16 White Pox Disease of the Caribbean Elkhorn Coral, *Acropora palmata*

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16.1
Introduction

Populations of the most common Caribbean reef-building coral, *Acropora palmata*, are being decimated by white pox disease, with losses of living cover in the Florida Keys National Marine Sanctuary (FKNMS) averaging 88%. Elkhorn coral plays a significant role in the structural and functional integrity of Caribbean coral reef ecosystems. *A. palmata* is an important shallow water species, providing elevated rates of calcium carbonate deposition (Adey 1978) and a highly complex three-dimensional structure of the shallow water fore reef. This keystone species provides shelter and food for reef organisms and aids in the protection of coastal regions by serving as a buffer between land and sea. Severe population declines of *A. palmata* in the FKNMS and elsewhere in the Caribbean have led to the identification of this species as a candidate for inclusion on the Endangered Species List (Diaz-Soltero 1999).

White pox disease, also termed acroporid serratiosis (Patterson et al. 2002), and patchy necrosis (Bruckner and Bruckner 1997), exclusively affects *Acropora palmata* and was first documented in 1996 on reefs off Key West, Florida (Holden 1996). White pox has since been observed throughout the Caribbean (Porter et al. 2001; Rodríquez-Martínez et al. 2001; Santavy et al. 2001; Patterson et al. 2002) and exclusively affects *A. palmata.* White pox disease is characterized by irregularly shaped, distinct white patches devoid of coral tissue (Fig. 16.1). Lesions can vary from a few to greater than 80 cm^2 and can develop simultaneously on all surfaces of the coral colony. Coral tissue loss occurs radially along the perimeter of the lesion at an average rate of 2.5 cm^2/day and is greatest during periods of seasonally elevated temperature. In heavily affected *A. palmata*, lesions can merge, resulting in tissue loss that spans the entire colony. White pox is highly contagious with nearest neighbors most susceptible to infection (Patterson et al. 2002). The disease spread rapidly within and between reefs in the FKNMS during the mid-1990s (Porter et al. 2001; Patterson et al. 2002).

The distinct white patches and the potential for tissue loss throughout the coral colony distinguishes white pox disease from white band disease, which also affects *A. palmata*, developing at the base of a coral branch and progressing upward toward the branch tip in a concentric ring. White band disease is characterized by a distinct white band of recently denuded coral skeleton (type I; Gladfelter 1982), or bleached tissue (type II; Ritchie and Smith 1998), fol-

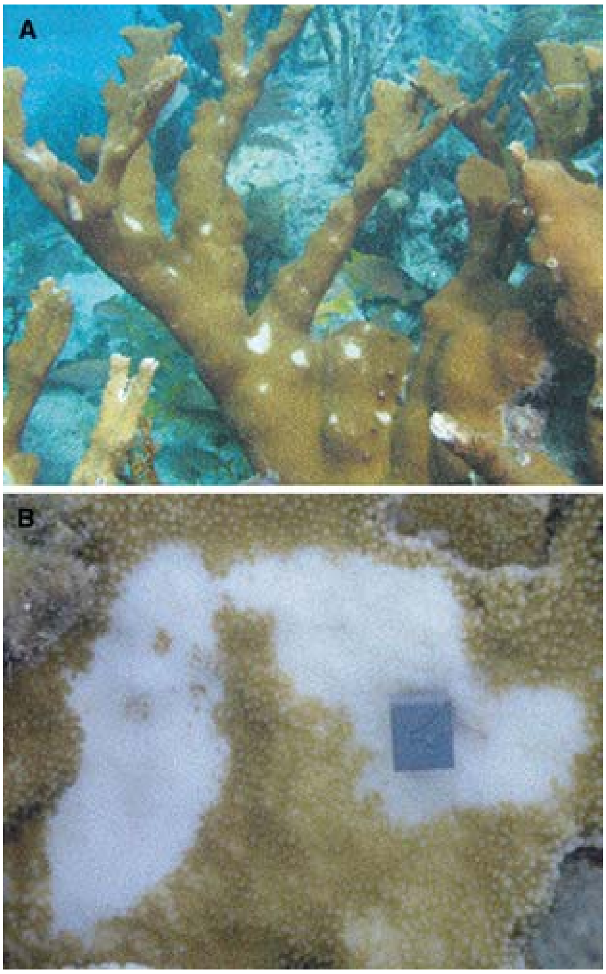

Fig. 16.1. A *Acropora palmata* colony affected with white pox disease. (Photograph by J.W. Porter). **B** White pox disease lesions on *A. palmata.* (Photograph by K.P. Sutherland)

lowed by a line of necrosis. White pox disease signs also clearly differ from coral bleaching and predation scars produced by the corallivorous snail, *Coralliophila abbreviata* (Knowlton et al. 1990; Miller 2001).

