

Emerging and recurring diseases in cetaceans worldwide and the role of environmental stressors

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ABSTRACT

Emerging and recurring infectious diseases known or suspected to have the potential to significantly impact cetacean populations, and possible synergistic effects of environmental factors are reviewed. Cetacean morbilliviruses and papillomaviruses and brucellosis may affect population densities through high mortality rates or interference with reproduction. Evidence is available for the role of environmental factors in the emergence/recurrence and severity of at least six infectious conditions i.e. lobomycosis, toxoplasmosis, tattoo skin disease, generalized bacterial infections, miscellaneous skin diseases and morbillivirus epizootics. Other micro-parasites of potential importance include rhabdo-, herpes- and parainfluenza-viruses as well as *Helicobacter* spp., *Streptococcus* spp., *Salmonella* spp. and *Mycobacterium marinum*. The population impact and aetiology of newly emerging skin diseases in South America are unknown and represent a cause of concern.

INTRODUCTION

Microparasites, including viruses, bacteria, and protozoans, may constrain the growth of wild animal populations (Anderson and May 1979; Anderson, 1982; Fenner, 1983; Gulland, 1995). The intensity of this effect is a function of the heightened mortality rate and/or reduction in reproductive capacity. Thus, microparasites may increase the risk of extinction of small populations in combination with other factors (May, 1986; Thorne and Williams, 1988; Gulland, 1995, Raga *et al.*, 1997). Here we review some emerging and recurring infectious diseases known or suspected to have the potential to significantly impact cetacean populations, and possible synergistic effects of environmental factors.

CETACEAN MORBILLIVIRUS

The dolphin, porpoise and pilot whale morbilliviruses (DMV, PMV and PWMV) are strains of a recently recognized member of the genus *Morbillivirus* (single, negative-strand RNA viruses of family *Paramyxoviridae*) called cetacean morbillivirus (CeMV) (Barrett *et al.*, 1993; Blixenkrone-Möller *et al.*, 1996; Kennedy, 1998; Taubenberger *et al.*, 2000; Di Guardo *et al.*, 2005). They trigger highly lethal epizootic diseases in cetaceans causing bronchiolo-interstitial pneumonia and interstitial pneumonia, non-suppurative encephalitis and lymphoid depletion (Kennedy *et al.*,

1991, Domingo *et al.*, 1992, Duignan *et al.*, 1992; Schulman *et al.*, 1997). CeMV caused epizootics in bottlenose dolphins (*Tursiops truncatus*) along the Atlantic USA coast in 1982, 1987-1988 and 1993-1994 (Lipscomb *et al.*, 1994; Krafft *et al.*, 1995, Duignan *et al.*, 1996). Over 50% of the inshore bottlenose dolphins off New Jersey and south to Canaveral National Seashore, Florida, USA, may have died during the 1987-1988 epizootic (Lipscomb *et al.*, 1994). PMV caused mortalities of an unknown number of harbour porpoises (*Phocoena phocoena*) along the coasts of the United Kingdom and the Netherlands in 1988-1990 (Kennedy *et al.*, 1988; Visser *et al.*, 1993). DMV was responsible for lethal epizootics in at least two small cetacean species from the Mediterranean Sea in 1990-1992 and again in 2006-2007 (Domingo *et al.*, 1990; Van Bresseem *et al.*, 1991, 1993a; Di Guardo *et al.*, 1992, 2005; Fernandez *et al.*, 2008; Raga *et al.*, 2008). The epizootic started along the central coast of Spain in 1990 and ended in Turkey and the Greek Islands in 1992 (Aguilar and Raga, 1993). It affected predominantly striped dolphins (*Stenella coeruleoalba*). Sexually mature individuals suffered the highest mortality though dependent calves also represented a significant portion of the toll probably because of the death of their mothers (Calzada *et al.*, 1994). DMV apparently did not persist as an endemic infection in Mediterranean striped dolphins after the epidemic was over (Van Bresseem *et al.*, 2001a). At the end of October 2006, an unusual high number of long-finned pilot whales (*Globicephala melas*) stranded along the coast of the Strait of Gibraltar (Fernandez *et al.*, 2008). The epizootic spread eastward and northeast till April 2007. The freshly dead *G. melas* examined by immuno-histology were positive for morbillivirus infection and a virus sequence very closely related to the DMV isolated in 1990-1992 was amplified by reverse-transcriptase polymerase chain reaction (RT-PCR) (Fernandez *et al.*, 2008). In early July 2007 dead or moribund striped dolphins and *G. melas* were detected in the Gulf of Valencia (Raga *et al.*, 2008). A DMV strain closely related to those isolated during the 1990-1992 epidemic was detected in seven of 10 *S. coeruleoalba* as well as in a juvenile *G. melas* by immunohistochemistry and RT-PCR (0.4% difference in the complete H gene) (Raga *et al.*, 2008; Tiwari, Banyard, Raga, and Barrett, unpublished work). In summer-autumn 2007, over two hundred striped dolphins were found dead along the coasts of Spain. Juvenile striped dolphins were significantly more frequently affected than adults in the current epizootic, likely because older dolphins were still protected by the immunity developed during the 1990-1992 epidemic (Raga *et al.*, 2008). The virus apparently reached the coasts of France in August (F. Dupraz, MARMAM message, day November 2007) and those of Italy (Ligurian Sea) in August-November 2007 (Garibaldi *et al.*, 2008). An isolated case was reported in Sardinia in 2007 (S. Appino, MARMAM message, day December 2007).

