

# Marine Science Review - 255

## Pathogens, disease and die-offs

### In this review:

- A. Recent articles – no abstract available
- B. Recent articles with abstracts

O/A denotes an open access article or journal

## A. Recent articles – no abstract available

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Morado, F. **Bitter crab syndrome: A major player in the global theater of marine crustacean disease.** *AFSC Quarterly Research Report* (July-August-September): 1-6, 2007. O/A

Jessup, D.A., Miller, M.A., Kreuder-Johnson, C., Conrad, P.A., Tinker, M.T., Estes, J., and Mazet, J.A.K. **Sea otters in a dirty ocean.** *Journal of the American Veterinary Medical Association* 231(11): 1648-1652, 2007.

Wohlsein, P., Puff, C., Kreutzer, M., Siebert, U., and Baumgärtner, W. **Distemper in a dolphin.** *Emerging Infectious Diseases* 13(12): 1959-1961, 2007. O/A

Prescott, J. **Characterization of *Klebsiella pneumoniae* isolates from New Zealand sea lion pups.** [Letter to the Editor] *Veterinary Microbiology* 125(3-4): 387, 2007.

Castinel, A., Pomroy, B., and Grinberg, A. **Hookworm infection and *Klebsiella pneumoniae* epidemics in New Zealand sea lion pups.** [Response to Letter to the Editor] *Veterinary Microbiology* 125(3-4): 388-389, 2007.

## B. Recent articles with abstracts

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Muzaffar, S.B., Hoberg, E.P., and Jones, I.L. **First record of *Alcataenia larina larina* (Cestoda: Dilepididae) in Atlantic puffins (Aves, Alcidae, *Fratercula arctica*) from Newfoundland, Canada.** *Comparative Parasitology* 74(2): 380-382, 2007.

**Notes:** The tapeworm *Alcataenia larina* (Cestoda: Cyclophyllidae: Dilepididae) is a parasite of gulls (Laridae) with a Holarctic distribution in oceanic and littoral habitats. Two subspecies *Alcataenia larina pacifica* and *Alcataenia larina larina* are recognized with the former occurring in the North Pacific basin and the latter in the North Atlantic. Alcids serve as incidental hosts for both species and infections are generally rare, usually involving few specimens. We report *A. l. larina* in Atlantic puffins (*Fratercula arctica*) collected from the Bay of Exploits, Newfoundland. The 14 strobilate and early gravid specimens were clearly distinguishable from the closely related *Alcataenia cerorbinae* and *Alcataenia fraterculae* by the smaller dimensions of the rostellar hooks and cirrus sac. Increasing numbers of gulls around seabird colonies in Newfoundland may result in more frequent contact between gulls and alcids, such as puffins (during foraging), promoting cross infections of *A. l. larina*.

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Dubey, J.R., Morales, J.A., Sundar, N., Velmurugan, G.V., Gonzalez-Barrientos, C.R., Hernandez-Morat, G., and Su, C. **Isolation and genetic characterization of *Toxoplasma gondii* from striped dolphin (*Stenella coeruleoalba*) from**

Costa Rica. *Journal of Parasitology* 93(3): 710-711, 2007.

**Notes:** *Toxoplasma gondii* infection in marine mammals is of interest because of mortality and mode of transmission. It has been suggested that marine mammals become infected with *T. gondii* oocysts washed from land to the sea. We report the isolation and genetic characterization of viable *T. gondii* from a striped dolphin (*Stenella coeruleoalba*), the first time from this host. An adult female dolphin was found stranded on the Pacific Coast of Costa Rica, and the animal died the next day. The dolphin had a high (1:6,400) antibody titer to *T. gondii* in the modified agglutination test. Severe nonsuppurative meningoencephalomyelitis was found in its brain and spinal cord, but *T. gondii* was not found in histological sections of the dolphin. Portions of its brain and the heart were bioassayed in mice for the isolation of *T. gondii*. Viable *T. gondii* was isolated from the brain, but not from the heart, of the dolphin. A cat fed mice infected with the dolphin isolate (designated TgSdCol) shed oocysts. Genomic DNA from tachyzoites of this isolate was used for genotyping at 10 genetic loci, including SAG1, SAG2, SAG3, BTUB, GRA6, c22-8, c29-2, L358, PK1, and Apico, and this TgSdCol isolate was found to be Type II.

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Guimaraes, P.R., de Menezes, M.A., Baird, R.W., Lusseau, D., Guimaraes, P., and dos Reis, S.F. **Vulnerability of a killer whale social network to disease outbreaks.** *Physical Review E* 76: art. 042901, 2007. O/A

**Notes:** Emerging infectious diseases are among the main threats to conservation of biological diversity. A crucial task facing epidemiologists is to predict the vulnerability of populations of endangered animals to disease outbreaks. In this context, the network structure of social interactions within animal populations may affect disease spreading. However, endangered animal populations are often small and to investigate the dynamics of small networks is a difficult task. Using network theory, we show that the social structure of an endangered population of mammal-eating killer whales is vulnerable to disease outbreaks. This feature was found to be a consequence of the combined effects of the topology and strength of social links among individuals. Our results uncover a serious challenge for conservation of the species and its ecosystem. In addition, this study shows that the network approach can be useful to study dynamical processes in very small networks.

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Toledo-Hernandez, C., Sabat, A.M., and Zuluaga-Montero, A. **Density, size structure and aspergillosis prevalence in *Gorgonia ventalina* at six localities in Puerto Rico.** *Marine Biology* 152(3): 527-535, 2007.

