

Marine Science Review – 231

Pathogens, disease and die-offs

In this review:

- A. Recent articles – no abstract available
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A. Recent articles – no abstract available

Forman, D., West, N., Powell, M., Francis, J., and Guy, E. ***Toxoplasma* in cetaceans around the British Isles.** *Veterinary Record* 161(8): 279, 2007.

Nugues, M. M. and Bak, R.P.M. **Dark spot syndrome: recent or old?** *Coral Reefs* 26(2): 359, 2007.

Aeby, G.S. **First record of coralline lethal orange disease (CLOD) in the Northwestern Hawaiian Islands.** *Coral Reefs* 26(2): 385, 2007.

Valentine, K.H., Harms, C.A., Cadenas, M.B., Birkenheuer, A.J., Marr, H.S., Braun-McNeill, J., Maggi, R.G., and Breitschwerdt, E.B. ***Bartonella* DNA in loggerhead sea turtles.** *Emerging Infectious Diseases* 13(6): 949-950, 2007.

B. Recent articles with abstracts

LaDeau, S.L., Kilpatrick, A.M., and Marra, P.P. **West Nile virus emergence and large-scale declines of North American bird populations.** *Nature* 447(7145): 710-713, 2007.

Notes: Emerging infectious diseases present a formidable challenge to the conservation of native species in the twenty-first century. Diseases caused by introduced pathogens have had large impacts on species abundances, including the American chestnut, Hawaiian bird species and many amphibians. Changes in host population sizes can lead to marked shifts in community composition and ecosystem functioning. However, identifying the impacts of an introduced disease and distinguishing it from other forces that influence population dynamics (for example, climate) is challenging and requires abundance data that extend before and after the introduction. Here we use 26 yr of Breeding Bird Survey (BBS) data to determine the impact of West Nile virus (WNV) on 20 potential avian hosts across North America. We demonstrate significant changes in population trajectories for seven species from four families that concur with a priori predictions and the spatio-temporal intensity of pathogen transmission. The American crow population declined by up to 45% since WNV arrival, and only two of the seven species with documented impact recovered to pre-WNV levels by 2005. Our findings demonstrate the potential impacts of an invasive species on a diverse faunal assemblage across broad geographical scales, and underscore the complexity of subsequent community response.

Richardson, L.L., Sekar, R., Myers, J.L., Gantar, M., Voss, J.D., Kaczmarek, L., Remily, E.R., Boyer, G.L., and Zimba, P.V. **The presence of the cyanobacterial toxin microcystin in black band disease of corals.** *FEMS Microbiology Letters* 272(2): 182-187, 2007.

Notes: Black band disease (BBD) is a migrating, cyanobacterial dominated, sulfide-rich microbial mat that moves across coral colonies lysing coral tissue. While it is known that BBD sulfate-reducing bacteria contribute to BBD pathogenicity by

production of sulfide, additional mechanisms of toxicity may be involved. Using HPLC/MS, the cyanotoxin microcystin was detected in 22 field samples of BBD collected from five coral species on nine reefs of the wider Caribbean (Florida Keys and Bahamas). Two cyanobacterial cultures isolated from BBD, *Geitlerinema* and *Leptolyngbya* sp. contained microcystin based on HPLC/MS, with toxic activity confirmed using the protein phosphatase inhibition assay. The gene *mcyA* from the microcystin synthesis complex was detected in two field samples and from both BBD cyanobacterial cultures. Microcystin was not detected in six BBD samples from a different area of the Caribbean (St Croix, USVI) and the Philippines, suggesting regional specificity for BBD microcystin. This is the first report of the presence of microcystin in a coral disease.

Van Dover, C.L., Ward, M.E., Scott, J.L., Underdown, J., Anderson, B., Gustafson, C., Whalen, M., and Carnegie, R.B. **A fungal epizootic in mussels at a deep-sea hydrothermal vent.** *Marine Ecology: An Evolutionary Perspective* 28(1): 54-62, 2007.

