# Can Access to Health Care Mitigate the Effects of Temperature on Mortality? *

Jamie T. Mullins
University of Massachusetts Amherst

Corey White
California Polytechnic State University, San Luis Obispo
& Monash University
& IZA

This version: August 14, 2020

**Abstract**

Understanding the sources of heterogeneity in the health effects of environmental exposure is critical for optimal policy design. Differential access to health care is commonly cited as a potential source of such heterogeneity. We test this hypothesis in a causal framework by combining random year-to-year fluctuations in local temperatures with variation in access to primary care services resulting from the idiosyncratic roll-out of Community Health Centers (CHCs) across US counties in the 1960s and 1970s. We find that the improved access to primary care services provided by CHCs moderates the heat-mortality relationship by 14.2%, but we find little evidence that CHC access mitigates the harmful effects of cold. In a supplementary analysis we find evidence that acute care – in contrast to primary care – may be especially effective at mitigating the cold-mortality relationship. 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: I10, I14, I18, Q50, Q52, Q54, Q58
Keywords: Health Care, Access, Climate, Temperature, Environment

*This paper has benefitted from discussions with Alan Barreca, Olivier Deschênes, Teevrat Garg, Andrew Goodman-Bacon, Sara Lowes, Maya Rossin-Slater, and Heather Royer. The paper has also benefitted from comments and suggestions by seminar participants at the NAREA annual meetings, the Hawaii Applied Micro Conference, University of Oregon, Cal Poly San Luis Obispo, UC Riverside, CU Denver, University of Nevada Reno, William & Mary, Melbourne Institute, Monash University, University of Sydney, and UNSW. Thanks also to two anonymous reviewers and the editor for providing comments and suggestions that led to a substantially improved paper. Neither author has received financial support or other funding for this project.

# 1 Introduction

The effects of exposure to adverse environmental conditions are heterogeneous across populations, with disadvantaged groups typically facing greater damages.[1] While this heterogeneity has been documented in a wide variety of settings, the underlying source of heterogeneity is rarely well understood. Differential access to health care is often posited as a potential source of such heterogeneity. We use a causal framework to test whether differential access to health care explains heterogeneity in the health impacts of extreme temperature exposure. Specifically, we test whether increased access to primary care services through Community Health Centers (hereafter, "CHCs") moderates the relationship between temperatures (both hot and cold) and mortality.

Understanding the sources of heterogeneity in environmental damages is important for the design of optimal policy as it informs the allocation of the "marginal dollar". For example, in the context of climate change, it may be more cost effective to invest in adaptation (e.g., health care) rather than mitigation (e.g., 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 of 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.

We leverage two sources of exogenous variation (CHC access and temperature) that have 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 due to a period known as the "great administrative confusion" at the federal agency which allocated CHC funding. Bailey and Goodman-Bacon (2015) find that CHCs reduced all-cause mortality in the years after initial establishment, that these ameliorative effects grew over time, and that the reductions in mortality rates were 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 identify causal impacts of temperature on a variety of outcomes including mortality (e.g., Deschênes and Greenstone, 2011; Dell et al., 2014).

[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.

Our analysis begins by successfully replicating the central results of both Bailey and Goodman-Bacon (2015) and prior studies on the temperature-mortality relationship (e.g., Deschênes and Greenstone, 2011) within a single model. We then construct a model to estimate the interaction between access to health care and temperature. Conceptually, we apply a difference-in-differences (henceforth, "DiD") design to estimate the effect of access to health care on the temperature-mortality relationship. Our interaction 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 a post-treatment indicator or time fixed effects in a standard DiD).[2]

We find that the presence of a CHC in a county mitigated the relationship between hot temperatures and all-cause mortality by approximately 14.2%. We find little evidence that CHCs affected the relationship between cold and all-cause mortality, though our estimates do suggest that CHCs mitigated cold-induced respiratory deaths. Given the services provided by CHCs (primary care, rather than direct treatment for acute conditions), our results suggest that access to primary care is more important for preventing deaths due to heat than cold.

Deaths triggered by heat and cold are driven by distinct mechanisms, and it is plausible that different types of health care may differentially impact heat- versus cold-related mortality. For example, cold-related mortality may be more sensitive to health care of a different type than was provided by CHCs. In a supplementary analysis, we leverage hospital desegregation in the American South as a source of variation in access to acute care (i.e., hospital care) and find that this type of care was especially effective at mitigating cold-related mortality. Taken together, these findings imply that primary care is more effective at reducing heat-related deaths whereas acute care is more effective at reducing cold-related deaths. We caution, however, that this conclusion is only suggestive given that the different settings in the CHC and desegregation analyses make direct comparisons difficult.

In general, our results demonstrate that increased access to health care can indeed mitigate environmental health damages. We therefore conclude that differential access to care is one driver behind the oft-noted differences in environmental damages across rich and poor populations. The different effects for heat- versus cold-related mortality imply that the potential for health care to mitigate environmental damages depends crucially on the specific nature of the damages and the health care intervention under consideration. Finally, our main finding – that improved access to primary care reduced the impacts of heat on mortality – suggests that increasing access to primary care may serve as a successful adaptive

[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.

mechanism for climate change.

This paper contributes to an active literature investigating heterogeneity in environmental damages. Several recent papers consider how income or nutrition supplementation programs contribute to heterogeneity in damages from environmental shocks in developing world contexts.[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) 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) investigate whether two Indian policies mitigate the impact of heat on infant mortality: a workfare program (NREGA) and a health worker program. They estimate that NREGA has no mitigating impact, whereas 9 months of pre-natal exposure to the health worker program mitigates the heat-infant-mortality relationship by over 80%. These estimates, however, do not account for potential pre-existing differences in the heat-infant-mortality relationship between treatment and control states and thus the estimates may partially reflect such differences.[4]

Cohen and Dechezleprêtre (2018) study the relationship between temperature and mortality in Mexico over the period 1998-2010. They find that while both cold and hot temperatures are associated with increases in mortality, the effects of cold (days with mean temperature $<50°$F) are much stronger. The authors then use a matching strategy to test whether enrollment in Mexico's national health insurance program – *Seguro Popular* – provides protective benefits against temperature-related mortality. They estimate 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.

Our setting is most similar to that studied by Barreca et al. (2016), in which the authors demonstrate a "remarkable" decline in the relationship between high temperatures and

[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. 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.

[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).

mortality over the course of the 20th century in the US, which was particularly dramatic during a period of rapid expansion in air conditioning technology (henceforth, "AC") after 1960. In Panel A of Figure 1, we use our own data to show that this decline was remarkable indeed: between 1959 and 1988, the heat-mortality relationship fell by 70%. Notably, the cold-mortality relationship also declined by 60%. Barreca et al. (2016) estimate the interaction between temperature and AC penetration and note that their estimates imply that the diffusion of AC technology can explain 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. As the authors note, quasi-experimental variation in AC penetration or the other potential modifiers does not exist, and thus they cannot rule out the possibility that other factors evolving simultaneous to the diffusion of AC technology could have contributed to the declining heat-mortality relationship.

The period of rapid expansion in AC technology coincided with substantial increases in access to – and spending on – health care. For instance, Panel B of Figure 1 shows the roll-out of the CHC program that we examine, which began in 1965. Furthermore, several other large public programs (e.g., Medicare and Medicaid) were implemented in the mid-1960s. Figure 1 shows that government health expenditures as a share of GDP more than tripled over our study period, 1959-1988. In principle, any of the expansions in access to care in this period could have mitigated health impacts of temperature exposures. 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 use 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.[5]

[5]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).

The remainder of this paper proceeds as follows: Section 2 provides a brief conceptual framework and background information on Community Health Centers. Section 3 outlines the details of our data and empirical approach. Section 4 presents our main results and a series of robustness checks. Section 5 provides a discussion of the mechanisms and the supplementary analysis examining Southern hospital desegregation, and Section 6 concludes.

# 2 Background & Conceptual Framework

## 2.1 Conceptual Framework

The conceptual goal of this paper can be illustrated using a slightly modified version of the framework presented by Graff-Zivin and Neidell (2013), which considered how environmental conditions affect health and human capital by building on the Grossman (1972) model of health production. Suppose that mortality ($M$) is a function of weather ($W$), access to pre-exposure primary/preventative care ($P$), and access to post-exposure acute treatment ($T$). Following Graff-Zivin and Neidell (2013), we make a distinction between the ultimate health outcome of interest ($M$) and an illness episode ($\phi$), yielding:

$$M = f\left(T, \phi(W, P)\right) \tag{1}$$

The purpose of this paper is to consider how the effect of weather on mortality (i.e., $\frac{\partial M}{\partial W}$) depends on access to health care (i.e., the levels of $P$ and $T$). The formulation of the mortality production function in Equation (1) highlights an explicit distinction between the roles of access to preventative care versus acute treatment (a distinction also made elsewhere, including Cutler (2001)). Preventative care ($P$) occurs prior to exposure and affects mortality by modifying the effect of weather on the probability of having an illness episode (e.g., a heart attack), whereas acute treatment ($T$) occurs after exposure and alters the probability of death conditional on experiencing an illness episode.

Because CHCs provided preventative care rather than acute treatment (a claim supported in Section 2.2), the central analysis of this paper estimates how exogenously improved access to preventative care modifies the effect of temperature on mortality: $\frac{\partial^2 M}{\partial W \partial P}$.[6] The distinction between preventative care and acute treatment is important for interpreting the mechanisms underlying our estimates. For example, we do not expect that CHCs mitigate $\frac{\partial M}{\partial W}$ by providing immediate treatment for specific temperature-induced illness episodes (e.g., heat stroke) because CHCs did not provide such acute (or emergency) care. Instead, we expect

[6] Hsiang et al. (2019) note that identifying sources of heterogeneity in environmental damages requires empirical identification of a second-order derivative of this form.

that CHCs improve an individual's health stock through preventiative care and better management of chronic health conditions, thereby decreasing the likelihood that temperature exposure induces an illness episode.

We return to this framework in Section 5 to discuss the mechanisms underlying our estimates and present a supplemental analysis with the goal of estimating $\frac{\partial^2 M}{\partial W \partial T}$.

## 2.2 What Are CHCs and How Were They Implemented?

The CHC program exists today but has changed substantially since its initial implementation during the period we analyze. During our study period, CHCs were networks of clinics that provided primary care services on an outpatient basis. Taylor (2004) notes that CHC funding was explicitly contingent on the provision of "comprehensive primary health care services", and Bailey and Goodman-Bacon (2015) use the Survey of Health Services Utilization and Expenditures to show that the proportion of the relevant population (older, low-income adults) reporting a "regular source of care" increased by 23% following CHC establishment. CHCs did not provide treatment for emergent conditions (Taylor, 2004). This is again supported by the analysis of Bailey and Goodman-Bacon (2015), who "show that CHCs had no measurable impact on accident-related mortality in any specification, which is consistent with their provision of primary (but not emergency) care". In the terminology of Equation (1), this suggests that CHCs significantly increased $P$, but not $T$.

