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Coral injury and recovery: matrix models link process to pattern

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Abstract

In corals, extant damage to colonies depends on the balance between injury and recovery rates. Sclerochronology – the study of scleractinian coral growth rings – provides information about these historical processes, since corals preserve traces of previous injuries and annual bands representing regrowth. Cross-sections of two Caribbean coral species, *Porites astreoides* (Ellis and Solander) and *Siderastrea siderea* Le Sueur, were examined to determine the size-frequency and incidence of partial mortality and the rate at which these injuries recover. Each year's injuries were divided into four size classes, and subsequent changes in lesion size were developed into probabilities of switching size classes each year. Matrix models, composed of a matrix of transition probabilities and a vector added annually to account for new damage, predicted a dynamic equilibrium of extant damage to corals that matches field censuses well. A census of corals was carried out at two (*P. astreoides*) or five (*S. siderea*) sites. In 23 of 28 comparisons, values of extant damage were within the 95% confidence intervals of model predictions. Discrepancies arose because the model underestimated small lesions in *P. astreoides*. Sclerochronology may fail in this instance because small, rapidly-recovering injuries do not leave permanent scars in coral skeleton, although they are common in point observations of reef damage. Based on sclerochronology, *P. astreoides* and *S. siderea* are differentially susceptible to injury, and *P. astreoides* tends to recover more rapidly. ©1997 Elsevier Science B.V.

Keywords: Disturbance; Partial mortality; *Porites astreoides*; Regeneration; Scleractinian coral; Sclerochronology; *Siderastrea siderea*

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1. Introduction

The morphology of colonial organisms allows modules to die while the colony as a whole persists and may subsequently regenerate. In coral reef systems, modular organisms include many of the most abundant animals (bryozoans, ascidians, zoanthids, scleractinian and gorgonian corals), as well as algae. Colonies suffer partial mortality from numerous natural (Porter, 1972; Woodley et al., 1981; Hayes, 1990; Bythell et al., 1993a,b) and anthropogenic (Jackson et al., 1989; Glynn, 1993) disturbances. The persistence of colonies depends on growth and recovery exceeding damage.

Although critical to colony survival, these rates of damage and recovery are difficult to discern. A colony showing a high level of partial mortality could have reached that state through severe injury or slow recovery; thus, single observations give little information about the causal processes leading to extant damage. In most colonial taxa, only long-term monitoring allows one to track shifts in colony size and damage, and therefore to deduce rates of injury and recovery. Recovery rates have been determined by making artificial lesions on colony surfaces and observing short-term recovery (Bak and Steward-van Es, 1980; Palumbi and Jackson, 1982; Bak, 1983; Wahle, 1983; Guzmán et al., 1991). Scleractinian corals however, exhibit characteristics that allow both injury and recovery rates to be determined. Each year's growth is laid down in permanent skeleton on the surface of the colony, and the resultant skeletal growth rings contain a record of previous injuries and regeneration.

Sclerochronology–the examination of annual growth rings preserved in coral skeletons–provides a valuable resource for information about the processes leading to partial mortality, but it has not yet been used for this purpose (although suggested by Buddemeier et al., 1974 and Jackson, 1983). This paper represents the first application of sclerochronology to injury and recovery rates, documented for a modest collection of coral colonies of two scleractinian species, *Siderastrea siderea* LeSueur and *Porites astreoides* (Ellis and Solander). This historical disturbance regime, integrated over time, should result in a characteristic level and pattern of damage visible at a single moment.

Simple transition matrices with parameters from sclerochronology were used to develop a prediction of extant damage. These matrices project partial mortality forward in time based on past injury and regeneration. Thus, the accuracy of sclerochronology can be tested by comparing predicted with actual levels of damage. In assessing these models, I used one-time field censuses of partial mortality on an independent set of coral colonies. Matrix models of partial mortality can distinguish species-specific responses to disturbance, lead to hypotheses about the injury and recovery occurring in populations of known extant damage, and forecast the effects of a change in disturbance regime.

2. Species, sites, and collection artifacts

The two coral species, *Porites astreoides* and *Siderastrea siderea*, differ in a number of obvious life-history characteristics. *P. astreoides* is hermaphroditic and broods larvae all year long. *S. siderea* has separate sexes and releases gametes seasonally into the water column (Soong, 1991). In tests of competitiveness with other corals, neither

species is highly aggressive, but *P. astreoides* does have the capacity to cover area quickly through growth or regeneration (Lang, 1973; Chornesky, 1989). *P. astreoides* has a higher growth rate than *S. siderea* (Guzmán et al., 1994) and a high turnover rate relative to other corals: only 6 to 13 years elapse between production and destruction of a given area of tissue (Hughes and Jackson, 1985; Bythell et al., 1993b). *P. astreoides* thus recruits readily, grows rapidly, but does not persist long. *S. siderea*, on the other hand, reaches ages of more than a century (Guzmán et al., 1993). Partial mortality is common in *S. siderea*, with larger colonies often fragmenting into smaller sub-colonies that are connected by old skeletal remains.

Both of these species are common on shallow coral reefs on the Caribbean side of Panama. I examined cross-sections of colonies that had been collected in 1988 from three different areas (Fig. 1). Punta Galeta, near the mouth of the Panama Canal, experienced a refinery leak of about 50 000 gallons of oil in 1986 (Jackson et al., 1989). Isla Grande lies approximately 20 km to the northeast, also along the Panama coast, and the San Blas Islands are an additional 20 km east and 5 km offshore. All of the colonies collected for sclerochronology came from depths of 1–3 m. The total sample included 79 colonies of *P. astreoides* and 89 colonies of *S. siderea* from eight reefs near Punta Galeta, four reefs near Isla Grande, and six reefs in the San Blas Islands (Table 1). The colonies had been collected after the refinery leak in order to assess the effects of oil on recent colony growth rates, sediment incorporation, and reproductive status (Guzmán et

