Health Care Inequality: Evidence of Differences in the Treatment of Whites and Blacks With Atrial Fibrillation

Kyle Dougher

Department of Economics
Professor William Evans

April 27, 2015

Abstract

Inequalities in health care and health outcomes have become a prominent concern for policy makers in the past several decades. Despite substantial efforts to eliminate these inequalities, recent literature suggests that blacks are still significantly less likely to receive proven treatments for numerous diseases even after controlling for patient, hospital, and physician characteristics. This paper examines whether there are treatment disparities between whites and blacks with atrial fibrillation. By controlling for hospital and physician fixed effects, I examine whether whites and blacks seeing the same physician at the same hospital receive different treatments. The findings indicate that disparities exist for whites and blacks even within the same hospital. However, these differences disappear when controlling for physician. This result suggests that blacks and whites are being sorted to different quality physicians within the hospitals from which they receive care.

Racial inequality in health and health care has been a well-documented and enduring concern in the United States. In 1984, the US Department of Health and Human Services published a report entitled *Health, United States 1983* that demonstrated the major disparities that existed in "the burden of death and illness experienced by blacks and other minority Americans" despite significant progress in the overall health of the nation. Since then, although health has continued to improve for all racial groups, inequity between groups has persisted. Almost three decades later, the Center for Disease Control (CDC) reports that blacks still fare worse than whites in terms of life expectancy, death rates, and infant mortality. In addition, blacks have higher mortality rates in nearly every major disease category including heart disease, cancer, stroke, diabetes, and respiratory diseases (CDC Health Disparities and Inequalities Report).

Increased efforts from both public and private entities have been made to address this persistent inequality. In 1998, President Clinton committed to a national goal of eliminating racial health disparities in six key areas by the year 2010. This initiative, named Healthy People 2000, targeted areas of health status including cardiovascular disease, diabetes, HIV/AIDS, immunizations, infant mortality, and cancer screening. To support the initiative, congress appropriated hundreds of millions of dollars to fund community interventions addressing health disparities (Erasing the Color Line 2003). Funding for similar interventions continued throughout the subsequent decade and was expanded under the Obama administration through projects such as the Racial and Ethnic Approaches to Community Health as well as the Health and Human Services (HHS) Action Plan to Reduce Racial and Ethnic Health Disparities (Ross). In addition, the Affordable Care Act includes numerous provisions explicitly designed to reduce racial health disparities. These provisions target racial data collection, diversity among health care practitioners, and cultural competence education, all intended to reduce racial gaps in the quality of health (Andrulis et al. 2010). Supporting these public efforts, private institutions such as the Robert Wood

Johnson Foundation, Kaiser Family Foundation, and the Commonwealth Fund have also begun initiatives to address racial disparities in health care.

Although racial health inequality has risen to the forefront in both public and private discussion, between 1990 and 2005 there has been a statistically significant increase in racial differences for 6 major health indicators including heart disease mortality and diabetes mortality (Orsi et al. 2010). Perhaps more disconcerting is the large number of studies that provide evidence of racial disparities in the receipt of proven treatments for a wide range of disorders. For example, Sonel et al. (2005) suggest that black patients who suffered from a myocardial infarction are significantly less likely than whites to receive life-saving coronary interventions such as cardiac catheterization, revascularization procedures, and statin therapy even after controlling for hospital, demographic, and patient characteristics. However, subsequent analysis using the same population found that controlling for hospital fixed effects narrowed or erased some of the differences in treatment (Barnato et al. 2005). Other analyses have found similar disparities in the receipt and outcomes of other surgical procedures. Notably, blacks have higher surgical mortality rates for a wide range of surgical procedures such as coronary artery bypass graft (CABG) and pancreatic resection even after controlling for hospital fixed effects (Lucas et al. 2006). Further, blacks were far more likely to undergo lower-extremity amputations rather than potentially limb-saving revascularization for lower-extremity arterial disease (Regenbogen et al. 2009).

These findings suggest that differences in the quality of hospitals that primarily serve blacks explain some of the gap in treatments and outcomes. In particular, care for black patients is concentrated among a small group of providers who have fewer resources and less capacity to provide high quality care. Nonetheless, provider-level segregation does not fully explain disparities observed in the studies above.

In this paper I seek to determine whether physician-level differences can explain the residual gap in treatments received. In particular, I compare inpatient[1] procedures administered to atrial fibrillation (AF) patients with Medicare who saw the same physician within the same hospital. To my knowledge, this paper is the first to use physician fixed effects in the context of AF treatment and one of the few papers to use physician fixed effects in examining treatment disparities for any disorder.

There are two main findings in this paper. The first is that the addition of hospital fixed effects explains little of the variation in AF treatments for whites and blacks. This finding is contrary to prior literature that suggests the discrepancies in treatments received are largely attributable to differences in hospital quality. The second finding is that controlling for physician and hospital fixed effects eliminates all evidence of treatment disparity. This finding is suggestive that within the same hospital blacks tend to see lower quality physicians on average. Thus, differences in physician quality are driving the disparity in treatments and outcomes.

The paper proceeds as follows. Section I provides background on AF and discusses the prior evidence of racial treatment disparities for AF patients. Sections II and III discuss the data and methods used in the analysis. Section IV presents the regression results. Section V discusses the implications of the findings and presents three hypotheses that could explain how blacks are sorted to different physicians. Finally, section VI discusses the limitations of the paper and concludes with implications for policymakers.

I. Background

AF is an irregular and typically rapid heartbeat during which the heart's upper two chambers (the atria) beat out of sync with the lower two chambers (the ventricles). As a result of this

[1] Inpatients are patients who are admitted to the hospital at least overnight.

irregularity, blood no longer pumps efficiently through the body causing shortness of breath, weakness, and blood clots. While AF is not usually life-threatening, patients with AF face significantly increased risk of thromboembolic stroke, ischemic stroke, and heart failure (Go et al. 2001). Further, AF causes a 4-5 fold increase in the risk of stroke and doubles the risk of heart-related deaths. An estimated 2 million individuals in the United States over the age of 65 are afflicted by the condition making AF the most common sustained arrhythmia. In the population of patients 65 and older admitted to hospitals, AF is the 5th most common primary diagnosis (The American Heart Association).