Notes: Mass mortalities due to disease are important determinants of population and community structure in marine ecosystems, but the speed at which an epizootic may sweep through a population, combined with rapid selection for disease-resistant stocks, can mask the ecological impact of disease in all but the most closely monitored populations. We document an emergent epizootic event in the deep sea that is occurring in mussels (*Bathymodiulus brevior*) at the Mussel Hill hydrothermal vent in Fiji Basin and we identify the causal agent as a black yeast (order Chaetothyriales) that elicits a pronounced host immune response and is associated with tissue deterioration. The yeast was not observed in other invertebrate taxa (the gastropods *Ifremeria nautilei*, *Alviniconcha* aff. *bessleri*; the limpets *Lepetodrilus schrolli*, *Symmetromphalus* aff. *hageni*; the polychaetes *Branchipolynoe pettiboneae*, *Amphisamytha* cf. *galapagensis*) associated with the mussel bed, nor in mussels (*Bathymodiulus brevior*) collected from adjacent Lau Basin mussel beds. Massive mussel mortality resulting from the fungal infection is anticipated at the Mussel Hill site in Fiji Basin; we expect that epizootic outbreaks in dense invertebrate communities have the potential to be major determinants of community structure in deep-sea chemosynthetic ecosystems. The possibility that submersible assets may serve as vectors for transport of the fungus warrants further attention.

Haapkyla, J., Seymour, A.S., Trebilco, J., and Smith, D. **Coral disease prevalence and coral health in the Wakatobi Marine Park, south-east Sulawesi, Indonesia.** *Journal of the Marine Biological Association of the United Kingdom* 87(2): 403-414, 2007.

Notes: This is the first study on coral diseases in the Wakatobi Marine National Park (WMNP), south-east Sulawesi. It aimed to provide baseline knowledge of coral disease prevalence and coral health in this remote region. Results indicate a low disease prevalence of 0.57% with only two known diseases occurring within the sampling unit, white syndrome (0.42%) and tumours (0.15%). They affected 15 taxonomic groups from a total of 32 taxonomic groups. The presence of black-band disease (BBD), skeletal eroding band (SEB) disease and Porites ulcerative white spot disease (PUWSD) was identified outside the study area. A large number of corals were affected by previously undescribed conditions (9.7% of colonies). The impact of lesions named as green spot, green band pigmented spot, and flatworm infestation is not known and represents an important area for future studies.

Kennedy, C.R. **The pathogenic helminth parasites of eels.** *Journal of Fish Diseases* 30(6): 319-334, 2007.

Notes: Although 63 and 55 species of helminths have been reported from each species of Atlantic eel and from 29 to 19 for each species of Pacific eel only the monogeneans *Pseudodactylogyryus bini* and *P. anguillae* and the nematode *Anguillicola crassus*, originally specific to species of Pacific eels, can be considered serious pathogens. None of the three are normally pathogenic to their preferred natural eel host species in the wild. *Pseudodactylogyryus* spp. only cause serious local gill damage when present on a host in large numbers under optimal conditions that facilitate transmission. This is the case in eel aquaculture, where infections can be controlled by drugs. *Anguillicola crassus* is only pathogenic to *Anguilla anguilla* and *A. rostrata* when Atlantic eels are introduced to the far east or when the parasites have been introduced to Europe. Here the parasite life cycle differs in that *A. crassus* can infect a wide range of intermediate hosts, employ paratenic hosts and survive as larvae for months in the swimbladder wall. This makes it an excellent colonizer. Its major pathogenic effects on eels result from haemorrhaging in, and thickening of, the swimbladder wall. It reduces the oxygen concentration in the swimbladder, reducing its ability to function as a hydrostatic organ, and increases the stress response of eels. In shallow lakes at warm temperatures this can result in mass

mortalities. It is also feared that the parasite affects the ability of eels to migrate to the Sargasso Sea and so contributes to the decline in eel populations. Control by drug treatment is possible in culture, but not in the wild.

Boyett, H.V., Bourne, D.G., and Willis, B.L. **Elevated temperature and light enhance progression and spread of black band disease on staghorn corals of the Great Barrier Reef.** *Marine Biology* 151(5): 1711-1720, 2007.

