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ABSTRACT

Hermatypic (reef building) corals live in an environment characterized by high ambient levels of photosynthetically active radiation (PAR) and ultraviolet radiation (UVR). Photoadaptive mechanisms have evolved to protect the sensitive cell structures of the host coral and their photosynthetic, endosymbiotic zooxanthellae. Environmental stressors may destabilize the coral–zooxanthellae system resulting in the expulsion of zooxanthellae and/or loss of photosynthetic pigment within zooxanthellae, causing a condition known as bleaching. It is estimated that 1% of the world's coral population is lost yearly, partly due to bleaching. Despite intensive research efforts, a single unified mechanism cannot explain this phenomenon. Although UVA and UVB cellular damage is well documented, UVC damage is rarely reported due to its almost complete absorption in the stratosphere. A small scale coral propagation system at the University of Maine was accidentally exposed to 15.5 h of UVC radiation (253.7 nm) from a G15T8 germicidal lamp, resulting in a cumulative surface irradiance of 8.39×10^4 J m^{-2}. An experiment was designed to monitor the progression of UVC induced damage. Branch sections from affected scleractinian corals, *Acropora yongei* and *Acropora formosa* were submitted to histopathology to provide an historical record of tissue response. The death of gastrodermal cells and necrosis resulted in the release of intracellular zooxanthellae into the gastrovascular canals. Zooxanthellae were also injured as evidenced by pale coloration, increased vacuolization and loss of membrane integrity. The recovery of damaged coral tissue likely proceeds by re-epithelialization and zooxanthellae repopulation of gastrodermal cells by adjacent healthy tissue.

1. Introduction

Shallow water reef building corals are continuously exposed to high ambient levels of photosynthetically active radiation (PAR: 400–700 nm) and ultraviolet radiation (UVR: 280–400 nm, Jokiel, 1980). This is primarily due to a higher angle of incidence of the sun near the equator, a thinner stratospheric ozone layer, and to characteristically low levels of organic particulate matter within the water column that absorb or deflect incoming irradiance and UVR (Browman et al., 2000). Clear, calm waters allow for deeper penetration of UVR with detrimental effects detectable to 20 m (Sinclair et al., 2005). The direct absorption of photons by DNA is wavelength-dependent and tends to drop off above 300 nm. The shorter wavelengths within the UVA range (320–400 nm) cause indirect damage to DNA, proteins and lipids through reactive oxygen intermediates. UVB (280–320) causes indirect and direct damage to DNA with the formation of cyclobutane pyrimidine dimers. These DNA lesions are lethal to the cell because they interfere with replication and transcription (Baruch et al., 2005; Lesser and Farrell, 2004).

Photoenzymatic repair (PER) and nucleotide excision repair (NER) mechanisms have been well described (Mitchell and Karentz, 1993). UVC (200–280 nm), although extremely dangerous, is largely absorbed by ozone in the stratosphere and currently has little measurable environmental impact (Dunlap et al., 1986) although concerns have been raised about UVC exposure in areas experiencing a denuded ozone layer such as Antarctica, Southern Chile and Southern Argentina (Staehelin et al., 2001).

Sessile, shallow water corals rely almost exclusively on physiological mechanisms to adapt to high levels of irradiance and ultraviolet radiation. The production of mycosporine-like amino acids and other pigments to selectively absorb, reflect, or fluoresce ultraviolet is a photoadaptive strategy to protect sensitive cell structures (Dunlap et al., 1986; Siebeck, 1988).

Endosymbiotic zooxanthellae mediate the flux of carbon and nutrients between the host and the environment. The photosynthetic activity of zooxanthellae results in a high pO_2 in the tissues and leads to the generation of reactive oxygen species. The enzymes superoxide dismutase, catalase, and ascorbate peroxidase

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are produced by the host and the zooxanthellae to protect against oxygen toxicity (Lesser et al., 1990). Maximum photosynthetic activity is the result of increased enzyme production, but is energetically costly. Increased levels of short wavelength, highly energetic UV place an additional stress on the coral organism to detoxify reactive oxygen species. If the coral is unable to compensate for this environmental stressor it may result in tissue damage or death and the expulsion of zooxanthellae (Gates et al., 1992). Bleaching, the mass expulsion of symbiotic zooxanthellae and/or the loss of photosynthetic pigment within individual zooxanthellae (Glynn and de Weerdt, 1991), has attracted world wide attention and is most often associated with the global warming phenomena. Low water temperatures, changes in salinity, irradiance (PAR) and increases in ultraviolet radiation can also act alone or synergistically to cause bleaching (Lesser et al., 1990; Glynn et al., 1985).

