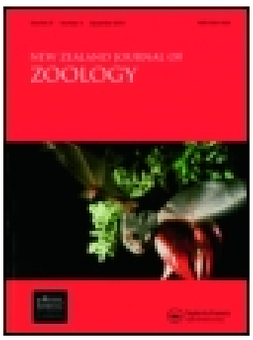

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Cause of death in blue penguins (*Eudyptula m. minor*) in North Otago, New Zealand

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Abstract I necropsied 213 blue penguins (*Eudyptula m. minor*) collected during 1994 to 1998, mainly from Oamaru Harbour and urban areas, the remainder from sites south to Otago Peninsula. The dominant categories were malnutrition (16%), and cause of death (15%). Where malnutrition was involved, it was usually unclear whether it was the direct cause of death or the consequence of undiagnosed natural disease or infestation. Twenty five per cent of birds died from animal attacks. Aspergillosis was the commonest natural disease (3.3%). Endoparasitism was uncommon (1.3%). This study provides significant evidence of geographic variation in infestation of blue penguins.

Keywords blue penguin; *Eudyptula minor*; New Zealand; mortality, starvation, predation

INTRODUCTION

North Otago, on the east coast of the South Island of New Zealand, supports a population of more than 3000 blue penguins (*Eudyptula m. minor*). Some of their colonies have been intensively monitored since 1992 (Dann 1994; Hocken 1997; D. M. Houston, unpubl.; see Kinsky & Falla (1976) for taxonomy). Because of intense public interest in the Oamaru Blue Penguin Colony, birds picked up dead and injured are frequently brought to the Oamaru Field Centre of the Department of Conservation.

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There is limited information on causes of mortality of New Zealand blue penguins (Crocket & Kearns 1975). This study is the first to categorise cause of death for a New Zealand blue penguin population. It also makes some comparisons between the known causes of death of blue penguins in Otago and in Australia (Harrigan 1992; Obendorf & McColl 1980).

MATERIALS AND METHODS

This work presents necropsy findings on 213 fledged blue penguins, opportunistically collected during 1994 to 1998. Of these, 137 (64.8%) came from Oamaru Harbour and the adjacent town, with a further 23 (to 75.6%) from the North Otago area and the rest from the Otago Coast south to Otago Peninsula.

Body weight and external body measurements were recorded. Birds were opened ventrally, skinned full length looking for subcutaneous injury (e.g., bruising or bite marks), then dissected. No examination for ectoparasites was made. Nutritional condition was assessed from fat stores and pectoral muscle bulk. The absence of any subcutaneous fat on the trunk, and loss of pectoral muscle bulk, defined the category 'poor nutrition' (Gill & Darby 1993; Obendorf & McColl 1980). Deduction of the cause of death was assisted by ancillary information on the circumstances of recovery; for example, wet lungs and fluid-containing air sacs in a bird collected from the surf line was taken to suggest death by drowning.

Eight major categories of cause of death were defined.

1. Unknown – indicates birds, which were not starving, in which there was no evidence of disease or physical injury.
2. Starvation – no morbid condition visible other than malnutrition. All birds' stomachs were opened and the contents examined particularly for residues, endoparasites, ulceration and evidence of bleeding. The intestine was examined externally only, noting the colour for indication of blood content.

3. Trauma – evidence of physical injury, without the characteristics of animal attack or vandalism. This category included injury by road or rail traffic. Trauma due to some unrecognised cause was categorised 'trauma, unspecified' (Table 1).
4. Predation – physical injuries characteristic of animal attack, such as a dog (*Canis familiaris*), mustelid (*Mustela* spp) or 'shark'. I acknowledge that predation is normally a food chain interaction, which will not generally apply to dog attack on penguins. The term 'shark' is taxonomically loose, referring to all predatory fish. It was applied to birds with linear skin incision (cut) of the body, accompanied by cuts in the plumage, reminiscent of cutting with scissors.
5. Natural – disease or morbid deformity, as distinct from predation, even though predation is a natural event for wildlife.
6. Drowned.
7. Vandalism – malignant human interference or an injury deliberately inflicted by human agency.
8. Trapped – killed or irrecoverably injured as a consequence of predator control measures.

