the number of medical malpractice claims paid by these hospitals. Our results are both statistically strong and “economically” meaningful: a one standard deviation reduction in PSI rates predicts a 16.2 percent fall in paid malpractice claims.

The association that we find is likely to be causal. We lack an external shock to patient safety, and thus cannot implement a true causal design. However, we have a strong empirical specification, with hospital FE and extensive patient-level covariates covering both health and demographic characteristics. Variation within hospitals across time in patient safety events, proxied by the Patient Safety Indicators developed by AHRQ, strongly predicts variation within hospitals across time in the same hospitals in paid malpractice claims. Reverse causality cannot explain our results – it would predict the opposite (negative) sign on our PSI measures. Higher malpractice risk should induce greater safety efforts, and hence reduce PSI rates. Two recent studies exploit legal shocks to malpractice risk and find evidence consistent with this prediction (Iizuka 2013; Zabinski and Black 2015; although Frakes and Jena – forthcoming – find no significant effect). This evidence suggests that our results may *understate* the causal effect of patient safety on medical malpractice claims.

Omitted variable bias cannot be ruled out, but it is unclear what omitted variable(s) might be time varying (our hospital FE control for time-invariant omitted variables) and

unrelated to patient safety yet predict time variation in both patient safety and malpractice claims within hospitals – both overall and specifically for childbirth claims – in a way that could explain our results. Any such omitted variable cannot be specific to Florida, because we obtain similar results for Texas. We also obtain very similar coefficient estimates across pooled OLS, hospital RE, and hospital FE models, suggesting that hospital characteristics – for example, larger, tertiary hospitals often see sicker patients – are not important drivers of our results. We also include extensive covariates, including the 17 measures that enter the Charlson comorbidity index. This should help to remove the effect of variation in patient mix within hospitals over time.

#### **4.7.2 Variation in PSI rates across hospitals**

We find large variation in PSI rates across similar-sized hospitals. This suggests that many hospitals can reduce adverse patient safety events at reasonable cost, because their peers are doing so. Why then don't more hospitals devote more effort to this important task?

Here we can only speculate, but in the big picture, the financial incentives for hospitals to improve patient safety, including the incentives provided by malpractice liability, are weak. Mello et al. (2007) find that hospitals are largely insulated from the financial costs of patient injuries. Krupka, Sandberg, and Weeks (2012) report that hospitals earn higher revenue when surgical patients suffer complications than when they do not. For PSI-5 (foreign body left during procedure), O'Connor (2012) reports that only about 1 percent of hospitals have installed inexpensive sponge-tracking systems for surgeries, which could reduce PSI-5 rates to nearly zero. For PSI-7 (central line-associated bloodstream infections), Herzer et al. (2014) conduct a cost-benefit analysis of a multi-hospital central line-associated bloodstream infections reduction program and estimate hospital cost at roughly \$20,000 per infection prevented. This cost is outweighed by the large benefits to patients from these serious, often fatal, infections. However, these prevention programs are costly to hospitals, which

both incur safety costs and lose the extra revenue that they often earn from treating these infections.<sup>20</sup>

### **4.7.3 Limitations of our study**

#### **4.7.3.1 No exogenous shock to PSI rates**

We have no exogenous shock to PSI rates, or plausible instrumental variables for these rates, and thus cannot implement a true causal research design. We do benefit, however, from the damage caps adopted by Florida and Texas during our sample period. These reforms are not a direct shock to PSI rates, but do provide a shock to hospital incentives to limit PSIs.

#### **4.7.3.2 Imperfect measure of patient safety**

We rely on imperfect measures of both patient safety and malpractice claim rates. We see the PSIs as proxies – constructs – for unobserved underlying patient safety. Noise in these proxies can be seen as a form of measurement error, which will bias our estimated coefficients toward zero.

#### **4.7.3.3 Imperfect measure of malpractice risk**

Our medical malpractice claims data are also not ideal. We have data from only two states, albeit large and diverse ones. We have hospital-level data only in Florida. We obtain data on claims only when they close, which limits the available sample period. We also have data only on paid claims. Data on all claims that are brought, including unpaid claims, would provide a useful and somewhat different measure of malpractice risk.

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<sup>20</sup>We are not aware of similar cost-benefit analyses for other PSIs.

## 4.8 Conclusion

We study whether PSI rates predict paid medical malpractice claim rates in Florida and Texas. We find a strong correlation between the individual PSI measures for most PSIs. This suggests that the PSIs, especially when pooled, are a reasonable proxy for overall patient safety in hospitals. We then find evidence for Florida of a strong positive association between PSI rates and medical malpractice claim rates, with a strong empirical specification that includes hospital fixed effects and extensive patient-level covariates. We confirm that a similar association holds for Texas, with county fixed effects.

These associations are likely to be causal. They suggest that hospitals that invest in patient safety can significantly reduce malpractice claims – a one standard deviation drop in PSI rates predicts a 16 percent drop in malpractice claims.



# Chapter 5

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# Appendix A

## Appendix to Chapter 1

### A.1 Precise definition for “due to” each ACSCC

Table A.1: Criteria for an admission, emergency department visit, or urgent care visit being counted as due to a condition

<b>Chronic condition</b>	<b>Description of poor health outcomes</b>	<b>Sources of ICD9 codes for poor health outcomes</b>
Asthma or COPD <sup>1</sup>	Principal diagnosis code of asthma, principal diagnosis code of COPD, or principal diagnosis code of acute bronchitis and an additional diagnosis code of COPD. Must be no diagnosis codes for cystic fibrosis or other abnormalities of the respiratory system.	AHRQ PQI5 (COPD or asthma in older adults admission rate) numerator technical specifications
Diabetes	Principal diagnosis code of diabetes complication, principal diagnosis of uncontrolled diabetes, or procedure code for a lower extremity amputation <sup>2</sup> (Agency for Healthcare Quality and Research, 2015) without a diagnosis code to indicate that the amputation was due to trauma.	AHRQ PQI1 (diabetes short-term complication admission rate), PQI3 (diabetes long-term complication admission rate), PQI14 (uncontrolled diabetes admission rate), PQI16 (lower extremity amputation among patients with diabetes) numerator technical specifications.
Heart failure	Principal diagnosis of heart failure, arrhythmia (atrial fibrillation, ventricular tachycardia, ventricular fibrillations, bradyarrhythmia), ischemic stroke, thromboembolism (deep vein thrombosis, atrial embolism and thrombosis in the extremities, pulmonary embolism), hepatic congestion, pulmonary congestion.	AHRQ PQI 8 (heart failure admission rate) numerator technical specifications; Watson et al. (2000) (complications of heart failure); ICD-9-CM Handbook (for codes corresponding to complications)
Hypertension	Principal diagnosis of hypertension, principal diagnosis of new myocardial infarction, principal diagnosis of heart failure in a beneficiary with no previous diagnosis of heart failure, principal diagnosis of stroke, or principal diagnosis of renal disease in a person with no previous diagnosis of renal disease.	AHRQ PQI 7 (hypertension admission rate) numerator technical specifications; James et al. (2013) (complications of hypertension); AHRQ PQI 8 (heart failure admission rate) numerator technical specifications; codes for other complications from the ICD-9-CM Handbook

## A.2 Determining and confirming NPIs in the SK&A data

Many physicians in the raw SK&A data have no National Provider Identifier (NPI) recorded and a few physicians have different NPIs recorded in different years. I exploit the National Plan & Provider Enumeration System (NPES) NPI database to remedy this issue. The NPES database removes physician details over time. Additionally, physicians have the option of updating their details, such as their location, at any time. I use a compilation of historical versions of the NPES files archived by the National Bureau of Economic Research. If the physician has no NPI in the SK&A data, I use the physician's name and location to match records in the NPES data. If the physician has one or more NPIs recorded in the SK&A data, I use the listed NPI to match the physician to the NPES data, and then confirm that the physician's name in the SK&A data matches the physician's name in the NPES data. When I am confirming an NPI that is already in the SK&A data, I do not require the location in the SK&A data to match the location in the SK&A data, since location is not necessarily kept up to date in the NPES file. Where the NPI cannot be confirmed in the NPES file, or remains missing, I drop the physician from the sample.

## A.3 Which physicians integrate?

Physicians that integrate may differ from those who do not both on observables and on unobservables.

To examine the differences on observables I focus on physicians that are not integrated in 2007, since integration may change the characteristics of the physician. I then compare physicians who integrate at some point between 2008 and 2013 to those that do not using a logit model. My independent variables are an indicator for the physician being a Car-

diologist (recall the sample has only Cardiologists and generalists); the size of the site the physician practices at in 2007, as measured by the number of physicians practicing there; the physician's number of Medicare claims in the 5% sample in 2006; the distance to the nearest hospital from the physician's zip code centroid in miles; the average gross income per household in the physician's zip code in 2006 in 2015 dollars; the population density in the physician's ZIP code in 2000 in people per square miles (approximated by the ZCTA density); and the hospital capacity in the physician's hospital service area, as measured by the number of beds in general acute care hospitals in 2006 divided by the population in 2000. I take the natural logarithm of all variables except the Cardiology indicator. I cluster the standard errors at the HSA level. It is also worth noting that there is substantial variation on observables among integrating and non-integrating physicians, much more so than between integrating and non-integrating physicians.

Table A.2: Characteristics of integrating physicians relative to non-integrating physicians - logit regression results

	Marginal effect	Standard error
1(Cardiologist)	8.1***	(2.3)
<i>at 1(Cardiologist)=0</i>		
ln(No. Medicare claims)	-1.0**	(0.4)
ln(No. physicians at site)	2.8***	(0.8)
ln(HSA beds per capita)	2.3***	(0.9)
ln(Miles to nearest hospital)	-1.9**	(1.0)
ln(Adjusted gross income 2015\$ thou.)	5.5**	(2.2)
ln(Pop. density ppl/mi <sup>2</sup> )	-2.2***	(0.5)
<i>at 1(Cardiologist)=1</i>		
ln(No. Medicare claims)	-1.2**	(0.5)
ln(No. physicians at site)	3.3***	(0.9)
ln(HSA beds per capita)	2.7***	(1.0)
ln(Miles to nearest hospital)	-2.3**	(1.1)
ln(Adjusted gross income 2015\$ thou.)	6.5**	(2.6)
ln(Pop. density ppl/mi <sup>2</sup> )	-2.6***	(0.7)

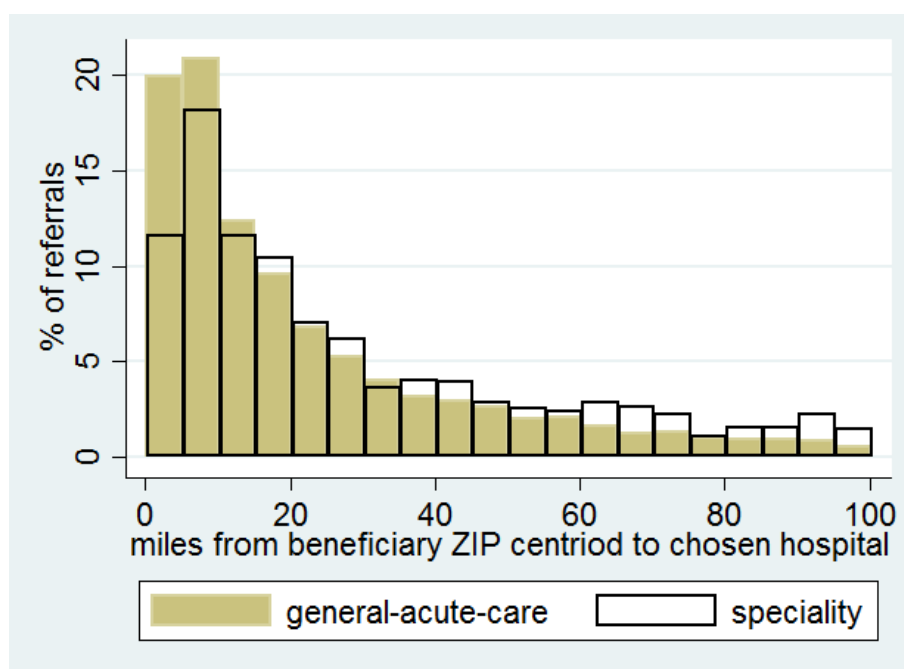
Marginal effects are at the means of all variables except 1(Cardiologist). All marginal effects and standard errors displayed above have been multiplied by 100 so they can be interpreted in terms of percentages. The sample is all generalists and cardiologists who were not integrated in 2007 from the states in the SK&A data. Standard errors are clustered on hospital service area.

