REVIEW

Cardiovascular Disease in Cetaceans

Daniel BENAK[1], Frantisek KOLAR[1], Marketa HLAVACKOVA[1]

[1]Laboratory of Developmental Cardiology, Institute of Physiology of the Czech Academy of Sciences, Prague, Czech Republic

Received July 14, 2026
Accepted August 4, 2026

Summary

Cetaceans possess a highly specialized cardiovascular system that supports prolonged apnea, selective perfusion of vital organs, and tolerance to marked hemodynamic fluctuations during diving. Nevertheless, pathological and clinical observations show that they can develop a broad spectrum of cardiovascular disease (CVD). This review summarizes current knowledge on CVD in cetaceans across species and ecological contexts. We discuss the major disease phenotypes described to date, including stress cardiomyopathy, chronic myocardial fibrosis and cardiomyopathy-like remodeling, inflammatory cardiac disease, vascular and ischemic lesions, valvular and functional abnormalities, and congenital defects. Among these, acute stress-associated myocardial injury is one of the best-supported phenotypes in stranded cetaceans, whereas chronic cardiomyopathy-like remodeling is most clearly documented in pygmy and dwarf sperm whales. Inflammatory cardiac disease appears less frequent and is usually associated with systemic infection, while vascular disease includes not only atherosclerotic and thrombotic lesions but also embolic and decompression-related pathology. Current knowledge remains limited by strong species and sampling biases, with most evidence derived from stranded animals and a smaller body of work from managed dolphins. Overall, cetacean cardiovascular pathology reflects the interaction between extreme physiological specialization, environmental exposure, systemic disease, and acute stress.

Key words

Cetaceans • Cardiovascular disease • Stress cardiomyopathy • Myocardial fibrosis • Myocarditis • Valvular disease • Diving physiology • Cardiovascular adaptation

Corresponding author

Daniel Benak, Institute of Physiology of the Czech Academy of Sciences, Videnska 1083, 14200 Prague 4, Czech Republic. E-mail: daniel.benak@fgu.cas.cz

Introduction

Cardiovascular disease (CVD) is commonly framed as a hallmark of human aging and industrialized lifestyles, closely linked to hypertension, metabolic dysfunction, obesity, and other consequences of modern living. At first glance, cetaceans would appear far removed from this paradigm. Nevertheless, pathological and clinical observations indicate that they can develop a broad and heterogeneous spectrum of CVD, including acute stress cardiomyopathy [1], chronic myocardial fibrosis and cardiomyopathy-like remodeling [2], inflammatory cardiac disease [3], vascular and ischemic injury [4], valvular and functional abnormalities [5], and congenital defects [6]. At the same time, accumulating comparative evidence suggests that cetaceans may also possess traits that confer relative protection against some cardiovascular phenotypes, particularly vascular aging and atherosclerosis-like disease in at least some species [7,8]. Cetacean cardiovascular biology therefore cannot be understood simply in terms of either protection or pathology; rather, it reflects a dynamic balance between resilience and vulnerability shaped by species, diving ecology, longevity, environmental exposure, systemic disease, context-specific stressors such as stranding, bycatch, or human handling, and the highly specialized cardiovascular and cardiorespiratory adaptations that support diving. These include profound bradycardia,

 An open access article under the CC BY 4.0 license https://creativecommons.org/licenses/by/4.0/ © 2026 by the authors. Published by the Institute of Physiology, Czech Academy of Sciences, Prague, Czech Republic. E-mail: physres@fgu.cas.cz, www.biomed.cas.cz/physiolres

selective redistribution of blood flow, large oxygen stores, and structural adaptations of the great vessels that help stabilize central hemodynamics under extreme physiological conditions [9]. This balance is likely to differ substantially across the cetacean clade. Cetaceans comprise two major evolutionary lineages: Mysticeti, the baleen whales, and Odontoceti, the toothed whales. Mysticetes include rorquals, right whales, the gray whale (*Eschrichtius robustus*), and the pygmy right whale (*Caperea marginata*), whereas odontocetes encompass the sperm whale (*Physeter macrocephalus*) and kogiids, beaked whales, monodontids, porpoises, and dolphins [10]. These groups differ markedly in body size, diving behavior, ecological niche, and lifespan, and such differences are likely to influence both cardiovascular adaptation and susceptibility to disease.

Unfortunately, the evidence base remains limited and uneven. Most current knowledge derives from stranded animals, whereas live-animal cardiovascular assessment is largely restricted to managed dolphins and a small number of field studies [11]. As a result, the true prevalence, spectrum, and natural history of CVD in free-ranging cetacean populations remain incompletely defined.

In this review, we first summarize the cardiovascular adaptations that support diving and the putative determinants of cardiovascular resilience in cetaceans. We then synthesize the available evidence for the major cardiovascular disease phenotypes currently recognized in this group, with emphasis on acute stress-associated myocardial injury, chronic cardiomyopathy-like remodeling, inflammatory cardiac disease, vascular and ischemic lesions, valvular and functional abnormalities, and congenital defects. By integrating comparative physiology with pathology, we aim to highlight both the exceptional cardiovascular adaptations of cetaceans and the distinctive disease processes to which they remain susceptible.

Cardiovascular adaptations to diving

The cetacean cardiovascular system is adapted to sustain prolonged apnea while preserving oxygen delivery to the most vulnerable organs, but also to tolerate the profound hydrostatic pressure changes associated with depth. These adaptations are not unique to cetaceans and, at least in part, are shared by other air-breathing diving vertebrates, including pinnipeds and marine birds [9]. At the same time, diving ecology is highly heterogeneous even within cetaceans: many dolphins and porpoises typically perform relatively short but more frequent dives, whereas large toothed whales such as Cuvier's beaked whales (*Ziphius cavirostris*) are among the most extreme mammalian divers, with recorded dive durations extending to approximately 222 min [12].

The core of the adaptation is the classic diving reflex, characterized by bradycardia, peripheral vasoconstriction, and redistribution of blood flow toward the brain and heart. In free-diving marine mammals, however, this response is graded rather than stereotypically fixed: the magnitude of bradycardia varies with species and dive type, and generally becomes more pronounced as dive duration increases [9].

Overall heart size does not appear to represent a uniform adaptation to diving in cetaceans. Despite the exceptional variation in cetacean body size – from approximately 50 kg in the vaquita (*Phocoena sinus*) to reported maximum values approaching 190,000 kg in the blue whale (*Balaenoptera musculus*) – relative heart mass generally falls within the range reported for terrestrial mammals [13]. Nevertheless, substantial interspecific variation exists: relatively large hearts have been reported in some dolphin species, including the white-beaked dolphin (*Lagenorhynchus albirostris*), whereas sperm whales (*Physeter macrocephalus*) have comparatively small hearts relative to their body size, suggesting that ecology, phylogeny, and other species-specific factors may influence cardiac size [13-15]. The principal cardiac adaptations to diving therefore appear to involve morphology rather than overall heart mass. Cetacean hearts are often dorsoventrally flattened, the right ventricular chambers are enlarged, and, depending on the species, the relative thickness of the right ventricular wall may also be increased. Dorsoventral flattening is thought to reposition the right ventricle so that its filling is not compromised by chest compression during dives, while changes in right ventricular chamber size and wall thickness are considered to improve its function during chest compression, lung collapse, and probable increases in pulmonary vascular resistance [9]. Recent anatomical analysis in bottlenose dolphins is consistent with this general pattern, demonstrating marked right ventricular cavity enlargement relative to terrestrial comparators [16]. Notably, fetal right-to-left shunts such as the *foramen ovale* and *ductus arteriosus* close shortly after birth in cetaceans, as in terrestrial mammals, and therefore do not appear to serve as a mechanism for bypassing potential increases in pulmonary vascular

resistance during diving [9].