Recent studies focusing on the waters surrounding the Antarctic continent and within the Antarctic ozone hole have found that the primary production of phytoplankton has decreased relative to similar areas during cycles of ozone depletion and increased UVB exposure. The early planktonic stages of various invertebrates and fish were also vulnerable to UVB induced injury (Karentz and Bosch, 2001). However, the overall effects on the ecosystem are far too complex to predict with any certainty. Despite atmospheric measurements that indicate the rate of ozone loss has slowed, total ozone is expected to drop through this decade resulting in a 5–10% increase in the average dose of biologically harmful UV radiation (de Gruijl, 1995). Although exposure to UVC may appear to be an environmentally unrealistic scenario, work with amphibians has demonstrated that UVC exposure of the egg nucleus induced developmental neural abnormalities in embryos, essentially the same as those induced by UVB damage (Light and Grant, 1997).

The purpose of this paper is to provide a histological reference of tissue damage and recovery in a scleractinian coral following an accidental UVC radiation exposure. This work may be useful in helping to establish the mechanisms involved in UVR damage repair within the coral–zooxanthellae system.

2. Materials and methods

The exposure occurred in small, warm water, environmentally controlled aquarium that houses a stony coral propagation system of approximately 465 l total capacity including sump, with an exposed surface area of 0.53 m^2. The system volume turnover rate is 11.7 per hour. Water quality parameters were intended to replicate ambient conditions of the tropical Indo-Pacific and were monitored with a Hach Systems Spectrophotometer and digital titrator. Supplemental sodium carbonate, calcium chloride and magnesium chloride/sulphate maintained alkalinity at 3.0–3.5 meq l^{-1}, calcium at 400 mg l^{-1}, and magnesium at 1250–1300 mg l^{-1}. Specific gravity and salinity were at 1.025 and 35 g l^{-1}, pH at 8.0–8.2, total ammonia and nitrite at undetectable levels and nitrate and phosphate at <10 mg l^{-1} and <0.03 mg l^{-1}, respectively. The temperature was kept at 26 °C. Artificial lighting was provided with a single DE 250 W, 10,000° (color temperature) metal halide fixture located 50 cm above the water's surface, on a 9 h light: 15 h dark cycle.

On the ceiling of each corner a fluorescent fixture housed a GE 15 W, mercury vapor, G15T8 Germicidal lamp. This lamp is rated at 49 μW cm^{-2} at 1 m, and emits monochromatic ultraviolet-C at 253.7 nm. This wavelength is close to the absorption maximum of nucleic acids and many other biological molecules and is energetic enough to initiate chemical reactions. It is therefore very destructive to living matter and is used for sterilization (Björn and Teramura, 1993). The closest lamp was approximately 1.73 m above the coral propagation tank.

An inadvertent exposure of the corals in this system occurred in the evening when the germicidal lights were accidentally switched on, and were not turned off until 08:30 the following morning, resulting in an exposure time of 15.5 h. Realizing that a dramatic effect had taken place, an experiment was designed to monitor the progression of UVC induced damage. Starting at day 1, 24 h after exposure, and on days 3,5,7,10,16, 22 and 32, 5 mm long cross sections of a coral branch (starting with the axial polyp) from an exposed *Acropora yongei* and *Acropora formosa* colony were removed, placed in 10% neutral buffered formalin, decalcified with CalExII, placed in a standard tissue processor for 16.5 h, then embedded in paraffin. Five μm sections were cut and then stained with hematoxylin and eosin.

