The Value of (Sub) Specialization: Evidence from Oncology

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Abstract

Specialization enhances professional productivity, but its benefits depend on access to the relevant expertise. In oncology, subspecialization—the narrowing of clinical focus within cancer care—has become increasingly common, yet its effects on patient outcomes remain poorly understood. This paper estimates the causal impact of access to subspecialized oncologists on treatment and survival using a differential distance instrument based on Medicare beneficiaries' proximity to subspecialists. Analyzing 6.1 million six-month chemotherapy episodes, we find that access to a relevant subspecialist reduces three-year mortality by 8.6% and episode spending by 3.5%. Potential mechanisms of reduced mortality are greater enrollment in disease-specific clinical trials and increased use of newer therapies. We find no evidence of increased care fragmentation and only modest indications of more intensive treatment near the end of life. Importantly, there are no mortality differences following initial oncology consultations, suggesting that survival benefits arise from differences in chemotherapy delivery rather than selective treatment of healthier patients. Our findings highlight the value of deep clinical expertise and raise important concerns about geographic disparities in access to subspecialized care.

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1 Introduction

Specialization is a fundamental determinant of the division of labor and the returns to skilled work, shaping how tasks are allocated and influencing productivity and economic outcomes (Smith, 1819). By allowing workers to develop expertise in narrowly defined areas, specialization often enhances efficiency and drives gains in output. However, it can also impose costs, including barriers to access, coordination inefficiencies, and a narrower focus that may neglect broader system-level objectives. These trade-offs raise the question of whether greater access to specialized services improves individual outcomes or if inefficiencies and coordination frictions reduce its benefits.

Despite the importance of specialization in modern economies, its implications within professions remain poorly understood. This paper examines specialization in medicine, focusing on medical oncology, to evaluate how high degrees of specialization affect patient outcomes. Medicine has a long history of specialization, with distinct medical specialties emerging as early as the 19th century (Weisz, 2006). Over time, physicians organized into specialties based on procedural similarities, diagnostic approaches, and disease patterns. More recently, "subspecialization" has emerged, further dividing specialists into narrower fields based on specific disease types. Medical oncology exemplifies this trend, with "subspecialists" focusing on cancers that affect the same anatomical region (e.g. breast cancer or prostate cancer) or share similar treatment approaches (e.g., leukemia and lymphoma). In 2008, 9% of chemotherapy episodes among Medicare fee-for-service patients were managed by subspecialized oncologists; by 2020, this share had doubled to 18% (Karadakic et al., 2025), reflecting the rapid expansion of novel treatments and cancer-specific clinical guidelines (Lozinski, 2024).[1]

Subspecialization can enhance precision of care when providers have the knowledge to tailor treatments to the biological and clinical nuances of specific and even rare, cancers or the specific needs of patients. However, it also presents challenges: achieving economies of scale in specialized care requires large patient volumes, leading to the uneven geographic distribution of subspecialists, with rural and underserved areas often facing limited access (Dingel et al., 2023; Karadakic et al., 2025). Additionally, many cancer patients have multiple chronic conditions and even multiple cancers, making care coordination increasingly complex. These dynamics make medical oncology an ideal setting for studying the trade-offs of specialization on patient outcomes, health care utilization, spending, and access disparities.

In this paper, we exploit exogenous variation in Medicare fee-for-service patients' dif-

[1]See Appendix Figure D1 for an overview in the rise of subspecialization within medical oncology in traditional Medicare between 2008 and 2020.

ferential distance to subspecialized oncologists for their specific cancer type versus general oncologists to estimate the causal effect of subspecialized oncologic care on patient outcomes. The growing trend of oncologic subspecialization, combined with geographic shifts in oncologists' practice locations over time, creates quasi-exogenous variation in access to subspecialists. Using a sample of 6.1 million six-month chemotherapy episodes (i.e., episodes initiated by oral or physician-administered systemic anticancer therapy) among 2.2 million patients, we compare individuals within the same ZIP Code Tabulation Area (ZCTA), diagnosed with the same cancer type, whose relative proximity to the relevant subspecialized oncologist versus a general oncologist changes over time. We show that differential distance strongly predicts whether a patient has access to or receives chemotherapy management from a subspecialized oncologist of the relevant cancer type. Under the plausible assumption that patients within the same ZCTA and with the same cancer type differ in subspecialist access only due to changes in differential distance over time, our instrumental variable strategy provides causal estimates of the effects of subspecialist access on health care utilization and patient health outcomes.

Our analysis finds that patients with greater access to subspecialists experience lower mortality in the years following treatment onset and are more likely to enroll in disease-specific clinical trials. Our instrumental variable estimates indicate that access to a subspecialized oncologist for the relevant cancer type reduces three-year mortality by 1.4 percentage points, an 8.6% reduction relative to the mean. However, we find no significant short-term mortality benefits within the first year, an expected result given that the benefits of differential treatment would take months to accumulate. In contrast, ordinary least squares (OLS) estimates suggest that access to subspecialists is associated with higher mortality, consistent with subspecialists treating more severe and potentially more advanced cancers.

Beyond improved survival, we also examine how access to subspecialized oncologists influences health care spending during six-month chemotherapy episodes. Using detailed Medicare claims, we construct comprehensive episode-level spending measures. Our two-stage least squares estimates indicate that subspecialist access reduces total spending, driven primarily by a $1,300 decline in Medicare Part B expenditures—largely from lower spending on injectable and infused chemotherapy agents and services related to injection and infusion.

To validate our instrumental variable, we conduct a series of robustness and falsification tests. First, we show that, conditional on ZCTA-level characteristics, beneficiary demographics, and fixed effects, the instrument is uncorrelated with more than three dozen patient health and demographic characteristics that influence clinical outcomes. Second,

we perform falsification exercises by randomly reassigning differential distances within years and find no significant effect on 1-year mortality. We further reassign differential distances to subspecialists of unrelated cancer types and again observe no systematic effects on mortality, supporting the exclusion restriction. Finally, our instrumental variable estimates on clinical trial enrollment reinforce the specialization mechanism: access to a subspecialized oncologist significantly increases enrollment in cancer trials specific to the patient's diagnosis, but has no effect on enrollment in non-cancer-related or generic multi-cancer trials. This suggests that the effects are primarily driven by specialized oncologic expertise rather than non-specific differences in physician quality or patient selection. Additionally, we demonstrate that our instrumental variable estimates do not impact health outcomes unrelated to oncologic care, reinforcing the interpretation that physician specialization, rather than general clinical skill or patient selection, is the key mechanism behind mortality reductions. For instance, access to a subspecialized oncologist has no effect on diagnoses of acute myocardial infarction, hip fractures, or strokes in the two years following chemotherapy initiation. Taken together, these results bolster our findings that oncologists' cancer-specific expertise is the primary driver of the observed mortality reductions.

To assess whether subspecialists systematically select healthier patients into chemotherapy, we examine a separate sample of new office visits with oncologists. Applying our identification strategy to this new sample, we find that initial consultations with subspecialized oncologists of the relevant cancer type increase the likelihood of initiating chemotherapy within 180 days. Importantly, we detect no impact of initial visits with subspecialists on the mortality of patients. These findings suggest that the observed survival gains are not driven by selection into treatment, but rather by differences in treatment of patients after the decision to initiate systemic therapy.

We explore several mechanisms that may explain the observed reductions in mortality and spending. First, we examine whether subspecialists facilitate greater access to cutting-edge treatments by linking Medicare claims to clinical trial data from ClinicalTrials.gov, using GPT-4 to classify trials by cancer type. We find that access to a subspecialized oncologist of the relevant cancer type significantly increases clinical trial enrollment, particularly in diagnosis-specific studies—suggesting improved access to novel therapies as a potential driver of better outcomes.

Second, we assess whether subspecialists are more likely to prescribe newer cancer therapies by analyzing the average FDA approval year of chemotherapy agents. Access to subspecialists is associated with a reduction in the average age of drugs by roughly half a year, primarily driven by the use of newer Part B drugs. Third, we assess end-of-

life treatment intensity and find that access to subspecialists reduces hospice use in the final 30 to 3 days of life by 29%, suggesting a shift toward slightly more aggressive care near death. This is accompanied by a 77.6% reduction in hospice spending relative to the mean, further indicating lower reliance on hospice services among patients with access to subspecialists. Finally, we examine whether subspecialist-led care alters the frequency of visits with providers during chemotherapy. Using episode-level claims data, we find no effect on the number of visits or the diversity of provider specialties, but observe a modest reduction in the number of unique providers. This suggests that concerns about increased care fragmentation with subspecialization are not borne out in oncology.

We further investigate the magnitude of specialization - the most subspecialized physicians should provide greater disease-specific benefit than less subspecialized physicians, even among the most specialized professionals. In our primary analysis, we focused on specialization in five groups of cancers: breast, gastrointestinal, leukemia/lymphoma, prostate/genitourinary, and thoracic. To examine whether the degree of specialization of the care-coordinating oncologist influences mortality outcomes, we used detailed cancer type classification beyond the five groups used in the main analyses to construct Herfindahl-Hirschman Indices (HHI) of oncologists' specialization, where a higher HHI indicates a greater concentration of a physician's caseload in a narrower set of cancer types. Instrumental variable estimates show that higher oncologist specialization significantly reduces mortality, with a 0.1-point increase in HHI (equivalent to moving from the 50th to the 64th percentile of the HHI distribution) lowering three-year mortality by one percentage point (a 2.6 percent reduction relative to the mean). These findings support the hypothesis that subspecialization could improve patient outcomes by facilitating access to advanced treatments and highly-specific physician expertise in a small number of cancers.

To understand how our estimated effects apply to different patient groups, we conduct a complier subgroup analysis, comparing compliers—those whose oncologist choice is influenced by differential distance—to the broader Medicare fee-for-service chemotherapy population. Compliers tend to be older, less likely to be female, and slightly more likely to be black. They also appear healthier on average, with fewer chronic condition flags for most chronic conditions. These differences help contextualize the impact of subspecialist access.

Our findings contribute to several strands of literature. First, we add to the broader economic literature on specialization and the division of labor (Smith, 1819; Becker and Murphy, 1992). Economic theory predicts that greater specialization enhances efficiency and expertise, yet also introduces potential trade-offs related to coordination costs and

accessibility (Rosen, 1983; Baumgardner, 1988). We examine these effects in a high-skilled professional setting—medicine, and specifically medical oncology—where specialization has expanded (Karadakic et al., 2025) at a time of rapid growth of medical knowledge and treatment options (Lozinski, 2024). Furthermore, our paper directly connects to Dingel et al. (2023) by providing empirical evidence on the benefits and constraints of higher specialization in health care markets. While their work highlights how larger markets facilitate specialization due to economies of scale, we quantify the patient-level implications of this specialization, showing that access to subspecialized oncologists improves survival, reduces spending and increases clinical trial enrollment.

Second, our research contributes to the broader literature on physician productivity, specialization, and its effects on treatment decisions and patient outcomes (Chan and Chen, 2022; Baicker and Chandra, 2004). While prior work has documented differences in health care practices between specialists and generalists—particularly in volume-outcome relationships among surgeons (Birkmeyer et al., 2002; Huckman and Pisano, 2006; Chandra and Staiger, 2007; Chowdhury, Dagash and Pierro, 2007; Sahni et al., 2016; Avdic, Lundborg and Vikström, 2019)—there is limited causal evidence on the effects of specialization in settings where expertise is defined not by procedural frequency, but by the breadth and depth of knowledge required for complex decision-making, as in medical oncology. In addition, subspecialization is a relatively recent phenomenon, and the absence of granular physician classifications and detailed patient outcomes has constrained prior efforts to measure its effects beyond associational studies on select malignancies (Shanafelt et al., 2012; Davidoff et al., 2020; Caswell-Jin et al., 2025). This paper addresses these limitations by constructing a new dataset on chemotherapy episodes, allowing us to directly classify oncologists into subspecialties using micro-level data and detailed patient characteristics—a level of granularity not documented in prior research. Our findings contribute to the growing literature on the division of labor in healthcare, providing new insights into how specialization influences the adoption of advanced treatments and impacts patient survival.

Finally, our study provides new evidence on how subspecialization shapes access to medical innovation. While prior research highlights disparities in clinical trial enrollment and treatment diffusion (Agha and Molitor, 2018; Alsan et al., 2022), it remains unclear whether subspecialists facilitate access to cutting-edge therapies. Linking Medicare claims to clinical trial data, we find that patients with greater access to subspecialized oncologists are significantly more likely to enroll in cancer trials. In addition, subspecialist access is associated with the use of newer chemotherapy drugs, as reflected by a lower average age of prescribed agents. These findings suggest that subspecialization enhances

access to medical innovation, with implications for both treatment equity and the role of physician expertise in the diffusion of new therapies.

The organization of this paper is as follows. Section 2 provides details on the context of medical oncology in the U.S. Section 3 describes our main data sources, the construction of our chemotherapy episodes (i.e., six-month episodes in which patients received systemic anti-cancer therapy), and the definition of our instrumental variable. In Section 4, we discuss our empirical strategy and the assumptions underpinning our identification approach. Section 5 presents our main results, Section 6 provides details on potential mechanisms, before concluding with Section 7.

2 Background on Medical Oncology

Cancer is the second leading cause of death in the United States, with older populations disproportionately affected. Among Medicare beneficiaries—predominantly comprised of individuals 65 and older—cancer care is a significant driver of health care utilization and costs. In 2015, cancer related health care spending was equivalent to 29% of overall Medicare spending, amounting to $183 billion, reflecting the high prevalence and complexity of cancer management in this population (Mariotto et al., 2020; Kaiser Family Foundation, 2025). The unique challenges posed by cancer in older adults, including comorbidities, frailty, and socioeconomic factors, necessitate specialized and coordinated approaches to care.[2]

Medical oncology is a cornerstone of cancer treatment. Medical oncologists—physicians trained in both internal medicine and oncology—primarily manage systemic therapies (i.e., cytotoxic chemotherapy, immunotherapy, targeted therapy, and hormone therapy). For simplicity, we refer to all of these as chemotherapy throughout the paper. Chemotherapy is typically delivered in one of two ways: infused or injected therapy, which is administered under the supervision of a healthcare professional, and oral therapy, which involves prescription medications taken by the patient in pill form.

In addition to prescribing and administering systemic anti-cancer and supportive care treatments, medical oncologists play a central role in coordinating care across multidisciplinary teams with surgical oncologists, radiation oncologists, and other health care professionals. Advances in treatment modalities over the last few decades—such as the development of targeted therapies addressing specific genetic mutations and im-

[2]The importance of subspecialization in ensuring up-to-date knowledge and optimal treatment is explicitly recognized by some cancer clinics and providers, who may emphasize it as part of their core mission statements (see e.g. Yale Cancer Clinic (2025)).

munotherapy leveraging the body's immune system—have transformed the landscape of cancer care (Sharma and Allison, 2015; Carroll et al., 2023). These innovations have improved survival rates for many cancers, including those in advanced stages (Emens et al., 2017). However, the increasing complexity of treatment regimens has also driven a trend toward subspecialization within oncology since oncologists face the challenge of keeping up-to-date with a rapidly expanding knowledge base (Lozinski, 2024) and availability of new anticancer therapies. For example, from 2000 to 2022, 573 agents were approved for various cancer indications (Scott et al., 2023). Similarly, Figure 1 demonstrates a sharp increase in medical guidelines for five distinct cancer categories. Guideline page counts between 2002 and 2020 have increased by over 300 percent for all cancer categories with leukemia and lymphoma experiencing a guideline page increase of 731 percent.

The increasing complexity of oncology care presents opportunities for oncologists to focus on specific cancer types or broader cancer categories, such as breast, gastrointestinal, or thoracic cancers, allowing them to develop expertise in managing the nuances of a set of specific malignancies. Subspecialization often centers around an anatomical region of the body (e.g. the breast, prostate, or the gastrointestinal tract) or cancer type (hematologic cancers). Subspecialized care may be particularly relevant for older patients, whose treatment plans require careful balancing of efficacy against potential risks associated with aging and comorbidities. However, subspecialization also may introduce challenges, particularly regarding equitable access to specialized care and potential fragmentation of care. Among Medicare beneficiaries, we find that the share of chemotherapy episodes treated by subspecialized oncologists in 2020 was more than three times higher in high income areas compared to low income areas (Karadakic et al., 2025).[3] This geographic disparity is especially consequential for Medicare beneficiaries, many of whom face barriers to travel or rely on local providers for care.

3 Data, Sample and Differential Distance Instrument

To define specialization of oncologists and assess the implications of access to subspecialized oncologists we draw upon a variety of data sources. The cornerstone of our analysis is a dataset containing chemotherapy episodes for the period 2008 to 2020, which is constructed utilizing 100% Medicare claims data accessed through the Center for Medicare and Medicaid Services' (CMS) Virtual Research Data Center (VRDC). We use data from

[3]See Appendix Figure D2 for differences in the share of chemotherapy episodes treated by highly subspecialized oncologists across ventiles of the U.S. population ordered from lowest income to highest income.

2007 to 2021 from Medicare Parts A, B and D. In addition we supplement our main sample with data on socioeconomic characteristics of ZCTAs obtained through the US Census Bureau. Furthermore, we are able to link information on clinical trials from clinicaltrials.gov to claims data using National Clinical Trial (NCT) numbers available in Medicare claims. Information on the Food and Drug Administration (FDA) approval year of novel cancer drugs was obtained from the National Cancer Institutes's (NCI) Surveillance, Epidemiology, and End Results (SEER) Program which provides FDA approval year through the Cancer Medication Enquiry Database (CanMED) and we link those to the relevant HCPCS and NDC codes in our data (National Cancer Institute, 2025).

3.1 Chemotherapy Episode Data

The foundation of our analysis is the construction of a dataset containing cancer care episodes as defined by the Oncology Care Model (OCM) (CMS, 2025). OCM was a value-based payment and care delivery model introduced by CMS that aimed to improve the quality and coordination of care for Medicare beneficiaries undergoing chemotherapy while reducing overall health care costs.[4] Following OCM methodology enables us to leverage the clinical and institutional experience of a large federal government initiative to capture an "industry standard" approach to measure cancer care. Using OCM definitions, we can define non-overlapping six month chemotherapy episodes for beneficiaries with cancer, assign episodes to a single cancer type, and assign a care coordinating principal medical oncologist based on the plurality of office visits during a chemotherapy episode.

In order to construct the final episode level data we include fee-for-service Medicare beneficiaries with cancer who received oral or physician-administered chemotherapy (including cytotoxic chemotherapy, targeted therapy, immunotherapy, and hormonal therapy) (CMS, 2020; Keating et al., 2021). Individuals in the episode sample are enrolled in Medicare Part A and B and do not receive the Medicare Endstage Renal Disease Benefit (ESRD).

Chemotherapy episodes are constructed using Medicare claims data, specifically physician-administered chemotherapy claims from Part B and Outpatient files (linked to a cancer diagnosis on the claim) and prescription fills for chemotherapy agents from the Part D event file.[5] To ensure alignment between Part D claims and active treatment,

[4] OCM was in operation from July 2016-December 2022, we applied the episode identification methodology throughout our study period.

[5] A full list of all Part B drugs can be found in Appendix Table D1. National Drug Codes (NDC) are available upon request, due to the significant number of codes used for drug identification.

prescription fills are included only if a corresponding Part B claim with a cancer diagnosis occurred within the prior 59 days. Using this approach, we define 180-day chemotherapy episodes starting from the initial chemotherapy claim. Each episode is then assigned to the medical oncologist who handled the plurality of evaluation and management (E&M) office visits during the episode. The cancer type for each episode is determined based on the plurality of cancer diagnoses from office visits within the episode (see Appendix Table D2). To account for the look back period and episode definitions, we utilized claims data from 2007 to 2021, restricting our final analysis sample to chemotherapy episodes initiated between 2008 and 2020.

3.2 Definition of Subspecialized Oncologists

To define whether a chemotherapy episode is coordinated by a subspecialized oncologist, we classify subspecialists as oncologists who provide at least 80 percent of their chemotherapy episodes within a single cancer category in a given year. The 80 percent threshold was chosen to reflect a balance between capturing clinicians whose work is dominated by a single cancer type or set of related cancer types while allowing for the reality that many oncologists have to treat common cancers for financial and clinical reasons despite their subspecialty. In additional analyses below, we also examine a continuous approach to defining specialization. Our dataset includes chemotherapy episodes for cancers split into 9 broad categories: breast cancer, gastrointestinal (GI) cancers, gynecologic cancers, head and neck cancer, hematologic cancers, prostate/genitourinary cancer, melanoma, thoracic cancer, and other cancers. This analysis focuses on five of these groups (breast cancer, GI cancer, hematologic cancers, prostate/genitourinary cancer, and thoracic cancers) for which systemic therapy is available and is typically coordinated by medical oncologists and together account for 90 percent of all identified episodes. We exclude head and neck, skin, gynecologic, and other cancers for two reasons: first, many of these cancers have care that is traditionally led by surgeons, not medical oncologists, particularly gynecologic and head/neck cancers. Second, the excluded groups have small sample sizes, which makes it difficult to define distinct subspecialties using a volume-based threshold. Under this classification, an oncologist is considered a breast cancer subspecialist if at least 80 percent of their chemotherapy episodes in a given year involve breast cancer episodes (Karadakic et al., 2025). The same 80 percent threshold applies analogously to oncologists specializing in the remaining cancer categories.

Our primary measure of access to a subspecialized oncologist of the relevant cancer type is an indicator for whether a beneficiary had any office visit with a subspecialized

oncologist of the relevant cancer type during the calendar year in which chemotherapy was initiated. As a secondary definition, we also consider whether the chemotherapy episode was managed by a subspecialist of the relevant cancer type. While these measures are highly correlated, they capture slightly different aspects of subspecialty involvement in care.

To illustrate the geographic distribution of subspecialist utilization, Figure 2 plots the share of chemotherapy episodes managed by subspecialized oncologists across Hospital Referral Regions (HRRs) in 2008 and 2020. Subspecialist-managed care is disproportionately concentrated in large metropolitan areas, particularly in the Northeast, Southwest, and other major urban centers. The figure highlights both the spatial clustering of subspecialist care and how its prevalence has expanded over time.

3.3 Main Sample and Variable Definitions

The sample of chemotherapy episodes includes beneficiary identifiers, the date of the initiating E&M claim for chemotherapy, the primary cancer type (see Table D2), whether the initial chemotherapy agent was administered via infusion or oral drugs, and the National Provider Identifier (NPI) of the care-coordinating oncologist.

We supplement this episode-level dataset with additional information from Medicare claims data and external sources. First, we incorporate beneficiary information including age, zip code, date of death, sex, and race—from the Master Beneficiary Summary File, along with binary indicators for all 27 chronic conditions. Furthermore, we construct a measure of predicted mortality using linear probability models trained on beneficiaries not included in our main sample and applied to our analytic sample.[6] Zip codes are converted into ZCTAs using publicly available crosswalks (Audirac, 2024). We also construct annual health care access measures at the ZCTA level using Medicare claims data. Finally, we merge in ZCTA-level population counts and annual median household income from the American Community Survey. Summary statistics of our main variables can be found in Table 1.

To measure spending associated with chemotherapy, we aggregate episode-level expenditures beginning on the date of chemotherapy initiation and continuing through

[6]To construct predicted mortality, we use Medicare claims and enrollment data from 2006–2021 to build a detailed beneficiary-level dataset with demographics, coverage indicators, chronic condition flags, and prior-year utilization measures (e.g., provider visits, ER use, hospitalizations). After addressing missingness through additional binary indicators equal to one if a variable is missing, we estimate a linear probability model of death in the current year using beneficiaries not included in our main sample. We then apply this model to all beneficiaries included in our main sample to generate individual-specific predicted mortality scores, capturing underlying health risk without reflecting treatment choices.

180 days post-initiation, or until the beneficiary's date of death if death occurs within that window. Our measure of total spending includes payments made by Medicare, out-of-pocket spending by beneficiaries, and payments from other primary non-Medicare payers. This approach captures a more comprehensive view of the financial burden associated with care, beyond government expenditures alone. We disaggregate spending by Medicare Parts (A, B, and D), by claims source (e.g., Carrier, Outpatient, Inpatient), and further by Restructured-Berenson-Eggers Type of Service Code subcategories to shed light on the underlying components and drivers of spending patterns (CMS, 2024).[7]

To examine other health outcomes and health care utilization, we leverage 100% Medicare samples, extracting enrollment in clinical trials (NCT number) from the Carrier file and constructing acute myocardial infarction, hip fracture, and stroke indicators using diagnostic related group codes from the Inpatient file.[8] We also use Medicare claims data to construct measures of prior healthcare utilization, including hospitalizations, primary care visits, emergency room visits, and cancer screening utilization, providing insight into beneficiaries' healthcare engagement before chemotherapy initiation.

