

THE IMPACT OF CORAL DISEASE ON THE SURVIVAL  
OF THE FLORIDA KEYS CORAL REEFS

by

CECILIA TORRES

(Under the Direction of James W. Porter)

ABSTRACT

In the Florida Keys, increases in disease abundance and decreases in coral cover were documented during the 1990s, raising concern about the contribution of disease to coral decline. The prevalence, severity, and lethality of coral diseases in the Florida Keys was quantified by following the fate of over 500 diseased colonies in 14 stations from 2002-2004, and assessing changes via digital photography. Disease prevalence ranged from 4.0-8.2%, and incidence of new infections fluctuated considerably from year to year. Between 2002-2004, disease lethality was low: 1% of the population died, and 3% suffered partial mortality from disease. Between 2002-2003, tissue loss to disease was small (0.4 m<sup>2</sup>), and monitored stations saw no significant changes in coral cover. However, unexpected long-term impacts of disease could be seen because 1) diseases targeted larger sized (more fecund) colonies, and 2) four of the most important reef building species accumulated most of the tissue loss.

**Index words:** coral reefs, Florida Keys, coral disease, tissue mortality, dark color syndrome, bleaching, white plague, white pox, cyanobacterial mat disease, Caribbean yellow band, skeletal anomaly, disease prevalence, disease incidence, disease severity, lethality.

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## TABLE OF CONTENTS

	Page
ACKNOWLEDGEMENTS .....	iv
LIST OF TABLES .....	vi
LIST OF FIGURES .....	vii
CHAPTER	
1 THE IMPACT OF CORAL DISEASE ON THE SURVIVAL OF THE FLORIDA KEYS CORAL REEFS .....	1
APPENDICES	
I .....	120
II .....	134
III .....	141
IV .....	155

## LIST OF TABLES

	Page
Table 1: Description of sites and stations selected for sampling and sampling dates.....	8
Table 2: Number of scleractinian coral colonies identified as diseased in 2002, 2003 and 2004 in 14 stations surveyed in the FKNMS.....	32
Table 3: Mean number of scleractinian coral colonies (greater than 3 cm in diameter) per m <sup>2</sup> at each of 14 stations surveyed in the FKNMS ( $\pm$ standard error).....	45
Table 4: Diseased area measurements (cm <sup>2</sup> ) based on downward-pointed overview, as well as close-up photographs, for all 14 stations surveyed in the FKNMS in 2002 and 2003.....	65
Table 5: Areas of scleractinian coral tissue loss or recovery due to disease conditions, and total net area changes at 14 stations surveyed in the FKNMS.....	67
Table 6: Prevalence, severity and lethality of diseases affecting the 4 scleractinian coral species which suffered most tissue loss during the survey.....	70
Table 7: Mean percent live scleractinian coral areas ( $\pm$ standard error) for all 14 stations surveyed in the FKNMS in 2002 and 2003.....	71
Table 8: Percent of projected live scleractinian coral areas with active disease lesions in 2002 and 2003 at 14 stations surveyed in the FKNMS.....	73
Table 9: Percent of the 2002 projected live scleractinian coral cover which was lost due to disease or recovered from disease by 2003, at 14 stations surveyed throughout the FKNMS.....	74

## LIST OF FIGURES

	Page
Figure 1: Map of the Florida Keys National Marine Sanctuary indicating the location of the selected sampling sites.....	9
Figure 2: Layout of the sampling stations.....	10
Figure 3: Photograph of the author filming a video transect by following the plastic chain on the surface of the reef from a distance of 40 cm.....	11
Figure 4: Photograph of three divers conducting scleractinian coral colony counts.....	13
Figure 5: Photograph of the author dropping weighed flags to mark diseased colonies during the inspection phase of the diseased coral survey.....	14
Figure 6: Photograph of the author checking the position of a diseased coral colony during the diseased coral survey.....	15
Figure 7: Photograph of the author relocating a colony marked as diseased during the preceding year's diseased coral survey.....	18
Figure 8: Image showing typical PointCount <sup>®</sup> for Coral Reefs display, with 30 random points displayed over each image to be identified.....	24
Figure 9: Image showing typical Image-Pro Plus <sup>®</sup> 4.5.1 display, in which tracings have been performed of all dark color syndrome disease lesions on a colony of <i>Siderastrea siderea</i> .....	29
Figure 10: Number of scleractinian coral colonies with disease signs per m <sup>2</sup> surveyed in 14 stations throughout the FKNMS in 2002, 2003, and 2004.....	33

Figure 11: Example of the potential disease condition affecting colonies of <i>Montastrea cavernosa</i> in several of the stations surveyed and referred to as “condition 1.” .....	35
Figure 12: Example of the potential disease condition affecting colonies of <i>Montastrea annularis</i> complex in several of the stations surveyed and referred to as “condition 2.” .....	36
Figure 13: Frequency of scleractinian coral disease types identified in 2002 in 14 stations surveyed in the FKNMS.....	37
Figure 14: Frequency of scleractinian coral disease types identified in 2003 in 14 stations surveyed in the FKNMS.....	38
Figure 15: Frequency of scleractinian coral disease types identified in 2004 in 14 stations surveyed in the FKNMS.....	38
Figure 16: Relative frequency of scleractinian coral species with signs of disease in 2002 in 14 stations surveyed in the FKNMS.....	40
Figure 17: Relative frequency of scleractinian coral species with signs of disease in 2003 in 14 stations surveyed in the FKNMS.....	41
Figure 18: Relative frequency of scleractinian coral species with signs of disease in 2004 in 14 stations surveyed in the FKNMS.....	42
Figure 19: Frequency of target scleractinian coral species affected by each of 11 coral disease types identified between 2002-2004 at 14 stations surveyed in the FKNMS.....	44
Figure 20: Percent of scleractinian coral colonies located in the first 20 m <sup>2</sup> of the offshore half of each station with signs of disease in 2002, 2003, and 2004.....	47
Figure 21: Percent of the population of 17 scleractinian coral species affected by disease in 14 stations surveyed in the FKNMS in 2002, 2003 and 2004.....	48

Figure 22: Status in 2003, of 237 scleractinian coral colonies identified as diseased in 2002.....	49
Figure 23: Status in 2004, of 237 coral colonies identified as diseased in 2002.....	50
Figure 24: Status in 2004, of 61 new scleractinian coral colonies identified as diseased in 2003.....	51
Figure 25: Status in 2004, of 194 scleractinian coral colonies identified as diseased with dark color syndrome in 2002 (N=155) and 2003 (N=39).....	52
Figure 26: Status in 2004, of 22 scleractinian coral colonies identified as diseased with white plague in 2002 (N=16) and 2003 (N=6).....	53
Figure 27: Status in 2004, of 46 scleractinian coral colonies identified in 2002 (N=38) and 2003 (N=8) with signs of bleaching.....	54
Figure 28: Status in 2004, of 9 colonies identified with ‘condition 1’ in 2002 (N=5) and 2003 (N=4).....	55
Figure 29: Status in 2004, of 25 scleractinian colonies marked with “unknown” disease signs in 2002 (N=16) and 2003 (N=9).....	57
Figure 30: Changes in coral tissue areas by 2003, of 237 colonies identified as diseased in 2002 (left), and by 2004 of 156 colonies identified as diseased in 2003 (right).....	59
Figure 31: Diseases affecting 127 scleractinian coral colonies which experienced tissue loss between 2002 and 2003, and 96 colonies which experienced tissue loss between 2003 and 2004.....	60
Figure 32: Disease types affecting 36 scleractinian coral colonies which died between 2002 and 2004. All colonies died 1 year after our initial disease observation.....	61

Figure 33: Disease conditions affecting 137 scleractinian coral colonies identified as diseased in 2002, and 60 identified in 2003, which experienced either no significant change in tissue area or a gain in tissue area by the subsequent survey year..... 62

Figure 34: Changes in coral area by 2003, in 155 scleractinian coral colonies marked with dark color syndrome in 2002, and by 2004, in 108 coral colonies marked in 2003..... 63

Figure 35: Differential impact of dark color syndrome on 51 colonies of *Montastrea annularis* complex (left), 182 colonies of *Siderastrea siderea* (center), and 26 colonies of *Stephanocoenia michelinii* (right)..... 64

Figure 36: Total net changes in coral tissue area due to disease at 14 stations surveyed in the FKNMS between 2002 and 2003..... 66

Figure 37: Percent of scleractinian coral tissue mortality attributed to 7 disease types in 14 stations surveyed in the FKNMS..... 68

Figure 38: Disease-related net tissue losses experienced by 13 scleractinian coral species across 14 stations surveyed throughout the FKNMS..... 69

Figure 39: Mean percent projected live scleractinian coral cover ( $\pm$  standard error) at 14 stations surveyed in the FKNMS in 2002 and 2003..... 72

Figure 40: Comparison of size class distribution of diseased colonies (all diseases during all 3 survey years) and coral population at all 14 stations surveyed in the FKNMS, and relative prevalence of disease per size class..... 75

Figure 41: Size class distribution of 2002-2004 <i>Siderastrea siderea</i> diseased colonies (all disease conditions) and total population of the species, and disease prevalence per size class.....	77
Figure 42: Size class distribution of 2002-2004 <i>Stephanocoenia michelinii</i> diseased colonies (all disease conditions) and total population of the species, and disease prevalence per size class.....	77
Figure 43: Size class distribution of 2002-2004 <i>Montastrea annularis</i> complex diseased colonies (all disease conditions) and total population of the species, and disease prevalence per size class.....	78
Figure 44: Size class distribution of 2002-2004 <i>Agaricia agaricites</i> complex diseased colonies (all disease conditions) and total population of the species, and disease prevalence per size class.....	78
Figure 45: Comparison of size class distribution of <i>Siderastrea siderea</i> colonies affected by dark color syndrome (2002-2004) and the general population for that species, and relative disease prevalence per size class.....	80
Figure 46: Comparison of size class distribution of <i>Stephanocoenia michelinii</i> colonies affected by dark color syndrome (2002-2004) and the general population for that species, and disease prevalence per size class.....	80
Figure 47: Comparison of size class distribution of total <i>Montastrea annularis</i> complex colonies affected by dark color syndrome (2002-2004) and the general population for that species, and disease prevalence per size class.....	81
Figure 48: Changes in percent live scleractinian coral cover at 14 stations in the Florida Keys between 1996 and 2003.....	87

Figure 49: Examples of colonies observed and followed through time with tissue mortality  
around the base of the colony and bisected polyps.....98

## CHAPTER 1

# THE IMPACT OF CORAL DISEASES ON THE SURVIVAL OF THE FLORIDA KEYS CORAL REEFS

## INTRODUCTION

### BACKGROUND

Coral reefs are among the most complex and biologically diverse ecosystems on the planet. They support 32 of the 34 known animal phyla, 93,000 known species of coral reef plants and animals (Reaka-Kudla *et al.* 1997; Porter and Tougas 2001) and an estimated 600,000 to 9.5 million undescribed species (Reaka-Kudla *et al.* 1997). In addition to harboring immense biological diversity, coral reefs provide significant sources of economic benefits and environmental services. They reduce coastal erosion by protecting shorelines from hurricanes and storms (Birkeland 1997; Hoegh-Guldberg 1999). They provide essential habitat for many commercially and recreationally important species of fish and shellfish, and shelter other associated ecosystems such as mangroves and seagrass beds, which are also important nursery grounds (Birkeland 1997). Coral reefs support a multibillion dollar tourism industry (Hoegh-Guldberg 1999), while providing critical sources of income to the local communities, particularly for small island nations with few exploitable resources (Porter and Tougas 2001). The

pharmaceutical industry is beginning to tap into the naturally occurring bioactive compounds among organisms found in coral reefs, which may prove to be a source of enormous biomedical potential (Birkeland 1997). Including all these resources and services, (Costanza *et al.* 1997) estimated the global annual value of coral reefs to the world economy at \$375 billion per year.

Coral reefs can only thrive in a very limited range of environmental conditions and are therefore extremely sensitive to environmental perturbations (Hallock 1997; Wilkinson 1999). Despite their importance, over the past three decades, coral reefs worldwide have experienced extensive degradation. Estimates suggest that 27% of the world's coral reefs have already been lost, with another 16% at serious risk of loss (Wilkinson 2002). Human activities lie at the very core of this decline. Increases in world population and global economic activity have coincided with the degradation of coral reefs, and in many cases there may be a direct causal relation (Wilkinson 1999). Major direct anthropogenic influences include increased sediment loading from terrestrial activities, organic and inorganic pollution from agricultural, urban and industrial discharges and consequent eutrophication, and overexploitation of fisheries, often with the use of extremely destructive techniques. Other increasingly significant threats include global climate change, which contributes to coral bleaching events, and coral disease. Although the latter are often considered non-anthropogenic factors, it is becoming ever more evident that these may be exacerbated by anthropogenic effects.

Many of the aforementioned threats to coral reefs can be seen at work in the Florida Keys. Over the past two decades, water quality in the south Florida reef system has been steadily declining. For instance, Sanctuary-wide, phosphorous has increased significantly, as have chloryphyll *a* concentrations and water turbidity in several places (Lapointe *et al.* 1994; Boyer and Jones 2002). There are currently over 30,000 on-site sewage disposal systems (septic tanks,

injection wells and cesspits) in the Florida Keys (Lapointe *et al.* 1990; Griffin *et al.* 1999; Kruczynski and McManus 2002). The majority of these are located in high density zones where they are often separated from the marine receiving waters by very short distances (25 m) (Lapointe *et al.* 1990). Studies have shown that nutrients from these sewage systems are seeping into nearshore surface waters (Lapointe *et al.* 1990; Lapointe and Clark 1992; Lapointe and Matzie 1996; Lapointe and Matzie 1997; Corbett *et al.* 1999) and have been correlated with a decline in coral cover (Porter and Meier 1992; Ogden *et al.* 1994). Human enteric bacteria and viruses have been found to be prevalent in nearshore, offshore and canal waters in the Florida Keys (Lapointe *et al.* 1990; Paul *et al.* 1995a; Paul *et al.* 1995b);(Paul *et al.* 1997; Griffin *et al.* 1999) as well as on coral surfaces (Lipp *et al.* 2002). Additional nutrients are also making their way into the waters of the Florida Keys from “upstream” terrestrial run-off. Human alteration of the south Florida watershed (Kissimmee-Okeechobee-Everglades drainage basin) for human settlement and large-scale agriculture has lead to a downstream nitrogen-overload problem, causing biotic phase shifts in the Florida Bay, algal blooms and eutrofication of the Florida Keys coral reef waters (Fourqurean and Robblee 1999; Lapointe *et al.* 2002). Paralleling this deterioration of water quality, it is becoming increasingly apparent that coral diseases are also emerging as a very significant threat to the reefs of the Florida Keys.

Over the past decade, reports of new and emerging coral diseases, especially in the Caribbean region, have emphasized the notion that an increase in coral diseases is seriously contributing to reef degradation (Richardson 1998). These emerging diseases, often epizootic in distribution, continue to appear with increasingly greater frequency and over wider geographical areas (Santavy and Peters 1997; Goreau *et al.* 1998; Hayes and Goreau 1998; Richardson 1998; Harvell *et al.* 1999; Williams and Bunkley-Williams 2000; Porter *et al.* 2001; Porter and Tougas

2001; Rosenberg and Ben-Haim 2002; Harvell *et al.* 2004). As of 2002, a total of 18 coral diseases had been described worldwide (13 of them reported since the mid-1990s), affecting at least 150 species of scleractinian, gorgonian and hydrozoan corals (Sutherland *et al.* 2004).

It has been suggested that mortality from coral diseases may have the potential of becoming more detrimental to the survival of coral reefs than all other threats combined (Hayes and Goreau 1998). Unfortunately, characterization of the causal agents, prevalence and consequences of coral diseases remains very limited. Part of the gap in our understanding of coral diseases stems from the fact that most studies are focused on describing observed disease epizootics (Santavy *et al.* 2001). This implies that quantitative observations are restricted to relatively few and small geographic areas, and often focus on the incidence of one single disease, during an observation period of limited duration (Dustan 1977; Gladfelter 1982; Edmunds 1991; Kuta and Richardson 1996; Bruckner and Bruckner 1997; Richardson *et al.* 1998a; Richardson *et al.* 1998b). Hence, little information exists concerning continuing baseline patterns of disease prevalence over large geographic extents and their influence on coral reef dynamics.

## OBJECTIVES

During a study conducted by Porter *et al.* (2001) as part of the EPA/NOAA Coral Reef Monitoring Project (CRMP), the Florida Keys National Marine Sanctuary saw a 404% increase in the number of locations exhibiting disease (from 26 to 131 stations) and a 218% increase in the number of coral species exhibiting disease (from 11 to 36 species) between 1996 and 1998. This increase in disease incidence paralleled a 37% decline in living coral cover over the same time period and geographic extent (Porter *et al.* 2002). Following documentation of these

massive coral losses over such broad geographic extents, attention was shifted from basic monitoring to detect changes in the reef health, to attempts at identifying and understanding the stressors producing this change. As part of this effort, the present study was designed with the objective of understanding the relationship between coral disease and observed coral reef change in the Florida Keys.

It is now widely recognized that coral disease epizootics can have devastating effects on coral populations. White band disease outbreaks decimated populations of staghorn and elkhorn corals *Acropora cervicornis* and *A. palmata* throughout the Caribbean in the late 1970s (Gladfelter 1982) and late 1980s (McClanahan and Muthiga 1998; Richardson 1998). Dustan (1977) documented the first white plague epizootic in coral reef populations off Key Largo, and reported high mortality among several coral species. Another white plague epizootic in the middle Florida Keys was reported in 1995, targeting a large number of coral species and with mortality rates up to 38% among colonies of *Dichocoenia stokesii*, the most affected species (Richardson *et al.* 1998b). Aspergillosis of sea fans, caused by the terrestrial fungus *Aspergillus sydowii* rapidly swept through reefs of the Florida Keys and the Caribbean in 1995 and 1996 causing mass mortalities (Smith *et al.* 1996; Nagelkerken *et al.* 1997a; Nagelkerken *et al.* 1997b; Slattery 1999). In the late 1990s, outbreaks of white pox disease decimated populations of *A. palmata* in the Florida Keys, where losses averaged 87% or greater (Miller *et al.* 2002; Patterson *et al.* 2002; Sutherland *et al.* 2004). The present study attempted to move beyond the type observations obtained during coral disease epizootics and quantify, in a more generalized fashion, the baseline abundance and distribution of all occurring diseases, as well as their impact on the reef, in a larger geographic area over a longer time scale.

The objectives of this study can be described as two-fold: 1) to quantify the extent of coral disease in the FKNMS and determine the prevalence of various diseases as well as their target species; and 2) to assess the impact such diseases are having on the health of coral populations throughout the FKNMS. To answer the first question, the prevalence of coral diseases of all types and on all scleractinian corals was surveyed at 14 stations (different reef types) throughout the FKNMS. This survey was conducted not only on a large geographic scale but also on a multi-annual scale. To achieve the second objective, it was necessary to determine what occurs to the coral colonies once they become infected with disease. Do they recover? Do they lose tissue? If so, is it a partial loss of tissue, or does disease lead inevitably to the death of the entire colony? Which syndromes seem to be more harmful to the coral, and which cause less damage? Which coral species seem to be more resistant to diseases? Are certain coral size classes more susceptible to become infected than others? To answer these questions the fate of individual diseased scleractinian coral colonies was followed over a three year time period, and changes to their health were quantitatively assessed by means of digital photography analysis.

## METHODS

### SITE SELECTION AND SAMPLING SCHEDULE

The present study was conducted within the scope of the Coral Reef Monitoring Project (CRMP). CRMP methodology involves yearly sampling of 40 different sites selected by stratified random EPA E-Map procedures (Overton *et al.* 1991) within the boundaries of the

Florida Keys National Marine Sanctuary. The sites include: a) offshore shallow and b) deep reefs, c) patch reefs and d) hardbottom sites. Each site is comprised of 2 to 4 stations, the first of which was selected by starting at the randomly generated latitude and longitude and swimming to the closest suitable habitat. Remaining stations were selected in adjacent habitat a minimum of 5 meters apart. Seven of the 40 CRMP sites were chosen as study sites for the present survey. Offshore deep sites were excluded from the disease survey due to depth and diving bottom time limitations in relation to the time requirements for completing survey work at each station. Hardbottom sites were also dropped due to the low percent living coral cover on this habitat type. Fourteen stations were selected among 7 sites including 3 offshore shallow reefs, and 4 patch reefs (Table 1). The sites are distributed along the Upper, Middle and Lower Keys as shown in Figure 1.

Sampling was conducted once a year during the summer months of 2002, 2003 and 2004 (Table 1). Previous disease surveys have suggested that seasonal differences exist in the incidence and prevalence of coral diseases (Rutzler *et al.* 1983; Kuta and Richardson 1996; Santavy and Peters 1997). During this survey, logistical considerations prevented a more frequent sampling schedule, which in turn precluded the study from drawing conclusions regarding patterns of seasonality in rates of coral disease infection or intensity of disease sign expression.

Table 1. Description of sites and stations selected for sampling and sampling dates.

Site Name	Station Number	Location	Habitat Type	Latitude	Longitude	Depth	Station Length	Sampling Dates
Grecian Rocks	1	Upper Keys	Offshore shallow	25° 06.450'	80° 18.410'	8'- 11'	22.80 m	7-16-2002 8-19-2003 6-15-2004
Grecian Rocks	2	Upper Keys	Offshore shallow	25° 06.450'	80° 18.410'	9'- 11'	23.20 m	7-16-2002 8-19-2003 6-15-2004
Porter Patch	1	Upper Keys	Patch	25° 06.1899'	80° 19.4586'	15'	24.40 m	7-25-2002 8-13-2003 6-16-2004
Porter Patch	2	Upper Keys	Patch	25° 06.1899'	80° 19.4586'	14'	23.00 m	7-25-2002 8-13-2003 6-16-2004
Tennessee	2	Middle Keys	Offshore shallow	24° 45.1621'	80° 45.4696'	18'- 21'	23.00 m	6-22-2002 5-06-2003 6-08-2004
Tennessee	3	Middle Keys	Offshore shallow	24° 45.1621'	80° 45.4696'	20'- 21'	22.25 m	6-22-2002 5-06-2003 6-08-2004
West Turtle Shoal Patch	3	Middle Keys	Patch	24° 41.9572'	80° 58.0127'	16'- 24'	22.00 m	7-10-2002 5-04-2003 6-09-2004
West Turtle Shoal Patch	4	Middle Keys	Patch	24° 41.9572'	80° 58.0127'	18'- 22'	21.50 m	7-10-2002 5-04-2003 6-09-2004
Western Sambo	1	Lower Keys	Offshore shallow	24° 28.7708'	81° 43.0293'	11'- 14'	22.50 m	8-07-2002 8-16-2003 7-26-2004
Western Sambo	2	Lower Keys	Offshore shallow	24° 28.7708'	81° 43.0293'	12'- 17'	24.00 m	8-07-2002 8-16-2003 7-26-2004
Cliff Green Patch	3	Lower Keys	Patch	24° 30.216'	81° 46.059'	22'- 25'	20.80 m	8-11-2002 7-15-2003 7-29-2004
Cliff Green Patch	4	Lower Keys	Patch	24° 30.216'	81° 46.059'	21'- 23'	20.50 m	8-11-2002 7-15-2003 7-29-2004
Jaap Patch	1	Lower Keys	Patch	24° 35.1421'	81° 34.9568'	7'- 9'	24.00 m	8-09-2002 7-18-2003 7-30-2004
Jaap Patch	2	Lower Keys	Patch	24° 35.1421'	81° 34.9568'	7'- 9'	24.10 m	8-09-2002 7-18-2003 7-30-2004

It should be noted that, time limitations and logistical considerations prevented full completion of the diseased coral survey at West Turtle Shoal Patch Reef Station 4 in 2002 and 2003. During those 2 years, only the offshore half of the station was surveyed for disease.

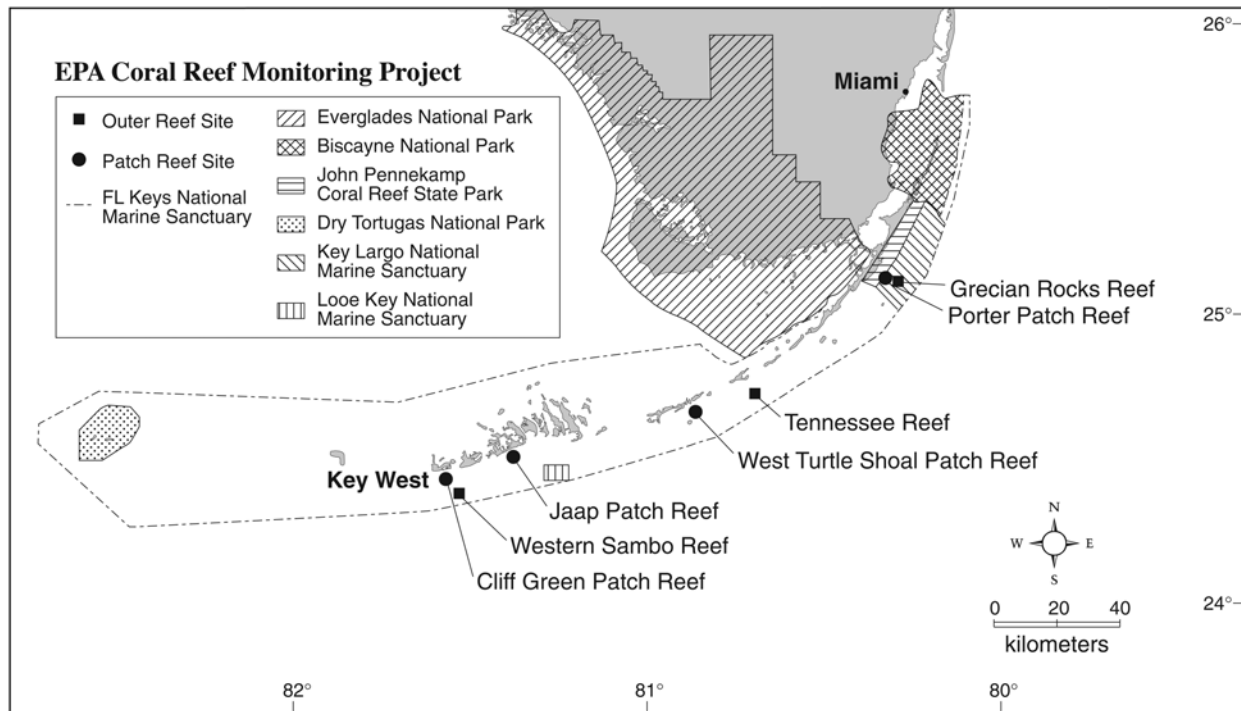


Figure 1. Map of the Florida Keys National Marine Sanctuary indicating the location of the selected sampling sites.

This study followed the fate of individual diseased scleractinian coral colonies during a three year period. Since observations on the condition of each colony were recorded only once per year, the following assumption was made: a coral colony which exhibited signs of disease at a particular point in time, and which during subsequent observations indicated loss of tissue in the same areas where the disease lesions were previously located, was considered to have experienced tissue mortality due to disease. To date, the causal agents and mechanisms of pathogenesis of most coral diseases remain unknown. Furthermore, no tools are yet available for the effective field diagnostic of a diseased state in many corals. In the context of this study, since the progressive death of the tissue was not witnessed on a weekly or even monthly basis, the

relation between diseased tissue and subsequent tissue death is merely a correlation and not, in fact, a causal relation.

## FIELD METHODS

### *CRMP Stations*

Each station consisted of an area approximately 2 m wide by 22 m long, permanently delineated by 1 inch square stainless steel stakes at either end of the station length. Stations are directed perpendicularly to the shoreline, with the stake furthest from the shoreline referred to as “offshore”. During sampling, a 2 meter long aluminum pole is placed over each of the two steel stakes marking the endpoints of the station. Three fiberglass tapes are stretched between the poles – one from each extremity of the poles and a third one down the center of the station – to delimit the station and the transects (Figure 2).

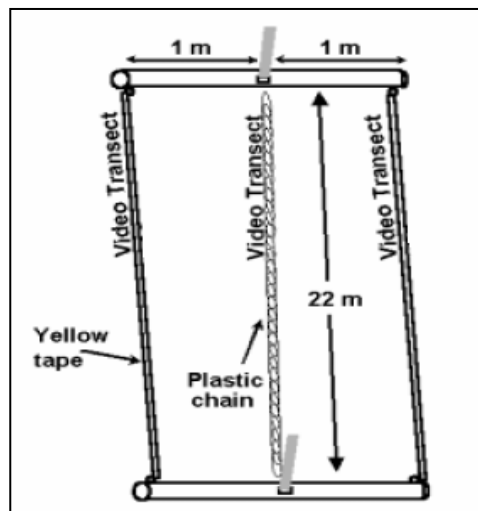


Figure 2. Layout of the sampling stations. The station is a 2 x 22 m rectangle, consisting of 3 parallel transects. Two steel stakes mark the endpoints of each station.

## Videography

The fiberglass tapes were used as a reference to carefully lay down a plastic chain on the surface of the reef, directly underneath each of the 3 transect lines, to serve as a guide for videography (Figure 2). The fiberglass tapes were subsequently removed. Video sampling was conducted using a Sony TRV 900 4-mm digital video camera. The camera was pointed straight downward and was maintained at a distance of 40 cm above the reef surface with the aid of a convergent laser light system (Figure 3). At this distance, the field of view is approximately 40 cm wide. Three parallel video transects approximately 22 m long were recorded at each station. Filming was conducted at a constant swim rate of about 4 m per minute, yielding approximately 9000 video frames per transect (Porter *et al.* 2002).

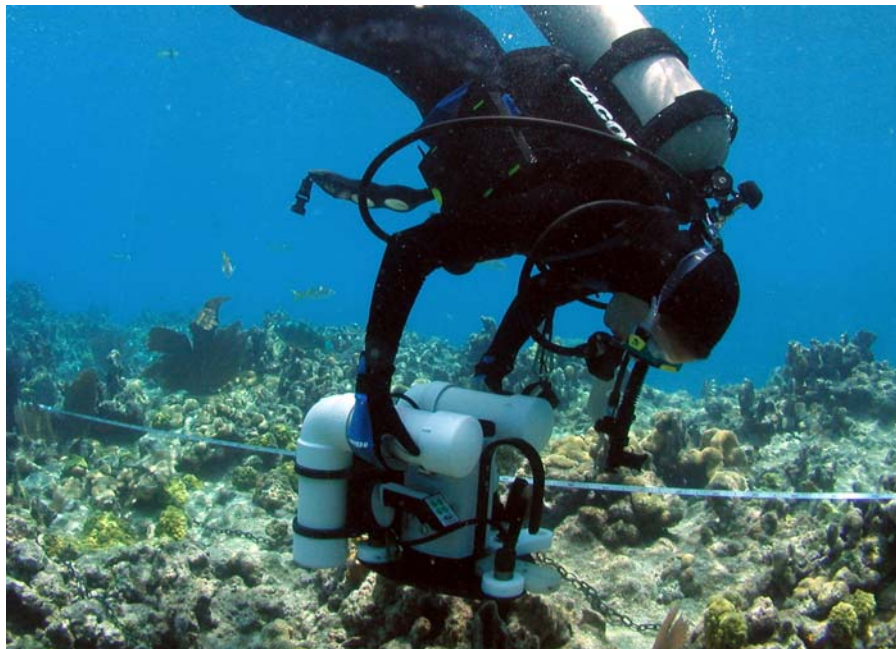


Figure 3. Photograph of the author filming a video transect by following the plastic chain on the surface of the reef from a distance of 40 cm.

### *Scleractinian Coral Colony Counts and Size Classes*

The purpose of this survey was to obtain an approximate count of the number of scleractinian coral colonies per area, with an estimate of their size. The survey required the attention of a minimum of two divers in order to obtain at least two replicate counts. It was conducted within the first 10 m of each station, from the offshore stake towards the center of the station. The area counted included the entire 2 m width of the station, for a total area of 20 m<sup>2</sup>. Two fiberglass tapes or polypropylene ropes were stretched tightly from pole to pole to delineate the station. Each diver used a 1 m × 1 m thin aluminum frame, subdivided by an elastic rope into four 0.5 m × 0.5 m quadrats to aid with the counts (Figure 4). The divers started by placing the frames side by side against the aluminum pole at the beginning of the station. Each diver counted the number of scleractinian coral colonies inside his/her quadrat. Then divers switched positions with each other and counted the colonies in the adjacent quadrat. The frames were then flipped over in the direction of the station's center and the new quadrat areas counted. The process was repeated 10 times. Each time a diver entered a colony onto the underwater datasheet, it was identified to species and classified within one of 4 size categories based on its maximum diameter: 0-3 cm, 3-10 cm, 10-50 cm and > 50 cm. Counters had stainless steel rulers available underwater to aid with colony size measurements. All quadrat counts were then totaled for each diver for the entire 20 m<sup>2</sup> area.



Figure 4. Photograph of three divers conducting scleractinian coral colony counts. The station was delineated by yellow propylene ropes and the divers used two 1 m × 1 m aluminum frames to mark the quadrats while counting.

#### *Diseased Scleractinian Coral Photographic Survey*

##### a) Inspection Phase

Two fiberglass tapes or polypropylene ropes were stretched tightly from pole to pole to delineate the station. This survey required the attention of at least two divers. In 2002 and 2003, 3 trained divers were available to aid with this survey, while in 2004 only 2 divers carried out all field components of the survey. The observers conducted a simultaneous inspection of the entire area of the station (approximately 2 m × 22 m) in search of scleractinian coral colonies exhibiting signs of disease. Weighed flags consisting of fluorescent flagging tape attached to 2.5 oz weights were placed to the side of each and all diseased colonies discovered inside the boundaries of the station (Figure 5). Due to time concerns and in the interest of consistency, only

scleractinian coral colonies with a minimum diameter of 3 cm were included in the survey. Only coral colonies exhibiting clear and unequivocal signs of disease were flagged.



Figure 5. Photograph of the author dropping weighed flags to mark diseased colonies during the inspection phase of the diseased coral survey.

b) Photography Phase

Beginning at the offshore stake of the station and swimming towards the center of the station, the observers examined each coral colony marked as diseased and entered on underwater data sheets the identity of the coral species, the name of the condition affecting the colony (if clearly identifiable), and the location of the colony. Colony location was referenced by: a) its distance from the stake, as measured with a fiberglass tape attached to the station's stainless steel

stake, and b) its direction or position relative to the width of the station. In 2002, this direction was measured in degrees with an underwater digital compass positioned at the stake and aimed directly towards the flagged colony (Figure 6). This technique required too much swimming back and forth between the stake and the diseased colonies. In subsequent years, the position was instead recorded as falling into one of 5 categories in relation to the width of the station as seen by someone positioned at the stake and facing the station: a) left, b) center-left, c) center, d) center-right and e) right. During the first year of the survey, an underwater clapperboard was used to enter the distance and direction of each colony for photography and colony tracking purposes. This was later found to be an unnecessary step and the clapperboard was not used in subsequent years.



Figure 6. Photograph of the author checking the position of a diseased coral colony during the diseased coral survey. The diver is positioned at the station's stake, aiming the digital compass in the direction of the coral colony of interest.

Diseased colony photographs were shot using an Olympus C-4040 digital camera fitted inside a Light and Motion Tetra 4000 underwater housing, with one Nikonos SB 105 underwater strobe light. All diseased colonies were photographed twice. The first photograph consisted of a downward-looking shot taken from approximately 40 cm directly above the coral. This angle and distance were intended to mimic the view obtained by the video camera transects in order to be able to relate data on the coral colony's projected surface area obtained from these two surveys. The distance was maintained with the aid of a 40 cm stainless steel wand mounted on the camera housing. In some instances, the size of the coral colony was too large to be included in one photograph. Large colonies required photographing from a greater distance than 40 cm above in order to incorporate the entire colony in the photograph. At its extremity, the removable stainless steel wand included a 2 cm × 2 cm square scale which was incorporated in each image in order to subsequently calibrate photographs for area measurements.

The wand was removed before shooting the second photograph, which in turn was a close-up of the diseased area. This photograph was taken from the angle which provided the greatest coverage of the affected area. Each close-up photograph included a stainless steel plate with six 3 cm × 3 cm black and white squares which served as the graduated scale for area calibrations. When the affected area was extensive, several close-up photographs of various sections were taken in order to create a composite of the area of interest. Each shot was reviewed using the instantaneous viewing feature on the digital camera before moving on to the next diseased colony. When all marked colonies in the offshore half of the station were photographed, the procedure was repeated for the onshore half of the station, with all distance and position measurements referenced to the onshore stake.

c) Diseased Colony Relocation

During the second and third years of the disease survey, the underwater protocol varied slightly from the aforementioned methods, since all diseased colonies photographed the previous year had to be relocated and newly diseased colonies identified as such.

Following the inspection phase, when all diseased colonies were marked with weighed flags, the observers searched for the colonies photographed the previous year based on their distance and direction from the stake. This information was available to the divers on underwater data sheets. Unless a colony photographed the previous year was no longer affected by disease or had since died, all colonies to be relocated were already marked during the inspection phase. When relocating a colony, the observers confirmed its correct identity by visual comparison to photographs taken the previous year, available to the divers on underwater sheets (Figure 7). When the colony of interest appeared to be “missing”, its death was established definitively by: a) location of the skeleton or b) confirmation of its absence relative to other identified neighboring features (other coral colonies, soft corals, sponges, *etc.*) observable on the previous year’s photograph. All relocated colonies were photographed with careful attention to reproducing, as accurately as possible, the angles and dimensions of the photographs taken the previous year.

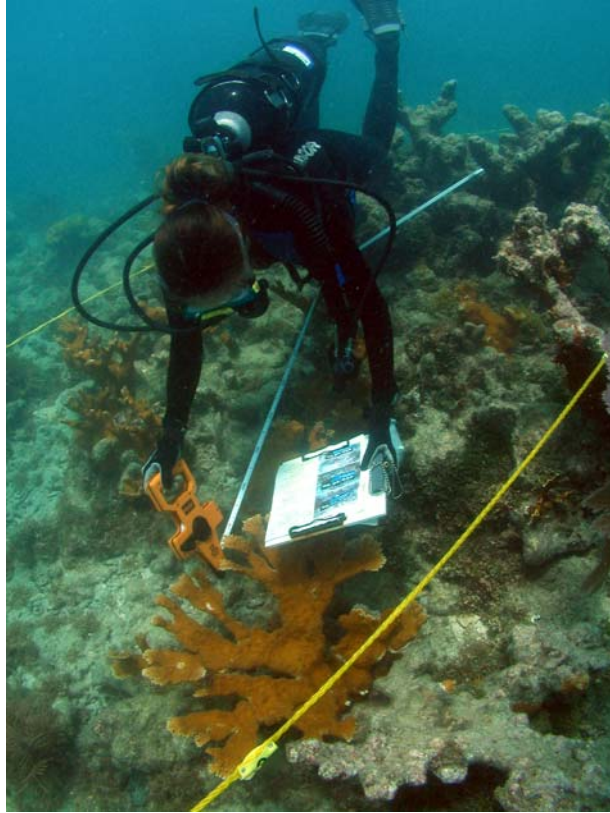


Figure 7. Photograph of the author relocating a colony marked as diseased during the preceding year's diseased coral survey. The diver was able to reference the distance, location and photograph of the diseased colony on an underwater data sheet.

Flagged colonies confirmed not to match reference coordinates for previous-year colonies were recorded as newly diseased colonies, identified by coral species and condition name. Their distance to the stake and position were measured, and they were photographed as described above.

#### d) Quality Assurance

During the inspection phase, the 3 observers (2 in 2004) surveyed the area of the station in search of disease in an independent manner. Observers were not allowed to remove weighed

flags dropped by other divers. During subsequent examination of each flagged colony, all three swimmers confirmed the identity of the coral and the identity of the disease or syndrome and resolved by consensus the inclusion of the colony in the survey and database.

e) Disease Identification

During field sampling, colonies were only marked as diseased if the lesions were active or if mortality was recent enough that the exposed bare coral skeleton had not yet been colonized by algae and remained bright white. Guidelines for identification of coral diseases or syndromes followed descriptions of each condition as published in the literature. Diseases were classified into the following categories:

**Bleaching**

Disease is defined as any impairment (interruption, cessation, proliferation, or other disorder) of vital body functions, systems, or organs (Stedman 2000). Bleaching, or the loss of symbiotic zooxanthellae from host coral tissue, has traditionally not been considered a disease, but rather a stress response resulting from changes in environmental conditions. Coral bleaching has recently also been associated with 2 bacterial pathogens of the genus *Vibrio* (Kushmaro *et al.* 1996; Kushmaro *et al.* 1997; Kushmaro *et al.* 1998; Rosenberg *et al.* 1998; Kushmaro *et al.* 2001; Ben-Haim and Rosenberg 2002; Ben-Haim *et al.* 2003a; Ben-Haim *et al.* 2003b), though this biotic origin has not, as yet, been shown in Caribbean coral species. Most often, once the abiotic stressor is removed, the coral regains its symbionts, resulting in full recovery. However, in certain instances, bleaching may result in coral mortality. Furthermore, bleaching has also been shown to reduce reproductive output, skeletal growth, calcification rates, and nutrition

(Porter *et al.* 1989; Brown 1997). For this reason, in the context of this study, bleaching was considered as a disease.

### **Caribbean yellow band disease**

Also known as yellow blotch and yellow band, this condition is characterized by circular to irregular patches or bands of discolored or yellow translucent coral tissue (Santavy *et al.* 1999; Cervino *et al.* 2001; Sutherland *et al.* 2004). Lesions are most common on the uppermost surfaces of the coral colony (Santavy *et al.* 1999). A patch of exposed skeleton is often at the center of each lesion or the edges of the band.

### **Cyanobacterial mat disease**

This name was used to group conditions known as black band disease and red band disease. Both syndromes are characterized by darkly pigmented microbial mats, which form a band (1 to 30 mm wide and 1 mm thick) which separates living tissue from dead skeleton (Rutzler *et al.* 1983; Carlton and Richardson 1995). These 2 syndromes only appear visually distinct by subtle differences in the coloration of the microbial mat. Given suggestions that red band disease may not be a distinct disease, but rather a variation of black band disease (Sutherland *et al.* 2004), no distinction was made between the two in this survey.

### **Dark color syndrome**

Also known as dark spots disease, dark band syndrome, and purple spots and purple band, the term dark color syndrome was used to refer to afflictions producing a darker than normal pigmentation response in the host tissues (Weil 2004). These syndromes are

characterized by irregularly shaped dark spots of purple or maroon coloration, or dark or purple bands advancing from the edges to the center of the colony (Sutherland *et al.* 2004). Sometimes the condition can be associated with a depression of the coral surface (Garzon-Ferreira and Gil-Agudelo 1998; Cervino *et al.* 2001). All aforementioned afflictions were grouped under the name “dark color syndrome.”

### **Skeletal anomaly**

Skeletal anomalies include tumors, galls, nodules and other abnormalities of coral tissue and skeleton (Sutherland *et al.* 2004). Tumors result from neoplasia (uncontrolled cell proliferation), hyperplasia (nonneoplastic controlled cell proliferation) and hypertrophy (nonneoplastic increase in cell size)(Sinderman 1990), though in this study, no distinction was made among them. Skeletal anomalies appear as lesions on the surface of corals, in which the shape of the polyps, the size of the tissue or skeleton, or the color of the tissue are markedly different from the surrounding skeleton or tissue.

### **White band disease**

Two types of white band disease have been described to date. Both exclusively affect branching acroporid corals (Sutherland *et al.* 2004) and are characterized by a white band of recently dead skeleton adjacent to a necrotic front of normally pigmented living tissue (Gladfelter 1982; Peters *et al.* 1983; Sutherland *et al.* 2004). Both develop at the base of the coral colony and progress upward towards the branch tips in a concentric ring (Gladfelter 1982; Sutherland *et al.* 2004), though white band type II can also develop at the tips of branches and progress downward (Ritchie and Smith 1998; Sutherland *et al.* 2004). White band type II is also

distinguished from type I by a band (2 to 20 cm wide) of living bleached tissue separating the skeleton from the living tissue, though sometimes the necrotic margin catches up to the normal tissue. When this occurs type I cannot be distinguished from type II (Ritchie and Smith 1998; Sutherland *et al.* 2004). For this reason, in the context of this study, no distinction was made between white band types.

### **White plague**

Three different types of white plague have been reported to date (White Plague I, II and III). All involve a line of tissue loss where tissue is adjacent to recently denuded skeleton (Sutherland *et al.* 2004). Distinctions are made between the 3 conditions mainly based on the speed at which the tissue loss progresses. White plague type II may sometimes show a narrow band (2 to 3 mm) of bleached tissue separating healthy tissue and dead skeleton, though this is not always the case. Also, White plague type III appears to affect exclusively large colonies of *Colpophyllia natans* and *Montastrea annularis*. During this study, no attempt was made to distinguish between white plague types.

### **White pox**

Also known as acroporid serratiosis (Patterson *et al.* 2002) and patchy necrosis (Bruckner and Bruckner 1997), white pox only affects *Acropora palmata* colonies. It is characterized by irregularly shaped white patches of recently dead skeleton surrounded by a necrotic front of normally pigmented living tissue (Sutherland *et al.* 2004). Lesions can develop simultaneously on all surfaces of the coral (Patterson *et al.* 2002). Because white pox lesions may sometimes be extremely difficult to distinguish from predation scars produced by the corallivorous snail

*Coralliophila abbreviata*, even in the absence of snail specimens on the coral colony, lesions located at the base of the colony or connected to the base of the colony by areas of dead tissue were not included in the survey.

## ANALYSIS METHODS

### *Video Image Analysis*

Due to time constraints, the subsequent analysis of video images was only conducted for station videos taken in 2002 and 2003. Still images (JPEG and Bitmap) were created from the video tape for analysis. From this library of images, frames were selected that abutted but did not overlap by more than 15% with the previous image (Porter *et al.* 2002). Approximately 60 abutting frames formed a complete mosaic of each of the three transects in a station. Each image included an area approximately 30 cm high by 40 cm wide.

Image analysis was conducted using a custom software application, PointCount for Coral Reefs<sup>®</sup>, developed specifically for the CRMP. This software provides a repeatable means for analyzing video images utilizing random point methodology. When each image was opened, the software was set to automatically display 30 random points over the image (Figure 8). This number of random points was selected based on a Patterson (1997) study which determined that the optimum number of random points per image necessary to precisely detect change in live coral cover over time was 35 for images taken from a distance of 1 meter from the reef surface, while as few as 17 points would give a reasonably accurate result. Patterson hypothesized that the number of points could be reduced if the distance to the reef surface was reduced. For the purpose of this study, only points which fell on scleractinian corals were identified by species,

leaving all other points blank (Figure 8). The software has a point-and-click feature which relays the identification data to a spreadsheet as a comma-separated value file (\*.csv), which was analyzed using Microsoft Excel.

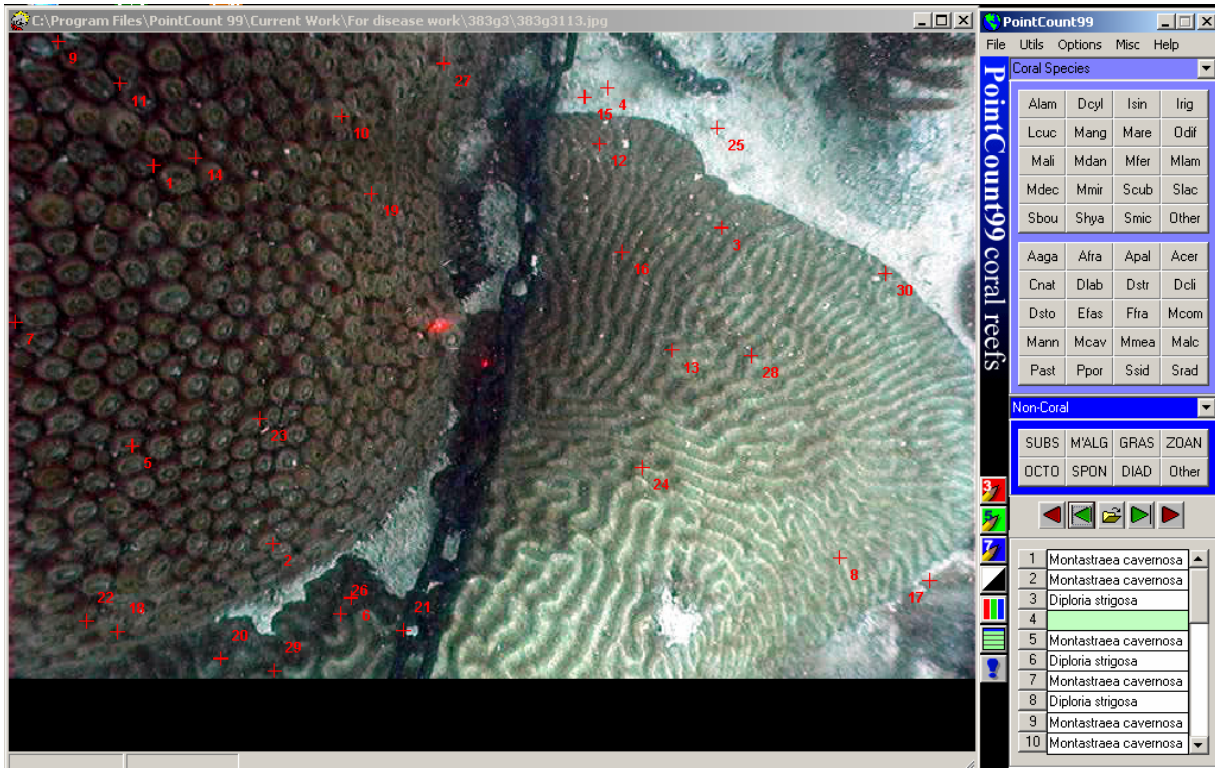


Figure 8. Image showing typical PointCount<sup>®</sup> for Coral Reefs display, with 30 random points displayed over each image to be identified.

### *Percent Cover of Scleractinian Coral Species*

The percent scleractinian coral cover of a given image was calculated as the number of points falling on scleractinian coral divided by the number of points counted for that frame. Any points which fell either before or after the station's end poles, in frames at the beginning and end of each transect, or on the plastic chain were not counted. Percent cover of scleractinian coral

species for a station was calculated as the mean of percent scleractinian coral covers for all images analyzed for a given station.

