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INVITED REVIEW

Climate change and health with an emphasis on interactions with ultraviolet radiation: a review

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Abstract

Climate change is increasingly recognized as a major risk to human health, and health concerns are assuming more importance in international debates on mitigation and adaptation strategies. Health consequences of climate change will occur through direct and indirect routes, and as a result of interactions with other environmental exposures. Heatwaves will become more common and are associated with higher mortality particularly in the elderly and those with pre-existing cardiovascular and respiratory illnesses. Warmer ambient temperatures will result in more dehydration episodes and increased risks of renal disease and, through effects on pollen seasons, there may be an increase in allergic disease such as asthma and hayfever. Other adverse effects including on air quality, food safety and security and an expanding distribution of some infectious diseases, including vector-borne diseases, are postulated. A related but separate environmental exposure is that of ultraviolet radiation (UVR). Interactions between climate change and stratospheric ozone (and the causes of ozone depletion) will cause changes to levels of ambient UVR in the future and warmer temperatures are likely to change sun exposure behaviour. Co-occurring effects on aquatic and terrestrial ecosystems have potential consequences for food safety, quality and supply. Climate change-related exposures are likely to affect the incidence and distribution of diseases usually considered as caused by UVR exposure; and changes in UVR exposure will modulate the climate change effects on human health. For example, in some regions warmer temperatures due to climate change will encourage more outdoor behaviour, with likely consequences for increasing skin cancer incidence. Although many of the health outcomes of both climate change and the interaction of climate change and UVR exposure are somewhat speculative, there are risks to over- or under-estimations of health risks if synergistic and antagonistic effects of co-occurring environmental changes are not considered.

Keywords: adaptation, air quality, cataracts, climate change, food quality and safety, heat, human health, infectious diseases, mitigation, skin cancer, skin cancers, ultraviolet radiation

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Introduction

Climate change has been described as 'the biggest global health threat of the 21st century' (Costello *et al.*, 2009). Some health risks are clear and direct, e.g. the effect of rising temperature on risk of heat stress, whereas others are less well-defined, occurring via indirect pathways and interactions with a range of other factors that will themselves be affected by climate change.

Previously, the two contemporary 'global environmental changes' resulting from anthropogenic activity – climate change and ozone layer depletion – have been considered separately. This is because climate change is largely driven through changes in the radiative properties in the lower atmosphere (troposphere) whilst ozone depletion occurs in the upper atmosphere (stratosphere). However, there is increasing recognition of various ways in which these atmospheric phenomena interact and how they can impact on human health (Fig. 1).

Herein we provide first an overview of the direct and indirect effects commonly considered under 'climate change and health', and then a brief discussion of the health effects of exposure to solar ultraviolet radiation (UVR). The following sections discuss the hitherto relatively neglected interacting effects of UVR and climate change and the potential risks to health.

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Effects of climate change on health

Noncommunicable diseases

Research on the potential consequences of climate change for human health has, to date, tended to focus

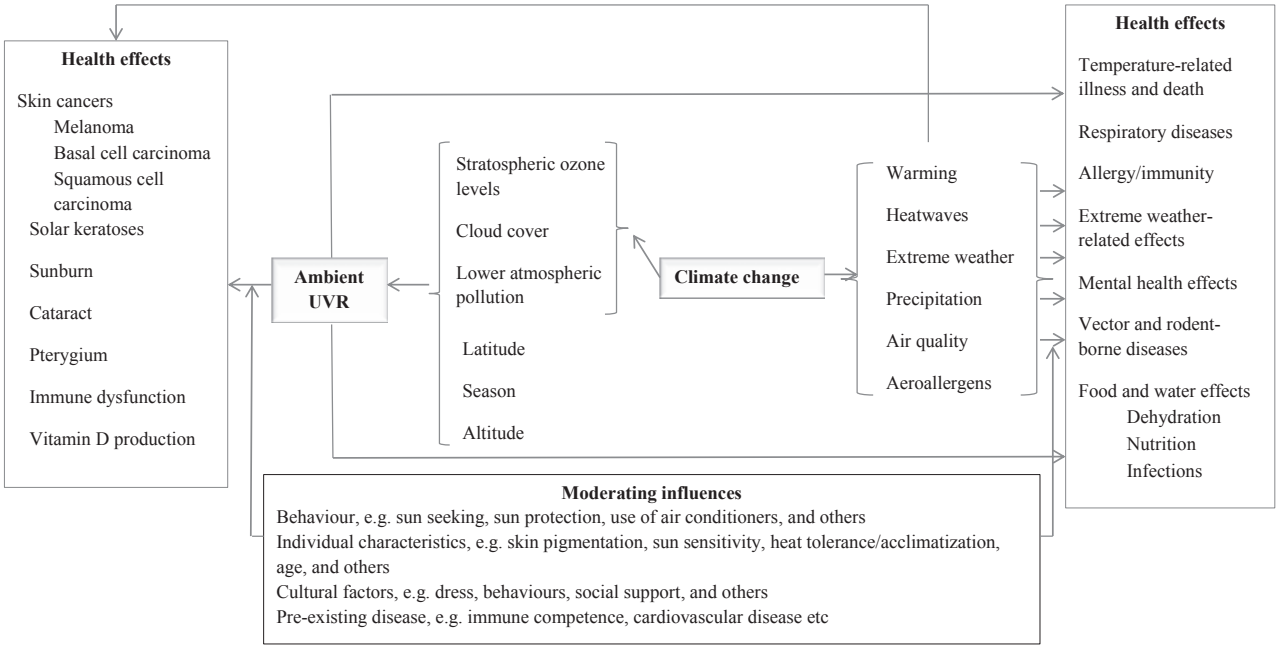

Fig. 1 Summary of the effects of climate change and ultraviolet radiation exposure on human health.

on communicable disease and injuries. However, there are also risks in relation to chronic noncommunicable diseases through direct and indirect pathways (Kjellstrom *et al.*, 2010).

