United States
Environmental Protection
Agency

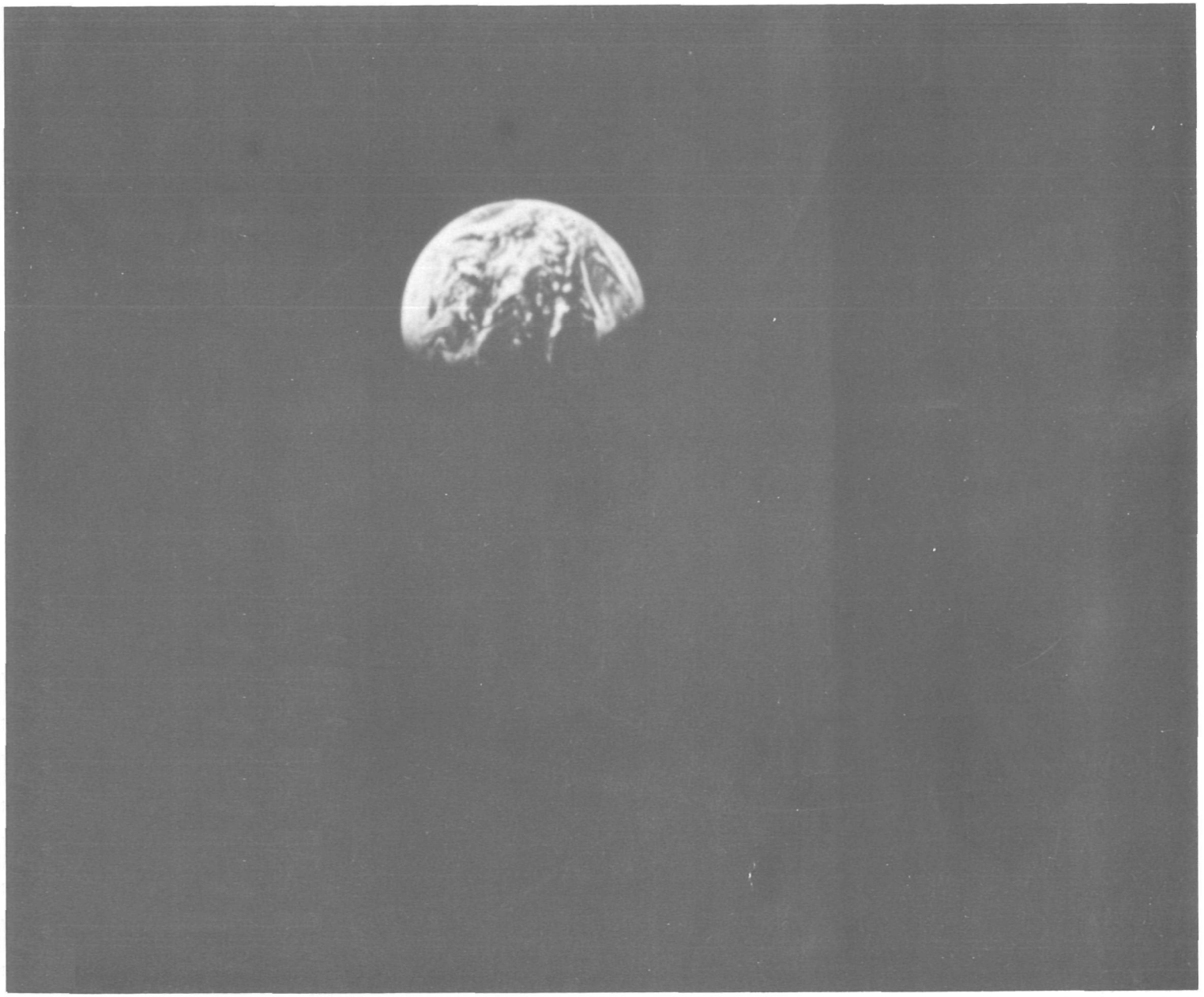
Policy, Planning,
And Evaluation
(PM-221)

EPA-230-05-89-057
June 1989

EPA

The Potential Effects Of Global Climate Change On The United States

Appendix G
Health



Printed on Recycled Paper

THE POTENTIAL EFFECTS OF GLOBAL CLIMATE CHANGE ON THE UNITED STATES:

APPENDIX G - HEALTH

Editors: Joel B. Smith and Dennis A. Tirpak

OFFICE OF POLICY, PLANNING AND EVALUATION
U.S. ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, DC 20460

MAY 1989

TABLE OF CONTENTS

PREFACE

The ecological and economic implications of the greenhouse effect have been the subject of discussion within the scientific community for the past three decades. In recent years, members of Congress have held hearings on the greenhouse effect and have begun to examine its implications for public policy. This interest was accentuated during a series of hearings held in June 1986 by the Subcommittee on Pollution of the Senate Environment and Public Works Committee. Following the hearings, committee members sent a formal request to the EPA Administrator, asking the Agency to undertake two studies on climate change due to the greenhouse effect.

> One of the studies we are requesting should examine the potential health and environmental effects of climate change. This study should include, but not be limited to, the potential impacts on agriculture, forests, wetlands, human health, rivers, lakes, and estuaries, as well as other ecosystems and societal impacts. This study should be designed to include original analyses, to identify and fill in where important research gaps exist, and to solicit the opinions of knowledgeable people throughout the country through a process of public hearings and meetings.

To meet this request, EPA produced the report entitled *The Potential Effects of Global Climate Change on the United States.* For that report, EPA commissioned fifty-five studies by academic and government scientists on the potential effects of global climate change. Each study was reviewed by at least two peer reviewers. The Effects Report summarizes the results of all of those studies. The complete results of each study are contained in Appendices A through J.

Appendix	Subject
A	Water Resources
B	Sea Level Rise
C	Agriculture
D	Forests
E	Aquatic Resources
F	Air Quality
G	Health
H	Infrastructure
I	Variability
J	Policy

GOAL

The goal of the Effects Report was to try to give a sense of the possible direction of changes from a global warming as well as a sense of the magnitude. Specifically, we examined the following issues:

- sensitivities of systems to changes in climate (since we cannot predict regional climate change, we can only identify sensitivities to changes in climate factors)
- the range of effects under different warming scenarios
- regional differences among effects
- interactions among effects on a regional level

- o national effects
- o uncertainties
- o policy implications
- o research needs

The four regions chosen for the studies were California, the Great Lakes, the Southeast, and the Great Plains. Many studies focused on impacts in a single region, while others examined potential impacts on a national scale.

SCENARIOS USED FOR THE EFFECTS REPORT STUDIES

The Effects Report studies used several scenarios to examine the sensitivities of various systems to changes in climate. The scenarios used are plausible sets of circumstances although none of them should be considered to be predictions of regional climate change. The most common scenario used was the doubled CO_2 scenario ($2XCO_2$), which examined the effects of climate under a doubling of atmospheric carbon dioxide concentrations. This doubling is estimated to raise average global temperatures by 1.5 to 4.5°C by the latter half of the 21st century. Transient scenarios, which estimate how climate may change over time in response to a steady increase in greenhouse gases, were also used. In addition, analog scenarios of past warm periods, such as the 1930s, were used.

The scenarios combined average monthly climate change estimates for regional grid boxes from General Circulation Models (GCMs) with 1951-80 climate observations from sites in the respective grid boxes. GCMs are dynamic models that simulate the physical processes of the atmosphere and oceans to estimate global climate under different conditions, such as increasing concentrations of greenhouse gases (e.g., $2XCO_2$).

The scenarios and GCMs used in the studies have certain limitations. The scenarios used for the studies assume that temporal and spatial variability do not change from current conditions. The first of two major limitations related to the GCMs is their low spatial resolution. GCMs use rather large grid boxes where climate is averaged for the whole grid box, while in fact climate may be quite variable within a grid box. The second limitation is the simplified way that GCMs treat physical factors such as clouds, oceans, albedo, and land surface hydrology. Because of these limitations, GCMs often disagree with each other on estimates of regional climate change (as well as the magnitude of global changes) and should not be considered to be predictions.

To obtain a range of scenarios, EPA asked the researchers to use output from the following GCMs:

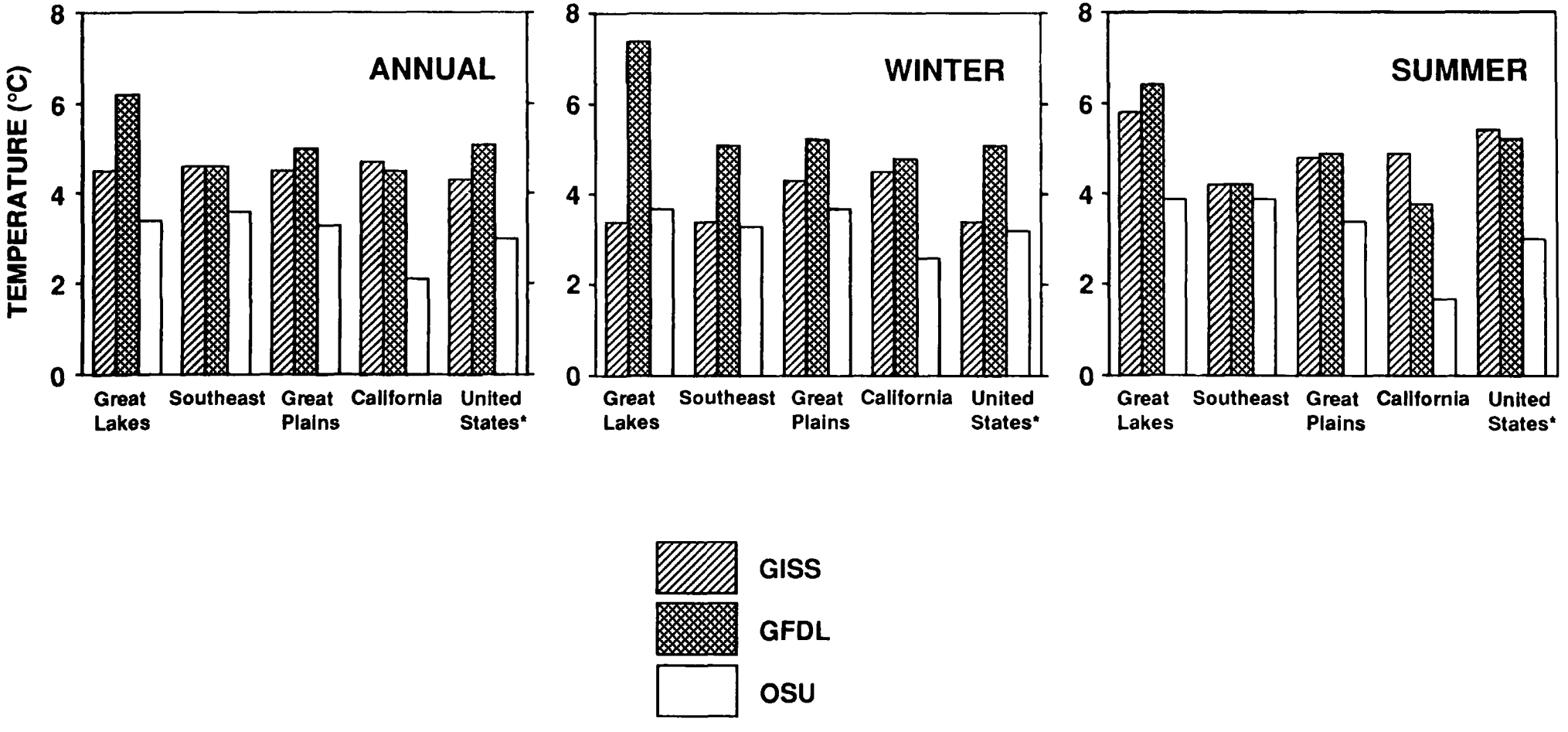
- o Goddard Institute for Space Studies (GISS)
- o Geophysical Fluid Dynamics Laboratory (GFDL)
- o Oregon State University (OSU)

Figure 1 shows the temperature change from current climate to a climate with a doubling of CO_2 levels, as modeled by the three GCMs. The figure includes the GCM estimates for the four regions. Precipitation changes are shown in Figure 2. Note the disagreement in the GCM estimates concerning the direction of change of regional and seasonal precipitation and the agreement concerning increasing temperatures.

Two transient scenarios from the GISS model were also used, and the average decadal temperature changes are shown in Figure 3.

FIGURE 1. TEMPERATURE SCENARIOS

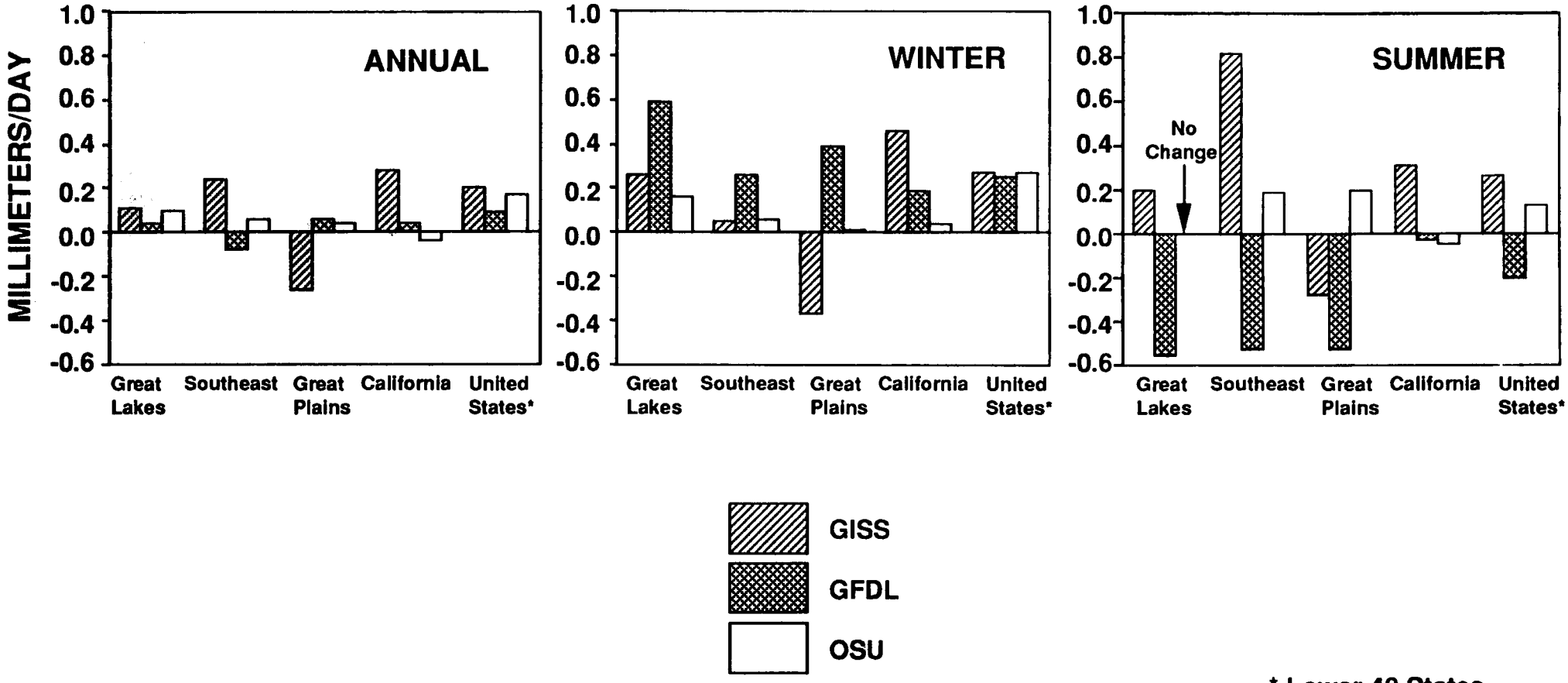
GCM Estimated Change in Temperature from $1xCO_2$ to $2xCO_2$

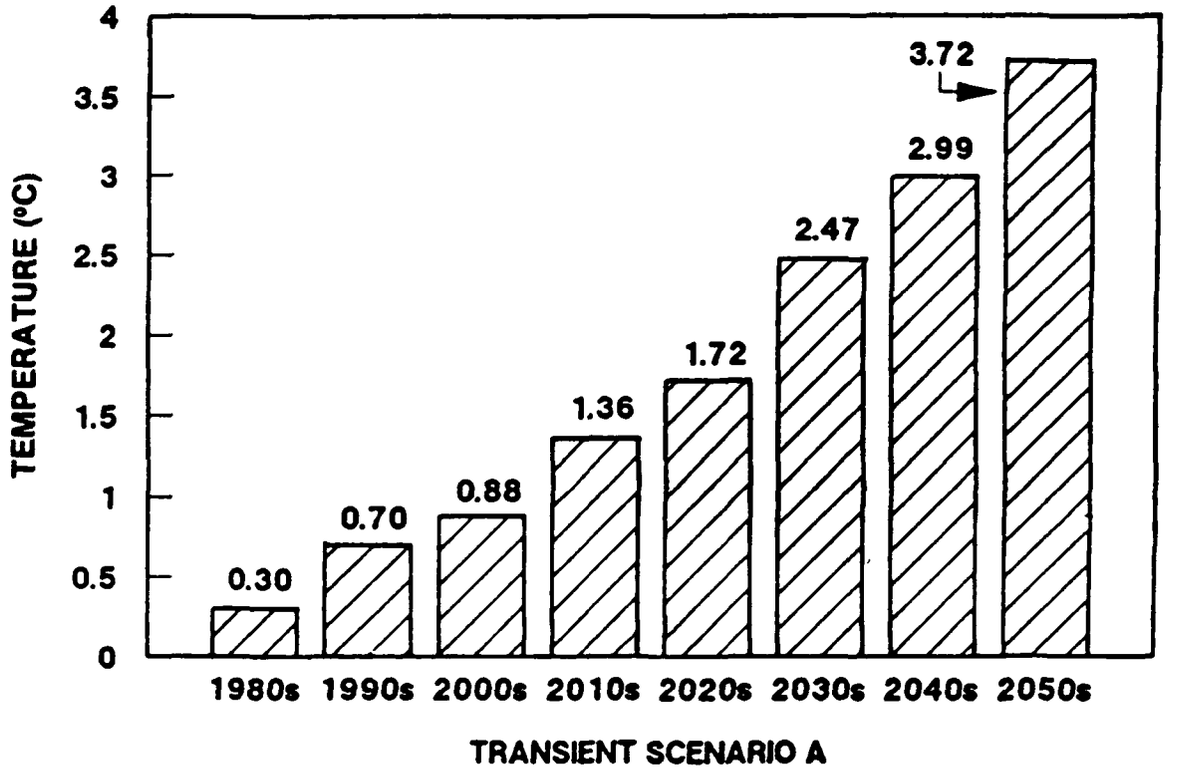


* Lower 48 States

FIGURE 2. PRECIPITATION SCENARIOS

GCM Estimated Change in Precipitation from $1xCO_2$ to $2xCO_2$





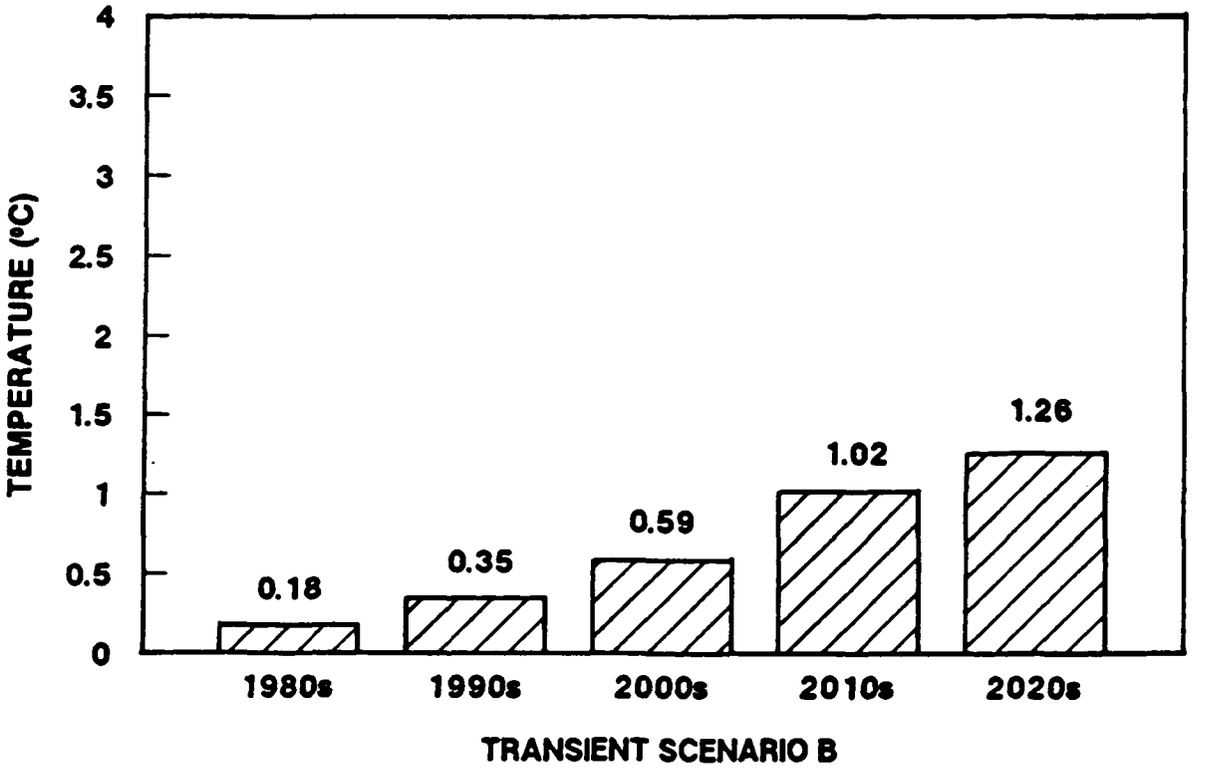


FIGURE 3. GISS TRANSIENTS "A" AND "B" AVERAGE TEMPERATURE CHANGE FOR LOWER 48 STATES GRID POINTS.

EPA specified that researchers were to use three doubled CO_2 scenarios, two transient scenarios, and an analog scenario in their studies. Many researchers, however, did not have sufficient time or resources to use all of the scenarios. EPA asked the researchers to run the scenarios in the following order, going as far through the list as time and resources allowed:

1. GISS doubled CO_2
2. GFDL doubled CO_2
3. GISS transient A
4. OSU doubled CO_2
5. Analog (1930 to 1939)
6. GISS transient B

ABOUT THESE APPENDICES

The studies contained in these appendices appear in the form that the researchers submitted them to EPA. These reports do not necessarily reflect the official position of the U.S. Environmental Protection Agency. Mention of trade names does not constitute an endorsement.

THE IMPACT OF CO_2 AND TRACE GAS-INDUCED CLIMATE CHANGES UPON HUMAN MORTALITY

by

Laurence S. Kalkstein
Center for Climatic Research
University of Delaware
Newark, DE 19716

Contract No. CR81430101

CONTENTS

FINDINGS[1]

The objective of this study is to estimate changes in human mortality attributed to potential changes in climate. The major result is an estimation of the number of deaths attributed to the increased incidence of extreme weather episodes predicted by numerous climate change models.

The evaluation covers 15 cities around the country, and daily mortality data for 11 summer and winter seasons were extracted and standardized in a manner that facilitates intercity comparisons. The mortality totals were divided into total and elderly categories, and separate evaluations were developed for all causes of death and those causes considered to be "weather-related."

A summary of the results follows:

1. Predictions of weather-induced mortality presently occurring during summer were attempted for the 15 cities exhibiting significant weather/mortality relationships. It is estimated that 1150 weather-induced deaths presently occur during an average summer season in the metropolitan areas of the 15 cities (these deaths are standardized totals, adjusted to the demography of a "standard city"). St. Louis, New york City, and Philadelphia rank first, second, and third, respectively, and each city presently averages over 100 standard city deaths per summer. The seven highest ranking cities are all found in the Midwest or Northeast. Four of the five lowest ranking cities are found in the South, with New Orleans and Oklahoma City experiencing virtually no deaths attributed to weather in summer.

2. Predicted future weather-induced mortality in summer rises rapidly as the scenarios become warmer and if people fail to acclimatize to the increased heat. The total weather-induced mortality estimate exceeds 7200 standard city deaths attributed to weather during an average summer under $2XCO_2$ conditions and assuming no acclimatization by the population. The magnitude of the increase varies significantly between cities. St. Louis exhibits the greatest number of deaths throughout all the warming scenarios. Other cities with predicted rapid rises in mortality with future warming are Los Angeles, Memphis, Philadelphia, and New York. New Orleans and Oklahoma City are cities least affected by predicted warming.

3. Predicted future weather-induced mortality in summer might actually decline in about half of the 15-city sample if the population fully acclimatizes to the increasing heat. Nevertheless, a moderate rise in mortality is predicted for the 15-city sample if full acclimatization occurs, and weather-induced mortality is predicted to double over present levels even if acclimatization is complete. Acclimatized mortality estimates for the 65+ age category are very similar to the total mortality estimates. By the design of the model, the comparatively modest rise in acclimatized mortality (as compared to unacclimatized predictions) parallels the response of people today who reside in southern cities with hot climates. Southern cities represent analogs of expected climate in northern cities, and these warmer cities exhibit fewer numbers of weather-induced deaths in summer. Thus, if people in northern cities fully acclimatize to the increased warmth, the rise in weather-induced mortality is predicted to be more modest. Estimates of future mortality assuming partial acclimatization (values midway between full and no acclimatization) indicate sizable increases in mortality as the weather became warmer.

---

[1]Although the information in this report has been funded wholly or partly by the U.S. Environmental Protection Agency under cooperative agreement number CR81430101 at the Center for Climatic Research, University of Delaware, it does not necessarily reflect the Agency's views, and no official endorsement should be inferred from it.

4. Present-day estimates of winter mortality indicate that weather-induced deaths are much less important in winter than in summer. The results differ from those uncovered in summer regarding the relative impact of weather. Most of the estimated winter deaths occur in regions with relatively severe winter climates, while the smallest number of deaths are found in mild weather cities. Conversely, in the summer, estimated weather-induced deaths are low in areas with severe summer climates.

5. Winter unacclimatized, partially acclimatized, and acclimatized predictions indicate that sharp drops in mortality are expected if the weather becomes warmer. The unacclimatized results differ from those uncovered in summer, when dramatic rises in mortality are predicted. The unacclimatized drop in winter may be related to fewer numbers of days below the threshold temperature.

Global warming could have an enormous impact upon human mortality through the 21st century. If the population does not fully acclimatize, over 7000 deaths attributed to the increasingly harsh weather can occur in the metropolitan areas of our 15-city sample. This figure is more startling when it is considered that these numbers correspond to average summer conditions. An analog of the very hot summer of 1980 occurring in the 21st century will no doubt increase weather-induced mortality to a much higher number than 7000.

Although fully acclimatized predictions are more modest, some general increases are still expected even under these conditions. If it is assumed that people partially acclimatize (possibly the most realistic scenario), the increases in mortality are larger, and weather-related deaths may increase by four to five times over present levels during the summer.

CHAPTER 1

INTRODUCTION

The impact of inadvertent climatic changes upon the human population has long been the subject of speculation. Although a large body of literature is devoted to the impact of variable climate upon the socioeconomic sector, very little has been done to estimate how predicted changes in climate might affect the health of the general population.

The objective of this study is to estimate changes in human mortality attributed to predicted changes in climate due to increased concentrations of CO_2 and other trace gases in the atmosphere. The result will be an estimation of the number of deaths attributed to the increased incidence of extreme weather episodes predicted by various climate change models.

The research presented here addresses two important priorities in climate/health studies. First, an assessment of CO_2/trace gas-induced climatic changes upon humans represents a timely and necessary aspect of air pollution-health analyses. The additional evaluation of climatic impact, rather than air pollution concentration alone, represents an important addition to any pollution/health research. Second, it is imperative that government agencies understand the implications of long-term climatic change in an attempt to develop mitigation policies. If the environmental impacts of inadvertent climatic change are quantified, regulatory action may be implemented with greater efficiency.

The following is a specific list of items addressed by this research:

1. Fifteen cities will be evaluated (Table 1), and present-day analogs that duplicate their predicted future climate regimes will be developed.

2. Weather/mortality relationships for the elderly (greater than 65 years of age) and the total population will be determined.

3. Estimates of future mortality assuming climatic warming will be presented. These estimates will be based on future weather scenarios as predicted by three Goddard Institute For Space Sciences (GISS) Global Circulation Models (GCMs).

There are several unavoidable limitations to this study. First, this evaluation concentrates on urban mortality variations. The scarcity of rural mortality data will curtail our development of an accurate rural mortality assessment procedure. Unforeseen future population changes and/or regional population shifts might represent a second limitation. Third, analog cities, used to approximate the future climate of our target cities, may be very different from the target cities in terms of architectural or structural makeup. Thus the microclimate within the dwellings of analog and associated target cities may vary. Fourth, the numerous interrelationships between weather, pollution, social factors, morbidity, and mortality are tremendously complex, leading to sharp disagreements among scholars involving the differential impacts of weather on human health. This has historically discouraged the development of deterministic weather/mortality models, which leads to increased difficulty with interpretation of results.

Although no previous study has attempted to predict the impact of future weather changes on mortality, considerable work relating to present climate/mortality relationships has been reported (White and Hertz-Picciotto, 1985; Munn, 1986; Kalkstein and Valimont, 1987). For example, studies at the Centers for Disease Control (CDC) have identified a number of factors that may inhibit the onset of heat stroke, including the increased use of air conditioning, consumption of fluids, and living in well-shaded residences (Kilbourne et al.,

Table 1. Fifteen Cities Evaluated in This Study

1. Atlanta, GA	9. Minneapolis, MN
2. Chicago, IL	10. New Orleans, LA
3. Cincinnati, OH	11. New York, NY
4. Dallas, TX	12. Oklahoma City, OK
5. Detroit, MI	13. Philadelphia, PA
6. Kansas City, MO	14. St. Louis, MO
7. Los Angeles, CA	15. San Francisco, CA
8. Memphis, TN	

1982). Some researchers have found that many causes of deaths other than heat stroke increase during extreme weather (Applegate et al., 1981; Jones et al., 1982). In addition, mortality attributed to weather seems to vary considerably with age, sex, and race, although there is disagreement among researchers in defining the most susceptible population group (Oechsli and Buechley, 1970; Bridger et al., 1976; Lye and Kamal, 1977).

The impact of cold weather is less dramatic than that of hot weather, although mortality increases have been noted during extreme cold waves (CDC, 1982; Fitzgerald and Jessop, 1982; Gallow et al., 1984; Kalkstein, 1984). Hypothermia is a major contributor to weather-related mortality in winter, but many other causes of death also increase including influenza, pneumonia, accidents, carbon monoxide poisoning, and house fires (National Center for Health Statistics, 1978).

A frequent criticism of these studies points to certain cultural adjustments through time that may have an impact on weather/mortality relationships, such as the lessened exposure of people to extreme weather owing to the increased use of air conditioning. Surprisingly, several studies indicate that these cultural adjustments may have a minimal impact. Ellis and Nelson (1978) have noted that during the past 30 years, mortality during heat waves in New York City has not changed significantly despite the increased use of air conditioning. Analysis by Marmor (1975) supports this finding, and his study covering a 22-year period implied that air conditioning may be decreasing excess mortality during initial summer hot spells only. Thus it is possible that people do not require direct exposure to hostile external environments to be negatively affected by these environments. The knowledge that external unpleasant conditions exist might be sufficient to contribute to negative reactions (Ulrich, 1984).

