

The Rise and Fall of a Six-Year Coral-Fungal Epizootic

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ABSTRACT: Drivers of disease cycles are poorly understood in marine ecosystems in spite of increasing outbreaks. We monitored a newly emerged fungal epizootic (aspergillosis) affecting sea fan corals (*Gorgonia ventalina* L.) in the Florida Keys to evaluate causes of its rise and fall over 6 years. Since August 1997, aspergillosis has nearly eradicated large sea fans at some sites. However, sea fan densities have remained relatively constant due to episodic recruitment replacing large fans with small. Recruitment itself was affected by infection and occurred only when prevalence of disease was low. This impact on recruitment occurred because the largest, potentially most fecund colonies had the highest prevalence of disease, and the pathogen significantly suppressed reproduction of infected fans. Moreover, high mortality among adults resulted in a demographic shift to smaller colonies. The most dramatic impact of aspergillosis was the Keys-wide loss of >50% of sea fan tissue from complete and partial mortality. Aspergillosis prevalence has declined steadily over the last 6 years, and we consider the following hypotheses for decline of the epizootic: change in environment, change in pathogen input, and increase in host resistance. We conclude that increasing host resistance is the most likely driver of the decline. However, a change in any of a number of factors, for example, recruitment of naïve hosts, rate of pathogen input, or environmental conditions (water quality and temperature), is likely to promote reemergence of the epizootic.

Keywords: marine epidemic, disease impact, sea fan corals, gorgonians, coral reefs, aspergillosis.

For humans and in many terrestrial systems, the causes of disease outbreaks and subsequent temporal dynamics (i.e., duration and decline) are generally well known (Anderson and May 1991; Grenfell and Dobson 1995; Hudson et al. 2002). Although introduction of new pathogens is a common cause of disease outbreaks in humans and ani-

mals, changes in environment can also trigger new outbreaks (Hudson et al. 2002). Similarly, disease outbreaks in crops and wild plant populations can result from a combination of factors including low diversity and high density of hosts (Garrett and Mundt 1999; Mitchell et al. 2002), evolution or introduction of new pathogen strains (Goodwin 1997; Rizzo and Garbelotto 2003), and climatic shifts (Coakley et al. 1999; Harvell et al. 2002). For many plants and animals, the duration and subsequent decline of epidemics are often determined by the rate at which susceptibles are removed (Hochachka and Dhondt 2000), changes in pathogen virulence and host resistance (Thrall and Burdon 2003), and changes in environmental conditions (Harvell et al. 2002).

In contrast, little is known about disease outbreaks in natural marine ecosystems. Although pathogens and other microorganisms now dominate the ecology of some coastal marine ecosystems (Jackson et al. 2001) and epizootics of marine taxa are increasing (Ward and Lafferty 2004), there has still been little progress in understanding the population biology of infectious diseases in the ocean (Harvell et al. 1999). In this regard, the work of Swinton et al. (1998) on harbor seals and Powell et al. (1996) on oysters stands out in predicting both the duration and decline of epizootics based on infection rate of susceptible individuals. In harbor seals, models predicted that the massive outbreak of phocine distemper caused by a morbillivirus in the mid-1980s was largely driven by transmission among seals at haul-outs. Once the population dipped below a threshold density, transmission did indeed slow, and the epizootic faded as predicted. In oysters, epizootics caused by the endoparasite *Perkinsus marinus* are sensitive to the environment and follow dry summers and warm winters. Models with information about parasite transmission, host resistance, and environmental drivers are successful in predicting timing of outbreaks (Powell et al. 1996; Hofmann et al. 2001).

For most other marine taxa, there is very little information regarding even the most basic properties of marine pathogens (e.g., identity, host specificity, transmission modes) or the factors affecting disease processes (e.g., environmental correlates). Because most studies of marine diseases report only the initial impacts of outbreaks (Har-

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vell et al. 1999), we are limited in evaluating factors that affect disease emergence, maintenance, and decline, or host population recovery. The situation with corals is particularly urgent because there have been large and more frequent outbreaks in the last two decades (Patterson et al. 2002; Harvell et al. 2004). Corals are considered a “canary in the coal mine” for climate warming because they have a narrow thermal tolerance and small warming trends have triggered large bleaching events (Hoegh-Guldberg 1999; bleaching is when corals expel their symbiotic algae and turn white because of thermal stress). This is the first epidemiologically structured study of a coral-pathogen interaction.

Aspergillosis, a fungal disease of sea fan corals (*Gorgonia* spp. L.; fig. 1), was first documented in 1995 near Saba, Bahamas, and subsequently was detected throughout the Caribbean basin, including in the Florida Keys (Nagelkerken et al. 1997a, 1997b). The pathogen responsible for the outbreak was identified as the fungus *Aspergillus sydowii* (Smith et al. 1996; Geiser et al. 1998), a common soil microbe. Detailed microbial work to satisfy Koch’s

postulates confirmed *A. sydowii* as the causative agent of the sea fan disease. Although aspergilli are successful opportunistic pathogens of a range of taxa, including humans, insects, and marine mammals (Sweeney et al. 1976; Pier and Richard 1992), none was known previously to affect marine invertebrates. Because *A. sydowii* is terrestrial and does not sporulate in seawater, this unusual outbreak is best classified as an emergent disease.

The identification of *A. sydowii* as the causative agent of the sea fan disease, the tractability of the fungus in experiments, and the abundance and sessile habit of the coral host have provided an opportunity to characterize this pathosystem (Kim et al. 2000; Alker et al. 2001, in press; Petes et al. 2003) and evaluate how resistance (Dube et al. 2002) and environmental conditions (Kim and Harvell 2002) may drive patterns of disease impact.

