

Article

Health Returns to Access Expansion: Evidence on the Role of Delivery Capacity Across U.S. States

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Abstract

Although expansions in healthcare coverage and affordability are widely expected to improve population health, their effects on health outcomes are conditional rather than automatic. Improved access increases the opportunity for care use, but measurable health gains depend on whether delivery systems can provide timely and effective treatment and whether the outcomes under study are responsive to clinical intervention. Using a balanced panel of U.S. state–year data from 2006 to 2020, this study examines how healthcare access interacts with delivery capacity to shape cause-specific mortality. Effective access to care is measured as a multidimensional construct encompassing coverage, affordability, and connection to care, while delivery capacity is proxied by lagged healthcare workforce density. The results indicate that gains in access are associated with reductions in amenable mortality primarily in higher-capacity settings. In contrast, mortality outcomes less directly responsive to clinical care exhibit weaker or different patterns. Instrumental-variable estimates are consistent with these findings, although the instruments fall short of conventional strength thresholds and are interpreted using weak-instrument-robust methods. Overall, the findings suggest that the health returns to access expansion are conditional on delivery capacity: they are more likely to materialize where healthcare systems can translate formal access into timely and effective clinical care and where outcomes are within the reach of medical intervention.

Keywords: healthcare access; delivery capacity; amenable mortality; health production; healthcare workforce; cause-specific mortality

JEL Classification: I12; I18; C23; C36



Academic Editor: Castiel Chen
Zhuang

Received: 24 April 2026

Revised: 1 July 2026

Accepted: 10 July 2026

Published: 15 July 2026

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1. Introduction

Expanding access to healthcare remains a central objective of contemporary health policy. Reforms that broaden insurance coverage, reduce out-of-pocket costs, and strengthen individuals' linkages to care are typically motivated by their potential to increase the utilization of appropriate services, alleviate financial barriers to treatment, and ultimately improve population health. While a substantial body of evidence documents positive effects on coverage, service use, and financial protection, findings on downstream health outcomes have been more mixed and frequently context-dependent ([Buchmueller et al., 2024](#); [Miller et al., 2021](#); [Courtemanche et al., 2018](#); [Baicker et al., 2013](#)).

From a health economics perspective, this raises a central question about the realized returns to access-oriented policy: under what conditions do improvements in access generate measurable health gains? Expanding coverage or affordability changes the demand side

of healthcare by increasing the ability and incentive to seek care. Health improvements, however, depend on whether delivery systems can convert that increased demand into timely diagnosis, treatment, and disease management. In this sense, the effects of access expansion are not determined by formal coverage alone. They also depend on whether the outcomes under study are plausibly responsive to medical care and whether the healthcare system has sufficient productive capacity to deliver care effectively once access improves. Evidence from the Oregon Health Insurance Experiment, subsequent Affordable Care Act studies, and more recent mortality research points in this direction: insurance expansions reliably improve access and financial protection, but their health consequences are mediated by the settings and mechanisms through which care is delivered (Sommers et al., 2012; Baicker et al., 2013; Courtemanche et al., 2018; Levy & Buchmueller, 2025; Miller et al., 2021; Tadesse & Dorose, 2026).

Two related sources of heterogeneity follow from this argument. First, the expected health returns from improved access should differ across outcomes, depending on their responsiveness to clinical care. For conditions in which timely diagnosis and effective treatment are especially consequential, expanded access is more likely to improve health outcomes. In contrast, outcomes shaped more heavily by prevention failures, environmental risks, violence, or other non-clinical determinants are less directly responsive to improved access alone, reflecting the broader role of social, behavioral, and environmental determinants in population health (McGinnis et al., 2002; Woolf & Braveman, 2011). The literature on amenable mortality captures this distinction by identifying deaths that are especially sensitive to timely and effective healthcare, and for that reason, useful for evaluating healthcare system performance (Nolte & McKee, 2004, 2012; Barber et al., 2017).

Second, even for clinically responsive outcomes, the health effects of improved access depend on delivery capacity. Access expansion can increase the demand for physician visits, diagnostics, follow-up care, and treatment, but those gains are less likely to produce measurable health improvements when provider supply, workforce availability, or system throughput are inadequate. In economic terms, the marginal return to access expansion depends on the availability of complementary inputs within the delivery system. Where those complementary inputs are stronger, improved access is more likely to yield realized health gains. Where they are weaker, access reforms may still improve insurance coverage or affordability while generating smaller effects on mortality or morbidity. This broader distinction between formal access and the effective delivery of high-quality care is consistent with international evidence showing that poor health outcomes often reflect not only a lack of contact with the healthcare system, but also failures in the quality and timeliness of care delivered (Kruk & Pate, 2020).

The United States offers a particularly informative setting for examining these issues. Despite repeated federal and state efforts to improve access to care, substantial interstate variation persists in affordability, connection to care, provider availability, workforce capacity, and cause-specific mortality (Woolf & Schoomaker, 2019). State-level differences in access and delivery capacity are important because they imply that a similar improvement in insurance coverage or affordability may not generate the same health return everywhere. States with stronger clinical labor supply, better care attachment, and greater system throughput may be more able to translate improved access into timely treatment, while lower-capacity states may experience smaller mortality effects even when formal access improves. At the same time, states operate within a common national environment characterized by broadly shared technologies, federal institutions, and regulatory structures. This combination of a shared institutional background and meaningful subnational variation provides a useful setting for studying how differences in access and delivery capacity are associated with differences in mortality outcomes.

This study is motivated by the gap between the policy expectation that access expansion should improve health and the empirical reality that health effects are often heterogeneous. U.S. access-oriented reforms have improved coverage and affordability, yet interstate differences in healthcare workforce density, access to usual sources of care, cost-related barriers, and cause-specific mortality remain substantial. These differences raise a policy-relevant question: whether expanding access yields larger mortality gains in settings where delivery systems have sufficient capacity to meet the additional demand for care.¹

Using U.S. state-year data from 2006 to 2020, we examine whether the health effects of improved access depend on delivery capacity and whether those effects differ across mortality outcomes that vary in their responsiveness to clinical care. Amenable mortality is the primary outcome because it captures deaths for which timely and effective medical care is especially consequential. Mortality outcomes that are less directly responsive to routine clinical interventions provide policy-relevant contrasts. The design makes it possible to assess not simply whether improved access is associated with lower mortality, but whether the gains are concentrated where healthcare delivery should matter most and where delivery systems are better able to convert formal access into realized care.

The results indicate that improvements in effective access to care are associated with reductions in amenable mortality, with the largest declines occurring in higher-capacity settings. Mortality outcomes less responsive to clinical care do not exhibit comparable capacity-conditioned patterns. These findings suggest that the health effects of access expansion are conditional rather than automatic. Policies that improve coverage and affordability can generate measurable health gains, but those gains depend on whether delivery systems have sufficient workforce and operational capacity to provide timely, clinically effective care.

The study contributes to the health economics literature on access, health production, and policy effectiveness in three ways. First, it helps explain why access-oriented reforms may improve intermediate outcomes such as coverage, utilization, and financial protection more consistently than final health outcomes. Second, it shows that the health payoff to improved access depends on complementary delivery capacity, so that the marginal return to access expansion is higher where healthcare systems can absorb and treat additional demand effectively. Third, it clarifies the conditions under which an access-oriented policy is most likely to generate larger health returns, thereby informing the efficient design and evaluation of healthcare policy.

The remainder of the paper proceeds as follows. Section 2 develops the conceptual framework. Section 3 describes the data and measurement strategy. Section 4 outlines the empirical approach. Section 5 reports the results. Section 6 discusses the findings in relation to the existing literature and policy implications, and Section 7 presents the conclusions.

2. Conceptual Framework

We frame healthcare access within a health-production framework in which health outcomes depend on both clinically delivered care and social, behavioral, environmental, and other non-clinical determinants of health (Grossman, 1972). Policies that expand insurance coverage, reduce affordability barriers, or improve individuals' access to care increase the likelihood of using medical services. However, improved access produces measurable health gains only if the healthcare delivery system can translate that access into timely and effective diagnosis, treatment, and disease management.