**Notes:** *Gorgonia ventalina*'s density, size structure and lesion prevalence was measured at six sites in Puerto Rico that exhibited variation in horizontal water transparency, sedimentation rates, suspended particle matter, scleractinian and macroalgal cover. *G. ventalina* density varied significantly among sites (between 0.84 and 0.007 colonies/m<sup>2</sup>), and was positively correlated with water transparency. Size structure did not vary much among sites, and reflects high mortality among the smaller size classes and high survivorship in large colonies. Prevalence of active fungi-induced lesions (type I) did not vary significantly among sites and was density-independent. However, prevalence of old lesions of unknown origin (type II) did vary among reefs and was negatively correlated with water transparency. Prevalence of types I or II lesions was independent of colony size. Our results suggest that (1) turbidity and sedimentation are important abiotic factors controlling the abundance of sea fans, (2) variation in settlement success and early survivorship of recruits has more impact on the sea fan populations than variation in the survivorship of large colonies and (3) prevalence of aspergillosis (type I) at the studied sites is similar to that reported for other Caribbean reefs and supports the epizootic nature of the disease and (4) lesions with exposed skeleton are more likely to be colonized by fouling organisms at impacted reefs. The combined effects of anthropogenic impacts and aspergillosis may cause local extinctions of sea fans, as is becoming evident in many reefs in Puerto Rico.

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Hurst, T.P. **Causes and consequences of winter mortality in fishes.** *Journal of Fish Biology* 71(2): 315-345, 2007.

**Notes:** Winter mortality has been documented in a large number of freshwater fish populations, and a smaller, but increasing, number of marine and estuarine fishes. The impacted populations include a number of important North American and European resource species, yet the sources of winter mortality remain unidentified in most populations where it has been documented. Among the potential sources, thermal stress and starvation have received the most research attention. Other sources including predation and pathogens have significant impacts but have received insufficient attention to date. Designs of more recent laboratory experiments have reflected recognition of the potential for interactions among these co-occurring stressors. Geographic patterns in winter mortality are, in some cases, linked to latitudinal clines in winter severity and

variability. However, for many freshwater species in particular, the effects of local community structure (predators and prey) may overwhelm latitudinal patterns. Marine (and estuarine) systems differ from freshwater systems in several aspects important to overwintering fishes, the most important being the lack of isolating barriers in the ocean. While open population boundaries allow fish to adopt migration strategies minimizing exposure to thermal stresses, they may retard rates of evolution to local environments. Geographic patterns in the occurrence and causes of winter mortality are ultimately determined by the interaction of regional and local factors. Winter mortality impacts population dynamics through episodic depressions in stock size and regulation of annual cohort strength. While the former tends to act in a density-independent manner, the latter can be density dependent, as most sources of mortality tend to select against the smallest members of the cohort and population. Most stock assessment and management regimes have yet to explicitly incorporate the variability in winter mortality. Potential management responses include postponement of cohort evaluation (to after first winter of life), harvest restrictions following mortality events and habitat enhancement. Future research should place more emphasis on the ecological aspects of winter mortality including the influences of food-web structure on starvation and predation. Beyond illuminating an understudied life-history phase, studies of overwintering ecology are integral to contemporary issues in fisheries ecology including ecosystem management, habitat evaluation, and impacts of climate change.

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Brooks, D.R. and Hoberg, E.P. **How will global climate change affect parasite-host assemblages?** *Trends in Parasitology* 23(12): 571-574, 2007.

**Notes:** Parasites are integral components of the biosphere. Host switching correlated with events of episodic climate change is ubiquitous in evolutionary and ecological time. Global climate change produces ecological perturbations, which cause geographical and phenological shifts, and alteration in the dynamics of parasite transmission, increasing the potential for host switching. The intersection of climate change with evolutionary conservative aspects of host specificity and transmission dynamics, called ecological fitting, permits emergence of parasites and diseases without evolutionary changes in their capacity for host utilization.

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Lyons, M.M., Smolowitz, R., Gomez-Chiarri, M., and Ward, J.E. **Epizootiology of Quahog Parasite Unknown (QPX) disease in northern quahogs (=hard clams) *Mercenaria mercenaria*.** *Journal of Shellfish Research* 26(2): 371-381, 2007.

**Notes:** The economically important marine bivalve mollusc, *Mercenaria mercenaria*, (commonly called a northern quahog or hard clam), has endured considerable mortalities caused by a thraustochytrid pathogen called Quahog Parasite X (QPX). Data on the percent prevalence of QPX infections were compiled from published reports along with our data to describe the epizootiology of QPX disease. QPX infections occurred in clams collected from both cultured beds and wild populations, but a higher percentage of QPX cases (76.5%) were from cultured clam beds. In addition, samples from cultured beds had a significantly higher prevalence ( $29.2 \pm 27.2\%$ ) of QPX infections compared with samples from wild populations ( $9.6 \pm 9.6\%$ ). The highest prevalence of QPX infections occurred in clams from samples with an intermediate size range (shell lengths 20-55 mm). QPX infections occurred in both male and female clams, but infection prevalence does not appear to be correlated with sex or sex ratios. The geographical range of QPX-related clam mortalities was Atlantic Canada to the Eastern Shore of Virginia, USA. Only marginally significant differences were detected between the prevalence of QPX at different locations. There were no latitudinal gradients in QPX prevalence or frequencies, suggesting local factors were important in determining its distribution. Although QPX infections occurred throughout the year no seasonal trends in the prevalence or frequencies of QPX were discernable. This summary of information available on QPX disease highlights the need for more thorough data collection regarding factors believed to be associated with its presence and severity in hard clams.

