Oral necrobacillosis (‘lumpy jaw’) in a free-ranging population of eastern grey kangaroos (Macropus giganteus) in Victoria

D. Borland, G. Coulson and I. Beveridge

Abstract. A high prevalence (54%) of oral necrobacillosis, commonly referred to as ‘lumpy jaw’ based primarily on the presence of cranial osteological lesions, is reported from a free-living population of Macropus giganteus in Victoria. Lesions were found primarily in the maxillary bone and the mandible, in association with the rostral region of the dental arcade. Autopsy data provided additional evidence that the osteological lesions observed were due to oral necrobacillosis. Lesions were more common in older kangaroos. The study was carried out during a drought with limited pasture availability and heavy faecal contamination of pasture, conditions that may have contributed to the high prevalence of the disease.

Additional keywords: disease, mortality, nutrition, parasites.

Received 1 October 2010, accepted 28 April 2011, published online 14 October 2011

Introduction

Oral necrobacillosis, colloquially referred to as ‘lumpy jaw’, is a well recognised disease of captive macropodids (Finnie 1976; Arundel et al. 1977; Samuel 1978, 1983; Miller et al. 1980; Burton 1981; Butler 1981; Vogelnest and Portas 2008). The disease commences as a periodontitis with invasion of the mucosa by saprophytic bacteria, and infection frequently extends into adjacent bones, resulting in osteomyelitis. Numerous species of predominantly gram-negative anaerobic bacteria have been isolated from lesions, with most members of the normal oral bacterial flora being present (Samuel 1978), but the disease has been reproduced experimentally only by the injection of Fusobacterium necrophorum (formerly Sphaerophorus necrophorus) into the gingival mucosa (Beveridge 1934). Samuel (1978) also established that F. necrophorum was absent in the oral bacterial flora of normal macropodids but was highly prevalent in macropodids with oral necrobacillosis, and that inoculation of other species of bacteria in the absence of F. necrophorum failed to produce the characteristic lesions. Thus it appears that the primary pathogen involved in the disease is F. necrophorum.

In spite of the extensive documentation of this disease in captive macropodids, there are relatively few well substantiated records of its occurrence in free-living animals. Tomlinson and Gooding (1954) reported ‘lumpy jaw’ in red kangaroos (Macropus rufus) in the Murchison region of Western Australia. However, their report is not detailed and the disease they reported also involved pelvic limbs, casting some doubt on the possibility that the syndrome they reported was oral necrobacillosis.

Individual cases of oral necrobacillosis in wild animals have been noted by Arundel et al. (1977) in the swamp wallaby (Wallabia bicolor) and the tammar wallaby (M. eugenii), although no substantiation was provided. Lesions in two individual wild rock wallabies (Petrogale assimilis and Petrogale sp.) were reported by Miles and Grigson (1990). Oral necrobacillosis has also been reported from the extinct kangaroo species M. titan, suggesting that periodontal disease in macropodids may not be a recent phenomenon (Horton and Samuel 1978).

Thus, while well known in captive macropodids, there is little substantiated evidence that the disease is widespread in wild populations. During a study of the causes of mortality in a free-ranging population of eastern grey kangaroos (M. giganteus) at Serendip Sanctuary, south-west of Melbourne, conducted during 2006, a significant proportion of skulls examined exhibited signs of osteomyelitis presumed to have arisen from oral necrobacillosis. Although there are no other diseases known to cause similar lesions in kangaroos, the presumed diagnosis was subsequently confirmed on the basis of autopsies of additional animals. We here present evidence for the common occurrence of oral necrobacillosis or ‘lumpy jaw’ in at least one population of free-ranging kangaroos.
Materials and methods

