Contrary to popular belief, stratospheric ozone depletion, and the resultant increase in solar UV-B (280-320 nm), are unlikely to fully recover soon. Notwithstanding the success of the Montreal Protocol in reducing the amount of ozone destroying chemicals into the stratosphere, the life-times of these compounds are such that even with full compliance with the Protocol by all countries, it will be decades before stratospheric ozone could return to pre-1980 levels. This raises the question, therefore, of what will happen to biological processes essential to the maintenance of life on earth which are sensitive to damage by increased UV-B radiation, particularly those involved with human health? The polar regions, because of the vagaries of climate and weather, are the bellwether for stratospheric ozone depletion and will, therefore, be the first to experience impacts due to increases in solar UV-B radiation. The impacts of these are incompletely understood and cannot be predicted with certainty. While some UV-B impacts on human health are recognized, much is unknown, unclear and uncertain. Thus, this paper attempts, as a first approximation, to point out potential impacts to the health and welfare of human inhabitants of the Arctic due to increased solar UV-B radiation associated with stratospheric ozone depletion. As will be seen, much more data is critically needed before adequate risk assessment can occur. (Int J Circumpolar Health 2005;64(5):509-522.)

Keywords: UV-B effects, ozone depletion, Arctic population
INTRODUCTION

Stratospheric ozone loss over the Arctic is a real phenomenon (Figure 1) and may continue to increase for decades, potentially leading to an Arctic ozone hole (1, 2).

Ozone depletion is important to human health because as stratospheric ozone decreases UV-B radiation increases (Figure 2). Stratospheric ozone loss appears to be directly linked to low stratospheric temperatures. As temperatures fall below −78°C, polar stratospheric ozone cloud (PSC) formation takes place (Figure 1, right ordinate). PSC promotes ozone depletion.

Ozone loss tends to occur at high latitudes, but recent ozone losses (2004-2005) were so extensive that, albeit at lower levels, they were seen throughout the mid latitudes of the Northern Hemisphere. Arctic ozone depletion tends to be sporadic, e.g. the previous past two winters (2002-2003 and 2003-2004) produced temperatures in the stratosphere over the Northern Hemisphere warm enough to prevent significant ozone loss. As Figure 1 shows, however, significant ozone loss in the Arctic is not an isolated event.

Most of the stratospheric ozone loss over the past decade has been observed during winter/spring months in Arctic regions. During this time, losses of up to 40% in Scandinavia and Siberia had been recorded. In Canada, sporadic losses of 10-20% or more also had been reported. In Point Barrow, Alaska (71.3° N), a daily total ozone value for March 1997 was approximately 6% below the previous ten year average. A recent report indicates a modest but stable recovery in the ozone layer everywhere except over the poles (3). With regard to UV-B improvement, however, not much progress is noted in reducing the increased UV-B radiation associated with ozone depletion (4). A wide-ranging comprehensive analysis on climate change impacts on the Arctic, including analysis on increased UV-B impacts on human health resulting from ozone depletion in the Arctic, has recently been published and has garnered world-wide attention (5).

Are ozone layer destruction and global warming related?

Ozone is a gas made up of 3 atoms of oxygen (O3). Tropospheric or ground level ozone is a major component of smog. Stratospheric O3 occurs 10 to 50 kilometers above the earth’s surface. Ozone is a very important part of the atmosphere as it efficiently absorbs UV-B radiation thus protecting the earth from this dangerous component of sunlight. Tropospheric ozone is also a greenhouse gas similar to CO2. Thus, it also has the capacity to trap heat at the earth’s surface. As a consequence, less outpouring of heat from the earth will result in a cooler stratosphere. Additionally, lower levels of stratospheric ozone result in less absorption of incoming solar radiation further cooling the stratosphere. As noted above, an important consequence of stratospheric cooling is the formation of polar stratospheric clouds resulting in ozone destruction, leading to more harmful UV-B transmitted to the Earth. Interestingly, ozone itself may affect climate change. For example, it is suggested that reduction in stratospheric ozone alters the temperature distribution in the stratosphere, especially in the southern Hemisphere, strengthening the polar jet stream and delaying the transition to summer-type circulation. This delays the breaking up of the polar vortex over Antarctic, slowing down the mixing of ozone rich air from higher latitudes with the
depleted Antarctic air. Climate change affects cloud cover, aerosol concentration and surface reflectance or albedo. All of these climate effects impact on the amount of UV-B at the Earth’s surface. It appears that as long as the greenhouse effect continues, it is likely that recovery of the ozone layer in the stratosphere will be delayed. The relationship between global climate change and ozone depletion is very complex and not all agree on the correct relationship (1). A better understanding is needed of the complicated interactions between climate change and ozone depletion in order to more adequately assess ozone loss and solar UV-B impacts.

