

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 examines the impact of physician subspecialization in medical oncology on patient outcomes, health care spending, and access to innovation. Using detailed US Medicare data on 2.2 million first-time chemotherapy episodes from 2008 to 2020, we exploit quasi-random variation in access to subspecialized oncologists through a differential distance instrument. We find that access to a subspecialist reduces three-year mortality by up to 10 percent relative to the mean, without increasing total Medicare spending. Subspecialist care also increases the use of newer chemotherapy agents and more than doubles enrollment in clinical cancer trials—particularly for trials aligned with the patient’s cancer type. Falsification and selection analyses support the identifying assumptions and suggest that observed gains reflect differences in treatment pathways rather than patient selection. These findings provide new evidence on the productivity effects of specialization in a non-routine, knowledge-intensive profession, and underscore the organizational trade-offs involved in delivering complex care.

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1 Introduction

Modern economies rely on increasingly narrow forms of specialization, with workers focusing on ever more specific tasks and domains. This paper examines specialization in medicine, focusing on medical oncology, to evaluate how high degrees of specialization affect patient outcomes. In recent decades, medical specialties have increasingly fragmented into narrower subspecialties defined by specific diseases and treatments. Medical oncology exemplifies this trend, with subspecialists focusing on particular cancer types or related groups of cancers (e.g., breast, thoracic, gastrointestinal, or hematologic cancers). Figure 1 shows that between 2008 and 2020, the share of chemotherapy episodes managed by subspecialized oncologists in Medicare nearly doubled from 9 percent to 18 percent, mirroring the rapid expansion of targeted treatments and cancer-specific guidelines (Karadakic et al., 2025; Lozinski, 2024).¹

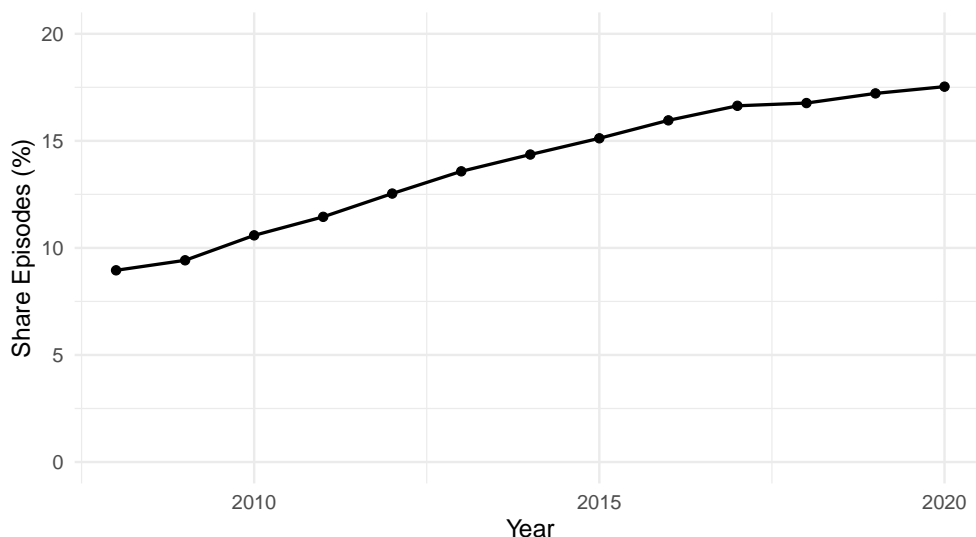


Figure 1: Trends in Subspecialization in Cancer Care 2008 - 2020

Note: This figure presents trends in subspecialization for beneficiary chemotherapy episodes based on results from Karadakic et al. (2025). The figure depicts the share of chemotherapy episodes managed by subspecialized oncologists of the patients' relevant cancer type. Subspecialized oncologists are defined as oncologists who manage more than 80% of chemotherapy episodes within a related group of cancers (e.g. gastrointestinal cancers) during a year.

Subspecialization has the potential to improve the precision of cancer care by enabling physicians to tailor treatments to the biological and clinical features of specific—and often rare—cancers. By concentrating their practice on a narrower set of cancers, physicians accumulate disease-specific expertise and remain closely aligned with

¹See Appendix Figure D1 for an overview of the rise in subspecialization within medical oncology in traditional Medicare between 2008 and 2020 across different cancer types.

rapidly evolving, cancer-specific treatment protocols. At the same time, subspecialists are unevenly distributed geographically, which may limit access for patients in rural or underserved areas (Dingel et al., 2023) and reduces utilization among individuals from lower-income areas (Karadakic et al., 2025). Moreover, many cancer patients present with multiple chronic conditions or overlapping diagnoses, increasing the complexity of coordinating care across providers (Cebul et al., 2008). These competing forces make oncology a particularly well-suited setting to study how increasing specialization affects patient outcomes, spending, and health care utilization.

In this paper, we estimate the causal effect of access to subspecialized oncologic care using quasi-random variation in Medicare patients' differential distance to oncologists who subspecialize in their cancer type relative to general oncologists. This distance-based empirical design follows a widely used approach in the health economics literature (McClellan, McNeil and Newhouse, 1994; Card, Fenizia and Silver, 2023; Gruber et al., 2025). Changes in oncologists' practice locations and the increasing degree of subspecialization across cancer types generate quasi-random variation in geographic access to subspecialists. Using a sample of 2.2 million Medicare beneficiaries initiating their first six-month chemotherapy episode between 2008 and 2020, we compare patients with the same detailed cancer type living in the same ZIP Code Tabulation Area (ZCTA) whose relative proximity to subspecialized oncologists changes over time as oncologists increasingly subspecialize across cancer types. Differential distance strongly predicts whether patients have access to a subspecialized oncologist relevant to their cancer diagnosis. Under the assumption that these changes in differential distance are unrelated to unobserved determinants of outcomes conditional on ZCTA, and cancer-type-by-year fixed effects, our instrumental-variable strategy identifies the causal effect of subspecialist access on patient outcomes, health care utilization and spending.

Our analysis shows that patients with access to subspecialized oncologists experience lower mortality and are more likely to enroll in disease-specific clinical trials. Instrumental-variable estimates indicate that access to a subspecialist reduces 1,080-day mortality by 4.5 percentage points, corresponding to a 10 percent decline relative to the mean. We do not detect statistically significant effects at shorter horizons, with the first measurable mortality effects emerging at one year following chemotherapy initiation, consistent with treatment differences taking time to translate into survival gains. Despite these improvements in outcomes, we find no evidence that subspecialist access increases health care spending. Using comprehensive Medicare claims from Parts A, B, and D, we estimate effects separately for each component and find no significant changes in total episode spending or in Part A, Part B, or Part D spending.

We perform a series of robustness and falsification tests to assess the validity of our

instrumental variable. Differential distance is uncorrelated with a wide range of patient health and demographic characteristics, conditional on fixed effects. In addition, placebo exercises—such as assigning distances to subspecialists treating unrelated cancers—yield no effects on mortality. Consistent with a specialization mechanism, access to subspecialized oncologists increases enrollment in diagnosis-specific cancer trials but has no effect on participation in unrelated or generic multi-cancer trials. Finally, we find no effects on health outcomes unrelated to oncology care, such as myocardial infarction, hip fractures, or strokes.

To assess whether survival gains reflect selective treatment of healthier patients, we analyze a separate sample of first office visits with medical oncologists linked to a cancer diagnosis. This setting allows us to examine whether subspecialists differentially select patients into chemotherapy based on unobserved health or prognosis. Applying our identification strategy to this sample, we find no evidence that patients seen by subspecialists are more likely to initiate chemotherapy following the visit. Among patients who do not initiate chemotherapy, mortality is also similar regardless of whether the initial visit was with a subspecialist or generalist. These results suggest that the survival benefits we document are unlikely to be driven by selection on unobserved patient characteristics.

We explore several mechanisms that may explain the observed mortality reductions. Patients with access to subspecialized oncologists are more likely to enroll in diagnosis-specific clinical trials and are more likely to receive recently approved cancer therapies, suggesting improved access to novel treatments as a potential driver of better outcomes. We also find no evidence that subspecialization increases care fragmentation: the number and diversity of providers involved in treatment remain unchanged, while the number of unique providers declines. Consistent with the role of disease-specific expertise, physicians who concentrate more heavily on a narrow set of cancers achieve larger mortality reductions. Finally, a complier analysis shows that patients whose treatment is influenced by differential distance closely resemble the broader chemotherapy population, supporting the external validity of our estimates.

Our findings contribute to several strands of literature. First, we contribute to the economic literature on specialization and the division of labor (Smith, 1819; Becker and Murphy, 1992; Rosen, 1983; Baumgardner, 1988; Garicano, 2000). Economic theory predicts that specialization increases efficiency and expertise but may create trade-offs related to coordination and access (Cebul et al., 2008). We study these trade-offs in medical oncology, a setting where specialization has expanded rapidly alongside the growth of medical knowledge and targeted therapies (Karadakis et al., 2025; Lozinski, 2024; Cutler and McClellan, 2001). Our results complement Dingel et al. (2023) by providing patient-

level evidence on the consequences of increased specialization in health care markets.

Second, our paper contributes to the literature on physician specialization and productivity (Chan and Chen, 2022; Baicker and Chandra, 2004). Much of the existing evidence on specialization and outcomes comes from surgical settings where expertise is closely linked to procedural volume (Birkmeyer et al., 2002; Huckman and Pisano, 2006; Chandra and Staiger, 2007; Halm, Lee and Chassin, 2002; Avdic, Lundborg and Vikström, 2019; Sahni et al., 2016). In contrast, there is limited causal evidence on specialization in settings where expertise primarily reflects knowledge and treatment decisions, such as medical oncology. Using newly constructed chemotherapy episodes that allow us to classify oncologists by cancer-specific subspecialties, we provide causal evidence on how disease-specific expertise affects treatment decisions and patient survival.

Finally, our study contributes to the literature on the diffusion of medical innovation (Coleman, Katz and Menzel, 1957; Agha and Molitor, 2018; Alsan et al., 2022; Chandra and Skinner, 2012). Linking Medicare claims to clinical trial data, we show that patients with greater access to subspecialized oncologists are more likely to enroll in cancer-specific clinical trials and to receive newer chemotherapy drugs. These findings suggest that physician subspecialization plays an important role in shaping access to medical innovation.

The remainder of the paper is organized as follows. Section 2 provides institutional background on medical oncology. Section 3 describes the data, construction of chemotherapy episodes and the instrumental variable. Section 4 outlines the empirical strategy. Section 5 presents the results, Section 6 examines mechanisms, and Section 7 concludes.

2 Background on Medical Oncology

Cancer is the second leading cause of death in the United States, with older populations disproportionately affected.² 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 percent 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

²The median age at cancer diagnosis in the United States is 67 years, meaning that half of all cancer cases occur among individuals aged 67 and older, even though this group represents less than 17 percent of the U.S. population (National Cancer Institute, 2025a).

approaches to care.³

Medical oncology is integral to cancer therapy. Medical oncologists—physicians trained in both internal medicine and oncology, treat patient with cancer using systemic therapies (i.e., cytotoxic chemotherapy, immunotherapy, targeted therapy, and hormonal 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, typically in pill form.

In addition to prescribing and administering systemic anti-cancer and supportive therapies, medical oncologists play a central role in coordinating care across multidisciplinary teams with surgical and radiation oncologists and other health professionals. Advances in treatment over the past decades—such as targeted therapies addressing specific genetic mutations and immunotherapies that harness the immune system—have transformed cancer care (Sharma and Allison, 2015; Carroll et al., 2023) and improved survival for many cancers (Horn et al., 2025; Emens et al., 2017). However, not all new therapies provide meaningful clinical benefit, and recent approvals often deliver only modest survival gains despite high costs (Mailankody and Prasad, 2015). As treatment options expand, clinical judgment becomes increasingly important to select appropriate therapies—sometimes under limited evidence—or to determine when the harms of treatment outweigh the benefits and palliative care is preferable.⁴ The growing complexity of cancer treatment has contributed to increasing subspecialization in oncology, as physicians must keep pace with a rapidly expanding knowledge base (Lozinski, 2024). Two developments illustrate this trend (Figure 2). Panel 2a shows that the cumulative number of cancer drugs—defined by 7-digit Anatomical Therapeutic Chemical (ATC) codes—more than doubled from 114 in 2008 to 241 in 2020. At the same time, clinical guidelines from the National Comprehensive Cancer Network (NCCN) expanded substantially: as shown in Panel 2b, guideline page counts for five major cancer types increased by more than 300 percent between 2002 and 2020, with hematologic cancer guidelines growing by over 700 percent.

The increasing complexity of oncologic care presents opportunities for oncologists to focus on specific individual or related cancer types, such as breast, gastrointestinal, thoracic cancers, or hematologic cancers, allowing them to develop expertise in managing

³The 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)).

⁴The guidelines of the National Comprehensive Cancer Network (NCCN) report the quality of evidence underlying recommendations, highlighting the uncertainty that often surrounds treatment decisions.

the nuances of a set of specific malignancies. Subspecialized care may be particularly relevant for older patients, whose treatment plans require careful balancing of treatment efficacy with 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, Karadakic et al. (2025) 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.⁵ This geographic disparity is especially consequential for Medicare beneficiaries, who may face barriers to travel or rely on local providers for care.

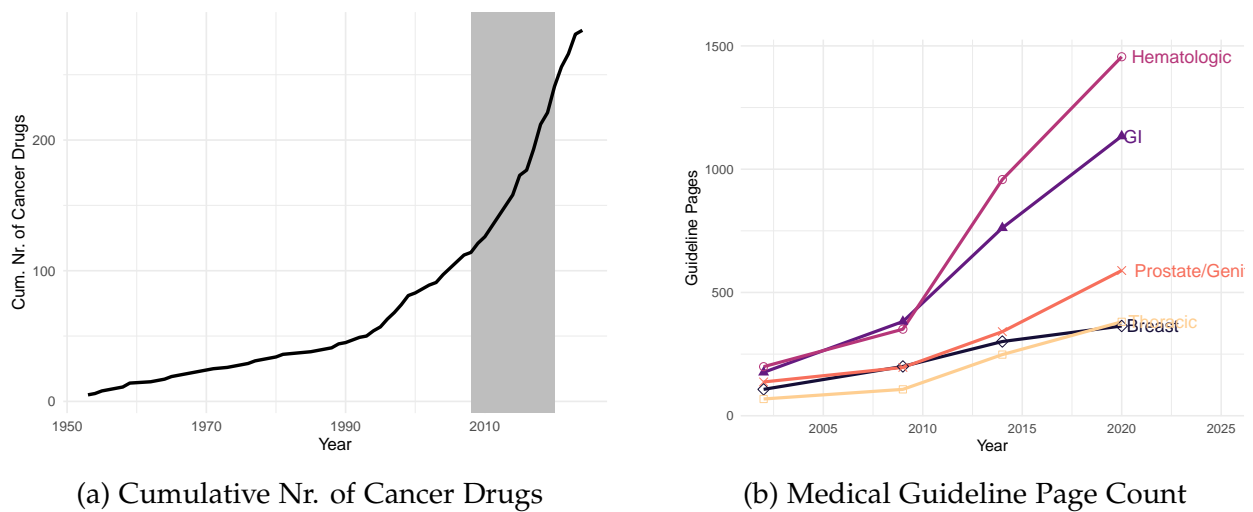


Figure 2: Cancer Drug and Medical Guideline Expansion across Time

Note: This figure presents two proxies for rising complexity in the treatment of cancer. Panel A displays the cumulative number of unique cancer drugs over time, defined by 7-digit Anatomical Therapeutic Chemical (ATC) classification codes, based on data from Pantziarka et al. (2021). Each unique ATC code corresponds to a distinct chemical substance. The shaded gray area highlights the time period of our main chemotherapy sample (2008–2020). Panel B 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 (hematologic, gastrointestinal, prostate/genitourinary, breast and thoracic cancer). Guideline page counts were obtained from Lozinski (2024). Abbreviations: GI=gastrointestinal, Prostate/Geni=prostate/genitourinary.

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 anal-

⁵See 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.

ysis is a dataset containing chemotherapy episodes for the period 2008 to 2020, which is constructed utilizing 100 percent Medicare claims data for fee-for-service beneficiaries accessed through the Center for Medicare and Medicaid Services' (CMS) Virtual Research Data Center (VRDC). We use data from 2007 to 2021 from Medicare Parts A, B and D. In addition we supplement our main sample with publicly available data on socioeconomic characteristics of ZCTAs obtained through the US Census Bureau. Furthermore, we linked 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, 2025b).

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; Keating et al., 2021). 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.⁶ 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.⁷

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).

⁶OCM was in operation from July 2016-December 2022, we applied the episode identification methodology throughout our study period.

⁷We only focus on office visits with medical oncologists defined using CMS specialty codes 82 (hematology), 83 (hematology/oncology), 90 (medical oncology) and 98 (gynecologic oncology). We include gynecologic oncologists because they often provide chemotherapy like medical oncologists.

Chemotherapy is identified using 100% Medicare claims data, specifically physician-administered chemotherapy claims from Carrier and Outpatient files (with a cancer diagnosis on the claim) and prescription fills for chemotherapy agents from the Part D event file.⁸ Part D prescription fills are included only if a corresponding Part B claim with a cancer diagnosis occurred within the prior 59 days to suggest the prescription is for cancer treatment rather than another indication. Using this approach, we define 180-day chemotherapy episodes starting from the initial chemotherapy claim. Each episode is then assigned to the medical oncologist with 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 E2). To account for the look back period and episode definitions, while utilizing 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 attributed to a subspecialized oncologist, we classify subspecialists as oncologists who provide at least 80 percent of their chemotherapy episodes within a single or related group of cancers in a given year. The 80 percent threshold was chosen to reflect a balance between capturing clinicians whose work is dominated by specific cancer types while allowing for the reality that many oncologists—even subspecialists—may have to treat common cancers for financial and clinical reasons (Karadakic et al., 2025). In additional analyses, we also examine a continuous approach to defining specialization based on a detailed set of cancers. Our episode level dataset includes chemotherapy episodes for cancers categorized into 9 broad categories: breast cancer, gastrointestinal (GI) cancers, gynecologic cancers, head and neck cancers, hematologic cancers, prostate/genitourinary cancers, melanoma, thoracic cancers, and other cancers. 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. The same 80 percent threshold applies analogously to oncologists specializing in the remaining cancer categories.

As noted earlier, the share of chemotherapy episodes managed by subspecialized oncologists nearly doubled between 2008 and 2020, rising from approximately 9 percent to 17.5 percent. This increase was especially pronounced in hematologic, breast, and prostate/genitourinary cancers, although subspecialization increased for all cancer types

⁸A full list of all Part B drugs can be found in Appendix Table E1. National Drug Codes (NDC) are available upon request, due to the large number of codes used for drug identification.

to varying degrees (see Appendix Figure D1).

To illustrate the geographic distribution of care received by subspecialist oncologists, Figure 3 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.

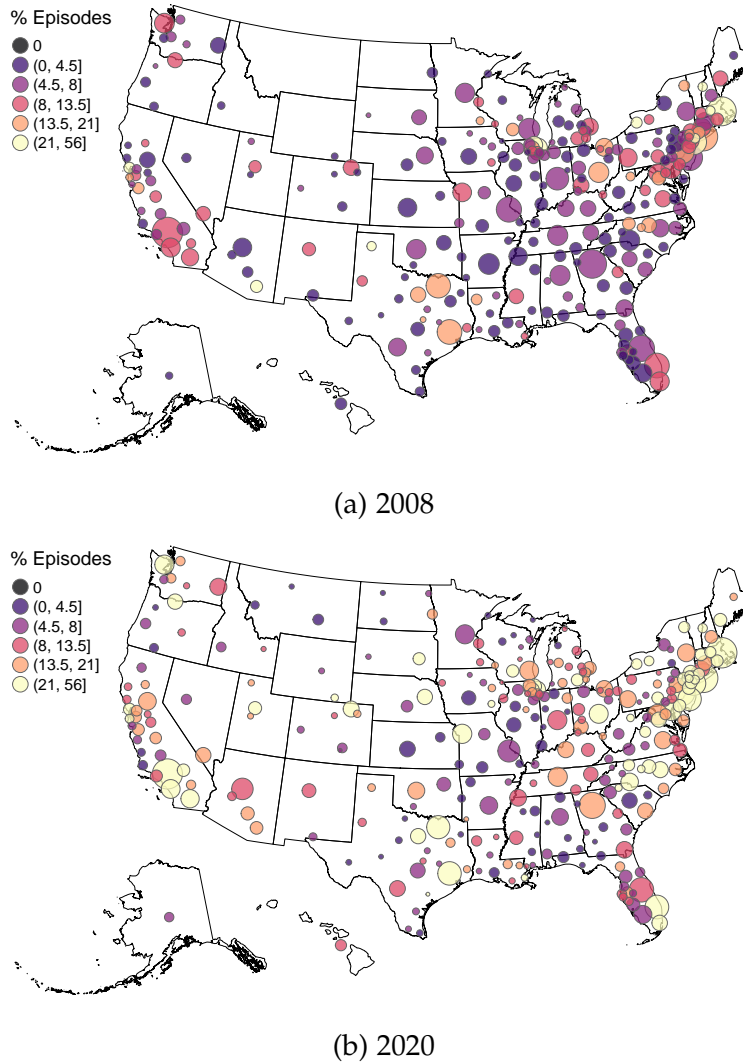


Figure 3: Utilization of Subspecialized Oncologists by Hospital Referral Region

Note: This figure shows the geographic distribution of chemotherapy episodes by Hospital Referral Region (HRR) for the years 2008 and 2020 based on data from Karadakic et al. (2025). Each bubble is positioned at the centroid of the largest polygon within the HRR (based on the beneficiaries location). The bubble size reflects the total number of chemotherapy episodes in the HRR, while the 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 omitted HRRs with less than eleven chemotherapy episodes managed by subspecialized oncologists in a each year.

Our primary measure of access to a subspecialized oncologist is an indicator for whether a beneficiary had any office visit with a subspecialist treating the relevant cancer type during the calendar year in which chemotherapy was initiated. This measure captures patient exposure to subspecialized oncologic expertise. As a secondary measure, we consider whether the chemotherapy episode itself was attributed to a subspecialist, which reflects a more intensive form of subspecialist involvement in treatment. Because the two measures are highly correlated and yield similar results, we focus on the office visit–based definition in our main analyses.

3.3 Main Sample and Variable Definitions

The sample of chemotherapy episodes includes beneficiary identifiers, the date of the initiating chemotherapy claim, the primary cancer type (see Table E2), whether the initial chemotherapy treatment was infused/injected or oral, and the National Provider Identifier (NPI) of the attributed oncologist.

Our main analysis focuses on five of nine cancer type groups—breast, GI, hematologic, prostate/genitourinary, and thoracic cancers—where systemic therapy is commonly used. We exclude head and neck, skin, gynecologic, and other cancers because small sample sizes limit the ability to define subspecialists based on volume. We restrict the sample to each beneficiary’s first chemotherapy episode to capture patients at a similar point in their treatment trajectory and to minimize confounding from prior care. This approach also ensures that mortality effects are estimated on unique individuals rather than repeat observations. Finally, we limit the sample to beneficiaries aged 67 and older to observe at least two full years of Medicare claims history prior to treatment initiation. These sample restrictions are summarized in Appendix Table E3.

