STEROIDAL SAPONIN TOXICITY IN EASTERN GREY KANGAROOS (MACROPUS GIGANTEUS): A NOVEL CLINICOPATHOLOGIC PRESENTATION OF HEPATOGENOUS PHOTORESENSITIZATION

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STEROIDAL SAPONIN TOXICITY IN EASTERN GREY KANGAROOS (MACROPODUS GIGANTEUS): A NOVEL CLINICOPATHOLOGIC PRESENTATION OF HEPATOGENOUS PHOTOSENSITIZATION

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ABSTRACT: We describe the clinicopathologic features of a mortality event characterized by blindness and dermatitis affecting eastern grey kangaroos (Macropus giganteus), secondary to hepatogenous photosensitization. Affected animals exhibited photophobic behavior, blindness, ataxia, recumbency, lethargy, ear shaking, and behavior consistent with distress or depression. The photophobia manifested as abnormal shade-seeking during the day, including finding refuge under or in structures used frequently by people. Severely affected kangaroos were jaundiced and had markedly elevated serum bilirubin and gamma glutamyl-transpeptidase concentrations. Blindness in affected animals was attributed to moderate to severe corneal opacity due to corneal edema and inflammation. Skin lesions were typically subtle on gross examination even in cases which had severe necrotizing dermatitis histologically. Histologic lesions in the liver of affected animals included the presence of acicular clefts typical of steroidal saponins. The outbreak was associated with pasture dominated by the invasive grass, Panicum gilvum, which is a recognized source of saponin-induced photosensitization in livestock.

Key words: Australia, blindness, hepatic photosensitization, macropod, marsupial, Panicum, plant poisoning, saponins.

INTRODUCTION

Hepatogenous photosensitization is a well-recognized pathophysiologic process in herbivorous eutherians, especially sheep, whereby hepatopathy induces distant tissue damage mediated through sunlight activation of the photoactive compound, phylloerythrin (Fløyen 1999; Quinn et al. 2014). Phylloerythrin is a derivative of ingested chlorophyll produced by gastrointestinal bacteria (Quin et al. 1935), which, in cases of photosensitization, has failed to be conjugated and safely excreted by the liver. Hepatogenous photosensitization is not etiologically pathognomonic and it can be thought of as primarily reflective of liver dysfunction. Diverse causes of hepatobiliary damage can cause hepatogenous photosensitization, although acute to subacute hepatic insult from toxic plants is the most widely reported etiology.

Despite the diversity of underlying etiologies, hepatogenous photosensitization has a moderately conserved clinical presentation in domestic herbivores (Kellerman et al. 1980; Casteel et al. 1991; Stegelmeier 2002). The most characteristic feature is necrotizing dermatitis in regions of skin exposed to sunlight, which are typically lacking in thick fur or fleece and are mostly dorsoventrally distributed, including the nose, eyelids, ears, muzzle, and tail, although the mammary glands and coronary regions of the hoof can also be affected (Quinn et al. 2014). Icterus is often observed and subcutaneous edema can be present. Clinical signs include photophobia, agitation, and inappetence. Severe cases are marked by secondary bacterial invasion of lesions, anorexia, and recumbency and can eventuate in death.

Relatively few plant toxicoses have been reported in wild marsupials, despite a diverse and abundant herbivorous marsupial fauna in Australia and New Guinea alongside a rich diversity of native and invasive toxic plants.
Early reports included pygmy possums (*Cercartetus* sp.), feeding on honey in regions infested with heavy ragwort (*Senecio jacobea*), that exhibited hepatosis, likely indirectly caused by pyrrolizidine alkaloids in the plant (Munday 1988). Clinical signs consistent with *Phalaris* spp. toxicity were described in western grey kangaroos (*Macropus fuliginosus*) on Kangaroo Island, South Australia (Munday 1988). Pyrrolizidine alkaloid toxicity was also thought to be the underlying etiology of cases of hepatopathies observed in free-ranging red kangaroos (*Macropus rufus*; Munday 1988; Ladds 2009). However, several outbreaks of plant toxicoses have occurred in populations of wild marsupials since 2011. Eastern grey kangaroos (*Macropus giganteus*) were identified with neurological signs including ataxia, head tremors, recumbency, and in some cases, death, suggested to be caused by chronic *Phalaris* spp. toxicity (Bacci et al. 2014). Hair loss and dermatitis in weak and emaciated southern hairy-nosed wombats (*Lasiorhinus latifrons*) between 2011 and 2014 in South Australia, was suggested to be caused by pyrrolizidine alkaloid hepatotoxicosis most likely associated with ingestion of the forb *Heliotropium europaeum* (Woolford et al. 2014). Other plant species might also have contributed to the pathology.

