CAPACITY STRAIN AND RACIAL DISPARITIES IN HOSPITAL MORTALITY

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ABSTRACT

A growing literature has documented racial disparities in health care. 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 biased care algorithms – are aggravated. Using detailed, time-stamped electronic health record data from two large hospitals, we document that in-hospital mortality increased more for Black patients than for White patients when hospitals approached capacity. We estimate that 8.5% of Black patient deaths were capacity-driven and thus avoidable. We then investigate the extent to which differential care inputs explain our findings. While strain exacerbated wait times similarly for Black and White patients, Black patients both waited the longest at high strain and faced greater mortality consequences from prolonged wait times. Finally, the largest racial disparities in mortality were among women and uninsured patients, highlighting biases in provider behavior and hospital processes as key mechanisms driving our results.

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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). Among this broad class 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; Sun et al., 2022).

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 examined whether patient outcomes worsen with hospital (or hospital ward) strain (Anesi et al., 2018; Eriksson et al., 2017; Evans and Kim, 2006; Hoe, 2022; Hoot and Aronsky, 2008; Marks and Choi, 2019; Song et al., 2020; Wilcox et al., 2020), the effects of capacity strain on racial disparities in health outcomes is not well known. As hospitals approach their capacity, increases in patient needs overwhelm providers and systems, potentially exacerbating racial biases in health care delivery and utilization (Dieleman et al., 2021). For example, hospital strain may challenge system resources and provider 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) or 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, 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 examined whether hospital capacity strain led to a worsening of racial disparities in hospital mortality, using detailed time-stamped electronic health records from two hospitals within a large, highly-regarded academic hospital system located in the Southeast United States that predominantly serve Black patients. These rich data enabled us to leverage hourly variation in patient exposure to hospital capacity strain, which we defined based on the share of all hospital beds occupied at the time of patient arrival. Time-stamped health record data are not generally not available to researchers (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. While the likelihood of hospitals reaching capacity at any given time is ex ante difficult to predict for hospital leaders and staff (Arogyaswamy et al., 2021; Hoe, 2022), we evaluated the potential for differential selection of patients on the basis of their mortality risk as capacity strain increases, a key challenge to causal inference in this, as well as other, research on the consequences of capacity strain on patient health. We then used a range of approaches to examine potential mechanisms for our findings, including examining data on inputs to care such as wait times and total charges, examining differences in the capacity-strain mortality relationship among sub-groups known to experience greater biases in health care delivery (specifically, Black women and uninsured patients), and estimating formal decomposition models.

Our core finding is that in-hospital mortality increased more for Black patients than for White patients when hospitals approached capacity. Our estimates suggest that 8.5% of all Black patient deaths in our sample were capacity-driven and thus avoidable. We show that these findings cannot be explained by strain-related racial differences in patient selection and were robust to a range of specification checks. Instead, we find suggestive evidence that increased wait times played a role in driving the observed strain-related racial disparities in mortality. In addition, the fact that the largest strain-related racial disparities in mortality were among women and uninsured patients highlights the role of biases in provider behavior and hospital processes as key mechanisms driving our results.

The remainder of the paper is as follows. We first discuss our data and main empirical strategy. We then present our results, starting with evidence in support of our causal identifying assumption, followed by our main estimates and results of sensitivity analyses, and ending with an assessment of

potential mechanisms. In the discussion section, we contextualize our findings in the literature, draw policy conclusions, and highlight areas for future work.

2   Materials and Methods

We used detailed electronic health record (EHR) data on all patients admitted to the hospital from two large hospitals within an academic hospital system in the Southeast United States over the period June 1, 2015- Sep 1, 2016. Both hospitals (which cannot be identified for privacy reasons per our data use agreement) are teaching hospitals with over 500 beds. They offer medical, surgical services, intensive care, and obstetric services and serve as level I trauma centers. One of the hospitals is highly ranked nationally in multiple specialty services.

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 admissions for patients who are identified as non-Hispanic Black or non-Hispanic White (hereafter, "White"). Patient race was self-reported (or staff-recorded in the event the patient was unable to self-report). While errors in race attribution are possible, they are likely infrequent and would generate a bias towards the null in our analyses. Institutional Review Board approval was granted by the institution that also provided the data.

Our final sample comprised of 52,880 admissions, of which 30,183 (57%) were for Black patients and 22,824 (43%) were for White patients. (This sample was obtained after excluding 1085 admissions because of missing on race/ethnicity, 1328 admissions for patients with Hispanic ethnicity, and 1569 admissions for recorded races other than Black or White). 71.1% of admissions came through the emergency room. Table 1 summarizes differences in patient characteristics by race (Table A.1 splits the sample by high and low levels of capacity strain). We note that, despite Black patients being on average younger, more likely to be female, and having lower mortality risk scores, their overall in-hospital mortality risk was similar to those of White patients (approximately 2% of all admissions).

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. While we were unable to obtain data on 30-day mortality, a frequently used outcome measure in the health services literature, we believe that in-hospital mortality is an informative outcome for this study for two reasons. First, in-

hospital mortality most proximally responds to hospital- and provider-level decision-making. Second, in-hospital mortality is considered an important quality measure (Jha et al., 2007) and is highly correlated with alternate measures, including 30-day mortality (Borzecki et al., 2010). Nevertheless, to account for any important mortality effects we may be missing with in-hospital mortality, we also separately considered discharge from the hospital to hospice in a sensitivity analysis. 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 to be overstated. (We also considered 30-day readmission as an additional secondary outcome, given its prominent use as a quality measure in the health services literature (Gupta, 2021)).

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 evidence of their strength in predicting health outcomes (Kohn et al., 2019). We first calculated strain at every hour of every day in our sample, based on which we then generated hospital-specific quintiles of our 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. The mean proportion of inpatient beds filled in the first quintile of strain in both hospitals ranged from 70-73%; at the fifth quintile the mean range of filled beds was 91-93% (Table A.2).

Empirical approach: Our core statistical model estimates 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:

$$Y_{ijht} = \beta_1 RACE_i + \boldsymbol{\beta_2}\mathbf{STRAIN_{iht}} + \boldsymbol{\beta_3} RACE_i \cdot \mathbf{STRAIN_{iht}}$$
$$+ \mathbf{X_i} + \delta_j + \eta_{ht} + \epsilon_{ijht} \tag{1}$$

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 and second order polynomial for age; sex, whether or not the patient was insured; and the number of Elixhauser

comorbidities, Elixhauser mortality index, and Elixhauser readmission index. The Elixhauser indices, which were constructed using data from current and any prior admissions during the data period, are used to capture various dimensions of patient acuity in the medical literature (Elixhauser et al., 1998; Moore et al., 2017) and are similarly predictive of mortality risk among admissions for Black and White patients in our sample (Table A.3). The interaction terms between race and hospital strain present the main exposure term, as they recover how the likelihood of in-hospital mortality (conditional on covariates) varies by race across different levels of capacity strain.

We further adjusted hospital-year (η_{ht}), and physician of record fixed effects (δ_j). We used physician of record fixed effects to adjust for specific service lines represented in our sample of admissions (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 also estimated more parsimonious versions of this model (including only age and hospital and year fixed effects), as well as more saturated models (e.g., including day-of-week, hour-of-day, and 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).

The key causal identification assumption of our approach is that hospital-wide strain at the hour of patient arrival is as-if random, i.e., independent of factors correlated with with patient mortality risk and patient race. This assumption is commonly invoked in the literature on the effects of hospital or ward capacity strain (Freedman, 2016; Hoe, 2022; KC and Terwiesch, 2012; Kim et al., 2014; Song et al., 2020), its validity supported by a growing literature that suggests that periods of capacity strain are difficult to anticipate ex ante even at the day to day level, let alone by the hour. For example, a recent qualitative analysis of hospital leaders at 13 U.S. academic medical centers - a setting that is similar to the present study - concluded that "hospital capacity strain is complex and difficult to predict" (Arogyaswamy et al., 2021).

Even if capacity strain is not predictable it is possible that, once it materializes, hospitals may selectively divert or admit patients on the basis of key medical characteristics, or that patients and ambulances may choose to obtain care at less strained hospitals. Importantly, for these processes to bias our findings, any strain-related selection on the basis of patient characteristics would have to differ by race. At the provider level, such coordinated processes to divert specific types of patients are unlikely to occur on an hour to hour basis, given known difficulties in responding capacity strain in real time (Arogyaswamy et al., 2021). In addition, studies examining racial differences in patient arrival

to hospitals find that Black patients with acute, life threatening health conditions may be more likely to be diverted to other hospitals during periods of high strain (Hsia et al., 2017), which would bias estimates of capacity strain-related racial disparities in hospital mortality in the opposite direction of what we hypothesized. Despite support of our causal identification assumption in the literature, we assessed for violations of this assumption in our sample before estimating our core models.

3   Results

Our results are structured as follows. First, we present results of tests designed to probe the validity of our research design. Second, we present our main findings of capacity-strain related racial disparities in hospital mortality. Third, we present results establishing the robustness of the findings to a range of sensitivity analyses. Fourth, we explore potential mechanisms.

3.1   Validity of the causal identifying assumption

We assessed for violations of our causal identifying assumption – namely that hospital capacity strain was associated with changes in the composition of admitted patients in ways that varied by patient race – in several ways. First, we found that distribution of Black and White patients was nearly identical in terms of hour of arrival to the hospital as well as capacity strain at the time of arrival (Figure A.1). Thus, the racial distribution of patients upon hospital arrival did not vary by capacity strain.

Second, while our data did not include information on patients who were not admitted to the hospital (e.g., patients diverted to other hospitals or patients arrived to the emergency room but were not admitted to the hospital), we were able to regress hospitalized patient characteristics (e.g., Elixhauser mortality indices, Elixhauser readmission index, number of Elixhauser comorbidities, age, sex, insurance status) on the main exposure terms, adjusting for all other covariates. This exercise assessed whether mortality-predicting patient characteristics among admitted patients changed at high levels of capacity strain, in ways that varied by patient race. (In addition to assessing differential patient selection, this approach also address the potential measurement error in covariates (Pei et al., 2019)). Though the average comorbidity burden of both Black and White patients admitted to the hospital decreased with high strain (Supplementary Figure A.4) - which may either reflect hospitals diverting/transferring sicker patients to other hospitals when they are at capacity or providers unable to prioritize high-risk patients and instead resorting to admitting patients first-come-first-serve when

strained - this tendency did not differ by race (Supplementary Figure A.2).

Collectively, these analyses support our literature-based contention (Arogyaswamy et al., 2021) that suggest that differential patient selection is unlikely to confound our main empirical model. The validity of our causal identification assumption was further supported by several other sensitivity analyses (described in Section 3.3).

3.2   Main result: strain-related racial disparities in hospital mortality

Figure I plots estimated differences in mortality between Black and White patients at each separate quintile of capacity strain as well as predicted mortality rates at each strain quintile for both groups of patients. Model estimates of the joint effects of race and high strain (i.e., the fifth quintile) on in-hospital mortality are presented in Table A.4. 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 capacity 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% greater likelihood of death for Black patients vis-a-vis White patients. Put differently, these estimates suggest that 8.5% of Black patient deaths were driven by high capacity strain. (We did not find evidence of strain-related racial differences in 30-day readmission likelihood; Figure A.3)

Strain-related racial differences in patient mortality appear to be driven by reductions in mortality among White patients in the fifth quintile of strain, while predicted mortality rates for Black patients are unchanged across levels of strain. The decrease in hospital mortality with elevated strain among White patients can be explained by decreases in average comorbidities levels at high levels of strain. However, we found the same pattern of decreasing comorbidities at high levels of strain for Black patients (Figure A.4). The fact that mortality did not fall in lock-step with decreasing comorbidities for Black patients while it did do so for White patients is consistent with racial biases in care patterns, which we evaluate in Section 3.4.