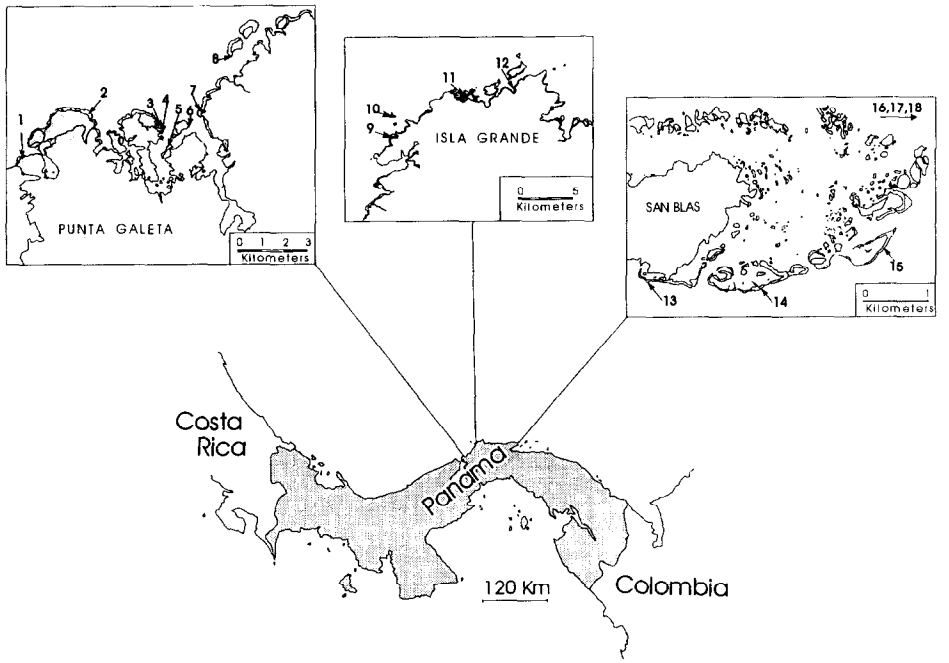

Fig. 1. Map of Panama, showing locations of reefs from which corals were collected. Numbers correspond to reefs listed in Table 1.

Table 1
Colonies collected in 1987–1988 for sclerochronology

Region	Reef	*Porites astreoides*			*Siderastrea siderea*		
		Number of colonies	Mean colony diameter (cm)	Collection depth (m)	Number of colonies	Mean colony diameter (cm)	Collection depth (m)
Punta Galeta	1 Margarita 3	4	21.5 (3.7)	1–2	5	33.5 (4.4)	1.5–2
	2 Galeta Channel	5	26.1 (2.2)	1.0	4	24.3 (5.1)	1–1.5
	3 Largo Remo East	5	20.0 (1.0)	1–1.5	5	28.5 (2.3)	1–1.3
	4 Toro Point 2	5	33.6 (2.7)	1.5	5	38.2 (4.9)	1.5
	5 Payardi West				5	26.0 (4.4)	1–1.5
	6 Payardi North	5	26.0 (3.1)	1.5	5	30.5 (3.3)	1–1.6
	7 Punta Muerto	5	18.7 (2.1)	1–1.5	5	26.8 (1.0)	0.85–1
	8 Naranjos South	4	22.8 (3.2)	1–1.5	5	28.8 (2.7)	0.6–1.5
Isla Grande	9 Doncella Reef	5	25.2 (2.9)	1–2	5	31.7 (2.4)	1.8–2.5
	10 Dos Marias	4	23.9 (2.8)	1.5	5	27.8 (3.5)	1.5–4
	11 Palina West	4	29.0 (3.6)	1.5	5	26.5 (3.3)	1–3
	12 Juan Gallego	5	28.6 (2.0)	1.5–2	5	29.7 (1.8)	1–3
San Blas	13 Ulag Sukun	5	32.0 (4.0)	1.5	5	38.5 (4.7)	1.5
	14 Korbiski	5	34.9 (4.2)	1.0	5	29.6 (3.9)	1.5
	15 Aguadargana	3	40.7 (3.3)	1.0	5	35.2 (2.6)	1.0
	16 Limones 3	5	39.9 (3.1)	1.5	5	36.0 (4.7)	1.5
	17 Limones 2	5	41.5 (4.4)	1.0	5	35.9 (2.3)	1.0
	18 Holandes	5	36.8 (2.4)	1.5	5	33.2 (3.6)	1–1.5

The table includes the actual number of colonies examined from each reef, their average diameter (SE), and range of collection depths.

al., 1991; Budd et al., 1993; Guzmán and Holst, 1993). One longitudinal section of 5–7 mm width was made through the diameter of each colony, and this section was x-rayed. The x-rays show clear growth rings, as well as injuries and recovery visible as deformations in the annual banding patterns (Fig. 2). Injuries are easily distinguished from bioerosion of old skeleton, because the latter eliminates sections of concentric growth rings without being associated with a redirection of coral growth.

Because the colonies were collected for a purpose unrelated to the assessment of partial mortality, they represent a biased sample of the disturbance experienced by corals at these reefs. The colonies are unrepresentative because (i) they include a restricted size range (30–50 cm in diameter), (ii) they are relatively intact and do not include any colonies that died completely, (iii) they are of variable ages, so the assessment of injury extending into the past is based on an ever smaller sample size, and (iv) they are sectioned only once, which precludes knowing the actual size of any intercepted lesions. In addition, LeTissier et al. (1994) have recently shown that many measurements based on sclerochronology can be obscured by x-rays, especially if cross-sections are not through the main growth axis of the colony.

Despite the constraints of the data set, however, this large collection of coral cross-sections provides a rare opportunity to assess the potential of sclerochronology to reconstruct dynamics of damage and recovery, as well as more conventional measurements of growth rate and bioerosion (Wells, 1963; Buddemeier et al., 1974; Highsmith, 1981; Highsmith et al., 1983; Beck et al., 1992; Risk and Pearce, 1992; Bosscher, 1993). The cross-sections provided 151 instances of *P. astreoides* injury, and 206 injuries to *S. siderea*, which I used in a simple matrix model to predict how much damage should be visible at the surface of corals at any given time. These predictions were then compared to in situ observations of damage gathered from two of the same areas in which colonies had been collected for sclerochronology - along transects at Largo Remo West, near Punta Galeta, and Aguadargana, in the San Blas Islands.

3. Models

The dynamics of injury and recovery can be likened to population dynamics, for which the use of matrix models is well-established (Bierzychudek, 1982; Hughes, 1984; Crouse et al., 1987; Caswell, 1989; Harvell et al., 1990; McFadden, 1991; Stacey and Taper, 1992). Each year, new injuries of various sizes occur (akin to individuals of different stages entering a population through migration, not reproduction), and over time these lesions shrink or expand, at the same time changing their size class membership (akin to organisms moving between different stage-classes).

The number of injuries occurring in each size class across a square meter of live coral each year can be represented by a vector V_t. After an injury occurs, the resulting lesion may shrink, stay the same, or expand. Given a group of injuries of size i, some proportion will move to each fate each year: a_{ij} shrink from size j to size i when $i < j$; a_{ii} stay at size i; and a_{ij} expand from size j to size i when $i > j$. These proportions are readily combined into a recovery matrix A, with elements a_{ij} representing all state-fate transitions that lesions could experience as they recover.

