

9. HUMAN HEALTH

The idea that CO₂-induced global warming is responsible for increases in a host of human maladies has become incorrectly entrenched in popular culture. Hardly a heat wave passes, for example, but what many are quick to blame global warming for any excess deaths that may have been associated with it. However, if the truth were told, and all of the scientific literature were justly examined, one would find that global warming will 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. What is more research has shown that there are also a number of *positive* effects of atmospheric CO₂ enrichment that tend to *enhance* people's quality of life, but about which little is spoken. In this next chapter we investigate such phenomena, beginning with a discussion on temperature-induced mortality.

9.1. Temperature-Induced Mortality

Which is more deadly ... *heat* or *cold*? ... *rising* temperatures or *falling* temperatures? The world's climate alarmists say that *warming* is the primary danger ... and that it should be avoided at essentially *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.

Additional information on this topic, including reviews on the health effects of CO₂ not discussed here, can be found at http://www.co2science.org/subject/h/subject_h.php under the heading Health Effects

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9.1.1. 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, and several others for Australia (Enquselassie *et al.*, 1993), Brazil (Sharovsky *et al.*, 2004), England (McGregor, 2005; Carder *et al.*, 2005; McGregor *et al.*, 2004; Kovats *et al.*, 2004;), Greece (Bartzokas *et al.*, 2004), Japan (Nakaji *et al.*, 2004), the United States (Cagle and Hubbard, 2005), and parts of Africa, Asia, Europe, Latin America and the Caribbean (Chang *et al.*, 2004), clearly demonstrate that global warming is actually *beneficial* to humanity, in that it reduces the incidence of cardiovascular disease related to low temperatures and wintry weather by a much greater degree than it increases the incidence of cardiovascular disease associated with high temperatures and summer heat waves.

Additional information on this topic, including reviews of newer publications as they become available, can be found at <http://www.co2science.org/subject/h/healtheffectscardio.php>.

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9.1.2. 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.

In another study, Donaldson (2006) studied the effect of annual mean daily air temperature on the length of the yearly RSV season in England and Wales for 1981-2004. Results indicated that "the seasons associated with laboratory isolation of respiratory syncytial virus (for 1981-2004) and RSV-related emergency department admissions (for 1990-2004) ended 3.1 and 2.5 weeks earlier, respectively, per 1°C increase in annual central England temperature ($P = 0.002$ and 0.043 , respectively)." Consequently, since "no relationship was observed between the start of each season and temperature," he reports that "the RSV season has become shorter." The implications of Donaldson's results are rather obvious. As he describes them, "these findings imply a health benefit of global warming in England and Wales associated with a reduction in the duration of the RSV season and its consequent impact on the health service."

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.

Additional information on this topic, including reviews of newer publications as they become available, can be found at <http://www.co2science.org/subject/h/healtheffectsresp.php>.

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9.1.3. Vector-Borne Diseases

In a research report in *Science*, Rogers and Randolph (2000) note what is probably well known to all, namely, 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." Indeed, such predictions comprise one of the major global-warming scare-stories of the world's climate alarmists. There are, however, several problems with this scenario.

According to Reiter (2000), claims that malaria resurgence is the product of CO₂-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 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."

Given such events, why was malaria so prevalent in Europe during some of the coldest centuries of the past millennium .. and why we have only recently 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. *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, use of insecticides, and the movement of people (Reiter, 2000; Reiter, 2001; Hay *et al.*, 2002).

Why, then, does the TSD and CSDs predict widespread future increases in malaria? They do it because nearly all of the analyses they cite have typically used only one, or at most two, climate variables to make predictions of the future distribution of the disease over the earth; and they generally do not include any of the non-climatic factors listed in the paragraph above. In one modeling study, for example, Rogers and Randolph (2000) employed a total of *five* climate variables and obtained very different results. Briefly, they used the present-day distribution of malaria to determine the specific climatic constraints that best define that distribution, after which the multivariate relationship they derived from this exercise was applied to future climate scenarios derived from state-of-the-art climate models, in order to map potential future geographical distributions of the disease.

The results of their study revealed very little change: a 0.84% increase in potential malaria exposure under the "medium-high" scenario of global warming and a 0.92% *decrease* under the "high" scenario. In consequence of these findings, Rogers and Randolph explicitly state that their quantitative 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."

Clearly, this study undercuts the climate-alarmist claim that any future warming of the globe will allow malaria to spread into current malaria-free regions, as do the studies of Hay *et al.* (2002) and Shanks *et al.* (2000). The first of these research groups 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. Likewise, Shanks *et al.* examined trends in temperature, precipitation and malaria rates in western Kenya over the period 1965-1997, finding absolutely no linkages among the variables.

Also working in Africa, Small *et al.* (2003) examined trends in a climate-driven model of malaria transmission between 1911 and 1995, using a spatially and temporally extensive gridded climate data-set to identify locations where the malaria transmission *climate suitability index*

had changed significantly over this time interval. Then, after determining areas of change, they more closely examined the underlying climate forcing of malaria transmission suitability for those localities. This protocol revealed that malaria transmission suitability did indeed increase because of climate change in specific locations of limited extent; but in Southern Mozambique, which was the only region for which climatic suitability consistently increased, the cause of the increase was increased *precipitation*, not temperature. In fact, Small *et al.* say that "climate warming, expressed as a systematic temperature increase over the 85-year period, does not appear to be responsible for an increase in malaria suitability over *any* [our italics] region in Africa." Hence, they concluded that "research on the links between climate change and the recent resurgence of malaria across Africa would be best served through refinements in maps and models of precipitation patterns and through closer examination of the role of nonclimatic influences," the great significance of which has recently been demonstrated by Reiter *et al.* (2003) for dengue fever, another important mosquito-borne disease.

