ABSTRACT: We reviewed prominent emerging infectious diseases of cetaceans, examined their potential to impact populations, re-assessed zoonotic risk and evaluated the role of environmental stressors. Cetacean morbilliviruses and papillomaviruses as well as *Brucella* spp. and *Toxoplasma gondii* are thought to interfere with population abundance by inducing high mortalities, lowering reproductive success or by synergistically increasing the virulence of other diseases. Severe cases of lobomycosis and lobomycosis-like disease (LLD) may contribute to the death of some dolphins. The zoonotic hazard of marine mammal brucellosis and toxoplasmosis may have been underestimated, attributable to frequent misdiagnoses and underreporting, particularly in developing countries and remote areas where carcass handling without protective gear and human consumption of fresh cetacean products are commonplace. Environmental factors seem to play a role in the emergence and pathogenicity of morbillivirus epidemics, lobomycosis/LLD, toxoplasmosis, poxvirus-associated tatt-too skin disease and, in harbour porpoises, infectious diseases of multifactorial aetiology. Inshore and estuarine cetaceans incur higher risks than pelagic cetaceans due to habitats often severely altered by anthropogenic factors such as chemical and biological contamination, direct and indirect fisheries interactions, traumatic injuries from vessel collisions and climate change.

KEY WORDS: Emerging diseases · Cetaceans · Morbilliviruses · Poxviruses · Papillomaviruses · *Brucella* spp. · *Lacazia loboi* · *Toxoplasma gondii* · Environmental stressors · Zoonosis
INTRODUCTION

Emerging infectious diseases (EIDs) are defined as those that (1) are newly recognized, (2) are evolving, (3) have recently shown an increase in incidence or expansion into new geographic locations or vectors, (4) have moved from one host species to another, (5) have increased in impact or severity, (6) or have undergone a change in pathogenicity (Bengis et al. 2004). EIDs are frequently the result of a change in the ecology of host and/or pathogen and are often driven by anthropogenic environmental modifications such as encroachment, animal movements across borders and global climate changes (Daszak et al. 2001, Bengis et al. 2004, Cunningham 2005, Munson et al. 2008, Datta et al. 2009, Johnson et al. 2009). Such diseases may trigger massive mortalities, constrain the growth of wild animal populations, increase the risk of extinction of small populations in combination with other factors and provoke loss of biodiversity (Plowright 1982, Thorne & Williams 1988, Daszak et al. 2000, Johnson et al. 2009).

In recent years EIDs have been reported in several cetacean species and populations worldwide provoking large-scale die-offs, affecting reproduction, causing disfiguring skin diseases and, in some cases, zoonosis. Here we review prominent cetacean EIDs, assess their potential to significantly impact populations as well as their zoonotic risk and consider the possible role of environmental factors.

VIRAL DISEASES

Cetacean morbillivirus

Cetacean morbillivirus (CeMV) belongs to the genus Morbillivirus (single, negative-strand RNA viruses of subfamily Paramyxovirinae, family Paramyxoviridae) and includes 3 strains: the dolphin morbillivirus (DMV, first isolated in Mediterranean striped dolphins Stenella coeruleoalba; Van Bressem et al. 1991), porpoise morbillivirus (PMV, first isolated in harbour porpoises Phocoena phocoena from Northern Ireland; McCullough et al. 1991) and pilot whale morbillivirus (PWMV, detected by PCR in a long-finned pilot whale Globicephala melas from New Jersey, USA; Taubenberger et al. 2000).

CeMV is endemic in several species of cetaceans worldwide (Duignan et al. 1995a, Van Bressem et al. 2001a). Pilot whales Globicephala spp. and other gregarious species are thought to be reservoirs of infection, act as vectors and spread the virus to other species with which they associate (Duignan et al. 1995a, Van Bressem et al. 1998). In the absence of population immunity, CeMV may trigger epidemics of lethal disease characterized by pneumonia, nonsuppurative meningo-encephalitis and prominent lymphoid cell depletion (Domínguez et al. 1990, Duignan et al. 1992).


The 1900–1992 DMV epidemic started along the central coast of Spain in 1990 and ended in Turkey and the Greek Islands in 1992, affecting predominantly Stenella coeruleoalba, currently the most abundant odontocete in the Mediterranean (Aguilar & Raga 1993, Aguilar 2000). Sexually mature individuals suffered the highest mortality, though dependent calves also represented a significant portion of the toll, possibly indirectly because of the deaths of their mothers (Calzada et al. 1994). DMV apparently did not persist as an endemic infection in Mediterranean striped dolphins after the epidemic terminated (Van Bressem et al. 2001a), presumably because the abundance (117 880, CI = 68 379 to 148 000) in the western Mediterranean Sea (Forcada et al. 1994) was too low to support endemic infection. Large numbers of striped dolphins have died in fisheries, which has greatly reduced their numbers (Aguilar 2000).

