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CHARACTERIZATION OF INFORMATION REQUIREMENTS FOR STUDIES OF CO2 EFFECTS: WATER RESOURCES, AGRICULTURE, FISHERIES, FORESTS AND HUMAN HEALTH

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7. HUMAN HEALTH: ANALYSIS OF CLIMATE RELATED TO HEALTH

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7.1 INTRODUCTION

This chapter is to determine the information and data needed to study future impacts of elevated carbon dioxide (CO₂) on human health. An increase in atmospheric CO₂ is predicted to have several direct or indirect effects on human health, including 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 CO₂), and possibly slight changes in human biochemistry and physiology (due to breathing air with elevated concentrations of CO₂).

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. Chapter 2 of this volume briefly describes the expected vegetation effects, and the *Direct Effects of Increasing Carbon Dioxide on Vegetation* (Strain and Cure 1985) has more detail regarding current knowledge in this field. The chapters on water resources, agriculture, and forest (3, 4, and 6 of this volume, respectively) point out how changes in vegetation may affect these issues.

Changes in ocean chemistry may affect fisheries that in turn may affect nutrition, especially in some of the developing regions of the world. Information on the carbon cycle is described briefly by Blasing in Chapter 2 of this volume. More detail will be found in *Atmospheric Carbon Dioxide and the Global Carbon Cycle* (Trabalka 1985). Potential effects on fisheries are described briefly in Chapter 5 of this volume.

It has been suggested that breathing elevated concentrations of CO₂ may change human physiology and biochemistry even though if the atmospheric CO₂ concentration were quadrupled (to approximately 1200 parts per million [ppm]), it would still be only about 1/40th of the concentration of CO₂ in the air expired from the lungs (approximately 5% or 50,000 ppm). Humans have been exposed to much higher concentrations of CO₂ than those expected in the atmosphere with no persistent deleterious effects. However, these exposures were intermittent or were for periods of 6 months or less. Two reviews of this literature (Bland et al. 1982; U.S. Department of Energy [U.S. DOE] 1982) indicate that, if there is an effect in breathing very low but elevated concentrations of CO₂ over prolonged periods, it is probably extremely small. In addition, the exposure limit to CO₂ concentration recommended by the National Institute of Occupational Safety and Health (NIOSH) is 10,000 ppm for up to 10 h/d (U.S. Department of Health and Human Services 1985). Thus, this possible effect of elevated CO₂ will not be discussed further in this document although eventually long-term, low-level laboratory studies may be needed to determine whether there is an effect.

This chapter, therefore, concentrates on the effects of climate change on humans. Climate and particularly weather elements influence human health because some diseases are quite prevalent in some climates but are either less prevalent or absent in others. Increased atmospheric CO₂ is expected to raise the average global temperature by about 1.5deg.-4.5deg.C (MacCracken and Luther 1985a, 1985b). In temperate zones this increase is expected to be about equivalent to the global average, whereas increases in tropical zones are expected to be smaller and increases in the polar areas are expected to be greater. Thus, the boundaries of the tropics may extend into the present semitropical regions, 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 CO₂-induced climate change on human health; we can only point to some of the known influences of climate, seasons, and weather on human health and specify the data and information needed, both from the CO₂ research program and from human health research, to eventually assess the effects of CO₂-induced climate change on human health.

It must be emphasized that nonmeteorological 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, probably continue to do so under a CO₂-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, although climate provides the underlying environment that determines the potential for the prevalence of some diseases in specific climates, human circumstances, attitudes, and interventions (or lack

thereof) can, in many cases, modify the prevalence of these diseases. For example, the socioeconomic status of regions and individuals, cultural practices, quality of health care and education, 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.

Pertinent information in the literature is limited in several ways. First, it would be desirable, for purposes of this study, to know how much 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. Therefore, most studies designed to measure the health of populations use mortality statistics. These studies state cause of death and where and when the person died; thus, these data can be matched with environmental conditions of the time, and inferences can be made on 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. There is little knowledge regarding whether climate or the prevailing weather conditions are synergistic with other factors in instigating or in determining the rate of progression of some diseases.

Second, in some cases, the literature referred to in this report is relatively old. This is a consequence of the fluctuations of interest in and funding for different areas of research. In particular fields, little research has been done in recent years; however, much of the older literature is creditable research and relevant to this study. In addition, there are many other studies that would reinforce the literature quoted that have not been included for the sake of conciseness.

Third, until the CO₂ issue arose, there was no particular reason to study the effects that regional climate change might have on human health 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 chapter will describe some of the ways that climate and weather influence human health, some of the modifying factors, and the information and data needed to initiate assessment of the possible impacts of elevated atmospheric CO₂ on human health. [Figure 7.1](#) indicates pathways by which climate change may modify human health.

7.2 CLIMATE AND WEATHER

7.2.1 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 indirectly through effects on disease-causing organisms or their vectors. Although the effects of variation of only one weather element may be examined in a particular study, that element does not act independently of other elements, for example, changes in humidity modify the effects of temperature.

Extreme temperatures endanger human health. The acute effects of excessive cold are frostbite and, for prolonged exposure, death from lowered 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 diseases, especially the elderly, are very susceptible to aggravation of the disease state from both excessively cold and excessively hot weather. Temperatures in tropical, subtropical, and warmer temperate zones are ideal for the survival and propagation of causative agents for some bacterial, viral, and parasitic diseases. Temperature also affects human health 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 is also affected by humidity. Certain levels of humidity are ideally suited to the survival and reproduction of pathogenic bacteria, viruses, parasites, and their vectors.

