Mortality events of large macropods in Australia

Fact sheet

Introductory statement

Mortality events of large macropods in Australia have been recorded sporadically since first official reports in the 1950s. The aetiology of the events varies, however there are often similar circumstances, such as extreme climatic conditions, high macropod population density and the presence of a parasitic or infectious agent. Significant events have been associated with heavy rainfall or flooding in inland areas of mainland Australia. Numbers affected are difficult to quantify but may be in the thousands or tens of thousands. When events occur in geographically isolated areas, investigation can be challenging, and in some of these events, a definitive diagnosis may not be reached.

This fact sheet provides a summary of available information, with a focus primarily on mass mortalities of large species of macropod, where aetiology is as yet unknown or unconfirmed. More work is required to understand the ecology and factors driving macropod mass mortalities, including those occurring in remote and inland areas. Wildlife Health Australian considers this to be a DRAFT Fact Sheet, with updates planned following significant new knowledge or developments in understanding. This fact sheet should not be relied upon as a sole source of information on the current situation.

Aetiology and host species affected

The cause of macropod mortalities may not always be known, and in many cases, may be difficult to determine. However, a variety of different aetiologies have been recognised, or hypothesised: the most common of these are associated with nutritional stress or infection (see Table 1 and Epidemiology). There may be multiple factors (many with synergistic effects) associated with occurrence of mortalities. There is a need for greater understanding of the inter-relationship between mortality events, normal population regulation and disease expression.

Red (Macropus rufus), eastern grey (M. giganteus), western grey kangaroo (M. fuliginosus), and common wallaroo (M. robustus) are often involved. This fact sheet does not consider causes of mortality in smaller macropod species (e.g. wallabies).
Table 1: Examples of mortality events of large macropods in Australia from 1970 to 2016

<table>
<thead>
<tr>
<th>Year</th>
<th>Species</th>
<th>Location</th>
<th>No. dead (estimates)</th>
<th>Duration of event</th>
<th>Cause (confirmed or suspected)</th>
<th>Clinical and or pathological signs *</th>
<th>Notes</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>2015</td>
<td>EGK</td>
<td>ACT, southern NSW</td>
<td>July - Sept</td>
<td>Malnutrition</td>
<td></td>
<td></td>
<td></td>
<td>Grillo et al. (2015)</td>
</tr>
<tr>
<td>2014</td>
<td>EGK</td>
<td>Wacol Qld</td>
<td>30</td>
<td>Days to weeks</td>
<td>Malnutrition, fascioliasis</td>
<td>HT, OT</td>
<td></td>
<td>Grillo et al. (2014a)</td>
</tr>
<tr>
<td>2014</td>
<td>EGK</td>
<td>Wagga Wagga NSW</td>
<td>85</td>
<td>6 weeks</td>
<td>Plant toxins (saponins)</td>
<td>BL, HT, OT</td>
<td></td>
<td>Grillo et al. (2014b)</td>
</tr>
<tr>
<td>2014</td>
<td>EGK</td>
<td>Lake Macquarie, NSW</td>
<td>300+</td>
<td></td>
<td>Babesia sp.</td>
<td>NS, SC, OT</td>
<td></td>
<td>Arthur (2014)</td>
</tr>
<tr>
<td>2011</td>
<td>EGK</td>
<td>ACT</td>
<td>&lt;100</td>
<td></td>
<td>Endoparasitism (Globocephaloides sp.)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2010</td>
<td>CW, RK, EGK</td>
<td>Tibooburra NSW SW Qld (around Quilpie and Cunnamulla)</td>
<td>400-1200</td>
<td>6 weeks</td>
<td>Not identified</td>
<td>BL, SC, JT, AT, OT</td>
<td>Heavy rain and increased insect activity.</td>
<td>Gordon et al. (2010); Grillo and Post (2010); Curran (2011); Grillo et al. (2011)</td>
</tr>
<tr>
<td>2010</td>
<td>EGK</td>
<td>Vic</td>
<td>&lt;100</td>
<td></td>
<td>Endoparasitism (Globocephaloides sp.)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2009</td>
<td>RK</td>
<td>Kununurra WA</td>
<td>Not reported</td>
<td></td>
<td>Not identified</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2009</td>
<td>EGK</td>
<td>Vic</td>
<td>&lt;100</td>
<td></td>
<td>Endoparasitism (Globocephaloides sp.)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2006</td>
<td>RK</td>
<td>Alice Springs NT</td>
<td>Not reported</td>
<td></td>
<td>Not identified</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Year</td>
<td>Species</td>
<td>Location</td>
<td>No. dead (estimates)</td>
<td>Duration of event</td>
<td>Cause (confirmed or suspected)</td>
<td>Clinical and or pathological signs *</td>
<td>Notes</td>
<td>Reference</td>
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<tr>
<td>2006</td>
<td>EGK</td>
<td>NSW</td>
<td>&lt;100</td>
<td></td>
<td>Endoparasitism (Globocephaloides sp.)</td>
<td></td>
<td>Presumptive starvation; drought conditions.</td>
<td></td>
</tr>
<tr>
<td>2005</td>
<td>RK</td>
<td>Meekatharra WA</td>
<td>3000</td>
<td></td>
<td>Presumptive starvation</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2005</td>
<td>EGK</td>
<td>Vic</td>
<td>&lt;100</td>
<td></td>
<td>Endoparasitism (Globocephaloides sp.)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Oct 1998</td>
<td>RK, WGS, CW</td>
<td>Marree SA – NW NSW – Hungerford Qld</td>
<td>&gt;1,000 (anecdotally 50,000+)</td>
<td>Several days</td>
<td>Not identified (no samples).</td>
<td>Stiffness, lameness, loss of condition.</td>
<td>Followed heavy rains and increased insect activity.</td>
<td>Curran et al. (1999)</td>
</tr>
<tr>
<td>1994-6</td>
<td>WGK, RK, EGK, CW</td>
<td>NSW, Vic, SA, WA, Qld</td>
<td>Not reported</td>
<td>Months</td>
<td>Wallali-Warrego blindness.</td>
<td>Blindness or other severe ocular pathology.</td>
<td>Notable insect activity.</td>
<td>Hooper et al. (1999); Reddacliff et al. (1999); Curran et al. (1997)</td>
</tr>
<tr>
<td>May 1990</td>
<td>CW, RK</td>
<td>Central western Qld</td>
<td>200+</td>
<td>Weeks</td>
<td>Not identified.</td>
<td>Weakness, ataxia; meningoencephalitis and pneumonitis.</td>
<td>Followed severe rains, flooding and increase in black fly numbers.</td>
<td>Clancy et al. (1990); Speare R et al. (1991);</td>
</tr>
<tr>
<td>1983</td>
<td>NR</td>
<td>Western Qld</td>
<td>Not reported</td>
<td>Not identified</td>
<td>Not reported</td>
<td>Following extended drought.</td>
<td></td>
<td>Speare R. et al. (1989)</td>
</tr>
<tr>
<td>1970</td>
<td>EGK</td>
<td>Vic</td>
<td>Not reported</td>
<td></td>
<td>Endoparasitism (Coccidiosis)</td>
<td>Haemorrhagic enteritis and hypoproteinaemia</td>
<td>Followed a flooding event.</td>
<td>Barker et al. (1972)</td>
</tr>
</tbody>
</table>