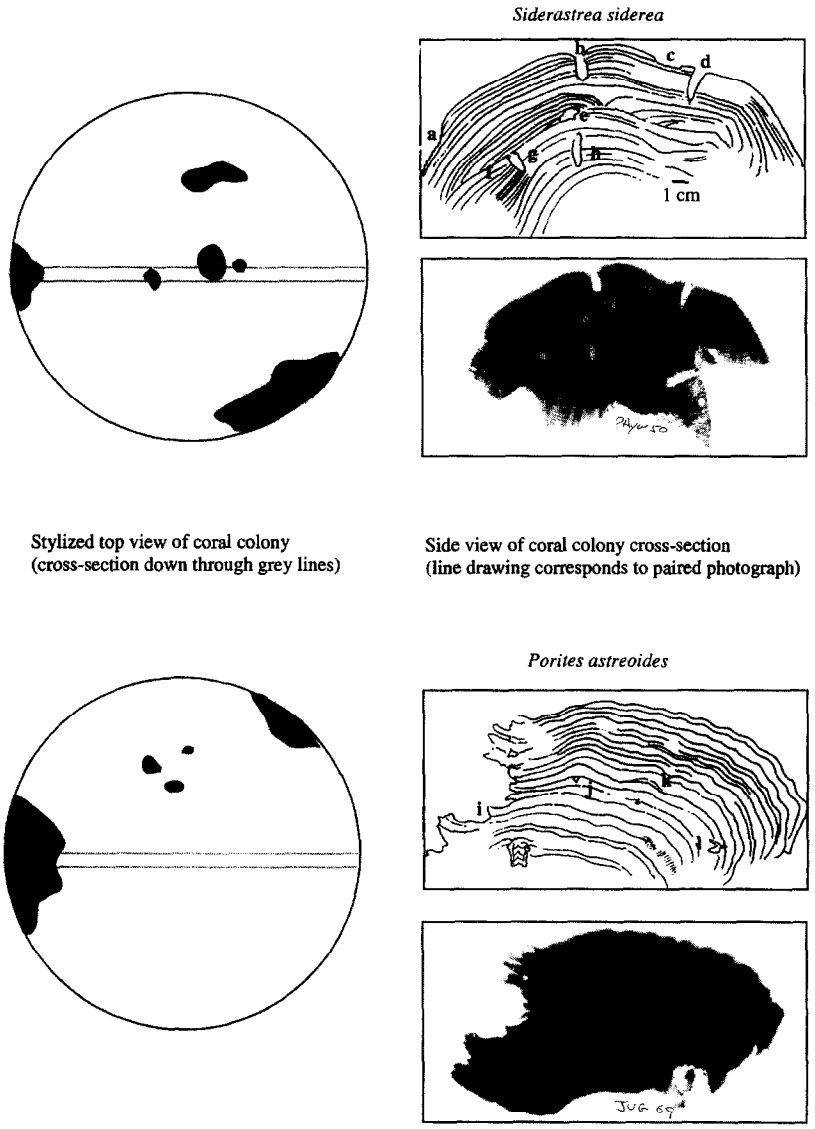

Fig. 2. Cross-sections of *Siderastrea siderea* and *Porites astreoides*. The photographs are examples of the sections that were available for describing historical damage and recovery. The line drawings, which highlight the annual growth rings in the two coral species, explain how injuries were measured. On the left is a schematic drawing of how the actual coral colony may have looked from above, before the longitudinal slice was taken to look at past tissue layers. The following letters refer to injuries in the line drawings: (a) 3 cm injury 2 yrs ago, not recovering; (b) 0.8 cm injury 2 yrs ago, followed by bioerosion of lower layers, not recovering; (c) 2 cm injury 1 yr ago; (d) calyces split, but no obvious injury; (e) 8 cm central injury 13 yrs ago, recovered in 5 yrs; (f) 4 cm peripheral injury 13 yrs ago, recovered in 6 yrs; (h) bioerosion; (i) 4 cm injury 15 yrs ago, followed by numerous small peripheral injuries and regrowth; (j) barnacle; (k) 0.3 cm injury 11 yrs ago recovered in less than 1 yr; (l) bioerosion.

The amount of dead skeleton found on coral colonies at any given time depends on the number of lesions from past years that have not yet completely recovered, added to new injuries of the year. If N_t is a vector representing the density of extant lesions in each size class, then extant damage changes through time as

$$N_t = A \cdot N_{t-1} + V_t.$$

In models of population dynamics, matrices are summarized by their dominant eigenvalue λ, which represents the asymptotic rate of growth of the population (increasing when $\lambda > 1$), and by their stable size distribution, which is the right eigenvector of the matrix (Caswell, 1989). The recovery matrix differs in subtle but important ways. Injuries, unlike organisms, neither reproduce nor die but simply fade away. Thus, there is no top row of the matrix representing age-specific fecundity, and shrinkage appears in the upper subdiagonal. In general, matrix columns must sum to unity in order to account for the fates of all lesions in a size class, although matrix columns representing small size classes will sum to less than one if some lesions recover completely. Because at least a few lesions in each size class shrink each year, the dominant eigenvalue of the matrix, λ, is always less than one and determines how fast one cohort of damage will disappear. The stable size distribution of lesions on coral depends not only on the recovery matrix, but on the vector of incident damage. With constant annual addition of new injuries (V), models eventually result in stable levels of extant damage, where $N_t = N_{t+1}$. If V is constant and I is the identity matrix,

$$N_{t+1} = N_t = (I - A)^{-1} \cdot V.$$

Thus a change in the rate of injury, V, will produce a corresponding change in the level of extant damage. If the incidence of injury varies from year to year, extant damage can nevertheless be summarized by a mean and variance of lesion densities.

Most matrix models of population dynamics are assessed by comparing the predicted size or age distribution to the same population that generated the parameters in the model (Bierzychudek, 1982; Crouse et al., 1987; Harvell et al., 1990; McFadden, 1991; Stacey and Taper, 1992). In contrast, the assessment presented here for models of partial mortality relies on stronger tests. Rather than predicting the relative abundances of larger and smaller lesions, they generate an absolute number of lesions of different size$\cdot m^{-2}$ of colony surface. Furthermore, predictions can be compared to an independent set of colonies not used in the construction of the model.