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 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 discomfort with symptoms such as headaches. There is some evidence that 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.

7.2.2 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) responses and ameliorative actions. Physiological changes begin within minutes of an abrupt change in temperature, but it takes time to become completely acclimated to a change. For example, changes in types of clothing can be a rapid response to climate stress; however, architectural changes in housing, particularly in existing structures, take long periods of time. New strains of plants can be developed that 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 will be difficult. If there is gradual change, over 25-50 years, many changes can occur, either passively or actively, to offset possible detrimental effects.

7.2.3 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 normal weather may be either beneficial or detrimental to human health. The extent and direction of changes in variability under a CO₂-induced climate change currently are not known, but this information will be very important in determining the effects of climate change on human health.

7.3 BASIC MECHANISMS IN HUMANS

7.3.1 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 physiological 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 a constant range (about 37deg.-37.5deg.C). These mechanisms react to changes in the surface body temperature. The surface temperature of the body changes, for example, as ambient temperature changes, or changes as we move in and out of areas with different temperatures, humidities, and air movements. Physical activity and exposure to solar radiation also affect surface body temperature. 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 important in determining the efficiency of heat loss in this manner. Air movement affects convection, and both air movement and humidity affect evaporation.

When the heat content of the body increases or decreases, the thermoregulatory mechanisms become active. These mechanisms involve interactions between the sweat glands and the nervous, hormonal, cardiovascular, and respiratory systems and are collectively termed the thermoregulatory system. In hot weather, the sweat glands become active 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 accelerate heat loss, 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 thermoregulation mechanism 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 adjust and acclimate to the new temperature fairly rapidly and adequately. After 5-10 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 equilibrium seems to be established (Lind 1964; Lee 1968). 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 or cooled chambers or on people who have moved (suddenly been exposed) to hot and cold climates. Research 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 CO₂-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 damage 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 CO₂-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 people, particularly the elderly and those who have diseased circulatory systems (see Section 7.4.2).

There may be many subtle differences between people living in different climates. For example, there are seasonal variations in conception that differ with climate and that could be considered to be physiological adaptation. In Chile, conception has been related to temperature, with the highest temperature coinciding with the peak of conception, except in the northern and southern extremes of that country, where 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 maximum conception rates occur at a mean monthly temperature of 14deg.-16deg.C and minimum conception rates occur at 23deg.C. In warm, temperate climates the maximum conception rate is at 13.6deg.C, and the minimum is at 23deg.C; in the tropics, the maximum is 26deg.C and the minimum is 28deg.C. He states that humidity in the hotter regions (above 25deg.C) is an important factor in depressing the conception rate. He believes that the finding of different conception rates at different temperatures in different climates is *prima facie* evidence of reproductive adaptation to environmental temperatures. Holiday seasons and other cultural activities probably have 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, although 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. A study of human sperm (Tjoa et al. 1982) found that, in Houston, Texas, sperm counts were lowest in the summer despite widespread availability of air-conditioning. Becker (1981) speculates that fecundity of women may change seasonally. Bernard et al. (1978) among others speculate that intercourse may be less frequent in uncomfortably hot weather. Shimura et al. (1981) hypothesize that seasonal diseases may have an influence on conception. However, 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.

Active measures are taken by humans to minimize thermal stress. These include heating and cooling indoor environments, modifying clothing and food type and intake, and modifying physical activity.

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 that specifically adapt races to extreme climates, although cultural practices (e.g., types of clothing, housing, food) mitigate the effects (Newman 1975b). Thus, it would not be expected that genetic selection, over a number of generations, would assist in physiologically adapting humans to a CO₂-induced climate warming.

7.3.2 Physiology and Biochemistry

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 homeostatic (blood-clotting) factors of the blood seem to vary seasonally (Bull et al. 1979). Seasonal variations have been found in-body weight (Billewicz 1967). In a London study, blood pressure was found to be highest in April and May, and lowest in September (Rose 1961). In some studies, 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 changes in biochemical and physiological seasonal variations to the seasonality of mortality in some diseases, the relationships are far from clear, and the effects of changes in climate are uncertain.

7.3.3 Birth Defects

Seasonal incidence of certain congenital malformations has been found (McKeown and Record 1951; Wehrung and Hay 1970; Cohen 1971); whereas in other malformations, seasonality has not been found (Slater et al. 1964). In one study the United States was divided into four climate zones, and differences among 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 occurrence rate 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 diseases have all been suggested as causes of the seasonal variations in these malformations. Regarding infections, however, McKeown and Record (1951) found no record of a specific fever in mothers during pregnancy relating to birth defects.

The birth months of people later diagnosed as 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 that in Monroe County, New York, there was an association between season of birth and risk of schizophrenia. 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.

It appears that climatological factors may have some, either direct or indirect, influence on birth defects; however, the precise relationships are very difficult to characterize, and the effects of a CO₂-induced climate change would be very difficult to predict considering the current lack of knowledge about the primary causative agents.

7.4 MORTALITY AND MORBIDITY RELATED TO CLIMATE/WEATHER

7.4.1 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 7.2](#), 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 (hygiene, medical care, nutrition, central heating, housing, etc.). The prominent summer peaks in the 1890-1930 curves for Japan were principally due 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 climate effects, but these may have been important factors in the cause of death. A warm climate may be more favorable for the propagation of airborne and waterborne communicable diseases. In areas where such diseases are prevalent, the mortality rate among the young is high, which leaves a smaller elderly population 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. Infant mortality 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.

