

### In this review:

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

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Bossart, G.D. **Marine mammals as sentinel species for oceans and human health.** *Oceanography* 19(2): 134-137, 2006.

## B. Recent articles with abstracts

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Mydlarz, L.D., Jones, L.E., and Harvell, C.D. **Innate immunity environmental drivers and disease ecology of marine and freshwater invertebrates.** *Annual Review of Ecology, Evolution, and Systematics* 37: 251-288, 2006.

**Notes:** Despite progress in the past decade, researchers struggle to evaluate the hypothesis that environmental conditions compromise immunity and facilitate new disease outbreaks. In this chapter, we review known immunological mechanisms for selected phyla and find that there are critical response pathways common to all invertebrates. These include the prophenoloxidase pathway, wandering phagocytic cells, cytotoxic effector responses, and antimicrobial compounds. To demonstrate the links between immunity and the environment, we summarize mechanisms by which immunity is compromised by environmental conditions. New environmental challenges may promote emergent disease both through compromised host immunity and introduction of new pathogens. Such challenges include changing climate, polluted environment, anthropogenically facilitated pathogen invasion, and an increase in aquaculture. The consequences of these environmental issues already manifest themselves as increased mortality on coral reefs, pathogen range expansion, and transmission of disease from aquaculture to natural populations, as we summarize in a final section on recent marine epizootics.

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Cerrano, C., Totti, C., Sponga, F., and Bavestrello, G. **Summer disease in *Parazoanthus axinellae* (Schmidt, 1862) (Cnidaria, Zoanthidea).** *Italian Journal of Zoology* 73(4): 355-361, 2006.

**Notes:** Climate change is affecting marine environments all over the world but scientists' attention is mainly devoted to tropical areas. In the Mediterranean Sea, species with a cold affinity are decreasing very fast from 0 to 40 m depth, while warm water species increase. From 2000, several populations of the zoanthid *Parazoanthus axinellae* (Schmidt 1882) have been showing signs of suffering along the Ligurian coast. Here we report a three-year monitoring, from June 2001 to September 2003, of a population of *P. axinellae* on the rocky cliff of the Portofino Promontory (Ligurian Sea). During this span of time the population, which covered an area of several square metres with a density of about 1 polyp cm<sup>-2</sup>, was severely reduced. In the meanwhile an encrusting sponge, *Crambe crambe*, rapidly colonized the free substrates abandoned by the zoanthid. Warm water and the massive proliferation of a cyanobacterium attributed to the genus *Porphyrosiphon* are hypothesized to be the main causes of this disease.

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Kane, A.S., Stine, C.B., Hungerford, L., Matsche, M., Driscoll, C., and Baya, A.M. **Mycobacteria as environmental portent in Chesapeake Bay fish species.** *Emerging Infectious Diseases* 13(2): 329-331, 2007.

**Notes:** Infection with environmental mycobacteria is increasing among many Chesapeake Bay fish species. Prevalence in juvenile Atlantic menhaden differed between tributaries and ranged from 2% to 57%. Mycobacterial infection may be a syndromic sentinel of altered environmental conditions that threaten aquatic animal health.

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Beineke, A., Siebert, U., Muller, G., and Baumgartner, W. **Increased blood interleukin-10 mRNA levels in diseased free-ranging harbor porpoises (*Phocoena phocoena*).** *Veterinary Immunology and Immunopathology* 115(1-2): 100-106, 2007.

**Notes:** Harbor porpoises from the North and Baltic Seas exhibit a higher incidence of bacterial infections compared to whales from less polluted arctic waters. Toxicological analysis revealed an association between elevated body burdens of environmental contaminants, such as polychlorinated biphenyls (PCB) and polybrominated diphenyl ether (PBDE) and lymphoid depletion in thymus and spleen of these whales. However, it remains undetermined if changes in the immune system are primarily contaminant-induced or a sequel of infectious diseases and emaciation. The aim of the present study was to investigate changes of blood cytokine mRNA levels in healthy and diseased harbor porpoises. Therefore, 29 by-caught and stranded whales were necropsied and the health status was evaluated based upon main pathological findings. Furthermore, the degree of thymic atrophy and splenic depletion was histologically graded using a semi-quantitative scoring system. Gene expression of interleukin (IL)-2, IL-4, IL-6, IL-10, transforming growth factor- $\beta$  and tumor necrosis factor- $\alpha$  in the blood was measured by real time reverse transcription-polymerase chain reaction. Thymic atrophy and splenic depletion were correlated with an impairment of the animals' health status. Additionally, a marked up-regulation of IL-10 was predominately found in severely diseased whales with evidence of chronic bacterial infections. Furthermore, increased IL-10 levels were associated with splenic depletion. Other investigated cytokines were not significantly associated with the health status or lymphoid depletion, respectively. The present study indicated that lymphoid depletion represents a sequel of chronic infectious diseases in a portion of investigated harbor porpoises. Regarding this, expression of anti-inflammatory cytokines, such as IL-10 might represent a consequence of continuous stimulation of the immune system and induction of immunomodulatory mechanisms in this cetacean species.

