A Spatial-Temporal Approach to Surveillance of Prostate Cancer Disparities in Population Subgroups

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Background: Prostate cancer mortality disparities exist among racial/ethnic groups in the United States, yet few studies have explored the spatiotemporal trend of the disease burden. To better understand mortality disparities by geographic regions over time, the present study analyzed the geographic variations of prostate cancer mortality by three Texas racial/ethnic groups over a 22-year period.

Methods: The Spatial Scan Statistic developed by Kulldorff et al was used. Excess mortality was detected using scan windows of 50% and 90% of the study period and a spatial cluster size of 50% of the population at risk. Time trend was analyzed to examine the potential temporal effects of clustering. Spatial queries were used to identify regions with multiple racial/ethnic groups having excess mortality.

Results: The most likely area of excess mortality for blacks occurred in Dallas-Metroplex and upper east Texas areas between 1990 and 1999; for Hispanics, in central Texas between 1992 and 1996; and for non-Hispanic whites, in the upper south and west to central Texas areas between 1990 and 1996. Excess mortality persisted among all racial/ethnic groups in the identified counties. The second scan revealed that three counties in west Texas presented an excess mortality for Hispanics from 1980–2001. Many counties bore an excess mortality burden for multiple groups. There is no time trend decline in prostate cancer mortality for blacks and non-Hispanic whites in Texas.

Conclusion: Disparities in prostate cancer mortality among racial/ethnic groups existed in Texas. Central Texas counties with excess mortality in multiple subgroups warrant further investigation.

Key words: health disparities ■ prostate cancer ■ spatial analysis ■ geographic information system

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BACKGROUND

Prostate cancer is among the most often diagnosed cancers in the United States, as it constitutes the second leading cause of cancer deaths among U.S. men.[1] More than 41,000 Americans died of prostate cancer in 2004.[2] Statistical studies have identified the racial/ethnic disparities of prostate cancer mortality across regions and over time. U.S. African-American males rank first and white men eighth in worldwide prostate cancer mortality rates.[3] National data indicate that prostate cancer mortality rates decreased during the 1990s, while the decreases were almost twice as great for whites and Asians/Pacific Islanders than for African Americans, American Indians/Alaska Natives and Hispanics.[4] Despite the evident general decline in prostate cancer mortality rates among all racial/ethnic groups during the '90s (possibly due to the wide adoption of prostate-specific antigen testing),[5] it is unclear whether the decline had any positive effect on those regions with an excess mortality. In Texas, for example, where approximately 11,000 new prostate cancer cases are diagnosed every year,[2] studies have found a decline in prostate cancer mortality consistent with national statistics in the late 1990s.[6] Despite the general decline in prostate cancer mortality, however, mortality disparities continue to exist by race and region. Texas-specific cancer research identified several counties in the Dallas-Fort Worth Metroplex region with an excess prostate cancer mortality between 1990 and 1997.[7] In terms of race, between 1970 and 1994 in the Dallas-Fort Worth state economic areas (SEAs), blacks had the highest rates of prostate cancer mortality, and the Dallas-Fort Worth,

Galveston and Potter/Randall SEAs had the highest rates among whites.[8] Abundant evidence relating to uneven geographic distribution of prostate cancer mortality underlines the importance of developing a more data-driven and evidence-based surveillance approach to monitoring potential health disparities, which renders an opportunity for a closer look at the distribution of mortality in diverse population groups over time. The purpose of the present study was to determine whether the declining prostate cancer mortality rates indicated regional variations of potential excess prostate cancer mortality over an extended period, and if so, at what level of excess mortality in terms of temporal duration, relative risk and affected regions by county. The information may be instrumental for planning for prostate cancer intervention and health resource allocation to address the regional health disparities.

METHODS

Location of the Study, Data Source and Management

The study was conducted in the state of Texas, a state with a diverse population, and included all counties in a study period between 1980 and 2001. All data were secondary and included prostate-cancer specific deaths, male population at risk and location. These data were identified from the following reliable sources and saved in separate files. The prostate-cancer-specific death cases file was obtained from an open source (Texas Vitalweb),[9] and included prostate cancer deaths (ICD-9 code 185 and ICD-10 code C61) by place of residence in 254 counties by four major racial/ethnic groups in Texas. These were coded as categorical data (representing blacks, Hispanics, non-Hispanic whites and others), indicating the four subgroups in each of the 22 study years. Each file contained an age-group categorical variable with values labeled 1–16, representing ages

Figure 1. Excess mortality of prostate cancer among Anglo males in Texas counties, 1980–2001

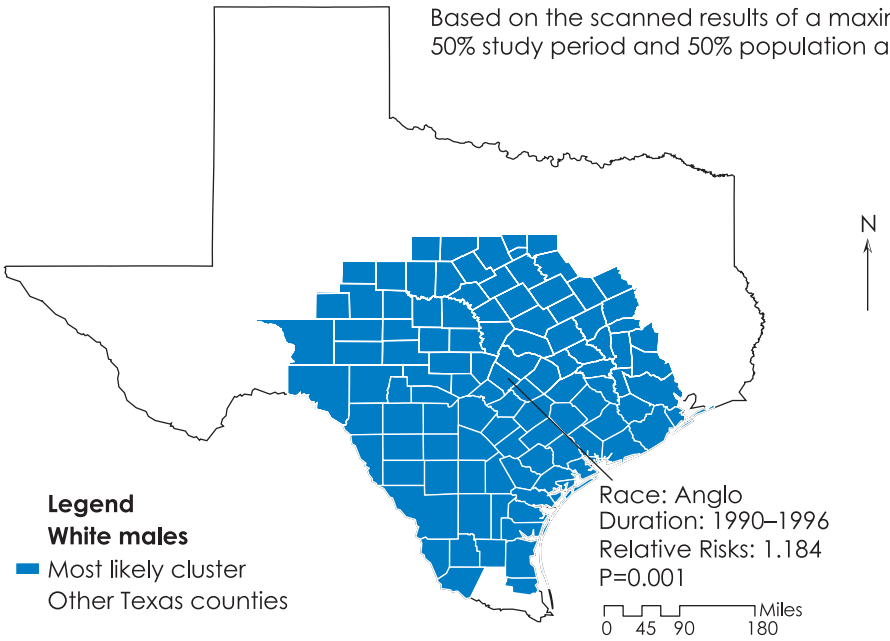


Table 1. Characteristics of male population under study and prostate cancer deaths, Texas, 1980–2001