This framework implies that the health return to access expansion is conditional in two ways. First, it depends on the type of health outcome being studied. Outcomes that are highly responsive to timely clinical intervention should be more affected by improved access than outcomes shaped mainly by social, behavioral, environmental, or other non-clinical risks. Second, it depends on delivery capacity. Even when access improves, the realized health effect may be limited if provider availability, staffing, or system throughput is insufficient to absorb the additional demand for care.

Let M_{jst} denote mortality in the outcome category j , state s , and year t . Mortality is assumed to depend on clinically delivered care, C_{st} , and mortality-increasing non-clinical determinants, N_{st} . The variable N_{st} is defined so that higher values correspond to less favorable social, behavioral, environmental, or policy conditions. Thus,

$$M_{jst} = M_j(C_{st}, N_{st}),$$

with

$$\frac{\partial M_j}{\partial C} < 0, \quad \frac{\partial M_j}{\partial N} > 0.$$

The first inequality indicates that more clinically delivered care reduces mortality, whereas the second indicates that mortality rises when non-clinical risks become more severe. A linear approximation to the mortality-production relationship can be written as:

$$M_{jst} = \alpha_j - \theta_j C_{st} + \lambda_j N_{st},$$

where $\theta_j > 0$ measures the responsiveness of the mortality category j to clinically delivered care, and $\lambda_j > 0$ captures the influence of non-clinical determinants on mortality. The magnitude of θ_j differs across mortality categories based on how much timely diagnosis, treatment, and clinical management can affect mortality risk. For amenable mortality, which includes causes of death that effective healthcare can plausibly prevent or delay, θ_j is expected to be relatively large. For preventable mortality categories shaped more heavily by injuries, violence, environmental conditions, behavioral risks, or other upstream factors, θ_j is expected to be smaller.

Clinically delivered care is itself produced by healthcare access and delivery capacity. Let A_{st} denote effective access to care and K_{st} denote delivery capacity. Access captures the ability to seek and obtain care through coverage, affordability, and attachment to a usual source of care. Capacity captures the supply-side ability of the healthcare system to convert potential demand into realized care. The production of clinically delivered care can therefore be represented as:

$$C_{st} = C(A_{st}, K_{st}),$$

with

$$\frac{\partial C}{\partial A} > 0, \quad \frac{\partial C}{\partial K} > 0, \quad \frac{\partial^2 C}{\partial A \partial K} > 0.$$

The first condition states that better access increases the potential for receiving care. The second states that greater delivery capacity increases the amount of care that can be provided. The third captures complementarity between access and capacity: the marginal effect of access on realized clinical care is larger when the delivery system has greater capacity. This provides the theoretical basis for including an interaction between access and delivery capacity in the empirical model.

A convenient linear approximation to the clinical-care production function is

$$C_{st} = \delta_0 + \delta_A A_{st} + \delta_K K_{st} + \delta_{AK} A_{st} K_{st},$$

where

$$\delta_A > 0, \quad \delta_K > 0, \quad \delta_{AK} > 0.$$

Substituting this expression into the mortality equation gives

$$M_{jst} = \alpha_j - \theta_j(\delta_0 + \delta_A A_{st} + \delta_K K_{st} + \delta_{AK} A_{st} K_{st}) + \lambda_j N_{st}.$$

Expanding and rearranging yields

$$M_{jst} = \mu_j - \theta_j \delta_A A_{st} - \theta_j \delta_K K_{st} - \theta_j \delta_{AK} A_{st} K_{st} + \lambda_j N_{st},$$

where

$$\mu_j = \alpha_j - \theta_j \delta_0.$$

The marginal effect of access on mortality is therefore:

$$\frac{\partial M_{jst}}{\partial A_{st}} = -\theta_j \delta_A - \theta_j \delta_{AK} K_{st}.$$

The marginal effect of delivery capacity is

$$\frac{\partial M_{jst}}{\partial K_{st}} = -\theta_j \delta_K - \theta_j \delta_{AK} A_{st}.$$

The cross-partial derivative is

$$\frac{\partial^2 M_{jst}}{\partial A_{st} \partial K_{st}} = -\theta_j \delta_{AK}.$$

Since $\theta_j > 0$ and $\delta_{AK} > 0$, the cross-partial derivative is negative, implying that greater delivery capacity strengthens the mortality-reducing effect of improved access. The magnitude of this complementarity depends on θ_j . It should be stronger for outcomes such as amenable mortality, where timely and effective healthcare is more likely to affect survival, and weaker for outcomes driven mainly by non-clinical risks.

The framework generates three empirical implications. First, the relationship between access and mortality should become more favorable as delivery capacity increases. Second, this capacity-conditioned relationship should be stronger for mortality outcomes that are more responsive to clinical care. Third, outcomes less directly affected by routine healthcare delivery should not exhibit the same access-capacity pattern. These implications motivate the empirical specification used below, which estimates the relationship between access, delivery capacity, and their interaction separately across mortality categories.

3. Data and Measurement

The analysis used an annual balanced panel of all 50 U.S. states and the District of Columbia from 2006 to 2020, the period for which comparable measures of healthcare access, delivery capacity, and mortality outcomes were jointly available. Variables were defined consistently across states and years to permit comparison on a common scale.

Mortality outcomes were constructed from the CDC WONDER Multiple Cause of Death files, which are based on death certificate data. State–year cause-specific death counts were used to compute age-adjusted mortality rates per 100,000 population using a common standard population throughout the study period.

The primary outcome was amenable mortality, defined as deaths for which timely and effective medical care should substantially reduce risk. This was treated as the primary outcome because it provides the clearest test of the paper’s central mechanism: whether improved access, together with sufficient delivery capacity, translates into realized clinical care and measurable health gains.

Two comparison categories were also examined. The first was narrowly defined preventable mortality, focused on external causes such as injuries, accidents, and violence. The second was more broadly defined preventable mortality, which extends the contrast category to causes driven more heavily by prevention and longer-run policy or social factors.

These outcome categories were defined using ICD-based groupings drawn from established amenable and preventable mortality frameworks, including [Nolte and McKee \(2004, 2012\)](#) and [OECD \(2019\)](#), and were applied consistently across states and years. Appendix A, Table A1 reports the specific ICD classifications used to construct each mortality category. The categories differ in the extent to which mortality should respond to clinically delivered care, making them useful for cross-outcome comparison. Amenable mortality captures deaths that timely and effective healthcare should be able to avert, whereas the preventable mortality categories increasingly reflect causes influenced by behavioral, environmental, and broader public-health factors. Table 1 summarizes the expected relationship between effective access and mortality, as well as the expected interaction between access and delivery capacity, for each mortality outcome category.

Table 1. Mechanism expectations by mortality outcome category.

Health Outcome Category (Mortality)	Expected Effect of Higher Effective Access	Expected Interaction with the Healthcare Delivery Capacity
Amenable	Weak/conditional	Capacity-activating
Preventable (narrow)	No systematic effect	No systematic interaction
Preventable (broad)	Weak/attenuated	Weak/imprecise

Note: “Capacity-activating” refers to an access–mortality relationship that becomes more favorable as delivery capacity increases because higher throughput improves the conversion of access into timely, clinically effective care.

Measures of access to care were drawn from the annual files of the CDC Behavioral Risk Factor Surveillance System (BRFSS). Three state–year indicators were used: the share of adults with health insurance coverage, the share reporting a usual source of care, and the share reporting cost-related barriers to needed care. These components were combined into an Effective Access to Care Index (EACI), defined as the unweighted mean of their standardized components, with the cost-barrier measure reverse-coded so that higher values indicate better access.² The index treats effective access as a multidimensional construct that combines coverage, attachment to care, and affordability while limiting the influence of variation in any single component.

Delivery capacity was proxied by healthcare workforce density, measured as the number of healthcare workers per 1000 residents. The measure was constructed from the U.S. Census Bureau’s American Community Survey Public Use Microdata Sample and aggregated to the state–year level. It includes healthcare practitioners and technical occupations (SOC 29-0000) and healthcare support occupations (SOC 31-0000), thereby capturing the broader clinical labor supply available to the state rather than hospital employment alone.

Capacity enters the analysis in lagged form to reflect adjustment frictions in labor supply and to reduce simultaneity with contemporaneous health outcomes.