Between October 2006 and April 2007, at least 27 Globicephala melas stranded along the southern Spanish Mediterranean coast and the Balearic Islands (Fernández et al. 2008). In early July 2007 dead or moribund Stenella coeruleoalba and G. melas were found in the Gulf of Valencia (Raga et al. 2008) (Fig. 1). Morbillivirus lesions and antigen were detected in all 9 examined G. melas and in 13 of 17 S. coeruleoalba. Fungal and bacterial superinfections were observed in 1 individual of each species. A DMV strain closely related to the virus isolated during the 1990 to 1992 epidemic was detected in 7 of 10 S. coeruleoalba as well as in 9 G. melas by reverse transcription-PCR (Fernández et al. 2008, Raga et al. 2008). During the summer and autumn 2007, more than 200 S. coeruleoalba were found dead along the coasts of
Spain. Juveniles were more frequently affected (Student’s test, \( t = 2.14, \text{df} = 49, p = 0.037 \)) than adults in the 2006–2007 outbreak, probably because older dolphins were still protected by the immunity developed during the 1990–1992 epidemic (Raga et al. 2008). The virus apparently reached the French Mediterranean coast in August and Italy's Ligurian Sea coast in the period August to November 2007 (Garibaldi et al. 2008).

Taking into account that both the 1990–1992 and 2006–2007 DMV epidemics started close to, or in, the Gibraltar Strait and that DMV was circulating in the North Sea in January 2007 (Wohlsein et al. 2007), we believe that DMV-infected cetaceans, possibly *Globicephala melas*, entered the Strait of Gibraltar and transmitted the infection to *Stenella coeruleoalba* with which they occasionally form mixed groups (Raga et al. 1991). Interestingly, the outbreak of PMV mortalities in the NE Atlantic and North Sea was also contemporaneous with the 1990 to 1992 DMV epidemic (Visser et al. 1993, Kennedy 1998).

Whether some environmental factor(s) triggered or influenced these outbreaks remains to be clarified. The *Stenella coeruleoalba* that succumbed to the 1990–1992 epidemic were in a poor nutritional state, with lipid reserves estimated to be only about 60% of usual values for this population, an energy depletion that could not be explained by the effect of the disease alone (Aguilar & Raga 1993). Also, the prevalence of ectoparasites and epibionts was much higher than reference values for this population, suggesting that during the epidemic more dolphins were susceptible to infestations, possibly because they were immunodepressed by the disease and were suffering from behavioural alterations such as reduced swimming speed (Aguilar & Raga 1993, Aznar et al. 2005). The abnormally high sea-surface temperatures (SSTs) and low rainfall conditions in the winter preceding the 1990–1992 outbreak in the western Mediterranean apparently depressed peak productivity that occurs each year in early spring and determines the regional abundance of fish (Aguilar & Raga 1993). This epidemic also started in regions containing unusually large numbers of inbred dolphins that were possibly more susceptible to diseases (Valsecchi et al. 2004).

Polychlorinated biphenyl (PCB) loads in *Stenella coeruleoalba* that died during the 1990–1992 epidemic were significantly higher (Mann-Whitney test, \( p < 0.001 \)) than in individuals that survived and, given their well-known immunosuppressive effect in mammals, it was suggested that PCBs may have compromised immune response and increased the severity of the outbreak (Aguilar & Borrell 1994). Though the role of environmental contaminants in the 2007 epidemic remains inconclusive, recent pollutant data obtained through analyses of biopsies from apparently healthy striped dolphins in 1987 to 2002 suggested that PCB and DDT concentrations have gradually decreased (Aguilar & Borrell 2005).

Thus, several environmental factors, i.e. fisheries interactions, inbreeding, migration, high contaminant...
loads, higher SSTs and limited prey availability may have synergistically interacted to increase the severity of the disease and may favor recurrent epidemics with a profound, accumulative impact on the population dynamics of Mediterranean *Stenella coeruleoalba*. Morbilliviruses are generally order specific (Barrett 1999) and there are no reports of CeMV disease in humans, suggesting that CeMV is not zoonotic.

**Genital papillomaviruses**

Papillomaviruses (PVs), small, non-enveloped, double-stranded DNA viruses (family *Papillomaviridae*) are epitheliotropic pathogens that may induce proliferation of the stratified squamous epithelia of the skin and mucosae and cause lesions known as warts, papillomas and condylomas in mammals and other vertebrates (Howley & Lowy 2001).