Further examining the reemergence of malaria in the East African highlands were Zhou *et al.* (2004), who addressed the issue via a nonlinear mixed-regression model study that focused on the numbers of monthly malaria outpatients of the past 10-20 years in seven East African highland sites and their relationships to the numbers of malaria outpatients during the previous time period, seasonality and climate variability. In doing so, they say that "for all seven study sites, we found highly significant nonlinear, synergistic effects of the interaction between rainfall and temperature on malaria incidence, indicating that *the use of either temperature or rainfall alone is not sensitive enough for the detection of anomalies that are associated with malaria epidemics* [our italics]," as has also been found by Githeko and Ndegwa (2001), Shanks *et al.* (2002) and Hay *et al.* (2002). What is more, *climate variability* -- not just *temperature* or not just *warming* -- contributed less than 20% of the temporal variance in the number of malaria outpatients, and at only two out of the seven sites studied.

In light of their findings, Zhou *et al.* concluded that "malaria dynamics are largely driven by autoregression and/or seasonality in these sites," and that "the observed large among-site variation in the sensitivity to climate fluctuations may be governed by complex interactions between climate and biological and social factors," including "land use, topography, *P. falciparum* genotypes, malaria vector species composition, availability of vector control and healthcare programs, drug resistance, and other socioeconomic factors," among which are "failure to seek treatment or delayed treatment of malaria patients, and HIV infections in the human population," which they say have "become increasingly prevalent." Hence, it would appear that the so-called *unprecedented* global warming of the past century or so, which is claimed in the TSD abd CSDs to have significantly accelerated over the past couple of decades, should be the *least* of our worries with respect to this subject ... *or* that the claimed acceleration of warming is more imagined than real.

Prefacing another revealing study, Kuhn *et al.* (2003) say "there has been much recent speculation that global warming may allow the reestablishment of malaria transmission in previously endemic areas such as Europe and the United States." In particular, they note that "the British Chief Medical Officer's recent report [*Getting Ahead of the Curve: A Strategy for*

Combating Infectious Diseases (Including Other Aspects of Health Protection), Department of Health (2002), London] asserted that 'by 2050 the climate of the UK may be such that indigenous malaria could become re-established'," which is the same mantra that is incessantly chanted by the world's climate alarmists. Consequently, to investigate the robustness of this hypothesis, they 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, this analysis indicated 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 explains "the malaria epidemics in the 'unusually hot summers' of 1848 and 1859." Nevertheless, the long-term near-linear temporal decline in malaria deaths over the period of study, in the words of the researchers, "was probably driven by nonclimatic factors," among which they list 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." They additionally note that the number of secondary 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.

Although simplistic model simulations may suggest that the increase in temperature predicted for Britain by 2050 is likely to cause an 8-14% increase in the *potential* for malaria transmission, Kuhn *et al.* say "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.

In the introduction to his review about what was known to this point in time about the putative link between global warming and the spread of infectious diseases, Zell (2004) stated that many people "assume a correlation between increasing disease incidence and global warming." However, as he concluded after studying the issue in considerable depth, "the factors responsible for the emergence/reemergence of vector-borne diseases are complex and mutually influence each other," citing as an example of this complexity the fact that "the incidence and spread of parasites and arboviruses are affected by insecticide and drug resistance, deforestation, irrigation systems and dams, changes in public health policy (decreased resources of surveillance, prevention and vector control), demographic changes (population growth, migration, urbanization), and societal changes (inadequate housing conditions, water deterioration, sewage, waste management)." Therefore, as he continues, "it may be over-simplistic to attribute emergent/re-emergent diseases to climate change and sketch the menace of devastating epidemics in a warmer world," such as Al Gore does in *An Inconvenient Truth*. Indeed, Zell states that "variations in public health practices and lifestyle

can easily [our italics] outweigh changes in disease biology," especially those that might be caused by global warming.

In a rather different type of study, but one that is extremely pertinent, Tuchman *et al.* (2003) (1) took leaf litter from *Populus tremuloides* (Michaux) trees that had been grown out-of-doors in open-bottom root boxes located within open-top aboveground chambers maintained at atmospheric CO₂ concentrations of either 360 or 720 ppm for an entire growing season, (2) incubated the leaf litter for 14 days in a nearby stream, and (3) fed the incubated litter to four species of detritivorous mosquito larvae to assess its effect on their development rates and survivorship. This work revealed that larval mortality was 2.2 times higher for *Aedes albopictus* (Skuse) mosquitos that were fed leaf litter that had been produced in the high-CO₂ chambers than it was for those fed litter that had been produced in the ambient-air chambers. In addition, they found that larval development rates of *Aedes triseriatus* (Say), *Aedes aegypti* (L.) and *Armigeres subalbatus* (Coquillett) were slowed by 78%, 25% and 27%, respectively, when fed litter produced in the high-CO₂ as opposed to the ambient-CO₂ chambers, so that mosquitoes of these species spent 20, 11 and 9 days longer in their respective larval stages when feeding on litter produced in the CO₂-enriched as compared to the ambient-CO₂ chambers. As for the reason behind these observations, the researchers suggest that "increases in lignin coupled with decreases in leaf nitrogen induced by elevated CO₂ and subsequent lower bacterial productivity [on the leaf litter in the water] were probably responsible for [the] decreases in survivorship and/or development rate of the four species of mosquitoes."

What is the significance of these findings?

In the words of Tuchman *et al.*, "the indirect impacts of an elevated CO₂ atmosphere on mosquito larval survivorship and development time could potentially be great," because longer larval development times could result in fewer cohorts of mosquitoes surviving to adulthood; and with fewer mosquitoes around, there should be lower levels of mosquito-born diseases, for which blessing we would have the ongoing rise in the atmosphere's CO₂ concentration to thank.

In another major review of the potential impacts of global warming on vector-borne diseases, Rogers and Randolph (2006) focus on recent upsurges of malaria in Africa, asking the question "Has climate change already had an impact?" They go on to demonstrate that "evidence for increasing malaria in many parts of Africa is overwhelming, but the more likely causes for most of these changes to date include land-cover and land-use changes and, most importantly, drug resistance rather than any effect of climate," noting that "the recrudescence of malaria in the tea estates near Kericho, Kenya, in East Africa, where temperature has not changed significantly [our italics], shows all the signs of a disease that has escaped drug control following the evolution of chloroquine resistance by the malarial parasite." They then go on to explain that "malaria waxes and wanes to the beat of two rhythms: an annual one dominated by local, seasonal weather conditions and a ca. 3-yearly one dominated by herd immunity," noting that "effective drugs suppress both cycles before they can be expressed," but that "this produces a population which is mainly or entirely dependent on drug effectiveness, and which suffers the

consequence of eventual drug failure, during which the rhythms reestablish themselves, as they appear to have done in Kericho."

