Chapter 5 General Discussion

The results presented in this work indicate that the thickness of the tissue layer (TTL) of massive *Porites* corals is a dynamic, constantly changing quantity. TTL has been shown here to vary in space and time and according to changing environmental conditions. Changes in TTL reflect both monthly (and longer term) growth cycles of the colony and the responses of the coral to its changed circumstances. Gradients in TTL between colonies located in environmental stress gradients can be used as a general assessment tool measuring the severity of impacts on coral community health. TTL changes are relative, however, and the scale of change varies greatly between individuals and episodes. Species of massive *Porites* is not a determinant of TTL; average values of TTL and the general patterns of variation are consistent between colonies of *P. australiensis*, *P. lobata* and *P. lutea*. Species-specific attributes may, however, play a role in determining the response of a colony to an environmental stress.

5.1 The thickness of the tissue layer of Porites varies in space and time.

Spatial variation in TTL is principally due to local variations in the quality of the coral's habitat, rather than to broader geographical factors (Chapter 2). Neither distance offshore, nor latitude appeared to affect the average thickness of the tissue layer in a survey of over 200 massive *Porites* colonies, controlled for species, depth of occurrence, size and shape of colony. These colonies

were sampled within a 3 month period, at sites widely-dispersed along the length of the Great Barrier Reef, thereby providing a snapshot of TTL variation on the GBR. Within-locality variation in environmental quality, such as found in inshore and offshore sides of a continental island or between the exposed and sheltered sides of a coral reef appear to have much more influence on the thickness of the tissue layer. The most ubiquitous influence on tissue thickness in terms of between-habitat variation appears to be light. As a general rule, the shallow-water habitats and those exposed to clearer waters seem to encourage development of thicker tissue layers than deep water or more turbid environments. Data presented elsewhere (e.g. Lough et al. 1999) indicate that calcification and linear extension rates are affected by latitude (attributed mainly to average SST: colonies in the highest latitudes of the species range are reported to cease skeletal growth during winter). The potential effects of latitudinal variations in insolation may be masked by habitat-based variations in conditions, acclimation to the local conditions, or may even be somewhat dependent on the local population of zooxanthellae (*sensu* Ulstrup & van Oppen, 2003). The lack of TTL correlation with latitude thus increases the potential usefulness of TTL as a monitoring tool, since putative TTL variation is linked only with departures from local environmental optima, rather than a strong, over-riding environmental parameter.

Availability of terrigenous nutrients, likewise, does not seem to be a strong influence, since offshore and oceanic habitats present average tissue thickness

levels equivalent to inshore and mid-shelf habitats. This finding is somewhat at odds with the expectation that cross-shelf gradients in water quality on the GBR would benefit mid-shelf corals more than inshore or offshore colonies (cf. Risk & Sammarco, 1991, Barnes & Lough, 1993).

Temporal variation in TTL is composed of monthly, yearly and multi-annual components. The shortest period variations are due to skeletal growth of the coral and a requirement for episodic tissue uplift. The longer cycles are due to a) seasonal and between-year variation in environmental parameters such as seawater temperature and insolation; and b) anomalous environmental variation due to pollution, *el niño/la niña* episodes, or large-scale climatic variations.

TTL varies within a month because of linear extension of the coral skeleton at the outer margin of the colony. During the monthly growth cycle, the thickness of the tissue layer increases incrementally with daily skeletal extension at the top of each calyx (S.Rotman, 2004 pers.com.). This increase is abruptly interrupted just after the full moon, when the tissue layer is reduced by approximately 1mm (Chapter 2). The hiatus in tissue increase coincides with the formation of a new dissepiment and the evacuation of tissue from the lower portion of the tissue layer.

Corals in the central GBR experience strongly seasonal environmental conditions. Seasonal cycles in linear extension and calcification are primarily attributed to temperature-dependence of the rate of the precipitation process (Reynaud et al. 2003) in shallow-water corals, since they are light-saturated for the entire year. Seasonal variations in the monthly mean TTL for corals in the central GBR coincide with these cycles. They indicate the response of the coral to annual cycles in SST, insolation, environmental conditions and planktonic food supply. It was not possible to separate the effects of all of these parameters in this study (Chapter 2, 3). Removal of a large proportion of the incident light from the corals, however, largely suppressed seasonal variation in TTL. Skeletal seasonality was retained in these colonies (Chapter 4), suggesting that the influence of temperature (and thus skeletal extension) on cycles of TTL is minor.