Hepatogenous photosensitization has not been widely reported in marsupials to date, possibly due to the difficulty in diagnosis, where clinical biochemistry and necropsy is required for definitive identification. Suspected pyrrolizidine alkaloid toxicoses in southern hairy-nosed wombats, and *Lantana camara* toxicity in three captive red kangaroos (Johnson and Jensen 1998), are the only confirmed cases of hepatogenous photosensitization in marsupials. Clinical signs observed in the captive red kangaroos included anorexia, lethargy and jaundice, exudative dermatitis of ear margins, eyelids, muzzle, scrotum, and corneal opacity (Johnson and Jensen 1998). Here we describe the clinicopathologic features of a 2014 mortality event characterized by blindness, dermatitis, and hepatic dysfunction affecting eastern grey kangaroos in the Riverina region of New South Wales, Australia.

**MATERIALS AND METHODS**

An outbreak of morbidity and mortality associated with blindness or photophobia in 95 eastern grey kangaroos occurred between 10 April and 12 May 2014 at a rural property in the Riverina region of New South Wales, Australia (Fig. 1). The property supported a large, permanent population of kangaroos and habitats included open woodlands and grasslands with native and introduced grass species.

The case definition for the outbreak and analyses was an eastern grey kangaroo with histologic evidence of hepatobiliary disease and moderate to severe corneal edema or moderate to severe dermatitis. Eight of the affected animals were euthanized during the outbreak and were considered acute cases (cases 1–8). Three animals were euthanized within 6 wk following the outbreak and were considered chronic cases (cases 9–11), and six further animals of local provenance, which were not from the site experiencing the outbreak and did not exhibit photophobia, corneal opacity, icterus, or hair loss, were also examined and euthanized due to other disease. These latter six animals were treated as unaffected controls. All animals were reported by members of the public, collected by an authorized wildlife carer (New South Wales WIRES Licence 14170) and were euthanized on humane grounds following veterinary examination (including blood collection and hematologic and biochemical analysis). Procedures not directly relating to the veterinary diagnosis and treatment of the animals...
were approved by the Charles Sturt University Animal Care and Ethics Committee (approval 12/075).

All animals were subjected to a full clinical examination following sedation with intramuscular zolazepam/tiletamine at approximately 10 mg/kg (Zoletil 100 mg/mL, Virbac, Milperra, New South Wales, Australia) and whole blood was collected by venipuncture into serum and ethylenediaminetetraacetic acid tubes for clinicopathologic examination. Animals were then euthanized and full necropsy performed. All procedures took place at the Veterinary Diagnostic Laboratory Charles Sturt University, Wagga Wagga, Australia, with clinical pathologic analyses performed using commercial diagnostic equipment (CellDyn 3700 Hematology System, Abbott Diagnostics, Abbott Park, Illinois, USA; and Konelab 30i Clinical Chemistry Analyser, Thermo Electro Corporation, Vantaa, Finland). Analysis occurred within 1 h of blood collection. Biochemical analytes included total protein, albumin, globulin, creatinine, urea, creatine kinase, aspartate aminotransferase, alkaline phosphatase (ALP), alanine transaminase, glucose, total bilirubin, gamma-glutamyl transpeptidase (GGT), cholesterol, potassium, sodium, bicarbonate, calcium, phosphate, chloride, amylase, and lipase. In addition, albumin-globulin, sodium:potassium, and calcium:phosphate ratios and anion gap were calculated. Selected tissues fixed in 10% buffered formalin were processed for paraffin embedding and were examined for histopathology. Both eyes were resected and fixed in Bouin’s solution.

Analytes from outbreak, postoutbreak, and unaffected kangaroos were statistically compared \((\alpha=0.05)\) using R (R Development Core Team 2013). Data for creatine kinase, aspartate aminotransferase, total bilirubin, GGT, and lipase were log transformed prior to multivariate analysis of variance of all analytes for the three case factor levels. Tukey multiple comparison procedure on the factor levels was used for the four analytes and boxplots were generated. The range of total bilirubin, GGT, and ALP values in outbreak and unaffected kangaroos was compared to 95% confidence intervals generated from published mean and standard deviation data from healthy, captive eastern grey kangaroos (Vogelnest and Woods 2008).

