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Effects of solar ultraviolet radiation on coral reef organisms†

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Organisms living in shallow-water tropical coral reef environments are exposed to high UVR irradiances due to the low solar zenith angles (the angle of the sun from the vertical), the natural thinness of the ozone layer over tropical latitudes, and the high transparency of the water column. The hypothesis that solar ultraviolet radiation (UVR, 290–400 nm) is an important factor that affects the biology and ecology of coral reef organisms dates only to about 1980. It has been previously suggested that increased levels of biologically effective ultraviolet B radiation (UVB, 290–320 nm), which is the waveband primarily affected by ozone depletion, would have relatively small effects on corals and coral reefs and that these effects might be observed as changes in the minimum depths of occurrence of important reef taxa such as corals. This conclusion was based on predictions of increases in UVR as well as its attenuation with depth using the available data on UVR irradiances, ozone levels, and optical properties of the water overlying coral reefs. Here, we review the experimental evidence demonstrating the direct and indirect effects of UVR, both UVB and ultraviolet A (UVA, 320–400 nm) on corals and other reef associated biota, with emphasis on those studies conducted since 1996. Additionally, we re-examine the predictions made in 1996 for the increase in UVB on reefs with currently available data, assess whether those predictions were reasonable, and look at what changes might occur on coral reefs in the future as the multiple effects (*i.e.* increased temperature, hypercapnia, and ocean acidification) of global climate change continue.

Introduction

Ultraviolet radiation as an evolutionary and ecological forcing factor

Ultraviolet radiation (UVR) is probably the single most influential abiotic factor that has shaped the evolution and ecology of the biosphere. The anoxic environment that characterized the early and mid Archean atmosphere was highly reduced and the

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absence of stratospheric ozone (O_3) allowed the transmission of all wavelengths of solar radiation including ultraviolet C radiation (UVC, International Commission on Illumination [CIE] definition: 190–280 nm) and the short wavelength region of UVB to reach the surface of the early prebiotic Earth.[1] These highly energetic wavelengths are absorbed by DNA[2] and can cause damage and mutations. Exposure to these wavelengths was therefore a strong selective pressure[3,4] that severely limited the ecological expansion of organisms until the evolution of biological photoprotective mechanisms and the development of the O_3 layer. In the mid to early Archean, cyanobacteria capable of oxygenic photosynthesis evolved[1,5] and molecular or di-oxygen (O_2) appeared in significant amounts in the Earth's atmosphere approximately 2.5 Gyr ago. As O_2 slowly accumulated in the upper atmosphere it was photochemically modified to O_3, which filtered out the shortest wavelengths of harmful UVR and changed the course of biological evolution. More recently, the thinning of the stratospheric O_3 layer as a result of anthropogenic inputs of chlorinated fluorocarbons has caused an increase in harmful UVB reaching the Earth's surface.[6–8] Also, long-term monitoring has clearly demonstrated the influences of local conditions and global climate change on the amount, and variability, of UVR reaching the Earth's surface.[9–11] It has been predicted that the currently observed decreases in UVB will continue due to increased stratospheric O_3 well into the 21st century.[10]

In the tropics the concentration of stratospheric O_3 is normally less than in the remainder of the globe (Fig. 1)[12] and together with a consistently lower solar zenith angle results in tropical ecosystems having a long evolutionary history of exposure to higher irradiances of UVR compared to other latitudes.[13,14] In the tropics (0–30° latitude) seasonal irradiances of UVR do not vary significantly and are more affected by changes in cloud cover, aerosols, and atmospheric pollution.[11] Shick *et al.*[15] had previously reported that satellite and ground data for 1979 to 1994 showed small, non-significant, changes in stratospheric O_3 of –2% per decade from 20°S to 20°N latitude, and –2 to –4% per decade from 20 to 30°N. However, the DNA-weighted doses of UVR at 15°S and 15°N latitude increased by 2.8 ± 2.1% (mean ± SD) and 6.3 ± 2.4% (mean ± SD) respectively during the same time period.[7] Many subtropical coral reefs (Caribbean, Hawaii, Red Sea) lie within these latitudes, and the data at that time showed that even small, non-significant, decreases in stratospheric O_3 at these latitudes could result in large increases in incident UVB.[7] Non-significant increases in equatorial UVR have also been reported through the early 1990s[16] with variability in the incident UVR of approximately 3.0% associated with minima and maxima of the 11 year solar cycle.[9] In recent assessments of changes in stratospheric O_3, however, it was again reported that total column O_3 over the tropics (25°S to 25°N latitude) had remained essentially unchanged from previous assessments.[10,17] These analyses indicate that from 1980 to 2006 there has been no statistically significant decrease in total column O_3 in the tropics but another assessment of tropical O_3 data from 1979 to 2005 using Stratospheric Aerosol and Gas Experiment (SAGE I and II) profile measurements and Total Ozone Mapping Spectrometer (TOMS)/solar backscatter ultraviolet (SBUV) column O_3 data has shown a significant decrease in tropical stratospheric O_3.[18] Depending on the dataset used, changes over the entire time period (1979 to 2005) for equatorial regions (25°S to 25°N latitude) range from 0 to 4% for TOMS/SBUV and from 4 to >8% for combined SAGE/sonde data.[18] While there is still uncertainty in the actual losses of stratospheric O_3 over equatorial regions where most of the world's coral reefs occur, these ecosystems still experience the highest irradiances of UVR anywhere on Earth at sea level even when compared to the lowest O_3 levels measured, and corresponding increases in UVR, reported from the Antarctic during an "ozone hole" event.[9]

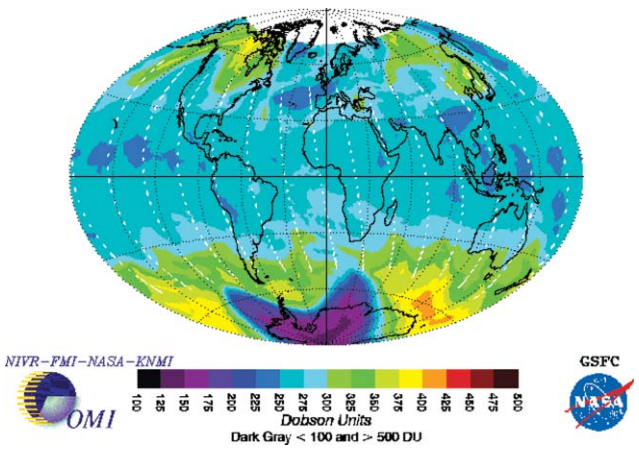

Fig. 1 Total ozone mapping spectrometer (TOMS) global projection for stratospheric ozone on October 26, 2008. Note the higher concentrations of ozone outside of the tropics except for the severe "ozone hole" over the Antarctic. One Dobson unit refers to a layer of ozone that would be 10 μm thick under standard temperature and pressure.

Other sources of variability of UVR that have recently been identified on a global scale include reports from land-based observations citing a decrease of 4 to 6% in total solar radiation incident on the surface of the Earth commonly known as "global dimming". This phenomenon was observed from the 1960s through the early 1990s predominantly in the United States and was believed to be primarily associated with urban areas.[19] Additional data, including satellite-based measurements from sites around the world, have shown that since 1990 a "global brightening" has actually occurred.[20,21] This dramatic change was attributed to decreases in atmospheric pollutants and aerosols that led to increased transmittance of solar radiation on a global basis and includes areas with coral reefs.[20,21] Another variable that significantly affects UVR reaching the Earth's surface on a regional scale is cloud cover,[22] with some areas reporting increased cloud cover[23] and others decreased cloud cover.[24] The results of trend analyses indicated statistically significant increases in UVR exposures of 10% per decade in the summer months in the tropics (specifically Darwin, Australia), which were associated with a simultaneous depletion of O_3 and a decrease in cloud cover and were also significantly influenced by the quasi-biennial oscillation (QBO). Whether this can be extrapolated to other areas such as the Great Barrier Reef (GBR) is debatable because Darwin is separated from the GBR by the Great Dividing Range, the barrier between the humid climate of the east coast and the arid climate of the west and interior of Australia, and results in lower levels of UVR than in the west.[24] Additionally, a recent study reported a <1% per decade increase in solar radiation for the southern Great Barrier Reef (18° to 26°S latitude).[25] As reported above, cloud cover is an important factor in tropical regions as increases

in cloud cover, which decreases both visible and UVR, have been suggested to decrease the probability of coral bleaching,[26] which is the breakdown in the symbiotic relationship between the coral host and its dinoflagellate partners (see below).

