UC-48
LBL-21625 c.1

Lawrence Berkeley Laboratory
UNIVERSITY OF CALIFORNIA

RECEIVED
LAWRENCE
BERKELEY LABORATORY

JUL 2 1986

LIBRARY AND
DOCUMENTS SECTION

CARBON DIOXIDE INCREASE AND HUMAN HEALTH: DATA AND RESEARCH REQUIREMENTS FOR DETERMINING CONSEQUENCES

M.R. White, I. Hertz-Picciotto, and M.E. Johnston

May 1986

For Reference

Not to be taken from this room

Donner Laboratory
Biology & Medicine Division

LBL-21625 c.1

Prepared for the U.S. Department of Energy under Contract DE-AC03-76SF00098

DISCLAIMER

This document was prepared as an account of work sponsored by the United States Government. While this document is believed to contain correct information, neither the United States Government nor any agency thereof, nor the Regents of the University of California, nor any of their employees, makes any warranty, express or implied, or assumes any legal responsibility for the accuracy, completeness, or usefulness of any information, apparatus, product, or process disclosed, or represents that its use would not infringe privately owned rights. Reference herein to any specific commercial product, process, or service by its trade name, trademark, manufacturer, or otherwise, does not necessarily constitute or imply its endorsement, recommendation, or favoring by the United States Government or any agency thereof, or the Regents of the University of California. The views and opinions of authors expressed herein do not necessarily state or reflect those of the United States Government or any agency thereof or the Regents of the University of California.

CARBON DIOXIDE INCREASE AND HUMAN HEALTH: DATA AND RESEARCH REQUIREMENTS FOR DETERMINING CONSEQUENCES

(Including A Bibliography for Additional Reading)

By

Margaret R. White, Irva Hertz-Picciotto, and Muriel E. Johnston

Division of Biology and Medicine
Lawrence Berkeley Laboratory
University of California
Berkeley, California 94720

TABLE OF CONTENTS

1. BACKGROUND 1
   1.1 Carbon Dioxide 1
   1.2 Carbon Dioxide and Trace Gas Increases 1
   1.3 Research on Carbon Dioxide 2
      1.3.1 Carbon Cycle 4
      1.3.2 Climate Effects 4
      1.3.3 Vegetation Response 6
      1.3.4 Indirect Effects of Carbon Dioxide Increase 7
2. AIMS, LOGIC, AND LIMITATIONS OF THE REPORT 9
3. CLIMATE AND WEATHER 13
   3.1 Climate and Weather Variables Relevant to Human Health 13
   3.2 Effects of Slow Versus Fast Climate Change 16
   3.3 Changes in the Variability of Weather 17
4. BASIC MECHANISMS IN HUMANS 17
   4.1 Thermoregulation, Acclimation, and Adaptation 17
   4.2 Biochemistry and Physiology 21
   4.3 Birth Defects 22
5. MORTALITY AND MORBIDITY RELATED TO CLIMATE/WEATHER 24
   5.1 Seasonality of Mortality and Morbidity 24
      5.1.1 Mortality from all Causes 26
      5.1.2 Mortality from Heart Disease 27
      5.1.3 Mortality from Cerebrovascular Disease 33
      5.1.4 Mortality and Morbidity from Respiratory Disease 35
      5.1.5 Mortality from Cancer 37
      5.1.6 Fetal and Infant Mortality 39
      5.1.7 Mortality from Other Causes 40
   5.2 Heat Waves 42
6. SPORADIC, EXTREME WEATHER EVENTS 53

7. AIRBORNE MATERIALS RELATED TO HUMAN HEALTH 54
7.1 Bacteria 56
7.2 Viruses 58
7.3 Fungi 59
7.4 Pollen 61
8. SEASONAL DISEASES CAUSED BY MICROORGANISMS 62
8.1 Airborne Diseases 62
8.2 Human Carrier Diseases 64
8.3 Vector-Borne Viral and Bacterial Diseases 65
8.4 Parasitic Diseases 66
8.4.1 Vector-borne Parasites 66
8.4.2 Parasites Requiring Intermediate Hosts 68
8.4.3 Parasites Which Need No Intermediate Host or Vector 69
9. NUTRITION 70
9.1 Climate and Weather Effects on Agriculture 70
9.2 Interactions of Disease and Nutrition 71
10. WATER 71
10.1 Quantity of Water 72
10.2 Quality of Water 72
11. SHELTER 74
12. AIR POLLUTION 75
13. RECREATION 75
14. SUMMARY 76
14.1 Seasonality of Mortality from Organic Diseases 77
14.2 Airborne Infectious Diseases 79
14.3 Human Carrier Disease 80
14.4 Vector-Transmitted and Parasitic Diseases 81
14.5 Airborne Spores, Fungi, and Pollen 82
14.6 Biochemical and Physiological Parameters 82
14.7 Conception and Birth Defects 82
14.8 Undernutrition and Malnutrition 83
14.9 Water Resources 83
14.10 Shelter 83
14.11 Air Pollution 84
14.12 Recreational Areas 84

15. DATA AND RESEARCH NEEDS 85
15.1 Climate Data Needs 85
15.2 Carbon Cycle Data Needs 85
15.3 Medical and Biological Research 86
15.3.1 Organic Diseases 87
15.3.2 Thermoregulation and Acclimation 88
15.3.3 Birth Defects and Infant and Fetal Deaths 89
15.3.4 Bacteria, Viruses, Parasites and Allergens 89
15.3.5 Nutrition and Disease 90
15.3.6 Shelter and Workplace 90
15.3.7 Recreational and Vacation Areas 90
ACKNOWLEDGMENT 91
REFERENCES 100
BIBLIOGRAPHY FOR ADDITIONAL READING 112

List of Figures

Figure 1.	Pathways by which CO_2-induced Climate Change may affect Human Health	14
Figure 2.	Historical Changes in Seasonal Variation Patterns of Mortality	25
Figure 3.	Generalized Relationship of Mortality to Temperature	27
Figure 4.	Relationship of Monthly Mortality Ratio to Monthly Mean Temperature	29
Figure 5.	Relationship of Temperature to Heart Disease Mortality, U.S. Cities: 1962-1966	31
Figure 6.	Relationship of Temperature to Stroke Mortality, U.S. Cities: 1962-1966	34
Figure 7.	Comparison of the Relationship of Death Rate from Cerebrovascular Disease to Temperature in London, Tokyo, and New York: 1960-1964	36
Figure 8.	Temperature and Death Rate During Extremely Hot Weather for Various U.S. Cities in 1936	43

List of Tables

Table 1.	Data from Heat Wave Studies	44
Table 2.	The Number of Deaths Per 1,000 Population by Age, Sex, Race, in St. Louis City, Missouri, for July 1966 (Annual Basis) and for the Year 1965	46
Table 3.	Mortality During Three September Heat Waves in Los Angeles	47
Table 4.	Knowns and Uncertainties Regarding Principal Climate Effects on Human Health	92-99

1. BACKGROUND

1.1 Carbon Dioxide

Carbon dioxide (CO_2) is a natural and essential constituent of Earth's atmosphere. It is a byproduct of human and animal metabolism and an essential nutrient for plants. Its presence in the atmosphere, along with water vapor and other trace gases, is responsible for maintaining the Earth's surface temperature within a range that supports life. This results from the fact that these gases reduce the rate of loss to space of the thermal energy emitted from the Earth's surface and the lower atmosphere.

Some of the natural sources of CO_2 are animal and human metabolism and the decay or burning of organic matter. Natural sinks for CO_2 include growing plants (especially forests), soils, and the oceans. Until recent history there appears to have been a dynamic balance between the natural sources and sinks of CO_2.

1.2 Carbon Dioxide and Trace Gas Increases

For approximately 10,000 years before the 19th Century, it appears that CO_2 concentration was relatively stable between 260 and 290 parts per million (ppm). In the 1800's, the atmospheric concentration of CO_2 began to increase. This is attributed to deforestation for agricultural expansion, to mechanized agriculture, urbanization with large population growth, and probably most importantly to the very large increase in the use of fossil fuels (i.e., coal, oil, and gas). Fossil fuel energy consumption increased approximately 10 fold from 1900 to 1984. The concen-

tration of atmospheric CO_2 has increased by about 25% in the last 150 years and it may increase to more than twice its current concentration within the next 100 years or so. In 1984, the concentration had reached 345 ppm. This increase in atmospheric CO_2 concentration is expected to have two major direct effects on the biosphere. First, it will increase the Earth's temperature. Second, it will affect vegetation because CO_2 is a fertilizer for plants. These direct effects will initiate many indirect effects which will affect mankind's environment and activities. Obviously, mankind is changing the environment by his actions.

In addition to increased atmospheric CO_2, trace gases are being introduced into the atmosphere by human activities. Some examples are methane, oxides of nitrogen and chlorocarbons. Although their concentrations are much lower than that of CO_2, model calculations suggest that their combined effects (including the effects of reactions with ozone) on climate could be as much as those estimated from the expected increase in CO_2. The effects of these gases are not considered in this study but it must be kept in mind that they also may be a source of climate change.

1.3 Research on Carbon Dioxide

Concern about the consequences of increasing CO_2 has led to a marked increase in research in this area in the past 10 years. The Department of Energy is the lead agency for this research in the United States but many other institutions and agencies both in the U.S. and abroad are participating in this research.

To determine the status of knowledge on the CO_2 issue and the directions research should take in the future, the Carbon Dioxide Research Division of the Department of Energy determined that it would be helpful to summarize the current state-of-the-art of knowledge and research. Consequently four state-of-the-art reports and two companion reports were planned. After several years of work and with input from many authors, both from the U.S. and abroad, these reports are now being published and are as follows:

Direct Effects of Increasing Carbon Dioxide on Vegetation (Strain and Cure, 1985)

Atmospheric Carbon Dioxide and the Global Carbon Cycle (Trabalka, 1985)

Projecting the Climatic Effects of Increasing Carbon Dioxide (MacCracken and Luther, 1985b)

Detecting the Climatic Effects of Increasing Carbon Dioxide (MacCracken and Luther, 1985a)

Characterization of Information Requirements for Studies of CO_2 Effects: Water Resources, Agriculture, Fisheries, Forests, and Human Health (White, 1985)

Glaciers, Ice Sheets and Sea Level: Effects of a CO_2-Induced Climatic Change (National Research Council, 1985)

The reader is referred to these reports for detailed information on the current status of CO_2 research. For purposes of introducing the subject matter of this report a very brief discussion follows.

1.3.1 Carbon Cycle

The global carbon cycle has been defined as the dynamic balance among global atmospheric sources and sinks which determines the rate of increase in the atmospheric CO_2 concentration. The major aim of carbon cycle research is to compute a balanced global carbon budget for the contemporary period. Research to determine the role of each source and sink is being actively pursued. The concentration of atmospheric CO_2 is being continuously monitored in a number of sites around the world. Models are being developed to accurately depict quantitative rates of carbon exchange among the major global reservoirs. Estimates of the amounts of CO_2 being generated by the use of fossil fuels and deforestation have been made. It has been found that the observed increase in atmospheric CO_2 is less than the estimated release of CO_2 from fossil fuel consumption and deforestation. Much of this CO_2 must be going into other reservoirs but to date it has not been possible to accurately determine the exact extent to which various reservoirs are involved. A better understanding of basic biological, chemical, and physical processes responsible for carbon cycling is needed. Extensive field and modeling research are planned and the results of that research will be necessary to achieve this understanding. Thus it is currently not possible to predict with any degree of certainty either the future rate of buildup of CO_2 in the atmosphere or its eventual maximum concentration.

1.3.2 Climate Effects

Climatalogists are in agreement that in theory an increase in atmospheric CO_2 should increase the global average near surface tempera-

ture to an extent that may be significant. Since atmospheric CO_2 has already increased by approximately 25%, it was thought that a CO_2-induced increase in temperature might be detectible at this time. Although there has been considerable research in an effort to determine whether, in fact, an increase has occurred, it has not been clearly identifiable to date, i.e., a cause and effect relationship between increased CO_2 and climate change has not been demonstrated. An effort to improve data bases and modeling is needed to establish this relationship.

Models which simulate the interactions among climate's most important processes have been developed and improved greatly in the past few years, but there are still large uncertainties because many processes (for example, cloud generation, atmosphere-ocean interactions) are not adequately treated. Thus the models still contain many important approximations and simplications. Nonetheless models do indicate that the global average temperature should increase. Estimates of the amount of increase fall between 1.5 and 4.5°C depending on the model. There are indications that the change in temperature (and other climate parameters) is likely to be larger in high latitude regions than in low latitude regions. It is expected that there will be changes in regional and seasonal precipitation patterns and in the variability of climate and weather, including the frequency of extreme events.

These projections, while important, are not adequate for predicting the consequences of climate change. While the models show similarity in their projections of the latitudinal distribution of the temperature change, they do not yet agree on the broad longitudinal seasonal and regional patterns of the projected changes. Comprehensive, verified models of the global climate system, that can estimate past and future

rates of climate change, are presently under development but results are not yet available. Thus the rate at which climate will change is not yet predictable. In addition it is not currently possible to predict the frequency distribution and variability of temperature and precipitation. This missing information, that is, extent of regional and seasonal climate change, rate of climate change, and changes in the variability of climate and weather, including occurrence of extreme events, is needed for determining the ecological and societal consequences of increasing CO_2.

1.3.3 Vegetation Response

Plants produce food and fiber through the process of photosynthesis. Essentials for the photosynthetic process are light and carbon from CO_2. CO_2 is assimilated by plants from the surrounding atmosphere. It has long been known that some plants will grow more rapidly in greenhouses where the CO_2 concentration has been enhanced over that of ambient air. However, intensive research to elucidate the effects of rising global CO_2 on plants has been initiated only recently. Very few crop species have been studied and those for a very short time. Little information is available to date on native species and ecosystems. Methodology and modeling approaches for determining the effects of elevated CO_2 have improved but still need much work.

Some of the physiological processes directly affected by CO_2 are not well understood nor are the nutrient requirements to sustain increased photosynthesis and growth at enriched levels of CO_2. Water-use efficiency of some plants is expected to increase. However, not all of

the species studied respond in the same manner or magnitude. Plant community composition will probably change but it is not yet possible to determine which plants may be dominant. This is especially important with respect to weed competition with crops. There may be changes in the nutrient value of crops. Whether there will be changes in plant vulnerability to insects and diseases is currently uncertain. Thus, although it appears that increased ambient CO_2 will accelerate the growth and increase the water-use efficiency of some plants, there are still many uncertainties about the extent to which plants will be benefited.

1.3.4 Indirect Effects of Carbon Dioxide Increase

There may be many effects resulting from CO_2-induced climate and vegetation change. Changes in both climate and vegetation will affect, for example, agriculture, water resources, fisheries, and unmanaged ecosystems. Human health will be affected by climate change and by changes in, for example, agriculture. These changes, in turn, may affect economics and other societal activities (for example, land use and recreational activities). Until there is more definitive information on climate change and vegetation response it is not possible to quantitate these indirect effects of CO_2 increase, though scenario studies have pointed out the possibilities for and the potential importance of some of these effects. Because of the current uncertainties about direct effects, no impact analysis of indirect effects can yet be done. However, due to the importance of some of the indirect effects in determining the ultimate consequences to humans of increasing CO_2 it was decided that some of the major potential effects should be examined. The aim was to

determine what information and research will be required to quantitate the effects from 1) the other CO_2 research programs and 2) the field of the indirect effect. The volume, Characterization of Information Requirements for Studies of CO_2 Effects: Water Resources, Agriculture, Fisheries, Forests, and Human Health (White 1985), was the result of that study. The technical information on human health in that volume is incorporated into this report. However, to keep the human health chapter of the D.O.E. report from becoming too lengthy, many of the pertinent references which were collected could not be used. This report is therefore published with an extensive bibliography for additional reading for those people interested in pursuing specific aspects of possible effects of climate change on human health. Even though extensive, it must be pointed out that the references and bibliography are certainly not exhaustive. There are, for instance, many more references to studies of climate effects on such diseases as schistosomiasis, malaria and encephalitis and to other parasitic, viral, and bacterial diseases which were not discussed. These were not referenced or discussed because we felt that the material that was referenced was adequate to illustrate the major directions of climate effects on human health. Also, though a number of abstracts of literature in foreign languages were read, there was not time to obtain translations of the articles for inclusion in the study; in most cases these reinforced or elaborated information already obtained. The other difference in the technical material from that in the human health chapter in the DOE report is that the summary and the data needs are separated here whereas they were combined in that report for the sake of conciseness.

2. AIMS, LOGIC AND LIMITATIONS OF THE REPORT

The purpose of this study was to determine the data and information needed to eventually define the consequences on human health of elevated CO_2. To attain this purpose, current knowledge of the effects of climate and weather on human health was reviewed.

An increase in atmospheric CO_2 is predicted to have several effects which may have an influence, directly and indirectly, on human health. These include climate change, changes in vegetation (due to changes in fertilization and water-use efficiency of plants), changes in ocean chemistry (due to increases in dissolved CO_2), and possibly slight changes in human biochemistry and physiology (due to breathing air with elevated concentrations of CO_2).

Changes in vegetation may affect water availability for agriculture and human use, human nutrition, materials for shelter, and so forth, by affecting water resources, agriculture, and forests. Changes in ocean chemistry may affect fisheries that in turn may affect nutrition, especially in some of the developing regions of the world.

It has been suggested that breathing elevated concentrations of CO_2 may change human physiology and biochemistry even though if atmospheric CO_2 concentration were quadrupled (to approximately 1200 parts per million [ppm]), it would still be only about 1/40th of the concentration of CO_2 in the air expired from the lungs (approximately 5% or 50,000 ppm). Humans have been exposed to much higher concentrations of CO_2 than those expected in the atmosphere with no persistent deleterious effects. However, these exposures were intermittent or for periods of six months or less. Two reviews of this literature (Bland et al. 1982; U.S. Department of Energy 1982) indicate that, if there is an effect of breathing

very low but elevated concentrations of CO_2 over prolonged periods, it is probably extremely small. In addition, the Natioanl Institute of Occupational Safety and Health's (NIOSH) recommended exposure limit to CO_2 is 10,000 ppm for up to 10h/d (U.S. Department of Health and Human Services 1985). Thus, this possible effect of breathing elevated CO_2 will not be discussed further in this document though eventually long-term, low level laboratory studies may be needed to determine whether, in fact, the effects are innocuous.

This report, therefore, concentrates on the effects of climate change on humans. Climate and particularly the weather elements influence human health because some diseases are quite prevalent in some climates but either less prevalent or absent in others. Increased atmospheric CO_2 is expected to raise the average global temperature by about 1.5° to 4.5°C. In temperate zones this increase is expected, on average, to be about equivalent to the global average while increases in tropical zones are expected to be smaller and increases in the polar areas are expected to be larger. Thus, the boundaries of the tropics may extend into regions now designated as semitropical, part of the current temperate zones may become semitropical, and so on. There are, as yet, no firm predictions regarding seasonal temperature changes in specific regions or regarding changes in other meteorological variables such as humidity and precipitation. In addition it is not known whether the variability of climate or weather will change, that is, whether there will be fewer or more periods of heavy or light precipitation, fewer or more excessively hot periods, and so on. Because of these uncertainties it is currently impossible to predict the impacts of CO_2-induced climate change on human health. At this time we can only point out some of the

known influences of climate, seasons, and weather on human health and disease-causing organisms and specify the data and information needed, both from the CO_2 research program and from human health research, to eventually assess the impacts of CO_2-induced climate change on human health.

Prior to discussing the relationships of meteorological variables to human health it must be pointed out that non-meteorological factors currently do, and in the future will, modify the effects of meteorological variables on health. Humans currently live in extremely cold and extremely hot climates and survive by modifying their ways of life (clothing, shelter, food, etc.). They will, of course, continue to do so under a CO_2-induced climate change. In addition to these behavioral adaptations, there are passive mechanisms in the human body, collectively termed the thermoregulatory system, which enable humans to adjust to some extent to adverse meteorological conditions.

Furthermore, though climate provides the underlying environment which determines the potential for prevalence of some diseases in specific climates, human circumstances, attitudes, and interventions (or lack thereof) can, in many cases, modify prevalence of these diseases. For example, the socioeconomic status of regions and individuals, cultural practices, quality of health care and education, and effort and funds expended for eradicating disease-causing organisms have a major influence on the prevalence of some diseases. Thus, although we can identify (but not always quantify) influences of meteorological variables on human disease, in many cases the end result of climate change on human health will be modified by many other factors.

Some caveats also need to be stated with regard to the pertinent information in the literature. First, it would be desirable, for purposes of this study, to know the extent to which climate and weather variables influence the onset and progression of organic diseases. However, mortality (death) statistics are much more readily available than are morbidity (illness) statistics, with the exception of some communicable and parasitic diseases that are required to be reported to public health authorities. For this reason, the majority of studies designed to measure the health of populations use mortality statistics. These studies state the causes of death and where and when the person died; thus these data can be matched with environmental conditions at the time of death and inferences can be made regarding whether these conditions influence the death rate from particular diseases. These mortality statistics do not tell us when the disease, which eventually killed these people, originated or what the environmental conditions were at that time. Knowledge is meager with respect to the influence of climate or the prevailing weather conditions on the onset and early progression of some diseases.

The second caveat is that, in some cases, the literature referred to in this report is relatively old. This is a consequence of the ebb and flow of interest in, and funding for, different areas of research. In some cases very little research has been done in a particular field in recent years. This older literature is still pertinent to the effects of climate and weather. In addition, there are many other studies that would reinforce the literature quoted, but which are not included for the sake of conciseness.

The third caveat is that, until the CO_2 issue arose, there was no particular reason to study the effects that climate change might have on human health in a particular region because climate change was not expected to occur, except possibly extremely slowly over hundreds of years. Therefore, most studies investigated the effects of large variations in weather (heat waves, cold spells, etc.) but did not look for possible effects of persistent climate change in a region.

This report describes some of the ways that climate and weather influence human health, some of the modifying factors, and the information and data needed to initiate studies of the possible consequences of elevated atmospheric CO_2 on human health. Figure 1 indicates some pathways by which climate change may modify human health.

3 CLIMATE AND WEATHER

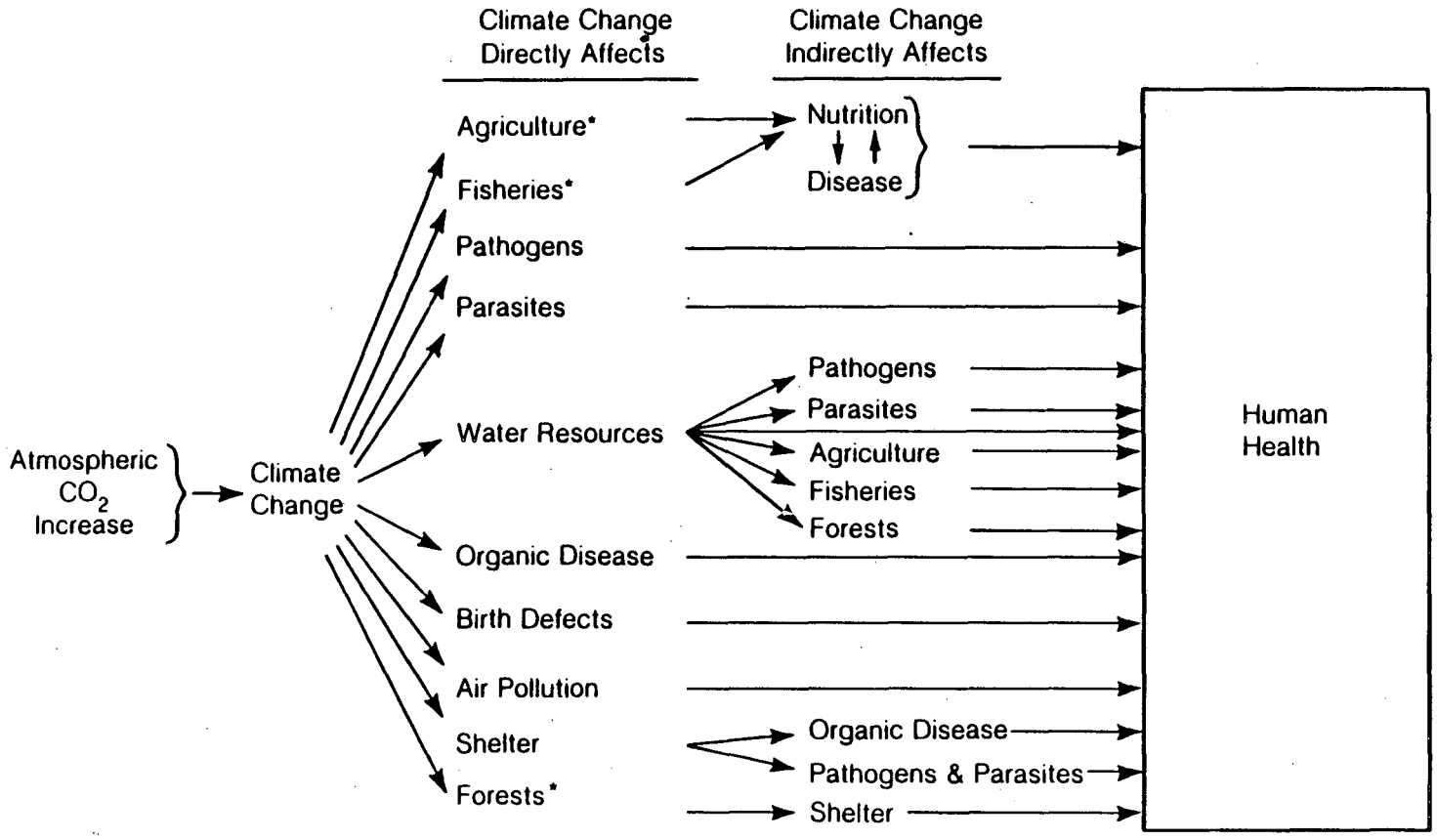
3.1 Climate and Weather Variables Relevant to Human Health

All climate and weather variables have some influence on human health. The effect may be either directly on the human body or through effects on disease-causing organisms or vectors of disease-causing organisms. Although the effects of variation of one weather element may be examined in a particular study, it should be kept in mind that that element does not act independently of other elements, for example, changes in humidity modify the effects of temperature.

Temperature at both extremes of the scale, either excessively hot or excessively cold, affects human health. The acute effects of excessive cold are frostbite and, for prolonged exposure, death from the lower-

Figure 1

Pathways by which CO_2-Induced Climate Change may Affect Human Health



XBL 845-8422

*Agriculture and forests may also be directly affected by increase in the concentration of atmospheric CO_2. Fisheries may be affected by CO_2-induced changes in ocean chemistry.

ing of body temperature. The acute effects of excessive heat, such as heat stroke and heat edema, if not treated quickly, can cause death. People with chronic disease, especially the elderly, are very susceptible to aggravation of the disease state from both cold and excessively hot weather. Temperatures in tropical, subtropical and warmer temperate zones are ideal for the survival and propagation of some bacterial, viral, and parasitic diseases. Temperature also affects human health indirectly by affecting agriculture, fisheries, and water resources.

The effects of high temperatures on human health are modified by the amount of moisture in the air (humidity). The degree to which thermoregulatory mechanisms must operate to keep body temperatures normal varies with humidity. Human comfort (feeling of well-being) is also affected by humidity. Pathogenic bacteria, viruses, parasites, and parasitic vectors have ideal humidities at which they survive and multiply to their best advantage.

Precipitation may increase humidity with consequent effects on humans. In cold weather, it may add to chilling of the human body, thus making the human more susceptible to disease, or it may aggravate chronic diseases. Depending on the amount and timing, it may modify the ecological habitat of parasites, their hosts, and insect vectors such that their growth and survival are affected. If there is too little or too much precipitation or the timing of it's occurrence is wrong, there may be crop damage. This may lead to food shortages or an increase in the cost of food, thus resulting in under- or malnutrition.