Although healthcare workforce density provides a practical and nationally comparable measure of delivery capacity in a state–year panel setting, it should be interpreted as a broad proxy rather than a comprehensive measure of healthcare system performance. The measure captures the availability of clinical labor resources but does not directly observe appointment availability, provider geographic distribution within states, primary care access, specialist availability, hospital infrastructure, care quality, or the operational throughput of healthcare organizations. Consequently, workforce density is best viewed as an indicator of the delivery system’s potential capacity to provide care rather than a direct measure of realized service availability or quality. Despite these limitations, workforce density remains closely aligned with the study’s conceptual framework because it captures a key supply-side input required to translate improved access into clinically delivered care.

Control variables were drawn from publicly available, nationally comparable U.S. sources and harmonized to the state–year panel. Behavioral risk measures, including smoking, obesity, and diabetes prevalence, were derived from BRFSS. Additional controls captured socioeconomic conditions, demographic composition, and baseline health risk, including poverty and unemployment rates, educational attainment, age structure, racial and ethnic composition, and the behavioral risk measures noted above. All controls were measured consistently across states and years and included symmetrically across outcome categories. Appendix A, Table A2 provides variable definitions, data sources, and summary statistics for the outcome variables, treatment variables, controls, and instruments used in the panel and instrumental-variable analyses.

4. Empirical Strategy

The analysis relates mortality to healthcare access, delivery capacity, and their interaction across outcome categories that differ in their expected responsiveness to clinically delivered care. The empirical design follows the conceptual framework by allowing the relationship between access and mortality to vary with delivery capacity rather than imposing a constant access effect across all states and years. If healthcare access improves health primarily through realized clinical care, the access–capacity relationship should be strongest for amenable mortality and weaker or absent for mortality outcomes less directly responsive to routine clinical intervention.

4.1. Core Specification

For each mortality category j , the baseline state–year specification is

$$M_{jst} = \beta_0 + \beta_1 A_{st} + \beta_2 K_{s,t-1} + \beta_3 (A_{st} \times K_{s,t-1}) + X'_{st} \Gamma + \mu_s + \tau_t + \varepsilon_{jst}.$$

Here, M_{jst} denotes the age-adjusted mortality rate in the outcome category j , state s , and year t . A_{st} is the Effective Access to Care Index, and $K_{s,t-1}$ is the lagged healthcare workforce density, measured as healthcare workers per 1000 residents. The vector X_{st} includes socioeconomic, demographic, and behavioral risk controls. State fixed effects, μ_s , absorb time-invariant differences across states, while year fixed effects, τ_t , absorb national shocks and common time trends. Standard errors are clustered at the state level.

The coefficient β_3 is the main parameter of interest. A negative value implies that the association between improved access and mortality becomes more mortality-reducing as the delivery capacity increases. The conceptual framework predicts this pattern most clearly for amenable mortality, where timely and effective medical care should have the greatest potential to reduce deaths.

4.2. Capacity Heterogeneity and Marginal Effects

The interaction term reflects the complementarity between access and capacity in the production of clinically delivered care. Access expansion increases the potential demand for services, while delivery capacity determines whether the healthcare system can translate that potential demand into realized diagnosis, treatment, follow-up, and disease management. The empirical model, therefore, allows the marginal association between access and mortality to vary over the observed distribution of delivery capacity.

The implied marginal association of access with mortality is

$$\frac{\partial M_{jst}}{\partial A_{st}} = \beta_1 + \beta_3 K_{s,t-1}.$$

At a given level of delivery capacity, the marginal relationship captures how mortality changes as access improves. A negative value indicates that improved access is associated with lower mortality. Since the model includes an interaction between access and capacity, the coefficient on access alone should not be interpreted as the overall access–mortality relationship. The relevant quantity is evaluated at substantively meaningful levels of capacity, such as the 25th, 50th, 75th, and 90th percentiles of the observed capacity distribution.

The same access measure, capacity proxy, control set, and fixed-effects structure are applied across all mortality categories. This symmetric design makes the cross-outcome comparison informative. Amenable mortality serves as the primary test of the clinical delivery mechanism, whereas preventable mortality categories yield contrasting outcomes that are expected to be less directly responsive to routine healthcare access and delivery capacity.

4.3. Instrumental-Variable Specification

The analysis also estimates instrumental-variable models to address potential endogeneity in effective access to care. Endogeneity may arise if states with worsening mortality trends adopt policies or programs that expand access, if unobserved state–year factors influence both access and mortality, or if measurement error in the access index attenuates the fixed-effects estimates.

The instrumental-variable specification preserves the same capacity-interaction structure as the fixed-effects model. Both effective access, A_{st} , and its interaction with lagged capacity, $A_{st} \times K_{s,t-1}$, are treated as endogenous. The excluded instruments are Medicaid expansion in force, hospital beds per 1000 residents, and CMS primary health care funding per capita, together with their interactions with lagged delivery capacity.³

The first stage excluded instruments are:

$$Z_{st} = \{\text{Medicaid}_{st}, \text{Beds}_{st}, \text{CMS}_{st}\},$$

with corresponding interaction instruments

$$Z_{st} \times K_{s,t-1} = \{\text{Medicaid}_{st} \times K_{s,t-1}, \text{Beds}_{st} \times K_{s,t-1}, \text{CMS}_{st} \times K_{s,t-1}\}.$$

Here, Medicaid_{st} denotes an indicator for Medicaid expansion in force, Beds_{st} denotes hospital beds per 1000 residents, and CMS_{st} denotes CMS personal healthcare spending per capita.

These instruments are intended to capture policy and institutional variation that affects effective access to care. The Medicaid expansion indicator affects access primarily through insurance coverage and affordability. CMS personal healthcare spending per capita captures broader public and payer spending on healthcare services. Hospital beds per 1000 residents proxy institutional supply conditions that may influence residents' ability

to connect with the healthcare system. The interaction terms instrument the endogenous access–capacity interaction in a manner consistent with the empirical specification.

The exclusion restriction requires that the instruments affect mortality through effective access to care, conditional on state fixed effects, year fixed effects, lagged delivery capacity, and the observed controls. State fixed effects absorb time-invariant differences in state health systems, political institutions, baseline mortality risks, and long-run socioeconomic conditions. Year fixed effects absorb national policy changes, macroeconomic shocks, and secular mortality trends. The control vector includes socioeconomic, demographic, and behavioral risk factors that may directly affect mortality.⁴

We interpreted the IV estimates as complementary evidence on the direction and capacity heterogeneity of the access–mortality relationship, rather than as standalone causal estimates. To assess sensitivity to weak identification, we report first-stage diagnostics, Anderson–Rubin tests, and LIML estimates.⁵

More broadly, all estimates should be read as state-level relationships rather than direct evidence on individual treatment receipt, quality of care, or timeliness of care. The strongest evidence for the proposed mechanism would be a negative access–capacity interaction for amenable mortality, combined with weaker or different patterns for preventable mortality outcomes. Such a pattern would be consistent with the view that access expansion yields larger mortality gains where delivery systems have greater capacity and where outcomes are responsive to timely clinical care.

5. Results

5.1. Descriptive Statistics

Table 2 reports the descriptive statistics for effective access to care, delivery capacity, and mortality outcomes across state–year observations grouped by quartiles of lagged healthcare workforce density. These comparisons provide a baseline description of how the leading indicators vary across capacity environments before turning to the regression analysis. Effective access to care increases monotonically with delivery capacity. State–years in the low-capacity group had substantially lower average access, with a mean EACI of -0.642 , whereas state–years in the average-capacity group displayed intermediate levels, with a mean EACI of -0.131 . High-capacity state–years had markedly higher access, with a mean EACI of 0.592 . Healthcare workforce density also increased across these categories, ranging from 31.3 per 1000 residents in low-capacity state–years to 47.4 per 1000 residents in high-capacity state–years.

Table 2. Descriptive means by healthcare delivery capacity category.