Finally, the definitions established in the 1930s by the CIE for different portions of the UVR spectrum are based entirely on the transmission characteristics of commonly available filters and are therefore largely arbitrary.[27,28] As pointed out by the technical committee and others, the CIE definitions are not exclusive for different effects and many biologists studying the photobiological effects of UVR use definitions that differ from those provided by the CIE.[27,28] The non-CIE definitions used here are based on the wavelengths incident on the biosphere, their biological effects, and consistency with the numerous data sets using more practical definitions since the 1980s.[15,27]

UVR in the water column over coral reefs

For many coral reefs the overlying water column allows UVR to penetrate to depths of 20 m or more because oligotrophic waters are dominated by the optical properties of the water itself and not by dissolved or particulate constituents in the water column.[15,29–35] While tropical waters are generally more transparent to UVR than temperate waters, the water column above coral reefs in coastal areas can be affected by terrigenous inputs, upwelling events, and variations in dissolved organic matter (DOM) that can modify its optical properties due to absorption and scattering, which will have profound effects on the attenuation of both photosynthetically active radiation (PAR, 400–700 nm) and UVR.[34–36] As an example, fringing reefs in the Bahamas exhibit a downwelling attenuation coefficient (K_d UVR) of 0.32 m^{-1} (Fig. 2) and Kaneohe Bay, Hawaii, which is actually a tropical estuary with high concentrations of DOM and particulate organic matter (POM), has a K_d UVR of 0.50 m^{-1} while Moku Manu, a nearby offshore reef has a K_d UVR of 0.08 m^{-1}.[37] UVB wavelengths are attenuated more rapidly than UVA wavelengths such that the maximum depth of UVB penetration can be much shallower than the maximum depth of UVA penetration and variations in the

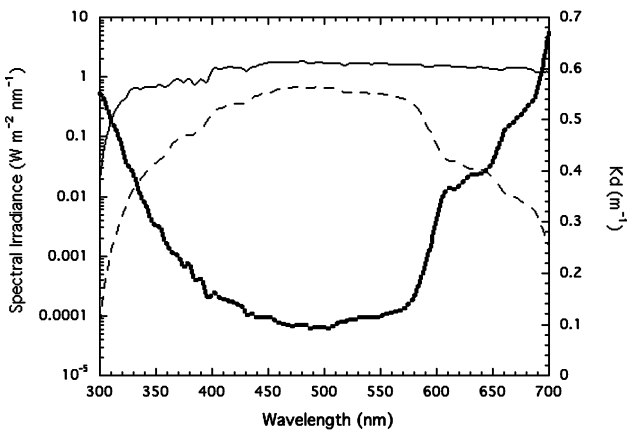

Fig. 2 Depth profile of spectral data (300–700 nm) recorded on the fringing reef at Horseshoe Reef (23° 46.5′ N lat., 76° 05.5′ W long.), July 2000, 1200–1230 h, using LiCor LI-1800UW scanning spectroradiometers (LiCor, USA) simultaneously placed at the surface (solid line) and a depth of 10 m (dotted line). Attenuation coefficients [K_d (m^{-1}), bold line] were calculated using the spectral data (300–700 nm).

optical properties of the water column may affect the attenuation of UVB more than UVA.[35,37] For example, in comparison with the attenuation coefficients reported above for UVR, the K_d UVB in Kaneohe Bay was 0.87 m^{-1} while in Moku Manu it was 0.18 m^{-1}.[37] In the Florida Keys, the attenuation coefficients for UVB, measured at 305 nm, are consistently higher than those for UVA and are controlled by the concentrations of colored DOM (CDOM) and dissolved organic carbon (DOC).[35]

The differences observed on these reefs are a result of the changes in the optical properties of the overlying water column, which can be more easily appreciated by the depth at which 10% of surface UVR ($Z_{10\%}$ UVR) occurs, which is 7.2, 4.6 and 28.7 m for the Bahamas, Kaneohe Bay and Moku Manu respectively.[37] Tedetti and Sempéré[34] estimated the $Z_{10\%}$ for UVA and UVB as well as the effective DNA damage dose for a wide variety of sites. They found a clear distinction between the open ocean characterized by a high $Z_{10\%}$ and coastal waters with a low $Z_{10\%}$ with no change in the UVB/UVA ratio when compared to Antarctic waters, which had higher UVB/UVA ratios under O_3 hole conditions.

Since 1996 there have been several additional descriptions of the underwater light environment including both PAR and UVR components of the spectrum for coral reef environments. Dunne and Brown[38] reported on downwelling UVR and PAR irradiances, and their K_d values, for several reefs in the Indian and Pacific oceans. While the spectral data only extend to a depth of 5 m and are reported as percentages of surface irradiance the attenuation coefficients for the study sites are provided and for the Maldives are greater than reported for offshore reefs in Hawaii[37] and the Bahamas,[39] with reefs in the Florida Keys and the Dry Tortugas having greater or smaller coefficients depending on the state of the local tide and location.[33,35,40] In addition, some reefs are located on tidal flats with recurrent exposures to ambient surface irradiances of PAR and UVR thus considerably increasing the damage potential, especially to critical targets like DNA, yet these coral communities survive.[38]

The most comprehensive studies describing the underwater light field of coral reefs as it relates to UVR, and its potential effects, come from work in the Florida Keys.[35,40] Lesser[40] examined in detail the vertical attenuation of UVR down to a depth of 30 m while Zepp *et al.*[35] examined many reef sites over a wide geographic area in the Florida Keys using both shipboard and continuous *in situ* measurements over a seven year period. Using an extensive dataset and analyses, Zepp *et al.*[35] showed that the waters of the Florida Keys had a spatially and temporally dynamic UVR environment primarily as a result of changes in the concentration of CDOM. When CDOM was highest the K_d for UVR was also high, which decreased the amount of biologically effective UVR incident on a shallow (3 m) coral reef.[35] CDOM concentrations also showed large spatial and temporal variability due to changes in the source of the principal absorbing compound being either of terrigenous or oceanic origin, as has been reported for several islands in French Polynesia[41] or with ebb and flood tides as has been described in the Bahamas,[42] Lower and Middle Keys and Dry Tortugas.[35] The effects of ocean acidification on the composition of CDOM and POM have not been investigated but could be another source of spatial and temporal variability of these constituents in the waters over coral reefs in the future.

A factor that has not been adequately studied is the water lensing effect by UVR on the subsurface light field due to flashes of

light, or sunflecks, created by the focusing of collimated solar radiation by surface waves.[37,43] Such enhanced UVB radiation events under the Antarctic ozone hole have been modeled and are an important component in the shallow photic zone[43] and could be a potentially important phenomenon in shallow water coral reef zones. Water lensing can be visualized on coral reefs as ripples of high irradiances and to date the UVB, UVA and PAR components have not been fully characterized.

Both reef morphology as well as coral morphology and orientation relative to the sun's position would also be expected to affect the microscale UVR environment due to differences in scattering and absorption from corals, pigmentation, and other benthic components such as surrounding sand. Because of such variability, accurate underwater measurements of UVR and/or modeling of spectral irradiance are crucial aspects of any photobiological study on the effects of UVR. Measurements of UVR on coral reefs should be combined with optical measurements of the water column to better describe changes in depth penetration of UVB and UVA. These measurements can be incorporated into long-term UVR monitoring networks that include satellite remote sensing techniques.[44,45] Continued global warming and ocean acidification will result in faster degradation of DOM and POM and potentially enhance the penetration of UVR, and in particular UVB into the water column and emphasizes the need for continuous monitoring on coral reef environments.[46] While biological and chemical dosimeters are useful and economical for short-term UVR determinations, they are not adequate for long-term monitoring as they work over a limited spectral range and doses.[47,48] In conclusion, despite the perceived constancy in tropical environments there is a clear lack of long-term data sets crucial to understanding the variability in ambient and underwater UVR over coral reefs. However, the lack of reasonably-priced instrumentation that is sensitive over the several orders of magnitude change in irradiance that occurs daily in the UVR spectrum is, unfortunately, not likely to be solved in the near future.

UVR photobiology of coral reef organisms

Effects of UVR on individuals

UVR is an important abiotic factor for coral reef organisms and, while many negative effects have been experimentally demonstrated, there are some positive aspects of exposure to UVR. For example, there are UVR receptors on the siphon of tridacnid mollusks[49] with unknown functions, and pomacentrid fish use UVR for vision to increase contrast for detection of zooplankton during daylight foraging by adults[50] and larval and juvenile fish.[51,52] UVR sensitivity in reef fish may also function in intraspecific communication where their predators are not capable of UVR vision.[53] Not all coral reef fish have visual UVR sensitivity as several species contain UVR absorbing compounds in their eyes, which would prevent these wavelengths from being useful in vision.[54] Of 211 species studied only 50% are capable of UVR perception.[55]

Several other studies stand out as unique in the coral reef literature because they document beneficial effects of UVR in coral reef organisms. Natural solar UVR was shown to increase the number of planula larvae released by *Pocillopora damicornis* when compared with conspecifics that were shielded from UVR.[56] UVR has also been shown to be required for the process of normal spicule formation in the gorgonian *Leptogorgia virgulata* as colonies that were maintained in the absence of UVR had significantly more "irregular" spicules when compared to colonies grown in the presence of UVR.[57] The spicules, at least in the colonial didemnid ascidian *Didemnum mole*, are potentially related to UVR photoprotection in shallow water colonies that contain higher densities of spicules than deeper water conspecifics,[58] but could also be related to an increase in hydrodynamic forces on shallow reefs. In another study UVR was shown to prevent bleaching in the coral *Oculina patagonica*.[59] In this Mediterranean coral, the causative agent of bleaching is believed to be the bacterium *Vibrio shiloi* whose virulence increases with increasing seawater temperature. Despite being exposed to temperatures that were higher by 2 °C than experienced by deep water conspecifics, colonies in shallow water (<1 m depth) showed negligible bleaching and an absence of *V. shiloi* in their tissues. In laboratory experiments it was shown that in infected corals exposed to solar radiation the intracellular bacteria were rapidly killed and bleaching was prevented, whereas in infected corals that were protected from UVR the bacterium multiplied and the corals bleached. It is debatable just how wide spread this phenomenon actually is because no other coral bleaching events have since been attributed to *V. shiloi*.