A further important adaptation is the high compliance of the ascending aorta, aortic arch, and proximal carotid arteries in whales. These structures have been proposed to function as a windkessel, helping maintain diastolic blood flow and pressure during the long cardiac pauses associated with slow heart rates [17]. This compliance is particularly relevant for coronary perfusion, which occurs mainly during diastole. It may also reduce left ventricular afterload during peripheral vasoconstriction, thereby limiting myocardial oxygen demand. In the largest species, this bradycardic response can be especially pronounced; in blue whales, dive-associated heart rates are typically 4-8 beats/min (bpm), can fall as low as 2 bpm, and rise to approximately 25-37 bpm during surface intervals [18].

These hemodynamic responses operate in parallel with unusually large oxygen stores, particularly in blood and skeletal muscle. Cetacean hemoglobin retains the basic tetrameric structure characteristic of mammalian hemoglobins, and its oxygen affinity is generally within the mammalian range. The size of the blood oxygen store is therefore determined largely by hemoglobin concentration and mass-specific blood volume, both of which vary markedly among species and tend to be greater in cetaceans with higher diving capacities. In the sperm whale, reported values include a hemoglobin concentration of approximately 22 g/dl and a mass-specific blood volume of approximately 200 ml/kg. By contrast, reported values for the common bottlenose dolphin (*Tursiops truncatus*) are approximately 14 g/dl and 71 ml/kg, respectively, overlapping the human range [9]. A particularly important diving adaptation is the high myoglobin content of locomotor muscle, which provides a local oxygen reserve, delays tissue oxygen depletion, and helps prolong aerobic metabolism during dives [19]. Myoglobin concentrations vary much more widely among cetacean species than hemoglobin concentrations and are influenced by species, age, muscle type, and diving ecology. Reported values range from approximately 0.9 g per 100 g wet muscle in blue whales, about twice the typical concentration in human skeletal muscle (approximately 0.47 g/100 g [20]), to approximately 7.9 g/100 g in narwhals (*Monodon monoceros*). Thus, in some deep-diving cetaceans, muscle myoglobin concentrations may exceed human values by more than an order of magnitude [9]. Blood-flow restriction to peripheral tissues conserves oxygen for organs with limited ischemic tolerance [21]. Cetaceans also appear to begin dives at high lung volume, unlike many phocid seals, which typically dive on expiration; this likely increases the respiratory oxygen store but also makes the regulation of gas exchange at depth especially important. With increasing pressure, progressive alveolar compression and pulmonary shunting reduce gas exchange, thereby limiting nitrogen uptake and helping preserve respiratory oxygen for ascent [9].

Overall, cetacean diving physiology reflects a coordinated cardiovascular strategy in which oxygen conservation, selective perfusion, hemodynamic stabilization, and tolerance of thoracic compression allow repeated exposure to apnea, fluctuating oxygen availability, and increased pressure at depth. This background is essential for interpreting cardiovascular disease in cetaceans, because findings that appear pathological in terrestrial mammals may in some cases overlap with highly specialized physiological adaptation.

Potential determinants of cardiovascular resilience and selective vulnerability in cetaceans

In contrast to the dominant human paradigm, in which CVD is strongly shaped by aging, sedentary behavior, and adverse dietary exposures, cetaceans combine habitual locomotor activity with a marine diet often rich in long-chain n-3 fatty acids, features that would generally be expected to favor cardiovascular health [22,23]. For example, analysis of the cardiac phospholipidome in small cetaceans revealed substantially higher n-3 fatty acid content than that observed in terrestrial mammals, with a predominance of phospholipid species containing esterified n-3 fatty acids, particularly EPA and DHA [24]. Moreover, whole-genome analyses have suggested enhanced antioxidant capacity in cetaceans, including expansion of the peroxiredoxin (PRDX) family and cetacean-specific amino acid changes in several genes related to glutathione metabolism [25]. Together, these molecular adaptations may contribute to relative protection against some vascular phenotypes, including atherosclerosis-like disease in whales [8].

In addition, the recurrent peripheral hypoperfusion and reoxygenation associated with diving may act as a natural ischemic-conditioning-like stimulus, potentially contributing to tolerance to hypoxemia and ischemia/reperfusion stress. Experimental studies in

laboratory animals indicate that both adaptation to chronic hypoxia and remote ischemic preconditioning induced by brief ischemic episodes in distant tissues can protect the myocardium against subsequent ischemia–reperfusion injury [26-28], although direct evidence for analogous cardioprotection in cetaceans remains lacking. Emerging evidence, however, suggests that cetaceans may possess a protective circulating milieu [7]. Serum from common bottlenose dolphins preserved endothelial function in *ex vivo* mouse carotid arteries, whereas age-equivalent human serum impaired endothelium-dependent dilation, supporting the idea that vascular protection may be an evolved component of diving adaptation [7]. Beyond its relevance to marine mammal biology, this raises the possibility that cetaceans may serve as a comparative model of endogenous vascular resilience with translational relevance to human CVD. From a comparative perspective, species such as the bowhead whale (*Balaena mysticetus*) are especially intriguing in the context of vascular aging and long-term cardiovascular resilience, as their exceptional longevity exceeds 200 years [29], making them the longest-lived mammals known.

At the same time, these potentially protective traits do not eliminate cardiovascular risk. Cetaceans are exposed to profound autonomic fluctuations, repeated changes in oxygen availability and shear stress, and, depending on species and environment, additional pressures such as aging, systemic disease, infection, toxic exposure, and stranding-associated stress [11]. Cardiac disease is common in aged cetaceans in both managed and free-ranging populations and includes multifocal myocardial degeneration and fibrosis, endocardiosis, valvular fibrosis, papillary fibro-elastomas, mitral leaflet thickening, or left ventricular hypertrophy [11]. Negative effects of toxins are particularly relevant because recent studies continue to document increasing exposure of cetaceans to persistent organic pollutants, including PFAS and legacy organochlorines, with evidence linking contaminant burden to oxidative stress biomarkers in marine mammals [30-32]. Acute cardiovascular injury may also accompany extreme stress states in stranded cetaceans, resulting in stress cardiomyopathy [33].

Cardiovascular pathology in cetaceans should therefore be understood not as a contradiction of their specialized biology, but as the product of competing resilience and injury mechanisms operating within a distinctive physiological and ecological context.

Cardiovascular disease in cetaceans

The study of CVD in cetaceans (Fig. 1) remains limited, and current knowledge is derived from a relatively small and heterogeneous body of evidence biased toward odontocetes [11]. Most available information comes from necropsy findings in stranded individuals, including animals involved in mass stranding events, whereas a smaller but important body of data has been generated in managed cetaceans, particularly dolphins, in which clinical cardiovascular assessment is feasible. Both sources are inherently biased: stranded animals disproportionately represent individuals affected by severe acute stress, systemic illness, trauma, or decomposition-related artifact. This limitation is particularly pronounced for elusive deep-diving species, for which live observations are scarce and biological knowledge often relies heavily on stranding records [34]. By contrast, managed populations reflect a restricted subset of species living under highly specific environmental conditions. As a result, the true prevalence, spectrum, and natural history of CVD in free-ranging cetacean populations remain poorly defined.