Genital papillomatosis has been observed in 9.7% of 31 sperm whales *Physeter macrocephalus* from Iceland (Lamberts et al. 1987), in 66.7% of 78 dusky dolphins *Lagenorhynchus obscurus*, 50% of 10 long-beaked common dolphins *Delphinus capensis*, 33% of 9 *Tursiops truncatus* and 48.5% of 33 Burmeister’s porpoises *Phocoena spinipinnis* from Peru (Fig. 2) (Van Bressem et al. 1996), in 3 *T. truncatus* from Florida (Bossart et al. 2005) and 28.7% of 251 *T. truncatus* from Cuba (Cruz et al. 2006), and in 1 Guiana dolphin *Sotalia guianensis* from Brazil (M. Marcondes pers. comm.). It was also diagnosed in captive *T. truncatus* in Europe and the USA (Bossart et al. 2005, Rector et al. 2008). Genital lesions macroscopically and microscopically consistent with PV-induced papillomas have also been seen in *P. phocoena*, short-beaked common dolphin *Delphinus delphis* and *Stenella coeruleoalba* from the British Isles (P. D. Jepson pers. obs.). Genital papillomatosis does not seem to occur in small cetaceans from New Zealand (P. J. Duignan pers. obs.) and the Spanish Mediterranean coast (J. A. Raga pers. obs.). Sexual variation in wart prevalence was found in *L. obscurus* and *P. spinipinnis*, with males being 2 and 3 times more-often infected than females, respectively (Van Bressem et al. 1996). Males may also be more frequently affected in Cuban *T. truncatus* (Cruz et al. 2006).

*Phocoena spinipinnis* papillomavirus type 1 (PsPV-1) and another still uncharacterized PV caused genital warts in *P. spinipinnis* (Van Bressem et al. 2007a). Three PVs (*Tursiops truncatus* [TtPV] papillomavirus type 1, 2 and 3) were associated with genital papillomatosis in dolphins from Europe and Atlantic USA (Rector et al. 2008). Other still uncharacterised PVs were detected in genital warts from Peruvian *T. truncatus* and *Lagenorhynchus obscurus* (Cassonnet et al. 1998). Recombination of an ancestor of PsPV-1 and an ancestor of TtPV-2 may have generated the common ancestor of TtPV-1 and -3 (Rector et al. 2008).

The high prevalence of genital warts in 33 *Phocoena spinipinnis* from Peru examined in 1993 to 1995 and

![Fig. 2. Phocoena spinipinnis. (a) Whitish-coloured warts in the vagina of porpoise JAS-50 (arrowheads). (b) Detection of group-specific papillomavirus antigen in superficial keratinocytes of a genital wart from porpoise JAS-44 using a polyclonal rabbit immune serum against disrupted particles of human papillomavirus type 1 (HPV–1). Nuclear staining (arrowheads) is visible in a cluster of differentiating cells and in adjacent, more superficially differentiated cells (magnification ×350)](image)
the detection of PV sequences in 5 of the 7 genital warts examined indicated that PV infection is very frequent in this population. Though PVs are ubiquitous and commonly found on the skin and mucosae of the genital tract of humans and other animal species, clinical disease only develops in a small minority of them and it is believed that additional factors (immune status, genetic predispositions, nutritional status, environmental factors) are involved in the process of tumour development (Mougin et al. 2001, Antonsson & Hansson 2002). The high prevalence of genital papillomas in *P. spinipinnis* may point to immune depression in this population. This would be consistent with results of a recent epidemiological study covering 17 cetacean species where the highest prevalence of poxvirus-associated tattoo skin disease (TSD) was also encountered in Peruvian *P. spinipinnis* (Van Bressem et al. 2009a). The significantly higher prevalence of genital warts in male *P. spinipinnis* may reflect the accumulation of immunosuppressive lipophilic contaminants through life in adult males and the depuration of their contaminant loads to their calves (Jepson et al. 2005, Wells et al. 2005). Chronic stress from intense fishery interactions may also play a role in the development of papillomas in cetaceans by depressing the immune system and acting synergistically with immunosuppressive contaminants (Clark et al. 2006, Hall et al. 2006, Martineau 2007).

*Phocoena spinipinnis* may become infected early in life through vertical and horizontal transmission (Van Bressem et al. 1996, 2007a). Genital warts of sufficient severity that may impede, or at least hamper, copulation affected 2 of 20 male *P. spinipinnis* examined but were not seen in other Peruvian small cetaceans. A sperm whale has also been reported to suffer extensive genital papillomatosis (Lambertsen et al. 1987). PVs in some circumstances (especially if non-randomly distributed) may exert an indirect impact on population dynamics (Lambertsen et al. 1987, Van Bressem et al. 1999).

As in other mammals (Howley & Lowy 2001) cetacean papillomaviruses appear to be species-specific (Van Bressem et al. 2007a, Rector et al. 2008). However, as recombination can occur between the *Papillomaviridae* leading to viable recombinants (Varsani et al. 2006, Rector et al. 2008), precaution is recommended when sampling genital warts in live cetaceans or fresh carcasses.

