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There is a lack of literature examining how the spatiotemporal trend of asthma may have impacted different ethnic /racial compositions of Texans. The present study sought to evaluate the geographic-temporal variations of asthma mortality in Texans over a 22-year period, retrospectively, and examine whether the trend of environmental Toxic Release Inventory (TRI) concentrations and their spatiotemporal persistence might place an uneven burden on particular racial groups. The study concentrates on the time period between 1980-2001 and first evaluates geographic excess of asthma mortality in different racial groups at the county level and characterizes the excess burden by spatiotemporal v ariations. After this assessment, the impact of TRI on asthma mortality over this period of time is analyzed. Based on these two analyses, this would identify which racial/ethnic groups in which Texas regions might have been affected the most by regarding mortality over time, and suggested priority geographic areas for policy intervention. At the end of the study, it could be said that there might be an association between the TRI release and increased asthma mortality in the Black male population.

ASTHMA MORTALITY AND TOXIC RELEASE IN TEXAS - AN ECOLOGICAL STUDY 1980-2001

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ASTHMA MORTALITY AND TOXIC RELEASE IN TEXAS - AN ECOLOGICAL STUDY 1980-2001

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CHAPTER I

INTRODUCTION

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CHAPTER II

BACKGROUND AND RATIONALE

Asthma is a complex lung disease that is characterized by epithelial shedding, airway smooth muscle hypertrophy and hyperplasia, overproduction of mucus and airway inflammation. Asthma is defined as a chronic inflammatory disease, which leads to symptoms such as coughing, episodes of wheezing, breathlessness and chest tightness. Asthma is a growing public health problem as its incidence; prevalence and mortality are still increasing in most of the world. Asthma is strongly related to immuno-genetic susceptibility. The immunomodulatory role of airway smooth muscle is mediated by the production of pro-inflammatory cytokines, chemokines, polypeptide growth factors, extra cellular matrix proteins, cell adhesion leukocyte activation and cell recruitment (Hirst et al., 1999). The pathophysiology of asthma has traditionally been attributed to an inflammatory process occurring predominately in the large airways (Crushmann et al., 1982). Early studies, conducted over 100 years ago, used autopsy specimens to study the macroscopic morphological and histological changes that occurred within large asthmatic airways. These studies clearly showed that asthma involves structural airway changes, including size and amount of airway smooth muscle, thickened basement membrane, mucus hypersecretion and edema of the airway wall (Crushmann et al., 1982). Asthma is clinically characterized by three factors reversible bronchial airway inflammation, increased mucous production and airway hyperresponsiveness (King et al., 1999). The inflammatory process

is regulated by CD4 +T cells, which are defensive cells in the body. These produce cytokines which stimulate growth, differentiation and recruitment of other defensive cells such as mast cells, basophils, eosinophils and B cells. The mast cells will degranulate, releasing histamine, prostaglandins, leukotriene, platelet activating factor, and bradykinin. This results in immediate response and inflammation of the airways. This also produces a late phase response, which involves inflammatory cells, i.e. eosinophils, to migrate and activate intercellular adhesion molecules (ICAM). These act to continually recruit and retain the inflammatory cells to certain regions causing the epithelium to be damaged and exposes the nerve endings. In asthmatics, this inflammatory response is amplified causing severe bronchial constriction (Hirst et al., 1999). The application of fibrooptic bronchoscopy in asthma has enabled us to obtain small human endobronchial biopsies from large airways. This, together with the development of molecular biology technologies (immunochemistry and insitu hybridization), has advanced our understanding of the pathogenesis of bronchial asthma (Djukanovic et al., 1991).

The first annual list from the Asthma and Allergy Foundation of America reveals the worst cities for people with asthma. Researchers ranked the top 100 metropolitan areas in the U.S. based on prevalence, risk, and medical factors related to asthma, including:1) prevalence of asthma; 2) asthma-related deaths; 3) outdoor air quality; 4) annual pollen measurements; 5) smoking laws; 6) number of asthma prescriptions filled per patient and 7) number of asthma specialists in that area. Knoxville, Tennessee., Little Rock, Arkansas., and St. Louis topped the list as the three worst cities for asthma sufferers. Conditions were found to be much more favorable for people with asthma in the San Francisco Bay area,

Miami, and Daytona Beach. Fla. (Asthma and Allergy Foundation of America, 2004).

Risk Factors of Asthma:

Researchers say that more than 20 million Americans suffer from asthma. About half of those suffer from the most common form, allergic asthma, but many may not know it. In people with allergic asthma, the coughing and wheezing of asthma attack is triggered by exposure to allergens, such as dust, pet dander, or mold spores rather than irritants, weather changes, viral or sinus infections, or exercise. There are many risk factors for asthma such as genetic factors, repeated lower respiratory tract infections, ethnicity, socioeconomic status, gender etc. Multiple twin and family analyses strongly imply a genetic basis for allergy related traits (Sanford et al., 1996). A recent study of 11, 688 Danish twin pairs (comparing identical and non-identical twin pairs) suggested that 73% of asthma susceptibility is due to genetic factors (Skadhauge et al., 1999). However, allergy-associated phenotypes, including asthma, do not appear to follow any Mendelian inheritance pattern, which is characteristic for complex genetic (multifactor) traits. The dissection of these traits is hampered by phenocopy, incomplete penetrance and genetic heterogeneity (Lander et al., 1994).

Repeated lower respiratory tract infections showed a positive association between subsequent wheeze and doctor's diagnosis of asthma. Reverse causation seems to be a plausible explanation for this, with lower respiratory tract infections being predictors of, rather than, risk factors for asthma. Thus, children already predisposed to asthma might simply be more likely to develop symptoms of lower respiratory tract when infected, rather than the virus causing the development of asthma. This explanation is supported by the

frequency of repeated infections (>2 infections before age three) being significantly higher in children with a family history of atopy (allergy) than in those with no atopic family member ($p<0.001$). Several authors have indeed reported that infections may enhance the development of asthma, particularly infections with respiratory syncytial virus (RSV) (Sigurs et al., 1995). The prevalence of asthma, allergic rhinitis and atopic dermatitis has dramatically increased over the past decades. This atopy- related diseases are most common chronic disorders in childhood in western societies. Several environmental studies have evaluated environmental risk factors that may explain the reasons for the steady increase of allergic disease with time.

Racial Disparities

Study by Hu et al., found boys to be significantly more likely to have physician diagnosed asthma than girls (odd ratio =1.7). This suggests different behavior such as variation in physical activity and biological differences exist between sexes. This study accounted for differences often seen in studies concerning racial differences, socioeconomic status. One possible mechanism is the amount of IgE (antibody) an individual possesses (Schwab et al. 2000). Certain studies have found a strong correlation between serum IgE and prevalence of asthma. These studies also suggest that blacks have higher IgE levels than whites and males have higher levels than females. These findings may explain the asthma severity in children but do not reflect in adults since females tend to have more mortality as adults. Another possibility for more prevalence in boys is, they tend to have smaller airways at any given lung size than girls. Other reasons are boys tend to have greater bronchial ability and they tend to have higher incidence of upper airway

infections (Hu et al., 1997) who also reported maternal smoking during pregnancy was significantly associated with childhood asthma (adjusted OR = 1.9, 95 % Confidence interval: 1.1 to 3.5). This study also found that cord blood IgE concentrations were elevated significantly in infants whose mothers smoked during pregnancy and this might predispose infants to subsequent sensitization and allergy (Hu et al., 1997). It has also been suggested that intrauterine exposure to smoking could cause changes in pulmonary structure and function. Yet another factor is genetic predisposition, as exemplified by the presence of maternal asthma. Researchers say that a better understanding of the role of IgE antibodies in allergic asthma now makes it easier for people with the condition to get effective treatment. IgE antibodies are produced by the body in response to exposure to allergens and may cause the release of histamines and other chemicals that can lead to inflammation of the airways and subsequent asthma attacks.

Asthma as a public health problem

Asthma currently affects 20 million people in United States, 5 million of who are under age 18 (Schwab et al, 2000). Asthma causes a great stress on the health care system. In the US, 9% to 16% of children use asthma medication regularly and 0.4% are hospitalized annually (Peat et al., 1999). The disease estimated to cost the US economy $ 11 billion in health care costs and lost productivity each year (Schwab et al., 2000). Due to increased prevalence of asthma, a multitude of studies have examined potential opportunities for primary and secondary prevention. Asthma is a major public health problem of increasing concern in the United States. Between, 1980 and 1994 prevalence of asthma has increased 75% overall and 74% among children 5-14 years of age

(www.cdc.gov). Low-income population, minorities, and people living in inner cities experience disproportionately higher morbidity and mortality due to asthma.

Effects of asthma on children and adolescents are huge. Asthma accounts for 14 million lost days of school missed annually and it is the third ranking cause of hospitalization among those younger than 15 years of age. The number of children dying from asthma increased almost three fold from 1993 in 1979 to 266 in 1996 and estimated cost of treating asthma in those younger than 18 years of age is $3.2 billion per year (www.cdc.gov/nchs). Asthma accounts for 14 million missed school days and it especially affects children belonging to a racial/ethnic minority group. Since 1978, the rate of mortality from asthma has increased substantially in the United States (Gadde et al., 1988). The rate of mortality from asthma decreased from 1.68 per 100,000 people in 1969 to 0.68 per 100,000 in 1977, but subsequently increased to 0.92 per 100,000 in 1978 and 2.41 per 100,000 in 1991 (Weiss et al., 1993).

Currently, there are no preventive measures for asthma; however, people with asthma could still lead quality, productive lives if they control their asthma. Asthma can be controlled by taking medication and by avoiding contact with environmental triggers. (www.cdc.gov/nchs). However, Indoor or outdoor environmental factors are essential to trigger asthma attacks. Several factors triggering asthma has been identified. Asthma is the most common chronic disease in the USA and evidence strongly suggests recent increases in prevalence and severity of the condition (Strachan et al., 1996). Etiologically, changes to the indoor environment have been implicated in both these increases; proliferation in American homes of soft furnishings, wall-to-wall carpets, central heating, and double

glazing, together with the tendency for asthmatics, especially children to spend more time indoors, have all combined to increase exposure to airborne indoor allergens (Woodcock et al., 1998). The allergen most commonly implicated in the USA is house dust mite, mattresses, bedding and carpets being its most important domestic reservoirs. A number of approaches to decreasing house dust mite exposure have been tried, of which dust mite impermeable bedding is the most promising (Custovic et al., 1998). There is increasing evidence that environmental factors contribute to the development of asthma, so the relationship was studied between home environment factors and asthma in children and adults people with socioeconomic background. In many studies the following factors were associated with asthma: damage caused by dampness in child's sleeping area (adjusted odd's ratio (OR) 4.9:95% confidence interval 2.0-11.7), air pollution at home (OR 2.5, 95% CI2.0 to 6.4), presence of rugs or carpets in child's bedroom (OR 3.6; 95 % CI 1.5 to 8.5) (Mohamed et al., 1995).

The importance of previous sensitization to various indoor allergens such as house dust mites (HDM) (Sporik et al., 1990), cockroaches (Duffy et al., 1998), fungi (Abramson et al., 1996), and cats (Saprong et al., 1997) as risk factors for asthma has been increasingly recognized. However, the impact of current allergen levels on sensitization and clinical activity of asthma is controversial and has not been investigated adequately, particularly in adults. Although it is widely accepted that symptoms of asthma are directly related to the current environmental allergen levels, this has not been established. Some studies have suggested that the current levels of HDM allergen (Custovic et al., 1996), cockroach allergen (Eggleston et al., 1998), fungi (Garrett et al., 1998), and cat allergen (Tunnicliffe

et al.1999), are related to sensitization and clinical activity of asthma, whereas others have contradicted these findings (Marks et al., 1995).

The literature suggests that indoor allergens may be inter-related. Fungi play an important role in the food chain of house dust mites as HDM flourish only on human dander that has been predigested by fungi (van Bronswijk et al., 1973). Concentrations of cat allergen were elevated in all houses with cats (Raunio et al., 1998) and cat ownership was found to be associated with high levels of viable airborne fungi and ergosterol levels in floor dust (Dharmage et al., 1999). None of the studies that examined the association between allergen levels and asthma has accounted for the confounding effects of inter-related exposures, whereas only few studies have accounted for other potential confounders such as medication use and parental asthma.

However, such factors as indicated in above discussion, do not totally explain the increasing morbidity and mortality of asthma. Asthma mortality rates are known to vary by age, geographic region, and race/ethnicity with higher rates for minorities. The present study focuses on the feasibility of using Geographic Information System (GIS) in conjunction with risk factors, exposures (Toxic Release Inventory) and demographic information (racial/ethnic differences) in asthma mortality. This study has two aspects; a) the assessment of the potential relationship between toxic releases and asthma mortality and b) the identification of existing disparities for asthma mortality by gender and race/ethnicity. We will attempt to determine whether environmental exposures have a different impact on these groups of the population over a time. Other outdoor environmental factors such as temperature, humidity, and air pollution will also be

considered and adjusted for, if necessary, to control confounding.

Racial /gender disparities:

The prevalence and severity of asthma are high among African-Americans (Weiss et al.1998) and Hispanics. Black children in the United States were more likely to have asthma or wheeze than white children (National health survey data). A large proportion of the racial disparity in asthma prevalence could be accounted for by the social and indoor environmental exposures like maternal smoking. Nevertheless, after adjustment for other significant exposures, black children still had 1.7 times the asthma rate of the white children (National Health and Nutrition Examination Survey (NHANES)). These national surveys may provide little detail on the environmental exposures and their relationship to the racial differences in disease prevalence within specific geographic areas. Asthma mortality rates increased despite our improved understanding of the pathophysiology of asthma and advanced methods of treatment (Wissow et al., 1988). The literature suggests that there has existed a disparity of asthma mortality in the racial groups over time in the U.S. Many explanations have been proposed to account for the uneven distribution of asthma admissions among racial and ethnic subpopulations. Some studies argued that low socio economic states are associated with small area variation in asthma mortality rates. The studies suggested that Black race /ethnicity and low socioeconomic status (SES) may be independent risk factors for asthma mortality (Maradar et al., 1992). However, other analyses suggest that both low SES (as measured by an ecological variable) and Black race/ethnicity are independently associated with excess asthma mortality and those independent contributions of race /ethnicity may be greater than that of SES (Smith et al,

1997). In 2001 over 3.2 million Hispanic Americans had been diagnosed with asthma in their lifetime. Two million Hispanic Americans reported that they still have the disease, and 1.2 million of those experienced an asthma attack in the past year. Prevalence rates in Hispanics were significantly lower than Non- Hispanic blacks and Non-Hispanic whites in 2001. Studies have suggested that within Hispanic subgroups, Puerto Ricans may have higher rates of asthma than other Hispanic subgroups and non-Hispanic whites (Ledogar et al, 2000). Across all age groups, females had higher death rates than males. In addition to age and sex inequalities, significant racial disparities in asthma mortality also exist in Texas. Mortality rates were highest among African-Americans (3.1 per 100,000), compared to 1.9 per 100,000 in Whites and 0.8 per 100,000 in Hispanics in 1998. The reason for these disparities is not entirely clear, although access to and quality of health care may play a role.

Lifetime prevalence based on the 2001 NHIS (National Health Interview Survey) sample, it was estimated that 31.3 million Americans or 113.4 per 1,000 persons had been diagnosed with asthma by a health professional within their lifetime. Between1997 and 1999 asthma lifetime prevalence rate decreased by 6%, however it has increased 25% since1999. Between 1997 and 2001, children 5-17 years of age have had the highest prevalence rates. In 2001, 144.2 per 1,000 children ages 5-17 had been diagnosed with asthma in their lifetime. Females have had consistently higher rates of asthma prevalence than males. In 2001, females were about 10% more likely than males to ever have been diagnosed with asthma. This was the first time that the difference between sexes for lifetime prevalence was statistically significant. In 2001, the prevalence rate in blacks was

close to 15 percent higher than in whites. Since the year 1997 the differences in lifetime asthma prevalence between races have been statistically significant. In 2000, 4,487 people died of asthma. Approximately 65% of these deaths occurred in women. The age adjusted death rate in 2000 was 1.6 per 100,000. The female death rate was 39 percent greater than the rate seen in males and the age-adjusted death rate for asthma in the black population (3.9 per 100,000) was three times the rate in the white population (1.3 per 100,000). Black women had the highest mortality rate due to asthma in 2000 (4.2 per 100,000). In this year, 292 Hispanics died of asthma - an age-adjusted death rate of 1.5 per 100,000 population. Age-adjusted death rates in Hispanics were 63% lower than non-Hispanic blacks, but 15% higher than non-Hispanic whites. Blacks and Hispanics are less likely to receive regular medical care than whites, and such care is essential for optimal control of asthma. Blacks and Hispanics are more likely than whites to obtain care for asthma in an emergency room (Coultas et al., 1994). They are also more likely to be less educated than whites, (Coultas et al., 1994) to be younger,(Bureau of the Census, 1992) and to live in more crowded conditions and thus to have more respiratory tract infections (Margolis et al., 1992). However, a recently published study found that Puerto Ricans had higher age-adjusted death rates than all other Hispanic subgroups and non-Hispanic whites and blacks. Asthma deaths are rare among children and highest among those over 85. In 2000, 223 children ages 0-17, or 0.3 per 100,000, died from asthma compared to 707 adults 85 and over, or 16.5 per 100,000 population. The rate in the 85 and over population was 139% greater than the second highest mortality rate of 6.9 per 100,000 population, seen in 75-84 year olds. Unlike morbidity estimates, which are drawn from sample populations and extrapolated to

the overall population, mortality data is obtained from the general population by way of death certificates. Therefore, the sex- and race-specific numbers and rates denote true differences, not estimates. As seen in recent years the number of deaths due to asthma continues to increase, even after the ICD-10 revision is taken into account.

Women have a greater prevalence of asthma than men (McWhorter et al., 1989). In the Philadelphia metropolitan area, increased morbidity among women with asthma has been reported (Skobeloff et al.1992). It was found that the death rate from asthma was significantly increased in census-tract areas with higher proportions of female residents. Substantially more men than women are undercounted in the census, and this undercounting is greater for black men than for other groups of men (Bureau of the Census, 2000). It was also believed that the association between death from asthma and female sex might in part be spurious, because of potential confounding by inaccurate census data.

Environmental Factors

The environmental factors that are implicated are overcrowding, old buildings structures, the presence of vermin, cockroach antigens, dust mites, rodent dander, air pollution, and poorly ventilated living spaces (Sly et al., 1996). In the United States, deaths from asthma occur predominantly in large cities, (Carr et al., 1995) suggesting an association with the urban environment. There were few studies that used the environmental exposures like TRI and this study gives us a unique opportunity to examine whether different environmental exposures including TRI, can account for the racial differences in asthma prevalence. Deaths from asthma increased in the United States from

2,598 in 1979 to 3564 in 1984 and rates have increased from 1.2 to 1.5 per 100,000 general population. Rates of death from asthma have continued to be substantially higher among blacks and Hispanics than whites (Sly et al., 1996). Mortality has increased for every age group (Sly et al., 1996). Asthma many be certified routinely as the underlying cause of death with greatest certainty from age 5-34 years (Jackson et al., 1989). To identify regional differences in mortality from asthma, data will be assembled by state, race, and year and data grouped for states into geographic regions. Studies have shown that air pollution is related to the worsening of asthma symptoms. One study of young campers with moderate to severe asthma revealed they were 40 percent more likely to have acute asthma episodes on high pollution summer days than on days with average pollution levels (Smith et al., 1999). Another recent study found that the number of daily hospital emergency room visits by older adults due to respiratory distress increased significantly as the air pollution levels in the summer months increased (Peat et al., 2001). This latter study tried to concentrate on TRI as the cause of asthma mortality in different racial groups. There were significantly higher rate of the corresponding asthma mortalities in women than men in the age groups 35-64 (Weiss et al., 1993). Asthma mortality rates were strongly associated with increasing age, but no consistent differences were observed between men and women. Mortality rates among blacks less than 65 years were 2-4 times the corresponding rate among whites between 1960 and 1989, but this difference not observed for those over 65 years of age (Schenker et al., 1999).

The Toxics Release Inventory (TRI) collects information about chemical release and waste management reported by major industrial facilities in the U.S. Toxic releases trigger

asthma because it is extremely irritating to the lungs and airways. It is well established that the concentrations of Toxic releases are directly related to asthmatic attacks, need for increased doses of asthma drugs, and emergency treatment for asthma (Baxter et al., 1990). Asthma attacks are most common in cities where automobile concentration is greatest, and in the summer when the right conditions of sunlight and low winds occur. Under these conditions, we become literally surrounded by a toxic release cloud. The United States Environmental Protection Agency (EPA) defines air pollution as "any visible or invisible particle or gas found in the air that is not part of the natural composition of air." The EPA using the Pollution Standard Index (PSI) reports air pollution. A PSI of 100 or more is dangerous for people with asthma and requires special precautions and planning. If you have asthma, you should also know that your symptoms could worsen even when pollution is moderate (PSI 50-100). You may still have to adjust your activities and medications.

