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ABSTRACT

Does Access to Health Care Mitigate Environmental Damages?*

Differential access to health care is commonly cited as a source of heterogeneity in the health effects of environmental exposure, yet little causal evidence exists to support such claims. We test this hypothesis by utilizing exogenous variation in both access to health care and environmental exposure. Variation in access to care is derived from the roll-out of Community Health Centers (CHCs) across US counties in the 1960s and 1970s, and variation in environmental exposure comes from random year-to-year fluctuations in ambient temperature within counties. We find that the provision of primary care through CHCs mitigates the relationship between heat and mortality by approximately 15%. Our results suggest that differential access to health care does contribute to observed heterogeneity in environmental health damages, and that improving access to primary care may be a useful means of mitigating harm from a warming climate.

JEL Classification: I10, I14, I18, Q50, Q52, Q54, Q58
Keywords: health care, access, climate, temperature, environment

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1 Introduction

In recent decades, economists have become increasingly involved in studying how the environment affects human health. Economists have played an especially pivotal role in drawing attention to the endogeneity of exposure to environmental insults. This focus has spawned a plethora of studies that employ exogenous variation in exposure to generate causal estimates of marginal damages (see Graff Zivin and Neidell (2013) for a review). In recent years it has also become increasingly recognized that environmental damages are unevenly distributed across populations (see Banzhaf et al. (2019) for a review). Indeed, many of the same studies that provide causal estimates of environmental damages also document substantial heterogeneity in the effects, often showing that disadvantaged populations suffer the greatest damages.[1] While such variation in environmental damages has been well documented in a variety of settings, economists' tools of causal identification are rarely utilized to investigate the drivers of this heterogeneity.

Understanding the sources of heterogeneity in damages is important for determining optimal environmental policy. First, predicting the benefits of an environmental policy requires understanding how the relevant population is likely to be affected. Second, understanding the drivers of heterogeneity can be informative for allocating the marginal dollar. For example, in the context of climate change, it may be more cost effective to invest in adaptation (e.g., investing in health care) rather than mitigation (e.g., investing in low carbon technology).

In a recent review, Hsiang et al. (2019) discuss the importance of precisely identifying the sources of heterogeneity in environmental damages, but also the difficulty in doing so. While causally estimating average marginal damages requires exogenous variation in environmental exposure, causally identifying heterogeneity in marginal damages requires exogenous variation in environmental exposure *and* in the source of heterogeneity.

While there are many potential sources of heterogeneity in environmental health damages, differential access to health care is often put forward as an important driver. We utilize a causal framework to test whether and to what extent a plausibly exogenous improvement in access to health care impacts temperature-induced health damages. Specifically, we test whether access to primary care through Community Health Centers (hereafter, "CHCs") moderates the relationship between (both hot and cold) temperatures and mortality.

We utilize two sources of exogenous variation (CHC access and temperature) that have

[1] For example: Chay and Greenstone (2003) and Currie and Walker (2011) each find larger health effects of air pollution among African Americans versus whites. Arceo et al. (2016) find that the effects of carbon monoxide on infant mortality are an order of magnitude larger in Mexico versus the US. Using sub-national data from 41 countries, Carleton et al. (2018) find that the effects of high temperatures on mortality are consistently larger for poorer populations.

each been analyzed rigorously in prior studies. Bailey and Goodman-Bacon (2015) study the initial round of CHC establishments, which took place from 1965 to 1974. During this period, Bailey and Goodman-Bacon (2015) show that the timing of CHC establishment in a county was effectively random. This was due, at least in part, to a period known as the "great administrative confusion" at the federal agency responsible for allocating CHC funding. Bailey and Goodman-Bacon (2015) find that CHCs reduce all-cause mortality in the years after initial establishment, that these ameliorative effects grow over time, and that the reductions in general mortality rates are driven primarily by older adults and cardiovascular/cerebrovascular causes of death. We unite the approach of Bailey and Goodman-Bacon (2015) with the panel-fixed effects methodology that has been widely used to causally identify impacts of temperature on a variety of outcomes including mortality (e.g., Deschênes and Greenstone, 2011; Dell et al., 2014).

We chose this setting explicitly because it satisfies a long list of requirements that are necessary for causally identifying the role of access to health care in environmental damages. First, there must exist exogenous variation in both the environmental shock and access to health care. Second, these sources of variation must overlap such that effects of the environmental shock can be estimated before and after the change in access, and for populations that did and did not experience the change. Third, both the environmental shock and the measure of access must plausibly affect the same outcome (e.g., the same causes of death) and the same population (e.g., the same age groups). Fourth, the direct effects of both the environmental shock and the measure of access should exhibit very high statistical power, which is necessary for identifying interaction effects of plausible magnitude. All of these requirements are satisfied in the setting we analyze.

Our analysis begins with a successful replication of the central results from both Bailey and Goodman-Bacon (2015) and studies of the effects of temperature on mortality (e.g., Deschênes and Greenstone, 2011) in a single estimating equation. We then construct a model to estimate the interaction between access to health care and temperature. Conceptually, our approach is to estimate a difference-in-differences (henceforth, "DiD") model for the effect of access to health care on the temperature-mortality relationship. To this end, our models allow for time-invariant differences in the temperature-mortality relationship across treatment groups (analogous to including a treatment group indicator or group fixed effects in a standard DiD), and group-invariant differences in the temperature-mortality relationship over time (analogous to the post-treatment indicator or time fixed effects in a standard DiD).[2]

[2]Analogous controls are used by Hornbeck and Keskin (2014) in the estimation of how aquifer access mitigates the impacts of drought on agricultural yields.

Our estimates indicate that the presence of a CHC in a county mitigates the relationship between hot temperatures and mortality by approximately 15%. We find no statistically significant evidence that the primary care services provided via CHC access affect the cold-mortality relationship, though the confidence intervals cannot rule out meaningful mitigation effects. These results are important in a general sense as they demonstrate that increased access to health care can indeed mitigate environmental health damages. As such, differential access to care may be one of the drivers behind oft-noted differences in environmental damages across rich and poor populations. More specific to our setting, the results suggest that increasing access to primary care is a policy lever that can be used to reduce temperature-related health damages. As such, increased access to health care may serve as a successful adaptive mechanism for climate change.

This paper contributes to an active literature seeking to identify heterogeneity in environmental damages. Several papers set in developing world contexts consider environmental shocks that affect outcomes through changes in income.[3] Most closely related to our work, however, is a set of papers that explicitly consider heterogeneity in the effects of temperature on mortality: Barreca et al. (2016); Burgess et al. (2017); Banerjee and Maharaj (2018); Cohen and Dechezleprêtre (2018).

Burgess et al. (2017) demonstrate that from 1957-2000, the heat-mortality relationship was much stronger in rural versus urban India. The authors use exogenous variation in bank access to show that a median increase in bank access mitigated the heat-mortality relationship in rural India by approximately 75%. These results imply that bank access was able to smooth temperature-induced shocks to agricultural income. Banerjee and Maharaj (2018) is similarly set in India and also focuses on agricultural income as an important mechanism. The authors investigate the relationship between high temperatures and infant mortality in India, and consider two factors that could potentially mitigate the relationship: a workfare program (NREGA) and a health worker program. They find that NREGA has no mitigating impact, whereas 9 months of pre-natal exposure to the health worker program mitigates the relationship between temperature and infant mortality by over 80%. While these estimates are interesting and important, they do not allow for pre-existing differences in

[3]Fetzer (2014) demonstrates that access to a workfare program in India (National Rural Employment Guarantee Act - "NREGA") successfully mitigates the relationship between agriculture-affecting rainfall shocks and violence; relatedly, Sarsons (2015) finds no evidence that dam (i.e., irrigation) access mitigates the rainfall-violence relationship in India. Garg et al. (2018) find that access to NREGA mitigates the relationship between high temperatures and test scores by approximately 38% in India. Adhvaryu et al. (2018) estimate that a conditional cash transfer program in Mexico (PROGRESA) mitigates the disadvantage caused by early life rainfall shocks by at least 20%. Garg et al. (2019) find that the same cash transfers from PROGRESA reduce the heat-induced homicide rate in Mexico by half to two-thirds. Gunnsteinsson et al. (2018) estimate that Vitamin A supplementation fully mitigates the effect of *in-utero* exposure to a tornado on infant/childhood growth outcomes in Bangladesh.

the heat-infant-mortality relationship between states that benefited from the health worker program and those that did not. As such, the estimates may partially reflect such differences.[4]

Cohen and Dechezleprêtre (2018) document the relationship between temperature and mortality in Mexico over the period 1998-2010. The authors find that both cold and hot temperatures are associated with increases in mortality, but the effects of cold (days with mean temperature <50°F) are much stronger. The authors then test whether enrollment in Mexico's national health insurance program – *Seguro Popular* – provides protective benefits against temperature-related mortality. This is done by matching enrolled decedents to non-enrolled decedents based on a set of observed characteristics, and then comparing the effects of temperature on mortality for each group. They find that enrollment in *Seguro Popular* mitigates the mortality effects of a cold day by 35%, and argue that any selection on unobservables likely attenuates this estimate.

In terms of setting, the foundational work of Barreca et al. (2016) is most closely related to ours. The authors demonstrate a "remarkable" decline in the relationship between high temperatures and mortality over the course of the 20th century in the US. This decline was particularly dramatic during a period of rapid expansion in air conditioning technology (henceforth, "AC") after 1960. In Figure 1, we use our own data to show that this decline was remarkable indeed: between 1959 and 1988, the heat-mortality relationship declined by 70%. Notably, in the same period the cold-mortality relationship declined by 60% as well. Barreca et al. (2016) estimate the interaction between temperature and AC penetration, and the results imply that the diffusion of AC technology explains the entire decline in the heat-mortality relationship. They also estimate interactions between temperature and the number of doctors per capita and electrification rates, but find no mitigating impacts. However, as the authors note, there does not exist quasi-experimental variation in AC penetration rates or the other potential modifiers. While the interaction estimates undoubtedly represent a real and substantial impact of AC on the heat-mortality relationship, contributions of other simultaneously evolving factors cannot be completely ruled out.

Coinciding with the period of rapid expansion in AC technology was a period of expanding access to – and spending on – health care. The CHC program, Medicare, and Medicaid were

[4]This may be a particularly important point given that the roll-out of the health worker program studied by Banerjee and Maharaj (2018) was explicitly non-random, as it was first implemented in Indian states identified as laggards in a variety of public health measures (Rao, 2014).

all implemented beginning in the mid-1960s.[5] Figure 1 shows trends in the US age-adjusted mortality rate ("AMR") and US government spending on health care for our study period, 1959-1988. During this period, government health expenditures as a share of GDP more than tripled, and there was a dramatic 28% decline in the overall AMR, reflecting increased life expectancy and resilience to potential health shocks. In principle, any of the expansions in access to care could have mitigated the health impacts of environmental exposure. We focus on the CHC program because of its credibly exogenous implementation and because Bailey and Goodman-Bacon (2015) have shown that the program had large mortality effects on the same population and causes of death that we expect to be impacted by exposure to extreme temperatures.

While Barreca et al. (2016), Banerjee and Maharaj (2018), and Cohen and Dechezleprêtre (2018) each provide an estimate of how access to health care mitigates the temperature-mortality relationship, to the best of our knowledge our paper is the first to utilize a natural experiment in access to health care to address this question. Our focus on causally identifying whether access to health care mitigates environmental damages is our primary contribution.[6]

The remainder of this paper proceeds as follows: Section 2 provides background on community health centers and the empirical setting. Section 3 outlines the details of our data and empirical approach. Section 4 presents our results and a series of robustness checks and extensions to the main analysis, and Section 5 concludes.

2 Background

In our description of CHCs, we provide only the detail necessary for interpretation of our analysis and we refer the reader to Bailey and Goodman-Bacon (2015) for additional detail.