Last of all, Childs *et al.* (2006) present a detailed analysis of malaria incidence in northern Thailand based on a quarter-century monthly time series (January 1977 through January 2002) of total malaria cases in the country's 13 northern provinces. Over this time period, when climate alarmists claim the world warmed at a rate and to a level that were *unprecedented over the prior two millennia*, they report there was an approximately constant rate of *decline* in total malaria incidence (from a mean monthly incidence in 1977 of 41.5 cases per hundred thousand people to 6.72 cases per hundred thousand people in 2001), due primarily to a reduction in cases positive for *Plasmodium falciparum* (mean monthly incidence in 1977 and 2001 of 28.6 and 3.22 cases per 100,000 people, respectively) and secondarily to a reduction in cases positive for *P. vivax* (mean monthly incidence in 1977 and 2001 of 12.8 and 3.5 cases per 100,000 people, respectively). Consequently, noting that "there has been a steady reduction through time of total malaria incidence in northern Thailand, with an average decline of 6.45% per year," they say this result "reflects changing agronomic practices and patterns of immigration, as well as the success of interventions such as vector control programs, improved availability of treatment and changing drug policies."

In light of the many findings described above, the claim that malaria will expand across the globe and intensify as a result of *CO₂-induced warming* is seen to be basically bogus. 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," independent of whatever tack earth's climate might take in the interim.

Additional information on this topic, including reviews of newer publications as they become available, can be found at <http://www.co2science.org/subject/m/malaria.php>, <http://www.co2science.org/subject/h/healtheffectsdengue.php>, and <http://www.co2science.org/subject/h/healtheffectsyellfev.php>.

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9.1.4. Tick-Borne Diseases

Similar to vector-borne diseases, climate alarmists also conclude that one of the likely consequences of the increase in temperature predicted to be produced by anthropogenic CO₂ emissions would be expanded geographic ranges of *tick-borne diseases*. We here consider the reliability of this claim as illuminated by some studies of the subject we have reviewed on our website (<http://www.co2science.org/subject/h/summaries/healtheffectstick.php>).

We begin with the study of Randolph and Rogers (2000), who report that tick-borne encephalitis (TBE) "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 disease is caused by a flavivirus (TBEV), which is maintained in natural rodent-tick cycles; and humans may be infected with it if bitten by an infected tick or by drinking untreated milk from infected sheep or goats.

Early writings on the relationship of TBE to global warming predicted it would expand its range and become more of a threat to humans in a warmer world. However, Randolph and Rogers indicate 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," so that "their continuing survival or expansion cannot be predicted from simple univariate correlations," as is commonly done by climate alarmists intent on scaring people into reducing CO₂ emissions on the basis of false premises. Hence, the two researchers decided to explore the subject in 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 distributions of five climatic variables: monthly mean, maximum and minimum temperatures, plus rainfall and saturation vapor pressure, "to provide a multivariate description of present-day areas of disease risk." Then, they 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 these operations indicated that the distribution of TBEV might expand both north and west of Stockholm, Sweden, in a warming world. For most other parts of Europe, however, the two researchers say that "fears for increased extent of risk from TBEV caused by global climate change appear to be unfounded." In fact, they found that "the precise conditions required for enzootic cycles of TBEV are predicted to be disrupted" in response to global warming, and that the new climatic state "appears to be lethal for TBEV." This finding, in their 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 report that the predicted change "appears to be to our advantage."

Also reporting 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," Estrada-Peña (2003) evaluated 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 revealed 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, Estrada-Peña found that "the simulations show a trend toward the destruction of the habitats of the four tick species," which is just the *opposite* of what is predicted by the TSD and CRDs.

Another scientist who has noted that many people "assume a correlation between increasing disease incidence and global warming" is Zell (2004), who reviewed the scientific literature pertaining to the subject and determined that "the factors responsible for the emergence/reemergence of vector-borne diseases are complex and mutually influence each other," citing as an example of this complexity the fact that "the incidence and spread of parasites and arboviruses are affected by insecticide and drug resistance, deforestation, irrigation systems and dams, changes in public health policy (decreased resources of surveillance, prevention and vector control), demographic changes (population growth, migration, urbanization), and societal changes (inadequate housing conditions, water deterioration, sewage, waste management)."

In light of these many complicating factors, Zell says "it may be over-simplistic to attribute emergent/re-emergent diseases to climate change and sketch the menace of devastating epidemics in a warmer world." Indeed, he concludes that "variations in public health practices and lifestyle can easily outweigh changes in disease biology," especially those that might be caused by global warming. What is more, these public health and lifestyle changes could be implemented *now*, if we chose to do so, and at only a tiny *fraction* of the cost that would be needed to make even the *smallest* of changes in the future course of earth's air temperature. If we are truly worried about the status of vector-borne diseases in a warmer world, this is the tack we should take.

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9.1.5. 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 (excluding 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 between 1971 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 CO₂ 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 was 10 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 who claim to be concerned about the health effects of climate change are being dishonest when they say that CO₂-induced global warming is killing people. In point of fact, CO₂-induced global warming -- if it is even occurring at all -- is enabling earth's populace to actually lead both *longer* and *more productive* lives.

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9.2. Non-Climatic Health Effects of Elevated CO₂

Even if atmospheric CO₂ 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 for the EPA to consider *other* potential health effects of atmospheric CO₂ enrichment that are *not* related to climate in order to determine the *net* effect of elevated levels of atmospheric CO₂ on human health and longevity. Hence, we next explore several aspects of this subject about which little or nothing at all is said -- *positive* effects of atmospheric CO₂ enrichment that tend to *enhance* people's quality of life.

9.2.1. Direct Health Effects of Elevated CO₂

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?

