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Coral bleaching: the role of the host

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Coral bleaching caused by global warming is one of the major threats to coral reefs. Very recently, research has focused on the possibility of corals switching symbionts as a means of adjusting to accelerating increases in sea surface temperature. Although symbionts are clearly of fundamental importance, many aspects of coral bleaching cannot be readily explained by differences in symbionts among coral species. Here we outline several potential mechanisms by which the host might influence the bleaching response, and conclude that predicting the fate of corals in response to climate change requires both members of the symbiosis to be considered equally.

Reefs under threat from global warming

Coral reefs are under severe threat from many sources, including mass coral mortality events caused largely by sea surface temperature (SST) anomalies associated with global warming [1]. Corals are a symbiosis between the animal host and micro-algae from the genus *Symbiodinium*. Under stress, such as elevated SST, the algal endosymbionts are expelled, a process that is known as coral bleaching. Projected increases in SST caused by global warming have led some authorities to predict that reefs might disappear entirely within 20–50 years [2]. However, this prediction is based largely on the assumption that corals will not be able to adapt to accelerating rates of environmental change [3] (Box 1). One mechanism that might allow corals to rapidly adjust to the projected rises in SST is for the host to switch to a more thermally tolerant symbiotic partner [4]. This mechanism has received considerable attention in the recent literature; however, experimental support remains inconclusive [5–7]. One positive result of this recent focus on the role of the symbiont has been a rapid increase in physiological research, and significant progress has been made toward understanding the mechanisms of bleaching (see next section). Unfortunately, there has yet to be a parallel increase in ecological research to explore the consequences of bleaching, particularly at the population level, possibly because the role of the host in influencing its fate is less well recognised. Here we outline evidence for a role of the host in determining a coral's bleaching response and propose several potential mechanisms to explain this role. We conclude that an understanding of the threat climate change poses to reef corals will remain incomplete unless both members of the symbiosis are considered equally. We argue that the holobiont (the animal and symbiont in combination) is the relevant unit of selection and therefore the appropriate target for the research required to determine the capacity of coral to respond to changing climate.

The photoinhibition model of coral bleaching

The recent emphasis on the role of the symbiont in thermal tolerance is justified by the finding that thermal bleaching begins with an accumulation of oxidative stress at photosystem II (PSII) in the symbiont, a process known as photoinhibition [8]. Because photoinhibition sensitivity to increased temperature varies among *Symbiodinium* clades, differences in sensitivity among corals are determined, in some species, by the symbiont [9,10]. However, the response of the holobiont is not always related to differences in the thermal tolerance of the symbiont. For example, coral species vary considerably in their response to thermal stress [11]. Some genera, such as *Stylophora*, *Pocillopora* and *Acropora*, are highly susceptible to bleaching, whereas *Cyphastrea*, *Goniopora*, *Galaxea* and *Pavona* are highly resistant, and this hierarchy of susceptibility is consistent over a wide geographic scale [12]. However, these differences in susceptibility among species are not caused by species hosting different types of symbionts. In particular, some common *Symbiodinium* types, such as C1 and C3, occur in many different host species with a divergent response to temperature, including the highly susceptible *Seriatopora hystrix*, and many taxa recognised as resilient to thermal stress, including *Favia*, *Goniastrea* and *Platygyra* [13]. Furthermore, the production of reactive oxygen species (ROS) in cells is a common early event in response to many types of stress and is not unique to photosynthetic organisms (Figure 1). Indeed, physiological changes in the host, such as reduced thickness of the epidermis and apoptosis of gastrodermal cells, might precede changes in symbionts when corals are exposed to heat [14]. Although the evidence for a primary role of the symbiont in coral bleaching is overwhelming, there are, nonetheless, many ways in which the host can limit the level of damage sustained by the symbionts, and thus influence the response of the holobiont.