3. Results

The total cumulative surface irradiance (total absorbed dosage) is the product of the fluence (energy per unit area) of this bulb at 1.73 m (0.016 mW cm^{-2}) and the total exposure time of the corals (5.58×10^4 s) and calculates to be 8.93×10^4 J m^{-2} at surface level. The affected corals were approximately 20 cm below the water surface. The attenuation of UVC at this depth is unknown, but is probably negligible, and was not adjusted into the final calculation. Corals that were not shaded by the presence of the overhead metal halide fixture appeared on clinical examination to have severe polyp retraction and loss of pigmentation, including zooxanthellae-contributed pigmentation, on their exposed surfaces. Severely injured specimens of *Montipora capricornis, Acropora millepora, A. formosa* and various zoanthid polyps failed to survive longer than 21 days post UVC exposure. Long term survivors include fragments of *A. yongei*,*A. millepora, Hydnophora* sp. an unidentified acroporid and a cleaner shrimp (*Lysmata amboinensis*). Due to the angle of incidence of the UV, a line of demarcation developed on the tank walls and on live rock, where pink coralline algae (Corallinaceae) were bleached white. Also, a calcareous green alga (Halimeda sp.) was bleached. Sadly, but perhaps not coincidentally, a large yellow tang (*Zebrasoma flavescens*) was observed to have an erythematous trunk lesion, but died and decomposed before it could be examined.

The following is a brief description of the sequence of events culminating in the recovery of an exposed *A. yongei* colony, and the death of an *A. formosa* colony. The DE 250 W, 10,000° (color temperature) metal halide light was kept at 50 cm above the water's surface on a 9 h Light: 15 h dark cycle for the duration of the experiment.

Days 1–7 *A. yongei* post exposure (Plate 1). Physical exam reveals severe tissue damage with polyp retraction and the loss of green fluorescent pigment of host cell origin on both exposed and unexposed surfaces. Apparently, tissue remains on both surfaces, but zooxanthellae survive only on the unexposed surface lower left arrow (Plates 2–4). At the microscopic level, tissue necrosis and the separation of epidermis and gastrodermis from the mesoglea result in the release and subsequent loss of zooxanthellae into the gastrovascular canals and into the water column (Coral Disease and Health Workshop, 2005). Note the swelling of the mesoglea as epithelia slough off and seawater is absorbed by this primitive connective tissue (Plate 5). Deeper sections reveal degenerating zooxanthellae adjacent to necrotic gastrodermal tissue, with an overall loss of normal tissue architecture.

Days 7–16 post exposure (Plates 6 and 7). Tissue shows unusual degeneration with an accumulation of amorphous, necrotic cellular debris within gastrovascular canals and pale, hypertrophic zooxanthellae, with increased vacuolization and the loss of membrane integrity, likely due to the effects of UVC exposure. Although tissue architecture appears disorganized, in some places the mesoglea is clearly lined by epithelia. A cell in mitosis (telophase) is present on

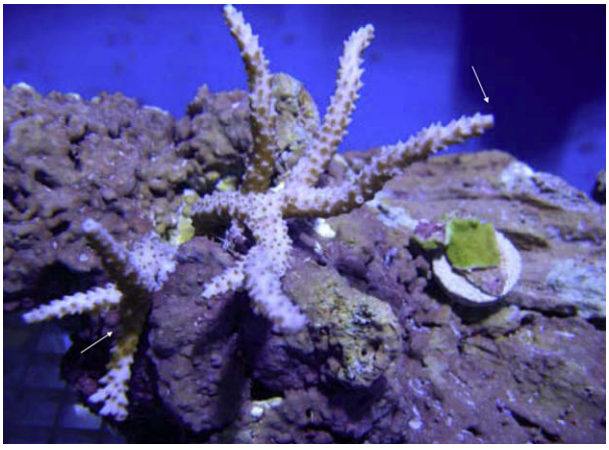

Plate 1. Day 1: *Acropora yongei*, 24 h post UVC exposure. Healthy tissue shaded by overhead branch (lower arrow). Site of tissue sections (upper arrow). Small fragment of *Montipora capricornis* was added later to act as a comparison for loss of pigmentation.