The cause of seasonal TTL variation does not, therefore, lie entirely with seasonal cycles in skeletal extension. It was not possible in this study to separate the influences of environmental parameters from endogenous cycles (such as reproduction, which coincides with the period of maximum tissue thickness). The monthly mean thickness of the tissue layer may be interpreted as an indication of the level of energy reserves held by the coral, allowing for the estimation of the relative performance of the colony in a given month. An important next step to discover the generality of seasonal cycles in TTL is to repeat the long term study of Chapter 2 and the light-deprivation experiment of

Chapter 3 in a locality which does not experience pronounced seasonal changes in environmental conditions. Only in this way can endogenous rhythms be identified and assessed.

Scales of variation in TTL greater than a year were detectable in the experiments described here (Chapter 2). Between year variation in skeletal growth and density is commonly recorded in skeletal proxy record research. It is usually attributed to differences in environmental conditions between years. The differences in TTL between years is likely also to be a product of inter-annual variation in conditions, although the results of Chapter 3 and 4 suggest that the response of the coral to changes in conditions (evidenced by the thickness of the tissue layer), rather than simple chemistry, may drive the skeletal patterns.

5.2 Tissue thickness and density bands

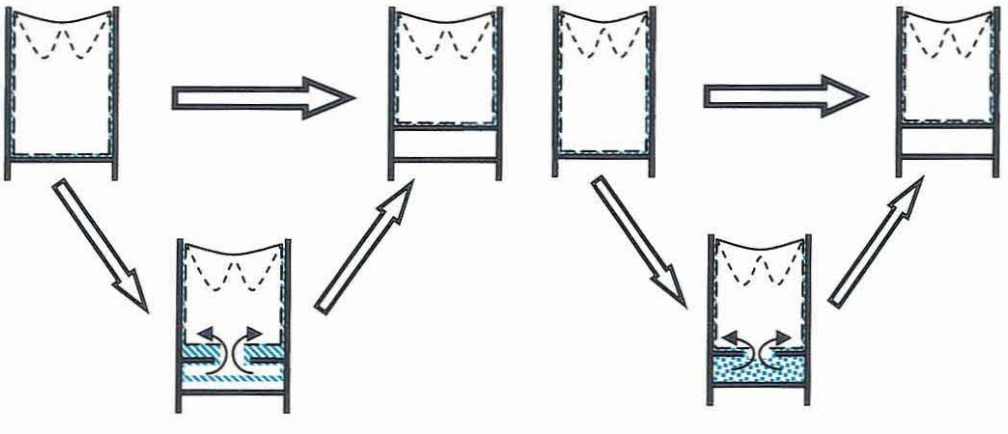
Faster growing corals form 12-13 dissepiments per year, corresponding to one every lunar month. Most colonies, however, form fewer dissepiments than this. It is in these colonies that we may see the strongest evidence for the mechanisms controlling density band formation. Taylor et al.'s (1993, 1995) models suggest that three main factors control the formation of density bands in massive *Porites*: linear extension rate, calcification rate and tissue thickness.

True (1995) had already shown that a gradient of tissue thickness exists between shallow and deep water colonies of *Porites*. In Chapter 3, I showed that excision of a large portion of a colony's energy intake by shading caused a reduction in the thickness of the tissue layer and a slowing-down of linear extension. Light is also therefore an important factor, probably determining in large part the energy level of the coral tissue. Moreover, the convergent decline between the thickness of the tissue layer and the rate of linear extension suggests further that one is dependent on the other.

