The Value of (Sub) Specialization: Evidence from Oncology*

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Abstract

We estimate the returns to specialization in medical oncology, in which subspecialists focus on specific cancer types. Using 2.2 million Medicare chemotherapy episodes from 2008–2020, we instrument for subspecialist access with differential distance to cancer-type-specific subspecialists versus general oncologists. Access to a relevant subspecialist reduces three-year mortality by 4.5 percentage points (10 percent), without increasing Medicare spending or measured provider fragmentation. Subspecialists also increase the use of newer therapies and diagnosis-specific clinical trials. These findings show that specialization raises productivity when expertise is closely matched to the task.

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Keywords: Physician Specialization, Mortality, Health Care Access

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1 Introduction

In many knowledge-intensive professions, workers increasingly specialize as the relevant knowledge frontier expands. This pattern is especially salient in medicine, where physicians have increasingly narrowed their practice around specific diseases, procedures, technologies, or treatment domains (Cassel and Reuben, 2011; Dalen, Ryan and Alpert, 2017; Ricketts, Fraher and Spero, 2016; Karadakic et al., 2025*b*). Subspecialization may improve outcomes when expertise closely matches the task, but it may also create access and coordination costs when expertise is unevenly distributed. Despite the growth of subspecialization across medicine, its consequences for patient outcomes, spending, access, and care coordination remain incompletely understood.

We study this tradeoff in medical oncology, where subspecialization has grown rapidly and treatment decisions are complex, evolving, and consequential for survival. Medical oncologists increasingly focus on specific cancer types or related groups of cancers, such as 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*a*; Lozinski, 2024).[1]

Subspecialization may improve the precision of cancer care by allowing physicians to tailor treatments to the biological and clinical characteristics of specific cancers as well as individual patient needs. By focusing on a narrower set of cancer types, physicians build disease-specific expertise and stay closely aligned with rapidly evolving, cancer-specific treatment protocols. At the same time, subspecialists are unevenly distributed geographically, which may limit access for patients in smaller or underserved markets (Dingel et al., 2023); descriptively, utilization of highly subspecialized oncology care is substantially higher in high-income areas than in lower-income areas (Karadakic et al., 2025*a*). Moreover, many cancer patients present with multiple chronic conditions or overlapping diagnoses, increasing the complexity of coordinating care across providers and possibly worsening survival (Cebul et al., 2008).[2] These competing forces make oncology a particularly well-suited setting for studying whether matched disease-specific expertise improves outcomes and whether any gains are offset by the costs that make subspecialization theoretically ambiguous: reduced access, higher spending, or more fragmented care.

[1] See Appendix Figure D1 for trends in medical oncology subspecialization by cancer type, 2008–2020.

[2] Cancer care often spans medical, surgical, radiation, supportive, and primary care teams; poor coordination can delay or disrupt care, weaken toxicity management, or generate inconsistent recommendations across clinicians (Institute of Medicine, 2013).

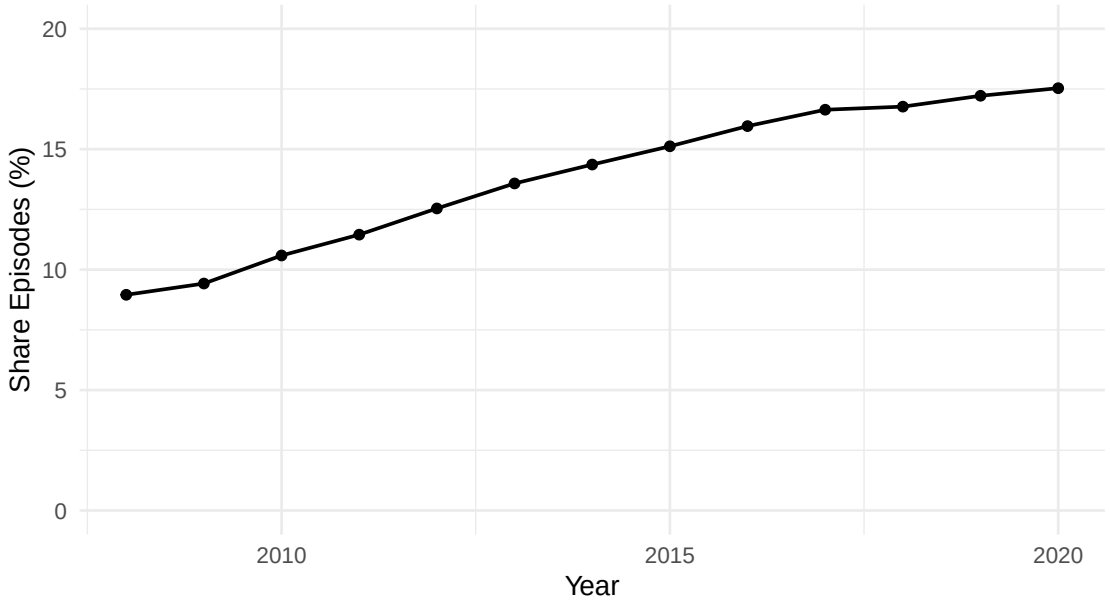


Figure 1: Trends in Subspecialization in Cancer Care, 2008-2020

Notes: This figure presents trends in subspecialization for beneficiary chemotherapy episodes based on results from Karadakic et al. (2025*a*). The figure depicts the share of chemotherapy episodes managed by subspecialized oncologists for patients with the relevant cancer type (e.g., a thoracic cancer subspecialist for patients with lung cancer). 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.

In this paper, we estimate the causal effect of access to cancer-type-specific subspecialized care, defined in our main specification as having at least one office visit with an oncologist who subspecializes in the patient's cancer type during the year chemotherapy begins. We study 2.2 million first six-month chemotherapy episodes among fee-for-service Medicare beneficiaries from 2008 to 2020 and use an instrumental variables (IV) strategy based on differential distance to the nearest relevant subspecialist relative to the nearest general oncologist. Our specification includes ZCTA and cancer type-by-year fixed effects, so identification comes from within-location variation in relative access to subspecialists across cancer types and over time, rather than from comparisons of high- and low-access regions. Differential distance strongly predicts access to a relevant subspecialist. The identifying assumption is that the residual variation in differential distance is otherwise unrelated to unobserved determinants of patient outcomes. Supporting this assumption, we show below that differential distance is balanced on predicted mortality, comorbidities, prior utilization, and socioeconomic characteristics after applying the design controls.

Our analysis shows that patients with access to relevant subspecialists experience lower mortality and are more likely to enroll in disease-specific clinical trials. IV es-

timates indicate that access to a relevant subspecialist reduces mortality by about 4 to 5 percentage points over medium-term horizons. Effects are small and statistically insignificant in the first six months, emerge around one year after chemotherapy initiation, and persist through three years, when mortality falls by 4.5 percentage points, or 10 percent relative to the mean. Despite these survival improvements, we do not find an increase in overall health care spending. Total episode spending and aggregate spending within Parts A, B, and D are statistically unchanged. Beneath these aggregate null effects, however, subspecialist access reallocates spending across categories, reducing chemotherapy spending while increasing other Part B spending.

The magnitude of these survival gains is economically meaningful. To benchmark their scale, major oncology innovations such as immunotherapy have generated large survival improvements in selected trial populations (Robert et al., 2011, 2015; Hamid et al., 2017), often at high treatment cost (Andrews, 2015). Although cross-study comparisons are necessarily imperfect, the three-year mortality reduction we estimate is roughly half the three-year survival gain reported for ipilimumab plus dacarbazine in previously untreated metastatic melanoma. Notably, in contrast to the novel therapeutics, survival gains from being treated by a subspecialist occur without detectable increases in Medicare spending.

Several additional analyses support the interpretation of the mortality results. The estimates are robust to allowing the sample to vary with the follow-up horizon, adding controls for oncologist volume, including ZCTA-specific linear time trends, and using an alternative treatment definition based on whether the episode-attributed oncologist is a relevant subspecialist. Falsification exercises that assign patients to differential distances corresponding to subspecialists of unrelated cancers yield no corresponding mortality effects. We also address the concern that subspecialists may selectively initiate chemotherapy among lower-risk patients. In a separate sample of first observed oncology visits—defined as the first observed encounters between patients and any oncologist prior to chemotherapy initiation—first visits with subspecialists have small and statistically insignificant effects on subsequent chemotherapy initiation. Among first visits that are followed by chemotherapy, differential distance is also balanced on predicted mortality.

We next examine both the mechanisms underlying the survival gains and the possible offsetting costs of subspecialization. Patients with access to relevant subspecialists are more likely to enroll in diagnosis-specific clinical trials and to receive recently approved cancer therapies, suggesting that improved access to novel treatments may be one pathway for better outcomes. Rather than fragmenting care, access to relevant subspecialists appears to concentrate provider involvement during chemotherapy episodes: the

number of visits and diversity of provider specialties remain unchanged, while the number of unique providers declines, consistent with relevant subspecialists serving as focal clinicians for disease-specific management. The mortality benefits are also larger when an oncologist's practice is more concentrated in the patient's specific cancer type, further supporting the disease-specific expertise interpretation. Finally, a complier analysis shows that patients whose provider type (subspecialist vs. generalist) is influenced by differential distance resemble the broader chemotherapy population in many respects, supporting the external validity of our estimates.

Our findings contribute to several strands of literature. First, we contribute to the economic literature on specialization, the division of labor, and the organization of knowledge (Smith, 1819; Becker and Murphy, 1992; Rosen, 1983; Baumgardner, 1988; Garicano, 2000). Economic theory predicts that specialization can increase efficiency and expertise but may create trade-offs related to coordination and access (Cebul et al., 2008). Related work on knowledge production argues that as knowledge accumulates, individual experts may face increasing burdens of expertise, leading to narrower specialization and greater reliance on teams (Jones, 2009; Wuchty, Jones and Uzzi, 2007). We study these trade-offs in medical oncology, a setting where specialization has expanded rapidly alongside the growth of medical knowledge and targeted therapies (Karadakic et al., 2025*a*; 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.[3] Among Medicare beneficiaries—predominantly individuals 65 and older—cancer care is a significant driver of health care utilization and costs. In 2015, national cancer-related health care spending was 29 percent of overall Medicare spending, or $183 billion, reflecting the high prevalence and complexity of cancer management in this population (Mariotto et al., 2020; Kaiser Family Foundation, 2025). Cancer spending has risen substantially over the past decade, reflecting in part the growing availability and use of increasingly expensive anticancer therapies (Kyle, Dusetzina and Keating, 2022). The unique challenges posed by cancer in older adults, including comorbidities, frailty, and socioeconomic factors, necessitate specialized and coordinated approaches to care.[4]

Medical oncologists—physicians trained in both internal medicine and oncology—treat patients 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 covered under Medicare Part B, and oral therapy, which involves prescription medications taken by the patient, typically in pill form and covered under Medicare Part D.

In addition to prescribing and administering systemic anti-cancer and supportive therapies, medical oncologists play a central role in coordinating care within multidisciplinary teams that include surgical and/or radiation oncologists and other health care professionals. Advances in treatment over the past decades—such as targeted therapies

[3]The 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, 2025*a*).

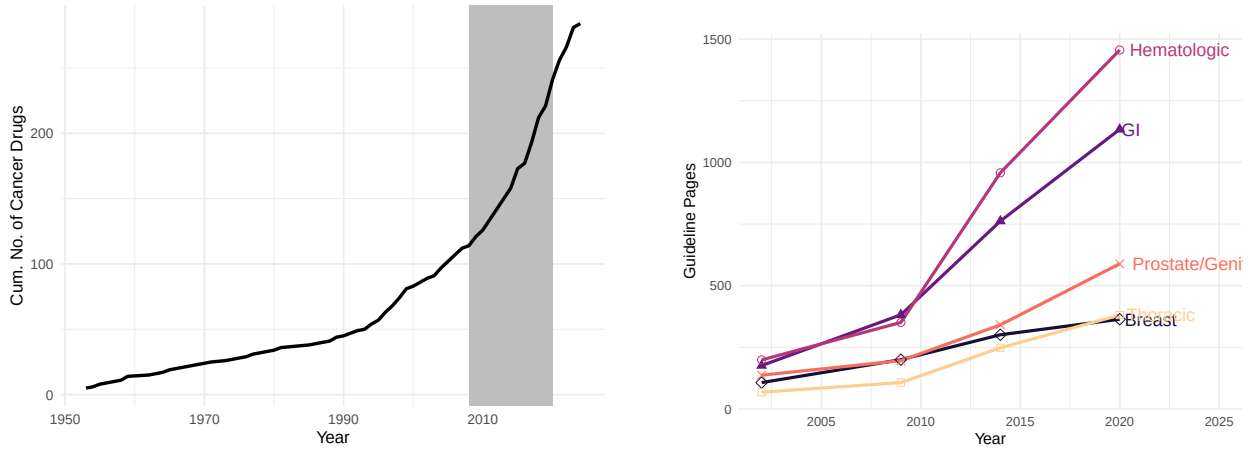
[4]The importance of subspecialization in ensuring up-to-date knowledge and optimal treatment is explicitly recognized by some cancer clinics and providers, who may emphasize it as part of their core mission statements (see e.g. Yale Cancer Clinic (2025)).

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.[5] 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) have 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, or hematologic cancers, allowing them to develop expertise in managing the nuances of these 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 may also introduce challenges, particularly regarding equitable access to specialized care and potential fragmentation of care (Karadakic et al., 2025*a*).[6] Appendix Figure D2 shows that chemotherapy episodes among patients residing in higher-income ZCTAs are more likely to be managed by highly subspecialized oncologists than episodes among patients residing in many lower-income ZCTAs. This geographic disparity is especially consequential for Medicare beneficiaries, who may face barriers to travel or rely on local providers for care. These descriptive differences motivate the access concern, but they are distinct from our causal design: our empirical strategy does not rely on cross-sectional differences or differential trends across regions; instead, it uses within-ZCTA, cancer-type-by-year variation in differential distance to estimate the consequences of access to the relevant cancer-type subspecialist.

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

[6]For example, if subspecialized oncologists require other specialists' expertise to manage care.



(a) Cumulative No. of Cancer Drugs

(b) Medical Guideline Page Count

Figure 2: Cancer Drug and Medical Guideline Expansion across Time

Notes: 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). We separated guideline page counts by the main cancer categories in our sample (hematologic, gastrointestinal, prostate/genitourinary, breast, and thoracic cancer). We obtained guideline page counts from Lozinski (2024). Abbreviations: GI=gastrointestinal, Prostate/Geni=prostate/genitourinary.

3 Data and Instrument Construction

We draw on multiple data sources, with the primary dataset consisting of chemotherapy episodes from 2008 to 2020 constructed using 100 percent Medicare fee-for-service claims accessed through the CMS Virtual Research Data Center. We use Medicare Parts A, B, and D data from 2007 to 2021. We supplement these data with ZCTA-level socioeconomic characteristics from the U.S. Census Bureau. We further link clinical trial information from ClinicalTrials.gov using National Clinical Trial (NCT) identifiers in claims, and merge data on FDA approval years of cancer drugs from the NCI SEER CanMED database to HCPCS and NDC codes in our sample (National Cancer Institute, 2025*b*).

3.1 Chemotherapy Episode Data

The foundation of our analysis is a dataset of cancer care episodes constructed following the Oncology Care Model (OCM) methodology (CMS, 2025; Keating et al., 2021). OCM was a value-based payment and care delivery model introduced by CMS to improve the quality and coordination of care for Medicare beneficiaries undergoing chemother-

apy while reducing costs.[7] Using OCM definitions allows us to adopt an established, "industry standard" approach to measuring cancer care. Specifically, we define non-overlapping six-month chemotherapy episodes, assign episodes to a single cancer type, and attribute care to a principal medical oncologist based on the plurality of office visits during the episode.[8] This OCM-based episode structure is central to our empirical design: chemotherapy initiation provides a common index date, claims during the episode permit assignment of cancer type and attribution to an oncologist, and the episode window allows us to measure spending, treatment choices, clinical trial enrollment, provider mix, and mortality on a comparable basis across patients.

We construct the episode-level dataset using 100 percent Medicare fee-for-service claims for beneficiaries with cancer who receive oral or physician-administered chemotherapy, including cytotoxic chemotherapy, targeted therapy, immunotherapy, and hormonal therapy (CMS, 2020; Keating et al., 2021). The sample is restricted to beneficiaries enrolled in Medicare Parts A and B who are not receiving the End-Stage Renal Disease (ESRD) benefit.

Chemotherapy is identified using physician-administered claims from the Carrier and Outpatient files (requiring a cancer diagnosis on the claim) and prescription fills for chemotherapy agents from the Part D event file.[9] Part D fills are included only if a corresponding Part B claim with a cancer diagnosis occurs within the preceding 59 days, increasing the likelihood that prescriptions are related to cancer treatment.[10] Using this approach, we define 180-day chemotherapy episodes beginning with the first chemotherapy claim. Each episode is attributed to the oncologist with the plurality of evaluation and management (E&M) office visits, and cancer type is assigned based on the plurality of cancer diagnoses codes on these office visit claims (see Appendix Table E1).

3.2 Definition of Subspecialized Oncologists

To classify whether a chemotherapy episode is attributed to a subspecialized oncologist, we define subspecialists as oncologists who provide at least 80 percent of their chemotherapy episodes for patients with a single cancer type (e.g., breast cancer) or

[7] OCM operated from July 2016 to December 2022; we apply its episode identification methodology throughout our study period.

[8] We restrict to office visits with medical oncologists defined using CMS specialty codes 82 (hematology), 83 (hematology/oncology), 90 (medical oncology), and 98 (gynecologic oncology); we included gynecologic oncologists because they frequently administer chemotherapy.

[9] A full list of Part B chemotherapy drugs and National Drug Codes (NDCs) are available upon request.