Abrupt changes in weather, such as those associated with the passage of a weather front, have been implicated in such things as feelings of malaise and onset of headaches, which are indicative of physiological

or biochemical disturbances. Some evidence has also accumulated indicating that these weather changes may be related to the onset of some diseases, such as common colds.

Wind in combination with temperature and humidity can affect human thermoregulation. It can also be a means of spreading the causative agents of disease, insect vectors, and allergens.

3.2 Effects of Slow Versus Fast Climate Change

The rate of climate change will be a major factor in the intensity of the effects and the efficiency of adaptive (passive) changes and ameliorative actions. Physiological changes begin within minutes of an abrupt change in temperature, but it may take much longer to become completely adapted to a changed climate. The types of clothing used to protect against climate stress can be changed rapidly, but architectural changes in housing, particularly in structures already built, may not be made until the structure is demolished and a new one built. New strains of plants can be developed which will tolerate a new climate, but this development may take a number of years. New water storage facilities take a number of years to plan and build. Thus, if there are appreciable and rapid changes in climate, adaptation may be difficult. If there is a very gradual change, over 25 to 50 years, many changes can occur, either passively or actively, to offset possible detrimental effects.

3.3 Changes in the Variability of Weather

Climate changes may either increase or decrease the variability of weather; that is, there may be fewer or more heat waves, excessively cold spells, excessively heavy rains, and cold or warm fronts. These changes from the normal weather may be either detrimental or beneficial to human health. The extent and direction of changes in variability under a CO_2-induced climate change are not known at this time, but this information will be very important in determining the effects of climate change on human health.

4. BASIC MECHANISMS IN HUMANS

4.1 Thermoregulation, Acclimation, and Adaptation

With climate change the microenvironment, particularly the temperature in which humans live, will change. The human body has a certain amount of adaptability to the environment. There are passive mechanisms that are triggered more or less continuously by changes in body temperature. For the human body to function properly the core (internal) body temperature must be kept in the normal range (about 37° to 37.5°C). The passive mechanisms react to changes in the body temperature. The surface temperature of the body changes as ambient temperature changes; as we move in and out of areas with different temperatures, humidities, and air movement; as we change our level of physical activity; as we are exposed to solar radiation, and so forth. Metabolic processes continually produce heat within the body, even in the resting condition.

The body normally loses heat by radiation, convection, and evaporation of water from its surfaces. Ambient temperature, air movement and humidity are all important in determining the efficiency of heat loss in this manner. Air movement affects convection and both air movement and humidity affect evaporation.

When external mechanisms fail to keep core temperature within the normal range, the thermoregulatory mechanisms become active. These mechanisms involve interactions between the sweat glands and the nervous, hormonal, and cardiovascular systems and are collectively called the thermoregulatory system. When the body becomes too warm this system is activated by heat sensors. The sweat glands become active in order to facilitate cooling of the skin by increased evaporation. The capillaries dilate to bring more blood to the body surface for cooling. The respiratory rate, blood volume, and heart rate increase.

When the ambient temperature is cold enough to lower the body temperature, the cold sensors activate the regulatory mechanisms. There is vasoconstriction to conserve heat. Shivering generates heat in the muscles, and the basal metabolic rate increases. There is increased cardiac output, stroke volume, and blood pressure (Buskirk 1978).

There may be adverse effects of thermal stress if any part of the thermoregulatory system fails or if the stress is great enough to overwhelm the system and active measures are not taken to bring body temperature back to normal. Generally, however, if the stress is not too great, healthy individuals acclimate to the new temperature fairly rapidly and adequately. After 5 to 10 days of continued heat stress, the body temperature and pulse rate are near normal. By about 14 days, blood volume and venous tone are approximately normal and, by about 3 weeks, a new equili-

brium seems to be established (Lee 1968; Lind 1964). If the heat stress is removed, most of the acclimation appears to be lost within a very few days. Whether there is any residual acclimation is not certain (Lind 1964). The way in which acclimation is accomplished and lost is not fully understood and needs more study (Lee 1968).

The studies on acclimation have usually been done either with artificially heated chambers or on persons who have moved (suddenly been exposed) to hot climates. The research done to date does not seem to be strictly applicable to a situation in which a very gradual temperature change occurs, which may be the case in a CO_2-induced climate change. Nor is there much information on whether, if the thermoregulatory system is frequently or for long periods activated to counteract extreme temperatures (either hot or cold), this might eventually be damaging to any of the components of the system (for example the circulatory system). That is, it is not known whether one climate is more or less stressful than another during a lifetime.

If a CO_2-induced climate change increases the variability of weather and the occurrence of heat waves, then it appears probable that acclimation will not be adequate to prevent morbidity and mortality of some persons, particularly the elderly and those who have diseased circulatory systems (see Section 5.2).

There may be many other subtle differences between people living in different climates. For example, there are seasonal variations in conception, which differ in different climates and which could be considered to be physiological adaptation. In Chili, these variations have been related to temperature, the highest temperature showing the highest peak of conception, except in the northern and southern extremes of that

country, in which there appears to be no seasonality for conception (Hajek et al. 1981). Macfarlane (1970) found a seasonal relation to the rate of conception in many areas of the world. He found that in cool, temperate climates the maximum conception rates occur at a mean monthly temperature of 14 to 16°C and minimum conception rates occur at 23°C. In warm, temperate climates the maximum conception rate is at 13.6°C, and the minimum is at 23°C. In the tropics, the maximum was 26°C and the minimum was 28°C. He states that humidity in the hotter regions (above 25°C) is an important factor in depressing conceptions. He feels that the finding of different conception rates at different temperatures in different climates is prima facie evidence of reproductive adaptation to different environmental temperatures. Holiday seasons and other cultural activities probably have some influence on conception, but he suggests that ambient temperature (and emotional) influences on the female hormones related to fertility may play an important part in the seasonality of conception.

Other researchers also found seasonal patterns in birth rates. In general, conception seems to be lowest in hot weather though there are some unexplained differences between the countries studied (Cowgill 1966; Calot and Blayo 1982), for example, seasonal patterns in the United States and Canada differ from those of Northern Europe. Seiver (1985) found that the seasonality of birth rates in the United States was most pronounced in the Southern (hottest) States and that after air-conditioning became common the maximum and minimum in birth-rate curves in the Southern States became less pronounced. The reasons for the effect of hot weather on conception have not been precisely determined. However, in animals (Johnston and Branton 1953; Glover 1956; Venkatachalam and

Ramanathan 1962) it has been found that sperm counts are depressed by increases in scrotal temperature. In a study of human sperm (Tjoa et al. 1982), sperm counts were found to be lowest in the summer in Houston, Texas, even though there is widespread air-conditioning in that city. Becker (1981) speculated that fecundity of women may change seasonally. Shimura et al. (1981) hypothesized that seasonal diseases may have an influence on conception. Cultural, socioeconomic, and behavioral factors also have some influence on seasonal patterns of conception. For example, Cowgill (1966) believes that urbanization has modified the seasonal pattern. Others (Bernard et al. 1978) speculate that intercourse may be less frequent in uncomfortably hot weather.

A number of studies have investigated the possibility of genetic differences in races who have lived for many generations in very cold or very hot climates. It appears that there are no appreciable physiological changes which specifically adapt races to extreme climates, although cultural practices (for example, types of clothing, housing, food) do enter into adaptation (Newman 1975b). Thus, it would not be expected that genetic selection would assist in adapting humans to a CO_2-induced warming over a number of generations.

4.2 Biochemistry and Physiology

Several studies of human biochemistry and physiology have found seasonal changes in some of these parameters. Blood volume increases in the summer and decreases in the winter (Doupe et al. 1957). Fibrinogen (protein in blood clots) levels have been found to be higher in warm weather than in cold weather (Tromp 1972). Some of the hemostatic

(blood-clotting) factors of the blood seem to vary seasonally (Bull et al. 1979). A seasonal variation has been found in body weight (Billewicz 1967). In a London study it was found that blood pressure peaked in April and May and was lowest in September (Rose 1961). The basal metabolic rate has been found to be higher in winter than in summer (Carlson and Hsieh 1965; Matsui et al. 1978). Some of these changes may be related to thermoregulation but the reasons for others are unclear.

Although there have been attempts (Bull et al. 1979) to relate some of these seasonal variation in biochemical and physiological parameters to the seasonality of mortality from some diseases, the relationships are far from clear, and the effects that a change in climate might have are very uncertain.

4.3 Birth Defects

Seasonal incidence of certain congenital malformations has been found (McKeown and Record 1951; Wehrung and Hay 1970; Cohen 1971), whereas for other malformations a lack of seasonality has been found (Slater et al. 1964). In one study the United States was divided into four climate zones, and differences in these zones were found (Wehrung and Hay 1970). Cohen found similarly shaped curves in several countries and two U.S. states (winter peak, summer trough); in Australia the monthly pattern was essentially reversed. Other investigators, although they found a seasonal relationship to the rate of occurrence of malformation, concluded that variations in climate were associated with only a small proportion of the seasonal variations (Elwood and MacKenzie 1971). Seasonal variations in hormones (possibly related to climatological variables),

toxic metals, and infectious disesases have all been suggested as causes of the seasonal variations in these malformations. However, with regard to infections, McKeown and Record (1951) have found no record of a specific fever in the mothers of malformed infants during pregnancy.

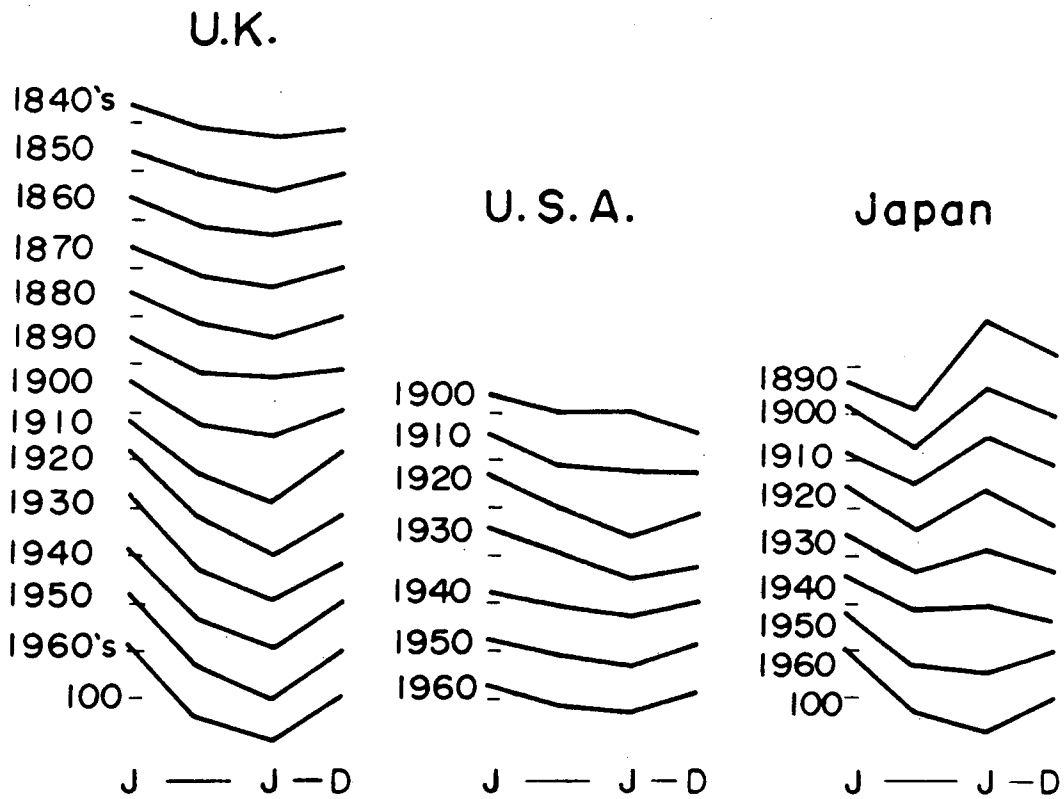
The birth months of persons later diagnosed as being schizophrenic were found to have a highly significant peak, most marked in March and April (Torrey et al. 1977). This seasonality was stronger in New England and the Midwest than in the South; it was also found in Northern Europe. Pulver et al. (1981) also found an association between season of birth and risk for schizophrenia in Monro County, New York. In addition to seasonally varying factors that may damage the central nervous system, they point out that mothers of schizophrenic patients may be more likely to conceive in early summer than are other women. Nutritional, genetic, environmental (e.g., climatological influence on the estrous cycle of women), and infectious disease factors have all been suggested as possible agents.

For birth defects, it appears that climatological factors may have some influence, either direct or indirect, but the precise relationships are very difficult to characterize, and the effects of a CO_2-induced climate change would be very difficult to predict considering the current lack of knowledge about the primary causative agents.

5. MORTALITY AND MORBIDITY RELATED TO CLIMATE/WEATHER

5.1 Seasonality of Mortality and Morbidity

Historically there has been a seasonal pattern for death rates, presumably due to climate and weather influences. These patterns have been changing, during the time in which reasonably good records have been kept. This is illustrated for three countries in Figure 2 from Momiyama-Sakamoto et al. (1977). These data are for all deaths in the particular country and are therefore for a mixture of climates, although the three countries have roughly the same mean annual temperature. The differences among the countries are attributed to differences in their rates of development (medical care, nutrition, central heating, housing, etc.). The prominent summer peaks in the 1890 to 1930 curves for Japan were due principally to communicable diseases. The winter peaks occurring in the later periods in all three countries are attributable principally to diseases of the aged, for example, heart and cerebrovascular diseases. For comparison, Momiyama and Kito (1963) examined the seasonality of death in Egypt, a relatively underdeveloped country. They found that there was a high summer mortality from gastroenteritis, dysentery, and avitaminosis, and a low (compared with the United Kingdom and Japan) mortality from heart disease and stroke (cerebrovascular disease). They did not discuss the age of the population and effects of the climate, but these may have been important factors in the cause of death. A warm climate may be more favorable for bacterial, viral, and parasitic diseases which are more prevalent in youth. Because of deaths at early ages from these diseases, the average age of death may have been lower.



XBL 845-1806

Fig. 2

Historical Changes in Seasonal Variation Patterns of Mortality: United Kingdom (1840 to 1960), United States (1900 to 1960), and Japan (1890 to 1960). Curves run from January to December.

Monthly Death Index = (Monthly Mortality)/(Annual Mortality) x 1200

This method of calculating death index arbitrarily sets the annual average death index at 100 (the -s on the curves), thus parts of the curves above the - are higer than the annual average for that period and vice versa.

Source: Momiyama-Sakamoto et al., 1977.

This would leave fewer persons to die of the diseases that are common to the elderly.

In other studies, Momiyama and Katayama (1966, 1967, 1972) examined the contribution of various age groups to the seasonality of death. Infant mortality contributed greatly to the summer peaks in the earlier years studied, but by the 1970s peaks in infant mortality had become much smaller, and was spread rather evenly throughout all the seasons. By the 1970s the largest contributors to the winter peaks were deaths of people 60 years of age and older, predominantly from heart and cerebrovascular diseases.

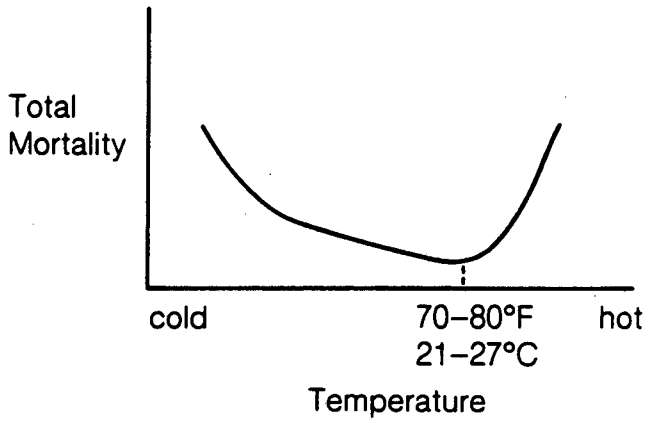
5.1.1 Mortality from all Causes

A number of investigators have found correlations between mortality from various diseases and weather and climate variables. Bull and Morton (1978) related death rates to the mean monthly temperatures in England and Wales. Mortality from vascular, heart, and respiratory diseases was most highly related to temperature. Deaths from asthma, cancer, and leukemia were the diseases least related to temperature. Rosenwaike (1966) found in the United States, for the period 1951 to 1960, that most diseases peaked in winter. The exceptions were cancer, which had no peak, and certain diseases of early infancy, which peaked in the summer.

In general, curves of temperature versus mortality from all causes, in the temperate and subtropical zones of developed countries, peak in the winter at the time of the coldest temperatures and have a minimum at about 21° to 27°C. However, if temperature goes much above

27°C, there may be upsurges in mortality. A generalized curve depicting this situation is shown in Figure 3.

Figure 3



Generalized Relationship of Mortality to Temperature

XBL 848-3469

5.1.2 Mortality from Heart Disease

Although this situation seems to be reversing somewhat, mortality from heart disease increased over several decades. For this reason, researchers have looked for contributing causes, and a number of studies of mortality from these diseases, relative to weather and climate variables, have been carried out. Results of a few of these studies are summarized below as illustrations of the knowledge and questions which are emerging.

Ambient temperature over a fairly wide range (below about 24°) and in several mid-latitude countries has been found to be negatively correlated with mortality from heart disease. That is, a rise in the mean monthly temperature is associated with a fall in mortality (Rose 1966; Dunnigan et al. 1970; Campbell and Beets 1979). Over the temperature range from -1° to 21° C (30° to 70°F), Rose obtained a correlation coefficient of -0.95 between the logarithm of the monthly mortality index and temperature. In London, West and Lowe (1976) found that between 2.3° and 17.9°C there was a 2.5% increase in mortality for each 1°C drop in mean monthly temperature. Dunnigan also found a winter peak for heart disease patients admitted to the hospital but discharged alive. The rate of change of mortality from heart disease per degree of temperature change appears to be different in different climates. Figure 4, from Anderson and LeRiche (1970), compares England and Wales with Australia (a hotter climate) and Ontario and Denmark (mean annual temperatures approximately equivalent to that of England, although Ontario is hotter in the summer and colder in the winter. They suggest that the amount of intercurrent respiratory disease may be responsible for the differences among countries. However, other investigators (Bainton et al. 1977, Rogot 1974) have found that intercurrent respiratory disease had only a minor influence on the relationship between temperature and heart disease mortality. States (1977) compared mortality in Pittsburg, Pennsylvania (temperate climate), and Birmingham, Alabama (subtropical climate) and related mortality to meteorological variables. He found deaths in Pittsburg due to ischemic heart disease to be highly correlated with weather (directly correlated with temperature change), but weather was poorly correlated with deaths from cerebrovascular disease. The reverse

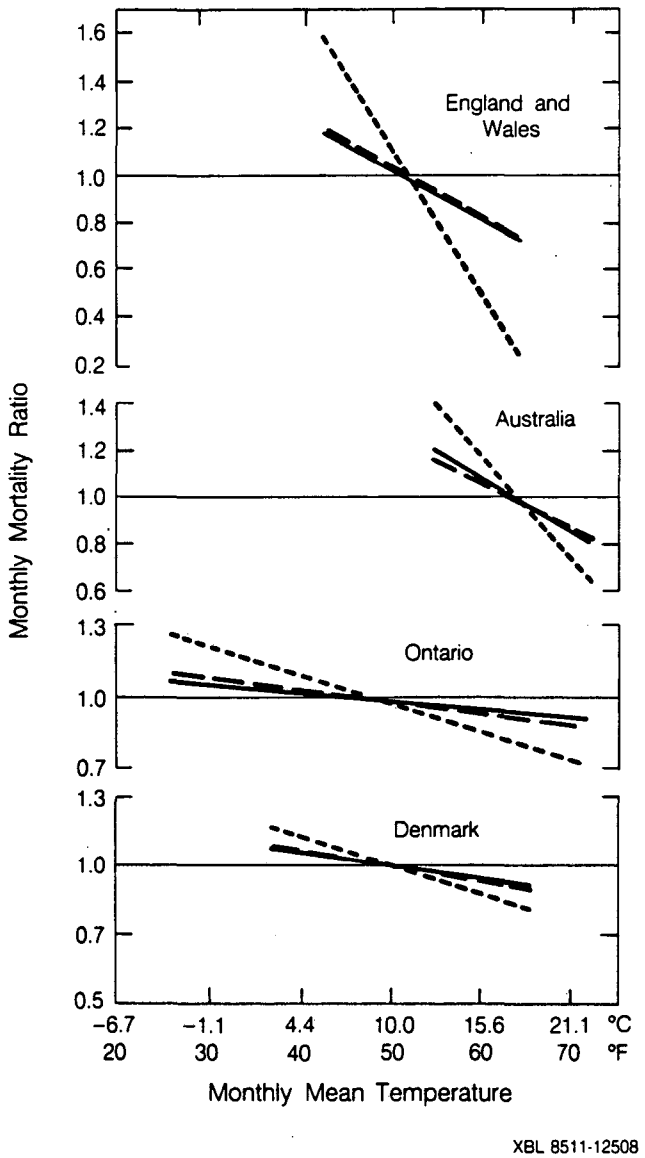


Fig. 4

Relationship of Monthly Mortality Ratio to Monthly Mean Temperature

Monthly mortality ratio is the ratio of the death rate/mo. to the mean death rate/yr. Both rates were expressed in terms of the number of deaths per day in order to allow for the variations in number of days in each month.

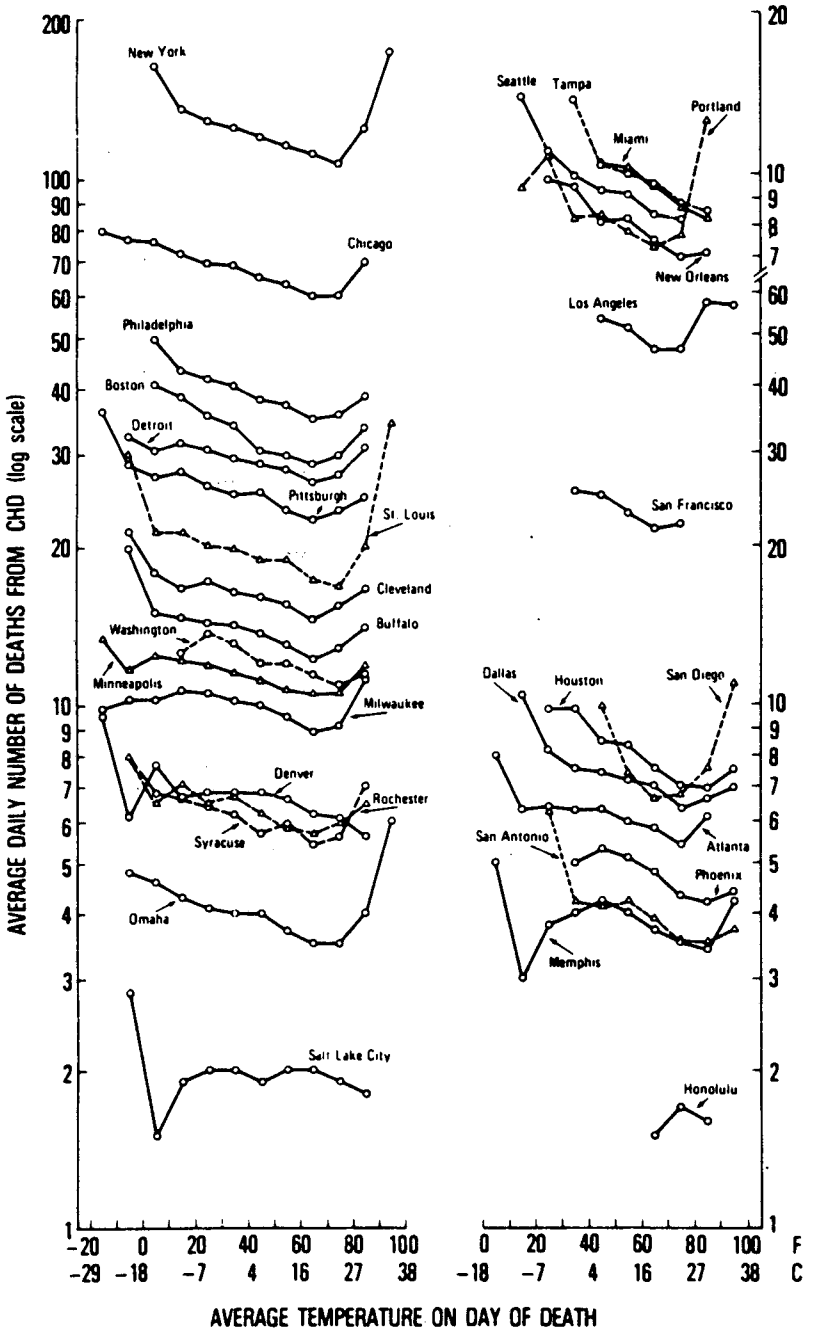
——— arteriosclerotic and degenerative heart disease (ICD 420-422)
— — vascular lesions of the central nervous system (ICD 330-334)
------- diseases of the respiratory system (ICD 470-527)
(ICD's from 7th Revision, International Classification of Disease, World Health Organization, 1955)

Source: Anderson and Le Riche, 1970.

was true in Birmingham. The correlation of mortality with weather variables was significant in both cities, but lower in Birmingham. There was a direct relationship of interdiurnal change in barometric pressure to mortality (for white males, for persons over 70, and for death from ischaemic heart disease). States (1976) surmises that the change in barometric pressure itself is probably not as important as the fact that it is an indication of more profound meteorological changes, such as changes in air mass. Hansen (1970) found that the more abrupt the change in barometric pressure the higher the probability was for the occurrence of peripheral arterial embolism; and that this did not change with seasons.

Even though heart disease mortality gradually declines as the temperature increases, at very hot temperatures there is a rather abrupt increase. The exact temperature at which this abrupt increase occurs appears to differ a few degrees in different areas. Figure 5, from Rogot and Padgett (1976), illustrates this for a number of cities in the United States. The cities on the left are in areas which have snow, those on the right are in areas where snow is unusual. Note that this figure uses numbers of deaths; therefore, death rates cannot be compared for the different cities.

Other meteorological variables also have been examined for their relationships to heart disease mortality. Although relationships of these variables to mortality have been found, it should be noted that the effects are due to synergistic actions with other variables, especially temperature and wind. It is the heat load on the body which is important.



XBL 845-1974

Fig. 5

Relationship of Temperature to Heart Disease Mortality, U.S. Cities, 1962-1966

Average daily deaths from coronary heart disease by average temperature on day of death for 32 selected Standard Metropolitan Statistical Areas: U.S., from 1962-1966.

Source: Roget and Padgett, 1976.

Relative Humidity has been found to be positively related to mortality in both hot and cold weather (Dudley et al. 1969; Rogot 1974; Campbell and Beets 1979). Anderson and Le Riche (1970), in their comparisons of countries with different climates (Figure 5) pointed out that during the winter months, England, Wales, and Australia, with the steepest regressions, had damper winters than Denmark and Ontario, thus implying that humidity may have modified the effects of temperature.

The relationship of rainfall to heart disease mortality has been studied by several investigators. For England and Wales (Rose 1966; Bull 1973) and for Memphis, Tennessee, (Rogot and Blackwelder 1970) no significant correlations were found. However, a positive correlation was found by West and Lowe (1976) and Roberts and Lloyd (1972) for England and Wales and by Rogot (1974) for Chicago.