Stress cardiomyopathy

Stress cardiomyopathy is a common cardiovascular phenotype in stranded or bycaught cetaceans and in animals handled during rescue. It is thought to result from stress-associated catecholamine release, with subsequent ischemia/reperfusion injury linked to spasm of small intramural coronary arteries, potentially leading to arrhythmias and death. Histologically, it is characterized by a recurring pattern of acute myocardial injury that includes contraction band necrosis, wavy myofibers, interstitial edema, and myocardial degeneration or necrosis (Fig. 2) [1,33, 35-39]. Interestingly, this stress-associated, catecholamine-mediated pattern parallels human Takotsubo syndrome, in which acute emotional or physical stress and catecholamine excess are associated with transient ventricular dysfunction and may occasionally produce contraction band necrosis [40].

An early indication of stress cardiomyopathy was provided by Turnbull and Cowan [1], who identified myocardial contraction band necrosis in all 52 stranded cetaceans examined along the western Gulf of Mexico coast. Cowan and Curry [35] subsequently expanded the histopathological framework in 407 small odontocetes

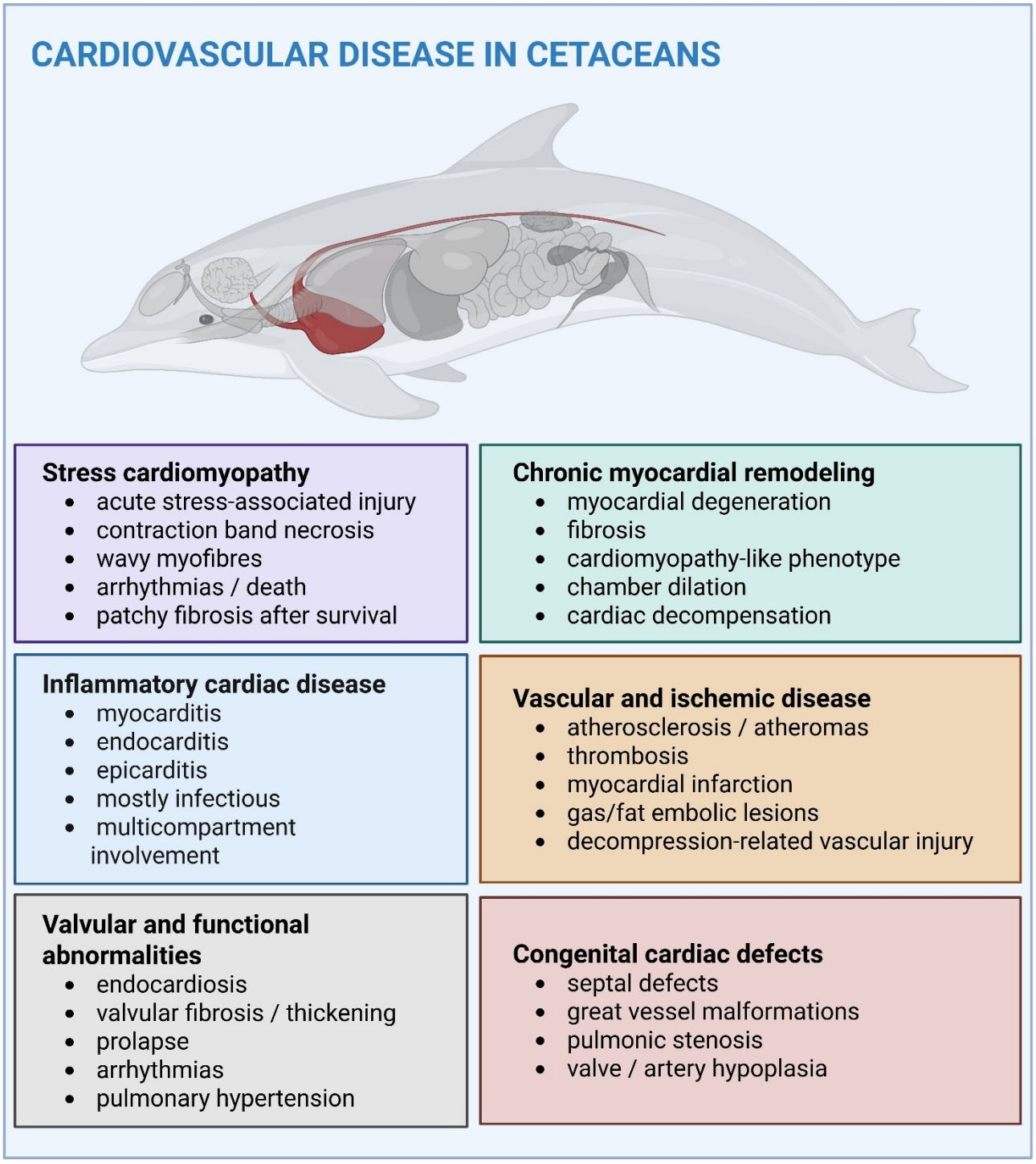


Fig. 1. Schematic overview of the major cardiovascular disease phenotypes currently described in cetaceans. The spectrum includes acute stress cardiomyopathy, chronic myocardial degeneration and cardiomyopathy-like remodeling, inflammatory cardiac disease, vascular and ischemic lesions, valvular and functional abnormalities, and congenital cardiac anomalies. The occurrence and interpretation of these phenotypes are influenced by species, diving ecology, age, infectious disease, environmental exposure, and stress-related contexts such as stranding, bycatch, and handling.

from 20 species, demonstrating a characteristic myocardial injury pattern with additional involvement of the atria and conduction system, together with lesions of small intramural coronary arteries and patchy myocardial scarring supporting a vasospastic and ischemic component of injury. Acute myocardial injury was most pronounced in *Kogia*, whereas scarring was most prominent in *Delphinus*. Herraez *et al.* [36] further supported the view that acute cardiac injury in stranded cetaceans may occur within a wider stress-associated