4. Sclerochronology

4.1. Sampling procedure

The matrix model parameters were derived from cross-sections cut vertically through the center of collected colonies. All colonies were described by basic colony measurements and by properties of each lesion where it was intercepted by the cross-section. Colony measurements included: (i) surface diameter, (ii) diameter of earliest distinct

growth ring, (iii) year of earliest growth ring, and (iv) length of the growth axis from the year of the earliest growth ring to the surface. Each lesion was measured for: (i) length, (ii) distance to the nearest colony edge, (iii) year of injury, (iv) total coral diameter in that year, and (v) change in lesion size from initial occurrence until the injury recovered or the colony was collected.

To convert these data into annual rates of change in lesion size, I assumed that the length of the lesion changed at a constant rate, unless the skeleton accreting after injury obviously reversed direction. This reversal occurred in 7% of lesions in both coral species. The assumption of a constant rate of shrinkage differs from studies of artificial lesions, in which recovery rates tend to decline over time. Recovery rates slow as energy stores in the tissue around the injury are exhausted and as new tissue encounters competitors (Bak et al., 1977; Bak and Steward-van Es, 1980; Bak, 1983). The difference between recovery rates in sclerochronology and observations of artificial lesions stems from the time frame involved. The recovery of artificial lesions over several months would not be perceptible in a cross-section that records annual events. Rather, the shrinkage of lesions I observed in sclerochronology represents growth across a substratum as new tissue extends at a constant rate. Artificial lesions tracked over a period of years should exhibit similar constant recovery rates.

A recovery rate (s) was calculated as $s = \frac{(l_0 - l_i)}{i}$, where l_0 is the most recent length of the lesion, l_i is the original length of the injury, and i is the number of years intervening. If the lesion recovered completely, $l_0 = 0$ and i is the number of years it took for coral tissue to regrow over the injury. For lesions that shrank in size, $s < 0$, and for lesions that expanded, $s > 0$. Lesion expansion after initial injury occurred relatively rarely in these cross-sections - in only 8% of *P. astreoides* and 5% of *S. siderea* lesions.

4.2. Recovery matrices

The recovery matrix for each species was based on all injuries encountered in cross-sections, integrated across both time and space; injuries were included regardless of where they occurred on the colony, where the colony had been collected, or, with some exceptions, the year of the injury. Although not ideal, particularly because some types of injuries appeared to recover more rapidly at Isla Grande (Table 2), combining across areas was required to generate a sufficient sample size of lesions for the recovery matrix.

Injuries that occurred in internal areas of the colony recovered more rapidly than peripheral lesions (Table 3), a consequence of overgrowth occurring from two directions instead of one. I corrected for this discrepancy by multiplying recovery rates (shrinkage and expansion) of all peripheral lesions by the average proportional difference in shrinkage between internal and peripheral injuries ($\times 1.45$ for *P. astreoides*, and $\times 1.36$ for *S. siderea* lesions ≥ 1 cm). In *S. siderea*, an unusually large proportion of 1 and 2 yr old injuries still remained static in size at the time of colony collection, perhaps due to short delays in the initiation of regrowth. In order to be included in the matrix, *S. siderea* injuries had to be at least 3 yr old, and *P. astreoides* injuries had to be at least 1 yr old (*P. astreoides* $n = 148$; *S. siderea* $n = 166$).

Lesions were divided into four size classes: 0–<1 cm, 1–<2 cm, 2–<3 cm and ≥ 3 cm. As lesions contracted or expanded, they changed their size class membership. I

Table 2
Regional recovery rates based on sclerochronology

	Species	Recovery rate ($cm \cdot yr^{-1}$)		
		Punta Galeta	Isla Grande	San Blas
Peripheral injuries	*P. astreoides*	0:79 (0.09) (26)	1.09 (0.12) (20)	0.60 (0.07) (27)
	S. siderea	0.40 (0.04) (30)	0.39 (0.04) (10)	0.40 (0.04) (23)
Central injuries	*P. astreoides*	1.29 (0.34) (6)	1.47 (0.25) (8)	1.25 (0.47) (5)
	S. siderea	0.60 (0.11) (6)	1.04 (0.22) (5)	0.45 (0.08) (10)

Porites astreoides and *Siderastrea siderea* colonies were collected from sets of reefs separated by over 20 km. Mean (SE) values are given, along with the number of lesions contributing to the values (in parentheses below). Species ($P=0.0001$), region ($P=0.0043$) and location of injury ($P=0.0001$) all have significant effects in ANOVA, but none of the factor interactions are significant. This table only presents data from lesions of at least 1 cm in length that were in the process of recovering.

based annual transitions of injuries among classes on whether an injury's average recovery rate (s) caused it to move into a new size class within 1 year. This strategy was not possible in the case of the largest size class, however, because these injuries spanned a wide size range and could not necessarily recover fast enough to shrink out of the size class in a year. For these largest injuries, transitions depended on the reciprocal of the average number of years it took an injury of average size to shrink to the lower limit of the size class (Table 4). This estimation problem and my solution are actually similar to the problem of estimating transition rates in stage-structured population models, for which organisms take several years to complete a stage (Crouse et al., 1987).

Recovery of injuries differs between *P. astreoides* and *S. siderea*. The differences are statistically significant when evaluated using log-linear analysis, which assesses the conditional independence of state-fate transitions in each species (Bishop et al., 1975; Wilkinson, 1989). The model for conditional independence

$$\log m_{ifs} = u + u_i + u_f + u_s + u_{if} + u_{is}$$

establishes whether the fate (t) of injuries in a given initial size class (i) depends on species (s). The full model shows significant species-specific differences in size class transitions (Likelihood ratio $X^2=29.6$; $P=0.02$), primarily due to size class 2, in which, analyzed separately, injuries to *P. astreoides* recover more rapidly than their counterparts in *S. siderea* (Likelihood ratio $X^2=16.61$; $P=0.001$).