Site description
Serendip Wildlife Sanctuary is located 60 km west-south-west of Melbourne (38°00′S, 144°24′E). The sanctuary covers 250 ha, of which 131 ha are made up of unimproved pasture: *Lolium rigidium* (annual ryegrass), *Trifolium subterraneum* (subterranean clover), *Avena fatua* (wild oats). A further 66 ha is open woodland while the remaining areas have been fenced off for revegetation. Dams forming a wetlands area are accessible to kangaroos, providing a reliable water source. At the time of this study there were estimated to be ~600 kangaroos on the site giving an approximate density of 3 kangaroos ha⁻¹. The sanctuary is surrounded by farmland and is enclosed by a low fence that provides no significant barrier to kangaroo movement. Average annual rainfall at the site between 2001 and 2005 was 480 mm. Rainfall at the site during 2006, when this study was conducted, was only 283 mm.

Skull collection and mortality records
Skulls were collected from all *M. giganteus* carcasses found in the study area during 2006 and the date or month of death was noted wherever possible. A molar progression formula (Kirkpatrick 1964) was used to determine the age of the animals at the time of death. Skulls from animals of unknown sex were compared with skulls of known sex and with a similar molar index to determine the sex (Coulson 1989), male skulls being significantly larger than those of females.

Autopsies were performed on all fresh carcasses obtained during 2006. The carcass was weighed, sex recorded and the pes, tail and total body length measured. Standard autopsy methods were used and, as all animals were expected to harbour significant numbers of internal parasites, total worm counts were performed as described by Arundel et al. (1979). Intensity data could not be collected for cestodes in the small intestine, which fragmented after the death of the host and for *Strongyloides* sp., which is an intramucosal parasite.

Skull lesions
Once the skulls obtained were cleaned, they were investigated for evidence of lesions and their locations recorded. In the case of the maxillary bone, the sites used were ‘caudal maxilla’ (that is, in the posterior part of the dental arcade) and ‘rostral maxilla’ (at the anterior extremity of the dental arcade). Skulls were classed into four categories according to the type and severity of the bone lesion observed. Skulls sometimes displayed lesions in several categories. However, each skull was placed into only one category, corresponding to the highest ‘ranked’ lesion observed. Lesions were ranked as either normal, exhibiting only minor osteolytic changes (Figs 3–5) or exhibiting major osteolytic (Figs 3–5) or osteoproliferative (Figs 5, 6) changes. In skulls that exhibited minor osteolytic lesions, there were significant bone defects but the lesions did not visibly alter the physical dimensions of the bone. In skulls that exhibited major osteolytic lesions, a significant bone defect was observed. In skulls that exhibited osteoproliferative lesions, the dimensions of the bone increased visibly.

The locations of lesions were also documented. Multiple locations were documented if lesions on a skull were observed on more than one bone. In instances where lesions occurred on different areas of the same bone or on multiple bones of the same type (e.g. right and left mandible), the location was recorded only once.

Pasture availability
Pasture availability was determined using samples taken from six random sites in each of two paddocks grazed by kangaroos (12 samples in total). All pasture in 50-cm² quadrants was clipped and collected. Photographs of the pasture were also taken at each sampling to record the extent of faecal contamination. The samples were rinsed to remove soil. The total dry weight (DW) of samples from each paddock was obtained by drying pasture samples in an oven at 60°C. The dried samples were then weighed. The estimated dry biomass was then calculated and is expressed as tonnes per hectare (t ha⁻¹).

Statistical analyses
The seasonal occurrence of bone lesions in skulls was examined using a Chi-square test. Fisher’s exact test was used to compare the proportion of kangaroos less than three years of age in two categories: kangaroos with bony lesions on their skull and kangaroos with no bony lesions on their skull. The Mann–Whitney test was used to compare the median ages of the kangaroos with skulls in the ‘proliferation’ category, with the kangaroos that had skulls in the ‘normal’ category.

