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Recent changes in the spatial pattern of prostate cancer in the United States

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Abstract

Introduction: Spatial-temporal trends in prostate cancer mortality are of interest because of the introduction and increasing use of the Prostate Specific Antigen (PSA) screening test after 1986. The purpose of this paper is to describe spatial-temporal changes in US prostate cancer mortality from 1968-1998.

Methods: Prostate cancer mortality data was obtained from Compressed Mortality Files available from the National Center for Health Statistics. In order to minimize potential problems such as small numbers or missing data, the analysis was limited to white males age 25 and over, and located in 2,970 counties with complete data. Statistical analyses included the global distance between observed and expected multinomial probabilities; Hoover's Index of Concentration; and a retrospective test for change in spatial patterns.

Results: Fairly steady declines were observed in prostate cancer mortality from 1968 until 1993, with an increasing tendency toward spatial uniformity. Spatial concentration increased from 1994-1998 had increasing spatial concentration, but by 1998, the level of spatial concentration had returned to levels that prevailed during the early to mid-1980s. Comparing 1991-98 to 1968-90, the observed number of prostate deaths increased most rapidly with respect to the expected number in Western Appalachia and the south central US.

Conclusions: The observed results are generally consistent with prior evaluations of prostate cancer spatial-temporal patterns. However, the current study identified a heretofore unnoticed recent pattern of change in Western Appalachia and the south central US.

Introduction

Prostate cancer is the number one incident cancer and second most common cause (after lung cancer) of cancer death for men in the United States. In 2003 alone, the American Cancer Society estimated 220,900 men to be diagnosed with prostate cancer; 28,900 deaths were directly attributable to the disease.[1] Epidemiologists have identified age, ethnicity, family history and dietary practices as important risk factors for prostate cancer incidence. Incidence rates vary 12-90 fold internationally[2,3] and have been on a general, worldwide increase during recent decades—even in low risk populations in Asia and Eastern Europe.[1,4-7]

Based mostly on prostate cancer incidence and mortality registries maintained by the SEER program (covering 14% of the U.S. population), a number of analyses of the trends in mortality and incidence over the past three decades are available for United States. Data indicate that incidence, survival, and mortality trends are similar for both blacks and whites[8]. Incidence rose through the mid-1980's among both blacks and whites. With the introduction of the Prostate Specific Antigen (PSA) testing in 1986, the incidence rates increased even more, but started declining in 1992 for white men, and in 1993 for black men. Incidence rates have declined since then[5-15]. Most of the increase was for early stage (localized and regional grade) prostate cancers. Distant stage disease rates remained relatively stable from 1973-1991; these rates have fallen dramatically for whites and somewhat less rapidly for other racial groups since 1992[11,12]. Data also indicate that the average age at diagnosis has fallen and the proportion of advanced stage tumors has declined, whereas the proportion of moderately differentiated tumors has increased.[10]

These data support the widely held hypothesis that the increase in early stage rates just after 1986 is attributable to the launch of aggressive PSA screening. The theory also predicts a decrease in incidence rates a few years after the start of the PSA era, once screening efforts start to be widely practiced. Data confirm this aspect as well. Moreover, data also confirm the hypothesis that successful early screenings lead to an eventual decrease in advanced stage cases.

Research also shows that mortality rates have also decreased from 1992 onwards[5,8,10,16,17], i.e., there is a time delay of about 5-6 years from the time of adoption of PSA screening to the time of first observable effects on mortality rates. For the age-group 50-84, mortality rates have actually fallen below the 1986 level after 1995 for white men, and after 1997 for black men. The decrease in distant disease mortality is directly attributable to a decrease in distant stage incidence, *and not* to improved survival of patients with distant disease. Therefore, the decrease in prostate cancer mortality rates also appears to reflect a change in medical practice (e.g. heightened screening) rather than a change in risk factors[10]. Thus, mortality rate data analysis also suggest that PSA screening has played its desired role in reducing prostate cancer rates. However, some researchers [10,17] caution against forecasting long term trends, given that the etiology of the disease is still unknown and public health benefits from current screening practices are yet to be proved conclusively.

Geographic Variation in Incidence and Mortality Rates

Although data and analyses on prostate cancer are available at both international[16-20] and, more commonly, national scales, less information is available on smaller scale spatial (or spatio-temporal) patterns. Most analyses have concentrated on temporal trends and have disregarded spatial patterns of incidence and mortality. The Atlas of Cancer Mortality for the U.S.[19] is still one of the best sources for maps of prostate cancer mortality in the US. However, as some analyses[3,20] have underscored, spatial pattern and cluster analyses can play an important role in identifying the impact of demographic and socioeconomic risk factors and can contribute significantly toward the identification of the etiology of prostate cancer.