One of the major questions that must be addressed when evaluating the impact of long-term changes in weather on human health involves the importance of acclimatization, which represents the increased ability of humans to withstand stressful conditions with repeated exposure. Several studies have evaluated acclimatization as a factor contributing to heat-related deaths. Gover (1938) reported that excess mortality during a second heat wave in any year will be slight in comparison to excess mortality during the first, even if the second heat wave is unusually extreme. Two possible explanations for this phenomenon are provided. First, the weak and susceptible members of the population die in the early heat waves of summer, thus lowering the population of susceptible people who would have died during subsequent heat waves. Second, those who survive early heat waves become physiologically or behaviorally acclimatized and hence deal more effectively with later heat waves (Marmor, 1975). Findings at the recent Williamsburg Conference on Susceptibility to Inhaled Pollutants support the second idea. Reactive subjects responded only at the beginning of the ozone season each year (spring and summer), and were generally not affected by exposure later in the year (fall). Rotton (1983) suggests that geographical acclimatization is also significant, and people moving from a cool to a subtropical climate will adapt rather quickly, often within two weeks. However, the population must still make behavioral and cultural adjustments (Ellis, 1972). Further support for geographical acclimatization is provided by Kalkstein

et al. (1986), who noted that mortality increased dramatically during heat waves in northern cities, but no mortality increase was observed in southern cities even under the hottest conditions.

This report will describe the methodology used in the development of weather-related mortality predictions into the future. In addition, results of the empirical evaluation will be presented and interpreted, and an evaluation of the potential socioeconomic implications will be attempted.

CHAPTER 2

PROCEDURE

MORTALITY DATA

A very detailed mortality data base is presently available from the National Center for Health Statistics (NCHS), which contains records for every person who has died in this country from 1964 to the present (NCHS, 1978). The data contain information such as cause of death, place of death, age, date of death, sex, and race. These data were extracted for the standard metropolitan statistical areas (SMSAs) of all the cities incorporated in this study for 11 years: 1964-66, 1972-78, and 1980 (during intervening years, a sizable amount of information was missing from many records). The number of deaths each day for each SMSA was tabulated and divided into total deaths and elderly deaths. Thus, the relative sensitivities for both categories could be determined, as weather probably exerts a differential influence upon mortality between categories.

Certain causes of death, deemed "weather-related," were factored out and evaluated separately using two procedures (Table 2). First, weather-related causes of death were subjectively identified after consultation with Dr. Melvyn Tockman, an epidemiologist from Johns Hopkins University, and Dr. Steven Parnes, head, Division of Otolaryngology, Albany Medical College. The medical experts examined a listing containing approximately 10,000 causes of death (Department of Health and Human Services, 1980) and identified the causes they considered to be directly or indirectly influenced by weather. Second, a more objective method to isolate weather-related causes was attempted by correlating annual fluctuations for every cause of death for the entire nation with population-weighted values of monthly mean temperature and precipitation. The population-weighted procedure for developing weather variables is used commonly by the National Oceanic and Atmospheric Administration (NOAA) to estimate national impacts of weather on society; refer to Warren and LeDuc (1981) for computational details.

There is conflicting evidence in the literature about the validity of factoring out weather-related causes of death. Many researchers continue to utilize total mortality figures in their analyses, as deaths from a surprisingly large number of causes appear to escalate with more extreme weather (Applegate et al., 1981; Jones et al., 1982). In an attempt to circumvent this apparent disagreement among researchers, weather-related and all causes categories were evaluated separately in this study for the total and elderly mortality categories.

Although they are probably less meaningful for inter-regional comparison than standardized values, unstandardized mortality values provide a better estimate of the total magnitude of weather's influence upon mortality. The unstandardized values are computed for each city by multiplying the death rate by the true population of the city's SMSA (using 1980 census data).

DETERMINATION OF WEATHER/MORTALITY RELATIONSHIPS

The procedural framework used in this study for developing and interpreting climate/mortality relationships is outlined in Figure 1, which should supplement the discussion in this subchapter. Prior to an evaluation of the effects of future warming on mortality, it is necessary to define the historical relationships between weather and mortality. This section first describes the procedure to develop historical relationships, and then outlines the procedure to evaluate future climate/mortality relationships.

Table 2. Causes of Death Considered to be Weather Related

1. Active rheumatic fever
2. Adverse effect of medicinal agents
3. Cerebrovascular disease
4. Complications of medical care
5. Complications of pregnancy, childbirth, and the puerperium
6. Contusion and crushing with intact skin surface
7. Diseases of the arteries, arterioles, and capaillaries
8. Diseases of the blood and blood-forming organs
9. Diseases of the digestive system
10. Diseases of the musculoskeletal system and connective tissue
11. Diseases of the nervous system and sense organs
12. Diseases of the skin and subcutaneous tissue
13. Diseases of the veins and lymphatics
14. Effects of foreign body, entering through orifice
15. Endocrine, nutritional, and metabolic diseases
16. Fractures of the skull, spine, trunk, and limbs
17. Hypertensive disease
18. Influenza
19. Injury to nerves and spinal cord
20. Intracranial injury
21. Ischemic heart disease
22. Neoplasms: benign
23. Neoplasms: malignant
24. Superficial injury
25. Toxic effect of substances of chiefly non-medical source

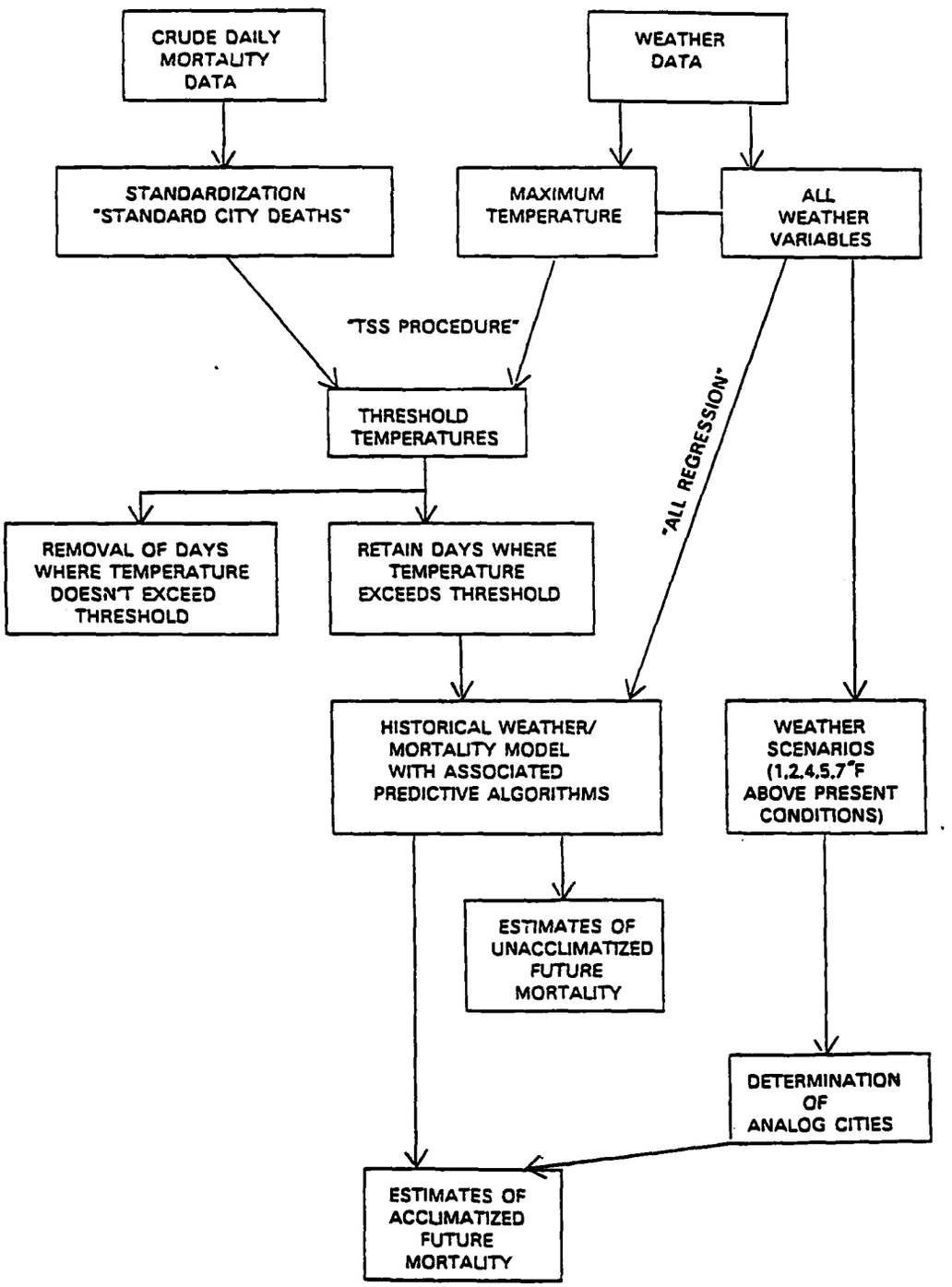


Figure 1. Procedural framework for climate/mortality analysis.

Threshold temperatures

Weather has been demonstrated to have some impact on daily mortality (Figure 2). During the heat wave of late July 1980 in New York City, deaths rose to over 50 percent above normal on the day with the highest maximum temperature (Kalkstein et al., 1986). Deaths among the elderly showed similar increases. In this study, daily changes in mortality were compared to 12 different weather elements which might have some influence on death rates (Table 3). One of these elements, a "time" variable (TIME), was also incorporated, which evaluated the intra-seasonal timing of the weather event. For example, it is hypothesized that a heat wave in August might have less of an influence than a similar heat wave in June, as the population would be unaccustomed to the June event. Thus, TIME simply assigns each day a number (e.g., June 1 is 1, June 2 is 2, July 1 is 31) representing its position in the summer (or winter) season.

Initial observations of daily deaths versus maximum temperature suggest that, in summer, weather has an impact on only the warmest 10-20 percent of the days; however, the relationship on those very warm days is impressive (Figure 3). Somewhat similar findings were uncovered for winter, and for certain SMSAs, the coldest 10 percent of days exhibited good weather/mortality relationships. Data were analyzed for the total and elderly mortality categories for each SMSA during summer and winter to compare the maximum temperature on the day of the deaths, as well as one, two, and three days prior to the day of the deaths to determine if a lag time exists between weather and the mortality response.

A unique aspect of this study involves the determination of a "threshold temperature," which represents that temperature beyond which mortality significantly increases (Kalkstein and Davis, 1985). The threshold temperature is calculated objectively by measuring the dissimilarity of mortality rates above and below a given temperature (refer to Kalkstein, in press, for a more detailed discussion). The threshold temperature for total deaths in New York City, for example, is 92°F (Figure 3), and mortality increases dramatically at temperatures above this level. This procedure can be repeated for winter, where the threshold temperature represents the temperature below which mortality increases.

Statistical manipulation

Once the threshold has been established, a procedure named "all regression" is used to determine which combination of weather elements (listed in Table 3) produces the best models ("best" is defined as possessing the highest R^2 value) for days beyond the threshold temperature (Draper and Smith, 1981). The next step involves choosing which regression model (for each combination of weather elements) best represents the historical relationships for that city. Complete multiple linear regressions were run for each model, which included regression diagnostics such as residuals plots and variance inflation factors (VIF) (SAS Institute, 1985). A high VIF indicates that two or more collinear independent variables are included in the model. When this is the case, one of the collinear variables is omitted from the model; the remaining variable explains a greater amount of the variance in mortality than the omitted variable. Because of this collinearity problem, CDH and maximum temperature are virtually never included in the same regression. The final selected model must meet the following criteria:

1. All included independent variables must be significant at the 0.05 level or better;

2. The VIF for all independent variables must be less than 5.00 (SAS Institute, 1985);

3. The residuals plots must indicate randomly distributed residuals;

4. The R^2 value of the final model must exceed 0.100.

An "adjusted" R^2 statistic was used in this study, which accounts for degrees of freedom in the regression model (Draper and Smith, 1981). Regression models possessing low degrees of freedom often exhibit inflated R^2

Table 3. Weather Variables Used in the Mortality Study

MAXIMUM TEMPERATURE (MAXT)
MINIMUM TEMPERATURE (MINT)
MAXIMUM DEWPOINT (MAXTD)
MINIMUM DEWPOINT (MINTD)
COOLING DEGREE HOURS (CDH): SUMMER ONLY*
HEATING DEGREE HOURS (HDH): WINTER ONLY**
3AM VISIBILITY (VISAM)
3PM VISIBILITY (VISPM)
3AM WIND SPEED (WNDAM)
3PM WIND SPEED (WNDPM)
CLOUD COVER (CLD)
TIME (TIME)

* CDH represents a measure of the day's warmth, and is calculated as follows:

$$CDH = \sum_{i=1}^{N} (T-90), \text{ where } T > 90$$

T represents the hourly temperature and N represents total hours with temperature above 90°.

** HDH represents a measure of the day's coldness, and is calculated as follows:

$$HDH = \sum_{i=1}^{M} (32-T), \text{ where } T < 32$$

T represents the hourly temperature and M represents total hours with the temperature below 32°F.

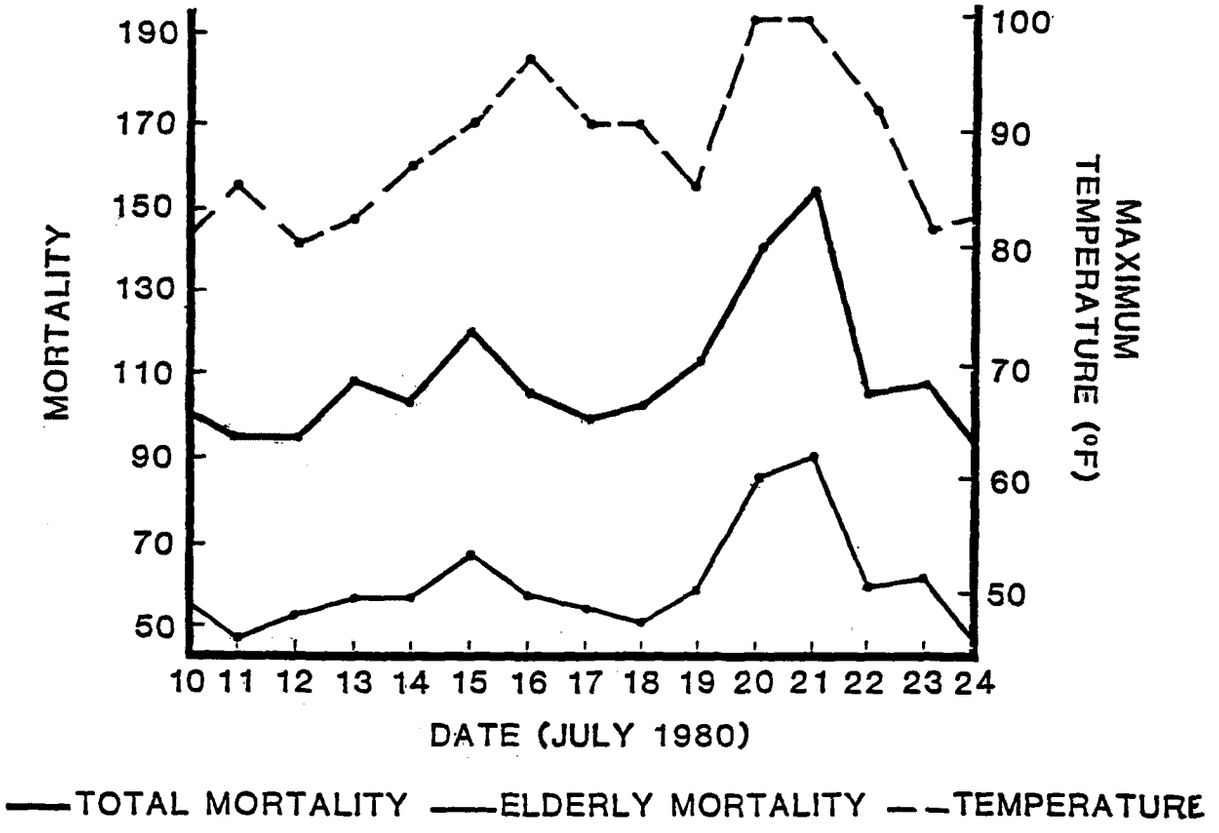


Figure 2. Mortality during a 1980 heat wave in New York City (Kalkstein et al., 1986).

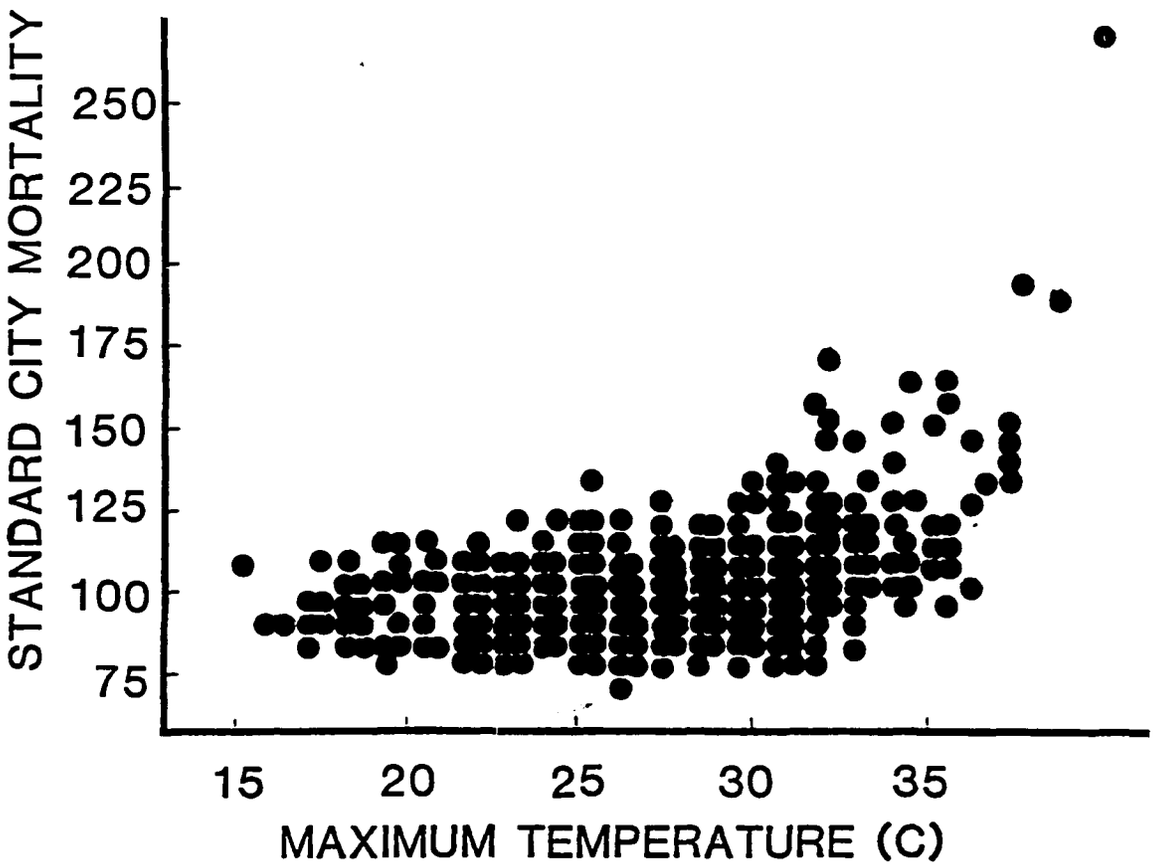


Figure 3. Daily summer-season standardized mortality vs. maximum temperature: New York City.

values, and the use of an adjusted R^2 statistic minimizes this problem. These guidelines insured some degree of quality control in the regression modeling, as thousands of regressions were computed to determine the best models.

Applying algorithms to weather scenarios

With historical relationships established, the next step is an attempt to estimate changes in mortality which might occur with predicted climatic warming. This study utilizes three GCM transient runs provided by NCAR/EPA (Jenne, 1987), and future predictions of climate have been developed for the cities in this study. The three runs are GISS transient A_1 (covering a 17-year period 30 years after the base period), GISS transient A_2 (covering a 17-year period 60 years after the base period), and GISS $2XCO_2$.

As described earlier, the base period for mortality includes a 17-year period extending from 1964 to 1980 (note that only 11 years of mortality data were available through this period). Thus the GISS transient A_1 scenario will estimate mortality for the period 1994-2010. The GISS transient A_2 scenario will estimate mortality for the period 2024-2040. The GISS $2XCO_2$ scenario will assume double CO_2 conditions occurring during the base period 1964-1980. New mortality estimates for each city were created for each scenario by using the algorithms developed from the historical data.

When measuring the impact of warming on future mortality, the question of acclimatization must be considered. Will people within each city respond to heat as they do today? Or will their reactions be similar to those of people who presently live in hotter climates? There is much disagreement in the literature concerning human acclimatization to changing weather. Some research indicates that acclimatization responses are very rapid (Marmor, 1975; Rotton, 1983), others think it is a much slower process (Kalkstein and Davis, 1985; Ellis, 1972), and a few imply that virtually no acclimatization occurs at all (Steadman, 1979). It is obvious that the full range of possibilities must be examined. First, the historical algorithms for each city that were developed from the previously described multiple regression procedure were applied to the three future weather scenarios. The mortality increases estimated from this procedure imply no acclimatization, as an assumption is made that people will respond to heat in the future in much the same way that they do today. Second, analog cities were established for each city evaluated to account for full acclimatization. For example, the use of one of the GISS scenarios to predict future weather in New York City will produce a regime which will approximate another city's present weather in the U.S. Thus using the GISS transient A_2 scenario to predict New York City's summer weather for the period 2024-2040 yields a weather regime approximating that of Kansas City, MO, today. Since Kansas City residents are fully acclimatized to this regime, the weather/mortality algorithm developed for Kansas City can be utilized for New York City to account for full acclimatization if New York's weather approximates that predicted by GISS transient A_2.

One potential problem that arises from the utilization of weather analogs involves the possible difference in racial and socioeconomic composition between the evaluated city and its analog. The utilization of standard city death totals minimizes this problem.

Selection of analog cities

Present-day analogs to account for full acclimatization were selected for each city evaluated in this study for the three GISS transient scenarios for summer and winter, and mortality models were created for them using the procedure described earlier. Each analog was selected from a pool of almost 50 cities around the country (Table 4), representing virtually every weather regime found in the continental United States. This large sample size of cities permits an inter-regional evaluation of human weather/mortality responses on a scale larger than ever before.

The analog cities for summer were determined by comparing three weather variables (mean maximum temperatures, mean minimum temperatures, and the mean number of days with maximum temperatures over

Table 4. Cities Used as Potential Analogs

1. ALBUQUERQUE, NM	25. LOS ANGELES, CA
2. AMARILLO/LUBBOCK, TX	26. LOUISVILLE, KY
3. ATLANTA, GA	27. MEMPHIS, TN
4. BILLINGS/GREAT FALLS, MT	28. MIAMI, FL
5. BIRMINGHAM, AL	29. MILWAUKEE, WI
6. BOISE, ID	30. MINNEAPOLIS, MN
7. BOSTON, MA	31. NASHVILLE, TN
8. CASPER, WY	32. NEW ORLEANS, LA
9. CHARLOTTE, NC	33. NEW YORK, NY
10. CHICAGO, IL	34. NORFOLK, VA
11. CINCINNATI, OH	35. OKLAHOMA CITY, OK
12. CLEVELAND, OH	36. PHILADELPHIA, PA
13. DALLAS, TX	37. PHOENIX, AZ
14. DENVER, CO	38. PITTSBURGH, PA
15. DES MOINES, IA	39. PORTLAND, OR
16. DETROIT, MI	40. ROCHESTER, NY
17. EL PASO, TX	41. ST. LOUIS, MO
18. FARGO, ND	42. SALT LAKE CITY, UT
19. INDIANAPOLIS, IN	43. SAN ANTONIO, TX
20. JACKSON, MS	44. SAN DIEGO, CA
21. JACKSONVILLE, FL	45. SAN FRANCISCO, CA
22. KANSAS CITY, MO, KS	46. SEATTLE, WA
23. LAS VEGAS, NV	47. SPOKANE, WA
24. LITTLE ROCK, AR	48. WICHITA, KS

90°F) for the three summer months (June, July, and August). This process was repeated using mean maximum temperatures, mean minimum temperatures, and the mean number of days with maximum temperatures below 32°F for the three winter months (December, January, and February) to determine winter analog cities. The statistical procedures used to determine the closest analog are described in detail within another manuscript (Kalkstein, in press), and these techniques produced analogs which were very close to the estimated future climate of the target cities.

Figure 4 illustrates the hypothetical differences expected in mortality with full, partial, and no acclimatization. It is probable that the acclimatized models (based on warmer city analogs) will show smaller increases in mortality than the unacclimatized models since residents have already adapted to the increased warmth. Thus, for warming scenarios of seven or more degrees, the differences in predicted deaths between full and no acclimatization situations may be very large (area hatched between lines 1 and 2). It is obviously necessary to consider a situation where partial acclimatization will be a likely result. It is possible that people will fully acclimatize to the increased warmth, but even if the population is capable of full behavioral acclimatization, it will take many years for the physical structure of the city to conform to the hotter climate (eg. total air conditioning of dwellings, construction of new structures with heating/cooling systems capable of meeting the demands of the new climate). It is improbable that people will not acclimatize at all to the increased warmth, as a majority of previous research rejects the notion that the population cannot acclimatize at least partially to changing weather conditions. For example, although it appears probable that people might adapt very well to the predicted warming, the urban structures will not be changed within the next 70 years to reflect the type of architecture best suited for the warmer climate. Today most poor inner city southerners reside in small single family dwellings which have adequate ventilation and often reflective aluminum roofs. However, their counterparts in large northeastern and midwestern cities live in row homes constructed of brick and possessing black roofs which readily absorb solar radiation. These structures become much hotter during extreme weather, and it is doubtful that the architectural makeup of these northern urban areas will change quickly enough to adapt to the predicted increasing warmth. Thus, a third estimate reflecting partial acclimatization (reflecting full acclimatization among the population but little change in the urban infrastructure) is included in Figure 4, with values intermediate between the full and no acclimatization possibilities.

In certain cases, it is possible that no extra deaths will be predicted for full acclimatization, as residents are conditioned to hot weather. For example, in Jacksonville, Florida, heat waves appear to produce no extra deaths (Figure 5); the relationship is so poor that it is virtually impossible to determine a threshold temperature.

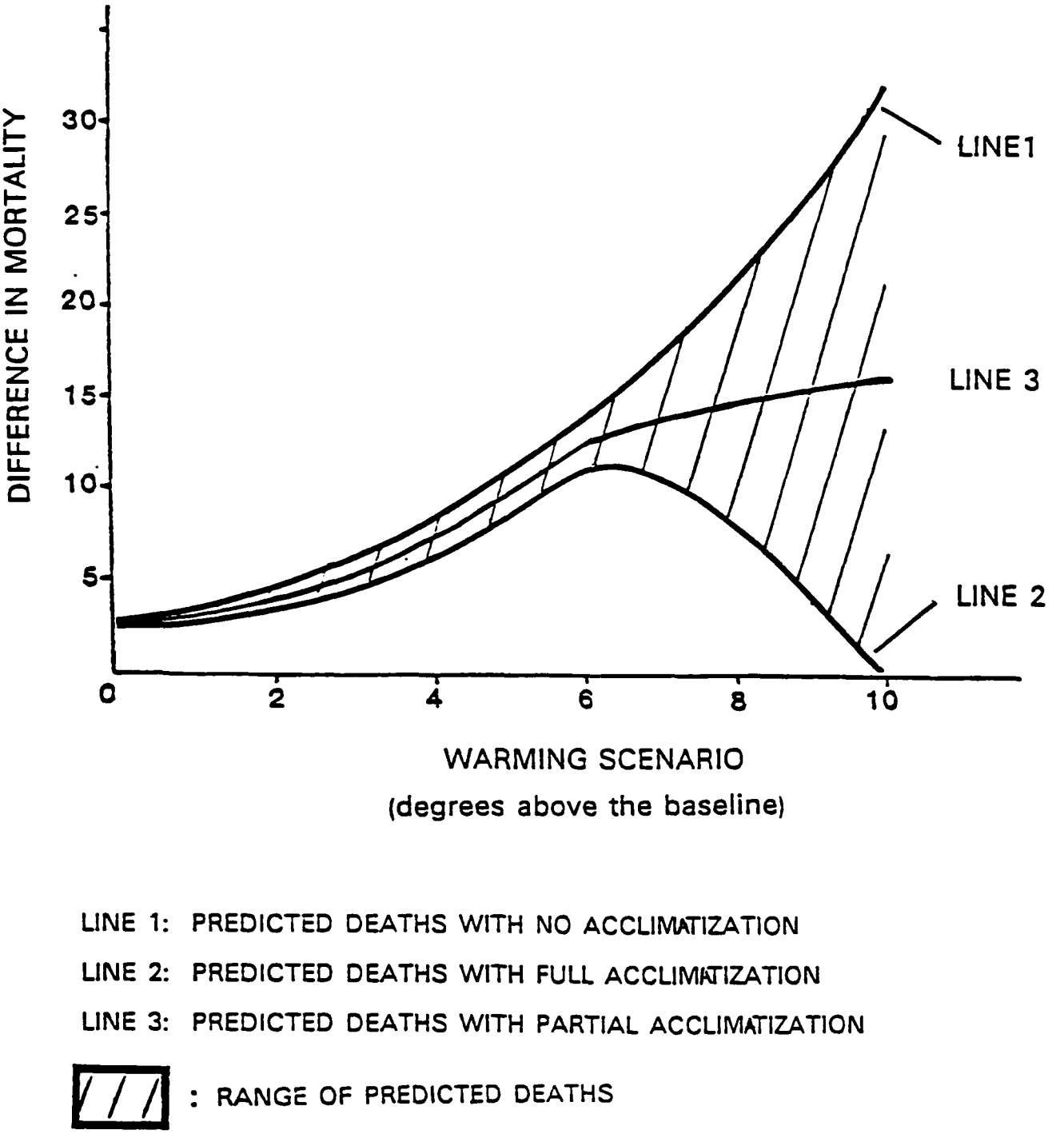


Figure 4. Expected increases in mortality in the target city for different warming scenarios.