Other than these relatively few studies that show the positive effects of UVR in coral reef systems, all other studies suggest that the effects of UVR are negative. In this review we consider primarily corals and coral reef organisms where appropriate and we refer you to other reviews that have assessed the effects of UVR on other aquatic organisms.[46,60–65] The negative effects of UVR for coral reef organisms range from mortality, to decreased growth and calcification, reduced photosynthesis and changes in respiration, DNA damage and oxidative stress, as well as adverse effects on reproduction and larval development and settlement. The assessment of tolerance or sensitivity to UVR for a particular species depends on the parameters or assays that are used such that for heterotrophic organisms DNA damage, growth and survival can be monitored, whereas for autotrophic organisms photosynthetic or photochemical parameters are measured. In the determination of tolerance for a single species, light history and nutrient status can also play a very important role.[66]

Coral reefs tend to be restricted to shallow waters (<30 m) and therefore to environments composed of high irradiances of both PAR and UVR. The experimental manipulations of corals and other coral reef organisms in the field, generally involving transplantations or the exposure of cryptic organisms to direct solar radiation, have revealed that the effects of acute exposure to UVR are almost universally negative. In 1980, Jokiel[67] first confirmed that UVR was an important abiotic stressor influencing the structure of shallow tropical benthic marine communities by demonstrating that cryptic reef epifauna from a shaded environment at 5 m were killed by exposure to unshaded solar UVR at a depth of 20 cm. UVR has also been implicated in the mortality of corals in transplantation experiments in the field. Examples are the movement of *Plerogyra sinuosa* from depths of 25 m to 5 m where 90% of the specimens unshielded from solar UVR died within one month, whereas those that were shielded from solar UVR as well as a 5–10% reduction in PAR remained healthy for six months.[68] Not all acute transplantion experiments

result in mortality. Torres *et al.*[69] transplanted branches of the coral *Acropora cervicornis* from 20 m to 1 m and, while the corals survived the sudden change in light conditions, their linear extension rates and skeletal densities were significantly reduced due to decreased photosynthetic capacity of their symbionts and a possible reallocation of resources.

The effects of natural solar UVR was also shown to decrease skeletal growth in colonies of *P. damicornis* when compared to those grown under a 400 nm cutoff filter,[56] which may be related to decreased calcification rates as determined by ^{45}Ca uptake for the same species in the presence of natural levels of UVA.[70] Inhibition of growth was not seen during a similar experiment by Glynn *et al.*[71] with this species, nor with *Acropora valida*, which were grown under 350 nm cutoff filters. In another study, exposure of *Porites astreoides* to natural UVR resulted in decreased skeletal growth in the more UVR sensitive brown morph compared to the green morph.[72]

The results of transplantation experiments suggest that the coral's prior history of exposure to solar UVR and PAR influences its tolerance to UVR.[73–76] Under artificial conditions of UVR exposure Siebeck[73,74] documented depth-related differences in tolerance to UVR (275–400 nm) for several coral genera as indicated by LD50 values where shallow specimens from 1.5 m were more tolerant to UVR than conspecifics whose depth of origin was from 18 to 20 m. We note, however, that short and unnatural wavelengths of UVR are included in these experiments that may not accurately reflect responses under natural solar radiation.[73,74] Acclimatization to solar UVR can also increase the synthesis of photoprotective UVR absorbing compounds known as mycosporine-like amino acids (MAAs) in colonies of *Montipora verrucosa*[75,76] and *A. cervicornis*[69] when exposed to UVR.

Autotrophs as well as heterotrophs that are in symbiosis with autotrophs, such as corals, must be exposed to adequate PAR to maintain the photosynthetic process, which in turn exposes them to UVR. Transplantation experiments measuring the effects of UVR on photosynthesis and respiration on corals and other coral reef organisms show similar results whether using natural solar[77] or artificial UVR.[78] Colonies of *Acropora microphthalma* that were collected at 20 and 30 m depths and maintained at a depth of 1 m showed ≥30% inhibition of photosynthesis when exposed to UVR compared to the paired colonies that were shielded from UVR.[77] In *Fungia* colonies collected at 30 m, photosynthesis was inhibited by artificial UVB compared to those colonies collected at 1 m.[78] In both cases, the isolated symbiotic algae from all depths were inhibited by UVR. Photosynthesis was also severely suppressed by artificial UVB in *Symbiodinium bermudense* isolated from the tropical sea anemone *Aiptasia pallida*,[79] *Symbiodinium* sp. from tridacnid clams,[80] and in the prochlorophyte *Prochloron* sp. isolated from the tropical ascidian, *Lissoclinum patella*,[81] but no effect was found when the symbionts were irradiated *in situ*[80–82] suggesting that the hosts play a crucial role in the success of these symbiotic associations in shallow tropical marine environments. Using a biological weighting function (BWF) for dinoflagellate photoinhibition, a weighted UVR (290–400 nm) irradiance of 12.5×10^{-3} W m^{-2} was used to simulate *in hospite* irradiances for *S. bermudense* growing in culture and resulted in the depression of maximum photosynthetic capacity, cell-specific content of chlorophyll *a*, the quantum yield of photosystem II (PSII) fluorescence, and ribulose 1,5-bisphosphate decarboxylase/oxygenase (Rubisco) activity.[83] The addition of exogenous antioxidants improved photosynthetic performance and chlorophyll-specific fluorescence, which suggests that the effects of UVR were both direct and indirect, in the latter case, mediated by reactive oxygen species (ROS). The results are consistent with the known effects of both UVR and ROS on the D1 protein in photosystem II[84,85] and on Rubisco.[86,87]

There is no consistent effect of UVR on respiration among corals, however, in freshly isolated zooxanthellae from UVR treated hosts the respiration rates are higher than in those hosts that have been shielded from UVR as has been shown in symbionts isolated from the anemones *A. pallida*[79] and *Phyllodiscus semoni.*[88] Similar to the results for the inhibition of photosynthesis, when the rates of respiration of freshly isolated symbiotic algae are compared to *in situ* respiration rates, significant decreases are seen in the former whether using natural solar radiation[75] or artificial UVB exposures.[78] These results suggest that the larger biomass, and hence greater rates of respiration of the host may mask any effects of UVR on respiration of zooxanthellae *in hospite.*[15]

The induction of photooxidative stress by UVR

In addition to its direct effects, UVR indirectly damages corals and other reef organisms *via* photochemical reactions that produce ROS. These reduced oxygen intermediates are produced by reactions that transform the electronic excitation resulting from the absorption of UVR into chemical energy by reducing molecular oxygen (O_2), and forming ROS such as hydrogen peroxide (H_2O_2), superoxide radicals ($O_2^{-\bullet}$), hydroxyl radicals ($^{\bullet}OH$) and singlet oxygen ($^1O_2$). Most of the production of ROS does not involve the direct activation of O_2 by UVR. Rather, various intermediate molecules in cells (*e.g.* aromatic amino acids) absorb UVR, and in particular UVA,[89] and enter into an excited state wherein the excitation energy can be used to form the various species of ROS, which in turn can lead to the production of extremely reactive $^{\bullet}OH$ in an iron-catalyzed Fenton reaction. UVA-generated ROS have multiple toxic effects on organisms, damaging DNA (see below), enzymes, membrane proteins and lipids (especially those containing polyunsaturated fatty acids) and photosystem components, resulting in what is more commonly known as photooxidative stress.[90] The intracellular production of ROS increases proportionally with O_2 concentration so that photoautotrophic symbioses such as corals producing an excess of O_2 in sunlight[91,92] are particularly vulnerable to the separate and interacting effects of UVR and ROS.[90,93]

Antioxidant defenses include the enzymes superoxide dismutase (SOD), catalase, and ascorbate or glutathione peroxidases, which detoxify $O_2^{-\bullet}$ and H_2O_2.[90,93] Various water-soluble, non-enzymatic, antioxidant molecules can also scavenge or quench ROS.[90,93] In this regard, early studies showed that the higher activity of host SOD in solar UVR exposed specimens of the temperate sea anemone *Anthopleura elegantissima* than in UVR shielded clone-mates was evidence of UVR induced oxidative stress.[94] The elevation of SOD activity in host and zooxanthellae, and of catalase and ascorbate peroxidase in the zooxanthellae from the UVR exposed reef anemone *P. semoni* was interpreted similarly.[88] Lesser *et al.*[95] found a significant UVR-related elevation of SOD and catalase in the zooxanthellae, but not for the activities in the host tissue of the reef zoanthid *Palythoa caribaeorum.* Lesser and Shick[79]

demonstrated that exposure to UVR at moderate irradiance (PAR = 375 μmol quanta m^{-2} s^{-1}) under a solar simulator significantly elevated SOD and catalase activity in cultured zooxanthellae (*S. bermudense*) from the tropical anemone *A. pallida*, but not in zooxanthellae exposed *in hospite*. This indicates a protective effect provided by the host for its symbionts under these experimental conditions. However, in full sunlight (1700 μmol quanta m^{-2} s^{-1}), zooxanthellae exposed to UVR *in hospite* had higher activities of SOD, catalase and ascorbate peroxidase compared to *A. pallida* that were shielded from UVR[96] such that host protection may not be as effective at the very high irradiances observed on shallow coral reefs where these anemones are generally found.