Here, we report on the relatively long-term study on the epidemiology of aspergillosis of sea fans in the Florida Keys. We predicted that the aspergillosis outbreak would be unusually destructive given the opportunistic nature and wide host range of most aspergilli, the emergence of

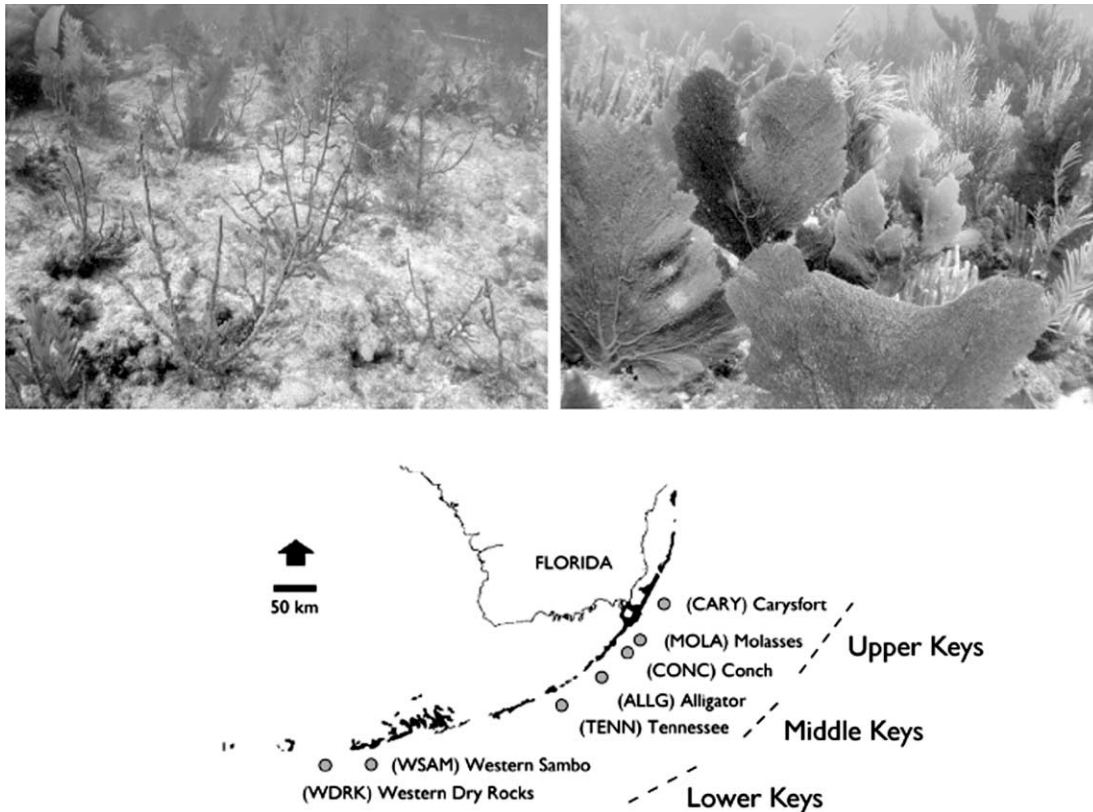


Figure 1: Aspergillosis of sea fan corals in the Florida Keys. The two photos contrast areas of high (*left*) and low (*right*) disease impact as indicated by high density of sea fan skeletal remains. The map shows locations and names (with abbreviations) of reefs with permanent sea fan monitoring transects. Regional designations (*Upper, Middle, Lower*) are also indicated.

aspergillosis in a new host, and the relatively high initial prevalence in this outbreak. An important objective of this study was to evaluate factors affecting the size-specific dynamics of an epizootic in a natural marine community and factors that underlie the rapid rise and slow fall of the outbreak.

Methods

To monitor aspergillosis of sea fans in the Florida Keys, permanent transects were established on shallow reefs (<8-m depth) spanning the length of the Keys (fig. 1). Sites were selected based on the presence of sea fan corals and the desire to monitor the length of the Keys across the three major regions—Upper, Middle, and Lower Keys—established by the U.S. Environmental Protection Agency (Hankinson and Conklin 1996). Data presented here are from seven reef sites (three transects per site) surveyed in August 1997, January 1998, May 1998, January 1999, May 1999, July 2000, May 2001, August 2002, and August 2003.

At each permanent transect, all sea fans with bases within 1 m of either side of the 25-m transect line were examined. For each colony, maximum height and number and sizes of lesions (i.e., percent disease damage) were recorded. All survey data presented here were collected by K.K. to minimize data collector error. Aspergillosis was diagnosed by the presence of distinctive lesions usually accompanied by purpling surrounding the lesions (Nagelkerken et al. 1997a, 1997b; Kim and Harvell 2002).

From the transect data, estimates of disease prevalence (percent of individuals infected) and severity (percent of colony area affected by disease) were derived. Colony area (in cm²) of individual sea fans and area affected by disease were estimated using a power function derived from image analysis relating surface area to colony height (Kim and Harvell 2002). Prior to analysis, data were summarized by transect, tested for normality and homogeneity of variances (Kolmogorov and Levene tests, respectively), and transformed as necessary.