3.4 Differential Distance Measure

The core of the empirical strategy relies on an instrumental variable constructed from the differential distance between a beneficiary's ZCTA and the nearest general oncologist versus the nearest subspecialized oncologist for the relevant cancer type. Because exact residential addresses are not available, we proxy beneficiary location using the centroid of their ZCTA in each year. For oncologists, we use the centroid of their modal ZCTA based on office visits recorded in Medicare Part B claims. Year-specific distance matrices are drawn from the NBER ZIP Code Distance Database (National Bureau of Economic Research, 2025).

$$\mathbf{DD}_{cit} = \text{Dist. Subspecialist}_{ct} - \text{Dist. Generalist}_{t}$$

Formally we define differential distance DD for a beneficiary with cancer type c and ZCTA i in year t as the difference between the nearest subspecialist with the relevant cancer subspecialization (e.g. breast cancer subspecialist for beneficiaries with breast cancer) and the distance to the nearest general oncologist.[9] Due to increases in subspecialization among medical oncologists over time differential distances between subspecialists and

[7] We provide a detailed overview of our spending definitions in Appendix A.

[8] See Appendix Table D3 for details on the definition of these outcomes.

[9] General oncologists are defined as all oncologists who do not manage 80% of cancers within one cancer type or set of related cancer types.

generalists become smaller over time (see Figure D3).

To construct our main sample, we restrict chemotherapy episodes to those with non-negative differential distances, meaning cases where subspecialists are located further away than general oncologists. Additionally, we exclude episodes with distances exceeding the 95th percentile of the annual distribution for either subspecialists or general oncologists.[10] This restriction avoids drawing inferences from outlier observations in geographically remote locations. After applying these criteria, our final sample retains 90% of the original chemotherapy episodes, encompassing 6.1 million unique chemotherapy episodes, 2.2 million unique beneficiaries treated by 17,679 distinct medical oncologists between the years 2008 to 2020.

Due to the non-linear relationship between the instrument and our measure of subspecialist access we additionally transform our measure of differential distance using the inverse-hyperbolic-sine (IHS) transformation. This transformation is frequently used to approximate the logarithmic transformation in regression models, while simultaneously allowing for negative and zero values of a variable. For cases where the IHS transformations enter regression models on the right hand side of the equation, as in our case, the interpretation of the slope parameter changes slightly particularly for small values of the explanatory variable (Bellemare and Wichman, 2020). We therefore define our instrument as follows:

$$z = \sinh^{-1}(x) = \ln\left(x + \sqrt{x^2 + 1}\right)$$

This transformation ensures that we are able to capture the non-linear relationship between differential distances and access measures while also capturing the significant number of chemotherapy episodes where distances to subspecialists and general oncologists are equivalent. In Figure 3 Panel 3a we present the unadjusted first-stage relationship between our instrument and the probability of having any office visit with a subspecialized oncologist of the relevant cancer type within the same year as chemotherapy initiation. Several aspects stand out, first, with increasing differential distance between subspecialists and general oncologists the share of episodes where the beneficiary has seen any subspecialized oncologist of the relevant cancer type declines. Second, the probability of having any office visit with a relevant subspecialist increases over time, reflecting the increasing subspecialization of medical oncologists in the US. Third, the negative relationship between differential distance and access to subspecialized oncologists is mostly increasing over time.[11] Notably, the instrument and a binary indicator of

[10] Only 118,527 chemotherapy episodes are assigned negative differential distances.

[11] In 2020 the negative relationship between the instrument and access to subspecialists attenuates

having a subspecialized oncologists of the relevant cancer type as the care coordinating oncologist is also highly correlated and extremely similar in magnitude to the first stage relationship between our main access measure and the instrument (see Figure 3 Panel 3b).

4 Empirical Strategy

The goal of our empirical analysis is to understand the effect of access to subspecialized oncologists on patient health outcomes and spending. Due to differential access and selection of patients to more specialized medical oncologists it is not possible to simply compare individuals who have access to subspecialized oncologists versus those who do not. Evidence on the benefits of access to highly specialized physicians mainly comes from surgical specialties, where there is a clear relationship between increased volume of a surgical procedure and patient outcomes (Halm, Lee and Chassin, 2002; Birkmeyer et al., 2002; Sahni et al., 2016; Avdic, Lundborg and Vikström, 2019). For medical oncologists there is only limited evidence on the effects of specialized oncologists on patient outcomes, with none of the studies significantly addressing selection effects with respect to access to subspecialized oncologists (see Shanafelt et al. (2012); Davidoff et al. (2020); Caswell-Jin et al. (2025)). However, due to the geographic concentration of subspecialized oncologists and the resulting differences in patient populations treated by subspecialized versus general medical oncologists, simple selection on observable strategies will not disentangle causal effects from adjusted associations.

Our approach to estimating the impact of access to subspecialized oncologists leverages variation in patients' exposure to subspecialized care based on their geographic location and the timing of chemotherapy initiation among cancer patients. We focus on patients undergoing chemotherapy because this group represents the "marginal" population of particular interest to policymakers due to their cost and high clinical risk. Chemotherapy is also central to the clinical implications of receiving specialized versus general oncologic care.

The empirical design in this study uses a distance-based instrument which has been employed in a variety of studies in the health economics literature (McClellan, McNeil and Newhouse, 1994; Card, Fenizia and Silver, 2023; Gruber et al., 2025). One of the main concerns with distance based instruments is the endogeneity of provider location. The distance of more subspecialized oncologists might be associated with patient char-

slightly towards zero compared to the year 2019. We attribute this to effects of the COVID-19 pandemic, which made physical access to cancer treatment and oncologic care more difficult (Nogueira et al., 2024).

acteristics that both influence our measure of access and specific health outcomes. For example, patients in more affluent suburban areas might live further away from subspecialized oncologists often located in academic medical centers in downtown areas, while also generally having better health outcomes. We address this issue in two ways. First, we do not solely base our empirical strategy on distance, but instead construct differential distance, a measure that captures the relative ease of access of one oncologist over the other. Second, we augment our instrumental variable strategy by including ZCTA fixed effects, so that we compare individuals in the same ZCTA at different points in time when access to subspecialized oncologists differed.[12] The variation in access therefore results from changes in differential distances within the same ZCTA over time, resulting from oncologists specializing, exiting and entering markets.

To illustrate the variation leveraged in our empirical strategy, Figure 4 presents the standard deviation of the residualized differential distance measure across selected metropolitan areas, aggregated at the Hospital Service Area (HSA) level.[13] We construct the residualized differential distance by regressing our instrument on ZCTA and cancer type-by-year fixed effects. We then standardize the resulting residuals to have mean zero and unit variance, average them by HSA and year, and finally calculate the standard deviation of these yearly HSA-level averages over time. This measure captures the within-HSA temporal variation in access to subspecialized oncologists that underlies our identification strategy.

Panels 4a (Raleigh, NC) and 4b (Dallas, TX) depict metropolitan areas with relatively high within-HSA variation, while Panels 4c (Atlanta, GA) and 4d (Seattle, WA) show areas with moderate variation. In contrast, Panels 4e (Boston, MA) and 4f (New York City, NY) illustrate regions with relatively low within-HSA variation. These maps provide visual intuition for the identifying variation: our empirical strategy relies on changes in access to subspecialized care that result from oncologist entry, exit, or shifts in specialization over time within the same geographic area.

We use two-stage least squares to estimate the effect of subspecialist access on outcomes of beneficiaries. In the first stage we estimate the effect our differential distance instrument on access to subspecialized oncologists:

$$\text{Access}_i = \alpha + \beta \text{DD}_{t(i)z(i)} + \delta X_i + \tau_{t(i)} + \gamma_{z(i)} + \psi \text{D}_{t(i)z(i)} + \varepsilon_i \tag{1}$$

[12] In Appendix Figure D4 we provide plots for the distribution of our instrument before and after residualizing for the relevant ZCTA and cancer type-by-year fixed effects.

[13] While our empirical strategy exploits variation at the ZCTA level, data use restrictions prevent us from displaying beneficiary-level ZCTA visualizations.

for episode i in ZCTA z in cancer type-by-year t, where DD captures the inverse hyperbolic sine of the differential distance between a subspecialized oncologist of the relevant cancer type and a general oncologist at the ZCTA and year level. The vector X_i includes beneficiary demographic and chronic conditions, as well as ZCTA level controls which vary over time. D is a simple distance measure capturing the distance to the nearest oncologist of any kind, $\tau_{t(i)}$ is a cancer type-by-year fixed effect and $\gamma_{z(i)}$ a ZCTA fixed effect.[14] The dependent variable Access_i is a binary indicator variable equal to one if a beneficiary has had any office visit with a subspecialized oncologist of the relevant cancer type during the year in which chemotherapy was initiated.

Next, we estimate the effect of access to a subspecialized oncologist on mortality, spending, clinical trial enrollment and drug use. We estimate:

$$Y_i = \alpha + \beta \widehat{\text{Access}}_i + \delta X_i + \tau_{t(i)} + \gamma_{z(i)} + \psi \text{D}_{t(i)z(i)} + \varepsilon_i \tag{2}$$

where Y_i are different mortality indicators, clinical trial enrollment indicators and other outcome measures.

Using this instrumental variable design allows us to estimate a local average treatment effect (LATE) for a complier subgroup for whom the instrument, differential distance, decreases the probability to have any office visit with a subspecialist of the relevant cancer type. The validity of our instrument necessitates the standard monotonicity and exclusion assumptions. Monotonicity in our setting means that individuals for whom the distance to the subspecialist versus a general oncologist increases the probability of access to a subspecialist weakly decreases. The exclusion restriction in our specific case requires that differential distance to a subspecialist versus a general oncologist only affects spending, mortality and clinical trial enrollment through access to subspecialized oncologists and we discuss this in more detail in Section 5.4.

Table 2 presents a direct test of the relevance condition by reporting first-stage estimates for our primary measure of access to subspecialized oncologists of the relevant cancer type. The first-stage coefficient remains highly significant, even after controlling for our full set of design covariates and fixed effects. Additionally, the first-stage F-statistic is large, indicating that our instrument generates sufficiently strong variation in access to subspecialists (Lee et al., 2022).

[14] We define fixed effects using 46 detailed cancer types, whereas subspecialist classification is based on five broader cancer categories. Table D2 provides an overview of the ICD codes corresponding to each category.

5 Main Results

In this section, we present our main findings on the effects of access to subspecialized oncologists of the relevant cancer type on patient outcomes. We find that access to a subspecialized oncologist significantly reduces mortality in the medium term, but has no significant effect on short-term mortality (under one year). These estimates contrast with the positive mortality effects suggested by structural form OLS estimates, indicating substantial selection effects in determining who receives care from subspecialists. The second part of our main results is comprised of a detailed look at health care spending per chemotherapy episode, where we find significant spending reductions driven by Part B chemotherapy spending. Following our main results on mortality and spending, we examine the characteristics of the complier subgroup and assess the validity of the exclusion restriction.

5.1 Mortality

Our primary health outcome measure is mortality, defined as the time from chemotherapy initiation until the date of death. We construct binary indicators for mortality at various time intervals, setting the variable to one if a patient dies within a given period after starting chemotherapy. These indicators range from 90-day mortality to 1,080-day mortality, in 90-day increments. To estimate the effect of subspecialist access, we apply Equation 2 and plot the 2SLS estimates in Figure 6 Panel 6a. The results indicate no statistically significant mortality effects within the first year after chemotherapy initiation. However, beyond this point, mortality declines steadily. Specifically, access to a subspecialized oncologist for the relevant cancer type reduces 1-year (360-day) mortality by 2 percentage points. Given an average 1-year mortality rate of 17 percent, this corresponds to an 11 percent reduction relative to the mean.

Notably, the mortality reduction continues to grow, reaching 3.7 percentage points at 2 years (720 days) post-initiation before stabilizing. Although the absolute reduction plateaus, baseline mortality continues to rise over time, reducing the relative effect size. To account for this, Figure 6 Panel 6b scales the mortality estimates relative to the population's mean mortality. The results show that relative mortality reductions peak at 12.8 percent for 630-day mortality before gradually declining again. This pattern suggests that while access to subspecialized oncologists mitigates medium-term mortality risk, its impact diminishes over time, likely reflecting the progression of age and underlying disease including comorbidities.

In contrast to the negative mortality effects estimated using 2SLS, the OLS estimates

of the structural form suggest that access to subspecialists is significantly positively correlated with mortality. For example, OLS results indicate that having access to a subspecialized oncologist is associated with a 0.6 percentage point increase in 1-year (360-day) mortality and a 1.4 percentage point increase in 3-year (1,080-day) mortality (see Table 3). This pattern suggests negative selection into treatment, where patients with more severe, complex, or advanced-stage cancers may be more likely to receive care from subspecialized oncologists.

We recognize that tumor characteristics such as cancer stage, grade, histology, and tumor markers are important drivers of cancer outcomes that we cannot measure. Thus, we conduct a series of robustness checks to ensure that our mortality results are not driven by selection into treatment or by the volume of cases treated by the coordinating oncologist within specific cancer types. First, we estimate models using samples that vary by the time window over which mortality is measured (Table D4), and find consistent results across specifications. Second, we control for the care-coordinating oncologist's episode volume across the five main cancer types by including inverse hyperbolic sine–transformed measures of episode volume for each category, and again find that results remain similar (Table D5). Finally, we restrict the sample to only the first chemotherapy episode per beneficiary, and the findings remain robust (Table D6).

Furthermore in Appendix C, we assess whether the observed mortality effects reflect differences in treatment rather than selective entry into chemotherapy.[15] Using a new sample of over 3.2 million initial oncology consultations, we find that patients whose first visit is with a subspecialist are more likely to initiate chemotherapy within 180 days—especially within the first 60 days. Importantly, we detect no mortality differences following the initial visit, either among all patients or among those who never initiate chemotherapy. Restricting the main chemotherapy sample to patients observed at the consultation stage, we recover mortality effects nearly identical to our main results, even after flexibly controlling for the time between consultation and treatment initiation. Taken together, these findings suggest that subspecialists do not selectively treat healthier patients; rather, the observed survival benefits stem from differences in how chemotherapy is delivered.

5.2 Episode Spending

Motivated by the observed mortality benefits of subspecialist access, we next examine its impact on chemotherapy episode spending. We construct comprehensive episode-level

[15] These results are preliminary, and we are actively conducting additional analyses to better understand the role of subspecialist consultation and selection into treatment.

spending measures that include Medicare payments, beneficiary out-of-pocket costs, and payments from non-Medicare primary payers, capturing all spending from chemotherapy initiation through 180 days or until death, if earlier. This approach reflects the total cost of care rather than Medicare spending alone. Appendix A provides details on measure construction and descriptive statistics. Briefly, average episode spending has risen over time, driven largely by increases in Part B and Part D expenditures. The share of Part D spending grew from 9.3% in 2008 to 32% in 2020, highlighting a shift toward oral chemotherapy agents.

Panel A of Table 4 presents estimates from our main 2SLS specification for total spending and its components by Medicare Part. We find that access to subspecialized oncologists reduces total episode spending by approximately $1,247, or 3.5% of the average, though the estimate is not statistically significant. Disaggregating by spending source, we observe no significant effect on Part A or Part D spending. However, Part B spending declines significantly by $1,342—equivalent to a 6.2% reduction relative to the average Part B episode spending—suggesting that subspecialist care may reduce use of high-cost physician-administered services.

We further decompose Part B spending and find that the overall reduction associated with subspecialist access is driven by lower spending in the Carrier file, while Outpatient spending increases (Appendix Table D8).[16] Using the Restructured BETOS classification, we identify chemotherapy agents and injection/infusion services as the primary sources of cost savings (Appendix Table D9). To assess whether reductions stem from high- or low-cost procedures, we classified procedure codes into average episode spending quintiles and aggregated episode-level spending by quintile. Results indicate that the decline in Part B spending is driven almost entirely by reduced use of high-cost drugs per episode (see Appendix Table D10).[17]

5.3 Complier Characteristics

Our instrumental variable estimates identify local average treatment effects (LATEs), capturing the effect of access to a subspecialized oncologist for the subgroup of compliers—patients who are quasi-randomly assigned to a subspecialist due to differential

[16]The higher average hospital outpatient spending associated with subspecialist care is consistent with the fact that many subspecialists practice in hospital-based settings, including NCI-designated cancer centers, and chemotherapy spending for these doctors is included in the hospital outpatient files.

[17]In preliminary analyses, we find that access to subspecialists reduces the use of biologics when lower-cost biosimilars are available. For instance, subspecialist access decreases the use of levoleucovorin in favor of the biosimilar leucovorin, which is commonly co-administered with 5-FU in colorectal cancer. We also observe reduced use of pegfilgrastim and filgrastim, white blood cell growth factors often used prophylactically during chemotherapy.

distance. To characterize these compliers, we follow (Gruber et al., 2025), estimating the first-stage relationship between our instrument and subspecialist access within subgroups defined by age, race, cancer type, and comorbidities. We then compute kappa values, weighting each subgroup's sample share by its first-stage coefficient relative to the full sample, to quantify its contribution to the complier population. Finally, we compare complier characteristics to the overall sample using reweighted means.

Table 5 presents this comparison, showing that compliers tend to be slightly older, less likely to be female, and more likely to be black. On average, they appear healthier based on chronic condition indicators, being less likely to have chronic condition flags for colorectal, endometrial, lung, and breast cancer, but more likely to have a chronic condition flag for prostate cancer.

5.4 Exclusion Restriction

In Section 4, we outlined the conditions necessary for the validity of our instrumental variable approach and for our 2SLS estimates to obtain a LATE interpretation. In our setting, the exclusion restriction requires that mortality—or any other relevant outcome—is affected by our instrument only through its impact on access to a subspecialized oncologist of the relevant cancer type. A broader interpretation, extending to an endogenous variable that is not strictly binary, is that the instrument should influence mortality solely through changes in the level of oncologist specialization available to a beneficiary, we will make use of this interpretation in a later section of this paper.

To assess the validity of this assumption, we provide balancing tests demonstrating that our instrument is uncorrelated with beneficiary characteristics, conditional on controls and fixed effects. Additionally, we show that our instrument is strongly correlated with many beneficiary characteristics in the absence of controls and fixed effects, underscoring the necessity of the conditional independence assumption for identification.

Figure 5 examines the relationship between our instrument and variables falling into three broad categories of beneficiary characteristics: chronic conditions, prior healthcare utilization, and other characteristics. We estimate these associations using both an unadjusted model, which excludes controls and fixed effects (solid purple dots), and an adjusted model (hollow orange dots), which incorporates our full set of controls (excluding chronic conditions and demographics) and fixed effects. The unadjusted results indicate that the instrument is significantly associated with many beneficiary characteristics, highlighting the potential for a violation of the independence assumption and exclusion restriction. However, once we include ZCTA-level controls and fixed effects,

these associations effectively disappear, suggesting that conditional independence holds for the set of observable characteristics presented in Figure 5.[18]

To further strengthen the validity of our exclusion restriction, we conduct two falsification tests. First, for each chemotherapy episode, we randomly reassign differential distances within years and re-estimate our main specification from Equation 2 using 1-year mortality as the outcome. We repeat this randomization 100 times and find that none of the 2SLS estimates are statistically significant at conventional levels (see Appendix Figure D6). The null results arise because randomizing differential distances breaks the first-stage relationship—differential distance becomes uncorrelated with access to a subspecialized oncologist of the relevant cancer type—supporting the idea that our instrument relies on meaningful geographic variation in access.

As a second falsification exercise, we reassign differential distances based on the nearest subspecialist of an unrelated cancer type. For example, we assign breast cancer patients the differential distance to the nearest gastrointestinal cancer subspecialist. We perform this reassignment for multiple cancer type combinations and estimate Equation 2 across different mortality outcomes (see Appendix Figure D5). The logic of this test is that if cancer-type-specific expertise is the key mechanism, differential distances to unrelated subspecialists should not systematically impact mortality. Consistent with this interpretation, we find no evidence of systematic mortality effects when using differential distances to subspecialists of unrelated cancer types.

6 Mechanisms

In this section, we examine potential mechanisms underlying our main mortality findings in greater detail. We separate these into three categories. First, we analyze differences in the utilization of health care during chemotherapy that could potentially be linked to differences in mortality. We particularly focus on the enrollment in clinical trials, the age of chemotherapy drugs used for chemotherapy, the role of end of life care and the importance of the health care provider mix for potential care fragmentation. Second, we analyze the effects of the intensive margin of oncologists' specialization on patient outcomes. Finally, we present additional evidence on health outcomes that should be unaffected by access to subspecialized oncologists, providing further support for the validity of our identification strategy.

[18] We also provide the balancing results as Appendix Table D7.

6.1 Health Care Utilization during Chemotherapy

6.1.1 Clinical Trial Enrollment

One potential mechanism through which subspecialist access improves survival is by facilitating access to novel and potentially life-saving treatments. While clinical trial enrollment alone is unlikely to account for the full mortality effect, it highlights meaningful differences in care utilization between patients with and without access to subspecialists. To assess this, we examine whether beneficiaries enrolled in clinical trials, using Medicare claims data from 2014 onward—when reporting of clinical trial identifier codes (NCTs) became mandatory for covered research services (CMS, 2014). We define clinical trial participation based on the presence of an NCT number on any claim during the year of chemotherapy initiation.

To supplement these administrative data, we incorporate information from ClinicalTrials.gov, focusing on non-observational trials and the primary condition each trial targets. Additionally, we use OpenAI's GPT-4 to classify trials into our broader cancer categories, enabling us to distinguish between overall cancer trial enrollment and enrollment in trials specific to a patient's cancer type. This classification helps assess whether access to subspecialists increases general clinical trial enrollment or selectively improves access to trials that are more directly relevant to a patient's diagnosis. Appendix B provides further details on the classification methodology.

Overall, subspecialized oncologists are more likely to enroll beneficiaries in clinical trials of any type, as shown in Figure 7. However, enrollment in unspecified cancer trials (i.e. trials targeted toward cancer treatment in general) and non-cancer trials remains low for both general and subspecialized oncologists (see Panels 7c and 7d). On average, general oncologists enroll approximately 1 percent of beneficiaries in cancer trials, with an even lower proportion enrolling in cancer trials specific to the patient's cancer type. In contrast, subspecialists enroll beneficiaries at roughly four times this rate (see Panels 7a and 7b).

To assess whether clinical trial enrollment is causally driven by access to subspecialists rather than selection into treatment, we estimate Equation 2, using as an outcome a binary indicator for whether a beneficiary enrolled in a clinical trial within the year of chemotherapy initiation.[19] Table 6 presents 2SLS and OLS estimates on the relationship between access to a subspecialized oncologist of the relevant cancer type and clinical trial enrollment across different trial types. Panel A suggests that access to a subspecialist of

[19]We do not directly test whether the oncologist is responsible for enrolling the beneficiary in a clinical trial, nor do we investigate whether claims with NCT numbers are specifically linked to oncologists coordinating the beneficiary's care.

the relevant cancer type increases enrollment in any cancer trial by 2.2 percentage points, a 116 percent increase relative to the sample mean. The effect is similar in magnitude for cancer-type concordant trials, with an estimated increase of 1.8 percentage points, or 113 percent relative to the mean. Notably, there is no statistically significant difference in enrollment for unspecified cancer trials or non-cancer trials, in contrast to the OLS estimates and raw unadjusted shares presented in Figure 7. While we do not directly test whether trial enrollment drives mortality reductions and few Medicare beneficiaries enroll in clincal trials, these findings reflect a key difference in healthcare utilization between individuals with access to subspecialized oncologists that may contribute modestly to the observed survival benefits of subspecialist care.

6.1.2 Age of Cancer Drugs

To assess the age of cancer drugs used during treatment, we linked FDA approval years—sourced from the NCI SEER Program and matched to relevant HCPCS and NDC codes—to Medicare claims data. We then calculated the average approval age of all chemotherapy drugs administered during the year of treatment initiation. For Part B drugs, we computed a weighted average based on the number of claim lines per HCPCS code; for Part D drugs, we used the number of prescription fills per NDC code as weights.

Due to limitations on the indication of cancer drugs and the respective approval year and the lack of clinical information available for our patient episodes, we are unable to match the FDA approval year specifically to the indication for the patient's cancer type; instead we focus on the approval year of the cancer drug.[20]

Table 7 presents both IV and OLS estimates of the effect of access to a subspecialized oncologist of the relevant cancer type on the average age of chemotherapy drugs used. Column (1) reports results for oral drugs (covered under Medicare Part D), column (2) for physician-administered drugs (covered under Part B), and column (3) combines both. We find no significant effect of subspecialist access on the age of Part D drugs. However, for physician-administered drugs, we observe a marginally statistically significant reduction in drug age at the 10 percent level. Specifically, access to a subspecialized oncologist is associated with a one-year decrease in the average age of the physician-administered drug. The combined measure of drug age across oral or physician-administered drugs also shows a marginally statistically significant reduction of approximately 0.6 years, suggesting that the effect is primarily driven by physician-administered therapies.

[20]The weighting approach does not account for differences in dosage, quantity administered, or intensity of use across drugs, and therefore captures the average approval year based on utilization frequency rather than total drug volume or dosage.