[10] To accommodate the look-back period and episode construction, we use claims data from 2007 to 2021 and restrict the final analysis sample to chemotherapy episodes initiated between 2008 and 2020.

a closely related group of cancer types (e.g., gastrointestinal cancers) in a given year. This threshold balances the need to identify clinicians whose practice is concentrated in specific cancer types with the reality that even subspecialists may treat common cancers for financial and clinical reasons (Karadakic et al., 2025*a*). In additional analyses, we also examine a continuous measure of specialization based on the share of chemotherapy episodes within cancer types. Our episode-level dataset includes chemotherapy episodes across nine broad cancer groups: breast, gastrointestinal (GI), gynecologic, head and neck, hematologic, prostate/genitourinary, melanoma, thoracic, and other cancers.

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 during the treatment period. As a secondary measure, we consider whether the chemotherapy episode itself is attributed to a subspecialist, reflecting a more intensive form of subspecialist involvement. Because the two measures are highly correlated and yield similar results, we focus on the office visit–based definition in our main analyses. Throughout, our estimates should be interpreted as the effect of access to a subspecialist whose practice is concentrated in the patient's own cancer type, not as the effect of specialist care in general or access to oncology subspecialists for unrelated cancers.

3.3 Main Sample and Variable Definitions

The chemotherapy episode sample includes beneficiary identifiers, the date of treatment initiation, primary cancer type (Table E1), treatment modality (physician administered vs oral), and the National Provider Identifier (NPI) of the attributed oncologist.

Our main analysis focuses on five cancer groups—breast, gastrointestinal (GI), hematologic, prostate/genitourinary, and thoracic cancers—where systemic therapy is commonly used. We exclude head and neck, skin, gynecologic, and other cancers due to limited sample sizes for defining subspecialists. The sample is restricted to each beneficiary's first chemotherapy episode to capture patients at a comparable point in their treatment trajectory and to avoid repeat observations. We further limit the sample to beneficiaries aged 67 and older to ensure at least two years of pre-treatment claims history. Appendix Table E2 summarizes these restrictions.

We augment the episode-level data with beneficiary characteristics from the Master Beneficiary Summary File, including age, ZIP code, date of death, sex, race, and indicators for 27 chronic conditions.[11] ZIP codes are mapped to ZCTAs using publicly

[11]We use end-of-year indicators for all 27 chronic conditions from the Chronic Conditions Warehouse

available crosswalks (Audirac, 2024). We also construct annual ZCTA-level measures of health care access using Medicare data, including beneficiary demographics, enrollment characteristics, and provider supply.[12] These are combined with ZCTA-level population counts and median household income from the American Community Survey, measured at the ZCTA–year level. Summary statistics are reported in Appendix Table E3.

To measure spending, we aggregate episode-level expenditures from treatment initiation through 180 days (or until death if earlier), including Medicare payments, beneficiary out-of-pocket spending, and payments from other payers. We further disaggregate spending by Medicare Part (A, B, and D), claim source, and Restructured Berenson-Eggers Type of Service (BETOS) categories (CMS, 2024). Additional details are provided in Appendix B.

Next, we construct additional outcomes and utilization measures using Medicare claims. Clinical trial participation is identified using National Clinical Trial (NCT) identifiers in Carrier claims. We also measure prior health care utilization—including hospitalizations, primary care visits, emergency department visits, and cancer screening—to capture pre-treatment health care engagement. To capture baseline health risk, we construct a measure of predicted mortality using out-of-sample predictions from year-specific linear probability models of death within the calendar year. The models are estimated using Medicare claims and enrollment data from a training sample of beneficiaries not included in the chemotherapy episode sample, and then applied to individuals in our analysis sample to generate predicted mortality scores. Covariates include demographics, chronic conditions, and prior-year health care utilization.[13] By construction, this measure reflects baseline mortality risk based on pre-treatment characteristics and is independent of treatment decisions in the chemotherapy episode.

3.4 Differential Distance Measure

The core of the empirical strategy relies on an instrumental variable constructed from a differential distance: the distance between a beneficiary's residence and the nearest general oncologist, minus the distance between the beneficiary's residence and the nearest subspecialized oncologist for the beneficiary's 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

(CCW) (CMS, 2021).

[12]Measures include average age, shares of beneficiaries who are full fee-for-service, disabled, male, or Black, total beneficiary counts, and provider supply (total providers, primary care, and mental health physicians).

[13]For a detailed overview of the variables used to construct predicted mortality, see Appendix Table E4.

visits recorded in Medicare Part B claims. Year-specific distance matrices are drawn from the NBER ZIP Code Distance Database (National Bureau of Economic Research, 2025).

Formally, we define differential distance (DD) for a beneficiary with cancer type c and ZCTA z 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 concentrate at least 80 percent of their chemotherapy episodes within a single group of related cancer types (e.g., breast, gastrointestinal, thoracic). Due to increases in subspecialization among medical oncologists over time, differential distances between subspecialists and generalists become smaller (see Appendix Figure D4).

$$\mathbf{DD}_{czt} = \sinh^{-1}(\text{Dist. Subspecialist}_{czt} - \text{Dist. Generalist}_{zt}) \tag{1}$$

To construct our main sample, we restrict chemotherapy episodes to those with non-negative differential distances, meaning cases in which subspecialists are located farther 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.[14]

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.[15] This transformation is frequently used to approximate the logarithmic transformation in regression models, while simultaneously allowing for zero values of a variable. When IHS transformations appear on the right-hand side of the equation, as in our case, the slope parameter's interpretation changes slightly, particularly for small values of the explanatory variable (Bellemare and Wichman, 2020).

[14] A table with detailed cancer types and corresponding ICD codes can be found in Appendix Table E1.

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

4 Empirical Strategy

Our empirical analysis aims to estimate the effect of access to subspecialized oncologists on patient health outcomes and spending. Simple comparisons between patients 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 oncologic care arising from differences in their geographic proximity to relevant subspecialized oncologists relative to general oncologists. The design uses a distance-based instrument, a common approach used in health economics (McClellan, McNeil and Newhouse, 1994; Card, Fenizia and Silver, 2023; Gruber et al., 2025). A central concern with such instruments is that distance to subspecialized physicians may be correlated with patient characteristics that also affect outcomes. For example, patients in more affluent suburban areas may live farther from subspecialists, who are often located in academic medical centers in downtown areas, while also having better underlying health. We address this concern in two ways. First, we use differential distance rather than absolute distance, measuring the distance to the nearest relevant subspecialist relative to the nearest general oncologist. Second, we include ZCTA fixed effects, so identification comes from comparisons among patients residing in the same geographic area.

The remaining variation in differential distance arises across cancer types within ZCTAs and over time. Subspecialists are unevenly distributed across disease areas, so two patients in the same ZCTA may face different relative access depending on their cancer type: a patient with breast cancer may live near a breast cancer subspecialist, while a patient with thoracic cancer in the same ZCTA may not live near a thoracic cancer subspecialist. Additional variation arises as oncologists enter, exit, move, or change the degree of concentration of their practice in particular cancers. Thus, the design does not compare high- and low-access regions, but instead uses cancer-type-specific variation in relative access within locations and over time.

We use two-stage least squares to estimate the effect of subspecialist access on beneficiaries' outcomes. In the first stage, we estimate the effect of differential distance on access to a subspecialized oncologist of the relevant cancer type:

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

for episode i in ZCTA z, cancer type c, and year t, where $\text{DD}_{c(i)t(i)z(i)}$ denotes the inverse hyperbolic sine of the differential distance between the nearest subspecialized oncologist of the relevant cancer type and the nearest general oncologist. The vector X_i includes

beneficiary demographic and clinical characteristics, as well as time-varying ZCTA-level controls. $D_{t(i)z(i)}$ denotes the natural logarithm of the distance to the nearest oncologist of any kind. $\lambda_{c(i)t(i)}$ is a cancer type-by-year fixed effect, and $\gamma_{z(i)}$ is a ZCTA fixed effect.[16] The dependent variable, Access_i, is a binary indicator for whether a beneficiary has at least one office visit with a subspecialized oncologist of the relevant cancer type during the year in which chemotherapy is initiated. As an alternative specification, we define treatment as whether the episode-attributed oncologist is a subspecialist in the relevant cancer type.

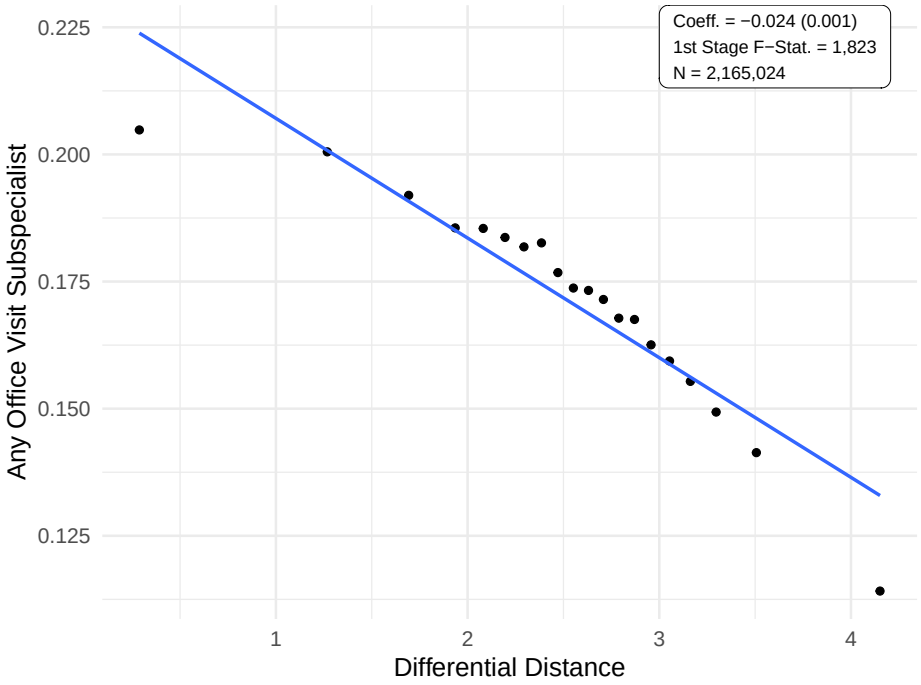


Figure 3: First Stage Relationship

Notes: This figure presents a binned scatter plot of the first-stage relationship after residualizing both the instrument and the treatment variable with respect to the design controls, including ZCTA fixed effects and cancer type-by-year fixed effects. Residuals are re-centered by adding back each variable's sample mean. The instrument is partitioned into ventiles, and each point represents the mean of the residualized instrument and treatment variable within a bin. The x-axis shows the inverse hyperbolic sine of differential distance, and the y-axis shows the probability of access to a subspecialized oncologist. The sample consists of 2,165,024 first chemotherapy episodes from 2008 to 2020. The estimated first-stage coefficient from Equation 2 is reported in the top-right corner of the figure, along with the corresponding Kleibergen–Paap F-statistic.

Figure 3 presents a binned scatter plot of the first-stage relationship after residualizing both the instrument and the treatment with respect to the design controls, specifi-

[16] We define fixed effects using 45 detailed cancer types, whereas subspecialist classification is based on five broader cancer categories. Table E1 provides an overview of the ICD codes corresponding to each category.

cally ZCTA fixed effects and cancer-type-by-year fixed effects. The figure shows a clear, negative relationship between differential distance and the probability of receiving care from a subspecialized oncologist, consistent with a strong and monotonic first stage. The corresponding first-stage estimate confirms 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.

Figure 3 also clarifies the access margin identified by the instrument. Even in the highest ventile of residual differential distance, roughly 11 percent of patients receive subspecialist care, indicating that some patients reach relevant subspecialists despite unfavorable distance. Even in the lowest ventile of residual differential distance, nearly 80 percent of patients do not receive subspecialist care. Thus, the difference in subspecialist use between the lowest and highest ventiles is approximately 19 percentage points, implying that differential distance shifts provider type for a meaningful but minority share of patients. For the complier group identified by the instrument, relative distance is the margin that shifts patients into or out of subspecialist care. The remaining variation in realized use suggests that referral, information, navigation, capacity, or other non-distance frictions may also matter.

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

$$Y_i = \alpha + \beta \widehat{\text{Access}}_i + \delta X_i + \lambda_{c(i)t(i)} + \gamma_{z(i)} + \psi \text{D}_{t(i)z(i)} + \varepsilon_i \tag{3}$$

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

To illustrate the variation underlying our empirical strategy, Appendix Figure D5 presents the distribution of within-ZCTA variation in the residualized differential distance measure across cancer types and over time. We first residualize the instrument with respect to ZCTA and cancer type-by-year fixed effects and then compute two complementary measures: (i) the standard deviation across cancer types within each ZCTA-year, capturing differences in access across cancer types within the same location and year, and (ii) the standard deviation over time within each ZCTA-cancer type, capturing changes in access within locations over time. Both measures are ZCTA-level averages, summarized using observation weights to reflect variation in the estimation sample. The figure shows substantial overlap between the two distributions, indicating that both cross-cancer and temporal variation contribute meaningfully to the instrument's variation. To quantify their relative importance, we decompose the residualized instrument using ZCTA-by-cancer type and ZCTA-by-year fixed effects. The decomposition indi-

cates that 45.8 percent of the explained variation in the residualized instrument is attributable to cross-cancer differences within ZCTAs, while 54.2 percent is attributable to temporal variation within ZCTAs. Taken together, these results highlight that the variation in the instrument reflects a combination of within-location differences across cancer types and changes in access to subspecialists over time.

To further 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 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 E5 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 we hold 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.[17]

[17]This 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*a*), 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 instrument's identifying variation.

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 relative distance to a subspecialist (compared to a general oncologist) must weakly reduce the likelihood of accessing subspecialized care for all individuals, ruling out "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 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 D6).

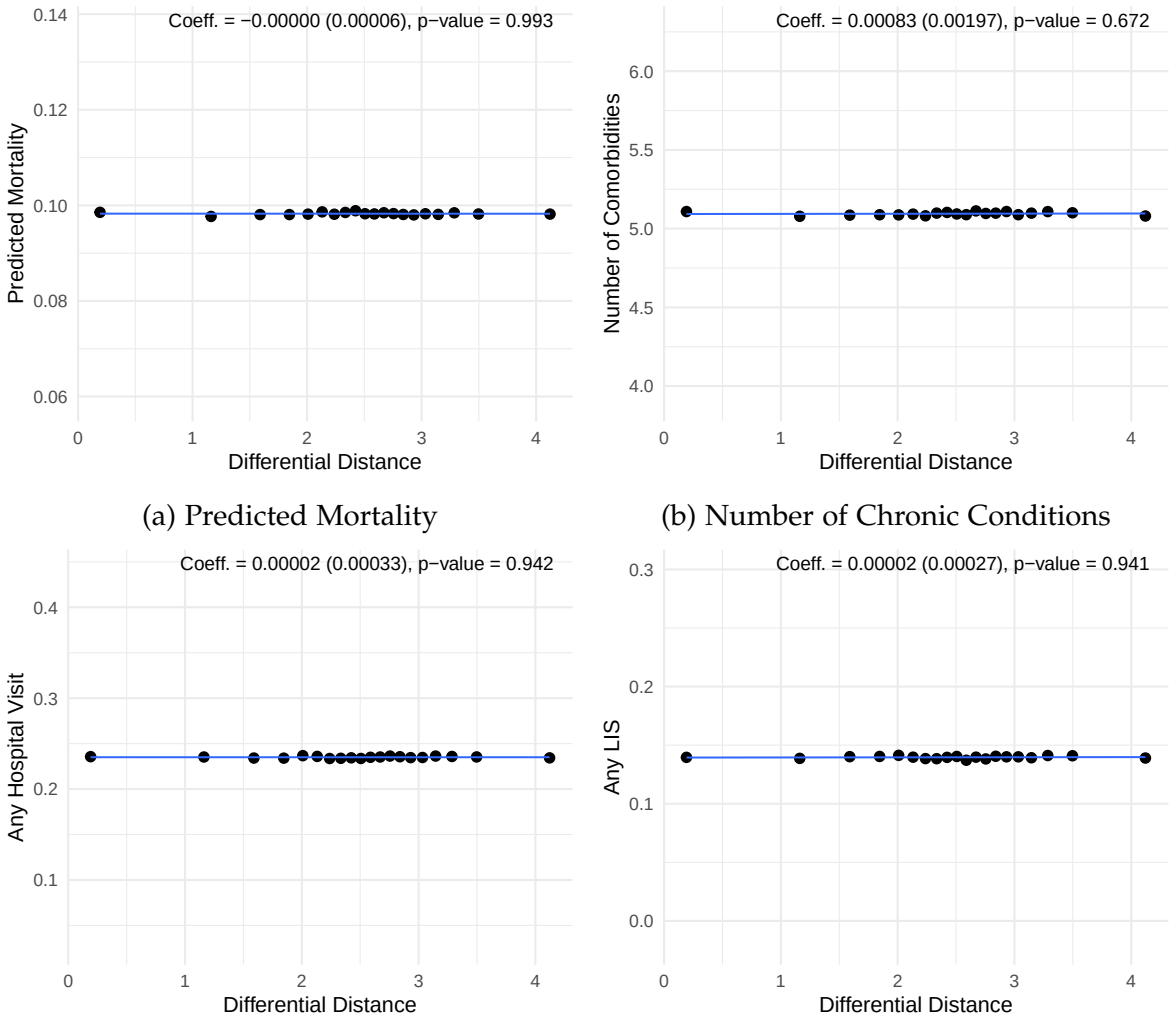
A second key requirement is the independence assumption: conditional on fixed effects, differential distance must be uncorrelated with unobserved determinants of patient outcomes. Violations of this assumption could arise if differential distance is systematically related to patient characteristics, 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 are similar in spirit to negative-control outcome tests, which probe whether the instrument predicts variables that should not be affected by treatment but may reveal alternative pathways linking the instrument to outcomes.