**Cetacean poxviruses and TSD**

TSD is characterised by very typical, irregular, grey, black or yellowish, stippled cutaneous lesions referred to as tattoos that may occur on any body part but show a preferential corporal distribution depending on the species (Fig. 3). Individual tattoo lesions may persist for months or years and recurrence is possible (Van Bressem & Van Waerebeek 1996, Van Bressem et al. 2003). TSD has been reported in 17 free-ranging species, including Delphinidae (n = 13), Phocoenidae (n = 2), Ziphiidae (n = 1) and Balaenidae (n = 1), as well as in captive *Tursiops truncatus* and Indo-Pacific bottlenose dolphins *Tursiops aduncus* (Van Bressem et al. 1999, 2007b, 2009a, Bracht et al. 2006). It is caused by poxviruses that may belong to a new genus of the subfamily *Chordopoxvirinae* (family *Chordopoxviridae*), but have a common, most immediate ancestor with terrestrial poxviruses of the genus *Orthopoxvirus* (Bracht

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**Fig. 3. Delphinus capensis.** Tattoo lesions (arrowheads) on the left flank of long-beaked common dolphin MFB-675 caught off central Peru in 1994
et al. 2006). TSD poxviruses are thought to induce humoral immunity protecting neonates and young calves (Smith et al. 1983, Van Bressem & Van Waerebeek 1996). A recent study in 17 cetacean species (1392 individuals) from 3 oceans and contiguous seas showed a common pattern for endemic TSD: a significant increase in prevalence in juveniles compared to calves, presumably due to juveniles that had lost maternal humoral immunity, as well as a significantly higher prevalence in juveniles than in adults, possibly because a high percentage of adults had acquired active immunity following infection. This epidemiological pattern was found inverted in samples of poor health odontocetes, possibly the result of a depressed immune system (Van Bressem et al. 2009a).

When endemic, TSD does not appear to induce a high mortality rate (Van Bressem & Van Waerebeek 1996, Van Bressem et al. 2003). However, it may kill neonates and calves without protective immunity and thus could interfere with host population dynamics (Van Bressem et al. 1999). A severe, generalized poxvirus infection, as apparent from a large number of disseminated tattoos in a calf *Sotalia guianensis* that live-stranded on the coast of Pará state, Brazil, in March 2008 (S. Siciliano pers. obs.) lends weight to this hypothesis. Super-infection by fungi or bacteria may exacerbate TSD and contribute to mortalities (Smith et al. 1983, Van Bressem et al. 2003, 2007b). The role of environmental factors in the course, severity and epidemiology of the disease is unknown. We hypothesize that immunosuppressive contaminants and severe, chronic stress levels among sociable cetaceans in areas with persistently elevated mortality rates from bycatch may contribute to higher prevalences of TSD in adults, cases of progressive TSD as evidenced by very large tattoo lesions, as well as to recurrence.

Although TSD poxviruses seem to be cetacean-specific (Bracht et al. 2006, Pearce et al. 2008) and no transmission to humans has been reported, it is recommended that persons (especially if immuno-compromised) who handle live cetaceans or fresh carcasses with tattoo lesions use gloves. Further research should include serological tests in conjunction with examination of tattoo lesions by electron microscopy and molecular techniques.

**BRUCELLOSIS**

Brucellosis is a globally distributed zoonotic disease of mammals that is pathogenic for the reticuloendothelial, reproductive, musculoskeletal and cutaneous systems and may cause generalized infection with sepsis in humans (Corbel 1997). The aetiological agent is a Gram-negative, facultative intracellular bacterium of the genus *Brucella*. From the 1990s onward, previously unknown strains of *Brucella* were detected in free-ranging pinnipeds and cetaceans from the Americas, Europe, Japan, New Zealand, the Solomon Islands and the Antarctic as well as in captive *Tursiops truncatus* (Ewalt et al. 1994, Ross et al. 1994, Ohishi et al. 2003, Duignan et al. 2005, Tachibana et al. 2006, Hernández-Mora et al. 2008). Phylogenetic analyses show that the marine *Brucella* isolates form a new group that originates very near the *B. ovis* branch and that the seal, porpoise and dolphin strains 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 2 *Brucella* species in marine mammals, *B. ceti* and *B. pinnipedialis* with, respectively, cetaceans and seals as preferred hosts. Groussaud et al. (2007) further suggested that brucellae isolated from cetaceans constitute 2 species with different preferred hosts, i.e. *B. phocoenae* in porpoises and *B. delphini* in dolphins.