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Bruno, J.F., Selig, E.R., Casey, K.S., Page, C.A., Willis, B.L., Harvell, C.D., Sweatman, H., and Melendy, A.M. **Thermal stress and coral cover as drivers of coral disease outbreaks.** *PLoS Biology* 5(5): art. e124, 2007.

**Notes:** Very little is known about how environmental changes such as increasing temperature affect disease dynamics in the ocean, especially at large spatial scales. We asked whether the frequency of warm temperature anomalies is positively related to the frequency of coral disease across 1,500 km of Australia's Great Barrier Reef. We used a new high resolution satellite dataset of ocean temperature and 6 y of coral disease and coral cover data from annual surveys of 48 reefs to answer this question. We found a highly significant relationship between the frequencies of warm temperature anomalies and of white syndrome, an emergent disease, or potentially, a group of diseases, of Pacific reef-building corals. The effect of temperature was highly dependent on coral cover because white syndrome outbreaks followed warm years, but only on high (>50%) cover reefs, suggesting an important role of host density as a threshold for outbreaks. Our results indicate that the frequency of temperature anomalies, which is predicted to increase in most tropical oceans, can increase the susceptibility of corals to disease, leading to outbreaks where corals are abundant.

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Romalde, J.L., Vilarino, M.L., Beaz, R., Rodriguez, J.M., Diaz, S., Villalba, A., and Carballal, M.J. **Evidence of retroviral etiology for disseminated neoplasia in cockles (*Cerastoderma edule*).** *Journal of Invertebrate Pathology* 94(2): 95-101, 2007.

**Notes:** Epizootiologic outbreaks of disseminated neoplasia have been reported in association with massive mortalities of various bivalve species. In cockles, *Cerastoderma edule*, this pathological condition was described in Ireland and France. Since 1997, different populations affected by this pathology have been detected in Galicia (NW Spain). Transmission electron microscopy allowed the visualization of virus-like particles in neoplastic cells, resembling a retrovirus-like agent. To confirm this hypothesis, we used a commercial kit for detection and quantification of reverse transcriptase (RT) activity, based on the use of bromo-deoxyuridine triphosphate (BrdUTP) and a BrdU binding antibody conjugated to alkaline phosphatase. In

addition, we developed a product-enhanced RT assay using RNA of hepatitis A virus as a template. These two assays showed positive RT activity in 90.9 and 81.8% of samples, respectively, from cockles displaying disseminated neoplasia as determined by light microscopy. These results strongly support the hypothesis of retroviral etiology for this pathological condition.

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Nievas, V.F., Leotta, G.A., and Vigo, G.B. **Subcutaneous clostridial infection in Adelie penguins in Hope Bay, Antarctica.** *Polar Biology* 30(2): 249-252, 2007.

**Notes:** During the 2000-2001 breeding season in Hope Bay, Antarctica, two adult Adelie penguins were found dead with lesions compatible with subcutaneous clostridial infection. *Clostridium cadaveris* was isolated from the musculature and the subcutaneous tissue of one of these two penguins, whereas *Clostridium sporogenes* was isolated from the subcutaneous tissue of the other penguin. *Escherichia coli* and *Staphylococcus* spp. were isolated from both animals. This is the first report of subcutaneous clostridial infection in Antarctic Adelie penguins.

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Goldstein, T., Lowenstine, L.J., Lipscomb, T.P., Mazet, J.A.K., Novak, J., Stott, J.L., and Gulland, F.M.D. **Infection with a novel gammaherpesvirus in northern elephant seals (*Mirounga angustirostris*).** *Journal of Wildlife Diseases* 42(4): 830-835, 2006.