Samples from the first affected animal during the early stages of the outbreak (and prior to the diagnosis of hepatogenous photosensitization) underwent ancillary testing for orbiviruses. Serum was examined for Wallal virus neutralization titer and pan- orbivirus reverse transcriptase-PCR was performed on spinal cord and brain material by a reference laboratory (Elizabeth MacArthur Agricultural Institute, New South Wales, Australia). Pasture grass samples were sent for botanical identification to the National Herbarium of New South Wales, Sydney, Australia.

RESULTS

Clinical signs

All animals at the outbreak site reported by the landholder as affected with clinical signs of blindness or photophobia but not collected \((n=88)\) died during the period of the outbreak and we were not able to examine them. Animals collected from the site of the outbreak (cases 1–6, 8) exhibited photophobic behavior, blindness, ataxia, recumbency, lethargy, ear shaking, and behavior consistent with distress or depression. Photophobia manifested as abnormal shade-seeking behavior including finding refuge in or under structures frequented by people.

All animals (7/7) had pale-colored corneal opacity (Fig. 2) and variable icterus on physical examination. Two of seven had poor body condition, including muscular atrophy in the trapezius and lateral femoral regions, and one kangaroo had regions of thickened, sparsely haired skin on the elbows and thorax. Some kangaroos had unilateral or bilateral mucoid ocular discharge.

One single joey (pouch emergent young) (case 7) had a fractured leg and had died at a nearby location during the period of the outbreak, likely the casualty of a road traffic accident. This animal exhibited histologic lesions consistent with hepatogenous photosensitization and was therefore considered an outbreak animal for clinical and anatomical analysis.

Two postoutbreak animals collected from the outbreak site (cases 10–11) were weak, in poor body condition, and had skin lesions but no corneal edema. A single animal near the outbreak site that had a fractured tibia due to vehicle impact (case 9) was also histologically consistent with a postoutbreak kangaroo (although lesions were subtle).

Clinical pathology

Routine veterinary diagnostic hematology and biochemistry performed from seven out-
break kangaroos (cases 1–7), two postoutbreak kangaroos (cases 9–10), and six unaffected control kangaroos revealed significant differences in absolute blood monocyte counts ($P=0.02$), total bilirubin concentration ($P=0.001$), and GGT level ($P=0.02$). Values were approaching significant differences in amylase ($P=0.06$) between outbreak kangaroos, postoutbreak kangaroos, and unaffected kangaroos (Fig. 3). Electrolyte ratios, bicarbonate, and anion gap values were not considered analytically useful because of outliers.

Total bilirubin and GGT concentrations were significantly greater in the outbreak kangaroos than either postoutbreak (total bilirubin, $P=0.020$; GGT, $P=0.050$) or unaffected (total bilirubin, $P=0.002$; GGT, $P=0.030$) kangaroos, which did not differ for these analytes. Monocyte counts were significantly greater in the postoutbreak kangaroos than either acute ($P=0.04$) or unaffected ($P=0.01$) groups. Significantly higher amylase was observed in the postoutbreak group than the outbreak group ($P=0.05$), although neither group differed significantly from unaffected kangaroos.

The range of total bilirubin values seen in outbreak animals were above 95% reference intervals for healthy eastern grey kangaroos, whereas considerable overlap was observed between outbreak, unaffected, and reference healthy kangaroos for GGT. All ALP values in this study ($n=15$), with one exception (1,057 U/L), were below the mean ALP value for healthy reference kangaroos (mean=1,037 U/L, SD=775 U/L, $n=46$; Table 1).

**Pathologic examination**

Outbreak ($n=8$) and postoutbreak kangaroos ($n=3$) exhibited variable body condition.
The majority of outbreak kangaroos \((n=7)\) had white corneal opacity (Fig. 2). The only animal without this lesion was a subadult. In some individuals, the corneal lesion had a central or ventrocentral horizontal distribution. Most animals \((n=6: four\ acute\ and\ two\ chronic)\) had subtle to obvious skin lesions, including excoriations, thickening, and scab formation. Three outbreak animals exhibited icterus. On postmortem, apart from icterus, the only significant finding was a discolored liver \((n=6)\) in outbreak kangaroos. The presence of these gross changes was inconsistently distributed across the 10 individuals examined; however, all affected animals except an individual with a fractured leg (case 7) had either corneal opacity or skin lesions. Case 7 did, however, have histologic lesions of both hepatobiliary disease and dermatitis (thereby meeting the case definition).