7.4.1.1 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, whereas deaths from asthma, cancer, and leukemia were related to temperature the least. Rosenwaike (1966)

found in the United States, for the period 1951-1960, 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, mortality from all causes in the temperate and subtropical zones of developed countries peaks in winter when temperatures are coldest and drops to its lowest levels when temperatures reach about 21deg.-27deg.C. However, if temperature goes much above 27[[ring]]C, there may be upsurges in mortality. A generalized curve depicting this situation is shown in [Figure 7.3](#).

7.4.1.2 Mortality From Heart Disease

Although this situation seems to be reversing somewhat, mortality from heart disease increased over several past 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 to illustrate the knowledge and the questions that are emerging.

Ambient temperature over a fairly wide range (below about 24deg.C) and in several midlatitude 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 -1deg. to 21deg.C (30deg.-70deg.F), Rose obtained a correlation coefficient of -0.95 between the logarithm of the monthly mortality index and temperature. West and Lowe (1976) found that in London, there was a 2.5% increase in mortality for each 1deg.C drop in mean monthly temperature when temperatures were between 2.3deg. and 17.9deg.C. Dunnigan also found a winter peak for heart disease patients admitted to the hospital but discharged alive. The change in the rate of mortality from heart disease per degree of temperature change appears to be different in different climates. [Figure 7.4](#), from Anderson and Le Riche (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 (which may be higher in one country than in another) may be responsible for the differences among countries. However, other investigators (Rogot 1974; Bainton et al. 1977) 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 Pittsburgh, Pennsylvania (temperate climate), and Birmingham, Alabama (subtropical climate), and related mortality to meteorological variables. He found deaths in Pittsburgh from ischemic heart disease to be highly correlated with weather (directly correlated with temperature change), but weather was poorly correlated with deaths from cerebrovascular diseases. The reverse was true in Birmingham. The correlation of mortality with weather variables was significant in both cities, but it was lower in Birmingham.

There was a direct relationship of interdiurnal change in barometric pressure to mortality (for white males, for people over 70, and for death from ischemic 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 was the probability for peripheral arterial embolism to occur; this observation did not change with seasons.

Even though heart disease mortality gradually declines as the temperature increases, it increases rather abruptly during periods of very hot temperature. The exact temperature at which this abrupt increase occurs appears to differ a few degrees in different regions. [Figure 7.5](#), from Rogot and Padgett (1976), illustrates this for a number of cities in the United States. The cities on the left are in areas that 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 these variables show a relationship to mortality, it should be noted that the effects are due to synergistic actions with other variables, especially temperature and wind; that is, it is the heat load on the body that is important.

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 7.4](#)), 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 mortality from heart disease 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 mortality from heart disease in Minneapolis-St. Paul, Minnesota (Baker-Blocker 1982), Chicago, Illinois (Rogot 1974), selected U.S. metropolitan areas (Rogot and Padgett 1976), and Toronto and Ontario in Canada (Anderson and Rochard 1979). It is probable that part of the effects of snowfall is 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.

Windspeed in January was found to have a positive correlation with the rate of mortality in 143 U.S. metropolitan areas (Campbell and Beets 1979). However, from studies carried out in England and Wales (Bull 1973) it was concluded that the relationship between windspeed and mortality 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 North-Central and Northeastern States in the United States (Driscoll 1971). In three different areas of the world--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 that follow cold fronts.

7.4.1.3 Mortality From Cerebrovascular Diseases

Cerebrovascular diseases, including strokes, also have been related to meteorological factors. [Figure 7.6](#) shows the relationship of stroke mortality to temperature (from the study of Rogot and Padgett [1976]). The lowest mortality occurred between 15.6deg. and 26.6deg.C (depending on the city); then there was a sharp rise as the temperature increased. These data are presented as average daily deaths; therefore, although the shape of the curves can be compared, the rate of death in cities, with different climates cannot be compared. Bull (1973) and Bull and Morton (1975, 1978) have obtained essentially the same results. For England and Wales, from -10deg. to +20deg.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 sharply, particularly in the older age groups. The investigators related mortality to temperatures during the 3 to 4 days before death.

Rogot and Padgett (1976) found that during periods of snow there was 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 in London, Tokyo, and New York City for 1960-1964 to temperature. These data are shown in [Figure 7.7](#). There is a negative relationship between stroke and temperature in all three cities. However, New York, with the lowest temperatures of the three cities, also had the least steep slope. The authors attributed this to better central heating in New York, which probably better protected the susceptible people from extreme weather.

7.4.1.4 Mortality and Morbidity From Respiratory Diseases

Rosenwaike (1966), using data from the United States, found the highest 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 -5deg. to about 20deg.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 were highest at low temperatures (<4deg.C) and gradually decreased up to temperatures of about 23deg.-24deg.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 people, occur in the winter both in 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, Louisiana, 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 preceded, 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 astmatogens and that the high pressure system tends to result in stagnant rain-free conditions that 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 humidity may instigate asthma attacks.

7.4.1.5 Mortality From Cancer

Mortality from cancer (data pooled from several types) in England and Wales (Bull and Morton 1978), Memphis, Tennessee (Rogot and Blackwelder 1970), and the entire United States (Rosenwaike 1966) was found to have very little association with temperature. The little association present may be due to temperature stress on people who are already terminally ill.

