Essays in Health Economics

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Essays in Health Economics

A dissertation presented

by

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Essays in Health Economics

Abstract

This thesis consists of three essays on topics in health economics. The first essay studies regulation and adoption of new pharmaceutical products in the context of physician decision-making in prescribing. The second essay focuses on socio-economic determinants of health and examines externalities from education on health and the mechanisms behind this relationship. The third chapter explores socio-economic, environmental, and health-related factors contributing to racial disparities in the COVID-19 pandemic burden, arguably one of the most pressing issues at the time this chapter was written.

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To my father Zoran, my mother Zorica, and my brother Milan

Introduction

This thesis consists of three essays on topics in health economics. The first essay studies regulation and adoption of new pharmaceutical products in the context of physician decision-making in prescribing. The second and third essays focus on socio-economic determinants of health in two different settings.

In the first essay, I examine the adoption of untested pharmaceuticals and how physicians respond to decisions made by the Food and Drug Administration (FDA) regarding clinical trials conducted in the pediatric population after initial market entry of the pharmaceutical drug. While FDA approval for the pediatric population at market entry increases the drug's market share relative to no approval, I show that physicians adopt drugs even in settings without FDA labeling information or certification. Even though physicians adopt ex-post ineffective or unsafe drugs, the ex-post effective drugs comprise the majority of FDA-unapproved uses, indicating concordance between physician prescribing and FDA decisions. Subsequent FDA certification or decertification of pediatric uses of drugs have small impacts on physicians' prescribing behavior. However, initial FDA approval meaningfully increases prescribing rates among pediatric patients. I document two reasons for low responses to subsequent FDA decisions about drugs' safety and effectiveness in the pediatric population: (i) high rates of off-label use and market learning prior to subsequent FDA decisions and (ii) large delays between market entry and subsequent labeling changes.

In the second essay, along with my co-authors Jacob Bor, David Cutler, and Edward Glaeser,

we ask whether there exist human capital spillovers on health. We document that people with the same level of education are healthier in more educated places – all-cause mortality declines by 65.6 deaths per 100,000 for each 10 percentage point increase in the share of individuals with a college degree in an area, controlling for individual education, demographics, and area characteristics. Area human capital is also associated with better quality of life and lower disease prevalence. These spillovers increased between 1990 and 2010 and are largest in the least educated areas. We show that almost all of the correlation between area human capital and health can be explained by differences in smoking and obesity rates across areas that are correlated with area human capital; these spillovers in health-related behaviors are partially driven by stricter regulations and stronger informational spillovers in more educated areas, each explaining approximately a quarter of the correlation between area human capital and smoking. We reject sorting on the basis of health and differences in demand and quality for health-related amenities as mechanisms driving human capital spillovers.

Lastly, in the third essay, I examine correlates of the disproportionate burden of COVID-19 among non-white communities in the U.S. At the onset of the COVID-19 pandemic, non-white individuals were 3-4 times more likely to contract COVID-19 and 1.5-2 times more likely to die from COVID-19. Using comprehensive data on COVID-19 cases and deaths by race reported by the CDC, aggregated to counties and combined with extensive county data on socio-economic, health-related, and environmental factors by race, I find that education rates among Black individuals are strongly negatively correlated with Black case rates, but not for any other non-white race. Additionally, rates of public transportation use and household density among Hispanic individuals are important correlates of Hispanic case and mortality rates, but less so for other racial groups. Health-related factors play a smaller role in explaining observed racial disparities in COVID-19 spread, but pre-existing comorbidities are strongly positively correlated with mortality among Black individuals infected with COVID-19.

Chapter 1

Drug Adoption under Uncertain Quality and Impact of FDA (De)Certification for Pediatric Patients

1.1 Introduction

A classic question in regulating product entry is the tradeoff between product quality and delays in valuable products entering the market due to regulatory compliance (Peltzman, 1973). While regulatory review ensures quality standards are met and lower quality products are screened out, it is also costly and time-consuming, which can prevent high-quality products from becoming available to consumers earlier. This question occurs in many industries where certification is needed for market entry, such as cars and airplanes. These tradeoffs are most salient in the case of pharmaceuticals. Regulatory approval by the Food and Drug Administration (FDA) for new drugs might mean delayed access to life-saving drugs or drugs that improve quality of life. On the other hand, lax regulatory requirements may allow unsafe, ineffective, or otherwise harmful drugs to enter the market and be sold

to consumers.

This paper sheds light on the benefits and harms of drug adoption under uncertain quality by: (a) examining the adoption of new drugs in cases with and without FDA approval, (b) evaluating the accuracy of physician prescribing decisions relative to subsequent FDA decisions based on clinical trials, and (c) estimating impacts of FDA certification and de-certification on prescribing controlling for market learning. We focus on pediatric (aged 0-17) drug prescribing for two reasons. First, under the 1999 Pediatric Research Equity Act (PREA), drug sponsors applying for new drug approval must conduct pediatric assessments of pharmacokinetics, pharmacodynamics, safety, and efficacy for every new drug. While these requirements are often deferred or delayed, completion is mandatory, which allows us to observe FDA decisions for different drugs, diseases, and ages. Second, unlike in other pharmaceutical settings where reporting of negative clinical trial findings might be low or biased (Zarin *et al.*, 2019; Oostrom, 2022), results from unsuccessful pediatric clinical trials must be reported to the FDA in order to comply with PREA requirements and are typically added to the drug label. Additionally, physicians are allowed to prescribe drugs off-label, defined as use of drugs in settings other than those indicated by the FDA, with limited legal liability or restrictions by pharmacies. The existence of relatively unrestricted off-label use allows us to observe prescribing patterns in the absence of FDA (de)certification and assess whether it aligns with subsequent FDA decisions.

We collect and combine data from Drugs@FDA, which is a publicly available database maintained by the FDA containing all drug label modifications by drug and date, with data from MicroMedex, one of the statutorily named medical compendia used by Centers for Medicaid and Medicare Services to create a historical record for each new drug approved between 1998-2013. This record contains all diseases and age ranges per disease that a drug was approved for, including dates of approval for each drug-disease-age combination. We call such approval dates “FDA certification events". We further supplement this data with information from the FDA’s Pediatric Labeling Changes database which contains drug

label changes associated with negative clinical trial results by drug-disease-age. We define "FDA decertification events" as drug labeling changes associated with lack of efficacy or safety-related negative results such as finding more or different adverse events relative to adults, contraindications, labeling changes advising against use in pediatric patients, long-term adverse events, or insufficient data for establishing safety.

To measure prescribing for FDA-approved vs. FDA-unapproved uses, we use the Truven MarketScan Commercial Claims and Encounters data from 1996-2013. This data includes health insurance claims for active employees and dependents (including children) for a sample of employer-sponsored plans. Drug claims in MarketScan do not include information on what uses each drug was prescribed for; thus, we define FDA-approved uses of a drug for a given disease as those for which: (i) the patient associated with the drug claim was diagnosed with the disease prior to receiving the drug and (ii) the drug is FDA-approved for treatment of the disease and for the patient's age at the time of the drug claim. We classify all other uses as FDA-unapproved. Since physicians can prescribe drugs for conditions never approved by the FDA, we limit the patient sample to those diagnosed with diseases the drug was indicated for at the time of market entry. This allows us to examine prescribing and rates of FDA-approved and unapproved uses as a function of age rather than disease area.

In our final drug sample of 440 drugs approved between 1999-2013, only 29% of drugs were approved for some pediatric age at market entry. Because of such low pediatric labeling events at market entry, 1 in every 5 pediatric drug claims in our data are for an FDA-unapproved use. Frequently used off-label drugs in children (aged 0-11) include drugs used to treat asthma, skin and respiratory infections, as well as depression and anxiety drugs. 4 out of the top 10 most frequently prescribed off-label drugs among adolescents (aged 12-17) are used to treat mental health conditions, particularly depression and anxiety. Furthermore, up to 10% of drugs record a negative trial result for some pediatric age range. Addition of both negative and positive trial results to the drug label can be significantly

delayed – conditional on FDA (de)certification at some point after initial market entry, the median drug is on the market for 8 years prior to the drug labeling change. Additionally, the average decertification event occurs 1.5 years later than the average certification event, suggesting that potentially harmful uses of a drug take longer to be labeled.

To examine drug adoption rates in the absence of FDA communication about safety and efficacy, we use an event study specification estimated around market entry of new drugs, specifically distinguishing between FDA-approved and FDA-unapproved uses. We find that patient and physicians adopt drugs even in the absence of FDA support (or lack thereof) for its use. Adoption of FDA-unapproved uses is fastest in the first year since market entry and remains constant and statistically significantly positive for up to three years after market entry; there is no de-adoption of FDA-unapproved uses. We also find that FDA-approved uses for pediatric age ranges at market entry are prescribed at twice the rate of unapproved uses. Drug adoption rates for both approved and unapproved uses are highest among treatment naive patients.

These results highlight one side of the tradeoff from regulatory inaction – untested and uncertified products are certainly adopted by patients and physicians. This is not necessarily welfare-reducing, as this could mean that untested but effective and safe drugs are adopted. To examine whether physicians' prescribing decisions are in concordance with subsequent FDA decisions, we estimate an event study specification at initial market entry separately by ex-post outcome documented for the drug in clinical trials, grouping drugs into three categories: ex-post effective, ex-post unsafe or ineffective, and drugs never tested. Though imprecisely estimated, we find that most unapproved uses of drugs are for drugs that are ex-post shown to be safe and effective. However, ex-post unsafe or ineffective drugs are also adopted at a statistically significant positive rate. We find no evidence of de-adoption of these drugs up to three years after initial approval and prior to subsequent FDA decertification. Assuming that the FDA decision is in alignment with a social planner's decision, higher rates of adoption of untested but ex-post unsafe or ineffective drugs would indi-

cate deviations from the social optimum. However, we find that even though prescribing rates for ex-post effective drugs are similar for more and less severe patients (as measured by prior emergency room and hospital utilization), ex-post unsafe/ineffective drugs are prescribed to more severe patients at higher rates. This may suggest that physicians are willing to try drug treatments that may not work for the average clinical trial patient but may work for more severe patients.

Under assumptions of Bayesian physicians with a mean-variance utility acting as perfect agents for their patients (McKibbin, 2020), subsequent FDA (de)certification events may not impact prescribing decisions if market learning already results in very precise physician beliefs prior to subsequent FDA labeling changes and physicians' beliefs are in concordance with the FDA's decision. To examine the empirical effects of subsequent FDA labeling changes on prescribing behavior, we separately estimate an event study specification estimating the impact of positive and negative FDA labeling changes on the probability of new drug claims. We find that subsequent FDA approval decisions increase prescriptions, though imprecisely estimated. FDA decertification events have no impact on prescribing. Given the median time of 8 years between market entry and subsequent labeling changes, our results are consistent with a model where long time periods of off-label prescribing and physician concordance with FDA decisions meaningfully reduces uncertainty around efficacy and safety so that subsequent FDA decisions have smaller impacts on prescribing relative to market learning.[1]

The paper proceeds as follows. Section 1.2 describes the regulatory setting and institutional background for drug approvals and pediatric prescribing. Section 1.3 outlines the empirical framework and event study specifications we estimate. Section 3.2 describes our data in

[1]Our findings are consistent with recent findings estimating the effect of secondary FDA approvals for new diseases prescribing behavior (McKibbin, 2020; Berger *et al.*, 2021). Ody and Schmitt (2019) also estimates the impact of subsequent positive and negative labeling changes for pediatrics and find similar though precisely estimated results due to the inclusion of all drug claims in the analysis rather than just those pertaining to the disease subject to the labeling change and thus may incorporate spillovers of FDA (de)certification across disease areas.

detail and Section 3.3 shows summary statistics. Section 1.6 contains our main findings and Section 3.6 concludes.

1.2 Setting and institutional background

1.2.1 Initial drug approvals and market entry

Regulatory agencies play a crucial role in ensuring that pharmaceutical drugs are safe and effective for use by patients. In the U.S., the Food and Drug Administration (FDA) is responsible for reviewing evidence submitted by drug sponsors to assess the drug's safety and efficacy (Burrows, 2006). Similarly, in the European Union, the European Medicines Agency (EMA) ensures that only safe and effective drugs are approved for use in the pharmaceutical drugs market (EMA, 2023). When reviewing evidence, regulatory agencies consider various factors, including dosage, pharmacokinetics, pharmacodynamics, safety, and efficacy submitted by drug sponsors for approval to enter the pharmaceutical drug market. For the remainder of the paper we will focus on the FDA as the regulator since our data comes from the U.S.[2]

Comprehensive data on various drug quality dimensions comes from clinical trials, which are often randomized, controlled, double-blind studies aimed at assessing the drug's safety and efficacy relative to a placebo or standard of care. As Pease *et al.* (2017) discuss, the FDA's usual requirement for approval includes more than one well-controlled clinical trial, but some of the clinical trial criteria for drug approval can be relaxed depending on the condition for which the treatment is under investigation, the standard of care, or the population size available for clinical trial enrollment (e.g., in the case for rare diseases).[3] The

[2]The regulatory setting in the European Union (EU) is quite similar as in the U.S. For a detailed comparison of the drug approval processes between the U.S. and EU, see Van Norman (2016).

[3]Recent trends suggest that these criteria are often relaxed to ensure faster market entry. For instance, between 2005-2012, over a third of drugs were approved based on a single pivotal trial and 44% were approved based on trials using surrogate endpoints instead of primary endpoints (Downing *et al.*, 2014). Recent studies also show that the FDA Breakthrough Therapy designation has enabled faster market entry by simplifying clinical trial criteria for particularly valuable new drugs seeking approval (Chandra *et al.*, 2022).

cost of conducting a clinical trial is entirely borne by the drug sponsor, which often is a pharmaceutical company and the drug patent holder.[4] The cost of a clinical trial can vary widely, depending on factors such as the complexity of the trial design, number of sites, the number of participants, and the duration of the trial (Moore *et al.*, 2020).

The FDA reviews the submitted evidence on the drug's safety and efficacy and decides whether to approve the drug for market entry. Upon approval, several regulatory features begin. First, the drug sponsor can enter the pharmaceutical drug market and sell the drug to patients, subject to post-marketing surveillance, typically for adverse events (Alomar *et al.*, 2020). Second, a 5-year market exclusivity period begins for the drug, which implies that only the drug sponsor is allowed to enter the market with the newly approved drug. The market exclusivity period may overlap with the drug patent period, but may also extend beyond the patent protection; it guarantees monopoly pricing to the drug sponsor (Kesselheim *et al.*, 2017). Third, the FDA and the drug sponsor determine the indicated use of a drug, also called an indication (discussed in detail below). Lastly, the drug sponsor can begin advertising the drug to patients and physicians for indicated uses only. Compliance with direct-to-consumer and physician advertizing regulations is also under FDA's purview (Li and Gibbs, 2021).

1.2.2 Drug indications

A drug's indication is a comprehensive description of all use cases of the drug for which the FDA has granted approval. The indication usually corresponds to the population in which the drug was tested in clinical trials.[5] Indications intend to highlight what uses are sanctioned by the FDA and act as salient, concrete, and easily accessible guidance for practitioners on cases in which the drug can be safely and effectively used. To achieve this

[4]Oostrom (2022) documents that half of treatment arms for psychiatric clinical trials can be considered as sponsored by industry.

[5]Clinical trials have extensive lists of exclusion and inclusion criteria that patients must meet in order to participate in the study, usually specified in a pre-analysis plan. The indication may be more encompassing than these criteria and typically corresponds to the average patient enrolled in the trial.

goal, the indications are generally included at the start of the drug label and at the start of the drug's package insert. Historically, drug labels have not included approved age ranges because most drugs have traditionally been first approved for adults and the pediatric population has generally been underrepresented in clinical trials (Bourgeois *et al.*, 2014). However, after the Best Pharmaceuticals for Children Act (BPCA) in 2002 and the Pediatric Research Equity Act (PREA) in 1999, pediatric indications have taken off.[6] All drug labels are publicly available in a database easily searchable by drug name called Drugs@FDA (discussed in more detail in Section 1.4.1), which ensures access to accurate and up-to-date information for all patients and practitioners.

As an example of an indication, Figure 1.1 shows the indications section of the drug label for Lexapro (escitalopram), one of the most frequently used antidepressants in the U.S. As Figure 1.1 shows, modern-day indications list the disease area and the ages for which a drug is indicated. For example, Lexapro is indicated for use for acute and maintenance treatment of major depressive disorder in adults and adolescents aged 12-17 years, as well as generalized anxiety disorder, but only in adults.

----------INDICATIONS AND USAGE----------

Lexapro is a selective serotonin reuptake inhibitor (SSRI) indicated for:

- Acute and Maintenance Treatment of Major Depressive Disorder (MDD) in adults and adolescents aged 12-17 years (1.1)
- Acute Treatment of Generalized Anxiety Disorder (GAD) in adults (1.2)

Figure 1.1: *Example indication section of a drug label*

1.2.3 Secondary approvals and indications

Drug sponsors may investigate additional indications beyond those granted at market entry. For instance, they may study and seek approval for a new disease area, patients with a specific treatment history, or a new age group. Each additional approved indication earns

[6]For more details on the regulatory background for pediatric indications, see Ristovska (2020) and FDA's Pediatric Report to Congress on BPCA and PREA.

the drug sponsor 3 years of market exclusivity. However, investigations of a drug's safety and efficacy for an already-approved disease but new pediatric age ranges earn the drug sponsor *at most* 6 months of additional market exclusivity, regardless of the outcome of the clinical trials conducted (i.e., regardless of whether the drug deemed safe and/or effective for children). Under BPCA, a drug sponsor can earn 6 months of market exclusivity if the FDA issues a written request for the drug sponsor to conduct pediatric assessments and the drug sponsor completes the studies outlined in the written request.[7] In addition to these incentives, since 1999 drug sponsors are required to complete a pediatric assessment *for every drug* at the time of market entry, compliance with which offers no market exclusivity rewards. However, this requirement can be deferred or delayed, with drug sponsors frequently citing difficult recruitment as one of the reasons for deferral (Hwang *et al.*, 2019, 2018).[8] Approximately 80% of all completed pediatric studies reported to the FDA are completed under PREA and not BPCA, indicating that compliance with the PREA requirements is a stronger incentive than the additional market exclusivity rewards provided under BPCA (FDA, 2020).

Additionally, any pediatric clinical trials that do not lead to pediatric approval must be reported to the FDA in order to comply with the requirements to conduct pediatric assessments under PREA. What is unique about secondary approvals in pediatrics is that results from unsuccessful pediatric clinical trials are added to the drug label as soon as they are reported to the FDA. While drug sponsors have been required to report trial results (positive or negative) for *any* indication to clinicaltrials.gov within 12 months of trial completion since 2017, only 66% of clinical trials complied with this requirement (Zarin *et al.*, 2019). Furthermore, reported results, particularly from industry sponsors, are biased towards finding safe and effective uses of drugs, suggesting that negative trials in settings

[7]The FDA issues written requests for drugs if it deems they will have "significant health benefits" in the pediatric population (FDA, 2022).

[8]This requirement is waived in cases where the disease rarely occurs in pediatric patients (Akalu *et al.*, 2021).

other than pediatrics (Oostrom, 2022). Thus, reporting of positive and negative pediatric trials provides an attractive setting to examine the impact of FDA certification and decertification of drugs since compliance with reporting negative results is higher in this setting and potential bias in reporting may be eliminated.

1.2.4 Off-label use

A unique feature of the pharmaceutical market is that physicians are allowed to prescribe drugs off-label, defined as use of drugs in settings other than those indicated by the FDA. Despite the potential risks associated with off-label use, physicians have only small legal liability with off-label prescribing, often solely in cases of serious adverse events (Syed *et al.*, 2021). Additionally, when dispensing drugs, pharmacies often do not know what disease the drug was prescribed for by the physicians and thus rarely restrict drug access based on whether the use is consistent with the FDA indication. Despite no restrictions on physician and pharmacy discretion to use or fill drugs used off-label, additional restrictions may be introduced by insurers. For instance, insurers may require physicians to obtain prior authorization from the insurer to use new or expensive drugs by submitting forms detailing the use of the drug to the insurer. Such prior authorization forms often ask the condition for which the drug is used and in theory are designed to screen out medically unacceptable uses of a drug (such as off-label uses without an accepted evidence base) as well as reduce spending (Brot-Goldberg *et al.*, 2023).

1.3 Empirical framework and estimation

1.3.1 Event study at initial market entry

Unlike in other product markets, the existence of off-label use in the market for pharmaceuticals allows us to directly observe whether there exists market learning and its speed in the absence of regulatory certification. Specifically, since off-label use is generally unrestricted for physicians and measurable in the data, we can observe adoption of drugs in the

absence of any FDA communication (certification or decertification) on efficacy and safety for a subset of drugs that are not approved for some or all pediatric age ranges at initial market entry. We can also compare such prescribing patterns to rates of use for pediatric age ranges where the FDA has granted approval at market entry.

To assess the extent of drug adoption with and without FDA certification, we estimate the following event study specifications around the time of market entry of a newly approved drug among pediatric patients who have ever been diagnosed with any of the approved diseases at market entry:

$$onlab_{ijdt} = \gamma_{jd}^{on} + \nu_t^{on} + \sum_{k=-6}^{12} \beta_k^{on} \mathbb{1}(t - E_j = k) + x_{ijt} + \varepsilon_{ijdt} \tag{1.1}$$

$$offlab_{ijdt} = \gamma_{jd}^{off} + \nu_t^{off} + \sum_{k=-6}^{12} \beta_k^{off} \mathbb{1}(t - E_j = k) + x_{ijt} + \varepsilon_{ijdt} \tag{1.2}$$

where i denotes a patient, j denotes drugs, d denotes diseases approved for drug j at market entry, t represents calendar quarters, and E_j denotes the market entry date of drug j.[9] $onlab_{ijdt}$ and $offlab_{ijdt}$ denote our measures of drug demand with and without FDA certification – $onlab_{ijdat}$ refers to whether patient i diagnosed with disease d was prescribed drug j at time t consistent with the drug label at time t, whereas $offlab_{ijdt}$ refers to receipt of FDA-unapproved uses of drug j at time t for patient i diagnosed with disease d. We estimate these models separately for on-label vs. off-label uses since diagnosed patients may receive no pharmaceutical drug treatment and we want to normalize our estimates relative to a denominator (diagnosed patient pool). γ_{jd} denote drug-disease fixed effects and ν_t denote calendar time fixed effects. x_{ijt} include the following controls for patient i's characteristics at time t: age by sex fixed effects, whether the patient has been treated with a drug in the same class as drug j prior to time t, which classifies patients into treatment naive and treatment experienced, whether the patient has had any emergency room (ER) or hospital visits in the 12 months prior to t, and fixed effects for the plan type the patient was

[9] While we refer to j as a drug, newly approved drugs may belong to multiple therapeutic drug classes, which is also included as an identifier but suppressed in the notation for clarity.

enrolled in at time t.[10] As discussed in Section 1.2.4, different insurers may have different policies regarding permitting uses of drugs that are not FDA approved; thus controlling for insurance plans is crucial. The majority of patients in our data do not have plan identifiers, so we only control for plan type, which is populated for the vast majority of patients.

The estimated β_k^{off} coefficients capture whether physicians and patients adopt off-label drug uses (for which no FDA information is provided at market entry), whereas β_k^{on} capture the adoption of FDA-approved uses. These coefficients are identified using variation in the market entry time across drugs. To estimate the models in Equation (1.1) and Equation (1.2), we only use quarters before any subsequent information from the FDA is available for the drug, which isolates the effect of the initial information provided in the drug label at market entry. β_{-1} is normalized to zero at the quarter before market entry; since periods prior to E_j have no drug claims because the drug is not yet on the market, the pre-trends for this specification and any other specification around market entry of a drug will mechanically be zero.[11]

We make strides in assessing whether drug adoption under uncertain quality is valuable by re-estimating Equation (1.2) for drugs and ages with subsequent FDA certification/decertification events where the β_k coefficients are interacted with the outcome of subsequent pediatric assessments:

$$offlab_{ijdt} = \gamma_{jd}^{off} + \nu_t^{off} + \sum_{k=-6}^{12} \beta_k^{off} \mathbb{1}(t - E_j = k) * expost_{jda} + x_{ijt} + \varepsilon_{ijdt} \tag{1.3}$$

where $expost_{jda}$ denotes one of the following three categories: whether the drug j was ex-post approved for use in age a and disease d, ex-post not approved (due to lack of safety or efficacy) but tested in age a and disease d, or never tested for use in age a and disease

[10] Plan types include basic/major medical, comprehensive, exclusive provider organization, health maintenance organization, non-capitated point-of-service, preferred provider organization, capitated or partially-capitated point-of-service, consumer-driven health plan, and high deductible health plan.

[11] Empirically, because of measurement error due to manually matching drug names to drug codes and since drug codes can be reused by drug sponsors/manufacturers for a new drug if prior drugs identified by the same drug code are discontinued, we are able to estimate non-zero pre-trends.

d. The β_k^{off} coefficients estimated separately for each of these categories will thus identify drug adoption at market entry for drugs that are ex-post determined to be safe and effective versus those shown ex-post to be ineffective or have higher rates of adverse events.[12] We use certification and decertification events beyond the time period of our data to classify drugs based on-expost outcomes. Higher rates of adoption of ex-post effective drugs indicates that patients and physicians are good at choosing effective and safe drugs in the absence of FDA certification; higher rates of adoption of ex-post ineffective or unsafe drugs indicates that in the absence of FDA (de)certification physicians may choose suboptimal drugs.

1.3.2 Event study at subsequent FDA (de)certification events

We also examine the extent to which subsequent FDA certification events i.e., approvals expand the drug's market size in the approved age and disease group as well using a similar event study approach as in Equation (1.1) and Equation (1.1):

$$rx_{ijdt} = \gamma_{jd} + \nu_t + \sum_{k=-6}^{12} \beta_k \mathbb{1}(t - A_{jdt} = k) + timeonmkt_j + x_{ijt} + \varepsilon_{ijdt} \tag{1.4}$$

where A_{jda} denotes the FDA approval date for drug j, disease d, and age a. In addition to patient characteristics x_{ijt}, we also control for the time drug j has been on the market, denoted by $timeonmkt_j$. This intends to proxy as a control for the extent of market learning due to off-label use prior to the drug's subsequent approval. Unlike Equation (1.3), which uses data on certification and decertification events to classify drugs into ex-post outcome categories, we only use certification events that occur during the span of our data. We also estimate as similar event study model for additions of negative clinical trial results to the drug label, i.e., decertification events in order to capture potential decreases in the drug's market share:

$$rx_{ijdt} = \gamma_{jd} + \nu_t + \sum_{k=-6}^{12} \beta_k \mathbb{1}(t - D_{jdt} = k) + timeonmkt_j + x_{ijt} + \varepsilon_{ijdt} \tag{1.5}$$

[12]Since there are no subsequent certification or decertification events for already approved uses of a drug, all drug claims with ex-post clinical trial outcomes are off-label.

where D_{jda} denotes the date the negative clinical trial result for drug j, disease d, and age a was added to the drug label.

Under assumptions of Bayesian physicians who update rationally in response to market learning and FDA communications about drug safety and efficacy, as well as under assumptions of physicians with a mean-variance utility function acting as perfect agents for their patients, FDA certification events should increase prescribing since they reduce uncertainty even if physicians are correct about drug efficacy (McKibbin, 2020). On the other hand, FDA decertification events also reduce uncertainty, but may decrease prescribing if physicians do not have accurate beliefs over drug efficacy. In such a model, if market learning already results in very precise physician beliefs prior to subsequent FDA approvals or labeling changes, additional FDA communications about drug safety and efficacy will have a negligible impact on prescribing if physicians have beliefs about drug safety and efficacy consistent with the FDA's decision.

1.4 Data and definitions

1.4.1 Sample drugs

We obtain a list of all drugs receiving their initial FDA approval for any age range and indication between 1998 and 2013 from Drugs@FDA, which is a publicly available database maintained by the FDA containing all FDA approvals, drug label modifications, and other FDA submissions by drug and date. We limit the sample to drugs in the 1998-2013 period because: (i) new indications and pediatric approvals were reliably recorded and time stamped starting in 1998, and (ii) 2013 is the end of the insurance claims data used to measure pharmaceutical demand (discussed below). Since drug names are not consistent across FDA submissions, we create a crosswalk between the 690 drug names gaining FDA approval and 661 unique active ingredients. 62 active ingredients were excluded because

the active ingredient was approved before 1998.[13] 14 drugs were excluded because they were permitted to be used over the counter at some point during our data, which makes it difficult to measure demand in our data. We will henceforth use the terms “active ingredients" and “drugs" interchangeably, even though there can be multiple drugs with separate indications and clinical trials for the same active ingredient.[14]

1.4.2 Approved ages and indications

Each active ingredient was mapped to all indications and ages approved for use by the FDA as of March 2023 using the MicroMedex database, which is one of the statutorily named medical compendia by Centers for Medicaid and Medicare Services (CMS). We include all indications approved for an active ingredient, regardless of whether they were approved for a specific formulation, route of administration, dosage, or drug.[15] 11 active ingredients in our sample did not have indication information available, but the remaining 574 active ingredients were mapped to 958 indications. Among these indications, 30 were excluded because they were used to assist in diagnostic or imaging procedures, 42 were procedure-related (e.g., for post-operative or pre-operative care), and 78 were prophylactic, all of which cannot be reliably identified in the data. The final sample contains 511 active ingredients mapped to 781 indications.

MicroMedex does not list the dates of FDA approval for different ages and diseases. However, Drugs@FDA provides a historical record of changes to the drug label, including changes in the indication. Following Berger *et al.* (2021), we manually review these records for our sample drugs to determine ages approved for different diseases in the indication

[13]Entries in the Drugs@FDA database are often manually entered by drug sponsors or FDA staff. If drug names differ across FDA submissions for the same active ingredient, it is possible to observe a drug name first approved in 1998 or after even though the same active ingredient was entered under a different drug name prior to 1998.

[14]For example, depending on whether the drug is administered orally or intravenously, it might have a different drug name and clinical trials despite having the same active ingredient.

[15]This implies that we will not be able to identify dosage-based, formulation-based, and route-based off-label use in our data, leading to an underestimate of the actual off-label use.

and dates of approval for each disease and age pair. This allows us to observe what ages and diseases were approved for a drug at market entry versus what diseases and age groups are added to the indication at a later point.

Indications can be quite detailed (e.g., whether to use in treatment-naive vs. experienced patients, specific disease sub-types etc.). Such specificity of indications are not easily identifiable in our data, so we map indications to coarser disease categories that ignore the patient's treatment experience, disease sub-types and other similar details of the indication and can plausibly be mapped to ICD-9-CM diagnosis codes that are used to identify diseases in the data discussed below.[16] We define 310 such diseases among 781 indications.[17]

1.4.3 Tested ages and indications

Information on pediatric events triggering a drug label change for drugs approved between 1998-2013 comes from FDA's Pediatric Labeling Changes database.[18] In addition to approvals, this database contains a list of negative results for any pediatric populations, such as: finding more or different adverse events than adults, occurrence of serious side effects or contraindicated/not recommended uses in any pediatric populations due to serious side effects or long-term adverse effects, insufficient data for establishing safety, and efficacy not demonstrated in clinical trials.

We define events associated with ineffective drugs as those where efficacy was not demonstrated in clinical trials.[19] Events associated with unsafe drugs include those where the

[16]This method of mapping drugs to indications and dates of approval closely follows methods used by Berger *et al.* (2021).

[17]Since multiple indications can be mapped to the same disease, and approved age ranges across indications may differ, we take the widest possible age range as approved for each disease (across indications). This biases us towards considering more drug uses as on-label and away from considering them off-label, leading to an underestimate of off-label use.

[18]This database contains any positive or negative labeling changes for any drugs on the market since 1998-present, not only for newly approved drugs. However, since our estimation strategies use variation across drugs, we use the same sample of drugs first approved between 1998-2013 for estimating the impact of FDA labeling changes at initial approval vs. at times after initial market entry.

[19]If efficacy was demonstrated at some subsequent point in time for the same disease, we do not consider

reported clinical trials found more or different adverse events relative to adults, contraindications, labeling changes advising against use in pediatric populations, additions of serious long-term adverse events, and insufficient data for establishing safety. For the purposes of this paper we exclude any events related to changes in dosing regimen or pediatric formulation release, which excludes 27 events and 14 drugs, because dosage information is typically bundled with safety/efficacy trials and is more difficult to determine changes in prescribed dosages in the data since dosages can more easily be adjusted outside of observable health care settings (e.g., taking half a pill or half an injection if a smaller dosage is recommended).

1.4.4 Drug claims

We use Truven MarketScan Commercial Claims and Encounters data from 1996-2013 to determine drug claims for sample drugs. This data contains all health insurance claims for active employees and dependents while insured through a sample of employer-sponsored plans. The data encompasses a large set of enrollees – historically, the data has included 500+ million claims a year from approximately 100 plans. Importantly, it includes health insurance claims of any dependents of employees, which typically include children of employees. Patient age is included on all insurance claims in the data. We observe all emergency room visits, inpatient stays, outpatient visits, and pharmacy claims for enrollees in the data.

Drugs were identified in the prescription drug claims and medical claims. Prescription drug claims include drugs obtained at pharmacies whereas medical claims include drugs administered by a physician or in an inpatient setting (e.g., cancer drugs, biosimilars, and other injectable drugs). Each active ingredient in our data was manually mapped to National Drug Codes (NDCs) used to identify pharmacy claims for sample drugs.[20] HCPCS

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the event as negative.

[20]To minimize measurement error, NDC codes need to be recorded in the RedBook drugs database on or after the initial approval year for each drug.

codes were used to identify drugs in the medical claims, which were also similarly manually mapped to active ingredients. Unique drug claims were identified by unique patient ID, NDC code, refill number, date prescription was filled, days' supply, and quantity, whereas unique physician/hospital administered drugs were identified by patient ID, HCPCS code, quantity, and date drug was administered.[21] A small subset of sample drugs was excluded because there were no NDC/HCPCS codes identifying them or because there were zero drug claims for those drugs in the data. To estimate demand for drugs, we use all drug claims for sample drugs filled by pediatric patients, defined as aged younger than 18, yielding a sample of 25.8 million drug claims among pediatric patients between 1996-2013.

1.4.5 Defining FDA-approved and unapproved (off-label) uses

The drug claims in MarketScan do not include information on what uses each drug was prescribed for. Thus, on-label vs. off-label uses cannot be directly identified just based on the drug claim. To identify uses for which a drug might be prescribed, we use diagnosis codes reported in inpatient admissions, outpatient claims, facility claims, and inpatient services for each patient with a drug claim for a drug in our sample. To ensure that we have enough information on each patient to determine potential uses for a drug claim, we only classify drug claims as on-label or off-label where the patient had at least 12 months of medical data prior to the prescription.[22] Lastly, we only consider drug claims occurring after the patient was diagnosed with the indicated condition at market entry.

We additionally limit our sample of patients and drug claims for patients diagnosed with the initial disease that a drug was approved for. Because drugs can be used to treat diseases not approved by the FDA, this criterion ensures that we examine the impact of FDA decisions on age-based off-label use rather than disease-based off-label use. In other words, we only examine off-label uses for already-approved diseases but in ages without FDA

[21] We cannot observe drugs that were prescribed but never filled by the patient.

[22] This medical enrollment periods need not be continuous.

approval.

For a drug claim to be classified as FDA-approved, it must satisfy the following criteria: (i) the patient associated with the drug claim must have been diagnosed with the indicated disease prior to the date associated with the drug claim, and (ii) the drug must be FDA-approved for treatment of the indicated disease for the patient's age at the time the prescription was filled or administered. This implies that drug uses not approved by the FDA but observable in the data include: (i) drug claims to ages never approved by the FDA and (ii) drug claims to ages not FDA-approved at the time of the drug claim, but approved at a later point.