Paradoxically, light is both the driving force of photosynthesis and the cause of photodamage to the photosynthetic machinery, primarily PSII. Photodamaged PSII is rapidly and efficiently repaired through the replacement of

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Box 1. Coral bleaching and rates of adaptation

The prediction that reefs might disappear as a result of global warming is based largely on the assertion that rates of environmental change are too rapid for corals to adjust before they succumb to high temperature [3]. Two key assumptions underlie such predictions: corals have comparatively long generation times and gene flow is too great for local adaptation. How correct are these assumptions?

Many features of coral life histories, such as extended life spans, delayed maturation and colony fission, should result in long generation times [49]. In species with these traits, generation times are between 33 and 37 years [50]. However, other corals, in particular those species most susceptible to thermal stress such as many species of *Acropora* and *Pocillopora*, mature early, grow rapidly and suffer whole-colony mortality, as opposed to colony fission, following mechanical disturbances and thermal stress [51]. The life histories of these ecologically important and abundant species suggest an as-yet undefined and underappreciated capacity to adapt rapidly to changing environments.

Furthermore, the assumption that the scale of dispersal is too large to allow for adaptation to local environments might also be incorrect. The relationship between the supply of propagules and larval recruitment suggests that the majority of recruits are likely to be of local origin [52], and this is further supported by experiments which demonstrate that settlement is very rapid in some species [53]. Genetic studies also indicate that populations of many species are highly subdivided, which is indicative of restricted gene flow [54].

The major appeal of the 'adaptive bleaching hypothesis' [4] is that it offers a mechanism enabling adjustment to stress over the course of weeks, as opposed to generations. However, evolution by natural selection can occur rapidly in some situations [55], and therefore novel mechanisms, such as switching symbionts, might not be required for corals to adjust to climate change.

photodamaged proteins with newly synthesised proteins [15]. Thus, photoinhibition occurs only when the rate of photodamage to PSII exceeds the rate of its repair. A moderate increase in temperature accelerates photoinhibition, primarily through inhibition of the repair process [16,17]. However, the primary site of thermal stress is still unresolved, with various authors arguing it is PSII [8], the thylakoid membrane [18] and/or the Calvin cycle [19]. Recent experimental evidence has demonstrated that photodamage to PSII occurs in two steps: the primary damage occurs in the oxygen-evolving complex of PSII caused by ultraviolet radiation (UV) and strong blue light (and less effectively by other visible light), whereas secondary damage is caused by light absorbed by photosynthetic pigments in the reaction centre of PSII [20]. Consequently, any mechanism that reduces UV and visible light flux to symbiotic cells will potentially prevent photodamage and therefore might prevent photoinhibition-dependent coral bleaching (Figure 2).

Hypothetical mechanisms by which the host might reduce bleaching damage

The coral host has several potential ways to reduce UV and light flux to symbionts, including the production of fluorescent pigments (FP) and the acquisition of mycosporine-like amino acids (MAA). Similarly, the host has several antioxidant systems and stress enzymes to deal with oxygen stress originating in the animal cell. Differences among host species in their capacity to utilise these mechanisms might therefore determine differences among species in response to stress.

Fluorescent pigments

The coral host produces a range of FPs belonging to a single family of proteins, closely related to the green fluorescent proteins [21]. FPs are highly abundant on reefs; up to 97% of corals in shallow water on the Great Barrier Reef contain fluorescent pigments [22,23]. By absorbing, scattering and dissipating high-energy solar radiation via fluorescence, FPs reduce photoinhibition and the severity of bleaching damage to corals [23,24]. Many aspects of bleaching appear to be related to differences in the abundance of FPs. For example, bleaching-susceptible taxa, such as pocilloporids