**Notes:** Twenty juvenile northern elephant seals (*Mirounga angustirostris*) that died between 1998 and 2004 had ulcers on the tongue, palatine mucosa, and/or tonsils. Histologic examination of the lesions revealed cytoplasmic swelling, nuclear pyknosis, and eosinophilic to amphophilic intranuclear inclusions bodies suggestive of herpesviral infection. Electron microscopic examination and polymerase chain reaction analysis confirmed the presence of a herpesvirus. Subsequent DNA sequencing identified this to be a new gammaherpesvirus that was similar to Porcine lymphotropic virus 2, Alcephaline herpesvirus 1 (malignant catarrhal fever virus from wildebeest), and Chlorocebus rhadinovirus 1. from African green monkeys. Identical herpesviral DNA was also detected in blood and mucosal swabs collected from five healthy elephant seal pups.

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Fujii, K., Sato, H., Kakumoto, C., Kobayashi, M., Saito, S., Kariya, T., Watanabe, Y., Sakoda, Y., Kai, C., Kida, H., and Suzuki, M. **Seroepidemiological survey of morbillivirus infection in Kuril harbor seals (*Phoca vitulina stejnegeri*) of Hokkaido, Japan.** *Japanese Journal of Veterinary Research* 54(2-3): 109-117, 2006.

**Notes:** Serological analysis was performed to detect morbillivirus infection in Kuril harbor seals in Hokkaido, Japan. Serum samples were collected from the seals at Nosappu (231 sera), Akkeshi (16) and Erimo (75) between 1998 and 2005. Antibodies to phocine distemper virus (PDV) were detected by ELISA in seals from Nosappu and Erimo. Antibodies to PDV were found in 56% (5/9) of the sampled seals from Nosappu in 1998 versus only 5% (3/66) in 2003, 1% (1/79) for 2004 and 1% (1/77) for 2005. This suggests an epidemic caused by the virus in or before 1998. As antibody-positive seals included juvenile seals in 2003 and 2005 sporadic infections of the virus are thought to have occurred in recent years. In Erimo, antibodies to PDV were found in 50% (14/28) of sampled seals in 2004 versus only 13% (1/8) for 1999, 7% (1/15) for 2003, and 0% (0/24) for 2005. These suggest sporadic infection by the virus before 2003 and the epizootic between autumn in 2003 when samples of 2003 were collected, and 2004. Since antibodies to canine distemper virus were detected in one adult seal from Nosappu in each year from 2003 to 2005 sporadic infections of the virus were suggested. There were no difference in incidence of seals with antibodies to the viruses between males and females and between juveniles and adults.

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Buckles, E.L., Lowenstine, L.J., Funke, C., Vittore, R.K., Wong, H.N., StLeger, J.A., Greig, D.J., Duerr, R.S., Gulland, F.M.D., and Stott, J.L. **Otarine herpesvirus-1, not papillomavirus, is associated with endemic tumours in California sea lions (*Zalophus californianus*).** *Journal of Comparative Pathology* 135(4): 183-189, 2006.

**Notes:** The purpose of this study was to determine if Otarine Herpesvirus-1 (OthV-1) is associated with the presence of urogenital carcinomas in California sea lions. Polymerase chain reaction (PCR) analysis with primers specific for OthV-1 was used to compare the prevalence of OthV-1 infection in 15 sea lions affected by urogenital carcinoma with that of age-matched and juvenile tumour-free animals, and animals with tumours of non-urogenital origin. The herpesvirus was more

prevalent (100%) and more widespread in the 15 animals with urogenital carcinoma than in 25 control animals, and was most often found in the urogenital tissue (vagina and prostate) and in the draining lymph nodes. Moreover, OtHV-1 DNA was not found in any juvenile animal, or in the neoplastic tissues of animals with non-urogenital tumours. Papillomavirus-specific PCR analysis of urogenital carcinoma tissues detected papillomavirus sequences in only one carcinomatous tissue. Further studies are needed to determine if OtHV-1 contributes to oncogenesis in the California sea lion; these data show, however, that OtHV-1 is associated with urogenital carcinomas, is preferentially present in urogenital tissues, and may be sexually transmitted. Papillomaviruses, which are known to contribute to urogenital tumours in other species, did not appear to be associated with the sea lion carcinomas.

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Ramaiah, N. **A review on fungal diseases of algae, marine fishes, shrimps and corals.** *Indian Journal of Marine Science* 35(4): 380-387, 2006.