A federal law called the Emergency Planning and Community Right to Know Act (EPCRA) gives you the right to know about toxic chemicals being released into the environment. The law requires facilities in certain industries, which manufacture, process, or use significant amounts of toxic chemicals, to report annually on their releases of these chemicals. The reports contain information about the types and amounts of toxic chemicals that are released each year to the air, water, and land as well as information on the quantities of toxic chemicals sent to other facilities for further waste management. As a component of EPCRA (Emergency Planning and Community Right to Know Act), certain manufacturers are required to report annually the total mass (pounds per year, lb/yr) of toxic chemicals released into the environment (air, water, land, or underground injection),

treated on-site, or shipped off-site for further waste treatment. This information is compiled by the U.S. Environmental Protection Agency (EPA) into a publicly accessible database known as the Toxic Release Inventory (TRI). The TRI database is designed to encourage pollution prevention and waste reduction by increasing public access to and knowledge of environmental chemical releases. TRI facilities are those that manufacture more than 25,000 pounds or use more than 10,000 pounds per year of certain toxic chemicals listed by the federal government. Many TRI facilities emit thousands of pounds of toxic substances to the atmosphere, to water, or to land in Texas. Begun in 1988, the Toxics Release Inventory contains information on releases of nearly 650 chemicals and chemical categories from industries including manufacturing, metal and coal mining, electric utilities, and commercial hazardous waste treatment, among others. You can easily find information on toxic chemical releases over the Internet at (www.epa.gov/triexplorer). For reporting year 1994, for instance, chemicals released into the air from Texas TRI facilities include 62,600 pounds of Trichloroethylene, 30,500 pounds of Tetrachlorothylene, 23,000 pounds of 1,1-Dichloro-1-Fluoroethane, 11,000 pounds of Sulfuric acid, 8,500 pounds of Hydrochloric acid, 6,650 pounds of Xylene, 4,800 pounds of Toluene, and 1,000 pounds of Glycol ethers. All of these chemicals are known to cause asthma. The current TRI toxic chemical list contains 582 individually listed chemicals and 30 chemical categories (including three delimited categories containing 58 chemicals). If the members of the three delimited categories are counted as separate chemicals then the total number of chemicals and chemical categories is 667 (i.e., 582 + 27 + 58). The TRI program has expanded significantly since its inception in 1987. The Agency has issued rules that roughly double

the number of chemicals included in the TRI to approximately 650 in 2002.TRI facilities are required to report releases and other waste management of specifically listed chemicals. They also are required to report transfers of toxic chemicals for waste management to off-site locations. Facilities that meet all three of the following criteria are subject to EPCRA Section 313 release and other waste management reporting: a) The facility has 10 or more full-time employees; b) The facility has a primary Standard Industrial Classification (SIC) code, in any of the groups listed in the table on the following page; and c) The facility manufactured (defined to include imported), processed, or otherwise used, in the course of a calendar year, any toxic chemical in quantities greater than the set threshold.

During 1982-1996 asthma increased by 84.8%. This growth rate is expected to continue. The cause of this increase has not yet been explained. The number of asthma deaths in America dramatically increased. During 1979-1997 the number of asthma patients in America increased by 109.2%. Different levels of asthma prevalence exist among races. Compared with whites, more blacks are affected by asthma. The gap of asthma prevalence between whites and blacks has increased from 2.6 % to 16.4% since 1985. Racial disparity in asthma mortality is more significant. Asthma mortality among blacks and other minorities is twice as high as that of other races. Asthma mortality among blacks is the highest. Feminization of asthma in America is highly significant. While male patients grew by 40% during 1982-1996, female patients expanded by 126.4% during the same period, this accounts for three times growth rate. The sex gap in the number of asthma patients has widened from 88 to 3094.Asthma prevails more among low-income than high income people. (http://www.lungusa.org/asthma).

A recent study by the American Lung Association Asthma Clinical Research Centers found that the inactivated influenza vaccine is safe to administer to adults and children with asthma, including those with severe asthma (American lung Association, 2000). Asthma is one of the most common chronic diseases in the United States, and it has increased in importance during the preceding 20 years. Despite its importance, no comprehensive surveillance system has been established that measures asthma trends at the state or local level. Asthma is a chronic inflammatory disorder of the airways characterized by variable airflow obstruction and airway hyperresponsiveness in which prominent clinical manifestations include wheezing and shortness of breath (Sheffer et al., 1993) is a multifactorial disease that has been associated with familial, infectious, allergenic, socioeconomic, psychosocial, and environmental factors (Weiss et al., 1993). Asthma morbidity and mortality are largely preventable with improved patient education regarding the factors associated with asthma and medical management (Barbee et al, 1985 and Weiss et al, 1992).

Socioeconomic factors that have been implicated in this trend of increasing asthma morbidity include limited health-care access, inadequate health insurance ,lack of recognition of asthma severity by patient or physician, psychosocial dysfunction of patient and family, overuse or inappropriate use of asthma medications , and exposure to indoor and outdoor environmental agents (Weiss et al., 1993).

Prevalence

Respondents to the Texas Behavioral Risk Factor Survey have been asked if a doctor has ever told them that they had asthma. According to the combined data from 1999

through 2001, approximately 1.5 million adults (10.5% of the population) have been told by a doctor, nurse or health professional that they had asthma. Among adults, females are about 20% more likely to have ever been diagnosed with asthma. Lifetime asthma prevalence was significantly higher in the youngest age group, 18 to 24 year olds. Hispanic adults are less likely to report having ever been diagnosed with asthma than Non-Hispanics. Prevalence of life time asthma diagnosis of asthma in Texas is for total population is 10.5, for males 9.4, for females 11.6, for whites 13.0, for blacks 11.4 and for Hispanics 7.0 (Texas Behavioral Risk Factor Surveillance System, 1999-2001.). Combining data from 1999 through 2001, approximately 900,000 (6.2 % of the population) adults currently have asthma. Estimated prevalence of current asthma among women is almost double that for adult males. Current asthma prevalence ranges from 8.6 – 5.4% across the age groupings with the highest prevalence among 18 to 24 year olds.

Asthmatic mortality during the years 1980-2000 has been significant In Texas. Death from asthma is uncommon but nationally and in Texas, the number of deaths and death rates has increased gradually during the last twenty years. The rate of increase for asthma deaths was sharpest among persons aged 65 years or older. In Texas, there were 4,831 deaths due to asthma from 1980 through 1998 (1.5 per 100,000 population). Non-Hispanic Blacks were the most likely to die from asthma with a death rate more than 200% higher than non-Hispanic Whites or Hispanics. Females had an asthma death rate about 40% higher than male (Texas Department of Health, Bureau of Vital Statistics).

One way we can assess the morbidity, or impact on quality of life, of asthma is by determining the rate of hospitalizations. Hospital Discharge data are useful for

characterizing the burden of asthma in Texas and for identifying the populations that are most severely affected. Between 1999 and 2001, there have been over 20,000 hospitalizations for asthma per year in Texas. In most cases, these hospitalizations could have been avoided with appropriate treatment and care. These hospitalizations unnecessarily create a burden not only in financial costs but also in terms of missed school, work, and other activities. Section 313 of the Emergency Planning and Community Right-To-Know Act (EPCRA) of 1986 established the TRI database. Under EPCRA, industrial facilities in specific sectors are required to report their environmental releases and waste management practices annually to the Environmental Protection Agency. Covered facilities must disclose their releases of approximately 650 toxic chemicals to air, water, and land, as well as the quantities of chemicals they recycle, treat, burn, or otherwise dispose of on-site and off-site. A more recent study also showed an association between air-pollutant concentrations and the number of visits to emergency departments for asthma (Schwartz et al., 1993).

The prevalence of asthma in the United States has increased over the past two decades, and data suggests trends in Texas have followed the same pattern (Centers for Disease Control and Prevention, 2002), with approximately 1.5 million adult Texans (10.5% of the population) have been told they have asthma. Among them, approximately 900,000 (6.2 % of the population) adults currently have asthma. Additional indicators of asthma morbidity include the number of emergency department visits, as well as limitations in activity among persons who have asthma and number of school or work days missed by persons with asthma (U.S. Department of Health and Human Services, 2000). Currently, sources of data

for these indicators have not been identified in Texas. From national data we know that asthma is the leading cause of school absenteeism due to chronic illness for children under 16 years of age. Children with asthma miss twice as many school days as do children without asthma (The American Lung Association of Texas, 1999). In 1993, direct and indirect expenditures for asthma exceeded $12.6 billion in the United States. Of importance is that 20% of people with asthma account for more than 80% of the total asthma direct costs (Smith etal, 1993). Based on national studies, the Allergy and Asthma Foundation of America estimates the total medical expenditures in Texas for asthma to be $763 million per year. $435 million of this is in direct medical expenditures, and $328 million is due to indirect costs such as school/work days lost and deaths (Allergy and Asthma Foundation of America, 2000). From 1980 to 1998, the asthma mortality rate nearly doubled in all age groups in Texas, with the highest death rates occurring in the population over 60 years of age. In 1998, 343 Texans died from asthma. The Environmental Protection Agency (EPA) has set National Ambient Air Quality Standards (NQAAS) for six "criteria pollutants" considered harmful to public health, including ground-level ozone (smog), particulate matter, lead, nitrogen oxides, sulfur oxides, and carbon monoxide. In Texas, four urban areas do not meet the federal standards for at least one of the criteria pollutants; these are Beaumont-Port Arthur, Dallas-Fort Worth, El Paso, and Houston-Galveston-Brazoria (The Environmental Protections Agency, 2002).

Web Geographic Information Systems (Web-GISs) is a geographic information system providing environmental, social economic and geographic information using technologies though the Internet. This technology has been successfully developed and applied by

Environmental Justice communities in co-operation with academic partners to provide citizens and activists with a new tool allowing people to overlay data layers and to understand possible relationships between them. The linking of population, pollution and outcome data allows Geographic Information Systems (GISs) researchers to go beyond simple mapping of disease rates by predetermined boundaries and to initiate the creation of meaningful spatial analyses (Briggs et al. 1995) GISs are hardware and software packages designed to integrate map feature with data that are related to defined geographical attributes (Vine et al. 1995). By linking environmental and health data, the investigator s may view GISs as tools for identifying high-risk areas, thus leading to proactive public health action (Waller et al., 1996). With surveillance provided by GIS, investigators can quickly identify areas for further study (Wittie et al, 1999), and environmental health hypotheses from spatial analysis of toxic exposure problems can be formulated and tested.(Briggs et al. 1995).Two other features of GIS are geocoding, or address-matching , which allows mapping residencies on to a Cartesian grid axis; and (buffering techniques, which distinguish "exposed" populations for analysis (Briggs et al, 1995).Geocoding uses a map layer, which contains a street network attributed with street names and types ,and it addresses ranges for both sides of the street. The software program interpolates along a particular street segment to place an address. Typical geocode rates will range between 20% up to nearly 100% for a rural state, depending on the urbanization of the area studied (Vine et al, 1995). This project is important because it is designed to identify and assess environmental risk factors that may play an important role in the increasing asthma problem. This research uses Geographic Information Systems (GIS) and Spatial Scan

Statistics software. These data will be analyzed and presented to illustrate primary (significant) clusters of asthma mortality for counties in Texas. GIS is a potent tool that has been under-utilized in medical studies. This study tries to explore the potential of GIS to investigate the relationship between environmental factors and the ethnic differences in asthma mortality. The results of this study will hopefully provide information to identify high-risk groups and environmental factors that could be modified to prevent asthma mortality.

From an Environmental Justice perspective, the Community University Consortium for Regional Environmental Justice (CUCREJ) views Web-GIS as a means providing EJ communities with key environmental, social, economic, and geographic information on the Web. Maps that correspond to their neighborhoods and, in an interactive framework, overlay the data they need to understand, to plan, and to undertake environmental actions. CUCREJ has engaged in Web-GIS projects as early as 1996 and continues to pursue such efforts and ensure that the projects are sustainable, extensible and accessible. It is important that the base map reflects the community's self-definition.

There has been a lack of studies that examine spatiotemporal trend of this disease burden, and that quantify the disparity that has been exerted on different racial groups over time. In the present study data developed through Geographic Information Systems (GIS) on locations of areas that release toxic chemicals were combined with information concerning asthma mortality and compared over a period of time for different racial groups. Black race/ethnicity appears to be associated, independently from low income and low education, with an elevated risk for asthma mortality (Grant et al., 2000). In the

present study, it could be identified as to which regions have more asthma related deaths and by studying race and ethnicity it can be shown which particular race is in proximity to a particular TRI site. From such data, it can also be concluded whether a particular race is more susceptible to the environmental TRI exposure reflected by increased asthma mortality. The environmental Protection Agency provides many kinds of environmental data including Toxic Release Inventories Air Release site, superfund site information and much more. These data can be accessed through http://www.epa.gov.

CHAPTER III

DATA COLLECTION AND TREATMENTS

US mortality data were obtained from the National Center for Health Statistics for years 1980 to 2001. Case identification was based on asthma (International Classification of Diseases, Ninth Revision [ICD-9]) as the cause of death. Additional vital information the study will utilize publicly available asthma mortality data, available through the Texas Department of Health website. A Spatial Scan Statistic (Sat SCAN) will be applied to differentiate excess asthma among counties in Texas between the year 1980 and 2001.

The analysis will adjust for covariates (demographic risk and vulnerability factors) to mortality such as age, gender and race/ethnicity. Gender will differentiate between male and female populations. Lastly, race/ethnicity will include Anglo, African American, and Hispanic/Latino populations. Geographic Information Systems (GIS) such as ArcGIS software will also be applied to present a visual representation of the analysis results in the format of Texas county clusters, both primary and secondary as determined through relative risks. These identified clusters will be used in recommendations to support a localized approach to screening interventions for reducing asthma mortality in Texas. Action plans produced by the Texas Cancer Council will be reviewed and based on the results of the analysis; recommendations will be produced. The spatial Scan Statistic factors in the uneven geographical population density and conditions the analyses on the total number of observed asthma deaths. In an analysis of rare case/deaths such as asthma

deaths, the Poisson model can be used for estimating probability distribution when case number is small compared to the population at risk. The null hypothesis of the Poisson model provides that, when there are no covariates, the expected death counts in each county are proportional to its population size or person years in that area. In the present study, for each location and size of the scanned space and time, the alternate hypothesis refers to elevated asthma mortality rates within space and time as compared to outside areas under study. Calculations can be performed using SatScan Program Version. The program first analyzes cases and adjusts for covariates to produce Log Odds Ratio (LOR) for observed cases. SatScan then aggregates these adjusted data by scanning through and plotting circles around geographic identifiers in a population size specified by the user across the study area. To identify potential excess asthma mortality in Texas counties between 1980 and 2001. The first, "Deaths Files by Race", included asthma deaths (ICD-9 Code 174 and ICD-10 Code C50), which reported the place of residence in 254 Texas counties of 4 racial groups, that were coded as categorical data (e.g., non-Hispanic White=1, Black=2, Hispanic=3 and Other=4) in each of the 22 study years. One file per racial group was created, including 5653 records reflecting the number of deaths for each race among the 16 age groups in 254 counties over the 22-year study period. The second file, the "Population File", contained data on the populations at risk in the study period. The population data for the remaining years were obtained from population estimates made available through the Texas State Data Center and the Center of Vital Statistics of the Texas Department of Health. [Population Data, TDH] The Population file contained a total of 195,072 records, representing the four races and in 254 counties for the 12-year study

period. Another file, the "Geographic File", was also obtained from the US Census Bureau. [US Gazetteers] This file contained the latitude and longitude information of Texas county centroids as a proxy that indicated the locality of each county. The Texas county shape files were obtained from the CDC Website (URL http://www.cdc.gov/epiinfo/usa/tx.exe) for further mapping analysis. Also, Medically Underserved Areas (MUA) counties data were gathered from the TDH Health Professional Resources Center (Thomas, 2001).MUA is an official designation for those counties having shortage of personal health services according to the U.S department of Health and Human Services rules. The criteria for MUA designation include: 1) percentage of elderly population (over 65 years); 2) poverty rate; 3) infant mortality rate; 4) ratio of primary care physicians per 1000 population (TDH). Both the MUA and TRI reporting county layers were overlaid on the asthma mortality clusters' layer to examine their potential associations.

To detect the effect of potential interventions on the asthma clusters, both Case and Population Data files also include surrogate predictors of interventions at the county level between the years 1996 and 2001, amount reported in pounds of total quantity of the toxic chemical released" (including offsite disposal) from the TRI Program of the US Environmental Protection Agency. There were (6,258 records) of TRI monitored facilities in 136 counties retrieved in the 6 year study period.[EPA] The toxic release data were first aggregated to the county level and then transformed to categorical data ranging from 1-10 with category (1) representing the lowest centile of measures and category (10) representing the highest for those Texas counties with TRI surveillance data. The non-reporting counties were treating as missing data and coded 0.

CHAPTER IV

STUDY DESIGN AND METHODOLOGY

The present research will be conducted by using an ecological study design based on geographic information systems and spatial statistical analysis. The geographic variation of disease rates was evaluated by a spatial scan statistic (Kulldorff et al., 2003), which uses a large number of scanning circles (more than 100K) of varying size (1% to 50% of the at-risk population) and location (independent of conventional geo-political boundaries) to search for places at which the number of observed cases deviated from a null hypothesis random incidence (i.e., proportional to population density). This test statistic was adopted previously for detecting excesses of breast (Kulldorff et al., 2003) and prostate cancers (Gregorio et al., 2000). When compared with other statistical methods for cluster detection, this statistic was found to have greater power for detecting localized hot spots of excess mortality. The Poisson model can be used for estimating the probability distribution when the number of deaths is substantially smaller than that of the population at risk. TRI State Data Files are sets of files containing all data submitted to the Toxic Chemical Release Inventory by facilities located in a selected state for a specific year. The data have been extracted from the Toxics Release Inventory System (TRIS). The data that were studied from 1996-2001 and then compared with the analysis of the asthma mortality. A handful of publications have used the GIS tool in epidemiological studies (White et al., 1999). These few studies give an indication of the potential of GIS as a tool to investigate the

relationship of environmental factors to the incidence of cancer. With GIS it is possible to superimpose layers of information (Krautheim et al., 1997). No study was done comparing the environmental factors and asthma.

This study used the Spatial Scan Statistic developed by Kulldorff and colleagues (Kulldorff et al., 1995) to detect potential excess asthma mortality. This statistic was found to have good power for detecting localized hot-spots type of excess events, when compared with other statistical methods for cluster detection, particular those in nonurban population, and was used in more than 40 health studies (Song et al., 2003). The Spatial Scan Statistic seems ideal for detecting potential excess asthma mortality in the state of Texas, which has a special combination of urban and rural populations. The Spatial Scan Statistic is unique since it factors uneven geographical population densities and conditions, and then analyzes the total number cases, which are asthma related deaths. Spatial Scan Statistic searches for clusters of cases without specifying their size or location ahead of time, which tests for their statistical significance at the same time adjusting for the multiple testing inherent in such a procedure. The Poisson model can be used for estimating the probability distribution when the number of cases/deaths is substantially smaller than that of the population at risk. The null hypothesis of the Poisson model provides the expected death counts in each county if there are no covariates. The null hypothesis provides the death counts that are proportional to the population size (or person-years) in that area. The alternative hypothesis states that deaths are not randomly distributed. In the present study, for each location and size of the scanned space and time; the alternative hypothesis refers to elevated adjusted mortality rates within space and time as compared to outside areas under study.

Calculations can be performed using the SatScan Program (version 4.0, freeware available from URL: http://www.satscan.org). SatScan first aggregates data with the scanning window of spatial (referring to the population at risk) – as a cylinder base, and temporal (years equal to seven in this study) – corresponding to the height of the cylinder as selected by the users. For each cylinder, the scan adjusts for covariates and calculates the Log Likelihood Ratio (LLR, formula described below) by scanning through and plotting circles around geographic identifiers (prepared in the Geographic File) in a population size specified by the user across the entire study area. The base is the same as defined in spatial statistics, while the height reflects the time period of potential clusters. The cylindrical window moves in space and time and scans through each possible geographic location, defined by county centroids in the present study. The overall relative risk for each cluster, along with a set of simulated values based on the same procedure within a specific space and time, are then calculated. The latter are used as a baseline against the LLR values of the observed values. SatScan employs the Monte-Carlo simulation to estimate the LLR. When the LLR values of observed windows are higher than LLR based on simulation, SatScan determines the deaths in a particular region that are significantly different from the rest of the study area for the particular time window by rejecting the null hypothesis. Under the Poisson assumption, the Likelihood function for a specific space-time is then proportional to:

$$LLR = (c/n)_c([C-c]/[C-n])_{(C-c)}I()$$

where C is the total number of asthma deaths, c is the number of cases within the space time window, and n is the covariate(s)-adjusted expected number of deaths within the

space-time analysis under the null-hypothesis. I () is an indicator function, whereby I() is equal to 1 when the timeframe has more deaths than is expected under the null-hypothesis, and is 0, otherwise. Based on a test statistic value of the LLR, a p-value is then calculated which suggests how well all the variables fit into the model at the same time. SatScan performs adjustments by indirect standardization. For the present study, the Poisson model was used to calculate the number of expected deaths in each county. The space-time retrospective analysis was conducted without prior assumptions as to the size or location of such areas or duration of excessive mortality. The scan setting was set at a maximum spatial cluster size of 90% of the study period (i.e., 10 years) and 50% of the population at risk. We employed the space-time retrospective analysis using the Poisson model and defined the maximum spatial cluster size at 50 percent of the population at risk suggested by Kulldroff (Kulldroff., 2003) as an optimal value setting that maximizes the effect of potential cluster detection. The test statistic analyzed cases by factoring in 1) space (calculated by lat/long); 2) time (by year); 3) background population (at risk); 4) race; 5) age-groups and 6) intervention variables. It performed adjustments by indirect standardization. This means that a cluster would comprise, at most, 50% of the population at risk. The study further tested the potential persistence of temporal clusters across the entire study period (i.e., 22 years) by holding constant the 50% maximum spatial cluster and scanning with the "purely spatial" option. For data processing, we developed a Visual Basic application to automate data collection and manipulation, and output the results to geographic information systems (GIS) for performing mapping and spatial queries. The SatScan program saved the output files, including cluster locations, relative risk for each

location, simulated LLRs, and the test statistic, in database (dbf format) files. Data were stored on a Microsoft SQL Server version 7.0, and The SatScan program calculated LLR by performing 999 instances of Monte Carlo replications. The automated process, including data input, scanning, and output, took an average of 45 minutes of computer time.