The tissue layer may reflect a certain degree of plasticity in the vertical architecture of the polyp, depending on the time of year and conditions. During reproduction, bulging gametogenic mesenteries can be observed deep within the subgastric tissue layer, in regions which are normally a tangled mass of coelenteric tubules. The season of reproduction on the Great Barrier Reef has, however, been linked to the period of formation of the less dense phase of skeletal density bands (Gagan et al. 1996**). Whether this represents a change in the architecture (to allow extra room for enlarged gametocytes) or change of utility of the tissue during reproduction is not known. Certainly, the continual mobilisation of tissue at the lower margin in response to its episodic uplift allows for this possibility. The explanation for this may lie in the mechanism of "tissue uplift" accompanying dissepiment formation.

5.3 Possible mechanisms for tissue uplift

Two main possible mechanisms exist for tissue uplift in *Porites*. Both of these possibilities involve the evacuation of tissue from the lower part of the tissue layer, below that part which would be sealed off by the formation of a new dissepiment. The first mechanism requires the involution of the tissue and physical withdrawal from the evacuation zone. The second involves catabolism of the somatic tissue of the lower region and the translocation of the catabolites into the remaining tissue above the dissepiment. It is likely that the two are not mutually exclusive; i.e. the actual process involves a combination of the two mechanisms.



a. b.

Figure 5.1 Uplift scenarios discussed in text. During the "uplift" event, a new dissepiment is created some distance above the floor of the corallite, sealing the polyp off from the older skeleton; tissue is evacuated from the zone below the new dissepiment. The mechanism for this evacuation is likely to be a combination of a) Involution scenario, in which the tissue is physically withdrawn from the evacuation zone; b) Catabolism scenario, in which tissue in the evacuation zone is digested before being transported into the remaining tissue layer.

5.3.1 Uplift process 1 – tissue involution

This scenario (Figure 5.1a) suggests involution of the lower portion of the tissue layer, possibly accompanied by the compression of the tissue in the polyp above the level of the new dissepiment to accommodate the extra tissue mass in the smaller space. Since the lower part of the tissue layer is a complex lattice of skeleton and tissue, and is highly interconnected to adjacent polyps, this process would require abscission and the complex involution of many canaliculae.

The mechanism of involution is likely to be similar to the "conveyor belt" model proposed by Barnes (pers. com.) for the oral/calicoblastic ectoderm around skeletal spinules at the oral surface of the polyp. Additionally, some amount of tissue compression is required to accommodate the extra 20-30% of tissue mass now occupying the thecae after the new dissepiment is formed. The simplest involution scenario could be modeled similarly to the process of tentacle retraction, albeit in a markedly more complicated manner of withdrawal. In this case, it might be that fluid is withdrawn from the coelenteron, creating a negative pressure gradient – in effect sucking the lower tissue back into the upper region. More interesting would be a scenario in which each tubule is involuted, turning in upon itself to migrate into the upper tissue mass.

5.3.2 Uplift process 2 – catabolism

This scenario involves the catablism of the bulk of the tissue in the evacuation zone and the transport of the disassembled structural components to storage areas in the upper tissue mass. This scenario, while still requiring the abscission of communicating canaliculae between polyps, does not require the complicated three-dimensional maneuvering of the first scenario. The catabolism of structural tissue releases quantities of lipids and proteins, which would be readily sequestered in the somatic tissue remaining to the polyp.

The second scenario appears to be more likely, given the apparent interchangeability of somatic tissue and lipid in stressful conditions (described in Chapter 3). In all likelihood, the actual mechanism of uplift contains elements of both scenarios: some amount of tissue catabolism, followed by the withdrawal of the remaining calicoblastic tissue. It might even be that the catabolism of somatic tissue is the fuel for uplift and dissepiment formation. Cessation of tissue uplift which has been reported in corals exposed to mine effluent supports this hypothesis. These corals did not form dissepiments when the uplift (about 0.4mm) would have caused the thickness of the tissue layer to diminish to less than 1.8mm, which is approximately the depth of the calyx. Uplift only occurred in those months where initial tissue thickness was great enough to leave >1.8mm after the uplift event (S. Rotman, pers. com.). Certainly, the availability of energy for renewed linear extension would be enhanced by the catabolism of approximately 15-25% of the tissue mass of a

polyp. Vago et al. (1997) showed that *Porites* from the central GBR extended their skeletons for only about 16 days in a month, commencing growth a few days subsequent to the full moon; this pulse could well be fuelled by energy liberated from sacrificial tissue during an uplift event. The bleaching experiments of Chapter 3 suggest that levels somatic tissue and lipids are related, and have a strong bearing on the vulnerability of corals to stress events.