Histologic sections of eyes, kidney, skin, liver, spleen, and integument were examined, except for one individual for which fixed integument was not available. Of the 11 clinically affected animals examined, all had a mild to severe hepatopathy, most \((n=8)\) with cholangiohepatitis. Seven individuals had variable numbers of acicular clefts present in portal regions. In addition, all affected animals had either mild to severe dermatitis \((n=10)\) or mild to severe corneal edema \((n=9)\). Nine kangaroos with dermatitis had moderate to severe necrotizing or ulcerative lesions. Only one individual had dermatitis without corneal edema, and one other animal with corneal edema did not exhibit skin lesions. Mild to moderate keratitis was also frequently observed \((n=5)\), and always was accompanied by corneal edema. Histologic lesions were more consistently represented across the 11 affected cases than were gross lesions. Abnormalities of the ocular lens, retina, or optic nerve were never observed.

No reportable lesions were observed in the spleen or kidney of any affected case. A selection of other formalin fixed tissues including lung, lymph node, heart, stomach, and pancreas were examined in individuals, and apart from a chronic fungal granuloma observed in a single case, no significant pathology was observed in these tissues.

Corneal lesions ranged from mild (cases 4, 8, 10) to moderate (cases 3, 5, 6, 11) or marked (cases 1, 2) focal to diffuse edema with variable accompanying mixed inflammation of the cornea, superficial limbus, or anterior uveal tract (Fig. 4). Congestion of limbic vessels and foci of hemorrhage were also occasionally present.

The integument of the eyelid (cases 2, 3, 5–8, 11) and pinna (cases 5–7, 9, 11) was variably affected, with moderate to severe focal to extensive inflammation and necrosis. Other regions of integument including, the chest and forearm (cases 2, 4, 8, 10), had mild to severe focally extensive necrotizing dermatitis with massive numbers of infiltrating and exudating neutrophils and other inflammatory cells (Figs. 5, 6), accompanied by serocellular crusting.

All cases had a degenerative hepatopathy, although the severity and extent of hepatic  

### Table 1. Range of values for bilirubin \((n=42)\), gamma glutamyl-transpeptidase \((n=35)\), and alkaline phosphatase \((n=46)\) measured during an outbreak of hepatogenous photosensitization in eastern grey kangaroos \((Macropus\ giganteus)\) apparently due to grazing on an introduced grass, Panicum gilvum in New South Wales, Australia. The range of values for outbreak \((n=7)\) and unaffected kangaroos \((n=6)\) are compared to 95% reference intervals for healthy eastern grey kangaroos.

<table>
<thead>
<tr>
<th>Analyte</th>
<th>Outbreak</th>
<th>Unaffected</th>
<th>Reference intervala</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bilirubin ((\mu\text{mol/L}))</td>
<td>7–499</td>
<td>1–10</td>
<td>0–7</td>
</tr>
<tr>
<td>Gamma glutamyl-transpeptidase (U/L)</td>
<td>17–49</td>
<td>9–24</td>
<td>0–37</td>
</tr>
<tr>
<td>Alkaline phosphatase (U/L)</td>
<td>141–1,057</td>
<td>91–501</td>
<td>0–2,615</td>
</tr>
</tbody>
</table>

lesions varied significantly. In cases 1, 9, and 10, there was a mild to moderate cholangiohepatitis, and hepatocytes contained mild (case 10) or moderate (cases 1, 9) amounts of yellow-brown intracytoplasmic material. The liver of case 2 had widespread accumulation of yellow-brown pigment in Kupffer cells, clustered hepatocellular necrosis associated with infiltrating neutrophils, and acicular clefts both in the hepatic parenchyma and associated with small bile ducts, sometimes associated with inflammation. In case 3, moderate inflammatory infiltration was present in periportal regions and was associated with mild to moderate fibrosis. There was moderate accumulation of yellow-brown pigment in and adjacent to hepatocytes. There was widespread disruption of hepatocellular cords in case 4. Many hepatocytes had eosinophilic intranuclear inclusions consistent with cytosegrosome bodies. There was a moderate amount of brownish, mostly intracellular pigment granules in both hepatocytes and Kupffer cells. There was a mild to moderate infiltration with lymphocytes and plasma cells in portal areas. Rare, mostly intracytoplasmic, acicular clefts were present in small biliary ducts. There was also scattered individual hepatocellular necrosis. Throughout the livers of cases 5, 6, and 7 there was mild congestion and scattered individual necrosis of hepatocytes. There was also a mild mixed inflammatory cellular infiltration mostly in portal areas, and several small bile ducts of cases 5 and 7 had intraluminal acicular clefts disrupting the epithelial cells. In case 6, there were focal periductular regions of marked hepatocellular collapse and replacement with amorphous acellular material. Similarly, case 8 had moderate to marked infiltration of peri-
portal regions with mixed inflammatory cells accompanied by scattered cellular necrosis, and at multiple foci, complete collapse of the hepatic parenchyma and replacement with amorphous acellular material (Fig. 7). There was mild to moderate accumulation of brown pigment in Kupffer cells and moderate scattered hepatocellular necrosis. Numerous intraluminal acicular clefts were seen in small bile ducts of case 8, often disrupting biliary epithelium, and clefts were also seen extracellularly in periportal regions (Fig. 8). Case 11 had moderate to marked multifocal predominantly lymphoplasmacytic infiltrations in the liver, principally associated with bile ducts. There was marked accumulation of yellow-brown pigment in hepatocytes, which was positive with Perls’ Prussian Blue stain.