[5] We sought to leverage the roll-outs of both Medicare and Medicaid for exogenous variation in access to health care in order to supplement the analysis in this paper. Neither were found to meet the standards necessary to identify the interaction effects of interest here. Specifically, Finkelstein and McKnight (2008) find no evidence that Medicare affected mortality, so it is unlikely that it significantly affected temperature-related mortality. Extending the approach used in Goodman-Bacon (2018b) to identify the effects of Medicaid implementation on non-white child mortality, we found that the interaction effects were too imprecise to draw any meaningful conclusions.

[6] Our paper also contributes to a broader literature in empirical economics aimed at identifying interaction effects by leveraging exogenous variation in multiple treatments. This strategy is particularly prominent in the literature on "dynamic complementarities" in early childhood development (Almond and Mazumder, 2013; Adhvaryu et al., 2018; Johnson and Jackson, 2019; Rossin-Slater and Wüst, 2018). Furthermore, our paper contributes to the rapidly expanding literature on the effects of temperature and climate on health in general. To date, this literature has identified such impacts across a wide variety of outcomes including: mortality (Barreca et al., 2016; Heutel et al., 2017), morbidity (White, 2017; Karlsson and Ziebarth, 2018), mental health (Mullins and White, 2019) and occupational health (Dillender, 2019), and across settings representing over half of the world's population (Carleton et al., 2018).

2.1 What Are CHCs and How Were They Implemented?

While the CHC program still exists, it has changed in character substantially since its initial implementation (i.e., the period in which our analysis is set). CHCs were clinics or networks of clinics that provided direct and comprehensive primary care services at little or no cost to the patient. These clinics were typically located in neighborhoods with few alternative health care options and served patients who were uninsured, underinsured, or low-income. CHCs often employed multiple clinic locations or mobile units, and over 90% provided home health care or transportation to appointments (Bailey and Goodman-Bacon, 2015).

The CHC program was initiated in 1965 as part of President Lyndon Johnson's "unconditional war on poverty". It was a grant reward program administered by the Office of Economic Opportunity ("OEO"), which provided direct grants to local organizations for War-on-Poverty programs. Like Bailey and Goodman-Bacon (2015), we rely on the chaotic period known as the "great administrative confusion" at the OEO as a source of quasi-random variation in access to health care. OEO funding decisions during this period (1965-1974) were characterized as "wild", and Bailey and Goodman-Bacon (2015) show little association between the timing of CHC establishment and pre-treatment county characteristics, mortality rates, changes in mortality rates, funding for other OEO programs, or local expansions in hospital capacity. Following Bailey and Goodman-Bacon (2015), we only utilize variation from the first wave of CHC establishments which took place between 1965 and 1974. The program was fundamentally altered by the 1975 Special Health Revenue Sharing Act which made CHC establishments much less plausibly exogenous and focused them in sparsely-populated rural settings; Bailey and Goodman-Bacon (2015) also argue that these later CHCs likely had much smaller impacts on mortality.

2.2 How Can CHCs Affect Mortality and Temperature-Related Mortality?

CHCs principally provided access to primary and preventative health care, and deaths that could be prevented by such care were heavily concentrated in older adult and elderly populations. Bailey and Goodman-Bacon (2015) note a number of ways in which CHCs may have reduced mortality rates. CHCs may have increased early detection of health conditions, or increased awareness about chronic yet manageable conditions like hypertension, which requires ongoing care and long-term medication. Patients would also have been able to get free or reduced-cost pharmaceuticals for the management of such conditions (e.g., beta blockers for hypertension) through CHCs, and the increased continuity of care likely made it easier for patients to maintain medication regimens. Indirect mechanisms are also plausible: CHC

access may have increased awareness about Medicaid and Medicare, thereby increasing access to treatments for acute conditions. It is also possible that CHCs decreased emergency department usage for non-emergency conditions and thus reduced emergency department crowding; as such, mortality even for non-users of CHCs could have been affected.

Given the dimensions of care provided by CHCs, access might be expected to increase the health stock of served populations. Improvements in the health stock of the population could in turn increase the population's resilience to health shocks of many kinds, including exposure to extreme temperatures. To take a concrete example, suppose that CHCs increased use of anti-hypertensive medication. Since hypertension is a risk factor for heart attack and stroke, an improvement in hypertension management would have made the population less susceptible to triggers for these events. Indeed, Bailey and Goodman-Bacon (2015) show that CHCs did lead to better hypertension management and that CHC-induced mortality reductions were primarily driven by cardiovascular disease (e.g., heart attack) and cerebrovascular disease (e.g., stroke). If either cold or hot temperatures trigger the events that CHCs prevented, then it is at least plausible that increasing CHC access could reduce temperature-related mortality from such events. The evidence supports this plausibility: we find that cardiovascular and cerebrovascular disease account for 47% of all cold-related deaths and 67% of all heat-related deaths in the pre-CHC period.[7] We also note that CHCs primarily benefitted the low-income population; to the extent that low-income individuals were also more temperature exposed (e.g., due to working conditions or lower levels of access to heating and air conditioning), interaction effects are even more plausible. We now turn to testing for such interactions empirically.

3 Data and Empirical Strategy

3.1 Data

This analysis brings together multiple data sources at the county-year-month level for the period 1959-1988. This sample matches that used by Bailey and Goodman-Bacon (2015), which covers the first wave of CHCs establishments in the US between 1965-1974 and the subsequent 14 year period. In our sample, CHCs were established in 114 counties between

[7] To arrive at these figures, we limit the sample to 1959-1964 and calculate the effects of days $<40°$F and $>80°$F, for both all-cause mortality and cardiovascular/cerebrovascular mortality. We find that one day $<40°$F increases the all-cause mortality rate by 0.241 (per 100,000 population) and the cardiovascular/cerebrovascular mortality rate by 0.113. The proportion of cold-related mortality attributable to cardiovascular and cerebrovascular causes is the ratio 0.113/0.241. One day $>80°$F increases the all-cause mortality rate by 0.339 and the cardiovascular/cerebrovascular mortality rate by 0.226

1965-1974.[8] Data on CHC establishment dates were provided by Martha Bailey and Andrew Goodman-Bacon and were collected from primary sources. County-year-month data on mortality is derived from the National Vital Statistics System mortality files. Age-adjusted mortality rates per 100,000 population are calculated using county-year population data from the U.S. Census and National Cancer Institute (SEER Program). County-year-month temperature and precipitation measures are derived from data constructed by the PRISM Climate Group and aggregated by Schlenker and Roberts (2009). Temperature is measured as the mean daily temperature in degrees Fahrenheit (calculated as the mean of the daily minimum and maximum). State-year data on air conditioning penetration rates are derived from U.S. Census data following Barreca et al. (2016). Additional details on the data are provided throughout this section, but for much greater detail on data sources and construction, see the Data Appendix. Summary statistics for mortality, climate variables, and air conditioning are provided for all counties in our sample, and separately for CHC and non-CHC counties in Table 1.

3.2 Empirical Strategy

3.2.1 Replication Model

We begin by replicating the effects of both CHC access on mortality *and* temperature on mortality in a single econometric model. The model specification follows primarily from Bailey and Goodman-Bacon (2015). Fortunately, the rich set of controls included in Bailey and Goodman-Bacon (2015) are well-suited for identifying ambient temperature effects as well. Our model is distinct from Bailey and Goodman-Bacon (2015) in two ways: (1) our model is estimated at the monthly rather than annual level, and thus some of the fixed effects are adjusted accordingly, and (2) our model includes climatic variables. Equation (1) describes the model.

$$\text{AMR}_{cym} = \gamma \text{CHC}_{cy}^{t \geq 0} + \pi g(\text{Temp}_{cym}) + \beta \text{X}_{cym} + \delta_{sy} + \delta_{cm} + \delta_{uy} + \delta_{ym} + \varepsilon_{cym} \qquad (1)$$

The outcome of interest is AMR_{cym}: the age-adjusted mortality rate in county c, year y

[8] CHCs were also established in New York City, Los Angeles and Chicago. We follow Bailey and Goodman-Bacon (2015) and omit these from our analytical sample given their large size and the disproportionate weight they would receive in the regressions.

and month m.[9] The first coefficient of interest is γ, where $\text{CHC}_{cy}^{t\geq 0}$ is an indicator equal to one in the years after CHC establishment in a particular county (superscripts indicate years relative to establishment; $t = 0$ represents the year in which a CHC was established). Additional models are presented in which $\text{CHC}_{cy}^{t\geq 0}$ is replaced with a set of binned event-study indicators for periods relative to the year of CHC establishment: $\text{CHC}_{cy}^{t\leq -2}, \text{CHC}_{cy}^{0\leq t\leq 4}, \text{CHC}_{cy}^{5\leq t\leq 9}$, and $\text{CHC}_{cy}^{t\geq 10}$ ($t = -1$ is the reference group). The binned specification follows the main specification of Bailey and Goodman-Bacon (2015) and allows for the assessment of differential pre-treatment trends and dynamic treatment effects.

The second coefficient of interest is π, where $g(\text{Temp}_{cym})$ is some function of mean daily temperatures in a given county-year-month. In the main specification, $g(\text{Temp}_{cym})$ is a vector of temperature bins measuring the number of days with mean temperatures within a given range. For example, $\text{Temp}_{cym}^{<40}$ and $\text{Temp}_{cym}^{>80}$ represent the number of days below 40°F and above 80°F, respectively. We estimate models that include only two temperature variables (i.e., $\text{Temp}_{cym}^{<40}$ and $\text{Temp}_{cym}^{>80}$), and models that include these in addition to intermediate 10°F bins (i.e., $\text{Temp}_{cym}^{<40}$, $\text{Temp}_{cym}^{40-50}$, $\text{Temp}_{cym}^{50-60}$, $\text{Temp}_{cym}^{70-80}$, and $\text{Temp}_{cym}^{>80}$). The simpler models are preferred due to the ease of interpretation and the fact that fewer parameters need to be estimated (this is especially important for the interaction models to follow). We also estimate models in which $g(\text{Temp}_{cym})$ represents a third-order polynomial in temperature; these models allow for nonlinear impacts of temperature across the entire temperature distribution, but only require estimating three parameters.[10]

The remaining controls ($\text{X}_{cy}, \delta_{sy}, \delta_{cm}, \delta_{uy}, \delta_{ym}$) are equivalent to the controls used in Bailey and Goodman-Bacon (2015), but adapted to the monthly time scale and additionally include controls for precipitation.[11] X_{cym} is a vector of county-level time-varying covariates (including precipitation). δ_{sy} are state-by-year fixed effects and δ_{uy} are urban-group-by-year

[9] Age-adjusted mortality rates hold fixed the age distribution of the population of a given county such that changes in the AMR reflect changes in the risk of death rather than changes in the age composition of the sample. The AMR for county c at time t is calculated as a weighted average of age-specific mortality rates (ASMR) for county c at time t and 5-year age group a. For concreteness, define $ASMR_{cta} = 100,000 \times \frac{Deaths_{cta}}{Pop_{cta}}$, and define $AMR_{ct} = \sum_{a=1}^{18} s_{ca} \times ASMR_{cta}$, where s_{ca} is the 1960 share of the population in 5-year age group a. Age-adjusting refers to holding the population age share s_{ca} fixed.

[10] We follow Carleton et al. (2018) in constructing these polynomials. Specifically, we first construct a third-order polynomial in temperature at the *daily* level, and then sum these three polynomial terms across the month. As such, these models allow us to exploit daily variation in local temperatures in models where the unit of observation is at the monthly level (i.e., similar to the temperature bin approach). In interpreting the estimates, we test whether the effect of an additional day at a given temperature is different from an additional day at 65°F. Higher order polynomials were considered, and the results were qualitatively unchanged.