Regarding the onset of cancer, there are two types of cancer in which meteorological influence seems apparent. 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 CO₂-induced climate change unless there is a large change in cloud cover), but may also have some relationship to female hormones because, 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 differ according to culture. The male/female ratios for melanoma differ with climate, 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 that varies somewhat 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 temperature is always above 15deg.C, and the annual rainfall is more than 50 cm. The Epstein-Barr virus is the etiological agent; however, the virus is not limited to the climate where the lymphoma is found. It was eventually found that the climatological factors favor malaria and that malaria depresses the immune system. The depressed immune system allowed the virus to initiate lymphoma. The tumor is common only in those areas where malaria is highly endemic. Thus, in this case, if a CO₂-induced climate change causes new areas of the world to have temperatures continuously above 15deg.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 well controlled.

7.4.1.6 Fetal and Infant Mortality

Perinatal (late fetal and less than 7 days of age) mortality and preterm delivery have been shown in the United States 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 because of infections that have a seasonal distribution (Slatis and DeCloux 1967; Janerich et al. 1971; Keller and Nugent 1983). However, protection of infants against exposure to extreme temperatures is very important. 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) and may be related to seasonally occurring infections.

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 early gestation in the fall and early winter.

7.4.1.7 Mortality From Other Causes

Deaths in the United States of people 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 preceding death, and it might be expected that any stress (including low temperatures) would precipitate death in people already very ill. However, the onset of insulin-dependent 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 CO₂-induced climate warming may prolong the lives of people with some of these diseases, but the effect would probably be small.

In the United States suicides tend to peak between March and May, and homicides have two peaks: July-September and December. Motor vehicle accidents are lower in the first half of the year (January-June) than in the last half, with the highest rate being in October through December. Nonmotor 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 CO₂-induced climate changes.

Thus, seasonal changes in weather variables have an important role in morbidity and mortality. In less developed countries there still is 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 weather elements (see Sections 7.6

and 7.7). In the more highly developed countries, where infectious and some parasitic diseases have been largely controlled, the effects of cold winters and hot summers are primarily in people with deficient thermoregulatory systems and, secondarily, in people who have undue exposure to, or activity in, extreme temperatures and in which the thermoregulatory system, although it may be functioning properly, is overwhelmed. It is uncertain how much the underlying climate is related to longterm (possibly damaging) stress on the thermoregulatory system, or how it relates to physiologically adapting people to better withstand thermal stress.

A first assumption might be that, if CO₂-induced climate change resulted in warmer winters, in currently temperate and cold zones, the life of some people might be prolonged. However, hotter summers might, to some extent, offset this. Hotter summers, in subtropical 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 regional and seasonal extent of changes in the climate and the weather elements, and the variability of weather are needed to predict the effects of CO₂-induced climate change on people with organic diseases.

7.4.2 Heat Waves

There is currently no information about possible changes in the variability of weather resulting from a CO₂-induced climate change. If variability decreases, even though the average temperature may be higher, there should be fewer heat waves. 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, these studies may be relevant to the effects of a CO₂-induced climate change.

Death rates can increase markedly as a result of heat waves, and the peaks correlate with maximum daily temperature 1-2 days before death; that is, there is a 1-2 day lag between the hottest temperatures and the peak in death rate (Bridger and Helfand 1968; Oechsli and Buechley 1970; Ellis 1972; Ellis et al. 1975). [Figure 7.8](#) 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 (nonheat 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 7.1](#) 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 most important meteorological factor is an increase in mean daily temperature of several degrees above normal for the region lasting 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 contribute to the effect on humans because they influence body temperature, and thus to the burden on the thermoregulatory system.

Illness (heat stroke, heat exhaustion, etc.) may occur in healthy people 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 people already ill, in particular those with circulatory problems, are most at risk during excessive heat (Schuman et al. 1964; Oechsli and Buechley 1970; Bridger et al. 1976; Jones et al. 1982). [Tables 7.2](#) and [7.3](#) illustrate the age influence on the death rate. Older people are more at risk because the main stress is on the circulatory system and many older people

have heart and vascular diseases. Because of circulatory problems their thermoregulatory systems are not as efficient as those of younger people (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 ischemic heart disease (Ellis and Nelson 1978) and cerebrovascular lesions (Schuman et al. 1964). In some regions or in some years the percentage rise in death due to heart disease was found to be higher (Ellis et al. 1975); in other regions or years cerebrovascular diseases accounted for more deaths (Bridger and Helfand 1968; Ellis and Nelson 1978). 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 people 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 7.8](#)) 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 people 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.

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

Within a region during a particular heat wave, investigators have examined various factors other than climate that 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). Several factors are probably involved here (see below), but the most predominant of these is thought to be urbanization, which has modified the climate (Clarke 1972a). There also may be relatively fewer air-conditioners or coolers per urban residence than in suburban residences. Daytime urban-rural temperature differences are small. However, the urban core retains heat at night because of the lack of air movement, higher near-surface air temperatures, and greater heat load. After sunset, rural surfaces cool more rapidly than do urban surfaces, because soil and grass have lower heat conductivity and storage capacity than do brick and concrete surfaces. Large urban structures also lose heat more slowly 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.

The racial makeup of people who died in heat waves has been studied with apparent contradictory results. In some cases, white people seemed to have higher excess mortality (Bridger et al. 1976, see [Table 7.2](#)); whereas in other cases mortality in nonwhites was higher (Henschel et al. 1969; Schuman 1972), and in yet others, there apparently were no differences (Schuman et al. 1964; Bridger and Helfand 1968). These apparent contradictions may have resulted, for example, from methodological or socioeconomic differences.