Population	Average Total Population	Percent in All Population (%)	Cumulative Deaths	Percentage of Males	Annual Adjusted* Rate (Per 100,000)
All males	8,677,605	49.37	35,242	100	18.5
Black males	991,066	5.638	6,493	18.42	29.8
Hispanic males	2,305,930	13.12	3,450	9.79	6.8
Non-Hispanic white males	5,187,210	29.51	25,219	71.56	22.1
Other males	193,397	1.10	80	0.23	1.9

* For overall rate (i.e., the population including all subgroups), both age and race/ethnicity are adjusted, while for race-specific rates age is adjusted.

ranging from "0–4" to "≥75," grouped by five-year intervals. A total of 89,408 records for each of the case files reflected the number of prostate cancer deaths per population group among 16 age groups in 254 counties over the 22-year study period. The second file, named "population file," included populations at risk in the study period; i.e., the male population in each county by all study years with the respective race and age group information corresponding to the deaths file. Centennial population data, including race/ethnicity and age group, were obtained from the year 1990/2000 Census American FactFinder Website originated by the U.S. Census Bureau.[10] The other 20 years of population data were obtained from population estimates made available through the Texas State Data Center and the Center of Vital Statistics of Texas Department of Health.[11] The "population file" contained a total of 357,632 records, representing four racial/ethnic groups by 16 age groups in 254 counties for the 22-year study period. In this file, age group variables were also coded as categorical data to enable adjustments for age in the Texas population. The third file, the "geographic file," also obtained from the U.S. Census Bureau,[12] contained latitude and longitude information of Texas county centroids as a proxy indicating the locality of each county.

Table 2. Relative risk of prostate cancer according to age group and race in Texas, 1980–2001

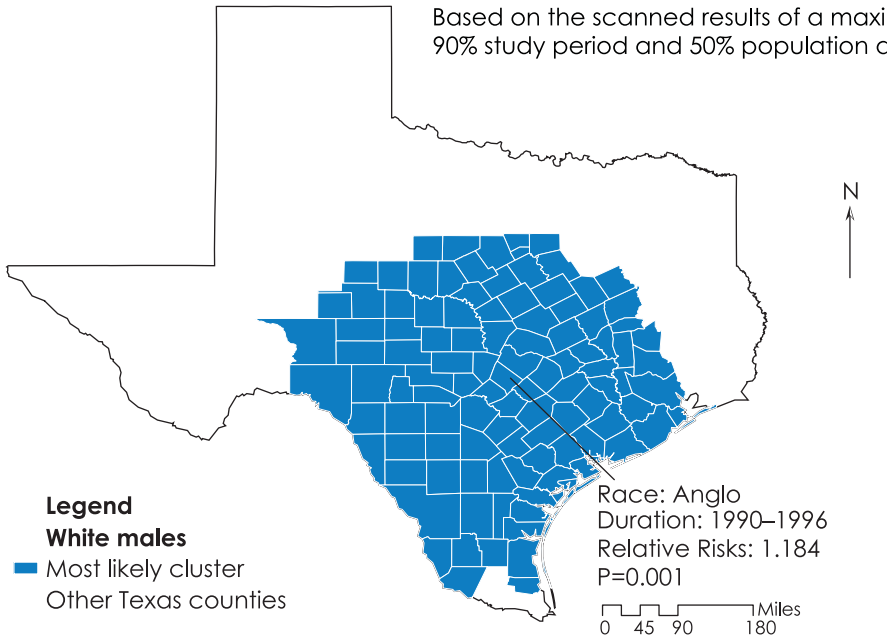
	Relative Risk	95% CI
Age Group		
20–39	0.05	(0.05, 0.06)
40–59	0.48	(0.47, 0.49)
60–69	referent	–
≥70	1.49	(1.46, 1.53)
Race		
Black	1.28	(1.25, 1.32)
Hispanic	0.67	(0.65, 0.69)
White	referent	–
Other	0.39	(0.35, 0.44)

Data Analysis

First, we evaluated the geographic excess of prostate cancer mortality in the selected population groups at the county level and characterized the excess burden by spatiotemporal variations represented by their respective relative risk, duration and p values indicating the homogeneity of prostate cancer mortality distribution. Second, we tested the potential persistence of excess deaths into the present decade. The resulting temporal patterns were compared with previous studies for a trend of declining mortality. Third, we performed spatial queries to identify the excess mortality affecting multiple subgroups over time. Based on the results of these analyses, we identified the racial/ethnic group(s) and the region(s) that might have been affected by a persistent excess mortality over time.

This study used the Spatial Scan Statistic developed by Kulldorff et al.[13-14] to detect potential excess mortality and adjust for covariates. The particular value of the spatiotemporal method is that it can detect "geographic" or "temporal" (or both) excess mortality that spanned only a limited portion of the full study period, or a selected time period within the study period as defined by the investigators. The method pinpoints the geo-

Figure 2. Excess mortality of prostate cancer among Anglo males in Texas counties, 1980–2001



graphic location, temporal duration, relative risk and statistical significance of the identified clusters. The method has been used elsewhere in similar studies of other types of cancer[15,16] and of accidental poisoning

Figure 3. Excess mortality of prostate cancer among black males in Texas Counties, 1980–2001

