

In this review:

A. Recent articles with abstracts

O/A denotes an open access article or journal

A. Recent articles with abstract

Thompson, R.C.A., Lymbery, A.J., and Smith, A. Parasites, emerging disease and wildlife conservation. *International Journal for Parasitology* 40(10): 1163-1170, 2010.

Notes: In this review some emerging issues of parasite infections in wildlife, particularly in Australia, are considered. We discuss the importance of understanding parasite biodiversity in wildlife in terms of conservation, the role of wildlife as reservoirs of parasite infection, and the role of parasites within the broader context of the ecosystem. Using a number of parasite species, the value of undertaking longitudinal surveillance in natural systems using non-invasive sampling and molecular tools to characterise infectious agents is illustrated in terms of wildlife health, parasite biodiversity and ecology.

Hoberg, E.P. Invasive processes, mosaics and the structure of helminth parasite faunas. *Revue scientifique et technique de l'Office international des Epizooties* 29(2): 255-272, 2010. O/A

Notes: The biosphere in evolutionary and ecological time has been structured by episodes of geographic and host colonisation that have determined distributions of complex assemblages of microparasites and macroparasites, including helminths circulating among vertebrates. Biological invasion is an intricate phenomenon often involving 'extra-range dispersal' and establishment of exotic (non-indigenous) species and populations substantially beyond their native range. Invasion may also involve the expansion or shifting of host and geographic distributions of an endemic (indigenous) species or fauna under changing environmental conditions. Invasions result in faunal interchange occurring under influences from both natural and anthropogenic forces where expansion on spatial/temporal continua bridges continents, regions and landscapes. Drivers for invasion are idiosyncratic, multifactorial, interactive, and opportunistic, with a powerful role for historical contingency. The life history patterns of helminths interact with invasion pathways to determine the potential for introduction. Human-mediated events, such as the global expansion of pathogens linked to development of agriculture, domestication of food animals, and European exploration have had a pervasive influence on the distribution of helminths. Globalisation, broad transport networks and environmental perturbation linked to climate change, along with other drivers, have accelerated these processes. A consequence of invasion and establishment of exotic species is that faunal structure will be a mosaic that includes admixtures of indigenous and non-indigenous species and populations; exemplified by helminth faunas among domestic and free-ranging ungulates and a diversity of host-parasite systems among vertebrates. Contemporary mosaics are evident where human-mediated events have brought assemblages of new invaders and relatively old endemic species into sympatry, highlighting interactions at ecotones, particularly those at borderlands between managed and natural ecosystems. Understanding the historical origins and complex components of mosaics is essential in formulating predictions about future responses to environmental change. Powerful tools are available which support the study of invasive species, the most important being systematics and our capacity to accurately identify parasites and to define evolutionary and biogeographic history. Faunal baselines derived from arrays of biological specimens, integrated surveys and informatics are a permanent

record of the biosphere when archived in museum collections. The absence of comprehensive taxonomic inventories of parasites, including molecular-genetic data, limits our ability to recognise the introduction of non-indigenous parasites, and to document patterns of expansion for local faunas under a regime of environmental perturbation.

Sarmiento-Ramírez, J.M., Abella, E., Martín, M.P., Tellería, M.T., López-Jurado, L.F., Marco, A., and Diéguez-Uribeondo, J. *Fusarium solani* is responsible for mass mortalities in nests of loggerhead sea turtle, *Caretta caretta*, in Boavista, Cape Verde. *FEMS Microbiology Letters* 312(2): 192-200, 2010.

Notes: The fungus *Fusarium solani* (Mart.) Saccardo (1881) was found to be the cause of infections in the eggs of the sea turtle species *Caretta caretta* in Boavista Island, Cape Verde. Egg shells with early and severe symptoms of infection, as well as diseased embryos were sampled from infected nests. Twenty-five isolates with similar morphological characteristics were obtained. Their ITS rRNA gene sequences were similar to the GenBank sequences corresponding to *F. solani* and their maximum identity ranged from 95% to 100%. Phylogenetic parsimony and Bayesian analyses of these isolates showed that they belong to a single *F. solani* clade and that they are distributed in two subclades named A and C (the latter containing 23 out of 25). A representative isolate of subclade C was used in challenge inoculation experiments to test Koch postulates. Mortality rates were c. 83.3% in challenged eggs and 8.3% in the control. Inoculated challenged eggs exhibited the same symptoms as infected eggs found in the field. Thus, this work demonstrates that a group of strains of *F. solani* are responsible for the symptoms observed on turtle-nesting beaches, and that they represent a risk for the survival of this endangered species.

Flint, M., Patterson-Kane, J.C., Limpus, C.J., and Mills, P.C. Health surveillance of stranded green turtles in southern Queensland, Australia (2006-2009): An epidemiological analysis of causes of disease and mortality. *EcoHealth* 7(1): 135-145, 2010. O/A

Notes: Causes of disease and mortality in marine turtles are frequently based on opportunistic investigations producing results that may not contribute to knowledge on how to protect their survival rate. Over a 4-year period (2006-2009), the major causes of stranding and morbidity in 100 green turtles (*Chelonia mydas*) from southern Queensland on the east coast of Australia were determined by comprehensive postmortem examination. Lesions were characterized for analysis using descriptive and probability statistics. Spirorchiid parasitism was found to be the most frequently occurring cause of mortality (41.8%), followed by gastrointestinal impaction (11.8%), microbiological infectious diseases (5.2%), and trauma (5.2%). Spirorchiid parasitism with associated inflammation (75%) was the most frequently occurring disease, followed by gastrointestinal impaction (5.1%). All other diseases were observed at a low prevalence. Assessment of the likelihood of disease being influenced by risk factors (season, maturity, and gender) showed that: (i) there were more observed cases of spirorchiid infection in summer when compared with the other seasons ($P = 0.029$); (ii) immature turtles had more severe spirorchiid parasite infections than mature turtles ($P = 0.032$); and (iii) respiratory disorders were more likely ($P = 0.01$) in summer and autumn than winter or spring. Number of observed cases and severity of spirorchiid lesions were highest in the brain compared with other histologically examined organ systems (all $P > 0.1$). Further investigation is required to build on these findings, aid management decisions, and determine the significance of these diseases for green turtle survivorship in Queensland.