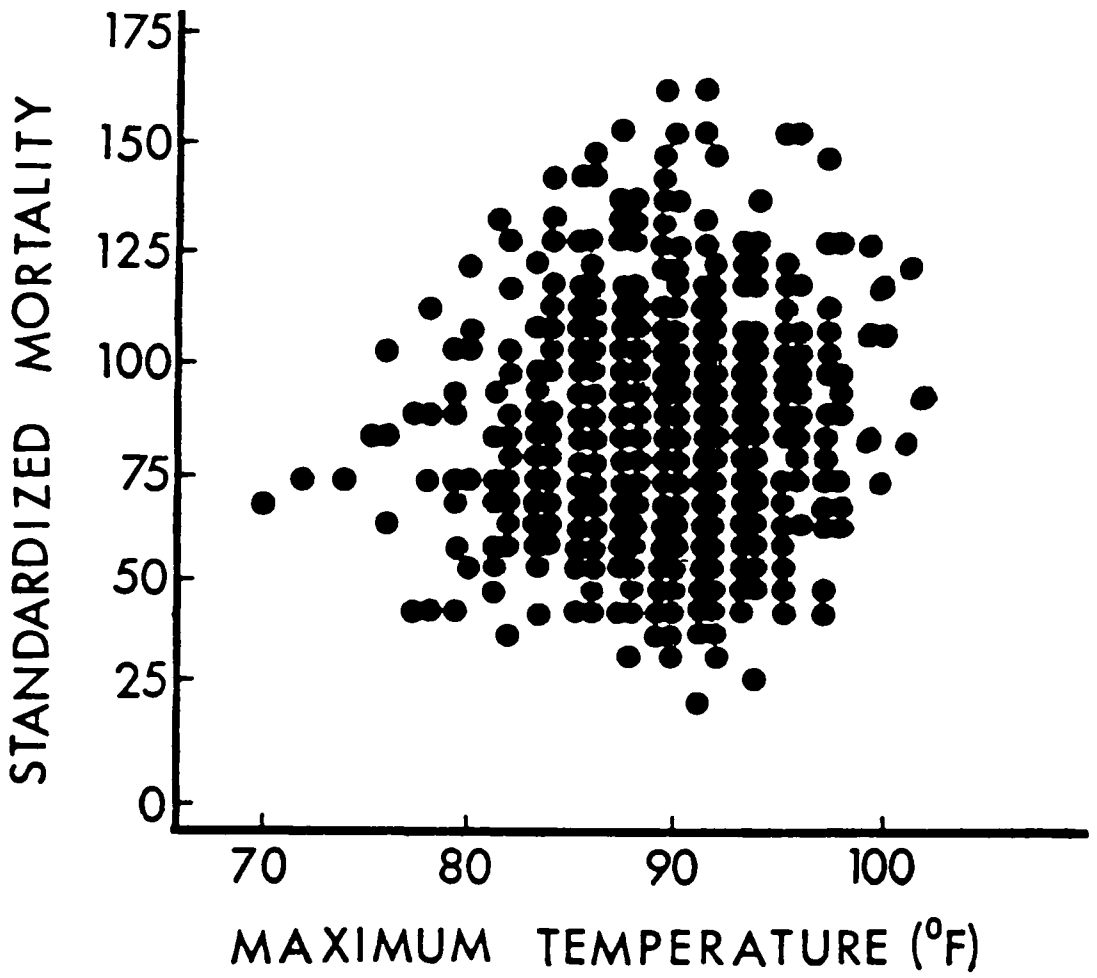


Figure 5. Daily summer-season standardized mortality vs. maximum temperature: Jacksonville.

CHAPTER 3

RESULTS AND DISCUSSION

HISTORICAL RELATIONSHIPS

Threshold temperatures were established for each of the 15 cities for total deaths and deaths among the elderly for the summer and winter seasons (Table 5). The threshold temperatures varied predictably between cities, and in the summer the southern and southwestern cities demonstrated the highest threshold temperatures. Similar findings were uncovered for threshold temperatures in the winter. Although there was considerable variation in threshold temperatures between cities, very little variation was detected between the two death categories within cities. For example, the summer threshold temperature in Atlanta was 94°F for the total and elderly death categories. It does not appear that any particular age group exhibits a distinctively high or low threshold temperature.

Very little lag time was noted between the weather mechanism and associated mortality response for all cities in summer (Table 5). In most cases, the mortality response occurred on the same day as the responsible weather mechanism (lag time = 0 days), although one-day lag responses were detected in some of the models. In winter, however, longer lag times were often noted, and for some cities the mortality response occurred three days after the responsible weather mechanism.

Once the threshold temperatures were established, the "all regression" procedure was performed for days above the threshold to determine the weather elements having the greatest impact on present-day mortality in each city. A large number of statistically significant models were uncovered for both seasons and for both death categories. Many of the relationships were more impressive than expected, with R^2 values frequently exceeding 0.250, especially in summer. During the warm season the most important weather variables proved to be CDH and TIME, which were directly and inversely related to mortality, respectively. The inverse TIME relationship suggests that the timing of the weather event is often as important as the magnitude. Hot weather occurring early in the season appears to have a more devastating impact than similar weather occurring in August, implying that acclimatization to hot weather might occur rapidly within a season. The importance of CDH and relative insignificance of maximum temperature are also noteworthy. This suggests that the intensity of the heat event might be of lesser importance than the duration of the event. Winter relationships appear to be substantially weaker than those in summer, and thermal variables (MAXT, MINT, HDH) appear to be much less important. Refer to Kalkstein, in press, for a more complete explanation.

Although similar numbers of statistically significant models were found for "all causes" and "weather-related" causes of death, the level of significance varied considerably between the two in both seasons. The proportion of statistically significant models with R^2 values exceeding 0.250 was much higher for the all causes category in summer. When significant all causes and weather-related models were uncovered for the same category within a city, the all causes model usually possessed the higher R^2 value. This is consistent with the findings of past studies on summer mortality as described previously, and the results of the all causes, rather than weather-related, models will be used exclusively here for the summer season. The winter season produced opposite results, and the "weather-related" category normally possessed a higher R^2 value than its "all causes" counterpart; thus the former will be emphasized here for the winter season.

The historical evaluation of mortality produced very interesting geographical distributions, indicating that the impact of weather on mortality varies considerably on an inter-regional level. For example, people living in the northern part of the country appear much more susceptible to heat-related mortality than those in the South. For a full discussion of these inter-regional variations, refer to Kalkstein (in press).

The algorithms developed through the regression analyses were employed to estimate the number of deaths attributed to the weather for each of the 15 evaluated cities. In addition, the use of future weather scenarios assuming long-term climatic warming permitted application of the algorithms to predict future trends in mortality. Both unacclimatized (using the historical algorithm for a city to predict future mortality in that city) and acclimatized (using the algorithms for analog cities to predict future mortality) estimates will be presented,

Table 5. Threshold Temperatures and Lag Times For the Fifteen Cities in Summer and Winter[a]

City	Summer				Winter			
	Total Deaths		Elderly Deaths		Total Deaths[b]		Elderly Deaths[b]	
	Threshold	Lag	Threshold	Lag	Threshold	Lag	Threshold	Lag
Atlanta	94°F	0	94°F	0	36°F	3		
Chicago	91	0	91	0	15	0	15	0
Cincinnati	92	0	93	1	18	3	18	3
Dallas	103	0	103	0	34	0	39	1
Detroit	90	0	90	1	16	2		
Kansas City	99	0	99	0	19	1	22	0
Los Angeles	81	0	82	0			57	2
Memphis	99	0	99	0	29	3	29	3
Minneapolis	93	1	93	0	1	1	4	3
New Orleans								
New York	92	1	93	1	27	0	24	2
Oklahoma City			104	1				
Philadelphia	92	1	92	1	24	1	24	1
St. Louis	96	1	98	1	22	3		
San Francisco	84	1	81	1	48	0	48	0

[a] If no value exists, the model was not statistically significant at the 0.05 level.

[b] Only "weather-related" models were utilized for winter since these provided better relationships than their "all causes" counterparts.

using the GISS warming scenarios described earlier. A statistically significant model at the 0.05 level or better was required if any estimate was attempted. If no statistically significant model was uncovered, it was assumed that weather has no impact on mortality, and a value of 0 deaths was assigned.

Analog cities were developed for summer and winter for each city to develop acclimatized predictions (Tables 6-7). In certain cases, the change in climate was so small (i.e., the GISS Transient A_1 run) that a city could be an analog of itself (refer to the Dallas summer analog).

SUMMER PREDICTIONS

Initially, an estimate of present-day mortality attributed to weather was attempted by utilizing the created regressions to develop historical algorithms for the 15 cities (Table 8). Using the algorithms, mortality was estimated for every day having temperatures above the threshold in the 11-year sample. These mortality estimates were compared to the 11-year mean mortality for the month, and the difference between the estimate derived from the algorithm and the long-term monthly mean was considered to be the day's weather-induced mortality. For example, assume that June 1, 1980, experienced temperatures above the threshold in Cincinnati. The historical algorithm estimated that 130 deaths occurred in Cincinnati on that day. The 11-year June mean mortality for Cincinnati is 115 deaths. Thus, 15 deaths were attributed to weather in Cincinnati on June 1.

The deaths attributed to weather were summed daily for all days above the threshold temperature for each month over the 11-year period. The average monthly mortality was extracted to represent the expected mortality attributed to weather during an average month in the evaluated city. Seasonal averages were derived from the monthly values.

For total deaths, it appears that New York experiences the greatest mortality totals, amounting to 320 deaths from all causes during an average summer attributed to weather. Chicago and Philadelphia ranked second and third, respectively, both averaging well over 100 standard city deaths during an average summer. Lowest values were found in New Orleans and Oklahoma City, where 0 deaths were attributed to the weather. During an average summer season over 1150 standard city deaths attributed to weather are estimated to occur in the SMSAs of the 15 cities. Of course this figure is much higher during extremely hot summers.

July is by far the most significant month, accounting for approximately two-thirds of all mortality in summer attributed to weather. Although August is generally much warmer than June in most of the cities, mortality for both months is quite similar, and in some cases, June's predicted mortality exceeds that of August. This reflects the importance of within-season acclimatization (Kalkstein, in press), indicating that early-season (June) heat waves generally exert a greater impact than late-season (August) heat waves of similar magnitude.

The evaluation of predicted future unacclimatized, acclimatized, and partially acclimatized mortality in summer based on the warming scenarios yielded interesting results (Table 9). Virtually all cities exhibited an increase in mortality as the scenarios became warmer for both total and elderly deaths, and the total unacclimatized mortality estimate exceeded 7400 attributed to weather during an average summer under $2XCO_2$ conditions. This is almost seven times higher than the number of deaths attributed to weather under average summer conditions today. However, the magnitude of the increase varied significantly between cities. New York continued to be the city with the greatest number of deaths through most of the warming scenarios, with over 1700 deaths attributed to weather during an average summer under $2XCO_2$ conditions. Other cities with predicted rapid rises in mortality with future warming were Los Angeles, Memphis, Philadelphia, and New York. New Orleans and Oklahoma City, with statistically non-significant mortality models, will probably not be affected significantly by future warming.

Estimates of future acclimatized mortality indicate that predicted warming might lessen weather-induced mortality in certain cities if acclimatization proceeds rapidly. In Atlanta, Detroit, Los Angeles, Memphis, New

Table 6. Summer Analogs for the Fifteen Cities Using the Three Warming Scenarios

Target City	Analog Cities		
	GISS Trans A_1	GISS Trans A_2	GISS $2XCO_2$
1. Atlanta	Kansas City	Wichita	New Orleans
2. Chicago	Des Moines	St. Louis	St. Louis
3. Cincinnati	Philadelphia	Nashville	Birmingham
4. Dallas	Dallas	Phoenix	Phoenix
5. Detroit	Minneapolis	Omaha	Omaha
6. Kansas City	Omaha	Memphis	Memphis
7. Los Angeles	San Diego	Pittsburgh	Norfolk
8. Memphis	Nashville	New Orleans	San Antonio
9. Minneapolis	Des Moines	Nashville	St. Louis
10. New Orleans	New Orleans	San Antonio	San Antonio
11. New York	Norfolk	Kansas City	Kansas City
12. Oklahoma City	Dallas	Dallas	Dallas
13. Philadelphia	Norfolk	Kansas City	Birmingham
14. St. Louis	Wichita	Memphis	Jacksonville
15. San Francisco	Seattle	Seattle	Pittsburgh

Table 7. Winter Analogs for the Fifteen Cities Using the Three Warming Scenarios

Target City	Analog Cities		
	GISS Trans A_1	GISS Trans A_2	GISS $2XCO_2$
1. Atlanta	Atlanta	Dallas	Dallas
2. Chicago	Rochester	Indianapolis	Philadelphia
3. Cincinnati	Cincinnati	Louisville	Louisville
4. Dallas	Dallas	Houston	New Orleans
5. Detroit	Cleveland	Pittsburgh	New York
6. Kansas City	Indianapolis	Louisville	Oklahoma City
7. Los Angeles	Los Angeles	Los Angeles	Miami
8. Memphis	Memphis	Dallas	New Orleans
9. Minneapolis	Minneapolis	Milwaukee	Chicago
10. New Orleans	New Orleans	San Diego	Los Angeles
11. New York	New York	Washington, DC	Norfolk
12. Oklahoma City	Little Rock	Birmingham	Jackson
13. Philadelphia	New York	Washington, DC	Norfolk
14. St. Louis	Philadelphia	Louisville	Memphis
15. San Francisco	San Francisco	San Francisco	San Diego

Table 8. Estimates of Total Present-Day Mortality Attributed to Weather During an Average Summer Season

Rank	City	Mortality			
		June	July	August	Total
1	New York	45	217	58	320
2	Chicago	44	98	31	173
3	Philadelphia	35	59	51	145
4	Detroit	21	67	30	118
5	St. Louis	1	80	32	113
6	Los Angeles	19	30	35	84
7	Minneapolis	9	27	10	46
8	Cincinnati	9	21	12	42
9	Kansas City	0	28	3	31
10	San Francisco	12	10	5	27
11	Memphis	0	18	2	20
12	Dallas	6	8	5	19
13	Atlanta	8	10	0	18
14	New Orleans	0	0	0	0
15	Oklahoma City	0	0	0	0

TABLE 9. ESTIMATES OF FUTURE MORTALITY IN SUMMER ATTRIBUTED TO WEATHER

City	Present Total	GISS Non-Acclim	TRANS Partial Acclim	A^1 Full Acclim	GISS Non-Acclim	TRANS Partial Acclim	A^2 Full Acclim	GISS 2x CO_2 Non-Acclim	2x CO_2 Partial Acclim	2x CO_2 Full Acclim	Change: Present to 2 x CO_2 Non-Acclim	Change: Present to 2 x CO_2 Partial Acclim	Change: Present to 2 x CO_2 Full Acclim
Atlanta													
Elderly	9	25	13	0	63	32	0	85	43	0	76	34	-9
Total	18	45	23	0	118	59	0	159	79	0	141	61	-18
Chicago													
Elderly	104	173	86	0	300	408	520	240	347	458	136	243	354
Total	173	295	145	0	511	725	940	412	622	835	239	449	662
Cincinnati													
Elderly	28	62	52	45	127	63	0	150	123	69	122	95	41
Total	42	93	83	72	195	97	0	226	195	116	184	153	74
Dallas													
Elderly	9	33	33	33	109	68	28	172	153	74	163	144	65
Total	19	61	61	61	197	213	67	309	244	179	290	225	160
Detroit													
Elderly	70	120	91	0	301	151	0	349	174	0	279	104	-70
Total	118	201	152	104	512	254	0	592	295	0	474	177	-118
Kansas City													
Elderly	20	20	24	27	41	16	49	38	66	18	93	46	73
Total	31	33	40	48	66	68	71	60	100	138	29	69	107
Los Angeles													
Elderly	53	94	50	7	194	132	71	1026	513	0	973	460	-53
Total	84	153	81	11	313	205	116	1654	824	0	1570	740	-84

Table 9. (page 2)

City	Present Total	GISS TRANS A[1] Non-Acclim	GISS TRANS A[1] Partial Acclim	GISS TRANS A[1] Full Acclim	GISS TRANS A[2] Non-Acclim	GISS TRANS A[2] Partial Acclim	GISS TRANS A[2] Full Acclim	GISS 2x CO_2 Non-Acclim	GISS 2x CO_2 Partial Acclim	GISS 2x CO_2 Full Acclim	Change: Present to 2 x CO_2 Non-Acclim	Change: Present to 2 x CO_2 Partial Acclim	Change: Present to 2 x CO_2 Full Acclim
Memphis													
Elderly	13	18	9	0	47	24	0	106	52	0	93	39	-13
Total	20	28	14	0	78	39	0	177	88	0	157	68	-20
Minneapolis													
Elderly	30	65	32	0	118	59	0	95	110	126	65	80	96
Total	46	96	47	0	175	87	0	142	186	235	96	140	189
New Orleans													
Elderly	0	0	0	0	0	0	0	0	0	0	0	0	0
Total	0	0	0	0	0	0	0	0	0	0	0	0	0
New York													
Elderly	212	514	257	0	897	448	6	1139	580	17	927	368	-195
Total	320	777	386	0	1375	689	8	1743	880	23	1423	560	-297
Oklahoma City													
Elderly	0	0	4	8	0	9	19	0	16	31	0	16	31
Total	0	0	6	12	0	19	29	0	23	47	0	23	47
Philadelphia													
Elderly	91	182	89	0	489	244	0	590	590	285	499	499	194
Total	145	288	142	0	778	388	0	938	700	466	793	555	321
St. Louis													
Elderly	71	203	102	0	471	373	278	459	226	0	388	155	-71
Total	113	325	162	0	754	564	375	744	372	0	631	259	-113

Table 9. (page 3)

City	Present Total	GISS Non-Acclim	TRANS Partial Acclim	A[1] Full Acclim	GISS Non-Acclim	TRANS Partial Acclim	A[2] Full Acclim	GISS 2x CO_2 Non-Acclim	2x CO_2 Partial Acclim	2x CO_2 Full Acclim	Change: Present to 2 x CO_2 Non-Acclim	Change: Present to 2 x CO_2 Partial Acclim	Change: Present to 2 x CO_2 Full Acclim
San Francisco													
Elderly	17	28	14	2	45	23	4	156	129	103	139	112	86
Total	27	44	23	3	71	38	7	246	202	159	219	175	132
Total													
Elderly	727	1537	856	122	3202	2050	975	4605	3122	1256	3878	2395	529
Total	1156	2439	1365	311	5143	3445	1613	7402	4810	2198	6246	3654	1042

Orleans, and St. Louis virtually no weather-induced deaths were predicted if the residents of these cities acclimatize. This is particularly surprising for St. Louis, where unacclimatized future mortality estimates and historical estimates were very high. In approximately half of the cities mortality totals declined as the scenarios became warmer, and in cities where increases did occur, they were much smaller than those uncovered under no acclimatization. The 15-city acclimatized totals showed a significant drop in mortality under conditions predicted in the early 21st century (GISS Transient A_1), followed by a relatively modest rise (when compared to unacclimatized results) for the mid 21st century (GISS Transient A_2). A more significant rise in acclimatized mortality is noted under $2XCO_2$ conditions, with values approximately double today's weather-induced mortality.

Estimates of future total mortality assuming partial acclimatization were calculated by computing the mortality totals exactly halfway between full and no acclimatization values. With partial acclimatization about 3800 standard city deaths are predicted for the 15-city sample under $2XCO_2$ conditions. This represents a substantial increase over present-day mortality estimates attributed to weather, and although these values should be viewed cautiously, indications are that mortality will increase substantially if partial acclimatization takes place.

Mortality estimates for the 65 and older category were very similar to the total mortality estimates. Substantial increases in unacclimatized mortality were noted in virtually all cities. Partial acclimatized values were less than unacclimatized, but a substantial increase of almost 2400 deaths above present conditions was noted for the 15-city sample under $2XCO_2$ conditions. Much like the results from the total death category, acclimatized mortality predictions for the 65 and older group were much smaller than the unacclimatized values.

The great disparity between acclimatized and unacclimatized predictions is troubling but not surprising. If people do not acclimatize to predicted warming, weather-induced mortality will rise at a very rapid rate because (1) the number of days exceeding the threshold temperature will increase, providing a larger group of total days when weather-induced mortality will be a factor; and (2) since CDH and maximum temperature are directly related to mortality in almost all the summer models, mortality will necessarily increase as the magnitudes of these weather variables increase. If acclimatization is complete, increasing warmth will produce a much smaller rise in mortality, which parallels the response of people today who reside in hot climates. In almost all of the southern cities in our 15-city sample, present-day weather-induced mortality was estimated to be lower than in the northern cities. These southern cities represent analogs of expected climate in the northern cities, and the lower present-day mortality rates indicate that southerners have indeed acclimatized to the frequent hot weather episodes that occur.

WINTER PREDICTIONS

The following winter mortality predictions are for weather-related causes of death only. Mortality predictions were also constructed for all causes of death, but these models generally possessed lower R^2 values than their weather-related counterparts. Thus a decision was made to use the weather-related predictions to ensure a more accurate result.

Present-day estimates of winter mortality indicate that weather-induced deaths are much less important in winter than in summer (Table 10). The city with the greatest predicted number of winter deaths, New York, averages only 56 standard city deaths per season. About half of the cities in the 15-city sample produce 10 or less weather-induced winter deaths during an average season. In some cases, statistically significant models were developed for winter for these cities, but the relationships were relatively weak, yielding mortality predictions that approached 0.

For total deaths, the four top ranking cities are all northern or midwestern locations. New York, St. Louis, Chicago, and Kansas City account for 170 of the 296 total weather-induced deaths which occur in the 15-city sample during a typical winter. The occasional severe cold waves encountered here obviously have a significant impact. Surprisingly, Minneapolis mortality totals are quite low compared to Midwestern cities located farther south. It is possible that residents of Minneapolis are so conditioned to winter cold that they are

Table 10. Estimates of Total Present-Day Mortality Attributed to Weather During an Average Winter Season

Rank	City	Mortality			
		January	February	December	Total
1	New York	38	10	8	56
2	St. Louis	15	20	12	47
3	Chicago	16	25	5	46
4	Kansas City	5	12	4	21
5	Dallas	3	10	3	16
6	Detroit	2	8	16	16
7	Cincinnati	3	8	3	14
8	San Francisco	8	1	1	10
9	Philadephia	10	0	0	10
10	Minneapolis	2	2	1	5
11	Atlanta	0	0	2	2
12	Los Angeles	0	0	0	0
13	Memphis	0	0	0	0
14	New Orleans	0	0	0	0
15	Oklahoma City	0	0	0	0

behaviorally adapted and take the proper precautionary steps to avoid cold weather impacts. The five cities which record the lowest number of weather-induced winter deaths are located in milder climates. In a previous study Los Angeles and San Francisco never exhibited winter conditions severe enough to produce weather-induced deaths (Kalkstein and Davis, 1985). However, the other southern cities recording few deaths do experience occasional cold waves, especially Atlanta, Memphis, and Oklahoma City. Perhaps the cold episodes are of too short a duration or are not extreme enough to create problems.

The sensitivity of the elderly to winter-induced mortality is surprisingly low, and only 157 elderly deaths attributed to weather are predicted during an average winter. Once again, the cities with the greatest predicted elderly mortality are located in northern or midwestern locations, while a majority of the low ranking cities are found in milder climates.

These results seem to counter some of the findings in the summer models regarding the relative impact of weather. During summer, many of the cities exhibiting the highest weather-induced mortality totals were located in the northern United States, where extreme heat occurs less often. There was a clear suggestion that people react to weather in a relative, rather than absolute, fashion in summer, as cities with low mortality estimates were found in some of the hottest regions in the country. During winter, the findings suggest that this relative response is less pronounced, as many of the cities with the highest winter mortality totals are located in regions with relatively severe winter climates, such as Chicago, Kansas City, New York, and St. Louis. Thus, the impact of weather on mortality in winter appears to be more absolute.

In both the total and elderly predictions for winter, February exhibits a surprisingly low number of winter weather-induced deaths. This occurs although February is normally colder than December; once again, an element of within-season acclimatization appears to be present and people are more sensitive to early cold waves than to later cold waves of similar (or greater) magnitude. Nevertheless, the TIME variable is less important in winter than in summer, and within-season acclimatization is less pronounced in winter.

Estimates of future winter mortality based on the warming scenarios were developed, producing unacclimatized, partially acclimatized, and acclimatized values for total deaths and elderly deaths. The results were very different than those uncovered in summer (Table 11). For total deaths assuming no acclimatization, a decline in weather-induced mortality was noted for most of the cities in the 15-city sample as the scenarios became warmer. Of the 15 cities, 10 registered five deaths or less under $2XCO_2$ conditions. These results differ markedly from summer, where unacclimatized total mortality showed a dramatic rise as the scenarios became warmer. The winter results suggest that a warmer climate will correspond with a lesser number of days below the threshold temperature, and the potential for weather-induced mortality will decrease correspondingly.

If full acclimatization is assumed, the impact of future warming on mortality also creates a pronounced drop. The lack of acclimatized mortality with increased warmth is consistent with previous winter findings, and mortality in many of the southern cities (which constitute the analogs for the warmer weather scenarios) is not significantly affected by winter weather. Values for partial acclimatization are similar to those for full acclimatization, and very few deaths are predicted under $2XCO_2$ conditions. Elderly response with climatic warming is very similar to the total population response.

TABLE 11 ESTIMATES OF FUTURE MORTALITY IN WINTER ATTRIBUTED TO WEATHER

City	Present	GISS	TRANS	A1	GISS	TRANS	A2	GISS	2x	CO_2	Change: Present to 2 x CO_2		
	Total	Non-Acclim	Partial Acclim	Full Acclim	Non-Acclim	Partial Acclim	Full Acclim	Non-Acclim	Partial Acclim	Full Acclim	Non-Acclim	Partial Acclim	Full Acclim
Atlanta													
Elderly	1	2	1	2	1	0	0	2	0	0	1	-1	-1
Total	2	3	2	2	2	1	0	2	0	0	0	-2	-2
Chicago													
Elderly	30	15	5	0	5	2	0	0	27	56	-30	-3	26
Total	46	20	9	0	7	2	0	2	48	96	-44	2	50
Cincinnati													
Elderly	9	5	5	5	3	2	0	4	2	0	-5	-7	-9
Total	14	7	7	7	4	2	0	6	3	0	-8	-11	-14
Dallas													
Elderly	10	8	8	8	1	0	0	0	0	0	-10	-10	-10
Total	16	14	14	14	4	1	0	1	0	0	-15	-16	-16
Detroit													
Elderly	10	3	1	0	1	0	0	2	8	18	-8	-2	8
Total	16	5	2	0	2	0	0	2	18	37	-14	2	21
Kansas City													
Elderly	14	15	7	0	9	4	0	4	1	0	-10	-13	-14
Total	21	20	10	0	13	6	0	5	2	0	-16	-19	-21
Los Angeles													
Elderly	0	0	0	0	0	0	0	0	0	0	0	0	0
Total	0	0	0	0	0	0	0	0	0	0	0	0	0

Table 11. (page 2)

City	Present Total	GISS TRANS A1 Non-Acclim	GISS TRANS A1 Partial Acclim	GISS TRANS A1 Full Acclim	GISS TRANS A2 Non-Acclim	GISS TRANS A2 Partial Acclim	GISS TRANS A2 Full Acclim	GISS 2x CO_2 Non-Acclim	GISS 2x CO_2 Partial Acclim	GISS 2x CO_2 Full Acclim	Change: Present to 2 x CO_2 Non-Acclim	Change: Present to 2 x CO_2 Partial Acclim	Change: Present to 2 x CO_2 Full Acclim
Memphis													
Elderly	0	0	0	0	0	1	1	0	0	0	0	0	0
Total	0	0	0	0	1	1	2	0	0	0	0	0	0
Minneapolis													
Elderly	2	0	0	0	0	1	3	0	0	0	-2	-2	-2
Total	5	2	2	2	1	3	6	1	0	0	-4	-5	-5
New Orleans													
Elderly	0	0	0	0	0	0	0	0	0	0	0	0	0
Total	0	0	0	0	0	0	0	0	0	0	0	0	0
New York													
Elderly	37	28	28	28	34	14	0	11	14	23	-26	-23	-14
Total	56	46	46	46	46	21	0	18	21	25	-38	-35	-31
Oklahoma City													
Elderly	0	0	0	0	0	1	1	0	0	0	0	0	0
Total	0	0	0	0	0	1	3	0	0	0	0	0	0
Philadelphia													
Elderly	6	8	14	21	1	0	0	1	0	1	-5	-6	-5
Total	10	12	25	36	1	0	0	1	0	1	-9	-10	-9
St. Louis													
Elderly	31	21	12	3	9	4	0	5	2	0	-26	-29	-31
Total	47	30	18	5	13	6	0	7	3	0	-40	-44	-47

Table 11. (page 3)

City	Present Total	GISS A1 Non-Acclim	TRANS A1 Partial Acclim	TRANS A1 Full Acclim	GISS A2 Non-Acclim	TRANS A2 Partial Acclim	TRANS A2 Full Acclim	GISS 2x CO_2 Non-Acclim	2x CO_2 Partial Acclim	2x CO_2 Full Acclim	Change: Present to 2 x CO_2 Non-Acclim	Change: Present to 2 x CO_2 Partial Acclim	Change: Present to 2 x CO_2 Full Acclim
San Francisco													
Elderly	7	6	6	6	7	7	7	5	2	0	-2	-5	-7
Total	10	8	8	10	9	9	10	7	2	0	-3	-8	-10
Total													
Elderly	157	111	87	73	71	36	12	34	56	98	-123	-101	-59
Total	243	167	143	122	103	53	21	52	97	159	-191	-146	-84

CHAPTER 4

CONCLUSIONS

The objective of this study was to estimate changes in human mortality attributed to predicted changes in climate due to increased concentrations of CO_2 and other trace gases in the atmosphere. The major result was an estimation of the number of deaths attributed to the increased incidence of extreme weather episodes predicted by numerous climate change models.

The evaluation covered 15 cities around the country, and daily mortality data for 11 summer and winter seasons were extracted. The mortality totals were divided into total and elderly mortality categories, and separate evaluations were developed for all causes of death and those causes considered to be "weather-related."

A summary of the results follows.

1. Predictions of weather-induced mortality occurring during summer were attempted for the 15 cities exhibiting significant weather/mortality relationships. It is estimated that approximately 1150 deaths occur during an average summer season in the SMSAs of the 15 cities. New York City, Chicago, and Philadelphia ranked first, second, and third, respectively, and each city averaged well over 100 standard city deaths per summer. The five highest ranking cities were all found in the Midwest or Northeast. The five lowest ranking cities were found in the South, with New Orleans and Oklahoma City experiencing virtually no deaths attributed to weather in summer.

2. Predicted future unacclimatized mortality in summer rose rapidly as the scenarios become warmer. The total mortality estimate exceeded 7400 deaths attributed to weather during an average summer under $2XCO_2$ conditions. The magnitude of the increase varied significantly between cities. New York exhibited the greatest number of deaths throughout all the warming scenarios. Other cities with predicted rapid rises in mortality with future warming were Los Angeles, Philadelphia, and St. Louis. New Orleans and Oklahoma City were cities least affected by predicted warming.

3. Predicted future acclimatized mortality in summer indicated that warming might lessen weather-induced mortality in about half of the 15-city sample if acclimatization is complete. A more modest rise in mortality is predicted for the 15-city sample if full acclimatization occurs; however, weather-induced mortality is predicted to double over present levels if acclimatization is complete. Acclimatized mortality estimates for the 65+ age category were very similar to the total mortality estimates. By the design of the model, the relatively small rise in acclimatized mortality paralleled the response of people today who reside in hot climates. Southern cities represented analogs of expected climate in northern cities, and these cities exhibited fewer numbers of weather-induced deaths in summer. Estimates of future mortality assuming partial acclimatization (values midway between full and no acclimatization) were also developed, and these indicated sizable increases in mortality as the weather became warmer.