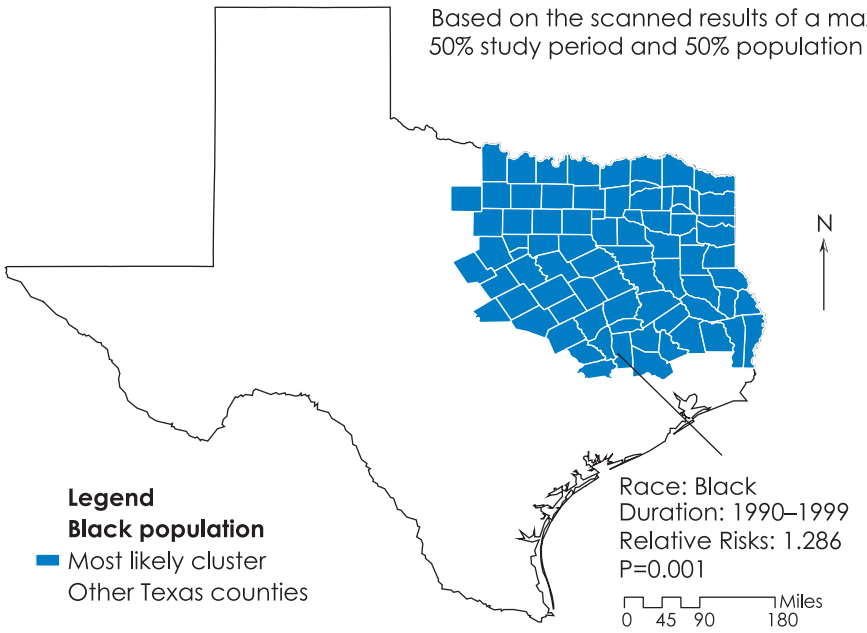


Table 3. Texas counties detected with excess prostate cancer mortality among non-Hispanic whites, blacks and Hispanics, 1980–2001

Population Subgroups	Years (Duration)	Number of Deaths	Annual Age-Adjusted Rate/100,000	P Value	Relative Risk	Number of Counties
Spatio-Temporal Excess Mortality						
Primary cluster for non-Hispanic white male population (50% and 90% study period, and 50% population at risk)	1990–1996 (7)	4,420	26.2	0.001	1.22	107
Primary cluster for black male population (50% and 90% study period, and 50% population at risk)	1990–1999 (10)	1,783	38.3	0.001	1.39	79
Primary cluster for Hispanic male population (50% of study period, and 50% population at risk)	1991–2001 (11)	1228	9.9	0.001	1.19	146
Secondary cluster for Hispanic male population (50% study period and 50% population at risk)	1992–1996 (5)	252	9.7	0.003	1.45	10
Primary cluster for Hispanic male population (90% of study period, and 50% population at risk)	1980–2001 (22)	523	9.8	0.001	1.52	3
Primary cluster for other male population (50% of study period, and 50% population at risk)	1989–1999 (11)	39	3.4	0.3	1.8	53

* Excess mortality is defined as up to 50% of study population, with temporal persistence of up to 50% or 90% of the study years. All included spatial only adjusting for time non-parametrically.

mortality disparities.[17] A "cluster" refers to a geographical area that during a specific timeframe has experienced a disproportionate excess in mortality when compared to surrounding areas.[18] A cluster can be either an increase or decrease in mortality, but this study only looked at increased mortality. This study used "clusters" and "excess mortality" interchangeably, with both terms referring to the statistical context of both spatial and temporal dimensions of excess mortality.

The key test of the spatial scan statistic proposes that by meeting the assumptions of a set of statistical models, the unusual increase (or reduction) of mortality in specific spatial and temporal windows (adjusted for risk factors) can be characterized by the affected regions,

Figure 4. Excess mortality of prostate cancer among black males in Texas counties, 1980–2001

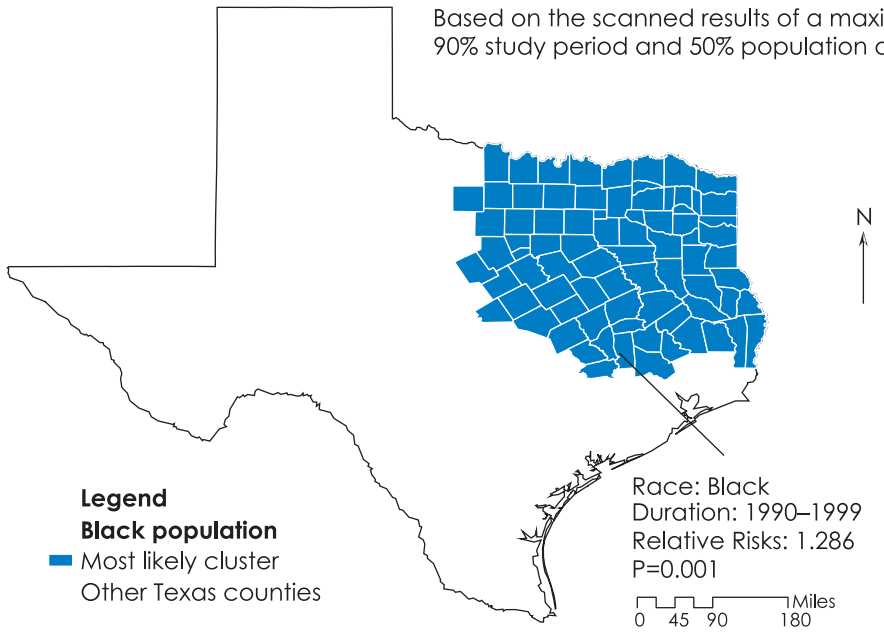
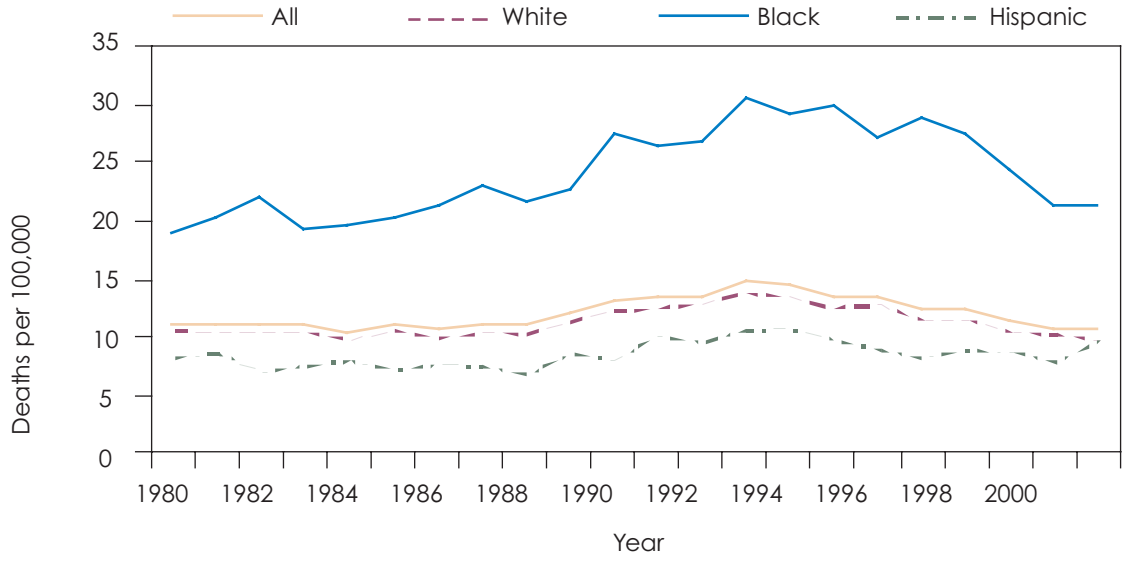