[11] Following Barreca et al. (2016), precipitation controls are indicators for whether total monthly precipitation was below the 25th percentile or above the 75th percentile of the county-month distribution.

fixed effects.[12] δ_{cm} are county-by-month fixed effects which are used in place of the county fixed effects in Bailey and Goodman-Bacon (2015). In addition to controlling for time-invariant differences across counties, the county-by-month fixed effects control for local-level seasonality. This is potentially important because both temperature and mortality exhibit substantial seasonality, which may differ between regions. δ_{ym} are year-by-month fixed effects which absorb any nationwide trends or shocks.

Standard errors are two-way clustered at both the county and year-month levels (Cameron et al., 2011). County clustering allows for arbitrary within-county serial correlation and year-month clustering allows for arbitrary spatial correlation within a year-month. All regressions are weighted by 1960 county populations.

Identification of γ in Equation (1) requires the usual parallel trends assumption for a DiD design: in the absence of treatment, trends in mortality would have been similar between counties in which CHCs were established at different dates or not at all. Bailey and Goodman-Bacon (2015) present substantial evidence supporting this identifying assumption, and we refer the inquisitive reader to their work for details. That said, we do present some of this evidence (e.g., pre-treatment effects in the event studies) for comparison with the interaction models that follow.

Identification of π in Equation (1) requires the assumption that within a given county-month, year-to-year weather realizations are uncorrelated with other unobserved determinants of mortality. Conditional on county-by-month fixed effects and other controls, year-to-year weather realizations are essentially random and thus this assumption is likely to be satisfied, though we again refer the reader to the work of others for a more detailed discussion (e.g., Deschênes and Greenstone, 2007).

In order to identify the interaction effect between temperatures and CHCs, we require the additional assumption that the two treatments are independent of one-another, conditional on our control regime. Because we are relying on random weather shocks within a county-month, there is little plausible reason to be concerned that temperature variation is related to the establishment or presence of CHCs. If the treatments are unrelated, as we expect, then the estimated impacts of each treatment should be insensitive to the inclusion of the other treatment variables in the regression. In our presentation of results, we separately estimate versions of Equation (1) with each treatment in isolation and then including both.

[12]The time-varying covariates were obtained from Bailey and Goodman-Bacon (2015) and include hospital beds per capita, hospitals per capita, public assistance transfers, and retirement transfers. Also included are annual time trends interacted with the levels of each of the following county characteristics measured in 1960: percent with income under $3,000, percent non-white, percent rural, percent urban, and number of physicians. The urban-group-by-year fixed effects are year dummies interacted with five categories of a county's 1960 population share in urban areas.

We find that the coefficient estimates for both treatments are effectively unchanged across these models.[13]

3.2.2 Interaction Model

Summary statistics in Table 1 reveal baseline differences across CHC and non-CHC counties. In particular, CHC counties had higher average mortality rates in the pre-CHC period (1959-1964) and were slightly warmer on average. These cross-sectional differences across counties in average mortality rates and climate conditions are accounted for through county fixed effects. While these controls are sufficient to separately estimate the effects of either CHC access or temperature on mortality, causally identifying the interaction requires additional controls. Conceptually, the empirical approach is to estimate a DiD model for *the effect of CHC access on the temperature-mortality relationship*. It is likely that there are cross-sectional differences in the temperature-mortality relationship that county fixed-effects would not account for (e.g., suppose the heat-mortality relationship is weaker in hot regions). Our preferred specification for the interaction model explicitly absorbs cross-sectional differences in the temperature-mortality relationship between the CHC and non-CHC counties by allowing baseline temperature effects to differ between these two groups. In this way we ensure our estimates are identified from the *change* in the temperature-mortality relationship before and after CHC establishment, rather than cross-sectional differences. Practically speaking we are estimating a triple-differences model, described below in Equation (2).

$$\begin{aligned} \text{AMR}_{cym} = {} & \phi(\text{CHC}_{cy}^{t\geq 0} \times g(\text{Temp}_{cym})) + \gamma \text{CHC}_{cy}^{t\geq 0} + \pi g(\text{Temp}_{cym}) \\ & + \theta(g(\text{Temp}_{cym}) \times \text{Treated}_c) + g(\text{Temp}_{cym}) \times \delta_y + \kappa(g(\text{Temp}_{cym}) \times \text{AC}_{sy}) \\ & + \beta \text{X}_{cym} + \delta_{sy} + \delta_{cm} + \delta_{uy} + \delta_{ym} + \varepsilon_{cym} \end{aligned} \tag{2}$$

The coefficient of interest in Equation (2) is ϕ. We allow for fixed differences in the temperature-mortality relationship between CHC and non-CHC counties through the inclusion of $g(\text{Temp}_{cym}) \times \text{Treated}_c$, where Treated_c is an indicator for whether a CHC was ever established in the county over the 1965-1974 period. We also estimate models that control for fixed differences across all counties in the temperature-mortality relationship (analogous to county fixed effects in a standard DiD) by including county-specific temperature effects: $g(\text{Temp}_{cym}) \times \delta_c$. This requires estimating over 3,000 additional parameters for each temperature variable, and as such the more parsimonious model is our preferred specification.

[13] Additionally, we test more directly for independence among the two treatments by regressing each treatment on the other and find no significant relationships. These results are presented in Table A1.

Furthermore, just as time fixed effects in a standard DiD specification absorb differences in mortality over time that are common across counties, our interaction model should account for differences in the temperature-mortality relationship over time. To this end, year-specific temperature effects are included: $g(\text{Temp}_{cym}) \times \delta_y$.

Finally, because the roll-out of CHCs occurred during a period of increasing AC penetration rates, it is potentially important to allow for differential effects of temperature across AC penetration rates. Table 1 shows that AC penetration rates increased slightly slower in CHC counties relative to non-CHC counties; as such, failing to control for AC effects would likely bias our estimates toward zero.[14] Following Barreca et al. (2016), we include the interaction between temperature and the state-year AC penetration rate: $g(\text{Temp}_{cym}) \times \text{AC}_{sy}$.

With these controls included, ϕ in Equation (2) identifies the change in the temperature-mortality relationship from before to after CHC establishment, relative to the change in the temperature-mortality relationship in counties where CHCs were established in different years or not at all. The identifying assumption is similar to that of a standard DiD approach: in the absence of treatment, trends in the temperature-mortality relationship would have been similar in counties where CHCs were established in different years or not at all. While this assumption is fundamentally un-testable, indirect tests support its plausibility. Most importantly, an event-study version of Equation (2) is estimated to test for differential trends in the temperature-mortality relationship prior to CHC establishment. We provide estimates of a binned event study (to maximize the power of the estimates), as well as a full year-by-year event-study.

Further note that the model described in Equation (2), $g(\text{Temp}_{cym})$ is a function of mean daily temperatures. Just as in the replication model, our preferred specifications utilize either a set of temperature bins or a third-order polynomial in temperature. See the appendix for a generalized empirical model that allows for J event-study indicators measuring CHC access and G temperature variables.

4 Results

4.1 Replication Model

The results of the most basic replication models are presented in Table 2. The outcome is the age-adjusted mortality rate (AMR) per 100,000 population, and all models include the set of fixed effects and controls described in Equation (1). The models vary in whether

[14] The likely attenuation bias is under the assumption that access to AC and access to CHCs both affect the temperature-mortality relationship in the same direction.

and how the effects of each treatment (CHCs and temperature) are incorporated. Column 1 starts with a simple model for the effect of CHC access on mortality with a single indicator for the presence of an established CHC in a county ($CHC^{t\geq 0}$), excluding all temperature variables. The coefficient estimate indicates that CHC establishment leads to a significant decrease in the monthly AMR by approximately 1.1. Relative to the pre-CHC mean AMR of 76.5 reported in Table 1, this represents a 1.4% decrease in the mortality rate.

Column 2 presents estimates from a simple model for the effects of temperature on mortality, excluding any measures of CHC access. The coefficient estimates indicate that both cold and hot temperatures lead to increases in mortality. The coefficient on $Temp^{<40}$ implies that one additional day under 40°F increases the monthly AMR by 0.11, relative to a day in the 40-80°F range. $Temp^{>80}$ implies one day over 80°F increases the monthly AMR by approximately 0.18. Importantly for the estimation of interaction effects that follow, the statistical power is extremely high for all estimates in Columns 1 and 2: the t-statistics for the coefficients on $CHC^{t\geq 0}$, $Temp^{<40}$, and $Temp^{>80}$ equal 3.6, 7.1 and 9.7, respectively (all are significant at the 0.1% level).[15]

Columns 1 and 2 demonstrate successful replications of Bailey and Goodman-Bacon (2015) and studies on the effects of temperature on mortality (e.g., Deschênes and Greenstone, 2011; Barreca et al., 2016) using a common econometric framework. Mirroring the results of the replicated studies, our estimates show that CHCs reduce mortality rates and extreme temperatures (both hot and cold) increase them. Column 3 includes both treatment variables in a single model. When the CHC and temperature variables are simultaneously included in the model, the coefficient estimates for each treatment remain virtually unchanged. This reinforces the notion that the variation utilized to identify the effects of these two treatments with respect to mortality are independent and thus identification of interaction effects between the treatments is unlikely to be confounded by some unaddressed interdependence.

The estimates in Columns 4 and 5 use models in which the single CHC indicator is replaced by four indicators for time relative to CHC establishment (i.e., a binned event-study model) following Bailey and Goodman-Bacon (2015). The coefficient on the pre-treatment CHC indicator ($CHC^{t\leq -2}$) is not statistically different from zero, indicating no evidence of differential trends in mortality prior to CHC establishment. The coefficient estimates on the three post-treatment CHC indicators are all negative, highly significant, and increasing with time relative to CHC establishment. The results in Column 5 (which includes temperature

[15]The extreme temperature bins ($Temp^{<40}_{cym}$ and $Temp^{>80}_{cym}$) were chosen primarily to maximize the power of the estimates since high statistical power is necessary to identify interaction effects in the models that follow. While more extreme temperatures such as temperatures >90°F lead to greater damages, these are rare events and the estimates have large standard errors in the binned specifications. The full distribution of temperature is explored in polynomial specifications.

variables) also closely mirror the results of Bailey and Goodman-Bacon (2015): our coefficient estimates on the $CHC^{0\leq t\leq 4}$, $CHC^{5\leq t\leq 9}$, $CHC^{t\geq 10}$ bins are -0.82, -1.51, and -1.63, respectively, whereas the coefficient estimates from the equivalent model in Bailey and Goodman-Bacon (2015) are -0.84, -1.58, and -1.46, respectively.[16] Our estimates for the effects of temperature are not directly comparable to those of Barreca et al. (2016) or similar papers because of differences in outcome measures, sample periods, and model specifications, but the estimates are of qualitatively similar character and magnitude.[17]

Results from more flexible replication models are presented in Figure 2. The first panel again replicates a result of Bailey and Goodman-Bacon (2015) and reports single-year event study estimates (as opposed to a binned event study) for the effect of CHCs on mortality. These estimates provide more detail on the dynamics of the treatment effects. The estimates again indicate little evidence of differential pre-treatment trends in mortality prior to CHC establishment, and a negative (i.e., ameliorative) treatment effect that emerges post-treatment and grows over time. A similar single-year event-study will be estimated in the next section for the effects of CHCs on the temperature-mortality relationship.

Panel B of Figure 2 reports coefficient estimates for five 10°F temperature bins (60-70°F is excluded), which were included in the model in place of the two extreme bins only. This yields the familiar U-shaped relationship between temperature and mortality that has been well documented in the prior literature.[18] Finally, Panel C of Figure 2 reports estimates from a specification that models temperature as a third-order polynomial. This model again yields the familiar U-shaped relationship, and allows us to analyze effects of temperature along every point of the temperature distribution including the most extreme temperatures.

[16] Because the Bailey and Goodman-Bacon (2015) model is estimated at the annual level, the coefficients from their paper were divided by 12 to scale the coefficients down to the monthly level.