# Appendix B

## Appendix to Chapter 2

### B.1 Distance of referrals

Figure B.1: Distribution of referrals by distance from the beneficiary's ZIP code centroid



The sample includes all referrals of sample patients from PCPs to hospitals within 100 miles of the PCP's practice over the period 2007-2013, where sample patients are Medicare FFS patients aged  $\geq 65$  who were covered by both Part A and Part B the whole portion of the year they were alive.



## B.2 Method for matching physician-years to hospital-owners

To determine the hospital-owner for each physician practice (where applicable) I map each hospital listed as the physician practice owner in the SK&A data, to its Medicare hospital identifier, using a fuzzy matching algorithm based on the hospital's name and state. Where there is missing data in a year (i.e. all even numbered years) I assume that if a practice was owned by hospital  $h$  in the previous year, it is still owned by hospital  $h$ . At this point I have a file that maps practice-years to Medicare hospital identifiers. Next I map the practice-years to physician-years using the physician-year to practice mapping I developed in chapter 1.<sup>1</sup> Now I have a file that maps physician-years to hospitals using physician and hospital identifiers found in the Medicare data. Using the Medicare data I construct a complete list of physician-hospital pairs where the hospitals are within 100 miles of the physician's practice.<sup>2</sup> To this list I add the SK&A names and Medicare IDs of the hospital-owner.

In some cases the hospital listed as the owner (based on the Medicare ID) does not appear in the choice set. One common reason for the hospital-owner's ID not appearing in the choice set is that many hospitals have similar names, which resulted in the incorrect ID being assigned to the hospital-owner listed in the SK&A data. For instance there are many St. John's Hospitals. Since respondents to the SK&A survey complete the survey while in their own practice they may not feel the need to specify which St. John's Hospital they are affiliated with if there is only one nearby, meanwhile my automated matching algorithm assigned the Medicare ID for a random St. John's Hospital within the same state. Other reasons for the hospital-owner ID not appearing in the choice set include that

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<sup>1</sup>The physician-year to practice mapping is based on a combination of the SK&A data and the NPPESS NPI registry. For more details refer to section A.2.

<sup>2</sup>Where physicians operate from multiple practices I include all hospitals within 100 miles of any of the physician's practices.

physician practices near state borders may be owned by a hospital across state lines, that some remote physician practices are more than 100 miles from their listed owner, that some hospitals report jointly with an affiliate in the Medicare data, and that VA hospitals are not included in the AHA survey from which I get my hospital location data. For physician's whose hospital-owner is not included in their choice set I perform manual adjustments. Where there is a hospital in the choice set with a similar name to the hospital listed as the owner in the SK&A data, I assign this Medicare ID to the hospital-owner variable. Where there are multiple hospitals in the choice set with a similar name to the hospital listed as the owner in the SK&A data I assign the Medicare ID of the closest of these hospitals to the hospital-owner variable. This solves the "many hospitals with the same name" and "hospitals near state borders" problems. When there is no hospital with a similar name listed in the choice set, I look up the hospital name in the American Hospital Directory website (<https://www.ahd.com/search.php>). Sometimes the notes sections of the results specify that the hospital is reporting jointly with an affiliated hospital. If the affiliated hospital is in the choice set, I assign the Medicare ID the affiliated hospital to the hospital-owner variable. If this still has not solved the problem I google the Medicare hospital ID and occasionally find that the hospital operates under another name that does appear in the hospital choice set. After this manual process about 3% of physician-years have a hospital-owner that is not included in their listed choice set – this includes the physician-years where their hospital-owner is a VA hospital or more than 100 miles away.

### **B.3 Comparing results of linear and non-linear models for the referral count regressions**

Unlike the adverse patient outcomes in the previous chapter, the referral count variables display considerable variation on the intensive margin. They approximately follow Poisson

distributions with low  $\lambda$ s. I prefer the linear model with fixed effect and a instrument presented in the body of the paper, due to the incidental parameters problem, accounting for changes in the mix of patient unobservables due to integration, and computational simplicity. However, as a check on my main results I also approximate the decision to refer using a Poisson model (see equation B.1) and a Poisson model with PCP fixed effects (see equation B.3). In case there is over-dispersion I also run a negative binomial regression. Unfortunately, the computational shortcut that allows efficient calculation of the Poisson model with fixed effects does not work for the negative binomial model hence it must be estimated by including dummy variables for each PCP. This is computationally infeasible given my equipment so I am unable to present negative binomial results with physician fixed effects.

$$r_{it} = \exp(\alpha + \beta v_{jt} + \gamma \mathbf{x}_{it} + \eta \mathbf{w}_{jt} + \theta \mathbf{w}_{h\tau} + \phi_t) + e_{it} \quad (\text{B.1})$$

$$r_{it} = \exp(\alpha + \beta v_{jt} + \gamma \mathbf{x}_{it} + \eta \mathbf{w}_{jt} + \theta \mathbf{w}_{h\tau} + \phi_t + \delta_j) + \varepsilon_{it} \quad (\text{B.2})$$

To account for changes in the mix of patient unobservables due to integration formally I could use a two-stage residual inclusion method, but the relationship between the endogenous regressor is extremely strong, as a rough check on the linear results I present a version of the model where I directly replace the endogenous variable with the instrument. In the linear model this is the reduced form. In the non-linear models this does not have a formal name.

In Tables B.1 I report the coefficients and average marginal effects of interest from the propensity to make referrals to specialists regressions. Among the models with no physician fixed effects it is apparent that changing the form of the model from linear to Poisson or negative binomial makes very little difference to the average marginal effect in either the model with no instrument or the “reduced form” model. In spite of the similarity between the Poisson and negative binomial results, a simple test of equidispersion found overdispersion

after the Poisson model for both referral count measures so the negative binomial model is technically a better fit. If anything using the negative binomial results suggest a slightly stronger relationship between PCP integration and propensity to refer than the OLS or Poisson models.

Producing an average marginal effect from the Poisson model with physician fixed effects is not possible – because I estimated the model using a method that does not estimate the fixed-effects it is not possible to integrate over them. (Estimating the fixed effects by including PCP dummies is computationally infeasible.) However, one can see that the direction, approximate magnitude, and significance of the coefficients is mostly unaffected by switching from the linear fixed effects to the Poisson fixed effects model. The only apparent difference is that the relationship between having an integrated PCP and being referred to a specialist appear significant at the 10% level in the Poisson model with physician fixed effects but not in the linear model with physician fixed effects. However, the results of the preferred specification are essentially the same between the linear and Poisson models. My main take-away from Table B.1 is that the bulk of my results for the relationship between physician integration and the volume of referrals they make to specialists is not attributable to my use of a linear model. However, marginally significant result in the Poisson fixed effects model with no instrument perhaps reinforces the importance of accounting for changes in the mix of patient unobservable characteristics that may result from a physician integrating with a hospital.

Table B.1: Influence of integration on PCP's propensity to make referrals to specialists – comparing linear and non-linear models

	Model				
	OLS	Poisson	Neg. Binom.	Linear FE	Poisson FE
<i>Panel A: No instrument</i>					
coefficient	0.099***	0.059***	0.060***	0.029	0.021*
SE on coeff	(0.017)	(0.010)	(0.010)	(0.019)	(0.011)
average marginal effect	0.099***	0.099***	0.101***	0.029	
SE on average marginal effect	(0.017)	(0.017)	(0.017)	(0.019)	
number of beneficiary-years	553,170	553,170	553,170	553,170	546,675
<i>Panel B: "Reduced form"</i>					
coefficient	0.043*	0.026*	0.026*	0.002	0.005
SE on coeff	(0.023)	(0.014)	(0.014)	(0.020)	(0.012)
average marginal effect	0.043*	0.043*	0.044*	0.002	
SE on average marginal effect	(0.023)	(0.023)	(0.023)	(0.020)	
number of beneficiary-years	435,796	435,796	435,796	435,796	429,281

# Appendix C

## Appendix to Chapter 4

### C.1 Calculating elasticity of medical malpractice claims given changes in PSI rates

We want to determine the elasticity of paid medical malpractice claims to small changes in PSI rates predicted by our model. We calculate the effect of a 0.001 standard deviation reduction in each of the PSIs on medical malpractice claims.

We begin with equation 4.1. One can express the residual number of PSI- $j$  events as the difference between the actual value of the dependent variable and its expectation, based on the estimated values of the regression parameters:

$$\text{PSI}j\text{Res}_{it} = \ln(1 + \text{PSI}j_{it}) - E[\ln(1 + \text{PSI}j_{it})]$$

Holding fixed the parameter estimates, if the number of PSI- $j$  occurrences in hospital (county for Texas)  $i$  and year  $t$  decreased by a fraction  $\Delta_{\text{PSI}j}$ , the new residual PSI- $j$  measure would be:

$$\text{PSI}j\text{Res}_{it}^* = \ln[1 + (1 - \Delta_{\text{PSI}j})\text{PSI}j_{it}] - E[\ln(1 + \text{PSI}j_{it})] \quad (\text{C.1})$$

We specify values for the  $\Delta_{\text{PSI}j}$  as follows:

$$\text{Let } \Delta_{\text{PSI}j} = 0.001 \times \frac{\sigma(\text{PSI}j\text{Rate}_{kt})}{\mu(\text{PSI}j\text{Rate}_{kt})},$$

where  $\text{PSI}j\text{Rate}_{kt}$  is the PSI- $j$  rate at hospital  $k$  in year  $t$  ( $\Delta_{\text{PSI}j}$  is computed at the hospital level for both Florida and Texas); and  $\sigma(X)$  and  $\mu(X)$  are the standard deviation and mean of variable  $X$ , respectively. In calculating the standard deviation and mean, we exclude hospital-years with PSI rates below the 10th and above the 90th percentile to eliminate the effect of outliers such as hospital-years with few cases at risk. The resulting average value across PSIs of  $\Delta_{\text{PSI}j}$  is 0.00046 in Florida and 0.00052 in Texas, or roughly a 0.05% change in PSI- $j$ , on average across the PSIs in both states.