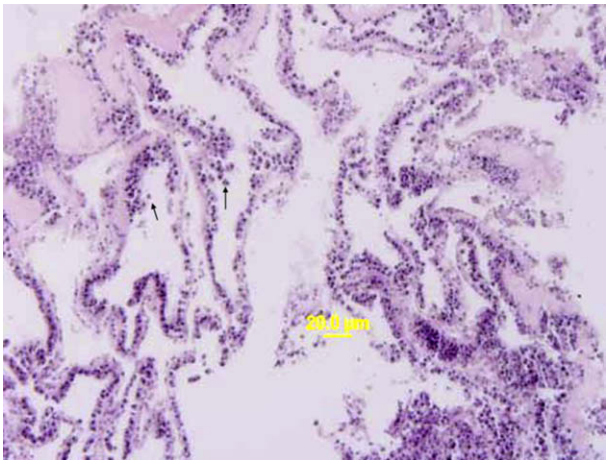

Plate 2. Day 1: *Acropora yongei* section through apical tissue with axial polyp. Free zooxanthellae (arrows) released from necrotic gastrodermal tissue, (10× – denotes objective, not total magnification).

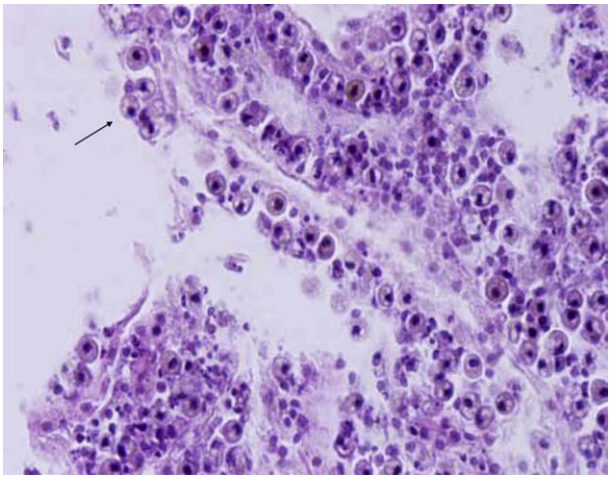

Plate 3. Day 1: *Acropora yongei* section through apical tissue with axial polyp, degenerating zooxanthellae and necrotic tissue, 40×.

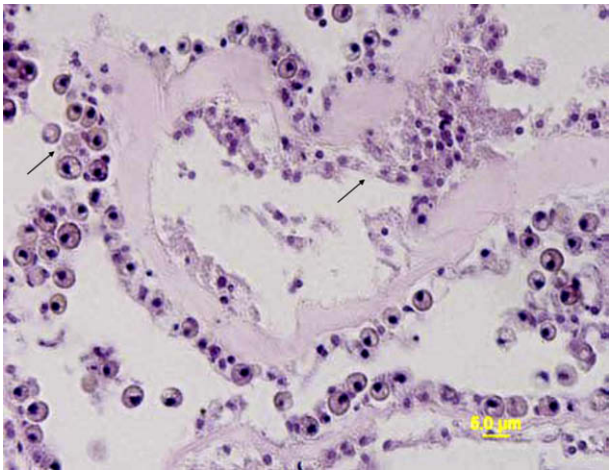

Plate 4. Day 1: *Acropora yongei* section through apical tissue. Free, degenerating zooxanthellae, necrotic tissue and loss of normal tissue architecture, 40×.

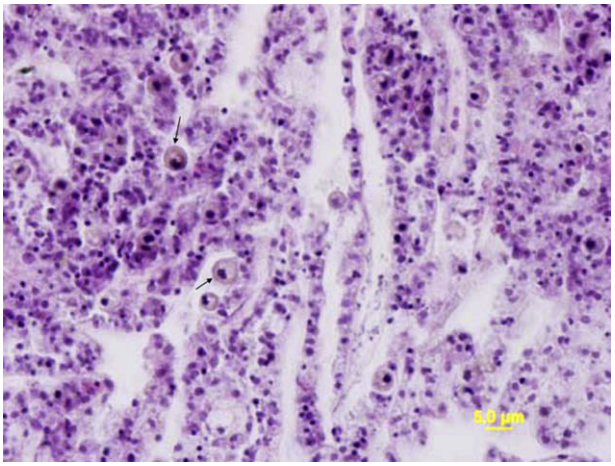

Plate 5. Day 3: *Acropora yongei*, degeneration and vacuolization of free zooxanthellae, 40×.