Lesser[83] established semi-continuous cultures of zooxanthellae isolated from *A. pallida* and simulated the *in hospite* nutrient conditions and PAR light regime while exposing the cultures to high temperature stress with and without exposure to UVR. During this experiment both the independent and interactive effects of thermal stress and UVR on photosynthesis were assessed in both the light and dark reactions as well as by measuring the flux of ROS and the activity of antioxidant enzymes. Photosynthesis, PSII function, growth rates, and Rubisco activity were shown to be significantly affected by exposure to an increase in temperature from 25 °C to 31 °C. Additionally, photosynthesis, PSII function, growth rates, and Rubisco activities further declined, significantly, when the same cells were exposed to elevated temperature and UVR. These observations were always accompanied by increases in the cellular concentration of ROS as well as increases in enzymatic antioxidant defenses. Exposing cultures to ascorbate, a non-enzymatic quencher of hydroxyl radicals, and catalase, which decomposes H_2O_2 to water and oxygen at the end of the experiment for only 1 h improved photosynthetic performance (*i.e.* P_{max}) by 24% in cultures exposed to 31 °C and by 37% in cultures exposed to elevated temperature and UVR.[83] For corals clear, direct, evidence shows that despite the protection afforded by host tissues and its constituents (*e.g.* MAAs) photosynthesis measured either directly,[40] or as photochemical efficiency using active fluorescence techniques,[97] shows a significant decline when exposed to UVR.

UVR induced DNA damage

DNA is a key target for solar UVB-induced damage either by direct absorption of the high-energy wavelengths or by indirect damage due to the production of ROS. The most common form of direct UVB damage is the generation of cyclobutane pyrimidine dimers (CPDs) by the formation of covalent bonds between adjacent pyrimidine (principally thymine) bases in DNA. DNA damage can also result from the formation of 6–4 photoproducts, photoisomerization, and Dewar valence isomers.[65] The presence of these lesions results in the deformation of the DNA helix and increases the probability of incorrect DNA replication or transcription that can lead to mutations or abnormal gene expression.[98] DNA photodamage can also be induced by exposure to $^1O_2$ that oxidizes the DNA and can result in strand breaks and DNA–protein crosslinks.[2,99] DNA damage arrests the cell cycle at the G1/S phase until the UVR-induced dimers are removed by DNA repair mechanisms such as photoreactivation or dark repair pathways,[100] and if the damage cannot be repaired it can then lead to apoptosis (programmed cell death) or cell necrosis.[101,102]

To date, the relatively few studies that have investigated DNA damage in coral reef organisms show that under natural conditions it is restricted to shallow waters. In the late-summer, however, doldrums conditions can result in lower attenuation coefficients and higher UVB doses at deeper depths,[32,103,104] with the possibility of greater damage if repair mechanisms are not increased proportionately. Under normal conditions there is a decline in those wavelengths that could damage DNA with increasing depth. For example, in the waters around coral reefs in the Bahamas the attenuation of pyrimidine dimer formation was shown to be exponential with depth. At 3 m UVB induced pyrimidine dimer formation was 17% of the surface values.[105]

The effects of UVR on DNA damage has been studied in bacterioplankton from the Gulf of Aqaba,[106,107] the Gulf of Mexico,[108] the Caribbean Sea[109] and a reef lagoon in New Caledonia.[110] Bacterioplankton play a critical role in nutrient cycling and energy flow to higher trophic levels, serving as both mineralizers and secondary producers that are consumed by higher organisms.[106,108] Bacterioplankton tend to be more susceptible to DNA damage than phytoplankton,[106,108–110] which can have significant consequences on the biodiversity and population dynamics of these important components of tropical ecosystems. DNA damage has been shown to increase over the diurnal cycle in bacterio- and phytoplankton when incubated in full surface solar radiation, and when UVB was excluded no DNA damage was observed, indicating that thymine dimers were only formed by UVB.[109] Additionally, DNA damage tended to be greater at the surface and in calm seas while no damage was detected below 10 m.[106,108,111] The extent of surface mixing determines the depth of DNA damage and no net accumulation of damage was observed in moderate seas with high rates of mixing, even at the surface.[106,108] Protein and DNA synthesis in bacterioplankton was inhibited by UVB, UVA and PAR while carbohydrate synthesis in phytoplankton was inhibited by both UVB and UVA.[106,109] Lyons *et al.*[112] estimated DNA damage in the microbial community associated with the coral surface microlayer (CSM), which extends a few millimetres above the surface of the corals *Montastraea faveolata* and *Colpophyllia natans*, and found that the extent of DNA damage was significant but consistently lower than in water column samples, suggesting that photoprotective mechanisms within the CSM may provide protection to the coral from UVR.

For the scleractinian coral *Porites porites* var. *porites* the levels of damage to DNA after exposure to solar simulated UVR for both host and algal tissue were dose-dependent when the UVR irradiances were weighted for DNA damage.[113] Using a UVB lamp as the light source and single cell gel electrophoresis (known as the comet assay), more DNA strand breakage was induced at higher doses in suspensions of host cells of the coral *Stylophora pistillata* containing symbiotic dinflagellates than in algal-free coral cells and coral-free algal cells.[114] The extremely high UVB exposures and the absence of UVA and PAR during the exposure to the UVB lamp may have resulted in greater levels of damage because photoreactive repair mechanisms that are normally induced by the longer wavelengths could not be activated.[115] Significantly more CPDs were formed in the host tissue fraction when the coral *M. faveolata* was exposed to full solar irradiance than when exposed to a light environment simulating 8 to 10 m depths.[101] In a study by Torregiani and Lesser[76] colonies of the coral *M. verrucosa* were collected from three different depths (1, 5 and 10 m) and exposed to

three different UVR treatments for three days under constant PAR equivalent to a depth of 0.15 m depth in Kaneohe Bay, Hawaii. Direct UVR damage to DNA was measured as CPDs and (6–4) pyrimidine–pyrimidone photoproducts for the holobiont. CPD accumulation in *M. verrucosa* was greatest in corals from 1 m, whereas corals originally from 10 m showed the lowest amount of DNA damage in response to exposure to UVR that was correlated with the greater concentrations of UVR absorbing compounds in 10 m samples. In another study of CPD formation in the Caribbean coral *P. astreoides* it was shown that damage to host tissue DNA is higher than symbiont DNA during exposure to natural UVR.[115]

DNA damage, when it has been found, appears to be restricted to shallow water conditions.[76,101,113,115] Organisms that experience UVR-induced DNA damage require a signaling pathway to detect the damage and induce the synthesis of DNA repair enzymes such as photolyase when the damage exceeds the repair capabilities of the constitutive levels of the enzyme.[65] If the damage is irreparable, apoptosis may be induced. Photoreactivation is the light-induced repair of DNA damage using the photolyase enzyme in the presence of UVA or blue light that provides the energy required to break the bonds formed between adjacent nucleotides that have formed dimers as a result of exposure to UVR.[65] When exposed to UVR, scleractinian corals can repair and recover from this damage but only in the presence of short wavelength PAR.[73,74] Repair and recovery of UVR exposed corals were greater when the corals were subsequently exposed to PAR compared to conspecifics maintained in the dark where the repair of DNA damage by photoreactivation could not occur. Therefore, the dose of UVR required to induce mortality in 50% of the colonies (LD_{50}) was lower than in colonies that were subsequently exposed to PAR.[73,74] These results are also consistent with the known action spectrum for enzymatic photorepair by photolyase.[116,117] To date there are no other studies published on photoreactivation, or on photolyase activities in corals and as suggested by Gleason[103] research in this area should be a priority but that has yet to be realized. Interspecific variation in photolyase-mediated repair activity has been measured in opisthobranch mollusks and found to vary between species and does not exceed 20% repair of the damaged DNA.[118] This suggests that the remaining 80% of damaged DNA would need to be repaired by other means such as nucleotide excision repair also known as dark repair.

With the genomic revolution being fully embraced by the coral reef community the availability of genomic databases and coral gene arrays (*i.e.* microarrays) are now available for various coral species such as *A. cervicornis*, *Acropora palmata* and *M. faveolata.*[119–121] These databases can be used to develop specific microarrays to examine gene networks up-regulated after exposure to stress and could be used to undertake research into changes in gene expression profiles in corals exposed to different environmental stressors such as UVR[119] and to quantify the expression of genes involved in DNA damage and repair.

Effects of UVR on reproduction and larval development

In marine ecosystems any changes in the environment, such as increased exposure to UVR or thermal stress, can exceed the physiological thresholds of organisms and cause significant mortality.[122] The most sensitive life history stages are generally believed to be eggs and embryos many of which develop in the shallow waters of the world's oceans including tropical environments.[123–125] Coral eggs from spawning species contain high concentrations of lipids (60–70% by weight) causing them to float to the surface of the water column once they are released from the parent colony during nocturnal spawning events. In some species, the eggs are released as packets together with sperm that is located in the center of the packet of eggs. Self-fertilization is inhibited and as the egg–sperm bundles reach the surface, they break up and fertilization with other genotypes can occur. The embryos develop into planula larvae, which stay afloat at the surface of the water column for several days, and are consequently exposed to the potentially harmful effects of solar UVR.

Fecundity of the coral *A. cervicornis* was estimated over a six month period in reciprocally transplanted branches between 20 m and 1 m to determine the effects of acute exposure to UVR on this ecologically important and threatened species.[126] The results showed that sexual reproduction was completely halted in branches that were transplanted to 1 m, whereas branches that were moved to deeper waters showed only slight delays in gamete release, possibly due to the change in their daily light cycle.[126] It is possible for *A. cervicornis* branches to be naturally transplanted from such depths due to storm surges or hurricanes with wave action strong enough to break the branches or colonies and move them to shallower waters. This also has implications for restoration efforts on this coral because the results show that transplants need to be placed initially at deeper depths to not only increase survival but also to increase the possibility that sexual reproduction will occur.