Notes: Rates of progression and transmission of black band disease (BBD) on the staghorn coral, *Acropora muricata*, were compared between months for seasonal in situ studies and between temperature treatments in experimental aquaria manipulations at Lizard Island on the Great Barrier Reef (GBR). In situ field experiments demonstrated that BBD progressed along branches approximately twice as fast (1.7-2.4 times) during the austral summer month of January (0.99 ± 0.04 cm/day) than in the cooler months of July (0.58 ± 0.04 cm/day) and May (0.41 ± 0.07 cm/day). Transmission of BBD between colonies was also accelerated in warmer months, with signs of infection becoming visible 1.2 days earlier in January compared to May. The greater seawater temperatures by ~ 2 to 3°C and light intensities by up to $650 \mu\text{E}/\text{m}^2/\text{s}$ in January, suggest that rates of progression and transmission of BBD are linked to one or both of these parameters. Manipulative experiments in summer provide corroborative evidence that elevated temperatures increase rates of BBD progression, with the disease progressing 1.3 times more rapidly in the 32°C elevated temperature treatment than in the 30°C ambient treatment (1.17 ± 0.06 cm/day versus 0.92 ± 0.07 cm/day; $F_{2,6} = 7.66$, $P = 0.022$). In contrast, although a trend for greatest BBD progression was measured in elevated temperature treatments of 29°C (0.46 ± 0.07 cm/day) and 31°C (0.52 ± 0.06 cm/day) in winter, these rates did not differ significantly ($F_{3,7} = 1.72$, $P = 0.249$) from those measured for the ambient 27°C treatment (0.37 ± 0.06 cm/day) or the field controls (0.41 ± 0.09 cm/day). The lower rates of BBD progression in the 31°C winter treatment (0.52 ± 0.06 cm/day) than in the 30°C (0.92 ± 0.07 cm/day) summer treatment, may have been a response to 28-fold decreased light irradiance in the former, suggesting that high irradiance in combination with elevated temperatures may promote progression of BBD. Results from this study indicate that the impact of elevated temperature on BBD progression is complex with a combination of environmental factors including temperature and light playing key roles in progression and transmission of the disease.

Vuori, K.A.M. and Nikinmaa, M. **M74 syndrome in Baltic salmon and the possible role of oxidative stresses in its development: Present knowledge and perspectives for future studies.** *Ambio* 36(2-3): 168-172, 2007.

Notes: Baltic salmon suffer from maternally transmitted yolk-sac fry mortality syndrome-M74. The incidence of M74 varies considerably on a year to year basis. In the 1990s the mortalities were 50-80% but in 2003-2005, below 10%. Before death, M74-affected fry have several typical symptoms. M74-eggs are characterized by low thiamine and carotenoid content, and affected fry show signs of oxidative stress. Although M74 is associated with thiamine deficiency and the symptoms of the fry can be alleviated with thiamine, the underlying causes of the syndrome have remained a mystery. We have studied the symptoms of M74 at the molecular level by investigating the global gene expression patterns using cDNA microarray and have quantified the changes in transcriptional regulation in M74-affected and healthy yolk-sac fry. Our and previous results suggest that M74 in Baltic salmon yolk-sac fry results from oxidative stresses disturbing several different developmental molecular pathways. Because the M74 syndrome is of maternal origin, factors in the Baltic Sea during salmon feeding and migration, i.e., the chemical composition of food, may be decisive in the development of M74. The possible mechanisms by which oxidative stresses may develop in adult salmon are discussed in the review.

Wohlsein, P., Müller, G., Haas, L., Siebert, U., Harder, T.C., and Baumgärtner, W. **Antigenic characterization of phocine distemper virus causing mass mortality in 2002 and its relationship to other morbilliviruses.** *Archives of Virology* 152(8): 1559-1564, 2007.

Notes: The antigenic relationship between the phocine distemper virus (PDV) strain causing the epidemic in 2002 and the PDV strain of 1988, canine distemper virus from two dogs and one marten, and one measles virus strain was investigated *in vivo* and *in vitro* using monospecific polyclonal and monoclonal antibodies directed against five different proteins of canine or phocine distemper virus (N, P, M, F, H). Epitopic mapping revealed no difference between the PDV strains causing the epidemics in 1988 or 2002. However, the use of these antibodies allowed discrimination between different morbilliviruses

including a vaccine strain of canine distemper virus. The major differences among the investigated morbilliviruses were found in the H protein.