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 each of 27 chronic conditions.⁹ Furthermore, we estimate linear probability models on a training set of beneficiaries that are excluded from our main sample and use the resulting coefficients to generate out-of-sample predictions of mortality for individuals in our main sample.¹⁰ Zip codes are converted into ZCTAs

⁹We used end-of-year indicators for all 27 chronic conditions provided by the Chronic Conditions Warehouse (CCW) (CMS, 2021).

¹⁰To construct predicted mortality, we use Medicare claims and enrollment data from 2006–2021 to build a detailed beneficiary-level dataset with demographics, full fee-for-service indicators, chronic condition flags, and prior-year utilization measures (e.g., provider visits, emergency department 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

using publicly available crosswalks (Audirac, 2024). We also construct annual measures of health care access at the ZCTA level using Medicare claims data.¹¹ 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 Appendix Table E4.

To measure spending associated with chemotherapy, we aggregate episode-level expenditures beginning on the date of chemotherapy initiation and continuing through 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). We provide a detailed overview of our spending definitions in Appendix B.

To examine other health outcomes and health care utilization, we leverage 100% Medicare samples, extracting enrollment in clinical trials using National Clinical Trial (NCT) numbers from the Carrier file and constructing acute myocardial infarction, hip fracture, and stroke indicators using diagnostic related group codes from the Inpatient file. 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

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.

¹¹These measures include the average age of Medicare beneficiaries; the shares of beneficiaries who are full fee-for-service, disabled, male, or black; the total number of beneficiaries; and the supply of health care providers, including the total number of providers, primary care physicians, and mental health physicians.

are drawn from the NBER ZIP Code Distance Database ([National Bureau of Economic Research, 2025](#)).

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. General oncologists are defined as all oncologists who do not manage 80 percent of cancers within a set of related cancer types. Due to increases in subspecialization among medical oncologists over time differential distances between subspecialists and generalists become smaller (see Appendix Figure D3).

$$DD_{cit} = \sinh^{-1}(\text{Dist. Subspecialist}_{ct} - \text{Dist. Generalist}_t) \quad (1)$$

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. This restriction avoids drawing inferences from outlier observations in geographically remote locations. Applying these criteria yields a final sample of 2.2 million first chemotherapy episodes, treated by 17,325 distinct medical oncologists across five major cancer groups and 45 detailed cancer types from 2008 to 2020.¹²

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.¹³ 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](#)).

4 Empirical Strategy

The goal of our empirical analysis is to estimate the effect of access to subspecialized oncologists on patient health outcomes and spending. Simple comparisons between pa-

¹²A table with detailed cancer types and corresponding ICD codes can be found in Appendix Table E2.

¹³The definition of the inverse hyperbolic sine transformation is as follows $\sinh^{-1}(x) = \ln(x + \sqrt{x^2 + 1})$.

tients with and without access to subspecialized oncologists are unlikely to yield causal estimates, as subspecialists may treat patients who differ systematically in disease severity, treatment preferences, or other characteristics that influence outcomes.

Our empirical strategy exploits variation in patients' exposure to subspecialized oncology care arising from differences in geographic proximity to subspecialized oncologists relative to general oncologists. We focus on patients receiving chemotherapy, as this is the stage at which treatment decisions are most complex and where differences in oncologist expertise are most likely to affect outcomes. This population is also clinically high-risk and accounts for a substantial share of cancer-related spending. Decisions about treatment selection and the management of treatment-related side effects are central to the care of these patients, making this setting particularly well suited to study the effects of subspecialization.

The empirical design in this study uses a distance-based instrument that 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 that the distance to more subspecialized oncologists might be associated with patient characteristics 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 rely solely on absolute distance, but instead construct differential distance, a measure that captures the relative ease of access to subspecialized versus general oncologists. Second, we augment our instrumental variable strategy by including ZCTA fixed effects, ensuring that identification comes from comparisons among individuals residing in the same geographic area. Importantly, differential distance varies across cancer types within the same ZCTA, reflecting that subspecialized oncologists are unevenly distributed across disease areas. As a result, patients living in the same location may face different access to subspecialized oncologists depending on their cancer diagnosis. Additional variation arises over time as oncologists specialize, exit, or enter local markets.

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:

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

for episode i in ZCTA z in cancer type-by-year t , where DD captures the inverse hyper-

bolic 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 clinical characteristics, as well as ZCTA level controls that 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.

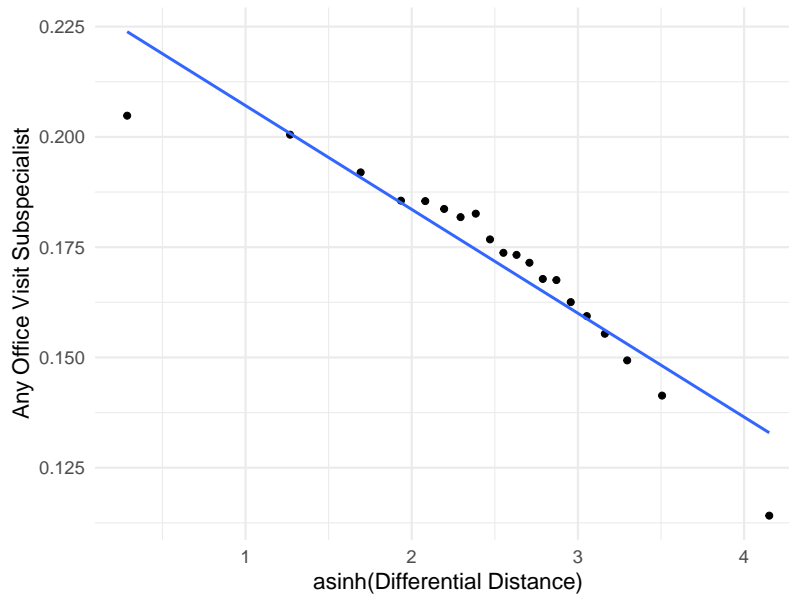


Figure 4: Residualized First Stage Relationship

Note: This figure presents a binned scatter plot of the first-stage relationship after residualizing all variables with respect to the design controls (ZCTA fixed effects and cancer-type-by-year fixed effects). Residuals are re-centered by adding back the sample mean of each variable. The instrument is partitioned into ventiles, and the plotted values correspond to bin-specific means of the instrument and outcome.

To provide intuition for the strength of the instrument, Figure 4 presents a binned scatter plot of the first-stage relationship after residualizing both the instrument and the treatment with respect to the design controls, specifically ZCTA fixed effects and cancer-type-by-year fixed effects. The plotted values correspond to bin-specific means of the residualized variables, re-centered at their respective sample means. The figure shows a clear and negative relationship between differential distance and the probability of receiving care from a subspecialized oncologist, consistent with a strong and mono-

¹⁴We define fixed effects using 45 detailed cancer types, whereas subspecialist classification is based on five broader cancer categories. Table E2 provides an overview of the ICD codes corresponding to each category.

tonic first stage. Appendix Table E7 reports the corresponding first-stage estimates, confirming a strong relationship between differential distance and access to subspecialized oncologists, with large and precisely estimated coefficients and an F-statistic well above conventional thresholds.

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

$$Y_i = \alpha + \beta \widehat{\text{Access}}_i + \delta X_i + \tau_{t(i)} + \gamma_{z(i)} + \psi D_{t(i)z(i)} + \varepsilon_i \quad (3)$$

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

To illustrate the variation leveraged in our empirical strategy, Figure 5 presents the standard deviation of the residualized differential distance measure across selected metropolitan areas, aggregated at the Hospital Service Area (HSA) level.¹⁵ We construct the residualized differential distance by regressing our instrument on our design controls, ZCTA and cancer type-by-year fixed effects. We then standardize the resulting residuals to have mean zero and unit variance. Next, we average these standardized residuals within each HSA and year. Finally, we compute the standard deviation of these HSA-year averages over time. This measure captures the variation in access to subspecialized oncologists that underlies our identification strategy, arising both from changes over time and from differences in differential distance across cancer types within the same geographic areas.

Figure 5 provides graphical intuition for our identifying variation. Panel 5a (Dallas, TX) highlights a metropolitan area with substantial variation in residualized differential distance across HSAs and over time, while Panel 5b (Boston, MA) depicts a metropolitan area with comparatively less variation. These maps illustrate the source of identifying variation in our design. Because our specification includes ZCTA and cancer type-by-year fixed effects, identification comes from differences in differential distance across cancer types within the same geographic area as well as changes in access over time—driven, for example, by physician entry, exit, or shifts in specialization—rather than broader, potentially confounded cross-sectional differences, such as those between rural upstate New York and Manhattan.¹⁶

This interpretation is further supported by Appendix Table E5, which decomposes

¹⁵While our empirical strategy exploits variation at the ZCTA level, data use restrictions prevent us from displaying beneficiary-level ZCTA data.

¹⁶Appendix Figure D6 shows the distribution of our instrument before and after residualizing for ZCTA and cancer type-by-year fixed effects, illustrating how our empirical design restricts identification to variation within ZCTAs across cancer types and over time.

the variation in the instrument across geographic, cancer-type, and time dimensions. Consistent with a distance-based measure, a large share of variation is explained by cross-sectional differences across ZCTAs (Adj. $R^2 = 0.527$), while cancer types account for a smaller portion (Adj. $R^2 = 0.102$). Our baseline specification, which includes ZCTA and cancer type-by-year fixed effects, explains approximately 63 percent of the variation in the instrument. Adding ZCTA \times cancer type fixed effects further increases the adjusted R^2 to 0.724,¹⁷ indicating that an important share of the remaining variation reflects differences in relative access across cancer types within the same ZCTA, whereas allowing for ZCTA-specific linear time trends leads to a more modest increase (Adj. $R^2 = 0.667$). Taken together, these results suggest that the identifying variation reflects both cross-cancer-type differences within locations and, to a lesser extent, changes over time in local access to subspecialized oncologists.

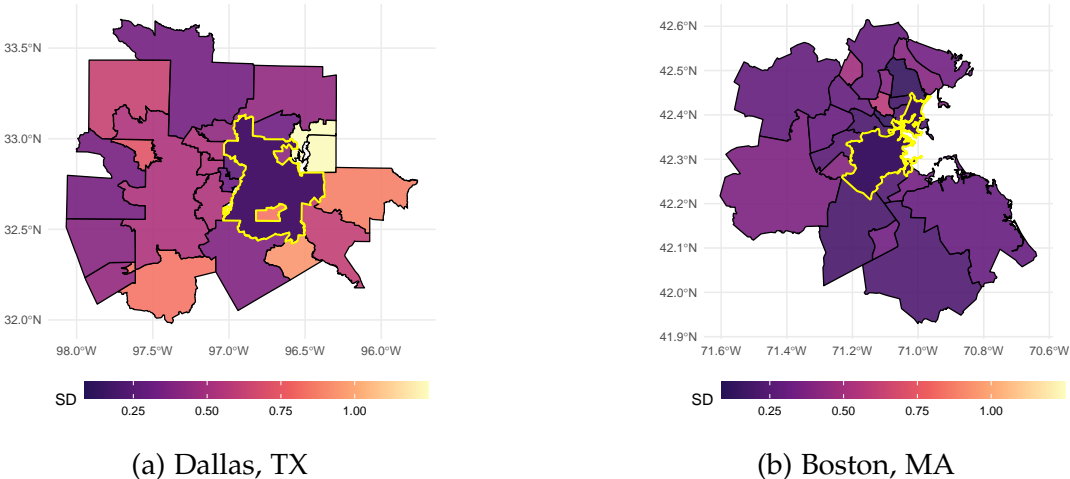


Figure 5: Variation in Residualized Differential Distance across Hospital Service Areas

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 of the data environment. The maps illustrate geographic variation in this measure across selected metropolitan areas. Panels A depicts the greater Dallas metropolitan areas and highlights regions with high variation in residualized differential distance over time. Panel B depicts the greater Boston metropolitan areas and highlights regions with comparably low variation in residualized differential distance over time. HSAs outlined in yellow represent the central HSA of the respective metropolitan area (e.g. downtown Boston for the Boston metropolitan area).

To isolate the sources of variation in our instrumental variable, we construct three alternative versions of the differential-distance measure. First, we hold the stock of oncologists fixed at its 2008 composition, preventing physicians from entering or leaving

¹⁷This specification is computationally intensive and drops approximately 97,000 singleton fixed-effect observations.

the workforce. This allows us to assess how much of the identifying variation reflects physician entry and exit. Second, we hold each oncologist’s practice location (ZCTA) fixed at the first year in which they appear in our data, thereby removing all variation arising from changes in where oncologists practice over time. Finally, we construct a version of the instrument that holds each oncologist’s subspecialization status fixed at the year they first enter our sample. This isolates how much of the baseline instrument’s variation is driven by changes in oncologists’ subspecialization patterns rather than changes in supply or location.

Appendix Table E6 shows that most of the identifying variation in our differential-distance instrument is driven by changes in oncologist subspecialization rather than workforce churn or physician relocation. When holding either the oncologist stock or their practice ZCTA fixed, the resulting instruments remain closely aligned with the baseline measure, exhibit nearly identical first-stage relationships, and retain substantial explanatory power in the within-specification (within-adjusted R^2 of 0.31 and 0.26). In contrast, fixing each oncologist’s subspecialization status substantially weakens the instrument: the correlation with the baseline measure declines, the within-adjusted R^2 falls to 0.13, and the first-stage coefficient decreases by more than half. Taken together, these results indicate that the identifying variation in our design is primarily driven by changes in subspecialization, both over time and across cancer types within the same ZCTA, rather than by shifts in the geographic distribution of oncologists.¹⁸

4.1 Instrument Validity

Our instrumental variable design identifies a local average treatment effect (LATE) for the subgroup of compliers whose probability of receiving care from a subspecialized oncologist is affected by differential distance. For this interpretation to hold, several assumptions must be satisfied. First, we require monotonicity: an increase in the relative distance to a subspecialist (compared to a general oncologist) must weakly reduce the likelihood of accessing subspecialized care for all individuals, ruling out the presence of “defiers” who would be more likely to seek subspecialized care as relative access worsens. In our setting, this assumption is plausible, as greater relative distance increases travel costs and reduces the convenience of accessing subspecialists, making systematic

¹⁸This pattern is intuitive. Entry and exit of oncologists primarily occur in markets that already have substantial oncology capacity, and oncologists are geographically concentrated (Milligan et al., 2024; Karadakic et al., 2025), so holding the workforce or practice locations fixed leaves most of the variation in differential distance intact. By contrast, subspecialization has evolved considerably over time, with many general oncologists transitioning into subspecialized practice. These changes alter relative distances to subspecialists versus generalists across cancer types within the same location, explaining why subspecialization accounts for the bulk of the identifying variation in the instrument.

substitution toward subspecialized care unlikely. Consistent with this interpretation, we find no evidence of systematic violations of the monotonicity assumption when estimating the first-stage relationship across a range of subsamples (Appendix Figure D4).

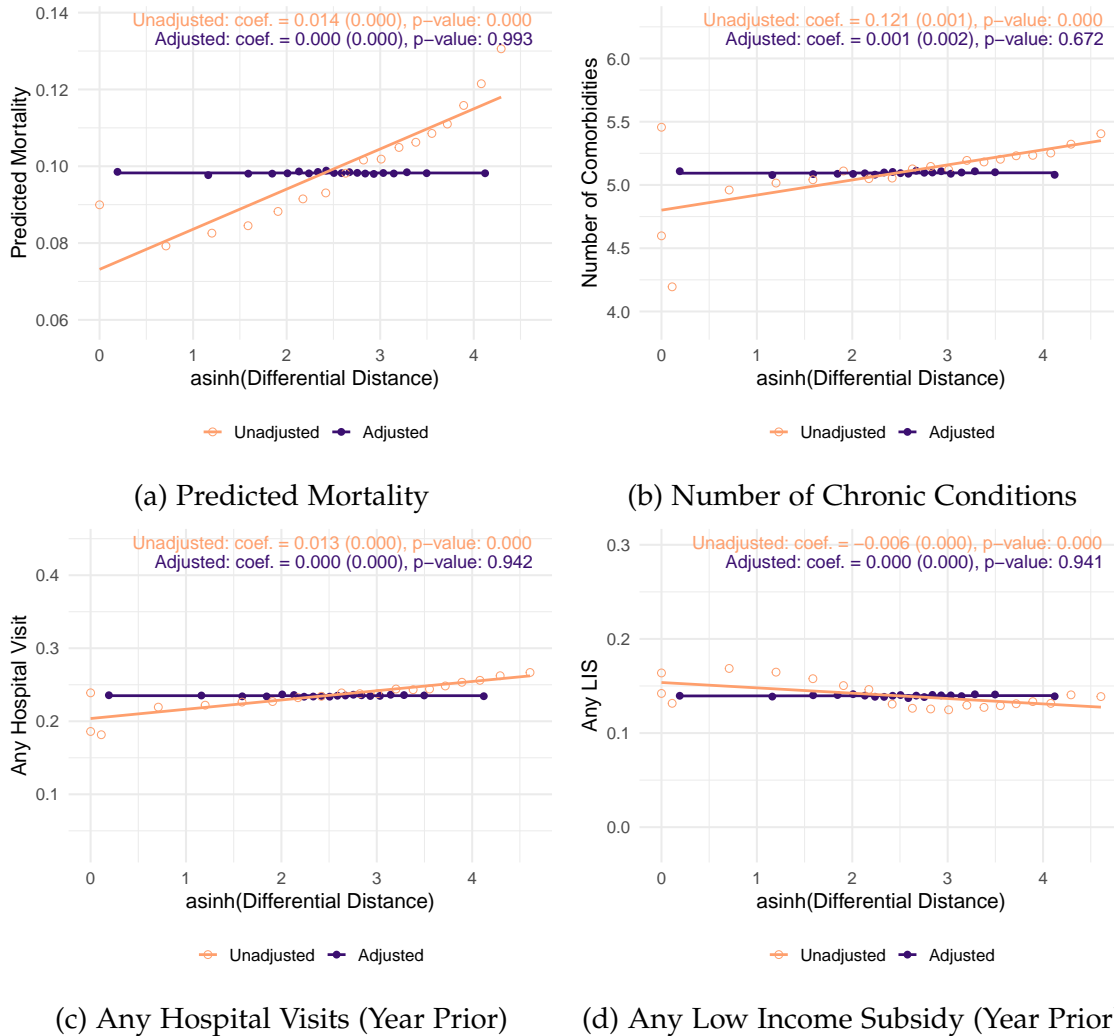


Figure 6: Distribution of Instrumental Variable

Note: These figures provide graphical representations of the balance-test regressions at the first chemotherapy episode level. Each panel presents both unadjusted and adjusted relationships between the instrument (differential distance) and a beneficiary characteristic. To construct the adjusted binned scatter plots, we residualize both axes with respect to ZCTA and cancer type-by-year fixed effects and add back the mean to aid interpretation. The unadjusted points correspond to simple bins of the raw variables. The middle number reports the sample mean, while the bottom and top numbers correspond to the mean minus and plus one-half of a standard deviation of the residualized variable. Within each panel, we report coefficients, standard errors, and p-values for both specifications. The unadjusted model reflects a simple regression of the instrument on the outcome, while the adjusted model includes the full set of design controls (ZCTA and cancer type-by-year fixed effects). Panel A presents predicted mortality; Panel B the number of chronic conditions; Panel C an indicator for any hospital visit in the prior year; and Panel D an indicator for any month with a low-income subsidy in the prior year.

A second key requirement is the independence assumption: conditional on fixed

effects, differential distance must be uncorrelated with unobserved determinants of patient outcomes. Violations of this assumption could arise if differential distance is systematically related to patient characteristics, such as baseline health or prior healthcare utilization, that independently affect outcomes. To assess the plausibility of this assumption, we examine balance in observable beneficiary characteristics. In the terminology of [Danieli et al. \(2026\)](#), these balance tests can be interpreted as negative control outcome tests, which probe the presence of alternative pathways linking the instrument to outcomes.

Figure 6 presents graphical representations of balance-test regressions for four selected variables that capture a broad range of patient characteristics, including predicted mortality, comorbidity burden, prior healthcare utilization, and socioeconomic status. Results are shown for both unadjusted and adjusted specifications. The unadjusted model corresponds to a simple regression of the instrument on the outcome, while the adjusted model includes ZCTA and cancer type-by-year fixed effects. The figure shows that, across all outcomes, there is a clear relationship between the instrument and the outcome in the unadjusted specification that becomes statistically insignificant once we include fixed effects. This pattern suggests that, after restricting variation to within ZCTAs over time and across cancer types, the instrument is not systematically related to patient characteristics. We present additional balance results for a broader set of variables in Appendix Figure D5 and Appendix Table E8.

While balance in observables does not guarantee independence with respect to unobservables, the absence of systematic relationships across a rich set of characteristics provides supportive evidence that, conditional on our empirical design, differential distance is plausibly independent of determinants of patient outcomes.

The exclusion restriction requires that differential distance affects outcomes only through its impact on access to subspecialized oncologists, rather than through other channels such as broader features of the local treatment environment. A key concern is that differential distance may proxy for characteristics of high-quality care settings—such as academic medical centers—that independently influence patient outcomes. In our setting, this concern is mitigated by the cancer-type specificity of the instrument, which varies within ZCTAs across cancer types and over time. We further assess the plausibility of this assumption using falsification exercises presented in Section 5.3.

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 medium-term mortality, but has no detectable effect on short-term mortality (within one year). These estimates contrast with positive mortality effects in OLS specifications, consistent with selection into subspecialized care. We also examine healthcare spending per chemotherapy episode and find no evidence of meaningful differences in overall spending. We then assess the exclusion restriction through a series of falsification tests, conduct additional robustness analyses, and characterize the complier subgroup.

5.1 Mortality

Our primary health outcome is mortality, defined as the time from chemotherapy initiation to death.¹⁹ We construct binary indicators for mortality at multiple horizons, setting the variable equal to one if a patient dies within a given period after starting chemotherapy. These indicators range from 90-day to 1,080-day mortality, in 90-day increments.

Figure 7 presents a residualized binned scatter plot of three-year mortality against differential distance. The figure shows a generally positive relationship, indicating that larger differential distances to subspecialized oncologists are associated with higher mortality. While the relationship is not perfectly monotonic across all bins, the overall pattern is consistent with a decline in mortality as access to subspecialized care improves, with somewhat larger differences at higher values of differential distance. These reduced-form patterns suggest that the instrument shifts mortality in the expected direction.

To estimate the effect of subspecialist access, we apply Equation 3 and plot the 2SLS estimates in Figure 8 Panel 8a. The results indicate no statistically significant mortality effects before 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 360-day mortality by 4 percentage points. Given an average 1-year mortality rate of 22.6 percent, this corresponds to a 17.7 percent reduction relative to the mean.