Health effects of rising ambient temperatures. There is a U-shaped relationship between mortality and temperature, with both colder and hotter temperatures increasing death rates, and acclimatization a key moderator (Iniguez *et al.*, 2010). Warmer climatic conditions may decrease cold-related mortality (Haines & Patz, 2004), and reduce respiratory tract infections such as pneumonia and influenza (Harley *et al.*, 2011) but will increase heat-related mortality. The balance of adverse and beneficial effects will vary by region, but the overall weighting will be towards increasing heat-related mortality (Baccini & Biggeri, 2009), under the influence of three correlated drivers: a warmer climate with an increase in heatwaves, increased urban heat island effects, and demographic trends (Luber & McGeehin, 2008).

Healthy adult bodies cope with increases in temperature (to a threshold) using internal heat regulatory mechanisms (Kovats & Hajat, 2008). However, high temperatures can cause heat cramps, heat syncope, heat exhaustion, heat stroke, and death, particularly in unhealthy bodies that are less able to cope with warmer temperatures (Kovats & Hajat, 2008; Luber & McGeehin, 2008). Heat stroke is particularly dangerous, causing permanent damage to multiple body organs and having a high case:mortality ratio (Kovats & Hajat, 2008).

Future climate change scenarios predict an increase in the frequency and length of heatwaves, with associated increases in mortality (Hajat *et al.*, 2006). For example, the 2003 European heatwave resulted in tens of thousands of additional deaths (Hoffmann *et al.*, 2008; Le Tertre *et al.*, 2006; Poumadère *et al.*, 2005), with much of the increase in mortality directly attributed to the health consequences of heat – dehydration, hyperthermia and heat stroke – often compounding pre-existing respiratory and cardiovascular conditions (Poumadère *et al.*, 2005). Heat-exacerbated air pollution (especially ozone) also contributed (Filleul *et al.*, 2006).

Urban centres experience higher temperatures than surrounding suburban and rural areas – the urban heat island effect – because buildings, roads and paved surfaces (at greatest concentration in urban areas) trap heat during the day and additional heat is generated from vehicles, industrial activity and air conditioning (Peng *et al.*, 2011). Urbanization is accelerating across the world (Grimm *et al.*, 2008), increasing both the exposure and the population-at-risk from elevated temperatures.

In most developed countries, a demographic trend towards an ageing population will have marked effects on heat-related mortality. The elderly are more susceptible to the effects of rising temperature (Vaneckova *et al.*, 2008) through not only less efficient internal heat regulation but also social conditions (such as isolation, poorer quality housing, and lack of access to air conditioning) that can decrease the capacity to adapt to

changes in ambient temperature (Kovats & Hajat, 2008).

In addition to these effects on overall mortality, some chronic diseases are likely to become more common as the climate warms, including kidney disease and hospitalizations for acute renal failure (Kjellstrom *et al*., 2010) – as seen during the 2006 California heatwave (Knowlton *et al*., 2008) – and renal calculi, with an additional 1.6–2.2 million new cases by 2050 predicted for the United States of America alone (Brikowski *et al*., 2008). Populations in low and middle income countries will be most at risk, particularly their often large numbers of heat-exposed workers, and bear the greatest burden of renal disease (Kjellstrom *et al*., 2009).

The full range of health impacts of warmer ambient temperatures due to climate change have yet to be fully understood. For example, heat stress reduces psychological performance resulting in more accidental physical injuries (Kjellstrom *et al*., 2010); thus warmer days and more hot days might lead to a rise in physical injuries. Recently, maternal exposure to warmer temperatures at various times during pregnancy has been linked to an increased risk of stillbirth or preterm birth (Strand *et al*., 2012).

Air quality and disease. Air quality (particularly concentrations of ozone and particulates) may be affected by future climate change as the formation and distribution of air pollutants is influenced by a range of weather variables, including temperature and cyclonic systems (see Spickett *et al*., 2011). In some areas, projected changes in air temperature and precipitation are likely to increase both the severity and frequency of fire events, which in turn lead to degraded air quality (Confalonieri *et al*., 2007). Ozone is a well-known respiratory irritant; exposure is associated with acute asthma attacks, hospital admission and premature mortality (Confalonieri *et al*., 2007; Kinney, 2008). Globally, anthropogenic ozone has been estimated to cause 700 000 premature deaths per year (Anenberg *et al*., 2010). Exposure to particulate matter is linked to increased risk of cardiovascular and respiratory diseases, including tuberculosis (Lin *et al*., 2007) and higher morbidity and mortality.

Aeroallergens and disease. Exposure and risk of reactions to common aeroallergens (e.g. ragweed), is likely to increase in the future: due to effects of climate change on lengthening the pollen season and the quantity of pollen produced (Beggs, 2004; Ziska *et al*., 2011) and because increasing atmospheric CO_2 concentrations will stimulate photosynthesis and plant growth, increasing the production, allergenicity and distribution of plant-based aeroallergens (Ziska & Beggs, 2012). The aeroallergen-related diseases of greatest concern are allergic asthma and allergic rhinitis.

Mental health. Climate change is now weakening or disturbing many of the social, economic and environmental conditions that underpin mental health (Fritze *et al*., 2008). More pronounced variability in weather, with extreme weather events, is likely to further compound risks to mental health (for example, see Berry *et al*., 2010). For example, 33% of Florida residents affected by Hurricane Andrew showed evidence of post traumatic stress disorder (PTSD) 4 months later (76% reported at least one PTSD symptom cluster) (Ironson *et al*., 1997). Such events may also exacerbate ongoing mental health problems (Berry *et al*., 2010) and promote uncertainty and anxiety even amongst those without pre-existing illness. Increased flood events are predicted to occur in regions where rainfall increases in a flood-prone area (Meehl *et al*., 2007), and flooding has been shown to have long-term effects on mental health (Kovats *et al*., 1999).

