

# Crassicaudosis: a parasitic disease threatening the health and population recovery of large baleen whales

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**Summary:** *This communication briefly reviews knowledge of the systemic disease caused by Crassicauda boopis in blue whales (Balaenoptera musculus), fin whales (B. physalus) and humpback whales (Megaptera novaeangliae). Infections with this giant nematode characteristically incite a chronic inflammatory reaction of the blood vessels which drain the kidneys. In this critical location, the parasite-induced lesion can cause complete vascular occlusion and kidney failure. Whale calves and juveniles typically suffer the heaviest parasite burdens following transplacental infection of the developing whale foetus. There is also probable whale-to-whale transmission post-partum, involving urinary contamination of the environment with C. boopis eggs and larvae. The frequency of the infection can exceed 95%. Haematological findings suggest that systemic pathological effects are typical at the population level. Gradual development of occlusive lesions in the renal veins appears to correlate with a major peak in natural mortality at about one year of age. To date, all findings support the conclusion that premature death caused by C. boopis infection is potentially a major impediment to population recovery of affected whale species. This suggests the interesting possibility of actively encouraging the population recovery of three species of large baleen whales. Such a restoration effort would entail remotely-deployed anthelmintic therapy administered, at sea, to infected whale cows and calves.*

**KEYWORDS:** Balaenoptera – Conservation – Crassicauda boopis – Disease – Kidney – Megaptera – Ocean systems management – Parasitism – Pathology – Whale.

## INTRODUCTION

The mammalian order Cetacea (whales, dolphins and porpoises) has evolved with a group of parasites belonging to the genus *Crassicauda* (order Spirurida). Infections with these nematodes are endemic in both the toothed and baleen whales. Such infections are a major cause of disease of the urinary, respiratory and digestive systems.

Of several known crassicaudid infections, those caused by *Crassicauda boopis* are especially pathogenic. This giant worm infects the highly endangered blue whale (*Balaenoptera musculus*), the humpback whale (*Megaptera novaeangliae*) and the fin

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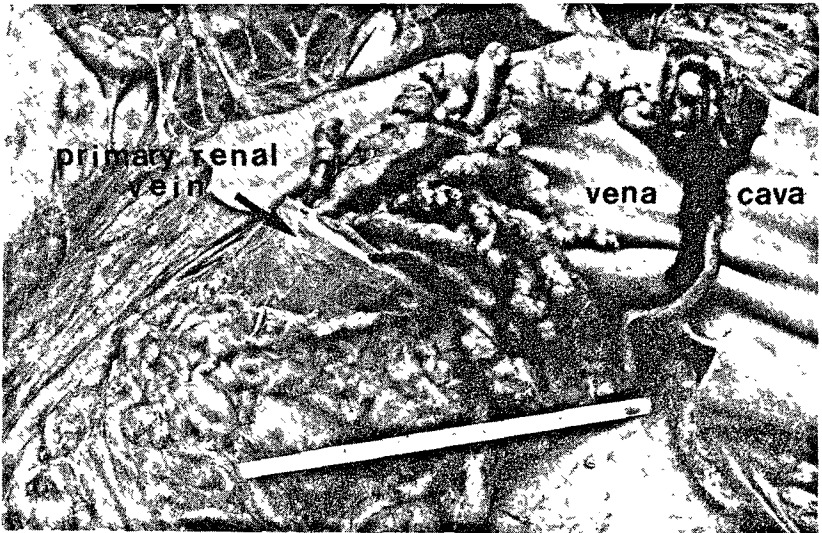
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whale (*B. physalus*) (2, 10, 11, 15, 23, 25, 26). Migrations of parasitic larvae in the tissues of the cetacean host are associated with extensive inflammation and sclerosis of the arteries supplying the gut (16). The maturing worms attain their definitive site in the vascular and ureteral system of the kidneys, where they grow to a length of two metres, feeding on venous blood (16).