CHAPTER V

RESULTS

The study included 5653 asthma deaths among an average population of 17,577,292 in Texas counties across the 21-year study period. The age-and-race-adjusted annual mortality rate of all races was 1.5/100,000 persons/year. Annual age-adjusted mortality rates for non-Hispanic Whites, Blacks, and Hispanics and for "Others" were 1.6, 2.7, 0.5 and 1.0 per 100,000 persons respectively. Of all the population all the Texas counties black males have the highest relative risk of asthma mortality at 1.411 with a p-value of 0.008.Of all the races in Texas white race has the highest relative risk of asthma mortality with a value of 1.351 (p-value =0.001). Black race has the highest temporal persistence for a period of 15 years from 1985-1999, and black females had the highest temporal persistence. Black race had temporal persistence for more than half of the study years. Two clusters have the longest temporal persistence to the present decade (Sabine, Fayette counties). The total numbers of cases were 5653 from 1980-2001.The number of cases found in females were 3458 and are more than males, which is 2195, reflecting the study and census reports about asthma mortality. The numbers of cases in White population are 3781, which are significantly higher than that of the black race, which has 1232 cases. Hispanics were 552 and other races were 88.Total cases in white males were 1366 and white females were 2415. The number of cases in Black males was 546 and Black females was 686.The total

number of cases for Hispanic males was 237 and females was 315.In other race category number of cases for males are 46 and for females 42.

With the adjustment of age and stratification by race, a scan window of a maximum of 90% of the study period (i.e., 10 years) and 50% of the population at risk revealed 23 regions of likely excess mortality in four racial groups within Texas population. Of these, four regions were statistically significant. Figures to 4 to 7 present these likely areas of excess mortality in this set of analysis. If scan is performed for all the population on a gender basis for the population as a whole, there were 11 clusters with 6 significant areas of excess mortality. Figures to 1 to 3 present these likely areas of excess mortality in this set of analysis. If the scan was performed with the gender basis in each race then 30 regions have been identified with likely excess mortality in four racial groups within Texas population. Among these, four regions were statistically significant in terms of both spatial and temporal excess. Figures to 8 to 15 present these likely areas of excess mortality in this set of analysis. To describe the extensive geographic regions of the state of Texas in a consistent manner, we adopt the term used in the "counties and regions cross reference" for each public health regions of Texas, as defined by the Texas Health and Human Services Commission (URL http://www.hhsc.state.tx.us/about_hhsc/HHS_Regions.html). For the whole population Texas counties, three potential excess mortality regions were identified. The most likely area of excess mortality with a Relative Risk (RR) of 1.310 (p=0.001) occurred between 1989-1998 in High Plains, Northwest Texas Metroplex, Upper Rio Grands, West Texas and Central Texas. These included 162 counties ranging from Briscoe to Robertson.

A secondary excess mortality region (RR= 1.495, p=0.001) was identified between years 1988-2001 in the 23 counties of Gulf Coast and upper South Texas. A second secondary excess mortality region was identified (RR=1.365, P=0.007) between years 1990-2000 in the 20 counties of Upper East Texas along South East Texas. Both the secondary clusters were significant.

For the non-Hispanic White population, five potential excess mortality regions were identified. The most likely area of excess mortality with a Relative Risk (RR) of 1.351 (p=0.001) occurred between 1989 and 1998 in High plains along Northwest Texas and Metroplex, West Texas and Upper Rio Grande. These included 151 counties ranging from Swisher to Bandera counties. A secondary excess mortality region (RR=2.415, p=0.014) was identified between 1995 and 1999 in the 12 counties of Lower South Texas. Another secondary potential excess area in the non-Hispanic White population was detected in 5 counties during the years 1990-1998 in Southeast Texas along Gulf Coast that was statistically significant (RR=1.785, p=0.019). Two other regions with excess mortality in the White population were identified but they were not statistically significant .One was at 4 counties in Central Texas during the years 1990-1996 (RR=2.781, p= 0.128) and another was in a Trinity county during the years 1993-1995 (RR=7.689,p=0.990).

For the Black population, the most likely area of excess mortality (RR=1.304, p=0.001) occurred between years1985 and 1999 in Northwest Texas, Metroplex and Central Texas. These included 110 counties ranging from Bosque to Lavaca. This cluster was statistically significant. Additionally, another region was identified with potential

excess mortality with a relative risk of 3.711 and p value of 0.089. This secondary cluster was found during the years 1988-1995 in the Lower South Texas, which included 16 counties ranging from Live oak to Dewitt. For the Hispanic population, the most likely area of excess mortality (RR=37.464, p=0.138) occurred between 1989 and 1997 in Yoakum. This cluster was not statistically significant. Additionally, seven other clusters were identified which were not statistically significant (p>0.2). For the "Other" population, two clusters were detected, and none of these were statistically significant (Primary cluster, p=0.103 and secondary cluster, p=0.504). For the white males, six potential excess mortality regions were identified. The most likely area of excess mortality with Relative Risk (RR) of 1.411 (p=0.001), occurred between 1991-1998 in High Plains, Northwest Texas, Metroplex and Upper East Texas. These include 119 counties ranging from Clay to Bowie. A secondary excess mortality region was identified with a Relative Risk of 4.628 (p=0.143) in Central Texas in counties of Fayette, Colorado, Lee and Bastrop. Additionally, four other clusters were identified with potential excess mortality but they were not statistically significant (p>0.2).

For the white females, the primary excess mortality region was identified with a relative risk (RR) of 1.398 (p=0.001) during the years 1989-1998 in High Plains, North West Texas, West Texas and Upper Rio Grande. These included 141 counties ranging from Terry to Williamson. A secondary excess mortality was identified with a Relative Risk (RR) of 2.462 (p= 0.003) in Lower South Texas in 17 counties ranging from Kleberg to Starr. Both these clusters of the white females were statistically significant. For the black males, the primary excess mortality region was identified with a Relative

Risk (RR) of1.444 (p=0.008). This occurred between 1990-1999 in Northwest Texas, Metroplex, Upper East Texas and Central Texas which included 110 counties ranging from Bosque to Sanjacinto.A secondary cluster was identified with a relative risk of 32.110 during the year 1997 but was not significant (p= 0.159).It was identified in the High plains and West Texas in ten counties ranging from Gaines to Ector. Two other regions were identified with excess mortality in the black males but none were significant. In black females, the primary excess mortality was identified with a Relative Risk (RR) of 1.322 (p=0.001) between the years 1985-1999 in Upper Rio Grande, West Texas, Northwest Texas, Metroplex, Central Texas Upper South Texas and Lower South Texas. These included 183 counties ranging from Terrell to Waller .A second cluster was also found with excess mortality (RR= 4.793), but it was not statically significant (p=0.716).In Hispanic males two potential regions were identified with excess asthma mortality (RR =51.387 and 2.730), but both were not statically significant (p>0.2).

In Hispanic females one primary cluster was identified with Relative Risk of 729.465 but with a P Value of 0.059 during the year 1982 in the Waller County. Seven other regions were identified with excess mortality but none was significant. In the "Other" male population two regions of excess mortality (RR= 9.531 and 86.859) were identified during year 2000 but none were significant (P>0.2). Similarly in the Other female population the primary region with excess mortality with a Relative Risk of 20.369 but with p-Value of 0.065 during the year 1997 in Upper South Texas, Gulf Coast and Lower South Texas. It included 24 counties ranging from Goliad and Bexar. Three other regions were identified with excess mortality but none was significant (p>0.2). Figure I present a

choropleth map illustrating the clusters in Texas Public Health Regions and figures in appendix B and C illustrate the relationship of asthma clusters of MUA and TRI, respectively.

CHAPTER VI

DISCUSSION

Asthma, a multifactorial disease associated with familial, infectious, allergenic, socioeconomic, psychosocial and environmental factors, is one of the leading chronic diseases that afflict the population of United States. According to NHIS (National Health Interview Survey, 2001) age adjusted death rates for the Texas population was 1.6, which is almost same as the result we got (1.5). The age adjusted rate for black population was three times that rate of the white population in that study, here blacks had (2.7) twice that of white (1.6) population. Female death rate was 39 percent higher than males. The age-adjusted death rates for Hispanic population were 1.5 per 100,000.Age adjusted death rates in Hispanics were 63%.

Lower than non Hispanic blacks but 15 % higher than non-Hispanic whites. In our study the age adjusted rates of that Hispanics (0.5) were far less than blacks but not higher than the non-Hispanic whites (1.6). Our analyses revealed some different patterns of racial and demographic inequalities with respect to asthma. The results indicate that between 1980 and 2001, three geographic regions were identified with excess mortality rates in Texas that were statistically significant for the whole population. One geographic region was identified with excess mortality that is statistically significant for males, which included 110 counties, and the regions were High Plains, Northwest Texas, Metroplex, West Texas and part of Central Texas. One region is statistically significant

for the females, which include 186 counties in all the regions of Texas except Upper East Texas, South East Texas and part of Gulf Coast. Of all the five regions with excess mortality for the white population only one geographic region was statically significant .It covered the areas from High Plains, Northwest Texas, Metroplex, Upper Rio Grande and West Texas.

For the Black race one geographical region was statistically significant which included 110 counties in the regions of Northwest Texas, Metroplex and Central Texas. Also the black race has the longest temporal persistence for a period of 15 years. For the white male one geographic region was identified which is statistically significant in the regions of Northwest Texas, Metroplex and Upper East Texas. For the White females one cluster is identified which is statistically significant in the regions of High Plains, Northwest Texas, Metroplex, Upper Rio Grande and West Texas.

This is very similar to the mortality in the white race as a whole. For Black males, a one region is identified that a temporal persistence of 9 years in the regions of Metroplex, Upper East Texas and Central Texas. For Black females a huge cluster with excess mortality that is statistically significant is found in all areas except Upper East Texas and South East Texas. Also Black females have the longest temporal persistence for 15 years from 1985-1999. Two clusters in the Texas counties have temporal persistence to the present decade. Based on this study it reiterates the fact that females are more affected by asthma than males. The White race has higher rates of mortality than any other race in Texas. Overall the number of deaths noticed in the white race is more than the Black

race. But Black males have the have the highest relative risk of asthma mortality if we compare according to the gender.

Also TRI Data is analyzed from 1996-2001 and was noticed that the areas with asthma mortality is directly involved with the areas where there is excess TRI exposure. There is also need to set up more TRI identifying sites in Texas so that more of the associations could be identified and necessary precautions could be taken. This study would be very effective in understanding and planning the measures to be taken effectively so as to reduce the asthma mortality not only in Texas; the same measures could then as well be tried in the whole country. The results of this analysis measures disease burden over time by both the spatial concentration (p values and relative risks) and temporal persistence (by duration of detected clusters), allowing us to quantify health disparity in a different perspective.

In the next stage of the study is the overlaying of the maps of asthma mortality with the maps of the TRI facilities in The Texas counties. For the Black population, the most likely area of excess mortality (RR=1.304, p=0.001) occurred between years 1985 and 1999 in Northwest Texas, Metroplex and Central Texas. These included 110 counties ranging from Bosque to Lavaca.80 percent of these counties are in the TRI reporting counties and 20 of those counties are in the grade 4 and 5 of the TRI emissions. This shows that African Americans live more in the areas in and around the emission sites and are more vulnerable to the toxic emissions. For the Black males, the primary excess mortality region was identified with a Relative Risk (RR) of 1.444 (p=0.008). This occurred between 1990-1999 in Northwest Texas, Metroplex, Upper East Texas and

Central Texas, which included 110 counties ranging from Bosque to Subjacent. Of these 82 percent of the counties come under the TRI surveillance and 35 of them in the serious toxic release zone Grade 4 and 5). In Black females, the primary excess mortality was identified with a Relative Risk (RR) of 1.322 (p=0.001) between the years 1985-1999 in Upper Rio Grande, West Texas, Northwest Texas, Metroplex, Central Texas Upper South Texas and Lower South Texas. These included 183 counties ranging from Terrell to Waller .58 percent of the counties come under TRI reporting counties and, of them, 30 counties are in the 4 and 5 grade toxic emission. For the whole population in Texas, a secondary excess mortality region (RR= 1.495, p=0.001) was identified between years 1988-2001 in the 23 counties of Gulf Coast and upper South Texas. Of these 20 counties are in TRI reporting counties and five counties are in the grade and 5 toxic releasing counties. That is more than 80 percent of the counties in the cluster come under the high TRI producing counties (grade 4 and 5). For the non-Hispanic White population, five potential excess mortality regions were identified. The most likely area of excess mortality with a Relative Risk (RR) of 1.351 (p=0.001) occurred between 1989 and 1998 in High plains along Northwest Texas and Metroplex, West Texas and Upper Rio Grande. These included 151 counties ranging from Swisher to Bandera counties. Of these 62 percent are in the TRI reporting counties. A secondary excess mortality region (RR=2.415, p=0.014) was identified between 1995 and 1999 in the 12 counties of Lower South Texas. Of these 10 counties are in TRI reporting counties and 3 are in the grade 5 counties. Another secondary potential excess area in the non-Hispanic White population was detected in 5 counties during the years 1990-1998 in Southeast Texas along Gulf

Coast that was statistically significant (RR=1.785, p=0.019) .Of these all 5 are in the TRI reporting counties. For the white males, six potential excess mortality regions were identified .The most likely area of excess mortality with Relative Risk (RR) of 1.411 (p=0.001), occurred between 1991-1998 in High Plains, Northwest Texas, Metroplex and Upper East Texas. These include 119 counties ranging from Clay to Bowie .Of this 74 percent of are in the TRI reporting counties and of these 25 counties are in the high emitters. For the White males, six potential excess mortality regions were identified .The most likely area of excess mortality with Relative Risk (RR) of 1.411 (p=0.001), occurred between 1991-1998 in High Plains, Northwest Texas, Metroplex and Upper East Texas. These include 119 counties ranging from Clay to Bowie. Of this 74 percent of them are in the TRI reporting counties and of these 25 counties are in the high emitters. For the White females, the primary excess mortality region was identified with a relative risk (RR) of 1.398 (p=0.001) during the years 1989-1998 in High Plains, North West Texas, West Texas and Upper Rio Grande. These included 141 counties ranging from Terry to Williamson. A secondary excess mortality was identified with a Relative Risk (RR) of 2.462 (p= 0.003) in Lower South Texas in 17 counties ranging from Kleberg to Starr. Both these clusters of the white females were statistically significant. 68 percent of the primary clusters come under the TRI counties and 30 of them are in high emitting regions.

Also, Medically Underserved Areas (MUA) counties data were gathered from the TDH Health Professional Resources Center (Thomas, 2001). MUA is an official designation for those counties having shortage of personal health services according to

the U.S department of Health and Human Services rules. The criteria for MUA designation include: 1) percentage of elderly population (over 65 years); 2) poverty rate; 3)infant mortality rate; 4)ratio of primary care physicians per 1000 population (TDH). Both the MUA and TRI reporting county layers were overlaid on the asthma mortality clusters layer to examine their potential associations. It was very clear that more asthma deaths are seen in the areas in Non-MUA counties. So by improving the medical facilities in these areas, which would eventually identify asthma cases at an earlier stage, we could effectively reduce the mortality.

With respect to suspected excess mortality, the regions detected with excess asthma mortality were consistent with those presented in the analyses of the Texas Department of Health, Bureau of Vital Statistics and the data reported by Texas State Data Center, Texas Population Estimates and Projections Programs. The results rendered supporting evidence that most counties that were previously suspected of having elevated asthma mortality do, indeed, have excessive mortality. The relative risk of this cluster was at the modest level of 1.18. Nevertheless, this region had the highest in relative risk, with the longest temporal persistence among detected potential clusters of all racial groups in this study. Based on this finding and on the comparisons of LLRs for the primary suspected clusters from both scan trials, it was determined that the Black female populations in the regions detected with clusters had the highest burden of asthma mortality, as evidenced by both temporal persistence and spatial concentration.

The verification of asthma excess mortality over time may prove beneficial for health policy and planning. Spatiotemporal analysis such as that described in this study will be

instrumental in planning and reaching the projected objective. For instance, the present analysis underscored the two regions with multiple racial groups that bear the persistent burden of asthma mortality, and detected a potential 15-year persistence of excess asthma with the highest relative risk in the black population. The results of spatiotemporal analysis quantified disease burden over time by both spatial concentration (as determined by p values, LLRs and relative risks) and temporal persistence (as determined by the duration of detected clusters), which presented another perspective of measuring health disparity. It contributed to an understanding of the persistent burden of the disease across space and time, as well as aiding in determining whether the mortality burden that may have persisted into the current decade. Compared with previous studies using Sat Scan for cluster detection, (Kulldorff et al., 1995) the relative risks revealed in the present study were apparently higher and no localized, hot spot clusters (with constant, high risks in the clusters) that persisted over time were detected. Nevertheless, this study offered baseline descriptions of persistently elevated asthma deaths in Texas, which may serve as a point of departure for policy deliberation and health resource allocation. Second, although this study focused primarily on statistically significant excess mortality, it by no means suggested that those non-statistically significant regions of excess mortality were less important. To be statistically significant at the 0.05 or 0.01 levels, outcome measures had to satisfy the Poisson distribution model and all independent variables of this study, including space, time and age, had to fit into the model simultaneously, and produce a large LLR as a result of spatial-temporal analysis. For example, the potential cluster detected among Blacks between 1991-1996 in Gulf Coast Texas (RR=1.15,p=0.12) was

for all age groups. However, the results may become statistically significant if analysis was conducted with the stratification of certain age groups, such as among Black females aged 25 to 40. Therefore, the p-value derived is construed as an indicator, suggesting the level of excess mortality that calls for further investigation. Third, the choice of county level analysis entailed the strengths and weaknesses intrinsic to this level of aggregation. Although sub-county level (such as census tracts or block groups) of analysis may be preferred in asthma mortality analysis, we chose the county-level data because this level of aggregation was used in other studies on detecting breast cancer clusters, (Kulldorff et al, 1997) and also because the disproportional demographic distribution of Texas population made sub-county level analysis less feasible. For example, there were seven border counties that averaged fewer than ten asthma deaths, and had a population of less than 900 residents during the study period. The rates based on these small numbers of events and small population sizes tend to be unpredictable and often inflated. The above findings are illustrated in the maps in appendix A from figure 1 to figure 16 and in appendix D from figures 17 to 25.The following tables from 1 to 4 gives a detailed description of all the asthma deaths in different counties in Texas.

Table 1: Asthma mortality in population of Texas and in males (Primary cluster).

Asthma Mortality	Cases	Annual Age –adjusted rates (per 100,000)	Year	Relative Risk of excess mortality	Total count ies	P value
Whole population (Primary cluster)	1753	1.9	1989-1998	1.310	162	0.001
1st Secondary cluster	311	2.2	1988-2001	1.495	23	0.001
2nd Secondary cluster	317	2.0	1990-2000	1.365	20	0.007
All Males (Primary cluster)	576	1.6	1991-1998	1.357	110	0.001

Table 2: Asthma mortality in males (secondary cluster), females and in Whites in Texas counties.

ASTHMA MORTALITY	NO. OF CASES	Annual Age – adjusted rates (per 100,000)	Year	Relative Risk of excess mortality	P value	Total counties
All Males (secondary cluster)	8	21.5	1989-1993	18.74	0.004	1
All Females (Primary cluster)	1175	2.3	1989-1999	1.309	0.001	186
All Females (Secondary cluster)	77	2.9	1989-1998	1.621	0.905	3
White race (Primary cluster)	1200	2.2	1989-1998	1.351	0.001	151
1st Secondary cluster	46	3.9	1995-1999	2.415	0.014	12
2nd Secondary cluster	94	2.9	1990-1998	1.785	0.019	5

Table 3: Asthma mortality in white males, white females, Blacks, Black males and Black females in Texas counties.

ASTHMA MORTALITY	NO. OF CASES	Annual Age – adjusted rates (per 100,000)	Year	Relative Risk of excess mortality	P value	Total count ies
White male (Primary cluster)	358	1.7	1991-1998	1.411	0.001	119
Secondary cluster	15	5.5	1990-1995	4.628	0.143	4
White female (Primary cluster)	716	2.9	1989-1998	1.398	0.001	141
Secondary cluster	49	5.0	1995-1999	2.462	0.003	17
Black race(Primary cluster)	526	3.6	1985-1999	1.304	0.001	110
Black male (Primary cluster)	177	3.6	1990-1999	1.444	0.008	110
Black female(Primary cluster)	299	3.9	1985-1999	1.322	0.001	183

Table 4: Asthma mortality in Hispanics, Hispanic males, Hispanic females and in other races in Texas counties.

ASTHMA MORTALITY	NO. OF CASES	Annual Age – adjusted rates (per 100,000)	Year	Relative Risk of excess mortality	P value	count ies
Hispanic race (Primary cluster)	4	20.5	1989-1997	37.464	0.138	1
Hispanic male (Primary cluster)	28	1.3	1993-1995	2.730	0.058	29
Hispanic female (Primary cluster)	2	458.7	1982-1982	729.465	0.059	1
Other Races (Primary cluster)	47	1.8	1987-1999	1.768	0.103	38
Other races female (Primary cluster)	5	20.0	1997-1997	20.369	0.065	24

CHAPTER VII

CONCLUSION

For the Black males, the primary excess mortality region was identified with a Relative Risk (RR) of1.444 (p=0.008). This occurred between 1990-1999 in Northwest Texas, Metroplex, Upper East Texas and Central Texas, which included 110 counties ranging from Bosque to Subjacent. Of these, 82 percent of the counties come under the TRI surveillance and 35 of them in the serious toxic release zone; we could say that there might be an association between the TRI release and the asthma mortality in the Black male population. The possible reasons for this may be Black males are more prone to the toxic releases or they may be working in and around those facilities more than other races or living in close proximity to the facilities. The reason for this conclusion is the Black race as such had high mortality occurring in the areas of the facilities is for whole of the Black population, the most likely area of excess mortality 80 percent of these counties are in the TRI reporting counties and 20 of those counties are in the grade 4 and 5 of the TRI emissions. But at the same time in black females, only 58 percent of the counties come under TRI reporting counties and of them 30 counties are in the 4 and 5 grade toxic emissions. It calls for an increased effort and investment in solidifying the toxic reporting systems in other counties. These results provide ample evidence that there might be an association between asthma mortality and toxic release and therefore warrant increased research in this area and need a better TRI reporting data in other counties .The primary

cluster of asthma mortality is also seen in other areas where there are no TRI reporting data. So by installing the TRI service stations in the remaining counties, we could get a more satisfying explanation about the relation and probably could intervene with policy changes. These recommendations might reduce the asthma mortality in these counties and provide a better future.