The few studies that have focused on the spatial pattern of prostate cancer find interesting and consistent patterns. The Atlas of Cancer Mortality[21] revealed that prostate cancer mortality rates were higher among white men in the Northwest, Rocky Mountain States, New England, North-Central and South Atlantic areas, and for black males in the South Atlantic region. An inverse rural-urban gradient was suggested, with high rates in less populated areas[5]. Similar patterns had been described by others prior to the atlas release[22]. It has also been noted that for the northwest, patterns for white males are more clustered in recent years than for the earlier years [5]. A recent study[3] detected five statistically significant clusters for whites and three significant clusters for blacks in the United States; the patterns observed could not be explained away by the selected demographic and socioeconomic factors, indicating that further spatial analysis of the risk factors and medical or reporting practices is required.

The purpose of this paper, therefore, is to describe the spatial changes in prostate cancer mortality that occurred during the 31-year study period, 1968-1998. Because of the relatively small number of non-whites in many regions of the country, the study is confined to white males. The analysis is carried out at the county level, and is based upon the annual death counts attributable to prostate cancer.

Methods

Data

Prostate cancer mortality based on death certificates was obtained from the Compressed Mortality File (CMF), produced by the National Center for Health Statistics[23].The CMF data are available at the county level for individual years for the period 1968-98, grouped by age, sex, race and underlying causes of death. The annual number of prostate cancer deaths (International Classification of Disease-9th edition codes (ICD-9), 185.0 -185.9) was obtained for each county in the *contiguous* United States for the period from 1968 through 1998, for *white men* aged 25 and over, by 10-year age groups (because of the difficulties associated with analyzing small numbers of nonwhites outside of the South, the analysis is confined to prostate cancer frequencies for white males). Thirty deaths were excluded with unknown age information.

The data are available for 3146 counties in the USA. However, due to incomplete records for some counties, only the contiguous 48 states were included in this study and only 2970 counties

of the possible 3111 were used for the final analyses. Data for 1972 were also excluded because of relatively low sampling frequencies used for that year.

The expected number of prostate cancer deaths for white males was derived using the population estimates were based on Bureau of the Census estimates of midyear county population provided with the CMF. The expected number of prostate cancer deaths in each county was obtained using the indirect standardization method, by multiplying national age-specific death rates and the county population in each age group.

Descriptive measures of change in the spatial pattern

Two descriptive approaches were used to evaluate temporal changes in the departure of mortality rates from spatial uniformity. With the first approach, for each year, multinomial probabilities (p_i) were defined as the likelihoods that a given prostate cancer death is located in county *i*. For each year , a χ^2 statistic was computed as a global measure of the difference between observed and expected county frequencies. To account for the fact that the number of deaths from prostate cancer essentially doubled during the time period, it is desirable to examine changes in the following quantity:

$$w = \sqrt{\sum_{i=1}^{m} \frac{\left(p_i^{obs} - p_i^{exp}\right)^2}{p_i^{exp}}} = \sqrt{\chi^2 / n} \qquad (1)$$

m is the number of counties, *n* is the number of deaths from prostate cancer, and where *p* represents the observed and expected proportions of all national cancer cases that fall in county *i*. The quantity *w* was calculated for each year, and represents a measure of the global distance between the observed and expected multinomial probabilities. As an aside, when the *p*'s in Equation 1 are replaced by the *p*'s expected under null and alternative hypotheses, *w* becomes a measure of effect size (i.e., a measure of the distance between null and alternative hypotheses[24]).

The second descriptive approach was to use Hoover's Index of Concentration[25,26] as a measure of the spatial concentration of prostate cancer cases. The Hoover index was calculated as

$$H = 50\sum_{i=1}^{m} | p_i^{obs} - p_i^{exp} | \qquad (2)$$

where the *p*'s are as previously defined. Conceptually, the Hoover index represents the percentage of all observed cancer cases that would have to be relocated to ensure that the spatial distribution of actual cases was identical to the spatial distribution of expected cases. In the special case where the observed and expected distributions are identical, the Hoover index is equal to zero. At the other extreme, imagine all cancer cases in one county, and no cases in all other counties; in this case, the Hoover index would approach its upper limit of 100.

Retrospective Detection of Change

A significant "changepoint" was defined as a point during the 31-year period where the spatial pattern of prostate cancer before the changepoint was substantially different from the spatial pattern of prostate cancer mortality after the changepoint.

Of interest is a test of the null hypothesis that the multinomial probabilities (representing the probabilities that a given case falls in a particular county) do not vary over time. In a spatial context, the null hypothesis is one of no spatial change, versus the alternative that there is a single changepoint dividing the temporal sequence of multinomial vectors of county-level prostate cancer deaths into two distinct temporal subsets ("before" and "after" the changepoint). Such a statistical test has been developed [27] and applied previously in a spatial context to the geographic pattern of county-level breast cancer mortality in the northeastern United States[28].