Brucellosis is endemic in several species and populations worldwide and, in Peruvian small cetaceans, affects significantly more sexually mature than immature individuals (Van Bressem et al. 2001b). *Brucella ceti* infection may measurably limit recruitment by compromising the normal functioning of male and female reproductive systems, inducing abortions and by killing neonates and sexually mature individuals. This could have significant consequences for endangered cetaceans such as Hector’s dolphins *Cephalorhynchus hectori hectori* and Maui’s dolphins *C. hec-
and serological tests for common diseases in Peru, no consultations and standard hematologic, parasitologic (Waerebeek pers. obs.). Despite numerous medical admissions to grossly identical clinical symptoms (K. Van summation, queried about her progressive weight loss, mediated in the trade of small cetaceans for human con- ger at the Pucusana port in central Peru who special-
Contemporaneously, a middle-aged female fishmon-
fatigue, anorexia and dramatic weight loss (>10%).
profuse night sweats, cephalgias, persistent chronic
severe myalgias and back aches, undulating fever,
and, in retrospect, consistent with brucellosis (Corbel clapham & van Waerebeek 2007, alfaro-shiguet et al. 2008) without any hygienic precautions. One of the authors (K. van waerebeek) suffered an undiagnosed but debilitating chronic disease in 1990 to 1992, a period when he necropsied hundreds of freshly dead small cetaceans in Peruvian fish markets (see van waerebeek & reyes 1994) without gloves or protective clothing. Clinical manifestations were severe, systemic and, in retrospect, consistent with brucellosis (corbel 1997), including seizures with loss of consciousness, severe myalgias and backaches, undulating fever, profuse night sweats, cephalgias, persistent chronic fatigue, anorexia and dramatic weight loss (>10%). Contemporaneously, a middle-aged female fishmonger at the Pucusana port in central Peru who special- ized in the trade of small cetaceans for human con-
sumption, queried about her progressive weight loss, admitted to grossly identical clinical symptoms (K. van waerebeek pers. obs.). Despite numerous medical consultations and standard hematologic, parasitologic and serological tests for common diseases in Peru, no
diagnosis was made for either patient. Generalized symptoms and prolonged illness have also been reported in several marine mammal workers during a global survey. Though most cases were not further diagnosed, brucellosis was suspected in at least 2 workers (Hunt et al. 2008). Restrictions should be applied to programmes where tourists are allowed to swim and interact closely with captive dolphins when Brucella spp. could be circulating in these colonies. Dolphins should at least be screened serologically for Brucella spp. before they are used for such purposes.
The role of environmental factors in the emergence of marine mammal brucellosis is unknown.

TOXOPLASMOSIS

Toxoplasma gondii is an obligate intracellular protozoan parasite belonging to the Apicomplexa phylum that causes toxoplasmosis in human and other warm-blooded animals worldwide including cetaceans (Dubey et al. 2003). Wild and domestic felids are the only animals known to serve as definitive hosts for T. gondii genotypes I through III, but many mammals can be intermediate hosts (Miller et al. 2008). Infection occurs through the ingestion of contaminated food or water, or transplacentally. In cetaceans, toxoplasmosis was first reported in a Sotalia guianensis from Brazil (Bandoli & de oliveira 1977). Infected free-ranging odontocetes have been reported in Europe, the americas and the Caribbean. They presented lymphadenitis, necrotizing adrenal adenitis, myocarditis, acute interstitial pneumonia, non-suppurative encephalitis (Fig. 4) and systemic disease. Transplacental fetal infection was reported in a free-ranging Risso’s dolphin Grampus griseus and a captive Tursiops aduncus (reviewed in Dubey et al. 2003). Toxoplasmosis was often, though not always, associated with immuno-suppression following a morbillivirus infection and/or high concentrations of environmental contaminants including PCBs (Di guarda et al. 1995, Mikaelian et al. 2000).

Toxoplasma gondii is the sole recognized species in the genus and, until recently, was composed of 3 major genotypes designed as Types I through III that have emerged as dominant strains worldwide (Conrad et al. 2005). Two new clades of T. gondii, named Type A and Type X, were recently detected in a Pacific harbour seal Phoca vitulina richardi, a California sea lion Zalophus californianus, southern sea otters Enhydra lutris nereis and northern sea otters E. lutris kenyoni from California and Washington State, as well as in a California mussel Mytilus californianus, the wild coastal felids Lynx rufus and Puma concolor, and a red fox Vulpes vulpes dwelling near Monterey and Estero.
Bays, California. These observations suggest that feline fecal contamination is flowing from land to sea through surface run-off and that sea otters may be infected through the consumption of infected marine invertebrates (Conrad et al. 2005, Miller et al. 2008, Johnson et al. 2009).

We believe that the infection of offshore species like *Grampus griseus* and *Stenella coeruleoalba* could be linked to ship run-off waters when hygienic conditions are poor and when rodents, cats or contaminated soil are present onboard. Waterborne transmission of *Toxoplasma gondii* may be much more common than previously thought (Jones & Dubey 2009) and the global maritime trade is thought to be responsible for the dissemination of *T. gondii* from its original niche in South America to other continents (Lehmann et al. 2006).

Degradation of the marine environment and food reduction may also play a role in the emergence of toxoplasmosis in marine mammals. A recent study in *Enhydra lutris nereis* from California suggests that high levels of *T. gondii* infection may be an adverse consequence of dietary specialization related to a resource-limited coastal system (Johnson et al. 2009).

Though the potential of *Toxoplasma gondii* to affect cetacean populations has not yet been investigated, its ability to cause lethal systemic diseases and abortions renders it highly suspect. *T. gondii* may contribute to the slow recovery rate of the southern sea otter population (Conrad et al. 2005). Besides, the possible reactivation of latent *T. gondii* infection during morbillivirus outbreaks may synergistically increase the severity and death rate of this viral disease. High levels of co-infection with the hemoprotozoan parasite *Babesia* spp. increased the severity of canine distemper virus epidemics in African lions *Panthera leo* (Munson et al. 2008).