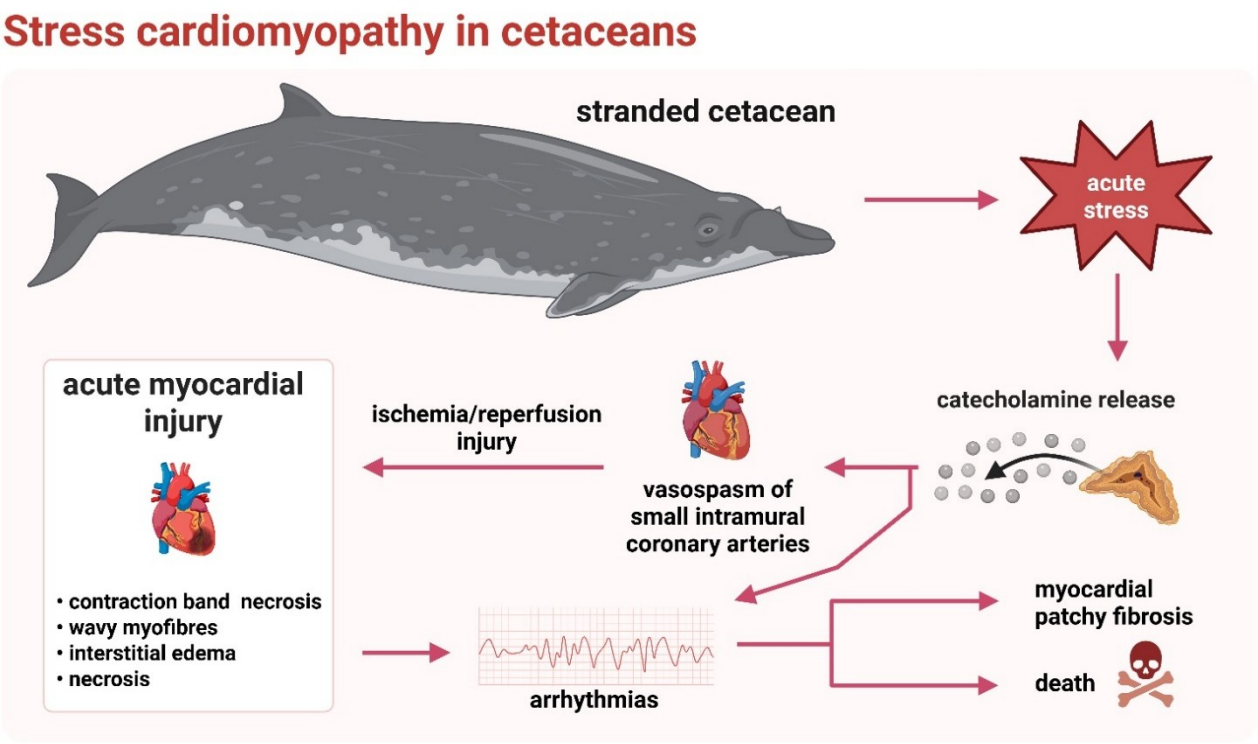


Fig. 2. Proposed pathophysiological framework of stress cardiomyopathy in cetaceans. Stranding-associated acute stress induces catecholamine release and vasospasm of small intramural coronary arteries, leading to ischemia/reperfusion injury and acute myocardial lesions characterized by contraction band necrosis, wavy myofibers, interstitial edema, and necrosis. These changes may result in arrhythmias, death, or subsequent patchy myocardial fibrosis.

multisystem syndrome. In a 12-year necropsy series of 51 live-stranded cetaceans from 13 species that underwent rescue-related capture, handling, transport, or short-term captivity, 25 animals showed a clinicopathological picture characterized by hemodynamic shock, degenerative lesions affecting both skeletal and cardiac muscle, and myoglobinuric tubular nephrosis. Cardiac lesions were detected in all 25 affected animals, including contraction band necrosis in Purkinje fibers. Camara *et al.* [33] strengthened the diagnosis of stress cardiomyopathy using histological, histochemical, and immunohistochemical analyses in 67 cetaceans from 19 species, demonstrating not only the characteristic lesion pattern but also depletion of cardiac troponins and myoglobin together with fibrinogen accumulation in damaged cardiomyocytes. In a subsequent study of nine live-stranded cetaceans, the same group showed that plasma cardiac troponin I was frequently elevated above the proposed cetacean reference range and correlated with histopathological evidence of acute cardiac injury [37]. A complementary case report in a rehabilitated live-stranded Risso's dolphin (*Grampus griseus*) further demonstrated rising cardiac troponin I and creatine kinase together with histological and immunohistochemical lesions consistent with capture myopathy and stress cardiomyopathy [38]. A larger retrospective stranding series from Taiwan reinforced the importance of this phenotype by identifying acute stress-induced cardiomyopathy in 41 of 73 stranded cetaceans, while also highlighting overlap with chronic and mixed cardiomyopathic phenotypes [39]. A further histopathological study in 20 stranded Cuvier's beaked whales (*Ziphius cavirostris*) showed a similar spectrum of stress-associated cardiac lesions, including wavy fibers in 75 %, vacuolar degeneration in 85 %, contraction band necrosis in 40 %, and vascular congestion and edema in 65 % and 55 % of cases, respectively, extending the stress cardiomyopathy phenotype to extreme deep-diving beaked whales [41].

In contrast to previous studies, which were dominated by stranded odontocetes and reported a high frequency of stress-associated myocardial lesions, contraction band necrosis was found in only 8 of 200 (4.0 %) hunted bowhead whales [42]. This suggests that the occurrence of acute stress-associated myocardial injury may be strongly species- and context-dependent in cetaceans. Additional evidence from a baleen whale comes from a live-stranded neonatal Bryde's whale

(*Balaenoptera brydei*), which showed elevated creatine kinase and cardiac troponin I together with histological and immunohistochemical evidence of acute skeletal and myocardial injury, including contraction band necrosis, wavy fibers, edema, and troponin/myoglobin depletion, consistent with stress cardiomyopathy and cardiac rhabdomyolysis in the setting of fetal distress and active stranding pathology [43].

Collectively, these studies identify stress cardiomyopathy as one of the best-supported forms of acute cardiac injury in stranded cetaceans. Importantly, survival of the acute insult does not necessarily imply full myocardial recovery, as ischemic and necrotic injury may progress to chronic patchy myocardial fibrosis [35,39].

Chronic myocardial remodeling

Chronic myocardial degeneration and cardiomyopathy-like remodeling have also been described in cetaceans as phenotypes distinct from acute stress cardiomyopathy, although they may in some cases develop after survival of acute stress-induced myocardial injury. These lesions are best documented in pygmy (*Kogia breviceps*) and dwarf sperm whales (*Kogia sima*), in which pathology ranges from mild myocardial degeneration to advanced cardiomyopathy with fibrosis and signs of cardiac decompensation.

Bossart *et al.* [2] first described a chronic cardiomyopathy-like syndrome in stranded kogiids, characterized grossly by pale, flabby, and sometimes dilated right ventricles and microscopically by moderate-to-extensive myocellular degeneration, atrophy, and fibrosis, predominantly affecting the right ventricle and ventricular septum. Associated centrilobular hepatic congestion, hepatocellular atrophy, and hemorrhagic necrosis were interpreted as consistent with primarily right-sided congestive heart failure, leading the authors to propose that myocardial decompensation likely preceded and contributed to stranding rather than arising secondarily as an acute consequence of the stranding event. A later study by Bossart *et al.* [44], which used a standardized examination protocol in 29 stranded *Kogia* hearts, further supported a chronic progressive continuum, with 14 animals classified as cardiomyopathy and 12 as mild myocardial degeneration. The predominant histological features included anisokaryosis with karyomegaly and nuclear rowing, interstitial edema, myofiber disarray, loss of cross-striations, eosinophilic sarcoplasmic homogenization, wavy-attenuated myofibers, and fibrosis. Because lesion types and anatomical distribution overlapped substantially between mild myocardial degeneration and cardiomyopathy, the authors interpreted these findings as different stages of the same chronic process rather than as an acute terminal lesion.