The test for a single changepoint, in a sequence of multinomial probabilities, may be described as follows. Given a *T* by *m* contingency table with ordered rows (where we have *T* equal to the number of time periods and *m* equal to the number of counties), the goal is to test for a change in the row proportions after an unknown row, *r*. Let Q^2 be the usual Pearson χ^2 statistic for testing association between rows and columns in the full table. Let Q_r^2 be the Pearson χ^2 statistic for testing association between rows and columns in the 2 by *m* table formed by aggregating the first 1,…, *r* rows and the remainder of the rows (*r* +1,…, *n*). The test statistic is : $Q_{\hat{r}}^2 = \max_r Q_r^2$

Extramultinomial variation also needs to be accounted for; such variation may arise in situations where the multinomial trials are correlated or where the multinomial probabilities are themselves not known with certainty. The associated variance inflation factor, $\hat{\sigma}^2$, may be estimated as

$$\hat{\sigma}_r^2 = (Q^2 - Q_r^2)/\{(T-2)(m-1)\}. \qquad (3)$$

The test statistic, adjusted for this extramultinomial variation, is $K_{\hat{r}}^2 = Q_{\hat{r}}^2 / \sigma_{\hat{r}}^2$, and the quantity $K_{\hat{r}}^2/(m-1)$ has an *F* distribution with *m*-1 and (*T*-2)(*m*-1) degrees of freedom.

Before computing the test statistic, the annual observed vectors of county deaths must be adjusted due to changes in expectations that occur as a result of population and age structure changes. This is done by multiplying the observed number of county deaths in year *t* by the ratio of the proportion of all expected deaths (for the entire time period) that occur in county *i* to the proportion of all expected deaths (for year *t*) that occur in county *i*. The result is an adjusted or standardized number of observed deaths (based upon the constant county shares of the expected deaths that are observed over the entire time period).

It is of interest to ask which counties contributed most significantly to the spatial changes that occurred. To assess this, for each county, annual, standardized *z*-scores associated were computed, using the observed and expected frequencies:

$$z = \frac{f_{obs} - f_{\exp}}{\sqrt{f_{\exp}}} \qquad (4)$$

These *z*-scores were computed for each county and for each year; they have a mean of zero and a variance of one, and they represent a measure of how far the observed mortality frequencies are from expectations. They are based on a Poisson model for the distribution of observed deaths; f_{exp} represents both the mean and the variance of the Poisson distribution. The z-scores are constructed in the usual fashion by subtracting the expected value from the observed value, and then dividing the result by the standard deviation. In addition, and more importantly from a

conceptual perspective, the *z*-scores are equal in absolute value to the square root of the individual county's contribution to a global chi-squared statistic that measures how far observed mortality is from expectations in a given year.

For a given county, the difference between its average z-score before and after the change represents how the county's prostate cancer mortality (relative to expectations) has changed. This difference in average *z*-scores has a variance of $(\hat{r}^{-1} + (T - \hat{r})^{-1})$ (under the assumption that the covariance of the *z*-scores for a county in two different years is equal to zero); hence for our data, where the changepoint occurs after the 22rd year of observations, the quantity

$$\tilde{z} = \frac{\overline{z}_{91-98} - \overline{z}_{68-90}}{\sqrt{22^{-1} + (30-22)^{-1}}} = \frac{\overline{z}_{91-98} - \overline{z}_{68-90}}{\sqrt{.168}} = .41 \qquad (5)$$

has, approximately, a standard normal distribution under the null hypothesis of spatial uniformity in both periods. (Although the assumption of normality is admittedly questionable for counties with small expected frequencies, the aggregation into two reasonably long time periods alleviates much of this concern). The assumption of a zero covariance was evaluated by computing for each county the serial correlation associated with the annual *z*-scores. Although the serial correlation was positive for many counties, it was also negative for many, and the average correlation was –0.02; this suggests that the assumption of a zero covariance is not unreasonable. In any event, the spatial distribution of the $\tilde{z}$ values was of primary interest; correcting for any covariance would effect the scale of the difference map, but not the spatial pattern.

To highlight spatial patterns, prior to mapping, the $\tilde{z}$ values were smoothed using a Gaussian kernel. This corresponds to placing a normal distribution at the center of each county, and then replacing the county's observation with the weighted sum of the observations in that county and surrounding counties (where the weights associated with each county are equal to the height of the normal distribution at that county's centroid). The standard deviation of the normal distribution is associated with the amount of smoothing. A large value will give substantial weight to even distant counties, and will result in a relatively large amount of smoothing. A small standard deviation will confine the large weights to a small neighborhood around each county, and little smoothing will occur. The standard deviation was a distance equal to the square root of the average area of a county (corresponding roughly to the average length of a county's border). This choice corresponds approximately to the common practice of using adjacent counties in measures of spatial autocorrelation.