Limitations

Most of the clusters for Hispanics and other race communities had fewer deaths per county or even if there was high relative risk but was not statistically significant. Some counties have less than 1000 residents in the study years. The rates are based on small numbers of events and population tends to be unpredictable and often inflated. Readers are therefore advised to be careful when interpreting health outcomes in these sparsely populated counties. Several limitations are worth noting in this study. The toxic release data were based on those counties with routine TRI surveillance reports. The data include 136 counties in Texas, which captures only half of all the Texas counties.

APPENDIX

APPENDIX A

ASTHMA MORTALITY MAPS IN TEXAS COUNTIES BY GEOGRAPHIC INFORMATION SYSTEMS

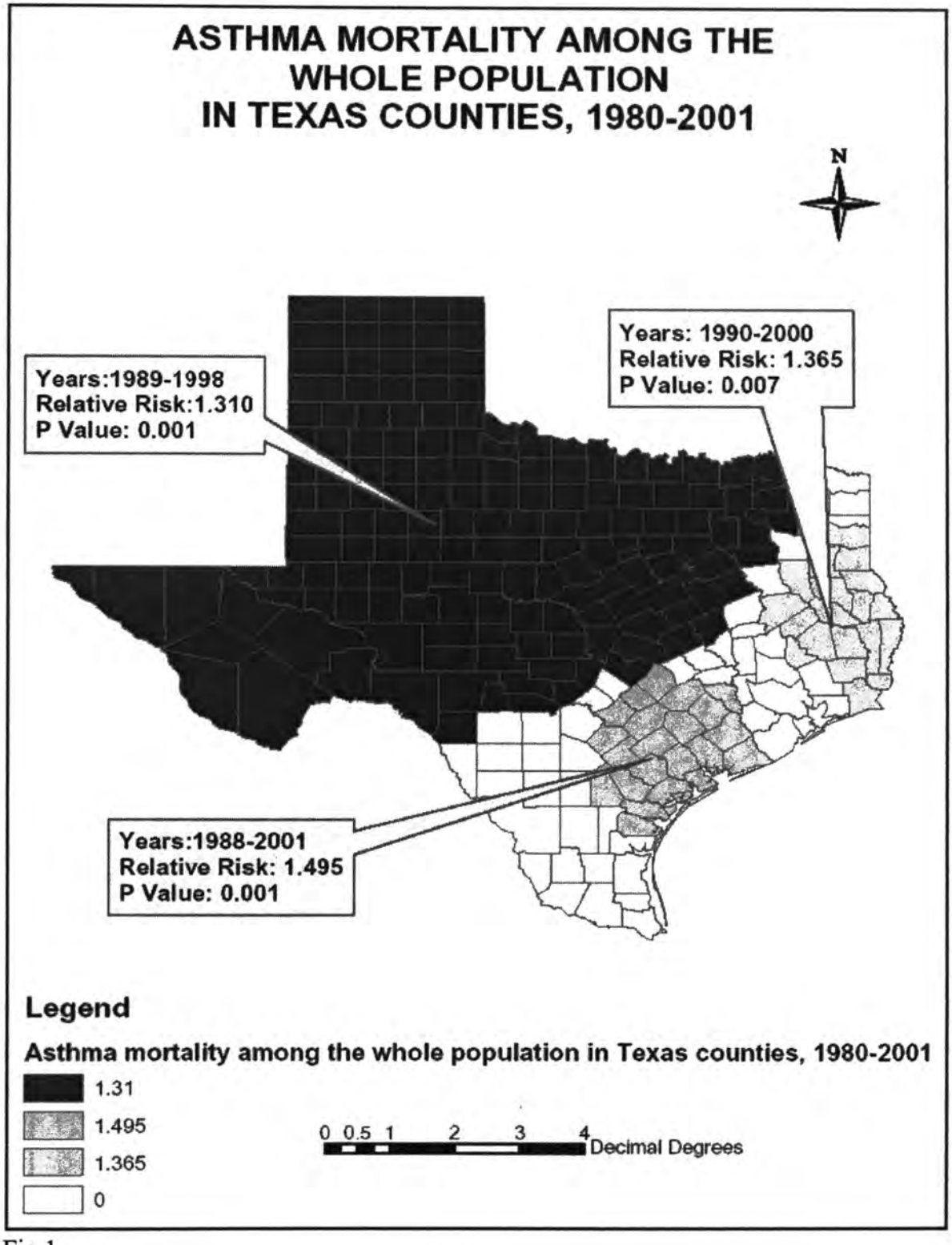

ASTHMA MORTALITY AMONG THE WHOLE POPULATION IN TEXAS COUNTIES, 1980-2001
N
Years:1989-1998
Relative Risk:1.310
P Value: 0.001
Years: 1990-2000
Relative Risk: 1.365
P Value: 0.007
Years:1988-2001
Relative Risk: 1.495
P Value: 0.001
Legend
Asthma mortality among the whole population in Texas counties, 1980-2001
1.31
1.495
1.365
0
0 0.5 1 2 3 4
Decimal Degrees

Fig 1

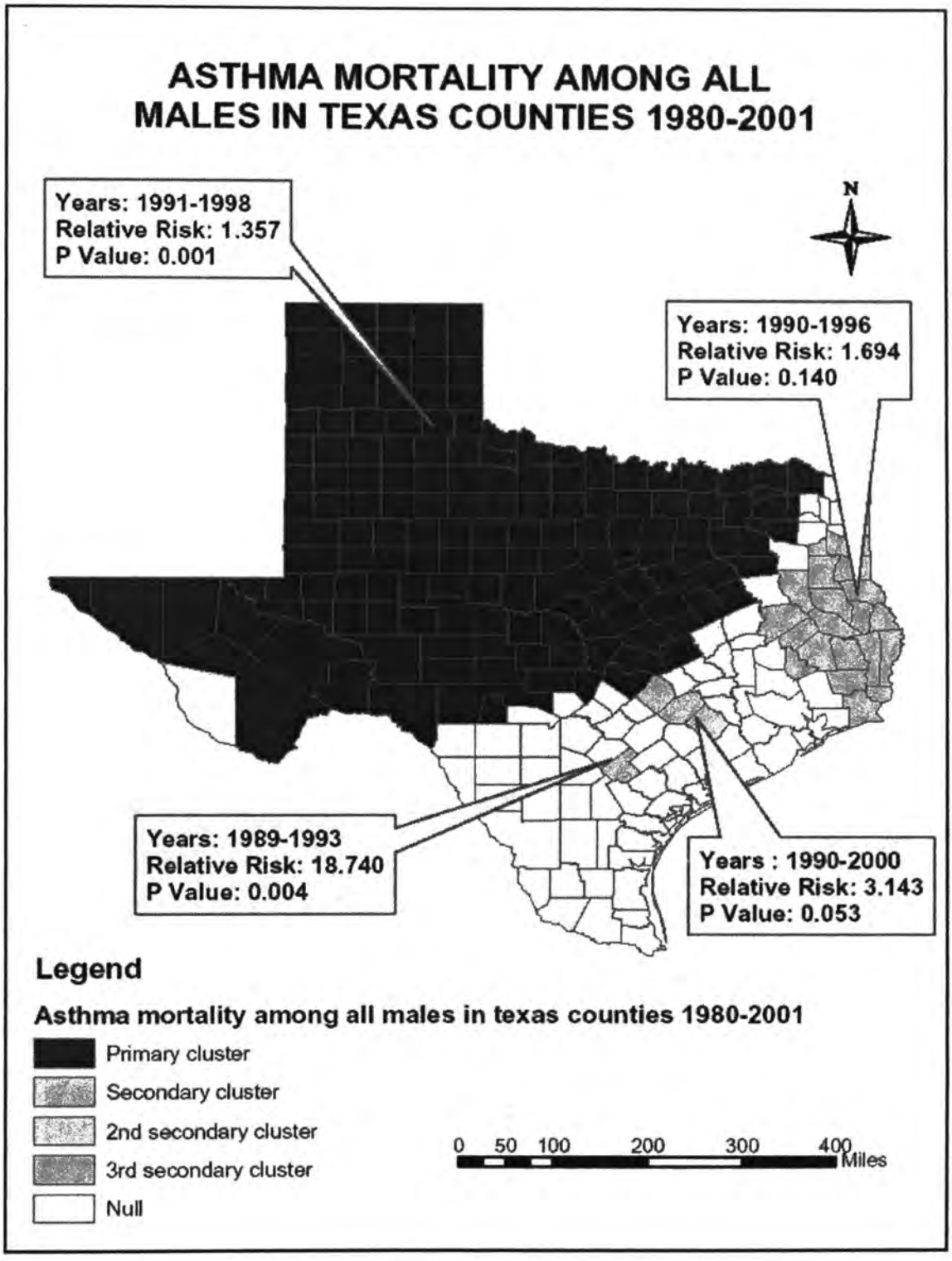

ASTHMA MORTALITY AMONG ALL MALES IN TEXAS COUNTIES 1980-2001
Years: 1991-1998
Relative Risk: 1.357
P Value: 0.001
N
Years: 1990-1996
Relative Risk: 1.694
P Value: 0.140
Years: 1989-1993
Relative Risk: 18.740
P Value: 0.004
Years : 1990-2000
Relative Risk: 3.143
P Value: 0.053
Legend
Asthma mortality among all males in texas counties 1980-2001
Primary cluster
Secondary cluster
2nd secondary cluster
3rd secondary cluster
Null
0 50 100 200 300 400 Miles

Fig 2

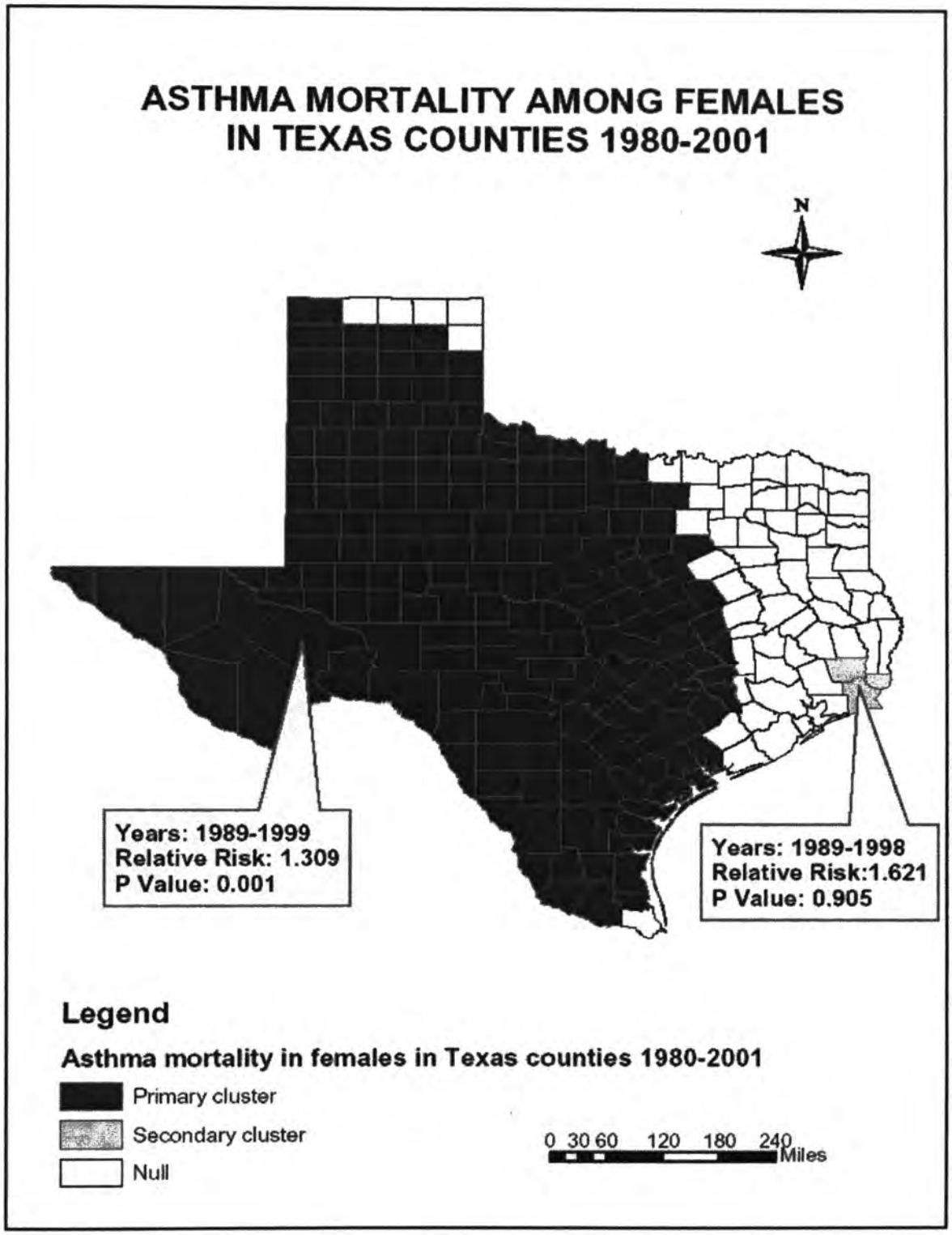

ASTHMA MORTALITY AMONG FEMALES
IN TEXAS COUNTIES 1980-2001
N
Years: 1989-1999
Relative Risk: 1.309
P Value: 0.001
Years: 1989-1998
Relative Risk:1.621
P Value: 0.905
Legend
Asthma mortality in females in Texas counties 1980-2001
Primary cluster
Secondary cluster
Null
0 30 60 120 180 240
Miles

Fig 3

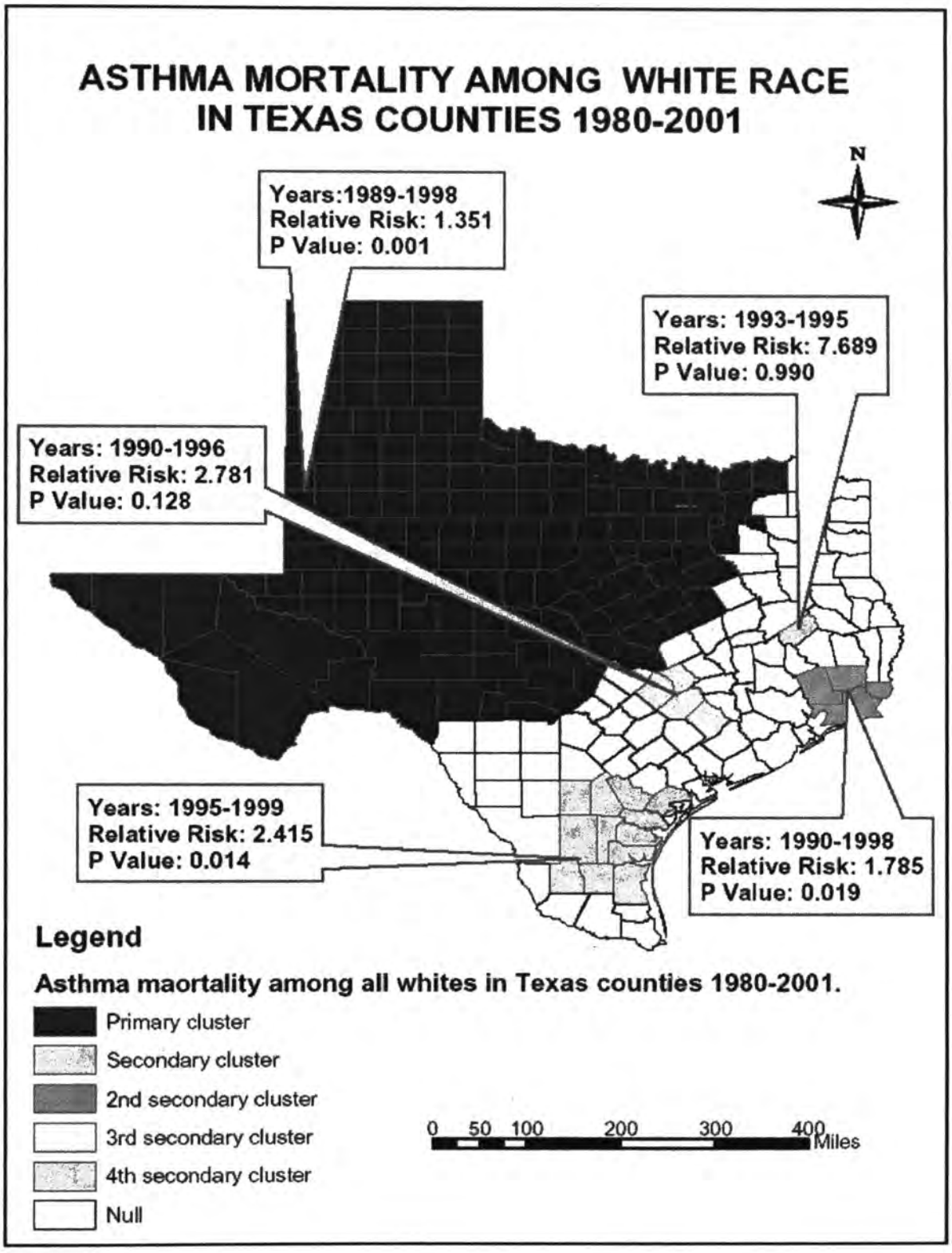

ASTHMA MORTALITY AMONG WHITE RACE
IN TEXAS COUNTIES 1980-2001
N
Years:1989-1998
Relative Risk: 1.351
P Value: 0.001
Years: 1993-1995
Relative Risk: 7.689
P Value: 0.990
Years: 1990-1996
Relative Risk: 2.781
P Value: 0.128
Years: 1995-1999
Relative Risk: 2.415
P Value: 0.014
Years: 1990-1998
Relative Risk: 1.785
P Value: 0.019
Legend
Asthma maortality among all whites in Texas counties 1980-2001.
Primary cluster
Secondary cluster
2nd secondary cluster
3rd secondary cluster
4th secondary cluster
Null
0 50 100 200 300 400 Miles

Fig 4

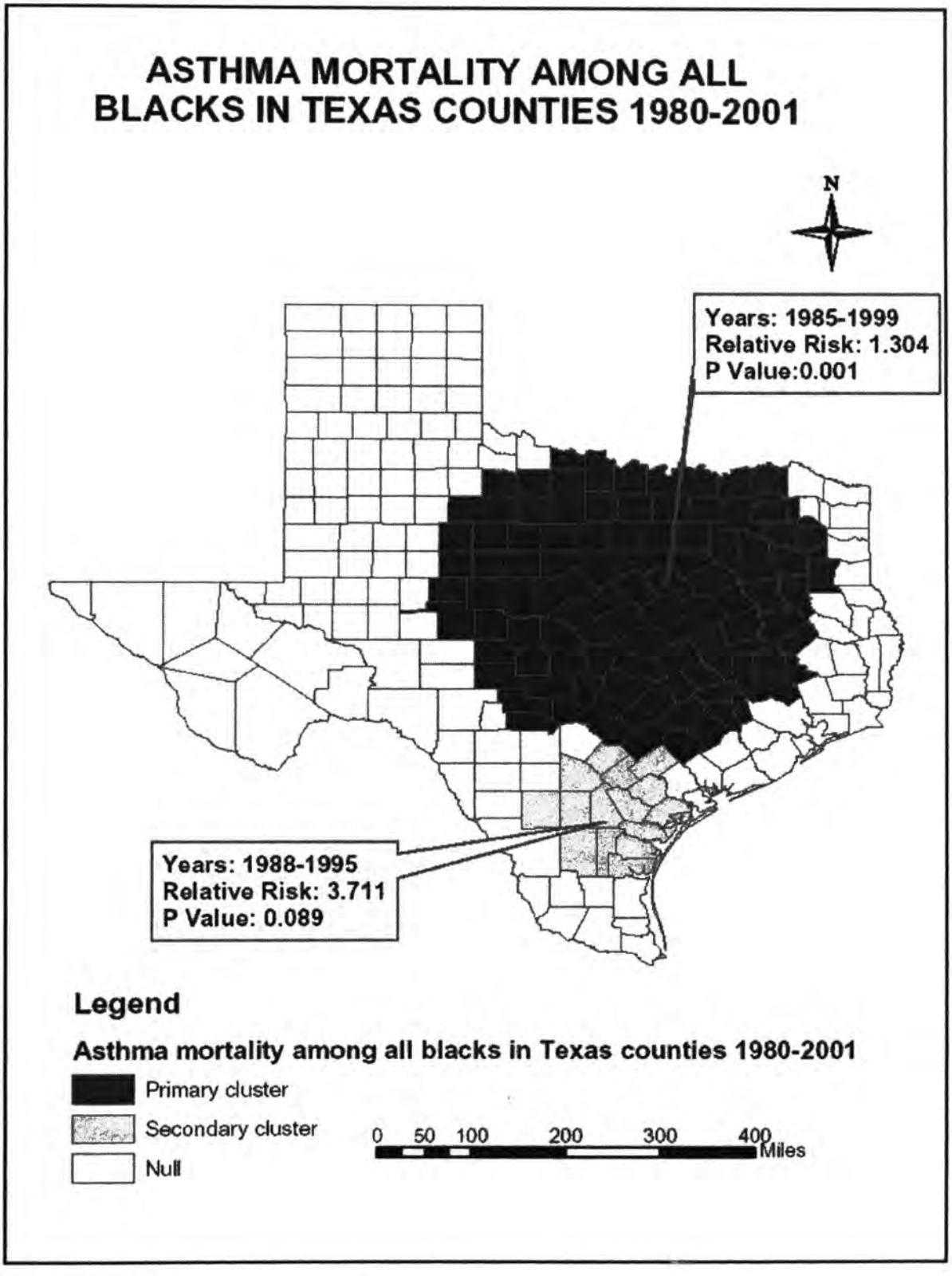

ASTHMA MORTALITY AMONG ALL BLACKS IN TEXAS COUNTIES 1980-2001
N
Years: 1985-1999
Relative Risk: 1.304
P Value:0.001
Years: 1988-1995
Relative Risk: 3.711
P Value: 0.089
Legend
Asthma mortality among all blacks in Texas counties 1980-2001
Primary cluster
Secondary cluster
Null
0 50 100 200 300 400
Miles