Ancillary testing

Serologic and reverse transcriptase-PCR testing for orbiviruses was performed on the first affected kangaroo examined in the outbreak. Although panorbivirus results were negative, the virus neutralization titer for Wallal virus was 512. This suggested previous exposure to Wallal virus, with establishment of protective adaptive immunity, but no current orbiviral infection was associated with the outbreak. Dominant pasture grass samples were identified as Panicum gilvum by the National Herbarium of New South Wales.

DISCUSSION

Secondary hepatogenous photosensitization was diagnosed as the proximal etiology of an outbreak of blindness in eastern grey kanga-
roos in the Riverina region of New South Wales, Australia. All examined affected animals had hepatobiliary lesions and mild to severe inflammation of regions of the cornea and integument that was exposed to sunlight.

Histopathologic lesions of the liver, skin, and cornea were highly consistent among outbreak individuals. All cases examined during the outbreak had hepatobiliary disease and most had both corneal and skin lesions. The histologic appearance of these lesions was relatively well conserved with mild keratitis accompanied by moderate to severe corneal edema and affected skin typically exhibiting a moderate to severe necrotizing or ulcerative dermatitis. Distal extremities such as the pinna and forearms, which, although pigmented, are more sparsely furred than other parts of the body, were most often affected. Gross findings by contrast were slightly more inconsistent, with fewer affected animals exhibiting both icterus or liver discoloration and skin or corneal lesions. This highlights the importance of histopathology as a more sensitive diagnostic method for the detection of liver, eye, and skin disease in wildlife than clinical or gross examination.

Corneal edema and keratitis causing impaired vision and blindness was a consistent feature of the cases of hepatogenous photosensitization in eastern grey kangaroos seen in this outbreak. By contrast, it is a very seldom observed sequela of photosensitization in domesticated herbivores, and was not reported in southern hairy-nosed wombats affected by hepatogenous photosensitization (Woolford et al. 2014). Corneal edema might be a feature of hepatogenous photosensitization in
a broader taxonomic group including other kangaroo species. This hypothesis is supported by the only other reported case of hepatogenous photosensitization in a red kangaroo, in which corneal opacity is also described (Johnson and Jensen 1998).

All except one of the outbreak kangaroos examined had mild to severe corneal edema. The exception was a joey that was too large to remain in the maternal pouch (pouch emergent young), although it might have been small enough to find more effective shade refuge during the day. The open woodland environment of the outbreak site, with large sun-exposed grazing areas, would likely have forced exposure to sunlight in outbreak adult animals. The free-ranging nature of the population would likely have exacerbated the impact of corneal lesions on survival due to misadventure and predation associated with poor vision.

Total bilirubin was elevated above normal reference ranges in outbreak animals, whereas GGT was less consistently elevated. Elevations of both analytes were correlated with outbreak cases, reflecting the biliary component of the disease. In contrast, ALP showed no association with either affected cases or, for that matter, with hepatobiliary disease more generally. This suggests that GGT is a moderately sensitive indicator of cholestasis in eastern grey kangaroos whereas ALP is insensitive to such lesions. This reflects the association these enzymes have with cholestatic disease in domestic herbivores (Bain 2011). The levels of ALP are often elevated in healthy captive macropods (mean 1,037 U/L, n=47; Vogelnest and Woods 2008) but in the

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**Figure 7.** Hepatic parenchymal replacement with amorphous acellular material and foci of mixed inflammation. These sites appeared to be predominantly associated with periportal regions as in the focus in the center right of the image. Similar lesions were observed in two of the eastern grey kangaroos (*Macropus giganteus*) affected during an outbreak of hepatogenous photosensitization in the Riverina of New South Wales, Australia (case 8). H&E stain.
wild kangaroos in our study, this was not the case (mean 416 U/L, \( n=15 \)). The reason for this difference is unclear because the pathology observed in our cohort would be assumed to be more significant that that encountered in captive populations. As such, ALP might act as a possible indicator of chronic (including endogenous glucocorticoid induced) hepatobiliary dysfunction, gastrointestinal, or bone pathology associated with captivity in macropods, and as such, might warrant further investigation in this regard.

