Cumulative Sperm Whale Bone Damage and the Bends

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Osteonecrosis, a chronic pathology of deep diving recognized in humans, is shown here to be a progressive condition in sperm whales, suggesting that the long-held dogma of complete immunity to decompression sickness ("the bends") in marine mammals should be revisited and that acute embolic disease may result from disruption of normal dive patterns.

Postmortem examination (*1*) of a 14.7-m adult male sperm whale, specimen NBWM 2003.95, showed that rib and chevron bone articulations, nasal bones, and deltoid crests (Fig. 1, fig. S1, and table S1) were pitted and eroded, much as described by Flower in 1868 (*2*). The lesions were multifocal and bilateral but asymmetric. Intervertebral disc and fused epiphyseal plate structure appeared normal, whereas a minority of zygapophyseal facets showed the same erosion and remodeling. Computer tomographs and x-rays showed good cortical bone density in the flipper, chevron bones, and vertebrae, with dense lamellar cortex and less dense medullary spongiform structure, but with joint surface erosions evident where the ribs and chevron bones articulated with the vertebrae and sternebrae. Histological examination revealed multifocal, chronic, and marked bone and cartilage erosion, ulceration, degeneration, and extensive proliferation and remodeling of cartilage and woven bone. Aetiology was unclear but premortem, with no histological indication of any infectious disease. Ziehl-Neelsen staining failed to show any definitive acid-fast bacteria. Bacterial cultures revealed no growth of organisms considered to be premortem pathogens. Culture for *Brucella* spp. was negative.

Consequent to these findings, we surveyed (*1*) 16 partial or complete sperm whale skeletons from the Pacific and Atlantic oceans, collected over a 111-year time span, of a broad size and age range. As body length increased, there was an increase in severity of bilateral erosion and remodeling of rib and chevron bone subarticular surfaces and deltoid crests (Fig. 1, fig. S1, and table S1). In calves, these showed regular small nutrient foramina. The nasal bones showed regular branching vessel indentations radiating from the lateral margin toward the midline. Early changes in juvenile rib (Fig. 1) and chevron bone (fig. S1A) subarticular surfaces included erosion around nutrient foramina and single, large, semispherical cavitations of 1.5 to 2.0 cm in diameter. In larger animals of both sexes, the regular pattern of nutrient artery foramina was further obscured by a mass of fenestrated, eroded, and remodeled bone in chevron and rib articular surfaces, deltoid crests (Fig. 1 and fig. S1), and nasal bones. The earliest stage of the nasal bone change was found in a 7.3-m male, in which the vascular tree was still evident, but substantial remodeling was already present.

Fig. 1. Progressive, erosive, and remodeling development of dysbaric osteonecrosis in sperm whale subarticular rib bone surfaces. The top to bottom panels show a progression from calf to mature adult. Scale bar 2 cm top panel, 1 cm rest. Dark areas indicate the vascular channels, which appear normal in the calf and increasingly eroded and enlarged in larger animals. Accession numbers top to bottom (table S1): MCZ1209, MCZ61406, NBWM 2003.95, and NBWM 2003.95.

Osteonecrosis best describes the bony changes we observed (supporting online text). Similar lesions have been attributed to dysbaric stress in fossil diving mosasaurs (*3*) and plesiosaurs (*4*).

In humans (*5*), osteonecrosis can follow dysbaric stress, hemoglobinopathy, hemopoietic disorders, hyperadrenocorticism, irradiation, or thermal injuries. Establishing the primary cause of osteonecrosis is usually based on history. All sperm whales routinely undergo dysbaric stress, whereas we have no cultural or histological evidence for any other single known cause of osteonecrosis that could have affected all the adult sperm whales we examined from the Atlantic and Pacific oceans over 111 years. Thus, the most parsimonious hypothesis is that nitrogen emboli induced the observed osteonecrosis.

It therefore appears that sperm whales may be neither anatomically nor physiologically immune to the effects of deep diving. This opens the question of decompression issues constraining surfacing behavior and implies that they and probably other cetacea may be open to acute embolic injury if forced to surface rapidly. The recent description of acute decompression-like sickness in beaked whales exposed to military sonar (*6*) may, therefore, reflect acute nitrogen embolism resulting when decompression sickness avoidance behavior, such as the dive traces previously described in sperm whales (*7*), is overridden by extended surfacing (supporting online text).

References and Notes

1. Materials and methods are available as supporting material on *Science* Online.
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Supporting Online Material

www.sciencemag.org/cgi/content/full/306/5705/2215/DC1
Materials and Methods
SOM Text
Fig. S1
Table S1

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