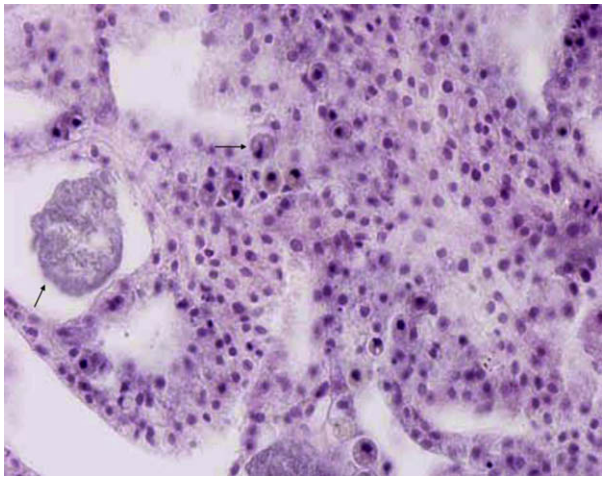

Plate 6. Day 7: *Acropora yongei*, amorphous debris (mesentery remnant) within coenenchyme, degenerate zooxanthellae. Note: amorphous debris negative for bacteria with Brown and Brenn gram stain (not shown).

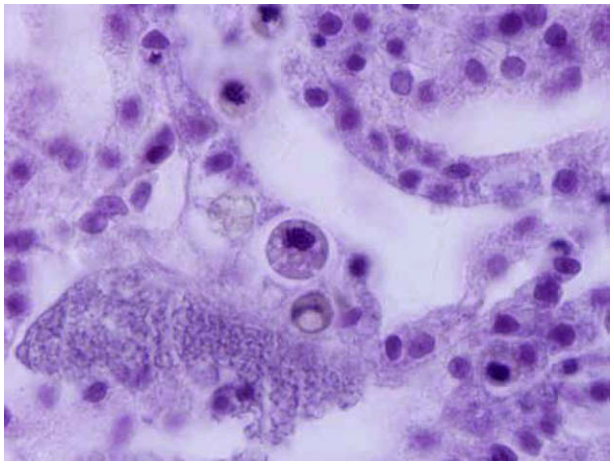

Plate 7. Day 7: *Acropora yongei*, detail of 6. Hypertrophic, vacuolated zooxanthellae, 100×.

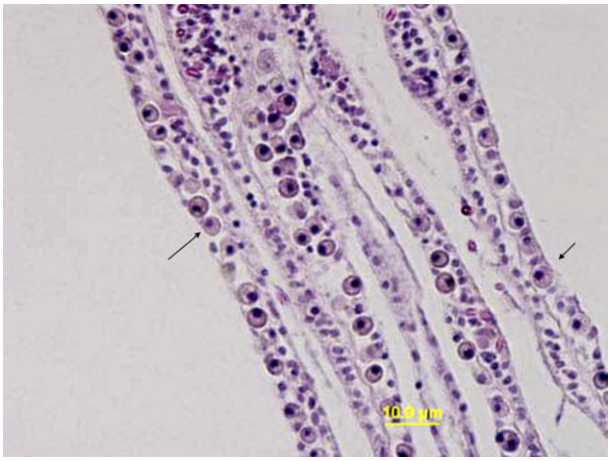

Plate 9. Day 22: *Acropora yongei*, Enlargement of 8 (central arrow area). Zooxanthellae within vacuoles, within normal appearing gastrodermal tissue, 40×.

the right side of, indicating that some cells are capable of division now.