Exposure to UVB results in low survival, long developmental durations or damage in eggs and larvae of corals[127,128] and other invertebrates such as polychaetes, crustaceans, oysters, echinoderms and fish.[46,129] The survivorship of eggs and planulae of three species of mass spawning reef corals, *A. palmata*, *Montastraea annularis*, and *M. franksi*, originating from deep sites was significantly lower than those from shallow sites when experimentally exposed for up to 4 days to ambient surface levels of UVR with UVB being responsible for most of the observed differences in larval survivorship.[128] Planulae of the reef coral *Agaricia agaricites* collected from colonies at 3 m were more resistant to UVR than were those from 24 m depth, which corresponded with tissue concentrations of MAAs with larvae from 3 m having higher concentrations of UVR absorbing compounds than those from 24 m depth.[129] The differential mortality in these planulae was related to exposure to UVB rather than to UVA or PAR. These results show that the sensitivity to high irradiances of UVB may affect the survival of coral larvae in shallow reef-waters and thus decrease rates of gene flow between coral populations.[130] This has potentially important implications for suggestions that deeper water populations could be a genetic pool for populating shallower areas affected by coral bleaching.

An important behavioral response to UVR exposure is phototaxis. While this would not be an important issue for developing eggs and embryos in the water column, the crawl away planulae of many species of brooding corals may also be affected, particularly in shallow water environments. It should be noted that these crawl away planulae can also be swept into the water column where they could be exposed to higher irradiances of UVR. It has been shown, however, that the crawl away planulae of the Caribbean coral, *P. astreoides* have the capacity to detect UVR. Experimentally

given the choice of three different light treatments to settle under: PAR or PAR + UVA or PAR + UVA + UVB, significantly greater numbers of larvae settled where UVR was reduced or absent.[131] The ability to detect UVR and avoid habitats with high UVR may be an important component that determines the successful recruitment of this species in Caribbean coral reef environments.[131] Most larvae respond negatively to UVR by delaying settlement or through direct physiological effects that inhibit the settlement process in some way, as was shown for *P. damicornis* where larvae exposed to UVR had no significant effect on mortality but did negatively affect settlement.[132] In contrast, the combined effect of UVR and temperature on the survival of planulae from soft corals from the Red Sea shows that the survival of aposymbiotic planulae increased with higher temperatures under UVR exposure.[133] As pointed out by Shick *et al.*[15] and Gleason,[103] the effects of UVR on coral reproduction and its role in larval dispersal and settlement has not been sufficiently studied and it is critical to obtain more data in order to understand the ecological and evolutionary significance of UVR in coral reef ecosystems for these life history characteristics. In the face of global climate change understanding these aspects of coral biology is even more critical.

UVR and coral bleaching

Bleaching in corals is the active expulsion of symbiotic algae from the tissues of the host caused by a range of environmental stressors.[90,122,134] In some cases bleaching may be due to the loss of pigmentation by the symbiotic algae. Elevated seawater temperature, often associated with El Niño warming events, is regarded as the most important primary factor in geographically widespread bleaching.[122,134] Evidence for bleaching caused by UVR in the field is anecdotal[135] but field experiments do show UVR as an independent abiotic factor that causes bleaching.[32] Gleason and Wellington[32] presented some of the best field data demonstrating that UVR could cause coral bleaching. This paper, however, has been criticized because of experimental problems related to differences in PAR irradiance between treatment groups that may have contributed to the observed effects.[136] The differences in PAR for the treatment with UVR (488 μmol quanta m^{-2} s^{-1}) and PAR in the treatment without UVR (442 μmol quanta m^{-2} s^{-1}) are physiologically insignificant and should not undermine the conclusion that UVR alone can induce coral bleaching under the right circumstances. One condition where UVR may cause coral bleaching independently is due to the deeper transmission of UVR into the water column during prolonged calm water conditions known as the doldrums.[32,137] Any of the direct effects of UVR will also very likely be species specific. While exposure to high irradiances of UVR decreased growth in colonies of *P. damicornis* collected from a depth of 1 m when these same colonies were exposed to unattenuated sunlight in shallow aquaria they could not be induced to bleach.[56]

UVR is also an important interactive factor in coral bleaching caused by thermal stress.[90,95,138] Corals are known to live at temperatures close to their lethal limits during the summer months.[134,138,139] Bleaching can be induced by short-term (several days) acute exposure to water temperatures several degrees above average summer temperatures or by longer-term exposures (several weeks) at exposures of 1 to 2 °C above average.[104,138] Lesser *et al.*[95] experimentally demonstrated significant independent effects of solar UVR and elevated temperature in reducing the number of zooxanthellae per polyp in the zoanthid *P. caribaeorum.* Glynn *et al.*[71] found that UVR (≈30% of direct solar irradiance) resulted in greater loss of symbiotic algae in *A. valida* and *P. damicornis* colonies collected from 2 m to 4 m than was observed in shielded conspecifics, but only at temperatures 1.3 to 1.8 °C above ambient in aquaria. These conditions also resulted in higher mortalities in *A. valida* colonies than in conspecifics shielded from UVR, although there was no independent effect of UVR on survival of *P. damicornis* colonies exposed to these experimental conditions.[71]

Not all coral species respond in the same way to the effects of ambient UVR and temperature. Elevated seawater temperatures caused bleaching in *Porites lobata* from the Pacific coast of Panama[140] or *Millepora alcicornis* from the Caribbean[104] but no significant effect was observed from exposure to ambient UVR. In the case of *M. alcicornis*, a hydrozoan coral, exposure to ambient levels of UVR does not have long term effects on photochemistry as the quantum yield of photosystem II (PSII) fluorescence for branches from naturally growing high light and low light samples decreased during the day and would fully recover by the same evening. Additionally, long term exposure (10 days) to elevated seawater temperatures up to 3 °C above the 7-year August average resulted in irreversible bleaching and death of colonies naturally growing in both sunny and shaded habitats.[104] In another coral, *M. faveolata*, an important Caribbean reef-building scleractinian coral, thermal stress in combination with high solar radiation (both PAR and UVR) resulted in greater host DNA damage and subsequent upregulation of genes involved in apoptosis or programmed cell death.[101] In another example under controlled laboratory experiments where the symbiotic gorgonian *Eunicea tourneforti* was exposed to different temperatures and UVB, the colonies bleached at temperatures ≥29.0 °C with UVB irradiances of 2.80 W m^{-2} that enhanced the bleaching response compared to temperature stress alone.[141] Lastly, D'Croz and Maté[142] showed that for experimentally bleached colonies of *P. damicornis* continued exposure to UVR prevented recovery even as temperatures decreased (30.3 °C). In general our knowledge on the response of the host to UVR stress with or without interacting factors (*e.g.* thermal stress) is wanting and the suggestion by Gleason[103] that studies to investigate how host damage contributes to coral bleaching has resulted in some recent investigations[143,144] but more research is needed for a better understanding of the role of the host in coral bleaching.

Biological weighting functions (BWFs) for UVR effects on coral reef organisms

The sensitivity of any biological process to UVR can be described mathematically using BWFs, which express the relative effect of radiation of the same photon flux density at different wavelengths[145] and therefore translate physical radiation measurements into biological effects. Originally described by Rundel[146] BWFs, unlike action spectra, include the interactive effects of other components of the spectrum such as repair mechanisms driven by simultaneous exposure to UVA and PAR.[145] BWFs are therefore important for modeling and making predictions about the effects of UVR in general on coral reefs and specifically the effects of enhanced UVB exposure as a result of stratospheric O_3 depletion or other factors.[15,35,40]

Halldal[147] presented the earliest available action spectrum for photosynthesis in symbiotic algae isolated from *Favia pallida*, which showed a significant amount of UVA-stimulated photosynthesis. However, Lesser[83] showed that BWFs for cultured symbiotic algae exhibit an inhibition of photosynthesis in both the UVA and UVB portions of the spectrum. This inhibition was further enhanced by exposure to elevated seawater temperatures consistent with those experienced by anthozoans symbiotic with dinoflagellates in the field. The BWF for the inhibition of photosynthesis by UVR in the coral *P. damicornis* from Hawaii[148] exhibited a significant increase in the effects of UVB (up to 310 nm), but a subsequent decrease in the remainder of the UVR spectrum when compared with the BWF for cultured zooxanthellae.[83] The shape and the magnitude of the BWFs for both *M. faveolata* and *P. damicornis* reflect both the accumulation of high concentrations of UVR-absorbing compounds in *P. damicornis* and the absorption properties of these compounds, which absorb principally in the UVA portion of the spectrum.[40,148,149] Because the irradiances of UVA are so much greater than UVB the weighted dose of biologically effective UVR (UVR_{BE}) is always greater for UVA for a specific effect such as photosynthesis despite the lower wavelength dependent effects compared to UVB.

When combined with spectral and *in situ* photosynthesis or DNA damage data, BWFs can be used to determine the UVR_{BE} as it relates to photoinhibition or DNA damage, respectively. By looking at both the shape and magnitude of the BWF across the UVR spectrum or by weighting the solar spectrum in the UVR with an appropriate BWF one can compare that weighted spectrum to the known MAA profile. For example, taking the BWF for photoinhibition of photosynthesis in corals[40,148] and using those BWFs to weight the solar spectrum at different depths for two species of coral (*M. faveolata* and *P. damicornis*) shows that MAAs provide most of their protection in the UVA portion of the spectrum (Fig. 3a, b). As depth increases the importance of MAAs decreases as the damaging wavelengths are filtered out by the water column, which is reflected in the UVR_{BE} at different depths (Fig. 3a, b).