#### *Statistical Analyses – Percent Cover Data*

All statistical analyses were performed using JMP<sup>®</sup> software. A Shapiro-Wilk goodness of fit test was conducted to determine whether data for percent scleractinian coral cover for all stations in 2002 and 2003 had a normal distribution. Since the test determined that the study data were not normally distributed ( $p < 0.0001$  for all 14 station in 2002 and 2003) (Appendix I), non-parametric tests were used to compare 2002 and 2003 data concerning changes in percent scleractinian coral cover. In order to determine if there were significant differences in the mean percent scleractinian coral cover for each station between 2002 and 2003, the difference between the two means was tested using the Wilcoxon's rank-sum test.

#### *Scleractinian Coral Colony Counts and Colony Sizes*

These data were intended for use in two manners: 1) to relate the prevalence of disease at each station to the population of various species present at that station, and 2) to relate the prevalence of disease on colonies of various size classes to the size class distribution of the population, in order to determine whether certain coral size classes are more susceptible to become infected than others.

Because precise field counts of coral colonies are known to be difficult to obtain, a certain amount of variation between observers was expected from this data. However, preliminary analyses of the data indicated considerably large variability in colony counts between observers. High variability was also encountered in colony counts between years. This

variability between years was often greater than what could conceivably be attributed to natural processes of coral settlement, growth and death. Some of the inconsistencies stemmed from species identification errors, others from the difficulty in discerning, for certain coral species such as *Montastrea annularis*, distinct colony boundaries, and yet others from variability in the visual assessment of colony size.

Consequently, individual counts could not be used directly as initially recorded. Instead, the data were averaged in the interest of reducing some of this variability. Therefore, instead of relating each year's disease data to that year's corresponding coral colony counts, high and low values were removed and data for all three years was compiled to obtain the average coral colony count for the period between 2002 and 2004. Disease data for each year was then related to an average population for the overall period.

#### *Statistical Analyses – Colony Sizes*

ChiSquared ( $G^2$  Likelihood-Ratio) analyses were performed to determine whether significant differences exist between the size class distribution of diseased colonies and that of all colonies in the population.

#### *Coral Species Identifications*

It is important to mention that due to inconsistencies in the literature (Vaughn 1901; Squires 1958; Wells 1973; Weil and Knowlton 1994; VanVeghel *et al.* 1996), and difficulties in the identification of certain species, both in the field and as well as on video images, for the purpose of this study, several groups of closely related forms were aggregated into complexes. The species *Agaricia agaricites*, *Agaricia carinata*, *Agaricia danai*, and *Agaricia purpurea* were

all listed as “*Agaricia agaricites* complex” (Wheaton *et al.* 1996). “*Montastrea annularis* complex” consisted of the species *Montastrea annularis*, *Montastrea faveolata*, and *Montastrea francksi* (Wheaton *et al.* 1996). The species *Porites porites*, *Porites furcata*, and *Porites divaricata* were grouped as “*Porites porites* complex” (Wheaton *et al.* 1996). Colonies of the species *Mycetophyllia danaana*, *Mycetophyllia ferox*, *Mycetophyllia lamarckiana*, and *Mycetophyllia aliciae* were only identified to the genus level and listed as “*Mycetophyllia sp.*” Finally, since colonies of *Siderastrea radians* were often difficult to distinguish from *Siderastrea siderea* colonies on video images, both were combined in a single category and listed as *Siderastrea siderea*.

#### *Diseased Area Measurements*

All diseased scleractinian coral survey data was entered into a Microsoft Excel database. Each diseased colony was assigned an identification and tracking label, which contained information regarding the year in which the photograph was taken, the code for the site where the colony was found, the station number, the colony number, and finally the photograph number. As an example, a diseased colony label reported as ‘03-9P3-2-12-b’ corresponds to a colony which was photographed in 2003 (‘03’), at Porter Patch station 2 (‘9P3-2’), and is colony number 12, photograph ‘b’ out several photographs taken. If that same colony was relocated and photographed again in 2004, the label for those photographs and data entries becomes ‘04-9P3-2-12-a, b or c....’

All photographs were first viewed using Adobe Photoshop<sup>®</sup> 5.0. This software allowed us to make changes in the lighting, color and contrast of each image and often vastly improve its

quality. Images were then labeled as explained above and saved onto CD's as JPEG and TIFF files.

As part of the quality assurance checks during the analysis phase, every marginal case of disease or dubious identification of disease was discussed among the observers involved in the field sampling. Any changes made to the information originally entered on the underwater data sheets was agreed upon by all observers.

Analysis of individual images was conducted using a specialized image analysis software, Image-Pro Plus<sup>®</sup> 4.5.1. Due to time constraints, this analysis was only performed for colonies photographed in 2002 and 2003. Each image was calibrated based on the graduated scale included in each photograph. Once the calibration was set for each image, tracings were made of all areas affected by disease or bleaching signs for both the overview and the close-up photographs (Figure 9). For colonies affected by line diseases such as white plague, the area of the lesion was measured as the area of the line of tissue death. For each photograph, all tracings were performed 3 times, and the mean of the 3 measurements was used. After each tracing, the resulting "measurement file" (\*.msr) and "outline file" (\*.out) were saved. The data associated with the mean area of disease lesions obtained from both the overview and close-up photographs of each colony was entered into the master disease database.

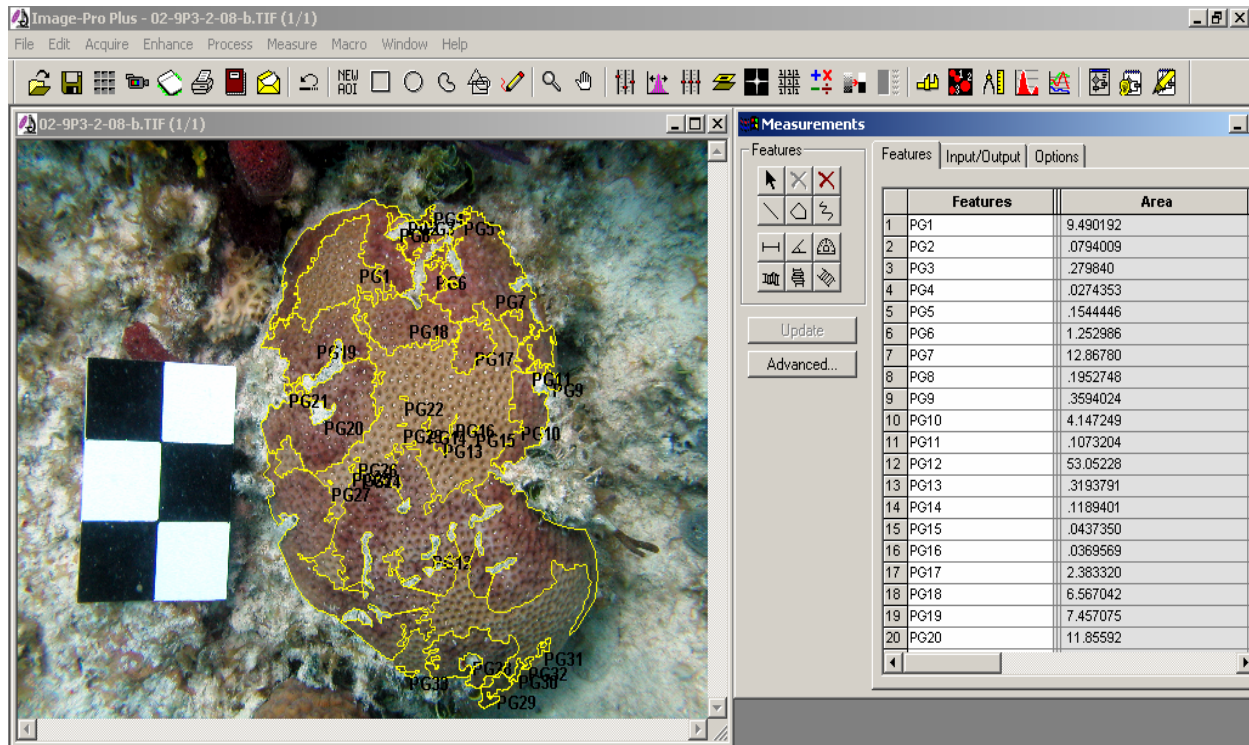


Figure 9. Image showing typical Image-Pro Plus<sup>®</sup> 4.5.1 display, in which tracings have been performed of all dark color syndrome disease lesions on a colony of *Siderastrea siderea*.

For colonies which seemed, upon visual inspection, to have lost tissue in the same regions where the lesions were located, or to have recovered from disease signs and gained tissue in those regions which were previously affected by disease, tracings were also performed of these changes in area. Because the photograph of a colony taken in 2003 could not possibly mimic *exactly* the one taken in 2002, some slight variation in the area of the colony was expected. Hence, only colonies with visibly considerable changes in area from year to year underwent analysis of tissue area changes. Whenever possible, live tissue area changes were assessed by tracing the entire area of the colony in 2002 and 2003, and calculating the difference. In some cases, the colonies photographed were too large to be entirely included within the

image. In those instances, tracings were estimated of the area in the 2002 photograph, which was dead by 2003, or of the area in the 2003 photograph, which was not alive in 2002.

### *Environmental conditions and population factors*

Disease prevalence at each of the 14 stations surveyed was compared to the following environmental conditions and population factors to determine if any correlations existed: 1) average depth at the stations, 2) water temperature (calculated as the mean of daily temperatures measured at the site during: a) the 30 days prior to the disease survey date, and b) all days of the year prior to the survey date), 3) mean percent cover of scleractinian corals, 4) H' Shannon-Weiner diversity index (calculated from video transect data), and 5) mean percent cover and relative frequency of target species. Least Squares Linear Regression analyses ( $R^2$ ) were used for comparisons.

## RESULTS

### SUCCESS OF FIELD METHODOLOGY

Contrary to many other coral disease studies, which involve tagging corals with nails to track disease progression, the non-invasive nature of this photography-based survey is considered one of its great advantages. During the field component of the diseased coral survey, the combination of: 1) the information gathered about colony distance and left-to-right location relative to the station's stake, and 2) the colony photographs available to the divers underwater,

proved to be sufficient to achieve successful relocation of diseased colonies in subsequent years. During the 2 years of the study in which colony relocation was conducted (2003 and 2004), only 2 diseased coral colonies were not relocated: 1 in 2003 (0.42% of colonies searched for in 2003), and 1 in 2004 (0.64% of colonies searched for in 2004). These were colonies for which neither presence nor death was confirmed (as indicated in the Methods section). This is a considerably high relocation rate, in comparison with surveys employing nail tags, which are often lost due to surge, wave action, predation or recreational divers, and for which relocation rates approximate 70% (Borger 2003). Hence, this can be considered a highly successful result, demonstrating the effectiveness of the field methodology developed for this survey. As with other quantitative study techniques such as the radial arc transect approach (Santavy *et al.* 2001), the major drawback to this method is the amount of time required to complete the entire station, which often amounted to more than one scuba tank per diver and more than 2 hours of underwater work per station.

## DISEASE PREVALENCE

A total of 238, 157 and 299 colonies were identified as diseased in 2002, 2003 and 2004 respectively, in all 14 stations surveyed. Table 2 shows the number of colonies with disease signs observed at each station during each year the survey was conducted. During years 2003 and 2004, the value for the number of diseased colonies includes both relocated colonies which continued to express disease signs, as well as newly diseased colonies.

Table 2. Number of scleractinian coral colonies identified as diseased in 2002, 2003 and 2004 in 14 stations surveyed in the FKNMS.

Station	Number of diseased colonies		
	2002	2003	2004
Jaap Patch Reef St. 1	14	15	104
Jaap Patch Reef St. 2	8	4	49
Western Sambo Offshore Shallow Reef St. 1	12	7	7
Western Sambo Offshore Shallow Reef St. 2	24	8	6
Cliff Green Patch Reef Station 3	12	12	5
Cliff Green Patch Reef Station 4	30	26	22
Tennessee Offshore Shallow Reef St. 2	5	2	1
Tennessee Offshore Shallow Reef St. 3	4	4	0
West Turtle Shoal Patch Reef St. 3	49	11	19
West Turtle Shoal Patch Reef St. 4	32 *	8 *	18
Grecian Rocks Offshore Shallow Reef St. 1	11	23	30
Grecian Rocks Offshore Shallow Reef St. 2	3	6	4
Porter Patch Reef St. 1	21	16	14
Porter Patch Reef St.2	13	15	20
<b>Total</b>	<b>238</b>	<b>157</b>	<b>299</b>

(\*) Only half of West Turtle Shoal Patch Station 4 was surveyed in 2002 and 2003.

Since not all stations were exactly the same length, the prevalence of diseased colonies was calculated per area surveyed. Figure 10 shows the number of diseased scleractinian coral colonies per m<sup>2</sup> in 2002, 2003 and 2004 at each of the 14 stations surveyed. This number ranged between 0.0 diseased colonies/m<sup>2</sup> at Tennessee Offshore Shallow Reef Station 3 in 2004, and 2.2 diseased colonies/m<sup>2</sup> at Jaap Patch Reef Station 1 in 2004. Tennessee Reef had the lowest number of diseased coral colonies per m<sup>2</sup> during all 3 survey years (Station 3 in 2002 and 2004, and Station 2 in 2003). West Turtle Shoal Patch Reef Station 3 had the highest number of

diseased coral colonies per m<sup>2</sup> in 2002, Cliff Green Patch Reef Station 4 in 2003, and Jaap Patch Reef Station 1 in 2004. Across all 14 stations combined, the average number of diseased colonies per m<sup>2</sup> was 0.4 in 2002, 0.3 in 2003 and 0.5 in 2004.

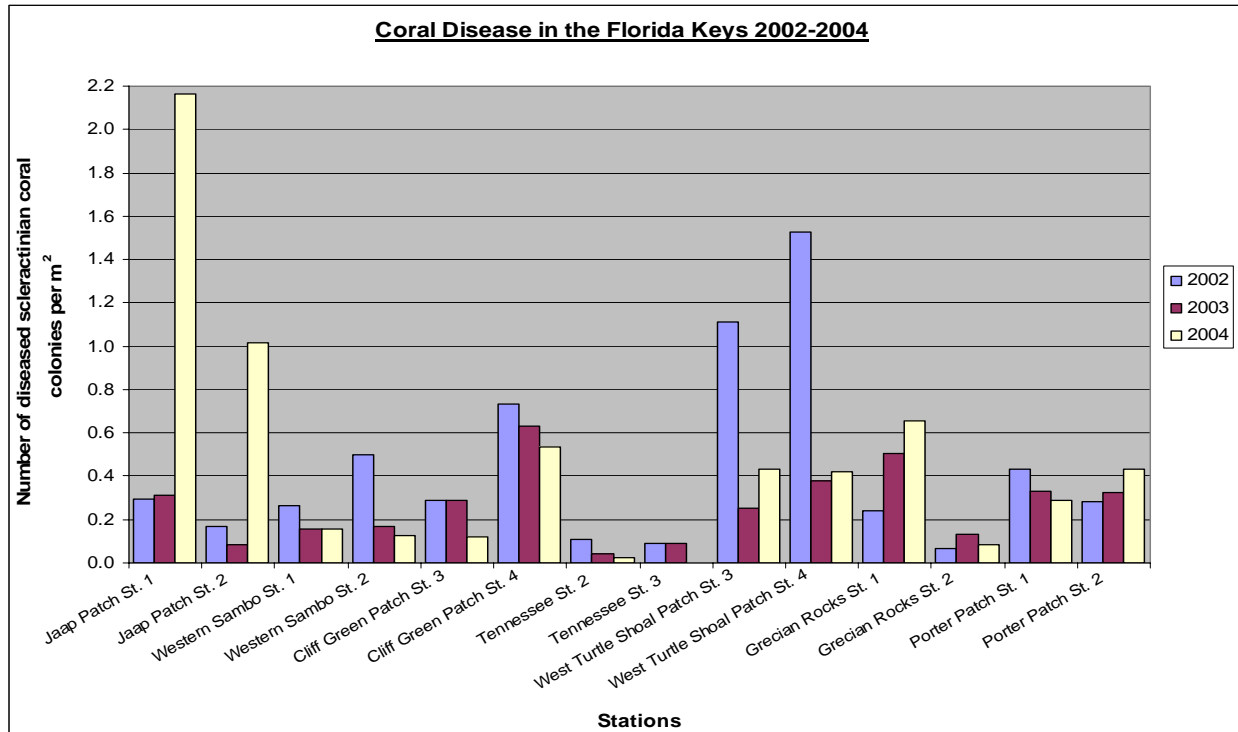


Figure 10. Number of scleractinian coral colonies with disease signs per m<sup>2</sup> surveyed in 14 stations throughout the FKNMS in 2002, 2003, and 2004.

## DISEASE TYPES

In addition to the 8 diseases listed in the Methods section, which have been described in the literature, 2 other potential disease conditions were encountered, which did not match descriptions of known diseases, but which were prevalent and widespread enough that it was

decided to include them in this disease survey. They are referred to as “condition 1” and “condition 2” because, at this point, insufficient information is available to characterize them, their causative agent, or in one case even whether it ultimately causes coral tissue loss. These potential diseases were common enough and their features sufficiently distinctive, that their inclusion and observation was warranted. However, they were not given specific names in an effort to avoid adding to the confusion that has been created by the plethora of *ad hoc* names in disease reports during the last decade (Richardson 1998). The conditions are described as follows:

### **Condition 1**

This potential disease was only observed on colonies of *Montastrea cavernosa*. Affected colonies show a pattern of tissue loss on individual polyps scattered, randomly it appears, across large regions of the colony. Affected polyps are not necessarily located next to one another. Dying polyps evidence a thinning and peeling of the live tissue as shown on Figure 11. In some cases, the colonies recovered from this condition from one survey year to the following without any tissue loss. However in other cases the condition was capable of causing loss of very large tissue areas, and 1 colony identified with this condition died during the survey.

### **Condition 2**

This potential disease was observed only on colonies of *Montastrea annularis* complex. It is characterized by a darkening of the coral tissue in small to large areas of the colony. This condition can sometimes appear very similar to dark color syndrome, and where both are present in close proximity, distinguishing between them can be tricky. In dark color syndrome affected

colonies of *M. annularis* complex, the darkening of the tissues is much more internal and profound, whereas in colonies affected with “condition 2” the darker coloration seems more superficial, almost as though it had been painted onto the coral (Figure 12). Furthermore, the edges of the darker coloration areas are very sharp, with individual polyps often only partially affected. It is possible that this condition may be a variant or a stage of dark color syndrome. However, this condition did not appear to cause tissue loss in the colonies affected. It is hence also likely that this may be a benign phenomenon and not a potential disease.

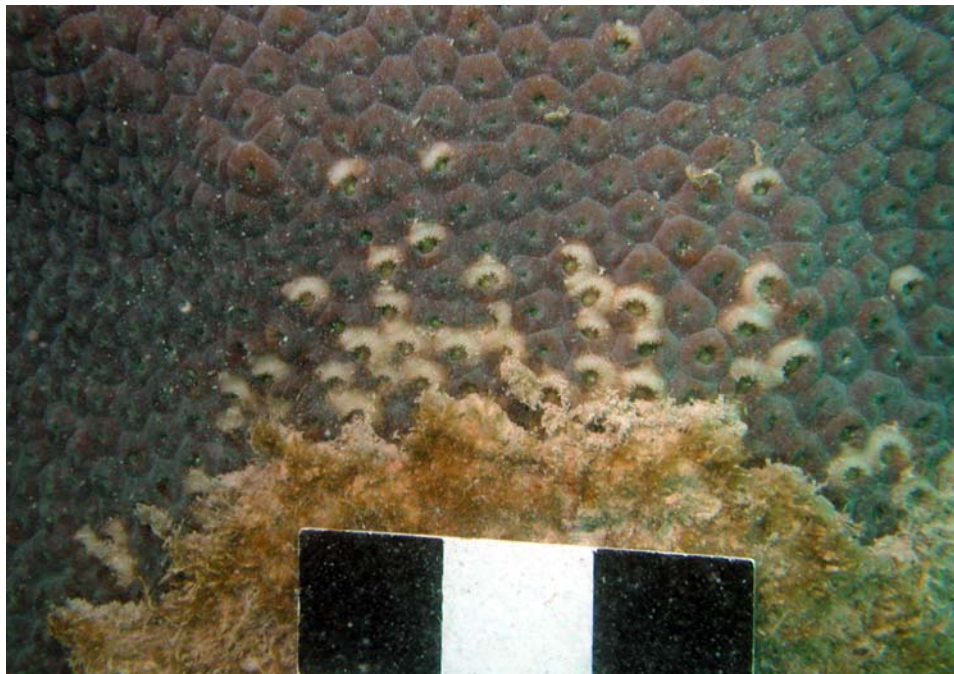


Figure 11. Example of the potential disease condition affecting colonies of *Montastrea cavernosa* in several of the stations surveyed and referred to as “condition 1.”

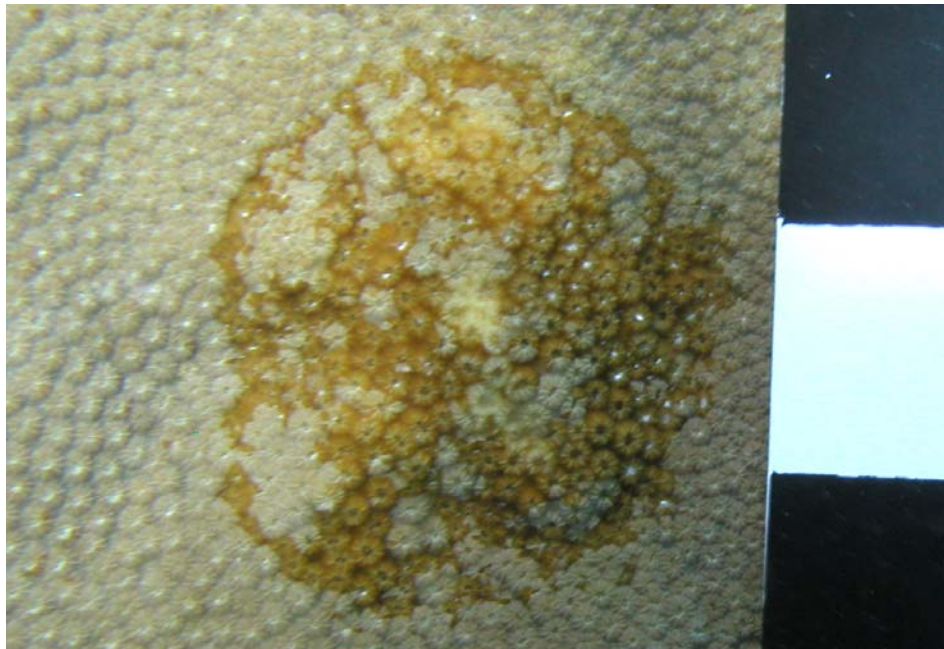


Figure 12. Example of the potential disease condition affecting colonies of *Montastrea annularis* complex in several of the stations surveyed and referred to as “condition 2.”

In addition to these 2 conditions, many examples were found during the study, of coral colonies which appeared most evidently diseased, yet the signs were confusing, not easily identifiable or did not match descriptions of known diseases. Since these cases were not widespread, but rather single cases of potential disease, they were grouped into one category and listed as “unknown.” Care was taken not to include any cases of potential predation.

Although the number of scleractinian coral colonies identified with disease decreased between 2002 and 2003, the number of diseases types found increased slightly. A total of 9, 11 and 11 scleractinian coral disease types were identified in 2002, 2003 and 2004 respectively. Disease types observed in 2002 included bleaching, Caribbean yellow band, conditions 1 and 2, dark color syndrome, skeletal anomalies, white plague, white pox and “unknown.” In addition to these, cyanobacterial mat disease and white band were also observed in 2003 and 2004. Dark

color syndrome was by far the most abundant disease type, affecting 65.1%, 66.1% and 82.5% of all diseased colonies identified in 2002, 2003 and 2004 respectively (Figures 13, 14 and 15).

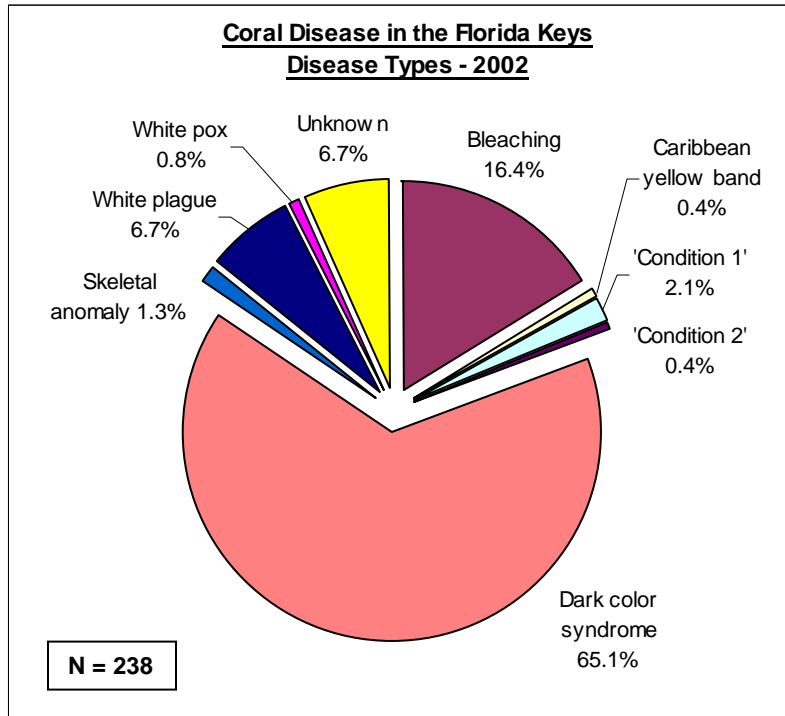


Figure 13. Frequency of scleractinian coral disease types identified in 2002 in 14 stations surveyed in the FKNMS.

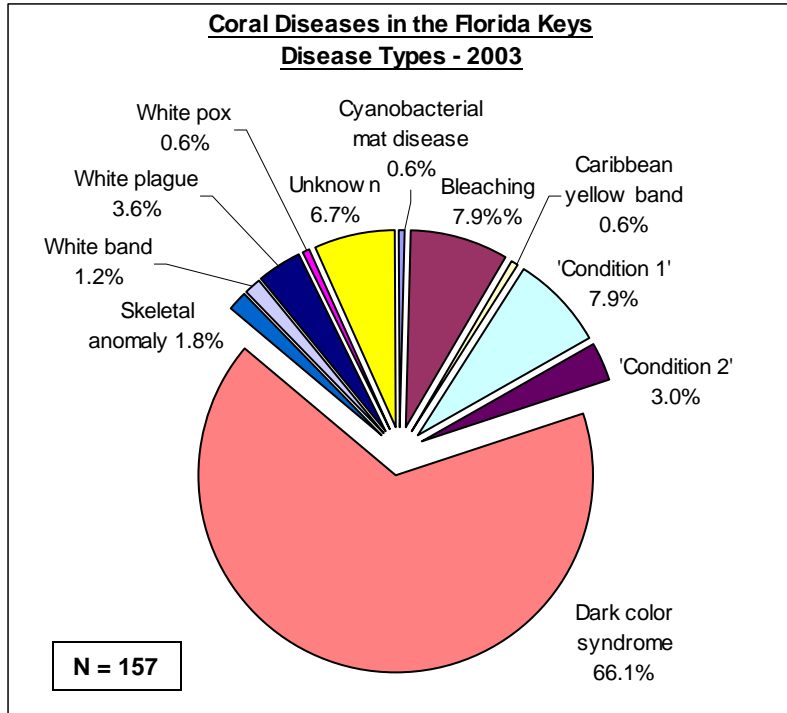


Figure 14. Frequency of scleractinian coral disease types identified in 2003 in 14 stations surveyed in the FKNMS.

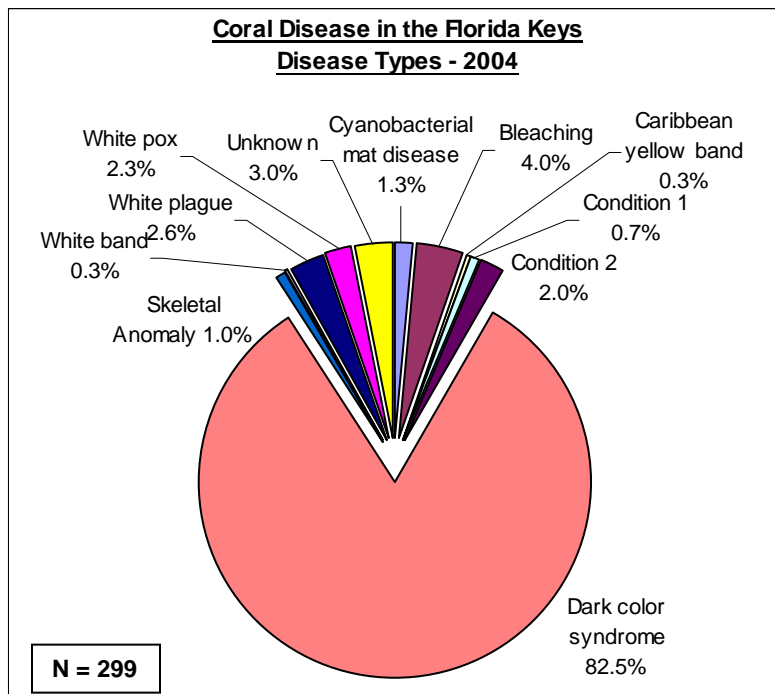


Figure 15. Frequency of scleractinian coral disease types identified in 2004 in 14 stations surveyed in the FKNMS.

## TARGET HOST SPECIES

Station species inventory counts revealed a total of 31, 29 and 27 scleractinian coral species found in the 14 stations surveyed in 2002, 2003 and 2004 respectively. Examples of disease were identified in a total of 17, 18 and 16 scleractinian coral species in 2002, 2003 and 2004 respectively. Affected species in 2002 included: *Agaricia agaricites* complex, *Acropora cervicornis*, *Acropora palmata*, *Colpolphyllia natans*, *Diploria clivosa*, *Diploria strigosa*, *Dichocoenia stokesii*, *Eusmilia fastigiata*, *Favia fragum*, *Montastrea annularis* complex, *Montastrea cavernosa*, *Meandrina meandrites*, *Oculina diffusa*, *Porites astreoides*, *Solenastrea bournoni*, *Stephanocoenia michelinii*, and *Siderastrea siderea*. In 2003, colonies of *Diploria labyrinthiformis* and *Porites porites* complex were also identified with disease signs, though no colonies of *Meandrina meandrites* were found with disease signs. In 2004, disease cases were found in all aforementioned species except *Diploria labyrinthiformis*, *Meandrina meandrites* and *Porites porites* complex.

In 2002, *Siderastrea. siderea* was the most frequently diseased species (55.0%), followed by *Montastrea annularis* complex (12.2%) and *Favia fragum* (8.4%) (Figure 16). In 2003, *S. siderea* was still the species most frequently diseased, though its relative frequency decreased to 37.6%, while the relative frequency of diseased *M. annularis* increased to 26.7%, followed by *S. michelinii* (8.5%) (Figure 17). In 2004, *M. annularis* complex became the most frequently diseased coral species, with a relative frequency of 60.7%, followed by *S. siderea* (22.4%) (Figure 18).

**Coral Disease in the Florida Keys**  
**Affected Coral Species - 2002**

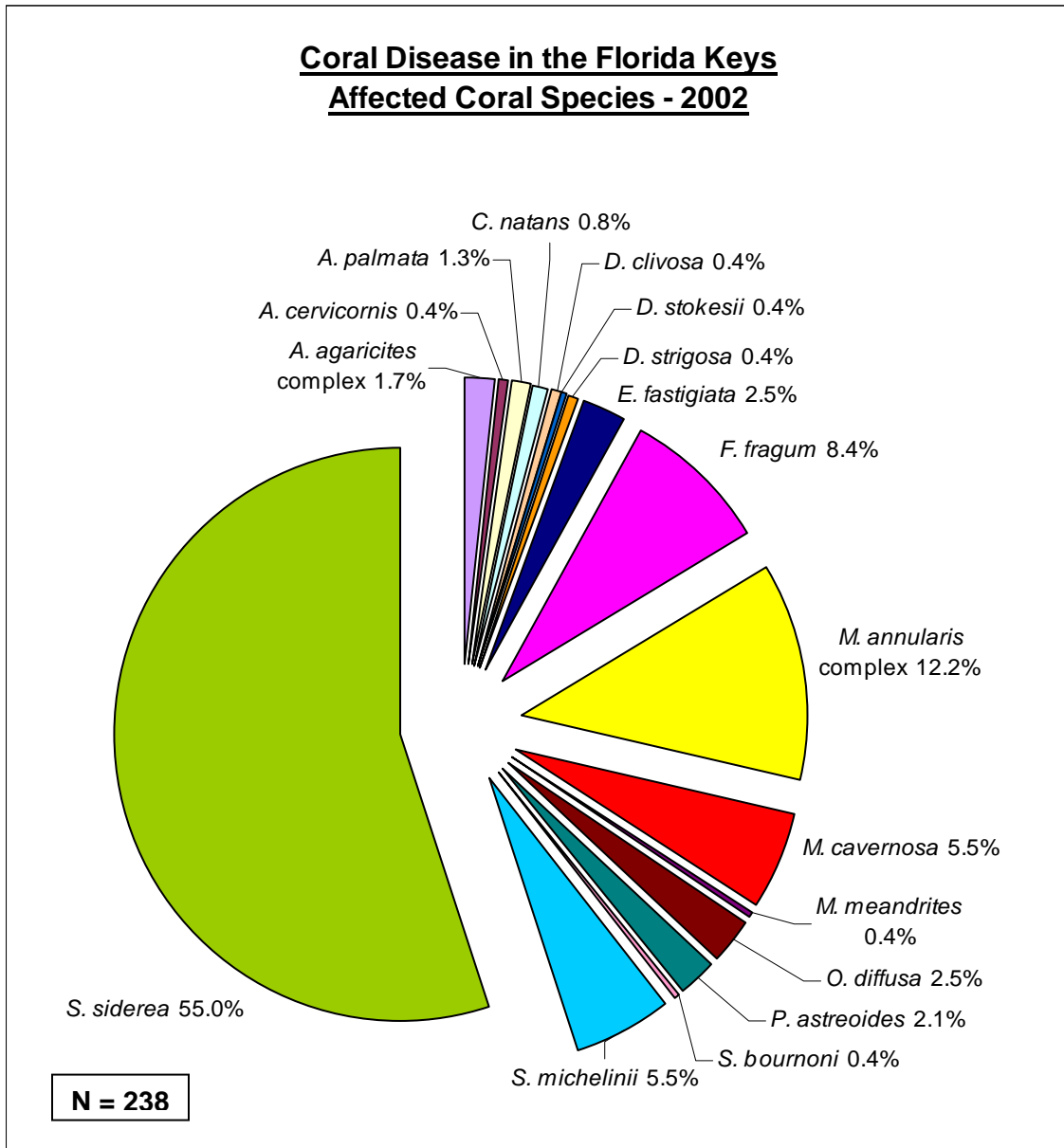


Figure 16. Relative frequency of scleractinian coral species with signs of disease in 2002 in 14 stations surveyed in the FKNMS.

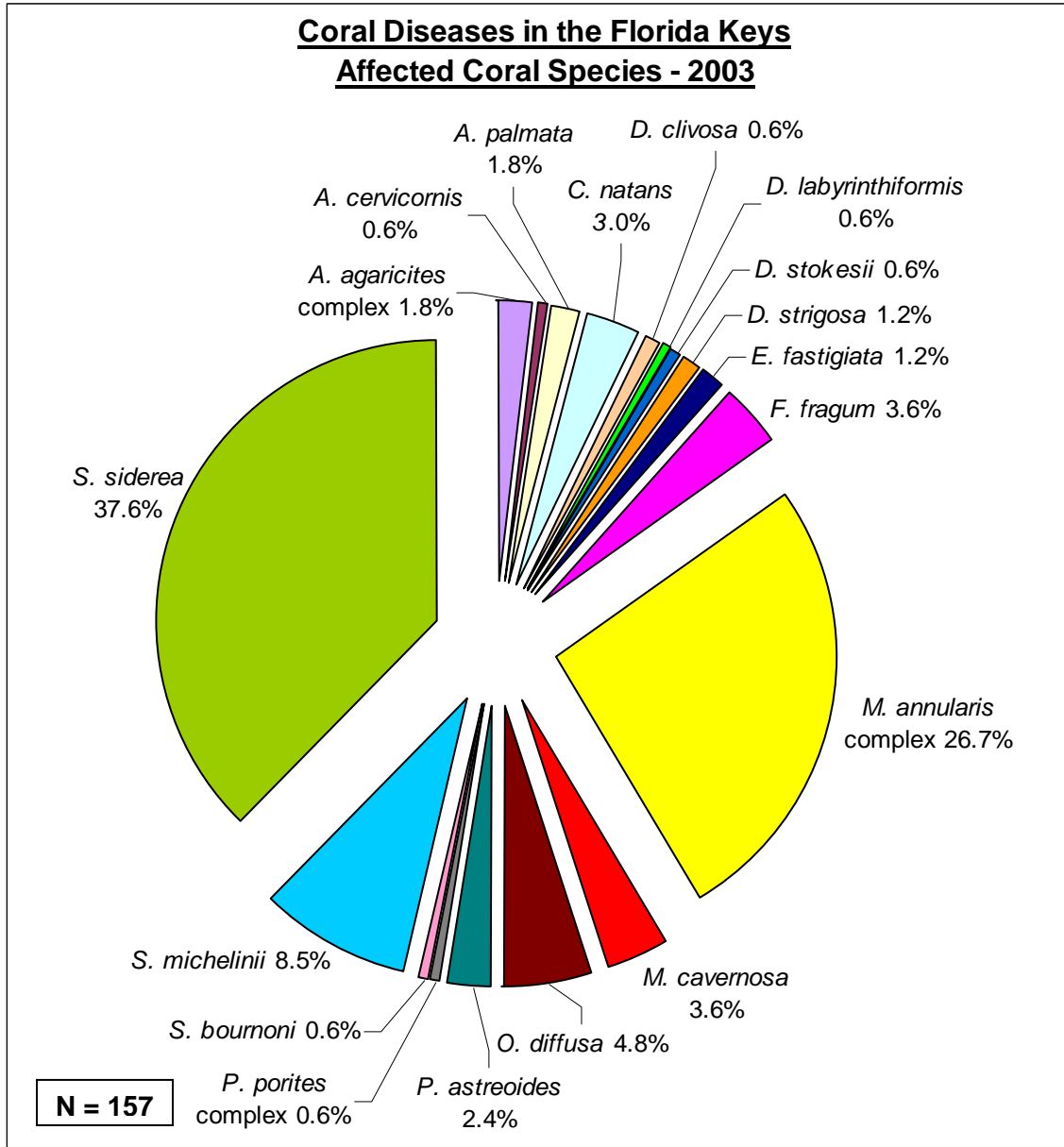


Figure 17. Relative frequency of scleractinian coral species with signs of disease in 2003 in 14 stations surveyed in the FKNMS.

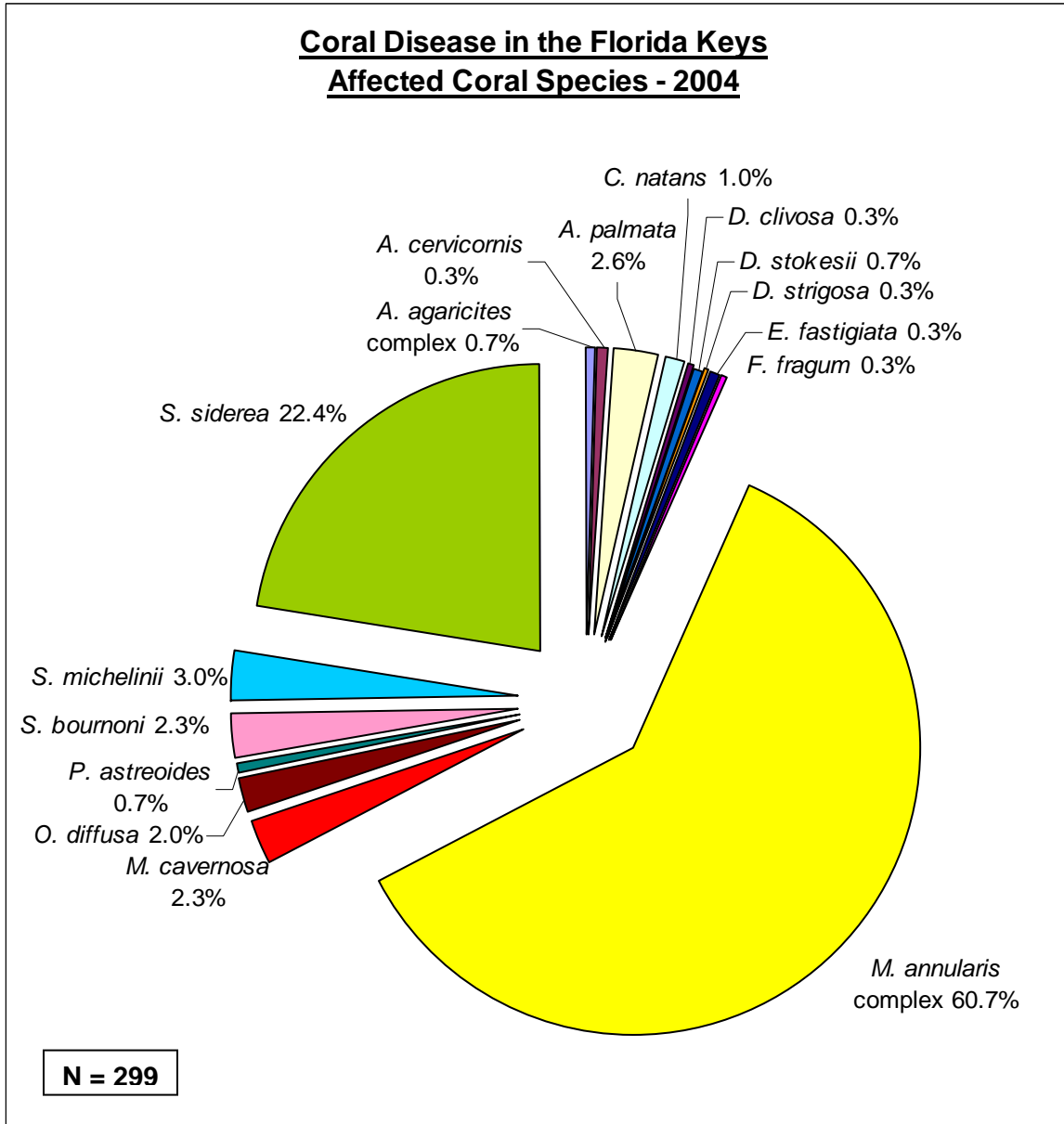


Figure 18. Relative frequency of scleractinian coral species with signs of disease in 2004 in 14 stations surveyed in the FKNMS.

In addition to the disease types known to date to affect only one or two coral species, such as white pox (affecting only *Acropora palmata*), white band (affecting only acroporid corals), or Caribbean yellow band (affecting only *Montastrea annularis* complex), several other disease types were found to target a few species preferentially (Figure 19). As previously mentioned, ‘condition 1’ was only found to affect *Montastrea cavernosa*, and ‘condition 2’ was only observed on colonies of *M. annularis* complex. Although cyanobacterial mat disease has been reported to affect 19 Caribbean species of scleractinian corals (Sutherland *et al.* 2004), during this survey, only 2 examples of this disease were observed, affecting colonies of *Colpolphyllia natans* and *M. cavernosa*. Similarly, skeletal anomalies and dark color syndrome are known to affect 16 and 11 different Caribbean scleractinian coral species respectively (Sutherland *et al.* 2004). During this survey, only 3 colonies exhibited skeletal anomalies, belonging to the species *A. palmata*, *Diploria strigosa*, and *Diploria clivosa*, the later of which had not yet been reported as a target species. Although dark color syndrome was identified on 6 different coral species (*Agaricia agaricites* complex, *Dichocoenia stokesii*, *M. annularis* complex, *Solenastrea bournoni*, *Stephanocoenia michelinii*, and *Siderastrea siderea*), 35.2% of affected colonies were *M. annularis* complex colonies, 32.1% were *S. siderea* colonies and 4.3% were *S. michelinii* colonies. In contrast, bleaching and white plague were found affecting a wide variety of coral species, in both cases 10 scleractinian coral species.

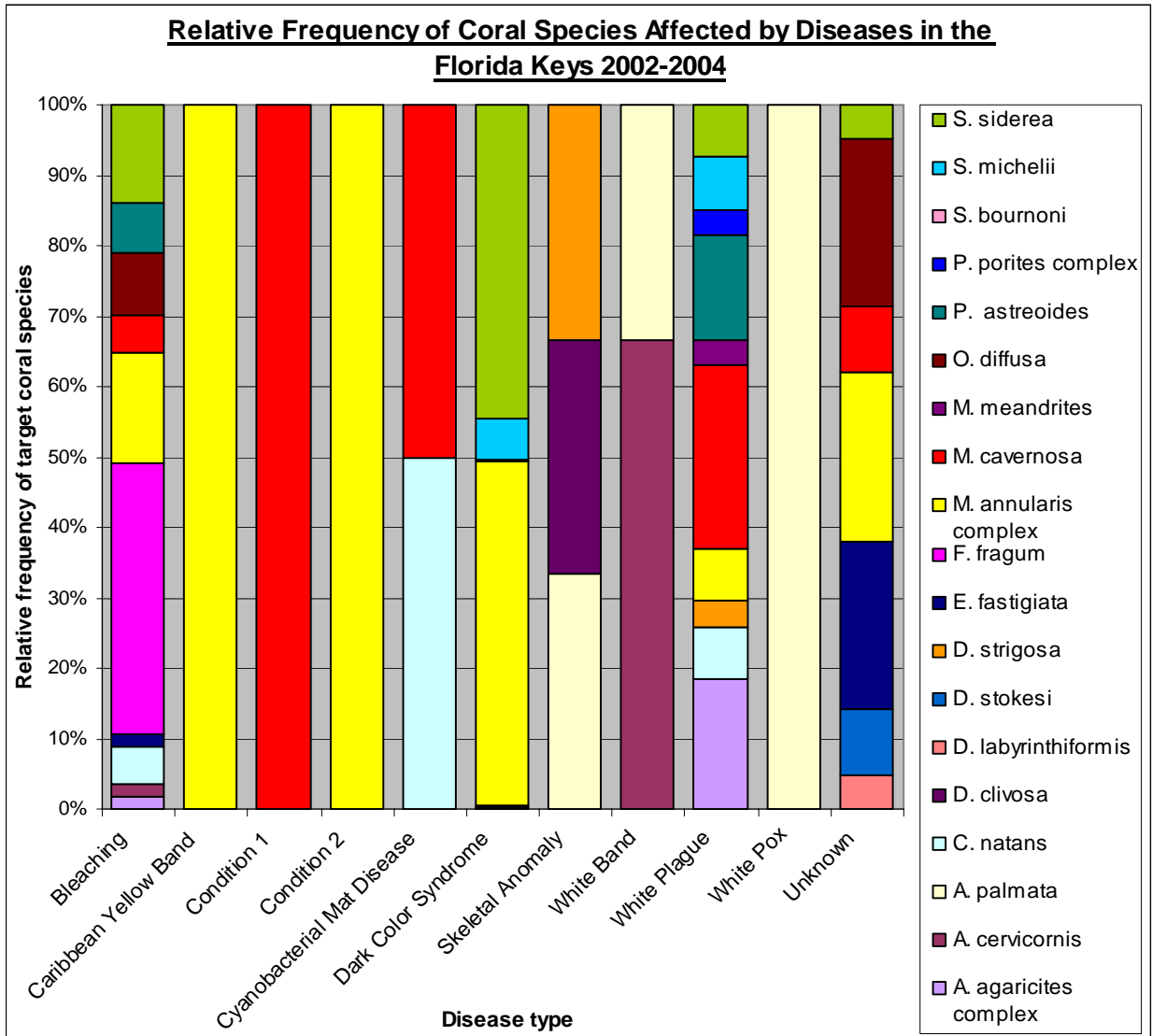


Figure 19. Frequency of target scleractinian coral species affected by each of 11 coral disease types identified between 2002-2004 at 14 stations surveyed in the FKNMS.

### SCLERACTINIAN CORAL POPULATION COUNTS

Based on the scleractinian coral colony counts performed in the first 20 m<sup>2</sup> of the offshore half of every station, colony density ranged between an average of 0.6 colonies per m<sup>2</sup> at Grecian Rocks Offshore Shallow Reef Station 2, and 20.4 colonies per m<sup>2</sup> at West Turtle

Shoal Patch Reef Station 3. The mean number of scleractinian coral colonies per m<sup>2</sup> ( $\pm$  standard error) at each of the 14 stations surveyed is listed in Table 3. Appendix II includes figures depicting the mean colony density of all scleractinian coral species at each of the 14 stations surveyed.

Table 3. Mean number of scleractinian coral colonies (greater than 3 cm in diameter) per m<sup>2</sup> at each of 14 stations surveyed in the FKNMS ( $\pm$  standard error).

Station	Mean number of scleractinian coral colonies per m <sup>2</sup> ( $\pm$ standard error)
Jaap Patch Reef St. 1	2.6 ( $\pm$ 0.1)
Jaap Patch Reef St. 2	4.0 ( $\pm$ 0.2)
Western Sambo Offshore Shallow Reef St. 1	3.5 ( $\pm$ 0.3)
Western Sambo Offshore Shallow Reef St. 2	9.3 ( $\pm$ 1.1)
Cliff Green Patch Reef St. 3	17.0 ( $\pm$ 0.6)
Cliff Green Patch Reef St. 4	17.9 ( $\pm$ 0.4)
Tennessee Offshore Shallow Reef St. 2	1.8 ( $\pm$ 0.1)
Tennessee Offshore Shallow Reef St. 3	2.3 ( $\pm$ 0.1)
West Turtle Shoal Patch Reef St. 3	20.4 ( $\pm$ 1.2)
West Turtle Shoal Patch Reef St. 4	10.2 ( $\pm$ 0.4)
Grecian Rocks Offshore Shallow Reef St. 1	1.5 ( $\pm$ 0.1)
Grecian Rocks Offshore Shallow Reef St. 2	0.6 ( $\pm$ 0.1)
Porter Patch Reef St. 1	3.5 ( $\pm$ 0.2)
Porter Patch Reef St. 2	5.0 ( $\pm$ 0.2)

In order to estimate the prevalence of diseased *versus* healthy colonies, the number of diseased colonies identified in the first 20 m<sup>2</sup> of the offshore half of each station was used. This treatment was selected rather than extrapolating the mean coral population values from the first

20 m<sup>2</sup> of the offshore half of the station to the entire station, because considerable variability in the density of coral colonies at either end of the transect was often observed. Based *only* on the diseased colonies found in the first 20 m<sup>2</sup> of the offshore half of the station for all 14 stations combined, and all scleractinian coral species combined, the percent of colonies in the population experiencing disease was 7.1% in 2002, 4.0% in 2003 and 8.2% in 2004.