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Myers, J.L., Sekar, R., and Richardson, L.L. **Molecular detection and ecological significance of the cyanobacterial genera *Geitlerinema* and *Leptolyngbya* in black band disease of corals.** *Applied and Environmental Microbiology* 73(16): 5173-5182, 2007.

**Notes:** Black band disease (BBD) is a pathogenic, sulfide-rich microbial mat dominated by filamentous cyanobacteria that infect corals worldwide. We isolated cyanobacteria from BBD into culture, confirmed their presence in the BBD community by using denaturing gradient gel electrophoresis (DGGE), and demonstrated their ecological significance in terms of physiological sulfide tolerance and photosynthesis-versus-irradiance values. Twenty-nine BBD samples were collected from

nine host coral species, four of which have not previously been investigated, from reefs of the Florida Keys, the Bahamas, St. Croix, and the Philippines. From these samples, seven cyanobacteria were isolated into culture. Cloning and sequencing of the 16S rRNA gene using universal primers indicated that four isolates were related to the genus *Geitlerinema* and three to the genus *Leptolyngbya*. DGGE results, obtained using Cyanobacteria-specific 16S rRNA primers, revealed that the most common BBD cyanobacterial sequence, detected in 26 BBD field samples, was related to that of an *Oscillatoria* sp. The next most common sequence, 99% similar to that of the *Geitlerinema* BBD isolate, was present in three samples. One *Leptolyngbya*- and one *Phormidium*-related sequence were also found. Laboratory experiments using isolates of BBD *Geitlerinema* and *Leptolyngbya* revealed that they could carry out sulfide-resistant oxygenic photosynthesis, a relatively rare characteristic among cyanobacteria, and that they are adapted to the sulfide-rich, low-light BBD environment. The presence of the cyanotoxin microcystin in these cultures and in BBD suggests a role in BBD pathogenicity. Our results confirm the presence of *Geitlerinema* in the BBD microbial community and its ecological significance, which have been challenged, and provide evidence of a second ecologically significant BBD cyanobacterium, *Leptolyngbya*.

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Baker, D.M., MacAvoy, S.E., and Kim, K. **Relationship between water quality,  $\delta^{15}\text{N}$ , and aspergillosis of Caribbean sea fan corals.** *Marine Ecology Progress Series* 343: 123-130, 2007.

**Notes:** It has been hypothesized that excess nitrogen (N) contributes to coral reef decline by exacerbating the impact of disease. We tested the relationship between N concentration and the prevalence and severity of aspergillosis of the sea fan coral *Gorgonia ventalina* at 9 reefs along the Florida Keys Reef Tract. Quarterly water quality data, averaged over 1 and 4 yr prior to a disease survey, were used to examine whether aspergillosis dynamics reflected short- or long-term N concentrations. A positive relationship was detected between prevalence of aspergillosis and long-term total nitrogen (TN) concentration; in contrast, disease severity was positively related to the ratio between dissolved inorganic nitrogen and total phosphate (DIN:TP) over both short- and long-terms. These results may reflect the differential influence of N on the host and pathogen. We also tested whether N isotope analysis ( $\delta^{15}\text{N}$ ) of sea fan coral tissue and skeleton could be used to assess the relative exposure to anthropogenic N inputs and its impact on disease. There was no relationship between  $\delta^{15}\text{N}$  and aspergillosis (either prevalence or severity). Furthermore, there was no relationship between  $\delta^{15}\text{N}$  and environmental concentrations of N. It is possible that the source of N (e.g. anthropogenic) does not affect the dynamics of sea fan aspergillosis, or that the  $\delta^{15}\text{N}$  signatures were suppressed by agricultural effluents and other N sources, thus confounding our analyses with disease.

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Barneah, O., Ben Dov, E., Kramarsky-Winter, E., and Kushmaro, A. **Characterization of black band disease in Red Sea stony corals.** *Environmental Microbiology* 9(8): 1995-2006, 2007.

**Notes:** Microbial communities associated with black band disease (BBD) in massive stony corals from the Northern Red Sea (Eilat) were examined for the first time using molecular tools and microscopy. A high microbial diversity was revealed in the affected tissue in comparison with the healthy area of the same colony. Microscopy revealed the penetration of cyanobacteria into the coral mesoglea and adjacent tissues. Cyanobacterial sequences from Red Sea BBD-affected corals formed a cluster with sequences previously identified from black band and red band diseased corals from the Indo-Pacific and Caribbean. In addition, 11 sequences belonging to the genus *Vibrio* were retrieved. This group was previously documented as pathogenic to corals. Sulfate-reducing bacteria, a group known to be associated with BBD and produce toxic sulfide, were studied using specific primers for the amplification of the dissimilatory sulfite reductase gene (*dsrA*). This technique facilitated and improved the resolution of the study of diversity of this group. All the sequences obtained were closely related to sequences of the genus *Desulfovibrio* and 46% showed high homology to *Desulfovibrio desulfuricans*. The complex nature of BBD and the lack of success in isolating a single causative agent suggest that BBD may be considered a polymicrobial disease.

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Bally, M. and Garrabou, J. **Thermodependent bacterial pathogens and mass mortalities in temperate benthic communities: a new case of emerging disease linked to climate change.** *Global Change Biology* 13(10): 2078-2088, 2007.