CHCs provided services at little or no cost to patients, served patients who were uninsured, underinsured, or low-income, and typically 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 the source of quasi-random variation in CHC establishments (and thus access). 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 use 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.

## 2.3 How Can CHCs Affect Mortality and Temperature-Related Mortality?

CHCs principally provided access to primary and preventative health care, and Bailey and Goodman-Bacon (2015) note that CHCs could have reduced mortality among older adults through several channels. CHCs may have increased early detection, awareness, and treatment of chronic yet manageable conditions like hypertension. CHCs provided free or reduced-cost pharmaceuticals for the management of such conditions (e.g., beta blockers for hypertension), making it easier for patients to maintain medication regimens. CHC access may have reduced mortality indirectly by increasing awareness of Medicaid and Medicare. It is also possible that CHCs could have reduced emergency department crowding by reducing usage for non-emergent conditions.

Given the type of care provided by CHCs, they might be expected to increase the health stock of served populations, which could in turn increase population resilience to health shocks of many kinds including exposures to extreme temperatures. For example, Bailey and Goodman-Bacon (2015) show that CHCs led to better hypertension management. Hypertension is a risk factor for cardiovascular disease (e.g., heart attack) and cerebrovascular disease (e.g., stroke), both of which are common causes of death triggered by extreme temperature exposure.[7] Bailey and Goodman-Bacon (2015) show that CHCs reduced mortality due to both cardiovascular and cerebrovascular disease, suggesting a means by which CHCs might impact mortality triggered by extreme temperatures.

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.

[7] We find that cardiovascular and cerebrovascular disease account for approximately 50% of all cold-related deaths and 71% of all heat-related deaths in the pre-CHC period. 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), the cardiovascular mortality rate by 0.0905 and the cerebrovascular mortality rate by 0.0311. The proportion of cold-related mortality attributable to cardiovascular and cerebrovascular causes is the ratio (0.0905+0.0311)/0.241. One day $>80°$F increases the all-cause mortality rate by 0.339, the cardiovascular mortality rate by 0.169, and the cerebrovascular mortality rate by 0.0726.

# 3 Data and Empirical Strategy

## 3.1 Data

Our main 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 CHC establishments in 114 counties between 1965-1974 and the subsequent 14 year period.[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 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 estimates of both CHC access *and* temperature on mortality in a single econometric model. We primarily follow the specification used by Bailey and Goodman-Bacon (2015). Their model includes a rich set of controls, making it well-suited for identifying ambient temperature effects in addition to CHC impacts. Our model is distinct from that of 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. Our unit of analysis is the county-year-month. Equation (2) describes the model, with subscripts $c$, $y$, $m$, and $u$ representing county, year, calendar month, and urban group (five categories of 1960 urbanicity).

[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.

$$\text{AMR}_{cym} = \gamma \text{CHC}_{cy}^{t \geq 0} + \pi g(\text{Temp}_{cym}) + \beta \text{X}_{cym} + \delta_{sy} + \delta_{cm} + \delta_{uy} + \delta_{ym} + \varepsilon_{cym} \tag{2}$$

The outcome of interest is $\text{AMR}_{cym}$: the age-adjusted mortality rate in county $c$, year $y$ and month $m$.[9] The first coefficient of interest is $\gamma$, 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. An important point here is that both mortality and CHC access are measured at the county level. Because only a portion of residents in a given county will utilize the services provided by CHCs, our estimates must be interpreted as intent-to-treat estimates.

The second coefficient of interest is $\pi$, 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 these two temperature variables, 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 omitted temperature category in the two-bin model is days with mean temperature between 40°F and 80°F, while the omitted temperature category in the five-bin model is days with mean temperature between 60°F and 70°F. The simpler models are sometimes preferred due the fact that fewer parameters need to be estimated, which 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

[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.

distribution, but only require estimating three parameters.[10]

The remaining controls ($X_{cy}, \delta_{sy}, \delta_{cm}, \delta_{uy}, \delta_{ym}$) 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.[12] $\delta_{sy}$ are state-by-year fixed effects and $\delta_{uy}$ are urban-group-by-year fixed effects.[13] $\delta_{cm}$ are county-by-month fixed effects which are used in place of the county fixed effects in Bailey and Goodman-Bacon (2015) and control for both time-invariant differences across counties and local seasonality. This is potentially important because both temperature and mortality exhibit substantial seasonality, which may differ between regions. $\delta_{ym}$ 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-by-month levels (Cameron et al., 2011). County clustering allows for arbitrary within-county serial correlation and year-by-month clustering allows for arbitrary spatial correlation within a year-month. All regressions are weighted by 1960 county populations.

Identification of $\gamma$ in Equation (2) 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 $\pi$ in Equation (2) 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 generally considered to be random, satisfying this assumption.

[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. These models therefore 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.

[12]The time-varying covariates include precipitation controls and variables obtained from Bailey and Goodman-Bacon (2015). These 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.

[13]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 again refer readers 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. Nevertheless, we show that estimates of both $\gamma$ and $\pi$ are stable across versions of Equation (2) in which both treatments are included and each is included separately.[14]

### 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, our empirical approach is to estimate a DiD design 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 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 specification, described below in Equation (3).

$$\begin{aligned} \text{AMR}_{cym} = {} & \phi(\text{CHC}_{cy}^{t\geq 0} \times g(\text{Temp}_{cym})) + \gamma \text{CHC}_{cy}^{t\geq 0} + \pi g(\text{Temp}_{cym}) \\ & + \theta(g(\text{Temp}_{cym}) \times \text{Treated}_c) + g(\text{Temp}_{cym}) \times \delta_y \\ & + \beta \text{X}_{cym} + \delta_{sy} + \delta_{cm} + \delta_{uy} + \delta_{ym} + \varepsilon_{cym} \end{aligned} \tag{3}$$

The coefficient of interest in Equation (3) is $\phi$. We allow for fixed differences in the

[14] 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.

temperature-mortality relationship between CHC and non-CHC counties through the inclusion of $g(\text{Temp}_{cym}) \times \text{Treated}_c$, where $\text{Treated}_c$ is an indicator for whether a CHC was ever established in the county over the 1965-1974 period. In an alternative specification, we 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: $g(\text{Temp}_{cym}) \times \delta_c$. These estimates are presented along with our main results, however because this specification adds over 3,000 additional parameters for each temperature variable, we opt for the more parsimonious model as our preferred specification.

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 also account for differences in the temperature-mortality relationship over time. To this end, year-specific temperature effects are included: $g(\text{Temp}_{cym}) \times \delta_y$.

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. Specifically, our estimates could be biased if the expansion of AC technology is correlated with the timing of CHC establishment (though we have little reason to expect this). To address this possibility, we also estimate a specification that includes the interaction between temperature and the state-year AC penetration rate: $g(\text{Temp}_{cym}) \times \text{AC}_{sy}$.

With these controls included, $\phi$ in Equation (3) 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 (3) 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 (3), $g(\text{Temp}_{cym})$ is a function of mean daily temperatures. Just as in the replication model, our preferred specifications rely upon 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 (2). The models vary in whether 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 led to approximately 1.14 fewer monthly deaths per 100,000 population. Relative to the pre-CHC mean AMR of 81.7 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 led to substantial increases in mortality during our sample period. The coefficient on $Temp^{<40}$ ($Temp^{>80}$) implies that one additional day under 40°F (over 80°F) increases the monthly AMR by 0.116 (0.182), relative to a day in the 40-80°F range. 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. 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 used 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

[15] The extreme temperature bins ($Temp_{cym}^{<40}$ and $Temp_{cym}^{>80}$) 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.

specification). 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, following Bailey and Goodman-Bacon (2015). The coefficient on the pre-treatment CHC indicator ($\text{CHC}^{t\leq -2}$) 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, all highly significant, and increasing with time since CHC establishment. The results in Column 5 (which includes temperature variables) also closely mirror the results of Bailey and Goodman-Bacon (2015): our coefficient estimates on the $\text{CHC}^{0\leq t\leq 4}$, $\text{CHC}^{5\leq t\leq 9}$, $\text{CHC}^{t\geq 10}$ 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. Panel A reports single-year event study estimates for the effect of CHC access 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.

Panels B and C report flexible estimates of the effects of temperature on mortality. For Panels B and C, we have limited the sample to the 114 counties that establish a CHC and the years prior to CHC establishment (1959-1964). These estimates can thus serve a valid baseline against which we can compare the interaction effects in the following section. Panel B reports coefficient estimates from a model with five 10°F temperature bins (60-70°F is omitted). This yields the familiar U-shaped relationship between temperature and mortality that has been well documented in the prior literature. Panel C reports estimates from a specification that models temperature as a third-order polynomial. This specification again yields the familiar U-shaped relationship with estimates of similar magnitude.

[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).

## 4.2 Interaction Model

Panel A of Table 3 presents estimates of the interaction models in which temperature is modelled using two bins ($\text{Temp}^{<40}$ and $\text{Temp}^{>80}$), and CHC access is modelled using a single post-treatment dummy. Column 1 is the baseline specification described in Equation (3), and Columns 2-5 represent a variety of alternative specifications. Column 2 tests whether the estimates are sensitive to allowing the effects of temperature to vary across AC penetration rates. Column 3 replaces the $g(\text{Temp}) \times \text{Treated}_c$ controls with the more general county-specific temperature effects ($g(\text{Temp}) \times \delta_c$); these represent over 3,000 additional parameters for each temperature variable, absorbing arbitrary fixed differences across all counties in the temperature-mortality relationship. Column 4 replaces the year-specific temperature effects ($g(\text{Temp}) \times \delta_y$) with state-year temperature effects ($g(\text{Temp}) \times \delta_{sy}$). This specification controls for anything changing differentially at the state level that could affect the temperature mortality relationship, including state-level health policy or factors correlated with state climate such as AC penetration. Finally, Column 5 includes a set of county-by-year fixed effects, which non-parametrically control for any county-level factors that vary at the annual (but not sub-annual) level.

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. In most specifications, we find no evidence that CHC access has a significant impact on the cold-mortality relationship. One exception is in Column 5, in which the coefficient of interest is negative and significant at the 5% level. The negative sign implies that CHC access did successfully mitigate the cold-mortality relationship, but this evidence is weak given the inconsistency across the various specifications.

For hot temperatures, the coefficient estimate on $\text{CHC}^{t\geq 0} \times \text{Temp}^{>80}$ in Column 1 (the main specification) yields a negative and statistically significant interaction term. The coefficient estimate changes little when AC interactions are included in Column 2. The magnitude of the estimate is also stable across Columns 3 and 4, though the standard errors increase substantially in these specifications due to the large number of additional parameters that need to be estimated (nevertheless, the estimates remain significant at the 10% and 5% levels, respectively). In Column 5, the estimate is slightly smaller in magnitude, though still significant at the 5% level. These estimates imply that CHC access successfully mitigated the heat-mortality relationship, and the consistency across these various specifications strengthens that conclusion.