Middlemas, S.J., Raffell, J.A., Hay, D.W., Hatton-Ellis, M., and Armstrong, J.D. Temporal and spatial patterns of sea lice levels on sea trout in western Scotland in relation to fish farm production cycles. *Biology Letters* 6(4): 548-551, 2010.

Notes: The relationship between aquaculture and infestations of sea lice on wild sea trout (*Salmo trutta*) populations is controversial. Although some authors have concluded that there is a link between aquaculture and lice burdens on wild fish, others have questioned this interpretation. Lice levels have been shown to be generally higher on Atlantic salmon farms during the second years of two-year production cycles. Here we investigate whether this pattern relates to lice burdens on wild fish across broad temporal and spatial axes. Within Loch Shieldaig across five successive farm cycles from 2000 to 2009, the percentage of sea trout with lice, and those above a critical level, were significantly higher in the second year of a two-year

production cycle. These patterns were mirrored in 2002-2003 across the Scottish west coast. The results suggest a link between Atlantic salmon farms and sea lice burdens on sea trout in the west of Scotland.

Krkošek, M. Sea lice and salmon in Pacific Canada: ecology and policy. *Frontiers in Ecology and the Environment* 8(4): 201-209, 2010.

Notes: The spread of sea lice (*Lepeophtheirus salmonis*) from salmon farms probably contributes to declines of some native Pacific salmon populations. Migration normally protects juvenile wild Pacific salmon from the marine ectoparasite in coastal waters by separating juvenile salmon from infected wild adults that are located offshore. Farmed salmon populations dwarf natural coastal host populations, particularly in winter, leading to biomagnification of louse populations. By spring, there may be large numbers of lice on farmed salmon, and this is associated with recurrent parasite infestations of wild juvenile salmon and depressed wild salmon stocks. Abiotic (eg temperature and salinity), biotic (eg predator abundance and food availability), and management (eg periodically emptying farms and applying chemical parasiticides) factors are thought to mediate the louse threat, but none have been well studied. Policy is needed that protects undeveloped juvenile salmon habitats and that supports long-term study of salmon ecosystems, to evaluate the sustainability of wild and farmed salmon.

Kangur, A., Kangur, P., Kangur, K., Jarvalt, A., and Haldna, M. *Anguillicoloides crassus* infection of European eel, *Anguilla anguilla* (L.), in inland waters of Estonia: history of introduction, prevalence and intensity. *Journal of Applied Ichthyology* 26(Suppl. 2): 74-80, 2010.

Notes: Eel fishery in Estonian inland waters depends entirely on the stocking of glass eels or pre-grown (farmed) eels. Via importation of live eels of 20-30 cm length the non-indigenous swimbladder nematode *Anguillicoloides crassus* was probably introduced via Germany into Lake Võrtsjärv in 1988, and has since spread to many inland waters of Estonia. In 1992, the parasite was found in eel caught from Lake Võrtsjärv. Between 1992 and 2002 and additionally in 2008, we examined in total 870 eels from Lake Võrtsjärv (270 km²) and in 2008, 63 eels from three small lakes for adult *A. crassus*. The aim of the study was to obtain information on the variation of *A. crassus* infection in eels in Estonian lakes, to determine the temporal dynamics of prevalence and intensity of infection, and to establish a relationship between the length of host and intensity of infection in the eels in Lake Võrtsjärv. There appeared to be a pronounced variation in prevalences of infected eels (from 3.7% to 100%) between the four investigated lakes. However, in Lake Võrtsjärv, the prevalence of adult *A. crassus* infection remained stable (mean about 65%) for many years. The average number of nematode per infected eel (mean intensity) ranged from 12.6 ± 2.5 in 1993 to 4.0 ± 0.6 in 1999 in Lake Võrtsjärv, while it was significantly higher ($P < 0.0001$) in the period 1992-1998 compared to 1999-2002 and 2008. The mean number of parasites per swimbladder was not related to eel length and no statistical difference was found in the condition factor of infected and non-infected eels. Although under normal environmental conditions *A. crassus* has not caused serious disease problems to eels in the study area, high intensity of parasite infection may contribute to eel kills due to oxygen deficiency in winter under the ice in Lake Võrtsjärv.

Smith, T.B., Blondeau, J., Nemeth, R.S., Pittman, S.J., Calnan, J.M., Kadison, E., and Gass, J. Benthic structure and cryptic mortality in a Caribbean mesophotic coral reef bank system, the Hind Bank Marine Conservation District, US Virgin Islands. *Coral Reefs* 29(2): 289-308, 2010. O/A