4. Present-day estimates of winter mortality indicated that weather-induced deaths were much less important in winter than in summer. The results differed from those uncovered in summer regarding the relative impact of weather. Most of the estimated winter deaths occurred in regions with relatively severe winter climates, while the smallest numbers of deaths were found in mild weather cities. It appeared that the impact of weather in winter is more absolute, while the impact of weather in summer tends to be relative.

5. Winter unacclimatized, partially acclimatized, and acclimatized predictions indicated that sharp drops in mortality are expected if the weather becomes warmer. The unacclimatized results differed

from those uncovered in summer, when dramatic rises in mortality were predicted. The unacclimatized drop in winter may be related to fewer numbers of days below the threshold.

It is quite obvious that predicted warming could have an enormous impact upon human health through the 21st century. If the population does not acclimatize, over 7000 deaths attributable to the increasingly harsh weather can be expected in the metropolitan areas of our 15-city sample. This figure is more startling when it is considered that these numbers correspond to average summer conditions. An analog of the very hot summer of 1980 occurring in the 21st century will no doubt increase weather-induced mortality to a much higher number than 7000.

Although fully acclimatized predictions are more modest, some general increases are still expected even under these conditions. If it is assumed that people will partially acclimatize (possibly the most realistic scenario), the increases in mortality are more impressive, and weather-related deaths will increase by four to five times over present levels. Thus it appears that specific policy decisions are necessary to prepare for a significant rise in human mortality if the warming scenarios come to pass.

REFERENCES

Applegate, W.B., Runyan, J.W., Jr., Brasfield, L., Williams, M.L., Konigsberg, C., and Fouche, C. Analysis of the 1980 heat wave in Memphis. Journal of the American Geriatrics Society. 29:337-342, 1981.

Bridger, C.A., Ellis, F.P., and Taylor, H.L. Mortality in St. Louis, Missouri, during heat waves in 1936, 1953, 1954, 1955, and 1966. Environmental Research. 12:38-48, 1976.

Centers for Disease Control. Exposure related hypothermia deaths--District of Columbia 1972-1982. In: Morbidity and Mortality Weekly Report. December, 1982. pp. 31-50.

Department of Health and Human Services. The International Classification of Diseases, 9th Revision, Clinical Modification, Volume 3: Procedures. Second edition, U.S. Dept. of Health and Human Services, September, 1980.

Draper, N., and Smith, H. Applied Regression Analysis, Second Edition. John Wiley and Sons, 1981. 673 pp.

Ellis, F.P. Mortality from heat illness and heat-aggravated illness in the United States. Environmental Research. 5:1-58, 1972.

Ellis, F.P., and Nelson, F. Mortality in the elderly in a heat wave in New York City, August, 1975. Environmental Research. 15:504-512, 1978.

Fitzgerald, F.T., and Jessop, C. Accidental hypothermia: A report of 22 cases and review of the literature. Advanced Internal Medicine. 27:127-150, 1982.

Gallow, D., Graham, T.E., and Pfeiffer, S. Comparative thermo-regulatory responses to acute cold in women of Asian or European descent. Human Biology. 56:19-34, 1984.

Gover, M. Mortality during periods of excessive temperatures. Public Health Reports. 53:112-1143, 1938.

Jenne, R. "GISS Transient Runs." NCAR/EPA Doc. 4, U.S. Environmental Protection Agency, Washington, DC, 1987, 3 pp.

Jones, T.S., Liang, Kilbourne, E.M., Griffin, M.R., Patriarca, P.A., Wassilak, G.G., Mullan,R.F., Herricck, R.F., Donnel, H.D., Jr., Choi, K., and Thacker, S.B. Morbidity and mortality associated with the July 1980 heat wave in St. Louis and Kansas City. Journal of the American Medical Associations. 247:3327-3330, 1982.

Kalkstein, L.S. The impact of winter weather on human mortality. In: Climate Impact Assessment: United States. U.S. Department of Commerce. December, 1984. pp. 21-23.

Kalkstein, L.S. The impact of CO_2 and trace gas-induced climate changes upon human mortality. U.S. EPA Office of Research and Development Report, in press.

Kalkstein, L.S., and Davis, R.E. The development of a weather/mortality model for environmental impact assessment. In: Proceedings of the 7th Conference of Biometeorology and Aerobiology, 1985. pp. 334-336.

Kalkstein, L.S., Davis, R.E., Skindlov, J.A., and K.M. Valimont. The impact of human-induced climatic warming upon human mortality: A New York City case study. Effects of Changes in Stratospheric Ozone and Global Climate, Volume 3: Climate Change. U.S. Environmental Protection Agency, October, 1986.

Kilbourne, E.M., Choi, K., Jones, T.S., Thacker, S.B., and the Field Investigation Team. Risk factors for heatstroke: A case-control study. Journal of the American Medical Association. 247:3332-3336, 1982.

Lilienfeld, A.M., and Lilienfeld, D.E. Chapter 4: Mortality statistics. Foundations of Epidemiology. Oxford University Press, 1980. pp. 51-65.

Lye, M., and Kamal, A. The effects of heat wave on mortality rates in elderly patients. The Lancet. 1:529-531, 1977.

Marmor, M. Heat wave mortality in New York City, 1949 to 1970. Archives of Environmental Health. 30:131-136, 1975.

Mausner, J.S., and Bahn, A.K. Chapter 7: Measures of morbidity and mortality. Epidemiology: An Introductory Text. W.B. Sanders Co., 1974.

Munn, R.E. The value of climatological information in assessments of the state of human health. Leningrad, U.S.S.R. In: Proceedings of the WMO/UNEP/WHO Symposium on Climate and Human Health. International Institute for Applied Systems Analysis, Luxenburg, Austria, 1986.

National Center for Health Statistics. Standardized Micro-Data Tape Transcripts. U.S. Department of Health, Education, and Welfare, Washington, DC, 1978.

Oechsli, F.W., and Buechley, R.W. Excess mortality associated with three Los Angeles September hot spells. Environmental Research. 3:277-284, 1970.

Rotton, J. Angry, sad, happy? Blame the weather. U.S. News and World Report. 95:52-53, 1983.

SAS Institute Inc. SAS User's Guide: Statistics; Version 5 Edition. SAS Institute Inc., 1985. 956 pp.

Steadham, R.G. The assessment of sultriness: Part I: A temperature-humidity index based on human physiology and clothing science. Journal of Applied Meteorology. 18:861-873, 1979.

Ulrich, R.S. View through a window may influence recovery from surgery. Science. 224:420-421, 1984.

Warren, H.E., and LeDuc, S.K. Impact of climate on energy sector in economic analysis. NOAA/Center for Environmental Assessment Services, Environmental Data and Information Service: United States. 1981. pp. 1431-1439.

White, M.R., and Hertz-Picciotto, I. Human health: Analysis of climate related to health. In: Characterization of Information Requirements for Studies of CO_2 Effects: Water Resources, Agriculture, Fisheries, Forests, and Human Health. M.R. White, ed., DOE/ER-0236, CO_2 Res. Div., U.S. Dept. of Energy, Washington, DC, 1985, pp. 172-205.

Willmot, C.F., Ackleson, S.G., Davis, R.E., Feddema, J.J., Klink, K.M., Legates, D.R., O'Donnell, J., and Rowe, C.M. Statistics for the evaluation and comparison of models. Journal of Geophysical Research. 90:8995-9005, 1985.

COMPUTER SIMULATION OF THE EFFECTS OF CHANGES IN WEATHER PATTERNS ON VECTOR-BORNE DISEASE TRANSMISSION

by

D. G. Haile
Insects Affecting Man and Animals Research Laboratory
Agricultural Research Service
U. S. Department of Agriculture
Gainesville, FL 32604

Project No. DW12932662-01-1

CONTENTS

FINDINGS[1]

Two weather-based models for simulation of the population dynamics of disease vectors were used to assess the possible effects of climate change due to increased levels of CO_2 in the atmosphere on vector-borne disease transmission in the United States. The first model (ADTSIM) simulates population dynamics of the American dog tick (ADT), Dermacentor variabilis, which is the primary vector of Rocky Mountain Spotted Fever (RMSF) in the eastern U. S. This model included the effects of temperature and atmospheric moisture on the life processes of the tick. The density of adult ticks was used as an indicator of RMSF transmission potential because this is the stage normally involved in transmission of human cases. The second model (MALSIM) simulates the population dynamics of Anopheline mosquitoes and the transmission of malaria between mosquitoes and humans. This model simulates direct incidence of malaria, assuming that the disease is reintroduced into a human population that is continuously exposed to mosquito bites. The effects of temperature, atmospheric moisture, and rainfall are included in MALSIM. The malaria vector considered in these simulations is Anopheles quadrimaculatus, which presently exists in many areas of the eastern half of the U. S. and was the primary vector of malaria in the south and east when the disease was endemic.

The ADTSIM results indicate that with the proposed climatic change scenarios at certain southern locations (Jacksonville, FL, and San Antonio, TX) ADT populations will disappear owing to adverse effects of high temperatures and low humidity, while at certain northern locations (Missoula, MT, North Bay, Ont., and Halifax, N.S.) populations will increase because of warming with adequate moisture. For most other U. S. locations, tick densities either declined moderately or remained the same with various weather scenarios. These results suggest that overall in the U. S. the problem of RMSF will decrease in certain areas, while others will remain unchanged.

The results of simulations of malaria transmission (assuming the disease is reintroduced into an unprotected population) show that there is little change in the transmission potential with modified weather scenarios. Areas in Florida with high transmission potential normally will remain high; very little increase was predicted for other areas.

Only the direct effects of weather on the disease vector were considered in these simulations. No consideration of the effects of weather on host densities or habitat were included. Consideration of these effects, along with other model refinements, would be necessary to improve confidence in the overall results. Also, much additional research on the biology, ecology, and modeling of these and other vector/disease complexes will be required for a more complete analysis of the impact of weather change on vector-borne diseases.

---

[1]Although the information in this report has been funded wholly or partly by the U.S. Environmental Protection Agency under Project Number DW12932662-01-1, it does not necessarily reflect the Agency's views, and no official endorsement should be inferred from it.

CHAPTER 1

INTRODUCTION

This study addresses the impact of climate change on vector-borne disease transmission in the United States as part of an overall study by the U. S. Environmental Protection Agency (EPA) the effects of climatic change resulting from the "greenhouse effect". Although at present there are no vector-borne diseases that are continuously transmitted to the human population in the United States, numerous arthropod vectors are present in the environment and the potential for major outbreaks exists in various regions as a result of increases in vector populations or prevalence of a disease.

Certain diseases are present in animal hosts, and others may be introduced from endemic areas by human immigrants or travelers. The arthropods most commonly involved in disease transmission in the United States involve various tick and mosquito species. The importance of weather variables on vector population dynamics and disease transmission has long been recognized (Harwood and James, 1979; Warren and Mahoud, 1984; Russell et al., 1963). Increases in temperature, up to an optimum level, normally increases the development and survival rate of vector and parasite life stages. Adequate atmospheric or soil moisture levels are important for survival of various vector stages and, in the case of mosquitoes, adequate levels of aquatic habitat are necessary for immature development.

Models have been used as a tool for understanding various aspects of vector population dynamics and disease transmission. In particular, the transmission of malaria has been studied extensively with mathematical models (Ross, 1911; MacDonald, 1957; Deitz et al., 1984; Molineaux and Grammiccia, 1980). Some efforts have been made to study mosquito and tick population dynamics using a computerized life history approach (Haile and Weidhaas, 1977; Fine et al., 1979; Sutherst et al., 1978; Sonenshine, 1975), but none of these efforts attempted to comprehensively model the effects of weather variables required for a quantitative assessment of climatic change. More recently, however, a comprehensive computer simulation model for population dynamics of the lone star tick, Amblyomma americanum, was developed, which included the direct effects of weather variables (Haile and Mount, 1987). This study was extended to develop a similar model for population dynamics of the American dog tick, Dermacentor variabilis, which is the principal vector of Rocky Mountain Spotted Fever (RMSF) in the U. S. (Mount and Haile, in press). In addition, the model for Anopheles albimanus by Haile and Weidhaas (1977) has been expanded to include the direct effects of weather variables and to allow simulation of malaria transmission to and from a human population with a variety of vector species (unpublished). These weather-sensitive models were used as the basis for the study on the impact of climatic change reported here. This report will describe the methodology used to simulate the effects of climatic change and an analysis of the implications of the results.

CHAPTER 2

METHODOLOGY

THE AMERICAN DOG TICK MODEL

The development and validation of the American dog tick model (ADTSIM) is reported in Mount and Haile (in press). Briefly, this model uses a dynamic life history approach with weekly age classes and time steps. Environmental variables used in the model include temperature, saturation deficit, daylength, host density, and habitat type. Relationships between environmental and biological variables include the following: (1) temperature-dependent development rates for eggs and engorged larvae, nymphs, and females; (2) the influence of temperature on fecundity; (3) the influence of habitat type, temperature, and saturation deficit on survival rates of free-living ticks; (4) the effect of host density, temperature, and daylength on host-finding rates; and (5) density-dependent survival of parasitic ticks during engorgement.

ADTSIM was developed to reflect the effects of average weekly weather data. These data may be actual values for a particular year or historical average data (values averaged over a period of years). As such the model was not designed to reflect the influence of extreme conditions on a diurnal or daily basis.

The effect of climate change in ADTSIM will indicate the direct effects of changes in ambient temperature and moisture on the tick life cycle with all other variables remaining constant. Any effect of climate change on habitat type and host densities are not simulated. An average of the number of adult ADT on hosts each week was used as an index to summarize each year of simulation. This index can also be used as an indicator of the transmission potential of RMSF because the adult tick is most often involved in transmission of the disease to humans.

Weather scenarios based on the results of three climate change models (GISS, GFDL, and OSU) that simulated the effects of doubling the level of CO_2 in the atmosphere, were used in the vector population models. Only simulations representing a step change in weather were used in this study. Actual data for the base period 1951-1980 were used for only one location (Richmond, VA) with ADTSIM. Historical average weather data for Richmond and other locations were used as the basis for evaluating the effect of the weather change models at each location. In this case the weekly weather data (normal or modified) were used in the population model each year until equilibrium was reached (usually ca. 15 years). A comparison of the equilibrium population with the population resulting from use of modified weather gave a direct measure of the impact of climate change.

THE MALARIA MODEL

A comprehensive model (MALSIM) to simulate the effects of environmental variables on population dynamics of Anopheline mosquitoes and on transmission of Falciparum malaria to a human population is currently being developed. Although this model is not fully validated, it contains the necessary relationships to provide a preliminary evaluation of the impact of climate change scenarios on this important disease.

Anopheles quadrimaculatus is the only malaria vector simulated in these studies. This species is still present in many areas of the eastern half of the United States and was the primary vector of malaria in the Southeast when the disease was endemic.

The following environmental relationships are included in MALSIM at present: (1) the effect of temperature on developmental rates of each vector stage and the parasite in the mosquito; (2) the effect of temperature on survival of immature stages; (3) the effect of temperature and saturation deficit on adult survival; (4) the relationship between rainfall, available area of aquatic habitat, immature density, and immature survival; and (5) temperature-induced hibernation of mated, nonblooded females.

Historical average weather data for a number of locations were used as the basis for simulations to compare the malaria transmission potential for normal vs. modified weather resulting from three climate change models (GISS, GFDL, and OSU). Although malaria is unlikely to become endemic in the United States, these simulations assume an introduction of 100 cases in an unprotected population of 10,000 along with initiation of mosquito population with 100,000 female adults. The level of transmission (incidence) in the 10,000 unprotected population after 3 years of simulation is used as an indication of transmission potential for comparative purposes.

CHAPTER 3

RESULTS

ADTSIM RESULTS

ADTSIM - Richmond, Virginia 1950-80

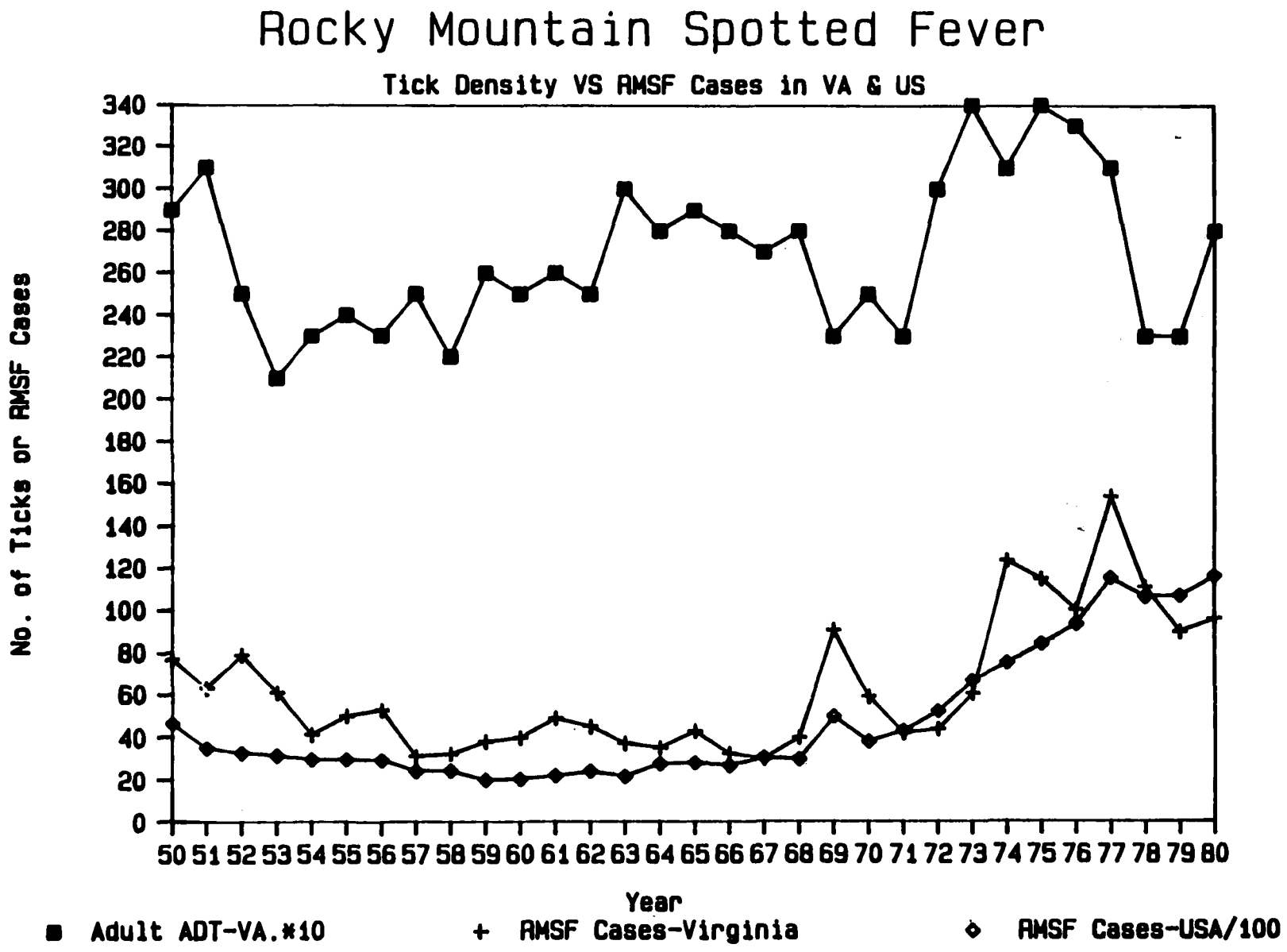
The simulated adult tick densities for the period 1950-1980 at Richmond with normal yearly weather data shows considerable variation between years as a result of weather alone (Figure 1). Cases of RMSF in Virginia and the United States (CDC data) are plotted in Figure 1 to show the correlation between simulated tick density and actual disease incidence. Figure 2 shows the comparison between the simulations with normal weather and the three models with modified weather. All modified weather scenarios produce a decrease in tick density for all years in the 30-year period. The OSU and GISS models show very similar results while the GFDL model produced a greater reduction. Comparisons of the average density with yearly data to the equilibrium density with historical average weather data showed that the results are very similar. Therefore, the equilibrium density method was used for evaluation of the impact of the modified weather scenarios at other locations.

ADTSIM Results at Various Locations

The equilibrium density of ADT adults on hosts at various U.S. locations and two Canadian locations with normal weather compared to the modified weather scenarios is shown in Figure 3. The results indicate a very clear shift in tick densities from south to north. The tick population was eliminated at the most southerly locations (Jacksonville and San Antonio) for all three modified weather scenarios. Simulations at Albuquerque showed no population with the normal dry weather and no change with any of the scenarios. Robust increases in tick density were indicated at the most northerly locations (Missoula, North Bay, and Halifax) for all scenarios. Missoula and North Bay had no tick population with normal weather and Halifax had only a low-level population. Simulations at U.S. locations where the normal tick density is relatively high produced a variety of results for the different scenario models. At some locations the three models produced similar results showing essentially no change in density at Los Angeles, Boston, and Medford or a moderate decrease at Richmond, New York, and Columbus. The results at Tulsa and Nashville indicated a decline in populations, with the OSU and GISS models predicting moderate declines and the GFDL model predicting population elimination. Simulation at Minneapolis gave the most mixed results between the different scenarios, with a population increase for GISS, no change for OSU, and population elimination for GFDL. In general, for locations where simulation indicated a moderate decline in population, the GFDL model showed the greatest decline.

RESULTS WITH MALSIM

Simulated levels of malaria transmission potential at various locations in the United States with normal weather and the three modified weather scenarios are presented in Figure 4. These results show that for the locations with the highest normal malaria transmission potential (Miami, Key West, and Orlando) there is very little change for any of the modified weather scenarios. For Jacksonville, which has a medium transmission level with normal weather, the results of the scenario models were mixed, with the GFDL model showing an increase, no change for GISS, and a decrease for OSU. As a practical matter, the changes at Jacksonville appear to be of little importance. At other locations with normally low transmission potential, only the GISS model appeared to result in significant increases in transmission potential. The GISS model produced increases at San Antonio, Atlanta, Nashville, and Richmond, with the largest increase at Atlanta. Simulations at Tulsa, Dallas, Baltimore, Indianapolis, and Boston indicated extremely low normal transmission with no significant change with any modified weather patterns. Overall, these simulations suggest that the proposed climatic changes will have little if any impact on transmission potential of malaria in the United States.



Haile

Figure 1. Graphs showing the yearly variation in simulated American dog tick density and the number of Rocky Mountain spotted fever cases in Virginia and in the U. S. with normal weather from 1950 to 1983.

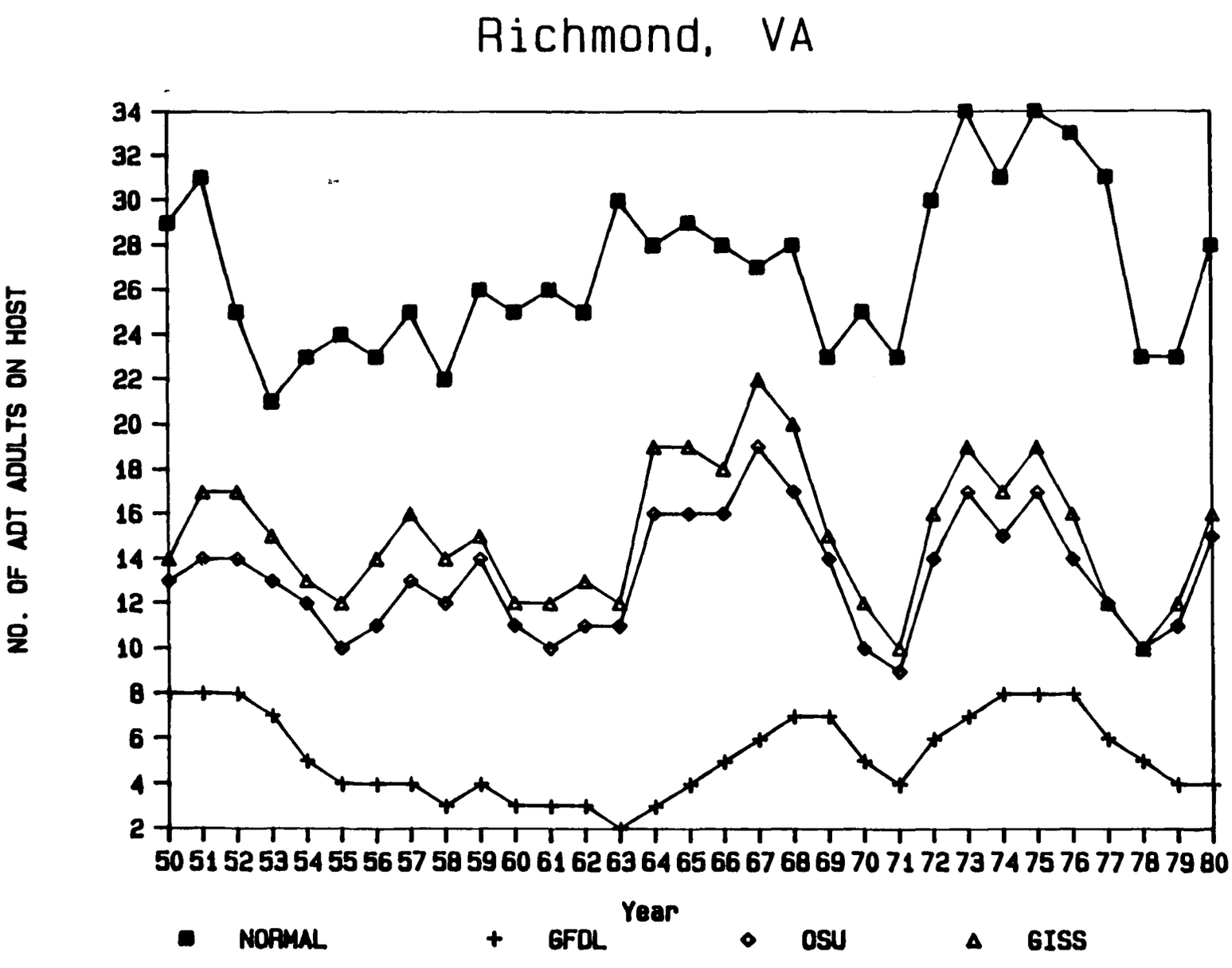


Figure 2. *Graphs showing the yearly variation in simulated American dog tick density at Richmond, VA, from 1950 to 1980 with normal GFDL, GISS and OSU weather scenarios.*

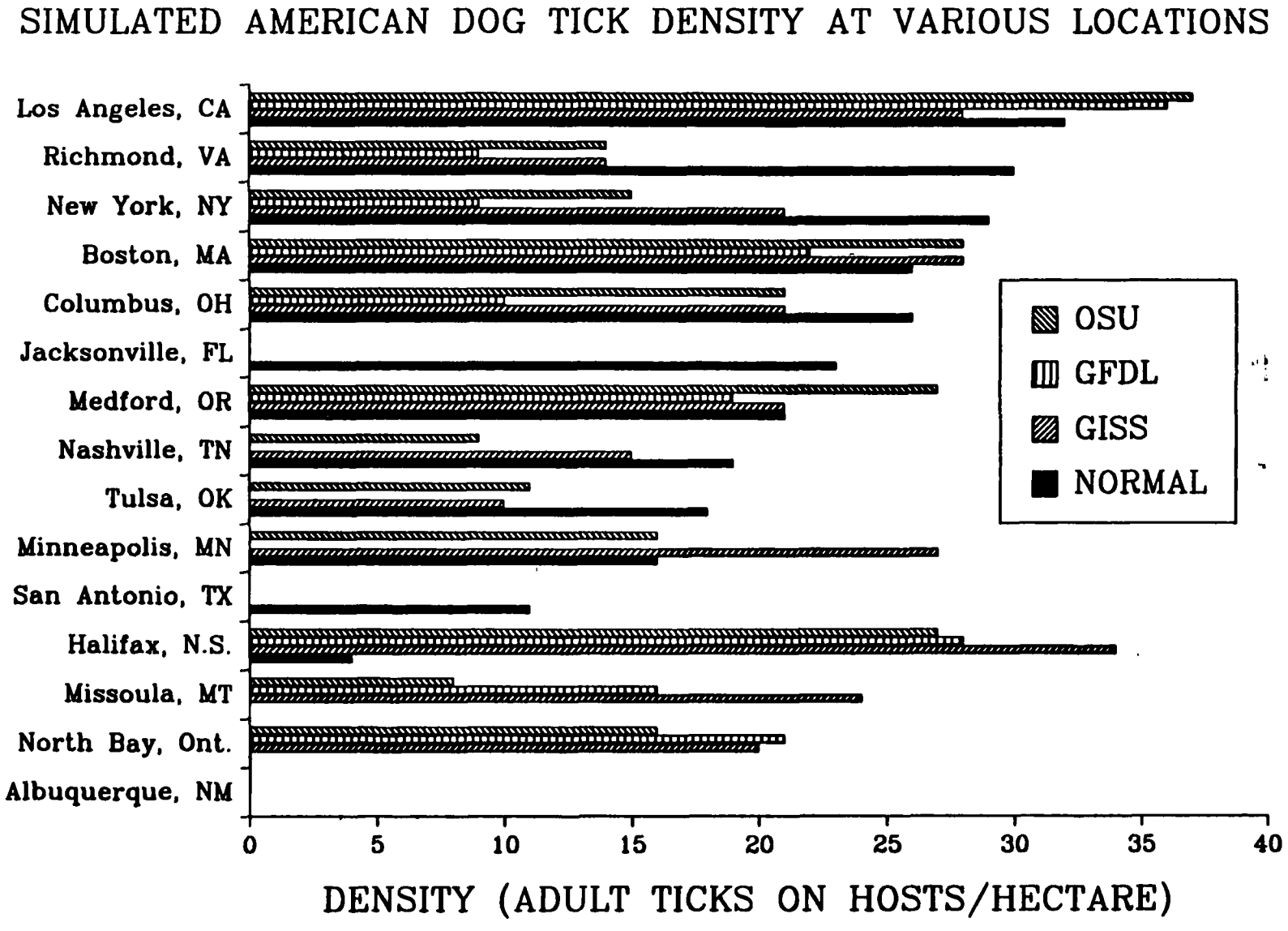


Figure 3. Simulated equilibrium density (after 15 years of simulation with constant weather) of American dog ticks at different locations with average, normal weather data and modified weather data for each climate change scenario.