[17] The differences between our analyses and those of Barreca et al. (2016) include the following. Our sample is 1959-1988 while the most-comparable Barreca et al. (2016) sample is 1960-2004. We use only <40°F and >80°F, whereas Barreca et al. (2016) use all 10°F bins between <10°F and >90°F. Finally, our outcome is the age-adjusted mortality rate in levels (at the county level), whereas Barreca et al. (2016) use the log crude mortality rate (at the state level).

[18] The estimate for the coefficient on $Temp^{<40}$ in this model with five bins is larger compared to the model with only the extreme bins in Table 2. The larger coefficient estimate in the five-bin model owes to the fact that the omitted group for the model in Table 2 includes all days with mean temperatures in the range of 40-80°F. Since temperatures in the 40-60°F range negatively impact mortality relative to the 60-70°F temperature bin, the coefficient estimate for $Temp^{<40}$ is attenuated when these days are included in the reference group. There is little difference in the coefficient estimate for $Temp^{>80}$ between the two models. For the interaction models, these results imply that a model including only the extreme temperature bins may provide conservative estimates for the interaction between CHCs and cold temperatures. While our preferred specification for the interaction model includes only the extreme temperature bins to facilitate interpretability, we also estimate interaction models with all five bins.

4.2 Interaction Model

Table 3 presents estimates of a basic version of the interaction models. Specifically, in this table temperature is modeled using two temperature bins ($\text{Temp}^{<40}$ and $\text{Temp}^{>80}$), and CHC access is modelled using a single post-treatment dummy (as opposed to a series of event-study dummies). The columns vary the controls that are included in the model, though all models include the same set of fixed effects and covariates that were included in the replication models. Column 1 is the simplest specification that includes all of the controls necessary for identifying the interaction effect: it allows for time-invariant differences in the effects of temperature across CHC and non-CHC counties ($g(\text{Temp}) \times \text{Treated}_c$) and for location-invariant differences in the effects of temperature over time ($g(\text{Temp}) \times \delta_y$). Column 2 allows for the effects of temperature to vary across AC penetration rates. Finally, Column 3 replaces the $g(\text{Temp}) \times \text{Treated}_c$ controls with the more general $g(\text{Temp}) \times \delta_c$ controls. These county-specific temperature effects represent over 3,000 additional parameters for each temperature variable, absorbing arbitrary fixed differences across all counties in the temperature-mortality relationship. Column 2 is our preferred specification as it includes all necessary controls while not asking as much of the data as the specification in Column 3. All interaction models include the main effects for both temperature and CHC access, however we present only the coefficients on the interactions for brevity.[19]

The coefficient on the $\text{CHC}^{t\geq 0} \times \text{Temp}^{<40}$ interaction represents the change in the effect of cold temperatures on mortality that can be attributed to CHC access. Across all three specifications, we find no evidence that CHC access has a significant impact on the cold-mortality relationship. We next turn to hot temperatures. The coefficient estimate on $\text{CHC}^{t\geq 0} \times \text{Temp}^{>80}$ in Column 1 yields a negative and statistically significant interaction term. The coefficient estimate changes little when AC interactions are included in Column 2. Finally, the magnitude of the estimate is also insensitive to inclusion of county-specific temperature effects in Column 3, though the standard errors increase substantially (nevertheless, the estimate remains significant at the 10% level).

The coefficient estimate in our preferred specification in Column 2 is -0.052; this represents the causal effect of CHC access on the heat-mortality relationship, and the negative sign indicates that CHC access mitigates the temperature-mortality relationship. In levels, the coefficient implies that CHC access reduces the effect of one day above 80°F on mortality by 0.052 deaths per 100,000 population. In relative terms, the estimate implies that CHC

[19] Furthermore, while the direct effects for CHC access in the interaction models have a meaningful interpretation, the interpretation of the direct effects for temperature are obscured by the controls that include temperature interactions (e.g., $g(\text{Temp}) \times \text{Treated}_c$ and $g(\text{Temp}) \times \delta_y$). In any case, these additional estimates are available upon request.

access mitigates the harmful effect of heat on mortality by 15.3%, measured relative to the heat-mortality relationship in CHC counties in the pre-CHC period.[20] It is worth noting that while we find no statistically significant evidence of mitigation for the effects of cold temperatures, our 95% confidence intervals cannot rule out implied mitigation impacts of up to 10.8%.

The estimates in Table 3 provide a relatively simple and straightforward interpretation. That being said, more flexible modelling of either temperature or the effects of CHC access can provide deeper insight. In Figure 3 we model temperature as a third-order polynomial; this requires estimating only one more parameter in comparison to the model from Table 3, but allows us to analyze the effects of CHC access on the temperature-mortality relationship across the entire temperature distribution rather than relying on specific cut points at 40°F and 80°F. The estimates presented in Figure 3 demonstrate again that CHC access significantly mitigates the heat-mortality relationship, and further, they show that the mitigating effects grow stronger (in absolute terms) at the extreme high end of the temperature distribution. Similar to Table 3, Figure 3 provides no evidence that CHC access effectively mitigates the cold-mortality relationship.

In Figure 4, we present estimates from a specification that allows for dynamic CHC treatment effects with a binned event-study framework.[21] Time relative to treatment is measured in 5-year bins, and the year prior to CHC establishment ($t = -1$) is the omitted category and so all dynamic effects are measured relative to that year. This specification follows Bailey and Goodman-Bacon (2015). In both the top and bottom panels of Figure 4, the first coefficient estimates ($\text{CHC}^{t<-1} \times \text{Temp}^{<40}$ and $\text{CHC}^{t<-1} \times \text{Temp}^{\geq 80}$) measure the "effect" of CHC access on the cold- and heat-mortality relationships in all periods more than one year before CHC establishment, relative to one year prior. These estimates therefore represent tests for differential trends in the temperature-mortality relationship prior to CHC establishment. If our identification strategy is valid, we expect the estimates of these coefficients to be near zero. Reassuringly, the estimates are small in magnitude and statistically insignificant for both cold and hot temperatures. The estimates for the three post-treatment interactions with <40°F days again yield no statistically significant evidence of an effect of CHCs on the cold-mortality relationship. The estimates for the post-treatment interactions with >80°F days are all negative (implying mitigation) and increase slightly in magnitude with years since establishment.

[20] To calculate the temperature-mortality relationship in CHC counties for the pre-CHC period, we limit the sample to only CHC counties in 1959-1964, and estimate the a model equivalent to the one presented in Column 2 of Table 2. The coefficient on $\text{Temp}^{>80}$ equals 0.339 (s.e.=0.070), and the coefficient on $\text{Temp}^{<40}$ equals 0.241 (s.e.=0.081).

[21] See Table A2 for these coefficient estimates.

The binned event-study approach in Figure 4 groups together years relative to CHC establishment to maximize the power of the dynamic estimates. As a final way of presenting these results, we estimate a full year-by-year event study (see Equation (3) in the Appendix for a full description of the specification) and present the results in Figure 5. These estimates are consistent with our other results. For cold temperatures, we again find evidence of neither differential pre-treatment trends in the cold-mortality relationship nor of a treatment effect of CHC establishment. Indeed, none of the 24 coefficient estimates are statistically different from zero. For hot temperatures, we also find no evidence of differential pre-treatment trends in the heat-mortality relationship: none of the eight pre-treatment coefficients are statistically different from zero. The effect of CHCs on the heat-mortality relationship emerges shortly after CHC establishment: the first statistically significant decline in the heat-mortally relationship comes in the third year after establishment ($t = 2$). In total, all 16 of the post-treatment estimates are negative and 11 are statistically significant at the 5% level.

4.3 Additional Results and Robustness Checks

Consider a series of robustness checks and additional results presented in Tables A3 to A5. Table A3 breaks out our estimates of the interaction effects by age and cause of death, following the categories used in Bailey and Goodman-Bacon (2015). For reference, we also include the baseline (pre-1965) effects of temperature on mortality for each group (for each group, the percent mitigation can be calculated as the interaction estimate divided by the baseline temperature estimate). In general, these estimates align with our expectations: we find the largest interaction effects among the age groups and causes of death that are most highly affected by both high temperatures and CHC access: individuals aged 50+, and deaths due to cardiovascular and cerebrovascular disease. Some caution should be taken in interpreting the interaction estimates by cause of death, which are imprecisely estimated. Combined with the main results, these estimates imply that access to health care can mitigate environmental health damages only if there is sufficient overlap in the populations and diseases affected by both the source of access to care and the environmental shock. Such overlap exists in our setting, and the overlap is particularly strong for hot temperatures.

Next, consider a test of whether the estimates are sensitive to the choice of counties that serve as the control group (i.e., non-CHC counties). To construct a control group that is more comparable to the treatment group, we follow Crump et al. (2009) and trim the sample based on the propensity of establishing a CHC. We construct two alternative propensity scores by

estimating a logit regression of a treatment indicator on various fixed county characteristics. The first is a "Standard" P-Score based on economic and demographic characteristics, and the second is a "Climate" P-Score based only on climatic variables.[22] With these propensity scores in hand, we then restrict the sample to counties with P-Scores in the following three ranges: [0.05,0.95], [0.1,0.9], and [0.2,0.8]. Note that [0.1,0.9] is the range suggested by Crump et al. (2009). The results of this exercise are presented in Table A4. Trimming using the "Standard" P-Scores dramatically limits the sample; for example, the [0.1,0.9] trimmed sample consists of 326 counties instead of 3,041 included in the main specification. Because climate is much less useful for predicting CHC establishment, samples trimmed based on the "Climate" P-Scores do not limit the sample as drastically. Reassuringly, the point estimates for the >80°F interactions are similar across all of the various samples, although estimates with the smaller samples are considerably less precise. Note that the large control group used in the main specification is beneficial along several dimensions: (1) a large control group makes for more 2X2 DiD comparisons that are un-confounded by prior treatment (Goodman-Bacon, 2018a), (2) a large control group allows for separate identification of the effects of time and time relative to treatment (Borusyak and Jaravel, 2017), and (3) in our specific setting, the large control group also contributes to the identification of the temperature effects.

Consider next a specification that models temperature bins more flexibly. Our main specification that utilizes temperature bins (i.e., in Table 3) only uses two bins, meaning days with temperatures in the wide 40-80°F range serve as the reference group. Table A5 shows that the results are qualitatively unchanged when we include three additional bins (40-50, 50-60, 70-80) such that days in the more narrow 60-70°F range represent the omitted category.

Finally, we consider a model that includes lagged measures of temperature. Note that – for the sake of simplicity – our main specifications include only contemporaneous monthly temperature measures. If there are delayed impacts or temporal displacement (i.e., harvesting) in the effects of temperature exposure on mortality, then our estimates may not fully capture the effects of interest. In the specifications presented in Table A6, we additionally

[22]The "Standard" P-Scores is calculated using the following variables (measured in 1960 unless otherwise noted): population density, population density squared, 1950-1960 % population growth, % nonwhite, % aged 0-4, % aged 21+, % aged 65+, % urban, % rural, 1959 % with income under $3,000, 1959 % with income over $10,000, % less than four years schooling, % 12 or more years schooling, % in labor force, unemployment rate, percent male in labor force, housing units per 1,000 population, % renting, % households with plumbing, % households with TV, % households with telephone, % households with automobile, median number of rooms, hospitals per 1,000 population, MDs per 1,000 population, and 1957 local government expenditure per 1,000 population. The "Climate" P-Scores is calculated using the following variables: mean temperature, mean days in the following bins: <20°F, 20-30°F, 30-40°F, 40-50°F, 50-60°F, 70-80°F, 80-90°F, >90°F, and mean precipitation.

include a one-month lag for each temperature variable, and report the sums of the coefficients on the contemporaneous and lagged temperature variables. The main results are qualitatively unchanged.

5 Discussion and Conclusion

This paper leverages two distinct sources of quasi-random variation in order to causally identify the impacts of an increase in health care access on temperature-induced mortality. We find that the county-level establishment of Community Health Centers mitigated the harmful relationship between high temperatures and general mortality rates by approximately 15%. We find no statistically significant evidence that CHCs affected cold-induced mortality.