Results

Disease Prevalence and Severity

At the start of our surveys in August 1997, approximately 31% (± 2.3 SEM) of all sea fans in our transects were infected with aspergillosis (fig. 2). Prevalence (percent of sea fans infected) varied across sites (repeated-measures ANOVA, $F = 16.75$, $P \leq .001$) and over time ($F = 34.41$, $P \leq .001$) with significant site-time interaction ($F = 3.646$, $P \leq .001$). In general, aspergillosis has been declining since the start of the surveys. However, on three occasions (May 1998, July 2000, and August 2002), isolated

mini-outbreaks were detected. The largest of the three occurred in July 2000 on Carysfort and Conch reefs, where aspergillosis increased approximately threefold compared to the previous year. By August 2003, Keys-wide prevalence of aspergillosis had declined to 5.9% ($\pm 1.4\%$).

Disease severity (percent of colony area affected by disease) also varied across sites ($F = 5.981$, $P \leq .0001$) and over time ($F = 2.195$, $P = .032$) with significant site-time interaction ($F = 1.571$, $P = .026$; fig. 2). Periods of high disease severity were noted sporadically but were less synchronous among sites and only nominally related to increases in prevalence ($R^2 = 0.079$, $P = .027$). Overall, average severity of aspergillosis in the Florida Keys has not declined. Despite high variance in prevalence among sites, there was no relationship between sea fan density and prevalence when examined in August 1997 and August 2003 (fig. 3).

Population Impact

Overall, Keys-wide sea fan population size remained unchanged over the course of the outbreak ($F = 1.883$, $P = .068$; fig. 4), largely due to sporadic (both among reefs and over time) but generally increasing levels of recruitment (estimated as the number of sea fans <10 cm in height; fig. 2). The total number of sea fans >10 cm declined by >27% between August 1997 and August 2003. If sea fans >20 cm are considered (i.e., discount recruitment over the last 2 years), the decline is even more dramatic at 39%. Recruitment was particularly important for two reefs. On Conch, the number of sea fans declined by >66% between August 1997 and May 2001 (0.84–0.28 sea fans m⁻²); however, two years of recruitment that were the highest among all reefs has apparently stopped the rapid decline. On Tennessee, the population declined by >53% between August 1997 and July 2000 (1.02–0.48 sea fans m⁻²) before returning to the August 1997 level in 2002.

Sea fan recruitment appears to be dependent on disease prevalence. Specifically, on six of seven reef sites, recruitment was inversely related to disease prevalence (fig. 5). When data were analyzed by monitoring dates ($N = 9$), only two showed a significant inverse relationship (data not shown).

Sea fan aspergillosis is highly size dependent (Kim and Harvell 2002). For instance, in August 1997, sea fans in the >80-cm-height class were >10 times more likely to be infected by aspergillosis than those in the <20-cm-height class. Once infected, the size of the infected area was 21 times greater among the larger sea fans. By August 2003, prevalence among the >80-cm-class was 25 times higher and infected area 14 times greater than those in the <20-cm-height class. The disproportionate impact of aspergil-