Exhibit 1. Age-adjusted prostate cancer mortality rates of Texas between 1980 and 2001 (standardized to 2000 U.S. population)



Decrease in mortality rates observed after 1998 may be reflective of the change in ICD 9/10 coding conventions. There is no significantly increasing or decreasing linear trend (RR=1.003, 95% confidence interval: 1.001, 1.004)

mortality rates, duration of excess, relative risk and statistical significance. The method factors in the uneven geographical population density and bases the analyses on the total number of observed prostate cancer deaths. In an analysis of relatively rare cases/deaths such as prostate cancer, the Poisson model can be used for estimating the probability distribution when the number of cases is small compared to the population at risk. The null hypothesis of the Poisson model provides that, when there are no confounding factors, the expected death counts in each county are proportional to its population size (or person-years) in that area. The alternative hypothesis is that deaths are not proportionately and randomly distributed.

In the present study, for each location and size of the scanned space and time, the null hypothesis is that the

Figure 5. Excess mortality of prostate cancer among Hispanic males in Texas counties, 1980–2001

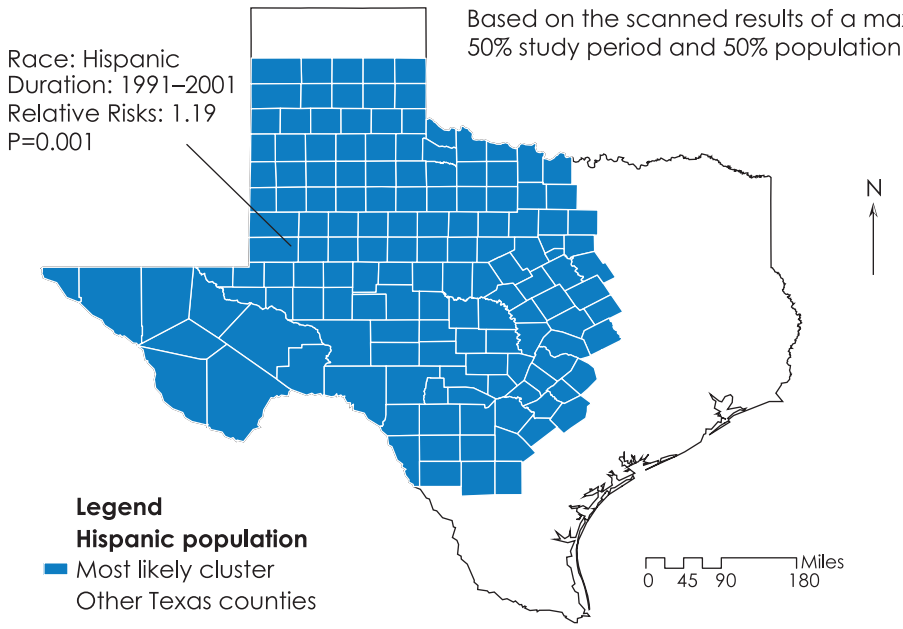
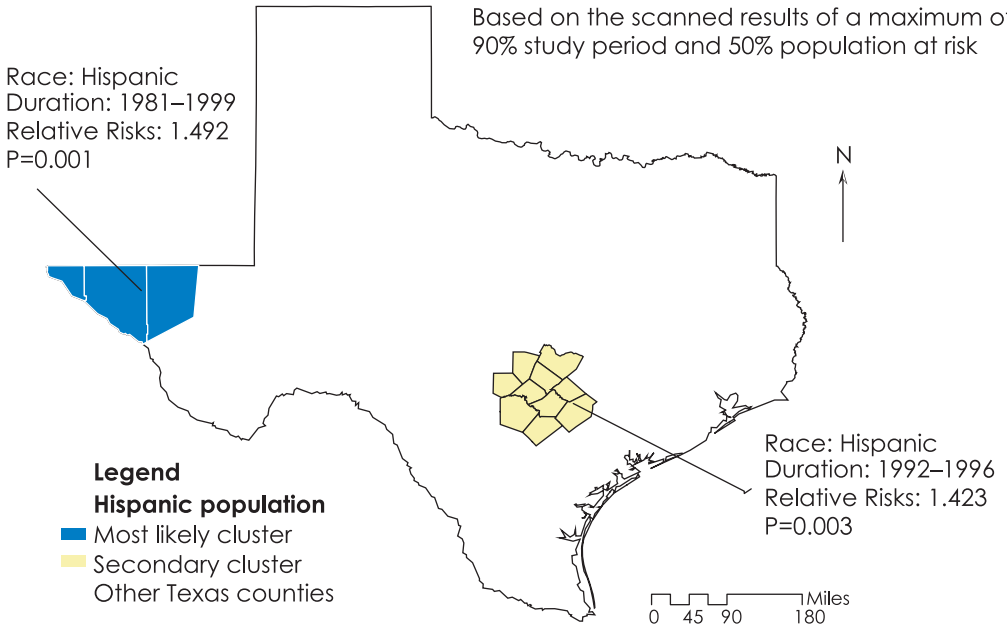