Haile

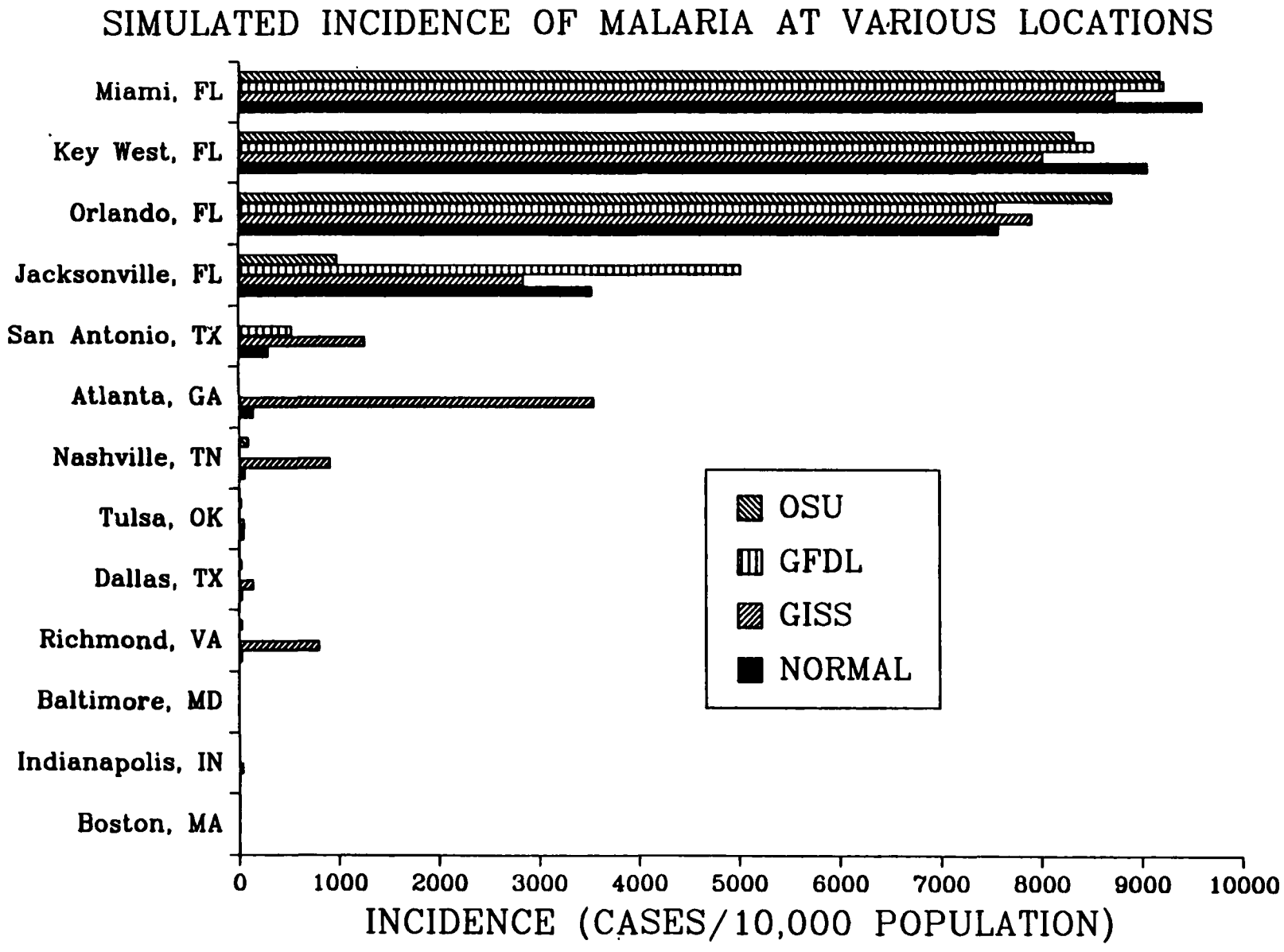


Figure 4. Simulated equilibrium incidence of malaria (after 3 years of simulation with constant weather) at different locations with average, normal weather data and modified weather data for each climate change scenario.

INTERPRETATION AND IMPLICATIONS OF RESULTS

These results suggest that potential problems associated with transmission of RMSF and malaria in the United States will be little worse with projected climatic change than they are today. However, these are only two of many vector-borne diseases that are a potential problem in the United States. Other modeling efforts would be required to address other tick or mosquito-borne diseases.

The results in this report are based on computer models, which although complex and useful from a research standpoint, have serious limitations for projecting the effects of climatic change. The limitations involve development of the models with average weather data and the lack of consideration of the climatic effects on host densities and habitat. Additional research on biology, ecology, and modeling of these and other vector/disease systems will be required for a more complete analysis of the potential effects of climate change.

REFERENCES

Dietz, K., L. Molineaux, and A. Thomas. A malaria model tested in the African savannah. Bull. WHO, (50):347 57, 1984.

Fine, P.E.M., M.M. Milbey, and W.C. Reeves. A general simulation model for genetic control of mosquito species that fluctuate markedly in population size. J. Med. Entomol., (16):189-199, 1979.

Haile, D.G. and D.W. Weidhaas. Computer simulation of mosquito populations (Anopheles albimanus) for comparing the effectiveness of control techniques. J. Med. Entomol., (13):553-567, 1977.

Haile, D.G. and G.A. Mount. Computer simulation of population dynamics of the lone star tick, Amblyomma americanum (Acari: Ixodidae). J. Med. Entomol. (24):356-369, 1987.

Harwood, R.F. and M.T. James. Entomology in Human and Animal Health. MacMillan Publishing Co. Inc., New York. 1979.

MacDonald, G. The Epidemiology and Control of Malaria. Oxford University Press, London. 1957. 201 pp.

Molineaux, L. and G. Grammiccia. The Garki Project: Research on the epidemiology and control of malaria in the Sudan savanna of West Africa. World Health Organization, Geneva, Switzerland. 1980.

Mount, G.A. and D.G. Haile. Computer simulation of population dynamics of the American dog tick, Dermacentor variabilis (Acari: Ixodidae). J. Med. Entomol. (in press)

Ross, R. The Prevention of Malaria, (2nd edition). London: Murray. 1911.

Russell, P.F., L.S. West, R.D. Manwell, and G. MacDonald. Practical Malariology, (2nd edition). Oxford Univeristy Press, London 1963.

Sonenshine, D.E. Influence of host-parasite interactions on the population dynamics of ticks. Misc. Pub. Entomol. Soc. Am. (9):243-249, 1975.

Sutherst, R.W., R.H. Wharton, and K.B.W. Utech. Guide to studies on tick ecology, CSIRO Aust. Div. Entomol. Tech. Pap. 14. 1978.

Warren, K.S. and A.A.F. Mahmoud. 1984. Tropical and Geographical Medicine. McGraw-Hill Book Company, New York, 1984.

THE POTENTIAL IMPACT OF CLIMATE CHANGE ON PATTERNS OF INFECTIOUS DISEASE IN THE UNITED STATES

by

Janice Longstreth
Joseph Wiseman
ICF/Clement Associates, Inc.
9300 Lee Highway
Fairfax, VA 22031-1207

Contract No. 68-01-7289

CONTENTS

FINDINGS[1]

The climate of the United States is expected to change as a result of the increased concentration of a number of "greenhouse gases." Although there will be an overall net global warming, the distribution of this warming will not be the same throughout all regions. Changes in temperature, rainfall, and other climatic elements will vary considerably by region.

The United States Environmental Protection Agency (EPA) is concerned that these climatic changes will increase the morbidity and mortality of infectious disease in the United States. A two-day workshop was convened to address the questions relating to this issue, including the following: Which infectious diseases will become a greater health problem; what public health measures can be taken to lessen the potential impact of these diseases; and what research agenda should be established in regard to these issues.

The workshop decided that given the present levels of sanitation, immunization, and nutrition in the United States, malaria, dengue, yellow fever, and several skin diseases pose the greatest threat of increased morbidity and mortality.

The workshop also concluded that the decrease in the support of public health programs, particularly those in disease surveillance and vector abatement, create the greatest threat to effectively combating increased morbidity and mortality caused by climate change. It was also concluded that the development of multidisciplinary teams including experts from the fields of behavioral science, entomology, and epidemiology, need to be established in order to develop programs to effectively meet the changes in infectious disease patterns caused by climate change.

[1]Although the information in this report has been funded wholly or partly by the U.S. Environmental Protection Agency under Contract No. 68-01-7289, it does not necessarily reflect the Agency's views, and no official endorsement should be inferred from it.

CHAPTER 1

INTRODUCTION

The climate[2] of the United States is expected to change due to an overall net global warming, which has been predicted to result from the increased global concentration of a number of "greenhouse gases" such as methane, carbon dioxide, and chlorofluorocarbons (CFCs) (EPA/UNEP, 1986; NAS, 1983). These gases are called greenhouse gases because they allow incoming solar radiation to pass through the earth's atmosphere but absorb outgoing infrared radiation thus resulting in warming within the confines of the atmosphere much as is seen within a greenhouse. Within the United States, it is expected that changes in temperature and rainfall will vary considerably by region, with some regions becoming colder and others warmer, as well as wetter or drier. Figure 1 presents one set of changes in temperature and rainfall, those predicted by the General Circulation Model (GCM) developed by NASA's Goddard Institute for Space Studies. Other GCM model predictions differ significantly from NASA's. While the quantitative and qualitative degree of regional climate change varies from model to model, it is relatively certain that temperature will increase by three to five degrees on average with a doubling in CO_2. This paper attempts to discuss, based on available data, how infectious disease may vary with normal changes in climatic factors.

EPA has been requested to prepare a report for Congress on the potential adverse effects that might be seen in the United States due to the global warming. One of EPA's concerns is that, associated with changes in climate throughout various regions of the United States, there will be changes in the morbidity or mortality of infectious diseases[3], both those endemic in the United States and those which may be introduced from other locations. This report addresses EPA's concerns with regard to the infectious diseases that currently occur in the U.S. or that plausibly might be imported; the worldwide impact of climate change on infectious diseases is beyond the scope of this report. The document has two goals. One goal is to attempt to determine the likelihood (with attendant uncertainties) that increased morbidity and mortality from particular infectious diseases will occur in the U.S. owing to changes in climate, and the second goal is to identify the information necessary to reduce the uncertainty in these conclusions.

It has been many decades since infectious diseases were the major causes of mortality in the United States, as they are now in large parts of the world. Figure 2 compares the 10 leading causes of death in the United States for 1900 and 1981. The leading causes of mortality at the turn of the century were (Mauser and Kramer, 1985) pneumonia and influenza, with tuberculosis second. By 1980, pneumonia and influenza were significantly reduced in importance and tuberculosis was not one of the ten leading causes of death. The differences in the leading causes of death in the United States between 1900 and 1980 are principally due to improved sanitation, housing, nutrition, immunization programs, and treatment. It seems unlikely that climate change will cause a return of these diseases as important causes of mortality.

Nevertheless, certain infectious diseases are not necessarily mitigated by improvements in sanitation, nutrition, immunization and treatment. These diseases present the greatest public health threat either because there are no vaccines or treatments currently available, because the infectious agents are not significantly

---

[2]Throughout this report, the terms 'climate' or 'climatic change' are used generically. Unless otherwise defined, use of the terms includes changes in temperature, rainfall, humidity, length of day, average daily solar radiation, and/or storm patterns, as well as changes in the frequency of rare events such as floods or droughts.

[3]This report addresses climatic changes likely to be induced by increases in the amount of greenhouse gases, with the exception of the probable impacts of increased UV-B brought about by stratospheric ozone depletion by CFCs. For an evaluation of that impact see Longstreth et al. (1987).

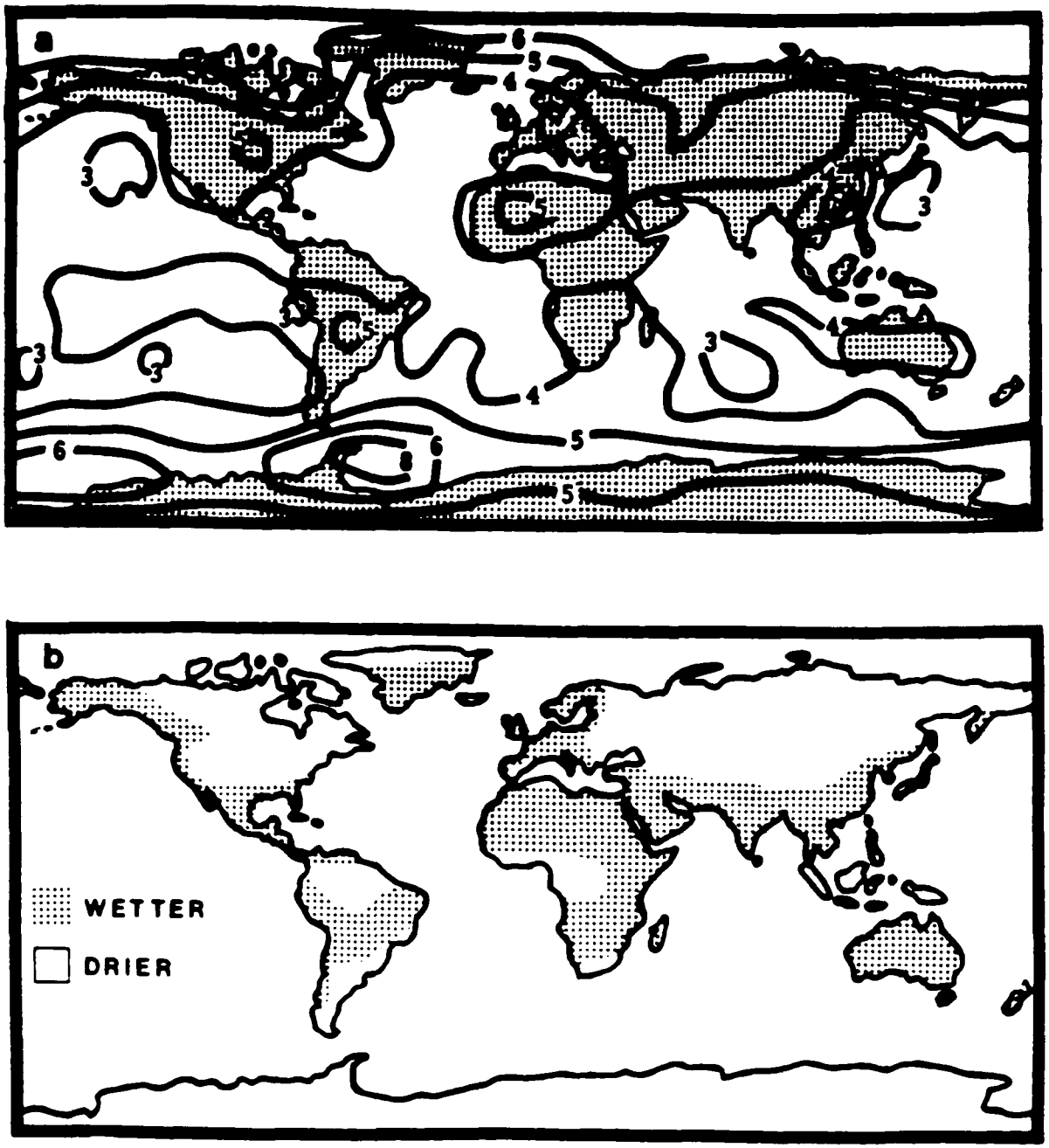


Figure 1. (a) Global patterns of surface temperature and (b) global changes in moisture patterns.

Source: Peters and Darling (1986).

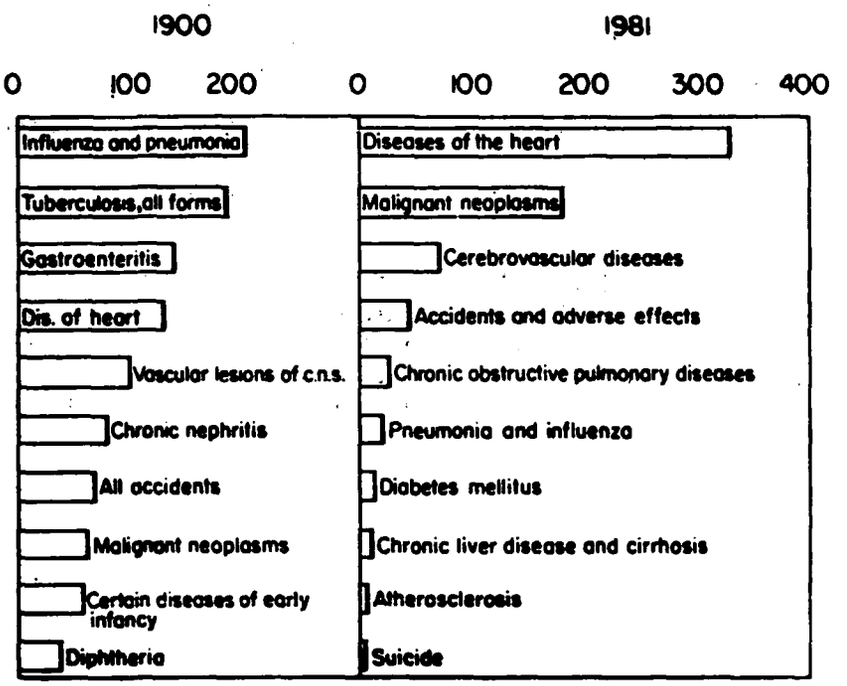


Figure 2. Death ratio for the ten leading causes of death in the United States 1900 and 1981.

Source: Mauser and Kramer (1985).

impacted by sanitation or nutrition, and/or because there may be significant portions of the United States population who have substandard access to such controlling factors. For the most part, diseases with these characteristics appear to be those that are vector-borne[4]. Thus this report focuses on this subset of infectious diseases in order to evaluate whether these diseases are likely to show changes of incidence within the United States following changes in climate. This evaluation will be based principally on information derived from five examples of vector-borne diseases, some of which currently exist in the United States and some of which either once existed in the U.S. and/or exist in locations near its borders. This subset of infectious diseases was chosen because a significant amount of information suggests that the prevalence and distribution of the vectors of these diseases can be affected by climatic variables, which in turn suggests that changes in climatic variables could change the prevalence and distribution not only of vectors but, most importantly, of the agents they carry.

There are at least two ways in which climatic change may impact vector-borne diseases. One mechanism of change is brought about by the direct impact of climatic change on the agent, vector, or host. For instance, changes in temperature, rainfall, humidity, or storm patterns that directly impact the multiplication or differentiation rate of the vector or the agent, increase the biting rate of the vector, or increase the amount of time that the host is exposed to the vector, would be considered direct impacts of climate change. A second mechanism of change is brought about by the indirect impacts of climate. In this category, climate influences some parameter that is important to vector spread or survival, such as the type of agriculture or the species of trees in a forest; this, in turn, changes the relationship between the parasite, vector, and host. Both types of impacts are considered in this report; clearly, however, the direct impacts are easier to evaluate than the indirect ones.

The report[5] first evaluates what is known about the impact of climatic factors on five vector-borne diseases: Lyme disease, Rocky Mountain spotted fever, malaria, dengue fever, and viral encephalitis. A discussion of several additional diseases determined by the workshop participants to be important is then presented. The information from these sections is analyzed for what can be generically stated about the effects of climate on agents, vectors and hosts. The final section presents the summary and policy implications.

---

[4]Vector-borne infectious diseases are those diseases in which the infectious agent is transmitted to the human host via an agent -- the vector. The vectors for most of the diseases likely to be observed in the U.S. are arthropods, e.g., fleas, ticks, and mosquitoes.

Notable examples of vector-borne diseases include malaria, which is transmitted to humans via mosquitoes, and bubonic plague, which is transmitted via infected fleas. (Plague is also transmitted directly from animals to animals, including humans, as a respiratory disease.)

[5]This report is limited to those diseases afflicting humans. Animal diseases, whether veterinary or of wildlife, will also be impacted by climatic change, and are the subject of a separate report.

CHAPTER 2

VECTOR-BORNE DISEASES

LYME DISEASE

Lyme disease, initially recognized in Lyme, Connecticut, in 1975, is caused by a spirochete, Borrelia burgdorferi, and is transmitted by ticks of the Ixodes ricinus complex.[6] It is an inflammatory disease, characterized by a distinctive skin lesion, erythema chronicum migran (ECM), systemic symptoms (profound fatigue, fever, chills, headache, and backache), polyarthritis, and cardiac and neurologic abnormalities which occur in varying combinations (Habicht et al., 1987). Symptoms are acute, lasting several weeks for most patients. Treatment with antibiotics is available and eventually complete recovery ensues (Braunwald et al., 1988).

The life cycle of the tick vector normally spans two years. Eggs are laid in the spring to hatch a month later into the larval form. During the first summer, the larva feed once on the blood of the host, then enter a resting stage with the onset of cold weather. The next spring, the larvae molts to become a nymph, which again attaches itself to an animal host. The nymph stage is primarily responsible for disease transmission (Steere, 1979). At the end of the summer, the nymphs molt into adults. They can be found in brush about one meter above the ground, where they easily attach to larger mammals (Habicht et al., 1987). Lyme disease is usually contracted between May 1 and November 30. The majority of cases are acquired in June and July (Steere, 1983b). This time frame corresponds with the peak questing period of Ixodes dammini, which for nymphs is May through July.

The principal hosts for juvenile and mature forms of I. dammini are the white-footed mouse (Peromyscus leucopus) and the white-tailed deer (Odocoileus virginianus), respectively. In addition, approximately 80 species of birds, mammals, and lizards have been identified as hosts of I. pacificus. It appears that lizards and Columbian black-tailed deer are the most important host of immature and adult I. pacificus, respectively (Westrom et al., 1985; Lane and Burgdorfer, 1986). The major importance of migrating birds as hosts is that they facilitate the movement of infected vectors over a large geographic radius, thereby substantially enlarging the geographic range of the vector and the agent (Hoogstraal et al., 1963; Spielman et al., 1985). In areas on the North Atlantic coastline, 80-90% of Ixodes ticks have B. burgdorferi in their gastrointestinal tracts. This rate contrasts with only a 3% rate of infection of I. pacificus on the West Coast. These rates correlate with the relative prevalence of the disease in the two areas.

Although Lyme disease has been reported from over 25 states, it has four major foci in the United States (Figure 3). It has also been reported in Germany, Switzerland, France, and Austria. In 1975, 59 cases were recorded in Connecticut; in 1985, the number had climbed to 863 cases (Habicht et al., 1987). Similar statistics are reported from New Jersey, with incidence increasing from 14 cases in 1980 to 39 in 1981 to 56 in 1982 (Bowen et al., 1984). Age-specific attack rates show that the risk of contracting Lyme disease is about equal in all age groups through age 50. After age 50, the risk lessens, probably because older persons are less likely to be present in tick-infested areas (Steere, 1983a). Better reporting may contribute to the increase in incidence statistics.

Lyme disease appears to occur wherever the vector is abundant. The presence of the vector in the northeast seems to depend on the presence of the white-tailed deer (Main et al., 1982). The range of the northeastern subspecies (O.v. borealis) is the known range of the vector I. dammini (Spielman et al., 1985),

---

[6] I. dammini in the northeastern and midwestern parts of the United States, I. pacificus in the western United States, and I. ricinus in Europe (Burgdorfer et al., 1985). It is also believed that other species of ticks, such as Dermacentor variabilis, Haemaphysalis leporispalustris, and Amblyomma americanum, serve as secondary vectors (Burgdorfer et al., 1985; Schulze et al., 1984; Schulze et al., 1986).

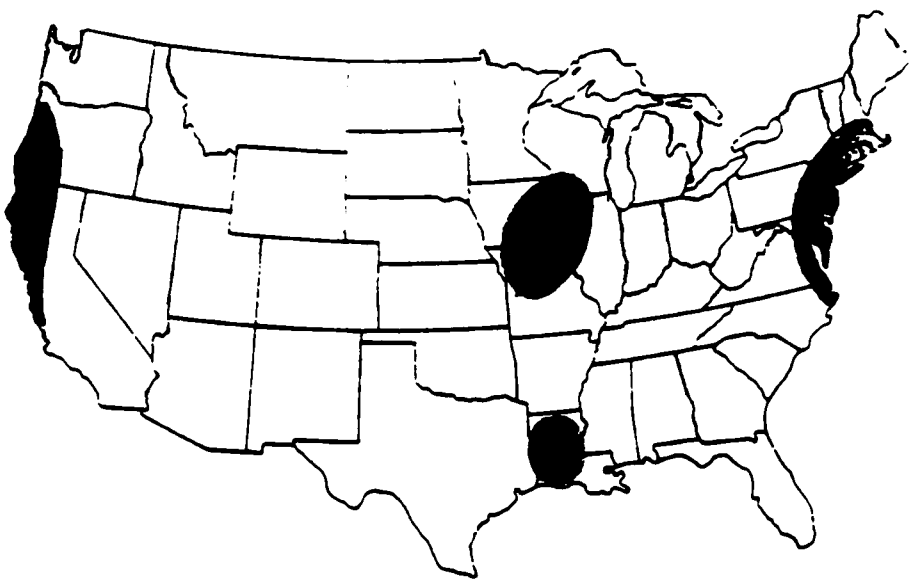


Figure 3. Geographic Distribution of Lyme Disease.

although formal proof of a cause and effect relationship is lacking (Spielman et al., 1984). Tick abundance mayalso depend upon environmental factors such as temperature, humidity, vegetation, and physiographic features of the environment (Spielman et al., 1985). Previous attempts to determine the abundance of I. dammini solely by climate (McEnroe, 1977) failed in that it ignored the distribution of the deer host and the presence of the tick in Wisconsin, which is a different climatic region (Spielman et al., 1985).

ROCKY MOUNTAIN SPOTTED FEVER

Rocky Mountain spotted fever[7] (RMSF) (also known as tick-typhus) is caused by the small, gram-negative, coccobacillus Rickettsiae rickettsii. The disease is characterized by fever, chills, headache and rash. Antibiotics are available for treatment, and vaccinations are available for those at greatest risk for developing the disease (Braunwald et al., 1988).

The two principal vectors of RMSF are the dog tick, Dermacentor variabilis, the distribution of which is shown in Figure 4; the wood tick Dermacentor andersoni, which is found mainly in the Rocky Mountain states; and Dermacentor occidentales which is found in parts of the western United States. Infection is maintained in ticks by transovarial transmission and by infectious feeding. The opportunity for a tick to acquire infectious rickettsiae is limited to the short period (3-4 days) in the life of a susceptible animal when the level of circulating R. rickettsii in the blood of the host is high enough for the tick to receive an infective dose[8] (McDade and Newhouse, 1986).

No data were found concerning the impact of environmental temperatures on the infectivity of R. rickettsii; however, data on R. mooseri, the agent for murine typhus, indicates that ambient temperature has a profound effect upon rickettsial growth in fleas, as well as the survival of fleas themselves. At 18°C, the rickettsial content of the fleas was below detectable levels for at least ten days and remained low throughout, whereas at 24° or 30°C, the rickettsial titer was consistently two or three times greater. In addition, if, after six days, the fleas were transferred from an environment of 18°C to one of 24° or 30°C, the rickettsial growth increased by two or three logs within one week (Farhang and Traub, 1985). The coccobacillus is heat tolerant.

Infection occurs in a large number of animal hosts. For example, in Maryland and Virginia, antibodies were found in 15 different mammals and 18 different types of birds (Bozeman et al., 1967; Sonenshine and Clifford, 1973). This large spectrum of animal reservoirs, the fact that several tick species are naturally infected with R. rickettsii (McDade and Newhouse, 1986), and man's role as an incidental host in the natural cycle makes eradication of RMSF unlikely. This is especially true since the United States has no tick control programs (Dr. Dan Haile, USDA; personal communication).[9]

Climatic, ecologic, and geophysical variables influence the timing of outbreaks. RMSF is a seasonal disease occurring in the warm periods that coincide with increased tick activity. In the west, the peak time is usually the spring and in the east the peak is from May through September.

Data for the United States population as a whole for 1970-1980 showed more than a doubling in incidence for the first half of this decade, going from less than 0.2 per 100,000 in 1970 to greater than 0.5 per

---

[7]RMSF is serologically related to R. conori, which causes Marseilles fever, Kenya tick typhus, and India tick typhus; to R. siberica, which causes North Asian tick-borne rickettsiosis; and to R. australis, which causes Queensland tick typhus. None of these diseases presently poses a problem in the United States.

[8]In order for a tick to receive an infective dose, the number of organisms has to be sufficient to overwhelm the so-called "gut barrier," allowing the organisms to invade the gut epithelium (McDade and Newhouse, 1986).

[9]This conclusion is also true for Lyme disease, as well as for other infectious tick-borne diseases.

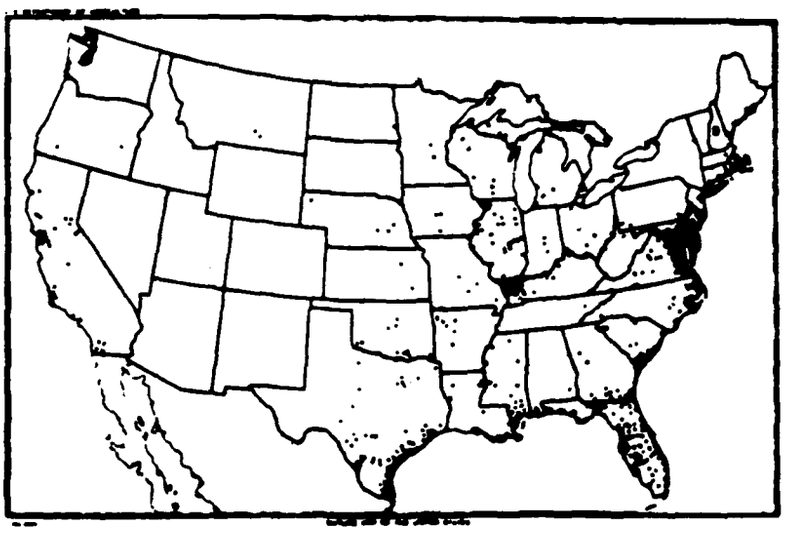


Figure 4. Distribution of Dermacentor variabilis.

100,000 in 1977 (CDC, 1986f). The higher rate was maintained until about 1983, falling to about 0.4 in 1984. Whether these changes were due to patterns of land use, microenvironmental changes, or other factors could not be determined from the available data.

Although the highest incidence of the disease is in children aged 5-9,[10] the highest mortality rates occur in the 40-59 age group[11] (D'Angelo et al., 1982). The fatality rate is about 15 to 20% in the absence of specific therapy; with prompt recognition and treatment, death is uncommon, yet between 4 and 6% of cases reported in the United States during recent years have died (APHA, 1980). Figure 5 shows the geographic distribution of the disease for 1982.

Prevalence of RMSF has been linked to natural vegetation, which reflects regional climatic conditions. The density of the principal tick vector also varies with the natural vegetation of the area. The highest incidence of RMSF has been associated with oak-hickory-pine forests. Within this general range, highest incidence is associated with drier, mesic forest types (Sonenshine et al., 1972).[12]

MALARIA

Malaria affects a large geographic area. Over the past 10-15 years, the prevalance and geographic distribution of malaria worldwide has increased slowly but steadily, sometimes in small foci, and sometimes over whole sub-continents. Its recent worldwide increase is due mainly to mosquito resistance to pesticides, break-down of control efforts, migration of the vectors, and irrigation. However, its spread is also environmentally related. Man's activities, including agriculture and road building, have created better habitats (e.g., more still water) and thus contribute to the spread of malaria into several areas where it was not previously present (De Zulueta, 1980).