Wheeler, K., Shields, J.D., and Taylor, D.M. **Pathology of *Hematodinium* infections in snow crabs (*Chionoecetes opilio*) from Newfoundland, Canada.** *Journal of Invertebrate Pathology* 95(2): 93-100, 2007.

Notes: Bitter crab disease (BCD) of snow crabs, *Chionoecetes opilio*, is caused by a parasitic dinoflagellate, *Hematodinium* sp. The disease has shown an alarming increase in prevalence in the commercial fishery in eastern and northeastern areas of Newfoundland and Labrador since it was first recorded there in the early 1990s. We documented histopathological alterations to the tissues in snow crabs with heavy infections of *Hematodinium* sp. and during sporulation of the parasite. Pressure necrosis was evident in the spongy connective tissues of the hepatopancreas and the blood vessels in most organs. In heavy infections, little remained of the spongy connective tissues around the hepatopancreas. Damage to the gills varied; in some cases it was severe, particularly during sporulation, involving apparent thinning of the cuticle, loss of epithelial cells, and fusion of the membranous layers of adjacent gill lamellae. Affected lamellae exhibited varying degrees of distention with a loss of trabecular cells, hemocyte infiltrations, and swelling or "clubbing" along the distal margins. Large numbers of zoospores were located along the distal margins of affected lamellae suggesting that sporulation may cause a lysis or bursting of the thin lamellar cuticle, releasing spores. Pressure necrosis, due to the build up of high densities of parasites, was the primary histopathological alteration in most tissues. *Hematodinium* infections in the snow crab are chronic, long-term infections that end in host death, during sporulation of the parasite.

Lesser, M.P., Bythell, J.C., Gates, R.D., Johnstone, R.W., and Hoegh-Guldberg, O. **Are infectious diseases really killing corals? Alternative interpretations of the experimental and ecological data.** *Journal of Experimental Marine Biology and Ecology* 346(1-2): 36-44, 2007.

Notes: Emerging infectious diseases are a worldwide problem and are believed to play a major role in coral reef degradation. The study of coral diseases is difficult but the use of culture-independent molecular techniques has been, and will continue to be, useful in a system where a limited number of visible signs are commonly used to define a "coral disease". We propose that coral "diseases", with rare exception, are opportunistic infections secondary to exposure to physiological stress (e.g. elevated temperature) that result in reduced host resistance and unchecked growth of bacteria normally benign and non-pathogenic. These bacteria are from the environment, the host, or the coral mucus layer and become opportunistic pathogens. While difficult and time consuming, we do not advocate abandoning the study of disease-causing pathogens in corals. However, these studies should include comprehensive efforts to better understand the relationship between coral diseases and environmental changes, largely anthropogenic in nature, occurring on coral reefs around the world. These environmental insults are the cause of the physiological stress that subsequently leads to coral mortality and morbidity by many mechanisms including overwhelming infections by opportunistic pathogens.

Luna, G.M., Biavasco, F., and Danovaro, R. **Bacteria associated with the rapid tissue necrosis of stony corals.** *Environmental Microbiology* 9(7): 1851-1857, 2007.

Notes: The rapid tissue necrosis (RTN) is a common disease of both wild and captive stony corals, which causes a fast tissue degradation (peeling) and death of the colony. Here we report the results of an investigation carried out on the stony coral *Pocillopora damicornis*, affected by an RTN-like disease. Total abundance of prokaryotes in tissue samples, determined by epifluorescence microscopy, was significantly higher in diseased than in healthy corals, as well as bacterial counts on MB2216 agar plates. Further experiments performed by fluorescent in situ hybridization using a 16S rDNA *Vibrio*-specific probe showed that vibrios were significantly more abundant in diseased than in healthy corals. Accordingly, bacterial counts on TCBS agar plates were higher in diseased than in healthy tissues. 16S rDNA sequencing identified as *Vibrio* colonies from diseased tissues only. Cultivated vibrios were dominated by a single ribotype, which displayed 99% of similarity with *Vibrio harveyi* strain LB4. Bacterial ribotype richness, assessed by terminal-restriction fragment length polymorphism analysis of the 16S rDNA, was significantly higher in diseased than in healthy corals. Using an in silico software, we estimated that a single terminal restriction fragment, putatively assigned to a *Vibrio* sp., accounted for > 15% and < 5% of the total bacterial assemblage, in diseased and healthy corals respectively. These results let us hypothesize that the RTN in stony corals can be an

infectious disease associated to the presence of *Vibrio harveyi*. However, further studies are needed to validate the microbial origin of this pathology.