EGK Eastern grey kangaroo, RK Red kangaroo, CW Common wallaroo, WGK Western grey kangaroo, NR not recorded or presumed mixed species; BL Blindness or other severe ocular pathology; SC Subcutaneous haemorrhage / bruising / oedema; JT Joint pathology (arthritis, oedema, haemorrhage); AT ataxia, stiff movements, recumbent; HT hepatic changes; NS Neurological signs; OT Other signs.
Occurrences in Australia

Mortalities may occur anywhere where there are large populations of susceptible macropod species, but several notable events have occurred in inland areas of NSW and Qld, and often in areas subject to flooding after heavy rain [see Table 1] (Lundie-Jenkins 2000; Gordon et al. 2010). There are earlier reports of large scale die-offs following major flooding in western Qld and western NSW (Speare R. et al. 1989). Mortalities associated with malnutrition have occurred during winter in temperate areas of south eastern Australia (Grillo et al. 2015; Roth 2015).

Mass mortalities of macropods have not been reported outside Australia.

Epidemiology

The epidemiology of macropod mortalities varies according to the aetiology. In most cases, multiple causal factors combine to result in a mass mortality event. Causes of previous macropod mass mortalities include:

- malnutrition (secondary to drought or seasonal scarcity of pasture, and may be age-dependent)
- heavy endoparasitic burdens (*Globocephaloides* spp., coccidia spp., fascioliasis)
- exposure to toxins, including plant toxins
- orbivirus infection (e.g. Wallal, Warrego or other serogroups)
- macropod herpes virus
- other possible arbovirus infections (unidentified)
- protozoal infections (e.g. *Babesia* sp.)
- other unknown aetiologies.

Influencing factors may be extreme climatic conditions (heat, cold, drought, flooding, which place more physiological stress on already compromised individuals); increased vector abundance; increased macropod population densities; poor food availability and other factors which favour persistence of endoparasites or grazing of toxic plants.