At the station level, with the exception of Jaap Patch Reef Station 1, where in 2004 approximately 100% of the coral colonies located in the first 20 m<sup>2</sup> of the offshore half of the station showed signs of disease, the percent of colonies in the population found to be diseased ranged between 0% (Tennessee Offshore Shallow Reef Stations 2 and 3 in 2004) and 33.9% (Jaap Patch Station 2 in 2004) (Figure 20). The high incidence of disease at Jaap Patch Reef in 2004 corresponds to an observed outbreak of dark color syndrome affecting colonies of *Montastrea annularis* complex. At Jaap Patch Reef Station 1, approximately 98% of the coral cover is composed of *M. annularis* complex colonies, most of which were affected by dark color syndrome in 2004.

A species-level analysis indicates that, with the exception of *Montastrea annularis* complex and *Favia fragum*, the percent of colonies diseased in the population of all other species was below 20% (Figure 21). In the case of *M. annularis* complex, 61.5% of the population (which was found in 11 of the 14 stations surveyed) was diseased in 2004, the majority of these at Jaap Patch reef, as mentioned previously. Though the population of *F. fragum* was small, with a total of approximately 3.8 colonies (located in only 3 of the 14 stations), all colonies of *F. fragum* found at Western Sambo Offshore Shallow Stations 1 and 2 were bleached, explaining the high percent disease prevalence for that species (Figure 21).

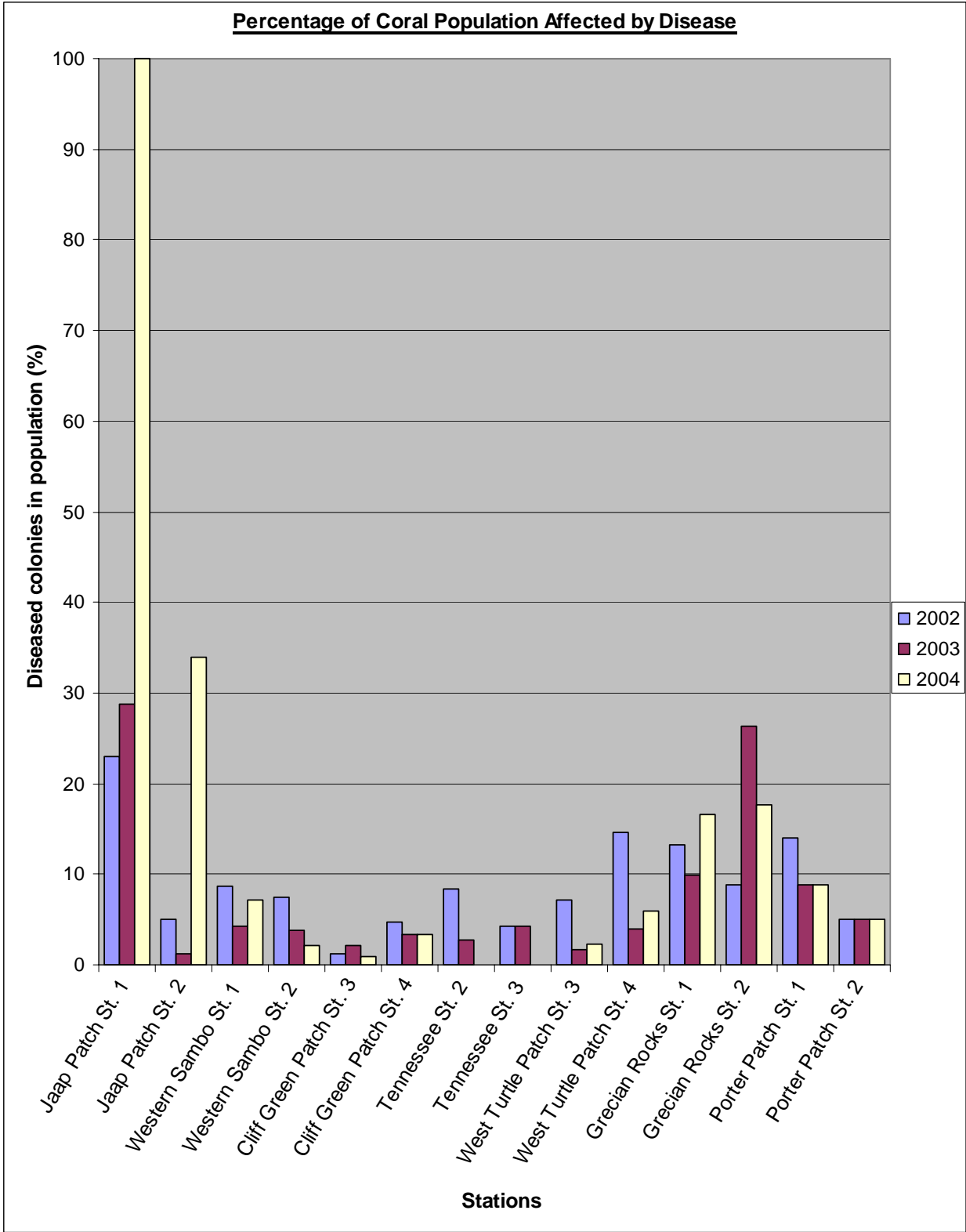


Figure 20. Percent of scleractinian coral colonies located in the first 20 m<sup>2</sup> of the offshore half of each station with signs of disease in 2002, 2003, and 2004.

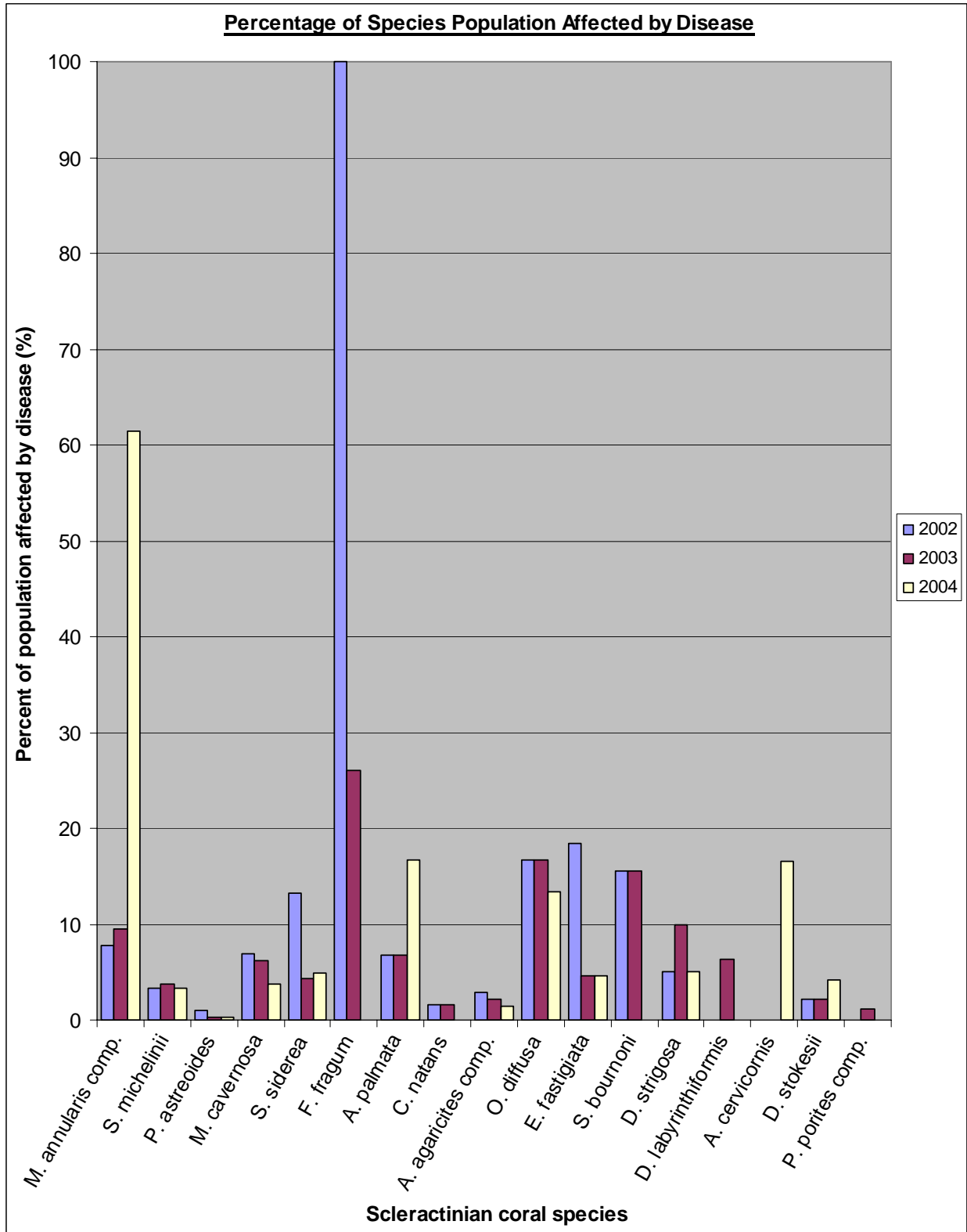


Figure 21. Percent of the population of 17 scleractinian coral species affected by disease in 14 stations surveyed in the FKNMS in 2002, 2003 and 2004.

## TEMPORAL FATE OF DISEASED CORAL COLONIES

### *Disease persistence*

Of 238 coral colonies identified with disease in 2002, 237 were successfully relocated in 2003. Of these, 95 (40.1%) continued to show signs of a disease, while 115 (48.5%) appeared visually healthy, and 27 (11.4%) had died by 2003 (Figure 22).

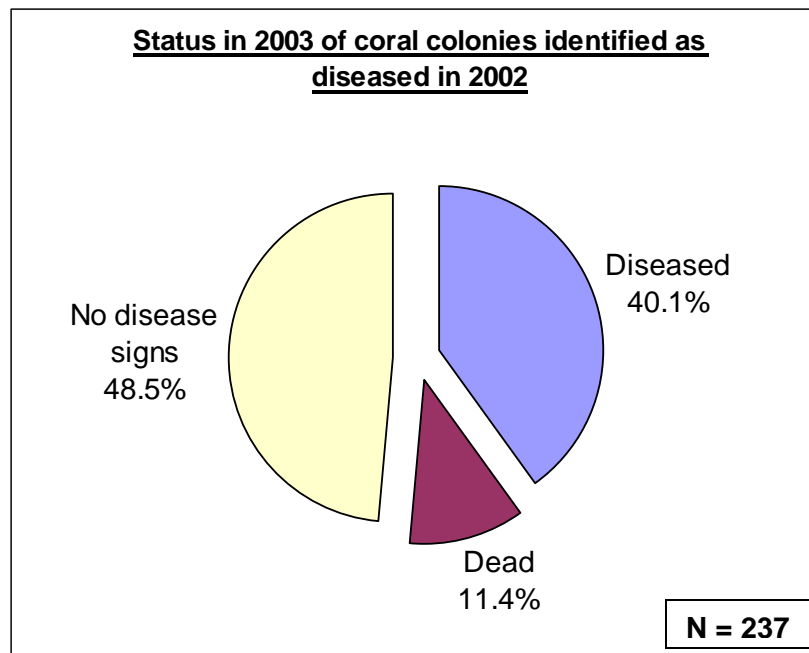


Figure 22. Status in 2003, of 237 scleractinian coral colonies identified as diseased in 2002.

Over the subsequent year, from 2003 to 2004, continued tracking of the original 237 colonies identified in 2002 revealed that, of the 95 colonies which continued to show disease signs in 2003, 63 (66.3%) were still diseased in 2004, while 26 (27.4%) appeared healthy in 2004 and 6 (6.3%) more colonies died. Interestingly, of the 115 diseased colonies identified in 2002

which appeared healthy in 2003, 17 (14.8%) once again exhibited signs of the same disease in 2004. In summary: 80 (33.8%) of the 237 colonies identified as diseased in 2002 still showed disease signs in 2004, 124 (62.3%) appeared healthy in 2004, and 33 (13.9%) died between 2002 and 2004 (Figure 23).

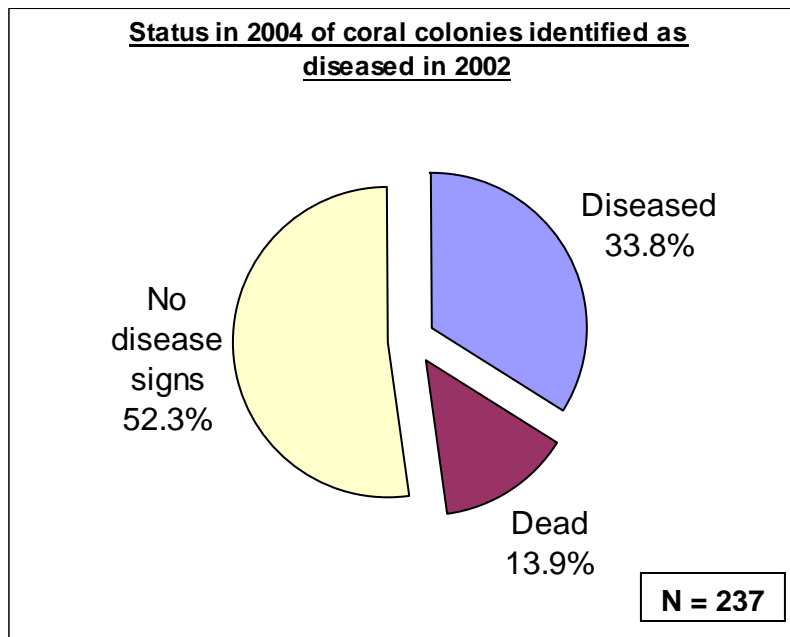


Figure 23. Status in 2004, of 237 coral colonies identified as diseased in 2002.

In 2003, 62 newly diseased colonies were added to the survey, of which 61 were successfully relocated in 2004: 34 (55.7%) continued to show disease signs, 3 (4.9%) died, and 24 (39.3%) were no longer diseased (Figure 24). In 2004, in addition to 97 relocated coral colonies still diseased from the previous year (63 originally marked in 2002, 34 marked in 2003), 202 newly diseased colonies were identified, 17 of which, as mentioned previously, had been diseased in 2002, but appeared healthy in 2003.

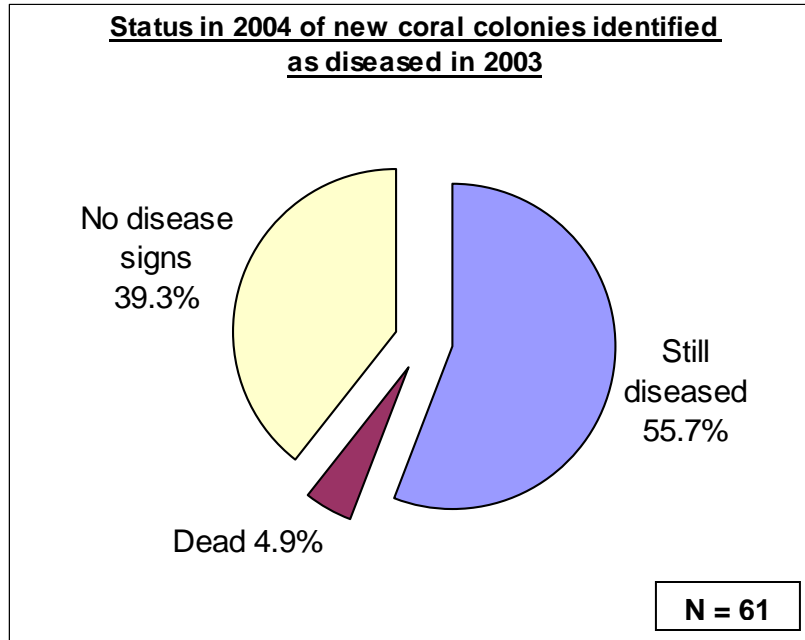


Figure 24. Status in 2004, of 61 new scleractinian coral colonies identified as diseased in 2003.

The following results provide a closer look at the persistence or cessation of specific disease types:

#### Dark color syndrome

Of 155 dark color syndrome diseased colonies identified in 2002, only 1 colony was marked as dead the subsequent year (0.6%). In 85 (54.8%) of the colonies affected by dark color syndrome in 2002, disease signs had completely disappeared by the following year. Of the 69 colonies which remained diseased in 2003, another 20 (29.0%) appeared healthy by 2004, while 3 more colonies died (4.3%). It should be noted that, out of the 17 cases of disease disappearance and subsequent reappearance mentioned previously, 16 were dark color syndrome infections.

In summary, after two years of observation: 62 (40.0%) of the original 155 dark color syndrome diseased colonies marked in 2002 were still diseased, 89 (57.4%) no longer showed signs of disease, and only 4 (2.6%) none had died.

In 2003, 39 new colonies were identified with dark color syndrome. By 2004, 11 (28.2%) were no longer affected by disease, and none had died. As seen in Figure 25, which summarizes the fate of 194 coral colonies identified with dark color syndrome in 2002 and 2003 (155 and 39 respectively), by 2004 the disease remained active in fewer than half of the colonies, and resulted in the death of only 4 colonies (2.1%).

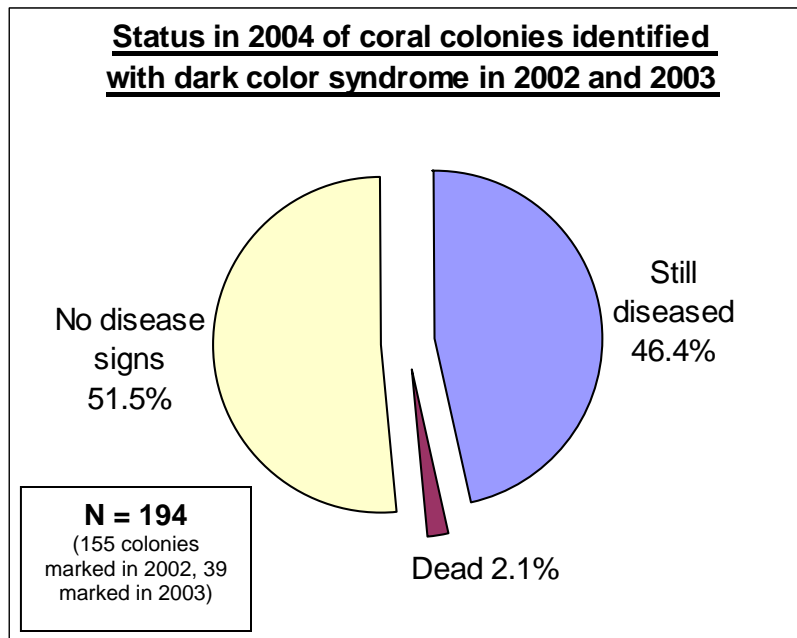


Figure 25. Status in 2004, of 194 scleractinian coral colonies identified as diseased with dark color syndrome in 2002 (N=155) and 2003 (N=39).

## White plague

In the case of white plague, by 2003, disease signs had disappeared in 9 (56.3%) of 16 colonies affected in 2002, while 5 colonies (31.3%) had died and 2 (12.5%) were still diseased (though both appeared fully recovered in 2004). Six newly diseased colonies were identified with white plague in 2003, of which only 1 continued to appear diseased in 2004 and none died.

Figure 26 summarizes the fate of 22 colonies identified with white plague in 2002 and 2003 (16 and 6 respectively).

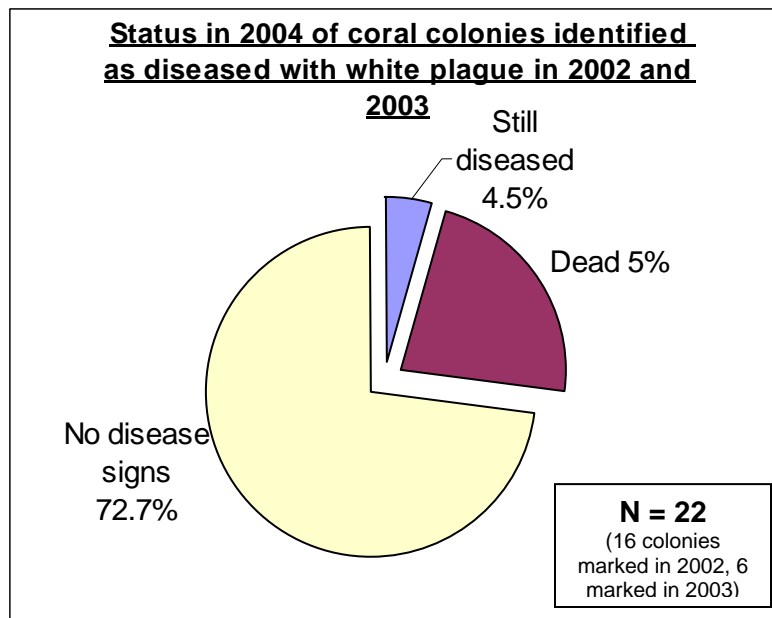


Figure 26. Status in 2004, of 22 scleractinian coral colonies identified as diseased with white plague in 2002 (N=16) and 2003 (N=6).

## Bleaching

In 2002, 38 coral colonies were identified as bleached, of which 4 (10.5%) were still bleached the subsequent year (though only 2 showed bleaching signs in 2004), 19 (50.0%) died,

and 15 (39.5%) were no longer bleached in 2003. One of the colonies which had been identified as diseased in 2002, and as healthy in 2003, appeared bleached again in 2004. Eight new colonies were identified as bleached in 2003, of which 2 died, 5 were no longer diseased and 1 continued to exhibit bleaching signs in 2004.

In summary, between 2002 and 2004, a total of 46 colonies were identified as bleached (38 in 2002 and 8 in 2003). By 2004, 21 (45.7%) of them were no longer bleached, 21 (45.7%) had died and 4 (8.7%) were still diseased, as can be seen in Figure 27.

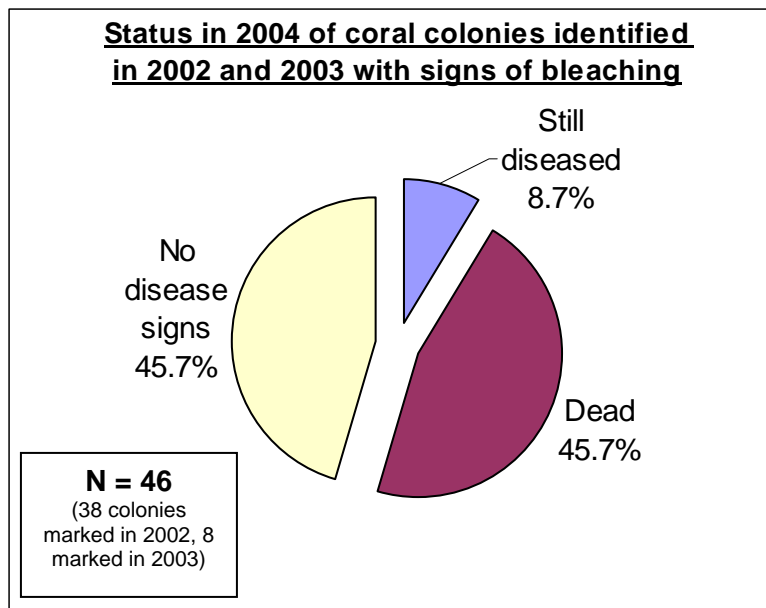


Figure 27. Status in 2004, of 46 scleractinian coral colonies identified in 2002 (N=38) and 2003 (N=8) with signs of bleaching.

### Condition 1

In 2002, 5 colonies were included in the survey with signs of what was termed as “condition 1”. All were *Montastrea cavernosa*, the only species affected by this condition. In

2003, only 3 of those colonies remained diseased, one of which died by 2004. Four newly diseased *M. cavernosa* colonies were added to the survey in 2003, and all appeared healthy the subsequent year. By 2004, of 9 colonies identified with “condition 1” (5 in 2002, 4 in 2003), 2 (22.2%) remained diseased, 6 (66.7%) appeared to show no signs of disease, and 1 (11.1%) had died (Figure 28).

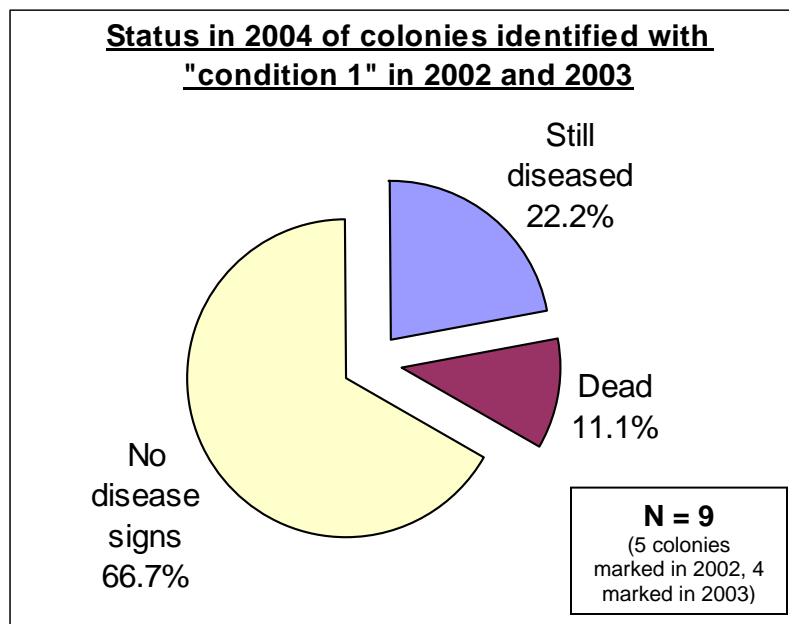


Figure 28. Status in 2004, of 9 colonies identified with ‘condition 1’ in 2002 (N=5) and 2003 (N=4).

#### Other disease types

- Only one colony was identified with Caribbean yellow band in 2002, and the disease persisted during the two subsequent years.

- In 2002, one *Montastrea annularis* complex colony was identified with ‘condition 2’, which also persisted during the two subsequent years. Five more colonies of *M. annularis* complex (the only target species of this condition) were identified with the condition in 2003, all of which continued to show disease in 2004.

- Three examples of skeletal anomalies were encountered in 2002, all of which persisted during the two subsequent survey years.

- Two cases of white pox were identified in 2002. The disease persisted on one colony until 2004, while the other colony no longer showed disease signs in 2003, although it was observed to have died by 2004. Its death may have been due to predation by the snail *Coralliophila abbreviate*. High densities of this corallivore were observed on that particular reef (Grecian Rocks Offshore Shallow Reef) in 2004. Six new white pox cases were identified in 2004, all at Western Sambo Reef.

- One case of cyanobacterial mat disease was observed in 2003, which persisted through 2004. A second colony was marked as diseased with the condition in 2004.

- Two cases of white band were identified in 2003. The condition had disappeared on one of the colonies by 2004, while it resulted in mortality in the second colony. One new colony was marked with the disease in 2004.

#### “Unknown” diseases

Finally, it is worth mentioning the fate of colonies marked as diseased during the survey, but for which a specific disease designation was not possible. Sixteen coral colonies were identified with ‘unknown’ disease signs in 2002, of which 2 (12.5%) died, 5 (31.3%) appeared healthy, and 9 (56.3%) were still diseased in 2003. By 2004, of these 9 diseased colonies, 6

(66.7%) remained diseased, 2 (22.2%) died and 1 (11.1%) appeared to show no signs of disease.

In 2003, 2 newly diseased colonies were identified with “unknown” signs, though the disease signs persisted in only 1 of them through the subsequent year.

As shown in Figure 29, which summarizes the fate of 25 colonies marked as diseased with “unknown” conditions (16 in 2002 and 9 in 2003), by 2004, 7 (38.9%) of the colonies remained diseased, while 3 (16.7%) had died, and 8 (44.4%) were no longer diseased.

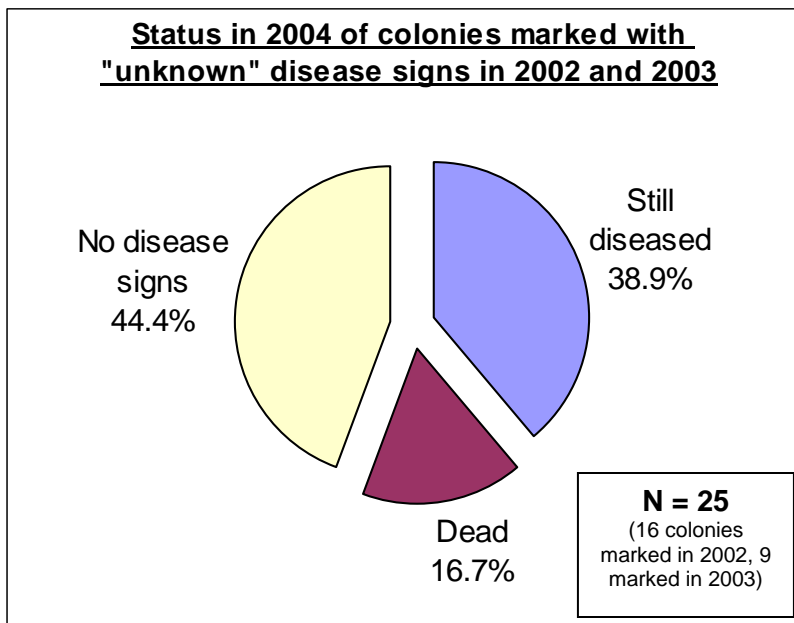


Figure 29. Status in 2004, of 25 scleractinian colonies marked with “unknown” disease signs in 2002 (N=16) and 2003 (N=9).

### *Multiple infections*

In addition to the 17 colonies mentioned earlier, for which 2002 disease signs disappeared in 2003 and reappeared in 2004 (16 dark color syndrome infections and one

bleached colony), several cases were observed of multiple concurrent or subsequent disease infections on the same colonies. Three colonies were found to recover from one disease infection (bleaching, dark color syndrome and white plague) and express signs of a new disease in a subsequent year (dark color syndrome, bleaching and cyanobacterial mat disease respectively). Three cases were found of colonies which acquired two simultaneous disease infections (skeletal anomalies and white pox in one case, and condition 2 and dark color syndrome in another colony).

### *Coral tissue mortality*

Based on comparison of photographs between years, colonies were determined to have either: lost tissue area, experienced no significant change in tissue area, or gained tissue area. The later case refers to colonies which recovered from disease and live tissue regenerated in those areas previously dying due to disease (areas previously denuded of live tissue). Normal colony growth in areas which did not previously show disease lesions was not included or traced.

Among the 237 colonies marked in 2002 and relocated in 2003, 107 (45.1%) experienced no significant tissue change, 100 (42.2%) suffered tissue loss, and 30 (12.7%) experienced tissue gain (Figure 30). Of the colonies which experienced either no tissue change or a tissue gain between 2002 and 2003, 20 did suffer a tissue loss during the second year (2003-2004).

In 2004, 156 coral colonies diseased the previous year were relocated. Fifty-five (35.3%) had experienced no significant change in tissue area, while 96 (61.5%) had lost tissue (of which 39 were colonies newly identified in 2003), and 5 (3.2%) had experienced gains in tissue area (Figure 30).

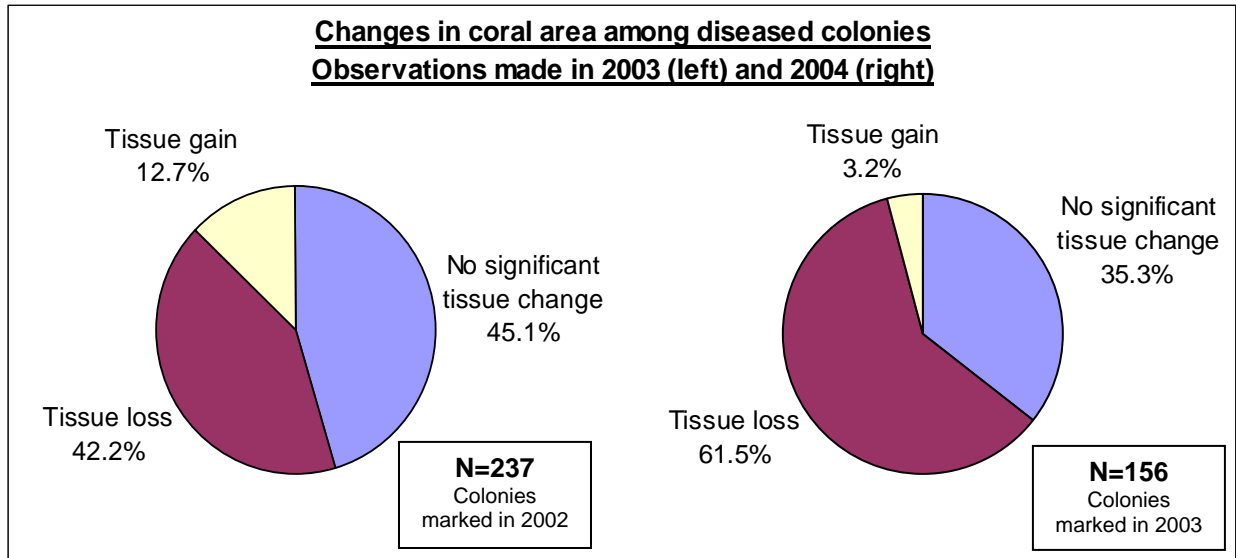


Figure 30. Changes in coral tissue areas by 2003, of 237 colonies identified as diseased in 2002 (left), and by 2004 of 156 colonies identified as diseased in 2003 (right).

Of 100 colonies which experienced tissue loss between 2002 and 2003, 52 (52.0%) were affected by dark color syndrome, 24 (24.0%) by bleaching, 10 (10.0%) by white plague, 2 (2.0%) by white pox, as well as 2 (2.0%) by “condition 1”, 1 (1.0%) by Caribbean yellow band, and 9 (9.0%) by “unknown” disease signs (Figure 31). Seventy-three of these cases (73.0%) involved only partial tissue mortality, while 27 (27.0%) were examples of complete colony mortality.

In contrast, among the 96 colonies which experienced a significant loss in tissue area between 2003 and 2004, 67 (69.8%) were affected by dark color syndrome, 6 (6.3%) by white plague, 5 colonies (5.2%) had bleaching signs, 5 colonies (5.2%) were affected by “condition 1”, 1 colony (1.0%) had evidence of cyanobacterial mat disease, 1 (1.0%) had Caribbean yellow band, 1 (1.0%) was affected by “condition 2”, 1 (1.0%) colony had signs of white band, 1 (1.0%) had white pox, and finally 8 colonies (8.3%) had “unknown” signs of disease (Figure 31).

Among the colonies which suffered tissue mortality, 87 (90.6%) were cases of partial tissue mortality and 9 were examples of complete colony mortality.

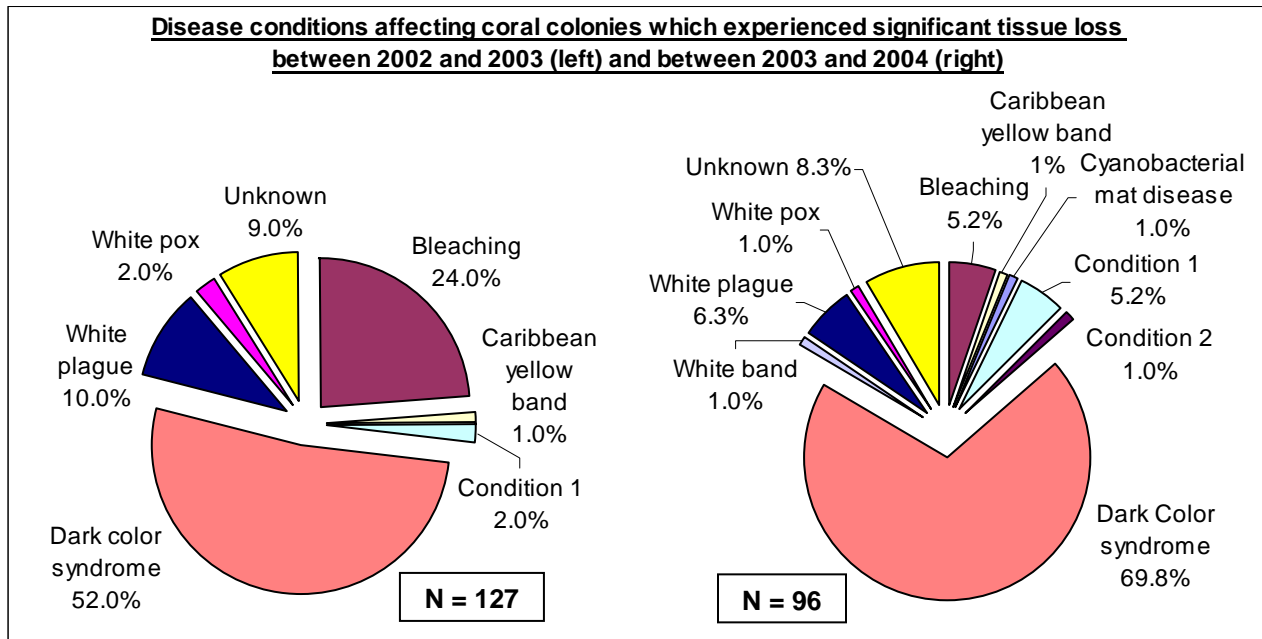


Figure 31. Diseases affecting 127 scleractinian coral colonies which experienced tissue loss between 2002 and 2003, and 96 colonies which experienced tissue loss between 2003 and 2004.

During the survey, 36 diseased coral colonies experienced complete colony mortality (all died one year after first observation): 21 (58.3%) were affected by bleaching, 5 (13.9%) by white plague, 4 (11.1%) by dark color syndrome, 1 (2.8%) by white band, 1 (2.8%) by “condition 1” and 4 (11.1%) by “unknown” disease conditions (Figure 32).

It is worth noting that 19 of the 21 bleached coral colonies which died during the duration of the survey were *Favia fragum* colonies, all located at Western Sambo Offshore Shallow Reef Stations 1 and 2. Only 3 (13.6%) bleached colonies of *F. fragum* in 2002 and 2003 remained

alive by 2004. By contrast, bleaching signs were observed on 24 colonies of other species, and this condition resulted in mortality of only 2 of them, one *Acropora cervicornis* and one *Siderastrea siderea*.

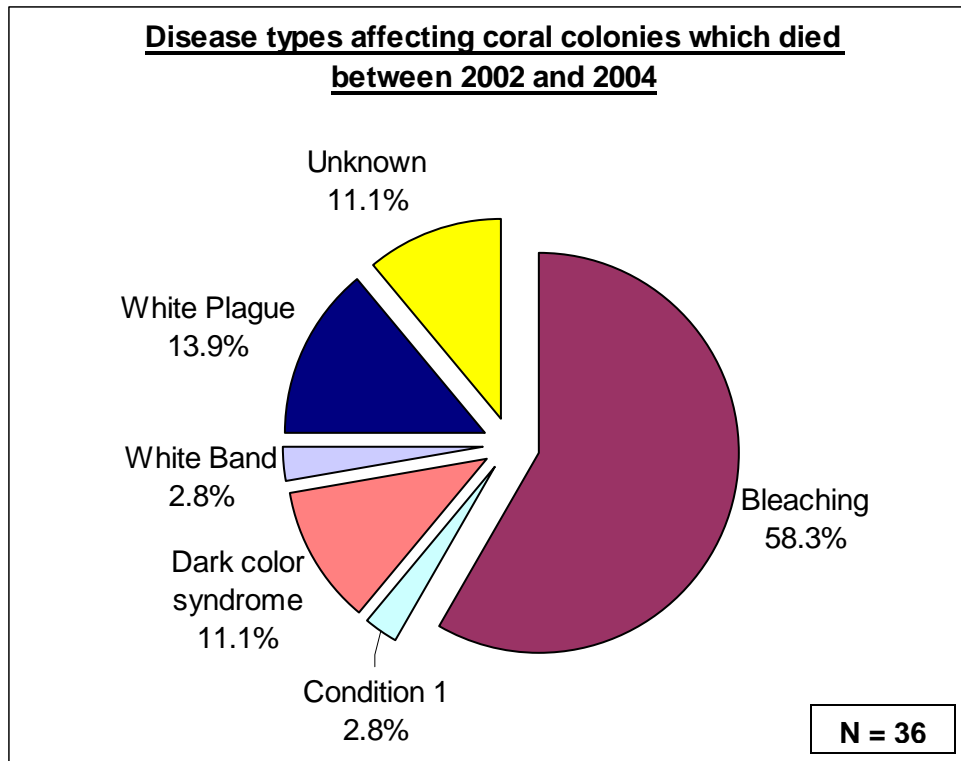


Figure 32. Disease types affecting 36 scleractinian coral colonies which died between 2002 and 2004. All colonies died 1 year after initial disease observation.

Of 137 diseased coral colonies which experienced either no significant tissue change, or a gain in live tissue area between 2002 and 2003, 103 (75.2%) were affected by dark color syndrome, 14 (10.2%) were affected by bleaching, 6 colonies (4.4%) had signs of white plague,

3 (2.2%) had growth anomalies, 3 (2.2%) were affected by “condition 1”, 1 colony was affected by “condition 2”, and 7 colonies had signs of “unknown” diseases (Figure 33).

Among 60 coral colonies which suffered no significant tissue change or a gain in live tissue area between 2003 and 2004, 40 (66.7%) were affected by dark color syndrome, 7 (11.7%) by bleaching, 4 colonies (6.7%) had signs of “condition 2”, 3 (5.0%) had growth anomalies, 2 colonies (3.3%) had signs of “condition 1”, 1 colony was affected by white band, and 3 colonies exhibited “unknown” signs of disease (Figure 33).

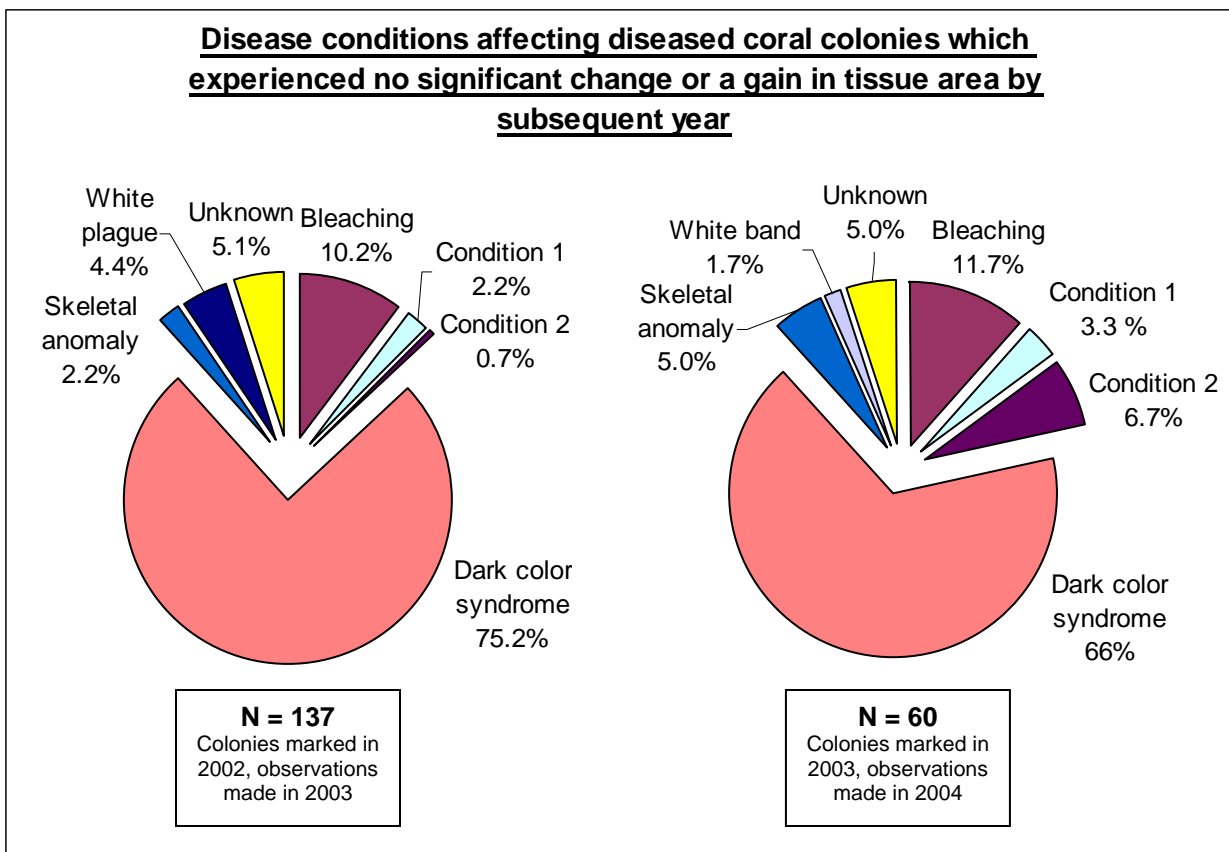


Figure 33. Disease conditions affecting 137 scleractinian coral colonies identified as diseased in 2002, and 60 identified in 2003, which experienced either no significant change in tissue area or a gain in tissue area by the subsequent survey year.

*Dark color syndrome*

Given the high prevalence of dark color syndrome among diseased colonies relative to all other conditions, a closer look at this disease and its effects is warranted. As mentioned previously, 155 coral colonies were identified with this condition in 2002. Of these, 84 (54.2%) experienced no significant changes in live tissue area by the subsequent year, 52 (33.5%) lost tissue, and 19 (12.3%) experienced significant gains in live tissue area by 2003 (Figure 34). Among 108 coral colonies identified with dark color syndrome in 2003, 38 colonies (35.2%) experienced no significant changes in live tissue area by 2004, 68 colonies (63.0%) suffered tissue mortality, and 2 colonies gained significant tissue area (1.9%) (Figure 34).

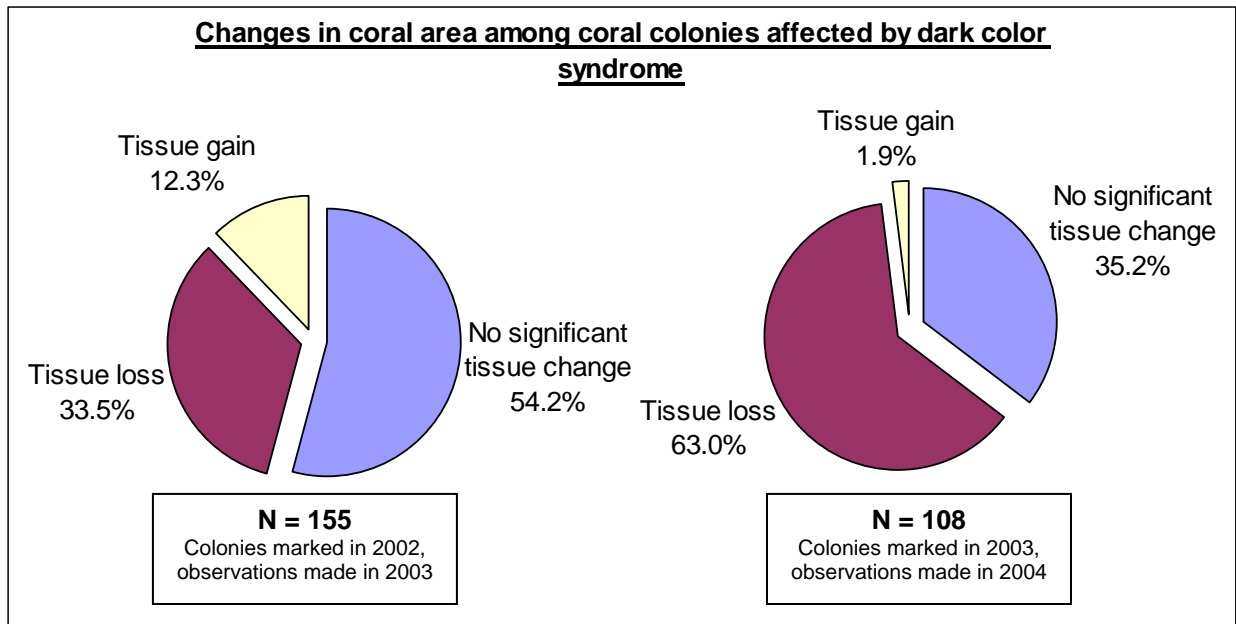


Figure 34. Changes in coral area by 2003, in 155 scleractinian coral colonies marked with dark color syndrome in 2002, and by 2004, in 108 coral colonies marked in 2003.

The considerable difference observed in the proportion of colonies suffering tissue losses due to this condition between these 2 survey years is related to the identity of the target species affected. It is evident, based on the results of this survey, that the condition known as dark color syndrome has a variable impact depending on its target coral host: of 51 colonies of *Montastrea annularis* complex observed to be affected by this condition during the duration of the survey, 94.1% experienced a significant loss of tissue (one year after disease observation). However, only 31.3% of 182 colonies of *Siderastrea siderea* and 42.4% of 26 colonies of *Stephanocoenia michelinii* affected by the condition experienced tissue mortality (Figure 35). In 2002, 80.6% of the dark color syndrome infected colonies were *S. siderea* colonies and 11.0% were *M. annularis* complex, while in 2003 only 53.7% were *S. siderea* colonies, and 31.5% were *M. annularis* complex colonies, explaining some of the observed differences in the impact of this condition throughout the survey years.