Figure 6. Excess mortality of prostate cancer among Hispanic males in Texas counties, 1980–2001



prostate cancer death rates in a scanned region are randomly distributed, meaning they do not exceed those outside the scanned region. The alternative hypothesis is that there are elevated cancer mortality rates within the scanned space and time as compared to outside areas, and that deaths are distributed heterogeneously in the entire study area, meaning that the deaths in the putative cluster of space and time exceeded those outside of the cluster.

Calculations were performed using SaTScan™ version 6.0.[18] Analytic approaches have been described elsewhere by the authors[17,19] and will only be briefly summarized here. The Poisson model was used to calculate expected deaths in each county. The space–time retrospective analysis was conducted without prior assumptions of the size, location or duration of excess mortality. First, the default scanning setting of a maximum spatial cluster size at 50% of the population at risk, and 50% of the study period (i.e., 11 years) was set. The "50% of population at risk" parameter was recommended by Kulldorff [18] as an optimal value setting that maximizes the effect of potential cluster detection. This would mean that a cluster would comprise at most 50% of the population at risk. The "study period" option was set at 50% and again at 90% for comparison. Secondly, the study further tested whether excess cases in any region may persist through the year 2000, by holding constant the 50% maximum spatial cluster but scanning for up to 90% of the study period of 19 years. Both analyses include spatial only with the adjustments of time trend nonparametrically to offset the effect of temporal trend. As implemented in SaTScan, the method employed for adjusting rate of age/race is indirect adjustment.

Appendix A describes the stepwise progression of the calculation. The output files, including cluster locations, the respective relative risk and the results of test statistic, were saved in database (.dbf format) files for exporting to geographic information systems (GIS) for

Appendix A. Descriptions of the stepwise progression of data processing

After downloading the cases/deaths, population and county centroids files, the VBA program:

1. Imports, aligns and exports data tables to SaTScan-compatible format: The program first creates three folders to be used as working directories and to save results of SaTScan analysis. They are "maps," "results" and "folders named by gender and race/ethnicity." It then imports all downloaded case/deaths files to the VBA, aligning and extracting data to a SaTScan-compatible format with each data table, including the fields of County ID and case/death numbers stratified by years, age, gender and race/ethnicity. Another data table with the same fields (but substituting cases/deaths with population numbers) is also prepared.

2. Deletes tables: After creating the cases and population files, all the downloaded files that were imported for this purpose in step 1 are then deleted. This step is necessary in order to reduce the size of the VBA and maximize the speed of ensuing steps. Once the files are deleted, the program invokes SaTScan scripts in the "scripts" folder.

3. Runs SaTScan: By invoking SaTScan scripts, the program opens four windows to perform four SaTScan concurrent sessions. These sessions include four windows of analysis, including two for all populations combined and two for non-Hispanic whites, each with windows of 50% spatial cluster and 50% temporal cluster, and 50% spatial and 90% temporal cluster, respectively.

4. Pauses: The program then pauses for 4,200 seconds (70 minutes) for SaTScan to process data. For this study, there were 725,000 records entered for processing.

5. Renames all GIS files to be Access compatible: After the SaTScan process is complete, the resultant GIS files are then renamed to MS-DOS compliant format of [filename].[extension] (for example, the default "all.gis.dbf" of the SaTScan output file was renamed "to all.dbf").

6. Imports GIS files to tables: To tabulate the results of cluster information, the program "imports" and "unions" (both SQL commands) all GIS files as one consolidated file and presents it in a report format.

7. Exports GIS files to map: The VBA then exports the aligned GIS result files to spatially join with Texas county boundary files and present the location of clusters in choropleth maps. It then invokes an ArcGIS map document of four data layers (representing "all population" "non-Hispanic white", "black" and "Hispanic" population by 50% population, 50% study year, and 50% population, 90% study year respectively) that were preformatted. For this study, the entire analysis took 70 minutes to run on a standard PC (1.5 MHz Pentium IV). The VBA outputs and prints all SaTScan result files upon the completion of the scan analysis.

further analysis. Data were stored in a Microsoft SQL Server version 7.0, and Visual Basic Application (VBA) code was developed by the first author to automate data manipulation, interface with SaTScan and output the results to a GIS. The automated process, from data input, scanning, to output, took a total of 70 minutes of computer time.

RESULTS

The study analyzed 35,242 prostate cancer deaths among an average population of 8,677,605 males in Texas for the 22-year time period. The age- and race-adjusted annual prostate cancer mortality rate was 18.5/100,000 men/year. Table 1 summarizes the characteristics of the study population and their subgroups. Blacks had the highest annual age-adjusted rate (29.8

Figure 7. Excess mortality of prostate cancer among "the other" males in Texas counties, 1980–2001