Let  $\Delta\text{PSI}j\text{Res}_{it} = (\text{PSI}j\text{Res}_{it}^* - \text{PSI}j\text{Res}_{it})$  and a normalized variation of this measure be:

$$\text{norm}(\Delta\text{PSI}j\text{Res}_{it}) = \frac{\Delta\text{PSI}j\text{Res}_{it}}{\sigma(\text{PSI}j\text{Res}_{it})}$$

where  $\sigma(\text{PSI}j\text{Res}_{it})$  is the standard deviation of the non-normalized residual PSI- $j$  measure across hospital-years (county-years). We sum across all PSIs and renormalize to obtain a normalized change in the pooled PSI measure for each hospital-year (county-year)  $it$ :

$$\Delta\text{PooledPSIMeasure}_{it} = \frac{\sum_{j \in \text{PSI}} \text{norm}(\Delta\text{PSI}j\text{Res}_{it})}{\sigma(\text{PSI}j\text{Res}_{it})}$$

Our main regression results (see equations 4.5 and 4.6) provide an estimate of the relationship between the change in the pooled PSI measure and the change in the medical malpractice risk measure:

$$\text{norm}(\Delta\text{MedMalRes}_{it}) = \gamma \times \Delta\text{PooledPSIMeasure}_{it}$$

where  $\gamma$  is the estimated coefficient on the pooled PSI measure. We can denormalize  $\Delta\text{MedMalRes}_{it}$  by multiplying by  $\sigma(\text{MedMalRes}_{it})$  across counties and years:

$$\Delta\text{MedMalRes}_{it} = \gamma \times \Delta\text{PooledPSIMeasure}_{it} \times \sigma(\text{MedMalRes}_{it})$$

Similar to equation C.1, we have, for medical malpractice claims, and a fractional decrease  $\Delta_{\text{MM}}$  in the number of claims in a county-year:

$$\ln[1 + (1 - \Delta_{\text{MM}})\text{MedMal}_{it}] = E[\ln(1 + \text{MedMal}_{it})] + \text{MedMalRes}_{it}^*.$$

However, this equation has no solution for counties with zero medical malpractice claims. We work around this problem by taking the expected value of both sides within the sample:

$$E\{\ln[1 + (1 - \Delta_{\text{MM}})\text{MedMal}_{it}]\} = E[\ln(1 + \text{MedMal}_{it})] + E[\text{MedMalRes}_{it}^*].$$

$$E\{\ln[1 + (1 - \Delta_{\text{MM}})\text{MedMal}_{it}]\} = E[\ln(1 + \text{MedMal}_{it})] + E[\text{MedMalRes}_{it}].$$

This has no analytic solution, so we estimate  $\Delta_{\text{MM}}$  numerically by searching for the value at which the equation holds in our sample. Lastly, we estimate the fractional change in medical malpractice claims due to a one standard deviation reduction in PSI rates as  $1000 \times \Delta_{\text{MM}}$ .

## C.2 Additional results for Florida

Tables C.1 and C.2 present first-stage regression results, from the regressions we use to compute the medical malpractice claims measure and the individual PSI measures.



Table C.1: First-stage regressions for PSIs in Florida

Dep. variable: $\ln(1+\text{PSI}_j \text{ count})$	j=2	j=3	j=4	j=5	j=6	j=7	j=8	j=9	j=10	j=11	j=12	j=13	j=14	j=15	j=17	j=18	j=19
$\ln(1+\text{cases at risk } j)$	0.072 (2.65)	0.640 (6.88)	0.715 (17.54)	1.093 (1.61)	-0.011 (-0.11)	0.302 (1.33)	0.014 (1.02)	-0.013 (-0.37)	0.074 (5.79)	0.322 (13.80)	0.150 (4.67)	0.306 (14.72)	-0.002 (-0.11)	0.339 (2.31)	0.089 (6.91)	0.591 (27.03)	0.401 (24.77)
% female	-0.001 (-0.16)	0.026 (5.20)	-0.006 (-1.05)	-0.001 (-0.42)	-0.009 (-0.48)	-0.004 (-0.36)	-0.001 (0.10)	0.001 (0.03)	-0.004 (-0.82)	0.005 (0.62)	0.008 (1.11)	0.001 (0.01)	0.007 (1.30)	0.010 (1.30)	-0.003 (-0.60)	0.004 (1.01)	0.007 (1.45)
% white	-0.003 (-2.27)	-0.008 (-4.25)	-0.001 (-0.56)	-0.000 (-0.34)	-0.000 (-0.03)	-0.003 (-1.15)	0.000 (0.38)	0.003 (0.19)	0.000 (0.09)	-0.000 (-0.08)	-0.003 (-1.30)	-0.001 (-0.66)	0.000 (0.38)	0.005 (1.80)	0.001 (0.47)	0.003 (1.05)	0.003 (1.44)
% Hispanic	0.001 (0.86)	0.008 (5.87)	0.000 (0.43)	-0.000 (-0.51)	-0.004 (-3.03)	0.003 (1.31)	-0.001 (-1.34)	-0.004 (-2.24)	-0.001 (-0.01)	0.001 (0.27)	0.003 (0.22)	0.000 (0.22)	0.000 (0.30)	-0.005 (-2.59)	-0.000 (-0.37)	-0.000 (-0.22)	-0.001 (-0.46)
% aged 0-4	0.003 (0.30)	-0.008 (-0.56)	-0.014 (-0.79)	0.031 (2.15)	0.008 (0.42)	0.008 (0.42)	0.007 (0.78)	0.007 (0.44)	0.003 (0.22)	0.003 (0.17)	-0.021 (-1.05)	0.024 (0.93)	0.006 (0.53)	0.021 (0.93)	0.044 (1.94)	0.052 (2.92)	0.044 (2.06)
% aged 5-19	0.005 (0.57)	-0.026 (-1.76)	0.014 (0.95)	0.035 (2.52)	0.011 (0.59)	-0.009 (-0.61)	0.011 (1.34)	0.021 (1.44)	0.024 (1.55)	0.030 (1.65)	0.000 (0.00)	0.030 (2.03)	0.023 (2.15)	0.005 (0.30)	0.027 (1.36)	0.055 (3.53)	0.025 (1.43)
% aged 35-49	0.006 (0.58)	-0.012 (-0.82)	-0.004 (-0.25)	0.011 (1.22)	0.001 (0.05)	0.007 (0.42)	0.005 (0.66)	-0.000 (-0.02)	-0.003 (-0.17)	0.020 (1.10)	-0.009 (-0.49)	0.018 (1.13)	0.009 (0.84)	-0.000 (-0.00)	0.022 (1.10)	0.068 (4.19)	0.041 (2.24)
% aged 50-64	0.001 (0.12)	-0.004 (-0.35)	-0.015 (-1.33)	0.019 (2.41)	0.009 (0.68)	-0.015 (-1.02)	0.009 (1.54)	0.011 (0.90)	0.014 (0.55)	0.008 (0.28)	0.004 (0.32)	0.032 (2.40)	0.011 (1.55)	0.019 (0.58)	0.008 (0.58)	0.052 (4.14)	0.012 (0.84)
% aged 65-84	0.004 (0.54)	-0.005 (-0.59)	-0.004 (-0.42)	0.012 (1.82)	0.011 (0.90)	-0.005 (-0.43)	0.009 (1.68)	0.008 (0.68)	0.005 (0.57)	0.009 (0.77)	-0.005 (-0.42)	0.019 (1.84)	0.008 (1.17)	0.014 (0.93)	0.004 (0.32)	0.044 (3.98)	0.015 (1.16)
% aged 85+	0.012 (1.43)	0.025 (1.97)	-0.003 (-0.28)	0.003 (0.42)	-0.007 (-0.52)	0.004 (0.25)	0.006 (0.97)	-0.016 (-1.21)	-0.000 (-0.04)	0.011 (0.77)	-0.004 (-0.32)	0.010 (0.73)	-0.004 (-0.51)	-0.030 (-1.75)	0.018 (1.12)	0.058 (4.47)	0.030 (2.13)
$\ln(\text{discharges})$	0.037 (0.51)	-0.007 (-0.06)	0.001 (0.01)	-1.036 (-1.59)	0.134 (1.49)	-0.179 (-0.73)	0.133 (2.70)	0.146 (1.29)	0.027 (0.38)	-0.046 (-0.31)	0.116 (1.14)	-0.010 (-0.09)	0.043 (0.79)	0.145 (1.07)	-0.187 (-2.23)	0.276 (3.87)	0.078 (0.87)
$\ln(1+\text{comorbidity } 1 \text{ count})$	0.041 (1.39)	-0.011 (-0.21)	0.039 (1.00)	0.019 (0.68)	0.099 (1.84)	-0.008 (-0.13)	0.058 (2.42)	0.165 (2.91)	0.046 (1.18)	0.059 (0.90)	0.098 (1.45)	0.044 (0.81)	0.095 (3.18)	0.248 (3.44)	0.015 (0.27)	-0.102 (-2.16)	0.005 (0.11)
$\ln(1+\text{comorbidity } 2 \text{ count})$	0.041 (0.92)	0.141 (1.57)	0.126 (1.99)	-0.016 (-0.35)	0.046 (0.63)	0.163 (1.65)	0.031 (0.97)	-0.064 (-0.69)	0.041 (0.80)	0.093 (0.94)	-0.039 (-0.51)	-0.033 (-0.48)	0.011 (0.27)	-0.139 (-1.35)	-0.010 (-0.17)	-0.034 (-0.60)	-0.002 (-0.04)
$\ln(1+\text{comorbidity } 3 \text{ count})$	0.031 (1.01)	0.239 (5.02)	0.113 (2.69)	0.085 (2.70)	0.103 (2.01)	0.269 (3.92)	0.054 (2.47)	0.179 (3.40)	0.100 (2.38)	0.235 (3.17)	0.252 (3.72)	0.200 (3.29)	0.008 (0.25)	0.190 (2.86)	-0.042 (-0.88)	0.055 (1.21)	0.005 (0.11)
$\ln(1+\text{comorbidity } 4 \text{ count})$	-0.045 (-1.04)	-0.054 (-0.69)	-0.034 (-0.60)	-0.060 (-1.45)	-0.163 (-2.34)	-0.188 (-2.33)	-0.093 (-2.76)	-0.144 (-1.70)	-0.129 (-2.32)	-0.157 (-1.68)	-0.227 (-3.02)	-0.230 (-3.01)	-0.093 (-2.19)	-0.329 (-3.28)	-0.043 (-0.67)	-0.027 (-0.51)	-0.012 (-0.19)
$\ln(1+\text{comorbidity } 5 \text{ count})$	0.026 (1.60)	-0.034 (-1.30)	0.074 (3.20)	0.044 (2.56)	0.071 (2.48)	-0.040 (-1.16)	0.021 (1.47)	0.039 (1.35)	0.048 (2.23)	0.066 (1.79)	0.039 (1.23)	0.050 (1.57)	0.048 (2.85)	0.040 (1.02)	0.007 (0.27)	0.018 (0.68)	0.057 (2.08)
$\ln(1+\text{comorbidity } 6 \text{ count})$	-0.072 (-1.49)	0.013 (0.12)	-0.243 (-3.26)	-0.012 (-0.24)	-0.104 (-1.06)	0.173 (1.38)	-0.089 (-2.25)	-0.156 (-1.49)	-0.182 (-2.59)	-0.173 (-1.29)	-0.199 (-1.92)	-0.000 (-0.00)	-0.016 (-0.33)	-0.044 (-0.35)	0.192 (2.48)	0.054 (0.76)	0.068 (0.74)

