

# Capacity Strain and Racial Disparities in Hospital Mortality\*

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## Abstract

A growing literature has documented racial disparities in health outcomes. We argue that racial disparities may be magnified when hospitals operate at capacity, when behavioral and structural conditions associated with poor patient outcomes (e.g., limited provider cognitive bandwidth or reliance on ex ante biased care algorithms) are aggravated. Using detailed, time-stamped electronic health record data from two major hospitals, we document a 20% relative increase in mortality for Black compared to White patients when hospitals operate at capacity, driven entirely by patients with more medical comorbidities. Put differently, 8.5% of Black patient deaths in our sample could have been avoided if Black patients had experienced the same capacity-mortality relationship as White patients. In terms of potential mechanisms, Black patients experienced longer wait times, lower likelihoods of ICU admissions, and had shorter lengths of stay and charges, though this was true at all levels of capacity strain. Dynamic decomposition analyses suggest that these findings are most likely driven by additional biases in provider behavior, hospital processes, and/or allocation of care resources that emerge or worsen as strain increases, which then interact with already lower intensities of care to produce differential mortality risks.

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# 1 Introduction

A large literature has documented racial disparities in health outcomes. These disparities are driven by exposure to structural racism in multiple domains over the life course ([Hardeman et al., 2016](#); [Lavizzo-Mourey et al., 2021](#)). Within this broader set of determinants, health care specific factors, including care patterns in hospitals, appear to play an important role in patterning racial health disparities ([Asch et al., 2021](#); [Chandra et al., 2020](#); [Hasnain-Wynia et al., 2007](#); [Institute of Medicine, 2003](#); [Ross et al., 2007](#)).

Racial disparities in health outcomes in hospital settings may arise due to differences in the quality and intensity of health care provided both across and within hospitals ([Asch et al., 2021](#); [Joynt et al., 2011](#); [Trivedi et al., 2005](#)). Focusing on within-hospital drivers of disparities, a growing body of work suggests the potential importance of implicit and explicit bias among providers ([Aysola et al., 2021](#); [Centola et al., 2021](#); [Johnson et al., 2016](#)), built-in biases in clinical decision algorithms ([Ashana et al., 2021](#); [Obermeyer et al., 2019](#)), differences in opportunities for patient self-advocacy ([Wiltshire et al., 2006](#)), staffing ratios ([Brooks Carthon et al., 2021](#)), and concordance in demographic background and life experiences between care providers and patients ([Alsan et al., 2019](#); [Greenwood et al., 2020](#)).

One hospital-level factor that has received surprisingly little attention is capacity strain, defined as a state of operations where patient needs exceed the clinical resources available to meet them. While a large literature has documented a robust adverse association between hospital (or hospital ward) strain and patient outcomes ([Anesi et al., 2018](#); [Eriksson et al., 2017](#); [Song et al., 2020](#); [Wilcox et al., 2020](#)), the effects of capacity strain on racial disparities in health outcomes is not well known. When hospitals operate under strain, increases in patient needs overwhelm providers and systems, exacerbating the effects of the aforementioned hospital-specific factors, as well as more general racial disparities in health care utilization ([Dieleman et al., 2021](#)), that produce racial disparities in outcomes. For example, hospital strain may strain provider and system resources and cognitive bandwidth ([Arogyaswamy et al., 2021](#)) and thereby lead to an increased reliance on concerning heuristics, such as pre-existing provider-level implicit racial biases ([Johnson et al., 2016](#)) and systems-level algorithms that (unintentionally) allocate resources in biased ways ([Obermeyer et al., 2019](#)). Increased strain may also raise the importance of patient self-advocacy in accessing needed care, potentially leading to worse outcomes for patients and families who face constraints in doing so. Through these and other mechanisms, hospital strain may produce or exacerbate racial disparities in intensity and quality of

care and thus, consequently, health outcomes. The potential importance of hospital strain as one driver of racial disparities in health among hospitalized patients is further underscored by the COVID-19 pandemic, during which unprecedented levels of clinical capacity strain has been associated both with worse inpatient outcomes overall (Bravata et al., 2021; Kadri et al., 2021; Rossman et al., 2021) and in which there has been marked widening of racial disparities in mortality (Asch et al., 2021; Miller et al., 2021; Song et al., 2021).

In this study, we assessed whether hospital strain led to worsening of racial disparities in hospital mortality. We use detailed, time-stamped hospital electronic health records, which allowed us to exploit hourly, plausibly exogenous variation in hospital capacity strain at the time of patient arrival. Time-stamped health record data are not readily available (Neprash et al., 2021; Song et al., 2020), which may be one reason why analyses of strain-related racial disparities in hospital mortality have not yet been conducted. Leveraging these novel data, we found that non-Hispanic Black (hereafter “Black”) patients faced a greater risk of in-hospital mortality than non-Hispanic White (hereafter “White”) patients as hospital strain increases to its highest level. These findings are robust to specification and cannot be explained by changes in patient composition. Black patients experienced longer wait times (e.g., from the emergency department to the inpatient ward or from the ward to the intensive care unit), lower likelihoods of being admitted to intensive care units, shorter lengths of stay, and reduced spending (charges) per episode than similarly comorbid White patients. However, these differences were similar across all levels of capacity strain and, thus, alone cannot explain the observed mortality patterns. Instead, decomposition analyses suggest the wide racial gap in hospital mortality that appears at higher levels of capacity strain can be explained by additional biases in provider behavior and/or hospital processes that emerge or worsen under greater stress. These biases, which are generally not measured in health record data (Rozier et al., 2021), may interact with lower intensities of care to produce racial mortality gaps at high levels of capacity strain.

## 2 Materials and Methods

We used detailed electronic health record (EHR) data on all visits from two large academic hospitals in the Southeast United States for the June 1, 2015- Sep 1, 2016. The uniqueness of these data relative to data used in other EHR-based research lies in the fact that they are time-stamped. That is, we were able to track each patient’s movement through each hospital wing from the time of arrival to discharge. Being able to do so is necessary to obtain precise and plausibly exogenous measures of hospital-wide

capacity strain. We restricted our sample to individuals who are identified as non-Hispanic Black or non-Hispanic White (hereafter, "White"). Institutional Review Board approval was granted by the institution that also provided the data.

*Outcome and Exposure:* Our primary outcome was in-hospital mortality, defined as death occurring anytime after arrival to the hospital but prior to hospital discharge. We chose to examine in-hospital mortality given that this outcome most proximally responds to hospital- and provider-level decision-making. In a sensitivity analysis, we also separately considered discharge to from the hospital to hospice as an additional outcome. We did so given the known higher rates of hospice referral for White compared to Black patients (Asch et al., 2021; Cohen, 2008), which may lead to estimates of racial disparities in in-hospital mortality alone to be overstated. As secondary outcomes, we considered a rich range of measures related to wait times (e.g., time to admission from the emergency department or to the intensive care unit) and intensity of care (e.g., total inpatient charges and length of stay) as a means to assess potential mechanisms.

Our primary exposure of hospital strain was calculated using the total number of patients occupying an inpatient bed in that hospital during the hour of the patient's arrival at the hospital. We explicitly chose this capacity-based measure given emerging literature that they better predict health outcomes than acuity- or turnover-based measures (Kohn et al., 2019). We then generated quintiles of the hospital strain measure with the top quintile specified as "high strain," an ex ante choice that follows the literature (Eriksson et al., 2017) and allows the effect of strain on health outcomes to be nonlinear. We calculated capacity strain quintiles separately for each hospital to create equivalent levels of strain; i.e., even though number of filled beds may vary at each quintile, the top quintile in each of the two hospitals equivalently identify hospitals that are close to capacity).

We assumed that hospital-wide strain at the hour of patient arrival captures random variation in strain, since time of patient arrival to the hospital is random as are hour-to-hour fluctuations in hospital capacity. This assumption, commonly used in the literature on the effects of hospital or ward capacity (Freedman, 2016; KC and Terwiesch, 2012; Kim et al., 2014; Song et al., 2020), is necessary to support causal interpretations of our findings. The assumption is supported by emerging research suggesting that periods of capacity strain are difficult to predict ex ante by hospital leadership, even at the day to day level, let alone by the hour (Arogyaswamy et al., 2021), which is the variation we leverage in the present study . A remaining threat to inference, however, is that hospitals faced with high levels of strain may selectively divert or admit of patients on the basis of key medical characteristics that may predict in-hospital mortality risk. While coordinated processes to divert

specific types of patients are less likely to occur on an hour to hour basis, given known difficulties in addressing capacity strain in real time (Arogyaswamy et al., 2021), we nevertheless interrogated this possibility in a series of robustness checks. Table 1 summarizes differences in patient characteristics by race.