As correctly pointed out in the EPA's Technical Support Document, inhaling very high concentrations of atmospheric CO₂ 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 CO₂ 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 CO₂ 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 CO₂ 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* CO₂ concentration of the air in many homes and buildings is often two to three times greater than the CO₂ concentration of outdoor air (Idso, 1997), which in large cities is itself often elevated by several tens of percent above the CO₂ concentration of rural air (Idso *et al.* 1998, 2002).

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9.2.2. Indirect Health Effects of Elevated CO₂

The effects of atmospheric CO₂ 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 CO₂ content. Here, we begin our investigation of this subject with a brief review of the well-known *aerial fertilization effect* of atmospheric CO₂ enrichment and how it impacts the human health issue.

9.2.2.1. Food Production

First and foremost, people must have *sufficient* food, simply to sustain themselves; and the rise in the atmosphere's CO₂ 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.

9.2.2.1.1. THE PAST

In a revealing study of the beneficial impact of mankind's *historical* CO₂ 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 CO₂ 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 CO₂ 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 CO₂ 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 CO₂-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 CO₂ concentration likely did for the productivity of other agricultural staples. Doing so, we find that the Industrial Revolution's flooding of the air with CO₂ resulted in mean yield increases on the order of 70% for other C₃ cereals, 28% for C₄ cereals, 33% for fruits and melons, 62% for legumes, 67% for root and tuber crops, and 51% for vegetables.

9.2.2.1.2. THE FUTURE

Clearly, the historical increase in the air's CO₂ 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 CO₂ Concentration*; and it was their conclusion that -- *after all that man can do* -- the aerial fertilization effect of the increase in the air's CO₂ 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 CO₂ content essential for the future well-being of man, *it is essential to the future well-being of the entire biosphere*.

Additional information on this topic, including reviews of newer publications as they become available, can be found at <http://www.co2science.org/subject/a/agriculture.php>.

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9.2.2.2. 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 CO₂ content play here?

9.2.2.2.1. PROTEIN CONTENT

In a review of the scientific literature related to effects of atmospheric CO₂ enrichment on plant constituents of significance to human health, Idso and Idso (2001) cited a number of studies where elevated levels of atmospheric CO₂ either increased, decreased or had no effect on the protein concentrations of various agricultural crops, the first two of which consequences have

subsequently also been observed by Kaddour and Fuller (2004) and Veisz *et al.* (2005), respectively, in wheat.

In the case of this particular crop - 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 CO₂ enrichment of the air, these experiments included increases and decreases in atmospheric ozone (O₃); and Pleijel *et al.* found that when increasing O₃ pollution reduced wheat grain yield, it simultaneously increased the protein concentration of the grain. They also found that when O₃ 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 concentration dropped when atmospheric CO₂ enrichment increased grain yield. Hence, it became clear that whenever the grain yield of the wheat was changed -- by CO₂, O₃ 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 CO₂, O₃ or water stress treatment.

In an earlier study of CO₂ and O₃ effects on wheat grain yield and quality, Rudorff *et al.* (1996) obtained essentially the same result. They observed, for example, that "flour protein contents were increased by enhanced O₃ exposure and reduced by elevated CO₂" but that "the combined effect of these gases was minor." Hence, they concluded that "the concomitant increase of CO₂ and O₃ 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 CO₂-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 CO₂ 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 CO₂ concentration on wheat grain nitrogen concentration and the baking properties of the flour derived from that grain throughout four years of free-air CO₂ 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 CO₂ 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 CO₂ 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 CO₂ enrichment can significantly increase wheat grain yield without sacrificing grain protein concentration in the process.

There are some situations, however, where atmospheric CO₂ enrichment has actually been found to *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 CO₂ concentrations of 350 and 600 ppm alone and in combination with 60 ppb SO₂ to study the interactive effects of elevated CO₂ and this major air pollutant on crop growth. They found that exposure to the elevated SO₂ caused a 13% decrease in foliar protein concentrations in both cultivars; but when the plants were concomitantly exposed to an atmospheric CO₂ 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 CO₂ enrichment. Likewise, they found no CO₂-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 CO₂-enriched wheat, rice and legumes was found by Jablonski *et al.* to be significantly increased above that of the same crops grown in normal air. Hence, there will likely be a vast increase in the total amount of protein that can be made available to humanity in a future CO₂-enriched world, both directly via food crops and indirectly via livestock.

With respect to the leguminous soybean, Thomas *et al.* (2003) additionally 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 CO₂ enrichment on soybean seed oil and oleic acid concentration."

Legumes and their responses to atmospheric CO₂ enrichment also figure prominently in a number of studies of *mixed* forage crops. In a study of nitrogen cycling in grazed pastures on the North Island of New Zealand, for example, Allard *et al.* (2003) report that under elevated CO₂, leaves of the individual species exhibited lower nitrogen concentrations but higher water-

soluble carbohydrate (WSC) concentrations. They also say "there was a significantly greater proportion of legume in the diet at elevated CO₂," and that this "shift in the botanical composition towards a higher proportion of legumes counterbalanced the nitrogen decrease observed at the single species scale, resulting in a nitrogen concentration of the overall diet that was unaffected by elevated CO₂." What is more, they report that "changes at the species level and at the sward level appeared to combine additively in relation to WSC." Hence, they note that "as there was a significant correlation between WSC and digestibility (as previously observed by Dent and Aldrich, 1963 and Humphreys, 1989), there was also an increase in digestibility of the high CO₂ forage," which result, in their words, "matches that found in a Mini-FACE experiment under cutting (Teyssonneyre, 2002; Picon-Cochard *et al.*, 2004)," where "digestibility also increased in response to CO₂ despite reduced crude protein concentration." These data, plus the strong relationship between soluble sugars (rather than nitrogen) and digestibility, led them to suggest that "the widespread response to CO₂ of increased soluble sugars might lead to an increase in forage digestibility."