Capacity Category (Definition)	Number of Observations (N)	Effective Access to Care Index (EACI)	Lagged Healthcare Workforce	Amenable Mortality	Preventable Mortality—Narrow	Preventable Mortality—Broad
Low capacity: Bottom Quartile of Workforce Density	179	-0.642 (0.823)	31.316 (3.015)	78.586 (19.039)	60.263 (13.89)	91.135 (16.525)
Average capacity: (Middle Quartile of Workforce Density)	357	-0.131 (0.695)	39.125 (2.702)	80.088 (19.623)	61.347 (15.224)	94.794 (18.707)
High capacity: (Upper Quartile of Workforce Density)	177	0.592 (0.458)	47.445 (3.488)	69.966 (19.343)	64.203 (19.26)	93.738 (21.535)
Total (All State–Years)	713	-0.08 (0.831)	39.244 (6.407)	77.184 (19.952)	61.786 (16.093)	93.611 (19.02)

Notes: Means with standard deviations in parentheses. Capacity categories are defined using quartiles of lagged healthcare workforce density (number of healthcare workers per 1000 residents). “Average capacity” pools the second and third quartiles. EACI is a standardized index (z-score units) in which higher values indicate better access. Mortality outcomes are age-adjusted rates per 100,000 population.

Amenable mortality was lower in high-capacity state-years than in low- and average-capacity groups. Mean amenable mortality was 78.6 deaths per 100,000 population in low-capacity state-years, 80.1 in average-capacity state-years, and 70.0 in high-capacity state-years. Preventable mortality did not show a comparable monotonic relationship with capacity. Under the narrow definition, mean preventable mortality was 60.3 in low-capacity state-years, 61.3 in average-capacity state-years, and 64.2 in high-capacity state-years. Under the broad definition, the corresponding values were 91.1, 94.8, and 93.7. These descriptive patterns are not causal, but they help motivate the capacity-heterogeneous analysis that follows.

5.2. Baseline Estimates

Table 3 presents the baseline state- and year-fixed-effects estimates. The results show little evidence of a common within-state association between effective access and mortality when the effects are constrained to be constant across settings. For amenable mortality, the coefficient on effective access was 1.27 with a standard error of 0.94. For preventable mortality, the corresponding estimates were 0.98 (standard error = 1.76) under the narrow definition and 0.47 (standard error = 1.74) under the broad definition. Across outcomes, the coefficients were small and statistically indistinguishable from zero.

Table 3. Baseline FE: Mechanism outcomes (amenable vs. preventable).

Variables	(1)	(2)	(3)
	Amenable Mortality	Preventable Mortality (Narrow)	Preventable Mortality (Broad)
Effective Access to Care Index (EACI)	1.268 (0.944)	0.977 (1.764)	0.466 (1.737)
Healthcare delivery capacity ($t - 1$)	−0.039 (0.065)	0.248 ** (0.117)	0.260 * (0.134)
Poverty rate	−4.745 (29.961)	95.237 *** (34.766)	81.370 ** (37.596)
Unemployment rate	−2.604 (54.893)	−92.404 (77.561)	−39.537 (76.184)
College BA share	−103.898 *** (37.603)	84.409 ** (36.545)	41.384 (41.215)
Share age 65+	123.299 (108.168)	−2.150 (136.963)	76.737 (154.975)
Share age 0–17	152.601 (97.591)	139.627 (102.626)	175.640 (111.867)
Share age 18–34	70.881 (79.730)	−197.733 * (102.889)	−154.726 (109.049)
Share age 35–49	55.635 (69.724)	−496.748 *** (81.933)	−388.388 *** (94.192)
Share White (NH)	−14.565 (13.652)	11.320 (23.376)	7.609 (24.724)
Share Black (NH)	−6.201 (41.675)	−286.420 *** (79.584)	−236.038 *** (79.222)
Share Asian (NH)	−476.258 *** (139.872)	−178.256 (191.943)	−299.710 (210.017)
Share Hispanic (H)	9.041 (45.433)	−133.080 (92.127)	−79.927 (102.833)
Share Male	−53.889 (96.893)	227.972 (153.035)	298.083 * (177.817)

Table 3. Cont.

Variables	(1) Amenable Mortality	(2) Preventable Mortality (Narrow)	(3) Preventable Mortality (Broad)
Smoking Prevalence	−25.486 (25.531)	−34.972 (29.306)	−6.470 (31.033)
Obesity Prevalence	11.580 (14.882)	61.093 ** (25.527)	53.330 ** (25.510)
Diabetes Prevalence	84.377 *** (28.389)	42.080 (48.222)	23.144 (47.992)
Constant	67.904 (79.352)	41.472 (107.498)	−3.911 (116.502)
Observations	713	713	713
Number of states	51	51	51
Within R ²	0.5389	0.7819	0.6244
F-statistic (df)	41.87	33.6	27.01
Rho (Fraction of variance due to state effects)	0.9661	0.9856	0.98

Standard errors in parentheses, clustered at the state level. Reference categories: age 50–64; non-Hispanic “other” race; female. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

These baseline results should be interpreted in light of the conceptual framework developed in Section 2, which predicts that the mortality effect of access is neither constant across settings nor uniform across outcomes. A baseline specification that imposes a single average access effect across all state–year observations was therefore not designed to capture the heterogeneous effects implied by the model. The remaining analysis relaxes this restriction by allowing the relationship between access and mortality to vary with delivery capacity and by comparing the resulting patterns across outcome categories.

5.3. Capacity-Heterogeneous Fixed-Effects Estimates

Table 4 reports the fixed effects estimates from interaction models that relate mortality to effective access, lagged delivery capacity, and their interaction. The specification tests whether the association between access and mortality depends on the delivery system’s ability to convert increased demand for care into timely and effective treatment.

Table 4. Capacity-conditioned effects of access on mortality (state and year FE).

Variables	(1) Amenable Mortality	(2) Preventable Mortality (Narrow)	(3) Preventable Mortality (Broad)
Effective Access to Care Index (EACI)	5.828 ** (2.508)	−8.920 *** (2.765)	−10.345 *** (2.761)
Healthcare delivery capacity ($t - 1$)	0.011 (0.070)	0.139 (0.117)	0.141 (0.131)
EACI $\times$ Capacity ($t - 1$)	−0.132 ** (0.065)	0.286 *** (0.084)	0.313 *** (0.087)
Poverty rate	−0.614 (30.746)	86.273 ** (32.956)	71.579 ** (34.833)
Unemployment rate	3.930 (57.344)	−106.587 (73.376)	−55.029 (72.529)

Table 4. Cont.

Variables	(1) Amenable Mortality	(2) Preventable Mortality (Narrow)	(3) Preventable Mortality (Broad)
College BA share	−103.204 *** (36.473)	82.903 ** (36.031)	39.739 (41.012)
Share age 65+	136.125 (107.302)	−29.986 (131.215)	46.333 (150.091)
Share age 0–17	197.863 ** (96.958)	41.393 (100.668)	68.341 (113.315)
Share age 18–34	90.595 (74.951)	−240.519 ** (100.706)	−201.461 * (105.041)
Share age 35–49	28.569 (68.534)	−438.005 *** (82.407)	−324.225 *** (94.812)
Share White (NH)	−9.939 (12.824)	1.278 (20.264)	−3.359 (21.172)
Share Black (NH)	9.277 (40.690)	−320.012 *** (76.997)	−272.730 *** (77.173)
Share Asian (NH)	−454.719 *** (136.028)	−225.005 (191.939)	−350.774 (212.218)
Share Hispanic (H)	−2.608 (46.186)	−107.795 (90.312)	−52.309 (101.634)
Share Male	−12.947 (83.337)	139.113 (147.593)	201.024 (165.524)
Smoking Prevalence	−23.517 (25.594)	−39.246 (28.587)	−11.138 (30.268)
Obesity Prevalence	10.995 (15.189)	62.363 ** (26.040)	54.717 ** (25.977)
Diabetes Prevalence	82.655 *** (29.481)	45.816 (46.056)	27.224 (46.156)
Constant	29.108 (70.426)	125.674 (99.213)	88.061 (104.367)
Observations	713	713	713
Number of states	51	51	51
Within R ²	0.5462	0.7896	0.6387
F-statistic (df)	42.33	38.98	29.23
Rho (fraction of variance due to state effects)	0.9627	0.9876	0.9838

Standard errors in parentheses, clustered at the state level. Reference categories: age 50–64; non-Hispanic “other” race; female. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

For amenable mortality, the coefficient on effective access was positive and statistically significant ($\beta = 5.828$, $SE = 2.508$), while the interaction between effective access and lagged delivery capacity was negative and statistically significant ($\beta = -0.1318$, $SE = 0.0646$). Appendix A, Figures A1 and A2 illustrate the implied marginal-effect pattern. The estimates suggest that the association between access and mortality becomes more favorable as delivery capacity increases, ultimately becoming mortality-reducing at sufficiently high levels of capacity. Expanded access may increase the potential for use of care, but measurable health gains are more likely when delivery systems can absorb that demand and provide timely, effective treatment.