Fig 5

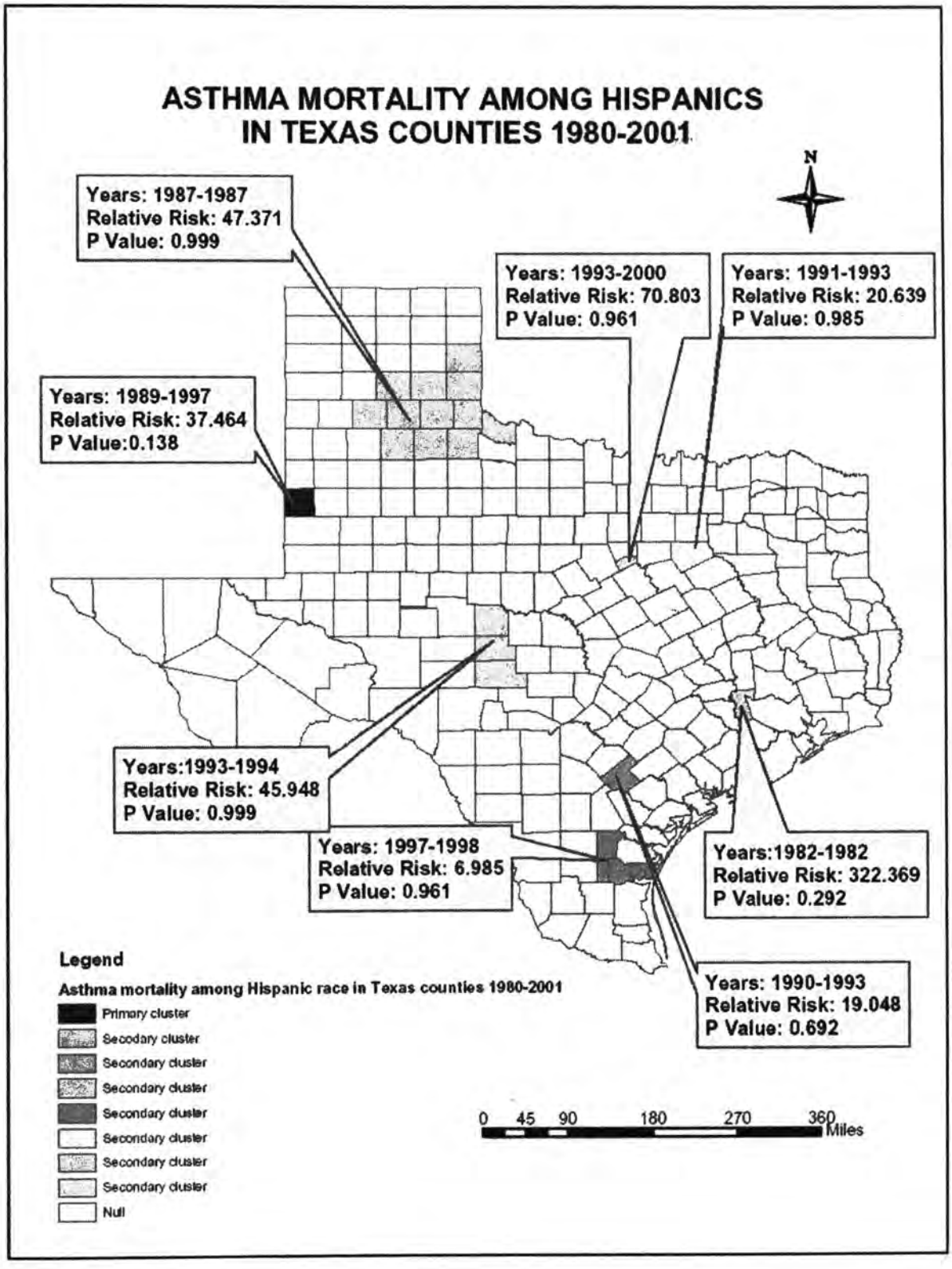

ASTHMA MORTALITY AMONG HISPANICS
IN TEXAS COUNTIES 1980-2001
N
Years: 1987-1987
Relative Risk: 47.371
P Value: 0.999
Years: 1993-2000
Relative Risk: 70.803
P Value: 0.961
Years: 1991-1993
Relative Risk: 20.639
P Value: 0.985
Years: 1989-1997
Relative Risk: 37.464
P Value:0.138
Years:1993-1994
Relative Risk: 45.948
P Value: 0.999
Years: 1997-1998
Relative Risk: 6.985
P Value: 0.961
Years:1982-1982
Relative Risk: 322.369
P Value: 0.292
Years: 1990-1993
Relative Risk: 19.048
P Value: 0.692
Legend
Asthma mortality among Hispanic race in Texas counties 1980-2001
Primary cluster
Secodary cluster
Secondary cluster
Secondary cluster
Secondary cluster
Secondary cluster
Secondary cluster
Secondary cluster
Null
0 45 90 180 270 360
Miles

Fig 6

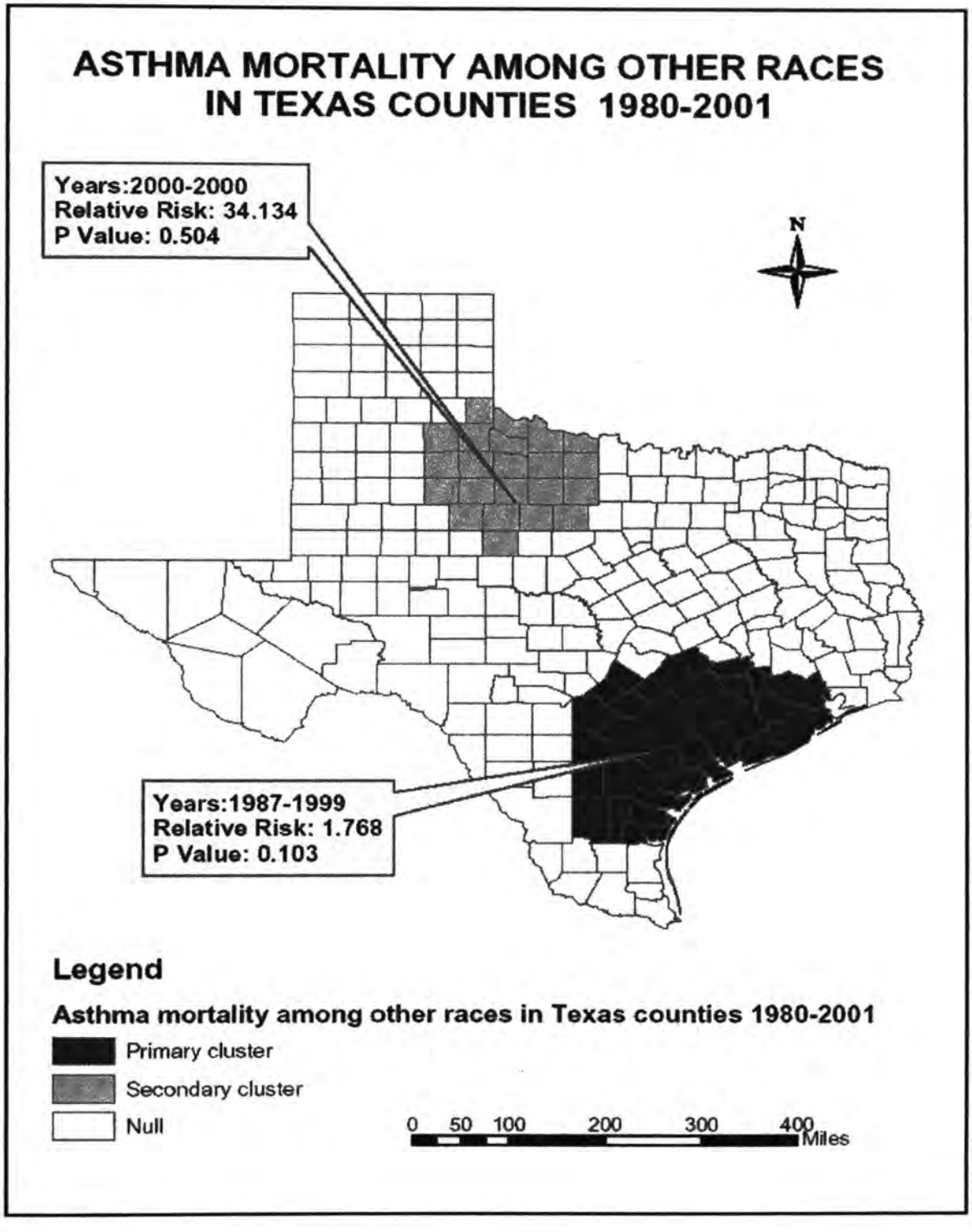

ASTHMA MORTALITY AMONG OTHER RACES
IN TEXAS COUNTIES 1980-2001
Years:2000-2000
Relative Risk: 34.134
P Value: 0.504
N
Years:1987-1999
Relative Risk: 1.768
P Value: 0.103
Legend
Asthma mortality among other races in Texas counties 1980-2001
Primary cluster
Secondary cluster
Null
0 50 100 200 300 400
Miles

Fig 7

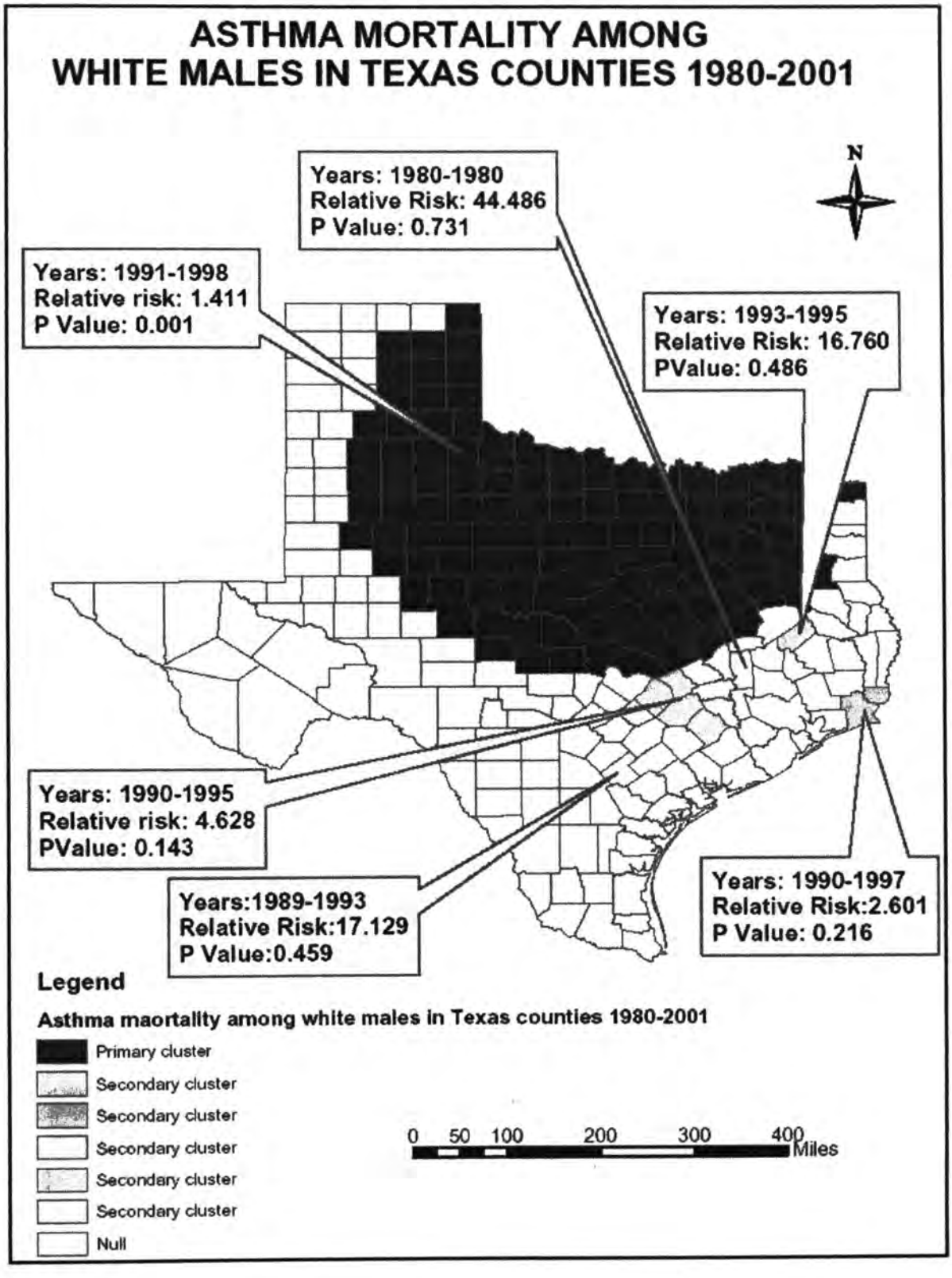

ASTHMA MORTALITY AMONG
WHITE MALES IN TEXAS COUNTIES 1980-2001
N
Years: 1980-1980
Relative Risk: 44.486
P Value: 0.731
Years: 1991-1998
Relative risk: 1.411
P Value: 0.001
Years: 1993-1995
Relative Risk: 16.760
PValue: 0.486
Years: 1990-1995
Relative risk: 4.628
PValue: 0.143
Years:1989-1993
Relative Risk:17.129
P Value:0.459
Years: 1990-1997
Relative Risk:2.601
P Value: 0.216
Legend
Asthma maortality among white males in Texas counties 1980-2001
Primary cluster
Secondary cluster
Secondary cluster
Secondary cluster
Secondary cluster
Secondary cluster
Null
0 50 100 200 300 400
Miles

Fig 8

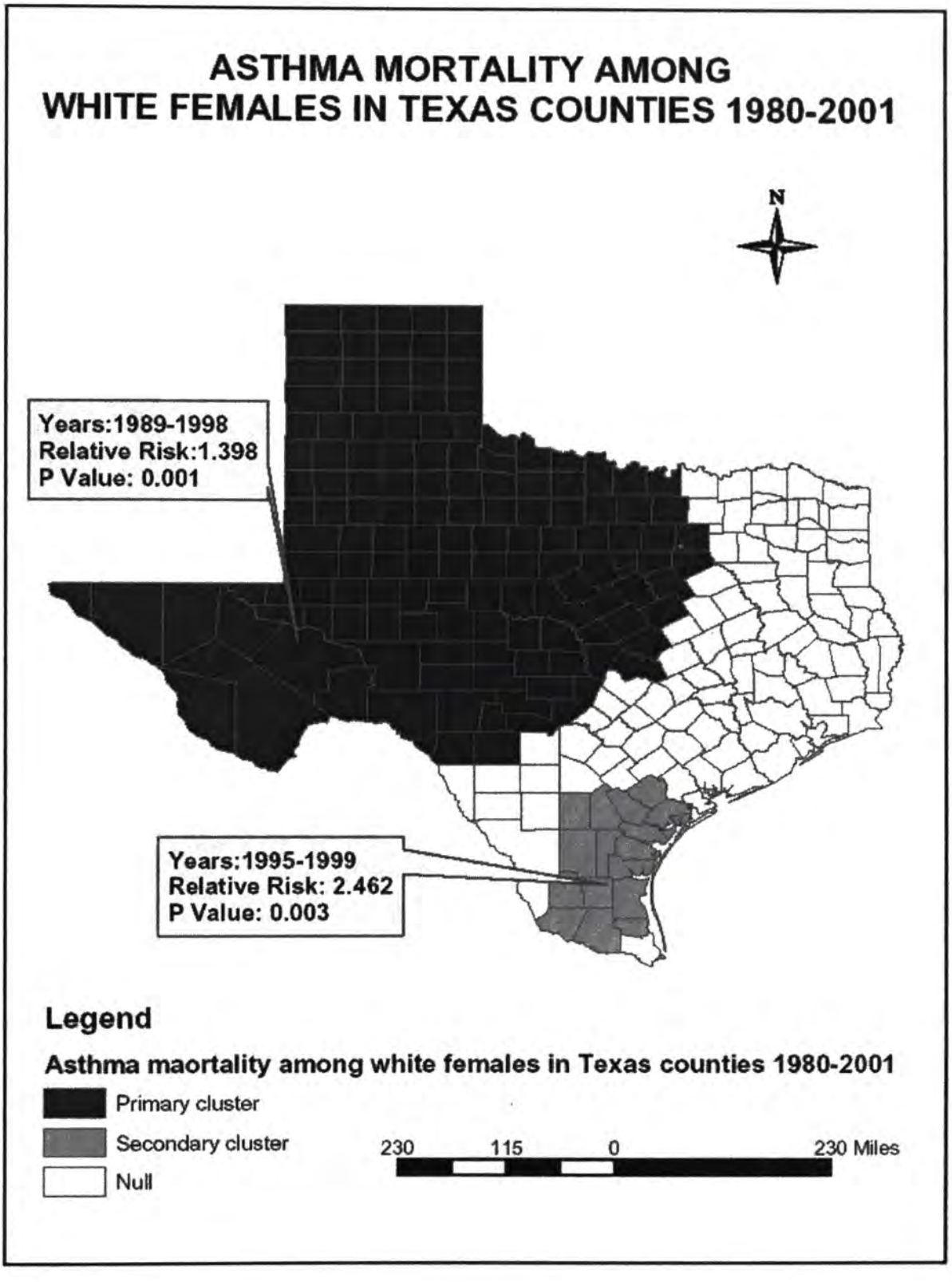

ASTHMA MORTALITY AMONG
WHITE FEMALES IN TEXAS COUNTIES 1980-2001
N
Years:1989-1998
Relative Risk:1.398
P Value: 0.001
Years:1995-1999
Relative Risk: 2.462
P Value: 0.003
Legend
Asthma maortality among white females in Texas counties 1980-2001
Primary cluster
Secondary cluster
Null
230
115
0
230 Miles

Fig 9

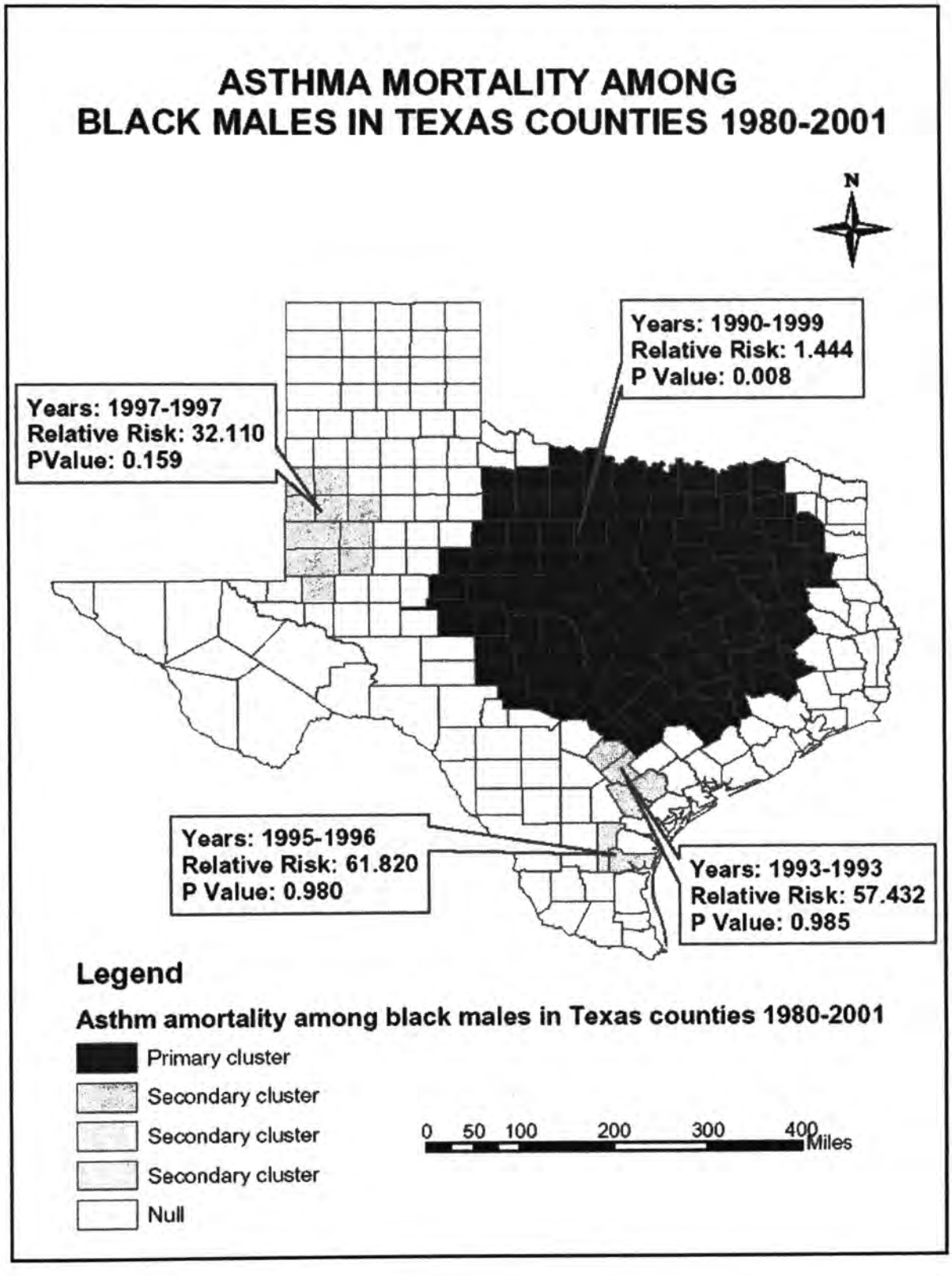

ASTHMA MORTALITY AMONG
BLACK MALES IN TEXAS COUNTIES 1980-2001
N
Years: 1990-1999
Relative Risk: 1.444
P Value: 0.008
Years: 1997-1997
Relative Risk: 32.110
PValue: 0.159
Years: 1995-1996
Relative Risk: 61.820
P Value: 0.980
Years: 1993-1993
Relative Risk: 57.432
P Value: 0.985
Legend
Asthm amortality among black males in Texas counties 1980-2001
Primary cluster
Secondary cluster
Secondary cluster
Secondary cluster
Null
0 50 100 200 300 400
Miles