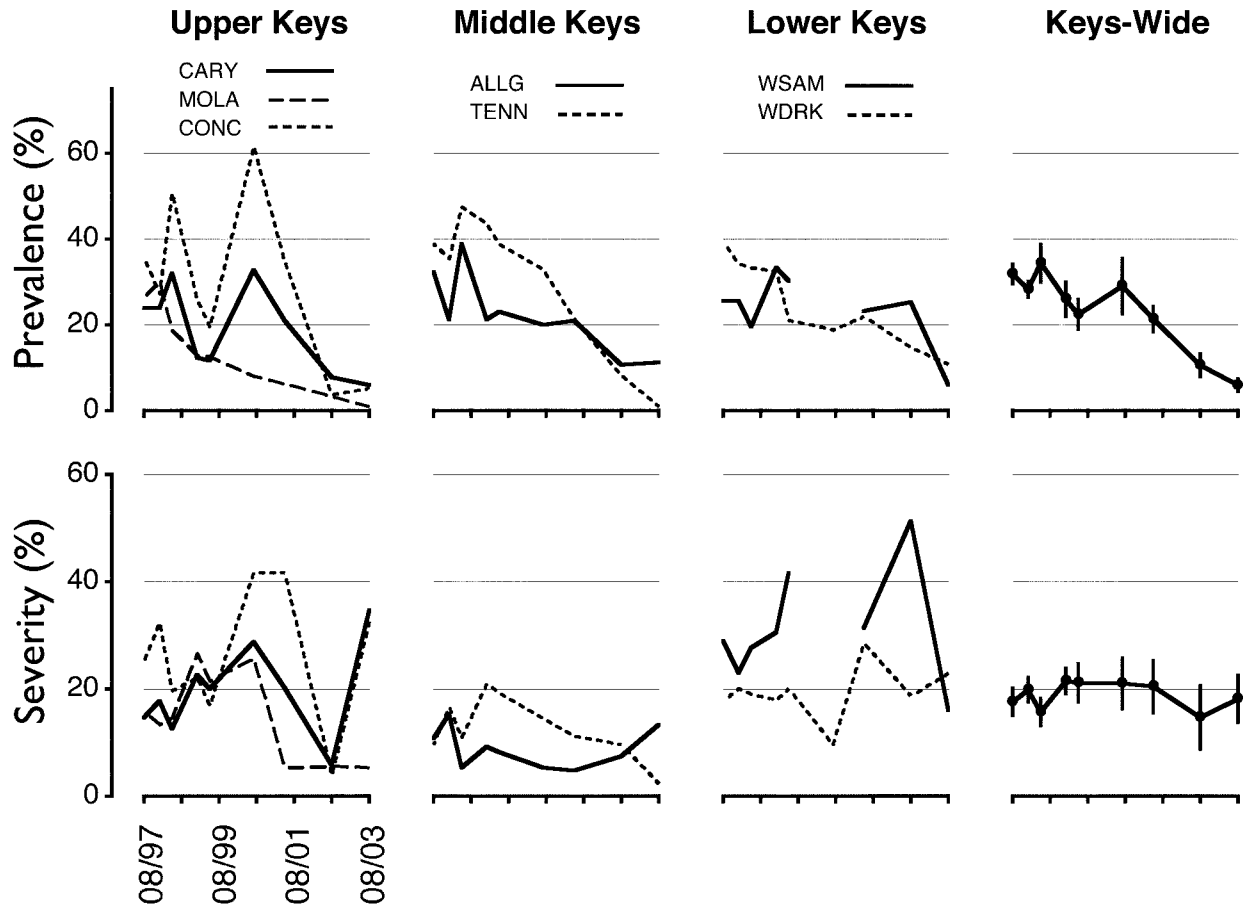


Figure 2: Aspergillosis prevalence (percent of colonies infected) and severity (percent of colony area affected by disease). Data for individual reefs are averages of three transects (error bars not shown). Keys-wide averages are also given with error bars indicating standard errors ($n = 7$ reefs). Reef abbreviations as in figure 1.

losis among larger sea fans and the increase in recruitment late in the epizootic resulted in a shift in the population structure to smaller sea fans (fig. 6). This trend was particularly dramatic on Conch Reef where sea fans <20 cm height now make up 90% of the population compared with 10% in August 1997. Overall, the Keys-wide median sea fan height declined from 40 cm in 1997 to 26 cm in August 2003. The impact of this downward shift in colony size is most evident when viewed as the change in sea fan tissue area (fig. 7). On average, partial and complete mortality resulted in a Keys-wide reduction in total sea fan tissue area of >50% ($\pm 12.7\%$) over the past 6 years, which translates to a loss rate of 8.4% ($\pm 2.1\%$) per year. This decline was particularly dramatic among reefs in the Middle and Lower Keys, with Conch losing >94% of total sea fan tissue area between August 1997 and August 2003.

Discussion

Impact of the Epizootic

Pathogens can be potent effectors of change in natural marine populations and communities (Harvell et al. 1999, 2004). However, the direct role of pathogens in population and community change has been demonstrated in only a few cases. For instance, although disease is widely accepted as the cause of major die-offs in *Diadema* sea urchins and acroporid corals in the Caribbean, causative microbial agents were never identified. Thus, the importance of this study is the demonstration of a significant disease impact on a coral population by a known pathogen. Indeed, the prevalence of aspergillosis, which ranged from 8% to nearly 60% (fig. 2), is among the highest reported for any coral disease (see also Nagelkerken et al. 1997b). Black

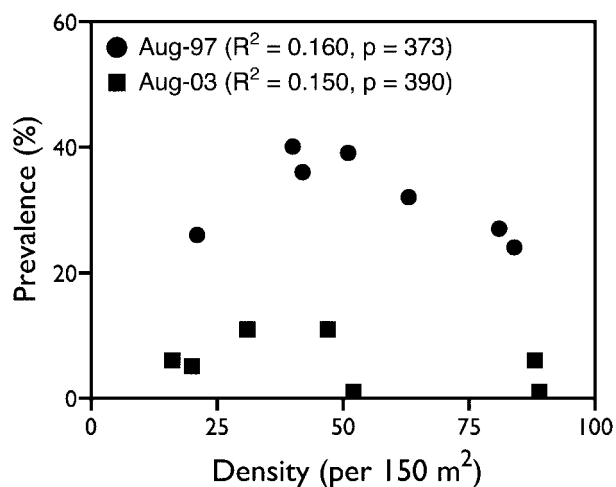


Figure 3: Density independence of aspergillosis. Data shown are from two monitoring dates (other dates are not shown but also reveal density independence).

band disease, which is a Caribbean-wide disease of scleractinian corals, occurs at frequencies $\leq 5.5\%$ (Garrett and Ducklow 1975; Edmunds 1991; Kuta and Richardson 1996). A recently reported bacterial disease referred to as “plague type II” had a prevalence as high as 33% in the elliptical star coral *Dichocoenia stokesi* in the Florida Keys (Richardson et al. 1998). In one study of white band disease, which is thought to be responsible for the virtual eradication of Acroporid corals from the Caribbean during the 1980s, prevalence among *Acropora palmata* in St. Croix, U.S. Virgin Islands, was 39%, with a mortality rate of $11\% \text{ year}^{-1}$ (Gladfelter 1982).

A population regulatory impact is one where the pathogen limits the intrinsic rate of increase of the host population (Tompkins et al. 2002). The regulatory role of aspergillosis is suggested by significant sea fan population and recruitment declines at some reefs. For instance, on Conch Reef, the sea fan population declined by $>72\%$ between August 1997 and May 2001 but later rebounded due to successful recruitment in the summers of 2002 and 2003 (fig. 4). Because sea fan corals are modular organisms, population dynamics and thus disease impact can be better understood in terms of colony size rather than simply the number of individuals (Hughes 1984). Partial mortality has the effect of reducing the size of sea fans and thus altering the size-class structure of the population. This effect can be relatively rapid. For instance, because many lesions form near the bases of colonies, even a small lesion can cause a large area of sea fan to be shed, thereby dramatically reducing colony size. Indeed, the combination of complete and partial mortality has resulted in the

loss of $>50\%$ of sea fan colony area over 6 years of monitoring (or $8.4\% \pm 2.1\% \text{ year}^{-1}$).