Hyperbilirubinemia was a consistent feature in acute to subacute cases of hepatogenous photosensitization in our cohort of eastern grey kangaroos and, although not specific for hepatogenous photosensitization, is likely to be useful diagnostically. Unexpectedly, the two postoutbreak cases showed significant elevations in circulating monocytes and amylase relative to both outbreak and unaffected individuals. The mechanism by which these were elevated specifically in postoutbreak cases is unclear.

The variable numbers of acicular clefts present in the portal regions of seven of the eight outbreak animals examined histologically were highly suggestive for steroidal saponin toxicity. This toxin is present in some plants at particular life stages and exists as various metabolites. Accumulation of the saponins in bile ducts causes structural damage to the biliary epithelium and cholestasis as well as an associated cholangiohepatitis. Steroidal saponin toxicity is well-recognized as an etiology for hepatogenous photosensitization in sheep in Australian, New Zealand, and South Africa.

**FIGURE 8.** Acicular clefts (arrows) associated with periportal regions, predominantly within the lumen of small bile ducts, of an eastern grey kangaroo (*Macropus giganteus*) affected during an outbreak of hepatogenous photosensitization in the Riverina of New South Wales, Australia (case 8). These were often observed disrupting biliary epithelial integrity and associated with mild to moderate mixed inflammatory cell infiltration. Scattered hepatocellular necrosis can be observed in the surrounding hepatic parenchyma. H&E stain.
Glastonbury et al. 1984; Holland et al. 1991; Miles et al. 1994).

Observations at the outbreak site identified pasture dominance by the introduced annual pasture grass Panicum gilvum, also known as sweet grass or sweet panic. Panicum grasses have historically been associated with steroidal saponin hepatotoxicity because they possess steroidal or lithogenic saponins that cause hepatobiliary dysfunction (Bridges et al. 1987; Button et al. 1987; Holland et al. 1991; Smith et al. 1992; Quinn et al. 2014). Despite such associations, little rigorous evidence exists confirming such causation in either domestic or wild herbivores. Biochemical profiling for toxic secondary metabolites in dominant plant species associated with a toxic outbreak is required to conclusively identify the causal species as etiologic agents of the disease, although Panicum spp. are likely candidates in this case.

We did not assess the chronic impact of severe corneal or hepatobiliary lesions on the fitness of affected animals. The deaths we observed were unlikely to represent the extent of morbidity and mortality associated with this outbreak in the affected kangaroo population. The liver is thought to be capable of significant regeneration; however, the energetic costs (in terms of healing, immune function, and lost nutrition) of cholangiohepatopathy might have effects on individual survival and recruitment and thus on the population.

One of the most significant aspects of identifying plant toxicosis as the cause of this outbreak in kangaroos is its close temporal association with two other reports of plant toxicity in wild herbivorous marsupials: pyrrolizidine alkaloid hepatotoxicosis in southern hairy-nosed wombats (Woolford et al. 2014), and chronic phalaris toxicity in eastern grey kangaroos (Bacci et al. 2014), both between 2011 and 2013. Despite a relatively rich, decades-long literature on the diseases of free-ranging wombats and kangaroos, these are the first reports of plant toxicities causing morbidity and mortality in any wild herbivorous marsupial. Such a temporal association is unlikely to be coincidental, especially in light of the independent investigation and reporting of all three cases in the context of ongoing wildlife disease surveillance. A prolonged period of drought in southeastern Australia ended in 2010 and conditions remained relatively wet across the region into 2014. All three events were associated with the dominance of degraded pastures with specific introduced grasses and forbs. We propose that opportunistic dominance of such grasslands by invasive species which are able to exploit wet seasons after prolonged drought is likely to be an increasing feature of Australian temperate semiarid environments, and we predict that these climatic events will be associated with an increasing frequency of plant toxicities in wild herbivorous marsupials.

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LITERATURE CITED


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