

**Two Sides of the Same Coin?
Employing Granger Causality Tests in a Panel Framework**

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Abstract: In this paper, we introduce a recently developed technology for assessing the assumption of causal homogeneity in panel Granger frameworks. Following a description of the procedure and the analytical contexts for which it is appropriate, we use this new technology to evaluate the recent claims of *relative advantage* theory regarding the causal relationship between black mobilization and GOP growth in the modern South. Specifically, we evaluate the cross-state robustness of earlier findings relating to the causal linkage between black mobilization and Republican growth and the possibility that GOP growth might have generated subsequent black mobilization in one or more Southern states. Our findings indicate black mobilization Granger caused Republican growth throughout the South, while Republican growth Granger caused black mobilization only in the deep South.

Introduction

Introduced nearly forty years ago, Granger models (see Granger 1969) of various types remain the most popular methodology for evaluating the nature of the causal relationship between two variables. Though originally designed for pairs of lengthy time series, Granger tests are increasingly used to evaluate causal relationships in panel data. The extension of the original Granger methodology to panel data has the potential to improve upon the conventional Granger analysis for all of the reasons that panel analysis is generally preferable to cross-sectional or traditional time series analysis. In general, panel models provide a number of improvements over the separate analysis of time series by cross-section. First, panel data allow for considerably more flexibility in the modeling of the behavior of cross-sectional units than conventional time series analysis (see Greene 2000). Second, the panel framework allows for the analytical incorporation of significantly more observations (and more degrees of freedom) than would a comparable analysis of individual time series. Finally, and most significantly for our purposes, panel Granger tests are significantly more efficient than conventional Granger tests (Hurlin and Venet 2001). The potential benefits of panel Granger tests are, therefore, considerable.

However, just as panel Granger tests share the benefits of other types of panel analysis, they also share an important potential flaw: the inappropriate assumption of causal homogeneity. As is often the case in panel analysis, this assumption is largely ignored. Failure to adequately analyze the empirical foundations for this assumption could easily lead to faulty substantive conclusions—inferring a causal relationship in all cross-sections when it is only present in a subset of cross-sections or rejecting the presence of a causal relationship for an entire group of observations when a subset of the sample does actually manifest the hypothesized causal

relationship. Following a brief discussion of Granger analysis and its extension to panel models, we describe a new procedure—developed in Hurlin and Venet (2001)—for evaluating the causal homogeneity of cross-sections in a panel framework. We then apply this new methodology to a particular research question in the literature on Southern politics. Specifically, we evaluate the causal claims of *relative advantage* theory as they relate to the relationship between the growth of Southern Republicanism and black mobilization in the South. We conclude with a discussion of the proper use of this new procedure—in our specific empirical context and more generally.

Assessing Causality—The Granger Framework

Granger testing is a common method of investigating causal relationships (Granger 1969). By estimating an equation in which y is regressed on lagged values of y and the lagged values of an additional variable x , we can evaluate the null hypothesis that x does *not* Granger-cause y . If one or more of the lagged values of x is significant, we are able to *reject* the null hypothesis that x does not Granger cause y . In more straightforward terms, we conclude that x Granger causes y . Though imperfect, it is, nevertheless a standard and useful tool for evaluating the character of the causal relationship between two variables.¹

Recently, econometricians have begun to modify Granger tests to incorporate panel dynamics (see for example Arellano and Bond 1991; Holtz-Eakin, Newey, and Rosen 1988;

¹These problems are discussed in some detail in Greene (2000) and Kmenta (1997). Aside from the fact that no technique can fully ascertain the nature of a causal relationship between two variables, the Granger procedure is a simple bivariate model. It is possible, for example, that the relationship between x and y is spurious (the correlation being caused by the impact of some other variable, z , on x and y). This renders the isolated use of Granger tests—whether or not in a panel framework—problematic. It does not, however, obviate the usefulness of Granger tests as a first step towards uncovering the character of the causal relationship between two variables. We discuss this issue in more detail in the conclusion.

Hurlin 2005; and Hurlin and Venet 2001).² Within panel frameworks, Granger tests generate meaningful results with significantly shorter time, incorporate significantly more observations, and produce more efficient results than Granger tests in the conventional context (Hurlin and Venet 2001).

Employing conventional Granger tests with panel data, however, generates two important inferential issues, both dealing with the potential heterogeneity of the individual cross-sections. The first potential type of cross-section variation is due to distinctive intercepts, and this type of variation is addressed with a fixed effects model. The other more problematic type of heterogeneity—causal variation across units—requires a more complex analytical response. Until recently, this type of heterogeneity was ignored (with unknown results). Erdil and Yetkiner (2005) identify two distinctive literatures dealing with panel VAR (vector autoregression models).³ The literature based on early work by Hsiao (1986) and Holtz-Eakin et al. (1988) largely ignores this type of heterogeneity.

The other strain of literature, that based on recent work by Hurlin and Venet (2001), explicitly addresses this type of heterogeneity. Hurlin and Venet (2001) outline a procedure for evaluating the character of the causal processes (homogenous vs. heterogenous) within a panel framework.⁴ Their analytical results are based on a panel Granger model of the following type, where for each of the individuals i and for all t in $[1, T]$:⁵

²Some substantive examples of this methodology include Podrecca and Carmeci (2001); Weinhold and Nair (2001); Davis and Hu (2004); Hurlin and Venet (2004); and Erdil and Yetkiner (2005).

³Granger models are one type of VAR model.

⁴See Davis and Hu (2004) or Erdil and Yetkiner (2005) for substantive examples of the methodology described in Hurlin and Venet (2001).

⁵Note that the residuals satisfy the conventional assumptions. See Hurlin and Venet (2001) for more details.

$$y_{i,t} = \sum_{k=1}^p \gamma^{(k)} y_{i,t-k} + \sum_{k=0}^p \beta_i^{(k)} x_{i,t-k} + v_{i,t}$$

Two important distinctions between the Hurlin and Venet model and the primary alternative to panel VAR modeling are the following:

1. the autoregressive coefficients ($\gamma^{(k)}$) are assumed constant and
2. the regression coefficients ($\beta_i^{(k)}$) are also assumed constant for all $k \in [1,p]$.

