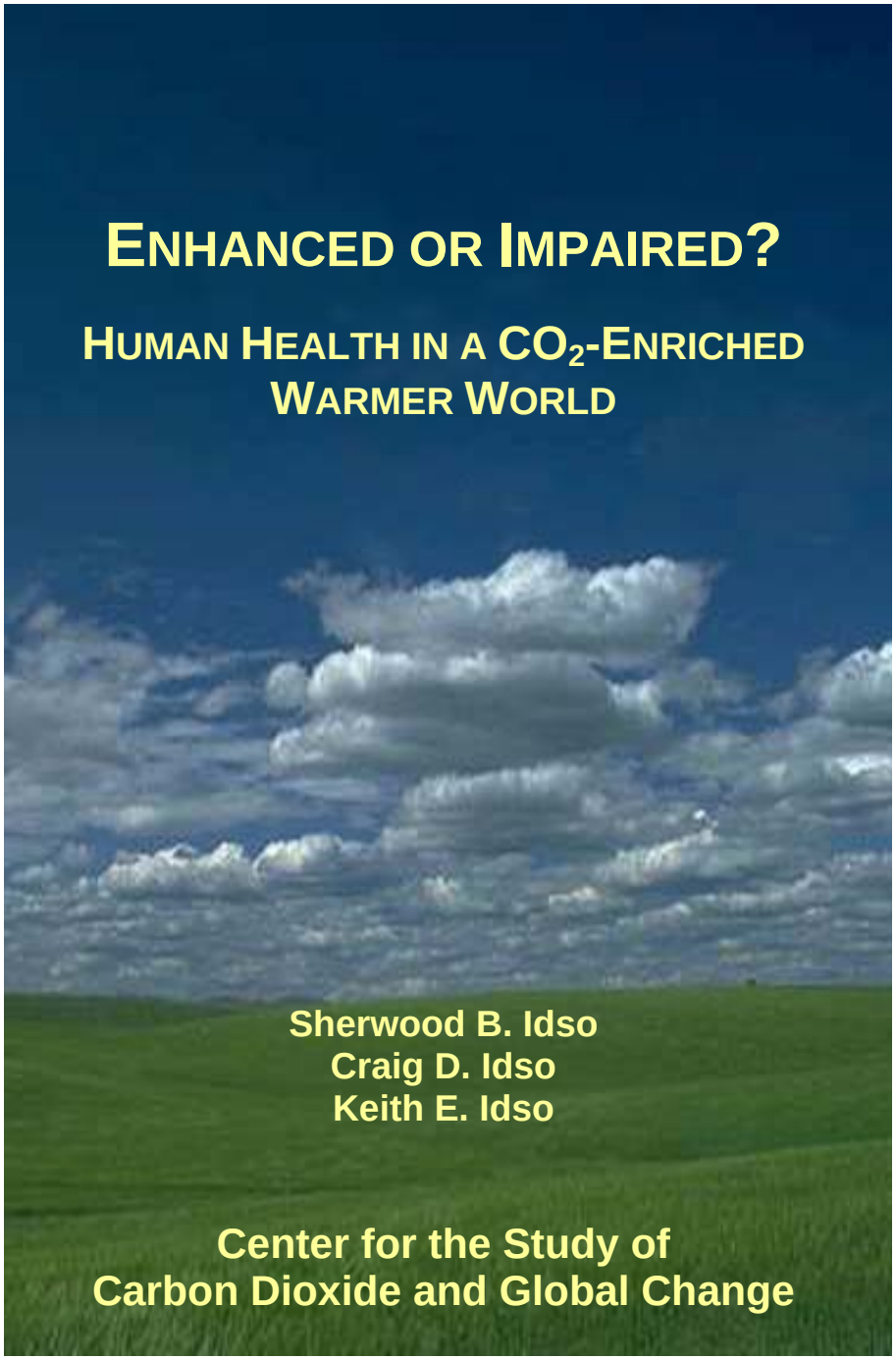

Enhanced or Impaired? Human Health in a CO_2-Enriched Warmer World

Sherwood B. Idso, Craig D. Idso and Keith E. Idso

Center for the Study of Carbon Dioxide and Global Change
P.O. Box 25697, Tempe, AZ 85285-5697, USA
www.co2science.org

About the Center

The Center for the Study of Carbon Dioxide and Global Change is a 501 (c)(3) non-profit science education organization created for the purpose of disseminating factual reports and sound commentary on new developments in the scientific quest to determine the climatic and biological consequences of the ongoing rise in the air's CO_2 content. It primarily meets this objective through the publication of *CO_2 Science Magazine*, a free online Internet publication that contains editorials on topics of current concern and mini-reviews of recently published peer-reviewed scientific journal articles, books and other educational materials (see www.co2science.org). In addition, the Center periodically publishes major reports on pressing scientific issues, such as the topic of this report. In all its endeavors, the Center attempts to separate reality from rhetoric in the emotionally-charged debate that swirls around the subject of carbon dioxide and global change.

TABLE OF CONTENTS

I. INTRODUCTION

Perhaps the best known thing about carbon dioxide or CO_2 is that it is a "greenhouse gas," possessing properties that endow it with the potential to enhance the atmosphere's *greenhouse effect* as its aerial concentration rises. A highly publicized consequence that is predicted to result from this phenomenon is *global warming*, which is typically characterized as undesirable by a cadre of climate alarmists. However, both the premise of catastrophic CO_2-induced global warming and its host of implied evils are untenable, as we continue to demonstrate in this second of our series of major reports on the subject.

In our first report (Idso *et al*., 2003), we evaluated -- and found wanting -- the climate-alarmist claim that CO_2-induced global warming will lead to a massive extinction of various species of plants and animals that are claimed to be unable to migrate poleward in latitude or upward in altitude fast enough to maintain a presence within the shifting climatic regimes to which they are currently adapted, as the planet warms at what is typically predicted to be an unprecedented rate. Here, we evaluate a second climate-alarmist scare story: the claim that CO_2-induced global warming will have numerous deleterious effects on human health.

The idea that CO_2-induced global warming is responsible for increases in a host of human maladies has become entrenched in popular culture. Hardly a heat wave passes, for example, but what climate alarmists are quick to blame global warming for any excess deaths that may have been associated with it. High temperature events are accused of increasing the number of cardiovascular-related deaths, of leading to enhanced respiratory problems, and of promoting the more rapid and widespread dissemination of a number of infectious diseases, such as malaria, dengue and yellow fever. If the truth be told, however, as we intend to do in this treatise, global warming would likely do just the *opposite* and actually *reduce* the number of lives lost to extreme thermal conditions, as many more people die from unseasonably cold temperatures than from excessive warmth.

We will also describe and evaluate a number of *non*-climatic effects of atmospheric CO_2 enrichment that impact human health. These are phenomena about which the world's climate alarmists say very little; for they produce *positive* effects that tend to *enhance* people's quality of life. Last of all, we will review the history of human lifespan and how it has changed over the past two centuries, during which time the air's CO_2 concentration and temperature both rose substantially and should therefore, according to climate-alarmist thought, have wrought a multitude of ills upon humanity.

II. TEMPERATURE-INDUCED MORTALITY

Which is more deadly … heat or cold? … rising temperatures or falling temperatures? The world's climate alarmists say it is warming that is to be avoided at all costs. Real-world data, however, suggest otherwise.

The positive health effects of heat have been well-documented over the past quarter-century. The early studies of Bull (1973) and Bull and Morton (1975a,b) in England and Wales, for example, demonstrated that even normal changes in temperature are typically associated with *inverse* changes in death rates, especially in older subjects. That is, when temperatures *rise*, death rates *fall*, while when temperatures *fall*, death rates *rise*. Also, Bull and Morton (1978) report "there is a close association between temperature and death rates from *most* diseases at *all* temperatures," and they say it is "very likely that changes in external temperature *cause* changes in death rates."

Another interesting finding of the study of Bull and Morton (1978) relates to *extremes* of heat and cold. They report that at the lower end of the temperature range, "there are more deaths the longer the 'run of days,' while at the higher end of the temperature range the reverse is true," i.e., "the longer the 'run' the fewer the deaths," suggesting that people adapt more readily to extreme heat than extreme cold. Among the various diseases that exhibit these relationships, they make particular note of "atherosclerotic diseases (strokes, ischemic heart disease, hypertension and diabetes)" and "respiratory diseases," which we will consider in more depth in that order.

A. CARDIOVASCULAR DISEASES

A good place to begin a review of temperature-related mortality is a *cold* location … like *Siberia*. Hence, we start with the study of Feigin *et al*. (2000), who examined the relationship between stroke occurrence and weather parameters in the Russian city of Novosibirsk, which has one of the highest incidence rates of stroke in the entire world.

Analyzing the health records of 2208 patients with a sex and age distribution similar to that of the whole of Russia over the period 1982-93, Feigin *et al*. found a statistically significant association between stroke occurrence and low ambient temperature. For ischemic stroke (IS), which accounted for 87% of all strokes recorded, they report that *the risk of IS occurrence on days with low ambient temperature is 32% higher than that on days with high ambient temperature*. Hence, they

suggested the implementation of "preventive measures … such as avoiding low temperature."

Hong *et al*. (2003) observed much the same thing in Incheon, Korea, over the period January 1998 to December 2000, reporting that "decreased ambient temperature was associated with risk of acute ischemic stroke," with the strongest effect being seen on the day after exposure to cold weather, further noting that "even a moderate decrease in temperature can increase the risk of ischemic stroke." In addition, they note that "risk estimates associated with decreased temperature were greater in winter than in the summer," which suggests, in their words, that "low temperatures as well as temperature changes are associated with the onset of ischemic stroke."

Nafstad *et al*. (2001) studied another cold place: Oslo, Norway. Thanks to Norwegian law, which requires all deaths to be examined by a physician who diagnoses cause and reports it on the death certificate, they were able to examine the effects of temperature on mortality due to all forms of cardiovascular disease for citizens of the country's capital over the period 1990 to 1995. Their analysis showed that *the average daily number of cardiovascular-related deaths was 15% higher in the winter months (October-March) than in the summer months (April-September)*, leading them to also conclude that "a milder climate would lead to a substantial reduction in average daily number of deaths."

To see if these relationships between cold temperatures and cardiovascular *mortality* are preceded by an even more general health-temperature relationship, Hajat and Haines (2002) set out to determine if mere cardiovascular-related *doctor visits* by the elderly bore a similar relationship to cold temperatures. Based on data obtained for registered patients aged 65 and older from several London, England practices between January 1992 and September 1995, they did indeed find that the mean number of general practitioner consultations was higher in the cool-season months (October-March) than in the warm-season months (April-September) *for all cardiovascular diseases*.

Of course, one might say, such findings are only to be expected in *cold* climates. What about *warm* climates, where summer maximum temperatures are often extreme, but summer minimum temperatures are typically mild?