Mortalities caused by *Globocephaloides* spp. generally occur in juvenile animals during winter, influenced by high population density and depleted food supplies (Arundel et al. 1990). Deaths due to intestinal coccidiosis may occur after flooding, which may drive increased population densities and food shortages; damp conditions and the use of feed supplementation onto the ground may be other factors influencing outbreaks (Barker et al. 1972).

Winter mortality events appear to occur as a result of combined effects of endoparasitism, reduced food resources and thermal stress (cold) in sub-adult (i.e. growing, inexperienced, recently weaned) eastern grey kangaroos. These events mortalities most often occur when population density is considered high (Grillo et al. 2015; Roth 2015).

Factors driving orbivirus outbreaks are not well understood but probably include increase in vector numbers following rain. For more information see WHA fact sheet ‘Epidemic blindness in kangaroos’.

Drivers of the large inland NSW and Qld mortality events have not been identified. Some events appear to be associated with heavier than average rainfall and flooding, and concurrent increased insect (possible vectors) populations. Individual events also occur in relatively confined geographical areas. Deaths typically occur over a 1-2 week period (sometimes longer) (Curran 2011). The extent of mortalities is difficult to quantify or even estimate with any accuracy, particularly in remote areas. Anecdotal reports range from hundreds to more than 50,000 animals estimate dead.
**Clinical signs**

Clinical signs will vary dependent on the causal factors.

Animals affected by **endoparasitism** (*Globocephaloides* sp. or coccidiosis) are generally weak (due to severe anaemia and hypoproteinaemia) or found dead.

Animals dying of **starvation** during drought or winter mortality events are thin with depleted fat and muscle stores. Animals appear unwell, with a poor coat condition. They are reluctant to move away from humans and continue to graze when disturbed.

In drought, animals are characteristically clustered around shade trees, in caves or near dried-up water sources.

Macropods infected with **orbiviruses** (Wallal/Warrego viruses) are blind, appear confused and have few other obvious clinical signs.

Macropods suffering from plant toxicity display varying signs, dependent on the toxins involved.

In some **large mortality events associated with rain** (but of unknown aetiology), animals may appear stiff, reluctant to move, with signs of lameness and blindness. Animals may be found dead despite abundant food resources. Some individuals in the 2010 Qld event had floppy ears and swollen joints.

**Diagnosis**

Confirming an aetiological diagnosis for macropod mass mortalities may be challenging, due in part to remote geographical location of many events, the difficulty in obtaining diagnostic specimens and lack of baseline information. Whenever practical, thorough investigation, including post mortem investigation and sampling should be undertaken for macropod mass mortality events (Lundie-Jenkins 2000).

**Clinical pathology**

Clinical pathology changes, if present, will vary according to the aetiology of the event.

Changes due to endoparasitism include anaemia and hypoproteinaemia. *Babesia* organisms may be visible in peripheral blood smears. For most other causes of macropod mass mortality, there are no known consistent clinical pathology changes.

**Pathology**

Gross and histopathological changes, if present, will vary according to the aetiology of the event.

**Starvation** (drought or winter mortality): depleted fat stores, reduced muscle mass, indicative of chronic starvation.

**Endoparasitism**: *Globocephaloides* - no fat stores and large numbers of red worms in the proximal duodenum. Coccidiosis - extensive haemorrhagic enteritis and identifiable coccidial life stages.

**Orbiviruses** (Wallal/Warrego viruses): non-suppurative panuveitis and retinitis, optic neuritis, and (in about 45% of cases) a non-suppurative meningoencephalitis (Durham et al. 1996).
Toxicity: changes will vary dependant on the toxins involved.

Pathology is variable in mortality events of unidentified cause. There is often no consistent clinical or pathological presentation within or between events. Changes reported across events are diverse in nature. Reported changes (which may not be present in each individual or event, include:

- severe polyarthritis of limbs
- subcutaneous haemorrhage
- congestion of bone marrow and body fat
- splenomegaly and pericardial effusion, myocardial necrosis, dermal vasculitis, myositis, anaemia (2010)
- parasitic larval tracts through hindlimb muscles (1998)
- focal necrosis of heart, liver and spleen, intravascular thrombi, mild acute mononuclear meningoencephalitis, choroid hyperplasia, interstitial pneumonitis, and chronic dermatitis consistent with bites from blackfly (*Austrosimulium pestilens*) (1990).

**Laboratory diagnostic procedures and specimens**

A wide range of procedures may be necessary for investigation of mortality events.

Where possible, live affected animals should be examined by an experienced wildlife veterinarian. Video and photographs, and detailed clinical records may assist if this is not possible.