Table C.1: First-stage regressions for PSIs in Florida - continued

Dep. variable: $\ln(1+\text{PSI}_j \text{ count})$	j=2	j=3	j=4	j=5	j=6	j=7	j=8	j=9	j=10	j=11	j=12	j=13	j=14	j=15	j=17	j=18	j=19
$\ln(1+\text{comorbidity } 7 \text{ count})$	-0.039 (-1.23)	-0.207 (-4.29)	-0.046 (-1.07)	0.082 (2.83)	0.230 (4.44)	0.203 (3.60)	0.015 (0.72)	0.228 (4.03)	0.097 (2.32)	0.162 (2.34)	0.278 (4.64)	0.106 (1.97)	0.043 (1.30)	0.277 (4.26)	0.100 (2.20)	0.151 (3.24)	0.106 (2.09)
$\ln(1+\text{comorbidity } 8 \text{ count})$	-0.063 (-2.20)	-0.073 (-1.51)	-0.152 (-4.58)	-0.103 (-3.87)	-0.166 (-4.23)	-0.186 (-3.10)	-0.062 (-2.92)	-0.223 (-5.38)	-0.130 (-3.39)	-0.162 (-2.71)	-0.217 (-4.26)	-0.130 (-2.54)	-0.123 (-4.55)	-0.282 (-5.60)	-0.189 (-4.56)	-0.100 (-2.07)	-0.188 (-2.37)
$\ln(1+\text{comorbidity } 9 \text{ count})$	0.070 (1.98)	0.124 (2.64)	0.095 (2.26)	0.065 (1.85)	0.093 (1.84)	0.128 (2.23)	0.034 (1.20)	0.054 (0.96)	0.054 (1.16)	0.062 (0.98)	0.158 (2.96)	0.041 (0.77)	0.072 (2.17)	0.012 (0.12)	0.024 (0.49)	-0.103 (-2.52)	-0.068 (-1.55)
$\ln(1+\text{comorbidity } 10 \text{ count})$	0.002 (0.04)	-0.271 (-3.13)	0.061 (0.99)	-0.121 (-2.58)	-0.226 (-3.06)	-0.569 (-5.30)	-0.021 (-0.63)	-0.227 (-3.03)	-0.007 (-0.13)	-0.056 (-0.53)	-0.233 (-3.07)	-0.029 (-0.35)	-0.102 (-2.43)	-0.437 (-4.84)	0.006 (0.09)	-0.158 (-2.87)	-0.116 (-1.59)
$\ln(1+\text{comorbidity } 11 \text{ count})$	-0.019 (-0.62)	0.113 (2.52)	0.013 (0.33)	-0.033 (-1.18)	0.058 (1.17)	0.068 (1.10)	0.021 (0.80)	0.049 (0.90)	-0.004 (-0.11)	-0.067 (-0.94)	-0.012 (-0.20)	-0.065 (-1.15)	0.031 (0.91)	0.094 (2.37)	-0.062 (-1.54)	-0.003 (-0.08)	-0.044 (-1.00)
$\ln(1+\text{comorbidity } 12 \text{ count})$	0.110 (3.82)	0.156 (3.73)	0.125 (4.04)	0.070 (2.76)	0.156 (3.68)	0.229 (4.37)	0.078 (4.10)	0.167 (4.05)	0.138 (3.88)	0.267 (4.88)	0.254 (5.45)	0.197 (4.34)	0.090 (3.57)	0.180 (3.23)	0.139 (4.00)	0.033 (1.04)	0.066 (1.59)
$\ln(1+\text{comorbidity } 13 \text{ count})$	-0.042 (-3.01)	-0.085 (-4.63)	-0.048 (-3.37)	0.007 (0.54)	-0.046 (-2.25)	-0.061 (-2.82)	-0.025 (-2.43)	-0.034 (-1.72)	-0.011 (-0.65)	-0.098 (-3.73)	0.084 (4.23)	0.092 (4.35)	-0.016 (-1.20)	-0.003 (-0.13)	-0.030 (-1.51)	-0.093 (-5.71)	-0.067 (-3.44)
$\ln(1+\text{comorbidity } 14 \text{ count})$	0.095 (2.88)	-0.052 (-0.91)	-0.031 (-0.59)	0.065 (1.50)	0.009 (0.15)	0.142 (1.91)	-0.013 (-0.51)	0.176 (2.43)	0.121 (2.27)	0.111 (1.19)	0.187 (2.62)	0.026 (0.38)	0.105 (2.37)	0.201 (2.54)	0.247 (4.35)	0.159 (3.23)	0.200 (3.98)
$\ln(1+\text{comorbidity } 15 \text{ count})$	-0.016 (-0.63)	-0.017 (-0.42)	-0.061 (-1.70)	0.021 (0.72)	0.025 (0.55)	-0.030 (-0.52)	-0.080 (-3.07)	0.022 (0.42)	-0.011 (-0.22)	0.022 (0.29)	0.064 (1.30)	-0.023 (-0.40)	0.010 (0.33)	0.062 (1.15)	0.038 (0.88)	0.029 (0.75)	0.024 (0.54)
$\ln(1+\text{comorbidity } 16 \text{ count})$	-0.049 (-1.60)	0.139 (2.59)	-0.092 (-1.87)	-0.004 (-0.10)	0.115 (1.89)	0.085 (1.22)	-0.006 (-0.22)	0.075 (0.98)	-0.125 (-2.42)	-0.117 (-1.51)	-0.037 (-0.57)	-0.145 (-2.30)	-0.022 (-0.60)	0.168 (1.83)	-0.113 (-2.34)	-0.112 (-2.63)	-0.042 (-1.01)
$\ln(1+\text{comorbidity } 17 \text{ count})$	0.034 (1.80)	0.065 (2.04)	0.026 (1.12)	0.029 (1.77)	0.098 (3.50)	0.197 (4.29)	0.067 (4.12)	0.102 (3.25)	0.071 (2.73)	0.073 (2.02)	0.140 (4.59)	0.064 (1.80)	0.008 (0.45)	0.043 (1.11)	0.035 (1.34)	-0.002 (-0.10)	0.004 (0.13)
constant	-0.744 (-0.90)	-2.060 (-1.88)	0.885 (0.79)	-1.333 (-1.66)	-0.602 (-0.42)	-0.005 (-0.00)	-1.300 (-1.99)	-1.273 (-0.91)	-0.366 (-0.34)	-1.576 (-1.06)	-0.592 (-0.39)	-2.169 (-1.66)	-1.428 (-1.77)	-3.355 (-1.65)	-1.105 (-0.77)	-6.308 (-4.70)	-3.404 (-2.33)
Adjusted $R^2$	0.280	0.859	0.843	0.286	0.639	0.689	0.222	0.686	0.360	0.724	0.860	0.630	0.261	0.794	0.588	0.866	0.912

Table shows results of “first-stage” regressions, used to construct the individual PSI measures for Florida. Sample is 2,484 hospital-years.  $t$ -statistics, with standard errors clustered on hospital, in parentheses. Significant results (at 5% level) in **boldface**. Base for percentage variables is number of hospital discharges.

Table C.2: First-stage regressions for medical malpractice claims in Florida

Dependent variable	<i>ln(1+adjusted malpractice claims)</i>	
	coefficient	<i>t</i> -stat
ln(1 + PSI 2 cases at risk)	0.037	(0.65)
ln(1 + PSI 3 cases at risk)	-0.023	(-0.29)
ln(1 + PSI 4 cases at risk)	<b>0.236</b>	<b>(4.34)</b>
ln(1 + PSI 5 cases at risk)	<b>-3.505</b>	<b>(-2.03)</b>
ln(1 + PSI 6 cases at risk)	1.120	(0.98)
ln(1 + PSI 7 cases at risk)	-0.141	(-0.57)
ln(1 + PSI 8 cases at risk)	0.059	(0.40)
ln(1 + PSI 9 cases at risk)	-0.079	(-0.32)
ln(1 + PSI 10 cases at risk)	0.253	(1.46)
ln(1 + PSI 11 cases at risk)	<b>-0.472</b>	<b>(-2.85)</b>
ln(1 + PSI 12 cases at risk)	-0.042	(-0.16)
ln(1 + PSI 13 cases at risk)	<b>0.164</b>	<b>(2.64)</b>
ln(1 + PSI 14 cases at risk)	0.060	(0.88)
ln(1 + PSI 15 cases at risk)	-1.030	(-0.91)
ln(1 + PSI 17 cases at risk)	0.071	(0.73)
ln(1 + PSI 18 cases at risk)	-0.065	(-1.51)
ln(1 + PSI 19 cases at risk)	-0.033	(-0.32)
% female	<b>0.020</b>	<b>(2.69)</b>
% white	-0.002	(-0.74)
% hispanic	<b>0.003</b>	<b>(2.08)</b>
% aged 0-4	-0.042	(-1.43)
% aged 5-19	-0.032	(-1.19)
% aged 35-49	0.010	(0.50)
% aged 50-64	-0.011	(-0.68)
% aged 65-84	-0.009	(-0.70)
% aged 85+	0.008	(0.52)
ln(discharges)	<b>3.484</b>	<b>(2.07)</b>
ln(1 + comorbidity 1 count)	<b>0.267</b>	<b>(3.64)</b>
ln(1 + comorbidity 2 count)	<b>-0.168</b>	<b>(-2.27)</b>
ln(1 + comorbidity 3 count)	0.091	(1.48)
ln(1 + comorbidity 4 count)	-0.124	(-1.57)
ln(1 + comorbidity 5 count)	0.016	(0.41)
ln(1 + comorbidity 6 count)	0.145	(1.38)
ln(1 + comorbidity 7 count)	0.086	(1.50)
ln(1 + comorbidity 8 count)	<b>-0.206</b>	<b>(-3.71)</b>
ln(1 + comorbidity 9 count)	0.032	(0.56)
ln(1 + comorbidity 10 count)	-0.078	(-0.87)
ln(1 + comorbidity 11 count)	-0.036	(-0.60)
ln(1 + comorbidity 12 count)	<b>0.224</b>	<b>(4.42)</b>
ln(1 + comorbidity 13 count)	-0.020	(-0.70)
ln(1 + comorbidity 14 count)	0.027	(0.41)
ln(1 + comorbidity 15 count)	0.012	(0.22)
ln(1 + comorbidity 16 count)	0.094	(1.55)
ln(1 + comorbidity 17 count)	0.033	(0.94)
constant	-0.804	(-0.49)
Adjusted R <sup>2</sup>	0.492	