The magnitude of the coefficient estimate on $\text{CHC}^{t\geq 0} \times \text{Temp}^{>80}$ in our preferred specification (Column 1) implies that CHC access reduced the effect of an additional day above 80°F on mortality by 0.048 deaths per 100,000 population (relative to a 40-80°F day). In

relative terms, the estimate implies that CHC access mitigated the harmful effect of heat on mortality by 14.2%, relative to the heat-mortality relationship in CHC counties in the pre-CHC period.[18]

As an alternative way to interpret our estimates, we ask how much of the overall decline in mortality attributable to CHC access comes from heat-related deaths? To answer this, we multiply our preferred interaction estimate (-0.048) by the average number of hot days per month (1.72). This product is the average number of heat-related deaths averted by CHC access per month, per 100,000 population. We then divide by the overall effect of CHCs from Column 1 of Table 2 (-1.136), implying that approximately 7.3% of deaths averted by access to CHCs were heat-related. Because CHCs were not targeted toward preventing heat-related mortality, it is not surprising that only a small portion of the deaths they prevented were heat-related.

In Panel B of Table 3, we present estimates from a specification that allows for dynamic CHC treatment effects in a binned event-study framework. For both cold and hot temperatures, the first coefficient estimates ($\text{CHC}^{t\leq -2} \times \text{Temp}^{<40}$ and $\text{CHC}^{t\leq -2} \times \text{Temp}^{\geq 80}$) 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. 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 yield no statistically significant evidence of an effect of CHCs on the cold-mortality relationship in any specification. The estimates for the post-treatment interactions with >80°F days are negative (across specifications), generally increase slightly in magnitude with years since establishment, and are statistically significant in the less demanding specifications (Columns 1, 2, and 5).

The binned event-study approach groups together years relative to CHC establishment to maximize the power of the dynamic estimates. We also present estimates of a full, year-by-year event study in Figure 3. 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 post-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

[18]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).

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.

By modeling temperature using only two variables, the estimates in Table 3 provide a relatively straightforward interpretation. In Figure 4, we model temperature more flexibly to provide additional insight. Panel A uses five 10°F bins and Panel B uses a third-order polynomial. Each panel displays the interaction effects of temperature with a post-CHC dummy. Both specifications reinforce the findings from Table 3. The estimate from the five-bin model (Panel A) implies that CHC access mitigated the effect of an >80°F day by 13.6% (relative to a 60-70°F day).[19] The polynomial specification (Panel B) is useful as it 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. In the polynomial specification, the mitigating effects grow larger in absolute terms at the high end of the distribution, but they are fairly constant (even decreasing) in relative terms. Specifically, the polynomial specification implies that the effect of days with mean temperatures of 80°F and 85°F (relative to a 65°F day) were mitigated by 16% and 13%, respectively.[20]

## 4.3 Additional Results and Robustness Checks

We present the estimates of a series of robustness checks and additional results in Tables A3 to A5. Details of these exercises are provided in Appendix Section A.3. To summarize: we only find evidence that CHCs mitigated the heat-mortality relationship for individuals aged 50 and older; results are robust to reliance on a series of more narrow, matched control groups; and neither lagged nor harvesting effects of temperature on mortality appear to be driving our estimates.

[19] This is relative to the baseline effects of temperature in CHC counties in the pre-CHC period, estimated using a comparable five-bin model (estimates from which are displayed in Panel B of Figure 2). The coefficient on the interaction is -0.0517 and the coefficient on the baseline effect is 0.380, thus the implied mitigation is -0.0517/0.380 = -0.136. The coefficient estimates for the five-bin model are presented in Table A2.

[20] This is relative to the baseline effects of temperature in CHC counties in the pre-CHC period at the same temperatures, estimated using a comparable polynomial model (estimates from which are displayed in Panel C of Figure 2). The interaction effects at 80°F and 85°F are estimated to be -0.028 and -0.044. The baseline effects of temperature on mortality at 80°F and 85°F are estimated to be 0.175 and 0.338. The percent mitigation is the interaction effect divided by the baseline.

# 5 Understanding Mechanisms

At this point, we have provided estimates of $\frac{\partial^2 M}{\partial W \partial P}$ (referring to Equation (1)) for both hot and cold temperature shocks. The evidence supports the conclusion that primary care (measured as access to CHCs) mitigates the heat-mortality relationship, but the evidence does not support strong mitigating effects for the cold-mortality relationship. Why is this the case? Answering this question requires understanding the different mechanisms underlying the mortality effects of heat versus cold, and how those differences interact with the type of care that we study.

## 5.1 Differences in the Effects of Heat and Cold

What are the different mechanisms underlying mortality triggered by heat versus cold? For heat, it is often posited that the body's thermoregulatory response imposes additional stress on the cardiovascular and cerebrovascular systems (Basu and Samet, 2002); in a multi-country epidemiological study, Gasparrini et al. (2015) state, "in the case of the association of heat with cardiovascular mortality... acute events seem to be triggered when the body exceeds its thermoregulatory threshold". For cold, there is evidence that suggests cold exposure can trigger physiological responses such as thrombosis (i.e., blood clotting), which can then lead to acute cardiovascular and cerebrovascular events (Keatinge et al., 1984). Cold weather can also induce mortality through cross-infection from indoor crowding, the adverse effects of cold exposure on the immune system, and increased survival of bacteria and viruses during cold temperatures (Eurowinter Group, 1997; Gasparrini et al., 2015).

We can see these distinctions to some extent in our own data. In Panel A of Table 4, we provide estimates of the effects of both cold and hot temperatures on mortality by six cause of death categories that are measured consistently over our sample period. Cardiovascular and cerebrovascular deaths account for approximately 50% of cold-related mortality and 71% of heat-related mortality.[21] Because Bailey and Goodman-Bacon (2015) find that CHCs reduced mortality primarily among these causes of death, this begins to uncover why CHCs had larger mitigating impacts for heat versus cold: heat-related mortality is more concentrated in the causes of deaths that were prevented by access to CHCs.

In Panel B of Table 4, we present estimates of the interaction model by cause of death. While power is limited, these estimates reveal at least one important finding: the <40°F interaction is negative and statistically significant for respiratory disease. This suggests that

[21] We calculate these percentages by summing the coefficients on Cardiovascular and Cerebrovascular mortality, and dividing by the coefficient on All-Cause mortality (e.g., for cold-related mortality: $(0.0905 + 0.0311)/0.242$).

important heterogeneity was hidden in our analysis of all-cause mortality, and that CHCs were indeed effective at preventing certain types of cold-related mortality.

This analysis by cause of death is useful, but limited, notably in that we can only construct consistent disease categories for very broad groups over our sample period. Furthermore, cause of death is an incomplete measure of the conditions that lead to mortality. It is possible, for example, that two deaths with the same ultimate cause of death were triggered by very different initial conditions. As an alternative method for differentiating between the mortality effects of heat and cold, we examine the daily dynamics of each relationship. This is possible because data on the exact date of death are available for most years in our sample.

To analyze the dynamic effects of temperature, we utilize daily data on weather and mortality (date of death was recorded for 1962-1966 and 1972-1988) in combination with a daily-level distributed lag model that is otherwise similar to Equation (2).[22] The results of this analysis are presented in Figure 5 for both cold days (Panel A) and hot days (Panel B). Each plot displays the daily effects (i.e., coefficient estimates) and the dynamic cumulative effects (i.e., sums of coefficient estimates). For a temperature shock on day 0, the daily effects represent the change in mortality $t$ days later, and the cumulative effects represent the total change in mortality from day 0 through day $t$. It is immediately clear that the dynamics are very different between the effects of heat versus cold. For a >80°F day, almost the entire increase in mortality comes on the day of the shock and the day after. For a <40°F day, there is a decrease in mortality on the day of the shock, and increases in mortality 2-20 days later. As an example, take note of the cumulative effect two days after the shock ($t = 2$): the cumulative effect peaks at this point for heat, whereas the cumulative effect is not statistically different from zero for cold.

The main takeaway is that the effects of heat tend to be immediate whereas the effects of cold tend to be delayed.[23] These different dynamics imply there are different pathways underlying the relationships between heat versus cold, and thus we should not necessarily expect that one type of health care should mitigate these relationships equally. Furthermore, these dynamics have implications for the potential mitigating effects of different types of care. To consider this, let us again refer back to Equation (1) and the distinction between acute treatment ($T$) and preventative care ($P$). Note that acute treatment can only be effective if

[22]The model we estimate is specified as follow:

$$\text{MR}_{ct} = \sum_{h=0}^{30} \pi_{t-h} g(\text{Temp}_{c,t-h}) + \beta \text{X}_{cym} + \delta_{sy} + \delta_{cm} + \delta_{uy} + \delta_{ym} + \delta_{day-of-week} + \delta_{day-of-year} + \varepsilon_{cym}$$

[23]This finding is consistent with prior work utilizing daily data on mortality (Anderson and Bell, 2009; Deschênes and Moretti, 2009; Gasparrini et al., 2015) and emergency department visits (White, 2017).

there is time to seek treatment between the onset of an illness episode and mortality. The immediate effects of heat on mortality suggest there is less time to seek treatment for heat-induced illness episodes, and thus increasing access to acute treatment may not be as effective as preventative care at mitigating heat-related deaths. On the other hand, the delayed impacts of cold suggest that access to treatment may be relatively more important. We explore this further in the following section, in which we present results from a supplementary analysis that has the goal of estimating the mitigating effect of acute treatment: $\frac{\partial^2 M}{\partial W \partial T}$.

## 5.2 The Effects of Increasing Access to Acute Treatment: Southern Hospital Desegregation

In this section, we provide a supplementary analysis in which we utilize the desegregation of Southern hospitals to test how access to acute treatment (specifically hospital care) mitigates the temperature-mortality relationship. We chose to focus on hospital desegregation for a number of reasons: (1) hospital desegregation represented a massive change in access to acute treatment for a well-defined group, (2) desegregation occurred during the same 30-year sample period as our CHC analysis and thus the setting and data are similar, and (3) we can build directly on prior work – Almond et al. (2006) – that studied the direct mortality effects of hospital desegregation.

While additional information on Southern hospital desegregation can be found in Almond et al. (2006), we briefly summarize several points regarding desegregation that are key to understanding our analysis. Hospital segregation in the US entailed a separate hospital building, wing, or floor for non-white patients. These "negro wards" were typically small and offered sub-standard care; they were often over-crowded and potential patients would be turned away if the ward was at capacity. Hospital segregation persisted in the Southern US largely unchecked for many years after the 1954 *Brown vs. Board of Education* decision that resulted in the desegregation of public schools. Most Southern hospitals were ultimately desegregated following the 1966 implementation of Medicare, which barred segregated hospitals from receiving reimbursement through the program.