**Notes:** It is a well-known fact that diseases affect health, survival and recruitment of any individual susceptible for diseases. As a consequence of disease, harvests from natural resources and, in particular, those from aquaculture dwindle quite severely. While an appreciable volume of information on variety of mycotic diseases in the marine organisms is available on global scale, studies from Indian waters are, at best, very few. This review is an attempt of bringing together a set of information deemed useful for stimulating marine mycopathological investigations in our waters. The information put together here is also to highlight the importance of pathology in general and fungal diseases in particular.

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Coral Disease Working Group of the Global Environmental Facility Coral Reef Targeted Research Program. **Coral disease, environmental drivers, and the balance between coral and microbial associates.** *Oceanography* 20(1): 172-195, 2007.

**Notes:** Across the globe, we are witnessing the decline of coral reef ecosystems. One relatively new factor contributing to this decline is the outbreak of destructive infectious diseases, especially on Caribbean reefs. As the Coral Disease Working Group of the Coral Reef Targeted Research Program, our research focuses on four priorities: (1) assessing the global prevalence of coral disease, (2) investigating the environmental drivers of disease, (3) identifying the pathogens that cause disease, and (4) evaluating the coral's ability to resist disease. Monitoring has revealed new coral-disease syndromes at each of four Global Environmental Fund Centers of Excellence: the Caribbean, the Philippines, Australia, and East Africa. Over the last 20 years, drastic (> 50 percent) loss of coral cover has occurred on the Yucatán Peninsula, even in pristine areas. Global surveys have revealed significant levels of disease and disease outbreaks occurring not only in the Caribbean "hotspots," but also in sites throughout the Pacific and Indian Oceans. By monitoring coral disease, we will create a baseline and long-term data set that can be used to test specific hypotheses about how climate and anthropogenic drivers, such as decreasing water quality, threaten coral reef sustainability. One such hypothesis is that high-temperature anomalies drive outbreaks of disease by hindering the coral's ability to fight infection and by increasing the pathogens' virulence. We observed recurrent outbreaks following the warm summer months of two of the most damaging diseases in the Caribbean. In addition, we found that coral disease in the Great Barrier Reef correlated with warm temperature anomalies. In the Caribbean and Mediterranean Seas, virulence of known coral pathogens and the normal coral flora changed during high-temperature periods. Other stresses such as high nutrients and sedimentation may similarly alter the balance between the coral and its resident microbial flora.

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Lester, S.E., Tobin, E.D., and Behrens, M.D. **Disease dynamics and the potential role of thermal stress in the sea urchin, *Strongylocentrotus purpuratus*.** *Canadian Journal of Fisheries and Aquatic Sciences* 64(2): 314-323, 2007.

**Notes:** Evidence from field and laboratory data indicates that warmer ocean temperatures likely play a critical role in the disease dynamics of intertidal populations of the sea urchin, *Strongylocentrotus purpuratus*. Urchin populations along the west coast of North America have experienced numerous disease epidemics in the past several decades, and yet little is known about disease transmission, the geographic extent, or contributing factors to these outbreaks. In this study, we examine disease in Pacific *Strongylocentrotus* urchins over a broad geographic range with repeated sampling. We suggest that what has been assumed to be a single disease might be two distinct diseases presenting two disparate pathologies. Both potential pathologies were extremely rare or absent north of Point Conception, California, in a region associated with strong upwelling and cooler water temperatures but were common at warmer sites in southern California and northern Baja California, Mexico. Furthermore, during the survey period, disease prevalence at some of the study sites was positively correlated with sea surface

temperatures as estimated from satellite data, leading us to hypothesize that heat stress may increase urchin susceptibility to disease. In experimentally elevated water temperatures, diseased individuals had significantly larger lesions and a significantly lower gonadal index, which could have important implications for urchin population dynamics.

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Webster, N.S. **Sponge disease: a global threat?** *Environmental Microbiology* 9(6): 1363-1375, 2007.

**Notes:** Sponges are the most simple and primitive metazoans, yet they have various biological and ecological properties that make them an influential component of coral-reef ecosystems. Marine sponges provide refuge for many small invertebrates and are critical to benthic-pelagic coupling across a wide range of habitats. Reports of sponge disease have increased dramatically in recent years with sponge populations decimated throughout the Mediterranean and Caribbean. Reports also suggest an increased prevalence of sponge disease in Papua New Guinea, the Great Barrier Reef and in the reefs of Cozumel, Mexico. These epidemics can have severe impacts on the survival of sponge populations, the ecology of the reef and the fate of associated marine invertebrates. Despite the ecological and commercial importance of sponges, the understanding of sponge disease is limited. There has generally been a failure to isolate and identify the causative agents of sponge disease, with only one case confirming Koch's postulates and identifying a novel *Alphaproteobacteria* strain as the primary pathogen. Other potential disease agents include fungi, viruses, cyanobacteria and bacterial strains within the *Bacillus* and *Pseudomonas* genera. There is some evidence for correlations between sponge disease and environmental factors such as climate change and urban/agricultural runoff. This review summarizes the occurrence of sponge disease, describes the syndromes identified thus far, explores potential linkages with environmental change and proposes a strategy for future research towards better management of sponge disease outbreaks.