16.2
The Coral Pathogen, *Serratia marcescens*

White pox disease is caused by *Serratia marcescens* (Patterson et al. 2002), a common Gram-negative bacterium classified as a coliform and a member of the Enterobacteriaceae family. *S. marcescens* is found in the intestines of humans, insects, and other animals, and in fresh water, soil, and plants (Grimont and Grimont 1994). *S. marcescens* is pathogenic to humans, cows, goats, chickens, fishes, insects, and plants (Baya et al. 1992; Grimont and Grimont 1994). *S. marcescens* is an opportunistic pathogen of humans associated with both waterborne infections in tropical waters (Hazen 1988) and hospital-acquired infections, including urinary tract infections, wound infections, pneumonia, and bacteremia (Grimont and Grimont 1994; Miranda et al. 1996; Shi et al. 1997).

The pathogenic mechanism of *S. marcescens* for elkhorn coral is currently unknown, thus our best available model for pathogenesis is derived from human clinical studies. *S. marcescens* is known to produce a number of virulence factors including serralysin (a peptidase), proteases, chitinases, extracellular lipases and nucleases (Hertle 2002). *S. marcescens* cell wall includes a lipopolysaccharide (LPS) that is responsible for endotoxin activity (Hejazi and Falkiner 1997). A hemophore secreted by *S. marcescens* was shown to be cytotoxic to cells other than red blood cells (Arnoux et al. 1999; Marty et al. 2002). Polson (2002) showed that the white pox isolate also produces lipases and nucleases, but their role in pathogenesis is presently unknown. There is an inverse relationship between pigment production (prodigiosin) and toxicity of *S. marcescens* strains against nematodes (Carbonelli et al. 2000). Likewise, human clinical isolates are rarely pigmented (Hejazi and Falkiner 1997). The white pox isolate is nonpigmented and, therefore, does not produce prodigiosin, but the role of the pigment in pathogenesis is also unclear. Hejazi and Falkiner (1997) described adherence and pathogenicity differences between *S. marcescens* strains exhibiting mannose-resistant and mannose-sensitive surface pili. Mannose-sensitive pili are important in bacterial colonization. In *Vibrio cholerae* mannose-sensitive hemagglutinin (MSHA) mediates the adherence to commensal zooplankton carapaces (Chiavelli et al. 2001).

Quorum sensing is a signaling process by which a number of marine (and other) bacteria turn on genes (including those for virulence factors) as a result of the production of the signal compound by other bacteria. Most known quorum sensing systems of Gram-negative bacteria involve the secretion of N-acyl-homoserine lactone or similar lactones and/or quinolones. Swarming and the production of protease (behaviors found in other quorum sensors) have been reported for *Serratia* species (Givskov et al. 1997; Lindum et al. 1998).

The prevalence of *S. marcescens* in the marine environment is unknown. However, this bacterium has been found in the marine environment in sewage-polluted estuaries. For example, *S. marcescens* has been linked to disease of white perch (*Morone americanus*) in the sewage-polluted Back River, Maryland (Baya et al. 1992). Other *Serratia* species are known to cause disease in marine fishes (Austin and Austin 1999), and to pose a serious threat as opportunistic pathogens of marine organisms (Ingles et al. 1993).

White pox is one of approximately 18 coral diseases documented worldwide (Sutherland et al., 2003), but *S. marcescens* is only the fifth pathogen to be confirmed as a coral disease agent through the fulfillment of Koch's postulates (Kushmaro et al. 1996; Smith et al. 1996; Richardson et al. 1998a, b; Ben-Haim and Rosenberg 2002; Patterson et al. 2002) and the first agent with a possible link to human sewage pollution (Patterson et al. 2002). Identification of *S. marcescens* as a coral pathogen marked the first time that a common member of the human gut microbiota was shown to be a marine invertebrate pathogen. While *S. marcescens* is ubiquitous, its noted association with human hosts prompts speculation that improperly treated sewage from the Florida Keys may be associated with white pox disease. Human sewage markers (e.g., human enteric bacteria and viruses) are prevalent on coral surfaces and in nearshore, offshore, and canal waters of the FKNMS (Paul et al. 1995a, b, 1997; Griffin et al. 1999; Lipp et al. 2002), suggesting land-based activities may affect reef coral health and coral reef survival.