Preventable mortality outcomes provide a relevant contrast. Under both the narrow and broad definitions, effective access was negative, while the interaction with capacity was positive and statistically significant. The marginal effect of access, therefore, becomes less favorable as capacity increases and does not exhibit the capacity-conditioned pattern observed for amenable mortality.

The contrast helps clarify the scope of the estimated relationship among access, delivery capacity, and mortality. Preventable mortality is shaped largely by behavioral, environmental, and social risk factors outside routine clinical care. Capacity-dependent associations for amenable mortality, coupled with their absence or reversal for preventable outcomes, are consistent with the interpretation that access reforms operate partly through clinical delivery and system-throughput channels. The preventable mortality results are therefore most appropriately read as supportive evidence regarding the boundaries of the proposed mechanism rather than as direct clinical effects. These findings should also be interpreted cautiously, as residual confounding, differences in state-level health risks, variation in policy environments, and compositional factors may contribute to the observed patterns.

5.4. Instrumental Variables Estimation Results

Table 5 addresses potential endogeneity in effective access using instrumental variable models that preserve the capacity-interaction structure. The IV estimates are intended to complement the fixed effects results rather than replace them. Since IV estimates are sensitive to instrument relevance, we report weak-identification diagnostics and weak-instrument-robust inference alongside the main estimates (Angrist & Pischke, 2009).

Table 5. Capacity-conditioned effects of access on mortality: IV (2SLS) estimates.

Variables	(1) Amenable Mortality	(2) Preventable Mortality (Narrow)	(3) Preventable Mortality (Broad)
Effective Access to Care Index (EACI)	9.324 ** (3.839)	−6.515 (6.179)	−9.390 (6.302)
Healthcare delivery capacity ($t - 1$)	−0.179 ** (0.085)	0.056 (0.166)	0.059 (0.170)
EACI $\times$ Capacity ($t - 1$)	−0.305 *** (0.104)	0.486 *** (0.161)	0.513 *** (0.165)
Poverty rate	−3.205 (34.940)	110.019 ** (43.655)	90.703 ** (45.146)
Unemployment rate	−1.103 (62.132)	−65.516 (71.918)	−21.808 (70.992)
College BA share	−115.046 *** (44.037)	−129.628 *** (47.363)	−79.131 * (47.520)
Share age 65+	191.915 * (114.325)	−195.405 (156.961)	−96.750 (181.106)
Share age 0–17	252.725 *** (96.331)	−10.291 (118.745)	13.856 (131.087)
Share age 18–34	136.041 ** (67.402)	−343.771 *** (126.015)	−293.547 ** (127.492)
Share age 35–49	44.046 (90.676)	−587.994 *** (125.720)	−444.788 *** (135.556)
Share White (NH)	−7.653 (10.702)	8.441 (20.293)	1.608 (21.962)
Share Black (NH)	16.344 (46.648)	−293.874 *** (82.212)	−254.271 *** (80.455)
Share Asian (NH)	−401.835 *** (142.801)	−349.911 (230.688)	−461.625 ** (226.570)

Table 5. Cont.

Variables	(1)	(2)	(3)
	Amenable Mortality	Preventable Mortality (Narrow)	Preventable Mortality (Broad)
Share Hispanic (H)	−5.410 (44.528)	−136.901 ** (65.115)	−74.188 (80.069)
Share Male	55.522 (84.448)	21.765 (149.949)	91.972 (157.138)
Smoking Prevalence	58.615 ** (27.704)	63.455 * (36.354)	70.230 * (37.029)
Obesity Prevalence	18.166 (17.793)	46.506 ** (22.418)	42.426 * (22.119)
Diabetes Prevalence	88.601 *** (32.726)	17.689 (45.460)	3.820 (43.503)
Constant	−40.976 (76.709)	318.661 *** (110.904)	268.140 ** (115.125)
Observations	713	713	713
Number of states	51	51	51
IV identification & weak-instrument diagnostics:			
Endogenous regressors	2	2	2
Excluded instruments	6	6	6
KP rk LM test (<i>p</i> -value)	0.0268	0.0268	0.0268
KP rk Wald F (weak-ID)	3.54	3.54	3.54
AR test <i>p</i> -value (weak-IV robust)	0.013	0.063	0.066

Notes: All specifications include state and year fixed effects. Standard errors clustered at the state level are reported in parentheses. The Kleibergen–Paap rk LM test rejects the null of under-identification, but the KP rk Wald F-statistic indicates potential weak-instrument concerns. Accordingly, Anderson–Rubin *p*-values provide weak-instrument-robust inference. The AR test rejects the joint null for amenable mortality at the 5% level and for both preventable mortality outcomes at the 10% level. (* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$).

For amenable mortality, instrumented effective access had a positive coefficient ($\beta = 9.324$, $SE = 3.839$), whereas its interaction with capacity was negative and statistically significant ($\beta = -0.305$, $SE = 0.104$). The coefficients indicate that the marginal effect of effective access on mortality becomes more pronounced as delivery capacity increases.

Lagged delivery capacity also enters with a negative coefficient, suggesting an independent role for capacity in clinically responsive outcomes. Weak-instrument diagnostics indicate that the instruments are relevant but fall short of conventional strength thresholds. Accordingly, the IV estimates were interpreted as supportive evidence and evaluated using weak-instrument-robust Anderson–Rubin tests and LIML robustness checks. The Anderson–Rubin results are consistent with the main conclusions for amenable mortality.

The contrast outcomes exhibited a different pattern. For narrowly defined preventable mortality, instrumented effective access was not statistically distinguishable from zero ($\beta = -6.515$, $SE = 6.179$), whereas the access–capacity interaction was positive and statistically significant ($\beta = 0.486$, $SE = 0.161$). The coefficient on capacity was small and not statistically distinguishable from zero ($\beta = 0.056$, $SE = 0.166$). Broadly defined preventable mortality showed the same qualitative pattern: instrumented effective access was not statistically distinguishable from zero ($\beta = -9.390$, $SE = 6.302$), the interaction was positive and statistically significant ($\beta = 0.513$, $SE = 0.165$), and the coefficient on capacity was again small and not statistically distinguishable from zero ($\beta = 0.059$, $SE = 0.170$). Weak-instrument-robust Anderson–Rubin *p*-values were correspondingly larger for the preventable mortality outcomes, at 0.063 and 0.066.

Appendix A, Figures A3 and A4 illustrate this pattern. For preventable mortality, the estimated marginal effect of access remained positive across the observed range of delivery capacity and increased with capacity. In contrast to the amenable mortality results, higher delivery capacity does not appear to shift the access effect toward reductions in mortality for these outcomes. This contrast is consistent with the view that capacity-conditioned gains from improved access are more closely linked to clinically responsive outcomes than to preventable mortality measures, which are shaped more heavily by non-clinical factors. However, these contrast estimates should be viewed as supportive rather than conclusive, given the possibility of residual confounding and the weaker IV evidence for the preventable mortality outcomes.

Overall, the results point to a conditional relationship rather than a uniform access effect. Expanded access is associated with mortality reductions primarily where delivery systems have sufficient capacity to translate access into effective treatment and where the outcomes under study remain within the institutional reach of clinical care.