To understand why CHCs might mitigate negative health effects of high temperatures but not low temperatures, we must understand the interplay between the specific services provided by CHCs and the channels through which extreme temperatures impact health. CHCs were focused on providing preventive care to poor and uninsured populations, and were successful in preventing deaths primarily due to cardiovascular and cerebrovascular diseases (Bailey and Goodman-Bacon, 2015). While both low and high temperatures are associated with increased mortality rates (see Figure 2), heat-induced mortality is more concentrated in the same causes of deaths that CHCs were effective at preventing. In other words, the preventative care provided by CHCs was particularly relevant for heat-induced health events.

The ameliorative effect of CHC access on the heat-mortality relationship suggests that expanding health care access may be an effective approach to mitigating the health damages of a warmer climate. However, the asymmetric responsiveness of temperature health damages to CHC openings implies a nuanced interplay between the channels of environmental harm and the health care services being provided. Taken together, these results imply that, to be effective, the specific dimension of health care for which access is improved must address the types of ailments triggered by the environmental shock of interest. As mentioned above, this appears to be the case in our setting for high temperatures but not for cold temperatures. This insight is crucial when considering both current inequities in environmental health damages and climate change adaptation, as it suggests that expanding health care access will be an effective approach to reducing the harmful effects of higher temperatures (or other adverse environmental conditions) only insofar as the mode of health care can be reasonably well-targeted.

Finally, access to health care varies dramatically both within and between countries. Given this reality, our results provide a clear, causal pathway through which heterogeneity

in environmental damages can be partially explained. Thus, even as our findings demonstrate how improvements in access to care can reduce the harm from specific environmental exposures, they underscore how existing differences in access to specific domains of care are likely contributing to widespread inequality in the incidence of environmental health damages.

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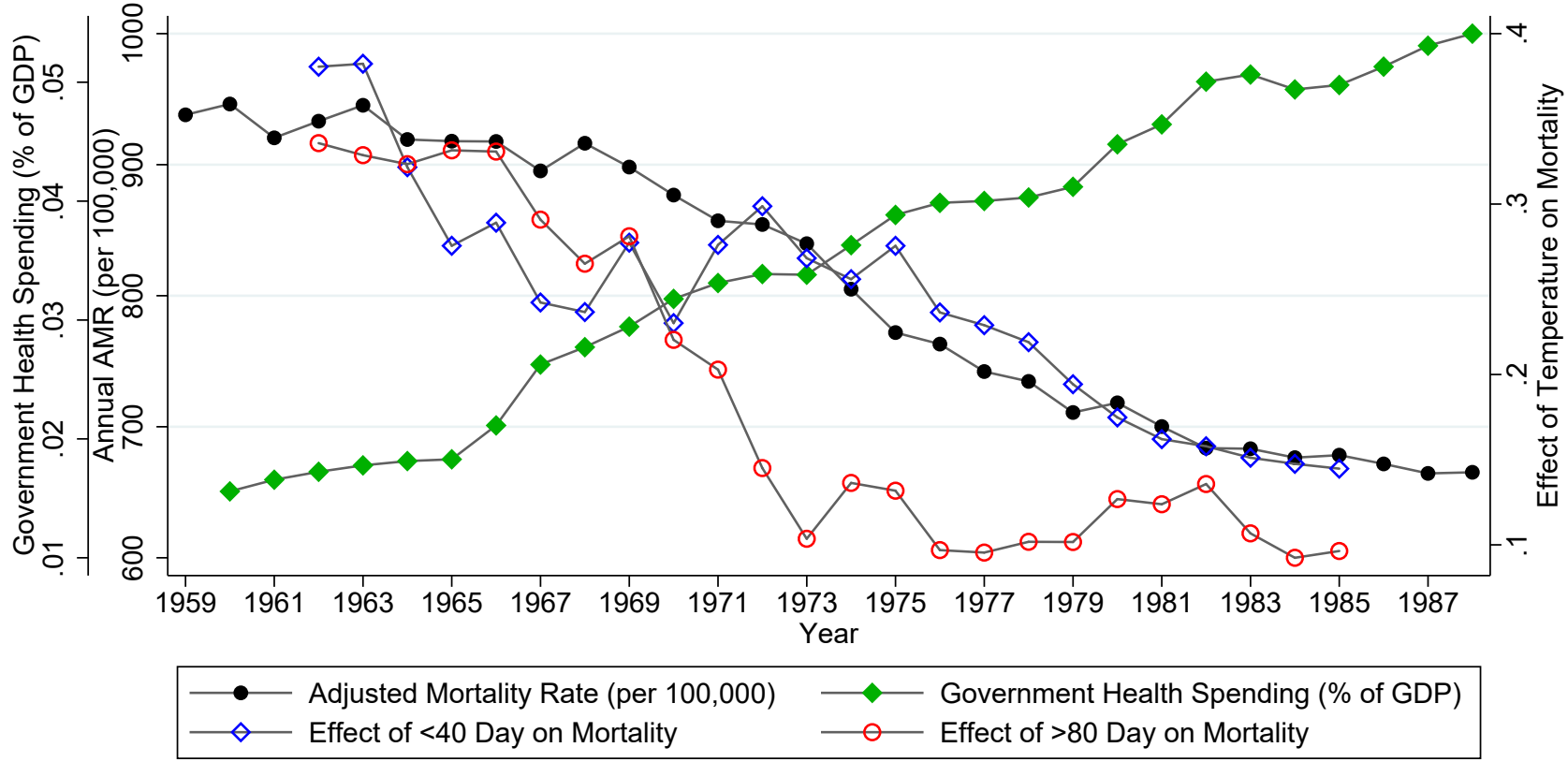
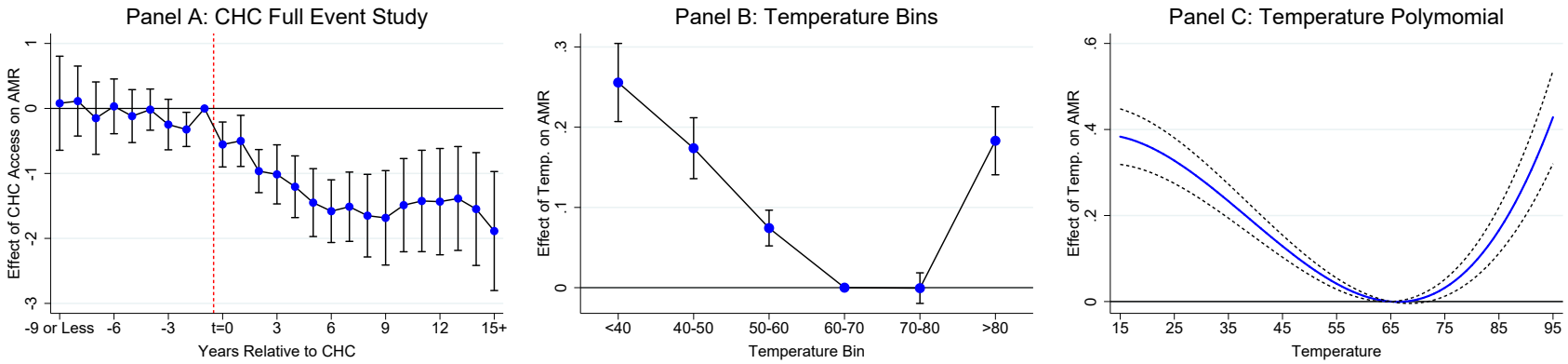
Figure 1: Trends in Mortality, Government Health Spending, and $\frac{\partial \text{Mortality}}{\partial \text{Temperature}}$



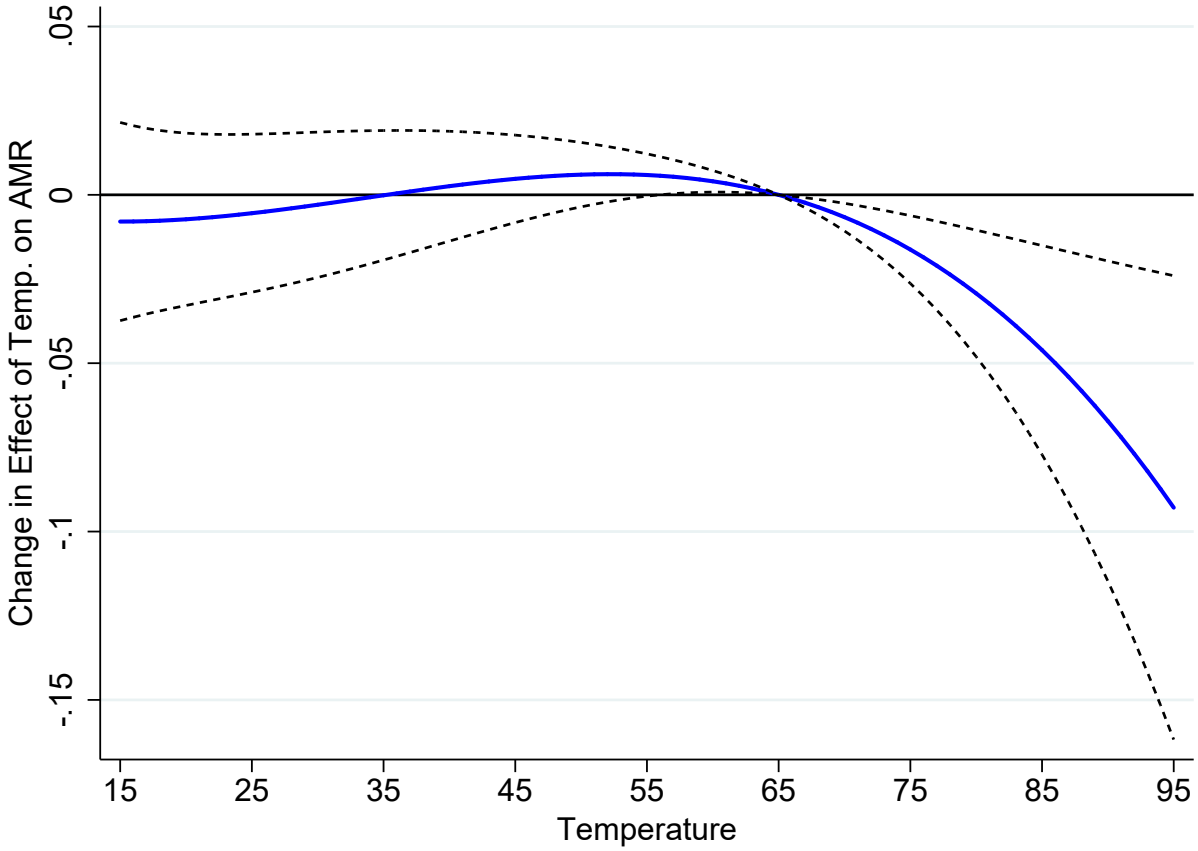
Notes: Coefficient estimates for the effects of temperature and mortality are from models described in Section 3.2.1 with five temperature bins and the sample limited to a 7 year period centered around the year in question. For example, the sample for the point labelled "1962" is 1959-1965. All of the estimates are significant at the 5% level; interpretation of the magnitudes of similar estimates is given in Section 4.1. Data on government health expenditures are from the National Health Expenditure Accounts, and include government spending on health insurance for the Department of Defense and the Department of Veterans Affairs, CMS programs (Medicaid and Medicare), government public health expenditures (including the CHC program), government health investments, and other programs. The age-adjusted mortality rate is calculated at the annual level for the entire US population.