Strain-related racial disparities in mortality were starkest 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)).

3.3 Additional sensitivity analyses

The main findings were robust to a number of sensitivity analyses. First, the results were not sensitive to the choice of covariates or estimation approach. Figure A.5 (a) only includes age, hospital-, and year- fixed effects in the model in addition to race and hospital strain; (b) fits a logistic regression model instead of our preferred linear probability model; (c) then adds in physician-of-record fixed effects back into the linear specification; and (d) then adds in DRG-fixed effects; and (e) then adds in day-of-week and hour-of-day fixed effects. The estimated coefficients are always 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.5 (f)). (This saturated specification addresses the possibility that strain-related changes in the relationship between specific patient attributes and mortality confounds estimates of the strain-mortality relationship by patient race.) Further, to address concerns that adjustment using weighted, summative indicies of comorbidities may be more prone to bias that adjusting for the individual components that make up these indicies (Möller et al., 2021), we estimated models in which we included each of the 31 comborbidities that comprise the Elixhauser indices as covariates. We also estimated models adjusting for the the first three principal components derived from the set of 31 comorbidities. The findings remained unchanged (Figures A.5 (g) and (h), respectively).

Our findings were also robust to accounting for potential bias from unobserved patient or hospital characteristics. Using Oster (2019)'s approach to quantifying bias from unobservables, we found that, assuming that unobserved covariates explain the joint effect of race and strain on mortality equally as much as the observed covariates, the lower bound of the estimated strain-mortality relationship implied similarly large effects as what we find in our main specification (Supplementary Table A.5). Further, the bias from unobserved confounders would have to be 17 times the relationship between observed characteristics and mortality to overturn our main findings, which is well above the rule-of-thumb threshold of 1 for this test (Oster, 2019). Notably, this finding further supports our causal identification assumption that strain-related differential patient selection is unlikely to bias our results.

To further interrogate the potential effects of unobserved patient characteristics, we calculated an alternate measure of capacity strain: hospital strain at the time of patient's hospital discharge (rather than at arrival). The rationale for this check is that capacity strain faced towards to the end

of a hospital stay is unlikely to influence the probability a given patient was admitted to begin with. In addition, any effects of capacity strain at the end of stay may more purely capture constraints on short-term provider or hospital decision-making with respect to individual patients. Moreover, while this measure is biased (providers are prone to selective discharge of patients during periods of high capacity strain), it does not share the same bias as our primary measure of strain (at patient arrival). Using this alternate measure of hospital strain, we again found evidence of greater mortality for Black patients relative White patients at the highest level of strain, with magnitudes even higher than those found in our core specification (Supplementary Figure A.6). This suggests that there may be something specific about hospital strain that differentially affects Black vs White patient mortality that cannot be simply explained away by unobserved mortality risk or differential admission practices.

Finally, Black-White differences in the likelihood of discharge to hospice actually declined as strain increased (Figure A.7). This finding runs in the opposite direction of the pattern that could explain our findings for the main outcome of in-hospital mortality, namely that relatively 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).

3.4 Potential mechanisms

Care inputs: Regarding potential mechanisms underlying the strain-related hospital mortality gap, we first examined racial disparities in wait times, given prior evidence that Black patients tend to wait longer for care than White patients and that time spent waiting to receive care (Lu et al., 2021) may be correlated with mortality risk (Guttmann et al., 2011; Kohn et al., 2019; Plunkett et al., 2011). Figure III (a) presents estimates from a model that uses wait times for inpatient admission as the dependent variable in our main empirical specification (adjusted for the same rich set of patient demographic characteristics, comorbidities, and hospital-year and physician-of-record fixed effects as in our main models). The results show that strain increases wait times for both Black and White patients, though Black patients waited longer than White patients at every level of hospital strain. We then decompose these effects by patient mortality risk. Strikingly, Black patients with higher risk of in-hospital mortality generally waited longer for a hospital bed than White patients with *lower* risk of in-hospital mortality at every level of hospital strain (Fig III c). These patterns are also seen for wait times in the ED (for patients admitted via the ED) and for ICU care (for patients admitted directly to the ICU) (Supplementary Figure A.8).

These patterns aside, we found no evidence of *differential* changes in wait times for Black vs.

White patients with increasing capacity strain (Figure III b and d). Nevertheless, it is still possible that changes in wait times may contribute to strain-related racial mortality gaps if the consequences of increased wait times are worse for Black patients. This could occur if, for example, Black patients were generally more likely to be mistriaged than White patients (Schrader and Lewis, 2013), and so longer wait times for potentially sicker patients could lead to elevated mortality risk. We find evidence that, as wait time increased, average likelihood of in-hospital mortality was higher for Black patients in our sample (Figure A.9). Thus, while both Black and White patients experienced higher wait times as a result of capacity strain, Black patients both waited longer (than White patients) at high strain as well as experienced potentially more unfavorable mortality risk (than White patients) when waiting longer. Based on estimates from Table A.6 (Col 2), accounting for the differential effect of wait times by race on mortality reduces the strain-related racial mortality gap by 20.5% (i.e., the estimate on the interaction decreases from 0.44 to 0.35 percentage points and loses statistical significance) .

We also evaluated strain-related differences measures of intensity of care as potential mechanisms (Figure A.10). We found that Black patients generally had lower rates of ICU admissions and ICU lengths of stay than White patients, similar overall lengths of stay, and lower inpatient charges than white patients. However, unlike wait times, several of these differences were relatively small to begin with, at all levels of strain, changed little (in absolute terms) with strain, and, if anything, tended to narrow at high levels of strain. Therefore, these specific inputs were less likely to explain strain-related racial disparities in mortality than wait times (this takeaway is supported by Table A.6, where each of these aforementioned inputs to care is interacted with race and included in the main model yet fails to significantly reduce the interaction coefficient between race and high capacity strain).

Provider and system biases : The findings of strain-related racial mortality gaps, as well as differences in wait times alone, highlight the importance of provider and systemic implicit/ explicit biases in generating racial disparities in hospital outcomes. While we did not have detailed data on specific clinical decisions, or attributes and actions of different providers and staff involved in each patient's care, we were able to interrogate the role of biases among providers and systems by examining outcomes for key subgroups who have been shown to be more prone to have their care affected by these biases. We focused on two groups: women and uninsured patients. Black women, relative to Black men, White women, and White men, are less likely to have their symptoms adequately recognized and receive therapies, with biased care documented for cardiovascular diseases (Schulman et al., 1999) and in the obstetric setting (Saluja and Bryant, 2021). Black women are also afforded fewer opportunities

to advocate for scarce care resources (Wiltshire et al., 2006). Similarly, uninsured patients have been shown to receive worse care (Doyle Jr, 2005), in ways that are racially patterned (Stepanikova and Cook, 2008). Consistent with this literature, we found that the overall strain-related racial disparities in hospital mortality were larger for women and uninsured patients (Figure IV). For both groups, racial mortality gaps arose at lower levels of capacity strain (quintile 4).

Decomposition analyses: We supplemented our analyses of care inputs and subgroup differences with formal econometric decompositions. Broadly, these approaches assess whether strain-related differences in mortality Black and White patients were due to differences in *levels* of patient characteristics and care inputs or due to differences in the *effect* of patient characteristics and care inputs on mortality. We used the dynamic Wellington decomposition method (Wellington, 1993), which decomposes the change in the mortality gap from low to high strain into (1) changes in the endowments of patient characteristics and care inputs, keeping coefficients constant (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), keeping endowments constant (referred to as "component 2"). We interpreted component 2 as being informative about the extent to which strain-related Black-White gaps in hospital mortality 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.11) 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 was 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 attributed to unmeasured covariates that may vary by race. (This finding is also mirrored when we use a separate decomposition method which explicitly examines the extent to which the emerging mortality gap at high strain is driven by changes in the race intercept (Figure A.12) (Kim, 2010)).

Following the literature in interpreting dynamic decompositions (Wellington, 1993), these findings suggest 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. That is, the strain-related racial mortality gap in our sample was likely driven by provider and system biases in the provision of health care. Similar results were obtained when using a standard Kitagawa-Oaxaca-Blinder decomposition approach (Blinder, 1973; Kitagawa, 1955; Oaxaca, 1973), which evaluated sources of racial differences in hospitals mortality separately at low and high levels of hospital strain Figure A.13. Notably at both low and high strain, this analysis shows 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. This finding further confirms other results that differential selection on patient characteristics is unlikely to explain main findings.

4   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 hospital mortality: hospital capacity strain. We found that Black-White disparities in hospital mortality markedly increased at the highest levels of capacity 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 8.5% of all Black deaths in our one-year sample – could have been avoided.

These findings were not explained by racial differences in the types of patients admitted or discharged at high levels of strain. Instead, we find that capacity strain worsened care processes for both Black and White patients, and Black patients received fewer care inputs all levels of strain. These changes were particularly marked for wait times. While the relative racial gap in wait times did not change with increasing strain, we find evidence that longer wait times for Black patients may have been more consequential for hospital mortality than similar changes for White patients, suggesting a

proximal mechanisms for our main results.

More generally, our findings suggest that shifts in hospital care processes or provider behavior that materialize or worsen as strain rises likely play a dominant role in the explaining the strain-induced racial mortality gap. This contention is underscored by larger impacts of capacity strain for Black women and Black uninsured patients, who have been shown to face the greatest implicit and explicit biases in health care provision (Doyle Jr, 2005; Schulman et al., 1999; Stepanikova and Cook, 2008). These biases may materialize at high levels of strain in several ways. For example, Black patients with similar illnesses may have different symptomatology or may be less able to advocate for themselves than White patients for scarce resources, which can translate into relatively 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.

Our findings – if replicated in other contexts – yield several implications. First, our results shed light on some of the mixed findings in the literature examining the consequences of hospital capacity strain on patient outcomes, which collectively examines a range of clinical contexts and patient populations (Anesi et al., 2018; Eriksson et al., 2017; Evans and Kim, 2006; Hoe, 2022; Hoot and Aronsky, 2008; Marks and Choi, 2019; Song et al., 2020; Wilcox et al., 2020). Our results indicate that strain may affect some patient groups more than others, underscoring the need to focus on ex ante at risk patient populations.

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, while our results shed some insight into potential mechanisms by which capacity strain may harm Black patients, 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 is sorely needed (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 can also mitigate long-standing biases in the structure and content of EHRs (Rozier et al., 2021; Sun et al., 2022). We note that the precise constellation of mechanisms that may explain strain-related disparities in health outcomes may vary across hospital systems, and that hospital and provider-specific mechanisms may themselves represent only a small portion of the sum of structural forces that pattern racial mortality disparities, more generally.