1.5 Summary statistics

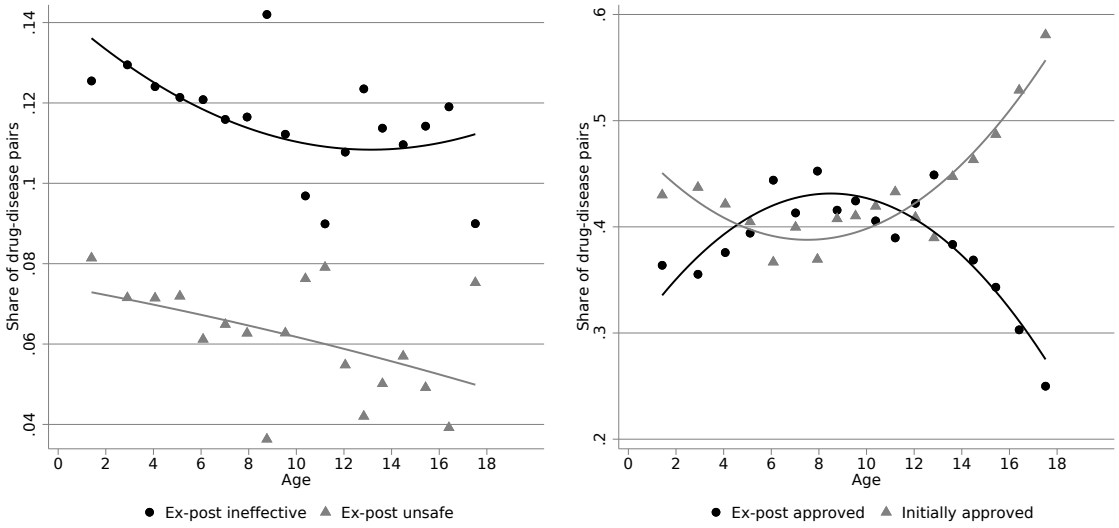
1.5.1 FDA certification and decertification rates across drugs

Our final drug sample contains 440 unique drugs and 852 approved drug-disease combinations, of which 624 were approved at market entry. Almost all indicated drug-disease combinations at initial market entry are approved for the adult population at the time of entry, defined as 18 years of age or older.[23] However, only 29% (180/624) of drug-disease combinations are approved for some subset of the pediatric population at market entry. An additional 132 drugs (21%) are approved for at least some at some point after entering the market without any pediatric approval and 10% of drugs have a negative labeling change for some pediatric age range.

Figure 1.2 shows the probability of initial approval, subsequent approval (certification), subsequent decertification associated with drug inefficacy, and subsequent decertification associated with adverse events as a function of pediatric age. All figures control for disease fixed effects and thus compare the drug certification and decertification probabilities within

[23] A small share of drugs, especially for those with high pediatric disease burden, were initially approved for the pediatric population only.

diseases but across drugs.



Note. *The left sub-figure plots the share of drug-disease pairs having a finding of inefficacy or lack of safety after initial market entry as a function of pediatric age. The right sub-figure plots the share of drug-disease pairs with FDA approval at initial market entry and share of drug-disease pairs with FDA approval at some point after initial market entry. The sample includes all new drugs approved by the FDA between 1998-2013 and includes all initial indications that were FDA-approved. Both figures include disease fixed effects. Data on indications and ages associated with certification and decertification events comes from MicroMedex database, Drugs@FDA, and FDA's Pediatric Labeling Changes database.*

Figure 1.2: *Probability of positive and negative events by age across drug-disease combinations*

The left sub-figure in Figure 1.2 shows that the highest rate of recording an ineffective result occurs between ages 2-6, and the probability of having a decertification event associated with drug inefficacy decreases with age. This sub-figure also shows that conditional on disease area, 5-8% of newly approved drugs are found to be unsafe in some pediatric age at some point after market entry; the share of drugs with decertification events associated with drug safety also decreases linearly with age.

The right sub-figure in Figure 1.2 shows that conditional on disease area, 50-60% of drugs are initially approved in some pediatric age and 40-60% are approved at some point after market entry. Since FDA approval is generally a terminal state, there is a negative relationship between initial FDA approval and subsequent FDA approval. The probability of

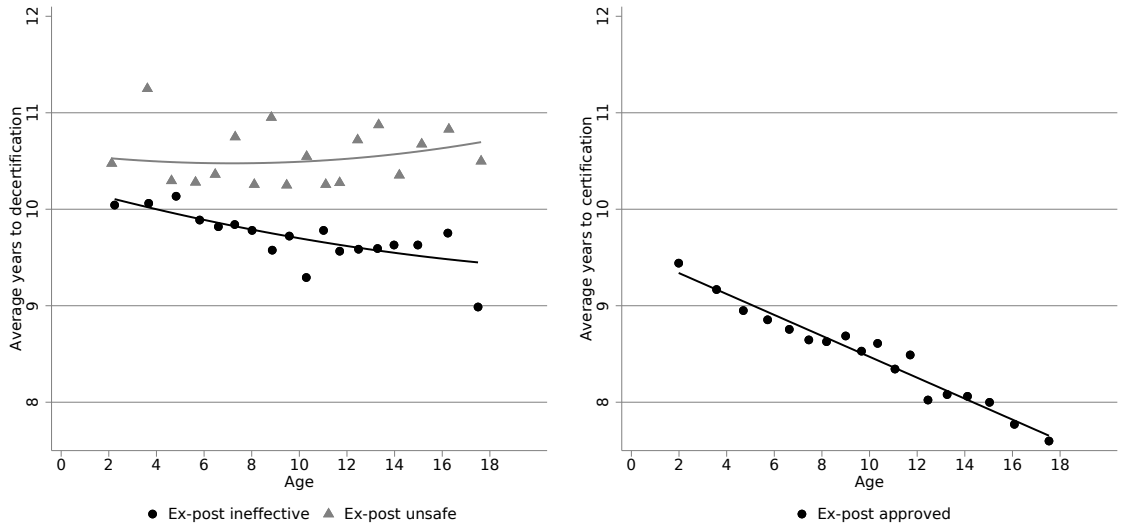
initial approval increases with age, corroborating patterns in our collected data showing that expansions of indications to pediatric age ranges typically first target adolescent ages (12-17), and subsequent indications examine safety and efficacy in younger ages. The non-linearities associated with the probability of initial approval as a function of age may be due to measurement error – when collecting the data on approved ages, we assume that a drug is approved for ages 0-18 if pediatric approval is mentioned but age ranges are not specifically mentioned.[24]

1.5.2 Timing of FDA certification and decertification events

Figure 1.3 shows the average number of years elapsed between initial drug approval and subsequent FDA certification and decertification events as a function of age for drug-disease pairs experiencing such an event. The left sub-figure in Figure 1.3 plots the average years elapsed since initial market entry for drug-disease pairs eventually documenting an ineffective or unsafe finding, whereas the right sub-figure plots the average time between initial drug approval and subsequent FDA approvals. As before, all figures control for disease fixed effects.

Figure 1.3 shows that, on average, a significant amount of time passes between initial drug approval and subsequent FDA certification or decertification events – less than 25% of all drug-disease pairs record a certification or decertification event within 5 years of initial market entry. This indicates that prior to any FDA decisions for unapproved pediatric age ranges that eventually end up getting tested, physicians can prescribe, experiment with, and learn about efficacy and safety for unapproved drugs for at least 8 years for the median drug. This also allows for a significant amount of time for the establishing of prescribing practices that may or may not be affected by subsequent FDA decisions. This is quite a meaningful duration given that physician decision-making as pertaining to prescribing has been established as "sticky", persistent, and inertial (Phelps, 2000; Janakiraman *et al.*, 2008;

[24]For example, if a drug label states "approved for children and adults", but does not specify ages for children, we assume it is approved for all pediatric ages.



Note. *The left sub-figure plots the average number of years since initial market entry for drug-disease pairs with a finding of inefficacy or lack of safety after initial market entry as a function of pediatric age. The right sub-figure plots the average number of years since initial market entry for drug-disease pairs with FDA approval at some point after initial market entry. The sample includes all new drugs approved by the FDA between 1998-2013 and includes all initial indications that were FDA-approved. Both figures include disease fixed effects. Data on indications and ages associated with certification and decertification events comes from MicroMedex database, Drugs@FDA, and FDA's Pediatric Labeling Changes database.*

Figure 1.3: *Average years elapsed between initial drug approval and positive and negative events by age across drug-disease combinations*

Chandra *et al.*, 2011).

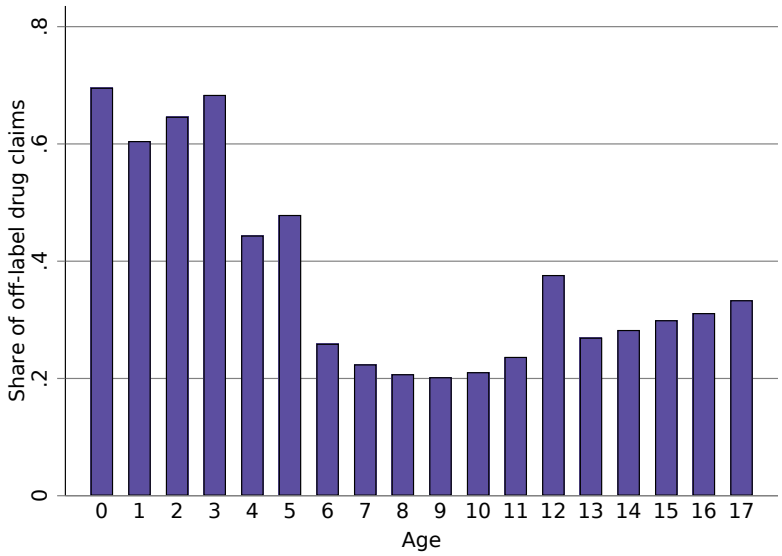
Second, Figure 1.3 indicates that across pediatric age ranges, both certification and decertification events tend to occur sooner for older pediatric age ranges relative to younger ages, which is a function of pediatric indication expansions first targeting adolescents and then younger pediatric patients. Additionally, the left sub-figure in Figure 1.3 shows that results pertaining to inefficacy tend to be submitted to the FDA sooner than events pertaining to safety. Decertification events related to safety and efficacy for pediatric patients younger than 5 years may take up to 10 years after market entry to be reported to the FDA. This may be a function of the our definition of safety-related decertification event, which includes adverse events related to long-term safety, which mechanically requires patients to be followed for longer periods of time. Additionally, it may also reflect difficulty with recruitment of younger pediatric patients for participation in clinical trials.

Lastly, certification events tend to occur sooner than decertification events. This could reflect endogenous responses by the drug sponsor such as delays in reporting due to fears of negative market response or attempts to further investigate the drug in modified clinical trials until a successful result is achieved.

1.5.3 Rates of off-label use

Among drug claims to pediatric patients, 22% of all drug claims are for off-label uses. This estimate is in line, if not lower, with other studies measuring pediatric off-label prescribing in various settings (Corny *et al.*, 2015; Yackey *et al.*, 2019; Cuddy and Currie, 2020). Figure 1.4 shows that the share of drug claims prescribed to pediatric patients falls with age and is highest among patients younger than 5. This result may be partly mechanical – as shown in Section 1.5.1, patients younger than 5 are least likely to have a drug approved. There is also a slight increase in the share of off-label drugs at age 12 since many sponsors initially test drugs among adolescents, often defined as 13-17 year olds.

Table 1.1 and Table 1.2 show the drugs with the highest on-label and off-label drug claims



Note. *This figure plots the rates of FDA approved and unapproved uses of new drugs entering the pharmaceutical drug market during 1998-2013. Drug claims were measured among pediatric patients (younger than 18 years of age) diagnosed with the FDA-approved diseases at initial market entry in the 1998-2013 MarketScan insurance claims data base. Data on approved indications and ages comes from MicroMedex database and Drugs@FDA. For a definition of FDA-approved vs. unapproved uses, see section Section 1.4.5.*

Figure 1.4: *Share of drug claims that are off-label by pediatric age*

(as a share of total drug claims to the drug) prescribed among children (0-11 years of age) and adolescents (12-17 years of age). Since many drugs have few pediatric prescriptions, to construct this table we only consider drugs in the top decile (>35,000 drug claims) in terms of number of pediatric drug claims.

As shown in Table 1.1, for pediatric patients younger than 12, the most frequently used on-label drugs mainly include drugs used to treat attention deficit hyperactivity disorder (ADHD), allergies, asthma, and skin and ear infections (idiopathic urticaria and otitis media/externa). Among adolescents, the most frequently used on-label drugs include drugs indicated for treatment of asthma, allergies, ADHD, and skin and ear infections, but also include drugs indicated for the treatment of acne and autoimmune conditions such as ankylosing spondylitis, Crohn's disease, psoriasis, rheumatoid arthritis, and ulcerative colitis. With the exception of infliximab, all of the top on-label drugs among both children and adolescents were approved for some pediatric age range at initial market entry.

Table 1.2 shows the ten most frequently used off-label drugs among children and adolescents. Two out of the ten drugs with the highest off-label use rates among children are among the most frequently used on-label drugs in adolescents and are used to treat asthma (budesonide + formoterol and levalbuterol), suggesting that some share of off-label use in children is for drugs approved in adolescents but not younger ages. Additionally, two of the ten drugs with the highest off-label drug claims among children are used to treat depression or anxiety (citalopram and escitalopram). Similarly, among adolescents, mental health drugs are among the most frequently used drugs with highest off-label shares, consistent with studies on off-label use rates across disease areas. In fact, four out of the top ten are used to treat mental health conditions (citalopram, duloxetine, escitalopram, and aripiprazole). While escitalopram was approved for treatment of depression in adolescents in the last four years of our data, citalopram has never been approved for treatment of depression in any pediatric age. Aripiprazole was approved for pediatric treatment of autism, bipolar disorder, Tourette's, and schizophrenia in the last 7 years of our data,

Table 1.1: *Top ten on-label drugs for children and adolescents*

Children (0-11 years of age)		
Drug name	*% on-label*	*Indicated for*
lisdexamfetamine	90%	ADHD
levothyroxine	88%	Hypothyroidism; Myxedema coma; Thyroid cancer
levocetirizine	88%	Idiopathic urticaria; Allergic rhinitis
ciprofloxacin + dexamethasone	86%	Otitis media and externa
fluticasone + salmeterol	86%	Asthma; Chronic bronchitis
dexmethylphenidate	86%	ADHD
ciprofloxacin + hydrocortisone	84%	Otitis externa
atomoxetine	84%	ADHD
oxcarbazepine	79%	Partial seizures
ciclesonide	70%	Asthma; Allergic rhinitis
Adolescents (12-17 years of age)		
Drug name	*% on-label*	*Indicated for*
clindamycin + tretinoin	95%	Acne
budesonide + formoterol	93%	Asthma
adapalene + benzoyl peroxide	93%	Acne
ciprofloxacin + hydrocortisone	93%	Otitis externa
ciprofloxacin + dexamethasone	92%	Otitis media and externa
dexmethylphenidate	90%	ADHD
infliximab	90%	Ankylosing spondylitis; Crohn's disease; Plaque psoriasis, Psoriatic arthritis; Rheumatoid arthritis; Ulcerative colitis
levalbuterol	89%	Asthma
atomoxetine	87%	ADHD

Note. *Drug claims were measured among pediatric patients (younger than 18 years of age) diagnosed with the FDA-approved diseases at initial market entry in the 1998-2013 MarketScan insurance claims data base. Percent on-label refers to the share of all drug claims that are for FDA-approved uses, conditional on having at least 1000 drug claims per drug. Data on approved indications and ages comes from MicroMedex database and Drugs@FDA. For a definition of FDA-approved vs. unapproved uses, see section Section 1.4.5.*

and duloxetine was never approved for any pediatric age ranges during the span of our data (although it was approved for anxiety and fibromyalgia after 2013). The high rate of off-label use among these mental health drugs in adolescents indicates that either they are being used for treatment of eventually-approved indications prior to approval or are used for treatment of other conditions not approved by the FDA. To solely isolate off-label uses for indicated conditions but for unapproved ages, our subsequent analyses exclude off-label uses among patients never diagnosed with an indicated condition, as discussed in Section 1.4.5.

1.6 Results

1.6.1 Adoption of FDA-approved and unapproved uses at initial market entry

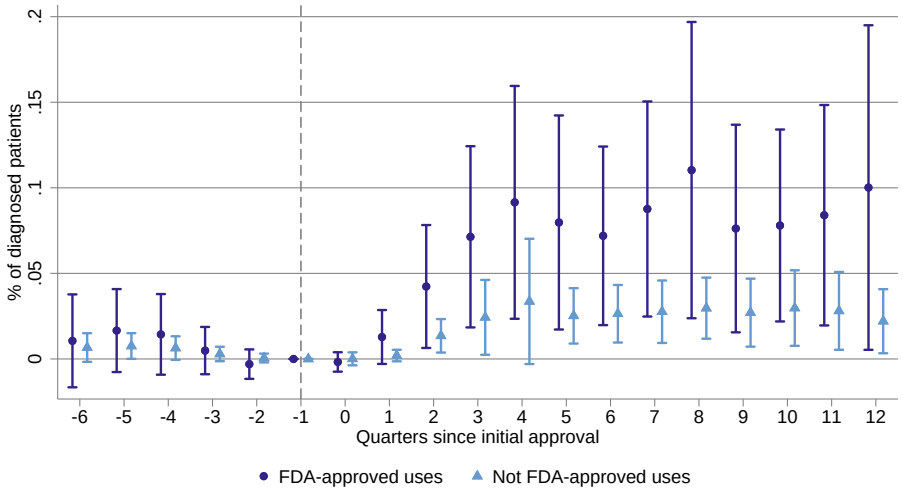
Figure 1.5 shows the effect of *initial* FDA approval on the probability of a pediatric patient diagnosed with an FDA-approved disease receiving a newly approved drug and the drug adoption rate for newly approved drugs in FDA-unapproved ages. This figure separately plots the effect of initial FDA approval on FDA approved uses vs. off-label (FDA unapproved) uses, which can include untested, unproven, and unapproved uses of the drug, as well as those that are ex-post tested, regardless of the ex-post outcome.

As shown in Figure 1.5, conditional on patient characteristics, patients and physicians adopt a drug even in the absence of FDA certification for the use. The rate of FDA-unapproved uses steadily increases during the first year after initial market entry and remains constant and statistically significantly positive at 0.05% for the following three years – we find no evidence of de-adoption of FDA-unapproved uses. Since data used to estimate these parameters only includes time periods when no subsequent FDA certification or de-certification events for pediatric events occurs, this figure isolates the drug adoption in the absence of subsequent FDA information. Although the increase in FDA-unapproved uses is small in absolute magnitude (0.05% increase), 70% of patients diagnosed with diseases in our data are never treated with pharmaceuticals and the average therapeutic drug class

Table 1.2: *Top ten off-label drugs for children and adolescents*

Children (0-11 years of age)		
Drug name	*% on-label*	*Indicated for*
budesonide + formoterol	100%	Asthma
polyethylene glycol	100%	Constipation
zonisamide	100%	Partial seizures
moxifloxacin	100%	Chronic bronchitis; Conjunctivitis; Acute sinusitis; Pneumonia; Skin infections; Abdominal infections; Plague
citalopram	100%	Depression
benzoyl peroxide + clindamycin	100%	Acne
escitalopram	100%	Depression; Anxiety
levalbuterol	48%	Asthma
pimecrolimus	45%	Atopic dermatitis
desloratadine	34%	Idiopathic urticaria; Allergic rhinitis
Adolescents (12-17 years of age)		
Drug name	*% on-label*	*Indicated for*
citalopram	100%	Depression
moxifloxacin	100%	Chronic bronchitis; Conjunctivitis; Acute sinusitis; Pneumonia; Skin infections; Abdominal infections; Plague
polyethylene glycol	100%	Constipation
duloxetine	100%	Depression; Anxiety; Fibromyalgia; Pain
aripiprazole	63%	Autistic disorder; Bipolar disorder; Tourette's; Depression; Schizophrenia
pantoprazole	56%	Erosive esophagitis; GERD; Gastric hypersecretion; Zollinger-Ellison syndrome
oxycodone	51%	Pain
escitalopram	50%	Depression; Anxiety
lisdexamfetamine	50%	ADHD; Binge eating disorder
levetiracetam	23%	Partial and generalized seizures

Note. *Drug claims were measured among pediatric patients (younger than 18 years of age) diagnosed with the FDA-approved diseases at initial market entry in the 1998-2013 MarketScan insurance claims data base. Percent on-label refers to the share of all drug claims that are for FDA-approved uses, conditional on having at least 1000 drug claims per drug. Data on approved indications and ages comes from MicroMedex database and Drugs@FDA. For a definition of FDA-approved vs. unapproved uses, see section Section 1.4.5.*



Note. *This figure plots the rates of FDA approved and unapproved uses of new drugs entering the pharmaceutical drug market during 1998-2013. Drug claims were measured among pediatric patients (younger than 18 years of age) diagnosed with the FDA-approved diseases at initial market entry in the 1998-2013 MarketScan insurance claims data base. Data on approved indications and ages comes from MicroMedex database and Drugs@FDA. For a definition of FDA-approved vs. unapproved uses, see section Section 1.4.5. Models include drug-class-disease fixed effects, calendar time fixed effects, patient age by sex fixed effects, patient treatment experience fixed effects, plan fixed effects at the time of drug claim, and fixed effects for whether the patient had an emergency room or hospitalization claim during the 12 months prior to receiving the drug.*

Figure 1.5: *Drug adoption at initial market entry*

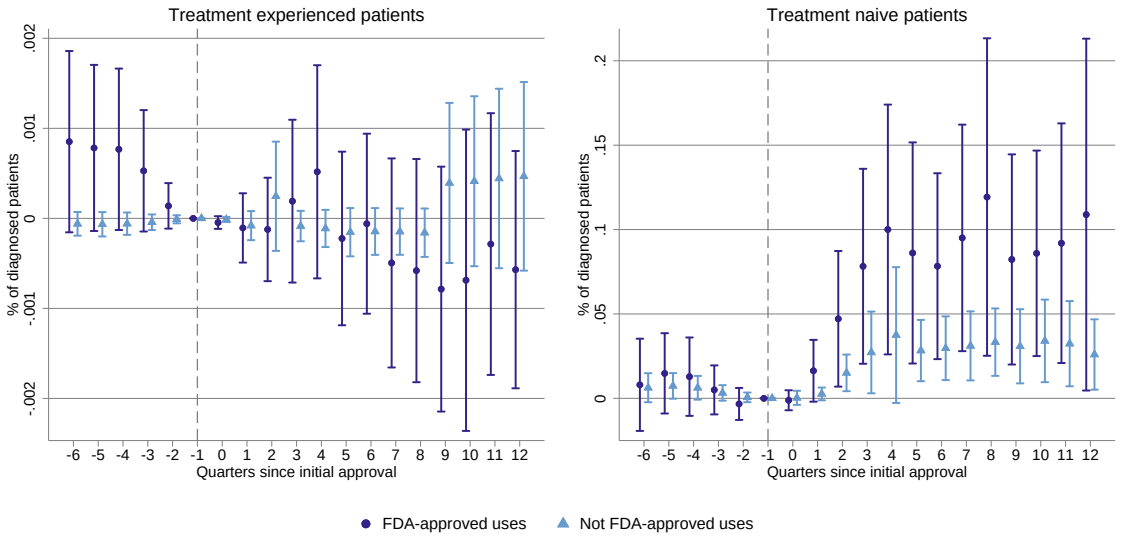
has 40 drugs, indicating that market shares per drug are low and a 0.05% increase is economically meaningful. Figure 1.5 provides suggestive evidence that one side of the tradeoff of regulatory (in)action – untested drugs being prescribed to patients – indeed occurs in practice.

Second, Figure 1.5 shows that an FDA approval for a pediatric age at the time of initial market entry is clearly informative and valuable to patients and physicians. Conditional on patient and plan characteristics, FDA approved uses in pediatric patients are prescribed at twice the rate of unapproved uses. This difference in the rate of FDA-approved vs. FDA-unapproved uses starts from the very moment of market entry and continues for the remainder of the first year since market entry. The on-label use rates also stabilize after the first year since initial approval at approximately 0.1% of all diagnosed patients.

Figure 1.6 shows that neither FDA-approved uses nor FDA-unapproved uses cannibalize market shares of existing drugs in a similar therapeutic class as the entrant – almost all of the prescribing of newly approved drugs, regardless of whether the drug was approved for pediatric age ranges or not, occurs among treatment naive patients who have never been treated with another drug in the same therapeutic class as the entrant. The finding that npatients who have never been treated with other drugs in a similar therapeutic class as the entrant start with an FDA-unapproved use is surprising since the therapeutic classes corresponding to sample drugs in our data on average encompass 40 drugs; thus many alternatives are available. However, it is possible that other competitors of the entrant within the same therapeutic class are also not approved for pediatric ages, so this finding may not necessarily correspond to substitution away from FDA-approved uses.[25]

Additionally, Figure 1.7 shows that conditional on plan and patient characteristics, drug uses without FDA-approval occur both among patients with high and low health care utilization rates. If we take health care utilization rates, specifically the occurrence or prior

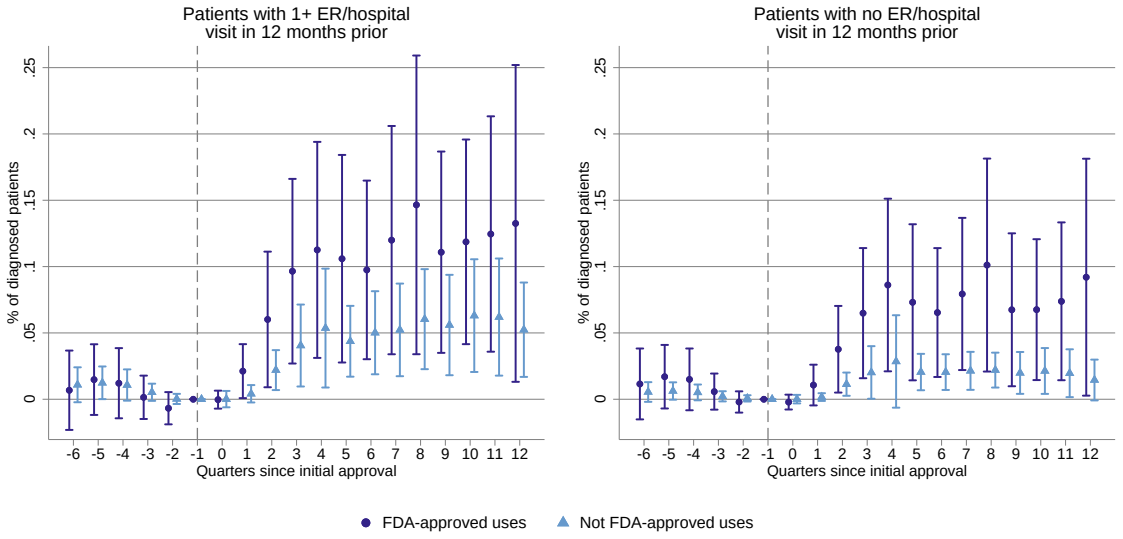
[25]Unfortunately, we do not have data on indications for competitors to our sample drugs and thus cannot determine whether substitution of this sort is occurring.



Note. *This figure plots the rates of FDA approved and unapproved uses of new drugs entering the pharmaceutical drug market during 1998-2013 by patient treatment status. Treatment experienced patients were defined as those with at least one drug claim for a drug in the same therapeutic drug class as the newly approved drug prior to receiving the newly approved drug. Drug claims were measured among pediatric patients (younger than 18 years of age) diagnosed with the FDA-approved diseases at initial market entry in the 1998-2013 MarketScan insurance claims data base. Data on approved indications and ages comes from MicroMedex database and Drugs@FDA. For a definition of FDA-approved vs. unapproved uses, see section Section 1.4.5. Models include drug-class-disease fixed effects, calendar time fixed effects, patient age by sex fixed effects, plan fixed effects at the time of drug claim, and fixed effects for whether the patient had an emergency room or hospitalization claim during the 12 months prior to receiving the drug.*

Figure 1.6: *Drug adoption at initial market entry by patient treatment status*

emergency room (ER) visits and hospitalizations as a proxy for disease severity, Figure 1.7 shows that even though the overall rate of prescriptions for newly approved drugs is twice as high among the more severely affected patients who have at least one claim for an ER or hospital visit prior to receiving the drug, the rate of FDA-unapproved uses is statistically significantly non-zero for the less severe patients as well, indicating that FDA-unapproved uses may not necessarily be targeted solely towards more severe patients.



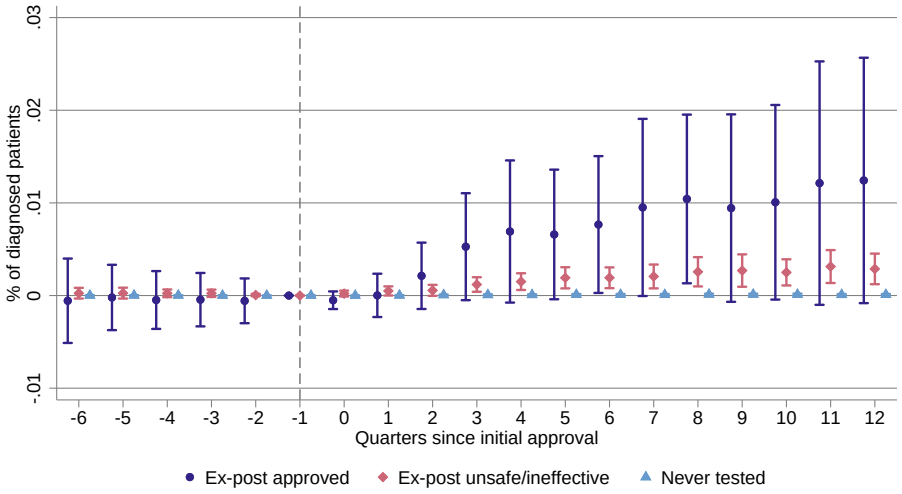
Note. *This figure plots the rates of FDA approved and unapproved uses of new drugs entering the pharmaceutical drug market during 1998-2013 by emergency room (ER) and hospital utilization of the patient in the 12 months prior to the drug claim. Drug claims were measured among pediatric patients (younger than 18 years of age) diagnosed with the FDA-approved diseases at initial market entry in the 1998-2013 MarketScan insurance claims data base. Data on approved indications and ages comes from MicroMedex database and Drugs@FDA. For a definition of FDA-approved vs. unapproved uses, see section Section 1.4.5. Models include drug-class-disease fixed effects, calendar time fixed effects, patient age by sex fixed effects, patient treatment experience fixed effects, and plan fixed effects at the time of drug claim.*

Figure 1.7: *Drug adoption at initial market entry by patient health care utilization*

Lastly, we find small differences in adoption of off-label uses between more and less restrictive plans, as shown in Figure A.1, although on-label use rates are higher among patients in less restrictive plans.

1.6.2 Drug adoption at initial market entry as a function of ex-post outcomes

To examine what types of off-label drugs are adopted by physicians, Figure 1.8 plots the rates of drug adoption, similar to Figure 1.5, broken down by whether the drug was ex-post found to be effective, ex-post ineffective or unsafe (combined into a single category to improve precision of our estimates), or never tested. Since this figure includes drug adoption at initial market entry conditional on *not* being approved at initial market entry, all drug adoption rates plotted in this figure refer to off-label rates. As previously, time periods where subsequent FDA certification or decertification occurs are excluded from estimation and estimates encompass drug adoption in the absence of subsequent FDA labeling changes.



Note. *This figure plots the off-label rates of new drugs entering the pharmaceutical drug market during 1998-2013 by ex-post FDA certification or decertification events. Drug claims were measured among pediatric patients (younger than 18 years of age) diagnosed with the FDA-approved diseases at initial market entry in the 1998-2013 MarketScan insurance claims data base. Data on indications and ages associated with certification and decertification events comes from MicroMedex database, Drugs@FDA, and FDA's Pediatric Labeling Changes database. Models include drug-class-disease fixed effects, calendar time fixed effects, patient age by sex fixed effects, patient treatment experience fixed effects, plan fixed effects at the time of drug claim, and fixed effects for whether the patient had an emergency room or hospitalization claim during the 12 months prior to receiving the drug.*

Figure 1.8: *Drug adoption at initial market entry by ex-post outcomes*

First, Figure 1.8 shows that physicians and patients deciding to prescribe off-label drugs are

largely adopting drugs that are later shown to be effective. Such drugs are being prescribed at an increasing rate continuing into three years after the initial drug approval and prior to any subsequent FDA decisions, reaching 0.01% of all diagnosed patients by the end of the third year since market entry. The adoption of ex-post effective drugs does not appear to slow down with time; however, our estimates for these types of drugs are imprecisely estimated.

Additionally, Figure 1.8 shows that drugs that are ex-post shown to be unsafe or ineffective are gradually adopted starting during the first year since market entry. Although the rate is much lower than ex-post effective drugs, it is precisely estimated and statistically significantly non-zero by the end of the first year since market entry. Figure 1.8 also shows that drugs that are never tested are not adopted, likely because such drugs are used in diseases mostly affecting adults (and are waived from PREA requirements).

Appendix Figure A.2 and Figure A.3 show that drug claims for both ex-post effective and ex-post ineffective/unsafe drugs are targeted primarily towards treatment naive patients and there appears to be no difference in the adoption of ex-post inefective drugs across plan types. Interestingly, Appendix Figure A.4 shows that while rates of off-label use of ex-post effective and safe drugs is similar between more severe and less severe patients, ex-post unsafe/ineffective drugs are primariy prescribed to more severe patients. This may suggest that physicians are willing to try treatments for more severe patients that may not work for the average clinical trial patient.

These findings highlight two sides of the lack of regulatory action in this setting. First, physicians appear to accurately adopt ex-post effective drugs, indicating that the existence of off-label use even in the absence of subsequent FDA action is valuable to patients as it allows for earlier access to effective drugs. On the other hand, a positive share of patients receive a drug that is ex-post shown to be ineffective or unsafe, indicating that the absence of regulatory action allows for adoption of harmful drugs.

1.6.3 Event study at subsequent FDA (de)certification

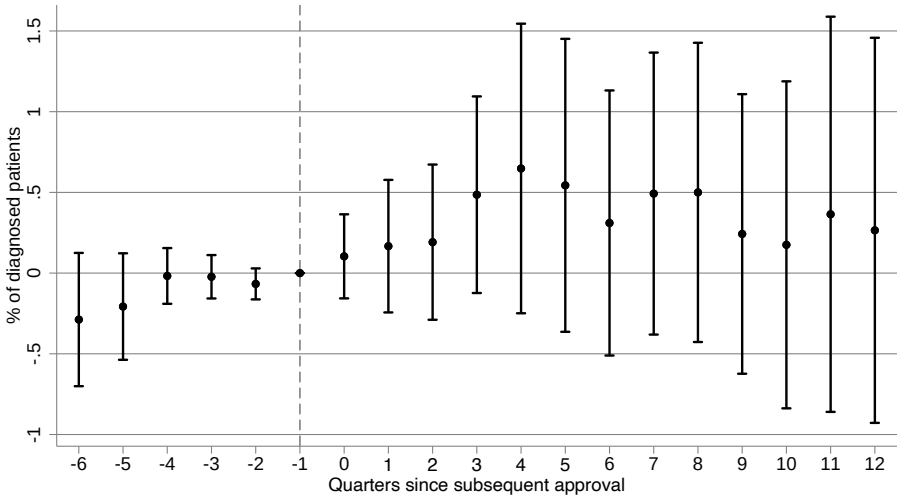
Given that even prior to subsequent trials we observe physicians and patients taking off-label drugs, we investigate whether subsequent FDA certification or decertification of drugs has an impact on demand for drugs or whether physicians who would have adopted the drug under FDA certification already adopted it as part of their off-label prescribing practice. Figure 1.9 shows the event study results at *FDA certification* for subsequent pediatric ages beyond ages approved at market entry, controlling for time the drug has been on the market as well as patient characteristics. This figure suggests that subsequent approvals increase the rate of new prescriptions slightly, although this increase is imprecisely estimated.[26] In fact, Figure 1.10 suggests that the drug already had a large off-label market share in the newly approved disease and age range and most drug claims simply get reclassified as on-label upon FDA approval, indicating that the same patients continue taking the drug even after FDA approval.