Fig 10

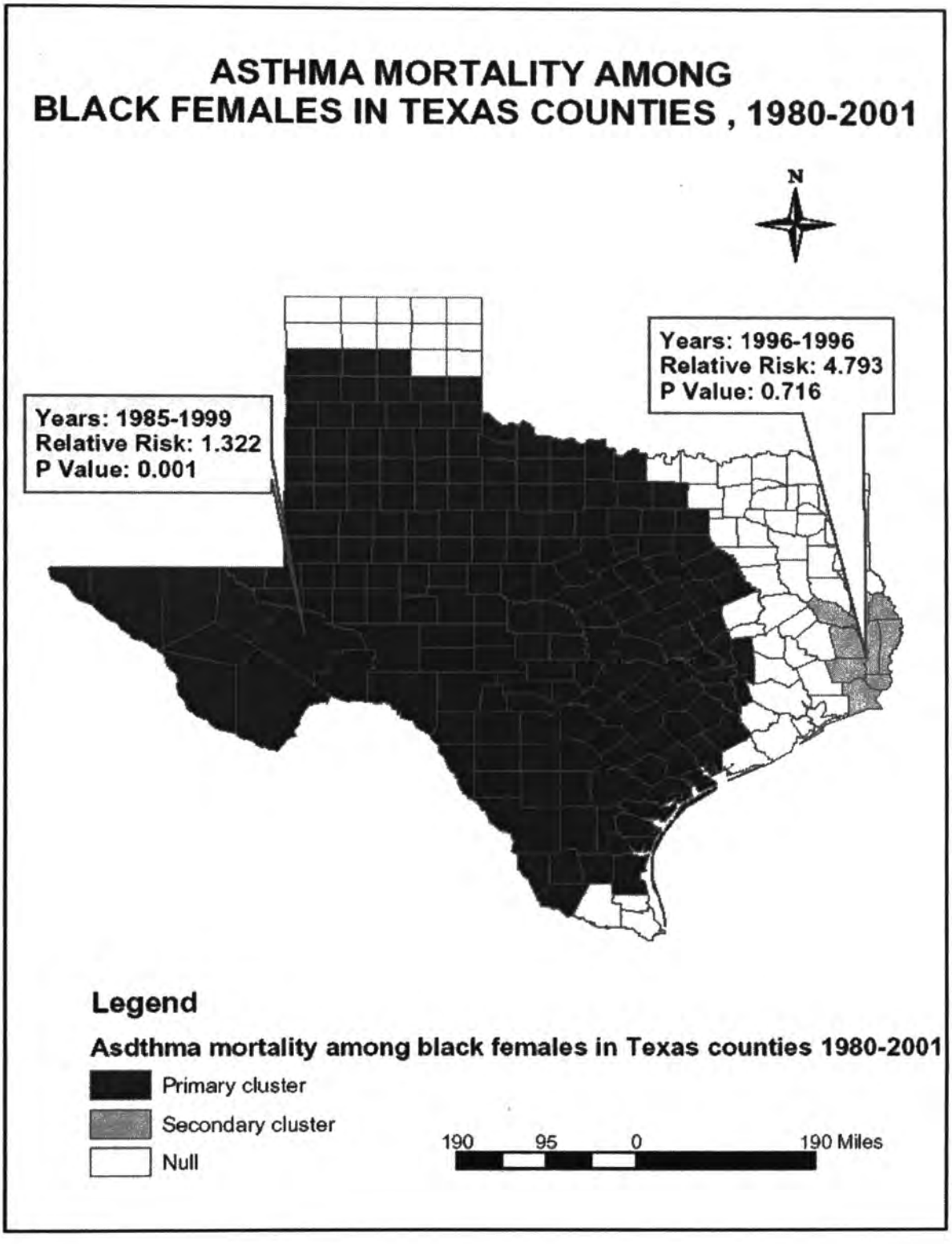

ASTHMA MORTALITY AMONG
BLACK FEMALES IN TEXAS COUNTIES , 1980-2001
N
Years: 1996-1996
Relative Risk: 4.793
P Value: 0.716
Years: 1985-1999
Relative Risk: 1.322
P Value: 0.001
Legend
Asdthma mortality among black females in Texas counties 1980-2001
Primary cluster
Secondary cluster
Null
190 95 0 190 Miles

Fig 11

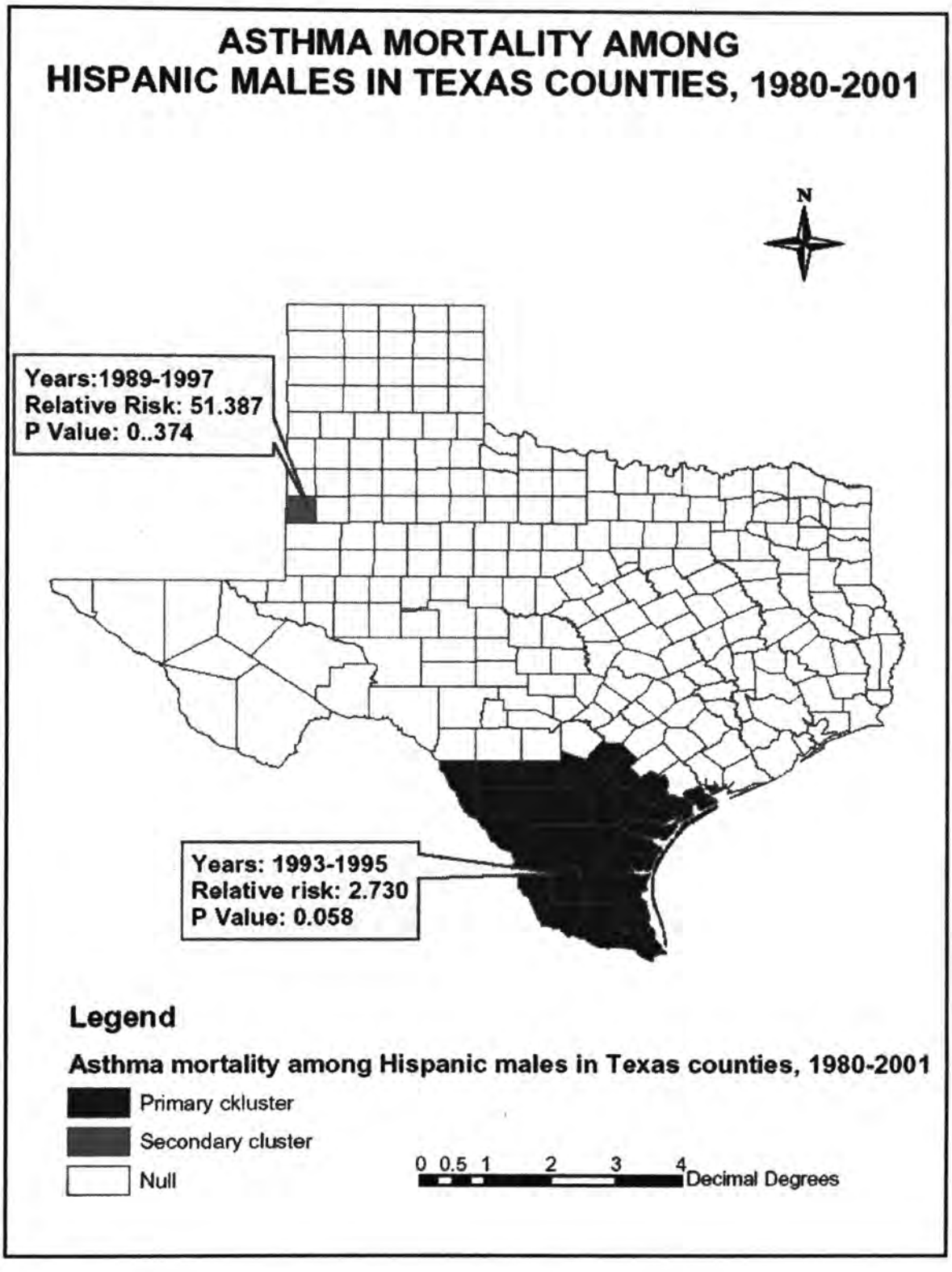

ASTHMA MORTALITY AMONG
HISPANIC MALES IN TEXAS COUNTIES, 1980-2001
N
Years:1989-1997
Relative Risk: 51.387
P Value: 0..374
Years: 1993-1995
Relative risk: 2.730
P Value: 0.058
Legend
Asthma mortality among Hispanic males in Texas counties, 1980-2001
Primary ckluster
Secondary cluster
Null
0 0.5 1 2 3 4
Decimal Degrees

Fig 12

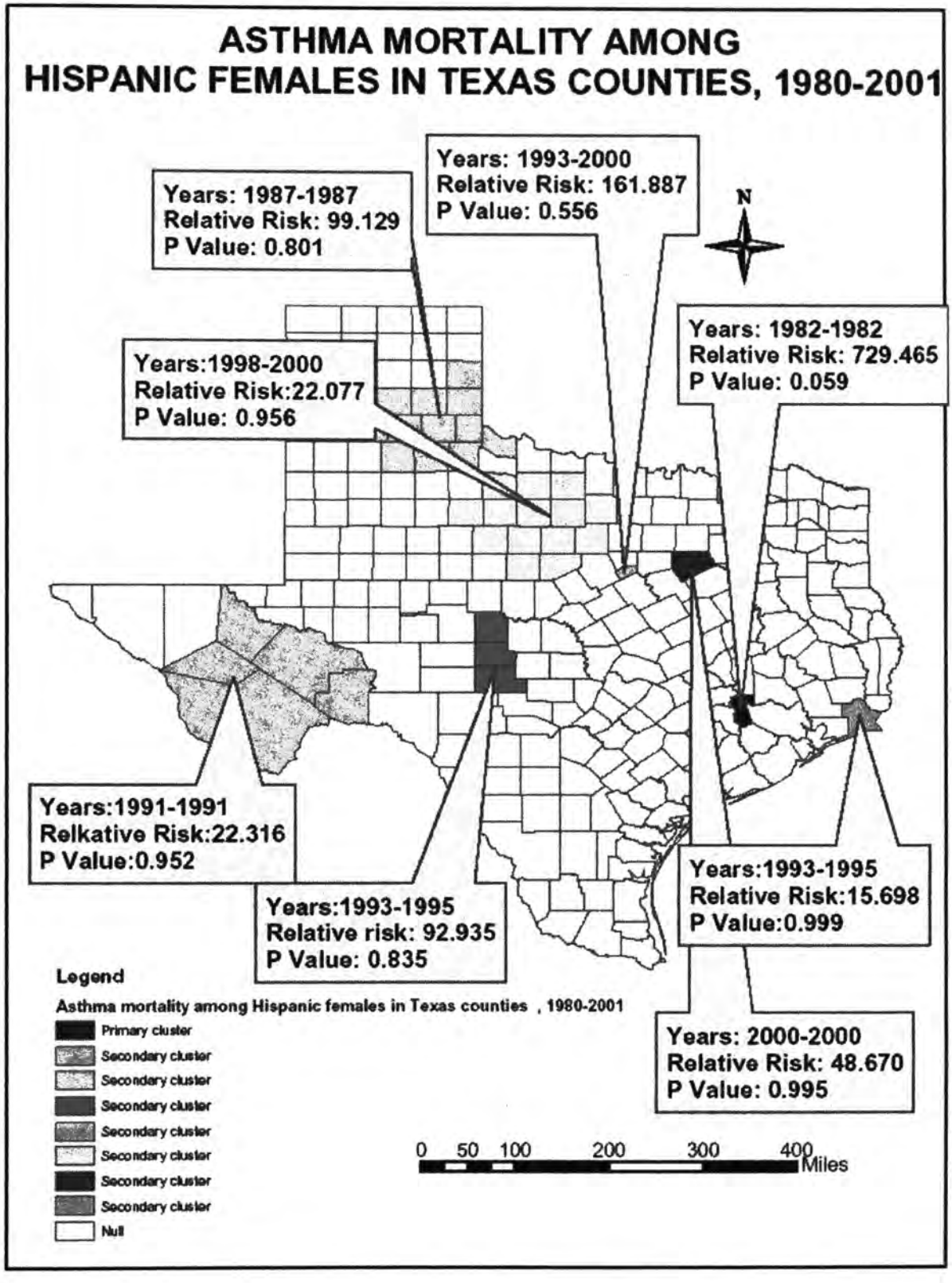

ASTHMA MORTALITY AMONG
HISPANIC FEMALES IN TEXAS COUNTIES, 1980-2001
Years: 1987-1987
Relative Risk: 99.129
P Value: 0.801
Years: 1993-2000
Relative Risk: 161.887
P Value: 0.556
N
Years: 1982-1982
Relative Risk: 729.465
P Value: 0.059
Years:1998-2000
Relative Risk:22.077
P Value: 0.956
Years:1991-1991
Relkative Risk:22.316
P Value:0.952
Years:1993-1995
Relative risk: 92.935
P Value: 0.835
Years:1993-1995
Relative Risk:15.698
P Value:0.999
Years: 2000-2000
Relative Risk: 48.670
P Value: 0.995
Legend
Asthma mortality among Hispanic females in Texas counties , 1980-2001
Primary cluster
Secondary cluster
Secondary cluster
Secondary cluster
Secondary cluster
Secondary cluster
Secondary cluster
Secondary cluster
Null
0 50 100 200 300 400
Miles

Fig 13

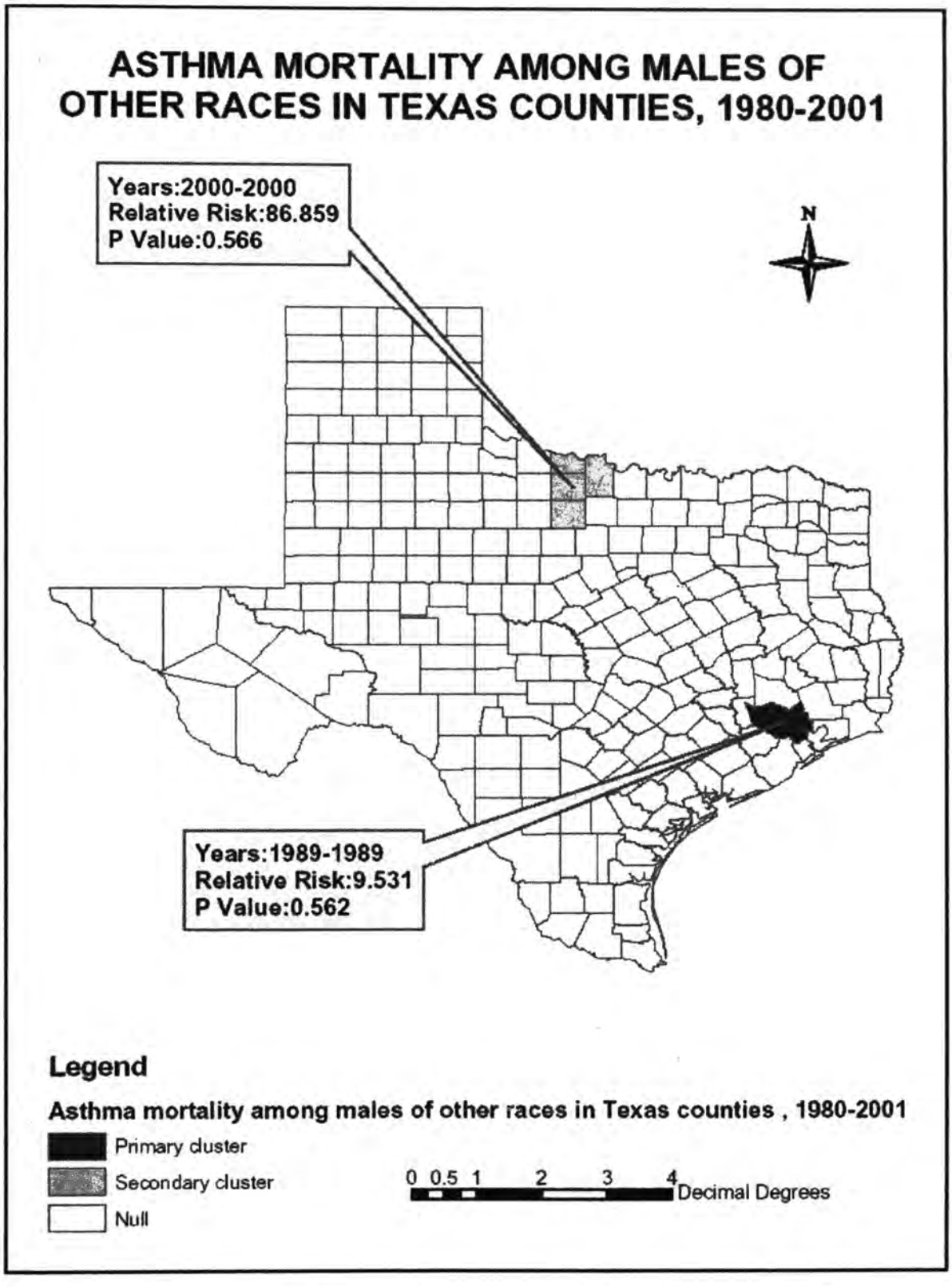

ASTHMA MORTALITY AMONG MALES OF OTHER RACES IN TEXAS COUNTIES, 1980-2001
Years:2000-2000
Relative Risk:86.859
P Value:0.566
N
Years:1989-1989
Relative Risk:9.531
P Value:0.562
Legend
Asthma mortality among males of other races in Texas counties , 1980-2001
Primary cluster
Secondary cluster
Null
0 0.5 1 2 3 4
Decimal Degrees

Fig 14

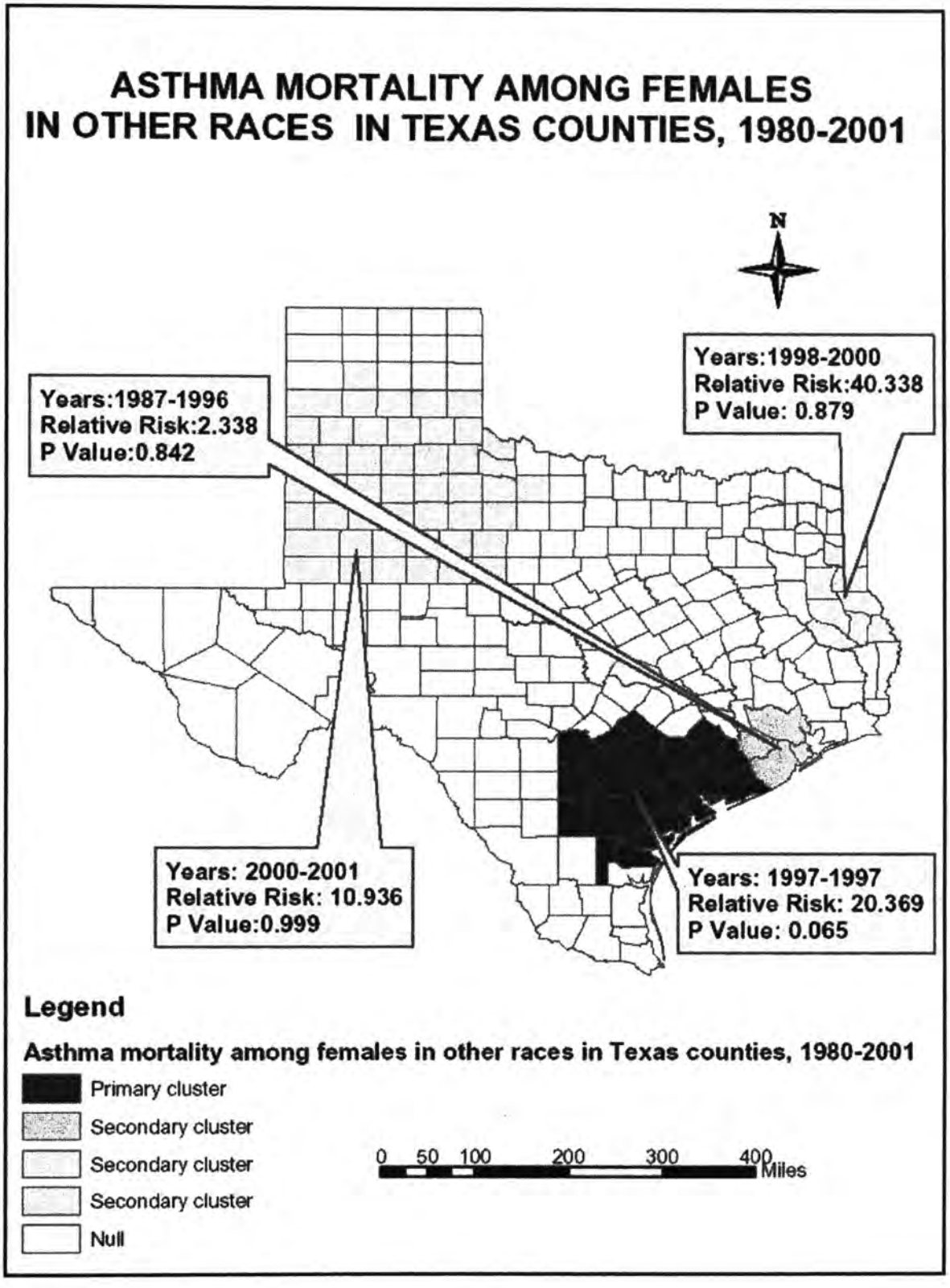

ASTHMA MORTALITY AMONG FEMALES
IN OTHER RACES IN TEXAS COUNTIES, 1980-2001
N
Years:1998-2000
Relative Risk:40.338
P Value: 0.879
Years:1987-1996
Relative Risk:2.338
P Value:0.842
Years: 2000-2001
Relative Risk: 10.936
P Value:0.999
Years: 1997-1997
Relative Risk: 20.369
P Value: 0.065
Legend
Asthma mortality among females in other races in Texas counties, 1980-2001
Primary cluster
Secondary cluster
Secondary cluster
Secondary cluster
Null
0 50 100 200 300 400
Miles