Notably, the mortality reduction reaches 5.1 percentage points at 540 days post chemotherapy initiation before marginally declining thereafter. Mortality rates continue

¹⁹Chemotherapy initiation is defined following the OCM methodology and corresponds to the date of the first qualifying chemotherapy claim. Specifically, the trigger date is the line first expense date on the chemotherapy drug line in Carrier claims, the revenue center date in Outpatient claims, and the fill date in Part D Event (PDE) data.

to rise over the time period following chemotherapy initiation, leading to a reduction in the overall effect size expressed in relation to mean mortality. To account for this, Figure 8 Panel 8b scales the mortality estimates relative to the population’s mean mortality. The results show that relative mortality reductions peak at 17.7 percent for 360-day mortality before declining slightly. 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 cancer and other comorbidities.

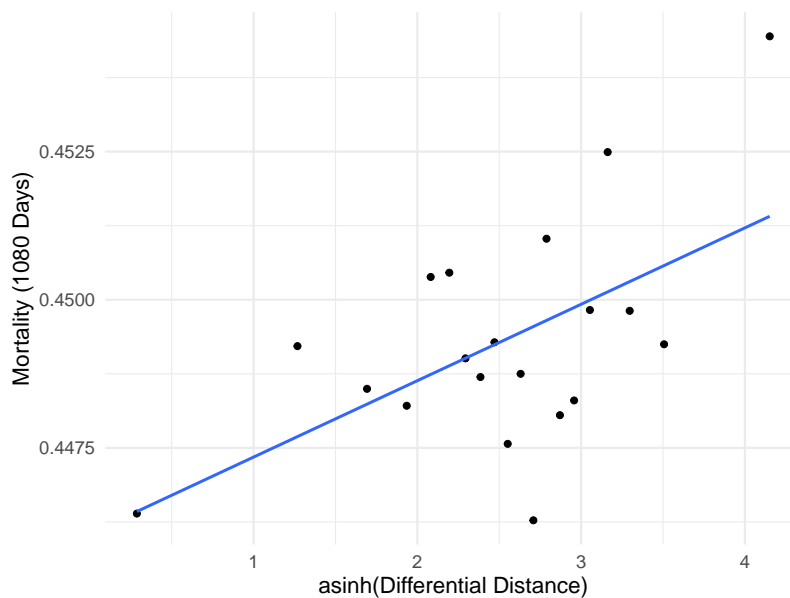


Figure 7: Reduced Form Relationship

Note: This figure presents a binned scatter plot of the reduced form relationship after residualizing both variables with respect to the design controls (ZCTA fixed effects and cancer-type-by-year fixed effects). Residuals are re-centered by adding back the sample mean of each variable. The instrument is partitioned into ventiles, and the plotted values correspond to bin-specific means of the instrument and outcome.

In contrast to the negative mortality effects estimated using 2SLS, the OLS estimates of the structural form suggest that access to subspecialists is associated with with higher mortality after 540 days. For example, OLS results indicate that having access to a subspecialized oncologist is associated with a 0.3 percentage point increase in 720-day mortality and a 0.5 percentage point increase in 1,080-day mortality (see Appendix Table E9). This pattern suggests 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 are not observed in claims

data. To assess the robustness of our results to potential selection into treatment, we compare our baseline estimates to alternative specifications and samples. In our main specification, all outcomes are estimated on a common sample covering chemotherapy episodes from 2008 to 2017. In Appendix Table E10, Panel B, we instead allow the estimation sample to vary with the mortality horizon, so that each outcome uses the maximum available follow-up (with longer horizons excluding later years due to data limitations). Results are similar, indicating that our findings are not driven by the common-sample restriction.

Second, we control for the episode-attributed oncologist’s treatment volume across the five main cancer categories by including inverse hyperbolic sine–transformed measures of episode volume in each category, including the patient’s own cancer type. Because volume in the index cancer type is closely related to our measure of subspecialization, this specification is potentially conservative, as it may absorb part of the variation in specialization. Nevertheless, our estimates remain largely unchanged (Panel C of Appendix Table E10), suggesting that our results are not driven by differences in oncologist volume. Third, we augment Equation 3 by allowing for ZCTA-specific linear time trends. Although the estimated effects are slightly attenuated, they remain close to our baseline results (Panel D of Appendix Table E10), indicating that our findings are not driven by differential trends across locations.

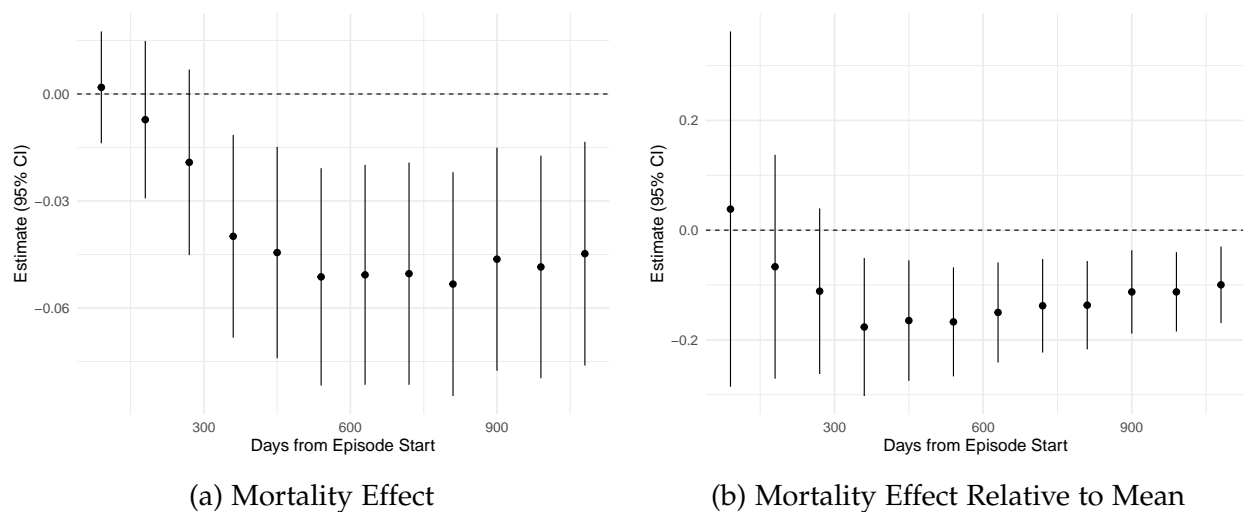


Figure 8: Effect of Subspecialist Access on Mortality

Note: The figure presents the effect of access to a subspecialized oncologist of the relevant cancer type on mortality, estimated using two-stage least squares (2SLS) in the sample of first chemotherapy episodes from 2008 to 2017. Each point reflects a separate regression estimated with the full set of controls and fixed effects specified in Equation 3. The x-axis denotes mortality measured in quarterly intervals (every 90 days), and the y-axis displays the corresponding 2SLS point estimates with 95% confidence intervals, based on standard errors clustered at the ZCTA level. Panel A plots the raw mortality estimates. Panel B rescales the estimates relative to the mean mortality rate at each time interval.

Finally, we consider an alternative treatment definition equal to one if the episode-attributed oncologist is a subspecialist of the relevant cancer type. This shifts the interpretation from access to subspecialized care to realized episode attribution to a subspecialist. Under this definition, estimated mortality effects are slightly larger, consistent with a more direct patient–physician linkage (see Panel E Appendix Table E10), and may reflect a modest change in the composition of compliers.

To address concerns that observed mortality improvements could reflect selective entry into chemotherapy rather than differences in care quality, we conduct a complementary analysis using a new sample of over 4.7 million first oncology consultation visits (Appendix A). This analysis allows us to examine whether subspecialists differentially select patients into chemotherapy—a margin not captured in our main specification, which conditions on treatment receipt. If subspecialists were more likely to initiate chemotherapy among patients with better underlying prognosis, this could generate spurious survival differences within the treated sample. We find no evidence that subspecialists are more likely to initiate chemotherapy within 90, 180, or 360 days following the first visit, suggesting no differential selection on the extensive margin.

Most importantly, we find no evidence of mortality effects following subspecialist consultations that are not followed by chemotherapy, and we replicate our main survival effects when restricting the consultation sample to visits followed by chemotherapy.²⁰ Taken together, these findings suggest that the survival benefits of care from subspecialized oncologists are unlikely to be driven by selective treatment of healthier patients, but instead reflect differences in how chemotherapy is delivered and how subspecialized oncologists treat patients relative to general oncologists.

5.2 Episode Spending

We next examine subspecialist access and its impact on chemotherapy episode spending. We construct comprehensive episode-level spending measures that include Medicare payments, beneficiary out-of-pocket payments, 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 B provides details on measure construction and descriptive

²⁰Restricting the consultation sample to visits followed by chemotherapy does not produce a sample identical to the chemotherapy episode sample. First, initial cancer-related visits are not always with medical oncologists (e.g., patients may first see a urologist, surgeon, or advanced practice provider) but may still go on to receive chemotherapy from an oncologist. Second, some initial consultations are not observed in claims data. Third, classifying physicians as subspecialists requires prior claims history, so some oncologists cannot be categorized at the time of the first visit. As a result, the two samples capture overlapping but not identical patient populations.

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 per episode grew from 8 percent in 2008 to 18 percent in 2020, consistent with increasing use of oral chemotherapy agents (Kyle, Dusetzina and Keating, 2022).

Table 1: Access to Subspecialized Oncologist and Log Spending

	Total	Part A	Part B	Part D
Panel A: First Stage				
$\sinh^{-1}(\text{DD})$	-0.024*** (0.001)	-0.024*** (0.001)	-0.024*** (0.001)	-0.024*** (0.001)
Panel B: Reduced Form				
$\sinh^{-1}(\text{DD})$	0.001 (0.001)	0.003 (0.003)	0.000 (0.001)	-0.001 (0.003)
Panel C: Structural Form				
Any Office Visit Subs.	0.113*** (0.002)	0.189*** (0.008)	0.132*** (0.002)	0.064*** (0.007)
Panel D: 2SLS				
Any Office Visit Subs.	-0.025 (0.037)	-0.145 (0.134)	-0.001 (0.043)	0.062 (0.117)
Adj. R ²	0.419	0.287	0.354	0.154
Observations	2,165,007	2,165,007	2,165,007	2,165,007
Mean Dep. Var.	10.07	3.78	9.59	5.16
F-Stat (1st Stage)	1,823	1,823	1,823	1,823

Notes: The table provides estimates on the effect of access to subspecialized oncologists on different measures of the natural logarithm of spending for chemotherapy episodes in our main sample. Column 1 provides estimates for total spending, column 2 Part A spending, column 3 Part B spending and column 4 Part D spending. Panel A presents first stage estimates, Panel B the respective reduced form estimates, Panel C the structural form estimates and Panel D provides 2SLS 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. First-stage strength is reported using the Kleibergen-Paap F-statistic. Standard errors are clustered at the ZCTA level. Signif. Codes: ***: 0.01, **: 0.05, *: 0.1.

Table 1 reports the estimated effect of access to a subspecialized oncologist on various measures of spending. We find no statistically significant impact on the natural logarithm of total spending at the episode level. When disaggregating by spending source, access to subspecialists is not significantly associated with changes in the natural logarithm of Part A, Part B, or Part D spending. However, while the 2SLS point estimates for Part A and Part B spending are negative and imprecisely estimated, they

contrast with the positive and statistically significant associations observed in OLS models. This divergence suggests that subspecialists may be more likely to treat patients with more complex or costly cancers, and that the null effects in the IV models likely reflect differences in treatment approach rather than differences in patient severity.

To isolate chemotherapy-related spending, we use HCPCS codes from the Oncology Care Model (OCM) to construct a measure of Part B chemotherapy drug spending. In addition, we apply the 2024 Restructured BETOS Classification System (RBCS) to identify injection and infusion services. These decompositions show that cost savings are concentrated in these two components: OCM-defined chemotherapy drug spending declines by approximately \$2,568, and injection and infusion services fall by about \$700 (Appendix Table E1). Despite these reductions, we find no overall effect on aggregate Part B spending. Importantly, these increases do not arise from other RBCS categories, but from a set of HCPCS codes that remain uncategorized under the RBCS classification, which collectively offset the reductions observed in chemotherapy drugs and infusion-related services in the aggregate Part B spending analysis.

5.3 Robustness

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. We have provided evidence for the fulfilment of the relevance, monotonicity and instrument independence assumption. One aspect we have so far not tested relates to the exclusion restriction, the fact that the instrument should affect outcomes (e.g. mortality) only through access to a subspecialist and not through alternative pathways (e.g. quality of locations where subspecialists practice).

To assess the validity of our exclusion restriction, we conduct additional tests. We reassign differential distances based on the nearest subspecialist for an unrelated cancer type. For example, we assign breast cancer patients the differential distance to the nearest gastrointestinal cancer subspecialist. We conduct this reassignment across multiple cancer-type pairings and re-estimate Equation 3 using 1,080-day mortality as the outcome (see Appendix Figure D7). The logic of this test is straightforward: if cancer-type-specific expertise is the key mechanism, then proximity to subspecialists of unrelated cancer types should not systematically affect mortality. Moreover, if mortality effects are instead driven by features of the broader practice environment—such as being treated at a high-quality hospital—we would expect to see similar effects regardless of the oncologist’s cancer type specialization. Consistent with the specificity of expertise as the main mechanism, we find no evidence of systematic mortality effects when using differential

distance to subspecialists in unrelated cancer types.

In addition to the placebo reassignment exercise above, we implement a complementary negative control instrument test following [Danieli et al. \(2026\)](#). Specifically, we construct a placebo instrument based on differential distance to subspecialists of unrelated cancer types and estimate its association with mortality conditional on the true instrument, controls, and fixed effects. This test evaluates whether residual variation in proximity to oncology providers—unrelated to the patient’s cancer type—predicts outcomes once the relevant variation of the true instrument is accounted for. Although differential distances across specialties are correlated due to shared geography and provider supply, they are not perfectly collinear. If broader features of the local care environment, such as general oncology capacity or hospital quality, drive our results, this residual variation could remain predictive of mortality. We present results for this falsification test in [Appendix Figure D8](#) where we find no evidence of such an association, supporting the validity of the exclusion restriction.

We further examine placebo outcomes that are unlikely to be directly affected by oncology care, including acute myocardial infarction, hip fracture, and stroke in the two years following chemotherapy initiation. While cancer is a systemic disease and its treatment can have wide-ranging effects, these acute vascular and orthopedic conditions are clinically unlikely to be closely related to cancer itself or its management. [Appendix Table E13](#) reports no statistically significant effects of subspecialist access on these outcomes. The estimates are small and imprecisely estimated, and where marginal significance arises, the sign does not align with a general improvement in health outcomes. Although these outcomes could, in principle, vary with overall hospital quality—given evidence that higher-quality hospitals attract more patients and deliver better outcomes across conditions ([Chandra et al., 2016](#))—we find no evidence of such patterns in our data.

Taken together, these findings support the exclusion restriction by indicating that differential distance affects mortality primarily through access to subspecialized oncologists with cancer-specific expertise. Consistent with this interpretation, we find no evidence that broader features of the care environment lead to improvements in outcomes unrelated to cancer.

5.4 Complier Characteristics

Our instrumental variable estimates identify LATEs for the subgroup of compliers—patients whose likelihood of receiving care from a subspecialized oncologist is affected by differential distance. To characterize this group, we follow [Gruber et al.](#)

(2025), estimating first-stage relationships within subgroups defined by cancer type, demographics, comorbidities, and other characteristics. We then construct kappa weights to quantify each subgroup’s contribution to the complier population and report reweighted means.

Table E14 shows that compliers are broadly similar to the overall sample across most observable characteristics. A useful summary of baseline health is predicted mortality. Compliers are slightly underrepresented in both the lowest- and highest-risk groups and more concentrated in the middle of the predicted mortality distribution, indicating that marginal patients affected by differential distance are neither the healthiest nor the sickest beneficiaries. This pattern suggests that the instrument primarily shifts access for patients with intermediate risk profiles, rather than selecting strongly on baseline health.

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 enrollment in clinical trials, the number of years since approval of chemotherapy drugs received by patients, the role of end-of-life care, and the health care provider mix as a marker for potential care fragmentation. Second, we analyze the effects of an oncologists’ degree of subspecialization on patient outcomes.

6.1 Health Care Utilization

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. To assess this, we examine beneficiary enrollment in clinical trials, using Medicare claims data from 2014 onward—when reporting of national 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 the Medicare data, we incorporate information from ClinicalTrials.gov, focusing on non-observational trials and their primary conditions. We use OpenAI’s GPT-4 to classify trials into our broader cancer categories, allowing us to

distinguish between overall cancer trial enrollment and enrollment in trials specific to a patient’s cancer type.²¹ This classification enables us to assess whether access to subspecialists increases general clinical trial participation or selectively improves access to trials that are more directly relevant to a patient’s diagnosis. Appendix C provides additional details on the classification methodology.

Table 2: Clinical Trial Enrollment and Access to Subspecialized Oncologists

	Cancer Trials				
	Any	Concordant	Discordant	Unspecified	Non-Cancer
Panel A: First Stage					
$\sinh^{-1}(\text{DD})$	-0.025*** (0.001)	-0.025*** (0.001)	-0.025*** (0.001)	-0.025*** (0.001)	-0.025*** (0.001)
Panel B: Reduced Form					
$\sinh^{-1}(\text{DD})$	-0.001*** (0.000)	0.000*** (0.000)	0.000 (0.000)	0.000 (0.000)	0.000 (0.000)
Panel C: Structural Form					
Any Office Visit Subs.	0.046*** (0.001)	0.041*** (0.001)	0.006*** (0.000)	0.002*** (0.000)	0.001*** (0.000)
Panel D: 2SLS					
Any Office Visit Subs.	0.023*** (0.007)	0.020** (0.006)	0.003 (0.002)	-0.001 (0.002)	0.001 (0.001)
Adj R ²	0.025	0.022	0.006	0.001	0.000
Observations	1,122,816	1,122,816	1,122,816	1,122,816	1,122,816
Mean Dep. Var.	0.021	0.018	0.003	0.001	0.001
F-Stat (1st Stage)	1,167	1,167	1,167	1,167	1,167

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 concordant to the patients cancer, column 3 any enrollment in a cancer trial discordant to the patients cancer, column 4 provides results for enrollment in in unspecified or multi-cancer trials and column 5 provides for enrollment in non cancer trials. Panel A presents first stage estimates, Panel B the respective reduced form estimates, Panel C the structural form estimates and Panel D provides 2SLS estimates. All models include demographic, ZCTA level and chronic conditions controls as well fixed effects for the beneficiaries’ ZCTA and cancer type by year. Standard errors are clustered at the ZCTA level. First-stage strength is reported using the Kleibergen-Paap F-statistic. Signif. Codes: ***: 0.01, **: 0.05, *: 0.1.

Patients with access to subspecialized oncologists are substantially more likely to enroll in clinical cancer trials. While overall enrollment rates are low, episodes attributed to general oncologists are linked to a cancer trial in roughly one percent of cases, with even fewer enrolling in trials specific to the patient’s cancer type. In contrast, episodes

²¹Some trials are multi-cancer and not specific to a single cancer type.

attributed to subspecialists are linked to clinical cancer trials at approximately five to six times this rate. Enrollment in unspecified cancer trials and non-cancer trials remains low for both groups (Appendix Figure D9).

To assess whether clinical trial enrollment is causally driven by access to subspecialists rather than selection into treatment, we estimate Equation 3, using as an outcome a binary indicator for whether a beneficiary enrolled in a clinical trial within the year of chemotherapy initiation.²² Table 2 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 D shows that access to a subspecialist of the relevant cancer type increases enrollment in any cancer trial by 2.3 percentage points, a 110 percent increase relative to the sample mean. The effect is similar in magnitude for cancer-type concordant trials, with an estimated increase of 2 percentage points, or 111 percent relative to the mean. In contrast, estimates for discordant, unspecified, and non-cancer trials are small and not statistically significant. While the corresponding structural form estimates are statistically significant, the point estimates are also close to zero, and the difference in significance largely reflects the greater precision of the structural form estimates. Relatively few Medicare beneficiaries enroll in clinical trials, and thus these findings are unlikely to explain the entire mortality benefits of subspecialized care; these findings reflect a key difference in healthcare utilization between individuals with access to subspecialized oncologists that may contribute modestly to the differences. Importantly it also suggests clear differences in practice patterns of subspecialized oncologists.

6.1.2 Age of Cancer Drugs

To assess whether differences in treatment choices contribute to the observed survival gains, we examine the age of cancer drugs administered during treatment. Subspecialized oncologists may be more likely to use newer therapies, which in some cases represent meaningful clinical advances, although not all recently approved treatments are necessarily more effective. In particular, certain innovations, such as immune checkpoint inhibitors, have been transformative (Horn et al., 2025), but may diffuse unevenly across oncologists.

To implement and assess the age of cancer drugs administered during treatment, we linked FDA approval years—sourced from the NCI SEER Program and mapped to corresponding HCPCS and NDC codes—to Medicare claims data. We conducted two com-

²²We 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 attributed oncologists.

plementary analyses. First, we examined whether access to subspecialized oncologists increases the likelihood that a patient receives a chemotherapy drug approved within the two years prior to treatment initiation. Second, we calculated the average number of years since approval for all chemotherapy drugs administered in the year of treatment initiation. For Part B drugs, we used a weighted average based on the number of claim lines per HCPCS code; for Part D drugs, we weighted by the number of prescription fills per NDC code. Due to limitations in linking approval dates to cancer-specific indications and the absence of detailed clinical information in Medicare claims, we focus on the first approval year of the generic substance rather than indication-specific approvals.

Appendix Table E15, Panel D, presents 2SLS estimates for the effect of subspecialist access on the probability of receiving a newly approved cancer drug (within two years of treatment initiation). Column 1 shows a marginally significant 0.2 percentage point increase in the probability of receiving a newly approved chemotherapy drug—corresponding to a 6.9 percent increase relative to the mean. While no significant effect is observed for newly approved Part B drugs (Column 2), we detect a marginally significant increase in the use of recently approved Part D drugs (Column 3). Together, these results provide suggestive evidence that subspecialist access may modestly increase the likelihood of receiving more recently approved therapies.

Appendix Table E16 reports 2SLS estimates for the effect of subspecialist access on the average approval age of chemotherapy drugs. Panel D shows no statistically significant effects on the weighted average age of drugs overall (Column 1), for Part B drugs (Column 2), or for Part D drugs (Column 3). These findings should be interpreted with caution, as the weighting methodology does not capture differences in drug dosage or treatment intensity, which likely limits the accuracy of this average age measure.

Taken together, the results suggest that while access to subspecialized oncologists does not substantially shift the overall age profile of chemotherapy drugs, it may slightly increase the likelihood of receiving more recently approved treatments, particularly oral agents covered under Part D.

6.1.3 Provider Mix and Fragmentation

Next, we assess whether access to subspecialized oncologists affects the composition and diversity of providers visited by patients who are undergoing 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: (i) the total number of office visits, (ii) the number of unique providers seen, and (iii) the number of unique provider specialties. We estimate the effect of subspecialist access on each

outcome using our main instrumental variable strategy (Equation 3).