Figure 1.11 shows our event study estimates around *FDA decertification* events, i.e., addition of negative trial results to the drug label. This figure shows that the addition of a negative trial result for a specific disease and age range has no impact on the demand for the decertified drug in the disease and ages which had a negative result. There does appear to be some de-adoption of the drug starting in the third year after the negative result, though this is also imprecisely estimated.

1.7 Discussion and conclusion

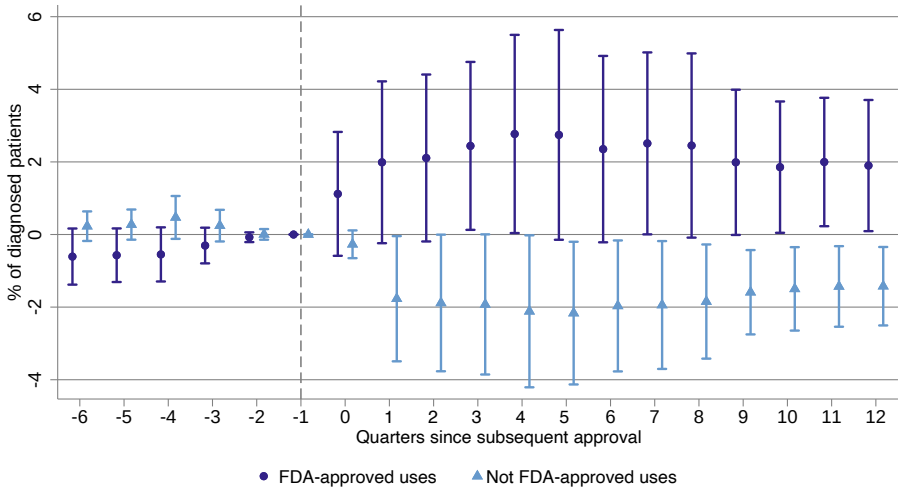
Our findings suggest that while physicians adopt drugs in pediatric patients without corresponding FDA labeling information, the majority of these prescriptions are for drugs that are ex-post shown to be effective. However, in equilibrium, physicians continue prescribing ex-post unsafe/ineffective drugs. FDA labeling changes adding such negative results

[26]We do not observe any patient groups that see a precisely estimated increase, as shown in Appendix Figure A.5-Figure A.7.



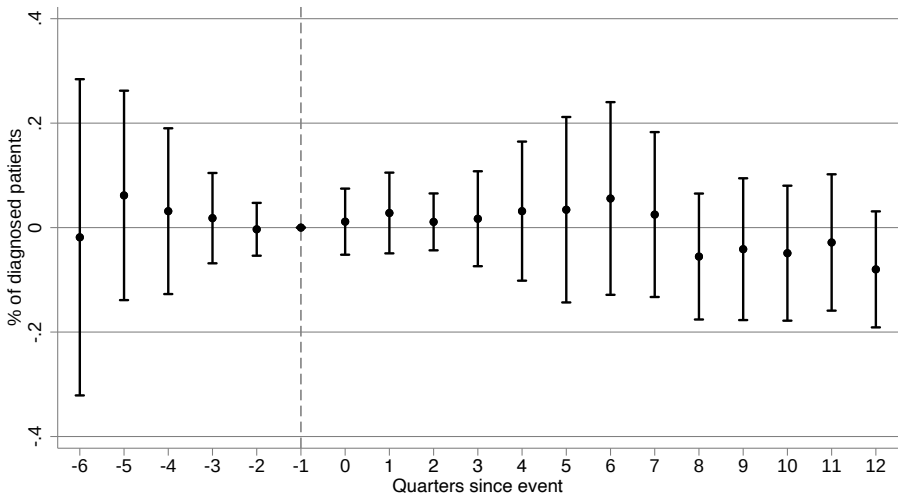
Note. *This figure plots the prescribing rates for drugs approved between 1996-2013 around FDA certification (approval) for some pediatric age. Drug claims were measured among pediatric patients (younger than 18 years of age) diagnosed with the FDA-approved diseases at initial market entry in the 1998-2013 MarketScan insurance claims data base. Data on approved indications and ages comes from MicroMedex database and Drugs@FDA. For a definition of FDA-approved vs. unapproved uses, see section Section 1.4.5. Models include drug-class-disease fixed effects, calendar time fixed effects, patient age by sex fixed effects, patient treatment experience fixed effects, plan fixed effects at the time of drug claim, and fixed effects for whether the patient had an emergency room or hospitalization claim during the 12 months prior to receiving the drug.*

Figure 1.9: *Drug demand at subsequent FDA certification*



Note. *This figure plots the prescribing rates for drugs approved between 1996-2013 around FDA certification (approval) for some pediatric age. Drug claims were measured among pediatric patients (younger than 18 years of age) diagnosed with the FDA-approved diseases at initial market entry in the 1998-2013 MarketScan insurance claims data base. Data on approved indications and ages comes from MicroMedex database and Drugs@FDA. For a definition of FDA-approved vs. unapproved uses, see section Section 1.4.5. Models include drug-class-disease fixed effects, calendar time fixed effects, patient age by sex fixed effects, patient treatment experience fixed effects, plan fixed effects at the time of drug claim, and fixed effects for whether the patient had an emergency room or hospitalization claim during the 12 months prior to receiving the drug.*

Figure 1.10: *Drug demand at subsequent FDA certification by approved vs. unapproved uses*



Note. *This figure plots the prescribing rates for drugs approved between 1996-2013 around FDA decertification (addition of a negative trial result to the drug label) for some pediatric age. Drug claims were measured among pediatric patients (younger than 18 years of age) diagnosed with the FDA-approved diseases at initial market entry in the 1998-2013 MarketScan insurance claims data base. Data on approved indications and ages comes from MicroMedex database and Drugs@FDA. For a definition of FDA-approved vs. unapproved uses, see section Section 1.4.5. Models include drug-class-disease fixed effects, calendar time fixed effects, patient age by sex fixed effects, patient treatment experience fixed effects, plan fixed effects at the time of drug claim, and fixed effects for whether the patient had an emergency room or hospitalization claim during the 12 months prior to receiving the drug.*

Figure 1.11: *Drug demand at subsequent FDA decertification*

to the drug label have little impact on decreasing such potentially harmful and costly uses; positive labeling changes slightly increase prescribing.

We find, however, that, conditional on drug treatment, more severe patients as measured by prior ER and hospital use have a larger share of drug claims for ex-post ineffective or unsafe drugs. Clinical trial results typically reflect treatment effects for the average enrolled patient. Off-label uses of may be effective for some patients and thus non-zero off-label uses in equilibrium may not necessarily be welfare reducing. Welfare calculations weighing the benefits and the costs of pediatric labeling changes are further complicated by the wide array of conditions included in the analysis for which there is no consistent endpoint used to measure outcomes. Extensions of this work will focus on (a) estimating the direct impact of off-label use on patient utilization and outcomes identifiable in insurance claims data and (b) combining data on clinical trial costs and estimated benefits to quantify returns to pediatric labeling with and without extensive delays in such labeling changes.

Chapter 2

Human Capital Spillovers and Health: Does Living Around College Graduates Lengthen Life?[1]

2.1 Introduction

Geographic disparities in health across the U.S. are large and growing (Murray *et al.*, 2005, 2006; Krieger *et al.*, 2008; Ezzati *et al.*, 2008; Kulkarni *et al.*, 2011; Chetty *et al.*, 2016; Dwyer-Lindgren *et al.*, 2017; Finkelstein *et al.*, 2021). The life expectancy gap between counties in the 1st vs. 99th percentile increased from 8.3 years in 1980 to 10.7 years in 2014 (Dwyer-Lindgren *et al.*, 2017) and a recent literature utilizing experimental and quasi-experimental methods has established that place of residence causally impacts both physical and mental health (Katz *et al.*, 2001; Kling *et al.*, 2007; Doyle, 2011; Ludwig *et al.*, 2011, 2012, 2013; Finkelstein *et al.*, 2021). While these studies document that place matters for health, there is little consensus on whether this relationship reflects environmental exposure, economic

[1]Co-authored with Jacob Bor, David Cutler, and Edward Glaeser.

conditions, health systems, local behavioral norms, or the role of public policies.

A robust literature finds a strong correlation between area-level education and earnings, holding individual-level education constant, typically referred to as human capital spillovers or externalities (Rauch, 1993; Moretti, 2004a,b; Canton, 2007; Rosenthal and Strange, 2008; Iranzo and Peri, 2009). Furthermore, the share of the population with a college degree strongly predicts the life expectancy of an area. In the data from (Ezzati *et al.*, 2008), county life expectancy rises by 1.3 years as the share of adults with a college degree increases by 10 percentage points. Much of this county-level correlation between education and health reflects the well-known individual-level relationship between years of schooling and mortality (Elo and Preston, 1996; Cutler and Lleras-Muney, 2006; Grossman, 2006, 2008; Meara *et al.*, 2008; Cutler *et al.*, 2011; Cutler and Lleras-Muney, 2012; Grossman, 2015).

In this paper, we ask whether human capital spillovers in health can help explain the relationship between area education and mortality. As area-level variation in education levels has widened over the past four decades (Berry and Glaeser, 2005; Moretti, 2013; Diamond, 2016), the existence of human capital spillovers in heath would help explain widening geographic health disparities. Documenting such spillovers and examining mechanisms driving these externalities highlights pathways through which local labor and educational policies may influence population health even in the absence of direct effects, which should be incorporated in any welfare analysis of such policies and in optimal local/place-based policy design.

We combine U.S. Census and American Community Survey data for 1990, 2000, and 2010 with complete mortality records containing cause of death information and individual education from the Multiple Cause Mortality Files; the U.S. Standard Certificate of Death only included information on the decedent's education after 1989. We find that after adjusting for individual-level educational attainment, a 10 percentage point increase in the percent of college graduates in an area is associated with a 5.6% lower all-cause mortality rate. Moreover, the correlation between area human capital and mortality has strengthened over

time.[2] This correlation is present across all demographic groups but is strongest for individuals younger than 65. Most of the human capital spillovers on health accrue to white and Hispanic individuals, but not African Americans. We also document that the human capital spillovers on all-cause mortality are largest for the least educated areas in 1990.

In addition to its impact on mortality, area human capital correlates strongly with non-fatal health outcomes and quality of life. After adjusting for individual-level education, a 10 percentage point increase in the percent college graduates in an area is associated with an 9% reduction in lung disease, 6% reduction in heart disease, and a 4% reduction in the number of days in poor physical or mental health. When examining human capital spillovers separately by cause of death, we find that are area human capital spillovers exist across almost all causes of death but are increasing over time only for medically-amenable causes of death (which includes deaths due to respiratory conditions and heart disease), as well as smoking-related and obesity-related causes of death. Consistent with our findings that the least educated areas drive the strengthening relationship between area human capital and health, we find that the correlation between these causes of death and area human capital is increasing among the least educated areas but is declining in other areas.

After presenting these facts, we examine three hypotheses that might explain the area human capital externality on health: i) spatial sorting, where innately healthier individuals move to high human capital areas, ii) higher human capital areas have higher quantity or quality amenities that improve health, and iii) individuals in better educated areas have fewer health-harming behaviors. Using data from the Health and Retirement Study for individuals 51 years of age and older and the National Longitudinal Survey of Young Women and Men for younger individuals, we reject the spatial sorting hypothesis. In contrast to the theory, we find that less healthy individuals, as measured by predicted mortality, are more likely to move counties. Furthermore, there are no differences in the area human capital between the areas to which healthier and sicker individuals move. We also

[2]Human capital earnings externalities also appear to be increasing over time (Glaeser *et al.*, 2004).

show that greater demand and quality for health-related amenities such as pollution, crime, health care demand, and health care quality cannot explain the human capital externality on health, rejecting our second hypothesis. We also reject wage effects operating through human capital externalities on income.

We find that differences across areas in health-related behaviors such as smoking and obesity explains almost all of the correlation between area human capital and mortality, after controlling for individual education. This result is presaged by the strong and robust correlation between area human capital and smoking, obesity, and physical activity we document. Using data from the Behavioral Risk Factor Surveillance System and the Current Population Survey, after controlling for individual-level education, we show that a 10 percentage points increase in area human capital is associated with a 10% decrease in the smoking rate, a 5% increase in smoking quit rate, a 9% decrease in the population share without any physical activity, and a 15%, 7%, and 2% decrease in the probability of being very obese, obese, and overweight, respectively. Consistent with our findings for mortality, we also find that the human capital spillovers on health-related behavior are highest for the least educated, rural areas in 1990.

Given the prominent role of health-related behaviors in explaining the correlation between area human capital and mortality, we examine two potential channels driving human capital spillovers on smoking: differential costs of health-related behaviors due to higher levels of education, and peer effects. To examine these hypotheses, we focus on smoking and tobacco taxes, clean indoor air laws, and workplace smoking bans as measures capturing differences in the cost of smoking across areas. Using the Current Population Survey, we find that individuals living in areas with a 10 percentage point higher share of college graduates are 3% more likely to be employed at workplaces with a smoking ban in all work and public areas, even after controlling for individual-level education. This effect persists even after controlling for state-year fixed effects, which pick up time-varying state policies such as tobacco taxes and clean indoor air laws. Directly controlling for smoking regulations

in regressions of smoking on area human capital and individual education can explain up about one-quarter of the correlation between area human capital and smoking rates.

Differences in behavior across areas with different levels of human capital might also be driven by peer effects, either directly or through informational spillovers. Using data from the National Health Interview Survey from 1987, 1992, and 2000, we find that, controlling for individual-level education, a 10 percentage point increase in the percent of college graduates is associated with a 11% increase in the share of individuals agreeing or strongly agreeing with the statement that smoking is harmful for pregnant women's babies and a 15% increase in the share agreeing or strongly agreeing that most lung cancer deaths stem from smoking. We also find that even after controlling for individual education, a 10 percentage point increase in area human capital is associated with a 8% increase in the probability of agreeing/strongly agreeing with smoking bans in bars, restaurants, and work areas, which we use as a proxy measure for beliefs about the harms of second-hand smoke. This relationship is strengthening over time and is noisy but highest in the least educated areas. Adding beliefs about second-hand smoke and smoking bans in regressions of smoking on area human capital, individual education, and smoking regulations can further explain 23% of the human capital spillovers on smoking.

Taken together, our results show that the large and growing relationship between education and health can be explained by the human capital externality on behaviors such as smoking and obesity, which are partially driven by more educated areas implementing stricter tobacco control policies and partly explained by informational spillovers about the harms of smoking. in contrast, wage spillovers, spatial sorting, and differences in health-related amenities are not as important.

The paper is structured as follows. Section 3.2 discusses our data sources for mortality, non-fatal health outcomes, smoking, obesity, migration, and area characteristics. Section 2.3 establishes the baseline relationship between area human capital and mortality and examines variation in human capital spillovers by cause of death, demographic groups, and

observable area characteristics. In this section we also discuss the correlation between area human capital and disease prevalence and non-fatal health. Section 2.4 presents a model of health-related behaviors and location choice that highlights the mechanisms behind human capital spillovers on health that we test empirically. In Section 2.5 empirically test the wage effects, spatial sorting, and health-related amenities hypotheses. Section 2.6 focuses on health-related behaviors and examines how human capital correlates with the cost of health-related behaviors across areas, as well as peer effects. Section 2.7 concludes.

2.2 Mortality and Area Characteristics Data

In this section, we discuss our mortality and area level data.

2.2.1 Mortality

Our central source on mortality data is the microdata on all deaths to U.S. residents occurring in the years 1990, 2000, and 2010, obtained from the National Center for Health Statistics Multiple Cause Mortality Files (MCMF). MCMF data are compiled from death certificates and include the underlying cause of death, as well as the age, sex, and educational attainment of the deceased (since 1988). Educational attainment on death certificates is typically reported by next-of-kin.[3] We also observe the deceased's county of residence in the data.[4]

We aggregated total deaths by county-age-sex-race-education cells. We excluded 3% of deaths that occurred among individuals younger than 25, as education is not reliably completed before age 25. Cells were defined by 5-year age categories (25-29, 30-34, ..., 85+), five levels of educational attainment based on completed years of school (<12, 12, 13-15,

[3]Some concerns have been raised regarding the accuracy of such reporting relative to self-reports, particularly the overstatement of high school graduation rates (Shai and Rosenwaike, 1989; Sorlie and Johnson, 1996; Rosamond *et al.*, 1997; Rostron *et al.*, 2010).

[4]County of residence is suppressed for deceased individuals residing in counties with population less than 100,000.

16, 17+), gender (M, F), and race/ethnicity (white non-Hispanic, Black non-Hispanic, other non-Hispanic, Hispanic).[5] We exclude deceased individuals with missing data on age (0.02%), county (0.16%), and education (10.5%) since we cannot match these deaths to a population denominator when calculating mortality rates. Most missing data on education occurred in 1990 for Louisiana, New York, and Georgia. Due to the exclusion of these observations, crude mortality rates in our sample are slightly lower than published estimates. This sample selection yielded 798,850 county-year-age-sex-race-education cells with non-zero deaths out of 22.8 million possible cells.

To mitigate bias from the number of deaths without reported education, we exclude any county-year-age-sex-race cells where the percent of deaths with missing education is 25% or more, which drops 1.9% of adult deaths with non-missing age, race, county, and education. In robustness checks, we only included county-year-age-sex-race cells where the percent of death certificates without reported education is 5% or less. Our regression analysis includes the percent of death certificates without education in each area-age-sex-race cell as a covariate.

To ensure comparability of geographic areas across years we aggregated counties into groups using definitions for consistent public use microdata areas (CONSPUMA), which represent the most detailed geographic areas that can be consistently identified between 1980-2011.[6]

Mortality rates were calculated by merging death counts for area-age-sex-race-education cells with corresponding population counts from the 1990 and 2000 U.S. Decennial Census (5% sample) and the pooled 2009-2011 American Community Survey (ACS) for 2010 (as

[5]Due to differences in data encoding over time in the MCMF data, in 1990 and 2000 we consider individuals with four years of high school as having completed high school, regardless of whether they were awarded a diploma. In 2010, we consider those with 12 years of education and no high school diploma as not having completed high school. Associate degrees were included in the 13-15 (some college) education category.

[6]For counties included in multiple CONSPUMAs, we use the CONSPUMA containing most of the countys population. Only 1.1% of counties in 1990, 1.3% in 2000, and 1.4% in 2010 map to multiple CONSPUMAs.

in Wheeler (2008)).[7] Due to random sampling in the Census and ACS data, 23,572 area-year-age-sex-race-education cells with non-zero deaths could not be matched to population denominators.[8] We exclude these cells (containing 0.7% of deaths) from the data. There are 188 cells with both death and population data but where total deaths exceed the estimated population, presumably due to sampling error. We censor the deaths at 100 percent for these cells (reducing total deaths by 0.05%).

Table 2.1 shows summary statistics in the mortality data. Our final dataset, pooled across 1990, 2000, and 2010, contains 478,000 area-year-age-sex-race-education cells and covers 5,928,470 deaths across all years, which represents 85% percent of deaths for people aged 25 and older.[9] The death rate was 1,162 deaths per 100,000.

Cause of death is also identified on death certificates. We classify deaths as medically amenable, smoking-related, obesity-related, or due to external causes based on causes identified in the literature (see Section B.1 for details). A cause of death can fall into multiple categories; for example, heart disease is both smoking-related and obesity-related. Approximately 56% of deaths are classified as smoking-related, 41% as obesity-related, 41% as medically amenable, and 6% as deaths due to external causes.

2.2.2 Data on Non-Fatal Health Outcomes

We obtain data on the prevalence of conditions such as cancer, lung disease, diabetes, heart disease, and stroke from the Health and Retirement Study (HRS), which provides a biennial, longitudinal survey of people aged 51 and older, over the 1992-2008 period. We also use data on individuals self-reporting good, very good, or excellent general health status and number of days over the last 30 days where poor physical or mental health

[7] We use the 3-year ACS due to the lack of data on individual education in the 2010 Decennial Census.

[8] This sampling issue is generally concentrated in the college-educated population aged 75 and older.

[9] The biggest drop from the 798,850 county-year-age-sex-race-education cells to the 478,000 area-year-age-sex-race-education cells is that counties are combined when aggregating the data to the area level.

Table 2.1: *Descriptive statistics on mortality and area characteristics*

	Mean	SD
Cell characteristics		
Age 25-64	80.40%	—
Age 65+	19.60%	—
Female	52.10%	—
No high school	15.90%	—
High school graduate	36.80%	—
Some college	22.50%	—
College graduate	15.80%	—
Graduate education	9.10%	—
Missing education on death certificate	3.60%	6.68%
Mortality rates by cause (per 100,000)		
All cause	1,162	2,613
Heart disease	339	952
Cancer	280	512
Medically amenable causes	474	1,186
Smoking-related causes	652	1,629
Obesity-related causes	479	1,180
External causes	67	120
Area characteristics		
% college graduates	24.80%	8.90%
% Black	10.50%	10.50%
% Hispanic	10.80%	13.00%
Density (persons per square mile)	1,805	6,040
Population	1,840,895	2,037,348
Industry share: manufacturing	11.30%	5.00%
Number of observations		
Area-year-age-sex-race-education cells	478,000	—
Areas	485	—
Population	510,096,733	—
Deaths	5,928,470	—

Note. *Death data by county-year-age-sex-education comes from the 1990, 2000, and 2010 Multiple Cause Mortality Files. Counties were aggregated to areas representing consistent public use microdata areas (CONSPUMAs). Mortality rates were calculated using population sizes from the 1990 and 2000 Census 5% samples, and the 2009-2011 ACS 5-year file for 2010. We exclude county-year-age-race-sex cells where 25% or more of reported deaths lacked education data. Statistics are weighted by cell size.*

interfered with daily activities from the 1999-2001 and 2009-2011 Behavioral Risk Factor Surveillance System (BRFSS).[10] Each respondent in the HRS and BRFSS data is mapped to an area using the same methodology as with the MCMF data.[11] As with the mortality data, we subset the BRFSS sample to individuals 25 years of age and older. All of these data sources also contain individual education and demographics.

2.2.3 Data on Health-Related Behaviors

We use self-reported data on smoking status, body mass index (BMI), and physical activity from the 1999-2001 and 2009-2011 BRFSS data described above. Since the BRFSS does not contain data from the 1990s, we supplement the BRFSS data with data on individual education, demographics, and smoking behavior from the Tobacco Use Supplement in the Current Population Survey (CPS) from waves 1995-1996, 1998-1999, 2001-2002, 2003, 2006-2007, 2010-2011, and 2014-2015.[12] These data contain geographic information for counties with population 100,000 or greater. As with all prior data sources, we include only individuals 25 years of age or older and match available counties to larger areas (CONSPUMAs).

2.2.4 Area Characteristics

We merge in several area-level attributes to the health data. Area human capital is defined as the percent of area residents aged 25 years or older with at least a college degree, using Census data from 1990 and 2000 and ACS data from 2009-2011. We obtain the area-level percent Black and Hispanic, and industry shares (proportion of workers who work in agriculture, forestry, fisheries, and mining; construction; manufacturing; transportation, communications, and other public utilities; trade; finance, insurance, and real estate; services; public administration; armed forces) from these data sources.

[10]The 1990 BRFSS does not contain data on self-reported health or number of days where poor health interfered with daily activities. It also does not contain consistent geographic identifiers.

[11]We utilize a restricted use HRS file with county identifiers. County identifiers are included in the BRFSS but are suppressed for areas with fewer than 50 respondents.

[12]The CPS data does not ask about height and weight and thus we cannot calculate BMI in this data set.

Data on area population and land area sizes were obtained from the Area Resource Files provided by the Bureau of Health Workforce for 1990, 2000, and 2010. We use this data to compute population density in each area. We also obtained data on non-federal physicians and hospital beds at the county level for 1990, 2000, and 2010 from the Area Resource Files, which we convert to numbers per 1,000 individuals.

County-level data on the average annual percent of Medicare enrollees having at least one annual ambulatory visit to a primary care clinician and average percent of female Medicare enrollees aged 67-69 having at least one mammogram over a two-year period for years 2003-2015 was obtained from the Dartmouth Health Atlas and was aggregated to areas using our previously discussed approach.

County-level reported homicides are taken from the Uniform Crime Reports. For each of 1990, 2000, and 2010, we average reported homicide in the three years centered around the decade (e.g., 1989-91 for 1990) to improve precision. We aggregate these data to areas and express as rates per 100,000 individuals.

Satellite data on air pollution for 1999-2001 and 2009-2011 are from van Donkelaar *et al.* (2019) and capture the concentration of suspended particulate matter of diameter 2.5ţm or less (PM-2.5). For 1989-1991, we obtain data on PM-10 measurements from the Environmental Protection Agency for counties with particulate matter monitoring agencies. We use the methodology from Meng *et al.* (2019) to generate predicted PM-2.5 measurements for 1989-1991 using the PM-10 and PM-2.5 data.

Hospital quality data comes from the Hospital Compare Database provided by Centers for Medicare and Medicaid Services for the period 2003-2008. The database contains information on process-of-care indicators for pneumonia, congestive heart failure, and acute myocardial infarction. These quality measures typically reflect usage of inexpensive, easy-to-implement practices that represent the standard of care for patients presenting with

these conditions.[13] For each hospital, quality scores were first averaged at the condition level, using condition-specific z-scores. We then average these z-scores for the three conditions, which provides a single hospital-specific metric for 3,861 hospitals, which we treat as roughly representing hospital quality for 2010. Finally, we calculate area-level hospital quality scores, weighting the hospital quality of all hospitals in the area by the number of discharges per hospital.

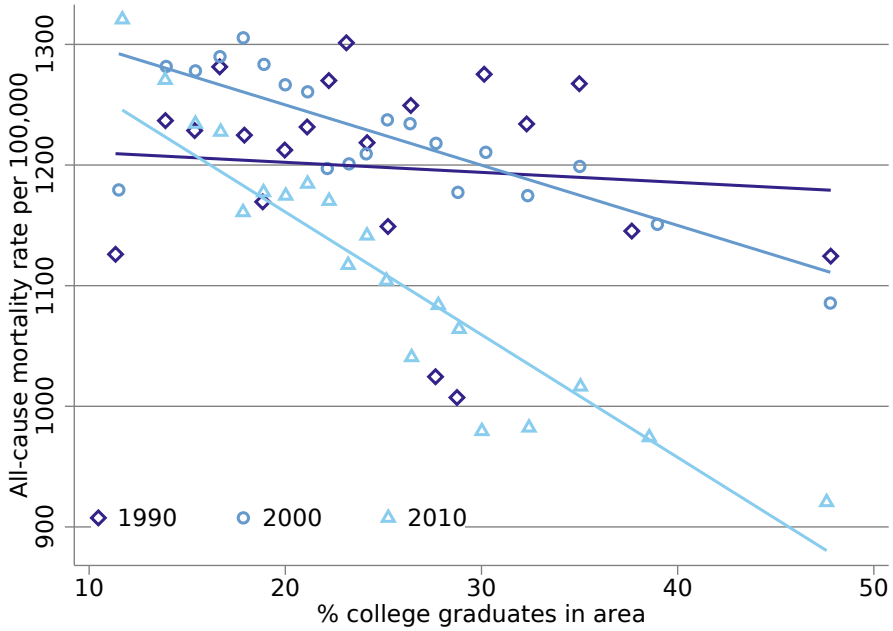
2.3 Area Human Capital and Mortality

We begin with an examination of the empirical relationship between area human capital and mortality. Figure 2.1 shows vigntiles of the relationship between area human capital and mortality across area-age-sex-race-education cells for each of 1990, 2000, and 2010. In forming these vigntiles, we control for age, sex, and race but not individual education. The figure shows no relationship between education and mortality in 1990 but a large increase in the relationship over time. In 2010, each 10 percentage point increase in the area-level share of adults with a college graduates – the equivalent to moving from the bottom quartile to the top quartile of the 2010 distribution of human capital – was associated with a decline of 103 deaths per 100,000 (statistically significant at the 1% level). This is equivalent to a 9% reduction in average mortality.

The major issue with interpreting these coefficients is that area education is clearly correlated with individual education, and individual education is clearly related to health.[14] Table 2.2 shows a variety of analyses separating individual and area level education. Each column of the table reports results of a regression model relating cell-level mortality rates to cell and area characteristics, using data for all area-year-age-sex-race-education cells. We

[13] For example, one measure is the percent of patients presenting with an acute myocardial infarction who are given aspirin upon arrival.

[14] The literature on the relationship between individual education and health is vast. For a comprehensive review of the theoretical background, as well as descriptive and quasi-experimental evidence on the relationship between education and health, see, for instance, Grossman (2006); Cutler and Lleras-Muney (2006); Grossman (2008, 2015); Galama *et al.* (2017).



Note. *This graph is a binned scatter plot across area-age-sex-race-education cells sorted by percent college graduates in the indicated year. Each point includes 5% of the population in that year, plotted at the mean percent college graduates and mean mortality rate (both residualized for age-sex-race fixed effects). The coefficients (and standard errors in parentheses) of the corresponding OLS regressions are -0.71 (3.12) in 1990, -4.78*** (0.66) in 2000, and -10.3*** (0.65) in 2010. *** p<0.01, ** p<0.05, * p<0.1. Standard errors are clustered at the area level.*

Figure 2.1: *The relationship between area human capital and age-sex-race-adjusted mortality per 100,000*

limit controls to demographic and geographic characteristics that are unlikely to be part of the causal pathway between area human capital and health: 5-year age-sex-race/ethnicity interactions, as well as year. We also control for the percent of death certificates in the cell with missing education data, population and population density (both log-transformed), and employment shares by industry at the area level.

Column 1 of the table examines the effect of individual education alone. Controlling for other cell-level and area-level covariates, the correlation between individual education and mortality is enormous. Individuals without a high school degree experience 699 additional deaths per 100,000 relative to individuals with graduate education. Mortality risk declines with each additional level of educational attainment; there is no evidence of a threshold effect. The second column shows the relationship between mortality and area human capital, without individual education controls. These results are closely related to Figure 2.1 and show that a 10 percentage point increase in the area-level percent of adult population with a college degree is associated with 102 fewer deaths per 100,000.

The third column presents the primary motivating fact for the paper. Even controlling for individual education, a 10 percentage point increase in the share of college graduates in an area is statistically significantly associated with fewer deaths, roughly a 6% decrease relative to average mortality.[15] The difference in the coefficients on the share of college graduates between the second and third columns of Table 2.2 implies that controlling for individual education explains just 36% of the relationship between area human capital and mortality shown in Figure 2.1.

Column 4 of Table 2.2 allows for the relationship between area human capital and mortality to vary by year. As with Figure 2.1, the relationship between area human capital and

[15]We obtain similar results when including a full set of individual-level age-sex-race-education interactions a 10 percentage point increase in the percent college graduates in an area is associated with a decrease of 66.4 deaths per 100,000. Even if we control for the changing relationship between individual education and mortality over time by including fixed effects for year interacted with individual education, we find that a 10 percentage point increase in the percent college graduates in an area is associated with a decrease of 63.2 deaths per 100,000.

Table 2.2: *Regression results of all-cause mortality rates per 100,000 on area human capital*

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Area characteristics							
% college grads		-10.24***	-6.56***	-1.45	-2.59***	1.64*	-23.73***
		(1.48)	(1.43)	(2.18)	(0.54)	(0.85)	(2.89)
% college grads * year=2000				-3.46		-3.70***	
				(2.14)		(0.74)	
% college grads * year=2010				-7.04***		-6.22***	
				(2.34)		(0.88)	
Cell characteristics							
High school graduate	-243.35***		-243.77***	-244.89***	-240.93***	-241.99***	-240.36***
	(38.28)		(38.35)	(38.45)	(38.53)	(38.60)	(38.52)
Some college	-422.62***		-421.66***	-423.52***	-421.08***	-422.50***	-420.16***
	(27.50)		(27.50)	(27.69)	(27.57)	(27.66)	(27.55)
College graduate	-519.02***		-514.63***	-515.93***	-513.80***	-514.73***	-512.14***
	(33.83)		(33.69)	(33.82)	(33.77)	(33.83)	(33.75)
Post-graduate educ	-698.68***		-693.08***	-694.05***	-690.61***	-691.12***	-689.99***
	(35.22)		(34.95)	(35.06)	(35.11)	(35.16)	(35.08)
% deaths with missing educ	-12.68***	-12.05***	-12.59***	-12.41***	-7.15***	-6.99***	-10.16***
	(1.59)	(1.64)	(1.62)	(1.69)	(0.71)	(0.70)	(1.19)
State-year FE	No	No	No	No	Yes	Yes	No
Area FE	No	No	No	No	No	No	Yes
Weighted obs.	510,096,733	510,096,733	510,096,733	510,096,733	510,096,733	510,096,733	510,096,733
Cells	478,000	478,000	478,000	478,000	478,000	478,000	478,000
Areas	485	485	485	485	485	485	485
R-squared	0.855	0.850	0.855	0.855	0.856	0.856	0.856
Dependent var. mean	1,162	1,162	1,162	1,162	1,162	1,162	1,162
% change from 10pp increase in % college grads		-8.8	-5.6		-2.2		-20.4

Note.*** p<0.01, ** p<0.05, * p<0.1. Standard errors are clustered at the area level. OLS regressions are estimated at the area-year-age-sex-race-education cell level, weighted by cell population, and pooled across 1990, 2000, and 2010. All regressions control for cell-level 5-year age (25-29, 30-34, . . . , 85+) by sex by race (white non-Hispanic, Black non-Hispanic, other non-Hispanic, Hispanic) interactions, year.*

mortality increases over time. Previous studies have found widening mortality disparities across individual-level education over time (Meara *et al.*, 2008; Cutler *et al.*, 2011; Olshansky *et al.*, 2012; Masters *et al.*, 2012; Hayward *et al.*, 2015; Sasson, 2016; Bor *et al.*, 2017). Our paper demonstrates that there exists a similarly increasing impact of area human capital on mortality over time.

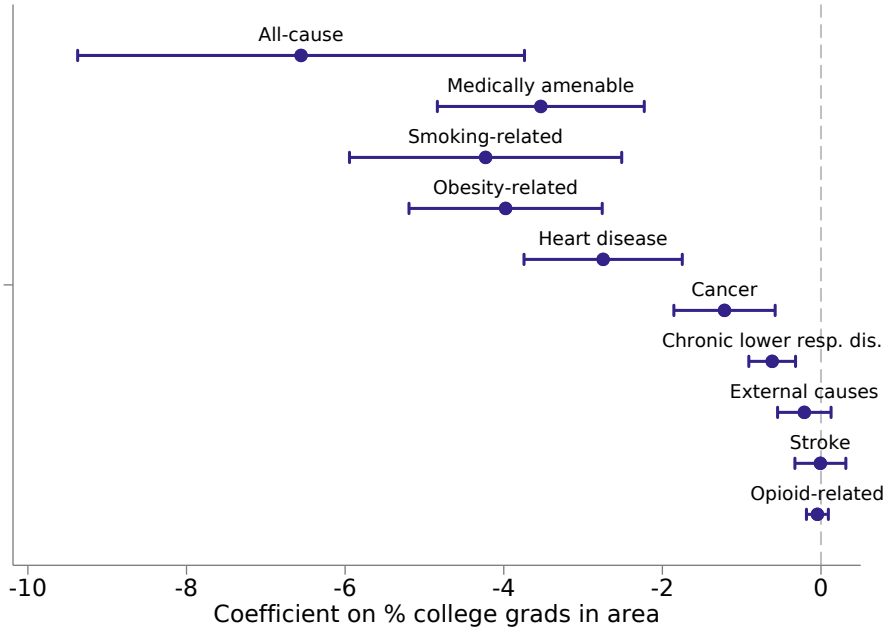
Columns 5 and 6 match the specifications in columns 3 and 4 but include state by year fixed effects, which account for time-varying state-level characteristics that may be correlated with both area human capital and health (e.g., changing state-level health or education policies such as Medicaid coverage, tobacco taxes, smoking regulations, etc.). The impact of area human capital on mortality falls in these specifications but remains statistically significant and increasing over time. Thus, differences in state-level policies cannot be the sole factor driving the correlation between area human capital and health nor the increase in this effect over time.