A. Description of AF and Treatments

Diagnostic tests for AF are relatively simple and painless, often involving a stress electrocardiogram (EKG) or an echocardiogram (echo). EKGs measure the heart's electrical efficiency and indicate whether the pattern of the heartbeat is irregular but do not accurately evaluate the pumping ability of the heart. An echo is an ultrasound that shows how blood flows through the heart. The echo indicates how well the valves are functioning and the speed of blood flow.

Once diagnosed, there are two broad goals of AF treatment: the prevention of circulatory instability and the prevention of stroke. The former can be accomplished through rate or rhythm control while the latter is achieved using anticoagulants such as heparin or warfarin (Prystowsky 2000).

Initial therapy for atrial fibrillation is typically intended to maintain sinus rhythm (normal heartbeat) through rate or rhythm control. In treating patients with AF, both of these methods of treatment have been shown to have comparable mortality and success rates (Van Gelder et al., Roy et al.) As its name suggests, rate control seeks to lower the patient's heart rate to normal levels by using beta blockers or calcium channel blockers. Beta blockers reduce the effects of epinephrine,

causing the heart to beat more slowly and with less force while calcium channel blockers relax blood vessels and thereby lower blood pressure (Mayo Clinic). On the other hand, rhythm control attempts to restore a normal heart rhythm through cardioversion, defibrillation, or ablation. Cardioversion is typically administered by sending low-energy electric shocks to the patient's heart through electrodes placed on the chest. Several studies suggest that cardioversion has a 90% success rate in restoring sinus rhythm. However, only 30% of those patients maintain normal rhythm a year later (Crandall et al.) Those who do not maintain sinus rhythm may necessitate additional cardioversion procedures. Defibrillation, while similar in application, involves high-energy shocks and is designed to treat very irregular and severe arrhythmias. In cases where rhythm control cannot be maintained by medication or electric shock, catheter ablation may be attempted (Leong-Sit et al. 2010). During the ablation procedure the physician delivers radiofrequency of energy to melt away unwanted tissue between the pulmonary veins and the left atrium. This excess tissue often plays a critical role in causing arrhythmia. Overall success rates for ablation range from 60% to 80% (Shapira 2009).

B. Evidence of Racial Disparities in Existing Literature

The existing literature provides substantial evidence of racial treatment disparities for AF consistent with findings for other diseases discussed above. For example, several studies have examined the impact of race on mortality and the receipt of anticoagulants for patients with AF. The Reasons for Geographic and Racial Differences in Stroke (REGARDS) is a national longitudinal study with oversampling from blacks and individuals from states with high rates of stroke. Using this data, Meschia et al. (2010) provide evidence that blacks are significantly less likely to be aware they have AF. Among those who were aware, the odds of blacks being treated with warfarin were only 25% that of whites. Using a different data set, Thomas et al. (2013) also found disparities in warfarin treatment (OR .76, $P<.001$) and found evidence that blacks had longer hospital stays.

Naderi et al. (2014) used nationally representative inpatient data of patients with a primary diagnosis of AF to examine differences in in-hospital procedures. The authors find blacks are significantly less likely to receive catheter ablation and cardioversion. Additionally, being black is an independent predictor of in-hospital mortality with black males being 90% more likely to die than white males. These estimates were obtained using hospital random effects and controlling for individual level characteristics.

While the papers discussed in the preceding paragraph all control for hospital and individual level characteristics, none include hospital or physician fixed effects. Therefore, one concern of these results is that they are biased due to omitted observable and unobservable characteristics at the physician and hospital levels. Further, these studies look at those diagnosed with AF from all age groups and insurance statuses. Unobserved differences in characteristics for patients in these populations may further bias their results. This paper addresses these limitations through the inclusion of hospital and physician fixed effects and through comparison of a population of Medicare beneficiaries.

II. Data

The data for this paper come from the 2009 Nationwide Inpatient Sample (NIS), a subset of the Healthcare Cost and Utilization Project (HCUP) database generated by the Agency for Healthcare Research and Quality (AHRQ). The NIS contains the largest collection of longitudinal all-payer data on hospital inpatient stays. This data approximates a 20% sample of short-term, non-federal hospitals. The universe of hospitals is stratified by hospital ownership, bed size, teaching status, urban or rural location, and geographic region to provide a national representation of all inpatient discharges in the United States. Although there are more recent NIS datasets available, I use the 2009 NIS for this paper because it is the last year that physician identification numbers were

available. This information is necessary in order to include physician fixed effects used in the analysis.

A. Sample

The 2009 NIS dataset contains 7,810,762 discharges between January 1, 2009 and December 31, 2009. Discharges came from 1,050 hospitals across each of the four geographic regions (129 Northeast, 306 Midwest, 418 South, and 197 West). Forty-six states are represented, capturing 96.1% of the US population. From this sample, I considered only patients with AF as the primary diagnosis. I further excluded patients below the age of 65 as well as patients who did not use Medicare as the primary payer to eliminate differences in healthcare coverage that could confound the results. Additionally, only patients with race listed as white or black were included. However, because some hospitals fail to supply race data, race is missing for 21% of the sample (8,334 observations). Dropping these observations may cause the race-specific estimates to be biased unless the omitted observations are representative of the non-missing observations. T-test comparisons indicate there are no significant differences in patient characteristics between patients with and without race information. Further, with the exception of paralysis, peripheral vascular disorders, and neurological disorders, there are no significant differences in risk factors. Therefore, there is little concern that the omission of observations missing race data will bias the results. The final sample included 40,263 observations with 38,270 white patients and 1,993 black patients. These observations come from 768 hospitals located in 40 different states.

B. Outcomes and Covariates

The outcomes of interest are listed in Table 1 and are segmented into two panels within the table. In panel A, I examine 6 common treatments and procedures for AF discussed in the section I:

echocardiogram, heparin, anticoagulants, ablation, cardioversion, and other shock[2]. I also include a variable that indicates whether the patient received no procedures at all. Additional information on these treatments is presented in Table I.

In panel B, I expand the list of outcomes to include variables that are measurable for any hospital analysis to discover the ways in which medical care received by blacks differs from that of whites. In particular, I examine length of stay, number of procedures, number of diagnoses, whether the patient had a major operating room procedure, total charges, whether the stay was an elective admission, and in-hospital mortality. These variables are largely determined by the physician and including them as controls would capture variation in the receipt of treatments that are attributable to physician differences. Therefore, I omit these variables from the list of controls to better estimate the physicians' full impact on treatments administered.