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Ainsworth, T.D., Kramasky-Winter, E., Loya, Y., Hoegh-Guldberg, O., and Fine, M. **Coral disease diagnostics: What's between a plague and a band?** *Applied and Environmental Microbiology* 73(3): 981-992, 2007.

**Notes:** Recently, reports of coral disease have increased significantly across the world's tropical oceans. Despite increasing efforts to understand the changing incidence of coral disease, very few primary pathogens have been identified, and most studies remain dependent on the external appearance of corals for diagnosis. Given this situation, our current understanding of coral disease and the progression and underlying causes thereof is very limited. In the present study, we use structural and microbial studies to differentiate different forms of black band disease: atypical black band disease and typical black band disease. Atypical black band diseased corals were infected with the black band disease microbial consortium yet did not show any of the typical external signs of black band disease based on macroscopic observations. In previous studies, these examples, here referred to as atypical black band disease, would have not been correctly diagnosed. We also differentiate white syndrome from white diseases on the basis of tissue structure and the presence/absence of microbial associates. White diseases are those with dense bacterial communities associated with lesions of symbiont loss and/or extensive necrosis of tissues, while white syndromes are characteristically bacterium free, with evidence for extensive programmed cell death/apoptosis associated with the lesion and the adjacent tissues. The pathology of coral disease as a whole requires further investigation. This study emphasizes the importance of going beyond the external macroscopic signs of coral disease for accurate disease diagnosis.

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Efrony, R., Loya, Y., Bacharach, E., and Rosenberg, E. **Phage therapy of coral disease.** *Coral Reefs* 26(1): 7-13, 2007.

**Notes:** At present there are no known procedures for preventing or treating infectious diseases of corals. Toward this end, the use of phage therapy has been investigated. Lytic bacteriophages (phages) were isolated for two bacterial pathogens that are responsible for coral diseases, *Vibrio coralliilyticus*, which is the causative agent of bleaching and tissue lysis of *Pocillopora damicornis*, and *Thalassomonas loyaeana*, which causes the white plague-like disease of *Favia fava*. By using these phages in controlled aquaria experiments, it was demonstrated that each of these diseases could be controlled by the pathogen-specific phage. The data indicate that initially the phages bind to the pathogen in seawater and are then brought to the coral surface where they multiply and lyse the pathogen. The phages remained associated with the coral and could prevent subsequent infections. These data suggest that phage therapy has the potential to control the spread of infectious coral diseases.

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Miller, M.W. and Williams, D.E. **Coral disease outbreak at Navassa, a remote Caribbean island.** *Coral Reefs* 26(1): 97-101, 2007.

**Notes:** In November 2004, a high prevalence of coral disease was observed at several sites around Navassa, an uninhabited Caribbean island between Haiti and Jamaica. At least fifteen mounding and foliaceous scleractinian species were affected with 'white disease' signs. Coral disease incidence was observed to be absent in quantitative surveys in 2002, but in 2004 average prevalence (i.e., % of colonies) of active disease ranged up to 15% and an additional 19% prevalence of colonies with patterns of recent mortality consistent with disease. Large and/or *Montastraea* spp. colonies were disproportionately affected and the anticipated loss of these large, reef-building colonies will impact coral community structure. One or more potential factors may influence the initiation and persistence of disease outbreak conditions at Navassa including recent hurricane disturbance, regional patterns of increasing disease impact in deep or remote Caribbean reefs, or vectoring of disease by the corallivorous worm, *Hermodice carunculata*.

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Littnan, C.L., Stewart, B.S., Yochem, P.K., and Braun, R. **Survey for selected pathogens and evaluation of disease risk factors for endangered Hawaiian monk seals in the main Hawaiian Islands.** *EcoHealth* 3(4): 232-244, 2006.