Fourth, these findings underscore the urgent need to ensure high quality patient care during periods of capacity strain. The COVID-19 pandemic has spurred new approaches to predicting periods of high capacity (Weissman et al., 2020), which may help hospitals respond to strain in advance by increasing staffing or other measures. Creating ex ante networks and decision rules to promote load-shifting to other hospitals during periods of elevated strain can also help ensure that hospital resources for existing patients are not stretched beyond capacity (Boudourakis et al., 2020). In addition, sustained multi-level approaches to address biases in care provision at all levels of strain (Vela et al., 2022) – which could include development and implementation of novel decision-support algorithms to improve allocation of care inputs to patients whose high mortality risks may be systematically missed by providers (Mullainathan and Obermeyer, 2022), de-adoption of de facto biased care algorithms (Obermeyer et al., 2021, 2019), development of provider peer networks to reduce biases in treatment decisions (Centola et al., 2021), and interventions to support patient self-advocacy – may have their largest impacts when hospital capacity strain is elevated, and biases in care provision become more rampant.

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). An ideal approach to examining the consequences of capacity strain on racial mortality disparities – as well as to identify potential mechanisms – would be to focus on a clinically defined scenario (e.g., chest pain) for which diagnostic and therapeutic interventions are known, readily measured, and time to care is of essence. Unfortunately, we lacked statistical power to conduct stratified analyses by specific medical condition, and leave this analysis to future work. Despite these limitations, 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.

5 Conclusion

We found that in-hospital mortality increased more for Black patients than for White patients as hospitals approached their capacity, and provide suggestive evidence that is pattern was likely driven by provider and system biases that emerge or worsen as hospitals face strain. These findings underscore the importance of interventions that equip hospitals to better predict and respond to periods of capacity strain as well as eliminate systematic biases in the care of Black patients.

Bibliography

Alsan, M., Garrick, O., and Graziani, G. (2019). Does Diversity Matter for Health? Experimental Evidence from Oakland. *American Economic Review*, 109(12):4071–4111.

Andrasfay, T. and Goldman, N. (2021). Reductions in 2020 us life expectancy due to covid-19 and the disproportionate impact on the black and latino populations. *Proceedings of the National Academy of Sciences*, 118(5).

Anesi, G. L., Liu, V. X., Gabler, N. B., Delgado, M. K., Kohn, R., Weissman, G. E., Bayes, B., Escobar, G. J., and Halpern, S. D. (2018). Associations of Intensive Care Unit Capacity Strain with Disposition and Outcomes of Patients with Sepsis Presenting to the Emergency Department. *Annals of the American Thoracic Society*, 15(11):1328–1335.

Arogyaswamy, S., Vukovic, N., Angela Keniston, A., Apgar, S., Bowden, K., Kantor, M., Diaz, M., McBeth, L., and Burden, M. (2021). The impact of hospital capacity strain: a qualitative analysis of experience and solutions at 13 academic medical centers. *Journal of General Internal Medicine*, epub before print.

Asch, D. A., Islam, M. N., Sheils, N. E., Chen, Y., Doshi, J. A., Buresh, J., and Werner, R. M. (2021). Patient and Hospital Factors Associated With Differences in Mortality Rates Among Black and White US Medicare Beneficiaries Hospitalized With COVID-19 Infection. *JAMA network open*, 4(6):e2112842.

Ashana, D. C., Anesi, G. L., Liu, V. X., Escobar, G. J., Chesley, C., Eneanya, N. D., Weissman, G. E., Miller, W. D., Harhay, M. O., and Halpern, S. D. (2021). Equitably Allocating Resources during Crises: Racial Differences in Mortality Prediction Models. *American Journal of Respiratory and Critical Care Medicine*, 204(2):178–186.

Aysola, J., Clapp, J. T., Sullivan, P., Brennan, P. J., Higginbotham, E. J., Kearney, M. D., Xu, C., Thomas, R., Griggs, S., Abdirisak, M., Hilton, A., Omole, T., Foster, S., and Mamtani, M. (2021). Understanding Contributors to Racial/Ethnic Disparities in Emergency Department Throughput Times: a Sequential Mixed Methods Analysis. *Journal of General Internal Medicine*.

Blinder, A. S. (1973). Wage Discrimination: Reduced Form and Structural Estimates. *The Journal of Human Resources*, 8(4):436–455. Publisher: [University of Wisconsin Press, Board of Regents of the University of Wisconsin System].

Borzecki, A. M., Christiansen, C. L., Chew, P., Loveland, S., and Rosen, A. K. (2010). Comparison of in-hospital versus 30-day mortality assessments for selected medical conditions. *Medical care*, pages 1117–1121.

Boudourakis, L., Silvestri, D., Natsui, S., Salway, R., Krouss, M., Uppal, A., Izaguierre, A., Siegler, M., Bernstein, S., Prieskorn, K., et al. (2020). Using interfacility transfers to 'level-load' demand from surging covid-19 patients: lessons from nyc health+ hospitals. *Health Affairs Blog*, 1377.

Bravata, D. M., Perkins, A. J., Myers, L. J., Arling, G., Zhang, Y., Zillich, A. J., Reese, L., Dysangco, A., Agarwal, R., Myers, J., Austin, C., Sexson, A., Leonard, S. J., Dev, S., and Keyhani, S. (2021). Association of Intensive Care Unit Patient Load and Demand With Mortality Rates in US Department of Veterans Affairs Hospitals During the COVID-19 Pandemic. *JAMA Network Open*, 4(1):e2034266.

Brooks Carthon, M., Brom, H., McHugh, M., Sloane, D. M., Berg, R., Merchant, R., Girotra, S., and Aiken, L. H. (2021). Better Nurse Staffing Is Associated With Survival for Black Patients and Diminishes Racial Disparities in Survival After In-Hospital Cardiac Arrests. *Medical Care*, 59(2):169–176.

Centola, D., Guilbeault, D., Sarkar, U., Khoong, E., and Zhang, J. (2021). The reduction of race and gender bias in clinical treatment recommendations using clinician peer networks in an experimental setting. *Nature communications*, 12(1):1–10.

Chandra, A., Kakani, P., and Sacarny, A. (2020). Hospital Allocation and Racial Disparities in Health Care. Working Paper 28018, National Bureau of Economic Research. Series: Working Paper Series.

Cohen, L. L. (2008). Racial/ethnic disparities in hospice care: A systematic review. *Journal of Palliative Medicine*, 11(5):763–768.

Dieleman, J. L., Chen, C., Crosby, S. W., Liu, A., McCracken, D., Pollock, I. A., Sahu, M., Tsakalos, G., Dwyer-Lindgren, L., Haakenstad, A., et al. (2021). Us health care spending by race and ethnicity, 2002-2016. *JAMA*, 326(7):649–659.

Doyle Jr, J. J. (2005). Health insurance, treatment and outcomes: using auto accidents as health shocks. *Review of Economics and Statistics*, 87(2):256–270.

Elixhauser, A., Steiner, C., Harris, D. R., and Coffey, R. M. (1998). Comorbidity measures for use with administrative data. *Medical care*, pages 8–27.

Eriksson, C. O., Stoner, R. C., Eden, K. B., Newgard, C. D., and Guise, J.-M. (2017). The Association Between Hospital Capacity Strain and Inpatient Outcomes in Highly Developed Countries: A Systematic Review. *Journal of General Internal Medicine*, 32(6):686–696.

Evans, W. N. and Kim, B. (2006). Patient outcomes when hospitals experience a surge in admissions. *Journal of Health Economics*, 25(2):365–388.

Freedman, S. (2016). Capacity and utilization in health care: the effect of empty beds on neonatal intensive care admission. *American Economic Journal: Economic Policy*, 8(2):154–85.

Greenwood, B. N., Hardeman, R. R., Huang, L., and Sojourner, A. (2020). Physician–patient racial concordance and disparities in birthing mortality for newborns. *PNAS*, 117(35):21194–21200.

Gupta, A. (2021). Impacts of performance pay for hospitals: The readmissions reduction program. *American Economic Review*, 111(4):1241–83.

Guttmann, A., Schull, M. J., Vermeulen, M. J., and Stukel, T. A. (2011). Association between waiting times and short term mortality and hospital admission after departure from emergency department: population based cohort study from ontario, canada. *Bmj*, 342.

Hardeman, R. R., Medina, E. M., and Kozhimannil, K. B. (2016). Structural Racism and Supporting Black Lives — The Role of Health Professionals. *New England Journal of Medicine*, 375(22):2113–2115.

Hasnain-Wynia, R., Baker, D. W., Nerenz, D., Feinglass, J., Beal, A. C., Landrum, M. B., Behal, R., and Weissman, J. S. (2007). Disparities in health care are driven by where minority patients seek care: examination of the hospital quality alliance measures. *Archives of Internal Medicine*, 167(12):1233–1239.

Hoe, T. P. (2022). Does hospital crowding matter? evidence from trauma and orthopedics in england. *American Economic Journal: Economic Policy*, 14(2):231–62.

Hoot, N. R. and Aronsky, D. (2008). Systematic review of emergency department crowding: causes, effects, and solutions. *Annals of emergency medicine*, 52(2):126–136.

Hsia, R. Y., Sarkar, N., and Shen, Y.-C. (2017). Impact of ambulance diversion: black patients with acute myocardial infarction had higher mortality than whites. *Health Affairs*, 36(6):1070–1077.

Institute of Medicine (2003). *Unequal Treatment: Confronting Racial and Ethnic Disparities in Health Care*. National Academies Press (US), Washington (DC).

Jha, A. K., Orav, E. J., Li, Z., and Epstein, A. M. (2007). The inverse relationship between mortality rates and performance in the hospital quality alliance measures. *Health affairs*, 26(4):1104–1110.

Johnson, T. J., Hickey, R. W., Switzer, G. E., Miller, E., Winger, D. G., Nguyen, M., Saladino, R. A., and Hausmann, L. R. M. (2016). The Impact of Cognitive Stressors in the Emergency Department on Physician Implicit Racial Bias. *Academic emergency medicine*, 23(3):297–305.

Joynt, K. E., Orav, E. J., and Jha, A. K. (2011). Thirty-day readmission rates for medicare beneficiaries by race and site of care. *JAMA*, 305(1):675–681.

Kadri, S. S., Sun, J., Lawandi, A., Strich, J. R., Busch, L. M., Keller, M., Babiker, A., Yek, C., Malik, S., Krack, J., et al. (2021). Association between caseload surge and covid-19 survival in 558 us hospitals, march to august 2020. *Annals of Internal Medicine*, 174(9):1240–1251.

KC, D. S. and Terwiesch, C. (2012). An econometric analysis of patient flows in the cardiac intensive care unit. *Manufacturing Service Operations Management*, 14(1):50–65.

Kim, C. (2010). Decomposing the change in the wage gap between white and black men over time, 1980-2005: An extension of the blinder-oaxaca decomposition method. *Sociological methods & research*, 38(4):619–651.

Kim, S.-H., Chan, C. W., Olivares, M., and Escobar, G. (2014). Icu admission control: An empirical study of capacity allocation and its implication for patient outcomes. *Management Science*, 61(1):19–38.

Kitagawa, E. M. (1955). Components of a Difference Between Two Rates*. *Journal of the American Statistical Association*, 50(272):1168–1194.

Kohn, R., Harhay, M. O., Weissman, G. E., Anesi, G. L., Bayes, B., Greysen, S. R., Ratcliffe, S. J., Halpern, S. D., and Kerlin, M. P. (2019). Ward Capacity Strain: A Novel Predictor of Delays in Intensive Care Unit Survivor Throughput. *Annals of the American Thoracic Society*, 16(3):387–390.

Kröger, H. and Hartmann, J. (2021). Extending the kitagawa–oaxaca–blinder decomposition approach to panel data. *The Stata Journal*, 21(2):360–410.