Results in Panel D of Appendix Table E17 show that subspecialist access has no discernible effect on the number of office visits or the diversity of unique provider specialties for which patients have office visits during the chemotherapy episode. However, it is associated with a modest but statistically significant 4.3% reduction in the number of unique providers relative to the mean.

These findings suggest that patients seeing subspecialists receive care from a 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 the number of different providers could suggest more streamlined care delivery among patients who visit subspecialists.

6.2 Degree of Oncologist Specialization

Our binary measure of subspecialist access captures the effect of specialization at the extensive margin, comparing patients who receive care from subspecialized oncologists to those who do not. However, this measure abstracts from important variation in the intensity of specialization across oncologists. To capture this variation, we move beyond a binary classification and construct a continuous measure of oncologist specialization. Specifically, we compute oncologist-level Herfindahl–Hirschman Indices (HHI) based on the distribution of chemotherapy episodes across cancer types. This measure reflects the degree to which an oncologist’s practice is concentrated within a narrow set of cancers, with higher values indicating greater specialization.

Importantly, this approach leverages a more granular classification of cancer types, distinguishing between 45 categories rather than the five broad groups used to classify subspecialists (see Appendix Table E2). This allows us to capture meaningful differences in specialization within cancer groups—for example, between oncologists focusing on specific gastrointestinal cancers—rather than treating all subspecialists within a broad category as homogeneous. The HHI is defined as follows:

$$\text{HHI}_i = \sum_{c=1}^C \left(\frac{n_{ic}}{\sum_{c=1}^C n_{ic}} \right)^2 \quad (4)$$

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 n_{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.²³

In Panel D of Table 3, we present 2SLS estimates of Equation 3, replacing our binary access measure with the care-coordinating physician's HHI as the treatment variable. 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 episode attributable oncologist's HHI is not statistically significant within the first 180 days but leads to significant mortality reductions thereafter. For instance, a 0.1 HHI point increase reduces 1,080-day mortality by 1.4 percentage points, corresponding to a 3.1 percent reduction relative to the respective mean mortality rate. For context, the average difference in HHI between a subspecialized and a general oncologist is 0.4 HHI points. Scaling our point estimate using this difference suggests a 5.6 percentage point decline in 1,080-day mortality, which is larger than the corresponding estimate in Figure 8, Panel 8a, suggesting potential non-linearities in the relationship between HHI and mortality. OLS estimates of the structural form in Panel C of Table 3 suggest smaller mortality reductions for the same increase in HHI, the degree of physician specialization remains an important determinant of patient survival.

²³Appendix Figure D10 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 percentiles—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.

Table 3: Mortality Effects of Oncologist Specialization

	180-Day	360-Day	720-Day	1080-Day
Panel A: First Stage				
$\sinh^{-1}(\text{DD})$	-0.008*** (0.000)	-0.008*** (0.000)	-0.008*** (0.000)	-0.008*** (0.000)
Panel B: Reduced Form				
$\sinh^{-1}(\text{DD})$	-0.000 (0.000)	0.001*** (0.000)	0.001*** (0.000)	0.001*** (0.000)
Panel C: Structural Form				
HHI	-0.014*** (0.001)	-0.024*** (0.001)	-0.039*** (0.002)	-0.048*** (0.002)
Panel D: 2SLS				
HHI	-0.023 (0.036)	-0.126*** (0.046)	-0.159*** (0.050)	-0.141** (0.051)
Adj R ²	0.120	0.224	0.302	0.325
Observations	1,681,119	1,681,119	1,681,119	1,681,119
Mean Dep. Var.	0.108	0.226	0.365	0.449
F-Stat (1st Stage)	715	715	715	715

Notes: This table reports estimates of the effect of the degree of cancer type subspecialization of the chemotherapy care coordinating oncologist on mortality for all first chemotherapy episodes between 2008 and 2017. Panel A shows first stage estimates. Panel B shows the reduced form estimates. Panel C shows the structural form estimates, and Panel D provides the 2SLS estimates. All models control for demographics, ZCTA characteristics, comorbidities, and include ZCTA and cancer type-by-year fixed effects. Standard errors are clustered at the ZCTA level. First-stage strength is reported using the Kleibergen-Paap F-statistic. Significance levels: *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$.

7 Conclusion

This paper provides causal evidence on the returns to specialization in a high-skill professional setting. Focusing on medical oncology, we show that access to subspecialized oncologists improves medium- and long-term survival without increasing episode-level spending. Leveraging quasi-random variation in differential distance to subspecialists versus general oncologists, we estimate that access to a subspecialist in the patient’s relevant cancer type reduces 1,080-day mortality by approximately 10 percent relative to the mean. These effects appear to operate through domain-specific expertise: we find no impact on non-cancer outcomes, and falsification tests assigning patients distances to subspecialists of unrelated cancer types yield no corresponding mortality effects.

The results are robust across specifications and supported by a range of validation exercises consistent with the identifying assumptions. Mechanism analyses suggest that subspecialists differ in treatment choices, with higher rates of enrollment in cancer-

specific clinical trials and greater use of newer therapies. While these channels explain only part of the observed survival gains, they point to meaningful differences in how care is delivered. At the same time, we find no evidence that subspecialization increases care fragmentation, suggesting that improved outcomes do not come at the cost of more dispersed care.

We further show that the benefits of specialization extend along a continuum: patients treated by more narrowly focused oncologists—measured by the concentration of treated cancer types—experience better outcomes. This pattern reinforces the interpretation that deeper clinical focus, rather than discrete subspecialty labels alone, drives improvements in care.

Despite these gains, access to subspecialized oncology care remains uneven. Utilization of subspecialists is disproportionately concentrated in higher-income areas (Karadkic et al., 2025), whereas patients in areas with the highest cancer mortality rates are less likely to receive such care. This mismatch between need and access raises important policy questions about how to expand the availability of specialized expertise while preserving equity in care delivery.

More broadly, our findings contribute to the economics of specialization and the division of labor. In contrast to settings where specialization may introduce fragmentation or inefficiencies, we show that in complex medical environments, deeper specialization can generate substantial improvements in outcomes without increasing costs. These results highlight the value of domain-specific expertise and have implications for how healthcare systems organize care, train physicians, and allocate specialized resources.

References

- Agha, Leila, and David Molitor.** 2018. "The local influence of pioneer investigators on technology adoption: evidence from new cancer drugs." *Review of Economics and Statistics*, 100(1): 29–44.
- Alsan, Marcella, Maya Durvasula, Harsh Gupta, Joshua Schwartzstein, and Heidi L Williams.** 2022. "Representation and Extrapolation: Evidence from Clinical Trials." National Bureau of Economic Research.
- Audirac, Michelle.** 2024. "Zip2zcta master xwalk."
- Avdic, Daniel, Petter Lundborg, and Johan Vikström.** 2019. "Estimating returns to hospital volume: Evidence from advanced cancer surgery." *Journal of health economics*, 63: 81–99.
- Baicker, Katherine, and Amitabh Chandra.** 2004. "The Productivity of Physician Specialization: Evidence from the Medicare Program." *American Economic Review*, 94(2): 357–361.
- Baumgardner, James R.** 1988. "The division of labor, local markets, and worker organization." *Journal of Political Economy*, 96(3): 509–527.
- Becker, Gary S, and Kevin M Murphy.** 1992. "The division of labor, coordination costs, and knowledge." *The Quarterly journal of economics*, 107(4): 1137–1160.
- Bellemare, Marc F, and Casey J Wichman.** 2020. "Elasticities and the inverse hyperbolic sine transformation." *Oxford Bulletin of Economics and Statistics*, 82(1): 50–61.
- Birkmeyer, John D, Andrea E Siewers, Emily VA Finlayson, Therese A Stukel, F Lee Lucas, Ida Batista, H Gilbert Welch, and David E Wennberg.** 2002. "Hospital volume and surgical mortality in the United States." *New England Journal of Medicine*, 346(15): 1128–1137.
- Card, David, Alessandra Fenizia, and David Silver.** 2023. "The Health Impacts of Hospital Delivery Practices." *American Economic Journal: Economic Policy*, 15(2): 42–81.
- Carroll, Caitlin E, Mary Beth Landrum, Alexi A Wright, and Nancy L Keating.** 2023. "Adoption of innovative therapies across oncology practices—evidence from immunotherapy." *JAMA oncology*, 9(3): 324–333.

- Cebul, Randall D., James B. Rebitzer, Lowell J. Taylor, and Mark E. Votruba.** 2008. "Organizational Fragmentation and Care Quality in the U.S. Healthcare System." *Journal of Economic Perspectives*, 22(4): 93–113.
- Chan, David C, and Yiqun Chen.** 2022. "The Productivity of Professions: Evidence from the Emergency Department." National Bureau of Economic Research.
- Chandra, Amitabh, Amy Finkelstein, Adam Sacarny, and Chad Syverson.** 2016. "Health Care Exceptionalism? Performance and Allocation in the US Health Care Sector." *American Economic Review*, 106(8): 2110–44.
- Chandra, Amitabh, and Douglas O Staiger.** 2007. "Productivity spillovers in health care: evidence from the treatment of heart attacks." *Journal of political Economy*, 115(1): 103–140.
- Chandra, Amitabh, and Jonathan Skinner.** 2012. "Technology growth and expenditure growth in health care." *Journal of Economic Literature*, 50(3): 645–680.
- CMS.** 2014. "Medicare Claims Processing Manual, Pub 100-04, Transmittal 2955." Baltimore, MD, Department of Health Human Services (DHHS), Change Request 8401: Mandatory Reporting of an 8-Digit Clinical Trial Number on Claims.
- CMS.** 2020. "OCM Performance-Based Payment Methodology." <https://www.cms.gov/priorities/innovation/files/x/ocm-cancerodelist.pdf>, Accessed 2023.
- CMS.** 2021. "27 CCW Chronic Conditions (1999–2021)." <https://www2.ccwdata.org/web/guest/condition-categories-chronic>, Accessed 12 March 2026.
- CMS.** 2024. "Restructured BETOS Classification System." <https://data.cms.gov/provider-summary-by-type-of-service/provider-service-classifications/restructured-betos-classification-system>, Accessed June 2025.
- CMS.** 2025. "Oncology Care Model." <https://www.cms.gov/priorities/innovation/innovation-models/oncology-care>, Accessed July 2025.
- Coleman, James, Elihu Katz, and Herbert Menzel.** 1957. "The diffusion of an innovation among physicians." *Sociometry*, 20(4): 253–270.
- Currie, Janet, and Hannes Schwandt.** 2016. "Mortality inequality: the good news from a county-level approach." *Journal of Economic Perspectives*, 30(2): 29–52.
- Cutler, David M, and Mark McClellan.** 2001. "Is technological change in medicine worth it?" *Health affairs*, 20(5): 11–29.

- Danieli, Oren, Daniel Nevo, Itai Walk, Bar Weinstein, and Dan Zeltzer.** 2026. "Negative Control Falsification Tests for Instrumental Variable Designs." *American Economic Review*, 116(4): 1380–1414.
- Dingel, Jonathan I, Joshua D Gottlieb, Maya Lozinski, and Pauline Mourot.** 2023. "Market Size and Trade in Medical Services." National Bureau of Economic Research.
- Emens, Leisha A, Paolo A Ascierio, Phillip K Darcy, Sandra Demaria, Alexander MM Eggermont, William L Redmond, Barbara Seliger, and Francesco M Marincola.** 2017. "Cancer immunotherapy: opportunities and challenges in the rapidly evolving clinical landscape." *European journal of cancer*, 81: 116–129.
- Garicano, Luis.** 2000. "Hierarchies and the Organization of Knowledge in Production." *Journal of political economy*, 108(5): 874–904.
- Gruber, Jonathan, David H Howard, Jetson Leder-Luis, and Theodore L Caputi.** 2025. "Dying or Lying? For-Profit Hospices and End-of-Life Care." *American Economic Review*, 115(1): 263–294.
- Halm, Ethan A, Clara Lee, and Mark R Chassin.** 2002. "Is volume related to outcome in health care? A systematic review and methodologic critique of the literature." *Annals of internal medicine*, 137(6): 511–520.
- Horn, Danae, Abby E Alpert, Mark Duggan, and Mireille Jacobson.** 2025. "The Impact of Immunotherapy on Reductions in Cancer Mortality: Evidence from Medicare." National Bureau of Economic Research Working Paper 34317.
- Huckman, Robert S, and Gary P Pisano.** 2006. "The firm specificity of individual performance: Evidence from cardiac surgery." *Management science*, 52(4): 473–488.
- Kaiser Family Foundation.** 2025. "What to Know About Medicare Spending and Financing." <https://www.kff.org/medicare/issue-brief/what-to-know-about-medicare-spending-and-financing/>, Accessed: 2025-02-27.
- Karadakic, René, Christopher Manz, Arno Cai, David C Chan, Bruce E Landon, Jukka-Pekka Onnela, Nancy L Keating, and Michael L Barnett.** 2025. "Geographic Variation in the Utilization of Cancer Care From Subspecialized Medical Oncologists in the United States, 2008 to 2020." *Annals of internal medicine*.
- Keating, Nancy L, Shalini Jhatakia, Gabriel A Brooks, Amanda S Tripp, Inna Cintina, Mary Beth Landrum, Qing Zheng, Thomas J Christian, Roberta Glass, Colleen M**

- Kummet, et al.** 2021. "Association of participation in the oncology care model with Medicare payments, utilization, care delivery, and quality outcomes." *Jama*, 326(18): 1829–1839.
- Kyle, Michael Anne, Stacie B Dusetzina, and Nancy L Keating.** 2022. "Evaluation of trends in oncology drug spending in Medicare, 2016 to 2020." *JAMA Network Open*, 5(7): e2221468.
- Lozinski, Maya.** 2024. "Knowledge Growth and Specialization: Evidence from Oncologists." <https://dx.doi.org/10.2139/ssrn.4960603>.
- Mailankody, Sham, and Vinay Prasad.** 2015. "Five years of cancer drug approvals: innovation, efficacy, and costs." *JAMA oncology*, 1(4): 539–540.
- Mariotto, Angela B, Lindsey Enewold, Jingxuan Zhao, Christopher A Zeruto, and K Robin Yabroff.** 2020. "Medical care costs associated with cancer survivorship in the United States." *Cancer epidemiology, biomarkers & prevention*, 29(7): 1304–1312.
- McClellan, Mark, Barbara J McNeil, and Joseph P Newhouse.** 1994. "Does more intensive treatment of acute myocardial infarction in the elderly reduce mortality?: analysis using instrumental variables." *Jama*, 272(11): 859–866.
- Milligan, Michael, Parsa Erfani, E John Orav, Stephen Schleicher, Gabriel A Brooks, and Miranda B Lam.** 2024. "Practice consolidation among US medical oncologists, 2015-2022." *JCO oncology practice*, 20(6): 827–834.
- National Bureau of Economic Research.** 2025. "ZIP Code Distance Database." <https://www.nber.org/research/data/zip-code-distance-database>, Accessed: 2025-02-27.
- National Cancer Institute.** 2025a. "Age and Cancer Risk." U.S. Department of Health and Human Services, National Institutes of Health.
- National Cancer Institute.** 2025b. "Observational Research in Oncology Toolbox: Cancer Medications Enquiry Database." <https://seer.cancer.gov/oncologytoolbox/>, Accessed 2025.
- Pantziarka, Pan, Rica Capistrano I, Arno De Potter, Liese Vandeborne, and Gauthier Bouche.** 2021. "An open access database of licensed cancer drugs." *Frontiers in pharmacology*, 12: 627574.
- Rosen, Sherwin.** 1983. "Specialization and human capital." *Journal of Labor Economics*, 1(1): 43–49.

- Sahni, Nikhil R, Maurice Dalton, David M Cutler, John D Birkmeyer, and Amitabh Chandra.** 2016. "Surgeon specialization and operative mortality in United States: retrospective analysis." *Bmj*, 354.
- Schwandt, Hannes, Janet Currie, Marlies Bär, James Banks, Paola Bertoli, Aline Bütikofer, Sarah Cattan, Beatrice Zong-Ying Chao, Claudia Costa, Libertad González, et al.** 2021. "Inequality in mortality between Black and White Americans by age, place, and cause and in comparison to Europe, 1990 to 2018." *Proceedings of the National Academy of Sciences*, 118(40).
- Sharma, Padmanee, and James P Allison.** 2015. "Immune checkpoint targeting in cancer therapy: toward combination strategies with curative potential." *Cell*, 161(2): 205–214.
- Smith, Adam.** 1819. *An inquiry into the nature and causes of the wealth of nations*. London:Printed for William Allason, and J. Maynard, and W. Blair.
- Yale Cancer Clinic.** 2025. "Delivering Cancer Care in our Communities." <https://medicine.yale.edu/news-article/delivering-cancer-care-in-our-communities/>, Accessed: 2025-07-11.

A Selection into Chemotherapy

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

To address this concern, we construct a complementary sample designed to capture the first point of contact between patients and medical oncologists. Specifically, we identify beneficiaries' first office visit with a medical oncologist between 2008 and 2020 in which the claim includes a cancer diagnosis corresponding to one of the major cancer types in our main analysis (breast, gastrointestinal, hematologic, prostate/genitourinary, or thoracic cancers). We retain only the earliest such visit per beneficiary. This "first visit" sample serves two purposes. First, it approximates the initial clinical encounter at which patients and oncologists discuss and decide on potential systemic therapy, capturing the key decision point preceding treatment initiation. Second, by focusing on the first oncologist visit associated with a relevant cancer diagnosis, we reduce the likelihood of including routine follow-up or surveillance visits that do not reflect an active treatment decision.

Table A1: Sample Construction First Visits

Sample Restriction	Nr. First Visits (remaining)
1. First Office Visits	6,940,287
2. Restrict to relevant cancer types	5,553,641
3. Restrict to visits with classified medical oncologist	5,287,063
4. Restrict to observations with valid ZCTA assignment	5,278,001
5. Remove negative differential distances	5,224,729
6. Remove outlier distances (>95th percentile)	4,805,241
7. Remove observations with missing controls	4,711,907

Notes: This table describes the construction of the analysis sample of first office visits with a medical oncologist using Medicare claims data from 2008 to 2020. Sample restrictions are applied sequentially as listed in the first column, and the second column reports the number of observations remaining after each step. ZCTA denotes ZIP Code Tabulation Area. Step 2 restricts the sample to visits associated with the cancer types included in the analysis (breast, gastrointestinal, hematologic, prostate/genitourinary, and thoracic cancers). Step 3 restricts to visits with medical oncologists who can be classified as either subspecialists or generalists. Step 4 restricts to observations with a valid ZCTA assignment. Step 5 excludes observations with negative differential distance (i.e., where the subspecialist is closer than the generalist). Step 6 excludes distance outliers where either the distance to the nearest subspecialist or generalist exceeds the 95th percentile of the annual distance distribution. Step 7 excludes observations with missing values in control variables.

Table A1 presents the sample construction process, including all exclusion criteria and the number of observations retained at each step. After restricting to the main cancer types, limiting the sample to classified medical oncologists,²⁴ and excluding outliers, the final sample consists of 4,711,907 first office visits with medical oncologists.

We then link the first-visit sample to the chemotherapy episode data.²⁵ This linkage allows us to determine whether a patient’s initial oncologist visit is followed by chemotherapy initiation. We do not require the cancer diagnosis recorded at the first visit to match the primary diagnosis in the chemotherapy sample. While most visits that lead to chemotherapy involve the same cancer type, discrepancies may arise due to coding differences or updates in diagnostic classification. Among all first office visits with a medical oncologist, 32.3 percent are followed by chemotherapy within 360 days. In a final step, we enrich the data with measures of oncologist subspecialization, beneficiary characteristics (including demographics and chronic conditions), and ZCTA-level variables. We also construct measures of geographic access—specifically, the distance to the nearest generalist and subspecialized oncologist for the relevant cancer type—using the same approach as in the main analysis. We then follow an identification strategy equivalent to our main analysis concerning chemotherapy episodes.

$$\text{Visit}_i = \alpha + \beta \cdot \text{DD}_{t(i)z(i)} + \delta X_i + \tau_{t(i)} + \gamma_{z(i)} + \psi D_{t(i)z(i)} + \varepsilon_i \quad (5)$$

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 first office visit with a subspecialized oncologist of the relevant cancer type and zero otherwise. The right hand side of this first stage is equal to Equation 2 in our chemotherapy analysis.

In a second step, we estimate the effect of initial subspecialist consultation on the

²⁴Subspecialization is defined empirically on a yearly basis using chemotherapy episode volume. Oncologists not observed in the chemotherapy episode data in a given year cannot be classified as either generalists or subspecialists.

²⁵Not all first chemotherapy episodes can be linked to a corresponding first office visit as defined above. This may arise for several reasons: (i) the initial visit occurs with an oncologist who cannot be classified as a subspecialist or generalist; (ii) the diagnosis code on the claim is misclassified or does not map to our set of included cancer types; or (iii) the initial visit falls outside our observation window for office visits (e.g., prior to 2008).

probability to initiate chemotherapy. We estimate:

$$Y_i = \alpha + \beta \cdot \widehat{\text{Visit}}_i + \delta X_i + \tau_{t(i)} + \gamma_{z(i)} + \psi D_{t(i)z(i)} + \varepsilon_i \quad (6)$$

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 90, 180, or 360 days after the initial consultation).

Table A2 reports first-stage estimates for the first-visit sample, separately for the full sample, visits that lead to chemotherapy within 360 days, and visits that do not. Differential distance is a strong predictor of subspecialist access across all samples, with F-statistics well above conventional thresholds. The first-stage coefficient is slightly larger in magnitude among patients who subsequently initiate chemotherapy (-0.020) than among those who do not (-0.017). This pattern suggests that geographic access is more closely related to subspecialist use in encounters that lead to treatment. In contrast, the inclusion of visits that do not result in chemotherapy—potentially reflecting a broader and more heterogeneous set of clinical interactions—attenuates the first stage in the full sample. Importantly, however, the instrument remains strong across all subsamples, supporting its relevance in the first-visit setting.

Table A2: First Office Visits: First Stage

	Full	With Chemo \leq 360	No Chemo \leq 360
$\sinh^{-1}(\text{DD})$	-0.018*** (0.000)	-0.020*** (0.001)	-0.017*** (0.000)
ZCTA FE	Yes	Yes	Yes
Cancer-Year FE	Yes	Yes	Yes
Adj. R ²	0.124	0.128	0.116
Observations	4,711,907	1,519,912	3,190,357
Mean Dep. Var.	0.108	0.118	0.103
F-Stat (1st stage)	1,842	1,438	1,668

Notes: This table reports first-stage estimates of the relationship between the inverse hyperbolic sine of differential distance—defined as the distance to the nearest subspecialized oncologist of the relevant cancer type relative to the nearest general oncologist—and the likelihood of seeing a subspecialist at the first office visit. The dependent variable is an indicator equal to one if the first visit occurs with a subspecialist of the relevant cancer type. Column 1 presents estimates for the full sample of first office visits. Column 2 restricts the sample to visits that result in chemotherapy initiation within 360 days. Column 3 reports estimates for visits that do not result in chemotherapy within 360 days. Standard errors are clustered at the ZCTA level. Reported first-stage F-statistics are Kleibergen-Paap statistics. Signif. Codes: ***: 0.01, **: 0.05, *: 0.1.