Toxoplasmosis is zoonotic globally. Humans become infected with *Toxoplasma gondii* mainly by ingesting uncooked meat containing viable tissue cysts or by ingesting food or water contaminated with oocysts from the feces of infected Felidae (Jones & Dubey 2009). The consumption of raw or incompletely cooked cetacean meat, customary in some countries (Muckle et al. 2001, Clapham & Van Waerebeek 2007), likely represents an incompletely assessed health hazard. Marine mammals may serve as sentinels of protozoan pathogen pollution.

**LOBOMYCOSIS**

Lobomycosis\(^1\) (lacaziosis) is caused by the yeast-like organism *Lacazia loboi* (Taborda et al. 1999) (syn. *Loboa loboi*; Caldwell et al. 1975). It is an uncultivated pathogen that belongs with the other dimorphic fungi to the order Onygenales, family Ajellomycetaceae (Herr et al. 2001, Vilela et al. 2009).

*L. loboi* naturally affects *Tursiops truncatus* and *Sotalia guianensis* from the Americas as well as humans (Caldwell et al. 1975, Symmers 1983, Paniz-Mondolfi et al. 2007, Van Bressem et al. 2007b). *L. loboi* cells found in infected tissues from *T. truncatus* are significantly smaller than those found in humans, which, according to Haubold et al. (2000), suggests that the organism may not be identical in the 2 hosts. However, serological data indicate that dolphins and humans are infected with similar *L. loboi* strains (Mendoza et al. 2008). In humans, lobomycosis is a 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 that may access the skin by penetration or accidental trauma (Paniz-Mondolfi et al. 2007). Patients with lobomycosis may suffer immunoregulatory disturbances that may be responsible for the lack of pathogen containment (Vilani-Moreno et al. 2004).

Lobomycosis in Delphinidae is characterized by greyish, whitish to slightly pink, verrucous lesions, often in pronounced relief (Fig. 5), that may ulcerate.

\(^1\)The disease’s nomenclature of lobomycosis (or Lobo’s disease) is a recommended nomen conservandum by virtue of its common usage since at least 1973 and the fact that it honours its discoverer, Jorge Lobo, who in 1931 described the first case of a chronic, cutaneous mycosis in man, which he then called keloidal blastomycosis.
and form plaques that may exceed 30 cm in their broadest dimension (Reif et al. 2008). Initially only observed in inshore/estuarine *Tursiops truncatus* and *Sotalia guianensis*, lobomycosis was recently detected in offshore *T. truncatus* off northern California, suggesting that its range may be expanding (Rotstein et al. 2009) or that its offshore occurrence has eluded scientists to date. Dolphins may live with this progressive disease for several years (Murdoch et al. 2008). However, their general condition may deteriorate markedly as the disease advances, with death following. Whether this is due to *L. loboi* infection or to other causes is still uncertain (Symmers 1983). A review of the literature (Migaki et al. 1971, Caldwell et al. 1975, Bossart 1984, Simões-Lopes et al. 1993, Bossart et al. 2003, Moreno et al. 2008, Rotstein et al. 2009) revealed that at least 11 *T. truncatus* (9 inshore, 2 offshore) died with advanced lobomycosis in North and South America. Besides the presence of *L. loboi* in the axillary lymph nodes of one specimen, none of the 5 dolphins investigated in detail had involvement of internal organs or mucous membranes. The disease is endemic in *T. truncatus* from the Indian River Lagoon, Florida, with prevalence levels oscillating between 6% (*n* = 484) and 12.4% (*n* = 186) in the period 1996 to 2006 (Murdoch et al. 2008). Affected dolphins from this region have significant impairment in adaptive immunity, possibly related to chronic exposure to environmental stressors (Reif et al. 2008).