## PATHOLOGY, CLINICAL LABORATORY MEDICINE AND EPIZOOTIOLOGY

Adult *C. boopis* in the renal veins typically induce a pronounced tissue reaction. Host cells of the vessel wall proliferate in an attempt to encapsulate the parasites. Lesions weighing five kilograms or more are formed in a process characterised by chronic severe occlusive phlebitis (Fig. 1). These lesions ultimately extend into the vena cava, mineralise and form a nidus for venous thrombosis (16, 17). Fragmentation of the intravascular lesions can lead to localised thromboembolic lesions in the lungs (5).

The mesenteric arterial lesions associated with this infection undoubtedly compromise digestive function, impairing vigour and health. However, the mechanism of death in severe cases is evidently due to complete occlusion of the renal veins, massive swelling and abscessation of the kidneys, and congestive kidney failure. Pyonephrosis involving both kidneys has been found in conjunction with bilateral occlusions of the renal veins (17).



Scale: 15 cm (6 in)

**FIG. 1**

### **Pathological lesion induced by adult *Crassicauda boopis* in the renal vein and vena cava of a fin whale**

The vena cava is opened. The tissue lesion surrounds the invading worms, obscuring them from view, and partially obstructs the vena cava. The primary renal vein is completely occluded by host reaction to the adult parasites

Biochemical studies corroborate this mechanism of death. In a clinical laboratory survey of thirty-one fin whales taken in a commercial whaling operation, serum from one sexually immature female showed chemical evidence of renal failure (19). The concentrations of serum urea nitrogen (114 mg/dl) and serum creatinine (3.5 mg/dl) were the highest found in any of the whales examined. Deviations consistent with renal failure occurred in serum electrolytes (elevated phosphate and potassium concentrations, with depressed calcium and chloride). Similarly, a 13.8 m female fin whale which stranded and died on the coast of New England, United States of America (USA) in 1977 showed a markedly elevated serum creatinine concentration (5.7 mg/dl). Judging from the body length, this animal was almost certainly a yearling calf. Large digitate masses were found in the renal veins and crassicaudids were present in the kidneys. The apparent cause of death of this animal was diagnosed as kidney failure secondary to *Crassicauda* infection (16; J.L. Dunn, personal communication). In another case, a moribund 18.9 m adult fin whale which stranded in Virginia, USA in 1987 had a markedly elevated serum creatinine concentration (5.2 mg/dl) and a very high eosinophil count (3,042 per  $\mu\text{l}$ ) (9). For comparison, chemical values determined in the serum of 29 apparently normal fin whales caught in a commercial whaling operation off south-west Iceland are given in Table I. White cell counts of animals belonging to the same population are given in Table II.

TABLE I

*Normal chemical values in serum of twenty-nine fin whales (Balaenoptera physalus) caught off south-west Iceland in 1985 (19)*

Substance	Mean	Range	SD
Na <sup>+</sup> (mEq/litre)	152.0	135–188	11.7
K <sup>+</sup> (mEq/litre)	5.2	3.6–9.3	1.4
Cl <sup>-</sup> (mEq/litre)	113.0	103–124	5.8
Ca <sup>++</sup> (mg/dl)	11.0	8.7–12.9	0.9
Mg <sup>++</sup> (mg/dl)	3.1	2.0–4.7	0.7
P (mg/dl)	8.2	4.7–12.1	1.6
SUN (mg/dl)	79.1	57–98	10.1
Creatinine (mg/dl)	1.3	0.3–3.0	0.6
Total protein (g/dl)	6.2	5.2–7.3	0.6
Albumin (g/dl)	4.0	2.8–4.8	0.4
Globulin (g/dl)	2.2	0.9–4.1	0.6
A/G ratio	2.0	0.7–5.1	0.8

SD: standard deviation  
mEq: milli-equivalent  
SUN: serum urea nitrogen

Quantitative parasitology and haematology provide evidence that *C. boopis* causes adverse systemic effects which are typical at the population level. Two of four blue whales examined in the British Isles by Hamilton (11) circa 1913-1914 had large *Crassicauda* lesions extending into the venae cavae. Matthews (26) reported that *Crassicauda* nematodes were found in the kidneys of 23 of 35 (66%) humpback whales examined for this infection as part of the Discovery Committee investigations in South Georgia Island and in Saldanha Bay (South Africa) between 1924 and 1931. On the