6.1.3 End of Life Care

Chemotherapy patients in our main sample—who are 67 and older with many having multiple chronic conditions—experience high mortality rates. Within one year of initiating chemotherapy, 17.3% of beneficiaries in our main sample have died. This increases to 30% at two years and 38.5% at three years.[21] Given these patterns, the intensity and nature of care at the end of life becomes an important aspect of cancer treatment, with significant implications for both quality of care and healthcare costs (Zeltzer et al., 2023). Treatment choices near the end of life may also reflect broader patterns in clinical decision-making influenced by access to subspecialized oncologists.

To examine this, we construct four measures of end-of-life care intensity, all defined over the last 30 days of life and limited to patients who die during or within 30 days after a chemotherapy episode. Specifically, we define (1) an indicator for any emergency room (ER) visit, (2) an indicator for any intensive care unit (ICU) stay, (3) an indicator for any hospice claim between 30 and 3 days before death, and (4) an indicator for hospice use in the last three days of life. ER and ICU visits are typically considered markers of high-intensity or aggressive care at the end of life (Jang et al., 2015). In contrast, earlier hospice enrollment (30–3 days before death) is generally viewed as a marker of less aggressive, more comfort-focused care. Hospice initiation in the final three days of life, however, is often considered too late to provide meaningful benefit.

To avoid conditioning on death, we construct end-of-life treatment measures for the full sample and interact them with an indicator for death during or within 30 days of chemotherapy.[22] This allows estimation on the full sample, improving power and generalizability. The interpretation changes: effects now reflect how subspecialist access influences both the probability of short-term mortality and, conditional on death, the intensity of end-of-life care.

Table 8 presents estimates for end-of-life outcomes. We find no significant effect of subspecialist access on ER or ICU visits in the last 30 days of life, nor on hospice use in the final 3 days. However, access to subspecialists significantly reduces the likelihood of any hospice claim between 30 and 3 days before death. The magnitude—representing a 29% reduction relative to the mean—suggests a shift away from comfort-focused care and toward slightly more intensive treatment at the end of life.[23]

[21] The age-adjusted death rate in the US in 2015 was only 733 per 100,000 which in comparison is less than one percent of the US population (Xu et al., 2016).

[22] Appendix Table D11 shows that, in the selected sample of decedents, access to subspecialists is associated with a higher likelihood of ICU admission in the last 30 days of life, but does not significantly affect other end-of-life health care utilization.

[23] Detailed spending results also indicate that access to subspecialists significantly reduces hospice-

6.1.4 Provider Mix and Fragmentation

Next, we assess whether access to subspecialized oncologists affects the composition and diversity of providers involved during chemotherapy. Using the Carrier and Outpatient files, we extract all office visits occurring within each beneficiary's chemotherapy episode and construct three episode-level measures: (1) the total number of office visits, (2) the number of unique providers, and (3) the number of unique provider specialties. We estimate the effect of subspecialist access on each outcome using our main instrumental variable strategy (Equation 2).

Results in Table 9 show that subspecialist access has no discernible effect on the number of office visits or the diversity of provider specialties involved. However, it is associated with a modest but statistically significant 4.5% reduction in the number of unique providers relative to the mean.

These findings suggest that subspecialists may deliver care through a more stable and coordinated provider team, without increasing provider diversity or visit frequency. While specialization is often associated with greater care fragmentation, we find no evidence of such an effect. Instead, the modest decline in provider count points to more streamlined care delivery under subspecialist management.

6.2 Degree of Oncologist Specialization

Our binary measure of access to a subspecialized oncologist provides a straightforward way to compare mortality outcomes between patients who receive care from subspecialists and those who do not. This measure captures the effect of access to specialization on mortality at the extensive margin. However, an important question remains: to what extent does the degree of specialization influence patient outcomes?

Rather than simply distinguishing between general oncologists and subspecialists—where subspecialists are defined as those treating at least 80 percent of chemotherapy episodes within a single cancer category—we investigate whether further specialization within a cancer type improves patient outcomes. The underlying hypothesis is that a narrower clinical focus may enhance expertise, leading to greater improvements in mortality. To test this hypothesis we constructed oncologist level Herfindahl-Hirschman Indices (HHI) of chemotherapy episodes of different cancer types, using a more detailed classification of cancer types capturing 46 different categories, as opposed to the 5 broad categories used above. The HHI is defined as

related expenditures during chemotherapy episodes—by an average of $291, corresponding to a 77.6% decline relative to mean hospice spending per episode.

follows:

$$\text{HHI}_i = \sum_{c=1}^{N} s_{ic}^2 \text{ where } s = \frac{\text{e}_{ic}}{\sum_{c=1}^{N} \text{e}_{ic}} \tag{3}$$

where HHI for oncologist i is calculated as the sum of the squared shares of chemotherapy episodes across cancer types. The episode share for cancer type c is defined as the number of chemotherapy episodes e_{ic} for that cancer type, divided by the total number of chemotherapy episodes the oncologist manages in a given year. The HHI ranges from 0 to 1, where higher values indicate greater specialization, meaning a larger share of an oncologist's caseload is concentrated within a single cancer type. We construct this measure separately for each year and each oncologist, using a more granular classification of cancer types than our five main cancer categories used to define subspecialists. The HHI is strongly correlated with subspecialist status. In 2020, at least 70 percent of oncologists in the top three deciles of the HHI distribution were classified as subspecialists, while the top two deciles consisted entirely of subspecialists.[24]

In Table 10, we present 2SLS estimates of Equation 2, replacing our binary access measure with the care-coordinating physician's HHI as the endogenous variable. Under the assumptions of instrument independence, monotonicity, relevance, and exclusion, this specification allows us to estimate the causal effect of increased physician specialization on patient mortality. Previously, we argued that differential distance should affect patient health outcomes only through access to a subspecialized oncologist, and that any alternative pathways would violate the exclusion restriction. In this context, one could argue that the degree of specialization is a downstream consequence of access to a subspecialist, implying that the instrument primarily affects outcomes by influencing our binary access measure, which in turn operates through the care coordinating oncologist's degree of specialization. This interpretation suggests that the degree of physician specialization is an integral mechanism through which subspecialist access impacts patient mortality.

Focusing on Panel A of Table 10, we observe patterns consistent with our main findings—mortality reductions relative to the population mean diminish over time, suggesting that specialization can only lower mortality rates to a certain extent. The causal effect of an increase in the care-coordinating oncologist's HHI is not statistically significant

[24]Appendix Figure D8 illustrates the distribution of oncologists' Herfindahl-Hirschman Index (HHI) values and their association with the subspecialist definition used in this study. As expected, higher HHI deciles—indicating greater concentration in treating a specific cancer type—are associated with a higher proportion of oncologists classified as subspecialists. Notably, some subspecialists also appear in the lower HHI deciles. This is consistent with our definition, which is based on broader cancer categories: an oncologist may qualify as a subspecialist even while treating a mix of detailed cancer types within a broader category.

within the first 180 days but leads to significant mortality reductions thereafter. For instance, a 0.1 increase in HHI reduces 3-year mortality by 1 percentage point, corresponding to a 2.6 percent reduction relative to the mean 3-year mortality rate. For context, the average difference in HHI between a subspecialized and a general oncologist is 0.45 points. Scaling our point estimate using this difference suggests a 4.6 percentage point decline in 3-year mortality, which is slightly larger than the corresponding estimate in Figure 6, Panel 6a, suggesting potential non-linearities in the relationship between HHI and mortality. OLS estimates in Panel B of Table 10 suggest smaller mortality reductions for the same increase in HHI, implying that despite potential negative selection of beneficiaries, the degree of physician specialization remains an important determinant of patient survival.

6.3 Placebo Outcomes

One potential concern is that patients with access to subspecialized oncologists may also have better access to higher-quality clinicians more broadly, which could independently contribute to improved survival. If true, the observed mortality benefits might reflect the overall quality of a patient's care team rather than the specific expertise of subspecialized oncologists. However, prior evidence—such as higher enrollment in cancer-specific clinical trials and improved outcomes among more narrowly focused oncologists—suggests that subspecialist access itself plays a meaningful role. To further test whether our results are driven by cancer-specific expertise rather than broader differences in care quality, we examine placebo outcomes unrelated to oncology care, such as mortality following hip fracture, stroke, and acute myocardial infarction.

If broader care quality is driving our results, we would expect to see improvements in health outcomes beyond mortality. However, because cancer and its treatments have significant systemic effects, particularly on the immune system and overall health of older individuals, there are few outcomes that can be completely isolated from cancer's influence. Given this limitation, we focus on three outcomes that are arguably less directly related to cancer treatment: acute myocardial infarctions (AMI), hip fractures, and strokes in the two years following chemotherapy initiation. These conditions are less likely to be directly influenced by cancer care because (1) myocardial infarctions and strokes are primarily driven by cardiovascular health and pre-existing risk factors rather than oncologic treatment decisions, (2) hip fractures are mostly (but not entirely) related to musculoskeletal health, falls, and osteoporosis, which are not primary concerns in oncologic care, and (3) while some cancer treatments may have secondary effects on car-

diovascular and bone health, these outcomes are generally not the focus of oncologists when managing cancer treatment regimens.

Table 11 presents 2SLS and OLS estimates from our main specification, examining the effect of subspecialist access on binary indicators for acute myocardial infarction, hip fracture, and stroke diagnoses using inpatient claims. Outcomes are measured separately for the first and second years following chemotherapy initiation. Panel A shows that the 2SLS estimates reveal no statistically significant relationship between subspecialist access and any of the placebo outcomes. Panel B reports OLS estimates, which—despite some statistical significance—indicate precisely estimated effects close to zero, suggesting no meaningful association. These results support the interpretation that subspecialist access affects cancer-specific outcomes without influencing unrelated health events.[25]

Taken together, these findings suggest that the overall skill of subspecialists—proxied by access to a subspecialist of the relevant cancer type—does not influence selected health outcomes across multiple organ systems unrelated to cancer. This strengthens the interpretation that mortality reductions are driven by oncologist specialization and expertise in cancer treatment, rather than general physician ability.

7 Conclusion

This paper provides novel causal evidence on the implications of specialization in high-skill professions. Focusing on medical oncology, we demonstrate that access to subspecialized oncologists improves medium- and long-term survival and reduces episode-level spending. Using quasi-exogenous variation in differential distance to subspecialists versus general oncologists, we estimate that access to a cancer-type–matched subspecialist reduces three-year mortality by 8.6% relative to the mean, without affecting non-cancer-related health outcomes—highlighting the value of specialized clinical expertise beyond general physician skill. We also find that subspecialist access lowers chemotherapy episode spending by approximately $1,250, a 3.5% reduction relative to the mean.

Our findings are robust to a range of model specifications and control variables, and we provide additional support for the validity of our instrument through falsification tests. While subspecialists initiate chemotherapy slightly earlier, we find no evidence that they selectively treat patients with lower baseline mortality risk, reinforcing the exogeneity of our identification strategy. Mechanistically, we document that patients with subspecialist access are significantly more likely to enroll in clinical trials—especially

[25] Using alternative definitions based on chronic condition indicators for AMI, hip fracture, and stroke yields similarly null results.

those relevant to their cancer type—and receive chemotherapy regimens composed of newer drugs. We also find suggestive evidence of more intensive end-of-life care, reflected in lower hospice use. Importantly, we do not observe increased fragmentation of care, as measured by provider diversity or visit patterns.

In additional analyses, we show that oncologists with a deeper focus on specific cancer types—as measured by Herfindahl-Hirschman Indices (HHI)—achieve better mortality outcomes, reinforcing the returns to deep clinical focus. At the same time, we find no impact of subspecialist access on unrelated health outcomes such as hip fractures, strokes, or myocardial infarctions, further underscoring the domain-specific nature of their effects.

Despite the benefits of subspecialization, our findings raise important concerns about equitable access subspecilist oncologists. Subspecialists are disproportionately located in higher-income and urban areas, limiting access for rural and underserved populations. This geographic concentration highlights the need to consider how healthcare systems can structure access to specialized expertise while ensuring equity. Overall, our study contributes to broader discussions in economics and health policy on specialization, labor markets, and productivity, offering new empirical evidence on the returns to deep expertise in complex professional services.

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Figures

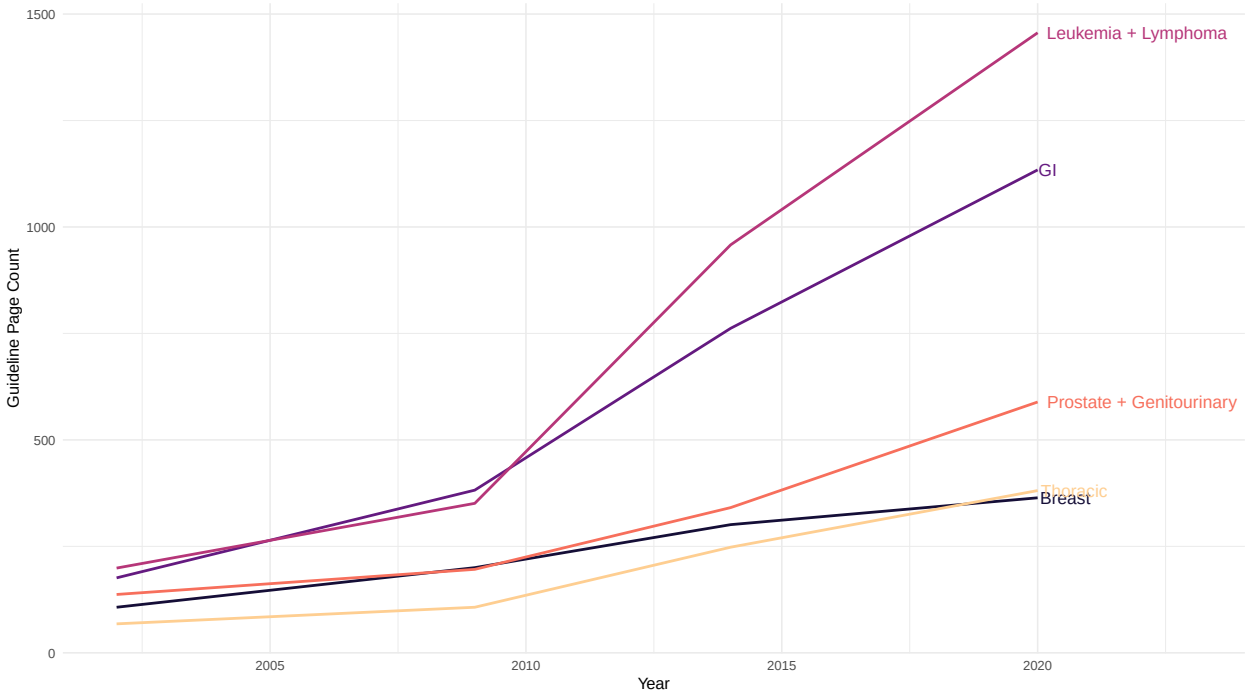
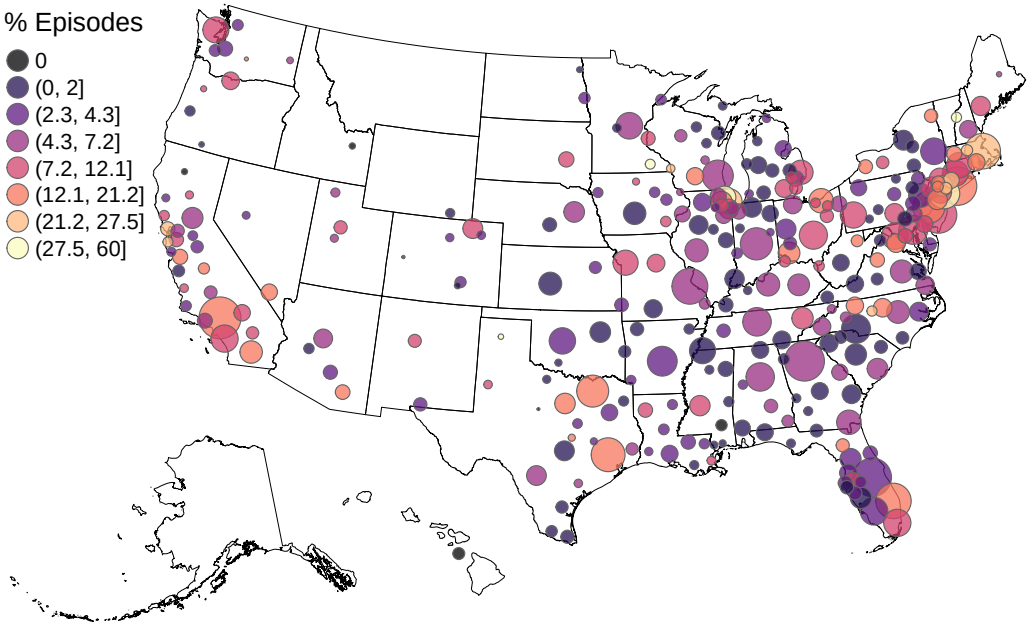
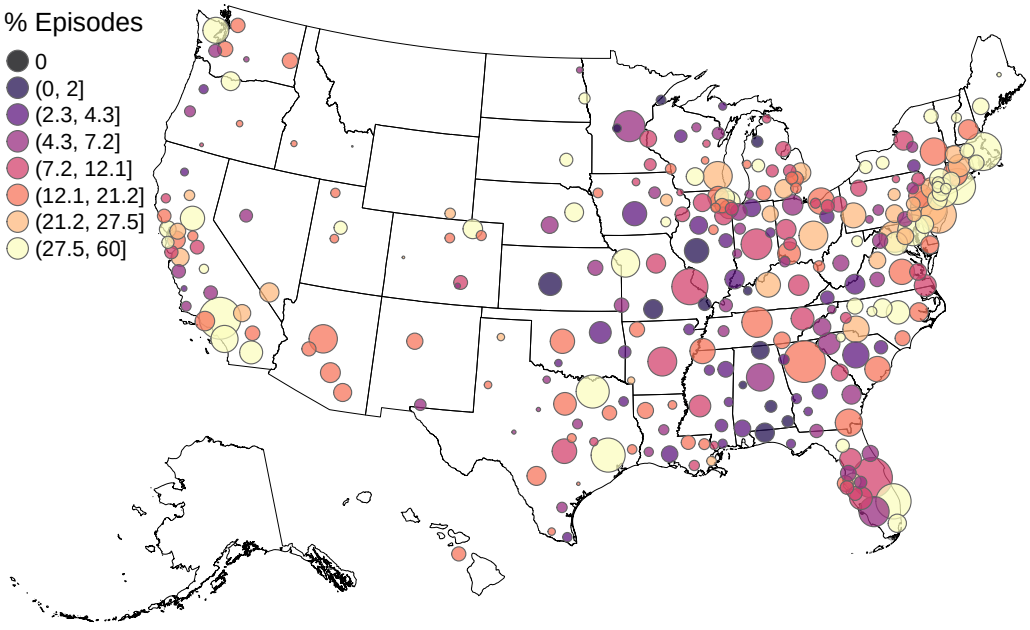


Figure 1: Guideline Page Counts by Cancer Category and Year, 2002 - 2020

Note: This figures shows the number of pages in clinical guidelines provided by the National Comprehensive Cancer Network (NCCN). The guideline page count was separated by the main cancer categories in our sample. Guideline page counts were obtained from Lozinski (2024)



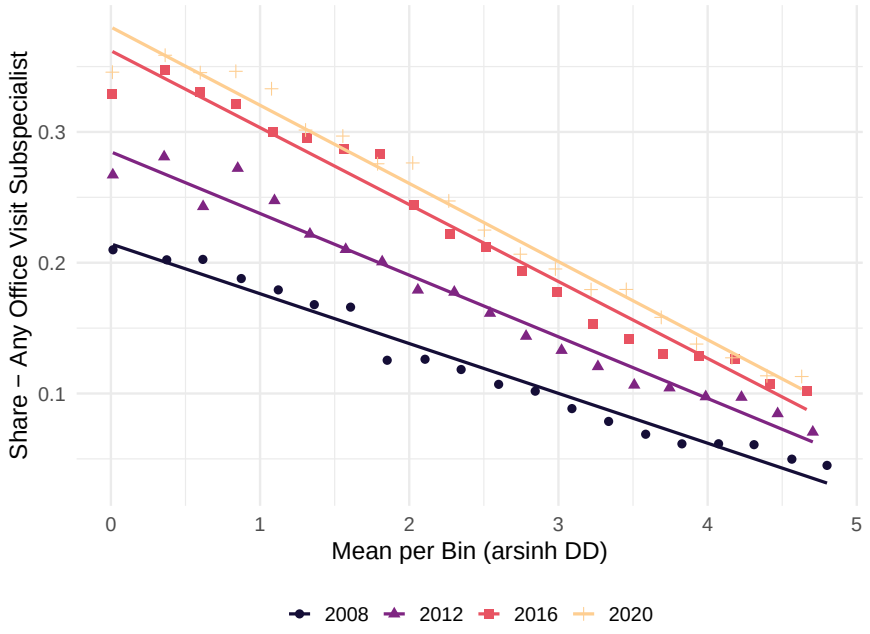
(a) 2008



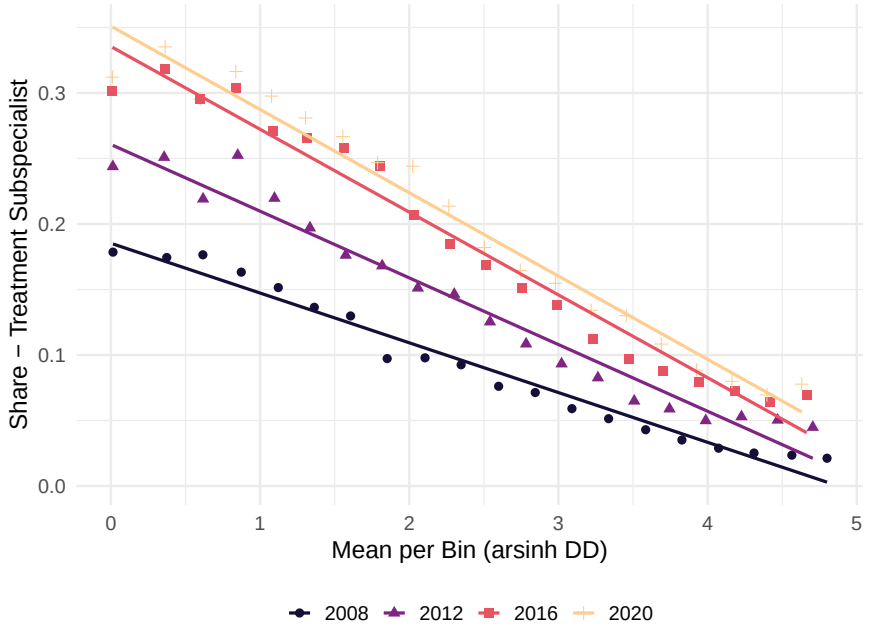
(b) 2020

Figure 2: Utilization of Subspecialized Oncologists by Hospital Referral Region

Note: This figure shows the geographic distribution of chemotherapy episodes by Hospital Referral Region (HRR) for the years 2008 and 2020. Each bubble is positioned at the centroid of the largest polygon within the HRR (based on the beneficiaries location). Bubble size reflects the total number of chemotherapy episodes in the HRR, while bubble color indicates the share of episodes managed by subspecialized oncologists of the relevant cancer type. State borders are included for geographic reference. Due to data output restrictions, we have omitted HRRs with less than eleven chemotherapy episodes managed by subspecialized oncologists.



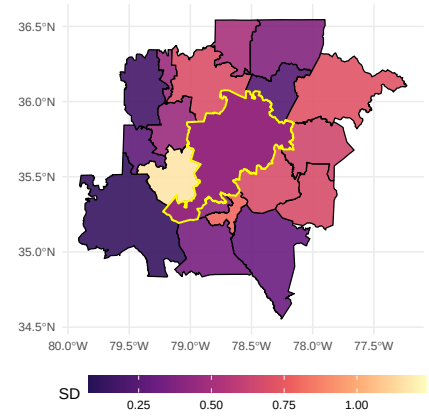
(a) Any Office Visit Subspecialist



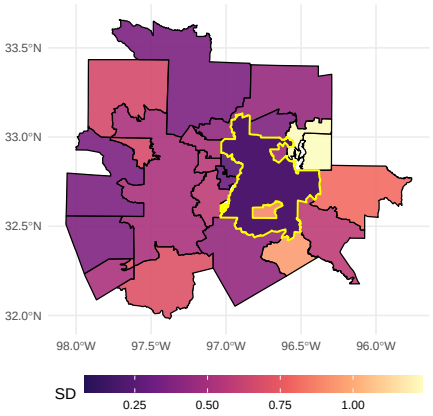
(b) Treated by Subspecialist

Figure 3: First Stage Relationship by Year

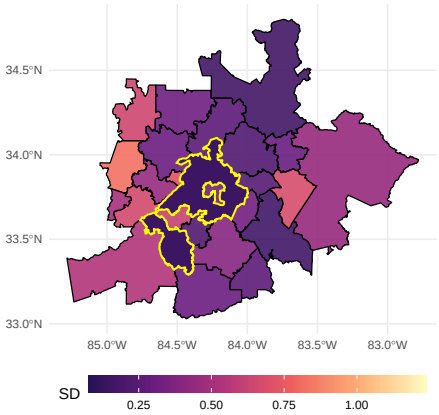
Note: The figure displays a binned scatter plot of the transformed differential distance instrument (x-axis) against measures of access to subspecialized oncologists (y-axis). Different colors represent distinct years, with linear best-fit lines plotted separately for each year. Panel A plots the share of episodes with any office visit with a subspecialized oncologist of the relevant cancer type per bin. Panel B plots the share of episodes where the care coordinating oncologist is a subspecialist of the relevant cancer type.