Yarden, O., Ainsworth, T.D., Roff, G., Leggat, W., Fine, M., and Hoegh-Guldberg, O. **Increased prevalence of ubiquitous ascomycetes in an acropoid coral (*Acropora formosa*) exhibiting symptoms of brown band syndrome and skeletal eroding band disease.** *Applied and Environmental Microbiology* 73(8): 2755-2757, 2007.

Notes: The prevalence of coral-associated fungi was four times higher in diseased *Acropora formosa* colonies than in healthy colonies. Since taxonomically related fungal species were isolated from diseased and healthy colonies, we suggest that their association with coral may be constitutive but that their abundance is dependent on coral health.

Fujii, K., Kakumoto, C., Kobayashi, M., Saito, S., Kariya, T., Watanabe, Y., Xuan, X. N., Igarashi, I., and Suzuki, M. **Seroepidemiology of *Toxoplasma gondii* and *Neospora caninum* in seals around Hokkaido, Japan.** *Journal of Veterinary Medical Science* 69(4): 393-398, 2007.

Notes: Serological analysis was performed to detect *Toxoplasma gondii* and *Neospora caninum* infection in seals in Hokkaido. Serum samples were collected from 322 Kuril harbor seals (*Phoca vitulina stejnegeri*) at Nosappu, Akkeshi and Erimo, from 46 spotted seals (*P. largha*) at Nosappu, Erimo, Yagishiri Island, Hamamasu and Syakotan, and from 4 ribbon seals (*P. fasciata*) and a bearded seal (*Erignathus barbatus*) at Nosappu between 1998 and 2006. Recombinant surface antigen of *T. gondii* (SAG2t) and *N. caninum* (NcSAG It) were used as antigens for ELISA to detect antibodies. Antibodies against SAG2t were detected from 4% of 77 Kuril harbor seals at Nosappu in 2005. Antibodies against NcSAG1t were detected from 2% (1/66) in 2003, 5% (4/79) in 2004 and 10% (8/77) in 2005 of Kuril harbor seals and 11% of 9 spotted seals in 2004 sampled at Nosappu. Eight percent of 12 Kuril harbor seals from Akkeshi and 25% of 4 spotted seals from Erimo in 2005 also contained antibodies against NeSAG1t. These suggest sporadic infection of *T. gondii* and *N. caninum* in Kuril harbor seals and spotted seals in Hokkaido. Of the ELISA-positive seals, 2 seals having anti-SAG2t antibodies and 3 seals having anti-NcSAG1t antibodies in 2005 were judged to be juveniles that have no maternal antibodies. These suggest that the protozoan infections have occurred in recent years. Infection of terrestrial protozoa such as *T. gondii* and *N. caninum* in seals indicates that the sea environment has been contaminated with protozoa.

Waller, L.J. and Underhill, L.G. **Management of avian cholera *Pasteurella multocida* outbreaks on Dyer Island, South Africa, 2002-2005.** *African Journal of Marine Science* 29(1): 105-111, 2007.

Notes: In 2002 there was a widespread epizootic involving seabirds on five of the offshore islands of the Western Cape, South Africa. Since then, avian cholera *Pasteurella multocida* outbreaks have been occurring annually on one of these islands, Dyer Island. This paper reports on the three subsequent summers, 2003/04, 2004/05 and 2005/06, during which further avian cholera outbreaks were recorded. It focuses on the outbreak in 2004/05, which was the largest in extent and the most closely monitored. The mortalities during 2005/06 were not as extensive as expected. The management measures used to bring these outbreaks under control are described. Removal of all the carcasses from the entire island in one day is important in reducing mortality. Management intervention is required to reduce the negative impacts of disturbance due to kelp gull *Lasus dominicanus* predation on other breeding seabirds, primarily the African penguin *Spheniscus demersus*, during the carcass collection process.