Figure 4 presents graphical representations of balance-test regressions for four selected variables capturing a broad range of patient characteristics: predicted mortality, an out-of-sample measure of baseline annual mortality risk based on pre-treatment characteristics,[18] the number of chronic conditions, prior healthcare utilization, and socioeconomic status, proxied by low-income subsidy (LIS) receipt. After residualizing the characteristics and differential distance with the design controls (i.e., ZCTA and cancer type-by-year fixed effects), the relationships between them are negligible and statistically insignificant. Additional balance results for a broader set of variables are presented in Appendix Figure D7 and Appendix Table E6.

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 other than access to

[18] See Appendix Table E4 for details on the construction of predicted mortality.

a subspecialized oncologist.



(a) Predicted Mortality

(b) Number of Chronic Conditions

(c) Any Hospital Visits (Year Prior)

(d) Any Low Income Subsidy (Year Prior)

Figure 4: Balance of Patient Characteristics

Notes: These figures provide graphical representations of the balance-test regressions at the first chemotherapy episode level. We residualize each variable with respect to ZCTA and cancer type $\times$ year fixed effects and add back the corresponding sample mean to facilitate interpretation. The y-axis is centered at the mean of the respective variable and spans ± 0.5 standard deviations of the residualized variable. Within each panel, we report regression coefficients, standard errors, and p-values for both specifications. Panel A presents yearly predicted mortality (see Section 3 and Appendix Table E4 for definitions); Panel B presents the number of chronic conditions; Panel C presents an indicator for any hospital visit in the prior year; and Panel D presents an indicator for any month with a low-income subsidy in the prior year.

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 set-

tings—such as academic medical centers—that independently influence patient outcomes. In our setting, this concern is mitigated by the instrument's cancer-type specificity, which varies across cancer types and over time within ZCTAs. We further assess the plausibility of this assumption using falsification exercises presented in Section 5.3.

5 Main Results

This section presents the main outcome analyses. We first examine mortality and Medicare spending, then report robustness and falsification exercises, address selection into chemotherapy, and characterize the complier population induced by differential distance.

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 after starting chemotherapy. These indicators range from 90-day to 1,080-day mortality, in 90-day increments.

Figure 5 presents a residualized binned scatter plot of 360-day mortality against differential distance, along with a comparison to predicted mortality as a measure of baseline health risk. The figure shows a positive relationship between differential distance and 360-day mortality, indicating that greater differential distance is associated with higher mortality. Because predicted mortality is constructed as a baseline measure of annual mortality risk within a calendar year and is not directly comparable in levels to episode-based 360-day mortality, we express both series as deviations from their respective sample means. This scaling facilitates a comparison of their comovement with the instrument without conflating differences in levels. While 360-day mortality varies systematically with the instrument, predicted mortality remains flat, supporting balance in baseline health risk. Together, these patterns suggest that the instrument shifts mortality in the expected direction while remaining uncorrelated with baseline patient characteristics.

To estimate the effect of access to relevant subspecialists, we apply Equation 3 and report the 2SLS estimates in Figure 6, Panel 6a. The results indicate no statistically significant effects on mortality before the end of the first year after chemotherapy initiation.

[19] Chemotherapy 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.

Beyond this horizon, however, mortality declines steadily. Specifically, access to a relevant subspecialist reduces 360-day mortality by 4 percentage points. Relative to a mean 1-year mortality rate of 22.6 percent, this corresponds to a 17.7 percent reduction.

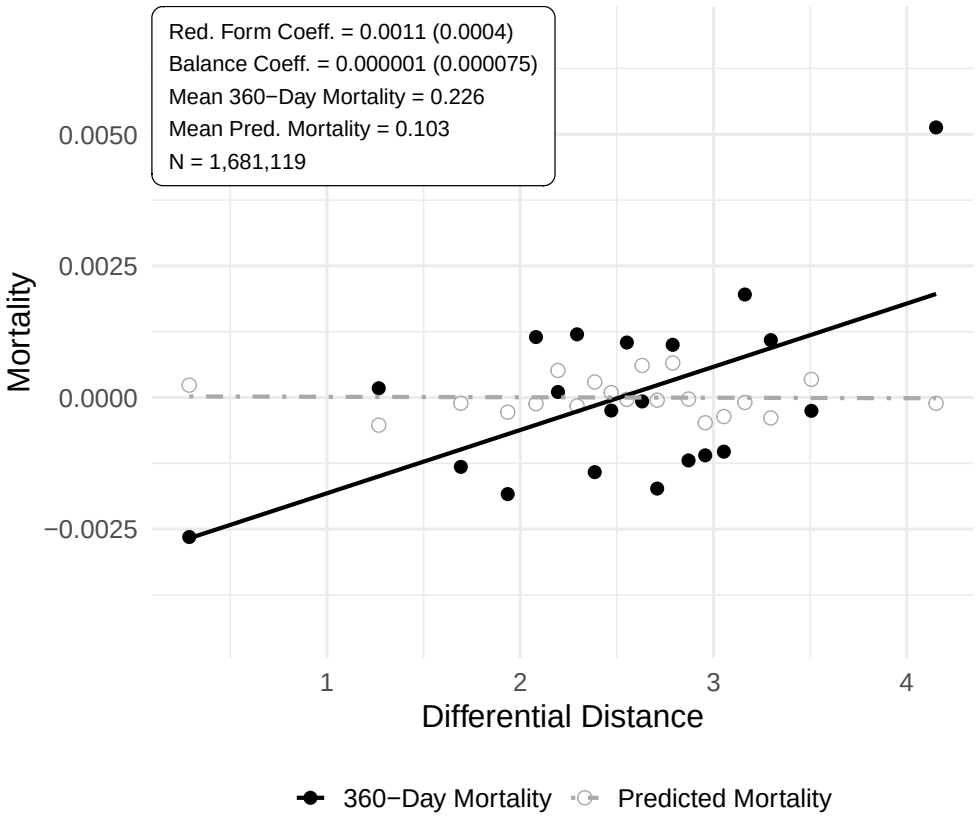


Figure 5: Balance and Reduced Form Relationship

Notes: This figure presents a binned scatter plot of the residualized relationship between differential distance and mortality outcomes for the sample of chemotherapy episodes from 2008 to 2017. All variables are residualized with respect to ZCTA fixed effects and cancer-type-by-year fixed effects. The instrument is partitioned into ventiles, and each point represents the mean of the residualized variables within a bin. The figure compares 360-day mortality (solid circles) to yearly predicted mortality (hollow circles), an out-of-sample measure of baseline annual mortality risk constructed from pre-treatment characteristics (see Section 3 and Appendix Table E4). While 360-day mortality is measured relative to chemotherapy initiation, predicted mortality captures baseline mortality risk within a calendar year. Because these measures are not directly comparable in level, we express both series as deviations from their respective sample means, allowing comparison of their comovement with the instrument. The annotation box reports the reduced-form and balance coefficients (incl. standard errors in parentheses) obtained from regressing the respective mortality outcome on the instrument (incl. ZCTA and cancer-type-by-year FEs), sample size, and the sample means of both outcomes.

The effect grows over time, reaching 5.1 percentage points at 540 days before stabilizing. As overall mortality increases with time since treatment, the effect becomes smaller when expressed relative to the rising baseline mortality rate. To examine the timing of these effects more directly, Panel 6b shows treatment-specific survival curves following

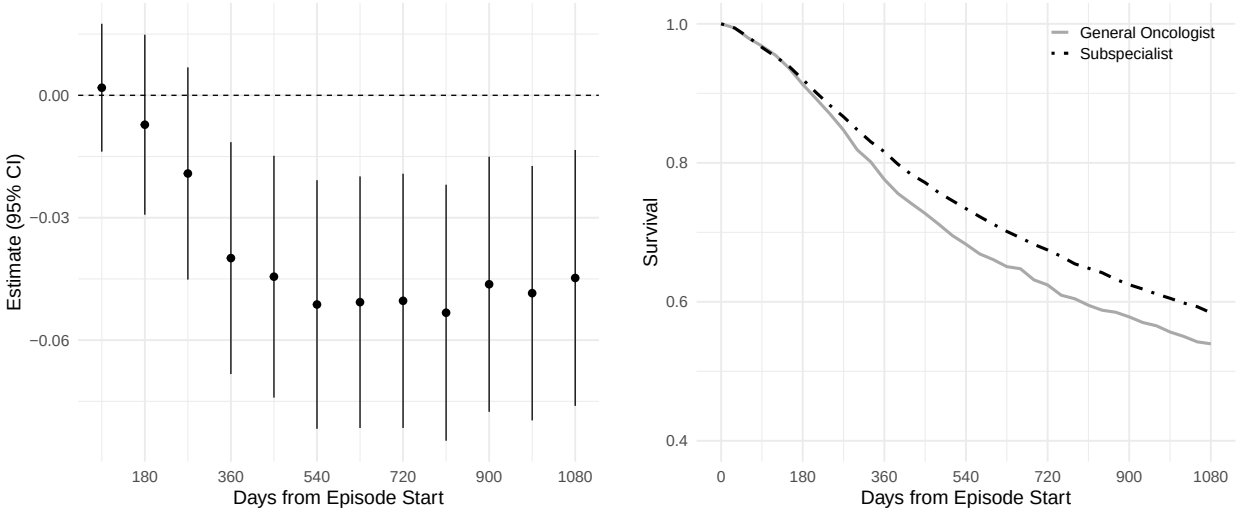
chemotherapy initiation. For each 30-day horizon t, we define an indicator $S_i(t)$ equal to one if beneficiary i survives up to t days after treatment initiation. We estimate survival under subspecialized and general oncologists' care using the same instrumental-variables framework as in the main specification, by interacting the survival indicator with the treatment assignment.

The survival curves show nearly identical outcomes in the short run, with no meaningful differences at 30 or 60 days. Differences begin to emerge after approximately six months and widen thereafter. At 360 days, survival is 81.6 percent for patients treated by relevant subspecialists compared with 77.6 percent for those treated by non-subspecialists, a gap of 4.0 percentage points. This difference is 5.1 percentage points at 540 days (73.4 percent versus 68.3 percent) and remains persistent at longer horizons, with survival at 1,080 days equal to 58.4 percent under subspecialized care and 53.9 percent under non-subspecialized care (4.5 percentage points). While these differences are stable in absolute terms, they are smaller when expressed relative to the rising baseline mortality risk at longer horizons. This pattern is consistent with the notion that, as time since treatment increases, competing health risks limit the extent to which improvements in cancer care can translate into proportional gains in survival. Overall, the survival curves suggest that mortality differences associated with subspecialized care are not immediate but emerge in the months after treatment initiation and persist through three years of follow-up.

OLS estimates in Appendix Table E7 suggest that selection into subspecialist care in the chemotherapy-episode sample may not be one-dimensional. At 180 days, the OLS estimate is more negative than the 2SLS estimate, consistent with favorable short-run selection into subspecialist care. By 360 days and beyond, however, the OLS estimates are much less negative than the 2SLS estimates and become positive at 720 and 1,080 days. This pattern suggests that patients seeing subspecialists may appear healthier or more treatment-appropriate in dimensions related to short-run mortality, while also having more severe, complex, or aggressive cancers that worsen longer-run prognosis. Such dimensions may include stage, grade, histology, biomarkers, performance status, or treatment complexity, which are not fully observed in claims.

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, we estimate all outcomes on a common sample covering chemotherapy episodes from 2008 to 2017. In Appendix Table E8, 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.



(a) Mortality (b) Survival

Figure 6: Effect of Subspecialist Access on Mortality and Survival

Notes: This figure presents 2SLS estimates of the effects of access to a relevant subspecialist for first chemotherapy episodes from 2008 to 2017. Panel A shows effects on mortality at 90-day horizons, with point estimates and 95% confidence intervals from regressions that include the full set of controls and fixed effects specified in Equation 3. Standard errors are clustered at the ZCTA level. Panel B shows estimated survival probabilities following initiation of chemotherapy under subspecialist versus general oncologist care. For each horizon t, we define an indicator $S_i(t)$ equal to one if beneficiary i survives up to t days. The subspecialist survival curve is estimated by interacting $S_i(t)$ with the treatment indicator D_i, while the general oncologist survival curve is estimated by interacting $S_i(t)$ with $(D_i - 1)$. Both sets of estimates are obtained using 2SLS specifications with the same controls and fixed effects as in the main analysis. The resulting estimates recover treatment-specific survival probabilities over time, conditional on the identifying variation provided by differential distance.

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 E8), suggesting that our results are not explained by observable differences in oncologist episode 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 E8), indicating that our findings are not driven by differential trends across locations.

Finally, we consider an alternative treatment definition that equals 1 if the episode-attributed oncologist is a subspecialist in the relevant cancer type. This shifts the inter-

pretation from any access to subspecialized care to the attribution of a realized episode 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 E8), and may reflect a modest change in the composition of compliers.

Appendix Table E9 examines how the estimated mortality effects vary across alternative fixed-effect structures designed to isolate different sources of identifying variation in the instrument. The baseline specification exploits both cross-cancer differences in access to subspecialists within locations and changes in access over time. Column 2 primarily relies on within-ZCTA cross-cancer variation by including ZCTA $\times$ year fixed effects, while Column 3 primarily relies on within-ZCTA changes over time by including ZCTA $\times$ cancer-type fixed effects. Both sources of variation generate negative mortality effects of similar magnitude, although estimates based on cross-cancer variation are more precise. Overall, the results indicate that the main findings are not driven exclusively by either temporal or cross-cancer variation in subspecialist access.

A remaining concern is that the mortality gains may reflect selection into chemotherapy rather than differences in care conditional on treatment. For example, subspecialists might initiate chemotherapy for a different set of patients than general oncologists, mechanically changing the risk composition of the chemotherapy episode sample. To assess this possibility, Appendix A constructs a complementary sample of over 4.7 million first office visits with medical oncologists with a cancer diagnosis and examines whether first visits with subspecialists affect subsequent chemotherapy initiation. This sample is useful for studying selection before chemotherapy begins, but it is not a substitute for the main OCM-based chemotherapy episode sample, which provides a standardized treatment start date, cancer-type assignment, oncologist attribution, and comparable outcome windows.[20] The estimated effects on chemotherapy initiation within 90, 180, and 360 days are small relative to baseline initiation rates and statistically insignificant (Appendix Table A3). In addition, differential distance is essentially unrelated to predicted mortality both among first visits followed by chemotherapy and among first visits not followed by chemotherapy (Appendix Figure A1a). Taken together, these analyses provide little support for the concern that differential selection of lower-risk patients into chemotherapy drives the main estimates.

[20]Mortality in the first-visit analyses is measured from the first oncology visit rather than from chemotherapy initiation, as in the main chemotherapy sample.

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 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				
DD	-0.0237***	-0.0237***	-0.0237***	-0.0237***
	(0.0006)	(0.0006)	(0.0006)	(0.0006)
Panel B: Reduced Form				
DD	0.0006	0.0034	0.0000	-0.0015
	(0.0009)	(0.0032)	(0.0010)	(0.0028)
Panel C: Structural Form (OLS)				
Any Subspecialist Visit	0.1131***	0.1892***	0.1323***	0.0642***
	(0.0021)	(0.0076)	(0.0024)	(0.0066)
Panel D: 2SLS				
Any Subspecialist Visit	-0.0251	-0.1447	-0.0011	0.0620
	(0.0369)	(0.1340)	(0.0426)	(0.1167)
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 relevant subspecialist 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 the relevant subspecialist is not significantly associated with changes in the natural logarithm of Part A, Part B, or Part D spending. However, the 2SLS point estimates for Part A and Part B spending are negative and imprecise, in contrast to the positive and statistically significant associations observed in OLS models. This contrast suggests that patients who see subspecialists are positively selected on dimensions associated with higher spending. In the IV estimates, however, access to the relevant subspecialist does not increase aggregate episode spending.

To isolate chemotherapy-related spending, we use HCPCS codes from the Oncology Care Model to construct a measure of Part B chemotherapy drug spending. We also apply the 2024 Restructured BETOS Classification System (RBCS) to identify injection and infusion services. These decompositions indicate that subspecialist access reallocates spending within outpatient care rather than increasing aggregate treatment intensity. OCM-defined Part B chemotherapy drug spending falls by $2,568, and injection and infusion spending falls by $656. These reductions are offset by higher spending in other Part B services, leaving aggregate Part B spending and total episode spending statistically unchanged (Appendix Table E11). The offsetting increase does not arise from other RBCS categories, but from HCPCS codes that remain uncategorized under the RBCS classification. The spending results suggest that subspecialist access shifts resource allocation within the chemotherapy episode, rather than increasing overall spending.

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 that the relevance, monotonicity, and instrument-independence assumptions hold. One aspect we have not tested so far is the exclusion restriction: the instrument should affect outcomes (e.g., mortality) only through access to a subspecialist, not through alternative pathways (e.g., the quality of the 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 2 and the reduced form using 1,080-day

mortality as the outcome (see Appendix Figure D8). The logic is straightforward: if cancer-type-specific expertise is the key mechanism, then proximity to subspecialists in unrelated cancer types should not systematically affect mortality. Moreover, if mortality effects instead reflect features of the broader practice environment—such as being treated at a high-quality hospital—we would expect similar effects regardless of the oncologist's cancer-type specialization. Consistent with the specificity of expertise, these placebo measures do not produce statistically significant mortality effects. In the first stage, placebo instruments exhibit either no relationship or a positive relationship with subspecialist access. In the reduced form, we find no statistically significant relationship between the placebo instruments and mortality. Taken together, these results provide no support for alternative explanations based on general care quality or geographic confounding and instead reinforce the interpretation that our main results reflect cancer-type-specific subspecialist expertise.

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 the differential distance to subspecialists of unrelated cancer types and estimate a reduced-form regression of its association with mortality, conditional on the true instrument, controls, and fixed effects. Including the true instrument ensures that variation in the placebo instrument is orthogonal to the relevant source of identifying variation, so that the coefficient on the placebo instrument is the object of interest and should be zero under a valid exclusion restriction. This test evaluates whether residual variation in proximity to subspecialists, unrelated to the patient's cancer type, predicts outcomes after accounting for the relevant variation in the true instrument. If broader features of the local care environment, such as general oncology capacity or hospital quality, drive our results, this residual variation could still predict mortality. Appendix Figure D9 does not indicate such an association, supporting the validity of the exclusion restriction.