(a) Raleigh, NC



(b) Dallas, TX



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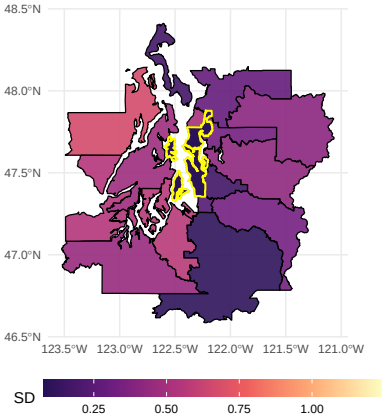
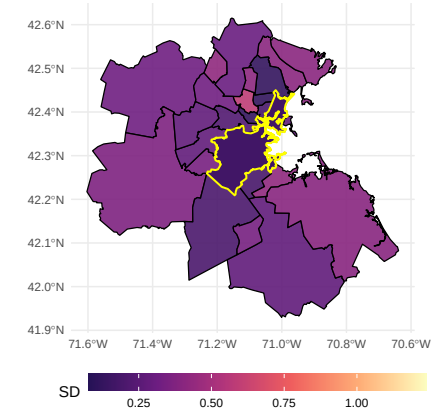
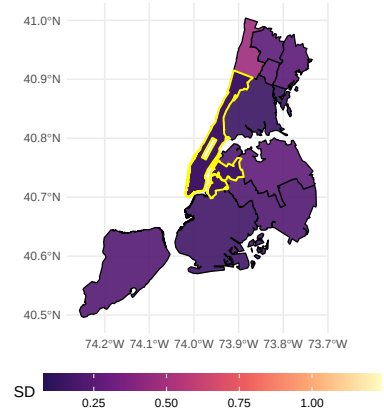


(d) Seattle, WA



(e) Boston, MA



(f) New York City, NY

Figure 4: Variation in Differential Distance across Hospital Service Areas (HSA)

Note: The figure displays the within-HSA standard deviation of our residualized instrumental variable. To construct this measure, we first regress the instrument on cancer type-by-year and ZCTA fixed effects. We then compute the average residual by HSA and year, and calculate the standard deviation of these HSA-level means over time. We have to rely on HSAs for this instead of ZCTAs due to file output restrictions. The maps illustrate geographic variation in this measure across selected metropolitan areas. Panels A and B highlight regions with high, Panels C and D show areas with moderate, and Panels E and F depict areas with low variation. HSAs outlined in yellow represent the core of the respective metropolitan area (e.g. Manhattan for the New York City metropolitan area).

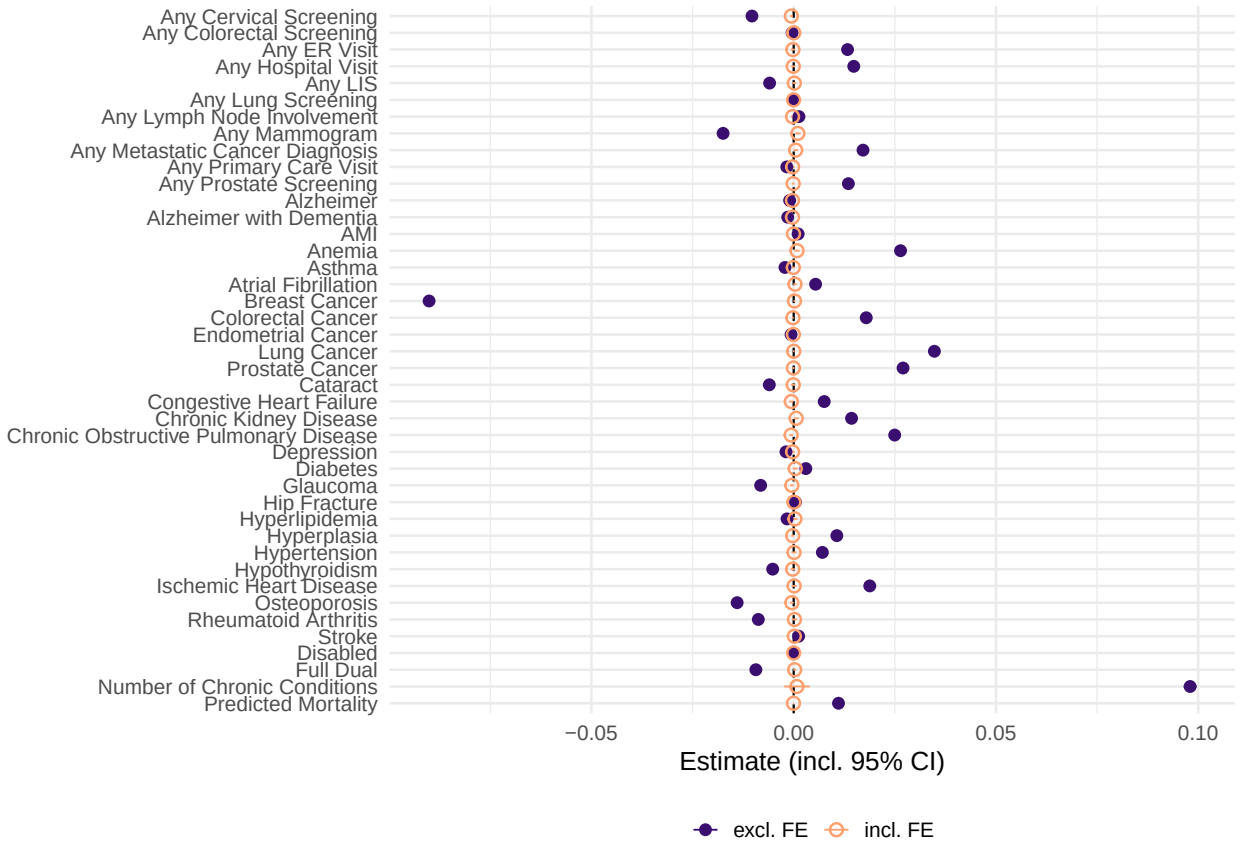
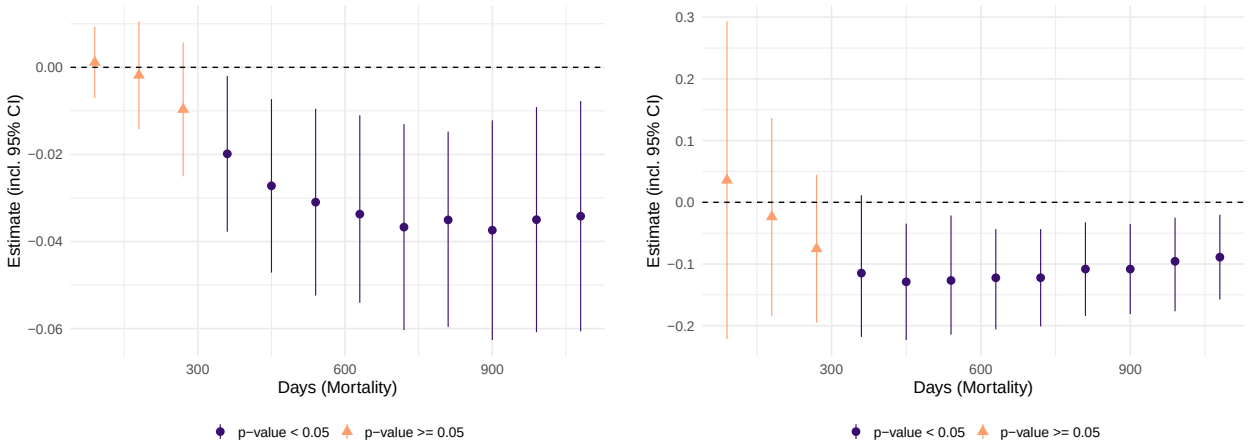


Figure 5: Instrument Balance across Beneficiary Characteristics

Note: This figure displays coefficients from separate regressions of the instrument on the beneficiary characteristics listed on the y-axis. Each point represents a distinct regression. Solid purple dots show unadjusted associations without controls or fixed effects. Hollow orange dots include our full set of ZIP Code Tabulation Area (ZCTA) controls, ZCTA fixed effects, and cancer type-by-year fixed effects. Confidence intervals are based on heteroskedasticity-robust standard errors in unadjusted regressions, and ZCTA-clustered standard errors in the adjusted specifications.

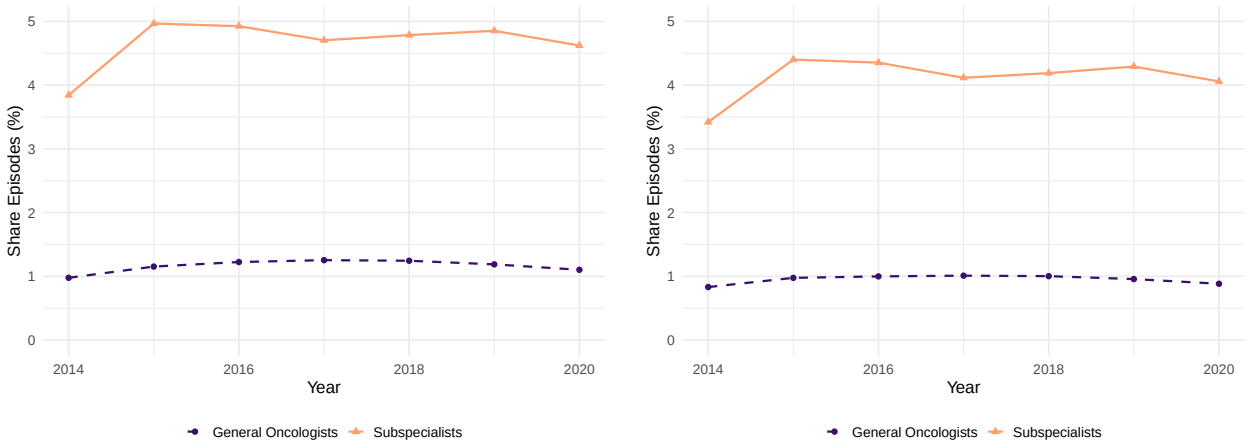


(a) Main Mortality Effect

(b) Relative to Mean Mortality

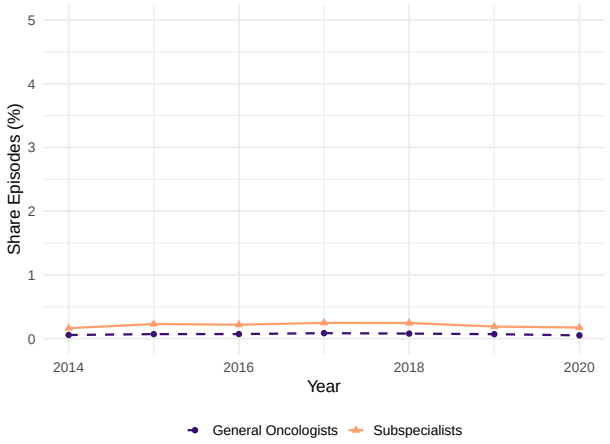
Figure 6: Effect of Subspecialist Access on Mortality

Note: The figure shows the mortality effect of access to a subspecialized oncologists of the relevant cancer type estimated using 2SLS. Each point is estimated using a separate regression. The x-axis represents different mortality measures in quarterly intervals (90-days). The y-axis represents the respective 2SLS estimates including 95% confidence intervals which are constructed using standard errors clustered at the ZCTA level. We used the same sample of all chemotherapy episodes from 2008 to 2017 for estimation.

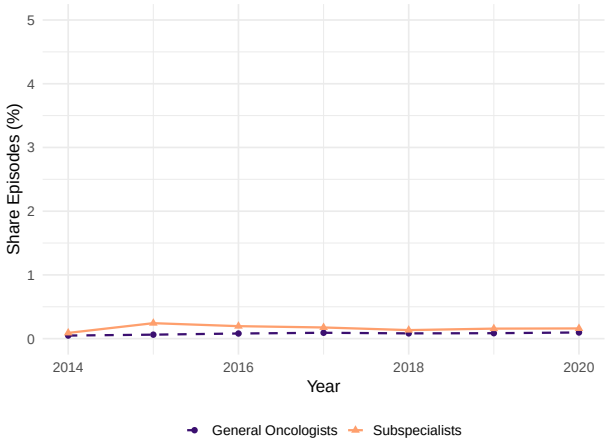


(a) Any Cancer Trial

(b) Concordant Cancer Trial



(c) Unspecified Cancer Trial



(d) Non-Cancer Trial

Figure 7: Share of Episodes with Cancer Trial Enrollment by Trial Type and Oncologist Subspecialization

Note: The figure displays the share of chemotherapy episodes between 2014 and 2020 where the beneficiary had any claim with a NCT number reported in the same year as the chemotherapy was initiated. The solid orange line shows the respective share for episodes coordinated by subspecialized oncologists, while the purple dashed line shows the share of episodes for general oncologists. Panel A reports the share for any cancer trial, Panel B for cancer trials concordant to a beneficiaries cancer type, Panel C the share for cancer trials with unspecified cancers and Panel D shows the share for non-cancer trials.

Tables

Table 1: Summary Statistics of Main Sample

Variable	Mean	SD	Min.	Max.
Panel A: Cancer Characteristics				
Breast Cancer	0.448	0.497	0.000	1.000
GI Cancer	0.102	0.303	0.000	1.000
Leukemia + Lymphoma	0.216	0.412	0.000	1.000
Prostate + Genitourinary Cancer	0.13	0.336	0.000	1.000
Thoracic Cancer	0.103	0.304	0.000	1.000
Panel B: Demographics				
Bene Age	75.977	6.545	67.000	115.000
Female Bene	0.652	0.476	0.000	1.000
Black Bene	0.080	0.271	0.000	1.000
Hispanic Bene	0.011	0.105	0.000	1.000
Asian Bene	0.015	0.121	0.000	1.000
Other non-White Race Bene	0.015	0.122	0.000	1.000
Panel C: Chronic Conditions				
Alzheimer's Disease	0.025	0.156	0.000	1.000
Alzheimer's Disease or Dementia	0.086	0.280	0.000	1.000
Acute Myocardial Infarction (Heart Attack)	0.011	0.104	0.000	1.000
Anemia	0.496	0.500	0.000	1.000
Asthma	0.062	0.241	0.000	1.000
Atrial Fibrillation	0.119	0.324	0.000	1.000
Breast Cancer	0.473	0.499	0.000	1.000
Colorectal Cancer	0.078	0.268	0.000	1.000
Endometrial Cancer	0.007	0.083	0.000	1.000
Lung Cancer	0.127	0.333	0.000	1.000
Prostate Cancer	0.141	0.348	0.000	1.000
Cataracts	0.231	0.421	0.000	1.000
Congestive Heart Failure	0.195	0.396	0.000	1.000
Chronic Kidney Disease	0.305	0.461	0.000	1.000
Chronic Obstructive Pulmonary Disease (COPD)	0.182	0.386	0.000	1.000
Depression	0.187	0.390	0.000	1.000
Diabetes	0.308	0.462	0.000	1.000
Hyperplasia	0.081	0.273	0.000	1.000
Glaucoma	0.116	0.32	0.000	1.000
Hyperlipidemia (High Cholesterol)	0.554	0.497	0.000	1.000
Hypertension (High Blood Pressure)	0.706	0.456	0.000	1.000
Hypothyroidism	0.199	0.399	0.000	1.000
Ischemic Heart Disease	0.364	0.481	0.000	1.000
Osteoporosis	0.129	0.336	0.000	1.000
Rheumatoid Arthritis/Osteoarthritis	0.391	0.488	0.000	1.000
Stroke (TIA)	0.042	0.201	0.000	1.000
Panel C: ZCTA Characteristics				
Distance Closest Oncologist	3.649	3.706	0.000	17.287
Mean Age	71.729	2.148	46.333	97.504
Share FFS	0.712	0.104	0.000	1.000
Share Full Dual	0.103	0.081	0.000	0.943
Share Disabled	0.142	0.071	0.000	0.918
Share Male	0.451	0.029	0.000	1.000
Share Black	0.097	0.167	0.000	1.000
Share Hispanic	0.019	0.044	0.000	0.680
Share Asian	0.02	0.045	0.000	1.000
Share Other non-White Race	0.020	0.029	0.000	1.000
Total Nr. Benes	4,904.062	3,477.523	1.000	43,028.000
Median Household Income	64,139.115	27,461.29	2,499.000	25,0001.000
Total Nr. Providers	109.696	198.904	0.000	7,211.000
Nr. Primary Care Providers	33.329	65.425	0.000	2,147.000
Nr. Mental Health Providers	10.487	20.801	0.000	338.000
Distance Closest Primary Care Provider	0.484	1.291	0.000	16.761

Notes: The table provides summary statistics our main sample of chemotherapy episodes.

Table 2: First Stage Estimates

	Any Office Visit Subspecialist	**Treatement Subspecialist**
$\sinh^{-1}(\text{DD})$	-0.026***	-0.018***
	(0.001)	(0.001)
ZCTA FE	Yes	Yes
Cancer-Year FE	Yes	Yes
Observations	6,144,379	6,144,379
R^2	0.176	0.183
Mean Dep. Var.	0.206	0.163
F-test (1st stage)	22,095.726	13,890.040

Notes: The table provides estimates of the first stage relationship between the inverse hyperbolic sine of the differential distance between a subspecialized oncologist of the relevant cancer type and a general oncologist. Column 1 provides estimates for our main access measure, while column 2 provides estimates of the relationship between the instrument and having a care coordinating oncologist who is a subspecialist of the relevant cancer type. Standard errors are clustered at the ZCTA level. Signif. Codes: ***: 0.01, **: 0.05, *: 0.1.

Table 3: Mortality Effects of Access to Subspecialized Oncologist - Constant Sample

	180-day	**360-day**	**720-day**	**1080-day**
Panel A: 2SLS				
Any Office Visit Subs.	-0.002	-0.020**	-0.037***	-0.034**
	(0.006)	(0.009)	(0.012)	(0.013)
R^2	0.118	0.218	0.292	0.317
F-test (1st stage)	13,911	13,911	13,911	13,911
Observations	4,456,173	4,456,173	4,456,173	4,456,173
Mean Dep. Var.	0.077	0.173	0.300	0.385
Panel B: OLS				
Any Office Visit Subs.	-0.004***	0.006***	0.014***	0.014***
	(0.000)	(0.001)	(0.001)	(0.001)
R^2	0.118	0.218	0.293	0.319
Observations	4,456,173	4,456,173	4,456,173	4,456,173
Mean Dep. Var.	0.077	0.173	0.300	0.385

Notes: The table provides estimates on the effect of access to subspecialized oncologists on various outcomes of mortality for the years 2008 to 2017 of our main sample. Due to the required look forward window when constructing measures of mortality we have fixed the sample to be constant for all measures of mortality. Panel A shows two-stage least squares estimates and Panel B presents the corresponding OLS estimates. All models include demographic, ZCTA level and chronic conditions controls as well fixed effects for the beneficiaries' ZCTA and cancer type by year fixed effects. Standard errors are clustered at the ZCTA level. Signif. Codes: ***: 0.01, **: 0.05, *: 0.1.

Table 4: Access to Subspecialized Oncologist and Spending

	Total	**Part A**	**Part B**	**Part D**
Panel A: 2SLS				
Any Office Visit Subs.	-1,246.52	415.57	-1,341.75**	-320.33
	(876.40)	(294.27)	(675.64)	(572.80)
R^2	0.358	0.164	0.243	0.325
F-test (1st stage)	19,061	19,061	19,061	19,061
Observations	6,144,329	6,144,329	6,144,329	6,144,329
Mean Dep. Var.	35,354.11	5,532.10	21,619.46	8,204.55
Panel B: OLS				
Any Office Visit Subs.	4,715.852***	1,289.665***	2,652.099***	774.088***
	(67.132)	(21.552)	(50.340)	(42.861)
R^2	0.361	0.165	0.246	0.326
Observations	6,144,329	6,144,329	6,144,329	6,144,329
Mean Dep. Var.	35,354.11	5,532.10	21,619.46	8,204.55

Notes: The table provides estimates on the effect of access to subspecialized oncologists on different measures of spending for chemotherapy episodes in our main sample. Column 1 provides estimates for total spending, column 2 Part A spending, column Part B spending and column 4 Part D spending. Panel A shows two-stage least squares estimates and Panel B presents the corresponding OLS estimates. All models include demographic, ZCTA level and chronic conditions controls as well fixed effects for the beneficiaries' ZCTA and cancer type by year fixed effects. Standard errors are clustered at the ZCTA level. Signif. Codes: ***: 0.01, **: 0.05, *: 0.1.

Table 5: Differential Distance IV Complier Characteristics

Variable	Share Among All	Share Among Compliers	Total Obs.	Conditional Obs.
Panel A: Demographics				
Age 67-69	18.289	17.469	6,144,379	1,123,773
Age 70-74	29.478	28.86	6,144,379	1,811,236
Age 75-79	23.868	23.753	6,144,379	1,466,516
Age 80-84	16.316	17.186	6,144,379	1,002,531
Age 85+	12.049	12.658	6,144,379	740,323
Asian Bene	1.484	1.539	6,144,379	91,170
Female Bene	65.172	56.436	6,144,379	4,004,390
Black Bene	7.958	8.669	6,144,379	488,948
Hispanic Bene	1.118	0.824	6,144,379	68,680
Other non-White Race Bene	1.508	1.364	6,144,379	92,640
Panel B: Chronic Conditions Indicators				
Alzheimer's Disease	2.507	2.191	6,144,379	154,055
Alzheimer's Disease or Dementia	8.570	7.828	6,144,379	526,593
Acute Myocardial Infarction (Heart Attack)	1.088	0.995	6,144,379	66,880
Anemia	49.609	45.304	6,144,379	3,048,179
Asthma	6.211	6.094	6,144,379	381,642
Atrial Fibrillation	11.925	11.848	6,144,379	732,715
Breast Cancer	47.275	34.326	6,144,379	2,904,775
Colorectal Cancer	7.783	5.599	6,144,379	478,200
Endometrial Cancer	0.687	0.684	6,144,379	42,216
Lung Cancer	12.698	9.114	6,144,379	780,191
Prostate Cancer	14.110	16.651	6,144,379	866,975
Cataracts	23.095	23.935	6,144,379	1,419,052
Congestive Heart Failure	19.525	17.934	6,144,379	1,199,697
Chronic Kidney Disease	30.530	30.472	6,144,379	1,875,872
Chronic Obstructive Pulmonary Disease (COPD)	18.179	16.981	6,144,379	1,116,957
Depression	18.660	17.922	6,144,379	1,146,528
Diabetes	30.850	29.437	6,144,379	1,895,527
Glaucoma	11.567	12.783	6,144,379	710,690
Hyperlipidemia (High Cholesterol)	55.433	56.433	6,144,379	3,405,997
Hyperplasia	8.105	7.394	6,144,379	498,023
Hypertension (High Blood Pressure)	70.57	70.622	6,144,379	4,336,103
Hypothyroidism	19.866	19.334	6,144,379	1,220,625
Ischemic Heart Disease	36.418	35.488	6,144,379	2,237,663
Osteoporosis	12.949	12.559	6,144,379	795,613
Rheumatoid Arthritis/Osteoarthritis	39.051	38.632	6,144,379	2,399,464
Stroke or Transient Ischemic Attack (TIA)	4.225	4.089	6,144,379	259,615

Notes: The table presents characteristics of the differential distance compliers, in comparison to the overall sample of chemotherapy beneficiaries. For categorical variables the mean in each bin is represented.