Periods with heavy snowfall had relatively high rates of heart disease mortality in Minneapolis-St. Paul, Minnesota, (Baker-Blocker 1982), Chicago, Illinois (Rogot 1974), selected U.S. metropolitan areas (Rogot and Padgett 1976), and Toronto, Canada (Anderson and Rochard 1979). It is probable that part of the effects of snowfall are due to increased physical activity in the cold. For the days of snow and immediately following, Anderson and Rochard found a larger increase in death rate for men under 65 than for those over 65. They pointed out that the men under 65 probably both worked and felt compelled to shovel snow, whereas those over 65 (presumably retired) did not feel compelled to be active in the snow.

The windspeed in January was found to have a positive relationship with mortality in 143 U.S. metropolitan areas (Campbell and Beets 1979).

However, in studies carried out in England and Wales, Bull (1973) felt that the relationship was of doubtful biological significance.

Prefrontal weather was found to coincide with increases in total mortality, and postfrontal weather was found to coincide with decreases in total mortality in the northcentral and northeastern states in the United States (Driscoll 1971). In three different areas of the world, that is, Japan, Houston, Texas, and Israel, Cech et al. (1976, 1977, 1979a, 1979b) found increased mortality due to heart disease at times that are typical for the intrusion of polar air during the winter anticyclones tat follow cold fronts.

5.1.3 Mortality from Cerebrovascular Disease

Cerebrovascular disease, including stroke, has also been related to meteorological factors. Figure 6 shows the relationship of stroke mortality to temperature from the study of Rogot and Padgett (1976). The lowest mortality was in the range 15.6° to 26.6°C (depending on the city); then there was a sharp rise as the temperature went higher. These data are in terms of average daily deaths; therefore, although the shape of the curves can be compared, cities with different climates cannot be compared with regard to the rate of death. Bull (1973) and Bull and Morton (1975, 1978) have obtained essentially the same results. For England and Wales, they found from -10° to +20°C they found a nearly linear decrease in the number of deaths as the temperature increased. Above and below this range the death rates rose steeply, particularly in the older age groups. They related mortality to temperature occurring 3 to 4 days before death.

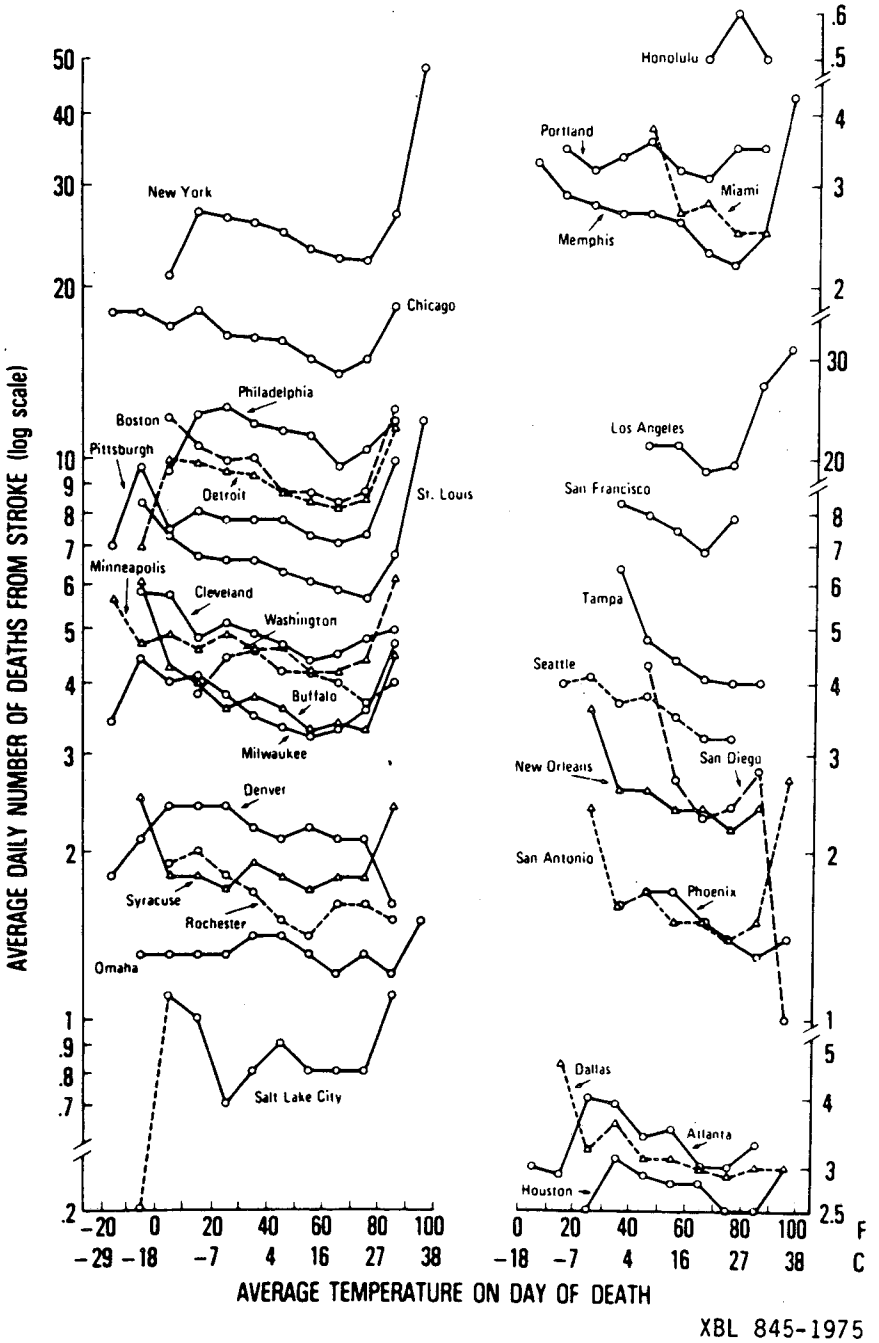


Fig. 6

Relationship of Temperature to Stroke Mortality, U.S. Cities, 1962-1966

Average daily deaths from stroke and average temperature on day of death for 32 selected Standard Metropolitan Statistical Areas: U.S., from 1962 to 1966.

Source: Roget and Padgett, 1976.

Rogot and Padgett (1976) found that periods of snow had a higher than expected stroke mortality. Bull (1973) found that wind speed had no significant relation to stroke mortality.

Cech et al. (1979b) found that increased stroke mortality was related to intrusions of the winter anticyclonic synoptic system, that is, low temperature, low relative humidity, and high wind speed and barometric pressure.

Momiyama and Katayama (1972) related the number of deaths from strokes to temperature in London, Tokyo, and New York City for the period 1960 to 1964. These data are shown in Figure 7. There was a negative relationship between stroke and temperature in all three cities. However, New York, with the lowest temperatures of the three cities, had the least steep slope. The authors attributed this to better central heating in New York than in the other cities.

5.1.4 Mortality and Morbidity from Respiratory Disease

Rosenwaike (1966), using data from the United States, has found peaks in the number of deaths from influenza and pneumonia (combined) and bronchitis in December and January and low death rates in June, July, and August. Bull and Morton (1978), using data from England, Wales, and New York, found high correlations between temperature and deaths from lobar pneumonia and acute and chronic bronchitis. The relationship was nearly linear and inverse from about -5° to about 20°C. When segregated into ages above and below 60 years, the slope of the line was much steeper for those over 60 years. Rogot and Blackwelder (1970), using data from Memphis, Tennessee, found that daily deaths from respiratory diseases

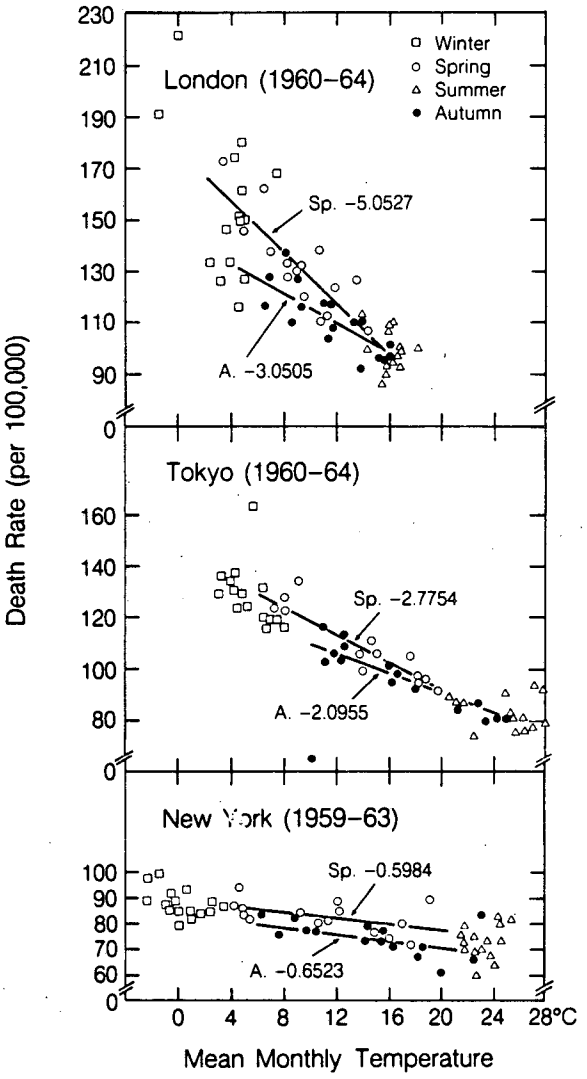


Fig. 7

Comparison of the Relationship of Death Rate from Cerebrovascular Disease to Temperature in London, Tokyo, and New York, 1960-1964

Sp is the regression coefficient for spring (3 months); A is the regression coefficient for autumn (3 months).

Source: Sakamoto-Momiyama, 1977.

were highest at low temperatures (<4°C), gradually decreased to about 23° to 24°C, and then abruptly increased. The data quoted above were not specifically related to epidemics, but epidemics of influenza which at times have killed thousands of persons occur in the winter in both the Northern and Southern Hemispheres and during the rainy season in the tropics. Research attempting to explain the relation of influenza epidemics to specific weather variables has been unproductive (Hope-Simpson 1979).

Goldstein (1980) studied asthma attacks in New Orleans, Lousiana and New York City. Asthma is related to airborne agents such as spores and molds, and there seems to be a relationship to meteorological variables. Goldstein found that clusters of attacks (epidemics) are preceeded, by 1 to 3 days, by the passage of a cold front followed by a high pressure system. It is suggested that the fronts bring in new air containing asthmatogens and that the high pressure system tends to result in stagnant rain-free conditions which retain the airborne particles. Tromp (1980) also implicated cold fronts in the onset of asthma in Europe and added that heat stress combined with high humdity may instigate asthma attacks.

5.1.5 Mortality from Cancer

Mortality from cancer (data from several types pooled) was found to have very little association with temperature in England and Wales (Bull and Morton 1978), Memphis, Tennessee (Rogot and Blackwelder 1970), or the entire United States (Rosenwaike 1966). The small amount of

association that is present may be due to temperature stress on persons who are already terminally ill.

With respect to the initiation of cancer, there are two types of cancer in which there appears to be a meteorological influence. The first is skin melanoma, which is thought to be related to the amount of solar radiation (ultraviolet radiation, which is not expected to change under a CO_2-induced climate change unless there is a large change in cloud cover), but may also have some relationship to female hormones since, especially under the age of 55, the incidence of skin melanoma is higher in women than in men (Scotto and Nam 1980; Cohen 1983; and Cohen et al. 1983). Cultural differences may also affect the rate of incidence of this cancer. The frequency of exposure and the amount of skin exposed to solar radiation differs in different cultures. The male/female ratios of melanoma are different in different climates, so there may be a climate effect on hormone balance; although this is currently only theoretical (Cohen 1983). Breast cancer detection (diagnosis) also has a seasonality which is somewhat different in different climates and may be related to seasonal changes in hormones. The seasonality of hormone levels to a certain extent may be related to ambient temperature, but this is an area in which further investigation is needed (Cohen et al. 1983).

Burkitt's lymphoma is a cancer that has been found to have a definite relationship to climatological factors. It is found in areas where the mean temperature is always above 15°C and the annual rainfall is more than 50 cm. The Epstein-Barr virus was found to be the etiological agent. However, the virus is not limited to the climate in which the lymphoma is found. It was eventually found that the climatological factors favored malaria and that malaria depressed the immune system. The

depressed immune system allowed the virus to initiate lymphoma. The tumor is common only in those areas in which malaria is highly endemic. Thus, in this case, if a CO_2-induced climate change causes new areas of the world to have temperatures above 15°C and rainfall of more than 50 cm, these areas might also become endemic for Burkitt's lymphoma (Burkitt 1983), unless malaria-carrying mosquitos are very well controlled.

5.1.6 Fetal and Infant Mortality

In the United States perinatal (late fetal and less than 7 days of age) mortality and preterm delivery have been shown to have a seasonal variation with major maxima in July and August, a smaller peak in January, and a minimum value in March and April. It has been suggested that this may partially be an indirect effect of meteorological variables, that is, because of infections that have a seasonal distribution (Slatis and DeCloux 1967; Janerich et al. 1971; Keller and Nugent 1983). In England an earlier seasonal trend in perinatal deaths has disappeared, but there is still a relationship between the variation in death rate at 1-11 months of age and temperature (Hare et al. 1981). In Australia, there is a high correlation between an index of heat stress and infant mortality, with the hotter areas having higher mortality (Dasvarma 1980). A seasonal effect has been noted in spontaneous abortions, which peak in the spring (McDonald 1971). The author suspects seasonally occuring infections to be the underlying cause.

Selvin and Janerich (1971) found a seasonality in birth weights, with high-weight infants born in March, April and May and low-weight infants born in June, July and August. They speculate that the low birth

weight seen in the summer may have been due to environmental factors occurring during in early gestation in the fall and early winter months.

5.1.7 Mortality from Other Causes

Deaths in the United States of persons with diabetes, tuberculosis, some renal diseases, cirrhosis of the liver, and senility (plus ill-defined conditions) are higher in winter than in summer (Rosenwaike 1966). In England and Wales deaths from gastric and duodenal ulcers and genitourinary diseases have been related inversely to mean temperatures (Bull and Morton 1978). In these diseases there is generally a prolonged illness preceeding death, and it might be expected that any stress (including low temperatures) would precipitate death in persons already very ill. However, the onset of insulindependent juvenile diabetes has been shown to have a seasonal variation (peaks in the summer), which has mirror image patterns in the Northern versus the Southern Hemispheres (Durruty et al. 1979; Fleegler et al. 1979). Aside from juvenile diabetes, a CO_2-induced climate warming may prolong the lives of persons with some of these diseases, but the effect would probably be small.

In the United States suicides tend to peak in the March to May period, and homicides have two peaks, one in July to September, the other in December. Motor vehicle accidents are lower in the first half of the year (January to June) than in the last half, with the highest rate being in October through December. Non-motor vehicle accidents are highest in June and July, probably because these months are peak vacation times (Rosenwaike 1966). Although meteorological factors may have some influence, these causes of death are so interrelated with psychological and

cultural factors that it would be difficult to determine the quantitative effects of CO_2-induced climate changes.

Thus, seasonal changes in weather variables have an important role in morbidity and mortality. In less developed countries there is still a large influence on health from communicable and parasitic diseases some of which peak in the summer and many of which are greatly influenced by the weather elements (see Sections 7 and 8). In the more highly developed countries, where infectious and some parasitic diseases to a great extent have been controlled, the effects of cold winters and hot summers are primarily in persons with deficient thermoregulatory systems and, secondarily, in persons who have undue exposure to, or activity in, extreme temperatures and in which the thermoregulatory system, although it may be functioning properly, is overwhelmed. How much the underlying climate is related to long-term (possibly damaging) stress on the thermoregulatory system or with acclimating people to better withstand thermal stress is uncertain.

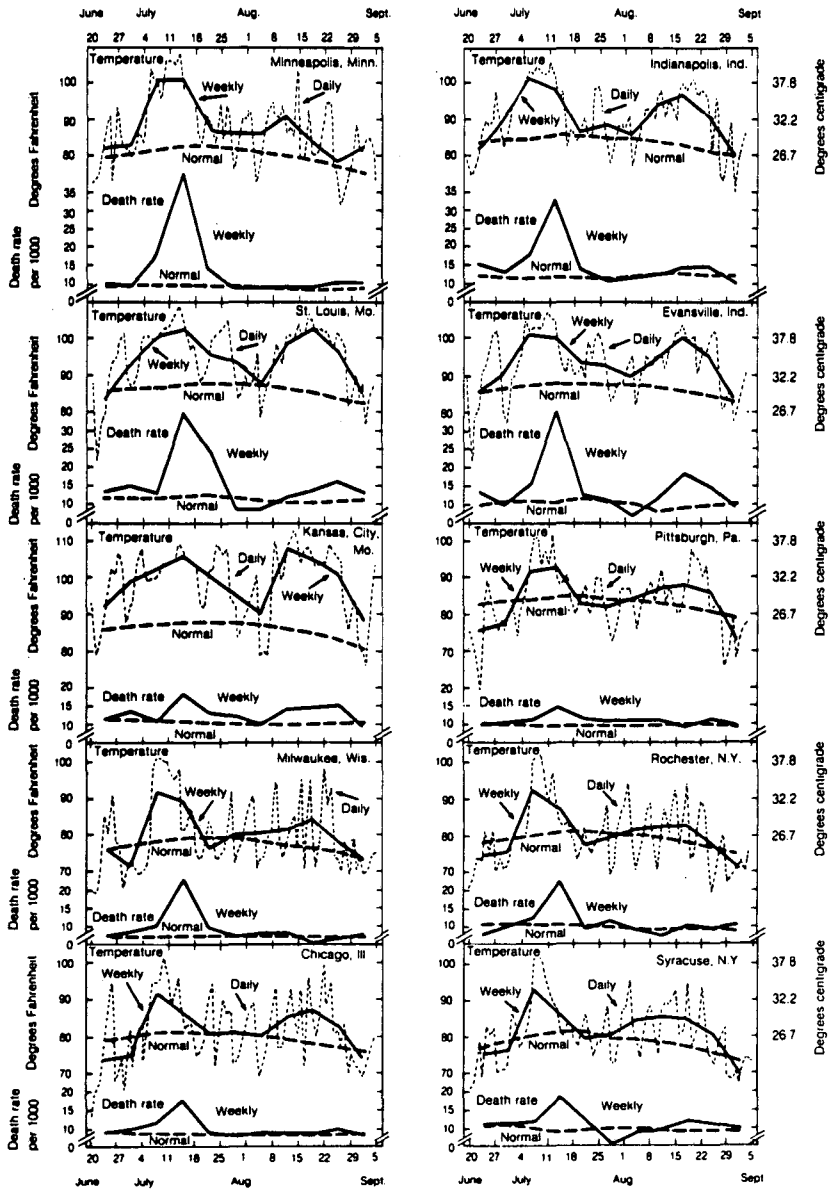
At first glance it would appear that, in currently temperate and cold zones, if CO_2-induced climate change resulted in warmer winters, this might prolong life in some persons. However, hotter summers might, to some extent offset this. Hotter summers, insubtropical and temperate climates, will most probably increase heat related deaths. In addition the other weather elements, that is, humidity, precipitation and wind act synergistically with temperature to modify (either beneficially or detrimentally) thermal stress. Thus, the rate of climate change, the extent of regional and seasonal changes in the climate, and the weather elements and the variability of weather are needed to predict the effects of CO_2-induced climate change on people with organic disease.

5.2 Heat Waves

There is currently no information about whether there may be changes in the variability of weather resulting from a CO_2-induced climate change. If variability decreases, even though the average temperature may be higher, there should be fewer heat waves (acute episodes of excessive heat). If variability increases there may be more heat waves. Because of these uncertainties and because studies of heat waves give some insights into the effects of higher than normal temperatures they may be relevant to the effects of a CO_2-induced climate change.

Death rates can increase markedly as a result of heat waves and the peaks are correlated with temperature 1-2 days before death (Bridger and Helfand 1968; Oechsli and Buechley 1970; Ellis 1972; Ellis et al. 1975). Figure 8 from an early study by Gover (1938) shows the death rates and temperatures in 1936, during excessive heat, for various cities in the United States. These are compared to normal death rates and temperatures (non-heat wave years).

Heat waves, acute enough to cause illness and increase death rates, vary in their meteorological characteristics from year to year in the same region and from region to region in the same year. The demographic characteristics also can vary appreciably from city to city and within the same city over a few years' time. Thus, it is difficult to compare the effects of one heat wave with those of another. Table 1 illustrates this with temperature and mortality data from heat waves in New York, Los Angeles, and St. Louis. In comparing excess deaths in the 1966 heat wave note, that the population of New York was about 10 times that of St. Louis and that the heat wave lasted about twice as long in St. Louis. The meteorological factor of most importance is an increase



XBL 845-9395

Fig. 8

Temperature and Death Rate During Extremely Hot Weather for Various U.S. Cities in 1936

Weekly death rate from all causes (annual basis) and daily average weekly maximum temperature for 10 cities in an area of extreme temperature for 11 summer weeks of 1936.

Source: Gover, 1938.

Table 1

Data from Heat Wave Studies

Ellis and Nelson (1978): New York Heat Wave, August 1975

Length of heat wave	(July 31-August 5)	6 days
Average maximum daily temperature	(July 31-August 5)	92.2° F (33°C)
Average minimum daily temperature	(July 31-August 5)	74.3° F (23°C)
Average dew-point temperature	(July 31-August 5)	68.5° F (20°C)
Average daily mortality	(August 1-6)	293
Excess deaths/day (compared to mortality in the same weeks in 1973 and 1974)	(August 1-6)	94
	(August 1-6)	

Schuman (1972): New York Heat Wave, July 1966

Length of heat wave	14
Temperature: 12 days	90°F (32°C)
Total excess mortality (increase over normal)	1181 (36.3%)
Excess deaths per day	84

Schuman (1972): St. Louis heat wave, July 1966

Length of heat wave	28 days
Temperature: 24 days	>90°F (32°C)
Total excess mortality (increase over normal)	618 (55%)
Excess deaths per day	22

(Note that population of St. Louis at the time of this study was approximately one 10th that of New York.)

Oechsli and Buechley (1970): Three Los Angeles heat waves

1939 September	
Length of heat wave:	7 days
Average maximum daily temperature:	103.4°F (40°C)
Total excess mortality (9 days):	546
Excess deaths per day (9 days):	61
1955 September	
Length of heat wave:	7 days
Average maximum daily temperature:	103.5°F (40°C)
Total excess mortality (9 days):	946
Excess deaths per day (9 days):	105
1963 September	
Length of heat wave:	4 days
Average maximum daily temperature:	106.0°F (41°C)
Total excess mortality (8 days):	580
Excess deaths per day (8 days):	72

in temperature of several degrees above normal for the region and which lasts for several days to several weeks (Bridger and Helfand 1968; Oechsli and Buechley 1970; Ellis 1972; Ellis et al. 1975). The length of the heat wave, maximum daytime temperature, minimum night temperature, humidity, and air movement all enter into the effects on humans in that they all influence body temperature and thus burden the thermoregulatory system.

Illness (heat stroke, heat exhaustion, etc.) may occur in healthy persons who are overexposed to, or overactive in, the heat. However, the majority of excess deaths that occur during heat waves are primarily from other illnesses in which heat stress accelerates death. Infants, the elderly, and persons already ill, in particular with circulatory problems, are most at risk in excessive heat (Schuman et al. 1964; Oechsli and Buechley 1970; Bridger et al. 1976; Jones et al. 1982). Tables 2 and 3 illustrate the age influence on death rate. Older people are more at risk since the main stress is on the circulatory system and many older people have heart and vascular disease. Probably mainly because of circulatory problems their thermoregulatory systems are not as efficient as those of younger persons (Crowe and Moore 1973; Ellis and Nelson 1978). Excess mortality in infants less than 24 hours old has been noted (Bridger et al. 1976); this is probably because their thermoregulatory systems are not yet functioning adequately to counteract the heat.

The illnesses of adults affected by heat are predominately ischaemic heart disease (Ellis and Nelson 1978) and cerebrovascular lesions (Schuman et al. 1964). In some regions or in some years the percent rise in heart disease deaths was found to be higher (Ellis et al. 1975), in other regions or years cerebrovascular disease accounted for more deaths

Table 2

The Number of Deaths per 1,000 Population by Age, Sex, Race, in St. Louis City, Missouri, for July 1966 (Annual basis) and for the year 1965

Age Group	Sex			Race		Male		Female	
	Total	Male	Female	White	Other	White	Other	White	Other
July 1966 (on annual basis)									
All Ages	27.1	29.2	25.3	30.5	21.0	33.7	21.4	27.8	20.6
Under 5 years	13.4	11.2	15.7	10.4	17.0	9.9	12.8	11.0	21.1
5 to 24 years	1.2	1.6	0.8	1.2	1.2	1.5	1.8	0.9	0.7
25 to 44 years	7.0	8.6	5.5	6.4	7.9	7.7	10.3	5.1	6.1
45 to 54 years	19.5	25.7	13.7	17.5	23.7	23.4	30.5	12.2	17.1
55 to 64 years	36.2	53.8	24.0	29.2	58.7	49.4	65.7	16.0	52.7
65 to 74 years	89.3	107.8	74.8	83.4	109.7	116.2	84.7	60.1	137.0
75 years and over	231.1	236.7	227.6	246.5	167.9	247.8	192.2	245.7	151.9
Resident 1965									
All Ages	14.2	16.5	12.2	15.6	11.6	17.9	13.8	13.6	9.7
Under 5 years	8.0	8.6	7.4	5.7	11.0	6.2	11.6	5.1	10.3
5 to 24 years	0.9	1.2	0.6	0.7	1.1	1.0	1.5	0.5	0.7
25 to 44 years	3.6	4.6	2.7	2.4	5.6	3.1	7.5	1.8	4.1
45 to 54 years	10.4	13.4	7.6	8.2	15.1	11.4	17.8	5.5	12.5
55 to 64 years	20.3	30.0	13.3	17.6	29.2	28.1	35.6	10.4	23.7
65 to 74 years	45.7	61.0	34.0	42.8	56.7	80.3	63.6	30.4	49.8
75 years and over	115.7	130.0	106.7	123.8	80.1	136.7	101.4	115.8	65.7

(from Bridger, Ellis and Taylor, 1976)

TABLE 3

Mortality during Three September Heat Waves in Los Angeles

	1939	1955	1963
Number of Days of Excessive Heat	7	7	4
Maximum Temperature	41°7C (107°F)	43.3°C (110°F)	42.8°C (109°F)
Day of Maximum Temperature	4	2	2
Day of Peak Mortality	5	3	5
Peak Mortality as percentage of Expected (All Ages)	271%	445%	172%
Peak Mortality as percentage of Expected (50-54 Years)	245%	307%	140%
Peak Mortality as percentage of Expected (85+ Years)	570%	910%	257%

Data from Oechsli and Buechley, 1970

(Ellis and Nelson 1978; Bridger and Helfand 1968). Whether this difference is due to the weather preceding the heat wave, the characteristics of the particular heat wave, or nonclimatic influences is unknown.

There has been speculation that most persons who die as a result of heat waves would have died very soon even without this stress. Some investigators state that there was no drop in overall mortality after heat waves which would have compensated for deaths during heat waves (Henschel et al. 1969; Schuman 1972; Ellis et al. 1975). In Gover's data

(Figure 8) only 3 of the 10 cities show a drop below normal within a few days after the first heat wave, and these were fairly small and probably not significant. However, Schuman et al. (1964) examined data for deaths of persons over 65 years of age from 108 cities in the United States and found an impressive drop in mortality 5 weeks after the heat wave. Thus, there is uncertainty regarding the extent by which life is shortened in those who die as a result of heat waves.