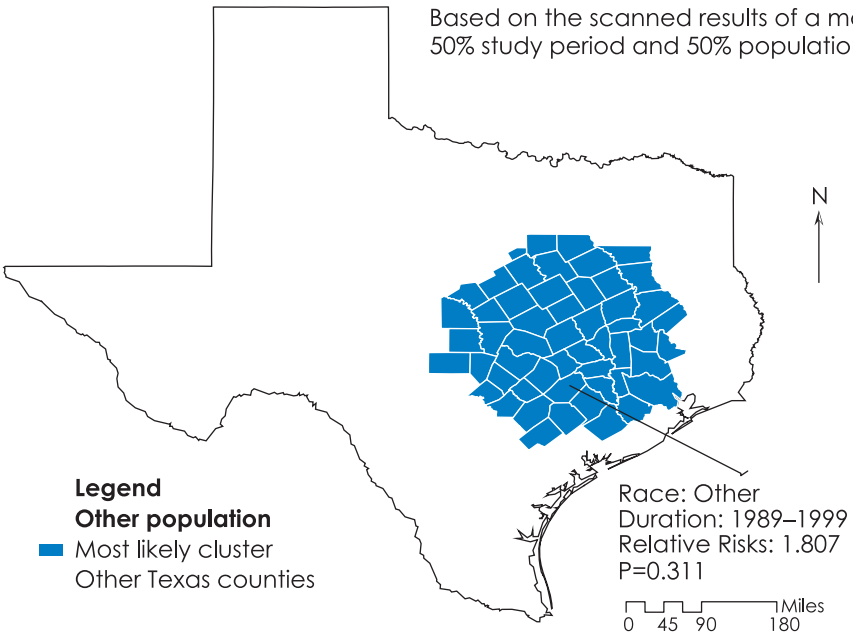
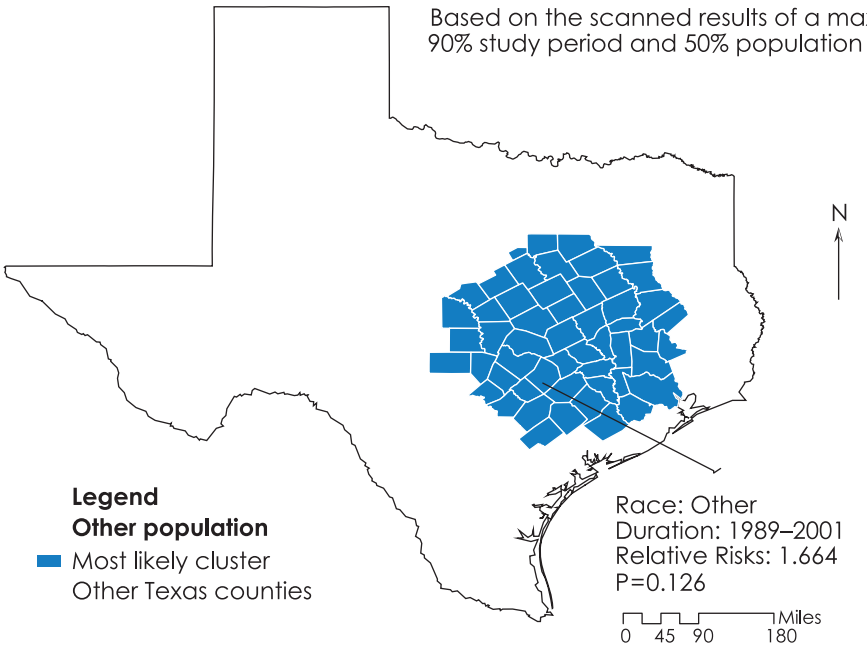


Figure 8. Excess mortality of prostate cancer among "the other" males in Texas counties, 1980–2001

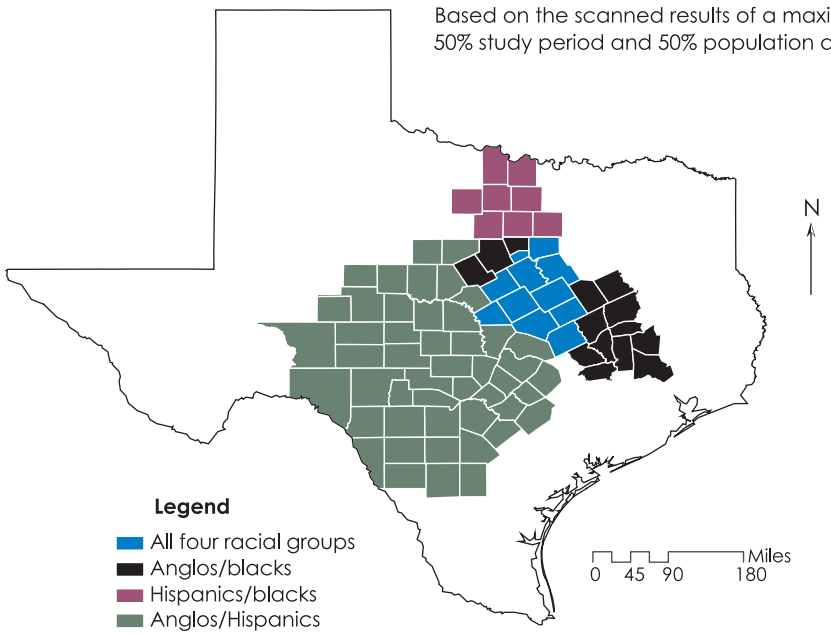


per 100,000). Exhibit 1 illustrates subgroup-specific rates over the study year and indicates that the rates in Texas counties were relatively stable over the 22-year study period. The risk of prostate cancer according to age group and race are presented in Table 2. The greatest risk was observed among black males and among people aged >70 years. However, the pattern of risk according to age group did not vary according to race/ethnicity.

With the adjustments of age and stratified by race, and with a scan window of a maximum of 50% of the study period (11 years) and 50% of the population at risk (4.4 million males in Texas), the first spatiotemporal analysis detected five regions of likely excess mortality in three racial/ethnic groups having statistical significance in terms of both spatial and temporal excess. Standardized mortality ratios ranged from 1.22–1.49. Figures 1, 3, 5 and 7 present these areas with likely excess mortality from the first analysis by each race, including: 1) the most likely (primary) cluster among each race, and 2) those clusters of statistical significance.