Results

The number of deaths due to prostate cancer among white males rose steadily and doubled during the period 1968-1994, with approximately 14,000 annual deaths at the beginning of this period, and slightly more than 28,000 by the end of the period. By 1998, the annual number of deaths had fallen slightly, to about 26,500.

Descriptive measures of change in the spatial pattern

Figures 1 and 2 display the annual values of *w* and *H* for the period 1968-1998 (in the figures, the vertical scale does not begin at zero, to exaggerate the vertical scale and allow for better visualization of temporal changes). Although there are no major changes in either index over time, general trends are apparent. Both descriptive approaches reveal fairly steady declines from the beginning of the study period until about 1993, indicating an increasing tendency toward spatial uniformity. The period 1994-1998 was one of increasing spatial concentration, and by 1998 the level of spatial concentration had returned to levels that prevailed during the early to mid-1980s.

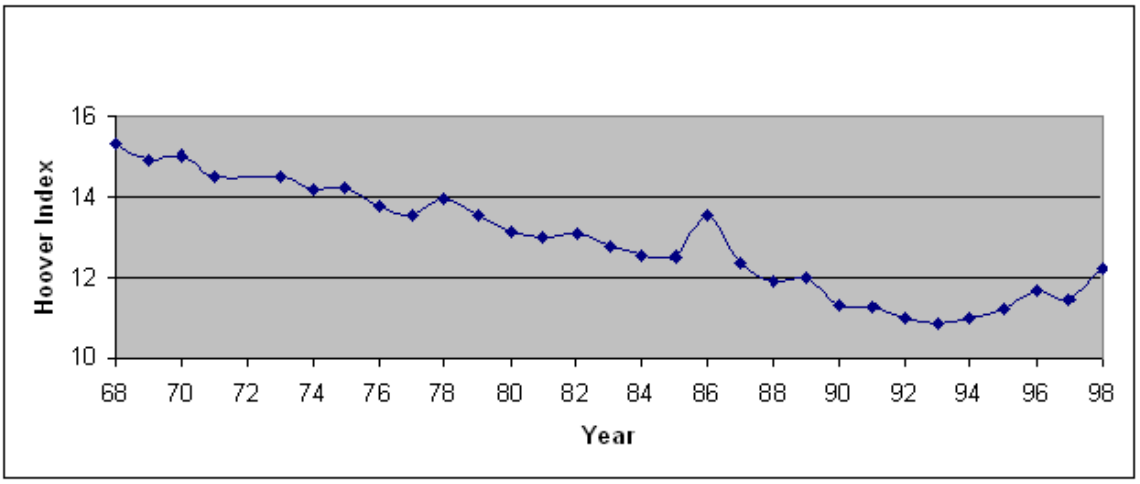

Figure 1. Hoover Index (1968-1998)

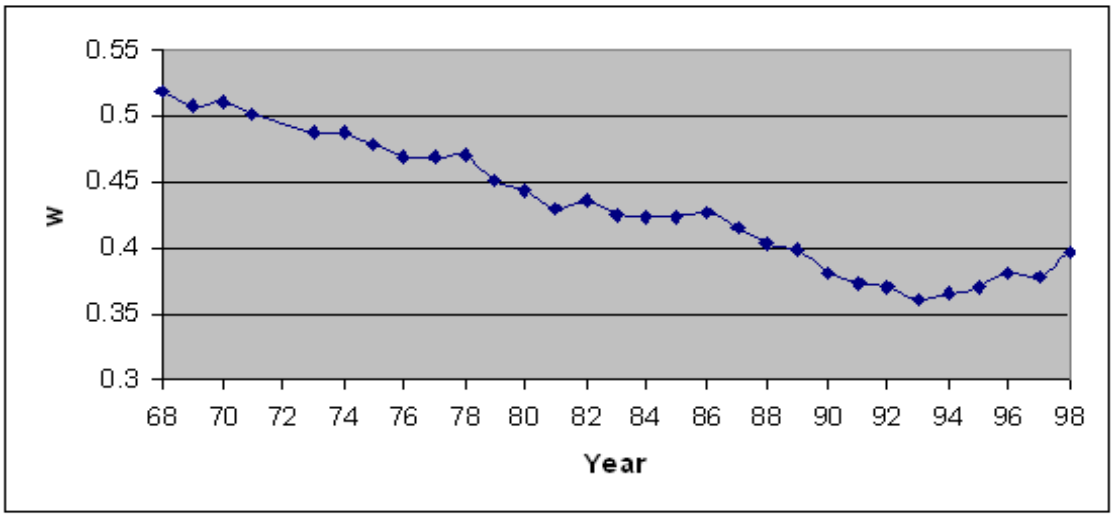

Figure 2. Distance from Expectations (w) (1968-98)