Results
Mortalities
From February to October 2006, all mortalities were recorded according to the month in which they occurred, including carcasses that were not autopsied. From March to October, 66 mortalities were recorded. Assuming no association of month and number of mortalities, the expected number of mortalities per month was eight. The observed number per month deviated from this expected number more than could be expected by chance ($\chi^2 = 15.5$, $P = 0.03$), with a greater than expected mortality in June and a lower than expected mortality in August. However, if seasonal mortalities (March–May, June–August, September–November) were compared, there were no significant differences ($\chi^2 = 0.026$, $P = 0.87$).

A total of 90 skulls was examined, comprising 66 for which the month of death was known and 24 for which this could not be ascertained. Eighteen of the skulls were obtained from animals that died as a result of vehicular accidents. Autopsies were carried out on 31 fresh carcasses. For these, no cause of death could be established for five animals, with other causes being determined as: vehicular accidents 14, bacterial pneumonia 1, oral necrobacillosis 2, parasitism 2, musculoskeletal disease 4, caval aneurysm 1, urethral obstruction 1, and haemochromatosis 1. Of these animals, 26% also exhibited signs of malnutrition, indicated by muscle wasting and the presence of excessive quantities of pale, straw-coloured fluid in the abdominal and thoracic cavities.

As expected, large numbers of internal parasites, principally gastric and large intestinal nematodes were encountered. The
most abundant species in the stomach was *Rugopharynx macropodis* in the stomach with a mean of 51,600 nematodes (range 100–284,000). In the large intestine, numbers of *Macropoxyuris brevigularis* and *Ma. longigularis*, oxyurid nematodes, ranged from 160 to 71,400 (mean 8,540). The numbers of parasites for each genus fell within the ranges established for healthy, free-ranging *M. giganteus* established by Beveridge and Arundel (1979). In only two individuals with extremely high numbers of parasites was this considered to have contributed to the death of the kangaroo.

Table 1. Number and proportion of *Macropus giganteus* skulls collected from Serendip Sanctuary that displayed bone lesions in the skull

<table>
<thead>
<tr>
<th>Category of bone lesions</th>
<th>No. of skulls (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>41 (46%)</td>
</tr>
<tr>
<td>Mild osteolytic change</td>
<td>11 (12%)</td>
</tr>
<tr>
<td>Major osteolytic change</td>
<td>21 (24%)</td>
</tr>
<tr>
<td>Osteoproliferative change</td>
<td>16 (18%)</td>
</tr>
</tbody>
</table>

Fig. 1. Lesions of osteomyelitis in skulls of *Macropus giganteus*. Mild osteolysis of maxillary region above dental arcade.

Fig. 2. Mild osteolysis of mandible associated with anterior region of dental arcade.

Fig. 3. Severe osteolysis of maxilla above dental arcade.

Fig. 4. Severe osteolysis of maxilla above dental arcade, with tooth loss and osteolysis of premaxilla.

Fig. 5. Severe osteolysis of the surface and osteoproliferation of the mandible, resulting in bone deformation.

Fig. 6. Severe osteoporosis of surface of mandible together with osteolysis and osteoproliferation, causing bone deformation.
Lesions in skulls

Of 89 relatively intact skulls examined, 54% exhibited observable osteological changes (Table 1). In skulls that exhibited mild osteolytic changes (Figs 1, 2), lesions consisted of brown discoloration and increased porosity or superficial roughening of the affected bone. In skulls that exhibited major osteolytic changes (Figs 3, 4), lesions showed varying degrees of osteolysis, often associated with tooth loss or the destruction of periodontal bone. In skulls that exhibited osteoproliferative changes (Figs 5, 6), there was an increase in bone mass due to reactive bone deposition, especially on the lateral aspect of one or both mandibles. These changes were associated with osteolytic cavities (Fig. 5) and often with a coarsely porous bone surface (Fig. 6). They ranged from minor local thickening of bone contours through to marked thickening and distortion of the mandibular contours.