Higher ambient temperatures as a result of climate change will reduce people's capacity to carry out physical work, leading to a loss of productivity at the population level and a loss of income at the household level with possible mental health risks for those unable to provide for themselves and their families (Berry *et al*., 2010). Hotter weather is associated with an increase in hospital admissions for dementia; mood affective disorders; neurotic, stress-related and somatoform disorders; disorders of psychological development; and senility (Hansen *et al*., 2008). Both hotter weather (Page *et al*., 2007) and prolonged drought (Nicholls *et al*., 2006) have been associated with an increased risk of suicide. As with other health effects of climate change, risks to mental health will be felt unevenly, with already disadvantaged populations generally at greatest risk (see Kjellstrom, 2009; Fritze *et al*., 2008).

Communicable diseases

Consequences of climate change, such as sea level rise and changes in humidity, rainfall and temperature, have the potential to affect the incidence of infections. Human factors, such as the mass movement of people and animals that might occur because of a changing climate will also mediate future patterns of infectious diseases (Patz *et al*., 2005).

Vector-borne diseases. The survival, reproduction and distribution of vectors are dependent on many factors including habitat destruction, land use, pesticide application, host density and climate (Semenza & Menne, 2009). For dengue, the most common arboviral

infection, temperature is an important factor for its transmission in urban areas (Semenza & Menne, 2009). Climate change, and in particular a warmer climate, may increase the available suitable habitat for the principal vector *Aedes aegypti* (Zhang *et al*., 2008), although other climate and non climate-related factors are also important, e.g. the prevalence of water tanks, use of air conditioning and urban density (Russell *et al*., 2009). Climate-suitable areas for dengue transmission will have a disproportionately greater impact on health in areas where there is urban poverty, than in those more economically developed places where surveillance and control measures can limit transmission (Van Kleef *et al*., 2010).

Malaria occurs in more than 92 countries and modelling studies predict that an additional 300 million and 150 million people could be exposed to *Plasmodium falciparum* and *Plasmodium vivax* malaria, respectively, as a result of future changes in the number of areas suitable for the spread of the vector and transmission of the disease (Martens *et al*., 1999). Most studies have found that the incidence of malaria is strongly (positively) related to temperature (Zhang *et al*., 2008). However, the disease dynamics of malaria are complex and non climatic factors will also affect future rates of malaria infections and infection outcomes (Confalonieri *et al*., 2007). The difficulty of incorporating non-climatic factors such as socio-economic status and the effectiveness of malaria-control programmes within modelling makes it difficult to establish precisely how malaria transmission will be affected by climate change at both the local and global scales (Confalonieri *et al*., 2007).

The effect of a warming climate on the distribution of tick-borne diseases is similarly uncertain. Modelling suggests that some tick-borne disease (e.g. tick-borne encephalitis) will in future be found in higher altitudes and latitudes (Randolph, 2001), and research has shown that tick-borne encephalitis has increased since the mid-1980s as a result of milder and shorter winters (Lindgren *et al*., 2000). However, tick-borne disease is dependent upon a host of causal pathways, not just temperature, so that there will be variation from place to place (Semenza & Menne, 2009). For example, milder winters might enable the expansion of Lyme borreliosis into higher altitudes, but such an expansion is dependent on host vertebrate species also shifting their population distribution (Semenza & Menne, 2009). In other places its distribution will decrease because of increases in droughts and severe floods (Semenza & Menne, 2009).

A dramatic increase in the potential transmission zone for the disease schistosomiasis has been postulated due to climatic warming. Zhou *et al.* (2008) estimated that a northern latitude movement of the freezing zone, due to climate warming, will allow the survival of the snail intermediate host of the parasite *Schistosoma japonicum*, to cover a further 8.1% of the Chinese land mass (Zhou *et al*., 2008).

Rodent-borne diseases. The potential effects that a changing climate will have on infectious diseases transmitted by mammals to humans have received less attention than vector-borne diseases. Previous research has indicated a possible link between drought followed by El Nino-driven heavy rainfall and the emergence in the southwest of the United States of hantavirus pulmonary syndrome (Glass *et al*., 2000). Infections with the virus follow human contact with excrement (urine, saliva, faeces) from rodents, e.g. deer mice. Climatic events such as periods of increased precipitation or drought affect food availability for rodent populations (Gubler *et al*., 2001). Warmer winters are likely to lead to increased rodent survival and areas that experience a decrease in precipitation might see rodents move into housing areas, thereby increasing the potential for human contact (Haines & Patz, 2004). These changing climatic dynamics mean that there is a potential for an increase in outbreaks of rodent-borne diseases such as hantavirus.

Water-borne disease. Climate change is predicted to alter rainfall patterns and the availability of surface water, and lead to a deterioration of water quality and an increase in water stress for some areas. Such changes are likely to result in increasing incidence of water-borne diseases, including diarrhoea (Confalonieri *et al*., 2007; Hitz & Smith, 2004). Nevertheless, the actual impacts of climate change on the incidence of water-borne diseases will depend on the success of implementation of prevention and control measures, including improved water supply and sanitation measures. Notably such systems can come under particular stress during extreme weather events (Wilbanks *et al*., 2007). For example, where flooding occurs, infrastructure (e.g. clean water supply, sewerage and electricity) can fail, increasing the risk of exposure to water-borne diseases such as cholera (Confalonieri *et al*., 2007).

Food systems

Food safety and quality. Climatic conditions are expected to become more variable, with extreme weather events (floods, heat waves, prolonged droughts, cyclones) occurring more frequently and with increased severity (Confalonieri *et al*., 2007). Such events have the potential to disrupt food supplies, affect regional food security (Schmidhuber & Tubiello, 2007) and to adversely affect the safe use of food.