Notes: Coral reef banks may form an important component of mesophotic coral ecosystems (MCEs) in the Caribbean, but remain poorly explored relative to shallower reefs and mesophotic habitats on slopes and walls. Consequently, the processes structuring mesophotic coral reef communities are not well understood, particularly the role of disturbance. A large and regionally important mesophotic system, the Hind Bank Marine Conservation District (MCD), St. Thomas, USVI, was systematically surveyed. Data were used to construct a comprehensive benthic habitat map for the MCD, describe the abiotic and biotic components of the benthos among habitats, and investigate patterns of coral health among habitats. Two-thirds of the MCD (23.6 km²) was found to be dense coral reef (Coral Cover = 24.1%) dominated by the *Montastraea annularis* species complex. Coral reef ecosystems were topographically complex, but could be classified into distinct habitat types, including high coral banks (35.8% of the MCD) and two large novel coral reef habitat types corresponding to an extremely flat basin (18%) and a highly rugose hillock basin (6.5%), containing thousands of coral knolls (2-10 m high). An extreme disease event

with undescribed signs of mortality occurred on 47% of coral reefs and reached a high prevalence in affected areas ($42.4\% \pm 6.3$ SE, $N = 26$). The disease was significantly clustered in the basin habitats of the western MCD (global Moran's $I = 0.32$, $P < 0.01$). Observations of the spatial pattern suggested that the driver was specific to the basin habitats and may have been caused by a coherent abiotic event.

Glas, M.S., Motti, C.A., Negri, A.P., Sato, Y., Froschio, S., Humpage, A.R., Krock, B., Cembella, A., and Bourne, D.G. Cyanotoxins are not implicated in the etiology of coral black band disease outbreaks on Pelorus Island, Great Barrier Reef. *FEMS Microbiology Ecology* 73(1): 43-54, 2010.

Notes: Cyanobacterial toxins (i.e. microcystins) produced within the microbial mat of coral black band disease (BBD) have been implicated in disease pathogenicity. This study investigated the presence of toxins within BBD lesions and other cyanobacterial patch (CP) lesions, which, in some instances (~19%), facilitated the onset of BBD, from an outbreak site at Pelorus Island on the inshore, central Great Barrier Reef (GBR). Cyanobacterial species that dominated the biomass of CP and BBD lesions were cultivated and identified, based on morphology and 16S rRNA gene sequences, as *Blennothrix*- and *Oscillatoria*-affiliated species, respectively, and identical to cyanobacterial sequences retrieved from previous molecular studies from this site. The presence of the cyanotoxins microcystin, cylindrospermopsin, saxitoxin, nodularin and anatoxin and their respective gene operons in field samples of CP and BBD lesions and their respective culture isolations was tested using genetic (PCR-based screenings), chemical (HPLC-UV, FTICR-MS and LC/MSn) and biochemical (PP2A) methods. Cyanotoxins and cyanotoxin synthetase genes were not detected in any of the samples. Cyanobacterial species dominant within CP and BBD lesions were phylogenetically distinct from species previously shown to produce cyanotoxins and isolated from BBD lesions. The results from this study demonstrate that cyanobacterial toxins appear to play no role in the pathogenicity of CP and BBD at this site on the GBR.

Negandhi, K., Blackwelder, P.L., Ereskovsky, A.V., and Lopez, J.V. Florida reef sponges harbor coral disease-associated microbes. *Symbiosis* 51(1): 117-129, 2010. [O/A](#)

Notes: Sponges can filter large volumes of seawater and accumulate highly diverse and abundant microbial communities within their tissue. Culture-independent techniques such as fluorescent in situ hybridization (FISH), 16S small subunit (SSU) rRNA gene analyses, and transmission electron microscopy (TEM) were applied to characterize the presence and distribution of microbes within sponges abundant on south Florida reefs. This study found that coral disease-associated bacteria (CDAB) are harbored within *Agelas tubulata* and *Amphimedon compressa*. FISH probes detected several potential bacterial pathogens such as *Aurantimonas corallicida*, *Cytophaga* sp., *Desulfovibrio* spp, *Serratia marcescans*, and *Vibrio mediterranei* within *A. compressa* and *A. tubulata* host sponges. Spatial differences in the distribution of targeted bacteria were seen within sponge hosts. Transmission electron microscopy of *A. compressa* indicated there was a higher concentration of bacteria in the choanosome compared to the ectosome. These observed spatial distributions support the presence of internal sponge niches, which could play a role in the location of the CDAB within the sponges.

Andersen, S.B., Vestergaard, M.L., Ainsworth, T.D., Hoegh-Guldberg, O., and Kuhl, M. Acute tissue death (white syndrome) affects the microenvironment of tabular *Acropora* corals. *Aquatic Biology* 10(1): 99-104, 2010.

Notes: White syndrome (WS) is a collective term for coral diseases that cause acute tissue loss, resulting in apparently healthy tissue bordering on exposed skeleton. In this study, the microenvironmental condition and tissue structure of WS-affected tabular acroporid corals were assessed by O_2 microelectrodes and histological techniques. The high spatial resolution of the microelectrode measurements enabled an evaluation of the extent of physiological changes at, and 2 cm away from, the WS border. Respiration of the coral host was decreased on the skeleton-tissue border but was comparable to that of healthy corals only 2 cm away from the border. Histological data, however, showed a decrease in mesoglea thickness on and 2 cm away from the WS border, which correlates with a previously observed allocation of photoassimilates away from the WS border. We suggest that there are colony-wide negative effects of WS which affect only the host physiology and, as disparate etiologies are evident in WS, these must be distinguished through the utilization of a multiple tool approach.

Littman, R.A., Bourne, D.G., and Willis, B.L. Responses of coral-associated bacterial communities to heat stress differ with *Symbiodinium* type on the same coral host. *Molecular Ecology* 19(9): 1978-1990, 2010.