Zepp *et al.*[35] used their extensive data set and the BWFs for the inhibition of photosynthesis in corals[40] and DNA damage[150] to model UVR damage in corals on Looe Key Reef in the Florida Keys at a depth of 3 m. They found that while the UVR_{BE} dose for DNA damage was much greater than for the inhibition of photosynthesis it decreased much more rapidly with depth as those UVB wavelengths responsible for DNA damage attenuate rapidly, whereas the spectral dependence for the inhibition of photosynthesis extends well into the UVA portion of the spectrum. Biological damage potential, when weighted by the DNA-damage action spectrum (E_{DNA}), also showed a more rapid attenuation with depth than downwelling UVB irradiance ($E_{d,UVB}$) with a 1% E_{DNA} depth of 9 m for an ocean atoll, 4.7 m for a coastal island, and 2.6 m for an inshore reef.[38] Increases in CDOM also affect the modeled DNA damage to a greater degree than the inhibition of photosynthesis as CDOM has significantly greater effects on UVB wavelengths, but both processes are predicted to be more strongly affected by changes in spectral attenuation by the water column than by changes in stratospheric O_3 concentration.[35] Additionally, we do not know how repair processes may change over depth, especially those repair pathways that are light dependent (*i.e.* photoreactivation).

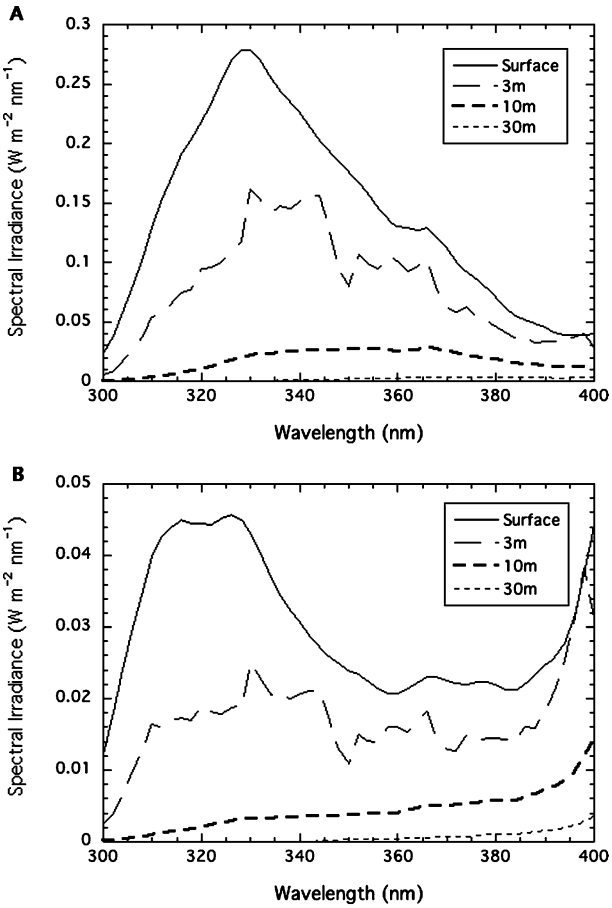

Fig. 3 Relative effective spectral irradiance (W m^{-2} nm^{-1}) at different depths for (A) *M. faveolata* and (B) *P. damicornis* obtained by weighting the irradiance at 0.05 m, 3, 10 m and 30 m (data from ref. 40) by the biological weights obtained for the inhibition of photosynthesis at 3 m for *M. faveolata* (data from ref. 40) and at 1 m for *P. damicornis* (data from ref. 146). Note the different scale used for *P. damicornis.*

One way to compare the sensitivities of different BWFs or to compare the same function among diverse taxa is to calculate a radiation amplification factor (RAF) that determines the proportional change in UVB_{BE} for a given decrease in O_3 concentration.[6] The RAF is the percentage increase in damage due to an increase in UVB caused by a 1% depletion in the O_3 layer.[64] For example, the RAF from 280 to 420 nm for the inhibition of photosynthesis in *P. damicornis* at 1 m depth in Hawaii is 0.20 ± 0.02 (mean ± SD), with little variation associated with the solar zenith angle.[148] As a comparison the RAF for DNA damage is ~2.0 which translates into a ten fold increase in the potential for biologically effective UVR for DNA damage compared to photoinhibition of photosynthesis for any incremental decrease in O_3 concentration.[6]

A more comprehensive study by Lesser[40] describes changes in the PAR and UVR components of the solar spectrum with increasing depth as it relates to primary productivity and the biosynthesis of MAAs in the reef-building coral *M. faveolata.* These data were used to examine depth-specific RAFs when combined with K_d values and Lesser[40] predicted that photosynthesis by *M. faveolata* at 5 m or deeper would be little affected, if at all, by any further increases in solar UVR. Shick *et al.*[15] applied the RAF for UVR inhibition of photosynthesis in *P. damicornis*[148] to a

10.8% decrease in stratospheric O_3, which resulted in a calculated increase of only 2.3% in UVB_{BE} at 307.5 nm, but a 25.7% increase in DNA-weighted dose using the Setlow and Setlow[150] DNA action spectrum. At the same rate of O_3 loss during the next decade, the cumulative loss from 1969 to 2006 would be 14.8%, which would expose corals at 1 m depth to a 3.2% greater photosynthesis-weighted dose, and a 37.8% higher DNA-weighted dose, than in 1969. However, even the worst-case scenario for equatorial regions has only shown a maximum decrease in stratospheric O_3 of 8%.[18] If we continue to use the exceptionally high rates of O_3 loss from Shick *et al.*[15] to calculate projected O_3 losses through the year 2008, corals at 2 m would then be exposed to greater irradiances of UVR than those calculated in 1969 for corals at 1 m but corals at 3 m would never be exposed to irradiances equivalent to the irradiances calculated for 1 m in 1969. Based on these liberal projections we anticipate only small additional independent effects of UVR on the biology and ecology of corals in the future despite the fact that we consistently see UVR effects in many experimental studies. This should not, however, be a signal to ignore the role of UVR and its interactive effects with other stressors such as elevated temperatures and ocean acidification. Tolerance to UVR exposure depends on how much of the damaging wavelengths reach critical internal cellular targets and in corals this may be enhanced due to the architecture of the internal skeleton.[151] The calcium carbonate skeleton effects the residence time of damaging photons through multiple scattering events, which is counterbalanced by the suite of photoprotective and photorepair mechanisms that the organism has available and the efficiency with which these mechanisms prevent or reverse UVR damage.

Photoprotective mechanisms that reduce the effects of UVR

The photoprotective mechanisms present in any organism must be specific enough to eliminate or reduce the exposure to UVR by using the basic physical principles of reflectance and absorption of UVR by constituents of the tissues and pigments of the coral host and their symbionts such as MAAs, before they can damage the sensitive cellular components and thus protect the organisms against the damaging UVR. In autotrophs or organisms symbiotic with autotrophs such as most corals, the photoprotective mechanisms must at the same time allow for the transmission of PAR to facilitate photosynthesis. Photoprotection in coral reef organisms can take many forms. Photoreactivation and DNA repair mechanisms have been covered in the section on UVR-induced DNA damage, whereas antioxidants and quenchers have already been discussed in the photooxidative stress section. Other protective mechanisms include physical barriers such as morphological or structural features that prevent the passage of UVR, and the production of compounds that absorb UVR.

The best known and commonly observed photoprotective response in a wide variety of marine organisms is the production of or presence of UVR absorbing compounds known as mycosporine-like amino acids (MAAs). Other UVR absorbing compounds that have been identified in marine organisms include scytonemin, carotenoids, 3-hydroxykynurenine, sporopollenin, melanin and fluorescent pigments. There are a number of reviews[152–160] on photoprotective mechanisms that can be referred to for further information in other marine organisms as well as freshwater taxa.

Physical barriers

Structural features such as the shells of gastropods and crustaceans and spines of echinoderms serve to protect invertebrates from predators in addition to providing protection from UVR. Corals produce mucous that may physically screen the host and symbiont from UVR damage particularly due to the presence of MAAs.[161–163] Mucous is produced by corals in response stress such as smothering due to sedimentation and desiccation that can occur at low tides when whole coral colonies can be exposed to air. Mucous may also decrease the damaging effects of UVR although it has been reported that the UVR screening effectiveness of MAAs in the mucous is poor.[163]

Symbiotic algae have the ability to produce multiple layered cell walls when exposed to artificial UVR in culture for four weeks, which disappear if UVR is removed.[164] The additional cell walls were hypothesized to protect the cellular contents by absorbing the damaging UVR as this species of dinoflagellate, *Symbiodinium californium*, does not synthesize MAAs in culture or *in hospite*.[165] Within the host, the algae do not produce these cell walls suggesting that MAAs found in the host tissues provide adequate protection under natural conditions.[165]

In the marine dinoflagellate *Scrippsiella sweeneyae*, cells exposed to UVR increase in cell volume possibly to lengthen the path that damaging photons have to travel and thus decrease the possibility that they would cause damage to sensitive cellular components such as DNA[166] and would also increase the possibility of being absorbed by the MAAs present in these cells. In another study Lesser and Shick[79] showed that an increase in cell size was associated with a decrease in the doubling times of zooxanthellae, in culture, exposed to UVR, which could be the result of DNA damage and cell cycle arrest.