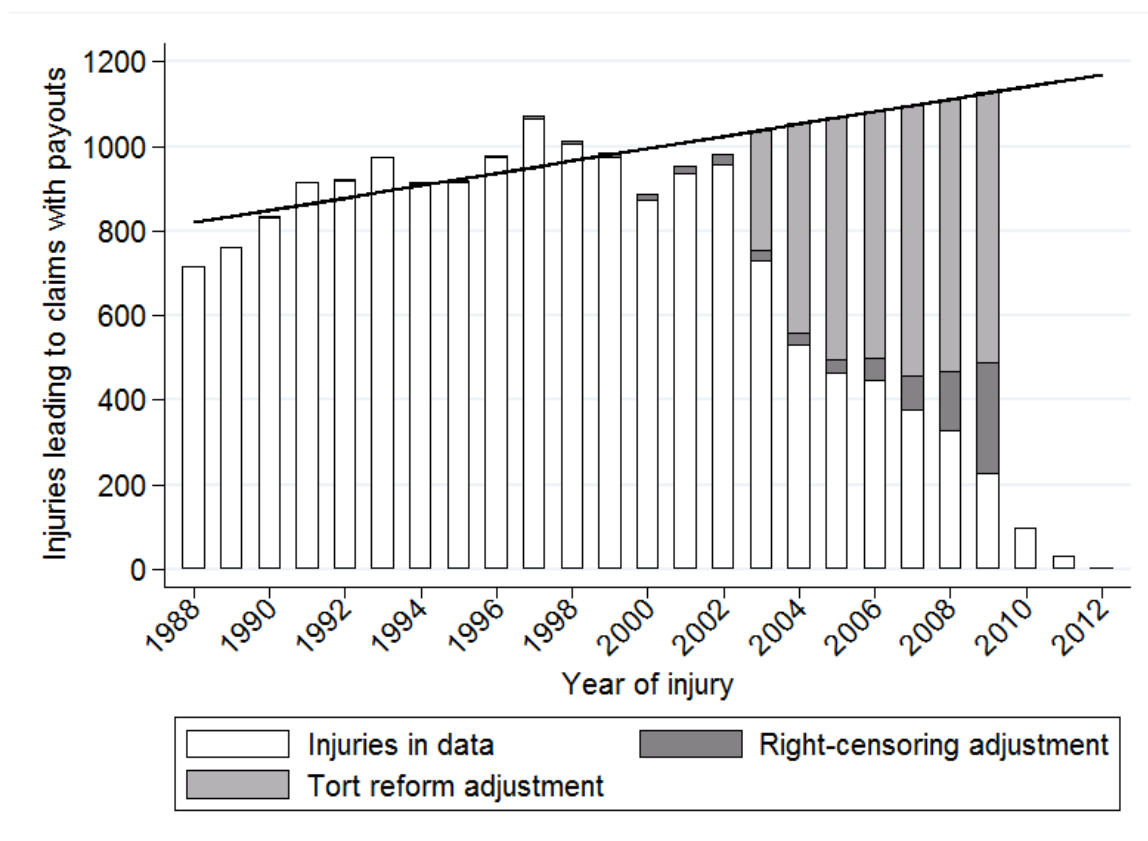
Table shows results of first-stage regressions, used to construct the medical malpractice claims measure for Florida. Regressions cover 2,484 hospital-years over 1999-2010. Percent aged 20-34 is the excluded age category. *t*-statistics, with standard errors clustered on hospital, in parentheses. The base for percentage variables is number of hospital discharges. Significant results (at 5% level) in **boldface**.

## **C.3 Additional Results for Texas**

### **C.3.1 Time Consistency and Adjusted Number of Claims Analysis**

Texas adopted a cap on non-economic damages and other limits on medical malpractice suits effective September 1, 2003. We therefore adjust the number of reported claims in the same way as for Florida, by adjusting both for the effect of the damages cap and for right-censoring due to the claims dataset including only closed paid claims. Figure C.1 is similar to Figure 4.1, and shows the number of closed, paid medical malpractice claims in Texas by injury year.

Figure C.1: Injuries leading to paid medical malpractice claims in Texas by year of injury



Number of closed paid medical malpractice claims included in Texas dataset, by injury year. Hollow bars show actual data. Drop in number of claims beginning with 2004 reflects two main factors: (i) the flow of new claims dropped after Florida adopted tort reforms, effective for suits filed after September 1, 2003; and (ii) some claims resulting from injuries in those years are not yet closed (right-censoring of the closed claims dataset). Adjustments for these factors are shown in light and dark shaded bars, respectively. See text for estimation procedure. Tort reform adjustment (light shading): Regression line shows estimated number of paid claims without tort reform, based on linear trend using data from 1988 to 2002: No. of claims =  $819.4 + 14.6 \times (\text{year} - 1988)$  [ $t = 3.50$ ].

### C.3.2 Texas Patient Safety Data

Table C.3 is similar to Table 4.2, and provides summary statistics on the number of PSI events, the number of cases at risk for each PSI, and the PSI rate (per 10,000 cases at risk) for Texas.

As in Florida we construct PSI counts using state inpatient data. We obtain the Texas



Table C.3: Summary statistics for medical malpractice claims and PSI events in Texas

	Total events	Cases at risk	Rate per 10,000 cases at risk	County level rates			
				Mean	Std. Dev.	Min	Max
PSI 2 : Death in Low-mortality DRGs	2,266	7,118,360	3.18	1.88	5.25	0	60
PSI 3 : Pressure Ulcer	210,233	7,545,093	278.64	174.47	520.84	0	5,564
PSI 4 : Death of Surgical Inpatients with Serious Treatable Complications	24,907	185,368	1,343.65	20.67	61.57	0	587
PSI 5 : Foreign Body Left during Procedure	1,747	24,381,028	0.72	1.45	4.13	0	38
PSI 6 : Iatrogenic Pneumothorax	10,700	18,817,811	5.69	8.88	25.36	0	223
PSI 7 : Central Venous Catheter-related Bloodstream Infection	27,792	16,936,240	16.41	23.06	84.89	0	961
PSI 8 : Postoperative Hip Fracture	1,082	4,209,569	2.57	0.90	2.79	0	34
PSI 9 : Postoperative Hemorrhage or Hematoma	15,541	6,177,664	25.16	12.90	39.04	0	398
PSI 10 : Postoperative Physiologic and Metabolic Derangement	2,637	3,352,285	7.87	2.19	7.65	0	74
PSI 11 : Postoperative Respiratory Failure	21,623	2,646,709	81.70	17.94	57.68	0	575
PSI 12 : Postoperative Pulmonary Embolism or Deep Vein Thrombosis	56,194	6,199,311	90.65	46.63	159.17	0	1,733
PSI 13 : Postoperative Sepsis	11,139	818,268	136.13	9.24	31.98	0	352
PSI 14 : Postoperative Wound Dehiscence	1,824	1,183,009	15.42	1.51	4.14	0	44
PSI 15 : Accidental Puncture or Laceration	55,563	19,512,195	28.48	46.11	127.76	0	968
PSI 17 : Birth Trauma - Injury to Neonate	5,239	3,877,979	13.51	4.35	13.67	0	147
PSI 18 : Obstetric Trauma - Vaginal Delivery with Instrument	40,106	255,126	1,572.01	33.28	91.97	0	957
PSI 19 : Obstetric Trauma - Vaginal Delivery without Instrument	72,982	2,431,500	300.15	60.57	175.31	0	1,446
<b>Total PSI events</b>	<b>561,575</b>	<b>125.6 mil.</b>	<b>44.69</b>	<b>466.04</b>	<b>1,356.27</b>	<b>0</b>	<b>12,709</b>
Population	229.5 mil.			190,484	465,786	2435	4,070,989
Hospital Discharges	30.6 mil.			25,424	67,960	54	564,915
Medical malpractice claims	6,547			5.43	16.69	0	202
Adjusted medical malpractice claims	10,948			9.09	25.51	0	247

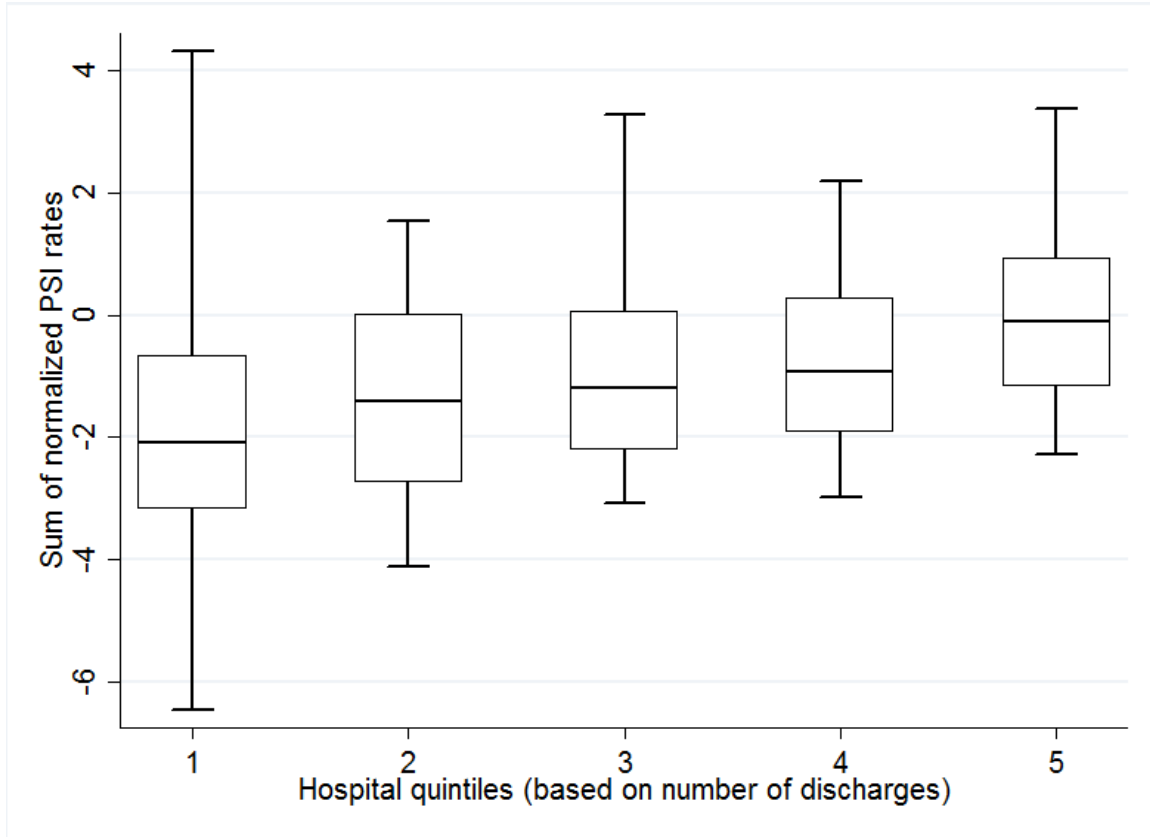
Table shows, for 1999-2009, total Texas statewide PSI events, cases at risk for each PSI, and statewide PSI rate per 10,000 cases at risk; and county-level mean, standard deviation, minimum, and maximum annual PSI rates. County-level data is based on 1,205 county-years with positive discharges (139 distinct counties). Bottom rows show: county population, hospital discharges, medical malpractice claims, and "adjusted medical malpractice claims," using adjustments described in text to remove estimated effects of right-censoring of claims and Texas's 2003 medical malpractice reforms.

inpatient dataset from the Texas Inpatient Public Use Data File, available for 1999-2013. The Texas dataset file is similar to the Florida dataset in structure, and contains a unique identifier for each discharge, the year and quarter of the discharge, hospital identifiers, patient demographic characteristics, and ICD-9-CM diagnosis and procedure codes. We map Texas hospitals to counties, compute county-level PSI rates, and then merge those rates with the county-level medical malpractice data. Many small Texas counties have no hospitals and therefore no PSI events. We therefore limit the Texas sample to county-years with positive discharges. This leaves a sample with 139 usable counties (out of 254 counties in Texas) and 1,205 county-years over 1999-2009. In four cases, affiliated hospitals in adjacent rural counties reported together. We combined these county pairs. The Texas inpatient data include roughly 31 million discharges between 1999 and 2009, and roughly 600,000 PSI events. The Texas malpractice claims data includes 6,547 raw claims and 10,948 adjusted claims.