Aspergillosis disproportionately affects large sea fans (Kim and Harvell 2002), probably due to larger colonies that have lower antifungal defenses (Kim et al. 2000; Dube et al. 2002), serve as larger targets for pathogen interception, and accumulate more pathogen hits over their longer lifetimes. The strong relationship between colony size and both disease prevalence and severity has significant implications for recovery potential. Petes et al. (2003) showed that even small fungal lesions can suppress reproduction of an entire colony. Given that colony size is an important determinant of reproductive output (Beiring and Lasker 2000), partial loss of the large sea fans plus suppression of reproduction should have a significant impact on the size of the larval pool. In particular, if sea fans in the Keys reefs are largely self-seeding (Sammarco and Andrews 1988; Cowen et al. 2000), a continued downward demographic shift to smaller colonies (fig. 6) is likely to increasingly slow recruitment and thus population recovery.

A reemergence of aspergillosis was detected between 1999 and 2000 at Carysfort and Conch (fig. 2A). A significant feature of this reemergence was its impact on small sea fans. In May 1999, we detected ~ 80 recruits (i.e., colonies ≤ 10 cm height) on our transects, none of which was infected. By July 2000, prevalence in this height class was $\sim 11\%$, suggesting a shift in age-specific force of infection (Anderson and May 1991). This shift is likely due to the input of susceptibles into the population rather than the appearance of more virulent strain of pathogen, given that prevalence among larger fans actually decreased during the same time.

The substantial impact of aspergillosis on sea fans may be due to two factors. First, given that aspergilli are terrestrial in origin, sea fans may be a naive host and thus highly susceptible to aspergillosis. Second, the density independence of aspergillosis (fig. 3) suggests that disease dynamics are dominated by external inputs of the pathogen rather than within-population transmission (although there is evidence of some secondary transmission among nearby sea fans in the 2–8-m range [Jolles et al. 2002]). For instance, *Aspergillus sydowii* may persist in reservoirs, biotic or environmental, so that epidemics are sustained even when hosts are rare. Opportunistic pathogens, like aspergilli, are likely to have broad host ranges. Indeed, susceptibility of a number of other gorgonian corals was suggested by the low efficacies of chemically mediated resistance against *A. sydowii* (Kim et al. 2000). Subsequent experimental work has confirmed a wider host range for *A. sydowii* among Caribbean gorgonians (K. Kim, K. Shuster, A. Alker, J. W. Ward, and C. D. Harvell, unpublished manuscript). Thus, the maintenance of aspergillosis is unlikely to be affected by the reduction in any