Table 2 presents the summary statistics for most of the important covariates used in the later analysis. The variables are divided into three categories: demographics, hospital, and risk factors. Examining the demographic characteristics, one can see that blacks came disproportionately from the center of large metro areas. Further, 70% of blacks in the sample live in areas with median income below $50,000. By contrast, whites were evenly distributed among the income quartiles.

Table 2 also reveals disparities in the characteristics of the hospitals from which blacks and whites tend to receive care. For example, blacks are significantly more likely to go to public hospitals and teaching hospitals than whites. Blacks also tended to go to hospitals in urban locations. Prior studies suggest that private, teaching, high volume hospitals typically provide better care than public, non-teaching institutions (Nelson 2002). Thus, while there is evidence of disparities in hospital

[2] Note that rate control therapies are not included as treatments. This omission is due to limitations in the NIS dataset. Rate control drugs are coded as CPT codes rather than as ICD-9 codes that are reported in the NIS. Additionally, EKGs are omitted due to extremely low incidence rate in the sample.

characteristics, it is unclear whether these differences imply that blacks go to hospitals of lower quality.

The final section of Table 2 highlights the substantial variation in the presence of risk factors between blacks and whites. Not only do blacks in the sample have higher mortality risk, they are also significantly more likely to have major or extreme loss of function due to their illness. Of the 27 comorbidities analyzed, blacks were significantly more likely ($p<.05$) to have higher prevalence in 15 of them. In particular, blacks were more likely to have complications with hypertension, congestive heart failure, coagulopathy, deficiency anemias, diabetes, and obesity. To the extent that these comorbidities covary with viable treatment options or patient outcomes, we should expect basic OLS specifications to be biased. Therefore, specifications in which these risk factors are controlled for may more accurately explain treatment differences observed.

III. Methods

A. Estimating Framework

To estimate the impact of being black on the probability of receiving the treatments outlined above, I fit linear probability models (LPM) using various levels of controls. The basic LPM specification takes the following form and serves as the foundation for later specifications:

$$Treatment_{ihp} = \alpha + black_{ihp}\beta + \epsilon_{ihp} \quad (1)$$

where $Treatment_{ihp}$ is an indicator for each of the seven outcomes listed in Table 1 and takes on the value of one if patient *i* at hospital *h* seeing physician *p* received that treatment. β is the coefficient of interest and indicates an incremental change in probability of receiving a given treatment associated with a patient being black. In this basic specification, β also represents the difference in means between white and black patients.

Each subsequent specification adds additional covariates to equation (1) to get closer to an estimate of the effect of being black on treatments received holding all else constant. I re-estimate the above model first by adding only demographic controls and then by controlling for a full set of covariates. These covariates control for demographic and hospital characteristics as well as individual risk factors. The remaining three specifications introduce hospital, physician, and hospital and physician fixed effects respectively. The addition of fixed effects to the model allows for the control of observed and unobserved characteristics within hospitals and physicians. The final model including both sets of fixed effects is estimated as follows:

$$Treatment_{ihp} = \alpha + black_{ihp}\beta + X_{ihp}\gamma + \lambda_h + \theta_p + \epsilon_{ihp} \quad (2)$$

where X is a vector of the demographic and risk factor controls (hospital characteristics are controlled for by the hospital fixed effects). λ_h designates the hospital fixed effects while θ_p designates the physician fixed effects. Equivalent analysis is conducted for the secondary outcome variables of interest in panel B.

One concern with prior studies is that differences in physicians are not properly controlled for. If blacks tend to go to lower quality physicians on average, failure to control for physician characteristics may bias results. The introduction of physician fixed effects allows for comparison of treatments for white and black patients seeing the same physician. By holding physician constant I eliminate all observable physician level characteristics such as experience, whether the physician is board-certified, and area of specialization that are not available in the NIS data. While certain studies include these controls, fixed effects also controls for unobservable differences such as physician skill, knowledge, or listening ability that may impact treatments administered. Any residual disparity found under this specification represents a difference in treatment that is not explained by patient characteristics, hospital quality, or physician quality.

IV. Results

A. Treatment outcomes

Table III presents the OLS coefficients on the dummy variable for black outlined above. As a means of comparing the magnitude of these coefficients, the first row of the table presents the proportion of whites receiving each treatment. Overall, the fraction of patients receiving each treatment was relatively low. Almost 60% of whites hospitalized for AF receive no procedures. Anticoagulation therapy was the most administered treatment with 17.31% of whites receiving some form of long-term antithrombic medication. Meanwhile, cardioversion and other electric counter shocks of the heart were administered 5.01% and 6.54% of the time respectively for whites.

In the second row of Table III, I present the OLS coefficients for the basic specification. Of the six treatments analyzed, blacks are significantly less likely to receive four of them. Only heparin therapy and ultrasound showed no evidence of a disparity. Not only is there a statistically significant gap in the proportion of whites and blacks receiving the other four treatments, these gaps are of substantial magnitudes. For example, the results from the basic specification imply that blacks are 1.8 percentage points or 35.9% less likely to receive cardioversion therapy than whites. Similarly blacks are 35.6%, 25.8%, and 12.9% less likely to receive other counter shock therapy, ablation therapy, and long-term anticoagulants respectively. These findings are consistent with the previous literature discussed in section I.

Of course, the findings of the most basic specification are subject to omitted variables bias. As demonstrated in the data section, there are significant differences in the characteristics and risk factors for the two populations of interest. Model (2) controls for demographic discrepancies but does not alter the findings of the previous model. Thus, even though blacks are poorer and more likely to come from large metro areas, these characteristics do not change the probability of receiving a given AF treatment in this sample. One exception to the previous statement is seen in

the coefficient for ultrasound that indicates a now statistically significant 20.7% lower probability that blacks will receive an echocardiogram.

Model (3) controls for observed patient and hospital characteristics. Notably, the coefficient on long-term anticoagulants is drastically attenuated and no longer statistically significant. This finding suggests that the disparities observed in models (1) and (2) are largely explained by differences in risk factors and hospital characteristics. Further, despite controlling for a broad range of covariates, the coefficients on cardioversion and other shock have not changed and remain highly statistically significant and negative.

In the final three models I include fixed effects to control for additional unobserved hospital and physician characteristics that could bias the previous results. Surprisingly, the addition of hospital fixed-effects in model (4) does little to explain cross-racial treatment differences. These findings provide evidence that white and black patients with AF in the same hospital have substantially different odds of receiving proven treatments even after factoring in a broad spectrum of risk factor controls. However, these discrepancies greatly diminish after controlling for physician fixed effects in model (5). Under this specification I find no evidence of differing treatments between whites and blacks for almost all procedures. Not only is there no longer a statistically significant difference, the magnitude of the coefficients is lessened as well. While model (5) suggests a treatment gap for cardioversion therapy, evidence of this disparity is no longer present once hospital and physician fixed effects are used in the final specification.