Similar to Florida (see Table 4.2), PSI rates exhibit substantial variation, suggesting that hospitals can improve their PSI rates at manageable cost, because some hospitals are achieving lower rates.

Figure C.2 is similar to Figure 4.2 in the text and provides box-and-whiskers plots showing the variation in PSI rates, both across hospital size quintiles and within each quintile. In Texas, only 9% of the variation in PSI rates is across quintiles, the remaining 91% is within quintiles.

Figure C.2: Box and whiskers plots for Texas hospital pooled PSI rates by hospital discharge quintiles



Box and whiskers plots of hospital pooled PSI rates for hospital size quintiles, based on mean discharges over 1999-2009, for 320 Texas hospitals (2,182 hospital-years) with cases at risk for each PSI. Pooled PSI rate is mean (across years) for hospital  $k$  of  $\sum_{j \in \text{PSI}} \{\text{norm}(\text{PSI-}j \text{ rate per 1,000 cases at risk})\}_{kt}$ , with PSI rates normalized to mean = 0 and standard deviation = 1. Boxes give 25th, 50th, and 75th percentiles; whiskers give 2.5th and 97.5th percentiles.

Table C.4 is similar to Table 4.3 and shows hospital-year level correlations among rates for individual PSIs. As in Florida, there is strong evidence that the PSIs contain a common core. They exhibit substantial correlation and the loadings the first principal component are positive and similar in magnitude for all PSIs. These loadings range from 0.171 to 0.302 for all PSIs except PSI-3 (loading = 0.043).



Table C.4: Correlations among individual PSI measures in Texas

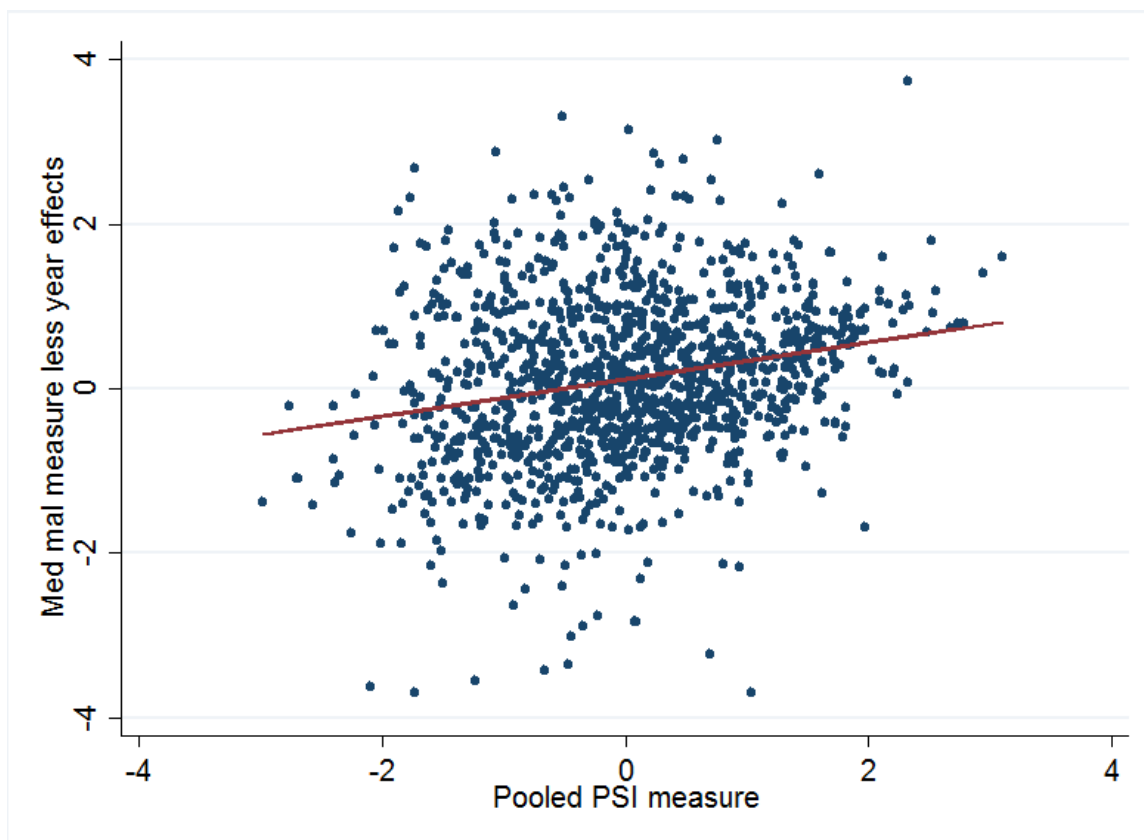
	PSI 2	PSI 3	PSI 4	PSI 5	PSI 6	PSI 7	PSI 8	PSI 9	PSI 10	PSI 11	PSI 12	PSI 13	PSI 14	PSI 15	PSI 17	PSI 18
PSI 3	-0.009															
PSI 4	0.200**	-0.011														
PSI 5	0.280**	0.053	0.225**													
PSI 6	0.214**	0.034	0.321**	0.250**												
PSI 7	0.149**	0.099**	0.363**	0.241**	0.409**											
PSI 8	0.208**	0.019	0.194**	0.258**	0.221**	0.226**										
PSI 9	0.136**	-0.009	0.273**	0.212**	0.377**	0.360**	0.229**									
PSI 10	0.261**	0.077**	0.306**	0.347**	0.300**	0.329**	0.325**	0.230**								
PSI 11	0.168**	0.038	0.412**	0.237**	0.310**	0.380**	0.219**	0.276**	0.492**							
PSI 12	0.104**	0.066*	0.203**	0.155**	0.212**	0.320**	0.103**	0.370**	0.181**	0.262**						
PSI 13	0.172**	0.049	0.352**	0.292**	0.296**	0.399**	0.234**	0.237**	0.501**	0.615**	0.198**					
PSI 14	0.220**	0.011	0.211**	0.308**	0.289**	0.279**	0.315**	0.289**	0.254**	0.172**	0.178**	0.170**				
PSI 15	0.0410	0.076**	0.253**	0.163**	0.327**	0.263**	0.061*	0.372**	0.135**	0.223**	0.231**	0.164**	0.154**			
PSI 17	0.184**	0.090**	0.211**	0.338**	0.358**	0.247**	0.210**	0.232**	0.282**	0.213**	0.144**	0.224**	0.304**	0.116**		
PSI 18	0.083**	-0.021	0.246**	0.201**	0.232**	0.247**	0.183**	0.221**	0.151**	0.207**	0.089**	0.174**	0.211**	0.136**	0.314**	
PSI 19	0.056	0.080**	0.221**	0.176**	0.243**	0.303**	0.158**	0.202**	0.169**	0.235**	0.087**	0.214**	0.160**	0.173**	0.283**	0.513**

Correlations among PSI measures. PSI measures are defined in the text. Counties are indexed by  $i$ , PSIs by  $j$ , Charlson comorbidities by  $m$ , years by  $t$ . Sample is 1,205 county-year observations over 1999-2009 with positive discharges, for 139 counties. Significance level: \* 5%, \*\* 1%.

### C.3.3 Simple correlation between malpractice measure and PSI measure

Figure C.3 is similar to Figure 4.3 in the text, and shows the simple correlation for Texas, across counties and years, between the residual malpractice claims measure and the residual PSI measure. As in Florida, we find a strong, positive relationship between the pooled PSI measure and the malpractice measure (coefficient = 0.225,  $t = 5.71$ ).

Figure C.3: Medical malpractice measure versus pooled PSI measure in Texas



Sample is 1,205 county-years over 1999-2009 with positive discharges. Linear trend:  $y = 0.115 + 0.225x$  ( $t = 5.71$ ), with standard errors clustered on county.

### **C.3.4 First stage regression results**

Tables C.5 and C.6 present first-stage regression results, from the regressions we use to compute the residual medical malpractice claims measure and the residual PSI measure.