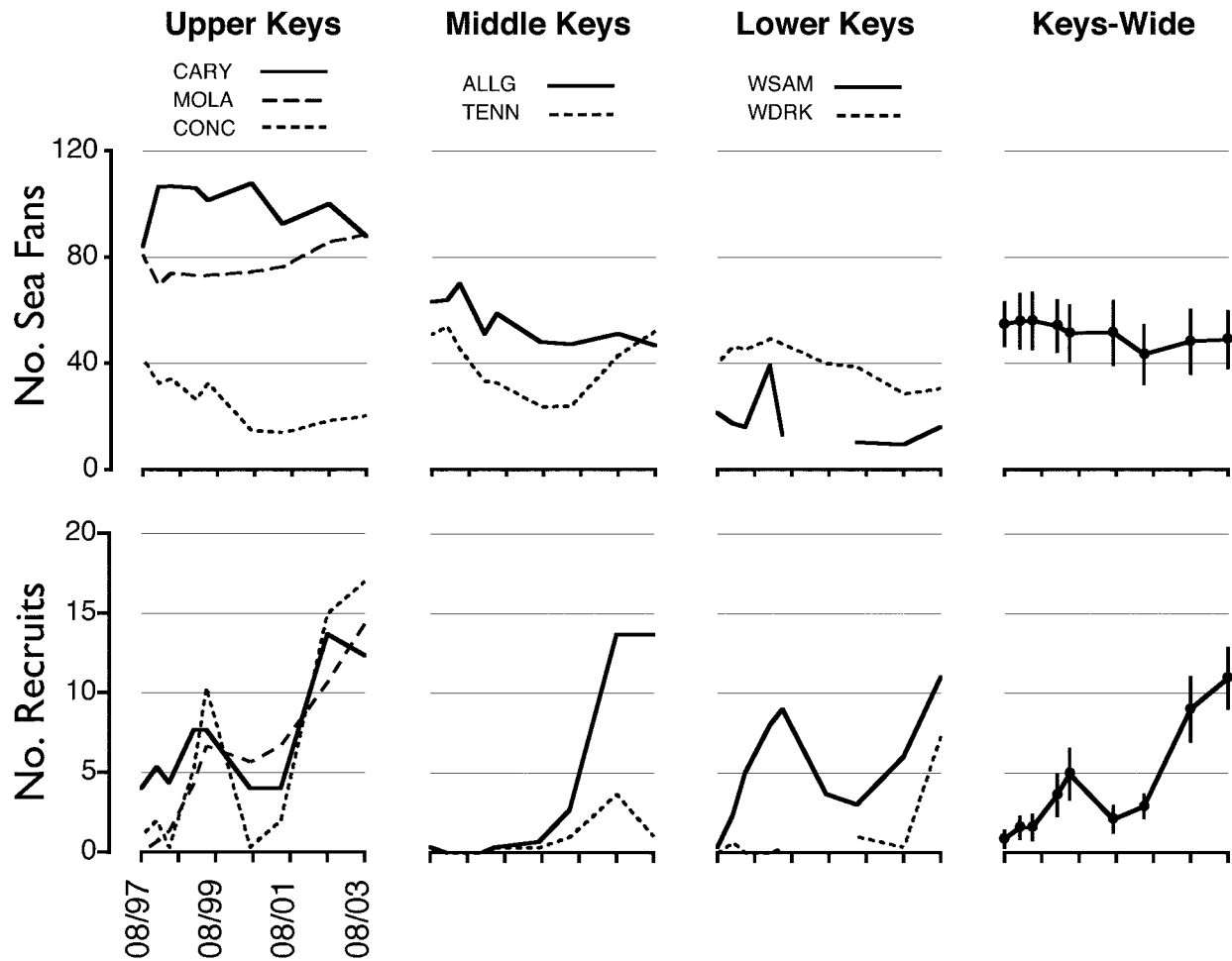


Figure 4: Impact of aspergillosis on the number of sea fans and number of recruits (sea fans <10 cm height). Data for individual reefs are averages of three transects (error bars not shown). Keys-wide averages are also given with error bars indicating standard errors ($n = 7$ reefs). Reef abbreviations as in figure 1.

one host species. The body of theory on two- or multihost, one-parasite systems supports this expectation (Hudson and Greenman 1998).

What Triggered the Epizootic?

The current epizootic is dated to approximately 1994. Because it was simultaneously detected throughout the Caribbean (Nagelkerken et al. 1997a), its geographical origin cannot be established by observation. There appears to have been a much more severe, albeit more localized, epizootic in the 1980s that eradicated sea fans from the Central American coastline (Guzmán and Cortéz 1984). Although the pathogen was never identified earlier, signs of the disease included lesions similar to those in the current epizootic.

There are a few possible triggers of these aspergillosis outbreaks. One is an increase in transport of terrestrial soil or dust and associated microbes. The terrestrial source might be local, as in the case of runoff (Smith et al. 1996), or exotic, such as the atmospheric transport of the pathogen-containing dust from Northern Africa to the Caribbean (i.e., African dust hypothesis; Shinn et al. 2000; Prospero and Lamb 2003). Given the worldwide distribution of *A. sydowii* and our current inability to distinguish strains from different locations, we cannot yet confirm the source of the pathogen responsible for the epizootics.

Another possible trigger of these aspergillosis outbreaks is a change in environmental conditions. One example is increasing sea surface temperature over the last several decades. In laboratory experiments, *A. sydowii* had an op-

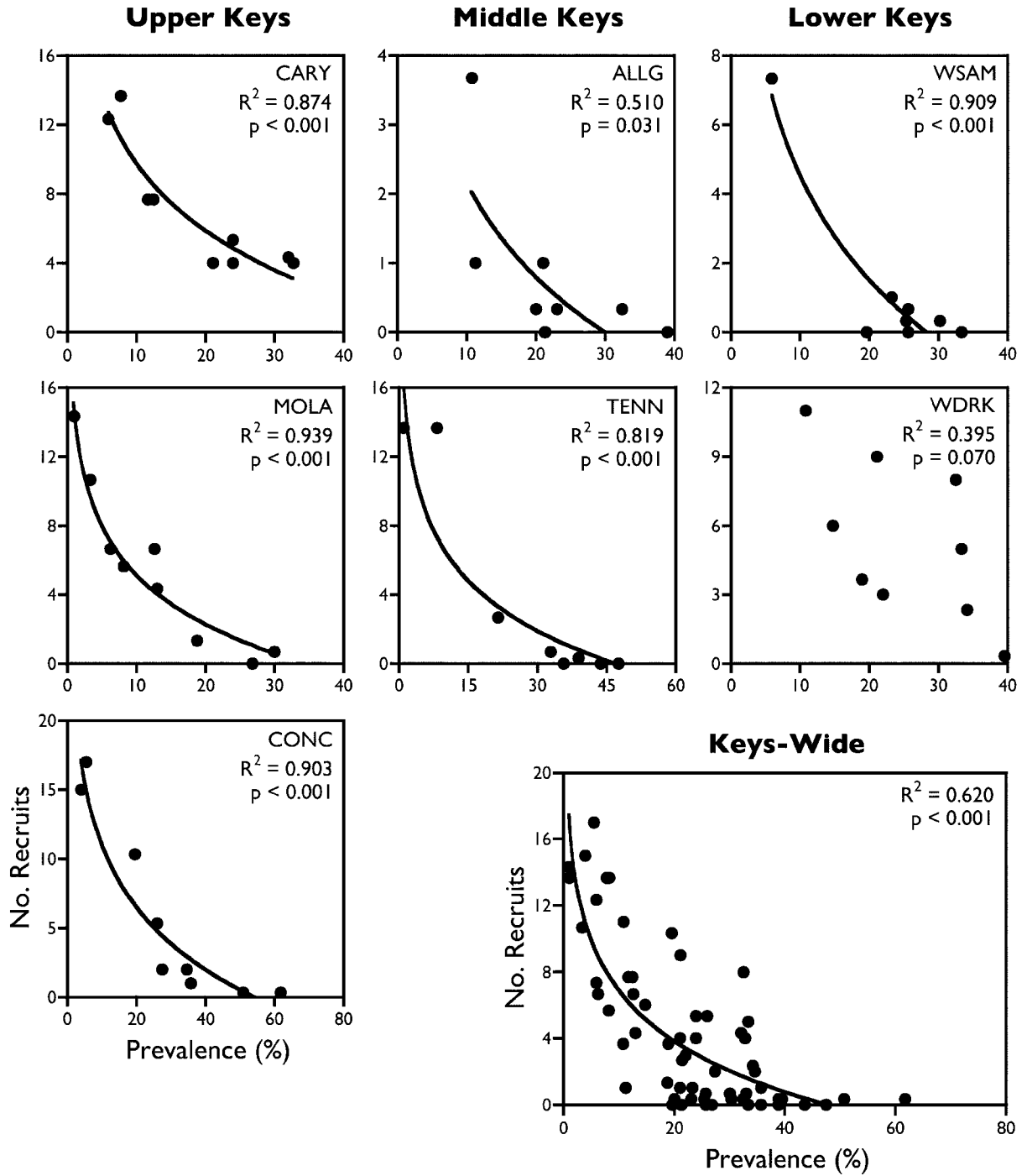


Figure 5: Relationship between aspergillosis and recruitment. On six of seven reefs, recruitment declined with disease prevalence. Data were combined for the Keys-wide analysis. Reef abbreviations as in figure 1.