In Israel, research conducted by Green *et al*. (1994) revealed that between 1976 and 1985, *mortality from cardiovascular disease was higher by 50% in mid-winter than in mid-summer, both in men and women and in different age groups*, in spite of the fact that summer

temperatures in the Negev, where much of the work was conducted, often exceed 30°C, while winter temperatures typically do not drop below 10°C. These findings are also substantiated by other Israeli studies that have been reviewed by Behar (2000), who states that "most of the recent papers on this topic have concluded that a peak of sudden cardiac death, acute myocardial infarction and other cardiovascular conditions is usually observed in low temperature weather during winter."

Evidence of a seasonal variation in cardiac-related mortality has additionally been noted in the relatively mild climate of southern California in the United States. In a study of all 222,265 death certificates issued by Los Angeles County for deaths caused by coronary artery disease from 1985 through 1996, Kloner *et al*. (1999) found that *death rates in December and January were 33% higher than those observed in the period June through September*. Likewise, based on a study of the Hunter region of New South Wales, Australia, that covered the period 1 July 1985 to 30 June 1990, Enquselassie *et al*. (1993) determined that "fatal coronary events and non-fatal definite myocardial infarction were 20-40% more common in winter and spring than at other times of year," while with respect to *daily* temperature effects, they found that "rate ratios for deaths were significantly higher for low temperatures," noting that "on cold days coronary deaths were up to 40% more likely to occur than at moderate temperatures."

In a study of both “hot” *and* “cold” cities in the United States -- where Atlanta, Georgia; Birmingham, Alabama; and Houston, Texas comprised the “hot” group, and where Canton, Ohio; Chicago, Illinois; Colorado Springs, Colorado; Detroit, Michigan; Minneapolis-St. Paul, Minnesota; New Haven, Connecticut; Pittsburgh, Pennsylvania; and Seattle and Spokane, Washington comprised the “cold” group -- Braga *et al*. (2002) determined both the acute effects and lagged influence of temperature on cardiovascular-related deaths. Their research revealed that in the hot cities, neither hot nor cold temperatures had much impact on mortality related to cardiovascular disease (CVD). In the cold cities, on the other hand, they report that both high and low temperatures were associated with increased CVD deaths, with the effect of cold temperatures persisting for days but the effect of high temperatures restricted to the day of the death or the day before. Of particular interest was the finding that *for all CVD deaths the hot-day effect was five times smaller than the cold-day effect*. In addition, the hot-day effect included some "harvesting," where the authors observed a deficit of deaths a few days later, which they did not observe for the cold-day effect.

Finally, in a study conducted in Sao Paulo, Brazil, based on data collected over the period 1991-1994, Gouveia *et al*. (2003) determined that the number of cardiovascular-related deaths in adults (15-64 years

of age) increased by 2.6% for each 1°C decrease in temperature below 20°C, while *there was no evidence for any heat-induced deaths due to temperatures rising above 20°C*. In the elderly (65 years of age and above), however, a 1°C warming above 20°C led to a 2% increase in deaths; but a 1°C cooling below 20°C led to a 6.3% increase in deaths, or *more than three times as many cardiovascular-related deaths due to cooling than to warming in the elderly*.

The results of these several studies clearly demonstrate that global warming is actually *beneficial* to humanity, in that it reduces the incidence of cardiovascular diseases related to low temperatures and wintry weather by a much greater degree than it increases the incidence of cardiovascular diseases associated with high temperatures and summer heat waves.

B. RESPIRATORY DISEASES

As with cardiovascular-related mortality, respiratory-related deaths are also more likely to be associated with cold conditions in cold countries. For example, in the Oslo study where Nafstad *et al*. (2001) found winter deaths due to cardiovascular problems to be 15% more numerous than similar summer deaths, they determined that *deaths due to respiratory diseases were fully 47% more numerous in winter than in summer*. Likewise, the London study of Hajat and Haines (2002) revealed that the number of doctor visits by the elderly was also higher in cool-season than warm-season months for all respiratory diseases. At mean temperatures below 5°C, in fact, the relationship between respiratory disease consultations and temperature was linear, and stronger at a time lag of 6 to 15 days, such that *a 1°C decrease in mean temperature below 5°C was associated with a 10.5% increase in all respiratory disease consultations*. In addition, Gouveia *et al*. (2003) found that death rates in Sao Paulo, Brazil, due to a 1°C cooling were *twice as great* as death rates due to a 1°C warming in adults, and *2.8 times greater* in the elderly.

Respiratory-related deaths were also investigated in the United States hot- and cold-city study of Braga *et al*. (2002), who found that increased temperature *variability* was the most relevant aspect of climate change with respect to this category of disease in this part of the world. Why is this finding important? Because Robeson (2002) has clearly demonstrated, from a 50-year study of daily temperatures at more than 1,000 U.S. weather stations, that temperature variability *declines* with warming, and at a very substantial rate, so that *reduced temperature variability in a warmer world would lead to reductions in temperature-*

related deaths at both the high and low ends of the daily temperature spectrum at all times of the year.

As is the case with human *cardiovascular* health, therefore, these several studies make it abundantly clear that a warming world should positively impact the *respiratory* health of the world's citizens.

C. VECTOR-BORNE DISEASES

In an article in *Science* entitled "The global spread of malaria in a future, warmer world," Rogers and Randolph (2000) note that "predictions of global climate change have stimulated forecasts that vector-borne diseases will spread into regions that are at present too cool for their persistence," which predictions comprise one of the major global-warming scare-stories of the world's climate alarmists. There are, however, several problems associated with this scenario.

According to Reiter (2000), claims that malaria resurgence is the product of CO_2-induced global warming ignore other important factors and disregard known facts. An historical analysis of malaria trends, for example, reveals that this disease was an important cause of illness and death in England during a period of *colder*-than-present temperatures throughout what has come to be called the Little Ice Age. What is more, its transmission began to decline only in the 19th century, during a *warming* phase, when, according to Reiter, "temperatures were already much higher than in the Little Ice Age."

We could well ask ourselves, therefore, why malaria was so prevalent in Europe during some of the coldest centuries of the past millennium and why we have witnessed malaria's widespread decline at a time when temperatures have been warming. Clearly, there must be other factors at work that are more important than temperature to the spread of malaria. And there are! -- factors such as the quality of public health services, irrigation and agricultural activities, land use practices, civil strife, natural disasters, ecological change, population change, the use of insecticides and the movement of people, as well as other climatic factors (Reiter, 2000, 2001; Hay *et al*., 2002).

These same sentiments are expressed by Kuhn *et al*. (2003), who analyzed the determinants of temporal trends in malaria deaths within England and Wales from 1840-1910. With respect to temperature changes over the period of study, they report finding that "a 1°C increase or decrease was responsible for an increase in malaria deaths of 8.3% or

a decrease of 6.5%, respectively," which they say explains "the malaria epidemics in the 'unusually hot summers' of 1848 and 1859." Nevertheless, there was a long-term near-linear temporal decline in malaria deaths over the period of study, which they say "was probably driven by nonclimatic factors." Foremost among the factors they list in this regard are increasing livestock populations (which tend to divert mosquito biting from humans), decreasing acreages of marsh wetlands (where mosquitoes breed), as well as "improved housing, better access to health care and medication, and improved nutrition, sanitation, and hygiene."

Kuhn *et al*. additionally note that the number of secondary malaria cases arising from each primary imported case "is currently minuscule," as demonstrated by the absence of any secondary malaria cases in the UK since 1953. Hence, they conclude that although the increase in temperature predicted for Britain by 2050 is likely to cause an 8-14% increase in the *potential* for malaria transmission, "the projected increase in proportional risk is clearly insufficient to lead to the reestablishment of endemicity." Expanding on this statement, they note that "the national health system ensures that imported malaria infections are detected and effectively treated and that gametocytes are cleared from the blood in less than a week." For Britain, therefore, they conclude that "a 15% rise in risk might have been important in the 19th century, but such a rise is now highly unlikely to lead to the reestablishment of indigenous malaria," since "socioeconomic and agricultural changes" have greatly altered the cause-and-effect relationships of the past.

Why, then, do climate alarmists predict widespread increases in malaria in response to global warming? They do it because nearly all of the studies they cite ignore these non-climatic factors and additionally use only one, or at most two, climate variables to characterize the current distribution of the disease when developing models to predict its future distribution. In contrast, Rogers and Randolph (2000) developed a predictive model that employs a total of *five* climate variables and obtained very different results: a mere 0.84% increase in potential malaria exposure under what they call the "medium-high" scenario of global warming and a 0.92% *decrease* under the "high" scenario. They thus rightly note that their model "contradicts prevailing forecasts of global malaria expansion" and that "it highlights the use [we would say *superiority*] of multivariate rather than univariate constraints in such applications."

Using a similar approach, Hay *et al*. (2002) investigated long-term trends in meteorological data at four East African highland sites that experienced significant increases in malaria cases over the past couple of decades, reporting that "temperature, rainfall, vapour pressure and the

number of months suitable for *P. falciparum* transmission have not changed significantly during the past century or during the period of reported malaria resurgence." Hence, these factors could not be responsible for the observed increases in malaria cases recently noted at these sites. Likewise, Shanks *et al*. (2000) examined trends in temperature, precipitation and malaria rates in western Kenya over the period 1965-1997, also finding absolutely no linkages among the variables.

It would thus appear that models used by climate alarmists to predict the spread of malaria in response to global warming are much too simplistic to reveal its true climatic dependency. In addition, the possibility that malaria expansion might occur as a result of rising temperatures is further severely weakened by the potential for effective human intervention. In the words of Dye and Reiter (2000), "given adequate funding, technology, and, above all, commitment, the campaign to 'Roll Back Malaria,' spearheaded by the World Health Organization, will have halved deaths related to [malaria] by 2010," so that "by 2050, the map of malaria distribution should bear little resemblance to the one drawn by Rogers and Randolph." In fact, if all goes well, there may not even be such a map!

Pretty much the same things can be said about dengue and yellow fever; and, in fact, *they have been said*. Reiter (2001), for example, notes that the natural history of these vector-borne diseases is highly complex; and the interplay of climate, ecology, vector biology and a number of other factors defies definition by the simplistic analyses utilized in the models employed by climate alarmists to generate predictions of future increases in the spread of these diseases under various global warming scenarios. There have been some reports of a recent resurgence of mosquito-born maladies in certain parts of the world; but, as Reiter states, it is "facile to attribute this resurgence to climate change." Indeed, he presents a case-by-case analysis demonstrating that factors associated with politics, economics and human activity -- *but not climate change* -- are the principal determinants of the spread of these diseases, going on to conclude that it is “inappropriate to use climate-based models to predict future prevalence."

There has also been some concern of late with respect to a potential cholera-climate connection. Pascual *et al*. (2002), for example, report that recent data analyses support a temporal association between the El Niño-Southern Oscillation (ENSO) phenomenon and the interannual variability of cholera in certain parts of the world, as well as a role of increased water temperature in enhancing the survival and growth of the pathogen that is responsible for the disease, although they say "it is not yet possible to assess the strength of particular climatic drivers." In

addition, they report that variations in water *volume* "can have dramatic effects on disease dynamics, perhaps more pronounced than those of factors affecting the pathogen's growth and survival."