6. Discussion

The findings highlight a central tension in research on healthcare access: while access expansions consistently improve coverage, affordability, and service utilization, their effects on health outcomes are more variable. Studies from the Oregon Health Insurance Experiment and the Affordable Care Act period document substantial gains in coverage and financial protection, yet the effects on mortality and other health measures have been heterogeneous across populations and settings (Baicker et al., 2013; Courtemanche et al., 2018; Sommers et al., 2017; Miller et al., 2021). Recent evidence suggests that expanded coverage can reduce mortality, but these effects differ meaningfully across states and demographic groups (Sommers, 2017; Lee et al., 2022; Levy & Buchmueller, 2025). This study contributes to this literature by identifying delivery capacity as a key source of heterogeneity in the health effects of expanded access. Access expansions appear to translate into mortality reductions primarily where the healthcare system has sufficient capacity to convert formal access into timely and effective clinical care.

This mechanism helps explain why expansions in access do not automatically produce uniform health gains. Expanded access increases opportunities to seek care, but achieving health improvements requires providers, staff, infrastructure, and organizational capacity to deliver timely diagnosis, treatment, and disease management. In higher-capacity settings, newly insured individuals are more likely to receive effective clinical interventions that can alter survival. In lower-capacity settings, access gains may still reduce financial barriers and increase utilization, yet the downstream effects on mortality are weaker or materialize more slowly. The health returns to access policy are therefore conditional on complementary delivery inputs rather than on access alone.

The distinction between amenable and preventable mortality reinforces this interpretation. Amenable mortality is specifically constructed to reflect deaths that should be responsive to timely and effective medical care (Nolte & McKee, 2004, 2012; Barber et al., 2017). The stronger access–capacity relationship observed for amenable mortality aligns with a clinical-delivery mechanism. In contrast, preventable mortality encompasses causes more heavily influenced by behavioral risks, injuries, violence, and environmental factors. The weaker and less consistent patterns for these outcomes suggest that the results do not simply reflect a general association between state resources and lower mortality. Instead, the access–capacity interaction is most pronounced where clinical care can plausibly change the survival trajectories.

These results also speak to the broader literature on healthcare quality and system performance. As Kruk et al. (2018) argue, contact with the healthcare system does not

guarantee improved health outcomes when the care received is of insufficient quality. The present findings extend this logic to state-level variation in access and delivery capacity. Formal access creates the possibility of care, but workforce availability and system throughput determine whether that possibility becomes realized treatment. This distinction has direct implications for policy evaluation: studies that report only average effects of access expansions may understate benefits in high-capacity environments and overstate expected mortality reductions in settings where delivery systems cannot readily absorb increased demand. Several limitations warrant caution. Delivery capacity is measured through healthcare workforce density, which captures a key supply-side input but does not directly reflect appointment availability, provider distribution, care quality, or operational efficiency. The instrumental-variable estimates should also be interpreted with care, as the instruments fall short of conventional strength thresholds, although the weak-instrument-robust inference and LIML estimates yielded qualitatively consistent conclusions. Finally, the state-year design cannot directly observe individual treatment decisions, timeliness of care, or clinical pathways. The findings should therefore be understood as evidence on state-level access–capacity relationships rather than as direct tests of patient-level mechanisms.

More broadly, the findings suggest that access expansion should be understood as one component of a broader health-production process rather than as a self-contained policy intervention. Access-oriented reforms are most likely to generate measurable improvements in mortality when two conditions are met: the outcome is responsive to clinical intervention, and the delivery system has adequate capacity to translate improved access into effective treatment. This perspective does not diminish the value of expanding access or reducing financial barriers. Rather, it clarifies the institutional conditions under which such policies are most likely to improve health. Access expansion and delivery capacity are therefore best viewed as complementary policy objectives: the former expands opportunities to receive care, while the latter determines whether those opportunities are translated into better health outcomes.

7. Conclusions and Policy Implications

This study shows that healthcare access operates as a conditional input to population health rather than as an independent policy endpoint. Expansions in coverage, affordability, and attachment to care increase individuals' opportunities to use medical services, but they do not, by themselves, ensure that care is delivered in a timely or effective manner. The health returns to expanded access therefore depend on the productive capacity of the delivery system and on whether the health outcome in question is responsive to clinical intervention.

The empirical results support this conditional interpretation. The access–capacity relationship is pronounced for amenable mortality, where timely diagnosis, treatment, and disease management provide a plausible pathway through which improved access can translate into improved survival. No comparable pattern emerged for preventable mortality, which is shaped more strongly by behavioral risks, injuries, environmental exposures, and broader public-health conditions. This contrast underscores a key distinction: access expansion reliably increases contact with the healthcare system, but mortality improvements are more likely to materialize when delivery systems have the capacity to convert that contact into effective clinical care.

These findings have direct implications for policy design. Access reforms and investments in delivery capacity should be treated as complementary instruments rather than sequential priorities. Coverage, affordability, and attachment to care shape the demand for healthcare services, while workforce availability, staffing ratios, and operational throughput determine the system's ability to respond. When delivery capacity is constrained, access ex-

pansions may still improve financial protection and utilization, yet the associated mortality gains are likely to be smaller, slower, or limited to specific outcomes. Strengthening delivery capacity is therefore not merely an implementation detail that follows access expansion—it is integral to whether access-oriented policy produces health improvements.

The results also inform how access policies should be evaluated. Average treatment effect estimates can obscure the institutional conditions that moderate policy effectiveness. Evaluations that average across states with heterogeneous delivery capacities risk understating benefits in high-capacity settings and overstating expected gains where systems cannot readily absorb increased demand. Similarly, evaluations that fail to distinguish clinically responsive outcomes from those driven primarily by upstream behavioral and environmental risks may mischaracterize the appropriate scope of healthcare policy. More informative evaluations should explicitly examine three interrelated conditions: whether access improved, whether the delivery system had the capacity to translate improved access into realized care, and whether the outcome was plausibly responsive to clinical intervention.

In sum, expanded access is best understood as a component of the health-production process rather than a standalone achievement. It creates the opportunity to obtain care; delivery capacity determines whether that opportunity can be realized at scale; and outcome responsiveness determines whether the care delivered can materially affect health. Health policy aimed at improving population health should therefore be designed around the conditions under which access translates into effective care, and effective care into measurable health gain.

Funding: This research received no external funding.

Institutional Review Board Statement: Not applicable.

Informed Consent Statement: Not applicable.

Data Availability Statement: The data used in this study are publicly available from the CDC WONDER Multiple Cause of Death database (available online: <https://wonder.cdc.gov/mcd.html>, accessed on 15 December 2025), the CDC Behavioral Risk Factor Surveillance System (available online: https://www.cdc.gov/brfss/annual_data/annual_data.htm, accessed on 15 December 2025), and the U.S. Census Bureau’s American Community Survey Public Use Microdata Sample (available online: <https://www.census.gov/programs-surveys/acs/microdata.html>, accessed on 15 December 2025). Additional publicly available data sources are identified in Appendix A Table A2. The processed analytic dataset and replication code are available from the corresponding author upon reasonable request.

Conflicts of Interest: The author declares no conflicts of interest.

Appendix A

Table A1. ICD-10 mortality classification framework.

Mortality Category	Principal ICD-10 Groupings	Conceptual Basis
Amenable Mortality	Selected infectious diseases (A15–A19, A35–A36, B05, B50–B54); selected malignant neoplasms amenable to treatment (e.g., colorectal, cervical, breast, testicular cancers); endocrine disorders including diabetes mellitus (E10–E14); epilepsy (G40–G41); cardiovascular diseases responsive to medical treatment (I00–I99); respiratory diseases including asthma and selected chronic respiratory conditions (J00–J99); digestive diseases including peptic ulcer and appendicitis (K25–K35); genitourinary diseases (N00–N39); maternal causes (O00–O99); selected perinatal conditions (P00–P96).	Deaths considered avoidable through timely and effective healthcare intervention following Nolte and McKee (2004, 2012) .

Table A1. Cont.