UV-B Radiation Health Risk in the Arctic: why is UV-B radiation a potential health problem?

Some of the most important biomolecules that exist in living cells are strong UV-B absorbers and, as such, are subject to change and modification when they absorb this radiation. In living systems where homeostasis or equilibrium is vital to a living cell, changes in biomolecules may introduce modifications which lead to alterations such that living cells can no longer function normally. For example, UV-B (280-320 nm) and UV-A (320-400 nm) initiate, in general, different spectra of photoproducts (6, 7) and damage is repaired.
by different pathways (8, 9). UV-B and UV-A elicit different transcriptional (10) and inflammatory responses (11); and UV-B and UV-A have different effects on the immune response.

Sunburn in the Arctic

Very little, if any, incidence data on sunburn in the Arctic (or elsewhere) can be found. For the Arctic, this is not entirely surprising since about half the year is spent in very low levels of sunlight. Previously, sunburns were not generally considered a problem in the Arctic although this may be changing due to ozone depletion. For example, some indigenous groups in the North are starting to report evidence of increased UV-B exposure and experiencing skin rashes and burns for the first time, and report a sense that the “sun is hotter” than they have experienced as the following quotations indicate:

“The sun burns us easily, it was not very hot in the past.” (Kuujjuaq, man age 62) (12)

“The sun was not that hot in the past. Nowadays, it’s really hot. My skin burns when I’m out for a while. Sometimes, we stay indoors in a shack.” (Kuujjuaq, man age 70) (12)

Furthermore, a recent study involving arctic field scientists who were engaged in biological and geological fieldwork, were monitored for erythemal(sunburn)-weighted exposure at latitude 75° N (13). It was estimated that about 80 standard erythemal doses (SED; see Figure 4) to the face of erythemally weighted sunlight could be received during a month of field work in July, 2000. Considering that exposure in an animal melanoma model showed 7 to 23 SED to be sufficient to induce melanoma with human-like pathology and genetic characteristics (14), this amount of sunburn exposure to humans might be significant for melanoma risk. Monitoring of sunburn episodes in northern populations on a more formal and systematic basis should be seriously considered and implemented quickly to establish valid base-line estimates and risk assessment for this important health effect.

UV-B and Cataracts

In 2002, there was an estimated 180 million people worldwide who were visually disabled. Between 40 and 45 million persons are declared blind and, by definition, cannot walk about unaided. Some 16 million people worldwide are currently blind as a result of cataracts; of these, the WHO estimates that as many as 20% may be due to UV radiation exposure [http://www.who.int/topics/cataract/en/].

Epidemiological reports and experimental studies indicate that cataract formation is a complicated process with many associated risk factors. The precise mechanism of action is not known although UV-B radiation is very strongly implicated and associations with latitude and climatically different countries have been reported (15-20).

Furthermore, a recent action spectrum indicated that after correcting for corneal transmittance, the biological sensitivity of the rat lens to UV-B is at least as great at 295 nm as at 300 nm. After correcting for transmittance by the atmosphere, UV-B at 305 nm is suggested to be the most likely wave band to injure the rat lens (21).

Several types of cataract exist with a varying degree of association with sunlight. In general, published evidence supports the concept that cortical cataracts are more likely to be related to UV-B (15-17, 20). One study, however,
suggests the opposite when life-time cumulative UV-B exposure and exposure after teenage years are considered (22) underscoring the need for further study including detailed wavelength or action spectrum studies on eye cataract. The effects of diet on cataract formation also needs to be considered but requires further research (18;23;24). Cataracts can also be a problem associated with diabetes (e.g. cataract is the second most common complication of diabetes mellitus) but little information is available on this association in Northern human populations.

Recently, Meyer-Rochow, called for an international effort to redress the complicated effects of UV radiation, particularly in regard to the risk of eye damage in the Arctic (25). To date there is still little known with regard to the causes of eye problems in northern indigenous populations (26). The amount of data available on Arctic populations and the incidences of cataracts is scarce. Given the potential for increased UV-B due to ozone depletion, which is projected to last for the foreseeable future, the call for a concerted international study on UV-B damage to the eye needs to be strongly reiterated.