Taken together, these findings support the exclusion restriction by indicating that differential distance affects mortality primarily through access to relevant subspecialists with cancer-specific expertise. Consistent with this interpretation, the falsification tests do not indicate that proximity to subspecialists for unrelated cancer types improves outcomes for patients outside those subspecialists' area of expertise.

5.4 Complier Characteristics

Our instrumental variable estimates identify local average treatment effects (LATEs) for compliers—patients whose likelihood of receiving care from a relevant subspecial-

ist varies with distance. To characterize this group, we follow Chan, Card and Taylor (2023) and estimate complier means for demographic characteristics, cancer types, and comorbidities using IV-based reweighting methods.

Table E12 shows that compliers are broadly similar to the overall chemotherapy sample across most observable characteristics. Compliers are somewhat more likely to reside in urban ZCTAs and exhibit slightly lower predicted mortality and fewer chronic conditions, suggesting modestly better baseline health on observable dimensions. Across cancer types, compliers are overrepresented among prostate/genitourinary cancer patients and underrepresented among hematologic cancers, while representation is similar for breast, gastrointestinal, and thoracic cancers. Comorbidity profiles are likewise broadly comparable, with only modest differences across individual chronic conditions. Overall, these patterns suggest that the instrument primarily shifts access among patients with similar observable health profiles and does not appear to strongly select on baseline illness severity. The greater concentration of compliers in urban areas is also consistent with the instrument's underlying geography, as relative distance is most likely to affect provider choice in markets where patients have access to both subspecialized and general oncologists.

6 Treatment Pathways and Disease-Specific Expertise

This section examines how relevant subspecialist access may improve survival and whether it changes care delivery. We first study treatment pathways, including cancer-specific clinical trial enrollment and use of recently approved therapies. We then examine provider mix and fragmentation, a potential cost of subspecialization if narrower expertise requires patients to interact with more clinicians. Finally, we ask whether mortality benefits increase with the intensity of oncologists' disease-specific focus, treating specialization as both a binary label and a continuous measure of experience in the patient's cancer type.

6.1 Treatment Pathways

6.1.1 Clinical Trial Enrollment

One potential mechanism by which relevant subspecialist access improves survival is by facilitating access to novel, 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 broader cancer categories, allowing us to distinguish between overall clinical trial participation and enrollment in trials specific to a patient's cancer type.[21] In total, we identify and classify 11,051 unique trials. This classification lets us assess whether access to subspecialists increases overall trial participation or specifically improves access to trials directly relevant to a patient's diagnosis. Appendix C provides additional details on the classification methodology.

Patients with access to relevant subspecialists 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 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 D10).

To assess whether clinical trial enrollment is causally driven by access to subspecialists rather than selection into treatment, we estimate Equation 3 using a binary indicator of whether a beneficiary enrolled in a clinical trial within the year of chemotherapy initiation as the outcome.[22] Table 2 presents 2SLS and OLS estimates on the relationship between access to a relevant subspecialist 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; thus, these findings are unlikely to explain the full mortality benefits of subspecialized care. They reflect a key difference in healthcare utilization among individuals with access to relevant subspecialists that may

[21] Some trials span multiple cancer types and are not specific to a single diagnosis.

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

contribute modestly to the differences. Importantly, it also suggests clear differences in the practice patterns of relevant subspecialists.

Table 2: Clinical Trial Enrollment and Access to Subspecialized Oncologists

	Cancer Trials				
	Any	**Concordant**	**Discordant**	**Unspecified**	**Non-Cancer**
Panel A: First Stage					
DD	-0.0249***	-0.0249***	-0.0249***	-0.0249***	-0.0249***
	(0.0007)	(0.0007)	(0.0007)	(0.0007)	(0.0007)
Panel B: Reduced Form					
DD	-0.0006***	-0.0005***	-0.0001	0.0010	-0.0000
	(0.0002)	(0.0002)	(0.0001)	(0.0015)	(0.0000)
Panel C: Structural Form (OLS)					
Any Subspecialist Visit	0.0461***	0.0405***	0.0056***	0.0018***	0.0013***
	(0.0006)	(0.0005)	(0.0002)	(0.0001)	(0.0001)
Panel D: 2SLS					
Any Subspecialist Visit	0.0226***	0.0197***	0.0029	-0.0010	0.0006
	(0.0067)	(0.0063)	(0.0025)	(0.0015)	(0.0014)
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.

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. Subspecialists 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 estimate 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 (National Cancer Institute, 2025*b*). We conducted two complementary analyses. First, we examined whether access to relevant subspecialists 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. Because we cannot link approval dates to cancer-specific indications and Medicare claims lack detailed clinical information, we focus on the chemotherapy agent's first approval year rather than indication-specific approvals.

Appendix Table E13, 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 statistically significant 0.15 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 statistically significant effect is observed for newly approved Part B drugs (Column 2), we detect a marginally statistically significant increase in the use of recently approved Part D drugs (Column 3).

Taken together, we find suggestive evidence that subspecialist access modestly increases exposure to recently approved therapies, particularly oral agents covered under Part D. These results suggest that adoption of newer drugs may contribute to the mortality gains, but they are unlikely to fully explain them.

6.1.3 Provider Mix and Fragmentation

A potential concern with subspecialization is that narrower expertise could increase coordination costs by requiring patients to interact with more clinicians during treatment (Becker and Murphy, 1992; Cebul et al., 2008; Agha, Frandsen and Rebitzer, 2019). Such fragmentation can be costly in oncology if it delays treatment decisions, complicates toxicity management, or diffuses responsibility for care.[23] Fragmentation may be particularly relevant in settings where specialization divides care across multiple procedures or body systems. In oncology, however, care may be organized around subspecial-

[23]Prior work links fragmentation in cancer care to higher costs (Hussain et al., 2015) and, in some settings, worse short-run outcomes (Graboyes et al., 2017). Qualitative studies describe related pathways, including delayed or skipped care, medical errors or near misses, and loss of trust (Dillon et al., 2024), as well as communication breakdowns across clinicians (Prouty et al., 2014).

ties: relevant cancer-type subspecialists may serve as focal providers for disease-specific treatment decisions and provide comprehensive management throughout the treatment episode. Whether subspecialization increases or reduces fragmentation in cancer care is therefore an empirical question.

To assess whether access to a relevant subspecialist increases measured provider fragmentation, we use the Carrier and Outpatient files to identify all office visits occurring within the six months following initiation of each beneficiary's first chemotherapy episode. We construct three episode-level measures of provider involvement based on all billing providers observed in claims: (i) the total number of office visits, (ii) the number of unique providers seen, and (iii) the number of unique provider specialties involved, measured using CMS provider specialty codes. We identify unique providers using NPIs and include all clinicians billing for care, including oncologists, advanced practice providers, and other physicians or clinicians. 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 E14 show that subspecialist access has no discernible effect on the number of office visits or the diversity of provider specialties involved during the chemotherapy episode. However, we find that subspecialist access *reduces* the number of unique providers involved in the patient's care by 4.3 percent. Decomposing provider counts by subspecialist involvement indicates that increased access shifts care toward subspecialist-led episodes, reducing the number of non-subspecialist providers observed during the episode. The net effect is a decline in the total number of unique providers,[24] consistent with slightly more concentrated provider involvement. One interpretation is that relevant cancer-type subspecialists may serve as focal providers for disease-specific treatment decisions, reducing the need for additional provider involvement when patients would otherwise be managed by oncologists whose practices are less focused on the patient's cancer type. These findings suggest that subspecialist access improves survival without increasing measured care intensity or claims-based measures of provider fragmentation within chemotherapy episodes.

6.2 The Intensive Margin of Disease-Specific Expertise

Our binary measure captures specialization at the extensive margin but abstracts from variation in the intensity of oncologists' disease-specific focus. To capture this dimension, we construct leave-one-out measures of the share of each oncologist's chemother-

[24] Unique providers are captured using unique NPIs and include oncologists, physician assistants, nurse practitioners, and other types of health care providers.

apy episodes in the patient's cancer type, using both five broad cancer groups and 45 detailed cancer types within those groups (Appendix Table E1 columns 1 and 2). We then estimate 2SLS models that replace the binary access measure with these continuous specialization shares.

In Panel A of Table 3, we re-estimate Equation 3 using a continuous measure of specialization defined as the share of the episode-attributed oncologist's chemotherapy episodes occurring in the patient's relevant cancer type, rather than our binary access measure. The results closely mirror the main findings: mortality effects are negligible in the short run, emerge after approximately 180 days, and remain statistically significant at longer horizons. Quantitatively, a 10 percentage point increase in the oncologist's relevant cancer-type episode share reduces 1,080-day mortality by 1.02 percentage points, corresponding to a 2.3 percent decline relative to mean mortality at this horizon. For context, oncologists classified as subspecialists have an average relevant cancer-type episode share of 94.9 percent, compared to 28.5 percent among general oncologists—a difference of 66.4 percentage points. Applying the estimated coefficient to this difference implies that moving from the average general oncologist to the average subspecialist would reduce 1,080-day mortality by approximately $0.102 \times 0.664 = 0.067$, or about 15 percent relative to the mean. These estimates closely align with the results using the alternative treatment definition reported in Panel E of Table E8, indicating that the mortality benefits increase smoothly with the degree of cancer-specific specialization rather than reflecting only a discrete distinction between subspecialists and generalists.

Panel B of Table 3 presents analogous estimates using specialization shares constructed from 45 more detailed cancer types. These measures capture a more granular form of cancer-specific expertise than the broader cancer groupings used in the main analysis. Consistent with this interpretation, the estimated mortality effects are larger. A 10-percentage-point increase in the detailed cancer-type share reduces 1,080-day mortality by 1.5 percentage points, compared with 1.02 percentage points using the broader classification. At the same time, the difference in average specialization shares between subspecialists and general oncologists is smaller under the more granular definition—72.2 percent versus 21.0 percent, a gap of 51.2 percentage points—reflecting that narrower cancer categories mechanically lower measured concentration. Nevertheless, the implied mortality reduction from moving from a generalist to a subspecialist remains substantial. Overall, these findings suggest that the benefits of subspecialized care increase with the degree to which oncologists concentrate their practice on the patient's specific cancer type, consistent with cancer-specific expertise as an important mechanism underlying the mortality effects.[25]

[25]Appendix Table E15 reports the full set of first-stage, reduced-form, OLS, and 2SLS estimates using

Taken together, these results reinforce the paper's central findings. While our baseline estimates show that access to a relevant subspecialist improves survival at the extensive margin of subspecialization, the continuous measures indicate that these benefits extend along an intensive margin of specialization. Patients with episodes attributed to oncologists whose practices are more concentrated in the relevant cancer domain experience better outcomes, with larger effects when specialization is measured more precisely at the level of detailed cancer types. This pattern is consistent with cancer-specific expertise as an important mechanism underlying the mortality effects, especially given the unrelated-cancer falsification tests and the robustness of the main mortality estimates to oncologist volume controls. These findings suggest that both access to subspecialists and the degree to which their practice is focused on the patient's specific cancer contribute meaningfully to improved survival.

Table 3: Mortality Effects of Oncologists' Relevant Specialization

	180-Day	**360-Day**	**720-Day**	**1080-Day**
Panel A: Broad Cancer Type Shares				
Cancer Type Share	-0.016	-0.091***	-0.115***	-0.102***
	(0.025)	(0.033)	(0.036)	(0.036)
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)	1,054	1,054	1,054	1,054
Panel B: Detailed Cancer Type Shares				
Cancer Type Share	-0.024	-0.134***	-0.169***	-0.150***
	(0.038)	(0.049)	(0.053)	(0.054)
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 2SLS estimates of the effect of cancer type–specific subspecialization on mortality for all first chemotherapy episodes between 2008 and 2017. Subspecialization is measured as the share of the episode attributed oncologist's chemotherapy episodes accounted for by the patient's relevant cancer type (e.g. the share of breast cancer episodes of the oncologist for patients with breast cancer). Shares are constructed using a leave-one-out approach that excludes the index episode to avoid mechanical bias. Panel A uses shares based on five broad cancer groups (breast, gastrointestinal, hematologic, prostate/genitourinary, and thoracic). Panel B uses shares based on 45 detailed cancer types within these groups (see Appendix Table E1). 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.

the broad cancer-type definition. Appendix Table E16 reports the corresponding results for the detailed cancer-type definition.

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 relevant cancer-type subspecialists improves medium-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. The results point to disease-specific expertise: mortality reductions are larger when oncologists concentrate more of their practice on the patient's cancer type; falsification tests using subspecialists for unrelated cancers yield no corresponding survival effects; and access to relevant subspecialists increases cancer-specific clinical trial enrollment and modestly increases exposure to recently approved therapies. These gains occur without increases in overall spending or measured provider fragmentation within chemotherapy episodes, suggesting that subspecialization improves outcomes through better-aligned expertise rather than through more intensive or more dispersed measured care.

Our findings suggest that organizing expertise can generate health gains meaningful relative to those produced by costly therapeutic innovation. New cancer therapies have transformed oncology but often carry high treatment costs; by contrast, the mortality reductions documented here arise from differences in physician expertise and care delivery, with no increase in overall Medicare spending. These mechanisms are likely complementary: subspecialists may improve outcomes in part because they are better positioned to adopt new therapies, identify appropriate patients for treatment, manage toxicities of systemic therapies, and connect patients to clinical trials.

Despite these gains, access to subspecialized oncology care remains uneven. Descriptively, utilization of highly subspecialized oncology care is higher in high-income areas than in many lower-income areas (Karadakic et al., 2025*a*), while patients in areas with the highest cancer mortality rates are less likely to receive such care. These area-level patterns underscore that realized access to specialized expertise differs across places. Geographic proximity alone is also insufficient for realized access. Among the 268,543 patients in our study population with zero differential distance—for whom the nearest relevant subspecialist and nearest general oncologist are equally distant—only 30.5 percent received subspecialist care. While this statistic does not identify which patients face the largest residual access barriers and is based on our stringent claims-based definition of a relevant subspecialist as an oncologist whose practice is highly concentrated in the patient's cancer type, it suggests that distance is not the only barrier to receiving highly

subspecialized care; referral patterns, information frictions, navigation barriers, patient preferences, or capacity constraints may also shape effective access.

As a rough benchmark, the area between the estimated subspecialist and generalist survival curves in Figure 6 implies an average gain of approximately 39 survival days per patient over the 1,080-day follow-up horizon.[26] If applied to zero-differential-distance Medicare beneficiaries who do not receive subspecialist care during our study period, this estimate would imply roughly 20,000 life-years gained over follow-up. Because this extrapolation assumes constant treatment effects and abstracts from capacity constraints, referral networks, patient preferences, and equilibrium responses, we interpreted it only as an illustration of the potential scale of the matching margin.

More broadly, our findings suggest that the returns to specialization depend not only on the amount of specialized expertise in a market, but also on whether patients reach the expertise relevant to their clinical problem. In our setting, geographic proximity to subspecialists shifts care for some patients, but many patients do not receive subspecialist care even when relevant subspecialists are nearby. Furthermore, our evidence suggests that specialization creates value when expertise is organized around the relevant task—in this case, the patient's cancer type—rather than simply when more specialists are present. This pattern suggests that referral, information, and care-delivery frictions can shape how available expertise translates into realized subspecialist care and, ultimately, patient outcomes.

[26] We calculate survival days gained as the area between the estimated survival curves for patients with access to subspecialized versus general oncologists over the 1,080-day follow-up period, approximating the area using the trapezoidal rule over the estimated mortality horizons.

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A Selection into Chemotherapy

Our main analysis focuses on first chemotherapy episodes constructed using the Oncology Care Model (OCM) framework. This sample provides a standardized claims-based unit of observation: chemotherapy initiation defines a common episode start date, cancer type can be assigned using claims during the episode, care can be attributed to a medical oncologist, and spending, treatment choices, clinical trial enrollment, provider mix, and mortality can be measured over comparable windows. These features make the chemotherapy episode sample an ideal setting in which to study the consequences of access to subspecialized oncology care.

A potential concern with this design, however, is that conditioning on chemotherapy initiation may introduce selection. If subspecialist oncologists differ from generalists in the patients for whom they initiate chemotherapy, the observed mortality advantage in the chemotherapy sample could reflect selection into treatment rather than differences in care or treatment decisions after the episode begins. For example, subspecialists might initiate chemotherapy only among patients with better underlying prognosis, mechanically improving observed survival among those who receive chemotherapy.

To address this concern, we construct a complementary sample designed to capture the first observed point of contact between patients and medical oncologists in Medicare claims. We use a lookback period beginning in 2006 and first identify all cancer-related office visits with medical oncologists observed from 2006 through 2020, where the claim includes a cancer diagnosis. We then retain the earliest such visit for each beneficiary. Finally, because subspecialist classifications are available beginning in 2008, we restrict the analysis sample to beneficiaries whose first observed visit with a medical oncologist occurs between 2008 and 2020. This "first visit" sample serves two purposes. First, it approximates the initial observed clinical encounter during which patients and oncologists discuss and decide on potential systemic therapy, capturing the key decision point preceding initiation of chemotherapy. Second, by focusing on the first observed medical oncology visit associated with a cancer diagnosis, we reduce the likelihood of including routine follow-up or surveillance visits that do not reflect an active treatment decision.

Table A1 presents the sample construction process, including all exclusion criteria and the number of observations retained at each step. After restricting to visits with classified medical oncologists and excluding outliers, the final sample consists of 4,711,907 first observed office visits with medical oncologists.[27]

[27]The first-visit sample should be interpreted as the first observed cancer-related medical oncology visit in Medicare claims, rather than a first-ever oncology visit. Subspecialization 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.

We then link the first-visit sample to the chemotherapy episode data to determine whether a patient's first observed medical oncology visit is followed by initiation of chemotherapy. We do not require that the cancer diagnosis recorded at the first visit match the primary diagnosis assigned to the chemotherapy episode, since differences may arise from claims coding, diagnostic refinement, or updates to the patient's cancer classification. Among all first office visits with a medical oncologist, 32.3 percent are followed by initiation of chemotherapy within 360 days.[28]

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.