Mortality Category	Principal ICD-10 Groupings	Conceptual Basis
Preventable Mortality (Narrow)	External causes of death (V01–Y89), including transport accidents, injuries, poisoning, homicide, suicide, and other external causes.	Causes largely outside the direct reach of routine clinical care and more responsive to prevention, safety regulation, and behavioral interventions.
Preventable Mortality (Broad)	External causes (V01–Y89), alcohol-related conditions (F10, K70), tobacco-related respiratory diseases (J40–J47), selected infectious diseases, nutrition-related conditions, and other causes commonly classified as preventable in OECD mortality frameworks.	Causes responsive primarily to public health, behavioral, environmental, and prevention-oriented policies rather than to clinical treatment alone.
Notes: Mortality categories were constructed using established ICD-10-based amenable and preventable mortality frameworks. Amenable mortality follows the approach developed by Nolte and McKee (2004, 2012), while the categories for preventable mortality are based on the OECD (2019) classifications. Age-adjusted mortality rates were calculated using the standard CDC age-adjustment methodology and applied consistently across states and years. The table reports the principal ICD-10 groupings underlying each mortality category rather than an exhaustive listing of individual diagnosis codes.		

Table A2. Variable definitions, data sources, and summary statistics for the panel and instrumental-variables analyses (2006–2020).

Category (Variable)	Mean (St. Dev.)	Min	Max	N	Source (URL)	Definition
Health Outcome Measures:						
Amenable Mortality	77.772 (20.119)	45.6	159.300	714	https://wonder.cdc.gov/mcd.html (accessed on 7 December 2025)	Age-adjusted mortality rate per 100,000 population from causes classified as amenable to timely and effective medical care (ICD-10: A15–A19 [tuberculosis]; C18–C21 [colorectal cancer]; C50 [breast cancer]; C53 [cervical cancer]; E10–E14 [diabetes mellitus]; I10–I15 [hypertensive disease]; I20–I25 [ischemic heart disease, below age thresholds]; I60–I69 [cerebrovascular disease]; J45–J46 [asthma]; K35–K38 [appendicitis]).
Preventable Mortality—Narrow	61.444 (15.970)	25	144.100	714	https://wonder.cdc.gov/mcd.html (accessed on 7 December 2025)	Age-adjusted mortality rate per 100,000 from preventable causes under the narrow definition, restricted to external causes (ICD-10 Chapter XX: V01–Y89), including injuries, transport accidents, falls, poisonings, assaults, and self-harm.
Preventable Mortality—Broad	93.640 (18.916)	53.5	183.900	714	https://wonder.cdc.gov/mcd.html (accessed on 7 December 2025)	Age-adjusted mortality rate per 100,000 from preventable causes under the broad definition, including all external causes (ICD-10: V01–Y89) plus selected disease-related causes strongly influenced by prevention and policy environments, such as lung cancer (C33–C34), alcoholic liver disease (K70), selected vaccine-preventable infectious diseases (selected A-codes), and selected cardiovascular causes (selected I-codes), following CDC/OECD practice.
Treatment Variables:						
(1) Access to Care						
Effective Access to Care Index (EACI), z-score	−0.099 (0.829)	−3.138	1.374	714	https://www.cdc.gov/brfss/ (accessed on 10 December 2025)	Composite effective access index constructed as the unweighted mean of three standardized (z-score) BRFSS measures: share of adults with health insurance coverage, usual source of care (who have a personal doctor), and affordability (<i>Adults who could not afford needed care, reverse-coded so that higher values indicate better access</i>).
(2) Delivery Capacity						
Healthcare Workforce Density (HCWs per 1000)	39.629 (6.499)	21.929	62.852	714	https://usa.ipums.org/usa/ (accessed on 10 December 2025)	Total healthcare workforce per 1000 residents constructed from ACS PUMS (SOC 29 + SOC 31).
Lagged Healthcare Workforce Density ($t - 1$)	39.244 (6.407)	21.929	62.852	713	https://usa.ipums.org/usa/ (accessed on 17 December 2025)	One-year lag of healthcare workforce density per 1000 residents.
(3) Interaction						
EACI × Lagged Workforce Density	−0.134 (31.752)	−106.626	69.217	714	Constructed	Interaction between effective access and lagged delivery capacity capturing capacity-conditioned effects.
Control Factors:						
Age Structure						

Table A2. Cont.

Category (Variable)	Mean (St. Dev.)	Min	Max	N	Source (URL)	Definition
Age share: 0–17	0.231 (0.022)	0.167	0.314	714	https://usa.ipums.org/usa/ (accessed on 17 December 2025)	Share of the population aged 0–17 in a given state–year.
Age share: 18–34	0.233 (0.020)	0.195	0.355	714	https://usa.ipums.org/usa/ (accessed on 17 December 2025)	Share of the population aged 18–34 in a given state–year.
Age share: 35–49	0.197 (0.015)	0.166	0.248	714	https://usa.ipums.org/usa/ (accessed on 17 December 2025)	Share of the population aged 35–49 in a given state–year.
Age share: 50–64	0.193 (0.016)	0.135	0.239	714	https://usa.ipums.org/usa/ (accessed on 17 December 2025)	Share of the population aged 50–64 in a given state–year.
Age share: 65+	0.146 (0.024)	0.069	0.219	714	https://usa.ipums.org/usa/ (accessed on 17 December 2025)	Share of the population aged 65 and older in a given state–year.
Socioeconomic Status						
Poverty rate ($\leq 100\%$ federal poverty line)	0.150 (0.032)	0.079	0.259	714	https://usa.ipums.org/usa/ (accessed on 20 December 2025)	Share of the population living at or below the federal poverty line in a given state–year.
Unemployment rate	0.034 (0.011)	0.012	0.075	714	https://www.bls.gov/lau/ (accessed on 20 December 2025)	State-level unemployment rate in a given year.
Share of adults 25+ with BA or higher	0.297 (0.064)	0.165	0.634	714	https://www.cdc.gov/brfss/ (accessed on 20 December 2025)	Educational attainment of the adult population.
Demographics						
Share of population: White	0.763 (0.138)	0.221	0.963	714	https://www.cdc.gov/brfss/ (accessed on 20 December 2025)	Share of the population identifying as White.
Share of population: Black	0.112 (0.108)	0.002	0.554	714	https://www.cdc.gov/brfss/ (accessed on 20 December 2025)	Share of the population identifying as Black.
Share of population: Hispanic	0.064 (0.081)	0.002	0.339	714	https://www.cdc.gov/brfss/ (accessed on 20 December 2025)	Share of the population identifying as Hispanic.
Share of population: Female	0.507 (0.008)	0.473	0.532	714	https://www.cdc.gov/brfss/ (accessed on 20 December 2025)	Share of the population that is female.
Health Risk Factors						
Diagnosed diabetes prevalence	0.096 (0.020)	0.052	0.162	714	https://www.cdc.gov/brfss/ (accessed on 14 December 2025)	Prevalence of diagnosed diabetes among adults.
Obesity prevalence (BMI ≥ 30)	0.285 (0.043)	0.185	0.435	714	https://www.cdc.gov/brfss/ (accessed on 15 December 2025)	Prevalence of adult obesity (BMI ≥ 30).
Current smoker prevalence	0.180 (0.038)	0.077	0.289	714	https://www.cdc.gov/brfss/ (accessed on 14 December 2025)	Prevalence of current smoking among adults.
Instrumental Variables:						
Hospital beds per 1000 population	3.277 (1.021)	1.883	9.150	714	https://data.hrsa.gov/topics/health-workforce/ahrf/ (accessed on 21 December 2025)	Number of hospital beds per 1000 residents in a given state–year.
CMS personal health care spending per capita	8125.498 (1762.822)	4635	14,381.000	714	https://www.cms.gov/ (accessed on 21 December 2025)	CMS personal health care spending per capita across all payers.
Medicaid expansion in force (dummy)	0.293 (0.455)	0	1.000	714	https://www.kff.org/ (accessed on 21 December 2025)	Indicator equal to 1 if Medicaid expansion under the ACA is in force in a given state–year.

Notes: Unit of observation is the U.S. state–year (2006–2020). The balanced panel includes 48 contiguous states plus the District of Columbia (N = 714). Specifications using lagged workforce capacity ($t - 1$) use N = 713 due to lag construction. Mortality rates are age-adjusted and reported per 100,000 population. The Effective Access to Care Index (EACI) is the unweighted mean of three standardized BRFSS measures (insurance coverage, usual source of care, and affordability, reverse-coded). Workforce and demographic variables were derived from ACS IPUMS using appropriate person weights. All data sources are publicly available.