A recent retrospective study of 45 stranded kogiids from the Canary Islands further strengthened the impression that chronic cardiomyopathy-like lesions are highly prevalent in this family. Gross and/or histological findings consistent with cardiomyopathy were identified in 31/45 animals (68.9 %), although cardiomyopathy was considered the primary cause of death in only 3 cases. In those fatal cases, right ventricular dilation, pulmonary edema and congestion, chronic passive hepatic congestion, cardiomyocyte degeneration, contraction band necrosis, myofiber disarray, and myocardial fibrosis supported terminal congestive cardiac failure rather than isolated acute myocardial injury [45]. Viricel and Rosel [46] explored the genetic basis of the apparent susceptibility of kogiids to cardiomyopathy and identified six variants in the sarcomeric genes *MYH7* and *MYBPC3*, encoding β-myosin heavy chain and cardiac myosin-binding protein C, respectively, that were predicted to affect protein function. The involvement of these genes is noteworthy from a comparative perspective, because pathogenic variants in *MYH7* and *MYBPC3* are among the principal genetic causes of inherited sarcomeric cardiomyopathy in humans, particularly hypertrophic cardiomyopathy [47]. However, because three of these variants were also detected in kogiid individuals without cardiomyopathy [46], the etiology is likely multifactorial, with possible contributions from age and environmental factors.

Similar pathologies have also been reported in cetaceans other than kogiids. In the Taiwanese stranding series, 38 of 73 cetaceans showed chronic or mixed cardiomyopathic phenotypes, including dilated cardiomyopathy and patchy myocardial fibrosis, while 25 animals were classified as having mixed-type cardiomyopathy combining acute and chronic features [39]. Additional support for chronic myocardial remodeling outside kogiids comes from bottlenose dolphins affected by the Deepwater Horizon oil spill, in which myocardial fibrosis was significantly more prevalent, suggesting that anthropogenic exposure may contribute to chronic myocardial injury in cetaceans [48].

These findings indicate that chronic myocardial remodeling in cetaceans is not restricted to kogiids, although the clearest and most repeatedly described

chronic cardiomyopathy-like syndrome remains in *Kogia* spp.

Inflammatory cardiac disease: myocarditis, endocarditis, and epicarditis

Compared with stress cardiomyopathy and chronic cardiomyopathy-like remodeling, inflammatory cardiac disease appears to be less frequently documented in cetaceans and is most often reported in association with infectious processes, sometimes involving the myocardium, endocardium, and epicardium simultaneously. The relative rarity of such lesions is illustrated by the study of Cowan and Curry [35], in which inflammation consistent with infectious myocarditis was observed in only one of 407 small odontocetes collected over 40 years.

In odontocetes, inflammatory cardiac lesions have been reported mainly in the context of septicemia or other systemic infectious disease and often involve multiple cardiac compartments simultaneously. Early reports noted incidental myocarditis in long-finned pilot whales (*Globicephala melas*) [49]. Subsequent case reports and stranding surveys showed that bacterial infections may be associated with myocarditis, endocarditis, and epicarditis, including lymphoplasmacytic myocarditis and vegetative mitral valve endocarditis in a short-beaked common dolphin (*Delphinus delphis*) with *Streptococcus phocae* septicemia and cetacean morbillivirus (CeMV) coinfection [3], necrotizing multivalvular endocarditis in another common dolphin with fatal *Wohlfahrtiimonas chitiniclastica* septicemia [50], and pyogranulomatous myocarditis with suppurative epicarditis in harbor porpoises (*Phocoena phocoena*) with *Staphylococcus aureus* septicemia [51]. Brucella-associated cardiac inflammation has also been documented in striped dolphins (*Stenella coeruleoalba*), ranging from severe vegetative mitral valve endocarditis with focal myocardial and pericardial involvement to myocarditis and myocardial necrosis in infected animals [52,53]. Viral and protozoal contributions have also been reported: CeMV-associated epicarditis and myocarditis were identified in striped dolphins [54], while *Toxoplasma gondii* was detected in the hearts of individual striped and bottlenose dolphins with lymphocytic epicarditis or mild lymphoplasmacytic myocarditis [55,56]. In addition, the pseudaliid nematode *Pseudalius inflexus* has been reported in the heart and pulmonary vasculature of cetaceans and has been associated with endocarditis, indicating that helminth infections may also contribute to inflammatory cardiovascular lesions [11]. Together, these observations indicate that inflammatory cardiac disease in odontocetes is uncommon but etiologically diverse and most often reflects systemic infectious processes rather than primary isolated cardiac pathology.

Among mysticetes, myocarditis has been identified as a probable cause of death in three southern right whale (*Eubalaena australis*) calves from Península Valdés, including one case with concurrent meningitis. Histologically, the myocardial lesions were multifocal to regionally extensive and consisted predominantly of degenerate neutrophils associated with cardiomyocyte necrosis and clusters of intralesional coccobacilli or short bacilli, supporting infectious suppurative myocarditis rather than stress-associated myocardial injury [57]. Another southern right whale calf off Brazil with septicemia caused by *Streptococcus dysgalactiae* also had myocarditis together with valvular endocarditis [58]. In bowhead whales, myocardial inflammation appears to be less prominent, with interstitial myocarditis reported in three and granulomatous myocarditis in one of 200 hunted animals [42].

Vascular and ischemic disease: atherosclerosis, thrombosis, and myocardial infarction

Although cetaceans, particularly large whales, may be relatively protected against atherosclerosis [8], vascular and ischemic lesions have nevertheless been described in several species. In addition, serum from common bottlenose dolphins has been reported to exert vasculoprotective effects in *ex vivo* assays, suggesting the presence of a circulating milieu that may help preserve vascular function and limit adverse vascular aging [7]. Despite these potentially protective traits, atherosclerosis of the coronary arteries and aorta has been described in long-finned pilot whales hunted off Newfoundland [4,49]. Atheromas have also been reported in short-finned pilot whales (*Globicephala macrorhynchus*), pygmy sperm whales, and killer whales (*Orcinus orca*), with the latter two species also showing severe vascular lesions, including thrombus formation [4]. Small myocardial infarctions not associated with overt functional disturbance were observed in several common bottlenose dolphins [4]. Thrombosis of peripheral arteries has also been reported, particularly in association with *Erysipelothrix rhusiopathiae* infection [4]. Also, the

pseudaliid nematode *Pseudalius inflexus* has been associated with vasculitis and thrombosis in cetaceans, indicating that parasitic infection may also contribute to vascular pathology [11].

In addition to atherosclerotic and thrombotic lesions, cetaceans may also develop acute embolic vascular disease as their vascular modifications may predispose them to gas bubble formation and embolization [11]. Pulmonary and systemic gas and fat embolic syndrome has been described in mass-stranded beaked whales exposed to anthropogenic sonar [59], while acute and chronic gas bubble lesions have also been reported in other stranded cetaceans including Risso's dolphins (*Grampus griseus*), common dolphins, Blainville's beaked whales (*Mesoplodon densirostris*), and harbor porpoises [60]. These findings indicate that vascular injury in cetaceans may also arise through decompression sickness-like mechanisms analogous to those affecting human divers, although the circumstances leading to pathological gas-bubble formation in cetaceans remain incompletely understood [61].

Valvular and functional cardiovascular abnormalities

Cetaceans may also exhibit valvular, structural, electrical, and hemodynamic cardiovascular abnormalities. Mitral valve endocardiosis was observed in two aged common bottlenose dolphins, and a holosystolic murmur of mitral insufficiency was also detected in another individual [4]. A stranded-dolphin series including striped dolphins and one common bottlenose dolphin from the Ligurian coast further documented a broader spectrum of structural and valvular lesions, including tricuspid and mitral endocardiosis, valvular fibrosis, mitral leaflet thickening, Lambl's excrescences, right ventricular dilation associated with hypoplasia of the tricuspid chordae, left ventricular hypertrophy, aneurysms of the pulmonary trunk/right sinus of Valsalva, and a cirsoid aneurysm of the coronary artery [5].