Mycosporine-like amino acids (MAAs)

MAAs are low molecular weight water-soluble compounds, derivatives of mycosporines—a group of compounds first identified in the mycelia of fungi and hypothesized to act as photoprotectants during sporogenesis.[167,168] MAAs were originally termed "S-320" due to their maximal wavelength of absorbance in five species of *Acropora*, one species of *Pocillopora* and a cyanobacterium.[169] Later, these compounds were identified as MAAs in the staghorn coral *Acropora formosa*.[170]

Most MAAs are composed of an aminocyclohexenimine ring, whereas a few, such as mycosporine-glycine and mycosporine-taurine, are mycosporines based on an aminocyclohexenone ring.[171,172] The core cyclohexenone unit is derived from the first steps of the shikimic acid pathway,[173] which is the same pathway involved in the synthesis of higher plant photoprotectants such as flavonoids.[174] This pathway has only been described in bacteria, cyanobacteria, fungi and algae and enzymes of the pathway have recently been described in the sea anemone *Nematostella* as being of bacterial and dinoflagellate origin.[175] The later biosynthetic steps in the shikimate pathway produce other types of MAAs. First, primary MAAs are produced[176,177] by the addition of free amino acids as side chains such as glycine to the core

produces mycosporine-glycine and from this compound shinorine is produced by the addition of serine.[178]

Symbiodinium appears to be restricted to producing five MAAs.[177] The biosynthesis of primary MAAs can be completely stopped or reduced in the presence of glyphosate, an inhibitor of the shikimic acid pathway,[179] while secondary MAAs are produced from primary MAAs such as the formation of palythene from porphyra-334.[180] Once primary MAAs accumulate, their conversion to secondary MAAs cannot be inhibited by glyphosate.[176] The continued production of some MAAs after the cessation of exposure to UVR indicates that these wavelengths are not required to catalyze the reactions of the shikimate pathway but rather act as a signal that induces the enzymes of the biosynthetic pathway.[176] The inhibitory effect of DCMU on *de novo* biosynthesis of MAAs indicates that photosynthesis is required for these steps.[181]

Although no research has been published on UVR receptors related to MAA synthesis in coral reef organisms, advances have been made in other systems. In cyanobacteria, a UVB specific photoreceptor for the induction of MAA synthesis called pterin has been described.[182] In the red alga *Chondrus crispus*, two photoreceptors for MAA synthesis have been hypothesized, one for blue light and one for UVA, which can act synergistically.[183] Using action spectra, Kräbs *et al.*[184] identified a UVA receptor with peaks at 320, 340 and 400 nm that induced the formation of shinorine, the principal MAA found in *C. crispus.* Two completely different signal transduction pathways have been proposed for the induction of MAAs: one that is activated by UVR and the other that does not require UVR activation but can be induced by salt stress.[159,185]

There are more than 20 formally identified MAAs and references to many unknowns or compounds characterized solely by their maximal absorbance are found throughout the literature and efforts should be made to identify them. Improvements in resolution of MAAs by high performance liquid chromatography (HPLC) have been developed[186,187] as well as improved techniques such as electrospray ionization mass spectrometry coupled with liquid chromatography (LC/MS) that will probably result in the identification of additional novel MAAs.[188–190] Substitution of the nitrogen of amino acids or imino alcohols at the C1 position of the core determines the peak of maximal absorption (λ_{max}) of each MAA. The absorption spectra of MAAs follow a normal distribution and their absorption bands are wide. The full width at half maximum (FWHM) values for Asterina-330 (λ_{max} = 330 nm), mycosporine-glycine (λ_{max} = 310 nm) and palythinol (λ_{max} = 332 nm) measure 40 nm wide whereas for purified shinorine (λ_{max}= 334 nm) and for palythine (λ_{max}= 320 nm) there values are closer to 50 nm wide[54,72,154,191] and compare to chlorophyll *a*, which has an FWHM value of 23 nm at room temperature.[192] Therefore, MAAs can individually provide a wide spectral screen against UVR and when found in combination the screening potential is even broader as the spectral absorbances of individual MAAs overlap and extend the spectral range of absorbance. The most recent MAA to be identified is Euhalothece-362 from cyanobacteria[193] thus extending the range of λ_{max} for all known MAAs from 309 to 362 nm.[172]

MAAs are found in taxonomically diverse marine and freshwater organisms ranging from cyanobacteria to vertebrates.[194] MAAs tend to be found in higher concentrations in tropical organisms than temperate species,[195] and cnidarians possess the highest diversity of MAAs among all phyla with at least 11 different MAAs characterized from scleractinian corals[157] and up to 10 MAAs identified in a single coral, *S. pistillata.*[179,181] Coral mucous[161–163,196] and the mucous of coral reef fish[197–199] also contain MAAs. Corals, like most marine and freshwater organisms, contain a suite of MAAs, thus extending the photoprotective potential across a broad spectrum. Palythine and mycosporine-glycine tend to be the most abundant MAAs in corals.[37,163,177,196,200–203] Various cnidarian species have been shown to contain MAAs exclusively in the host component,[80,165,201,204] which may be accounted for by trophic accumulation through their diet[165,176] or due to the presence of bacteria such as *Vibrio.*[152] To examine the potential contribution of prokaryotes in the synthesis of MAAs in animal tissue, one group of larvae from the scleractinian coral *Goniastrea retiformis*, whose symbiotic algae do not contain MAAs, were treated with the antibiotic rifampicin (an inhibitor of DNA transcription). MAA synthesis and conversion occurred in the larvae; therefore these results indicate a possible contribution of prokaryotes associated with the animal tissue to these processes[204] or transfer of the genes to the host.[175] The host has an important role in protecting symbionts from UVR damage with the presence of MAAs in the host tunic, which prevents the photoinhibition of photosynthesis in the *Prochloron* symbionts of the host ascidian.[205] Only ascidians in symbiosis with *Prochloron* have UVR absorbing compounds in their tunics, whereas asymbiotic ascidians have either tunics that are transparent to UVR and PAR, or the tunics are pigmented and absorb both UVR and PAR equally.[206] In didemnid ascidians MAAs are accumulated in specific cells called tunic bladder cells as a protection against UVR.[207]

MAAs are highly photostable,[208] even in the presence of heat[209] and photosensitizers,[210] such that several sunscreen candidates have been examined for sunscreen suitability and a limited selection is being tested for potential use in human skin-care and cosmetic products.[211,212] MAAs should be ideal photoprotectants because they are efficient absorbers of the potentially damaging wavelengths before they can reach sensitive cellular components. In addition these compounds harmlessly dissipate the UVR energy thus preventing the formation of singlet oxygen or superoxide radicals, which would otherwise damage cellular components. The majority (up to 97%) of the high energy photons absorbed by shinorine, porphyra-334 and palythine are rapidly dissipated as heat by very fast internal conversion processes as indicated by their low fluorescence quantum yields, absence of free radical or triplet state formation and as measured by direct photoacoustic calorimetric determinations there is no energy transfer to chlorophyll *a*.[208,210,213–216] Due to their high molar absorptivity (ε ranges from 28 100 to 50 000 l mol^{-1} cm^{-1}), MAAs absorb very efficiently within the wavelength range from 309 to 362 nm. In addition, the high amount of packaging of these compounds in intact cells of dinoflagellates around UVR-sensitive organelles increases the efficiency of UVR absorption by MAAs.[217] The disparity between the relatively low spectral absorbance in the UVR by intact dinoflagellates in growth medium with the high concentrations of these compounds as determined by HPLC suggests that the MAAs are tightly packaged within the cells.[217]

To show that MAAs are effective as photoprotectants it needs to be demonstrated that the accumulation of these compounds results in a concomitant increase in UVR resistance. This has been clearly established using BWFs for the inhibition of

photosynthesis in a bloom-forming dinoflagellate *Akashiwo sanguinea* (= *Gymnodinium sanguineum*) under cultured conditions[218] as well as in the green sea urchin *Strongylocentrotus droebachiensis*.[191,219,220] High concentrations of MAAs in high light cultures of *A. sanguinea* eliminated the sensitivity of photosynthesis to UVR in the range from 320 to 360 nm, whereas the low light cultures with low concentrations of MAAs were highly sensitive within this wavelength range.[218] The MAA absorption spectrum corresponded almost exactly to the estimated biologically-effective spectrum in the UVR, strongly suggesting that these optical screens provided complete protection within the most biologically damaging wavelength range.[218] First cleavage delay in eggs as well as the level of UVR-induced developmental abnormalities in embryos and larvae of *S. droebachiensis* are negatively correlated with MAA concentration also demonstrating the photoprotective role of MAAs.[191,219] However, in this case the MAAs do not provide complete protection because sea urchin larvae with high MAA concentrations suffered some cleavage delays as well as abnormalities.[191,219] The accumulation of DNA damage could be responsible for some of the cleavage delays in embryos exposed to UVR.[220]

Many studies provide supporting evidence for the photoprotective role of MAAs. The extracts of UVR absorbing substances from the marine red alga *Porphyra yezoensis* reduce the production of UVR-dependent thymine photodimers by a direct molecule-to-molecule energy transfer process.[221] In most corals there is a positive correlation between MAA concentration and solar UVR. MAA concentrations vary seasonally,[224] and decrease with increasing depth[37,40,72,77,129,161,222,223] as well as in experimental manipulations that filter out UVR.[88,165] Interestingly, MAA concentrations in corals decrease with low water flow and this flow modulation of MAA synthesis is directly related to rates of photosynthesis.[149,225,226] The higher production of MAAs in female soft corals prior to spawning in comparison to male colonies (up to 67% and 56% for *Lobophytum compactum* and *Sinularia flexibilis*, respectively) indicates that the parent is providing photoprotectants to its offspring,[224] which is important for larval survival in *L. compactum*[227] and *Heteroxenia fuscescens*.[203]