Figure 2: Replication Models – Flexible Specifications



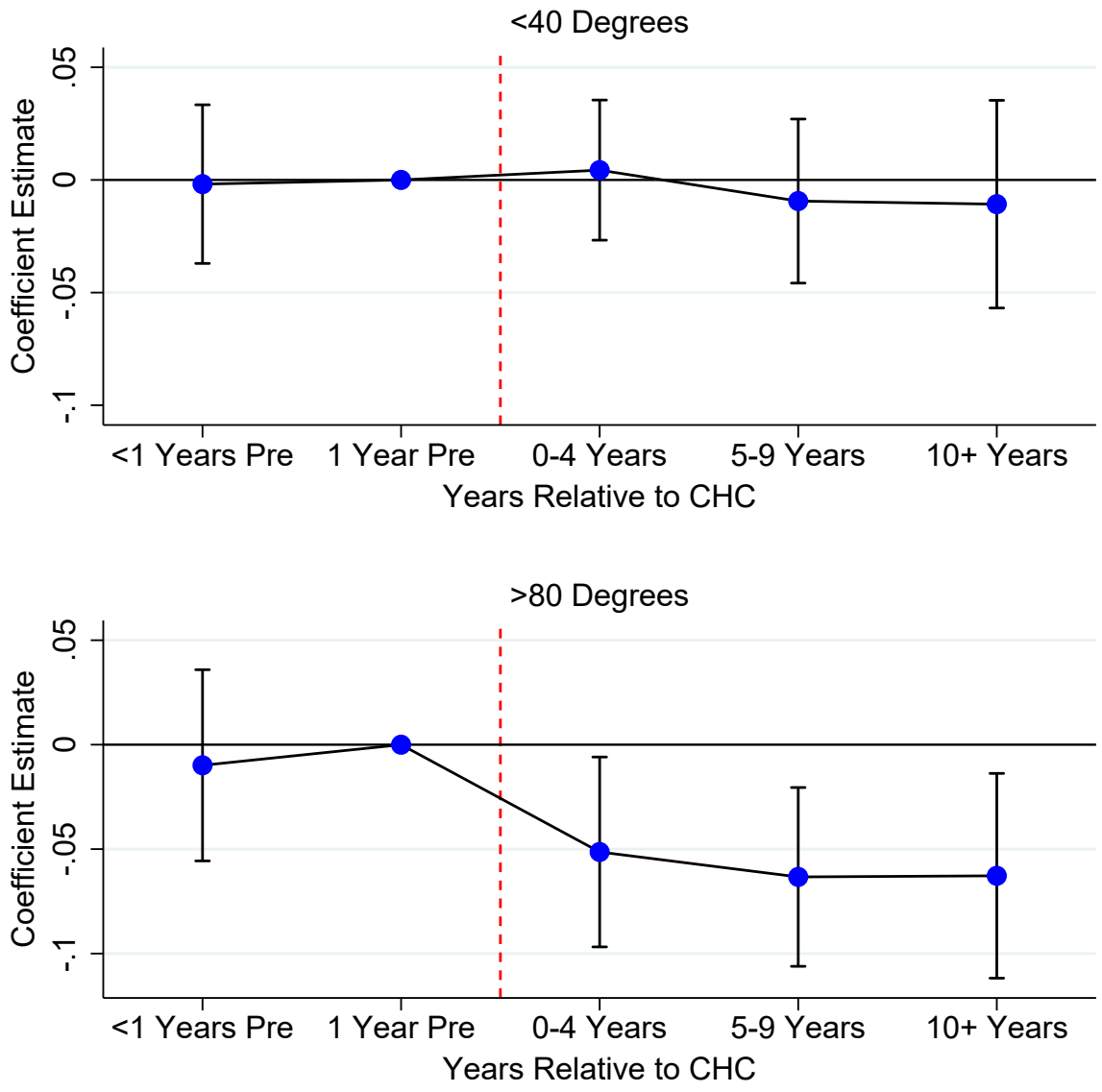
Notes: In Panel A, period $t = 0$ represents the year in which a CHC was established in a given county. A balanced panel of counties identify all event study coefficients between t-6 and t+14 (i.e., the same number of counties identify these coefficients). In Panel B, variables measuring the number of days in each of five 10°F temperature bins are included (60-70°F days are excluded as the reference group). Panel C allows for analysis across the entire temperature distribution in a parsimonious manner following (Carleton et al., 2018). These regressions include a third-order polynomial in temperature, where each polynomial term is constructed at the daily level and then summed over months. Each point on the plot represents a test of the hypothesis that the effect of a single day at the given temperature is equal to the effect of a day with temperature equal to 65°F (i.e., the interpretation is analogous to the interpretation of the coefficients plotted in Panel B). Bars and dashed lines represent 95% confidence intervals.

Figure 3: Interaction Model – Temperature Polynomial



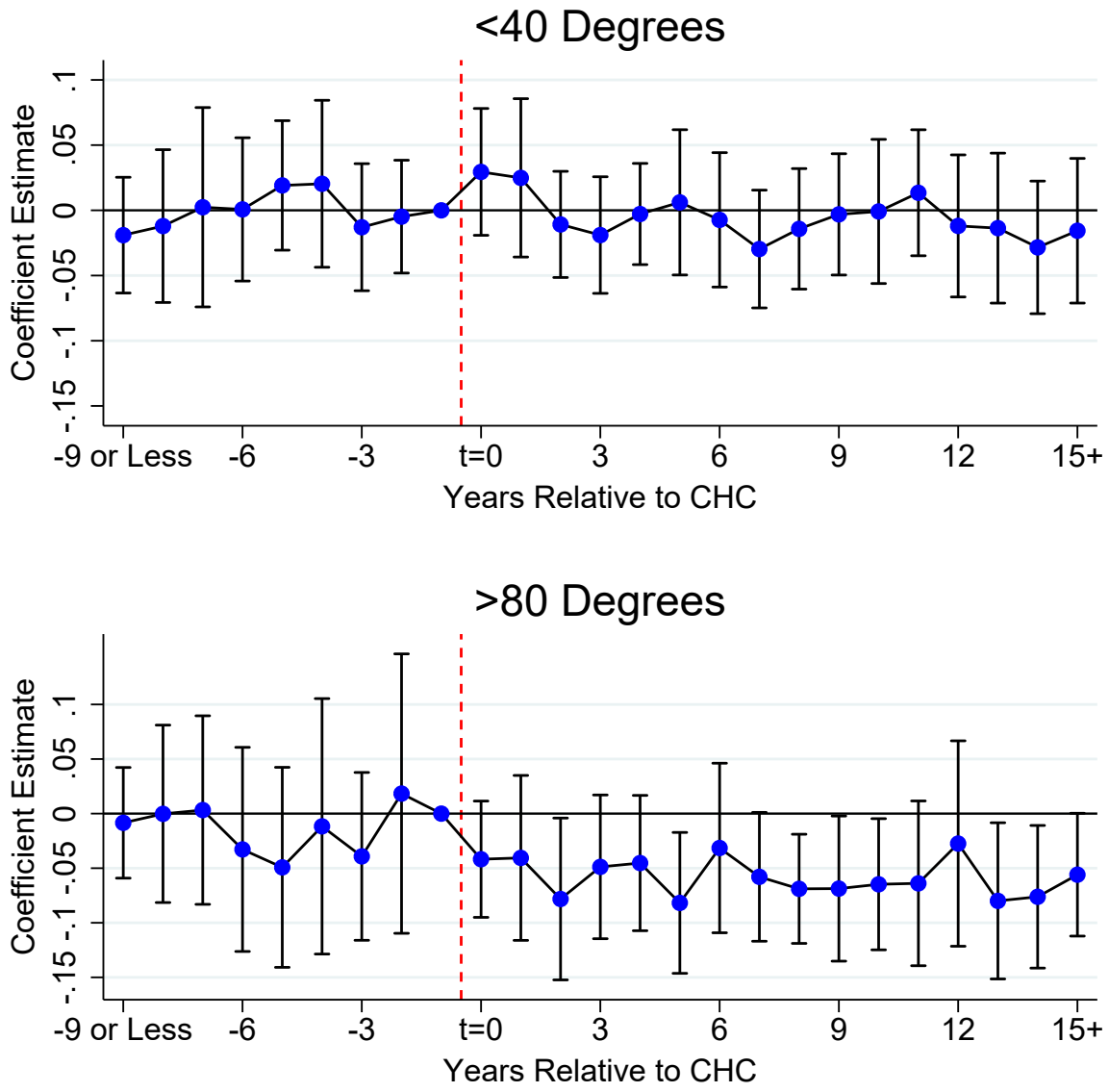
Notes: These estimates represent the effect of CHC access on the temperature-mortality relationship (i.e., the estimates of the interaction are plotted). The estimates are derived from a model equivalent to Column 2 of Table 3, but with a third-order polynomial in place of the two temperature bins. Each point represents a test of whether CHC access changes the impact of a day at the given temperature relative to a day with temperature equal to 65°F. Dashed lines represent 95% confidence intervals.

Figure 4: Binned Event Study for the Effects of CHC Access on $\frac{\partial \text{Mortality}}{\partial \text{Temp}}$



Notes: All estimates in both panels are derived from a single regression. The estimates are derived from a model equivalent to Column 2 of Table 3, but with indicators for time relative to CHC access in place of the single post-CHC indicator. Period $t = 0$ represents the first year in which a CHC was established in a given county; year $t = -1$ is omitted as the reference group. Bars represent 95% confidence intervals.

Figure 5: Full Event Study for the Effects of CHC Access on $\frac{\partial \text{Mortality}}{\partial \text{Temp}}$



Notes: All estimates in both panels are derived from a single regression. Period $t = 0$ represents the first year in which a CHC was established in a given county. A balanced panel of counties identify all event study coefficients between t-6 and t+14 (i.e., the same number of counties identify these coefficients). Bars represent 95% confidence intervals.

Table 1: Summary Statistics

	All Counties All Years 1959-1988		CHC Counties Pre-CHC Years 1959-1964		Non-CHC Counties Pre-CHC Years 1959-1964	
	Mean	S.D.	Mean	S.D.	Mean	S.D.
AMR	68.3	(16.46)	81.7	(11.17)	76.75	(17.53)
Infant MR	146.9	(146.8)	212.5	(61.42)	210.21	(177.74)
AMR Age 1-14	14.93	(12.97)	21.51	(6.28)	5.13	(7.56)
AMR Age 15-49	18.34	(11.17)	20.58	(5.52)	19.41	(12.7)
AMR Age 50+	237.3	(56.4)	282.03	(40.68)	264.02	(64.27)
AMR Heart Dis.	26.31	(9.01)	33.41	(6.39)	31.44	(10.42)
AMR Cerebro. Dis.	7.38	(4.49)	9.83	(2.63)	10.05	(5.63)
AMR Cancer	12.89	(4.75)	13.34	(2.29)	11.82	(5.5)
AMR Infectious Dis.	3.34	(2.83)	3.57	(1.83)	2.98	(3.31)
AMR Diabetes	1.27	(1.52)	1.5	(0.81)	1.37	(1.88)
AMR Accidents	4.10	(3.93)	3.88	(1.53)	4.57	(4.53)
Temperature (°F)	54.5	(17.28)	54.87	(16.88)	54.29	(17.69)
# Days <40°F	7.08	(10.44)	6.73	(10.36)	7.45	(10.72)
# Days 40-50°F	4.61	(5.74)	4.58	(5.82)	4.48	(5.60)
# Days 50-60°F	5.26	(6.05)	5.61	(6.63)	4.88	(5.67)
# Days 60-70°F	5.99	(6.7)	6.16	(6.92)	5.84	(6.5)
# Days 70-80°F	5.72	(8.10)	5.65	(8.04)	6.06	(8.23)
# Days ≥ 80°F	1.77	(5.31)	1.72	(5.25)	1.74	(5.18)
Precipitation (mm)	83.76	(56.15)	75.43	(53.54)	81.77	(54.53)
AC (1959-1964)	0.13	(0.07)	0.13	(0.07)	0.13	(0.07)
AC (1965-1988)	0.44	(0.28)	0.41	(0.26)	0.45	(0.28)
Counties	3,041		114		2,927	

Notes: All summary statistics represent monthly averages for counties included in our analytic sample. AMR represents the age-adjusted mortality rate per 100,000 population. AC is the air conditioning penetration rate measured at the state level. Summary statistics are weighted by the county's 1960 population.