Desegregation would have had the largest mortality impacts on potentially fatal illnesses for which effective in-hospital treatments were available. Almond et al. (2006) tailor their analysis to a specific population that was especially likely to benefit: post-neonatal infants (1-12 months old). Many post-neonatal deaths in this period were due to the contraction of infectious diseases leading to pneumonia and gastroenteritis, and deaths due to these conditions could be avoided with timely medical treatment. Another advantage of focusing on post-neonatal infants (rather than older children or adults) is that they would not have

been directly impacted by other policy changes at the time such as the implementation of Medicare or the desegregation of public schools.[24] In our analysis, we follow Almond et al. (2006) and focus only on post-neonatal infants. Desegregation could have influenced the temperature-mortality relationship if the types of deaths triggered by temperature exposure (1) overlap with deaths that could have been prevented by desegregation and (2) exhibit a sufficient delay between exposure and death such that successful treatments could be implemented.

In estimating the effects of desegregation, Almond et al. (2006) employ two strategies. The first strategy simply compares post-neonatal mortality among non-whites and whites in Southern states, before and after desegregation occurred in 1966. The second strategy is a more rigorous approach that utilizes the actual year of desegregation by hospital, but confines the analysis to Mississippi where the necessary data is available. Our analysis builds off of the first strategy because we need to simultaneously estimate the effects of temperature, which requires sufficient variation in weather in each period of study. Given that Almond et al. (2006) find very similar results with their two strategies, we are confident that our approach primarily captures the causal effects of desegregation. That being said, we acknowledge that our estimates may capture other factors that contributed to the reduction in the racial gap in post-neonatal mortality in Southern states in the 1960s to 1970s. As such, our estimates should be considered an upper bound, and we proceed with this caveat in mind.

The structure of our analysis of desegregation closely mirrors our analysis of CHCs. Specifically, we first estimate the effects of temperature and desegregation on post-neonatal mortality in separate replication models and then build models to estimate the interaction. The empirical specifications are very similar to Equations (2) and (3), and are described in Appendix B.

The results for both the replication and interaction models are presented in Figure 6. In all panels, the outcome is the post-neonatal mortality rate (PNMR) per 100,000 births. Panels A, B, and C represent the replication estimates (mirroring Figure 2), and Panels D and E represent the interaction estimates (mirroring Figure 4). Panel A demonstrates a successful replication of Almond et al. (2006): hospital desegregation led to a massive decline in post-neonatal mortality among non-whites compared to whites. The corresponding difference-in-differences estimate is a decrease in the PNMR of 71.77, representing an approximate 44% decline relative to the pre-desegregation mean non-white PNMR of 163.9. Panels B and C represent the effects of temperature on the non-white PNMR in the pre-desegregation period, using temperature bins (Panel B) and a polynomial specification (Panel C). Both panels

[24] As noted by Almond et al. (2006), Medicaid didn't become available in many Southern states until years after 1966. In Mississippi, for instance, Medicaid was not available until 1970 (Almond et al., 2006).

indicate that cold temperatures led to large increases in the PNMR. This is consistent with the fact that most deaths in this population were due to causes linked to infectious disease.

Panels D and E represent estimates of the interaction between hospital desegregation and temperature. Both panels indicate that hospital desegregation was highly effective at mitigating the relationship between cold and post-neonatal mortality. The magnitude of the mitigating effects is large, though consistent with the large overall effects of desegregation. The implied mitigation from the polynomial specification is 83% at 35°F, and the binned specification implies complete mitigation of the effects of days <50°F.

The analysis of desegregation provides an estimate of the mitigating effects of acute care ($\frac{\partial^2 M}{\partial W \partial T}$), whereas the CHC analysis provides an estimate of the mitigating effects of primary care ($\frac{\partial^2 M}{\partial W \partial P}$). Comparing these two analyses implies that acute care is relatively more important for cold-related illness and that primary care is relatively more important for heat-related illness. We emphasize, however, that these estimates were derived from two health care experiments that took place in very different contexts, and thus we view any conclusions resulting from this comparison as only suggestive in nature.

# 6 Conclusion

The goal of this paper is to understand how access to health care can mitigate the health impacts of exposure to extreme temperatures. Utilizing quasi-experimental variation in access to care through the roll-out of the Community Health Center (CHC) program, our main finding is that the establishment of a CHC in a county mitigated the relationship between heat and all-cause mortality by approximately 14%. We find little evidence that CHC access mitigated the effects of cold on all-cause mortality.

We note that health care is not one-dimensional. In particular, we make a distinction between preventative (or primary) care and acute treatment. CHCs only provided preventative care, and thus our main analysis is only relevant to the provision of preventative care. This is an important consideration because different types of care may be more relevant to certain environmental shocks. Our main analysis demonstrates that preventative care is effective at reducing heat-induced mortality, and a supplementary analysis – leveraging variation in access to acute treatment arising from hospital desegregation in the American South – shows that acute treatment is effective at reducing cold-induced mortality.

This paper uses a causal framework to demonstrate that improved access to health care (defined generally) can indeed mitigate the effects of exposure to adverse environmental conditions. The results also suggest that the mode of care (i.e., primary or acute care) is important. 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. 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 adverse environmental conditions only insofar as the mode of health care can be reasonably well-targeted. In the context of climate change, the ameliorative effect of primary care on the heat-mortality relationship suggests that expanding access to primary care may be an effective approach to mitigating the health damages of a warmer climate.

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.

# References


**Adhvaryu, Achyuta, Anant Nyshadham, Teresa Molina, and Jorge Tamayo**, "Helping Children Catch Up: Early Life Shocks and the PROGRESA Experiment," Working Paper 24848, National Bureau of Economic Research July 2018.

**Almond, Douglas and Bhashkar Mazumder**, "Fetal origins and parental responses," *Annu. Rev. Econ.*, 2013, *5* (1), 37–56.

**_ , Kenneth Y Chay, and Michael Greenstone**, "Civil rights, the war on poverty, and black-white convergence in infant mortality in the rural South and Mississippi," 2006.

**Anderson, Brooke G and Michelle L Bell**, "Weather-related mortality: how heat, cold, and heat waves affect mortality in the United States," *Epidemiology (Cambridge, Mass.)*, 2009, *20* (2), 205.

**Arceo, Eva, Rema Hanna, and Paulina Oliva**, "Does the effect of pollution on infant mortality differ between developing and developed countries? Evidence from Mexico City," *The Economic Journal*, 2016, *126* (591), 257–280.

**Bailey, Martha J and Andrew Goodman-Bacon**, "The War on Poverty's experiment in public medicine: Community health centers and the mortality of older Americans," *American Economic Review*, 2015, *105* (3), 1067–1104.

**Banerjee, Rakesh and Riddhi Maharaj**, "Heat, Infant Mortality and Adaptation: Evidence from India," 2018.

**Barreca, Alan, Karen Clay, Olivier Deschenes, Michael Greenstone, and Joseph S Shapiro**, "Adapting to Climate Change: The Remarkable Decline in the US Temperature-Mortality Relationship over the Twentieth Century," *Journal of Political Economy*, 2016, *124* (1), 105–159.

**Basu, Rupa and Jonathan M Samet**, "Relation Between Elevated Ambient Temperature and Mortality: A Review of the Epidemiologic Evidence," *Epidemiologic Reviews*, 2002, *24* (2), 190–202.

**Borusyak, Kirill and Xavier Jaravel**, "Revisiting event study designs," *Available at SSRN 2826228*, 2017.

**Burgess, Robin, Olivier Deschenes, Dave Donaldson, and Michael Greenstone**, "Weather, climate change and death in India," *University of Chicago*, 2017.

**Cameron, A Colin, Jonah B Gelbach, and Douglas L Miller**, "Robust inference with multiway clustering," *Journal of Business & Economic Statistics*, 2011, *29* (2), 238–249.

**Carleton, Tamma, Michael Delgado, Michael Greenstone, Trevor Houser, Solomon Hsiang, Andrew Hultgren, Amir Jina, Robert E Kopp, Kelly McCusker, Ishan Nath et al.**, "Valuing the Global Mortality Consequences of Climate Change Accounting for Adaptation Costs and Benefits," 2018.

**Chay, Kenneth Y and Michael Greenstone**, "The impact of air pollution on infant mortality: evidence from geographic variation in pollution shocks induced by a recession," *The quarterly journal of economics*, 2003, *118* (3), 1121–1167.

**Cohen, François and Antoine Dechezleprêtre**, "Mortality inequality, temperature and public health provision: evidence from Mexico," 2018.

**Crump, Richard K, V Joseph Hotz, Guido W Imbens, and Oscar A Mitnik**, "Dealing with limited overlap in estimation of average treatment effects," *Biometrika*, 2009, *96* (1), 187–199.

**Currie, Janet and Reed Walker**, "Traffic congestion and infant health: Evidence from E-ZPass," *American Economic Journal: Applied Economics*, 2011, *3* (1), 65–90.

**Cutler, David M**, "The economics of better health: the case of cardiovascular disease," *Mimeo*, 2001.

**Dell, Melissa, Benjamin F Jones, and Benjamin A Olken**, "What Do We Learn from the Weather? The New Climate–Economy Literature," *Journal of Economic Literature*, 2014, *52* (3), 740–798.

**Deschênes, Olivier and Enrico Moretti**, "Extreme Weather Events, Mortality, and Migration," *The Review of Economics and Statistics*, 2009, *91* (4), 659–681.

_ **and Michael Greenstone**, "The economic impacts of climate change: evidence from agricultural output and random fluctuations in weather," *American Economic Review*, 2007, *97* (1), 354–385.

_ **and** _ , "Climate Change, Mortality, and Adaptation: Evidence from Annual Fluctuations in Weather in the US," *American Economic Journal: Applied Economics*, 2011, pp. 152–185.

**Dillender, Marcus**, "Climate Change and Occupational Health: Are There Limits to Our Ability to Adapt?," 2019.

**Eurowinter Group**, "Cold exposure and winter mortality from ischaemic heart disease, cerebrovascular disease, respiratory disease, and all causes in warm and cold regions of Europe," *The Lancet*, 1997, *349* (9062), 1341–1346.

**Fetzer, Thiemo**, "Can workfare programs moderate violence? Evidence from India," 2014.

**Garg, Teevrat, Gordon McCord, and Aleister Montfort**, "Adaption Through Social Policy: Temperature, Violence and Cash Transfers in Mexico," 2019.

_ **, Maulik Jagnani, and Vis Taraz**, "Temperature and human capital in India," 2018.

**Gasparrini, Antonio, Yuming Guo, Masahiro Hashizume, Eric Lavigne, Antonella Zanobetti, Joel Schwartz, Aurelio Tobias, Shilu Tong, Joacim Rocklöv, Bertil Forsberg et al.**, "Mortality risk attributable to high and low ambient temperature: a multicountry observational study," *The Lancet*, 2015, *386* (9991), 369–375.

