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Poor Survival Rate of Eastern Gray Kangaroos (*Macropus giganteus*) Affected by Chronic Phalaris Toxicity

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ABSTRACT: Chronic phalaris toxicity (CPT) is a neurological disease caused by animals ingesting toxins produced by early growth stages of *Phalaris aquatica*, a pasture plant introduced to the southeastern regions of Australia postcolonization. Little is known about the clinical progression of CPT in wildlife, as incidents are sporadic and predominantly reported when animals are in the end stages of disease and in a poor welfare state. We studied a cohort of 35 eastern gray kangaroos (*Macropus giganteus*) affected by CPT to clarify clinical prognosis and survival rates. Kangaroos were captured in May, June, and July of 2022 at Plenty Gorge Parklands, Victoria, Australia. Each animal was radiotracked for 180 d, clinical progression and disease outcomes monitored twice a week. By the conclusion of the study, 24 animals had died (19 by euthanasia due to deterioration, five found dead). Ten animals survived, with two demonstrating a reduction in clinical signs and eight showing full resolution of clinical signs. One animal was disqualified from the study. The overall survival rate was 29.4% (95% confidence interval 17.5–49.5%). The survival duration of animals that died ranged from 5 to 133 d. There was no difference in survival rate based on sex ($P=0.2$), age class ($P=0.49$) or the month of capture ($P=0.49$). These results suggest that CPT is an important health and welfare concern for at-risk macropod populations, with high case-fatality rates and prolonged clinical durations. Further research to manage the disease via methods such as reducing *Phalaris aquatica* plant coverage and preventative treatments for animals is warranted to reduce disease incidences and improve disease outcomes in wildlife populations.

Key words: Disease, introduced, kangaroo, plant, survival, toxicity, tracking, welfare.

INTRODUCTION

Anthropogenic environmental modifications due to urbanization and agriculture have had many important impacts on wildlife species, such as an increase in emerging infectious diseases, loss of biodiversity, and the introduction of invasive plant species (Daszak et al. 2001). Invasive weeds and pasture species may affect native animal species through alterations of their food availability, natural behavior and toxicities (Yan et al. 2001; Crawford et al. 2015; Stewart et al. 2021). Toxicosis from introduced plants as a form of anthropogenic wildlife disease has been documented in Australian mammals ingesting novel plants such as *Panicum gilvum* (Steventon et al. 2018), *Heliotropeum europaeum* (Woolford et al. 2014), and *Phalaris aquatica* (Bacci et al. 2014).

Phalaris aquatica (*Phalaris tuberosa*), a perennial grass, was introduced into Australia in the early 1900s to improve pasture for livestock farming (Oram et al. 2009). In the 1940s, a "staggers"-like neurological disease was identified as being associated with sheep (*Ovis aries*) grazing the pasture and was later referred to as chronic phalaris toxicity (CPT; McDonald 1942). Experimental evidence suggests that CPT is caused by the ingestion of tryptamine alkaloids produced by the plant, causing hyperstimulation of serotonergic receptors within neurons (Bourke et al. 1990). The alkaloids identified in *Phalaris* spp. include dimethyltryptamine (Culvenor et al. 1964), 5-methoxy-N-methyltryptamine (Wilkinson 1958; Culvenor et al. 1964), gramine (Culvenor et al. 1964), hordenine (Wilkinson 1958), bufotinine (Culvenor et al.

1964), and β-carboline (Frahn and O'Keefe 1971). These toxins, mostly contained in the leaves, are at their highest concentration during early phases of growth, which often occurs in March and April in southeastern Australia and can be further increased during periods of moisture insufficiency (Marten 1973; Read et al. 2020). Affected animals present with ataxia, muscle tremors, head nodding, and hyperexcitability (McDonald 1942; Lee and Kuchel 1953; Alden et al. 2014).