We provide additional evidence supporting the independence assumption. Figure A1a shows that the instrument is not systematically related to observable beneficiary

characteristics once we condition on ZCTA and cancer type-by-year fixed effects. We further assess the plausibility of the monotonicity assumption in Figure A1b, which reports first-stage estimates across a range of subsamples. The consistently signed coefficients indicate that the instrument shifts access to subspecialized care in the same direction across subpopulations, providing support for the monotonicity assumption.

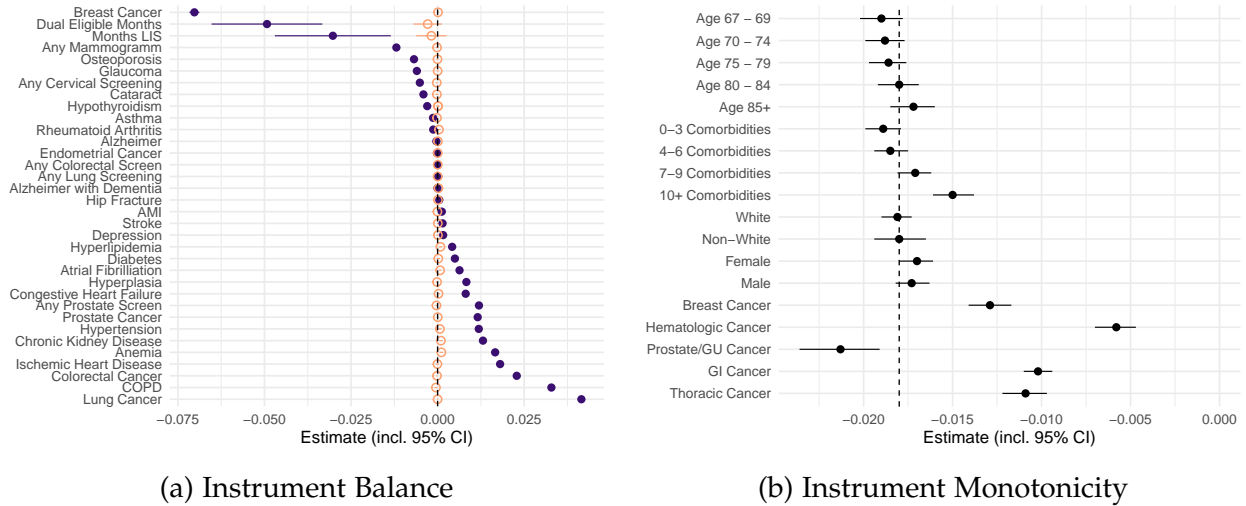


Figure A1: Instrument Balance and Monotonicity in First Visit Sample

Note: Panel A reports coefficients from separate regressions of the instrument on the beneficiary characteristics listed on the y-axis, using the full sample of first office visits. Each point corresponds to a distinct regression. Solid purple markers show unadjusted associations without controls or fixed effects, while hollow orange markers report estimates from specifications that include ZCTA and cancer type-by-year fixed effects. Panel B presents first-stage coefficients estimated across different subsamples of the first-visit sample. Each coefficient is obtained from a separate regression and displayed with 95% confidence intervals. The vertical dashed line indicates the corresponding first-stage estimate from the full sample for reference. Confidence intervals are based on heteroskedasticity-robust standard errors in the unadjusted specifications and ZCTA-clustered standard errors in the adjusted specifications.

We estimate Equation 6 using binary indicators for chemotherapy initiation within 90, 180, and 360 days following a patient’s first office visit with an oncologist as the outcome. As shown in Table A3, we find no statistically significant effect of having this first visit with a subspecialist on the likelihood of initiating chemotherapy within these time frames. This result holds for any chemotherapy initiation and initiation of chemotherapy that corresponds to the main cancer diagnosis of the first visit. These findings suggest that subspecialists do not initiate chemotherapy at higher rates than generalists.

Table A3: First Office Visits and Probability of Chemotherapy Initiation

	≤ 90	≤ 180	≤ 360
Panel A: Any Chemotherapy			
First Office Visit Subs.	0.022 (0.013)	0.013 (0.014)	0.014 (0.014)
Adj R ²	0.094	0.098	0.096
Observations	4,711,907	4,711,907	4,711,907
Mean Dep. Var.	0.266	0.299	0.323
F-Stat (1st Stage)	1,842	1,842	1,842
Panel B: Cancer Type Concordant Chemotherapy			
First Office Visit Subs.	0.021 (0.013)	0.011 (0.014)	0.011 (0.014)
Adj R ²	0.097	0.101	0.101
Observations	4,711,907	4,711,907	4,711,907
Mean Dep. Var.	0.256	0.287	0.308
F-Stat (1st Stage)	1,842	1,842	1,842

Notes: This table reports 2SLS estimates of the effect of having a first office visit with a subspecialized oncologist of the relevant cancer type on indicators for chemotherapy initiation. Each column represents a separate regression. Column 1 reports effects on initiation within 90 days, Column 2 within 180 days, and Column 3 within 360 days of the office visit. Panel A estimates effects on initiating any chemotherapy, while Panel B restricts the outcome to chemotherapy corresponding to the main diagnosis of the first visit. All models include demographic controls, ZCTA-level characteristics, indicators for chronic conditions, and fixed effects for ZCTA and cancer type-by-year. Standard errors are clustered at the ZCTA level. First-stage strength is measured using the Kleibergen–Paap F-statistic. Significance levels: *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$.

Next, we use the first office visit sample to examine the effect of a first office visit with a subspecialist on patient mortality. We construct binary indicators for mortality within 180, 360, 720, and 1080 days following a patient’s first office visit with a medical oncologist. Figure A2 presents the corresponding 2SLS estimates for four samples: (i) all first office visits from 2008 to 2017 (FV – Full), (ii) first visits followed by chemotherapy initiation (FV – With Chemo), (iii) first visits not followed by chemotherapy (FV – No Chemo), and (iv) our main sample of first chemotherapy episodes (Main Chemo). Three key patterns emerge. First, there is no statistically significant mortality effect in the short-term (up to 180-days). Second, the largest mortality reductions are consistently observed in the main chemotherapy sample. Third, while the estimates differ in magnitude across samples, the overall direction of effects is consistent, lending support to the idea that subspecialized oncologic care may improve longer-term outcomes, particularly for patients actively receiving chemotherapy. Importantly, we find no mortality effects among patients whose first visit does not result in chemotherapy initiation, supporting the notion that subspecialists are not selecting patients based on unobserved mortality

risk. This lends further credibility to our interpretation that differences in outcomes are driven by treatment pathways rather than selection into subspecialist care.

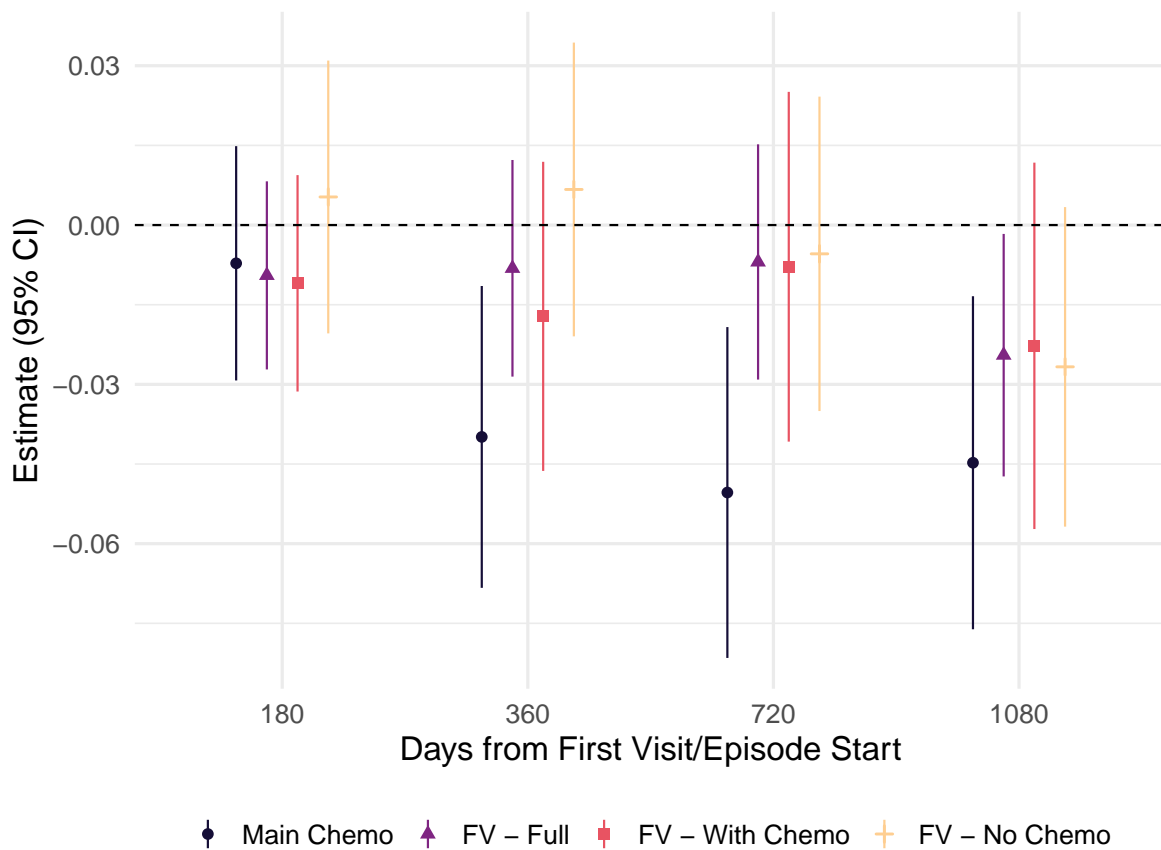


Figure A2: First Office Visit with a Subspecialist and Mortality

Note: This figure presents 2SLS estimates of the effect of a first office visit with a subspecialized oncologist on mortality, based on regressions of Equation 6. Each point reflects a separate regression for mortality measured at 180, 360, 720, and 1080 days after the initial visit. Black circles represent estimates from our main analysis sample of first chemotherapy episodes (“Main Chemo”). Purple triangles depict estimates for the full sample of first office visits (“FV – Full”). Red squares correspond to the subsample of first visits followed by chemotherapy initiation (“FV – With Chemo”), and yellow crosses show estimates for first visits not followed by chemotherapy (“FV – No Chemo”). All models include our standard set of controls: beneficiary demographics, ZCTA characteristics, chronic conditions, as well as ZCTA and cancer type-by-year fixed effects. 95% confidence intervals are based on standard errors clustered at the beneficiary ZCTA level.

As a final exercise, we implement a control function approach to assess the role of selection into the chemotherapy sample. We begin by estimating the first stage equation in the first-visit sample and retain the resulting first-stage residual for each individual. We then merge these residuals into the first chemotherapy episode sample for the corresponding beneficiaries, restricting the analysis to episodes that can be linked to a first-visit observation between 2008 and 2017. Using this linked sample, we first re-estimate

our IV model for mortality outcomes. We then augment this specification by including the first-stage residual from the first-visit sample as an additional control. This procedure allows us to assess whether unobserved factors influencing subspecialist access at the initial consultation stage affect our mortality estimates in the chemotherapy sample. Results from this exercise are reported in Appendix Table A4.

Table A4: Access to Subspecialized Oncologist and Mortality (Chemotherapy Sample)

	180-Day	360-Day	720-Day	1080-Day
Panel A: Chemotherapy Sample (2008-2017)				
Any Office Visit Subs.	-0.007 (0.011)	-0.040*** (0.014)	-0.050*** (0.016)	-0.045** (0.016)
First-Stage Residual Control	No	No	No	No
Adj. R ²	0.134	0.224	0.302	0.335
Observations	1,681,119	1,681,119	1,681,119	1,681,119
Mean Dep. Var.	0.108	0.226	0.365	0.449
F-Stat (1st Stage)	1,649	1,649	1,649	1,649
Panel B: Linked Chemotherapy Sample (2008-2017)				
Any Office Visit Subs.	-0.007 (0.014)	-0.037** (0.019)	-0.035* (0.020)	-0.029 (0.020)
First-Stage Residual Control	No	No	No	No
Adj R ²	0.117	0.220	0.303	0.328
Observations	1,138,750	1,138,750	1,138,750	1,138,750
Mean Dep. Var.	0.114	0.239	0.381	0.463
F-Stat (1st Stage)	1,329	1,329	1,329	1,329
Panel C: Linked Chemotherapy Sample (2008-2017)				
Any Office Visit Subs.	-0.006 (0.016)	-0.039* (0.020)	-0.036* (0.022)	-0.030 (0.022)
First-Stage Residual Control	Yes	Yes	Yes	Yes
Adj R ²	0.117	0.224	0.303	0.328
Observations	1,138,750	1,138,750	1,138,750	1,138,750
Mean Dep. Var.	0.114	0.239	0.381	0.463
F-Stat (1st Stage)	3,273	3,273	3,273	3,273

Notes: This table reports estimates of the effect of access to subspecialized oncologists on mortality for first chemotherapy episodes between 2008 and 2017. Panel A presents the baseline estimates using the full chemotherapy sample. Panel B reports analogous estimates for the subset of chemotherapy episodes that can be linked to the first office visit sample. Panel C augments this specification by including the first-stage residual from the first-visit sample as a control function. All models include demographic controls, ZCTA-level characteristics, and comorbidities, and incorporate ZCTA and cancer type-by-year fixed effects. Standard errors are clustered at the ZCTA level. First-stage strength is reported using the Kleibergen–Paap F-statistic. Significance levels: *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$.

The estimates in Table A4, Panel A, present our baseline results using the full chemotherapy sample for episodes between 2008 and 2017. Restricting the analysis to

the linked sample in Panel B yields coefficients that are smaller in magnitude, reflecting differences in sample composition, but remain qualitatively similar and generally statistically significant. Importantly, augmenting the specification with the first-stage residuals from the first visit sample in Panel C has little impact on the estimated effects of subspecialist access on mortality. If anything, the coefficients increase slightly in magnitude, suggesting that unobserved factors influencing selection into subspecialist care at the initial consultation stage do not materially drive our results. Taken together, these findings indicate that our estimates are robust to sample restrictions and provide no evidence that selection into the chemotherapy sample biases the estimated effects of subspecialist access on mortality.

B 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 B2. In order to better understand drivers of spending we also constructed episode level spending of different RBCS subcategories (CMS, 2024).

Table B1: Spending Measures Comparison with Prior Literature

	Keating et al. (2021)	Main Sample
Total	30,946	39,456
Part A	5,966	7,664
Part B	18,503	27,183
Part D	7,794	4,609

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 in 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 year 2014 corresponding to first year of observation 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 B1, we compare average episode-level spending in our sample for the year 2014 to OCM benchmarks presented in Table 2 of Keating et al. (2021). We observe slightly higher spending in Medicare Part B and Part A and lower average spending for Part D services, 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 and focus on a more narrow set of cancers.

Next, we provide an overview of episode level spending for all episodes in our main sample. In Figure B1 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. Part A spending remains almost unchanged over the entire time period.

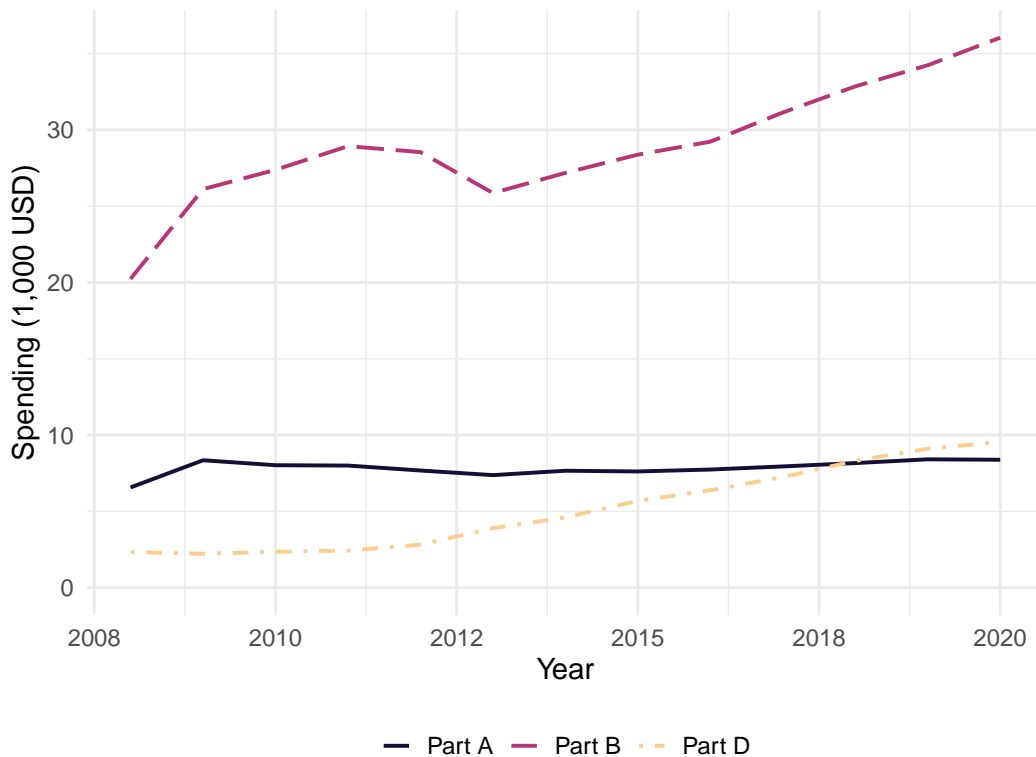


Figure B1: Spending per Episode by Medicare Part and Year

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

Figure B2 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 8% of total episode-level spending, compared to

69% for Part B and 23% for Part A. By 2020, the share of Part D spending had increased substantially to 18%, while the share of Part B spending declined to 67%, and Part A spending fell to 16%. This shift reflects the growing importance of oral and self-administered drugs in cancer treatment and the evolving structure of Medicare-financed oncology care.

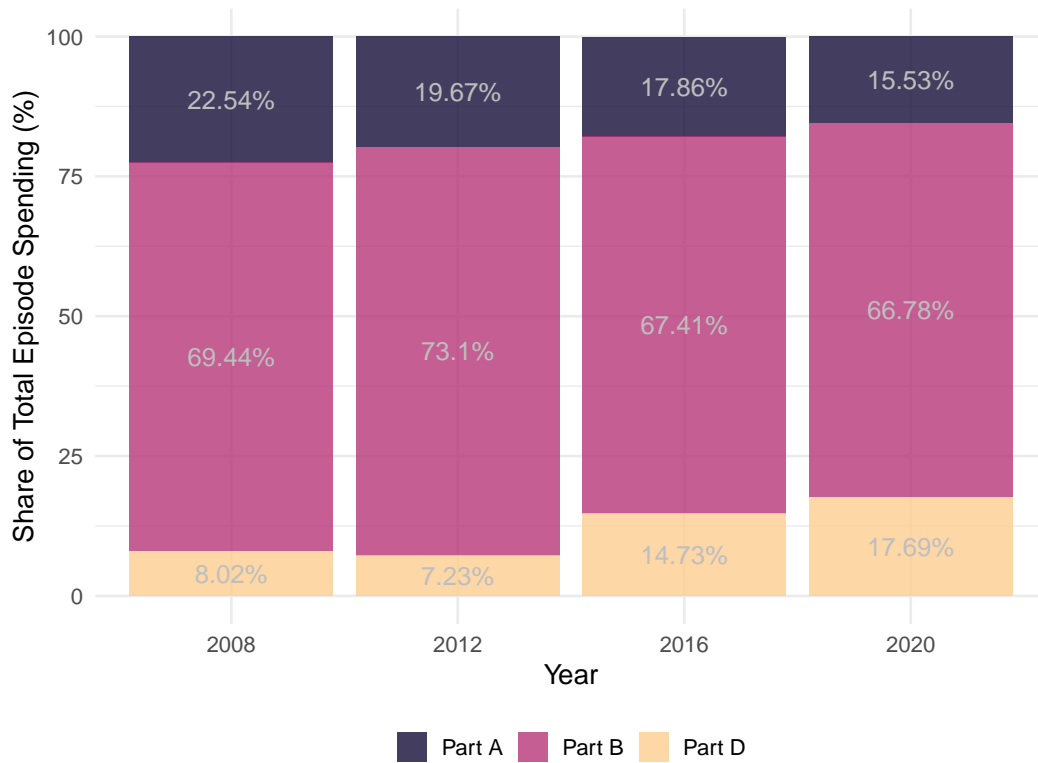


Figure B2: Share of Total Spending by Medicare Part over Time

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 first chemotherapy episodes from our main sample.

Table B2: 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_PTBL_DDCTBL_AMT	Bene.	
Line Bene. Coinsurance Amt.	LINE_COINSRNC_AMT	Bene.	
Line Bene. Part B Deductible Amt.	LINE_BENE_PTBL_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_PTBL_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_DSPRPRNTNT_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_PTBL_DDCTBL_AMT	Bene.	
NCH Bene. Part B Coinsurance Amt.	NCH_BENE_PTBL_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.

C 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 E2.

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 C1. 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).