**Goodman-Bacon, Andrew**, "Difference-in-differences with variation in treatment timing," Technical Report, National Bureau of Economic Research 2018.

**Graff-Zivin, Joshua and Matthew Neidell**, "Environment, Health, and Human Capital," *Journal of Economic Literature*, 2013, *51* (3), 689–730.

**Grossman, Michael**, "On the concept of health capital and the demand for health," *Journal of Political Economy*, 1972, pp. 223–255.

**Gunnsteinsson, Snaebjorn, Achyuta Adhvaryu, Parul Christian, Alain Labrique, Jonathan Sugimoto, Abu Ahmed Shamim, and Keith P West Jr**, "Protecting Infants from Natural Disasters: The Case of Vitamin A Supplementation and a Tornado in Bangladesh," 2018.

**Heutel, Garth, Nolan H Miller, and David Molitor**, "Adaptation and the Mortality Effects of Temperature Across US Climate Regions," Technical Report, National Bureau of Economic Research 2017.

**Hornbeck, Richard and Pinar Keskin**, "The historically evolving impact of the ogallala aquifer: Agricultural adaptation to groundwater and drought," *American Economic Journal: Applied Economics*, 2014, *6* (1), 190–219.

**Hsiang, Solomon, Paulina Oliva, and Reed Walker**, "The Distribution of Environmental Damages," *Review of Environmental Economics and Policy*, 2019.

**Johnson, Rucker C and C Kirabo Jackson**, "Reducing Inequality through Dynamic Complementarity: Evidence from Head Start and Public School Spending," *American Economic Journal: Economic Policy*, 2019.

**Karlsson, Martin and Nicolas R Ziebarth**, "Population health effects and health-related costs of extreme temperatures: Comprehensive evidence from Germany," *Journal of Environmental Economics and Management*, 2018, *91*, 93–117.

**Keatinge, WR, SR Coleshaw, F Cotter, M Mattock, M Murphy, and R Chelliah**, "Increases in platelet and red cell counts, blood viscosity, and arterial pressure during mild surface cooling: factors in mortality from coronary and cerebral thrombosis in winter.," *Br Med J (Clin Res Ed)*, 1984, *289* (6456), 1405–1408.

**Mullins, Jamie T and Corey White**, "Temperature and mental health: Evidence from the spectrum of mental health outcomes," *Journal of Health Economics*, 2019, *68*, 102240.

**Rao, Tanvi**, "The Impact of a Community Health Worker Program on Childhood Immunization: Evidence from India's 'ASHA' Workers," *SSRN Electronic Journal*, 01 2014.

**Rossin-Slater, Maya and Miriam Wüst**, "What is the added value of preschool? Long-term impacts and interactions with an infant health intervention," *NBER Working Paper No. 22700, IZA Discussion Paper No*, 2018, *10254*.

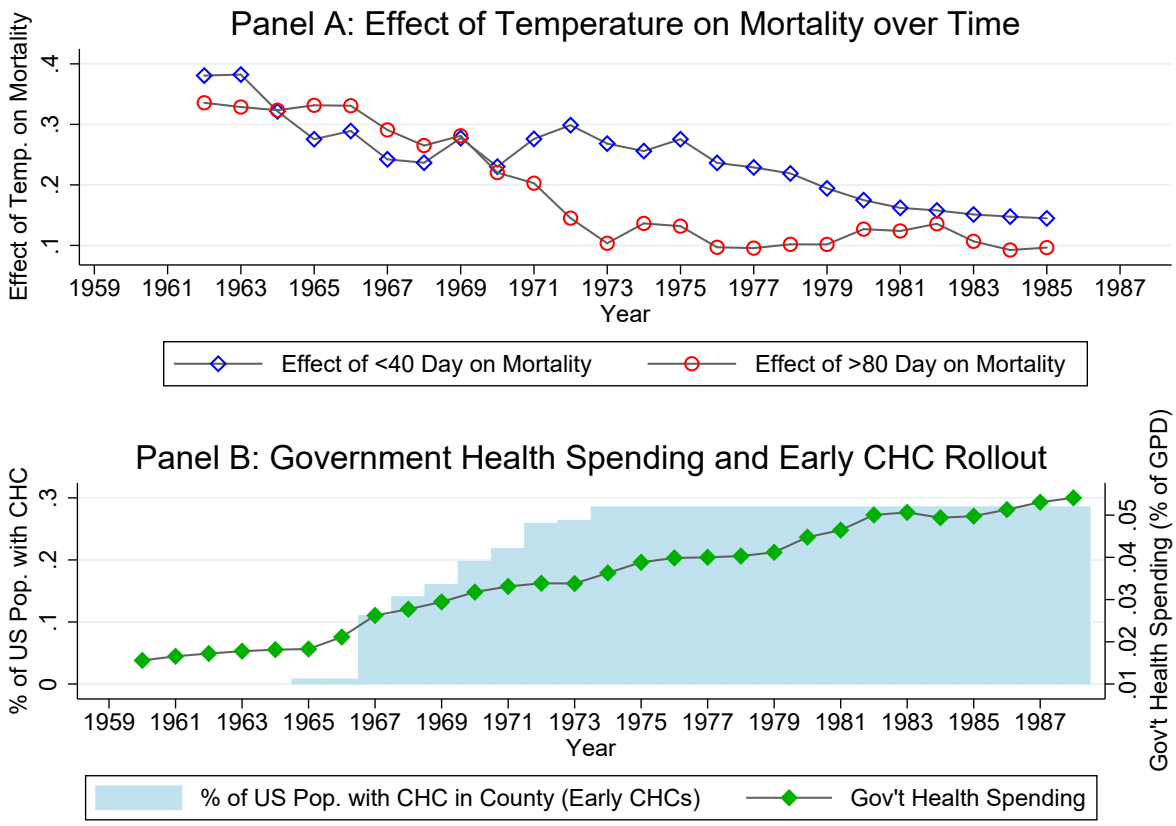
**Sarsons, Heather**, "Rainfall and conflict: A cautionary tale," *Journal of development Economics*, 2015, *115*, 62–72.

**Schlenker, Wolfram and Michael J Roberts**, "Nonlinear temperature effects indicate severe damages to US crop yields under climate change," *Proceedings of the National Academy of Sciences*, 2009, *106* (37), 15594–15598.

**Taylor, Jessamyn**, "Fundamentals of Community Health Centers," *National Health Policy Forum Background Paper*, 2004.

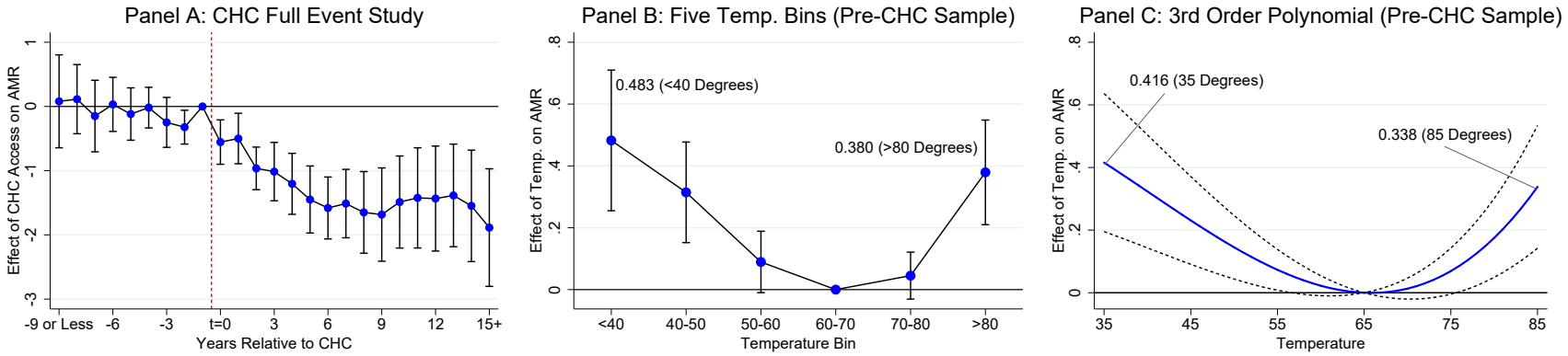
**White, Corey**, "The dynamic relationship between temperature and morbidity," *Journal of the Association of Environmental and Resource Economists*, 2017, *4* (4), 1155–1198.

Figure 1: Trends in $\frac{\partial \text{Mortality}}{\partial \text{Temperature}}$, Gov't Health Spending, and CHC Access