**Notes:** In the temperate north-western Mediterranean Sea, large-scale disease outbreaks in benthic invertebrate species have recently occurred during climatic anomalies characterized by elevated seawater temperatures. One of the most affected species was the red gorgonian *Paramuricea clavata*, a key species of highly diverse communities dwelling in dim-lit habitats in the Mediterranean. From diseased *P. clavata* colonies, we isolated culturable bacteria associated to tissue lesions in order to

investigate their potential as pathogens. Inoculation of four bacterial isolates onto healthy *P. clavata* in aquaria caused disease signs similar to those observed during the 2003 mortality event. The infection process was dependent on elevated seawater temperatures, in a range of values consistent with recordings performed in the field during the climatic anomalies. Among the four isolates, we identified a *Vibrio coralliilyticus* strain that showed virulence to *P. clavata*. This strain was re-isolated from diseased colonies during the experimentations. *V. coralliilyticus* has been previously identified as a thermodependent pathogen of a tropical coral species, emphasizing a causal role of this infectious agent in the *P. clavata* disease. Taking into consideration predicted global warming over the coming decades, a better understanding of the factors and mechanisms that affect the disease process will be of critical importance in predicting future threats to temperate gorgonian communities in the Mediterranean Sea.

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Pedersen, A.B., Jones, K.E., Nunn, C.L., and Altizer, S. **Infectious diseases and extinction risk in wild mammals.** *Conservation Biology* 21(5): 1269-1279, 2007.

**Notes:** Parasite-driven declines in wildlife have become increasingly common and can pose significant risks to natural populations. We used the IUCN Red List of Threatened and Endangered Species and compiled data on hosts threatened by infectious disease and their parasites to better understand the role of infectious disease in contemporary host extinctions. The majority of mammal species considered threatened by parasites were either carnivores or artiodactyls, two clades that include the majority of domesticated animals. Parasites affecting host threat status were predominantly viruses and bacteria that infect a wide range of host species, including domesticated animals. Counter to our predictions, parasites transmitted by close contact were more likely to cause extinction risk than those transmitted by other routes. Mammal species threatened by parasites were not better studied for infectious diseases than other threatened mammals and did not have more parasites or differ in four key traits demonstrated to affect parasite species richness in other comparative studies. Our findings underscore the need for better information concerning the distribution and impacts of infectious diseases in populations of endangered mammals. In addition, our results suggest that evolutionary similarity to domesticated animals may be a key factor associated with parasite-mediated declines, thus, efforts to limit contact between domesticated hosts and wildlife could reduce extinction risk.

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Palstra, A.P., Heppener, D.F.M., van Ginneken, V.J.T., Szekely, C., and van den Thillart, G.E.E.J. **Swimming performance of silver eels is severely impaired by the swim-bladder parasite *Anguillicola crassus*.** *Journal of Experimental Marine Biology and Ecology* 352(1): 244-256, 2007.

**Notes:** Infection with the swim-bladder parasite *Anguillicola crassus* is suggested as one of the principal causes of the collapse of the European eel population. This nematode has been introduced in Europe from Asia in the 80s and parasitized in a short time *Anguilla* eel species in different geographical regions across the globe. The parasites drain energy due to their sanguivorous feeding and they cause mechanical damage on the swim-bladder wall. These two effects are hypothesized to impair the spawning migration of the European eel. In this study, we have investigated both effects on swimming performance. We hypothesized that parasitic sanguivorous activities - related to parasite weight - reduce swimming endurance, while mechanical damage of the swim-bladder impairs buoyancy control. Eighty eels suffering various degrees of infection were introduced in swim-tunnels and subjected to a swimming fitness test. The relation between *A. crassus* infection and swimming efficiency was measured for large female silver eels swimming at various speeds. Infected eels had lower cruising speeds and a higher cost of transport. Eels without parasites, but with a damaged swim-bladder showed similar effects. Almost half of the eels that contained damaged swim-bladders (43%) stopped swimming at low aerobic swimming speeds (< 0.7 m/s). Simulated migration trials in a recent related study have confirmed that eels with a high parasite level or with damaged swim-bladder show early migration failure (< 1000 km). Reduced swimming performance appears to be associated with swim-bladder dysfunction. As we found that especially silver eels have much higher infection levels than yellow eels, it is concluded that migrating silver eels with severely infected or damaged swim-bladders are unable to reach the spawning grounds.

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Belden, L.K. and Harris, R.N. **Infectious diseases in wildlife: the community ecology context.** *Frontiers in Ecology and Environment* 5(10): 533-539, 2007.

**Notes:** Species diversity can have important effects on disease dynamics. While these effects are often considered with respect to alternate hosts and predators, the influence of diversity may also be seen at the level of the parasite or pathogen. Pathogenic microbes face an array of abiotic and biotic challenges, both within their host and, often, in the external environment. Here, we examine the role of microbial ecology in maintaining health and in contributing to disease. As suggested by some medical scientists and others, we argue that placing pathogens in an ecological context can contribute to our understanding of emerging infectious diseases in natural systems. In addition, we suggest that this view could provide important insights for the conservation of species, including many amphibians, that are threatened by disease outbreaks.