Kannan, K., Perrotta, E., Thomas, N.J., and Aldous, K.M. **A comparative analysis of polybrominated diphenyl ethers and polychlorinated biphenyls in southern sea otters that died of infectious diseases and noninfectious causes.** *Archives of Environmental Contamination and Toxicology* 53(2): 293-302, 2007.

Notes: Southern sea otters (*Enhydra lutris nereis*) from the California coast continue to exhibit a slower population regrowth rate than the population in Alaska. Infectious diseases have been identified as a frequent cause of death. Infectious diseases caused by varied pathogens including bacteria, fungi, and parasites were suggestive of compromised immunological health of mature animals in this population. To test the hypothesis that elevated exposure to immunotoxic contaminants such as

polybrominated diphenyl ethers (PBDEs) and polychlorinated biphenyls (PCBs) contribute to disease susceptibility via immunosuppression, we determined concentrations of PBDEs and PCBs in livers of 80 adult female sea otters that died of infectious diseases, noninfectious causes, or emaciation. Concentrations of PBDEs and PCBs in sea otter livers varied widely (10-26,800 ng/g and 81-210,000 ng/g, lipid weight, respectively). Concentrations of PBDEs in sea otters were some of the highest values reported for marine mammals so far. Although PCB concentrations in sea otters have declined during 1992-2002, the mean concentration was at the threshold at which adverse health effects are elicited. Concentrations of PBDEs and PCBs were significantly correlated, suggesting co-exposure of these contaminants in sea otters. No significant association was found between the concentrations of PBDEs and the health status of sea otters. Concentrations of PCBs were significantly higher in otters in the infectious disease category than in the noninfectious category, suggesting an association between elevated PCB concentrations and infectious diseases in Southern sea otters.

Spraker, T.R., Delong, R.L., Lyons, E.T., and Melin, S.R. **Hookworm enteritis with bacteremia in California sea lion pups on San Miguel Island.** *Journal of Wildlife Diseases* 43(2): 179-188, 2007.

Notes: Large breeding populations of California sea lions (*Zalophus californianus*) are located on San Miguel and San Nicolas Islands in the Southern California Bight. In 2001, there was a substantial increase in pup mortality in late summer and fall. From June 2002 to January 2003, 208 freshly dead pups were examined on San Miguel Island, the most western of the Channel Islands off the coast of southern California. Tissues from 186 of these pups were examined histologically. The primary lesions in 133 (72%) of the pups were an enteritis associated with hookworms and infections in major organs. Emaciation/starvation in 43 pups (26%) was the second most important cause of death.

Aguirre, A.A., Keefe, T.J., Reif, J.S., Kashinsky, L., Yochem, P.K., Saliki, J.T., Stott, J.L., Goldstein, T., Dubey, J.P., Braun, R., and Antonelis, G. **Infectious disease monitoring of the endangered Hawaiian monk seal.** *Journal of Wildlife Diseases* 43(2): 229-241, 2007.

Notes: As part of conservation efforts between 1997 and 2001, more than 25% (332 animals) of the endangered Hawaiian monk seal (*Monachus schauinslandi*) population was sampled in the northwestern Hawaiian Islands. Serum samples were tested for antibodies to viruses, bacteria, and parasites known to cause morbidity and mortality in other marine mammal species. Antibodies were found to phocine herpesvirus-1 by using an enzyme-linked immunosorbent assay, but seropositive results were not confirmed by virus neutralization test. Antibodies to *Leptospira bratislava*, *L. hardjo*, *L. icterohaemorrhagiae*, and *L. pomona* were detected in seals from several sites with the microagglutination test. Antibodies to *Brucella* spp. were detected using 10 conventional serologic tests, but because of inconsistencies in test results and laboratories used, and the lack of validation by culture, the *Brucella* serology should be interpreted with caution. Antibodies to *B. canis* were not detected by card test. *Chlamydophila abortus* antibodies were detected by complement fixation (CF) test, and prevalence increased significantly as a function of age; the low sensitivity and specificity associated with the CF make interpretation of results difficult. Antibodies to *Toxoplasma gondii* and *Dirofilaria immitis* were rarely found. There was no serologic evidence of exposure to four morbilliviruses, influenza A virus, canine adenovirus, caliciviruses, or other selected viruses. Continuous surveillance provides a means to detect the introduction or emergence of these or other infectious diseases, but it is dependent on the development or improvement of diagnostic tools. Continued and improved surveillance are both needed as part of future conservation efforts of Hawaiian monk seals.