The first-visit-with-chemotherapy sample overlaps with, but is not identical to, the main chemotherapy episode sample. Some chemotherapy episodes cannot be linked to a qualifying first office visit because the initial cancer-related encounter may occur with a surgeon, urologist, advanced practice provider, or oncologist who cannot be classified at the time of the visit; because the relevant visit is not observed in claims; or because diagnosis coding differs between the initial visit and the chemotherapy episode. Thus, the first-visit analyses should be interpreted as complementary checks on selection into chemotherapy rather than as re-estimations of the main estimand using the same sample.

We enrich the linked first-visit data with measures of oncologist subspecialization, beneficiary characteristics, chronic conditions, ZCTA-level variables, and geographic access to generalist and relevant subspecialized oncologists, constructed using the same

[28]The share followed by chemotherapy may be lower than would be expected for true first oncology visits, reflecting the limitations of identifying first visits in Medicare claims.

approach as in the main analysis. We then estimate specifications parallel to the main chemotherapy-episode analysis.

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

for first visit i in ZCTA z in year t with cancer type c, 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 demographics and chronic conditions, as well as ZCTA-level controls that vary over time. D is the natural logarithm of distance to the nearest oncologist of any kind, $\lambda_{c(i)t(i)}$ is a cancer type by year fixed effect, and $\gamma_{z(i)}$ is a ZCTA fixed effect. The dependent variable Visit_i is a binary indicator variable for whether 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 4 in our chemotherapy analysis.

In a second step, we estimate the effect of initial subspecialist consultation on the probability of initiating chemotherapy. We estimate:

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

where Y_i is the outcome variable, for example, a binary indicator for whether chemotherapy was initiated within a specific time window (e.g., within 90, 180, or 360 days after the initial consultation).

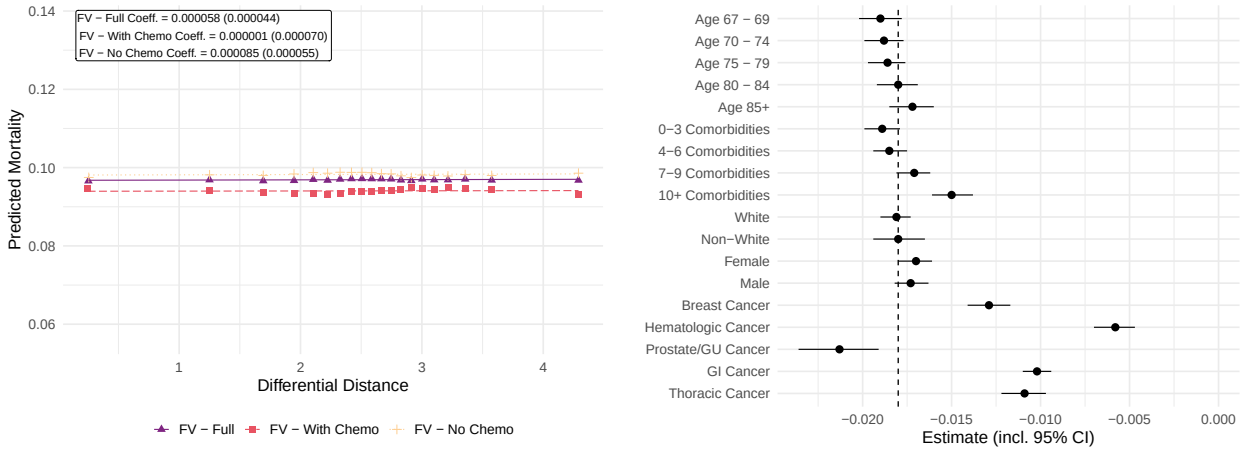
Table A2 reports first-stage estimates for the first-visit sample, separately for the full sample, visits that lead to chemotherapy within 360 days, and visits that do not. Differential distance is a strong predictor of subspecialist access across all samples, with F-statistics well above conventional 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 associated with subspecialist use in encounters that result in 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**
DD	-0.018***	-0.020***	-0.017***
	(0.000)	(0.001)	(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. Due to fixed effect singletons the number of observations in columns 2 and 3 do not perfectly match column 1. 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 two additional diagnostics for the first-visit design. First, Figure A1a examines balance on predicted mortality, a summary measure of baseline health risk constructed from pre-visit characteristics. Predicted mortality differs across the full first-visit sample, the subsample followed by chemotherapy, and the subsample not followed by chemotherapy, reflecting that these groups capture different clinical populations. However, within each sample, differential distance is essentially unrelated to predicted mortality after conditioning on ZCTA and cancer type-by-year fixed effects. The between-sample differences in predicted mortality are larger than the within-sample changes across instrument values. Thus, among first visits followed by chemotherapy, the instrument does not appear to select patients with lower observed baseline mortality risk into treatment. Second, Figure A1b assesses the plausibility of monotonicity by estimating the first-stage relationship across patient subgroups. The coefficients are consistently signed across these subsamples, indicating that greater relative distance to a relevant subspecialist shifts access to subspecialized care in the same direction across observable groups.



(a) Instrument Balance

(b) Instrument Monotonicity

Figure A1: Instrument Balance and Monotonicity in First Visit Sample

Notes: Panel A presents binned scatterplots of yearly predicted mortality against residualized differential distance in the first-visit sample. Results are shown separately for the full first visit sample (FV - Full), first visits followed by chemotherapy initiation within 360 days (FV - With Chemo), and first visits not followed by chemotherapy (FV - No Chemo). Both yearly predicted mortality and differential distance are residualized with respect to ZCTA and cancer type-by-year fixed effects, after which sample-specific means are added back. Points represent ventiles of residualized differential distance, and fitted lines show linear relationships within each sample. Reported coefficients and standard errors are obtained from regressions including ZCTA and cancer type-by-year fixed effects. The y-axis is centered at the sample-specific mean and spans plus and minus one-half of a standard deviation of observation-level residualized yearly predicted mortality. 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 next estimate the effect of a first office visit with a subspecialist using 2SLS, with binary indicators for chemotherapy initiation at 90, 180, and 360 days following a patient's first office visit with an oncologist as the outcome. As shown in Table A3, the estimated effects are statistically insignificant at each horizon. The point estimates are positive but small relative to baseline initiation rates: for any chemotherapy within 360 days, the estimate is 1.4 percentage points on a baseline initiation rate of 32.3 percent. These estimates do not support the simplest favorable-selection story in which subspecialists improve survival in the chemotherapy sample by initiating chemotherapy for lower-risk patients. Alternatively, even if the additional marginal patients had zero potential mortality, the mechanical reduction in treated-sample mortality would be less than one percentage point at one year and about two percentage points at three years, below the main mortality estimates.

The effects on chemotherapy initiation do not rule out more complex forms of selection, such as a greater likelihood of treating patients with favorable unobserved prognos-

tic features while treating fewer frailer patients. However, for these more complex forms of selection, it is helpful to compare OLS and 2SLS estimates in the main chemotherapy sample, reported in Appendix Table E7, which reflect both selection into the chemotherapy sample and selection to subspecialists within that sample. In the main chemotherapy sample, OLS estimates are more favorable than the 2SLS estimate at 180 days but become much less favorable at longer horizons, turning positive at 720 and 1,080 days while the 2SLS estimates remain negative. The pattern does not support a simple story in which selection explains the longer-term mortality benefits of subspecialists.

Table A3: First Office Visits and Probability of Chemotherapy Initiation

	≤ 90	≤ 180	≤ 360
Panel A: Any Chemotherapy			
Subspecialist Visit	0.022	0.013	0.014
	(0.013)	(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			
Subspecialist Visit	0.021	0.011	0.011
	(0.013)	(0.014)	(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 episode cancer type corresponding to the cancer type of the first medical oncology 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 mortality following the initial medical oncology encounter. We construct binary indicators for mortality at 180, 360, 720, and 1,080 days after the first visit. Figure A2 reports 2SLS estimates for four samples: (i) all first office visits from 2008 to 2017 (FV–Full), (ii) first visits followed by chemotherapy initiation within 360 days (FV–With Chemo), (iii) first visits not followed by chemotherapy within 360 days (FV–No Chemo), and (iv) the main sample of first

chemotherapy episodes (Main Chemo). For the first-visit samples, mortality is measured from the date of the first oncology visit; for the main chemotherapy sample, mortality is measured from the date of chemotherapy initiation. As discussed above, the first-visit-with-chemotherapy sample overlaps with, but is not identical to, the main chemotherapy episode sample.

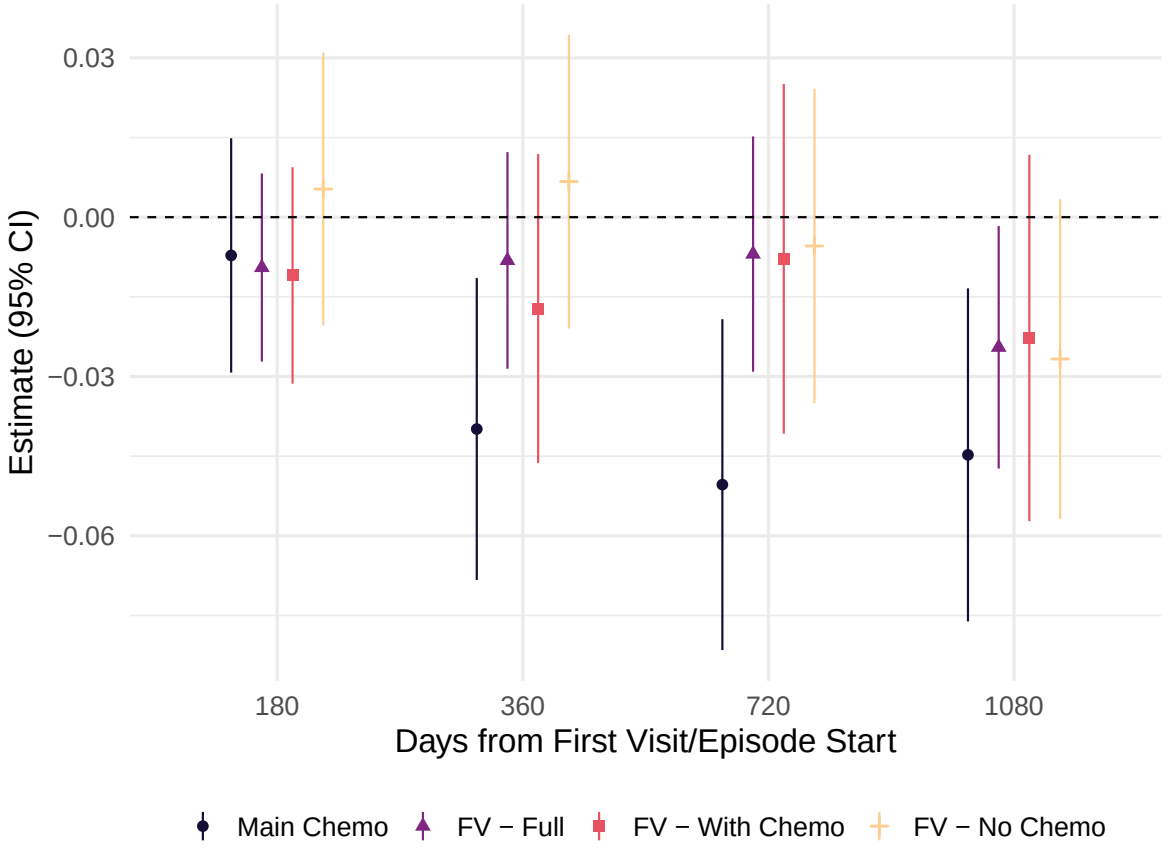


Figure A2: First Office Visit with a Subspecialist and Mortality

Notes: This figure presents 2SLS estimates of the effect of a first office visit with a subspecialized oncologist on mortality. 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, and ZCTA- and cancer type-by-year fixed effects. 95% confidence intervals are based on standard errors clustered at the beneficiary ZCTA level.

The first-visit mortality estimates are imprecise and generally not statistically significant. This is not surprising, because the first-visit sample is less standardized for outcome measurement than the OCM chemotherapy episode sample: the first visit may precede treatment by varying amounts of time, may not lead to systemic therapy, and

may be followed by heterogeneous care pathways. Consistent with this distinction, the largest and most precise mortality reductions appear in the main chemotherapy episode sample, while estimates in the first-visit subsamples are smaller in magnitude and less precise, particularly at shorter horizons. At longer horizons, some first-visit estimates are directionally negative, including among patients who do not initiate chemotherapy. This pattern is not necessarily inconsistent with an effect of subspecialized oncologic care, since patients who do not initiate systemic therapy may still receive surgery, radiation, surveillance, supportive care, palliative care, or additional diagnostic workup, and medical oncologists may influence these pathways through referrals, staging, care coordination, or treatment recommendations. We therefore view Figure A2 as descriptive evidence of mortality patterns across related samples; the more direct test of selection into chemotherapy is Table A3, which examines whether first visits with subspecialists change the probability of subsequent initiation of chemotherapy.

Overall, the first-visit analyses provide little support for the specific selection mechanism in which differential access to subspecialists improves measured survival by shifting lower-risk patients into the chemotherapy episode sample. Table A3 shows small and statistically insignificant effects on chemotherapy initiation, while Figure A1a shows that differential distance is balanced on predicted mortality among first visits followed by chemotherapy. These analyses do not eliminate the possibility of selection on unobserved cancer severity or treatment appropriateness, but they make it less likely that the main mortality estimates are driven by observable baseline risk differences or large changes in entry into chemotherapy.

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 and beneficiary level to enable 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. To better understand the drivers of spending, we also constructed episode-level spending for different RBCS subcategories (CMS, 2024).

Table B1: Episode-Level 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 in 2016-2019 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 by Keating et al. (2021), who evaluate spending in Oncology Care Model (OCM)- participating practices compared with 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 most common cancer types and the use 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 2014 with OCM benchmarks presented in Table 2 of Keating et al. (2021). We observe slightly higher spending in Medicare Parts A and B 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 beneficiary populations and a narrower focus on 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 increases significantly, driven primarily by higher Part B and Part D spending. Part A spending remains almost unchanged throughout the period.

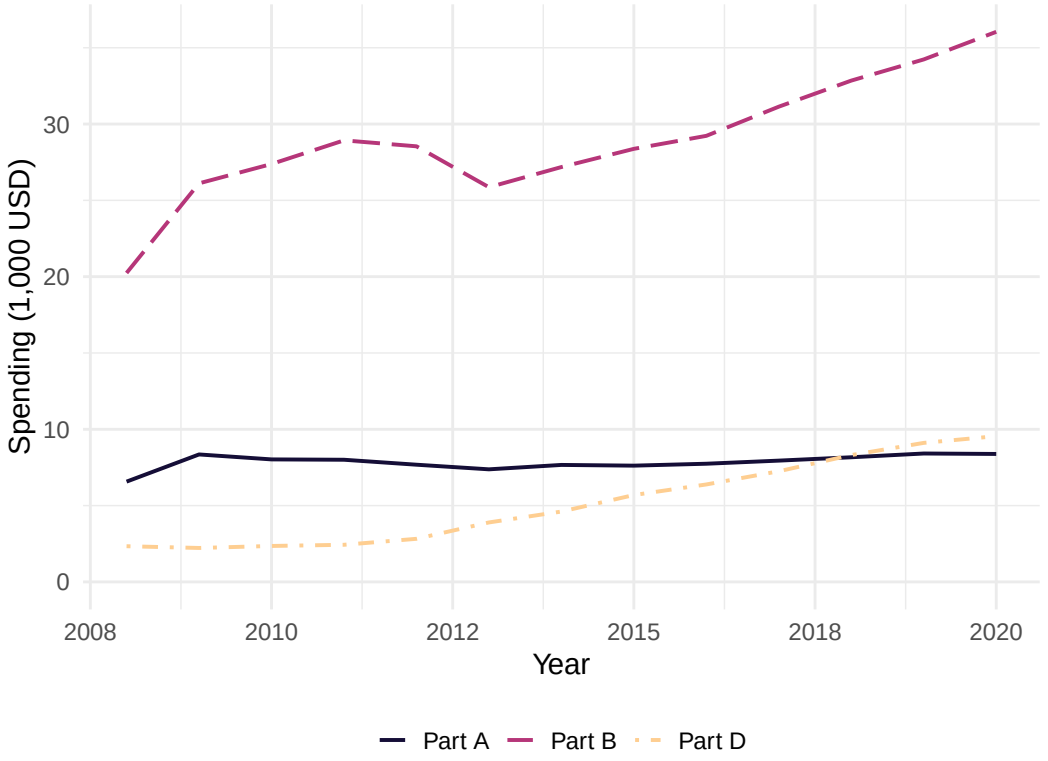


Figure B1: Spending per Episode by Medicare Part and Year

Notes: 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.