Lavizzo-Mourey, R. J., Besser, R. E., and Williams, D. R. (2021). Understanding and Mitigating Health Inequities — Past, Current, and Future Directions. *New England Journal of Medicine*, 384(18):1681–1684.

Lu, F. Q., Hanchate, A. D., and Paasche-Orlow, M. K. (2021). Racial/ethnic disparities in emer-

gency department wait times in the united states, 2013–2017. *The American Journal of Emergency Medicine*, 47:138–144.

Marks, M. and Choi, M. K. (2019). Baby boomlets and baby health: Hospital crowdedness, hospital spending, and infant health. *American Journal of Health Economics*, 5(3):376–406.

Mateo, C. M. and Williams, D. R. (2021). Racism: a fundamental driver of racial disparities in health-care quality. *Nature Reviews Disease Primers*, 7(1):1–2.

Miller, S., Wherry, L. R., and Mazumder, B. (2021). Estimated mortality increases during the covid-19 pandemic by socioeconomic status, race, and ethnicity: Study examines covid-19 mortality by socioeconomic status, race, and ethnicity. *Health Affairs*, 40(8):1252–1260.

Möller, S., Bliddal, M., and Rubin, K. H. (2021). Methodical considerations on adjusting for charlson comorbidity index in epidemiological studies. *European Journal of Epidemiology*, 36(11):1123–1128.

Moore, B. J., White, S., Washington, R., Coenen, N., and Elixhauser, A. (2017). Identifying increased risk of readmission and in-hospital mortality using hospital administrative data. *Medical care*, 55(7):698–705.

Mullainathan, S. and Obermeyer, Z. (2022). Diagnosing physician error: A machine learning approach to low-value health care. *The Quarterly Journal of Economics*, 137(2):679–727.

Neprash, H. T., Everhart, A., McAlpine, D., Smith, L. B., Sheridan, B., and Cross, D. A. (2021). Measuring primary care exam length using electronic health record data. *Medical care*, 59(1):62–66.

Oaxaca, R. (1973). Male-Female Wage Differentials in Urban Labor Markets. *International Economic Review*, 14(3):693–709. Publisher: [Economics Department of the University of Pennsylvania, Wiley, Institute of Social and Economic Research, Osaka University].

Obermeyer, Z., Nissan, R., Stern, M., Eaneff, S., Bembeneck, E. J., and Mullainathan, S. (2021). Algorithmic bias playbook. *Center for Applied AI at Chicago Booth*.

Obermeyer, Z., Powers, B., Vogeli, C., and Mullainathan, S. (2019). Dissecting racial bias in an algorithm used to manage the health of populations. *Science (New York, N.Y.)*, 366(6464):447–453.

Oster, E. (2019). Unobservable selection and coefficient stability: Theory and evidence. *Journal of Business & Economic Statistics*, 37(2):187–204.

Pei, Z., Pischke, J.-S., and Schwandt, H. (2019). Poorly measured confounders are more useful on the left than on the right. *Journal of Business & Economic Statistics*, 37(2):205–216.

Plunkett, P. K., Byrne, D. G., Breslin, T., Bennett, K., and Silke, B. (2011). Increasing wait times predict increasing mortality for emergency medical admissions. *European Journal of Emergency Medicine*, 18(4):192–196.

Ross, J. S., Cha, S. S., Epstein, A. J., Wang, Y., Bradley, E. H., Herrin, J., Lichtman, J. H., Normand, S.-L. T., Masoudi, F. A., and Krumholz, H. M. (2007). Quality Of Care For Acute Myocardial Infarction At Urban Safety-Net Hospitals. *Health Affairs*, 26(1):238–248.

Rossman, H., Meir, T., Somer, J., Shilo, S., Gutman, R., Ben Arie, A., Segal, E., Shalit, U., and Gorfine, M. (2021). Hospital load and increased COVID-19 related mortality in Israel. *Nature Communications*, 12(1).

Rozier, M. D., Patel, K. K., and Cross, D. A. (2021). Electronic health records as biased tools or tools against bias: A conceptual model. *The Milbank Quarterly*.

Saluja, B. and Bryant, Z. (2021). How implicit bias contributes to racial disparities in maternal morbidity and mortality in the united states. *Journal of Women's Health*, 30(2):270–273.

Schrader, C. D. and Lewis, L. M. (2013). Racial disparity in emergency department triage. *The Journal of emergency medicine*, 44(2):511–518.

Schulman, K. A., Berlin, J. A., Harless, W., Kerner, J. F., Sistrunk, S., Gersh, B. J., Dube, R., Taleghani, C. K., Burke, J. E., Williams, S., et al. (1999). The effect of race and sex on physicians' recommendations for cardiac catheterization. *New England Journal of Medicine*, 340(8):618–626.

Song, H., Tucker, A. L., Graue, R., Moravick, S., and Yang, J. J. (2020). Capacity Pooling in Hospitals: The Hidden Consequences of Off-Service Placement. *Management Science*, 66(9):3825–3842. Publisher: INFORMS.

Song, Z., Zhang, X., Patterson, L. J., Barnes, C. L., and Haas, D. A. (2021). Racial and ethnic disparities in hospitalization outcomes among medicare beneficiaries during the covid-19 pandemic. 2(12):e214223–e214223.

Stepanikova, I. and Cook, K. S. (2008). Effects of poverty and lack of insurance on perceptions of racial and ethnic bias in health care. *Health services research*, 43(3):915–930.

Sun, M. J., Oliwa, T., Peek, M. E., and Tung, E. L. (2022). Negative patient descriptors: Documenting racial bias in the electronic health record. epub ahead of print.

Trivedi, A. N., Zaslavsky, A. M., Schneider, E. C., and Ayanian, J. Z. (2005). Trends in the Quality of Care and Racial Disparities in Medicare Managed Care. *New England Journal of Medicine*, 353(7):692–700.

Vela, M. B., Erondu, A. I., Smith, N. A., Peek, M. E., Woodruff, J. N., and Chin, M. H. (2022). Eliminating explicit and implicit biases in health care: evidence and research needs. *Annual review of public health*, 43:477.

Weissman, G. E., Crane-Droesch, A., Chivers, C., Luong, T., Hanish, A., Levy, M. Z., Lubken, J., Becker, M., Draugelis, M. E., Anesi, G. L., et al. (2020). Locally informed simulation to predict hospital capacity needs during the covid-19 pandemic. *Annals of internal medicine*, 173(1):21–28.

Wellington, A. J. (1993). Changes in the male/female wage gap, 1976-85. *Journal of Human resources*, pages 383–411.

Wilcox, M. E., Harrison, D. A., Patel, A., and Rowan, K. M. (2020). Higher ICU Capacity Strain Is Associated With Increased Acute Mortality in Closed ICUs. *Critical Care Medicine*, 48(5):709–716.

Williams, D. R. and Mohammed, S. A. (2009). Discrimination and racial disparities in health: evidence and needed research. *Journal of Behavioral Medicine*, 32(1):20–47.

Wiltshire, J., Cronin, K., Sarto, G. E., and Brown, R. (2006). Self-advocacy during the medical encounter: use of health information and racial/ethnic differences. *Medical Care*, 44(2):100–109.
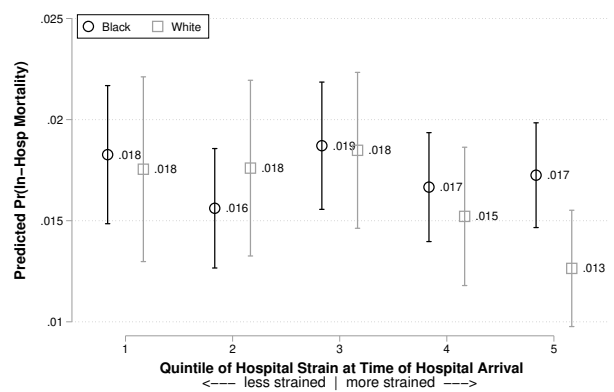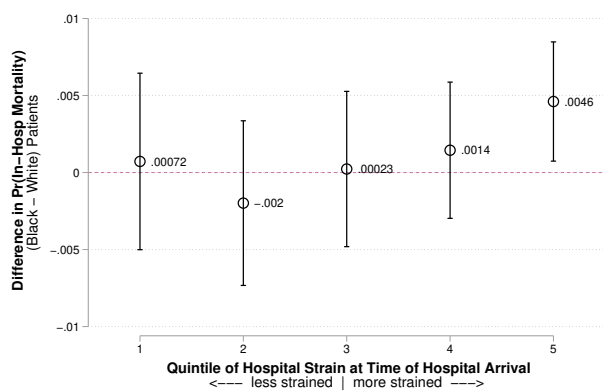
(b)

FIGURE I: Racial gap in in-hospital mortality by hospital strain

(a) Difference in in-hospital mortality between Black and White patients. (b) Predicted probabilities of in-hospital mortality for Black and White patients. Hospital strain at time of patient arrival to the hospital is used as the exposure. Patient covariates include age, age squared, sex, Elixhauser readmission index, Elixhauser mortality index, number of Elixhauser comorbidities, and insurance status. Hospital-year and physician-of-record fixed effects are included. 95% robust standard errors are presented.
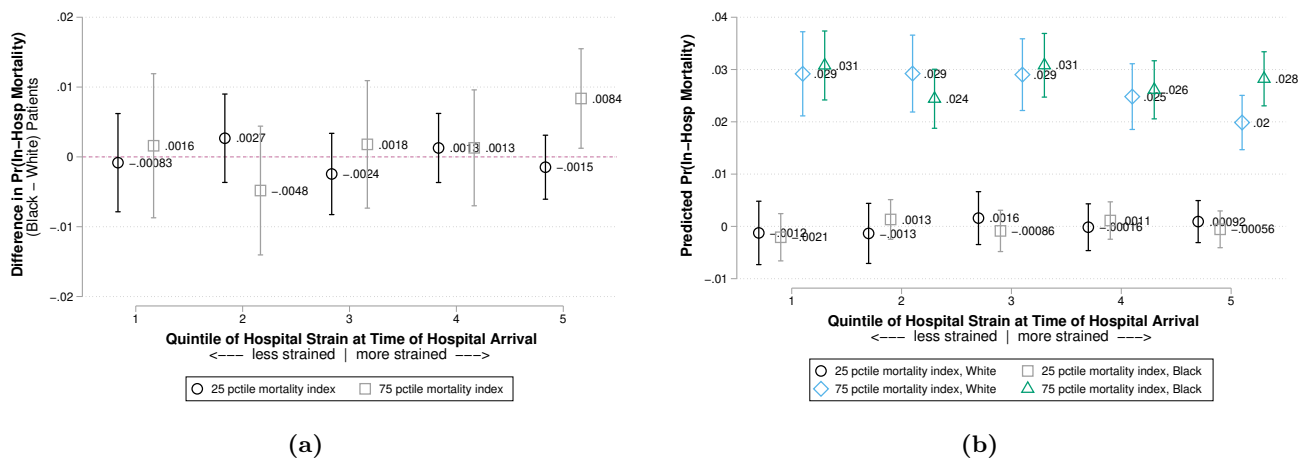
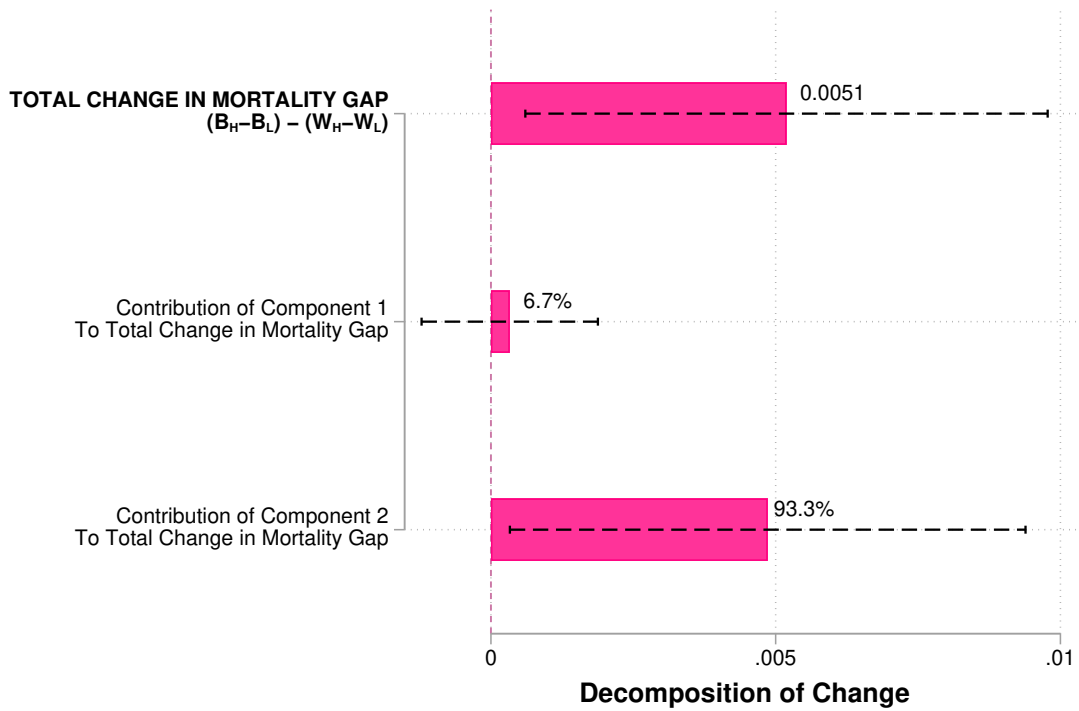**FIGURE II**: Racial gap in in-hospital mortality by hospital strain –
decomposed by ex-ante mortality risk based on comorbidities