*Estimation of Main Models:* In our main models, we estimated the differences in the likelihoods of in-hospital mortality between Black and White patients at each quintile of hospital strain using the following linear probability regression model:

$$Y_{ijht} = \beta_1 RACE_i + \beta_2 STRAIN_{iht} + \beta_3 RACE_i \cdot STRAIN_{iht} + \mathbf{X}_i \Gamma + \delta_j + \eta_{ht} + \epsilon_{ijht} \quad (1)$$

Specifically, we regressed in-hospital mortality for each admission  $i$  on patient race, quintile of hospital strain at the hour arrival to the hospital, the full series of interaction between patient race and quintiles of hospital strain, and a series of additional patient-level characteristics, including age, second order polynomial for age, sex, whether or not the patient was insured, and the number of Elixhauser comorbidities, Elixhauser mortality index, Elixhauser readmission index (all of which are used to capture various dimensions of patient acuity in the medical literature (Elixhauser et al., 1998; Moore et al., 2017)). The interaction terms between race and hospital strain present the main exposure term, as they recover how mortality risk (conditional on covariates) varies by race across different levels of capacity strain, with the interaction between race and fifth quintile of strain recovering the difference in mortality risk at what we specified as high levels of strain.

We further adjusted for hospital-year ( $\eta_{ht}$ ), and physician of record fixed effects ( $\delta_j$ ). We used physician of record fixed effects to adjust for specific service lines (e.g., surgery, internal medicine, labor and delivery) and, to the extent that the physician of record participated in the entire care episode, fixed physician-specific differences in practice patterns. To make sure our results are not driven by the choice of covariates, we additionally estimated more parsimonious versions of this model (including only age, sex, and hospital and year fixed effects), as well as more saturated models (e.g., including diagnosis related-group (DRG) fixed-effects to ensure comparisons within a specific health condition, interacting hospital strain with all the control variables in  $X_i$ , etc).

### 3 Results

Estimates of the joint effects of race and high strain (i.e., the fifth quintile) on in-hospital mortality are presented in Table A.1. The differences in mortality between Black and White patients at each separate quintile are plotted in Figure I (a). Black patients were 0.4 percentage points more likely than White patients to die in-hospital if they are arrived to the hospital at its highest quintile of strain ( $p$ -value on the interaction term = 0.04) than when they arrived at lower quintiles of strain. Given a baseline mortality rate of 2 per 100 admissions, this estimate represents an 20% relative increase in likelihood of death for Black patients vis-a-vis White patients.

Our central finding of increased mortality for Black vs. White patients at the highest levels of hospital strain – was not sensitive to inclusion or exclusion of various covariates. Figure A.1 (a) only includes age, hospital-, and year- fixed effects in the model in addition to race and hospital strain; (b) then adds in sex; (c) then uses a logit specification; (d) then adds in physician-of-record fixed effects back into the linear specification; and (e) then adds in DRG-fixed effects. The estimated coefficients are similar in magnitude - if not larger - than in our main specification. The coefficient estimates are similar even in a highly saturated specification that includes an interaction between every covariate in the main model with quintiles of hospital strain (Figure A.1 (f)).

We found that these mortality patterns were most stark when comparing Black and White patients with higher ex ante mortality risks (as measured by the Elixhauser mortality index, Fig II). There was no significant difference in the probability of in-hospital mortality between Black and White patients for patients at the 25th percentile of Elixhauser mortality indices at any level of hospital strain. However, for patients at the 75th percentile of Elixhauser mortality indices, Black patients were 0.8 percentage points more likely to die in-hospital at the highest quintile of hospital strain (i.e., a 2% vs. 2.8% predicted probability of in-hospital mortality for White vs. Black patients, respectively, Figure II (b)). (This difference materializes because in-hospital mortality risk for high-comorbidity Black patients remains similar for Black patients at high levels of strain, while it decreases for White patients. We discuss reasons for this pattern below, noting that it is unlikely to be driven by differential admission of patient-types across race as strain increases).

### 4 Potential Explanations and Mechanisms

We interrogated a number of potential explanations and mechanisms for our findings. One reason Black patients may experience greater in-hospital mortality rates than White patients at the highest

levels of hospital strain may be patient selection. For example, if hospitals are selectively visited by sicker patients at high levels of strain, and those patients happen to be predominantly Black, then this differential selection alone may account for our findings (rather than any direct effects of strain on patient care). We assess the possibility of differential selection into our sample in several ways. We estimated versions of our main regression model in which we regressed each patient characteristics (e.g., Elixhauser mortality indices, Elixhauser readmission index, number of Elixhauser comorbidities, age, sex, insurance status) on the main exposures while including *all* other covariates (Figure A.2). In addition to assessing potential patient selection, this specification also helps address the potential measurement error in key covariates (Pei et al., 2019). Though the average comorbidity burden of both Black and White patients admitted to the hospital decreases with high strain – which may reflect hospitals transferring sicker patients to other hospitals when they are at capacity – this tendency does not differ between Black and White patients. While decreasing average comorbidity burdens with increasing strain may explain why the likelihood of in-hospital mortality decreases slightly at high levels of strain for White patients, it cannot explain why the likelihood of in-hospital mortality does not change at high levels of strain for Black patients. In fact, Figure A.2 (a) shows that, Black patients always have a lower mortality index (i.e., have fewer comorbidities that are highly predictive of in-hospital mortality) across all levels of hospital strain, adjusting for all other characteristics. Similarly, we find no evidence that other patient characteristics are differentially changing for Black vs White patients with increasing hospital strain. Finally, we find no differential changes in the likelihood of discharge to hospice care at high strain (Figure A.3), as higher rates of discharge to hospice among White patients at higher levels of strain may mechanically increase Black-White disparities in in-hospital mortality (Asch et al., 2021; Cohen, 2008).

To address potential selection on patient characteristics that were not observed in our data, we calculated and used as an alternate exposure measure hospital strain at the time of hospital discharge, as potential differential patient selection at the time of arrival to the hospital should not be correlated with capacity strain later during the hospital stay. Using this alternate measure of hospital strain, we again found evidence of greater mortality for Black vis-a-vis White patients at the highest level of strain (Figure A.4).

Regarding potential mechanisms underlying the strain-related mortality gap, we first examined racial disparities wait times, as time spent waiting for necessary care may be correlated with mortality risk (Guttman et al., 2011; Kohn et al., 2019; Plunkett et al., 2011). Figure III displays racial disparities in wait times while in the ED (for those admitted through the ED), for inpatient admission

(for all patients admitted through and from outside the ED), and for intensive care unit admission (for those admitted to the ICU). Increasing capacity strain was generally associated with increased wait times for all patients. Black patients waited longer than White patients at every level of hospital strain; in fact Black patients with elevated risk of in-hospital mortality generally waited longer for both inpatient admission and ICU admission than White patients with *lower* risk scores at every level of hospital strain. However, we found no evidence of differential changes in any of the wait time measures for Black vs. White patients with increases in capacity strain.

We also evaluated strain-related differences in intensity of care as a potential mechanism. We found that, across the board, the likelihood of ICU admission decreased with increasing strain, and that Black patients with higher baseline Elixhauser mortality scores are, once again, less likely to be admitted to the ICU than their White counterparts at almost all levels of hospital strain. We again did not find any differential changes in the likelihood of ICU admission or length of stay across racial groups with increasing hospital strain (Figures A.5). (However, in analyses where we decomposed hospital strain into general ward- and ICU-specific strain at the time of patient arrival, we found that Black patients were less likely to be admitted to the ICU as ICU strain, but not general ward strain, increased (Figure A.6). These results suggest that ward-specific measures of strain may have heterogeneous effects on racial disparities in health care, though we were not powered to decompose impacts on mortality by these ward-specific measures.)

We also examined other outcomes that may reflect intensity of care provided: total inpatient length of stay, total inpatient charges, and 30-day readmission rates. Figure A.7 does not show evidence of changing racial disparities in any of these outcomes as a function of hospital strain. Mirroring patterns for wait times and ICU admissions, inpatient charges and lengths of stay were lower for Black versus White patients with similar comorbidity burdens). Additionally, given that these outcomes also are correlated with patient complexity (i.e., readmission rates and inpatient expenditures may be elevated among more medically complex patients), the lack of relative changes in these outcomes for Black vs. White patients as strain increases also supports previously mentioned findings of no differential patient selection by hospital strain.

#### 4.1 Decomposition Analyses

Racial disparities in care inputs and intensity cannot alone drive the main findings, given that these disparities persist equally across all levels of strain. We thus conducted formal decomposition analyses to better elucidate underlying mechanisms.



First, we employed a standard Kitagawa-Oaxaca-Blinder decomposition (Blinder, 1973; Kitagawa, 1955; Oaxaca, 1973) to assess whether static differences in mortality rates between Black and White patients at low, and then high, levels of hospital capacity were due to differences in *levels* of patient characteristics and care inputs (i.e., differences in “endowments”) or due to differences in the *effect* of patient characteristics and care inputs on mortality (i.e., differences in “coefficients”). More intuitively, the “endowments” portion assesses how mortality for White patients would change if they had the same *mean* characteristics and care inputs as Black patients (keeping the coefficients constant), while the “coefficients” portion assesses how White mortality risk would change if they had the same *mapping* of covariates to mortality outcomes as Black patients (keeping the endowments constant).