Luscher *et al.* (2004) found much the same thing in their review of the subject, which was based primarily on studies conducted at the Swiss FACE facility that hosts what has become the world's longest continuous atmospheric CO₂ enrichment study of a naturally-occurring grassland. In response to an approximate two-thirds increase in the air's CO₂ concentration, leaf nitrogen (N) concentrations of white clover (*Trifolium repens* L.) and perennial ryegrass (*Lolium perenne* L.) were reduced by 7% and 18%, respectively, when they were grown separately in pure stands. However, as Luscher *et al.* report, "the considerably lower concentration of N under elevated CO₂, observed for *L. perenne* leaves in pure stands, was found to a much lesser extent for *L. perenne* leaves in the bi-species mixture with *T. repens* (Zanetti *et al.*, 1997; Hartwig *et al.*, 2000)." Furthermore, as they continue, "under elevated CO₂ the proportion of N-rich *T. repens* (40 mg N g⁻¹ dry matter) increased in the mixture at the expense of the N-poor *L. perenne* (24 mg N g⁻¹ dry matter when grown in monoculture)," the end result being that "the concentration of N in the harvested biomass of the mixture showed no significant reduction."

That this phenomenon is likely ubiquitous is suggested by the still more comprehensive review of the subject produced by Campbell *et al.* 2000), who analyzed research conducted between 1994 and 1999 by a worldwide network of 83 scientists associated with the Global Change and Terrestrial Ecosystems (GCTE) Pastures and Rangelands Core Research Project 1 (CRP1). This program had resulted in the publication of more than 165 peer-reviewed scientific journal articles; and Campbell *et al.* determined from this massive collection of data that the legume content of grass-legume swards was typically increased by approximately 10% in response to a doubling of the air's CO₂ content.

Also of interest within this context, Luscher *et al.* (2004) state that "the nutritive value of herbage from intensively managed grassland dominated by *L. perenne* and *T. repens* ... is well above the minimum range of the concentration of crude protein necessary for efficient digestion by ruminants (Barney *et al.* 1981)." Hence, they conclude that "a small decrease in the concentration of crude protein in intensively managed forage production systems [which

may never occur, as noted above] is not likely to have a negative effect on the nutritive value or on the intake of forage." In addition, in a CO₂-enriched world of the future there would be much *more* such forage produced per unit of land and water devoted to the enterprise, clearly making the ongoing rise in the air's CO₂ content a big plus for animal husbandry.

One final forage study we have reviewed on our website is that of Newman *et al.* (2003), who investigated the effects of two levels of nitrogen fertilization and an approximate doubling of the air's CO₂ content on the growth and chemical composition of tall fescue (*Festuca arundinacea* Schreber cv. KY-31), both when infected and uninfected with a mutualistic fungal endophyte (*Neotyphodium coenophialum* Morgan-Jones and Gams). They found that the elevated CO₂ reduced the crude protein content of the forage by an average of 21% in three of the four situations studied: non-endophyte-infected plants in both the low and high nitrogen treatments, and endophyte-infected plants in the high nitrogen treatment. However, there was *no* protein reduction for endophyte-infected plants in the *low* nitrogen treatment.

This latter point is very important; for as noted by Newman *et al.*, "the endophyte is present in many native and naturalized populations and the most widely sown cultivars of *F. arundinacea*," so that the first two situations in which the CO₂-induced protein reduction occurred (those involving non-endophyte-infected plants) *are not typical of the real world*. In addition, since the dry-weight biomass yield of the forage was increased by fully 53% under the low nitrogen regime, and since the *ten-times-greater* high nitrogen regime only boosted yields by an additional 8%, there would appear to be no need to apply any extra nitrogen to *F. arundinacea* in a CO₂-enriched environment. Consequently, under best management practices in a doubled-CO₂ world of the future, little to no nitrogen would be added to the soil and there would be little to no reduction in the crude protein content of *F. arundinacea*, but there would be more than 50% more of it produced on the same amount of land.

With respect to the final plant quality studied by Newman *et al.*, i.e., forage digestibility, increasing soil nitrogen lowered *in vitro* neutral detergent fiber digestibility in both ambient and CO₂-enriched air; and this phenomenon was most pronounced in the elevated CO₂ treatment. Again, however, under *low* nitrogen conditions there was *no* decline in plant digestibility. Hence, there is a *second* good reason to not apply extra nitrogen to *F. arundinacea* in a high CO₂ world of the future and, of course, little to no *need* to do so. Under best management practices in a future CO₂-enriched atmosphere, therefore, the results of this study suggest that much greater quantities of good quality forage should be able to be produced without the addition of any -- or very little -- extra nitrogen to the soil.

But what about the *unmanaged world of nature*? Increases in the air's CO₂ content often - but not always (Goverde *et al.*, 1999) - lead to greater decreases in the concentrations of nitrogen and protein in the foliage of C₃ as compared to C₄ grasses (Wand *et al.*, 1999); and as a result, in the words of Barbehenn *et al.* (2004a), "it has been predicted that insect herbivores will increase their feeding damage on C₃ plants to a greater extent than on C₄ plants (Lincoln *et al.*, 1984, 1986; Lambers, 1993).

To test this hypothesis, Barbehenn *et al.* (2004a) grew *Lolium multiflorum* Lam. (Italian ryegrass, a common C₃ pasture grass) and *Bouteloua curtipendula* (Michx.) Torr. (sideoats gramma, a native C₄ rangeland grass) in chambers maintained at either the ambient atmospheric CO₂ concentration of 370 ppm or the doubled CO₂ concentration of 740 ppm for two months, after which newly-molted sixth-instar larvae of *Pseudaletia unipuncta* (a grass-specialist noctuid) and *Spodoptera frugiperda* (a generalist noctuid) were allowed to feed upon the grasses. As expected, foliage protein concentration decreased by 20% in the C₃ grass, but by only 1% in the C₄ grass, when grown in the CO₂-enriched air. However, and "contrary to our expectations," according to Barbehenn *et al.*, "neither caterpillar species significantly increased its consumption rate to compensate for the lower concentration of protein in [the] C₃ grass," noting that "this result does not support the hypothesis that C₃ plants will be subject to greater rates of herbivory relative to C₄ plants in future [high-CO₂] atmospheric conditions (Lincoln *et al.*, 1984)." In addition, and "despite significant changes in the nutritional quality of *L. multiflorum* under elevated CO₂," they report that "no effect on the relative growth rate of either caterpillar species on either grass species resulted," and that there were "no significant differences in insect performance between CO₂ levels."