Finally, column 7 includes area fixed effects, which control for time-invariant area-level characteristics that may be correlated with area human capital and health. Within areas, there is an even larger correlation between the growth of the share with a college degree and the reduction in mortality.

2.3.1 Heterogeneity in the Relationship Between Education and Mortality

To help motivate the theories we explore, we examine how the relationship between area human capital and health varies across causes of death and demographic groups. Figure 2.2 presents estimates from our baseline regression, with mortality rates separated by cause of death. Area human capital is statistically significantly negatively correlated with mortality rates for medically amenable, smoking-related and obesity-related deaths, as well as deaths from heart disease, cancer, and chronic lower respiratory disease. In contrast, area human capital is not correlated with death due to stroke, opioid-related deaths, and deaths due to external causes. Figure B.2 shows that the correlation between area human capital

and mortality strengthens over time for medically amenable deaths, cancer-related, chronic respiratory disease, as well as deaths due to opioid-related or external causes to a minor degree. These results suggest that behavioral differences are a key reason for the externalities of human capital on health we document in this paper.

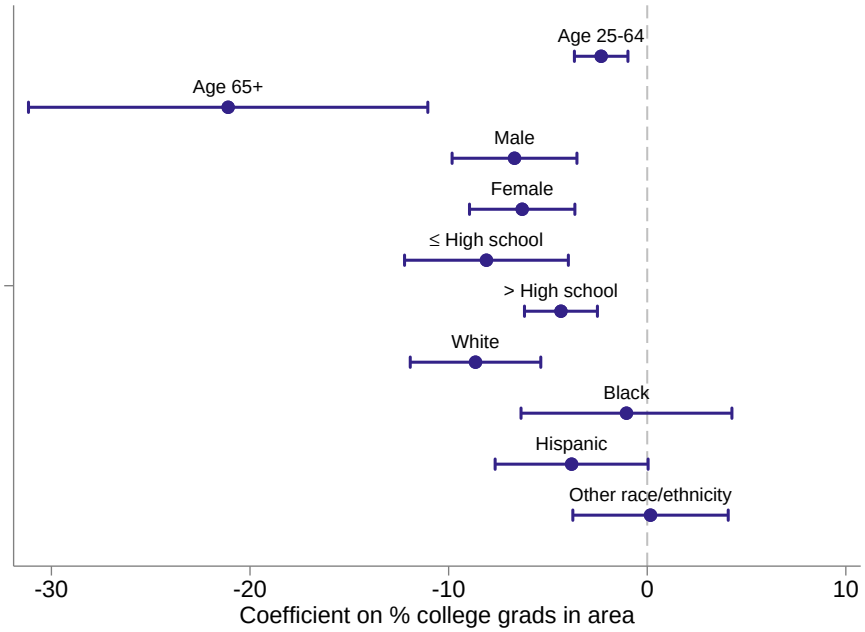


Note. *This figure plots the coefficient on area human capital estimated separately for each cause of death. OLS regressions are estimated at the area-year-age-sex-race-education cell level, weighted by cell population, and pooled across 1990, 2000, and 2010. All regressions control for cell-level 5-year age (25-29, 30-34, , 85+) by sex by race (white non-Hispanic, Black non-Hispanic, other non-Hispanic, Hispanic) interactions, individual education, percent of death certificates without education information, and year. We also include controls for area log density and log population, percent Black, percent Hispanic, and industry shares. Smoking-related, medically amenable, and obesity-related causes of death include all deaths to causes associated with that risk factor and are not mutually exclusive categories (see Section B.1 for detail). Confidence intervals are clustered at the area level.*

Figure 2.2: *Regression results of cause-specific mortality rates per 100,000 on area human capital*

Figure 2.3 shows estimates of area human capital using the same regression as in column 3 of Table 2.2 estimated separately by age, gender, individual education, and race. In absolute terms, the relationship between area human capital and mortality is larger for older than younger individuals. However, in relative terms, the relationship between area human capital and health is slightly stronger for younger individuals. A 10 percentage point

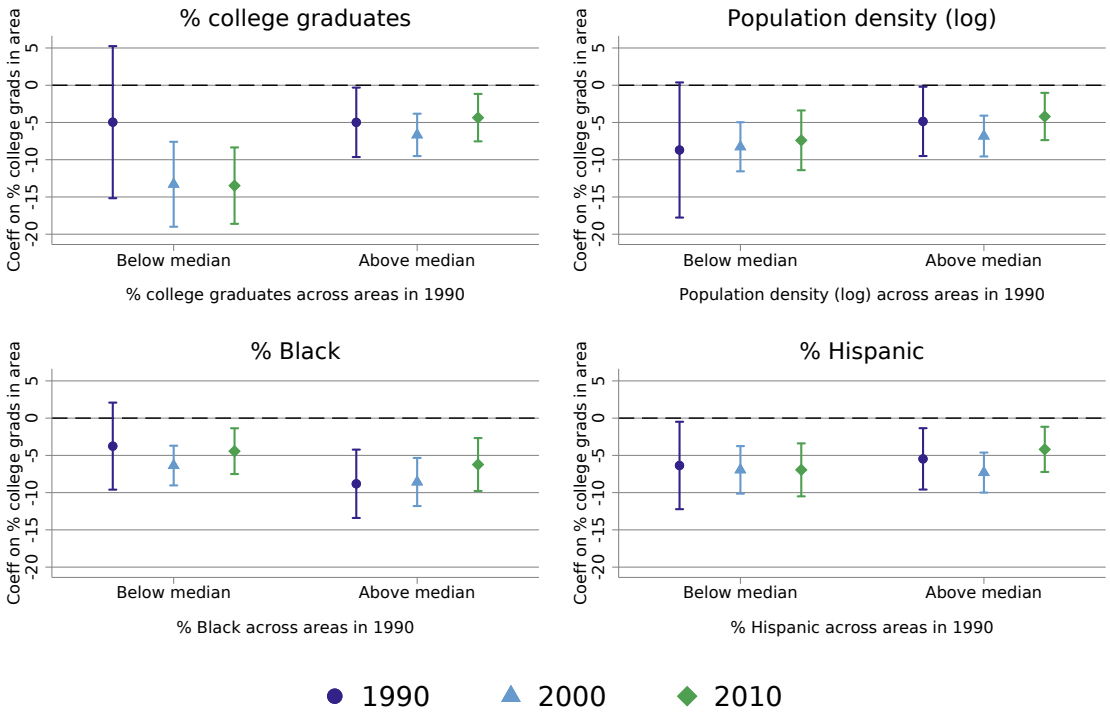
increase in the area-level percent college graduates is associated with a 6.6% decrease in the mortality rate among individuals younger than 65 and a 4.7% decrease in the mortality rate among those 65 and older. The relationship between area human capital and mortality is similar by gender and individual education, in both absolute terms and relative to the average mortality rate in each subgroup. The coefficient estimates are bigger for white and Hispanic individuals, however the standard errors are large for Black individuals and people of other races, so equality of the coefficients cannot be rejected. Figure B.1 shows that the relationship between area human capital and mortality increased over time for almost all demographic groups. Overall, there are very few differences in area effects by demographics.



Note. *This figure plots the coefficient on area human capital estimated separately for each subgroup. OLS regressions are estimated at the area-year-age-sex-race-education cell level, weighted by cell population, and pooled across 1990, 2000, and 2010. All regressions control for cell-level 5-year age (25-29, 30-34, , 85+) by sex by race (white non-Hispanic, Black non-Hispanic, other non-Hispanic, Hispanic) interactions, individual education, percent of death certificates without education information, and year. We also include controls for area log density and log population, percent Black, percent Hispanic, and industry shares. Confidence intervals are clustered at the area level.*

Figure 2.3: *Regression results of all-cause mortality rates per 100,000 on area human capital by demographic subgroups*

We similarly examined how the impact of area education varies with area characteristics. Figure 2.4 shows the coefficient on the interaction of area human capital with being above or below median on four area characteristics: area human capital, percent of the area population that is Black, percent of the population that is Hispanic, and population density. We use the specification where the coefficients differ by year (column 4 of Table 2.2) to examine both levels and changes in the relationship.



Note. *This figure plots the coefficient on area human capital interacted by whether the area-year-age-sex-race-education cell is in an area above/below median percent college graduates, population density (log), percent Black, or percent Hispanic across areas in 1990, weighted by population. OLS regressions are estimated at the area-year-age-sex-race-education cell level, weighted by cell population, and pooled across 1990, 2000, and 2010. All regressions control for cell-level 5-year age (25-29, 30-34, , 85+) by sex by race (white non-Hispanic, Black non-Hispanic, other non-Hispanic, Hispanic) interactions, individual education, percent of death certificates without education information, and year. We also include controls for area log density and log population, percent Black, percent Hispanic, and industry shares. Confidence intervals are clustered at the area level.*

Figure 2.4: *Regression results of all-cause mortality rates per 100,000 on area human capital by area characteristics*

Parallel to our findings for individual education, there is a negative correlation between

area human capital and mortality across all area characteristics. Thus, there is no threshold based on observable characteristics at which the negative correlation between area human capital and mortality tapers off. In general, less educated areas in 1990 and areas with above median Black population have a larger negative correlation between area human capital and all-cause mortality, although the coefficients are not statistically different across the groups.

Over time, the largest increase in the coefficient on area human capital is in areas with a low share of college graduates in 1990. In these areas, which are largely white and rural, a 10 percentage point increase in area human capital in 2010 is associated with a three times larger decrease in all-cause mortality than a 10 percentage point increase in 1990. For areas with a high share of college graduates in 1990, the change is almost exactly the same between 1990 and 2010.

2.3.2 Area Human Capital and Non-Fatal Health Outcomes

While the bulk of our analysis focuses on mortality, we also examine the relationship between area human capital and health for non-fatal health outcomes. To some extent, such relationships are presaged by the findings for mortality, but these are also of independent interest because they allow us to compare the magnitude of the impact on disease prevalence relative to disease outcomes.

We examine the relationship between area human capital and health conditions using data from the HRS. We focus on new diagnoses of cancer, lung disease, diabetes, heart disease, and stroke – the major conditions asked about in the survey. With the BRFSS, we examine self-reported health.

Health-associated mobility is an issue in these analyses. To control for this, our HRS analysis characterizes people by the human capital of the area the respondent lived in when first entering the HRS, generally around ages 51-55. We discuss selective migration in more detail below.

Table 2.3 shows the relationship between area human capital and disease prevalence, controlling for individual education, other demographics (age, sex, race/ethnicity), and survey year. Columns 1-5 show the results for the HRS diagnosis measures. Area human capital is negatively and statistically associated with new onset of lung and heart disease cases. The effects are large, roughly 7-9 percentage points decline in incidence for a 10 percentage point increase in the share of people with a college degree. Area level human capital is not associated with the onset of other conditions. Both lung and heart disease are smoking and obesity-related, suggesting that these behaviors are part of the causal pathway. Columns 6 and 7 show that area human capital is associated with an increase in the percent of BRFSS respondents self-reporting good, very good, or excellent health (roughly 0.75 percentage points for a 10 percent increase) and a reduction in the number of days in the last 30 days where the BRFSS respondent reported having poor physical or mental health (roughly 4%).

The question that we turn to now is why these results are found. We first present the theories and then discuss the empirical evidence.

2.4 Model of human capital spillovers and health

We posit three potential explanations for the observed relation between area-level education and health. The first is selective migration – innately healthier individuals move to better educated areas. The second theory is that higher human capital areas have more and/or better health-related amenities, such as less pollution and violent crime or better medical care. The third theory is that there are spillovers in information, time preferences, or other factors that lead people living in higher human capital areas to engage in more health-promoting behaviors. Legislation that increases the costs of healthy behavior, such as rules pertaining to smoking in public, can be seen as either a health-related amenity or a local behavioral norm, albeit one with the force of law.

We do not focus specifically on incomes, which are higher in high human capital areas. The reason is that any spatial equilibrium implies that high wages are offset by higher

Table 2.3: *Regression results of non-fatal health outcomes on area human capital*

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	Cancer per 100,000	**Lung dis per 100,000**	**Diabetes per 100,000**	**Heart dis per 100,000**	**Stroke per 100,000**	**Good, v good, or excellent health**	**Poor health, days in last 30 days**
	HRS 1992-2008	HRS 1992-2008	HRS 1992-2008	HRS 1992-2008	HRS 1992-2008	BRFSS 1999-2001, 2009-2011	BRFSS 1999-2001, 2009-2011
Area chars							
% college grads	6.5	-18.7**	-7.4	-37.8**	9.1	0.00075***	-0.01551***
	(11.5)	(8.2)	(10.5)	(16)	(9.1)	(0.00026)	(0.00474)
Individual educ							
High school	-135.9	-883.2***	-1,051.5***	-1,080.2***	-461***	0.10853***	-2.19451***
	-132.6	-140.6	-181.1	-220.1	-102.2	-0.00221	-0.11605
Some college	56.9	-980.1***	-1,185.6***	-959.4***	-625.9***	0.14766***	-2.56244***
	-135.5	-158.6	-213	-282.1	-130.4	-0.00264	-0.12364
College graduate	-101.7	-1,676.3***	-1,843.4***	-2,372.7***	-879.8***	0.23511***	-3.94072***
	-162.7	-152.3	-195.7	-256.7	-118	-0.00363	-0.12414
Observations	113,890	115,694	108,075	100,174	119,554	1,553,212	1,459,505
R-squared	0.0307	0.0228	0.0321	0.0579	0.0346	0.115	0.047
Dependent var mean	2,800	2,200	3,300	6,200	1,700	0.834	3.77
% increase from 10pp increase in % college	*2.30%*	*-8.50%*	*-2.20%*	*-6.10%*	*5.30%*	*0.90%*	*-4.10%*

Note.*** p<0.01, ** p<0.05, * p<0.1. Standard errors are clustered at the area level. Cancer, lung disease, diabetes, heart disease, stroke, and hospitalizations in the Health and Retirement Survey (HRS) were defined as conditions or hospitalizations reported since the prior wave. Area characteristics in the HRS are measured at the time of HRS entry. OLS regressions use sampling weights and include individual-level controls for 5-year age (25-29, 30-34, , 85+, missing) by sex by race (white non-Hispanic, Black non-Hispanic, other non-Hispanic, Hispanic, missing race/ethnicity) interactions, Behavioral Risk Factor Surveillance System (BRFSS) year or HRS wave. We exclude individuals with missing education. We also include controls for area log density and log population, percent Black, percent Hispanic, and industry shares.*

amenity prices.[16] Thus, we can reject this channel theoretically. That said, we also test this empirically later in the paper.

We start with some notation to be precise about the theories. Assume that individuals potentially live for two periods ($t = 1, 2$) and make health-related consumption decisions in the first period that impact their probability of survival in the second period. At $t = 1$, expected utility for a representative individual living in area k equals:

$$U(R, N, B, B_k) + b(B)h_i Q_k \beta V(E) \tag{2.1}$$

where T refers to traded goods bought at a numeraire price of 1, Nrefers to non-traded goods bought at an endogenous price of p_k^N, B refers to health-related behaviors (e.g., smoking, overeating, and taking medication) which are bought at an exogenous price of p_k^B, and B_k refers to the average level of health-related behaviors in area k. At $t = 2$, the individual achieves nonnegative utility equal to $V(E)$ if the person is living and zero otherwise, where E denotes the individual's human capital, perhaps translated into wages.

We assume that there are two levels of human capital (E_H and E_L) and we denote $V(E_x) = V_x$ for $x = L, H$ where $V_H > V_L$. Second period utility is discounted by a discount factor β and multiplied by the survival probability,$b(B) * h_i * Q_k$, which has three components: $b(B)$, a decreasing function of the individual's health-related behavior B; h_i which denotes the individual's innate well-being, which determines the probability of not dying from causes unrelated to the behavior; and Q_k, which denotes area-specific health-related factors such as health care quality and other health-related area attributes, including the social and physical environment (e.g., pollution, crime, health-related regulations). Assume that $b(B) = \max(1 + d_0 B, 0)$, with $d_0 < 0$; thus, $b_B(B) \leq 0$ and B represents harmful health-

[16]This is true empirically. A 10 percentage point increase in the percent college graduates in an area measures (controlling for our baseline set of covariates) is associated with: 1.3 fewer homicides per 100,000; 0.6 additional physicians per 1,000; 1 percentage point increase in the share of women receiving mammograms; 0.06 for hospital quality z-score; we find no relationship between pollution and primary care visits after controlling for our baseline set of covariates. The one exception is that areas with higher human capital have fewer hospital beds per 1,000, perhaps because people are healthier.

related behaviors.

To derive exact solutions, we assume that $U(T, N, B, B_k) = T + g(N) + b_0 B - \frac{b_1}{2} B^2 - \frac{b_2}{2}(B - B_k)^2$, where all parameters are positive. Utility is linear in traded goods consumption and concave in non-traded goods consumption ($g'(N) > 0; g''(N) < 0$). Without reference effects, utility is concave in consumption of health-harming goods. In addition to that concavity, people get utility from having similar consumption as their peers. In this model, there are direct peer effects in health-related behaviors (i.e., $B - B_k$ enters utility directly); empirically, we test whether more educated individuals shift the behavioral norm in the community via information spillovers or policies and legislation targeting health-related behaviors that make unhealthy behaviors costlier.

For convenience, there is no saving between the two periods, so $Y_k^x - p_k^N N - p_k^B B$ equals consumption of the traded good, where Y_k^x refers to the earnings in location k of an individual with education $x = L, H$. At an interior equilibrium, all individuals consume all three goods, so that every person's consumption of N satisfies $g\prime(N) = p_k^N$.

Assume that $\frac{d^2 U(T,N,B,B_k)}{dB dB_k} > 0$, so that individuals enjoy higher utility from any health-related behavior when more individuals in their area also engage in the health-related behavior. Previous studies suggest that smoking, obesity, healthy eating, depressive symptoms, sleep, substance abuse, and other related behaviors are complementary across individuals with close social or geographical ties (Christakis and Fowler, 2007, 2008; Fowler and Christakis, 2008b,a; Cacioppo *et al.*, 2009; Mednick *et al.*, 2010; Cutler and Glaeser, 2010; Rosenquist *et al.*, 2011).

Low-human capital individuals are immobile and have an area-specific health-level of $h_L = h_L^k$ reflecting environmental factors in their area. They work providing non-traded goods and are each able to produce n_k units of non-traded services. Highly educated individuals produce traded goods, where are produced using a constant returns to scale technology where productivity and wage per worker equals W_H^k. All high human capital individuals

have health of $h_H > h_L^k$.

In Proposition 2.4.1 below, we assume that there is an exogenous share of high education individuals living in area k, denoted σ_k. In Proposition 2.4.2, we allow the highly educated to move and impose a spatial equilibrium so that their lifetime expected utility must equal a reservation value of U_H.

Proposition 2.4.1. *Unhealthy behavior is higher for the less educated group, and the levels of unhealthy behavior for both groups and for the area overall are decreasing with the share of the population that is highly educated.*[17]

Human capital spillovers stem from peer effects in unhealthy behavior. Better educated people engage in less unhealthy behavior because they value longevity and thus derive more utility form an increased probability of survival. A greater share of the population that is educated then shifts the behavioral norm in the community, which makes unhealthy behavior costlier for everyone. The desire to conform with the area-wide average means that factors that increase the share of the population that is educated will shape the health of the area, as we show in Proposition 2.4.2:

Proposition 2.4.2. *Increases in W_k^H, h_k and Q_k will cause (1) the share of the area that is educated to increase, (2) the level of unhealthy behavior for both high and low education groups in the area to decline and (3) the probability of survival for both groups to increase.*

Proposition 2.4.2 highlights three forces that can induce an increase in the education level of a place. First, a place with a more productive skill-intensive export sector will attract more educated individuals, which is unsurprising. Second, a place where less educated people are innately healthier will attract more skilled individuals. This is because health is associated with less engagement in health-harming behavior, and people like to move to areas where others consume like them. Thus, higher skilled people will move to healthier

[17] Proposition proofs are contained in Section B.3.

areas more – recall that only highly educated people are mobile in this model. Third, a place with better health-related amenities will also attract the educated.[18]

Higher education levels in an area may induce more skilled doctors to move to that area since they might prefer living around other skilled individuals or because demand for health care services is higher. Individuals may also be more willing to vote for public investments related to medical care, public health, or external stressors in areas with higher human capital. Additionally, better educated patients may provide more discipline for doctors, hospitals, and insurers in terms of providing high-quality care. The spatial aggregation of the highly educated (and better paid) may also generate greater demand for medical care and lead to quality improvements associated with scale and specialization. Higher area human capital may be associated with healthier physical and social environments, which we also consider to be health-related amenities. Note that our model assumes that our model assumes that health-related amenities are exogenously given for each area, but we empirically examine how differential investment in health-related amenities across areas with different human capital relates to externalities to health.

2.5 Testing Explanations: Sorting, Behaviors, and Amenities

Our first hypothesis is that the relationship between area human capital and health is explained by spatial sorting: healthy individuals move to areas with higher human capital or less healthy individuals move to areas with lower human capital. We focus on this hypothesis first because under this hypothesis, area human capital would not have a direct effect on health, but rather would attract healthier migrants for other reasons, e.g., because they can afford the higher housing prices, have preferences for amenities catering to healthier individuals (e.g., healthier food, gyms, etc.), or because they prefer living among similar individuals.

[18]This result can easily persist in a model where both education groups are mobile, as long as the highly educated individuals value health more than less educated individuals.

To test this hypothesis, we use data from the HRS. We create a measure of health status as the predicted probability of death in the next two years, given information on demographics and health conditions.[19] We then estimate a probit model for migration to another county in the next two years, using our baseline health measure as the main explanatory variable and individual demographic and area-level controls as in the previous analyses as controls.

The results are shown in Table 2.4. The first two columns show that people who are less healthy are more likely to move across counties that people who are healthier. This is consistent with Finkelstein *et al.* (2016, 2021), who report similar findings using Medicare data. The key question is how health status relates to the health of the county that people move to. The second column relates baseline health status to the difference in average human capital of the destination county minus average human capital in the origin county for movers. Contrary to the selection theory, there is no statistically significant effect of baseline health status on the relative human capital of the origin and destination counties.

We also consider shorting at younger ages, using data from the NLSY. The NLSY sample was aged 26-38 in 1990 and 46-58 in 2010. Thus, the ages just precede the HRS. Young men were asked in 1969-1971 and 1976 whether they had moved to a different SMSA or county since the last interview, and young women were asked annually or every two years between 1968-2001 whether they had moved to a different standard metropolitan statistical area (SMSA) or county since the last interview. We use a similar approach to the HRS. We start by predicting the probability of dying between the current and next interview using a probit model relating death to demographic and health characteristics in the current interview wave. Because the surveys asked different health-related questions for men vs. women, we use different predictors for the two groups and report results separately. For men, the controls include 5-year age categories by race/ethnicity interactions,

[19]Specifically, we estimate a probit model for mortality including indicators for whether the respondent was working, baseline risk factors such as high blood pressure, ever and current smoker, BMI, and medical history (ever had heart disease, lung disease, cancer, stroke, arthritis, psychological conditions, hospitalizations).

Table 2.4: *Spatial sorting, baseline health status, and selective migration*

	(1)	(2)	(3)	(4)	(5)
Dependent variable	**Migrated to new county in next 2 years**	**% college in destination - minus - % college in origin**	**Migrated to new SMSA/county**	**Stayed in SMSA or moved to SMSA from non-SMSA**	**Migrated to new SMSA/county**
	HRS 1992-2008	HRS 1992-2008	NLSY Young Women 1968-2001	NLSY Young Women 1968-2001	NLSY Young Men 1969-1971 1976
Model	Probit, dy/dx (SE)	OLS, coef. (SE)	Probit, dy/dx (SE)	Probit, dy/dx (SE)	Probit, dy/dx (SE)
Baseline health	0.051**	0.092	0.162**	0.568	-0.114
	(0.022)	(5.23)	(0.064)	(0.385)	(0.172)
Observations	71,717	3,101	50,772	3,010	4,527
R-squared	0.0031	0.017	0.0341	0.08	0.0318
Dependent var mean	0.043	-0.42	0.094	0.573	0.092

Note.*** p<0.01, ** p<0.05, * p<0.1. Standard errors are clustered at the area level. All regressions use sampling weights and control for individual education, 5-year age (25-29, 30-34, , 85+) by sex by race (white non-Hispanic, Black non-Hispanic, other non-Hispanic, Hispanic) interactions, National Longitudinal Survey of Youth (NLSY) wave or Health and Retirement Survey (HRS) survey wave, Black, and Hispanic. In the NLSY Young Men regressions, we use 1-year age categories instead of 5-year due to similar ages in the sample. Columns 1-2 additionally control for area characteristics: log population, log density, and industry shares. Baseline health in the HRS regressions was measured as the probability of death between the current and next interview and as probability of dying by 2011 in the NLSY regressions; it is estimated in a separate probit regression of mortality on measures of health status in the current interview and demographics*

individual education, year, whether the individual had any health limitations interfering with work, school or other activities, and the type and duration of health limitations. Additional controls for women include BMI, whether they were a current smoker, whether they currently have angina, hypertension, congestive heart failure, whether they have ever had an acute myocardial infarction or cancer, and whether they have any health limitations affecting school, work, or other activities. We then relate baseline health to the probability of moving to a new SMSA or county before the next interview, using a probit model.

The results are shown in columns 3-5 of Table 2.4. Column 3 shows that young women in worse baseline health are more likely to move to a new SMSA or county. This is not true among young men, as shown in column 5, but this estimate is noisy. We do not observe geographic identifiers in the NLSY, but column 4 further shows that among young women who move to a new SMSA or county, those of worse baseline health are more likely to remain in an SMSA or move to a SMSA from a location that is not an SMSA. If we consider SMSAs to be urban, high human capital areas relative to non-SMSAs, this is consistent with the idea that those with worse baseline health are more likely to move to high human capital areas. We thus take the NLSY results as suggestive evidence that the sorting hypothesis might not hold for younger adults either.

2.5.1 Health Behaviors

We now turn to our second hypothesis, which suggests that area human capital affects health-related behaviors. We focus on the two behavioral health risk factors that contributed the most to mortality in the U.S. in 2000 – smoking and obesity (Mokdad *et al.*, 2004; Cutler and Lleras-Muney, 2010) – and which already relate to area human capital as shown in Figure 2.2.

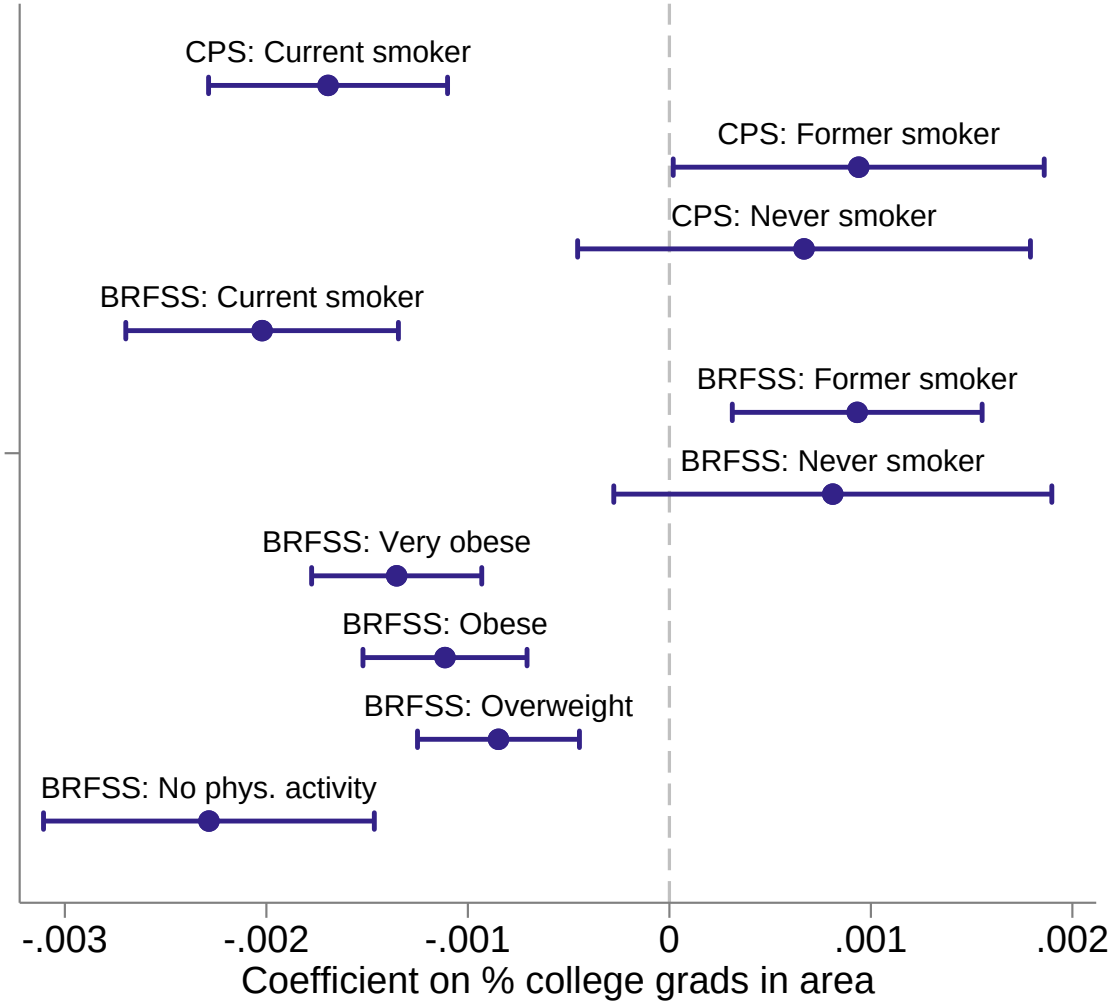
We use data on smoking status and obesity from the BRFSS and smoking status from the CPS, each matched to area characteristics measured in the decennial census or ACS wave

immediately preceding the given year.[20] We use similar regression models as for our baseline mortality regressions in column 3 of Table 2.2 but instead of mortality as the dependent variable we use whether the individual was a current, former, or never smoker, three categories of obesity based on BMI (very obese [BMI>35], obese [35≥BMI>30], and overweight [30≥BMI>25] vs. normal or underweight), and whether the individual reported being mostly physically inactive (vs. being physically active). We also use probit instead of OLS for estimation since all outcomes are binary variables.

Figure 2.5 shows the coefficients and standard errors for area human capital. Area human capital is strongly negatively correlated with the probability of being a current smoker and being obese. The coefficient on area human capital for current smoking is similar in the two data sets and implies that individuals living in areas with 10 percentage points more college graduates are 1.7 percentage points less likely to be currently smokers, equivalent to a 10.3% decrease in the probability of smoking relative to the average smoking rate. People who live in high human capital areas are both more likely to have never smoked and more likely to have quit smoking. Area human capital is also statistically significantly associated with lower probability of being overweight or obese – a 10 percentage point increase in the percent college graduates in an area is associated with a 15% lower probability of being very obese and a 7% lower probability of being obese. Consistent with these findings, people are also less likely to engage in no physical activity in areas with higher human capital. These findings closely align with causal neighborhood effects on obesity from the Moving to Opportunity experiment (Ludwig *et al.*, 2013).

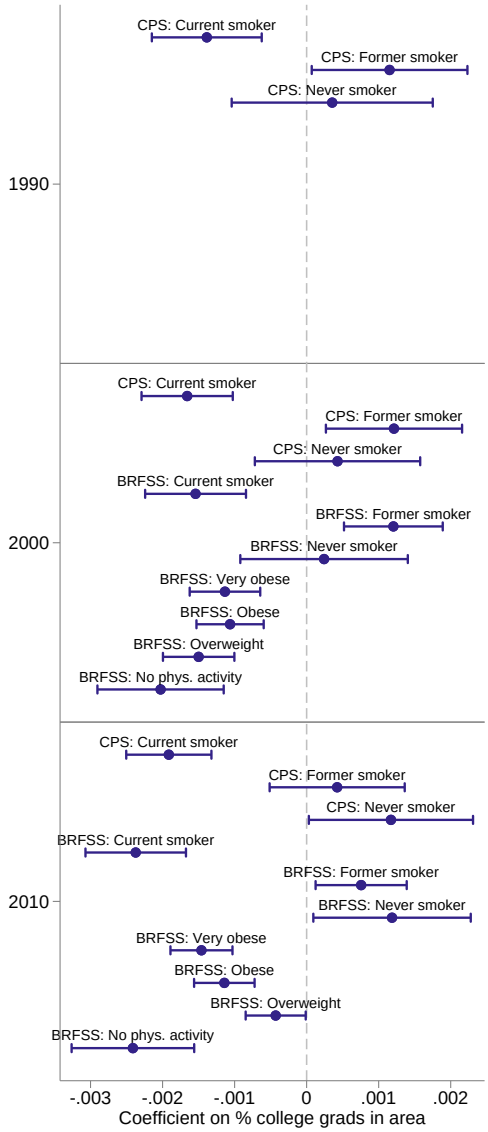
Not only do are these results apparent in the cross-section, they are true in the time series as well. Figure 2.6 shows that the correlation between area human capital and the probability of being a current smoker, never smoker, very obese, and physically active increases over time.

[20] E.g., 1995 is merged to area data from the 1990 census, 2003 to area characteristics from the 2000 census, and 2014 to area data from the 2009-2011 ACS.



Note. *This figure plots the coefficient on area human capital estimated separately for each smoking-related and obesity-related behavior, all of which are defined as binary variables. All probit regressions pool data from the 1999-2001 and 2009-2011 Behavioral Risk Factor Surveillance System (BRFSS) or the Tobacco Use Supplement in the Current Population Survey (CPS) from waves 1995-1996, 1998-1999, 2001-2002, 2003, 2006-2007, 2010-2011, and 2014-2015. All regressions use sampling weights and include individual-level controls for 5-year age (25-29, 30-34, , 85+, missing) by sex by race (white non-Hispanic, Black non-Hispanic, other non-Hispanic, Hispanic, missing race/ethnicity) interactions, individual education, and year. We exclude individuals with missing education. Area-level percent college graduates in each year was measured using data from the immediately preceding census or 3-year ACS. We also include controls for area log density and log population, percent Black, percent Hispanic, and industry shares, defined similarly as percent college graduates.*

Figure 2.5: *Regression results of health-related behaviors on area human capital*



Note. *This figure plots the coefficient on area human capital estimated separately for each smoking-related and obesity-related behavior, all of which are defined as binary variables. All probit regressions pool data from the 1999-2001 and 2009-2011 Behavioral Risk Factor Surveillance System (BRFSS) or the Tobacco Use Supplement in the Current Population Survey (CPS) from waves 1995-1996, 1998-1999, 2001-2002, 2003, 2006-2007, 2010-2011, and 2014-2015. All regressions use sampling weights and include individual-level controls for 5-year age (25-29, 30-34, , 85+, missing) by sex by race (white non-Hispanic, Black non-Hispanic, other non-Hispanic, Hispanic, missing race/ethnicity) interactions, individual education, and year. We exclude individuals with missing education. Area-level percent college graduates in each year was measured using data from the immediately preceding census or 3-year ACS. We also include controls for area log density and log population, percent Black, percent Hispanic, and industry shares, defined similarly as percent college graduates.*

Figure 2.6: *Regression results of health-related behaviors on area human capital by year*

To see how much these variables can explain of the area effect on mortality, we estimate our central model in column 3 of Table 2.2 including measures of smoking and obesity in the area. For smoking, we use the CPS data for the 1990s and the BRFSS data after 1999, when the CPS data is missing for a given area. Area-level data on obesity comes from the 1999-2001 and 2009-2011 from the BRFSS and is only available for those years.[21] Even with these noisy measures of smoking and obesity, Table 2.5 shows that controlling differential rates of smoking explains about 40% of the effect of area human capital on all-cause mortality (as demonstrated by difference in the coefficients between column 1 and column 2). Further controlling for obesity explains 88% of the correlation between area human capital and mortality in 2000 and 2010, with roughly equal contributions from the two behaviors. Smoking and obesity are particularly good for explaining the correlation between area human capital and deaths due to medically amenable, smoking-related, cancer, and chronic lower respiratory disease deaths, as shown in Table B.2. Furthermore, if we control for trends in smoking and obesity in regressions of all-cause mortality on area human capital by year, differences in these trends can also explain the strengthening correlation between area human capital and mortality, shown in Table B.1.