The most readily-available energy source to the polyps is almost certainly lipids (Anthony and Fabricius, 2000), whether supplied by the zooxanthellae of by catabolism of somatic tissue reserves. Several studies have suggested that the supply of photosynthates to shallow water corals vastly exceeds their ability to use or store it, causing them to purge the excess in mucus secretions (e.g. Crossland, 1987; Edmunds & Davies, 1986). Personal observations suggest that mucus tunic production in *Porites* is concentrated around the time of the full moon (cf. Coffroth, 1988, 1990) – possibly reflecting a surplus of material resulting from the catabolism of tissue. Testing of the mechanism of uplift would require the continuous monitoring of both tissue thickness and lipid and protein levels over the course of several uplift events.

5.3 Tissue thickness and stress in *Porites*

In Chapter 3 I examined several sources of stress in massive *Porites*. A unifying factor amongst the stress sources was that they all – in some way – caused a decrease in the amount of energy available to the coral. Whether this

decrease was direct (such as the need to increase expenditure to combat oxidative stress or sedimentation) or indirect (the reduction of zooxanthella-derived nutrition because of shading), it appears that the severity of the stress is reflected in a reduction in the tissue mass and/or the lipid content of the tissue.

Both lipid content and tissue thickness appear to have threshold or minimum values. In the stress studies described in Chapter 3, a bleaching response only occurred when *both* tissue and lipid approached their minima. The story is not quite that simple, however. Examination of the relationships between tissue thickness and lipid concentration during both artificial and natural bleaching events presented in Chapter 3 (Figs. 3.10, 3.12) indicates that the lipid threshold appears to precede the tissue threshold in terms of the bleaching response. That is, bleaching appears to occur when the lipid levels of the tissue layer decrease to a certain point (approximately 6-8mg.cm^{-3}), without regard for the thickness of the tissue layer. Colonies which had bleached, whether they had relatively thick or thin tissue layers, exhibited similarly reduced concentrations of tissue lipids. Colonies with initially thicker tissue, however, contained comparatively less lipid. During both the natural and artificial bleaching events, the tissues of colonies with comparatively thicker tissue layers contained lipid levels not significantly different from bleaching concentrations. These colonies uniformly lost tissue thickness, whereas colonies from the other group (thin tissue, high lipid concentrations) lost only

lipids. This result is somewhat surprising, since it suggests that somatic tissue and lipids are more-or-less interchangeable as an energy reserve for the coral.

Adopting the "catabolism model" of tissue uplift described above provides a mechanism for this interchangeability. It also provides a context to understand why colonies can bleach while still having an apparent reserve of somatic tissue (Figure 3.9). If catabolism of somatic tissue can occur only in the context of tissue uplift (cued by the full moon), then corals under stress have a single opportunity each month to "top up" the readily available energy reserve provided by the lipids sequestered in the tissue layer. If the level of expenditure is severe enough to deplete that combined supply, then the coral may take steps to dramatically reduce energy expenditure by constraining the major energy-depleting functions – either by reducing or halting linear skeletal growth (see Chapter 4), or by expelling potentially costly zooxanthellae (the additional respiratory expenditure prioritized against potential energetic gains). This last action might be interpreted as a final act of desperation, since it also removes what is potentially the greatest source of ongoing energy replacement for the coral. It is probably, however, that this action is accompanied by a virtual shutdown of metabolism – in effect, waiting for the next opportunity to catabolize the somatic energy reserve.