Taking into account that both 1990-1992 and 2006-2007 DMV epidemics started close to, or in the Gibraltar Strait (Raga *et al.*, 2008) and that DMV was circulating in the North Sea earlier that year (Wohlstein *et al.*, 2007), we believe that DMV-infected cetaceans, possibly *G. melas*, entered the Strait of Gibraltar and propagated the infection to striped dolphins, currently the most abundant odontocete in the Mediterranean (Notarbartolo di Sciarra *et al.*, 1993; Aguilar, 2000). *G. melas* has been proposed as the reservoir and vector of infection to other cetacean species of the North Atlantic (Duignan *et al.*, 1995; Van Bresseem *et al.*, 1998). Long-finned pilot whales are commonly seen in the western Mediterranean including the Strait of Gibraltar and may form mixed groups with striped dolphins (Duguy, 1989; Raga *et al.*, 1991, pers. observations). Interestingly, the outbreak of PMV mortalities in the NE Atlantic and North Sea was also contemporaneous with the 1990-1992 DMV epidemic. Whether these facts indicate that some environmental factor(s) triggered or influenced these outbreaks remains to be clarified. The dolphins that succumbed to the 1990-1992 epizootic were in a poor nutritional state with their lipid reserves estimated to be only about 60% of the usual values for this population, an energy depletion that could not be explained by the effect of the disease alone (Aguilar *et al.*, 1992; Aguilar and Raga, 1993). Also, the prevalence of ectoparasites and epizoids was much higher than reference values for this population, suggesting that the dolphins had gone through a period of restricted mobility (Aguilar and Raga, 1993; Aznar *et al.*, 1994). These unusual factors were associated with abnormally high temperatures and low rainfall conditions that prevailed in the western Mediterranean in the winter preceding the outbreak, and which apparently depressed the peak of productivity that occurs each year in early spring and determines the regional abundance of fish (Aguilar and Raga, 1993). Polychlorinated biphenyls (PCBs) loads of striped dolphins that died during the 1990-92 epidemic were higher than in the individuals that survived it (Kannan *et al.*, 1993; Aguilar and Borrell, 1994), and given their well-known immunosuppressive effect in mammals (Busbee *et al.*, 1999), it was suggested that the high PCB loads may have compromised immune response and increased the severity of the outbreak (Aguilar and Borrell, 1994). Though the role of pollutants in the 2007 epidemic remains to be investigated, recent pollutant data obtained through analyses of biopsies from apparently healthy striped dolphins in 1987-2002 suggested that PCB and DDT concentrations have gradually decreased in this species (Aguilar and Borrell, 2005). Recurrent epizootics with high mortalities among all age classes may have a profound impact on the population dynamics of Mediterranean *S. coeruleoalba*.

Morbilliviruses may also have been involved in the mass strandings of Fraser's dolphins (*Lagenodelphis hosei*) observed along the Atlantic coast of South America in 1997-1999 (Moreno *et al.*, 2003). Indeed, three of four *L. hosei* that died during these events had serum antibodies against DMV (Van Bresseem *et al.*, 2001a). The role of these viruses in cetacean mass-stranding worldwide should be considered.

BRUCELLA SPP.