Socioeconomic status has been found to be a factor in the death rate in heat waves. People in the lower socioeconomic status generally were 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, possibly 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 regarding 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 (Henschel et al. 1969; Oechsli and Buechley 1970; 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, although significant and negative, was small. There is also some indication that residence in air-conditioned premises may interfere with acclimation to heat (Marmor 1975) and thus make people more vulnerable to heat when they are in non-air-conditioned environments. For example, 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 7.8](#), which were collected before air-conditioning had an appreciable effect, indicate that in the second heat wave of one summer the death rate was lower than in the first heat wave; this partly may have been due to the most vulnerable people being eliminated 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 1 year in both the south and the north-central regions the weekly average temperature was 35.6deg.C (4deg.C above normal for the south and 7.6deg.C above normal for the north-central region). During this period the death rate in the south increased by only 12%; whereas in the north-central region it 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 that was associated with the marked increase in weekly mortality and that acclimation had an influence.

Unfortunately, literature on heat waves is inadequate in relating the precise contributions of individual weather and climate variables to the increase in morbidity and mortality. First, weather conditions, that is, the length and intensity of the heat wave, the suddenness of its onset, the degree to which the temperature deviates from the normal for the season and the 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; the data available to and the methodology used by investigators also can differ. Until the CO₂ issue arose, there was no reason to investigate the effects of a changing climate in a particular region; therefore, with the exception of temperature, climate and weather variables were not usually considered in the studies.

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. However, several indices have been used as predictors of heat stress. Quayle and Doehring (1981) discuss several of the indices 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 37deg.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 terrestrial 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. However, presumably the index should be a means for warning that people with chronic disease and the elderly might be at high risk and should take precautions to reduce exposure and limit physical activity.

Thus, heat wave studies point out that abrupt increases in temperature can accelerate death in many people; however, there are many uncertainties about 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 buildup within a season have? With the exception of the inner city and the socioeconomic 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 differ in a CO₂-induced climate change? However, perhaps the most important question is whether the variability in weather will increase or decrease; thus, whether the frequency of abrupt changes in temperature will change.

7.5 SPORADIC AND 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 forage, consequently affecting nutrition and economics. Failure to obtain adequate nutrition, either because nutritionally adequate foods are in short supply or are too expensive, increases susceptibility to disease and early death.

Floods also may disturb nutritional status by destroying crops. In addition, in many cases homes are lost, leaving people exposed to the elements or crowded into temporary shelter. These situations increase the likelihood of disease and epidemics. Floods may contaminate water supplies, adding to the possibility of an increase in the occurrence of disease.

Tornadoes and hurricanes may kill and maim people, as well as destroy crops and housing, and may also increase the probability of the spread of disease.

To some extent, droughts are covered in Chapter 4 (this volume) and are referred to in this chapter with respect to the relationship of nutrition to disease. However, the effects of floods, tornadoes, and hurricanes 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 CO₂-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 extreme events in a particular region.

7.6 AIRBORNE MATERIALS RELATED TO HUMAN HEALTH

Many airborne bacteria, viruses, fungi, and allergens that cause human illnesses are distributed in the atmosphere. Atmospheric conditions, especially temperature, precipitation, humidity, and wind, will affect the multiplication, dispersal, and survival of these organisms. Thus, a CO₂-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, 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 the numbers and types 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 can serve 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 (Lidwell 1964; Akers et al. 1979).

The transport of biological materials suspended in air is the same for 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 (still) atmosphere allows buildup of pollutants. The degree of stability depends on the rate of temperature change with height (Chatigny and Dimmick 1979).

7.6.1 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/m³ at an altitude of 3127 m. However, it appears that no major bacterial epidemics in humans are propagated by spread in the atmosphere. 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 bacteria are dispersed. Air temperature does not appear to be the usual limiting factor because most organisms can live at extreme temperatures (between -20deg. and 48deg.C); however, the effect of temperature is modified by relative humidity. The lethal effects of ultraviolet radiation are well known, but 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 next.

Hyslop (1978) aerosolized various bacteria to test their viability in air. Tests on a mycoplasma at various relative humidities showed that survival was shortest at 80% relative humidity. *Escherichia coli*, at 20deg.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 28deg.C and 50% relative humidity. A significant amount of *Salmonella typhimurium* remained viable for 24 hours at 21deg.-28deg.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 air-water 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 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 who are in close 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 fanshaped, 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 that 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 1-4 bacteria/ml. At the time the snow fell, there was a stable high-pressure system over Russia and a low-pressure system west of Scandinavia, 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 that 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 may contribute to the spread of diseases over long distances but are not as effective in causing diseases as are bacteria found in high concentrations in indoor environments. This is probably due to factors in the outdoor atmosphere (e.g., ultraviolet light, air pollutants) that kill the bacteria, as well as the great dilution caused by air turbulence.

7.6.2 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 poliomyelitis 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 these influences.

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, humidity, temperature control, and so forth.