In addition, while the autoregressive slope coefficients are identical for all individuals, the regression coefficients are allowed to vary across individual cross-sections. Hurlin and Venet identify four distinct scenarios to describe the causal processes: *Homogenous Non-Causality*, *Homogenous Causality*, *Heterogeneous Causality*, and *Heterogeneous Non-Causality*.⁶

In the *Homogenous Non-Causality* case, there is no linear causal relationship between x and y for any of the individual cross-sections. More formally, the condition of *Homogenous Non-Causality* is defined as one in which the following is true:

$$\forall i \in [1, N] \quad E(y_{i,t} / \bar{y}_{i,b}, \bar{x}_{i,b}, \alpha_i) = E(y_{i,t} / \bar{y}_{i,b}, \bar{x}_{i,b}, \alpha_i)$$

where $E(y_{i,t} / \bar{y}_{i,b}, \bar{x}_{i,t})$ is the best linear predictor of $y_{i,t}$ given the past values of $y_{i,t}$ (denoted $\bar{y}_{i,t}$) and the past values of $x_{i,t}$ (denoted $\bar{x}_{i,t}$).⁷

⁶ Heterogeneity arising from level differences between cross-sections is addressed by including unit-specific (fixed) effect parameters.

⁷ Hurlin and Venet (2001) (inclusion of x_t as a regressor) explicitly define the best linear predictor for the *instantaneous* case (includes current value of x as a regressor). Our definition of the best linear predictor for the more conventional Granger case (does not include current value of x as a regressor) is a straightforward extension.

In the case of the *Homogenous Causality*, there are N linear causal relationships (each cross-section manifests a causal relationship). More formally, the condition of *Homogenous Causality* is defined as one in which the following is true:

$$\forall i \in [1, N] \quad E(y_{i,t} / \bar{y}_{i,b} \alpha_i) = E(y_{i,t} / \bar{y}_{i,b} \bar{x}_{i,b} \alpha_i)$$

In accordance with Hurlin and Venet's assumption that the individual predictors ($\bar{y}_{i,b}$, $\bar{x}_{i,b}$ and α_i) are identical, then the following is also true:

$$\forall (i, j) \in [1, N] \quad E(y_{i,t} / \bar{y}_{i,b} \bar{x}_{i,b} \alpha_i) = E(y_{j,t} / \bar{y}_{j,b} \bar{x}_{j,b} \alpha_j)$$

The case of *Heterogeneous Causality* corresponds to the presence of at least *one* causal relationship (up to a maximum of N causal relationships). So, the following is true:

$$\exists i \in [1, N] \quad E(y_{i,t} / \bar{y}_{i,b} \alpha_i) \neq E(y_{i,t} / \bar{y}_{i,b} \bar{x}_{i,b} \alpha_i)$$

Unlike the preceding case, the individual predictors are assumed to be heteroogenous, so we also have:

$$\exists (i, j) \in [1, N] \quad E(y_{i,t} / \bar{y}_{i,b} \bar{x}_{i,b} \alpha_i) \neq E(y_{j,t} / \bar{y}_{j,b} \bar{x}_{j,b} \alpha_j)$$

The final case refers to that situation in which at least one individual (and up to $N-1$ individuals) does *not* manifest a causal relationship. In this case, there must be at least one equality consistent with the following form:

$$\exists i \in [1, N] \quad E(y_{i,t} / \bar{y}_{i,b} \alpha_i) = E(y_{i,t} / \bar{y}_{i,b} \bar{x}_{i,b} \alpha_i)$$

This exhausts the theoretically possible outcomes associated with panel Granger tests. We now move to a more detailed description of the implementation of the Hurlin and Venet procedure for empirically distinguishing between these various cases.

A Method for Causality Testing in Panel Settings

Hurlin and Venet's methodology for evaluating causal heterogeneity in panel data is composed of a set of nested tests. These tests are easily implemented using the constrained regression technique in Stata® or in similar programs such as E-Views®. Before outlining the specific hypotheses and requisite tests, we outline the basic process below:

1. Conduct panel nonstationarity tests for each variable to be examined.⁸
2. Create a dummy variable for each panel member.
3. Create a set of slope parameters by multiplying each unit-specific variable by lags of the independent variable.
4. Specify the necessary equations with the proper constraints, excluding the constant term.
5. For each model, record the sum of squared residuals or save as a new variable.
6. Calculate the required test statistics and determine significance.

A flowchart outlining the nested testing procedure described in the previous section is presented in Figure 1.⁹ The first hypothesis to be tested is the *Homogenous Non-Causality Hypothesis*. More specifically, the *Homogenous Non-Causality Hypothesis* tests the proposition that across all the members of our panel, x does not Granger cause y . Rejection of the

⁸These models operate under the assumption that all series to be tested do not contain a unit root. Popular statistical software packages (such as Stata) include standard tests to assess stationarity in panel data. Two examples would be the Levin, Lin, Chu Test or the Im, Pesaran, and Shin Test. See Baltagi (2005) for a discussion of unit root tests for panel data.

⁹Nomenclature and equations come directly from Hurlin and Venet (2001).

Homogenous Non-Causality Hypothesis, therefore, indicates that for at least one or more members of the panel ($n \geq 1$), x does Granger cause y .¹⁰

<Figure 1 about here>

We assess the *Homogenous Non-Causality Hypothesis* by constructing a test statistic comparing the sum of squared residuals from a set of restricted models (RSS_2) to the sum of squared residuals produced by a set of baseline (unrestricted) models (RSS_1). A significant test statistic indicates that for at least some (and possibly all) of the cross-sections, there exists a causal relationship. Failure to reject the *Homogenous Non-Causality Hypothesis* is an indication that x does not Granger cause y for any of the panel members and, subsequently, the testing process ends here. As in the traditional Granger causality test, the unrestricted model includes lags of $y_{i,t-k}$, lagged values of the interactive terms ($x_{i,t-k}$), and the fixed effects themselves to predict current values of $y_{i,t}$. Lagged values of y are constrained to equality ($\beta x_{i,t-1} = \beta x_{i,t-k}$) for all models presented. In the unrestricted model, subsequent lags for within-panel slope coefficients are also set to equality ($\beta x_{i,t-1} = \beta x_{i,t-k}$). In the restricted model slope coefficients and lags are constrained to zero ($\beta x_{i,t-1} = 0$), leaving only the unit specific effects and the various lags of the dependent variable to predict current values of y .¹¹

The *Homogenous Non-Causality* test statistic (F_{hnc}) is calculated as:

$$F_{hnc} = \frac{(RSS_2 - RSS_1)/(Np)}{RSS_1/[NT - N(1 + p) - p]}^{12}$$

¹⁰It is important to note that rejection of the *Homogenous Non-Causality Hypothesis* does not indicate the presence of a homogenous causal process for the entire panel (or that x Granger causes y for all members of the panel).