B. Secondary Outcomes

The results from the previous analysis indicate that blacks and whites tend to go to different physicians. Further, it is this variation in physicians that explains much of the treatment disparities seen in earlier specifications of the model and discussed in the prior literature. This section expands

upon this finding to determine whether any of the observable physician-determined outcomes available in the NIS dataset reveal the ways in which these physicians differ. Table IV presents the results from this subsequent analysis. The construction of this table exactly mirrors Table III with the sole difference being that the dependent variables are replaced with various outcome variables outlined in the data section.

The coefficients from model (3) suggest that there is significant variation across outcomes for blacks and whites even once controls are added to the model. In particular, blacks have longer hospital stays on average, fewer total procedures, and fewer diagnoses. Blacks are less likely to have major operating room procedures and to have been admitted to the hospital through elective admission. Additionally, total expenditures for blacks are 10.97% higher on average compared to those of whites. I include in-hospital mortality as an indicator of suboptimal care, however there is no evidence that blacks are more likely to have died in-hospital. This finding perhaps is not surprising. Poor in-hospital care may lead to complications later on that would not result in death during the stay of the patient. Additionally, patients do not die from AF itself but from other conditions that result from having AF such as stroke or heart failure. As the sample of interest includes patients with a primary diagnosis of AF we should expect mortality in the short term to be relatively low while the longer term mortality risk for these individuals to be elevated.

The narrative for this secondary analysis is similar to that of the first. Within-hospital characteristics do not explain much of the white/black gap for the outcomes that were significant in model (2) with the exception of total charges. In other words, while there are statistically significant differences in care received, there is no evidence that blacks and whites at the same hospital will pay different amounts. This finding could reflect offsetting effects between the longer length of stay for blacks and the lower number of procedures received; however there is no way to substantiate that hypothesis with the NIS data.

As seen in models (5) and (6), the addition of physician fixed effects eliminates the evidence of outcome disparity for most of the dependent variables of interest. These results suggest blacks tend to go to physicians that administer fewer procedures on average and who have patients with longer average hospital stays. These physicians are also more likely to admit patients to the hospital who did not elect to do so themselves. However, even within the same hospital and the same physician, blacks still receive fewer diagnoses and are less likely to undergo a major operating room procedure.

C. Sensitivity Check

Two issues are commonly raised with the LPM. The first is that OLS estimation necessarily imposes heteroskedasticity in the case of a binary variable. The second is that predicted values from LPM estimates are not constrained to the unit interval. The former concern is easily fixed by using heteroskedasticity consistent robust standard errors (Angrist and Pischke 2008). The latter is potentially problematic and could bias predicted value results, particularly if a large proportion of the LPM predicted probabilities fall outside the unit interval (Horrace and Oaxaca 2005). However, given the limitations of alternative non-linear specifications discussed below, the LPM is preferable to conduct fixed effects analysis used in this paper despite predicted values for some observations falling outside the unit interval.

In the papers discussed in section I, non-linear specifications are exceedingly more common when estimating binary choice models. The selection of using LPM in this paper instead of a non-linear specification was done to avoid inducing incidental parameters bias as well as to more reliably calculate marginal effects in the fixed effects specifications. In nonlinear maximum likelihood models, estimators for all variables in the regression are inconsistent when the number of these incidental parameters is fixed, such as the number of hospitals and physicians in this analysis. This

form of bias is known as incidental parameters bias and is almost certain to be present in probit estimations of marginal effects (Lancaster 2000).

The more common approach within the social sciences for binary choice data with fixed effects is to estimate logit models and employ a method of conditional fixed effects. Unlike probit models, conditional logit models eliminate the fixed effect term and are consistent (Heckman 1981). However, there is not a reliable way to calculate average marginal effects under this specification.[4] Nonetheless, as a sensitivity check, I fit a logit model and present the coefficient estimates and p values alongside those of the LPM estimates for model (2) that controlled for patient and hospital characteristics but not fixed effects. These results are presented in Table A-1 in the appendix. The estimates from the comparison of model (2) are not constrained by unreliable marginal effects estimates and suggest strong correlations between the pairs of probability vectors for each outcome variable (correlations not presented). As previously mentioned, comparison of marginal effects are not appropriate when using the fixed effects logit specification. Instead, I present the odds ratio results alongside the p values to compare the magnitude and statistical significance of the results. The results of the conditional logit specification appear to correspond with the OLS results with the exception of heparin treatment. Overall, however, the results from these tests support the robustness of the findings from the LPM estimates to other specifications.

V. Discussion

A paper by Gardner and Vishwasrao (2010) outlines two mechanisms by which patients may be treated by lower quality physicians: the patient goes to a hospital, where average physician quality is lower, or upon admission to a hospital the patient is directed to a lower quality physician within

[4] This limitation is due to the fact that, in a conditional logit model, fixed effects are conditioned out of the likelihood function. Because the fixed effects are not actually estimated as they are in a LPM, it is impossible to estimate the marginal effects unless we assume fixed effects are zero.

that hospital. In the context of the quality of care gap for whites and blacks, the former has an intuitive appeal. Previous research suggests that hospitals located in areas with large populations of minorities, poor, or uninsured patients employ lower-quality physicians on average. As shown in the data section, blacks in the sample tend to live in lower income neighborhoods in urban environments, disproportionately receive care from southern hospitals, and are more likely to receive care from public, government-controlled hospitals. To the extent that these factors are correlated with hospitals with lower-quality physicians, we should expect to see significant variation in physician quality across hospitals that serve blacks or whites. Yet, despite this intuitive explanation, the data suggest that hospital fixed effects explain little of the variation in treatment and outcomes. If the previous hypothesis were true and blacks went to hospitals with lower average quality physicians, these disparities would be controlled for by the hospital fixed effects. The failure of that specification to do so suggests that instead, patients are being directed to lower quality physicians within the hospitals. Although it is not possible to determine how physicians are assigned to patients from the NIS data, the remaining of the discussion section expands upon the latter hypothesis and offers three potential mechanisms through which blacks may be sorted to lower-quality physicians.