These counts of injury fates are changed into probabilities for use in the recovery matrices. The full models representing partial mortality of *P. astreoides* and *S. siderea* are:

P. astreoides

$$\begin{pmatrix} 0.47 & 0.33 & 0.18 & 0 \\ 0 & 0.24 & 0.32 & 0.05 \\ 0 & 0.04 & 0.41 & 0.25 \\ 0 & 0 & 0.045 & 0.7 \end{pmatrix} \cdot N_{t-1} + V_t = N_t$$

Table 3
Linear recovery rates (SE) of injuries to *P. astreoides* and *S. siderea*

	Peripheral injuries		Central injuries		
	Recovery rate(cm/yr)	Number of injuries	Recovery rate(cm/yr)	Number of injuries	Unpaired *t*-test
All injury sizes:					
P. astreoides	0.80 (0.06)	73	1.16 (0.15)	27	$P=0.006$
S. siderea	0.45 (0.03)	73	0.43 (0.05)	56	$P=0.66$
Injuries $1 \leq x \leq 8$ cm:					
P. astreoides	0.81 (0.06)	64	1.39 (0.18)	19	$P=0.0002$
S. siderea	0.45 (0.03)	63	0.61 (0.08)	18	$P=0.02$

Injuries were measured in all cross-sections available for sclerochronology, and were grouped as peripheral or central (surrounded by growing coral tissue). The sample includes only those injuries that were at least 1 (*P. astreoides*) or 3 (*S. siderea*) years old at the time of colony collection and had begun to recover. Relatively recent *S. siderea* lesions are excluded because a disproportionate number had failed to begin recovering. Recovery rate (s) is dependent on injury size (l) in only one case: central injuries to *S. siderea* ($s = -0.13\,l - 0.27$). The restriction of lesion sizes to between 1 and 8 cm eliminates the small-size internal lesions in *S. siderea* that recover particularly slowly. When injuries <1 cm are no longer present in the analysis, the significant regression between size and recovery rate disappears, and central injuries recover more rapidly than peripheral injuries.

Table 4
State-fate transitions for peripheral and central lesions based on injuries observed in cross-sections of *Porites astreoides* and *Siderastrea siderea*

Initial size	Size 1 yr later				
Porites astreoides	Gone	1	2	3	≥3
<1 cm	10	9			
1–<2 cm	18	15	11	2	
2–<3 cm	1	4	7	9	1
≥3 cm			3	15	43
Siderastrea siderea	Gone	1	2	3	≥3
<1 cm	19	29	2		
1–<2 cm	2	20	18		
2–<3 cm			16	14	1
≥3 cm				7	38

Transitions of the largest lesions were based on the number shrinking (for *P. astreoides* and *S. siderea*, respectively, $n=37$ and $n=29$), their mean size (5.3 cm and 5.2 cm) and mean recovery rate (1.11 cm·yr^{-1} and 0.67 cm·yr^{-1}).

S. siderea

$$\begin{pmatrix} 0.58 & 0.62 & 0 & 0 \\ 0.04 & 0.33 & 0.58 & 0 \\ 0 & 0 & 0.49 & 0.2 \\ 0 & 0 & 0.03 & 0.8 \end{pmatrix} \cdot N_{t-1} + V_t = N_t$$

4.3. Injury vectors

Although all lesions visible in cross-section were used to construct the recovery matrix, I based injury vectors on internal injuries only. This restriction stemmed from the difficulty of measuring edge lesions in the field, where peripheral damage rapidly becomes obscured by encrusting organisms and is difficult to score for size. Using only injuries that were intercepted by cross-sections away from peripheral edges made the model predictions more consistent with field observations. It is conceivable that some of these central injuries actually touched the edge of the colony outside the range of the single cross-section, so the model may over estimate the central injuries. Also in contrast to the recovery matrix, separate injury vectors were calculated for each year. This allowed environmental stochasticity to be incorporated into the model through variable incidence, although not through variable recovery.

Colonies are smaller when younger and some had only settled a few years before collection. Consequently, the amount of coral cross-section available for analysis is reduced in earlier samples relative to more recent years. Because cross-sections of early growth rings effectively sample less coral area, estimates of injury are poorer in the past. I extracted 15 different years of injury occurrence from *P. astreoides* cross-sections and 25 years from *S. siderea* (Table 5).

Table 5
Counts of injuries from cross-sections

Year	*P. astreoides*		Size class				*S. siderea*		Size class			
	n	*L* (cm)	1	2	3	4	*n*	*L* (cm)	1	2	3	4
1987	38	1099	1	1	1		30	1011	1		1	1
1986	79	2195	1	3	1	1	89	2650		4	1	5
1985	79	2108	3	1		1	89	2570	3	1	1	
1984	79	2021			1	1	89	2490	2			
1983	79	1935		1			89	2409	2	1	1	1
1982	78	1826			1		89	2329	2	2		
1981	73	1702	1	1			89	2249	2	1		
1980	73	1563		2			89	2169	3	1	2	1
1979	68	1382	1	1			89	2089	2			1
1978	68	1310		1			89	2009		1		
1977	63	1178					89	1928	4		1	2
1976	53	975	1	1			89	1848	2			1
1975	51	893					87	1744	2	1	1	1
1974	47	779	1	1			86	1666	1		2	
1973	40	623					85	1585	4		1	
1972							85	1512	1	2		
1971							83	1422	2	1		
1970							80	1293	1			
1969							77	1190	2			
1968							71	1047	1			
1967							66	964				4
1966							60	857				
1965							56	786	2			
1964							51	714	1			1
1963							46	625				

Table includes length of cross-section examined (L) and number (n) of colonies used to calculate incidence of centrally-located injuries in each year for *P. astreoides* and *S. siderea*.

To calculate the incidence of injuries in each year, I needed to account both for the smaller size and number of colonies in earlier years, and for the fact that longitudinal slices were used to estimate damage occurring across a planar area. The first issue was solved by normalizing the number of injuries counted in each year by the total length of cross-section (L) examined in that year, which was:

$$L_i = \sum_{colonies\ of\ age \geq i} \left(d_t + [d_0 - d_t] \cdot \left[1 - \frac{i}{t} \right] \right),$$

where i is the number of growth rings counting backwards from the colony surface, t is the total number of rings, d_t and d_0 are the diameters at the colony base and surface respectively. The transformation from linear to planar estimates of injury must take into consideration that large lesions are more likely to be intercepted by a random slice through the colony than are small ones, simply because they span more area. Injury incidence for each size class in each year ($I_{c,i}$) was:

$$I_{c,i} = \frac{n_{c,i}}{L_i \cdot (2 \cdot r_c + 0.6)},$$

where $n_{c,i}$ is the number of injuries in size class c in year i, L_i is the length of coral cross-section examined, and r_c is the mean radius of the size class. The denominator in this equation comes from noting that the actual width of the longitudinal slice taken from each colony was approximately 0.6 cm. In addition, these slices effectively sampled an area on either side that was, combined, as wide as each size class ($2*r_c$) – any injury whose midpoint fell within the distance of its radius from the cross-section would be visible on the x-ray (Fig. 3).

The two coral species show differences in damage incidence. *S. siderea* sustains more small (<1 cm) lesions, whereas *P. astreoides* is injured more often in size class 2. Injury rates in the larger size classes are indistinguishable for the two species (Fig. 4). In both species, annual damage was temporally uncorrelated among size classes (Runs test: $P>>0.1$ for all pair-wise combinations of size classes within each species).