Figure IV presents the decomposition of the mortality gap separately for low and high strain. We find a suggestive pattern: at both low and high strain, the endowment portion informs us that White patients would have a lower mortality rate if they had the same characteristics and care inputs as Black patients (suggesting White patients are indeed sicker), and the coefficient portion informs us that White patients would have a higher mortality rate if they had the same mapping of characteristics and care inputs onto mortality as Black patients (suggesting better “returns to care” for White patients), which implies that White patients are awarded a degree of protection against mortality that Black patients are not. However, the contribution of the coefficients portion to the mortality gap at high strain is substantially larger than it is at low strain, suggesting that the protective effect that White patients experience (or, what can equivalently be also called the harmful effect that Black patients experience) increases at high strain, leading to the higher mortality observed for Black patients vis a vis White patients.

We further examined why this pattern occurred by using dynamic decomposition methods that decompose the changes in endowments and coefficients from low to high strain. Specifically, we used the Wellington decomposition method (Wellington, 1993) which allows us to separately assess whether the coefficient on the interaction between patient race and the indicator for highest quintile of strain was driven by (1) changes in the endowments of patient characteristics and care inputs (referred to as “component 1”) versus (2) changes in the coefficients on patient characteristics and care inputs as well as the constant term (for race) (referred to as “component 2”). We interpret component 2 as being informative about the extent to which strain-related Black-White gaps in mortality risk might be ameliorated if behavioral and procedural responses to capacity strain – reflected by “returns” to patient characteristics and care inputs – evolved similarly for Black and White patients.

Figure V (with component-specific contributions broken out by patient characteristics, care inputs, hospital-time factors, and the race intercept in Figure A.8) shows that 93% of the effect of high strain on increasing racial disparities in mortality can be explained by component 2 - i.e., changes in the coefficients (including the constant term) on patient characteristics and care inputs as the hospital moves from states of low to high capacity strain, an estimate that is statistically significant. This estimate is driven entirely by differential changes in the race-specific intercept term. (This finding was unchanged by the inclusion of different covariate sets, all of which are correlated with mortality risk in our main regression models. Thus, the loading of the decomposition on the intercept term cannot (entirely) be due to unmeasured covariates that may vary by race (Kim, 2010).) Following the literature in interpreting dynamic decompositions (Wellington, 1993), we conclude that nearly all of the strain-related mortality gap would be eliminated if Black patients experienced the same strain-related changes in provider care patterns and access to hospital resources that White patients did.

These findings are consistent with de facto or de jure racial disparities in hospital processes, provider behavior, and/or returns to care that either emerge or worsen with capacity strain as being a primary driver of strain-related racial disparities in mortality. For example, provider biases or resource allocation choices (that cannot be observed in standard EHR data) that arise at higher levels of strain may put Black patients at higher mortality risk than White patients with similar demographic and comorbidity profiles. These emergent biases - which are typically not measured in electronic health records - may interact with racial disparities in care inputs and intensity that exist at all levels of strain, to produce heightened mortality risk for Black patients vis-a-vis White patients at high levels of capacity strain.

In contrast, less than 7% of the change in racial mortality gap from low to high levels of strain can be attributed to changes in patient characteristics or levels of care (a finding that, too, is highly robust to varying different sets of controls). This is consistent with our findings in prior sections that show no statistically detectable differences in racial disparities in care inputs and patient demographic characteristics as hospitals move from low to high levels of capacity strain. We find similar results using an alternate five-way decomposition method, which addresses potential challenges in cleanly separating coefficient and endowment effects in dynamic settings (Kim, 2010) - see Figure A.9.

## 5 Discussion

Racial disparities in health outcomes arise from myriad systemic, institutional, and interpersonal causes (Institute of Medicine, 2003; Lavizzo-Mourey et al., 2021; Williams and Mohammed, 2009). A large literature has focused on the specific role played by health care systems in patterning these disparities, with evidence suggesting that hospital processes and provider-biases may be important targets for interventions to ameliorate them (Lavizzo-Mourey et al., 2021; Mateo and Williams, 2021). Our study illuminates the importance of these factors by focusing on a hitherto unexplored driver of racial disparities in in-hospital mortality: hospital capacity strain. We found that Black-White disparities in in-hospital mortality markedly increased at the highest levels of hospital strain, a substantively large effect size that was equivalent to 20% of overall in-hospital mortality in our sample. In other words, if Black patients had experienced the same trend in hospital mortality by strain as White patients, 40 excess Black deaths – or 8.3% of all Black deaths in our one-year sample – could have been avoided. These findings could not be explained by racial differences in the types of patients admitted or discharged at high levels of strain. We find that capacity strain worsens care processes for both Black and White patients (such as by increasing wait times and reducing care intensity), and Black patients receive fewer care inputs relative to similarly comorbid White patients at all levels of strain. However, the *difference* in care inputs between Black and White patients remains unchanged with increasing capacity strain, suggesting that our main findings are likely explained by changes in care processes not usually captured by EHR systems.

Instead, our findings suggest that shifts in hospital care processes or provider behavior that either materialize or worsen as strain rises play a dominant role in the explaining the strain-induced racial mortality gap. That is, strain-related shifts in care patterns may either directly differ by patient race, or may interact with the already higher wait times and lower intensity of care that Black patients experience relative to White patients, to produce relatively worse health outcomes for Black patients at higher levels of strain. For example, Black patients may describe their symptoms differently and/or may be less likely to advocate for themselves than White patients, which can translate into higher mortality for Black patients at high strain. This may be either because providers *become* less likely to account for these differences as strain increases, or alternatively, physicians may be *always* less likely to account for these differences but it only “matters” at high strain because it compounds the poor quality of care Black patients already receive at high strain, which leaves no room for such error by physicians. Similarly, implicit provider biases (Johnson et al., 2016) or reliance on algorithmic

allocation of care inputs (Obermeyer et al., 2019), both of which may generate racial disparities in care inputs at all levels of strain, may have deadly consequences when hospitals and hospital staff are under great stress.

These findings – if replicated in other contexts – yield several implications. First, our results suggest that, in addition to addressing factors that generate racial disparities in care more generally, additional safeguards may need to be put in place at high levels of hospital strain to reduce the risk of negative outcomes for Black patients. Second, our findings were observed in a sample of admissions that occurred prior to the COVID-19 pandemic. Thus, strain-related racial disparities in hospital mortality may in fact have been larger during the COVID-19 pandemic, a time of record capacity strain and provider burnout across many US hospitals (Kadri et al., 2021) and a historic reversal of 20 years of progress in narrowing the Black-White life expectancy gap (Andrasfay and Goldman, 2021). Third, the widening of the racial hospital mortality gaps with increasing capacity strain warrants a further examination of the key mechanisms by which such disparities in hospital-level outcomes materialize. Doing so will require more detailed quantitative and qualitative data on changes in hospital and provider care patterns and how these materialize across patients of different backgrounds than what EHRs typically collect (Aysola et al., 2021). For example, emerging literature on hospital capacity strain illustrates a rich set of challenges (e.g., unpredictability of the occurrence capacity strain, variation in nature of strain by source and patient mix, inadequate or unbalanced staffing within and across care units), responses (which are often low-yield, including capping the number of patients providers can care for, prioritizing discharge, geographic reshuffling of patients, and bringing in temporary staff or moonlighters), and consequences (reduced provider bandwidth, competing messages, burnout, and conflict among hospital staff) (Arogyaswamy et al., 2021), all of which may exacerbate racial disparities in health outcomes. Collecting such data will likely be critical for further identifying the underlying drivers behind our findings, and doing so will also serve to mitigate long-standing biases in the structure and content of EHRs (Rozier et al., 2021). We also note that the precise constellation of mechanisms that may explain strain-related disparities in health outcomes may vary across hospital systems.

Our use of dynamic decomposition methods (which are generally underutilized in these applications) was an effort to get around the inherent limitations of EHR data (Rozier et al., 2021). These decompositions can sharpen hospital and provider efforts to identify and ameliorate biases by identifying the classes of potential factors that may explain disparities in health outcomes. A complementary approach to better identifying underlying bias-driven mechanisms would be to focus on a clinically

defined scenario - e.g., chest pain - for which diagnostic and therapeutic interventions (depending on the cause) are known and time to care is of essence. Unfortunately, we lacked statistical power to conduct stratified analyses by medical condition, and leave this analysis to future work.

Limitations of our study include the possibility of unmeasured confounders (even given the tight research design and findings from the health services literature that hospital capacity strain is hard to predict *ex ante*); the lack of higher-resolution data on care inputs, hospital processes, and clinical decision-making; possible differential measurement error in specific patient characteristics (e.g., patient comorbidity indices); and the fact that our data come from two hospitals from a single hospital system (whose patient population is predominantly Black). Nevertheless, our study adds new insights into the drivers of racial disparities in hospital outcomes by identifying hospital capacity strain as a state where built-in structural biases in the health care system may be most dangerous for the health of Black patients.