**Notes:** A recently reestablished and increasing population of Hawaiian monk seals in the main Hawaiian Islands (MHI) is encouraging for this endangered species. However, seals in the MHI may be exposed to a broad range of human, pet, livestock, and feral animal pathogens. Our objective was to determine the movement and foraging habitats of Hawaiian monk seals in the MHI relative to the potential exposure of seals to infectious diseases in near-shore marine habitats. We captured 18 monk seals in the MHI between January 27, 2004 and November 29, 2005, tested them for various infectious diseases, and then monitored the foraging movements of 11 of them using satellite-linked radio transmitters for the next 32-167 days. All seals tested negative for canine adenovirus, calicivirus, four morbilliviruses, phocine herpes virus, *Leptospira* sp., and feline and canine heartworm antigen/antibody. Six of the seals tested positive on complement fixation for *Chlamydia abortus* (formerly *Chlamydia psittaci*). Four seals demonstrated positive titers to *Sarcocystis neurona*, two to *Neospora caninum*, and two to *Toxoplasma gondii*. Fecal cultures showed approximately half ( $n = 6$ ) positive for *E. coli* 0157, no *Salmonella* sp., and only one with *Campylobacter* sp. Satellite monitored seals spent considerable time foraging, traveling, and resting in neritic waters close to human population centers, agricultural activity, and livestock ranges, and sources of land-based water runoff and sewage dispersal. Consequently, Hawaiian monk seals in the MHI may be at risk of exposure to several infectious disease agents associated with terrestrial animals that can contaminate marine habitats from runoff along drainages and that are known to cause disease in marine mammals. Further, some seals overlapped substantially in their use of coastal habitats and several moved among islands while foraging and were seen on beaches near each other. This suggests that diseased seals could infect healthy conspecifics throughout the MHI.

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Despommier, D., Ellis, B.R., and Wilcox, B.A. **The role of ecotones in emerging infectious diseases.** *EcoHealth* 3(4): 281-289, 2006.

**Notes:** Recognition of the significance of the boundary between ecological systems, often referred to as the ecotone, has a long history in the ecological sciences and in zoonotic disease research. More recent research in landscape ecology has produced an expanded view of ecotones and elaboration of their characteristics and functions in ecosystems. Parallel research on emerging infectious diseases (EIDs) and the causes of increased rates of pathogen transmission, spread, and adaptation suggests a correspondence between ecotonal processes and the ecological and evolutionary processes responsible for zoonotic and vector-borne emerging infections. A review of the literature suggests that ecotones play a role in a number of the most important EIDs. Yet these are the only diseases for which specific landscape ecological information exists in the literature or disease reports. However, the similar disease ecologies of these with about half of the approximately 130 zoonotic EIDs suggests ecotones, particularly their anthropogenic origination or modification, may be generally associated with ecotones and the global trend of increasing EIDs.

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Ford, S.E. and Smolowitz, R. **Infection dynamics of an oyster parasite in its newly expanded range.** *Marine Biology* 151(1): 119-133, 2007.

**Notes:** Over a 2-year period in 1990 and 1991, coincident with a pronounced warming episode, Dermo disease outbreaks in the oyster, *Crassostrea virginica*, caused by the parasite *Perkinsus marinus*, occurred over a 500-km range from Delaware Bay to Cape Cod, in the northeastern United States. The parasite had not previously been recorded or known to cause mortalities in this region. To document infection patterns and levels in this region several years after the initial outbreaks, and to compare them with those in the parasite's historic southern range, we deployed and sampled oysters from 1996 to 1998 at multiple sites spanning the expanded range. During this 2-year period, the parasite was documented to occur in oysters at high prevalences throughout the new range, in sites varying from small, enclosed embayments to large estuaries, and in both cultured and wild-set oysters. Infection and mortality patterns, and levels were similar to those in southern locations where the parasite has been enzootic for at least decades. The persistence of high *P. marinus* infection levels in the new range after the initial expansion is probably due to several factors: (1) winter temperatures continued to increase during the 1990s and early 2000s, albeit at a slower rate than in 1990-1991, facilitating overwinter survival of the parasite; (2) many oyster-growing sites in the northeast are in relatively shallow water in which summer temperatures offer ample time for the parasite to proliferate and spread; and (3) the combination of high parasite burdens and high host densities in oyster farms results in an abundance of parasites and high transmission rates. Colder winters and high rainfall after 2002 reduced prevalences in some regions, but *P. marinus* can survive low temperatures and low salinities, and epizootic conditions are likely to return if temperatures rise again, as predicted by climate-change models.