¹¹Slope coefficients for x can be either constrained to zero or excluded from the model altogether.

¹² N =number of panel members; p =number of lags; T =number of time periods

Interpretation of the statistic relies on the Fischer distribution with Np , $NT-N(1+p)-p$ degrees of freedom. If evidence exists to reject the *Homogenous Non-Causality Hypothesis*, the next step proceeds to test the hypothesis of homogenous causality.

To test the *Homogenous Causality Hypothesis*, we calculate another test statistic hereafter referred to as F_{hc} . Acceptance of the *Homogenous Causality Hypothesis* (insignificant test statistic) indicates that a common causal process is manifest for *all* panel members. At this juncture further testing is unnecessary as x is said to Granger cause y for all panel members. Rejection of the *Homogenous Causality Hypothesis* (significant test statistic) would indicate that for at least one or more panel members, x does not Granger cause y . The F_{hc} test statistic is calculated using the sum of squared residuals from the unrestricted model described above (RSS_1) along with the sum of squared residuals (RSS_3) from a restricted model in which the slope terms are constrained to equality for all the panels in the sample ($\beta_{x_{t-1}} = \beta_{x_{t-k}}$). Calculation of the F_{hc} test statistics is as follows:

$$F_{hc} = \frac{(RSS_3 - RSS_1) / [p(N - 1)]}{RSS_1 / [NT - N(1 + p) - p]}$$

Again, the test statistic is interpreted using a Fischer distribution with Np , $NT-N(1+p)-p$ degrees of freedom.

If the *Homogenous Causality Hypothesis* is rejected, a third hypothesis is tested in order to determine for which panel members x Granger causes y . For each panel member (i), the *Heterogeneous Non-Causality Hypothesis* is conducted using the unrestricted sum of squared residuals estimated previously (RSS_1) in addition to the sum of squared residuals ($RSS_{2,i}$) from a model in which the slope coefficient ($\beta_{x_{i,t-k}} = 0$) for the panel member in question is constrained to zero (or excluded from the model equation). The statistic to test the *Heterogeneous Non-*

Causality Hypothesis (hereafter referred to as F_{henc}^i) for panel member (i) is calculated as follows:

$$F_{\text{henc}}^i = \frac{(RSS_{2,i} - RSS_1) / p}{RSS_1 / [NT - N(1 + 2p) + p]}$$

If the F_{henc}^i test statistic is significant using a F-distribution with Np , $NT - N(1+p) - p$ degrees of freedom then we can reject the *Heterogeneous Non-Causality Hypothesis*, indicating that x does Granger cause y for panel member (i). If the F_{henc}^i test statistic is insignificant, then x does not Granger cause y for panel member (i). Testing of the *Heterogeneous Non-Causality Hypothesis* is conducted for each panel member across the requisite number of time periods ($t-k$) in order to determine which members contributed to the finding of causality (denoted by the rejection of the *Homogenous Non-Causality Hypothesis*).

When theory suggests grouping members of a panel in a specific manner, a second *Heterogeneous Non-Causality Hypothesis* test statistic can be specified to examine the causal nature for some subset of panel members. In this case, the slope coefficients for the subset of panel members (j) in question are constrained to zero ($\beta_{x_j,t-k} = 0$). The residual sum of squared residuals from this restricted equation (RSS_4) is again compared to those from the unrestricted model (RSS_1) to produce the F_{henc}^j test statistic:

$$F_{\text{henc}}^j = \frac{(RSS_4 - RSS_1) / (n_{nc}p)}{RSS_1 / [NT - N(1 + p) - n_c p]} \quad 13$$

At this juncture, a significant F_{henc}^j test statistic allows for the rejection of the *Non-Causality Hypothesis* for sub-group (j) and the inference that x Granger causes y for this subset of panel members. We now apply this panel Granger procedure to test one recent theory of political development in the post-World War II South.

¹³ n_{nc} =number of panel members where β is constrained to 0. n_c =number of panel members where β is not constrained to 0.

Substantive Application: Understanding the Transformation of the Southern Party System

Theoretical Underpinnings

Though dramatic, the rise of Southern Republicanism and the electoral mobilization of Southern blacks were not unexpected. Key's (1949) work alludes to this transformation, and by the end of the 1970s, both dynamics were the foci for a considerable body of scholarship (see Black and Black 2002 for a description of this literature). Undoubtedly, national political dynamics played an important role in the formative stages of the development of Southern Republicanism (see Black and Black 1987, 1992, and 2002 and Carmines and Stimson 1989), but national level forces cannot fully explain sub-regional variation in GOP growth or the electoral mobilization of Southern blacks.¹⁴

Until recently, state-level explanations of Republican growth focused solely on demographic and economic factors such as immigration, the transformation and growth of the Southern economy, the growth in religious conservatism, and racial context. The literature had no *political* explanation for an inherently political transformation. One new perspective, *relative-advantage* theory (Hood, Kidd, and Morris 2004), does offer one such example of a political explanation. This theory provides a mechanism to explain the political behavior of both African Americans and conservative whites in the post-VRA South. In order to fully develop this explanation, we must first briefly discuss the political dynamics of the pre-VRA South before outlining the political behavior that the theory would lead us to expect in a post-VRA South.

¹⁴Hood, Kidd, and Morris (2004), Nadeau and Stanley (1993), Rhodes (2000) and Shafer and Johnston (2001) provide useful overviews of this literature.

In the one-party, pre-VRA South, the Democratic Party served as the institutional vehicle both for white power and black disenfranchisement. While the goals of conservative whites were surely many and varied, prominent among them was the maintenance of their political power via the prevention of black political empowerment (see Key 1949). Blacks in the pre-VRA South obviously had different goals. Blacks sought political power by advocating and protesting for the removal of barriers to free political competition. Though blacks received some support from outside forces—the federal government and the national Democrats—pre-VRA, the political dynamics in the South remained opposed to the political participation of blacks.

Passage of the VRA in 1965 completely transformed the political dynamics of the region by breaking down the institutional and legal barriers that had limited black political participation. Suddenly, a situation very similar to free trade existed in that the artificial constraints to mobilization began to be removed. The theory of *relative advantage* advances expectations about the response of white conservatives and blacks to this new political situation.