Photokeratitis or “sunburn” of the eye
Damage to the clear front surface of the eye, the cornea, can occur by UV-B radiation, whether the UV-B comes from natural sunlight (e.g. at high altitudes), or from artificial sources such as welder’s arc or from sun tanning beds. It can also occur when UV-B is reflected from snow cover (snow blindness). Symptoms include a gritty feeling in the eye (eye feels like it has sand in it) and can be painful. Redness, tearing and swollen eyelids may be noted. These symptoms usually start about a few hours after exposure and normal vision usually returns after about 24 hours or less. Depending on severity, the damage to the cornea is usually repaired after about 1 or 2 days. UV-B filtering wrap-around sunglasses (side blocking of UV-B light) is considered good protection. UV-B exposure to the eye should be limited as much as possible (minutes not hours).

Pollution and UV-B
Pollutants of a wide variety of types are well described in the Arctic such as the polyaromatic hydrocarbons (PAHs), polychlorinated biphenyls (PCBs), and heavy metals (See Chapters 6 and 10 in reference (27)). In some cases interaction between UV-B and chemical pollutants have been seen to occur in aquatic organisms following ingestion of UV-B-absorbing PAHs. When exposed to UV-B, these organisms show phototoxic effects (28). A number of persistent organic pollutants (POP’s) which include PCB’s, may be immunologically damaging without UV-B. At present there is little information available on potential combined effects between these agents and immunosuppression caused by UV-B alone. In view of such findings and the high levels of POPS in some Arctic regions, research on combinatorial effects between UVB and PAH’s and PCB’s is critically needed.

UV-B and Immunosuppression
UV-B radiation alone can initiate a selective down regulation of cell-mediated immunity in mammals, including humans. The unusual feature of UV-B-induced form of immunosuppression is that it redirects cell-mediated immunity (CMI) to sensitizing antigens from an effector-type response to a suppressor response. These can include chemical antigens, viral antigens, and tumor antigens (29). Skin pigmentation is not an efficient protective factor.
against UV-B induced immune suppression. Immunosuppressive effects by UV-B play an important role in UV-B induced skin cancer by preventing the destruction of highly antigenic skin cancers by the immune system (30, 31) (See also Skin Cancer and UV-B below). In addition, sunlight modulation of cell-mediated immunity may involve susceptibility to certain infectious diseases (32). A light-absorbing molecule found on the surface of skin, identified as trans-urocanic acid (t-UCA), has been shown to be the initiator of UVB-induced immunosuppression (33). Immunosuppression is activated when t-UCA isomerizes to its cis-form (c-UCA). Cis-UCA is known to cause immunosuppression in humans similar to the immunosuppression in mice caused by UV-B (34).

**Wavelength dependence or “action spectrum” for UV-B induced biological effects**

Photobiological responses are by definition wavelength dependent. However, to accurately compare biological-inducing activity of the many spectrally different sources available, from sunlight to sunlamps, it is necessary to consider differences in wavelength efficiency in initiating the biological response be it skin cancer, sunburn, photosynthesis, immune suppression to name a few. In order to make such comparisons, it is necessary to deliver, “biologically-effective” doses from the optical source (Figure 4). As described in the legend to figure 4, such differences in wavelength efficiency can be taken into account with use of an appropriate wavelength-dependence or “action spectrum”. Action spectra, in addition

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**Derivation of Biologically Effective Radiation**

To determine biologically effective doses, the spectral output of a source (in this example the F40 sunlamp) is multiplied by an action spectrum of interest, in this case the CIE sunburn action spectrum (45). The area under the Product Curve is integrated and the resulting effective irradiance (W m$^{-2}$) is used to calculate the effective dose as follows: $\text{Dose}_{\text{eff}}$ (Joules m$^{-2}$) = Irradiance$_{\text{e}}$ (W m$^{-2}$) x exposure time (s). When using the CIE standard erythema action spectrum the Dose$_{\text{e}}$ may be expressed as the Standard Erythemal Dose (SED). One standard SED is defined as 100 Joules/m$^2$ (Note: 1 Watt = 1 Joule (J)/sec). In this example, 23 SED was used to induce cutaneous malignant melanoma, with strong similarity to human melanoma, in an experimental transgeneic mouse model using the F40 sunlamp (46). Twenty three SED could have been received in ~2.5 h on 4 July 2000, at 32.7° N. Longitude: -117.1° W. Terrestrial irradiance provided courtesy Biospherical Instruments Inc, San Diego CA.
to providing a weighting function to determine biologically-effective doses, are very useful in providing information about the identity of the initial photoreceptor responsible for triggering a light-driven response (35). Such information can provide critical direction for research on molecular mechanism (33).