Although climatic factors undoubtedly are involved in the dynamics of cholera, they are not well defined; and even when they ultimately are understood, the importance of socio-economic factors for the development and spread of cholera -- which is often described, in the words of Pascual *et al*., as "the disease of poverty" -- will likely far outweigh them. As the researchers describe the situation, "the importance of sanitary conditions is clearly indisputable," in support of which declaration they cite the fact that "infrastructure providing safe water and sewage treatment in industrialized nations has made the sustained transmission of cholera extremely unlikely." Hence, it would appear that the best long-term preventive measures to take against cholera would be those that enhance the wealth of nations and their citizens.

Another major vector-borne disease is tick-borne encephalitis (TBE), which according to Randolph and Rogers (2000) "is the most significant vector-borne disease in Europe and Eurasia," having "a case morbidity rate of 10-30% and a case mortality rate of typically 1-2% but as high as 24% in the Far East." The flavivirus (TBEV) that causes TBE is maintained in natural rodent-tick cycles; and humans may be infected if bitten by an infected tick or by drinking untreated milk from infected sheep or goats.

Early writings on the relationship of this disease to global warming predicted that TBE -- like so many other vector-born diseases -- would expand its range and become more of a threat to humans in a warmer world. However, Randolph and Rogers draw our attention to the fact that "like many vector-borne pathogen cycles that depend on the interaction of so many biotic agents with each other and with their abiotic environment, enzootic cycles of TBEV have an inherent fragility," such that "their continuing survival or expansion cannot be predicted from simple univariate correlations." Hence, the two researchers decided to explore the subject in significantly greater detail than had ever been done before.

Confining their analysis to Europe, Randolph and Rogers first matched the present-day distribution of TBEV to the present-day distribution of *five* climatic variables: average monthly mean, maximum and minimum temperatures, plus rainfall and saturation vapor pressure, "to provide a multivariate description of present-day areas of disease risk." They then applied this understanding to outputs of a general circulation model of

the atmosphere that predicted how these five climatic variables may change in the future.

The results of this effort indicated that the distribution of TBEV may expand both north and west of Stockholm, Sweden, in a warming world. Elsewhere, however, the authors say that "fears for increased extent of risk from TBEV caused by global climate change appear to be unfounded." In fact, they note that "the precise conditions required for enzootic cycles of TBEV are predicted to be disrupted" in response to global warming, while the new climatic state "appears to be lethal for TBEV."

This analysis, in Randolph and Rogers' words, "gives the lie to the common perception that a warmer world will necessarily be a world under greater threat from vector-borne diseases." In the case of TBEV, in fact, they note that the predicted change "appears to be to our advantage."

A similar conclusion was reached by Estrada-Peña (2003), who studied the effects of various abiotic factors on the habitat suitability of four tick species that are major vectors of livestock pathogens in South Africa. This work led to the development of species-specific models of tick habitat suitability, which indicated that "year-to-year variations in the forecasted habitat suitability over the period 1983-2000 show a clear decrease in habitat availability, which is attributed primarily to increasing temperature in the region over this period." In addition, when climate variables were projected to the year 2015, it was determined that "the simulations show a trend toward the destruction of the habitats of the four tick species."

Commenting on this finding, Estrada-Peña notes that "it is often suggested that one of the most important societal consequences of climate change may be an increase in the geographic distribution and transmission intensity of vector-borne disease." In the cases of the four disease-carrying ticks of South Africa described in this study, however, just the *opposite* was observed.

In considering the several findings described in this section, it is clear that vector-borne diseases are unlikely to be affected in any major way by a continuation of the most recent -- and possibly still on-going -- spate of global warming that has brought about the welcome demise of the Little Ice Age and ushered in the productive and prosperous Modern Warm Period.

D. ALL DISEASES

In a study of mortality in general, Keatinge and Donaldson (2001) analyzed the effects of temperature, wind, rain, humidity and sunshine during high pollution days in the greater London area over the period 1976-1995 to determine what weather and/or pollution factors have the biggest influence on human mortality. Their most prominent finding was that simple plots of mortality rate versus daily air temperature revealed a linear increase in deaths as temperatures fell from 15°C to near 0°C. Mortality rates at temperatures above 15°C were, in the words of the researchers, "grossly alinear," showing no trend. Days with high pollutant concentrations were colder than average, but a multiple regression analysis revealed that no pollutant was associated with a significant increase in mortality among people over fifty years of age. Indeed, *only low temperatures were shown to have a significant effect on both immediate (1 day after the temperature perturbation) and long-term (up to 24 days after the temperature perturbation) mortality rates*.

In a closely allied study, Keatinge *et al*. (2000) examined heat- and cold-related mortality in north Finland, south Finland, southwest Germany, the Netherlands, Greater London, north Italy, and Athens, Greece, in people aged 65-74. For each of these regions, they determined the 3°C temperature interval of lowest mortality and then evaluated mortality deviations from that base level as temperatures rose and fell by 0.1°C increments. The result, according to the researchers, was that "all regions showed more annual cold related mortality than heat related mortality." In fact, over the seven regions studied, *annual cold related deaths were nearly ten times greater than annual heat related deaths*. The scientists also note that the very successful adjustment of the different populations they studied to widely different summer temperatures "gives grounds for confidence that they would adjust successfully, with little increase in heat related mortality, to the global warming of around 2°C predicted to occur in the next half century." Indeed, they say their data suggest that "any increases in mortality due to increased temperatures would be outweighed by much larger short term declines in cold related mortalities." For the population of Europe, therefore, an increase in temperature would appear to be a climate change for the better.

Gouveia *et al*. (2003) conducted a similar study in Sao Paulo, Brazil, where they tabulated the numbers of daily deaths from all causes (excepting violent deaths and deaths of infants up to one month of age), which they obtained from the city's mortality information system for the period 1991-1994. They then analyzed these data for *children* (less than 15 years of age), *adults* (ages 15-64), and the *elderly* (age 65 and above) with respect to the impacts of warming and cooling. For each

1°C increase above the minimum-death temperature of 20°C for a given and prior day's mean temperature, there was a 2.6% increase in deaths from all causes in children, a 1.5% increase in deaths from all causes in adults, and a 2.5% increase in deaths from all causes in the elderly. For each 1°C decrease *below* the 20°C minimum-death temperature, however, *the cold effect was greater*, with increases in deaths from all causes in children, adults and the elderly registering 4.0%, 2.6% and 5.5%, respectively, which cooling-induced death rates are 54%, 73% and 120% *greater* than those attributable to warming.

In a similar study conducted in Shanghai, China, from 1 Jun 2000 to 31 Dec 2001, Kan *et al*. (2003) found a V-like relationship between total mortality and temperature that had a minimum mortality risk at 26.7°C. Above this temperature, they note that "total mortality increased by 0.73% for each degree Celsius increase; while for temperatures below the optimum value, total mortality decreased by 1.21% for each degree Celsius increase." Hence, it can be appreciated that the net effect of a warming of the climate of Shanghai would likely be *reduced* mortality on the order of 0.5% per degree Celsius increase in temperature, *or perhaps even more*, in light of the fact that the warming of the past few decades has been primarily due to increases in daily *minimum* temperatures.

In the United States, Goklany and Straja (2000) studied deaths due to all causes over the period 1979-97, finding there were no trends due to either extreme heat or cold in the entire population or, even more remarkably, in the older more susceptible age groups, i.e., those aged 65 and over, 75 and over, and 85 and over. Nevertheless, *deaths due to extreme cold exceeded those due to extreme heat by 80% to 125%*. With respect to the absence of *trends* in U.S. death rates attributable to either extreme heat or cold, Goklany and Straja say this observation "suggests that adaptation and technological change may be just as important determinants of such trends as more obvious meteorological and demographic factors."

Donaldson *et al*. (2003) suggest much the same thing. For three areas of the world -- North Carolina, USA; South Finland; and Southeast England -- they determined the mean daily May-August 3°C temperature bands in which deaths of people aged 55 and above were at a minimum. Then they compared heat- and cold-related deaths that occurred at temperatures above and below this optimum temperature interval for each region, after which they determined how heat-related deaths in the three areas changed between1971 and 1997 in response to: (1) the 1.0°C temperature rise that was experienced in North Carolina over this period (from an initial temperature of 23.5°C), (2) the 2.1°C temperature rise experienced in Southeast England (from an initial

temperature of 14.9°C), and (3) the unchanging 13.5°C temperature of South Finland.

First, it was determined that the 3°C temperature band at which mortality was at its local minimum was lowest for the coolest region (South Finland), highest for the warmest region (North Carolina), and intermediate for the region of intermediate temperature (Southeast England). This finding suggests that the populations of the three regions were somewhat acclimated to their respective thermal regimes. Second, for *each* of the three regions, it was determined that *cold*-related mortality (expressed as excess mortality at temperatures *below* the region's optimum 3°C temperature band), was **greater** than *heat*-related mortality (expressed as excess mortality at temperatures *above* the region's optimum 3°C temperature band).

As for the third aspect of the study, i.e., changes in heat-related mortality from 1971 to 1997, it was determined that in the coldest of the three regions (South Finland, where there was *no change* in temperature over the study period), heat-related deaths per million inhabitants in the 55-and-above age group *declined* from 382 to 99. In somewhat warmer Southeast England, however, where it warmed by a whopping 2.1°C over the study period, heat-related deaths per million of the at-risk age cohort *still declined*, but this time from only 111 to 108. Last of all, in the warmest of the three regions (North Carolina, USA, where mean daily May-August temperature rose by 1.0°C over the study period), corresponding heat-related deaths *also fell*, and this time from 228 to a mere 16 per million.

From these several observations we learn that most people can adapt to both warmer and cooler climates and that cooling tends to produce many more deaths than warming, irrespective of the initial temperature regime. As for the reason behind the third observation -- the dramatic decline in heat-related deaths in response to warming in the hottest region of the study (North Carolina) -- Donaldson *et al*. (2003) attribute it to the increase in the availability of air conditioning in the South Atlantic region of the United States, where they note that the percentage of households with some form of air conditioning rose from 57% in 1978 to 72% in 1997. With respect to the declining heat-related deaths in the other two areas, they say "the explanation is likely to lie in the fact that both regions shared with North Carolina an increase in prosperity, which could be expected to increase opportunities for avoiding heat stress."

Another revealing investigation into the comparative dangers of unseasonably hot and cold temperatures was conducted by Huynen *et al*. (2001), who analyzed mortality rates in the entire population of Holland. For the 19-year period 1 January 1979 through 31 December

1997, the group of five scientists compared the numbers of deaths in people of all ages that occurred during well-defined heat waves and cold spells. Their bottom-line findings indicated there was a total excess mortality of 39.8 deaths *per day* during heat waves and 46.6 deaths *per day* during cold spells.

