Prominent Emerging Diseases within the United States

Rocco C. Cipriano[1], Paul R. Bowser[2], Alistair Dove[3], Andy Goodwin[4], and Corey Puzach[5]

[1]U.S. Geological Survey, Leetown Science Center, National Fish Health Research Laboratory, 11649 Leetown Road, Kearneysville, West Virginia 25430; [2]Aquatic Animal Health Program, Department of Microbiology and Immunology, College of Veterinary Medicine, Cornell University, Ithaca, New York 14853; [3]Georgia Aquarium Inc., 225 Baker Street, Atlanta Georgia 30313; [4]University of Arkansas at Pine Bluff, Aquaculture/Fisheries Center, 1200 N. University Drive, P.O. Box 4912, Pine Bluff, Arkansas 71601; [5]U.S. Fish and Wildlife Service, La Crosse Fish Health Center, 555 Lester Avenue, Onalaska, Wisconsin 54650

Abstract

This manuscript reviews disease syndromes that have become significant aquatic animal health issues within the United States since 2003. The emergence of Viral Hemorrhagic Septicemia (VHS) disease among wild fish in the Great Lakes is probably the most problematic and political issue. The emergence of this pathogen resulted in the issuance of a 2006 VHSV Federal order that placed restrictions on the movement of certain species of fish in the eight states that border the Great Lakes (New York, Pennsylvania, Ohio, Indiana, Illinois, Michigan, and Wisconsin and Minnesota) as well as the movement of live fish into the United States from the Ontario and Quebec Provinces, Canada. Spring Viremia of Carp (SVC) was identified for the first times in the United States during 2002. It was diagnosed as the source of mortality among koi at a private facility in North Carolina as well as from feral carp in Cedar Lake (WI). In 2004, Koi Herpesvirus (KHV) killed 8,000 adult common carp (*Cyprinus carpio*) in the Chadakoin River (NY); it reoccurred the next year within Chautauqua Lake (NY), killing an estimated 25,000 carp (20–30 lbs. apiece). During the summers of 2007 and 2008, KHV epizootics also occurred among carp in Ontario (Canada). Finally, outbreaks of epizootic shell disease in American lobster (*Homarus americanus*) have generated concern along the southern New England coast and eastern Long Island Sound. The prevalence and severity of shell disease have increased within inshore areas of southern New England and resulted in significant decreases in lobster catches and marketability.

Key words: emerging disease, United States, Viral Hemorrhagic Septicemia, Spring Viremia of Carp, Koi Herpesvirus, epizootic shell disease

Introduction

Objectives of the bilateral symposia between the United States and Russia are designed to facilitate awareness and collaborations among scientists from both countries by allowing invited participants to review their specific areas of expertise that are relevant to national

trends of aquatic animal population health and disease. Specifically, the proceedings of each symposium are intended to provide a historical synopsis and geographic review of the priority health and disease issues affecting fish and aquatic animals within specific inland, coastal, and marine regions of the United States and Russia. It is also intended to identify priority and emergent health and disease issues that would benefit from collaborative research efforts and develop proposals to successful research such problems/issues. The first conference was convened at the Laboratory of Ichthyopathology in Rybnoe from 12-19 July 1998. The second conference was hosted at the National Fish Health Research Laboratory in Kearneysville, West Virginia during September 21-28, 2003. This presentation reviews disease syndromes that have arisen within the United States since that time.

Three viral pathogens that have emerged within the United States which include Viral Hemorrhagic Septicemia (VHS), Koi Herpesvirus (KHV) and Spring Viremia of Carp (SVC). Viral Hemorrhagic Septicemia (VHS) has been the most problematic and also the most political aquatic animal disease issue within the United States during the past five years. The SVC virus was confirmed from a koi/carp (*Cyprinus carpio*) near the North Carolina and Virginia border in 2002, shortly before the second bilateral conference was convened. It has since become a significant problem with additional reports increasing in regularity within the United States. In 2004, Koi Herpesvirus (KHV) caused great concern when it was initially reported as the cause of a massive kill among common carp in New York state. In addition to the aforementioned viruses, outbreaks of epizootic shell disease in American lobster (*Homarus americanus*) have generated concern along the southern New England coast and eastern Long Island Sound. The prevalence and severity of shell disease has increased since the mid 1990's and resulted in significant decreases in catch and marketability.