In recent years, there has been an increase in reports of free-ranging eastern gray kangaroos (*Macropus giganteus*) around Victoria, Australia, showing similar signs to sheep affected by CPT (N. Davis, Parks Victoria, pers. comm. 2021). Clinical signs have included ataxia, muscle tremor, hypermetria, and erratic tail sways. A histological diagnosis of CPT was made for a representative cohort by visualizing pigment accumulation in neurons within the thalamus and locomotion centers of the brain of affected animals (Bacci et al. 2014). Although the presenting signs and pathology have been described, the clinical progression of the disease in kangaroos and associated time frames have not been reported.

In sheep, clinical progression can vary considerably. Animals from a given flock may have different outcomes; some animals die within a range of 2–21 wk after showing clinical signs, but others have survived until the end of studies (Lee and Kuchel 1953; Bourke et al. 1987). Some animals have continued to show clinical signs for up to 3 mo after removal from *Phalaris* pasture without either further improvements or deterioration (Le Souef 1948), and others with milder signs who were removed quickly from the pasture have been able to recover fully (McDonald 1942).

The clinical progression of CPT in kangaroos may differ significantly from that in livestock because of two factors. First, sheep often have little choice but to graze pastures of *P. aquatica* with minimal alternative feed, because of agricultural practices (Le Souef 1948; Milne 1955), whereas free-ranging macropods are likely to have wider home ranges that have reduced availability and coverage of *P. aquatica* compared to farms. This may lead to a difference in the rate of alkaloid toxin intake. Secondly, sheep are likely to be removed from *Phalaris* pasture and put on alternative feed at the onset of clinical signs of CPT (Bourke et al. 1987), but this is not the case with wildlife, because relocation or exclusion of entire at-risk populations from existing habitats poses significant technical, ecological, and welfare challenges (Cowan et al. 2020). As a result, although the rate of intake might be slower, macropods are likely to be exposed to *Phalaris* spp. plants during the toxin-producing growth phase for longer periods, potentially leading to very different clinical outcomes. The unknown progression of disease combined with additional practical challenges in surveillance of at-risk wild macropod populations further raises concerns about the animal welfare impacts of CPT, as these animals may potentially be affected by debilitating neurological signs for prolonged periods of time. It is therefore valuable to examine the health and welfare impacts of CPT directly in at-risk macropod populations.

Detection of cases of CPT is more difficult in wildlife than in livestock; reporting of cases is more sporadic and often lacks longitudinal information for individuals. In addition, affected animals tend to show cryptic behavior and experience reduced mobility, further lowering the chance of an affected animal being detected. When a case is reported, the animal often is already displaying severe clinical signs, hence it is not possible to determine the duration of clinical disease. Our study aimed to address these knowledge gaps in clinical progression and disease outcomes of CPT, including survival rates, by radiotracking and observing initially mildly affected wild eastern gray kangaroos throughout the clinical course of the disease.