These numbers indicate that a typical cold-spell *day* kills at a rate that is 17% greater than a typical heat-wave *day* in the Netherlands. In addition, the researchers note that the heat waves they studied ranged from 6 to 13 days in length, while the cold spells lasted 9 to 17 days, making the average cold spell approximately 37% longer than the average heat wave. Adjusting for this duration differential thus makes the number of deaths *per cold spell* in the Netherlands fully *60% greater* than the number of deaths *per heat wave*. What is more, excess mortality *continued* during the whole month *after* the cold spells, leading to even *more* deaths; while in the case of heat waves, there actually appeared to be mortality *deficits* in the following month, which suggests, in the words of the authors, "that some of the heat-induced increase in mortality can be attributed to those whose health was already compromised" or "who would have died in the short term anyway." This same conclusion has also been reached in a number of other studies (Kunst *et al*., 1993; Alberdi *et al*., 1998; Eng and Mercer, 1998; Rooney *et al*., 1998). It is highly likely, therefore, that the 60% greater death toll we have calculated for Dutch cold spells as compared to Dutch heat waves is a vast *underestimate* of the true differential killing power of these two extreme weather phenomena.

The Dutch could well ask themselves, therefore, "Will global climate change reduce thermal stress in the Netherlands?" ... which is exactly what the senior and second authors of the Huynen *et al*. paper did in a letter to the editor of *Epidemiology* that bore that very title (Martens and Huynen, 2001). Based on the predictions of nine different GCMs for an atmospheric CO_2 concentration of 550 ppm in the year 2050 -- which implied a 50% increase in Dutch heat waves and a 67% drop in Dutch cold spells -- they calculated a total mortality *decrease* for Holland of approximately *1100 people per year* at that point in time.

Yes, global warming -- if it continues, and for whatever reason -- will result, not in more lives *lost*, but in more lives *saved*. And it's not just the Dutch that will be thus blessed; data from all over the world tell the same story.

Take Germany, for instance. Laschewski and Jendritzky (2002) analyzed daily mortality rates of the population of Baden-Wurttemberg (10.5 million inhabitants) over the 30-year period 1958-1997 to determine the sensitivity of the people living in this moderate climatic zone of

southwest Germany to long-and short-term episodes of heat and cold. With respect to long-term conditions, they note that the mortality data "show a marked seasonal pattern with a minimum in summer and a maximum in winter." With respect to short-term conditions, they report that "cold spells lead to excess mortality to a relatively small degree, which lasts for weeks," and that "the mortality increase during heat waves is more pronounced, but is followed by lower than average values in subsequent weeks." These scientists also say this latter observation suggests that people who died from short-term exposure to heat likely "would have died in the short term anyway."

With respect to this short-term mortality displacement in the case of heat-related deaths, we note that the authors' data demonstrate it is precisely that, i.e., merely a *displacement* of deaths and not an overall increase. They found, for example, that the mean duration of above-normal mortality for the 51 heat episodes that occurred from 1968 to 1997 was10 days, with a mean increase in mortality of 3.9%, after which there was a mean *decrease* in mortality of 2.3% for 19 days. Hence, the *net* effect of the two perturbations was essentially nil (actually, a calculated overall *decrease* in mortality of 0.2% over the full 29-day period).

In light of the knowledge gained from these several studies of the effects of temperature on human mortality due to *all* health problems, it can readily be appreciated that it is *cooling* that kills, not warming. Hence, those people who claim to be concerned about the health effects of climate change are being dishonest with both themselves and others when they say that CO_2-induced global warming is killing people. In point of fact, CO_2-induced global warming -- if it is even occurring at all -- is enabling earth's populace to actually lead both *longer* and *more productive* lives.

III. NON-CLIMATIC HEALTH EFFECTS OF ELEVATED CO_2

Even if atmospheric CO_2 enrichment caused significant global warming (which is highly unlikely), and even if global warming caused an increase in human death rate (which it clearly does not), it would still be necessary to consider *other* potential health effects of atmospheric CO_2 enrichment that are *not* related to climate in order to determine the *net* effect of elevated levels of atmospheric CO_2 on human health and longevity. Hence, we explore several aspects of this subject in the sections that follow.

A. DIRECT HEALTH EFFECTS OF ELEVATED CO_2

Almost all trace elements and compounds, even beneficial ones, can be poisonous if ingested or inhaled in large enough concentrations. So what about carbon dioxide? Do we have to worry about any deleterious health effects as its atmospheric concentration continues to climb?

Inhaling *very* high concentrations of atmospheric CO_2 can induce a state of *hypercapnia* in people (Nahas *et al*., 1968; Brackett *et al*., 1969; van Ypersele de Strihou, 1974). Characterized by an excessive amount of CO_2 in the blood, which typically results in *acidosis*, this condition is accompanied by headache, nausea, visual disturbances, and is sometimes fatal (Poyart and Nahas, 1968; Turino *et al*., 1974). Several studies have demonstrated, however, that these problems do not seriously impact human health until the air's CO_2 concentration reaches approximately 15,000 ppm (Luft *et al*., 1974; Schaefer, 1982), which is approximately 40 times greater than its current concentration.

Clearly, therefore, we do not have to worry about there being any direct adverse health effects associated with the ongoing rise in the air's CO_2 content, even if it were to increase by a factor of ten, which is probably all that could be achieved by burning the entire supply of fossil fuels in the crust of the earth. In fact, the *current* CO_2 concentration of the air in many homes and buildings is often two to three times greater than the CO_2 concentration of outdoor air (Idso, 1997), which in large cities is itself often elevated by several tens of percent above the CO_2 concentration of rural air (Idso *et al*. 1998, 2002).

B. INDIRECT HEALTH EFFECTS OF ELEVATED CO_2

The old saying "you are what you eat" suggests that effects of atmospheric CO_2 enrichment on *food production* must also be considered in any assessment of the health effects of the historical and still-ongoing rise in the air's CO_2 content. Hence, we begin our investigation of this subject with a brief review of the well-known *aerial fertilization effect* of atmospheric CO_2 enrichment and how it impacts the human health issue.

1. Quantity of Food

First and foremost, people must have *sufficient* food, simply to sustain themselves; and the rise in the atmosphere's CO_2 concentration that has occurred since the inception of the Industrial Revolution (an increase of approximately 100 ppm) has done wonders for humanity in this regard.

a. The past

In a revealing study of the beneficial impact of mankind's historical CO_2 emissions on world food production, Mayeux *et al*. (1997) grew two cultivars of commercial wheat in a 38-meter-long soil container topped with a transparent tunnel-like polyethylene cover within which a CO_2 gradient was created that varied from approximately 350 ppm at one end of the tunnel to about 200 ppm at the other end. Both of the wheat cultivars were irrigated weekly over the first half of the 100-day growing season, so as to maintain soil water contents near optimum conditions. Over the last half of the season, however, this regimen was maintained on only half of the wheat of each cultivar, in order to create both water-stressed and well-watered treatments.

At the conclusion of the experiment, the scientists determined that the growth response of the wheat was a linear function of atmospheric CO_2 concentration in both cultivars under both adequate and less-than-adequate soil water regimes. Based on the linear regression equations they developed for grain yield in these situations, we calculate that the 100-ppm increase in atmospheric CO_2 concentration experienced over the past century and a half should have increased the mean grain yield of the two wheat cultivars by about 72% under well-watered conditions and 48% under water-stressed conditions, for a mean yield increase on the order of 60% under the full range of moisture conditions likely to have existed throughout the entire real world.

It is also important to note that this CO_2-induced yield enhancement was not restricted to wheat. Based on the voluminous amount of data

summarized by Idso and Idso (2000) for the world's major food crops, the calculations we have made for wheat can be scaled to determine what the past 150-year increase in atmospheric CO_2 concentration likely did for the productivity of other agricultural staples. Doing so, we find that the Industrial Revolution's flooding of the air with CO_2 resulted in mean yield increases on the order of 70% for other C_3 cereals, 28% for C_4 cereals, 33% for fruits and melons, 62% for legumes, 67% for root and tuber crops, and 51% for vegetables.

b. The future

Clearly, the historical increase in the air's CO_2 content that has been experienced to date has vastly benefited mankind and enabled our numbers to grow considerably. In fact, the very existence of many of the people who read these words may well be attributed to that phenomenon. But what of the future? The population explosion of our species has not yet subsided; and there is real concern about our ability to feed the projected population of the world a mere fifty years hence.

Tilman *et al*. (2001) address this problem in an analysis of the global environmental impacts of agricultural expansion that may occur over the next half-century. Based on projected increases in population, *and even accounting for expected concomitant advances in technological expertise*, they conclude that the task of meeting the global food demand expected to exist in the year 2050 will likely exact a heavy environmental toll and produce great societal impacts.

What are the specific problems? Tilman and his colleagues report that "humans currently appropriate more than a third of the production of terrestrial ecosystems and about half of usable freshwaters," noting that this usurpation of natural resources will increase even more in the future. In terms of the amount of land devoted to agriculture, they calculate an 18% increase over the present by the year 2050; but because developed countries are expected to *withdraw* large areas of land from farming over the next fifty years, the net loss of natural ecosystems to cropland and pasture in *developing* countries will amount to about half of all potentially suitable remaining land, which would "represent the worldwide loss of natural ecosystems larger than the United States."

The scientists go on to say that this phenomenon "could lead to the loss of about a third of remaining tropical and temperate forests, savannas, and grasslands." What is more, in a worrisome reflection upon the consequences of these land-use changes for both plants and animals, they remind us that species extinction follows rapidly on the heels of habitat destruction. Finally, in another acknowledgement of just how

serious the situation is, Tilman and his associates report that "even the best available technologies, fully deployed, cannot prevent many of the forecasted problems."

So what can possibly be done to avert this future food production shortfall and its devastating consequences that "even the best available technologies, fully deployed," cannot prevent? This is the question that was addressed by Idso and Idso (2000) in their treatise entitled *Forecasting World Food Supplies: The Impact of the Rising Atmospheric CO_2 Concentration*; and it was their conclusion that -- *after all that man can do* -- the aerial fertilization effect of the increase in the air's CO_2 content that is expected to occur by the year 2050 would be *just barely sufficient*, in the mean, to assure the agricultural productivity required to prevent mass starvation in many parts of the globe without usurping what little of the natural world would remain at that time.

In view of these observations, not only is the ongoing rise in the air's CO_2 content essential for the future well-being of man, *it is essential to the future well-being of the entire biosphere*.

2. Quality of Food

Clearly, *quantity* of food is mankind's number one concern when it comes to survival; but after survival is assured, *quality* of food rises to the fore. What role does the ongoing rise in the air's CO_2 content play here?

a. Protein content

In a review of the scientific literature related to effects of atmospheric CO_2 enrichment on plant constituents of significance to human health, Idso and Idso (2001) cited a number of studies that indicated elevated levels of atmospheric CO_2 may at times increase, decrease or have no effect upon the protein contents of various foods.