Four protozoan agents potentially cause malaria in humans: Plasmodium vivax, P. malariae, P. ovale, and P. falciparum. The most serious form of malaria is induced by P. falciparum and presents a very varied clinical picture including fever, chills, sweats, and headache. It may progress to liver damage, coagulation defects, shock, renal and liver failure, acute encephalitis and coma. Case fatality among untreated children and nonimmune adults exceeds 10%. Malaria induced by the other Plasmodium sp. is generally not life threatening except in the very young, the very old, or those with concurrent disease. The less severe forms of malaria are characterized by an initial feeling of malaise, usually accompanied by headache and nausea and ending with profuse sweating. This pattern of symptoms occurs in cycles which appear daily, every other day, or every third day. Treatment of acute attack can be accomplished with quinine or chloroquinine (Braunwald et al., 1988).

Plasmodia are transmitted to humans via the bite of anopheline mosquitoes. In the mosquito, plasmodia go through a complicated life cycle involving a number of differentiation stages. P. vivax and P. malariae require environmental temperatures of at least 15°C for their development within the anopheline mosquito. P. falciparum requires temperatures of at least 17° or 18° C for its development to take place (Macdonald, 1957). With gradual increases in global temperatures since the last ice age (+8.0-9.5°) transmission has migrated from Africa to southern Europe.

---

[10]In the 5-9 age group, the incidence of RMSF is from 5.4 to 8.5 per 100,000 population in endemic areas. For the 10-19 age group, the incidence is from 2.5 to 4.0, and for the group 20 and older, the incidence is from 1.0 to 2.8 (D'Angelo, 1982).

[11]Approximately 10-12%.

[12]The effect of climatic change on forest growth has been modeled. See Solomon and West (1984); Botkin et al. (1972).

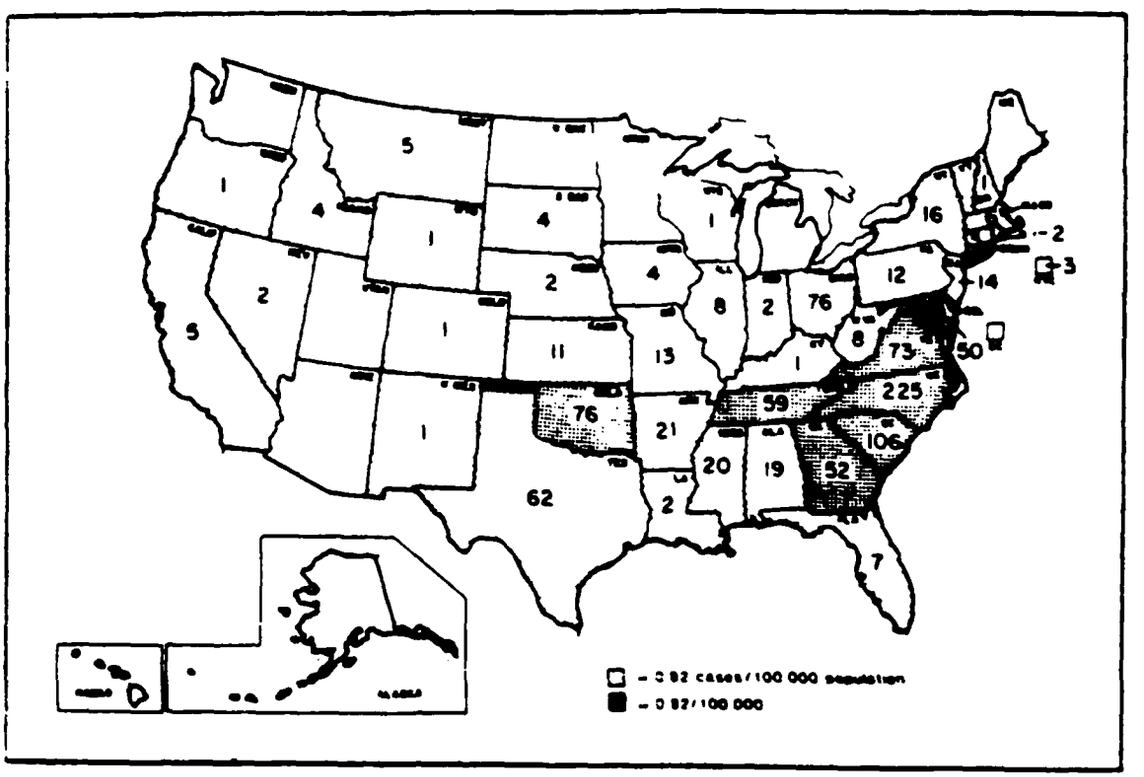


Figure 5. Distribution of Rocky Mountain Spotted Fever for 1982.

A number of different anopheline mosquitoes can serve as vectors for the malaria agents. In the United States, these include Anopheles quadrimaculatus and A. freebornei. In addition, Plasmodium species and isolates are known to adapt in order to be able to develop in particular Anopheles species or subspecies (WHO, 1987).

Malaria was once a significant health problem in the southern and western United States (Faust, 1938). Figure 6 illustrates mortality due to malaria in the south for the period 1925-35. With the realization of the role played by the mosquito in malaria transmission, breeding of the mosquito was controlled by better drainage and ambient spraying (Williams, 1938; Williams, 1937). Resistance of the vector to pesticides can make present-day control programs more difficult. Both P. falciparum and P. vivax were present in the United States.

Indigenous transmission of malaria within the United States, although rare, is still possible. Some or all of the 27 reported case of P. vivax that occurred in San Diego County in June-September 1986 occurred through indigenous transmission. A competent vector, Anopheles freeborni, is found in the southern California. In addition, there have been reports of local transmission among Punjabi immigrants in the Sacramento Valley. This represent the largest outbreak of malaria in the United States since 1952 (Brunetti et al., 1954; CDC, 1986a,f).

With a large influx of immigrants, sufficiently large amounts of the malaria parasite could be introduced into the United States in the presence of competent vectors. These vectors include An. freeborni in California and the states that border Mexico, as well as A. quadrimaculatus[13] in the entire Southeast during the warmer months (CDC, 1986a). As the climate becomes warmer, it may be expected that these vectors will increase in their geographic range throughout the entire Southeast during the warmer months (CDC, 1986a), and that An. quadrimaculatus will be present for a larger part of each year. In addition, raising the ambient temperature could provide conditions more favorable to the replication of the various Plasmodium sp. within a competent vector. If more irrigation is required in areas with increasing temperature and adequate drainage is not concurrently supplied, more mosquitoes could breed (Gratz, 1973). Longer seasons for exposure of the host human to infected vectors could also result.

DENGUE FEVER

Dengue is a mosquito-borne virus with four serotypes (Dengue 1-4). It is classified as a Group B arbovirus and is antigenically related to St. Louis encephalitis, yellow fever, Japanese B encephalitis, and other viruses. Classic dengue fever, which usually is not fatal, is characterized by the abrupt onset of fever and generalized body aching as well as severe headache and retro-orbital pain. Dengue fever can be complicated by dengue hemorrhagic fever (DHF) and dengue shock syndrome (DSS), both of which can be fatal, particularly in young children.

The principal vector for dengue is the Aedes aegypti mosquito, whose distribution in the United States is shown in Figure 7. A potential vector, Aedes triseriatus, is found throughout the eastern half of the United States. In addition, Aedes albopictus, a vector for dengue, originally of northern Asiatic origin, and resistant to winter freezing temperatures, has now been found in the United States. Aedes albopictus[14] has both rural and urban habitats and is a competent vector for dengue (Anon., 1987a). The distribution of A. albopictus in the U.S. is shown in Figure 8.

---

[13] A. quadrimaculatus is highly susceptible to both P. vivax and P. falciparum.

[14] A. albopictus has been shown experimentally to transmit dengue transovarially.

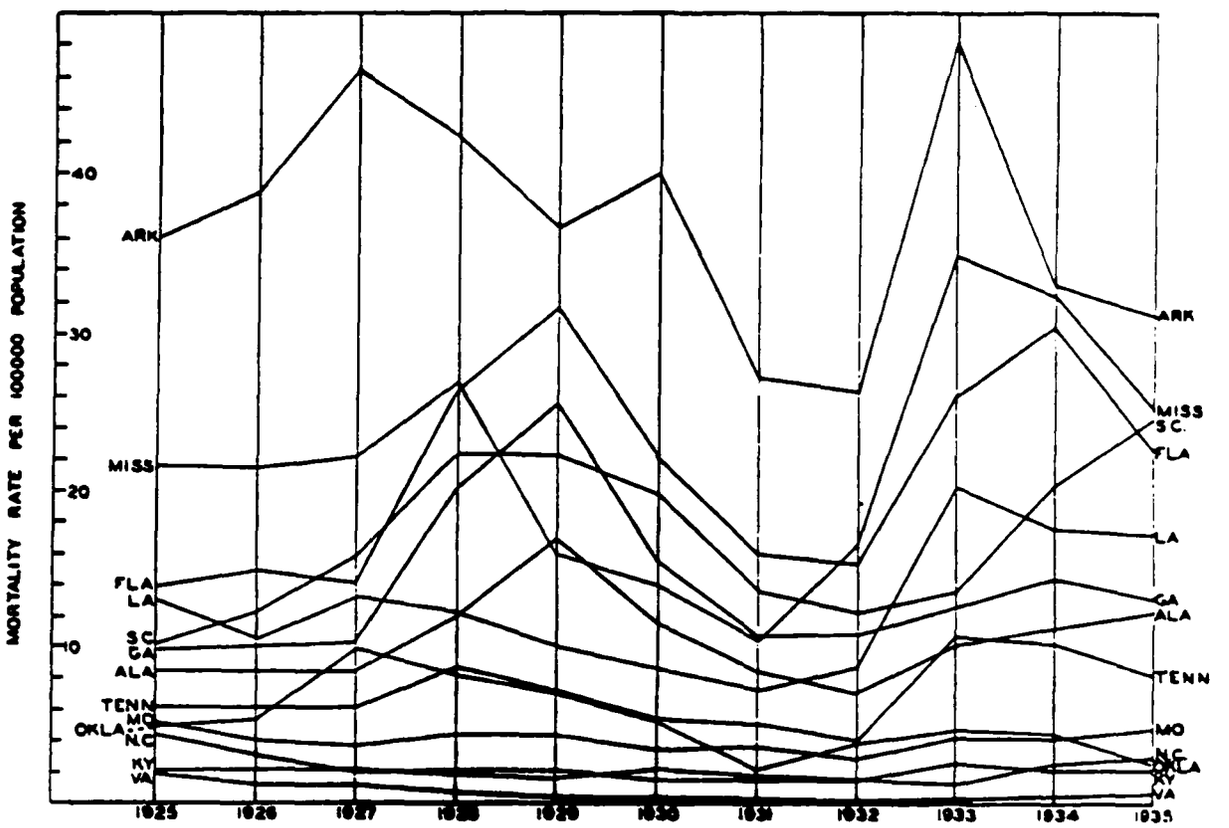


Figure 6. Malaria mortality in the southern states, 1925-1935.

Source: Dauer and Faust (1937).

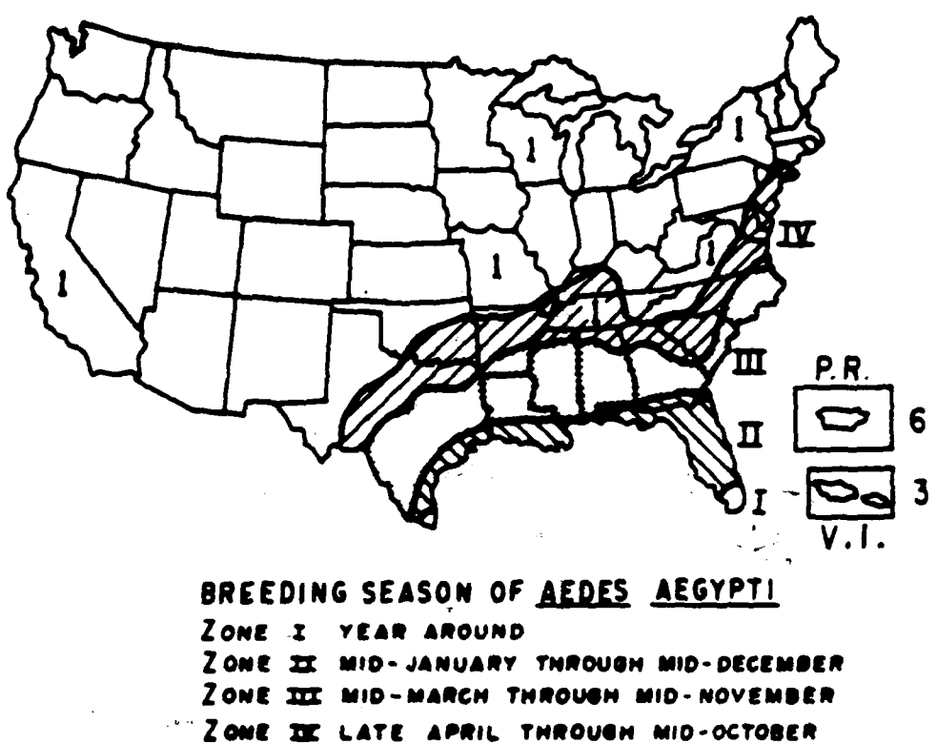


Figure 7. Confirmed Dengue cases imported into the United States in 1984 and Aedes aegypti distribution.

Source: CDC (1986e).

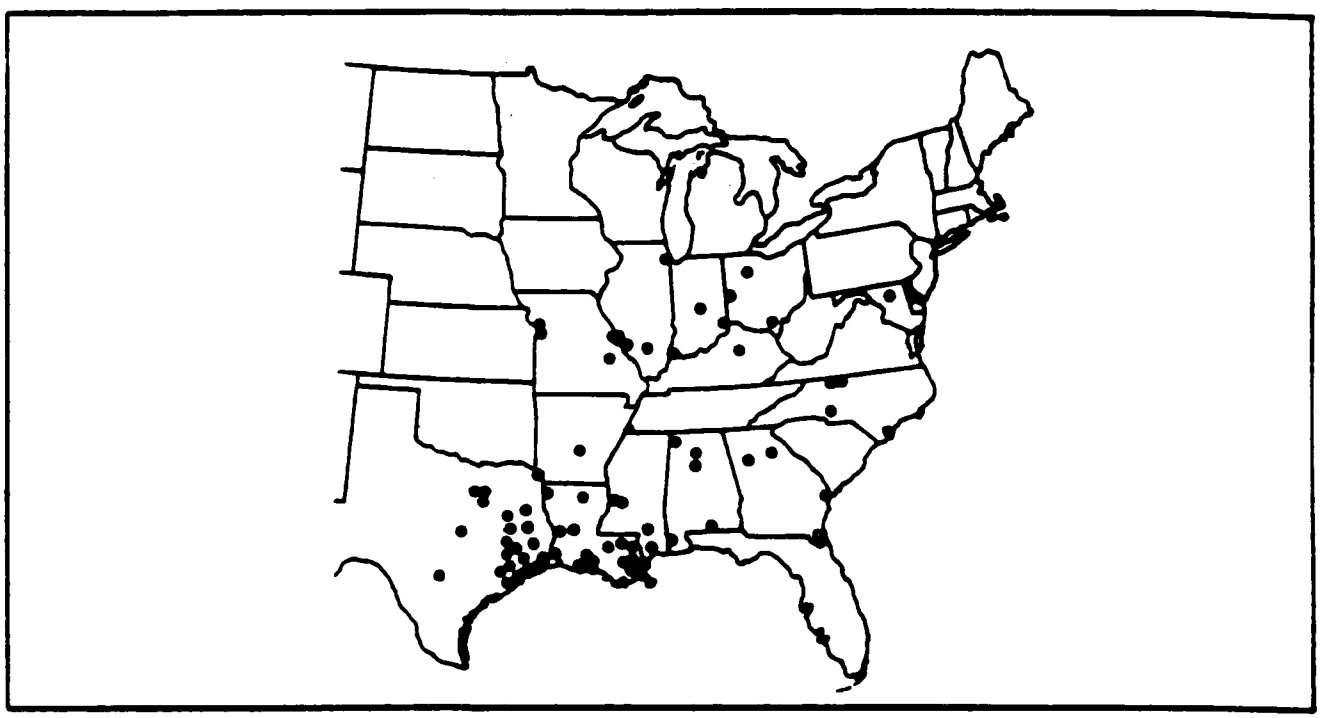


Figure 8. Counties with confirmed Aedes albopictus infestation in the United States.

Source: CDC (1987).

Longstreth

Dengue is not presently endemic in the continental United States. However, epidemics have occurred in the past.[15] The last such large epidemic occurred in Texas in 1945 (Anon., 1987a). Nine cases of indigenous transmission of dengue within the United States were reported from Texas in 1986 (Reiter, 1988).

Although dengue is not currently indigenously transmitted in the continental United States, it is a problem in Puerto Rico and also several localities that provide large numbers of immigrants to the United States. For example, 2,371 cases of dengue (DEN-1, 2 and 4) were reported from Puerto Rico in 1985 (Anon., 1987b). In addition, dengue had not been reported in Cuba for 30 years, when a DEN-1 epidemic occurred in 1977. This was followed in 1981 by an epidemic of DHF caused by DEN-2, which resulted in at least 158 deaths (CDC, 1981). Similarly, in 1978, Mexico reported its first cases of dengue in many years. The disease became endemic in most coastal areas of Mexico, and in 1984 cases of DHF were reported (CDC, 1986e). The spread of dengue and DHF in these countries is following a pattern similar to that found in Southeast Asia, where DHF is presently a leading cause of hospitalization and death of children.

Dengue is frequently introduced into the United States by people who have traveled abroad, who return to areas containing a competent vector for dengue (illustrated in Table 1 for 1982). Although travelers importing dengue virus into the United States have not introduced a large amount of the virus into a small geographic area, large numbers of immigrants from dengue endemic areas tend to settle in small geographic areas. A competent vector for dengue may be present in these areas.

Specific experiments have been conducted on the effect of temperature[16] on the ability of Aedes aegypti to transmit DEN-2 virus. These experiments showed that the DEN-2 virus was transmitted by A. aegypti only if the mosquitoes were kept at 30°C. The required extrinsic incubation period was shortened if the temperature was increased to 32°C and 35°C (Watts et al., 1987). This pattern of temperature and vector efficiency parallels the climatic pattern of DHF outbreaks in Bangkok, Thailand, where case rates rise during the hot season (with daily mean temperatures of 28°-30°C) and decrease during the cool season (with daily mean temperatures of 25°-28°C) (Watts et al., 1987).

ARBOVIRUS-RELATED ENCEPHALITIS

Arthropod-borne viruses (arboviruses) are associated with several major clinical syndromes,[17] including encephalitis. Arbovirus infections were responsible for 65% of diagnosed encephalitis cases reported to CDC between 1969 and 1979 (Shope, 1980). At least 18 arboviruses cause encephalitis; of these, at least 7 are present in the United States, including western equine encephalitis (WEE), St. Louis encephalitis (SLE), eastern equine encephalitis (EEE), California encephalitis (CE), La Crosse encephalitis, Powassan encephalitis, and Venezuelan equine encephalitis (VEE).

---

[15]Previous large epidemics include: Pensacola, Charleston, Savannah, and New Orleans (1827-1828); New Orleans, Mobile, Charleston, Augusta, and Savannah (1850); New Orleans, Savannah, Charleston, and Augusta (1879-1880); Augusta and Galveston (1885-1886); Texas (1897); Houston, Galveston, and Brownsville (1907 and 1918); Texas, Louisiana, Florida, and Georgia (1922); Miami, Florida, and Georgia (1934). Many smaller outbreaks have also occurred. For a complete history, see Ehrenkranz et al. (1971). Outbreaks also occurred in Hawaii in 1913-1915 and in 1943 (Usinger, 1944).

[16]Temperatures required to maintain maximum vector efficiency may vary depending on the specific disease/vector system.

[17]These include encephalitis, yellow fever, hemorrhagic fevers, hepatitis, arthritis, rash, and undifferentiated tropical fevers.

Table 1. Reported Cases of Dengue-Like Illness in the United States in 1982

State	Number of cases reported	Number of cases confirmed*	Travel history for patients with confirmed cases
Alabama†	7		
Arizona	2		
Arkansas†	2		
California	3	3	South America, Jamaica Philippines
Colorado	1	-	
Connecticut	2	2	Jamaica, Puerto Rico
D. C.	2		
Florida†	2		
Georgia†	11	2	French Guiana, Kenya
Hawaii†	2		
Idaho	1		
Illinois	11	2	Dominican Republic India
Indiana	3		
Maryland	2		
Massachusetts	16	5	Burma, China, Jamaica El Salvador, Sri Lanka
Michigan	2	1	Puerto Rico
Minnesota	3	-	
Mississippi†	1	-	
Missouri	6		
Nebraska	1	1	Thailand
New Jersey	5	2	Puerto Rico
New York	31	11	Suriname, Puerto Rico, India
Ohio	4	3	Puerto Rico, Sri Lanka
Oklahoma	1	1	
North Dakota	1	-	
Pennsylvania	7	5	
Texas†	12	5	Suriname, Michoacan, Mexico Tamaulipas, Mexico
Virginia	2	2	Venezuela, India
Vermont	1		
Total	144	45	

*Confirmed either serologically or virologically

†States with *Aedes aegypti* during much of the year

Source: Gubler (1983).

The effects of infections with these viruses range from a mild influenza-like syndrome to central nervous system (CNS) disease, which can be fatal. Encephalitis is characterized by acute febrile illness (oral temperature $\geq$100°F), with signs of brain parenchymal inflammation that include one or more signs of decreased level of consciousness (confusion, disorientation, delirium, lethargy, stupor, coma) and/or objective signs of neurologic dysfunction (convulsion, cranial nerve palsy, dysarthria, rigidity, paresis, paralysis, abnormal reflexes, tremor, etc.). Meningeal irritation is often seen in encephalitis patients; symptoms may include aseptic meningitis (acute febrile illness and signs of meningitis, such as stiff neck) and febrile headache (Monath, 1980). Delayed or latent CNS sequelae in children who have had encephalitis is also a severe complication (Finley and Longshore, 1958).

Outbreaks of encephalitis caused by the different viruses are normally limited to specific geographic locations and seasons, including seasons in which arthropod breeding and feeding occur. Most cases of encephalitis occur in late summer/early fall when mosquitoes, the primary vectors, are prevalent. In addition, infection depends on certain variables, including the species of mosquitoes that are susceptible to a specific virus, and the viral concentration in the susceptible vertebrate host's blood. More than 100,000 infectious units per milliliter are usually required in order for virulent strains to infect mosquitoes. An extrinsic incubation (EI) period, i.e., the interval between ingestion of the virus and subsequent transmission through biting, of 4 days to 2 weeks at summer temperatures is normally required, before the virus is fully multiplied and can be transmitted to a new host.

Many of these variables are affected by climatic change. For example, environmental temperature affects the EI (the species discussed here are active at temperatures of 13°-35°C). Increased temperature can decrease the EI, thereby quickening the transmission process and promoting epidemic disease. Moisture, present as rainfall or irrigation, affects the growth of plant life for feeding of host animals, and the presence of insect breeding sites. Changes in these environmental conditions affect the ability of the vectors to transmit the virus effectively. Finally, viruses carried by the same vectors appear to occur in the same climatic conditions and geographical distributions. It would follow, then, that the effective spread of the viruses, and usual subsequent epidemic disease, is dependent upon optimal environmental conditions in which the vectors may breed, feed, and transmit the viruses. Those conditions, wet or dry, mild or warm climates, vary with the particular species of mosquito vector.

WEE is transmitted in a mosquito-bird cycle[18] by the vector Culex tarsalis. C. tarsalis mosquitoes breed in groundwater ponds and abound in irrigated areas of California, Texas, and other parts of south-central and southwestern United States, where SLE occurs endemically (Shope, 1980). SLE accounts for occasional small outbreaks each year in humans and for periodic large epidemics. Warm weather is required to complete the extrinsic incubation of the virus in the mosquito. As the C. tarsalis naturally feeds on large vertebrates, equine and human cases occur annually. WEE, which can be severe but is not normally fatal, is seen primarily in children under 2 years of age, causing retardation, seizures and spasticity (Johnson, 1977).

Rainfall[19] strongly affects the numbers of infected individuals, since C. tarsalis breed mainly in ground pools and irrigated ponds. Environmental temperature also affects the activity of the arbovirus. The maximum temperature permissible for the WEE vector to transmit the virus effectively was $\leq$ 25°C. Above 32°C, virus transmission rates rapidly decreased (Hardy et al., 1980; Kramer et al., 1983). A study of EI temperatures of C. tarsalis showed decreased vector competence after two to three weeks of EI at 32°C as compared with vector

---

[18]WEE has an alternative cycle in jackrabbits and A. melanimon.

[19]A method used to predict Murray Valley Encephalitis (MVE) in southeast Australia involves the use of the Southern Oscillation (SO), which is a mode of climatic fluctuations in the area and is used to predict rainfall. Clinical cases of MVE have tended to occur in summers and autumns following periods of above-average rainfall. Darwin atmospheric pressure, an index of the SO, had been below average in the seasons preceding epidemics of MVE. This correlation can be used to predict outbreaks of MVE, therefore serving as an early warning system (Nicholls, 1986).

competence after EI at 18° or 25°C. The high temperature did not, however, affect preexisting infection (Kramer et al., 1983). Studies also demonstrate lower effectiveness in WEE virus transmission to humans at higher ambient temperatures (Hardy et al., 1980; Kramer et al., 1983). The cooler temperatures at which the WEE virus is better able to replicate allow for epidemic disease much earlier in the summer, and eventually much farther north in cooler climates later in the season (Shope, 1980).

SLE is the most geographically widespread arbovirus in the United States and the most common cause of epidemic arbovirus encephalitis in humans (Johnson, 1977). The C. tarsalis mosquito transmits the virus in the western United States and C. nigripalpus in Florida in the same manner in which WEE is transmitted. The two viruses carried by these vectors are therefore affected in the same way by climatic change. Thus, the vector and the infection are prevalent in areas and seasons of cooler climate and high precipitation.

SLE[20] in humans has been reported throughout most of the United States; however, the first reported Canadian case in humans occurred in 1975 (Spence et al., 1977). During that same year, a widespread epidemic occurred, with over 2,000 reported cases and 171 deaths, predominantly throughout the eastern two-thirds of the United States. The epidemic was primarily between late June and October. During that year, precipitation was below the long-term (1941-1970) average in June and July, and temperatures exceeded the normal in May. Figure 9 shows the percentage of cases of arboviral encephalitis by month of onset and the etiology reported to the CDC between 1955 and 1971. SLE encephalitis occurred mainly in the late summer and tended to follow those due to CE and WEE, but tended to precede cases of EEE (Monath, 1980). Nearly all epidemics have shown that the incidence of SLE is 5-40 times higher in persons over 60 years than those in the 0-9 age group (Monath, 1980).

Mosquitoes belonging to the Culex pipiens complex (C.p. pipiens, C.p. quinquefasciatus, and C. nigripalpus) are the principal vectors for SLE in urban areas. These species breed in polluted water of high organic content; therefore, these species are suited to urban and suburban areas (Monath, 1980; Shope, 1980), unlike the primarily rural C. tarsalis. In contrast to the effect of rainfall on C. tarsalis, C. pipiens epidemics occur more often in times of drought and high temperatures. The lack of rainfall results in poorly draining and stagnant water, providing increased breeding grounds for the vector (Monath, 1980; Shope, 1980; Johnson, 1977). The year of the first major epidemic in the United States, 1933, was the driest year recorded since 1837 (Shope, 1980).

High temperatures favor virus transmission for C. pipiens and C. tarsalis by decreasing the EI time, as well as the time required for larval maturation and development of viral infectivity (Monath, 1980). Studies of the relationships between incubation time of SLE virus in C. pipiens and mean temperature found that daily exposures to increased temperatures from a constant of 25°C decreased incubation time, thereby increasing effectiveness of viral transmission (Hardy et al., 1980). In addition, a review of all the arbovirus encephalitis cases found in the United States showed that most WEE outbreaks have occurred at or above the 70 F June isotherm, whereas most SLE cases have occurred in warmer latitudes at or below the June isotherm (Hess, 1963).

Eastern equine encephalitis (EEE) is transmitted in a mosquito-bird cycle by Cuiseta melanura, and is present in the freshwater marshes along the Gulf and Atlantic coasts, including the Great Lakes region (Johnson, 1977; Shope, 1980). The vector mosquitoes do not feed on large vertebrates; therefore, infection in humans is very rare. However, climatic changes altering the conditions of the wetlands, such as rainfall, could introduce changes in mosquito breeding or types of susceptible birds. The virus might then spill over to a species of mammal-feeding mosquitoes, most likely Aedes or other Coque tidia, via infected birds (Shope, 1980). This

---

[20]In Florida, the primary urban, as well as rural vector is C. nigripalpus, occurring more frequently in rural areas. Eight species of SLE-carrying mosquitoes in Florida were studied and found to transmit vertically, with A. taeniorhynchus transmitting venereally, although there is no evidence that this occurs in nature. Transstadial transmission by this species was observed at larval rearing at 18°C but not at 27°C. This is a possible over-wintering host for the virus in Florida because of the abundance and high transmission rate.

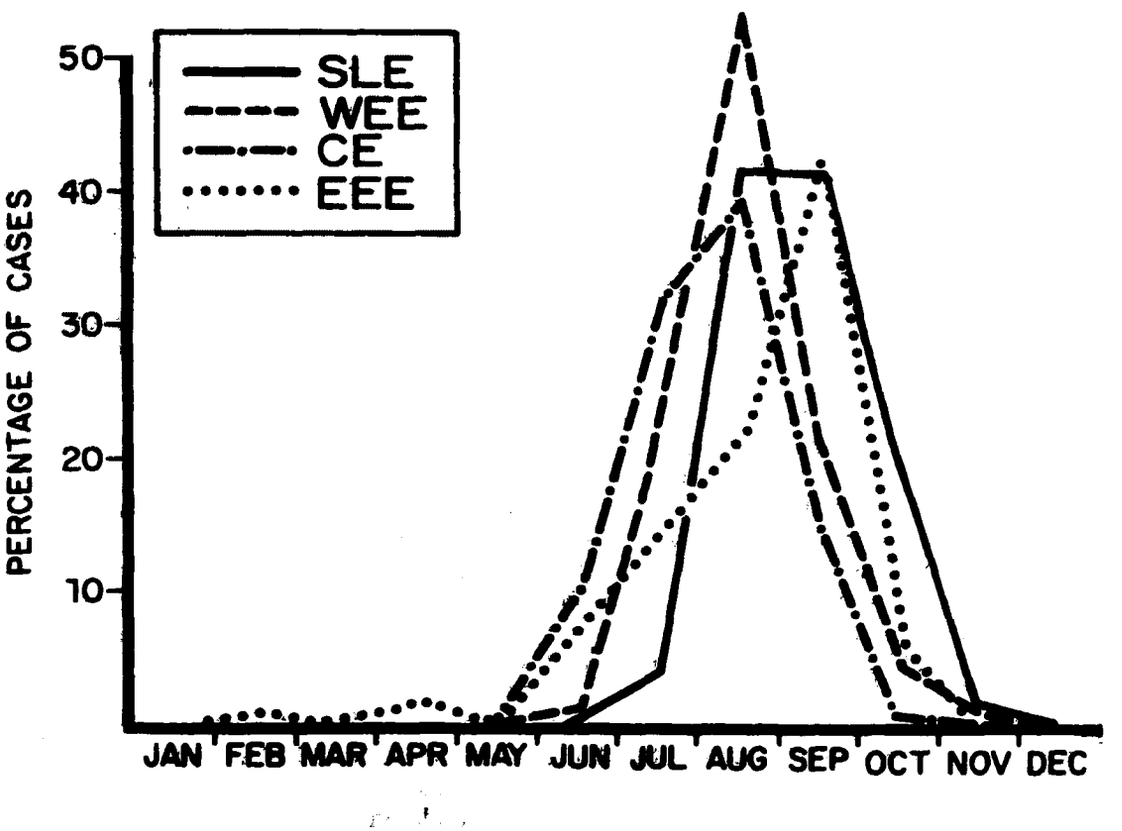


Figure 9. Percentage of cases of Arboviral Encephalitis by month of onset, 1955-1977.