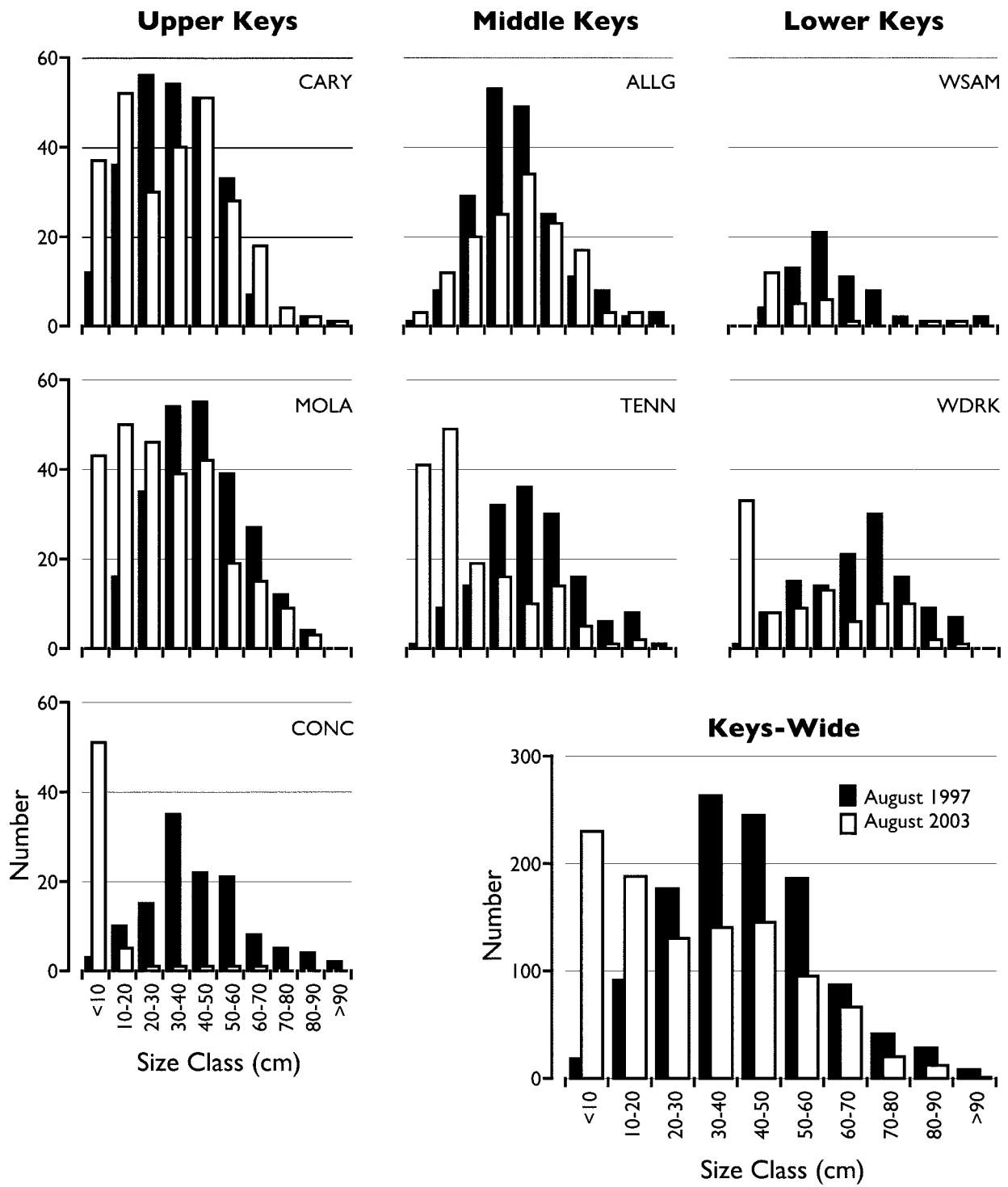


Figure 6: Changes in size class structure of sea fans between August 1997 and August 2003. Note the general decline in larger sea fans. Data were combined for the Keys-wide analysis. Reef abbreviations as in figure 1.