Viral Hemorrhagic Septicemia

Viral Hemorrhagic Septicemia is caused by an enveloped, bullet-shaped rhabdovirus (approximately 180 nm x 60 nm) comprised of five structural proteins encoded by five distinct genes located on a single segment of single-stranded RNA (McAllister 1979, de Kinkelin 1983). Affected fish may present with clinical signs that include anemia, exophthalmia, lethargy, darkened coloration, and hemorrhage of the eyes, skin, gills and fins (Wolf 1988). Internally, petechiae may be present in the peritoneum, swimbladder, visceral musculature and fat. The liver may appear mottled, the spleen is often enlarged, and the kidneys are

generally a pronounced reddish coloration (Wolf 1988). The disease is common in Europe where it is principally expressed as an acute to chronic infection among cultured rainbow trout (*Oncorhynchus mykiss*) when water temperatures are near or below 10^{o}C (Wolf 1988). More recently, the virus has been associated with marine fishes from the northern Atlantic and Pacific Oceans (Takano et al. 2000, Dopazo et al. 2002, King et al. 2001, Hedrick et al. 2003) and four genotypes have been identified (Snow et al. 1999, 2004, Einer-Jensen et al. 2004, 2005). Strains of VHS isolated from North America (genotype IV) are different from the first three genotypes of European origin (Benmansour et al. 1997, Stone et al. 1997, Einer-Jensen et al. 2004).

In the late 1980's, VHS virus was first detected in the United States, among clinically normal chinook salmon (*O. tshawytscha*) and coho salmon (*O. kisutch*) that had returned from the Pacific Ocean on spawning migrations to rivers in Washington state (Brunson et al. 1989, Hopper 1989). Subsequent North American isolations were predominantly made from herring (*Clupea harengus*) and cod (*Gadus macrocephalus*) in the Pacific and Atlantic Oceans and most of these strains had low pathogenicity for salmonids (Meyers and Winton 1995).

In 2005, VHS was the cause of mortality among muskellunge (*Esox masquinongy)* within the northwest portion of Lake St. Clair, Michigan. Affected fish exhibited congestion of internal organs associated with hemorrhages in the inner wall of the swim bladder. Sequence analysis revealed that the isolates were a distinct sublineage of the North American genotype IV (Elsayed et al. 2006). Elsayed et al. (2006) proposed that the newly emergent Great Lakes isolate of VHSV be referred to as VHSV genotype IVb and also suggested that all previous genotype IV isolates from the marine environment be referred to as VHSV genotype IVa. Elsayed et al. (2006) also reported virus suspect fish samples collected in 2003, which when assayed in 2005 also contained VHSV IVb. In 2005, another VHS-mortality event occurred in the Bay of Quinte, Lake Ontario, Canada that involved approximately 100 metric tons of freshwater drum (*Aplodinotus grunniens*), round goby (*Neogobius melanostomus*) and muskellunge (Lumsden et al. 2007). Similar to what Elsayed et al. (2006) had reported, the virus isolated from this latter event belonged to genotype IVb. In May 2006, mortality was reported among round gobies from the St. Lawrence River and Lake Ontario, which were also caused by the VHS IVb (Groocock et al. 2007a). In October 2006, emergence

Table 1: List of Species Regulated by the Viral Hemorrhagic Septicemia Virus interim rule (list effective to 9 September 2008, from Bowser 2009)

Family Centrarchidae
Black Crappie, *Pomoxis nitromaculatus*
Bluegill, *Lepomis macrochirus*
Largemouth Bass, *Micropterus salmoides*
Pumpkinseed, *Lepomis gibbosus*
Rock Bass, *Ambloplites ruprestris*
Smallmouth Bass, *Micropterus dolomieu*