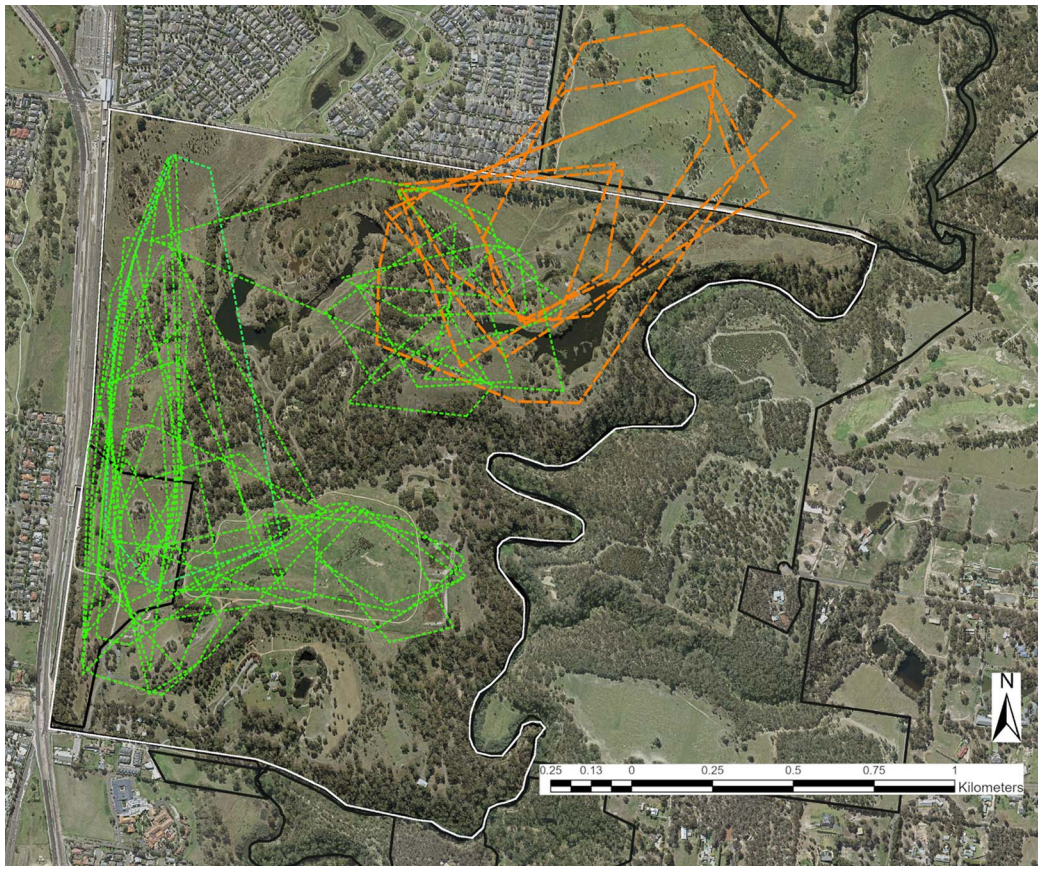

FIGURE 1. Aerial map of Plenty Gorge Parkland (37°37′S, 145°06′E), Bundoora, Victoria, Australia where eastern gray kangaroos (*Macropus giganteus*) cohort with chronic phalaris toxicity were VHS radiocollared and tracked. Dashed and dotted lines: Minimum convex polygon showing the area each animal (n=34) was located during diurnal tracking (total GPS coordinates n=728). Dashed lines represent the six animals that roamed outside the study site during tracking; dotted lines represent the remaining cohort. Continuous line: study site boundary.

MATERIALS AND METHODS

The study was conducted at Plenty Gorge Parklands (37°37′S, 145°06′E), Bundoora, Victoria, Australia, which is situated in the periurban fringe of Melbourne. The northern region of the park consists of a combination of open grassland and grassy woodland, with large patches of *Phalaris aquatica* present because of previous use of the site for farming. The focus for this study was the northern region of the park, which consists of the Morang Wetlands and the Hawkstowe Picnic Area (230 ha; Fig. 1). A wire fence runs along the northern, western, and southern borders of this area with a deep gorge on the eastern side. Despite these barriers, macropods were seen during this study to be moving in and out of the study site onto adjacent, non–*P. aquatica*–covered grasslands. The predominant macropod species present is the eastern gray kangaroo, with an estimated population of 1,000 animals (N. Davis, Parks Victoria, pers. comm.) and a smaller number (unknown population size) of swamp wallabies (*Wallabia bicolor*). Each year, approximately 10–20 kangaroos with clinical signs consistent with CPT are reported by members of the public and park authorities at this site (N. Davis, Parks Victoria, pers. comm.). In 2021, 15 such clinically affected animals were diagnosed with CPT via histopathology by the University of Melbourne Veterinary School, Werribee, Victoria, Australia.