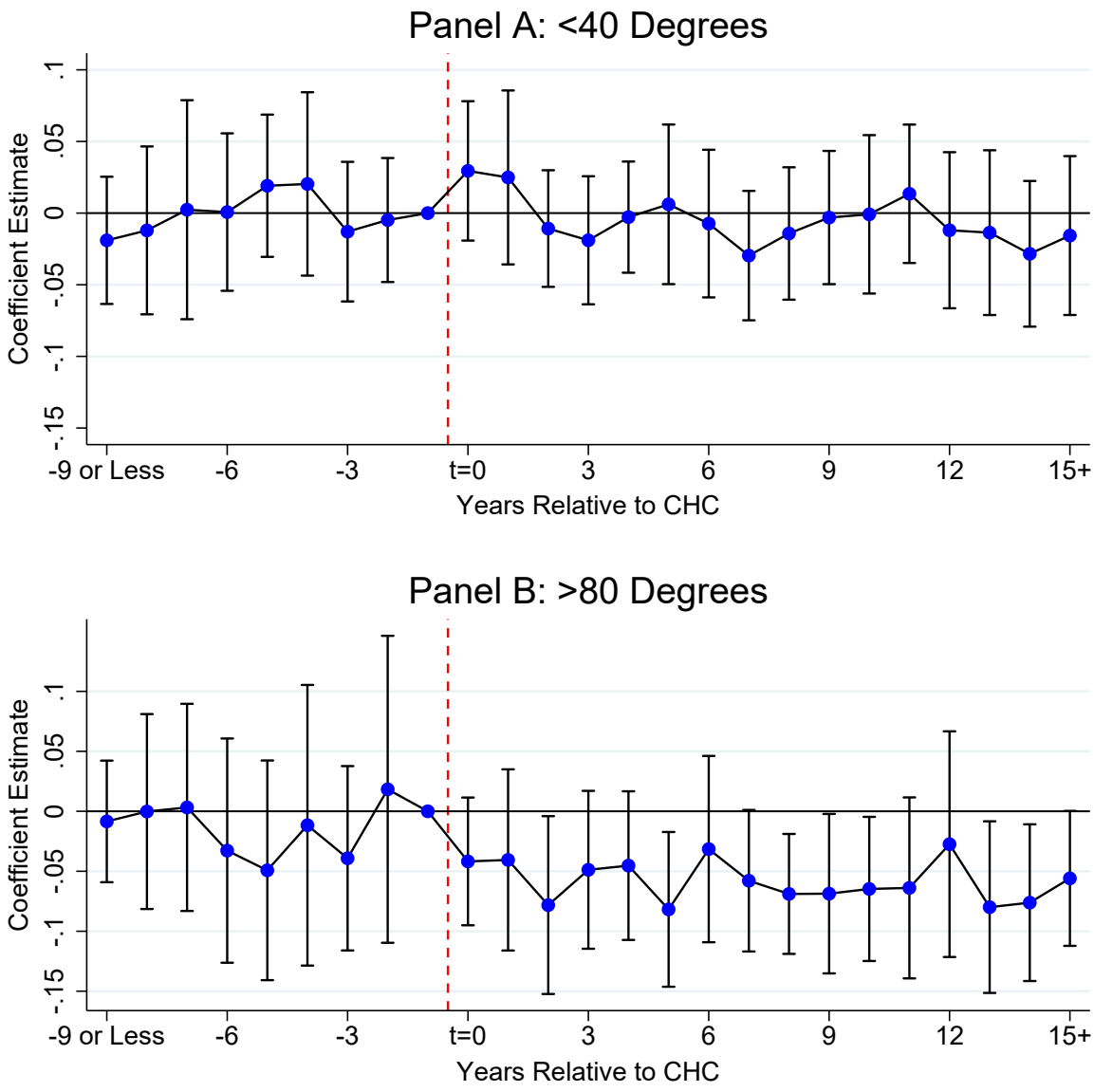

Notes: For Panel A, coefficient estimates for the effects of temperature and mortality are from models described in Section 3.2.1 with five temperature bins and the sample limited to a 7 year period centered around the year in question. For example, the sample for the point labeled "1962" is 1959-1965. All of the estimates are significant at the 5% level; interpretation of the magnitudes of similar estimates is given in Section 4.1. For Panel B, 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, Medicaid, Medicare, government public health expenditures (including the Community Health Center program), government health investments, and other programs. The CHC roll-out variable measures the share of the US population with a CHC in their county (using the fixed 1960 population); the focus is on the first wave of CHC establishments during in 1965-1974 as described in Section 2.2.

Figure 2: Replication Models – Flexible Specifications



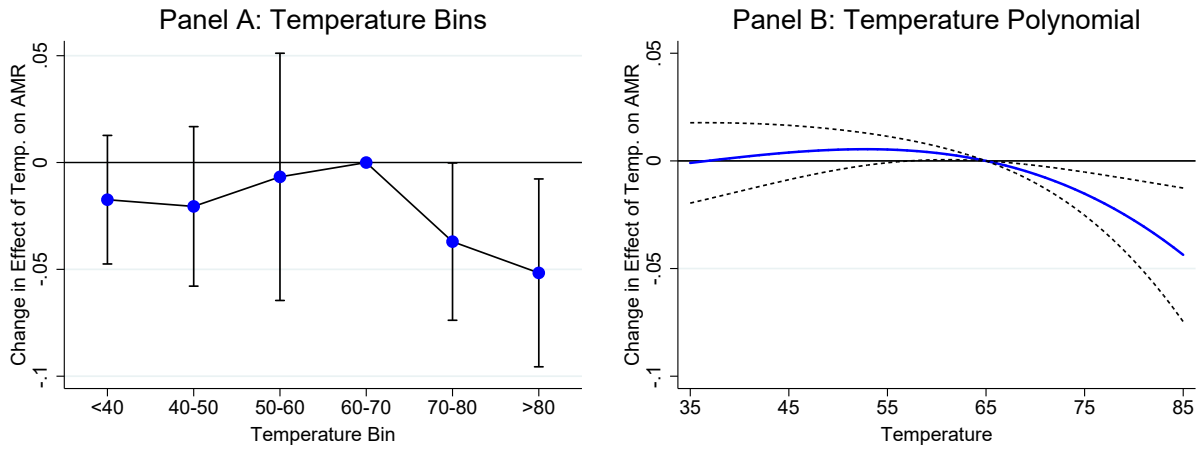

Notes: Panel A represents the effects of access to Community Health Centers (CHCs) on the age-adjusted mortality rate per 100,000 population (AMR); in this plot, period $t = 0$ represents the year in which a CHC was established in a given county. A balanced panel of counties identify all event study coefficients between t-6 and t+14 (i.e., the same number of counties identify these coefficients). Panels B and C represent the effects of temperature on the AMR using a sample limited to counties in which CHCs were established and years prior to CHC establishment (1959-1965); these estimates are intended to serve as a reference point for the interaction estimates presented in Table 3 and Figure 4. 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 daily mean 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: Event Study for the Effects of CHC Access on $\frac{\partial \text{Mortality}}{\partial \text{Temp}}$



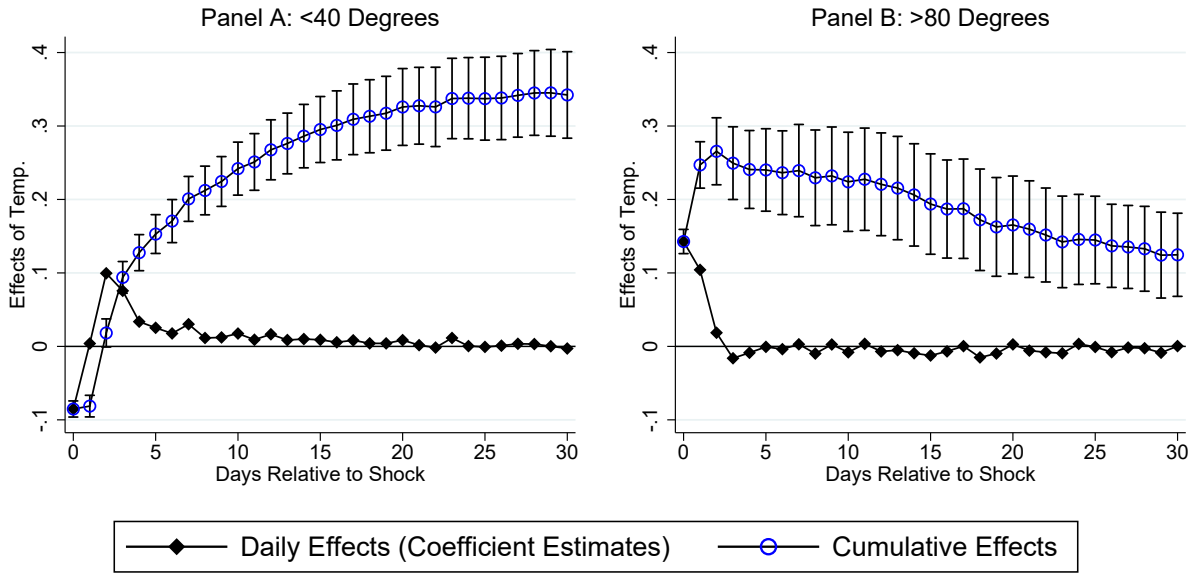

Notes: All estimates in both panels are derived from a single regression with the age-adjusted mortality rate per 100,000 population (AMR) as the outcome variable. Period $t = 0$ represents the first year in which a Community Health Center (CHC) was established in a given county. A balanced panel of 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.

Figure 4: Interaction Model – Flexible Nonlinear Specifications



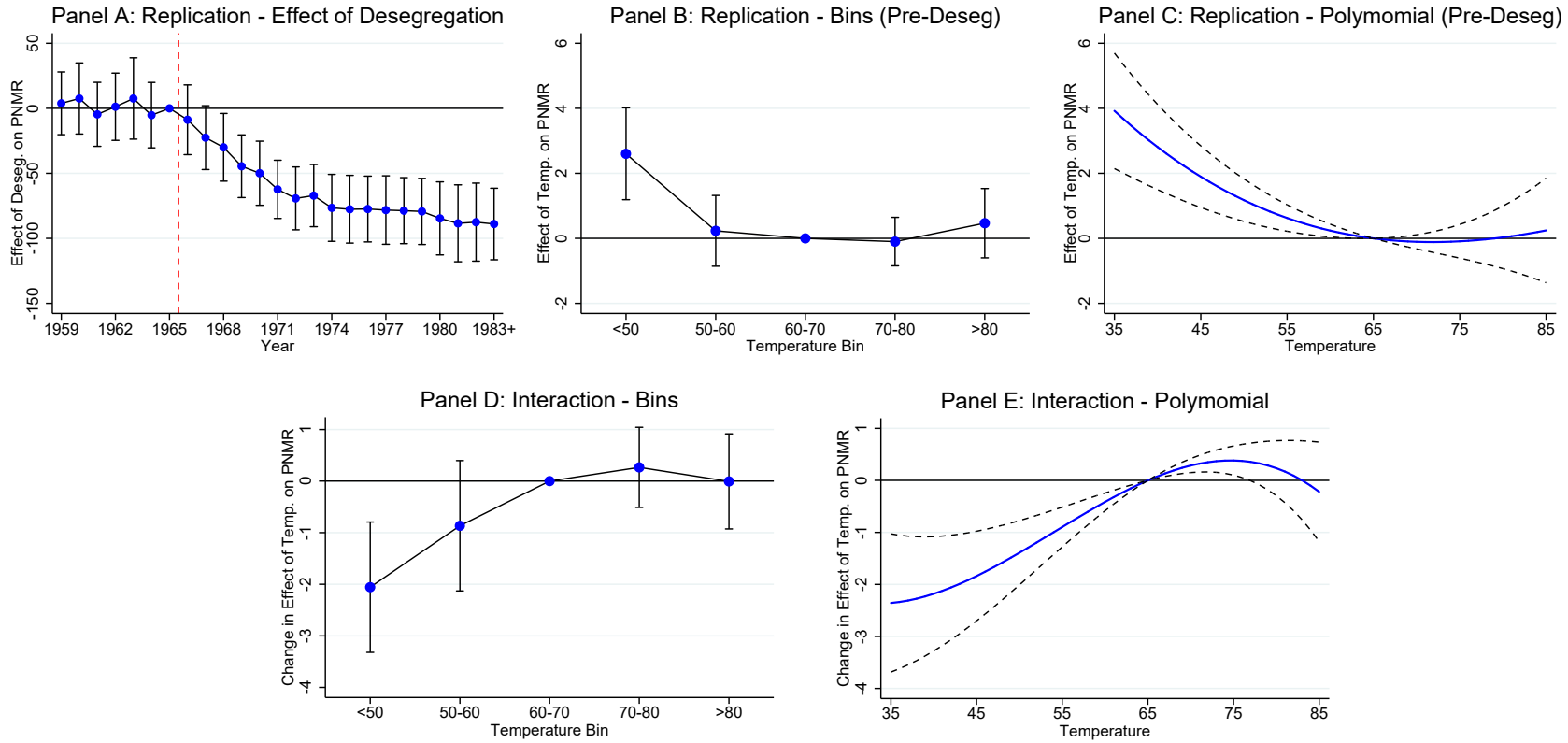

Notes: These estimates represent the effect of Community Health Center (CHC) access on the temperature-mortality relationship (i.e., the estimates of the interaction are plotted). Mortality is measured using the age-adjusted mortality rate per 100,000 population (AMR). Instead of parameterizing temperature using two bins as in Table 3, these plots represent estimates with five temperature bins (Panel A) and a third-order polynomial in temperature (Panel B). In Panel A, each point represents a test of whether CHC access changes the impact of a day with a mean temperature in the given range relative to a day between 60-70°F. In Panel B, each point represents a test of whether CHC access changes the impact of a day with the exact given mean temperature relative to a day with a mean temperature equal to 65°F. The controls included in these specifications are equivalent to those presented in Column 1 of Table 3. Bars and dashed lines represent 95% confidence intervals.