Table 6: Clinical Trial Enrollment and Access to Subspecialized Oncologists

	Any Cancer	**Concordant Cancer**	**Unspecified Cancer**	**Non Cancer**
Panel A: 2SLS				
Any Office Visit Subs.	0.022***	0.018**	0.000	0.001
	(0.005)	(0.005)	(0.000)	(0.001)
R^2	0.052	0.048	0.016	0.015
F-test (1st stage)	10,984	10,984	10,984	10,984
Observations	3,737,072	3,737,072	3,737,072	3,737,072
Mean Dep. Var.	0.019	0.016	0.001	0.001
Panel B: OLS				
Any Office Visit Subs.	0.044***	0.038***	0.002***	0.001***
	(0.000)	(0.000)	(0.000)	(0.000)
R^2	0.056	0.051	0.016	0.015
Observations	3,737,072	3,737,072	3,737,072	3,737,072
Mean Dep. Var.	0.019	0.016	0.001	0.001

Notes: The table provides estimates of the effect of access to a subspecialized oncologist of the relevant cancer type on measures of clinical trial enrollment. Estimates for different outcomes are presented in different columns. Column 1 presents results referring to any enrollment in a cancer trial, column 2 any enrollment in a cancer trial of the concordant cancer type, column 3 any enrollment in a cancer trial where cancer types are not clearly specified and column 4 shows results for non cancer trials. Panel A presents 2SLS estimates and Panel B presents results for simple OLS estimates. All models include demographic, ZCTA level and chronic conditions controls as well fixed effects for the beneficiaries' ZCTA and cancer type by year fixed effects. Standard errors are clustered at the ZCTA level. Signif. Codes: ***: 0.01, **: 0.05, *: 0.1.

Table 7: Subspecialist Access and Age of Cancer Drugs

	Combined	**Part B**	**Part D**
Panel A: 2SLS			
Any Office Visit Subs.	-0.555*	-1.114*	-0.670
	(0.324)	(0.594)	(0.436)
R^2	0.203	0.187	0.260
F-test (1st stage)	19,043	12,991	10,213
Observations	6,130,968	4,253,503	3,819,076
Mean Dep. Var.	24.795	30.148	23.641
Panel B: OLS			
Any Office Visit Subs.	-0.469***	-0.818***	-0.379***
	(0.022)	(0.036)	(0.027)
R^2	0.203	0.187	0.26
Observations	6,130,968	4,253,503	3,819,076
Mean Dep. Var.	24.795	30.148	23.641

Notes: The table provides estimates on the effect of access to subspecialized oncologists on the average age of cancer drugs used during the year of chemotherapy initiation. Column 1 indicates the effect on the average age of Part B and Part D drugs combined, column 2 provides estimates of the effect on the average age of Part B drugs and column 3 provides estimates for the effect on the average age of Part D drugs. Panel A shows two-stage least squares estimates and Panel B presents the corresponding OLS estimates. All models include demographic, ZCTA level and chronic conditions controls as well fixed effects for the beneficiaries' ZCTA and cancer type by year fixed effects. Standard errors are clustered at the ZCTA level. Signif. Codes: ***: 0.01, **: 0.05, *: 0.1.

Table 8: Subspecialist Access and End of Life Care (30 Days before Death)

	Any ER	**Any ICU**	**Any Hospice (30-3)**	**Any Hospice (3-0)**
Panel A: 2SLS				
Any Office Visit Subs.	0.004	0.001	-0.009**	-0.002
	(0.004)	(0.003)	(0.003)	(0.002)
R^2	0.083	0.055	0.051	0.027
F-test (1st stage)	19,061	19,061	19,061	19,061
Observations	6,144,379	6,144,379	6,144,379	6,144,379
Mean Dep. Var.	0.050	0.026	0.031	0.014
Panel B: OLS				
Any Office Visit Subs.	-0.003***	-0.001***	0.000**	0.000***
	(0.000)	(0.000)	(0.000)	(0.000)
R^2	0.083	0.166	0.051	0.027
Observations	6,144,379	6,144,379	6,144,379	6,144,379
Mean Dep. Var.	0.050	0.026	0.031	0.014

Notes: The table provides estimates on the effect of access to subspecialized oncologists on different measures of end of life care within the last 30 days of a beneficiaries life. Column 1 shows the effect on the probability of emergency room admission, column 2 on the effect of intensive care unit admission, column 3 shows the effect on whether a beneficiary has had a hospice claim within the last 30 to 3 days before death (30-3) and column 4 whether a beneficiary had any claim within the last 3 days of life (3-0). All outcomes additionally include the condition that a person died during an ongoing chemotherapy episode or within 30 days after. Panel A shows two-stage least squares estimates and Panel B presents the corresponding OLS estimates. All models include demographic, ZCTA level and chronic conditions controls as well fixed effects for the beneficiaries' ZCTA and cancer type by year fixed effects. Standard errors are clustered at the ZCTA level. Signif. Codes: ***: 0.01, **: 0.05, *: 0.1.

Table 9: Access to Subspecialized Oncologist and Provider Mix

	Visits	**Unique Providers**	**Unique Specialties**
Panel A: 2SLS			
Any Office Visit Subs.	0.238	-0.198***	-0.017
	(0.203)	(0.064)	(0.050)
R^2	0.266	0.188	0.187
F-test (1st stage)	19,061	19,061	19,061
Observations	6,144,379	6,144,379	6,144,379
Mean Dep. Var.	10.252	4.415	3.942
Panel B: OLS			
Any Office Visit Subs.	0.757***	0.566***	0.354***
	(0.014)	(0.005)	(0.003)
R^2	0.266	0.201	0.192
Observations	6,144,379	6,144,379	6,144,379
Mean Dep. Var.	10.252	4.415	3.942

Notes: The table provides estimates on the effect of access to subspecialized oncologists on different measures relevant for the beneficiary provider mix. All outcomes are defined at the episode level. Column 1 provides estimates for the effect on the number of E&M office visits, column 2 the number of unique provider NPIs, column 3 the number of unique specialties and column 4 the effect on the standard deviation of the time between office visits. Panel A shows two-stage least squares estimates and Panel B presents the corresponding OLS estimates. All models include demographic, ZCTA level and chronic conditions controls as well fixed effects for the beneficiaries' ZCTA and cancer type by year fixed effects. Standard errors are clustered at the ZCTA level. Signif. Codes: ***: 0.01, **: 0.05, *: 0.1.

Table 10: Mortality Effects of Oncologist Specialization

	180-day	**1-Year**	**2-Year**	**3-Year**
Panel A: 2SLS				
HHI	-0.006	-0.065**	-0.114***	-0.103**
	(0.020)	(0.029)	(0.038)	(0.042)
R^2	0.118	0.219	0.293	0.319
F-test (1st stage)	4,775	4,775	4,775	4,775
Observations	4,456,173	4,456,173	4,456,173	4,456,173
Mean Dep. Var.	0.077	0.175	0.303	0.388
Panel B: OLS				
HHI	-0.008***	-0.015***	-0.029***	-0.039***
	(0.001)	(0.001)	(0.001)	(0.002)
R^2	0.118	0.220	0.294	0.319
Observations	4,456,173	4,456,173	4,456,173	4,456,173
Mean Dep. Var.	0.077	0.175	0.303	0.388

Notes: The table provides estimates on the effect of oncologist level cancer type HHIs on various outcomes of mortality for the years 2008 to 2017 of our main sample. Panel A shows two-stage least squares estimates and Panel B presents the corresponding OLS estimates. All models include demographic, ZCTA level and chronic conditions controls as well fixed effects for the beneficiaries' ZCTA and cancer type by year fixed effects. Standard errors are clustered at the ZCTA level. Signif. Codes: ***: 0.01, **: 0.05, *: 0.1.

Table 11: Access to Subspecialists and Placebo Outcomes

	Year + 1			Year + 2		
	AMI	**Hip Fracture**	**Stroke**	**AMI**	**Hip Fracture**	**Stroke**
Panel A: 2SLS						
Any Office Visit Subs.	0.002	-0.002	0.002	0.000	-0.002	0.000
	(0.001)	(0.002)	(0.002)	(0.001)	(0.001)	(0.001)
R^2	0.016	0.011	0.027	0.012	0.009	0.016
F-test (1st stage)	19,061	19,061	19,061	19,061	19,061	19,061
Observations	6,144,379	6,144,379	6,144,379	6,144,379	6,144,379	6,144,379
Mean Dep. Var.	0.003	0.004	0.004	0.002	0.003	0.003
Panel B: OLS						
Any Office Visit Subs.	0.000***	0.000***	0.000***	0.000***	0.000***	0.000***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
R^2	0.017	0.011	0.028	0.012	0.009	0.016
Observations	6,144,379	6,144,379	6,144,379	6,144,379	6,144,379	6,144,379
Mean Dep. Var.	0.003	0.004	0.004	0.002	0.003	0.003

Notes: The table provides estimates on the effect of access to subspecialized oncologists of the relevant cancer type on binary indicators for having any diagnosis for acute myocardial infarction (AMI), hip fracture and stroke in the first year (Year + 1) and second year (Year + 2) after chemotherapy initiation in the Part A Inpatient file. Panel A shows two-stage least squares estimates and Panel B presents the corresponding OLS estimates. All models include demographic, ZCTA level and chronic conditions controls as well fixed effects for the beneficiaries' ZCTA and cancer type by year fixed effects. Standard errors are clustered at the ZCTA level. Signif. Codes: ***: 0.01, **: 0.05, *: 0.1.

8 Appendix

A Spending Definitions

We construct detailed measures of healthcare spending using individual claims from Medicare Parts A, B, and D. This includes inpatient, hospice, home health, and skilled nursing facility (SNF) claims from Part A; carrier, outpatient, and durable medical equipment (DME) claims from Part B; and prescription drug claims from Part D. For each claims file, we identify relevant records and associated payment variables, classifying payments into three categories: (1) Medicare payments, (2) beneficiary out-of-pocket payments, and (3) payments by non-Medicare primary payers. These spending amounts are aggregated by date at the beneficiary level to allow for analysis over defined time periods.

Episode-level spending is defined as the total spending from the date of chemotherapy initiation through 180 days post-initiation, or until the date of death if the beneficiary dies within that period. For each claims file, we select relevant variables and exclude records (at the claim or line level) that do not contribute to healthcare spending. Spending is calculated separately for each file and disaggregated by payer type (Medicare, beneficiary, or other primary payer). A full overview of variable definitions and selection criteria is provided in Table A2. In order to better understand drivers of spending we also constructed episode level spending of different RBCS subcategories (CMS, 2024).

Table A1: Spending Measures Comparison with Prior Literature

	Keating et al. (2021)	**Main Sample**
Overall	30,946	37,630
Part A	5,966	5,502
Part B	18,503	22,009
Part D	7,794	10,200

Notes: The table provides the average spending for chemotherapy episodes as defined by the Oncology Care Model (OCM) in USD. Column 1 provides spending measures obtained from Table 2 Keating et al. (2021), where we averaged the OCM intervention group baseline and intervention columns. In column 2 we provide spending per chemotherapy episode from our main sample for the years 2014 to 2019 corresponding to the time period used in the comparison article.

We benchmark our spending measures against those reported in Keating et al. (2021), who evaluate spending in Oncology Care Model (OCM) participating practices compared to propensity-matched non-OCM practices. While their analysis covers a shorter

time period (2014–2019), their definition of chemotherapy episodes closely aligns with ours. Due to our focus on the largest cancer types and the application of additional sample selection criteria, exact comparability is not expected. However, our spending estimates closely track those reported in Keating et al. (2021), supporting the consistency of our measures. In Table A1, we compare average episode-level spending in our sample between 2014 and 2019 to the OCM benchmarks presented in Table 2 of Keating et al. (2021). We observe slightly higher spending in Medicare Part B and Part D, which we attribute to the inclusion of both beneficiary out-of-pocket payments and non-Medicare primary payer spending in our estimates, as well as differences in the beneficiary populations included.

Next, we provide an overview of episode level spending for all episodes in our main sample. In Figure A1 we show average spending per chemotherapy episode by Medicare Parts A, B and D from 2008 to 2020 in 1,000 USD. Over time spending per episode significantly increases, which is particularly driven by higher Part B and Part D spending.

Figure A1: Spending per Episode by Medicare Part and Year

Note: The figure presents average spending per chemotherapy episode separately for Medicare Part A, B and D for the years 2008 to 2020.

Figure A2 shows the evolving share of total spending accounted for by each part of Medicare over time. In 2008—just two years after the introduction of Medicare Part

D—Part D spending comprised only 9% of total episode-level spending, compared to 69% for Part B and 22% for Part A. By 2020, the share of Part D spending had increased substantially to 32%, while the share of Part B spending declined to 56%, and Part A spending fell to 12%. This shift reflects the growing importance of oral and self-administered drugs in cancer treatment and the evolving structure of Medicare-financed oncology care.

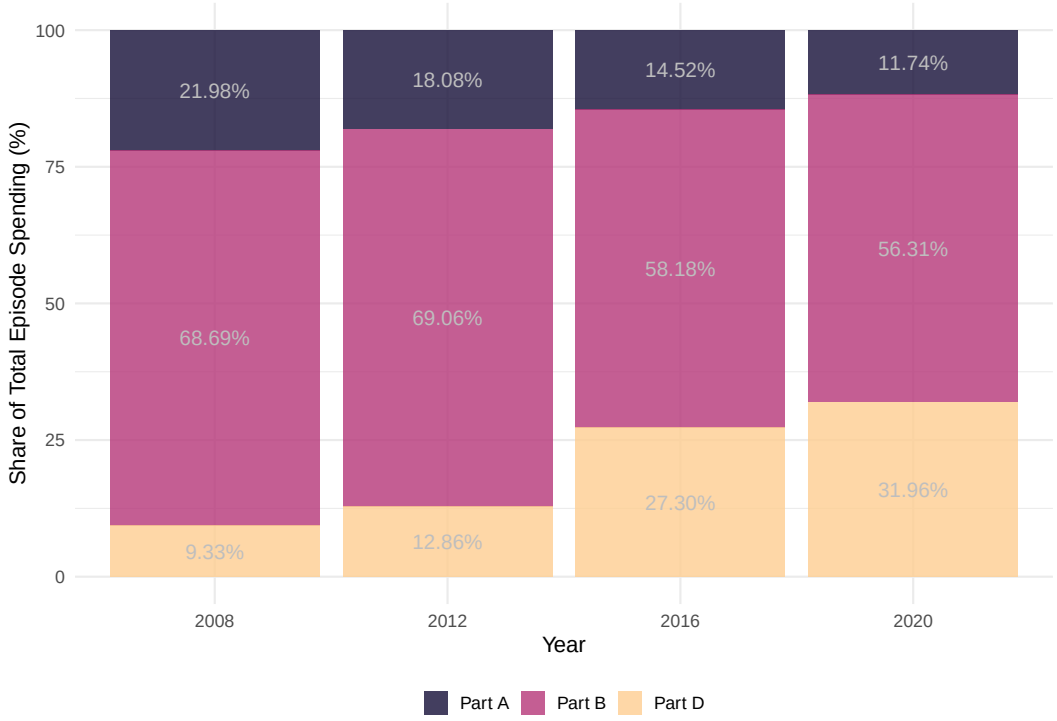


Figure A2: Share of Total Spending by Medicare Part over Time

Note: The figure presents the share of total spending by year by Medicare Part A, B and D for the years 2008, 2012, 2016 and 2020 and includes relevant episodes from our main sample.

Table A2: Variable List and Selection Criteria for Spending Definitions

Label	Variable	Payer Type	Exclude
Carrier			
Carrier Claim Payment Denial Code	CARR_CLM_PMT_DNL_CD		0 or D through Y
Line Processing Indicator Code	LINE_PRCSG_IND_CD		NOT A, R or S
Line NCH Medicare Payment Amt.	LINE_NCH_PMT_AMT	Medicare	
Line Bene. Part B Deductible Amt.	LINE_BENE_PTB_DDCTBL_AMT	Bene.	
Line Bene. Coinsurance Amt.	LINE_COINSRNC_AMT	Bene.	
Line Bene. Part B Deductible Amt.	LINE_BENE_PTB_DDCTBL_AMT	Bene.	
Line Primary Payer (if not Medicare) Paid Amt.	LINE_BENE_PRMRY_PYR_PD_AMT	Primary Payer	
Line Last Expense Date	LINE_LAST_EXPNS_DT		
Durable Medical Equipment (DME)			
Carrier Claim Payment Denial Code	CARR_CLM_PMT_DNL_CD		0 or D through Y
Line Processing Indicator Code	LINE_PRCSG_IND_CD		NOT A, R or S
Line NCH Medicare Payment Amt.	LINE_NCH_PMT_AMT	Medicare	
Line Bene. Part B Deductible Amt.	LINE_BENE_PTB_DDCTBL_AMT	Bene.	
Line Bene. Coinsurance Amt.	LINE_COINSRNC_AMT	Bene.	
Line Primary Payer (if not Medicare) Paid Amt.	LINE_BENE_PRMRY_PYR_PD_AMT	Primary Payer	
Line Last Expense Date	LINE_LAST_EXPNS_DT		
Home Health			
Claim Medicare Non-Payment Reason Code	CLM_MDCR_NON_PMT_RSN_CD		non-blank
Claim Facility Type Code	CLM_FAC_TYPE_CD		4, 5
Claim (Medicare) Payment Amt.	CLM_PMT_AMT	Medicare	
Revenue Center Non-Covered Charge Amt.	REV_CNTR_NCVRD_CHRG_AMT	Bene.	
NCH Primary Payer (if not Medicare) Claim Paid Amt.	NCH_PRMRY_PYR_CLM_PD_AMT	Primary Payer	
Claim Through Date	CLM_THRU_DT		
Hospice			
Claim Medicare Non-Payment Reason Code	CLM_MDCR_NON_PMT_RSN_CD		non-blank
Claim (Medicare) Payment Amt.	CLM_PMT_AMT	Medicare	
Revenue Center Non-Covered Charge Amt.	REV_CNTR_NCVRD_CHRG_AMT	Bene.	
NCH Primary Payer (if not Medicare) Claim Paid Amt.	NCH_PRMRY_PYR_CLM_PD_AMT	Primary Payer	
Claim Through Date	CLM_THRU_DT		
Inpatient			
Claim Medicare Non-Payment Reason Code	CLM_MDCR_NON_PMT_RSN_CD		non-blank
Claim (Medicare) Payment Amt.	CLM_PMT_AMT	Medicare	
Claim PPS Capital Disproportionate Share Amt.	CLM_PPS_CPTL_DSPRPRTNT_SHR_AMT	Medicare	
Claim PPS Capital Indirect Medical Education (IME) Amt.	CLM_PPS_CPTL_IME_AMT	Medicare	
Operating Indirect Medical Education (IME) Amt.	IME_OP_CLM_VAL_AMT	Medicare	
Operating Disproportionate Share (DSH) Amt.	DSH_OP_CLM_VAL_AMT	Medicare	
Revenue Center Non-Covered Charge Amt.	REV_CNTR_NCVRD_CHRG_AMT	Bene.	
NCH Bene. Inpatient (or other Part A) Deductible Amt.	NCH_BENE_IP_DDCTBL_AMT	Bene.	
NCH Primary Payer (if not Medicare) Claim Paid Amt.	NCH_PRMRY_PYR_CLM_PD_AMT	Primary Payer	
Claim Through Date	CLM_THRU_DT		
Outpatient			
Claim Medicare Non-Payment Reason Code	CLM_MDCR_NON_PMT_RSN_CD		non-blank
Claim Facility Type Code	CLM_FAC_TYPE_CD		4, 5
Claim (Medicare) Payment Amt.	CLM_PMT_AMT	Medicare	
NCH Bene. Part B Deductible Amt.	NCH_BENE_PTB_DDCTBL_AMT	Bene.	
NCH Bene. Part B Coinsurance Amt.	NCH_BENE_PTB_COINSRNC_AMT	Bene.	
Revenue Center Non-Covered Charge Amt.	REV_CNTR_NCVRD_CHRG_AMT	Bene.	
NCH Primary Payer (if not Medicare) Claim Paid Amt.	NCH_PRMRY_PYR_CLM_PD_AMT	Primary Payer	
Claim Through Date	CLM_THRU_DT		
Skilled Nursing Facility (SNF)			
Claim Medicare Non-Payment Reason Code	CLM_MDCR_NON_PMT_RSN_CD		non-blank
Claim (Medicare) Payment Amt.	CLM_PMT_AMT	Medicare	
Revenue Center Non-Covered Charge Amt.	REV_CNTR_NCVRD_CHRG_AMT	Bene.	
NCH Primary Payer (if not Medicare) Claim Paid Amt.	NCH_PRMRY_PYR_CLM_PD_AMT	Primary Payer	
Claim Through Date	CLM_THRU_DT		
Part D			
Amt. paid for by Part D low income subsidy	LICS_AMT	Medicare	
Amt. Paid by Patient	PTNT_PAY_AMT	Bene.	
Other True Out-of-Pocket (TrOOP) Amt.	OTHR_TROOP_AMT	Primary Payer	
Reduction in patient liability (PLRO)	PLRO_AMT	Primary Payer	
Amt. paid by Part D plan for the PDE	CVRD_D_PLAN_PD_AMT	Primary Payer	
RX Service Date	SRVC_DT		

Notes: The table provides an overview of the variables and criteria used to construct spending measures for our main analysis from individual claims. The first column describes the variable, the second column provides the variable label as outlined in the Chronic Condition Warehouse (CCW) codebooks for fee-for-service claims and Part D events. The third column indicates which payer is attributed the respective payment variable and column four indicates exclusion criteria for claim and lines for relevant variables.

B Clinical Trial Classification

To classify clinical trials obtained from ClinicalTrials.gov, we utilized OpenAI's GPT-4 API. The model was prompted with the primary condition listed for each trial and tasked with assigning it to one of several predefined cancer categories. The classification process followed these steps:

First, we linked the ClinicalTrials.gov dataset to a list of clinical trial identifiers observed in Medicare claims, obtained from the Virtual Research Data Center (VRDC). This allowed us to subset the data, retaining only trials that were present in Medicare claims. We then extracted key trial characteristics, including study type, study status, conditions, interventions, and trial identifiers, for further processing.

Since some trials list multiple conditions, we focused on the primary condition recorded in the dataset to ensure consistency in classification. We then defined a set of predefined cancer categories: breast cancer, GI cancer, leukemia/lymphoma, skin cancer, head/neck cancer, prostate/genitourinary cancer, thoracic cancer, gynecologic cancer, other cancer, general cancer, and no cancer. With the exception of the last two categories, these classifications align with the definitions used in Table D2.

The general cancer category was used to classify trials that were clearly cancer-related but did not fall into a specific cancer type. For example, some trials listed broad terms such as "neoplasm" or "malignancies" as their primary condition. While these terms indicate a cancer-related trial, they do not provide enough specificity to assign the trial to a distinct cancer category. The no cancer category was used for trials that were entirely unrelated to cancer.

For each classification request, we set the model's temperature to zero to ensure deterministic outputs and limited responses to a maximum of 20 tokens. The API was prompted with the following format:

Label as one of the following: breast cancer, GI cancer, leukemia/lymphoma, skin cancer, head/neck cancer, prostate/genitourinary cancer, thoracic cancer, gynecologic cancer, other cancer, general cancer, no cancer – for medical condition: non-small cell lung cancer.

We manually reviewed a subset of the clinical trials and obtained correct classification for more than 90% of our requests. In the final step, we merged the classification results back into the clinical trial dataset, ensuring each trial was assigned a cancer category for further analysis.

The final share of classified trials which have a corresponding clinical trial number both among beneficiaries in our chemotherapy sample and in clinical trials is presented

in Figure B1. Of the 11,049 classified trials relevant for our sample 24 percent are related to leukemia and lymphoma, 15 percent are not related to cancer, 12 percent are related to other cancers (cancers not covered by our broader categories).