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Ainsworth, T.D., Kvennefors, E.C., Blackall, L.L., Fine, M., and Hoegh-Guldberg, O. **Disease and cell death in white syndrome of Acroporid corals on the Great Barrier Reef.** *Marine Biology* 151(1): 19-29, 2007.

**Notes:** White syndromes (WS) are among the most prevalent coral diseases, and are responsible for reef demise on the Great Barrier Reef. The disease manifests as a clear differentiation between tissue and exposed skeleton and results in rapid tissue loss. Fluorescence in situ hybridisation (FISH) was used in conjunction with histology and transmission electron microscopy (TEM) to investigate bacterial communities and cell death associated with WS. No evidence of bacterial communities or microbial association (using six bacterial probes, TEM and histopathology) was evident within the lesion or adjacent tissues, despite the presence of dense possible secondary invaders in the exposed skeletal regions. Despite widespread reference to necrosis in coral disease literature, there was no evidence of necrosis in any WS lesion or the adjacent tissues in this study. However, in situ end labelling, light microscopy and TEM of WS and healthy coral tissue sections showed evidence of extensive programmed cell death (PCD) exclusively in WS. This study provides the first evidence of intrinsic or PCD as a primary mechanism of cell death in WS, and may provide some explanation for the failure to isolate pathogens from over 80% of identified coral diseases, many of which show similar lesion patterns and WS characteristics.

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Jacobson, E.R., Homer, B.L., Stacy, B.A., Greiner, E.C., Szabo, N.J., Chrisman, C.L., Origgi, F., Coberley, S., Foley, A.M., Landsberg, J.H., Flewelling, L., Ewing, R.Y., Moretti, R., Schaf, S., Rose, C., Mader, D.R., Harman, G.R., Manire, C.A., Mettee, N.S., Mizisin, A.P., and Shelton, G.D. **Neurological disease in wild loggerhead sea turtles *Caretta caretta*.** *Diseases of Aquatic Organisms* 70(1-2): 139-154, 2006.

**Notes:** Beginning in October 2000, subadult loggerhead sea turtles *Caretta caretta* showing clinical signs of a neurological disorder were found in waters off south Florida, USA. Histopathology indicated generalized and neurologic spirorchidiasis. In loggerhead sea turtles (LST) with neurospirorchidiasis, adult trematodes were found in the meninges of the brain and spinal cord of 7 and 3 affected turtles respectively, and multiple encephalic intravascular or perivascular eggs were associated with granulomatous or mixed leukocytic inflammation, vasculitis, edema, axonal degeneration and occasional necrosis. Adult spirorchids were dissected from meningeal vessels of 2 of 11 LST brains and 1 of 10 spinal cords and were identified as *Neosporichis* sp. Affected LST were evaluated for brevetoxins, ciguatoxins, saxitoxins, domoic acid and palytoxin. While tissues from 7 of 20 LST tested positive for brevetoxins, the levels were not considered to be in a range causing acute toxicosis. No known natural (algal blooms) or anthropogenic (pollutant spills) stressors co-occurred with the turtle mortality. While heavy metal toxicosis and organophosphate toxicosis were also investigated as possible causes, there was no evidence for their involvement. We speculate that the clinical signs and pathologic changes seen in the affected LST resulted from combined heavy spirorchid parasitism and possible chronic exposure to a novel toxin present in the diet of LST.

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Tops, S., Lockwood, W., and Okamura, B. **Temperature-driven proliferation of *Tetracapsuloides bryosalmonae* in bryozoan hosts portends salmonid declines.** *Diseases of Aquatic Organisms* 70(3): 227-236, 2006.

**Notes:** Proliferative kidney disease (PKD) is an emerging disease of salmonid fishes. It is provoked by temperature and caused by infective spores of the myxozoan parasite *Tetracapsuloides bryosalmonae*, which develops in freshwater bryozoans. We investigated the link between PKD and temperature by determining whether temperature influences the proliferation of *T. bryosalmonae* in the bryozoan host *Fredericella sultana*. Herein we show that increased temperatures drive the proliferation of *T. bryosalmonae* in bryozoans by provoking, accelerating and prolonging the production of infective spores from cryptic stages. Based on these results we predict that PKD outbreaks will increase further in magnitude and severity in wild and farmed salmonids as a result of climate-driven enhanced proliferation in invertebrate hosts, and urge for early implementation of management strategies to reduce future salmonid declines.