Relative advantage theory predicts that blacks would mobilize as Democrats as institutional and legal barriers to political participation begin to disintegrate. Blacks in the region aligned with the Democratic Party for two reasons. First, it was the national Democrats who made passage of the VRA possible and who advocated for black political enfranchisement in the South. Additionally, for blacks the *relative advantage* of aligning with the Democratic Party was much greater than aligning with the GOP because the Republican Party in the South was almost non-existent and lacked any political power. If political power was their goal, then it made little sense to seek that power via the Republican Party in the South.

For conservative whites *relative advantage* theory also predicts two actions, namely movement away from the Democratic Party and migration toward the GOP. White conservatives

were not interested in sharing power with blacks within the Democratic Party. In addition, white conservatives felt increasingly alienated from national Democrats over the VRA and civil rights in general. Again, the *relative advantage* of the Republican Party increased for conservative whites as the relative advantage of the Democratic Party (increasingly populated by blacks) decreased. In addition, the national Republicans (via Goldwater, Nixon, and others) appeared more and more sympathetic to conservative whites in the region.

Thus, *relative advantage* theory argues that both blacks and conservative whites responded to the *political* opportunities and constraints available in the late-twentieth century South in straightforward and understandable ways. As blacks mobilized and became Democrats, it became increasingly difficult for white conservatives to maintain control of the party. In increasing numbers, white conservatives left for the relatively more attractive Republican Party (see Heard 1952). As conservative whites left the Democratic Party, the opportunities for southern blacks in the Democratic Party increased. With the Democratic Party an increasingly attractive option, southern blacks mobilized in greater numbers. In a straightforward extension of the theory of *relative advantage*, the usefulness of the Republican Party increases over time for white conservative voters as the attractiveness of the Democratic Party decreased. *At the same time and because of that*, the attractiveness of political mobilization, and membership in, the Democratic Party increases over time for black voters. This mobilization dynamic is most likely to play out in those Southern states with the largest black populations—especially those with black populations large enough (if mobilized) to control the Democratic Party in an increasingly Republican environment.¹⁵

¹⁵These would be the deep South states or Alabama, Georgia, Louisiana, Mississippi, and South Carolina.

To investigate the potentially endogenous relationship between GOP growth and black mobilization in the South, we turn to a panel Granger analysis to examine the extent to which: (1) GOP growth Granger causes black mobilization and (2) black mobilization Granger causes GOP growth for the Southern states from 1960-2004. We provide a fuller description of our data in the next section.

Variable Operationalization

For this study, the former states of the Confederacy serve as our unit of analysis, producing a total of 11 panels. For each Southern state we have collected biennial data over a 44-year time period, from 1960 through 2004, for two variables.¹⁶ The first of these variables, *Black Electoral Mobilization*, taps into the potential influence that the political mobilization of blacks may have produced in regard to politics in the region. *Black Electoral Mobilization* is calculated at the state level as the number of black registered voters divided by the total number of registered voters, or:

$$\text{Number of Blacks Registered to Vote} / \text{Total Number of Registered Voters}^{17}$$

Operationalized as it is, our measure of black electoral strength places blacks within the context of the existing electorate—a much more precise method for estimating the potential influence of blacks as an electoral presence than alternative indicators (i.e. the percentage of blacks registered to vote).

The second indicator of interest, *GOP Strength*, is measured at the state level utilizing a index developed by David (1972). General election vote percentages for Republican candidates

¹⁶Our time series consist of two-year election cycles (i.e. 1960, 1962,...2004).

¹⁷Interpolation was used to fill in the gaps between missing years for both the number of blacks who were registered to vote and for the total number of registered voters in each Southern state. [Data Sources: *VEP News* (Various Years); *Statistical Abstract* (Various Years); Current Population Reports: P-20 Series on Voting and Registration (Various Years)].

in gubernatorial, senate, and congressional elections were utilized to create a composite state-level index of GOP strength.¹⁸ Following the construction of each GOP state index, a 10-year (5-time point) moving average was applied to smooth any sharp variations present in each series.¹⁹ The David Index of Party Strength was the method of choice for Lamis (1988) in his detailed study of party change in the South. Specifically, *GOP Strength* is calculated as:

$$\frac{(\% \text{ Republican Vote [Senate Election]} + \% \text{ Republican Vote [Gubernatorial Election]} + \% \text{ Republican Vote [Average Republican Congressional Vote]})}{3}$$

These two series are plotted by state from 1960 through 2004 in Figure 2, with *GOP Strength* represented by black squares and *Black Mobilization* represented by white triangles. As evidenced by this set of eleven plots there is a great deal of variation, both temporal and cross-sectional, for each of these series. The minimum and maximum values for *GOP Strength* are .01 (Mississippi-1960) and .57 (Tennessee-2004) respectively. For *Black Mobilization* the minimum value is .04 found in 1960 Mississippi, while the maximum value of .38 is also associated with Mississippi in 1994.

<Figure 2 about here>

¹⁸Estimates from 1960 through 1970 are obtained from David's work, while estimates for the remaining years are calculated by the authors. [Data Sources: David (1972); *Guide to U.S. Elections* (1994); *America Votes* (Various Years)].

¹⁹Comparisons between our measure of Republican Party strength and actual party registration data from Louisiana and Florida from 1950-2000 (the only two Southern states that did track party registration during the time of our study) indicate a high level of congruity ($r = .94$ for LA and $.94$ for FL) [Data available from the authors upon request].

²⁰Special transformations had to be made for Louisiana for each election following the 1978 institution of an open primary system. We used the following method to calculate our index of GOP party strength for 1978 through 2004:

1. If there was only one election (open primary): $\text{GOP} = \text{Percent of Total Republican Vote (Including votes won by other Republican Candidates in the Primary)}$
2. If there was both a primary and a general election and
 - a. The general election contained both a Republican and a Democrat: $\text{GOP} = \text{Percent of Total Vote Won by Republican Candidate}$
 - b. The general election contained two Democratic candidates: $\text{GOP} = 0\%$
 - c. The general election contained two Republican candidates: $\text{GOP} = 100\%$

Testing for Nonstationarity

Before proceeding with the panel Granger tests outlined above, we need to establish that both panel series are stationary (do not contain a unit-root). We utilize two different tests designed to detect the presence of unit-roots specifically in panel data. Table 1 presents test statistics from the Levin, Lin, and Chu and the Im, Pesaran, and Shin procedures, both of which indicate that nonstationarity is not an issue for either series.