**TABLE II**  
***Absolute and differential white-cell counts of fin whales***  
***(Balaenoptera physalus) caught off south-west Iceland in 1985***

Cell type/form	No. of whales	White-cell count	
		Range	Mean ( $\pm$ SD)
		per $\mu$ l	per $\mu$ l
Total leukocytes	47	2,770 – 16,340	5,464 ( $\pm$ 2,493)
Total neutrophils	47	432 – 4,623	1,822 ( $\pm$ 986)
Metamyelocytes	47	0 – 110	4 ( $\pm$ 18)
Band forms	47	0 – 89	15 ( $\pm$ 24)
Segmenters	47	402 – 4,534	1,803 ( $\pm$ 981)
Lymphocytes	47	603 – 3,448	1,533 ( $\pm$ 571)
Monocytes	47	34 – 651	219 ( $\pm$ 146)
Eosinophils	47	507 – 9,477	1,876 ( $\pm$ 1,445)
		%	%
Total neutrophils	48	8.8 – 63.0	32.7 ( $\pm$ 11.1)
Metamyelocytes	48	0 – 2.0	0.09 ( $\pm$ 0.35)
Band forms	48	0 – 1.1	0.26 ( $\pm$ 0.38)
Segmenters	48	8.8 – 62.6	32.6 ( $\pm$ 11.3)
Lymphocytes	48	15.0 – 56.5	29.7 ( $\pm$ 8.6)
Monocytes	48	0.5 – 20.4	4.4 ( $\pm$ 3.2)
Eosinophils	48	14.5 – 58.0	32.7 ( $\pm$ 9.9)

SD: Standard deviation

Note: All counts corrected for presence of nucleated red blood cells; 200-400 cells scored per differential determination. Data collected by Dr D. Atkins. Expanded data set from (17), with subcounts for neutrophilic series

basis of inspections of whale carcasses examined aboard floating factories between 1948 and 1952, Cockrill (5) described *Crassicauda* infections to be "common and widespread" in fin, blue and humpback whales in the southern hemisphere. In a more recent study (1981-1987), the frequency of *C. boopis* infection in fin whales caught in the Icelandic coastal whaling operation was found to average over 95% ( $n = 196$ ) (17).

Although the existing data are not sufficient to determine with certainty whether the frequency of *C. boopis* infection has increased, there are indications that the health of fin whales is deteriorating in the North Atlantic Ocean. In their analysis of biological data collected from fin whales caught by Icelandic whalers, Lockyer and Sigurjonsson (24) found significant changes in vital parameters from 1967 to 1988. The average age at sexual maturity of females increased from 6-7 to 9-10 years, while that of males increased from 7-8 to 12 years. This indicates a significant slowing in the rate of body growth, because body length at sexual maturity remained constant during the same period (24).

Haematological studies of the same fin whale population in 1985 revealed the pronounced eosinophilia typical of heavy internal parasitism (17). Mean eosinophil counts exceeded 1,800 cells/ $\mu$ l of blood despite surprisingly low total white cell counts (5,464 cells/ $\mu$ l) (Table II). Although comparable haematological data do not exist for humpback or blue whales, the mean relative eosinophil count (33%) found in fin whales

landed in Iceland represents the highest known in any cetacean. In contrast, blood eosinophils are reportedly rare in the grey whale (*Eschrichtius robustus*), a baleen whale known to be free of crassicaudosis (29). In the North Atlantic fin whale, extensive mesenteric arteritis, chronic occlusive phlebitis and endemic *C. boopis* infection were all associated with haematological evidence of systemic disease.

From these combined findings, it appears that the severity of crassicaudosis, and perhaps also the frequency of the infection, may be increasing in the North Atlantic. Increased disease could account for the noted trend of declining growth rates in fin whales over the past two decades. If host populations are expanding, one would naturally expect increases in the rate of transmission of this infection and in consequent rates of morbidity and mortality (17). However, another factor of critical importance has been established by long-term oceanographic studies. Since 1958, there has been a consistent, gradual 15-50% decline in population numbers of essentially all major zooplankton taxa in the north-eastern Atlantic and North Sea (6). Whether this is due to climatic, anthropogenic or other influences, it points directly to a reduction in food supply as a primary cause of the deteriorating condition of North Atlantic fin whales. Notably, poor nutrition could lead to an increase in the severity of endemic parasitism by a compromising effect on disease resistance, even if numbers of whales are decreasing.