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Hoberg, E.P., Polley, L., Jenkins, E.J., Kutz, S.J., Veitch, A.M., and Elkin, B.T. **Integrated approaches and empirical models for investigation of parasitic diseases in northern wildlife.** *Emerging Infectious Diseases* 14(1): 10-17, 2008. O/A

**Notes:** The North is a frontier for exploration of emerging infectious diseases and the large-scale drivers influencing distribution, host associations, and evolution of pathogens among persons, domestic animals, and wildlife. Leading into the International Polar Year 2007-2008, we outline approaches, protocols, and empirical models derived from a decade of integrated research on northern host-parasite systems. Investigations of emerging infectious diseases associated with parasites in northern wildlife involved a network of multidisciplinary collaborators and incorporated geographic surveys, archival collections, historical foundations for diversity, and laboratory and field studies exploring the interface for hosts, parasites, and the environment. In this system, emergence of parasitic disease was linked to geographic expansion, host switching, resurgence due to climate change, and newly recognized parasite species. Such integrative approaches serve as cornerstones for detection, prediction, and potential mitigation of emerging infectious diseases in wildlife and persons in the North and elsewhere under a changing global climate.

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Castinel, A., Grinberg, A., Pattison, R., Duignan, P., Pomroy, B., Rogers, L., and Wilkinson, I. **Characterization of *Klebsiella pneumoniae* isolates from New Zealand sea lion (*Phocarctos hookeri*) pups during and after the epidemics on Enderby Island, Auckland Islands.** *Veterinary Microbiology* 122(1-2): 178-184, 2007.

**Notes:** The 2001/2002 and 2002/2003 breeding seasons of New Zealand sea lions (NZSLs) on the Auckland Islands were marked by a high pup mortality caused by acute bacterial infections. As part of a health survey from 1998/1999 to 2004/2005, tissues and swabs of lesions had been collected at necropsy to identify the bacteria associated with pup mortality. *Klebsiella pneumoniae* was grown in pure culture from 83% of various organs and lesions in 2001/2002 and 76% in 2002/2003, and less frequently in the following seasons (56% in 2003/2004 and 49% in 2004/2005). Pup isolates of *K. pneumoniae* showed identical minimal inhibitory concentrations (MIC) of cefuroxime, neomycin, cephalotin, cephalixin and dihydrostreptomycin, suggesting clonal aetiology of the pathogen. Isolates also tested negative for production of extended-spectrum beta-lactamases (ESBLs), which was not in favour of an anthropogenic origin of the epidemic strain. Pulsed-field gel electrophoresis (PFGE) of XbaI DNA macrorestriction fragments was performed on isolates of *K. pneumoniae* and *Klebsiella oxytoca* from 35 pups, three NZSL adult females, and from three human patients for comparison. PFGE showed that pup isolates of *K. pneumoniae* were genetically indistinguishable but were neither related to *K. pneumoniae* from humans and from NZSL adults, nor to *K. oxytoca* from NZSLs. It is concluded that the 2001/2002 and 2002/2003 epidemics at Sandy Bay rookery were caused by a single *K. pneumoniae* clonal lineage, genetically different from the strain carried by adult NZSLs. An anthropogenic origin of the *K. pneumoniae* clone could not be confirmed, but further investigations are required to rule-out such occurrence.

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McKenzie, V.J. and Townsend, A.R. **Parasitic and infectious disease responses to changing global nutrient cycles.** *EcoHealth* 4(4): 384-396, 2007.

**Notes:** Parasitic and infectious diseases (PIDs) are a significant threat to human, livestock, and wildlife health and are changing dramatically in the face of human-induced environmental changes such as those in climate and land use. In this article we explore the little-studied but potentially important response of PIDs to another major environmental change, that in the global nutrient cycles. Humans have now altered the nitrogen (N) cycle to an astonishing degree, and those changes are causing a remarkable diversity of environmental and ecological responses. Since most PIDs are strongly regulated by ecological interactions, changes in nutrients are likely to affect their dynamics in a diversity of environments. We show that while direct tests of the links between nutrients and disease are rare, there is mounting evidence that higher nutrient levels frequently lead

to an increased risk of disease. This trend occurs across multiple pathogen types, including helminths, insect-vector-borne diseases, myxozoa, and bacterial and fungal diseases. The mechanistic responses to increased nutrients are often complex and frequently involve indirect responses that are regulated by intermediate or vector hosts involved in disease transmission. We also show that rapid changes in the N cycle of tropical regions combined with the high diversity of human PIDs in these regions will markedly increase the potential for N to alter the dynamics of disease. Finally, we stress that progress on understanding the effects of nutrients on disease ecology requires a sustained effort to conduct manipulative experiments that can reveal underlying mechanisms on a species-specific basis.

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Piskorska, M., Smith, G., and Weil, E. **Bacteria associated with the coral *Echinopora lamellosa* (Esper 1795) in the Indian Ocean - Zanzibar Region.** *African Journal of Environmental Science and Technology* 1(5): 93-98, 2007.