Brucellosis is a globally distributed zoonotic disease of mammals that i.a. is pathogenic for the reproductive system and may cause abortion. The aetiologic agent is a Gram-negative, facultative intracellular bacteria of the genus *Brucella*. In the 1990s, previously unknown strains of *Brucella* were detected in free-ranging pinnipeds and cetaceans from the Americas, Europe, the Antarctic and western North Pacific as well as in captive bottlenose dolphins (Ewalt *et al.*, 1994; Ross *et al.*, 1994; Jahans *et al.*, 1997; Clavareau *et al.*, 1998; Miller *et al.*, 1999; Bricker *et al.*, 2000; Van Bresseem *et al.*, 2001b; Foster *et al.*, 2002; Prenger-Berninghoff *et al.*, 2003; Ohishi *et al.*, 2003, 2004; Davison *et al.*, 2008). Phylogenetic analyses of the marine *Brucella* isolates show that these form a new group that originate very near the *B. ovis* branch and that the seal, porpoise and dolphin brucellae have diverged a very long time ago, concurrent with the evolution of their hosts (Bourg *et al.*, 2007). On the basis of biological and molecular characteristics, Foster *et al.* (2007) proposed two *Brucella* species in marine mammals: *Brucella ceti* with cetaceans as preferred hosts and *Brucella pinnipedialis* with seals as the preferred hosts.

Disorders associated with brucellosis in cetaceans include placentitis, orchitis, abortion, lung infection and non-suppurative meningo-encephalitis (Miller *et al.*, 1999; Gonzalez *et al.*, 2002; Prenger-Berninghoff *et al.*, 2003; Ohishi *et al.*, 2004; Davison *et al.*, 2008). Brucellosis is endemic in several species and populations worldwide and, in Peruvian small cetaceans affects significantly more sexually mature than immature individuals (Van Bresseem *et al.*, 2001b, 2007a). When enzootic *B. ceti* infection may constitute a measurable limiting factor among environmental variables reducing population growth through negatively influencing reproduction (Van Bresseem *et al.*, 2001b, 2007a).

TOXOPLASMOSIS

The protozoan *Toxoplasma gondii* is widespread in marine mammals including odontocetes (reviewed in Dubey *et al.*, 2003). In cetaceans, toxoplasmosis was first reported in a Guiana dolphin (*Sotalia guianensis*) from Brazil (Bandoli and Oliveira, 1977). Infected free-ranging dolphins have been found in Europe, the Americas and the Caribbean (Bandoli and Oliveira, 1977; Domingo *et al.*, 1990; Cruickshank *et al.*, 1990; Inskeep *et al.*, 1990; Di Guardo *et al.*, 1995a,b; Resendes *et al.*, 2002; Cabezón *et al.*, 2004; Dubey *et al.*, 2007). They presented lympho-adenitis, lesions in the adrenal glands, myocarditis, acute interstitial pneumonia and non-suppurative encephalitis (Inskeep *et al.*, 1990; Dubey *et al.*, 2003; 2007). Disseminated toxoplasmosis was reported in a female *T. truncatus* and her calf from Florida, in a female Risso's dolphin (*Grampus griseus*) from the Mediterranean and in two beluga whales (*Delphinapterus leucas*) from the St Lawrence estuary, Quebec (Inskeep *et al.*, 1990; De Guise *et al.*, 1995; Resendes *et al.*, 2000). Two pregnant infected females (a captive *Tursiops aduncus* and the Mediterranean *Grampus griseus*) aborted fetuses with generalized toxoplasmosis (Jardine and Dubey, 2002; Resendes *et al.*, 2002). Clinical toxoplasmosis was generally associated with immunosuppression following a morbillivirus infection (Domingo *et al.*, 1990; Di Guardo *et al.*, 1995b; Schulman *et al.*, 1997) and/or high concentrations of environmental contaminants including PCBs (Mikaelian *et al.*, 2000). Serological data demonstrated that *T. gondii* circulates in Mediterranean *T. truncatus*, *D. delphis* and *S. coerulealba* (Cabezón *et al.*, 2004), coastal bottlenose dolphins from the USA (Dubey *et al.*, 2003, 2005) and belugas from Quebec (Mikaelian *et al.*, 2000).