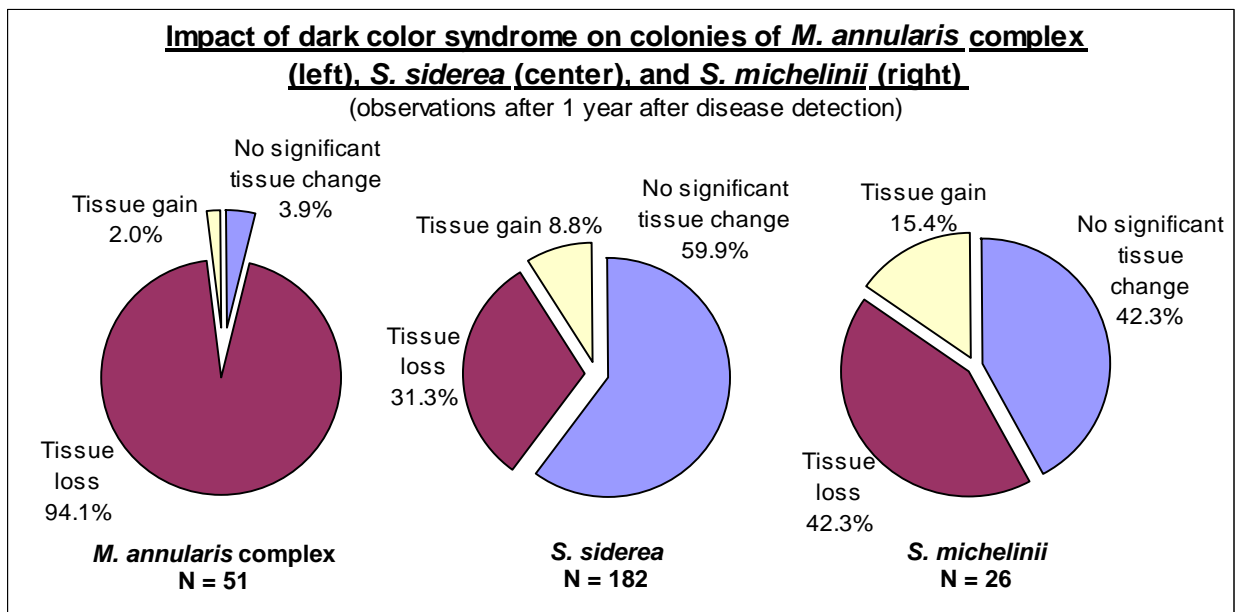


Figure 35. Differential impact of dark color syndrome on 51 colonies of *Montastrea annularis* complex (left), 182 colonies of *Siderastrea siderea* (center), and 26 colonies of *Stephanocoenia michelinii* (right).

## QUANTIFICATION OF DISEASED AREAS

Measurement of areas affected by active disease lesions were performed on all colonies identified as diseased in 2002 and 2003. Diseased areas (based on close-up photographs) totaled 7,529 cm<sup>2</sup> at all 14 stations in 2002, and 7,048 cm<sup>2</sup> in 2003. At the station level, in 2002, total coral areas with active disease ranged from 22 cm<sup>2</sup> at Tennessee Offshore Shallow Reef Station 3, to 1,393 cm<sup>2</sup> at West Turtle Shoal Patch Reef Station 3. In 2003, total coral areas affected by diseased ranged from 43 cm<sup>2</sup> at Tennessee Reef Station 3, to 2,520 cm<sup>2</sup> at Grecian Rocks Offshore Shallow Reef Station 1. Table 4 includes disease areas per station, based on close-up and overview photographs. Diseased area measurements calculated from overview photographs did not differ largely from measurements based on close-up photographs.

Table 4. Diseased area measurements (cm<sup>2</sup>) based on downward-pointed overview, as well as close-up photographs, for all 14 stations surveyed in the FKNMS in 2002 and 2003.

Stations	2002		2003	
	Downward-pointing overview (cm <sup>2</sup> )	Close-up (cm <sup>2</sup> )	Downward-pointing overview (cm <sup>2</sup> )	Close-up (cm <sup>2</sup> )
Jaap Patch Reef St. 1	620	651	169	173
Jaap Patch Reef St. 2	410	413	42	44
Western Sambo Offshore Shallow Reef St. 1	312	299	138	137
Western Sambo Offshore Shallow Reef St. 2	201	273	1,193	1,193
Cliff Green Patch Reef St. 3	356	375	173	176
Cliff Green Patch Reef St. 4	320	340	381	387
Tennessee Offshore Shallow Reef St. 2	42	44	45	45
Tennessee Offshore Shallow Reef St. 3	23	22	43	43
West Turtle Shoal Patch Reef St. 3	1,345	1,393	158	159
West Turtle Shoal Patch Reef St. 4	1,300	1,300	831	829
Grecian Rocks Offshore Shallow Reef St. 1	345	342	1,971	2,520
Grecian Rocks Offshore Shallow Reef St. 2	128	159	92	117
Porter Patch Reef St. 1	757	901	486	541
Porter Patch Reef St. 2	912	1,016	640	686
<b>Total</b>	<b>7,072</b>	<b>7,529</b>	<b>6,360</b>	<b>7,048</b>

## DISEASE-ASSOCIATED CHANGES IN CORAL AREAS

The extent of coral tissue mortality and areas recovered from disease between 2002 and 2003 was totaled for all 14 stations. Disease-associated changes in coral areas ranged between a net gain of 86 cm<sup>2</sup> at Jaap Patch Reef Station 2 and a net loss of 584 cm<sup>2</sup> of coral tissue at West Turtle Shoal Patch Reef Station 4 (corresponding to only half the station)(Figure 36). Net changes totaled 4,215 cm<sup>2</sup> of tissue lost throughout all 14 stations (Table 5), with an average of 301 cm<sup>2</sup> of net tissue mortality per station.

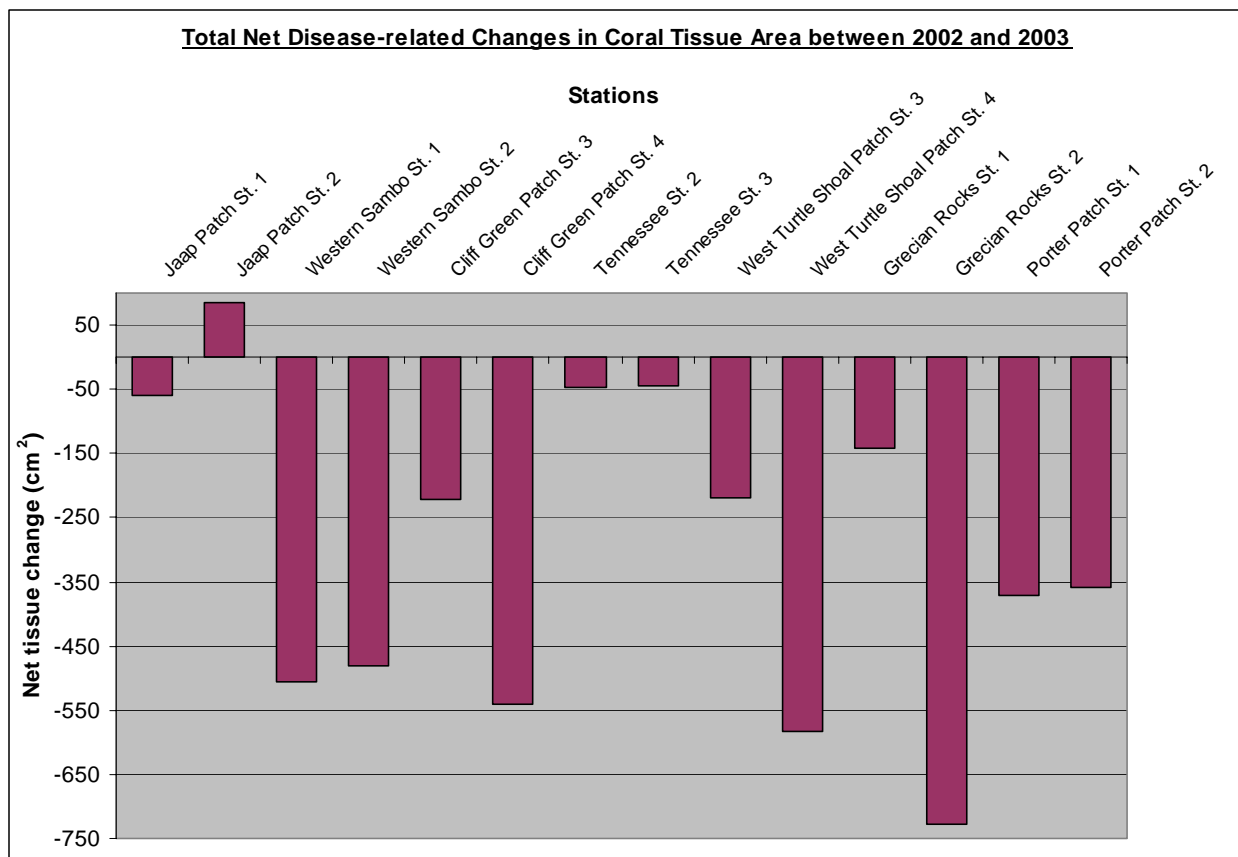


Figure 36. Total net changes in coral tissue area due to disease at 14 stations surveyed in the FKNMS between 2002 and 2003.

Table 5. Areas of scleractinian coral tissue loss or recovery due to disease conditions, and total net area changes at 14 stations surveyed in the FKNMS.

Stations	Area lost to disease	Area gained from disease recovery	Net total area change
Jaap Patch Reef St. 1	-61.7	0.0	-60.7
Jaap Patch Reef St. 2	-13.9	+99.8	+85.8
Western Sambo Offshore Shallow Reef St. 1	-506.4	0.0	-506.4
Western Sambo Offshore Shallow Reef St. 2	-480.2	0.0	-480.2
Cliff Green Patch Reef St. 3	-220.5	0.0	-220.5
Cliff Green Patch Reef St. 4	-549.8	+8.8	-540.9
Tennessee Offshore Shallow Reef St. 2	-46.1	0.0	-46.1
Tennessee Offshore Shallow Reef St. 3	-44.4	0.0	-44.4
West Turtle Shoal Patch Reef St. 3	-284.1	+65.4	-218.7
West Turtle Shoal Patch Reef St. 4	-701.3	+117.2	-584.1
Grecian Rocks Offshore Shallow Reef St. 1	-141.0	0.0	-141.0
Grecian Rocks Offshore Shallow Reef St. 2	-727.2	0.0	-727.2
Porter Patch Reef St. 1	-398.8	+27.1	-371.7
Porter Patch Reef St. 2	-359.7	+0.4	-359.3
<b>Total</b>			<b>-4215</b>

Dark color syndrome was involved in 46.2% of the tissue mortality (1,948 cm<sup>2</sup> lost). Although bleaching affected 24.0% of the colonies which experienced tissue loss between 2002 and 2003, the condition was only responsible for the loss of 409 cm<sup>2</sup> (9.7%). This disparity between the frequency of incidence and the tissue mortality is in part explained by the fact that 75% of the colonies which experienced tissue loss due to bleaching between 2002 and 2003 were *Favia fragum* colonies. Colonies of this species are characteristically small (<10 cm diameter), and hence even complete colony mortality only involved small tissue losses. White pox was involved in the loss of 866 cm<sup>2</sup> of coral tissue (20.5%), while the disease affected only 2.0% of the colonies which lost tissue between 2002 and 2003, pointing both to the severity of the condition's impact in terms of tissue loss at the colony level and its significant contribution to coral tissue mortality at the reef level relative to other diseases. A total of 745 cm<sup>2</sup> of coral tissue

were lost to white plague (17.7%), 26 cm<sup>2</sup> to Caribbean yellow band (0.6%), which affected only one colony, 19 cm<sup>2</sup> to “condition 1” (0.5%) and 202 cm<sup>2</sup> to unknown diseases (4.8%) (Figure 37).

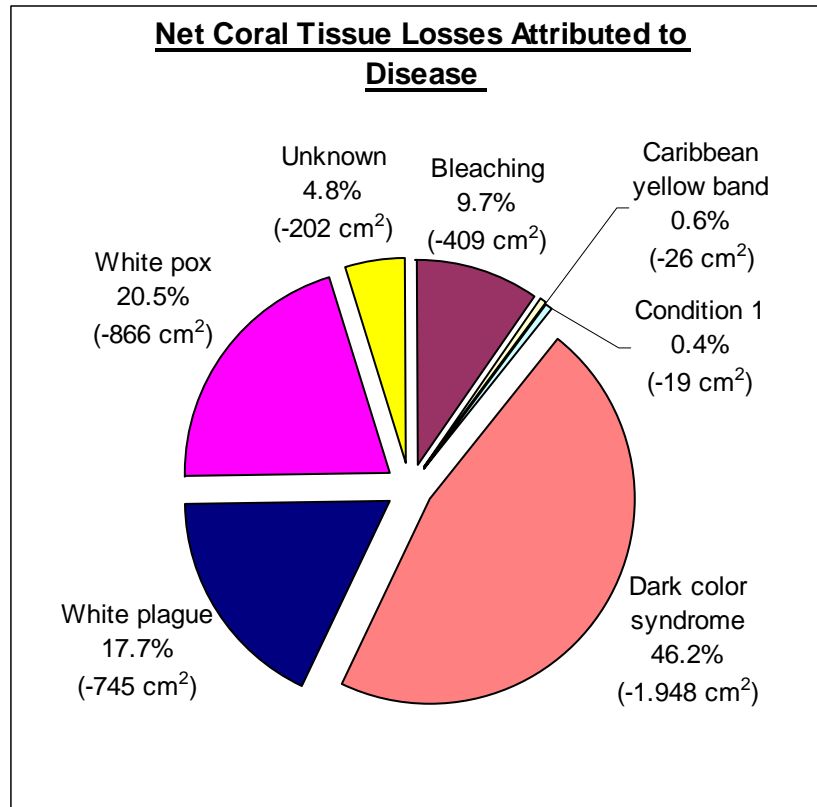


Figure 37. Percent of scleractinian coral tissue mortality attributed to 7 disease types in 14 stations surveyed in the FKNMS.

Net coral tissue losses were observed for 13 coral species. The greatest net tissue mortality was seen on *Acropora palmata* (20.5% of total tissue losses, with a loss of 866 cm<sup>2</sup>), *Montastrea annularis* complex (20.2% of losses, with 855 cm<sup>2</sup>), *Montastrea cavernosa* (19.8% of losses, with 840 cm<sup>2</sup>) and *Siderastrea siderea* (19.3% of losses, with 818 cm<sup>2</sup>). Net tissue

losses across all 14 stations for these and 9 other coral species are listed in Figure 38. Net total tissue gains from tissue recovery to disease, were observed on two coral species: *Meandrina meandrites* (1 cm<sup>2</sup>) and *Porites astreoides* (16 cm<sup>2</sup>). Table 6 summarizes the data on prevalence, severity and lethality of diseases affecting the 4 coral species which suffered most tissue loss.

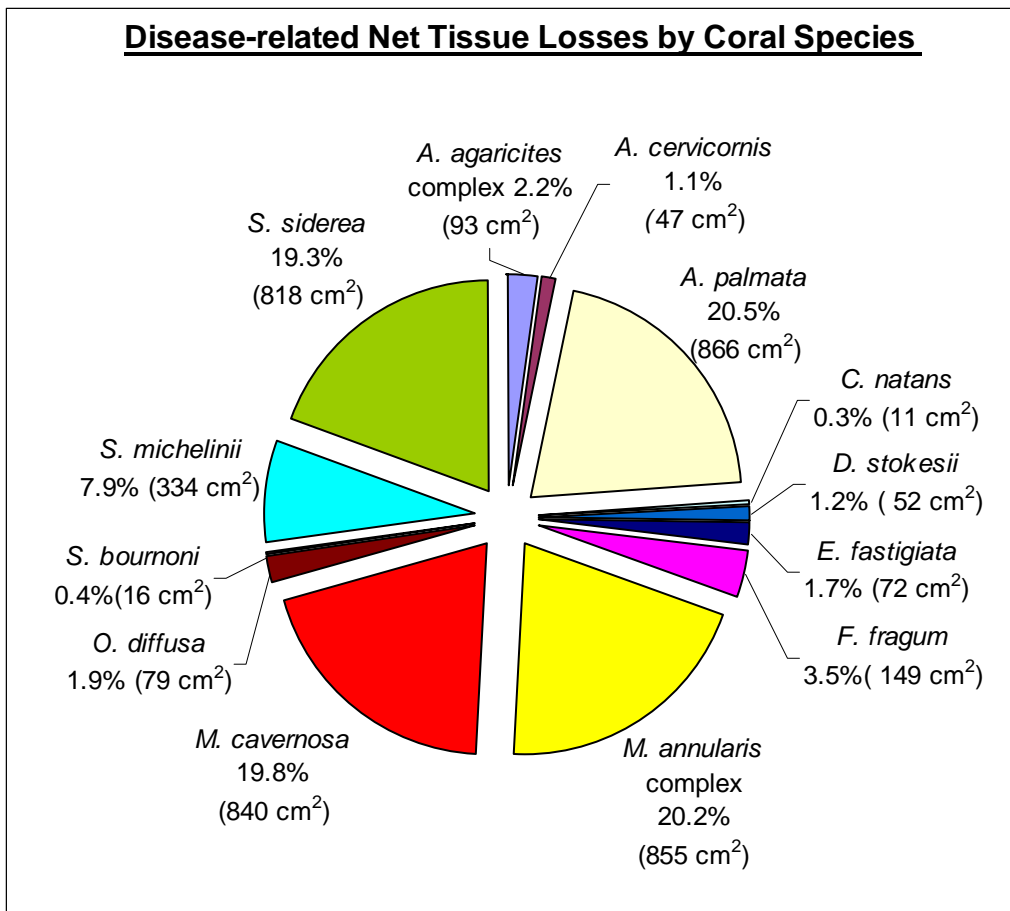


Figure 38. Disease-related net tissue losses experienced by 13 scleractinian coral species across 14 stations surveyed throughout the FKNMS.

Table 6. Prevalence, severity and lethality of diseases affecting the 4 scleractinian coral species which suffered most tissue loss during the survey. Prevalence and partial/whole mortality percents were calculated based on populations of healthy and diseased corals in the first 20m<sup>2</sup> of each station. Areas of tissue diseased and lost were calculated based on diseased coral colonies from the entire station.

Coral species	Disease prevalence				Disease severity				Disease lethality			
	2002	2003	2004	Average (± standard error)	2002		2003		2002-2003			
					Disease area (cm <sup>2</sup> )	% of species total	Disease area (cm <sup>2</sup> )	% of total	Tissue lost (cm <sup>2</sup> )	% of total	Partial mortality (% of species population)	Whole mortality (% of species population)
<i>Montastrea annularis</i> complex	7.8%	9.5%	61.5%	26.3% (±17.6)	1410.2	0.4%	2245.9	0.7%	855.3	0.3%	11.3%	0%
<i>Montastrea cavernosa</i>	6.9%	6.2%	3.7%	5.6% (±1.0)	133.1	0.1%	154.9	0.1%	839.7	0.8%	3.1%	1.2%
<i>Siderastrea siderea</i>	13.3%	4.3%	4.9%	7.5% (±2.9)	4444.9	5.6%	2740.6	4.0%	818.2	1.0%	3.2%	0.3%
<i>Acropora palmata</i>	6.7%	6.7%	16.8%	10.1% (±3.4)	248.8	1.5%	144.8	0.9%	866.0	5.1%	3.4%	0%

## PROJECTED LIVE CORAL SURFACE AREAS

Mean projected live coral surface areas were estimated from the 2002 and 2003 videos.

In 2002, mean percent coral cover ranged from 1.2% at Tennessee Offshore Shallow Reef Station 3, to 23.0% at Grecian Rocks Offshore Shallow Reef Station 1, with an average of 10.9% across all 14 stations. In 2003, the average percent cover for all 14 stations had not changed significantly, remaining static at 10.8%, with a range from 0.6% at Tennessee Offshore Shallow Reef Station 3 to 25.1% at Jaap Patch Reef Station 2 (Table 7).

Table 7. Mean percent live scleractinian coral areas ( $\pm$  standard error) for all 14 stations surveyed in the FKNMS in 2002 and 2003.

Stations	2002		2003	
	Average	St. Error	Average	St. Error
Jaap Patch Reef St. 1	17.4%	1.4	19.2%	1.2
Jaap Patch Reef St. 2	21.2%	1.6	25.1%	1.5
Western Sambo Offshore Shallow Reef St. 1	3.6%	0.8	4.0%	0.8
Western Sambo Offshore Shallow St. 2	5.3%	0.9	5.0%	0.9
Cliff Green Patch Reef St. 3	18.2%	1.6	15.9%	1.3
Cliff Green Patch Reef St. 4	14.7%	1.5	14.3%	1.3
Tennessee Offshore Shallow Reef St. 2	1.5%	0.3	1.3%	0.3
Tennessee Offshore Shallow Reef St. 3	1.2%	0.3	0.6%	0.2
West Turtle Shoal Patch Reef St. 3	13.4%	1.1	13.6%	1.1
West Turtle Shoal Patch Reef St. 4	22.9%	1.7	19.5%	1.5
Grecian Rocks Offshore Shallow Reef St. 1	23.0%	2.1	24.2%	2.3
Grecian Rocks Offshore Shallow Reef St. 2	3.8%	1.0	2.5%	0.7
Porter Patch Reef St. 1	2.1%	0.4	2.2%	0.6
Porter Patch Reef St. 2	5.0%	0.9	4.5%	0.7

The mean percent cover for the following stations appeared lower for 2003 than 2002: Western Sambo Station 2, Cliff Green Stations 3 and 4, Tennessee Stations 2 and 3, West Turtle Station 4, Grecian Rocks Station 2 and Porter Patch Station 2. Meanwhile, Jaap Stations 1 and 2, Western Sambo Station 3, Grecian Rocks Station 1 and Porter Patch Station 1 slightly increased their mean percent live coral cover between 2002 and 2003 (Figure 39). However, Wilcoxon's rank-sum tests indicate that there was no significant difference between 2002 and 2003 percent means for any of the 14 stations ( $p > 0.05$  for all stations) (Appendix III).

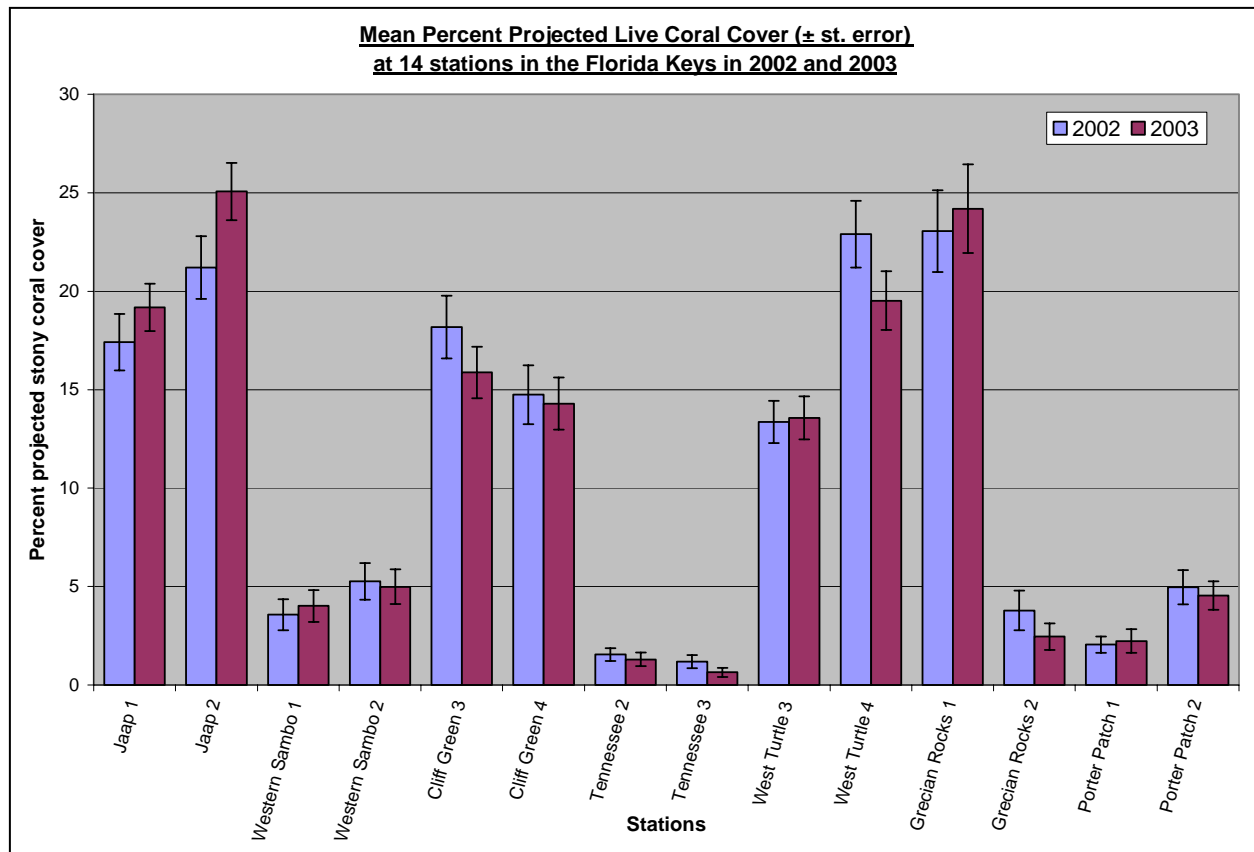


Figure 39. Mean percent projected live scleractinian coral cover ( $\pm$  standard error) at 14 stations surveyed in the FKNMS in 2002 and 2003.

Based on percent live coral cover and data on areas of active disease lesions (based on overview photographs, as seen in Table 4), it was estimated that the percent live coral area under active disease expression ranged between 0.3% at Grecian Rocks Station 1 and 4.0% at Porter Patch Station 2 in 2002, and between 0.2% at Jaap Patch Station 1 and 5.0% at Western Sambo Station 2 in 2003. Average values across all 14 stations equaled 1.7% and 1.5% in 2002 and 2003 respectively (Table 8).

Table 8. Percent of projected live scleractinian coral areas with active disease lesions in 2002 and 2003 at 14 stations surveyed in the FKNMS.

Stations	2002		2003	
	Live Coral Cover (cm <sup>2</sup> )	Percent live coral area with active disease lesions	Live Coral Cover (cm <sup>2</sup> )	Percent live coral area with active disease lesions
Jaap Patch Reef St. 1	83,600	<b>0.7%</b>	92,040	<b>0.2%</b>
Jaap Patch Reef St. 2	102,162	<b>0.4%</b>	120,803	<b>0.0%</b>
Western Sambo Offshore Shallow Reef St. 1	16,055	<b>1.9%</b>	18,057	<b>0.8%</b>
Western Sambo Offshore Shallow Reef St. 2	25,235	<b>0.8%</b>	23,963	<b>5.0%</b>
Cliff Green Patch Reef St. 3	75,616	<b>0.5%</b>	66,038	<b>0.3%</b>
Cliff Green Patch Reef St. 4	60,447	<b>0.5%</b>	58,600	<b>0.6%</b>
Tennessee Offshore Shallow Reef St. 2	7,119	<b>0.6%</b>	6,003	<b>0.7%</b>
Tennessee Offshore Shallow Reef St. 3	5,295	<b>0.4%</b>	2,853	<b>1.5%</b>
West Turtle Shoal Patch Reef St. 3	58,779	<b>2.3%</b>	59,665	<b>0.3%</b>
West Turtle Shoal Patch Reef St. 4	48,090	<b>2.7%</b>	40,992	<b>2.0%</b>
Grecian Rocks Offshore Shallow Reef St. 1	105,107	<b>0.3%</b>	110,279	<b>1.8%</b>
Grecian Rocks Offshore Shallow Reef St. 2	17,561	<b>0.7%</b>	11,406	<b>0.8%</b>
Porter Patch Reef St. 1	10,024	<b>7.6%</b>	10,887	<b>4.5%</b>
Porter Patch Reef St. 2	22,846	<b>4.0%</b>	20,897	<b>3.1%</b>
<b>Mean</b>		<b>1.7%</b>		<b>1.5%</b>

Similarly, based on percent live coral cover and data concerning area changes due to disease, it was estimated that between 2002 and 2003 the percent of live coral tissue cover which was lost due to disease, or gained by disease recovery, ranged between -3.71% (of 2002 live coral cover) at Porter Patch Station 1, and +0.08% (of 2002 live coral cover) at Jaap Patch Station 2 (Table 9). The total tissue area lost to disease across all 14 stations, which equaled 4,215 cm<sup>2</sup>, constitutes a loss of 0.7% of 2002 projected live coral cover for all stations combined. This loss is well inside the margin of error calculated for projected coral cover for all stations, which also demonstrated no significant change in coral cover between 2002 and 2003.

Table 9. Percent of the 2002 projected live scleractinian coral cover which was lost due to disease or recovered from disease by 2003, at 14 stations surveyed throughout the FKNMS.

Stations	Percent of 2002 live coral cover lost or gained by 2003 due to disease
Jaap Patch Reef St. 1	- 0.07%
Jaap Patch Reef St. 2	+ 0.08%
Western Sambo Offshore Shallow Reef St. 1	- 3.15%
Western Sambo Offshore Shallow Reef St. 2	- 1.90%
Cliff Green Patch Reef St. 3	- 0.29%
Cliff Green Patch Reef St. 4	- 0.89%
Tennessee Offshore Shallow Reef St. 2	- 0.65%
Tennessee Offshore Shallow Reef St. 3	- 0.83%
West Turtle Shoal Patch Reef St. 3	- 0.37%
West Turtle Shoal Patch Reef St. 4	- 1.21%
Grecian Rocks Offshore Shallow Reef St. 1	- 0.13%
Grecian Rocks Offshore Shallow Reef St. 2	- 4.14%
Porter Patch Reef St. 1	- 3.71%
Porter Patch Reef St. 2	- 1.57%

## COLONY SIZES

With information from the colony count survey on the size class distribution of all coral colonies in the half-transect, and from the diseased coral survey on the size of all diseased coral colonies, the prevalence of disease as a function of colony size was analyzed. Results indicate that total coral colony population sizes are skewed towards smaller colonies, with 55% of the colonies falling in the 3-10 cm<sup>2</sup> (maximum diameter) range, 37% in the 10-50 cm<sup>2</sup> range, and 8% in the > 50 cm<sup>2</sup> range. In contrast, diseased colonies (including all 3 survey years) were distributed as follows: 28% of diseased colonies fell within the 3-10 cm size class, 60% were middle-sized colonies (10-50 cm), and 12% fell within the > 50 cm class (Figure 40). Chi-

Square analyses confirm that the differences in size class distribution between diseased and total population corals are statistically significant ( $G^2=88.41$ ,  $DF=2$ ,  $p<0.0001$ ) (Appendix IV).

Relative to the population of their size class, large and middle-sized colonies were disproportionately affected by disease (21% and 23% prevalence respectively) (Figure 40). This leads to the general observation that larger individuals are more susceptible to disease than smaller corals.

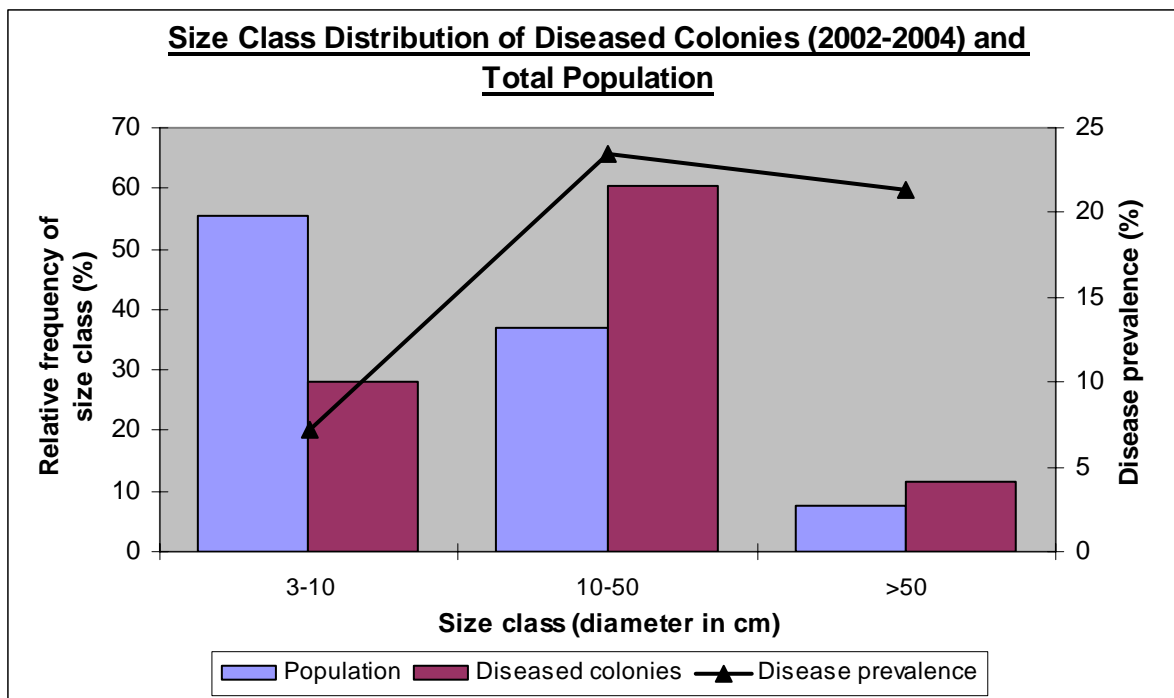


Figure 40. Comparison of size class distribution of diseased colonies (all diseases during all 3 survey years) and coral population at all 14 stations surveyed in the FKNMS, and relative prevalence of disease per size class.

The following coral species had a large enough number of affected colonies to conduct Chi-Square analyses of colony size distribution at the species level (for all diseases combined):

*Montastrea annularis* complex, *Stephanocoenia michelinii*, *Siderastrea siderea*, *Montastrea cavernosa*, and *Agaricia agaricites* complex. ChiSquare analyses indicate that size class distributions in diseased and population colonies are significantly different for *S. siderea*, *S. michelinii*, *M. annularis* complex, and *A. agaricites* complex colonies ( $G^2=222.88$ ,  $DF=2$ ,  $p<0.0001$ ;  $G^2=19.22$ ,  $DF=2$ ,  $p<0.0001$ ; and  $G^2=46.70$ ,  $DF=2$ ,  $p<0.0001$ ,  $G^2=6.78$ ,  $DF=2$ ,  $p=0.0337$  respectively), but not for *M. cavernosa* or ( $G^2=2.27$ ,  $DF=2$ ,  $p=0.3207$ ) (Appendix IV).

Although the majority of colonies of *S. siderea* and *S. michelinii* affected by disease were middle-sized colonies, in relative terms, larger-sized colonies were preferentially affected, with 54% and 28% of large colonies affected respectively (Figures 41 and 42).

Among colonies of *Montastrea annularis* complex, middle-sized colonies appeared to be relatively more affected by disease than small or large colonies (Figure 43). However, it should be noted that discerning distinct colony boundaries in this species can be far more difficult than for other coral species. Hence, it is possible that this pattern of disproportionate disease prevalence among middle-sized colonies (in contrast to large ones, as is the case with among other coral species) may be an artifact resulting from the observer error which could conceivably lie in assessments of colony size for both healthy and diseased colonies for this species.

Among colonies of *Agaricia agaricites* complex, a species for which no large colonies were found, the size class distribution of the population is skewed towards smaller colonies, yet middle-sized colonies were disproportionately more affected than smaller ones (Figure 44)

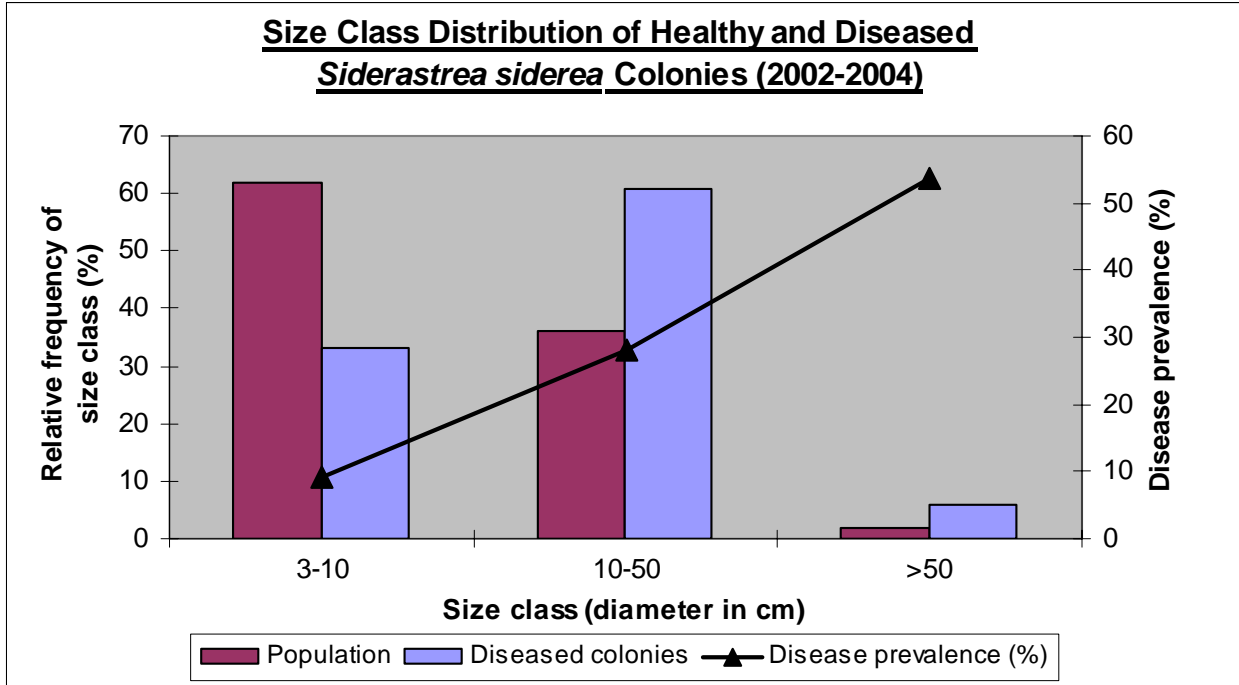


Figure 41. Size class distribution of 2002-2004 *Siderastrea siderea* diseased colonies (all disease conditions) and total population of the species, and disease prevalence per size class.

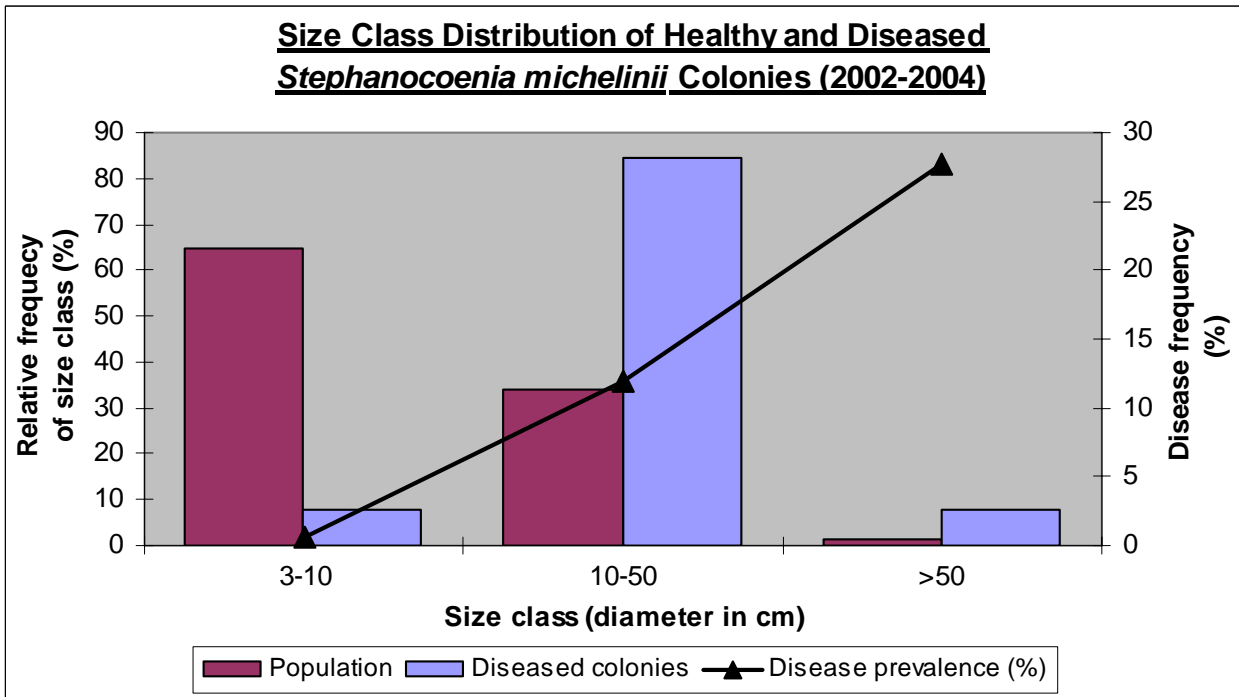


Figure 42. Size class distribution of 2002-2004 *Stephanocoenia michelinii* diseased colonies (all disease conditions) and total population of the species, and disease prevalence per size class.

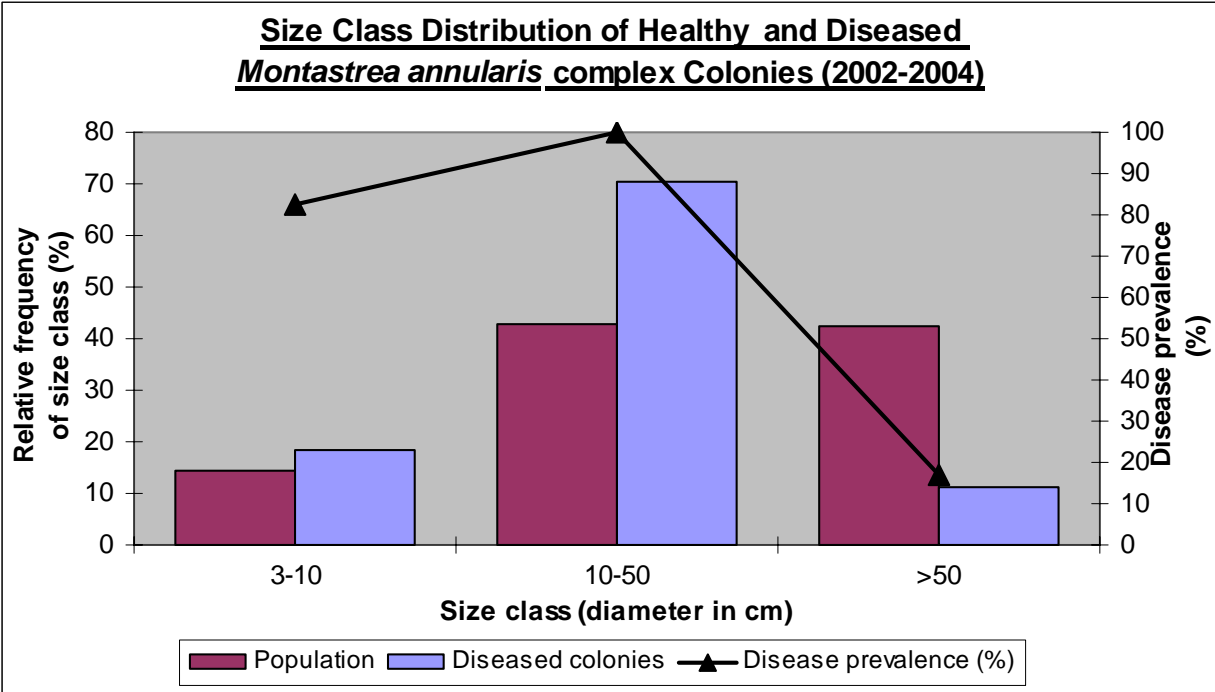


Figure 43. Size class distribution of 2002-2004 *Montastrea annularis* complex diseased colonies (all disease conditions) and total population of the species, and disease prevalence per size class.

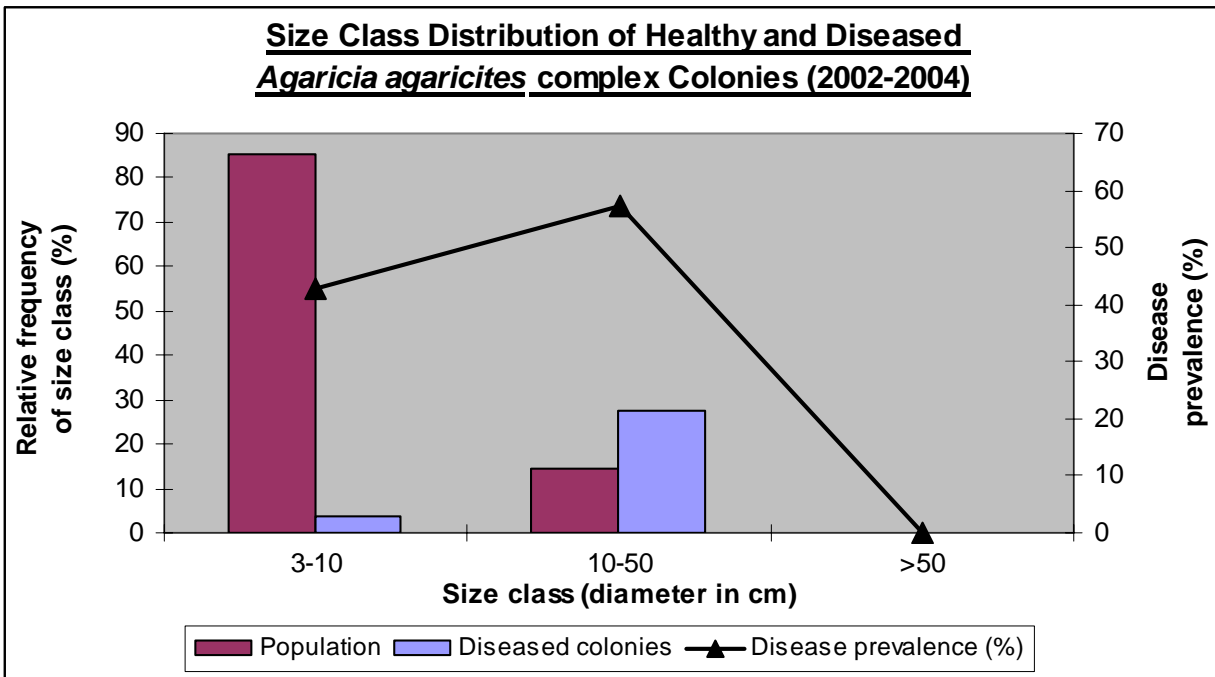


Figure 44. Size class distribution of 2002-2004 *Agaricia agaricites* complex diseased colonies (all disease conditions) and total population of the species, and disease prevalence per size class.

Dark color syndrome was the only disease with a large enough number of affected colonies to conduct Chi-Square analyses of colony size distribution. Results show statistically significant differences in the size class distribution of healthy *versus* dark color syndrome diseased *Siderastrea siderea*, *Stephanocoenia michelinii* and *Montastrea annularis* complex colonies ( $G^2=39.92$ ,  $DF=2$ ,  $p<0.0001$ ;  $G^2=17.42$ ,  $DF=2$ ,  $p=0.0002$  and  $G^2=46.70$ ,  $DF=2$ ,  $p<0.0001$ ) (Appendix IV). Results relating specifically to dark color syndrome mirror those presented previously for all combined diseases:

- Though the majority of colonies affected by the disease among the species *Siderastrea siderea* and *Stephanocoenia michelinii* were middle-sized colonies, in relative terms, dark color syndrome disproportionately affected larger-sized colonies, with 45% and 28% of large colonies affected respectively (Figures 45 and 46).
- Among colonies of *Montastrea annularis* complex, middle sized colonies were significantly more affected than small or large colonies, with 98% of disease prevalence in the middle-sized class (Figure 47). Here again, it should be noted that difficulties in distinguishing colony boundaries and assessing colony size for this species may have contributed to the differences observed in target colony size between this species and *Siderastrea siderea* and *Stephanocoenia michelinii*.

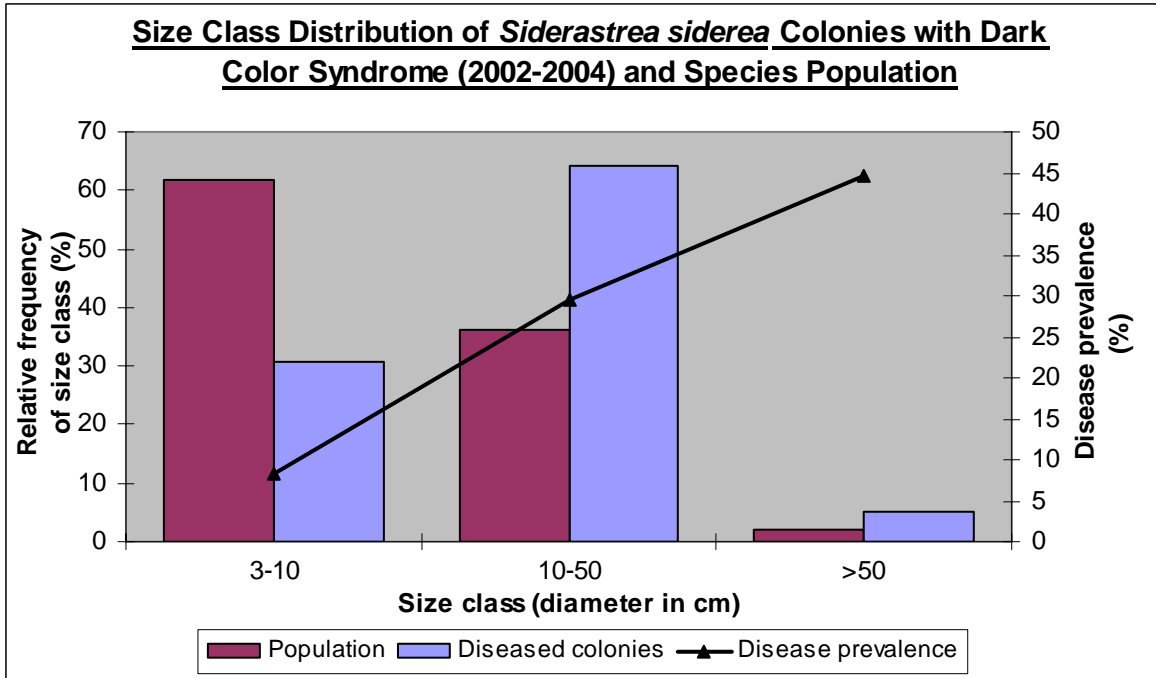


Figure 45. Comparison of size class distribution of *Siderastrea siderea* colonies affected by dark color syndrome (2002-2004) and the general population for that species, and relative disease prevalence per size class.