2.5.2 Health-Related Amenities

We next turn to whether some of the difference in mortality across areas can be explained by a correlation between area education and health amenities. We focus on two external stressors – air pollution and crime – while acknowledging that more environmental factors beyond these two may affect health. We also control for health care quality. Adverse health effects due to exposure to air pollution include increased lung disease incidence or aggravation of existing lung disease, cancer, and premature death (Environmental Protection Agency, 2023). High levels of air pollution may also discourage outdoor exercise and thus indirectly impact mortality through obesity. Higher area human capital could also

[21]Since not all areas are represented in the CPS and BRFSS, we estimate the models including smoking, obesity, and physical activity among cells where we have available data on these behaviors. Thus, the number of observations and average mortality rates used in Figure 2.7 is lower than the ones reported in Table 2.2.

Table 2.5: *Regression results of all-cause mortality per 100,000 on area human capital and health-related behaviors*

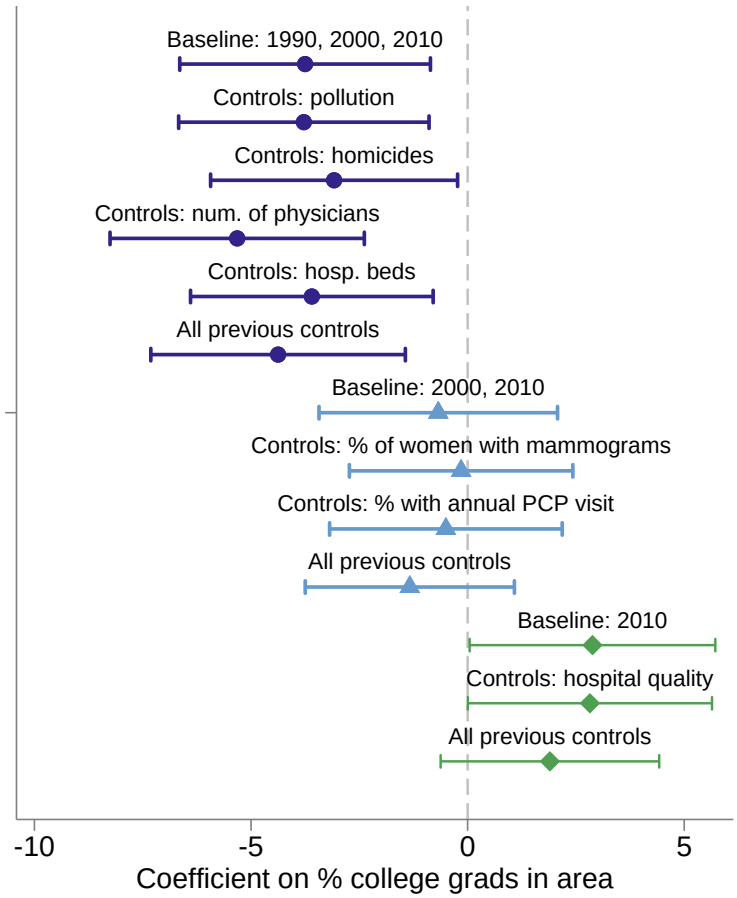
	(1)	(2)	(3)	(4)	(5)	(6)
	Cells with non-missing data on smoking behavior		**Cells with non-missing data on smoking behavior and obesity-related behaviors**			
	1990-2010		2000-2010			
Area characteristics						
% college graduates	-6.49***	-3.89***	-5.96***	-3.00*	-4.85***	-0.67
	(1.31)	(1.45)	(1.35)	(1.55)	(1.39)	(1.4)
% current smoker		5.86***		7.19***		8.06***
		(2.05)		(2.51)		(1.75)
% former smoker		-3.88***		-2.68*		-3.53***
		(1.45)		(1.41)		(1.36)
% overweight, obese, very obese					3.30**	5.68***
					(1.3)	(1.58)
Cells	391,485	391,485	325,708	325,708	325,708	325,708
Areas	484	484	484	484	484	484
R-squared	0.87	0.87	0.875	0.875	0.875	0.876
Dependent var. mean	1,164	1,164	1,173	1,173	1,173	1,173
% change from 10pp increase in % college	*-5.60%*	*-3.30%*	*-5.10%*	*-2.60%*	*-4.10%*	*-0.60%*

Note.*** p<0.01, ** p<0.05, * p<0.1. *Standard errors are clustered at the area level. OLS regressions are estimated at the area-year-age-sex-race-education cell level, weighted by cell population, and pooled across 1990, 2000, and 2010. All regressions control for cell-level 5-year age (25-29, 30-34, , 85+) by sex by race (white non-Hispanic, Black non-Hispanic, other non-Hispanic, Hispanic) interactions, individual education, percent of death certificates without education information, and year. We also include controls for area log density and log population, percent Black, percent Hispanic, and industry shares. The percent of individuals that were current or former smokers were calculated using the 1995-1996, 1998-1999 CPS, 1999-2001 BRFSS, and 2009-2011 BRFSS. The percent of individuals that were overweight, obese, or very obese, and those with no physical activity were calculated using the 1999-2001 and 2009-2011 BRFSS.*

be associated with less pollution because air quality may be priced into property values, leading to selection of the better educated (and wealthy) into such areas. While homicides are a crude measure that may not capture all aspects of crime, they are more reliably reported than other crimes (Bureau of Justice Statistics, 1994). Crime could also decrease health through indirect channels; for example, unsafe streets could increase stress, lead residents to stay inside and get less exercise, or make it difficult to obtain necessary health care or management of chronic conditions. Additionally, pollution and crime might be lower in more educated areas for similar reasons that demand for high-quality medical care might be higher – people in higher human capital areas may vote more for public goods addressing environmental stressors and may possess the political clout to regulate crime and pollution.

Figure 2.7 examines whether controlling for differences in health-related amenities across areas can explain the correlation between area human capital and mortality above and beyond what is explained by behaviors such as smoking and obesity. The first set of models in Figure 2.7 are our most comprehensive models. They work from the models in Table 2.5 and sequentially add in pollution and crime data. The second set of models subsets the sample to 2000-2010, where we also have obesity data. The last set of models uses data for 2010 only, and also controls for health care demand and quality.

In total, external factors such as pollution and homicide rates explain a very small share of the relationship between area human capital and mortality after controlling for smoking and obesity. Both pollution and homicides are correlated with mortality, but neither explains much of the relationship between area human capital and mortality. Similarly, measures of health care demand and quality such as number of physicians, hospital beds, and health care quality, which are also correlated with mortality, cannot explain the effect of area human capital on mortality beyond what is explained by differences in smoking and obesity.



Note. *This figure plots the coefficient on area human capital estimated separately with the specified controls. In addition to our baseline set of covariates (discussed in detail below), the baseline regression for 1990-2010 controls for percent of population that is currently smoking or formerly a smoker, the baseline regression for 2000-2010 and the baseline regression for 2010 control for smoking behavior and the percent of the population that is overweight, obese, or very obese. All regressions are estimated at the area-year-age-sex-race-education cell level, weighted by cell population, and pooled across 1990, 2000, and 2010. All regressions further control for cell-level 5-year age (25-29, 30-34, , 85+) by sex by race (white non-Hispanic, Black non-Hispanic, other non-Hispanic, Hispanic) interactions, individual education, percent of death certificates without education information, and year. We also include controls for area log density and log population, percent Black, percent Hispanic, and industry shares. Confidence intervals are clustered at area level.*

Figure 2.7: *Regression results of all-cause mortality per 100,000 on area human capital and health-related amenities*

2.6 Understanding Health-Related Behaviors

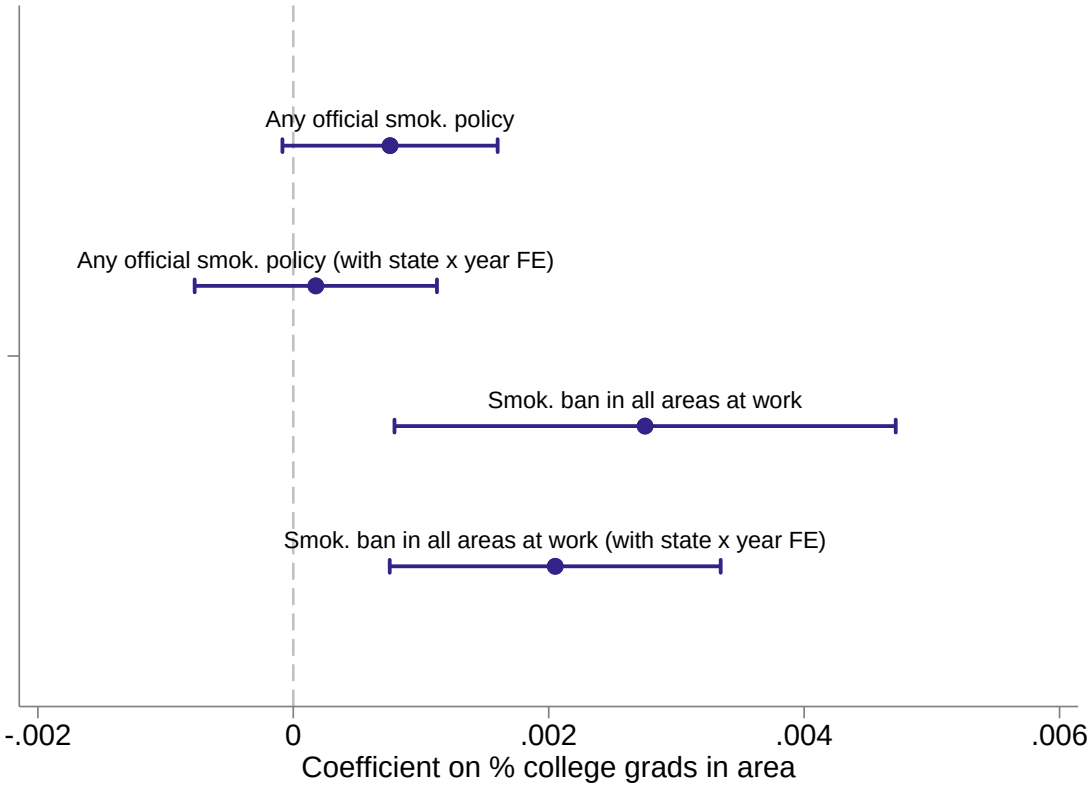
2.6.1 Prices

Given the prominent role of health-related behaviors in explaining the correlation between area human capital and mortality, we consider why they are so related to area human capital. One possible theory is through prices. More educated areas may be more likely to support legislation and regulations aimed at improving health. For example, this may include tobacco control policies such as tobacco taxes, clean indoor air laws, and workplace smoking bans. Tobacco taxes are typically regulated at the federal or state level. States may also mandate clean indoor area laws in some places (e.g., in workplaces, restaurants, and bars). Thus, tobacco taxes and state clean indoor air laws and regulations will typically vary by state and year. Private workplace smoking bans can be implemented as company policy independent from law, and thus may vary within states and years.

From our results that include state by year fixed effects, we know that not all of the impact of area human capital is through state-level policies. Thus, we focus on workplace smoking bans. The CPS data described above ask questions on workplace smoking policies for indoor workers. We focus on whether the workplace has an official smoking policy in place (which is likely a regulation) and whether the workplace bans smoking in all public and work areas.

Figure 2.8 shows the impact of area human capital on these policies. Controlling for the individual's own education, individuals living in more educated areas are more likely to work at places with a complete ban on smoking in all public and work areas. A worker with a 10 percentage point higher share of college graduates is 3% more likely to be employed at places with a complete smoking ban. Part of this relationship can be explained by state-level smoking legislation, as shown by the coefficients that include state by year interactions in Figure 2.8. However, the relationship between area human capital and workplace smoking bans is large and statistically significant even after including state-year fixed

effects. The relationship between smoking bans and area human capital is much stronger for low education workers living in high education area than for the highly educated.



Note. *This figure plots the coefficient on area human capital estimated separately for each outcome, all of which are defined as binary variables. All probit regressions pool data from the Tobacco Use Supplement in the Current Population Survey (CPS) from waves 1995-1996, 1998-1999, 2001-2002, 2003, 2006-2007, 2010-2011, and 2014-2015, use sampling weights, and include individual-level controls for 5-year age (25-29, 30-34, , 85+, missing) by sex by race (white non-Hispanic, Black non-Hispanic, other non-Hispanic, Hispanic, missing race/ethnicity) interactions, individual education, and year. We exclude individuals with missing education. Area-level percent college graduates in each year was measured using data from the immediately preceding census or 3-year ACS. We also include controls for area log density and log population, percent Black, percent Hispanic, and industry shares, defined similarly as percent college graduates. Confidence intervals are clustered at the area level.*

Figure 2.8: *Regression results of workplace smoking policies on area human capital*

A number of papers discuss the effectiveness of these bans in reducing smoking. For example, Evans *et al.* (1999) show that compared to a firm with little restrictions on smoking, adopting a smoke-free policy at a workplace reduces the probability of smoking by 5.7 percentage points and decreases the daily number of cigarettes smoked by 14% on average.

In our own data, we can examine how general state policies and working smoking bans affect tobacco use. Table 2.6 shows the relationship between area human capital and the probability of smoking, controlling for various state policies. Without state controls, an area with a 10 percentage point greater share of college graduates has a 1.4 percentage point lower smoking rate (column 1). Controlling for time-varying state-level policies such as tobacco taxes and state-level clean indoor air laws (through state-year fixed effects) and the presence of a workplace ban reduces the impact of area human capital by about a third (columns 2 and 3). Columns 4-6 show that some of this comes from increased quitting, while the rest comes from non-initiation.

2.6.2 Peer Effects

A second theory is that area human capital drives peer effects. For instance, the proximity of more educated individuals undertaking healthy behaviors may encourage individuals across the education distribution to undertake healthy behaviors themselves. Furthermore, differences in information and beliefs about the harmful effects of smoking and obesity, which may correlate with area human capital, may also be driving these differences in smoking behavior across areas. While we cannot directly assess direct peer effects because these inherently reflect preferences not captured in our data, we can examine informational spillovers. The 1987, 1992, and 2000 National Health Interview Surveys (NHIS) asked individuals were asked about their agreement with a series of statements about the effects of smoking on health: smoking by pregnant women is harmful for baby, someone else's smoke is harmful, and most lung cancer deaths are caused by smoking, among others. We consider how these are related to area education.[22]

Table 2.9 and Table 2.8 show regression results. NHIS respondents living in counties with a 10 percentage point higher percent of college graduates are 11% more likely to agree with

[22]Each year in the NHIS data was merged to area characteristics measured in the decennial census immediately prior to the given year (i.e., 1987 is merged to area characteristics from 1980, 1992 to area data from 1990, 2000 to area data from 2000).

Table 2.6: *Regression results of smoking behavior on area human capital and workplace smoking policies*

	(1)	(2)	(3)	(4)	(5)	(6)
	Current smoker			**Former smoker**		
Area characteristics						
% college graduates	-0.00134***	-0.00100**	-0.00092**	0.00051	0.00023	0.00024
	(0.00032)	(0.00041)	(0.00042)	(0.00044)	(0.00044)	(0.00044)
Individual characteristics						
Smoking banned in			-0.04036***			-0.00123
public/work areas at work			(0.00284)			(0.00312)
Individual education						
High school graduate	-0.03806***	-0.03795***	-0.03646***	0.04201***	0.04161***	0.04165***
	(0.00682)	(0.0067)	(0.00665)	(0.00537)	(0.00527)	(0.00525)
Some college	-0.07992***	-0.07888***	-0.07633***	0.05820***	0.05740***	0.05747***
	(0.00829)	(0.00816)	(0.00804)	(0.0055)	(0.00539)	(0.00538)
College graduate	-0.18248***	-0.18193***	-0.17770***	0.02314***	0.02235***	0.02247***
	(0.0112)	(0.01109)	(0.01088)	(0.00536)	(0.00534)	(0.00531)
Post-graduate education	-0.23795***	-0.23775***	-0.23216***	0.01086*	0.01007*	0.01023*
	(0.01167)	(0.01152)	(0.01124)	(0.00584)	(0.00587)	(0.00585)
State-year FE	No	Yes	Yes	No	Yes	Yes
Cells	172,907	172,817	172,817	172,928	172,927	172,927
Areas	297	297	297	297	297	297
R-squared	0.071	0.076	0.078	0.057	0.061	0.061
Dependent variable mean	0.158	0.158	0.158	0.196	0.196	0.196
% change from 10pp increase in % college grads	*-8.50%*	*-6.30%*	*-5.80%*	*2.60%*	*1.20%*	*1.20%*

Note.*** p<0.01, ** p<0.05, * p<0.1. *Standard errors are clustered at the area level. All probit regressions pool data from the Tobacco Use Supplement in the Current Population Survey (CPS) from waves 1995-1996, 1998-1999, 2001-2002, 2003, 2006-2007, 2010-2011, and 2014-2015, use sampling weights, and include individual-level controls for 5-year age (25-29, 30-34, , 85+, missing) by sex by race (white non-Hispanic, Black non-Hispanic, other non-Hispanic, Hispanic, missing race/ethnicity) interactions, individual education, and year. We exclude individuals with missing education. Area-level percent college graduates in each year was measured using data from the immediately preceding census or 3-year ACS. We also include controls for area log density and log population, percent Black, percent Hispanic, and industry shares, defined similarly as percent college graduates*

the statement that smoking is harmful for pregnant women's babies and 15% more likely to agree that most lung cancer deaths stem from smoking, controlling for individual education. These results are statistically significant at the 10% level and 5% level, respectively. As the next columns show, individuals living in more educated areas are also more likely to support smoking bans in bars, restaurants, and work areas.

In a similar exercise as for smoking regulations, we examine whether area human capital is correlated with smoking behavior after controlling for individual education and beliefs about second-hand smoking, specifically whether smoking should be banned in bars, restaurants, and workplaces. These results, shown in Table 2.9, suggest that controlling for state-year fixed effects and smoking bans at work explain 5.5% and 4.5% of the correlation between area human capital and the probability of being a current smoker and former smoker, respectively. The relationship between area human capital and quitting smoking is quite noisy. Further controlling for agreement with the statement that smoking should be banned in bars, restaurants, and workplaces explains 23.4% of the correlation between area human capital and smoking behavior.

2.7 Conclusion

Our paper documents a strong and robust relationship between area human capital and mortality, even after controlling for individual education. This relationship appears to be a recent phenomenon, having emerged in 2000 and particularly 2010. The correlation between area human capital and mortality is strongest, on a relative scale, for individuals younger than 65 and white and Hispanic individuals and further extends to non-fatal health outcomes such as lung disease, heart disease, and number of days in poor physical or mental health. Medically-amenable, smoking-related, and obesity-related causes of death experience the largest spillovers from area human capital, which are also highest in the least educated areas in 1990.

We consider several pathways through which area human capital may impact health. Al-

Table 2.7: *Regression results of beliefs about smoking on area human capital*

	(1)	(2)	(3)
	Strongly agree or agree with the following statement:		
	Smoking by pregnant women is harmful	**Someone else's smoke is harmful**	**Most lung cancer deaths are caused by smoking**
Area characteristics			
% college grads	0.0103*	0.00328	0.0112***
	(0.00537)	(0.00336)	(0.00412)
Individual education			
High school graduate	0.218***	0.182***	0.122***
	(0.0455)	(0.0294)	(0.0413)
Some college	0.500***	0.335***	0.234***
	(0.0595)	(0.0345)	(0.0471)
College graduate	0.597***	0.541***	0.387***
	(0.0721)	(0.0377)	(0.0591)
Post-graduate	0.771***	0.695***	0.552***
	(0.0782)	(0.0422)	(0.0595)
Dependent variable mean	0.922	0.922	0.74
% change from 10pp increase in % college	*11.20%*	*3.60%*	*15.10%*

Note.*** p<0.01, ** p<0.05, * p<0.1. *Standard errors are clustered at the area level. Outcomes were defined relative to having no opinion, disagreeing, or strongly disagreeing. All probit regressions in columns 1-3 pool data from National Health Interview Survey (NHIS) from 1987, 1992, and 2000. All probit regressions in columns 4-6 pool data from the Tobacco Use Supplement in the Current Population Survey (CPS). All regressions, use sampling weights and include individual-level controls for 5-year age (25-29, 30-34, , 85+, missing) by sex by race (white non-Hispanic, Black non-Hispanic, other non-Hispanic, Hispanic, missing race/ethnicity) interactions, and year. We exclude individuals with missing education. Percent college graduates was measured at the county level using data from the immediately preceding census for the given year for NHIS regressions and from the immediately preceding census or 3-year ACS for the CPS regressions. We also include controls for area log density and log population, percent Black, percent Hispanic, and industry shares, defined similarly as percent college graduates.*

Table 2.8: *Regression results of beliefs about smoking on area human capital*

	(1)	(2)	(3)
	Strongly agree or agree with the following statement:		
	Smoking should be banned in bars	**Smoking should be banned in restaurants**	**Smoking should be banned in work areas**
Area characteristics			
% college grads	0.00381***	0.00313*	0.00285***
	(0.00124)	(0.00162)	(0.00107)
Individual education			
High school graduate	-0.03241***	-0.00892	0.01466***
	(0.00518)	(0.00581)	(0.00439)
Some college	-0.01654**	0.03696***	0.06028***
	(0.00664)	(0.00791)	(0.00654)
College graduate	0.04482***	0.09166***	0.11960***
	(0.00699)	(0.00991)	(0.008)
Post-graduate	0.08092***	0.13279***	0.15032***
	(0.00728)	(0.01146)	(0.00965)
Dependent variable mean	0.451	0.607	0.775
% change from 10pp increase in % college	*8.50%*	*5.20%*	*3.70%*

Note.*** p<0.01, ** p<0.05, * p<0.1. *Standard errors are clustered at the area level. Outcomes were defined relative to having no opinion, disagreeing, or strongly disagreeing. All probit regressions in columns 1-3 pool data from National Health Interview Survey (NHIS) from 1987, 1992, and 2000. All probit regressions in columns 4-6 pool data from the Tobacco Use Supplement in the Current Population Survey (CPS). All regressions, use sampling weights and include individual-level controls for 5-year age (25-29, 30-34, , 85+, missing) by sex by race (white non-Hispanic, Black non-Hispanic, other non-Hispanic, Hispanic, missing race/ethnicity) interactions, and year. We exclude individuals with missing education. Percent college graduates was measured at the county level using data from the immediately preceding census for the given year for NHIS regressions and from the immediately preceding census or 3-year ACS for the CPS regressions. We also include controls for area log density and log population, percent Black, percent Hispanic, and industry shares, defined similarly as percent college graduates.*

Table 2.9: *Regression results of smoking behavior on area human capital and beliefs about smoking bans*

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Current smoker				**Former smoker**			
Area chars								
% college grads	-0.00145***	-0.00143***	-0.00137***	-0.00105**	0.00074*	0.00071	0.00071	0.00077
	(0.00033)	(0.00048)	(0.00048)	(0.00046)	(0.00043)	(0.00048)	(0.00048)	(0.00047)
Individual chars								
Work smoking ban			-0.04331***	-0.02895***			-0.00055	0.00239
			(0.0034)	(0.0033)			(0.00353)	(0.00353)
Agree				-0.19492***				-0.03955***
with smoking ban				(0.00689)				(0.00259)
State-year FE	No	Yes	Yes	Yes	No	Yes	Yes	Yes
Cells	120,636	120,552	120,552	120,552	120,647	120,646	120,646	120,646
Areas	297	297	297	297	297	297	297	297
R-squared	0.073	0.077	0.08	0.14	0.059	0.063	0.063	0.065
Dependent variable mean	0.167	0.168	0.168	0.168	0.204	0.204	0.204	0.204
% change from 10pp increase in % college	*-8.60%*	*-8.50%*	*-8.20%*	*-6.30%*	*3.60%*	*3.50%*	*3.50%*	*3.80%*

Note.*** p<0.01, ** p<0.05, * p<0.1. *Standard errors are clustered at the area level. All probit regressions pool data from the Tobacco Use Supplement in the Current Population Survey (CPS) from waves 1995-1996, 1998-1999, 2001-2002, 2003, 2006-2007, 2010-2011, and 2014-2015, use sampling weights, and include individual-level controls for 5-year age (25-29, 30-34, , 85+, missing) by sex by race (white non-Hispanic, Black non-Hispanic, other non-Hispanic, Hispanic, missing race/ethnicity) interactions, individual education, and year. We exclude individuals with missing education. Area-level percent college graduates in each year was measured using data from the immediately preceding census or 3-year ACS. We also include controls for area log density and log population, percent Black, percent Hispanic, and industry shares, defined similarly as percent college graduates.*

most all of the correlation between area human capital and mortality can be explained by differences in smoking rates and obesity rates across areas due to the strong spillovers of area human capital on these health-related behaviors. We theoretically and empirically examine two channels driving these spillovers and find empirical evidence for both: policies that increase the price of unhealthy behaviors such as smoking, and peer effects about the harms of smoking.

As such, our findings suggest that even in the absence of direct effects of local and place-based labor or educational policies on health, any welfare analysis of such policies should incorporate spillovers on health. Since the spillovers we find are very large, and current estimates of the value of a statistical life are $7.4 million (from the EPA), we expect that the payoff of any local policy that increases the educational level of an area brings massive returns in terms of lives saved.

Chapter 3

Racial Disparities in COVID-19 Cases and Deaths: The Role of Socio-Economic vs. Health-Related Factors

3.1 Introduction

Throughout the COVID-19 pandemic in the U.S., large racial disparities in cases and deaths have emerged.[1] The reasons behind the high COVID-19 burden among non-white individuals are numerous. Non-white individuals are more likely to live in densely populated geographic areas or in denser living quarters due to housing segregation, high housing costs, or higher rates of incarceration, which may facilitate COVID-19 transmission. On the other hand, many COVID-19 outbreaks have occurred in nursing homes, and most nursing home

[1]For an excellent and thorough review of the most recent studies on COVID-19 disparities by race, see Racial Disparities in COVID-19: Key Findings from Available Data and Analysis by the Kaiser Family Foundation.

residents are white and non-Hispanic (Feng *et al.*, 2011).[2] Non-white individuals are more likely to work in essential occupations or occupations for which working from home is not possible (Rogers *et al.*, 2020). Non-white individuals may live further away from grocery stores, medical facilities, and workplaces, resulting in increased use of public transportation and higher risk of COVID-19 infection. These differences might further be amplified by or correlated with differences in income by race. Conditional on these factors, non-white individuals might be less likely to adhere to social distancing recommendations, less likely to follow guidelines for preventing infections, or be less informed about COVID-19 spread (Alsan *et al.*, 2020). Non-white individuals might be disproportionately affected by underlying comorbidities that increase illness severity, leading to higher COVID-19 mortality rates.[3] Pollution has also been shown to be highly correlated with COVID-19 mortality (Wu *et al.*, 2020), and non-white individuals may be disproportionately more likely to live in more polluted areas. Non-white individuals may have less access to proper health care, or have access to facilities of worse quality than white individuals. Finally, racial bias in the health care system, as well as other forms of systemic racism not captured by the aforementioned factors may affect infection spread, illness severity, and health care allocation among non-white populations.

This paper aims to examine the role of large set of socio-economic, health-related, and environmental factors on racial disparities in COVID-19 spread and disease severity across counties in the U.S., with a particular emphasis on disentangling the role of socio-economic vs. health-related factors in the differential COVID-19 spread by race. We use individual-level data on COVID-19 cases and deaths in the U.S., collected by the Center for Disease

[2]According to the Centers for Medicaid and Medicare Services, nursing home residents accounted for 3.95% of all COVID-19 cases but 25.31% of all COVID-19 deaths in the U.S. at the beginning of the COVID-19 pandemic.

[3]The CDC reports that asthma, cancer, cerebrovascular disease, chronic kidney, liver, and lung diseases, cystic fibrosis, diabetes, heart conditions, HIV, disabilities, depression, schizophrenia, dementia, obesity, physical inactivity, pregnancy, smoking, immunodeficiencies, tuberculosis, use of corticosteroids or other immunosuppressive medication, and solid organ or blood stem cell transplantation are the comorbidities with the strongest and most consistent evidence on increasing illness severity.

Control (CDC) and the National Center for Health Statistics and aggregated to age-race-county categories, which we combine with a variety of data sources on household density, population density, income, education, public transportation use, pollution, health care quality, comorbidities, and social distancing at the age-race-county level. We exclude small counties (<100,000 population) since these do not have population denominators by race used for the calculation of COVID-19 case and mortality rates per 100,000. We focus on the initial six months of the COVID-19 pandemic to (a) minimize confounding from endogenous COVID-19 prevention policies at the individual and county level and (b) focus on factors that contribute to the differential initial impact of the pandemic by race that can be addressed by policies implemented *today*.

First, we document stark racial disparities in COVID-19 in the first six months of the COVID-19 pandemic: Black and Hispanic individuals were 2.9 and 3.9 times more likely to contract COVID-19 relative to white individuals and were 2.2 and 1.3 times more likely to die from COVID-19 after adjusting for age. Similarly, after age adjustment, AIAN individuals were 2.9 times more likely to contract COVID-19 and 1.6 times more likely to die from COVID-19 relative to white individuals. While NHPI individuals were 3.2 times more likely to have COVID-19 compared to white individuals, they were less likely to die from COVID-19. Asian individuals also had a higher case and mortality rate relative to white individuals.

We find that after controlling for other socio-economic and health characteristics, both white and non-white individuals living in denser counties and counties with higher race-specific rates of public transportation use have higher COVID-19 case and mortality rates, highlighting the importance of density-related factors in the initial spread of COVID-19. Race-specific average household income is also negatively correlated with case and mortality rates across all racial groups, as are pollution levels.

Public transportation use is particularly strongly correlated with the Hispanic case rate – counties where the share of Hispanic individuals commuting to work using public trans-

portation is 10 percentage points higher are associated with a 13% higher average Hispanic COVID-19 case rate, controlling for other factors. Another factor that is strongly correlated with the Hispanic case rate, but not any other racial groups, is household density. Controlling for other county characteristics, counties where Hispanic individuals are 10 percentage points more likely to live in dense households, defined as living in group quarters, multigenerational household, or multifamily households are associated with a 11% higher Hispanic COVID-19 case rate. Furthermore, the correlation between education and case and mortality rates among Black individuals is particularly striking – counties where Black individuals are 10 percentage points more likely to have a college degree have, on average, a 10% lower Black case rate after controlling for other county characteristics. The correlation between education and case rates is much smaller for other racial groups. Because most counties with AIAN and NHPI populations are small and thus excluded from our analysis, many estimates for these racial groups are noisy, although population density and average AIAN/NHPI income are statistically significantly correlated with case and mortality rates for these groups.

We also find that health care quality and comorbidity rates are uncorrelated with COVID-19 case and mortality rates across all racial groups. However, conditional on infection, counties with lower health care quality have higher COVID-19 mortality rates for white individuals. Importantly, counties with an additional 10 percentage points of Black individuals with an underlying comorbidity are associated with a 3.75% higher share of Black individuals dying from COVID-19 if infected.

Given the differences in socio-economic, health-related, and environmental characteristics between white and non-white individuals, as well as the differences in correlates for race-specific case and mortality rates, we formalize the contributions of each to the observed case and death rate differentials by race using the Oaxaca-Blinder decomposition (Oaxaca (1973), Blinder (1973)). We decompose the average differences in COVID-19 case and mortality rates between Black and white and Hispanic and white individuals into three components:

one due to differences in average characteristics by race ("differences in endowments"), differences due to differential correlations between characteristics and case/mortality rates across racial groups ("differences in coefficients"), and differences due to the interaction between the levels and coefficients for each characteristic ("differences in interactions").

The Oaxaca-Blinder decomposition suggests that if Black individuals had the same observed characteristics as white individuals, keeping correlations between characteristics and COVID-19 burden constant, the case rate and mortality rate difference between Black and white individuals would decrease by 25% and 45% (respectively) relative to the average. This reduction largely stems from equalizing education and income levels, at to a smaller extent household density, which are particularly strong correlates of case and mortality rates among Black individuals. Similarly, equalizing observed characteristics between Hispanic and white individuals would reduce the average COVID-19 case and mortality rate difference between Hispanic and white individuals by 23% and 26%, respectively, relative to the average observed case and mortality rate differential. This decrease can be attributed to equalizing household density and income between white and Hispanic individuals.

On the other hand, if Black individuals had the same correlation between various factors and case and mortality rates as white individuals, but keeping the levels of characteristics across race as observed, the difference between Black and white individuals would fall by 33% for case rates and would even become negative for mortality rates. This is largely due to the fact that the correlation between population density (and to a smaller extent comorbidities) and case/mortality rates is stronger for Black individuals than for white individuals. Additionally, for Hispanic individuals, having equal correlations between characteristics and case/mortality rates as white individuals would explain away the entire case and mortality rate differential observed between Hispanic and white individuals. This result comes from the fact that population density and household density are highly correlated with case rates and mortality rates among Hispanic individuals.

Few papers have examined the role of a comprehensive set of socio-economic, environmen-

tal, and health-related factors in the increased COVID-19 burden among non-white individuals. Several papers explore the relationship between demographic, socio-economic, and health characteristics and COVID-19 cases and deaths and find strong correlations between racial composition, public transportation, and density-related measures and COVID-19 burden across all levels of geography (Borjas, 2020; Knittel and Ozaltun, 2020; McLaren, 2020; Benitez *et al.*, 2020; Almagro *et al.*, 2020). However, these studies do not explicitly focus on the differential COVID-19 case and mortality rates by race. This gap comes from the unavailability of data sources that include both an extensive set of socio-economic and health-related factors. Several studies using individual-level data from a variety of states and hospital systems have documented racial disparities in COVID-19 infections, hospitalizations, and deaths (Price-Haywood *et al.*, 2020; Yehia *et al.*, 2020; Ogedegbe *et al.*, 2020; Gu *et al.*, 2020; Alsan *et al.*, 2021). Some of these studies show that race is uncorrelated with COVID-19 illness severity after controlling for socio-economic and clinical factors; however, most socio-economic characteristics are limited to only education and household income. Our paper addresses this gap by: (a) analyzing a larger set of socio-economic and environmental characteristics, and (b) focusing on separating the role of socio-economic vs. health factors in both COVID-19 spread and COVID-19 illness severity.

The paper proceeds as follows. Section 3.2 describes the data used in this paper in detail. Section 3.3 documents racial disparities in COVID-19 nationwide and over time. It also describes differences in demographic, socio-economic, environmental and health characteristics by race. Section 3.4 explores correlates of COVID-19 case and mortality rates for each racial group. Section 3.5 presents the results from the Oaxaca-Blinder decomposition for Black and Hispanic individuals relative to white individuals. Finally, Section 3.6 concludes.