<Table 1 about here>

Causality Testing

The *Homogenous Non-Causality Hypothesis* is the first test conducted. In our case, we want to know if:

$$\text{GOP Strength}_{(i,t-k)} \xrightarrow{\text{granger causes}} \text{Black Electoral Mobilization}_{(i,t)}$$

and if

$$\text{Black Electoral Mobilization}_{(i,t-k)} \xrightarrow{\text{granger causes}} \text{GOP Strength}_{(i,t)}$$

collectively for the eleven states in our sample. In order to test the *Homogenous Non-Causality Hypothesis* we calculated the F_{hnc} test statistics using the sum of squared residuals from an unrestricted model defined below:

$$\begin{aligned} y_{i,t} = & \beta_{i,t-1}y_{i,t-1} \dots \beta_{i,t-k}y_{i,t-k} + \beta_{AL,t-1}x_{AL,t-1} \dots \beta_{AL,t-k}x_{AL,t-k} + \beta_{AR,t-1}x_{AR,t-1} \dots \beta_{AR,t-k}x_{AR,t-k} + \\ & \beta_{FL,t-1}x_{FL,t-1} \dots \beta_{FL,t-k}x_{FL,t-k} + \beta_{GA,t-1}x_{GA,t-1} \dots \beta_{GA,t-k}x_{GA,t-k} + \beta_{LA,t-1}x_{LA,t-1} \dots \beta_{LA,t-k}x_{LA,t-k} + \\ & \beta_{MS,t-1}x_{MS,t-1} \dots \beta_{MS,t-k}x_{MS,t-k} + \beta_{NC,t-1}x_{NC,t-1} \dots \beta_{NC,t-k}x_{NC,t-k} + \beta_{SC,t-1}x_{SC,t-1} \dots \beta_{SC,t-k}x_{SC,t-k} + \\ & \beta_{TN,t-1}x_{TN,t-1} \dots \beta_{TN,t-k}x_{TN,t-k} + \beta_{TX,t-1}x_{TX,t-1} \dots \beta_{TX,t-k}x_{TX,t-k} + \beta_{VA,t-1}x_{VA,t-1} \dots \beta_{VA,t-k}x_{VA,t-k} + \\ & \beta_{AL} + \beta_{AR} + \beta_{FL} + \beta_{GA} + \beta_{LA} + \beta_{MS} + \beta_{NC} + \beta_{SC} + \beta_{TN} + \beta_{TX} + \beta_{VA} + \\ & \varepsilon_{i,t-1} \dots \varepsilon_{i,t-k} \end{aligned}$$

where the slope coefficients for subsequent lags within states are constrained to equality (i.e. $\beta_{AL,t-1}x_{AL,t-1} = \beta_{AL,t-k}x_{AL,t-k}$; $\beta_{AR,t-1}x_{AR,t-1} = \beta_{AR,t-k}x_{AR,t-k}$...) and a second model identical to the one specified in Equation 1, where the slope coefficients and subsequent lags for all the states in our sample are constrained to 0 (i.e. $\beta_{AL,t-1}x_{AL,t-1} \dots \beta_{AL,t-k}x_{AL,t-k} = 0$; $\beta_{AR,t-1}x_{AR,t-1} \dots \beta_{AR,t-k}x_{AR,t-k} = 0$...).

$1 \dots \beta_{AR,t-k} X_{AR,t-k} = 0 \dots$).²¹ Again, a significant *Homogenous Non-Causality Hypothesis* test statistic indicates that for at least one (and possibly all) of the states in our analysis, x Granger causes y.

The *Homogenous Non-Causality* test statistics are presented in Table 2. The first test of the *Homogenous Non-Causality Hypothesis* analyzes whether *GOP Strength*, for the members of our panel collectively, Granger causes *Black Mobilization*. The F_{hnc} test statistic is statistically significant at one lag (t-1), allowing us to reject the homogenous non-causality hypothesis. So for at least one state (and possibly all), there is statistical evidence that *GOP Strength* does Granger cause *Black Mobilization*. Test statistics, however, are insignificant at two and three lags, an indication that there is little evidence to support the claim that this relationship exists beyond a single lag period.

<Table 2 about here>

The second half of Table 2 details the *Homogenous Non-Causality* test statistics used to examine the hypothesis that *Black Mobilization* Granger causes *GOP Strength*. At one lag, the F_{hnc} test statistic is significant, indicating again that this causal process is at work in one or more of the states in our panel. Again, there appears to be no evidence that this relationship exists in subsequent time periods (i.e. t-2, t-3). In summary, we can reject the *Homogenous Non-Causality Hypothesis* for the relationships under study and proceed next to determine whether the causal process is heterogeneous or homogenous across all eleven states of the South.

In order to test the *Homogenous Causality Hypothesis*, we calculate another set of test statistics, hereafter referred to as F_{hc} , using the sum of squared residuals from the unrestricted model specified above and those from a model where the slope coefficients are constrained to equality for all states and lag periods (i.e. $\beta_{AL,t-1} X_{AL,t-1} = \beta_{AL,t-k} X_{AL,t-k} = \beta_{AR,t-1} X_{AR,t-1} = \beta_{AR,t-k} X_{AR,t-k} \dots$).

²¹For all the models specified, lagged values of y should be constrained to equality ($\beta_{i,t-1} Y_{i,t-1} = \beta_{i,t-k} Y_{i,t-k}$).

Acceptance of the *Homogenous Non-Causality Hypothesis* (insignificant test statistic) indicates that the causal process is homogenous for all the units in our panel. At this juncture, if the *Homogenous Causality Hypothesis* is confirmed, further testing is unnecessary as x is said to Granger cause y for all the states in our sample. On the other hand, rejecting the *Homogenous Causality Hypothesis* (significant test statistic) would indicate that for at least one or more states ($n \geq 1$), x does not Granger cause y .

The results of our tests for the *Homogenous Causality Hypothesis* are located in Table 3. Again, the bi-directional relationship between *Black Mobilization* and *GOP Strength* is examined up to three lag periods. In the case of *GOP Strength* Granger causing *Black Mobilization*, the *Homogenous Causality Hypothesis* is rejected at one lag. Therefore, we must conclude that the causal process in this case is heterogeneous, or does not exist across all the states in our sample. Reversing the equation, however, we find that the *Homogenous Causality Hypothesis* is accepted in the case of *Black Mobilization* Granger causing *GOP Strength*. So, for this relationship, the causal process appears to be sufficiently homogenous across all eleven states, making further examination unnecessary.