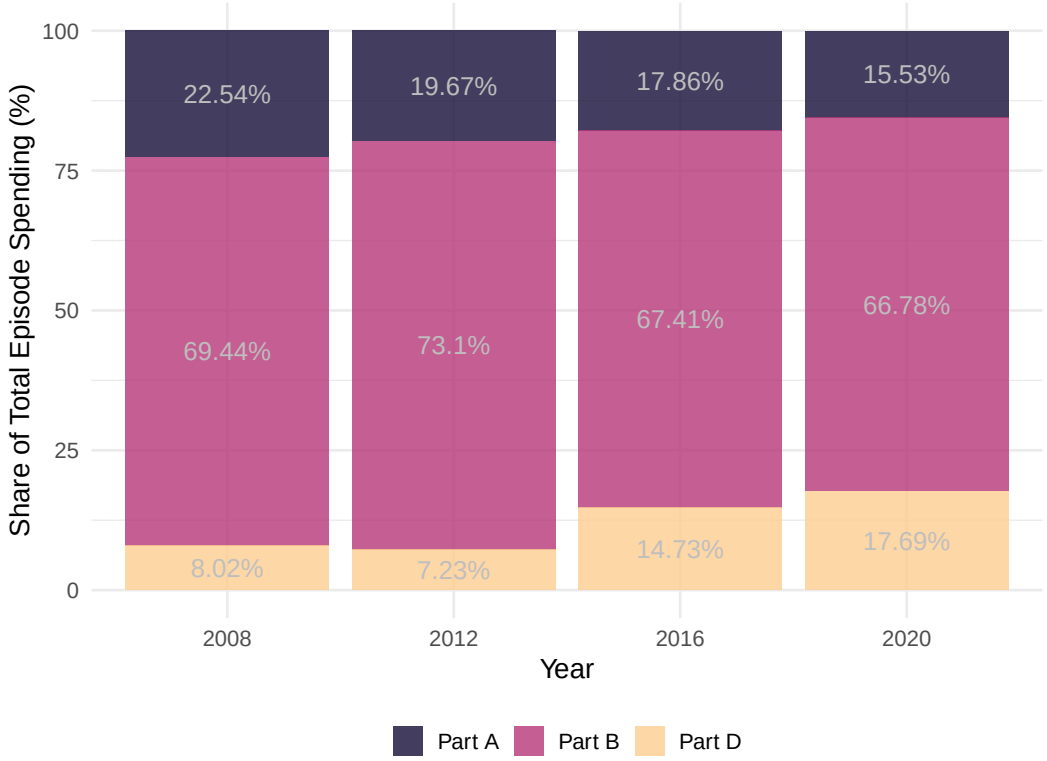


Figure B2: Share of Total Spending by Medicare Part over Time

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

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

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 E1.

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 sufficient specificity to assign it 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 with a corresponding clinical trial number, across both beneficiaries in our chemotherapy sample and clinical trials, is shown in Figure C1. Of the 11,049 classified trials relevant to our sample, 24 percent are related to leukemia

and lymphoma, 15 percent are unrelated to cancer, and 12 percent are related to other cancers (those not covered by our broader categories).

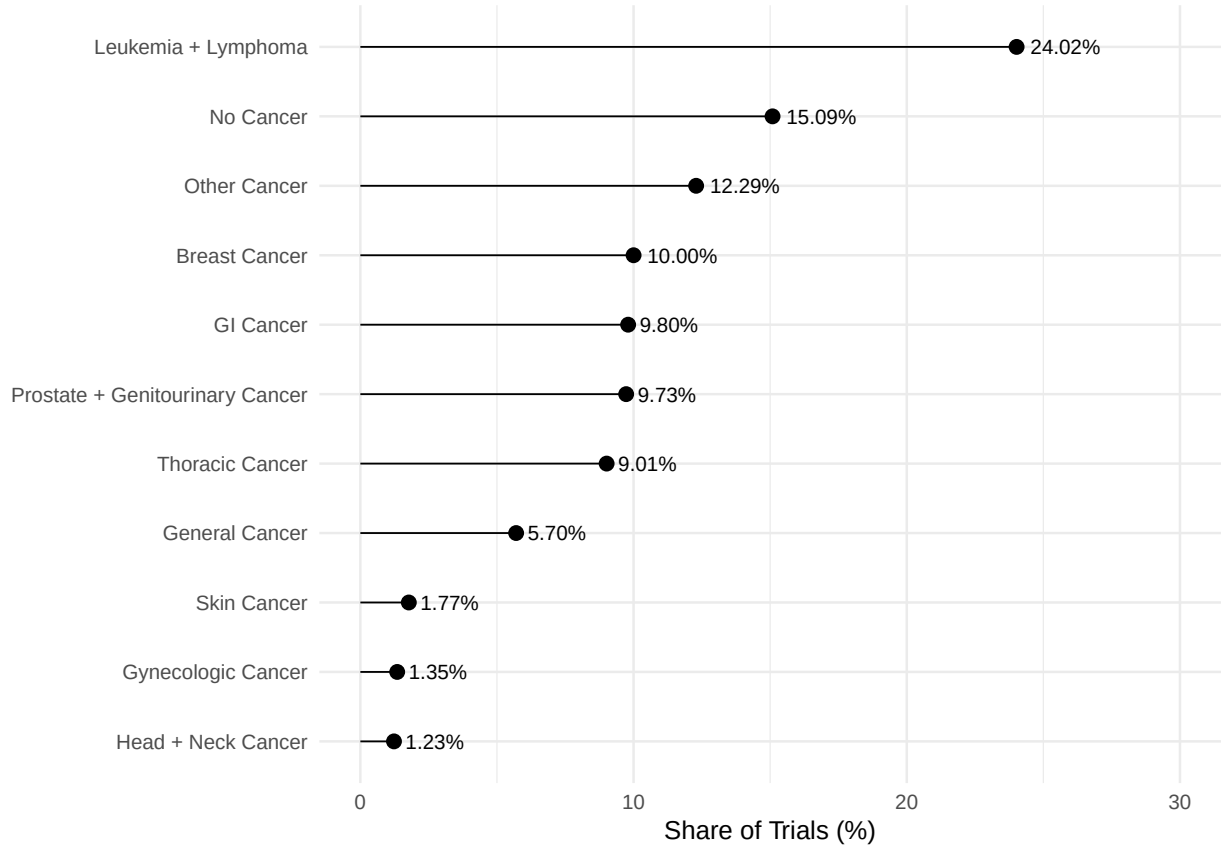


Figure C1: Share of Clinical Trials by Cancer Classification

Notes: This figure presents the share of clinical 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

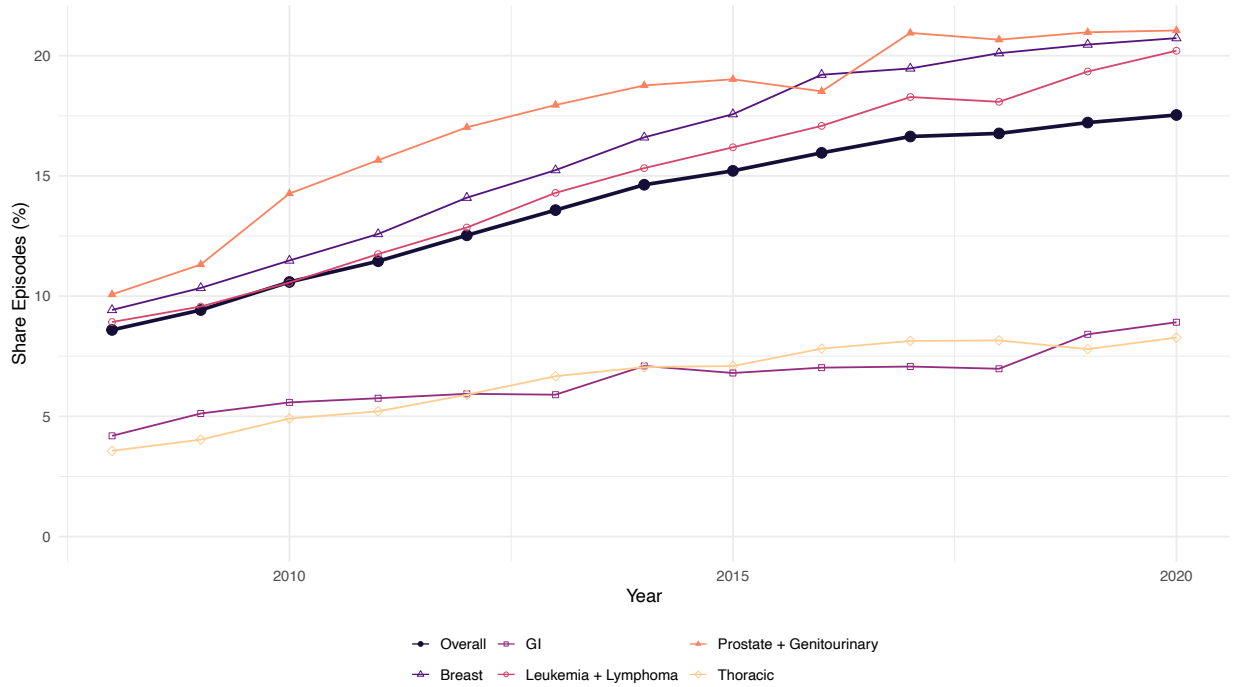


Figure D1: Trends in Subspecialization in Cancer Care, 2008-2020

Notes: This figure presents trends in subspecialization for beneficiary chemotherapy episodes of different cancer types based on results from Karadakic et al. (2025*a*). The figure depicts the share of chemotherapy episodes managed by highly subspecialized oncologists for each relevant cancer type. The "Overall" group (black line with black dots) indicates the trend in subspecialization for all cancer types combined. Abbreviations: GI=gastrointestinal.

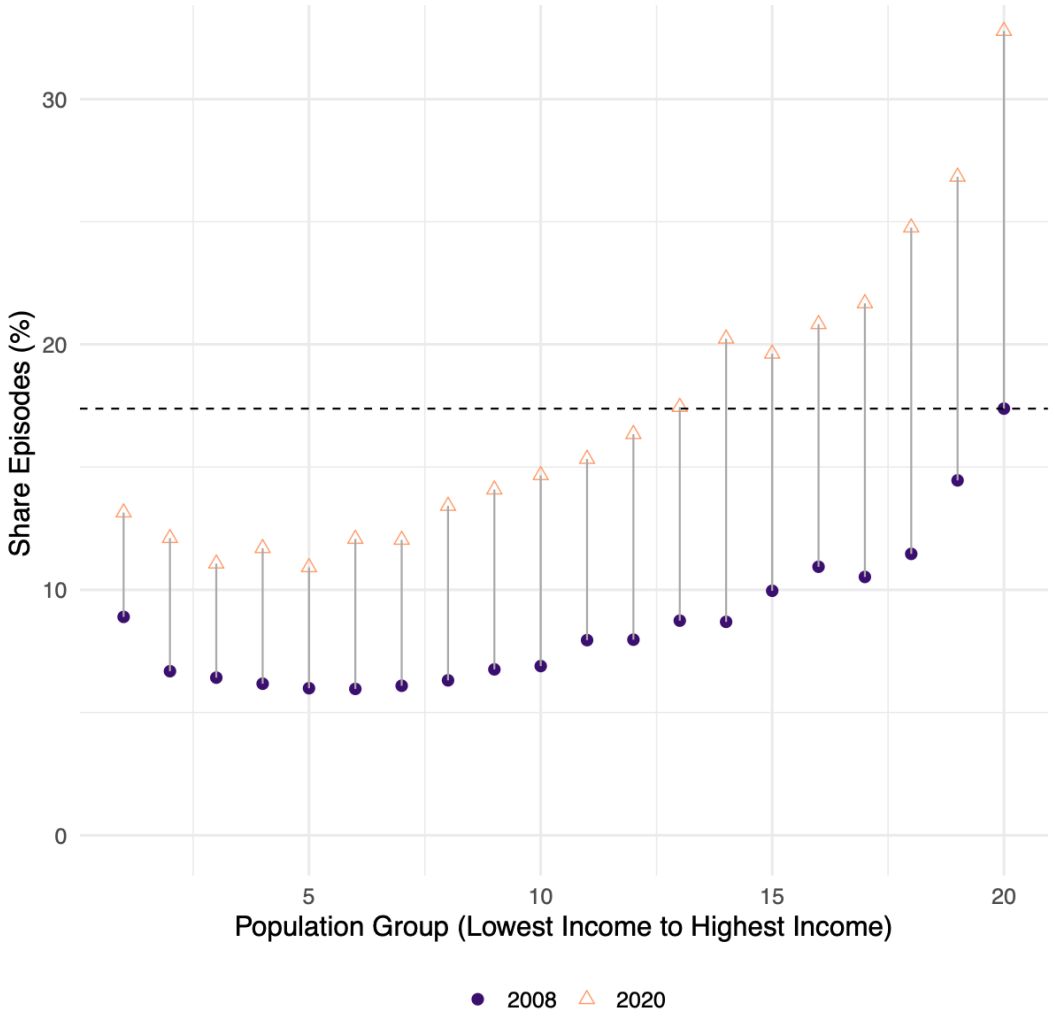
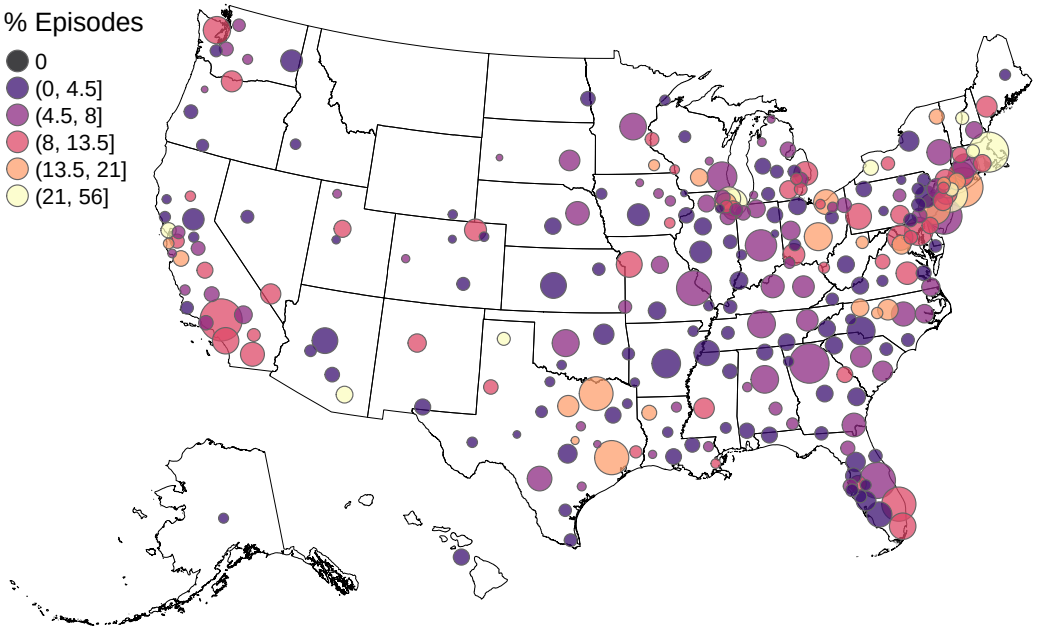
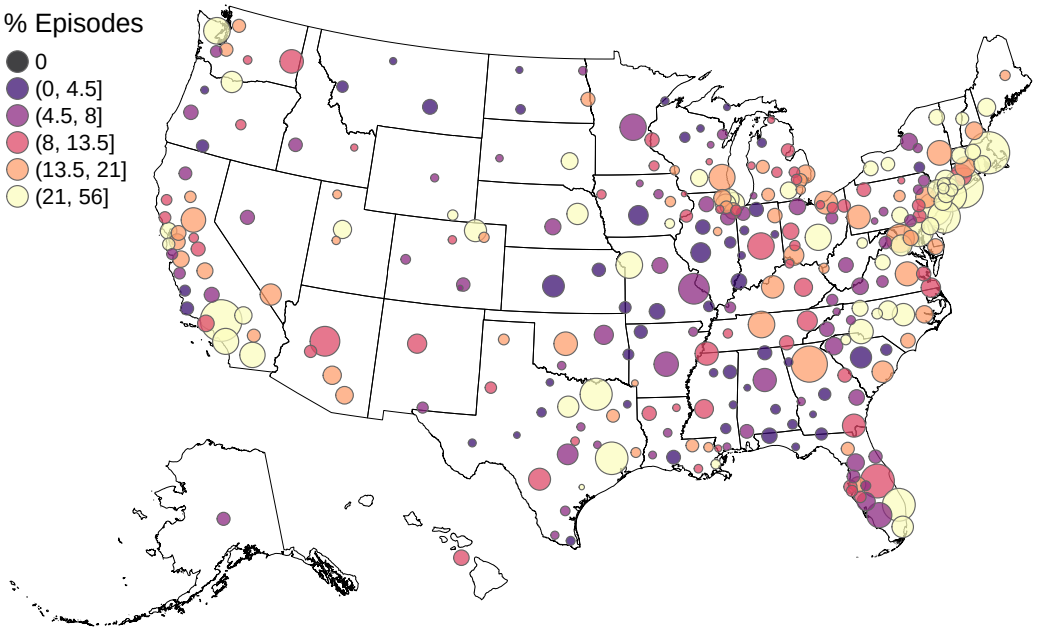


Figure D2: Area-Income Differences in Access to Highly Subspecialized Cancer Care

Notes: This figure illustrates area-income differences 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 areas in the U.S. (Currie and Schwandt, 2016; Schwandt et al., 2021).



(a) 2008



(b) 2020

Figure D3: Utilization of Subspecialized Oncologists by Hospital Referral Region

Notes: 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*a*). 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.

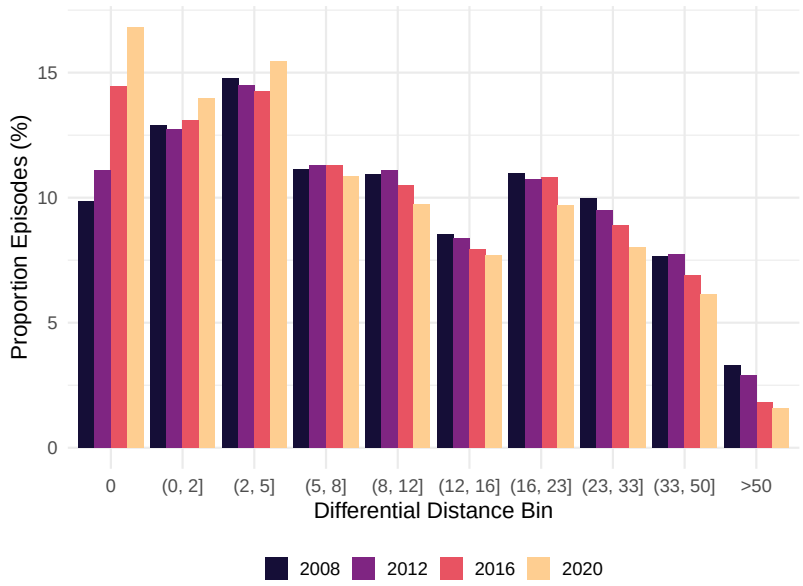


Figure D4: Distribution of Differential Distances over Time

Notes: 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.