Family Cyprinidae
Bluntnose Minnnow, *Pimephales notatus*
Emerald Shiner, *Notropis atherinoides*
Spottail Shiner, *Notropis hudsonius*
Shorthead Redhorse, *Moxostoma macrolepidotum*
Silver Redhorse, *Moxostoma anisurum*

Family Ictaluridae
Brown Bullhead, *Amieurus nebulosus*
Channel Catfish, *Ictalurus punctatus*

Family Esocidae
Muskellunge, *Esox masquinongy*
Northern Pike *Esox niger*

Family Percidae
Walleye, *Sander vitreus*
Yellow Perch, *Perca flavescens*

Family Salmonidae
Brown Trout, *Salmo trutta*
Chinook Salmon, *Oncorhynchus tshawytscha*
Lake Whitefish, Coregonus *clupeaformis*
Rainbow Trout, *Oncorhynchus mykiss*

Family Gadidae
Burbot, *Lota lota*

Family Scianidae
Freshwater Drum, *Aplodinotus grunniens*

Family Gobiidae
Round Goby, *Neogobius melanostomus*

Family Clupeidae
Gizzard Shad, *Dorosoma cepedianum*

Family Moronidae
White Bass, *Morone chrysops*
White Perch, *Morone americana*

Family Percopsidae
Trout-Perch, *Percopsis omiscomacys*

of this pathogen resulted in the issuance of a VHSV Federal order by the Animal and Plant Inspection Service of the United States Department of Agriculture (APHIS USDA) that placed restrictions on the movement of certain species of fish in the eight states bordering on the Great Lakes (New York, Ohio, Indiana, Illinois, Pennsylvania, Michigan, and Wisconsin) as well as the movement of live fish into the United States from the Canadian Provinces of Ontario and Quebec. Further survey of multiple bodies of water across New York State indicated that VHS virus was widespread in a diverse number of fishes from each of the Great Lakes of New York State as well as from one inland location, Lake Conesus (Groocock et al. 2007b). As a reportable pathogen, there are very specific diagnostic protocols which must be followed to

document that a new fish species has been infected with VHSV or that an infected fish is found in body of water in which the infection was not previously known (OIE 2006). Currently, VHSV genotype IVb has been documented in the Great Lakes Basin of North America from 28 species of fish (Table 1).

Spring Viremia of Carp (SVC)

Spring Viremia of Carp (SVC), caused by the bullet-shaped *Rhabdovirus carpio* (Ahne et al. 2002), was first described in Yugoslavia by Fijan et al. (1971). Prior to 2002, SVC was a disease that commonly affected carp and other cyprinids within Europe, the Middle East, Russia, and the former territories of the Soviet Union (Fijan 1999). Phylogenetic analysis of the partial G gene sequence identified four distinct subgroups; isolates from Western Europe formed one sub-group, those from Russia and the former Soviet states formed another two subgroups, and Asian isolates formed a fourth group (Stone et al. 2003). Like many other diseases, the clinical signs of SVC are non-specific, but may include exophthalmia, darkened coloration, anemia, ascites, and hemorrhage in the gills, skin, and eyes. Internally, affected fish may present with edema and inflammation with petechial hemorrhages in many organs, especially characteristic of the swim bladder (Fijan et al. 1971, Fijan 1999).

In 2002, SVC was first reported from North America as a result of mortality among koi at a private production facility in North Carolina (Goodwin 2003). Affected fish displayed severe ascites, petechiae in the skin, and SVCV was detected by cell culture and immunocytochemistry with an anti-SVCV antibody. Final confirmation was made by the Office International des Epizooties (OIE) SVCV reference laboratory (Centre for Environment, Fisheries, and Aquaculture Science, Weymouth, England).