Notes: This study compared the effect of heat stress on coral-associated bacterial communities among juveniles of the coral, *Acropora tenuis*, hosting different *Symbiodinium* types. In comparison to a control temperature treatment (28 °C), we documented dramatic changes in bacterial associates on juvenile corals harbouring ITS 1 type D *Symbiodinium* when placed in a high (32 °C) temperature treatment. In particular, there was a marked increase in the number of retrieved *Vibrio* affiliated sequences, which coincided with a 44% decline in the photochemical efficiency of the D-juveniles. Interestingly, these *Vibrio* sequences affiliated most closely with the coral pathogen, *Vibrio coralliilyticus*, which has been implicated in some coral disease outbreaks. In contrast, *A. tenuis* hosting ITS 1 type C1 *Symbiodinium* did not exhibit major bacterial shifts in the elevated temperature treatment, indicating a more stable bacterial community during thermal stress; concomitantly a decline (10%) in photochemical efficiency was minimal for this group. D juveniles that had been exposed to moderately elevated sea temperatures (30 °C) in the field before being placed in the control temperature treatment displayed a decrease in the number of *Vibrio* affiliated sequences and bacterial profiles shifted to become more similar to profiles of corals harbouring type C1 *Symbiodinium*. In combination, these results demonstrate that thermal stress can result in shifts in coral-associated bacterial communities, which may lead to deteriorating coral health. The lower resilience of *A. tenuis* to thermal stress when harbouring *Symbiodinium* D highlights the importance of inter-kingdom interactions among the coral host, dinoflagellate endosymbiont and bacterial associates for coral health and resilience.

Arkoosh, M.R., Boylen, D., Dietrich, J., Anulacion, B.F., Ylitalo, G., Bravo, C.F., Johnson, L.L., Loge, F.J., and Collier, T.K. Disease susceptibility of salmon exposed to polybrominated diphenyl ethers (PBDEs). *Aquatic Toxicology* 98(1): 51-59, 2010.

Notes: The health effects of the flame retardant polybrominated diphenyl ethers (PBDEs) in fish are not well understood. To determine the potential effects of this ubiquitous contaminant class on fish health, juvenile subyearling Chinook salmon (*Oncorhynchus tshawytscha*) were fed a diet that reflected the PBDE congeners found in the stomach contents of subyearling Chinook salmon collected from the highly urbanized and industrialized lower Willamette River in the Columbia River Basin of North America. The diet, consisting of five PBDE congeners (BDE-47, BDE-99, BDE-100, BDE-153 and BDE-154), was fed to the salmon at 2% of their body weight in food per day for 40 days. Two concentrations of the diet (1 x and 10x PBDE) were fed to the salmon. The 1x PBDE diet reflected the concentration of PBDEs (190 ng PBDEs/g food) found in the stomach contents of juvenile subyearling Chinook salmon; the 10x diet was prepared at 10 times that concentration. The fish were then exposed to the marine bacterial pathogen *Listonella anguillarum* to assess susceptibility to infectious disease. Juvenile Chinook salmon fed the 1x PBDE diet were more susceptible to *L. anguillarum* than salmon fed the control diet. This suggests that juvenile salmonids in the lower Willamette River exposed to PBDEs may be at greater risk for disease than nonexposed juvenile salmonids. In contrast, salmon that consumed the 10x PBDE diet were not more susceptible to the pathogen than salmon fed the control diet. The mechanisms for the dichotomous results observed in disease susceptibility between salmon fed the 1x and 10x PBDE diets are currently not known but have also been observed in other species exposed to PBDEs with respect to immune function.

Bossart, G.D., Reif, J.S., Schaefer, A.M., Goldstein, J., Fair, P.A., and Saliki, J.T. Morbillivirus infection in free-ranging Atlantic bottlenose dolphins (*Tursiops truncatus*) from the Southeastern United States: Seroepidemiologic and pathologic evidence of subclinical infection. *Veterinary Microbiology* 143(2-4): 160-166, 2010.

Notes: From 2003 to 2007, sera (n = 234) from free-ranging Atlantic bottlenose dolphins (*Tursiops truncatus*) inhabiting two southeast Atlantic estuarine regions, the Indian River Lagoon (IRL), FL and Charleston, SC (CHS) were tested for antibodies to cetacean morbilliviruses as part of a multidisciplinary study of individual and population health. Positive morbillivirus titers were found on initial capture in 12 of 122 (9.8%) IRL dolphins in the absence of an epizootic. All CHS dolphins were seronegative. Positive fluctuating morbillivirus titers and seroconversion were found in IRL dolphins. Seropositivity was detected in dolphins 8-13 years of age as well as in dolphins that were alive during the 1987-1988 epizootic. During the study

period, pathologic and immunohistochemical findings from stranded IRL dolphins (n = 14) did not demonstrate typical morbillivirus-associated lesions or the presence of morbillivirus antigen. The findings suggest that morbillivirus infections are occurring in the absence of widespread mortality in IRL dolphins.

Rejmanek, D., Miller, M.A., Grigg, M.E., Crosbie, P.R., and Conrad, P.A. Molecular characterization of *Sarcocystis neurona* strains from opossums (*Didelphis virginiana*) and intermediate hosts from Central California. *Veterinary Parasitology* 170(1-2): 20-29, 2010.

Notes: *Sarcocystis neurona* is a significant cause of neurological disease in horses and other animals, including the threatened Southern sea otter (*Enhydra lutris nereis*). Opossums (*Didelphis virginiana*), the only known definitive hosts for *S. neurona* in North America, are an introduced species in California. *S. neurona* DNA isolated from sporocysts and/or infected tissues of 10 opossums, 6 horses, 1 cat, 23 Southern sea otters, and 1 harbor porpoise (*Phocoena phocoena*) with natural infections was analyzed based on 15 genetic markers, including the first internal transcribed spacer (ITS-1) region; the 25/396 marker; *S. neurona* surface antigen genes (snSAGs) 2, 3, and 4; and 10 different microsatellites. Based on phylogenetic analysis, most of the *S. neurona* strains segregated into three genetically distinct groups. Additionally, fifteen *S. neurona* samples from opossums and several intermediate hosts, including sea otters and horses, were found to be genetically identical across all 15 genetic markers, indicating that fatal encephalitis in Southern sea otters and equine protozoal myeloencephalitis (EPM) in horses is strongly linked to *S. neurona* sporocysts shed by opossums.