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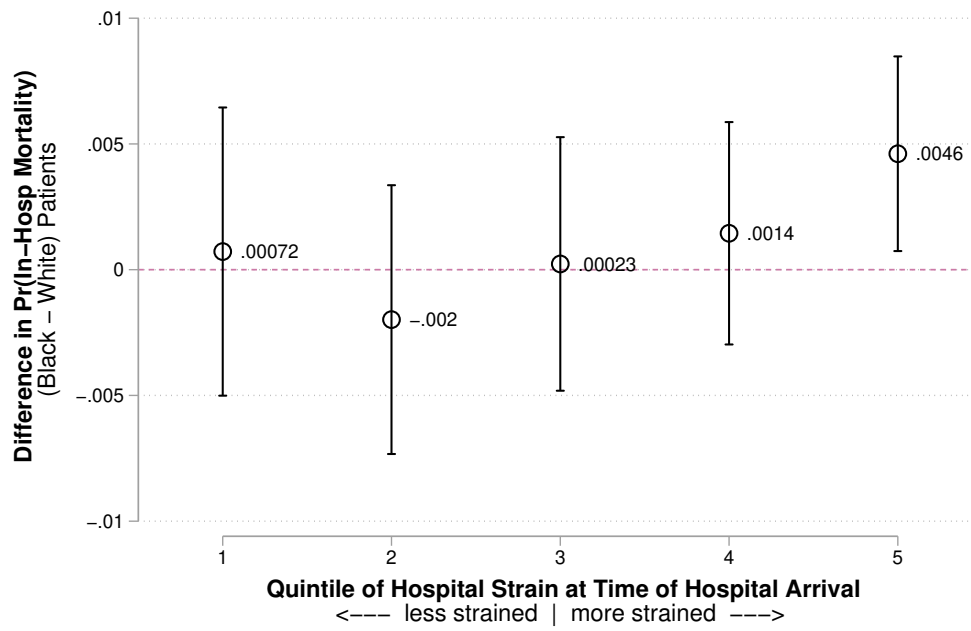
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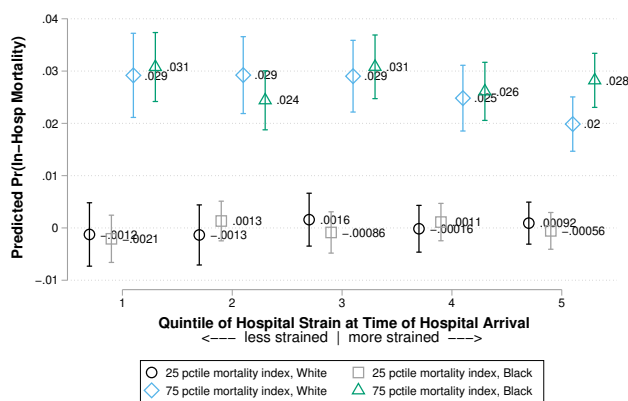
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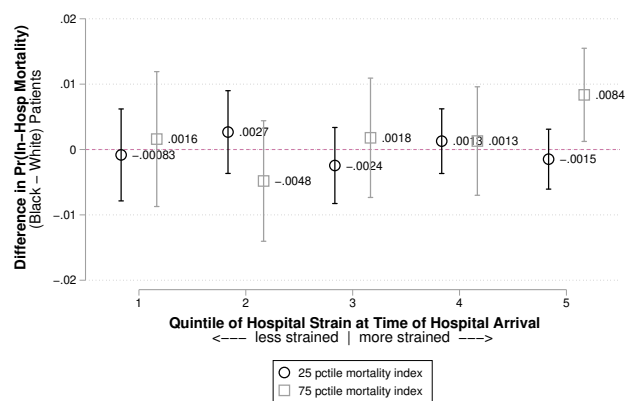


**FIGURE I**  
Racial Gap in In-hospital Mortality By Hospital Strain

This figure plots the difference in in-hospital mortality between Black and White patients using hospital strain at time of patient arrival as the exposure. Patient covariates include age, age squared, sex, Elixhauser readmission index, number of Elixhauser comorbidities, and insurance status. Hospital-year and physician-of-record fixed effects are included. 95% robust standard errors are presented.



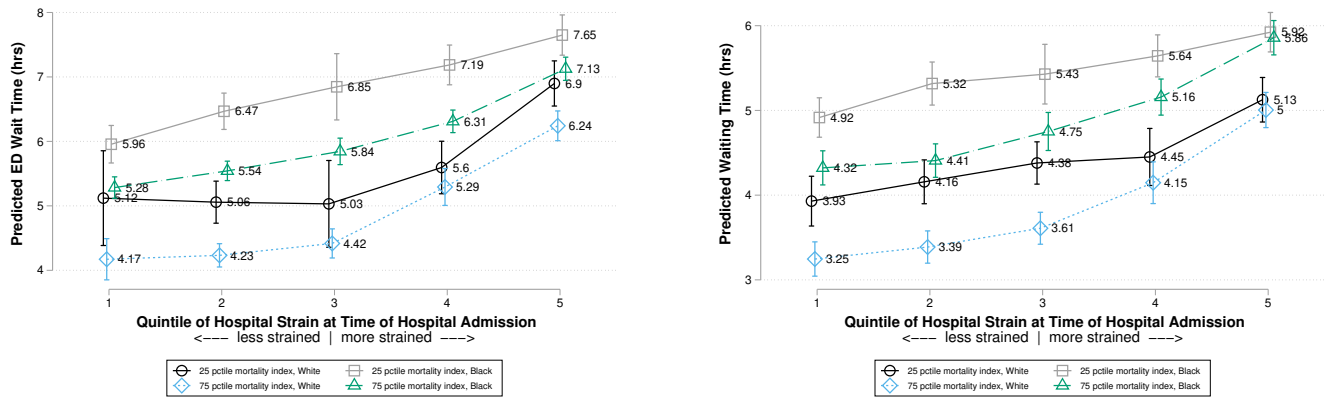
(a) Predicted In-Hospital Mortality



(b) Difference in In-Hospital Mortality

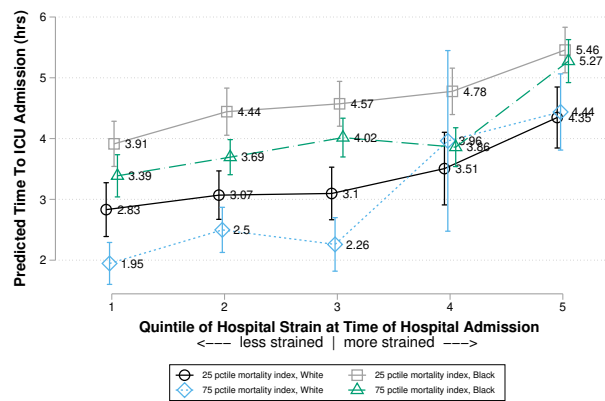
**FIGURE II**  
 Racial Gap in In-hospital Mortality By Hospital Strain –  
 By Ex-Ante Mortality Risk Based on Comorbidities

Figure (a) and (b) plot the difference in, and predicted probabilities of, in-hospital mortality respectively, between Black and White patients, for those at the 25th and 75th percentile of overall Elixhauser mortality index using hospital strain at time of patient arrival as the exposure. Patient covariates include age, age squared, sex, Elixhauser readmission index, number of Elixhauser comorbidities, and insurance status. Hospital-year and physician-of-record fixed effects are included. 95% robust standard errors are presented.