Fig 15

APPENDIX B

MAP OF TEXAS MEDICALLYUNDERSERVED COUNTIES

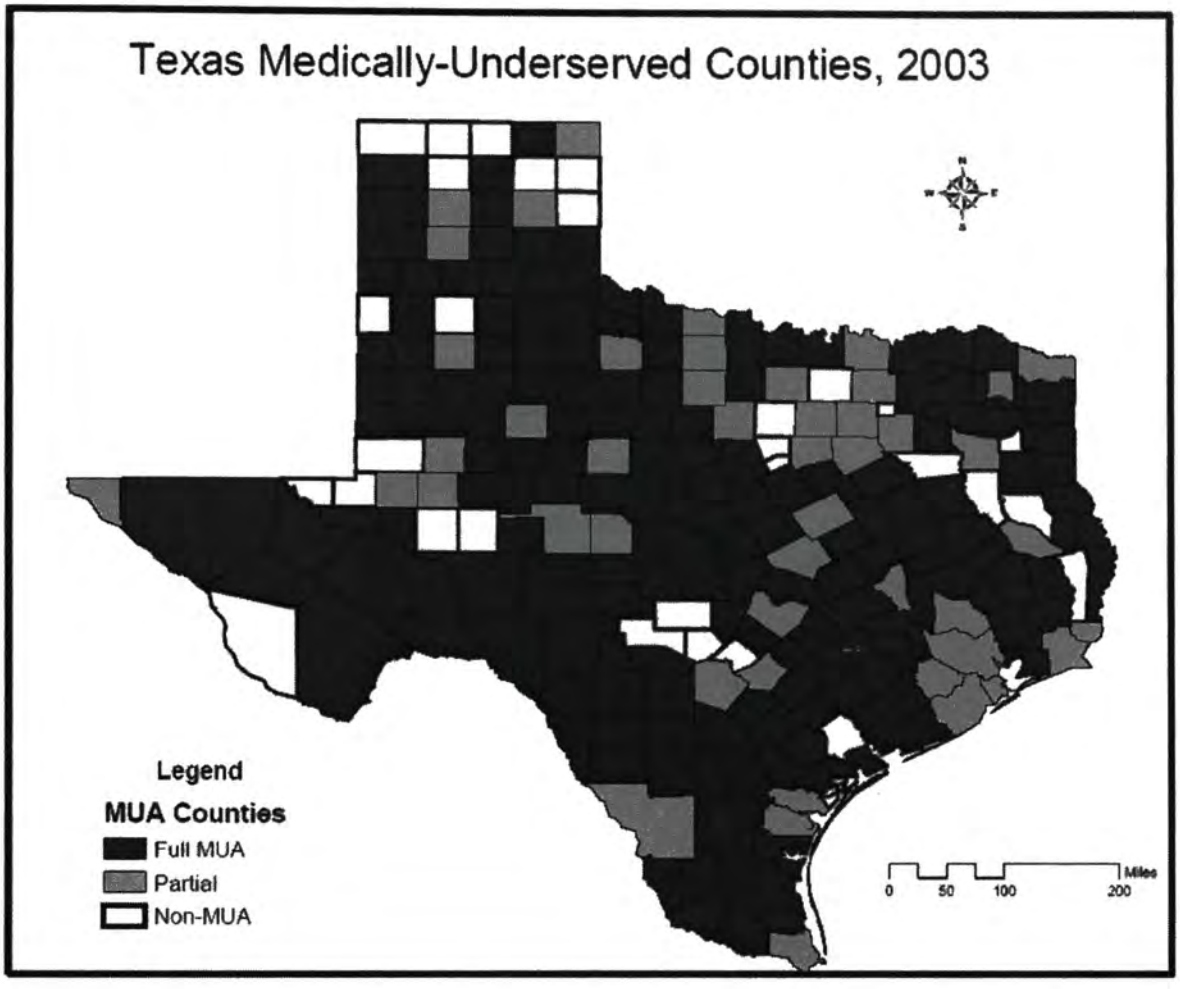
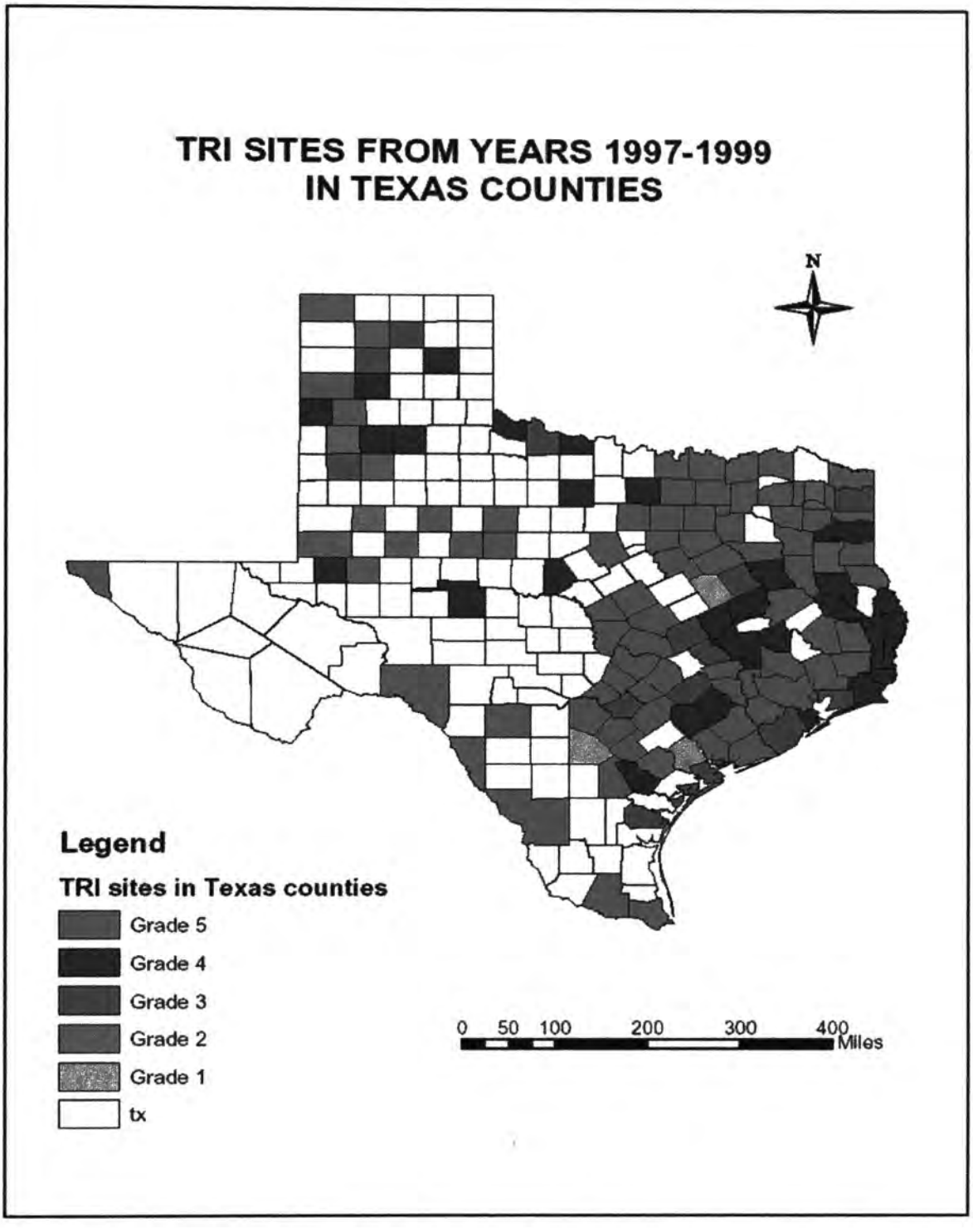

Texas Medically-Underserved Counties, 2003
Legend
MUA Counties
Full MUA
Partial
Non-MUA
0
50
100
200
Miles

Fig 16

APPENDIX C

TOXIC RELEASE INVENTORY SITES FROM YEARS 1997-19999 IN TEXAS COUNTIES


TRI SITES FROM YEARS 1997-1999
IN TEXAS COUNTIES
N
Legend
TRI sites in Texas counties
Grade 5
Grade 4
Grade 3
Grade 2
Grade 1
tx
0 50 100 200 300 400
Miles