In bottlenose dolphins affected by oil spill, echocardiography and electrocardiography have identified thinner left ventricular walls, smaller left atrial size, increased prevalence of tricuspid valve prolapse and valvular thickening [48].

Congenital cardiac defects

Congenital cardiac anomalies appear to be rare in cetaceans and are documented almost exclusively in isolated case reports, limiting any conclusions about their true prevalence or species distribution. Nevertheless, the available reports indicate that structural malformations of the heart and great vessels do occur and, in some cases, may be severe enough to contribute to stranding or early death. An early report in bottlenose dolphins described a female fetus with transposition of the pulmonary artery and aorta and an interventricular septal defect [6]. Congenital ventricular septal defect with left-to-right shunting has also been described in a live-stranded harbor porpoise from the North Sea [62]. In addition, necropsy of a neonatal bottlenose dolphin stranded on the coast of South Carolina revealed multiple congenital cardiac malformations, including right ventricular hypertrophy, ventricular septal defect, aortic dilation, an atrial septal defect associated with a functionally common atrium and left atrial remnant, subvalvular pulmonic stenosis, and hypoplasia of the pulmonary artery and mitral valve [63]. Together, these reports indicate that although congenital heart disease is rarely detected and poorly characterized in cetaceans, it should be considered among the differential diagnoses of cardiovascular compromise, particularly in neonatal or juvenile animals.

Conclusions

Cetaceans combine exceptional cardiovascular specialization with a surprisingly broad, although unevenly documented, spectrum of cardiovascular disease. The literature reviewed here indicates that cetacean CVD does not conform to a single pattern. Instead, it comprises several partially overlapping phenotypes, including acute stress cardiomyopathy, chronic myocardial degeneration and fibrosis, infectious inflammatory carditis, vascular and ischemic disease, valvular abnormalities, and rare congenital defects. Among these, acute stress-associated myocardial injury is the most consistently documented lesion complex, whereas chronic cardiomyopathy-like remodeling is most clearly established in kogiids. At the same time, comparative and mechanistic evidence suggests that cetaceans may be relatively protected from some vascular phenotypes, particularly adverse vascular aging and atherosclerosis-like disease in at least some taxa. Overall, cetacean cardiovascular biology is best understood as a balance between resilience and vulnerability, shaped by species, diving ecology, lifespan, environmental exposure, infection, and acute stress. A better understanding of cetacean cardiac physiology and

pathology is therefore important for interpreting disease, improving health assessment, and evaluating the effects of stranding, bycatch, pollution, and other anthropogenic stressors on cetacean populations. At the same time, the study of cetaceans offers a valuable comparative perspective on cardiovascular responses to hypoxia, ischemia-reperfusion, extreme autonomic and hemodynamic regulation, vascular aging, and stress-associated myocardial injury across mammals, including humans.

Acknowledgements
Figures were created with BioRender.com.

Declaration of the use of AI
During the preparation of this work, the authors used ChatGPT (OpenAI) to assist with language editing and readability. After using this tool, the authors reviewed and edited the content as needed and take full responsibility for the content of the published article.

Conflict of Interest
There is no conflict of interest.

References

1. Turnbull BS, Cowan DF. Myocardial contraction band necrosis in stranded cetaceans. J Comp Pathol 1998;118:317-327. https://doi.org/10.1016/S0021-9975(07)80007-7
2. Bossart GD, Odell DK, Altman NH. Cardiomyopathy in stranded pygmy and dwarf sperm whales. J Am Vet Med Assoc 1985;187:1137-1140. https://doi.org/10.2460/javma.1985.187.11.1137
3. Díaz-Delgado J, Sierra E, Vela AI, Arbelo M, Zucca D, Groch KR, Fernández A. Coinfection by Streptococcus phocae and cetacean morbillivirus in a short-beaked common dolphin Delphinus delphis. Dis Aquat Organ 2017;124:247-252. https://doi.org/10.3354/dao03124
4. Sweeney JC, Ridgway SH. Common diseases of small cetaceans. J Am Vet Med Assoc 1975;167:533-540. https://doi.org/10.2460/javma.1975.167.07.533
5. Scaglione FE, Bollo E, Pregel P, Chiappino L, Sereno A, Mignone W, et al. Heart pathologies in dolphins stranded along the northwestern Italian coast. Dis Aquat Organ 2013;107:31-36. https://doi.org/10.3354/dao02672
6. Gray KN, Conklin RH. Multiple births and cardiac anomalies in the bottle-nosed dolphin. J Wildl Dis 1974;10:155-157. https://doi.org/10.7589/0090-3558-10.2.155
7. Bernaldo de Quirós Y, Mahoney SA, VanDongen NS, Greenberg NT, Venkatasubramanian R, Saavedra P, et al. The bottlenose dolphin (Tursiops truncatus): a novel model for studying healthy arterial aging. Am J Physiol Heart Circ Physiol 2024;327:H660-H665. https://doi.org/10.1152/ajpheart.00464.2024
8. Tjandrawinata RR, Vencio ICC, Nugroho ABM, Hartanto J, Widjanarko ND, Nurkolis F. Guardians of the sea: molecular and biochemical adaptations in sharks and whales for atherosclerosis resistance and their potential in human cardioprotection. Diabetol Metab Syndr 2025;17:293. https://doi.org/10.1186/s13098-025-01868-5
9. Ponganis P. *Diving Physiology of Marine Mammals and Seabirds*. Cambridge University Press, 2015, 334 p. https://doi.org/10.1017/CBO9781139045490
10. McGowen MR, Tsagkogeorga G, Álvarez-Carretero S, dos Reis M, Struebig M, Deaville R, et al. Phylogenomic Resolution of the Cetacean Tree of Life Using Target Sequence Capture. Systematic Biology 2020;69:479-501. https://doi.org/10.1093/sysbio/syz068
11. St. Leger J, Raverty S, Mena A. Cetacea. In: *Pathology of Wildlife and Zoo Animals*. TERIO KA, MCALOOSE D, ST LEGER J (eds), Academic Press, 2018, pp 533-568. https://doi.org/10.1016/B978-0-12-805306-5.00022-5
12. Quick NJ, Cioffi WR, Shearer JM, Fahlman A, Read AJ. Extreme diving in mammals: first estimates of behavioural aerobic dive limits in Cuvier's beaked whales. J Exp Biol 2020;223:jeb222109. https://doi.org/10.1242/jeb.222109
13. McAlpine D. Size and growth of heart, liver, and kidneys in North Atlantic fin (Balaenoptera physalus), sei (B. borealis), and sperm (Physeter macrocephalus) whales. Can J Zool 1985;63:1402-1409. https://doi.org/10.1139/z85-210
14. Galatius A, Ijsseldijk L, Siebert U, Kinze C. External proportions, body and organ masses, and foetal growth of North Sea white-beaked dolphins. Mammal Biol 2025;106:441-453. https://doi.org/10.1007/s42991-025-00545-4