(a) Time in the ED

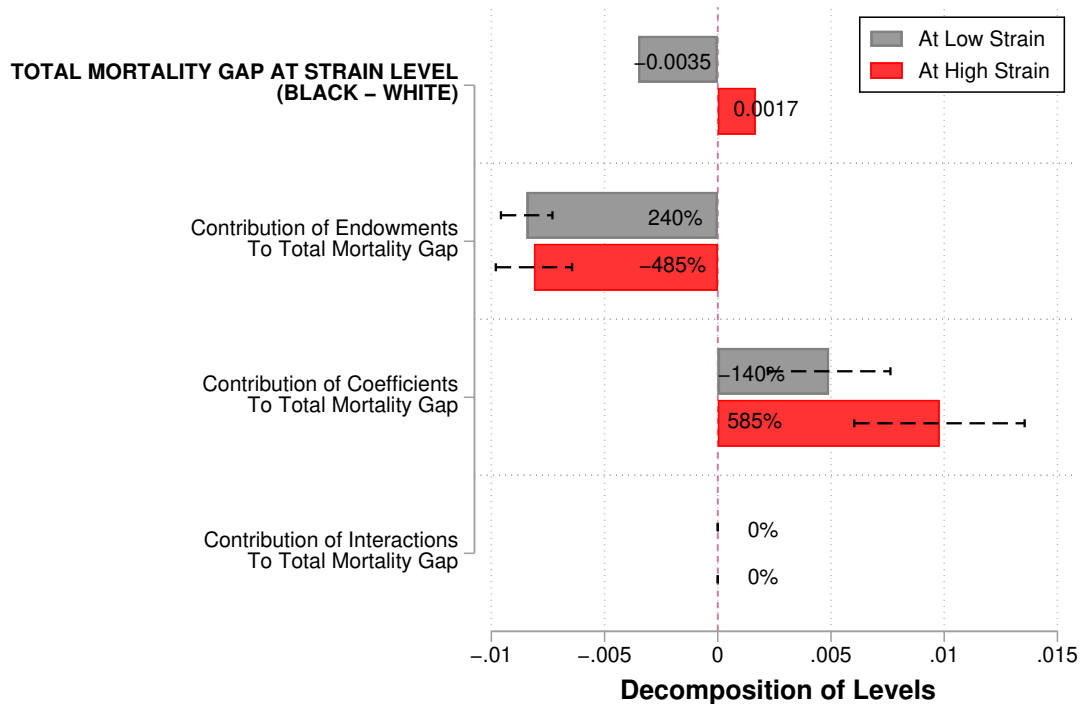
(b) Time to Hospital Floor



(c) Time to ICU Admission

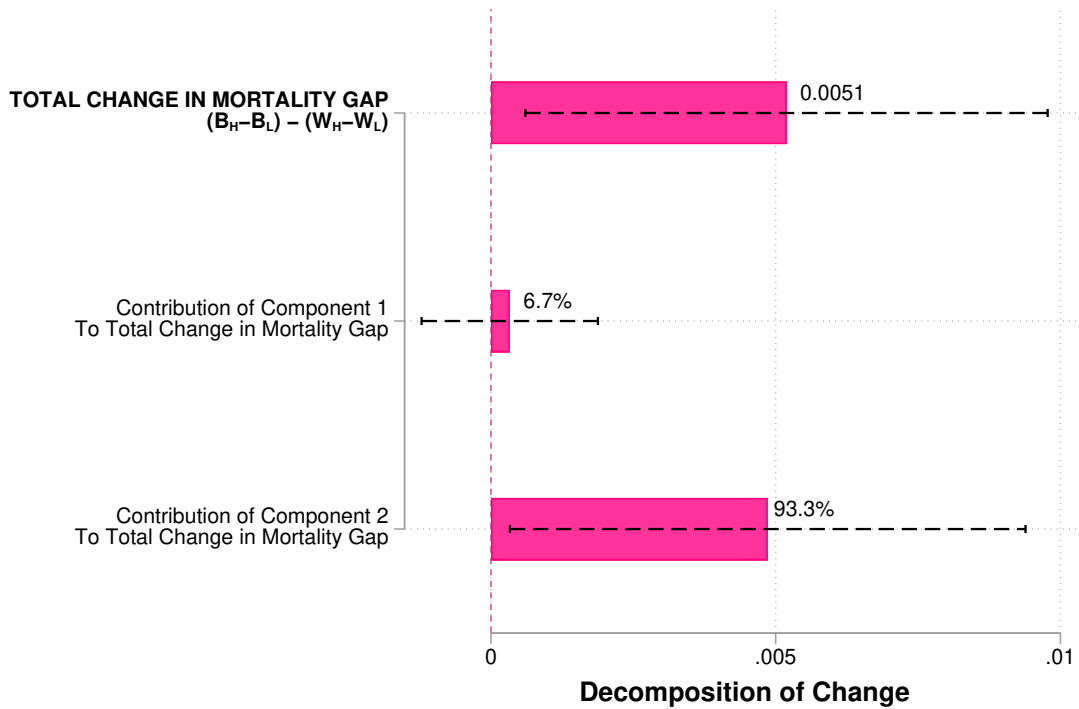
**FIGURE III**  
Inputs to Care: Waiting Time

This figure plots the predicted (a) waiting time in the ED (for those admitted through the ED) (b) predicted time to a hospital ward (for all patients), and (c) waiting time to ICU admission (for those who were directly admitted to the ICU) for Black and White patients at the 25th and 75th percentile of overall Elixhauser mortality index using hospital strain at time of patient arrival as the exposure. Patient covariates include age, age squared, sex, Elixhauser readmission index, number of Elixhauser comorbidities, and insurance status. Hospital-year and physician-of-record fixed effects are included. 95% robust standard errors are presented.



**FIGURE IV**  
 Static Kitagawa-Oaxaca-Blinder Decomposition  
 of the Racial Gap in Hospital Mortality  
 at Low and High Strain

KOB decomposes the racial gap in hospital mortality at high strain into three components: (i) the “endowments”: how much would hospital mortality for Black patients change if Black patients had the same levels of patient characteristics (i.e., age, age squared, sex, uninsurance, Elixhauser readmission index, and Elixhauser mortality index) or the same levels of inputs to care (i.e., waiting time, ICU admission, length of inpatient stay, total inpatient charges) as White patients; (ii) the “coefficients”: how much would hospital mortality for Black patients change if Black patients had the same mapping of patient characteristics/inputs to care to hospital mortality; and (iii) the “interaction”: how much would hospital mortality for Black patients change if Black patients had the same levels and the same mapping of patient characteristics/care inputs to hospital mortality. Positive and negative values on the X axis denote the potential increase and decrease, respectively, in White patients’ mortality if they had the same endowments/coefficients/interactions as Black patients. The percentage amounts indicate the contribution of each component towards the Total Mortality Gap (and should thus add up to 100%, accounting for rounding errors) at low and high strain.



**FIGURE V**  
Wellington Decomposition of the Change in Mortality Gap from Low to High Strain

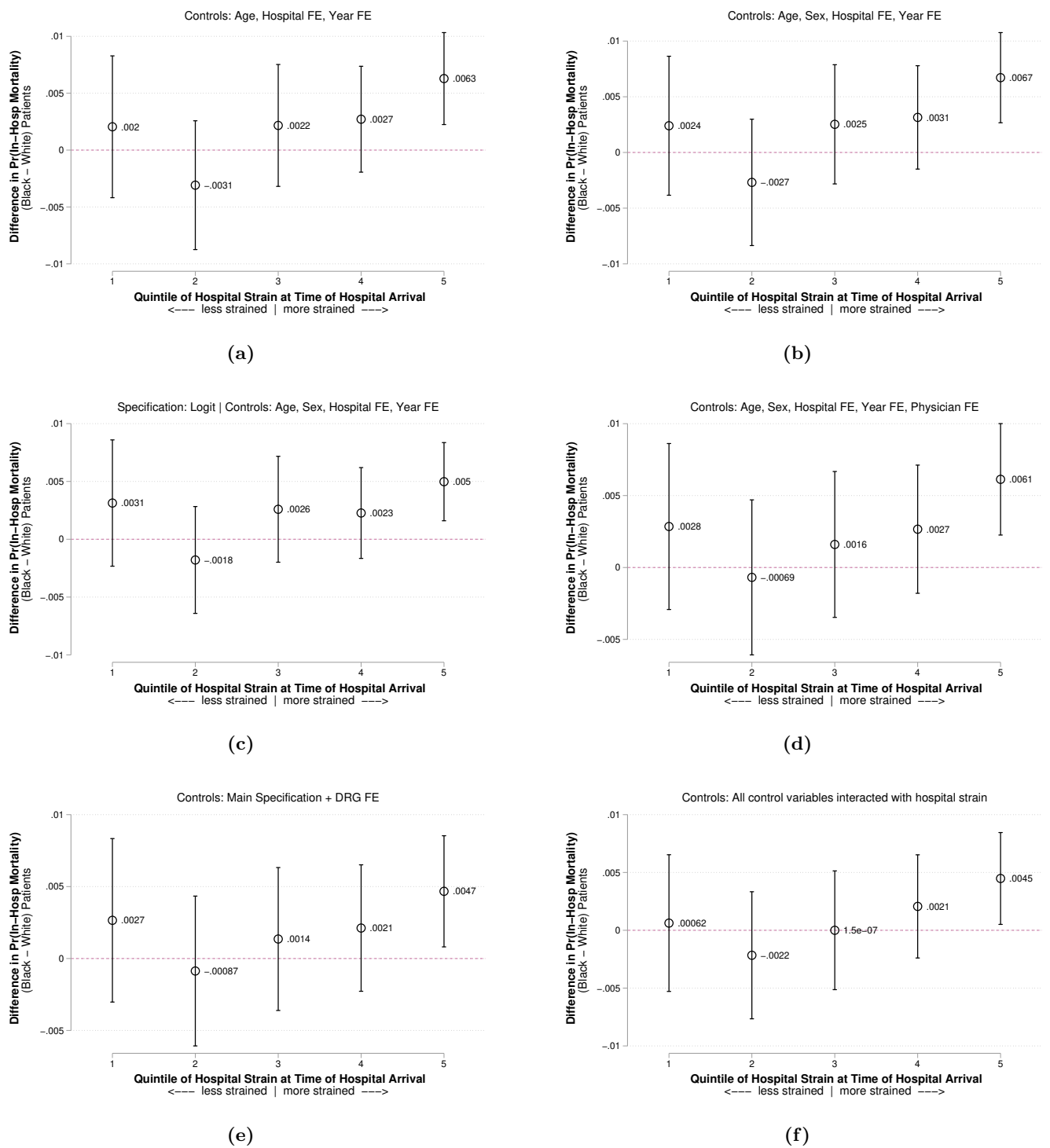
The Wellington Decomposition decomposes the change in the racial gap in hospital mortality from low to high strain into two components: (i) Component 1: the portion of the change in mortality gap that can be explained by changes in the means of patient characteristics (i.e., age, age squared, sex, uninsurance, Elixhauser readmission index, and Elixhauser mortality index), inputs to care (i.e., waiting time, ICU admission, length of inpatient stay, total inpatient charges) or hospital factors (i.e., hospital-year fixed effects); and (ii) Component 2: the portion of the change in mortality gap that can be explained by changes in the coefficients (including the constant term) of patient characteristic, care inputs, and hospital factors. Positive values on the X axis denote the potential increase in the change in White patients' mortality (from low to high strain) if they had the same component 1/ component 2 as Black patients. The percentage amounts indicate the contribution of each component towards the Total Change in Mortality Gap (and should thus add up to 100%, accounting for rounding errors).