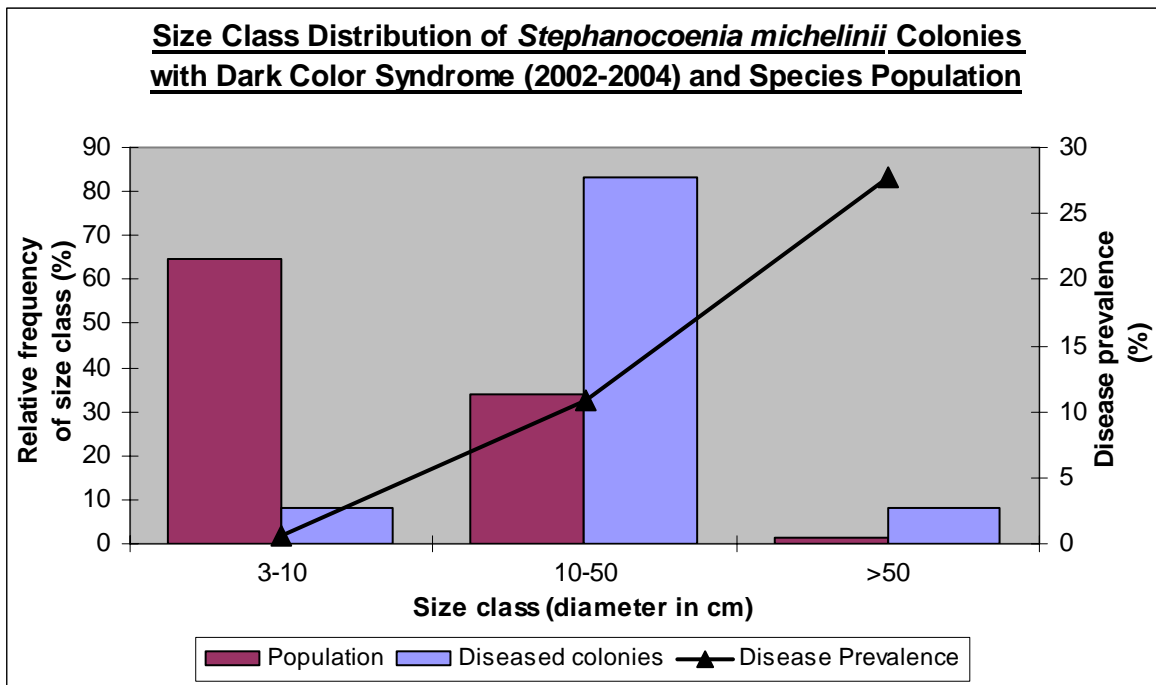


Figure 46. Comparison of size class distribution of *Stephanocoenia michelinii* colonies affected by dark color syndrome (2002-2004) and the general population for that species, and disease prevalence per size class.

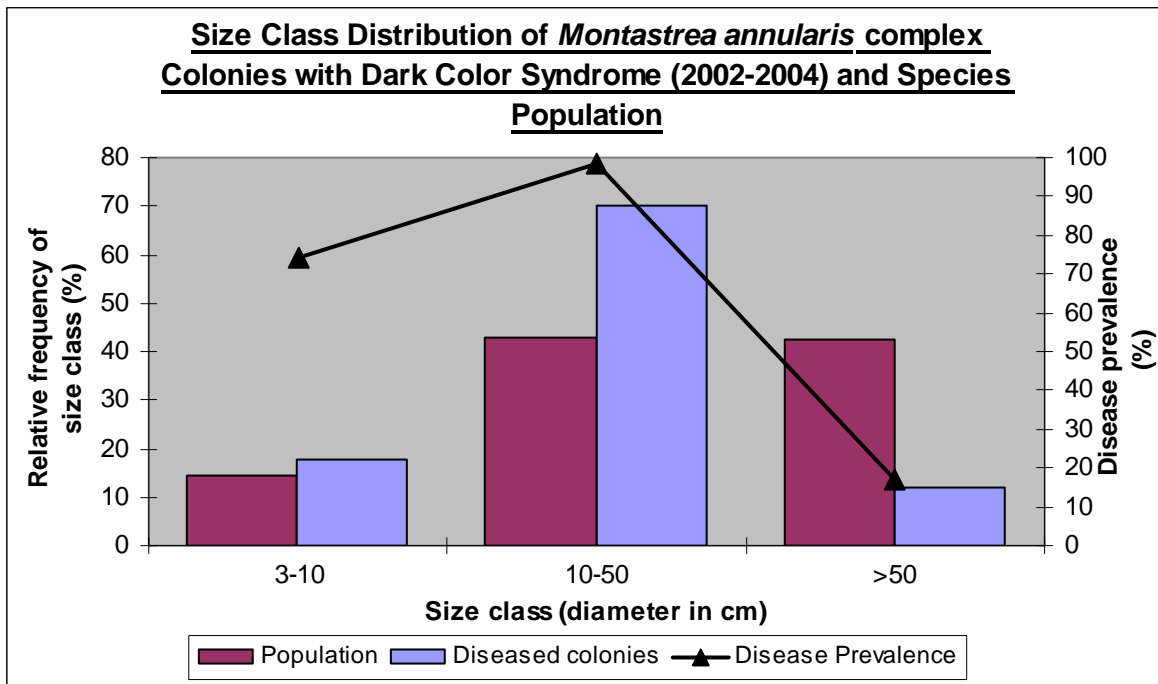


Figure 47. Comparison of size class distribution of total *Montastrea annularis* complex colonies affected by dark color syndrome (2002-2004) and the general population for that species, and disease prevalence per size class.

## ENVIRONMENTAL CONDITIONS AND POPULATION FACTORS

No significant correlations were found between the prevalence of disease at each station throughout the 3 years in which the study was conducted and either of the following factors:

- Average depth at the station
- Water temperature, calculated as the mean of daily temperatures measured at the site during: a) the 30 days prior to the disease survey date, and b) all days of the year prior to the survey date. The data was only available for 2003 and 2004, and hence was only compared to the prevalence of disease for those two years.

- Mean percent cover of scleractinian corals

- H' Shannon-Weiner diversity index

Similarly, no significant correlations were found specifically between the prevalence of dark color syndrome at each station throughout the 3 survey years and either of the following factors:

- Average depth at the station
- Water temperature, calculated as the mean of daily temperatures measured at the site during: a) the 30 days prior to the disease survey date, and b) all days of the year prior to the survey date. This was compared to the prevalence of dark color syndrome for 2003 and 2004.
- Mean percent cover of the 3 species considered as main target species: *Siderastrea siderea*, *Montastrea annularis* complex, and *Stephanocoenia michelinii*. The average of 2002 and 2003 percent covers was used in these calculations, since it was established that they were not significantly different.
- H' Shannon-Weiner diversity index
- Relative frequency of the 3 target species. Since colony counts were only available for the first 20m<sup>2</sup> of the offshore side of the station, only dark color syndrome cases located within that same area were included in calculations.

## DISCUSSION

### DISEASE DYNAMICS

Over the past 20 years there has been a plethora of studies describing increases in coral disease occurrence throughout the wider Caribbean. Many of these studies report on disease epizootics, which we now know can have devastating effects on the populations of target species (Dustan 1977; Gladfelter 1982; Dustan and Halas 1987; Smith *et al.* 1996; Aronson and Precht 1997; Bruckner and Bruckner 1997; Bruckner *et al.* 1997; Nagelkerken *et al.* 1997a; Nagelkerken *et al.* 1997b; McClanahan and Muthiga 1998; Richardson *et al.* 1998a; Slattery 1999; Richardson *et al.* 2001; Miller *et al.* 2002; Patterson *et al.* 2002; Sutherland and Ritchie 2004). Fewer reports document the general background abundance of numerous diseases over large geographic distributions and their increase over time (Garzon-Ferreira *et al.* 2001; Porter *et al.* 2001). Between 1996 and 1998 for example, Porter *et al.* (2001) observed a dramatic increase in the number of localities and the number of species with coral disease in the Florida Keys. They noted 404% more stations and 218% more species affected by disease in 1998 than in 1996. In a similar series of surveys conducted on Colombian reefs during the 1990s, Garzon-Ferreira *et al.* (2001) also described an increase in the number of disease types and species affected. Yet, most of these reports are based on surveys which score only the presence or absence of disease within survey areas. They do not provide information on the percent of colonies in the population that are affected by disease (prevalence), the amount of tissue affected (severity), or the amount of partial or whole mortality caused by the conditions (lethality).

The initial years of the Coral Reef Monitoring Project (1996-2000), provided important information attesting to the increases in disease diversity and distribution in the Florida Keys.

However, due to the type of surveys conducted by the CRMP, no information was available to quantify how common diseases were among colonies on the reef, nor the lethality of disease types. Hence the ultimate impact of these diseases on the health of the reef remained unknown. By following all diseased coral colonies through time on 14 of the CRMP stations in the Florida Keys, and by comparing information on the number of diseased coral colonies with the number of coral colonies on these same reefs, quantification of disease prevalence, severity and lethality was achieved. These data provide an answer to the fundamental question of whether coral disease is significantly affecting coral survival in the Florida Keys. Other than a few studies which focus exclusively on specific diseases (Edmunds 1991; Bruckner *et al.* 1997; Cervino *et al.* 2001; Kim and Harvell 2002; Borger 2003), there exists very little data in the literature to compare these results with, since no other study, has systematically followed the fate of individual diseased colonies through time on a long-term basis and within the context of a thorough whole ecosystem survey.

In summary, the prevalence of disease among all species was 0.4 diseased coral colonies per m<sup>2</sup> or 7.1% of the population diseased in 2002, 0.3 diseased coral colonies per m<sup>2</sup> or 4.0% of the population in 2003, and 0.5 diseased coral colonies per m<sup>2</sup> or 8.2% of the population in 2004. In quantitative terms, the prevalence of diseases at the community level in the Florida Keys is similar to the total average prevalence of disease of 5.3%, calculated by Weil (2004) for 28 reef sites throughout the wider Caribbean between 1999 and 2002. Disease lethality was low, with only 12% of the colonies diseased in 2002 and 2003 resulting in complete colony mortality by the end of the survey (1% of the coral population), and 41% experiencing partial mortality (3% of the coral population). The remaining 47% of diseased colonies showed either no change in live tissue (78%) or a gain in tissue cover (22%).

Considering previous CRMP reports indicating massive increases in disease during the late 1990s, a high incidence of disease or a continued increase in disease occurrence during the initial years of the 21<sup>st</sup> century was also expected. The indices of disease extent were indeed slightly higher in 2004 than when the survey was started in 2002. However, disease prevalence actually decreased between 2002 and 2003, indicating fluctuations in the patterns of disease incidence from year to year. Indeed, baseline patterns and trends in disease expression may be more complex and difficult to predict than expected.

Some of the complexity in annual patterns of disease incidence and prevalence can be appreciated in analyses of disease persistence at the colony level. This study showed that 40-50% of disease cases did not persist one year after initial observation, with another 10% ceasing two years after initial observation. Seventeen cases were observed in which disease signs disappeared, but reappeared in a subsequent survey year. Simultaneously, new disease infections emerged every year with widely variable incidence levels (62 new cases in 2003 *versus* 202 new cases in 2004), proving this to be a highly dynamic phenomenon. It is of critical importance to determine whether these annual fluctuations in disease incidence and prevalence are part of the general upward trend shown by Porter *et al.* (2001) during the late 1990s, or are otherwise random fluctuations, within an overall dynamic equilibrium. This question will only be answered through the continuation of this yearly disease survey over a longer period of time.

Analyses of percent live coral cover at each station can be very indicative of trends in the health of the reef. It is very noteworthy that, during the period of time in which Porter *et al.* (2001) noted the increases in both stations and coral species exhibiting disease, that is, 1996 to 1998, they also noted statistically significant decreases in the scleractinian coral cover at 7 of the stations which were surveyed in the current study (Jaap Patch Station 1, Cliff Green Station

3, Western Sambo Stations 1 and 2, West Turtle Patch Station 3 and Grecian Rocks Stations 1 and 2), while the other 7 experienced no significant change (Porter *et al.* 2002). However, statistical analysis of coral cover data for 2002 and 2003 indicates that none of the 14 stations surveyed experienced a significant change in coral cover between 2002 and 2003. Furthermore, unpublished CRMP data concerning percent coral cover for these same stations for the period between 1999 and 2003 indicates that most of the stations have approximately maintained their coral cover since 1999/2000, or even experienced slight increases in coral cover (Figure 48). This is a highly important observation, because, although the number of locations surveyed is limited, their geographic distribution, reef type, and species composition are representative of the coral reefs of the Florida Keys, and there is no reason to believe these 14 stations would behave differently from the rest of the Sanctuary's reefs. These trends in coral cover are indicative of an encouraging scenario of improvement in general reef health during recent years, and certainly contrast with the high rates of coral loss seen on these reefs during the late 1990s.

The results of quantitative analysis of disease-associated tissue loss are very much compatible with the observations of no-change in coral cover at any of the stations surveyed between 2002 and 2003. The net coral tissue loss caused by disease during this study was found to be a mere 0.4 m<sup>2</sup> between 2002 and 2003 for all stations combined. This constitutes only 0.7% of the 2002 projected live coral cover for the 14 stations surveyed, a loss well within the margin of error for coral cover estimates for the same stations. If disease had been shown to cause extensive amounts of tissue loss throughout the surveyed stations, the unlikely scenario of a very high coral recruitment rate and significant new coral tissue growth would have had to take place simultaneously to explain a lack of change in coral cover at all the stations. Instead, these results demonstrate of a rate of tissue loss to disease that is insignificant enough to fall below the

threshold of changes detectable with the techniques used to monitor coral cover. Ultimately, what this means is that although background levels of coral disease throughout the Sanctuary may appear significant, at the moment, under non-epizootic conditions, these diseases are not having a major impact on coral survival.

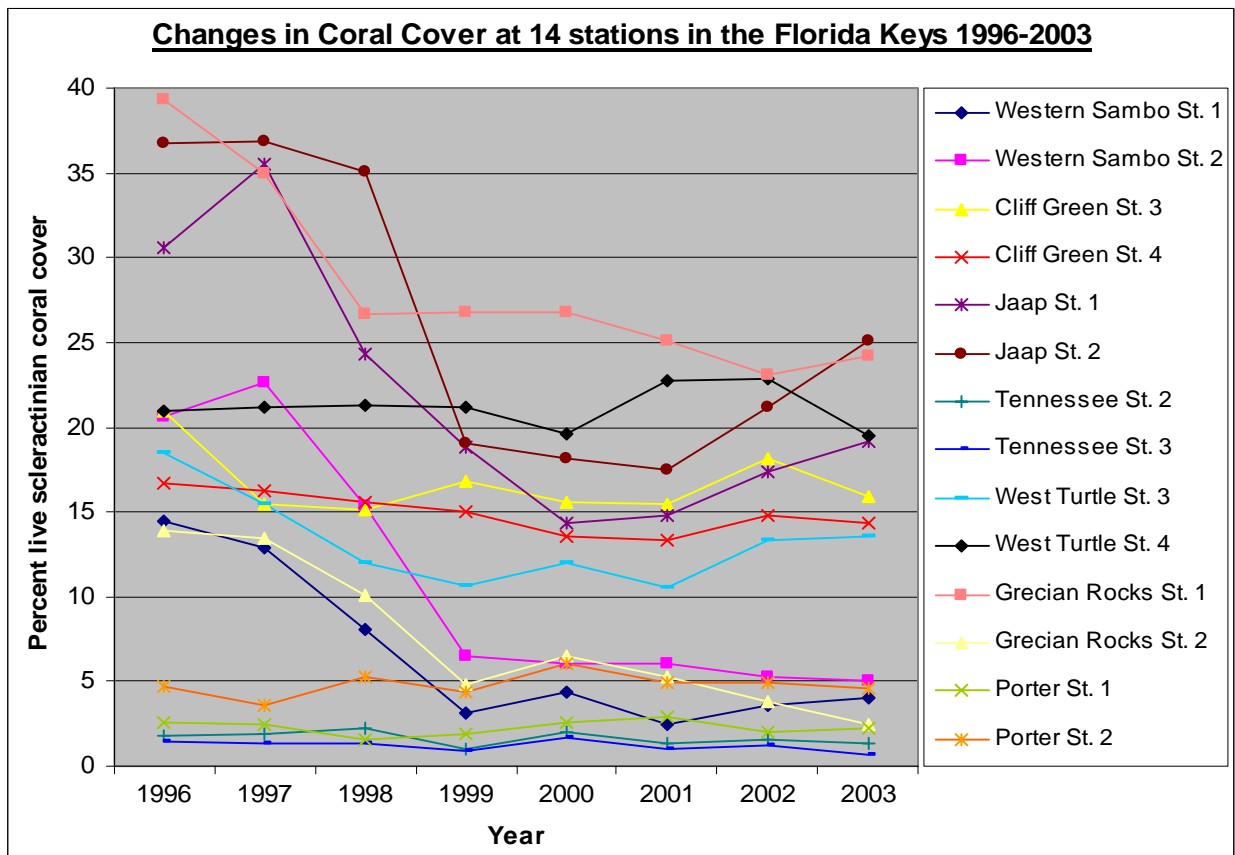


Figure 48. Changes in percent live scleractinian coral cover at 14 stations in the Florida Keys between 1996 and 2003.

That said, there are many dimensions to coral disease, and what we do not know about the range of effects of emerging coral diseases far exceeds our current and limited understanding. The impacts of coral diseases, beyond simple tissue loss, may be felt in the long-term on many

other levels that have not been quantified. These may potentially prove to pose serious threats at the reef community level. For example, disease may have consequences on reproduction or fecundity, or species-specific disease pressures may lead to shifts in species abundance or loss of diversity. Examples of this variety of impacts were observed in St. Croix, USVI, where changes brought about by white band disease in the late 1970s included a decrease in the structural complexity of the reef surface (Gladfelter 1982), or in the Belizean Barrier Reef, where the same disease caused a massive *Acropora*-to-*Agaricia* transition during the late 1980s (Aronson and Precht 1997).

Disease was observed on a variety of species during this survey, although disease pressures were not distributed evenly across species. Of the 32 scleractinian coral species found during this survey, 20 were affected by 8 known disease types and 2 potentially new disease types. This constitutes 62.5% of the species present in the 14 stations surveyed in 2002 and 2003. Although this survey was conducted over an extensive geographical area, species susceptibility did not vary greatly over the spatial scale. Furthermore, unlike other reports of increasing species susceptibility (Garzon-Ferreira *et al.* 2001; Porter *et al.* 2001), no upward trend was detected in species susceptibility over time. The total number and identity of the species affected remained very similar during the 3 years in which this survey was conducted. Three species were only found to be infected during one of the survey years, and 5 species were only represented by a single diseased colony. Two species, *Siderastrea siderea* and *Montastrea annularis* complex represented more than 60% of the diseased colonies during each year of the survey. These two species, *M. annularis* complex and *S. siderea*, in addition to *Acropora palmata* and *Montastrea cavernosa*, accumulated the greatest tissue losses between 2002 and 2003, with 19-20% of the tissue losses in each case. From an ecological perspective, these two observations are significant

because they indicate that: a) disease pressures are species-specific, and b) the species suffering the heaviest burden of tissue loss are among the four most important reef building species in the Florida Keys. These are massive or structurally important coral species, which are essential to reef framework formation and stability (Glynn 1973; Ginsburg *et al.* 1996). If disease pressures persist for many years with the same species-specificity, as mentioned previously, long-term effects could be seen on species richness, three-dimensional structure, or any number of as-yet unidentified characteristics.

Furthermore, disease does not need to result in complete colony mortality for the effects of tissue loss to be considered a stress and a long-term threat. Many of the diseased colonies in this survey only suffered losses of a few centimeters of tissue. As small as these tissue losses may have been at the individual colony level, the impacts of partial mortality on corals are not well understood. In corals, like other colonial organisms, disease and mortality can occur at the individual polyp level or at the colony level, which further complicates the understanding of their dynamics (Weil 2004). It is known that corals usually delay reproduction until they have achieved sufficient size (Szmant 1991). Furthermore, regeneration of damage lesions has been found to affect colony reproduction and fecundity due to a reallocation of energy resources (Rinkevich and Loya 1989; Van Veghel and Bak 1994). Hence, partial mortality may still have significant consequences for reproduction by reducing fecundity at the polyp level, reducing total colony reproductive output, or reducing colony size to below the reproductive threshold (Weil 2004). Although this study shows that lethality of coral diseases in the Florida Keys was low during the years in which this survey was conducted, approximately 53% of all diseased colonies suffered some degree of tissue mortality. Though this constitutes only 4-5% of the coral population, in the long-term, the continued persistence of disease over many years resulting in

small but widespread partial mortality, could have unexpected consequences at the reef population level.

## DISEASE TYPES

The following is a brief summary of the most common coral diseases observed during this study, their impact on target species, and an assessment of their influence on coral reef health.

### *Dark color syndrome*

Dark color syndrome is considered a relatively “new” disease, although it has become, in recent years one of the most common diseases throughout the wider Caribbean. The observations made in this study concerning dark color syndrome seem to be in accordance with recent reports which indicate an increasingly greater distribution and higher prevalence of this condition throughout the Caribbean (Garzon-Ferreira and Gil-Agudelo 1998; Gil-Agudelo 1998; Cervino *et al.* 2001; Garzon-Ferreira *et al.* 2001; Weil *et al.* 2004b). During this survey, examples of dark color syndrome were found in all 14 stations surveyed throughout the Florida Keys, and 65-82% of all colonies identified as diseased constituted cases of infection with this condition.

During this survey, the syndrome was observed in 6 scleractinian coral species, though *Montastrea annularis* complex, *Siderastrea siderea* and *Stephanocoenia michelinii* were the principal target species. This observation is consistent with previous reports of the disease (Garzon-Ferreira and Gil-Agudelo 1998; Gil-Agudelo and Garzon-Ferreira 2001; Weil *et al.* 2004b). Together, *M. annularis* complex and *S. siderea* accounted for 85-93% of dark color

syndrome cases between 2002 and 2004, a result strikingly similar to the 94% reported by Gil-Agudelo and Garzon-Ferreira (2001) for those 2 species in Colombian reefs. In this study, up to 59% of the *M. annularis* complex colonies and 13% of the *S. siderea* colonies in the population were found to be affected by this condition. In comparison, reports of dark color syndrome in Colombia indicate a prevalence of 28% of among *S. siderea* colonies and 17-20% among *M. annularis* complex colonies (Garzon-Ferreira and Gil-Agudelo 1998; Gil-Agudelo 1998); in Dominica 100% of all *S. siderea* colonies surveyed in 2000 were infected with the disease (Borger 2003); and in Puerto Rico prevalence ranged from 4-17% among susceptible species (Weil *et al.* 2004b).

It has been repeatedly suggested that dark color syndrome may be more prevalent during periods of warmer water temperature, and may disappear during the winter months (Gil-Agudelo and Garzon-Ferreira 2001; Borger 2003). Given the sampling schedule of this survey, visiting the reefs only during summer months, it was not feasible to make observations regarding the seasonality of this condition. It is possible that some of the infected colonies observed during this study maintained lesions that persisted during the winter season into successive survey years. However, it is more likely that colonies showing dark color syndrome lesions one or two years after initial detection constitute cases of disease reemergence during the summer survey months. Borger (2003) also reported that dark color syndrome signs could disappear, and sometimes reappear again. During this study, more than half of the colonies marked with dark color syndrome in 2002 and 2003 were no longer diseased by 2004. Furthermore, confirming Borger's observations, 16 cases of infection were observed in 2002, for which disease signs disappeared in 2003, and subsequently reappeared in 2004. It is possible that dark color syndrome may be a persistent affliction, which remains in the host tissues for long periods of time, years possibly,

but whose expression is influenced by environmental variables. Expression of this disease during summer months could indeed be triggered by water temperatures, heavy rainfall, or even the nutrient level alterations which usually correlate with periods of elevated rainfall. Alternatively, disease disappearance and reemergence could also be an indication that certain colonies are more susceptible than others to become infected, and hence during periods of favorable environmental conditions, the same coral colonies become infected recurrently. From a management perspective, this observation highlights the importance of monitoring diseases over extended periods of time. A static look at disease prevalence may not reveal enough about this highly dynamic phenomenon.

Little is known about the putative pathogen causing dark color syndrome. The finding of a reduced number of zooxanthellae in corals with dark color syndrome, as well as a reduced mitotic index of zooxanthellae, led Cervino *et al.* (2001) to suggest that perhaps the etiology of this syndrome may be initiated in the zooxanthellae, and the affliction may primarily affect zooxanthellae, and the coral tissue only secondarily. Alternatively, Weil *et al.* (2004b) reported differences in the bacterial communities living in association with healthy and diseased colonies of *Siderastrea siderea* and *Montastrea annularis*. Most notably, diseased colonies contained bacterial strains metabolically related to *Vibrio carchariae*, not found on healthy colonies. However, experimental bacterial inoculations of healthy corals have so far not resulted in disease. During the present survey, it was observed that the impact of dark color syndrome varied greatly depending on the target species. Among colonies of *S. siderea* and *Stephanocoenia michelinii*, approximately 60-70% of the infections did not cause tissue mortality, whereas close to 95% of infected *M. annularis* complex colonies sustained tissue necrosis. It is possible that different coral species may be responding differently to the same

pathological agent, but these observations also bring to light the possibility that similar dark coloration signs on various species may actually be produced by different pathogens. Gil-Agudelo *et al.* (2004) also concluded, based on monitoring of the disease in Colombian reefs, that signs of the syndrome could be present in some corals for several years, showing little to no change, while in other species, dark color syndrome showed higher rates of progression, resulting in larger lesions and greater tissue loss. Presently, it can be concluded from this study that the impact of dark color syndrome, in terms of tissue mortality, is generally more significant in *M. annularis* complex colonies than in other target species.

Though dark color syndrome was identified across all the stations surveyed, several significant outbreaks were witnessed during the duration of the survey. In 2002, very high incidence of the syndrome was observed at West Turtle Patch Reef Stations 3 and 4, where a considerable number of *Siderastrea siderea* colonies were affected (36 in Station 3 and 26 in half of Station 4). The subsequent year, the incidence of dark color syndrome at that reef was considerably lower, with most colonies of *S. siderea* no longer showing disease signs. Very high incidence of dark color syndrome was also observed on *Montastrea annularis* complex colonies at Grecian Rocks Station 1 in 2003 (14 diseased colonies), and at Jaap Patch Reef Stations 1 and 2 in 2004 (101 and 47 infected colonies respectively). These highly localized cases of high disease incidence could be interpreted as suggesting that the condition is possibly infectious. Surveys by Gil-Agudelo and Garzon-Ferreira (2001), and Borger (2003) all reported a clumped distribution of dark color syndrome in Colombian reefs and Dominican reefs respectively, which they suggested could be an indicator that the disease is contagious and possibly that a microorganism may be associated with it.

Although dark color syndrome was a highly prevalent disease throughout the entire area surveyed, its impact was not very significant in terms of tissue mortality. Between 2002 and 2004, only 2% of the colonies affected by dark color syndrome died. This represents only 3% of the dark color syndrome-affected colonies which suffered tissue necrosis, with the other 97% constituting cases of partial rather than whole mortality. Throughout the 14 stations surveyed from 2002 to 2003, the loss of coral tissue attributed to dark color syndrome corresponded to 1,948 cm<sup>2</sup> (approx. 0.2 m<sup>2</sup>), which is 2.8% of the projected cover of the 3 main target species in 2002 or 0.3% of the total scleractinian coral projected area in 2002. Studies of the syndrome on Colombian reefs also concluded that due to the small size of the lesions and their slow growth rate, it appeared that the effect of the disease in corals was very limited, and perhaps almost insignificant for the reef ecosystem as a whole (Gil-Agudelo *et al.* 2004).

Although dark color syndrome was not observed to cause extensive tissue necrosis during the period of time in which this survey was conducted, this disease may still have other significant effects. Given the extremely high prevalence of this condition and the fact that its target species are among the most important reef building species in the Florida Keys, any detrimental effects other than direct tissue necrosis could still pose a significant long-term threat to coral population dynamics. For example, the persistence of this condition is thought to affect the calcification process in the host coral, producing depressions on the coral surface (Garzon-Ferreira and Gil-Agudelo 1998; Weil *et al.* 2004b). It has also been suggested that this disease may decrease the regeneration capability of corals (Borger 2003). Ultimately, nothing is known about the influence of this condition on the reproductive capabilities of target corals. A potential effect of this disease on the fecundity of infected colonies could have very significant implications for the perpetuation of the reefs, given the nature of the target species. From a

management perspective, it is hence important not to dismiss this condition on the basis of its low mortality rates, but rather to focus research efforts on achieving some understanding of its pathology, microbiology, etiology and epizootiology.

### *Bleaching*

Unlike the mass mortalities of the scale observed in the Indo-Pacific (Wilkinson *et al.* 1999; Wilkinson 2002), bleaching in the Caribbean has caused variable, but generally low coral mortality (Weil 2004). Most studies of bleaching impacts have taken place following mass bleaching events, and not much is known about background levels of bleaching during non-mass bleaching years. Bleaching was seen to affect a variety of species, though the large majority of colonies which died as a result of bleaching were *Favia fragum* colonies, mostly affected at Western Sambo Reef between 2002 and 2003. Due to the characteristically small colony size of this species, it is difficult to determine to what extent the death of these colonies was directly caused by the bleaching condition, or by a stress state (which could also be expressed as bleaching) reducing the capacity of the small colonies to fight algae encroachment or sediment deposition. Among colonies of other species, bleaching did not pose a significant threat, since the colonies generally recovered without visible damage. Since most of the bleached colonies that died were of small size, tissue loss was low, amounting to only 409 cm<sup>2</sup> (0.04 m<sup>2</sup>), or 0.06% of 2002 projected live scleractinian coral cover.

### *White plague*

White plague disease has become widespread throughout the wider Caribbean during the past couple of decades (Dustan and Halas 1987; Richardson *et al.* 1998a; Richardson *et al.*

1998b; Richardson *et al.* 2001; Pantos *et al.* 2003). Most reports refer to epizootic events, and very few studies provide information on the prevalence and impact of this disease during non-epizootic conditions. In the Florida Keys, four large white plague epizootics have been reported since the disease was discovered. The first, in 1975, infected high numbers of *Mycetophyllia ferox* colonies (Dustan 1977). Ten years later, a second epizootic in the same area was reported, affecting mostly *Montastrea* colonies (Dustan and Halas 1987). A third, and more virulent, white plague event in the 1990s rapidly killed colonies of *Dichocoenia stokesii* (Richardson *et al.* 1998a). Within 2 years, the disease had affected 16 other coral species in the Florida Keys. More recently, a fourth epizootic, involving an even more virulent strain, produced significant coral tissue mortality in colonies of the *Montastrea* genus (Richardson *et al.* 2001). No significant outbreaks of white plague were observed during this study, although the disease was present in many of the stations surveyed. White plague was observed affecting a variety of species, as is consistent with other reports of this condition (Dustan 1977; Bruckner and Bruckner 1997; Richardson *et al.* 1998a; Richardson *et al.* 1998b; Richardson *et al.* 2001). Although this disease continues to be associated with epizootics and massive coral tissue loss (Richardson *et al.* 2004), this study revealed that, at non-epizootic levels, the impact of this disease throughout the Florida Keys was not significant in terms of total tissue mortality. Tissue losses directly attributable to white plague totaled 745 cm<sup>2</sup> (0.07 m<sup>2</sup>) throughout the 14 stations surveyed. This corresponds to 0.12% of the 2002 projected live scleractinian coral cover. Only 5 colonies died as a result of white plague, 3 of which were small colonies (3-10 cm diameter), and 2 were 10-50 cm in maximum diameter. By 2004, the disease persisted in only 4.5% of the colonies identified in the first 2 years of the survey and only 5 new cases were identified in 2004.

It should be noted that, during the 3 years in which this study was conducted, numerous colonies were noted to be experiencing tissue mortality from the base of the colony upwards in the manner that is typical of white plague infections. A sharp line demarcated live from dead tissue, often bisecting the edge polyps. However, because the dead tissue was not recently dead, but rather covered by filamentous algae and/or sediment, they were not included in the survey. Because freshly exposed skeleton was not visible at the time of the survey, they were considered to be examples of old disease infections, whose progression had been halted. However, tracking of several of these colonies indicated that they continued to show slow tissue loss over time (Figure 49).

The pattern of tissue mortality just described occurred frequently, under circumstances where an exact cause for the loss could not be determined. This condition occurred throughout the 14 stations surveyed, and therefore it is important to suggest possible explanations for these tissue losses. It is possible that these losses may constitute cases of tissue mortality due to algal-sediment overgrowth (Dustan 1977). A coral colony that is weakened, or subjected to a high rate of sedimentation may not be able to cleanse itself of sediment, and tissue damage and death may result (Dustan 1977). Smothering could also be occurring due to overgrowth by filamentous algae, which can trap fine sediments (Dustan 1977). Though encroaching sediment accumulation or growth of sediment-trapping algae was not observed on these colonies during this survey, no proof exists that such a phenomenon did not occur during other times. Another alternative is that these colonies were infected with white plague, yet disease progression rates were slow enough that growth of filamentous algae and sediment deposition caught up with the edge of receding tissue, so that no freshly exposed coral skeleton was ever apparent. It is also possible that the colonies were repeatedly affected by recurrent white plague infections, taking place during

periods of the year other than when the surveys were conducted. The fact that several of the colonies on which this phenomenon was observed were *Dichocoenia stokesii* colonies, which were first reported by Dustan (1977) to be among the most common white plague target species, adds validity to the hypothesis that white plague may have been involved in the tissue necrosis. If this were indeed the case, then relative impact of white plague on tissue mortality at the reef level would surely be more significant than what was quantified during this study.

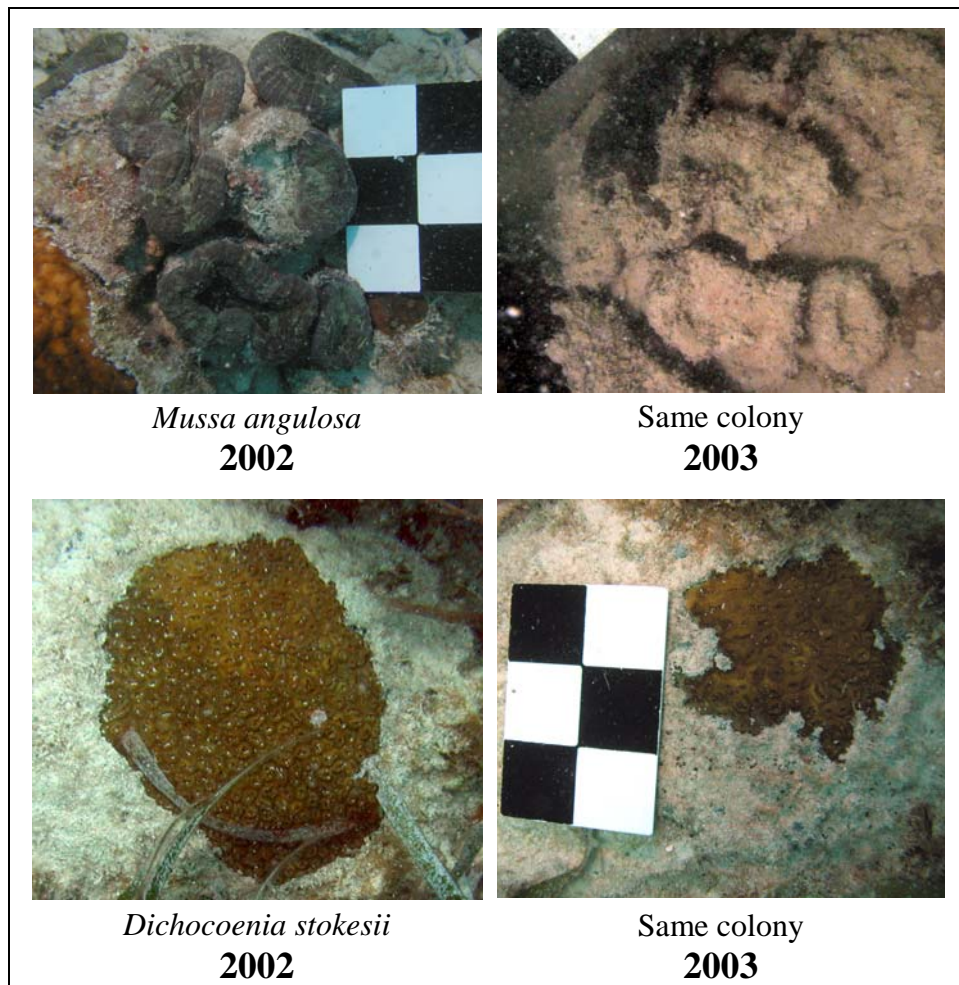


Figure 49. Examples of colonies observed and followed through time with tissue mortality around the base of the colony and bisected polyps. Such colonies were not included in the survey because no white bare skeleton was visible, and cause of death could not be confirmed.

### *Caribbean yellow band*

Along with dark color syndrome, Caribbean yellow band is also considered one of the relatively “new” diseases. Similarly, it has been observed with increasing frequency, over an increasingly wide distribution in recent years. The disease was first reported in 1994 from corals in the Florida Keys (Santavy and Peters 1997). It has since been observed throughout the wider Caribbean (Weil *et al.* 2002; Gil-Agudelo *et al.* 2004; Weil 2004) and major outbreaks have been reported in Panama (Santavy *et al.* 1999), Bonaire, Turks and Caicos, and the USVI (Cervino *et al.* 2001). Although in some of the Caribbean reefs where it is found, this is the most prevalent disease (Gil-Agudelo *et al.* 2004), during this survey, only one case of Caribbean yellow band was observed, affecting a colony of *Montastrea annularis* complex. This is the species most often reported as a target species, though the condition has been identified in 10 other species (Garzon-Ferreira *et al.* 2001; Weil *et al.* 2002; Gil-Agudelo *et al.* 2004).

Despite how common Caribbean yellow band has become in recent years, the etiology of this disease remains a mystery. As is the case with the majority of coral diseases, the causal agent of Caribbean yellow band is still unknown. Cervino *et al.* (2001) proposed, as with dark color syndrome, that Caribbean yellow band could actually be a zooxanthellae disease, which could kill the coral tissue by damage produced to its symbiont. They also reported a reduced number of zooxanthellae, with a reduced mitotic index in corals with Caribbean yellow band (Cervino *et al.* 2001). Preliminary histological studies revealed a degeneration of the tissue in affected areas, and the presence of a crystalline-like material in the gastric cavity (Santavy and Peters 1997). It is known that Caribbean yellow band is a slow progressing disease, with spreading rates of about 1 cm per month (Weil *et al.* 2004a), compared to other diseases such as white band or cyanobacterial mat disease, which can grow several centimeters per month.

Between 2002 and 2003, 26 cm<sup>2</sup> of coral tissue were lost on the affected colony, or approximately 2.2 cm<sup>2</sup> per month, which is similar to previous reported rates.

Many colonies can respond to diverse disease infections, stop their advance, and often recover and regrow lost tissue. However, previous studies of Caribbean yellow band have reported that, although the width of the band may be reduced during the winter, infected colonies were never completely rid of the infection during several years of observation (Weil 2004). Though only one case of infection by Caribbean yellow band was located during this survey, the signs persisted throughout the 3 years of observation, indicating that though this may indeed be a slow advancing syndrome, it has the potential of being very destructive in the long-term.

#### *Skeletal anomalies*

Though skeletal anomalies are not a common condition, they are potentially important indicators of changing environmental conditions. Skeletal anomalies have been recognized in many species of corals from around the world (Peters 2004). Three cases were observed in this survey, affecting the species *Diploria strigosa*, *Diploria clivosa* and *Acropora palmata*, which were already known to be among the typical target species for this syndrome (Peters 2004). Although the etiology of these conditions remains unknown, genetic mutations and UV exposure are hypothesized (Peters *et al.* 1986; Peters 2004). Reports of this type of lesions are apparently on the rise, which has led to the suggestion that globally or locally changing environmental conditions might be contributing to their formation, possibly by causing mutations in genes involved in controlling different aspects of the cell cycle. In the 3 cases identified here, lesions persisted throughout the duration of the survey, but no tissue necrosis was ever observed. Although these anomalies do not seem to pose any immediate or significant threat to the reef, it

is important to keep track of any increases in their occurrence. Because of their sensitivity to changes in their environment, corals are often considered “canaries in the coal mine.” If these types of anomalies are indeed proved to result from environmentally-induced genetic mutations, monitoring their occurrence could provide important indicators of global or local changes in environmental conditions.

### *White pox*

White pox is a recently emerged disease, first documented in the Florida Keys in 1996 (Holden 1996). It is now known to be among the most highly virulent diseases. White pox is blamed for decimating populations of *Acropora palmata* in the Florida Keys, as well as other reefs throughout the Caribbean. In the Florida Keys National Marine Sanctuary, white pox was responsible for the loss of an average 88% of the *A. palmata* populations between 1996 and 2002 (Sutherland and Ritchie 2004). Consequently, the species has now become a candidate for inclusion in the U.S. Endangered Species List (Diaz-Soltero 1999; Bruckner 2003). During this survey, 8 cases of white pox were identified. This prevalence is substantial considering the low abundance of *A. palmata* colonies and their distribution in only 3 of the 14 stations surveyed. Six of the cases detected occurred at Western Sambo Reef in 2004, where an outbreak of the disease was observed. Confirming what is already known about this disease, the tissue mortality due to white pox was significant in this survey: 866 cm<sup>2</sup> of tissue loss from only 2 affected colonies between 2002 and 2003. For comparison, among the 4 species which suffered the greatest total tissue mortality between 2002 and 2003, *A. palmata* suffered approximately as much tissue loss on only 2 colonies due to white pox, as a variety of diseases caused on 35 colonies of *Siderastrea siderea* or 18 colonies of *Montastrea annularis* complex. Clearly, this disease – known to be

caused by the enterobacterium *Serratia marcescens*, and possibly associated to human sewage pollution – continues to pose a significant threat to elkhorn coral populations in the Florida Keys. These populations have already been decimated in recent years, and the persistence of this disease could pose severe threats to the potential recovery of the species.

#### *Cyanobacterial mat disease*

Cyanobacterial mat disease was the first described coral disease (Antonius 1973). Although it has been reported in a wide variety of coral species around the world and extensive outbreaks have been reported in South Florida and the Caribbean (Peters 1993; Bruckner *et al.* 1997; Bruckner 1999), it is usually found at low prevalence (<1%) on scattered coral colonies on a reef (Garrett and Ducklow 1975; Edmunds 1991; Kuta and Richardson 1996; Bruckner *et al.* 1997). Results of this survey are consistent with this observation, with only 2 examples of cyanobacterial mat disease identified between 2002 and 2004. One case was found affecting a colony of *Montastrea cavernosa* and while the second affected a colony of *Colpolphyllia natans*. Both species are common targets for this disease (Santavy and Bruckner 2004). Because neither of the cases was detected in 2002, no tissue loss is attributed to this disease between 2002 and 2003. This disease is known to be able to progress over the coral tissue at a rapid rate (Antonius 1973; Richardson 1996), and may even cause the mortality of the entire colony (Rutzler *et al.* 1983; Bruckner *et al.* 1997). However, given its low prevalence under non-epizootic conditions, this disease is not considered to be a grave threat in the Florida Keys in terms of tissue mortality at the level of the ecosystem as a whole.

Although cyanobacterial mat disease may occur year round, it usually exhibits a distinct seasonal cycle. It is present primarily during the warmest months of the year, when water

temperatures rise above 28° C (Rutzler *et al.* 1983; Edmunds 1991; Kuta and Richardson 1996).

The optimal temperature for the proliferation of the dominant cyanobacterial mat disease pathogen, *Phormidium corallyticum*, is between 30°C and 37°C (Richardson and Kuta 2003). It should be noted that some of the stations, those in the Middle Keys, were surveyed as early as May in 2003, which may have precluded observation of some disease infections (of cyanobacterial mat or other diseases) which only appear later in the summer months.

Furthermore, average water temperatures for the 30 days preceding all 2003 and 2004 survey dates did not rise beyond 30°C and 31°C respectively (water temperatures were not available for 2002). Finally, it is interesting to note that reports of physical injury or stress appear to make the corals most susceptible to infection (Antonius 1985). Although this may not constitute more than a coincidence, during this survey, one of the two colonies affected by cyanobacterial mat disease had been infected with white plague the previous year.

#### *New potential diseases*

Reports of new and emerging coral diseases have proliferated in recent years. It is possible that climate- and human-mediated changes to the marine environment may be increasing the rate of contact between novel or known pathogens and susceptible hosts, or creating an environment more favorable for the proliferation of new and existing pathogens (Harvell *et al.* 1999). Whatever the causes may be, the emergence of new diseases in the marine ecosystem is no longer in question (Harvell *et al.* 1999; Harvell *et al.* 2004), and it is thus important for field coral biologists to keep an eye out for new and developing conditions. Two such potentially new disease conditions were observed during this survey. They were termed “condition 1” and “condition 2”, in order to avoid adding to the confusion already created by so

many new disease names reported in a very *ad hoc* fashion during the last decade (Richardson 1998). These potential diseases affect exclusively *Montastrea cavernosa* and *Montastrea annularis* complex colonies respectively. Prevalence of these conditions (9 and 6 colonies infected respectively between 2002 and 2004) warranted their inclusion in this study. Resulting observations of tissue mortality (including 1 case of complete colony mortality by “condition 1”) reaffirm the need for scientists to continue looking out for these conditions during field surveys, in order to better describe disease signs, their impact on host corals, rates of tissue loss and general incidence.

## COLONY SIZE

Studies have shown that in colonial organisms such as corals, often the fate of an individual can be predicted more accurately from its size than its age (Connell 1973; Hughes and Jackson 1980; Highsmith 1982). The processes of fragmentation, partial mortality, fusion and fission have been shown to combine to decouple simplistic size-age correlations (Hughes and Jackson 1980), such that conventional age-related population analyses are not suited to the demographic characteristics of coral colonies (Hughes 1984). Small or medium-sized individuals are frequently older than large ones, especially if they are crowded or injured (Hughes 1984). Size is often a stronger driver of population dynamics (Hughes 1984; Hughes and Connell 1987; Babcock 1991). Large individuals are typically more fecund, while small individuals usually have higher rates of mortality, regardless of age (Lefkovitch 1965; Jackson 1977; Highsmith *et al.* 1980). In this study, both healthy and diseased coral colonies were measured and correlations were found between disease incidence and coral colony sizes.

As is often the case with coral population structure (Bak and Meesters 1998), size distribution of all colonies counted during this survey was shown to be skewed towards smaller-sized colonies, with 55% of the colonies in the 3-10 cm diameter size class, 37% in the 10-50 cm diameter class, and only 8% of the population in the > 50 cm diameter size class. These observations match those made by Ginsburg *et al.* (2001), who also concluded that in Florida Keys patch reefs, coral size distribution was skewed towards smaller sizes. They hypothesized that the smaller number of large colonies could be the result of the cumulative effects of disease, bioerosion, and removal by storm waves.

This study shows that in the Florida Keys, coral diseases disproportionately affect the larger-sized colonies (10-50 cm diameter). Similar patterns of size-specific prevalence were noted by Kim and Harvell (2002) in aspergillosis-infected *Gorgonia* sea fans in the Florida Keys, and by Nugues (2002) among white plague diseased corals in St. Lucia. Kim and Harvell (2002) suggested three hypotheses to explain this pattern of colony size-dependent disease prevalence. One hypothesis proposes an increased disease prevalence with age, simply because older individuals tend to accumulate infections over time (Anderson and May 1998). Another explanation posits that the larger the colony, the larger the target will be for waterborne pathogen transmission. Finally, they also suggest that large colonies may be chemically more susceptible than smaller ones, because they contain more poorly defended areas chemically, notably the edges of the colony, than smaller colonies (Kim *et al.* 2000). Whatever the cause, the fact that diseases disproportionately affect larger colony sizes has profound demographic implications, because larger colonies have been shown to have greater fecundity than smaller ones (Hughes 1984; Szmant-Froelich 1985). In fact, the ability to reproduce can be size-dependent in some corals regardless of age (Connell 1973; Szmant 1986). This would suggest, in addition to the

direct contribution of coral diseases to tissue mortality, an indirect contribution to coral reef decline by repressing reproductive capabilities. It is very likely that, as indicated by Ginsburg *et al.* (2001), the skewed distribution of coral colonies in the Florida Keys toward smaller sizes is indeed partially caused by many years of coral disease pressures.

It has also been suggested that rates of partial and whole colony mortality are strongly dependent on colony size for most species (Hughes and Jackson 1980; Rylaarsdam 1983; Hughes and Jackson 1985; Babcock 1991; Meesters *et al.* 1997; Nugues 2002). Typically small colonies are either unharmed or die, while large colonies survive but accumulate injuries year after year (Hughes and Jackson 1985). In accordance with these reported patterns, during this study, no large corals suffered whole-colony mortality. In fact, approximately 80% of the colonies that died were in the 3-10 cm diameter size class. This suggests that small colonies are more likely to escape infections than large ones, however, once the infection occurs, small colonies are more likely to suffer complete mortality.

## ENVIRONMENTAL AND POPULATION FACTORS

Comparisons between disease prevalence and environmental parameters (depth and water temperature) and population factors (target species cover and frequency, and species diversity) did not produce significant correlations. In a similar disease survey carried out in Dominica, Borger (2003) also failed to demonstrate correlations between physical measurements (water temperature, wave height, turbulence, depth, visibility and currents) and disease densities. Borger did find a significant negative relationship between dark color syndrome prevalence and coral diversity values (Shannon-Weiner Index,  $H'$ ), but such a correlation was not seen in this study. It

is possible that the absence of correlations in this study between disease and population and environmental parameters is due to the limited number of stations sampled, the small range in depths or the fact that all stations were not consistently sampled the same month of the year.

Although few environmental parameters were examined in this study, analyses of nutrient concentrations and other water quality parameters, and how they relate to long-term disease incidence trends are of critical interest. Anthropogenic impacts are considered potential causes for worldwide rises in disease occurrence, but quantitative data to support such assertions is still very limited. Warming temperatures have been linked with increased disease outbreaks in corals and other marine organisms (Hayes *et al.* 2001; Harvell *et al.* 2002; Kuta and Richardson 2002; Rosenberg and Ben-Haim 2002). Nutrient enrichment has been shown to significantly increase the severity of two coral diseases, aspergillosis of sea fans and Caribbean yellow band (Bruno *et al.* 2003) (Kim and Harvell 2002). Cyanobacterial mat disease and white pox may be associated with fecal contamination of possible human origin (Frias-Lopez *et al.* 2002; Patterson *et al.* 2002). The links however, are not always clear. White band disease outbreaks in Belize and the USVI, for example, occurred on reefs distant from human impacts (Gladfelter 1982; Weil 2004). The white pox outbreak that affected elkhorn corals in southwestern Puerto Rico (Weil *et al.* 2002; Weil and Ruiz 2003) also occurred in reefs distant from direct pollution. Though cyanobacterial mat disease appears to increase in abundance in polluted waters near industrialized areas (Antonius 1988), the disease has also been found on relatively pristine reefs with minimal exposure to human activity (Edmunds 1991). The question of the relationship between disease incidence and environmental changes remains one of the most important aspects of any disease studies and should remain at the forefront of future research efforts.