16.3
Loss of *Acropora palmata* in the Florida Keys National Marine Sanctuary: 1994–2002

Annual monitoring of Eastern Dry Rocks Reef (24°27.715′N, 81°50.801′W) in the FKNMS began in 1994 (Patterson et al. 2002). Seven additional reef sites in the FKNMS have been monitored on an annual basis since 1996: Carysfort Reef (25°13.205′N; 80°12.628′W), Grecian Rocks Reef (25°06.450′N; 80°18.410′W), Molasses Reef (25°00.525′N; 80°22.589′W), Rock Key Reef (24°27.285′N; 81°51.589′W), Sand Key Reef (24°27.119′N; 81°52.650′W), Sombrero Reef (24°37.531′N; 81°06.624′W), and Western Sambo Reef (24°28.771′N; 81°42.970′W; Patterson et al. 2002; Porter et al. 2002).

Between 1994 and 2002, the percent cover of *A. palmata* at Eastern Dry Rocks declined by 97%, from 23.9% in 1994 to 0.82% in 2002 (Fig. 16.2). Between 1996 and 2001, the percent cover of *A. palmata* dramatically declined at each of the seven additional reef sites: Carysfort, 90%; Grecian Rocks, 89%; Molasses, 90%; Rock Key, 79%; Sand Key, 100%; Sombrero, 100%; Western Sambo, 90% (Fig. 16.3). The complete loss of *A. palmata* at Sombrero Reef and Sand Key occurred by 1999 and 2001, respectively. Loss of *A. palmata* at all eight surveyed reefs in the FKNMS averaged 92% between 1996 and 2001 (Fig.16 3). Between 2001 and 2002, the percent cover of *A. palmata* increased at Carysfort, Molasses, and Rock Key and continued to decline at Grecian Rocks and Western Sambo. The continued loss was especially pronounced at Grecian Rocks where an additional 53% of *A. palmata* cover was lost, with a total loss at this reef of 95% between 1996 and 2002 (Fig. 16.3).

Populations of *Acropora palmata* in the FKNMS sustained losses averaging 88% between 1996 and 2002. These losses are approximately double the loss caused by white plague type II disease on *Dichocoenia stokesii* colonies in the FKNMS (Richardson et al. 1998a, b) and are comparable to *A. palmata* losses at other locations throughout the Caribbean (Bythell and Sheppard 1993; Aronson and Precht 2001; Miller et al. 2002). While *A. palmata* population declines elsewhere in the Caribbean were attributed to hurricanes, bleaching, and white band disease (Gladfelter 1982; Porter and Meier 1992; Bythell and