Table C.5: First-stage regressions for PSIs in Texas

Dep.var.: ln(1+PSIj count)	j=2	j=3	j=4	j=5	j=6	j=7	j=8	j=9	j=10	j=11	j=12	j=13	j=14	j=15	j=17	j=18	j=19
ln(1+cases at risk j)	-0.089 (-1.71)	<b>0.624</b> (6.37)	<b>0.459</b> (10.33)	<b>0.671</b> (2.36)	<b>0.880</b> (4.46)	0.431 (1.32)	<b>-0.063</b> (-2.57)	<b>-0.097</b> (-2.97)	0.007 (0.32)	<b>0.166</b> (4.45)	-0.034 (-0.65)	<b>0.183</b> (5.86)	<b>-0.050</b> (-2.06)	0.512 (1.97)	-0.026 (-1.39)	<b>0.507</b> (16.13)	<b>0.176</b> (6.75)
% white	-1.030 (-2.45)	-0.775 (-1.80)	-0.480 (-1.42)	-1.283 (-2.82)	-1.012 (-2.03)	-1.501 (-3.48)	-0.877 (-1.98)	-0.734 (-1.59)	-1.565A (-3.01)	-1.778 (-3.86)	-0.576 (-1.18)	<b>-1.672</b> (-4.00)	-0.839 (-1.86)	-0.420 (-0.71)	<b>-1.430</b> (-2.71)	0.043 (0.09)	0.140 (0.26)
% Hispanic	<b>0.516</b> (2.78)	<b>0.501</b> (2.33)	<b>0.613</b> (2.77)	<b>0.738</b> (4.45)	<b>0.691</b> (3.03)	<b>0.970</b> (3.91)	<b>0.590</b> (3.12)	<b>0.275</b> (1.21)	<b>1.029</b> (3.67)	<b>1.164</b> (3.42)	<b>0.475</b> (1.53)	<b>1.437</b> (5.26)	<b>0.545</b> (3.45)	-0.013 (-0.05)	<b>1.109</b> (4.49)	<b>0.630</b> (2.19)	0.289 (0.85)
% aged 62+	<b>2.010</b> (2.44)	<b>-2.102</b> (-2.02)	<b>2.066</b> (2.96)	<b>1.634</b> (2.66)	<b>2.659</b> (3.21)	<b>3.080</b> (3.38)	<b>1.215</b> (2.71)	<b>2.101</b> (2.68)	<b>3.155</b> (3.46)	<b>2.219</b> (2.24)	<b>2.161</b> (2.05)	<b>2.276</b> (2.98)	<b>1.177</b> (2.16)	0.251 (0.24)	0.467 (0.47)	-1.149 (-1.11)	-1.713 (-1.51)
% rural	-0.054 (-0.26)	0.315 (1.66)	-0.341 (-1.86)	0.009 (0.06)	-0.263 (-1.25)	-0.276 (-1.25)	-0.103 (-0.88)	-0.334 (-1.71)	-0.001 (-0.00)	-0.208 (-0.93)	-0.450 (-1.59)	-0.104 (-0.56)	-0.022 (-0.17)	-0.372 (-1.61)	0.027 (0.13)	-0.340 (-1.55)	-0.422 (-1.70)
income per capital	<b>0.289</b> (2.56)	0.104 (0.95)	<b>0.184</b> (2.09)	<b>0.372</b> (3.77)	<b>0.325</b> (2.23)	<b>0.785</b> (6.52)	<b>0.266</b> (2.63)	<b>0.374</b> (3.14)	<b>0.478</b> (3.35)	<b>0.529</b> (3.77)	<b>0.496</b> (3.77)	<b>0.604</b> (5.55)	<b>0.312</b> (2.83)	<b>0.484</b> (3.74)	<b>0.737</b> (4.80)	<b>0.488</b> (3.11)	<b>0.445</b> (2.93)
ln(discharges)	<b>0.520</b> (4.00)	<b>-0.506</b> (-3.70)	<b>-0.327</b> (-2.53)	<b>-0.465</b> (-2.00)	<b>-0.621</b> (-3.78)	<b>-0.576</b> (-2.10)	-0.040 (-0.56)	<b>0.314</b> (2.38)	<b>-0.203</b> (-2.12)	<b>-0.437</b> (-2.91)	0.192 (1.15)	<b>-0.433</b> (-3.38)	0.122 (1.36)	0.182 (0.77)	0.302 (1.67)	-0.271 (-1.72)	<b>0.819</b> (3.29)
ln(1+comorbidity 1 count)	0.038 (0.97)	-0.086 (-1.50)	0.001 (0.03)	0.035 (1.12)	0.039 (0.76)	0.012 (0.18)	0.011 (0.39)	0.073 (1.52)	0.001 (0.02)	0.033 (0.55)	0.097 (1.30)	-0.011 (-0.21)	0.000 (0.01)	0.112 (1.72)	-0.031 (-0.52)	0.089 (1.52)	0.143 (1.96)
ln(1+comorbidity 2 count)	0.151 (1.58)	0.178 (1.62)	-0.012 (-0.14)	0.021 (0.28)	-0.024 (-0.22)	-0.114 (-1.02)	0.065 (0.98)	-0.058 (-0.61)	0.153 (1.67)	0.178 (1.62)	-0.040 (-0.34)	<b>0.270</b> (2.70)	0.121 (1.68)	-0.169 (-1.39)	0.175 (1.43)	0.007 (0.07)	0.094 (0.87)
ln(1+comorbidity 3 count)	0.013 (0.30)	<b>0.134</b> (2.46)	-0.021 (-0.42)	-0.003 (-0.09)	-0.090 (-1.50)	-0.080 (-1.17)	-0.021 (-0.67)	-0.061 (-1.32)	-0.019 (-0.39)	0.016 (0.24)	0.021 (0.31)	0.011 (0.18)	-0.027 (-0.71)	0.007 (0.10)	0.118 (1.92)	0.010 (0.15)	0.138 (1.82)
ln(1+comorbidity 4 count)	-0.084 (-1.22)	0.114 (1.26)	<b>0.309</b> (4.02)	-0.081 (-1.51)	-0.036 (-0.44)	0.059 (0.60)	0.027 (0.56)	-0.113 (-1.54)	0.023 (0.34)	0.175 (1.74)	0.012 (0.12)	0.155 (1.86)	-0.069 (-1.31)	<b>-0.263</b> (-2.67)	-0.057 (-0.69)	<b>0.180</b> (2.07)	-0.212 (-1.97)
ln(1+comorbidity 5 count)	<b>0.093</b> (3.53)	0.005 (0.19)	-0.003 (-0.13)	0.031 (1.50)	-0.015 (-0.44)	-0.015 (-0.46)	0.030 (0.46)	-0.018 (-0.75)	-0.011 (-0.41)	<b>-0.107</b> (-3.17)	-0.008 (-0.20)	-0.037 (-1.21)	0.033 (1.66)	-0.045 (-1.25)	-0.000 (-0.01)	0.029 (0.79)	-0.006 (-0.15)
ln(1+comorbidity 6 count)	0.032 (0.33)	0.147 (1.13)	0.054 (0.52)	0.052 (0.56)	0.180 (1.32)	0.190 (1.34)	0.096 (0.75)	-0.098 (-0.75)	0.203 (1.97)	0.215 (1.39)	-0.056 (-0.39)	0.120 (1.00)	-0.006 (-0.06)	-0.250 (-1.65)	-0.055 (-0.35)	-0.190 (-1.20)	<b>-0.694</b> (-4.07)
ln(1+comorbidity 7 count)	-0.032 (-0.79)	0.082 (1.26)	-0.023 (-0.42)	-0.013 (-0.38)	0.012 (0.18)	0.060 (0.91)	0.011 (0.37)	0.060 (1.19)	-0.060 (-1.34)	-0.077 (-1.24)	0.018 (0.28)	-0.083 (-1.54)	0.005 (0.14)	0.038 (0.57)	0.030 (0.53)	0.030 (0.40)	0.042 (0.50)
ln(1+comorbidity 8 count)	<b>-0.060</b> (-2.09)	0.032 (0.68)	-0.066 (-1.60)	<b>-0.063</b> (-2.63)	-0.021 (-0.57)	<b>-0.106</b> (-2.49)	-0.004 (-0.15)	-0.004 (-0.16)	<b>-0.078</b> (-2.26)	-0.070 (-1.24)	-0.076 (-1.34)	-0.033 (-0.70)	-0.026 (-0.98)	0.039 (0.62)	0.014 (0.27)	0.038 (0.84)	0.015 (0.30)
ln(1+comorbidity 9 count)	-0.028 (-0.91)	0.007 (0.17)	<b>-0.103</b> (-2.54)	-0.042 (-1.61)	<b>-0.084</b> (-2.18)	-0.004 (-0.08)	-0.036 (-1.16)	-0.028 (-0.69)	-0.025 (-0.74)	<b>-0.105</b> (-2.16)	-0.008 (-0.17)	<b>-0.113</b> (-2.64)	-0.024 (-0.92)	-0.059 (-1.08)	<b>-0.092</b> (-2.43)	<b>-0.092</b> (-2.04)	-0.102 (-1.91)
ln(1+comorbidity 10 count)	-0.151 (-1.59)	-0.045 (-0.34)	0.036 (0.40)	-0.048 (-0.56)	0.021 (0.17)	0.141 (1.20)	-0.025 (-0.39)	0.031 (0.29)	0.069 (0.89)	0.052 (0.50)	0.041 (0.26)	0.124 (1.11)	-0.076 (-1.01)	-0.198 (-1.15)	-0.244 (-1.81)	0.093 (0.95)	-0.132 (-0.86)
ln(1+comorbidity 11 count)	<b>-0.087</b> (-2.08)	0.029 (0.29)	<b>-0.080</b> (-2.07)	<b>-0.075</b> (-2.01)	-0.013 (-0.13)	-0.079 (-1.79)	<b>-0.087</b> (-2.08)	-0.018 (-0.45)	<b>-0.118</b> (-2.91)	<b>-0.167</b> (-3.86)	-0.021 (-0.56)	<b>-0.185</b> (-4.43)	-0.056 (-1.43)	0.040 (1.00)	-0.085 (-2.08)	<b>-0.110</b> (-2.71)	-0.068 (-1.70)

Table C.5: First-stage regressions for PSIs in Texas - continued

Dep.var.: $\ln(1+\text{PSI})$ count	j=2	j=3	j=4	j=5	j=6	j=7	j=8	j=9	j=10	j=11	j=12	j=13	j=14	j=15	j=17	j=18	j=19
$\ln(1+\text{comorbidity } 12 \text{ count})$	(-2.42) <b>0.067</b> (2.15)	(0.63) <b>0.187</b> (3.73)	(-2.07) 0.004 (0.13)	(-2.22) 0.048 (1.70)	(-0.29) 0.020 (0.45)	(-1.35) 0.064 (1.42)	(-2.90) <b>0.098</b> (4.51)	(-0.43) 0.066 (1.72)	(-2.73) 0.062 (1.71)	(-3.18) <b>0.097</b> (2.22)	(-0.36) <b>0.187</b> (3.87)	(-4.12) 0.074 (1.77)	(-1.76) 0.049 (1.97)	(0.76) 0.064 (1.16)	(-1.79) 0.037 (0.97)	(-2.17) 0.081 (1.70)	(-0.98) <b>0.133</b> (2.75)
$\ln(1+\text{comorbidity } 13 \text{ count})$	0.013 (0.68)	0.023 (1.30)	0.039 (1.88)	<b>0.032</b> (2.13)	0.023 (1.14)	0.051 (1.93)	-0.010 (-0.64)	0.007 (0.36)	<b>0.065</b> (3.91)	<b>0.101</b> (4.48)	<b>0.102</b> (3.77)	<b>0.097</b> (4.76)	0.003 (0.21)	0.024 (1.07)	<b>0.047</b> (2.03)	0.003 (0.15)	-0.046 (-1.85)
$\ln(1+\text{comorbidity } 14 \text{ count})$	-0.033 (-0.57)	-0.062 (-0.86)	<b>0.151</b> (2.60)	0.077 (1.51)	0.075 (1.08)	<b>0.259</b> (3.43)	<b>0.131</b> (2.74)	<b>0.294</b> (4.26)	0.103 (1.64)	<b>0.214</b> (3.25)	<b>0.177</b> (2.38)	0.127 (1.96)	<b>0.145</b> (2.79)	<b>0.261</b> (2.86)	0.083 (1.18)	0.100 (1.29)	0.082 (0.94)
$\ln(1+\text{comorbidity } 15 \text{ count})$	-0.044 (-1.38)	0.001 (0.02)	-0.056 (-1.29)	-0.020 (-0.75)	-0.034 (-0.74)	-0.013 (-0.32)	-0.051 (-1.81)	-0.008 (-0.20)	-0.029 (-0.95)	-0.034 (-0.71)	-0.059 (-1.12)	-0.040 (-1.12)	0.000 (0.01)	0.056 (1.02)	-0.017 (-0.41)	<b>-0.105</b> (-2.28)	-0.037 (-0.73)
$\ln(1+\text{comorbidity } 16 \text{ count})$	<b>-0.086</b> (-2.00)	0.052 (0.87)	0.068 (1.55)	-0.004 (-0.13)	<b>0.111</b> (2.04)	<b>0.135</b> (2.10)	0.011 (0.32)	<b>0.149</b> (3.24)	0.009 (0.21)	0.031 (0.51)	0.127 (1.95)	0.048 (0.94)	0.021 (0.64)	<b>0.244</b> (3.75)	-0.019 (-0.39)	0.049 (0.89)	0.026 (0.40)
$\ln(1+\text{comorbidity } 17 \text{ count})$	<b>0.175</b> (6.62)	0.025 (0.68)	<b>0.316</b> (11.00)	<b>0.174</b> (6.38)	<b>0.265</b> (8.14)	<b>0.351</b> (10.03)	<b>0.130</b> (4.68)	<b>0.251</b> (7.58)	<b>0.255</b> (8.11)	<b>0.391</b> (10.42)	<b>0.243</b> (5.79)	<b>0.301</b> (9.30)	<b>0.167</b> (6.33)	<b>0.193</b> (4.89)	<b>0.260</b> (7.65)	<b>0.238</b> (6.68)	<b>0.318</b> (7.37)
Constant	<b>-2.826</b> (-4.64)	-0.094 (-0.15)	-0.104 (-0.19)	<b>-1.472</b> (-3.30)	<b>-2.897</b> (-4.61)	<b>-1.916</b> (-2.39)	-0.790 (-1.98)	<b>-2.457</b> (-4.03)	<b>-1.369</b> (-2.06)	-0.675 (-0.84)	<b>-3.091</b> (-3.54)	-0.871 (-1.28)	<b>-1.304</b> (-2.85)	<b>-2.584</b> (-3.32)	<b>-2.081</b> (-2.97)	0.202 (0.30)	<b>-2.302</b> (-2.59)
Adjusted R <sup>2</sup>	0.668	0.929	0.918	0.682	0.834	0.884	0.595	0.886	0.725	0.867	0.911	0.848	0.681	0.900	0.752	0.903	0.906