Samples may be collected from live, affected individuals, or at point of euthanasia, including blood for haematology, biochemistry, serology, PCR, virology and toxicological investigation. Full post mortem investigation and sampling should be undertaken on euthanased or freshly dead individuals. Ideally, investigation should be undertaken by trained and experienced field personnel. If necessary, appropriately trained and authorised lay staff may undertake post mortem investigation, under the supervision of a veterinarian. Detailed written protocols will assist this process.

A number of animals, comprising a representation of species, ages and sexes affected, should be sampled from each site. A minimum sample set of ten individuals (more where possible) will allow for more robust interpretation and analysis of results.

Samples collected should include:

- representative samples of all organs - both fixed in formalin and stored fresh (chilled)
- faeces - fresh, held chilled for faecal floatation
- gastrointestinal contents – fresh, chilled and fixed in formalin
- gastrointestinal tract parasites - relaxed in water then into 70-90% ethanol
- gastrointestinal contents
- a range of swabs, collected using aseptic technique, in transport medium for bacteriology, virology and additional investigation
- additional samples, where possible, of brain, lung, and liver - fixed in glutaraldehyde for electron microscopy and frozen in liquid nitrogen for bacteriology, virology and toxicology
- a sterile sample of aqueous humour for toxicology
- specimens of insects (in liquid nitrogen or 70% ethanol), plants and water (in sterilised sealed containers) should be collected at each site, where possible.
Please refer to the document “Response procedure for sporadic epidemic mortalities in macropod populations in Queensland” (Lundie-Jenkins 2000) for full details. Investigations should include a structured epidemiological approach to identification and recording of risk factors. Veterinary epidemiological advice should be sought. Whenever possible, samples should be collected from a number of unaffected individuals as well.

**Prevention, control and treatment**

In most cases of macropod mass mortality, prevention and control methods are generally not feasible. Treatment of individual macropods involved in mortality events is rarely undertaken but will be dependent on the aetiology. Euthanasia may be required for welfare reasons. Prevention and control will depend upon identification of causal factors and the ability for a treatment to be applied. For arboviruses, control of vectors may be of use, but presents logistical difficulties in field situations.

**Surveillance and management**

Wildlife disease surveillance in Australia is coordinated by Wildlife Health Australia. The National Wildlife Health Information System (eWHIS) captures information from a variety of sources including Australian government agencies, zoo and wildlife parks, wildlife carers, universities, industry and members of the public. Coordinators in each of Australia’s States and Territories report monthly on significant wildlife cases identified in their jurisdictions. NOTE: access to information contained within the National Wildlife Health Information System dataset is by application. Please contact admin@wildlifehealthaustralia.com.au.

Kangaroo meat processors closely monitor animal health through observation and carcass inspection to ensure that diseased animals do not enter the meat supply chain. Food safety is taken seriously by state, territory and commonwealth governments in Australia. All export and domestic establishments processing kangaroo carcasses for human consumption must comply with the Australia Standard for the Hygienic Production of Wild Game Meat for Human Consumption. This standard includes a post-mortem assessment of every carcase for abnormalities and evidence of disease to determine whether the meat is fit for human consumption.

A response procedure for investigation of macropod mortalities has been described (Lundie-Jenkins 2000). No active surveillance has been identified.

**Statistics**

Information on some recent mortality events in large macropods is summarised in Table 1. The national wildlife health information system (eWHIS) contains details of mortality events in large macropod species around Australia.

**Research**

Key research questions include:

- What are the proximate and ultimate causes of macropod mortality events?
- Should there be a management action associated with these events, or are they part of the normal ecology of large macropods in Australia?
• What are the infectious agents involved, how are they transmitted and what risk factors are associated?
• What other causal factors contribute to outbreaks?

**Human health implications**

There are no recognised zoonotic risks related to the known infectious agents contributing to large macropod mortalities. Appropriate personal protective equipment and good hygiene is recommended as normal best practice.

**Conclusions**

Mortality events in large macropod species have been reported in many areas of Australia. In some cases, aetiology has been determined, in others, causes remain unknown or unconfirmed. It is likely that multiple causal factors contribute to most mortality events. Ongoing investigation is required to better understand the drivers and proximate causes of mortality events in large macropod species in Australia.

**References and other information**


Acknowledgements

We are extremely grateful to the many people who had input into this fact sheet.

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To provide feedback on this fact sheet

Wildlife Health Australia would be very grateful for any feedback on this fact sheet. Please provide detailed comments or suggestions to [admin@wildlifehealthaustralia.com.au](mailto:admin@wildlifehealthaustralia.com.au). We would also like to hear from you if you have a particular area of expertise and would like to produce a fact sheet (or sheets) for the network (or update current sheets). A small amount of funding is available to facilitate this.

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