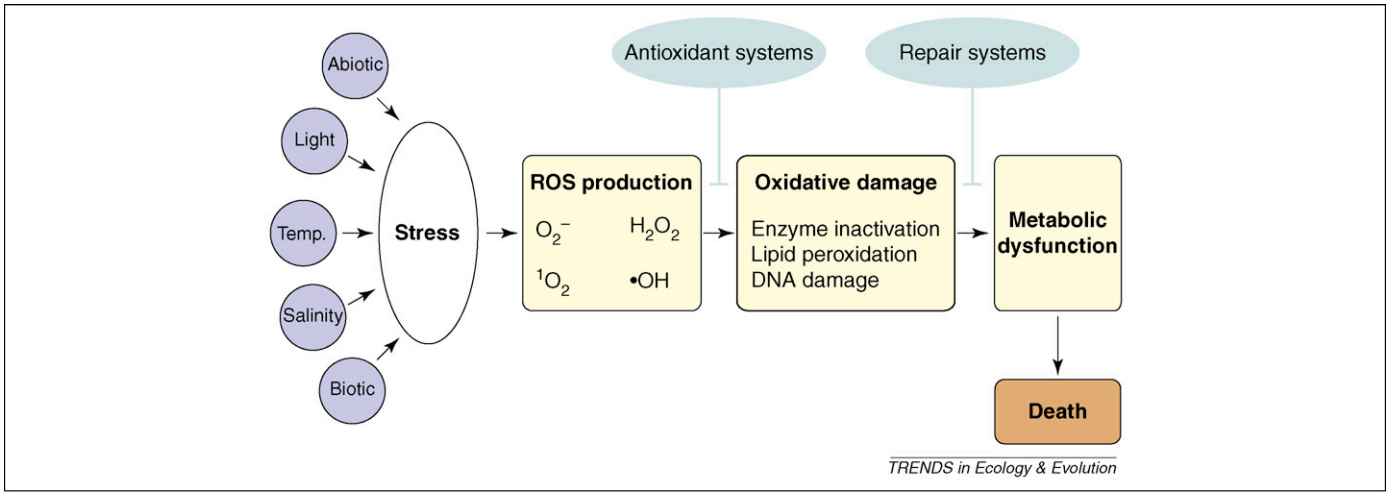

Figure 1. A generalised sequence of stress. Stress was originally defined as a nonspecific response independent of the cause of stress [46]. For example, corals bleach in response to high temperature, high light or high salinity and so forth, and therefore bleaching can be considered a stress response. In most stress responses, the production of reactive oxygen species (ROS) in cells is a common early event. Superoxide is the most commonly produced ROS. Hydrogen peroxide (H_2O_2), which is the only diffusive ROS molecule, is produced secondarily. In animal cells, ROS production is associated with the mitochondria; in photosynthetic organisms, it is also associated with the chloroplasts [27]. Potentially toxic ROS are removed by antioxidant systems, which include enzymatic antioxidants such as superoxide dismutase and catalase, ascorbic acid, carotenoids [27], FPs [47] and mycosporine glycine [28]. As long as these scavenging mechanisms are functional, ROS will not accumulate. Under severe stress, however, these antioxidant systems might not be able to destroy all ROS produced, in which case oxidative damage will occur, leading to metabolic dysfunction, cell destruction or mutation. In addition to antioxidant systems, there are repair systems to remove damaged molecules and to replace them with new ones. In this context, antioxidant systems function as the primary line of defence and the repair systems act as a secondary line of defence against oxidative stress. If these mechanisms cannot limit or suppress stress damage, living organisms will eventually die.