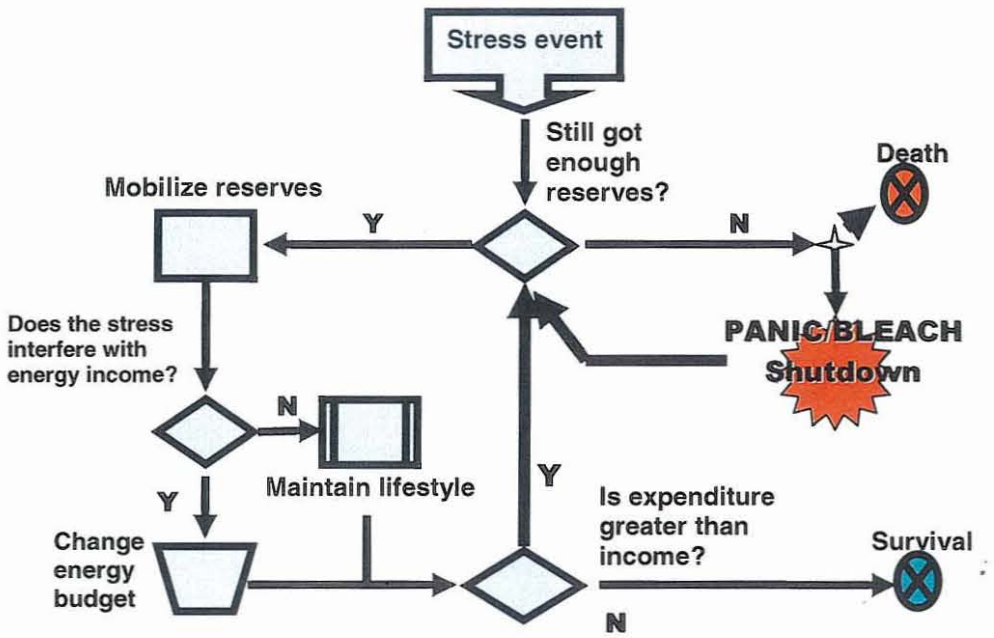


Figure 5.2 Hypothetical stress-management protocol for corals. The onset of stress invokes a status check, to determine the ability of the coral to meet the impact. The first option for corals is to mobilize available energy reserves, whether in the form of lipids, or by the catabolism of somatic tissue. As well, the coral determines whether its ability to acquire *more* energy is affected by the stress event (e.g. shading or damage to zooxanthellae). If energy acquisition is severely compromised, then some sort of adjustment is made, such as curtailment of skeletogenesis. Repeated application of this protocol allows the corals to deal with any sort of sub-lethal stress event. Failure at the first checkpoint, however, leaves the coral with a one-time option of bleaching, which involves a shutdown of all major non-metabolic functions, such as growth, reproduction and upkeep of symbionts (hence, zooxanthellae are expelled). If this process frees up enough energy, then the coral may survive the stress event. Corals such as *Porites* have an ability to repeatedly mobilize energy reserves during monthly tissue uplift episodes, and so exhibit greater durability than corals that cannot.

The minimum tissue thickness of live *Porites* corals in this study was 2.1mm (Chapter 3). Although Rotman (2004, pers. com.) has observed unbleached colonies suffering from sedimentation stress with as little as 1.8mm of tissue – far below the minimum depth of tissue reported here or in other works (e.g. Barnes & Lough, 1997; 2-3mm) – which did not form a dissepiment until tissue thickness increased beyond 2.2mm, these cases must be viewed as exceptional. Even under the stress from a mine sediment plume, these

colonies did not bleach or die (compare this with Barnes & Lough's 1997 findings based on corals in a similar situation in southern PNG in which they found a trend of declining tissue thickness approaching the source of the plume, culminating in community mortality). Barnes & Lough (1997) noted that the colonies in the mine plume with TTL below 2mm died. Pre-stress linear extension rates for Rotman's colonies approached 20mm per annum (compare with various values for *Porites* spp. in Harriott, 1999). At the time of writing, no information is available about any changes in linear growth rate in Rotman's corals. Such rapid skeletogenesis suggests that normal energy uptake of the Rotman's colonies was extremely high. The energy supply of the colonies post impact must, therefore, have been sufficient to maintain skeletal growth even under stressful conditions, since Rotman subsequently reported that they recommenced dissepiment formation once the tissue thickness increased past 2.2mm. A corollary to this observation is that there must be some architectural or structural constraints on the formation of dissepiments such that they cannot form within ~2mm of the upper surface of the corallite. Apart from the exceptional cases reported by Rotman, it seems likely that after experiencing prolonged or extreme stress, if the colony cannot mobilize enough energy to combat ongoing stress (by shutdown and bleaching behaviour), it will die.