The first step in constructing an action spectrum usually involves identifying the active waveband, for example separating UV-A from UV-B. Following this, narrowband irradiation is used across the active waveband to create dose-response curves from which an action spectrum is constructed. Data on identification of the specific waveband for melanoma induction has recently been published (14). This information is a necessary prerequisite for a melanoma action spectrum which in turn, can be used as a biological “weighting function” for risk assessment of solar radiation induction of melanoma. (See also Figure 4 above).

**Genetic interaction with UV-B**

Genetically determined susceptibilities to UV-induced immunosuppression have been shown to exist and appear to be controlled by a number of interacting Uvs genes involving autosomal and X-linked genes. Such an interaction for UV-immunosuppression had not been described previously and may be unique for this mechanism (36). Genetically determined high susceptibility to UV immunosuppression may be an important risk factor for UV-related human diseases not only in arctic populations but other populations as well.

**Skin cancer and UV-B**

There are three main types of skin cancer. Two generally do not metastasize and are called basal and squamous cell carcinoma often referred to collectively as non-melanoma skin cancer. The third type, which shows a higher mortality, and which can metastasize aggressively, is malignant melanoma of which several subtypes exist (37, 38).

There is much experimental evidence which provide a clear connection between sunlight exposure and non-melanoma skin cancer, and which implicates UV-B as a carcinogen (37, 39, 40). A relationship between sunlight and melanoma, while less clear, is considered a near certainty (37, 41-44). Supporting this contention is the recent development of an HGF/SF transgenic melanoma mouse model in which melanocytes are distributed ectopically throughout the dermis and epidermis, comparable to human skin. Following UV radiation melanomas arise with an etiology, histopathology and molecular pathogeneses remarkably reminiscent of human melanoma (46) New studies using this model show that a single exposure to neonates of an erythemally-weighted dose of precisely defined and controlled UV-B is able to initiate cutaneous melanoma development, while UV-A cannot, even at doses sufficiently high to be predicted to be melanomagenic (14)

The molecular/genetic mechanisms underlying the relationship between malignant melanoma and exposure to UV-B versus UV-A are, at present, unclear.

It should be pointed out that not all sunburns lead to melanoma, as other predisposing factors are often needed. For example, immunosuppressive effects of UV-B, as described above, play an important role in UV-induced non-melanoma skin cancer by preventing the destruction of highly antigenic skin cancers by the immune system (30, 31). The relationship between UV-B induced immunosuppression and melanoma is less clear.
Skin cancer in the northern populations, ozone depletion and risk evaluation

Epidemiological evidence indicates that sporadic or intermittent sunlight exposure can be a very important factor in malignant melanoma development especially in childhood (47). Although epidemiological studies on melanoma in northern latitudes tend to be scarce, and skin cancer incidence rates in northern latitudes tend to be low, melanoma rates world-wide are increasing including in some Northern countries. The World Health Organization (WHO) estimates that melanoma incidence rates in Norway and Sweden have more than tripled in the last 45 years, while in the United States the rate has doubled in the last 30 years [http://www.who.int/media-centre/news/notes/2005/np07/en/].

In Saskatchewan Canada, a dramatic increase in incidence of melanoma has been reported between 1970 and 1999 (48). And in one recent study in northwest Russia (north of the 60th latitude) melanoma incidence rates for men were seen to be very different from melanoma rates in European countries (49). With regard to risk assessment, a skin cancer action spectrum has been used to predict increases in UV-B induced non-melanoma skin cancer resulting from ozone destruction between 1979-1994 (50). Whether observed increases in melanoma world-wide are due to increased UV-B due to ozone depletion remains to be determined.

Skin cancer is rare in Arctic indigenous populations consistent with findings elsewhere that skin pigmentation is protective against skin cancer. A recent study, however, involving Danes working in Greenland and cancer risk indicated an elevated risk of melanoma in females. A role for excessive UV exposure in this regard has been suggested (51). With increasing numbers of non-indigenous populations living in the Arctic (52) the incidence of melanoma and non-melanoma skin cancer needs to be carefully monitored in both indigenous as well as non-indigenous inhabitants. It should be noted that because of significant warming in the arctic over recent years any potential increase in skin cancer incidence which may be related to reflectance from snow may be mitigated by the projected decrease in snow cover.