<Table 3 about here>

In order to determine which states in our panel contribute to the causal finding leading from *GOP Strength* to *Black Mobilization* we must utilize the *Heterogeneous Non-Causality Hypothesis*. Rejection of the *Heterogeneous Non-Causality Hypothesis* indicates the presence of a causal relationship for the state under consideration. The F_{henc}^i test statistics are calculated using the sum of squared residuals from the unrestricted model in Equation 1 and a second model in which the slope coefficients and subsequent lags for the state under testing are constrained to 0 (i.e. $F_{\text{henc}}^{\text{AL}}: \beta_{\text{AL},i-1}X_{\text{AL},i-1} \dots \beta_{\text{AL},i-k}X_{\text{AL},i-k} = 0$). We present state by state results in the upper portion of

Table 4 for one lag period (t-1).²² The test results indicate that for Alabama, Georgia, Louisiana, Mississippi, North Carolina, and South Carolina the relationship *GOP Strength* Granger causes *Black Mobilization* appears to hold. For the remaining five states in our study there does not appear to be enough statistical evidence to reject the *Heterogeneous Non-Causality Hypothesis*.

<Table 4 about here>

What one quickly notices about these two sets of states is that they divide nicely into two well-identified geographic subregions, namely the deep and rim South. The sole exception is North Carolina, typically considered a rim South state. The bottom portion of Table 4 tests the *Heterogeneous Non-Causality Hypothesis* by subregion. By convention, we classify North Carolina as a rim state along with Arkansas, Florida, Tennessee, Texas, and Virginia with the remaining states comprising the deep South. In order to determine whether *GOP Strength* Granger causes *Black Mobilization* for each subregion, we derive the F_{henc}^j using the sum of squared residuals from Equation 1 and from a second model in which the slope coefficients for the states comprising the subregion are constrained to 0 (i.e. $F_{\text{henc}}^{\text{Deep}}: \beta_{\text{AL},t-1}X_{\text{AL},t-1} = 0; \beta_{\text{GA},t-1}X_{\text{GA},t-1} = 0; \beta_{\text{LA},t-1}X_{\text{LA},t-1} = 0; \beta_{\text{MS},t-1}X_{\text{MS},t-1} = 0; \beta_{\text{SC},t-1}X_{\text{SC},t-1} = 0$).²³ As indicated, the *Heterogeneous Non-Causality Hypothesis* is rejected collectively for the deep South states and accepted for the rim states. So, testing by subregion one may conclude that at t-1, *GOP Strength* Granger causes *Black Mobilization* in the deep South, but not the rim South.

²²There is little evidence that this causal relationship exists beyond the first lag period (see Table 2). We therefore restrict tests of the *Heterogeneous Non-Causality Hypothesis* to t-1.

²³In this case we only calculate the F_{henc}^j test statistic to one lag (t-1).

Discussion of Results

In summary, we find that *Black Mobilization* Granger causes *GOP Strength* and that *GOP Strength* Granger causes *Black Mobilization*. The former causal process can be characterized as homogenous for the eleven states in our sample. The later process, conversely, is heterogeneous, existing in the deep South (and North Carolina), but not in the rim states. In the case of the rim South, the causal process is one-sided and can be characterized as:

$$\text{Black Electoral Mobilization}_{(\text{Rim}, t-1)} \xrightarrow{\text{granger causes}} \text{GOP Strength}_{(\text{Rim}, t)}$$

In the case of the deep South states, this process is reciprocal:

$$\text{Black Electoral Mobilization}_{(\text{Deep}, t-1)} \xrightarrow{\text{granger causes}} \text{GOP Strength}_{(\text{Deep}, t)}$$

$$\text{GOP Strength}_{(\text{Deep}, t-1)} \xrightarrow{\text{granger causes}} \text{Black Electoral Mobilization}_{(\text{Deep}, t)}$$

Not only is there a geographical differentiation associated with these subregions, but a substantive one as well. While the size of the black population is much higher in the South compared to other regions of the United States, within the South the highest concentrations of black citizens are located in the deep South states. This fact becomes clear looking at Figure 3, which arrays the eleven states in the region according to the relative size of their average black populations from 1960 through 2000. The average black population in the region varies from a high of 37% in Mississippi to a low of 12% in Texas.

<Figure 3 about here>

The horizontal line in Figure 3 separates states based on the significance of their F_{henc} test statistics in Table 4. These test statistics were significant for those states above the horizontal line and insignificant for the reminder located below this division. This line also suggests a potential threshold for the noted counter-mobilization effect to manifest itself. In states with an

average black population over 20%, expansion of the Republican Party produced increased levels of mobilization within the black electorate. Below this level, it is possible that a necessary critical mass does not exist to trigger the noted counter-mobilization reaction found in the deep South states.

As Key noted decades ago:

The range of the Negro population—from 49.2 percent in Mississippi to 14.4 percent in Texas—suggests that even “the South” is by no means homogenous and that if the Negro influences the politics of the South, there ought to be wide variations in political practices from state to state (1949:10-11).

This variation has traditionally been thought of in quantitative terms. We suggest that a qualitative distinctiveness also exists, and that the theory of *relative advantage* explains why. These results, taken in tandem, suggest the transformation of Southern politics during the last half of the twentieth century can be viewed from a theoretical perspective that attributes the same logic of action to both blacks and conservative whites. This is a simple and straightforward feedback loop (in over half of the Southern states), but it is also a novel characterization of the twin pillars of the transformation of Southern politics: Republican growth and black mobilization.

With few exceptions, research on Republican growth in the South has focused on other types of causal explanations including economic dynamics, religious or cultural orientations, migration patterns, or the geographic concentration of blacks (i.e. the black-belt hypothesis), while largely overlooking more overtly political explanations. Similarly, existing work on black mobilization in the South rarely focuses explicitly on the party dynamics highlighted in this paper.

In an important respect, our theoretical perspective (and results) is consistent with the black empowerment literature (Bobo and Gilliam 1990; Browning, Marshall, and Tabb 1984; and Harris, Sinclair-Chapman, McKenzie 2005). However, instead of focusing on the outcome

produced by black mobilization—the election of black officials (in the South)—and then viewing that outcome as an inducement to further black mobilization, we focus on a more primary strategic dynamic: the change in the benefits for blacks of Democratic Party membership and activism (and, implicitly, electoral mobilization). Clearly, Republican growth (and the exodus of white conservatives from the Democratic Party) opened up significant opportunities for blacks.²⁴ Our research suggests that Southern blacks saw these opportunities—where they were greatest—and took advantage of them.