Diarrhoea is the second leading cause of death amongst children under five: around 1.5 million deaths per year (UNICEF, WHO, 2009). Higher ambient temperatures have been associated with increased incidence of diarrhoeal disease and more frequent outbreaks of food poisoning such as salmonellosis (for example, Hijoka *et al.* in Hitz & Smith, 2004). In one study in the Pacific Islands a 1 °C rise in temperature was associated with a 3% increase in diarrhoea incidence in infants (Singh *et al.*, 2001). Predicted climate change, with increased temperature and more frequent excessive rainfall events, is thus likely to increase the risk of outbreaks of diarrhoeal diseases of both food-borne and water-borne origins (Edwards *et al.*, 2011; Schmidhuber & Tubiello, 2007).

Food security. Healthy and nutritious food is fundamental to good health. Food production is highly dependent on climate, and variations in precipitation, temperature and solar radiation have the potential to significantly disrupt food production. The effects of climate change on food production are likely to be geographically uneven: in general, developed countries seem set to benefit from increased crop yields whereas developing countries are likely to see crop yields decrease (Parry *et al.*, 2004; Rosenzweig & Parry, 1994). An unstable and less effective food system will leave some people, predominantly in the developing world, in danger of increased hunger and malnutrition and therefore more susceptible to disease pressures.

Increased disruption to global food supply chains will result in higher food prices, leaving many people unable to afford enough nutritious food to keep them healthy. Already researchers are attributing disruption of global food systems to climate change (for example, see Darnton-Hill & Cogill, 2010). The historical evidence shows us that periods of unprecedented seasonal heat cause damage to food systems and lead to food shortages and higher food prices (Battisti & Naylor, 2009), for example during the food crisis of 2006–2008 (see Godfray *et al.*, 2010). Climate change is likely to lead to disruption occurring on a more frequent basis and in regions most vulnerable to fluctuations in food supply and food cost. The continued growing global population and the increased levels of consumption will ensure that future demand for food will increase for at least another 40 years (Godfray *et al.*, 2010), leading to a likely increase in the number of people at risk of hunger (Schmidhuber & Tubiello, 2007).

Immune function

The complex, multi-layered mammalian immune system acts to protect the host from pathogen and aberrant-cell induced disease. Suppression of the immune system predisposes individuals to infection and cancer, whereas over-activity can lead to autoimmune disease and allergy. The immune system is sensitive to numerous endogenous and exogenous factors – including under-nutrition (Chandra, 2002), physiological (McEwen, 1998) and psychological stress (Glaser & Kiecolt-Glaser, 2005) and UVR exposure (Hart *et al.*, 2011). Many of these exposures are also sensitive to global climate change (e.g. food insecurity, heat stress, post traumatic stress following extreme weather events), as has been detailed above. Given the critical role of the immune system in maintaining the primacy of the 'healthy state' over the 'diseased state' at an individual level, immune system-modulating environmental exposures felt at a population level could have significant public health ramifications. This will be particularly relevant to the already vulnerable populations of the developing world.

Health effects of UVR exposure

Herein we provide a brief summary of the effects of UVR exposure on human health to provide context for later sections. A more detailed review is provided elsewhere (Norval *et al.*, 2011).

Solar UVR incident on Earth's atmosphere is comprised of UVC (wavelength 100–280 nm), UVB (280–315 nm) and UVA (315–400 nm) (Diffey, 2002). UVC and most UVB (>90%) is absorbed in the stratosphere, particularly through reactions involving ozone. Ambient UVR levels and the relative amounts of UVA and UVB vary according to geographical position, season, time of day and altitude due to variation in the path-length through the atmosphere. Cloud cover and lower atmospheric (tropospheric) pollution as well as reflection from surfaces (albedo) (e.g. snow) further modulate ground-level UVR (both attenuating and magnifying). Human sun exposure behaviour further modifies the UVR dose received. Worldwide, outdoor-working adults receive about 10%, and indoor working adults and children about 3%, of available ambient UVR (Godar, 2005). Across a population, this dose can vary widely, from one tenth to ten times the mean value (Gies *et al.*, 1999). Darker skin pigmentation and clothing habits further alter the received biologically effective dose of UVR.

Effects on the skin, eyes and immune system

Direct effects on health from UVR exposure primarily involve the skin and eyes, and through them, the immune system. The UVB wavelengths are thought to be most damaging, although UVA may be important in

some skin cancers (Wang *et al*., 2001) and for immune suppression (Norval & Halliday, 2011). Both exposures and health outcomes can be separated into short-lived (acute) and long-term (chronic) time frames. Acute responses to high dose UVR exposure include DNA damage, inflammation and cell death – manifest as sunburn in the skin and photokeratitis and photoconjunctivitis in the eye (WHO, 1994). Immune suppression occurs and may allow the reactivation of latent viruses (e.g. herpes simplex, leading to the development of 'cold sores' on the lips) (Norval & Halliday, 2011).

Recurrent acute exposures or chronic UVR exposure also cause DNA damage and immune suppression resulting in the development of skin tumours: solar keratoses (common premalignant lesions capable of spontaneous regression), basal and squamous cell carcinomas (the non-melanoma skin cancers, NMSC) and cutaneous malignant melanoma (Armstrong & Kricker, 2001). The largely nonlethal NMSCs are the most common cancers in many countries, whereas malignant melanoma is less common, but more lethal. Incidence of all forms of these UVR-induced skin tumours continues to increase in many countries, despite strong public health sun protection programmes (Brewster *et al*., 2007; Coory *et al*., 2006; Richardson *et al*., 2008; Van Hattem *et al*., 2009). In the eye, chronic UVR exposure causes pterygium (Threlfall & English, 1999) which may contain pre malignant foci (Chui *et al*., 2011), carcinomas of the cornea and conjunctiva (Sun *et al*., 1997), some types of cataract (Norval *et al*., 2007) and ocular melanomas of superficial eye tissues (Norval *et al*., 2011). Ocular malignancies are uncommon, but pterygia and cataracts are extremely common (McCarty *et al*., 2000, 1999), particularly in high ambient UVR settings and, untreated, can cause blindness. UV irradiation is potently immunosuppressive in animal models (Hart *et al*., 2011), but the effects of chronic UVR exposure in humans at doses relevant to daily activity is less obvious. Immunosuppressed hosts (e.g. those with HIV/AIDS, organ transplant recipients) are at greatest risk of UVR-induced infections (i.e. Human papilloma virus, herpes infection) (Sleijffers *et al*., 2002) and decreased vaccine efficacy among children immunized in summer seasons is described (Norval & Woods, 2011). Conversely, there is growing evidence that higher UVR exposure is linked to decreased risk of some autoimmune diseases [e.g. multiple sclerosis (Lucas *et al*., 2011)], although a causal association is not yet proven.