7.6.3 Fungi

Fungal lung infections are relatively common occurrences in certain areas of the United States. It has been estimated (Furcolow 1965) that annually 500,000 people 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 occurrence are not available. However, this disease, along with other fungal diseases, probably accounts for much time lost from 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), in investigating *Histoplasma capsulatum*, found prevalence of this fungus in an area of the United States (Midcentral and Mideastern States) where annual precipitation is 76 to 127 cm and the average summer temperature is between 21deg. and 27deg.C. They think that the direction of the prevailing winds tends to limit the fungus to this area. However, Hasenclever (1979) believes that it is distributed worldwide 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 foci are disturbed (digging, plowing, etc.), epidemics occur in areas downwind.

Coccidioides immitis, another soil fungus, 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 climatic factors to this prevalence. The fungus apparently thrives in climates where summer temperatures are hot enough to sterilize the surface soil, thereby eliminating competing plants (July ambient air temperatures from 26deg. to about 32deg.C), and where there is very little freezing at ground level (January ambient air temperatures range from about 4deg. to 12deg.C). Rainfall is also an important factor, with 13-50 cm being ideal. Temperature and rainfall outside these ranges are less favorable for the fungus. Hugenholtz (1957) also found that winds that raise dust storms, mainly in the spring and fall (especially after a period of precipitation), increase the incidence of infection.

Climate changes could either extend or limit 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.

7.6.4 Pollen

Pollen from plants are found worldwide and are one of the most common causes of allergies in humans. Pollen grains are minute and are thus easily airborne and dispersed over long distances. Meteorological conditions may limit or expand their dispersal and, in some cases, their production.

Hay fever afflicts approximately 6% of the U.S. population, being most prevalent among 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 most common causes of hay fever. 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 hay fever. The pollen season for ragweed is August-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.

7.7. SEASONAL DISEASES CAUSED BY MICROORGANISMS

7.7.1 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 the survival of the causative pathogens; (2) the depression of the 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 because of the reduced availability of fresh vegetables. It has also been suggested that the 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 have been sought, it has not been found (Hope-Simpson 1979). However, Tromp (1980) stated that influenza virus and grampositive 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 more 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 incidences of these diseases are unknown, although trends may be fairly well established. With CO₂-induced global warming, it would generally be assumed that summer diseases would increase and winter diseases would decrease. However, other meteorological variables, for example, humidity and rainfall, also influence the prevalence of these diseases. CO₂-induced changes in these meteorological parameters, on a regional and seasonal basis, are currently uncertain. Thus, the effects of a CO₂-induced climate change on the prevalence of these diseases are also uncertain.

7.7.2 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. A few examples of the relationship of climate to these diseases follow.

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 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 15deg.-27deg.C (average temperature for the hottest months). Rainfall and humidity also have been implicated in influencing the incidence of cholera.

The group of *Salmonella* organisms (causative agents of typhoid, paratyphoid, 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 CO₂-induced climate change.

7.7.3 Vector-Borne Viral and Bacterial Diseases

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

Although large epidemics of plague have not occurred 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. Plague epidemics have occurred when temperatures have been about 19deg.-26deg.C with high relative humidity; for example, during the plague epidemic in Brazil, the relative humidity was between 66 and 83% (Pollitzer 1954). When the temperature increases above about 26deg.C, the incidence of plague decreases, apparently because fleas are susceptible to desiccation. A study conducted in Vietnam (Olson 1969) showed that the incidence of plague was inversely related to the amount of rainfall. For unknown reasons, as the amount of rainfall increases, the flea population decreases; it is speculated that the flea drowns or gets caught in the mud in rat holes.

There are several different strains of viruses that cause encephalitis and are transmitted to humans by the culicine mosquito. Birds are a reservoir for the virus. The virus strain 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 the epidemics. Rainfall and flooding provide breeding ponds for mosquitoes. Incubation periods for the virus in the mosquitoes are much longer at 20deg. than at 32deg.C. Thus, as the temperature increases, when the viruses and mosquitoes are present, the probability of epidemics becomes greater. At least one mosquito vector stops biting when the temperature drops below 15deg.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 increased above about 26deg.C with low humidity; 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.

7.7.4 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, improved sanitary conditions, and the education of the populace assist the control of vectors and parasites. However, there is a large climate influence that 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, decreased work ability, and greater susceptibility to other diseases.

7.7.4.1 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

where malaria is endemic and where epidemics occasionally occur. 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); there is a temporary increase in abortions, and a reduction in conceptions and birth rates. The plasmodia (parasite) develops in *Anopheles* mosquitoes and is transmitted to humans by the mosquito bite. There are some 50 species of *Anopheles* that are important vectors. One of the problems in controlling mosquitoes is that different species have different susceptibilities to the chemicals used for control, and each has a different ecological niche. 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. A crude generalization (Learmonth 1977) regarding temperature and humidity is that mosquitoes breed and are active at temperatures above 16deg.C, and a relative humidity of about 60% is ideal. Temperatures in excess of about 35deg.C and average relative humidity of less than 25% will cause either death or dormancy. Also, the rate of development of the parasite (sporogony) in the mosquito depends on temperature; for example, for *P. falciparum*, it takes 9 days at 30deg.C, 11 days at 24deg.C, and 20 days at 20deg.C (Garnham, 1964).