(a) Difference in in-hospital mortality between Black and White patients by mortality risk. (b) Predicted probability of in-hospital mortality for Black and White patients by mortality risk. Mortality risk is measured using the 25th and 75th percentile of overall Elixhauser mortality index. Hospital strain at time of patient arrival to the hospital is used 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.
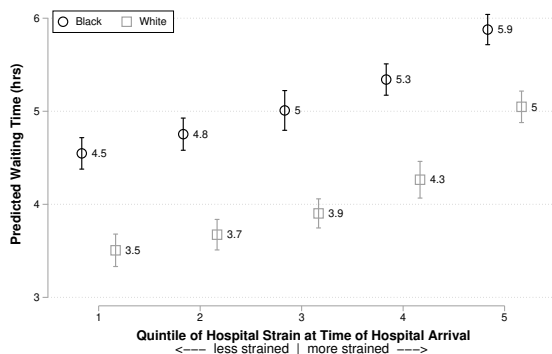
**(a)** Predicted wait times – overall
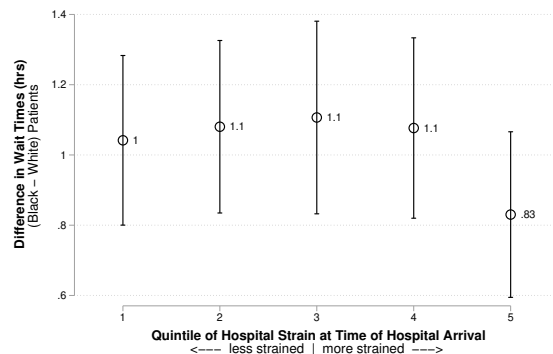
(b) Difference in wait times – overall
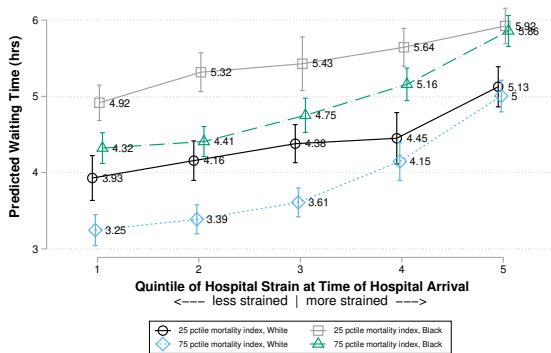
(c) Predicted wait times – by mortality risk
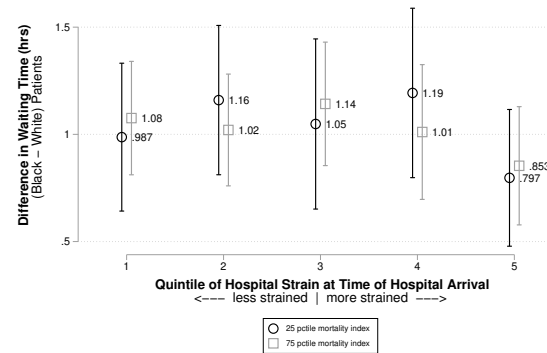
(d) Difference in wait times – by mortality risk

FIGURE III

Inputs to Care: Wait Time for Hospital Bed

Subfigure (a) plots the predicted wait times for Black and White patients, and subfigure (b) plots the difference in wait times between Black and White patients. Subfigures (c) and (d) presents these estimates decomposed by the 25th and 75th percentile of Elixhauser mortality index. Hospital strain at time of patient arrival is used 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.
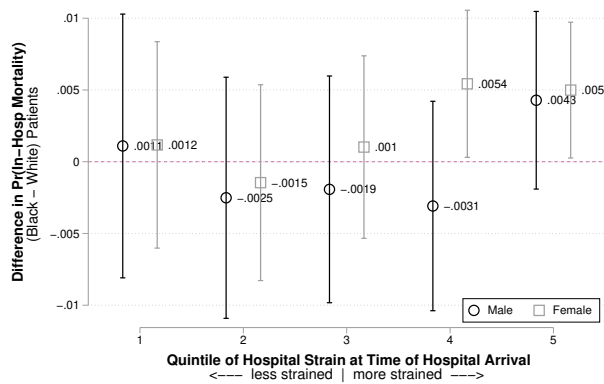
(a) Decomposition by gender
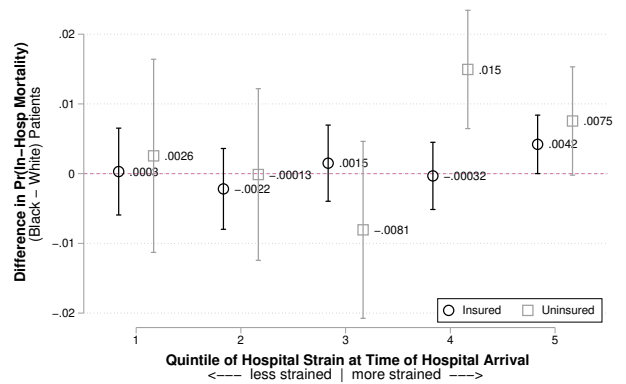
(b) Decomposition by insurance status

FIGURE IV

Racial gap in in-hospital mortality decomposed by (a) gender and (b) insurance status

This figure plots the difference in in-hospital mortality between Black and White patients decomposed by (a) gender and (b) insurance status. Patient covariates include age, age squared, sex, Elixhauser readmission index, Elixhauser mortality 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 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, Elixhauser mortality index, number of Elixhauser comorbidities), 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 of patient characteristic, care inputs, and hospital factors as well as the constant term for race. "Group 1" is Black patients and "Group 2" is White 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	52.32
	(18.03)	(19.25)
Uninsured	0.10	0.14
	(0.30)	(0.35)
Female	0.49	0.63
	(0.50)	(0.48)
Elixhauser mortality index	13.21	12.78
	(12.98)	(13.41)
Elixhauser 30-day readmission index	23.19	25.18
	(19.43)	(21.74)
# of comorbidities	4.00	4.05
	(2.66)	(2.97)
ICU Admission	0.27	0.22
	(0.45)	(0.42)
In-hospital mortality	0.02	0.02
	(0.13)	(0.12)
Length of ICU stay (days)	3.87	3.60
	(6.87)	(5.58)
Total inpatient length of stay (days)	7.51	6.99
	(9.82)	(12.46)
ED length of stay (hours)	3.77	4.84
	(7.23)	(5.19)
Hospital strain at time of hospital arrival (quintiles)	2.28	2.21
	(1.42)	(1.42)
Observations	22824	30183

Appendix
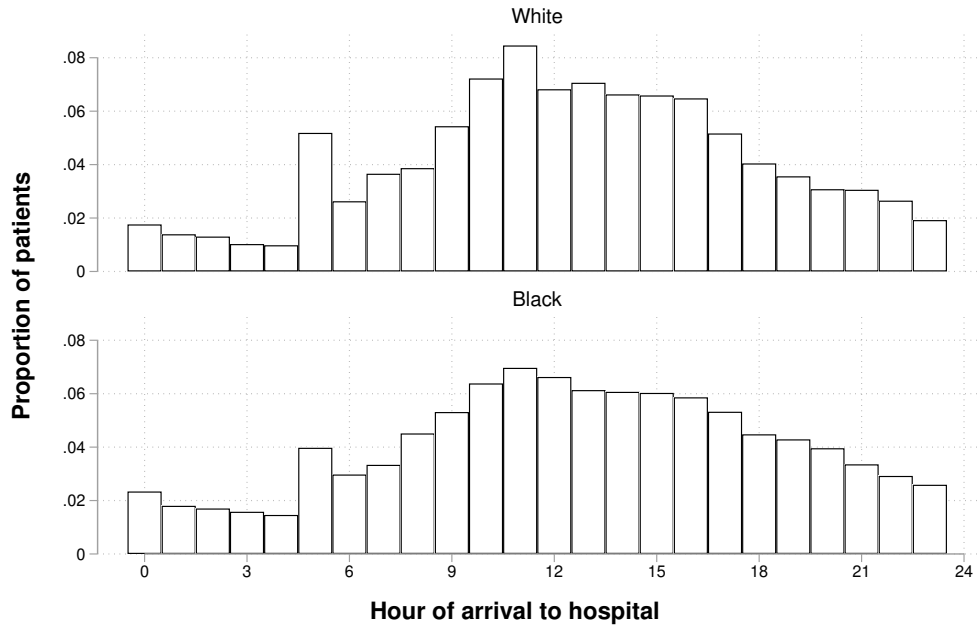
(a) Hour of arrival
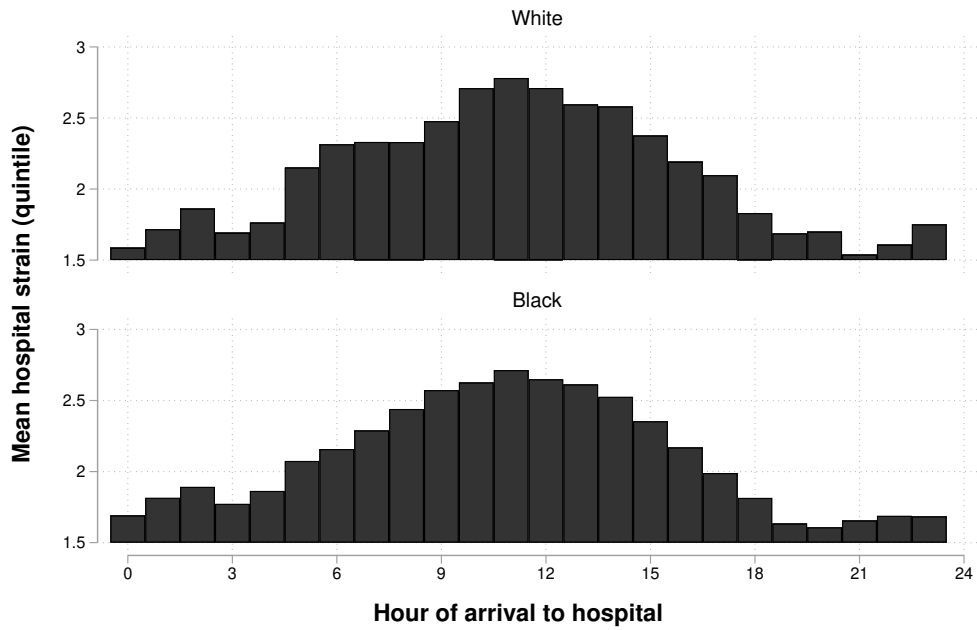
(b) Mean strain at hour of arrival