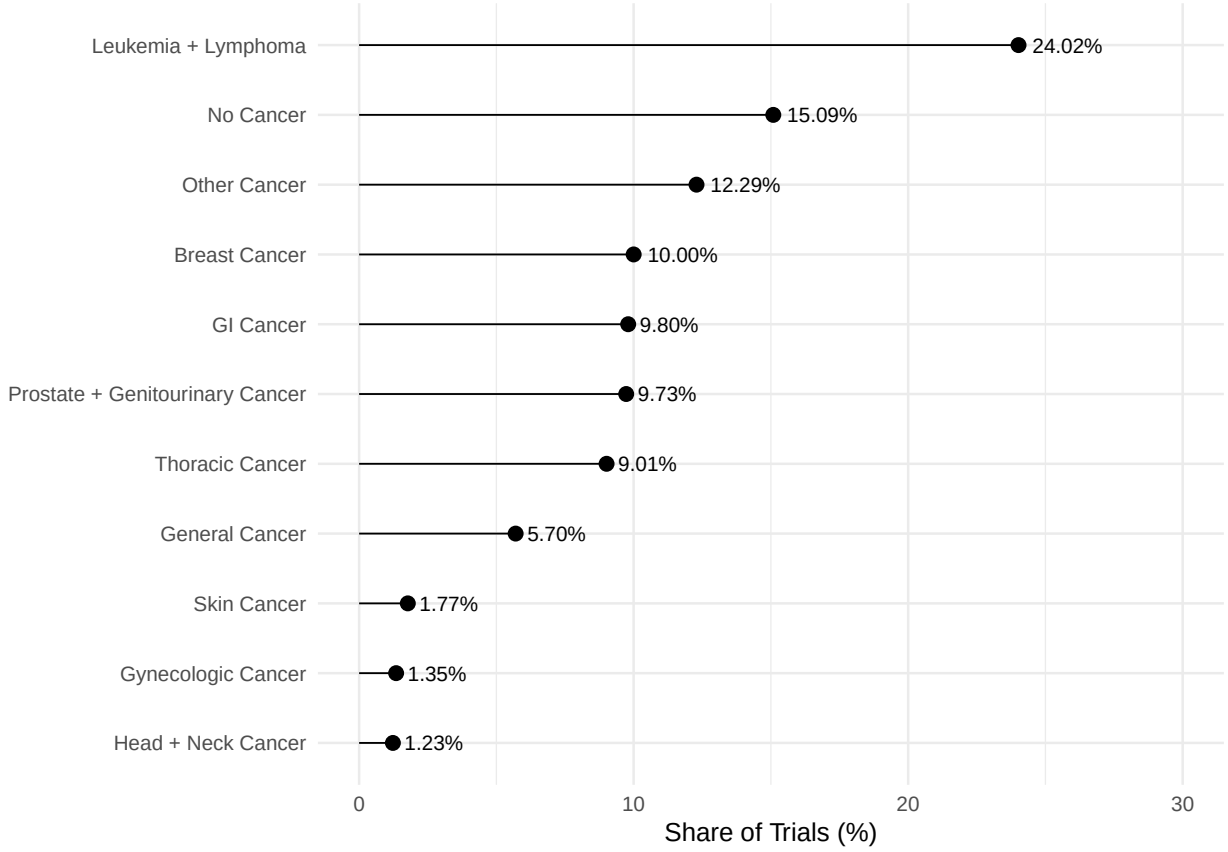


Figure B1: Share of Clinical Trials by Cancer Classification

Note: This figure presents the share of cancer trials within different categories as classified using GTP-4 in combination with data from clinicaltrials.gov. In total there are 11,049 classified trials, which are both linked to a beneficiary in our sample during the year their chemotherapy was initiated and where information is available via clinicaltrials.gov

C Selection into Chemotherapy

Our main analysis focuses on beneficiaries who initiate chemotherapy, as defined by the OCM framework. However, a potential concern is that improved mortality outcomes associated with subspecialist access could reflect selection into chemotherapy rather than differences in treatment quality. For instance, subspecialists may be more selective, initiating treatment only for healthier patients, thereby improving observed survival among those who receive chemotherapy.

To examine this, we construct a new sample capturing the point of initial oncology consultation. Specifically, we identify all new office visits (HCPCS codes 99202–99205) with oncologists in our OCM dataset between 2008 and 2020. This restriction ensures that we can classify oncologists as subspecialists across the full sample. We further restrict to patients with a cancer diagnosis and no initiation start of a new chemotherapy episode in the preceding 12 months, retaining only the first qualifying new office visit per beneficiary. This sample allows us to test whether exposure to a subspecialist at the consultation stage affects the likelihood of receiving chemotherapy. This leaves us with a sample of 3,205,399 new office visits of which 15.3% were with a subspecialized oncologist of the relevant cancer type.[26]

We follow a similar identification strategy as in our main analysis, but specifically we estimate the following equation:

$$\text{Visit}_i = \alpha + \beta \cdot \text{DD}_{t(i)z(i)} + \delta X_i + \tau_{t(i)} + \gamma_{z(i)} + \psi \text{D}_{t(i)z(i)} + \varepsilon_i \tag{4}$$

for visit i in ZCTA z in year by cancer type t, where DD captures the inverse hyperbolic sine of the differential distance between a subspecialized oncologist of the relevant cancer type and a general oncologist at the ZCTA and year level. The vector X_i includes beneficiary demographic and chronic conditions, as well as ZCTA level controls which vary over time. D is a simple distance measure capturing the distance to the nearest oncologist of any kind, $\tau_{t(i)}$ is a cancer type by year fixed effect and $\gamma_{z(i)}$ a ZCTA fixed effect. The dependent variable Visit_i is a binary indicator variable equal to one if a beneficiary had the new office visit with a subspecialized oncologist of the relevant cancer type and zero otherwise.

In a second step, we estimate the effect of initial subspecialist consultation on time to chemotherapy initiation. We estimate:

[26] It is important to recognize that the new office visit sample captures a different subset of beneficiaries than our main chemotherapy sample. As a result, direct comparisons between the two samples should be interpreted with caution.

$$Y_i = \alpha + \beta \cdot \widehat{\text{Visit}}_i + \delta X_i + \tau_{t(i)} + \gamma_{z(i)} + \psi \text{D}_{t(i)z(i)} + \varepsilon_i \tag{5}$$

where Y_i is the outcome variable for example a binary indicator equal to one if chemotherapy was initiated within a specific time windows (e.g. within 30, 60, 90 days after the initial consultation).

We provide evidence that our instrument satisfies both the relevance and independence assumptions in this new office visit sample. Table C1 shows a strong first-stage relationship between the instrument and the likelihood of having a first new office visit with a subspecialized oncologist, with the association remaining robust after controlling for covariates and fixed effects; the large F-statistic confirms sufficient instrument strength (Lee et al., 2022). In support of the independence assumption, Figure D7 demonstrates that the instrument is uncorrelated with observable beneficiary characteristics once we condition on ZCTA and cancer type-by-year fixed effects.

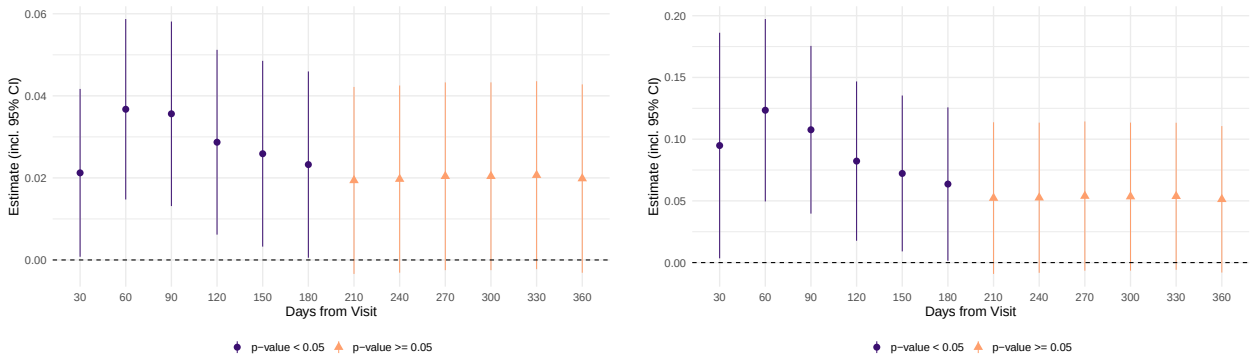
Table C1: First Stage Estimates Selection into Chemotherapy

	New Visit Subspecialist
$\sinh^{-1}(\text{DD})$	-0.027***
	(0.000)
Observations	3,205,399
R^2	0.161
Mean Dep. Var.	0.153
F-test (1st stage)	16,961

Notes: The table provides estimates of the first stage relationship between the inverse hyperbolic sine of the differential distance between a subspecialized oncologist of the relevant cancer type and a general oncologist and the outcome of having a first new office visit with a subspecialist or the relevant cancer type. All models include demographic, ZCTA level and chronic conditions controls as well fixed effects for the beneficiaries' ZCTA and cancer type by year fixed effects. Standard errors are clustered at the ZCTA level. Signif. Codes: ***: 0.01, **: 0.05, *: 0.1.

We estimate Equation 5 using binary indicators for chemotherapy initiation within 30 to 360 days after the first new office visit. As shown in Figure C1, beneficiaries initially seen by a subspecialized oncologist of the relevant cancer type are significantly more likely to start chemotherapy within 180 days, with the largest effect—3.7 percentage points or 12.3% relative to the mean—occurring within 60 days. Differences dissipate after 180 days, suggesting that subspecialists accelerate initiation without affecting

overall treatment rates. Earlier initiation, however, may reflect a clinically meaningful improvement in timeliness of care.



(a) Main Estimates

(b) Relative to Mean

Figure C1: New Subspecialist Office Visit and Time to Chemotherapy Initiation

Note: The figure displays the effect of having a first new office visit with a subspecialized oncologist of the relevant cancer type on the likelihood of initiating chemotherapy, measured at different time intervals following the visit. Estimates are derived from separate 2SLS regressions for each binary outcome. The x-axis indicates whether chemotherapy was initiated within 30, 60 up to 360 days of the initial visit. Panel A presents raw 2SLS point estimates, while Panel B scales these estimates relative to the mean of each respective outcome. The y-axis shows the estimated coefficients with 95% confidence intervals, using standard errors clustered at the ZCTA level. All models control for beneficiary demographics, ZCTA-level characteristics, and chronic conditions, and include fixed effects for ZCTA and cancer type by year.

Using the sample of first new office visits, we examine whether mortality differs based on whether the initial consultation was with a subspecialized oncologist. This analysis tests whether the mortality effects observed in the chemotherapy sample might stem from earlier selection into care. We assess mortality for all new visits between 2008 and 2017, and separately for individuals who never initiate chemotherapy. As shown in Table C2, we find no statistically significant differences in mortality up to three years post-consultation. These null results suggest that subspecialist involvement at the initial visit does not meaningfully influence survival, reinforcing the view that observed mortality benefits emerge during or after chemotherapy.

Table C2: Mortality Effects of New Office Visit with Subspecialist

	180-day	360-day	720-day	1080-day
Panel A: All New Visits (2008 - 2017)				
New Visit Subspecialist	-0.003	-0.015	-0.007	-0.010
	(0.009)	(0.010)	(0.011)	(0.011)
R^2	0.174	0.251	0.307	0.322
F-test (1st stage)	13,464	13,464	13,464	13,464
Observations	2,397,805	2,397,805	2,397,805	2,397,805
Mean Dep. Var.	0.143	0.229	0.335	0.404
Panel B: New Visits without Chemotherapy (2008 - 2017)				
New Visit Subspecialist	-0.008	-0.004	-0.013	-0.014
	(0.012)	(0.013)	(0.014)	(0.014)
R^2	0.249	0.302	0.335	0.344
F-test (1st stage)	8,663	8,663	8,663	8,663
Observations	1,475,609	1,475,609	1,475,609	1,475,609
Mean Dep. Var.	0.185	0.250	0.326	0.383

Notes: The table presents two-stage least squares (2SLS) estimates of the effect of having a first new office visit with a subspecialized oncologist on mortality outcomes between 2008 and 2017. Mortality is measured as a binary indicator of death within a given time frame following the first new office visit, with time to death calculated from the date of that visit. To ensure comparability across specifications, the sample is fixed based on the maximum follow-up window. Panel A reports estimates for the full sample; Panel B restricts to individuals who did not initiate chemotherapy within 360 days of the office visit. All models control for demographics, chronic conditions, and ZCTA-level characteristics, and include fixed effects for ZCTA and cancer type by year. Standard errors are clustered at the ZCTA level. Significance codes: *** p<0.01, ** p<0.05, * p<0.1.

To complete the analysis, we focus on the subset of individuals observed in both the new office visit and chemotherapy samples and re-estimate our main mortality model (Equation 2 from Section 4), measuring time to death from chemotherapy initiation. In this subgroup, we recover mortality effects nearly identical to our main results, suggesting that the observed survival benefit is driven by how chemotherapy is delivered, not by differential selection into treatment. These findings remain robust when we flexibly control for the time between the initial consultation and treatment initiation. Taken together, this evidence indicates that while subspecialists may initiate chemotherapy slightly earlier, they do not select patients with different underlying mortality risk—highlighting the importance of subspecialist-led chemotherapy delivery in improving outcomes.

D Additional Figures and Tables

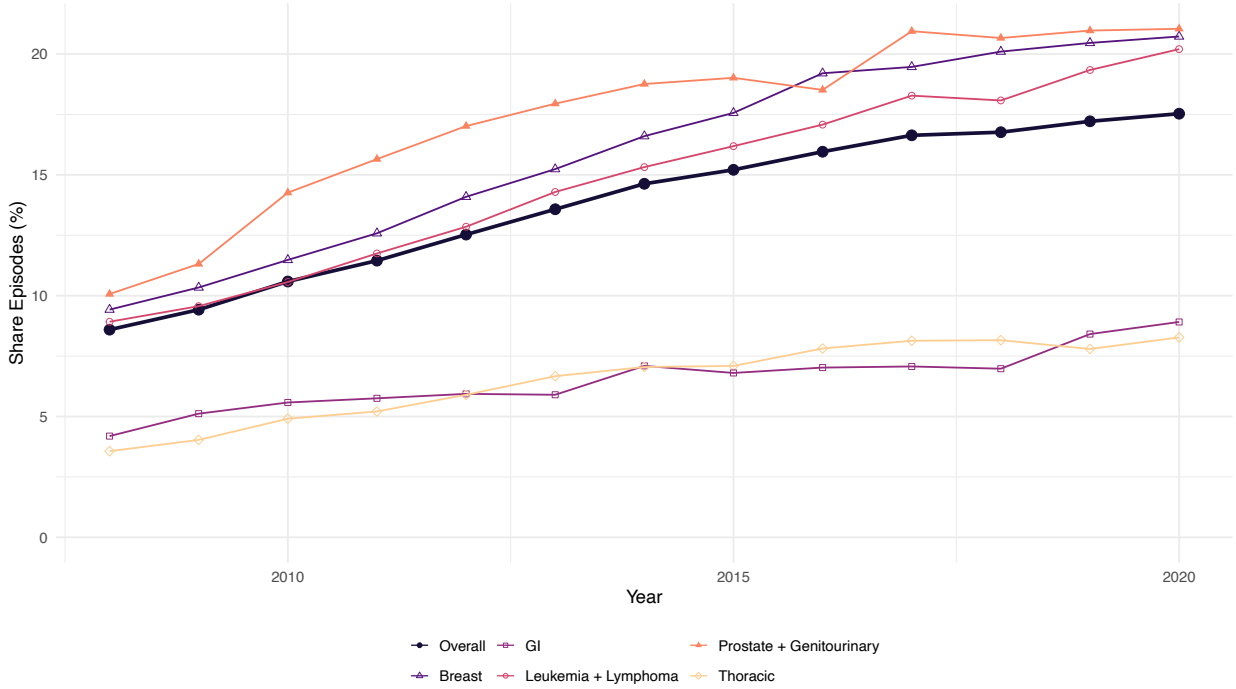


Figure D1: Trends in Subspecialization in Cancer Care 2008 - 2020

Note: This figure presents trends in subspecialization for beneficiary chemotherapy episodes of different cancer types. The figure depicts the share of chemotherapy episodes managed by highly subspecialized oncologists of the relevant cancer type separately by cancer type. The "Overall" group (black line with black dots) indicates the trend in subspecialization for all cancer types combined. Abbreviations: GI=gastrointestinal.

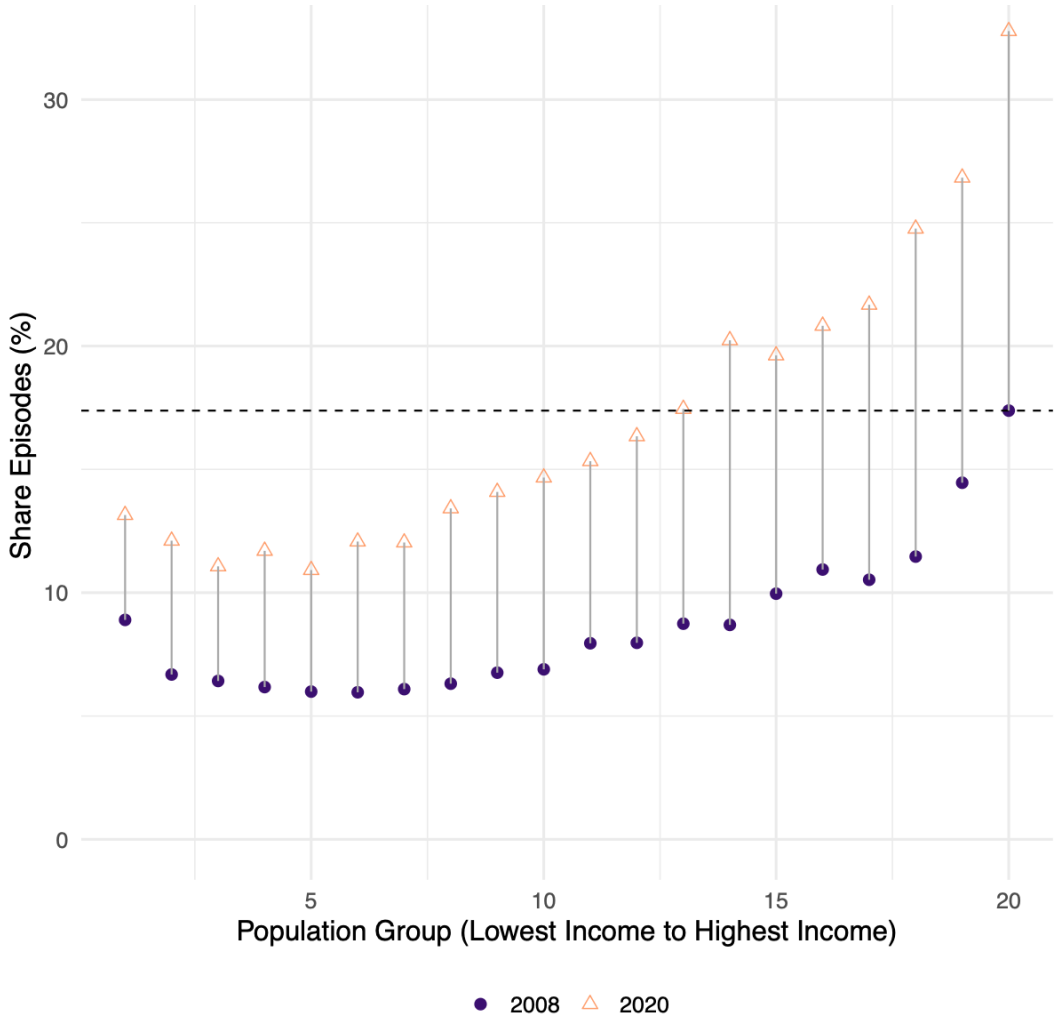


Figure D2: Socioeconomic Gradients in Access to Highly Subspecialized Cancer Care

Note: This figure illustrates the socioeconomic gradients in access to highly subspecialized cancer care, using beneficiary ZCTA and median household income data from the American Community Survey. The figure plots the share of chemotherapy episodes managed by highly subspecialized oncologists on the y-axis, with the x-axis representing population groups (ventiles), ordered from lowest income to highest income, for the years 2008 and 2020. Vertical grey lines indicate differences in access to highly subspecialized cancer care across years for each population group. The horizontal dashed line represents the level of access to highly subspecialized cancer care for the highest income population group in 2008. This figure follows prior work on mortality differences across different areas in the U.S. (Currie and Schwandt, 2016; Schwandt et al., 2021).

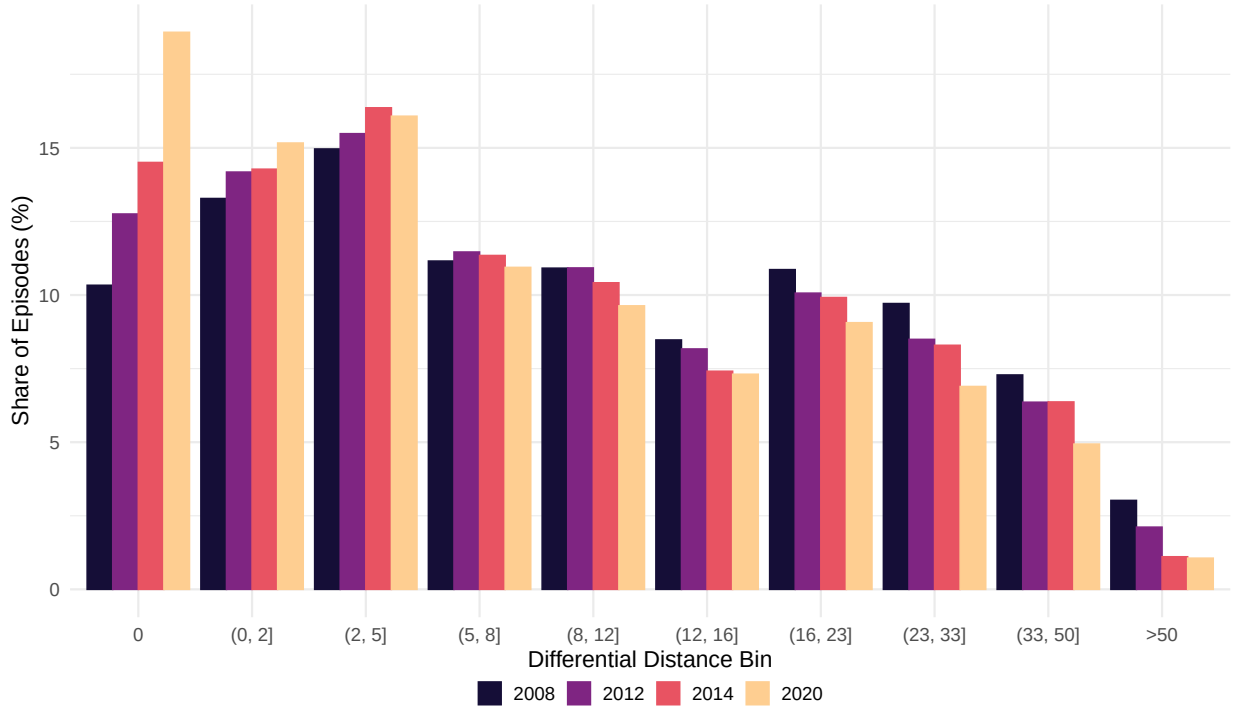
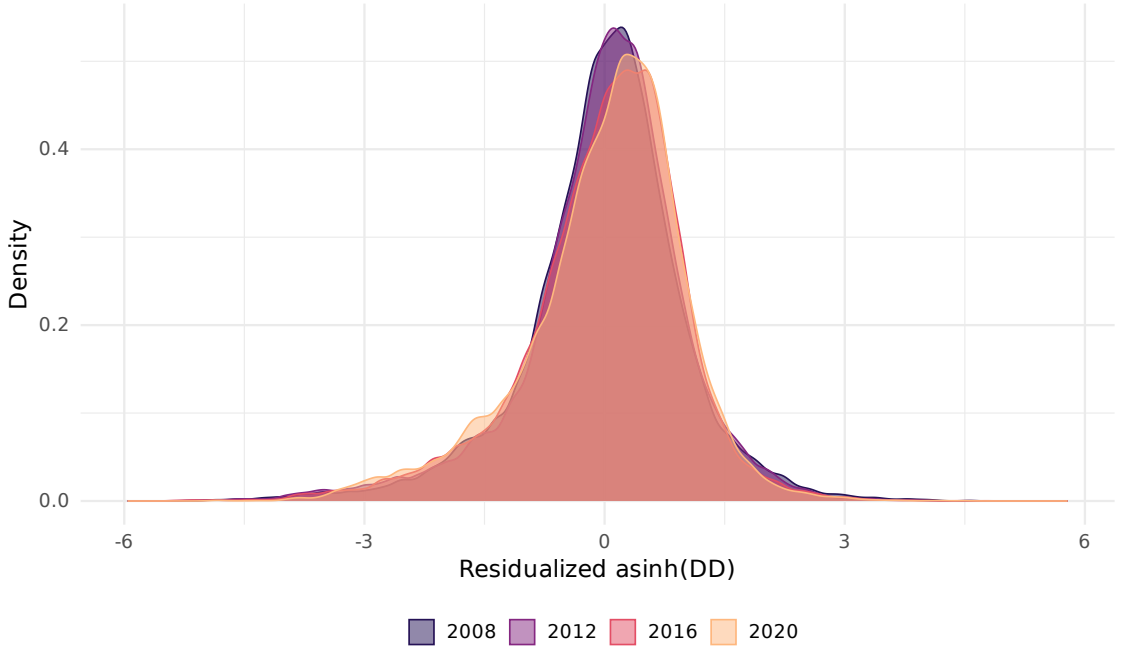


Figure D3: Distribution of Differential Distances over Time

Note: This figure presents the distribution of differential distances for four different years (2008, 2012, 2016, 2020) for our entire main sample. It shows the distribution of differential distances in miles by different bins. Panel B shows the histogram of arcsinh-transformed differential distances across different years.