**Notes:** Infectious diseases are now known to have major effects on the structure and function of coral reef ecosystems throughout the world. The number of recognized coral diseases has increased dramatically. The problem was first recognized in the Caribbean in the early 1970's but has now been reported to affect coral communities worldwide. There is little information regarding bacteria associated with diseased corals in the Indian Ocean. However, one of the most common disease signs observed is a rapid loss of tissue leaving exposed white skeleton in contact with compromised tissue, followed by necrosis. These signs have been referred to as white plague in the Caribbean. Similar signs have been observed in the Indo-Pacific and are referred to as white syndrome. The pathogens associated with these disease signs depend on the species and geographic location of the corals. In the Caribbean, the disease was associated with *Aurantimonas corallicida* and in the Red Sea with *Thalassomonas loyaeana*, both newly described species. During exploratory surveys in the reefs near Zanzibar in the Indian Ocean, mucus samples were collected from healthy and apparently diseased *Echinopora lamellosa* (with signs of white syndrome) colonies. Samples were plated on two solid media: GASW (a nonspecific medium) and TCBS (*Vibrio* selective medium). Growth on TCBS was only found with diseased samples. Culturable isolates were characterized using metabolic profiling. A relatively high prevalence of Class Gamma Proteobacteria was found with diseased samples compared with healthy samples and *Vibrio* species were well represented in diseased samples.

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Lloyd-Smith, J.O., Greig, D.J., Hietala, S., Ghneim, G.S., Palmer, L., St. Leger, J., Grenfell, B.T., and Gulland, F.M.D. **Cyclical changes in seroprevalence of leptospirosis in California sea lions: endemic and epidemic disease in one host species?** *BMC Infectious Diseases* 7(art. 125), 2007. **O/A**

**Notes: Background:** Leptospirosis is a zoonotic disease infecting a broad range of mammalian hosts, and is re-emerging globally. California sea lions (*Zalophus californianus*) have experienced recurrent outbreaks of leptospirosis since 1970, but it is unknown whether the pathogen persists in the sea lion population or is introduced repeatedly from external reservoirs. **Methods:** We analyzed serum samples collected over an 11-year period from 1344 California sea lions that stranded alive on the California coast, using the microscopic agglutination test (MAT) for antibodies to *Leptospira interrogans* serovar Pomona. We evaluated seroprevalence among yearlings as a measure of incidence in the population, and characterized antibody persistence times based on temporal changes in the distribution of titer scores. We conducted multinomial logistic regression to determine individual risk factors for seropositivity with high and low titers. **Results:** The serosurvey revealed cyclical patterns in seroprevalence to *L. interrogans* serovar Pomona, with 4-5 year periodicity and peak seroprevalence above 50%. Seroprevalence in yearling sea lions was an accurate index of exposure among all age classes, and indicated on-going exposure to leptospires in non-outbreak years. Analysis of titer decay rates showed that some individuals probably maintain high titers for more than a year following exposure. **Conclusion:** This study presents results of an unprecedented long-term serosurveillance program in marine mammals. Our results suggest that leptospirosis is endemic in California sea lions, but also causes periodic epidemics of acute disease. The findings call into question the classical dichotomy between maintenance hosts of leptospirosis, which experience chronic but largely asymptomatic infections, and accidental hosts, which suffer acute illness or death as a result of disease spillover from reservoir species.

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Wulff, J.L. **Disease prevalence and population density over time in three common Caribbean coral reef sponge species.** *Journal of the Marine Biological Association of the United Kingdom* 87(6): 1715-1720, 2007.

**Notes:** Reports of disease in sponges are increasing, but the paucity of data on disease prevalence over time makes it uncertain how much this trend reflects increased attention to sponges rather than increased sponge disease. Population and community influences on disease dynamics, and the consequences of disease at these levels, are also little known. Five censuses, over 14 y, of a small plot on a shallow coral reef at San Blas, Panama, provide data for the three most abundant species on population dynamics (number of individuals and total volume) and disease prevalence (number of individuals with active lesions). Although data for the three species, combined in broad categories (i.e. high vs low), support a general conclusion that disease prevalence was greater from 1994-1998 than from 1984-1988, the data do not demonstrate a steady increase over time, and disease prevalence for two of the species decreased in each of the final two censuses from a high in 1994. Fluctuations in population density (total volume) and disease prevalence were nearly synchronous within individual species, but asynchronous among the three species, suggesting that population density, measured as total sponge volume per unit area, may influence disease dynamics in these sponges.

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Neimanis, A., Gavier-Widen, D., Leighton, F., Bollinger, T., Rocke, T., and Morner, T. **An outbreak of type C botulism in Herring Gulls (*Larus argentatus*) in southeastern Sweden.** *Journal of Wildlife Diseases* 43(3): 327-336, 2007.

**Notes:** From 2000 to 2004, over 10,000 seabirds, primarily Herring Gulls (*Larus argentatus*), died from an undetermined cause in the Blekinge archipelago in southeastern Sweden. In June 2004, 24 affected Herring Gulls were examined clinically, killed humanely, and 23 were examined by necropsy. Seven and 10 unaffected Herring Gulls collected from a local landfill site and from Iceland, respectively, served as controls. All affected birds showed similar neurologic signs, ranging from mild incoordination and weakness to severe flaccid paralysis of legs and wings, but generally were alert and responsive. All affected gulls were in normal nutritional condition, but were dehydrated and had empty stomachs. No gross or microscopic lesions, and no bacterial or viral pathogens were identified. Type C botulinum toxin was detected in the sera of 11 of 16 (69%) affected gulls by mouse inoculation. Type C botulism was the proximate cause of disease in 2004. Sera from 31% of birds tested from outbreaks in 2000 to 2003 also had detectable type C botulinum toxin by mouse inoculation. No large-scale botulism outbreak has been documented previously in this area. The source of toxin, initiating conditions, and thus, the ultimate cause of this outbreak, are not known. This epidemic might signal environmental change in the Baltic Sea.