Source: Monath (1980).

is the only route of epidemic transmission of EEE to horses and to humans, in whom infection is rare, but fatal in 60% of the cases (Johnson, 1977). EEE affects primarily children, and death usually occurs in the first 2-5 days after onset, characterized by periorbital edema during the acute disease (Johnson, 1977).

The La Crosse strain of California encephalitis virus causes mild to severe cases of encephalitis in children ranging from a benign aseptic meningitis to a severe, but rarely fatal, form of encephalitis (Johnson, 1977). There is little information on the effect of climate on the occurrence of the disease. The virus is widely distributed in the Midwest and the middle Atlantic and Appalachian states (Shope, 1980), and is transmitted by Aedes triseriatus mosquitoes, which reside in woodland treeholes and feed on small woodland mammals. A. triseriatus transmits the virus transovarially,[21] (Shope, 1980) as well as venereally through infected seminal fluid (Thompson, 1978), and thus the adult progeny can then transmit the infection by bite upon adult emergence (Shope, 1980). This vertical transmission cycle allows for human La Crosse infection during early summer, and is amplified by infection and viremia in small mammals. The mosquito has a very limited flight range, so humans are infected only when exposed to the woods or to old discarded tires, which serve as "synthetic treeholes" (Johnson, 1977).

[21]In a study of transovarial transmission of CE viral strains in A. dorsalis and A. melanimon, no consistent transmission rates were found relating to the time of year or location of collection (Turell, 1982).

CHAPTER 3

ELEMENTS OF INFECTIOUS DISEASE

In the attempt to predict what the potential impacts of climate change might be on infectious diseases, we decided that the analysis would be facilitated through the development of a conceptual model that looked at the interrelationship between climate and the elements necessary for a vector-borne disease to occur. Accordingly, given below are brief summaries of information drawn from the five diseases discussed above, which apply to the three elements of a vector-borne disease: the agent, the vector, and the host (or hosts).

THE AGENT

The agent in a vector-borne disease may be a virus, protozoa, bacteria, or helminth. Infectious agents are transmitted to their hosts by vectors. The role of the vector may not only be one of transport, but may also be required for completion of the lifecycle of the agent. As indicated above, there are two potential concerns regarding the impact of climate change on disease agents: one is that climate change will allow the establishment of new agents in the United States; the other is that agents that are presently endemic will flourish, thereby favoring either enlargement of the geographic area where they are found or greater infectivity in the current geographic area.

Agents Not Currently Present in the U.S.

Malaria and dengue are two vector-borne diseases not currently established in the United States, although they are introduced regularly and have been established in the United States in the past. Two questions must be considered in evaluating whether the establishment[22] of new infectious diseases in the United States is possible. The first is whether the agent is currently present or is likely to become present (by virtue of immigration, etc.) within the geographic range of a competent vector; the second is whether conditions are or will become favorable to the development and spread of the agent. The minimum amount of agent in a geographic area needed to assure transmission of the disease varies with each specific disease.

Although from time to time infectious diseases may be introduced into the United States by the return of travelers, generally the geographic distribution of these individuals is sufficiently broad that the critical mass of the agent necessary for establishment is unlikely to occur. For malaria, climatic conditions do not favor the differentiation of the agent within the vectors which are present.

Currently, the two most likely sources of introduction of a disease[23] into the United States are via legal immigrants and refugees (principally from Asia) and via migrant workers and illegal immigrants. Between 1975 and 1981, 0.5 million Asian refugees settled in the United States. Roughly one third of that population lives in California; one third lives in the states of Texas, Washington, Pennsylvania, Illinois, Minnesota, Virginia and Oregon; and one third have settled throughout the remaining states (Cantanzaro, 1982). If immigrants continue to congregate in nearby areas, a critical mass could be approached. In addition to these refugees, between 1977 and 1985, the largest number of immigrants admitted to the United States have been from Asia (INS, 1985). Of the 50 metropolitan statistical areas specifically listed by the Immigration and Naturalization Service as areas where immigrants intended to reside, 14 are areas presently known to be infested with Aedes aegypti (INS,

---

[22]Establishment is used here to mean the indigenous transmission of the disease by local vectors to sizable population.

[23]In addition to the diseases discussed elsewhere, the introduction of JBE from Asia, Ross River fever from Australia, Chikungunya virus from Asia, and Rift Valley fever from Africa should be considered possible candidates.

1985; CDC, 1986e,f). The fact that a second vector, Aedes albopictus, has become established in northern, as well as southern states (CDC, 1986b,d), broadens the area in which a dengue virus could become established given appropriate conditions.

Another source for the introduction of new infectious agents into the U.S. is via migrant workers (border commuters) and illegal immigration. It is estimated that between 4.5 and 12 million illegal aliens reside in the United States (Bos, 1984), as well as another 1.3 million seasonal workers and border commuters (Corwin, 1982). It is assumed that these individuals carry the parasites commonly found in their home countries. In addition to dengue and malaria, large percentages of immigrants and refugees are infected with intestinal parasites (15% to 78%), positive tuberculin test (49% to 55%) (Arfaa, 1981), and hepatitis B (12% to 36%) (CDC, 1979a,b; 1980c,e; Barry, 1983; McGlynn, 1985; Cantanzaro, 1982; Jones et al., 1980).

Medical screening of immigrants and refugees does not include testing for arbovirus, malaria, or most parasitic diseases. The CDC guidelines for medical examination of non-United States citizens seeking entry into the United States do not include such diseases as one of the "dangerous contagious diseases."[24] Nevertheless, it is estimated that within a single refugee detention center, several cases of dengue are identified each year, with an occasional large outbreak. There are also confirmed cases of malaria each year.

This section has illustrated that sufficiently large amounts of parasites are introduced into the country, making outbreaks of diseases not previously endemic to the United States possible. Although other factors also need to be present, the first element of the infectious disease relationship is presence of the parasite.

Agents Currently Found in the U.S.

For agents of diseases such as RMSF, Lyme, arbovirus-transmitted encephalitis, plague, and other endemic agents, the critical question is whether changes in climate will either enlarge the area in which they are present or increase the number of cases seen in the endemic areas. With regard to the role of the agent, is it likely that replication of the agent in vectors (or hosts) will be more favored under new climatic conditions? If the answer to this question is yes, then this could result either in larger amounts of agent per vector, a shorter time for the agents to reach infectivity within the vector, or a wider geographic area that supports development of infectious agents in the vector. Given below are brief summaries of what was found in the literature relevant to assessing these questions. Clearly, much is not known. (A discussion of additional data needs is presented in the Conclusions and Recommendations section.)

Lyme disease currently has four major foci in the United States. The climatic conditions within the foci are dissimilar and the transmission of the disease seems to be more dependent on the presence of infected ticks and their principal hosts, the white-footed mouse and the white-tailed deer, than on the competence of the agent within the vector. No data were found to address whether climatic factors can affect the reproductive and/or developmental processes of this spirochete within its tick vector. However, the incidence of the disease is increasing.

The incidence of Rocky Mountain Spotted Fever in the United States doubled in the decade of 1970 to 1980, from less than 0.2 per 100,000 to more than 0.5 per 100,000; however, by 1984 the rate was 0.4 per 100,000. These are nationwide statistics and it is unclear what they reflect in terms of the agent. Clearly the agent is present; however, very little information was found upon which to base an assessment of how climate change could affect its distribution or infectivity. If R. rickettsia, the agent for RMSF, behaves like R. mooseri, the agent for tick typhus, then one would expect to see increased rickettsial titers in ticks under warmer conditions; however, as discussed below, warmer temperatures could disrupt diapause in the vector life cycle, which could adversely affect tick survival or rickettsial cycles.

[24]"Dangerous contagious diseases" include AIDS, tuberculosis, VD, and leprosy.

THE VECTOR/ANIMAL HOST

Vector-borne diseases were selected for study in this review principally because the greatest amount of information found about the relationship of climatic variables to infectious diseases was found for this subset of infectious diseases, and the judgment was made that the high levels of nutrition, health care, and hygiene found in the U.S. were likely to prevent a significant impact of climate changes on other more common bacterial and viral diseases. The discussion that follows focuses principally on the impacts that climatic change could potentially have on the tick and the mosquito. In addition, this section includes information on the impact of climatic variables on intermediate vertebrate hosts.

The introduction of a vector into a disease process means that there are now three places (agent, vector/intermediate host, and human host) that require attention to control a disease, instead of the standard two (agent and host). Because of this intermediate step between the agent and the host, control of these diseases becomes more difficult than control of diseases such as pneumonia or tuberculosis, which are induced directly by the agent in the host.

The two most important factors related to a vector's transmission of disease are the geographic range (both in distance and in amount of time during the year that the vector is present) and the vector's rate of infectivity by any parasite. The geographic distribution of tick vectors in the United States is determined by a complex set of factors which are both geographic and climatic in nature. For example, D. andersoni, the vector of Rocky Mountain spotted fever (RMSF) in the western part of the United States, is associated with "shallow soils, moderate shrub cover, exposed rock, steep slopes, numerous pines, log litter" and decreasing grass cover (McDade and Newhouse, 1986). Those same factors influence the abundance of the vertebrate host for the larval tick.

Temperature and relative humidity are also important in the vector's spatial distribution. The relative humidity must be high enough to prevent desiccation of ticks and their eggs, and the ambient temperature must be high enough (app. 20°C) to allow the life cycle of the vector to be completed. However, winter soil temperatures must be low enough to release the adult ticks from their diapause, a period of lowered metabolism, that prevents their feeding in the fall (Wilkinson, 1967; McDade and Newhouse, 1986). This appears to be less than 5°C. Shorter survival rates have been observed at 75% relative humidity than at 85-95%, although the optimum relative humidity for vector reproduction and survival has not yet been determined (Wilkinson, 1967).

The difference in the timing of onset of disease, as contrasted by the pattern of RMSF in Virginia, Massachusetts, and Nova Scotia, may be explained by the direct or indirect effect of environmental changes. For example, a change in the temperature isotherms in the various areas could have modified a vector's life cycle, or a change in the composition of the flora and/or fauna could have resulted in the introduction of an additional vector whose life cycle differs temporally from the original vector so that maturing and overwintering ticks overlap considerably in terms of the time of activity. The definitive combination of factors that are conducive to the continued geographic spread of the vector has not yet been determined (Garvie et al., 1978).

The impact of climatic factors on mosquitoes serving as vectors has been studied for encephalitis virus infections. Transmission of arboviruses by mosquitoes is influenced by a number of factors. Among these are population levels, biting habits, and the intrinsic factors affecting the actual biological transmission of the specific agents. These variables are usually very closely related to climatic change, especially temperature and rainfall, which in turn determine the geographical distribution of the individual species and the viruses for which they are specific (specific viruses are carried only by specific susceptible vectors).

The population levels and biting habits of virus-specific mosquitoes vary according to climate, genetic determination, and abundance of hosts. In species which undergo diapause[25] and are thus capable of

[25]Diapause is a physiological state of suspended activity or arrested development that facilitates survival through a period of unfavorable conditions, but is initiated before the onset of these conditions (Bailey, 1982).

overwintering, the population levels[26] vary throughout the year. Viral activity is usually increased during the active summer months and decreased during winter months because of reduced feeding levels related to the onset of diapause. Diapause is induced in late-stage larvae and pupae by a combination of shortened photoperiod and cooler temperatures, and thus varies temporally with latitude (Bailey, 1982). In a study of Culex pipiens, indigenous to temperate climates and capable of overwintering, and Culex quinquefasciatus, found in pantropical regions (Florida) and incapable of hibernation, it was found that although both species develop fat reserves on a sugar diet, only C. pipiens within this complex of species underwent the additional physiologic changes associated with diapause in response to exposure to shortened photoperiod and decreased temperatures. The C. p. quinquefasciatus is apparently unable to utilize its fat reserves or decrease its activity in order to survive temperate-zone winters. The C. p. pipiens, on the other hand, showed signs of overall decreased activity, including reduced blood-feeding[27] (Eldridge, 1968).

In addition to influencing population levels and biting habits, climate variables strongly influence the capability and rate of transmission in vectors that undergo biologic transmission. Effective biologic transmission (or extrinsic incubation, EI) depends on several variables such as the infectivity threshold, virus multiplication rates, and susceptibility and dissemination barriers, many of which are temperature-dependent.

The EI period is the time required after ingestion of an infective blood-meal, during which the viral multiplication takes place and before the virus can be transmitted orally by the vector (Hardy et al., 1983). The specific time varies for each virus and mosquito, and the length of time the mosquito must be able to survive after ingestion in order to transmit the virus can be determined from it. Longer survival rates of mosquito populations, then, are required to maintain cycles of viruses with long EI periods (Hardy et al., 1983). Viral multiplication rates vary directly with temperature, as shown in a study of vector capability of Aedes aegypti for California encephalitis virus and dengue viruses at various temperatures. A. aegypti is indigenous to warmer climates and is a highly effective vector of dengue, a tropical arbovirus, but is an ineffective vector of CE[28], which is endemic at latitudes where frigid conditions prevail. At a range of temperatures, the EI period of DEN-2 virus increased from 6-13 days with a decrease in temperature from 90°-75°F. Transmission of the Yukon strain of CE was very ineffective, with EI periods of 3 weeks at 80°F, and 4 weeks at 55°F (McLean, 1977). In a similar study of A. aegypti, viral replication of Yukon and Norway strains of CE and MVE (found in moderate climates) was seen in its full viable range (13-39°C), with rates of viral multiplication decreased at lower temperatures and increased at higher (McLean, 1975a,b). Therefore, although the A. aegypti is susceptible to infections from regions of the world in which the species is absent, the decreased temperature has such a negative effect on the EI period that this warm-climate vector would not be a competent vector in colder climates.

The infectivity threshold (IT), or concentration of virus that must be ingested by the vector in order for strains to become infective, varies among viruses and mosquito species. There are many barriers, such as salivary gland and mesenteronal barriers,[29] preventing infection of certain tissues, and so the most effective viruses have the lowest infectivity thresholds. However, a low IT is not necessarily a requirement for successful transmission

---

[26]An investigation of seasonal biting habits of Aedes aegypti in Bangkok, Thailand, showed little fluctuation in the seasonal population levels of the mosquito. The increased occurrence of dengue hemorrhagic fever during monsoon season could not be attributed to an increase in population due to the heavy rainfall, but rather to the increased activity of the existing population under favorable climatic conditions (Yasuno and Tonn, 1972).

[27]The same study indicated a correlation of lower body fat with more normal ovarian development.

[28]It is ineffective only because it cannot diapause. However, A. albopictus is not so affected by colder conditions.

[29]These barriers are not directly affected by climate change, but rather by dose and time, and are primarily genetically determined. They are therefore beyond the scope of this report and will not be discussed.

if the virus produces very high levels of viremia in its vertebrate host, or if transovarial (vertical) transmission is possible (Hardy et al., 1983).

Transovarial transmission has been observed in many mosquito species and is considered to be a primary means by which some arboviruses are maintained during winter, dry seasons, and under adverse environmental conditions under which their arthropod hosts are either inactive or unable to survive. This is especially important in species in cooler climates and incapable of overwintering. Transovarial transmission was first observed for yellow fever virus in A. aegypti in 1905 (Rosen, 1981). Vertical transmission of SLE has also been widely demonstrated in eight species which occur in Florida. In this study, larval rearing temperatures affected such transmission of the virus in A. taeniorhynchus, with effective transmission at 18°C, but not at 27°C (Nayar et al., 1986). In a different study, vertical transmission of SLE by female A. albopictus and A. epactius occurred. Minimal infection rates of F_1 adults reared at 18°C were much higher than in those reared at 27°C. This type of transmission is postulated to be a mechanism for overwintering of the SLE virus in temperate regions of North America. Dengue, JBE, and yellow fever viruses are also transmitted vertically at low levels by Aedes mosquitoes (Hardy et al., 1980).

It is difficult to make any generalizations regarding the geographic distributions of specific vectors without addressing each one individually. However, based on the affects of temperature changes, one can make a broad conclusion that species that are capable of overwintering or of transmitting vertically, and which have higher rates of transmission at lower EI temperatures, are more likely to be found in latitudes corresponding to temperate-zone to arctic-zone climates. The viruses for which they are specific will also be endemic to those areas.

DEFINITIVE HOST

The definitive host is the organism where the infectious agent grows and sexually multiplies. For these vector-borne diseases, the human host provides nutrition and the necessary environment for proliferation. The information on the five vector-borne diseases was reviewed; very little information was found specifically relating to the impact of climatic factors on modifying the role of the host in the infectious disease process.

> ...it is becoming increasingly evident that man rather than nature is likely to be responsible for the direct or indirect dispersal of an arbovirus over great distances. Although by no means proved, it is distinctly possible that recent major and singular epizootic epidemic outbreaks of Venezuelan equine encephalitis in Central and North America and of Rift Valley fever in Egypt were caused by human behaviour. In any event, it is now clear that most disease-producing arboviruses are not introduced into a known endemic region annually by migratory birds or other long-distance non-human travellers (WHO, 1985a).

Theoretically, there are two types of human activity that could impact the disease process and could in turn be modified by changes in climate: those activities that humans as society undertake and those activities that humans as individuals undertake.

Human activities in the societal sense, particularly as they relate to the environment, increase or decrease the amount of disease. Clearings, power lines, logging roads, and campsites frequently become heavily infested with ticks carrying RMSF (Hoogstraal, 1981). Increased use of irrigation may lead to increases in mosquito populations (Gratz, 1973).

> The disappearance of plague from Uganda coincided with the introduction of antibiotics and DDT but probably had nothing to do with either, just as the virtual disappearance of malaria from the USA in the 1950s (which stood at 4 million cases per year in the 1930s) had little, if anything to do with the new anti-malarials and DDT. I well remember L.W. Hackett emphasizing this point when I was studying epidemiology at Berkeley in 1955. In the USA, as in Western Europe, changes in the ways of man were probably involved.

> Perhaps some of us should be making a special study of the factors involved in bringing about these great recessions with as much energy as we bring to the study of the factors responsible for the establishment of endemic and epidemic conditions (Gillett, 1985).

The environmental changes resulting from global climate change as well as the activities taken in response to these changes could potentially result in changes in the pattern of infectious disease. All the implications of global climate change are not yet known; however, within EPA's program to evaluate the potential impacts, consideration is being given to how hydrology, agriculture, forestry and infrastructure will change on a regional level (EPA, 1987). Potential changes which would result from sea level rise and changes in temperature and rainfall patterns include increased use of irrigation and impacts on wetlands and coastal, lake, and river ecosystems, such as geographic shifts and changes in composition; changes in the composition and growth rate of forests; and changes in crop yields, geographic distribution, and pesticide use. All of these changes have potential for impact on disease patterns and the growth and development of agent-vector pairs.

Perhaps of equal importance in the transmission of infectious disease in the United States is man's behavior as an individual. The best studied example of this is the use of air conditioning and television as it relates to mosquito-borne encephalitis in the San Joaquin Valley (Gahlinger et al., 1986). It was found that the use of these appliances decreased the amount of time people spent outdoors on summer evenings, the feeding time for the vector Culex tarsalis. The decreased prevalence of WEE and SLE in humans, together with the virus' continued presence in avian and equine populations, as well as the prevalence of the vector, can be accounted for only by the individual activities of man.

ADDITIONAL DISEASES

Participants at the workshop identified three additional diseases that they felt deserved attention in this report. A brief description of these diseases is given below.

Rift Valley fever (RVF) is a mosquito-borne, acute febrile disease of cattle, sheep, and man. It is characterized by fever, vomiting, hemorrhagic manifestations, vascular retinitis, encephalitis, and CNS complications (WHO, 1985b). RVF has long been recognized as a self-limited human febrile disease in veterinarians, butchers and shepherds. A 1975 RVF outbreak in South Africa produced the first known outbreak of RVF with encephalitis and fatal hemorrhagic fever (WHO, 1985a).

Although RVF has generally been confined to sub-Saharan Africa, outbreaks occurred in 1977-78 in the irrigated region of Egypt with devastating consequences. It is estimated that between 20,000 and 200,000 cases and 600 deaths occurred. Tests confirmed that RVF had not previously occurred in Egypt (WHO, 1985a).

RVF has not yet been reported outside Africa. However, the 1977-78 outbreak in Egypt demonstrated that it can occur in new areas with devastating results. Although the natural reservoir of RVF is unknown, it is believed that cattle and sheep act as amplifiers of the disease. At least 26 species of mosquito have been implicated as potential vectors of the virus by isolation in laboratory experiments including C. pipiens (WHO, 1985b). Its penetration into Egypt represents movement into a totally new ecosystem and combined with the severity of the human epidemic, it demonstrates disastrous potential for areas outside Africa.

Yellow fever, which shares clinical features with other hemorrhagic fevers, but has a more severe hepatic involvement, is a viral disease endemic to the tropical regions of the Americas and Africa. It is transmitted by mosquitoes from monkey to man and from man to man. At present, yellow fever has been reported from ten Latin American countries with most cases coming from Bolivia, Brazil, Columbia, and Peru (WHO, 1985a; Monath, 1987).

Of particular importance is the outbreak of yellow fever in urban areas infested with A. aegypti, which is present in large parts of the United States. Several outbreaks of urban yellow fever have been traced to persons who were infected in forested areas and subsequently brought the disease to the city (WHO, 1985b).

Although an effective vaccine for yellow fever exists, supplies are not adequate. Should an outbreak occur in the continental United States, it has been predicted that supplies could not be produced in sufficient amounts to prevent an epidemic (Drs. Robert Shope and William Reeves, personal communication).

The changes in climate will increase the incidence and severity of skin infections and infestations. Skin infections, including dermatophytosis,[30] candidiasis,[31] streptococcal pyoderma, erythrasma, tinea versicolor,[32] and scabies are altered by temperature and humidity (Taplin et al., 1987). The importance of lifestyle and behavior upon contracting disease cannot, however, be dismissed.

Skin diseases were the single greatest cause of outpatient visits to Army medical facilities during the Vietnam War (Allen, 1977). When compared with temperature, relative humidity, and rainfall, outpatients' visits coincided with the mean monthly index values for rainfall and relative humidity, but were four months different from variations in monthly temperature (Figure 10). Thus, outpatient skin diseases were directly affected by rainfall and relative humidity, but not by temperature changes (Allen, 1977).

---

[30]The dermatophytes consist of three genera of fungi - Microsporum, Trichophyton and Epidermophyton. The infections caused by these fungi are usually referred to as ringworm or Tinea (Stein, 1983).

[31]Of the seven species of Candida known to infect man, the majority of infections are composed of Candida albicans. Although Candida albicans is a common inhabitant of the human body, it is increasingly a cause of serious infection (Stein, 1983).

[32]Tinea versicolor is caused by the infection of the superficial layer of the epidermis of the yeast Malassezia furfur (Stein, 1983).

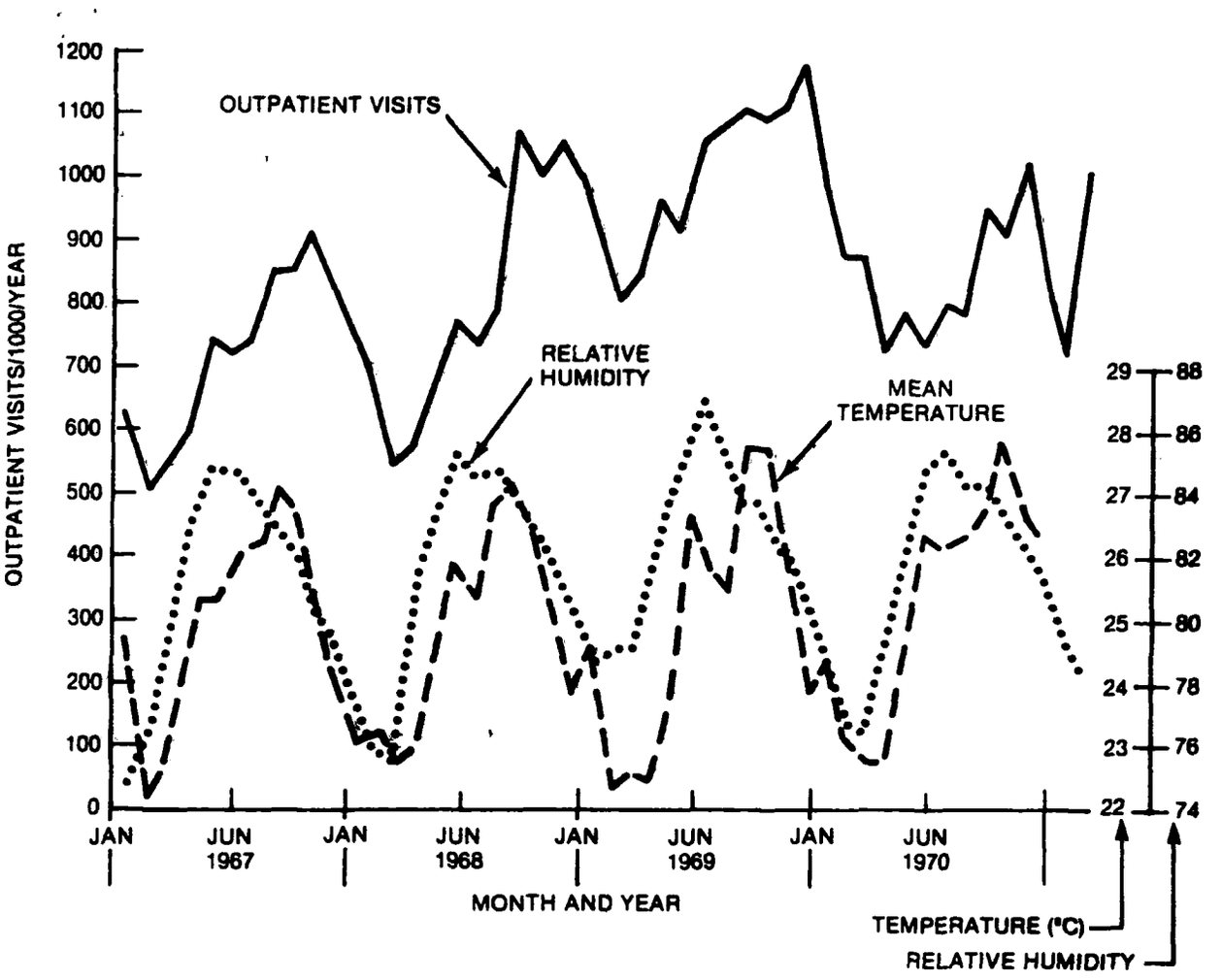


Figure 10. Outpatient visits for skin diseases in relation to mean monthly temperature, rainfall, and relative humidity for U.S. Army personnel in Vietnam, 1967-70.

Source: Allen (1977).

CHAPTER 4

POLICY IMPLICATIONS

A major focus of the two-day workshop was to address the issue of what policies would need to be implemented to lessen the increase in morbidity and mortality from infectious disease caused by climate change. It was felt that there was already a significant amount of information known that could be preventively used to combat the spread of infectious disease in the United States.

The workshop participants felt that disease and vector surveillance programs are a cornerstone to dealing with the issues. Several states have in place surveillance programs which are adequate to deal with the problems (e.g., the California Encephalitis Surveillance Program). However, these programs are in danger of not being able to deal effectively with the situation because of a decrease in state and federal funding and a lack of trained personnel. There are also many areas in the country where these programs do not exist and may need to be established.

A major concern of the workshop was the deterioration in the centers of excellence that could be called upon to spearhead drives against the increases in infectious disease. For example, the number of arboviral research units in the United States has decreased, and it is the belief of the workshop attendees that the expertise may not be present in sufficient quantities to deal effectively with the situation.

The workshop attendees found that these issues are multidimensional in nature, and effective solutions would also have to be interdisciplinary. It was, therefore, felt that interdisciplinary teams need to be established, so that the groups can be effectively integrated. The workshop felt that an effective integration necessitates working together for a period of time and, therefore, these groups need to be formed now.

The workshop participants expressed great concern that EPA's Endangered Species Act would limit pesticide use and thereby curtail the ability of various agencies to control vector-borne disease.

CHAPTER 5

SUMMARY AND CONCLUSIONS

As has been discussed, climate has a great impact on patterns of infectious disease in the extent of their geographic radius as well as in their seasonal duration. Although the importance of certain climatic conditions has been defined for a number of infectious diseases, it is not well understood how changes in climate, particularly gradual change, will affect disease patterns.

This report has reviewed what is known on many of the issues and has presented our best estimates of how climatic changes could alter the patterns of infectious disease in the United States. Without knowing exactly what changes in climate will occur (information which the EPA is in the process of generating), it is not possible to predict what the impacts of the global climate changes due to the emissions of "greenhouse gases" will be. In addition, recommendations are given below with regard to the data gaps which need to be filled.

ADDITIONAL RESEARCH NEEDED

In the course of developing this report, it was realized that there are many areas that need additional research. These research areas are outside the traditional health literature, but are issues that directly impact on the question of climatic change and infectious disease.

- Research as to the living conditions of the poor in the United States with specifics as to sanitation, differences between rural and urban poor as well as use of air conditioning and types of housing. All these elements impact on the interaction of the vector with man and will influence disease patterns.

- Research on the impact climatic change will have on intermediate animal hosts such as the white-footed mouse, white-tailed deer, and resident and migratory birds. As the range of the vector is tied to the range of the intermediate host, the increase or decrease in the animal host's range may correspondingly increase or decrease the area in which the disease is present.

- Research in behavioral science as to the impact of climatic change on man's daily life. This issue is related to the amount of time a person would be outdoors and in the vicinity of a disease-carrying vector.

- Research as to specific changes in agriculture patterns, forest growth and type of forests as these factors impact on the presence of vectors and animal hosts.

GLOSSARY

anopheline mosquitoes - mosquitoes of the genus Anopheles; many are malarial vectors.

antibiotic - a chemical derived from a fungus or bacteria which is inhibitory to other microorganisms.

antigen - substance which induces an immune response upon contact with the immune system

arbovirus - arthropod-borne virus.

arthropod - member of the invertebrate phylum Arthropoda which includes insects, crustaceans, spiders, and ticks.

candidiasis - infection caused by yeast species of the genus Candida, often Candida albicans (Stein, 1983).

CDC - Centers for Disease Control.

CE - California encephalitis.

chloroquinine - see quinine.

CNS - central nervous system.

coagulation - the clotting of blood.

coccobacillus - a type of bacteria.

definitive host - organism in which the infectious agent grows and sexually multiplies; for the diseases discussed in this paper, the definitive host is often a human.