In the case of wheat -- which according to Wittwer (1995) is "the most widely grown plant in the world today," contributing "more calories and protein to the human diet than any other food" -- Pleijel *et al*. (1999) were able to bring some semblance of order to this confusing situation by analyzing the results of 16 open-top chamber experiments that had been conducted on spring wheat in Denmark, Finland, Sweden and Switzerland between 1986 and 1996. In addition to CO_2 enrichment of the air, these experiments included increases and decreases in atmospheric ozone (O_3); and Pleijel *et al*. found that *when increasing O_3 pollution reduced wheat grain yield, it simultaneously increased the*

protein concentration of the grain. They also found that *when O_3 was scrubbed from the air and grain yield was thereby increased, the protein concentration of the grain was decreased*. Moreover, *this same relationship described the degree to which grain protein concentrations dropped when atmospheric CO_2 enrichment increased grain yield*. Hence, it became clear that whenever the grain yield of the wheat was changed -- by CO_2, O_3 or even water stress, which was also a variable in one of the experiments -- grain protein concentrations either moved up or down along a common linear relationship in the opposite direction to the change in grain yield elicited by the CO_2, O_3 or water stress treatment.

In an earlier study of CO_2 and O_3 effects on wheat grain yield and quality, Rudorff *et al*. (1996) obtained essentially the same results. They observed, for example, that "flour protein contents were increased by enhanced O_3 exposure and reduced by elevated CO_2" but that "the combined effect of these gases was minor." Hence, they concluded that "the concomitant increase of CO_2 and O_3 in the troposphere will have no significant impact on wheat grain quality."

Earlier still, Evans (1993) had found similar relationships to exist for several other crops, further observing them to be greatly affected by soil nitrogen availability. It is highly likely, therefore, that the differing availability of soil nitrogen could have been responsible for some of the differing results observed in the many other studies reviewed by Idso and Idso (2001); and, in fact, that is precisely what the study of Rogers *et al*. (1996) suggests. Although the latter investigators observed CO_2-induced reductions in the protein concentration of flour derived from wheat plants growing at low soil nitrogen concentrations, no such reductions were evident when the soil nitrogen supply was increased to a higher rate of application. Hence, Pleijel *et al*. concluded that the oft-observed negative impact of atmospheric CO_2 enrichment on grain protein concentration would probably be alleviated by higher applications of nitrogen fertilizers; and the study of Kimball *et al*. (2001) confirmed their hypothesis.

Kimball *et al*. studied the effects of a 50% increase in atmospheric CO_2 concentration on wheat grain nitrogen concentration and the baking properties of the flour derived from that grain throughout four years of free-air CO_2 enrichment experiments. In the first two years of their study, soil water content was an additional variable; and in the last two years, soil nitrogen content was a variable. The most influential factor in reducing grain nitrogen concentration was determined to be low soil nitrogen; and under this condition, atmospheric CO_2 enrichment further reduced grain nitrogen and protein concentrations, although the change was much less than that caused by low soil nitrogen. When soil nitrogen was *not* limiting, however, increases in the air's CO_2 concentration did

not affect grain nitrogen and protein concentrations; neither did they reduce the baking properties of the flour derived from the grain. Hence, it would appear that *given sufficient water and nitrogen, atmospheric CO_2 enrichment can significantly increase grain yield without sacrificing grain protein concentration in the process*.

In some situations, however, atmospheric CO_2 enrichment may actually *increase* the protein concentration of wheat. Agrawal and Deepak (2003), for example, grew two cultivars of wheat (*Triticum aestivum* L. cv. Malviya 234 and HP1209) in open-top chambers maintained at atmospheric CO_2 concentrations of 350 and 600 ppm alone and in combination with 60 ppb SO_2 to study the interactive effects of elevated CO_2 and this major air pollutant on crop growth. They found that exposure to elevated SO_2 caused an average 13% decrease in foliar protein concentrations in both cultivars. However, when plants were concomitantly exposed to an atmospheric CO_2 concentration of 600 ppm, leaf protein levels only decreased by 3% in HP1209, while *they actually increased by 4%* in Malviya 234.

In the case of rice -- which according to Wittwer (1995) is "the basic food for more than half the world's population," supplying "more dietary energy than any other single food" -- Jablonski *et al*. (2002) conducted a wide-ranging review of the scientific literature, finding that it too appeared to suffer no reduction in grain nitrogen (protein) concentration in response to atmospheric CO_2 enrichment. Likewise, they found no CO_2-induced decrease in seed nitrogen concentration in the studies of *legumes* they reviewed. This finding is also encouraging, since according to Wittwer (1995), legumes "are a direct food resource providing 20% of the world's protein for human consumption," as well as "about two thirds of the world's protein concentrate for livestock feeding." What is more, the biomass of the CO_2-enriched wheat, rice and legumes was found by Jablonski *et al*. to be significantly increased. Hence, there will likely be a vast increase in the total amount of protein that can be made available to humanity in a future CO_2-enriched world, both directly via food crops and indirectly via livestock.

With respect to the specific legume *soybeans*, Thomas *et al*. (2003) note that "oil and protein comprise ~20 and 40%, respectively, of the dry weight of soybean seed," which "unique chemical composition," in their words, "has made it one of the most valuable agronomic crops worldwide." In addition, they say that "the intrinsic value of soybean seed is in its supply of essential fatty acids and amino acids in the oil and protein, respectively;" and in this regard they report that Heagle *et al*. (1998) "observed a positive significant effect of CO_2 enrichment on soybean seed oil and oleic acid concentration," although they could find no such effect in their study.

b. Antioxidant content

Antioxidants are also of great importance to human health; and one of the most prominent of these plant products is *ascorbate* or vitamin C. In the early studies of Barbale (1970) and Madsen (1971, 1975), a tripling of the atmospheric CO_2 concentration produced a modest (7%) increase in this antioxidant in the fruit of tomato plants. Kimball and Mitchell (1981), however, could find no effect of a similar CO_2 increase on the same species, although the extra CO_2 of their study stimulated the production of vitamin A. In bean sprouts, on the other hand, a mere one-hour-per-day doubling of the atmospheric CO_2 concentration actually *doubled* plant vitamin C contents over a 7-day period (Tajiri, 1985).

Probably the most comprehensive investigation of CO_2 effects on vitamin C production in an agricultural plant -- a tree crop (sour orange) -- was conducted by Idso *et al*. (2001). In an atmospheric CO_2 enrichment experiment begun in 1987 and still ongoing, a 75% increase in the air's CO_2 content was observed to increase sour orange juice vitamin C concentration by approximately 5% in run-of-the-mill years when total fruit production was typically enhanced by about 80%. In aberrant years when the CO_2-induced increase in fruit production was much greater, however, the increase in fruit vitamin C concentration was also greater, rising to a CO_2-induced enhancement of 15% when fruit production on the CO_2-enriched trees was 3.6 times greater than it was on the ambient-treatment trees.

These findings take on great significance when it is realized that scurvy -- which is induced by low intake of vitamin C -- may be resurgent in industrial countries, especially among children (Ramar *et al*., 1993; Gomez-Carrasco et al., 1994), and that subclinical scurvy symptoms are increasing among adults (Dickinson *et al*., 1994). Furthermore, Hampl *et al*. (1999) have found that 12-20% of 12- to 18-year-old school children in the United States "drastically under-consume" foods that supply vitamin C; while Johnston *et al*. (1998) have determined that 12-16% of U.S. college students have marginal plasma concentrations of vitamin C. Hence, since vitamin C intake correlates strongly with the consumption of citrus juice (Dennison *et al*., 1998), and since the only high-vitamin-C juice consumed in any quantity by children is orange juice (Hampl *et al*., 1999), the modest role played by the ongoing rise in the air's CO_2 content in increasing the vitamin C concentration of orange juice could ultimately prove to be of considerable significance for public health in the United States and elsewhere.

Another important study to assess the impact of elevated levels of atmospheric CO_2 on plant antioxidant production was that of Wang *et al*.

(2003), who evaluated the effects of elevated CO_2 on the antioxidant activity and flavonoid content of strawberry fruit in field plots at the U.S. Department of Agriculture's Beltsville Agricultural Research Center in Beltsville, Maryland, where they grew strawberry plants (*Fragaria x ananassa* Duchesne cv. Honeoye) in six clear-acrylic open-top chambers, two of which were maintained at the ambient atmospheric CO_2 concentration, two of which were maintained at ambient + 300 ppm CO_2, and two of which were maintained at ambient + 600 ppm CO_2 for a period of 28 months (from early spring of 1998 through June 2000). The scientists harvested the strawberry fruit, in their words, "at the commercially ripe stage" in both 1999 and 2000, after which they analyzed them for a number of different antioxidant properties and flavonol contents.

Before reporting what they found, Wang *et al*. provide some background by noting that "strawberries are good sources of natural antioxidants (Wang *et al*., 1996; Heinonen *et al*., 1998)." They further report that "in addition to the usual nutrients, such as vitamins and minerals, strawberries are also rich in anthocyanins, flavonoids, and phenolic acids," and that "strawberries have shown a remarkably high scavenging activity toward chemically generated radicals, thus making them effective in inhibiting oxidation of human low-density lipoproteins (Heinonen *et al*., 1998)." In this regard, they note that previous studies (Wang and Jiao, 2000; Wang and Lin, 2000) "have shown that strawberries have high oxygen radical absorbance activity against peroxyl radicals, superoxide radicals, hydrogen peroxide, hydroxyl radicals, and singlet oxygen." In their experiment, therefore, they were essentially seeking to see if atmospheric CO_2 enrichment could make a good thing even better.

So what did the Agricultural Research Service scientists find? They determined, first of all, that strawberries had higher concentrations of ascorbic acid (AsA) and glutathione (GSH) "when grown under enriched CO_2 environments." In going from ambient to ambient + 300 ppm CO_2 and ambient + 600 ppm CO_2, for example, AsA concentrations increased by 10 and 13%, respectively, while GSH concentrations increased by 3 and 171%, respectively. They also learned that "an enriched CO_2 environment resulted in an increase in phenolic acid, flavonol, and anthocyanin contents of fruit." For nine different flavonoids, for example, there was a mean concentration increase of 55 ± 23% in going from the ambient atmospheric CO_2 concentration to ambient + 300 ppm CO_2, and a mean concentration increase of 112 ± 35% in going from ambient to ambient + 600 ppm CO_2. In addition, they report that the "high flavonol content was associated with high antioxidant activity." As for the significance of these findings, Wang *et al*. note that "anthocyanins have been reported to help reduce damage caused by free radical activity, such as low-density lipoprotein oxidation, platelet aggregation, and

endothelium-dependent vasodilation of arteries (Heinonen *et al*., 1998; Rice-Evans and Miller, 1996)."

In summarizing their findings, Wang *et al*. say "strawberry fruit contain flavonoids with potent antioxidant properties, and under CO_2 enrichment conditions, increased the[ir] AsA, GSH, phenolic acid, flavonol, and anthocyanin concentrations," further noting that "plants grown under CO_2 enrichment conditions also had higher oxygen radical absorbance activity against [many types of oxygen] radicals in the fruit." Hence, they determined that atmospheric CO_2 enrichment truly did "make a good thing better."