Dietary factors and UV-B-induced immunosuppression

Another factor which may be important in UV-B effects in Arctic populations is diet. Experimental evidence indicates that UV immunosuppression in mice can be increased by increasing dietary histidine (53) High dietary fat has also been demonstrated to enhance UV-induced immunosuppression in experimental systems (54) and may also be a factor in Arctic populations along with high histidine concentrations in some fish (55) and seal species (56).

Non-Hodgkin’s lymphoma and UV-B radiation

The incidence of non-Hodgkin’s lymphoma (NHL) has been increasing rapidly over the past 30 years. The reasons for this trend are not known although increasing exposure to sunlight has been postulated (57). And, indeed, certain epidemiological evidence suggests that NHL shows a relationship with sunlight exposure (58, 59). This relationship is suggested to be via the immunosuppressive effects of UV-B (58-61) A correlation between the occurrence of skin cancer and the occurrence of NHL
also has been described (62). NHL does not, however, show a latitudinal gradient in the U.S. (63). This is in contrast to non-melanoma skin cancer, suggesting that an effect of UV-B radiation may be as a co-factor rather than as a primary causative agent of this disease. Recent data suggest less of a direct association between NHL and UV radiation than previously believed. Indirectly, however, evidence showing a correlation between NHL and melanoma has been reported and suggests a possible sunlight association. One recent large Australian study found, in individuals with NHL, an increased risk of a second primary cancer, which include melanoma and nonmelanoma skin cancers, cancer of the lip and cancer of the tongue (57). Conversely, in a different study, an increased risk was noted of NHL developing among patients with skin cancers (both melanoma and nonmelanoma skin cancers (64). These data provide some support for a sunlight association with NHL. The authors conclude, however, that support for a direct relationship between UV and the incidence of NHL was weak and inconsistent (64). Reports continue to be published regarding correlations between NHL and UV radiation and, while results are mixed, there appears to be sufficient linkage (directly or indirectly) to warrant further investigation regarding ozone loss over the Arctic, increased UVB and NHL (65). More research needs to be conducted to determine if UV radiation in sunlight exposure plays a role in the development of NHL. With regard to other lymphatic disease, Danish women working in Greenland are reported to show an excess of lymphatic malignancies raising the question of a role for excess UV-B (51).

Autoimmune diseases such as Type-I diabetes and Multiple Sclerosis (MS) may also have an immunosuppressive connection with UV-B. For example, the prevalence of type 1 diabetes mellitus was found associated with latitude of residence and decreasing UV in the southern hemisphere (66). The inverse association of type 1 diabetes and UV radiation is consistent with that previously reported in Australia for multiple sclerosis, another autoimmune disease, as well as type 1 diabetes across latitudinal gradients in the Northern Hemisphere (67). This association agrees with photoimmunologic evidence of UV-induced immunosuppression and may suggest a positive beneficial effect of UV radiation in reducing the incidence of some autoimmune conditions. More studies focusing on immune disorders and personal UV exposure in humans, particularly in the Arctic, are to be recommended.

**UV-B and Viral Interactions**

Herpes simplex virus (HSV) is found in all races, cultures and continents. HSV affects 80-90% of the world’s population. Most people have herpes simplex as either cold sores or as genital herpes. In experimental animal models UV-B has been shown to release immune inhibition on HSV expression and lead to HSV manifestation. A similar effect may also be at work in the human populations (68).

Indigenous populations appear to be predisposed to cancers such as nasopharyngeal and salivary gland cancers (69). These cancers are thought to be associated with the high viral load in these populations, and genetic factors appear to be involved. Salivary gland cancer has been linked to UV exposure in several studies on non-Arctic populations (51) although, again, a latitude gradient has not been demonstrated suggesting UV may act as
a co-factor. One early study examined HSV in a Canadian Inuit population and reported an early age of acquisition and high prevalence of infection (70).

Whether enhanced UV-B due to ozone depletion will exacerbate HSV or other viral infections in Arctic populations remains to be determined.