Location of lesions

The location of lesions on 48 affected skulls was documented (Fig. 7). However, not all skulls were complete; some had extensive fractures. Lesions observed were predominantly located on bones in close association with the teeth: the maxilla, mandible and premaxilla were the bones most commonly affected. Lesions observed in the maxillary bone occurred primarily at the rostral extremity of the dental arcade in the caudal maxilla. Likewise, lesions on the mandible occurred predominantly in the area ventral to the rostral half of the dental arcade.

Age of kangaroos

The sex and age at time-of-death of *M. giganteus* were investigated for all mortalities at Serendip Sanctuary with bone lesions on their skulls (Fig. 8). In total, 47 skulls with lesions were examined. There was no significant difference in median age at time-of-death, sex proportions or proportion of skulls from kangaroos under three years of age at time-of-death, between skulls with bone lesions and skulls without bone lesions. When different categories of bone lesions were considered, the median age of the 16 kangaroos with skulls in the osteoproliferative category was significantly higher than the median age for the 41 kangaroos with normal skulls. Differences in the osteolytic category were not significant (Table 2).

Pasture availability

Visual assessment of the pasture available was that it was extremely short except in October during the spring flush. Visual assessment was confirmed by data on dry matter ha\(^{-1}\) (Fig. 9), which was below 1 t ha\(^{-1}\) between February and May, 2006, increased to >1 t ha\(^{-1}\) between June and September, reached a peak in October and then declined to ~1–2 t ha\(^{-1}\) in November–January. The pastures were heavily contaminated with faeces throughout the year.

Discussion

Bone lesions were seen in a significant proportion of skulls obtained from *M. giganteus* at Serendip Sanctuary. The lesions ranged from mild porosity and brown discoloration to large proliferative bone masses. The severe bone lesions were considered to be manifestations of oral necrobacillosis; the mild lesions probably represented cases with predominantly soft tissue involvement before significant osteological involvement, although it was not possible to confirm this supposition from the autopsy material available. Cases of severe active necrobacillosis were also identified in two of the 31 (6%) animals autopsied.
during the study, providing evidence in support of the hypothesis that the skeletal lesions observed were the result of oral necrobacillosis. However, the examination of bone lesions only almost certainly underestimates the prevalence of oral necrobacillosis in the population as soft tissue lesions can be present in the absence of lesions in bone.

The lesions found on skulls from Serendip Sanctuary were almost entirely observed on bones in direct contact with dental tissues. In many cases in the current study, alveolar bone had been destroyed and teeth had been shed or there was destruction of tooth roots. Proliferative lesions observed were found predominantly on the mandibles, particularly in the area ventral to the rostral half of the dental arcade. It was observed generally that bone lesions rarely involved the caudal, erupting molars and that lesions tended to be associated with the more rostral teeth in the dental arcade. These data therefore suggest that molar eruption may not play an important role as a route of infection for lumpy jaw, but rather that loss of premolars, deciduous premolars and leading molars provide a route of infection for the disease. This observation contradicts the hypothesis of Finnie (1976), who specifically identified erupting molars as a major route of infection, and of Arundel et al. (1977), who identified ‘tooth eruption’ as a major route of infection for lumpy jaw. Miller and Beighton (1979) suggested that food packing around postfunctional molar teeth may predispose the animal to opportunistic infection by opportunistic oral microorganisms, a hypothesis consistent with observations reported here.

Some animals in the population under study died from the effects of lumpy jaw. On post-mortem examination, these animals were severely emaciated and exhibited large bony

---

Table 2. Average and median ages (years) of kangaroos with either no skull lesions, those with skull lesions of any type and those with osteoproliferative lesions

<table>
<thead>
<tr>
<th></th>
<th>Mean</th>
<th>Median</th>
</tr>
</thead>
<tbody>
<tr>
<td>Without lesions</td>
<td>3.4</td>
<td>2.5</td>
</tr>
<tr>
<td>With lesions</td>
<td>4.4</td>
<td>3.2</td>
</tr>
<tr>
<td>With osteoproliferative lesions</td>
<td>6.0^</td>
<td>5.6^</td>
</tr>
</tbody>
</table>

^Indicates significant difference from normal.
swellings on the mandibles or severe destruction of periodontal structures. They were thought to have been unable to feed, causing them to starve to death. It is also possible that the mortalities may have been caused by septicemia with the spread of bacteria to other tissues (Arundel et al. 1977). However, in this study, lumpy jaw was investigated predominantly on the basis of bone lesions observed on skulls, precluding observations on the cause of death in most cases.