Before addressing how black and white patients could be sorted, a natural question that arises is whether there is any previous indication that whites and blacks really do receive care from different physicians. One paper by Bach et al. (2004) provides such evidence. Looking at white and black Medicare beneficiaries, the authors find that 80% of visits by black patients were accounted for by 22% of physicians who provided a small percentage of care to whites. This finding suggests that the care received by whites and blacks largely comes from different physicians. The disparities in treatment found above could emerge if, as Bach et al. argue in their paper, "these two groups of physicians differed in their ability to provide high quality care, either because of differences in their

clinical training or because of differences in their access to resources." Given that blacks are receiving care from different physicians, I next turn to the means by which sorting patients could occur.

Disparities in primary care physicians may explain how blacks and whites are referred to different physicians in-hospital. In analyzing care for the uninsured, Gardner and Vishwasrao suggest that sorting could result from network effects. Namely, when a referral to a specialist or attending physician is required, physicians interviewed stated that doctors saw patients either when it was their turn through rotation of assignments or as a favor for the primary physician requesting the consultation. The authors find that if the primary physician is younger or from a lower-ranked school he or she was more likely to consult a physician from the same cohort. Thus, if the primary care physicians of blacks and whites differ in quality, these physicians are more likely to send their patients to in-hospital physicians of similar quality.

Bach et al. analyze differences in primary care physicians for blacks and whites and find that the primary physicians that serve blacks are less likely to provide high-quality care. In particular, they are less likely to be board certified (adjusted OR .55), less likely to report they are able to obtain access for their patients to high-quality subspecialists (adjusted OR .77), to have access to nonemergency hospital admission (adjusted OR .69), and to report they are able to provide high-quality care to all their patients (adjusted OR .67). Therefore, to the extent that network effects hold in the population analyzed above, one explanation for the findings could be that blacks are being referred to lower quality physicians and specialists in-hospital by their primary care physicians who are also of lower quality on average.

In addition to being seen by lower quality physicians, a previous study suggests that blacks also have less access to specialists. If having a specialist as an attending physician is positively correlated with receiving a greater number of treatments, this disparity in access could partially

explain the treatment gap observed in the data. In their paper on racial variation in treatment for strokes, Mitchell et al. (2000) found that black patients were significantly less likely to have a neurologist as their attending physician even after controlling for hospital and patient characteristics. Further, patients treated by neurologists were more likely to receive diagnostic testing, and of the patients who had AF, were more likely to receive anticoagulant therapy. Given the limitations of the dataset used, I cannot test the hypothesis that blacks in the sample above have less access to cardiologists. Nonetheless, the paper by Mitchell et al. is suggestive that this access gap could be a potential mechanism that explains variation in attending physicians. More research is needed on how patients are referred to specialists to better understand why blacks could have less access and what the implications are for patient outcomes.

Changing physician incentives through the expansion of pay-for-performance (P4P) and public quality-reporting programs offers a third pathway that blacks would see different physicians than whites. These programs are designed to improve quality of care and reduce health disparities. However, they may have the unintended consequence of incentivizing physicians to avoid patients, such as minorities, perceived as likely to lower quality scores. Previous literature suggests that physicians believe minority patient to be less likely to comply with treatments and recommendations and are more likely to have poorer outcomes. Further, a paper by Omoigui et al. (1996) suggests there is evidence that physicians who are exposed to P4P and quality reporting incentives will try to avoid minority patients as a result of these perceived poorer outcomes. For example, when New York State began a quality-reporting program that measured death rates from CABG surgery for surgeons, the gap between CABG rates for whites and blacks increased. If some physicians in the sample above were exposed to these incentive programs, these physicians may be less likely to accept referrals for black patients. As a result, blacks could be more likely to receive care from younger or lower quality physicians who have less input over which patients they treat.

VI. Conclusion

While this paper is suggestive that variations in treatment by race can be explained by differences in physicians, later specifications of analysis may suffer from low power due to the addition of fixed effects and the low incidence rates associated with the treatments of interest. Low power increases the chance of type II error and could contribute to the lack of statistically significant results in the final specification. To more precisely estimate the specifications with fixed effects, subsequent analysis should be conducted that pools together multiple years of NIS data in order to increase the number of observations compared within each physician.

Moreover, although these results control for a broad range of observed and unobserved factors, it is impossible to fully eliminate the concern of omitted variables bias in the estimates. A physician's decision to administer a particular treatment is determined by a host of indicators for patient health and contraindications that are perceived by hospital staff and that may not be reflected in the data.

This analysis examined treatments in the inpatient context. However, AF is largely treated in an outpatient setting. While the findings of this paper are representative of AF Medicare patients in hospitals, there are concerns over the external validity of the results. Thus, it is not clear that the implications drawn here can be generalized to patients treated for AF as outpatients.

Finally, this paper sought to identify whether there was a difference in treatments received by race. Perhaps a more important question is what the long-term impacts of that disparity are for the health of the individual. Although, this analysis finds no evidence of in-hospital mortality disparities, it is yet to be determined whether blacks are at a higher risk of dying due to future complications resulting from inadequate treatment. To the extent that low treatment does lead to worse health indicators, this finding could imply that physician quality explains the failure of policy efforts of the past few decades to close the racial health gap.

Nonetheless, despite concerns over low power, the data suggest highly significant differences in receipt of cardioversion, other shock therapy, ablation, and diagnostic ultra sounds within hospital. This finding contrasts with those in prior studies. As a result, previous suggestions to provide greater amounts of resources to hospitals that primarily serve blacks may not be a complete solution. In particular, if blacks are still being sorted to lower quality physicians within the hospitals, inequities would persist.

There is a growing literature supporting the hypothesis that patients are sorted to physicians of different quality. Greater investigation into the extent to which the above pathways are true is necessary to determine the optimal ways to address this disparity. However, I present a few preliminary policy implications of the findings above. If health differentials are originating at the primary care physician level, policies targeted at hospital resources may be too little too late. Instead, addressing the limitations in the quality of primary care physicians that serve blacks may have the dual effect of earlier detection of disease and referral to better quality physicians in-hospital. Finally, the full impact of P4P initiatives must be examined. While efforts to improve transparency and physician quality are valuable, the evidence of harmful unintended consequences may mitigate societal gains from the policy and perpetuate racial disparities in health.