3.2 Data

3.2.1 COVID-19 cases and deaths

We obtain the most comprehensive data set on individual-level COVID-19 cases and deaths available in the U.S., collected by the Center for Disease Control (CDC) and the National Center for Health Statistics.[4] The data contains all COVID-19 cases reported to the CDC since the beginning of the pandemic (January 2020) until April 17, 2022, amounting to a total of 71.4 million cases. Each case is dated and reported alongside the state and county of residence of the individual, as well as age, sex, whether the case was hospitalized, whether the case ended up in the ICU, and whether the individual associated with the case report subsequently died from COVID-19.[5] Each case is also associated with race information: white, Black, Asian, American Indian/Alaska Native, Native Hawaiian/Pacific Islander, Hispanic, and other races.[6] After excluding COVID-19 cases with missing race data, our data contains 46 million cases, all of which are included in the descriptive statistics at the national level reported in Section 3.3.1.

We use the cumulative COVID-19 cases per 100,000 individuals, henceforth referred to as the "case rate" and the cumulative COVID-19 deaths per 100,000 individuals, henceforth referred to as the "mortality rate", as the main outcomes of interest. We use population counts by race from the 2010 Decennial Census (10% sample) to calculate the case and

[4]COVID-19 was added to the Nationally Notifiable Condition List on April 5, 2020 and was classified as "immediately notifiable, urgent (within 24 hours)". However, COVID-19 case surveillance data are collected by jurisdictions and shared voluntarily with CDC.

[5]Geography and demographics (including race) are suppressed for low frequency (<5) records.

[6]Figure C.1 plots the share of cumulative COVID-19 cases reported to the CDC up to April 17, 2022 that have missing race information. As this figure shows, North Dakota did not report race information for its COVID-19 cases. Washington, Texas, New York, and Connecticut also have a particularly high rate of missing race data. As Desmet and Wacziarg (2020) discuss, if measurement error in cases and deaths due to testing and reporting is random, then the estimates in this paper will have higher variance but will remain unbiased. However, if there are systematic differences in testing and reporting across racial groups, all analyses in this paper would combine the effect of the COVID-19 incidence, testing, and reporting into one effect. Conditional on reporting race data, there is little reason to suspect measurement error in reporting of race information in the CDC data since all COVID-19 case report forms used the same race categories, so we would expect unbiased but higher variance estimates.

mortality rates nationwide.

For all regression analyses, cases and deaths were first aggregated to 10-year age by race by county categories. To calculate cases and deaths per 100,000 individuals at the county level, we again use population counts by race, 10-year age categories, and county from the 2010 10% Decennial Census. In the CDC data, 2.5% of cases have missing state or county identifiers which we exclude from the analysis. Furthermore, because the Census data suppresses county identifiers for small counties (population <100,000), around 41% of reported COVID-19 cases were excluded (36% of cases during the first wave of the pandemic), yielding a final sample of 27.2 million cases for all regression analyses, of which 1.2 million were during the first six months of the pandemic.

3.2.2 Socio-economic, health-related, and environmental characteristics

We use the 2016-2019 American Community Survey (ACS) data to obtain the percent of the population that is 65 years of age or older, percent unemployed, percent not in labor force, percent living in group quarters or multigenerational households (2+ generations), percent living in a household with 2+ families, percent uninsured, percent college graduates, percent using public transportation to travel to work, and average household income. These statistics were aggregated at the 10-year age categories by race by county level.

Our second main data source focuses on health-related factors, in particular comorbidities associated with more severe COVID-19 disease as documented by the CDC.[7] We use age-race-county-specific prevalence rate of asthma, cancer, cardiovascular disease (heart attack, angina, coronary heart disease, stroke), diabetes, obesity (BMI $\geq$ 30), smoking, and other chronic illnesses leading to immune suppression from the 2010-2012 Behavioral Risk Factor Surveillance System (BRFSS).[8]

[7]For a complete list of these comorbidities, see CDC's Underlying Medical Conditions Associated with Higher Risk for Severe COVID-19 – Information for Healthcare Professionals. We have excluded comorbidities that cannot be identified in the BRFSS data.

[8]Later years of the BRFSS do not consistently report counties.

Land area size from the 2018 Census Gazetteer Files, combined with county-level population data from the 2010 Census, was used to calculate population density for counties. As a measure of county-level pollution, we use the average satellite-measured PM2.5 levels across counties as measured in van Donkelaar *et al.* (2019) to capture pollution levels across counties for 2012-2019.

We use mobile device tracking data from SafeGraph through May 31, 2020 to calculate the median time spent at home during January-May as a proxy measure of stay-at-home order adherence. The SafeGraph data reports the daily discrete distribution of time spent at home at the census block-level for devices whose home has been determined to be in that census block.[9] We aggregate this daily distribution to the median time spent at home in the period prior to state-wide shutdowns and widespread media coverage (in January and February, which we call the "pre-shutdown period") and after (in March-May, "post-shutdown period") within each county using Pareto interpolation. We do not use Safegraph data beyond the early months of the pandemic as social distancing became replaced by masking as the main way of preventing COVID-19 spread in the later part of the pandemic. To our knowledge, comprehensive county-level masking data for the U.S. is unavailable.

As a proxy measure for health care quality and access, we use the average annual county-level age-adjusted Prevention Quality Indicator (PQI) rate per 100,000 individuals for Medicare beneficiaries for 2014-2018, obtained through the Centers for Medicare and Medicaid Services.[10]

[9]SafeGraph is a data company that aggregates anonymized location data from numerous applications in order to provide insights about physical places. To enhance privacy, SafeGraph excludes census block group information if fewer than five devices visited an establishment in a month from a given census block group. The data was generated via a series of GPS pings from anonymous mobile devices. A device's home census block was determined as the common nighttime location of the mobile device over a 6-week period.

[10]The PQI is calculated using inpatient data and captures conditions for which hospitalization could be prevented via appropriate outpatient care and conditions that could be less severe if treated early and appropriately. As a result, higher PQI values correspond to worse health care quality and access.

3.3 Summary statistics

3.3.1 Nationwide cases and deaths

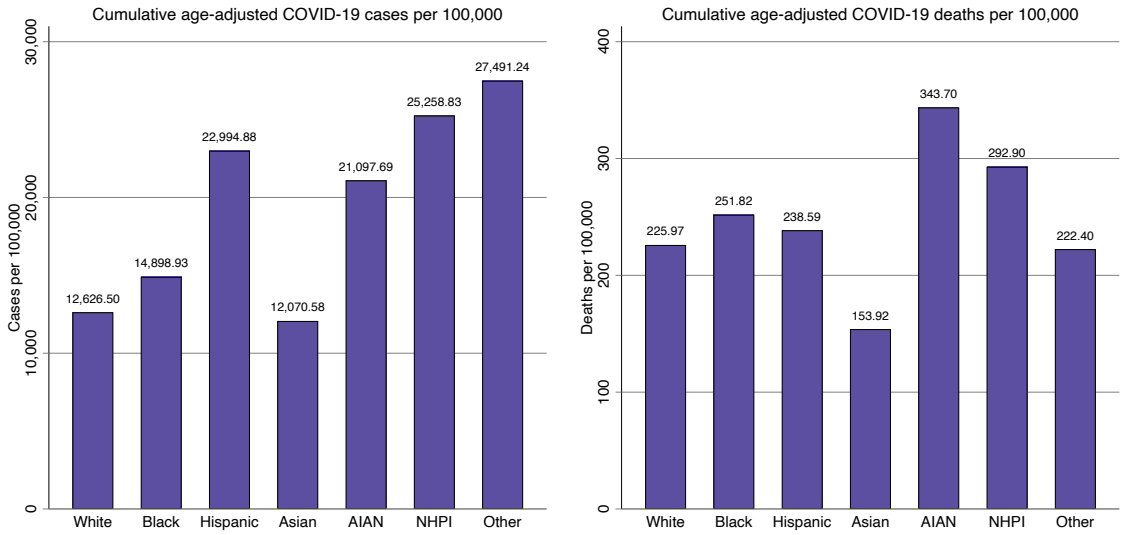
Figure 3.1 reports *age-adjusted* COVID-19 case and mortality rates per 100,000 individuals documented between January 2020 and April 2022 for white, Black, Hispanic, Asian, American Indian/Alaskan Native (AIAN), Native Hawaiian/Pacific Islander (NHPI), and other racial groups.[11] Nationwide, all non-white racial groups except Asian have higher case rates than white individuals. The case rates among AIAN and NHPI individuals are the highest across all racial groups and are 1.7 and 2 times higher than the case rate for white individuals, respectively. The case rate for Hispanic individuals is 1.8 times higher than for white individuals (22,994 vs. 12,626 cases per 100,000) and the case rate for Black individuals (14,898 cases per 100,000) is 1.2 times higher than the case rate for white. White individuals, however, have a similar case rate as Asian individuals.

These racial disparities also persist in the mortality rate, as shown in the second chart in Figure 3.1. The mortality rates among AIAN and NHPI individuals are 1.5 and 1.3 times higher than the mortality rate among white individuals and are the highest mortality rates across all racial groups. The mortality rates among white, Black, and Hispanic individuals are roughly similar. Asian individuals are the least likely to die from COVID-19, with mortality rates approximately 30% lower than that of white individuals.[12]

Figure 3.2 and Figure 3.3 show how these disparities have evolved throughout the duration of the pandemic. Across all waves of the pandemic, nonwhite individuals were more likely to contract COVID-19, in particular Hispanic, AIAN, and NHPI individuals. Death rates

[11] Age adjustment was performed using the 2010 Census population by age and race as the standard population. Since age is the main risk factor for severe COVID-19, and since white individuals tend to be older than non-white individuals, not adjusting for age generally results in smaller disparities in COVID-19 burden across racial groups.

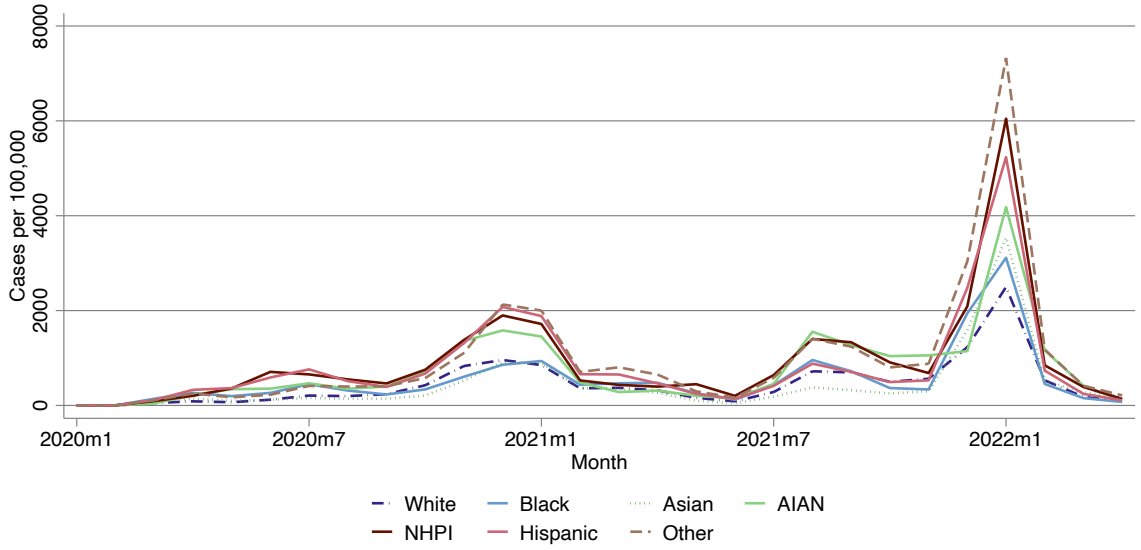
[12] Appendix Figure C.2 shows that similar, if not starker, disparities persist when looking at COVID-19 related hospitalizations and ICU rates per 100,000 reported to the CDC. However, hospitalizations and ICU stays may not be as reliably reported as cases and deaths; see Appendix Figure C.3 and Figure C.4.



Note. *This figure shows cumulative age-adjusted COVID-19 cases and deaths per 100,000 individuals reported up to April 17, 2022 to the National Vital Statistics and the Centers for Disease Control. Population counts by 10-year age categories and race were obtained from the 2010 Decennial Census (10%). Age adjustment was performed using the 2010 Decennial Census population.*

Figure 3.1: *National cumulative COVID-19 case and mortality rates per 100,000 by race*

were also higher among nonwhite individuals – Black individuals were almost twice as likely to die from COVID-19 in the first wave of the pandemic as compared to white. AIAN and NHPI individuals had some of the highest observed mortality rates during the subsequent COVID-19 waves.

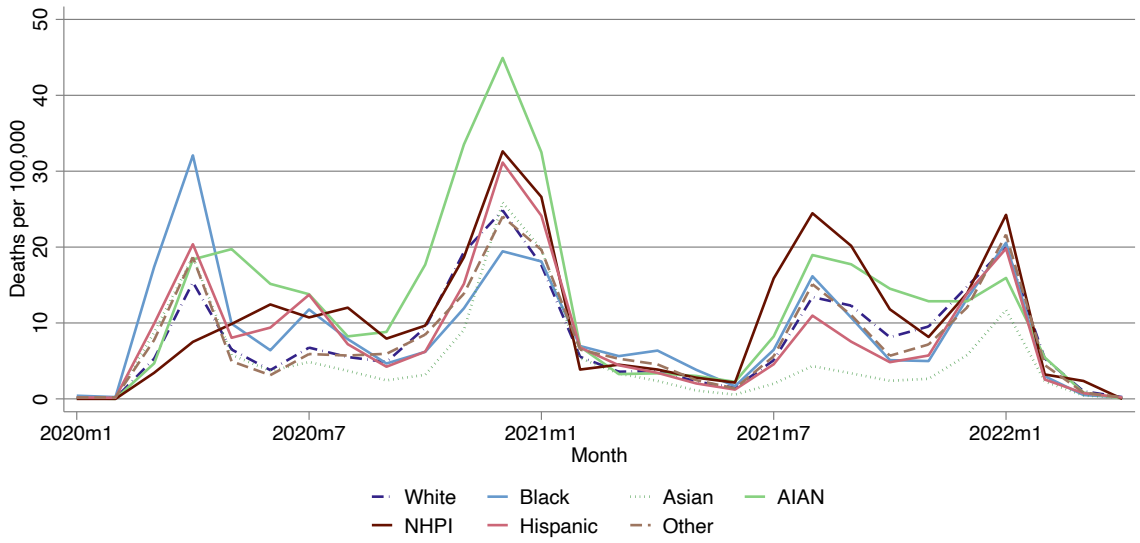


Note. *his figure shows monthly age-adjusted COVID-19 cases per 100,000 individuals reported up to April 17, 2022 to the National Vital Statistics and the Centers for Disease Control. Population counts by 10-year age categories and race were obtained from the 2010 Decennial Census (10%). Age adjustment was performed using the 2010 Decennial Census population.*

Figure 3.2: *National age-adjusted COVID-19 case rates per 100,000 by race over time*

Focusing specifically on the first wave of the pandemic, Figure 3.4 shows that the racial disparities in the beginning of the pandemic are even starker: Black and Hispanic individuals were 2.9 and 3.9 times more likely to contract COVID-19 relative to white individuals.[13] They were also 2.2 and 1.3 times more likely to die from COVID-19 relative to white individuals. AIAN individuals were 2.9 times more likely to contract COVID-19 and 1.6 times more likely to die from COVID-19 relative to white individuals. While NHPI individuals were 3.2 times more likely to have COVID-19 compared to white individuals, they were less likely to die from COVID-19. Interestingly, Asian individuals had a higher COVID-

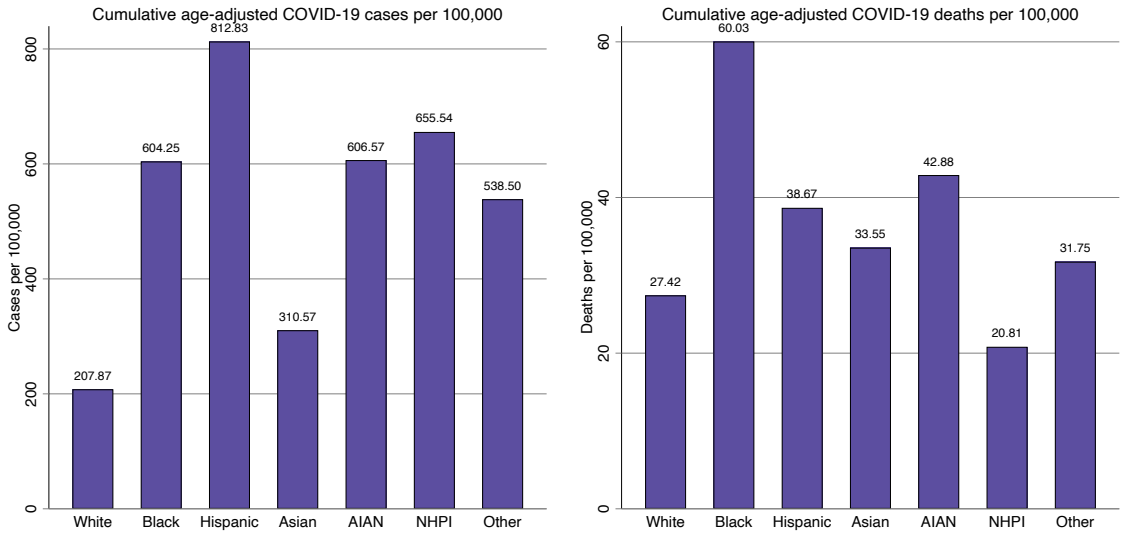
[13]We define the first wave of the pandemic as January 2020 to June 2020.



Note. *This figure shows monthly age-adjusted COVID-19 deaths per 100,000 individuals reported up to April 17, 2022 to the National Vital Statistics and the Centers for Disease Control. Population counts by 10-year age categories and race were obtained from the 2010 Decennial Census (10%). Age adjustment was performed using the 2010 Decennial Census population.*

Figure 3.3: *National age-adjusted COVID-19 mortality rates per 100,000 by race over time*

19 case and mortality rate relative to white individuals in the first wave of the pandemic. Because COVID-19 policy and prevention responses across counties and at the individual level are endogenous to the spread of COVID-19, the remainder of the paper focuses on the relationship between socio-economic, environmental, and health-related factors and COVID-19 cases and deaths during the first wave of the pandemic, with a particular focus on what factors and characteristics were associated with the *initial* racial disparity in COVID-19 burden.



Note. *The map plots cumulative COVID-19 cases and deaths per 100,000 individuals reported as of April 17, 2022 to the National Vital Statistics and the Centers for Disease Control. Population counts by race were obtained from the 2016-2019 American Community Survey.*

Figure 3.4: *National cumulative COVID-19 case and mortality rates per 100,000 by race*

3.3.2 Socio-economic and health-related factors

Table 3.1 presents summary statistics of race-specific socio-economic, health-related, and environmental characteristics across counties in our sample. We choose characteristics that may be correlated with COVID-19 transmission or mortality as documented by the literature or by epidemiological modeling (e.g., (Desmet and Wacziarg, 2020), (Benitez *et al.*, 2020)) and group these characteristics into the following broad categories: age-related (per-

cent elderly), housing-related (percent residing in group quarters, which includes nursing homes, percent residing in households with two or more families, and percent residing in multigenerational households), density-related (population density and percent using public transportation), income-related (unemployment rate, percent not in labor force, uninsured rate, household income, and percent working in service industry), knowledge-related (percent with college degree), environmental factors and social distancing (pollution and percent change in median time spent at home between Jan-Feb and March-May of 2020), and, lastly, health (health care quality, measured by PQI rate per 100,000, comorbidities associated with severe COVID-19 disease). Population density, pollution, the PQI rate, and the median time spent at home do not vary by race because this data was unavailable at the county level.

As is widely known, non-white racial groups are less likely to be elderly, which aligns with our earlier note that adjusting COVID-19 case and mortality rates by age exacerbates the differences in COVID-19 burden by race. Non-white racial groups are more likely to live in denser households, more likely to use public transportation, more likely to be uninsured, more likely to have lower income and less education (except for Asians). Non-white individuals are also more likely to be unemployed and work in the service industry. Comorbidity rates are highest among Black and AIAN individuals, while all other racial groups have relatively similar comorbidity rates. Rates of diabetes, obesity, and smoking are particularly high among Black and AIAN individuals.

These differences in the levels of various characteristics across racial groups may contribute to differences in COVID-19 case and mortality rates by race even if the size of the correlation between these characteristics and COVID-19 burden is the same across racial groups. This motivates the first part of the Oaxaca-Blinder decomposition, which will determine how much these differences in *levels* of various characteristics correlated with COVID-19 burden explain COVID-19 race differentials *holding the correlation between characteristics and case/mortality rates constant*.

Table 3.1: *Summary statistics across counties by race*

	White		Asian		Black		Hispanic		AIAN		NHPI	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Characteristics that vary by race												
Socio-economic												
Age 65+, %	27.3	41.4	24.2	39.8	25.1	40.4	25.2	40.4	18.8	36.3	12.6	31.1
Lives in dense housing, %	14.7	8.0	23.1	17.6	24.0	16.2	22.1	12.9	24.3	28.5	32.6	32.9
In group quarters, %	2.8	4.2	2.8	9.1	7.5	12.8	3.0	6.8	5.7	16.3	4.6	16.3
2+ families in household, %	6.7	6.3	6.7	10.3	7.5	9.2	8.3	8.2	9.8	20.0	12.8	24.4
Multigenerational household (2+ gen), %	6.0	3.1	14.5	14.2	10.4	10.2	12.0	9.9	10.7	20.3	18.5	26.6
Unemployed, %	2.6	2.2	2.2	4.2	4.6	6.9	3.2	4.2	4.1	12.2	2.9	10.5
Not in labor force, %	45.5	30.7	47.7	32.2	49.3	30.6	45.7	30.4	48.7	36.2	36.2	36.8
Works in service industry, %	33.8	21.9	36.6	27.4	32.4	23.5	30.8	22.3	33.5	32.6	37.6	35.9
Average household income, $	107,025	37,811	116,086	48,393	73,052	36,738	78,344	28,997	83,623	57,010	105,556	77,727
Uninsured, %	5.4	5.4	7.2	9.8	8.7	11.0	15.1	14.8	12.2	21.8	11.6	23.6
College graduates, %	27.0	19.0	37.5	28.3	16.2	15.4	14.4	14.7	16.4	24.8	17.3	28.1
Use public transit, %	2.7	6.8	5.0	11.4	7.2	13.1	4.5	10.8	6.5	19.1	6.6	19.4
Health-related												
Any comorbidity, %	55.9	16.9	33.2	36.1	64.4	30.4	55.6	32.8	67.9	39.8	51.7	43.3
Asthma, %	14.2	12.1	8.1	20.7	16.2	22.8	14.5	23.2	18.3	32.2	13.9	30.2
Cancer, %	16.1	16.3	4.5	17.1	6.4	15.2	7.5	18.6	12.8	29.0	5.5	20.0
Cardiovascular disease, %	10.5	12.1	6.0	19.5	11.0	20.2	9.9	21.4	15.2	30.4	5.7	20.4
Diabetes, %	9.8	9.1	11.1	24.5	17.7	24.3	15.0	24.4	16.2	30.7	13.9	30.7
Obese, %	23.2	13.6	8.8	21.6	35.0	29.3	27.5	28.7	31.7	38.5	27.8	39.1
Current smoker	38.8	27.8	34.2	41.2	49.4	37.9	39.7	38.0	50.2	44.1	41.3	44.0
Other chronic disease	12.8	14.9	6.9	20.4	13.4	24.7	13.1	24.8	22.5	39.3	15.6	34.0
Characteristics that do not vary by race												
Population density (log)	6.2	1.3	6.2	1.3	6.2	1.3	6.2	1.3	6.2	1.3	6.3	1.4
Median time spent at home (Mar 2020-May 2020), hrs	12.4	1.8	12.4	1.8	12.4	1.8	12.4	1.8	12.4	1.8	12.8	1.7
Median time spent at home (Jan 2020-Feb 2020), hrs	10.6	1.3	10.6	1.3	10.6	1.2	10.6	1.2	10.6	1.2	10.8	1.2
% change in median time spent at home	16.7	8.8	16.8	8.9	16.7	8.9	16.8	8.8	16.8	8.8	18.2	8.5
Average PM 2.5	7.6	1.2	7.6	1.2	7.6	1.2	7.6	1.2	7.5	1.2	7.5	1.3
Average PQI rate	4,269	1,058	4,250	1,051	4,288	1,057	4,266	1,060	4,232	1,087	3,923	1,120

Note. *Socio-economic characteristics were obtained from the 2016-2019 American Community Survey (ACS). Health-related characteristics come from the 2010-2012 Behavioral Risk Factor Surveillance System (BRFSS). Data on time at home was obtained from SafeGraph as of May 31, 2020. Pollution data comes from van Donkelaar et al. (2019) for 2012-2019. Age-adjusted Prevention Quality Indicator (PQI) data was obtained through Centers for Medicare and Medicaid Services for 2014-2018. Land area size from the 2018 Census Gazetteer Files was combined with county-level population data from the 2010 Census to calculate population density for counties.*

3.4 Relationship between socio-economic and health characteristics and COVID-19 burden by race

Even if racial groups had the same socio-economic, health, and environmental characteristics, we can observe racial disparities in COVID-19 case and mortality rates if the impact of these characteristics on COVID-19 case and mortality rates varies by race. To examine whether this may be the case, Table 3.2 and Table 3.3 present the relationships between COVID-19 case rates and mortality rates, respectively, and race-specific characteristics across the health, socio-economic, and environmental domains. In other words, the results in Table 3.2 and Table 3.3 represent OLS regression coefficients from separately regressing county-level COVID-19 case and mortality rates for white, Asian, Black, Hispanic, AIAN, NHPI individuals on county-level characteristics for the corresponding racial group. Counties with large AIAN and NHPI populations are much smaller in size and thus are frequently excluded from the data as discussed in Section 3.2; the estimates for these racial groups are as a result noisier. Table 3.2 and Table 3.3 use data before June 2020 and include fixed effects for 10-year age categories. Density, pollution, and the percent change in median time spent at home between January-February and March-May are not available separately by race and are thus included as an overall measure for each county. Lastly, because the full set of characteristics discussed in Section 3.3 are likely to be correlated, we minimize the number of included covariates by combining the percent of individuals in group quarters, in households with 2+ families, and in multigenerational households into a single measure of percent of individuals living in dense households. We also include a covariate for the presence of any comorbidity associated with increased COVID-19 risk instead of including all comorbidities separately.

3.4.1 COVID-19 case rates

Table 3.2 indicates that residing in denser households is positively associated with higher case rates among all non-white racial groups (controlling for all other factors), but is par-

ticularly large for Hispanic individuals, even though noisy and marginally significant. Our estimates imply that counties with a 10 percentage point increase in the percent of Hispanic individuals living in dense households are associated with an 11 percent higher average Hispanic COVID-19 case rate. The correlation between household size and case rate is negative for white individuals; however, when breaking down this measure into percent of individuals in group quarters vs. other types of dense households, the number of white individuals living in group quarters such as nursing homes is strongly positively associated with the white COVID-19 case rate at the county-level.

Average household income is generally negatively associated with the case rate across all racial groups, but is statistically significant for white, Hispanic, and NHPI individuals. Education is particularly negatively correlated with the COVID-19 case rate among white and Black individuals – a 10 percentage point increase in the percent of Black individuals with a college degree is associated with a 10% decrease in the average Black COVID-19 case rate (around 5% for white individuals), controlling for other factors. As documented in Alsan *et al.* (2020), knowledge about COVID-19 prevention differs by demographic groups, but our results complement these findings by suggesting education gradients in COVID-19 prevention conditional on race.

Use of public transportation and population density are strongly correlated with case rates across almost all races, indicating that density-related measures are another important factor in the initial spread of COVID-19. Public transportation use is particularly strongly correlated with the Hispanic case rate – an additional 10 percentage point of Hispanic individuals using public transportation to travel to work is associated with a 13% increase in the average Hispanic COVID-19 case rate. The correlation between population density and case rates is among the largest across all characteristics and all racial groups. Pollution is another significant predictor of COVID-19 spread among Asian and Hispanic individuals, but not white, Black, or AIAN individuals. Lastly, the percent change in the time spent at home during March 2020-June 2020 relative to January-February 2020 is positively associ-

Table 3.2: *Regressions of county-level cumulative COVID-19 cases per 100,000 by race on race-specific characteristics*

	(1) White b/se	(2) Asian b/se	(3) Black b/se	(4) Hispanic b/se	(5) AIAN b/se	(6) NHPI b/se
Characteristics that vary by race						
% in dense household	-3.8305***	2.0491	4.0565	14.8991**	2.1278*	6.6221
	(0.9399)	(1.7822)	(6.1003)	(7.0933)	(1.2165)	(6.7170)
Avg. hh income	-0.0017***	-0.0015*	-0.0021*	-0.0069***	-0.0000	-0.0038**
	(0.0002)	(0.0009)	(0.0011)	(0.0024)	(0.0007)	(0.0019)
% using public transit	11.4820***	4.7483	-1.0099	17.0793**	1.9383	-3.4751
	(1.9279)	(4.2248)	(3.1412)	(7.6259)	(1.3152)	(10.8372)
% college graduates	-1.0564**	0.4766	-8.7027**	1.1885	0.4602	-4.6751
	(0.5327)	(2.0646)	(4.2354)	(7.1427)	(1.9072)	(8.0611)
Comorbidities						
% with any comorbidity	-0.2493	-1.4038	2.7560	-1.8243	-0.6629	-14.4640
	(0.3039)	(1.1855)	(2.4915)	(2.2870)	(0.7716)	(11.2994)
Characteristics that do not vary by race						
Avg. % change in time spent at home	4.6471***	15.4275***	24.3035***	16.1030***	2.2959	-40.3280***
	(0.8160)	(5.1799)	(8.7322)	(6.0882)	(3.4384)	(12.7682)
Population density (log)	58.3885***	11.1536	136.2395***	156.9672**	13.8910	185.4459**
	(8.6956)	(48.2267)	(46.7492)	(60.9726)	(24.6221)	(85.3714)
Avg. PQI rate	0.0032	-0.0264	0.0233	0.0062	-0.0220	-0.1353
	(0.0054)	(0.0469)	(0.0506)	(0.0777)	(0.0210)	(0.1268)
Avg. PM 2.5	-9.3628*	127.3362***	-110.4963	131.8469**	-67.0725**	166.7627
	(5.5927)	(43.0527)	(68.0643)	(56.1364)	(27.5767)	(128.6960)
10-year age FE	Yes	Yes	Yes	Yes	Yes	Yes
Missing: housing vars	Yes	Yes	Yes	Yes	Yes	Yes
Missing: employment vars	Yes	Yes	Yes	Yes	Yes	Yes
Missing: transit vars	Yes	Yes	Yes	Yes	Yes	Yes
Missing: education vars	Yes	Yes	Yes	Yes	Yes	Yes
Missing: comorbidity vars	Yes	Yes	Yes	Yes	Yes	Yes
Missing: other vars	Yes	Yes	Yes	Yes	Yes	Yes
N	3,816	3,639	3,705	3,747	3,011	1,368
R-squared	0.3326	0.0246	0.0615	0.0770	0.0262	0.0506
Dependent var. mean	219	511	843	1,331	266	770

*Standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1*

Note. *Cases represent the cumulative COVID-19 cases per 100,000 individuals reported up to June 1, 2020 to the National Vital Statistics and the Centers for Disease Control. Population counts by 10-year age categories and race were obtained from the 2010 Decennial Census (10%). Socio-economic characteristics were obtained from the 2016-2019 American Community Survey (ACS). Health-related characteristics come from the 2010-2012 Behavioral Risk Factor Surveillance System (BRFSS). Data on time at home was obtained from SafeGraph as of May 31, 2020. Pollution data comes from van Donkelaar et al. (2019) for 2012-2019. Age-adjusted Prevention Quality Indicator (PQI) data was obtained through Centers for Medicare and Medicaid Services for 2014-2018. Land area size from the 2018 Census Gazetteer Files was combined with county-level population data from the 2010 Census to calculate population density for counties.*

ated with the case rate across almost all racial groups with the exception of NHPI, possibly reflecting endogenous response at the individual level. Health-related factors such as co-morbidities and health care quality are uncorrelated with the COVID-19 case rate across all racial groups.[14]

3.4.2 COVID-19 mortality rates

Table 3.3 shows the results from the same OLS models as for case rates discussed above, but using mortality rates instead. We find that similar factors correlate with COVID-19 mortality rates as for case rates, suggesting that the mortality rate measure might be picking up increased COVID-19 spread. Neither health care quality nor comorbidities are correlated with mortality rates, with the exception of Black individuals, where counties with a 10 percentage point higher share of Black individuals with an underlying comorbidity are associated with a 13% higher COVID-19 mortality rate among Black individuals, although only marginally significant.

To address the fact that the mortality rate may be capturing overall COVID-19 burden rather than severity of disease, we estimate the same models as in Table 3.3 but instead use the mortality rate conditional on COVID-19 infection, i.e., the percent of individuals that die conditional on being infected with COVID-19. These results, shown in Table 3.4, indicate that counties with lower health care quality are particularly positively correlated with higher mortality rates among white individuals conditional on infection. Lower health care quality is also positively associated with higher mortality rates (conditional on infection)

[14]Under the assumption that within counties racial groups interact with each other, characteristics of a given racial group may correlate with COVID-19 case and death rates of other racial groups, even when controlling for the characteristics of the other racial groups. Policy interventions to curb the COVID-19 spread in certain racial groups may magnify in impact if behavior or characteristics of targeted racial groups spills over to other groups. For instance, Table C.1 and Table C.2 show the OLS regression results from regressing the COVID-19 case and death rate for the listed racial group on the characteristics of white individuals, controlling for own-group characteristics. These results suggest that non-white individuals living in counties where white individuals are richer and more educated have lower COVID-19 case rates, even controlling for non-white income and education levels. However, regressions of this sort may capture area factors that we are not controlling for in our regressions that affect all racial groups equally and are correlated with income and education of white individuals.