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Karesh, W.B., Cook, R.A., Gilbert, M., and Newcomb, J. **Implications of wildlife trade on the movement of avian influenza and other infectious diseases.** *Journal of Wildlife Diseases* 43(3): S55-S59, 2007. **O/A**

**Notes:** The global trade in wildlife provides disease transmission mechanisms that not only result in human disease outbreaks, but also threaten livestock, international trade, rural livelihoods, native wildlife populations, and the health of ecosystems. Global movement of animals for the pet trade is estimated at some 350 million live animals, worth approximately US\$20 billion per year. Approximately one-quarter of this trade is thought to be illegal, hence not inspected or tested. Disease outbreaks resulting from trade in wildlife have caused hundreds of billions of dollars of economic damage globally. Rather than attempting to eradicate pathogens or the wild species that may harbor them, a practical approach would include decreasing the contact rate among species, including humans, at the interface created by wildlife trade. Wild animals are captured, transported, and sold either live or dead and commingled throughout the process in a system of scale-free networks with major hubs rather than random or evenly distributed supply systems. As focal points for distribution and sales, the hubs provide control opportunities to maximize the effects of regulatory efforts as demonstrated with domestic animal trading systems (processing plants and wholesale and retail markets, for example). Focusing efforts at markets to regulate, reduce, or in some cases, eliminate the commercial trade in wildlife could provide a cost-effective approach to decrease the risks for disease in humans, domestic animals, wildlife, and ecosystems.

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Hall-Spencer, J.M., Pike, J., and Munn, C.B. **Diseases affect cold-water corals too: *Eunicella verrucosa* (Cnidaria: Gorgonacea) necrosis in SW England.** *Diseases of Aquatic Organisms* 76(2): 87-97, 2007. **O/A**

**Notes:** The first recorded incidence of cold-water coral disease was noted in *Eunicella verrucosa*, a coral on the international 'red list' of threatened species, at a marine protected area in SW England in 2002. Video surveys of 634 separate colonies at 13 sites revealed that disease outbreaks were widespread in SW England from 2003 to 2006. Coenchyme became necrotic in diseased specimens, leading to tissue sloughing and exposing skeletal gorgonin to settlement by fouling organisms. Sites where necrosis

was found had significantly higher incidences of fouling. No fungi were isolated from diseased or healthy tissue, but significantly higher concentrations of bacteria occurred in diseased specimens. Of 21 distinct bacteria isolated from diseased tissues, 19 were Vibrionaceae, 15 were strains of *Vibrio splendidus* and 2 others closely matched *Vibrio tasmaniensis*. Vibrios isolated from *E. verrucosa* did not induce disease at 15°C, but, at 20°C, controls remained healthy and test gorgonians became diseased, regardless of whether vibrios were isolated from diseased or healthy colonies. Bacteria associated with diseased tissue produced proteolytic and cytolytic enzymes that damaged *E. verrucosa* tissue and may be responsible for the necrosis observed. Monitoring at the site where the disease was first noted showed new gorgonian recruitment from 2003 to 2006; some individuals had died and become completely overgrown, whereas others had continued to grow around a dead central area.

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Shields, J.D., Taylor, D.M., O'Keefe, P.G., Colbourne, E., and Hynick, E. **Epidemiological determinants in outbreaks of bitter crab disease (*Hematodinium* sp.) in snow crabs *Chionoecetes opilio* from Conception Bay, Newfoundland, Canada.** *Diseases of Aquatic Organisms* 77(1): 61-72, 2007.

**Notes:** Bitter crab disease (BCD) is caused by *Hematodinium* sp., an endoparasitic dinoflagellate. It lives within the hemocoeloms of snow crabs *Chionoecetes opilio* and Tanner crabs *C. bairdi*, making them unmarketable due to their bitter flavor. Two recent outbreaks of BCD have occurred in Conception Bay, Newfoundland, one from 1999 to 2000 and another from 2003 to 2005. In the earlier outbreak, prevalence was highest in juvenile and primiparous females and juvenile males. It was thought to be highest in these hosts because they molt more frequently than larger males and the disease is transmitted to newly molted crabs. In the 2003 to 2005 outbreak, the prevalence of BCD changed and was at its highest, 24 % in trapped males and 13.5 % in trawled males. This apparent shift in the dynamics of the infection between the earlier 1999 to 2000 and later 2003 to 2005 outbreaks was highly correlated with 2 factors: an increase in bottom temperatures, associated with the recent climatic warming trend in the Northwest Atlantic, and an increase in molting activity of the snow crabs due presumably to the temperature increase within Conception Bay. That is, rising temperatures occurring from 2003 to 2005 likely stimulated molting activity in snow crabs, which led to an increase in susceptible hosts in the population. Given the positive correlation between increased bottom temperature, increased molting activity, and the latest outbreak of BCD, we predict that further trends in climatic warming will enhance transmission, spreading the parasite into additional fishing areas.

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Sterud, E., Forseth, T., Ugedal, O., Poppe, T.T., Jorgensen, A., Bruheim, T., Fjeldstad, H.P., and Mo, T.A. **Severe mortality in wild Atlantic salmon *Salmo salar* due to proliferative kidney disease (PKD) caused by *Tetracapsuloides bryosalmonae* (Myxozoa).** *Diseases of Aquatic Organisms* 77(3): 191-198, 2007.