Table 2: Effects of CHC Access and Temperature on Mortality

	(1)	(2)	(3)	(4)	(5)
$\text{CHC}^{t\geq 0}$	-1.136		-1.146		
	(0.307)		(0.307)		
$\text{CHC}^{t\leq -2}$				-0.0976	-0.102
				(0.168)	(0.168)
$\text{CHC}^{0\leq t\leq 4}$				-0.836	-0.850
				(0.157)	(0.158)
$\text{CHC}^{5\leq t\leq 9}$				-1.554	-1.566
				(0.271)	(0.270)
$\text{CHC}^{t\geq 10}$				-1.562	-1.578
				(0.390)	(0.390)
$\text{Temp}^{<40}$		0.116	0.116		0.116
		(0.0159)	(0.0158)		(0.0158)
$\text{Temp}^{\geq 80}$		0.182	0.183		0.183
		(0.0187)	(0.0187)		(0.0187)
N	1,094,760	1,094,760	1,094,760	1,094,760	1,094,760

Notes: Estimates from each column are from a single regression. The covariates and fixed effects described in Equation (1) are included in all specifications. Standard errors in parentheses are two-way clustered at the county and year-month levels.

Table 3: Effects of CHC Access on the Temperature-Mortality Relationship

	(1)	(2)	(3)
$\text{CHC}^{t\geq 0} \times \text{Temp}^{<40}$	-0.00294	-0.00346	-0.00420
	(0.0114)	(0.0115)	(0.0157)
$\text{CHC}^{t\geq 0} \times \text{Temp}^{\geq 80}$	-0.0484	-0.0518	-0.0477
	(0.0201)	(0.0197)	(0.0286)
N	1,094,760	1,094,760	1,094,760
Temp × Treated	X	X	
Temp × δ_y	X	X	X
Temp × AC		X	X
Temp × δ_c			X

Notes: Each column reports coefficient estimates from a single regression. The main effects for temperature and CHC access are included in all specifications. The interacted temperature controls represent controls for all temperature variables included in the model; for example, in Panel C models including "Temp × Treated" include both $\text{Temp}^{<40}$ × Treated and $\text{Temp}^{>80}$ × Treated. Standard errors are two-way clustered at the county and year-by-month level. For reference, the baseline estimates for CHC counties in the pre-CHC period (1959-1964) for the effect of a <40 and >80 day are 0.241 (s.e.=0.081) and 0.339 (s.e.=0.070), respectively.

Appendix

Table A1: Association between CHCs and Temperature Shocks

	$\text{Temp}^{>80}$	$\text{Temp}^{<40}$	CHC	CHC	CHC
CHC	0.0192	0.0229			
	(0.0279)	(0.0392)			
$\text{Temp}^{<40}$			0.0000466		0.0000328
			(0.0000793)		(0.0000978)
$\text{Temp}^{>80}$				0.0000626	0.0000472
				(0.0000897)	(0.000114)
N	1,094,760	1,094,760	1,094,760	1,094,760	1,094,760

Notes: Column labels denote the outcome variable of each regression. "CHC" represents an indicator for whether a CHC was in place in the given county and year. All models include county and year-month fixed effects. Standard errors are clustered at the county level.

Table A2: Effects of CHC Access on the Temperature-Mortality Relationship – Binned Event Study Estimates

	(1)	(2)	(3)
$\text{CHC}^{t\leq -2} \times \text{Temp}^{<40}$	-0.00207	-0.00186	-0.00286
	(0.0179)	(0.0179)	(0.0247)
$\text{CHC}^{0\leq t\leq 4} \times \text{Temp}^{<40}$	0.00452	0.00436	0.00329
	(0.0157)	(0.0158)	(0.0217)
$\text{CHC}^{5\leq t\leq 9} \times \text{Temp}^{<40}$	-0.00916	-0.00938	-0.0115
	(0.0185)	(0.0185)	(0.0259)
$\text{CHC}^{t\geq 10} \times \text{Temp}^{<40}$	-0.0102	-0.0108	-0.0126
	(0.0235)	(0.0234)	(0.0315)
$\text{CHC}^{t\leq -2} \times \text{Temp}^{\geq 80}$	-0.0116	-0.00987	-0.00703
	(0.0234)	(0.0233)	(0.0453)
$\text{CHC}^{0\leq t\leq 4} \times \text{Temp}^{\geq 80}$	-0.0506	-0.0514	-0.0463
	(0.0231)	(0.0231)	(0.0369)
$\text{CHC}^{5\leq t\leq 9} \times \text{Temp}^{\geq 80}$	-0.0627	-0.0633	-0.0520
	(0.0220)	(0.0218)	(0.0344)
$\text{CHC}^{t\geq 10} \times \text{Temp}^{\geq 80}$	-0.0597	-0.0628	-0.0579
	(0.0249)	(0.0249)	(0.0384)
N	1,094,760	1,094,760	1,094,760
Temp × Treated	X	X	
Temp × δ_y	X	X	X
Temp × AC		X	X
Temp × δ_c			X

Notes: Each column reports coefficient estimates from a single regression. The main effects for temperature and CHC access are included in all specifications. The interacted temperature controls represent controls for all temperature variables included in the model; for example, the models in Columns 1 and 2 include both $\text{Temp}^{<40} \times$ Treated and $\text{Temp}^{>80} \times$ Treated. Standard errors are two-way clustered at the county and year-by-month level. For reference, the baseline estimates for CHC counties in the pre-CHC period (1959-1964) for the effect of a <40 and >80 day are 0.241 (s.e.=0.081) and 0.339 (s.e.=0.070), respectively.

Table A3: Estimates By Age and Cause of Death

	Infant	1-14	15-49	50+		
Panel A1: Interaction Effects by Age						
$CHC^{t\geq 0} \times Temp^{<40}$	0.0723	0.00544	-0.000605	-0.0182		
	(0.0917)	(0.00752)	(0.00535)	(0.0493)		
$CHC^{t\geq 0} \times Temp^{\geq 80}$	-0.126	-0.00637	-0.00310	-0.243		
	(0.125)	(0.0130)	(0.0116)	(0.0783)		
Panel A2: Pre-1965 Temperature Effects by Age						
$Temp^{<40}$	0.346	0.00743	0.00725	0.696		
	(0.231)	(0.00884)	(0.0131)	(0.234)		
$Temp^{>80}$	0.274	0.0128	0.0405	1.306		
	(0.234)	(0.00890)	(0.0140)	(0.172)		
	Panel B1: Cause of Death					
	Heart Dis.	Cerebro. Dis.	Cancer	Infectious Dis.	Diabetes	Accidents
$CHC^{t\geq 0} \times Temp^{<40}$	-0.00454	0.000174	0.00114	-0.00714	-0.000576	-0.00280
	(0.00842)	(0.00309)	(0.00289)	(0.00271)	(0.000833)	(0.00195)
$CHC^{t\geq 0} \times Temp^{\geq 80}$	-0.0111	-0.0108	-0.00411	-0.00921	-0.00281	-0.00601
	(0.0134)	(0.00536)	(0.00561)	(0.00425)	(0.00146)	(0.00370)
Panel B2: Pre-1965 Temperature Effects by Cause						
$Temp^{<40}$	0.0835	0.0293	0.00651	0.0212	0.00360	0.00850
	(0.0272)	(0.00939)	(0.00562)	(0.0197)	(0.00290)	(0.00613)
$Temp^{>80}$	0.153	0.0728	0.0100	0.0321	0.00603	0.0151
	(0.0181)	(0.0106)	(0.00596)	(0.00739)	(0.00236)	(0.00576)

Notes: Panels A1 and B1 display the interaction effects by age and cause of death, respectively. For reference, panels A2 and B2 display the baseline (pre-1965) effects of temperature on mortality by age and cause of death. The interaction specification corresponds to Column 2 of Table 3. Standard errors are two-way clustered at the county and year-by-month level.

Table A4: CHC Interaction Model – Trimmed Samples

	(1)	(2)	(3)	(4)	(5)	(6)
	Panel A: Simple DiD					
$CHC^{t\geq 0} \times Temp^{<40}$	-0.000620	0.0174	0.0220	-0.00315	-0.00372	-0.000765
	(0.0135)	(0.0167)	(0.0247)	(0.0116)	(0.0115)	(0.0124)
$CHC^{t\geq 0} \times Temp^{\geq 80}$	-0.0343	-0.0330	-0.0239	-0.0598	-0.0584	-0.0568
	(0.0187)	(0.0225)	(0.0310)	(0.0199)	(0.0199)	(0.0206)
	Panel B: Binned Event Study					
$CHC^{t\leq -2} \times Temp^{<40}$	-0.0182	-0.0355	-0.0160	-0.000598	-0.00114	0.000762
	(0.0295)	(0.0185)	(0.0265)	(0.0203)	(0.0204)	(0.0220)
$CHC^{0\leq t\leq 4} \times Temp^{<40}$	-0.00911	-0.0319	-0.0117	0.00471	0.00445	0.00917
	(0.0242)	(0.0171)	(0.0329)	(0.0178)	(0.0179)	(0.0189)
$CHC^{5\leq t\leq 9} \times Temp^{<40}$	-0.0176	-0.00214	0.0226	-0.00735	-0.00834	-0.00408
	(0.0294)	(0.0204)	(0.0332)	(0.0208)	(0.0209)	(0.0232)
$CHC^{t\geq 10} \times Temp^{<40}$	-0.0233	-0.00752	0.0131	-0.00881	-0.0105	-0.00562
	(0.0368)	(0.0224)	(0.0314)	(0.0265)	(0.0264)	(0.0292)
$CHC^{t\leq -2} \times Temp^{\geq 80}$	-0.0247	-0.0262	-0.0286	-0.0101	-0.0106	-0.0163
	(0.0231)	(0.0404)	(0.0538)	(0.0233)	(0.0233)	(0.0239)
$CHC^{0\leq t\leq 4} \times Temp^{\geq 80}$	-0.0528	-0.0532	-0.0411	-0.0536	-0.0532	-0.0532
	(0.0227)	(0.0420)	(0.0579)	(0.0227)	(0.0226)	(0.0228)
$CHC^{5\leq t\leq 9} \times Temp^{\geq 80}$	-0.0567	-0.0504	-0.0413	-0.0721	-0.0714	-0.0760
	(0.0220)	(0.0371)	(0.0558)	(0.0209)	(0.0209)	(0.0211)
$CHC^{t\geq 10} \times Temp^{\geq 80}$	-0.0566	-0.0599	-0.0589	-0.0741	-0.0728	-0.0777
	(0.0248)	(0.0367)	(0.0580)	(0.0249)	(0.0249)	(0.0253)
N	210,600	117,720	56,160	1,093,680	1,078,920	894,240
Standard P-Score	X	X	X			
Climate P-Score				X	X	X
P-Score Range	[0.05,0.95]	[0.1,0.9]	[0.2,0.8]	[0.05,0.95]	[0.1,0.9]	[0.2,0.8]

Notes: This table replicates the findings from Column 2 of table Table 3, with samples limited to counties within the given propensity score range. Standard errors are two-way clustered at the county and year-by-month level.

