WITHERING SYNDROME AND RESTORATION OF SOUTHERN CALIFORNIA ABALONE POPULATIONS

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ABSTRACT

Withering syndrome is a chronic wasting disease of California abalone (*Haliotis* spp.) first observed in the Channel Islands in the mid-1980s. This fatal disease played a significant role in the demise of southern California black abalone and may also be contributing to the lack of recovery of other species following their severe depletion by overfishing. The causative agent of withering syndrome is an intracellular Rickettsiales-like prokaryote that infects gastrointestinal epithelia. The entire southern California region is considered endemic for this pathogen, and therefore all abalone restoration plans for this area need to consider the implications of its presence. Here we review the current state of knowledge regarding withering syndrome and discuss its potential impact on species recovery programs.

INTRODUCTION

Before the arrival of Europeans, large abalone in southern California were preyed upon primarily by aboriginal peoples and sea otters. During the 1800s the aboriginal peoples were relocated from the Channel Islands and the sea otters were hunted to local extinction. This resulted in higher local population densities of black abalone (*Haliotis cracherodii*) and probably other species than previously experienced in recent history (Davis et al. 1992). Throughout the mid- to late-1900s various abalone species were serially depleted by commercial harvesting (Karpov et al. 2000). Withering syndrome first appeared in the mid-1980s at the Channel Islands in high-density populations of black abalone. Here we review the current state of knowledge regarding withering syndrome and the implications of this disease for abalone recovery programs.

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WITHERING SYNDROME

In the mid-1980s large numbers of dying black abalone with severe pedal atrophy and the empty shells of recently deceased black abalone were observed in populations along the central Channel Islands. The term *withering syndrome* (WS) was coined to describe the phenomenon (Haaker et al. 1992). The disease spread throughout the Channel Islands (Lafferty and Kuris 1993; VanBlaricom et al. 1993) and to the mainland (Steinbeck et al. 1992; Alstatt et al. 1996) throughout the early 1990s. Although overfishing had already greatly reduced many black abalone populations in southern California, WS nearly eliminated the remaining populations throughout the Channel Islands and off central California (Haaker et al. 1992; Richards and Davis 1993; Alstatt et al. 1996). Overfishing was responsible for population declines of pink (*H. corrugata*), green (*H. fulgens*), and red abalone (*H. rufescens*) in southern California prior to the appearance of WS (Karpov et al. 2000). WS signs have been observed in all three species (Pete Haaker, pers. comm.), and the impact of WS on the natural recovery of their populations remains unclear. Overfishing had also depleted populations of white abalone (*H. sorenseni*) before the appearance of WS (Davis et al. 1998). In 2001 this species became the first marine invertebrate to be listed under the federal Endangered Species Act (Federal Register 66 (103), 29046-29055, 29 May 2001). Although two shrunken white abalone were collected from Santa Catalina Island in 1993 (cited in Hobday et al. 2001), and 20 freshly dead, undamaged shells were collected from Farnsworth Bank in 1990 (Tegner et al. 1996), shrunken animals can occur for reasons other than WS. Further studies are needed to determine the susceptibility of white abalone to WS. During the 1997–98 El Niño, red abalone farms from Mexico to central California experienced high mortality rates, and animals showed signs of WS (Moore et al. 2000).