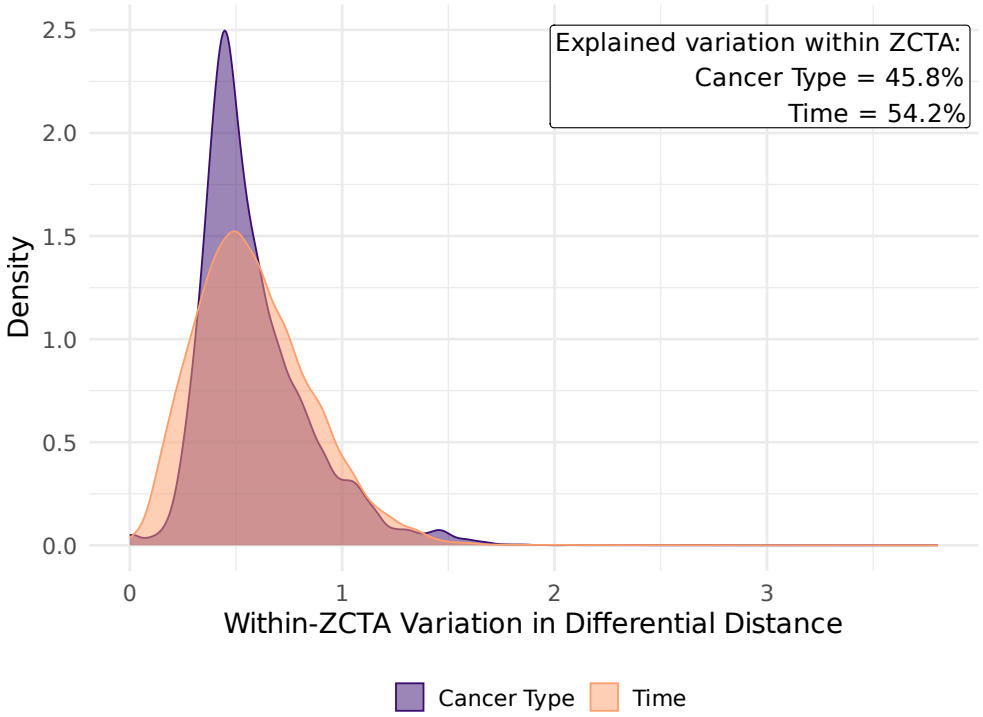


Figure D5: Identifying Variation in Differential Distance

Notes: This figure shows the distribution of within-ZCTA variation in the residualized differential distance measure. The instrument is first residualized with respect to ZCTA and cancer-type-by-year fixed effects. We then construct two measures of variation: (i) the standard deviation across cancer types within each ZCTA-year, capturing differences in access across cancer types within a given location and year, and (ii) the standard deviation over time within each ZCTA-cancer type, capturing changes in access within locations over time. Both measures are averaged at the ZCTA level and summarized using observation weights to reflect the variation in the estimation sample. The two distributions exhibit substantial overlap, indicating that both cross-cancer and temporal variation contribute to identification. A Shapley variance decomposition of the residualized instrument attributes 45.8 percent of the explained variation to cross-cancer differences within ZCTAs and 54.2 percent to temporal variation within ZCTAs.

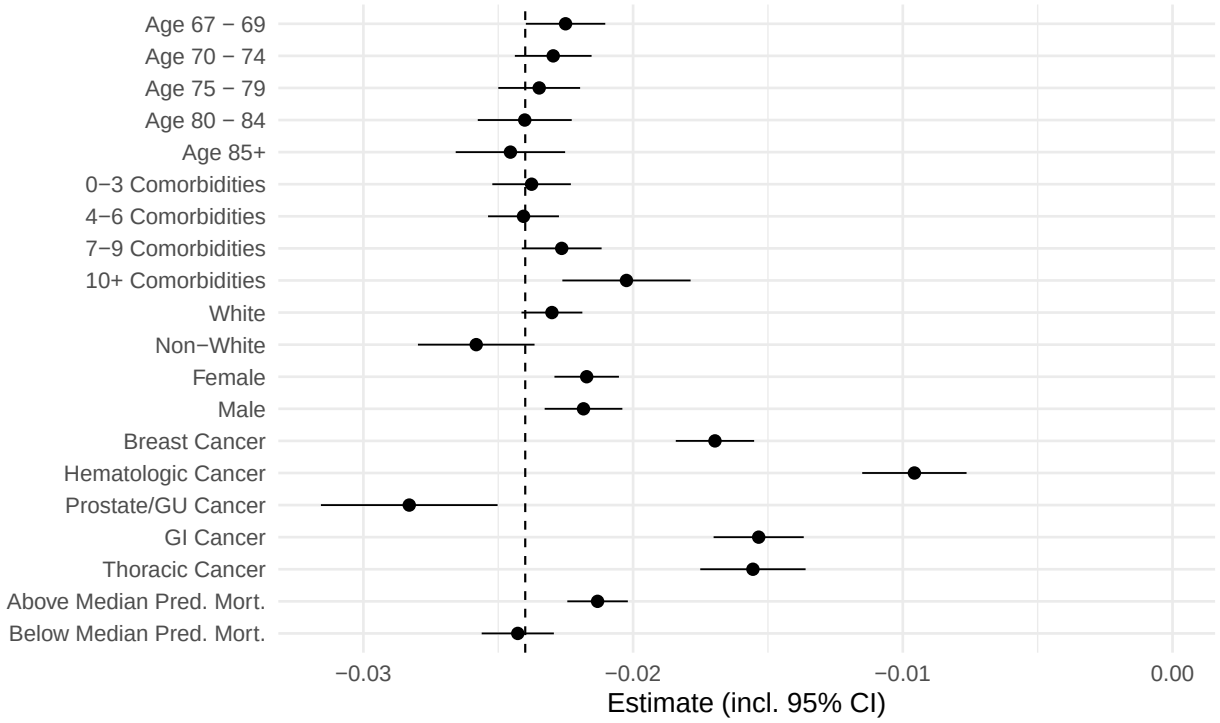


Figure D6: Instrument Monotonicity

Notes: 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.

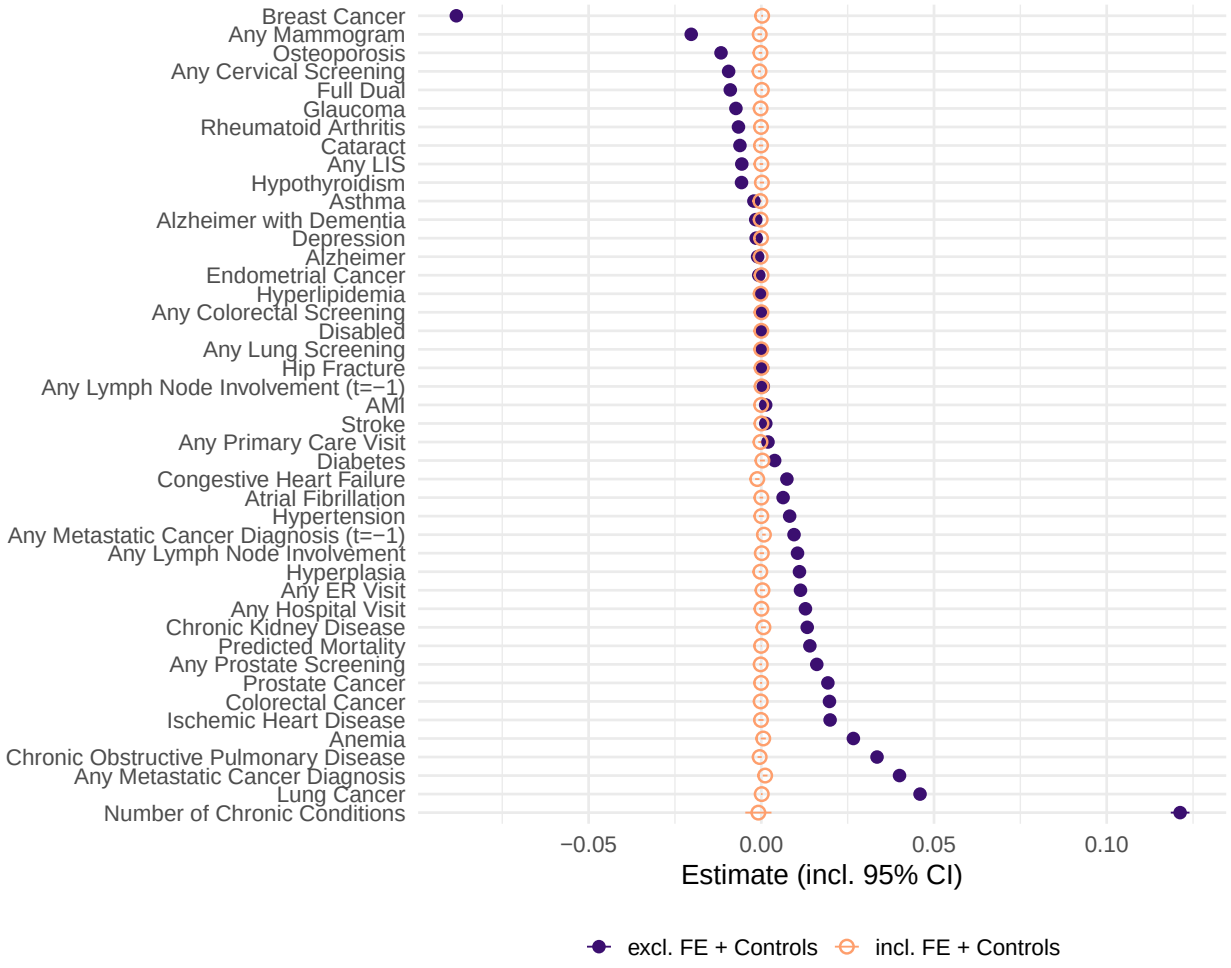


Figure D7: Instrument Balance across Beneficiary Characteristics

Notes: 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.

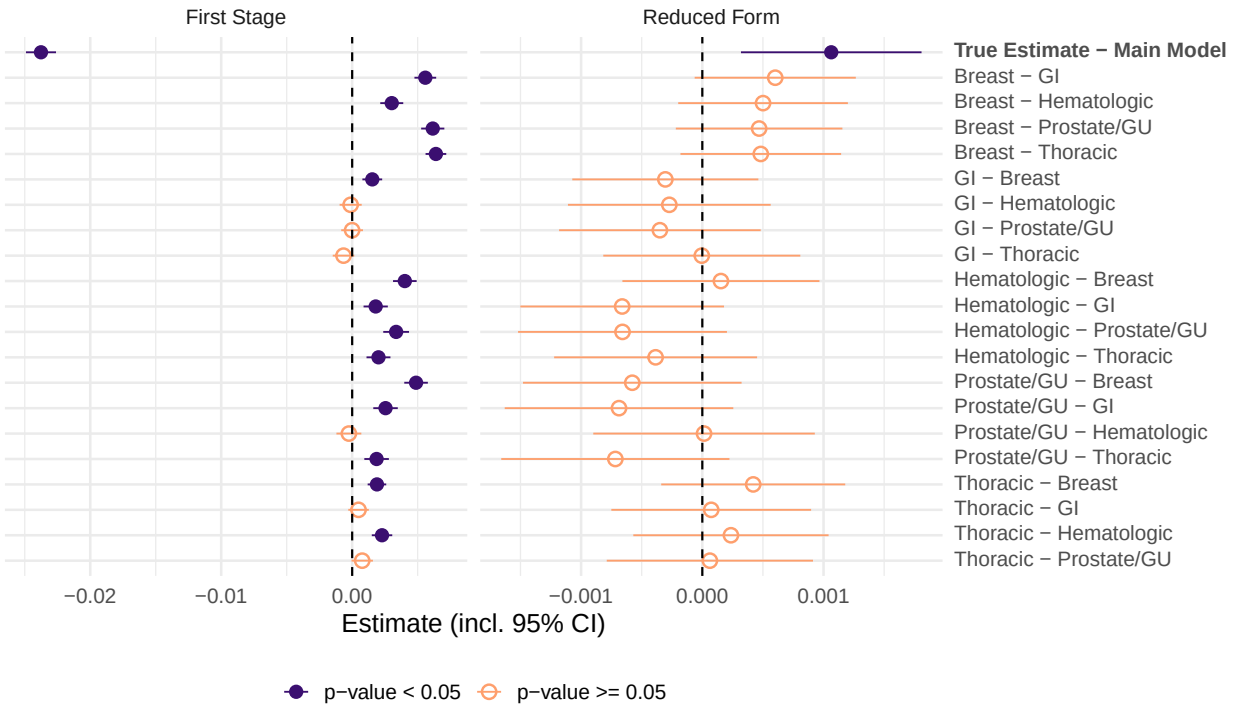


Figure D8: Falsification Test - Assignment of Distance to Unrelated Subspecialist

Notes: This figure presents first-stage and reduced-form 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 based on the true distance to the nearest subspecialist for 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.

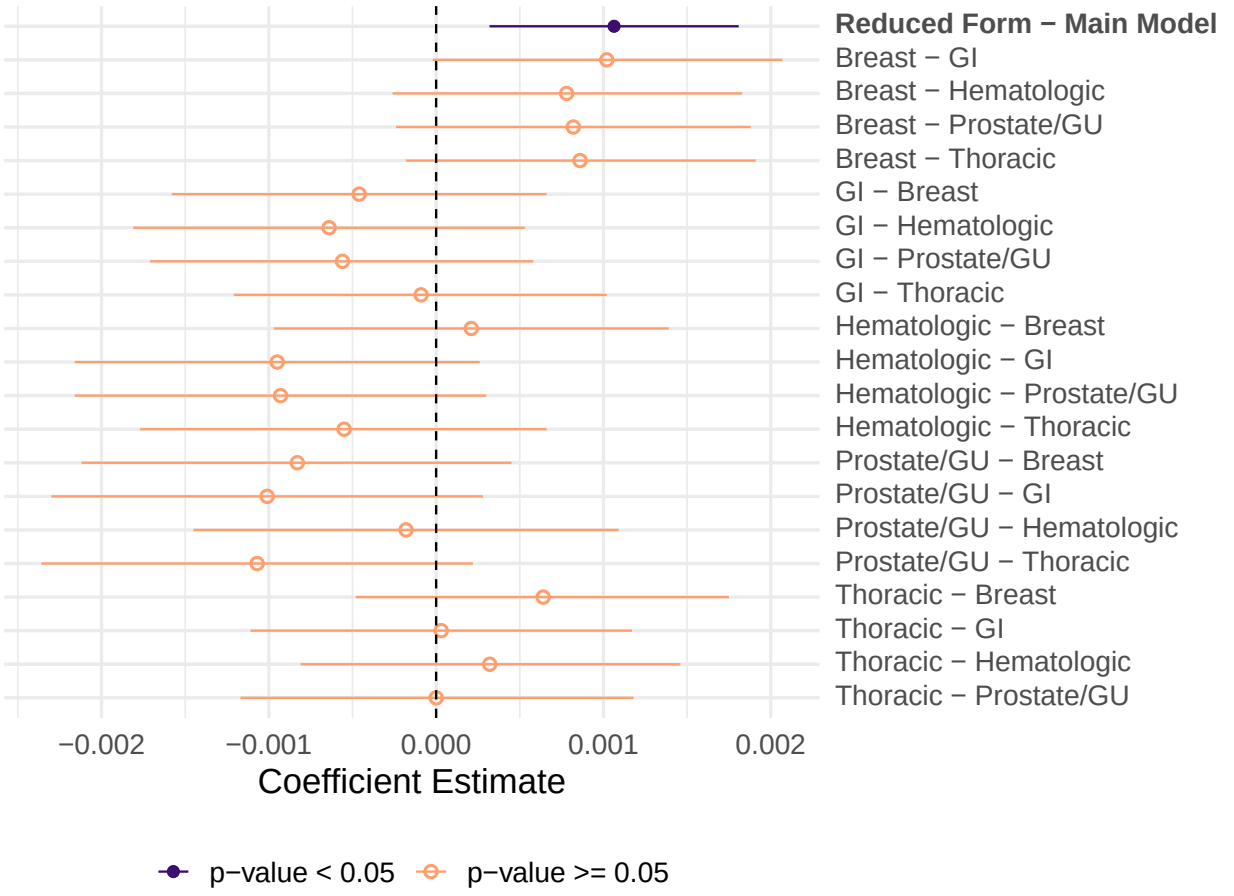
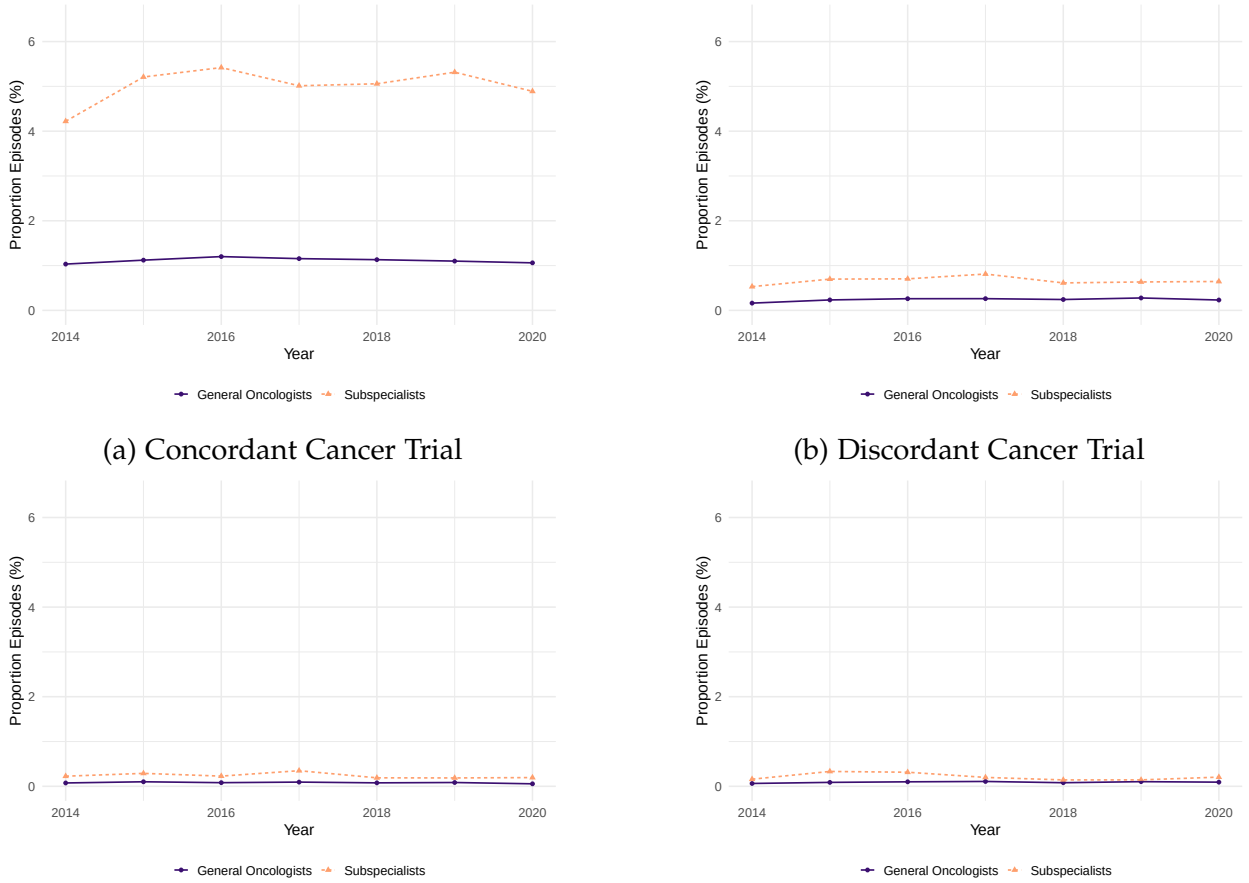