FIGURE A.1

Distribution by race of (a) hour of arrival and
(b) mean quintile of hospital strain at hour of arrival to hospital

Subfigure (a) presents the distribution of White and Black patients by the hour of their arrival to the hospital. Subfigure (b) presents the mean quintile of hospital strain for White and Black patients by the hour of their arrival to the hospital.
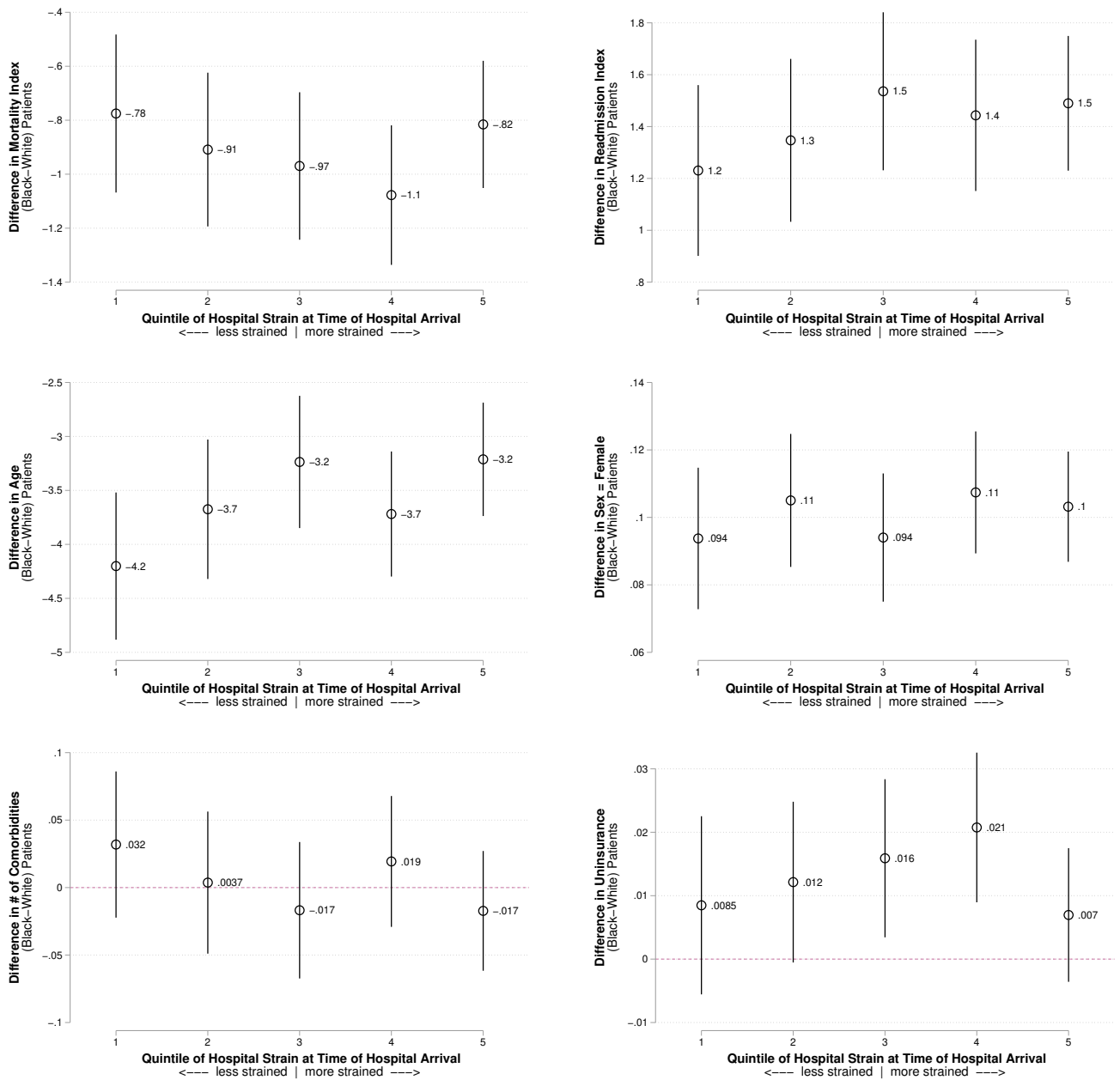
FIGURE A.2

Testing for patient selection using control variables as dependent variables

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 in the main specification are still 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.
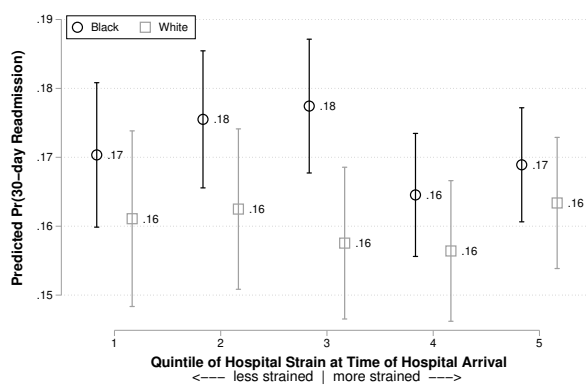
(a) Predicted 30-day readmission
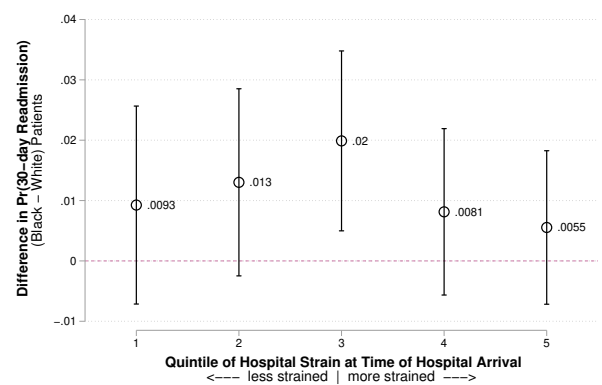
(b) Differences in 30-day readmission

FIGURE A.3

30-day Readmission

This figure plots the (a) predicted 30-day readmission for Black and White patients and (b) difference in 30-day readmission between Black and White patients, using hospital strain at time of patient's hospital arrival as the exposure. Patient covariates include age, age squared, sex, Elixhauser readmission index, Elixhauser mortality index, number of Elixhauser comorbidities, and insurance status. Hospital-year and physician-of-record fixed effects are included. 95% robust standard errors are presented.
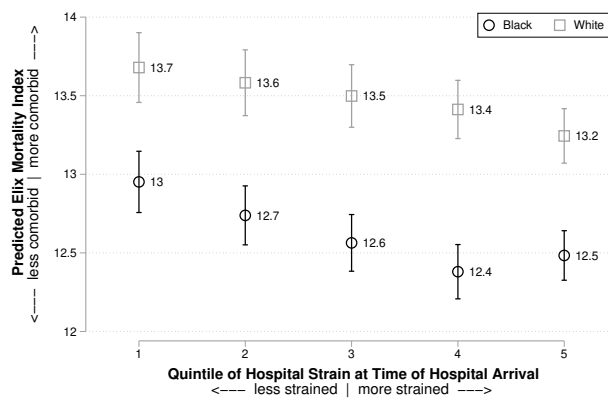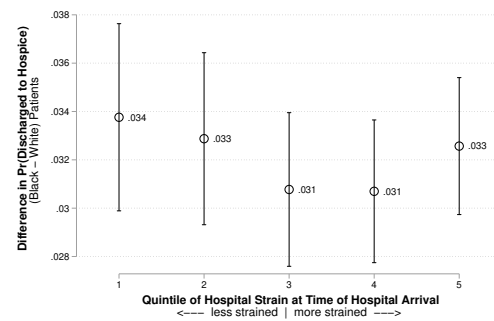
FIGURE A.4

In-hospital mortality using hospital strain at end of inpatient stay

This figure plots the predicted Elixhauser mortality index for Black and White patients using hospital strain at the hour of patient's hospital 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.
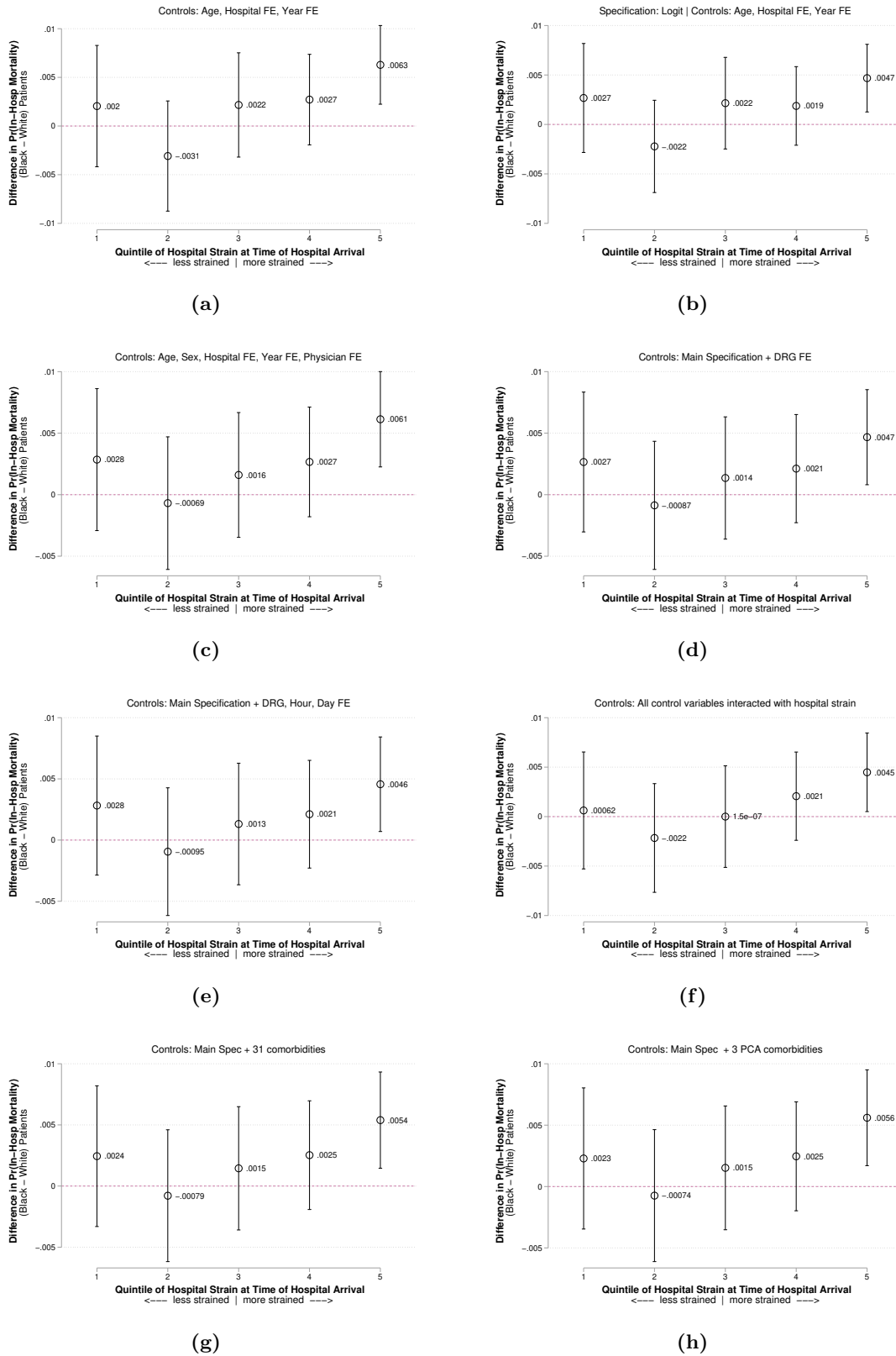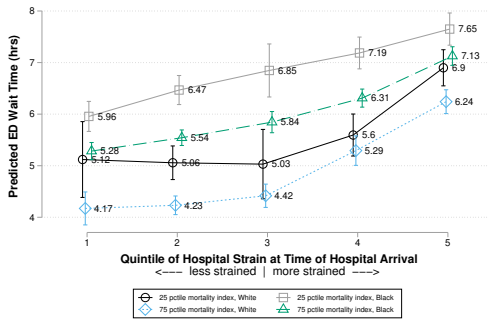
FIGURE A.5