## CONCLUSIONS

- Disease incidence in the coral reefs of the Florida Keys fluctuates considerably from year to year. Prevalence of disease varied from 4.0% to 8.2% between 2002 and 2004.
- Disease lethality was low, with only 12% of the colonies identified with disease in 2002 and 2003 sustaining complete colony mortality by the end of the survey (1% of the coral population), and only 41% of diseased colonies experiencing partial mortality (3% of the coral population).
- In 40-50% of diseased colonies, disease signs did not last for more than one year.
- Percent cover of live coral in all surveyed stations did not change significantly between 2002 and 2003. Trends in coral cover during the past 3-4 years are indicative of an encouraging scenario of slight improvement in general reef health during recent years, and vastly contrast with the high rates of coral loss measured on these same reefs during the late 1990s.
- Measurements of tissue loss due to disease are highly consistent with this non-significant change in total coral cover: only 0.4 m<sup>2</sup> of coral tissue were lost due to disease between 2002 and 2003. This constitutes only 0.7% of the 2002 projected live coral cover for the 14 stations surveyed, and is well within the margin of error associated with live coral cover surveys.
- *Siderastrea siderea* and *Montastrea annularis* complex represented more than 60% of the diseased colonies during each year of the survey. These two species, in addition to *Montastrea cavernosa*, and *Acropora palmata* suffered the highest disease-associated

tissue losses. All four are among the most important reef building species in the Florida Keys.

- Dark color syndrome was the most prevalent disease (65-83% of diseased colonies), affecting mostly *Siderastrea siderea* and *Montastrea annularis* complex colonies. In over half of the cases the disease did not persist for more than 1 year, but several cases of reoccurrence were observed. Tissue necrosis was not extensive for this disease, but varied depending on the target species: tissue loss was seen in 95% of *M. annularis* colonies, but only in 40% of *S. siderea* colonies. It is not clear whether dark color syndrome signs seen on different species are a result of the same pathogen. Overall total tissue mortality amounted to only 0.2 m<sup>2</sup>, which is 0.3% of the total scleractinian coral area in 2002.
- White pox still poses a serious threat to the recovery of already-low *Acropora palmata* populations.
- Diseases disproportionately affected larger sized coral colonies. This could have significant demographic implications, since larger sized colonies are more fecund than smaller ones.
- No correlations were observed between disease prevalence and several environmental and population factors. Further analysis of this potential relationship is of critical importance, in order to ascertain whether anthropogenic impacts are contributing to a rise in coral disease incidence.
- Currently, under non-epizootic levels, disease does not appear to pose a significant threat to coral survival in the Florida Keys. Nevertheless, the species-specificity of disease pressures, and sustained long-term partial tissue losses may have unforeseen

consequences on reproduction capabilities, species richness, three-dimensional reef structure, or any number of as-yet unidentified characteristics.

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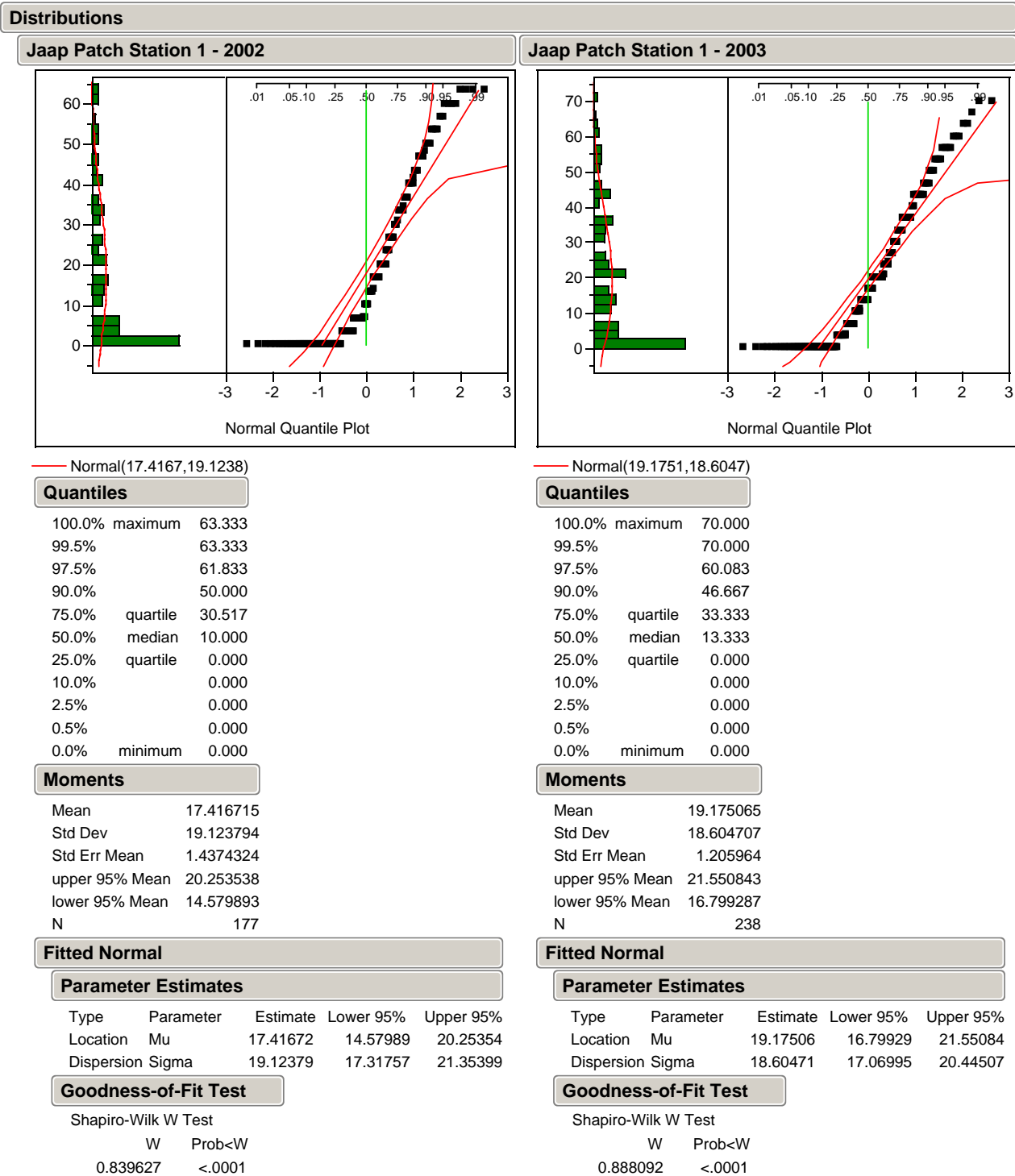
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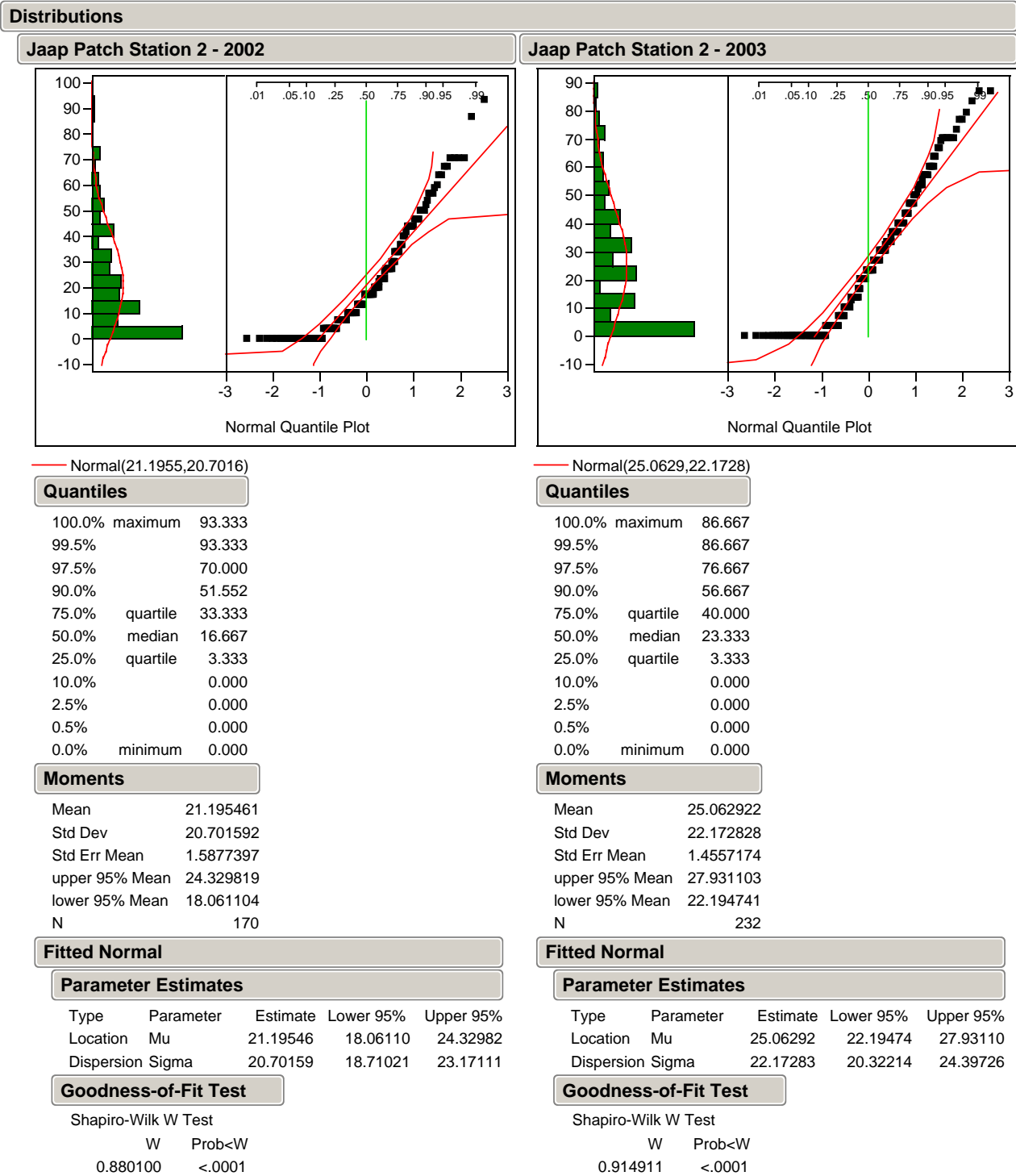
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## APPENDIX I

Distribution of scleractinian coral percent cover data and results of Shapiro-Wilk goodness of fit tests for Jaap Patch Reef Station 1 for 2002 and 2003.



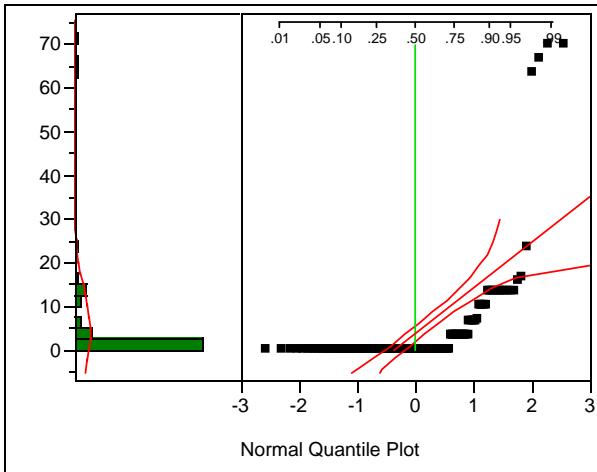
Distribution of scleractinian coral percent cover data and results of Shapiro-Wilk goodness of fit tests for Jaap Patch Reef Station 2 for 2002 and 2003.



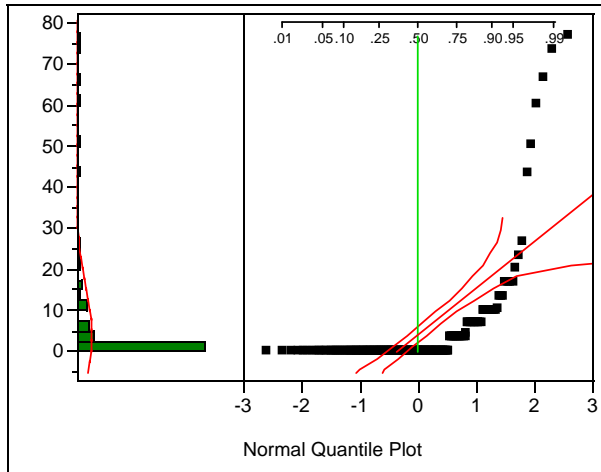
Distribution of scleractinian coral percent cover data and results of Shapiro-Wilk goodness of fit tests for Western Sambo Offshore Shallow Reef Station 1 for 2002 and 2003.

**Distributions**

**Western Sambo Station 1 - 2002**



**Western Sambo Station 1 - 2003**



— Normal(3.56774, 10.5487)

**Quantiles**

100.0%	maximum	70.000
99.5%		70.000
97.5%		39.333
90.0%		13.333
75.0%	quartile	3.333
50.0%	median	0.000
25.0%	quartile	0.000
10.0%		0.000
2.5%		0.000
0.5%		0.000
0.0%	minimum	0.000

**Moments**

Mean	3.5677407
Std Dev	10.548677
Std Err Mean	0.7797806
upper 95% Mean	5.1063135
lower 95% Mean	2.029168
N	183

**Fitted Normal**

**Parameter Estimates**

Type	Parameter	Estimate	Lower 95%	Upper 95%
Location	Mu	3.56774	2.029168	5.10631
Dispersion	Sigma	10.54868	9.567346	11.75609

**Goodness-of-Fit Test**

Shapiro-Wilk W Test		
W	Prob<W	
0.361116	0.0000	

— Normal(4.01257, 11.3664)

**Quantiles**

100.0%	maximum	76.667
99.5%		76.667
97.5%		50.000
90.0%		10.000
75.0%	quartile	3.333
50.0%	median	0.000
25.0%	quartile	0.000
10.0%		0.000
2.5%		0.000
0.5%		0.000
0.0%	minimum	0.000

**Moments**

Mean	4.0125738
Std Dev	11.366421
Std Err Mean	0.8057442
upper 95% Mean	5.6015154
lower 95% Mean	2.4236321
N	199

**Fitted Normal**

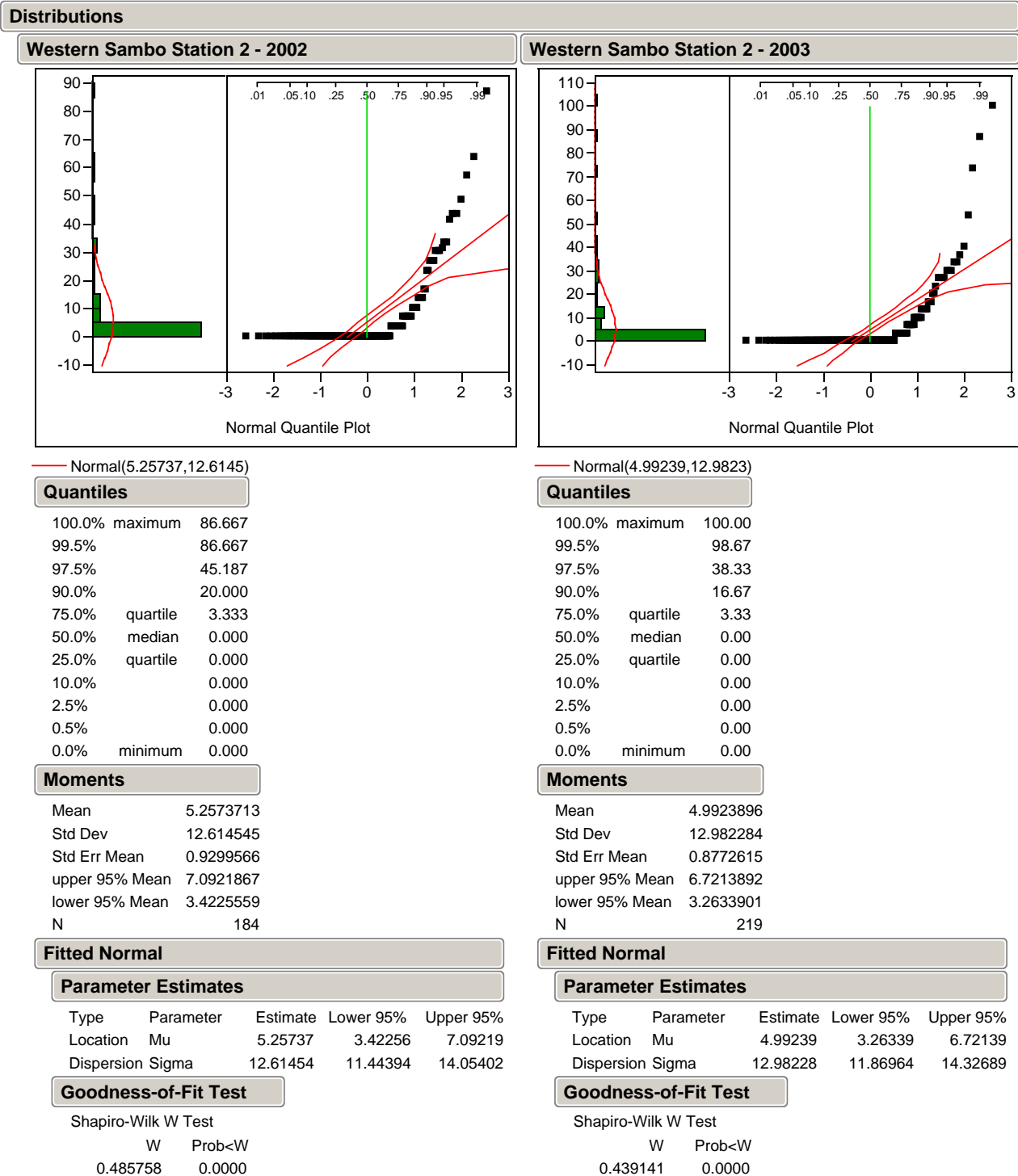
**Parameter Estimates**

Type	Parameter	Estimate	Lower 95%	Upper 95%
Location	Mu	4.01257	2.42363	5.60152
Dispersion	Sigma	11.36642	10.34865	12.60797

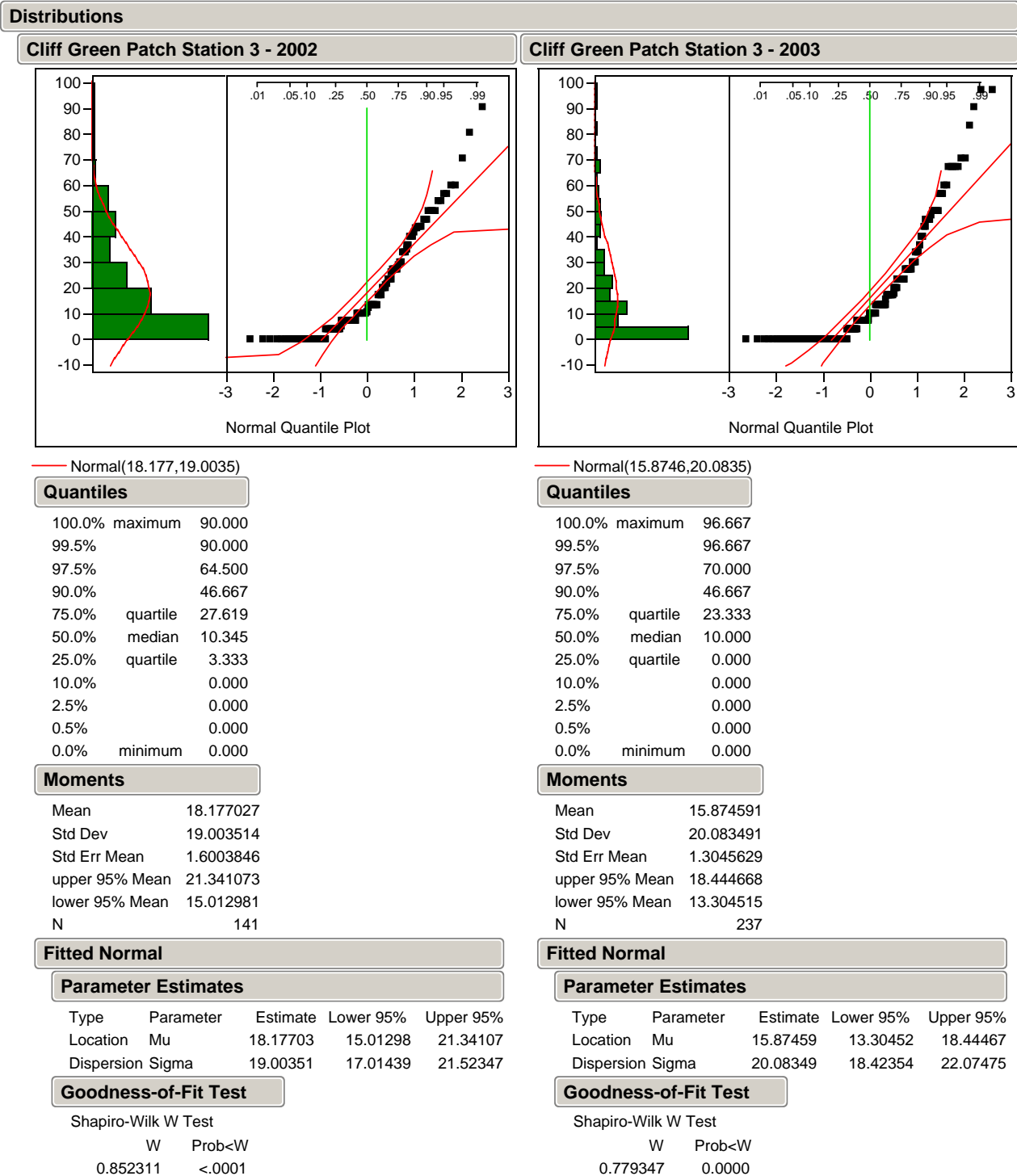
**Goodness-of-Fit Test**

Shapiro-Wilk W Test		
W	Prob<W	
0.389420	0.0000	

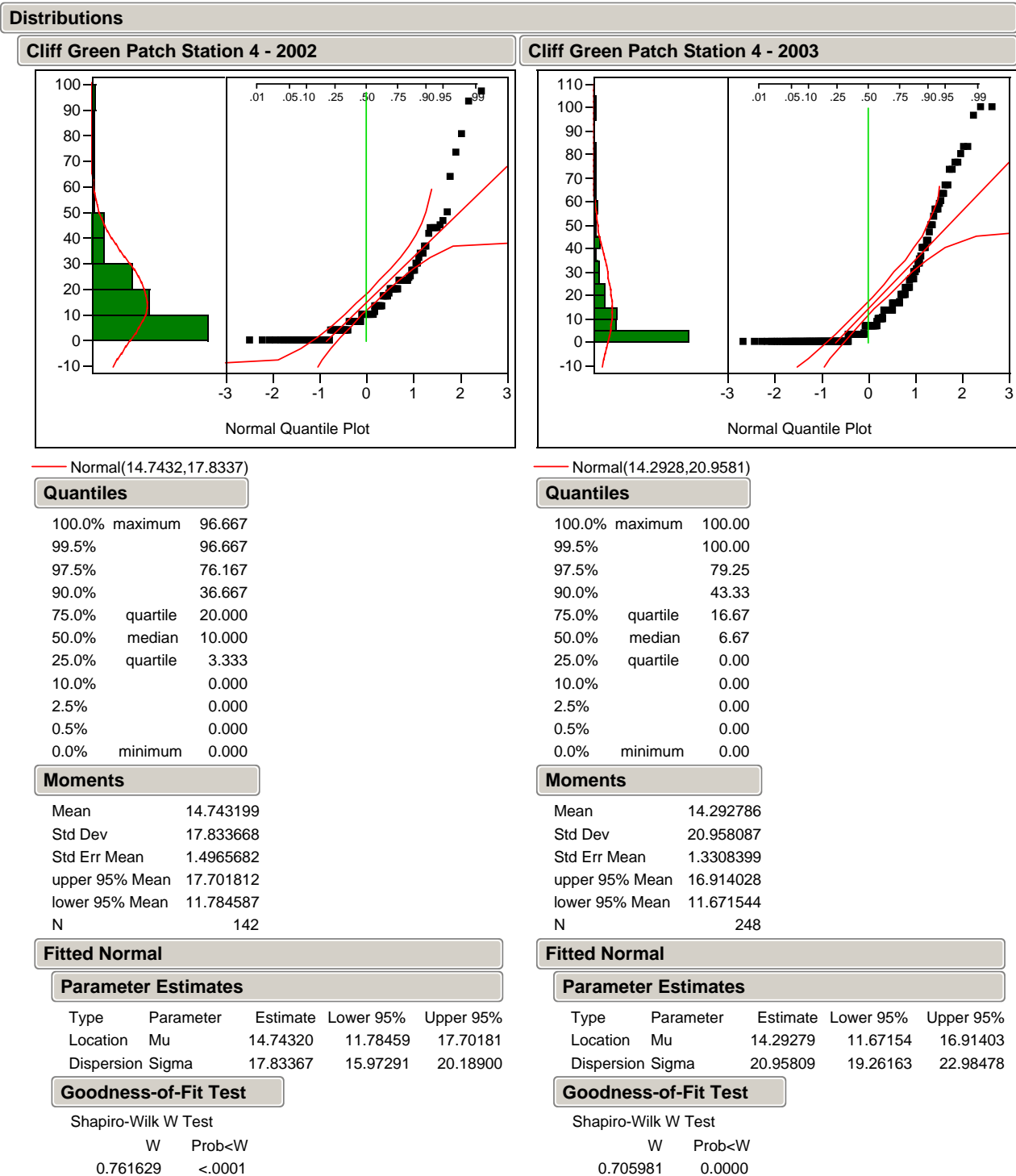
Distribution of scleractinian coral percent cover data and results of Shapiro-Wilk goodness of fit tests for Westen Sambo Offshore Shallow Reef Station 2 for 2002 and 2003.



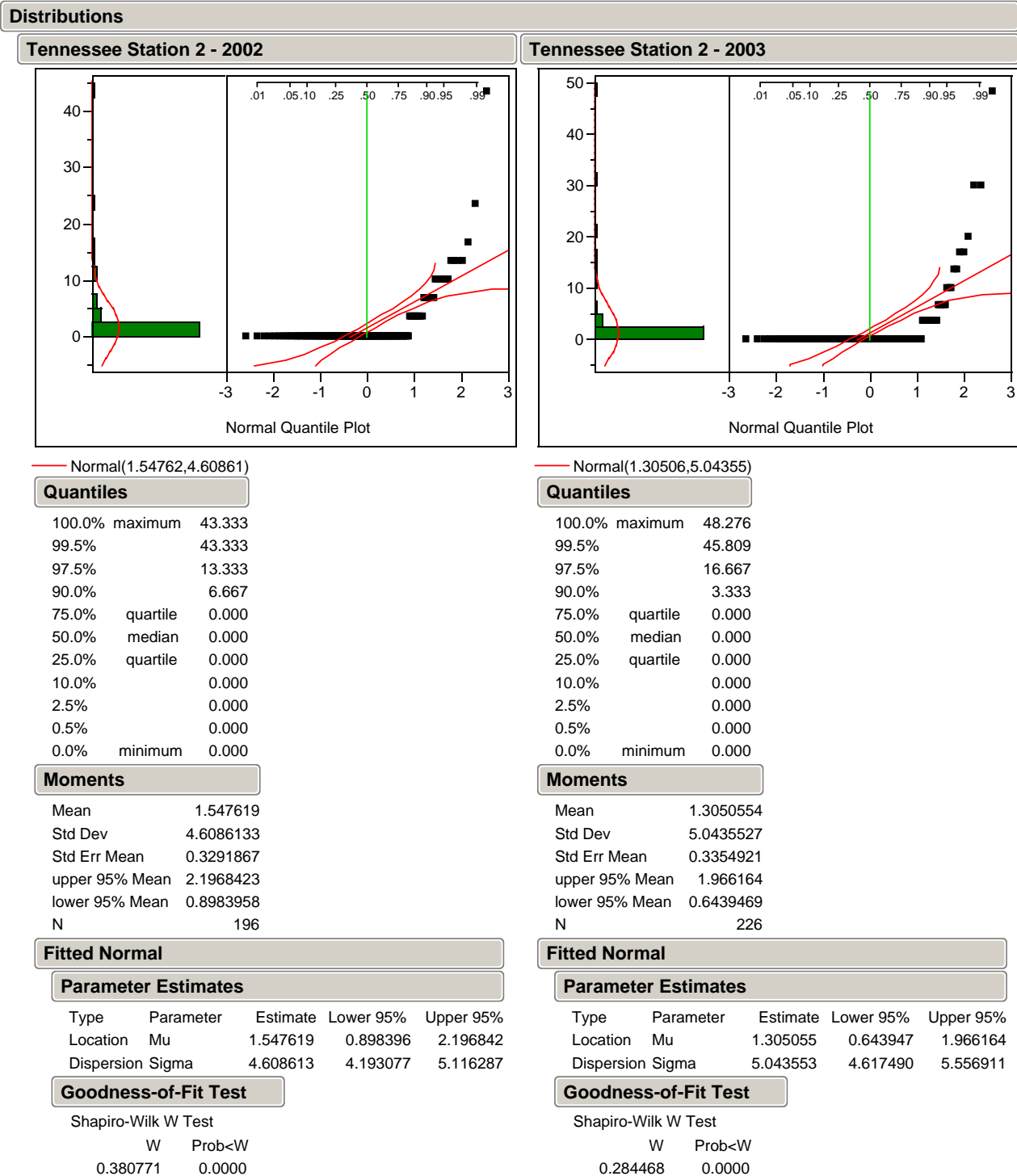
Distribution of scleractinian coral percent cover data and results of Shapiro-Wilk goodness of fit tests for Cliff Green Patch Reef Station 3 for 2002 and 2003.



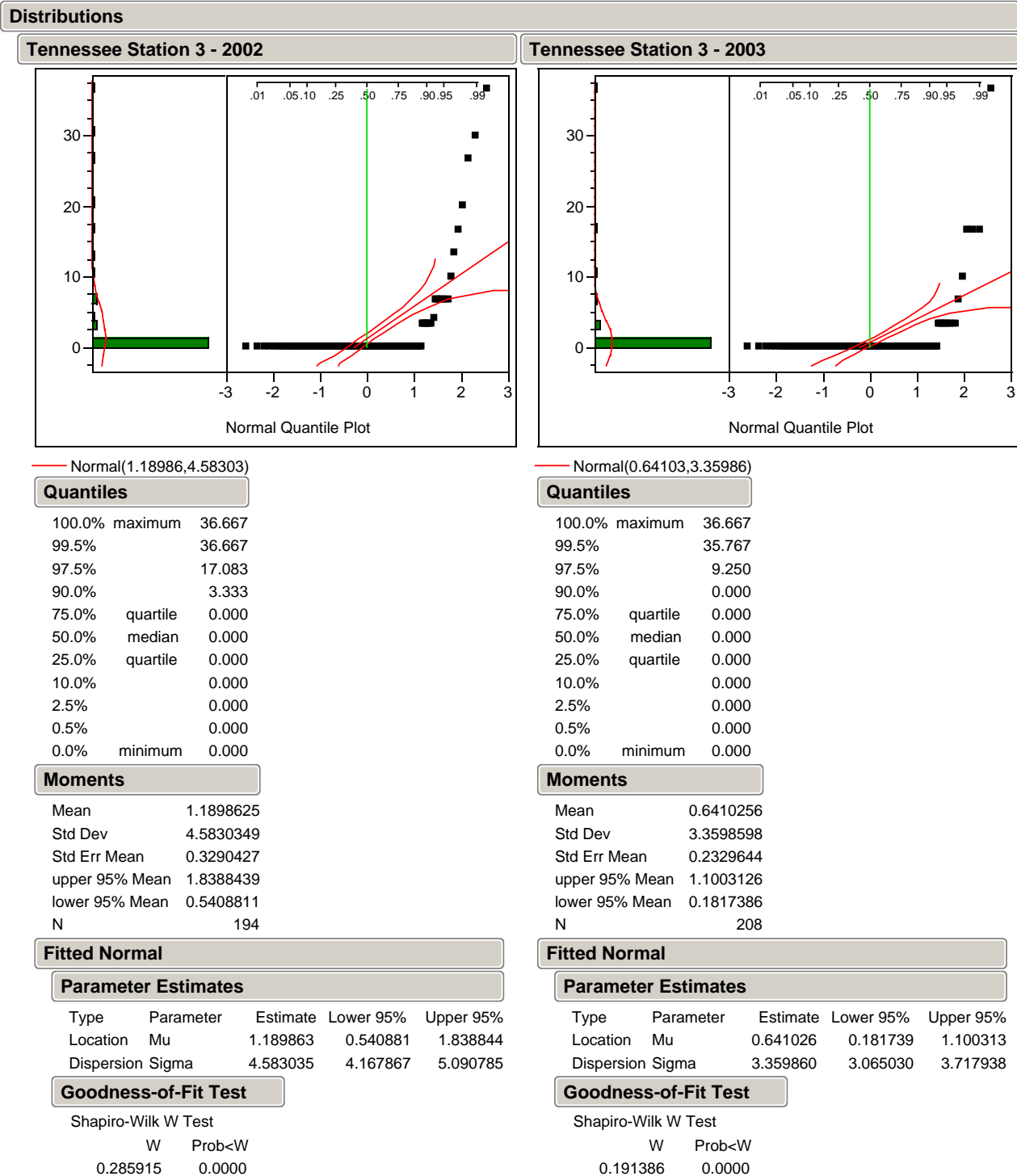
Distribution of scleractinian coral percent cover data and results of Shapiro-Wilk goodness of fit tests for Cliff Green Patch Reef Station 4 for 2002 and 2003.



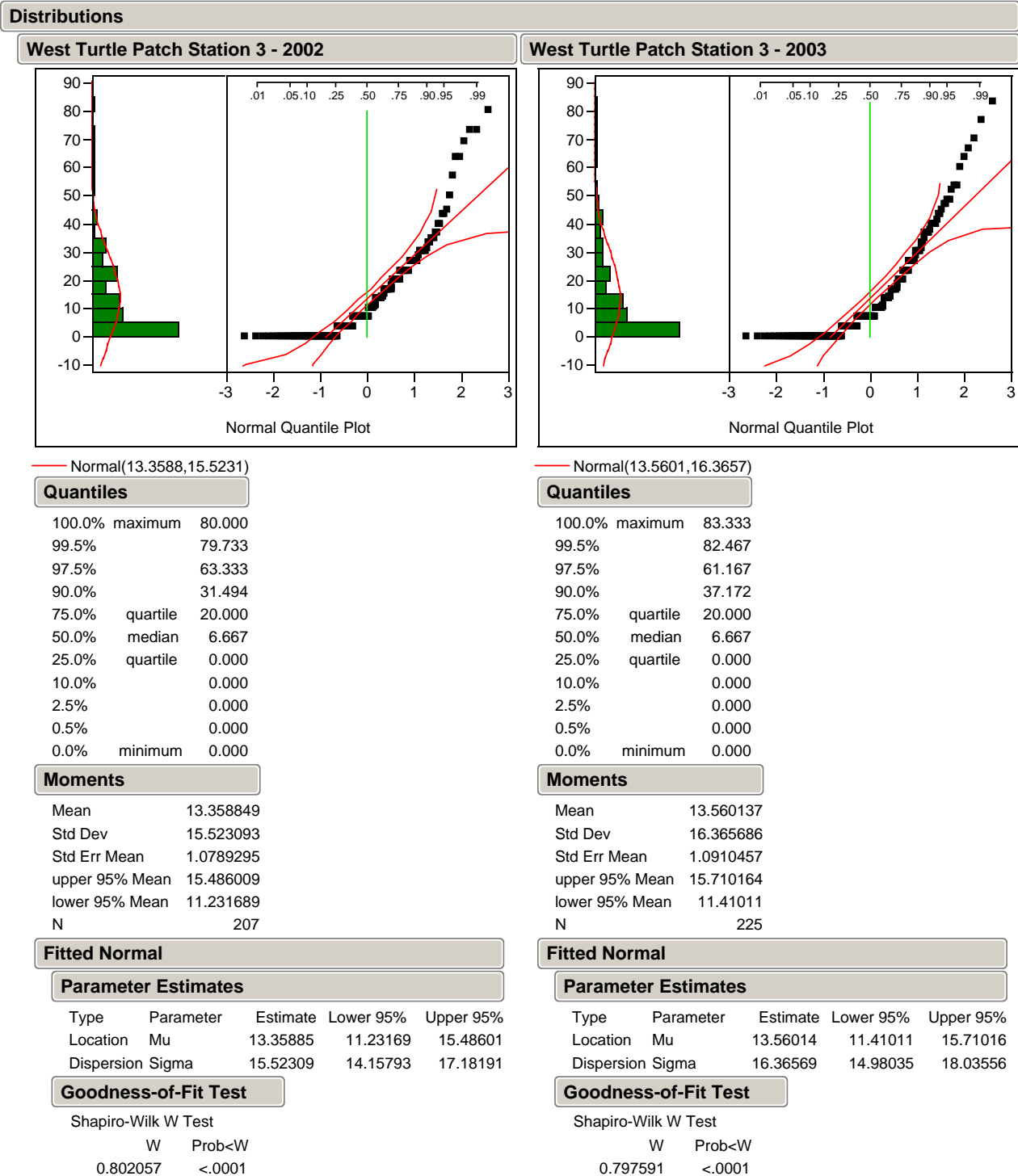
Distribution of scleractinian coral percent cover data and results of Shapiro-Wilk goodness of fit tests for Tennessee Offshore Shallow Reef Station 2 for 2002 and 2003.



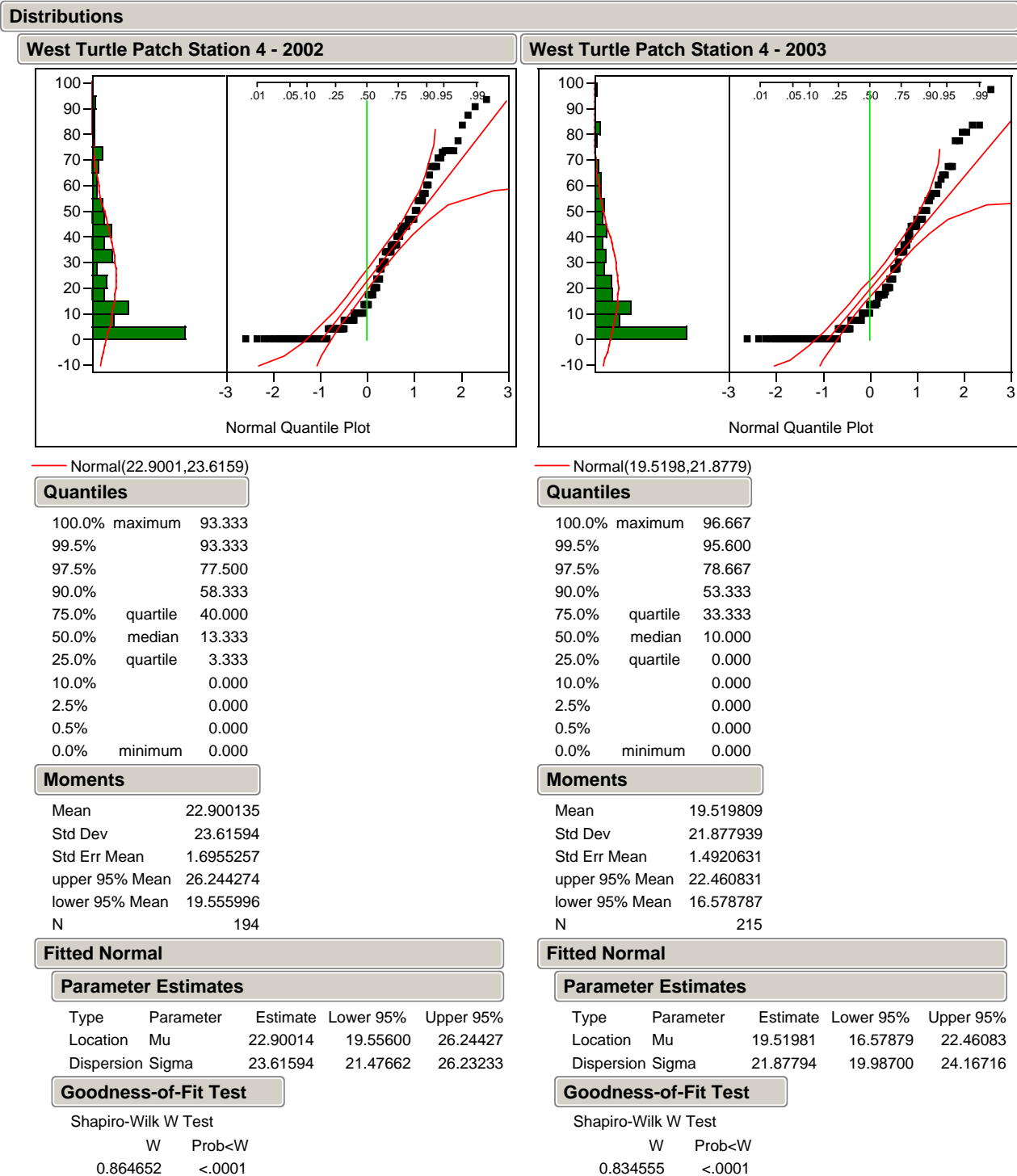
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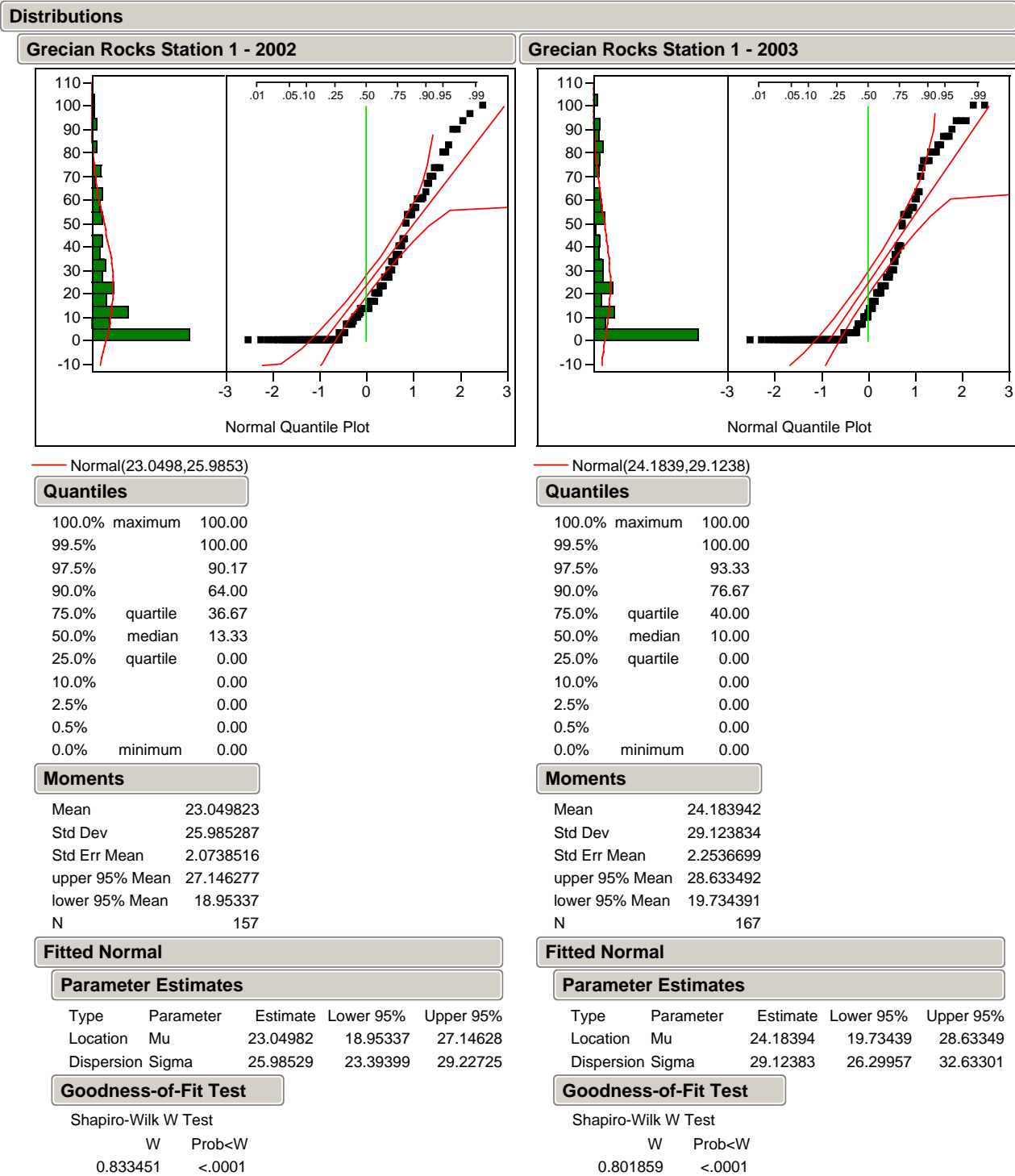
Distribution of scleractinian coral percent cover data and results of Shapiro-Wilk goodness of fit tests for West Turtle Shoal Patch Reef Station 3 for 2002 and 2003.



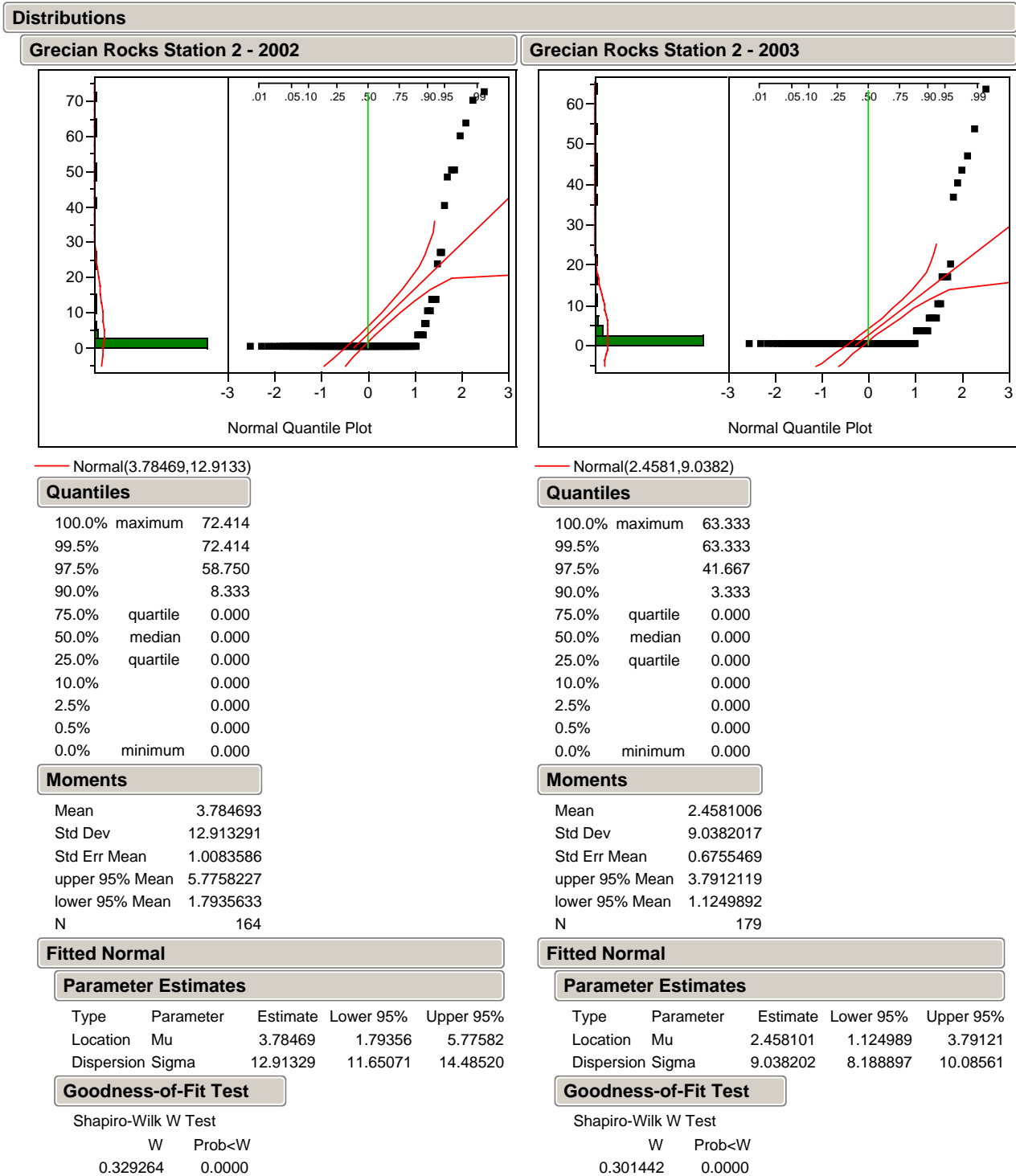
Distribution of scleractinian coral percent cover data and results of Shapiro-Wilk goodness of fit tests for West Turtle Shoal Patch Reef Station 4 for 2002 and 2003.