4.4. Predictions of extant damage

The models' predictions depend on both the intensity and order of injury, since extant damage is a function of how much injury occurred in the most recent year and in prior years. Thus, a single prediction would not represent the variety of outcomes that can be generated by different sequences of injury. However, the realm of possible outcomes can be constructed by simulating multiple injury scenarios.

In this study, each simulation began with the average field values of extant damage for the species and ended when initial conditions no longer affected predictions. This point was 23 years for *P. astreoides* and 42 years for *S. siderea*, since it takes that long for

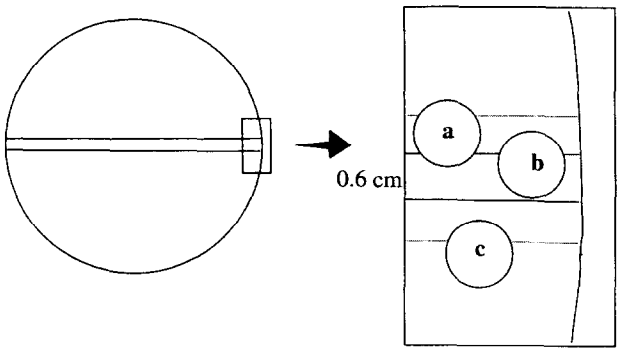

Fig. 3. Schematic drawing of coral colony surface, to show rationale behind transformation of injury data from 2- to 3-dimensions. Although the cross-sections taken through corals were thin slices, they nevertheless sampled an area of coral for damage. The cross-section samples an area that is its own width (about 0.6 cm) plus one injury radius on either side. Any injury of a given size whose midpoint falls within one radius of the cross-section will appear on the x-ray photograph (injuries a and b), but those falling outside the radius will not be included (injury c).

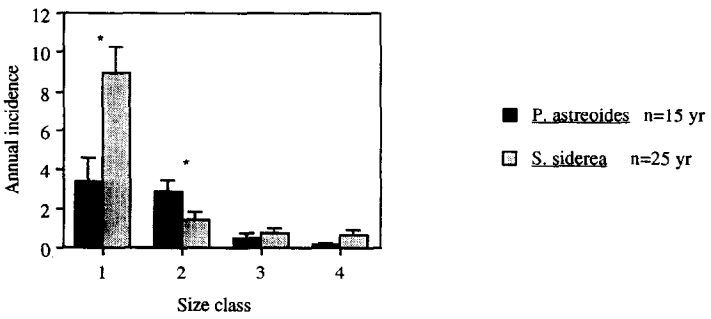

Fig. 4. Incidence of injury ($m^{-2} \cdot yr^{-1}$) in each of four size classes, based on coral cross-sections. Error bars are standard errors. * = Significant interspecific difference in injury.

average field damage, in the absence of new injury, to decline to <1% of original damage. During each year of the simulation, a different randomly-chosen injury vector was added, and the densities of lesions in each size class at the end of the 23 or 42 yr period were recorded. This procedure was repeated 1000 times for each species to generate distributions of possible levels of extant damage. The 95% confidence interval falls between the 25th and the 975th ordered value of the density of lesions of a given size (Table 6). The models predict differences in extant damage between the two species, with *S. siderea* showing significantly more internal extant damage than *P. astreoides* for all damage size classes (Unpaired *t*-tests between species, within each size class: $P<0.05$).

Table 6
Model outputs and censuses of extant damage (number of centrally-located lesions of each size found in 1 m^2 of coral)

Size (cm)	Mean	Range	95% CI	Punta Galeta	San Blas
Porites astreoides					
<1	9.55	0.91–24.62	2.52–20.45	106.1	94.9
1–<2	4.82	0.22–10.19	0.70– 9.12	20.4	12.3
2–<3	1.59	0.14– 5.46	0.37– 4.07	3.3*	4.0*
≥3	1.08	0.08– 3.30	0.26– 2.26	1.1*	0.4*
Siderastrea siderea					
<1	30.06	10.3–62.19	15.25–46.96	13.9	40.3*
1–<2	7.43	2.09–19.22	3.39–13.74	5.1*	5.6*
2–<3	2.74	0.27– 7.75	0.77– 9.83	2.7*	3.2*
>3	4.55	0.43–14.50	1.30– 9.83	7.2*	8.2*

Model results are presented as confidence intervals and ranges of possible extant damage, based on randomization tests. Census values are based on several 20×2 m transects at Punta Galeta and San Blas reefs.
* Census value within 95% CI predicted from randomization tests.

5. Censuses

Model outputs were compared to actual levels of extant damage in the field. The models should be able to forecast extant damage if certain conditions hold. First, the disturbance regime of past decades must be consistent over time. The models will account for random year-to-year fluctuations in injury, but they will fail if damage has followed consistent upward or downward trends. Second, all damage must leave permanent records in the skeleton. Any injuries that recover in less than a year would not deform the annual banding pattern and thus would fail to be incorporated in the model, even though they would be visible in a single observation of a colony's surface. Third, the models assume that recovery is a Markovian process; that is, the transition probabilities depend only on lesion size, not age. In contrast, calculated transitions do not necessarily reflect the fates of older lesions, because they were generated using only injuries that were initially that size.

Field patterns of partial mortality came from censuses of *P. astreoides* and *S. siderea* in April–June 1992 at some of the same sites from which colonies had been collected for cross-sections in 1987 and 1988. Three 20×2 m transects were established both at Largo Remo West, near Punta Galeta (2–3 m depth), and at Aguadargana, in the San Blas Islands (3–5 m depth). A census of *S. siderea* was carried out on all transects and *P. astreoides* along two in each area. The censuses generated a representative sample of 30–50 colonies (many of which had fragmented into smaller sub-colonies) of each species at each site. The total area of coral sampled, which depends on colony size as well as number, varied much more widely among sites, from less than 1 m^2 for *P. astreoides* at San Blas, to more than 17 m^2 for *S. siderea* at Galeta (Table 7).

Table 7
Summary of censuses

	Porites astreoides				*Siderastrea siderea*			
	Colonies	Sub-colonies	Surface area (m^2)	Lesions	Colonies	Sub-colonies	Surface area (m^2)	Lesions
Punta Galeta transects								
1	44	49	0.440	15 (1)	25	105	4.743	118 (66)
2	42	47	0.4698	101 (2)	27	183	9.602	150 (57)
3					26	103	2.969	72 (37)
San Blas transects								
1	60	78	1.011	126 (15)	10	27	0.654	16 (8)
2	37	38	1.229	76 (33)	12	43	1.507	50 (13)
3					11	24	0.509	58 (8)
Mangles					50	73	3.504	48 (50)
Salar 1					143	179	3.458	76 (47)
Salar 2					98	161	5.558	93 (88)

Both coral species were censused at Punta Galeta and San Blas. *S. siderea* colonies were also censused during 2 h dives at other San Blas sites (Mangles, Salar 1 and 2). Table includes the number of colonies and sub-colonies (colony fragments) found on each transect, along with the number of centrally-located lesions (followed by the number of holes occupied by blennies, crabs, fanworms, or other motile organisms).