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Coma, R., Linares, C., Ribes, M., Diaz, D., Garrabou, J., and Ballesteros, E. **Consequences of a mass mortality in populations of *Eunicella singularis* (Cnidaria: Octocorallia) in Menorca (NW Mediterranean).** *Marine Ecology Progress Series* 327: 51-60, 2006.

**Notes:** At the end of the boreal summer of 1999, many invertebrates in hard-bottom communities in the NW Mediterranean Sea suffered an episode of mass mortality. Our study examined the effects of this event on populations of the temperate octocoral *Eunicella singularis* at Menorca (Balearic Islands). The event affected colonies over the entire depth range where the species is present (15 to 40 m). Four years after the occurrence of the event, 59 % of the colonies still exhibited some damage. The extent of injury of the colonies varied among locations, ranging between 37 and 67 % with a mean of 50 % of the colony surface. The proportion of dead colonies also varied among locations from 26 to 62 %, with an overall mean of 46 % of the population. The proportion of dead colonies was inversely correlated with density. Current size distribution of the colonies indicated the occurrence of low recruitment during the years subsequent to the mass mortality event. Our study demonstrates that the 1999 mass mortality event was geographically more extensive than previously thought (Menorca is located about 400 to 700 km from areas where mass mortality was previously reported, along the coast of Provence and Liguria). The mortality rate of *E. singularis* estimated for the Menorca populations was the highest value recorded to date for this species, showing that the delayed effects of the mortality event exerted a much greater impact on these populations than the immediate effects. Additional impacts result from a reduction in recruitment. These delayed consequences may lead to important changes in community composition, structure, processes and function, as gorgonians are key structural and functional organisms within these communities.

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Miner, C.M., Altstatt, J.M., Raimondi, P.T., and Minchinton, T.E. **Recruitment failure and shifts in community structure following mass mortality limit recovery prospects of black abalone.** *Marine Ecology Progress Series* 327: 107-117, 2006.

**Notes:** Mass mortalities of species can fundamentally alter the structure of natural communities, which can in turn negatively impact species' recovery. Beginning in 1994, some of the largest remaining populations of black abalone *Haliotis cracherodii* on the mainland coast of California, experienced mass mortalities due to the fatal disease called 'withering syndrome', which led to its listing as a species of concern by the USA National Marine Fisheries Service. We have been monitoring black abalone populations along the coast of southern and central California since 1992, and detection of withering syndrome at our southernmost site prompted us to investigate how the impending decline of this dominant grazer might correlate with changes in black abalone recruitment and the rocky intertidal community in which it lives. Quantitative surveys before and after mass mortalities revealed that, after black abalone declined, there was a shift in the composition of the intertidal species assemblage from one dominated by bare rock and crustose coralline algae (good quality abalone habitat) to one with increased cover of sessile invertebrates and sea urchins. Declines in abalone abundance were also correlated with a lack of recruitment to areas affected by withering syndrome, despite the presence of healthy adult populations only tens of kilometers away. This suggests that abalone recruitment might be limited by dispersal, a lack of quality habitat for settlement and early survival, or the continued presence of the disease agent. Recruitment failure and these dramatic shifts in habitat quality indicate that the outlook for recovery of black abalone is poor.

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Ward, J.R., Kim, K., and Harvell, C.D. **Temperature affects coral disease resistance and pathogen growth.** *Marine Ecology Progress Series* 329: 115-121, 2006.

**Notes:** Temperature anomalies on coral reefs now routinely exceed coral stress thresholds, making temperature a critical variable to consider in coral host-pathogen systems. While temperature is widely hypothesized to drive coral disease outbreaks by decreasing coral resistance and increasing pathogen growth rates, tests of the temperature hypothesis are rare. Here we report evidence from the sea fan coral *Gorgonia ventalina-Aspergillus* host-pathogen system that temperature stress increases one component of sea fan resistance. Experimentally infecting sea fan fragments while increasing temperatures to reflect summertime highs in the Florida Keys, USA, caused a 176% increase in activity of host-derived antifungal compounds. Thus, temperature stress and infection induce higher levels of resistance. However, pathogen growth rate also increases over the same temperature range, providing an opportunity for pathogen establishment before host resistance is maximal. This dual effect of temperature emphasizes the need to test intact host-pathogen systems. Given predictions for future warming events, aspergillosis is predicted to continue causing sea fan mortality in the Caribbean Sea.

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