THE ETIOLOGIC AGENT OF WS

During the late 1980s to early 1990s three potential causes of WS were proposed: pollution, starvation (reduced drift algae supply in association with the severe 1982–83 El Niño), and an infectious agent (Davis 1993). Lafferty and Kuris (1993) integrated data sets on WS presence in black abalone, black abalone mortality rates, temperature, kelp abundance, urchin abundance, distance to ports, and current patterns. They concluded that an infectious agent must be responsible for the disease and that elevated temperature, though not a sole cause, resulted in accelerated mortality rates. A coccidian parasite of the kidney (Friedman et al. 1995) was examined as a potential cause but was found to be unassociated with signs of WS (Friedman et al. 1993). Gardner et al. (1995) reported that a Rickettsiales-like prokaryote (WS-RLP) infecting gastrointestinal tissue was present in black abalone from a WS-affected (Channel Islands) population and absent in black abalone from an unaffected population (Ano Nuevo Island, north of Santa Cruz), and that infection intensity was correlated with severity of disease signs. In studies with laboratory-held black abalone, Friedman et al. (1997) reported complex relationships between WS signs, WS-RLP intensity, temperature, and food supply, with some evidence for a role of WS-RLP. In a subsequent study, Friedman et al. (in press) verified that WS-RLP presence is required for WS expression in black abalone, suggesting its etiologic role in this species. Correlation between disease signs and WS-RLP intensity were observed in farmed red abalone during the 1997–98 El Niño (Moore et al. 2000). A series of studies directly implicating WS-RLP in red abalone with WS was reported by Moore et al. (2001). Clinical WS signs and WS-related mortality were seen only in animals with severe WS-RLP infections whereas WS-RLP-free animals remained healthy. Data and observations on pathogenesis, ultrastructure (Friedman et al. 2000), and DNA-based detection (Andree et al. 2000; Antonio et al. 2000) indicate that the bacterium infecting red and black abalone hosts is a single species. WS-RLP infections have been detected in pink and green abalone (CDFG, unpubl. observ.), whereas the susceptibility of white, flat, and pinto abalone remains unknown. The region from central California to northern Mexico can be considered endemic for WS-RLP. Therefore, any abalone restoration activities in southern California need to address the potential impact of this disease.

PHYLOGENY OF WS-RLP

Friedman et al. (2000) named WS-RLP "*Candidatus* Xenohaliotis californiensis" and described its phylogenetic placement within the order Rickettsiales based on morphological characteristics and the DNA sequence of its 16sRNA gene. The term "*Candidatus*" in the taxon indicates that the species was described largely on morphological and DNA sequence-based data and that the serological and biochemical analyses necessary for a complete description is lacking (Murray and Stackebrandt 1995). The order Rickettsiales comprises a large and diverse group of obligate intracellular, gram-negative bacteria, often pathogenic for one or more host species while exhibiting benign infections in closely related species. This group contains the agents of Rocky Mountain Spotted Fever, scrub typhus, Q fever, and typhus in humans, a wide variety of diseases in other mammals (Krieg and Holt 1984), piscirickettsiosis in salmon (Fryer and Lannan 1994), stained prawn disease (Bower et al. 1996), necrotizing hepatopancreatitis in shrimp (Brock et al. 1986), and a large number of poorly described pathogens in many aquatic animal taxa (Sparks 1985). Rickettsiales-like prokaryotes are common in branchial and digestive epithelia of marine molluscs where they often appear benign (Elston 1986; Fryer and Lannon 1994; Sparks 1985).

Nearly all Rickettsiales-like prokaryotes with terrestrial hosts require an arthropod vector (e.g., mosquitoes, fleas, lice, ticks) for spread between individuals. This characteristic is related to their intracellular location and to the inability of most to survive even transient desiccation. Our studies with WS-RLP in black and red abalone suggest that no arthropod vector is required—that is, direct transmission occurs between individuals (Moore et al. 2001; Friedman et al., in press). This finding is in agreement with findings on other marine Rickettsiales-like prokaryotes (e.g., *Piscirickettsia salmonis* of salmon [Cvitanich et al. 1991] and two that infect shrimp [Brock et al. 1986; Bower et al. 1996]). WS-RLP infects gastrointestinal epithelium. WS-RLP inclusions, each containing thousands of individual bacteria, are readily observed bursting into the lumen of the gastrointestinal tract. As with most gastrointestinal pathogens the spread of WS-RLP between individuals is likely fecal-oral.

ORIGIN AND POTENTIAL RESERVOIRS OF WS-RLP

The devastating impact of WS-RLP on black abalone populations suggests that it is a nonnative pathogen. Although we lack historical data to determine whether WS-RLP was present in Channel Island abalone populations before the arrival of WS, we have documented its sudden appearance off the central California coast. Until the mid-1990s, high population densities of black abalone at Vandenberg Air Force Base, resulting from restricted access and the absence of sea otters, were similar to population densities at the Channel Islands prior to the appearance of WS. WS-RLP was absent from this population until approximately 1994, when the first

observations of WS were made (Friedman, unpubl. obs.; Alstatt et al. 1996). Abalone are taxonomically placed within the family Haliotidae, which is in the order Archeogastropoda of class Gastropoda, phylum Mollusca. Intracellular bacterial pathogens such as those in the order Rickettsiales do not have extremely broad host ranges, and the original host species of WS-RLP is likely another haliotid, archeogastropod, or gastropod mollusc. It is possible that nonhaliotid gastropods in southern California are susceptible to infection and could act as reservoirs for the pathogen even in the absence of abalone. This could have important implications for abalone species recovery programs. We have examined a limited number of limpets and other gastropods after cohabitation with infected abalone and have not detected WS-RLP, although further studies are needed to conclude a lack of susceptibility of nonhaliotid gastropods.