With respect to the age-specific measures of severity, worm counts obtained in detailed examinations of the kidneys of 93 fin whales in Iceland indicated that, on average, young whales experience the heaviest *C. boopis* burdens (unpublished findings). This age variation is evidently attributable to an initial mode of infection by the transplacental route. Although urinary contamination of the marine environment with *C. boopis* eggs and larvae is known and implies oral infection of post-natal whales (16), transplacental infection is indicated by the finding of nearly adult-sized *C. boopis* in the vena cava and renal veins of a new-born fin whale calf. This animal (USNMNH 484994) was only 6.65 m long and washed ashore dead in Delaware, USA in 1973 (J. Mead, personal communication).

Transplacental infection, followed by chronic occlusive phlebitis of the renal veins in the post-natal calf, corresponds to a major peak in mortality at approximately one year of age. An analysis of beachings of 148 fin whales and 54 humpback whales in the North Atlantic revealed this peak (data provided by the National Museum of Natural History, Smithsonian Institution, Washington D.C., USA). When demographically weighted under the conventional assumption of a 4% mortality rate (1), 53% (fin whale) to 86% (humpback whale) of this stranding mortality was attributable to calves which were estimated (on the basis of body length) to be 0.5-1.5 years old (i.e. 5-10 m humpback, 7-14 m fin whale).

Serious adverse effects of crassicaudosis may also be occurring on evolutionary time scales. Histopathological evidence points to the likely importance of cellular immune defense mechanisms, including the monocyte-macrophage system, in reducing successful larval migration into the kidneys and foetus (16). Even so, the occlusive nature of the lesions which form in the renal veins (Fig. 1) suggests that, paradoxically, *C. boopis* would kill those whales which mount the strongest defence reaction against the adult worm. It is the exuberance of the chronic inflammatory reaction to the giant parasite in this critical location which has the potential to cause host death.

In this way, *C. boopis* appears specially adapted for maintaining affected whale species in an abnormal state characterised by a genetic susceptibility to systemic

infection. This may account for the very high frequency of the infection (95%), the gigantic size of the mature parasite (2 m), its obvious evolutionary success and apparent virulence.

## PROPOSED CONSERVATION INITIATIVE

In view of all these findings, a veterinary programme at sea should be explored as a conservation measure (18). Current knowledge supports the conclusion that such action might succeed in reducing the morbidity and mortality caused by *C. boopis* in blue, humpback and fin whale populations. Appropriate veterinary action would involve single dose anthelmintic therapy administered by ballistic syringe to free-ranging whale calves. Treatment of the whale cow in order to reduce transplacental infection of the foetus would also be appropriate if the problem of large dose volume can be managed. In either case, the potent but safe anthelmintics known as avermectins (4) would probably be the medication of choice.

The avermectins are fermentation products of the soil microbe *Streptomyces avermitilis* which have a very short environmental half-life when exposed to light (a number of hours or days) (4). Avermectins which appear especially well-suited for use in marine systems have been developed experimentally and may soon be produced on a larger scale. Since such preparations are available, a series of closely monitored experimental treatments should be conducted to confirm the safety of these substances for use in baleen whales. The commercially-produced avermectin (known as ivermectin) may also be suited for use in the marine system.

The primary therapeutic objective would be to eliminate *C. boopis* infection in the calf prior to the development of life-threatening lesions. This should improve the chances of the calf surviving through the brief but critical weaning period. Preventing premature death of only one female calf could result in an increment of ten or more new animals to the population over the course of the reproductive life span of the female. **Single dose** avermectin therapy can also significantly increase the rate of growth of young land mammals – a benefit which correlates with an accelerated onset of sexual maturity, increased fertility and increased fecundity (12). If the same therapeutic effect is achieved, a comparatively modest human effort could substantially increase the reproductive capacity of depleted whale herds.