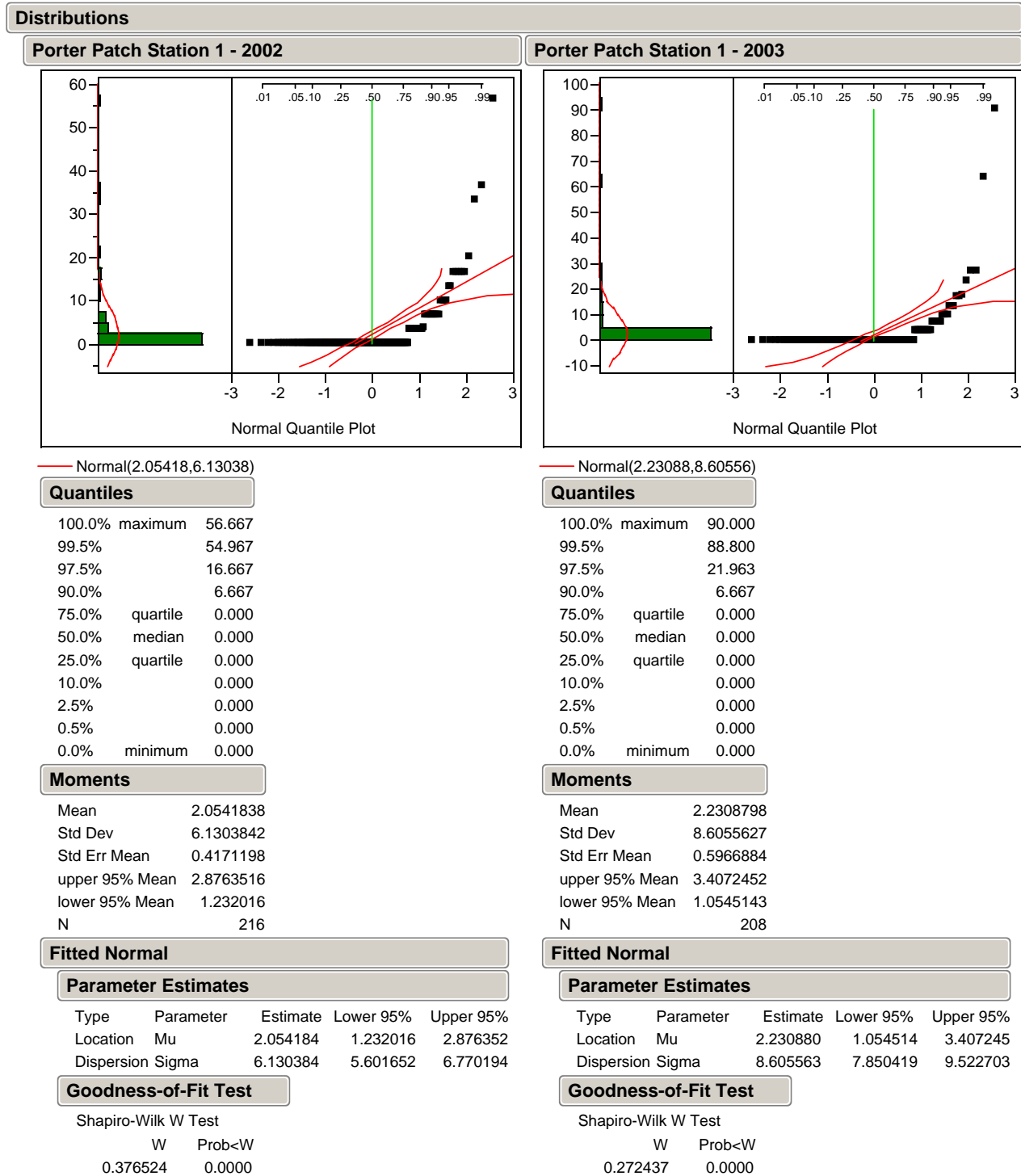
Distribution of scleractinian coral percent cover data and results of Shapiro-Wilk goodness of fit tests for Grecian Rocks Station 1 for 2002 and 2003.



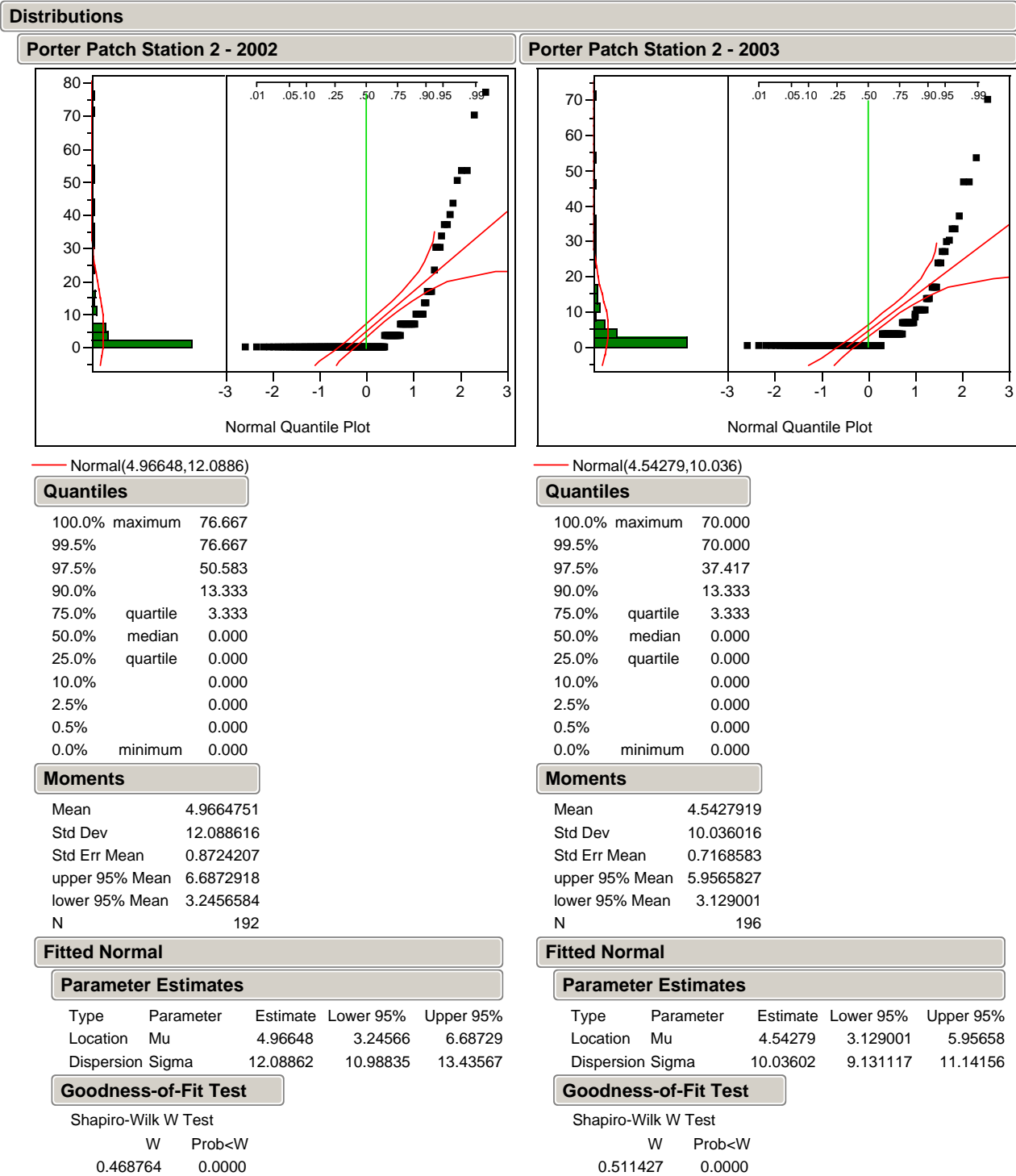
Distribution of scleractinian coral percent cover data and results of Shapiro-Wilk goodness of fit tests for Grecian Rocks Offshore Shallow Reef Station 2 for 2002 and 2003.



Distribution of scleractinian coral percent cover data and results of Shapiro-Wilk goodness of fit tests for Porter Patch Reef Station 1 for 2002 and 2003.

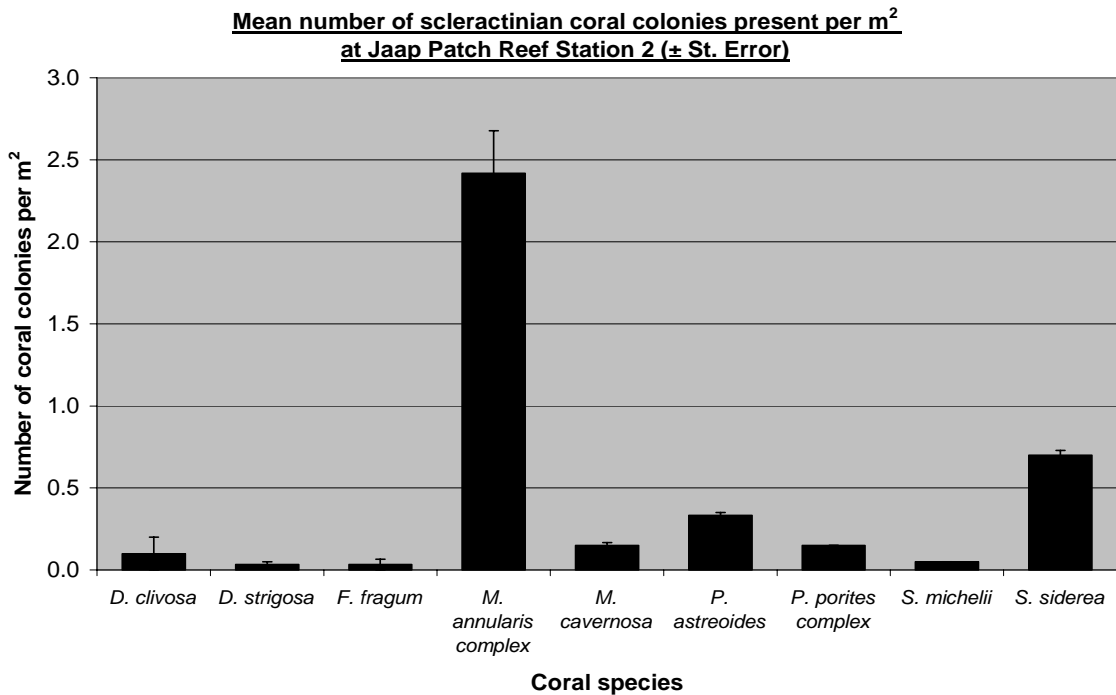
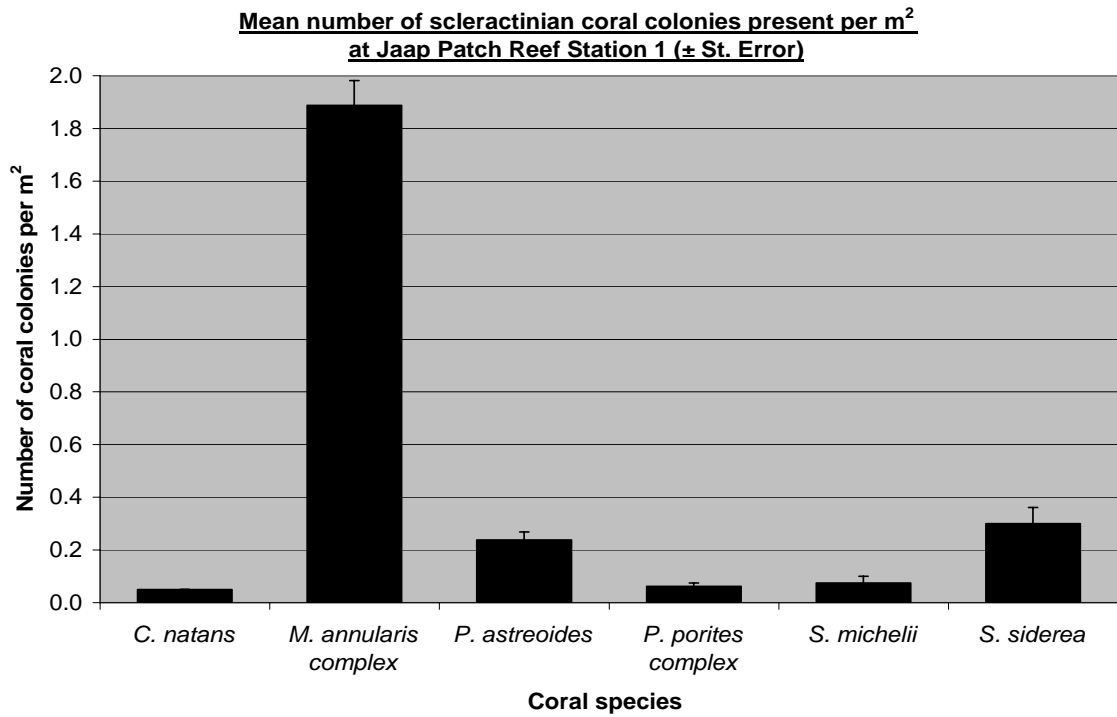


Distribution of scleractinian coral percent cover data and results of Shapiro-Wilk goodness of fit tests for Porter Patch Reef Station 2 for 2002 and 2003.



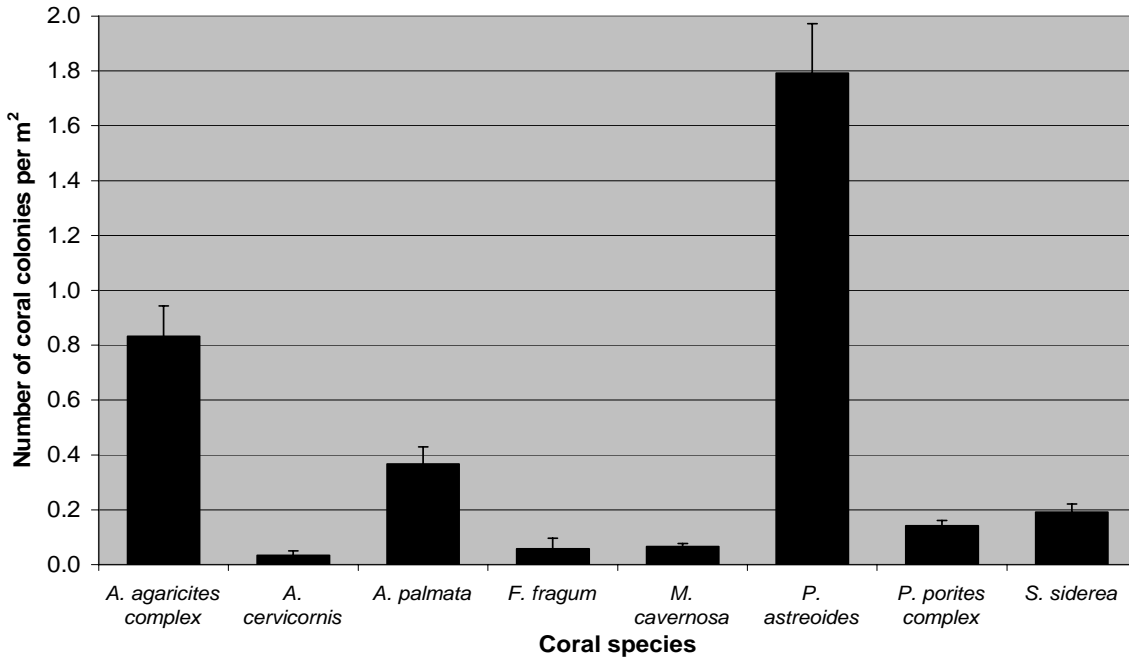
## APPENDIX II

Mean number of scleractinian coral colonies present per m<sup>2</sup> at Jaap Patch Reef Stations 1 (above) and 2 (below).

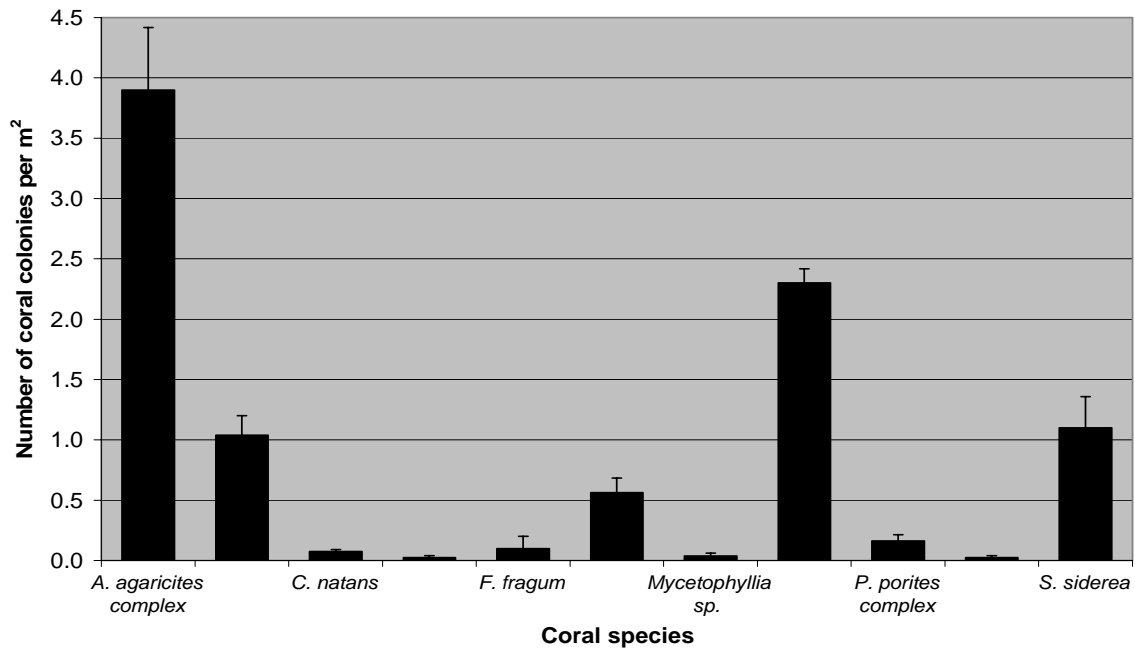


Mean number of scleractinian coral colonies present per m<sup>2</sup> at Western Sambo Offshore Shallow Reef Stations 1 (above) and 2 (below).

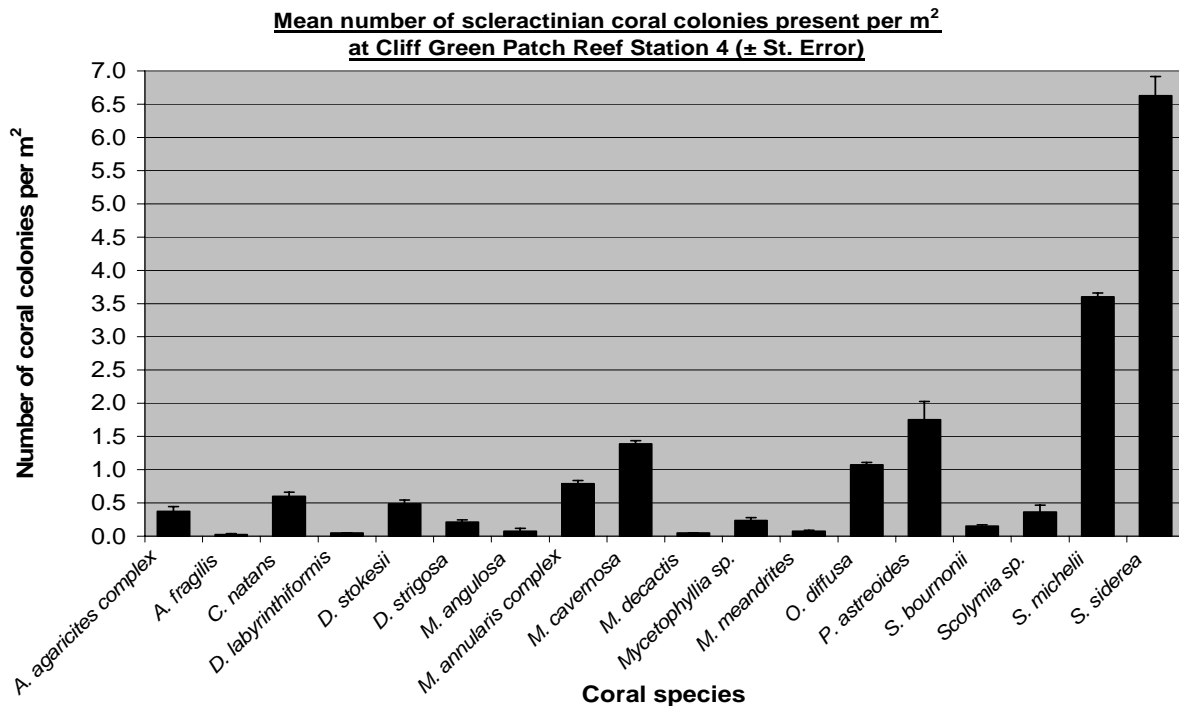
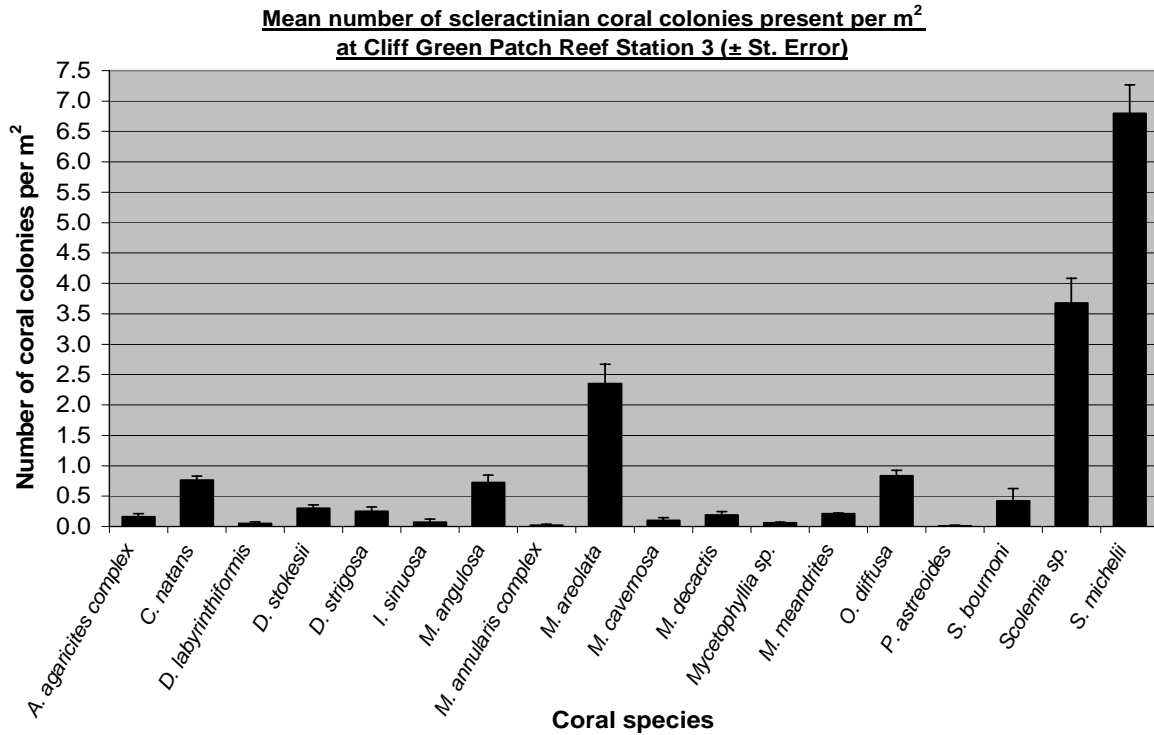
**Mean number of scleractinian coral colonies present per m<sup>2</sup> at Western Sambo Reef Station 1 (± St. Error)**



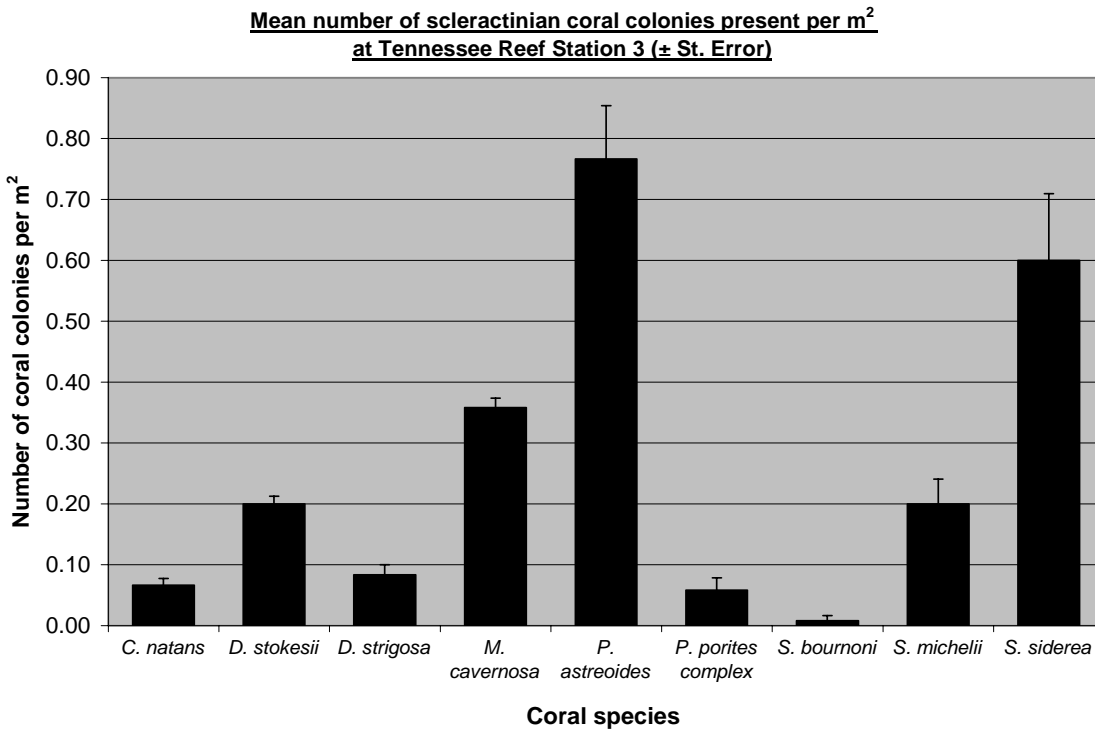
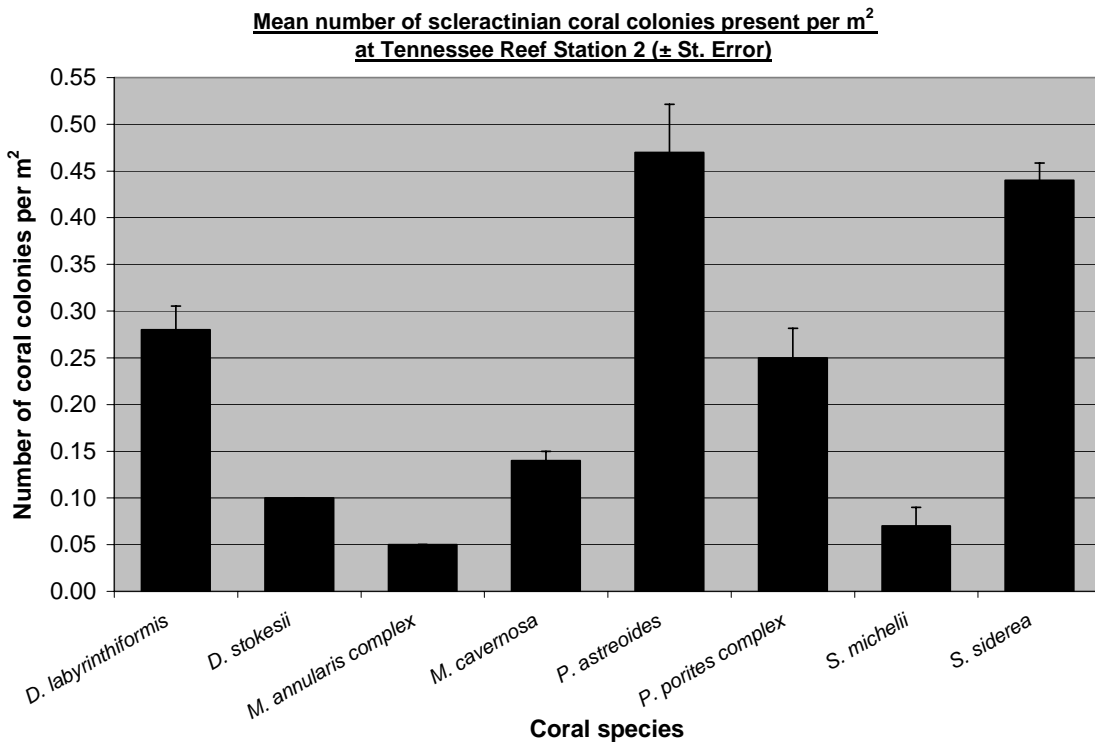
**Mean number of scleractinian coral colonies per m<sup>2</sup> at Western Sambo Reef Station 2 (± St. Error)**



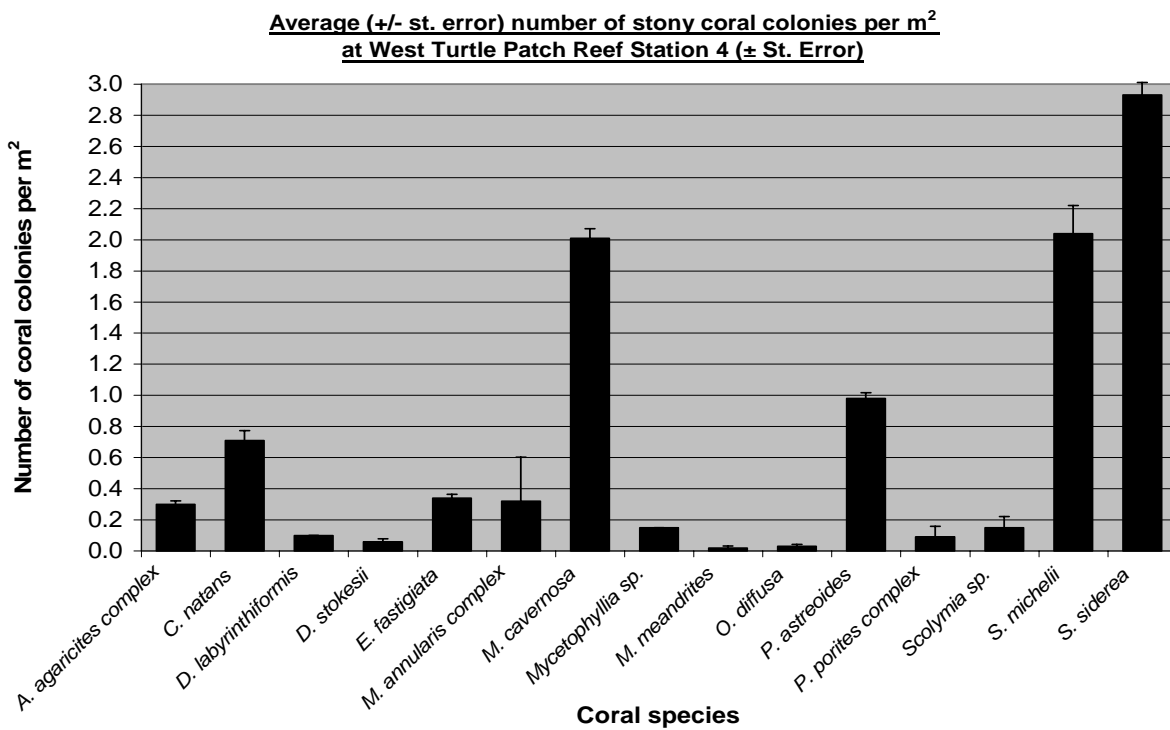
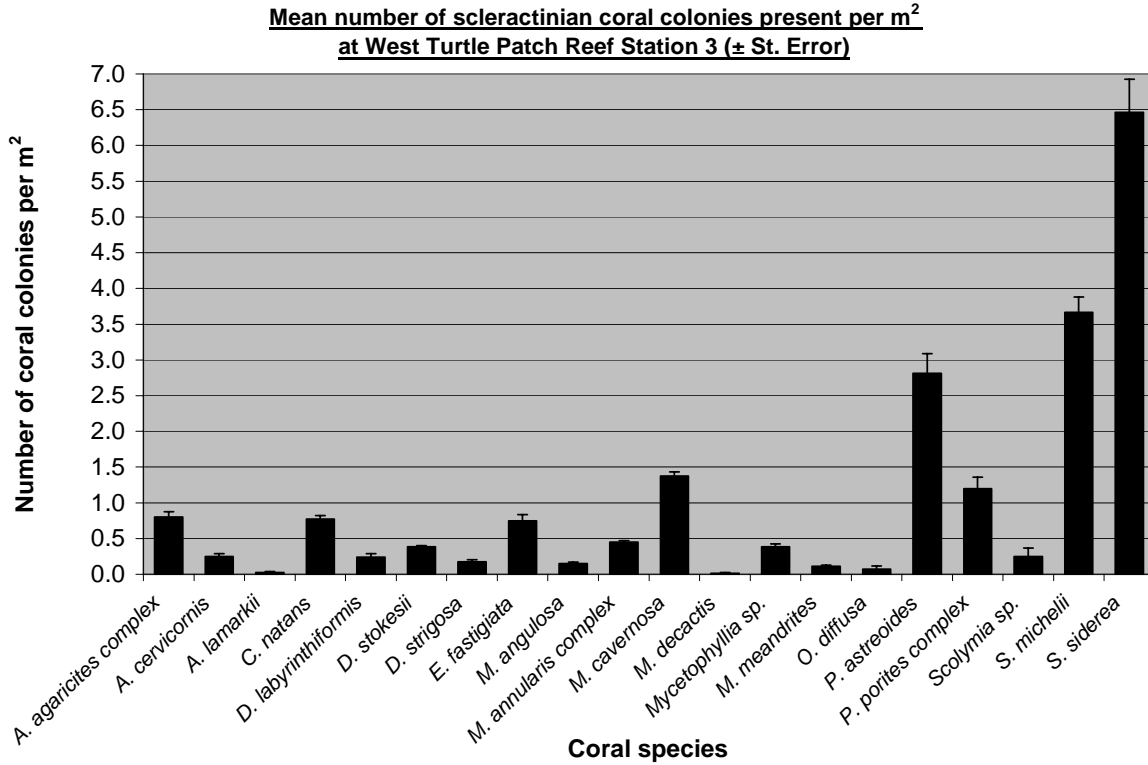
Mean number of scleractinian coral colonies present per m<sup>2</sup> at Cliff Green Patch Reef Stations 3 (above) and 4 (below).



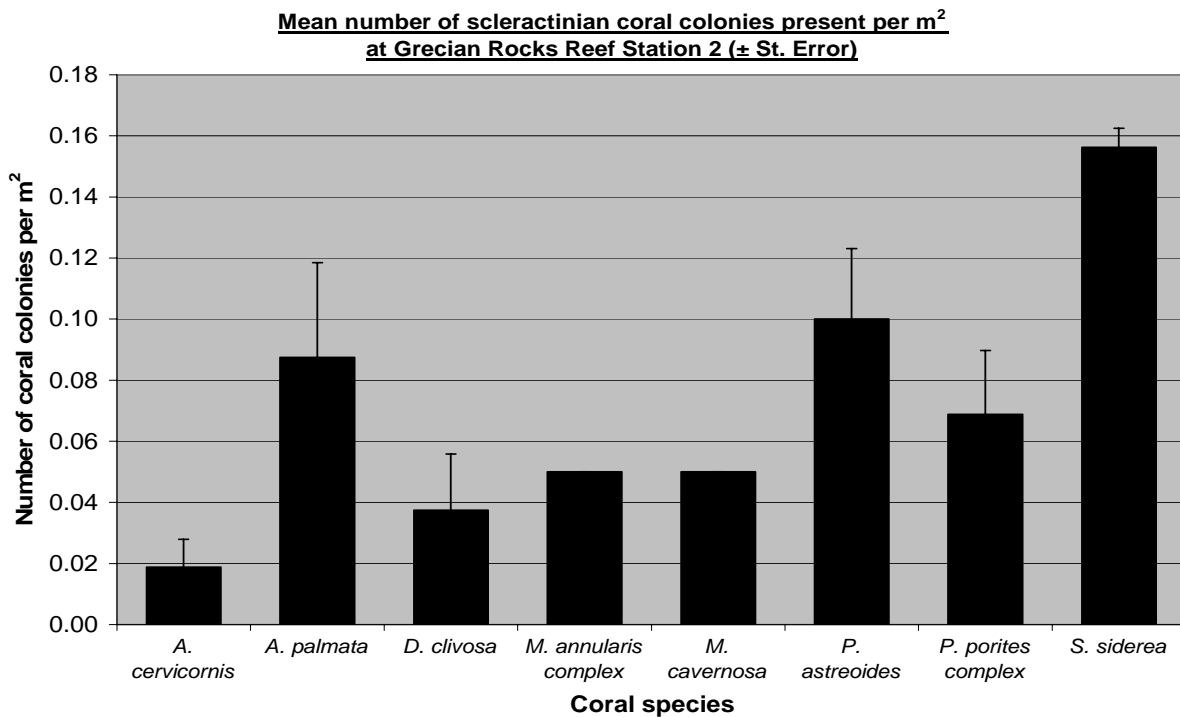
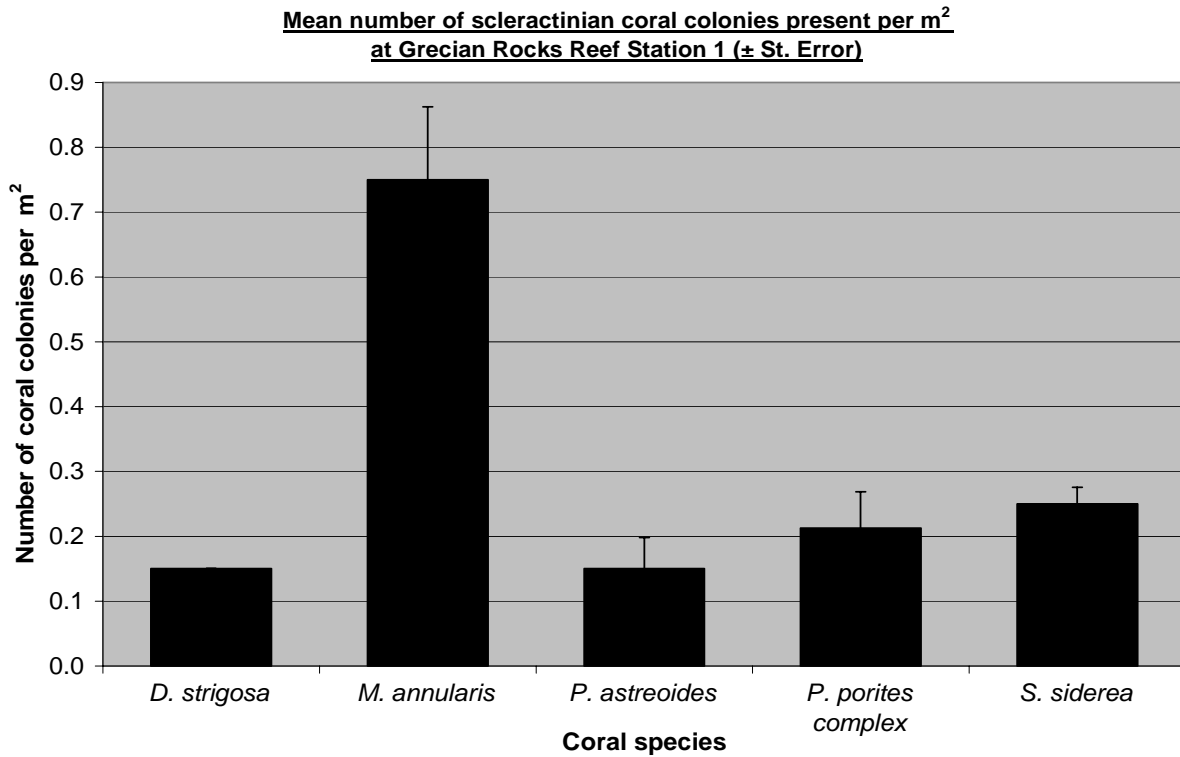
Mean number of scleractinian coral colonies present per m<sup>2</sup> at Tennessee Offshore Shallow Reef Stations 2 (above) and 3 (below).



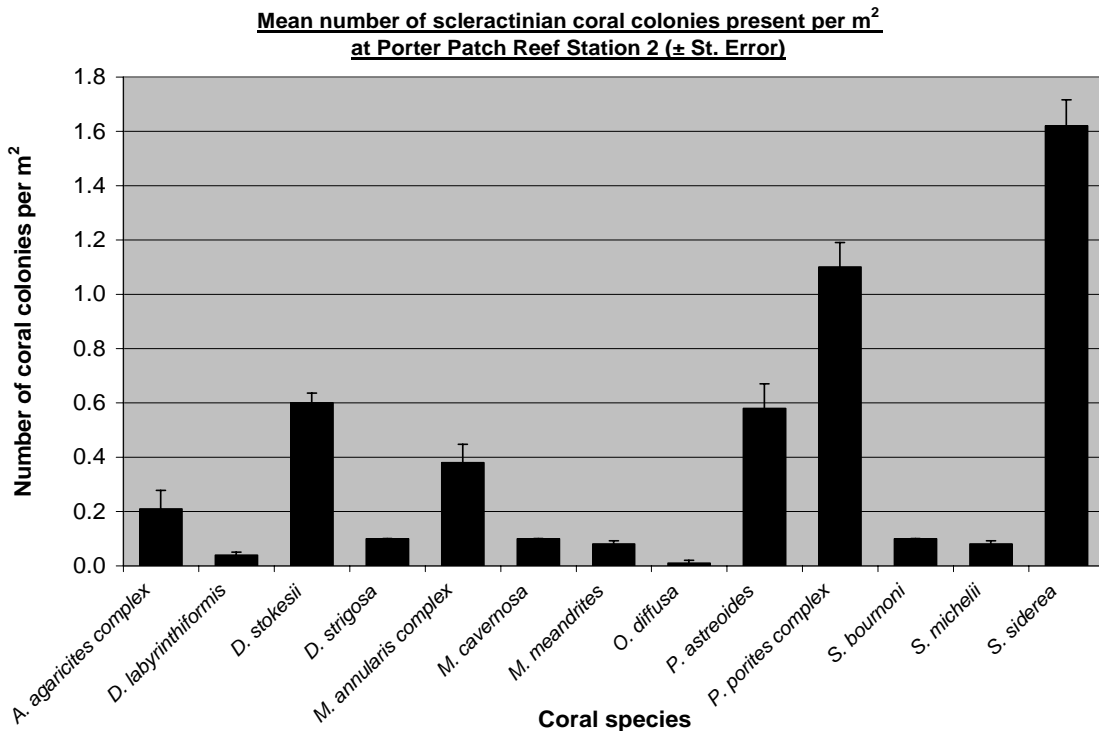
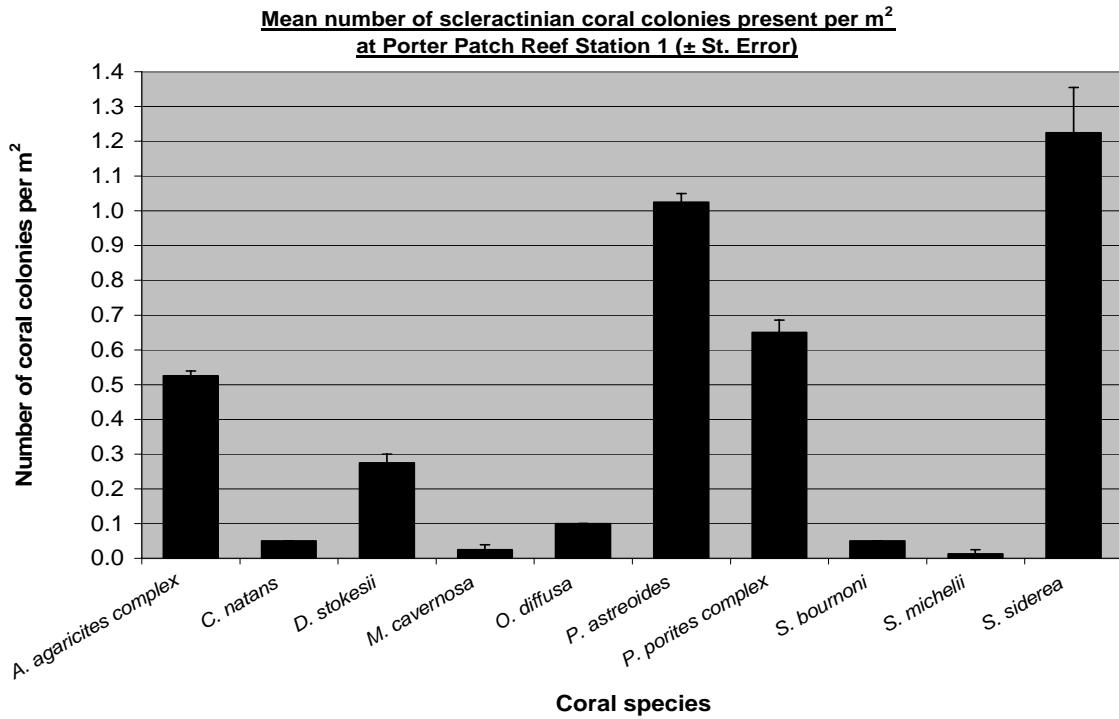
Mean number of scleractinian coral colonies present per m<sup>2</sup> at West Turtle Shoal Patch Reef Station 3 (above) and 4 (below).



Mean number of scleractinian coral colonies present per m<sup>2</sup> at Grecian Rocks Offshore Shallow Reef Station 1 (above) and 2 (below).

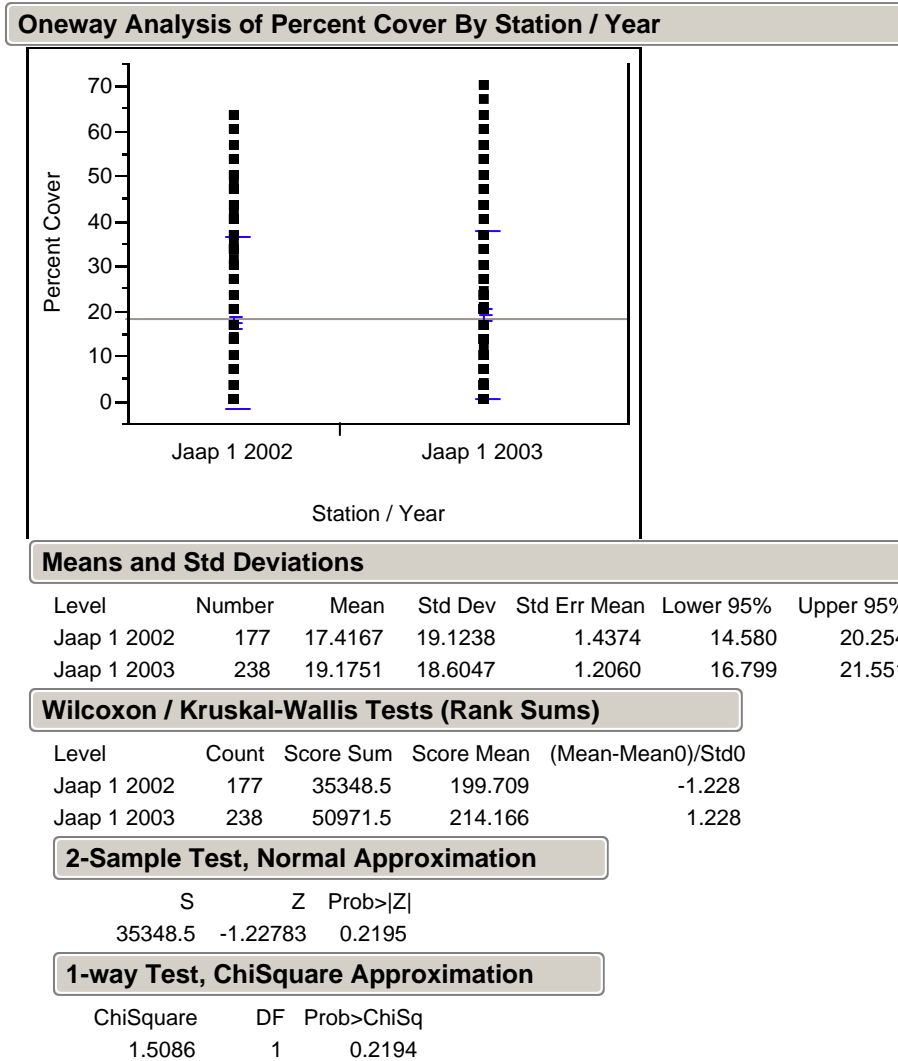


Mean number of scleractinian coral colonies present per m<sup>2</sup> at Porter Patch Reef Station 1 (above) and 2 (below).



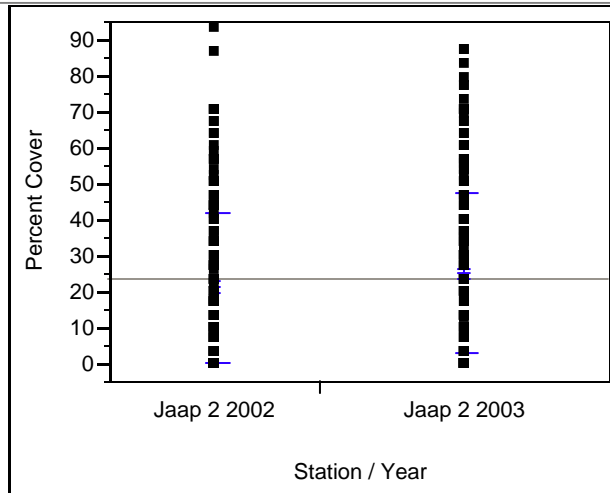
### APPENDIX III

Wilcoxon's Rank Sum tests comparing mean percent live coral cover for Jaap Patch Reef Station 1 in 2002 and 2003.



Wilcoxon's Rank Sum tests comparing mean percent live coral cover for Jaap Patch Reef Station 2 for 2002 and 2003.