For each colony on the transect, length (L) and width (W) (maximum and perpendicular dimensions) were measured, and its shape was recorded as flat or rounded. Similar measurements were taken on all colony fragments if colonies had split into smaller sub-colonies, from which I derived estimates of total area of live coral. Most sub-colonies were flat with roughly elliptical shapes, so:

$$area = \sum_{all\ live\ colonies} L \cdot W \cdot \frac{\pi}{4}$$

Lesions were defined as dead areas of coral surrounded by live tissue. For each lesion, I recorded length (l), width (w), and depth. Lesions that descended into the colony surface were considered to have occurred in prior years, while lesions at the surface probably occurred in the year of observation. Some lesions had eccentric shapes, in which case their size class membership would clearly depend on where they were sliced, so I used the average diameter of each lesion to determine its size class ($\sqrt{l \cdot w}$). By dividing counts of lesions in each size class by the total amount of coral tissue examined, I generated data of the actual density of lesions of each size.

6. Model–census comparisons

The models generated predictions about extant damage that were qualitatively, and in many cases quantitatively accurate (Fig. 5). As predicted, field data showed small lesions predominating in both species, and larger lesions to be more common in *S. siderea* than in *P. astreoides*. *S. siderea* showed more larger lesions in both model predictions and field censuses even though neither the incidence nor shrinkage of size class 4 differed statistically between coral species-the final difference was a result of two slight non-significant differences in the matrix model.

Not only were general patterns of damage consistent with model predictions, but actual densities of lesions often matched. Damage to *S. siderea* fell within 95% confidence intervals generated by the model for each size class except the smallest at Galeta. Censuses of size classes 3 and 4 in *P. astreoides* also matched model predictions, but the models substantially underestimated damage in the smallest size classes (Fig. 5).

7. Discussion

7.1. Why use sclerochronology and disturbance models?

Sclerochronology allows a sophisticated understanding of the factors leading to partial mortality of scleractinian corals. Unlike single censuses that show a static level of damage, sclerochronology reveals the dynamics of injury and recovery. Long-term studies to generate comparable data would take over a decade. To be of value, however, sclerochronology must provide reasonable estimates for injury and recovery rates. One way of assessing the information from sclerochronology is to determine how well it predicts one-time observations and distinguishes among species.

The value of incorporating injury and recovery rates, developed from sclerochronolo-

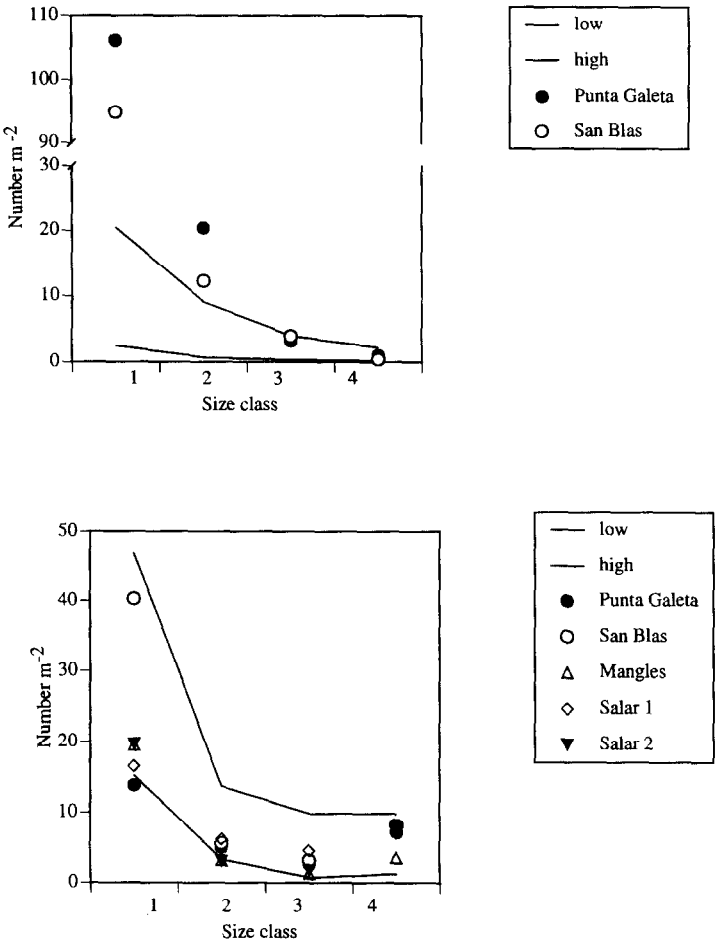

Fig. 5. (a) Extant damage to *Porites astreoides*. The solid lines represent an envelope of damage of different sizes within which 95% of all randomization tests fell. The points are census data of actual damage visible at two reefs. Note break in scale affecting size class 1. (b) Extant damage to *Siderastrea siderea*, from randomization tests and censuses at five reefs.

gy, into a model stems from prediction and generalization. The model output can be used to indicate whether the parameters are reasonable, to assess whether coral damage has accelerated, to forecast how a change in injury or recovery might affect extant damage, or to infer disturbance regimes in populations where only extant damage is known.

7.2. Comparing species

According to sclerochronology, *P. astreoides* sustains less injury to internal regions of the colony and recovers more rapidly than *S. siderea*. As a consequence, it shows less

partial mortality in an instantaneous census of extant damage. *P. astreoides*' faster recovery rate may derive from its faster growth rate (Guzmán et al., 1994), as new tissue extends from the live area around an injury. Lower levels of partial mortality might also be expected in *P. astreoides* because injury either recovers rapidly or kills colonies completely (Bythell et al., 1993b). Thus, in a sample of live colonies, extant damage would rarely be observed. Small differences between the species in injury and recovery rates translate into substantially higher predictions for extant damage to *S. siderea*.