15. Plön S, Albrecht K, Cliff G, Froneman PW. Organ weights of three dolphin species from South Africa - implications for ecological adaptation? J Cetac Res Manag 2012;12:265-276. https://doi.org/10.47536/jcrm.v12i2.584
16. Kinoshita R, Ebisawa K, Okabayashi K, Narita T, Nakayama S, Koie H. Aquatic environments change the cardiac morphology of dolphins. J Vet Med Sci 2023;85:334-339. https://doi.org/10.1292/jvms.22-0367
17. Shadwick RE, Gosline JM. Arterial mechanics in the fin whale suggest a unique hemodynamic design. Am J Physiol 1994;267:R805-R818. https://doi.org/10.1152/ajpregu.1994.267.3.R805
18. Goldbogen JA, Cade DE, Calambokidis J, Czapanskiy MF, Fahlbusch J, Friedlaender AS, et al. Extreme bradycardia and tachycardia in the world's largest animal. Proc Natl Acad Sci U S A 2019;116:25329-25332. https://doi.org/10.1073/pnas.1914273116
19. Wright TJ, Davis RW. Myoglobin oxygen affinity in aquatic and terrestrial birds and mammals. J Exp Biol 2015;218:2180-2189. https://doi.org/10.1242/jeb.119321
20. Möller P, Sylvén C. Myoglobin in human skeletal muscle. Scand J Clin Lab Invest 1981;41:479-482. https://doi.org/10.3109/00365518109090486
21. Allen KN, Vázquez-Medina JP. Natural Tolerance to Ischemia and Hypoxemia in Diving Mammals: A Review. Front Physiol 2019;10:1199. https://doi.org/10.3389/fphys.2019.01199
22. Štursová P, Budinská X, Nováková Z, Dobšák P, Babula P. Sports activities and cardiovascular system change. Physiol Res 2023;72(Suppl. 5):S429-S444. https://doi.org/10.33549/physiolres.935238
23. Jiang L, Wang J, Xiong K, Xu L, Zhang B, Ma A. Intake of Fish and Marine n-3 Polyunsaturated Fatty Acids and Risk of Cardiovascular Disease Mortality: A Meta-Analysis of Prospective Cohort Studies. Nutrients 2021;13:2342. https://doi.org/10.3390/nu13072342
24. Monteiro J, Maciel E, Maia R, Torres-Pereira A, Calado R, Domingues P, et al. Characterization of the cardiac phospholipidome of small cetaceans provides adaptational insight and a foundation for indirect population health screening. Mar Mam Sci 2021;37:1406-1427. https://doi.org/10.1111/mms.12823
25. Yim HS, Cho YS, Guang X, Kang SG, Jeong JY, Cha SS, et al. Minke whale genome and aquatic adaptation in cetaceans. Nat Genet 2014;46:88-92. https://doi.org/10.1038/ng.2835
26. Lang JA, Kim J. Remote ischaemic preconditioning - translating cardiovascular benefits to humans. J Physiol 2022;600:3053-3067. https://doi.org/10.1113/JP282568
27. Alanova P, Alan L, Neckar J, Ostadal B, Kolar F. Cardioprotective Effect of Chronic Hypoxia Involves Inhibition of Mitochondrial Permeability Transition Pore Opening. Physiol Res 2024;73:881-884. https://doi.org/10.33549/physiolres.935427
28. Alanova P, Alan L, Opletalova B, Bohuslavova R, Abaffy P, Matejkova K, et al. HIF-1α limits myocardial infarction by promoting mitophagy in mouse hearts adapted to chronic hypoxia. Acta Physiol (Oxf) 2024;240:e14202. https://doi.org/10.1111/apha.14202
29. Keane M, Semeiks J, Webb AE, Li YI, Quesada V, Craig T, et al. Insights into the evolution of longevity from the bowhead whale genome. Cell Rep 2015;10:112-122. https://doi.org/10.1016/j.celrep.2014.12.008
30. Remili A, McKinney MA, Maldonado-Rodriguez A, Ferguson SH, Caputo M, Kiszka JJ. Legacy persistent organic pollutants among multiple cetacean species in the Northwest Atlantic. Sci Total Environ 2024;954:176746. https://doi.org/10.1016/j.scitotenv.2024.176746
31. Galatius A, Bossi R, Sonne C, Rigét FF, Kinze CC, Lockyer C, et al. PFAS profiles in three North Sea top predators: metabolic differences among species? Environ Sci Pollut Res Int 2013;20:8013-8020. https://doi.org/10.1007/s11356-013-1633-x
32. Lemos LS, Manfrin da Silva E, Steinman KJ, Robeck TR, Quinete N. Assessment of per- and poly-fluoroalkyl substances and physiological biomarkers in aquarium-based bottlenose dolphins and killer whales. Chemosphere 2024;364:143038. https://doi.org/10.1016/j.chemosphere.2024.143038
33. Câmara N, Sierra E, Fernández-Maldonado C, Espinosa de Los Monteros A, Arbelo M, Fernández A, Herráez P. Stress cardiomyopathy in stranded cetaceans: a histological, histochemical and immunohistochemical study. Vet Rec 2019;185:694. https://doi.org/10.1136/vr.105562
34. Benak D, Carter P, Ebr J, Ebrova I, Van Gaever K, Kao B, et al. Sighting of a large group of pygmy sperm whales (Kogia breviceps) and a recent record of a dwarf sperm whale (K. sima) in the Azores. J Marine Biol Assoc United Kingdom 2025;105:e129. https://doi.org/10.1017/S0025315425100830