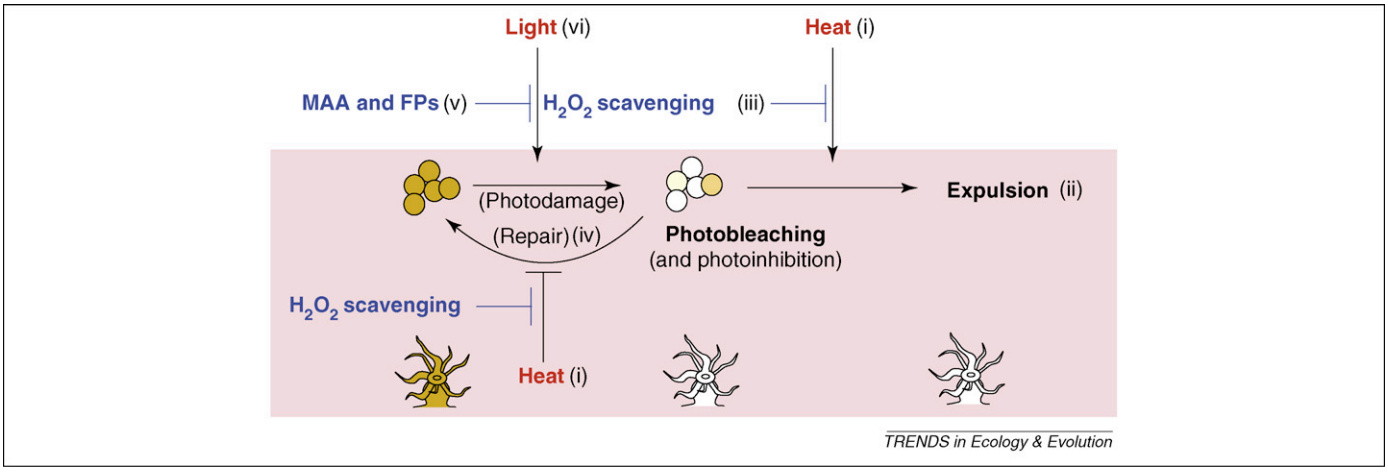

Figure 2. The role of the coral host in preventing bleaching caused by moderate heat stress. Moderate heat (i) accelerates the production of hydrogen peroxide (H_2O_2) in the chloroplasts of the algal symbionts either by damaging the thylakoid membrane or disrupting the Calvin cycle. H_2O_2 can spread from here to the host cell, where it activates a cellular cascade akin to an innate immune response, which results in expulsion of symbionts (ii) [38]. The potential to scavenge H_2O_2 (iii) varies among host species and, therefore, might influence difference in susceptibility to H_2O_2-dependent coral bleaching. In addition, H_2O_2 inhibits the repair of photodamage in PSII (iv), causing an acceleration of photoinhibition and photobleaching of pigments in symbionts [48]. Finally, by accumulating UV-absorbing compounds, such as MAAs and FPs, the host can intercept UV light (v), thereby limiting photodamage to PSII and preventing light-dependent coral bleaching (vi).

and acroporids, have relatively low densities of FPs, whereas poritids, faviids and other less-susceptible taxa have relatively high densities of FPs [22]. Similarly, highly fluorescent colonies suffer less partial mortality following bleaching than weakly fluorescent conspecifics [24]. Although the support for a role of FPs in reducing bleaching damage is compelling, a direct role of FPs in preventing bleaching, for example by providing photoprotection, remains to be tested experimentally. Furthermore, it is not known whether FPs can provide relief from heat stress alone.

Mycosporine-like amino acids

MAAs absorb UV and dissipate UV energy as heat without forming toxic intermediates [25]. MAAs are synthesised via the shikimic acid pathway and, because animals lack this pathway, symbionts are presumed to be the source of MAAs in corals [25]. Alternatively, they are acquired via heterotrophic feeding. Whatever the source of these compounds, MAAs are far more abundant in host tissues than in freshly isolated symbionts [25]. In addition, the diversity of MAAs found in holobionts is far greater than that found in *Symbiodinium* in isolation [25]. Whether this results from the host stimulating symbionts to produce a greater diversity of MAAs *in hospite* or because the host can modify MAAs translocated from the symbiont remains unknown [26]. However, it is clear that the host has a major influence on the complement and distribution of MAAs in the holobiont, thereby moderating the amount of UV that reaches symbiont cells and, thus, we hypothesise, influencing the amount of damage sustained by symbionts.