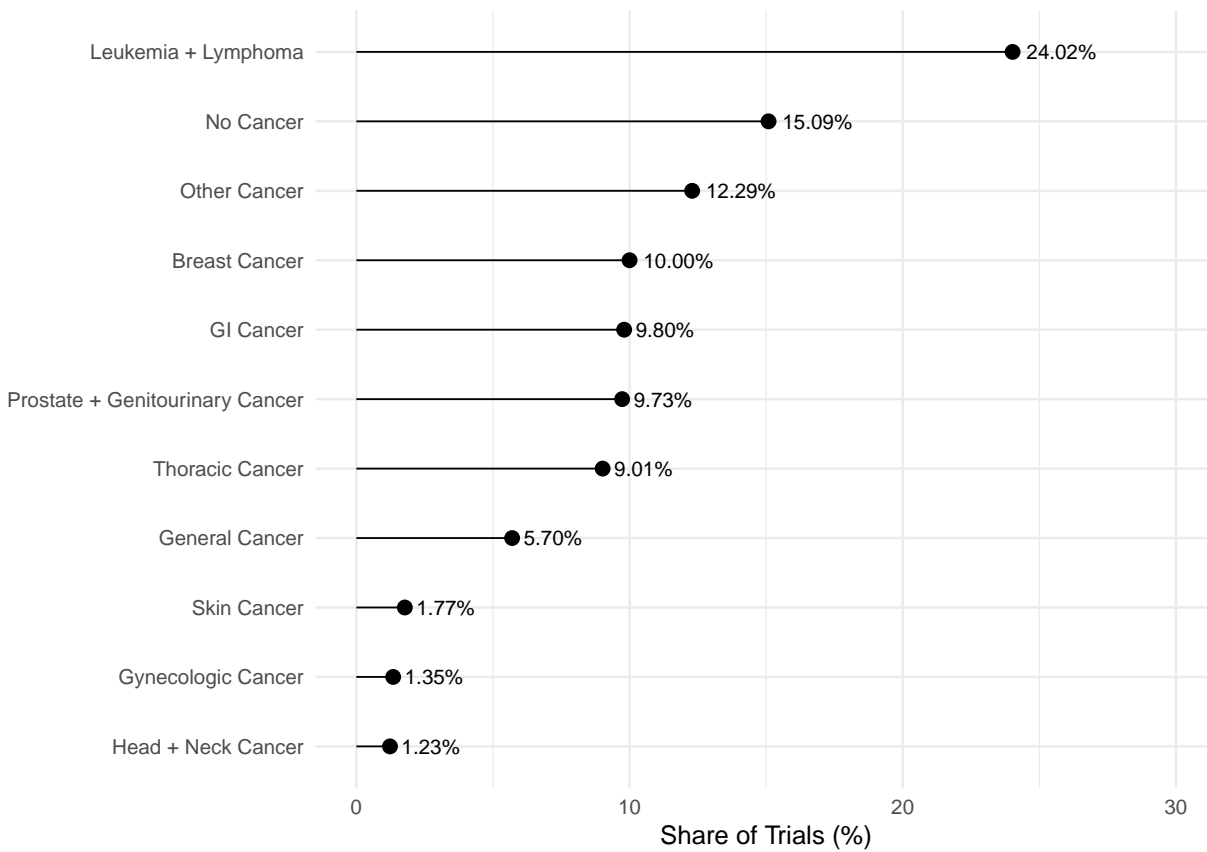


Figure C1: Share of Clinical Trials by Cancer Classification

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

D Additional Figures

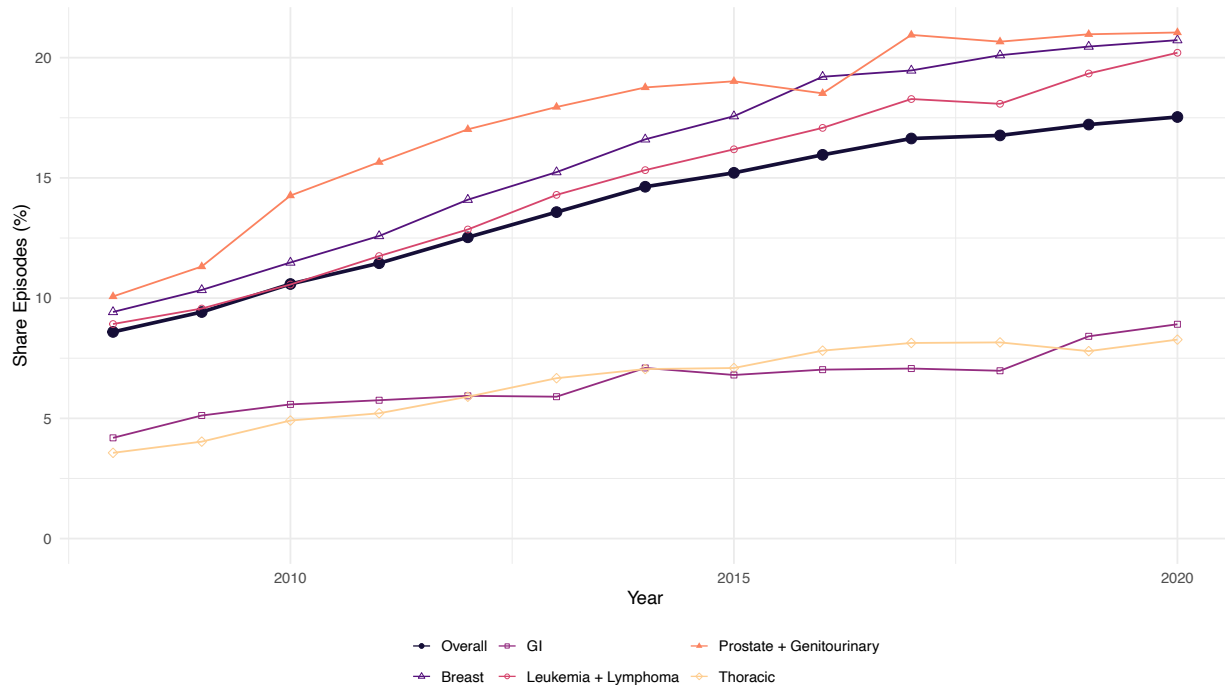


Figure D1: Trends in Subspecialization in Cancer Care 2008 - 2020

Note: This figure presents trends in subspecialization for beneficiary chemotherapy episodes of different cancer types based on results from Karadakic et al. (2025). The figure depicts the share of chemotherapy episodes managed by subspecialized oncologists of the relevant cancer type separately by cancer type. Subspecialized oncologists are defined as oncologists who manage more than 80% of chemotherapy episodes within a related group of cancers (e.g. gastrointestinal cancers). The “Overall” group (black line with black dots) indicates the trend in subspecialization for all cancer types combined. Abbreviations: GI=gastrointestinal.

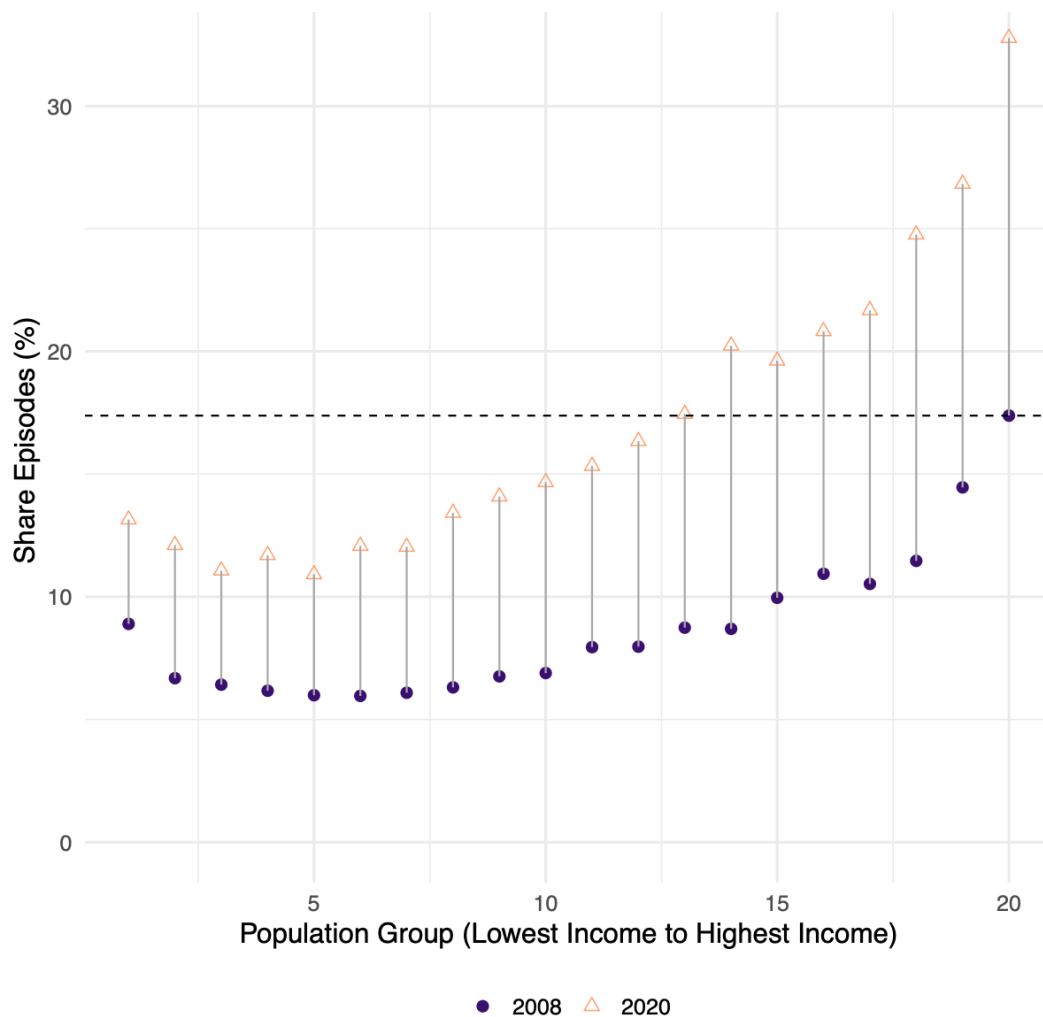


Figure D2: Socioeconomic Gradients in Access to Highly Subspecialized Cancer Care

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).

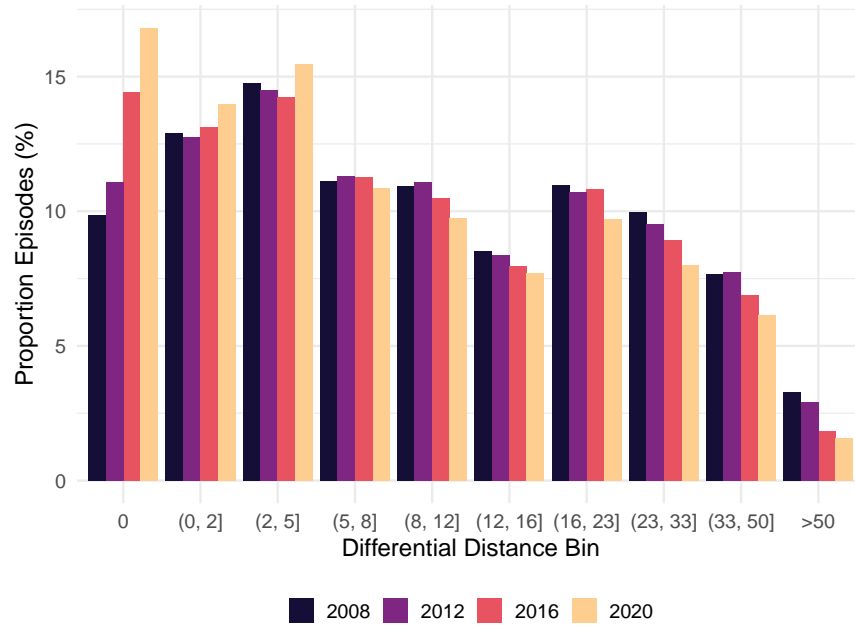


Figure D3: Distribution of Differential Distances over Time

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.

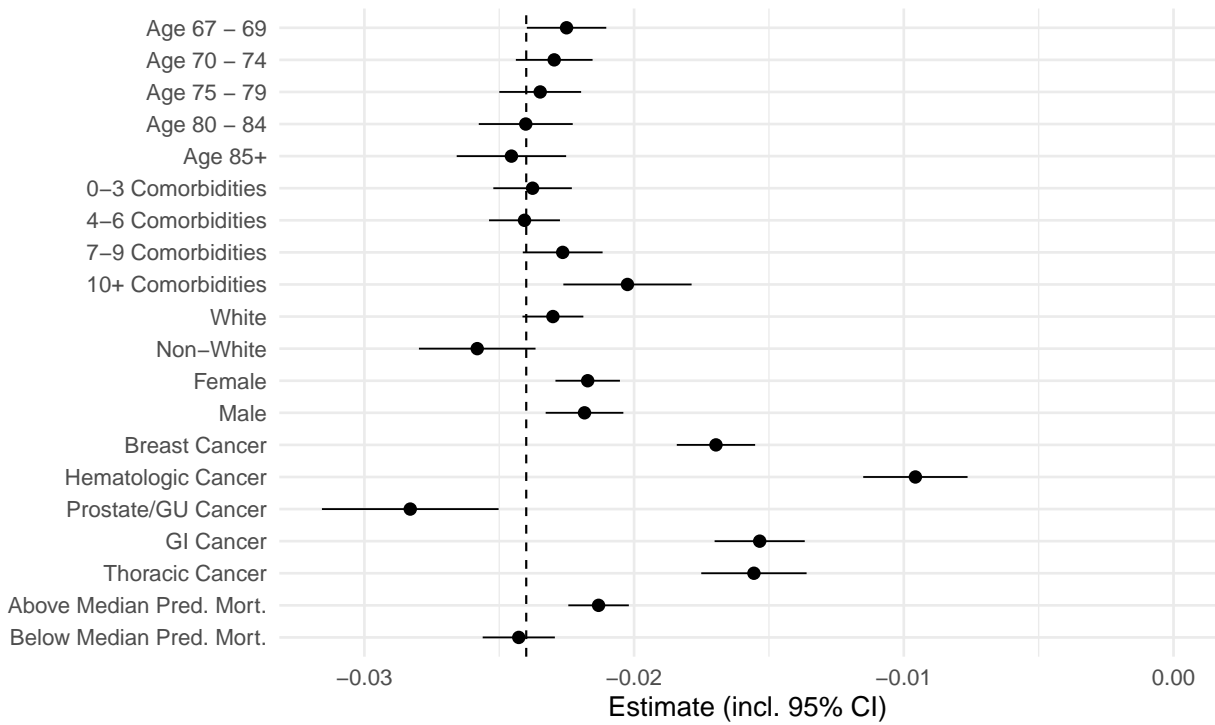


Figure D4: Instrument Monotonicity

Note: This figure presents first-stage estimates across different subsamples to assess the plausibility of the monotonicity assumption. Each point corresponds to the coefficient from a separate estimation of the first-stage equation for the subsample indicated on the y-axis. Error bars represent 95% confidence intervals based on standard errors clustered at the ZCTA level. The vertical dashed line represents the point estimate of the first stage relationship for the full sample and serves as a reference point.

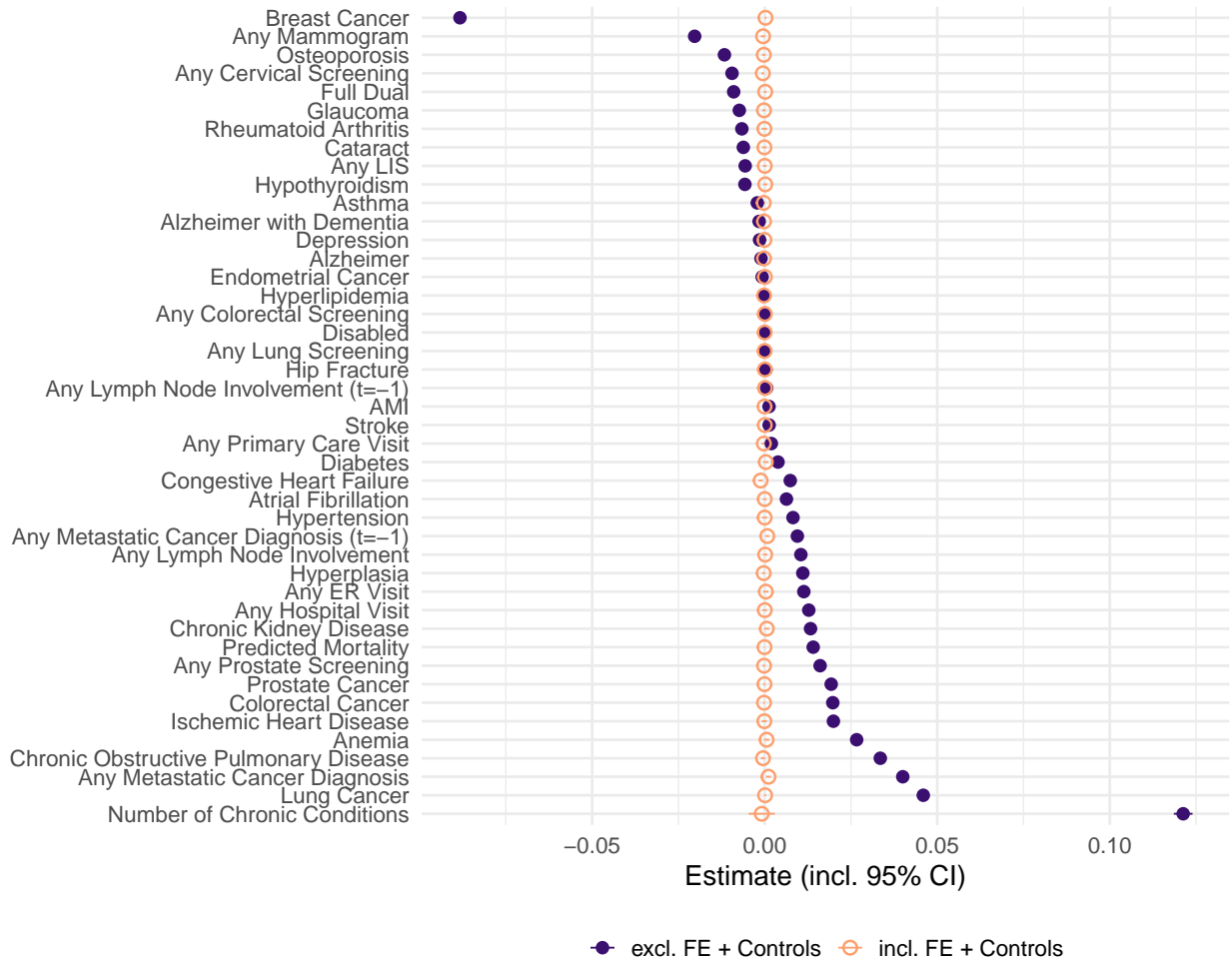
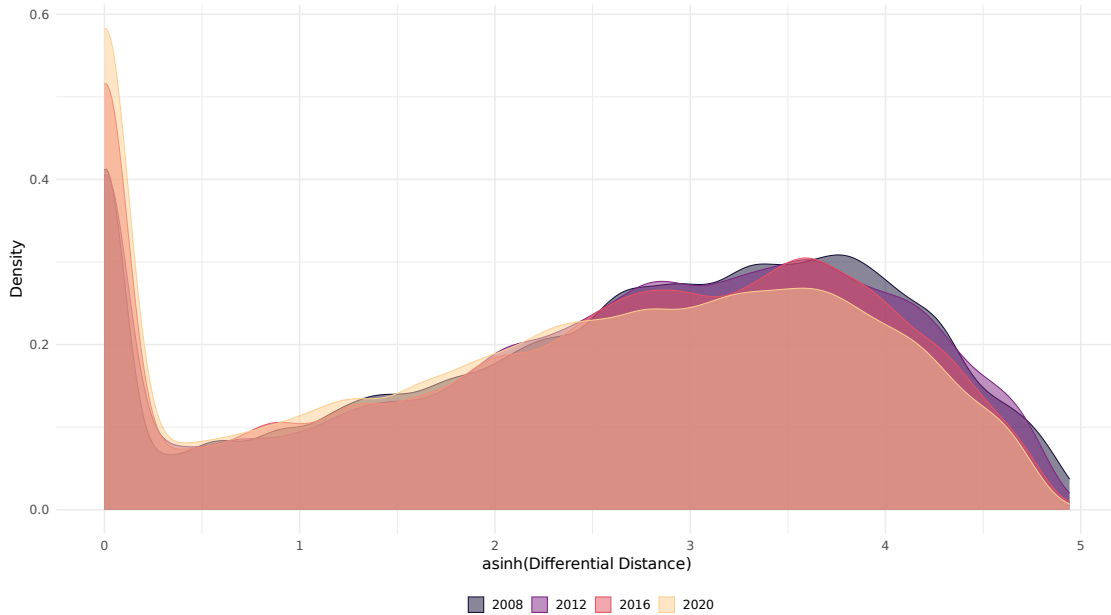
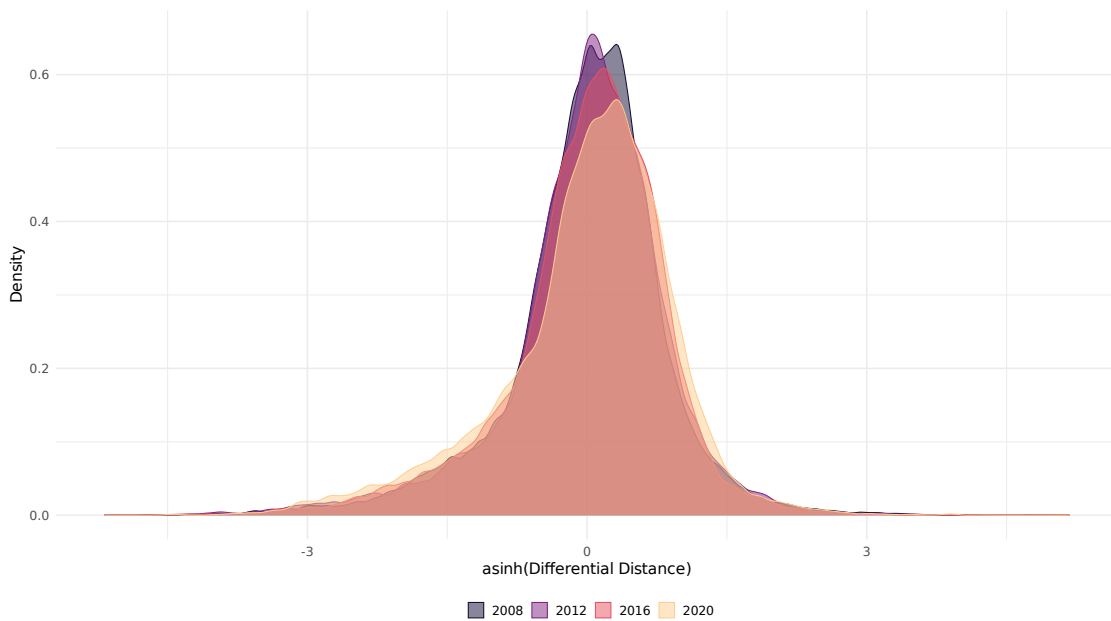


Figure D5: Instrument Balance across Beneficiary Characteristics

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) $\text{asinh}(\text{Differential Distance})$



(b) Residualized $\text{asinh}(\text{Differential Distance})$

Figure D6: Distribution of Instrumental Variable

Note: This figure displays the distribution of the instrumental variable—the inverse hyperbolic sine (IHS) of differential distance—for the main sample of first chemotherapy episodes. 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.