Sensitivity of estimates 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 the exposure, under various combinations of covariates, fixed effects, and specifications. 95% robust standard errors are presented.
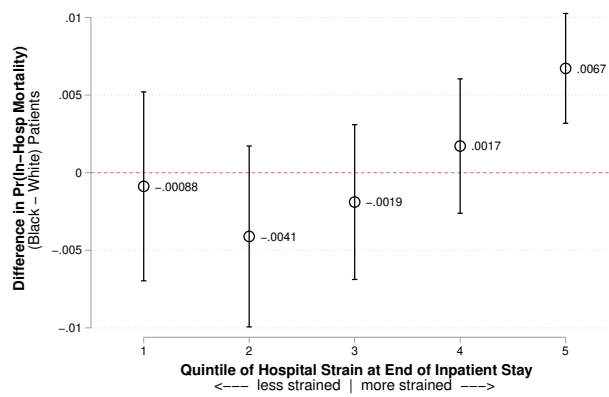
FIGURE A.6

In-hospital mortality using hospital strain at end of inpatient stay

This figure plots the difference in in-hospital mortality between Black and White patients using hospital strain at the hour of the end of the patient's inpatient stay (as opposed to at the hour of patient arrival to the hospital) as the exposure. Patient covariates include age, age squared, sex, Elixhauser mortality and readmission indices, number of Elixhauser comorbidities, and insurance status. Hospital-year and physician-of-record fixed effects are included. 95% robust standard errors are presented.
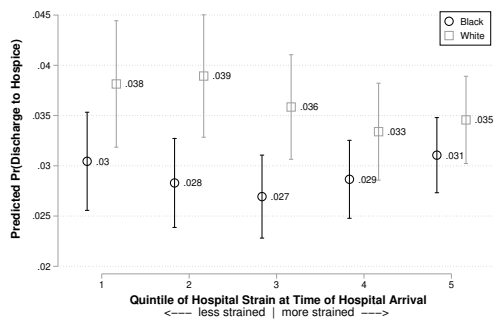
(a) Predicted values   **(b)** Differences by race
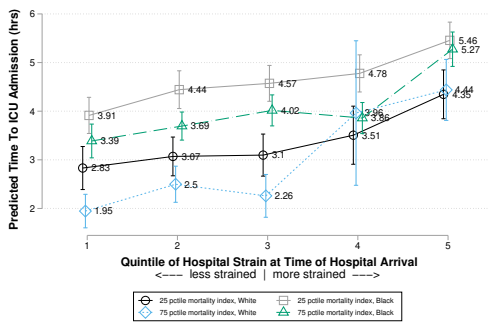
FIGURE A.7

Discharge to hospice

Sufbigure (a) plots the predicted probabilities of discharge to hospice for Black and White patients, and subfigure (b) plots the difference in probabilities of discharge to hospice 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, Elixhauser mortality index, number of Elixhauser comorbidities, and insurance status. Hospital-year and physician-of-record fixed effects are included. 95% robust standard errors are presented.
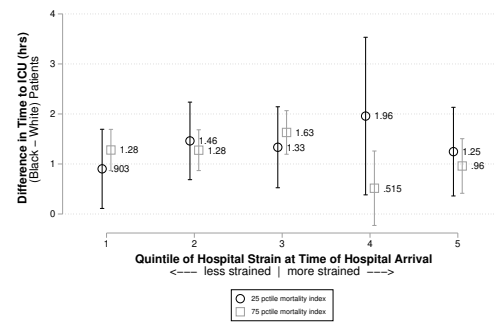
(a) Time in the ED: Predicted Values
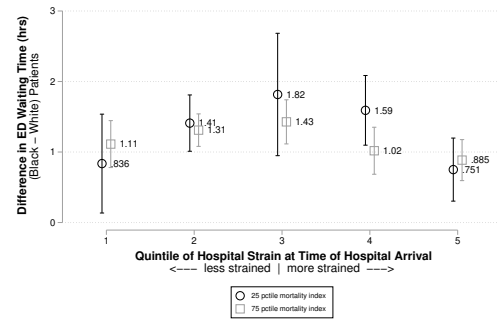
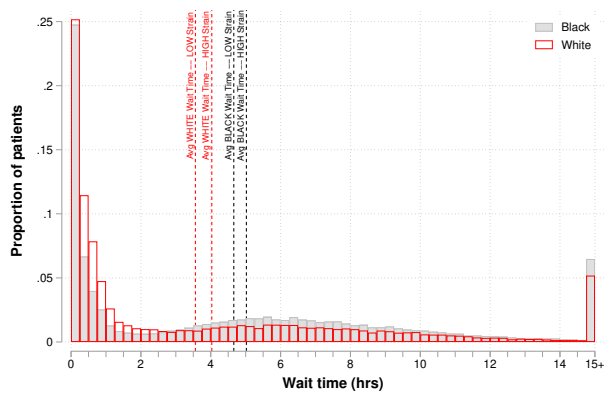**(b)** Time in ED: Racial Differences

(c) Time to ICU Admission: Predicted values

(d) Time to ICU Admission: Racial Differences

FIGURE A.8
Inputs to Care: Wait Time in the ED/ to the ICU

Subfigure (a) plots the predicted wait time in the ED (for those admitted through the ED) for Black and White patients, and subfigure (b) plots the difference in wait times in the ED between Black and White patients, at the 25th and 75th percentile of overall Elixhauser mortality index. Subfigure (c) plots the predicted wait time for ICU admission (for those who were directly admitted to the ICU) for Black and White patients, and subfigure (d) plots the difference in wait times for ICU admission between Black and White patients, at the 25th and 75th percentile of overall Elixhauser mortality index. Hospital strain at time of patient arrival is used 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) Density of wait times
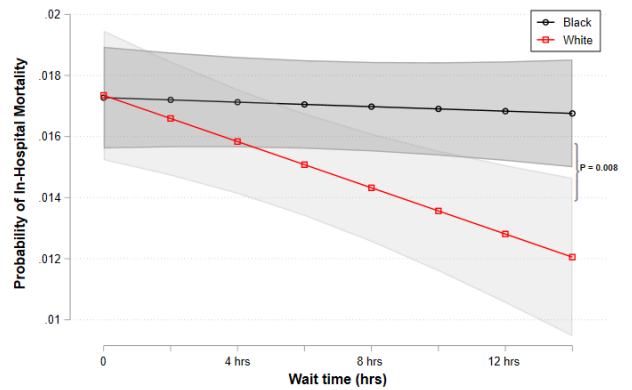
(b) Wait times and mortality
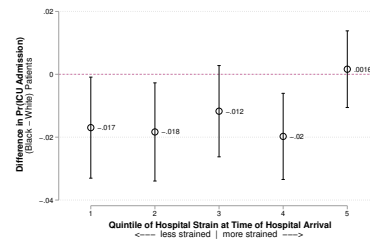
FIGURE A.9

Racial differences in the relationship between wait times and in-hospital mortality

Subfigure (a) plots the distribution of wait times for Black and White patients. Subfigure (b) plots the predicted probability of in-hospital mortality for Black and White patients using wait times as the exposure, (with the *p*-value of the difference in effects overlaid on the graph). Patient covariates in subfigure (b) include age, age squared, sex, Elixhauser readmission index, Elixhauser mortality index, number of Elixhauser comorbidities, and insurance status. Hospital-year and physician-of-record fixed effects are included. 95% robust standard errors are presented.
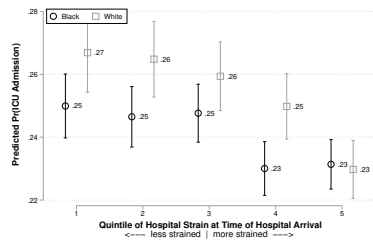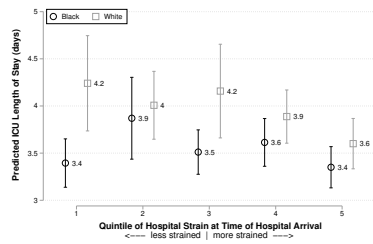
(a) Predicted ICU admission

(b) Difference in ICU admission

(c) Predicted ICU length of stay

(d) Difference in ICU length of stay

(e) Predicted length of stay

(f) Difference in length of stay
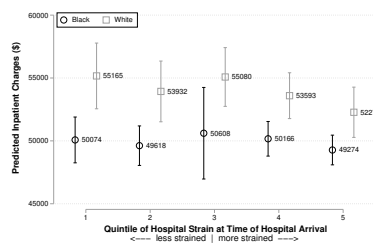
(g) Predicted inpatient charges
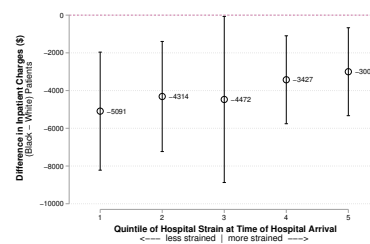
(h) Difference in inpatient charges