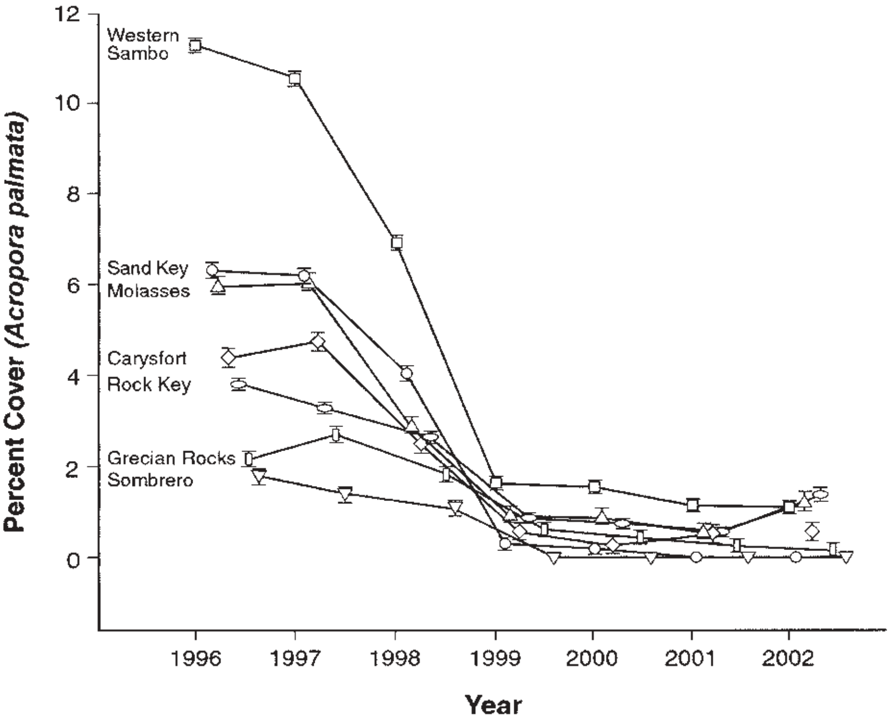

Fig. 16.2. Percent cover of *Acropora palmata* at seven reef sites in the Florida Keys National Marine Sanctuary, 1996–2002: Western Sambo Reef (*squares*), Sand Key Reef (*circles*), Molasses Reef (*triangles*), Carysfort Reef (*diamonds*), Rock Key Reef (*ovals*), Grecian Rocks Reef (*rectangles*), Sombrero Reef (*inverted triangles*). Data are presented as mean ± SD

Sheppard 1993; Aronson and Precht 2001; Miller et al. 2002), losses in the FKNMS were primarily caused by white pox disease. By 1997, 1 year after the first documentation of the disease (Holden 1996), white pox was found at all surveyed reefs in the FKNMS (Patterson et al. 2002; Porter et al. 2002). Signs of active white pox disease were observed on *A. palmata* colonies at Eastern Dry Rocks every year between 1996 and 2000 (Patterson et al. 2002) and at each of the other reefs with living cover of *A. palmata* every year between 1997 and 2002. One exception was Carysfort Reef, where white pox disease signs were not observed at the time of the 2000 survey. Observations of white band disease, on the other hand, were rare at monitored reefs in the FKNMS between 1996 and 2002.

In late September 1998, reefs in the FKNMS were impacted by both Hurricane Georges and mass-bleaching (Wilkinson et al. 1999). These events may have contributed to the coral decline observed between 1998 and 1999 (Fig. 16.3), though the 82% loss of *A. palmata* recorded at Eastern Dry Rocks between 1994 and 1998 (Fig. 16.2) occurred prior to both the hurricane and the bleaching event. Hurricane or bleaching damage may have contributed to the further

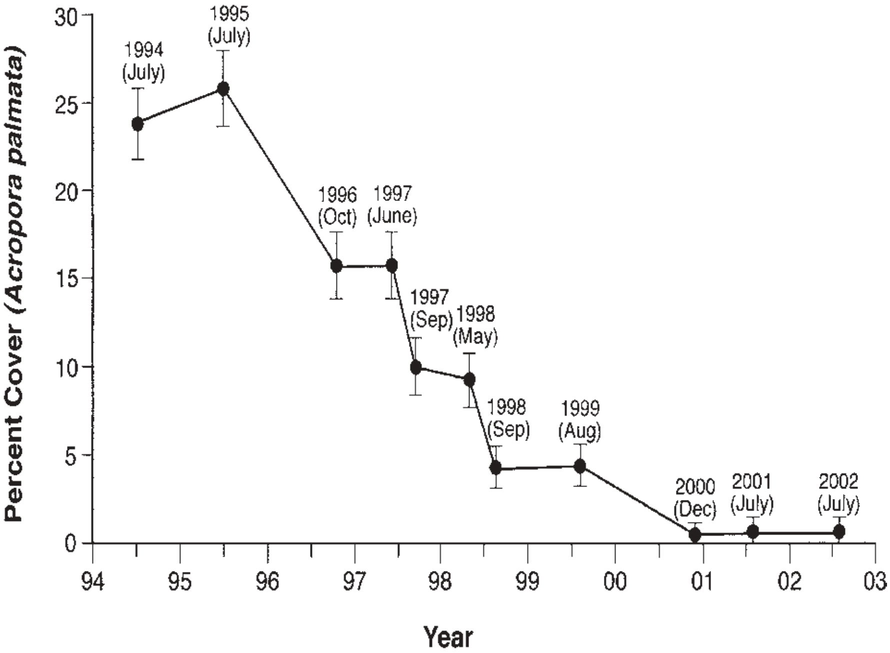

Fig. 16.3. Percent cover of *Acropora palmata* at Eastern Dry Rocks Reef, Key West, FL, 1994–2002. Data are presented as mean ± SD

decline of *A. palmata* on this reef after 1998. However, it is important to note that the first post-hurricane/post-bleaching survey (August 1999) showed a 3% increase in percent live cover of *A. palmata* (Fig. 16.2).