RESULTS

The causes of death assigned to the 213 adult and fledged juvenile blue penguins are summarised in Table 1. The category 'Unknown' (32/213, 15%) is a major group, whilst nearly 16% of birds died with or of starvation.

Gastrointestinal bleeding was a feature (16/34, 47%) of birds dying with or of starvation. Neither gastric parasites nor ulceration were a feature of such bleeding. I found gastric nematodes in only 1.3% (3/213) of blue penguins, compared with the 37% infestation rate in 48 yellow-eyed penguins (*Megadyptyes antipodes*) over the same period and collection environment.

The presence of well rounded gravel (stone particles ≥ 2.0 mm) in the stomachs of penguins is not associated with morbidity. By contrast, angular pieces of shell and glass are apparently morbid, usually associated with other bizarre gastric contents (pica) such as vegetable material and/or feathers, and are seen only, albeit less frequently (6/34, 18%), in starvation.

Signs of dog attack are characteristic: pectoral muscle damage (dislocation or rupture), accompanied by internal skin lacerations that are not necessarily evident externally (Harrigan 1992). Sometimes the only external sign of visceral crushing (as in the jaws of a large dog) is a bloody oral discharge. Mustelid predation upon blue penguins in North Otago is invariably due to ferrets (*Mustela furo*), and produces a characteristic sub occipital bite injury. Careful searching has failed to find any evidence of cranial puncture. The injury can appear trivial to external examination. Skinning the dorsum

Table 1 Cause of death in 213 adult blue penguins from northern Otago; identifying 138 birds from Oamaru, North Otago.

Cause of death	Total birds	% of total	Oamaru birds	% Oamaru birds
Unknown	32	15	19	13.8
Starvation	34	15.9	16	11.6
Trauma				
Road	22	10.3	20	14.6
Rail	8	3.75	8	5.8
Unspecified	20	9.4	16	11.6
Predation				
Dog	30	14	16	11.6
Mustelid	20	9.4	15	10.9
Shark	8	3.75	3	2.2
Unspecified	5	2.3	2	1.4
Natural	11	5.1	4	2.9
Drowned	10	4.7	8	5.8
Vandalism	10	4.7	10	7.3
Trapped	3	1.4	1	0.7
Totals	213 (100%)		138 (64.8%)	

of the cranium and upper neck reveals minor sub occipital muscle damage and bruising, with fine punctures of the skin. Mustelids feeding on a penguin carcase characteristically strip the cervical skeleton toward the pectoral girdle. The head may be isolated, and even (rarely) removed from the attack site.

The category 'natural' (Table 1) included any natural disease process. Starvation and predation are also recognised as naturally wildlife processes, but in this study were frequent enough to justify independent consideration. An outstanding negative result of this study, under the category of natural disease processes, was the virtual absence of any macroscopic evidence of endoparasitism in the Otago blue penguin population over the period of the collection.

Of the 11 birds classed as dying of natural causes (Table 1), one fledged juvenile had a congenitally deformed bill (shortening and crossover of the mandibles), which would have made feeding difficult for the independent bird. Of the other ten, seven died with respiratory mycosis, two of acute non-specific respiratory infection, and one of possible encephalitis. Mycotic respiratory tract disease, probably *Aspergillum* sp., (J. M. Gill, pers. comm.) was found in seven birds (3.3%), and took two forms. Chronic mycosis (six cases) presented either as a tumorous, encapsulated, fungal granuloma, or as a caseous lining of one or more air sacs, usually with active mould on the free surface. Granulomata varied from pea sized in one air sac up to one tumour of 80 × 70 mm, which, at 178.5 g, represented 16% of body weight, causing mechanical pressure on the heart and lungs. Acute mycosis (one case) presented as an exudative tracheo-bronchitis, causing acute inflammatory secretion obstructing the air passages.

I found that the physical signs of drowning (wet lungs, free bloody fluid in the air sacs) are not reliably diagnostic, so drowning may be a difficult cause of death to establish particularly in the absence of recovery information.