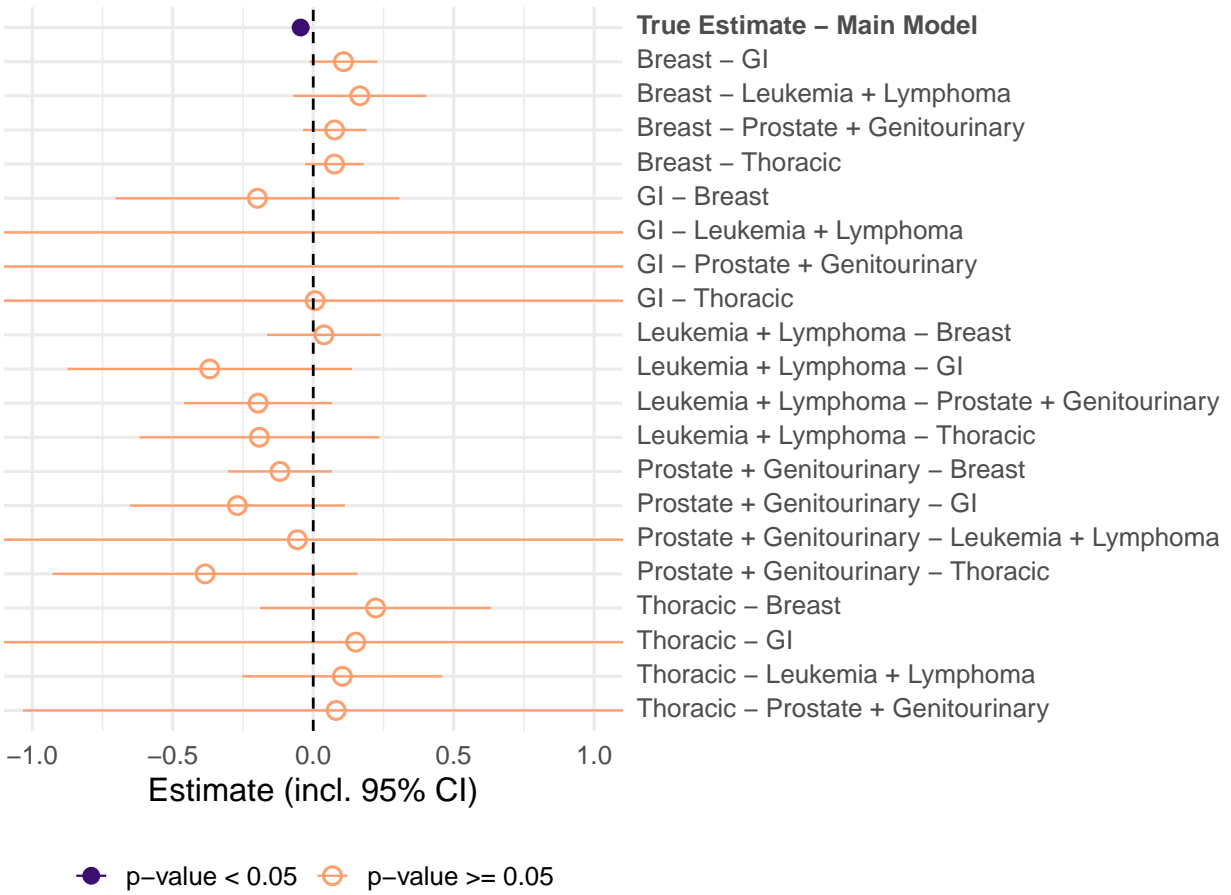


Figure D7: Falsification Test - Assignment of Distance to Unrelated Subspecialist

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. 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 first 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.

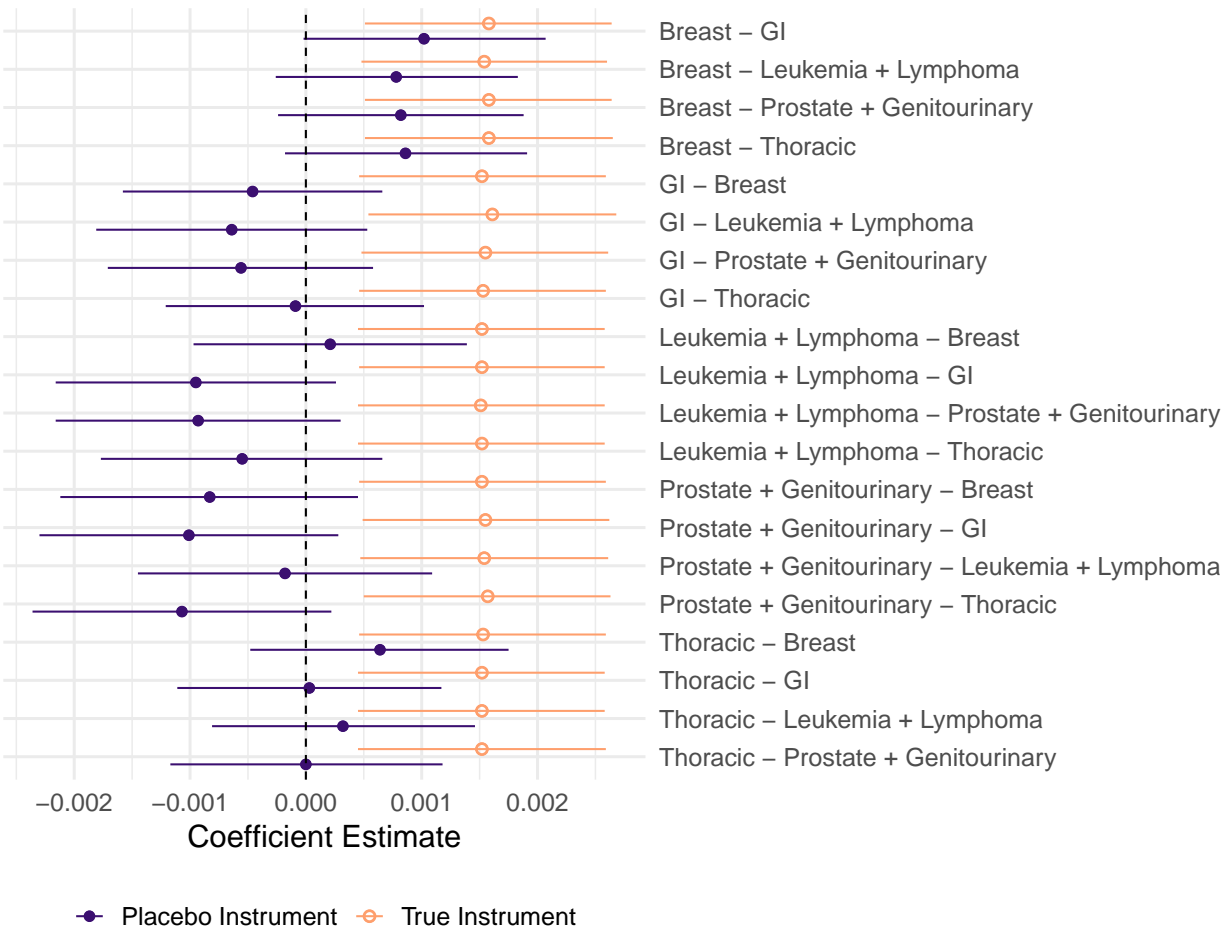
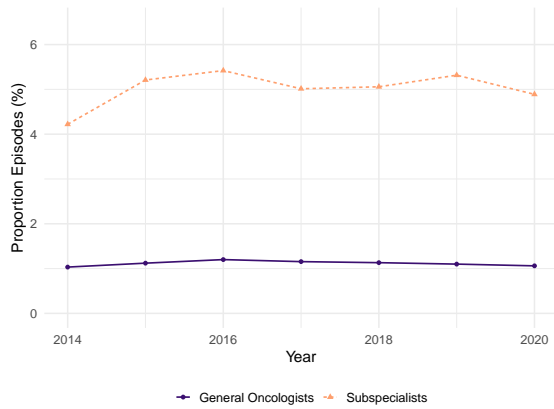
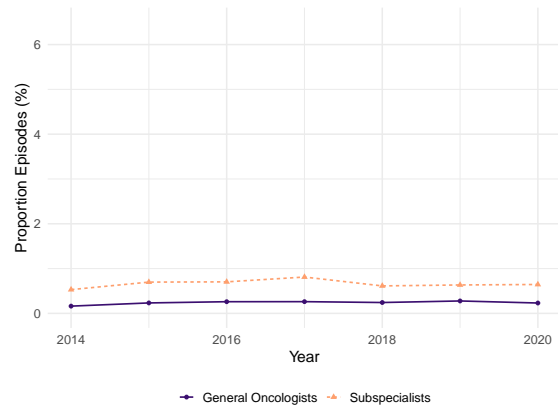


Figure D8: Negative Control Instrument Test

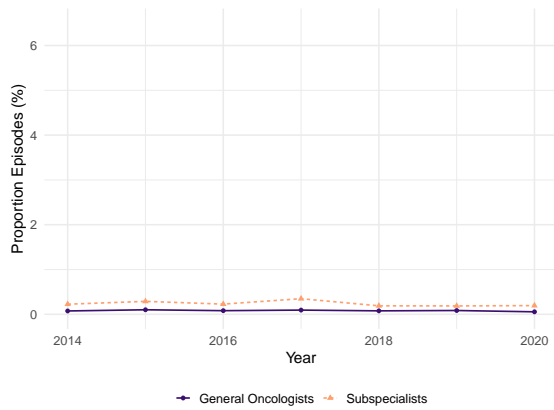
Note: This figure reports coefficient estimates and 95% confidence intervals from regressions of 1,080-day mortality on the true instrument and placebo instruments constructed using differential distance to subspecialists of unrelated cancer types, following the negative control instrument (NCI) approach of [Danieli et al. \(2026\)](#). All specifications condition on the true instrument and include the same controls and fixed effects as in the main analysis. Each pair of estimates corresponds to one placebo specification. Each row represents a specific reassignment of distances across cancer types. For example, “Breast – GI” indicates that beneficiaries are assigned the distance to the nearest breast cancer subspecialist, except for breast cancer patients, who are instead assigned the distance to the nearest GI subspecialist. The analysis sample consists of first chemotherapy episodes from 2008 to 2017. All models control for demographic characteristics, ZCTA-level covariates, and chronic conditions, and include ZCTA fixed effects and cancer type-by-year fixed effects. Standard errors are clustered at the ZCTA level.



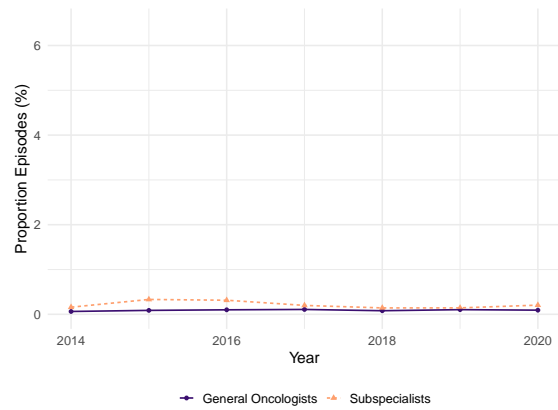
(a) Concordant Cancer Trial



(b) Discordant Cancer Trial



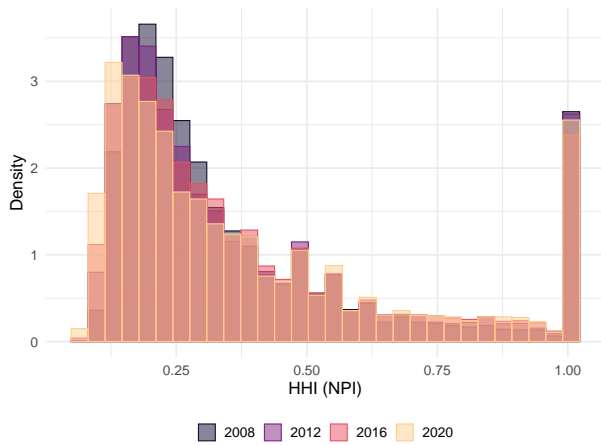
(c) Unspecified Cancer Trial



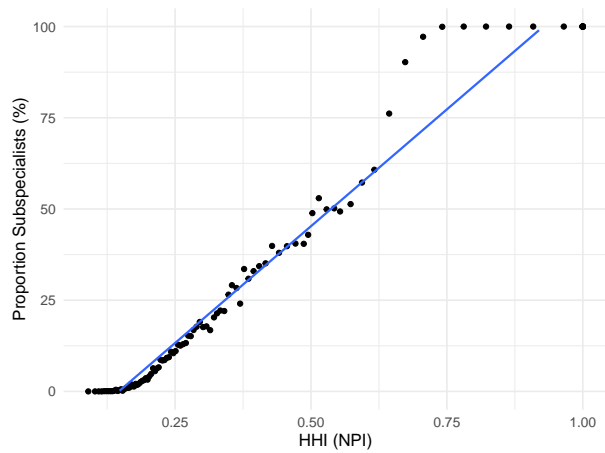
(d) Non-Cancer Trial

Figure D9: Proportion of Episodes with Cancer Trial Enrollment by Trial Type and Oncologist Subspecialization

Note: The figure shows the share of chemotherapy episodes from 2014 to 2020 in which the beneficiary had any claim containing an NCT number in the same year as chemotherapy initiation. The solid orange line represents episodes coordinated by subspecialized oncologists, while the dashed purple line represents episodes managed by general oncologists. Panel A reports the share of claims linked to cancer trials concordant with the beneficiary's cancer type; Panel B shows discordant cancer trials; Panel C depicts trials for unspecified cancers; and Panel D presents the share associated with non-cancer trials.



(a) Distribution of HHI



(b) HHI and Subspecialist Share

Figure D10: Descriptive Measures of Herfindahl–Hirschman Index

Note: This figure presents descriptive statistics of the cancer-type Herfindahl-Hirschman Index (HHI), a measure of oncologist specialization within a specific cancer type. Panel A plots the distribution of HHI values for all providers in our first chemotherapy sample for the years 2008, 2012, 2016, and 2020. Panel B displays the relationship between HHI and the share of subspecialized oncologists by plotting the average HHI and corresponding average subspecialist share across percentiles of the HHI distribution, constructed separately for each year.

E Additional Tables

Table E1: 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

(Table E1 continued)

TRIPTORELIN	J3316
ANTI-THYMOCYTE GLOBULIN, EQUINE	J7504
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

(Table E1 continued)

CYCLOPHOSPHAMIDE	J9096
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 E1 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
TRASTUZUMAB-DTTB	Q5112

(Table E1 continued)

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 E3: Sample Construction

Sample Restriction	Nr. Chemotherapy Episodes
Initial number of Episodes	9,250,813
Subset to focus cancer types	8,287,162
Remove episodes with missing ZCTA or HRR	8,267,999
Remove negative differential distances	8,149,437
Remove outlier distances above 95th distance percentile	7,744,706
Restrict to beneficiaries aged 67 and older	6,144,379
First episodes only	2,165,024
Final analysis sample	2,165,024

Notes: This table reports the construction of the analysis sample of chemotherapy episodes from Medicare claims data between 2008 and 2020. We sequentially apply the sample restrictions listed in the first column. The second column reports the number of chemotherapy episodes remaining after each restriction. ZCTA refers to ZIP Code Tabulation Area and HRR refers to Hospital Referral Region. The final analysis sample consists of first chemotherapy episodes for beneficiaries aged 67 and older with valid geographic information and non-outlier distances to the nearest generalist and subspecialized oncologists.

Table E4: Summary Statistics of Main Sample

Variable	Mean	SD	Min.	Max.
Panel A: Cancer Characteristics				
Breast Cancer	0.355	0.479	0	1
GI Cancer	0.158	0.365	0	1
Hematologic Cancer	0.222	0.416	0	1
Prostate + Genitourinary Cancer	0.127	0.333	0	1
Thoracic Cancer	0.163	0.369	0	1
Panel B: Demographics				
Bene Age	75.175	6.626	67	114
Female Bene	0.585	0.493	0	1
Black Bene	0.080	0.271	0	1
Hispanic Bene	0.011	0.104	0	1
Asian Bene	0.014	0.119	0	1
Other Non-White Race Bene	0.015	0.121	0	1
Panel C: Chronic Conditions				
Acute Myocardial Infarction	0.014	0.118	0	1
Alzheimer's Disease	0.023	0.150	0	1
Alzheimer's or Dementia	0.082	0.274	0	1
Anemia	0.559	0.496	0	1
Asthma	0.069	0.253	0	1
Atrial Fibrillation	0.129	0.335	0	1
Cataracts	0.206	0.405	0	1
Chronic Kidney Disease	0.314	0.464	0	1
COPD	0.231	0.422	0	1
Colorectal Cancer	0.102	0.303	0	1
Congestive Heart Failure	0.209	0.407	0	1
Depression	0.196	0.397	0	1
Diabetes	0.315	0.465	0	1
Endometrial Cancer	0.008	0.087	0	1
Glaucoma	0.104	0.305	0	1
Hyperlipidemia	0.570	0.495	0	1
Hyperplasia	0.112	0.315	0	1
Hypertension	0.726	0.446	0	1
Hypothyroidism	0.188	0.391	0	1
Ischemic Heart Disease	0.395	0.489	0	1
Lung Cancer	0.188	0.391	0	1
Osteoporosis	0.107	0.309	0	1
Prostate Cancer	0.135	0.342	0	1
Rheum. Arthritis / Osteoarthritis	0.372	0.483	0	1
Stroke (TIA)	0.047	0.213	0	1
Panel D: ZCTA Characteristics				
Distance to Closest Oncologist	3.703	3.756	0	17.287
Distance to Closest PCP	0.492	1.307	0	16.761
Mean Age	71.643	2.160	46.987	91.902
Median Household Income	64443.452	27127.046	2499	250001
Nr. of Mental Health Providers	9.985	20.118	0	338
Nr. of PCPs	31.271	61.304	0	2147
Share Asian	0.019	0.044	0	1
Share Black	0.098	0.168	0	1
Share Disabled	0.146	0.071	0	0.885
Share FFS	0.713	0.103	0.024	1
Share Full Dual	0.103	0.080	0	0.943
Share Hispanic	0.018	0.043	0	0.680
Share Male	0.451	0.029	0	1
Share Other Race	0.019	0.029	0	1
Total Beneficiaries	4734.646	3383.978	1	43028
Total Providers	103.964	189.181	0	7211

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

Table E5: Explained Instrument Variation

Fixed Effects	Adjusted R^2
ZCTA	0.527
Cancer type	0.102
Year	0.009
ZCTA + Year	0.535
ZCTA + Cancer type	0.625
Cancer type \times Year	0.110
ZCTA + Cancer type \times Year (design controls)	0.634
ZCTA-specific linear trends + Cancer type \times Year	0.667
ZCTA \times Cancer type + Cancer type \times Year	0.724
Design controls + other controls	0.642

Notes: Entries report the Adjusted R^2 from regressions of the differential distance instrument on alternative sets of fixed effects. Higher Adjusted R^2 values indicate that the corresponding set of fixed effects explains a larger share of the variation in the instrument.

Table E6: Drivers of Variation in Instrumental Variable

	Baseline	Fixed Stock	Fixed ZCTA	Fixed Specialization
Correlation with Baseline Z	1.000	0.737	0.711	0.645
First Stage β	-0.024***	-0.023***	-0.022***	-0.010***
First Stage F	1,823	3,036	2,613	684
Simple Adj. R^2	—	0.528	0.501	0.416
Full Adj. R^2	—	0.746	0.730	0.682
Within Adj. R^2	—	0.307	0.263	0.133

Notes: The table reports how much of the variation in the baseline differential–distance instrument (Z) is reproduced when holding different components of the oncology workforce fixed. Column 1 (Baseline) shows results for the original instrument. Column 2 (Fixed Stock) holds the set of oncologists constant at the 2008 workforce. Column 3 (Fixed ZCTA) holds each oncologist’s practice ZIP code (ZCTA) fixed at the first year they appear in our chemotherapy episode data. Column 4 (Fixed Specialization) holds only the oncologist’s subspecialization status fixed at first appearance. The correlation column reports pairwise correlations with the baseline instrument. The first-stage coefficient (β) and Kleibergen–Paap F-statistic are taken from regressions of access to a subspecialist on each instrument using the main specification. The “Simple Adj. R^2 ” is the adjusted R^2 from a univariate regression of the baseline instrument on each alternative instrument. The “Full Adj. R^2 ” is from the full first-stage model including demographic controls, chronic conditions, ZCTA controls, ZCTA fixed effects, and cancer-type-by-year fixed effects. The “Within Adj. R^2 ” reports the adjusted R^2 after absorbing fixed effects. Significance levels: *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$.

Table E7: First Stage Estimates

	Any Office Visit Subspecialist	Treatment Subspecialist
$\sinh^{-1}(\text{DD})$	-0.024*** (0.001)	-0.018*** (0.001)
ZCTA FE	Yes	Yes
Cancer-Year FE	Yes	Yes
Adj. R ²	0.174	0.161
Observations	2,165,024	2,165,024
Mean Dep. Var.	0.186	0.141
F-Stat (1st stage)	1,823	1,176

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 for our main sample of first chemotherapy episodes. 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. Reported first-stage F-statistics are Kleibergen-Paap statistics. Signif. Codes: ***: 0.01, **: 0.05, *: 0.1.