We note additionally in this regard that elevated levels of atmospheric CO_2 also make *more* of that good thing. Deng and Woodward (1998), for example, report that after growing strawberry plants in air containing an additional 170 ppm of CO_2, total fresh fruit weights were 42 and 17% greater than weights displayed by control plants grown at high and low soil nitrogen contents, respectively; while Bushway and Pritts (2002) report that a two- to three-fold increase in the air's CO_2 content boosted strawberry fruit yield by an average of 62%. In addition, Campbell and Young (1986), Keutgen *et al*. (1997), and Bunce (2001) report positive strawberry photosynthetic responses to an extra 300 ppm of CO_2 ranging from 9% to 197% (mean of 76% $\pm$ 15%); and Desjardins *et al*. (1987) report a 118% increase in photosynthesis in response to a 600 ppm increase in the air's CO_2 concentration.

3. Medicinal Constituents of Plants

Primitive medical records indicate that extracts from many species of plants have been used for treating a variety of human health problems for perhaps the past 3500 years (Machlin, 1992; Pettit *et al*., 1993, 1995). In modern times the practice has continued, with numerous chemotherapeutic agents being isolated (Gabrielsen *et al*., 1992a). Until recently, however, no studies had investigated the effects of atmospheric CO_2 enrichment on specific plant compounds of direct medicinal value.

This situation changed when Stuhlfauth *et al*. (1987) studied the individual and combined effects of atmospheric CO_2 enrichment and water stress on the production of secondary metabolites in the woolly foxglove (*Digitalis lanata* EHRH), which produces the cardiac glycoside *digoxin* that is used in the treatment of cardiac insufficiency. Under controlled well-watered conditions in a phytotron, a near-tripling of the air's CO_2 content increased plant dry weight production in this medicinal plant by 63%, while under water-stressed conditions the CO_2–induced dry weight increase was 83%. In addition, the *concentration* of digoxin

within the plant dry mass was enhanced by 11% under well-watered conditions and by 14% under conditions of water stress.

In a subsequent whole-season field experiment, Stuhlfauth and Fock (1990) obtained similar results. A near-tripling of the air's CO_2 concentration led to a 75% increase in plant dry weight production per unit land area and a 15% increase in digoxin yield per unit dry weight of plant, which combined to produce an actual doubling of total digoxin yield per hectare of cultivated land.

Equally impressive was the study of Idso *et al*. (2000), who evaluated the response of the tropical spider lily (*Hymenocallis littoralis* Jacq. Salisb.) to elevated levels of atmospheric CO_2 over four growing seasons. This plant has been known since ancient times to possess anti-tumor activity; and in modern times it has been shown to contain constituents that are effective against lymphocytic leukemia and ovary sarcoma (Pettit *et al*., 1986). These same plant constituents have also been proven to be effective against the U.S. National Cancer Institute's panel of 60 human cancer cell lines, demonstrating greatest effectiveness against melanoma, brain, colon, lung and renal cancers (Pettit *et al*., 1993). In addition, it exhibits strong anti-viral activity against Japanese encephalitis and yellow, dengue, Punta Tora and Rift Valley fevers (Gabrielsen *et al*., 1992a,b).

Idso *et al*. determined that a 75% increase in the air's CO_2 concentration produced a 56% increase in the spider lily's belowground bulb biomass, where the disease-fighting substances are found. In addition, for these specific substances, they observed a 6% increase in the concentration of a two-constituent (1:1) mixture of 7-deoxynarciclasine and 7-deoxy-*trans*-dihydronarciclasine, an 8% increase in pancratistatin, an 8% increase in trans-dihydronarciclasine, and a 28% increase in narciclasine. Averaged together and combined with the 56% increase in bulb biomass, these percentage concentration increases resulted in a total mean active-ingredient increase of 75% for the plants grown in air containing 75% more CO_2.

4. Other Plant Constituents

A number of other plant constituents also perform important functions in maintaining human health, including sugars, lipids, oils and fatty acids, as well as macro- and micro-nutrients. Although concerns have been raised about the availability of certain of the latter elements in plants growing in a CO_2-enriched world (Loladze, 2002), the jury is still out with respect to this subject as a consequence of the paucity of pertinent data. Literally thousands of studies have assessed the impact of elevated levels of atmospheric CO_2 on the *quantity* of biomass produced by

agricultural crops, but only a tiny fraction of that number have looked at any aspect of food *quality*. From what has been learned about plant protein, antioxidants and the few medicinal substances that have been investigated in this regard, however, there is no reason to believe that these other plant constituents would be present in any *lower* concentrations in a CO_2-enriched world of the future than they are currently. Indeed, there is ample evidence to suggest they may well be present in significantly *greater* concentrations, and *certainly* in greater *absolute amounts*.

IV. A BRIEF HISTORY OF HUMAN LONGEVITY

The last 150-200 years have seen a significant degree of global warming, as the earth has recovered from the global chill of the Little Ice Age and entered the Modern Warm Period. Simultaneously, the planet has experienced a rise in its atmospheric CO_2 concentration that has taken it to levels not experienced for eons. What effects have these "twin evils" of the climate-alarmist crowd had on human health, as represented by perhaps the best integrative measure of their myriad possible influences, i.e., *human lifespan*?

Obviously, no one can give a precise quantitative answer to this question. Nevertheless, there are ways to assess the relative importance of the *wrongly-presumed negative health influences* of global warming and atmospheric CO_2 enrichment by considering the history of human longevity.

Tuljapurkar *et al*. (2000), for example, examined mortality over the period 1950-1994 in the G7 countries -- Canada, France, Germany (excluding the former East Germany), Italy, Japan, the United Kingdom, and the United States. The authors found that "in every country over this period, mortality at each age has declined exponentially at a roughly constant rate."

In discussing these findings, Horiuchi (2000) notes that the average lifespan of early humans was approximately 20 years, but that in the major industrialized countries it is now about 80 years, with the bulk of this increase having come in the past 150 years. He then notes that "it was widely expected that as life expectancy became very high and approached the 'biological limit of human longevity,' the rapid 'mortality decline' would slow down and eventually level off," but he states the now obvious fact that "such a deceleration has not occurred."

"These findings give rise to two interrelated questions," says Horiuchi: (1) "Why has mortality decline not started to slow down?" and (2) "Will it continue into the future?"

Some points to note in attempting to answer these questions are the following. First, in Horiuchi's words, "in the second half of the nineteenth century and the first half of the twentieth century, there were large decreases in the number of deaths from infectious and parasitic diseases, and from poor nutrition and disorders associated with pregnancy and childbirth," which led to large reductions in the deaths of infants, children and young adults. In the second half of the twentieth century, however, "mortality from degenerative diseases, most notably heart diseases and stroke, started to fall," and the reduction was most

pronounced among the elderly. Some suspected that this latter drop in mortality might have been achieved "through postponing the deaths of seriously ill people," he said; but data from the United States demonstrate that "the health of the elderly greatly improved in the 1980s and 1990s, suggesting that *the extended length of life in old age is mainly due to better health* [our italics] rather than prolonged survival in sickness."

Providing additional support for this conclusion is the study of Manton and Gu (2001). With the completion of the latest of the five National Long-Term Care Surveys of disability in U.S. citizens over 65 years of age -- which began in 1982 and extended to 1999 at the time of the writing of their paper -- these researchers were able to discern two most interesting trends: (1) disabilities in this age group decreased over the entire period studied, and (2) disabilities decreased at a rate that grew ever larger with the passing of time.

Specifically, over the entire 17-year period of record, there was an amazing relative decline in chronic disability of 25%, as the percentage of the over-65-years-of-age group that was disabled dropped from 26.2% in 1982 to 19.7% in 1999. What is more, the percentage disability decline rate per year for the periods 1982-1989, 1989-1994 and 1994-1999 was 0.26, 0.38 and 0.56% per year, respectively. Commenting on the ever-accelerating nature of this disability decline, the authors say "it is surprising, given the low level of disability in 1994, that the rate of improvement accelerated" over the most recent five-year interval.

Finally, Oeppen and Vaupel (2002) report that "world life expectancy more than doubled over the past two centuries, from roughly 25 years to about 65 for men and 70 for women." What is more, they note that "*for 160 years, best-performance life expectancy has steadily increased by a quarter of a year per year* [our italics]," and they emphasize that this phenomenal trend "is so extraordinarily linear that it may be the most remarkable regularity of mass endeavor ever observed." They also report there are no indications of the worldwide life-extension trend leveling off anytime soon.

To summarize to this point, it appears that *in countries with highly developed market economies*, such as the G7 nations, where good health care is readily available, deaths of infants, children and young adults have been dramatically reduced over the last century or so, to the point where average life expectancy is now largely determined by what happens to elderly people; and it is evident that under these circumstances, the elderly are living longer and longer with the passing of time. It is further evident that this phenomenon -- which is an observed empirical reality -- is likely due to ever-improving health in older

people, which in turn is likely the result of continuing improvements in their bodily systems for repairing cellular damage caused by degenerative processes associated with old age.

What is responsible for this incredible phenomenon? Nobody knows for sure. But what we do know for sure is that *it has operated unimpeded with unwavering consistency ever since the inception of the Industrial Revolution*, concomitant with simultaneous significant increases in both air temperature and atmospheric CO_2 concentration. Hence, we can confidently conclude that the “twin evils” of the climate-alarmist crowd have had not the slightest negative influence on this most welcome development. In fact, their general coherence in time with the 160-year linear increase in best-performance life expectancy almost leads one to suspect that one or both of them might even be partially *responsible* for the lengthening lifespan of mankind.

V. CONCLUSIONS

Although historical and projected future increases in the air's CO_2 concentration and its wrongly-predicted ability to lead to catastrophic global warming have been universally hailed by climate alarmists as diabolically detrimental to human health, scientific studies clearly demonstrate that such is not the case. Throughout the entire course of the Industrial Revolution, during which time the air's CO_2 content rose by 35% and its near-surface temperature by about 0.6°C, there has been no detectable negative impact on human longevity. In fact, human lifespan has concurrently experienced an almost unbelievable increase that shows no signs of ultimately leveling off or even slowing down. What is more, warming has been shown to *positively* impact human health, while atmospheric CO_2 enrichment has been shown to *enhance* the health-promoting properties of the food we eat, as well as stimulate the production of more of it. In addition, elevated levels of atmospheric CO_2 have been shown to increase the amounts and effectiveness of disease-fighting substances found in plants that protect against various forms of cancer, cardiovascular and respiratory diseases.

In light of these many well-documented observations, it is abundantly clear we have nothing to fear from increasing concentrations of atmospheric CO_2 and global warming, i.e., the "twin evils" of the extreme environmental movement. Indeed, these phenomena would appear to be *our friends* … and friends of the entire biosphere.

REFERENCES

Agrawal, M. and Deepak, S.S. 2003. Physiological and biochemical responses of two cultivars of wheat to elevated levels of CO_2 and SO_2, singly and in combination. *Environmental Pollution* 121: 189-197.

Alberdi, J.C., Diaz, J., Montero, J.C. and Miron, I. 1998. Daily mortality in Madrid community 1986-1992: relationship with meteorological variables. *European Journal of Epidemiology* 14: 571-578.