APPENDIX D

OVERLAP MAPS OF ASTHMA MORTALITY AND TOXIC RELEASES IN TEXAS COUNTIES

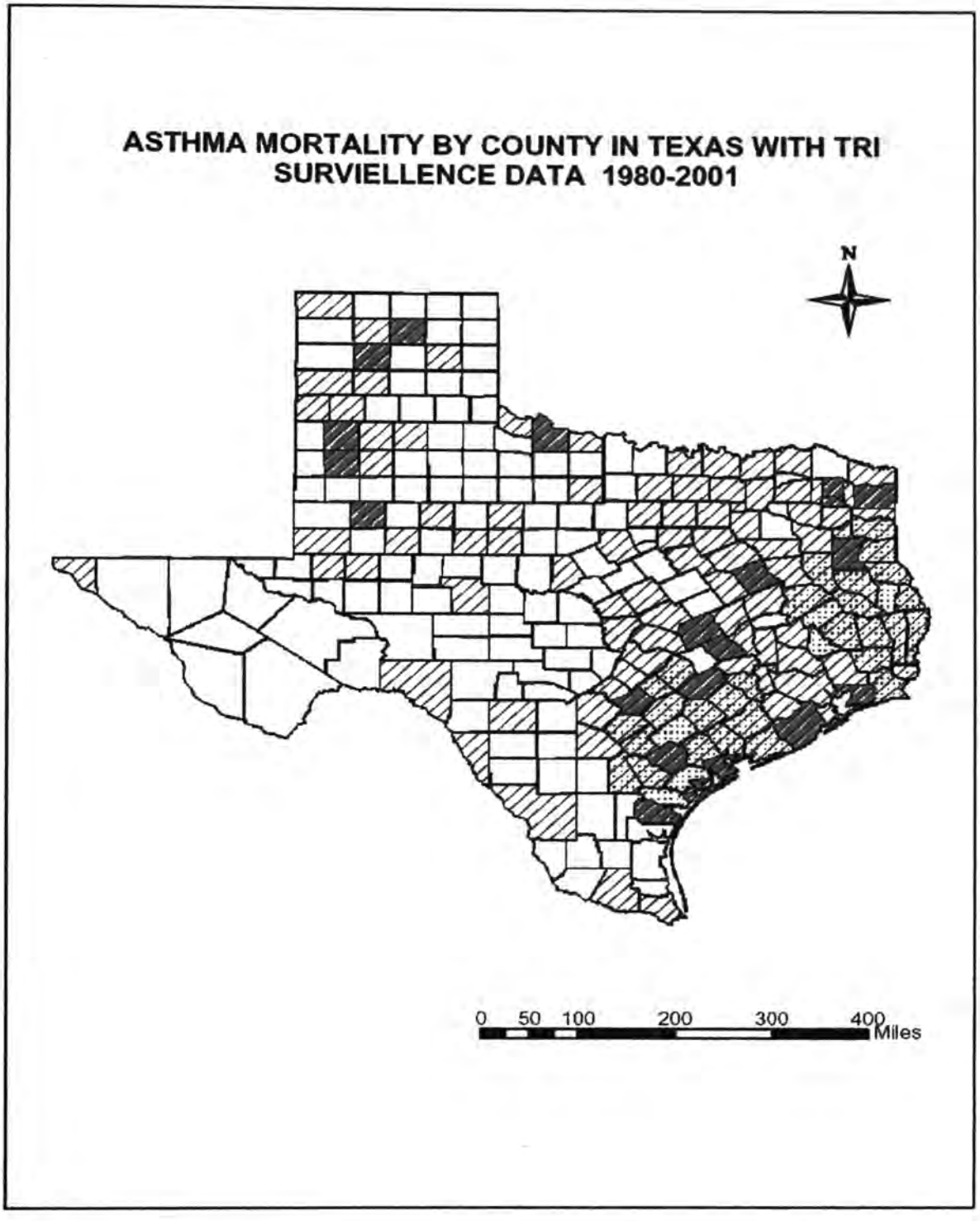

ASTHMA MORTALITY BY COUNTY IN TEXAS WITH TRI SURVIELLENCE DATA 1980-2001
N
0 50 100 200 300 400
Miles

Fig 17

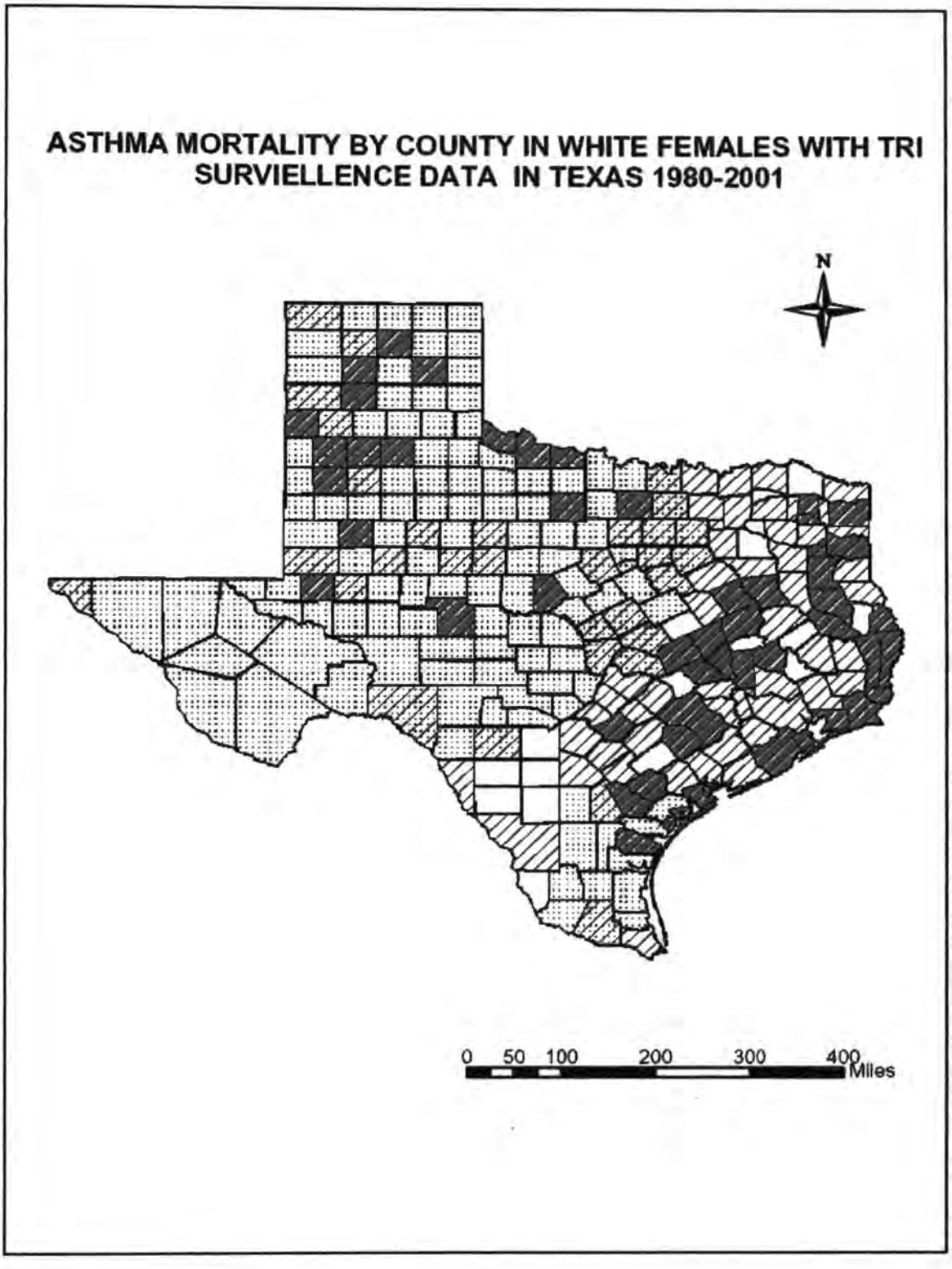

ASTHMA MORTALITY BY COUNTY IN WHITE FEMALES WITH TRI SURVIELLENCE DATA IN TEXAS 1980-2001
N
0 50 100 200 300 400
Miles

Fig 18

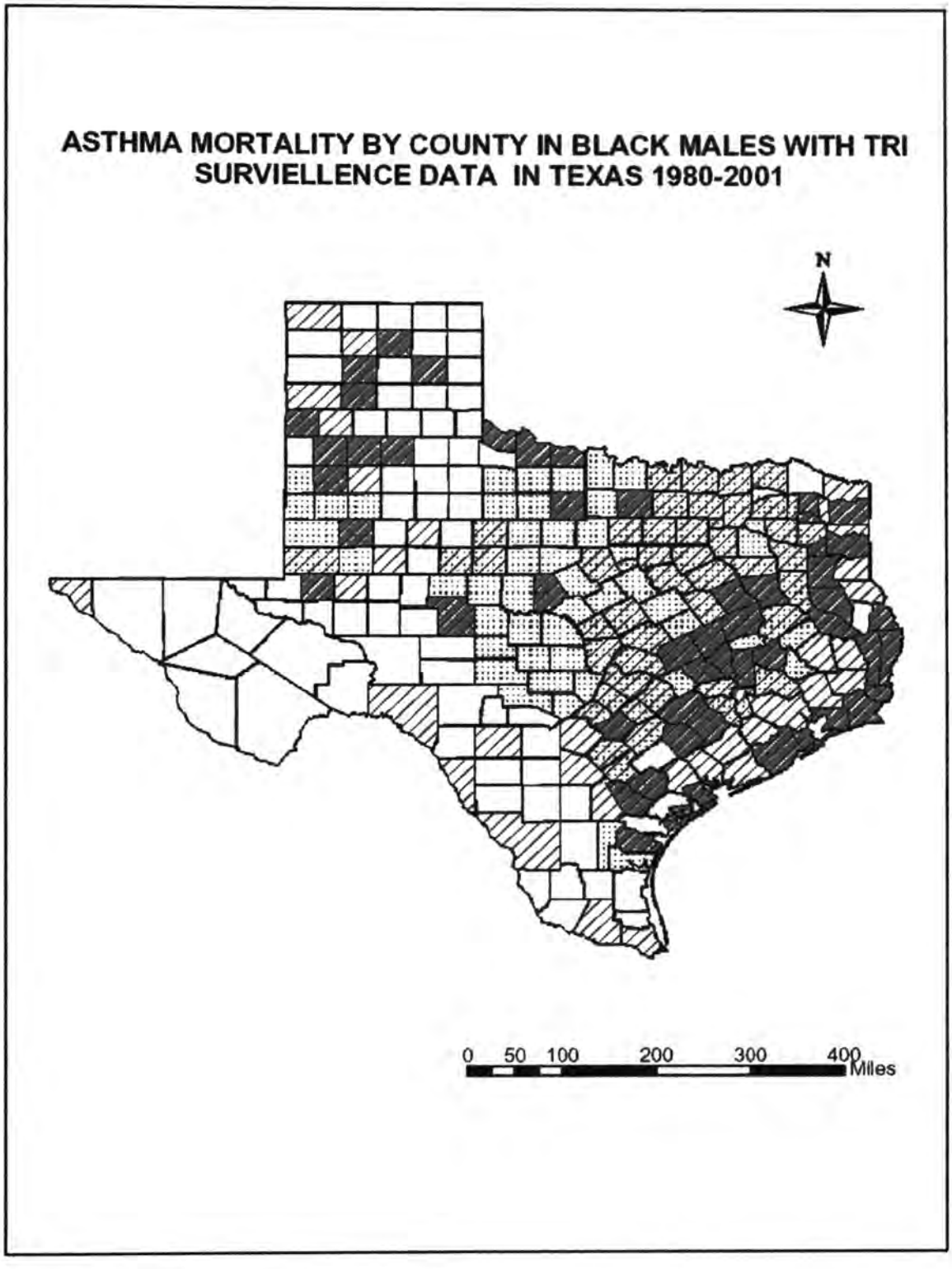

ASTHMA MORTALITY BY COUNTY IN BLACK MALES WITH TRI SURVIELLENCE DATA IN TEXAS 1980-2001
N
0 50 100 200 300 400
Miles

Fig 19

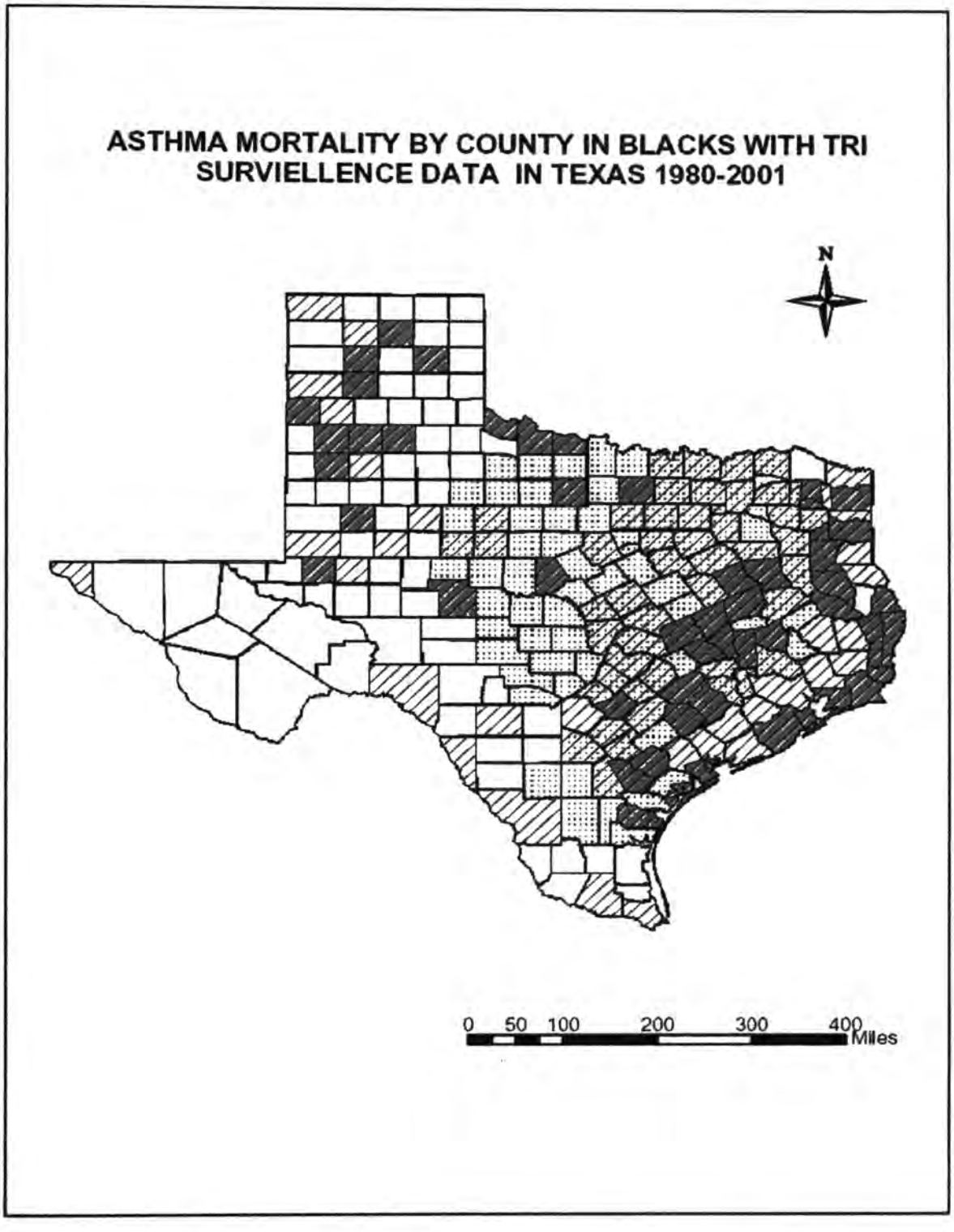

ASTHMA MORTALITY BY COUNTY IN BLACKS WITH TRI SURVIELLENCE DATA IN TEXAS 1980-2001
N
0 50 100 200 300 400
Miles

Fig 20

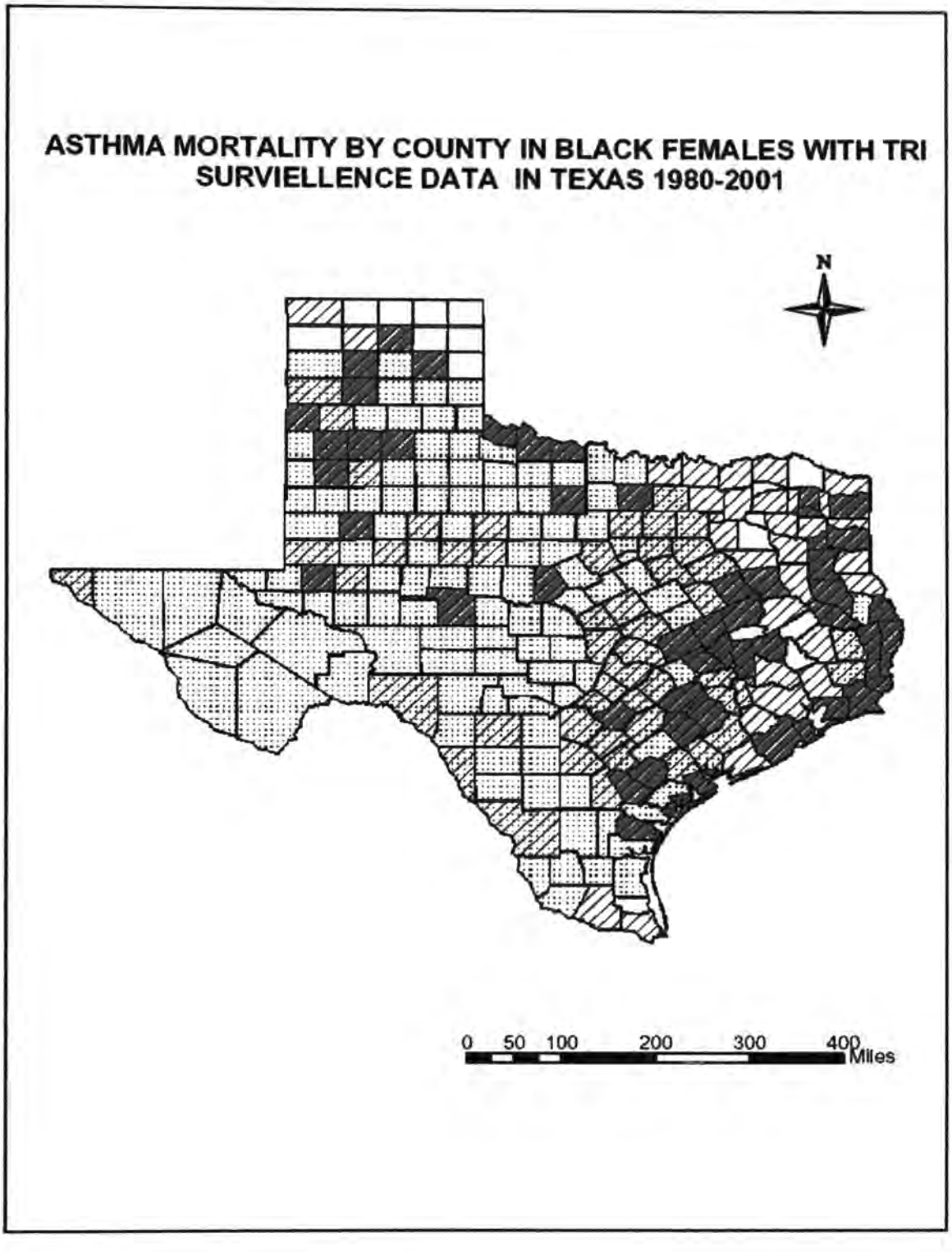

ASTHMA MORTALITY BY COUNTY IN BLACK FEMALES WITH TRI SURVIELLENCE DATA IN TEXAS 1980-2001
N
0 50 100 200 300 400
Miles

Fig 21

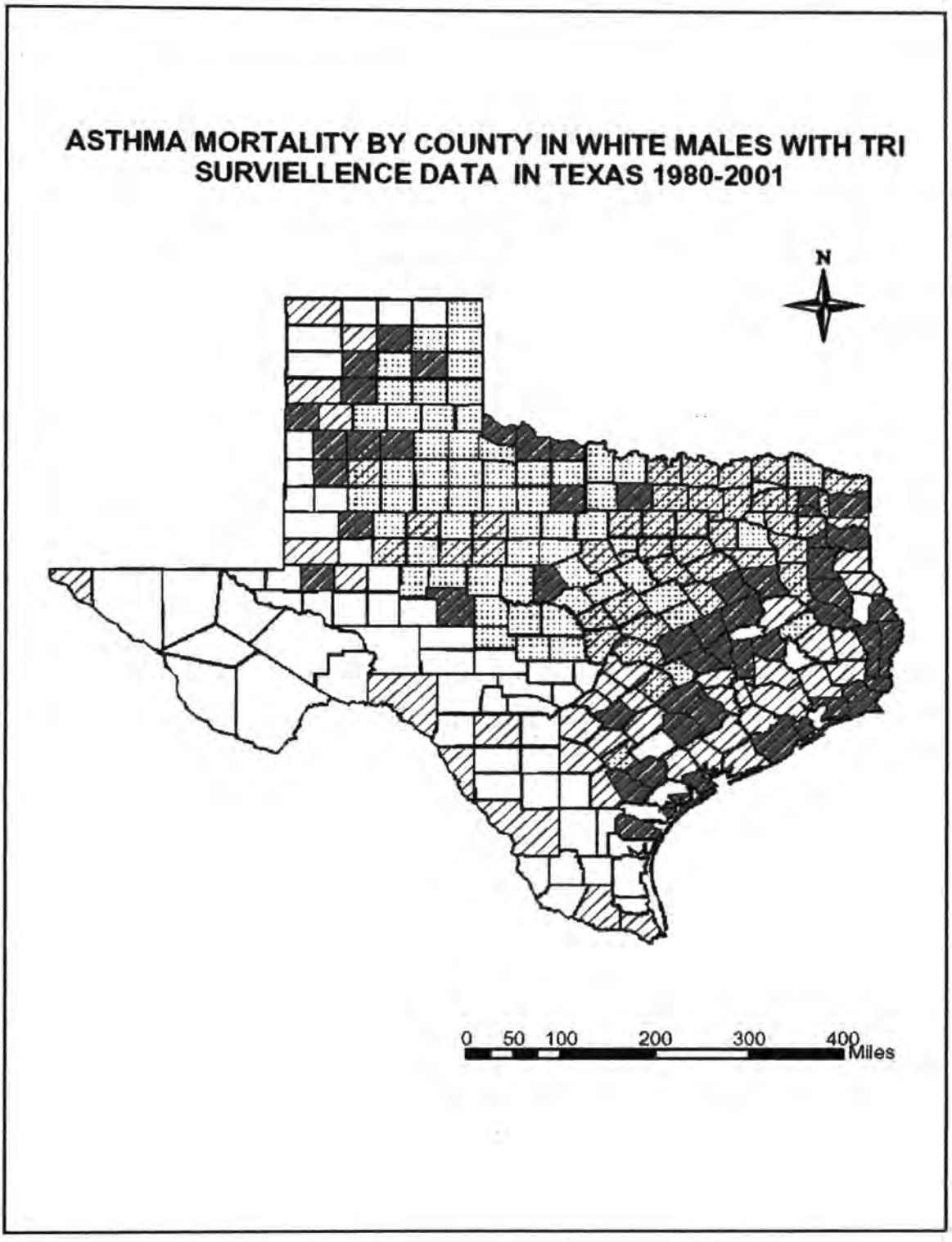

ASTHMA MORTALITY BY COUNTY IN WHITE MALES WITH TRI SURVIELLENCE DATA IN TEXAS 1980-2001
N
0 50 100 200 300 400
Miles

Fig 22

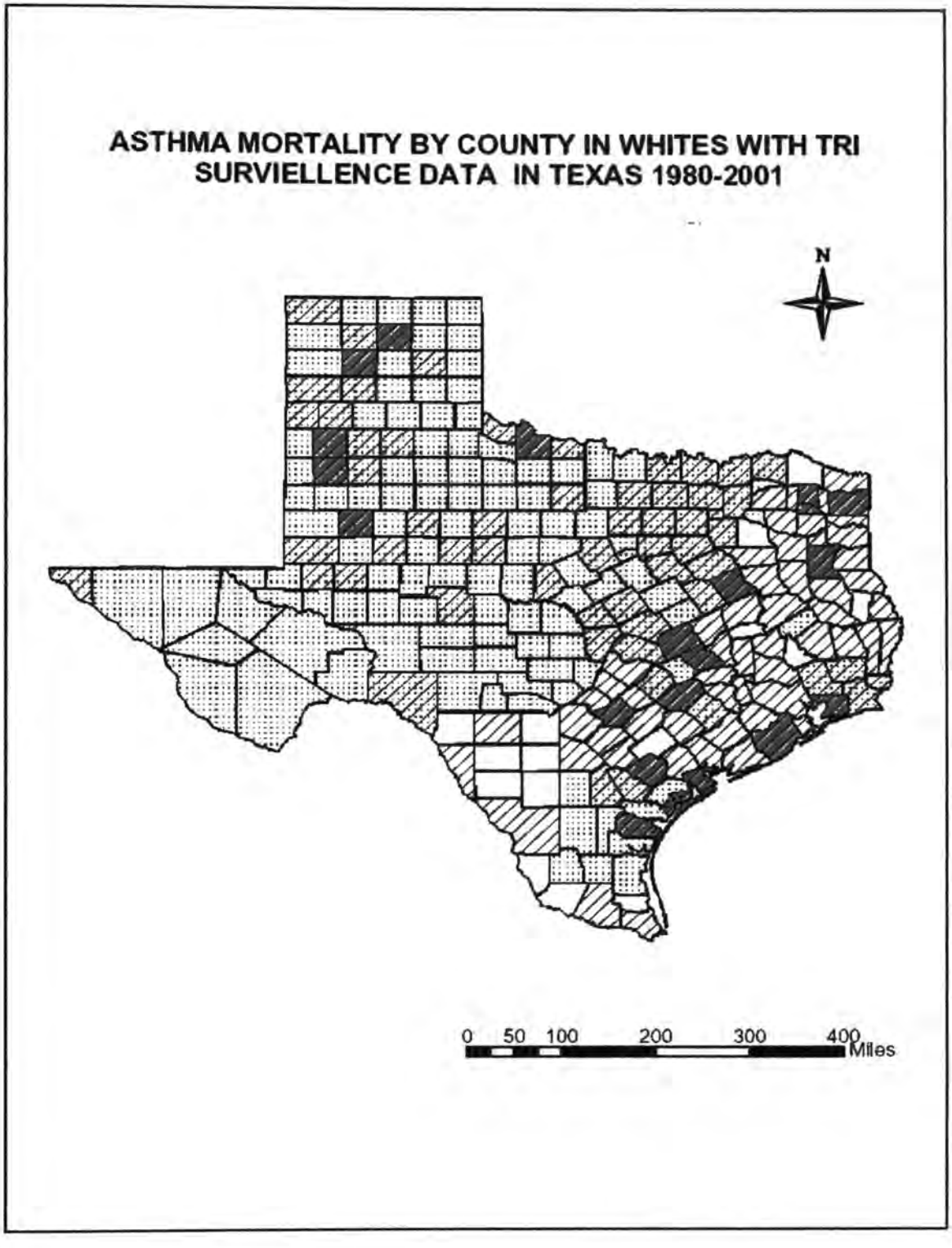

ASTHMA MORTALITY BY COUNTY IN WHITES WITH TRI SURVIELLENCE DATA IN TEXAS 1980-2001
N
0 50 100 200 300 400
Miles

Fig 23

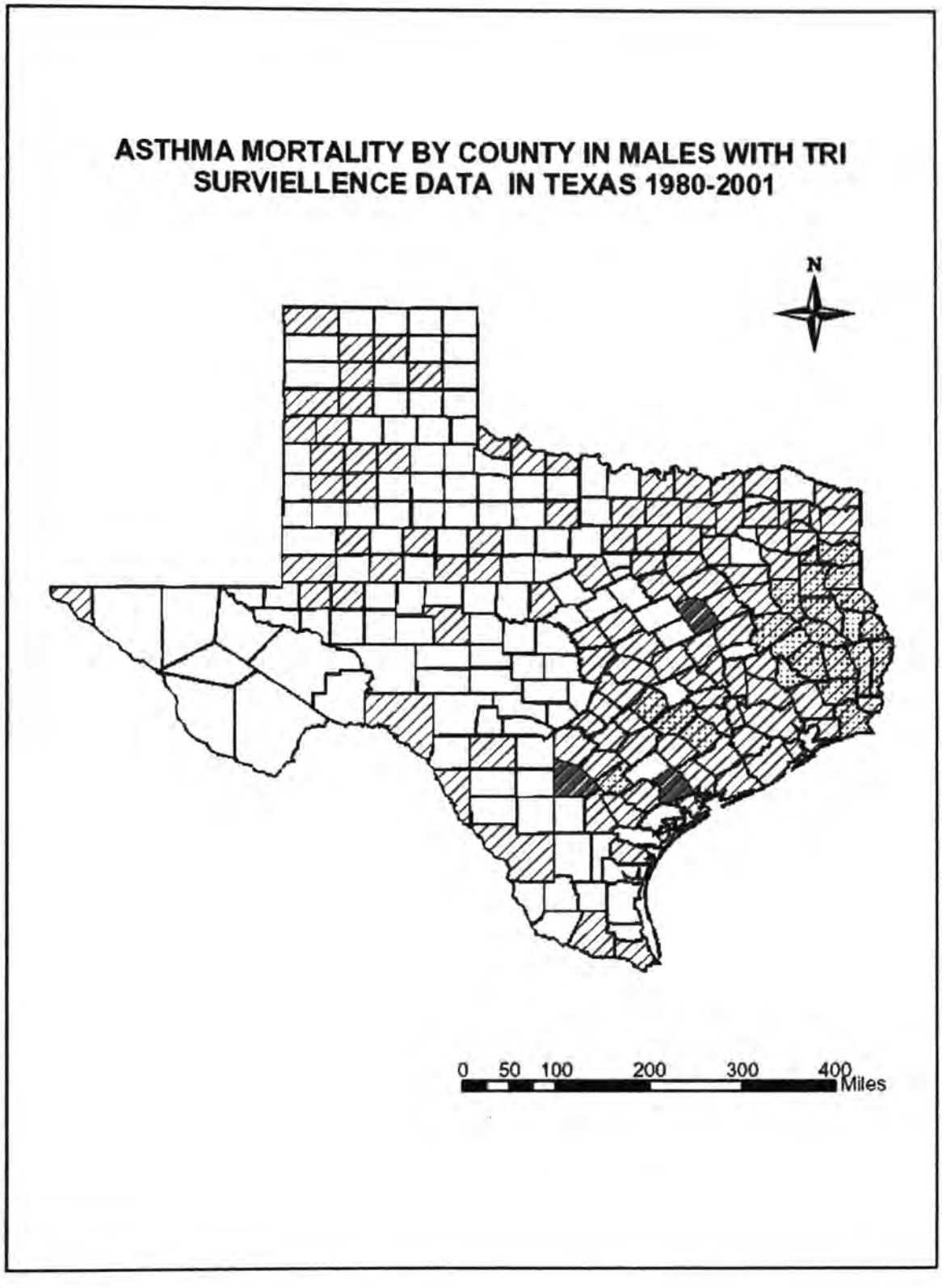

ASTHMA MORTALITY BY COUNTY IN MALES WITH TRI SURVIELLENCE DATA IN TEXAS 1980-2001
N
0 50 100 200 300 400
Miles

Fig 24

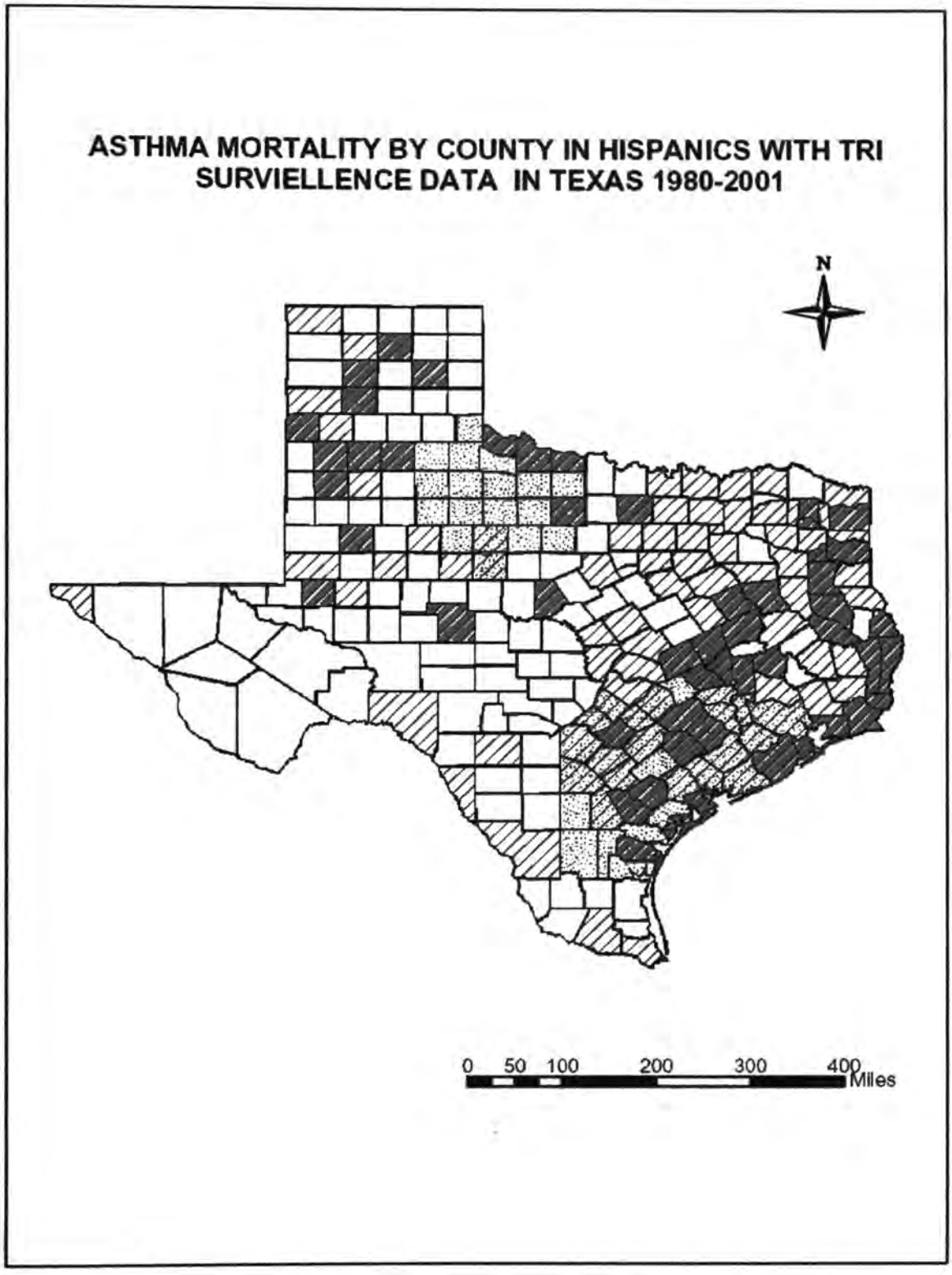

ASTHMA MORTALITY BY COUNTY IN HISPANICS WITH TRI SURVIELLENCE DATA IN TEXAS 1980-2001
N
0 50 100 200 300 400
Miles

Fig 25

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