It is difficult to determine the number of people who become ill because of heat stress but do not die, because generally these cases are not listed in public health records. However, it appears that at least as many as die, become ill but survive. (Cook 1955; Jones et al. 1982).

Within a region during a particular heat wave investigators have examined various factors other than climate which seem to influence heat wave effects. Death rates increase much more in inner cities than they do in suburban and rural districts (Henschel et al. 1969; Schuman 1972; Jones et al. 1982). There are probably several factors involved here (see below), but the most predominant of these is thought to be that urbanization has modified the climate (Clark 1972a). There also may be relatively fewer air-conditioners in urban residences than in suburban residences. Daytime urban-rural differences in temperature are small. However the urban core retains heat at night because of lack of air movement, higher near surface air temperatures and a greater heat load. After sunset, the rural surfaces cool at a more rapid rate than do urban surfaces, because soil and grass have a lower heat conductivity and storage capacity than do brick and concrete surfaces. Large urban structures also loose heat slower than do single-family dwellings. More heat stroke victims were found in upper levels of multistory buildings than in

residences well shaded by trees and shrubs (Kilbourne et al. 1982). Thus it appears that the urban environment contributes substantially to excess morbidity and mortality from heat.

The racial composition of people who died in heat waves has been studied with apparently contradictory results. In some cases white people seemed to have higher excess mortality (Bridger et al. 1976, see Table 2); whereas in others mortality in non-whites was higher (Henschel et al. 1969; Schuman 1972), and in yet others, there appeared to be no differences (Schuman et al. 1964; Bridger and Helfand 1968). These apparent contradictions may have resulted, for example, from the methodology used in the studies or socioeconomic differences among the populations studied.

Socioeconomic status has been found to be a factor in the death rate in heat waves. People in the lower socioeconomic status were generally found to have the largest increases in deaths (Buechley et al. 1972; Schuman 1972; Jones et al. 1982). However, these people live predominately in the inner cities, frequently in high density inadequately ventilated housing, and probably a high percentage of this population perform manual labor in the heat.

The male/female ratio of victims of heat waves has also been examined but without consensus in the results (Schuman et al. 1964; Bridger and Helfand 1968; Henschel et al. 1969; Schuman 1972; Bridger et al. 1976). Some of these studies were not age-adjusted which may have influenced the results because there are usually more women in the older age groups.

Some studies have suggested that widespread use of air-conditioning has reduced the effects of heat waves (Oechsli and Buechley 1970;

Henschel et al. 1969; Kilbourne et al. 1982). However, one study (Buechley et al. 1972) found that the correlation between percentage of cases with air-conditioning and the mortality rate ratio in counties of the New York - New Jersey area, while negative was very small. There is also some indication that residence in air-conditioned premises may interfere with acclimation (see Section 4) to heat (Marmor 1975) and thus make people more vulnerable to heat when they are in nonair-conditioned environments. Thus a power outage during excessive heat might result in disproportionate numbers of deaths in people accustomed to air-conditioning (Bridger et al. 1976). Nonetheless, air-conditioning undoubtedly does lessen stress, thus protecting susceptible people when there are heat waves.

There are physiological adjustments to extreme heat (Wyndham et al. 1970; Folk 1974). However, the extent to which these mechanisms increase the heat tolerance of humans and modify morbidity and mortality for normal (for a particular climate) hot weather and for heat waves is uncertain. The data of Gover (1938), Figure 8, which was collected before air-conditioning had an appreciable effect, indicate that in the second heat wave of a summer the death rate was lower than in the first heat wave. Some of this effect may have been due to the most vulnerable people being elimated during the first heat wave. However, it appears that, at least partially, this lower death rate may have been due to acclimation acquired during the earlier part of the season.

Gover found that for particular weeks during the 5 years in her study, both normal temperatures and death rates were higher in the south than in the north-central, north-Atlantic, and western areas of the United States. However, in one year in both the south and the north

central regions the weekly average temperature was 35.6°C (4°C above normal for the south and 7.6°C above normal for the north central region). During this period the death rate in the south increased by only 12% whereas that in the north central region increased by 132%. Variables other than temperature were not examined but Gover thought it was the excess over normal temperature rather than the actual temperature which was associated with a marked increase in weekly mortality and that acclimation had an influence.

Unfortunately literature on heat waves is inadequate for determining the precise contributions of individual weather and climate variables to the extent of morbidity and mortality. First, the weather conditions, that is, the length and intensity of the heat wave, the suddenness of the onset, the degree to which the temperature deviates from the normal for the season and region, and the accompanying humidity differ with each heat wave. Second, different regions and the same region in different years may have different demographic characteristics. Also, the data available to, and the methodology used by, different investigators differ.

Until the CO_2 issue arose, there was no reason to investigate the effects of a changing climate in a particular region and thus, with the exception of temperature, climate and weather variables were not usually considered in the studies. However, several indices have been used as predictors of heat stress. Quayle and Doehring (1981) discuss several of the idices and conclude that one, termed apparent temperature (Steadman 1979a, 1979b), is probably the most comprehensive. Steadman based his research on human physiology and clothing science, assuming a typical adult human in the shade and a base windspeed. He determined the rate at

which heat is transferred from the body to the surrounding air (to keep body core temperature at approximately 37°C) at different temperatures and humidities. Apparent temperature can be read from a chart of ambient temperature versus relative humidity. Changes in windspeed or in amounts of direct radiation (insolation or terretsrial and sky radiation) can be factored in. However, he found that the effects of changes of windspeed in the summer are slight. The effect of barometric pressure was found to be negligible. He related apparent temperature to heat syndromes such as heat stroke and heat exhaustion. He did not discuss the relationship of apparent temperature to illness or death from other diseases that might be brought about by heat stress, but presumably the index should be a means for warning that persons with chronic disease and the elderly might be at high risk and should take precautions to reduce exposure and limit physical activity. Lee (1980) reviewed models intended to relate climatological variables to human health and comfort and concluded that, of those developed so far, all had inadequacies.

Thus, heat waves studies point out that abrupt increases in temperature can accelerate death in many people but that there are many uncertainties regarding the influence of variables other than temperature. Is the higher death rate in inner cities wholly due to higher temperatures in those areas, or do socioeconomic factors have a large influence? What influence does the underlying climate have? That is, if one climate is normally hotter than another, are people in the hotter climate better acclimated and thereby appreciably less vulnerable to sudden increases in temperature than those in a cooler climate? How much do such variables as humidity and air movement influence the extent of morbidity and mortality? How much influence does the rate of heat build-

up within a season have? With the exception of the inner city and the socio-economic questions, all these questions are pertinent to climate and related weather changes. That is, how much will regional and seasonal temperature, humidity, and air movement change in a CO_2-induced climate change? However, perhaps the most important question with regard to this is whether the variability in weather will increase or decrease and thus whether there will be fewer or more abrupt changes in temperature.

6. SPORADIC, EXTREME WEATHER EVENTS

There are other sporadic, extreme weather events in addition to heat waves whose frequency and intensity may change with climate change:

Droughts affect human health principally by the failure of crops and grazing lands, consequently affecting nutrition and economics. The failure to obtain adequate nutrition, either because the essential foods are unavailable or are too expensive, increases susceptibility to disease and early death.

Floods also may influence nutritional status by destroying crops. In addition, in many cases housing is lost, leaving people exposed to the elements or crowded into temporary shelter. These situations increase the likelihood of disease and epidemics. Water supplies may be contaminated by floods, adding to the possibility of an increase in the occurrence of disease.

Tornados and hurricanes may kill and maim people, as well as destroy crops and housing, and may, as with floods, increase the probability of the spread of disease.

Flood, tornado, and hurricane effects depend strictly on the force of the individual events, the physical damage done, and the area affected. There is no way to quantify their effects or predict their frequency under a CO_2-induced climate change at this time. When there is more information about regional and seasonal climate change it may be possible to predict the probability of an increase or a decrease in frequency of these events in a particular region.

7. AIRBORNE MATERIALS RELATED TO HUMAN HEALTH

Many airborne bacteria, viruses, fungi, and allergens that cause human illnesses are found in the atmosphere. Atmospheric conditions, especially temperature, precipitation, humidity, and wind, will affect the multiplication, dispersal, and survival of these organisms. Thus, a CO_2-induced climate change may affect their prevalence.

The bacteria and viruses causing human diseases can become airborne through direct dispersal such as coughing and sneezing. They may also be disseminated on dry skin particles and from bedding, and so on. The majority of human diseases from airborne infectious materials are transmitted indoors, where there is close contact among people. Meteorological variables are related to this only to the extent that they influence the indoor environment by effects on air temperature and humidity. However, infectious materials may also become airborne outdoors by the injection of materials in the air by, for example, water cooling towers, sprinkler irrigation systems, spray from waves, raindrop splashes, and industrial processes (especially those involving animals and animal byproducts). Although few measurements of organisms injected directly into

the atmosphere from rural locations have been done, it has been conjectured that harrowing operations, movement of large herds of animals over dry terrain, and other farm practices may contribute to rural loading of the atmosphere. Dust at high humidity can serve as a site for bacterial survival or as a resting place from which such organisms as bacteria and viruses can be redispersed by the splash of raindrops or by vigorous air movement (Akers et al. 1979; Lidwell 1964).

The transport of biological materials suspended in air is the same as that of other materials of comparable size. Dispersal is dependent on atmospheric turbulence. Both horizontal and vertical eddies are involved. Turbulence intensity depends on the roughness of terrain, the change of wind speed with height (wind shear), and the vertical temperature profile of the atmosphere. Insolation and wind speed principally determine the temperature profile near the surface. The amount of insolation determines the thermal turbulence, and the wind speed modifies the intensity of the turbulence. The dispersive ability of the atmosphere depends on whether it is unstable or stable, that is, whether forces acting on it enhance or reinforce its vertical motion (unstable) or suppress its motion and restore it to its initial position (stable). The unstable atmosphere increases dispersive ability. A stable atmosphere allows buildup of pollutants. The degree of stability is dependent on the rate of change of temperature with height (Chatigny and Dimmick 1979).

7.1 Bacteria

Bacteria survive outdoors in appreciable numbers for variable periods of time, and they are found fairly high in the atmosphere. For example, Fulton (1966) found as many as 200 microorganisms per m^3 at an altitude of 3127 m. However, it appears that no major bacterial epidemics in humans are propagated by spread in the atmosphere (see below regarding minor epidemics). The survival of bacteria in the atmosphere is a function of many variables, including temperature, relative humidity, ultraviolet radiation, air pollutants, and the media in which the bacteria are dispersed. Air temperature does not appear to be the usual limiting factor because most organisms can live at quite extreme temperatures (between -20° and 48°C); however, the effect of temperature is modified by relative humidity. The lethal effects of ultraviolet radiation are well known, although high relative humidity may protect some bacteria from these effects. Sampling and plating have been a problem in determining survival of airborne bacteria, and there is still much work to be done before the meteorological conditions involved can be quantified (Akers et al. 1979). However, a few examples related to airborne bacteria are summarized below.

Hyslop (1978) aerosolized various bacteria to test their viability in air. A mycoplasma was tested at various relative humidities and survival was shortest at 80% relative humidity. Escherichia coli, at 20°C and 63% relative humidity, survived less than 3 hours. At least 10% of two mycoplasma strains of human pulmonary origin survived for 5 hours at 28°C and 50% relative humidity. A significant amount of Salmonella typhimurium remained viable for 24 hours at 21 to 28°C and 50% relative humidity (in shaded daylight or darkness).

Katzenelson and Teltch (1976) examined the bacterial quality of air in the vicinity of wastewater spray irrigation in Israel. Viable coliform bacteria were found at a distance of 350 m, and Salmonella was found at a distance of 60 m downwind from the source.

Blanchard and Syzdek (1970) found that air bubbles breaking at an airwater interface contained bacteria concentrated from 10 to 1000 times over that of the suspension from which they originated.

Wellock (1960) described a minor epidemic of Q fever (an influenza-like disease which is caused by airborne rickettsia Coxiella burnetii) in an urban area of the San Francisco Bay area. This disease is usually transmitted to humans from animals and animal products, and in rural areas the majority of cases are found in people that are in close in contact with animals. Dust from lambing pens is often the source. The source of this particular outbreak was apparently a slaughterhouse and the fumes from a fat-rendering process. Cases were found in a fan-shaped, 10 block area downwind from the plant, and there was evidence that airborne dissemination occurred up to 16 km from the source.

Swedish scientists investigated a "red" snow which fell in February 1969 (Bovallius et al. 1978). It had from 70 to 120 bacteria/ml (Bacillus sp.), depending on where it was sampled. Snow that fell before and after the red snow had a count of only from 1 to 4 bacteria/ml. At the time the snow fell, there was a stable high-pressure system over Russia and a low-pressure system west of Scandanavia, with a strong southerly air flow between them. The air transport route was traced, and the airborne material was found to originate in an area north of the Black Sea, where there were sandstorms at that time. Other investigators found pollen and minerals in the snow which were also compatible with

those originating in the Black Sea. The authors did not state whether the bacteria were pathogenic.

Thus, bacteria are present in the atmosphere and contribute to the spread of diseases over long distances but not as effective in causing diseases as are bacteria found in high concentration in indoor environments. This is probably due to factors in the outdoor atmosphere (e.g. ultraviolet light, air pollutants), which kill the bacteria and the great dilution caused by air turbulence.

7.2 Viruses

Viruses in nature generally only multiply in living organisms and are usually transmitted to humans by close contact. As with bacteria, laboratory studies have been performed to find the type of environment in which they can survive. For example, Hyslop (1978) reported that polio and other viruses tested showed progressively decreasing resistance to desiccation after aerosolization; poliovirus longevity in air was sufficient to permit dissemination across distances of several miles; relative humidity, altitude, and solar radiation are among some of the principal determinants of virus survival; other meteorological factors may modify the influence of these.

Falk and Hunt (1980) state that airborne viruses are associated with two types of particles, that is, droplet nuclei (propagated from sneezes, coughs, etc.) and dust particles (shed from skin and clothing), and that the dust particles are associated with localized epidemics. Harper (1963) aerosolized poliovirus and vaccinia virus at 20, 50, and 80% relative humidity. The poliovirus survived best at 80% relative

humidity, but the vaccinia virus survived best at 20% relative humidity. Rechsteiner (1970) found, for respiratory syncytial virus, high infectivity at high relative humidities and low infectivity at low relative humidities.

Several viral diseases of animals have been shown to cause epidemics at some distance from the source, and the viruses were almost certainly airborne. By implication, this could happen in human diseases, but atmospheric (outdoor) spread of human pathogenic viruses has not been shown to be a factor in major epidemics. Indoor airborne spread of viral disease has been implicated in epidemics, and indoor humidity and temperature are related to the viability of viruses. Although indoor environments are influenced by climate and weather, each microenvironment will be different, depending on ventilation, heating, and so forth.

7.3 Fungi

Fungal lung infections are relatively common occurrences in certain areas of the United States. It has been estimated (Furcolow 1965) that 500,000 people annually acquire histoplasmosis (caused by the fungus Histoplasma capsulatum), and that about one-third of them develop a clinical illness that ranges from a very mild respiratory disorder to a serious and frequently fatal condition in which the fungus spreads to other areas of the body. Possibly 90% recover without chemotherapy or hospitalization, and many cases are misdiagnosed as viral diseases. It is not required that this disease be reported to public health officials; therefore reliable statistics on its incidence are not available. However, this, along with other fungal diseases, probably accounts for much

loss of time at work and school (Hasenclever 1979). The different fungi responsible for these infections apparently require different climatic conditions for survival and dispersion.

Furcolow and Horr (1956), investigating the prevalence of Histoplasma capsulatum, found prevalence of this fungus in an area of the United States (middle central and middle eastern states) where annual precipitation is 76 to 127 cm and the average summer temperature is between 21° and 27° C. They think that the direction of the prevailing winds tends to limit the fungus to this area. However, Hasenclever (1979) believes that it has a worldwide distribution in temperate, subtropical, and tropical regions but that there are foci where the microclimate is ideal for rapid multiplication of the fungus and that when these are disturbed (digging, plowing, etc), epidemics occur in areas downwind.

Coccidioides immitis, another soil fungi, is common in certain areas of the southwest and parts of California. Maddy (1957) investigated the areas where the fungus was most prevalent and related the climatic factors to this prevalence. The fungus apparently thrives in a climate where summer temperatures are hot enough to sterilize the surface soil, thereby disposing of competing plants (July ambient air temperatures from 26° to about 32°C), and where there is very little freezing at ground level (January ambient air temperatures range from about 4 to 12°C). Rainfall is also an important factor, with 13 to 50 cm being ideal. Temperature and rainfall outside these ranges are less favorable for the fungus. Hugenholtz (1957) also found that winds, stirring up dust storms, mainly in the spring and fall (especally after a period of precipitation), increase the incidence of infection.

Climate changes could either extend or make more limited the areas in which these fungi are prevalent and responsible for widespread respiratory disease. Separate studies would be needed for each fungus to determine its survival under new climatic conditions.

7.4 Pollen

Pollen from plants are found worldwide and are one of the most common causes of allergies in humans. They are very minute in size and thus are easily airborne and dispersed over long distances. Meteorological conditions may limit or expand their dispersal and, in some cases, their production. Hayfever afflicts approximately 6% of the U.S. population, with the maximum prevalence being in the 25- to 44-year age group. The economic impact of the work loss of so large a fraction of the work force is substantial, the discomfort is great, and many of the cases may develop into asthma. Ragweed pollen is one of the commonest causes of hayfever. Most of the eastern two-thirds of the United States is exposed to the pollen of this plant (Dingle 1964). Because ragweed grows best in cultivated land, it is largely a product of farming practices. Weather is a determining factor in the aeroallergen problem. Rain, wind, and humidity affect the dispersal of the pollen in the air and thus modify the extent of human exposure to pollen and the severity of the hayfever. The pollen season for ragweed is from August to September. The crop is dependent on rainfall in June, July, and August. Drought will delay the onset of the pollen season. As in agriculture precipitation, temperature, and soil moisture are important. In Michigan, for example, high

May rainfall and low July rainfall will produce a maximum yield of ragweed pollen.

8. SEASONAL DISEASES CAUSED BY MICROORGANISMS

8.1 Airborne Diseases

Many infectious diseases are transmitted from human to human by the airborne route. Most respiratory diseases are more prevalent in cold or changeable weather than in warm weather. The reasons for this climatic influence are debatable. Ford (1981) offers the following as possible reasons: (1) a positive effect of cold weather on survival of the causative pathogens; (2) the depression of the human immune system at lower temperatures; (3) increased opportunities for transmission as people are confined indoors (with higher concentrations of infectious agents) for longer periods; and (4) lack of vitamin C due to the reduced availability of fresh produce. It has also been suggested that drying out of the mucous membranes, from low humidity in heated environments, may be partially responsible for the seasonality of these diseases.

Influenza epidemics occur in the winter in both the Northern and Southern hemispheres and, although the climate relationships to the rather abrupt appearance of new and virulent strains has been sought, it has not been defined (Hope-Simpson 1979). However, Tromp (1980) has stated that influenza virus and gram positive bacteria (including pneumonia-causing bacteria) die more rapidly with high humidity and vigorous air movement. Thus, the winter months with very low humidities and

little air movement in centrally heated buildings are favorable for the transmission of infectious respiratory diseases.

The occurrence of epidemic cerebral meningitis, a winter disease, has been correlated with the influx of warm, humid air masses (Tromp 1963).

Streptococcal infections and diphtheria are more prevalent in the autumn and early winter, but meteorological influences have not been determined.

Poliomyelitis is prevalent in the summer (Tromp 1963, 1980). There is evidence from studies carried out in the United States that the incidence of this disease is related to relative humidity, that is, it occurs when the relative humidity rises above about 28% (Armstrong 1952). In the tropics, the maximum incidence is during the rainy season.

In most cases, the precise relationships of meteorological variables to the incidence of these diseases is unknown, although trends may be fairly well established. With CO_2-induced global warming, it would generally be assumed that summer diseases would become more prevalent and winter diseases less so. However, other meteorological variables, for example, humidity and rainfall, also influence the prevalence of these diseases. CO_2-induced changes in these meteorological parameters, on a regional and seasonal basis, are currently uncertain. Thus, the changes in prevalence of these diseases that might be caused by a CO_2-induced climate change are also uncertain.

8.2 Human Carrier Diseases

Communicable diseases, which are not usually transmitted by air, are influenced by climate and weather conditions. These diseases are usually transmitted by contact (e.g., hands touching food) and by water contaminated with urine or fecal matter.

Cholera is a disease that is prevalent in the summer. It is transmitted by the fecal contamination of water, but there is recent evidence that the organism can be found in water which is not fecally contaminated. It becomes dormant when water is cold and multiplies rapidly when the water warms. May (1958) defined the summer isotherms for cholera as approximately 15° to 27°C (average temperature for the hottest months). Rainfall and humidity have also been implicated as influencing the incidence of cholera.

The group of Salmonella organisms (causative agents of typhoid, paratyphiod, and other intestinal diseases) are usually transmitted by contaminated water and food. These bacteria survive longer when the weather is cold than when it is hot (May 1958), and at thawing time there may be invasions of lakes and streams by bacteria-contaminated materials.

Bacillary dysentery is prevalent in temperate countries during warm, moist months and in subtropical and tropical countries during the rainy season (Sangster 1977).

Each of these diseases needs to be examined individually, and regional and seasonal climate changes must be known in order to define the effects of a CO_2-induced climate change.

8.3 Vector-Borne Viral and Bacterial Diseases

In vector-borne viral and bacterial diseases it is usually the climate and weather influences on the vector or the vector intermediate host, rather than the microorganism, that determine the prevalence of disease.

Although there have been no large epidemics of plague in recent years, it is endemic in many parts of the world including the Southwestern and Western states in the United States. The disease usually is transmitted from rodents to humans by flea bites. Epidemics occur when the temperature is in the range of about 19 to 26°C and relative humidity is high; for example in Brazil, when the relative humidity is between 66 and 83% (Pollitzer 1954). When the temperature increases above about 26°C, the prevalence of plague decreases, apparently because fleas are susceptible to desiccation. In a study conducted in Vietnam (Olson 1969), it was shown that plague incidence was inversely related to the amount of rainfall. As the amount of rainfall increases, the flea population decreases. The reason is unknown, but it was speculated that the flea drowns or gets caught in the mud in ratholes.

There are several different strains of viruses that cause encephalitis and that are transmitted to humans by the culicine mosquito. Birds serve as a reservoir for the virus. The strain of virus and the species of the carrier mosquito determine the prevalence of the disease. There are periodic outbreaks of encephalitis in the United States and many other countries. Rainfall (Burnet 1952) and temperature (Hess et al. 1963) are factors in epidemics. Rainfall and flooding provide breeding ponds for mosquitos. Incubation periods for the virus in the mosquitoes are much longer at 20° than at 32°C. Thus, as the temperature increases,

when the viruses and mosquitoes are prevalent, the probability of epidemics becomes greater. At least one mosquito vector stops biting when the temperature drops below 15°C.

The prevalence of these two diseases would be affected differently by climate change, depending on the combination of meteorological variables. Plague would probably be less prevalent if the temperature became hotter than about 26°C and the humidity was low; although large amounts of rainfall might also decrease the prevalence. On the other hand, encephalitis would be expected to increase with an increase in temperature and rainfall.

8.4 Parasitic Diseases

Parasitic diseases are currently more prevalent in tropical and subtropical climates than in temperate climates, although some are a problem in temperate areas. Technology, the improvement of sanitary conditions, and the education of the populace assist in the control of vectors and parasites. However, there is a large climate influence which contributes to the prevalence of these diseases in the warmer and sometimes more humid areas of the world. Chronic infections with parasites frequently contribute to malnutrition, a decreased ability to work, and an increased susceptibility to other diseases.

8.4.1 Vector-Borne Parasites

Malaria is still one of the world's largest health problems, despite the fact that it has been controlled in many areas of the world

by changes in agriculture and by mosquito abatement programs. There are still many areas in which malaria is endemic and where there are occasional epidemics. In hyperendemic regions (where 75% of the children have enlarged spleens) there is high childhood morbidity and mortality, high immunity in adults, high abortion rates, and low birth rates (Learmonth 1977). In epidemics there is high mortality and morbidity at all ages (although children and the aged are most at risk), a temporary increase in abortions, and a reduction in conceptions and birth rates. The plasmodia (parasite) develops in Anopheline mosquitos and is transmitted to humans by the mosquito bite. There are some 50 species of Anopheles which are important vectors. One of the problems in controlling the mosquitoes is that different species have different susceptibilities to the chemicals used for control and different ecological niches. For example, some like brackish water, some like clear water, some like sunlit water, some like still water, and others like fast-running water. Thus, an increase or decrease in rainfall may modify the habitats to the advantage of some species and to the disadvantage of others. With respect to temperature and humidity, a crude generalization (Learmonth 1977) is that the mosquitoes breed and are active at temperatures above 16°C and a relative humidity of about 60% is ideal. Temperatures in excess of about 35°C and average relative humidity of less than 25% will cause either death or dormancy. Also, the rate of development of the parasite in the mosquito depends on temperature; for example, for P. Falciparum, it takes 9 days at 30°C, 11 days at 24°C and 20 days at 20°C (Garnham, 1964).

Other vectors of parasites and the parasites they transmit require somewhat different climatic conditions.

8.4.2 Parasites Requiring Intermediate Hosts

Schistosomiasis was estimated to have a world prevalence of about 200 million cases in 1975 (Markell and Voge 1981). Part of the life cycle of the parasite occurs in humans, part in water, and part in snails living in still or slow-moving water contaminated by human urine or fecal material. There are several species of schistosomes and the snails which carry them. The parasite enters humans by penetrating the skin when the human comes in contact with schistosome-contaminated water. They then may cause pulmonary, hepatic, intestinal, urinary, or central nervous system problems. The environmental factors that influence the prevalence of the disease act on the water phase of the parasite and on the snail host. Temperature influences various stages of the cycle. Below about 9°C snails are not usually infected by the parasite. When the temperature rises above about 35° to 39°C the snails die. The temperature most favorable for the water/snail cycle of the parasite is 26° to 28°C (Kendall 1964; Purnell 1966; Anderson and May 1979). Rainfall, if heavy enough, can wash the snails out of their ecological niches, but generally rainfall is advantageous because it keeps their areas wet enough for survival. The snail population usually peaks during the season of high rainfall (Anderson and May 1979). Unfortunately, artificial water resources such as dams, irrigation canals, water impoundments (built for health and economic reasons) are ideal breeding and living areas for the water and snail phases of the cycle. Despite attempts to control it, schistosomiasis continues to spread world-wide (Heyneman 1982). It is also frequently carried to new areas by migrants, some of whom are escaping from drought-stricken areas. Thus, changes in climate may influence the spread of this parasite in many ways. Whether a climate change is

advantageous or disadvantageous to the parasite and its host will depend on the direction of regional and seasonal changes, in particular temperature and precipitation. Health education of the population in areas contaminated with the parasite could also influence its prevalence.

8.4.3 Parasites Which Need No Intermediate Host or Vector

The hookworm, endemic in parts of the United States, is an example of this type of parasite. In 1975 the hookworm was estimated to infect about 450 million people worldwide. The eggs are passed from human feces to soil where, if conditions are favorable, they hatch, and the larvae develop and transform to the infective stage. Given the opportunity they then penetrate human skin. They may cause anemia and occasionally pneumonitis if the infecting dose is large, as well as diarrhea. They are generally confined to those parts of the Northern Hemisphere south of the 36th parallel, and in the Southern Hemisphere, north of the 30th parallel (Markell and Voge 1981). Eggs and larvae are subject to freezing and desiccation, and are very climate sensitive. The optimal temperature range for the hatching of eggs is from 27° to 32°C, and they need at least 127 cm of rain per year (Gilles 1984).