T. gondii is the sole recognized species in the genus and, until recently, was composed of three major genotypes designed as types I-III that have emerged as dominant strains worldwide (Conrad *et al.*, 2005). Two new clades of *T. gondii*, named type A and X, were recently detected in the southern sea otter (*Enhydra lutris nereis*) from California and *Enhydra lutris kenyoni* from Washington State (Conrad *et al.*, 2005; Sundar *et al.*, 2008). Members of the *Felidae* family are the only known definitive hosts for *T. gondii* genotypes I-III but many mammals can be intermediate hosts if infected through the ingestion of contaminated food or water, or transplacentally. The origin and transmission of types A and X are not yet known. Some *T. gondii* infections of marine mammals, especially in coastal species, have been linked to freshwater runoffs contaminated with cat excrements containing oocysts (Miller *et al.*, 2002). Oocysts are very resistant and likely to survive in the sea and may be concentrated by molluscs (Dubey *et al.*, 2007). The infection of offshore species like *G. griseus* and *S. coerulealba* may be linked to ship run-off waters when hygienic conditions are low and when rodents, cats or contaminated soil are present onboard. The global maritime trade is thought to be responsible for the dissemination of *T. gondii* from its original niche in South America to the other continents (Lehman *et al.*, 2006).

Though the potential of *T. gondii* to impact cetacean populations has not yet been investigated, its ability to cause abortions and lethal systemic diseases renders it highly suspect. The possible reactivation of latent *T. gondii* infection during morbillivirus outbreaks may synergistically increase the severity of the viral disease and death rate. *T. gondii* is thought to contribute to the slow rate recovery of the southern sea otter population (Conrad *et al.*, 2005).

LOBOMYCOSIS AND LOBOMYCOSIS-LIKE DISEASE

Caused by a yeast-like organism known as *Lacazia loboi* (Taborda *et al.*, 1999) (syn. *Loboa loboi*; Caldwell *et al.*, 1975), lobomycosis (or lacaziosis) naturally affects humans, *T. truncatus* and *S. guianensis* (de Vries and Laarman, 1973; Caldwell *et al.*, 1975; Simões-Lopes *et al.*, 1993; Reif *et al.*, 2006; Van Bressem *et al.*, 2007b). *Lacazia loboi* cells found in *T. truncatus* infected tissues are significantly smaller than those found in humans, suggesting that the organism may not be identical in the two hosts (Haubold *et al.*, 2000). In humans, lobomycosis is a self-limited, chronic fungal infection of the skin, endemic in rural regions in South and Central America. Water, earth and vegetation are considered ecological habitats of the fungus and the agent accesses the skin by penetration or accidental trauma (Paniz-Mondolfi *et al.*, 2007). Patients with lobomycosis may have immunoregulatory disturbances possibly responsible for the lack of pathogen containment (Vilani-Moreno *et al.*, 2005; Honda *et al.*, 2007). Lobomycosis in dolphins is characterized by grayish, whitish to slightly pink, verrucous lesions, often in pronounced relief that may ulcerate (Migaki *et al.*, 1971). In *T. truncatus* from Florida the disease was associated with an impaired immune function possibly caused by anthropogenic factors (Bossart, 1984; Reif *et al.*, 2006). Variation in salinity and water temperature may also play a role (Reif *et al.*, 2006). The disease has been reported in dolphins from the Atlantic coast of the USA, Europe and South America (De Vries and Laarman, 1973; Caldwell *et al.*, 1975; Symmers, 1983; Simões-Lopes *et al.*, 1993; Reif *et al.*, 2006; Van Bressem *et al.*, 2007b). In South America, several cases highly reminiscent of lobomycosis were observed in free-ranging coastal *T. truncatus* and *S. guianensis*. In the absence of a histological diagnosis, the disease was called lobomycosis-like disease (LLD; Van Bressem *et al.*, 2007b). LLD seem to have been emerging recently over a relatively short span of time in Colombia, Ecuador, Peru, Brazil and Venezuela (Van Bressem *et al.*, 2007b, 2008a; Bermúdez *et al.*, 2008). Prevalence in inshore *T. truncatus* varied from 1.6% (Gulf of Guayaquil, Ecuador) to 20% (Tramandai estuary, Brazil) (Van Bressem *et al.*, 2007b; Moreno *et al.*, 2008). Most of the populations affected inhabited polluted waters harbouring ports or cities and, in the case of Guayaquil, intense shrimp farming activities (CPPS, 2000; CVC, 2003; UNEP, 2006; WHO/UNICEF/WSSCC, 2001). The origin of the fungus in coastal waters is unknown. The role of increased shipping in introducing *Lacazia loboi* to new ecological niches should be explored. Indeed, the discharge of water, sediments and biofilms from ships' ballast water tanks is a prominent vector of aquatic invasive species (Ruiz *et al.*, 2000; Drake *et al.*, 2007) and several cases of LLD have recently been diagnosed in small cetaceans in the vicinity of large ports in Colombia, Ecuador and Brazil (Van Bressem *et al.*, 2007b; 2008a). The appearance of new cases of LLD in the lagoon of Mayotte, southwest Indian Ocean and possibly off Amakusa-Shimoshima Island, East China Sea and gives weight to this hypothesis (Kiska and Van Bressem, 2008; Shirakihara *et al.*, 2008).