We opportunistically selected 35 eastern gray kangaroos for the study during 1 May to 31 July 2022 (winter), based on the presence of clinical

signs consistent with mild CPT. Areas with accessible terrain (avoiding water bodies, cliffs, and dense untraversable understory, equating to approximately 70% of the parkland area) were searched on foot weekly for animals that matched the clinical criteria of mild stages of CPT (see Supplementary Material Table S1), which included mild head nodding, flaccid ears, decreased flight zone, and ataxia. It was not possible to select animals representing different areas or mobs systematically; because of the scarcity of diseased animals; based on past reports, the prevalence is estimated to be less than 5% of the population

Once identified, animals were immobilized (with) using a combination of xylazine (2 mg/kg Randlab, New South Wales, Australia) and tiletamine–zolazepam (1.25-2 mg/kg Zoletil (Virbac Australia, Milperra, New South Wales, Australia) injected intramuscularly via 1-mL darts (Pneudart type P gel collared triport darts, Pneudart, Williamsport, Pennsylvania, USA) fired from a Pneudart G2 X-Caliber dart gun (Pneudart). Age class (subadult or adult) was determined by on-site observations of the animal's behavior and appearance before capture as described (Jaremovic and Croft 1991); sex was determined by the presence of a pouch or descended testicles. Body condition assessment was made based on muscle and fat coverage around the tail base, at the dorsal spinous processes between scapulae, and at the hip bone (Edwards et al. 2013; Brandimarti et al. 2021). A clinical examination was performed on each individual after capture; only animals that were of a body condition score of 2.5/5 or above and without any other clinical findings (e.g., physical injuries, infections) were selected for marking and collaring with a VHF telemetry device (see the following for details). Animals were monitored for heart rate, respiratory rate, and temperature during sedation.

All study animals had a VHF telemetry collar fitted around the neck under sedation. The devices used (V6C 173 series, Lotek, Havelock North, New Zealand) weighed 130 g +/−3% (<1% of body weight of study animals) which was within the guidelines of <5% body weight for a telemetry device (Cornelsen et al. 2022). The frequencies of the VHF collars were set to be between 150 and 152 MHz. A latex weak link (consisted of a 6-cm-long, 1-cm-wide, 4-mm-thick latex tube) with similar design to Cowan et al. (2020) was installed on the collars; this acted as a passive drop-off unit to prevent entanglement and to achieve remote release of the collar at the end of the study (within 12 mo). An extra 2 cm of space was left between the collar and neck to allow movement of the collar. This space and the short lifespan of the weak link accommodated any growth of subadult animals collared and was expected to mitigate any negative impacts of the collar on the survival of all studied animals.

The animals were also ear tagged and microchipped for identification. A color-coded two-piece goat ear tag (Mini male 10, 51 mm (W) × 17 mm (H) and a Mini female button, Allflex, Queensland, Australia) was placed within the lower proximal region of the pinna that had minimal blood vessels (Silvy et al. 2012). The button tags that were attached onto the concave side of the ear were slightly modified by trimming two sides of the circle down to create an oval shape to fit the shape of the ear. After tagging, a microchip (ISO FDX-B, Virbac Australia, Milperra, New South Wales, Australia) was implanted subcutaneously between the scapulae.

Partial reversal of chemical sedation via intramuscular injection of atipamezole (100 μg/kg) was performed after marking. After the reversal agent was given, animals were transported to a safe recovery location within the grassland. Recovery was monitored from a distance of 50 m, with full recovery indicated when the animal was able to stand up and move away from the observers without falling. Kangaroos were radiotracked 24 h after the capture event to ensure there were no obvious adverse outcomes from the capture.