Table A5: CHC Interaction Model – All Temperature Bins

	(1)	(2)
	Panel A: Simple DiD	
$CHC^{t\geq 0} \times Temp^{<40}$	0.00573	-0.0177
	(0.0175)	(0.0152)
$CHC^{t\geq 0} \times Temp^{\geq 80}$	-0.0657	-0.0536
	(0.0230)	(0.0221)
	Panel B: Binned Event Study	
$CHC^{t\leq -2} \times Temp^{<40}$	-0.0192	0.00493
	(0.0367)	(0.0338)
$CHC^{0\leq t\leq 4} \times Temp^{<40}$	0.00431	0.00388
	(0.0330)	(0.0295)
$CHC^{5\leq t\leq 9} \times Temp^{<40}$	-0.0172	-0.0131
	(0.0375)	(0.0341)
$CHC^{t\geq 10} \times Temp^{<40}$	-0.0200	-0.0281
	(0.0418)	(0.0394)
$CHC^{t\leq -2} \times Temp^{\geq 80}$	-0.00403	0.00443
	(0.0336)	(0.0309)
$CHC^{0\leq t\leq 4} \times Temp^{\geq 80}$	-0.0631	-0.0389
	(0.0315)	(0.0272)
$CHC^{5\leq t\leq 9} \times Temp^{\geq 80}$	-0.0689	-0.0487
	(0.0356)	(0.0299)
$CHC^{t\geq 10} \times Temp^{\geq 80}$	-0.0724	-0.0556
	(0.0357)	(0.0303)
N	1,094,760	1,094,760
40-50, 50-60, 70-80 Bins & Interactions	X	X
Temp × Treated Controls	X	X
Temp × δ_y Controls	X	X
Temp × AC Controls		X

Notes: In addition to the <40°F and >80°F temperature bins included in the main specifications, temperature variables with counts of days with mean temperatures 40-50°F, 50-60°F, and 70-80°F are included as well. All relevant interactions are also included for each temperature bin (i.e., the CHC interactions in all specifications and the additional interactions depending on the column). The 60-70°F range is the omitted group. No specification with Temp × δ_c controls is estimated; while it was computationally possible to estimate such a model with two temperature bins, doing so with five temperature bins is infeasible as this would require estimating over 15,000 parameters. Standard errors are two-way clustered at the county and year-by-month level.

Table A6: CHC Interaction Model – Including Lags in Temperature

	(1)	(2)
	Panel A: Simple DiD	
$\text{CHC}^{t\geq 0} \times \text{Temp}_t^{<40} + \text{CHC}^{t\geq 0} \times \text{Temp}_{t-1}^{<40}$	0.0009	0.0008
	(0.0126)	(0.0128)
$\text{CHC}^{t\geq 0} \times \text{Temp}_t^{\geq 80} + \text{CHC}^{t\geq 0} \times \text{Temp}_{t-1}^{\geq 80}$	-0.0549	-0.0565
	(0.0219)	(0.0217)
	Panel B: Binned Event Study	
$\text{CHC}^{t\leq -2} \times \text{Temp}_t^{<40} + \text{CHC}^{t\leq -2} \times \text{Temp}_{t-1}^{<40}$	0.0008	0.0009
	(0.0178)	(0.0178)
$\text{CHC}^{0\leq t\leq 4} \times \text{Temp}_t^{<40} + \text{CHC}^{0\leq t\leq 4} \times \text{Temp}_{t-1}^{<40}$	-0.0063	-0.0060
	(0.0294)	(0.0292)
$\text{CHC}^{5\leq t\leq 9} \times \text{Temp}_t^{<40} + \text{CHC}^{5\leq t\leq 9} \times \text{Temp}_{t-1}^{<40}$	0.0088	0.0087
	(0.0165)	(0.0165)
$\text{CHC}^{t\geq 10} \times \text{Temp}_t^{<40} + \text{CHC}^{t\geq 10} \times \text{Temp}_{t-1}^{<40}$	-0.0529	-0.0536
	(0.0270)	(0.0272)
$\text{CHC}^{t\leq -2} \times \text{Temp}_t^{\geq 80} + \text{CHC}^{t\leq -2} \times \text{Temp}_{t-1}^{\geq 80}$	0.0041	0.0040
	(0.0168)	(0.0169)
$\text{CHC}^{0\leq t\leq 4} \times \text{Temp}_t^{\geq 80} + \text{CHC}^{0\leq t\leq 4} \times \text{Temp}_{t-1}^{\geq 80}$	-0.0619	-0.0626
	(0.0267)	(0.0268)
$\text{CHC}^{5\leq t\leq 9} \times \text{Temp}_t^{\geq 80} + \text{CHC}^{5\leq t\leq 9} \times \text{Temp}_{t-1}^{\geq 80}$	-0.0059	-0.0061
	(0.0213)	(0.0214)
$\text{CHC}^{t\geq 10} \times \text{Temp}_t^{\geq 80} + \text{CHC}^{t\geq 10} \times \text{Temp}_{t-1}^{\geq 80}$	-0.0629	-0.0648
	(0.0303)	(0.0301)
N	1,091,719	1,091,719
40-50, 50-60, 70-80 Bins & Interactions	X	X
Temp × Treated Controls	X	X
Temp × δ_y Controls	X	X
Temp × AC Controls		X

Notes: In this specification, a one-month lag in temperature is included for each temperature variable. All relevant interactions are also included for each lagged bin (i.e., the CHC interactions in all specifications and the additional interactions depending on the column). The reported estimates represent the sums of the contemporaneous and lagged variables. The 60-70°F range is the omitted group. No specification with Temp × δ_c controls is estimated; while it was computationally possible to estimate such a model with two temperature bins, doing so with the lagged temperature bins as well is infeasible as this would require estimating over 12,000 parameters. Standard errors are two-way clustered at the county and year-by-month level.

Online Appendix

A.1 Data Details

A.1.1 Community Health Centers and Covariates

Data on the timing and location of CHC establishments, as well as data on all covariates used in Bailey and Goodman-Bacon (2015), were graciously shared by Martha Bailey and Andrew Goodman-Bacon. These data were painstakingly collected through a variety of archival sources including the National Archives Community Action Program, hand-entered Public Health Service Reports, and other primary sources. For all CHCs established in 1965-1974, these data indicate the county in which services are provided, and the year in which the county received its first CHC services grant (as opposed to planning grants). For the purposes of this paper, it is only important that this data provide accurate information on the year and location in which CHC services were first offered. CHC establishments are coded as beginning in January of the relevant year. We refer readers to Bailey and Goodman-Bacon (2015) for more detail on the data collection.

A.1.2 Mortality Rates

Mortality data are derived from the 1959-1988 National Vital Statistics System (NVSS) mortality files maintained by the National Center for Health Statistics (NCHS). For years through 1988, these files are publicly available with county identifiers. We use the a crosswalk between NCHS county codes and FIPS county codes to deal with changes in county coding over time (ICPSR 36603). The NVSS files contain individual-level information on all deaths in the US. Deaths are matched to weather and CHC data based on the year, month, and county of occurrence.

The primary outcome of interest is the age-adjusted mortality rate per 100,000 population. Annual county-level population data by 5-year age groups for the period 1969-2016 are obtained from the Surveillance, Epidemiology, and End Results Program (SEER). Because these data are only available for the period 1969 and beyond, we also use data from the U.S. Census Bureau on county-level population in 1950 and 1960; population data are linearly interpolated for the missing years between 1950 and 1969.

The main outcome of interest is the age-adjusted mortality rate (AMR). Age-adjusted mortality rates hold fixed the age distribution of the population of a given county such that changes in the AMR reflect changes in the risk of death rather than changes in the age structure. In particular, the AMR for county c at time t is calculated as a weighted average of age-specific mortality rates (ASMR) for county c at time t and 5-year age group

a. $ASMR_{cta} = 100,000 \times \frac{Deaths_{cta}}{Pop_{cta}}$; $AMR_{ct} = \sum_{a=1}^{18} s_{ca} \times ASMR_{cta}$, where s_{ca} is the 1960 share of the population in 5-year age group a. Age-adjusting refers to holding the population age share s_{ca} fixed.

A.1.3 Weather

The assignment of local weather conditions to population groups is central to our empirical investigation. Our main data source on weather is derived from the PRISM Climate Group (aggregated by Schlenker and Roberts, 2009). This contains daily data on temperature and precipitation for points on a 2.5-by-2.5 mile grid for the U.S. over the period 1959-1988. We aggregate the data to the county level by taking a weighted average of daily temperature and precipitation for all grid points within a county, where the values from each grid point are weighted by the inverse of the squared distance from the grid point to the county's population centroid. Our main temperature variable of interest is the daily mean temperature (the mean of the minimum and maximum temperature). Daily mean temperatures are grouped into 10°F-wide bins, ranging from <40°F to >80°F. The numbers of days in each temperature bin are summed for each county-month in the sample. The independent variables of interest are therefore counts of days for which a given county had a mean temperature in each bin in a given month and year. Precipitation is measured as the monthly sum.

The third order polynomials in mean temperatures are constructed following Carleton et al. (2018). Specifically, we first calculate a third-order polynomial in temperature at the *daily* level, and then sum these three polynomial terms across the month. This approach allows us to leverage daily variation in local temperatures in models where the unit of observation is at the monthly level. Estimates are all interpreted as the effect of an additional day with a given mean temperature relative to an additional day at 65°F.

A.1.4 AC Data

We follow Barreca et al. (2016) in constructing our measure of AC penetration at the state-year level. Data on AC penetration are derived from the 1960, 1970, and 1980 Censuses. State-year AC penetration rates are interpolated between census years and extrapolated to the ends of the sample. Note that AC penetration rates are also extrapolated across months within the year to avoid discontinuous jumps at the beginning of each year.

A.2 Empirical Strategy Details

A.2.1 Generalized CHC Interaction Model

Please see the following for a generalized empirical model for identifying the interaction between CHC access and temperature. This model allows for J event-study indicators and G temperature bins.

$$\begin{aligned} \text{AMR}_{cym} = &\sum_{j=1}^{J}\sum_{g=1}^{G}\phi^{jg}(\text{CHC}_{cy}^{j} \times \text{Temp}_{cym}^{g}) + \sum_{j=1}^{J}\gamma^{j}\text{CHC}_{cy}^{j} + \sum_{g=1}^{G}\pi^{g}\text{Temp}_{cym}^{g} \\ &+ \sum_{g=1}^{G}\theta^{g}(\text{Temp}_{cym}^{g} \times \text{Treated}_{c}) + \sum_{g=1}^{G}\kappa^{g}(\text{Temp}_{cym}^{g} \times \text{AC}_{sy}) + \sum_{g=1}^{G}(\text{Temp}_{cym}^{g} \times \delta_{y}) \\ &\beta \text{X}_{cym} + \delta_{sy} + \delta_{cm} + \delta_{uy} + \delta_{ym} + \varepsilon_{cym} \end{aligned} \tag{3}$$

In practice, our primary specification includes the four event-study indicators ($\text{CHC}_{cy}^{t\leq -2}$, $\text{CHC}_{cy}^{0\leq t\leq 4}$, $\text{CHC}_{cy}^{5\leq t\leq 9}$, and $\text{CHC}_{cy}^{t\geq 10}$) used in Bailey and Goodman-Bacon (2015) and two temperature variables ($\text{Temp}_{cym}^{<40}$ and $\text{Temp}_{cym}^{>80}$) representing both cold and hot temperatures. In this specification, $J = 4$ and $G = 2$, and the estimates of the eight ϕ^{jg} coefficients are of primary interest. The interpretation of one of the ϕ^{jg} coefficients is similar to that of a standard event-study coefficient. For example, the interpretation of the coefficient on the interaction $\text{CHC}_{cy}^{0\leq t\leq 4} \times \text{Temp}_{cym}^{>80}$ is as follows: the difference in the effect of one additional day >80°F on the age-adjusted mortality rate between the year prior to CHC establishment and the period 0-4 years after. The coefficients on the pre-treatment interactions (e.g., $\text{CHC}_{cy}^{t\leq -2} \times \text{Temp}_{cym}^{>80}$) are expected to be near zero if no differential pre-treatment trends exist in the temperature-mortality relationship. In addition to the binned event study approach, we also estimate a full annual event study, with indicators for each year relative to treatment from $t - 9$ to $t + 15$ ($J = 24$).