## ECOLOGICAL CONSIDERATIONS

The important ecological roles played by these species should also be accepted as good reason for action (7). Calculations performed by Laws (20) suggest that depletion of baleen whale populations by man has caused a significant reduction in the natural transport of ecologically useful biomass from productive polar regions to nutrient-impooverished lower latitudes. This transport of biomass is due to natural mortality in low latitudes of whales which feed mostly in the higher latitudes of the Southern Ocean. The figures provided by Laws for “initial” and “present” (1977) stocks of blue, fin and humpback whales yield an estimated annual reduction of 1.1 million metric tons in one-way transport of biomass in the Southern Hemisphere alone (20), which corresponds to a decrease of 89% compared with the estimate for the period prior to pelagic whaling.

Since 1977, the magnitude of this annual adverse impact appears to have changed very little. Shipboard surveys conducted between 1978 and 1984 found very few fin and blue whales in Antarctic waters despite the considerable effort invested in attempted sightings (14). On this basis, the noted reduction in yearly transport of ecologically useful biomass into nutrient-poor lower latitudes will presumably have undesirable repercussions well into the next century.

While prolonged global depletion of large baleen whales may not be widely perceived as an “eco-crisis” when compared with many of the more visible environmental problems facing the Earth, the result is a marine system which is seriously out of balance. The above estimates suggest that pelagic whaling has altered not only the composition, but also the spatial dynamics of marine communities. One very clear point is that the initial biotic shift which occurred in reaction to this perturbation now serves to impede systems recovery. In the long term, the character and size-structure of this biotic shift will probably reduce the useful production of the sea.

A dramatic increase in the abundance of local populations of seals and penguins promptly followed human depletion of large baleen whales in the Southern Ocean (8, 21, 22, 27, 28). These indigenous species serve no major function in biomass transport across latitudes, because their home range is mostly restricted to near-polar habitats. Of little use to humans, they also are far less metabolically efficient than baleen whales because of their comparatively small body size.

In this context, the ecological consequences of the depletion of large baleen whales can be better understood by applying the principle of allometric scaling of metabolism. With reasonable consistency between taxonomic groups, basal metabolic rate varies in proportion to body mass raised to the power of 0.75 (3). Hence, it can readily be estimated that the replacement of one 100,000 kg blue whale with an equal mass of crabeater seals (*Lobodon carcinophagus*) each weighing 200 kg **increases** the rate of food and oxygen consumption required for survival by about 470%. For biomass structured in the form of 5 kg chinstrap penguins (*Pygoscelis antarctica*), the corresponding change is a 1,200% increase. In terms of metabolic output, the small species which characterise this biotic shift likewise excel as producers of “waste” heat and carbon dioxide (3, 13). These inefficiencies all reduce the useful productivity of the sea and may have serious environmental consequences when combined with the massive change in community structure which has occurred (13).

In conclusion, the cumulative adverse impact of past human excess in the catching of large baleen whales still appears to be increasing. It is exacerbated by any factor which favours the persistence of unnaturally large populations of warm-blooded competitors of small body size. Crassicaudosis, without doubt, is one such factor.

Crassicaudosis is a potentially lethal endemic disease which afflicts blue, fin and humpback whales throughout the world, causing substantial morbidity in those animals which survive. Thus, it impedes the population recovery of these gigantic species and favours the proliferation of smaller-bodied, reproductively active competitors which are less energy efficient.

In this knowledge, some immediately see the promise of an effective method to help restore the balance of the Earth’s perturbed marine systems. For, unlike many ecological problems which confront us today, diseases caused by nematodes can usually be controlled by (medical) technology. Especially important is the fact that veterinary

action at sea to stimulate the population recovery of large baleen whales could bring **self-sustaining** improvements in the vigour and efficiency of global systems. Each therapeutic success would establish new potential for perpetually increasing ecological gain. In all probability, this would lead to further environmental improvements which might otherwise be impossible to achieve and sustain.

In this light, the challenge for modern society is to comprehend the whale problem in all its facets and to appreciate the full cost of a failure to act. The challenge is also to recognise that describing the problem is only one step in the development of solutions. The uniquely positive option of creative stewardship needs to be actively explored.