Retrospective Detection of Change

The results – in the form of the chi-squared statistic associated with a breakpoint after each potential year -- are shown in Figure 3. The changepoint statistic, which is based upon the maximum chi-squared value in the figure, is significant, and indicates a change in the spatial pattern of prostate cancer following 1990. In addition, the maximum illustrated in the figure is quite peaked, instilling confidence in the accurate identification of the changepoint, and indicating possible changes in the spatial pattern in a relatively narrow time window around 1990

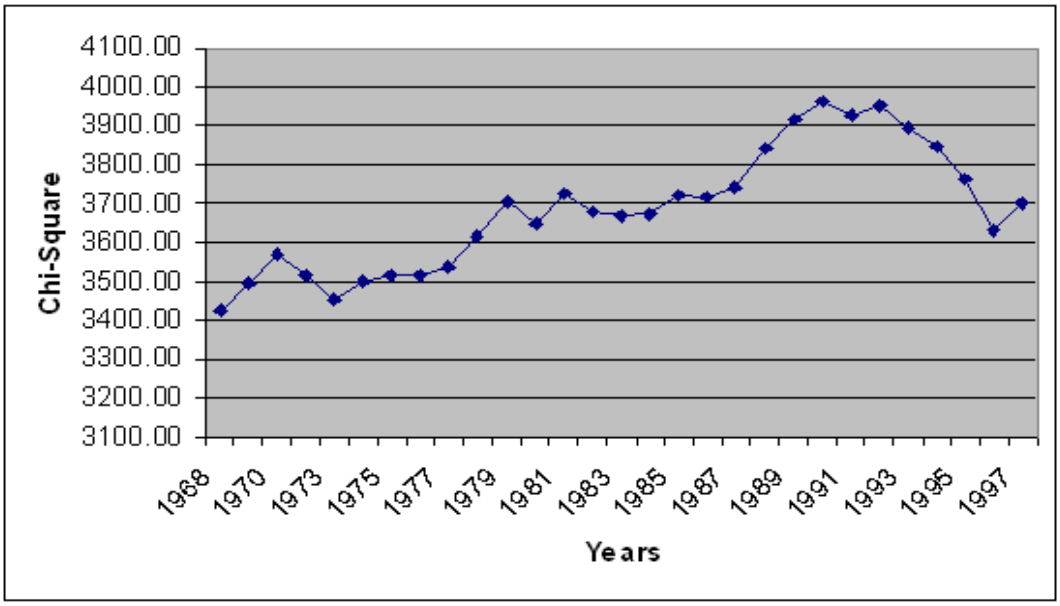

Figure 3. Chi-Square Values for Potential Breakpoints

Figure 4 represents a map of the standardized differences between the two time periods. The map shows high positive values in western Appalachia and down through the south central portion of the country. These are areas where the observed number of cases has grown the most rapidly, with respect to the expected number of cases. Areas that display the most negative values include much of New England and scattered portions of the upper Midwest and West; these are areas where the largest reductions in cancer mortality have occurred relative to expectations.

Figures 5 and 6 display the smoothed values of $\overline{z}_{68-90}$ and $\overline{z}_{91-98}$; each has been adjusted by dividing by the standard deviation of the mean *z*-score, so that the quantities mapped represent standard normal variables (the difference of these maps is shown in Figure 4). It is interesting to note that many of the declines in mortality (shown in blue in Figure 4) have occurred in the northern part of the country, where rates are highest. Likewise, many of the increases in mortality, relative to the expectations brought about by assuming spatial uniformity, have occurred in the southern portion of the country, where rates have historically been lower. In sum, the map of mortality observed for the period 1991-1998 shows less spatial variability than the