Miller, M.A., Conrad, P.A., Harris, M., Hatfield, B., Langlois, G., Jessup, D.A., Magargal, S.L., Packham, A.E., Toy-Choutka, S., Melli, A.C., Murray, M.A., Gulland, F.M., and Grigg, M.E. A protozoal-associated epizootic impacting marine wildlife: Mass-mortality of southern sea otters (*Enhydra lutris nereis*) due to *Sarcocystis neurona* infection. *Veterinary Parasitology* 172(3-4): 183-194, 2010.

Notes: During April 2004, 40 sick and dead southern sea otters (*Enhydra lutris nereis*) were recovered over 18 km of coastline near Morro Bay, California. This event represented the single largest monthly spike in mortality ever recorded during 30 years of southern sea otter stranding data collection. Because of the point-source nature of the event and clinical signs consistent with severe, acute neurological disease, exposure to a chemical or marine toxin was initially considered. However, detailed postmortem examinations revealed lesions consistent with an infectious etiology, and further investigation confirmed the protozoan parasite *Sarcocystis neurona* as the underlying cause. Tissues from 94% of examined otters were PCR-positive for *S. neurona*, based on DNA amplification and sequencing at the ITS-1 locus, and 100% of tested animals (n = 14) had elevated IgM and IgG titers to *S. neurona*. Evidence to support the point-source character of this event include the striking spatial and temporal clustering of cases and detection of high concentrations of anti-*S. neurona* IgM in serum of stranded animals. Concurrent exposure to the marine biotoxin domoic acid may have enhanced susceptibility of affected otters to *S. neurona* and exacerbated the neurological signs exhibited by stranded animals. Other factors that may have contributed to the severity of this epizootic include a large rainstorm that preceded the event and an abundance of razor clams near local beaches, attracting numerous otters close to shore within the affected area. This is the first report of a localized epizootic in marine wildlife caused by apicomplexan protozoa.

Robles-Sikisaka, R., Bohonak, A.J., McClenaghan, L.R., and Dhar, A.K. Genetic signature of rapid IHHNV (infectious hypodermal and hematopoietic necrosis virus) expansion in wild *Penaeus* shrimp populations. *PLoS ONE* 5(7): art. e11799, 2010. [O/A](#)

Notes: Infectious hypodermal and hematopoietic necrosis virus (IHHNV) is a widely distributed single-stranded DNA parvovirus that has been responsible for major losses in wild and farmed penaeid shrimp populations on the northwestern Pacific coast of Mexico since the early 1990's. IHHNV has been considered a slow-evolving, stable virus because shrimp populations in this region have recovered to pre-epizootic levels, and limited nucleotide variation has been found in a small number of IHHNV isolates studied from this region. To gain insight into IHHNV evolutionary and population dynamics, we analyzed IHHNV capsid protein gene sequences from 89 *Penaeus* shrimp, along with 14 previously published sequences. Using Bayesian coalescent approaches, we calculated a mean rate of nucleotide substitution for IHHNV that was unexpectedly high

(1.39×10^{-4} substitutions/site/year) and comparable to that reported for RNA viruses. We found more genetic diversity than previously reported for IHHNV isolates and highly significant subdivision among the viral populations in Mexican waters. Past changes in effective number of infections that we infer from Bayesian skyline plots closely correspond to IHHNV epizootiological historical records. Given the high evolutionary rate and the observed regional isolation of IHHNV in shrimp populations in the Gulf of California, we suggest regular monitoring of wild and farmed shrimp and restriction of shrimp movement as preventative measures for future viral outbreaks.

Van Houtan, K.S., Hargrove, S.K., and Balazs, G.H. Land use, macroalgae, and a tumor-forming disease in marine turtles. PLoS ONE 5(9): art. e12900, 2010. [O/A](#)

Notes: Wildlife diseases are an increasing concern for endangered species conservation, but their occurrence, causes, and human influences are often unknown. We analyzed 3,939 records of stranded Hawaiian green sea turtles (*Chelonia mydas*) over 28 years to understand fibropapillomatosis, a tumor-forming disease linked to a herpesvirus. Turtle size is a consistent risk factor and size-standardized models revealed considerable spatial and temporal variability. The disease peaked in some areas in the 1990s, in some regions rates remained constant, and elsewhere rates increased. Land use, onshore of where the turtles feed, may play a role. Elevated disease rates were clustered in watersheds with high nitrogen-footprints; an index of natural and anthropogenic factors that affect coastal eutrophication. Further analysis shows strong epidemiological links between disease rates, nitrogen-footprints, and invasive macroalgae and points to foraging ecology. These turtles now forage on invasive macroalgae, which can dominate nutrient rich waters and sequester environmental N in the amino acid arginine. Arginine is known to regulate immune activity, promote herpesviruses, and contribute to tumor formation. Our results have implications for understanding diseases in aquatic organisms, eutrophication, herpesviruses, and tumor formation.

O'Hara, T.M., Holcomb, D., Elzer, P., Estep, J., Perry, Q., Hagius, S., and Kirk, C. *Brucella* species survey in polar bears (*Ursus maritimus*) of northern Alaska. Journal of Wildlife Diseases 46(3): 687-694, 2010.