Table 3.3: *Regressions of county-level cumulative COVID-19 deaths per 100,000 by race on race-specific characteristics*

	(1) White b/se	(2) Asian b/se	(3) Black b/se	(4) Hispanic b/se	(5) AIAN b/se	(6) NHPI b/se
Characteristics that vary by race						
% in dense household	-0.2351	0.8678*	2.7108	1.6191	0.9410**	0.0389
	(0.3811)	(0.4628)	(3.7637)	(1.1393)	(0.3828)	(0.1606)
Avg. hh income	-0.0007***	0.0001	-0.0005	-0.0013***	0.0001	-0.0002*
	(0.0001)	(0.0002)	(0.0005)	(0.0004)	(0.0002)	(0.0001)
% using public transit	2.8804***	3.2826**	-1.3383	4.5448**	-0.4649*	-0.6348**
	(1.0372)	(1.6679)	(1.4492)	(2.0932)	(0.2560)	(0.2692)
% college graduates	-0.7157***	0.5470	-3.5002	1.9622	0.2342	-0.4085**
	(0.2342)	(0.4099)	(2.7125)	(2.0850)	(0.4228)	(0.1779)
Comorbidities						
% with any comorbidity	-0.0870	0.0294	2.1284*	0.3094	-0.2206	-0.2785
	(0.1414)	(0.2637)	(1.1857)	(0.4878)	(0.2669)	(0.5535)
Characteristics that do not vary by race						
Avg. % change in time spent at home	1.7376***	2.3957***	9.9982*	5.6576***	-0.1542	-1.0421
	(0.3763)	(0.8256)	(5.3706)	(1.1921)	(0.7354)	(0.7296)
Population density (log)	28.4934***	25.7133***	82.2192***	43.3244***	3.3571	10.0066
	(4.4349)	(6.9361)	(23.6833)	(12.1070)	(5.4286)	(8.1848)
Avg. PQI rate	0.0001	0.0060	0.0224	0.0100	0.0029	-0.0035
	(0.0025)	(0.0070)	(0.0261)	(0.0095)	(0.0054)	(0.0063)
Avg. PM 2.5	-9.1581***	-5.9512	-54.1331	-13.9766	-9.6941*	-1.8203
	(2.2968)	(8.0933)	(34.2647)	(11.1905)	(5.0727)	(13.7443)
10-year age FE	Yes	Yes	Yes	Yes	Yes	Yes
Missing: housing vars	Yes	Yes	Yes	Yes	Yes	Yes
Missing: employment vars	Yes	Yes	Yes	Yes	Yes	Yes
Missing: transit vars	Yes	Yes	Yes	Yes	Yes	Yes
Missing: education vars	Yes	Yes	Yes	Yes	Yes	Yes
Missing: comorbidity vars	Yes	Yes	Yes	Yes	Yes	Yes
Missing: other vars	Yes	Yes	Yes	Yes	Yes	Yes
N	3,816	3,639	3,705	3,747	3,011	1,368
R-squared	0.3121	0.1521	0.0463	0.1440	0.0319	0.0477
Dependent var. mean	43	64	160	129	27	30

*Standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1*

Note. *Deaths represent the cumulative COVID-19 deaths per 100,000 individuals reported up to June 1, 2020 to the National Vital Statistics and the Centers for Disease Control. Population counts by 10-year age categories and race were obtained from the 2010 Decennial Census (10%). Socio-economic characteristics were obtained from the 2016-2019 American Community Survey (ACS). Health-related characteristics come from the 2010-2012 Behavioral Risk Factor Surveillance System (BRFSS). Data on time at home was obtained from SafeGraph as of May 31, 2020. Pollution data comes from van Donkelaar et al. (2019) for 2012-2019. Age-adjusted Prevention Quality Indicator (PQI) data was obtained through Centers for Medicare and Medicaid Services for 2014-2018. Land area size from the 2018 Census Gazetteer Files was combined with county-level population data from the 2010 Census to calculate population density for counties.*

for all other racial groups, but these results are not statistically significant. Importantly, counties where Black individuals had a higher rate of comorbidities had a higher conditional Black mortality rate. An additional 10 percentage points of Black individuals with an underlying comorbidity are associated with a 3.75% increase in the average percent of Black individuals dying from COVID-19 if infected.

3.5 Oaxaca-Blinder decomposition

Given the differences in county-level characteristics and coefficients on those characteristics by race, the Oaxaca-Blinder decomposition introduced in Oaxaca (1973) and Blinder (1973) allows for a systematic examination of the extent to which observed differences in COVID-19 case rates between white and non-white racial groups stem from the differences in levels of observed characteristics by race discussed in Section 3.3 vs. the differences in the strength of the correlation between these characteristics and COVID-19 burden across racial groups demonstrated in Section 3.4. In Table 3.5 and Table 3.6, we decompose the average differences in COVID-19 case and mortality rates between Black and white and Hispanic and white individuals into three components: one due to differences in average characteristics (or levels of characteristics documented in Section 3.3) by race ("differences in endowments"), differences due to the "impact" of characteristics on case rates across racial groups ("differences in coefficients"), as estimated in Section 3.4, and differences due to the interaction between the levels and coefficients for each characteristic ("differences in interactions"). We exclude AIAN and NHPI individuals since sample sizes for these racial groups are small.

In Table 3.5 and Table 3.6, the columns labeled "endowments" show the change in the average case or death rate differential by race if levels of the non-white racial group were equal to the levels for whites, either of individual socio-economic, health-related, or environmental factors or across all factors. The columns labeled "coefficients" show the change in the case or mortality rate differential between non-white and white if the correlations

Table 3.4: *Regressions of county-level cumulative COVID-19 deaths conditional on infection by race on race-specific characteristics*

	(1) White b/se	(2) Asian b/se	(3) Black b/se	(4) Hispanic b/se	(5) AIAN b/se	(6) NHPI b/se
Characteristics that vary by race						
% in dense household	0.0312	0.0069	0.0312	-0.0199	0.0929	0.0052
	(0.0243)	(0.0366)	(0.0319)	(0.0327)	(0.0583)	(0.0436)
Avg. hh income	-0.0000	0.0000	-0.0000	-0.0000	0.0000	-0.0000*
	(0.0000)	(0.0000)	(0.0000)	(0.0000)	(0.0000)	(0.0000)
% using public transit	0.0766**	0.0985**	0.0233	0.0767***	-0.0688	-0.0160
	(0.0317)	(0.0403)	(0.0185)	(0.0285)	(0.0471)	(0.0457)
% college graduates	-0.0082	0.0088	-0.0049	-0.0500	-0.0425	0.0087
	(0.0146)	(0.0263)	(0.0408)	(0.0355)	(0.0478)	(0.0349)
Comorbidities						
% with any comorbidity	0.0126	0.0035	0.0267***	-0.0035	-0.0068	0.0076
	(0.0086)	(0.0127)	(0.0094)	(0.0096)	(0.0262)	(0.0211)
Characteristics that do not vary by race						
Avg. % change in time spent at home	0.0488**	0.0214	0.0717**	0.0989***	-0.0437	-0.0924
	(0.0200)	(0.0483)	(0.0309)	(0.0278)	(0.0996)	(0.1093)
Population density (log)	0.8440***	0.3248	0.7630***	0.2941	0.3514	-1.0760
	(0.2126)	(0.3650)	(0.2410)	(0.2090)	(0.6390)	(1.0591)
Avg. PQI rate	0.0004***	0.0006	-0.0000	0.0003	0.0005	0.0004
	(0.0002)	(0.0004)	(0.0002)	(0.0002)	(0.0008)	(0.0009)
Avg. PM 2.5	-0.2644	-0.8888***	-0.5386**	-0.5449***	-0.0738	1.4381
	(0.1646)	(0.3368)	(0.2632)	(0.1982)	(0.7357)	(0.8817)
10-year age FE	Yes	Yes	Yes	Yes	Yes	Yes
Missing: housing vars	Yes	Yes	Yes	Yes	Yes	Yes
Missing: employment vars	Yes	Yes	Yes	Yes	Yes	Yes
Missing: transit vars	Yes	Yes	Yes	Yes	Yes	Yes
Missing: education vars	Yes	Yes	Yes	Yes	Yes	Yes
Missing: comorbidity vars	Yes	Yes	Yes	Yes	Yes	Yes
Missing: other vars	Yes	Yes	Yes	Yes	Yes	Yes
N	3,202	1,766	2,619	2,823	543	291
R-squared	0.6463	0.4918	0.4720	0.4387	0.2530	0.3706
Dependent var. mean	8	8	8	6	7	4

*Standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1*

Note. *Conditional deaths represent the cumulative COVID-19 deaths divided by the cumulative COVID-19 cases (multiplied by 100) reported up to June 1, 2020 to the National Vital Statistics and the Centers for Disease Control. Population counts by 10-year age categories and race were obtained from the 2010 Decennial Census (10%). Socio-economic characteristics were obtained from the 2016-2019 American Community Survey (ACS). Health-related characteristics come from the 2010-2012 Behavioral Risk Factor Surveillance System (BRFSS). Data on time at home was obtained from SafeGraph as of May 31, 2020. Pollution data comes from van Donkelaar et al. (2019) for 2012-2019. Age-adjusted Prevention Quality Indicator (PQI) data was obtained through Centers for Medicare and Medicaid Services for 2014-2018. Land area size from the 2018 Census Gazetteer Files was combined with county-level population data from the 2010 Census to calculate population density for counties.*

between individual or aggregate factors for the non-white racial group were equal to those of whites. And lastly, the columns labeled "interactions" show the change in the average case or mortality rate differential by race from changing both the levels and the coefficients. Differences in the constants comprise the "unexplained" component of the decomposition – the part that is unrelated to any of the factors included in the models. Note that all models also include 10-year age category fixed effects and controls for missing values for included covariates, which may also contribute to explaining part of the observed case rate differentials by race but are not of interest for this analysis. In a similar vein, we do not discuss the impact of the interactions in explaining the case rate differential in as much detail.

Table 3.5: *Oaxaca-Blinder decomposition: COVID-19 cases per 100,000*

	Black vs. white			**Hispanic vs. white**		
	Endowments	Coefficients	Interactions	Endowments	Coefficients	Interactions
Case rate difference		624			1112	
Total explained by included covariates	-152	-208	93	-259	-1645	219
% in dense household	-29	-147	56	-85	-321	106
Avg. hh income	-58	27	13	-156	318	118
% using public transit	3	57	-33	-17	-16	6
% college graduates	-74	96	65	12	-25	-22
% with any comorbidity	7	-123	-8	-14	57	12
Avg. % change in time spent at home	0	-291	0	-1	-170	0
Population density (log)	-4	-427	2	0	-538	0
Avg. PQI rate	0	-76	0	0	-11	0
Avg. PM 2.5	2	675	-2	2	-938	-2
Total unexplained (constant)		-609			1521	

Note. *Cases represent the cumulative COVID-19 cases per 100,000 individuals reported up to June 1, 2020 to the National Vital Statistics and the Centers for Disease Control. Population counts by 10-year age categories and race were obtained from the 2010 Decennial Census (10%). Socio-economic characteristics were obtained from the 2016-2019 American Community Survey (ACS). Health-related characteristics come from the 2010-2012 Behavioral Risk Factor Surveillance System (BRFSS). Data on time at home was obtained from SafeGraph as of May 31, 2020. Pollution data comes from van Donkelaar et al. (2019) for 2012-2019. Age-adjusted Prevention Quality Indicator (PQI) data was obtained through Centers for Medicare and Medicaid Services for 2014-2018. Land area size from the 2018 Census Gazetteer Files was combined with county-level population data from the 2010 Census to calculate population density for counties.*

3.5.1 COVID-19 case rates

As shown Table 3.5, if Black individuals had the same observed characteristics as white individuals, but different coefficients, we would expect the Black-white case rate difference to *decrease* by 152 cases per 100,000 relative to the observed case rate difference of 624

Table 3.6: *Oaxaca-Blinder decomposition: COVID-19 deaths per 100,000*

	Black vs. white			Hispanic vs. white		
	Endowments	Coefficients	Interactions	Endowments	Coefficients	Interactions
Mortality rate difference		117			86	
Total explained by included covariates	-55	-303	23	-22	-188	-3
% in dense household	-19	-55	21	-9	-32	11
Avg. hh income	-14	-11	-5	-30	37	14
% using public transit	4	19	-11	-4	-5	2
% college graduates	-30	35	24	19	-30	-26
% with any comorbidity	6	-91	-6	2	-14	-3
Avg. % change in time spent at home	0	-122	0	0	-58	0
Population density (log)	-2	-294	1	0	-81	0
Avg. PQI rate	0	-85	0	0	-38	0
Avg. PM 2.5	1	300	-1	0	32	0
Total unexplained (constant)		335			185	

Note. *Deaths represent the cumulative COVID-19 deaths per 100,000 individuals reported up to June 1, 2020 to the National Vital Statistics and the Centers for Disease Control. Population counts by 10-year age categories and race were obtained from the 2010 Decennial Census (10%). Socio-economic characteristics were obtained from the 2016-2019 American Community Survey (ACS). Health-related characteristics come from the 2010-2012 Behavioral Risk Factor Surveillance System (BRFSS). Data on time at home was obtained from SafeGraph as of May 31, 2020. Pollution data comes from van Donkelaar et al. (2019) for 2012-2019. Age-adjusted Prevention Quality Indicator (PQI) data was obtained through Centers for Medicare and Medicaid Services for 2014-2018. Land area size from the 2018 Census Gazetteer Files was combined with county-level population data from the 2010 Census to calculate population density for counties.*

additional cases per 100,000 among Black individuals, which represents a 25% decrease relative to the average case rate difference between Black and white individuals reported at the top of Table 3.5. This increase largely stems from equalizing the education and income levels (and to some extent household density) between Black and white individuals, which are particularly strongly correlated with case rates among Black individuals.

If, however, observed characteristics between Black and white individuals remained at levels reported in Section 3.3, but instead Black individuals had the same magnitude of correlations between characteristics and case rates as white individuals, the difference in case rates between Black and white individuals would fall by 208 cases per 100,000, which is a 33% reduction in the case rate differential by race. These reductions largely stem from the fact that population density, social distancing, comorbidity rates, and household density are more strongly correlated with case rates for Black individuals than white individuals. Because the correlation between many factors and the case rate for white individuals are

higher than those for Black individuals, equalizing the coefficients across the two racial groups would be associated with an approximately fivefold increase in the Black-white case rate difference, as indicated by the difference in coefficients.

The second panel of Table 3.5 shows that if Hispanic individuals had the same levels of socio-economic, environmental, and health-related characteristics as white individuals documented in Section 3.3, but holding the race-specific correlations between these factors and case rates constant, the difference in the COVID-19 case rate between Hispanic and white individuals would fall by 259 cases per 100,000, which is a 23% reduction in the case rate differential of 1,112 cases per 100,000. This reduction stems from equalizing the household density and income levels of white and Hispanic individuals, both of which are highly correlated with case rates among Hispanic individuals. On the other hand, if we allow for Hispanic and white individuals to have different endowments of the characteristics considered in this decomposition, but instead assume that Hispanic individuals had the same correlations between these factors and case rates as white individuals, the case rate differential between Hispanic and white individuals would fall by 1,645 cases per 100,000, leading to higher case rates among white individuals. As for Black individuals, this is largely due to the stronger correlation between population density, social distancing, pollution, and household density for Hispanic individuals relative to white individuals.

3.5.2 COVID-19 mortality rates

Table 3.6 performs the Oaxaca-Blinder decomposition for the COVID-19 mortality rate instead of the case rate. If Black individuals had the same levels of socio-economic, health-related, and environmental characteristics as white individuals, the average mortality rate difference between Black and white individuals would fall by 55 deaths per 100,000, representing a 47% decrease in the average difference in mortality rates between Black and white individuals. Similar to cases, this is achieved by equalizing education, income, and household density between Black and white individuals, as well as comorbidity rates, but to a smaller extent. The second panel of Table 3.6 shows that if Hispanic individuals had

the same endowments as white individuals reported in Section 3.3, the average mortality rate difference between Hispanic and white individuals would decrease by 22 deaths per 100,000 (a 26% decrease relative to the average mortality rate differential).

On the other hand, Table 3.6 shows that if Black and Hispanic individuals had the same correlations between characteristics and mortality rates as white individuals, but holding the levels of these characteristics constant, then the mortality rate differential would fall by twice as much as the observed mortality rate differential, leading to higher mortality rates among whites relative to Black and Hispanic individuals. As for cases, this is largely driven by the stronger correlation between population density, social distancing, and household density and mortality rates for non-white individuals relative to white individuals.

3.6 Conclusion

At the onset of the COVID-19 pandemic, non-white individuals were 3-4 times more likely to contract COVID-19 and 1.5-2 times more likely to die from COVID-19. While our findings suggest that population density, household income, and pollution are important factors for the spread of COVID-19 across all racial groups, we also find that Hispanic case rates are particularly high in counties where Hispanic individuals were more likely to commute to work using public transportation and more likely to live in denser or multifamily households. Furthermore, counties where Black individuals are more educated also have a lower Black case rate, and this correlation is much weaker for all other racial groups. We find little evidence that health-related factors are correlated with the initial COVID-19 case and mortality rate disparities; the notable exception is the fact that the rate of underlying comorbidities among Black individuals is highly correlated with the Black mortality rate from COVID-19. If Black and Hispanic individuals had the same socio-economic characteristics as white individuals, observed case and mortality rate differentials by race would fall by 25-35%.

An important caveat of this paper is that our findings are not causal and use data aggre-

gated at the county level. Additionally, we do not account for endogenous individual and county- or state-level response to the COVID-19 pandemic. However, our findings suggest that even though we find little evidence that health-related factors were associated with an increased COVID-19 spread among non-white individuals, direct interventions targeting the higher rates of diabetes, obesity, and smoking among Black individuals, which we show are correlated with more severe COVID-19 illness and mortality in Black individuals conditional on infection, may decrease susceptibility to infectious disease in future generations. Importantly, our results imply that policies beyond the health care and public health domains may play a huge role in the disparate impact of pandemic spread by race. For instance, housing policies targeted to Hispanic individuals that decrease the rate of multi-family or multi-generational households (e.g., by increasing housing affordability) may also be associated with lower risk of disease spread among this racial group. Similarly, educational and informational policies on infectious disease prevention might play a much larger role for improving and maintaining public health for Black individuals than for any other racial group.

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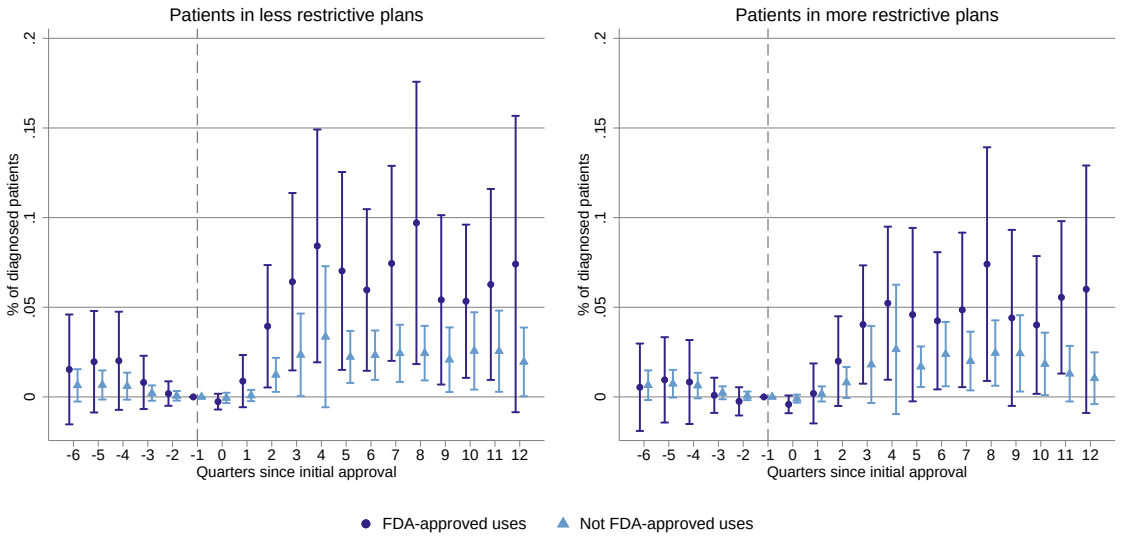
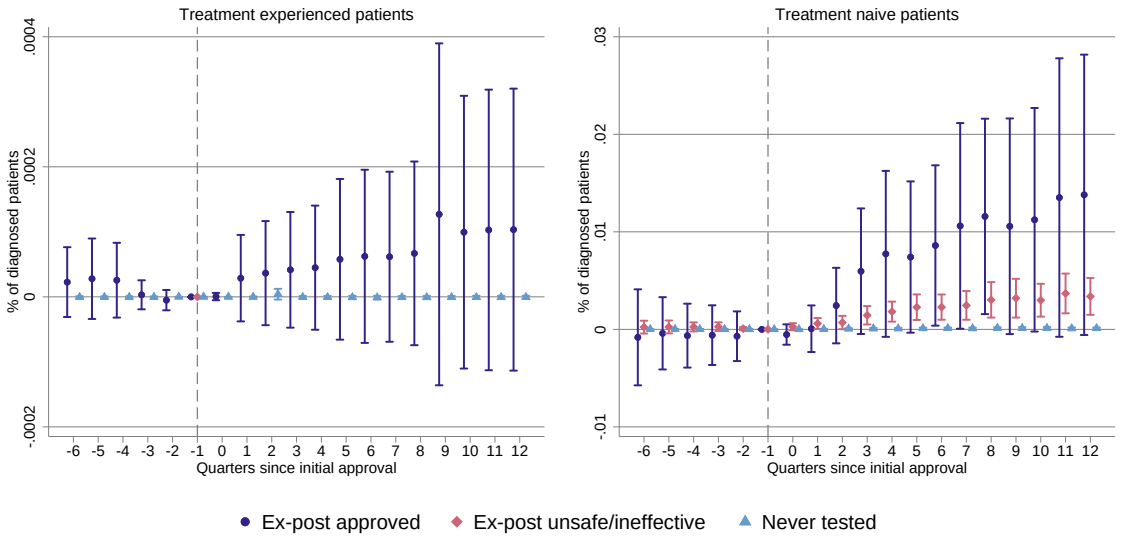
Appendix A

Appendix to Chapter 1



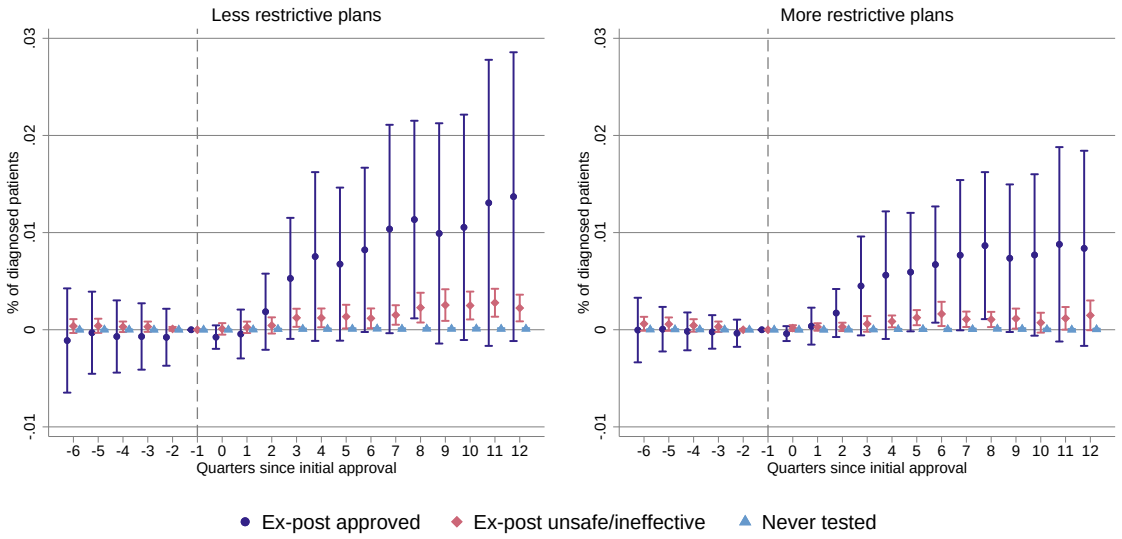
Note. *This figure plots the rates of FDA approved and unapproved uses of new drugs entering the pharmaceutical drug market during 1998-2013 by patient plan enrollment. Less restrictive plans include all Preferred Provider Organization (PPO) plans and more restrictive plans include all Health Maintenance Organization (HMO) plans. Drug claims were measured among pediatric patients (younger than 18 years of age) diagnosed with the FDA-approved diseases at initial market entry in the 1998-2013 MarketScan insurance claims data base. Data on approved indications and ages comes from MicroMedex database and Drugs@FDA. For a definition of FDA-approved vs. unapproved uses, see section Section 1.4.5. Models include drug-class-disease fixed effects, calendar time fixed effects, patient age by sex fixed effects, plan fixed effects at the time of drug claim, and fixed effects for whether the patient had an emergency room or hospitalization claim during the 12 months prior to receiving the drug.*

Figure A.1: *Drug adoption at initial market entry by patient plan enrollment*



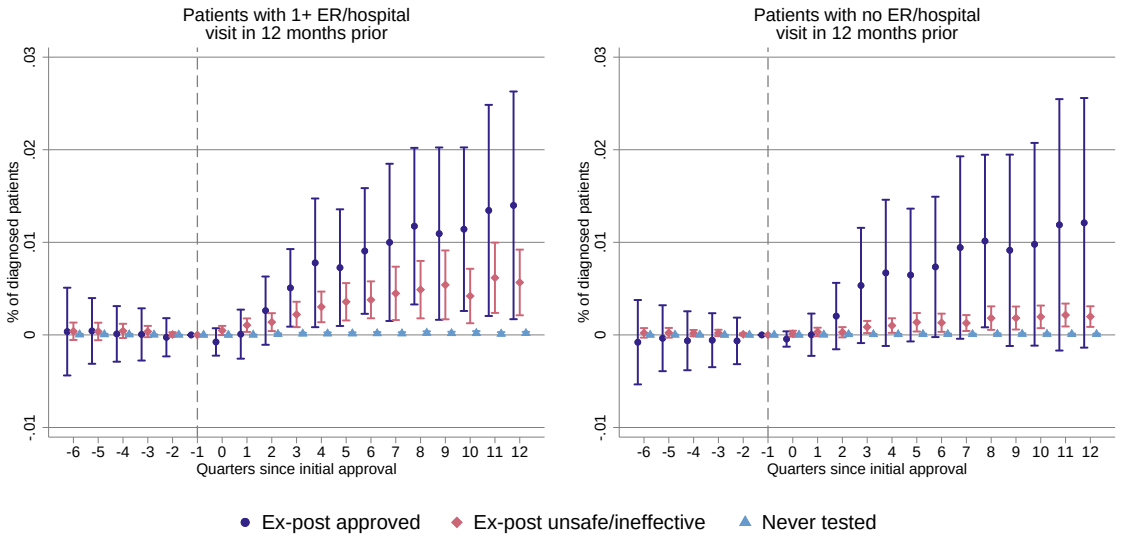
Note. *This figure plots the off-label rates of new drugs entering the pharmaceutical drug market during 1998-2013 by ex-post FDA certification or decertification events and by patient treatment status. Treatment experienced patients were defined as those with at least one drug claim for a drug in the same therapeutic drug class as the newly approved drug prior to receiving the newly approved drug. Drug claims were measured among pediatric patients (younger than 18 years of age) diagnosed with the FDA-approved diseases at initial market entry in the 1998-2013 MarketScan insurance claims data base. Data on approved indications and ages comes from MicroMedex database and Drugs@FDA. For a definition of FDA-approved vs. unapproved uses, see section Section 1.4.5. Models include drug-class-disease fixed effects, calendar time fixed effects, patient age by sex fixed effects, plan fixed effects at the time of drug claim, and fixed effects for whether the patient had an emergency room or hospitalization claim during the 12 months prior to receiving the drug.*

Figure A.2: *Drug adoption at initial market entry by ex-post outcomes and patient treatment status*



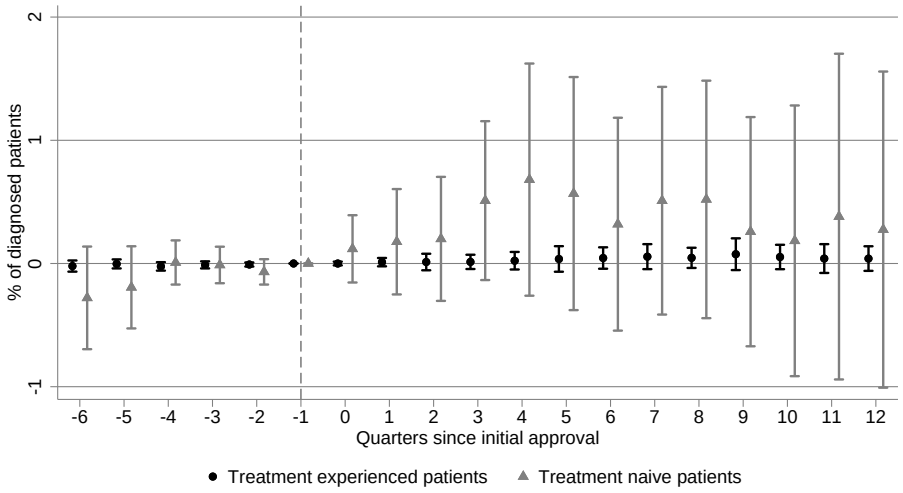
Note. *This figure plots the off-label rates of new drugs entering the pharmaceutical drug market during 1998-2013 by ex-post FDA certification or decertification events and by patient plan enrollment. Less restrictive plans include all Preferred Provider Organization (PPO) plans and more restrictive plans include all Health Maintenance Organization (HMO) plans. Drug claims were measured among pediatric patients (younger than 18 years of age) diagnosed with the FDA-approved diseases at initial market entry in the 1998-2013 MarketScan insurance claims data base. Data on approved indications and ages comes from MicroMedex database and Drugs@FDA. For a definition of FDA-approved vs. unapproved uses, see section Section 1.4.5. Models include drug-class-disease fixed effects, calendar time fixed effects, patient age by sex fixed effects, plan fixed effects at the time of drug claim, and fixed effects for whether the patient had an emergency room or hospitalization claim during the 12 months prior to receiving the drug.*

Figure A.3: *Drug adoption at initial market entry by ex-post outcomes and patient plan enrollment*



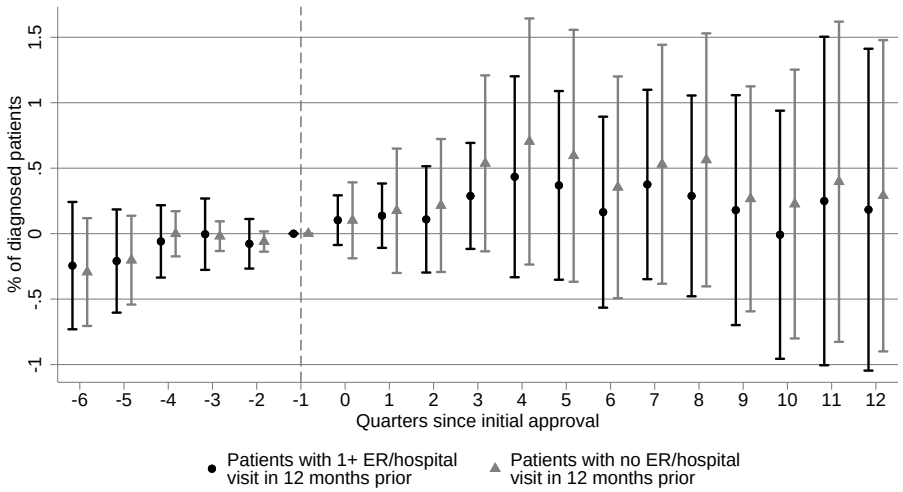
Note. *This figure plots the off-label rates of new drugs entering the pharmaceutical drug market during 1998-2013 by ex-post FDA certification or decertification events and by emergency room (ER) and hospital utilization of the patient in the 12 months prior to the drug claim. Drug claims were measured among pediatric patients (younger than 18 years of age) diagnosed with the FDA-approved diseases at initial market entry in the 1998-2013 MarketScan insurance claims data base. Data on approved indications and ages comes from MicroMedex database and Drugs@FDA. For a definition of FDA-approved vs. unapproved uses, see section Section 1.4.5. Models include drug-class-disease fixed effects, calendar time fixed effects, patient age by sex fixed effects, plan fixed effects at the time of drug claim, and fixed effects for whether the patient had an emergency room or hospitalization claim during the 12 months prior to receiving the drug.*

Figure A.4: *Drug adoption at initial market entry by ex-post outcomes and patient health care utilization*



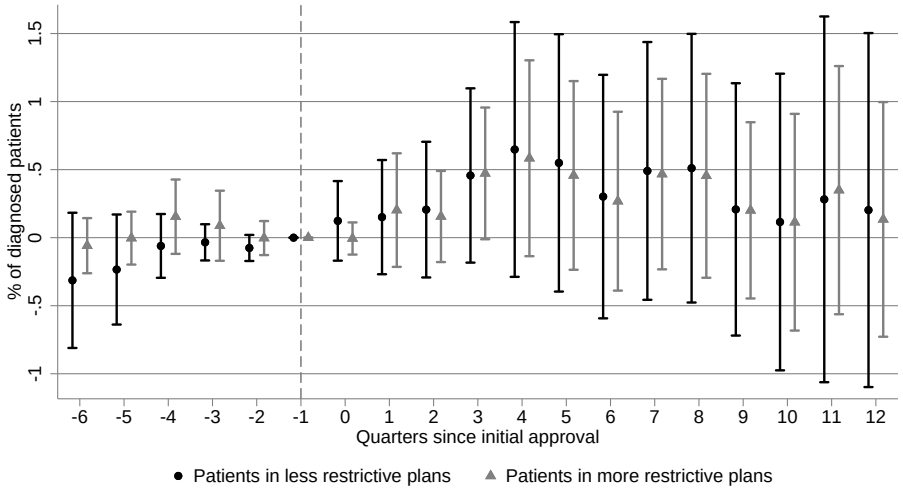
Note. *This figure plots the prescribing rates for drugs approved between 1996-2013 around FDA certification (approval) for some pediatric age. Treatment experienced patients were defined as those with at least one drug claim for a drug in the same therapeutic drug class as the approved drug prior to receiving the drug. Drug claims were measured among pediatric patients (younger than 18 years of age) diagnosed with the FDA-approved diseases at initial market entry in the 1998-2013 MarketScan insurance claims data base. Data on approved indications and ages comes from MicroMedex database and Drugs@FDA. For a definition of FDA-approved vs. unapproved uses, see section Section 1.4.5. Models include drug-class-disease fixed effects, calendar time fixed effects, patient age by sex fixed effects, patient treatment experience fixed effects, plan fixed effects at the time of drug claim, and fixed effects for whether the patient had an emergency room or hospitalization claim during the 12 months prior to receiving the drug.*

Figure A.5: *Drug demand at subsequent FDA certification by patient treatment status*



Note. *This figure plots the prescribing rates for drugs approved between 1996-2013 around FDA certification (approval) for some pediatric age by emergency room (ER) and hospital utilization of the patient in the 12 months prior to the drug claim. Treatment experienced patients were defined as those with at least one drug claim for a drug in the same therapeutic drug class as the approved drug prior to receiving the drug. Drug claims were measured among pediatric patients (younger than 18 years of age) diagnosed with the FDA-approved diseases at initial market entry in the 1998-2013 MarketScan insurance claims data base. Data on approved indications and ages comes from MicroMedex database and Drugs@FDA. For a definition of FDA-approved vs. unapproved uses, see section Section 1.4.5. Models include drug-class-disease fixed effects, calendar time fixed effects, patient age by sex fixed effects, patient treatment experience fixed effects, plan fixed effects at the time of drug claim, and fixed effects for whether the patient had an emergency room or hospitalization claim during the 12 months prior to receiving the drug.*

Figure A.6: *Drug demand at subsequent FDA certification by patient health care utilization*



Note. *This figure plots the prescribing rates for drugs approved between 1996-2013 around FDA certification (approval) for some pediatric age. Less restrictive plans include all Preferred Provider Organization (PPO) plans and more restrictive plans include all Health Maintenance Organization (HMO) plans. Treatment experienced patients were defined as those with at least one drug claim for a drug in the same therapeutic drug class as the approved drug prior to receiving the drug. Drug claims were measured among pediatric patients (younger than 18 years of age) diagnosed with the FDA-approved diseases at initial market entry in the 1998-2013 MarketScan insurance claims data base. Data on approved indications and ages comes from MicroMedex database and Drugs@FDA. For a definition of FDA-approved vs. unapproved uses, see section Section 1.4.5. Models include drug-class-disease fixed effects, calendar time fixed effects, patient age by sex fixed effects, patient treatment experience fixed effects, plan fixed effects at the time of drug claim, and fixed effects for whether the patient had an emergency room or hospitalization claim during the 12 months prior to receiving the drug.*

Figure A.7: *Drug demand at subsequent FDA certification by patient plan enrollment*

Appendix B

Appendix to Chapter 2

B.1 Definitions of mortality due to smoking-related, obesity-related, medically amenable, and external causes

Smoking-related

Malignant Neoplasms: of the Lip, Oral Cavity, Pharynx, Esophagus, Stomach, Pancreas, Larynx, Trachea, Lung, Bronchus, Cervix Uteri, Kidney and Renal Pelvis, Urinary Bladder, and Acute Myeloid Leukemia; Cardiovascular Diseases: Ischemic Heart Disease, Other Heart Disease, Cerebrovascular Disease, Atherosclerosis, Aortic Aneurysm, Other Arterial Disease; Respiratory Diseases: Pneumonia, Influenza, Bronchitis, Emphysema, Chronic Airway Obstruction.