When categorised according to their most severe lesion, all three classes of skulls were seen in similar proportions (Table 1). Kangaroos with proliferative lesions had a significantly higher median age at time-of-death than did kangaroos with no bony lesions on their skulls (Table 2). This suggests that proliferative lumpy jaw lesions are likely to be present in the later stages of the disease, while osteolytic lesions are likely to predominate in the earlier stages. If it is assumed that the different categories of lesions represent different stages of the disease, affected animals are likely to have been affected with the disease for a lengthy period.

The presence of bone lesions on skulls collected in years before this study (data not included), as well as the higher age of kangaroos with proliferative bony lesions, suggests that the disease has been present in the population for some time rather than having occurred as a recent epidemic. This conclusion is significant, as lumpy jaw has been observed previously in wild populations predominantly as epidemics (Arundel et al. 1977). Although no additional wild populations with a low prevalence of lumpy jaw were included in this study, Miller and Beighton (1979), in describing dental abnormalities in M. giganteus, examined 81 skulls from Yan Yean in Victoria and 38 from Grey Mare in Queensland, without finding any evidence of lumpy jaw.

The high prevalence of lumpy jaw lesions on skulls in this study was notable as previous substantiated records from wild animals have involved individuals rather than populations. It is commonly believed that harsh feed, which is more common in dry conditions, stress and faecal contamination are all predisposing factors in the development of lumpy jaw infection in captive animals (Vogelnest and Portas 2008). The current study was conducted during a severe drought when feed was apparently limited, the feed available was predominantly dry and the pasture was heavily contaminated with faecal material (Borland 2006). However, it is not possible from the evidence available to determine which, if any, of these factors contributed to the high prevalence of oral necrobacillosis observed in this study.

The database on pasture biomass is crude and does not take into account species composition and palatability of the component species for kangaroos. In addition, while extensive data exist for calculating the nutritive value of available pasture for ruminants, the extrapolation of such data to macropods is currently uncertain (Grigg 2002). However, Fletcher (2006) showed that the population density of eastern grey kangaroos in temperate grasslands in the Australian Capital Territory would remain in long-term equilibrium if the biomass (dry weight) of green grass was 143 000 t ha\(^{-1}\) per head. The biomass of all pasture (regardless of species) available to the estimated 600 kangaroos on Serendip Sanctuary at the time was two orders of magnitude less than this equilibrium value in all but one month of the year, suggesting that the population was under considerable nutritional stress. The sparse pasture was also generally dry due to the prevailing drought conditions and there was invariably heavy faecal contamination of pastures. The contribution of these factors to the occurrence of the disease in free-living populations of kangaroos clearly warrants additional investigation.

The current study was able to identify a high density of kangaroos, limited feed availability and heavy faecal contamination of pasture associated with the high prevalence of oral necrobacillosis seen in the kangaroo population. It was, however, not possible to determine any specific causal relationships.

Acknowledgements

We thank M. Helman and M. Smith, Parks Victoria, for invaluable support in conducting this study, J. Charles for advice on the skull lesions, and R. Cooke for help with autopsies. The study was conducted under a research permit (10004787) from the department of Sustainability and Environment.

References


Beveridge, W. I. B. (1934). A study of twelve strains of Bacillus necrophorus, with observations on the oxygen tolerance of the organism. The Journal of Pathology and Bacteriology 38, 467–491. doi:10.1002/path.1700380314