Lobomycosis and LLD may eventually contribute to the death of severely affected dolphins (Simões-Lopes *et al.*, 1993; Van Bressem *et al.*, 2007b; Bermúdez-Villapol *et al.*, 2008). All dolphins that died with extensive lobomycosis or LLD were adult, and all LLD affected individuals sighted have been large, presumably adult, individuals, except for a calf *S. guianensis* from the Lagamar estuary, Brazil (Van Bressem *et al.*, 2008a). As the rates of increase of dolphin population size appears to be more sensitive to the non-calf survival rate than to the calf survival rate (Reilly and Barlow, 1986), lobomycosis and LLD are likely to lower the growth of coastal populations, and may have a significant impact on small, isolated ones.

GENITAL PAPILLOMAVIRUSES

Papillomaviruses (PVs), small, non-enveloped, double-stranded DNA viruses (family *Papillomaviridae*) are epitheliotropic pathogens which may induce proliferation of the stratified squamous epithelia of the skin and mucosa and cause lesions known as warts, papillomas and condylomas in mammals and birds (Antonsson and Hansson, 2002; Terai *et al.*, 2002; Bernard, 2005).

Genital papillomatosis was observed in sperm whales (*Physeter macrocephalus*) caught off Iceland (Lambertsen *et al.*, 1987), in dusky dolphins (*Lagenorhynchus obscurus*), long-beaked common dolphins (*Delphinus capensis*), common bottlenose dolphins and Burmeister's porpoises (*Phocoena spinipinnis*) from Peru (Van Bressem *et al.*, 1996), in *T. truncatus* from Florida (Bossart *et al.*, 2005) and Cuba (Cruz *et al.*, 2006) and in a Guiana dolphin from Brazil (M. Marcondes, pers. commn. May 2007) as well as in *T. truncatus* kept in captivity in Europe and the USA (Bossart *et al.*, 2005; Rector *et al.*, 2006). Genital lesions macroscopically and microscopically consistent with PV-induced papillomas have also been seen in *P. phocoena*, *D. delphis* and *S. coeruleoalba* stranded along the coasts of the United Kingdom (Jepson, P.D., pers. obs.). Prevalence of the disease ranged from high to medium in cetaceans from Peru (33-66.7%; Van Bressem *et al.*, 1996), Cuba (28.7%; Cruz *et al.*, 2006) and Iceland (10%; Lambertsen *et al.*, 1987). Sexual variation in wart prevalence was found in *L. obscurus* and *P. spinipinnis* with males being two and three times more infected than females, respectively (Van Bressem *et al.*, 1996). The same possibly occurs in *T. truncatus* from Cuba (Cruz *et al.*, 2006).

Phocoena spinipinnis papillomavirus type 1 and another still uncharacterized PV cause genital warts in *P. spinipinnis* (Van Bressem *et al.*, 2007c). At least three PVs are associated with genital papillomatosis in *T. truncatus*

from Europe and Atlantic USA (Chiers *et al.*, 2004; Rector *et al.*, 2006; Rehtanz *et al.*, 2006;). Other still uncharacterised PVs were detected in papillomas from Peruvian *T. truncatus* and *L. obscurus* (Cassonnet *et al.*, 1998). The high prevalence (48.5%) of genital warts in 33 Burmeister's porpoises examined in 1993-1995 (Van Bressem *et al.*, 1996) and the detection of PV sequences in 5 of 7 genital warts sampled in this study indicate that PV infection is frequent in this species. Porpoises may become infected early in life through vertical and horizontal transmission (Van Bressem *et al.*, 1996, 2007c). Genital lesions of sufficient severity that may impede, or at least, hamper copulation were seen in two of 20 male *P. spinipinnis* but not in other species from Peru. One sperm whale also suffered extensive genital papillomatosis (Lambertsen *et al.*, 1987). PVs in some circumstances (especially if non-randomly distributed) may exert an indirect impact on the dynamics of this population (Lambertsen *et al.*, 1987; Van Bressem *et al.*, 1999).