For the diseases briefly described above, as well as a number of other parasitic diseases, CO_2-induced increases in temperature may provide new geographic areas suitable for the survival and multiplication of vectors or intermediate hosts. In addition, current endemic areas may be modified so that there are more or fewer life cycles of parasites or vectors during a year. Changes in rainfall could also affect the prevalence of parasites by affecting breeding sites of vectors or hosts. If migra-

tion of humans and new water projects are a consequence of CO_2-induced climate changes, the control of these diseases may become greatly complicated.

9. NUTRITION

9.1 Climate and Weather Effects on Agriculture

Much of the world's population, particularly in the developing countries, is either undernourished (low caloric intake) or malnourished (lacking essential nutrients). This may result in retardation of growth and development beginning in the fetus and continuing throughout childhood and adolescence. Many humans whose caloric intake is adequate are malnourished because the predominant foods are from plant sources that lack or are low in essential nutrients, such as proteins, or have the nutrients (e.g., iron) in such chemical form that they are not easily absorbed. Although these problems are being addressed both on a local and worldwide basis, they are far from being solved (Newman 1975a).

Agriculture, both plant and animal, will be affected by climate changes. Whether these changes are beneficial or detrimental will depend on the extent and type of regional and seasonal changes. For plants, either the amount of crop production or the type of plant which will grow efficiently may change. Both domestic and aquatic animals may be affected. If in a particular region the amount of food production declines or production is switched to foods that are nutritionally less well-balanced, then the nutrition of the regional population may suffer.

In addition to changes in food production, society's need for nutritional energy may increase under a CO_2-induced warming. It has been demonstrated by Consolazio et al. (1961) that more calories are needed for a heavy work load in ambient temperatures over 30°C (86° F) than are needed at lower temperatures.

9.2 Interactions of Disease and Nutrition

There are also interactions between infectious and parasitic diseases and nutritional deficiencies. Nutritional status can influence both susceptibility to disease and the outcome of disease. The disease may also alter the nutritional status of the person affected (Scrimshaw et al. 1968; Mata et al. 1972). The effects of a CO_2-induced climate change on nutrition and disease prevalance may either help the situation or make it worse, depending on regional and seasonal effects on the diseases and on food production.

10. WATER

Water is essential to all forms of life, including that of humans. Precipitation is variable both seasonally and regionally. Regional changes in the amounts of precipitation, temperatures, humidity, and wind influence the amount and quality of fresh water available for humans, thereby affecting the quality of life.

10.1 Quantity of Water

The quality and quantity of nutrition is very dependent on the amounts of precipitation in a region because of the dependence of agriculture (both plant and animal) on water resources. In addition to the total amount of precipitation, agriculture is dependent on the regional and seasonal timing of this precipitation. At opposite ends of the precipitation spectrum, droughts and floods can adversely affect the agriculture of a region. In some areas of the world, the products of fisheries are vital sources of protein, and the fisheries may be affected by the amounts of organic matter carried by runoff from precipitation.

Forests and other nonmanaged vegetation are dependent on precipitation in much the same way as agriculture. The forests products comprise many things needed to maintain or improve the quality of human health; for example, shelters are vital for assisting in body thermoregulation and are helpful in protecting humans from harmful elements of the environment.

The availability of water, which is essential for drinking, cooking, sanitation, and so forth is dependent on precipitation for groundwater recharging, on the continuing flow of water in streams, and on the filling of lakes and reservoirs. When these resources are not adequately replenished, human health and life are endangered.

10.2 Quality of Water

The quality of water is dependent largely on the quantity of precipitation. Groundwater that has percolated through soil and rock and has had impurities filtered out is usually the purest water for drinking, cooking, and sanitation. Groundwater also frequently has essential trace

elements added as it moves through soil and rock. When there is insufficient precipitation to replace the amounts withdrawn, and groundwater is depleted, less pure water from streams, lakes, and reservoirs will be used. These sources are also dependent on precipitation for replenishment and on the dilution of ipurities accumulated from biological or industrial sources. In developed nations, surface water usually goes through purification processes before use by humans, but these processes do not always remove all noxious materials. In developing nations, frequently the water used for drinking, bathing, and so forth, is taken directly from the surface sources that may be highly contaminated with harmful materials (biological and chemical) and the amount of precipitation in these localities will largely determine the concentration of these materials. If there is fast-flowing water, the material may be diluted or washed out. Stagnant water, sometimes created by rain collecting in depressions or by flooding, is an ideal environment for many pathogenic organisms. In some cases groundwater wells may be contaminated by biological materials when rains are heavy and runoff flows into them. Cultural practices, sanitation practices, and poverty all contribute to the problems created by the use of impure water.

If water becomes scarce in a particular region or if there is too much or poorly timed precipitation, there may be adverse effects on human health. Conversely, if areas that are now deficient in precipitation receive more precipitation, or if areas that now get too much seasonal water receive precipitation more evenly over the seasons, then the effects of climate change may be beneficial to those areas. The rate of change of precipitation will also be important; that is, if precipitation declines in a region and does so slowly, it may allow time for adaptive

measures such as construction of reservoirs, modifying methods of irrigation, development of more drought- or moisture-tolerant plants. The effects of a CO_2-induced climate change on water resources will depend on regional and seasonal changes, which are currently uncertain.

11. SHELTER

Shelter is one of the ways in which humans protect themselves from extremes in temperature. In tropical climates well-planned residences are built for the maximum ventilation, residences in extremely cold climates for the maximum insulation, and residences in temperate climates such that they can be open in summer and closed and insulated in winter. Unfortunately, there are many residences and work places that are not well planned. In particular, the inner cities in many areas are built such that they absorb and retain heat. These are the so-called heat islands. This situation has been highlighted in several studies of heat waves (see Section 4.2) where it was found that there were many more deaths in the midcity than in the suburban and rural areas (Henschel et al. 1969; Clarke 1972b).

CO_2-induced warming of the Earth, particularly if there should be more heat waves (which will depend on currently unknown changes in the variability of weather) may make this situation more acute. The planning of cities and revisions of building codes should take this situation into consideration to avoid or alleviate heat absorption and retention (World Meteorological Organization 1970a, 1970b). Meteorologists should be intimately involved in the planning. This is a situation that currently needs attention, regardless of a potential CO_2-induced climate change.

12. AIR POLLUTION

A CO_2-induced climate change, in itself, will not add noxious air pollutants to the atmosphere. However, changes in local weather due to a change in climate may affect the concentration of air pollutants either by dispersing them more rapidly or by allowing concentrations to build up. Air pollutants which are harmful to human health tend to be generated and more concentrated in the atmosphere of industrial and metropolitan areas. Generally, the areas that are in most danger from health-threatening pollutants are those in low-lying areas, that is, basins in hilly or mountainous areas or river valleys which are somewhat protected from the wind. However, if the meteorological factors are unfavorable, life-threatening episodes can occur in other types of terrain. In several acute episodes of air pollution where there were excess deaths, there was fog combined with high pressure systems (Prindle 1964) which held the smoke and other air pollutants near the ground.

The effects of CO_2-induced climate changes on the occurrence of acute air pollution episodes in a region will depend on the seasonal changes in meteorological variables in that region. If these changes cause air pollutants to disperse more quickly than occurs in the current climate, then the change should be beneficial. If the changes are conducive to the buildup of air pollutants, then they will be detrimental.

13. RECREATION

There are many possibilities for changes in outdoor recreation and tourist areas if there are changes in climate. If the climate is hotter and precipitation does not increase, such things as low lake and low

river levels will occur. Alternatively, if precipitation does increase in some areas, there may be improved conditions for water-related recreation. Forest areas, including national and local parks, and snow areas for winter sports may also change. The effects of a CO_2-induced climate change in these examples and in other recreation and tourist areas will depend on regional and seasonal changes in climate, which, however, are currently uncertain.

14. SUMMARY

There are a number of aspects of human health that are related to climate and weather variables and that may be affected by a climate change. In many instances research has been (and is being) performed to improve knowledge regarding the relationship of meteorological variables to human health. There is, however, much more to learn about these relationships and how they may be used to improve human health. In addition, there are areas of the world where current knowledge and technological advances are not being used as efficiently as possible for various reasons, for example, the lack of education, the lack of funds, and the influence of cultural practices. In some of these situations, climate change may intensify the problems, in others it may be beneficial. In many human health situations, even if CO_2-induced regional and seasonal climate changes were known, the background information needed to predict effects of these changes is unknown or uncertain. However, at least some of this background information is obtainable by defining and comparing the relationships of meteorological variables to health and mortality in current, but differing, climates.

14.1 Seasonality of Mortality from Organic Diseases

It appears, at least on the surface, that climate warming in the cold regions of the world might delay death from organic disease (e.g., heart, cerebrovascular, respiratory diseases). This is based on the fact that, over a fairly wide range of temperatures, mortality increases as the ambient temperature becomes colder and has peaks when the weather abruptly becomes colder. However, there are also increases in mortality in the summer when the temperature becomes excessively hot or when there are sudden upswings in temperature (heat waves). Thus, the extent to which a warming of the climate would counteract the effects of cold weather is uncertain, not only because of uncertainty about the extent and variability of regional and seasonal CO_2-induced changes, but also because the relationships of mortality to meteorological factors (and combinations of these factors) in different climates are not well defined.

Other meteorological factors such as humidity and wind, which will probably also change in a CO_2-induced climate change, modify the effects of temperature stress.

The ambient temperature at which stress becomes excessive for humans appears to differ within and between climate regions, but the reasons for this have not been adequately defined. The amount of stress may be related to the amount of acclimation to the regional climate or to the meteorological conditions immediately preceeding the extreme conditions. Research is needed to clarify these relationships.

The effect on morbidity and mortality of the length of the hot spell, the variation in temperature during this time, and the extent of

deviation from the normal temperature for the season and climate are not precisely defined.

The high mortality rate in inner cities during hot weather is probably due principally to the fact that these areas absorb more heat during the day and lose heat more slowly at night. However, there are other differences between these areas and suburban and rural areas, such as age of the population, type of work, cultural habits, socioeconomic status, and so forth. These differences may influence outcomes and bias temperature versus mortality data.

There is evidence that in nursing homes and hospitals air-conditioning protects from or eliminates heat stress. It is also a factor in comfort during hot weather. However, the extent to which it protects healthy persons from debilitating heat stress and interferes with acclimation to heat is uncertain. If there is interference with acclimation, air-conditioning may lead to vulnerability to the effects of heat when it is not available, that is, due to mechanical or power failure or when people are outdoors.

Older persons are much more vulnerable to temperature stress than are younger ones. This, in most cases, probably is due to the progressive deterioration of the cardiovascular and respiratory systems. However, there is evidence that, in some older persons, thermoregulatory mechanisms other than cardiovascular are inefficient. It is not clear whether this is true in the majority of older persons. If so, it may contributie substantially to mortality from temperature stress.

It has been speculated that the majority of excess deaths, during periods of heat stress, are those of persons already ill who would have died shortly, regardless of the heat stress. The alternative is that

life is appreciably shortened for some persons. Studies intended to resolve this question have produced inconsistent results. Related to this situation is the fact that a second heat wave of approximately the same intensity and duration usually has a lower mortality rate than that of the first one. It is not known whether this is due to mortality of most of the vulnerable persons in the earlier heat wave (leaving a healthier population) or whether the earlier heat wave acclimated people such that they could better withstand the effects of the second heat wave. Both of these factors probably influence mortality in subsequent heat stress.

In contrast to mortality studies, there have been relatively few investigations of the extent to which meteorological factors contribute to the onset and progression of organic disease. This is an important area of research which needs to be accelerated for better understanding of the reasons for the high incidence rate of these diseases in temperate climates of developed countries.

Until uncertainties, such as those discussed above, are resolved, predicting the effects of a CO_2-induced climate change on organic diseases in a quantitative way will be very difficult.

14.2 Airborne Infectious Diseases

Many infectious diseases (e.g., measles, influenza, poliomyelitis) are carried through the air from one human to another. Illness from most of these diseases tends to peak during colder weather. This is generally thought to be due to closer contact between humans confined indoors and to the lack of dilution of the infectious agents with fresh, uncontam-

inated air. In addition, low humidity in the winter may also contribute to susceptibility to disease by drying out mucous membranes, as may the lowering of body temperature when outdoors. Some pathogenic bacteria and viruses survive better in low humidity and still air. Some infectious diseases peak in other seasons. For example, poliomyelitis occurs in summer, and the incidence has been correlated with high humidity. Rainfall and sudden changes in weather are also implicated in the onset of some infectious diseases.

Since the majority of infectious diseases peak in winter, a CO_2-induced warming of the climate might lower the incidence of these diseases. In the cases of those diseases prevalent at other seasons, the regional and seasonal CO_2-induced climate changes would probably influence the prevalence of those diseases. However, the direction of the influence will depend on the direction of the climate changes in particular regions and whether these are favorable or unfavorable for survival and spread of particular disease-causing organisms.

14.3 Human Carrier Disease

Most of these diseases (e.g., cholera and dysentery) are caused by the contamination (usually fecal) of hands, water, and food. They are found in warm, moist environments and may be dispersed to water supplies by rain runoff. Thus, they may be more prevalent with a CO_2-induced warming of the climate in regions where the humidity is high. However, it will be necessary to examine each disease-causing organism with regard to the combination of meteorological factors which pertain to determine the effect which climate change may have on the disease.

14.4 Vector-Transmitted and Parasitic Diseases

Vectors, whether they cause viral, bacterial, or parasitic disease, are usually insects who have no internal temperature-regulating mechanisms. They are, therefore, generally very sensitive to macro- and microclimate; the rate of multiplication of the disease-causing organisms within the vectors is also very temperature sensitive. In some cases, the temperature, humidity, and precipitation requirements for survival of the vector and its larvae, and for multiplication of the microorganism or parasite are reasonably well known; in other cases, more research is needed. In general, vector-borne diseases are more common in warm tropical and subtropical, relatively humid climates, and thus CO_2-induced warming may expand the areas in which they survive and thrive, but regional and seasonal rainfall would also have an influence on their prevalence.

Parasites that have intermediate hosts, such as schistosomes in which part of their life cycle is in snails, part in water, and part in humans, also need specific climatic conditions for survival and to become infective. Thus, a CO_2-induced climate change would probably affect their prevalence, but the direction of this effect will depend on the regional and seasonal situation.

If increased irrigation and construction of reservoirs for agriculture are mandated by a CO_2-induced climate change, this may expand the snail habitat and mosquito breeding sites, thus complicating control of schistosomes and diseases carried by mosquitoes.

14.5 Airborne Spores, Fungi, and Pollen

These airborne materials cause respiratory diseases and allergies. Their abundance in the air may be affected in many ways by meteorological factors. Temperature, humidity, precipitation, and wind are all involved in propagation and dispersal. Regional and seasonal changes in meteorological factors may affect the abundance in the air of all these materials, but each one possibly in a different way, depending on the local combination of meteorological factors.

14.6 Biochemical and Physiological Parameters

A number of human biochemical and physiological parameters vary seasonally. Some are on adoptive response and are related to thermoregulation, but the significance of changes in the other parameters is uncertain, and their relationships to seasonal mortality is unclear. Thus, the effects of climate change are unknown.

14.7 Conception and Birth Defects

There are relationships between the season of birth and the number of abortions, the number of stillbirths, some birth defects, and schizophrenia arising later in life. These amount to a small but significant portion of births. However, the underlying causes and the relationships to meteorological factors are unknown. Whether a CO_2-induced climate change would influence these outcomes is very uncertain and not likely to be clarified until the underlying causes and the influence of current meteorological conditions are defined.

14.8 Undernutrition and Malnutrition

Nutrition is a great problem worldwide, but it is especially acute in underdeveloped countries. This problem is intimately related to agricultural productivity, poverty, and disease. Climate is a large factor in these relationships. A CO_2-induced climate change may have an appreciable impact on nutrition, but the direction of the impact in a particular region will depend on the seasonal changes in that region.

14.9 Water Resources

The amount and purity of the water available regionally for personal human use have a large impact on human health and welfare. The quantity and quality of this water is greatly dependent on the ambient temperature and wind, which affect evaporation, and on the amount and timing of precipitation, which affect water supplies and soil moisture. Higher temperatures will cause increased evaporation from soil and free water surfaces. Regional and seasonal precipitation may or may not be adequate to offset this loss of surface water. The effects of CO_2-induced climate change and whether these are beneficial or detrimental will depend on the direction of regional and seasonal changes.

14.10 Shelter

Both residences and places of work currently are, in many instances, inadequate to protect humans from excessive heat. A CO_2-induced warming of the world could make this situation more acute. This situation should be a major factor in future planning for cities, residences,

and work places. Meteorologists should be involved in this planning, along with architects and engineers, so that such things as prevailing winds, seasonal temperatures, the probability of excessive heat episodes, and so forth, enter into the planning. This is a situation which needs attention, regardless of a CO_2-induced climate change.

14.11 Air Pollution

The concentration of air pollutants builds up when there is stagnant air over pollutant-producing areas. The frequency of occurrence of acute air pollution episodes in a CO_2-induced climate change will depend on whether the frequency of the particular weather types associated with these episodes increases or decreases in regions where air pollution is high.

14.12 Recreational Areas

Recreational areas (including tourist areas) are usually places for rest and relaxation, the use of which is beneficial to both mental and physical human health. The appeal and usability of these areas is frequently dependent on climate (temperature, amount of precipitation), and a change in climate will probably be detrimental to some areas and beneficial to others. Until regional and seasonal changes are known, it is impossible to judge the direction of these effects.

15. DATA AND RESEARCH NEEDS

Table 4 tabulates the major knowns and uncertainties regarding the climate relationship to human health.

15.1 Climate Data Needs

Many aspects of human health and welfare are affected by meteorological variables. Effects of climate change on agriculture, water resources, fisheries and forests may also impinge on human health. To specify qualitatively and quantitatively the effects of a CO_2-induced climate change on human health, it is necessary to know the regional and seasonal climate changes, changes in the variability of climate and weather, and the rate at which climate change will occur. The state of climate research, at this time, is such that it is not possible to predict this information with any certainty. Until such time as definitive regional and seasonal changes are available, it would be useful to have an estimate of the limits of climate change, that is, the range of probable change in meteorological variables for regional health effect studies.

15.2 Carbon Cycle Data Needs

The CO_2 fertilization of plants, which is related to agriculture and thereby to human nutrition, is being studied separately. The data needs from studies of the carbon cycle will be found in the Vegetation Effects State-of-the-Art Report (U.S. Department of Energy (U.S. DOE) 1985a).

The direct effects of breathing increased levels of atmospheric CO_2 on human health have been addressed elsewhere (U.S. DOE 1982; Bland et al. 1982), and on the basis of current knowledge, it appears that the effects will not be deleterious. However, should new information appear indicating a need to further study the direct effects of CO_2 on human health, information from the carbon cycle research will be needed. The rate at which the atmospheric CO_2 concentration will increase and the maximum expected concentration will be needed if these studies are undertaken.

15.3 Medical and Biological Research

Information on the extent and direction is needed to quantify the effects of increased CO_2 on human health. However, there are medical and biological fields in which work pertinent to the effects of climate change has been done or is in progress. This is due to current concern about the influence of climate/weather variables on human health. This research should be continued and intensified, by the agencies already involved, to obtain information that will not only be valuable currently but that will be helpful when more is known about the directions of CO_2-induced regional and seasonal climate changes. The prospect of a CO_2-induced climate change adds some urgency to solving these problems and finding ways to ameliorate them. Research related to meteorological influences which should have high priority is outlined below.

15.3.1 Organic Diseases

Meteorological variables influence mortality rates and, to a largely unknown extent, influence morbidity from a number of organic diseases, such as heart and cerebrovascular diseases. Research should continue and should ideally include (1) the effects of all meteorological variables over the range found within a given climate, including the effects of interactions of the variables, (2) the effects of changes in the variability of meteorological factors, (3) the effects of rate of change of meteorological variables, and (4) studies of whether prevalence of diseases differ in differing climates, and, if so, the extent to which meteorological variables are responsible. The data used in these studies should be adjusted for such factors as age of population, socioeconomic status, sex, race, type of housing, place of residence (urban, suburban, rural), air-conditioning and central heating availability. Meteorologists should be an integral part of the team of public health officials (epidemiologists, biostatisticians, etc.) involved in planning and executing the studies.

Mortality data are relatively easy to obtain from public records; however, useful morbidity data are much more difficult to obtain. For this reason, cooperation of the medical profession will be required to determine the approximate time of onset of the diseases and the age of the patient at the time of onset. The meteorological factors influencing initiation and progression of the diseases are probably more important than those responsible for mortality from a disease already developed to a stage where acute stress of many kinds (especially temperature extremes) causes death. This kind of information will probably be very difficult to obtain, but an alternative, which might produce surrogate

information, is to relate to climate, and compare between climates, the percentage of the population who die of particular diseases within certain categories such as age and sex. This could determine whether particular climates are more or less conducive to the onset of specific diseases at earlier ages. However, there are factors other than meteorological that contribute to the death rate from these diseases. Some examples are variable proportions of a particular race, sex, age, or socioeconomic class. The contributions of such factors to the variance in death rates must be resolved before the influence of meteorological variables can be precisely determined.

15.3.2 Thermoregulation and Acclimation

Thermoregulation and acclimation are related to the organic diseases discussed above. There has been considerable research in this area, and much is known about the physiology of thermoregulation and acclimation; however, it appears uncertain whether a long residence time in either a hot summer or cold winter climate causes cumulative stress on the physiological systems involved and if so, whether this may lead to the early onset of, for example, diseases of the circulatory system. There is also uncertainty about whether time spent in air-conditioned premises leads to the loss of acclimation. If this is the case, it may lead to vulnerability in situations where air-conditioning is, at least temporarily, unavailable (e.g., outdoors, or when there is a mechanical or power failure). Research to elucidate these uncertainties is needed.

15.3.3 Birth Defects and Infact and Fetal Deaths

The role of meteorological variables in adverse reproductive outcomes is uncertain and needs to be elucidated. However, the underlying causes (which have the major influence) are also generally unknown and need to be understood before the role of meteorological variables can be determined.

15.3.4 Bacteria, Viruses, Parasites and Allergens

The meteorological factors involved in the survival and multiplication of the various microorganisms which cause human diseases, and their hosts and vectors, are in some cases reasonably well defined. In other cases, the geographic areas in which these diseases are endemic are reasonably well known, but the precise meteorological conditions in which they survive and thrive are uncertain. Mapping the current areas of endemicity and defining the meteorological, and other variables which are not currently well known and which limit the habitat (or allow it to expand), would currently assist in controlling diseases. In addition, this information would assist in predicting areas in which new health problems might arise or in which current health problems might be alleviated, should there be a climate change for whatever reason. This would allow monitoring techniques to be put into place and preventive measures to be planned.

15.3.5 Nutrition and Disease

Many areas of the world currently are plagued with a combination of nutritional deficiencies and disease. These contributors to poor health reinforce one another. Climate change may be either beneficial or detrimental depending on regional and seasonal changes. The current work of local, national, and international agencies aimed at determining the meteorological factors involved and alleviating these problems should be continued and, where possible, accelerated.

15.3.6 Shelter and Workplace

City planners, architects, and engineers need to work closely with meteorologists on research to design new structures and their surroundings and to remodel old structures such that meteorological factors are used to the best advantage. This is a current need that may become more critical, especially for cooling human habitats, if global warming results from an increase in atmospheric CO_2.

15.3.7 Recreational and Vacation Areas

Until regional and seasonal CO_2-induced climate changes (or at least an estimate of the direction and ranges of these changes) are known, research regarding the effects on these areas probably would not be productive and is not recommended.

ACKNOWLEDGMENT

We are grateful to Drs. Robert L. Dimmick of the Naval Biological Laboratory, Oakland (retired), Dr. Dennis M. Driscoll of Texas A & M University, Dr. Edward K. Markell, Stanford University and Kaiser Foundation Medical Center (retired), and Dr. Stanley H. Schuman, Medical University of South Carolina for their excellent reviews of some of this material. However, the reviewers are not to be held responsible for the contents of this report. We also thank Ms. Judy Barule and Ms. Tennessee Gock for their patient and competent preparation of the manuscript.

This work was supported by the Director, Office of Energy Research, Carbon Dioxide Research Division of the U.S. Department of Energy, under contract number DE-AC03-76SF00098.

Table 4

Knowns and Uncertainties Regarding Principal Climate Effects on Human Health

Health Concerns	Climate Variables Involved	Knowns	Unknowns and Uncertainties
Organic Diseases and Heat and Cold Effects	Temperature Humidity Precipitation (including Snow) Wind Persistence and Speed Various Weather Types (fronts, etc.)	As temperature becomes progressively colder or excessively hot, mortality increases. All climate variables listed influence mortality to some extent. Older age groups are particularly vulnerable to the effects of extremes in meteorological variables or rapid changes in weather. Abrupt increases (heat waves) or decreases (cold snaps) in temperature markedly increase mortality in persons with underlying organic disease. Increases in morbidity and occasionally mortality in persons in apparently good health can result from extreme heat (heat stroke).	The extent to which meteorological variables influence the onset and progression of organic diseases is largely unknown. For example, does the onset of heart disease occur (on average) at an earlier age in one climate as compared with a different climate? Is a particular disease more prevalent in one type of climate than in another? Which meteorological variables are responsible and how do they interact? When there is temperature stress, the extent of underlying climatic influence on age-adjusted mortality rates from various organic diseases is very uncertain, i.e., does mortality from a particular disease occur at earlier ages in one climate compared with another? When there is temperature stress, the extent of interaction of the various meteorological factors in contributing to mortality from various diseases is uncertain. For example, what influence do variations in humidity at a particular temperature have? The temperature (hot or cold) at which there are abrupt increases in mortality appears to differ in different climates, but the influence of modifying meteorological factors is not completely resolved, nor are many of the non-climatic regional factors that may influence mortality.

Table 4

(Continued)

Health Concerns	Climate Variables Involved	Knowns	Unknowns and Uncertainties
Organic Diseases and Heat and Cold Effects (cont.)		During heat waves the following variables influence the extent of mortality and morbidity: Maximum and minimum daily temperature. The variation in temperature from day to day. The length of the heat wave. Sex, race, socio-economic status, housing, physical activity, air-conditioning.	During heat waves, the relative contributions to morbidity and mortality of the following are uncertain: Maximum and minimum (night) temperature. Variation in temperature. Length of the heat wave. Pattern of preheat wave temperature buildup. Effect of underlying climate, e.g., the normal, non-heat wave temperature for the season. Effect of acclimation due to earlier heat waves. Influence of such things as sex, race, socioeconomic status, housing, physical activity, air-conditioning.

Table 4

(Continued)

Health Concerns	Climate Variables Involved	Knowns	Unknowns and Uncertainties
Thermoregulation and Acclimation	Temperature Humidity Wind Speed	Initiated when core body temperature moves outside normal range (cooler or warmer). Many of the physiological and nervous system mechanisms. of thermoregulation are known. Ambient temperature, wind speed and humidity all tend to modify body heat content and through this, thermoregulation. Acclimation to abrupt change in climate essentially takes place in a few weeks in healthy individuals.	Frequent variation in ambient temperature or long-term residence in extreme climates may put appreciable demands on thermoregulatory systems. It is not known whether these demands are beneficial or whether they may eventually lead to disease of, for example, the circulatory system. Effects on thermoregulatory and acclimation mechanisms of moving in and out of hot and cool areas (e.g., work and air-conditioned areas) is uncertain.
Physiology and Biochemistry	Temperature Humidity	There are seasonal variations in physiological systems and biochemistry.	The reasons for these variations are frequently uncertain, as is their relationship to morbidity and mortality.