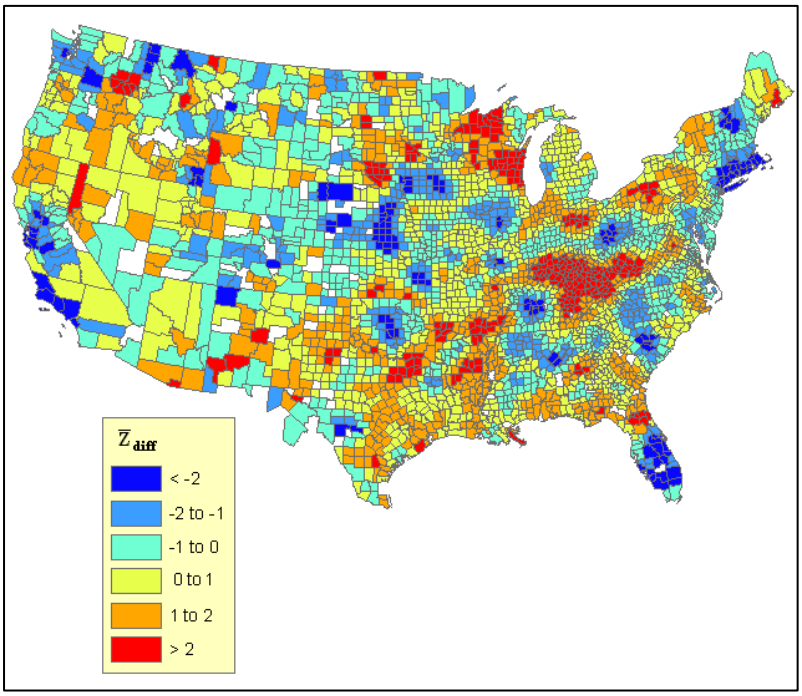

Figure 4. Difference in Smoothed Z-Scores (1968-90 to 1991-98)

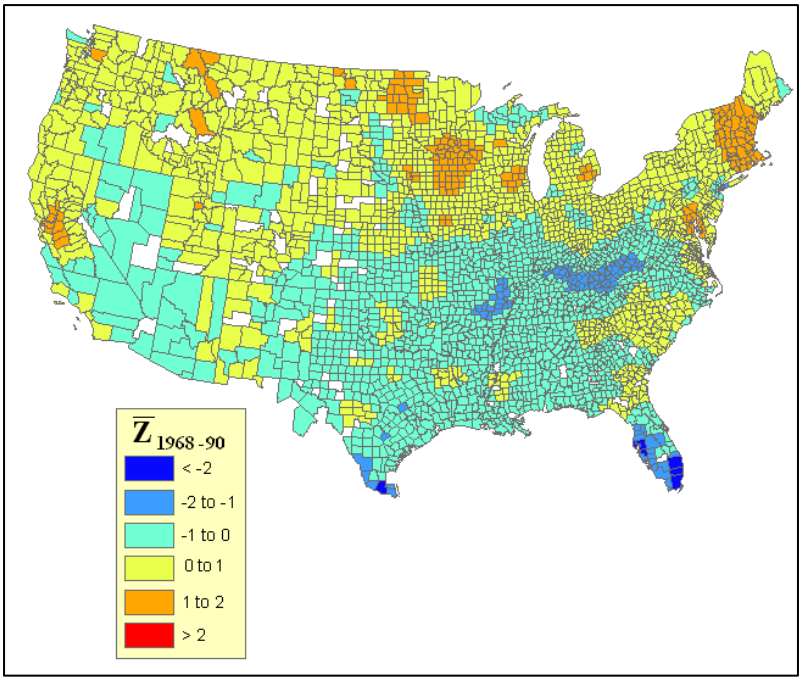

Figure 5. Smoothed Z-scores (1968-1990)

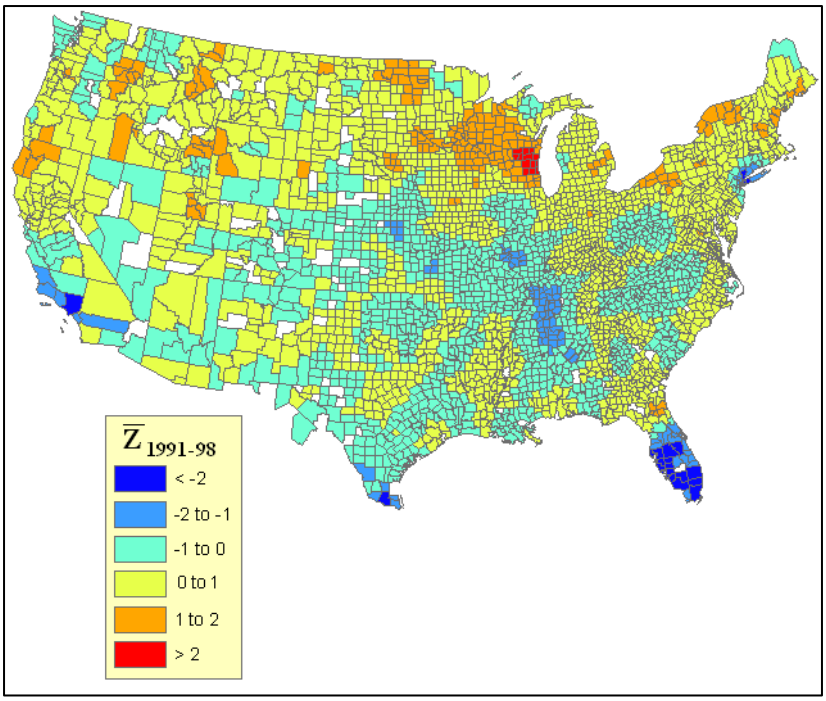

Figure 6. Smoothed Z-scores (1991-1998)