'Vandalism' describes malignant human interference, such as gunshot or arrow wounds. (1) Three penguins died, over two weeks, from 0.22 calibre gun shot wounds. (2) There were two apparent episodes of crossbow killings, the first causing seven deaths. A crossbow attack was easily identified from the wound made by the bolt penetrating through the thorax, leaving a pencil-sized diameter track, plus the major lateral crush injury inflicted when the perpetrator stood on the injured bird to withdraw the bolt. A second episode was identified from circumstantial evidence, but the birds were too putrefied to examine and are not included among the findings here.

DISCUSSION

I could not determine whether birds diagnosed as dying of starvation died with or of the malnutrition. This category may obscure natural disease, such as avian malaria as suggested by Graczyk et al. (1995) and Fiennes (1967), or viral infections (Morgan et al. 1985). I found that chronic aspergillosis was always associated with malnutrition. Graczyck & Cockrem (1995) found a 100% prevalence of aspergillosis seropositivity among New Zealand blue penguins. It might be expected that cases of malnutrition would increase in the post breeding season, when some inexperienced juveniles starve and adults inadequately prepared for the moult are especially at risk (D'Amore & Jessop 1995). However, only 30 of the 34 birds in this category (Table 1) were documented sufficiently well to show season of death, insufficient numbers to confirm this expectation. The association between malnutrition and gastro-intestinal bleeding was firm, and also noted by Crocket & Kearns (1975). I could not deduce how often intestinal haemorrhage was the terminal event but it was not associated either with nematode infestation or visible gastric ulceration, in contrast to the findings of Obendorf & McColl (1980).

The frequency of road and rail traffic injury is related to the proximity of Oamaru blue penguin colonies to the main railway and the roads serving the harbour and the town. The monitored colonies are protected by fencing, but it is impossible to fence in the general population of birds using town buildings and industrial complexes as breeding sites, which are thereby exposed to the town (road) traffic. These hazards are reflected in the higher incidence of 20% (28/137) Oamaru birds killed on the road or railway, compared with 14% (30/213) of the whole sample (Table 1).

The figure given for 'shark' predation can only present that proportion of the sample I examined which died as a result of shark attack; it is not an estimate of the incidence of shark predation. Such reservations need not apply either to dog or mustelid attack from a defined area such as Oamaru, well covered by observers.

Ferrets (*Mustela furo*) are the most significant blue penguin mustelid predator in North Otago. The grassland habitat favoured by ferrets (Lavers &

Clapperton 1990) predominates in the district and is characteristic of blue penguin habitat. Trapping to remove ferrets from around penguin colonies has eliminated mustelid predation on the nesting birds and their eggs. Stoats (*Mustela erminea*) are extremely rare in these areas, and although a solitary stoat was sighted some years ago, none has been trapped in the Oamaru penguin colonies. Ratz et al. (2000) did not describe the physical signs associated with mustelid attack on yellow eyed penguins, but the dorsal upper cervical attack pattern (see above) is quite characteristic (C. N. Challies unpubl.). Dead penguins showing such signs are found at times and sites where ferrets are turning up in traps (C. N. Challies, unpubl.; A. G. Hocken, pers. obs.). Ferrets trapped less than 12 hours after yellow eyed penguins were found, killed and eaten out in the characteristic pattern, had remains of penguins among their stomach contents both in North Otago (A. G. Hocken, unpubl. 1998) and on Otago Peninsula (Moller et al. 1995). Carcases showing the distinctive pattern of eating out of the carcass by feral cats (*Felis catus*), described by Fitzgerald (1990), are extremely rare among the Oamaru penguin populations. Hence, the evidence for ferret predation of blue penguins is circumstantial but compelling.

The incidence of aspergillosis in this study was comparable with that described by Obendorf & McColl (1980). By contrast, Harrigan (1992) did not find respiratory mycosis in 41 "collected" birds and 26 picked up dead from Philip Island in 1983 and 1986, possibly a reflection of the smaller sample. Morbidity due to mycosis in captive populations suggests that it is a contagious disease (Fiennes 1967; Flach et al. 1990). However, *Aspergillus* is a ubiquitous, sporulating organism and, bearing in mind the findings of Graczyk & Cockrem (1995), the evidence could as well be interpreted as an immunocompetence break down in stressed birds, e.g., in captivity (Flach et al. 1990) and/or under transportation (Fiennes 1967). In a wild population, a similar degree of stress might be imposed by malnutrition, a common feature of this study (Table1).