Sosa, E.R., Landsberg, J.H., Stephenson, C.M., Forstchen, A.B., Vandersea, M.W., and Litaker, R.W. ***Aphanomyces invadans* and ulcerative mycosis in estuarine and freshwater fish in Florida.** *Journal of Aquatic Animal Health* 19(1): 14-26, 2007.

Notes: In the spring of 1998, the Florida Fish and Wildlife Research Institute received numerous reports of lesioned or ulcerated fish primarily from the St. Lucie Estuary on the southeast coast of Florida, an area known since the late 1970s for lesions of the ulcerative mycosis (UM) type. From these and archived reports, as well as others received from different areas of Florida, we documented that diseased specimens had randomly distributed skin ulcers (usually reddened or hemorrhagic) with raised irregular margins and, in some cases, deeply penetrating hyphae in the surrounding muscle tissue. Since 1998, 256 fish (comprising 19 species) with ulcerative lesions (from 15 different locations) were confirmed with hyphae in fresh squash

preparation or by histological evaluation. Squash preparations revealed nonseptate, sparsely branching, thick-walled hyphae; histological sections revealed incyctic granulomas in the dermis that occasionally penetrated into the skeletal muscle. These pathological characteristics were consistent with UM caused by the oomycete *Aphanomyces invadans* in Southeast Asia, Japan, Australia, and the United States. For specific identification, six isolates from ulcerated fish were cultured and prepared for molecular characterization using established diagnostic methods. Ribosomal RNA gene sequence analysis identified three isolates as *Aphanomyces invadans*, one as the oomycete *Achlya bisexualis*, and two as the ascomycete *Phialemonium dimorphosporum*. A more extensive survey of 67 ulcerated skin samples from fish collected between 1998 and 2003 was performed using a polymerase chain reaction assay specific for *Aphanomyces invadans*. Of these, 26 (38.8%) samples from seven fish species and nine collection locations were positive. Confirmation of UM associated with *Aphanomyces invadans* represents new host records in Florida for the sheepshead *Archosargus probatocephalus*, striped mullet *Mugil cephalus*, white mullet *Mugil curema*, silver perch *Bairdiella chrysoura*, black drum *Pogonias cromis*, largemouth bass *Micropterus salmoides*, and American shad *Alosa sapidissima*.

Sosa, E.R., Landsberg, J.H., Kiryu, Y., Stephenson, C.M., Cody, T.T., Dukeman, A.K., Wolfe, H.P., Vandersea, M.W., and Litaker, R.W. **Pathogenicity studies with the fungi *Aphanomyces invadans*, *Achlya bisexualis*, and *Phialemonium dimorphosporum*: Induction of skin ulcers in striped mullet.** *Journal of Aquatic Animal Health* 19(1): 41-48, 2007.

Notes: Based on isolations from naturally infected fish in Florida, we investigated the role of the fungi *Aphanomyces invadans*, *Achlya bisexualis*, and *Phialemonium dimorphosporum* in the etiology of ulcerative mycosis (UM) in striped mullet *Mugil cephalus*. We injected healthy striped mullet subcutaneously with secondary zoospores of four oomycete isolates: two concentrations (50 and 115 zoospores/mL) of SJR (an endemic isolate of *Aphanomyces invadans* in American shad *Alosa sapidissima* from the St. Johns River); two concentrations each of CAL (25 and 65 zoospores/mL) and ACH (1,400 and 2,000 zoospores/mL; endemic isolates of *Aphanomyces invadans* and *Achlya bisexualis*, respectively, in striped mullet from the Caloosahatchee River); and two concentrations of the ascomycete culture MTZ (2,500 and 3,500 zoospores/mL; endemic isolate of *P. dimorphosporum* from whirligig mullet *M. gyrans* in the Matanzas Inlet). All fish injected with either concentration of SJR developed granulomatous ulcers after 8 d and died within 21 d. Eighty percent (8/10) of fish injected with the high dose of CAL developed ulcers after 13 d and died within 28 d, but only 30% (3/10) of fish injected with the low dose of CAL developed ulcers. Four of the ulcerated fish died within 28 d, and the remaining fish were terminated after 32 d. Fish injected with zoospores of *Aphanomyces invadans* developed ulcers that were grossly and histologically similar to those observed in naturally infected striped mullet with UM from several estuaries or rivers in Florida. These hemorrhagic skin ulcers were characterized by myonecrosis and the presence of mycotic granulomas. None of the fish injected with ACH, MTZ, or sterile water developed ulcers. This study fulfilled Koch's postulates and demonstrated that ulcers could be experimentally induced in striped mullet after exposure via injection to secondary zoospores of an endemic Florida strain of *Aphanomyces invadans*.