Source: CDC's National Center for Chronic Disease Prevention and Health Promotion (2014).

Obesity-related

Coronary Heart Disease, Other Cardiovascular Disease; Cancers: of the Colon, Breast, Esophagus, Uterus, Ovaries, Kidney, and Pancreas; Diabetes, and Kidney Disease.

Source: Flegal *et al.* (2007).

Medically amenable

Intestinal Infections, Tuberculosis, Other Infections (Diphtheria, Tetanus, Septicaemia, Poliomyelitis), Whooping Cough, Measles; Malignant Neoplasms of: Colon and Rectum, Skin, Breast, Cervix Uteri, Uterus, Testis; Hodgkins Disease, Leukaemia, Diseases of the Thyroid, Diabetes, Epilepsy, Chronic Rheumatic Heart Disease, Hypertensive Disease, Ischaemic Heart Disease (50% of all such deaths), Cerebrovascular Disease, All Respiratory Diseases, Peptic Ulcer, Appendicitis, Abdominal Hernia, Cholelithiasis and Cholecystitis, Nephritis and Nephrosis, Benign Prostatic Hyperplasia, Misadventures to Patients during Surgical and Medical Care, Maternal Death, Congenital Cardiovascular Anomalies, Perinatal Deaths (excl. stillbirths).

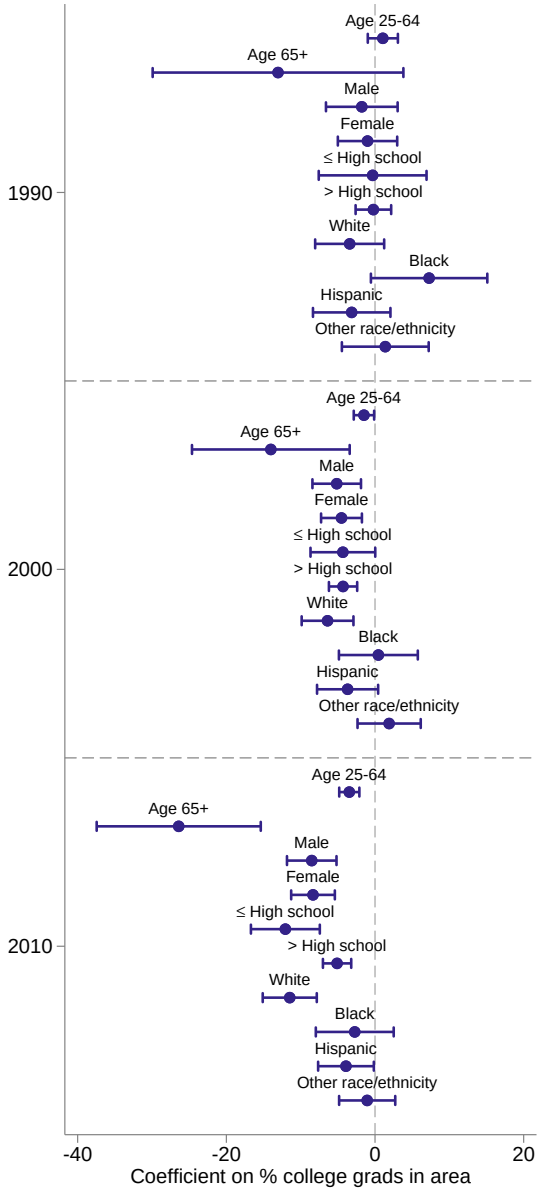
Source: Nolte and McKee (2008).

External causes

Accidents, Intentional Self-Harm, Assault, Events of Undetermined Intent, Legal Intervention, Operations of War and Their Sequelae, Complications of Medical and Surgical Care.

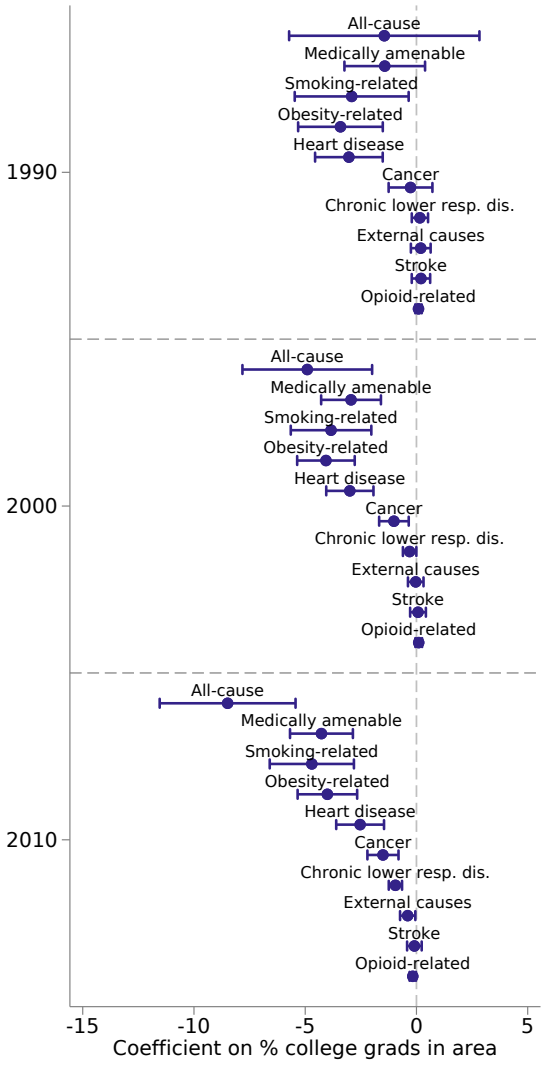
Source: ICD-10-CM Codes V01-Y98.

B.2 Additional tables and figures



Note. *This figure plots the coefficient on area human capital interacted by year and estimated separately for each subgroup. OLS regressions are estimated at the area-year-age-sex-race-education cell level, weighted by cell population, and pooled across 1990, 2000, and 2010. All regressions control for cell-level 5-year age (25-29, 30-34, , 85+) by sex by race (white non-Hispanic, Black non-Hispanic, other non-Hispanic, Hispanic) interactions, individual education, percent of death certificates without education information, and year. We also include controls for area log density and log population, percent Black, percent Hispanic, and industry shares. Confidence intervals are clustered at the area level.*

Figure B.1: *Regression results of all-cause mortality rates per 100,000 on area human capital by subgroup and year*



Note. *This figure plots the coefficient on area human capital interacted by year and estimated separately for each cause of death. OLS regressions are estimated at the area-year-age-sex-race-education cell level, weighted by cell population, and pooled across 1990, 2000, and 2010. All regressions control for cell-level 5-year age (25-29, 30-34, , 85+) by sex by race (white non-Hispanic, Black non-Hispanic, other non-Hispanic, Hispanic) interactions, individual education, percent of death certificates without education information, and year. We also include controls for area log density and log population, percent Black, percent Hispanic, and industry shares. Smoking-related, medically amenable, and obesity-related causes of death include all deaths to causes associated with that risk factor and are not mutually exclusive categories (see Section B.1 for details). Confidence intervals are clustered at the area level.*

Figure B.2: *Regression results of cause-specific mortality rates per 100,000 on area human capital, by year*

Table B.1: *Regression results of all-cause mortality per 100,000 on area human capital and trends in health-related behaviors*

	(1)	(2)	(3)	(4)	(5)	
	Cells with non-missing data on smoking behavior		**Cells with non-missing data on smoking behavior and obesity-related behaviors**			
	1990-2010		2000-2010			
Area characteristics						
% college graduates	0.36	0.41	-3.18**	-1.08	-2.49*	0.28
	(2.19)	(2.03)	(1.41)	(1.4)	(1.46)	(1.41)
% college graduates * year=2000	-4.92***	-2.88*				
	(1.77)	(1.49)				
% college graduates * year=2010	-9.18***	-5.93***	-4.56***	-3.44***	-4.11***	-1.57
	(1.79)	(1.96)	(0.71)	(1.3)	(0.94)	(1.04)
Covariates						
% current smoker		Yes		Yes		Yes
% current smoker by year		Yes		Yes		Yes
% former smoker		Yes		Yes		Yes
% former smoker by year		Yes		Yes		Yes
% overweight, obese, very obese					Yes	Yes
% overweight, obese, v obese by year					Yes	Yes
Cells	391,485	391,485	324,029	324,029	324,029	324,029
Areas	484	484	484	484	484	484
R-squared	0.87	0.87	0.876	0.876	0.876	0.876

Note. **** p<0.01, ** p<0.05, * p<0.1. Standard errors are clustered at the area level. OLS regressions are estimated at the area-year-age-sex-race-education cell level, weighted by cell population, and pooled across 1990, 2000, and 2010. All regressions control for cell-level 5-year age (25-29, 30-34, , 85+) by sex by race (white non-Hispanic, Black non-Hispanic, other non-Hispanic, Hispanic) interactions, individual education, percent of death certificates without education information, and year. We also include controls for area log density and log population, percent Black, percent Hispanic, and industry shares. The percent of individuals that were current or former smokers were calculated using the 1995-1996, 1998-1999 CPS, 1999-2001 BRFSS, and 2009-2011 BRFSS. The percent of individuals that were overweight, obese, or very obese, and those with no physical activity were calculated using the 1999-2001 and 2009-2011 BRFSS.*

Table B.2: *Regression results of mortality per 100,000 by cause of death on area human capital and trends in health-related behaviors*

	(1)	(2)	(3)	(4)	(5)	(6)
	Medically-amenable		**Smoking-related**		**Obesity-related**	
Area characteristics						
% college graduates	-3.29***	-0.89	-3.82***	-1.07	-3.69***	-1.97***
	(0.65)	(0.64)	(0.81)	(0.89)	(0.58)	(0.65)
% current smoker, BRFSS and CPS		3.39***		4.34***		2.41***
		(0.81)		(1.08)		(0.7)
% former smoker, BRFSS and CPS		-2.08***		-1.85**		-1.98**
		(0.7)		(0.92)		(0.78)
% overweight, obese, very obese, BRFSS		2.78***		2.80***		1.86***
		(0.72)		(0.93)		(0.65)
Cells	325,708	325,708	325,708	325,708	325,708	325,708
Areas	484	484	484	484	484	484
R-squared	0.838	0.838	0.844	0.844	0.827	0.827
Dependent var. mean	475	475	631	631	465	465
% change from 10pp increase in % college grads	*-6.9*	*-1.9*	*-6.1*	*-1.7*	*-7.9*	*-4.2*

Note. **** p<0.01, ** p<0.05, * p<0.1. Standard errors are clustered at the area level. Regressions were estimated among areas with non-missing smoking and obesity data. OLS regressions are estimated at the area-year-age-sex-race-education cell level, weighted by cell population, and pooled across 1990, 2000, and 2010. All regressions control for cell-level 5-year age (25-29, 30-34, , 85+) by sex by race (white non-Hispanic, Black non-Hispanic, other non-Hispanic, Hispanic) interactions, individual education, percent of death certificates without education information, and year. We also include controls for area log density and log population, percent Black, percent Hispanic, and industry shares. The percent of individuals that were current or former smokers were calculated using the 1995-1996, 1998-1999 CPS, 1999-2001 BRFSS, and 2009-2011 BRFSS. The percent of individuals that were overweight, obese, or very obese, and those with no physical activity were calculated using the 1999-2001 and 2009-2011 BRFSS.*

B.3 Proofs of propositions

B.3.1 Proof of Proposition 1

The first order condition for unhealthy behavior is $B_x = \frac{b_0+b_2B_k-d_0Q_kh_x\beta V_x-p_k^B}{b_1+b_2}$, where B_x for $x = H, L$ refers to the optimal level of B for the two groups (high human capital and low human capital individuals). Given the exogenous share of high human capital individuals σ_k, the first order condition implies that:

$$
\begin{aligned}
B_k &= \frac{1}{b_1}(b_0 - p_k^B - d_0Q_k\beta(\sigma_k h_H V_H + (1-\sigma_k)h_k V_L) \\
B_H &= \frac{b_0 - p_k^B}{b_1} - \frac{d_0Q_k\beta}{b_1(b_1+b_2)}((b_1+b_2\sigma_k)h_H V_H + b_2(1-\sigma_k)h_k V_L) \\
B_L &= \frac{b_0 - p_k^B}{b_1} - \frac{d_0Q_k\beta}{b_1(b_1+b_2)}(b_2\sigma_k h_H V_H + (b_1+b_2(1-\sigma_k))h_k V_L))
\end{aligned}
$$

All of these terms are decreasing with σ_k, p_k^B, h, Q_k,β, d_0, V_L and V_H, and increasing with b_0. The difference $B_L - B_H = \frac{d_0Q_k\beta}{b_1+b_2}(h_H V_H - h_k V_L) > 0$ and $B_k - B_H = \frac{(1-\sigma_k)}{b_1+b_2}d_0Q_k\beta(h_H V_H - h_k V_L) > 0$ as $V_H h_H > V_L h_k$, and $\frac{dB_k}{d\sigma_k} = -\frac{Q_k\beta d_0}{b_1}(h_H V_H - h_k V_L)$.

B.3.2 Proof of Proposition 2

The only endogenous price is the price of non-traded services, which must clear the market, and this requires $g\prime(p_k^N)^(- 1) = (1-\sigma_k)n_k$ so that the per capita production of non-traded services equals per capita consumption. This implies that $\frac{dp_k^N}{d\sigma_k} = -n_k g''((1-\sigma_k)n_k)$, which is positive.

The spatial equilibrium for the highly educated workers implies that:

$$
\begin{aligned}
W_k^H - p_k^N(1-\sigma_k)n_k + g((1-\sigma_k)n_k) - \frac{(b_0 - p_k^B)^2}{2b_1} \\
- \frac{d_0Q_k\beta^2}{2b_1(b_1+b_2)^2}(((b_1+b_2\sigma_k)h_H V_H + b_2(1-\sigma_k)h_k V_L)^2 \\
- b_1b_2(1-\sigma_k)^2(h_H V_H - h_k V_L)^2) = U_H
\end{aligned}
$$

Using the fact that we have $\frac{d\sigma_k}{dW_k^H}$ equal to 1 divided by

$$-n_k^2(1-\sigma_k)g''((1-\sigma_k)n_k) + \frac{b_2}{b_1(b_1+b_2)}(d_0Q_k\beta)^2(h_HV_H - h_kV_L)(\sigma_k h_H V_H + (1-\sigma_k)h_kV_L)$$

which is positive. As W_k^H does not directly impact health, we know that $\frac{dB_x}{dW_k^H} = \frac{\partial B_x}{\partial \sigma_k}\frac{d\sigma_k}{dW_k^H} < 0$, and $\frac{dB_k}{dW_k^H} = \frac{\partial B_k}{\partial \sigma_k}\frac{d\sigma_k}{dW_k^H} < 0$.

The probability of survival is $Q_k h_x(1-d_0B_x)$ and denoted S_x for each type, and for the area overall equals $Q_k(\sigma_k h_H(1-d_0B_H) + (1-\sigma_k)h_k(1-d_0B_L))$. Consequently, the effect of W_k^H for the survival rate of each group is $\frac{dS_x}{dW_k^H} = -Q_k h_x d_0 \frac{\partial B_x}{\partial \sigma_k}\frac{d\sigma_k}{dW_k^H} > 0$ and overall is $\frac{dS_k}{dW_k^H} = Q_k(h_H(1-d_0B_H) + h_k(1-d_0B_L))\frac{d\sigma_k}{dW_k^H} - Q_k d_0(h_H\sigma_k\frac{dB_H}{dW_k^H} + h_k(1-\sigma_k)\frac{dB_L}{dW_k^H})$ with both terms positive.

We also have that $\frac{d\sigma_k}{dh_k}$ equal $\frac{b_2(1-\sigma_k)}{b_1(b_1+b_2)}(d_0Q_k\beta)^2V_L(\sigma_k h_H V_H + (1-\sigma_k)h_kV_L)$ divided by

$$-n_k^2(1-\sigma_k)g''((1-\sigma_k)n_k) + \frac{b_2}{b_1(b_1+b_2)}(d_0Q_k\beta)^2(h_HV_H - h_LV_L)(\sigma_k h_H V_H + (1-\sigma_k)h_kV_L)$$

which is also positive.

We know that $\frac{dB_H}{dh_k} = \frac{\partial B_H}{\partial \sigma_k}\frac{d\sigma_k}{dh_k} - V_L\frac{b_2}{(b_1+b_2)b_1}(1-\sigma_k)Q_k\beta d_0$, $\frac{dB_L}{dh_k} = \frac{\partial B_L}{\partial \sigma_k}\frac{d\sigma_k}{dh_k} - \frac{(b_1+(1-\sigma_k)b_2)}{(b_1+b_2)b_1}Q_k\beta d_0V_L$ and $\frac{dB_k}{dh_k} = \frac{\partial B_k}{\partial \sigma_k}\frac{d\sigma_k}{dh_k} - \frac{1}{b_1}Q_k\beta d_0V_L(1-\sigma_k)$. As $\frac{d\sigma_k}{dh_k} > 0$, $\frac{\partial B_L}{\partial \sigma_k} < 0$, $\frac{\partial B_H}{\partial \sigma_k} < 0$, and $\frac{\partial B_k}{\partial \sigma_k} < 0$, these terms are all negative.

For the survival rates we have $\frac{dS_H}{dh_k} = -d_0Q_kh_H\frac{\partial B_H}{\partial h_k} > 0$, $\frac{dS_L}{dh_k} = Q_k(1-d_0B_L) - d_0Q_kh_L\frac{\partial B_L}{\partial h_k} > 0$, and $\frac{dS_k}{dh_k} = Q_k(h_H(1-d_0B_H) - h_k(1-d_0B_L))\frac{d\sigma_k}{dh_k} - \sigma_kQ_kh_Hd_0\frac{\partial B_H}{\partial h_k} - (1-\sigma_k)Q_kh_kd_0\frac{\partial B_L}{\partial h_k} + (1-\sigma_k)Q_k(1-d_0B_L)$ and all terms are positive.

Finally, we have that $\frac{d\sigma_k}{dQ_k} > 0$, $\frac{dB_H}{dQ_k} < 0$, $\frac{dB_L}{dQ_k} < 0$, and $\frac{dB_k}{dQ_k} < 0$. For the survival rates we have $\frac{dS_H}{dQ_k} > 0$, $\frac{dS_L}{dQ_k} > 0$, and $\frac{dS_k}{dQ_k} > 0$.

These magnitudes imply that for $x = H, L$ and k

$$\left|\frac{\frac{dB_x}{dQ_k}}{\frac{d\sigma_k}{dQ_k}}\right| > \left|\frac{\frac{dB_x}{dW_k^H}}{\frac{d\sigma_k}{dW_k^H}}\right|, \left|\frac{\frac{dS_x}{dQ_k}}{\frac{d\sigma_k}{dQ_k}}\right| > \left|\frac{\frac{dS_x}{dW_k^H}}{\frac{d\sigma_k}{dW_k^H}}\right|$$
$$\left|\frac{\frac{dB_x}{dh_k}}{\frac{d\sigma_k}{dh_k}}\right| > \left|\frac{\frac{dB_x}{dW_k^H}}{\frac{d\sigma_k}{dW_k^H}}\right|, \left|\frac{\frac{dS_x}{dh_k}}{\frac{d\sigma_k}{dh_k}}\right| > \left|\frac{\frac{dS_x}{dW_k^H}}{\frac{d\sigma_k}{dW_k^H}}\right|$$

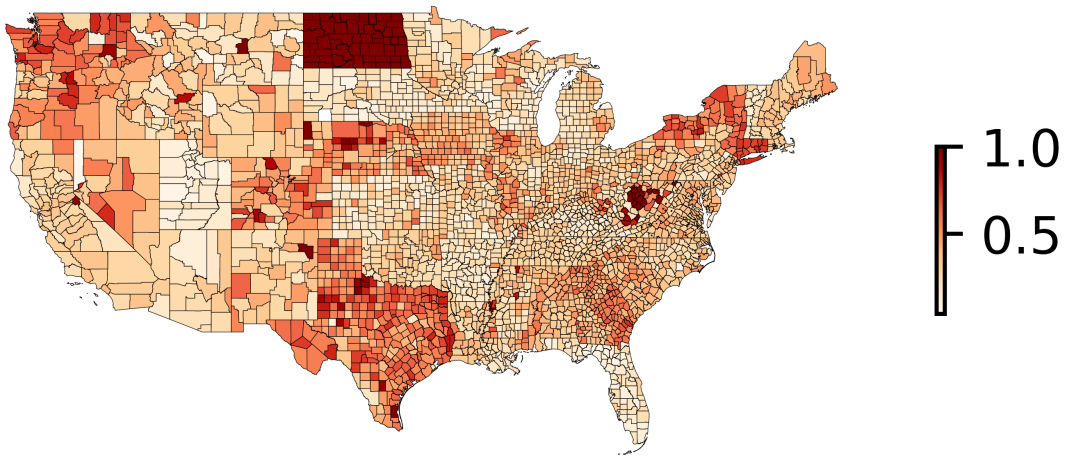
As $1 > \frac{d_0}{b_1}(b_0 - p_k^B - Q_k h_H \beta d_0 V_H) + \frac{d_0 b_2}{(b_1+b_2)b_1}(1-\sigma_k)Q_k \beta d_0 (h_H V_H - h_k V_L)$, it follows that

$$\left|\frac{Q_k}{\sigma_k}\frac{\partial \sigma_k}{\partial Q_k}\right| > \left|\frac{h_k}{\sigma_k}\frac{\partial \sigma_k}{\partial h_k}\right|, \left|\frac{Q_k}{B_H}\frac{\partial B_H}{\partial Q_{!k}}\right| > \left|\frac{h_k}{B_H}\frac{\partial B_H}{\partial h_k}\right|$$
$$\left|\frac{Q_k}{B_L}\frac{\partial B_L}{\partial Q_{!k}}\right| > \left|\frac{h_k}{B_H}\frac{\partial B_H}{\partial h_k}\right|, \left|\frac{Q_k}{B_k}\frac{\partial B_k}{\partial Q_{!k}}\right| > \left|\frac{h_k}{B_k}\frac{\partial B_k}{\partial h_k}\right|$$

From these, it follows that $\left|\frac{Q_k}{S_H}\frac{\partial S_H}{\partial Q_{!k}}\right| > \left|\frac{h_k}{S_H}\frac{\partial S_H}{\partial h_k}\right|$, $\left|\frac{Q_k}{S_L}\frac{\partial S_L}{\partial Q_{!k}}\right| > \left|\frac{h_k}{S_L}\frac{\partial S_L}{\partial h_k}\right|$, and $\left|\frac{Q_k}{S_k}\frac{\partial S_k H}{\partial Q_{!k}}\right| > \left|\frac{h_k}{S_k}\frac{\partial S_k}{\partial h_k}\right|$.

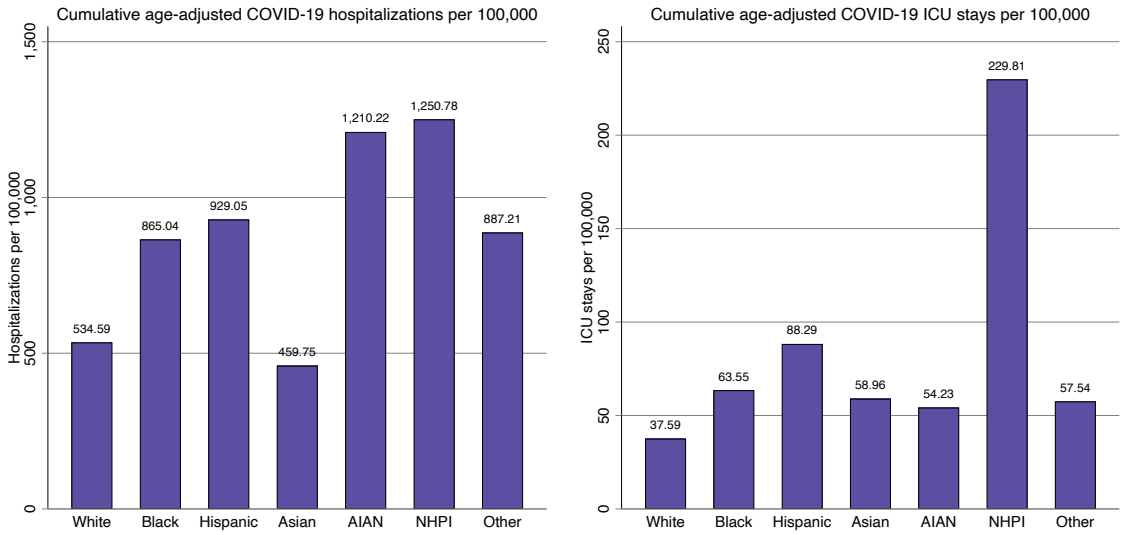
Appendix C

Appendix to Chapter 3



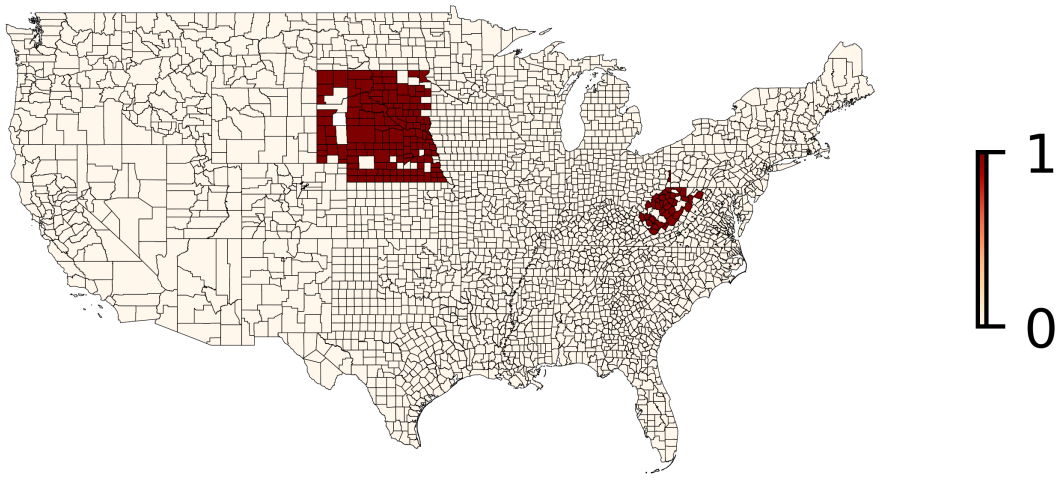
Note. *The map plots the share of cumulative COVID-19 cases with missing race data by county, reported as of April 17, 2022 to the National Vital Statistics and the Centers for Disease Control.*

Figure C.1: *Share of COVID-19 cases with missing race information*



Note. *This figure shows cumulative age-adjusted COVID-19 hospitalizations and ICU stays per 100,000 individuals reported as of April 17, 2022 to the National Vital Statistics and the Centers for Disease Control. Population counts by 10-year age categories and race were obtained from the 2010 Decennial Census (10%). Age adjustment was performed using the 2010 Decennial Census population.*

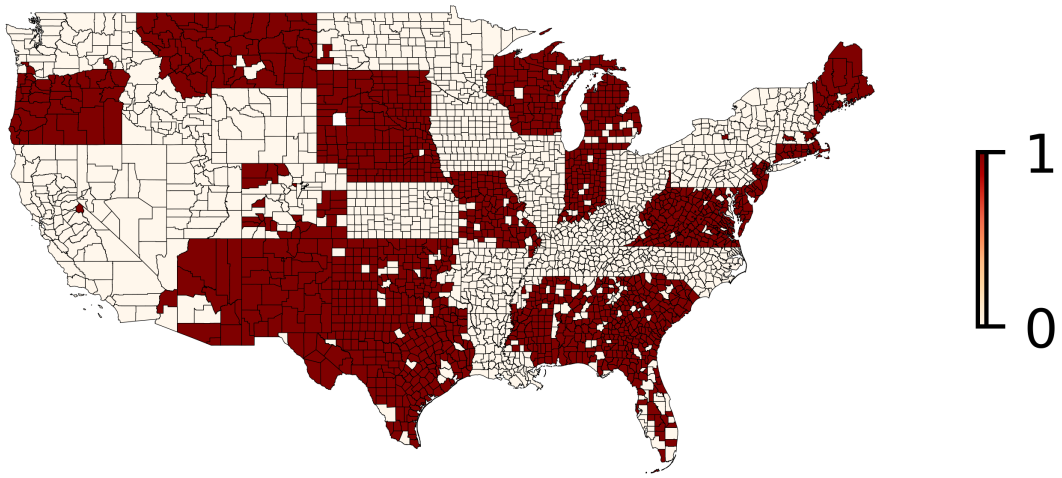
Figure C.2: *National cumulative COVID-19 hospitalization and ICU rates per 100,000 by race*



Note. *The map plots the counties reporting at least one hospitalization associated with COVID-19 as of April 17, 2022 to the National Vital Statistics and the Centers for Disease Control.*

Figure C.3: *Counties with missing hospitalizations data*

Figure C.4: *Counties with missing ICU data*



Note. *The map plots the counties reporting at least one ICU stay associated with COVID-19 as of April 17, 2022 to the National Vital Statistics and the Centers for Disease Control.*

Table C.1: *Regressions of county-level cumulative COVID-19 cases per 100,000 by race on characteristics of other racial groups, controlling for own-race characteristics*

	(1) Asian b/se	(2) Black b/se	(3) Hispanic b/se	(4) AIAN b/se	(5) NHPI b/se
Characteristics that vary by race					
% in dense household for whites	-2.834	-32.867***	-50.112***	-2.061	-55.922**
	(12.304)	(6.629)	(12.151)	(3.941)	(23.479)
Avg. hh income for whites	-0.008***	-0.004*	-0.010***	-0.003**	-0.021***
	(0.002)	(0.002)	(0.003)	(0.001)	(0.006)
% using public transit for whites	-5.497	44.175**	-7.747	6.219	31.395
	(5.719)	(22.336)	(22.419)	(6.271)	(24.974)
% college graduates for whites	-3.025	-4.403	-3.328	6.495**	13.199
	(3.542)	(6.495)	(8.376)	(2.651)	(13.624)
Comorbidities					
% with any comorbidity for whites	-2.420	-1.710	-3.436	-2.189**	3.491
	(2.401)	(1.812)	(2.322)	(0.853)	(7.856)
Characteristics that do not vary by race					
Avg. % change in time spent at home	15.405***	14.647***	15.565**	1.660	-18.257
	(5.345)	(4.805)	(7.442)	(3.814)	(14.697)
Population density (log)	228.041***	134.326**	405.938***	25.387	349.679**
	(30.401)	(54.803)	(56.566)	(37.846)	(140.282)
Avg. PQI rate	-0.045	-0.030	-0.213**	-0.008	-0.438**
	(0.057)	(0.045)	(0.091)	(0.027)	(0.199)
Avg. PM 2.5	46.005	-89.282*	124.279**	-58.940*	181.580
	(29.370)	(52.716)	(56.126)	(32.302)	(156.743)
Own-race characteristics	Yes	Yes	Yes	Yes	Yes
10-year age FE	Yes	Yes	Yes	Yes	Yes
Missing: housing vars	Yes	Yes	Yes	Yes	Yes
Missing: employment vars	Yes	Yes	Yes	Yes	Yes
Missing: transit vars	Yes	Yes	Yes	Yes	Yes
Missing: education vars	Yes	Yes	Yes	Yes	Yes
Missing: comorbidity vars	Yes	Yes	Yes	Yes	Yes
Missing: other vars	Yes	Yes	Yes	Yes	Yes
N	2,496	2,542	2,568	2,006	844
R-squared	0.0337	0.0786	0.0762	0.0247	0.0297
Dependent var. mean	548	1,022	1,646	302	1,149

*Standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1*

Note. *Cases represent the cumulative COVID-19 cases per 100,000 individuals reported up to June 1, 2020 to the National Vital Statistics and the Centers for Disease Control. Population counts by 10-year age categories and race were obtained from the 2010 Decennial Census (10%). Socio-economic characteristics were obtained from the 2016-2019 American Community Survey (ACS). Health-related characteristics come from the 2010-2012 Behavioral Risk Factor Surveillance System (BRFSS). Data on time at home was obtained from SafeGraph as of May 31, 2020. Pollution data comes from van Donkelaar et al. (2019) for 2012-2019. Age-adjusted Prevention Quality Indicator (PQI) data was obtained through Centers for Medicare and Medicaid Services for 2014-2018. Land area size from the 2018 Census Gazetteer Files was combined with county-level population data from the 2010 Census to calculate population density for counties.*

Table C.2: *Regressions of county-level cumulative COVID-19 deaths per 100,000 by race on characteristics of other racial groups, controlling for own-race characteristics*

	(1) Asian b/se	(2) Black b/se	(3) Hispanic b/se	(4) AIAN b/se	(5) NHPI b/se
Characteristics that vary by race					
% in dense household for whites	3.102*	-0.714	0.337	-0.941	-0.447
	(1.736)	(3.007)	(2.051)	(0.771)	(1.264)
Avg. hh income for whites	-0.001***	0.000	-0.001**	-0.001*	-0.000
	(0.000)	(0.001)	(0.001)	(0.000)	(0.000)
% using public transit for whites	-1.719	16.076	7.808	-1.209	-2.392*
	(2.482)	(10.923)	(7.189)	(1.287)	(1.279)
% college graduates for whites	-3.183***	-5.459**	-4.757**	2.514**	0.819
	(0.861)	(2.586)	(2.368)	(0.999)	(1.138)
Comorbidities					
% with any comorbidity for whites	-0.343	1.016	-0.482	-0.459*	-0.045
	(0.373)	(1.095)	(0.492)	(0.267)	(0.383)
Characteristics that do not vary by race					
Avg. % change in time spent at home	3.108***	5.459*	6.173***	0.189	-1.344
	(1.020)	(2.898)	(1.426)	(0.981)	(1.354)
Population density (log)	66.548***	58.746***	91.385***	7.824	19.543
	(9.435)	(22.512)	(15.030)	(10.772)	(12.517)
Avg. PQI rate	-0.011	-0.026	-0.022*	0.009	-0.007
	(0.009)	(0.022)	(0.013)	(0.008)	(0.011)
Avg. PM 2.5	-8.635	-15.588	-14.586	-11.156*	-6.423
	(8.792)	(15.543)	(12.408)	(6.650)	(17.955)
Own-race characteristics	Yes	Yes	Yes	Yes	Yes
10-year age FE	Yes	Yes	Yes	Yes	Yes
Missing: housing vars	Yes	Yes	Yes	Yes	Yes
Missing: employment vars	Yes	Yes	Yes	Yes	Yes
Missing: transit vars	Yes	Yes	Yes	Yes	Yes
Missing: education vars	Yes	Yes	Yes	Yes	Yes
Missing: comorbidity vars	Yes	Yes	Yes	Yes	Yes
Missing: other vars	Yes	Yes	Yes	Yes	Yes
N	2,496	2,542	2,568	2,006	844
R-squared	0.2008	0.0592	0.1976	0.0344	0.0621
Dependent var. mean	84	201	164	36	48

*Standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1*

Note. *Deaths represent the cumulative COVID-19 deaths per 100,000 individuals reported up to June 1, 2020 to the National Vital Statistics and the Centers for Disease Control. Population counts by 10-year age categories and race were obtained from the 2010 Decennial Census (10%). Socio-economic characteristics were obtained from the 2016-2019 American Community Survey (ACS). Health-related characteristics come from the 2010-2012 Behavioral Risk Factor Surveillance System (BRFSS). Data on time at home was obtained from SafeGraph as of May 31, 2020. Pollution data comes from van Donkelaar et al. (2019) for 2012-2019. Age-adjusted Prevention Quality Indicator (PQI) data was obtained through Centers for Medicare and Medicaid Services for 2014-2018. Land area size from the 2018 Census Gazetteer Files was combined with county-level population data from the 2010 Census to calculate population density for counties.*