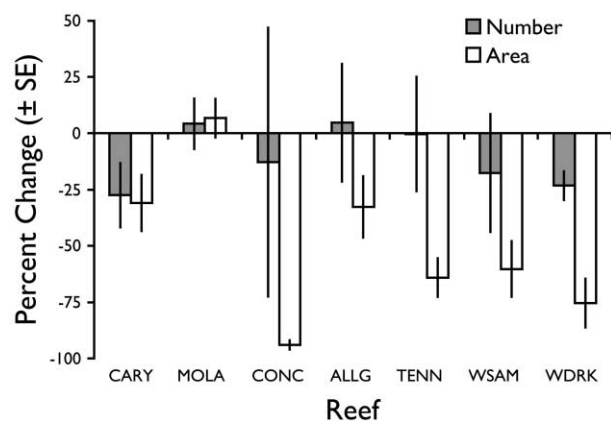


Figure 7: Impact of aspergillosis as indicated by percent changes in population size (number) and absolute sea fan tissue area between August 1997 and August 2003. Changes in tissue area result from a combination of complete and partial mortality. Reef abbreviations as in figure 1.

timal growth temperature of 30°C, corresponding to the mean summer temperature in the Florida Keys, and was less affected by host chemical defenses at 30°C than at 25°C (Alker et al. 2001). In contrast, corals exposed to 30°C water for prolonged periods display signs of stress such as bleaching (Brown 1997; Hoegh-Guldberg 1999), which appears to increase disease susceptibility (Harvell et al. 2001). It is noteworthy that warming has occurred in two relatively rapid increases during the early 1980s and 1990s (Levitus et al. 2001), coincident with the two documented epizootics affecting sea fans.

Another potential environmental trigger is the increased nutrient pollution of coastal waters (Howarth et al. 2000), including the Florida Keys (Jones and Boyer 2000; Lapointe et al. 2002). Evidence supporting the role of nitrates in facilitating aspergillosis comes from field experiments showing that nutrient enrichment increases the severity of aspergillosis (Bruno et al. 2003). Other coral diseases also seem to increase under high nutrient conditions (Kuta and Richardson 1996). Unfortunately, the lack of both long-term baseline data (environmental and disease) and an understanding of the lag time between a triggering event and an outbreak limit our ability to identify the trigger for these aspergillosis outbreaks.

What Caused the Fall of the Epizootic?

For many marine disease outbreaks, a reduction in susceptible individuals occurs rapidly (possibly concomitant with high rates of disease spread [McCallum et al. 2003]) and significantly reduces host population density. For instance, catastrophic decline of the carnivorous sea star *Heliaster* in the early 1980s occurred over a 2-week period

(Dungan et al. 1982). Similarly, significant epizootics (i.e., >50% mortality) affecting several species of sponges (Galtsoff et al. 1939), the scallop *Argopectin gibbus* (Moyer et al. 1993), and the herring *Clupea harengus* (Sinderman 1956) all lasted <6 months. It took approximately 1 year for the near elimination of the abalone *Haliotis cracherodii* (Lafferty and Kuris 1993) and the sea urchin *Diadema antillarum* (Lessios et al. 1984). White band disease nearly eradicated Caribbean acroporid corals but was of a much longer duration, lasting throughout the 1980s (Gladfelter 1982; Aronson and Precht 2001).

Aspergillosis of sea fans is a comparatively slow epizootic; it has been ongoing in the Caribbean since approximately 1994 (Nagelkerken et al. 1997a). Since its discovery in the Florida Keys in 1996, aspergillosis has only recently reached endemic (as opposed to epizootic) levels (fig. 2). The duration of aspergillosis is comparable to white band disease, both of which are of substantially longer duration than diseases affecting unitary species described above. Indeed, the extended duration of aspergillosis may result from the colonial nature of corals (i.e., the ability to endure substantial partial mortality), which can slow the rate at which hosts die. Aspergillosis also differs from the marine diseases described above in that it has not significantly decreased its hosts' density at most sites (fig. 3) because of high sea fan recruitment in recent years.

There are several plausible hypotheses for the decline of the epizootic, including changes in the environment, in pathogen input or pathogen virulence, and in host resistance. For many marine diseases, a warming ocean (Levitus et al. 2001) is predicted to increasingly favor pathogens (Harvell et al. 2002). The combination of accelerated pathogen growth and reduced host resistance at higher water temperatures is likely to result in disease outbreaks of greater frequency and impact. However, water temperatures remain relatively high in the Florida Keys, and there is no correlation between the decline phase and water temperature. Similarly, there is no indication that nutrient levels have decreased over the last 3 years. Although it remains unclear whether and what environmental conditions may have triggered the epizootic, we can reject the hypothesis that its decline is related to an improvement in nutrient level or temperature.

It is possible that the decline in the epizootic was due to decreasing pathogen input. However, this hypothesis is inconsistent with continuing trends of increasing inputs of African dust and nutrients (Howarth et al. 2000; Lapointe et al. 2002; Prospero and Lamb 2003). We have no information to address whether the pathogenicity of *A. sydowii* has declined, but we are investigating variation in virulence among strains.

A common factor causing decline of epizootic is the

reduction of susceptible individuals (Tompkins et al. 2002). This appears to be the case for sea fan aspergillosis. Antifungal secondary chemicals are an important mechanism for aspergillosis resistance in sea fans (Kim et al. 2000), and while the level of resistance itself does not appear to be related to disease intensity (measured as the product of prevalence and severity), there is a marked relationship between variance in resistance and disease intensity (i.e., lower variance in resistance at reefs with greater disease intensity; Dube et al. 2002). This suggests that there has been selection for disease resistance, especially at the more heavily impacted sites like Conch Reef (Dube et al. 2002). Theoretically, the combination of very high mortality among susceptibles and strong suppression of reproduction among infected colonies (Petes et al. 2003) should lead to intense selection for resistant sea fans. Recruitment (fig. 4) of apparently resistant sea fans during 2002 and 2003 (prevalence among colonies <20 cm height was <1% in August 2003) could have further contributed to decline of the epizootic. Similar dynamics are an important component of *Perkinsus* epizootics affecting oysters (Powell et al. 1996). Continued monitoring of these sea fan recruits will reveal whether they really are resistant to aspergillosis and thus whether the disease will continue to decline (fig. 2).

Acknowledgments

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