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#### LA CRASSICAULOSE, UNE MALADIE PARASITAIRE MENAÇANT LA SANTÉ ET LA RECONSTITUTION DES POPULATIONS DE GROS MYSTICÈTES. – R.H. Lambertsen.

*Résumé* : L'auteur fait le point sur les connaissances concernant les effets systémiques des infestations à *Crassicauda boopis* chez la grande baleine bleue (*Balaenoptera musculus*), le rorqual commun (*B. physalus*) et la jubarte (*Megaptera novaeangliae*). Les infestations par ce nématode géant entraînent une réaction caractéristique d'inflammation chronique des vaisseaux sanguins irriguant les reins. Dans cette localisation critique, la lésion due au parasite peut provoquer une occlusion vasculaire totale, puis une insuffisance rénale. Les nouveau-nés et les jeunes sont généralement les plus parasités, par suite des contaminations fœtales in utero. La transmission entre baleines est également probable après la naissance, en raison de la contamination urinaire de l'environnement par les œufs et les larves de *C. boopis*. La fréquence de l'infestation peut dépasser 95 %. Les résultats des analyses hématologiques tendent à indiquer qu'à l'échelle des populations, les effets systémiques du parasite sont pathognomoniques. Le développement progressif de lésions oblitérantes des veines rénales apparaît en corrélation avec l'un des principaux pics de mortalité naturelle que l'on observe vers l'âge d'un an. A ce jour, toutes les études confirment que la mort prématurée due aux infestations par *C. boopis* est une entrave potentielle majeure à la reconstitution des espèces de cétacés



atteintes. Il pourrait donc paraître intéressant d'encourager activement la reconstitution de trois espèces de gros mysticètes. Cet effort impliquerait l'administration de traitements anthelminthiques à distance, en mer, aux femelles et aux nouveau-nés.

MOTS-CLÉS : Baleinoptère – Cétacé – Conservation – *Crassicauda boopis* – Gestion des systèmes océaniques – Maladie – Mégaptere – Parasitisme – Pathologie – Rein.

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## LA CRASICAUDOSIS, UNA ENFERMEDAD PARASITARIA QUE REPRESENTA UNA AMENAZA PARA LA SALUD Y PARA LA RECONSTITUCIÓN DE LAS POBLACIONES DE LOS GRANDES MISTICETOS. – R.H. Lambertsen.

**Resumen:** El autor resume los conocimientos relativos a la enfermedad sistémica provocada por *Crassicauda boopis* en las ballenas azules (*Balaenoptera musculus*), los rorcuales (*B. physalus*) y los jubartes (*Megaptera novaeangliae*). Las infecciones por este nematodo gigante provocan una reacción característica de inflamación crónica de los vasos sanguíneos que drenan el riñón. En esa región crítica, la lesión producida por el parásito puede provocar oclusión vascular total e insuficiencia renal. Las crías recién nacidas y los animales jóvenes son por lo general los más invadidos por el parásito como consecuencia de la contaminación fetal in utero. La transmisión postparto de ballena a ballena es también probable dada la contaminación urinaria del medio ambiente con huevos y larvas de *C. boopis*. La frecuencia de la infección puede ser superior a un 95%. Los resultados de los análisis hematológicos parecen indicar que los efectos sistémicos de la enfermedad son típicos a nivel de la población. El desarrollo progresivo de lesiones oclusivas en las venas renales parece correlacionado con el incremento de la mortalidad natural observado cuando los animales tienen alrededor de un año de edad. Hasta la fecha, todos los resultados confirman que la muerte prematura debida a la infección por *C. boopis* constituye en potencia un gran obstáculo para la reconstitución de las especies de ballenas afectadas. Interesa por lo tanto fomentar activamente la reconstitución de la población de tres especies de grandes misticetos. Esa reconstitución implicaría la administración a distancia de una terapia antihelmíntica a las hembras y las crías infectadas en el mar.

PALABRAS CLAVE: *Balaenoptera* – Ballena – Conservación – *Crassicauda boopis* – Enfermedad – Gestión de sistemas oceánicos – *Megaptera* – Parasitismo – Patología – Riñón.

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