Antioxidant systems

A similarly complex interplay between host and symbiont is evident in response to oxidative stress. Potentially toxic ROS caused by stress are removed by antioxidant systems, which include enzymatic antioxidants such as superoxide dismutase (SOD) and catalase, ascorbic acid, carotenoids (reviewed in Ref. [27]) and mycosporine glycine [28]. Symbiotic hosts, such as the coral *Stylophora pistillata* and the anemone *Anemonia viridis*, have many different types of SOD, some of which are not found in non-symbiotic animals, and these act in combination with the antioxidant defences [28] of the symbiont to minimise oxidative damage [29]. In particular, more types of SOD are active in symbionts in isolation than when *in hospite*, indicating that protective mechanisms of the host limit oxidative damage sustained by the symbionts [29].

Heat-shock proteins

Stress proteins, such as the ubiquitous heat-shock proteins (HSP), are another way that organisms cope with stress, including temperature and high light. HSPs act as molecular chaperones, which maintain protein structure and cell function, particularly following stress. Many different HSPs are found in coral tissue and their activity influences the bleaching response. For example, high-light-acclimatised tissues of the coral *Goniastrea aspera* have higher concentrations of HSPs and these tissues do not bleach, unlike areas of the same colony that had not acclimatised to high light [30]. Importantly, neither the clade of symbiont nor algal antioxidant defences varied within colonies, highlighting the role of host tissue in the bleaching response of these colonies [30].

Change in diet in response to bleaching stress

Whereas much of the energy required for coral metabolism is derived from *Symbiodinium*, many corals are also effective carnivores. Species which can increase carnivory survive experimental bleaching better than species which cannot [31], presumably because the host can compensate for the loss of energy caused by reduced densities of symbionts by increased heterotrophic feeding. Alternatively, by increasing heterotrophic feeding, the host's demands on the symbiont might be reduced, allowing the symbiont to allocate more energy to its own antioxidant defences and potentially limiting damage to the algal cell. Nonetheless, changes in the transfer of photosynthetic products from the symbiont to the host in response to stress have yet to be explored.

Coral bleaching: a communication breakdown under stress

Coral bleaching presents biologists with many conundrums. For example, many corals bleach at 1–2 °C above historical mean summer maximum sea temperatures, although the bleaching threshold might be modified by ambient light levels. Whereas the actual temperature threshold depends upon location, at most locations mass bleaching thresholds are in the range of 29–32 °C, which is a surprisingly low temperature for tropical organisms to suffer high mortality. Indeed, it is only symbiotic organisms, including corals, sea anemones, zoanthids and sponges, that die during thermal anomalies [32]. Macroalgae do not bleach or die during coral mass bleaching events, and photosynthesis in free-living micro-algae is mostly unaffected below 35 °C [33]. Similarly, the critical thermal maximum for tropical fish is between 34.7 °C and 40.0 °C [34]. Does living together present unique problems, particularly when life becomes stressful?

Perhaps the bleaching phenomenon is best perceived as a breakdown in communication. Under normal conditions, symbiosis is presumably maintained by the release of signalling compounds from the symbiont [35]. The host-derived symbiosome membrane might mediate this signal transduction, although the role of membrane components in interpartner communication remains to be fully explored [36,37]. When the condition of the symbiont is compromised under stress, signalling is disrupted and the animal host defends itself, by expelling the symbiont or killing the animal cells that contain symbionts [38,39]. Alternatively, bleaching might be the result of a breakdown in the host's normal processes of population regulation of the symbiont at high temperature. Densities of *Symbiodinium* in coral cells are generally low and stable [40], despite the fact that the potential population growth rate of symbiont cells is much higher than host cells [41]. The host must, therefore, have mechanisms that regulate symbiont densities, for example 'host factors' that limit symbiont cell division [42] or are potentially toxic to *Symbiodinium* cells [43]. Other host regulatory mechanisms include limiting the supply of essential nutrients to symbionts [41] or digesting or expelling symbionts [43]. At non-stressful temperatures, the majority of symbionts released are morphologically degraded and have low photosynthetic efficiency [44]. By contrast, at stressful temperatures, the majority of released symbionts appear healthy [45] and photosynthetically active [44]. This suggests that under stress, the coral host's ability to discriminate between healthy and underperforming symbionts is diminished. Whatever the details of this relationship, living together clearly presents the organism with unique challenges, particularly when under stress. Bleaching is clearly an emergent property of the holobiont, the consequences of which cannot be fully resolved when considering either partner in isolation.