In South America and the SW Indian Ocean, several cases highly reminiscent of lobomycosis were observed in free-ranging inshore *Tursiops truncatus*, *T. aduncus* and *Sotalia guianensis*. In the absence of a histological diagnosis, the disease was called lobomycosis-like disease (LLD) (Van Bressem et al. 2007b, 2009b, Kiszka et al. 2009). As in the case of lobomycosis, LLD evolves over years and is associated with the death or disappearance of severely affected dolphins. Two *T. truncatus* stranded dead with generalized LLD in Brazil and Venezuela in 2003 and 2004, while 2 other well-known individuals with severe LLD disappeared in 2003 (Moreno et al. 2008). A severely affected calf from Mayotte Lagoon also disappeared in early 2006 (Kiszka et al. 2009). Prevalence of LLD in inshore South American *T. truncatus* varied from 1.6% (Gulf of Guayaquil, Ecuador) to 20% (Tramandaí estuary, Brazil) (Van Bressem et al. 2007b, Moreno et al. 2008). Along the Atlantic coast of South America, lobomycosis was first observed in an adult female *S. guianensis* caught in the estuary of the Surinam River in 1971 and subsequently in an adult female *T. truncatus* stranded in Santa Catarina State, southern Brazil, in 1990 (de Vries & Laarman 1973, Simões-Lopes et al. 1993). Though no other cases were reported until recently (Van Bressem et al. 2007b), a retrospective study in southern Brazil provided evidence that cutaneous mycosis, including lobomycosis and LLD, affected *T. truncatus* in Rio Grande do Sul State (south of Santa Catarina) from at least 1995 to 2008 (Moreno et al. 2008). LLD was also observed in 2 *T. truncatus* from Baía Norte and in 4 of 103 *S. guianensis* inhabiting the Paranaguá estuary in 2003 to 2007 (Van Bressem et al. 2007b, 2009b). Thus, lobomycosis and LLD in Brazilian Delphinidae seem to be concentrated in southern Brazil, from 25°22′S, 48°25′W to 31°17′S, 50°57′W (Moreno et al. 2008, Van Bressem et al. 2009b). A single case of LLD occurred in 2004 in a *T. truncatus* from Margarita Island, Venezuelan Caribbean, where lobomycosis or LLD had never before been encountered in humans nor dolphins (Bermudez et al. 2009). On the Pacific coast of South America, the first described cases of LLD date back to 1991, in an inshore *T. truncatus* community from the Estuary of Guayaquil, Ecuador (Van Bressem et al. 2007b). Further cases of LLD were reported in 2 *T. truncatus* from Bahía Malaga, Colombia, and in 1 *T. truncatus* from central Peru in 2005 and 2006 (Van Bressem et al. 2007b). The disease has also been present in *T. aduncus* in the Mozambique Channel at Mayotte since at least 1999, with a prevalence of 8.4% in 71 adults for the period 2004 to 2008 (Kiszka et al. 2009).

Though initially described only in large, presumably adult dolphins, LLD has recently also been seen in 2 calves, a *Sotalia guianensis* from the Paranaguá estuary and a *Tursiops aduncus* from Mayotte. This latter specimen was extensively affected and disappeared (Van Bressem et al. 2009b, Kiszka et al. 2009).
tions is unknown but the disease may have contributed to the death of several specimens.

A unique case of transmission of lobomycosis from *Tursiops truncatus* to a human was reported in Europe in the early 1970s (Symmers 1983). Norton (2006) believed that the transmission rate of lobomycosis from animals to humans is very low. Recently, a second case of zoonotic transmission was reported in a laboratory worker who most likely acquired lobomycosis after manipulating experimentally infected mice (Rosa et al. 2009). As for brucellosis, additional cases may remain undiagnosed in South and Central American countries. Close contact with infected dolphins may be risky and precautionary restrictions should apply with regard to programmes that allow thousands of tourists of unknown immunological status to swim with dolphins (Samuels & Spradling 1995). Till recently, the distribution and epidemiology of lobomycosis in humans and dottocetes seemed to be unconnected, affecting mostly humans inhabiting the Amazon basin and inshore and estuarine dolphins from the tropical and subtropical western Atlantic coast (Van Bressem et al. 2007b, Reif et al. 2008). However, recent findings suggest that humans may contract lobomycosis from the marine environment in South America (Bermudez et al. 2009).

The factors driving the apparent emergence of lobomycosis and LLD in South America and Mayotte are unknown. Several of the affected populations inhabit biologically and chemically polluted waters around major ports and cities and, in the case of Guayaquil, waters harbouring intense shrimp farming (Van Bressem et al. 2007b, Reif et al. 2008). The role of increased shipping in introducing *Lacazia loboii* to new ecological niches must be explored. The discharge of water, sediments and biofilms from ships’ ballast water tanks is a prominent known vector of aquatic invasive species (Ruiz et al. 2000, Drake et al. 2007). Global warming may also play a role as it expands the tropical belt (Seidel et al. 2008) and hence, possibly, the range of lobomycosis. Global warming-driven ecological modifications may affect local soil ecology and hydrology, resulting in the persistence of invasive fungal pathogens and release of infectious spores (Greer et al. 2008). Variation in salinity and water temperature are other parameters to consider in *L. loboii* infection (Reif et al. 2006).

**DISEASES OF MULTIFACTORIAL AETIOLOGY**

Recent studies have demonstrated a significant association between both chronic PCB and trace metal exposure and infectious diseases (including parasitic, bacterial and mycotic pneumonia and generalized bacterial infections) in *Phocoena phocoena* from the North and Baltic Seas (Siebert et al. 1999, Bennett et al. 2001, Jepson et al. 2005, Hall et al. 2006). *P. phocoena* from the British Isles that died in poor health had a significantly higher sum of the concentrations of 25 individual chlorobiphenyl congeners (Σ25CBs) than those that perished by traumatic death. Adult females in both groups had significantly lower Σ25CBs levels than adult males because of off-loading of organochlorines during gestation and lactation (Jepson et al. 2005). Pollutants also negatively affected the immune and endocrinical systems of *P. phocoena* in these waters (Beineke et al. 2005, Das et al. 2006), but much remains to be learned about the full impact on these populations.