**TABLE 1**  
Summary Statistics

	White mean/sd	BLack mean/sd
Age (yrs.)	59.14 (18.03)	52.32 (19.25)
Uninsured	0.10 (0.30)	0.14 (0.35)
Female	0.49 (0.50)	0.63 (0.48)
Elixhauser mortality index	13.21 (12.98)	12.78 (13.41)
Elixhauser 30-day readmission index	23.19 (19.43)	25.18 (21.74)
# of comorbidities	4.00 (2.66)	4.05 (2.97)
ICU Admission	0.27 (0.45)	0.22 (0.42)
In-hospital mortality	0.02 (0.13)	0.02 (0.12)
Length of ICU stay (days)	3.87 (6.87)	3.60 (5.58)
Total inpatient length of stay (days)	7.51 (9.82)	6.99 (12.46)
ED length of stay (hours)	3.77 (7.23)	4.84 (5.19)
Hospital strain at time of hospital arrival (quintiles)	2.28 (1.42)	2.21 (1.42)
Observations	22824	30183

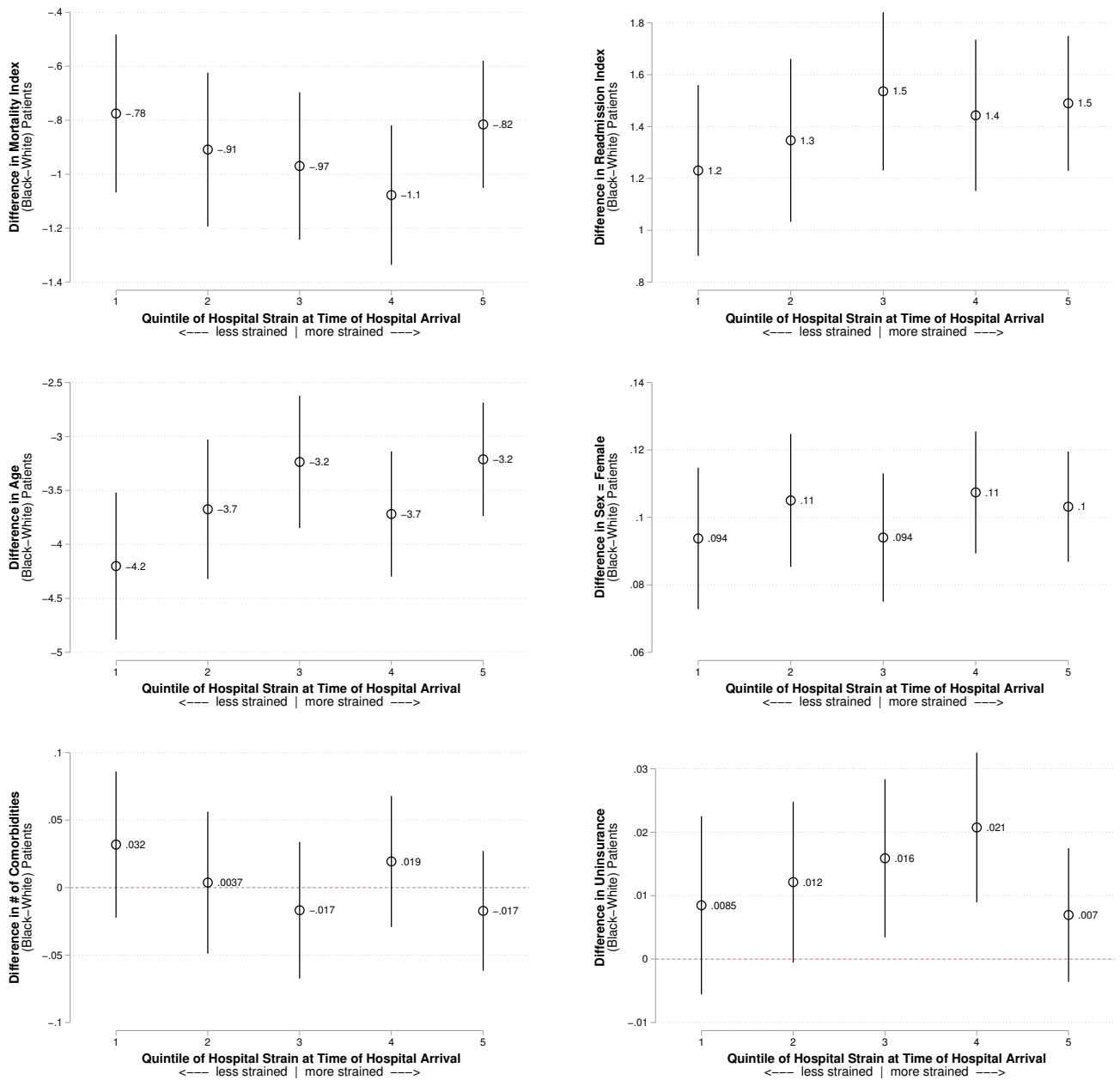


# A Appendix



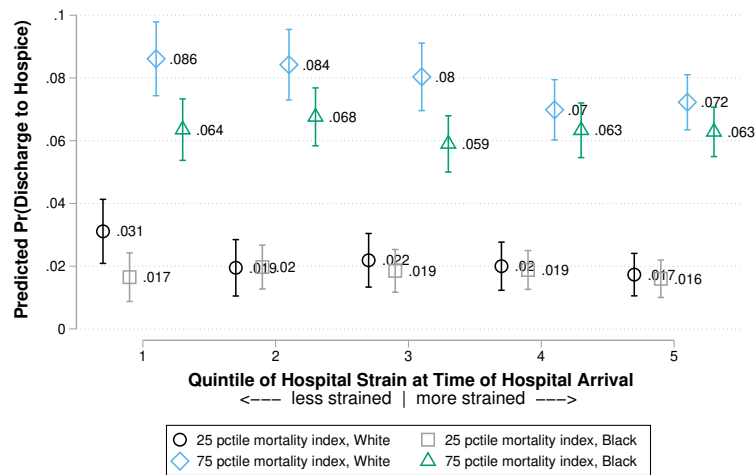
**FIGURE A.1**  
Sensitivity to Alternative Specifications

This figure plots the difference in in-hospital mortality between Black and White patients using hospital strain at time of patient hospital arrival as exposure, under various combinations of covariates and fixed effects. 95% robust standard errors are presented.



**FIGURE A.2**  
Using control variables on the LHS

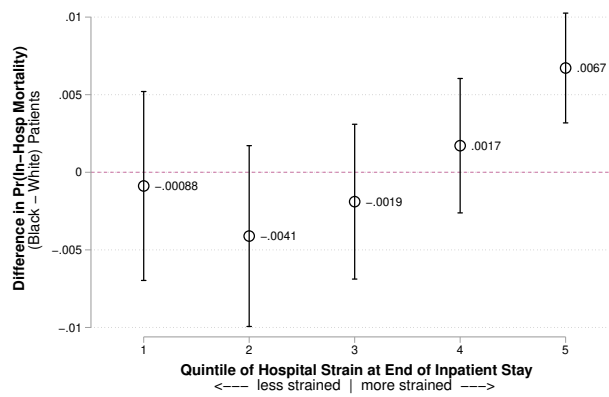
This figure plots the difference in each covariate between Black and White patients using hospital strain at time of patient's hospital arrival as the exposure. In each subfigure, excluding the covariate on the left-hand side (LHS), all other covariates are included in the RHS: Elixhauser mortality index, age, number of Elixhauser comorbidities, Elixhauser readmission index, sex and insurance status. Hospital-year and physician-of-record fixed effects are included. 95% robust standard errors are presented.



(a) Predicted Hospice Discharge

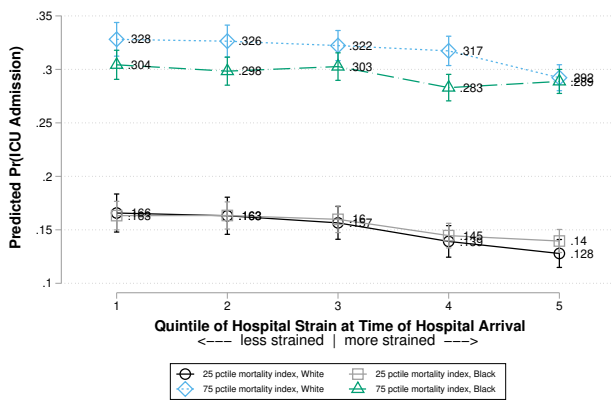
**FIGURE A.3**  
Discharge to Hospice

This figure plots the predicted probabilities of discharge to hospice for Black and White patients at the 25th and 75th percentile of overall Elixhauser mortality index using hospital strain at time of patient arrival as the exposure. Patient covariates include age, age squared, sex, Elixhauser readmission index, number of Elixhauser comorbidities, and insurance status. Hospital-year and physician-of-record fixed effects are included. 95% robust standard errors are presented.