Also in 2002, SVC was determined to cause a mortality-event involving more than 1,500 dead carp in Cedar Lake (Wisconsin). Affected fish displayed clinical signs of SVC including petechial and ecchymotic skin hemorrhages, ascites, and edema of the kidney and spleen (Dikkeboom et al. 2004). A virus was isolated in the fat head minnow (FHM) cell line and the isolate was confirmed to be SVC by the CEFAS/OIE Reference Laboratory. The isolate displayed a 98.6% nucleotide identity with the isolate from the koi epizootic in North Carolina and was most similar to Asian rather than European strains (Dikkeboom et al. 2004). Because of the kill in Cedar Lake, personnel from the Wisconsin Department of Natural Resources sent archived histological tissue blocks from historic carp kills in Wisconsin to the CEFAS laboratory and tissues from a

1989 carp kill in the Petenwell Flowage (Wisconsin River) tested positive for SVCV. In that instance, the virus was more similar to the European rather than the Asian genotype (Marcquenski et al. 2003).

In response to these isolations, the United States Fish and Wildlife Service (USFWS) initiated surveillance for SVCV in 2003 (Puzach et al. 2009) in conjunction with the National Wild Fish Health Survey and funding was provided from the Animal and Plant Health Inspection Service (APHIS). Almost 7,000 fish representing 20 species were screened at the USFWS Fish Health Center in La Crosse (WI). Fish were sampled from an eight state area including: Minnesota, Wisconsin, Iowa, Missouri, Illinois, Michigan, Ohio, and Montana. The virus was isolated from common carp from the Calumet Sag Channel (IL) in 2003, as well as from carp in Pool 8 of the Upper Mississippi River after reports of lethargic fish were observed near the surface. In 2007, SVCV was isolated from emerald shiners (*Notropis atherinoides*) in the Ohio River (OH) during 2007. Personnel from the USFWS Fish Health Center at Lamar (PA) also isolated SVCV in largemouth bass (*Micropterus salmoides*) and bluegill (*Lepomis macrochirus*) in Ohio. Other isolations of SVCV in fish in North America include a private ornamental carp pond in Washington State, a commercial pond in Missouri and in Hamilton Harbor, Lake Ontario, Hamilton, Ontario, Canada. Despite its detection in the Great Lakes and Mississippi River watersheds, there is very little evidence that SVCV is a significant problem in North America. Since its first detection in 2003, the only major fish kill was the event in Cedar Lake. That kill involved only common carp, a species widely considered as an aquatic nuisance by wildlife agencies. There have been no reports of SVCV in commercial aquaculture since the 2003 and 2004 cases in North Carolina and Missouri and no SVC kills have been reported in the Great Lakes or Mississippi River.

Koi Herpesvirus

Koi herpesvirus (KHV) is a relatively newly recognized disease that affects koi and common carp (Bretzinger et al. 1999, Hedrick et al. 1999, 2000, 2005). The virus is a member of the Family *Herpesviridae* and has an icosahedral inner capsid (100-110 nm diameter). A tegument between the nucleocapsid and viral envelope produces an overall diameter from 170-230 nm (Hedrick et al. 2005).

The disease is characterized as a highly contagious infection with severe mortality at water temperatures between 22° and 27 °C (Hedrick et al. 2000, OATA 2001, Ronen et al. 2003). Affected fish may exhibit necrotic gill filaments with

associated bleeding, sunken eyes, excessive dermal mucous with discolored patches or dermal blisters and, in some cases, heavy secondary bacterial or fungal infections can mask the underlying viral infection (Hedrick et al. 2000, 2005, Goodwin 2003). Internal signs of KHV-infected fish vary and are often non-specific, but organs may be mottled, the kidney and spleen may be enlarged, and there may also be a larger than normal number of adhesions in the body cavity (Hedrick et al. 2000, 2005, Goodwin 2003).

In 2004, a mass mortality event occurred in the Santee-Cooper Reservoir (South Carolina) that involved thousands of common carp (Terhune et al. 2005). Affected fish displayed hemorrhages of their ventral surfaces and severe gill necrosis from which *Flavobacterium columnare* was isolated and determined to be the principal cause of mortality. Although cytopathic effects were not observed in cell cultures, PCR results were positive for KHV among several fish designating the first time that this virus was identified from wild carp in the United States (Terhune et al. 2005).