Table 3 summarizes the results of analysis for non-Hispanic whites, blacks and Hispanics by potential excess mortality, duration of occurrence, observed deaths, counties and relative risk information in each cluster of excess mortality. For the non-Hispanic white population, the most likely area of excess mortality with a relative risk (RR) of 1.22 (p=0.001) occurred between 1990 and 1996 and included 107 counties ranging from upper south Texas, west to central Texas. For blacks, the most likely area of excess mortality (RR=1.39, p=0.01) occurred between 1990 and 1999 in the Dallas–Fort Worth Metroplex and upper east Texas. These included 79 counties with 10 years of persistence. For Hispanics, the most likely area of excess mortality (RR=1.19, p=0.001) occurred between 1991 and 2001 from the upper Rio Grande, west, high plains, northwest to central Texas, including 146 counties; and a secondary excess mortality cluster was found in 10 upper south counties (RR=1.45, p=0.003) between 1992 and 1996. For the "other" racial/ethnic/ethnic population, the most likely cluster (RR=1.80, p=0.31) was found in the counties of the Gulf Coast and Central Texas. This cluster included 53 counties and was not statistically significant. In the first scan trial, suspected clusters for Hispanics and "other" persisted for 11 years, and the cluster for blacks persisted for 10 years, which was around the maximum temporal size (50% of the study years) set for this analysis. To detect whether these or other clusters might have persisted across most of the study period, a second scan analysis was performed with 90% of the study period as the maximum temporal size (i.e., 19 years), and with a 50% population at risk setting as previously adopted. Figures 2, 4, 6 and 8 present the results of likely clusters in this second scan. The results indicate that the temporal excess level and the counties of the primary cluster among non-Hispanic whites and blacks did not change. However for Hispanics, the most likely cluster (RR=1.52, p= 0.001) narrowed down to three southwest counties (El Paso, Hudspeth and Culberson) between 1980 and 2001. For the "other" racial/ethnic group, the most likely cluster (RR=1.68, p=0.30) was detected with a temporal persistence of 13 years, between 1989 and 2001. This was not statistically significant.

Figure 9. Excess mortality of prostate cancer among multiple racial groups in Texas counties, 1980–2001



To detect the cluster regions that affected multiple racial/ethnic groups, we further conducted spatial queries on those counties for which clusters were detected for racial/ethnic group(s). This spatiotemporal analysis found that many counties bore a higher mortality burden in multiple races. Figure 9 is a color-coded map that presents overlaid cluster layers for each race. Among them, excess prostate cancer mortality was found in 11 counties in central and the Dallas–Fort Worth Metroplex areas among all four racial/ethnic groups; 25 counties in central-to-Gulf Coast Texas were found to have an excess among non-Hispanic whites and blacks; 62 counties in west, central and upper south Texas among non-Hispanic whites and Hispanics; and 22 counties in northwest and the Dallas–Fort Worth Metroplex among blacks and Hispanics.

DISCUSSION

This study identified spatiotemporal trends of excess prostate cancer mortality in Texas that mirrored national and local trends, while revealing new information that warrants further investigation. First, the results indicated that between 1981 and 2001, five geographic regions experienced excess prostate cancer mortality rates that were statistically significant. The geographic areas identified with excess mortality generally parallel the analysis of 1970–1994 data by the National Institutes of Health[8] and 1990–1997 data by Zhan,[7] and confirm most of the counties that were previously suspected of having elevated prostate cancer mortality. Furthermore, the present study additionally identified the west-to-central Texas region as having excess mortality from prostate cancer in the Hispanic population, which persisted for >11 years. This had not been previously reported. The statistically significant relative risks of these clusters were at levels of 1.19–1.49 between 1981 and 2001, which are the highest relative risks with the longest temporal persistence detected among any of the racial/ethnic groups. The trend of excess mortality seems to persist into the current decade for Hispanics in the detected region. The findings that no "hot-spots" (i.e., RR >2) excess prostate cancer mortality were found paralleled the results of our previous spatial analysis of breast cancer[19] and colorectal cancer[20] clusters in Texas. Third, as a caveat of Exhibit 1, there may have been points in time where the trend increased or decreased significantly (i.e., among blacks), but, overall, we did not observe this trend. Fourth, the suspected cluster of excess mortality among blacks in upper east Texas persisted for 10 years but decreased after 1999 (i.e., the mortality rate declined in that year). The non-Hispanic white cluster (with a RR of 1.22) was only present for 7 years, vanishing after 1996. The declining deaths for non-Hispanic whites (starting in 1996) and blacks (starting in 1999) observed by this study are generally consistent with national and state-level studies,[6,15,21] although temporal analysis (Exhibit 1) suggested that there is no temporal trend of the decrease. Based on these findings, it appears that the Hispanic population had the highest burden of prostate cancer mortality during the study period in the identified regions, as evidenced by both spatial concentration and temporal persistence. Despite Hispanics having the lowest age-adjusted annual rate of prostate cancer, they nevertheless were strongly affected in terms of clustering in space and time. More studies are needed to find out why, as the clusters could provide a clue to prostate cancer etiology.

The clustering of prostate cancer mortality detected in this study could reflect underlying disparities in the causation of this disease, and/or delay in diagnosis and treatment. The analysis controls for age by race; thus, higher prostate cancer mortality rates cannot be explained by a higher proportion of older men in a geographic area per se. However, since age is a risk factor for this disease, the relative importance of mortality from competing causes of death could play a significant role in prostate cancer mortality. Moreover, mortality rates for various diseases differ by race/ethnicity.[22] This may be especially true in older men where conditions such as cardiovascular disease, stroke and diabetes more aggressively take their toll, while prostate cancer is often a slowly progressing disease. Highly differential causes of death have been found between racial/ethnic groups in one study of Texas prostate cancer registry patients [see Miller, JA Prostate Cancer in Texas: Addressing Racial/Ethnic and Ethnic Disparities. (unpublished dissertation). University of Texas School of Public Health, 2004. http://digitalcommons.library.tmc.edu/dissertations/AAI3143613/]. Additionally, an unusually low number of males in an area could introduce statistical instability in mortality rates from other diseases. This study did not consider the age of incident prostate cancer or survival times after diagnosis.