Conclusions

Corals have been in symbiosis with photosynthetic organisms for up to 200 million years, a selective pressure that has modified both host and symbiont [26,35]. Many features of the symbiosis, such as oxidative defences, MAA abundance and symbiont density per cell, are the result of complex molecular interactions between the partners. As outlined above, there is abundant evidence that both partners are involved in determining the organisms' response to stress and, therefore, understanding the threat climate change poses requires the organism to be treated as a whole. In particular, population ecological studies of corals are lacking and, consequently, the effects of temperature on even the most basic vital rates in corals, such as growth, mortality and fecundity, are largely unknown. To effectively address the issue of rates of adaptation in corals, and whether or not they will be exceeded by rates of environmental change, long-term demographic studies are required to detect temporal trends in life-history traits and to explore the sensitivity of population growth to these changes. The past ten years have seen great progress made by physiologists and molecular biologists toward understanding the mechanism of coral bleaching. Now it is time for ecologists to match this progress with studies designed to determine the consequences of coral bleaching for coral demography and, furthermore, to predict how climate-induced changes in coral demography will influence the future of coral reefs.

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References

1 Hughes, T.P. *et al.* (2003) Climate change, human impacts, and the resilience of coral reefs. *Science* 301, 929–933
2 Hoegh-Guldberg, O. (1999) Climate change, coral bleaching and the future of the world's coral reefs. *Mar. Freshw. Res.* 50, 839–866
3 Hoegh-Guldberg, O. *et al.* (2007) Coral reefs under rapid climate change and ocean acidification. *Science* 318, 1737–1742
4 Buddemeier, R.W. and Fautin, D.G. (1993) Coral bleaching as an adaptive mechanism—a testable hypothesis. *Bioscience* 43, 320–326
5 Sotka, E.E. and Thacker, R.W. (2005) Do some corals like it hot? *Trends Ecol. Evol.* 20, 59–62
6 Coles, S.L. and Brown, B.E. (2003) Coral bleaching—capacity for acclimatization and adaptation. *Adv. Mar. Biol.* 46, 183–223
7 Douglas, A.E. (2003) Coral bleaching—how and why? *Mar. Pollut. Bull.* 46, 385–392
8 Iglesias-Prieto, R. *et al.* (1992) Photosynthetic response to elevated temperature in the symbiotic dinoflagellate *Symbiodinium microadriaticum* in culture. *Proc. Natl. Acad. Sci. U. S. A.* 89, 10302–10305
9 Warner, M.E. *et al.* (1996) The effects of elevated temperature on the photosynthetic efficiency of zooxanthellae *in hospite* from four different species of reef coral: a novel approach. *Plant Cell Environ.* 19, 291–299
10 Fitt, W.K. and Warner, M.E. (1995) Bleaching patterns of four species of Caribbean reef corals. *Biol. Bull.* 189, 298–307
11 Loya, Y. *et al.* (2001) Coral bleaching: the winners and the losers. *Ecol. Lett.* 4, 122–131
12 McClanahan, T.R. *et al.* (2004) Comparing bleaching and mortality responses of hard corals between southern Kenya and the Great Barrier Reef, Australia. *Mar. Pollut. Bull.* 48, 327–335
13 LaJeunesse, T.C. *et al.* (2003) Low symbiont diversity in southern Great Barrier Reef corals, relative to those of the Caribbean. *Limnol. Oceanogr.* 48, 2046–2054
14 Ainsworth, T.D. *et al.* (2008) Early cellular changes are indicators of pre-bleaching thermal stress in the coral host. *J. Exp. Mar. Biol. Ecol.* 364, 63–71
15 Aro, E.M. *et al.* (2005) Dynamics of photosystem II: a proteomic approach to thylakoid protein complexes. *J. Exp. Bot.* 56, 347–356