In a similar study with the same two plants, Barbehenn *et al.* (2004b) allowed grasshopper (*Melanoplus sanguinipes*) nymphs that had been reared to the fourth instar stage to feed upon the grasses; and once again, "contrary to the hypothesis that insect herbivores will increase their feeding rates disproportionately in C₃ plants under elevated atmospheric CO₂," they found that "*M. sanguinipes* did not significantly increase its consumption rate when feeding on the C₃ grass grown under elevated CO₂," suggesting that this observation implies that "post-ingestive mechanisms enable these grasshoppers to compensate for variable nutritional quality in their host plants," and noting that some of these post-ingestive responses may include "changes in gut size, food residence time, digestive enzyme levels, and nutrient metabolism (Simpson and Simpson, 1990; Bernays and Simpson, 1990; Hinks *et al.*, 1991; Zanotto *et al.*, 1993; Yang and Joern, 1994a,b)." In fact, their data indicated that *M. sanguinipes* growth rates may have actually *increased*, perhaps by as much as 12%, when feeding upon the C₃ foliage that had been produced in the CO₂-enriched air.

In conclusion, with respect to both managed agricultural crops and the wild plants of earth's natural ecosystems, it would appear that the ongoing rise of the air's CO₂ concentration will have few negative impacts of any consequence on the nutritive value of their grains and foliage in terms of protein concentration. In fact, in tree crops such as citrus, CO₂-induced changes in the activities of certain foliar proteins could well lead to vast increases in yield potential, as elucidated by the work of Idso *et al.* (2001).

Additional information on this topic, including reviews of newer publications as they become available, can be found at <http://www.co2science.org/subject/p/protein.php>.

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9.2.2.2.2. ANTIOXIDANT CONTENT

Environmental stresses induced by exposure to pollutants, drought, intense solar radiation and high air or water temperatures generate highly-reactive oxygenated compounds that damage plants. Ameliorating these stresses typically involves the production of antioxidant enzymes that scavenge and detoxify the highly-reactive oxygenated compounds. Hence, when stresses are present, concentrations and/or activities of antioxidants in plants are generally observed to be high; and as Lesser (1997) has noted in corals, they tend to counter the effects of the stress. A good question to ask, therefore, is how atmospheric CO₂ enrichment impacts this relationship and what the observed results imply.

In a study of two soybean genotypes, Pritchard *et al.* (2000) reported that three months' exposure to twice-ambient CO₂ concentrations reduced the activities of superoxide dismutase and catalase by an average of 23 and 39%, respectively. Likewise, Polle *et al.* (1997) showed that two years of atmospheric CO₂ enrichment reduced the activities of several key antioxidative enzymes, including catalase and superoxide dismutase, in beech seedlings. Moreover, Schwanz and Polle (1998) demonstrated that this phenomenon can persist indefinitely, as they discovered similar reductions in these same enzymes in mature oak trees that had been growing near natural CO₂-emitting springs for 30 to 50 years.

The standard interpretation of these results is that the observed reductions in the activities of antioxidative enzymes under CO₂-enriched conditions imply that plants exposed to higher-than-current atmospheric CO₂ concentrations experience less oxidative stress and thus have a reduced need for antioxidant protection. This conclusion further suggests that "CO₂-advantaged" plants will be able to funnel more of their limited resources into the production of other plant tissues or processes essential to their continued growth and development.

On the other hand, when oxidative stresses do occur under high CO₂ conditions, the enhanced rates of photosynthesis and carbohydrate production resulting from atmospheric CO₂ enrichment generally enable plants to better deal with such stresses by providing more of the raw materials needed for antioxidant enzyme synthesis. Thus, when CO₂-enriched sugar maple seedlings were subjected to an additional 200 ppb of ozone, Niewiadomska *et al.* (1999) reported that ascorbate peroxidase, which is the first line of enzymatic defence against ozone,

significantly increased. Likewise, Schwanz and Polle (2001) noted that poplar clones grown at 700 ppm CO₂ exhibited a much greater increase in superoxide dismutase activity upon chilling induction than clones grown in ambient air. In addition, Lin and Wang (2002) observed that activities of superoxide dismutase and catalase were much higher in CO₂-enriched wheat than in ambiently-grown wheat following the induction of water stress.

Antioxidants are also of great importance to human health. In some cases, the additional carbon fixed during CO₂-enrichment is invested in antioxidative compounds, rather than enzymes; 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 CO₂ 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 CO₂ increase on the same species, although the extra CO₂ 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 CO₂ concentration actually *doubled* plant vitamin C contents over a 7-day period (Tajiri, 1985).

Probably the most comprehensive investigation of CO₂ effects on vitamin C production in an agricultural plant -- a tree crop (sour orange) -- was conducted by Idso *et al.* (2002). In an atmospheric CO₂ enrichment experiment begun in 1987 and still ongoing, a 75% increase in the air's CO₂ 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 CO₂-induced increase in fruit production was much greater, however, the increase in fruit vitamin C concentration was also greater, rising to a CO₂-induced enhancement of 15% when fruit production on the CO₂-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 CO₂ 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 CO₂ on plant antioxidant production was that of Wang *et al.* (2003), who evaluated the effects of elevated CO₂ 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 CO₂ concentration, two of which were maintained at ambient + 300 ppm CO₂, and two of which were maintained at ambient + 600 ppm CO₂ 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 CO₂ 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 CO₂ environments." In going from ambient to ambient + 300 ppm CO₂ and ambient + 600 ppm CO₂, 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 CO₂ 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 $55 \pm 23\%$ in going from the ambient atmospheric CO₂ concentration to ambient + 300 ppm CO₂, and a mean concentration increase of $112 \pm 35\%$ in going from ambient to ambient + 600 ppm CO₂. 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 CO₂ enrichment conditions, increased the[ir] AsA, GSH, phenolic acid, flavonol, and anthocyanin concentrations," further noting that "plants grown under CO₂ enrichment conditions also had higher oxygen radical absorbance activity against [many types of oxygen] radicals in the fruit." Hence, they determined that atmospheric CO₂ enrichment truly did "make a good thing better."