Barbale, D. 1970. The influence of the carbon dioxide on the yield and quality of cucumber and tomato in the covered areas. *Augsne un Raza (Riga)* **16**: 66-73.

Behar, S. 2000. Out-of-hospital death in Israel - Should we blame the weather? *Israel Medical Association Journal* **2**: 56-57.

Brackett, N.C., Jr., Wingo, C.F., Muren, O. and Solano, J.T. 1969. Acid-base response to chronic hypercapnia in man. *New England Journal of Medicine* **280**: 124-130.

Braga, A.L.F., Zanobetti, A. and Schwartz, J. 2002. The effect of weather on respiratory and cardiovascular deaths in 12 U.S. cities. *Environmental Health Perspectives* **110**: 859-863.

Bull, G.M. 1973. Meteorological correlates with myocardial and cerebral infarction and respiratory disease. *British Journal of Preventive and Social Medicine* **27**: 108.

Bull, G.M. and Morton, J. 1975a. Seasonal and short-term relationships of temperature with deaths from myocardial and cerebral infarction. *Age and Ageing* **4**: 19-31.

Bull, G.M. and Morton, J. 1975b. Relationships of temperature with death rates from all causes and from certain respiratory and arteriosclerotic diseases in different age groups. *Age and Ageing* **4**: 232-246.

Bull, G.M. and Morton, J. 1978. Environment, temperature and death rates. *Age and Ageing* **7**: 210-224.

Bunce, J.A. 2001. Seasonal patterns of photosynthetic response and acclimation to elevated carbon dioxide in field-grown strawberry. *Photosynthesis Research* **68**: 237-245.

Bushway, L.J. and Pritts, M.P. 2002. Enhancing early spring microclimate to increase carbon resources and productivity in June-bearing strawberry. *Journal of the American Society for Horticultural Science* **127**: 415-422.

Campbell, D.E. and Young, R. 1986. Short-term CO_2 exchange response to temperature, irradiance, and CO_2 concentration in strawberry. *Photosynthesis Research* **8**: 31-40.

Deng, X. and Woodward, F.I. 1998. The growth and yield responses of *Fragaria ananassa* to elevated CO_2 and N supply. *Annals of Botany* **81**: 67-71.

Dennison, B.A., Rockwell, H.L., Baker, S.L. 1998. Fruit and vegetable intake in young children. *J. Amer. Coll. Nutr.* **17**: 371-378.

Desjardins, Y., Gosselin, A. and Yelle, S. 1987. Acclimatization of ex vitro strawberry plantlets in CO_2-enriched environments and supplementary lighting. *Journal of the American Society for Horticultural Science* **112**: 846-851.

Dickinson, V.A., Block, G., Russek-Cohen, E. 1994. Supplement use, other dietary and demographic variables, and serum vitamin C in NHANES II. *J. Amer. Coll. Nutr.* **13**: 22-32.

Donaldson, G.C., Keatinge, W.R. and Nayha, S. 2003. Changes in summer temperature and heat-related mortality since 1971 in North Carolina, South Finland, and Southeast England. *Environmental Research* **91**: 1-7.

Dye, C. and Reiter, P. 2000. Temperatures without fevers? *Science* **289**: 1697-1698.

Eng, H. and Mercer, J.B. 1998. Seasonal variations in mortality caused by cardiovascular diseases in Norway and Ireland. *Journal of Cardiovascular Risk* **5**: 89-95.

Enquselassie, F., Dobson, A.J., Alexander, H.M. and Steele, P.L. 1993. Seasons, temperature and coronary disease. *International Journal of Epidemiology* **22**: 632-636.

Estrada-Peña, A. 2003. Climate change decreases habitat suitability for some tick species (Acari: Ixodidae) in South Africa. *Onderstepoort Journal of Veterinary Research* **70**: 79-93.

Evans, L.T. 1993. *Crop Evolution, Adaptation and Yield*. Cambridge University Press, Cambridge, UK.

Feigin, V.L., Nikitin, Yu.P., Bots, M.L., Vinogradova, T.E. and Grobbee, D.E. 2000. A population-based study of the associations of stroke occurrence with weather parameters in Siberia, Russia (1982-92). *European Journal of Neurology* **7**: 171-178.

Gabrielsen, B., Monath, T.P., Huggins, J.W., Kefauver, D.F., Pettit, G.R., Groszek, G., Hollingshead, M., Kirsi, J.J., Shannon, W.F., Schubert, E.M., Dare, J., Ugarkar, B., Ussery, M.A., Phelan, M.J. 1992a. Antiviral (RNA) activity of selected Amaryllidaceae isoquinoline constituents and synthesis of related substances. *Journal of Natural Products* **55**: 1569-1581.

Gabrielsen, B., Monath, T.P., Huggins, J.W., Kirsi, J.J., Hollingshead, M., Shannon, W.M., Pettit, G.R. 1992b. Activity of selected Amaryllidaceae constituents and related synthetic substances against medically important RNA viruses. In: Chu, C.K. and Cutler, H.G. (Eds.), *Natural Products as Antiviral Agents*. Plenum Press, New York, NY, pp. 121-35.

Goklany, I.M. and Straja, S.R. 2000. U.S. trends in crude death rates due to extreme heat and cold ascribed to weather, 1979-97. *Technology* **7S**: 165-173.

Gomez-Carrasco, J.A., Cid, J.L.-H., de Frutos, C.B., Ripalda-Crespo, M.J., de Frias, J.E.G. 1994. Scurvy in adolescence. *J. Pediatr. Gastroenterol. Nutr.* **19**: 118-120.

Gouveia, N., Hajat, S. and Armstrong, B. 2003. Socioeconomic differentials in the temperature-mortality relationship in Sao Paulo, Brazil. *International Journal of Epidemiology* **32**: 390-397.

Green, M.S., Harari, G., Kristal-Boneh, E. 1994. Excess winter mortality from ischaemic heart disease and stroke during colder and warmer years in Israel. *European Journal of Public Health* **4**: 3-11.

Hajat, S. and Haines, A. 2002. Associations of cold temperatures with GP consultations for respiratory and cardiovascular disease amongst the elderly in London. *International Journal of Epidemiology* **31**: 825-830.

Hampl, J.S., Taylor, C.A., Johnston, C.S. 1999. Intakes of vitamin C, vegetables and fruits: Which schoolchildren are at risk? *J. Amer. Coll. Nutr.* **18**: 582-590.

Hay, S.I., Cox, J., Rogers, D.J., Randolph, S.E., Stern, D.I., Shanks, G.D., Myers, M.F. and Snow, R.W. 2002. Climate change and the resurgence of malaria in the East African highlands. *Nature* **415**: 905-909.

Heagle, A.S., Miller, J.E. and Pursley, W.A. 1998. Influence of ozone stress on soybean response to carbon dioxide enrichment: III. Yield and seed quality. *Crop Science* **38**: 128-134.

Heinonen, I.M., Meyer, A.S. and Frankel, E.N. 1998. Antioxidant activity of berry phenolics on human low-density lipoprotein and liposome oxidation. *Journal of Agricultural and Food Chemistry* **46**: 4107-4112.

Hong, Y-C., Rha, J-H., Lee, J-T., Ha, E-H., Kwon, H-J. and Kim, H. 2003. Ischemic stroke associated with decrease in temperature. *Epidemiology* **14**: 473-478.

Horiuchi, S. 2000. Greater lifetime expectations. *Nature* **405**: 744-745.

Huynen, M.M.T.E., Martens, P., Schram, D., Weijenberg, M.P. and Kunst, A.E. 2001. The impact of heat waves and cold spells on mortality rates in the Dutch population. *Environmental Health Perspectives* **109**: 463-470.

Idso, C.D. and Idso, K.E. 2000. Forecasting world food supplies: The impact of the rising atmospheric CO2 concentration. *Technology* **7S**: 33-55.

Idso, C.D., Idso, S.B. and Balling Jr., R.C. 1998. The urban CO_2 dome of Phoenix, Arizona. *Physical Geography* **19**: 95-108.

Idso, S.B. 1997. The poor man's biosphere, including simple techniques for conducting CO_2 enrichment and depletion experiments on aquatic and terrestrial plants. *Environmental and Experimental Botany* **38**: 15-38.

Idso, S.B., Idso, C.D. and Balling Jr., R.C. 2002. Seasonal and diurnal variations of near-surface atmospheric CO_2 concentrations within a residential sector of the urban CO_2 dome of Phoenix, AZ, USA. *Atmospheric Environment* **36**: 1655-1660.

Idso, S.B., Idso, C.D. and Idso, K.E. 2003. *The Specter of Species Extinction: Will Global Warming Decimate Earth's Biosphere?* George C. Marshall Institute, Washington, DC.

Idso, S.B. and Idso, K.E. 2001. Effects of atmospheric CO_2 enrichment on plant constituents related to animal and human health. *Environmental and Experimental Botany* **45**: 179-199.

Idso, S.B., Kimball, B.A., Pettit III, G.R., Garner, L.C., Pettit, G.R., Backhaus, R.A. 2000. Effects of atmospheric CO_2 enrichment on the growth and development of *Hymenocallis littoralis* (Amaryllidaceae) and the concentrations of several antineoplastic and antiviral constituents of its bulbs. *American Journal of Botany* **87**: 769-773.

Idso, S.B., Kimball, B.A., Shaw, P.E., Widmer, W., Vanderslice, J.T., Higgs, D.J., Montanari, A. and Clark, W.D. 2002. The effect of elevated atmospheric CO_2 on the vitamin C concentration of (sour) orange juice. *Agriculture, Ecosystems and Environment* **90**: 1-7.

Jablonski, L.M., Wang, X. and Curtis, P.S. 2002. Plant reproduction under elevated CO_2 conditions: a meta-analysis of reports on 79 crop and wild species. *New Phytologist* **156**: 9-26.

Johnston, C.S., Solomon, R.E., Corte, C. 1998. Vitamin C status of a campus population: College students get a C minus. *J. Amer. Coll. Health* **46**: 209-213.

Kan, H-D., Jia, J. and Chen, B-H. 2003. Temperature and daily mortality in Shanghai: A time-series study. *Biomedical and Environmental Sciences* **16**: 133-139.

Keatinge, W.R. and Donaldson, G.C. 2001. Mortality related to cold and air pollution in London after allowance for effects of associated weather patterns. *Environmental Research* **86A**: 209-216.

Keatinge, W.R., Donaldson, G.C., Cordioli, E., Martinelli, M., Kunst, A.E., Mackenbach, J.P., Nayha, S. and Vuori, I. 2000. Heat related mortality in warm and cold regions of Europe: Observational study. *British Medical Journal* **321**: 670-673.

Keutgen, N., Chen, K. and Lenz, F. 1997. Responses of strawberry leaf photosynthesis, chlorophyll fluorescence and macronutrient contents to elevated CO_2. *Journal of Plant Physiology* **150**: 395-400.