The cohesive, persistent and statistically significant nature of the clusters detected in this study would suggest that there are underlying contributing factors to prostate cancer mortality that might not exist outside the clusters and that may be addressed through public health research and policy. Racial/ethnic disparities continue to call for attention as one of the top-10 leading health priorities identified by the Healthy People 2010 National Health Objectives.[23] Socioeconomic factors, social environment and access to clinical preventive services contribute to racial/ethnic disparities in disease mortality. In areas of high immigration, such as Texas, lack of familiarity with the U.S. healthcare system, different cultural attitudes about the use of traditional and conventional medicine and lack of familiarity with English can pose barriers to accessing appropriate healthcare and receiving prostate cancer screening.

The identification of clusters of prostate cancer

excess mortality over time may prove beneficial for health policy and planning. For instance, Texas has yet to meet the Healthy People 2010 objective of reducing prostate cancer death rates to below 28.7 per 100,000 men.[23] By looking ahead into the current decade and working to reach this objective, the detection of excess prostate cancer mortality and identification of achievable interventions may assist in reaching the established goal. The spatiotemporal analysis presented here identified the regions with a concentrated, persistent excess burden of prostate cancer mortality among various racial/ethnic groups, particularly in those 11 counties that are bearing an excess mortality burden in all four racial/ethnic groups. In addition, the second scan, with a maximum of 90% of the study period, detected a possible 19-year persistence of excess prostate cancer mortality in Hispanics in the upper Rio Grande area of Texas. This finding, in combination with the results of the 50% temporal persistence, suggests that the "cluster trend" may be ongoing and has persisted into the current decade. The trend may be due to a lack of access to prostate cancer screening among uninsured Hispanics living in the detected region. This area of "active" excess mortality burden warrants further investigation and policy intervention.

The likely cluster of excess mortality among Hispanics in those western counties should also be considered in the context of migration patterns in this area of Texas. Cross-border migration is common, and rate denominators taken from census estimates may not reflect undocumented migration and actual population. Using accurate mortality counts while underestimating the denominator population at risk could inflate mortality rates.

Two technical notes warrant elaboration in the present study. First, although the study focused primarily on statistically significant excess mortality, it by no means suggests that those regions of nonstatistical significance of excess mortality were less important. To determine statistical significance at the 0.05 or 0.01 level, outcome measures had to satisfy the Poisson distribution model, and all predictors/covariates in this study, including space, time, age and race, would have fitted into the model simultaneously and produced a large log-likelihood ratio in the spatial-temporal analysis. The p value denoting significance is only an indicator suggesting that the level of excess mortality calls for further investigation.

Lastly, this study was completed by using the software ArcView from Environmental Science Research Institute (ESRI) and the free SaTScan. The mapping tasks implemented in ArcViews can be completed using the EpiMap component of EpiInfo, freely available from the Centers for Disease Control and Prevention. The free system might benefit medically underserved regions in the US, as well as developing countries where limited resources are available yet have pressing needs for health surveillance.

LIMITATIONS

Several research issues warrant further discussion. First, the modest relative risks occurring over a large area of contiguous counties do not necessarily indicate "clusters" of conventional epidemic intensity. Prostate cancer may have a substantial developmental period, and has causes and risk factors that are not fully understood and which may operate over various time scales. The mortality captured here is only an endpoint in that process. Further epidemiologic study and effective policy interventions to address this mortality might ideally be applied outside the windows of space or time considered here. Nevertheless, this study offered baseline descriptions of persistently elevated prostate cancer deaths in Texas, which may serve as a point of departure for further investigation and planning of health resource allocations.

Second, the study identified seven counties (e.g., Loving and King counties) averaging <10 prostate cancer deaths and <900 residents in the study period. Rates based on small numbers of events and population tend to be unpredictable and often inflated. Readers are advised to use caution when trying to interpret health outcomes, including the excess disease mortality, in these sparsely populated counties.

Third, this study considered three major population subgroups of Texas, in addition to the "other" group. Most state-level data collection and surveillance systems include two (white/blacks or African American) or three (non-Hispanic white, black or African American, and Hispanic) population groups. However, this is not consistent with the U.S. Office of Management and Budgeting Statistical Directive[15,24] which provides guidance for the collection and reporting of health data by six races (including American Indian or Alaska Native, Asian, black or African American, Native Hawaiian, Pacific Islander and white) and two ethnicities (Hispanic or Latino versus non-Hispanic or Latino). Misclassification of race/ethnicity may account for some persons being categorized as "other," in which case the mortality rates for correctly categorized race/ethnicities may be over- or underestimated, although how this affects the detection of clusters (i.e., relative rates between racial/ethnic groups) remains unclear. Misclassification may come from two sources: census and cancer registry.

CONCLUSION

Between 1980 and 2001, five regions of potential excess prostate cancer mortality were detected in Texas counties among non-Hispanic white and Hispanic groups with statistical significance. Among all racial/ethnic groups, Hispanics in three counties of the upper Rio Grande portion of Texas had the highest burden of this disease mortality as evidenced by spatial concentration and temporal persistence. Researchers and health authorities may wish to direct particular

attention to the excess mortality among Hispanics in these three counties that have extended for >19 years and may have persisted into the current decade. In addition, 11 counties in central Texas that had multiple races affected by excess mortality over time warrant further epidemiologic investigation and policy intervention. Future spatiotemporal studies may control for other risk factors, as explanatory variables would explain more of the variation seen in the data, resulting in smaller p values for clusters.

Authors' Contributions

All authors collaborated intensively and contributed equally for this study. Hsu contributed to the conceptualization of this study, including data analysis and interpretation. Miller, Soto Mas and Nkhoma all contributed to the data analysis, interpretation and epidemiological analysis of this study. All read and approved the final manuscript.

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