We note additionally in this regard that elevated levels of atmospheric CO₂ 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 CO₂, 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 CO₂ 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 CO₂ ranging from 9% to 197% (mean of 76% ± 15%); and Desjardins *et al.* (1987) report a 118% increase in photosynthesis in response to a 600 ppm increase in the air's CO₂ concentration.

Other researchers have found similar enhancements of antioxidative compounds under enriched levels of atmospheric CO₂. Estiarte *et al.* (1999), for example, reported that a 180-ppm increase in the air's CO₂ content increased the foliar concentrations of flavonoids, which protect against UV-B radiation damage, in field-grown spring wheat by 11 to 14%. Caldwell *et al.* (2005), on the other hand, found that an ~75% increase in the air's CO₂ content increased the total isoflavone content of soybean seeds by 8% when the air temperature during seed fill was 18°C, by 104% when the air temperature during seed fill was 23°C, by 101% when the air temperature was 28°C, and by 186% and 38%, respectively, when a drought-stress treatment was added to the latter two temperature treatments. Lastly, in an experiment conducted under *very high* atmospheric CO₂ concentrations, Ali *et al.* (2005) found that CO₂ levels of 10,000 ppm, 25,000 ppm and 50,000 ppm increased total flavonoid concentrations of ginseng roots by 228%, 383% and 232%, respectively, total phenolic concentrations by 58%, 153% and 105%, cysteine contents by 27%, 65% and 100%, and non-protein thiol contents by 12%, 43% and 62%, all of which substances are potent antioxidants. What is more, it is interesting to note that the increased consumption of such plant material - naturally enriched with antioxidative compounds as a consequence of the historical rise in the air's CO₂ content - may have played a role in the observed decline in human mortality rates over the period 1950-1994 (Tuljapurkar *et al.*, 2000).

In summary, as the CO₂ content of the air rises, plants typically experience less oxidative stress; and since they thus need fewer antioxidants for protection, antioxidant levels in their leaves decline, which enables them to use more of their valuable resources for other important purposes. However, when oxidative stresses are present, elevated CO₂ helps to increase the synthesis and activities of antioxidants that tend to alleviate the problems the stresses cause. Moreover, the possibility exists that the greater concentrations of antioxidative compounds in plant tissues caused by the historical rise in the air's CO₂ content may have contributed to the human life span extension experienced over the course of the Industrial Revolution.

Additional information on this topic, including reviews of newer publications as they become available, can be found at <http://www.co2science.org/subject/a/antioxidants.php>.

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9.2.3. 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 CO₂ 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 CO₂ 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 CO₂ content increased plant dry weight production in this medicinal plant by 63%, while under water-stressed conditions the CO₂-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 CO₂ 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 CO₂ 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 CO₂ 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 CO₂.

Additional information on this topic, including reviews of newer publications as they become available, can be found at <http://www.co2science.org/subject/h/co2healthpromoting.php>.

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9.2.4. 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 CO₂-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 CO₂ 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 CO₂-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*.

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9.3. A Brief History Of Human Longevity

The past two centuries have witnessed 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 seen an increase in its atmospheric CO₂ 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? Although no one can give a precise quantitative answer to this question, it is possible to assess their *relative* importance by considering the history of human longevity.

Tuljapurkar *et al.* (2000) examined mortality over the period 1950-1994 in Canada, France, Germany (excluding the former East Germany), Italy, Japan, the United Kingdom, and the United States, finding 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, Horiuchi notes that "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," but data from the United States demonstrate, in his words, 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 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.

With respect to the population of the entire planet, 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," and they emphasize that this phenomenal trend "is so extraordinarily linear that it may be *the most remarkable regularity of mass endeavor ever observed* [our italics]."

These observations clearly demonstrate that if the increases in air temperature and CO₂ concentration of the past two centuries were indeed bad for our health, their combined negative influence was *minuscule* compared to whatever else was at work in promoting this vast increase in worldwide human longevity; and it is that "whatever else" to which we now turn our attention.

To summarize to this point, in countries with highly developed market economies 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 ever longer with the passing of time. It is further evident that this phenomenon is likely due to ever-improving health in older people, which in turn is likely the result of continuing improvements in the abilities of their bodies to repair cellular damage caused by degenerative processes associated with old age, i.e., stresses caused by the reactive oxygen species that are generated by normal metabolism (Finkel and Holbrook, 2000). What is responsible for this incredible lengthening of human life span? It is probably a number of things acting in concert, with no single phenomenon overpowering the others. Nevertheless, the multi-faceted force has operated with unwavering consistency since the inception of the Industrial Revolution, which leads us to wonder if the "twin evils" of the climate-alarmist crowd might actually be responsible for some portion of the longer and healthier lives that are being experienced by the planet's elderly. In what follows we briefly review some materials that illustrate some of the means by which elevated atmospheric CO₂ concentrations may help to extend human life span.

Wentworth *et al.* (2003) report they found "evidence for the production of ozone in human disease," specifically noting that "signature products unique to cholesterol ozonolysis are present within atherosclerotic tissue at the time of carotid endarterectomy, suggesting that ozone production occurred during lesion development." As Marx (2003) describes it, "researchers think that inflammation of blood vessels is a major instigator of plaque formation," that "ozone contributes to plaque formation by oxidizing cholesterol," and that the new findings "suggest new strategies for preventing atherosclerosis." Further, according to Marx, Daniel Steinberg of the University of California, San Diego, says that although it's still too early to definitively state whether ozone production in plaques is a major contributor to atherosclerosis, he expresses his confidence that once we know for sure, we'll know which antioxidants will work in *suppressing* plaque formation.