Days 22 and 32 post exposure (Plates 8–10). Healthy tissue from the adjacent unexposed tissue margins, or perhaps from cryptic tissue in the exposed area, overgrows denuded corallum. This appears dramatic due to the retrograde sectioning. Sagittal section of radial polyp reveals normal appearing gastrodermis and intracellular zooxanthellae within vacuoles (Plate 11). Apparently healthy tissue overgrows bare skeleton at transection sites. Green fluorescent pigmentation is returning and polyps are beginning to open. Note the presence of spirocysts beginning to aggregate (Plate 10).

A. formosa Days 1–5 post exposure (Plates 12–16). Severe tissue injury results in the separation of the epidermis from the gastrodermis and the release of degenerating zooxanthellae. The coral was likely dead on Day 1 due to the more severe tissue damage resulting from the exposure and the entire skeleton of this colony was denuded of tissue by 10 days post exposure.

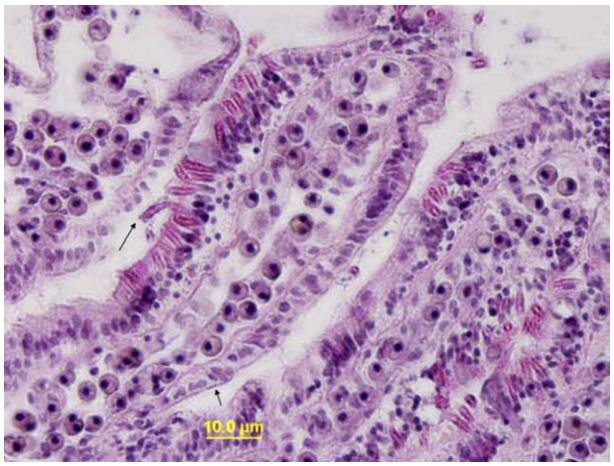

Plate 10. Day 32: *Acropora yongei*, Zooxanthellae. Spirocysts (upper arrow). Epithelial cells (lower arrow), 40×.

4. Discussion

The heightened interest in ultraviolet photobiology has been preceded by concern from atmospheric scientists in the 1970s that

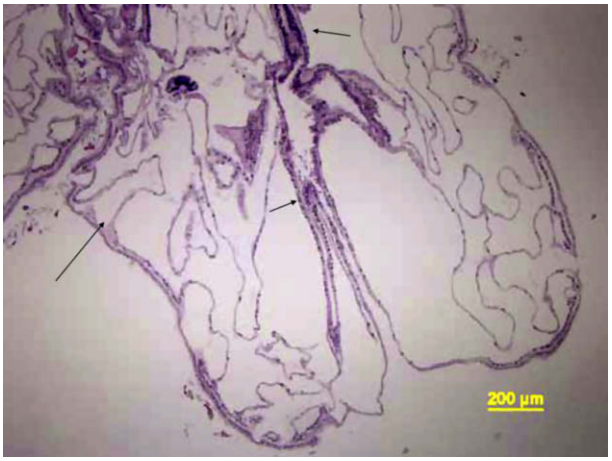

Plate 8. Day 22: *Acropora yongei*, Sagittal section of radial polyp. Normal appearing gastrodermis with zooxanthellae. Gastrovascular (middle arrow) and canals (left arrow). Mesentery with mesenterial filaments (top arrow), 10×.

Plate 11. *Acropora yongei*, 90 days post UVC exposure. Upper right arrow: site of sectioning for experiment. Middle right arrow: site of deliberate transection for propagation of fragment. Left arrow: note normal zooxanthellae coloration and denuded corallum, etiology unknown. Lower right arrow: coral tissue overgrowth onto rock.

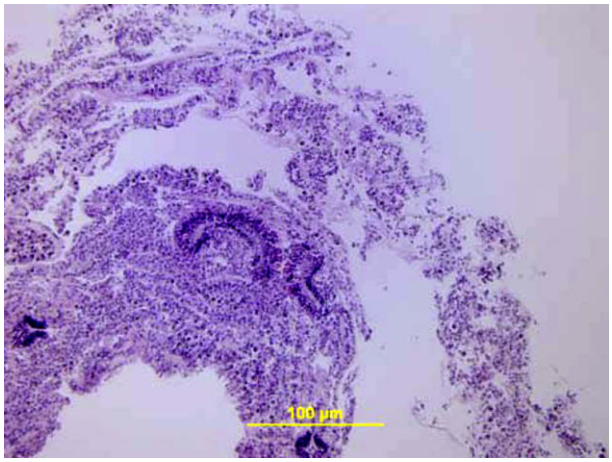

Plate 12. Day 1: *Acropora formosa*, Epidermis separating from gastrodermis, 10×.