Table I
Description of Treatment Variables

Treatment	Description	ICD 9 Code
Ultrasound	Diagnostic ultrasounds (echocardiograms) use high-pitched sound waves that are sent through the ultrasound device held onto the chest. This device picks up echoes of the sound waves as they reverberate off the patient's heart. Ultrasounds allow physicians to diagnose atrial fibrillation as well as detect clots in the atria.	88.72
Heparin	Heparin is an anticoagulation treatment administered intravenously or subcutaneously in-hospital. Anticoagulation therapy can provide significant clinical benefit by reducing stroke or systemic embolism risk in patients with AF.	99.19
Anticoagulant	Oral anticoagulants, such as warfarin, are commonly administered as a long-term treatment for atrial fibrillation and have similar benefits to those of heparin.	V58.61
Ablation	Catheter ablation involves the insertion of a series of thin wires through the patient's blood vessels into the heart. Radiofrequency energy is sent through the catheters to destroy small areas of heart tissue where abnormal heartbeats may cause arrhythmia. Often several procedures are needed to raise the success rate to above 70%, particularly among older AF patients.	37.34
Cardioversion	Electrical cardioversion is a procedure that uses synchronized low-voltage electric current to reset the heart's rhythm back to normal sinus rhythm. Cardioversion is a defibrillation that occurs at a specific point in the electrical cycle. In 90% of patients normal sinus rhythm is restored but may not last. If an irregular heart rhythm returns, a second or third treatment of cardioversion is possible. Cardioversion may not be recommended if the patient has valve problems, an enlarged heart, or long-term AF.	99.61
Other Shock	Other electric counter shock refers to defibrillation techniques whereby electrical current is used to take the patient out of fibrillation. This form of defibrillation occurs without regard to the electrical cycle.	99.62
No Procedure	No Procedure indicates that the inpatient data showed no procedural code listed for that patient.	

Table II
Summary of Statistics for Covariates

	Whole Sample	White (n=38,270)	Black (n=1,993)
Demographics			
Age	78.54	78.60	77.38
	(7.79)	(7.74)	(7.96)
Female	0.620	0.618	0.655
	(0.485)	(0.486)	(0.476)
Patient Location			
Center of large metro areas	0.214	0.201	0.4524
	(0.406)	(0.396)	(0.497)
Fringe of large metro areas	0.272	0.274	0.222
	(0.445)	(0.445)	(0.416)
Medium metro area	0.189	0.193	0.107
	(0.391)	(0.395)	(0.309)
Small metro area	0.103	0.103	0.088
	(0.303)	(0.305)	(0.282)
Micropolitan	0.136	0.139	0.074
	(0.343)	(0.346)	(0.262)
Neither metropolitan nor micropolitan	0.087	0.089	0.057
	(0.282)	(0.285)	(0.231)
Median household income of patient's ZIP code			
<$39,999	0.239	0.225	0.508
	(0.424)	(0.415)	(0.500)
$40,000-$49,999	0.266	0.268	0.208
	(0.442)	(0.443)	(0.406)
$50,000-$65,999	0.248	0.252	0.156
	(0.432)	(0.435)	(0.363)
$66,000+	0.246	0.252	0.128
	(0.431)	(0.434)	(0.334)
Hospital			
Hospital Control			
Government, public	0.091	0.089	0.141
	(0.285)	(0.281)	(0.340)
Private, non-profit	0.770	0.774	0.683
	(0.421)	(0.418)	(0.465)
Private	0.139	0.137	0.176
	(0.346)	(0.344)	(0.381)
Number of Beds[5]			
Small	0.140	0.142	0.102
	(0.344)	(0.346)	(0.296)
Medium	0.247	0.246	0.261
	(0.431)	(0.431)	(0.439)
Large	0.613	0.611	0.636
	(0.487)	(0.487)	(0.481)
Urban	0.851	0.848	0.918
	(0.356)	(0.359)	(0.274)
Hospital Region			
Northeast	0.247	0.248	0.222
	(0.431)	(0.432)	(0.416)
Midwest	0.203	0.205	0.167
	(0.402)	(0.404)	(0.373)
South	0.398	0.392	0.524
	(0.490)	(0.488)	(0.500)

[5] Bedsize categories depend on region, location, and teaching status.

	Whole Sample	White	Black
West	0.152	0.155	0.087
	(0.359)	(0.362)	(0.282)
Teaching Hospital	0.3843	0.3770	0.5281
	(0.4864)	(0.4846)	(0.4993)
Risk Factors			
Risk of Mortality			
Minor likelihood of dying	0.301	0.307	0.188
	(0.459)	(0.461)	(0.391)
Moderate likelihood of dying	0.421	0.431	0.420
	(0.494)	(0.494)	(0.495)
Major likelihood of dying	0.231	0.228	0.296
	(0.422)	(0.419)	(0.456)
Extreme likelihood of dying	0.047	0.045	0.086
	(0.212)	(0.207)	(0.280)
Severity of Illness			
Minor loss of function	0.189	0.193	0.102
	(0.391)	(0.395)	(0.303)
Moderate loss of function	0.423	0.425	0.379
	(0.494)	(0.494)	(0.485)
Major loss of function	0.337	0.332	0.424
	(0.473)	(0.471)	(0.494)
Extreme loss of function	0.052	0.049	0.095
	(0.222)	(0.217)	(0.294)
Hypertension	0.692	0.687	0.790
	(0.462)	(0.464)	(0.407)
Congestive Heart Failure	0.006	0.005	0.014
	(0.014)	(0.074)	(0.118)
Coagulopathy	0.029	0.028	0.044
	(0.168)	(0.166)	(0.205)
Deficiency Anemias	0.146	0.142	0.240
	(0.354)	(0.349)	(0.427)
Chronic blood loss Anemia	0.006	0.006	0.006
	(0.074)	(0.074)	(0.074)
Diabetes, uncomplicated	0.209	0.204	0.319
	(0.407)	(0.403)	(0.466)
Diabetes with chronic complications	0.028	0.027	0.058
	(0.166)	(0.161)	(0.233)
Drug Abuse	0.002	0.002	0.008
	(0.044)	(0.040)	(0.089)
Alcohol Abuse	0.018	0.018	0.025
	(0.133)	(0.131)	(0.156)
Hypothyroidism	0.194	0.199	0.101
	(0.395)	(0.399)	(0.301)
Obesity	0.080	0.075	0.120
	(0.271)	(0.267)	(0.325)
Peripheral vascular disorders	0.083	0.083	0.091
	(0.276)	(0.275)	(0.287)
Pulmonary circulation disorders	0.002	0.002	0.004
	(0.045)	(0.045)	(0.059)
Renal failure	0.134	0.129	0.278
	(0.343)	(0.335)	(0.448)
Valvular disease	0.003	0.003	0.005
	(0.051)	(0.050)	(0.067)