Table 4

(Continued)

Health Concerns	Climate Variables Involved	Knowns	Unknowns and Uncertainties
Conception and Birth Defects	Largely unknown	There are climate (seasonal) influences on the extent of adverse reproductive outcomes, e.g., stillbirths, malformations, mental disease.	The influence of season is small but significant; however, the influence of factors unrelated to climate is much larger, and these need to be determined before specific effects of meteorological factors can be determined.
Airborne Pathogens, Viruses, and Bacteria	Temperature Humidity Precipitation Wind Speed	Most airborne pathogens are transmitted from person to person indoors, where humidity is probably the most important factor both for survival of of the organism and for infecting humans. Survival and dispersal outdoors is dependant on humidity, precipition, wind speed and direction, ultraviolet radiations, and other nonclimatic factors.	Ranges of climatic conditions for the survival and spread of most pathogens in the outdoors are not well defined. Distances over which pathogens can be dispersed before being inactivated are uncertain. Reasons for disappearance and reappearance of many pathogenic diseases (e.g., influenza epidemics) are not known.

Table 4

(Continued)

Health Concerns	Climate Variables Involved	Knowns	Unknowns and Uncertainties
Pollen and Fungi	Temperature Humidity Precipitation Wind Seasonal difference in climate variables Timing of peak temperatures and precipitation	Temperature, humidity, and precipitation requirements of some pathogenic fungi are known. Timing of temprature and precipitation for the highest production of some pollen are relatively well known.	Climate variables needed for some pathogenic fungi to grow and be dispersed. Reasons for variations in the extent of production and dispersal for some pollen. Precise relationships of airborne allergens to asthma.
Vectorborne microorganisms	Temperature Humidity Precipitation Wind Speed and Direction	The climatic conditions necessary for some vectors to thrive and for the microorganisms to multiply within the vector are known. Some microorganisms are are known to have alternate hosts, e.g., birds.	Ideal environmental conditions for the survival and multiplication of the microorganism in the vector are uncertain in some cases. The role of the alternate hosts is not always well-defined. The ideal climate conditions for the host, and the multiplication of the microorganisms in the host are frequently uncertain.

Table 4

(Continued)

Health Concerns	Climate Variables Involved	Knowns	Unknowns and Uncertainties
Diseases transmitted by human contact or through fecal or urine contamination of food or water	Temperature Humidity Precipitation	In many cases it is known that ambient temperature and humidity are related to the survival of these disease organisms outside the body. Heavy rains can be responsible for dispersing these organisms into drinking and bathing water. Insects spread some of these diseases via their contaminated legs.	For many of the organisms the exact ambient conditions in which the organisms survive and may be transmitted is not known. These diseases are thought to be more virulent in areas in which there is under- or malnutrition, but inter-relationships are not well specified.
Parasitic diseases	Temperature Humidity Precipitation Wind Speed and Direction	Environmental conditions required by some pathogens, their vectors, or intermediate hosts are relatively well known and the areas in which they are endemic are fairly precisely mapped.	For some parasites, their vectors, and intermediate hosts, the environmental requirements or the areas in which they are endemic are not precisely defined. The areas into which these diseases could could move, on the basis of current climate, given appropriate circumstances (e.g., migration of vectors or carriers) is not completely determined in many cases.

Table 4

(Continued)

Health Concerns	Climate Variables Involved	Knowns	Unknowns and Uncertainties
Nutrition	Temperature Humidity Precipitation Wind Timing of peak temperature and precipitation	Failure of crops, underproduction of agriculture, or lack of production of foods with essential nutrients, will lead to mal- or undernutrition. Poverty, even though nutritious foods are available, will affect nutritional status. Some diseases interfere with nutrition. Under- or malnutrition may influence susceptibility to, or progression of, some diseases.	Regional and seasonal CO_2-induced climate change and the effects of an increased ambient CO_2 concentration on agriculture must be known before effects of increased CO_2 on agriculture and thus on nutrition can be predicted. The interrelationships of disease and nutritional status are in many cases poorly understood.
Water Quality and Quantity	Temperature Humidity Precipitation Wind	Water availability and its quality affect, in some degree, almost all aspects of human health.	Information on regional and seasonal CO_2-induced climate change is required before predictions of the effects on water resources and the consequent effects on human health, can be determined.

Table 4

(Continued)

Health Concerns	Climate Variables Involved	Knowns	Unknowns and Uncertainties
Shelter	Temperature Humidity Precipitation	Properly planned shelter can protect from some of the adverse effects of meteorological variables.	The extent to which types of housing enters into morbidity and mortality from extremes in temperature are not well defined. Research into innovative ways to heat and cool new buildings and cities is progressing, but research with meteorological input is not being as actively pursued, as is needed, for older structures (both commercial and residential).
Air Pollution	Stagnant weather conditions Precipitation Wind	The extent of air movement (turbulence, wind) will influence the rate of dispersal of air pollutants.	The effects of a CO_2-induced climate change on air pollution will depend on regional and seasonal changes. Until this information is available, no predictions of the effect of change can be made.
Recreational	All meteorological variables	Recreational areas are very dependent on climate for their usefulness and appeal, and they could be altered by climate change.	The beneficial or detrimental effects of a CO_2-induced climate change on recreational areas cannot be predicted until regional and seasonal information is available.

REFERENCES

Akers, T.G., Edmonds, R.L., Kramer, C.L., Lighthart, B., McManus, M.L., Schlichting, H.E. Jr., Solomon, A.M., and Spendlove, J.C. 1979. "Sources and Characteristics of Airborne Materials." 11-84. In R.L. Edmonds (ed.), Aerobiology, The Ecosystems Approach. Dowden, Hutchinson and Ross, Straussburg, Pennsylvania.

Anderson, R.M., and May, R.M. 1979. "Prevalence of Schistosome Infections Within Molluscan Populations: Observed Patterns and Theoretical Predications." Parasitology 79: 63-94.

Anderson, T.W., and Le Riche, W.H. 1970. "Cold Weather and Myocardial Infarction." Lancet 1: 291-296.

Anderson, T.W., and Rochard, C. 1979. "Cold Snaps, Snowfall and Sudden Death from Ischaemic Heart Disease." Canadian Medical Association Journal 121: 1580-1583.

Armstrong, C. 1952. "Poliomyelitis and the Weather." Proceedings of the National Academy of Science 38: 613-618.

Bainton, D., Moore, F., and Sweetnam, P. 1977. "Temperature and Deaths from Ischaemic Heart Disease." British Journal of Preventive and Social Medicine 31: 49-53.

Baker-Blocker, A. 1982. "Winter Weather and Cardiovascular Mortality in Minneapolis-St. Paul." American Journal of Public Health 72: 261-265.

Becker, S. 1981. "Seasonality of Fertility in Matlab, Bangladesh." Journal of Biosocial Science 13: 97-105.

Bernard, R.P., Bhatt, R.V., Potts, D.M., and Rao, A.P. 1978. "Seasonality of Birth in India." Journal of Biosocial Science 10: 409-421.

Billewicz, W.Z. 1967. "A Note on Body Weight Measurements and Seasonal Variation." Human Biology 39: 242-250.

Blanchard, D.C., and Syzdek, L. 1970. "Mechanism for the Water-to-Air Transfer and Concentration of Bacteria." Science 170: 626-628.

Bland, M.K., Bailey, H.C., and Lipsett, M.J. 1982. The Direct Biological Effects of Increased Atmospheric Carbon Dioxide Levels. (Contract Number 68-02-3716). (Prepared by Stanford Research Institute, International), U.S. Environmental Protection Agency, Washington, D.C.

Bovallius, A., Roffey, R., and Henningson, E. 1978. "Long Range Air Transmission of Bacteria." Applied and Environmental Microbiology 35: 1231-1232.

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

Bridger, C.A., and Helfand, L.A. 1968. "Mortality from Heat During July 1966 in Illinois." International Journal of Biometeorology 12: 51-70.

Buechley, R.W., Van Bruggen, J., and Truppi, L.E. 1972. "Heat Island = Death Island?" Environmental Research 5: 85-92.

Bull, G.M. 1973. "Meteorological Correlates with Myocardial and Cerebral Infarction and Respiratory Disease." British Journal of Preventive and Social Medicine 27: 108-113.

Bull, G.M., Brozovic, M., Chakrabarti, R., Meade, T.W., Morton, J., North, W.R.S., and Sterling, Y. 1979. "Relationship of Air Temperature to Various Chemical, Haematological and Haemostatic Variables." Journal of Clinical Pathology 32: 16-20.

Bull, G.M., and Morton, J. 1975. "Relationships of Temperature with Death Rates from All Causes and from Certain Respiratory and Arteriosclerotic Diseases in Different Age Groups." Age and Aging 4: 232-246.

Bull, G.M., and Morton, J. 1978. "Environment, Temperature, and Death Rates." Age and Aging 7: 210-224.

Burkitt, D.P. 1983. "The Discovery of Burkitt's Lymphoma." Cancer 51: 1777-1786.

Burnet, F.M. 1952. "Murray Valley Encephalitis." American Journal of Public Health 42: 1519-1521.

Buskirk, E.R. 1978. "Cold Stress: A Selective Review." 249-266. In L.J. Folinsbee (ed.), Environmental Stress-Individual Human Adaptations. Academic Press, New York, New York.

Calot, G., and Blayo, C. 1982. "Recent Course of Fertility in Western Europe." Population Studies 36: 345-372.

Campbell, D.E., and Beets, J.L. 1979. "The Relationship of Climatological Variables to Selected Vital Statistics." International Journal of Biometeorology 23: 107-114.

Carlson, L.D., and Hsieh, A.C.L. 1965. "Cold." 15-51. In O.G. Edholm (ed.), The Physiology of Human Survival. Academic Press, New York, New York.

Cech, I., Smolensky, M., Lane, R., Halevy, B., and Samueloff, S. 1976. "Biometeorological Aspects of Short-Term Fluctuations of Cardiac Mortality in Jerusalem and Tel Aviv Studied by Lagged Cross-Covariance Analysis." Israel Journal of Medical Science 12: 828-831.

Cech, I., Smolensky, M., Lane, B.S., Halevy, B., and Samuelhoff, S. 1977. "Meteorologic Factors and Temporal Variation of Cardiac Mortality in an Urban Setting in a Desert Climatic Zone." Israeli Journal of Medical Science 13: 451-459.

Cech, I., Smolensky, M.H., Lane, R., Nagata, H., Takahashi, Y., and Morimoto, T. 1979a. "Day to Day and Seasonal Fluctuations of Urban Mortality in Houston, Texas." International Journal of Biometeorology 23: 77-87.

Cech, I., Youngs, K., Smolensky, M.H., and Sargent, F. 1979b. "Day to Day and Seasonal Fluctuations of Urban Mortality in Kyoto, Japan." International Journal of Biometeorology 23: 89-105.

Chatigny, M.A., and Dimmick, R.L. 1979. "Transport of Aerosols in the Intramural Environment." 95-109. In R.L. Edmonds (ed.), Aerobiology, The Ecosystems Approach. Dowden, Hutchinson and Ross, Straussburg, Pennsylvania.

Clarke, J.F. 1972a. "Some Climatological Aspects of Heat Waves in the Contiguous United States." Environmental Research 5: 76-84.

Clarke, J.F. 1972b. "Some Effects of the Urban Structure on Heat Mortality." Environmental Research 5: 93-104.

Cohen, P. 1971. "Seasonal Variations of Congenital Dislocation of the Hip." Journal of Interdisciplinary Cycle Research 2: 417-425.

Cohen, P. 1983. "Cancer and Seasonal Patterns." American Journal of Epidemiology 118: 785-786.

Cohen, P., Wax, Y., and Modan, B. 1983. "Seasonality in the Occurrence of Breast Cancer." Cancer Research 43: 892-896.

Consolazio, C.F., Shapiro, R., Mastinson, J.E., and McKinzie, P.S.L. 1961. "Energy Requirements of Men in Extreme Heat." Journal of Nutrition 73: 126-134.

Cook, E.L. 1955. "Epidemiological Approach to Heat Trauma." Military Medicine 116: 317-322.

Cowgill, U.M. 1966. "Season of Birth in Man. Contemporary Situation with Special Reference to Europe and the Southern Hemisphere." Ecology 47: 614-623.

Crowe, J.P., and Moore, R.E. 1973. "Physiological and Behavioral Responses of Aged Men to Passive Heating." Journal of Physiology 236: 43.

Dasvarma, G.L. 1980. "Socio-Demographic Correlates of Infant Mortality in Australia." Social Science and Medicine 14D: 151-164.

Dingle, A.N. 1964. "Allergens." 96-130. In S. Licht (ed.), Medical Climatology. E. Licht, New Haven, Connecticut.

Doupe, D., Ferguson, M.H., and Hildes, J.A. 1957. "Seasonal Fluctuations in Blood Volume." Canadian Journal of Biochemistry and Physiology 35: 203-213.

Driscoll, D.M. 1971. "The Relationship Between Weather and Mortality in Ten Major Metropolitan Areas in the United States, 1962-1965." International Journal of Biometeorology 15: 23-39.

Dudley, E.F., Beldin, R.A., and Johnson, B.C. 1969. "Climate, Water Hardness and Coronary Heart Disease." Journal of Chronic Disease 22: 25-48.

Dunnigan, M.G., Harland, W.A., and Fyfe, T. 1970. "Seasonal Incidence and Mortality of Ischaemic Heart Disease." Lancet 2: 793-797.

Durruty, P., Ruiz, F., and de Los Rios, G. 1979. "Age at Diagnosis and Seasonal Variation of the Onset of Insulin-Dependent Diabetes in Chili (Southern Hemisphere)." Diabetologia 17: 357-360.

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

Ellis, F.P., and Nelson, F. 1978. "Mortality in the Elderly in a Heat Wave in New York City, August 1975." Environmental Research 15: 504-512.

Ellis, F.P., Nelson, F., and Pincus, L. 1975. "Mortality During Heat Waves in New York City, July, 1972 and August and September, 1973." Environmental Research 10: 1-13.

Elwood, J.H., and MacKenzie, G. 1971. "Comparisons of Secular and Seasonal Variations in the Incidence of Anencephalus in Belfast and Four Scottish Cities, 1956-1966." British Journal of Preventive and Social Medicine 25: 17-25.

Falk, L.A., Jr., and Hunt, R.D. 1980. "Overview of Airborne Contagion in Animals." Annals of the New York Academy of Science 353: 174-178.

Fleegler, F.M., Rogers, K.D., Drosh, A., Rosenbloom, A.L., Travis, L.B., and Count, J.M. 1979. "Age, Sex and Season of Onset of Juvenile Diabetes in Different Geographic Areas." Pediatrics 63: 374-379.

Folk, G.E., Jr. 1974. Textbook of Environmental Physiology. Lee and Febiger, Philadelphia, Pennsylvania.

Ford, M.J. 1981. The Changing Climate: Responses of the Natural Fauna and Flora. George Allen and Urwin, London, United Kingdom.

Fulton, J.D. 1966. "Microorganisms of the Upper Atmosphere. III. Relationship Between Altitude and Micropopulation." Applied Microbiology 14: 237-240.

Furcolow, M.L. 1965. "Environmental Aspects of Histoplasmosis." Archives of Environmental Health 10: 4-10.

Furcolow, M.L., and Horr, W.H. 1956. "Air and Water in the Natural History of Histoplasma Capsulation." 282-288. Proceedings of the Conference on Histoplasmosis, 1952, Washington, D.C., U.S. Government Printing Office, Washington, D.C.

Garnham, P.C.C. 1964. "Factors Influencing the Development of Protozoa in Their Anthropodan Hosts." 33-50. In A.E.R. Taylor (ed.) Host-Parasite Relationships in Invertebrate Hosts, Second Symposium, British Society for Parasitology. Blackwell Scientific Publications, Oxford, United Kingdom.

Gilles, H.M. 1984. "Intestinal Nematode Infections." In G.T. Strickland (ed.), Hunter's Tropical Medicine. W.B. Saunders, Philadelphia, Pennsylvania.

Glover, T.D. 1956. "The Effect of Scrotal Insulation and the Influence of the Breeding Season upon Fructose Concentration in the Semen of Ram." Journal of Endocrinology 13: 235-242.

Goldstein, I.F. 1980. "Weather Patterns and Asthma Epidemics in New York City and New Orleans, U.S.A." International Journal of Biometeorology 24: 329-339.

Gover, M. 1938. "Mortality During Periods of Excessive Temperature." U.S. Public Health Reports 53: 1122-1143.

Hajek, E.R., Gutierrez, J.R., and Espinosa, G. 1981. "Seasonality of Conception in Human Populations in Chili." International Journal of Biometeorology 25: 281-291.

Hansen, J.B. 1970. "The Relation Between Barometric Pressure and the Incidence of the Peripheral Arterial Embolism." International Journal of Biometeorology 14: 391-397.

Hare, E.H., Moran, P.A.P., and Macfarlane, A. 1981. "The Changing Seasonality of Infant Deaths in England and Wales 1912-1978 and Its Relation to Seasonal Temperature." Journal of Epidemiology and Community Health 35: 77-82.

Harper, G.J. 1963. "Some Observations on the Influence of Suspending Fluids on the Survival of Airborne Viruses." 335-341. Proceedings of the 1st International Symposium on Aerobiology 1963, October 3-5, 1963, University of California, Berkeley, Naval Biological Laboratory, Oakland, California.

Hasenclever, H.F. 1979. "Impact of Airborne Pathogens in Outdoor Systems: Histoplasmosis." 199-208. In R.L. Edmonds (ed.), Aerobiology, The Ecosystems Approach. Dowden, Hutchinson and Ross, Straussburg, Pennsylvania.

Henschel, A., Burton, L.L., Margolies, L., and Smith, J.E. 1969. "An Analysis of the Heat Deaths in St. Louis During July 1966." American Journal of Public Health 59: 2232-2242.

Hess, A.D., Cherubin, C.E., and Lamotte, L.C. 1963. "Relation of Temperature to Activity of Western and St. Louis Encephalitis Viruses." American Journal of Tropical Medicine 12: 657-667.

Heyneman, D. 1982. "Parasitic Diseases of the Tropics." 6-23. In Medical and Health Annual, Encyclopedia Britannica, Inc., Chicago, Illinois.

Hope-Simpson, R.E. 1979. "The Influence of Season Upon Type A Influenza." 170-184. In S.W. Tromp and J.J. Bounna (eds.), Biometeorological Survey Vol I, 1973-1978, Part A. Heyden, London, United Kingdom.

Hugenholtz, P.G. 1957. "Climate and Coccidioidomycosis." Proceedings of Symposium on Coccidioidomycosis, February 11-13, 1957, Phoenix, Arizona. Publication No. 575: 136-157. Public Health Service, Atlanta, Georgia.

Hyslop, N.St.G. 1978. "Observations on the Survival of Pathogens in Water and Air at Ambient Temperatures and Relative Humidity." 197-205. In M.W. Loutit and J.A.R. Miles (eds.), Microbial Ecology. Springer-Verlag, Berlin, The Federal Republic of Germany.

Janerich, D.T., Porter, I.H., and Logrillo, V. 1971. "Season of Birth and Neonatal Mortality." American Journal of Public Health 61: 1119-1125.

Johnston, J.E., and Branton, C. 1953. "Effects of Seasonal Climatic Changes on Certain Physiological Reactions, Semen Production and Fertility of Dairy Bulls." Journal Dairy Science 36: 934-942.

Jones, T., Lang, A.P., Kilborne, E.M., Griffin, M.R., Patrianca, P.A., Wassilak, S.G.F., Mullin, R.J., Herrick, R.F., Donnell, H.D., Choi, K., and Thacker, S.B. 1982. "Morbidity and Mortality Associated with the July 1980 Heat Wave in St. Louis and Kansas City Missouri." Journal of the American Medical Association 247: 3327-3331.

Katzenelson, E., and Teltch, B. 1976. "Dispersion of Enteric Bacteria by Spray Irrigation." Journal of Water Pollution Control 48: 710-716.

Keller, C.A., and Nugent, R.P. 1983. "Seasonal Patterns in Perinatal Mortality and Preterm Delivery." American Journal of Epidemiology 118: 689-698.

Kendall, S.B. 1964. "Some Factors Influencing the Development and Behavior of Trematodes in Their Molluscan Hosts." British Society of Parasitology 2: 51-73.

Kilbourne, E.M., Choi, K., Jones, T.S., Thacker, S.B., and The Field Investigation Team. 1982. "Risk Factors for Heatstroke - A Case-Control Study." Journal of the American Medical Association 247: 3332-3336.

Learmonth, A.T.A. 1977. "Malaria." 61-108. In G.M. Howe (ed.), A World Geography of Human Diseases. Academic Press, New York, New York.

Lee, A.R. 1968. "Human Adaptations to Arid Environments." 517-557. In G.W. Brown, Jr. (ed.), Desert Biology. Academic Press, New York, New York.

Lee, D.H.K. 1980. "Seventy-five Years of Searching for a Heat Index." Environmental Research 22: 331-356.

Lidwell, O.M. 1964. "Microbiology of the Atmosphere and Airborne Infection." 131-158. In S. Licht (ed.), Medical Climatology, E. Licht, New Haven, Connecticut.

Lind, A.R. 1964. "Physiological Responses to Heat." In S. Licht (ed.), Medical Climatology, E. Licht, New Haven, Connecticut.

MacCracken, M.C., and Luther, F.M. (eds). 1985a. Detecting the Climatic Effects of Increasing Carbon Dioxide (DOE/ER-0235). U.S. Department of Energy, Washington, D.C. Available from NTIS, Springfield, Virginia.

MacCracken, M.C., and Luther, F.M. (eds). 1985b. Projecting the Climatic Effects of Increasing Carbon Dioxide (DOE/ER-0237). U.S. Department of Energy, Washington, D.C. Available from NTIS, Springfield, Virginia.

Macfarlane, W.V. 1970. "Seasonality of Conception in Human Populations." International Journal of Biometeorology 14: Suppl.4, Part I: 167-182.

Maddy, K.T. 1957. "Ecological Factors Possibly Relating to the Geographic Distribution of Coccidioides Immitis." Proceedings of Symposium on Coccidioimycosis, February 11-13, 1957, Phoenix, Arizona. Publication No. 575: 136-157. Public Health Service, Atlanta, Georgia.

Markell, E.K., and Voge, M. 1981. Medical Parasitology. W.B. Saunders, Philadelphia, Pennsylvania.

Marmor, M. 1975. "Heat Wave Mortality in New York City 1949-1970." Archives of Environmental Health 30: 130-136.

Mata, L.J., Ursutia, J.J., Albertazzi, C., Pellecer, O., and Arellano, E. 1972. "Influence of Recurrent Infections on Nutrition and Growth of Children in Guatamala." American Journal of Clinical Nutrition 25: 1267-1275.

Matsui, H., Shimaoka, K., Migamura, M., and Kobayashi, K. 1978. "Seasonal Variation of Aerobic Work Capacity in Ambient and Constant Temperature." 279-291. In L.J. Folinsbee (ed.), Environmental Stress, Individual Human Adaptations Academic Press, New York, New York.

May, J. 1958. The Ecology of Human Disease. MD Publications, New York, New York.

McDonald, A.D. 1971. "Seasonal Distribution of Abortions." British Journal of Preventive and Social Medicine 25: 222-224.

McKeown, T., and Record, R.G. 1951. "Seasonal Incidence of Congenital Malformations of the Central Nervous System." Lancet 1: 192-196.

Momiyama (Sakamoto), M., and Katayama, K. 1966. "A Medico-Climatological Study in the Seasonal Variation of Mortality in the United States of America (I). Features of Seasonal Variation of Mortality." Papers in Meteorology and Geophysics 17: 276-286.

Momiyama (Sakamoto), M., and Katayama, K. 1967. "A Medico-Climatological Study in the Seasonal Variation of Mortality in the United States of America (II). Signs of Deseasonality Seen in Mortality." Papers in Meteorology and Geophysics 18: 209-232.

Momiyama, M., and Katayama, K. 1972. "Deseasonality of Mortality in the World." International Journal of Biometeorology 16: 329-342.

Momiyama-Sakamoto, M., Katayama, K., Hashiya, N., and Sato, T. 1977. "Seasonality in Recent Mortality in Japan, U.K. and U.S.A. A Study in

Human Mortality/Season Association." Papers in Meteorology and Geophysics 28: 105-123.

Momiyama (Sakamoto), M., and Kito, H. 1963. "A Geographic Study of Seasonal Disease Calendar Models by Period and Country." Papers in Meteorology and Geophysics 14: 109-118.

Newman, M.T. 1975a. "Nutritional Adaptation in Man." 210-259. In A. Damon (ed.), Physiological Adaptation. Oxford University Press, London, United Kingdom.

Newman, R.W. 1975. "Human Adaptation to Heat." 80-91. In A. Damon (ed.), Physiological Anthropology. Oxford University Press, London, United Kingdom.

Oechsli, F.W., and Buechley, R.W. 1970. "Excess Mortality Associated with Three Los Angeles September Hot Spells." Environmental Research 3: 277-284.

Olson, W.P. 1969. "Rainfall and Plague in Vietnam." International Journal of Biometeorology 14: 357-360.

Pollitzer, R. 1954. Plague. World Health Organization (WHO) Monograph Series Number 22. WHO, Geneva, Switzerland.

Prindle, R.A. 1964. "Air Pollution and Community Health." 505-518. In S. Licht (ed.), Medical Climatology, E. Licht, New Haven, Connecticut.

Pulver, A.E., Sawyer, J.W., and Childs, B. 1981. "The Association between Season of Birth and Risk for Schizophrenia." American Journal of Epidemiology 114: 735-749.

Purnell, R.E. 1966. "Host-Parasite Relationships in Schistosomiasis." Annals of Tropical Medicine and Parasitology 60: 90-93.

Quayle, R., and Doehring, R. 1981. "Heat Stress, A Comparison of Indices." Weatherwise 34: 120-124.

Rechsteiner, J. 1970. "The Recovery of Airborne Respiratory Syncytial Virus." 269. In H. Silver (ed.), Aerobiology Proceedings of the Third International Symposium, September, 1969, University of Sussex, England. Academic Press, London, United Kingdom.

Roberts, C.J., and Lloyd, S. 1972. "Association Between Mortality from Ischaemic Heart-Disease and Rainfall in South Wales and in the County Boroughs of England and Wales." Lancet 1: 1091-1093.

Rogot, E. 1974. "Associations Between Coronary Mortality and the Weather, Chicago, 1967." U.S. Public Health Reports 89: 330-338.

Rogot, E., and Blackwelder, W.C. 1970. "Associations of Cardiovascular Mortality with Weather in Memphis, Tennessee." U.S. Public Health Reports 85: 25-39.

Rogot, E., and Padgett, S.J. 1976. "Associations of Coronary and Stroke Mortality with Temperature and Snowfall in Selected Areas of the United States, 1962-1966." American Journal of Epidemiology 103: 565-575.

Rose, G. 1961. "Seasonal Variation in Blood Pressure in Man." Nature 189: 235.

Rose, G. 1966. "Cold Weather and Ischaemic Heart Disease." British Journal of Preventive and Social Medicine 20: 97-100.

Rosenwaike, I. 1966. "Seasonal Variation of Deaths in the United States, 1951-1960." Journal of the American Statistical Association 61: 706-719.

Sakamoto-Momiyama, M. 1977. Seasonality In Human Mortality, A Medico-Geographic Study. University of Tokyo Press, Tokyo, Japan.

Sangster, G. 1977. "Diarrhoeal Diseases." 145-174. In G.M. Howe (ed.), A World Geography of Human Diseases. Academic Press, New York, New York.

Schuman, S.H. 1972. "Patterns of Urban Heat-wave Deaths and Implications for Prevention: Data from New York and St. Louis During July, 1966." Environmental Research 5: 59-75.