FACTORS AFFECTING DISEASE EXPRESSION

The key factors governing WS expression in abalone are (1) the presence of WS-RLP, (2) host species, and (3) temperature, clearly the most important environmental factor. Among local black abalone populations, animals with WS signs were seen exclusively within the heated seawater discharge plume of the Diablo Canyon Power Plant, where temperatures measured up to 11°C above ambient (Steinbeck et al. 1992). Mortality was positively correlated with temperature among Channel Islands black abalone populations experiencing WS (Lafferty and Kuris 1993). Elevated mortality and lower relative weights occurred during El Niño events in black abalone populations at Santa Cruz Island (Tissot 1995). Friedman et al. (1997) reported that elevated water temperature accelerated the mortality rate in laboratory-held black abalone, but animals in colder water also eventually succumbed. The red abalone culture industry has provided insight into the dynamics of WS in this species. Southern and central California red abalone farms typically experience some degree of WS in the summer or fall. Severity is associated with water temperature; the 1997–98 El Niño with temperatures above 23°C was particularly devastating. In a 220-day experiment, red abalone held at 18.5°C had higher mortality, more WS signs, and higher WS-RLP burdens than those held at 14.7°C (Moore et al. 2000). Although low temperature clearly provides a thermal refuge from the pathogenicity of WS-RLP infection in red abalone during the first several years of life, the effect of low-level, chronic infection over a multidecade potential life span remains undetermined. Temperature fluctuations due to seasonality, El Niño/La Niña events, and potential global warming make the long-term impact of WS in red abalone difficult to assess. Temperature likely plays a significant role in WS expression in pink and green abalone, though these species have yet to be studied. The true success of future southern California abalone restoration activities can only be gauged after populations have experienced the broad range of environmental temperatures that occur over multiyear or multidecade cycles. We have detected WS-RLP in experimentally exposed red abalone as small as 3 mm shell length (J. Moore and C. Finley, unpubl. obs.), but further research is needed to determine the life stages at which different species of abalone are susceptible to WS-RLP infection and clinical expression of WS.

POTENTIAL DEVELOPMENT OF RESISTANCE

Black abalone have nearly disappeared from southern California. Since WS-RLP is now endemic throughout southern California, restoration of black abalone (and probably other species) will depend upon natural or hatchery-assisted development of resistant stocks. When a severe selection pressure, such as epidemic disease, sweeps through a population, genetic diversity between individuals can result in survival of a proportion that are "resistant." This previously indistinguishable subset can become the founder population if sufficient numbers of survivors with sufficient genetic diversity remain. The resistance is typically graded rather than absolute—for example, some individuals may survive only slightly longer than average before succumbing to a disease, and others may be able to reproduce before doing so. The disease caused by *Bonamia ostreae*, a protozoan that parasitizes hemocytes in European oysters, *Ostrea edulis*, provides an example. The disease, bonamiasis, was first described in Brittany in 1979 (Comps et al. 1980), the pathogen apparently arriving via the introduction of infected oyster seed from California (Elston et al. 1987). Bonamiasis caused catastrophic declines in native oyster populations throughout most of Europe, reaching Ireland in the 1980s. Culloty et al. (2001) challenged two naive Irish oyster strains and one selectively bred from survivors that had been exposed to *B. ostreae* since the 1980s. In laboratory and field trials, the selected strain showed lower pathogen prevalence, infection intensity, and mortality compared to the naive strains. Similarly, some selective development of resistance to infectious agents of *Crassostrea virginica* has been detected (e.g., Ford 1988; Davis and Barber 1999). Bower et al. (1999) reported on laboratory and field trials investigating the virulence of the protozoan parasite *Perkinsus qugwadi* to nonnative Japanese scallops *Patinopecten yessoensis* that were either naive (with no history of exposure) or progeny of survivors of a *P. qugwadi* epidemic. The latter group of animals had a lower prevalence and lower intensities of infection after challenge by injection or following deployment in a *P. qugwadi* endemic region. The resistant strain tended to have more intense hemocyte responses

to infection, suggesting that resistance was associated with immune system function.