5.5 Extraction of proxy records from coral skeletons

5.5.1 Corals record stress events as isotope anomalies in their skeletons

In the study presented in Chapter 4, I described the use of varying ratios of carbon and oxygen isotopes within the aragonite skeleton as proxy records to reconstruct temperature/ salinity and "productivity" history of massive *Porites*. These techniques have been developed in geochemical labs over the last several decades to a high level of sophistication, yet they had never been tested against histories of known biological variation until now.

This study (Chapter 4) was the first in which it was demonstrated that corals can record chronic sub-lethal stress as changes in the isotopic composition of their skeletons. Moreover, the results presented in this study were the first to match physiological assessment of coral condition to skeletal and isotopic anomalies.

The experiment which was the cause of the stress analysed in this study was described in Chapter 3. In it, I changed a single parameter (light) in the environment of a group of large *Porites* colonies in a field experiment. Reduction in incident PAR caused a profound reduction in the average thickness of the tissue layer of the study colonies relative to unshaded controls (Chapter 3). It also triggered changes in both the rate of growth and isotopic composition of the corals' skeletons during the shading period (Chapter 4). These changes highlight a period of skeletogenesis which contrasts strongly

with the period of skeletal deposition corresponding to the wide-scale coral bleaching event of 1998. During the acute stress period of the mass bleaching event, growth of the previously-shaded corals was almost indistinguishable from non-stressed periods. On the contrary, the growth of the unshaded colony appeared to cease briefly during the event, leaving an isotopic signature similar to that described by Suzuki et al. (2003).

Critical examination of certain aspects of the reconstruction of past environments using coral-based isotope proxy techniques revealed several strengths and weaknesses of the techniques.

5.5.2 There are differences between the colonies and species

Superficially, the sensitivity of tissue layer thickness to changes in environmental conditions provides a promise of the ability to discriminate between chronic and acute stress events in the life of a coral. In practice, however, the finding is more equivocal. There was little agreement in the fine detail of the isotopic records of the two shaded colonies analysed in this study, in either the $\delta\,^{18}O$ or the $\delta^{13}C$ traces. Neither did the two shaded colonies agree substantially with the unshaded control colony's traces, except during the pre-experimental period. There may be several explanations for this: 1) there are significant between colony differences, which will render comparisons of records from individual colonies across locations meaningless, since there is no way to discriminate environmental from individual variation;

2) there are significant differences between the various species of massive *Porites* in the way they deal with stress and changing environments, with similar restrictions (i.e. colonies which have not been identified to species level cannot be directly compared); 3) exposure to the chronic stress of the shading experiment somehow inured the shade colonies to the energy crisis engendered by the bleaching event, resulting in a minimal reaction to the bleaching event (even though they did bleach).

This last possibility is quite intriguing: it is supported by the results of the lipid analyses described in Chapter 3, in which the lipid content of the tissue layer of the previously-shaded group was much higher than the unshaded control group (this difference disappeared two years after the bleaching event). In part it implies that environmental impacts occurring at short intervals will not be faithfully recorded in the skeleton. Isotopic signatures such as described by Suzuki et al. (2003) may only be distinguishable if they are separated by sufficient intervals of recovery time. Skeletal proxy techniques will therefore underestimate the frequency of environmental impacts. Such a failure means that reliable fine-scale hindcasting of environmental variation is not possible, or at least severely compromised. It also means, however, that corals may be more resilient than previously thought, and able to invoke a variety of strategies for survival under stressful conditions.

These findings shed some light on why it has so far been difficult to reconcile proxy records from different studies (e.g. Lough 2004). Previous studies (e.g. Grottoli, 2000, Linsley et al. 1999) have indicated that between-colony variation is marked. These studies have also shown that skeletal isotope records vary between colonies located in different depths of water and in different habitats. If corals lay down their skeleton differently according to species, individual resilience (perhaps partly dependent on their algal symbionts: e.g. Little et al. 2004) or environmental history, then little wonder that consonance between proxy-derived environmental histories is so rare.