UVR-induced production of vitamin D and other beneficial effects

Skin exposure to UVB radiation initiates the endogenous synthesis of vitamin D, and this is the primary source of vitamin D in many regions of the world (Holick, 2003). Vitamin D deficiency is a risk factor for bone diseases (rickets in children, osteomalacia and osteoporosis in adults) and may also increase the risk of cancers (with the evidence strongest for colorectal cancer (Gandini *et al*., 2011)) and autoimmune (particularly multiple sclerosis and type 1 diabetes (Ponsonby *et al*., 2005)), cardiovascular (especially hypertension (Holick, 2005)) and metabolic (e.g. type 2 diabetes and the metabolic syndrome (Gagnon *et al*., 2011)) diseases.

Solar UVR is the most important natural germicide in the environment (Davies *et al*., 2009; Sagripanti *et al*., 2009). In both shallow sea-water and fresh river water, sunlight inactivates disease-causing micro-organisms: *Cryptosporidium parvum*, the causative organism for the diarrheal disease cryptosporidiosis (Connelly *et al*., 2007; King *et al*., 2008); *Burkholderia pseudomallei,* the causative organism for melioidosis (a disease with high mortality in some situations or particular groups) (Sagripanti *et al*., 2009); *Escherichia coli* and enteric viruses (Sinton *et al*., 2002). Many communities in rural and remote regions rely on surface waters and groundwater for their domestic and agricultural needs.

Interactions of climate change and UV radiation

In the 1970s the risks and then the reality of depletion of stratospheric ozone by anthropogenic emissions of chlorofluorocarbons (CFCs) were recognized. Rapid international action (through the Montreal Protocol and its amendments) to replace ozone depleting substances (ODSs) was driven by fears of large increases of biologically damaging UVB radiation reaching Earth's surface with consequences for human health and environmental damage. Although some increases in UVB have been observed (McKenzie *et al*., 2011), the effectiveness of the Montreal Protocol is evidenced by the lack of major health and environmental effects. Estimates of the 'world avoided' (Newman *et al*., 2009) remind us of the potential risks of inaction. Recent models indicate recovery of stratospheric ozone to at least 1980 levels in most regions by the end of the 21st century (McKenzie *et al*., 2011).

Global climate change, through chemical and dynamical influences, will alter the trajectory of recovery of stratospheric ozone (and thus UVB levels at Earth's surface) with specific effects variable by region (McKenzie *et al*., 2011). For example, lower stratospheric temperatures resulting from climate change are expected to decrease the rate of chemical destruction of ozone outside of polar regions but, through an increase in polar clouds, to enhance ozone loss in polar regions (McKenzie *et al*., 2011). Changes in precipitation, cloud cover and aerosols (e.g. air pollution associated with

the burning of fossil fuels) as a result of climate change or mitigation activities will alter both ground-level UVR and its spectral composition, i.e. it will affect both UVA and UVB levels. Loss of snow cover and sea ice will reduce surface albedo and reflected UVR.

There are bidirectional effects of climate change and stratospheric ozone depletion. Ozone depleting CFCs were also greenhouse gases – the phase-out of these chemicals through the Montreal Protocol has thus already provided significant protection from global warming although the trajectory of this effect is now weakening. Replacement chemicals are also greenhouse gases, albeit with lower global warming potential (McKenzie *et al.*, 2011). Depletion of stratospheric ozone may have contributed to cooler-than-expected Antarctic temperatures. However, as stratospheric ozone recovers, melting of the west Antarctic ice sheet may accelerate (Shindell & Schmidt, 2004) and, as sea ice retreats and snow cover lessens, exposed surfaces will absorb a greater fraction of incoming solar energy and contribute to warming. Photodegradation involving both UVA and UVB radiation is important to litter decomposition in arid and semi-arid ecosystems (~40% of Earth's land surface). Changes in ambient UVR could alter the carbon sequestration potential of terrestrial ecosystems and CO_2 losses to the atmosphere (Ballare *et al.*, 2011).

The most recent predictions taking account of recovery of ozone depletion and climate change scenarios, suggest that by the end of the 21st century, erythemal UVR (the combination of UVA and UVB weighted towards effectiveness at causing erythema of the skin) will decrease by 9% in northern high latitudes, but increase by 4% in the tropics and up to 20% in southern high latitudes in late spring and early summer (Hegglin & Shepherd, 2009). However, recent models that include predicted changes in cloud cover suggest there will be further reduction in erythemal UV of ~10% at northern high latitudes (due to increasing cloudiness) and an additional increase of ~3–6% in the tropics (McKenzie *et al.*, 2011).

Health effects of climate change and UV radiation

Research to date on the health effects of the co-occurrence of climate change and changes in ambient UVR is scanty, requiring a broad range of inputs: predictions of temperature, precipitation and clouds from climate models and of UVR scenarios with inclusion of ozone effects; consequential changes in human behaviour; and modelling the combined effects on the distribution of exposure and human health outcomes. Herein we first consider the moderating effect of UVR exposure on the climate change-related health outcomes noted in the first section of this article. We follow by considering how the environmental consequences of climate change might affect the UVR-related health outcomes reviewed in the second section of this article.