Also in 2004, approximately 8,000 adult common carp died in the Chadakion River, the river that drains Chautauqua Lake, which was followed in June of 2005 with another mortality event was reported among carp on Chautauqua Lake, New York. (Grimmett et al. 2006). In the latter episode, some 25,000 adult carp weighing an estimated 20 – 30 pounds apiece succumbed to infection over a two-week period. The KHV virus was diagnosed from the affected fish by cell culture and by quantitative PCR. The etiology of KHV was subsequently confirmed by the USDA-APHIS, National Veterinary Services Laboratories in Ames, Iowa (Grimmett et al. 2006). Other unpublished reports have also identified KHV to have caused epizootics among carp from Wolf Lake (Indiana) in 2005, as well as from Lake Mohave and Lake Havasu in Arizona during June, 2009. Though not in the United States, additional KHV epizootics have occurred among carp within the Kawartha district in Ontario, Canada during summers of 2007 and 2008 as well as in 2008 in the Lundar Beach area of Lake Manitoba (Al-Hussinee et al. 2009). In each of these Canadian cases, KHV was detected by nested PCR and the viral load was estimated by quantitative PCR.

Epizootic Shell Disease

Serious outbreaks of epizootic shell disease have affected the American lobster (*Homarus americanus*) within southern New England. Hess (1937) was the first to describe shell disease among lobsters from tidal impoundments, but that condition was somewhat different than what

has been observed in the current situation. In southern New England, the prevalence and severity of the shell disease during the mid-1990's was relatively low (0% to 5.6%), but reached epizootic proportions and exceeded 20% in near shore coastal areas from southern Massachusetts to eastern Long Island Sound by the year 2000 (Cobb and Castro 2006). Glenn and Pugh (2006) reported that the disease remained at relatively constant levels among lobsters from the Gulf of Maine and outer regions of Cape Cod. By 2001, some of the most affected populations were reported along inshore areas of Rhode Island (42.0%), eastern Long Island Sound (22.7%) and Buzzards Bay (11.6%; Castro & Angell 2000, CT-DEP1999, Estrella 1991, Cobb and Castro 2006). At Buzzards Bay, a significant correlation was noted between disease incidence and a series of warmer than average water temperatures from 1999-2003, which suggested that temperature may affect the severity of the disease (Glenn and Pugh 2006). Effects of temperature were also evident in Long Island Sound where lobsters displayed a non-fatal calcinosis stimulated by above normal seawater temperatures in 2002. The increased temperatures were thought to have induced changes in hemolymph pH and caused formation of calcium deposits in gills and antennal glands (Dove et al. 2004).

Females are more likely to exhibit disease than males and larger lobsters were more likely to be affected (Estrella 1991, Glenn and Pugh 2006). The carapace is generally affected and may appear to be pitted or, in extreme cases, have many irregular deep erosions with extensive melanization (Smolowitz et al. 2005, Cobb and Castro 2006). Bacteria invade the carapace from the surface of the shell initiating an inflammatory response evidenced by hemocyte infiltration at the erosive lesions (Smolowitz et al. 2005). Fewer bacteria are found on the carapaces of healthy as compared to diseased lobsters (Hsu and Smolowitz 2003), but the microbial flora are similar and suggests invasion by an opportunistic pathogen (Chistoserdov et al. 2005, Sullivan and Nelson 2005). However, members of the F*lavobacteriaceae* were extremely predominant within the affected areas of the shell (Chistoserdov et al. 2005). In a more recent study, Quinn et al. (2009) found bacteria in the lesions of shell diseased lobsters that included the Bacteroidetes, Gamma-proteobacteria and Alphaproteobacteria. Members of the latter two classes were determined to be transient opportunists, but one member of the Bacteriodetes, which was tentatively referred to as *Aquamarina 'homaria,'* was present in all shell disease lesions. Pending further delineation of bacterial etiology, the increased prevalence of shell disease has been speculated to

result from changes in susceptibility of lobsters to one or more species in the bacterial flora that may be enhanced by fluctuations within environmental parameters (Tlusty et al. 2007).

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