UV-B and vitamin D
Vitamin D (calciferol) is a fat-soluble vitamin (for a comprehensive review on the mechanism of Vitamin D formation see (71)). The major biologic function of vitamin D is to maintain normal blood levels of calcium and phosphorus. Vitamin D aids in the absorption of calcium, helping to form and maintain a strong skeletal structure. Without vitamin D, bones can become thin, brittle, soft, or deformed. Vitamin D prevents rickets in children and osteomalacia in adults, which are skeletal diseases that result in defects that weaken bones. In some new studies, Vitamin D has been linked to cell regulatory properties. Interestingly, a recent study reports a link between UV-B exposure and synthesis of calcitriol, (vitamin D hormone) in human skin, a first of its kind (72).

Vitamin D is found in food, but also can be made in human skin after exposure to UV-B from the sun. In general, skin synthesis provides most of the vitamin D to the body (80%–100%) (73) and with adequate sunlight exposure dietary vitamin D may be unnecessary (74). Many factors can affect vitamin D production such as season of the year, latitude, age, skin color, time spent outdoors, and sun angle. Few foods contain significant amounts of vitamin D that can act as a substitute for sunshine exposure. Fish with a high fat content such as sardines, salmon, herring and mackerel are excellent sources of vitamin D. Other sources of importance are meat, milk and eggs, and fortified foods. Fortified foods, however, may not be sufficient to preclude sunlight exposure. In one study it was reported that dark-skinned, veiled, pregnant women, their infants and elderly in residential care had the highest vitamin D deficiency in subjects studied (75). This suggests that vitamin D deficiency may be a bigger risk factor in populations world-wide when factors reducing exposure to sunlight are considered (e.g. Arctic populations during long, dark winters). Clearly, a balance is needed between sunshine exposure and the increased risk of excessive sunlight leading to skin cancer or other UV-B health effects and not enough sunlight exposure leading to vitamin D deficiency for diseases such as rickets (76, 77). The vast potential array of biological activity for vitamin D, in addition to the well-known calcium/bone connection, would indicate that studies in arctic communities on UV-B induction of vitamin D in liver and kidney and now, vitamin D hormones in skin, should be given high priority. This takes on additional significance given the 6 months of darkness in winter followed in Spring/Summer by potential excess exposure to UV-B due to ozone depletion in Arctic communities. The consequences of this are not known.

SUMMARY OF RELATED IMPACTS
A lack of in-depth information on health effects due to increased UV-B in the Arctic precludes any in-depth evaluation of risk assessment at present. Skin cancer appears to be a low risk phenomenon in the Arctic partic-
ularly in indigenous populations. However, with the recent discovery that UV-B is the active waveband for melanoma induction (14) one may reasonably ask how long will such low risk be maintained? Both indigenous and non-indigenous populations should be monitored routinely for skin cancers, cataracts and precursors to such conditions. The effect that UV-B-induced immune suppression, alone or in combination with Arctic stressors and pollutants will have on human or animal populations is unknown. The implications of increased UV-B on the incidence of viral diseases in Arctic populations needs also to be addressed particularly as climate change may introduce viral-carrying insect species, unknown in Arctic climes, under a global warming scenario (see Bradley et al., this edition). UV activation of viruses is well-documented (59, 78-80). Further troubling is the emergence of infectious disease, new and old, in the Arctic (81). Given an association between the lowering of resistance to some infectious diseases by UVB–induced immune suppression in experimental animal systems, (82) and a potential role for cis-UCA (83) (see UVB and immnosuppression above) attention to increases in infectious disease as a consequence of increased UV-B due to ozone depletion needs to be given high priority.

Clearly, population health impacts that might develop in response to increased UV-B, due to stratospheric ozone depletion is both a major concern and a major area for research. What is particularly disconcerting is that the stratospheric ozone layer, over the Arctic, is projected to remain vulnerable for the next several decades even with full compliance of the Montreal Protocol: http://www.unep.org/ozone/pdf/Press-Backgrounder.pdf).

Little doubt now exists that global climate change, and in particular global warming, is a real phenomenon. If suggested links between ozone depletion and global warming turn out to be correct (and presently this is not universally accepted) (1, 2, 4) (see also Figure 1 and the following link: http://www.realclimate.org/index.php?p=151) risk assessment of UV-B induced health impacts to Arctic populations will be even more demanding than previously thought. Aside from the question of atmospheric studies needing more research, the almost completely neglected question of UVB-impacts to human health as related to stratospheric ozone depletion is in even greater need of study (84).

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