Figure D9: Negative Control Instrument Test

Notes: This figure reports coefficient estimates and 95% confidence intervals from reduced-form regressions of 1,080-day mortality on 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. The coefficient on the placebo instrument is the object of interest and should be zero under a valid exclusion restriction. Each pair of estimates corresponds to a single placebo specification, in which distances are reassigned across cancer types. Each row represents a specific reassignment; for example, "Breast – GI" assigns all beneficiaries the distance to the nearest breast cancer subspecialist, except for breast cancer patients, who are assigned the distance to the nearest GI subspecialist instead. The analysis sample consists of the first chemotherapy episodes from 2008 to 2017. All models control for demographic characteristics, ZCTA-level covariates, and chronic conditions, and include ZCTA 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 D10: Proportion of Episodes with Cancer Trial Enrollment by Trial Type and Oncologist Subspecialization

Notes: 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.

E Additional Tables

Table E1: 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 unspecific 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

Notes: 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 E2: 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 E3: Summary Statistics of Main Sample

Variable	Mean	SD	Min.	Max.
Panel A: Cancer Characteristics				
Breast Cancer	0.330	0.479	0	1
GI Cancer	0.158	0.365	0	1
Hematologic Cancer	0.223	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 E4: Variables for Predicted Mortality Construction

Category	**Variables**
Demographics	Binary indicators for disability status; sex; age categories (0–64, 65–69, 70–74, 75–79, 80–84, 85–89, 90+); race and ethnicity (Black, Hispanic, Asian, Native, other)
Insurance Status	Binary Indicators for Fee-for-service status; dual eligibility (full and partial)
Chronic Conditions	Binary indicators for 27 chronic conditions based on Medicare Chronic Conditions Warehouse (CCW) definitions, including Alzheimer's disease, acute myocardial infarction, anemia, asthma, atrial fibrillation, cancers (breast, colorectal, endometrial, lung, prostate), cataract, congestive heart failure, chronic kidney disease, COPD, depression, diabetes, hyperlipidemia, hypertension, hypothyroidism, ischemic heart disease, osteoporosis, rheumatoid arthritis/osteoarthritis, and stroke/transient ischemic attack
Summary Health Measures	Total number of chronic conditions
Prior Health Care Utilization	Number of unique visits; visits to specialists; visits to primary care physicians; outpatient emergency department visits; inpatient emergency department visits; hospitalizations; intensive care unit stays
Missingness Indicators	Binary indicators for missing values for all covariates, with missing values set to zero

Notes: This table describes the covariates included to construct our predicted morality measure. Predicted mortality is constructed using out-of-sample predictions from year-specific linear probability models of death within the calendar year. For each year, the model is estimated on a training sample of Medicare beneficiaries who are not part of the chemotherapy episode sample and is then applied to individuals in the analysis sample. Covariates are measured prior to treatment and include demographic characteristics, insurance status, indicators for chronic conditions based on Medicare Chronic Conditions Warehouse (CCW) definitions, summary measures of comorbidity burden, and prior-year health care utilization. To account for missing data, we include indicator variables for missingness for all covariates and set missing values to zero. All utilization measures are constructed using claims in the year prior to the observation year.

Table E5: 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 E6: 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 healthcare indicators prior to chemotherapy. Column 1 indicates the variable name, column 2 the mean within the overall sample, column 3 the standard deviation, column 4 the estimate of an unadjusted regression of our instrument on the variable in the respective row (without any controls and without fixed effects), column 5 the corresponding heteroskedasticity robust standard error, column 6 shows estimates from a regression of our instrument on the outcome in the respective row including cancer type by year, as well as ZCTA fixed effects and demographic controls as well as ZCTA level controls, finally column 7 shows the corresponding standard error clustered at the ZCTA level.

Table E7: Mortality Effects of Access to Subspecialized Oncologist

	180-Day	**360-Day**	**720-Day**	**1080-Day**
Panel A: First Stage				
DD	-0.0238***	-0.0238***	-0.0238***	-0.0238***
	(0.0017)	(0.0017)	(0.0017)	(0.0017)
Panel B: Reduced Form				
DD	0.0002	0.0010***	0.0012***	0.0011***
	(0.0003)	(0.0004)	(0.0004)	(0.0004)
Panel C: Structural Form (OLS)				
Any Subspecialist Visit	-0.0142***	-0.0052***	0.0029***	0.0047***
	(0.0006)	(0.0008)	(0.0009)	(0.0009)
Panel D: 2SLS				
Any Subspecialist Visit	-0.0072	-0.0399***	-0.0504***	-0.0448**
	(0.0113)	(0.0145)	(0.0159)	(0.0160)
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 E8: Robustness of Mortality Outcomes by Samples and Specifications

	180-Day	360-Day	720-Day	1080-Day
Panel A: Fixed Sample (2008-2017)				
Any Subspecialist Visit	-0.007	-0.040***	-0.050***	-0.045**
	(0.011)	(0.014)	(0.016)	(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 Subspecialist Visit	-0.001	-0.024*	-0.041***	-0.045***
	(0.010)	(0.031)	(0.015)	(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 Subspecialist Visit	-0.005	-0.046**	-0.058***	-0.050***
	(0.014)	(0.018)	(0.020)	(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 Subspecialist Visit	0.003	-0.032**	-0.048***	-0.039***
	(0.012)	(0.015)	(0.016)	(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	-0.054***	-0.068***	-0.061**
	(0.015)	(0.020)	(0.022)	(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 E9: Mortality Effects - Sources of Identifying Variation

	Baseline	Cancer Type Variation	Time Variation
Panel A: First Stage			
DD	-0.0235***	-0.0275***	-0.0163***
	(0.0005)	(0.0007)	(0.0005)
Panel B: Reduced Form			
DD	0.0012***	0.0010***	0.0006
	(0.0004)	(0.0005)	(0.0005)
Panel C: Structural Form (OLS)			
Any Subspecialist Visit	-0.0313***	-0.0305***	-0.0336***
	(0.0010)	(0.0010)	(0.0011)
Panel D: 2SLS			
Any Subspecialist Visit	-0.0529***	-0.0358**	-0.0354
	(0.0169)	(0.0182)	(0.0305)
Adj R^2	0.251	0.250	0.256
Observations	1,681,119	1,641,871	1,590,985
Mean Dep. Var.	0.449	0.449	0.444
F-Stat (1st Stage)	1,621	1,431	903
ZCTA	Yes	No	No
Cancer Type	No	Yes	No
Year	No	No	Yes
ZCTA × Cancer Type	No	No	Yes
ZCTA × Year	No	Yes	No
Cancer Type × Year	Yes	No	No

Notes: This table reports estimates of the effect of access to subspecialized oncologists on 1,080-day mortality for all first chemotherapy episodes between 2008 and 2017. Panel A shows first-stage estimates. Panel B reports reduced-form estimates. Panel C reports structural-form OLS estimates, and Panel D reports 2SLS estimates. Column 1 (Baseline) presents results for the specification with ZCTA and cancer type × year fixed effects. Column 2 (Cancer Type Variation) presents results primarily relying on within-ZCTA cancer type variation (ZCTA × year and cancer type 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: Access to Subspecialized Oncologist and Spending by Subcategory

	Part A				Part B			
	Inpatient	**HHA**	**Hospice**	**SNF**	**Carrier**	**Outpatient**	**DME**	**Part D**
Panel A: First Stage								
DD	-0.024***	-0.024***	-0.024***	-0.024***	-0.024***	-0.024***	-0.024***	-0.024***
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
Panel B: Reduced Form								
DD	-16.66	-0.93	6.44*	-8.57***	123.42***	-105.64***	-0.80	11.21
	(10.52)	(1.50)	(3.68)	(2.87)	(20.57)	(21.19)	(1.35)	(11.20)
Panel C: Structural Form (OLS)								
Any Subspecialist Visit	1,932.95***	-29.99***	-69.27***	-191.17***	-4,207.06***	6,900.43***	33.21***	444.74***
	(33.258)	(3.62)	(5.58)	(6.86)	(57.33)	(74.64)	(3.53)	(32.71)
Panel D: 2SLS								
Any Subspecialist Visit	704.42	39.30	-272.21*	362.27***	-5,217.32***	4,465.74***	33.92	-474.06
	(444.37)	(63.55)	(155.94)	(121.50)	(874.14)	(899.49)	(57.14)	(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 E11: Access to Subspecialized Oncologist and Spending on Chemotherapy Drugs and Services

	Chemotherapy Drugs	**Injections & Infusions (RI)**
Panel A: First Stage		
DD	-0.024***	-0.024***
	(0.001)	(0.001)
Panel B: Reduced Form		
DD	60.74***	15.51***
	(13.88)	(5.835)
Panel C: Structural Form (OLS)		
Any Subspecialist Visit	-1,023.72***	-458.82***
	(37.01)	(14.24)
Panel D: 2SLS		
Any Subspecialist Visit	-2,567.73***	-655.78***
	(587.17)	(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 E12: Complier Characteristics Overview

	Complier			Complier/Sample	
Variable	**Mean**	**SE**	**Sample Mean**	**Ratio**	**SE (Ratio)**
Panel A: Chemotherapy Episode Cancer Type					
Breast Cancer	0.346	0.015	0.330	1.049	0.046
GI Cancer	0.147	0.006	0.158	0.931	0.039
Hematologic Cancer	0.137	0.009	0.223	0.617	0.041
Prostate/GU Cancer	0.211	0.008	0.127	1.659	0.066
Thoracic Cancer	0.159	0.007	0.163	0.976	0.043
Panel B: Other Characteristics					
Age	75.219	0.102	75.175	1.001	0.001
Female	0.577	0.012	0.585	0.986	0.020
Black	0.090	0.006	0.080	1.134	0.068
Urban ZCTA	0.923	0.007	0.814	1.133	0.009
Predicted Mortality	0.088	0.002	0.098	0.898	0.022
Nr. Chronic Cond.	5.029	0.049	5.095	0.987	0.010
Panel C: Chronic Condition Indicators					
Alzheimer	0.022	0.002	0.023	0.974	0.076
Alzheimer Dementia	0.074	0.004	0.082	0.905	0.044
AMI	0.012	0.002	0.014	0.885	0.106
Anemia	0.479	0.008	0.559	0.857	0.015
Asthma	0.071	0.004	0.069	1.025	0.055
Atrial Fibrillation	0.123	0.005	0.129	0.953	0.036
Cancer Breast	0.366	0.015	0.355	1.030	0.041
Cancer Colorectal	0.076	0.004	0.102	0.744	0.035
Cancer Endometrial	0.008	0.001	0.008	1.013	0.181
Cancer Lung	0.175	0.007	0.188	0.929	0.038
Cancer Prostate	0.193	0.008	0.135	1.428	0.056
Cataract	0.218	0.006	0.206	1.055	0.030
CHF	0.193	0.006	0.209	0.923	0.028
CKD	0.296	0.008	0.314	0.943	0.024
COPD	0.208	0.006	0.231	0.898	0.026
Depression	0.196	0.006	0.196	0.999	0.031
Diabetes	0.292	0.007	0.315	0.926	0.022
Hyperplasia	0.111	0.005	0.112	0.990	0.047
Glaucoma	0.113	0.005	0.104	1.089	0.045
Hip Fracture	0.009	0.001	0.012	0.748	0.119
Hyperlipidemia	0.572	0.008	0.570	1.004	0.013
Hypertension	0.709	0.007	0.726	0.977	0.010
Hypothyroidism	0.199	0.006	0.188	1.060	0.032
Ischemic Heart Disease	0.366	0.008	0.395	0.926	0.020
Osteoporosis	0.108	0.005	0.107	1.005	0.050
Rheumatoid Arthritis	0.359	0.007	0.372	0.964	0.019
Stroke	0.040	0.003	0.047	0.833	0.061

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

Table E13: Subspecialist Access and Use of Newly FDA Approved Drugs

	Any	**Part B**	**Part D**
Panel A: First Stage			
DD	-0.02366***	-0.02366***	-0.02366***
	(0.00055)	(0.00055)	(0.00055)
Panel B: Reduced Form			
DD	-0.00004*	-0.00017	-0.00017
	(0.00002)	(0.00013)	(0.00010)
Panel C: Structural Form (OLS)			
Any Subspecialist Visit	0.00034***	0.00500***	0.00538***
	(0.00006)	(0.00030)	(0.00028)
Panel D: 2SLS			
Any Subspecialist Visit	0.00155*	0.00728	0.00700*
	(0.00085)	(0.00529)	(0.00423)
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 E14: Access to Subspecialized Oncologist and Provider Mix

	Nr. Visits	**Unique Providers**	**Unique Specialties**
Panel A: First Stage			
DD	-0.02366***	-0.02366***	-0.02366***
	(0.00055)	(0.00055)	(0.00055)
Panel B: Reduced Form			
DD	-0.00223	0.00460***	0.00007
	(0.00641)	(0.00196)	(0.00152)
Panel C: Structural Form (OLS)			
Any Subspecialist Visit	0.78900***	0.66602***	0.41672***
	(0.01811)	(0.00566)	(0.00395)
Panel D: 2SLS			
Any Subspecialist Visit	0.09417	-0.19446***	-0.00285
	(0.27097)	(0.08318)	(0.06409)
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.

Table E15: Oncologist Specialization and Mortality - Broad Cancer Types

	180-Day	360-Day	720-Day	1080-Day
Panel A: First Stage				
DD	-0.01041***	-0.01041***	-0.01041***	-0.01041***
	(0.00032)	(0.00032)	(0.00032)	(0.00032)
Panel B: Reduced Form				
DD	0.00017	0.00095***	0.00120***	0.00106***
	(0.00027)	(0.00035)	(0.00038)	(0.00038)
Panel C: Structural Form (OLS)				
Cancer Type Share	-0.03628***	-0.05010***	-0.05365***	-0.05404***
	(0.00093)	(0.00123)	(0.00139)	(0.00142)
Panel D: 2SLS				
Cancer Type Share	-0.01645	-0.09110***	-0.11501***	-0.10221***
	(0.02579)	(0.03303)	(0.03621)	(0.03648)
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)	1,054	1,054	1,054	1,054

Notes: This table reports estimates of the effect of cancer type–specific subspecialization on mortality for all first chemotherapy episodes between 2008 and 2017. Subspecialization is measured as the share of the treating oncologist's chemotherapy episodes accounted for by the patient's relevant cancer group (breast, gastrointestinal, hematologic, prostate/genitourinary or thoracic cancer). This measure is constructed using a leave-one-out approach that excludes the index episode to avoid mechanical bias. 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 E16: Oncologist Specialization and Mortality - Detailed Cancer Types

	180-Day	360-Day	720-Day	1080-Day
Panel A: First Stage				
DD	-0.00707***	-0.00707***	-0.00707***	-0.00707***
	(0.00026)	(0.00026)	(0.00026)	(0.00026)
Panel B: Reduced Form				
DD	0.00017	0.00095***	0.00120***	0.00106***
	(0.00027)	(0.00035)	(0.00038)	(0.00026)
Panel C: Structural Form (OLS)				
Cancer Type Share	-0.04055***	-0.06383***	-0.07463***	-0.07866***
	(0.00106)	(0.00142)	(0.00163)	(0.00171)
Panel D: 2SLS				
Cancer Type Share	-0.02422	-0.13414***	-0.16934***	-0.15050***
	(0.03785)	(0.04879)	(0.05344)	(0.05375)
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 cancer type–specific subspecialization on mortality for all first chemotherapy episodes between 2008 and 2017. Subspecialization is measured as the share of the treating oncologist's chemotherapy episodes accounted for by the patient's relevant detailed cancer type. Detailed cancer types are presented in Appendix Table E1. This measure is constructed using a leave-one-out approach that excludes the index episode to avoid mechanical bias. 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.