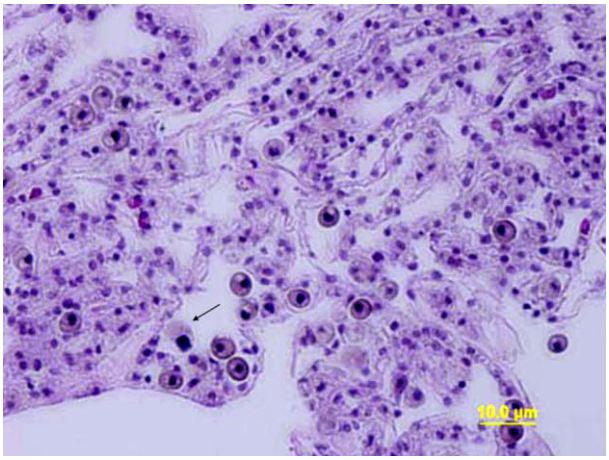

Plate 15. Day 3: *Acropora formosa* unknown cell type (arrow), 40×.

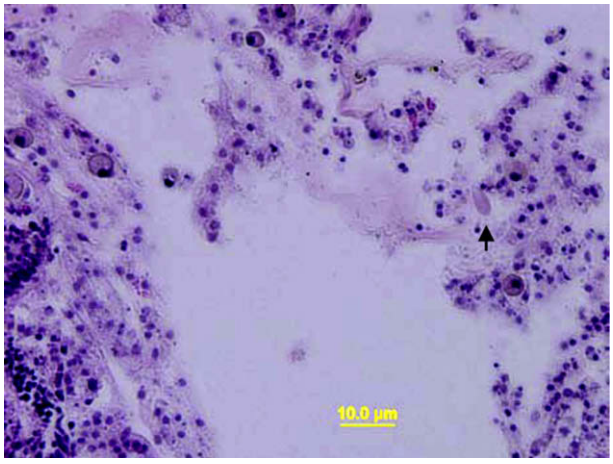

Plate 13. Detail of 12 – tissue necrosis and sloughing. Zooxanthellae degeneration, possible organism (arrow), 40×.

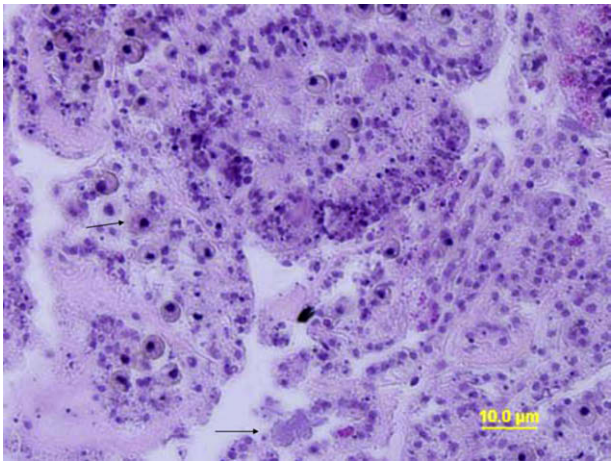

Plate 16. Day 5: *Acropora formosa*, degenerating zooxanthellae (upper arrow). Amorphous debris (lower arrow), 40×.