Modulation of climate change-induced health outcomes by UVR exposure

Air quality and disease. UVR acts upon NO_x produced in motor vehicle exhaust gases to form photochemical smog, which includes ozone, peroxyacetylnitrate (a potent eye irritant) and aerosols. Tropospheric ozone concentrations are predicted to increase by 13–31%, particularly in polluted regions (Shindell *et al.*, 2008) several of which are at low latitude where ambient UVR levels are likely to also increase (McKenzie *et al.*, 2011).

On the other hand, UVR also initiates the production of hydroxyl radicals, that 'clean' the atmosphere by converting major air pollutants (e.g. methane) into more water-soluble forms that are removed in precipitation (Tang *et al.*, 2011). Stratospheric ozone depletion may have led to an approximate 3.5% increase in the concentration of hydroxyl radicals (Shindell *et al.*, 2006), but for the period from 1900 to 2100 a (regionally variable) decrease of ~20% is predicted (Wild & Palmer, 2008). The future balance of UVR-induced production of photochemical smog and of hydroxyl radicals, with adverse and beneficial effects, respectively, on ambient air quality, will have important flow-on effects for human health.

UVR is involved in the formation and destruction of organic aerosols from volatile organic compounds released from plants and other organisms; in turn aerosols can scatter and absorb incoming solar UVR, modify the optical properties of clouds, their precipitation efficiency and lifetimes, thereby altering the ground-level UVR (Tang *et al.*, 2011). Any reductions in ambient UVR, as noted above, may result in reductions in the UV-induced breakdown of both organic and inorganic contaminants in the atmosphere and in surface waters (e.g. mercury, copper, pesticides), that may increase or decrease the toxicity of pollutants (depending on the toxicity of the photoproduct compared to the parent compound) and their dispersion (Zepp *et al.*, 2011).

Aeroallergens and disease. In the United States of America, lengthening of the ragweed season has been greater at higher latitudes, in keeping with projections of greater rises in global temperatures at higher latitudes and altitudes (IPCC, 2008) One consequence of lower UVR levels predicted for higher latitude regions in the future would be a decrease in UVR-induced dampening of the allergic response (Hart *et al.*, 2011; Hollams

et al., 2011), potentially exacerbating the adverse effect of climate change on allergic disease.

Vector-borne diseases. UVB irradiation inhibits the effectiveness of two strains of a bacterium (*Bacillus spaericus)* that is used as a biopesticide to control mosquito larvae (Hadapad *et al*., 2008). Risks from mosquito-borne diseases are largely confined to low latitude locations where UVB levels are predicted to increase over the next century – consideration of these interacting effects of different environmental factors will be important to health protection in these regions.

Water-borne and food-borne disease. UVB irradiation increases mortality amongst juveniles of the snail that is the intermediate host of *Schistosoma mansoni* (causing schistosomiasis or bilharzia) (Ruelas *et al*., 2006), and adverse effects of UVB on other stages of *Schistosoma* are also described (Ariyo & Oyerinde, 1990; Ruelas *et al*., 2007). Nevertheless, any effect in natural systems will depend not only on ambient UVB but also on water transparency for these wavelengths. As previously noted *Cryptosporidium parvus* is deactivated by UVR exposure, so that decreases in UV transparency, as well as changes in temperature and pH, of surface drinking water supplies as a result of climate change may favour survival of this human pathogen (Connelly *et al*., 2007).

There is some evidence to support these interactions. Paediatric (<6 year) emergency department visits for gastroenteritis in Sydney, Australia increased with higher maximum daily temperature, but decreased in relation to the UV index (Lam, 2007), a pattern possibly explained by poorer survival of the infecting organism in higher ambient UVR conditions.

In Philadelphia, seasonal variation in the incidence of invasive pneumonia (caused by *Streptococcus pneumonia)* and of invasive meningitis was better correlated with the UV Index than with temperature (Kinlin *et al*., 2009; White *et al*., 2009). Possible contributing factors include impaired innate immunity due to lower vitamin D status in winter (Norval *et al*., 2011) and UVR effects on the infectivity or transmission of the organism (Kinlin *et al*., 2009), although the major explanation of the seasonal pattern remains likely to be increased close contact indoors.

Food safety and quality and food security. Climate change may benefit crop production in arctic and sub-arctic regions as environmental conditions become milder and more hospitable (McBeath, 2011). However, in these same regions, UVR levels are predicted to decrease (McKenzie *et al*., 2011). Attenuation of UVB irradiation is associated with increased intensity of herbivory by insects (twofold increase with a 25% reduction in UVB irradiance (Ballare *et al*., 2011)) and UVR exposure may decrease plant diseases through its antimicrobial effects. Meanwhile higher UVB levels are associated with a modest decrease in plant biomass (1% reduction for each 3% increase in weighted UVB irradiance (Ballare *et al*., 2011)). Thus, the net effect on crop production at higher latitudes of the combined influences of changes in climate and UVR is complex – but both influences must be considered.

Enhanced UVB irradiation improved water economy in one plant species (*Mediterranean pines*), alleviating the adverse effects of drought (Manetas *et al*., 1997) but it is not clear how generalizable this finding is, particularly to crop species. Nevertheless, the finding may have importance in low latitude regions where UVR is expected to increase. Furthermore, there is considerable uncertainty over the effects of climate change on vegetation cover and thus UVR penetration. Changes are likely to be latitude dependent, with a decline in woodland and reduced vegetation cover (i.e. increased UVR penetration) in low and mid-latitude regions, but increased forest and woodland at northern high latitudes (i.e. lower UVR penetration) (Zepp *et al*., 2011).