Table III
OLS Estimates of Race and Treatment Disparities: Basic Results

		Dependent Variable:						
Model	Specification	Ultrasound	Anticoagulant	Heparin	Ablation	Cardioversion	Other Shock	No Procedure
	Sample Mean Whites	0.1033	0.1731	0.0045	0.0406	0.0501	0.0654	0.5947
(1)	Basic LPM	-0.01649	0.0225**	0.0025	-0.0105*	-0.0180**	-0.0233***	-0.0217
(2)	Demographic Controls	-0.0214*	-0.0176*	0.0006	-0.0107*	-0.0180**	-0.0239***	-0.0096
(3)	Patient and Hospital Controls	-0.0262**	-0.0040	0.0007	-0.0090	-0.0217***	-0.0233***	0.0349**
(4)	Hospital FE	-0.0206**	0.0036	0.0008	-0.0179**	-0.0244***	-0.0209***	0.0394**
(5)	Physician FE	-0.0084	0.0260	-0.0002	-0.0114	-0.0181*	-0.0098	0.0003
(6)	Hospital and Physician FE	-0.0020	0.0250	-0.0029	-0.0126	-0.0105	-0.0093	0.0015

Notes: This table reports OLS estimates for a dummy variable for blacks in the indicated specifications. The dependent variables are binary variables denoted in the column headings. Robust standard errors clustered by hospital in specifications 1-4 and by physician id in specification 5 and 6 were used. The sample includes whites and blacks over the age of 65 diagnosed with atrial fibrillation using Medicare as their primary payer.

*** Significant at the 1 percent level
** Significant at the 5 percent level
* Significant at the 10 percent level

Table IV
OLS Estimates Of Race and Quality Disparities: Basic Results

Model	Specification	Dependent Variable: Length of Stay	Number of Procedures	Number of Diagnoses	Major OR Procedure	Log Total Charges	Elective Admission	Died in Hospitalization
	Sample Mean Whites	3.7938	0.9778	10.1014	0.0758	9.7246	0.1356	0.0112
(1)	Basic LPM	1.2007***	0.0879*	0.9985***	-0.0015	0.2643***	-0.0512***	0.0053*
(2)	Demographic Controls	1.0623***	0.0259	0.9273***	-0.0056	0.1952***	-0.0480***	0.0042
(3)	Patient and Hospital Controls	0.3183**	-0.1518**	-0.4333**	-0.0145**	0.1097**	-0.0281**	-0.0030
(4)	Hospital FE	0.2564**	-0.1587***	-0.2985***	-0.0281***	-0.1082	-0.0370***	-0.0018
(5)	Physician FE	0.3456	-0.0640	-0.3794**	-0.0149	0.0035	-0.0152	0.0028
(6)	Hospital and Physician FE	0.2682	-0.1013	-0.4026**	-0.0200*	-0.0185	-0.0222	0.0063

Notes: This table reports OLS estimates for a dummy variable for blacks in the indicated specifications. The dependent variables are binary variables denoted in the column headings. Robust standard errors clustered by hospital in specifications 1-4 and by physician id in specification 5 and 6 were used. The sample includes whites and blacks over the age of 65 diagnosed with atrial fibrillation using Medicare as their primary payer.

*** Significant at the 1 percent level
** Significant at the 5 percent level
* Significant at the 10 percent level

References

Andruilis, P. Dennis, Nadia J. Siddiqui, Jonathan P. Purtle, and Lisa Duchon. "Patient Protection and the Affordable Care Act of 2010." Joint Center For Political and Economic Studies

Angrist, D. Joshua., and Jörn-Steffen Pischke. *Mostly Harmless Econometrics: An Empiricist's Companion*. Princeton: Princeton UP, 2009. Print.

Bach, B. Peter, Hoangmai H. Pham, Deborah Schrag, Ramsey C. Tate, and J. Lee Hargraves. 2010. "Primary Care Physicians Who Treat Blacks and Whites." *New England Journal of Medicine* 351.6: 575-84.

Barnato, E. Amber, F. Lee Lucas, Douglas Staiger, David E. Wennberg, and Amitabh Chandra. 2005. "Hospital-Level Racial Disparities in Acute Myocardial Infarction Treatment and Outcomes." *Medical Care* 43.4: 308-19.

CDC Health Disparities and Inequalities Report. Rep. Center For Disease Control, 2013.

Crandall, Mark A., David J. Bradley, Douglas L. Packer, and Samuel J. Asirvatham. 2009. "Contemporary Management of Atrial Fibrillation: Update on Anticoagulation and Invasive Management Strategies." Mayo Clinic Proceedings 84.7: 643-62.

Erasing the Color Line. Rep. Grant Makers in Health, Nov. 2003.

Gardner, Lara, and Sharmila Vishwasrao. 2010. "Physician Quality and Health Care for the Poor and Uninsured." *Inquiry* 47.1: 62-80.

Go, S. Alan. 2001. "Prevalence of Diagnosed Atrial Fibrillation in Adults: National Implications for Rhythm Management and Stroke Prevention: The AnTicoagulation and Risk Factors In Atrial Fibrillation (ATRIA) Study." *JAMA: The Journal of the American Medical Association* 285.18: 2370-375.

Heckman, J. James. 1981. *The Incidental Parameters Problem and the Problem of Initial Conditions In Estimating a Discrete Time-Discrete Data Stochastic Process*.

"High Blood Pressure (hypertension)." *Beta Blockers*. Mayo Clinic, 01 Feb. 2014. Web.

Horrace, William C., and Ronald L. Oaxaca. 2006. "Results on the Bias and Inconsistency of Ordinary Least Squares for the Linear Probability Model." *Economics Letters* 90.3: 321-27.

Introduction to the HCUP Nationwide Inpatient Sample (NIS). Rep. HCUP, 2009.

Lancaster, Tony. 2000. "The Incidental Parameters Problem since 1948." *Journal of Econometrics* 95: 391-413.

Leong-Sit, P., E. Zado, D. J. Callans, F. Garcia, D. Lin, S. Dixit, R. Bala, M. P. Riley, M. D. Hutchinson, J. Cooper, E. P. Gerstenfeld, and F. E. Marchlinski. 2010. "Efficacy and Risk

of Atrial Fibrillation Ablation Before 45 Years of Age." *Circulation: Arrhythmia and Electrophysiology* 3.5: 452-57.