Notes: We report on the presence of specific antibodies to *Brucella* spp. and *Yersinia enterocolitica* in polar bears (*Ursus maritimus*) from northern Alaska (southern Beaufort Sea) during 2003-2006. Based on numerous known stressors (e.g., climate change and loss of sea ice habitat, contaminants), there is increased concern regarding the status of polar bears. Considering these changes, it is important to assess exposure to potentially pathogenic organisms and to improve understanding of transmission pathways. *Brucella* or specific antibodies to *Brucella* spp. has been reported in marine mammals. Various assays were used to elucidate the pathway or source of exposure (e.g., "marine" vs "terrestrial" *Brucella* spp.) of northern Alaska polar bears to *Brucella* spp. The standard plate test (SPT) and the buffered *Brucella* antigen card test (BBA) were used for initial screening for antibodies specific to *Brucella*. We then evaluated positive reactors (presence of serum antibody specific for *Brucella* spp.) using immunoblots and competitive enzyme-linked immunosorbent assay (cELISA, based on pumped-derived *Brucella* spp. antigen). Annual prevalence of antibody (BBA and SPT) for *Brucella* spp. ranged from 6.8% to 18.5% over 2003-2006, with an overall prevalence of 10.2%. Prevalence of *Brucella* spp. antibody did vary by age class. Western blot analyses indicated 17 samples were positive for *Brucella* spp. antibody; of these, 13 were negative by marine (primed) derived *Brucella* antigen cELISA and four were positive by marine cELISA. Of the four samples positive for *Brucella* antibody by marine cELISA, three cross-reacted with *Y. enterocolitica* and *Brucella* spp. (one sample was *Brucella* negative and *Y. enterocolitica* positive). It appears the polar bear antibody does not react with the antigens used on the marine cELISA assay, potentially indicating a terrestrial (nonprimed) source of *Brucella* spp.

Deem, S.L., Merkel, J., Ballweber, L., Vargas, F.H., Cruz, M.B., and Parker, P.G. Exposure to *Toxoplasma gondii* in Galapagos penguins (*Spheniscus mendiculus*) and flightless cormorants (*Phalacrocorax harrisi*) in the Galapagos Islands, Ecuador. Journal of Wildlife Diseases 46(3): 1005-1011, 2010.

Notes: *Toxoplasma gondii* is one of the most common protozoan parasites of humans and warm-blooded animals. Members of the family Felidae are the only definitive hosts of this parasite and, thus, important in the epidemiology of the disease. Previous studies on Pacific islands have found *T. gondii* infections in a number of avian species where domestic cats (*Felis catus*) have been introduced. Little is known about *T. gondii* in the Galapagos Islands, although introduced domestic cats in the archipelago

are known to be *T. gondii* antibody-positive. In this study, we quantified prevalence of antibody to *T. gondii* in two threatened avian marine species, Galapagos penguins (*Spheniscus mendiculus*) and flightless cormorants (*Phalacrocorax harrisi*), and tested the hypothesis that this parasite is more prevalent on Isabela Island (with cats) than on Fernandina Island (without cats). Overall, antibody prevalence was 2.3% in both Galapagos penguins and flightless cormorants from samples collected during 2003-2005, and in 2008. In Galapagos penguins (n = 298), a significantly higher antibody prevalence was found in penguins on Fernandina Island (free of cats) than on Isabela Island (with cats, Fisher's exact test, P = 0.02). In flightless cormorants (n = 258), there was a higher antibody prevalence in cormorants living on Isabela than on Fernandina, although this difference was not statistically significant (Fisher's, P = 0.19). This study is the first to show exposure to *T. gondii* in endemic avian species in the Galapagos Islands, providing evidence for disease-related risks associated with the feral cat population in the archipelago. We provide possible explanations for these findings and recommendations for future studies towards a better understanding of the epidemiology of *T. gondii* in the Galapagos Islands.

Himworth, C.G., Haulena, M., Lambourn, D.M., Gaydos, J.K., Huggins, J., Calambokidis, J., Ford, J.K.B., Zaremba, K., and Raverty, S. Pathology and epidemiology of phocid herpesvirus-1 in wild and rehabilitating harbor seals (*Phoca vitulina richardsi*) in the Northeastern Pacific. *Journal of Wildlife Diseases* 46(3): 1046-1051, 2010.

Notes: Phocid herpesvirus-1 (PhHV-1, subfamily Alpha herpesvirinae) was isolated from harbor seals (*Phoca vitulina vitulina*) in the Netherlands in 1985, and was subsequently identified in Pacific harbor seals (*Phoca vitulina richardsi*) from California, USA in the 1990s. PhHV-1-associated pathology was first recognized in harbor seal carcasses submitted to a veterinary diagnostic laboratory in Abbotsford, British Columbia, Canada in 2000, and 63 cases were identified by 2008. A review of these cases indicated that PhHV-1-associated disease is widespread in harbor seals in the wild and within rehabilitation facilities in the coastal northeastern Pacific (including British Columbia, Canada, and Washington, USA). Morbidity and mortality occurred primarily in neonatal and weanling seal pops, and was due to PhHV-1 alone, or in combination with other disease processes. All cases occurred between July and October, corresponding to the pupping and weaning seasons in this area. Although previous publications have described the prevalence of antibody to PhHV-1 in harbor seals from British Columbia, Canada and Washington, USA this is the first study to focus on the epidemiology and pathology of the virus in this region.