Dengue fever - infectious disease caused by the dengue virus, which is transmitted by mosquitoes.

DEN-1, -2, -3, -4 - the four serotypes of the dengue virus.

dermatophytosis - infection caused by fungus of one of three genera: Microsporum, Trichophyton, or Epidermophyton; may be referred to as ringworm or Tinea (Stein, 1983).

desiccation - drying.

DHF - dengue hemorrhagic fever.

diapause - physiological state of suspended activity or arrested development that facilitates survival through a period of unfavorable conditions, but is initiated before the onset of these conditions (Bailey, 1982).

dysarthria - malformation or disturbance of a joint due to emotional stress, paralysis, or spasticity of muscles.

edema - swelling as a result of accumulation of fluid.

EEE - eastern equine encephalitis.

encephalitis - inflammation of the brain.

endemic - present in a certain area or among a specific population.

GLOSSARY (continued)

epithelium - layer of cells covering all free surfaces of an organism.

epizootic - a disease which simultaneously attacks or is present in a large number of animals.

erythema chronicum migran (ECM) - skin lesion consisting of an inflamed, red ring with advancing hardened edges leaving a central clear area; emanates from an insect bite; characteristic of Lyme disease.

erythrasma - eruption of reddish brown patches as a result of Corynebacterium minutissimum.

etiology - cause of disease.

extrinsic incubation (EI) period - interval between host's ingestion of the virus and subsequent transmission through biting; varies for each virus and vector.

febrile - relating to fever.

gram-negative - a defining characteristic of a group of bacteria; after being Gram stained, gram-negative cells do not readily retain crystal violet dye.

helminth - an intestinal parasite.

hemorrhagic - relating to or characterized by bleeding.

hepatic - relating to the liver.

host - organism from which infectious agent gains sustenance.

indigenous - originating in a particular area.

infectious feeding - introduction of the agent to the vector when the vector is feeing on the blood of an infected organism.

infectious agent - the virus, bacteria, protozoan, or other microorganism which induces disease.

infectivity threshold - concentration of virus that must be ingested by the vector in order for strains to become infective; varies among viruses and vectors.

JBE - Japanese B encephalitis.

larva - immature form of certain organisms (e.g., insects and ticks) which emerge from the egg.

Lyme disease - inflammatory infectious disease caused by the tick-transmitted spirochete, Borrelia burgdorferi.

meningeal - relating to the membranes surrounding the spinal cord and brain.

meningitis - inflammation of the membranes of the brain and/or spinal cord.

mesenteronal - relating to the layers of tissue surrounding the abdominal viscera.

mesic - characterized by a moderate amount of moisture.

GLOSSARY (continued)

nymph - immature form of certain organisms (e.g., ticks); the larva molts to become a nymph, which is adult-like in form though usually smaller.

pantropical - relating to areas which are predominantly tropical.

parenchymal - relating to the distinguishing cells of a gland or organ.

paresis - partial or incomplete paralysis.

periorbital - relating to the area around the eye socket.

photoperiod - duration of light.

plasmodia - members of the family Plasmodiae; blood parasites of vertebrates.

protozoa - unicellular eukaryotic microorganisms.

pupa - inactive form of an organism during which the larva transforms into an adult.

questing period - time when ticks--either nymphs or adults--are seeking hosts.

quinine/chloroquinine - antimalarial chemical effective against the asexual and red blood cell-attacking forms of the plasmodia.

resistance - ability of an organism to remain unaffected by a toxic substance.

retinitis - inflammation of the retina.

rickettsia - a type of coccobacillus (bacteria); all but one of this group are vector-borne parasites.

RMSF (Rocky Mountain spotted fever; also know as tick typhus) - infectious disease caused by the tick-borne bacteria Rickettsiae rickettsii

RVF - Rift Valley fever.

scabies - skin irritation accompanied by intense itching caused by the female Sarcoptes scabiei var. hominis burrowing into the skin.

sequela - effect of a disease.

serotype - subdivision of a species or virus identifiable on the basis of antigenic character.

SLE - St. Louis encephalitis.

spirochete - spiralling bacteria.

streptococcal pyoderma - a pus-forming skin infection caused by a member of the genus Streptococcus.

systemic - relating to the organism as a whole.

tinea versicolor - infection of the superficial layer of the epidermis by the yeast Melassezia furfur (Stein, 1983).

GLOSSARY (continued)

transovarial transmission - passage of infectious agent to eggs within the ovaries; larvae are subsequently infected.

transstadial transmission - transmission of agent between different stages in the life history of an organism.

vector - organism which transmits the infectious agent to the host; examples include ticks and mosquitoes.

VEE - Venezuelan equine encephalitis.

venereally - relating to sexual intercourse.

vertical transmission - see transovarial transmission.

viremia - the presence of a virus in the bloodstream.

WEE - western equine encephalitis.

yellow fever - acute, harmful infectious disease caused by a mosquito-borne virus.

REFERENCES

Allen, A.M. Skin Diseases in Vietnam, 1965-72. United States Army, 1977.

American Public Health Association. APHA. Control of Communicable Diseases in Man (ed.) Berenson, A.S. (1980)

Anon. Leads from the MMWR, Imported and indigenous Dengue fever - United States, 1986. JAMA 258:1712-1713 (1987a).

Anon. Leads from the MMWR; Dengue in the Americas, 1985. JAMA 257:166 (1987b).

Arfaa, F. Intestinal parasites among Indochinese refugees and Mexican immigrants resettled in Contra Costa County, California. J Fam Prac 12:223-226 (1981)

Bailey, C.L., Faran, M.E., Gargan, T.P., and Hayes, D.E. Winter survival of blood-fed and nonblood-fed Culex pipiens L. Am J Trop Med Hyg 31:1054-1061 (1982).

Barry, M., Craft, J., Coleman, D., Coulter, H.O., and Horwitz, N. Clinical findings in Southeast Asian refugees. JAMA 249:3200-3203 (1983).

Bos, E. Estimate of the number of illegal aliens: An analysis of the sources of disagreement. Pop Res Pol Rev 3:239-254 (1984).

Botkin, D.B., Janak, F.F. and Wallis, J.R. Some ecological consequences of a computer model of forest growth. J Ecol 60:849-872 (1972).

Bowen, G.S., Schultz, T.L., Hayne, C., and Parkin, W.E. Focus of Lyme disease in Monmouth County, New Jersey. Am J Epi 120:387-394 (1984).

Bozeman, M.F., Shirai, A., Humphries, J.W., et al. Ecology of Rocky Mountain spotted fever. 2. Natural infection of wild mammals and birds in Virginia and Maryland. Am J Trop Med Hyg 16:48 (1967).

Braunwald, E., Isselbacher, K.I., Persdorf, R.G., Wilson, J.D., Martin, J.B., and Fauci, A.S. Harrison's Principles of Internal Medicine. Eleventh Edition. McGraw-Hill (1988).

Brunetti, R., Fritz, R.F., and Hollister, A.C. An outbreak of malaria in California, 1952-1953. Am J Trop Med 3:779-788 (1954)

Burgdorfer, W., Lane, R.S., Barbour, A.G., Gresbrink, R.A., and Anderson, J.R. The western black-legged tick, Ixodes pacificus: A vector of borrelia burgdorferi. Am J Trop Med Hyg 34:925-930 (1985).

Cantanzaro, A. and Moser, R.J. Health status of refugees from Vietnam, Laos and Cambodia. JAMA 247:1303-1308 (1982).

CDC. Health Status of Indochinese refugees. MMWR 28:385-398 (1979a).

CDC. Survey of intestinal parasites-Illinois. MMWR 28:346 (1979b).

CDC. Viral hepatitis type B, tuberculosis and dental care of Indonesian refugees. MMWR 29:1-3 (1980c).

CDC. Health screening resettled Indochinese refugees-Washington, D.C., Utah. MMWR 29:4-11 (1980e).

CDC. Dengue-Cuba. MMWR 30:317 (1981).

CDC. USPHS Guidelines for medical examination of aliens. June, 1984.

CDC. Plasmodium viral malaria-San Diego County, California, 1986. MMWR 35:679-681 (1986a)

CDC. Update: Aedes albopictus infestation-United States. MMWR 35:649-651 (1986b).

CDC. Aedes albopictus introduction-Texas. MMWR 35:141-142 (1986c).

CDC. Aedes albopictus infestation-United States. MMWR 35:493-495 (1986d).

CDC. Dengue - the Americas, 1984. MMWR 35:51-57 (1986e).

CDC. Annual Summary, 1984. MMWR 33:54 (1986f).

CDC. Update: Aedes albopictus infestation-United States. MMWR 36:769-773 (1987).

Corwin, A.F. The numbers game: Estimates of illegal aliens in the United States, 1970-1981. Prepared for the House Judiciary Committee. Feb 1982.

D'Angelo, L.J., Bregman, D.J. and Winkler, W.G. Rocky Mountain spotted fever in the United States: Use of age-specific incidence to determine public health policy for a vector-borne disease. So Med J 75:3 (1982)

Dauer, C.C. and Faust, E.C. Symposium on malaria - Part 2. Malaria mortality in the United States, with especial reference to the Southeastern United States. So Med J 30:939-943 (1937).

de Zulueta, J. Man and malaria. In: Stanley, N.F., and Joske, R.A. (eds), Changing Disease Patterns and Human Behavior, Academic Press, New York (1980).

Ehrenkranz, N.J., Ventura, A.K., Cuadrado, R.R., Pond, W.C., and Porter, J.E. Pandemic dengue in Caribbean countries and the southern United States-past, present, and potential problems. N Eng J Med 285:1460-1469 (1971).

Eldridge, B.F. The effect of temperature and photoperiod on blood-feeding and ovarian development in mosquitoes of the Culex pipiens complex. Am J Trop Med Hyg 17:133-143 (1968).

EPA/UNEP. Effects of changes in stratospheric ozone and global climate. Vol. 1-4 (1986).

EPA. U.S.Environmental Protection Agency. Work Plan for a Report to Congress on the Effects of Global Climate Change. September 16, 1987 (1987)

Farhang, A.A., and Traub, R. Transmission of murine typhus rickettsiae by Xenopsylla cheopsis, with notes on experimental infection and effects of temperature. Am J Trop Med Hyg 34:555-563 (1985)

Faust, E.C. The 1930 status of malaria in the southern United States, as determined by mortality data. So Med J 25:544-548 (1932).

Faust, E.C. Malaria mortality in the southern United States for the year 1936. So Med J 31:816-818 (1938).

Faust, E.C. Malaria mortality in the southern United States for the year 1937. Am J Trop Med 19:447-455 (1939).

Finley, K. and Longshore, W.A. A progress report on five years of follow up of western equine and St. Louis encephalomyelitis cases in California with special reference to children. Trans Amer Neur Soc 83:45-49 (1958).

Gahlinger, P.M., Reeves, W.C., and Milby, M.M. Air conditioning and television as protective factors in arboviral encephalitis risk. Am J Trop Med Hyg 35:601-610 (1986).

Garvie, M.B., McKiel, J.A., Sonenshine, D.E., and Campbell, A. Seasonal dynamics of American dog tick, Dermacentor variabilis (Say), populations in southwestern Nova Scotia. Can J Zool 56:28-39 (1978)

Gillett, J.D. Direct and indirect influences of temperature on the transmission of parasites from insects to man. In: Taylor, A.E.R. and Muller, R. (eds), The Effects of Meteorological Factors upon Parasites, Blackwell Scientific Publications, London (1974).

Gillett, J.D. The behaviour of Homo sapiens, the forgotten factor in the transmission of tropical disease. Trans R Soc Trop Med Hyg 79:12-20 (1985).

Gratz, N.G. Mosquito-borne disease problems in the urbanization of tropical countries. CRC Crit Rev Environ Control 3:455-495 (1973)

Gubler, D.J. Dengue in the United States. MMWR 33(1SS):9SS-12SS (1983).

Habicht, G.S., Beck, G., and Benach, J.L. Lyme disease. Sci Am 256:78-83 (1987)

Hardy, J.L., Rosen, L., Kramer, L.D., Presser, S.B., Shroyer, D.A. and Turell, M.J. Effect of rearing temperature on transovarial transmission of St. Louis encephalitis virus in mosquitoes. Am J Trop Med Hyg 29:963-968 (1980).

Hardy, J.L., Houk, E.J., Kramer, L.D., and Reeves, W.C. Intrinsic factors affecting vector competence of mosquitoes for arboviruses. Ann Rev Entomol 28:229-62 (1983).

Hess, A.D., Cherubin, C.E., and LaMotte, L.C. Relation of temperature to activity of Western and St. Louis encephalitis viruses. Am J Trop Med Hyg 12:657-667 (1963).

Hoogstraal, H. Changing patterns of tick-borne diseases in modern society. Ann Rev Entomol 26:75-99 (1981).

Hoogstraal, H., Kaiser, M.N., Traylor, M.A., Gaindy, E., and Gaber, S. Ticks (Ixodidae) on birds migrating from Europe and Asia to Africa, 1959-1961. Bull WHO 28:235-262 (1963).

Immigration and Naturalization Service. INS. Statistical Yearbook (1985).

Johnson, R.T. Viral Infections of the Nervous System, Raven Press, New York; pp. 107-119 (1977).

Jones, M.J., Thompson, J.H., and Brewer, N.S. Infectious diseases of Indochinese refugees. Mayo Clin Proc 55:482-488 (1980)

Kramer, L.D., Hardy, J.L. and Presser, S.B. Effect of temperature of extrinsic incubation on the vector competence of Culex tarsalis for western equine encephalomyelitis virus. Am J Trop Med Hyg 32:1130-1139 (1983).

Lane, R.S. and Burgdorfer, W. Potential role of native and exotic deer and their associated ticks (Acari: Ixodidae) in the ecology of Lyme disease in California, USA. Zbl Bakt Hyg A 263:55-64 (1986).

Longstreth, J.D., Lill, P., Wiseman, J.A., and Laurenson, J. UV-B Induced Immunosuppression: Potential Impacts on Infectious Disease. Report prepared for U.S. EPA (1987).

Macdonald, G. The Epidemiology and Control of Malaria. Oxford University Press, Oxford (1957).

Main, A.J., Carey, A.B., Carey, M.G., and Goodwin, R.H. Immature Ixodes dammini (Acari: Ixodidae) on small animals in Connecticut, USA. J. Med Entomol 19:655-664 (1982).

Mauser, J.S., and Kramer, S. Epidemiology: An Introductory Text. (2nd ed.) W.B. Saunders Co., St. Louis, MO (1985).

McDade, J.E., and Newhouse, V.F. Natural history of Rickettsia rickettsii. Ann Rev Microbiol 40:287-309 (1986).

McEnroe, W.D. The restriction of the species range of Ixodes scapularis, Say, in Massachusetts by fall and winter temperature. Acarologia 18:618-625 (1977).

McGlynn, K.A., Lustbader, E.D., and London, W.T. Immune responses to hepatitis B virus and tuberculosis infections in Southeast Asian refugees. Am J Epi 122:1032-1036 (1985).

McLean, D.M., Grass, P.N., Judd, B.D, Cmirlova, D., and Stuart, K.M. Natural foci of California encephalitis virus activity in the Yukon Territory. Can J Pub Health 68:69-73 (1977).

McLean, D.M., Miller, M.A., and Grass, P.N. Dengue virus transmission by mosquitoes incubated at low temperatures. Mosq News 35:322-327 (1975a).

McLean, D.M., Grass, P.N., Miller, M.A., and Wong, K.S.K. Arbovirus growth in Aedes aegypti mosquitoes throughout their viable temperature range. Arch Virol 49:49-57 (1975b).

Monath, T.P. Yellow fever: A medically neglected disease. Report on a seminar. Rev Infect Dis 9:165-175 (1987).

Monath, T.P. Epidemiology. In: Monath, T.P. (ed.), St. Louis Encephalitis, APHA, Washington, D.C. pp. 239-312 (1980).

National Academy of Sciences (NAS). Changing climate. National Academy Press, Washington, D.C. (1983).

Nayar, J.K., Rosen, L., and Knight, J. Experimental vertical transmission of Saint Louis encephalitis virus by Florida mosquitoes. Am J Trop Med Hyg 35:1296-1301 (1986).

Nicholls, N. A method for predicting Murray Valley encephalitis in Southeast Australia using the southern oscillation. Aust J Exp Biol Med Sci 64:587-594 (1986).

Peters, R.L., and Darling, J.D.S. Potential effects of greenhouse warming on natural communities. In: Titus, J. (ed.), Effects of Changes in Stratospheric Ozone and Global Climate, Vol. 3, EPA, Washington, D.C. pp. 137-159 (1986).

Rosen, L. Transmission transovarienne des arbovirus par les moustiques. Med Trop 41:23-29 (1981).

Schulze, T.L., Bowen, G.S., Lakat, M.F., Parkin, W.E., and Shisler, J.K. Seasonal abundance and hosts of Ixodes dammini (Acari: Ixodidae) and other Ixodid ticks from an endemic Lyme disease focus in New Jersey, USA. J Med Entomol 23:105-109 (1986).

Schulze, T.L., Bowen, G.S., Bosler, E.M., Lakat, M.F., Parkin, W.E., Altman,. R., Ormiston, B.G., and Shisler, J.K. Amblyomma americanum: A potential vector of Lyme disease in New Jersey. Science 224:601-603 (1984).

Shope, R.E. Arbovirus-related encephalitis. Yale J Biol Med 53:93-99 (1980).

Solomon, A.M. and West, D.C. Simulating forest ecosystem responses to expected climate change in eastern North America: Applications to decision making in the forest industry. Publ. No. 2398, Environmental Science Division, Oak Ridge National Lab, Oak Ridge, Tenn. (1984)

Sonenshine, D.E., Peters, A.H., and Levy, G.F. Rocky Mountain spotted fever in relation to vegetation in the Eastern United States. Am J Epi 96:59-69 (1972)

Sonenshine, D.E., and Clifford, C.M. Contrasting incidence of Rocky Mountain spotted fever in ticks infesting wild birds in eastern U.S. Piedmont and coastal areas, with notes on the ecology of these ticks. J Med Entomol 10:497-502 (1973)

Spence, L., Artsob, H., Grant, L., and Th'ng, C. St. Louis encephalitis in southern Ontario: Laboratory studies for arboviruses. Can Med Assoc J 116:35-37 (1977)

Spielman, A., Wilson, M.L., Levine, J.F., and Piesman, J. Ecology of Ixodes dammini-borne human babesiosis and Lyme disease. Ann Rev Entomol 30:439-460 (1985)

Spielman, A., Levine, J.F., and Wilson, M.L. Vectorial capacity of North American Ixodes ticks. Yale J Biol Med 57:507-513 (1984)

Steere, A.C., Burtenhagen, N.H., Craft, J.E., et al. The early clinical manifestations of Lyme disease. Ann Intern Med 99:76-82 (1983a).

Steere, A.C., and Malawista, S.E. The epidemiology of Lyme disease, In: Lawrence, N.E., and Schulman, L.E. (eds). Current Topics in Rheumatology: Epidemiology of the Rheumatic Diseases, Gower Med Publ Ltd., New York p 33 (1983b).

Steere, A.C., and Malawista, S.E. Cause of Lyme disease in the United States: Locations correlated with distribution of Ixodes dammini. Ann Intern Med 91:730-733 (1979).

Stein, D.H. Superficial fungal infections. Ped Clin N A 30:545-561 (1983).

Taplin, D., Meinking, T.C., and Mertz, P.M. Skin infections in tropical climates: Current patterns and future predictions. Manuscript in preparation. (1987)

Thompson, W.H. and Beaty, B.J. Venereal transmission of La Crosse virus from male to female Aedes triseriatus. Am J Trop Med Hyg 27:187-196 (1978)

Turell, M.J., Reeves, W.C. and Hardy, J.L. Evaluation of the efficiency of transmission of California encephalitis viral strains in Aedes dorsalis and Aedes melanimon. Am J Trop Med Hyg 31:382-388 (1982).

Usinger, R.L. Entomological phases of the recent dengue epidemic in Honolulu. Pub Health Rep 59:423-430 (1944)

Watts, D.M., Burke, D.S., Harrison, B.A., Whitmire, R.E., and Nisalak, A. Effect of temperature on the vector efficiency of Aedes aegypti for Dengue 2 virus. Am J Trop Med Hyg 36:143-152 (1987)

Westrom, D.R., Lane, R.S., and Anderson, J.R. Ixodes pacificus (Acari: Ixodidae): Population dynamics and distribution on Columbian black-tailed deer (Odocoileus hemionus Columbianus). J Med Entomol 22:507-511 (1985).

WHO. Arthropod-borne and rodent-borne viral diseases. Tech Rep Ser #719. World Health Organization, Geneva, Switzerland (1985a).

WHO. Viral hemorrhagic fevers. Tech Rep Ser #721. World Health Organization, Geneva, Switzerland (1985b).

WHO. The biology of malaria parasites. Tech Rep Ser #743. World Health Organization, Geneva, Switzerland (1987).

Wilkinson, P.R. The distribution of Dermacentor ticks in Canada in relation to bioclimatic zones. Can J Zool 45:517-537 (1967).

Williams, L.L. Report of the subcommittee on malaria prevention activities, 1937. So Med J 31:818-819 (1938).

Williams, L.L. Symposium on malaria - Part 2; malaria prevention activities, 1936. So Med J 30:938-939 (1937).

Yasuno, M., and Tonn, R.J. A study of biting habits of Aedes aegypti in Bangkok, Thialand. Bull WHO 43:319-325 (1972).

APPENDIX

AGENDA OF WORKSHOP

MONDAY
NOVEMBER 9, 1987

8:30 - 9:00 am	Coffee
9:00 - 9:30 am	Opening remarks - Mr. Joel Smith, EPA; Mr. Dan Lasoff, EPA
9:30 - 10:00 am	Presentation - Ms. Eleanor Cross, Naval Medical Research Station, "Use of Climate and Weather for Predicting Vector-Borne Disease Distribution"
10:00 - 10:30 am	Presentation - Dr. Ronald Schwarz, TRAIN, Inc., "Anthropology and Medical Ecology in Infectious Disease Research"
10:30 - 10:45 am	Coffee
10:45 - 12:30 am	Discussion
12:30 - 1:30 pm	Lunch
1:30 - 2:00 pm	Presenation - Dr. Dan Haile, USDA, "Computer Simulation of Vector Population Dynamics and Disease Transmission"
2:00 - 2:30 pm	Presentation - Dr. Byron Wood, TGS Technology, "Use of Remote Sensing to Monitor the Effects of Climate Change"
2:30 - 5:00 pm	Discussion

AGENDA OF WORKSHOP

TUESDAY
NOVEMBER 10, 1987

8:30 - 9:00 am	Coffee
9:00 - 9:30 am	Presentation - Dr. David Taplin, University of Miami, "Effects of Climate on Skin Infections and Infestations"
9:30 - 10:00 am	Presentation - Dr. Paul Reiter, CDC, " Weather, Vector Biology and Arboviral Recrudescence"
10:00 - 10:30 am	Presentation - Dr. Thomas Chambers, St. Jude's, Memphis, "Seasonal Effects on Influenza Epidemics"
10:30 - 10:45 am	Break
10:45 - 12:30 am	Discussion
12:30 - 1:30 pm	Lunch
2:00 - 3:30 pm	Discussion
3:30 - 4:00 pm	Conclusion

Longstreth

GOAL OF THE PROGRAM AND ISSUES FOR THE WORKSHOP

GOAL:

The primary goals of the workshop were to determine which infectious diseases would become significantly greater human health problems due to climate change and to determine what public health policy decisions can presently be made to lessen the morbidity and mortality of these infectious diseases due to changes in climate. The workshop also had the goal the defining of a research agenda for the future.

ISSUES:

1. Which infectious diseases pose the greatest threat in terms of morbidity and mortality within the United States due to climate change?

2. To what climate factors do these infectious diseases show the greatest sensitivity and which elements (i.e., agent, vector, reservoir host, human host) are most sensitive?

3. How significant could the infectious disease problem become in the future if temperatures rise by 2 to 4°C and precipitation changes by $\pm$ 20 percent? What would a 1°C rise and a 10% change in precipitation do?

4. Are there any short or long term public health policy decisions currently being made which are likely to be impacted by climate change? Examples might be implementation of health screening of immigrants, or planning and/or implementing vector control programs.

5. What research needs are most critical in order to develop an assessment of the problem?

6. How would your answers change if these questions were applied to other developed countries? To underdeveloped countries?

1. Which infectious diseases pose the greatest threat in terms of morbidity and mortality within the United States due to climate change?

   The consensus of the workshop was that the United States would again have endemic (and epidemic) malaria and dengue (which is already present in Puerto Rico) in significantly greater numbers than is true today. These diseases would be mainly focused in the south-southeast and California, although the workshop did not rule out that these diseases would also occur in other areas of the United States.

   The workshop also believed that yellow fever would again become a problem, even though a vaccine exists for the disease. This is so because very little of the vaccine exists in the United States (or throughout the world) and the supplies would easily be exhausted in the United States should a major outbreak occur.

   It was generally felt that tick-borne diseases such as Lyme disease and RMSF would not become a greater problem due to climate change, but that the geographic focus of the diseases could change.

2. To what climate factors do these infectious diseases show the greatest sensitivity and which elements (i.e., agent, vector, reservoir host, human host) are most sensitive?

   The workshop felt that temperature was the most important climatic variable and that rainfall was the second most important variable. Small changes in these variables significantly impact on the geographic range and temporal duration of the vector which was considered to be the element most sensitive to climatic change.

3. How significant could the infectious disease problem become in the future if temperatures rise by 2 to 4°C and precipitation changes by $\pm$ 20 percent? What would a 1°C rise and a 10% change in precipitation do?

   The workshop felt that these relatively small changes would have a dramatic impact in changing patterns of infectious disease. This is due to the real impact that small changes in climate can have on the vector. It was felt that the most important impact would be that small increases in temperature would increase the geographic range in which the vector would be present throughout the entire year.

4. Are there any short or long term public health policy decisions currently being made which are likely to be impacted by climate change? Examples might be implementation of health screening of immigrants, or planning and/or implementing vector control programs.

   Several public health decisions are presently being made which will undermine the ability of the United States to adequately deal with the changes in patterns of infectious disease brought about by climatic change.

   One element is the lessening of the disease surveillance system in the United States. Without good surveillance data, preventive measures cannot be put into operation effectively.

   There are becoming less centers of excellence, such as the arboviral research centers, in the United States that are conducting research on these questions and that will be able to advise and respond as necessary.

   Due to the lack of career paths and adequate funding, fewer and fewer individuals, both in the United States and throughout the world, are being trained in vector eradication techniques. As climate change increases the importance of these programs, there will be a lack of adequately trained personnel to deal with the problem.

   The issues discussed are multi-dimensional in nature. There are at present no multi-dimensional centers or groups that have integrated epidemiologists, entomologists, behavioral scientists and physicians that will be able to respond to the new problems raised by climate change.

Longstreth

5. What research needs are most critical in order to develop an assessment of the problem?

   The workshop felt that the most critical research needs were the collection of better data as to the incidence of infectious disease, particularly in those groups which traditionally do not use the health care system, i.e., the poor and recent immigrants.

   It was also felt that greater surveillance of the various disease-carrying vectors is needed so that abatement programs can be maintained or developed as needed.

6. How would your answers change if these questions were applied to other developed countries? To underdeveloped countries?

   In relation to other developed countries, it was generally felt that the same or similar problems as predicted for the United States would occur in those countries. Similar influxes of immigrants from highly endemic areas occur throughout Europe as in the United States, and a similar strain on the public health systems of those countries is presently taking place.

   The workshop believed that the impact of climate change on patterns of infectious disease in underdeveloped countries would be significantly greater than in developed countries. This was because the public health systems and vector abatement programs in these countries are already overburdened, and any additional burdens on these systems will result in significantly greater incidence of disease. In addition, the sanitation and nutrition levels in many of the underdeveloped countries will promote a greater incidence of infectious disease than in the more developed countries.

WORKSHOP ATTENDEES

Dr. Joan Aron
Department of Population Dynamics
Johns Hopkins University School of Public Health
615 North Wolfe
Baltimore, MD 21205
301/955-3708

Dr. Thomas Chambers
St. Jude's Children's Medical Research Center
Department of Virology
Box 318
Memphis, TN 38101
901/522-0300

Dr. James Childs
Department of Immunology and Infectious Disease
Johns Hopkins University School of Public Health
615 North Wolfe
Baltimore, MD 21205
301/955-3708

Dr. Jeanne Courval
Division of Epidemiology
School of Public Health
Columbia University
600 West 168 Street
New York, NY 10032
212/305-3921

Ms. Eleanor Cross
Naval Medical Research
Mail Stop 7
Bethesda, MD 20814
301/295-1731

Ms. Margo Daniel
United States Environmental
Protection Agency
Washington, DC
202/382-2776

Dr. I.F. Goldstein
School of Public Health
Columbia University
600 West 168 Street
New York, NY 10032
212/928-7674

Dr. John Grayzell
ST/RD/RRD
Room 608
Dept. of State
Washington, DC 20523
202/235-8860

Dr. Dan Haile
USDA
1600 SW 23rd Drive
Box 14565
Gainesville, FL 32604
904/374-5928

Dr. Charles Hughes
Department of Family and Preventive Medicine
University of Utah Medical Center
Salt Lake City, UT 84132
801/581-5310

Ms. Terri Lavin
Department of Geography
University of Delaware
Newark, DE 19716

Dr. Janice Longstreth
ICF/Clement Associates
9300 Lee Highway
Fairfax, VA 22031
703/934-3102

Ms. Terry Meinking
Department of Dermatology and Cutaneous Surgery
University of Miami School of Medicine
Box 016960 R-117
Miami, FL 33101
305/547-6214

Dr. Greg Mertz
Tufts School of Veterinary Medicine
200 Westboro Road
North Grafton, MA 10536
617/839-5302

Ms. Marilyn Milby
School of Public Health
Warren Hall
University of California
Berkeley, CA 94720
415/642-3938

Dr. William Reeves
School of Public Health
Warren Hall
University of California
Berkeley, CA 94720
415/642-3938

Dr. Paul Reiter
CDC Laboratory
GPO Box 4532
San Juan, PR 00936
809/781-3636

Dr. Ronald Schwarz
TRAIN, Inc.
2816 Cheswolde
Baltimore, MD 21209
301/358-9561

Dr. Robert Shope
Yale University School of Medicine
Department of Epidemiology and Public Health
Box 3333
New Haven, CT 06510
203/785-4821

Dr. David Slade
Deputy Director
Physical and Technical Research Div. ER-74
Office of Health and Environmental Research
Department of Energy
Washington, DC 20545
202/353-4375

Dr. Chip Stem
Tufts School of Veterinary Medicine
200 Westboro Road
North Grafton, MA 10536
617/839-5302

Dr. David Taplin
Department of Dermatology and Cutaneous Surgery
University of Miami School of Medicine
Box 016960 R-117
Miami, FL 33101
305/547-6214

Ms. Kathleen Valimont
Department of Geography
University of Delaware
Newark, DE 19716

Dr. Byron Wood
TGS Technology Inc.
Mail Stop 242-4
NASA
Ames Research Center
Moffit Field, CA 94035
415/694-6184