Acute non specific inflammation of the respiratory tract could not be proven macroscopically, and would not lend itself to histological assessment in frozen material. The signs of acute infection could be confused with those of drowning, for either condition will present wet lungs and blood stained free fluid in the air sacs. Fluid may also be produced during the later stage of post mortem changes (autolysis). It was, therefore, surprisingly difficult to formulate criteria for the diagnosis of drowning, a problem also noted by Darby in yellow-eyed penguins (J. T. Darby, pers. comm.). This problem illustrates the necessity of adequate recording of the details of the recovery. It is impossible to determine how many birds drowned, for, as with 'shark' attack, the number lost at sea is unknown. Of the ten so classified in this study, three were trapped in industrial drains, six picked up on beaches, and the recovery details of one were incomplete. Presumably most drowned birds recovered on beaches were trapped in nets and discarded at sea.

A principal finding of this study is that there was no macroscopic evidence of gastric, hepatic or renal endoparasitism. Obendorf & McColl (1980), found pathology in 77% of 48 birds opportunistically collected from Victoria, describing gastric ulceration, parasitic hepatitis and cholangitis, as well as respiratory aspergillosis. Harrigan (1992) reported a high incidence of hepatic trematode parasitism, which caused gross liver enlargement in birds from around Phillip Island, and suggested some temporal variation of infestation within that population. By contrast, my findings, in the light of the Australian studies, (Obendorf & McColl 1980; Harrigan 1992), imply regional rather than temporal variation in endoparasitism in blue penguins. The Otago samples were collected over three and a half years, which should have picked up any temporal variation here. Crocket & Kearns (1975) did not detect any hepatic infestation in their New Zealand birds.

It is difficult to understand the significance attributed by Obendorf & McColl (1980) to "enlarged gall bladders" in starving birds. The gall bladder of a healthy fasting blue penguin is typically full. It is sausage-shaped, and up to 60 mm long, lying superficially in the ventral abdomen. Its distension is not morbid and could not of itself be related to hepatitis or cholangitis, which would cause inflammation and thickening of the gall bladder. The same picture was described in fasting but not starving yellow-eyed penguins by Gill & Darby (1993). Conversely, in birds killed with full stomachs, the gall bladder is empty. The variation in gall bladder size is physiological; the gall bladder functions as a storage organ, and when the digestive tract is inactive it makes no call upon bile reserves.

Crocket & Kearns (1975) examined beach-wrecked birds in Northland in the late winter of 1973, and another in late autumn 1974, and found heavy parasitism by renal trematodes. They described small (2.0 mm) parasites, inducing destruction with little inflammatory reaction. My

examination routinely included saggital section and inspection of kidneys and liver and I doubt that such changes could have been overlooked. Harrigan (1992) described renal coccidiosis in "many" of 26 emaciated birds picked up dead. In two cases I found macroscopic lesions, apparently similar, but which histology showed to be collections of intraluminal trematode eggs, of no morbid significance (J. M. Gill, pers. comm.).

I considered it appropriate to record trapped birds (killed or irrecoverably injured in predator traps) as a separate category, that the risk to penguins of predator control operations might be ascertained. However, of the complete collection of 213 birds, only two were killed in Fenn traps set in wooden tunnels and another was the only bird from the Oamaru group (0.7% of 147) which was put down after being irrecoverably injured in a leg-hold trap. Since that time, trapping operations on the Oamaru sites have used only Fenn traps in anchored "Philproof"™ plastic tunnels. The low incidence of accidental deaths of penguins, compared with the large number of mustelid predators killed, suggest that active predator control around penguin colonies is on the whole advantageous. There is already a public awareness programme in Oamaru emphasising education, regulation and the need for dog control around the penguin colony and nearby beaches.

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