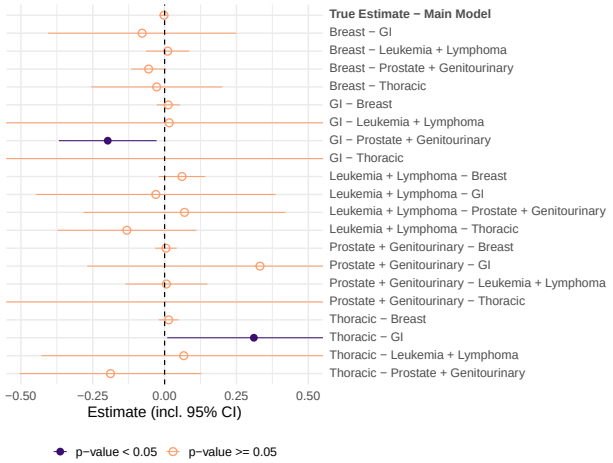
(a) asinh(Differential Distance)



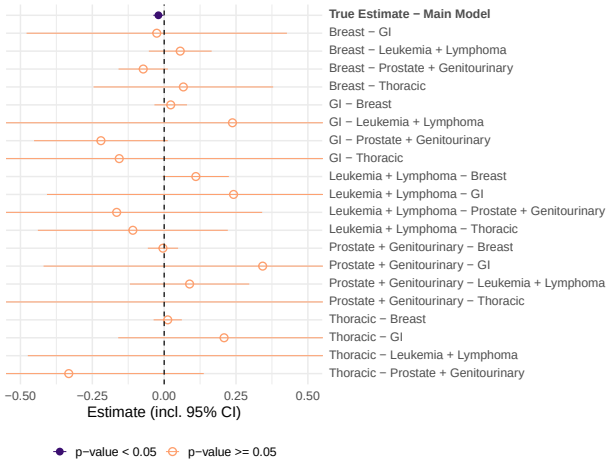
(b) Residualized asinh(Differential Distance)

Figure D4: Distribution of Instrumental Variable

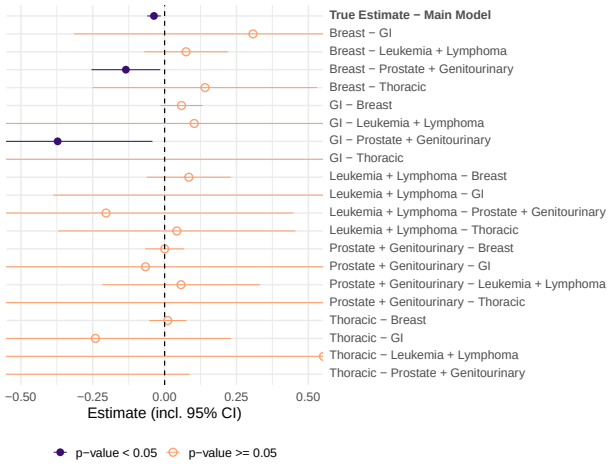
Note: This figure displays the distribution of the instrumental variable—the inverse hyperbolic sine (IHS) of differential distance—for individuals in the main sample. All values are standardized to have a mean of 0 and a standard deviation of 1. Panel A shows kernel density plots of the raw IHS-transformed differential distance for the years 2008, 2012, 2016, and 2020. Panel B shows the corresponding residualized values, obtained by regressing the instrument on cancer type-by-year and ZCTA fixed effects.



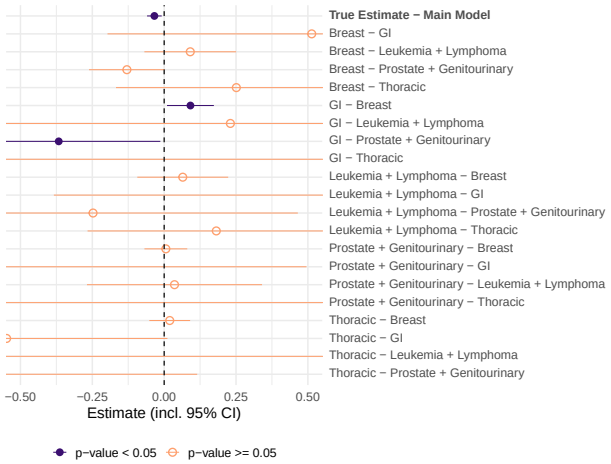
(a) 180-Day Mortality



(b) 1-Year Mortality



(c) 2-Year Mortality



(d) 3-Year Mortality

Figure D5: Falsification Test - Assignment of Distance to Unrelated Subspecialist

Note: This figure presents falsification tests in which beneficiaries are assigned distances to subspecialists for unrelated cancer types (e.g., the distance to the nearest breast cancer subspecialist for a beneficiary with thoracic cancer). Each dot is estimated from a separate regression. Each row on the y-axis represents a specific reassignment of distances; for example, "Breast – GI" means all beneficiaries were assigned the distance to the nearest breast cancer subspecialist, except those with breast cancer, who were assigned the distance to the nearest GI subspecialist. The top row shows the main estimates using the true distance to the nearest subspecialist of the relevant cancer type. Each panel corresponds to one of our four main mortality outcomes. Solid purple circles indicate statistically significant estimates ($p < 0.05$), while hollow orange circles indicate statistically insignificant results ($p \geq 0.05$). The analysis sample includes all chemotherapy episodes from 2008 to 2017. All models control for demographics, ZCTA-level characteristics, and chronic conditions, and include ZCTA fixed effects and cancer type-by-year fixed effects. Standard errors are clustered at the ZCTA level.

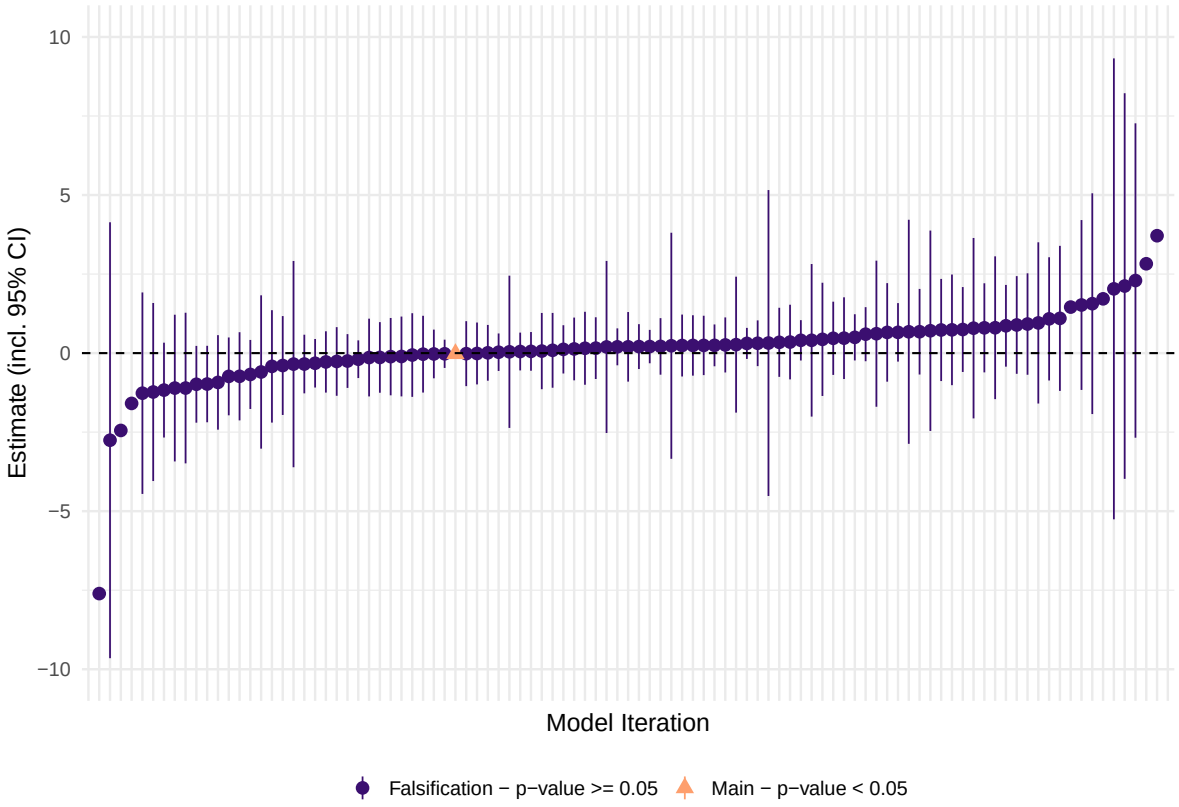


Figure D6: Falsification Test - Randomly Reassign Differential Distances

Note: This figure presents 2SLS estimates of the effect of access to a subspecialized oncologist of the relevant cancer type on 1-year mortality. Each dot represents the result from a separate regression using our main specification, where differential distances were randomly reassigned to chemotherapy episodes within year. This randomization was repeated 100 times. For reference, the figure also includes our main (non-randomized) estimate. All models control for beneficiary demographics, ZCTA-level characteristics, and chronic conditions, and include ZCTA fixed effects and cancer type-by-year fixed effects. Standard errors are clustered at the ZCTA level.

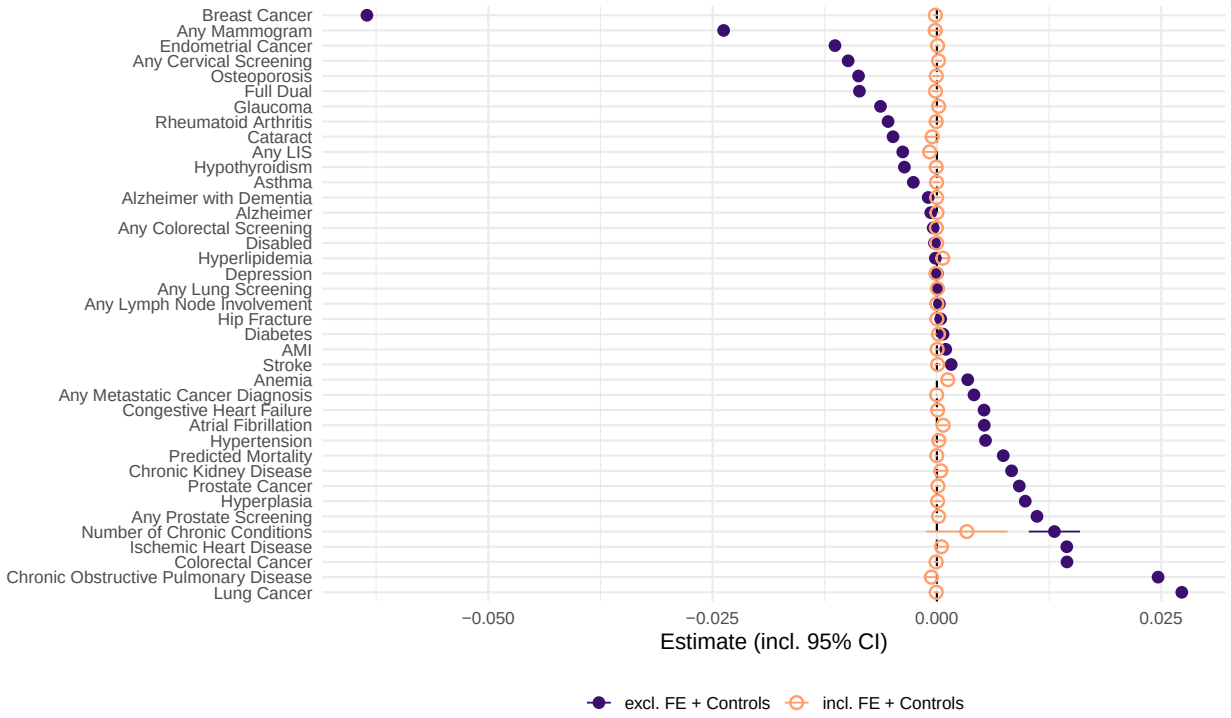
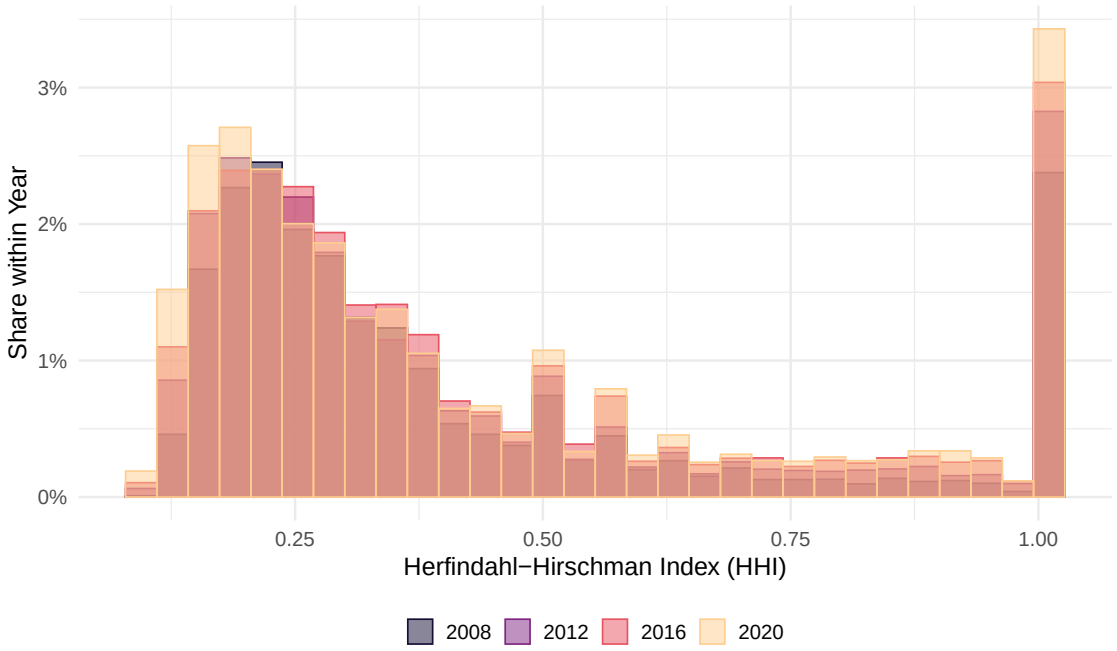
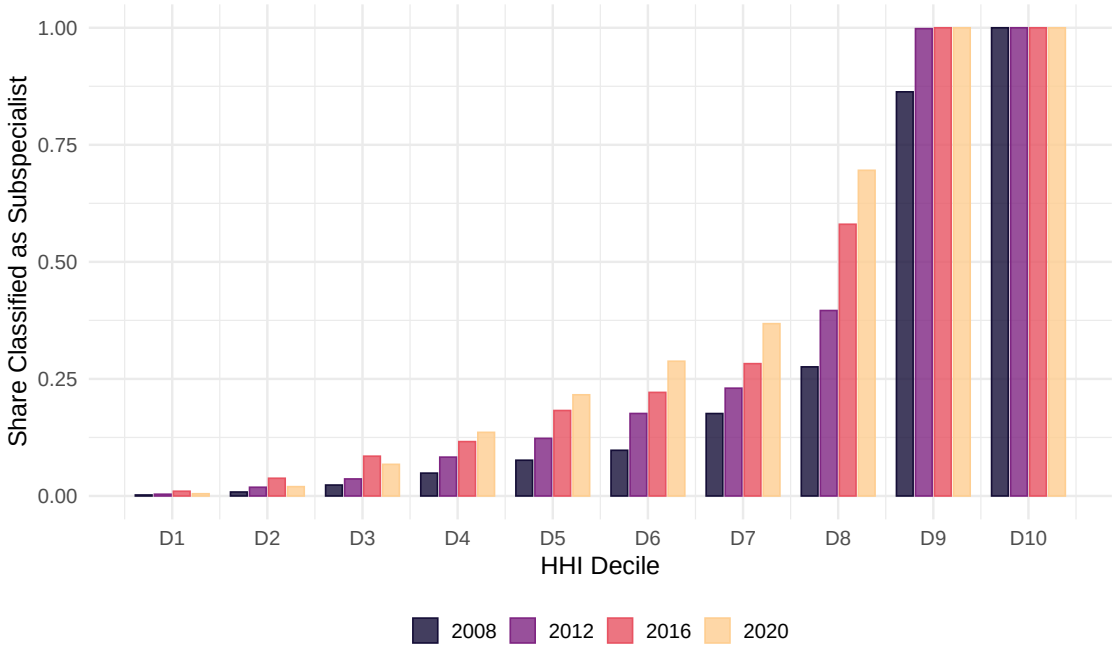


Figure D7: Instrument Balance across Beneficiary Characteristics

Note: This figure displays coefficients from separate regressions of the instrument on the beneficiary characteristics listed on the y-axis. Each point represents a distinct regression. Solid purple dots show unadjusted associations without controls or fixed effects. Hollow orange dots include our full set of ZIP Code Tabulation Area (ZCTA) controls, ZCTA fixed effects, and cancer type-by-year fixed effects. Confidence intervals are based on heteroskedasticity-robust standard errors in unadjusted regressions, and ZCTA-clustered standard errors in the adjusted specifications.



(a) HHI Distribution



(b) Share of Subspecialists by HHI Decile

Figure D8: Oncologist Cancer Type Concentration and Subspecialization

Note: The figure displays information about the Herfindahl-Hirschman Indices (HHI of detailed cancer type concentration (46 unique cancer types) for the years 2008, 2012, 2016 and 2020. In Panel A we plot the distribution of oncologist's HHI of cancer type concentration by year. In Panel B we plot the share of subspecialist oncologists by decile of the respective HHI distribution.

Table D1: Chemotherapy Drug HCPCS Codes

Generic Drug Name	HCPCS Code
LUTETIUM LU 177 DOTATATE	A9513
IBRITUMOMAB	A9543
TOSITUMOMAB	A9545
IOBENGUANE I 131	A9590
TRIPTORELIN	C9016
OBINUTUZUMAB	C9021
DAUNORUBICIN AND CYTARABINE	C9024
RAMUCIRUMAB	C9025
PEMBROLIZUMAB	C9027
INOTUZUMAB OZOGAMICIN	C9028
COPANLISIB	C9030
LUTETIUM LU 177 DOTATATE	C9031
BENDAMUSTINE	C9042
CEMIPLIMAB-RWLC	C9044
MOXETUMOMAB PASUDOTOX-TDFK	C9045
TAGRAXOFUSP-ERZS	C9049
ADO-TRASTUZUMAB EMTANSINE	C9131
BRENTUXIMAB VEDOTIN	C9287
ASPARAGINASE ERWINIA	C9289
PERTUZUMAB	C9292
CARFILZOMIB	C9295
ZIV-AFLIBERCEPT	C9296
OMACETAXINE	C9297
IOBENGUANE I 131	C9408
BELINOSTAT	C9442
BLINATUMOMAB	C9449
NIVOLUMAB	C9453
SILTUXIMAB	C9455
RITUXIMAB AND HYALURONIDASE	C9467
TALIMOGENE LAHERPAREPVEC	C9472
IRINOTECAN, LIPOSOMAL	C9474
NECITUMUMAB	C9475
DARATUMUMAB	C9476
ELOTUZUMAB	C9477
TRABECTEDIN	C9480
ATEZOLIZUMAB	C9483
OLARATUMAB	C9485
AVELUMAB	C9491
DURVALUMAB	C9492
ALEMTUZUMAB	J0202
BUSULFAN	J0594
DECITABINE	J0894
HISTRELIN	J1675
LANREOTIDE	J1930
LEUPROLIDE	J1950
OCTREOTIDE	J2353
OCTREOTIDE	J2354
SILTUXIMAB	J2860
TRIPTORELIN	J3315
TRIPTORELIN	J3316
ANTI-THYMOCYTE GLOBULIN, EQUINE	J7504

(Table D1 continued)

ANTI-THYMOCYTE GLOBULIN, RABBIT	J7511
BUSULFAN	J8510
CAPECITABINE	J8520
CAPECITABINE	J8521
CYCLOPHOSPHAMIDE	J8530
ETOPOSIDE	J8560
FLUDARABINE	J8562
GEFITINIB	J8565
MELPHALAN	J8600
TEMOZOLOMIDE	J8700
TOPOTECAN	J8705
ANTINEO, NOC	J8999
DOXORUBICIN	J9000
DOXORUBICIN, LIPOSOMAL	J9001
DOXORUBICIN, LIPOSOMAL	J9002
ALEMTUZUMAB	J9010
ALDESLEUKIN	J9015
ARSENIC TRIOXIDE	J9017
ASPARAGINASE ERWINIA	J9019
ASPARAGINASE	J9020
ATEZOLIZUMAB	J9022
AVELUMAB	J9023
AZACITIDINE	J9025
CLOFARABINE	J9027
BCG (BACILLUS CALMETTE-GUERIN)	J9030
BCG (BACILLUS CALMETTE-GUERIN)	J9031
BELINOSTAT	J9032
BENDAMUSTINE	J9033
BENDAMUSTINE	J9034
BEVACIZUMAB	J9035
BENDAMUSTINE	J9036
BLINATUMOMAB	J9039
BLEOMYCIN	J9040
BORTEZOMIB	J9041
BRENTUXIMAB VEDOTIN	J9042
CABAZITAXEL	J9043
BORTEZOMIB	J9044
CARBOPLATIN	J9045
CARFILZOMIB	J9047
CARMUSTINE	J9050
CETUXIMAB	J9055
COPANLISIB	J9057
CISPLATIN	J9060
CISPLATIN	J9062
CLADRIBINE	J9065
CYCLOPHOSPHAMIDE	J9070
CYCLOPHOSPHAMIDE	J9080
CYCLOPHOSPHAMIDE	J9090
CYCLOPHOSPHAMIDE	J9091
CYCLOPHOSPHAMIDE	J9092
CYCLOPHOSPHAMIDE	J9093
CYCLOPHOSPHAMIDE	J9094
CYCLOPHOSPHAMIDE	J9095
CYCLOPHOSPHAMIDE	J9096

(Table D1 continued)

CYCLOPHOSPHAMIDE	J9097
CYTARABINE, LIPOSOMAL	J9098
CYTARABINE	J9100
CALASPARGASE PEGOL-MKNL	J9118
CEMIPLIMAB-RWLC	J9119
DACTINOMYCIN	J9120
DACARBAZINE	J9130
DACARBAZINE	J9140
DARATUMUMAB	J9145
DAUNORUBICIN	J9150
DAUNORUBICIN, LIPOSOMAL	J9151
DAUNORUBICIN AND CYTARABINE	J9153
DEGARELIX	J9155
DENILEUKIN DIFTITOX	J9160
DOCETAXEL	J9170
DOCETAXEL	J9171
DURVALUMAB	J9173
ELOTUZUMAB	J9176
ENFORTUMAB VEDOTIN-EJFV	J9177
EPIRUBICIN	J9178
ERIBULIN	J9179
ETOPOSIDE	J9181
ETOPOSIDE	J9182
FLUDARABINE	J9185
FLUOROURACIL	J9190
GEMCITABINE	J9198
GEMCITABINE	J9199
FLOXURIDINE	J9200
GEMCITABINE	J9201
GOSERELIN	J9202
GEMTUZUMAB OZOGAMICIN	J9203
IRINOTECAN, LIPOSOMAL	J9205
IRINOTECAN	J9206
IXABEPILONE	J9207
IFOSFAMIDE	J9208
IDARUBICIN	J9211
INTERFERON, GAMMA 1-B	J9216
LEUPROLIDE	J9217
LEUPROLIDE	J9218
LEUPROLIDE	J9219
HISTRELIN	J9225
IPILIMUMAB	J9228
INOTUZUMAB OZOGAMICIN	J9229
MECHLORETHAMINE	J9230
MELPHALAN	J9245
MELPHALAN	J9246
NELARABINE	J9261
OMACETAXINE	J9262
OXALIPLATIN	J9263
PACLITAXEL, PROTEIN-BOUND	J9264
PACLITAXEL	J9265
PEGASPARGASE	J9266
PACLITAXEL	J9267
PENTOSTATIN	J9268

(Table D1 continued)

TAGRAXOFUSP-ERZS	J9269
PEMBROLIZUMAB	J9271
MITOMYCIN	J9280
OLARATUMAB	J9285
MITOMYCIN	J9290
MITOMYCIN	J9291
MITOXANTRONE	J9293
NECITUMUMAB	J9295
NIVOLUMAB	J9299
GEMTUZUMAB OZOGAMICIN	J9300
OBINUTUZUMAB	J9301
OFATUMUMAB	J9302
PANITUMUMAB	J9303
PEMETREXED	J9305
PERTUZUMAB	J9306
PRALATREXATE	J9307
RAMUCIRUMAB	J9308
POLATUZUMAB VEDOTIN-PIIQ	J9309
RITUXIMAB	J9310
RITUXIMAB AND HYALURONIDASE	J9311
RITUXIMAB	J9312
MOXETUMOMAB PASUDOTOX-TDFK	J9313
ROMIDEPSIN	J9315
STREPTOZOCIN	J9320
TALIMOGENE LAHERPAREPVEC	J9325
TEMOZOLOMIDE	J9328
TEMSIROLIMUS	J9330
THIOTEPA	J9340
TOPOTECAN	J9350
TOPOTECAN	J9351
TRABECTEDIN	J9352
ADO-TRASTUZUMAB EMTANSINE	J9354
TRASTUZUMAB	J9355
TRASTUZUMAB AND HYALURONIDASE-OYSK	J9356
VALRUBICIN	J9357
FAM-TRASTUZUMAB DERUXTECAN-NXKI	J9358
VINBLASTINE	J9360
VINCRISTINE	J9370
VINCRISTINE, LIPOSOMAL	J9371
VINCRISTINE	J9375
VINCRISTINE	J9380
VINORELBINE	J9390
FULVESTRANT	J9395
ZIV-AFLIBERCEPT	J9400
Not otherwise classified, antineoplastic drugs	J9999
TENIPOSIDE	Q2017
TISAGENLECLEUCEL	Q2040
AXICABTAGENE CILOLEUCEL	Q2041
TISAGENLECLEUCEL	Q2042
SIPULEUCEL-T	Q2043
DOXORUBICIN, LIPOSOMAL	Q2048
DOXORUBICIN, LIPOSOMAL	Q2049
DOXORUBICIN, LIPOSOMAL	Q2050
BEVACIZUMAB-AWWB	Q5107

(Table D1 continued)

TRASTUZUMAB-DTTB	Q5112
TRASTUZUMAB-PKRB	Q5113
TRASTUZUMAB-DKST	Q5114
RITUXIMAB-ABBS	Q5115
TRASTUZUMAB-QYYP	Q5116
TRASTUZUMAB-ANNS	Q5117
BEVACIZUMAB-BVZR	Q5118
RITUXIMAB-PVVR	Q5119
ALEMTUZUMAB	Q9979
TEMOZOLOMIDE	WW002
TEMOZOLOMIDE	WW003
TEMOZOLOMIDE	WW004
TEMOZOLOMIDE	WW005
TEMOZOLOMIDE	WW006
TEMOZOLOMIDE	WW007
TEMOZOLOMIDE	WW008
TEMOZOLOMIDE	WW009
BUSULFAN	WW020
ETOPOSIDE	WW030
ETOPOSIDE	WW031
ETOPOSIDE	WW032
MELPHALAN	WW080
MELPHALAN	WW081
CAPECITABINE	WW089
CAPECITABINE	WW090
CAPECITABINE	WW091
CAPECITABINE	WW093
CAPECITABINE	WW094
CAPECITABINE	WW096
TOPOTECAN	WW140

Notes: The table provides the generic drug names and HCPCS codes for chemotherapy drugs used to construct chemotherapy episodes following the Oncology Care Model.