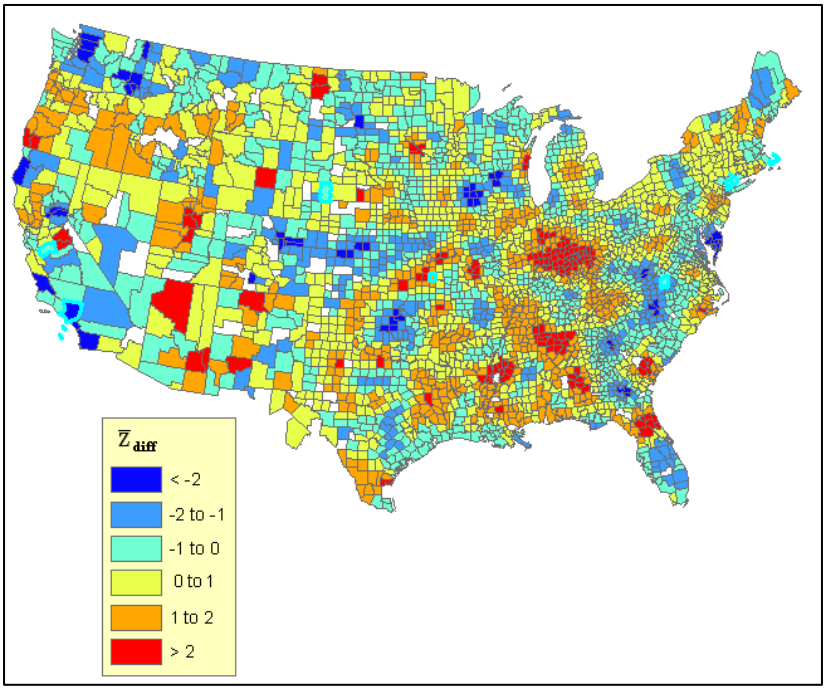

Figure 7. Difference in Smoothed Z-Scores (1991-94 to 1995-98)

map observed for the period 1968-1990. These results are consistent with the descriptive results presented earlier, which showed, from 1968 onwards, a tendency toward lesser geographic diversity and greater spatial uniformity over time, at least until about 1994. The data for the period 1991-1998 was then examined using the same method, and a significant changepoint was discovered in 1994. The spatial differences in prostate cancer mortality between the 1991-1994 and 1995-1998 periods are highlighted in Figure 7. Although distinct spatial patterns are not readily apparent, the declines in southern California and Florida are notable because these constitute large urban areas; declines in these areas of already low prostate cancer mortality contributed to the decreasing spatial uniformity that occurred during the period 1995-1998.

Year	State	County	Observed	Expected	Z
1968	ND	Dunn	6	1.2	3.55
1969	MA	Barnstable	17	10.3	3.52
1970	OR	Klamath	12	3.7	3.7
1971	NC	Davidson	15	5.54	3.53
1973	TX	Grimes	16	5.54	3.87
1974	MO	Wright	10	2.32	4.16
1975	ND	Barnes	8	1.97	3.58
1976	IA	Pocohantas	8	1.95	3.6
1977	MO	Bollinger	7	1.48	3.71
1978	MS	Scott	11	1.71	5.57
1979	MD	Baltimore City	80	49.28	4.11
1980	VT	Lamoille	3	1.39	3.78
1981	VA	Pittsylvania	13	3.96	3.89
1982	NE	Deuel	12	3.71	3.69
1983	IA	Calhoun	9	2.46	3.51
1984	ND	Pembina	9	1.72	4.47
1985	ID	Jefferson	8	1.44	4.37
1986	IA	Emmet	10	1.89	4.74
1987	IA	Webster	18	6.17	4.14
1988	NC	Oswego	7	1.12	4.36
1989	UT	Salt Lake	85	55.76	3.71
1990	MS	Choctaw	7	1.02	4.6
1991	MN	Benton	14	4.06	4.2
1992	IA	Bremer	14	4.1	4.16
1993	GA	Emanuel	9	2.03	4.03
1994	OR	Morrow	7	1.22	4.15
1995	MS	Madison	12	3.09	4.24
1996	WI	Kewaunee	13	3.18	4.57
1997	TN	Marion	12	2.77	4.57
1998	WI	Calumet	14	3.64	4.55