It is important to note that nearly all work concerning development of disease resistance in molluscs has focused on bivalves and the enhancement of managed populations for aquaculture, not on restoring wild populations. In fact, despite the observations above, most experience with oyster disease paints a relatively grim picture with respect to recovery of natural populations via the development of resistance. Two protozoan parasites, *Haplosporidium nelsoni* and *Perkinsus marinus*, remain virulent pathogens severely impacting Atlantic *Crassostrea virginica* populations decades following initial mortality events, and no European populations of *Ostrea edulis* have recovered to levels approaching those before the introduction of bonamiasis, although other pathogens have also contributed. As a result of these experiences, the deployment of a naturally resistant nonnative oyster species is being considered to boost the Atlantic oyster culture industry (Calvo et al. 2000).

There is anecdotal evidence for potential WS-RLP resistance in black abalone. WS was first observed in the Vandenberg Air Force Base population in 1994 (Alstatt et al. 1996). During the 1997–98 El Niño, a large mortality event occurred with up to 80% of individuals exhibiting signs of WS (CDFG, unpubl. obs.). By 1999 densities were less than 1% of the pre-epidemic levels, but those that remained appeared healthy. Animal densities may be too low for this population to recover on its own, and hatchery-based production may be necessary to determine whether these abalone harbor genetically based resistance to WS-RLP. It is important to note that the heritability of this resistance should not be assumed; micro-geographic conditions, food availability, and any number of other nonheritable factors may contribute to realized disease resistance.

SPECIFIC RESTORATION APPROACHES IN SOUTHERN CALIFORNIA

Restoration activities being considered for the recovery of California abalone populations include (1) establishing (or continuing the existence of) protected areas where populations could recover on their own; (2) aggregation of animals to create densities necessary for successful fertilization; and (3) outplanting of hatchery-reared larvae, juveniles, or adults. For white abalone and southern California black abalone, passive methods alone are unlikely to achieve population recovery. Although aggregation increases the likelihood of WS-RLP transmission between individuals, this approach to restoration may be preferable to taking no action because current population densities of white abalone and southern California black abalone are too low to achieve successful fertilization. Outplanting activities for any species must consider the effect of WS on the outplanted animals, taking into account the inevitable occurrence of severe El Niño events. Outplanting of abalone into the WS-RLP-endemic region will be a viable option only if animals do not succumb to WS, which is dependent on thermal regime and species susceptibility. For black abalone, outplanting of animals of any age that are not resistant to WS-RLP may be futile. For red, pink, green, and white abalone, the ecology of WS in each species, particularly with respect to thermal modulation, needs to be determined and considered in the restoration planning process. The health status of any farm-reared abalone should be closely assessed before they are considered for restoration activities. Outplanting of WS-RLP-infected individuals of any species should be carefully considered even if the animals appear healthy. Fortunately, recently developed oral and injection treatments with oxytetracycline have been shown to be very effective against WS-RLP (Friedman et al. 2000) and could be used to eliminate the pathogen from broodstock. While outplanting efforts will always carry some degree of infectious disease risk, this can be minimized by applying knowledge gained about interactions between hosts and potential pathogens.

KEY RESEARCH NEEDS

White Abalone

White abalone is the first marine invertebrate to be listed as endangered under the federal Endangered Species Act. Studies are needed to determine the susceptibility of this species to WS-RLP infection and the role of temperature in WS progression. This information is essential for devising a rational recovery plan.

Pink and Green Abalone

As with white abalone, the relationship between temperature and WS progression in pink and green abalone needs to be determined in order to guide the design of recovery plans for these species.

Nonlethal Diagnostic Methods

To minimize further loss while monitoring natural populations, we need to develop nonlethal diagnostic methods for detecting WS-RLP. These would also be useful for monitoring the WS-RLP status of captive broodstock and their progeny in abalone culture facilities. A polymerase chain reaction-based detection assay (Andree et al. 2000) could provide a foundation for such methods.

Identification and Production of Resistant Stocks

For black abalone and perhaps other species, identification and production of WS-RLP-resistant stocks may

offer the only hope for population recovery in southern California. Resistance should be investigated among survivors of WS epidemics.

Variation Among WS-RLP Isolates from Different Regions or Host Species

WS is currently being managed under the assumption that the pathogen is homogeneous throughout its range. This could be investigated through the development of a polymerase chain reaction-based method of detecting nucleic acid variation and periodic sampling within the endemic region.

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