35. Cowan DF, Curry BE. Histopathology of the alarm reaction in small odontocetes. J Comp Pathol 2008;139:24-33. https://doi.org/10.1016/j.jcpa.2007.11.009
36. Herráez P, Espinosa de los Monteros A, Fernández A, Edwards JF, Sacchini S, Sierra E. Capture myopathy in live-stranded cetaceans. Vet J 2013;196:181-188. https://doi.org/10.1016/j.tvjl.2012.09.021
37. Câmara N, Sierra E, Fernández A, Arbelo M, Andrada M, Espinosa de los Monteros A, Herráez P. Increased Plasma Cardiac Troponin I in Live-Stranded Cetaceans: Correlation with Pathological Findings of Acute Cardiac Injury. Sci Rep 2020;10:1555. https://doi.org/10.1038/s41598-020-58497-3
38. Câmara N, Sierra E, Fernández A, Arbelo M, Bernaldo de Quirós Y, Arregui M, et al. Capture Myopathy and Stress Cardiomyopathy in a Live-Stranded Risso's Dolphin (Grampus griseus) in Rehabilitation. Animals (Basel) 2020;10:220. https://doi.org/10.3390/ani10020220
39. Li W-T, Chou L-S, Chiou H-Y, Chen IH, Yang W-C. Analyzing 13 Years of Cetacean Strandings: Multiple Stressors to Cetaceans in Taiwanese Waters and Their Implications for Conservation and Future Research. Front Mar Sci 2021;8:606722. https://doi.org/10.3389/fmars.2021.606722
40. Ghadri JR, Wittstein IS, Prasad A, Sharkey S, Dote K, Akashi YJ, et al. International Expert Consensus Document on Takotsubo Syndrome (Part I): Clinical Characteristics, Diagnostic Criteria, and Pathophysiology. Eur Heart J 2018;39:2032-2046. https://doi.org/10.1093/eurheartj/ehy076
41. Câmara N, Fernández A, Herráez P, Arbelo M, Andrada M, Suárez-Santana CM, Sierra E. Microscopic Findings in the Cardiac Muscle of Stranded Extreme Deep-Diving Cuvier's Beaked Whales (Ziphius cavirostris). Microsc Microanal 2022;28:1641-1648. https://doi.org/10.1017/S1431927622000605
42. Stimmelmayr R, Rotstein D, Sheffield G, Brower HK, George JC. Diseases and parasites. In: *The Bowhead Whale*. GEORGE JC, THEWISSEN JGM (eds), Academic Press 2021, pp 471-498. https://doi.org/10.1016/B978-0-12-818969-6.00030-3
43. Câmara N, Sierra E, Fernández A, Suárez-Santana CM, Puig-Lozano R, Arbelo M, Herráez P. Skeletal and Cardiac Rhabdomyolysis in a Live-Stranded Neonatal Bryde's Whale With Fetal Distress. Front Vet Sci 2019;6:476. https://doi.org/10.3389/fvets.2019.00476
44. Bossart G, Hensley G, Goldstein J, Kroell K, Manire C, Defran R, Reif J. Cardiomyopathy and Myocardial Degeneration in Stranded Pygmy (Kogia breviceps) and Dwarf (Kogia sima) Sperm Whales. Aquat Mam 2007;33:214-222. https://doi.org/10.1578/AM.33.2.2007.214
45. Díaz-Santana P, Fernández A, Díaz-Delgado J, Suárez-Santana C, Quesada-Canales Ó, Martin V, et al. Pathology and Cause of Death in Stranded Kogiids: A Retrospective Study from the Canary Islands (1999-2018). Animals (Basel) 2026;16:594. https://10.3390/ani16040594.
46. Viricel A, Rosel PE. Looking into a whale's heart: investigating a genetic basis for cardiomyopathy in a non-model species. Genome 2017;60:695-705. https://doi.org/10.1139/gen-2016-0203
47. Wilde AAM, Semsarian C, Márquez MF, Shamloo AS, Ackerman MJ, Ashley EA, et al. European Heart Rhythm Association (EHRA)/Heart Rhythm Society (HRS)/Asia Pacific Heart Rhythm Society (APHRS)/Latin American Heart Rhythm Society (LAHRS) Expert Consensus Statement on the state of genetic testing for cardiac diseases. Europace 2022;24:1307-1367. https://doi.org/10.1093/europace/euac030
48. Linnehan BK, Gomez FM, Huston SM, Hsu A, Takeshita R, Colegrove KM, et al. Cardiac assessments of bottlenose dolphins (Tursiops truncatus) in the Northern Gulf of Mexico following exposure to Deepwater Horizon oil. PLoS One 2021;16:e0261112. https://doi.org/10.1371/journal.pone.0261112
49. Cowan DF. Pathology of the pilot whale. Globicephala melaena. A comparative survey. Arch Pathol 1966;82:178-189.
50. Díaz-Delgado J, Sierra E, Vela AI, Domínguez L, Andrada M, Arbelo M, Fernández A.. Endocarditis associated with Wohlfahrtiimonas chitiniclastica in a short-beaked common dolphin (Delphinus delphis). J Wildl Dis 2015;51:283-286. https://doi.org/10.7589/2014-03-072
51. Siebert U, Müller G, Desportes G, Weiss R, Hansen K, Baumgärtner W. Pyogranulomatous myocarditis due to Staphylococcus aureus septicaemia in two harbour porpoises (Phocoena phocoena). Vet Rec 2002;150:273-277. https://doi.org/10.1136/vr.150.9.273
52. González-Barrientos R, Morales JA, Hernández-Mora G, Barquero-Calvo E, Guzmán-Verri C, Chaves-Olarte E, Moreno E. Pathology of striped dolphins (Stenella coeruleoalba) infected with Brucella ceti. J Comp Pathol 2010;142:347-352. https://doi.org/10.1016/j.jcpa.2009.10.017

53. Grattarola C, Petrella A, Lucifora G, Di Francesco G, Di Nocera F, Pintore A, et al. Brucella ceti Infection in Striped Dolphins from Italian Seas: Associated Lesions and Epidemiological Data. Pathogens 2023;12:1034. https://doi.org/10.3390/pathogens12081034
54. Esposito E, Oliviero M, Iaccarino D, Paduano G, Serra F, Levante M, et al. Post Mortem Findings of Cetaceans Stranded Along the Campania Coast from 2016 to 2022. Animals (Basel) 2025;15:1812. https://doi.org/10.3390/ani15121812
55. Grattarola C, Gallina S, Giorda F, Pautasso A, Ballardini M, Iulini B, et al. First report of Salmonella 1,4,[5],12:i:- in free-ranging striped dolphins (Stenella coeruleoalba), Italy. Sci Rep 2019;9:6061. https://doi.org/10.1038/s41598-019-42474-6
56. Grattarola C, Minoia L, Giorda F, Consales G, Capanni F, Ceciarini I, et al. Health Status of Stranded Common Bottlenose Dolphins (Tursiops truncatus) and Contamination by Immunotoxic Pollutants: A Threat to the Pelagos Sanctuary-Western Mediterranean Sea. Diversity 2023;15:569. https://doi.org/10.3390/d15040569
57. McAloose D, Rago MV, Di Martino M, Chirife A, Olson SH, Beltramino L, et al. Post-mortem findings in southern right whales Eubalaena australis at Península Valdés, Argentina, 2003-2012. Dis Aquat Organ 2016;119:17-36. https://doi.org/10.3354/dao02986
58. Bianchi MV, Ehlers LP, Vargas TP, Lopes BC, Taunde PA, de Cecco BS, et al. Omphalitis, urachocystitis and septicemia by Streptococcus dysgalactiae in a southern right whale calf Eubalaena australis, Brazil. Dis Aquat Organ 2018;131:227-232. https://doi.org/10.3354/dao03293
59. Fernández A, Edwards JF, Rodríguez F, Espinosa de los Monteros A, Herráez P, Castro P, et al. "Gas and fat embolic syndrome" involving a mass stranding of beaked whales (family Ziphiidae) exposed to anthropogenic sonar signals. Vet Pathol 2005;42:446-457. https://doi.org/10.1354/vp.42-4-446
60. Jepson PD, Deaville R, Patterson IA, Pocknell AM, Ross HM, Baker JR, et al. Acute and chronic gas bubble lesions in cetaceans stranded in the United Kingdom. Vet Pathol 2005;42:291-305. https://doi.org/10.1354/vp.42-3-291
61. Hooker SK, Fahlman A, Moore MJ, Aguilar de Soto N, Bernaldo de Quirós Y, Brubakk AO, et al. Deadly diving? Physiological and behavioural management of decompression stress in diving mammals. Proc Biol Sci 2012;279:1041-1050. https://doi.org/10.1098/rspb.2011.2088
62. Szatmári V, Bunskoek P, Kuiken T, van den Berg A, van Elk C. Echocardiographic diagnosis and necropsy findings of a congenital ventricular septal defect in a stranded harbor porpoise. Dis Aquat Organ 2016;118:177-183. https://doi.org/10.3354/dao02973
63. Powell JW, Archibald RT, Cross CA, Rotstein DS, Soop VM, McFee WE. Multiple congenital cardiac abnormalities in an Atlantic bottlenose dolphin (Tursiops truncatus). J Wildl Dis 2009;45:839-842. https://doi.org/10.7589/0090-3558-45.3.839



Fig. 3. Sperm whale (Physeter macrocephalus), Pico, Azores



Fig. 4. Sperm whale (Physeter macrocephalus), Pico, Azores