5.5.3 Analysts MUST study more than one core

The evident power of fine-scale isotopic techniques to detect isolated stress events which would normally escape proxy techniques based solely on densitometry means that the discussion above is not so much "why use them?" as "how do we improve them?". The obvious first solution to much of the question is to use multiple records from each location, selecting only conspecific colonies. The records from each colony could then be reconciled against one another using standard techniques already in use in terrestrial paleo-climatology and dendrochronology (such as "wiggle matching" of isotopic records to match established chronologies using Bayesian maximum likelihood strategies: e.g. Blaauw et al 2004). The results presented in this study suggest that the use of single proxy records to reconstruct an environmental history across multiple locations is doomed to failure.

5.5.5 Identification to species level is vital for comparative studies

Perhaps more important than replication of samples, is identification of the source of the sample used for proxy record reconstruction. Previous studies (e.g. Barnes et al. 1999) have indicated that little or no difference exists between the five species of massive *Porites* commonly used in densitometry-based proxy records on the GBR (viz. *P. lobata, P. lutea, P.australiensis, P. mayeri, P. solida*). In contrast, this study has found that strong differences in the isotopic composition of coral skeletons laid down in periods of stress from corals of different species (cores from the two conspecific corals in this study agree more substantially with each other than with that from the out-species). It may be that densitometric techniques are not sensitive enough to detect such differences (which matters little, since the primary goal of such a study is to measure annual calcification increments and thereby derive SST variations). The continued use of more sensitive techniques, however, should be based on the robust identification of the source colony to species level. This caveat almost certainly applies also to the non-Poritid species used in similar studies in the Caribbean and elsewhere. Certainly, comparison between disparate proxy records should be undertaken only with great caution if the source species for each record is not positively identified.

5.6 Evaluation of TTL as a monitoring tool:

The results presented in this study suggest that variation in the thickness of the tissue layer of massive *Porites* corals is a sensitive indicator of the health or resilience of the colonies. Adaptation of this sensitivity into a simple reactive monitoring tool is, however, not a simple matter.

One of the primary criteria to identify a potential general monitoring tool is ease of use. Ideally, a monitoring protocol should be able to deliver a simple, easy-to-interpret index representing the status of the community under study. Unfortunatel massive *Porites* falls short of this ideal in several ways.

5.6.1 Inter-locality, inter-colony, intra-annual and intra-monthly variability approximates stress-induced variability

While average TTL of colonies varies consistently over the course of a year (Chapter 2) and under stress (Chapter 3), individual colonies present quite different profiles of TTL variation (Chapter 2, 3). TTL varies between colonies and between localities in ways that are difficult to predict. Different localities support colonies of vastly different TTL without apparent differences in environmental conditions. Formation of new dissepiments, while predictable, constrains sampling to a brief period in each month (although Rotman, (2004, pers. com.) reports that TTL increases in a linear fashion prior to uplift, suggesting that it may be possible to derive correction factors). Moreover, seasonal variations are of the same order of magnitude as variations due to the impact of some stress factor. Added to this is evidence that

previous stress events in the life of the coral can influence both skeletal and tissue parameters (Chapter 3, 4).

5.6.2 Previous history can influence the sensitivity of the measure

Corals which have previously been influenced by some energy crisis appear to adopt slightly different energy use strategies than corals without incipient stress, perhaps decreasing their sensitivity to new stress impacts. Therefore, in order to discriminate the impact of a stress agent, the observer must know quite a lot about the colonies to be sampled. This criterion suggests that conversion of TTL variation into a tool for monitoring community health on an *ad hoc* basis is more complex than is desirable. Each measurement must be put into the context of locality, habitat (within locality), depth of water, time of year, time of month, size of colony and recent history of the colony to become meaningful. A measurement of the thickness of the tissue layer is thus very much a relative measure of the coral's "health".