**Notes:** Extensive mortality in Atlantic salmon fry was reported in the River Aelva from 2002 to 2004. Dead fish were collected in late summer 2006, and live fish were sampled by electrofishing in September the same year. At autopsy and in histological sections, the fish kidneys were found to be pale and considerably enlarged. Proliferative lesions with characteristic PKX cells were seen in a majority of the fish. DNA from kidney samples of diseased fish was subjected to PCR and sequencing, and the amplified sequences matched those of *Tetracapsuloides bryosalmonae*. We concluded that this myxozoan transmitted from bryozoans was the main cause of the observed mortality in salmon fry in 2006. Results from quantitative electrofishing in 2005 and 2006, combined with the observed fry mortality from 2002 to 2004, show that the smolt production in the river is severely reduced and that *T. bryosalmonae* is the most likely explanation for this decline. The present study is the first to report a considerable negative population effect in wild Atlantic salmon due to proliferative kidney disease (PKD). It also represents the northernmost PKD outbreak in wild fish. The river is regulated for hydroelectric power purposes, causing reduced water flow and elevated summer temperatures, and the present PKD outbreak may serve as an example of increased disease vulnerability of northern fish populations in a warmer climate.

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Toplu, N., Aydogan, A., and Oguzoglu, T.C. **Visceral leishmaniosis and parapoxvirus infection in a Mediterranean monk seal (*Monachus monachus*).** *Journal of Comparative Pathology* 136(4): 283-287, 2007.

**Notes:** A Mediterranean monk seal was shown by immunohistochemical and polymerase chain reaction techniques to be dually infected with a *Leishmania* sp. and parapoxvirus. The pathological findings included a deep ulcer on the side of the head, ulcers on the gingival and inner aspect of the lower lip, enlarged lymph nodes and tonsils, and respiratory lesions (pulmonary consolidation, oedema, haemorrhages and emphysema; tracheal and bronchial congestion, exudates and haemorrhage).

Amastigotes were demonstrated in macrophages in the lymph nodes and spleen, and intracytoplasmic inclusion bodies were observed in the tracheal and oral mucosa.

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Siebert, U., Wohlsein, P., Lehnert, K., and Baumgartner, W. **Pathological findings in harbour seals (*Phoca vitulina*): 1996-2005.** *Journal of Comparative Pathology* 137(1): 47-58, 2007.

**Notes:** Between 1996 and 2005 the carcasses of 355 harbour seals originating from the coast of Schleswig-Holstein, Germany, were investigated for pathological changes. The animals were collected before ( $n = 280$ ) and after ( $n = 75$ ) the second phocine distemper virus (PDV) epizootic in 2002. The seals were either found dead or were killed due to severe illness. Necropsy was performed in each case, in addition to histopathological, immunohistochemical, microbiological and parasitological examinations. Throughout the period of study, the respiratory and alimentary tracts were the organ systems most consistently affected by pathological change. The most common cause of death was bronchopneumonia caused by parasitic and/or bacterial infection of the lung. Less frequently identified changes included: trauma, gastroenteritis, uterine torsion or dystocia, polyarthritis/polymyositis, intestinal torsion, septicaemia, dermatitis, and keratitis. The most frequent causes of bronchopneumonia, gastroenteritis, polyarthritis, dermatitis and septicaemia were infections with a/b-haemolytic streptococci, *Escherichia coli* and *Clostridium perfringens*. A number of changes were more frequently identified after 2002. These included the presence of parasites in the lung, stomach and intestine; bronchopneumonia, gastritis, enteritis, septicaemia and perinatal death. The increased prevalence of these changes may have been related to the preceding PDV epidemic.

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Thomas, N.J., Dubey, J.P., Lindsay, D.S., Cole, R.A., and Meteyer, C.U. **Protozoal meningoencephalitis in sea otters (*Enhydra lutris*): a histopathological and immunohistochemical study of naturally occurring cases.** *Journal of Comparative Pathology* 137(2-3): 102-121, 2007.

**Notes:** Protozoal meningoencephalitis is considered to be all important cause of mortality, in the California sea otter (*Enhydra lutris*). Thirty nine of 344 (11.3%) California (CA) and Washington state (WA) sea otters examined from 1985 to 2004 had histopathological evidence of significant protozoal meningoencephalitis. The aetiological agents and histopathological changes associated with these protozoal infections are described. The morphology of the actively multiplicative life stages of the organisms (tachyzoites for *Toxoplasma gondii* and merozoites for *Sarcocystis neurona*) and immunohistochemical labelling were used to identify infection with *S. neurona* ( $n = 22$ , 56.4%) *T. gondii* ( $n = 57$ , 12.8%) or dual infection with both organisms ( $n = 12$ , 30.8%). Active *S. neurona* was present in all dual infections while most had only the latent form of *T. gondii*. In *S. neurona* meningoencephalitis, multifocal to diffuse gliosis was widespread in grey matter and consistently present in the molecular layer of the cerebellum. In *T. gondii* meningoencephalitis, discrete foci of gliosis and malacia were more widely separated, sometimes incorporated pigment-laden macrophages and mineral, and were found predominantly in the cerebral cortex. Quiescent tissue cysts of *T. gondii* were considered to be incidental and not a cause of clinical disease and mortality. Protozoal meningoencephalitis was diagnosed more frequently in the expanding population of WA sea otters (10 of 31, 32.3%) than in the declining CA population (29 of 313, 9.3%). Among sea otters with protozoal meningoencephalitis, those that had displayed neurological signs prior to death had active *S. neurona* encephalitis, supporting the conclusion that *S. neurona* is the most significant protozoal pathogen in the central nervous system of sea otters.