Kimball, B.A., Mitchell, S.T. 1981. Effects of CO_2 enrichment, ventilation, and nutrient concentration on the flavor and vitamin C content of tomato fruit. *HortScience* **16**: 665-666.

Kimball, B.A., Morris, C.F., Pinter Jr., P.J., Wall, G.W., Hunsaker, D.J., Adamsen, F.J., LaMorte, R.L., Leavitt, S.W., Thompson, T.L., Matthias, A.D., Brooks, T.J. 2001. Wheat grain quality as affected by elevated CO_2, drought, and soil nitrogen. *New Phytologist* **150**: 295-303.

Kloner, R.A., Poole, W.K. and Perritt, R.L. 1999. When throughout the year is coronary death most likely to occur? A 12-year population-based analysis of more than 220,000 cases. *Circulation* **100**: 1630-1634.

Kuhn, K.G., Campbell-Lendrum, D.H., Armstrong, B. and Davies, C.R. 2003. Malaria in Britain: Past, present, and future. *Proceedings of the National Academy of Science, USA* **100**: 9997-10001.

Kunst, A.E., Looman, W.N.C. and Mackenbach, J.P. 1993. Outdoor temperature and mortality in the Netherlands: a time-series analysis. *American Journal of Epidemiology* **137**: 331-341.

Laschewski, G. and Jendritzky, G. 2002. Effects of the thermal environment on human health: an investigation of 30 years of daily mortality data from SW Germany. *Climate Research* **21**: 91-103.

Loladze, I. 2002. Rising atmospheric CO_2 and human nutrition: toward globally imbalanced plant stoichiometry? *Trends in Ecology & Evolution* **17**: 457-461.

Luft, U.C., Finkelstein, S. and Elliot, J.C. 1974. Respiratory gas exchange, acid-base balance, and electrolytes during and after maximal work breathing 15 mm Hg $PICO_2$. In: *Carbon Dioxide and Metabolic Regulations*. G. Nahas and K.E. Schaefer (Eds.). Springer-Veriag, New York, NY, pp. 282-293.

Machlin, L.G. 1992. Introduction. In: Sauberlich, H.E. and Machlin, L.J. (Eds.), Beyond deficiency: New views on the function and health effects of vitamins. *Annals of the New York Academy of Science* **669**: 1-6.

Madsen, E. 1971. The influence of CO_2-concentration on the content of ascorbic acid in tomato leaves. *Ugeskr. Agron.* **116**: 592-594.

Madsen, E. 1975. Effect of CO_2 environment on growth, development, fruit production and fruit quality of tomato from a physiological viewpoint. In: P. Chouard, N. de Bilderling (Eds.), *Phytotronics in Agricultural and Horticultural Research*. Bordas, Paris, pp. 318-330.

Manton, K.G. and Gu, XL. 2001. Changes in the prevalence of chronic disability in the United States black and nonblack population above age 65 from 1982 to 1999. *Proceedings of the National Academy of Science, USA* **98**: 6354-6359.

Martens, P. and Huynen, M. 2001. Will global climate change reduce thermal stress in the Netherlands? *Epidemiology* **12**: 753-754.

Mayeux, H.S., Johnson, H.B., Polley, H.W. and Malone, S.R. 1997. Yield of wheat across a subambient carbon dioxide gradient. *Global Change Biology* **3**: 269-278.

Nafstad, P., Skrondal, A. and Bjertness, E. 2001. Mortality and temperature in Oslo, Norway. 1990-1995. *European Journal of Epidemiology* **17**: 621-627.

Nahas, G., Poyart, C. and Triner, L. 1968. Acid base equilibrium changes and metabolic alterations. *Annals of the New York Academy of Science.* **150**: 562-576.

Oeppen, J. and Vaupel, J.W. 2002. Broken limits to life expectancy. *Science* **296**: 1029-1030.

Pascual, M., Bouma, M.J. and Dobson, A.P. 2002. Cholera and climate: revisiting the quantitative evidence. *Microbes and Infection* **4**: 237-245.

Pettit, G.R., Gaddamidi, V., Herald, D.L., Singh, S.B., Cragg, G.M., Schmidt, J.M., Boettner, F.E., Williams, M., Sagawa, Y. 1986. Antineoplastic agents, 120. *Pancratium littorale*. *Journal of Natural Products* **49**: 995-1002.

Pettit, G.R., Pettit III, G.R., Backhaus, R.A., Boyd, M.R., Meerow, A.W. 1993. Antineoplastic agents, 256. Cell growth inhibitory isocarbostyrils from *Hymenocallis*. *Journal of Natural Products* **56**: 1682-1687.

Pettit, G.R., Pettit III, G.R., Groszek, G., Backhaus, R.A., Doubek, D.L., Barr, R.J. 1995. Antineoplastic agents, 301. An investigation of the Amaryllidaceae genus *Hymenocallis*. *Journal of Natural Products* **58**: 756-759.

Poyart, C.F. and Nahas, G. 1968. Inhibition of activated lipolysis by acidosis. *Molecular Pharmacol.* **4**: 389-401.

Pleijel, H., Mortensen, L., Fuhrer, J., Ojanpera, K., Danielsson, H. 1999. Grain protein accumulation in relation to grain yield of spring wheat (*Triticum aestivum* L.) grown in open-top chambers with different concentrations of ozone, carbon dioxide and water availability. *Agriculture, Ecosystems and Environment* **72**: 265-270.

Ramar, S., Sivaramakrishman, V., Manoharan, K. 1993. Scurvy – a forgotten disease. *Arch. Phys. Med. Rehabil*. **74**: 92-95.

Randolph, S.E. and Rogers, D.J. 2000. Fragile transmission cycles of tick-borne encephalitis virus may be disrupted by predicted climate change. *Proceedings of the Royal Society of London Series B* **267**: 1741-1744.

Reiter, P. 2000. From Shakespeare to Defoe: Malaria in England in the Little Ice Age. *Emerging Infectious Diseases* **6**: 1-11.

Reiter, P. 2001. Climate change and mosquito-borne disease. *Environmental Health Perspectives* **109**: 141-161.

Rice-Evans, C.A. and Miller, N.J. 1996. Antioxidant activities of flavonoids as bioactive components of food. *Biochemical Society Transactions* **24**: 790-795.

Robeson, S.M. 2002. Relationships between mean and standard deviation of air temperature: implications for global warming. *Climate Research* **22**: 205-213.

Rogers, D.J. and Randolph, S.E. 2000. The global spread of malaria in a future, warmer world. *Science* **289**: 1763-1766.

Rogers, G.S., Milham, P.J., Gillings, M. and Conroy, J.P. 1996. Sink strength may be the key to growth and nitrogen responses in N-deficient wheat at elevated CO_2. *Australian Journal of Plant Physiology* **23**: 253-264.

Rooney, C., McMichael, A.J., Kovats, R.S. and Coleman, M.P. 1998. Excess mortality in England and Wales, and in greater London, during the 1995 heat wave. *Journal of Epidemiology and Community Health* **52**: 482-486.

Rudorff, B.F.T., Mulchi, C.L., Fenny, P., Lee, E.H., Rowland, R. 1996. Wheat grain quality under enhanced tropospheric CO_2 and O_3 concentrations. *Journal of Environmental Quality* **25**: 1384-1388.

Schaefer, K.E. 1982. Effects of increased ambient CO_2 levels on human and animal health. *Experientia* **38**: 1163-1168.

Shanks, G.D., Biomndo, K., Hay, S.I. and Snow, R.W. 2000. Changing patterns of clinical malaria since 1965 among a tea estate population located in the Kenyan highlands. *Transactions of the Royal Society of Tropical Medicine and Hygiene* **94**: 253-255.

Stuhlfauth, T., Fock, H.P. 1990. Effect of whole season CO_2 enrichment on the cultivation of a medicinal plant, *Digitalis lanata*. *Journal of Agronomy and Crop Science* **164**: 168-173.

Stuhlfauth, T., Klug, K. and Fock, H.P. 1987. The production of secondary metabolites by *Digitalis lanata* during CO_2 enrichment and water stress. *Phytochemistry* **26**: 2735-2739.

Tajiri, T. 1985. Improvement of bean sprouts production by intermittent treatment with carbon dioxide. *Nippon Shokuhin Kogyo Gakkaishi* **32**(3): 159-169.

Thomas, J.M.G., Boote, K.J., Allen Jr., L.H., Gallo-Meagher, M. and Davis, J.M. 2003. Elevated temperature and carbon dioxide effects on soybean seed composition and transcript abundance. *Crop Science* **43**: 1548-1557.

Tilman, D., Fargione, J., Wolff, B., D'Antonio, C., Dobson, A., Howarth, R., Schindler, D., Schlesinger, W.H., Simberloff, D. and Swackhamer, D. 2001. Forecasting agriculturally driven global environmental change. *Science* **292**: 281-284.

Tuljapurkar, S., Li, N. and Boe, C. 2000. A universal pattern of mortality decline in the G7 countries. *Nature* **405**: 789-792.

Turino, G.M., Goldring, R.M. and Heinemann, H.O. 1974. The extracellular bicarbonate concentration and the regulation of ventilation in chronic hypercapnia in man. In: *Carbon Dioxide and Metabolic Regulations*. G. Nahas and K.E. Schaefer (Eds.). Springer-Verlag, New York, NY, pp. 273 - 281.

Van Ypersele de Strihou, C. 1974. Acid-base equilibrium in chronic hypercapnia. In: *Carbon Dioxide and Metabolic Regulations*. G. Nahas and K.E. Schaefer (Eds.). Springer-Verlag, New York, NY, pp. 266.

Wang, S.Y., Bunce, J.A. and Maas, J.L. 2003. Elevated carbon dioxide increases contents of antioxidant compounds in field-grown strawberries. *Journal of Agricultural and Food Chemistry* **51**: 4315-4320.

Wang, H., Cao, G. and Prior, R.L. 1996. Total antioxidant capacity of fruits. *Journal of Agricultural and Food Chemistry* **44**: 701-705.

Wang, S.Y. and Jiao, H. 2000. Scavenging capacity of berry crops on superoxide radicals, hydrogen peroxide, hydroxyl radicals, and singlet oxygen. *Journal of Agricultural and Food Chemistry* **48**: 5677-5684.

Wang, S.Y. and Lin, H.S. 2000. Antioxidant activity in fruit and leaves of blackberry, raspberry, and strawberry is affected by cultivar and maturity. *Journal of Agricultural and Food Chemistry* **48**: 140-146.

Wittwer, S.H. 1995. Food, Climate, and Carbon Dioxide: The Global Environment and World Food Production. CRC Press, Boca Raton, FL.

INDEX

Human Health in a CO_2-Enriched Warmer World

CENTER FOR THE STUDY OF
CARBON DIOXIDE AND GLOBAL CHANGE

P.O. BOX 25697
TEMPE, AZ 85285-5697, USA

TELEPHONE
480-966-3719
FAX
480-966-0758
EMAIL
INFO@CO2SCIENCE.ORG
WEBSITE
WWW.CO2SCIENCE.ORG

NOVEMBER 2003