16 Takahashi, S. *et al.* (2004) Repair machinery of symbiotic photosynthesis as the primary target of heat stress for reef-building corals. *Plant Cell Physiol.* 45, 251–255
17 Warner, M.E. *et al.* (1999) Damage to photosystem II in symbiotic dinoflagellates: a determinant of coral bleaching. *Proc. Natl. Acad. Sci. U. S. A.* 96, 8007–8012
18 Tchernov, D. *et al.* (2004) Membrane lipids of symbiotic algae are diagnostic of sensitivity to thermal bleaching in corals. *Proc. Natl. Acad. Sci. U. S. A.* 101, 13531–13535
19 Jones, R.J. *et al.* (1998) Temperature induced bleaching of corals begins with impairment of dark metabolism in zooxanthellae. *Plant Cell Environ.* 21, 1219–1230
20 Nishiyama, Y. *et al.* (2006) A new paradigm for the action of reactive oxygen species in the photoinhibition of photosystem II. *Biochim. Biophys. Acta* 1757, 742–749
21 Matz, M.V. *et al.* (1999) Fluorescent proteins from nonbioluminescent Anthozoa species. *Nat. Biotechnol.* 17, 969–973
22 Salih, A. *et al.* (1998) Photoprotection of symbiotic dinoflagellates by fluorescent pigments in reef corals. In *Australian Coral Reef Society 75th Anniversary Conference* (Greenwood, J.G. and Hall, N.J., eds), pp. 217–230, The University of Queensland
23 Salih, A. *et al.* (2000) Fluorescent pigments in corals are photoprotective. *Nature* 408, 850–853
24 Salih, A. *et al.* (2006) The role of host-based color and fluorescent pigments in photoprotection and in reducing bleaching stress in corals. *Proc. 10th Int. Coral Reef Symp.* 746–756
25 Shick, J.M. and Dunlap, W.C. (2002) Mycosporine-like amino acids and related gadusols: biosynthesis, accumulation, and UV-protective functions in aquatic organisms. *Annu. Rev. Physiol.* 64, 223–262
26 Furla, P. *et al.* (2005) The symbiotic anthozoan: a physiological chimera between alga and animal. *Integr. Comp. Biol.* 45, 595–604
27 Lesser, M.P. (2006) Oxidative stress in marine environments: biochemistry and physiological ecology. *Annu. Rev. Physiol.* 68, 253–278
28 Yakovleva, I. *et al.* (2004) Differential susceptibility to oxidative stress of two scleractinian corals: antioxidant functioning of mycosporine-glycine. *Comp. Biochem. Physiol. B Biochem. Mol. Biol.* 139, 721–730
29 Richier, S. *et al.* (2005) Symbiosis-induced adaptation to oxidative stress. *J. Exp. Biol.* 208, 277–285
30 Brown, B.E. *et al.* (2002) Exploring the basis of thermotolerance in the reef coral *Goniastrea aspera*. *Mar. Ecol. Prog. Ser.* 242, 119–129
31 Grottoli, A.G. *et al.* (2006) Heterotrophic plasticity and resilience in bleached corals. *Nature* 440, 1186–1189
32 McClanahan, T. *et al.* (2009) Consequences of coral bleaching for sessile reef organisms. In *Ecological Studies: Coral Bleaching: Patterns, Processes, Causes and Consequences* (van Oppen, M.J.H. and Lough, J.M., eds), pp. 121–138, Springer-Verlag
33 Berry, J. and Bjorkman, O. (1980) Photosynthetic response and adaptation to temperature in higher plants. *Plant Physiol.* 31, 491–543
34 Mora, C. and Ospina, A.F. (2001) Tolerance to high temperatures and potential impact of sea warming on reef fishes of Gorgona Island (tropical eastern Pacific). *Mar. Biol. (Berl.)* 139, 765–769
35 Yellowlees, D. *et al.* (2008) Metabolic interactions between algal symbionts and invertebrate hosts. *Plant Cell Environ.* 31, 679–694