Table D2: ICD 9 and ICD 10 Codes for Cancer Type Classification

Cancer Type Label	**Cancer Types Included**	**ICD-9/ICD-10 Codes**
Breast		174.xx, 175.xx, 233.0x, C50.xx, D05.xx
GI	Esophagus, stomach, pancreas, liver, small intestine, colon, rectum	154.2x, 154.3x, 154.8x, 230.0x, 230.1x, 230.2x, 230.3x, 230.4x, 230.5x, 230.6x, 230.7x, 230.8x, 230.9x, 150.xx, 151.xx, 155.xx, 156.0x, 156.1x, 156.2x, 156.8x, 156.9x, 195.2x, 159.xx, 157.xx, 152.xx, 153.xx, 154.0x, 154.1x, C21.xx, D00.xx, D01.xx, C15.xx, C16.xx, C22.xx, C23.xx, C24.xx, C76.2x, C25.xx, C17.xx, C18.xx, C19.xx, C20.xx
Gynecologic	Ovaries, uterus, cervix, vulva, vagina	233.1x, 179.xx, 180.xx, 182.xx, 184.0x, 184.1x, 184.2x, 184.3x, 184.4x, 183.2x, 183.3x, 183.4x, 183.5x, 183.8x, 183.9x, 184.8x, 184.9x, 181.xx, 183.0x, D06.xx, C51.xx, C52.xx, C53.xx, C54.xx, C55.xx, C57.xx, C58.xx, C56.xx
Head and Neck	Lip, tongue, salivary gland, gum, floor of mouth, oropharyngeal, nasopharyngeal, hypopharyngeal, sinuses, laryngeal, trachea, nasal cavity, middle ear, eye, adnexa, head, face, neck, palate, parotid gland	231.xx, 140.xx, 141.0x, 141.1x, 141.2x, 141.3x, 141.4x, 141.5x, 141.6x, 141.8x, 141.9x, 142.0x, 142.1x, 142.2x, 142.8x, 142.9x, 143.xx, 144.xx, 145.0x, 145.1x, 145.2x, 145.3x, 145.4x, 145.5x, 145.6x, 145.8x, 145.9x, 146.0x, 146.1x, 146.2x, 146.3x, 146.4x, 146.5x, 146.6x, 146.7x, 146.8x, 146.9x, 147.xx, 148.0x, 148.1x, 148.2x, 148.3x, 148.8x, 148.9x, 149.xx, 160.0x, 160.1x, 160.2x, 160.3x, 160.4x, 160.5x, 160.8x, 160.9x, 161.xx, 162.0x, 190.xx, 195.0x, D02.xx, C00.xx, C01.xx, C02.xx, C03.xx, C04.xx, C05.xx, C06.xx, C07.xx, C08.xx, C09.xx, C10.xx, C11.xx, C12.xx, C13.xx, C14.xx, C30.xx, C31.xx, C32.xx, C33.xx, C69.xx, C76.0x
Leukemia + Lymphoma	Acute leukemia, chronic leukemia, lymphoma, multiple myeloma	205, 205.01, 205.02, 204.0x, 205.3x, 206.0x, 207.0x, 207.2x, 208.0x, 205.2x, 204.1x, 205.1x, 208.1x, 206.1x, 238.71, 208.2x, 208.8x, 208.9x, 204.9x, 238.72, 238.73, 238.74, 238.75, 206.2x, 206.9x, 203.81, 203.0x, 203.1x, 289.83, 205.9x, 238.76, 289.89, 202.3x, 202.5x, 202.6x, 202.9x, 204.2x, 204.8x, 206.8x, 205.8x, 207.8x, 207.1, 207.11, 207.12, 238.4x, 202.8, 202.81, 202.82, 202.83, 202.84, 202.85, 202.86, 202.87, 202.88, 203.8, 203.82, 200.0x, 200.1x, 200.2x, 200.3x, 200.4x, 200.5x, 200.6x, 200.7x, 200.8x, 201.xx, 202.0x, 202.1x, 202.2x, 202.4x, 202.7x, 273.3x, C91.0x, C91.3x, C91.5x, C91.6x, C91.ax, C92.0x, C92.3x, C92.4x, C92.5x, C92.6x, C92.ax, C93.0x, C94.0x, C94.2x, C94.3x, C95.0x, C94.4x, C92.2x, C91.1x, C92.1x, C95.1x, C93.1x, D47.1x, D47.3x
Prostate + Genitourinary	Kidney, bladder, ureter, prostate, testis, penis	188.xx, 189.1x, 189.2x, 189.3x, 189.4x, 189.8x, 189.9x, 233.2x, 233.3x, 233.4x, 233.5x, 233.6x, 189.0x, 187.1x, 187.2x, 187.3x, 187.4x, 187.5x, 187.6x, 187.7x, 187.8x, 187.9x, 186.xx, 185.xx, C65.xx, C66.xx, C67.xx, C68.xx, D07.xx, C64.xx, C60.xx, C63.xx, C62.xx, C61.xx
Skin	Melanoma, non-melanoma skin cancers	232.xx, 172.xx, 209.31, 209.32, 209.33, 209.34, 209.35, 209.36, 173.xx, D04.xx, C43.xx, D03.xx, C4A.xx, C44.xx
Thoracic	Lung, pleura, mediastinum, thymus	162.2x, 162.3x, 162.4x, 162.5x, 162.8x, 162.9x, 165.xx, 163.xx, 164.1x, 164.2x, 164.3x, 164.8x, 164.9x, 195.1x, 164.0x, C34.xx, C39.xx, C45.xx, C38.xx, C76.1x, C37.xx
Other	All other cancers	170.4x, 170.5x, 170.7x, 170.8x, 170.0x, 170.1x, 170.2x, 170.3x, 170.6x, 170.9x, 209.3, 193.xx, 194.0x, 194.1x, 194.3x, 194.4x, 194.5x, 194.6x, 194.8x, 194.9x, 209.0x, 209.1x, 209.2x, 191.xx, 192.0x, 192.1x, 192.2x, 192.3x, 192.8x, 192.9x, 233.7x, 233.9x, 234.xx, 176.xx, 195.5x, 195.8x

Note: The table presents the classification of cancer types used in our main episode data. Column 1 provides the cancer type label, column 2 provides the included cancer types and column 3 the corresponding ICD-9 and ICD-10 codes used for identification of cancers in the Medicare claims.

Table D3: Diagnostic Related Group Codes for Placebo Outcomes

Condition	**DRG Code Nr.**
Acute Myocardial Infarction	280, 281, 282
Hip Fracture	480, 481, 482
Stroke	061, 062, 063, 064, 065, 066

Notes: The table provides the diagnostic related group codes used to define placebo outcomes from Medicare Inpatient files.

Table D4: Mortality Effects of Access to Subspecialized Oncologist - Varying Sample

	180-day	1-Year	2-Year	3-Year
Panel A: 2SLS				
Any Office Visit Subs.	-0.001	-0.015*	-0.034***	-0.033**
	(0.005)	(0.008)	(0.011)	(0.013)
R^2	0.114	0.214	0.290	0.318
F-test (1st stage)	17,177	17,177	15,478	13,911
Observations	5,598,571	5,598,571	5,016,515	4,456,173
Mean Dep. Var.	0.074	0.168	0.298	0.388
Panel B: OLS				
Any Office Visit Subs.	-0.004***	0.006***	0.013***	0.014***
	(0.000)	(0.000)	(0.001)	(0.001)
R^2	0.114	0.215	0.292	0.319
Observations	5,598,571	5,598,571	5,016,515	4,456,173
Mean Dep. Var.	0.074	0.168	0.298	0.388

Notes: The table provides estimates on the effect of access to subspecialized oncologists on various outcomes of mortality for the years 2008 to 2019 of our main sample. Due to the required look forward window when constructing measures of mortality the exact sample varies for the different mortality outcomes. Panel A shows two-stage least squares estimates and Panel B presents the corresponding OLS estimates. All models include demographic, ZCTA level and chronic conditions controls as well fixed effects for the beneficiaries' ZCTA and cancer type by year fixed effects. Standard errors are clustered at the ZCTA level. Signif. Codes: ***: 0.01, **: 0.05, *: 0.1.

Table D5: Mortality Effects of Access to Subspecialized Oncologist - Including Volume Controls

	180-day	**360-day**	**720-day**	**1080-day**
Panel A: 2SLS				
Any Office Visit Subs.	-0.000	-0.022**	-0.040***	-0.035**
	(0.008)	(0.011)	(0.015)	(0.017)
R^2	0.118	0.220	0.295	0.320
F-test (1st stage)	12,004	12,004	12,004	12,004
Observations	4,456,173	4,456,173	4,456,173	4,456,173
Mean Dep. Var.	0.077	0.175	0.303	0.388
Panel B: OLS				
Any Office Visit Subs.	-0.003***	0.012***	0.024***	0.026***
	(0.000)	(0.001)	(0.001)	(0.001)
R^2	0.118	0.220	0.295	0.320
Observations	4,456,173	4,456,173	4,456,173	4,456,173
Mean Dep. Var.	0.077	0.175	0.303	0.388

Notes: The table provides estimates on the effect of access to subspecialized oncologists on various outcomes of mortality for the years 2008 to 2017 of our main sample. Due to the required look forward window when constructing measures of mortality we have fixed the sample to be constant for all measures of mortality. Panel A shows two-stage least squares estimates and Panel B presents the corresponding OLS estimates. All models include demographic controls, ZCTA-level characteristics, and indicators for chronic conditions, as well as fixed effects for the beneficiary's ZCTA and cancer type-by-year. In addition, we control for the volume of cancer episodes of the care coordinating oncologist in each of the five cancer categories using five variables, each transformed using the inverse hyperbolic sine function. Standard errors are clustered at the ZCTA level. Signif. Codes: ***: 0.01, **: 0.05, *: 0.1.

Table D6: Mortality Effects of Access to Subspecialized Oncologist - Including Volume Controls

	180-day	**1-Year**	**2-Year**	**3-Year**
Panel A: 2SLS				
Any Office Visit Subs.	-0.007	-0.038**	-0.051***	-0.045**
	(0.011)	(0.015)	(0.016)	(0.016)
R^2	0.134	0.238	0.313	0.335
F-test (1st stage)	5,422	5,422	5,422	5,422
Observations	1,682,382	1,682,382	1,682,382	1,682,382
Mean Dep. Var.	0.108	0.229	0.368	0.452
Panel B: OLS				
Any Office Visit Subs.	-0.014***	-0.005***	0.003***	0.005***
	(0.001)	(0.001)	(0.001)	(0.001)
R^2	0.130	0.239	0.315	0.336
Observations	1,682,382	1,682,382	1,682,382	1,682,382
Mean Dep. Var.	0.103	0.229	0.368	0.452

Notes: The table provides estimates on the effect of access to subspecialized oncologists on various outcomes of mortality for the years 2008 to 2017 for the first chemotherapy episode of each beneficiary in our main sample. Due to the required look forward window when constructing measures of mortality we have fixed the sample to be constant for all measures of mortality. Panel A shows two-stage least squares estimates and Panel B presents the corresponding OLS estimates. All models include demographic controls, ZCTA-level characteristics, and indicators for chronic conditions, as well as fixed effects for the beneficiary's ZCTA and cancer type-by-year. Standard errors are clustered at the ZCTA level. Signif. Codes: ***: 0.01, **: 0.05, *: 0.1.

Table D7: Balancing Test of Instrumental Variable

Variable	Mean	SD	Est. Unadj.	Std. Err. Unadj.	Est. Adj.	Std. Err. Adj.
Panel A: Chronic Conditions Indicators						
Alzheimer	0.025	0.156	-0.001	0.000	0.000	0.000
Alzheimer with Dementia	0.086	0.280	-0.001	0.000	0.000	0.000
AMI	0.011	0.104	0.001	0.000	0.000	0.000
Anemia	0.496	0.500	0.026	0.000	0.001	0.000
Asthma	0.062	0.241	-0.002	0.000	0.000	0.000
Atrial Fibrillation	0.119	0.324	0.005	0.000	0.000	0.000
Breast Cancer	0.473	0.499	-0.090	0.000	0.000	0.000
Colorectal Cancer	0.078	0.268	0.018	0.000	0.000	0.000
Endometrial Cancer	0.007	0.083	-0.001	0.000	0.000	0.000
Lung Cancer	0.127	0.333	0.035	0.000	0.000	0.000
Prostate Cancer	0.141	0.348	0.027	0.000	0.000	0.000
Cataract	0.231	0.421	-0.006	0.000	0.000	0.000
Congestive Heart Failure	0.195	0.396	0.008	0.000	-0.001	0.000
Chronic Kidney Disease	0.305	0.461	0.014	0.000	0.001	0.000
Chronic Obstructive Pulmonary Disease	0.182	0.386	0.025	0.000	-0.001	0.000
Depression	0.187	0.390	-0.002	0.000	0.000	0.000
Diabetes	0.308	0.462	0.003	0.000	0.000	0.000
Glaucoma	0.116	0.320	-0.008	0.000	0.000	0.000
Hip Fracture	0.011	0.104	0.000	0.000	0.000	0.000
Hyperlipidemia	0.554	0.497	-0.002	0.000	0.000	0.000
Hyperplasia	0.081	0.273	0.011	0.000	0.000	0.000
Hypertension	0.706	0.456	0.007	0.000	0.000	0.000
Hypothyroidism	0.199	0.399	-0.005	0.000	0.000	0.000
Ischemic Heart Disease	0.364	0.481	0.019	0.000	0.000	0.000
Osteoporosis	0.129	0.336	-0.014	0.000	0.000	0.000
Rheumatoid Arthritis	0.391	0.488	-0.009	0.000	0.000	0.000
Stroke	0.042	0.201	0.001	0.000	0.000	0.000
Panel B: Prior Healthcare Use and Diagnosis						
Any Cervical Screening	0.068	0.252	-0.010	0.000	-0.001	0.000
Any Colorectal Screening	0.022	0.147	0.000	0.000	0.000	0.000
Any Lung Screening	0.002	0.050	0.000	0.000	0.000	0.000
Any Mammogram	0.193	0.394	-0.017	0.000	0.001	0.000
Any Prostate Screening	0.057	0.232	0.014	0.000	0.000	0.000
Any Primary Care Visit	0.853	0.354	-0.002	0.000	0.000	0.000
Any ER Visit	0.348	0.476	0.013	0.000	0.000	0.000
Any Hospital Visit	0.253	0.435	0.015	0.000	0.000	0.000
Any Lymph Node Involvement	0.087	0.282	0.001	0.000	0.000	0.000
Any Metastatic Cancer Diagnosis	0.208	0.406	0.017	0.000	0.000	0.000
Panel C: Other Measures						
Any LIS	0.15	0.357	-0.006	0.000	0.000	0.000
Full Dual	0.080	0.271	-0.009	0.000	0.000	0.000
Disabled	0.000	0.002	0.000	0.000	0.000	0.000
Number of Chronic Conditions	4.859	2.932	0.098	0.001	0.001	0.002
Predicted Mortality	0.078	0.107	0.011	0.000	0.000	0.000

Notes: The table provides summary statistics for different beneficiary characteristics and healthcare indicators prior to chemotherapy. Column 1 indicates the variable name, column 2 the mean within the overall sample, column 3 the standard deviation, column 4 the estimate of an unadjusted regression of our instrument on the variable in the respective row (without any controls and without fixed effects), column 5 the corresponding heteroskedasticity robust standard error, column 6 shows estimates from a regression of our instrument on the outcome in the respective row including cancer type by year, as well as ZCTA fixed effects and demographic controls as well as ZCTA level controls, finally column 7 shows the corresponding standard error clustered at the ZCTA level.

Table D8: Access to Subspecialized Oncologist and Detailed Spending

	Part A				Part B			
	Inpatient	**HHA**	**Hospice**	**SNF**	**Carrier**	**Outpatient**	**DME**	**Part D**
Panel A: 2SLS								
Any Office Visit Subs.	470.35*	22.17	-291.15***	214.19***	-5,313.87***	4,015.80***	-43.68	-320.33
	(249.96)	(41.12)	(78.53)	(68.68)	(771.31)	(729.53)	(41.67)	(572.80)
R^2	0.131	0.140	0.028	0.080	0.213	0.144	0.061	0.325
F-test (1st stage)	19,061	19,061	19,061	19,061	19,061	19,061	19,061	19,061
Observations	6,144,329	6,144,329	6,144,329	6,144,329	6,144,329	6,144,329	6,144,329	6,144,329
Mean Dep. Var.	3,994.25	578.84	375.30	584.70	11,815.07	9,475.52	329.88	8,204.55
Panel B: OLS								
Any Office Visit Subs.	1,442.61***	-11.85***	-24.66***	-116.43***	-2,932.87***	5,558.99***	25.98***	774.09***
	(19.93)	(2.49)	(2.75)	(3.76)	(46.10)	(55.48)	(2.71)	(42.86)
R^2	0.132	0.140	0.029	0.081	0.215	0.145	0.062	0.326
Observations	6,144,329	6,144,329	6,144,329	6,144,329	6,144,329	6,144,329	6,144,329	6,144,329
Mean Dep. Var.	3,994.25	578.84	375.30	584.70	11,815.07	9,475.52	329.88	8,204.55

Notes: The table provides estimates on the effect of access to subspecialized oncologists on different measures of spending for chemotherapy episodes in our main sample. Column 1 provides estimates for inpatient, column 2 for home health, column 3 for hospice, column 4 for skilled nursing facilities, column 5 for carrier file spending, column 6 for outpatient, column 7 for durable medical equipment and column 9 for Part D spending. Panel A shows two-stage least squares estimates and Panel B presents the corresponding OLS estimates. All models include demographic, ZCTA level and chronic conditions controls as well fixed effects for the beneficiaries' ZCTA and cancer type by year fixed effects. Standard errors are clustered at the ZCTA level. Signif. Codes: ***: 0.01, **: 0.05, *: 0.1.

Table D9: Access to Subspecialized Oncologist RBCS Subcategory Spending

	Chemotherapy (RH)	**Injections & Infusions (RI)**	**Uncategorized**
Panel A: 2SLS			
Any Office Visit Subs.	-2,498.50***	-768.17***	1,852.46***
	(491.74)	(171.45)	(368.74)
R^2	0.214	0.095	0.404
F-test (1st stage)	19,061	19,061	19,061
Observations	6,144,379	6,144,379	6,144,379
Mean Dep. Var.	8,501.45	2,314.60	5,093.14
Panel B: OLS			
Any Office Visit Subs.	-742.58***	-231.71***	2,786.57***
	(29.83)	(10.26)	(28.10)
R^2	0.215	0.136	0.406
Observations	6,144,379	6,144,379	6,144,379
Mean Dep. Var.	8,501.45	2,314.60	5,093.14

Notes: The table provides estimates on the effect of access to subspecialized oncologists on different measures of spending for chemotherapy episodes in our main sample. The spending subcategories follow the Restructured BETOS Classification System (RBCS) and we present select estimates here. Column 1 covers RBCS codes falling into the subcategory "Treatment - Chemotherapy", column 2 covers spending in the category "Treatment - Injections and Infusions (nononcologic)" and column 3 covers HCPCS codes that are not categorized in the BETOS classification (e.g. mostly retired chemotherapy codes). Panel A shows two-stage least squares estimates and Panel B presents the corresponding OLS estimates. All models include demographic, ZCTA level and chronic conditions controls as well fixed effects for the beneficiaries' ZCTA and cancer type by year fixed effects. Standard errors are clustered at the ZCTA level. Signif. Codes: ***: 0.01, **: 0.05, *: 0.1.

Table D10: Access to Subspecialized Oncologist and Effects on Spending by Cost Quintile

	Q1	Q2	Q3	Q4	Q5
Panel A: 2SLS					
Any Office Visit Subs.	-1.45	-1.88	-62.68**	-354.67***	-2,833.96***
	(1.13)	(6.56)	(25.66)	(95.97)	(522.22)
R^2	0.045	0.088	0.218	0.133	0.210
F-test (1st stage)	19,061	19,061	19,061	19,061	19,061
Observations	6,144,379	6,144,379	6,144,379	6,144,379	6,144,379
Mean Dep. Var.	13.82	68.11	457.79	1,578.72	8,710.25
Panel B: OLS					
Any Office Visit Subs.	0.56***	8.69***	-9.31***	-190.50***	-798.46***
	(0.07)	(0.41)	(2.03)	(6.10)	(31.34)
R^2	0.046	0.088	0.219	0.133	0.211
Observations	6,144,379	6,144,379	6,144,379	6,144,379	6,144,379
Mean Dep. Var.	13.82	68.11	457.79	1,578.72	8,710.25

Notes: The table reports estimates of the effect of access to subspecialized oncologists on Part B chemotherapy drug spending within the RBCS sub-categories "Treatment – Chemotherapy" and "Treatment – Injections and Infusions." We first calculate the average Medicare spending per HCPCS code across all episodes and assign each code to a spending quintile. We then aggregate episode-level spending by summing across all HCPCS codes within each quintile and estimate treatment effects separately for each spending tier. The table provides both 2SLS only. Results for different measures of spending are presented across different panels. All models include demographic, ZCTA level and chronic conditions controls as well fixed effects for the beneficiaries' ZCTA and cancer type by year fixed effects. Standard errors are clustered at the ZCTA level. Signif. Codes: ***: 0.01, **: 0.05, *: 0.1.

Table D11: Subspecialist Access and End of Life Care (30 Days before Death) - Decedent Sample

	Any ER	**Any ICU**	**Any Hospice (30-3)**	**Any Hospice (3-0)**
Panel A: 2SLS				
Any Office Visit Subs.	0.056	0.095**	-0.040	0.024
	(0.048)	(0.044)	(0.048)	(0.038)
R^2 0.116	0.161	0.087	0.070	
F-test (1st stage)	929	929	929	929
Observations	440,475	440,475	440,475	440,475
Mean Dep. Var.	0.579	0.299	0.352	0.168
Panel B: OLS				
Any Office Visit Subs.	-0.021***	-0.004*	0.021***	0.000
	(0.002)	(0.002)	(0.002)	(0.002)
R^2	0.119	0.166	0.089	0.071
Observations	440,475	440,475	440,475	440,475
Mean Dep. Var.	0.579	0.299	0.352	0.168

Notes: The table provides estimates on the effect of access to subspecialized oncologists on different measures of end of life care within the last 30 days of a beneficiaries life for a selected sample of decedents. Decedents are included in this sample if they died during or within 30 days of chemotherapy. Column 1 shows the effect on the probability of emergency room admission, column 2 on the effect of intensive care unit admission, column 3 shows the effect on whether a beneficiary has had a hospice claim within the last 30 to 3 days before death (30-3) and column 4 whether a beneficiary had any claim within the last 3 days of life (3-0). Panel A shows two-stage least squares estimates and Panel B presents the corresponding OLS estimates. All models include demographic, ZCTA level and chronic conditions controls as well fixed effects for the beneficiaries' ZCTA and cancer type by year fixed effects. Standard errors are clustered at the ZCTA level. Signif. Codes: ***: 0.01, **: 0.05, *: 0.1.