Navas-Camacho, R., Gil-Agudelo, D.L., Rodríguez-Ramírez, A., Reyes-Nivia, M.C., and Garzón-Ferreira, J. Coral diseases and bleaching on Colombian Caribbean coral reefs. *Revista de Biología Tropical* 58(Suppl. 1): 95-106, 2010. [O/A](#)

Notes: Since 1998 the National Monitoring System for the Coral Reefs of Colombia (SIMAC) has monitored the occurrence of coral bleaching and diseases in some Colombian coral reefs (permanent stations at San Andres Island, Rosario Islands, Tayrona, San Bernardo Islands and Uraba). The main purpose is to evaluate their health status and to understand the factors that have been contributing to their decline. To estimate these occurrences, annual surveys in 126 permanent belt transects (10 x 2 m) with different depth intervals (3-6 meters, 9-12 meters and 15-18 meters) are performed at all reef sites. Data from the 1998-2004 period, revealed that San Andres Island had many colonies with diseases (38.9 colonies/m²), and Uraba had high numbers with bleaching (54.4 colonies/m²). Of the seven reported coral diseases studied, Dark Spots Disease (DSD), and White Plague Disease (WPD) were noteworthy because they occurred in all Caribbean monitored sites, and because of their high interannual infection incidence. Thirty five species of scleractinian corals were affected by at least one disease and a high incidence of coral diseases on the main reef builders is documented. Bleaching was present in 34 species. During the whole monitoring period, *Agaricia agaricites* and *Siderastrea siderea* were the species most severely affected by DSD and bleaching, respectively. Diseases on species such as *Agaricia fragilis*, *A. grahamae*, *A. humilis*, *Diploria divosa*, *Eusmilia fastigata*, *Millepora complanata*, and *Mycetophyllia aliciae* are recorded for the first time in Colombia. We present bleaching and disease incidences, kinds of diseases, coral species affected, reef localities studied, depth intervals of surveys, and temporal (years) variation for each geographic area. This variation makes difficult to clearly determine defined patterns or general trends for monitored reefs. This is the first long-term study of coral diseases and bleaching in the Southwestern Caribbean, and one of the few long term monitoring studies on coral diseases worldwide.

Navas-Camacho, R., Rodríguez-Ramírez, A., and Reyes-Nivia, M.C. Agents of coral mortality on reef formations of the Colombian Pacific. *Revista de Biología Tropical* 58(Suppl. 1): 133-138, 2010. O/A

Notes: The National Monitoring System for Coral Reefs of Colombia (SIMAC) monitors the impact of some of the most important agents of coral tissue loss (bleaching and/or disease) in the Colombian Pacific coral formations since 1998. Physiological bleaching is among the main results of stress in the area. Signs of coral diseases resembling bacterial bleaching such as White Plague and White Band, were observed in Malpelo and Gorgona islands. Damage to the Pacific gorgonian *Pacificorgia* spp., similar to those produced by Aspergillosis in Caribbean corals, was detected in Utria Bay. The presence of tumors in colonies of massive corals was also recorded. Even though coral diseases are globally widespread, their occurrence in American Pacific reefs has been poorly documented to date.

Sánchez, J.A., Herrera, S., Navas-Camacho, R., Rodríguez-Ramírez, A., Herron, P., Pizarro, V., Acosta, A.R., Castillo, P.A., Montoya, P., and Orozco, C. White plague-like coral disease in remote reefs of the Western Caribbean. *Revista de Biología Tropical* 58(Suppl. 1): 145-154, 2010. O/A

Notes: The health of coral reef communities has been decreasing over the last 50 years, due the negative effects of human activities combined with other natural processes. We present documentation of a White Plague Disease (WPD) outbreak in the Serrana Bank, an isolated Western Caribbean atoll with presumably inexistent pollutant inputs from local human settlements. In addition, this study summarizes seven years of observations on diseased corals in the nearby island of San Andrés, which in contrast is one of the most populated islands of the Caribbean. There was a massive coral mortality in the atoll lagoon (14°27'53.24", 80°14'22.27" W, and 12m depth) due to WPD on May 4 of 2003. Seventeen species were found dead or largely affected by the disease. The information resulting from GPS and manta-tow transects revealed that approximately 5.8ha of reticulate *Montastraea* spp. patch reefs were lethally affected by the disease in the atoll. On May 8 of the same year we observed and calculated a mean coral cover of 7.03% (SD ± 2.44), a mean diseased coral tissue cover of 5.5% (SD ± 1.1) and a 13.4% (SD ± 8.05) of recently dead coral covered with a thin filamentous algae layer; approximately 73% of mortalities caused by the disease occurred before the end of the outbreak. A rough estimate of 18.9% in recent coral cover reduction can be attributed to WPD. This represents about 82% of the total coral cover decline since 1995. Semi-enclosed environments such as atoll lagoons and the reticulate patch-reefs of *Montastraea* spp. seem to be particularly vulnerable to this kind of coral disease, which constitute an alert to increase the monitoring of the same kind of atoll environments. The WPD has been present in the area of the nearby island of San Andrés at a low prevalence level, with sporadic increasing peaks of disease proliferation. The peaks observed during 1999 and 2004 comprised increases of 266% and 355% respectively, suggesting an alarming progression of the disease in this area. This study includes new information of the epizootiology of White Plague Disease and documents the permanent prevalence and progression of the WPD in the area of San Andres Island.

Wetchateng, T., Friedman, C.S., Wight, N.A., Lee, P.Y., Teng, P.H., Sriurairattana, S., Wongprasert, K., and Withyachumnarnkul, B. Withering syndrome in the abalone *Haliotis diversicolor supertexta*. *Diseases of Aquatic Organisms* 90(1): 69-76, 2010.