Table 1. Maximum z-scores (1968-1998)

Year	State	County	Observed	Expected	Z
1968	WI	Douglas	0	5	-3.37
1969	FL	Pinellas	72	120.2	-4.68
1970	NY	Queens	123	171.92	-3.89
1971	NY	Bronx	79	115.08	-3.52
1973	NY	Kings	150	199.73	-3.64
1974	NY	Bronx	69	109.56	-4.1
1975	MO	Franklin	0	6	-3.67
1976	TX	Hidalgo	2	18.12	-4.74
1977	FL	Dade	140	189.31	-3.72
1978	VA	Augusta	0	7.62	-4.14
1979	PA	Warren	0	5.23	-3.43
1980	TX	Bell	1	9.67	-3.5
1981	NY	Kings	140	188.88	-3.69
1982	WI	Marinette	0	5.91	-3.65
1983	FL	Dade	166	215.88	-3.51
1984	FL	Broward	146	202.66	-4.14
1985	NY	Kings	141	190.25	-3.7
1986	NY	Queens	144	206.07	-4.52
1987	TX	Hidalgo	8	32.62	-5.04
1988	IL	Ogle	0	5.97	-3.67
1989	NY	Kings	149	200.49	-3.77
1990	TX	Hidalgo	16	40.67	-4.31
1991	NY	Kings	153	207.6	-3.93
1992	NY	Kings	144	106.92	-4.57
1993	FL	Broward	227	286.39	-3.61
1994	TX	Taylor	2	13.83	-3.9
1995	CA	Los Angeles	599	708.89	-4.21
1996	FL	Palm Beach	180	250.08	-4.61
1997	CA	Los Angeles	572	676.42	-4.1
1998	CA	Los Angeles	527	655.68	-5.16

Table 2. Minimum observed z-scores (1968-1998)

It is also instructive to look at the locations of the maximum and minimum (unsmoothed) z-scores in each year; despite the limitations associated with looking at outliers, some trends emerge when looking at these values over a 31-year period. The maximum standardized scores that were observed in each year are shown in Table 1. Also shown are the observed and expected counts for those counties where the maximum was observed. The scores in later years appear to be generally higher than those in earlier years.

The minimum standardized scores are shown in Table 2. It is interesting to note that the minima typically occur in large counties, while the maxima tend to occur in small counties; this is in keeping with a reverse rural-urban gradient that has been noted previously (with high values in rural areas and low value in urban regions). Also notable is the fact that Los Angeles county had the lowest score in three of the last four years for which data were available. It seems plausible that a part of the increasing spatial diversity in prostate cancer mortality witnessed during the mid to late-1990s may be due to the declining mortality in Los Angeles county, as well as declining mortality in parts of Florida. This would be consistent with what one might expect from the early implementation of screening programs; they are more likely to be adopted early in urban areas, and hence any early improvements in mortality might be more likely to occur in such places.

Conclusions

The spatial changes determined here through the use of statistical methods have occurred at about the time changes would have been expected to show up in mortality patterns, given an approximate time lag of five years from the start of widespread screening. Although there are alternative ways to interpret these results, one possible summary is:

i. Spatial variation in cancer mortality declined for a long period of time, so that the pattern of deaths observed during the early 1990s was considerably more uniform than that observed earlier. This was largely the result of long steady declines in spatial concentration that occurred until about 1994.
ii. Spatial concentration began increasing in 1994. An interesting and open question is whether these changes since about 1993 or 1994 can be attributed to spatial variability in the effectiveness of screening programs. This could come about, for instance, if screening programs were initially more widely adopted in areas of low mortality – and there is a possibility of this given that a) there is some support for a reverse rural-urban gradient in prostate cancer mortality, and b) it seems likely that screening would at least initially be more effective in urban than in rural areas. Spatial concentration would increase as a result of possibly stronger declines in mortality in urban areas (due to the early effects of screening programs), where mortality is already low relative to rural areas.

There are some notable limitations to this study. The retrospective test for change in spatial patterns is based upon the alternative hypothesis that there is a single point in time that divides the spatial patterns into “before the change” and “after the change”. Although Figures 1 and 2 seem to indicate gradual changes in descriptive measures of spatial concentration, Figure 3 seems to imply a more well-defined changepoint.

These figures, therefore, need to be interpreted with caution; they are not necessarily inconsistent, but rather are simply limited in their ability to reveal a fuller picture of spatial change. It is possible to have a substantial change in geographic pattern, with little or no change in systemwide measures of spatial concentration (this would occur, for example, if the individual counties where concentration was high shifted from one location to another). An important next step is to relate the results described here to changes in prostate cancer incidence.

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