TATTOO SKIN DISEASE

Tattoo skin disease (TSD) is characterised by very typical, irregular, grey, black or yellowish, stippled lesions that may occur on any part of the body but show a preferential corporal distribution depending on the species (Van Bressem and Van Waerebeek, 1996). It has been observed in several species of free-ranging Delphinidae, Phocoenidae and Ziphiidae worldwide as well as in captive bottlenose dolphins (*Tursiops truncatus*) and a bowhead whale (*Balaena mysticetus*) from Alaska (see Van Bressem *et al.*, 1999, 2008b; Bracht *et al.*, 2006). TSD is caused by poxviruses (Flom and Houk, 1979, Geraci *et al.*, 1979, Van Bressem *et al.*, 1993, 2006) that may belong to a new genus of *Chordopoxvirinae*, but have a common, most immediate ancestor with terrestrial poxviruses of the genus *Orthopoxvirus* (Bracht *et al.*, 2006; Pearce *et al.*, 2008). These viruses are thought to induce humoral immunity that protects neonates and young calves from the disease (Smith *et al.*, 1983, Van Bressem and Van Waerebeek, 1996; Van Bressem *et al.*, 2006). Individual tattoo lesions may persist for months or years and recurrence may occur (Van Bressem *et al.*, 2003).

A still unpublished study (Van Bressem *et al.*, 2008b) suggests that the epidemiological pattern of TSD may be an indicator of a degrading and stressful aquatic environment. In odontocetes that had died a traumatic death (TD) as well as in free-ranging *T. truncatus* from the Sado estuary, Portugal, prevalence varied with the age class, being generally null in neonates and young calves, peaking among juveniles and decreasing in adults. In contrast, prevalence increased or remained high in adult dolphins and porpoises that had died in poor health (PH) as well as in coastal *P. spinipinnis* from Peru. Prevalence of TSD was significantly higher in adult dolphins and porpoises from the PH category than in adults from the TD category. In coastal dolphins and porpoises, prevalence did not decrease significantly in adults while it did in adult neritic and oceanic cetaceans. These findings suggest that adult odontocetes that died in PH and Peruvian *P. spinipinnis* had a depressed immune system with reduced capacity to fight infections.

Clinical and epizootiological data do not indicate that poxvirus infection induces a high mortality rate when enzootic (Van Bressem and Van Waerebeek, 1996; Van Bressem *et al.*, 2003). However, it may kill neonates and calves without protective immunity and thus interfere with host population dynamics. An apparent, severe, generalized poxvirus infection in a calf *S. guianensis* stranded alive along the coast of Pará state, Brazil in March 2008 gives weight to this hypothesis. Super-infection by fungi or bacteria may exacerbate the disease (Smith *et al.*, 1983; Van Bressem *et al.*, 2003, 2007b).

INFECTIOUS DISEASES IN *PHOCOENA PHOCOENA* FROM THE NORTH AND BALTIC SEAS

Recent studies have demonstrated a significant association between chronic PCB exposure and infectious diseases (including parasitic, bacterial and mycotic pneumonia and generalized bacterial infections) in harbour porpoises from the North and Baltic Seas (Jepson *et al.*, 1999, Siebert *et al.*, 1999; Das *et al.*, 2004; Jepson *et al.*, 2005; Hall *et al.*, 2006; Valderrama *et al.*, 2008). The infectious disease group of harbour porpoises from the British Isles had a significantly higher sum of the concentrations of 25 individual chlorobiphenyl congeners ($\Sigma 25CBs$) than the TD group. The mean $\Sigma 25CBs$ level in TD animals was 13.6mg/kg lipid, whereas the mean level in the PH group was 27.6mg/kg lipid. Adult females in both groups had significantly lower $\Sigma 25CBs$ levels than adult males because of off-loading of organochlorines during gestation and lactation (Jepson *et al.*, 2005). The full impact of PCBs on harbour porpoise populations in European waters is unknown.