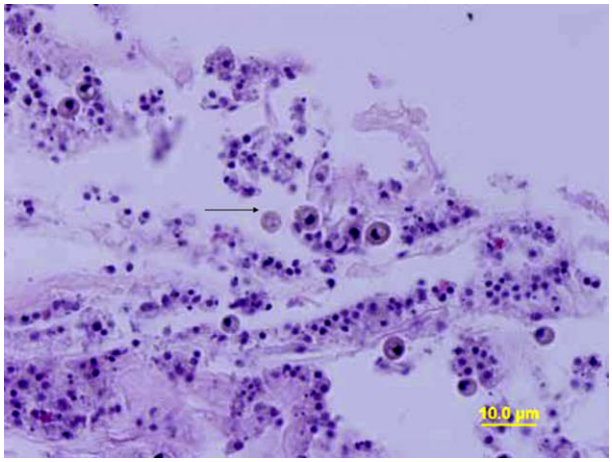

Plate 14. Day 1: *Acropora formosa* apical section, including remnant of axial polyp. Degenerating zooxanthellae (arrow), 40×.

our protective ozone layer is in jeopardy due to the dramatic increase in stratospheric supersonic transportation (McDonald, 1971). Molina and Rowland (1974) raised the spectre of anthropogenic emission of chlorofluorocarbons into the atmosphere as a potential threat to the ozone layer. Discovery of the Antarctic ozone hole in the mid-1980s led to similar findings in the Arctic and in middle latitudes. It is speculated that due to depletion at high latitudes, ozone is drawn poleward resulting in mid-latitude thinning (Björn et al., 1998).

As was noted in the introduction, UVC radiation (220–280 nm) normally does not reach the terrestrial level and therefore would not be expected to have an impact on reef invertebrates. Although a discussion of the ecology of microbial assemblages is beyond the scope of this paper, it is interesting that bacteria have been identified that are resistant to the sterilizing effects of UVC (Joux et al., 1999). This extreme example of photoadaptive repair mechanisms suggests that other planktonic organisms may have a similar ability (Smith et al., 1992). The vast majority of work however, has focused on UVB (280–320 nm) and the shorter wavelengths within the UVA range (320–400 nm). Based on the research to date, it is reasonable to conclude that most organisms are living under ambient UVR stress and that enhanced levels due to environmental changes may cause cellular damage (Voytek, 1989).

In this accidental UVC exposure experiment, gastrodermal cell death and necrosis may result in the release of intracellular zooxanthellae into the gastrovascular canals and likely into the water column, causing the observed bleaching effect of the exposed tissue (however, it is also possible that the initial damage to zooxanthellae pigmentation by UVC exposure led to gastrodermal cell

death). This is one of five proposed mechanisms of release described by Gates et al. (1992). It is also apparent that zooxanthellae may be injured by UVC radiation. An increase in zooxanthellae vacuolization was also observed by Hayes and Bush (1990) in a coral bleaching event of unknown etiology in the Cayman Islands.

Recovery of the *A. yongei* (commonly known as "Bali green slimer" for its copious mucus production) proceeded rapidly, likely due to photoreactivating repair mechanisms in the presence of intense light, excellent water quality, and the lack of secondary bacterial infection. In addition, forceful, chaotic water movement facilitated the removal of necrotic debris. Reepithelialization was aided by the translocation of photosynthetic by-products and zooxanthellae from unexposed healthy tissue. This new tissue may express substances (antimicrobial peptides) to kill and overgrow bacterial and algal biofilms. It is interesting to note that the *A. yongei* colony, in this experiment expressed green fluorescent pigment uniformly prior to exposure. The *A. formosa* colony only had fluorescent pigment on the axial polyps. Fluorescent pigment granules attain their highest density in areas of rapid cellular division and areas immediately above reproductive organs, suggesting their role in photoprotection (Salih et al., 2000). Although both hard coral species typically occupy turbulent, shallow water, high solar irradiance reefs, there are likely species-specific variations in sensitivity to ultraviolet that may be correlated to the production of mucus, fluorescent pigments and/or mycosporin-like amino acids.

Work with mammalian cell lines has demonstrated that the cell killing effects of UVC and UVB depend mostly on nuclear damage, whereas UVA incites cytoplasmic damage (Beer et al., 1993) At this time only a tenuous connection can be made to injury and photoprotective mechanisms in coral tissue and zooxanthellae based on data derived from UVA and UVB studies. Further work is needed to more clearly define the differences between ultraviolet induced injury and injury caused by other environmental stressors.

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