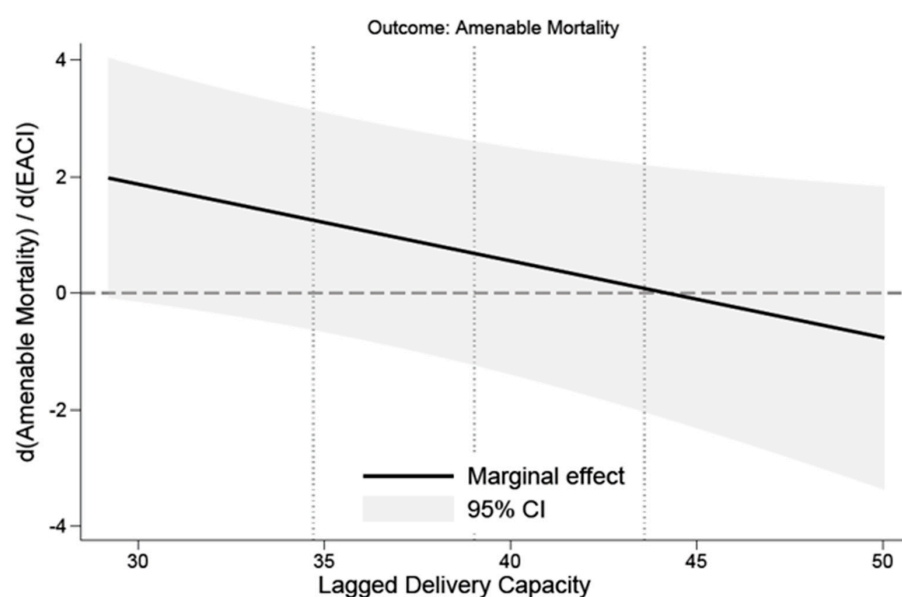


Figure A1. FE estimate of marginal effects of EACI across capacity.

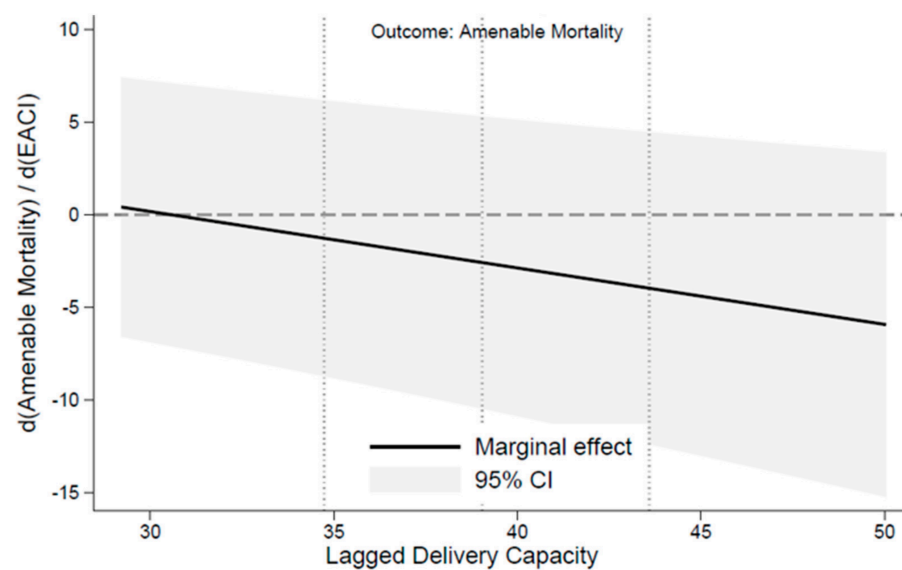


Figure A2. Marginal effect of EACI across capacity. Results from the FE IV regression model.

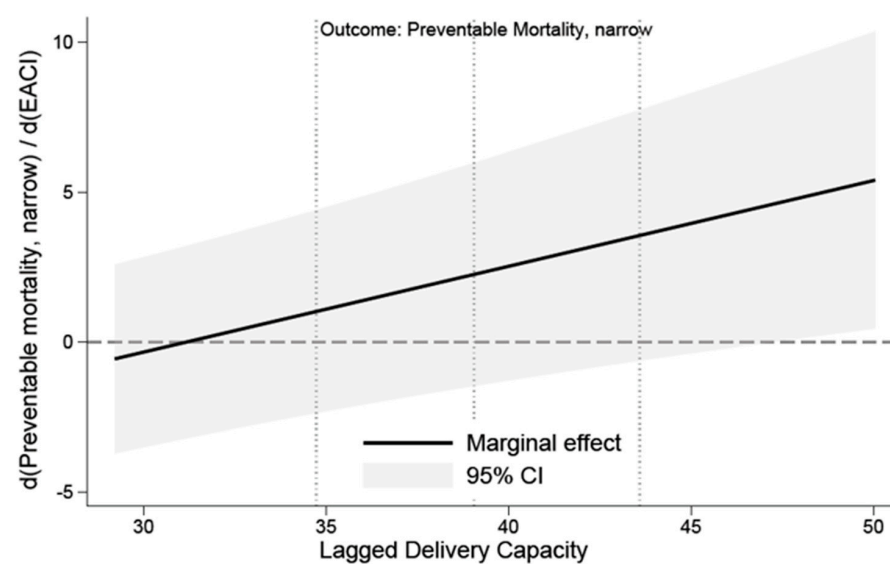


Figure A3. Marginal effect of EACI across capacity. Results from the FE regression estimation.

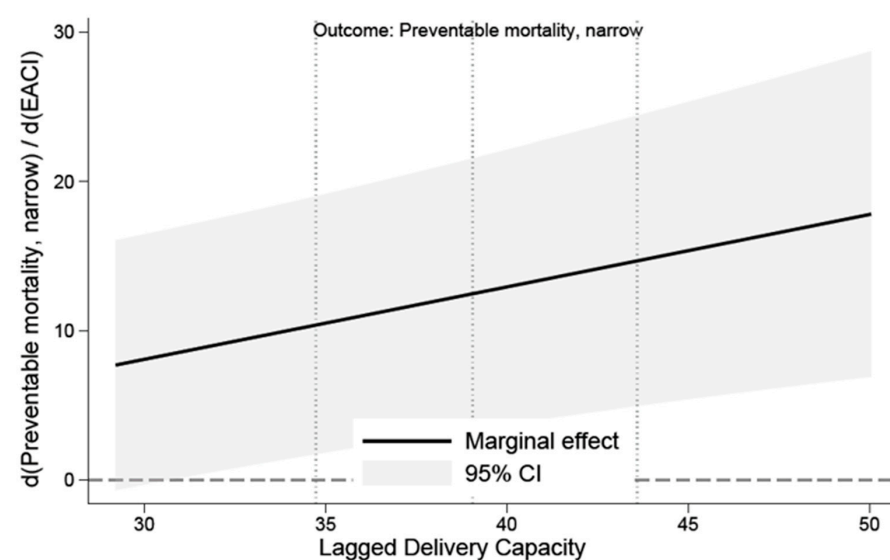


Figure A4. Marginal effect of EACI across capacity. Results from the FE IV regression estimation.

Notes

- ¹ Governance quality may also influence how effectively health systems translate resources into outcomes. The present study focuses on delivery capacity because it represents a proximate healthcare-system mechanism through which access improvements may affect the delivery of clinical care, and ultimately, health outcomes.
- ² The EACI is calculated as $[z(\text{Insurance}) + z(\text{Usual Care}) + z(\text{Cost Access})]/3$, where Cost Access denotes the reverse-coded cost-barrier component and $z(\cdot)$ denotes standardization. Higher values indicate greater effective access to healthcare services.
- ³ Hospital beds per 1000 residents may also reflect broader health-system capacity. For this reason, the IV estimates are interpreted cautiously and evaluated alongside the fixed-effects estimates, weak-instrument-robust tests, and LIML robustness checks.
- ⁴ As in all observational IV designs, the exclusion restriction is not directly testable. The identifying assumption is defended on institutional grounds and by including state fixed effects, year fixed effects, lagged capacity, and time-varying controls.
- ⁵ Anderson–Rubin tests provide inference that remains valid under weak identification, while LIML is reported because it is often less biased than conventional 2SLS when instruments are weak.

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