Schuman, S.H., Anderson, C.P., and Oliver, J.T. 1964. "Epidemiology of Successive Heat Waves in Michigan in 1962 and 1963." Journal of the American Medical Association 189: 733-738.

Scotto, J., and Nam, J. 1980. "Skin Melanoma and Seasonal Patterns." American Journal of Epidemiology 118: 785-786.

Scrimshaw, N.S., Taylor, C.E., and Gordon, J.E. 1968. Interaction of Nutrition and Infection. World Health Organization (WHO), Monograph Series Number 57, WHO, Geneva, Switzerland.

Seiver, D.A. 1985. "Trend and Variation in the Seasonality of U.S. Fertility, 1947-1976." Demography 22: 89-100.

Selvin, S., and Janerich, D.T. 1971. "Four Factors Influencing Birth Weight." British Journal of Preventive and Social Medicine 25: 12-16.

Shimura, M., Richter, J., and Miura, T. 1981. "Geographical and Secular Changes in the Seasonal Distribution of Births." Social Science and Medicine 15D: 103-109.

Slater, B.C.S., Watson, G.I., and McDonald, J.C. 1964. "Seasonal Variation in Congenital Abnormalities, Preliminary Report of a Survey Conducted by the Research Committee of Council of the College of General Practitioners." British Journal of Preventative and Social Medicine 18: 1-7.

Slatis, H.M., and DeCloux, R.J. 1967. "Seasonal Variation in Stillbirth Frequencies." Human Biology 39: 284-294.

States, S.J. 1976. "Weather and Death in Birmingham, Alabama." Environmental Research 12: 340-354.

States, S.J. 1977. "Weather and Deaths in Pittsburgh, Pennsylvania: A Comparison with Birmingham, Alabama." International Journal of Biometeorology 21: 7-15.

Steadman, R.G. 1979a. "The Assessment of Sultriness. Part I: A Temperature-Humidity Index Based on Human Physiology and Clothing Science." Journal of Applied Meteorology 18: 861-873.

Steadman, R.G. 1979b. "The Assessment of Sultriness. Part II. Effects of Wind, Extra Radiation and Barometric Pressure on Apparent Temperature." Journal of Applied Meteorology 18: 874-885.

Strain, B.R., and Cure, J.R. (eds.). 1985. Direct Effects of Increasing Carbon Dioxide on Vegetation (DOE/ER-0238). U.S. Department of Energy, Washington, D.C. Available from NTIS, Springfield, Virginia.

Tjoa, W.S., Smolensky, M.H., Hsi, B.P., Steinberger, E., and Smith, K.D. 1982. "Circannual Rhythm in Human Sperm Count Revealed by Serially Independent Sampling." Fertility and Sterility 38: 454-459.

Torrey, E.F., Torrey, B.B., and Peterson, M.R. 1977. "Seasonality of Schizophrenic Births in the United States." Archives of General Psychiatry 34: 1065-1070.

Trabalka, J.R. (ed.) 1985. Atmospheric Carbon Dioxide and the Global Carbon Cycle (DOE/ER-0239). U.S. Department of Energy, Washington, D.C. Available from NTIS, Springfield, Virginia.

Tromp, S.W. 1963. Medical Biometeorology. Elsevier Publishing, New York, New York.

Tromp, S.W. 1972. "Influence of Weather and Climate on the Fibrinogen Content of Human Blood." International Journal of Biometeorology 16: 93-95.

Tromp, S.W. 1980. Biometeorology: The Impact of Weather and Climate on Humans and Their Environment. Heyden and Son, Ltd., London, United Kingdom.

U.S. Department of Energy. 1982. Brown, E.B., Gull, C.D., and Carlston, C.B. (eds.), Effects of CO_2 on Mammalian Organisms, Report of A Workshop, June 5-6, 1980, Bethesda, Maryland, Department of Energy CONF.-8006249.

U.S. Department of Health and Human Services. 1985. "NIOSH Recommendations for Occupational Safety and Health Standards." Morbidity and Mortality Weekly Report Supplement 34: No.1S.

Venkatachalam, P.S., and Ramanathan, K.S. 1962. "Effects of Moderate Heat on the Testes of Rats and Monkeys." Journal of Reproduction and Fertility 4: 51-56.

Wehrung, D.A., and Hay, S. 1970. "A Study of Seasonal Incidence of Congenital Malformations in the United States." British Journal of Preventive and Social Medicine 24: 24-32.

Wellock, C.E. 1960. "Epidemiology of Q Fever in the Urban East Bay Area." California's Health 18: 73-76.

West, R.R., and Lowe, C.R. 1976. "Mortality from Ischaemic Heart Disease - Inter-town Variation and its Association with Climate in England and Wales." International Journal of Epidemiology 5: 195-201.

White, M.R. (ed.) 1985. Characterization of Information Requirements for Studies of CO_2 Effects: Water Resources, Agriculture, Fisheries, Forests, and Human Health (DOE/ER-0236). U.S. Department of Energy, Washington, D.C. Available from NTIS, Springfield, Virginia.

World Health Organization (WHO). 1955. International Classification of Diseases; 7th Revision. WHO, Geneva, Switzerland.

World Meteorological Organization. 1970a. Urban Climate. Proceedings of the WMO Symposium on Urban Climates and Building Climatology, Brussels, October, 1968 (Vol. I). Technical Note 108. Secretariat of the World Meteorological Organization, Geneva, Switzerland.

World Meteorological Organization. 1970b. Building Climatology. Proceedings of the WMO Symposium on Urban Climates and Building Climatology, Brussels, October, 1968 (Vol. II). Technical Note 109. Secretariat of the World Meteorological Organization, Geneva, Switzerland.

Wyndham, C.H., Strydom, N.B., Benade, A.J.S., and Van Rensburg, A.J. 1970. "Tolerance Times of High Wet Bulb Temperatures in Acclimatized and Unacclimatized Men." Environmental Research 3: 339-352.

BIBLIOGRAPHY FOR ADDITIONAL READING

(If title does not adequately describe relation to subject, a parenthetic phrase will be found at end of reference)

Akers, T.G. 1963. "Survival of Airborne Virus, Phage and Other Minute Microbes." 296-339. In R.L. Dimmick and A. Akers (eds.) An Introduction to Experimental Aerobiology. J. Wiley and Sons, New York, New York.

Anderson, T.W., and Le Riche, W.H. 1970. "Seasonal Variation in Ischaemic-Heart-Disease Mortality." Lancet 2: 1140.

Andrewes, C.H. 1964. "The Complex Epidemiology of Respiratory Virus Infections." Science 146: 1274-1277. (Seasonality of infections).

Andrewes, C.H. 1967. "Climate, Weather and Season in Relation to Respiratory Infection." 56-62. In S.W. Tromp and W.H. Weihe (eds.), Biometeorology Vol. 2, pt. 1.

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

Arthur, D.R. 1962. Ticks and Disease. Row, Peterson and Co., Evanston, Illinois. (Seasonality of ticks).

Assaad, F., and Cockburn, W.C. 1972. "Four-year Study of WHO Virus Reports on Enteroviruses Other Than Poliovirus." Bulletin of the World Health Organization 46: 329-336. (Seasonality of viruses).

Assaad, F. and Cockburn, W.C. 1974. "A Seven-year Study of WHO Virus Laboratory Reports on Respiratory Viruses." Bulletin of the World Health Organization 51: 437-445. (Seasonality of respiratory tract infections).

Assaad, F., and Borecka, I. 1977. "Nine year Study of WHO Virus Laboratory Reports on Fatal Viral Infections." Bulletin of the World Health Organization 55: 445-453.

Aycock, W.L., Lutman, G.E., and Foley, G.C. 1945. "Seasonal Prevalence as a Principle in Epidemiology." American Journal of Medical Science 209: 395-411.

Aycock, W.L., and Mueller, J.H. 1950. "Meningococcus Carrier Rates and Meningitis Incidence." Bacteriological Review 14: 115-160. (Seasonal variation in transmission of meningococcus).

Bagni, N., Mandrioli, P., and Puppi, G.L. 1976. "Atmospheric Spore Content at Mount Cimone, Italy, in Relation to the Winds." International Journal of Biometeorology 20: 184-189.

Bailey, S.F., Eliason, D.A., and Hoffmann, B.L. 1965. "Flight and Dispersal in the Mosquito Culex tarsalis Coquillet in the Sacramento Valley of California." Hilgardia 37: 73-113. (Effects of temperature, light intensity and wind).

Baranowska, M. and Gabryl, B. 1981. "Biometeorological Norm as Tolerance Interval of Man to Weather Stimuli." International Journal of Biometeorology 25: 123-126.

Barnes, A.M. and Poland, J.D. 1983. "Plague in the United States, 1982." Morbidity and Mortality Weekly Report 32: 19SS-24SS.

Barnicot, N.A. 1959. "Climatic Factors in the Evolution of Human Populations." Cold Spring Harbor Symposia on Quantitative Biology 24: 115-129.

Bashford, D.J., Donovan, T.J., Furniss, A.L., and Lee, J.V. 1979. "Vibrio Colerae in Kent." Lancet 1: 436-437. (Related to water temperature).

Bass, D.E., Kleeman, C.R., Quinn, M., Henschel, A., and Hegnauer, A.H. 1955. "Mechanisms of Acclimatization to Heat in Man." Medicine 34: 323-380.

Bellamy, R.E., Reeves, W.C., and Scrivani, R.P. 1958. "Relationships of Mosquito Vectors to Winter Survival of Encephalitis Viruses." American Journal of Hygiene 67: 90-100.

Berg, A. 1973. The Nutrition Factor. The Brookings Institution, Washington, D.C. (Nutrition and disease).

Biersteker, K. amd Evendijk, J.E. 1976. "Ozone, Temperature and Mortality in Rotterdam in the Summers of 1974 and 1975." Environmental Research 12: 214-217.

Black, R.E., Brown, K.H.W., Becker, S., and Yunus, M. 1982. "Longitudinal Studies of Infectious Diseases and Physical Growth of Children in Rural Bangladesh." American Journal of Epidemiology 115: 305-314. (Disease and malnutrition).

Blake, P.A. 1980. "Diseases of Humans (Other than Cholera) Caused by Vibrios." Annual Review of Microbiology 34: 341-367. (Prevalence related to water temperature).

Bleibtreu, H.K. (ed.) 1969. Evolutionary Anthropology - A Reader in Human Biology. Allyn and Bacon, Inc., Boston, Massachusetts. (Adaptation to temperature extremes included).

Bliss, C.I. and Blevins, D.L. 1959. "The Analysis of Seasonal Variation in Measles." American Journal of Hygience 70: 328-334.

Bowie, C. and Prothero, D. 1981. "Finding Causes of Seasonal Diseases Using Time Series Analysis." International Journal of Epidemiology 10: 87-92. (Ischaemic heart disease, temperature, and rainfall).

Boyd, J.T. 1960. "Climate, Air Pollution, and Mortality." British Journal of Preventive and Social Medicine 14: 123-135. (Respiratory disease, temperature, humidity, pollution, fog).

Bradley, A.K., Gilles, H.M., and Shehu, U. 1977. "Malumfashi Endemic Diseases Research Project, I. Some Ecological and Demographic Considerations." Annals of Tropical Medicine and Parasitology 71: 443-449.

Brandt, C.D., Kim, H.W., Rodriguez, W.J., Arrobio, J.O., Jeffries, B.C., and Parrott, R.H. 1982. "Rotovirus Gastroenteritis and Weather." Journal of Clinical Microbiology 16: 478-482.

Breeland, S.G. 1974. "Population Patterns of Anopheles Albimanus and Their Significance to Mosquito Abatement." Bulletin of the World Health Organization 50: 307-315. (Seasonal patterns).

Brenner, M.H. and Mooney, A. 1982. "Economic Change and Sex-specific Cardiovascular Mortality in Britain 1955-1976." Social Science and Medicine 16: 431-442. (Includes physical environmental disturbances, especially very cold temperatures).

Brown, G.W., Jr. (ed.). 1968. Desert Biology. Academic Press, New York, New York.

Bryson, R.A., and Murray, T.J. 1977. Climates of Hunger. The University of Wisconsin Press, Madison, Wisconsin. (Drought).

Bull, G.M. 1980. "The Weather and Deaths from Pneumonia." Lancet 1: 1405-8.

Bull, G.M., and Morton, J. 1975. "Seasonal and Short-Term Relationships of Temperature with Deaths from Myocardial and Cerebral Infarction." Age and Aging 4: 19-31.

Burnet, M. 1962. Natural History of Infectious Disease. Cambridge University Press, London, United Kingdom.

Burch, G.E. and De Pasquale, M.D. 1962. Hot Climates, Man and His Heart. Charles C. Thomas, Springfield, Illinois.

Bursell, E. 1957. "The Effect of Humidity on the Activity of Tsetse Flies." Journal of Experimental Biology 34: 42-51.

Center for Environmental Assessment Services. 1980. U.S. Social and Economic Effects of the Great 1980 Heat Wave and Drought. U.S.

Department of Commerce, National Oceanic and Atmospheric Administration, Washington, D.C.

Chambers, R., Longhurst, R., Bradley, D., and Feachem, R. 1979. "Seasonal Dimension to Rural Poverty: Analysis and Practical Implications." Journal of Tropical Hygiene 82: 156-172. (Nutrition, disease, migration, agriculture).

Chandler, T.J. 1967. "Temperature Regime in Towns - London's Heat Island." 589-597. In S.W. Tromp and W.H. Weihe (eds.), Biometeorology Vol. 2, pt. 2, Permagon Press, New York, New York.

Clarke, J.F. and Bach, W. 1971. "Comparison of the Comfort Conditions in Different Urban and Suburban Microenvironments." International Journal of Biometeorology 15: 41-54.

Collins, K.J., Dore, C., Exton-Smith, A.N., Fox, R.H., McDonald, I.C., and Woodward, P.M. 1977. "Accidental Hypothermia and Impaired Temperature Homeostasis in the Elderly." British Medical Journal 1: 353-356.

Curtiss, J.R.B. and Grahn, D. 1980. "Population Characteristics and Environmental Factors That Influence Level and Cause of Mortality/A Review." Journal of Environmental Pathology and Toxicology 4: 471-511. (Climate, geographic residence).

Czeizel, A., Vizkelety, T., and Szentpeteri, J. 1972. "Congenital Dislocation of the Hip in Budapest, Hungary." British Journal of Preventive and Social Medicine 26: 15-22. (Seasonality).

Davey, M.L. and Reid, D. 1972. "Relationship of Air Temperature to Outbreaks of Influenza." British Journal of Preventive and Social Medicine 26: 28-32.

Davies, J.E. 1982. "Sleeping Sickness and the Factors Affecting it in Botswana." Journal of Tropical Medicine and Hygiene 85: 63-71. (Tsetse fly, rainfall, temperature, migration, drought).

Davies, R.R. and Smith, L.P. 1974. "Weather and the Grass Pollen Content of the Air." Clinical Allergy 4: 95-108.

Davies, S.F.M., Joyner, L.P., and Kendall, S.B. 1963. Coccidiosis. Oliver and Boyd, London, United Kingdom. (Rainfall, humidity, temperature seasons).

Davis, T.R.A. and Joy, R.J.T. 1962. "Natural and Artifical Cold Acclimatization in Man" 286-303. In S.W. Tromp (ed.), Biometeorology, Permagon Press, Oxford, United Kingdom.

Derrick, E.H. 1965. "The Seasonal Variation of Asthma in Brisbane: Its Relation to Temperature and Humidity." International Journal of Biometeorology 9: 239-251.

de Torres, B.V., de Ilja, R.M., and Esparza, J. 1978. "Epidemiological Aspects of Rotavirus Infection in Hospitalized Venezuelan Children with Gastroenteritis." American Journal of Tropical Medicine and Hygiene 27: 567-572. (Seasonality, temperature, rainfall).

DePaolo, A. 1981. "Vibrio cholerae in Marine Foods and Environmental Waters: A Literature Review." Journal of Food Science 46: 66-70. (Temperature, seasons).

DeWitt, W.B. 1955. "Influence of Temperature on Penetration of Snail Hosts by Schistosoma mansoni Miracidia." Experimental Parasitology 4: 271-276.

Dimmick, R.L. and Akers, A.B. (eds.) 1963. An Introduction to Experimental Aerobiology, John Wiley and Sons, New York, New York.

Driscoll, D.M. 1983. "Human Biometeorology in the 1970's." International Journal of Environmental Studies 20: 137-147.

Duckworth, F.S. and Sandberg, J.S. 1954. "The Effect of Cities upon Horizontal and Vertical Temperature Gradients." Bulletin American Meteorological Society 35: 198-207.

Edholm, O.G. (ed.). 1965. The Physiology of Human Survival. Academic Press, New York, New York.

Edmonds, R.L. (ed.) 1979. Aerobiology, The Ecosystems Approach. Dowden, Hutchinson and Ross, Inc. Stroudsburg, Pennsylvania.

Ehrlich, R., Miller, S., and Walker, R.L. 1970. "Effects of Atmospheric Humidity and Temperature on the Survival of Airborne Flavobacterium." Applied Microbiology 20: 884-887.

Ehrlich, R., Miller, M., and Walker, R.L. 1970. "Relationship Between Atmospheric Temperature and Survival of Airborne Bacteria." Applied Microbiology 19: 245-249.

El-Eman, M.A. and Madsen, H. 1982. "The Effect of Temperature, Darkness, Starvation and Various Food Types on Growth, Survival and Reproduction of Helisoma duryi, Biomphalaria alexandrina, and Bulinus truncatus (Gastropoda: Planorbidae)." Hydrobiologia 88: 265-275. (Snail hosts for schistosomes).

Ellis, F.P., Exton-Smith, A.N., Foster, K.G., and Weiner, J.S. 1976. "Eccrine Sweating and Mortality During Heat Waves in Very Young and Very Old Persons." Israeli Journal of Medical Science 12: 815-817.

Ellis, F.P., Prince, H.P., Lovatt, G., and Whittington, R.M. 1980. "Mortality and Morbidity in Birmingham (England) During the 1976 Heat Wave." Quarterly Journal of Medicine 49: 1-8.

Ellis, F.P. 1973. "Mortality from Heat Illness in the United States: A Progress Report." Archives Des Sciences Physiologiques 27: A573-A577.

Ellis, F.P. 1976. "Mortality and Morbidity Associated with Heat Exposure." International Journal of Biometeorology 20, Supplement: 36-40.

Feachem, R., Miller, C., and Drasar, B. 1981. "Environmental Aspects of Cholera Epidemiology. II. Occurrence and Survival of Vibrio cholerae in the Environment." Tropical Diseases Bulletin 78: 865-880. (Survival in relation to water temperature).

Fellowes, D.S.B. and Proctor, I.R.D. 1973. "The Incidence of the Common Cold in Relation to Certain Meteorological Parameters." International Journal of Biometeorology 17: 193-203.

Fleischer, S.L.M. and Asnani, G.C. 1978. "The Influence of Weather on Asthma in Nairobi." International Journal of Biometeorology 22: 263-270.

Folinsbee, L.J. (ed.). 1978. Environmental Stress - Individual Human Adaptations. Academic Press, New York, New York.

Folk, G.E., Jr. 1974. Textbook of Environmental Physiology. Lee and Febiger, Philadelphia, Pennsylvania.

Ford, M.J. 1981. The Changing Climate: Responses of the Natural Fauna and Flora. George Allen and Urwin, London, United Kingdom.

Fox, R.H., MacGibbon, R. Davies, L., Woodward, P.M. 1973. "Problem of the Old and the Cold." British Medical Journal 1: 21-24.

Foy, H.M., Hall, C.E., Cooney, M.K., Allan, I.D. and Fox, J.P. 1983. "Influenza Surveillance in the Pacific Northwest 1976-1980." International Journal of Epidemiology 12: 353-356. (Seasonality).

Fulton, J.D. and Mitchell, R.B. 1966. "Microorganisms of the Upper Atmosphere II. Microorganisms in Two Types of Air Masses at 690 Meters Over a City." Applied Microbiology 14: 232-236.

Fulton, J.D. 1966. "Microorganisms of the Upper Atmosphere V. Relationship Between Frontal Activity and the Micropopulation at Altitude." Applied Microbiology 14: 245-250.

Furcolow, M.L. 1961. "Airborne Histoplasmosis." Bacteriologic Review 25: 301-309.

Garrett-Jones, C. 1962. "The Possibility of Active Long-distance Migrations by Anopheles pharoensis Theobald." Bulletin of the World Health Organization 27: 299-302.

Gemmell, M.A. 1978. "The Effect of Weather on Tapeworm Eggs and Its Epidemiological Implications." 83-94. In T.E. Gibson (ed.), World Meteorological Organization Technical Note No.159, World Meteorological Organization, Geneva, Switzerland.

Gentilli, J. 1980. "Some Climatic Factors in Australian Health." Social Science and Medicine 14D: 85-100.

Gillett, J.D. 1974. "Direct and Indirect Influences of Temperature on the Transmission of Parasites from Insects to Man." 79-95. In A.E.R. Taylor and R. Muller (eds.), The Effects of Meteorological Factors Upon Parasites, Blackwell Scientific Publications, London, United Kingdom.

Gironi, B. 1976. Man, Climate, and Architecture 2nd edition. Applied Science Publishers, Ltd., London, United Kingdom.

Glaser, E.M. 1963. "Circulatory Adjustments in the Arid Zone." 131-148. In Environmental Physiology and Psychology in Arid Conditions. Reviews of Research. United Nations Educational, Scientific and Cultural Organization, Unesco, Paris, France.

Goldsmith, J.R. and Perkins, N.M. 1967. "Seasonal Variations in Mortality." 97-114. In S.W. Tromp and W.H. Weihe (eds.), Biometeorology, Vol. 2, pt. 1, Permagon Press, New York, New York.

Gordon, J.E. and Erhardt, C.L. 1958. "Weather and Death." American Journal of the Medical Sciences 236: 383-399.

Gordon, R.M. and Lavoipierre, M.M.J. 1972. "The Adaptation of Insects to Climatic Conditions." 71-74. In Entomology for Students of Medicine, Blackwell Scientific Publications, Oxford, United Kingdom.

Greenberg, J.H., Bromberg, J., Reed, C.M., Gustafson, T.L., and Beauchamp, R.A. 1983. "The Epidemiology of Heat-Related Deaths, Texas 1950, 1970-79, and 1980." American Journal of Public Health 73: 805-807.

Greenberg, L., Field, F., Reed, J.I., and Erhardt, C.L. 1967. "Asthma and Temperature Change." 3-6. In S.W. Tromp and W.H. Weihe (eds.), Biometeorology Vol. 2, pt. 1, Permagon Press, New York, New York.

Gregorczuk, M. and Cena, K. 1967. "Distribution of Effective Temperature Over the Surface of the Earth." International Journal of Biometeorology 11: 145-149. (Temperature, humidity, thermal stress in man).

Gregorczuk, M. 1968. "Bioclimates of the World Related to Air Enthalpy." International Journal of Biometeorology 12: 35-39. (Human thermal sensations).

Gregory, P.H. 1973. The Microbiology of the Atmosphere. John Wiley and Sons, New York, New York.

Griffiths, J.F. 1983. "Climatic Classification." International Journal of Environmental Studies 20: 115-125. (Crops, mammals, building design, microscale climates).

Gumble, A., Otori, U., Ritchie, L.S., and Hunter, G.W., III. 1957. "The Effect of Light, Temperature, and pH onthe Emergence of Schistosoma Japonicum Cercariae from Oncomelania Nosophoro." American Mircoscopy Society Transactions 76: 87-92.

Haberman, S., Capildeo, R., and Rose, F.C. 1981. "The Seasonal Variation in Mortality from Cerebrovascular Disease." Journal of the Neurological Sciences 52: 25-36.

Hammel, H.T. 1969. "Terrestrial Animals in the Cold: Recent Studies of Primative Man." 322-344. In H.K. Bleibtreu (ed.), Evolutionary Anthropology - A Reader in Human Biology, Allyn and Bacon, Inc., Boston, Massachusetts.

Hardy, J.L., Rosen, L., Kramer, L.D., Presser, S.B., Shroyer, D.A., and Turell, M.J. 1980. "Effect of Rearing Temperature on Transovarial Transmission of St. Louis Encephalitis Virus in Mosquitoes." Journal of Tropical Medicine and Hygiene 29: 963-968.

Hare, E., Price, J., and Slater, E. 1974. "Mental Disorder and Season of Birth: A National Sample Compared with the General Population." British Journal of Psychiatry 124: 81-86.

Harlan, W.R. 1981. "Physical and Psychosocial Stress and the Cardiovascular System." Circulation 63: 266A-271A.

Haufe, W.O. 1964. "Quantitative Measurements of Activity of Aedes Aegypti (L.) (Culicidae: Diptera) in Response to Changes in the Hygrothermal Environment." International Journal of Biometeorology 7: 245-264. (Malaria).

Haufe, W.O. 1972. "Meteorological Effects on Arthropods (Including Insects, Ticks, and Mites) as Vectors of Human and Animal Diseases." 109-117 and 148-151. In S.W. Tromp and J.J. Bourma (eds.), Progress in Biometeorology, Vol. 1, pt. III, Period 1963-1970, Swets and Zeitlinger, Amsterdam, The Netherlands.

Helfand, L.A. and Bridger, C. 1971. "Hippocrates, Thermal Stress, and Stroke Mortality - 1966." Weatherwise 24: 100-105.

Hess, A.D. and Hayes, R.O. 1967. "Seasonal Dynamics of Western Encephalitis Virus." American Journal of Medical Science 253: 333-348.

Hieber, J.P., Shelton, S., Nelson, J.D., Leon, J., and Mohs, E. 1978. "Comparison of Human Rotavirus Disease in Tropical and Temperate Settings." American Journal of Diseases of Childhood 132: 853-858. (Gastroenteritis in different climates).

Hira, P.R. and Muller, R. 1966. "Studies of the Ecology of Snails Transmitting Urinary Schistosomiasis in Western Nigeria." Annals of Tropical Medicine and Parasitology 60: 198-211.

Holland, W.W. and Spicer, C.C. 1967. "Influence of Weather on Respiratory Disease." 30-40. In S.W. Tromp and W.H. Weihe (eds.), Biometeorology, Vol. 2, pt. 1, Permagon Press, New York, New York.

Hollis, N.R.S., and Goldman, R.F. 1977. Clothing Comfort. Ann Arbor Science, Ann Arbor, MI.

Hope-Simpson, R.E. 1981. "The Role of Season in the Epidemiology of Influenza." Journal of Hygiene, Cambridge 86: 35-47.

Hori, S., Tsujita, J., Mayuzumi, M., Tanaka, N., Toda, Y., and Araki, T. 1979. "Effect of Long Term Residence in the Temperate Zone on the Physique and Sweating Reaction of Subtropical Natives." International Journal of Biometeorology 23: 255-261. (Acclimation).

Horvath, S.M., and Rochelle, R.D. 1977. "Hypothermia in the Aged." Environmental Health Perspectives 20: 127-130.

Howe, G.M. (ed.) 1977. A World Geography of Human Diseases. Academic Press, New York, New York.

Ingold, C.T. 1971. Fungal Spores, Their Liberation and Dispersal. Clarendon Press, Oxford, United Kingdom. (Wind, rain, water supply).

Jankowiak, J. 1964. "Effects of Wind on Man." 343-357. In S. Licht (ed.), Medical Climatology, E. Licht, New Haven, Connecticut.

Jusatz, H.J. 1958. "Medical Cartography as a Scientific Method for the Investigation of Tropical Diseases." 124-127. Proceedings of the 6th International Congress on Tropical Medicine and Malaria, Institute Medical Tropical, Lisbon, Portugal. (Dengue, leishamania, yellow fever).