Table shows results of “first-stage” regressions, used to construct the residual PSI measure for Texas. Sample is 1,205 county-years. *t*-statistics, with standard errors clustered on county, in parentheses. Significant results (at 5% level) in **boldface**. Base for percentage variables is county population.

Table C.6: First-stage regressions for medical malpractice measure in Texas

Dependent variable	<i>ln</i> (1+adjusted malpractice claims)	
	coefficient	<i>t</i> -stat
ln(1 + PSI 2 cases at risk)	<b>-0.118</b>	<b>(-2.10)</b>
ln(1 + PSI 3 cases at risk)	-0.116	(-1.02)
ln(1 + PSI 4 cases at risk)	0.077	(1.56)
ln(1 + PSI 5 cases at risk)	1.274	(0.98)
ln(1 + PSI 6 cases at risk)	1.998	(1.00)
ln(1 + PSI 7 cases at risk)	0.135	(0.36)
ln(1 + PSI 8 cases at risk)	<b>0.375</b>	<b>(2.35)</b>
ln(1 + PSI 9 cases at risk)	0.121	(0.60)
ln(1 + PSI 10 cases at risk)	<b>-0.469</b>	<b>(-2.19)</b>
ln(1 + PSI 11 cases at risk)	0.344	(1.61)
ln(1 + PSI 12 cases at risk)	<b>-0.631</b>	<b>(-2.48)</b>
ln(1 + PSI 13 cases at risk)	<b>0.180</b>	<b>(3.64)</b>
ln(1 + PSI 14 cases at risk)	0.084	(1.28)
ln(1 + PSI 15 cases at risk)	-2.648	(-1.27)
ln(1 + PSI 17 cases at risk)	-0.243	(-1.33)
ln(1 + PSI 18 cases at risk)	0.060	(1.55)
ln(1 + PSI 19 cases at risk)	0.260	(1.34)
% white	-0.070	(-0.11)
% hispanic	0.499	(1.72)
% over 62	-0.512	(-0.53)
% rural	0.051	(0.25)
income per capita	<b>0.331</b>	<b>(2.17)</b>
ln(discharges)	-0.391	(-0.61)
ln(1 + comorbidity 1 count)	-0.010	(-0.22)
ln(1 + comorbidity 2 count)	<b>0.209</b>	<b>(2.37)</b>
ln(1 + comorbidity 3 count)	-0.034	(-0.64)
ln(1 + comorbidity 4 count)	0.044	(0.51)
ln(1 + comorbidity 5 count)	0.003	(0.09)
ln(1 + comorbidity 6 count)	0.056	(0.50)
ln(1 + comorbidity 7 count)	-0.068	(-1.04)
ln(1 + comorbidity 8 count)	0.018	(0.42)
ln(1 + comorbidity 9 count)	0.007	(0.12)
ln(1 + comorbidity 10 count)	-0.002	(-0.02)
ln(1 + comorbidity 11 count)	-0.083	(-1.83)
ln(1 + comorbidity 12 count)	<b>0.134</b>	<b>(2.94)</b>
ln(1 + comorbidity 13 count)	0.002	(0.08)
ln(1 + comorbidity 14 count)	0.094	(1.05)
ln(1 + comorbidity 15 count)	<b>-0.127</b>	<b>(-2.31)</b>
ln(1 + comorbidity 16 count)	0.030	(0.60)
ln(1 + comorbidity 17 count)	<b>0.147</b>	<b>(3.60)</b>
constant	-2.895	(-3.34)
Adjusted R <sup>2</sup>	0.737	

Table shows results of first-stage regressions, used to construct the residual medical malpractice claims measure for Texas. Regressions include 1,205 county years over 1999-2009. Percent aged 20-34 is the excluded age category. *t*-statistics, with standard errors clustered on county, in parentheses. The base of percentage variables is county population. Significant results (at 5% level) in **boldface**.

### C.3.5 Baby claims

Table C.7: Texas neonate birth trauma PSI and newborn injury claims

Dependent variable	Newborn malpractice claims measure		
	(1)	(2)	(3)
Regression model	OLS	County RE	County FE
PSI 17 measure: Birth Trauma - Injury to Neonate	<b>0.212***</b> (3.62)	<b>0.150***</b> (3.68)	0.072* (1.73)

Each cell is from a separate regression of the newborn medical malpractice measure on the PSI-17 measure. Malpractice and PSI measures are defined in the text. t-statistics, with standard errors clustered on county, in parentheses. Sample is same as in Table 4.7. Significance level: \* 10%, \*\* 5%, \*\*\* 1%. Significant results (at 5% level) in **boldface**.

#### C.3.5.1 Individual PSI measures and robustness checks

In Table C.8, we provide results for Texas for the individual PSI measures. All 17 coefficients are positive with pooled OLS and county RE, and 14 of 17 are positive with county FE. As in Florida, none of the negative coefficients are significantly different from zero. Of the positive coefficients, 13 are significant and positive with pooled OLS, 12 with county RE, and 3 with county FE. Weaker statistical significance with FE is expected, because the FE model relies only on “within” variation.

If we include all 17 PSI measures in a single regression, as separate explanatory variables, including all the PSIs individually, the sum of coefficients is 0.459 ( $t = 6.30$ ) for pooled OLS; 0.447 ( $z = 6.27$ ) for county RE, and 0.244 ( $t = 2.57$ ) for county FE.

If we conduct a principal components analysis, the coefficient on the first principal component (coeff. = 0.128;  $t = 2.68$ ) is similar to the 0.130 coefficient on the pooled PSI measure reported in the text, Table 4.7.



Table C.8: Texas regressions of medical malpractice measure on individual PSI measures

Dependent variable	Medical malpractice measure		
	(4)	(5)	(6)
Regression model	OLS	County RE	County FE
PSI 2 : Death in Low-mortality DRGs	<b>0.173***</b> (4.42)	<b>0.155***</b> (4.49)	<b>0.101***</b> (2.97)
PSI 3 : Pressure Ulcer	<b>0.0809**</b> (2.39)	<b>0.0669**</b> (2.07)	0.0430 (1.10)
PSI 4 : Death of Surgical Inpatients with Serious Treatable Complications	0.0569* (1.80)	0.0371 (1.30)	-0.00324 (-0.10)
PSI 5 : Foreign Body Left during Procedure	<b>0.163***</b> (3.99)	<b>0.139***</b> (3.68)	0.0546 (1.35)
PSI 6 : Iatrogenic Pneumothorax	<b>0.124***</b> (3.21)	<b>0.102***</b> (2.86)	0.0412 (1.02)
PSI 7 : Central Venous Catheter-related Bloodstream Infection	<b>0.139***</b> (3.79)	<b>0.120***</b> (3.35)	0.0615 (1.38)
PSI 8 : Postoperative Hip Fracture	<b>0.186***</b> (4.66)	<b>0.172***</b> (4.50)	0.0244 (0.86)
PSI 9 : Postoperative Hemorrhage or Hematoma	<b>0.134***</b> (3.31)	<b>0.134***</b> (3.95)	<b>0.121***</b> (3.73)
PSI 10 : Postoperative Physiologic and Metabolic Derangement	<b>0.172***</b> (4.24)	<b>0.149***</b> (4.00)	0.0587 (1.40)
PSI 11 : Postoperative Respiratory Failure	<b>0.138***</b> (3.97)	<b>0.125***</b> (3.87)	<b>0.0915**</b> (2.31)
PSI 12 : Postoperative Pulmonary Embolism or Deep Vein Thrombosis	0.0376 (1.09)	0.0361 (1.06)	0.0112 (0.27)
PSI 13 : Postoperative Sepsis	<b>0.131***</b> (3.65)	<b>0.110***</b> (3.32)	0.0547 (1.49)
PSI 14 : Postoperative Wound Dehiscence	<b>0.133***</b> (3.09)	<b>0.106***</b> (2.63)	0.0206 (0.49)
PSI 15 : Accidental Puncture or Laceration	0.0426 (1.06)	0.0339 (0.99)	0.00890 (0.26)
PSI 17 : Birth Trauma - Injury to Neonate	<b>0.137***</b> (4.05)	<b>0.116***</b> (3.67)	0.0594 (1.45)
PSI 18 : Obstetric Trauma - Vaginal Delivery with Instrument	0.0312 (0.76)	0.0111 (0.31)	-0.0299 (-0.73)
PSI 19 : Obstetric Trauma - Vaginal Delivery without Instrument	<b>0.0859**</b> (2.11)	0.0556 (1.44)	-0.00783 (-0.17)

Each cell is from a separate regression of the medical malpractice measure on the indicated PSI measure. Medical malpractice and PSI measures are defined in the text. Sample is 1,205 county-years (139 counties) over 1999-2009 with positive discharges. t-statistics, with standard errors clustered on hospital, in parentheses. Significance level: \* 10%, \*\* 5%, \*\*\* 1%. Significant results (at 5% level) in boldface.



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