**Oneway Analysis of Percent Cover By Station / Year**



**Means and Std Deviations**

Level	Number	Mean	Std Dev	Std Err Mean	Lower 95%	Upper 95%
Jaap 2 2002	170	21.1955	20.7016	1.5877	18.061	24.330
Jaap 2 2003	232	25.0629	22.1728	1.4557	22.195	27.931

**Wilcoxon / Kruskal-Wallis Tests (Rank Sums)**

Level	Count	Score Sum	Score Mean	(Mean-Mean0)/Std0
Jaap 2 2002	170	32378	190.459	-1.637
Jaap 2 2003	232	48625	209.591	1.637

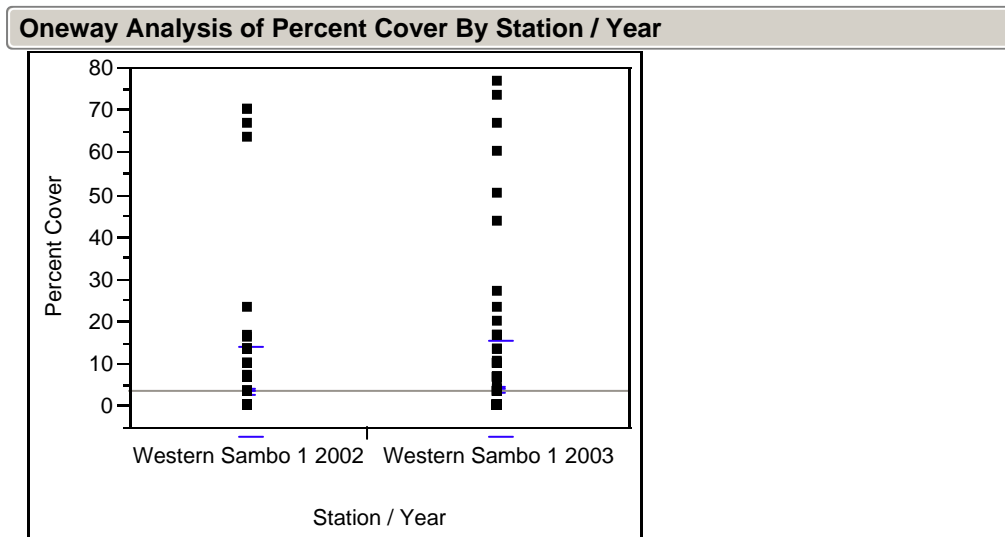
**2-Sample Test, Normal Approximation**

S	Z	Prob> Z
32378	-1.63718	0.1016

**1-way Test, ChiSquare Approximation**

ChiSquare	DF	Prob>ChiSq
2.6818	1	0.1015

Wilcoxon's Rank Sum tests comparing mean percent live coral cover for Western Sambo Offshore Shallow Reef Station 1 in 2002 and 2003.



**Means and Std Deviations**

Level	Number	Mean	Std Dev	Std Err Mean	Lower 95%	Upper 95%
Western Sambo 1 2002	183	3.56774	10.5487	0.77978	2.0292	5.1063
Western Sambo 1 2003	199	4.01257	11.3664	0.80574	2.4236	5.6015

**Wilcoxon / Kruskal-Wallis Tests (Rank Sums)**

Level	Count	Score Sum	Score Mean	(Mean-Mean0)/Std0
Western Sambo 1 2002	183	34697.5	189.604	-0.405
Western Sambo 1 2003	199	38455.5	193.244	0.405

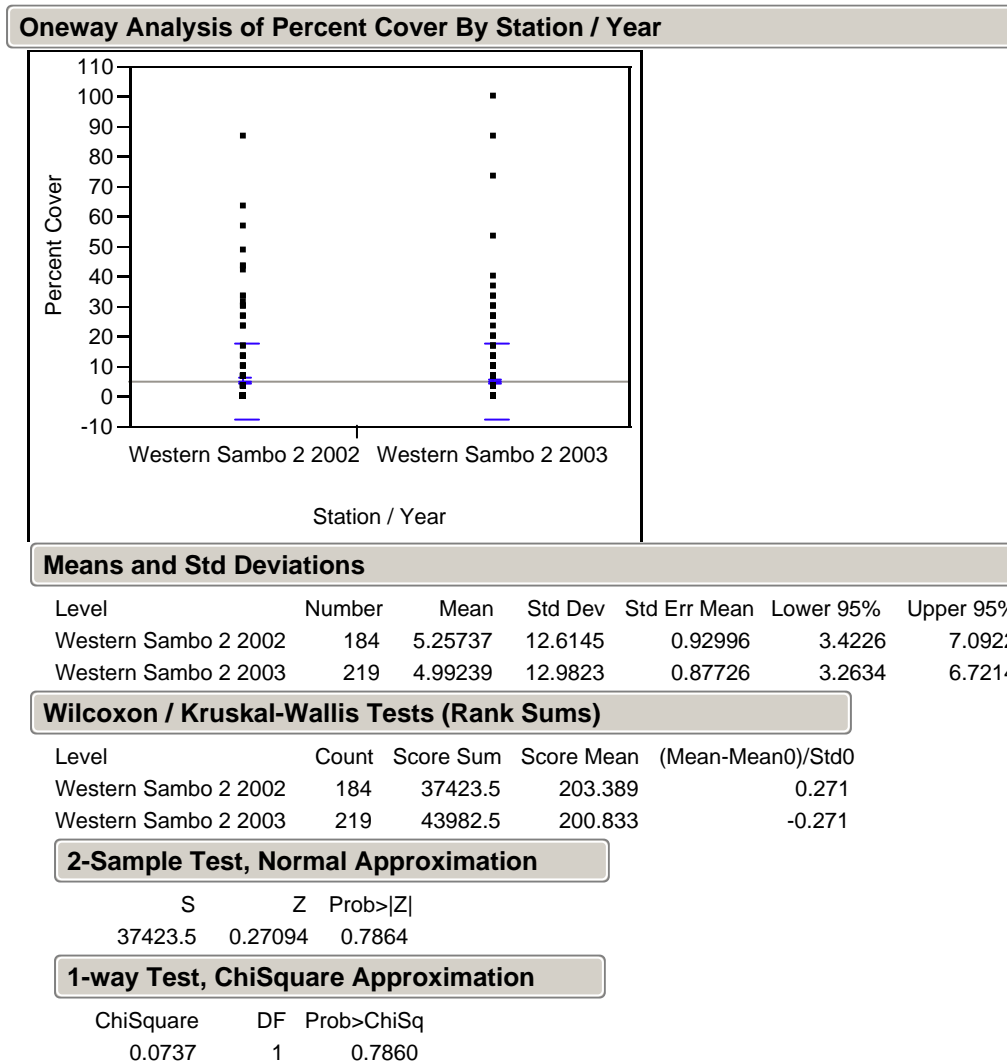
**2-Sample Test, Normal Approximation**

S	Z	Prob> Z
34697.5	-0.40492	0.6855

**1-way Test, ChiSquare Approximation**

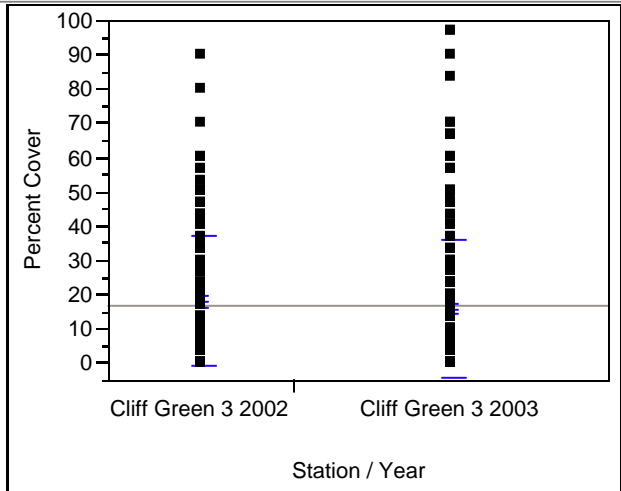
ChiSquare	DF	Prob>ChiSq
0.1644	1	0.6851

Wilcoxon's Rank Sum tests comparing mean percent live coral cover for Western Sambo Offshore Shallow Reef Station 2 in 2002 and 2003.



Wilcoxon's Rank Sum tests comparing mean percent live coral cover for Cliff Green Patch Reef Station 3 in 2002 and 2003.

**Oneway Analysis of Percent Cover By Station / Year**



**Means and Std Deviations**

Level	Number	Mean	Std Dev	Std Err Mean	Lower 95%	Upper 95%
Cliff Green 3 2002	141	18.1770	19.0035	1.6004	15.013	21.341
Cliff Green 3 2003	237	15.8746	20.0835	1.3046	13.305	18.445

**Wilcoxon / Kruskal-Wallis Tests (Rank Sums)**

Level	Count	Score Sum	Score Mean	(Mean-Mean0)/Std0
Cliff Green 3 2002	141	28696.5	203.521	1.946
Cliff Green 3 2003	237	42934.5	181.158	-1.946

**2-Sample Test, Normal Approximation**

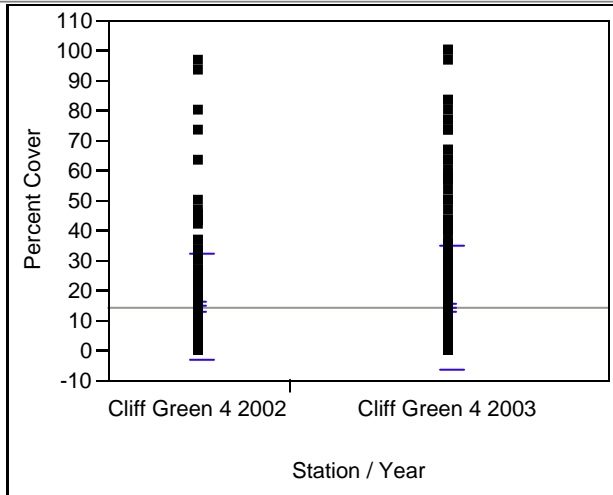
S	Z	Prob> Z
28696.5	1.94576	0.0517

**1-way Test, ChiSquare Approximation**

ChiSquare	DF	Prob>ChiSq
3.7879	1	0.0516

Wilcoxon's Rank Sum tests comparing mean percent live coral cover for Cliff Green Patch Reef Station 4 in 2002 and 2003.

**Oneway Analysis of Percent Cover By Station / Year**



**Means and Std Deviations**

Level	Number	Mean	Std Dev	Std Err Mean	Lower 95%	Upper 95%
Cliff Green 4 2002	142	14.7432	17.8337	1.4966	11.785	17.702
Cliff Green 4 2003	248	14.2928	20.9581	1.3308	11.672	16.914

**Wilcoxon / Kruskal-Wallis Tests (Rank Sums)**

Level	Count	Score Sum	Score Mean	(Mean-Mean0)/Std0
Cliff Green 4 2002	142	29802	209.873	1.935
Cliff Green 4 2003	248	46443	187.270	-1.935

**2-Sample Test, Normal Approximation**

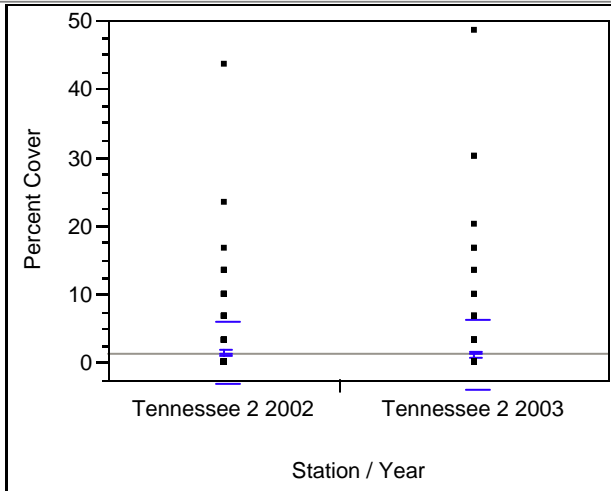
S	Z	Prob> Z
29802	1.93460	0.0530

**1-way Test, ChiSquare Approximation**

ChiSquare	DF	Prob>ChiSq
3.7445	1	0.0530

Wilcoxon's Rank Sum tests comparing mean percent live coral cover for Tennessee Offshore Shallow Reef Station 2 in 2002 and 2003.

**Oneway Analysis of Percent Cover By Station / Year**



**Means and Std Deviations**

Level	Number	Mean	Std Dev	Std Err Mean	Lower 95%	Upper 95%
Tennessee 2 2002	196	1.54762	4.60861	0.32919	0.89840	2.1968
Tennessee 2 2003	226	1.30506	5.04355	0.33549	0.64395	1.9662

**Wilcoxon / Kruskal-Wallis Tests (Rank Sums)**

Level	Count	Score Sum	Score Mean	(Mean-Mean0)/Std0
Tennessee 2 2002	196	42570.5	217.196	1.432
Tennessee 2 2003	226	46682.5	206.560	-1.432

**2-Sample Test, Normal Approximation**

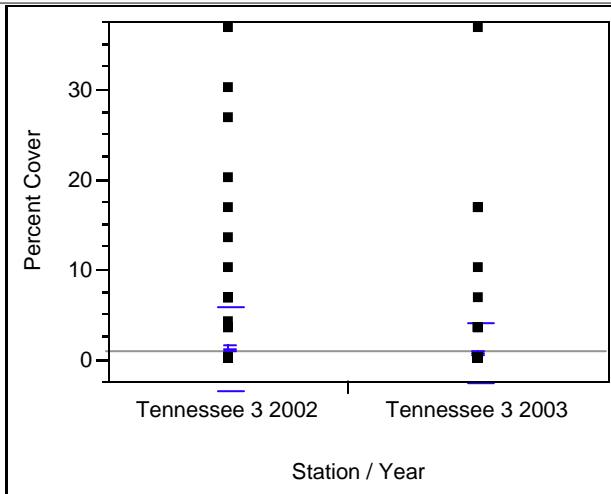
S	Z	Prob> Z
42570.5	1.43161	0.1523

**1-way Test, ChiSquare Approximation**

ChiSquare	DF	Prob>ChiSq
2.0513	1	0.1521

Wilcoxon's Rank Sum tests comparing mean percent live coral cover for Tennessee Offshore Shallow Reef Station 3 in 2002 and 2003.

**Oneway Analysis of Percent Cover By Station / Year**



**Means and Std Deviations**

Level	Number	Mean	Std Dev	Std Err Mean	Lower 95%	Upper 95%
Tennessee 3 2002	194	1.18986	4.58303	0.32904	0.54088	1.8388
Tennessee 3 2003	208	0.64103	3.35986	0.23296	0.18174	1.1003

**Wilcoxon / Kruskal-Wallis Tests (Rank Sums)**

Level	Count	Score Sum	Score Mean	(Mean-Mean0)/Std0
Tennessee 3 2002	194	40057.5	206.482	1.635
Tennessee 3 2003	208	40945.5	196.853	-1.635

**2-Sample Test, Normal Approximation**

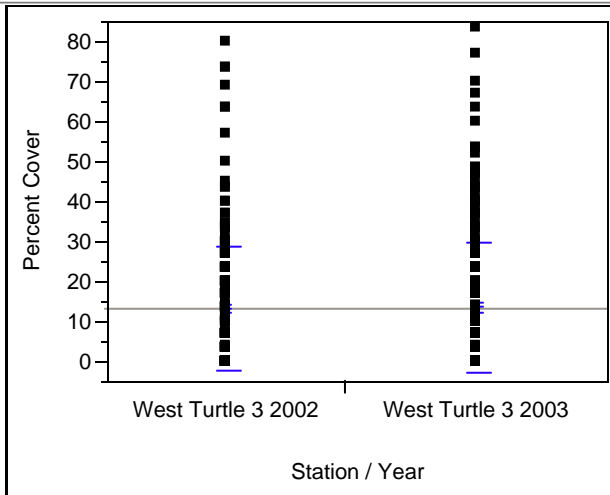
S	Z	Prob> Z
40057.5	1.63517	0.1020

**1-way Test, ChiSquare Approximation**

ChiSquare	DF	Prob>ChiSq
2.6765	1	0.1018

Wilcoxon's Rank Sum tests comparing mean percent live coral cover for West Turtle Shoal Patch Reef Station 3 in 2002 and 2003.

**Oneway Analysis of Percent Cover By Station / Year**



**Means and Std Deviations**

Level	Number	Mean	Std Dev	Std Err Mean	Lower 95%	Upper 95%
West Turtle 3 2002	207	13.3588	15.5231	1.0789	11.232	15.486
West Turtle 3 2003	225	13.5601	16.3657	1.0910	11.410	15.710

**Wilcoxon / Kruskal-Wallis Tests (Rank Sums)**

Level	Count	Score Sum	Score Mean	(Mean-Mean0)/Std0
West Turtle 3 2002	207	45024	217.507	0.162
West Turtle 3 2003	225	48504	215.573	-0.162

**2-Sample Test, Normal Approximation**

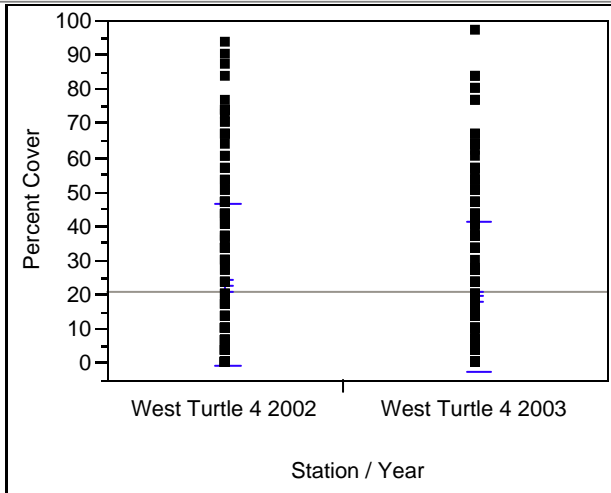
S	Z	Prob> Z
45024	0.16233	0.8710

**1-way Test, ChiSquare Approximation**

ChiSquare	DF	Prob>ChiSq
0.0265	1	0.8707

Wilcoxon's Rank Sum tests comparing mean percent live coral cover for West Turtle Shoal Patch Reef Station 4 in 2002 and 2003.

**Oneway Analysis of Percent Cover By Station / Year**



**Means and Std Deviations**

Level	Number	Mean	Std Dev	Std Err Mean	Lower 95%	Upper 95%
West Turtle 4 2002	194	22.9001	23.6159	1.6955	19.556	26.244
West Turtle 4 2003	215	19.5198	21.8779	1.4921	16.579	22.461

**Wilcoxon / Kruskal-Wallis Tests (Rank Sums)**

Level	Count	Score Sum	Score Mean	(Mean-Mean0)/Std0
West Turtle 4 2002	194	41327	213.026	1.313
West Turtle 4 2003	215	42518	197.758	-1.313

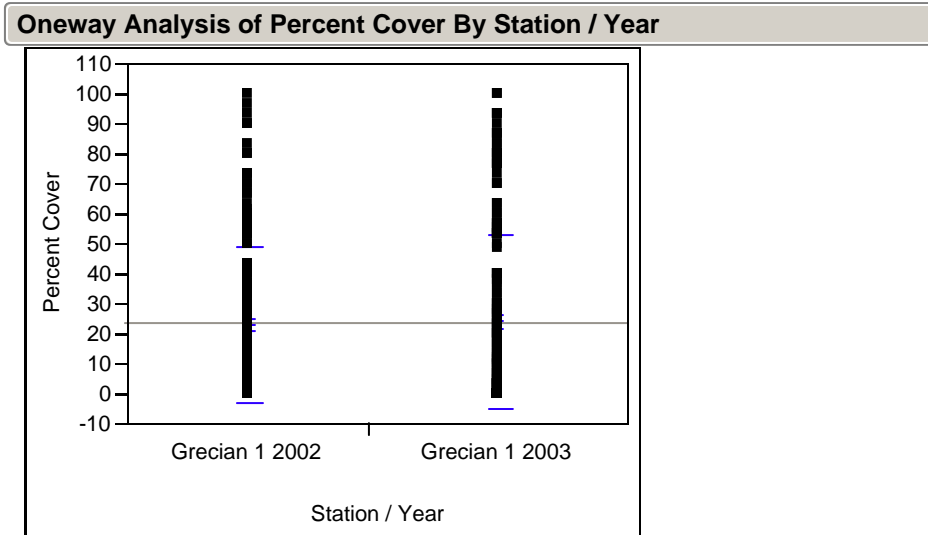
**2-Sample Test, Normal Approximation**

S	Z	Prob> Z
41327	1.31344	0.1890

**1-way Test, ChiSquare Approximation**

ChiSquare	DF	Prob>ChiSq
1.7262	1	0.1889

Wilcoxon's Rank Sum tests comparing mean percent live coral cover for Grecian Rocks Offshore Shallow Reef Station 1 in 2002 and 2003.



**Means and Std Deviations**

Level	Number	Mean	Std Dev	Std Err Mean	Lower 95%	Upper 95%
Grecian 1 2002	157	23.0498	25.9853	2.0739	18.953	27.146
Grecian 1 2003	167	24.1839	29.1238	2.2537	19.734	28.633

**Wilcoxon / Kruskal-Wallis Tests (Rank Sums)**

Level	Count	Score Sum	Score Mean	(Mean-Mean0)/Std0
Grecian 1 2002	157	25716	163.796	0.244
Grecian 1 2003	167	26934	161.281	-0.244

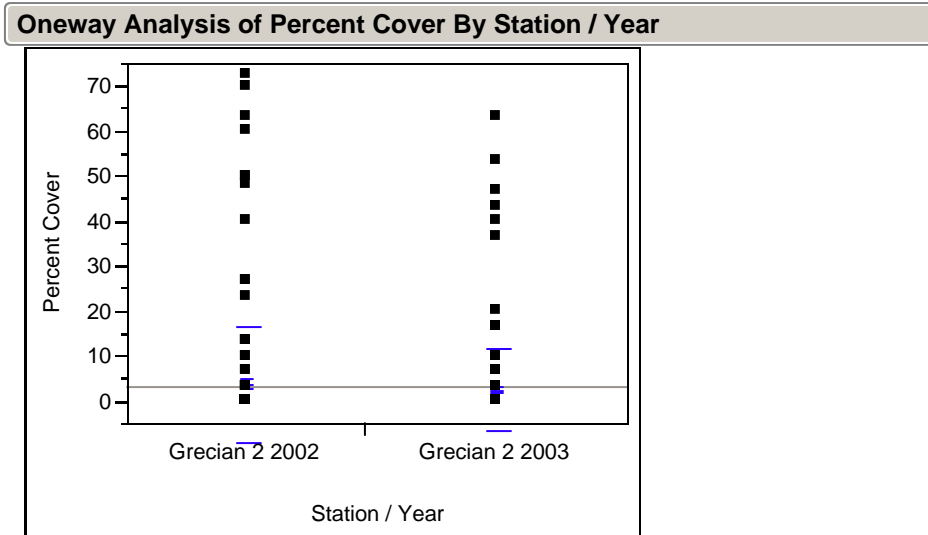
**2-Sample Test, Normal Approximation**

S	Z	Prob> Z
25716	0.24423	0.8071

**1-way Test, ChiSquare Approximation**

ChiSquare	DF	Prob>ChiSq
0.0599	1	0.8066

Wilcoxon's Rank Sum tests comparing mean percent live coral cover for Grecian Rocks Offshore Shallow Reef Station 2 in 2002 and 2003.



**Means and Std Deviations**

Level	Number	Mean	Std Dev	Std Err Mean	Lower 95%	Upper 95%
Grecian 2 2002	164	3.78469	12.9133	1.0084	1.7936	5.7758
Grecian 2 2003	179	2.45810	9.0382	0.6755	1.1250	3.7912

**Wilcoxon / Kruskal-Wallis Tests (Rank Sums)**

Level	Count	Score Sum	Score Mean	(Mean-Mean0)/Std0
Grecian 2 2002	164	28142	171.598	-0.116
Grecian 2 2003	179	30854	172.369	0.116

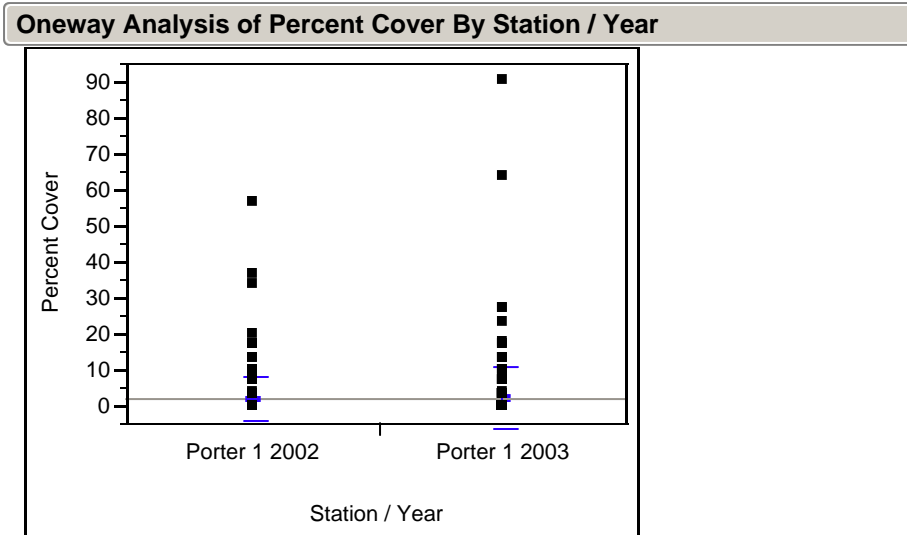
**2-Sample Test, Normal Approximation**

S	Z	Prob> Z
28142	-0.11635	0.9074

**1-way Test, ChiSquare Approximation**

ChiSquare	DF	Prob>ChiSq
0.0137	1	0.9067

Wilcoxon's Rank Sum tests comparing mean percent live coral cover for Porter Patch Reef Station 1 in 2002 and 2003.



**Means and Std Deviations**

Level	Number	Mean	Std Dev	Std Err Mean	Lower 95%	Upper 95%
Porter 1 2002	216	2.05418	6.13038	0.41712	1.2320	2.8764
Porter 1 2003	208	2.23088	8.60556	0.59669	1.0545	3.4072

**Wilcoxon / Kruskal-Wallis Tests (Rank Sums)**

Level	Count	Score Sum	Score Mean	(Mean-Mean0)/Std0
Porter 1 2002	216	46462.5	215.104	0.638
Porter 1 2003	208	43637.5	209.796	-0.638

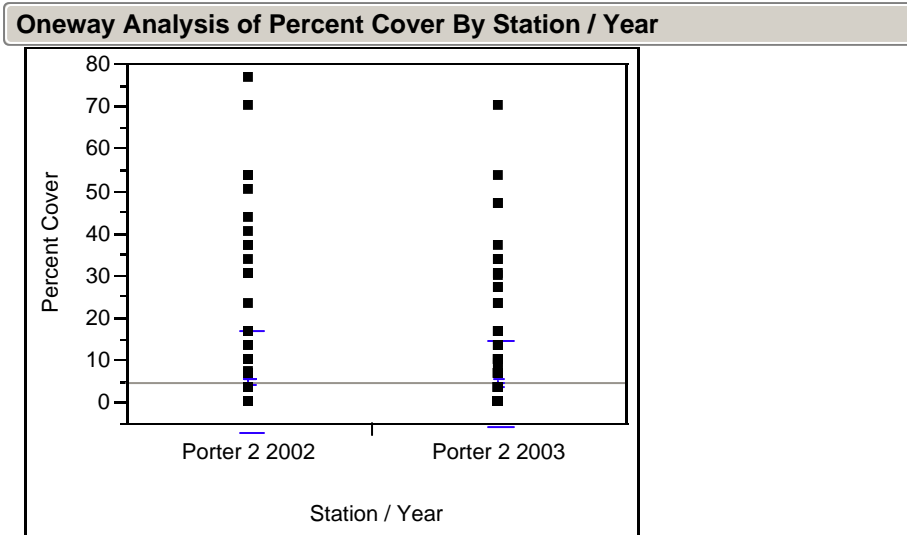
**2-Sample Test, Normal Approximation**

S	Z	Prob> Z
43637.5	-0.63756	0.5238

**1-way Test, ChiSquare Approximation**

ChiSquare	DF	Prob>ChiSq
0.4072	1	0.5234

Wilcoxon's Rank Sum tests comparing mean percent live coral cover for Porter Patch Reef Station 2 in 2002 and 2003.



**Means and Std Deviations**

Level	Number	Mean	Std Dev	Std Err Mean	Lower 95%	Upper 95%
Porter 2 2002	192	4.96648	12.0886	0.87242	3.2457	6.6873
Porter 2 2003	196	4.54279	10.0360	0.71686	3.1290	5.9566

**Wilcoxon / Kruskal-Wallis Tests (Rank Sums)**

Level	Count	Score Sum	Score Mean	(Mean-Mean0)/Std0
Porter 2 2002	192	36716.5	191.232	-0.662
Porter 2 2003	196	38749.5	197.702	0.662

**2-Sample Test, Normal Approximation**

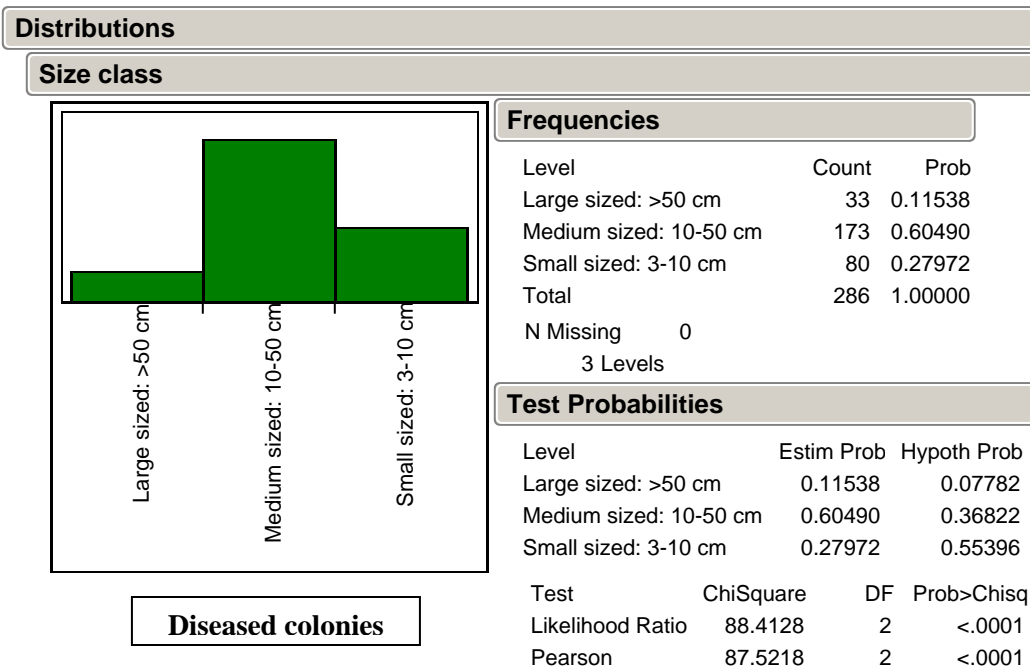
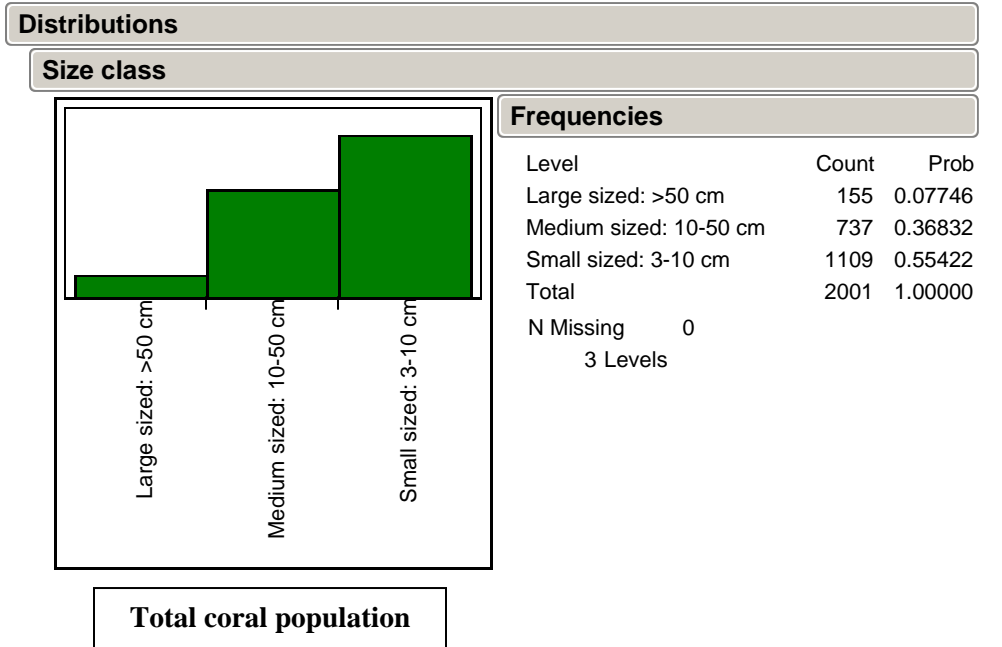
S	Z	Prob> Z
36716.5	-0.66168	0.5082

**1-way Test, ChiSquare Approximation**

ChiSquare	DF	Prob>ChiSq
0.4385	1	0.5078

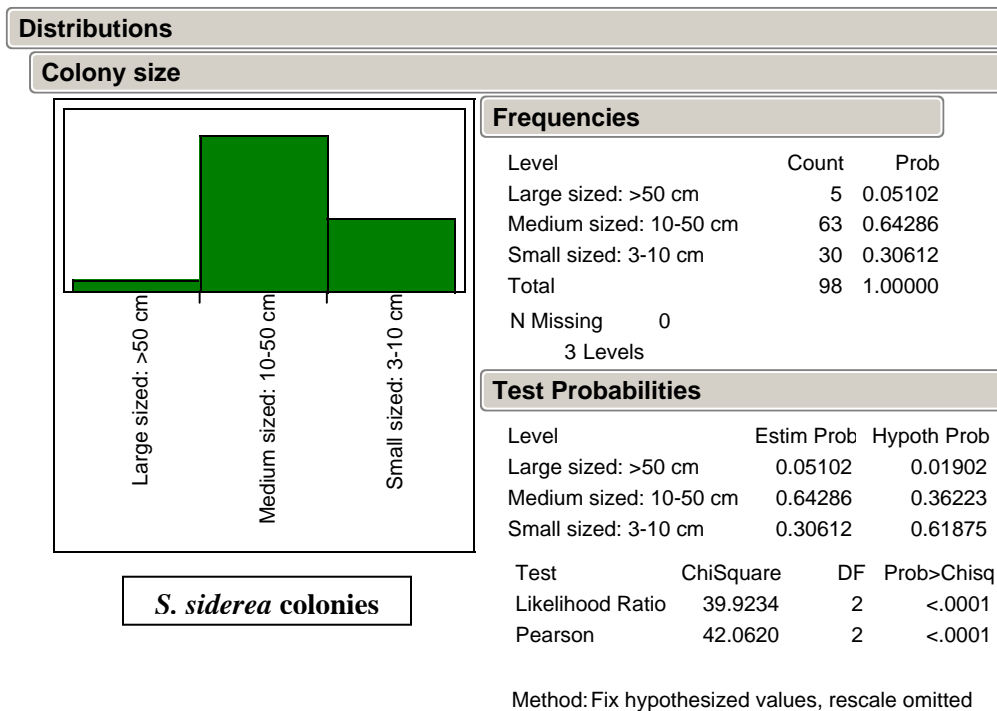
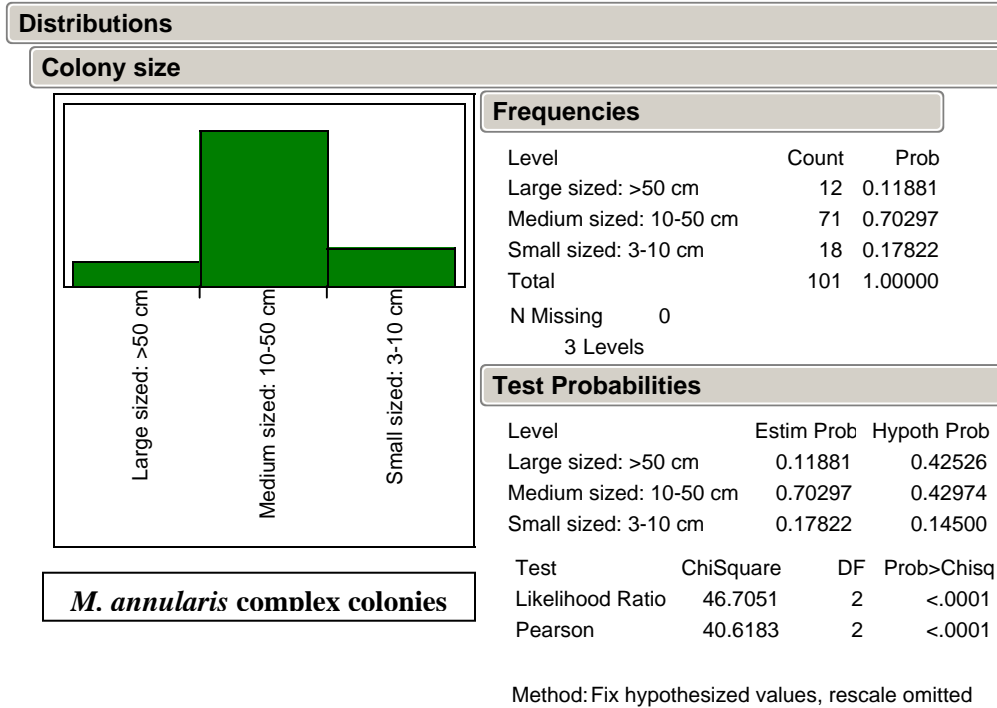
## APPENDIX IV

Size class distribution of all healthy (above) and diseased (below) scleractinian coral colonies identified in the first 20 m<sup>2</sup> of all 14 stations surveyed between 2002 and 2004, and results of G<sup>2</sup> Likelihood-Ratio Chi-Square tests.

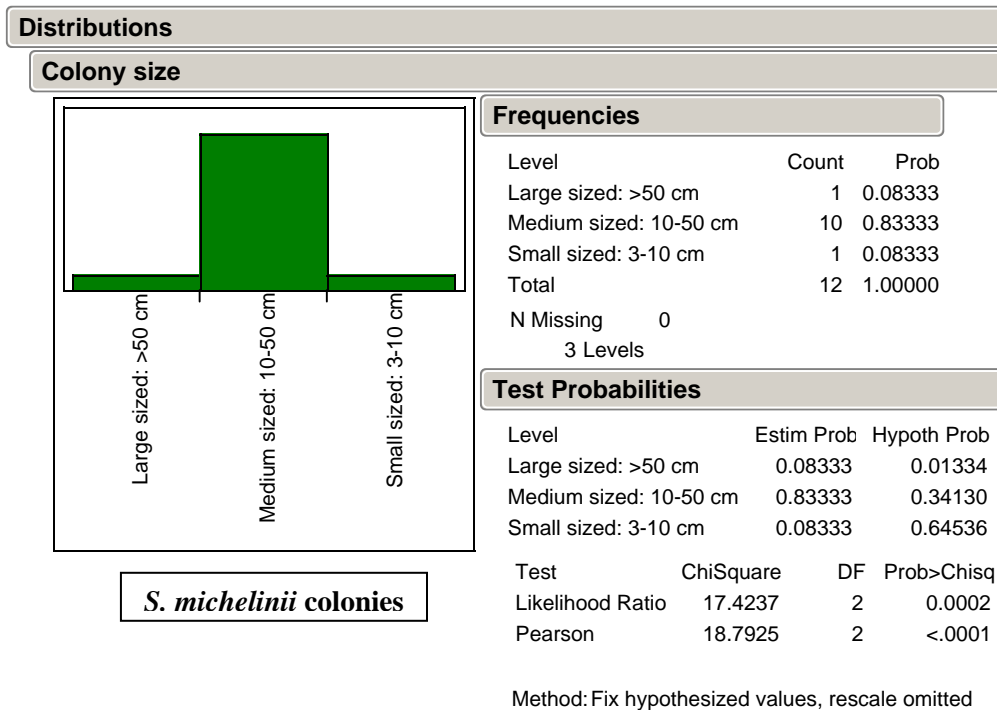


Method: Fix hypothesized values, rescale omitted

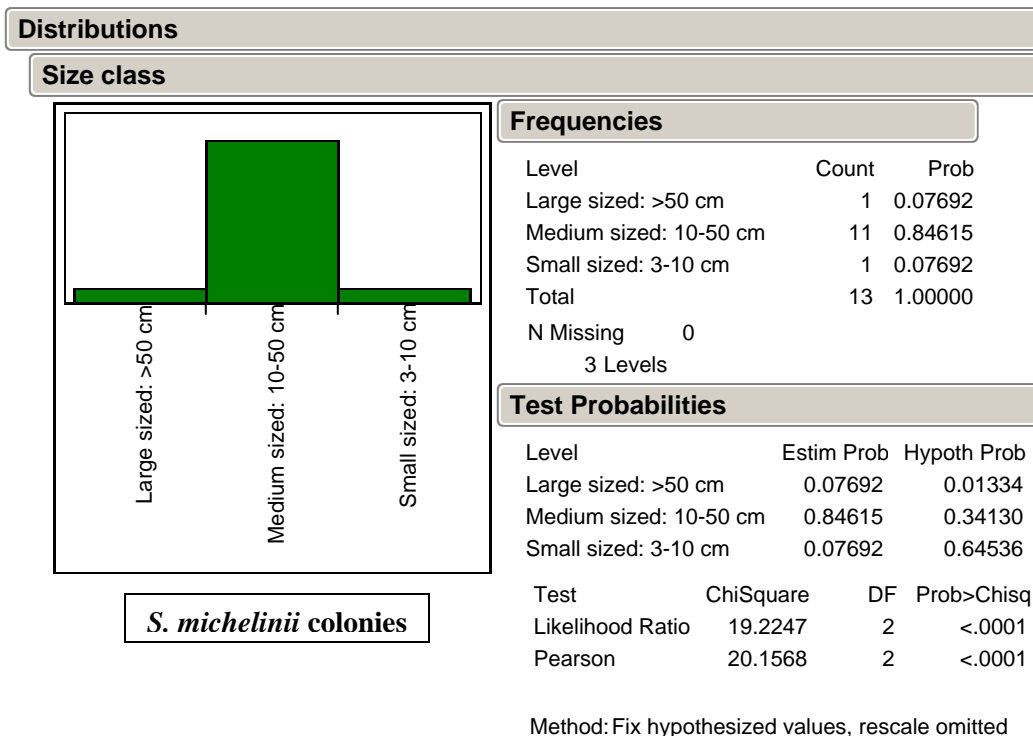
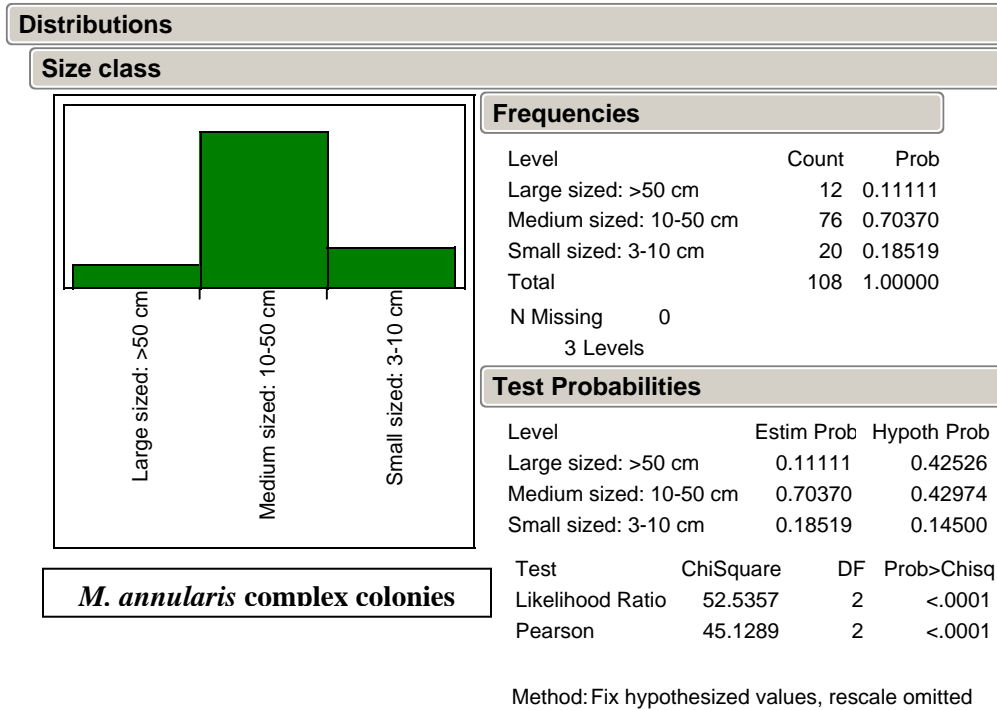
Size class distribution and results of  $G^2$  Likelihood-Ratio Chi-Square tests for *Montastrea annularis* complex (above) colonies and *Siderastrea siderea* colonies (below) identified with dark color syndrome in the first 20 m<sup>2</sup> of all 14 stations surveyed from 2002 to 2004.



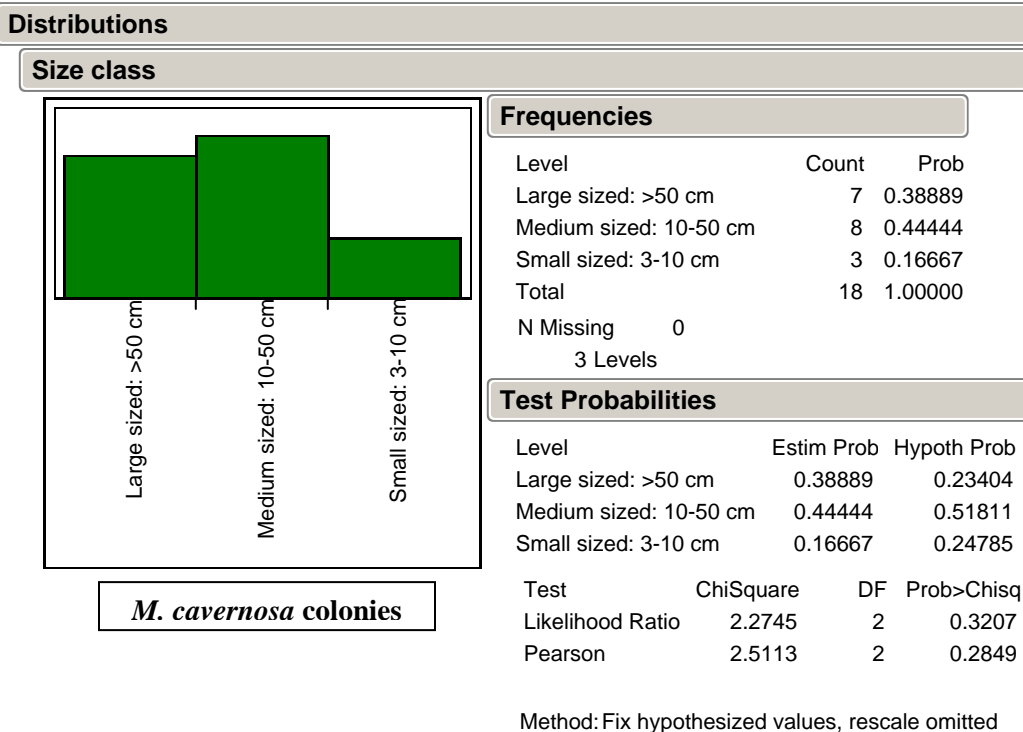
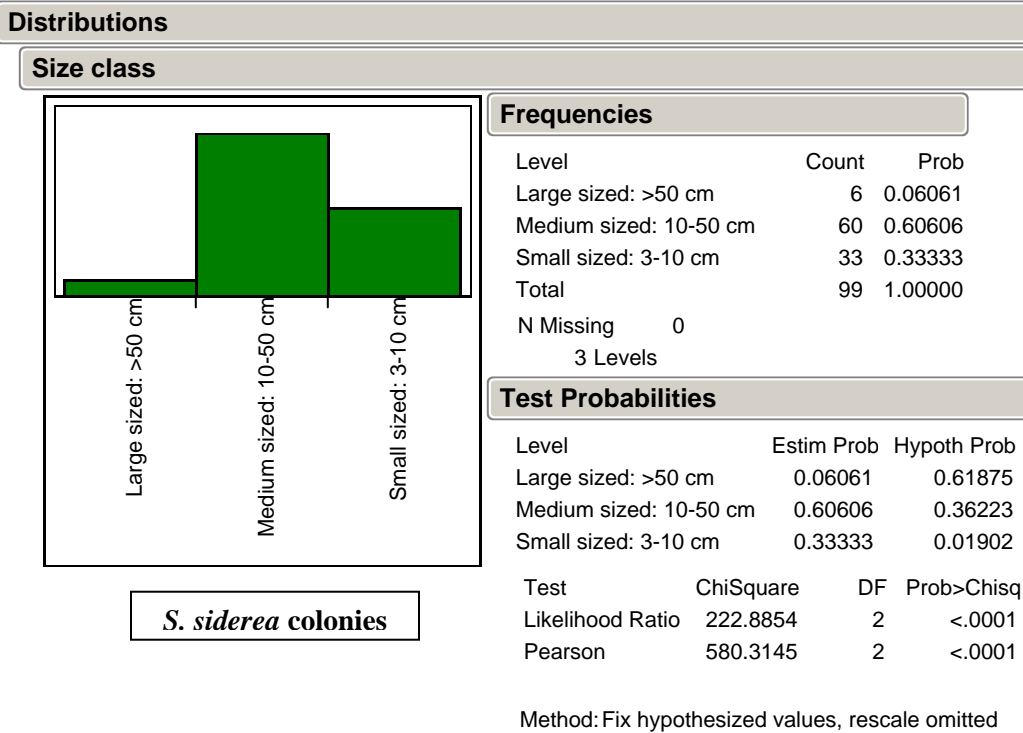
Size class distribution and results of  $G^2$  Likelihood-Ratio Chi-Square tests for *Stephanocoenia michelinii* colonies identified with dark color syndrome in the first 20 m<sup>2</sup> of all 14 stations surveyed from 2002 to 2004.



Size class distribution and results of  $G^2$  Likelihood-Ratio Chi-Square tests for all diseased colonies (including all diseases) of *Montastrea annularis* complex (above) and *Stephanocoenia michelinii* (below) in the first 20 m<sup>2</sup> of all 14 stations surveyed from 2002 to 2004.



Size class distribution and results of  $G^2$  Likelihood-Ratio Chi-Square tests for all diseased colonies (including all diseases) of *Siderastrea siderea* (above) and *Montastrea cavernosa* (below) in the first 20 m<sup>2</sup> of all 14 stations surveyed from 2002 to 2004.



Size class distribution and results of  $G^2$  Likelihood-Ratio Chi-Square tests for all diseased colonies (including all diseases) of *Agaricia agaricites* complex in the first 20 m<sup>2</sup> of all 14 stations surveyed from 2002 to 2004.