7.3. Model-census discrepancies

Although matrix models generally made accurate predictions of levels of extant damage, the discrepancies may be more instructive than the consistencies. The models are least successful in predictions of the smallest size classes for *P. astreoides*. The discrepancy may arise because the model fails to account for numerous small injuries. Indeed, whereas the model predicts that new injuries should make up 25–50% of the lesions in the smallest size class, field data indicates that 80–90% of all small lesions are new (Fig. 6 Fig. 7 Fig. 8). The incidence of injury derived from sclerochronology is probably underestimated because many of these small injuries recover within weeks to months (Bak and Steward-van Es, 1980) and leave no skeletal trace. What the mismatch between model and census reveals is that much of the damage to *P. astreoides* lasts only a short while - not even long enough to mark the skeleton. Recovery may take energy from the colony (Meesters et al., 1994), but injury does not reduce the area of live tissue for long.

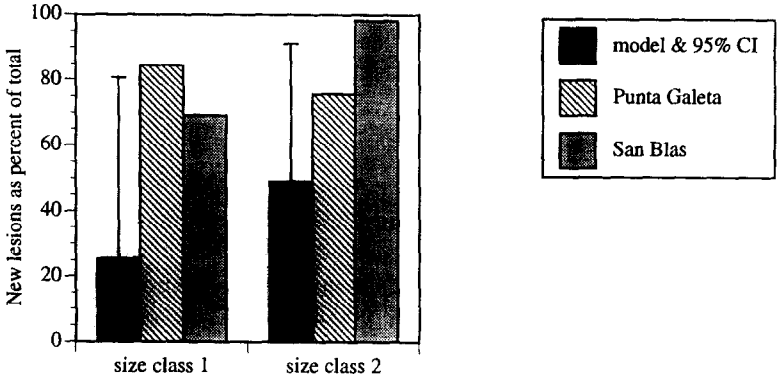

Fig. 6. New injuries sustained by *Porites astreoides* in the smallest lesion size classes, as a percent of the total number of lesions of that size. In the field, new injuries can be distinguished from older ones that have not yet recovered because new ones sit at the surface of colonies, without any coral tissue having accreted around them. The censuses show that recently-formed injuries <2 cm are much more common on reefs than the matrix model allows.

7.4. Turnover rates

The injuries visible in coral cross-sections can be used to define turnover rates in these coral species. Turnover rate (T) is calculated as the inverse of the proportion of tissue injured each year. It corresponds to the average time between consecutive injuries to a particular area of coral (rotation period; Paine and Levin, 1981) or, equivalently, to the time required to accumulate a total lesion area equal to the total coral area. Sclerochronology indicates a turnover rate of about 70 years for *P. astreoides* ($T = \frac{1}{0.014\ \mathrm{yr}^{-1}}$) and 120 years for *S. siderea* ($T = \frac{1}{0.0085\ \mathrm{yr}^{-1}}$) (Table 8). Alternative measures of turnover rates, measured directly as the proportion of injured tissue seen over long-term observations, only exist for *P. astreoides* and suggest a much shorter period of 6–13 years (Hughes and Jackson, 1985; Bythell et al., 1993b). Several possible factors could account for the discrepancy. Perhaps the corals in these Panamanian reefs have simply experienced less injury than in other areas. Quarterly transects in this region of Panama in 1987–88 showed no more than 5% of *P. astreoides* colonies had been damaged recently, and these few had suffered less than 10% mortality (Guzmán et al., 1993). If these censuses captured most new mortality, then less than 1% of *P. astreoides* was damaged annually, for a turnover rate of more than 100 years. Longer turnover times are to be expected from calculations that only include partial mortality, especially since total colony mortality has been shown in other populations to be an important component of damage in *P. astreoides* (Hughes and Jackson, 1985; Bythell et al., 1993b).

7.5. Spatial variation in damage and recovery

S. siderea predictions fare well when confronted by actual levels of partial mortality in areas where colonies were collected for sclerochronology. Furthermore, these same patterns of partial mortality are found at sites more than 20 km distant. At Salar 1, Salar

Table 8
Turnover rates for *Porites astreoides* and *Siderastrea siderea* based on sclerochronology

		Injury size			
		<1 cm	1–<2 cm	2–<3 cm	>3 cm
P. astreoides	Count	16	43	24	59
	Number m^{-2}	7.0	9.8	3.7	4.9
	Area of damage·m^{-2}	0.00014	0.0017	0.0018	0.010
		Turnover rate = 71 yr			
S. siderea	Count	44	42	39	57
	Number m^{-2}	9.7	4.9	3.1	2.1
	Area of damage m^{-2}	0.00019	0.00086	0.0015	0.0059
		Turnover rate = 120 yr			

For *P. astreodies*, up to 15 yr of annual growth rings were used (about 210 m of cross-section that could have been damaged, integrated over time and over all colonies), and damage occurring up to 25 yr before collection was included for *S. siderea* (about 410 m of cross-section that could have been damaged). Transformations from counts of intercepted damage to density and area assumed that injuries were circular, with a diameter equal to their intercepted length. The mid-point of the size class was used as the average diameter.

2, and Mangles reefs (10–12 m depth), I carried out a census of *S. siderea* colonies during 2 h diving at each site (Table 7). Extant damage at these sites also fell within the 95% confidence intervals generated by the *S. siderea* model (Fig. 4). Although one should be wary of extrapolating local disturbance processes to other locations, these additional censuses imply that the sorts of small scale injury sustained by *S. siderea* and its pace of recovery may be regionally similar.

The generality of these models, combined with their ability to predict a realm of possibility for extant damage that encompasses field observations, gives them a potential role in elucidating the dynamics of coral reef disturbance (Connell, 1978). The obvious drawbacks arise because of what is not preserved in coral skeletons: small, rapidly-recovering injuries and total colony death.

The absence of total mortality from these models is particularly troubling because of documented decreases in coral cover throughout tropical waters due to onslaughts of disease, pollution, and competition (Jackson et al., 1989; Glynn, 1993). Based only on partial mortality, sclerochronology indicates no obvious trends in injury rates over the past several decades, even at the site of several oil spills. Past injury and recovery rates gave predictions of extant damage indistinguishable in most cases from current levels. However, future studies using the record of disturbance in coral cross-sections could still profit from temporal comparisons, since longer records, random samples or larger colonies might be more likely to reveal changes in injury and recovery rates associated with declining environmental conditions.

Sclerochronology works on the same principle as dendrochronology: information about the history of organisms is preserved in growth rings. Dendrochronology has proven extraordinarily useful in reconstructing disturbance by forest fires (Heinselman, 1973; Romme, 1982; Swetnam, 1993), since fires leave tell-tale scars when they injure trees. Although coral cross-sections do not distinguish among the myriad sources of damage, they nevertheless preserve traces of injury and recovery. This study shows that those traces are likely to be accurate indicators of small-scale disturbance, as long as injuries do not immediately recover.

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