Lucas, F. L., Therese A. Stukel, Arden M. Morris, Andrea E. Siewers, and John D. Birkmeyer. 2006. "Race and Surgical Mortality in the United States." *Annals of Surgery*. U.S. National Library of Medicine.

Meschia, J. F., P. Merrill, E. Z. Soliman, V. J. Howard, K. M. Barrett, N. A. Zakai, D. Kleindorfer, M. Safford, and G. Howard. 2010. "Racial Disparities in Awareness and Treatment of Atrial Fibrillation: The REasons for Geographic and Racial Differences in Stroke (REGARDS) Study." *Stroke* 41.4: 581-87.

Mitchell, B. Janet, David J. Ballard, David B. Matchar, Jack P. Whisnant, and Gregory P. Samsa. 2000. "Racial Variation in Treatment for Transient Ischemic Attacks: Impact of Participation by Neurologists." *Health Services Research* 34.7: 1413-428.

Naderi, Sahar, Fatima Rodriguez, Yun Wang, and JoAnne Foody. 2014. "Racial Disparities in Hospitalizations, Procedural Treatments and Mortality of Patients Hospitalized with Atrial Fibrillation." *Ethnicity & Disease*: 144-49.

Omoigui, N. A., D. P. Miller, K. J. Brown, K. Annan, D. Cosgrove, B. Lytle, F. Loop, and E. J. Topol. 1996. "Outmigration For Coronary Bypass Surgery in an Era of Public Dissemination of Clinical Outcomes." *Circulation* 93.1: 27-33.

Orsi, Jennifer M., Helen Margellos-Anast, and Steven Whitman. 2010. "Black–White Health Disparities in the United States and Chicago: A 15-Year Progress Analysis." *American Journal of Public Health* 100.2: 349-56.

Prystowsky, Eric N. 2000. "Management of Atrial Fibrillation: Therapeutic Options and Clinical Decisions." *The American Journal of Cardiology* 85.10: 3-11.

Regenbogen, Scott E., Atul A. Gawande, Stuart R. Lipsitz, Caprice C. Greenberg, and Ashish K. Jha. 2009. "Do Differences in Hospital and Surgeon Quality Explain Racial Disparities in Lower-Extremity Vascular Amputations?" *Transactions of the Meeting of the American Surgical Association* 127: 68-75.

Ross, Janell. "Affordable Care Act May Help Close Gap on Health Disparities." *www.nationaljournal.com*. National Journal, 9 Apr. 2014.

Roy, Denis, Mario Talajic, Stanley Nattel, George Wyse, Paul Dorian, Kerry L. Lee, Martial G. Bourassa, Malcolm O. Arnold, Alfred E. Buxton, John Camm, Stuart J. Connolly, Marc Dubuc, Anique Ducharme, Peter G. Guerra, Stefan H. Hohnloser, Jean Lambert, Jean-Yves Le Heuzey, Gilles O'Hara, Ole Dyg Pedersen, Jean-Lucien Rouleau, Bramah N. Singh, Lynne Warner Stevenson, William G. Stevenson, Bernard Thibault, and Albert L. Waldo. 2008. "Rhythm Control versus Rate Control for Atrial Fibrillation and Heart Failure" *New England Journal of Medicine*. 358.25: 2667-2677.

Shapira, Adam R. 2009. "Catheter Ablation of Supraventricular Arrhythmias and Atrial Fibrillation." *American Family Physician* 10th ser. 15.80: 1089-094.

Smedley, Brian D., Adrienne Y. Stith, and Alan R. Nelson. *Unequal Treatment: Confronting Racial and Ethnic Disparities in Health Care*. Washington, D.C.: National Academy, 2003.

Sonel, Ali F., Chester B. Good, Jyotsna Mulgund, Matthew T. Roe, Brian Gibler, Sidney C. Smith, Jr., Mauricio G. Cohen, Charles V. Pollack, Jr., Magnus Ohman, and Eric Peterson. 2005. "Racial Variations in Treatment and Outcomes of Black and White Patients With High-Risk Non-ST-Elevation Acute Coronary Syndromes: Insights From CRUSADE (Can Rapid Risk Stratification of Unstable Angina Patients Suppress Adverse Outcomes With Early Implementation of the ACC/AHA Guidelines)." *Circulation* 111.10: 1225-232.

Thomas, Kevin L., Jonathan P. Piccini, Li Liang, Gregg Fonarow, Clyde Yancy, Eric Peterson, and Adrian Hernandez. 2013. "Racial Differences in the Prevalence and Outcomes of Atrial Fibrillation Among Patients Hospitalized With Heart Failure." *Arrhythmia and Electrophysiology* 2: 1-9.

"What Is Atrial Fibrillation?" The American Heart Association, 16 Apr. 2014.

Appendix

Table A-1
Comparison of OLS Estimates to Logistic Estimates

Treatment	Mean Value for whites (1)	Patient and Hospital Controls: Primary Specification (2)	P Value (3)	Logistic Specification (4)	P Value (5)	Final Model: Primary Specification (6)	P Value (7)	Logistic Specification (OR) (8)	P Value (9)
Panel A									
Ultrasound	0.1025	-.0262	0.037	-0.0278	0.049	-0.0020	0.888	0.988	0.943
Heparin	0.0047	.0007	0.761	-0.0014	0.510	-0.0029	0.345	0.253	0.028
Anticoagulant	0.1719	-.0040	0.705	-0.0054	0.641	0.0250	0.173	1.200	0.122
Ablation	0.0401	-.0090	0.105	-0.0109	0.122	-0.1263	0.201	0.633	0.118
Cardioversion	0.0492	-.0217	0.000	-0.0265	0.002	-0.0105	0.302	0.788	0.313
Other Shock	0.0643	-.0233	0.001	-0.0294	0.005	-0.0093	0.429	0.855	0.406
No Procedure	0.5937	.0349	0.041	0.0350	0.039	0.0015	0.948	1.01	0.912

Notes: This table repeats the OLS estimates from model (3) and (6) presented in table III along with the p values for those estimates. Column (4) presents the average marginal effects from the logistic model estimation. The average marginal effects are calculated by predicting the outcome as a treatment and as a control for each individual, taking the difference in the two predictions, and averaging those differences across the whole sample. Model (6) is compared to odds ratio results from conditional logistic regressions. The results are robust to the choice of model specification.