Conclusion

The utilization of panel data for the exploration of political phenomena has grown exponentially over the last decade. Diagnostic tests and other extensions to deal with known problems have sometimes been slower to develop. This manuscript examines the use of one such extension: the application of Granger causality tests to panel data. In addition, the panel Granger tests presented allow one to control for, and detect, the possibility that the causal process, if present, is not homogenous across all members of the panel under study (i.e. heterogenous causality). The application of Granger causality tests to panel data can help inform researchers both about the nature of causal relationships between sets of variables and the extent to which such relationships are heterogenous or homogenous across panel members prior to the application of more extensive multivariate modeling techniques.

In the case of the application presented in the previous section, the logical next step would involve specifying a set of models within a multivariate framework in order to determine if the causal relationships uncovered stand up to the addition of control variables. Having

²⁴For example, in 2004 blacks comprised 58% of the turnout in the South Carolina Democratic Primary and 47% in the Georgia Democratic Primary (Bullock and Gaddie 2005a, 2005b).

already determined the nature of the causal process between the two primary variables of interest, development of such multivariate models can, therefore, proceed with this knowledge in hand.

We now know that a non-recursive relationship exists between these two factors of interest, however, this two-way causal flow is not homogenous across all the states in our sample. Again, for the rim South states there is no endogeneity issue, as *Black Mobilization* was found to Granger cause *GOP Strength*, with no evidence for the converse. For those states located in the deep South the panel Granger tests employed did point to evidence of a non-recursive relationship between these two variables of interest. In formulating multivariate explanatory models the best course of action would call for separate analyses, one for the panel of deep South states and another for the rim South panel. For the latter, instrumental variables will need to be employed to alleviate known problems associated with the use of endogenous regressors, while for the former a more traditional panel regression framework can be utilized (see Baltagi 2005 for a discussion of panel models using instrumental variables).

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Table 1. Panel Unit-Root Tests

Test	Test Statistics
<i>Levin, Lin, and Chu:</i>	
GOP Strength	-2.93 ^{***}
Black Mobilization	-4.06 ^{***}
<i>Im, Pesaran, and Shin:</i>	
GOP Strength	-1.99 ^{**}
Black Mobilization	-2.23 ^{***}

Notes: * p<.10; ** p<.05; *** p<.01

For both tests, a significant test statistic allows for the rejection of the null hypothesis that the series is nonstationary.

Table 2. Homogenous Non-Causality Test Statistics

Lags	F_{hnc}
GOP Strength Granger Causes Black Mobilization	
t-1	3.214***
t-2	.532
t-3	.668
Black Mobilization Granger Causes GOP Strength	
t-1	1.652*
t-2	1.197
t-3	.979

Notes: * p<.10; ** p<.05; *** p<.01

Critical Values: F_{hnc} : Based on an F-Distribution with $Np, NT-N(1+p)-p$ degrees of freedom (Hurlin and Venet 2001)

Table 3. Homogenous Causality Test Statistics

Lags	F_{hc}
GOP Strength Granger Causes Black Mobilization	
t-1	1.966**
t-2	.493
t-3	.594
Black Mobilization Granger Causes GOP Strength	
t-1	.748
t-2	.559
t-3	.619

Notes: * p<.10; ** p<.05; *** p<.01

Critical Values: F_{hc} : Based on an F-Distribution with $Np, NT-N(1+p)-p$ degrees of freedom (Hurlin and Venet 2001)

Table 4. Heterogenous Non-Causality Test Statistics:
 GOP Strength Granger Causes Black Mobilization (t-1)

State	F _{henc}
Alabama	7.500***
Arkansas	.190
Florida	.002
Georgia	13.932***
Louisiana	10.008***
Mississippi	16.660***
North Carolina	4.752***
South Carolina	3.736***
Tennessee	.550
Texas	.121
Virginia	1.38
Sub-Region	
Deep	6.464***
Rim	1.147

Notes: * p<.10; ** p<.05; *** p<.01

Critical Values: F_{henc}: Based on an F-Distribution with Np, NT-N(1+p)-p
 degrees of freedom (Hurlin and Venet 2001)

Deep South: AL, GA, LA, MS, SC

Rim South: AR, FL, NC, TN, TX, VA

Figure 1. Granger Causality Testing for Panel Data

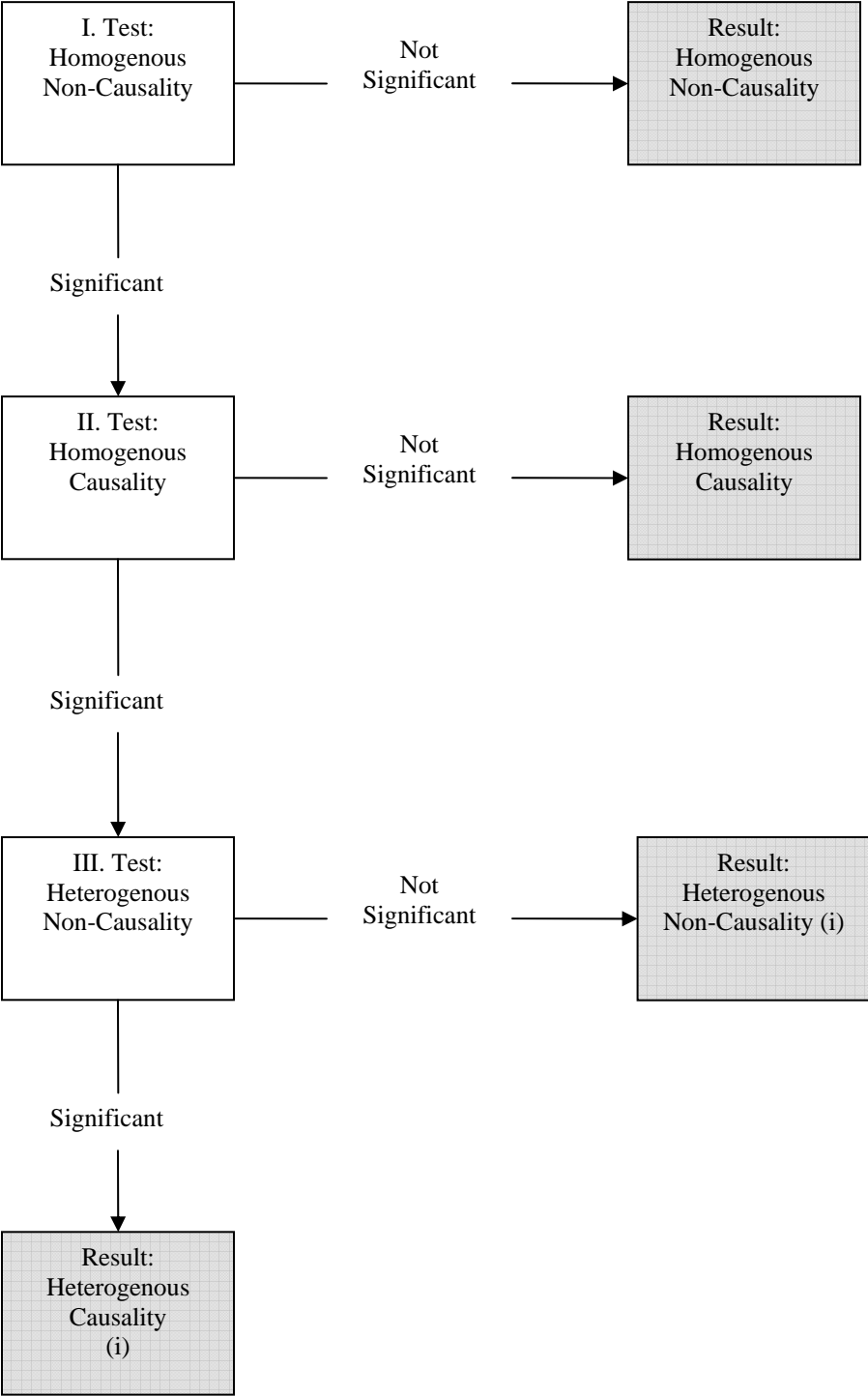
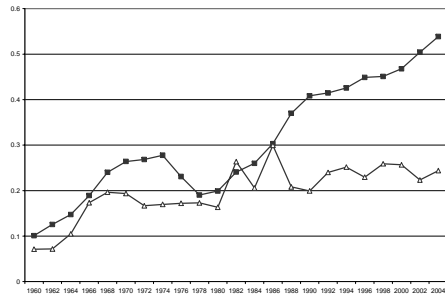
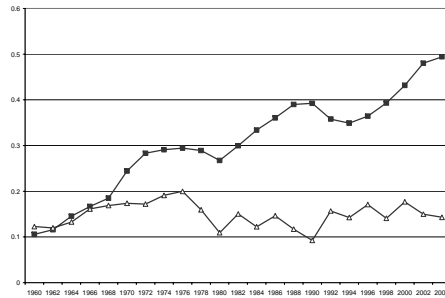