Notes: Abalone aquaculture is a small but growing industry in Thailand and is based on both the exotic *Haliotis diversicolor supertexta* and the native *H. asinina*. Withering syndrome (WS) in abalone is caused by an infection with the Rickettsia-like organism (RLO) 'Candidatus *Xenohaliotis californiensis*' and has been spread to many countries globally. The present study reports the first observation of the WS-RLO agent in the small abalone, *H. diversicolor supertexta* in Thailand, Taiwan (ROC) and the People's Republic of China (PRC). Under light microscopy, the RLO was observed as intracytoplasmic inclusions within epithelial cells lining the post-esophagus and, to a minor extent, the intestine of *H. diversicolor*. Under transmission electron microscopy, inclusions were characterized as colonies of rod-shaped bacteria, 200 x 1800 nm in size, within a vesicle in the cytoplasm of the infected cell. The RLO from the small abalone bound with WS-RLO-specific in situ hybridization probes and was amplified by polymerase chain reaction (PCR), using primers designed from the 16S rDNA sequence of the original WS-RLO from California, USA. The PCR product of RLO samples from both the PRC and Thailand showed extremely high identity with the California WS-RLO (100 and 99%, respectively). These data combined with the history of abalone movements for aquaculture purposes indicate that RLOs observed in Thailand, Taiwan and the PRC are the WS-RLO that originated from California.

Reed, K.C., Muller, E.M., and van Woesik, R. Coral immunology and resistance to disease. *Diseases of Aquatic Organisms* 90(2): 85-92, 2010.

Notes: Scleractinian corals (phylum Cnidaria, class Anthozoa) have innate immunological responses against infections. Research has recently suggested that corals also possess an adaptive-like immunological repertoire that recognizes specific pathogens and allografts. While evolutionarily distinct, the corals' innate and adaptive-like immunity systems are not mutually exclusive because the phagocytic cells of the non-specific, innate immune system may activate specific adaptive immunological responses. Warming oceans may immunocompromise coral hosts, making them more susceptible to tropical marine diseases, independent of the virulence of the pathogen. The ability of corals to ward off both primary and opportunistic infections, through adaptive-like mechanisms, may play a critical role in the corals' ability to fight future disease infection. Here we show evidence that corals possess immunological repertoires that extend well beyond simple innate defenses. The extent to which corals have developed such an adaptive-like immune repertoire will determine whether corals will survive climate change and other anthropogenic disturbances.

Arboleda, M.D.M. and Reichardt, W.T. *Vibrio* sp causing *Porites* ulcerative white spot disease. *Diseases of Aquatic Organisms* 90(2): 93-104, 2010.

Notes: The causative agent of the Indo-Pacific coral disease, *Porites* ulcerative white spot syndrome (PUWS), that affects *Porites* spp. and a few other coral genera has so far remained unidentified. Inoculation of thiosulphate citrate bile sucrose (TCBS) agar with tissue material from *Porites cylindrica* infected with white spot produced colonies of approximately 3 mm diameter consisting of Gram-negative, motile, non-sucrose-fermenting, slightly curved rods with a minimum NaCl requirement of 0.3%. Three of these putative *Vibrio* sp. isolates were used for infection trials that included different stages of cell growth. Four modes of inoculation and 3 stages of bacterial cell growth were considered for testing Koch's postulates. Stationary phase cells proved more consistently infectious than did exponentially growing or starved cells using a 1-step immersion technique at cell concentrations of 10^4 cells ml⁻¹. A 1-step immersion technique proved more reliable in producing signs of white spot than did other techniques, such as injection, smearing and 2-step immersion of the inoculum. At inoculum densities $>10^4$ cells ml⁻¹ further signs of disease, such as tissue degradation and bleaching, also became evident. At elevated temperatures (>29 °C) bleaching remained absent for at least 2 mo from non-inoculated corals serving as controls, but was observed in artificially infected coral fragments. Of the 9 seawater aquaria containing healthy specimens of *P. cylindrica*, 6 showed signs of white spot 15 d after infection with an isolate tentatively identified as *Vibrio* sp. Based on 99% similarity of its 16S rRNA gene sequence and selected phenotypical features, this isolate revealed a close relationship to *V. natriegens* and *V. parahaemolyticus*.

Aeby, G.S., Ross, M., Williams, G.J., Lewis, T.D., and Work, T.M. Disease dynamics of *Montipora* white syndrome within Kaneohe Bay, Oahu, Hawaii: distribution, seasonality, virulence, and transmissibility. *Diseases of Aquatic Organisms* 91(1): 1-8, 2010.

Notes: We report on an investigation of *Montipora* white syndrome (MWS), which is a coral disease reported from Hawaii, USA, that results in tissue loss. Disease surveys of *Montipora capitata* within Kaneohe Bay (Oahu) found colonies that were affected by MWS on 9 reefs within 3 regions of Kaneohe Bay (south, central, north). Mean MWS prevalence ranged from 0.02 to 0.87% and average number of MWS cases per survey site ranged from 1 to 28 colonies. MWS prevalence and number of cases were significantly lower in the central region as compared to those in the north and south regions of Kaneohe Bay. There was a positive relationship between host abundance and MWS prevalence, and differences in host abundance between sites explained ~27% of the variation in MWS prevalence. Reefs in central Kaneohe Bay had lower *M. capitata* cover and lower MWS levels. MWS prevalence on reefs was neither significantly different between seasons (spring versus fall) nor among 57 tagged colonies that were monitored through time. MWS is a chronic and progressive disease causing *M. capitata* colonies to lose an average of 3.1% of live tissue mo⁻¹. Case fatality rate was 28% after 2 yr but recovery occurred in some colonies (32%). Manipulative experiments showed that the disease is acquired through direct contact. This is the first study to examine the dynamics of MWS within Hawaii, and our findings suggest that MWS has the potential to degrade Hawaii's reefs through time.