Figure 5: Daily Dynamic Effects of Temperature




Notes: Estimates derived from a distributed lag model as described in Section 5.1. The model estimated at the daily level with 30 lags in each temperature variable, and the outcome is the mortality rate per 100,000 population. The coefficient estimates (labeled "daily effects") represent the change in mortality on a given day due to a temperature shock $t$ days prior, and are estimated directly as the coefficients on the lag terms. The "cumulative effects" represent the sum of all coefficients up to and including $t$, and thus represent the total change in mortality between day 0 and day $t$. All values in both panels are estimated based on a single regression. Bars on the cumulative estimates represent 95% confidence intervals.

Figure 6: Desegregation Analysis




Notes: These plots represent the analysis of Southern hospital desegregation. The outcome in all panels is the post-neonatal mortality rate (PNMR) per 100,000 live births. Panels A-C represent the direct effects of both desegregation and temperature on the PNMR, and mirror Figure 2 from the CHC analysis. Panels D-E represent the interaction between desegregation and temperature, and mirror Figure 4 from the CHC analysis. Bars and dashed lines represent 95% confidence intervals, and standard errors are two-way clustered at the state and year-by-month levels.

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 Resp. 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 separate regression. All use county-level, age-adjusted mortality rate per 100,000 population (AMR) as the outcome variable. The covariates and fixed effects described in Equation (2) are included in all specifications. Standard errors in parentheses are two-way clustered at the county and year-by-month levels.

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

| | (1) | (2) | (3) | (4) | (5) |
|---|---|---|---|---|---|
| | Panel A: DD Estimates | | | | |
| $\text{CHC}^{t\geq 0} \times \text{Temp}^{<40}$ | -0.00294 | -0.00346 | -0.00336 | -0.00324 | -0.0221 |
| | (0.0114) | (0.0115) | (0.0151) | (0.0155) | (0.0101) |
| $\text{CHC}^{t\geq 0} \times \text{Temp}^{\geq 80}$ | -0.0484 | -0.0518 | -0.0499 | -0.0603 | -0.0314 |
| | (0.0201) | (0.0197) | (0.0273) | (0.0288) | (0.0131) |
| | Panel B: Binned Event-Study Estimates | | | | |
| $\text{CHC}^{t\leq -2} \times \text{Temp}^{<40}$ | -0.00207 | -0.00186 | -0.00261 | -0.0109 | 0.00400 |
| | (0.0179) | (0.0179) | (0.0245) | (0.0213) | (0.0184) |
| $\text{CHC}^{0\leq t\leq 4} \times \text{Temp}^{<40}$ | 0.00452 | 0.00436 | 0.00350 | -0.00949 | -0.00627 |
| | (0.0157) | (0.0158) | (0.0215) | (0.0193) | (0.0175) |
| $\text{CHC}^{5\leq t\leq 9} \times \text{Temp}^{<40}$ | -0.00916 | -0.00938 | -0.0106 | -0.0142 | -0.0216 |
| | (0.0185) | (0.0185) | (0.0256) | (0.0211) | (0.0212) |
| $\text{CHC}^{t\geq 10} \times \text{Temp}^{<40}$ | -0.0102 | -0.0108 | -0.0107 | -0.0161 | -0.0255 |
| | (0.0235) | (0.0234) | (0.0310) | (0.0256) | (0.0234) |
| $\text{CHC}^{t\leq -2} \times \text{Temp}^{\geq 80}$ | -0.0116 | -0.00987 | -0.00616 | 0.00812 | -0.0438 |
| | (0.0234) | (0.0233) | (0.0450) | (0.0339) | (0.0267) |
| $\text{CHC}^{0\leq t\leq 4} \times \text{Temp}^{\geq 80}$ | -0.0506 | -0.0514 | -0.0471 | -0.0321 | -0.0586 |
| | (0.0231) | (0.0231) | (0.0370) | (0.0291) | (0.0260) |
| $\text{CHC}^{5\leq t\leq 9} \times \text{Temp}^{\geq 80}$ | -0.0627 | -0.0633 | -0.0531 | -0.0627 | -0.0662 |
| | (0.0220) | (0.0218) | (0.0339) | (0.0354) | (0.0244) |
| $\text{CHC}^{t\geq 10} \times \text{Temp}^{\geq 80}$ | -0.0597 | -0.0628 | -0.0601 | -0.0565 | -0.0795 |
| | (0.0249) | (0.0249) | (0.0393) | (0.0351) | (0.0263) |
| $N$ | 1,094,760 | 1,094,760 | 1,094,760 | 1,094,760 | 1,094,760 |
| Temp × Treated | X | X | | X | X |
| Temp × $\delta_y$ | X | X | X | | X |
| Temp × AC | | X | | | |
| Temp × $\delta_c$ | | | X | | |
| Temp × $\delta_{sy}$ | | | | X | |
| $\delta_{cy}$ | | | | | X |

Notes: Each column in each panel reports coefficient estimates from a separate regression. All use county-level, age-adjusted mortality rate per 100,000 population (AMR) as the outcome variable. 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, "Temp × Treated" include both $\text{Temp}^{<40} \times$Treated and $\text{Temp}^{>80} \times$Treated. The specification in Column 5 includes county-by-year fixed effects in place of all county-level annually-varying covariates, including the post-treatment indicator for presence of a CHC. Standard errors in parentheses are two-way clustered at the county and year-by-month levels. For reference, the baseline estimates for CHC counties in the pre-CHC period (1959-1964) for the effect of a <40°F and >80°F day are 0.241 (s.e.=0.081) and 0.339 (s.e.=0.070), respectively.

Table 4: Estimates by Cause of Death

| | Panel A: Effects of Temperature (CHC Counties, 1959-1964) | | | | | | |
|---|---|---|---|---|---|---|---|
| | All-Cause | Cardiovascular | Cerebrovascular | Cancer | Respiratory | Diabetes | Accidents |
| $\text{Temp}^{<40}$ | 0.242 | 0.0905 | 0.0311 | 0.0144 | 0.0415 | 0.00102 | 0.00367 |
| | (0.0805) | (0.0360) | (0.0142) | (0.00986) | (0.0198) | (0.00503) | (0.00900) |
| $\text{Temp}^{\geq 80}$ | 0.339 | 0.169 | 0.0726 | 0.00151 | 0.0383 | 0.00551 | 0.0106 |
| | (0.0699) | (0.0299) | (0.0147) | (0.0137) | (0.0115) | (0.00435) | (0.00834) |
| $N$ | 8,208 | 8,208 | 8,208 | 8,208 | 8,208 | 8,208 | 8,208 |
| Mean Dep. Var. | 81.48 | 33.30 | 9.79 | 13.32 | 3.97 | 1.49 | 4.03 |
| | Panel B: Interaction Estimates | | | | | | |
| | All-Cause | Cardiovascular | Cerebrovascular | Cancer | Respiratory | Diabetes | Accidents |
| $\text{CHC}^{t\geq 0} \times \text{Temp}^{<40}$ | -0.00294 | -0.00459 | 0.000325 | 0.00104 | -0.00661 | -0.000628 | -0.00268 |
| | (0.0114) | (0.00831) | (0.00309) | (0.00288) | (0.00268) | (0.000825) | (0.00196) |
| $\text{CHC}^{t\geq 0} \times \text{Temp}^{\geq 80}$ | -0.0484 | -0.00967 | -0.0101 | -0.00384 | -0.00911 | -0.00279 | -0.00608 |
| | (0.0201) | (0.0134) | (0.00520) | (0.00561) | (0.00432) | (0.00146) | (0.00369) |
| $N$ | 1,094,760 | 1,094,760 | 1,094,760 | 1,094,760 | 1,094,760 | 1,094,760 | 1,094,760 |

Notes: This table presents estimates by cause of death. For each column, the outcome is the number of deaths in the given disease category per 100,000 population. All-cause mortality is displayed for reference in Column 1. Panel A displays the direct effects of temperature on mortality by cause of death; the sample is limited to CHC counties in the pre-CHC period (1959-1964). Panel B displays the interaction effects, and the specification corresponds to Column 1 of Table 3. Standard errors in parentheses are two-way clustered at the county and year-by-month levels.

# 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: CHC Interaction Model – Five Temperature Bins

| | (1) | (2) |
|---|---|---|
| | Panel A: Simple DiD | |
| $CHC^{t\geq 0} \times Temp^{<40}$ | -0.0174 | -0.0177 |
| | (0.0153) | (0.0152) |
| $CHC^{t\geq 0} \times Temp^{\geq 80}$ | -0.0517 | -0.0536 |
| | (0.0224) | (0.0221) |
| | Panel B: Binned Event Study | |
| $CHC^{t\leq -2} \times Temp^{<40}$ | 0.00439 | 0.00493 |
| | (0.0338) | (0.0338) |
| $CHC^{0\leq t\leq 4} \times Temp^{<40}$ | 0.00375 | 0.00388 |
| | (0.0296) | (0.0295) |
| $CHC^{5\leq t\leq 9} \times Temp^{<40}$ | -0.0132 | -0.0131 |
| | (0.0343) | (0.0341) |
| $CHC^{t\geq 10} \times Temp^{<40}$ | -0.0281 | -0.0281 |
| | (0.0396) | (0.0394) |
| $CHC^{t\leq -2} \times Temp^{\geq 80}$ | 0.00369 | 0.00443 |
| | (0.0306) | (0.0309) |
| $CHC^{0\leq t\leq 4} \times Temp^{\geq 80}$ | -0.0383 | -0.0389 |
| | (0.0271) | (0.0272) |
| $CHC^{5\leq t\leq 9} \times Temp^{\geq 80}$ | -0.0478 | -0.0487 |
| | (0.0299) | (0.0299) |
| $CHC^{t\geq 10} \times Temp^{\geq 80}$ | -0.0536 | -0.0556 |
| | (0.0305) | (0.0303) |
| $N$ | 1,094,760 | 1,094,760 |
| 40-50, 50-60, 70-80 Bins & Interactions | X | X |
| Temp × Treated | X | X |
| Temp × $\delta_y$ | X | X |
| Temp × AC | | 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. Standard errors in parentheses are two-way clustered at the county and year-by-month levels. Both columns use county-level, age-adjusted mortality rate per 100,000 population (AMR) as the outcome variable.