Jusatz, H.J. 1961. World Atlas of Epidemic Diseases. Falk Verlag, Hamburg, The Federal Republic of Germany.

Jusatz, H.J. 1962. "The World Atlas of Epidemic Diseases and Its Significance for Bioclimatological Classifications." 141-149. In S.W.

Tromp (ed.), Biometeorology of the 2nd International Bioclimatological Congress, 1960, Permagon Press, New York, New York.

Jusatz, H.J. 1966. "The Importance of Biometeorological and Geomedical Aspects in Human Ecology." International Journal of Biometeorology 10: 323-334.

Jusatz, H.J. 1967. "Seasonal Diseases and Bioclimatologic Classification." 944-947. In S.W. Tromp and W.H. Weihe (eds.), Biometeorology 2, part 2, Proceedings of the 3d International Biometeorological Congress, September 1963, Permagon Press, New York, New York.

Kadar, L., Paldy, A., and Desi, I. 1982. "Relationship Between Variations of Sunspot Activity, Climatic and Environmental Conditions and the Incidence of Some Infectious Diseases." Geographic Medicine (Hungary) 12: 121-140.

Karl, T.R. and Quayle, R.G. 1981. "The 1980 Summer Heat Wave and Drought in Historical Perspective." Monthly Weather Review 109: 2055-2073.

Karl, T.R. and Koscielny, A.J. 1982. "Drought in the United States: 1895-1981." Journal of Climatology 2: 313-329.

Kartman, L. and Prince, F.M. 1956. "Studies on Pasturella Pestis in Fleas. V. The Experimental Plague-Vector Efficiency of Wild Rodent Fleas Compared with Xenopaylla Cheopis, together with Observations on the Influence of Temperature." American Journal of Tropical Medicine and Hygiene 5: 1058-1070.

Katayama, K. and Momiyama-Sakamoto, M. 1972. "The Seasonal Variation of Stroke Mortality and Its Relation to its Temperature in Japan." Papers in Meteorology and Geophysics 23: 329-345.

Katayama, K. and Momiyama-Sakamoto, M. 1973. "A Biometeorological Study in the Seasonal Variation of Mortality from Cerebral Haemorrhage, and Cerebral Thrombosis and Embolism." Papers in Meteorology and Geophysics 24: 311-329.

Kerslake, D. McK. 1972. The Stress of Hot Environments. University Press, Cambridge, United Kingdom.

Kew, M.C. 1976. "Temperature Regulation in Heatstroke in Man." Israeli Journal of Medical Science 12: 759-764.

Kevan, S.M. 1979. "Season of Life-Season of Death." Social Science and Medicine 13D: 227-232.

King, H.H. and Pandit, C.G., 1931. "A Summary of the Rat-Flea Survey of the Madras Presidency with a Discussion on the Association of Flea

Species with Climate and with Plague." Indian Journal of Medical Research 19: 357-392.

Kloos, H., Lemma, A., Kirub B., Gebre, A., Mazengia, B., Feleke, G., and DeSole, G. 1980. "Intestinal Parasitism in Migrant Farm Labor Populations in Irrigation Schemes in the Awash Valley, Ethiopia, and in Major Labour Source Areas." Ethiopian Medical Journal 18: 53-61. (Effects of migration and irrigation).

Klumpp, R.K. and Chu, K.Y. 1977. "Ecological Studies of Bubinus rohlfsi, the Intermediate Host of Schistosoma haematobium in the Volta Lake." Bulletin of the World Health Organization 55: 715-730. (Seasonality of transmission).

Knight, V. (ed.) 1973. Viral and Mycoplasmal Infections of the Respiratory Tract. Lea and Febiger, Philadelphia, Pennsylvania.

Knight V. 1980. "Viruses as Agents of Airborne Contagion." Annals of the New York Academy of Science 353: 147-156. (Seasonality).

Knochel, J.P. 1974. "Environmental Heat Illness, An Eclectic Review." Archives of Internal Medicine 133: 841-863.

Knox, E.G. 1981. "Meteorological Associations of Cerebrovascular Disease Mortality in England and Wales." Journal of Epidemiology and Community Health 35: 220-223.

Konno, T., Suzuki, H., Katsushima, N., Imai, A., Tazawa, F., Kitaoka, T., Sakamato, M., Yazaki, N., and Ishida, N. 1983. "Influence of Temperature and Relative Humidity on Human Rotavirus Infection in Japan." The Journal of Infectious Diseases 147: 125-128.

Kuntz, R.E. 1947. "Effect of Light and Temperature on Emergence of Schistosoma Mansoni Cecariae." American Microscopic Society Transactions 66: 37-49.

Kutschenreuter, P.H. 1959. "A Study of the Effect of Weather on Mortality." Transactions of the New York Academy of Science 22: 126-138.

Kuzma, R.J., Kuzma, C.M., and Buncher, C.R. 1977. "Ohio Drinking Water Source and Cancer Rates." American Journal of Public Health 67: 725-729. (Water quality - surface versus ground water).

Lambrecht, F.L. 1974. "Entomological Aspects of Filariasis Control in Sri Lanka." Bulletin of the World Health Organization 51: 133-143. (Rainfall - wet zone, dry zone).

Landsberg, H.E. 1969. Weather and Health: An Introduction to Biometeorology. Doubleday and Co., Garden City, New York.

Landsberg, H.E. 1970. "Man-Made Climatic Changes." Science 170: 1265-1274.

Landsberg, H.E. 1971. "Interaction of Man and His Atmospheric Environment." 65-72. In Meteorology as Related to the Human Environment. WMO Special Environmental Report No. 2. World Meteorological Organization No. 312.

Landsberg, H.E. 1972. The Assessment of Human Bioclimate, A Limited Review of Physical Parameters. WMO Technical Note No. 123. World Meteorological Organization, Geneva, Switzerland.

Lebowitz, M.D. 1973. "A Comparative Analysis of the Stimulus-Response Relationship between Mortality and Air Pollution-Weather." Environmental Research 6: 106-118.

Leck, I. and Record, R.G. 1966. "Seasonal Incidence of Anencephalus." British Journal of Preventive and Social Medicine 20: 67-75.

Lee, D.H.K. 1982. "Epidemic Heat Effects." Journal of the American Medical Association 247: 3354-3355.

Leithead, C.S., and Lind, A.R. 1964. Heat Stress and Heat Disorders. Cassell and Co. Ltd., London, U.K.

Levine, J.A. 1969. "Heat Stroke and the Aged." American Journal of Medicine 47: 251-258.

Licht, S. (ed). 1964. Medical Climatology. E. Licht, New Haven, Connecticut.

Lidwell, O.M., Morgan, R.W., and Williams, R.E.O. 1965. "The Epidemiology of the Common Cold IV. The Effect of Weather." Journal of Hygiene, Cambridge 63: 427-439.

Loutit, M.W., and Miles, J.A.R. (eds). 1978. Microbial Ecology. Springer-Verlag, Berlin, The Federal Republic of Germany.

Lye, M. and Kamal, A. 1977. "Effects of a Heatwave on Mortality-rates in Elderly Inpatients." Lancet 2: 529-531.

Lyster, W.R. 1976. "Death in Summer." Lancet ii: 469. (Heat waves - London).

Macfarlane, A. and Waller, R.E. 1976. "Short Term Increases in Mortality During Heat Waves." Nature 264: 434-436.

Macfarlane, A. 1977. "Daily Mortality and Environment in English Conurbations. I: Air Pollution, Low Temperature, and Influenza in Greater London." British Journal of Preventive and Social Medicine 31: 54-61.

Macfarlane, A. 1978. "Daily Mortality and Environment in English Conurbations. II: Death During Summer Hot Spells in Greater London." Environmental Research 15: 332-341.

Macpherson, R.K. and Ofner, F. 1965. "Heat and the Survival of the Aged and Chronically Ill." The Medical Journal of Australia, Feb: 292-295.

Macpherson, R.K., Ofner, F., and Welch, J.A. 1967. "Effect of the Prevailing Air Temperature on Mortality." British Journal of Preventive Medicine 21: 17-21.

Markell, E.K., and Voge, M. 1981. Medical Parasitology. W.B. Saunders, Philadelphia, Pennsylvania.

Marmor, M. 1978. "Heat Wave Mortality in Nursing Homes." Environmental Research 17: 102-115.

Mason, T.J., Fraumeni, J.F., Hoover, R., and Blot, W.J. 1981. An Atlas of Mortality from Selected Diseases, NIH Publication No. 81-2397. U.S. Department of Health and Human Services. Washington, D.C.

Mata, L.J., Urrutia, J.J., and Lechtig, A. 1971. "Infection and Nutrition of Children of a Low Socioeconomic Rural Community." American Journal of Clinical Nutrition 24: 249-259.

Mata, L.J., and Behar, M. 1975. "Malnutrition and Infection in a Typical Rural Guatemalan Village: Lessons for the Planning of Preventive Measures." Ecology of Food and Nutrition 4: 41-47.

May, J. 1958. The Ecology of Human Disease. MD Publications, New York, New York.

McElroy, A. and Townsend, P.K. 1979. Medical Anthropology in Ecological Perspective, Duxbury Press, North Scituate, Massachusetts. (Climatic stress, agriculture, parasites, etc.).

McLaughlin, J.T. and Shulman, M.D. 1977. "An Anthropocentric Summer Severity Index." International Journal of Biometeorology 21: 16-28.

McLintock, J., Burton, A.N., Dillenberg, H., and Rempel, J.G. 1966. "Ecological Factors in the 1963 Outbreaks of Western Encephalitis in Saskatchewan." Canadian Journal of Public Health 57: 561-575.

Metropolitan Life Insurance Company. 1974. "Mortality from Tornados, Hurricanes, and Floods." Metropolitan Life Insurance Company Statistical Bulletin, December 1974: 4-7.

Middleton, P.J., Szymanski, M.T., and Petric, M. 1977. "Viruses Associated with Acute Gastroenteritis in Young Children." American

Journal of Diseases of Childhood 131: 733-737. (Relationship to ambient temperature).

Miller, C.J., Drasar, B.S., and Feachem, R.G. 1982. "Cholera and Estuarine Salinity in Calcutta and London." Lancet 1: 1216-1218. (Seasonality).

Miura, T. and Shimura, M. 1980. "Epidemic Seasonal Infertility - A Hypothesis for the Cause of Seasnal Variation of Births." International Journal of Biometeorology 24: 91-95.

Moe, K. and Shirley, J.A. 1982. "The Effects of Relative Humidity and Temperature on the Survival of Human Rotavirus in Faeces." Archives of Virology 72: 179-186.

Mogi, M. 1983. "Relationship between Number of Human Japanese Encephalitis Cases and Summer Meteorological Conditions in Nagasaki, Japan." American Journal of Tropical Medicine and Hygiene 32: 170-174.

Momiyama, M. 1968. "Biometeorological Study of the Seasonal Variation of Mortality in Japan and Other Countries on the Seasonal Disease Calendar." International Journal of Biometeorology 4: 377-393.

Mousa, A.H. and Abou El-Hassan, A.A. 1972. "The Effect of Water Temperature on the Snail Intermediate Hosts of Schistosomiasis in Egypt." Journal of the Egyptian Medical Association 55: 148-165.

Neill, W.A., Duncan, D.A., Kloster, F., and Mahler, D.J. 1974. "Response of Coronary Circulation to Cutaneous Cold." American Journal of Medicine 56: 471-476.

Neutra, R. 1974. "Meteorological Factors and Eclampsia." Journal of Obstetrics and Gynaecology of the British Commonwealth 81: 833-840. (Seizures during pregnancy).

O'Donnell, T.F. 1975. "Acute Heat Stroke - Epedemiologic, Biochemical, Renal, and Coagulation Studies." Journal of the American Medical Association 234: 824-828.

Ohno, Y. 1969. "Biometeorological Studies on Cerebrovascular Diseases. I. Effects of Meteorological Factors on the Death from Cerebrovascular Accident." Japanese Circulation Journal 33: 1285-1298.

Ohno, Y. 1969. "Biometeorological Studies on Cerebrovascular Diseases. II. Seasonal Observation on Effects of Meteorologic Factors on the Death from Cerebrovascular Accident." Japanese Circulation Journal 33: 1299-1308.

Ohno, Y. 1969. "Biometeorological Studies on Cerebrovascular Diseases. III. Effects by the Combination of Meteorological Changes on the Death

from Cerebrovascular Accident." Japanese Circulation Journal 33: 1309-1314.

Ohno, Y., Horibe, H., Hayakawa, N., Aoki, N., and Okada, H. 1970. "Biometeorological Studies on Cerebrovascular Diseases. IV. Evaluation of Meteorological Factors, Their Changes or Combinations on the Occurence of Cerebrovascular Accident." Japanese Circulation Journal 34: 69-75.

Olson, W.P. 1969. "Rat-Flea Indices, Rainfall, and Plague Outbreaks in Vietnam, with Emphasis on the Pleiku Area." American Journal of Tropical Medicine and Hygiene 18: 621-628.

Onori, E. and Grab, B. 1980. "Indicators for the Forecasting of Malaria Epidemics." Bulletin of the World Health Organization 58: 91-98. (Including air temperature, relative humidity and importation of parasites).

Osiba, S. 1957. "The Seasonal Variation of Basal Metabolism and Activity of the Thyroid Gland in Man." Japanese Journal of Physiology 7: 355-365.

Osterman, P.O., Lovstrand, K.G., Lundberg, P.O., Lundquist, S., and Muhr, C. 1981. "Weekly Headache Periodicity and the Effect of Weather Changes on Headache." International Journal of Biometeorology 25: 39-45.

Page, T., Harris, R.H., and Epstein, S.S. 1976. "Drinking Water and Cancer Mortality in Louisiana." Science 193: 55-57. (Water quality).

Parry, M. 1967. "The Urban "Heat Island"." 616-624. In S.W. Tromp and W.L. Weihe (eds.), Biometeorology, Vol. 2, pt. 2, Permagon Press, New York, New York.

Paul, M.O. and Erinle, E.A. 1982. "Influence of Humidity on Rotavirus Prevalence Among Nigerian Infants and Young Children with Gastroenteritis." Journal of Clinical Microbiology 15: 212-215.

Pedgley, D.E. 1980. Weather and Airborne Organisms. Technical Note No. 173." World Meteorological Organization No. 562, Geneva, Switzerland.

Perkins, W.A. and Vaughan, L.M. 1961. "Public Health Implications of Airborne Infection: Physical Aspects." Bacteriological Review 25: 347-355. (Effects of atmospheric conditions).

Posey, C. 1980. "Heat Wave." Weatherwise 33: 112-116.

Pyle, G.F. and Cook, R.M. 1978. "Environmental Risk Factors of California Encephalitis in Man." Geography Reviews 68: 157-170. (Relationship of outbreaks to rainfall).

Ragab, H.A. 1949. "Effect of Humidities and Temperatures on the Size and Numbers of Oocysts of Plasmodium gallinaceum Transmitted by a Mosquito." Nature 163: 643-644.

Rainey, R.C. 1973. "Airborne Pests and the Atmospheric Environment." Weather 28: 224-239.

Rao, B.L., Kadam, S.S., Parni, K.M., and Kothavale, V.S. 1982. "Epidemiological, Clinical and Virological Features of Influenza Outbreaks in Pune, India, 1980." Bulletin of the World Health Organization 60: 639-642. (Relationship to summer, rainy and winter seasons).

Robins-Browne, R.M. 1984. "Seasonal and Racial Incidence of Infantile Gastroenteritis in South Africa." American Journal of Epidemiology 119: 350-355.

Robson, J.R.K., Larkin, F.A., Sandretto, A.M., and Tadayyon, B. 1972. Malnutrition, Its Causation and Control. Vol. I and II. Gordon and Breach, New York, New York. (Relationship to climate and interrelationships with diseases).

Rose, V., Hewitt, D., and Milner, J. 1972. "Seasonal Influences on the Risk of Cardiac Malformation. Nature of the Problem and some Results from a Study of 10,077 Cases." International Journal of Epidemiology 1: 235-244.

Rowland, M.G.M., Leung, T.S.M., and Marshall, W.C. 1980. "Rotavirus Infection in Young Gambian Village Children." Transactions of the Royal Society of Tropical Medicine and Hygiene 74: 663-671. (Related to rainy season).

Sakamoto-Momiyama, M. 1977. Seasonality in Human Mortality, A Medico-Geographic Study. University of Tokyo Press, Tokyo, Japan.

Sargent, F., II. 1960. "Changes in Ideas on the Climate Origin of Disease." Bulletin American Meteorological Society 41: 238-244.

Scragg, R. 1981. "Seasonality of Cardiovascular Disease Mortality and the Possible Protective Effect of Ultra-violet Radiation." International Journal of Epidemiology 10: 337-341.

Scrimshaw, N.S., Taylor, C.E., and Gordon, J.E. 1968. Interaction of Nutrition and Infection. World Health Organization (WHO), Monograph Series Number 57, WHO, Geneva, Switzerland.

Segal, M. and Mahrer, Y. 1979. "Heat Load Conditions in Israel - A Numerical Mesoscole Model Study." International Journal of Biometeorology 23: 279-284. (Discomfort index, skin temperature, hot dry conditions).

Service, M.W. 1977. "Ecological and Biological Studies on Aedes Cantans (Meig.) (Diptera: Culicidae) in Southern England." Journal of Applied Ecology 14: 159-196. (Development at different temperature, seasonal incidence, vector for arbovirus).

Service, M.W. 1978. "The Effect of Weather on Mosquito Biology." 151-167. In T.E. Gibson (ed.), Weather and Parasitic Animal Disease. World Meteorological Organization Technical Note No. 159. World Meteorological Organization, Geneva, Switzerland.

Shadrin, A.S., Marinich, I.G., and Taros, L.Y. 1977. "Experimental and Epidemiological Estimation of Seasonal and Climato-Geographical Features of Non-specific Resistance of the Organism to Influenza." Journal of Hygiene, Epidemiology, Microbiology and Immunology 21: 155-161.

Shattuck, G.C. and Hilferty, M.M. 1936. "Distribution of Heat Effects In Various Parts of the World." New England Journal of Medicine 214: 458-467.

Shattuck, G.C. and Hilferty, M.M. 1932. "Sunstroke and Allied Conditions in the United States." American Journal of Tropical Medicine 12: 223-244.

Shibolet, S., Lancaster, M.C., and Danon, Y. 1976. "Heat Stroke: A Review." Aviation, Space, and Environmental Medicine 47: 280-301.

Shiff, C.J. 1964. "Studies on Bulinus (Physopsis) Globosus in Rhodesia. I. The Influence of Temperature on the Intrinsic Rate of Natural Increase." Annals of Tropical Medicine and Parasitology 58: 94-105. (Schistosomiasis).

Shiffman, M.A., Schneider, R., Turner, A.G., and Helms, R.W. 1976. "Seasonality in Water Related Intestinal Disease in Guatemala." International Journal of Biometeorology 20: 223-229. (Contaminated water, disease, and nutritional interactions).

Silver, I.H. (ed.) 1970. Aerobiology, Proceedings of the Third International Symposium, University of Sussex, England, September 1969. Academic Press, New York, New York.

Singh, I., Chohan, I.S., Lal, M., Khanna, P.K., Srivastava, M.C., Nanda, R.B., Lamba, J.S., and Malhotra, M.S. 1977. "Effects of High Altitude Stay on the Incidence of Common Diseases of Man." International Journal of Biometeorlogy 21: 93-122. (Comparison of diseases in relatively stable, dry, cold climate (high altitude) with hot, humid plains).

Smith, C.E., Pappagianis, D., Levine, H.B., and Saito, M. 1961. "Human Coccidioidomycosis." Bacteriological Review 25: 310-320. (Survival in low humidity, high temperature climates).

Smith, L.P. 1974. "Meteorological Factors of Importance in Biological Systems." 1-12. In A.E.R. Taylor and R. Muller (eds.), The Effects of Meteorological Factors upon Parasites, Symposia of the British Society for Parasitology, Vol. 12, Blackwell Scientific Publications, Oxford, United Kingdom.

Sohar, E., Birenfeld, C., Shoenfeld, Y., and Shapiro, Y. 1978. "Description and Forecast of Summer Climate in Physiologically Significant Terms." International Journal of Biometeorology 22: 75-81. (Discomfort index).

Sprung, C.L. 1979. "Hemodynamic Alterations of Heat Stroke in the Elderly." Chest 75: 362-366.

Staden, O.D. 1952. "Experimental Infection of Australorbis Glabratus with Schistcsoma Mansoni. I. Individual and Mass Infection of Snails and the Relationship of Infection to Temperature and Season." Annals of Tropical Medicine and Parasitology 46: 48-53.

Steegman, A.T., Jr. 1975. "Human Adaptation to Cold." 130-166. In A. Damon (ed.), Physiological Anthropology, Oxford University Press, London, United Kingdom.

Strickland, G.T. (ed.). 1984. Hunter's Tropical Medicine. W.B. Saunders, Philadelphia, Pennsylvania.

Sulman, F.G. 1976. Health, Weather and Climate. S. Karger, Basel, Switzerland.

Sulman, F.G. 1981. Short- and Long-term Changes in Climate Vol. II, Chapter 1: "Man's Reaction to Short-term Climate Changes," 1-20. Chapter 2: "Medical Impact of Evil Winds." 21-38. CRC Press, Inc., Boca Raton, Florida.

Taylor, A.E.R. and Muller, R. (eds). 1974. The Effects of Meteorological Factors upon Parasites. Blackwell Scientific Publishers, Oxford, United Kingdom.

Terjung, W.H. 1967. "The Geographic Application of Some Selected Physio-Climatic Indices to Africa." International Journal of Biometeorology 11: 5-19.

Terjung, W.H. 1968. "World Patterns of the Distribution of the Monthly Comfort Index." International Journal of Biometeorology 12: 119-151.

Terjung, W.H. and Louie, S.S-F. 1971. "Potential Solar Radiation Climates of Man." Annals of the Association of American Geographers 61: 481-500. (Solar heat load on man).

Thom, E.C. 1959. "The Discomfort Index." Weatherwise 12: 57-60.

Tillett, H.E., Smith, J.W.G., and Clifford, R.E. 1980. "Excess Morbidity and Mortality Associated with Influenza in England and Wales." Lancet 1: 793-795. (Excess mortality due to cold weather).

Tout, D.G. 1977. "Effective Temperature and the Hot Spell of June-July 1976." Weather 32: 67-72.

Tout, D.G. 1978. "Mortality in the June-July 1976 Hot Spell." Weather 33: 221-226.

Tout, D.G. 1980. "Aspects of the Human Biometeorology of the 1978/79 Winter in the United Kingdom." Weather 35: 235-241.

Tout, D.G. 1980. "The Discomfort Index, Mortality and the London Summers of 1976 and 1978." International Journal of Biometeorology 24: 323-328.

Tromp, S.W. 1964. "Weather, Climate, and Man." 283-293. In D.N. Dill (ed.), Handbook of Physiology, section 4. Adaptation to the Environment, Williams and Wikins, Baltimore, Maryland.

Tromp, S.W. and Bouma, J. 1965. "Effect of Weather on Asthmatic Children in the Eastern Part of the Netherlands." International Journal of Biometeorology 9: 233-238.

Tromp, S.W. 1968. "Influence of Weather and Climate on Asthma and Bronchitis." Review of Allergy 22: 1027-1044.

Tromp, S.W. and Bouma, J.J. (eds.). 1977. Progress in Biometeorology, Vol. I and II. Swets and Zeitlinger, Amsterdam, The Netherlands.

Tromp, S.W. and Bouma, J.J. 1979. Biometeorological Survey, Vol. 1, pt. A, Human Biometeorology, Heyden, Philadelphia, Pennsylvania.

Tromp, S.W. 1980. Biometeorology. Heyden and son, Philadephia, Pennsylvania.

U.S. Department of Health and Human Services. 1981. An Atlas of Mortality from Selected Diseases, National Institute of Health, Publication No. 81-2397. Washington, D.C.

Ulberg, L.C. 1958. "The Influence of High Temperature on Reproduction." Journal of Heredity 49: 62-64.

United Nations Educational, Scientific and Cultural Organization (UNESCO). 1963. Environmental Physiology and Psychology in Arid Conditions. UNESCO, Geneva, Switzerland.

Velimirovic, B. and Subramanian, M. 1972. "The Pattern of Morbidity after Typhoons in a Tropical City." International Journal of Biometeorology 16: 343-360.

Wallen, C.C. 1974. A Brief Survey of Meteorology as Related to the Biosphere. World Meteorological Organization (WMO) Special Environmental Report No. 4. WMO, Geneva, Switzerland. (Chapter VI - Human Biometeorology).

Weihe, W.H. 1976. "The Application of Meteorology in Medical Sciences." International Journal of Biometeorology 20: 157-165.

Weihe, W.H. 1979. "Climate, Health, and Disease." 313-364. In Proceedings of the World Climate Conference, Geneva, February 12-23, 1979, World Meteorological Organization No. 53, Geneva, Switzerland.

West, P.A. and Lee, J.V. 1982. "Ecology of Vibrio Species, Including Vibrio cholerae, in Natural Waters of Kent, England." Journal of Applied Bacteriology 435-448. (Seasonality and effects water temperature).

West, R.R., Lloyd, S., and Roberts, C.J. 1973. "Mortality from Ischaemic Heart Disease-Assocation with Weather." British Journal of Preventive Medicine 27: 36-40.

World Health Organization. 1972. Health Hazards of the Human Environment. WHO, Geneva, Switzerland.

Wyndham, C.H., Strydom, N.B., Morrison, J.F., Williams, C.G., Bredell, G.A.G., Von Rahden, M.J.E., Holdsworth, L.D., Van Graan, C.H., Van Rensburg, A.J., and Munro, A. 1964. "Heat Reactions of Caucasians and Bantu in South Africa." Journal of Applied Physiology 19: 598-606.

Wyndham, C.H., Strydom, N.B., Munro, A., MacPherson, R.K., Metz, B., Schraff, G., and Schieber, J. 1964. "Heat Reactions of Caucasians in Temperate, in Hot, Dry, and in Hot, Humid Climate." Journal of Applied Physiology 19: 607-612.

Wyndham, C.H. 1966. "Southern African Ethnic Adaptation to Temperature and Exercise." 201-244. In P.T. Baker and J.S. Weiner (eds.), The Biology of Human Adaptability, Clarendon Press, Oxford, United Kingdom.

Yanagawa, H., Hara, N., Hashimoto, T., Yokoyama, H., and Tachibana, K. 1981. "Geographic Pattern of Tuberculosis and Related Factors in

Japan." Social Science and Medicine 15D: 141-148. (Temperature, humidity, sunshine possibly correlated with occurence).

Yasuno, M., Rajagopalan, P.K., Russel, S., and Labrecque, G.C. 1973. "Influence of Seasonal Changes in Climate on Dispersal of Culex pipiens fatigans - A Study of Villages in Delhi Union Territory, India." Bulletin of the World Health Organization 48: 317-321. (Mosquitos).

Zahar, A.R. 1974. "Review of the Ecology of Malaria Vectors in the WHO Eastern Mediterranean Region." Bulletin of the World Health Organization 50: 427-440.

This report was done with support from the Department of Energy. Any conclusions or opinions expressed in this report represent solely those of the author(s) and not necessarily those of The Regents of the University of California, the Lawrence Berkeley Laboratory or the Department of Energy.

Reference to a company or product name does not imply approval or recommendation of the product by the University of California or the U.S. Department of Energy to the exclusion of others that may be suitable.

LAWRENCE BERKELEY LABORATORY
TECHNICAL INFORMATION DEPARTMENT
UNIVERSITY OF CALIFORNIA
BERKELEY, CALIFORNIA 94720