Between 2000 and 2001, living cover of *Acropora palmata* at Eastern Dry Rocks increased 66.7% from 0.49 to 0.82% (Fig. 16.2). This increase in living cover was due to growth of remaining fragments of living tissue (Fig. 16.4A, B). Coral recruitment was not observed at Eastern Dry Rocks at any time during the 9-year survey. All seven elkhorn colonies that exhibited tissue gain in 2001 showed either complete or partial tissue loss in 2002 (Fig. 16.4C). This loss may be attributed to disease or predation. While signs of active white pox disease were not observed on this reef at the time of the 2001 and 2002 surveys, percent cover of *A. palmata* was only 0.82% during these two survey years, and therefore, minimal surface area of living tissue was available for the disease to affect. Declining population numbers may make *A. palmata* especially vulnerable to white pox disease and predation by *Coralliophila abbreviata*, which preferentially feeds on this coral species (Knowlton et al. 1990; Miller 2001). Decimation of *A. palmata* populations may also limit the reproductive capacity of this species which reproduces almost exclusively by fragmentation (Aronson and Precht 2001). While vegetative reproduction may be well adapted to recolonization following mechanical disturbances such as hurricanes, colony fragmen-

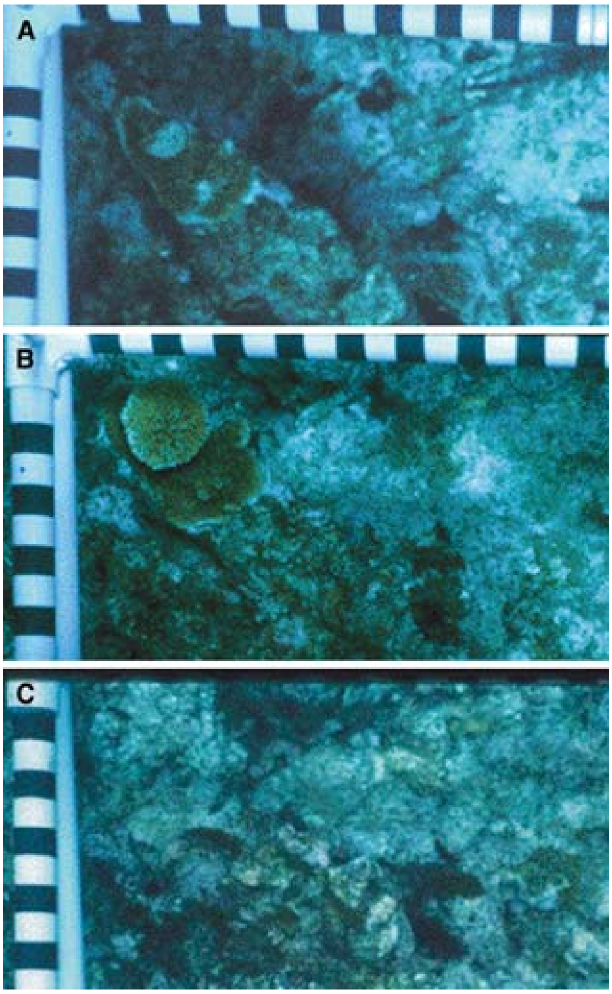

Fig. 16.4. Photographic time series of gain and loss of percent cover *Acropora palmata* at Eastern Dry Rocks Reef, Key West, FL, 2000–2002. The *A. palmata* fragment was photographed on **A** 28 December 2000 and **B** 30 July 2001. Despite the growth observed between 2000 and 2001, the fragment had disappeared by **C** 29 July 2002. This same pattern of tissue gain between 2000 and 2001, followed by tissue loss between 2001 and 2002 was observed on seven *A. palmata* colonies at this reef. (Photographs by J.W. Porter and K.P. Sutherland)

tation is ineffectual following severe population declines due to disease, which frequently kills the entire coral colony.