Koenig, W.D., Marcus, L., Scott, T.W., and Dickinson, J.L. **West Nile virus and California breeding bird declines.** *EcoHealth* 4(1): 18-24, 2007.

Notes: Since it was first detected in 1999, West Nile virus (WNV) quickly spread, becoming the dominant vector-borne disease in North America. Sometimes fatal to humans, WNV is even more widespread among birds, with hundreds of species known to be susceptible to WNV infection in North America alone. However, despite considerable mortality and local declines observed in American crows (*Corvus brachyrhynchos*), there has been little evidence of a large regional association between WNV susceptibility and population declines of any species. Here we demonstrate a correlation between susceptibility to WNV measured by large-scale testing of dead birds and two indices of overall population change among bird species following the spread of WNV throughout California. This result was due primarily to declines in four species of Corvidae, including all species in this family except common ravens (*Corvus corax*). Our results support the hypothesis that susceptibility to WNV may have negative population consequences to most corvids on regional levels. They also provide confirmation that dead animal surveillance programs can provide important data indicating populations most likely to suffer detrimental impacts due to WNV.

Swift, L., Hunter, P.R., Lees, A.C., and Bell, D.J. **Wildlife trade and the emergence of infectious diseases.** *EcoHealth* 4(1): 25-30, 2007.

Notes: Most recent emerging infectious diseases have been zoonotic in origin. It is our contention that one of the factors responsible for such emergence is the trade in wildlife and bushmeat in particular. This article considers the effect of increasing diversity in the species hunted on the probability of global epidemics such as SARS. In particular, we develop a mathematical model of the probability of such an outbreak in terms of the number of species hunted, the number of susceptibles, and the rate of contact. Hence, we postulate that local biodiversity loss and increasing rates of animal trafficking, and trade and transportation of animals to large cities -- where there is a greater potential for person-to-person transmission -- may increase the probability of such outbreaks dramatically.

Gulland, F.M.D. and Hall, A.J. **Is marine mammal health deteriorating? Trends in the global reporting of marine mammal disease.** *EcoHealth* 4(2): 135-150, 2007.

Notes: A recent rise in the reporting of diseases in marine organisms has raised concerns that ocean health is deteriorating. The goal of this study was to determine whether or not there has been a recent deterioration in marine mammal health by investigating the trends in disease reports over the past 40 years (categorized by the method of study, the species affected, and the etiology of the disease) and by exploring the changes in frequency of mass mortality events among marine mammals reported in the United States since 1978. The number of papers on marine mammal disease published each year has increased since 1966, although the annual publication rate appears to have stabilized since ~1992. Those published in the 1960s and 1970s were largely about helminth and bacterial disease, those investigating viruses emerged in the late 1970s and increased in the 1980s and 1990s, whereas protozoal diseases and harmful algal toxins were largely not reported until the 1990s. The annual number of mass mortality events in the U.S. approximately doubled between 1980 and 1990 but since 2000 has been between seven and eight events per year. Causes of mass mortality events have included biotoxins, viruses, bacteria, parasites, human interactions, oil spills, and changes in oceanographic conditions. Events due to biotoxins appear to be increasing, but the change in the frequency of mass mortality events from other causes over the past 40 years cannot be determined from the available published literature due to changes in marine mammal abundance, inconsistencies in effort and extent of resources for pathological investigation, and advances in technology that have allowed improved detection of pathogens and toxins in more recent years. To ensure future information on the true incidence of marine diseases and their underlying causes is more reliable, specific and directed marine health monitoring programs, well-equipped stranding networks, and dedicated diagnostic laboratories are needed.