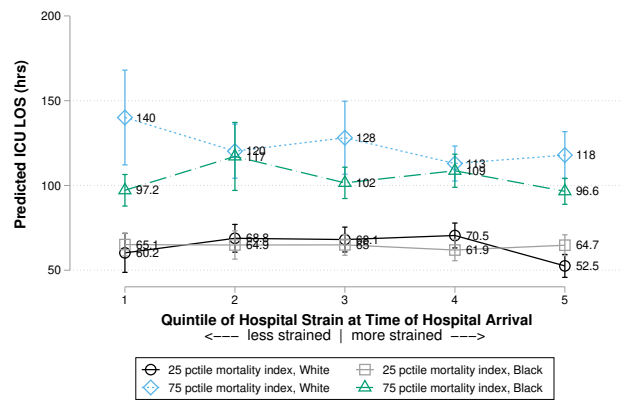


**FIGURE A.4**  
In-hospital Mortality and Hospital Strain at End of Stay

This figure plots the difference in in-hospital mortality between Black and White patients using hospital strain at the end of the patient's inpatient stay as the exposure. Patient covariates include age, age squared, sex, Elixhauser readmission index, number of Elixhauser comorbidities, and insurance status. Hospital-year and physician-of-record fixed effects are included. 95% robust standard errors are presented.



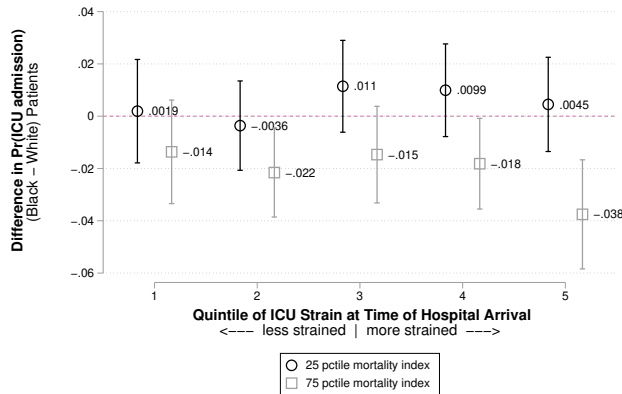
(a) ICU Admission



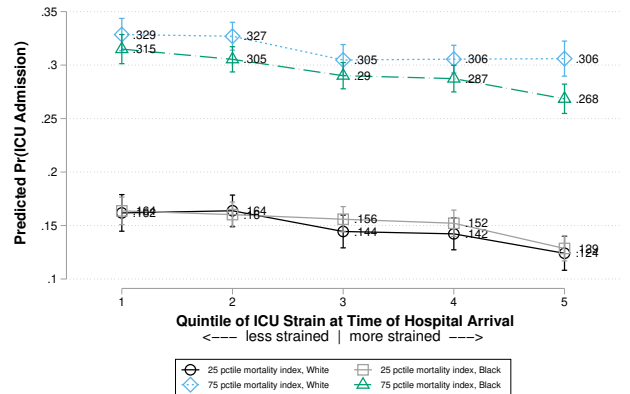
(b) ICU Length of Stay

**FIGURE A.5**  
Inputs to Care: Intensity of Care

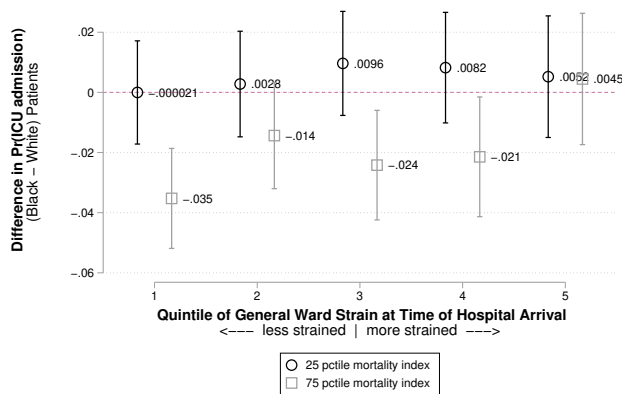
Figures (a) and (b) plot the predicted probabilities of (a) ICU admission, and (b) ICU Length of Stay respectively for Black and White patients at the 25th and 75th percentile of overall Elixhauser mortality index using hospital strain at time of patient arrival as the exposure. Patient covariates include age, age squared, sex, Elixhauser readmission index, number of Elixhauser comorbidities, and insurance status. Hospital-year and physician-of-record fixed effects are included. 95% robust standard errors are presented.



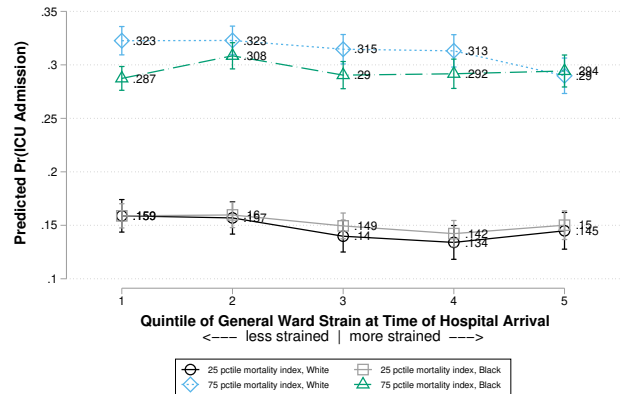
(a) Predicted ICU Admission - Using ICU Strain at Time of Arrival



(b) Predicted ICU Length of Stay - Using ICU Strain at Time of Arrival



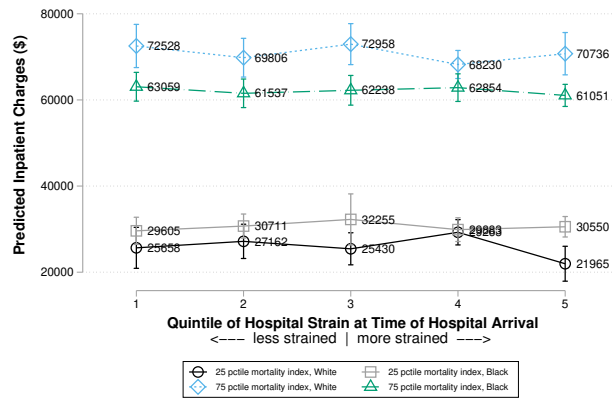
(c) Predicted ICU Admission Using General Ward Strain at Time of Arrival



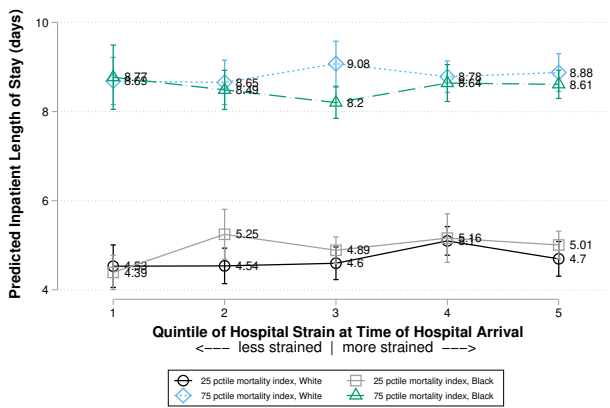
(d) Predicted ICU Length of Stay - Using General Ward Strain at Time of Arrival

**FIGURE A.6**  
Inputs to Care: ICU Admission Decomposed

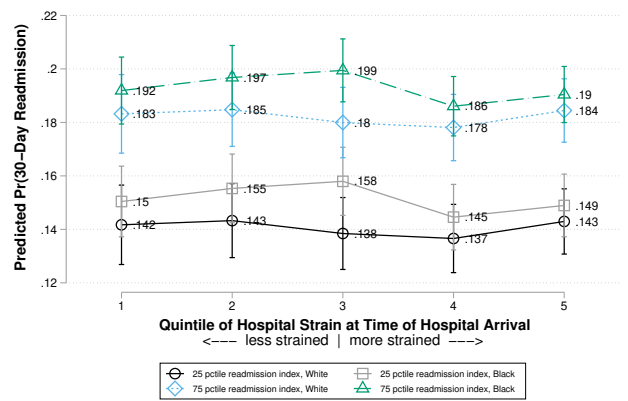
This figure decomposes the effect of hospital-wide strain into the effects of general ward- and ICU-specific strain. Figures (a) and (b) plot the difference in and predicted probabilities of ICU admission respectively, between Black and White patients, for those at the 25th and 75th percentile of overall Elixhauser mortality index using ICU strain at time of patient arrival as the exposure. Figures (c) and (d) plot the same, using general ward strain at the time of patient arrival as the exposure instead. Patient covariates include age, age squared, sex, Elixhauser readmission index, number of Elixhauser comorbidities, and insurance status. Hospital-year and physician-of-record fixed effects are included. 95% robust standard errors are presented.