A. palmata colonies at Eastern Dry Rocks exhibited reduced tissue loss in the winter followed by accelerated tissue loss in the summer. This seasonal trend in percent change of *A. palmata* is evidenced by the stair-step pattern of tissue loss (Fig. 16.2) and may be attributable to the effects of seasonal seawater temperatures and precipitation on white pox disease pathogenicity. Elevated seawater temperature is a stressor in corals, causing thermally induced breakdown in the coral–zooxanthellae host–symbiont relationship (Brown 1997), promoting growth and virulence of pathogens (Kushmaro et al. 1996, 1998; Toren et al. 1998; Alker et al. 2001; Banin et al. 2001; Israely et al. 2001), and reducing immune response in host corals (Toren et al. 1998; Alker et al. 2001). Increased rainfall may increase seepage of sewage from septic tanks (Rose et al. 2001) and seed the marine environment with *Serratia marcescens* and other human fecal enteric bacteria and viruses.

The white pox disease epizootic has caused catastrophic losses of *A. palmata* in the FKNMS in just 9 years. These losses illustrate the impact that disease can have on coral communities. The susceptibility of *A. palmata* to disease and predation, combined with the reproductive strategies of this species, exacerbate the impact of declining populations on the stability of Caribbean coral reef ecosystems. The monumental losses of *A. palmata* in the FKNMS and elsewhere in the Caribbean signify an urgent need for the protection of this keystone species under the Endangered Species Act.

16.4
Potential Sources of the White Pox Pathogen

Potential sources of the white pox disease pathogen include wastewater influent, septic tank effluent, feces of reef fishes, canal water, reef water column, and white pox diseased and apparently healthy *A. palmata*. Accumulating evidence suggests that te health of reef organisms in the FKNMS and elsewhere in the Caribbean is affected by pollution of fecal origin. White pox disease is caused by a fecal enteric bacterium of possible human origin (Patterson et al. 2002) and human sewage markers are concentrated on coral surfaces in nearshore waters of the FKNMS (Lipp et al. 2002). Bacteria associated with human fecal contamination have been found within the microbial mat that causes black band disease of corals (Frias-Lopez et al. 2002). Since 1996, populations of the sewage consuming reef sponge *Cliona delitrix* have increased by a factor of 10 on reefs in the FKNMS (Ward-Paige and Risk 2003) while, concurrently, corals have declined by 37% (Porter et al. 2002).

It is tempting to speculate that poor waste disposal practices may be associated with the declining health of tropical marine invertebrates in the FKNMS. Full-scale sewage treatment plants service the communities of Key West, Key Colony Beach on Marathon, and Ocean Reef Resort on Key Largo. The remainder of the Florida Keys utilizes on-site waste disposal practices including septic systems, injection wells, and illegal cesspools. Keys-wide, there are at least 24,000 septic tanks, 600–700 injection wells, and 5000–10,000 cesspools (Lapointe et al. 1990; Shinn et al. 1994). On-site waste disposal contaminates the waters of the FKNMS with nutrients and microorganisms of human fecal origin (Lapointe et al. 1990; Paul et al. 1995a, b, 1997; Griffin et al. 1999). Fecal bacteria and viruses migrate quickly (0.57–140.9 mph) from the site of disposal through the porous limestone bedrock of the Florida Keys and finally to the marine environment (Paul et al. 2000). The general direction of contaminant flow from land is toward the reef tract (Paul et al. 1995a, 1997, 2000).

16.5
Unresolved Questions and Future Research

Although *S. marcescens* occurs in human sewage, the source of the *S. marcescens* strain that causes white pox disease is uncertain (Patterson et al. 2002). Given the magnitude of *A. palmata* loss in the FKNMS, research is needed to identify the source of this pathogen. A variety of potential sources are presently being screened for biotypes similar to the *Serratia* pathogen. These potential sources include wastewater influent, septic tank effluent, feces of reef fishes, seabird guano, canal water, reef water column, and white pox diseased and apparently healthy *A. palmata*. Coral reef managers and wastewater treatment engineers require certainty of the human origin of the white pox pathogen in order to recommend expensive sewage treatment upgrades in Florida and around the Caribbean.

Research is continuing to determine mechanisms of pathogenesis of *S. marcescens* against *A. palmata*. Future research will identify virulence genes in the coral pathogen and prevalence of these genes in environmental isolates of *S. marcescens*. In addition, future studies will reveal the host range of the Serratia pathogen. It is possible that this Serratia isolate does not exclusively affect *A. palmata*, but instead affects other coral species on which disease signs are manifested differently. Since the etiologies of the majority of the coral disease conditions described to date are unknown (Richardson 1998), the extent of the pathogenicity of the white pox pathogen warrants examination.

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