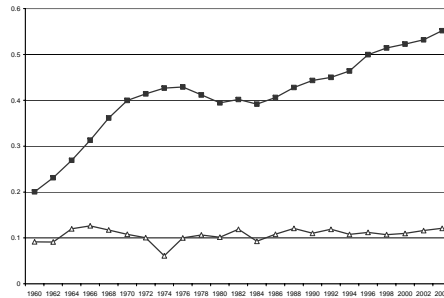
Figure 2. GOP Strength and Black Mobilization by State, 1960-2004



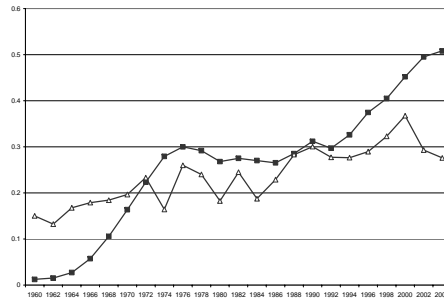
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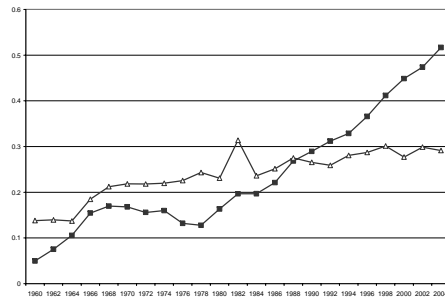
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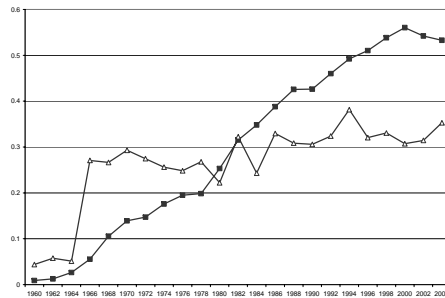
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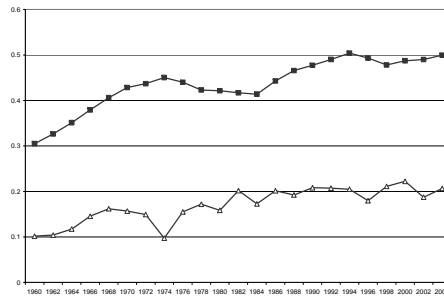
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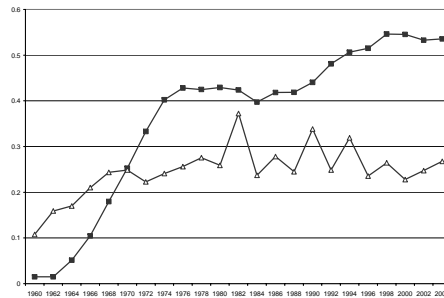
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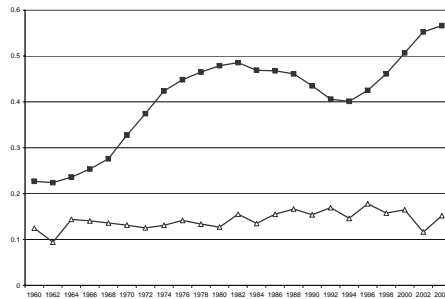
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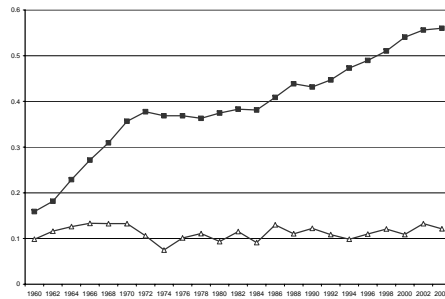
North Carolina



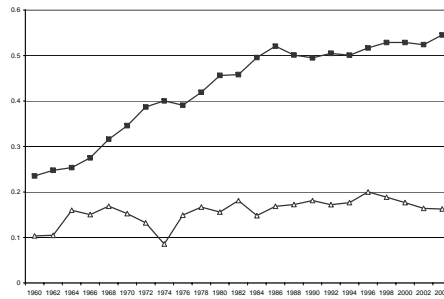
South Carolina



Tennessee



Texas



Virginia

Figure 3. Mean Black Population by State, 1960-2000