5.6.3 TTL offers a relative measure of environmental stress gradients

While sampling requirements and colony variability may be too restrictive for TTL to be a generic monitoring tool, it offers great potential as an indicator of environmental impacts. Gradients in TTL towards point sources of environmental stress, such as mine sites (discussed in Chapter 3) offer potential to estimate the relative impact of such stressors on coral communities. The response of coral communities to the effects of sedimentation (Barnes &

Lough 1997), turbidity/ light reduction (Rotman 2004) and fluvial salinity gradients (True, unpublished data) are all amenable to proxy assessment by TTL measurement. In this respect TTL is demonstrated to be a much more responsive monitoring parameter than skeletal growth rate and density (e.g. Chapter 4; Barnes & Lough, 1997; Anthony et al. 2002) or visible signs of stress (Chapter 3; Rotman, 2004).

Further work needs to be done to identify the physiological processes surrounding tissue uplift and energy sequestration in *Porites* to clarify issues about density banding and stress responses. The contribution of reproduction to annual cycles in massive *Porites* has not been rigorously examined. The threshold of light saturation is a natural starting point to examine mechanisms involved in seasonal cycles of both TTL and skeletogenesis. Likewise, it will be important to examine biological cycles in corals without strongly seasonal environments (particularly with regards to temperature). A major finding of this work is how much is yet unknown about the responses of corals to stress and seasonal environmental fluctuations.

5.7 General conclusions

- Tissue layer thickness of massive *Porites* corals varies in space and time;
- Spatial variation in TTL is mostly a manifestation of local (habitat-level) environmental conditions rather than an indication of broad geographic trends in conditions;
- Seasonal trends in TTL variation are similar to seasonal patterns (i.e. summer maximum, winter minimum) reported for calcification and linear extension in *Porites* on the central GBR, but are ;
- SST and insolation are the factors most likely to be responsible for seasonal cycles in TTL; long term monitoring of TTL in low latitude environments (where seasonal variation in temperature is not pronounced) may yield insight into the importance of tissue versus skeletal variation in the formation of skeletal density bands;
- TTL variation is a sensitive indicator of incipient or sublethal stress in massive *Porites*. It is not possible, however, to distinguish between chronic and acute stress solely from the thickness of the tissue layer;
- Continual mobilization of the lower portion of the tissue layer allows massive *Porites* some degree of control over their response to stressful conditions: tissue thickness and lipid levels appear to be interchangeable indications of the energy level of the polyp under stress, although extreme responses such as bleaching are more closely linked to tissue thickness than lipid levels;

- The response of corals to environmental impacts is dictated in large part by their available energy reserve. Diminishment of the ability of the coral to acquire energy leads to a reduction in linear extension as the coral adapts;
- Reduction of the coral's somatic reserve past a critical point leads to a bleaching response concomitant with shutdown of other functions;
- Proxy environmental reconstruction techniques based on isotope ratios within the coral skeleton are able to capture stress events in the life of a coral identified by TTL changes. Their sensitivity is diminished, however, by previous stress events. Likewise, interpretation of the nature of the stress event from the proxy record is problematic: proximal environmental variation is not recorded, and non-related stress events can leave identical signatures in the isotope record.
- The use of TTL variation as a reactive monitoring technique is not really feasible. Between colony and habitat and seasonal variation, as well as the need to contextualize measurements to previous history place severe constraints on its utility as a simple technique;
- Gradients in TTL corresponding to environmental gradients are a simple means of identifying those environmental gradients and mapping their severity.

5.7 Publications arising...

•True, Barnes, Willis (JEMBE: accepted manuscript)

–Measuring stress in corals: the thickness of the tissue layer is a proxy for coral condition

•True, Gagan, Barnes (Geochim. Cosmochim: in prep)

–Stress in the life of a massive coral affects stable isotope ratios in its skeleton

•True, Gagan, McCulloch (Science/Geochim. Cosmochim: in prep)

–Fidelity of geochemical proxies in coral skeletons during stress events

•True (Coral Reefs: in prep)

-Coral tissue as an energy reserve during stress

•True (Coral Reefs: in prep)

-Lesion healing strategies in massive *Porites* corals