FIGURE A.10

Racial gap in inputs to care by hospital strain

This figure plots the predicted inputs to care for, and the difference in inputs to care between, Black and White patients. Inputs to care examined are: (a,b) ICU admission, (c,d) ICU length of stay, (e,f) inpatient length of stay, and (g,h) inpatient charges. 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.
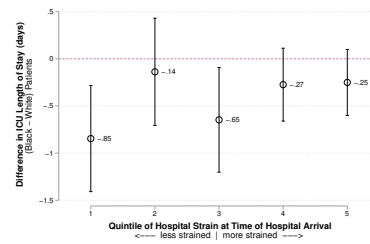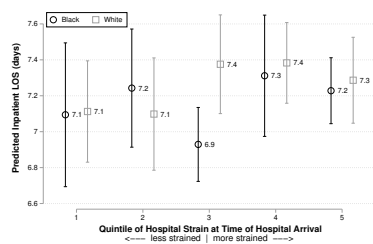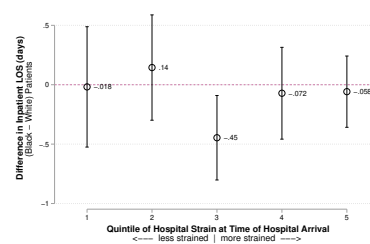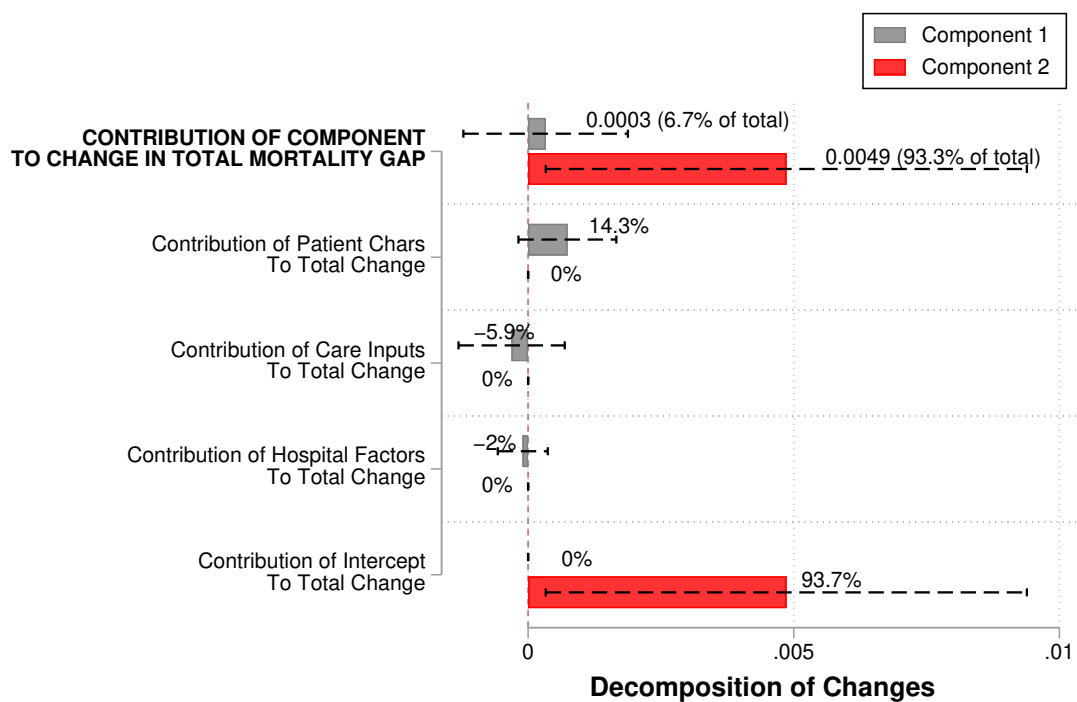
FIGURE A.11
Detailed Wellington Decomposition

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, Elixhauser mortality index, number of Elixhauser comorbidities), 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 of patient characteristic, care inputs, and hospital factors as well as the constant term for race. "Group 1" is Black patients and "Group 2" is White 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.12

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 (taken directly from Kröger and Hartmann (2021)): (i) Intercept Effect: 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. "Group 1" is Black patients and "Group 2" is White 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).
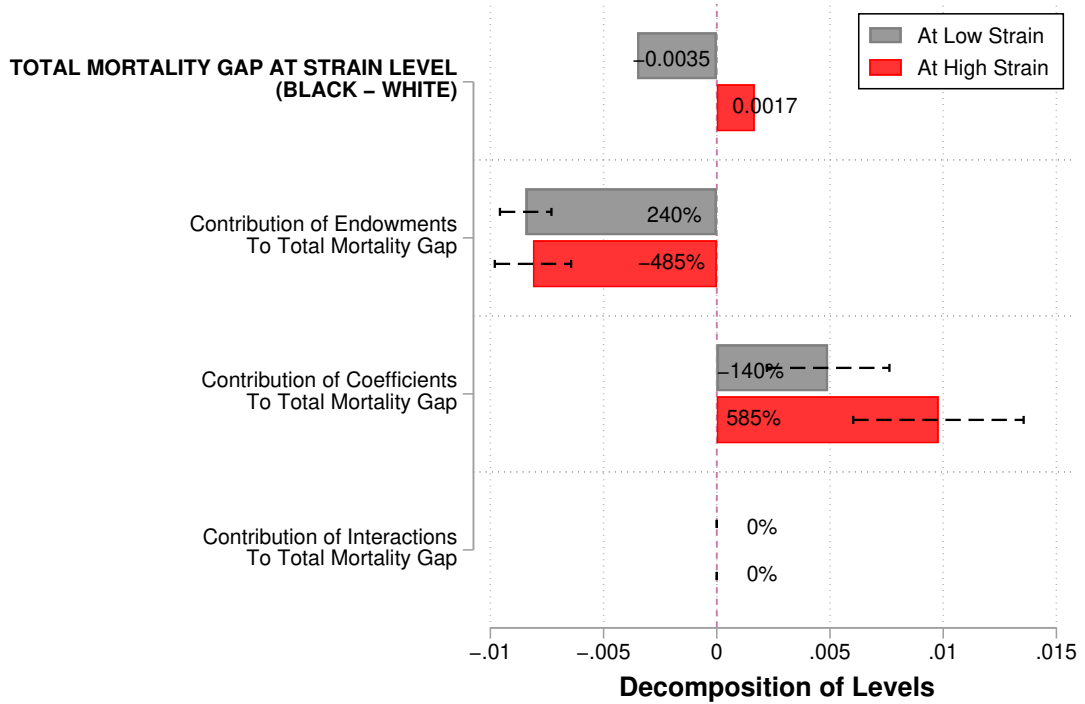
FIGURE A.13
Static Kitagawa-Oaxaca-Blinder Decomposition
of the Racial Gap in Hospital Mortality
at Low and High Strain

KOB decomposes the racial gap in hospital mortality at high strain into three components: (i) the "endowments": how much would hospital mortality for White patients change if White 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 Black patients; (ii) the "coefficients": how much would hospital mortality for White patients change if White patients had the same mapping of patient characteristics/inputs to care to hospital mortality as Black patients; and (iii) the "interaction": how much would hospital mortality for White patients change if White patients had the same levels and the same mapping of patient characteristics/care inputs to hospital mortality as Black patients. "Group 1" is Black patients and "Group 2" is White 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.

TABLE A.1

Black and White patient characteristics by low and high strain

	White		Black		
	Low Strain	High Strain	Low Strain	High Strain	$p < 0.05$?
Age (yrs.)	59.06	59.34	52.07	53.07	No
	(18.17)	(17.66)	(19.33)	(18.98)	
Uninsured	0.10	0.10	0.15	0.13	No
	(0.31)	(0.30)	(0.36)	(0.34)	
Female	0.49	0.48	0.63	0.63	No
	(0.50)	(0.50)	(0.48)	(0.48)	
Elixhauser mortality index	13.34	12.86	12.77	12.79	No
	(12.99)	(12.95)	(13.35)	(13.58)	
Elixhauser readmission index	23.15	23.32	25.00	25.70	No
	(19.45)	(19.39)	(21.65)	(21.98)	
# of comorbidities	3.97	4.06	4.02	4.16	No
	(2.66)	(2.65)	(2.96)	(2.99)	
Observations	16570	6254	22463	7720	

This table shows the unadjusted differences in patient characteristics between Black and White patients at low and high strain. The last column indicates whether the $p-value$ on the interaction term (from regressing the patient characteristic on $Black$, $HighStrain$, and an interaction between the two) is statistically significant at the 5% level.

TABLE A.2

Proportion Full at Each Quintile of Strain At Each Hospital

	Quintile 1		Quintile 2		Quintile 3		Quintile 4		Quintile 5	
	H1	H2	H1	H2	H1	H2	H1	H2	H1	H2
Proportion Full	0.70	0.73	0.80	0.85	0.83	0.88	0.86	0.91	0.91	0.94
	(0.10)	(0.14)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.02)	(0.02)
Observations	4183	4307	4694	4625	4913	5079	5774	5458	6691	7283

For hospitals H1 and H2, this table presents the mean proportion of beds filled at each of the five quintiles of strain, with SD in parentheses.

TABLE A.3

Predictive power of Elixhauser measures for Black and White Patients

	(1)	(2)
	Pr(In-Hospital Mortality)	
	White	Black
# of comorbidities	0.00674***	0.00564***
	(0.000712)	(0.000724)
Elixhauser mortality index	0.00199***	0.00219***
	(0.000179)	(0.000144)
Elixhauser readmission index	-0.000987***	-0.000804***
	(0.000133)	(0.000114)
N	22822	30182
r2	0.0331	0.0443
FE	H-Y	H-Y

Standard errors in parentheses

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

This table presents estimates from regressing in-hospital mortality on the three Elixhauser measures: number of comorbidities, mortality index, and readmission index separately for Black and White patients. All patient covariates used in the primary specification are used, along with hospital-year fixed effects.

TABLE A.4

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)
High strain	-0.00437**
	(0.00175)
Black X High strain	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
r2	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.

TABLE A.5

Oster Test for Selection on Unobservables

| | (Main Model) | | | | |
	(1)	(2)	(3)	(4)	(5)
Estimate (β)	0.00441	0.00417	0.00364	0	0
Coefficient of Proportional Selection (δ)	-	1	1	16.51	5.1
R_{max}	$R_{adj} = 0.187$	$1.3 * R_{adj}$	$2 * R_{adj}$	$1.3 * R_{adj}$	$2 * R_{adj}$

This tables presents the results from the (Oster, 2019) Pscalc option in STATA. Col (1) presents the regression estimate (β, the interaction on $HighStrain*Black$) from our main model. Cols (2) and (3) present the same regression coefficient under the assumption of equal proportional selection (i.e., $\delta = 1$). Cols (4) and (5) present the degree of proportional selection (δ) necessary to give an effect size of 0.

TABLE A.6

Contribution of Care Inputs to Racial Mortality Gap at High Strain

	Dep Var: In-Hospital Mortality					
	(1: Main Model)	(2)	(3)	(4)	(5)	(6)
Black X Highest level of hospital strain	0.00441**	0.00350	0.00420*	0.00405*	0.00480**	0.00311
	(0.00224)	(0.00224)	(0.00223)	(0.00224)	(0.00225)	(0.00223)
N	52880	52337	52880	52861	52880	52337
r2	0.187	0.190	0.189	0.188	0.188	0.197
Care inputs included in model	None	Wait Time	ICU Admission	Charges	Total LOS	All

Standard errors in parentheses

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

This table presents our primary estimate of interest, i.e., the interaction between Black and highest quintile of strain, after accounting for the differential effect by race of various care inputs on patient in-hospital mortality. Col (1) presents estimates from our main model. Cols 2- 5 add the following care inputs respectively into our main model: wait time, ICU admission, charges, and total inaptient length of stay. Column 6 includes all 3 care inputs. Models 2-6 include our main specification with two additional terms on the RHS: care input, and race interacted with care input.