**CONCLUSIONS**

Cetaceans are infected by a wide variety of pathogens that may be order-, family- or species-specific (e.g. morbilli-, pox- and papillomaviruses) or opportunistic (bacteria, fungi, protozoa). Infections 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). Environmental degradation, including biological and chemical pollution, climate change, fisheries, noise and heavy boat traffic, is thought to have disturbed this equilibrium by lowering the population immune response, depressing food supplies, increasing stress and facilitating the introduction of alien pathogens, among others (Fair & Becker 2000, Bossart 2007, Burek et al. 2008, Miller et al. 2008, Reif et al. 2008, Johnson et al. 2009). The reported number and severity of cetacean diseases have increased, in part because of more dedicated research in this field (Gulland & Hall 2007). However, taking this bias into account, some morbidity rates seem to have accrued, such as, for example, those resulting from exposure to harmful algal blooms (HABs), morbillivirus outbreaks in the North Atlantic and contiguous seas and LLD cases in South American cetaceans (Gulland & Hall 2007, Van Bressem et al. 2007b, Raga et al. 2008). Trend analysis for poorly studied or recently recognized diseases is often hampered by the lack of adequate quantitative data of long-term (background) prevalence levels, even though observed present-day prevalence may seem alarmingly high.

There is mounting circumstantial evidence that chemical pollution has increased the emergence and severity of several diseases in pinnipeds and cetaceans (Aguilar & Borrell 1994, Ross et al. 2000, Ross 2002, Jepson et al. 2005, Hall et al. 2006). Biological pollution is an emerging issue, with the findings of terrestrial pathogens in marine mammals, of a significant increased fecal coliform count in harbour seals living...
near urban developments and of cutaneous disorders of miscellaneous aetiology in coastal odontocetes (Mos et al. 2006, Van Bressem et al. 2007b, Miller et al. 2008). Climate changes can increase water temperatures, modify the distribution of vectors and reservoir species, change pathogen and host interaction dynamics and alter pathogen transmission cycles (Greer et al. 2008). Such changes have facilitated the establishment, emergence or possible resurgence of Cryptococcus gattii in Canada and promoted a multi-species outbreak (including the Dall’s porpoise Physeter macrocephalus and Phocoena phocoena) (Kidd et al. 2004). Climate changes have also affected the geographical distribution of arthropod-borne viruses (arboviruses) (Gould & Higgs 2008). Whether these viruses may emerge in cetaceans is currently unknown. However, St. Louis encephalitis virus (arboviruses Family Flaviviridae, genus Flavirus) has caused the death of a captive killer whale Orcinus orca (Buck et al. 1993). The louse Lepidophthirus macrorhiini is likely the reservoir and vector of the Southern elephant seal alphavirus (Family Togaviridae, genus Alphavirus; La Linn et al. 2001). Whether marine arthropods like Cyamidae (whale lice), parasitic on cetaceans, carry arboviruses remains to be investigated. Marine birds, especially gulls Larus spp. may serve as vector and reservoir of pathogens such as influenza viruses (Ohishi et al. 2006) and Escherichia coli (Nelson et al. 2008).

Brucella spp. and other bacteria (i.e. Mycobacterium spp. and Erysipelothrix rhusiopathiae), St. Louis encephalitis virus, Lactazia loboi and Toxoplasma gondii are zoonotic pathogens (Flowers 1970, Chastel et al. 1975, Kuno & Chang 2005). Standard sanitary precautions are recommended during physical contact with live cetaceans, and especially with stranded or other individuals exhibiting clinical signs of infectious pathologies. When handling fresh cetacean products such as blood, tissues, biopsy samples, muscle (meat) and skin (blubber), the systematic use of gloves and protective clothing would be optimal. However, it is clear that in field conditions and communal or aboriginal environments such preventative measures may be unrealistic, especially in developing countries. In humans, an estimated 75% of emerging diseases are zoonotic, with wildlife representing a large and mostly unknown reservoir (Cunningham 2005). Consumption of bushmeat (including cetacean meat), development of ecotourism and access to petting zoos favour the transmission of zoonotic diseases. Unidentified long-lasting or recurrent illnesses in humans working with dolphins or handling cetacean carcasses have recently been described (Hunt et al. 2008; this paper).

We conclude that anthropogenic environmental changes may increase the prevalence and severity of infectious illnesses in dolphins, porpoises and whales worldwide. Inshore, estuarine and riverine cetaceans seem particularly at risk because coastal and fluvial ecosystems are often dramatically degraded by human activities, are more heavily polluted both biologically and chemically, and are most prone to the effects of climate change. Neritic and fluvial species are also more likely killed, injured, disturbed or stressed by frequent, direct and indirect fisheries interactions, and by collisions with vessels (Van Waerebeek et al. 2007). A high prevalence of traumatic injuries, even minor skin lacerations, in concert with a compromised immune system create ideal targets for opportunistic pathogens.

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