Table E8: Balancing Test of Instrumental Variable

Variable	Mean	SD	Est. Unadj.	Std. Err. Unadj.	Est. Adj.	Std. Err. Adj.
Panel A: Chronic Conditions Indicators						
Alzheimer	0.023	0.150	-0.001	0	0	0
Alzheimer with Dementia	0.082	0.274	-0.002	0	0	0
AMI	0.014	0.118	0.001	0	0	0
Anemia	0.559	0.496	0.027	0	0.001	0
Asthma	0.069	0.253	-0.002	0	0	0
Atrial Fibrillation	0.129	0.335	0.006	0	0	0
Breast Cancer	0.355	0.479	-0.088	0	0	0
Colorectal Cancer	0.102	0.303	0.020	0	0	0
Endometrial Cancer	0.008	0.087	-0.001	0	0	0
Lung Cancer	0.188	0.391	0.046	0	0	0
Prostate Cancer	0.135	0.342	0.019	0	0	0
Cataract	0.206	0.405	-0.006	0	0	0
Congestive Heart Failure	0.209	0.407	0.007	0	-0.001	0
Chronic Kidney Disease	0.314	0.464	0.013	0	0.001	0
Chronic Obstructive Pulmonary Disease	0.231	0.422	0.034	0	0	0
Depression	0.196	0.397	-0.001	0	0	0
Diabetes	0.315	0.465	0.004	0	0	0
Glaucoma	0.104	0.305	-0.007	0	0	0
Hip Fracture	0.011	0.107	0	0	0	0
Hyperlipidemia	0.570	0.495	0	0	0	0
Hyperplasia	0.112	0.315	0.011	0	0	0
Hypertension	0.726	0.446	0.008	0	0	0
Hypothyroidism	0.188	0.391	-0.006	0	0	0
Ischemic Heart Disease	0.395	0.489	0.020	0	0	0
Osteoporosis	0.107	0.309	-0.012	0	0	0
Rheumatoid Arthritis	0.372	0.483	-0.007	0	0	0
Stroke	0.047	0.213	0.001	0	0	0
Panel B: Prior Healthcare Use and Diagnosis						
Any Cervical Screening	0.061	0.239	-0.009	0	-0.001	0
Any Colorectal Screening	0.021	0.142	0	0	0	0
Any ER Visit	0.315	0.464	0.011	0	0	0
Any Hospital Visit	0.235	0.424	0.013	0	0	0
Any Lung Screening	0.003	0.055	0	0	0	0
Any Lymph Node Involvement (t=-1)	0.056	0.229	0.001	0	0	0
Any Lymph Node Involvement	0.187	0.390	0.010	0	0	0
Any Mammogram	0.182	0.386	-0.020	0	0	0
Any Metastatic Cancer Diagnosis (t=-1)	0.118	0.323	0.009	0	0.001	0
Any Metastatic Cancer Diagnosis	0.356	0.479	0.040	0	0.001	0
Any Primary Care Visit	0.810	0.393	0.002	0	0	0
Any Prostate Screening	0.078	0.268	0.016	0	0	0
Panel C: Other Measures						
Any LIS	0.140	0.347	-0.006	0	0	0
Disabled	0.000	0.002	0	0	0	0
Full Dual	0.073	0.260	-0.009	0	0	0
Number of Chronic Conditions	5.095	2.930	0.121	0.001	-0.001	0.002
Predicted Mortality	0.098	0.119	0.014	0	0	0

Notes: The table provides summary statistics for different beneficiary characteristics and health-care 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 E9: Mortality Effects of Access to Subspecialized Oncologist

	180-Day	360-Day	720-Day	1080-Day
Panel A: First Stage				
$\sinh^{-1}(\text{DD})$	-0.024*** (0.001)	-0.024*** (0.001)	-0.024*** (0.001)	-0.024*** (0.001)
Panel B: Reduced Form				
$\sinh^{-1}(\text{DD})$	0.000 (0.000)	0.001*** (0.000)	0.001*** (0.000)	0.001*** (0.000)
Panel C: Structural Form				
Any Office Visit Subs.	-0.014*** (0.001)	-0.005*** (0.001)	0.003*** (0.001)	0.005*** (0.001)
Panel D: 2SLS				
Any Office Visit Subs.	-0.007 (0.011)	-0.040*** (0.014)	-0.050*** (0.016)	-0.045** (0.016)
Adj R ²	0.120	0.224	0.302	0.325
Observations	1,681,119	1,681,119	1,681,119	1,681,119
Mean Dep. Var.	0.108	0.226	0.365	0.449
F-Stat (1st Stage)	1,649	1,649	1,649	1,649

Notes: This table reports estimates of the effect of access to subspecialized oncologists on mortality for all first chemotherapy episodes between 2008 and 2017. Panel A shows first stage estimates. Panel B shows the reduced form estimates. Panel C shows the structural form estimates, and Panel D provides the 2SLS estimates. All models control for demographics, ZCTA characteristics, comorbidities, and include ZCTA and cancer type-by-year fixed effects. Standard errors are clustered at the ZCTA level. First-stage strength is reported using the Kleibergen-Paap F-statistic. Significance levels: *** p<0.01, ** p<0.05, * p<0.1.

Table E10: Robustness of Mortality Outcomes by Samples and Specifications

	180-Day	360-Day	720-Day	1080-Day
Panel A: Fixed Sample (2008-2017)				
Any Office Visit Subs.	-0.007 (0.011)	-0.040*** (0.014)	-0.050*** (0.016)	-0.045** (0.016)
Adj. R ²	0.134	0.224	0.302	0.335
Observations	1,681,119	1,681,119	1,681,119	1,681,119
Mean Dep. Var.	0.108	0.226	0.365	0.449
F-Stat (1st Stage)	1,649	1,649	1,649	1,649
Panel B: Varying Sample				
Any Office Visit Subs.	-0.001 (0.010)	-0.024* (0.031)	-0.041*** (0.015)	-0.045*** (0.016)
Adj. R ²	0.119	0.223	0.302	0.325
Observations	2,014,625	2,014,625	1,845,326	1,681,119
Mean Dep. Var.	0.105	0.220	0.361	0.449
F-Stat (1st Stage)	1,767	1,767	1,731	1,649
Panel C: Volume Controls				
Any Office Visit Subs.	-0.005 (0.014)	-0.046** (0.018)	-0.058*** (0.020)	-0.050*** (0.020)
Adj. R ²	0.119	0.225	0.302	0.325
Observations	1,681,119	1,681,119	1,681,119	1,681,119
Mean Dep. Var.	0.108	0.226	0.365	0.449
F-Stat (1st Stage)	1,540	1,540	1,540	1,540
Panel D: ZCTA Slopes				
Any Office Visit Subs.	0.003 (0.012)	-0.032** (0.015)	-0.048*** (0.016)	-0.039*** (0.016)
Adj. R ²	0.120	0.224	0.302	0.325
Observations	1,681,119	1,681,119	1,681,119	1,681,119
Mean Dep. Var.	0.108	0.226	0.365	0.449
F-Stat (1st Stage)	1,605	1,605	1,605	1,605
Panel E: Alternative Treatment				
Attributed Subspecialist	-0.010 (0.015)	-0.054*** (0.020)	-0.068*** (0.022)	-0.061** (0.022)
Adj R ²	0.120	0.224	0.302	0.325
Observations	1,681,119	1,681,119	1,681,119	1,681,119
Mean Dep. Var.	0.108	0.226	0.365	0.449
F-Stat (1st Stage)	982	982	982	982

Notes: This table reports 2SLS estimates of the effect of access to subspecialized oncologists on mortality. Panel A presents the baseline results for first chemotherapy episodes from 2008–2017. Panel B allows the estimation sample to vary with the mortality horizon based on available follow-up. Panel C adds controls for the treating oncologist’s episode volume across cancer groups. Panel D includes ZCTA-specific linear time trends. Panel E uses an alternative treatment definition equal to one if a patient’s chemotherapy episode is attributed to a subspecialist of the relevant cancer type. All models control for demographics, ZCTA characteristics, comorbidities, and include ZCTA and cancer type-by-year fixed effects. Standard errors are clustered at the ZCTA level. First-stage strength is reported using the Kleibergen-Paap F-statistic. Significance levels: *** p<0.01, ** p<0.05, * p<0.1.

Table E2: ICD 9 and ICD 10 Codes for Cancer Type Classification

Cancer Type Label	Cancer Types Included	ICD-9/ICD-10 Codes
Breast	Breast Cancer; Carcinoma in situ of breast	174.xx, 175.xx, 233.0x, C50.xx, D05.xx
GI	Anal Cancer; Carcinoma in situ of oral cavity, esophagus and stomach; Carcinoma in situ of other and unspecified digestive organs; Gastro/Esophageal Cancer; Liver Cancer; Malignant neoplasm of abdomen; Malignant neoplasm of other and ill-defined digestive organs; Pancreatic Cancer; Small Intestine / Colorectal Cancer	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	Carcinoma in situ of cervix uteri; Female GU Cancer other than Ovary; Malignant neoplasm of other and unspecified female genital organs; Malignant neoplasm of placenta; Ovarian Cancer	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	Carcinoma in situ of middle ear and respiratory system; Head and Neck Cancer	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; Acute panmyelosis with myelofibrosis; Atypical chronic myeloid leukemia, BCR/ABL negative; Chronic Leukemia; Chronic leukemia of unspecified cell type; Chronic myelomonocytic leukemia; Chronic myeloproliferative disease; Essential (hemorrhagic) thrombocythemia; Juvenile myelomonocytic leukemia; Leukemia, unspecified; Lymphoid Leukemia, unspecified; Lymphoma; MDS; Monocytic Leukemia, unspecified; Multiple Myeloma; Myelofibrosis; Myeloid leukemia, unspecified; Osteomyelofibrosis; Other and unspecified malignant neoplasms of lymphoid, hematopoietic and related tissue; Other lymphoid leukemia; Other monocytic leukemia; Other myeloid leukemia; Other specified leukemias; Polycythemia vera; Secondary and unspecified malignant neoplasm of lymph nodes	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	Bladder Cancer; Carcinoma in situ of other and unspecified genital organs; Kidney Cancer; Malignant neoplasm of penis, other, and unspecified male organs; Malignant neoplasm of testis; Prostate Cancer	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	Carcinoma in situ of skin; Malignant Melanoma; Melanoma in situ; Merkel cell carcinoma; Other and unspecified malignant neoplasm of skin	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 Cancer; Malignant neoplasm of heart, mediastinum and pleura; Malignant neoplasm of thorax; Malignant neoplasm of 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	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 used for subspecialist classification, column 2 provides the more detailed cancer types which are used for statistical modeling purposes (e.g. inclusion of cancer type fixed effects) and column 3 the corresponding ICD-9 and ICD-10 codes used for identification of cancers in the Medicare claims.

Table E11: Access to Subspecialized Oncologist and Spending by Subcategory

	Part A				Part B			Part D
	Inpatient	HHA	Hospice	SNF	Carrier	Outpatient	DME	
Panel A: First Stage								
$\sinh^{-1}(\text{DD})$	-0.024*** (0.001)	-0.024*** (0.001)	-0.024*** (0.001)	-0.024*** (0.001)	-0.024*** (0.001)	-0.024*** (0.001)	-0.024*** (0.001)	-0.024*** (0.001)
Panel B: Reduced Form								
$\sinh^{-1}(\text{DD})$	-16.66 (10.52)	-0.93 (1.50)	6.44* (3.68)	-8.57*** (2.87)	123.42*** (20.57)	-105.64*** (21.19)	-0.80 (1.35)	11.21 (11.20)
Panel C: Structural Form								
Any Office Visit Subs.	1,932.95*** (33.258)	-29.99*** (3.62)	-69.27*** (5.58)	-191.17*** (6.86)	-4,207.06*** (57.33)	6,900.43*** (74.64)	33.21*** (3.53)	444.74*** (32.71)
Panel D: 2SLS								
Any Office Visit Subs.	704.42 (444.37)	39.30 (63.55)	-272.21* (155.94)	362.27*** (121.50)	-5,217.32*** (874.14)	4,465.74*** (899.49)	33.92 (57.14)	-474.06 (474.08)
Adj. R ²	0.146	0.136	0.024	0.082	0.197	0.114	0.068	0.242
Observations	2,165,024	2,165,024	2,165,024	2,165,024	2,165,024	2,165,024	2,165,024	2,165,024
Mean Dep. Var.	5,699.26	778.14	527.70	764.10	15,370.09	12,670.91	420.14	5,048.72
F-Stat (1st Stage)	1,823	1,823	1,823	1,823	1,823	1,823	1,823	1,823

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 3 Part B spending and column 4 Part D spending. Panel A presents first stage estimates, Panel B the respective reduced form estimates, Panel C the structural form estimates and Panel D provides 2SLS 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. First-stage strength is reported using the Kleibergen-Paap F-statistic. Standard errors are clustered at the ZCTA level. Signif. Codes: ***: 0.01, **: 0.05, *: 0.1.

Table E12: Access to Subspecialized Oncologist and Spending on Chemotherapy Drugs and Services

	Chemotherapy Drugs	Injections & Infusions (RI)
Panel A: First Stage		
$\sinh^{-1}(\text{DD})$	-0.024*** (0.001)	-0.024*** (0.001)
Panel B: Reduced Form		
$\sinh^{-1}(\text{DD})$	60.74*** (13.88)	15.51*** (5.835)
Panel C: Structural Form		
Any Office Visit Subs.	-1,023.72*** (37.01)	-458.82*** (14.24)
Panel D: 2SLS		
Any Office Visit Subs.	-2,567.73*** (587.17)	-655.78*** (246.70)
Adj R ²	0.167	0.104
Observations	2,165,024	2,165,024
Mean Dep. Var.	9,411.18	3,332.22
F-Stat (1st Stage)	1,822	1,822

Notes: This table reports estimates of the effect of access to subspecialized oncologists on different categories of spending for the full sample of first chemotherapy episodes. Column 1 focuses on Part B spending for chemotherapy-related HCPCS codes, defined according to the Oncology Care Model. Column 2 presents Part B spending estimates for HCPCS codes related to the infusion and injection of drugs, based on the 2024 Restructured BETOS Classification System (RBCS). Panel A shows first-stage results; Panel B reports reduced-form estimates; Panel C provides structural-form estimates; and Panel D presents the 2SLS results. All specifications control for beneficiary demographics, ZIP code-level characteristics, and comorbidities, and include fixed effects for ZIP code and cancer type-by-year. Standard errors are clustered at the ZIP code (ZCTA) level. First-stage strength is summarized using the Kleibergen-Paap F-statistic. Significance levels: *** p<0.01, ** p<0.05, * p<0.1.

Table E13: Access to Subspecialists and Placebo Outcomes

	Year + 1			Year + 2		
	AMI	Hip Fracture	Stroke	AMI	Hip Fracture	Stroke
Panel A: First Stage						
$\sinh^{-1}(\text{DD})$	-0.024*** (0.001)	-0.024*** (0.001)	-0.024*** (0.001)	-0.024*** (0.001)	-0.024*** (0.001)	-0.024*** (0.001)
Panel B: Reduced Form						
$\sinh^{-1}(\text{DD})$	0.000 (0.000)	0.000 (0.000)	0.000* (0.000)	0.000 (0.000)	0.000 (0.000)	0.000 (0.000)
Panel C: Structural Form						
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)
Panel D: 2SLS						
Any Office Visit Subs.	-0.001 (0.002)	-0.001 (0.002)	0.005* (0.003)	0.000 (0.002)	-0.001 (0.002)	0.001 (0.003)
Adj R ²	0.006	0.002	0.002	0.003	0.002	0.001
Observations	2,014,625	2,014,625	2,014,625	1,845,326	1,845,326	1,845,326
Mean Dep. Var.	0.005	0.005	0.008	0.003	0.004	0.006
F-Stat (1st Stage)	1,767	1,767	1,767	1,731	1,731	1,731

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. Health conditions were identified using diagnostic related group (DRG) group codes. Acute Myocardial Infarction: 280, 281, 282; hip Fracture: 480, 481, 482; stroke: 061, 062, 063, 064, 065, 066. Results are estimated on the sample of first chemotherapy episode. Panel A shows first stage estimates. Panel B shows the reduced form estimates. Panel C shows the structural form estimates, and Panel D provides the 2SLS estimates. All models control for demographics, ZCTA characteristics, comorbidities, and include ZCTA and cancer type-by-year fixed effects. Standard errors are clustered at the ZCTA level. First-stage strength is reported using the Kleibergen-Paap F-statistic. Significance levels: *** p<0.01, ** p<0.05, * p<0.1.

Table E14: Complier Characteristics Overview

Variable	Share	Share Among Compliers	Share Among Treated	N (Conditional)	N (Unconditional)
Panel A: Chemotherapy Episode Cancer Type					
Breast Cancer	0.330	0.241	0.347	713,977	2,166,050
GI Cancer	0.158	0.104	0.103	342,279	2,166,050
Hematologic Cancer	0.222	0.094	0.309	481,932	2,166,050
Prostate/Genito. Cancer	0.127	0.155	0.154	274,981	2,166,050
Thoracic Cancer	0.163	0.111	0.096	352,881	2,166,050
Panel B: Demographic Information					
Bene. Female	0.585	0.548	0.577	1,267,710	2,166,050
Bene. Black	0.080	0.090	0.081	172,722	2,166,050
Bene. Hispanic	0.011	0.008	0.011	23,713	2,166,050
Bene. Asian	0.014	0.011	0.019	31,227	2,166,050
Bene. Other/Non-White	0.015	0.013	0.020	32,070	2,166,050
Bene. Age 67–70	0.309	0.302	0.375	668,247	2,166,050
Bene. Age 70–74	0.274	0.269	0.299	593,753	2,166,050
Bene. Age 75–79	0.220	0.224	0.204	475,614	2,166,050
Bene. Age 80–84	0.151	0.156	0.118	327,810	2,166,050
Bene. Age 85+	0.105	0.105	0.071	228,195	2,166,050
Bene. Full Dual	0.073	0.072	0.063	157,284	2,166,050
Panel C: Chronic Conditions Indicators					
Alzheimer	0.023	0.021	0.016	49,912	2,166,050
Alz. with Dementia	0.082	0.076	0.065	177,334	2,166,050
AMI	0.014	0.013	0.012	30,411	2,166,050
Anemia	0.559	0.501	0.526	1,210,975	2,166,050
Asthma	0.069	0.069	0.071	149,164	2,166,050
Atrial Fibrillation	0.129	0.126	0.116	278,790	2,166,050
Colorectal Cancer	0.102	0.066	0.052	221,423	2,166,050
Endometrial Cancer	0.008	0.010	0.009	16,684	2,166,050
Lung Cancer	0.188	0.136	0.116	407,495	2,166,050
Prostate Cancer	0.135	0.153	0.156	292,052	2,166,050
Cataract	0.206	0.217	0.223	447,094	2,166,050
CHF	0.209	0.192	0.177	453,140	2,166,050
CKD	0.314	0.304	0.311	679,220	2,166,050
COPD	0.231	0.211	0.156	501,019	2,166,050
Depression	0.196	0.193	0.206	425,230	2,166,050
Diabetes	0.315	0.302	0.284	682,425	2,166,050
Hyperplasia	0.112	0.107	0.121	242,540	2,166,050
Glaucoma	0.104	0.119	0.121	225,445	2,166,050
Hip Fracture	0.011	0.008	0.010	24,866	2,166,050
Hyperlipidemia	0.570	0.573	0.569	1,233,660	2,166,050
Hypertension	0.726	0.718	0.689	1,572,380	2,166,050
Hypothyroidism	0.190	0.186	0.191	406,991	2,166,050
Ischemic Heart Disease	0.395	0.381	0.364	855,986	2,166,050
Osteoporosis	0.107	0.107	0.115	232,089	2,166,050
Rheumatoid Arthritis	0.372	0.372	0.380	806,324	2,166,050
Stroke	0.047	0.044	0.042	102,774	2,166,050
Panel D: Other Characteristics					
Chronic Cond. (0–3]	0.299	0.304	0.348	646,574	2,166,050
Chronic Cond. (3–4]	0.129	0.137	0.135	279,510	2,166,050
Chronic Cond. (4–6]	0.260	0.266	0.253	563,048	2,166,050
Chronic Cond. (6–8]	0.184	0.176	0.163	399,571	2,166,050
Chronic Cond. >8	0.128	0.122	0.108	277,347	2,166,050
Pred. Mortality Q1	0.200	0.205	0.260	433,210	2,166,050
Pred. Mortality Q2	0.200	0.193	0.243	433,210	2,166,050
Pred. Mortality Q3	0.200	0.198	0.211	433,210	2,166,050
Pred. Mortality Q4	0.200	0.192	0.173	433,210	2,166,050
Pred. Mortality Q5	0.200	0.161	0.118	433,210	2,166,050

Notes: The table presents characteristics of the differential distance compliers, in comparison to the overall sample of chemotherapy beneficiaries.

Table E15: Subspecialist Access and Use of Newly FDA Approved Drugs

	Any	Part B	Part D
Panel A: First Stage			
$\sinh^{-1}(\text{DD})$	-0.024*** (0.001)	-0.024*** (0.001)	-0.024*** (0.001)
Panel B: Reduced Form			
$\sinh^{-1}(\text{DD})$	0.000* (0.000)	0.000 (0.000)	0.000 (0.000)
Panel C: Structural Form			
Any Office Visit Subs.	0.000*** (0.000)	0.005*** (0.000)	0.005*** (0.000)
Panel D: 2SLS			
Any Office Visit Subs.	0.002* (0.001)	0.007 (0.005)	0.007* (0.524)
Adj R ²	0.016	0.114	0.064
Observations	2,165,024	2,165,024	2,165,024
Mean Dep. Var.	0.029	0.026	0.017
F-Stat (1st Stage)	1,823	1,823	1,823

Notes: The table provides estimates on the effect of access to subspecialized oncologists on the probability to utilize a cancer drug where the generic substance has received FDA approval within the last two years. Column 1 indicates the effect on the probability to receive any such drug, either through infusion (Part B) or orally (Part D). Column 2 provides estimates of the effect for Part B drugs only and column 3 provides estimates for Part D drugs. Panel A presents first stage estimates, Panel B the respective reduced form estimates, Panel C the structural form estimates and Panel D provides 2SLS estimates. All models include demographic, ZCTA level and chronic conditions controls as well fixed effects for the beneficiaries' ZCTA and cancer type by year. Standard errors are clustered at the ZCTA level. First-stage strength is reported using the Kleibergen-Paap F-statistic. Signif. Codes: ***: 0.01, **: 0.05, *: 0.1.

Table E16: Subspecialist Access and Average Age of Cancer Drugs

	All	Part B	Part D
Panel A: First Stage			
$\sinh^{-1}(\text{DD})$	-0.024*** (0.001)	-0.024*** (0.001)	-0.024*** (0.001)
Panel B: Reduced Form			
$\sinh^{-1}(\text{DD})$	0.012 (0.009)	0.000 (0.013)	0.018 (0.012)
Panel C: Structural Form			
Any Office Visit Subs.	-0.871*** (0.023)	-1.217*** (0.031)	-0.377*** (0.029)
Panel D: 2SLS			
Any Office Visit Subs.	-0.502 (0.386)	-0.011 (0.552)	-0.797 (0.524)
Adj R ²	0.240	0.239	0.353
Observations	2,159,340	1,726,189	1,052,397
Mean Dep. Var.	27.46	31.68	25.65
F-Stat (1st Stage)	1,827	1,690	1,218

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 presents first stage estimates, Panel B the respective reduced form estimates, Panel C the structural form estimates and Panel D provides 2SLS estimates. All models include demographic, ZCTA level and chronic conditions controls as well fixed effects for the beneficiaries' ZCTA and cancer type by year. Standard errors are clustered at the ZCTA level. First-stage strength is reported using the Kleibergen-Paap F-statistic. Signif. Codes: ***, 0.01, **, 0.05, *, 0.1.

Table E17: Access to Subspecialized Oncologist and Provider Mix

	Nr. Visits	Unique Providers	Unique Specialties
Panel A: First Stage			
$\sinh^{-1}(\text{DD})$	-0.024*** (0.001)	-0.024*** (0.001)	-0.024*** (0.001)
Panel B: Reduced Form			
$\sinh^{-1}(\text{DD})$	-0.002 (0.006)	0.005*** (0.002)	0.000 (0.002)
Panel C: Structural Form			
Any Office Visit Subs.	0.789*** (0.018)	0.666*** (0.006)	0.417*** (0.004)
Panel D: 2SLS			
Any Office Visit Subs.	0.094 (0.271)	-0.194*** (0.083)	-0.003 (0.064)
Adj R ²	0.221	0.175	0.169
Observations	2,165,024	2,165,024	2,165,024
Mean Dep. Var.	12.08	4.56	4.02
F-Stat (1st Stage)	1,822	1,822	1,822

Notes: The table provides estimates on the effect of access to subspecialized oncologists on different measures relevant for the beneficiary provider mix for our full sample of first chemotherapy episodes. 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. Panel A shows first stage estimates. Panel B shows the reduced form estimates. Panel C shows the structural form estimates, and Panel D provides the 2SLS estimates. All models control for demographics, ZCTA characteristics, comorbidities, and include ZCTA and cancer type-by-year fixed effects. Standard errors are clustered at the ZCTA level. First-stage strength is reported using the Kleibergen-Paap F-statistic. Significance levels: *** p<0.01, ** p<0.05, * p<0.1.