Tropospheric ozone (see above, related to both UVR and vehicular emissions) causes crop damage, with losses estimated currently of 3.5–6.1 billion dollars annually (Murphy *et al*., 1999) but forecast to be ~$20 billion by 2030 (Tang *et al*., 2011; Van Dingenen *et al*., 2009).

Exposure to UVB radiation has generally negative effects on aquatic plants and animals (Hader *et al*., 2011), potentially affecting the quantity and quality of nutrients and human food supplies. However, UVR effects are dependent on the location and mobility of the organism and the transparency of the water column, with the latter affected by dissolved materials, the density of suspended particles, the concentration of phytoplankton and the depth of the surface mixed layer (Hader *et al*., 2011). In the clearest waters, UVR at 320 nm can penetrate as deep as 84 m (Tedetti *et al*., 2007). Warming temperatures, changes in precipitation, melting of sea ice and an increase in storm events under climate change conditions will alter surface mixing depths, possibly increasing UVR exposure to aquatic organisms living in the upper layers (Hader *et al*., 2011). As sea ice melts, organisms accustomed to living under it will be exposed to higher doses of UVR than previously, whereas those living above it will receive lower UVR doses due to decreased surface albedo and reflection. Furthermore, although many aquatic organisms have developed protection strategies against UV-induced damage, climate change effects such as acidification due to increased atmospheric CO_2 concentrations may compromise such strategies [e.g. calcification in

molluscs, reviewed in (Hader *et al.*, 2011)], with flow-on effects into the food web.

The net effects of combined climate change and UVR exposure on individual aquatic organisms is not clear, but consideration at the ecosystem level is also important. These environmental changes may have marked effects on community and trophic level structure (e.g. shifts in species composition as a result of higher sea surface temperatures, shorter sea-ice seasons and deeper migration of organisms to avoid UVR exposure) that are also important (Hader *et al.*, 2011).

Modulation of UVR-related health outcomes by climate change effects

Effects on the skin, eyes and immune system. Ambient UVR levels are predicted to increase by 2100 at low latitudes where they are already high and decrease at higher latitudes where they are already low (McKenzie *et al.*, 2011). Based only on this change, risks of the adverse effects of UVR exposure, i.e. skin cancers, cataracts, will increase in the tropics. Importantly, however, in humans, sun exposure behaviour is a key modifier of the received dose of UVR. There are less data on the effect of changes in temperature on sun exposure behaviour and intuitively we would expect that this will vary according to the usual temperature to which we are acclimatized. In an Australian study, the risk of sunburn increased with increasing ambient temperature (over three times more likely to be sunburned with temperatures >28 °C) (Dobbinson *et al.*, 2008) but at 'hotter temperatures' people sought refuge indoors. Warmer ambient temperatures due to climate change are likely to be associated with less clothing to protect skin and more time outdoors – thus increasing the risks of the adverse effects of sun exposure at least in some regions. Warmer ambient temperatures may also accelerate skin cancer genesis (Boukamp *et al.*, 1999; Van Der Leun & De Gruijl, 2002; Van Der Leun *et al.*, 2008) but additional data are required to quantify this effect and to evaluate its importance. Increasing urbanization (Department of Economic & Social Affairs, 2010) or migration to higher latitude regions may be associated with lower UVR exposure and related health risks (McCarty *et al.*, 2000).

Dehydration has been implicated as a risk factor for cataract in developing countries (Minassian *et al.*, 1989) and dehydration episodes are likely to become more common in hotter, climate change-induced conditions. Diminishing snow cover in some areas, with decreased surface albedo and thus dose to the eye of reflected UVR may be beneficial, as reflection bypasses the natural sun protection afforded by the eyebrow ridge (Norval *et al.*, 2007). Cataract risk may be increased with chronic exposure to higher ambient temperature (Sliney, 1986), although this has not yet been confirmed. The net effects for UV-related eye diseases of environmental changes associated with climate change, including warmer temperatures and changes in cloud cover and precipitation, will vary by location.

Higher temperatures and humidity, as well as increased UVR predicted for the tropics in the future (Ilyas, 2007), could result in immune suppression with adverse effects on protection against vaccine-preventable diseases, other infectious diseases and skin cancers. In higher northern latitudes, decreased levels of UVR-induced suppression of autoimmunity may result in higher incidence of relevant autoimmune diseases, e.g. multiple sclerosis and type 1 diabetes (Ponsonby *et al.*, 2005). Alternatively, in these regions, warmer temperatures that encourage more time outdoors could increase sun exposures, ameliorating these effects. For multiple sclerosis warmer temperatures exacerbate symptoms and may precipitate relapses through non-immune mechanisms (Kjellstrom *et al.*, 2010).

UVR-induced production of vitamin D

Vitamin D deficiency is more common at higher latitudes where ambient UVR levels are lower (Hagenau *et al.*, 2009). Lower ambient UVR predicted for higher northern latitudes may increase vitamin D deficiency in these regions, although this may be balanced by more efficient vitamin D production at higher ambient temperatures (Tsiaras & Weinstock, 2011). Nevertheless, increasing prevalence of vitamin D deficiency in these regions could exacerbate the already high incidence of multiple sclerosis and other autoimmune diseases, unless other factors, such as warmer temperatures encouraging time outdoors, are compensatory. Urban migration from rural areas may result in lower sun exposure and vitamin D (Mckinley *et al.*, 2011), with consequent risks to health (Gross, 2002).

Mitigation and adaptation

There are two main strategies for countering the adverse (and promoting the positive) health and ecological effects of climate change – mitigation and adaptation. 'Mitigation' aims to limit the magnitude of climate change by reducing atmospheric emissions of greenhouse gases from anthropogenic sources (i.e. industry, motor vehicles and agriculture). Such strategies are most effectively driven and enforced at an international, national or regional level and have been the impetus behind the establishment of multi-national agreements (i.e. the Kyoto Protocol). To meet agreed targets, many countries have introduced strict emissions regulation

legislation and/or 'priced' carbon dioxide emissions via taxation or emissions trading schemes (Ellerman, 2008). 'Adaptation' aims to reduce impacts of climate change that have already occurred or are projected to occur. Given that it is not possible to mitigate against all projected changes to climate (some have already occurred, and others are already 'locked in' because of the long atmospheric half-life of greenhouse gases) or adapt to the full consequences of unmitigated climate change, the challenge is to develop complementary mitigation and adaptation options.