In organisms such as invertebrates and vertebrates that are incapable of producing MAAs, the photoprotective compounds can be acquired from their diet and translocated to different tissues as demonstrated in holothuroids,[228] tridacnid clams[80] and teleost fish.[229] The diet of coral reef fish strongly influences the composition of UVR-absorbing compounds in mucous and as long as they are available in the diet the fish can adapt to UVR exposure.[230] Diet does not however affect the MAA content in the scleractinian coral *S. pistillata*.[181] In symbiotic species, MAAs may also be acquired from symbionts as is the case in the upside-down jellyfish *Cassiopeia xamachana*.[165] The scyphistomae stage is aposymbiotic and does not contain MAAs. Under cultured conditions the symbiont, *Symbiodinium microadriaticum*, produces MAAs and leaks them into the surrounding medium. After infection of the scyphistoma with symbiotic algae, the ephyrae and adult jellyfish stages contain the same suite of MAAs as the symbiotic algae in culture.[165]

Due to the inherent difficulties of identifying *Symbiodinium* spp. sequence diversity of the small subunit ribosomal DNA gene (SSU rDNA) has been used to assign *Symbiodinium* into clades.[231] Currently, the number of recognized clades is eight and are designated A to H,[232] with clades A through D maintaining associations with scleractinian corals. Although clade assignment allows for the grouping of different *Symbiodinium* into phylogenetically similar types there have been some attempts, albeit unsuccessful, to assign physiological traits to these clades. The only case in which clade designation aligns with a physiological trait is in the synthesis of MAAs by *Symbiodinium* under specific conditions.[233] In culture, Clade A *Symbiodinium* synthesize MAAs, whereas clade B and C *Symbiodinium* do not,[233] but *in hospite* at least one MAA is present in all *Symbiodinium* regardless of clade designation,[177] although corals hosting clade A symbionts contain higher concentrations and diversity of MAAs under experimental conditions.[97]

While the physiological role of MAAs is clearly the protection from harmful UVR by physical screening, additional roles, as antioxidant molecules scavenging ROS and as osmolytes to cope with salt stress, amongst others, have led them to be considered as secondary metabolites due to their apparent multiple functions. Mycosporine-glycine has been shown to have moderate, concentration-dependent antioxidant activity by scavenging free radicals in extracts from marine organisms[234] and quenching singlet oxygen.[235] Mycosporine-glycine was able to effectively suppress the inactivation of mitochondrial electron transport, lipid peroxidation of microsomes, hemolysis of erythrocytes and growth inhibition of *Escherichia coli* by markedly reducing the levels of singlet oxygen ($^1O_2$) produced by photosensitizers under illumination.[235] Mycosporine-glycine also provides rapid protection against oxidative stress before antioxidant enzymes are produced in two species of scleractinian corals, *Platygyra ryukyuensis* and *S. pistillata*.[236] It may not be a coincidence then that mycosporine-glycine is the most frequently observed MAA and has the highest concentration of those MAAs observed in a diverse range of cnidarian species.[37,72,77,88,129,170,196,222] A study by Portwich and Garcia-Pichel[185] suggests that, although the MAAs mycosporine-glycine and shinorine could be induced by salt stress, they play no significant role in attaining osmotic homeostasis, although in other studies they have been considered to be osmolytes.[237] The role of MAAs as osmolytes in coral symbioses has not been investigated but the coral host does need to maintain a compatible osmotic environment both with its endosymbionts as well as with the external environment.[238]

MAAs have also been considered as nitrogen reservoirs due to the rapid mobilization of ammonium to MAAs.[239] Due to their high nitrogen content, MAAs have also been suggested to be an intracellular nitrogen source when nitrogen is limiting.[66,240,241] Starved corals continued to accumulate MAAs and conserved them disproportionally compared with declining protein and chlorophyll *a*, indicating the priority placed on maintaining this UVR-sunscreen defense.[181] Ammonium consistently affected only the accumulation of primary *Symbiodinium* MAAs (mycosporine glycine, shinorine, porphyra-334, and mycosporine-2 glycine), and not secondary MAAs derived from the former.[181] Lastly, MAAs have been proposed to be the so-called "host factor" in species symbiotic with *Symbiodinium* which facilitates the transfer of photosynthate between symbiont and host.[242]

Fluorescent proteins

The brilliant colors of scleractinian corals are due, in part, to a family of green fluorescent-like proteins (GFPs) that fluoresce in

the presence of UVR or PAR. UVR induced fluorescence has been reported in diverse species and locations such as the Pacific, Caribbean and Red Sea by Catala-Stucki,[243] Logan *et al.*[244] and Schlichter *et al.*,[245] and are now known to be a part of a large family of fluorescent proteins that are widely distributed amongst symbiotic cnidarians.[246]

Originally isolated and described from the hydromedusae *Aequorea victoria*, the 238 amino acid protein, within which three residues at positions 65–67 form the active chromophore, is extremely resistant to extremes in pH and temperature.[247] In corals these fluorescent proteins are located principally in the epithelial cells of the cnidarian host,[248–251] although they can be observed in gastrodermal tissue as well,[252] and little is known about the function of this protein in corals. Kawaguti[248,249] demonstrated that the dissipation of high-energy UVR *via* its absorption and subsequent fluorescence at longer (PAR) wavelengths in corals is accomplished by green pigments concentrated in granules. It was proposed that these fluorescent proteins provide photoprotection under high-light conditions[252–254] or enhance photosynthesis under low-light conditions,[254] or both depending on the position of the fluorescent pigment relative to the zooxanthellae. Of the proposed functions described above there is little possibility that fluorescent proteins could improve photosynthetic performance as fluorescence resonance energy transfer (FRET) between fluorescent proteins occurs but transfer from fluorescent proteins to chlorophyll does not occur in corals.[251,254] Additionally, for the Caribbean coral *M. faveolata* the lack of any depth-dependent differences in the expression of GFP does not support the hypothesis that these proteins can protect the holobiont from the deleterious effects of UVR.[251] It has however been recently demonstrated that GFPs can quench $O_2^{\bullet-}$.[255] The modest SOD-like activity of GFP may well be compensated by its high concentration in corals,[251,256] making it a significant contributor to the overall antioxidant defenses of corals. GFP protein concentration[257] also decreases in corals exposed to thermal stress. This is consistent with an *in hospite* environment where high fluxes of $O_2^{\bullet-}$ occur during thermal stress, and GFPs can quench $O_2^{\bullet-}$ but not without a decrease in GFP concentrations, which is caused by oxidative degradation of the protein[255] along with a decrease in transcription of the gene.[258]

Conclusions

Any increase in UVB irradiance around the globe is likely to stabilize and then decrease due to the continued increases in stratospheric ozone. Despite continued global climate change that could delay the rate of expected increases in stratospheric ozone, UVB irradiances will not increase significantly in the tropics where most coral reefs occur and alone are not likely to cause coral reef degradation. We now have significantly more biological data since the last comprehensive review on the effects of UVR on coral reef organisms.[15] Based on the data available we would make the following conclusions. First, we can confirm the hypothesis originally posed by Jokiel[67] that UVR is an important influence on the community structure in shallow water coral reef environments and has been over the entire evolutionary history of coral reefs. Due to the attenuation of UVR by the water column and its constituents, the prediction by Shick *et al.*,[15] that UVR will only have significant effects in shallow reef environments, is reaffirmed with additional data since 1996. Second, in response to UVR as a constant evolutionary selective pressure, shallow water dwelling coral reef organisms have been shown to have developed several effective defence mechanisms, in particular the synthesis or accumulation of MAAs. Other photoprotective mechanisms have not been adequately studied in coral reef dwelling organisms nor has the interaction between different photoprotective mechanisms been adequately addressed. Third, the relatively recent changes in global climate due to continued increases in anthropogenic CO_2 emissions will lead to more bleaching events in which UVR, especially UVA, will have a very important interacting role. As corals bleach the host tissues are potentially exposed to higher UVR irradiances, thus accelerating the bleaching process. We have the following observations and recommendations for future studies: (1) The recommendations outlined in Shick *et al.*[15] and Gleason[103] regarding the need for a quantitative understanding of the population and community level changes influenced by UVR alone, or interacting with other abiotic factors, has not been realized. Fully understanding the independent and interactive effects of UVR on coral reef community structure may not be practical or possible at this time as the coral reef research community deals with multiple stressors affecting coral reefs at unprecedented spatial and temporal scales such as increases in seawater temperature and ocean acidification. Nevertheless, the interaction between these stressors and UVR has not been adequately studied, but it is essential to conduct these studies for better planning of coral reef restoration efforts. (2) In regard to UVR resistance, no work to our knowledge has been conducted on UVR resistant phenotypes or species as suggested by Shick *et al.*,[15] and this may be another component that will be essential for plans directed at restoration of coral reefs. Lastly, (3) one principle area of UVR photobiology is woefully understudied and that is the effect of UVR exposure on DNA damage, both directly as thymine dimers, and indirectly as strand breaks caused by exposure to ROS. This should include a comprehensive examination of repair mechanisms such photoreactivation and dark repair. Emphasis should also be placed on determining the energetic costs associated with the various photoprotective and repair mechanisms as trade-offs to growth and reproduction.

Undertaking studies on the effects of UVR exposure for coral reef organisms is an important and difficult endeavor. The requirements for excellent spectroradiometric measurements, accurate simulation of naturally occurring PAR : UVA : UVB ratios, with or without UVB enhancement due to changes in stratospheric ozone, and good experimental design using appropriate filter material still elude many studies. But these components are necessary if we are to understand the evolutionary and ecological importance of this critical abiotic factor on coral reefs.

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