Table A3: Estimates by Age

| | Panel A: Baseline Effects of Temp. | | | |
|---|---|---|---|---|
| | Infant | 1-14 | 15-49 | 50+ |
| $\text{Temp}^{<40}$ | 0.361 | 0.0178 | 0.0240 | 0.866 |
| | (0.362) | (0.0142) | (0.0177) | (0.308) |
| $\text{Temp}^{\geq 80}$ | -0.303 | -0.000271 | 0.0556 | 1.448 |
| | (0.401) | (0.0241) | (0.0341) | (0.279) |
| $N$ | 7920 | 7920 | 8208 | 8208 |
| Mean Dep. Var. | 212.5 | 5.0 | 20.6 | 290.0 |
| | Panel B: Interaction Estimates | | | |
| | Infant | 1-14 | 15-49 | 50+ |
| $\text{CHC}^{t\geq 0} \times \text{Temp}^{<40}$ | 0.0798 | 0.000213 | -0.000232 | -0.0189 |
| | (0.0906) | (0.00238) | (0.00535) | (0.0485) |
| $\text{CHC}^{t\geq 0} \times \text{Temp}^{\geq 80}$ | -0.135 | 0.000731 | -0.00309 | -0.225 |
| | (0.123) | (0.00557) | (0.0116) | (0.0840) |
| $N$ | 1072440 | 1072440 | 1094760 | 1094760 |

Notes: This table presents estimates by age. For each column, the outcome is the number of deaths in the given age group per 100,000 population in that age group. Panel A displays the direct effects of temperature on mortality by age; the sample is limited to CHC counties in the pre-CHC period (1959-1964). Panel B displays the interaction effects, and the specification corresponds to Column 1 of Table 3. Standard errors in parentheses are two-way clustered at the county and year-by-month levels.

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 1 of Table 3, with samples limited to counties within the given propensity score range. Standard errors in parentheses are two-way clustered at the county and year-by-month levels.

Table A5: CHC Interaction Model – Including Lags in Temperature

| | (1) | (2) |
|---|---|---|
| | Panel A: Coef. Estimates | |
| $\text{CHC}^{t\geq 0} \times \text{Temp}_t^{<40}$ | -0.0166 | -0.0172 |
| | (0.0142) | (0.0143) |
| $\text{CHC}^{t\geq 0} \times \text{Temp}_{t-1}^{<40}$ | 0.0175 | 0.0179 |
| | (0.0142) | (0.0142) |
| $\text{CHC}^{t\geq 0} \times \text{Temp}_t^{\geq 80}$ | -0.0350 | -0.0407 |
| | (0.0270) | (0.0266) |
| $\text{CHC}^{t\geq 0} \times \text{Temp}_{t-1}^{\geq 80}$ | -0.0199 | -0.0158 |
| | (0.0265) | (0.0265) |
| | Panel B: Summed Estimates | |
| $\text{CHC}^{t\geq 0} \times \text{Temp}_t^{<40} + \text{CHC}^{t\geq 0} \times \text{Temp}_{t-1}^{<40}$ | 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) |
| $N$ | 1,091,719 | 1,091,719 |
| Temp × Treated | X | X |
| Temp × $\delta_y$ | X | X |
| Temp × AC | | 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). Panel A reports both contemporaneous and lagged coefficients, and Panel B reports the sum of the two coefficient estimates (i.e., the total two-month effect). Standard errors in parentheses are two-way clustered at the county and year-by-month levels. The outcome is the age-adjusted mortality rate per 100,000 population (AMR).

# Online Appendix

# A CHC Analysis Details

## 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 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.

$$\begin{aligned} \text{AMR}_{cym} = &\sum_{j=1}^{J}\sum_{g=1}^{G}\phi^{jg}(\text{CHC}_{cy}^{j} \times \text{Temp}_{cym}^{g}) + \sum_{j=1}^{J}\gamma^{j}\text{CHC}_{cy}^{j} + \sum_{g=1}^{G}\pi^{g}\text{Temp}_{cym}^{g} \\ &+ \sum_{g=1}^{G}\theta^{g}(\text{Temp}_{cym}^{g} \times \text{Treated}_{c}) + \sum_{g=1}^{G}\kappa^{g}(\text{Temp}_{cym}^{g} \times \text{AC}_{sy}) + \sum_{g=1}^{G}(\text{Temp}_{cym}^{g} \times \delta_{y}) \\ &\beta \text{X}_{cym} + \delta_{sy} + \delta_{cm} + \delta_{uy} + \delta_{ym} + \varepsilon_{cym} \end{aligned} \tag{4}$$

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 $\phi^{jg}$ coefficients are of primary interest. The interpretation of one of the $\phi^{jg}$ 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 $t+15$ ($J = 24$).

## A.3 Robustness Checks and Additional Analyses

We present the estimates of a series of robustness checks and additional results in Tables A3 to A5. Table A3 presents estimates of the direct effects of temperature and the interaction effects by age, following the categories used in Bailey and Goodman-Bacon (2015). In general, these estimates align with our expectations: we only find evidence that CHCs mitigated the heat-mortality relationship for individuals aged 50 and older.

Next, consider a test of whether the estimates are sensitive to forcing the treatment and control groups to be more comparable. To construct more comparable samples, 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.[25] 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, 2018), (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.

For the sake of simplicity and precision, our main specifications only include temperature in the contemporaneous month. 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 A5, we additionally include a one-month lag for each temperature variable. The coefficient on the lagged >80°F interaction is insignificant and negative, suggesting the mitigating effects of CHCs on the heat-mortality relationship are not offset by future increases in mortality. Panel B reports the summed contemporaneous and lagged coefficients, and these are qualitatively very similar to our main estimates.

[25] 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, % 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.

# B Desegregation Analysis Details

## B.1 Empirical Design

Our analysis of Southern hospital desegregation proceeds in a similar manner to our analysis of CHCs. We begin with a "replication" model that estimates the direct effects of hospital desegregation and temperature on post-neonatal mortality. Note that while our estimation of the effects of desegregation on post-neonatal mortality is a replication of results from Almond et al. (2006), we are aware of no other studies that estimate the effects of temperature on post-neonatal mortality. Our replication model is described below with subscripts $r$, $s$, $y$, and $m$ representing race, state, year, and calendar month.

$$\begin{aligned} \text{PNMR}_{rsym} = \gamma(\text{NW}_r \times \text{Deseg}_y^{t\geq 0}) + \pi_1 g(\text{Temp}_{sym}) + \pi_2(g(\text{Temp}_{sym}) \times \text{NW}_r) \\ + \mu \text{X}_{smy} + \delta_{rsm} + \delta_{sy} + \varepsilon_{rsym} \end{aligned} \tag{5}$$

In this model, $\text{PNMR}_{rsym}$ is the post-neonatal mortality rate (per 100,000 births) for race $r$, in state $s$, year $y$ and month $m$. An important distinction between this model and our model for CHCs is that here the unit of observation is the race-by-location-by-time, rather than just location-by-time as in the CHC model. Race in defined as either white or non-white. For Southern states in this time period the non-white category is overwhelmingly comprised of African Americans. The sample in this model is limited to Southern states (defined as Alabama, Arkansas, Florida, Georgia, Louisiana, Mississippi, North Carolina, South Carolina, Tennessee, and Virginia).

Our estimates of the effects of desegregation on mortality are derived from a simple DiD design in which we compare non-whites and whites, before and after desegregation. The coefficient of interest is $\gamma$, where $\text{NW}_r$ is an indicator for non-white and $\text{Deseg}_y^{t\geq 0}$ is an indicator for years 1966 and later ($t = 0$ is 1966). If we were not interested in simultaneously estimating the effects of temperature on mortality, this model could be aggregated to the race-by-time level. Estimating temperature effects requires geographical variation, however, and thus we disaggregate to the state level. In principle the model could be estimated at the county level, however because the denominator in the outcome is the annual number of births, the outcome is undefined for some counties in which there were zero births for a particular race-year.

$\pi_1$ and $\pi_2$ represent the effects of temperature, which are allowed to be different for the white and non-white populations. We are primarily interested in the effects of temperature on the non-white population (i.e., $\pi_1 + \pi_2$). $g(\text{Temp}_{sym})$ is either a set of temperature bins

or a 3rd order polynomial in temperature. Because Southern states are warmer on average than the rest of the US, we use the number of days <50°F to represent cold temperatures in the binned specifications. Note that the average number of days <50°F in the South is approximately equal to the number of days <40°F for the entire US. $\mathrm{X}_{cym}$ represents precipitation controls: two indicators for monthly precipitation below the 25th percentile or above the 75th percentile of the state-month distribution respectively.

Identification of $\gamma$ in a difference-in-differences framework requires controlling for unobserved time-invariant differences across race, and unobserved race-invariant differences over time. This is accomplished through race-state-month fixed effects ($\delta_{rsm}$) and state-year fixed effects ($\delta_{sy}$). These controls are specified to facilitate causal identification of $\pi$ as well; in particular, the race-state-month fixed effects control for differences in average temperature and mortality across states, and allow seasonality to vary across both states and races. The state-year fixed effects obviate the need for any additional state-level annually-varying covariates. Standard errors are two-way clustered at the state and year-by-month levels.

### B.1.1 Interaction Model

The model used to estimate the interaction between desegregation and temperature exposure is very similar in spirit to the interaction model for CHCs, and is described below:

$$\begin{aligned}\mathrm{PNMR}_{rsym} = {} & \phi(\mathrm{NW}_r \times \mathrm{Deseg}_y^{t\geq 0} \times g(\mathrm{Temp}_{sym})) + \gamma(\mathrm{NW}_r \times \mathrm{Deseg}_y^{t\geq 0}) \\ & + \pi_1 g(\mathrm{Temp}_{sym}) + \pi_2(g(\mathrm{Temp}_{sym}) \times \mathrm{NW}_r) + g(\mathrm{Temp}_{sym}) \times \delta_y \\ & + \mu \mathrm{X}_{smy} + \delta_{rsm} + \delta_{sy} + \varepsilon_{rsym}\end{aligned} \tag{6}$$

This is essentially a triple-differences model in which the coefficient of interest ($\phi$) is on the three-way interaction. We interpret $\phi$ as the effect of desegregation on the temperature-PNMR relationship. Similar to our model for CHCs, we are again careful to control for fixed differences in the effects of temperature across treated and untreated groups. In this case, race defines the treatment group, and thus $\mathrm{Temp}_{sym} \times \mathrm{NW}_r$ allows for these differences. We also allow for the effect of temperature to vary over time ($\mathrm{Temp}_{sym} \times \delta_y$). Although not included in our main specification, we can also allow the effects of temperature to vary across AC penetration rates ($\mathrm{Temp}_{smy} \times AC_{rsy}$), and the results are insensitive to these additional controls (results available upon request). The precipitation controls and fixed effects are the same as in the replication model.