Reactive oxygen species (ROS) generated during cellular metabolism or peroxidation of lipids and proteins also play a causative role in the pathogenesis of cancer, along with coronary heart disease (CHD), as demonstrated by Slaga *et al.* (1987), Frenkel (1992), Marnett (2000), Zhao *et al.* (2000) and Wilcox *et al.* (2004). However, as noted by Yu *et al.* (2004), "antioxidant treatments may terminate ROS attacks and reduce the risks of CHD and cancer, as well as other ROS-related diseases such as Parkinson's disease (Neff, 1997; Chung *et al.*, 1999; Wong *et al.*, 1999; Espin *et al.*, 2000; Merken and Beecher, 2000)." As a result, they say that "developing functional foods rich in natural antioxidants may improve human nutrition and reduce the risks of ROS-associated health problems."

Consider, in this regard, the common strawberry. Wang *et al.* (2003) report that strawberries are especially good sources of natural antioxidants. They say 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)." They also note that Wang and Jiao (2000) and 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." And they say 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)."

Our reason for citing all of this information is that Wang *et al.* (2003) have recently demonstrated that enriching the air with carbon dioxide increases both the concentrations and activities of many of these helpful substances. They determined, for example, that strawberries had higher concentrations of ascorbic acid and glutathione when grown in CO₂-enriched environments. They also learned that "an enriched CO₂ environment resulted in an increase in phenolic acid, flavonol, and anthocyanin contents of fruit." For nine different flavonoids, in fact, there was a mean concentration increase of 55% in going from the ambient atmospheric CO₂ concentration to ambient + 300 ppm CO₂, and a mean concentration increase of 112% in

going from ambient to ambient + 600 ppm CO₂. Also, they report that "high flavonol content was associated with high antioxidant activity."

There is little reason to doubt that similar concentration and activity increases in the same and additional important phytochemicals in other food crops would occur in response to the same increases in the air's CO₂ concentration. Indeed, the *aerial fertilization effect* of atmospheric CO₂ enrichment is a near-universal phenomenon that operates among plants of all types, *and it is very powerful*. Since the dawning of the Industrial Revolution, for example, the work of Mayeux *et al.* (1997) and Idso and Idso (2000) suggests that the concomitant historical increase in the air's CO₂ content has led to mean yield increases of approximately 70% in C₃ cereals, 28% in C₄ cereals, 33% in fruits and melons, 62% in legumes, 67% in root and tuber crops, and 51% in vegetables. Hence, there must have been significant concomitant increases in the concentrations and activities of the various phytochemicals in these foods that act as described by Wang *et al.* (2003).

Our reason for reporting these observations is that atmospheric CO₂ enrichment has been documented to enhance the concentrations of a number of antioxidants in many of the foods we eat. In what follows, therefore, we merely report the results of two studies that have confirmed the positive effects of antioxidant-induced decreases in animal ROS concentrations on animal life span.

Melov *et al.* (2000) tested the theory that reactive oxygen species cause aging by examining the effects of two superoxide dismutase-/catalase-like mimetics (EUK-8 and EUK-134) on the life-span of normal and mutant *Caenorhabditis elegans* worms that ingested various concentrations of the mimetics. In all of their experiments, treatment of normal worms with the antioxidant mimetics significantly increased both mean and maximum life-span. Treatment of normal worms with but 0.05 mM EUK-134, for example, increased their mean life-span by fully 54%; and in mutant worms whose life-span had been genetically shortened by 37%, treatment with 0.5 mM EUK-134 restored their life-span to normal by increasing their mutation-reduced life-span by 67%. It was also determined that these effects were not due to a reduction in worm metabolism, which could have reduced the production of oxygen radicals, but "by augmenting natural antioxidant defenses without having any overt effects on other traits." In other words, in the words of the authors, "these results suggest that endogenous oxidative stress is a major determinant of the rate of aging," the significance of which statement resides in the fact that antioxidants tend to reduce such stresses in animals, including man, and in the observation that atmospheric CO₂ enrichment has been shown to significantly enhance the concentrations of many of these important ROS-fighting constituents of the foods we eat.

Another study to address the subject was conducted by Larsen and Clarke (2002), who fed diets with and without coenzyme Q to wild-type *Caenorhabditis elegans* and several mutants during the adult phases of their lives, while they recorded the lengths of time they survived. This work revealed that "withdrawal of coenzyme Q from the diet of wild-type nematodes extends adult life-span by ~60%." In addition, they found that the life-spans of the four different mutants they studied were extended by a Q-less diet. More detailed experiments led them to conclude that

the life-span extensions were due to *reduced generation* and/or *increased scavenging* of reactive oxygen species, leading them to conclude in the final sentence of their paper that "the combination of reduced generation and increased scavenging mechanisms are predicted to result in a substantial decrease in the total cellular ROS and thereby allow for an extended life-span."

In light of these many diverse observations of both plants and animals, there is ample reason to believe that the historical increase in the air's CO₂ content has played a prominent role in enhancing many aspects of human health over the course of the Industrial Revolution, and that its continued upward trend will provide ever more of the same benefits, not the least of which is *increased longevity*. Consequently, we end this section with the encouraging thought that perhaps the best way to obtain these benefits is to directly consume foods that are known to contain high levels of antioxidants, i.e., fruits and vegetables, since with each passing year these foods will likely contain ever greater concentrations of ROS-fighting substances, thanks to the ongoing rise in the air's CO₂ content that comes primarily from the burning of fossil fuels.

Additional information on this topic, including reviews of newer publications as they become available, can be found at <http://www.co2science.org/subject/h/humanlifespan.php>.

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9.4. Conclusions

Although historical and projected future increases in the air's CO₂ 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 CO₂ content rose by 35% and its near-surface temperature by about 1°C, there has been not the slightest negative impact on human lifespan. In fact, human lifespan has concurrently experienced an almost unbelievable increase that shows no sign of ultimately leveling off or even slowing down. What is more, warming has been shown to positively impact human health, while atmospheric CO₂ enrichment has been shown to enhance the health-promoting properties of the food we eat (as well as produce more of it) and increase the presence and effectiveness of disease-fighting substances found in plants. Clearly, we have absolutely nothing to fear from the "twin evils" (CO₂ and warming) of the extreme environmental movement. Indeed, they would appear to be our *friends*.