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

7.7.4.2 Parasites Requiring Intermediate Hosts

There was an estimate of about 200 million cases, worldwide, of schistosomiasis in 1975 (Markell and Voge 1981). The life cycle of the parasite is spent partly in humans, partly in water, and partly 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 that 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. Environmental factors, which 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 9deg.C snails are not usually infected by the parasite. When the temperature rises above about 35deg.-39deg.C the snails die. The temperature most favorable for the water/snail cycle of the parasite is 26deg.-28deg.C (Kendall 1964; Purnell 1966; Anderson and May 1979). Heavy rainfall can wash the snails from their ecological niches, but, generally, rain is advantageous for snails 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, and water impoundments (built for health and economic reasons) are ideal breeding and living areas for the schistosomes during the water and snail phases of the life cycle. Despite attempts to control it, schistosomiasis continues to spread worldwide (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, changes in temperature and precipitation. Health education of the population in areas contaminated with the parasite could also influence its prevalence.

7.7.4.3 Parasites That Do Not Need Intermediate Hosts or Vectors

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 the human feces to soil where, with favorable conditions, they hatch, and the larvae develop and transform to the infective stage. Given the opportunity, they then penetrate human skin. They may cause diarrhea, anemia, and occasionally pneumonitis, if the infecting dose is large. They are generally confined to those parts of the Northern Hemisphere south of the 36th parallel, and north of the 30th parallel in the Southern Hemisphere

(Markell and Voge 1981). Eggs and larvae are subject to freezing and desiccation and are very sensitive to climate. The optimal temperature for eggs to hatch is from 27deg. to 32deg.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, CO₂-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 migration of humans and new water projects are a consequence of CO₂-induced climate changes, the control of these diseases may become greatly complicated.

7.8 NUTRITION

7.8.1 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 that 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 not as balanced nutritionally, then the nutrition of the regional population may suffer (see Chapters 4 and 5 of this volume for more discussion of the effects of climate change).

In addition to changes in food production, society's need for nutritional energy may increase under a CO₂-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 30deg.C (86deg.F) than are needed at lower temperatures.

7.8.2 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 CO₂-induced climate change on nutrition and disease prevalence may either help a particular situation or make it worse, depending on regional and seasonal changes and their effects on the diseases and on food production.

7.9 WATER

Water is essential to all forms of life, including that of humans. Precipitation is variable both seasonally and regionally. Regional changes in the amount and timing of precipitation, temperature, humidity, and wind influence the amount and quality of fresh water available for humans, thereby affecting the quality of life. See Callaway and Currie, Chapter 3 of this volume, for a discussion of the effects of climate change on water resources.

7.9.1 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 (see Chapters 4 and 5 of this volume for more details).

Forests and other nonmanaged vegetation are dependent on precipitation in much the same way as agriculture. Many forest products are needed for maintaining or improving the quality of human health; for example, shelters are vital for alleviating stress on the thermoregulation mechanisms and are helpful in protecting humans from harmful elements of the environment (see Chapter 6 of this volume for details of climate effects on forests).

The availability of water, which is essential for drinking, cooking, sanitation, and so forth is dependent on precipitation for groundwater recharging, as well as for maintaining streamflow and filling lakes and reservoirs. When these resources are not adequately replenished, human health and life are endangered.

7.9.2 Quality of Water

The quality of water is dependent largely on the quantity of precipitation. Groundwater, which has percolated through soil and rock and has had impurities filtered out, is usually the purest water for drinking, cooking, and sanitation. Frequently groundwater also 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 also are dependent on precipitation for replenishment and for the dilution of impurities accumulated from biological or industrial sources. In developed nations, surface water usually goes through purification processes before human use, 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, which 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 and sanitation practices as well as poverty 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 in precipitation will also be important; that is, if precipitation declines in a region and does so slowly, there may be time for adaptive measures such as construction of reservoirs, modification of methods of irrigation, and development of plants that are more drought tolerant. The effects of a CO₂-induced climate change on water resources will depend on regional and seasonal changes, which are currently uncertain.

7.10 SHELTER

Shelter is one of the ways in which humans protect themselves from extremes in temperature. Well-planned residences in tropical climates are built for maximum ventilation; whereas those in extremely cold climates are built for maximum insulation, and those in temperate climates are built 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 7.4.2) where there was a higher percentage of deaths in the midcity than in the suburban and rural areas (Henschel et al. 1969; Clarke 1972b).

CO₂-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 the potential for CO₂-induced climate change.

7.11 AIR POLLUTION

A CO₂-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 accumulate. Air pollutants that are harmful to human health usually are generated and are more concentrated in the atmosphere of industrial and metropolitan areas. Generally, areas most in danger from health-threatening pollutants are those in low-lying areas, that is, basins in hilly or mountainous areas or river valleys that 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 excess deaths occurred, there was fog combined with high pressure systems (Prindle 1964), which held the smoke and other air pollutants near the ground.

The effects of CO₂-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.

7.12 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 CO₂-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.

7.13 SUMMARY AND DATA AND RESEARCH NEEDS

A number of aspects of human health that are related to climate and weather variables may be affected by climate change. These include the relationship of climate and weather variables to the mortality rate from diseases such as heart, cerebrovascular and respiratory; birth defects and fetal and infant mortality; survival and geographic prevalence of disease-causing organisms such as bacteria, viruses, fungi, pollen, and parasites (and their vectors and hosts); nutrition; and shelter needs. Often research has been (and is being) performed to determine more precisely the relationship of meteorological variables to human health. For example, the knowledge gained in this research is being used in many cases in the control of parasites. 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 is possible because of factors such as lack of education, lack of funds, and 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 CO₂-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. [Table 7.4a, b, c](#) tabulates knowns and uncertainties regarding the climate relationship to human health. Data and research needs are discussed next.