EMERGING SKIN DISEASES IN SOUTH AMERICA

Miscellaneous skin diseases of unknown aetiologies have been reported from several regions in South America (Flach, 2006; Brownell *et al.*, 2007; Van Bressem *et al.*, 2007b, 2008a; Flach *et al.*, 2008; S. Siciliano, unpublished data). Many are associated with unrelated wounds, scars and tooth rakes. Though these lesions may eventually heal, their effect on the overall health, fitness and reproductive success of individuals is unknown. A possible relation with poor water quality is suggested as lesions were observed in individuals living in waters heavily contaminated by organic material

originating from fishmeal factories, municipal sewage and aquaculture as well as ballast water from cargo ships (Van Bressem *et al.*, 2007b).

Several bacteria including the common opportunistic pathogens *Vibrio* spp., *Aeromonas hydrophila* and *Pseudomonas aeruginosa* that thrive in the aquatic environment have been isolated from skin lesions in cetaceans worldwide (reviewed in Van Bressem *et al.*, 2008c). In the lower Chesapeake Bay, USA, coastal ecosystems are frequently invaded by micro-organisms from ballast waters with an estimated 10^{20} bacteria (including *V. cholerae*) and viruses annually discharged (Drake *et al.*, 2007). Salmon and shrimp farms heavily use prophylactic antibiotics resulting in the increase of antibiotic resistance in fish and human pathogens including *Vibrio cholerae* and *Aeromonas hydrophila* (Weber *et al.*, 1994; Rhodes *et al.*, 2000; Cabello, 2004, 2006). The combination of aquaculture, intense ship traffic and the release of untreated run-off waters likely favour a high density of pathogenic bacteria, some of which are antibiotic-resistant, that may infect wounds. In this context, we believe that combined microbial and organic contamination is possibly at the origin of the emergence of frequent miscellaneous skin lesions in coastal cetaceans from South America. The worldwide dissemination of fungi and antibiotics-resistant marine bacteria through water ballast and sea product transportation may favour the emergence and spread of skin diseases to other areas of the world.

CONCLUSIONS

As for any other mammals, cetaceans are normally infected by a wide variety of pathogens that are order or family-specific (i.e. morbilli-, papilloma- and poxviruses as well as *Brucella ceti*) or opportunistic (bacteria, fungi, protozoans). Infection by specific pathogens are likely to have occurred for thousands of years with some equilibrium between populations and pathogens as in other species (Begon *et al.*, 1996; Blaser and Kirschner, 2007). Environmental degradation including biological and chemical pollution, climate change, fisheries and heavy boat traffic, may have changed this pattern by lowering the immune response of the populations, shortening the food supply, increasing stress and facilitating the introduction of alien pathogens, among others. The number and severity of diseases have increased, in part because of more dedicated research in this field (Gulland and Hall, 2007). However, taking this bias into account, there has been an increase in the frequency and severity of some diseases as for example those resulting from exposure to harmful algal blooms (HABs) as well as lobomycosis and whitish velvety cutaneous lesions in cetaceans from the Americas to cite a few (Reif *et al.*, 2006; Gulland and Hall, 2007; Van Bressem *et al.*, 2007b; Flach *et al.*, 2008; Siciliano *et al.*, 2008).

There is conclusive evidence that chemical pollution has increased the emergence and severity of several diseases (Martineau *et al.*, 1988; Aguilar and Borrell, 1994; De Guise *et al.*, 1998; Jepson *et al.*, 1999, 2005; Ross, 2002; Hall *et al.*, 2006; Van Bressem *et al.*, 2008b). Biological pollution is an emerging issue (Mos *et al.*, 2006; Pereira *et al.*, 2007; Van Bressem *et al.*, 2007b). Coastal cetaceans seem particularly more at risk because, besides living in an environment that is prone to be more polluted, they may face higher concentrations of pathogenic micro-organisms in the waters they inhabit and may also be more often wounded and stressed by interactions with boats and fisheries. With a depressed immune system and a ruptured skin, these animals would make ideal targets for opportunistic pathogens. The continued degradation of the marine environment and increased pressure on its inhabitants will possibly provoke more frequent epizootics, help disseminate fungal and bacterial diseases and increase the prevalence and severity of infectious illnesses in cetaceans worldwide.

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