Mitigation strategies

Health co-benefits of mitigation activities are now being promoted (Haines *et al*., 2009). However, it is also important to consider potential health risks of such activities. We briefly discuss two examples below.

1. *Light bulbs, cataract and skin cancer*: Many countries have moved to replace energy-inefficient incandescent light bulbs with compact fluorescent lights (CFLs) that use 75–80% less electricity for an equivalent amount of light (Javorniczky *et al*., 2011; Walls *et al*., 2011). However, CFLs emit low levels of UVR, particularly single envelope lamps (Javorniczky *et al*., 2011). A recent estimate suggested that use of CFLs in Australia could result in 2970 additional annual cases of cataract and 7480 additional pterygia (Walls *et al*., 2011). Similar concerns have previously been raised about increased risks of squamous cell carcinoma (SCC) from close use of fluorescent lighting, e.g. in desk lamps (Sharma *et al*., 2009). Nevertheless, in assessing possible health risks, care needs to be given to both the actual received dose and the wavelength distribution of the exposure. In desk lamps, the output is primarily UVA, whereas UVB is thought to be most important for SCC development. These studies do, however, provide a warning of the need to consider the breadth of effects of new technologies – beneficial for climate change mitigation but possibly adverse for human health.

2. *Atmospheric seeding*: One major type of proposed geo-engineering solution to limit the effects of greenhouse gas accumulation in the short-term involves limiting planetary solar radiation reaching Earth's surface through the use of space-borne reflectors or injection of aerosols, e.g. sulphur particles into the stratosphere (Fox & Chapman, 2011; Izrael *et al*., 2009). Such approaches could provide 'shade' for the planet and buy time for mitigation activities, but limiting visible light as well as UVR will have widespread effects on human health (e.g. possible great increase in diseases of vitamin D deficiency, but decrease in sunburn events) and ecosystems (including plant-food yields).

Formulating appropriate adaptation options

The design and prioritization of adaptation strategies will largely depend on the population's underlying level of vulnerability to climate change. Vulnerability

Table 1 Hierarchy of public health adaptation responses

Adaptation response	Definition	Examples	Enabling agencies/tools
Zero-order (Mitigation)	Mitigation of greenhouse gas emissions	• Mandating improved fuel efficiency of new cars • Promote renewable energy sources • Implement financial impost of CO_2 emissions	• International agreements • Government policy • Private industry
Primary	Reducing *exposure* of populations to climate change and its environmental impacts	• Redesign/modification of cities to lessen the urban heat island effect • Improve barriers against floods • Improve irrigation Promote education of 'sun smart' behaviour to reduce UVR-associated morbidity	• Government policy • Development partners • Urban designers • Public health authorities
Secondary	Reducing the *health impact* of climate change exposures	• Strengthening surveillance and eradication programmes for vector-borne infections • Early warning and response systems for floods and storms • Improving education of villagers for prevention and management of common diseases • Improved services to urban poor	• Government ministries • Development partners • Academia • Community level health and education centres
Tertiary	*Managing* the adverse health outcomes of climate change	• Effective management and treatment programmes for disease (i.e. malaria) • Rapid emergency response capability for natural disaster or disease outbreak	• Government ministries • Community level health facilities

depends on exposure to climate variables (i.e. heat waves, UVR, extreme weather events), sensitivity of the population to those exposures (i.e. poverty, poor housing, extremes of age, dependence on subsistence agriculture) and 'adaptive capacity' (ability to adjust practices, processes or structures to moderate or offset potential damage and take advantage of opportunities presented by the consequences of climate change). For these reasons, populations in the developing world are at particular risk of the adverse health effects of climate change.

An integrated multi-level approach to adaptation will be required to reduce the overall impacts of climate change-related adverse health outcomes (e.g. Table 1). In planning adaptation options, programmes must address a current (and future) climate-sensitive burden of disease (illness, injury or death) with explicit objectives and expected outcomes – there must be a measurable gain. They should also be practicable, cost effective, and able to be monitored and evaluated. It is important that these projects are not viewed and undertaken as 'stand alone' climate change efforts, but instead are mainstreamed into existing priorities and strategies across relevant sectors (e.g. to meet Millennium Development Goals).

As for the potential mitigation strategies, adaptation strategies may themselves have detrimental impacts on public health. For example, an irrigation and dam project to improve resilience against famine may contribute to an increased risk of malaria. Climate change adaptation programmes (including in non health sectors) therefore, should also be assessed in terms of potential short, medium and long-term detrimental health impacts.

Conclusion

Concerns about potential health risks are assuming greater importance in climate change discussions (Lancet, 2011). Here it is important to think broadly about possible changes in risk exposures and resulting health outcomes and to consider flow-on effects of single, interacting or co-occurring exposures. The field is difficult to research, with necessary reliance on ecological or correlational studies, and on climate prediction modelling. Health concerns were a major driver of international action to limit stratospheric ozone depletion (Dotto & Schiff, 1978) and there are now calls to factor in the health co-benefits of climate change mitigation to economic models, so that the cost savings from a healthier population can offset economic concerns about mitigation efforts (Lancet, 2011).

For most of the health considerations discussed herein, populations in developing countries are more vulnerable to the effects of climate change. However, even in wealthy countries, methods of coping, e.g. with warming temperatures by staying indoors and using air conditioners, may have unintended consequences such as vitamin D deficiency. Importantly, we are dealing with complex systems and a systems approach to understanding and managing the effects of climatic changes and interactions across the climate system is essential.

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