7.13.1 Climate Data Needs

Many aspects of human health and welfare are affected by meteorological variables. To specify qualitatively and quantitatively the effects of a CO₂-induced climate change on human health, it is necessary to know the rate at which climate change will occur, regional and seasonal climate changes, and changes in the variability of weather. The state of current climate research is such that it is not possible to predict this information with any certainty. Until such time as definitive regional and seasonal changes are predictable, 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. For the climate needs of water resources, agriculture, fisheries, and forests related to human health, see Chapters 3, 4, 5 and 6 of this volume.

7.13.2 Carbon Cycle Data Needs

The CO₂ 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 report on vegetation effects (Strain and Cure 1985).

The direct effects of breathing increased levels of CO₂ 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 study further the direct effects of CO₂ on human health, information from carbon cycle research will be needed, including the rate at which the atmospheric CO₂ concentration will increase and the maximum expected concentration.

7.13.3 Medical and Biological Research

Determining the effects of a CO₂-induced climate change on human health, beneficial and detrimental, will depend on the magnitude of regional and seasonal changes in climate and changes in the variability of meteorological variables. Although the CO₂ Climate Program is making progress in this direction, apparently, it will be some time before this kind of information will be available. 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 CO₂-induced regional and seasonal climate changes. The prospect of a CO₂-induced climate change adds some urgency to solving these problems and finding ways to ameliorate them. High-priority research related to meteorological influences is outlined next.

7.13.3.1 Organic Diseases

Meteorological variables influence mortality and, to a largely unknown extent, influence morbidity from a number of organic diseases, such as heart and cerebrovascular diseases. Research should continue in this field 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 the rate of change of meteorological variables; and (4) studies of whether prevalence of diseases differs with climate and, if so, the extent to which meteorologic variables are responsible. The data used in these studies should be adjusted for factors such as age of population, socioeconomic status, sex, race, type of housing, place of residence (urban, suburban, rural), air-conditioning, and the availability of central heating. 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. Therefore, cooperation of the medical profession will be required to determine the approximate time of the onset of the diseases and the age of the patient at that time. 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 stresses of many kinds (especially temperature extremes) cause 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 diseases at earlier ages. However, there are factors other than meteorological that contribute to regional differences in death rate from these diseases. Some examples are different percentages among regions of a particular race, sex, age, socioeconomic class, or city dwellers. The contribution of these factors to the variance in death rates must be resolved before the influence of meteorological variables can be precisely determined.

7.13.3.2 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.

7.13.3.3 Birth Defects and Infant 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.

7.13.3.4 Bacteria, Viruses, Parasites, and Allergens

The meteorological factors involved in the survival and multiplication of various microorganisms that cause human diseases and the hosts and vectors of these microorganisms 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 current areas of endemicity and defining the meteorological variables, and other variables not currently well known, 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 may 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.

7.13.3.5 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 to this situation, 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.

7.13.3.6 Shelter and Workplace

City planners, architects, and engineers need to work closely with meteorologists on research to design new structures and their surroundings (e.g., green areas) 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 CO₂.

7.13.3.7 Recreational and Vacation Areas

Until regional and seasonal CO₂-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.

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

- 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.
- Cech, I., Smolensky, M., Lane, R., Halevy, B., and Samuelhoff, S. 1976. "Biometeorological Aspects of Short-Term Fluctuations of Cardiac Mortality in Jerusalem and Tel Aviv Studied by Lagged Cross-Covariance Analysis." *Israeli 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., Youngs, K., Smolensky, M.H., and Sargent, F. 1979a. "Day to Day and Seasonal Fluctuations of Urban Mortality in Houston, Texas." *International Journal of Biometeorology* 23:77-87.
- Cech, I., Smolensky, M.H., Lane, R., Nagata, H., Takahashi, Y., and Morimoto, T. 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, Strausburg, 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*:25-18.
- 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 Chile (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. 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."

- Garnham, P.C.C. 1964. "Factors Influencing the Development of Protozoa in Their Arthropodan 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* 53:1112-1143.
- Hajek, E.R., Gutierrez, J.R., and Espinosa, G. 1981. "Seasonality of Conception in Human Populations in Chile." *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, Strausburg, 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.
- Hughenoltz, 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, 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.
- Lee, A.R. 1968. "Human Adaptations to Arid Environments." 517-557. In G.W. Brown, Jr. (ed.), *Desert Biology*. Academic Press, 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 in 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 Guatemala." *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.
- May, J. 1958. *The Ecology of Human Disease*. MD Publications, 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., and Kito, H. 1963. "A Geographic Study of Seasonal Disease Calendar Models by Period and Country." *Papers in Meteorology and Geophysics* 14:109-118.
- 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.
- 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. 1975b. "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 Monograph Series Number 22. World Health Organization, 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.
- 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*

- Scrimshaw, N.S., Taylor, C.E., and Gordon, J.E. 1968. *Interaction of Nutrition and Infection*. World Health Organization, Monograph Series Number 57. World Health Organization, 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 Preventive 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/ER0238). 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.
- 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. E.B. Brown, C.D. Gull, C.B. Carlston (eds.), *Effects of CO2 on Mammalian Organisms*, Report of a Workshop, June 5-6, 1980, Bethesda, Maryland. (CONF. 8006249). Department of Energy, Washington, D.C. Available from NTIS, Springfield, Virginia.
- 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.
- World Health Organization (WHO). 1955. *International Classification of Diseases*, 7th Revision. WHO, Geneva, Switzerland.
- World Meteorological Organization (WMO). 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 (WMO). 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.