(a) Inpatient Charges



(b) Inpatient Length of Stay



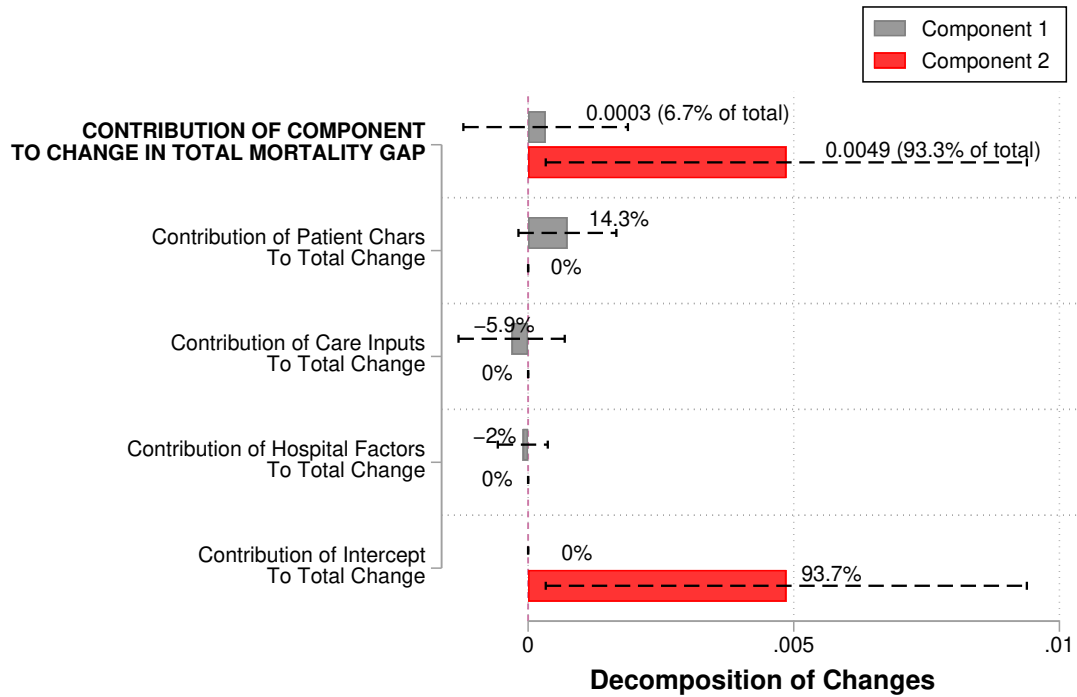
(c) 30-Day Readmission

**FIGURE A.7**

Inputs to Care: Other Indicators of Intensity of Care

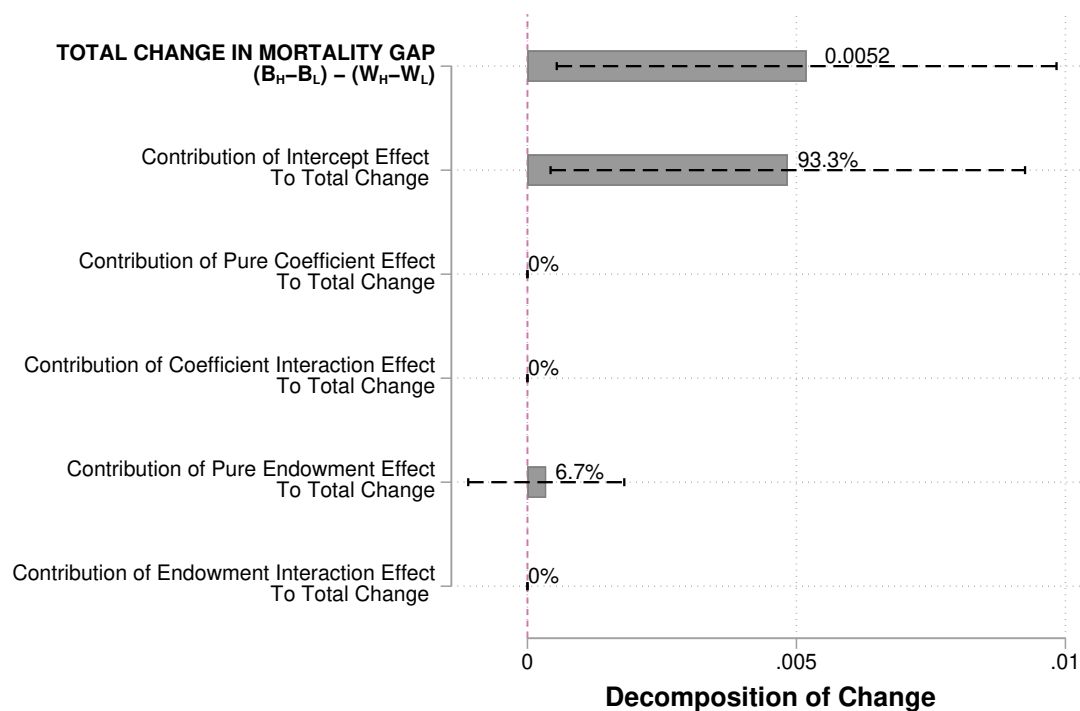
This figure plots the predicted (a) inpatient charges, (b) inpatient length of stay, and (c) 30-day readmission for Black and White patients at the 25th and 75th percentile of overall Elixhauser mortality or readmission index using hospital strain at time of patient arrival as the exposure. Patient covariates include age, age squared, sex, Elixhauser readmission index, number of Elixhauser comorbidities, and insurance status. Hospital-year and physician-of-record fixed effects are included. 95% robust standard errors are presented.





**FIGURE A.8**  
Detailed Wellington Decomposition

The Wellington Decomposition decomposes the change in the racial gap in hospital mortality from low to high strain into two components: (i) Component 1: the portion of the change in mortality gap that can be explained by changes in the means of patient characteristics (i.e., age, age squared, sex, uninsurance, Elixhauser readmission index, and Elixhauser mortality index), inputs to care (i.e., waiting time, ICU admission, length of inpatient stay, total inpatient charges) or hospital factors (i.e., hospital-year fixed effects); and (ii) Component 2: the portion of the change in mortality gap that can be explained by changes in the coefficients (including the constant term) of patient characteristic, care inputs, and hospital factors. Positive values on the X axis denote the potential increase in the change in White patients' mortality (from low to high strain) if they had the same component 1/ component 2 as Black patients. The percentage amounts indicate the contribution of each component towards the Total Change in Mortality Gap (and should thus add up to 100%, accounting for rounding errors).



**FIGURE A.9**

Kim Decomposition of Change in Racial Mortality Gap from Low to High Strain

The Kim decomposition (Kim, 2010) decomposes the racial gap in hospital mortality at high strain into five components (quoted from Kröger and Hartmann (2021)): (i) Intercept Effect: purely the difference in differences between group and overall intercepts; (ii) Pure Coefficient Effect: measures how much the gap between groups changes due to changes in the coefficients if there were no differences in the endowments at all, neither between groups nor over time; (iii) Coefficient Interaction Effect: measures how much the gap between groups changes due to the average change in endowment combined with the difference in the averaged coefficient; (iv) Pure Endowment Effect: measures how much the gap between groups changes due to changes in the endowments if there were no differences in the coefficients at all, neither between groups nor over time; (v) Endowment Interaction Effect: measures how much the gap between groups changes due to the average change in coefficients combined with the difference in the averaged endowments. Positive values on the X axis denote the potential increase in the change in White patients' mortality (from low to high strain) if they had the same component 1/ component 2 as Black patients. The percentage amounts indicate the contribution of each component towards the Total Change in Mortality Gap (and should thus add up to 100%, accounting for rounding errors).

**TABLE A.1**  
Estimates from Main Model

	In-Hospital Mortality
Age (yrs.)	-0.000637*** (0.000164)
Age (squared)	0.00000599*** (0.00000154)
Female	-0.000863 (0.00119)
Uninsured	0.00366** (0.00140)
Black	0.000174 (0.00143)
Highest level of hospital strain	-0.00437**  (0.00175)
<b>Black X Highest level of hospital strain</b>	0.00441** (0.00224)
# of comorbidities	0.00396*** (0.000532)
Elixhauser mortality index	0.00129***  (0.0000981)
Elixhauser readmission index	-0.000430*** (0.0000836)
N	52880
r <sup>2</sup>	0.187
FE	H-Y, Phys

Standard errors in parentheses

\*  $p < 0.10$ , \*\*  $p < 0.05$ , \*\*\*  $p < .001$

This table presents the estimates from regressing in-hospital mortality on race and an indicator for high hospital strain (i.e., the fifth quintile of within-hospital strain). Col(1) presents estimate from the main specification, including covariates, hospital-year- and physician-of-record fixed effects.