36 Schwarz, J.A. and Weis, V.M. (2003) Localization of a symbiosis-related protein, sym32, in the *Anthopleura elegantissima-Symbiodinium muscatinei* association. *Biol. Bull.* 205, 339–350
37 Chen, M.C. *et al.* (2005) ApRab11, a cnidarian homologue of the recycling regulatory protein Rab11, is involved in the establishment and maintenance of the *Aiptasia-Symbiodinium* endosymbiosis. *Biochem. Biophys. Res. Commun.* 338, 1607–1616
38 Dunn, S.R. *et al.* (2007) Apoptosis and autophagy as mechanisms of dinoflagellate symbiont release during cnidarian bleaching: every which way you lose. *Proc. Biol. Sci.* 274, 3079–3085
39 Perez, S. and Weis, V. (2006) Nitric oxide and cnidarian bleaching: an eviction notice mediates breakdown of a symbiosis. *J. Exp. Biol.* 209, 2804–2810
40 Muscatine, L. *et al.* (1998) Cell specific density of symbiotic dinoflagellates in tropical anthozoans. *Coral Reefs* 17, 329–337
41 Falkowski, P.G. *et al.* (1993) Population-control in symbiotic corals. *Bioscience* 43, 606–611
42 Gates, R.D. *et al.* (1995) Free amino-acids exhibit anthozoan host factor activity—they induce the release of photosynthate from symbiotic dinoflagellates in-vitro. *Proc. Natl. Acad. Sci. U. S. A.* 92, 7430–7434
43 Dunn, S.R. *et al.* (2002) Programmed cell death and cell necrosis activity during hyperthermic stress-induced bleaching of the symbiotic sea anemone *Aiptasia* sp. *J. Exp. Mar. Biol. Ecol.* 272, 29–53
44 Bhagooli, R. and Hidaka, M. (2004) Release of zooxanthellae with intact photosynthetic activity by the coral *Galaxea fascicularis* in response to high temperature stress. *Mar. Biol. (Berl.)* 145, 329–337
45 Reimer, A.A. (1971) Observations on the relationships between several species of tropical zoanthids (Zoanthidea, coelenterata) and their zooxanthellae. *J. Exp. Mar. Biol. Ecol.* 7, 207–214
46 Selye, H. (1946) The general adaptation syndrome and the diseases of adaptation. *J. Clin. Endocrinol.* 6, 117–230
47 Bou-Abdallah, F. *et al.* (2006) Quenching of superoxide radicals by green fluorescent protein. *Biochim. Biophys. Acta* 1760, 1690–1695
48 Takahashi, S. and Murata, N. (2005) Interruption of the Calvin cycle inhibits the repair of photosystem II from photodamage. *Biochim. Biophys. Acta* 1708, 352–361
49 Hughes, T.P. *et al.* (1992) The evolutionary ecology of corals. *Trends Ecol. Evol.* 7, 292–295
50 Babcock, R.C. (1991) Comparative demography of three species of scleractinian corals using age-dependent and size-dependent classifications. *Ecol. Monogr.* 61, 225–244
51 Baird, A.H. and Marshall, P.A. (2002) Mortality, growth and reproduction in scleractinian corals following bleaching on the Great Barrier Reef. *Mar. Ecol. Prog. Ser.* 237, 133–141
52 Hughes, T.P. *et al.* (2000) Supply-side ecology works both ways: the link between benthic adults, fecundity, and larval recruits. *Ecology* 81, 2241–2249
53 Miller, K. and Mundy, C. (2003) Rapid settlement in broadcast spawning corals: implications for larval dispersal. *Coral Reefs* 22, 99–106
54 Ayre, D.J. and Hughes, T.P. (2004) Climate change, genotypic diversity and gene flow in reef-building corals. *Ecol. Lett.* 7, 273–278
55 Carroll, S.P. *et al.* (2007) Evolution on ecological time-scales. *Funct. Ecol.* 21, 387–393