Each animal was tracked on foot using a VHF receiver (TR-2 telonic receiver, Mesa, Arizona, USA) and assessed visually during daylight hours twice per week for 180 d. The location of the animal and stage of disease were recorded in line with the clinical description (Supplementary Material Table S1) along with any observed complications related to the tracking collar or ear tags, such as alopecia, dermatitis, or entanglement. Because not all affected animals exhibit all clinical signs, the overall clinical stage of an animal was considered to have progressed to the next stage when any clinical sign satisfied the next stage's description. The clinical duration of each stage of disease was also recorded. Duration was calculated as the number of days between the first

observation of a certain stage of disease until the day it progressed to the next stage.

Kangaroos at the severe stage of disease (Supplementary Material Video 1) had significantly compromised ambulatory functions with perceived increased risk of misadventure or motor vehicle accident. They were also considered a potential safety risk to park visitors. Thus, if a tracked animal progressed to the severe stage of disease during the study, the animal was euthanized on safety and welfare grounds. Kangaroos to be euthanized were sedated remotely as described above. Pentobarbitone sodium 325 mg/mL (Lethabarb, Virbac Australia, Milperra, New South Wales, Australia) at a dose of 2 mL/kg was injected intravenously until cardiac arrest was confirmed via auscultation.

Euthanized animals were transferred to the Melbourne Veterinary School for a full necropsy. Animals were examined for physical injuries, body condition, gross changes to organs, and the presence of other disease processes. A diagnosis of CPT was made at necropsy by the detection of gray–green discoloration of the cerebral cortex and/or thalamic regions of the brain, followed by the microscopic identification of a granular brown pigment within neurons in histologic sections prepared from the grossly affected regions, as described (Bacci et al. 2014). The histologic sections were subsequently stained with periodic acid-Schiff in an attempt to exclude the noted pigment being a polysaccharide or lipopigment and also Fontana–Masson stains to confirm that the identified pigments were melanin-like. All histological examinations were conducted by a board-certified veterinary pathologist (RP). A Kaplan–Meier estimate, including 95% confidence intervals, was conducted on the entire cohort as well as subgroups (month of capture, sex, and age class) using data analysis program Jamovi v2.3 (The jamovi project 2024).

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RESULTS

One animal was disqualified from the data set, as we could not determine if a prolonged anesthetic recovery and reduction in ambulatory functions was iatrogenic and contributed to the death, and no diagnostic information was obtainable from the heavily scavenged carcass. Of the remaining animals, 24/34 died during the 180-d monitoring period (Supplementary Material Figs. S1 and S2). Of these, 19 were euthanized for welfare reasons because of the presence of severe clinical signs. Three animals were found dead and displayed evidence of trauma, suspected to be due to predation, as blood was evident around the carcass, and there were injuries to the neck region with suspected bite marks on the collar. One animal was found dead with signs of vehicle collision (found near a road with no signs on the carcass or tracking collar of animal attack) and human interference (collar antenna was cut at the base with the cut surface suggesting the use of a tool). One animal was found drowned in a lake. No complications related to the telemetry collar or ear tags were observed throughout the study.

The estimated survival rate was over 180 d and mean survival duration for the entire cohort was 29.4% (95% confidence interval [CI]=17.5– 49.5%; Fig. 2) and 89.5 d (standard deviation [SD]=66.5 d), respectively. The mean survival duration for the animals that died was 51.8 d (SD=36 d). The mean number of days animals were at the mild and moderate stages of disease for the entire cohort was 48.1 d (SD=55.4 d) and 26.6 d (SD=26.5 d), respectively. The mean number of days animals were at the mild and moderate stages of disease for the animals that died was 19.5 d (SD=21.3 d) and 31.6 d (SD=27.9 d), respectively. Finally, the mean number of days animals were at the mild and moderate stages of disease for the group that survived was 117 d (SD=52.1 d) and 17.2 d (SD=20.7 d), respectively.

There was no statistical difference from the Kaplan–Meier estimate between the survival of male versus female (P=0.20) or adult versus subadult (P=0.49) kangaroos. There was also no statistical difference between the capture month of May versus June versus July

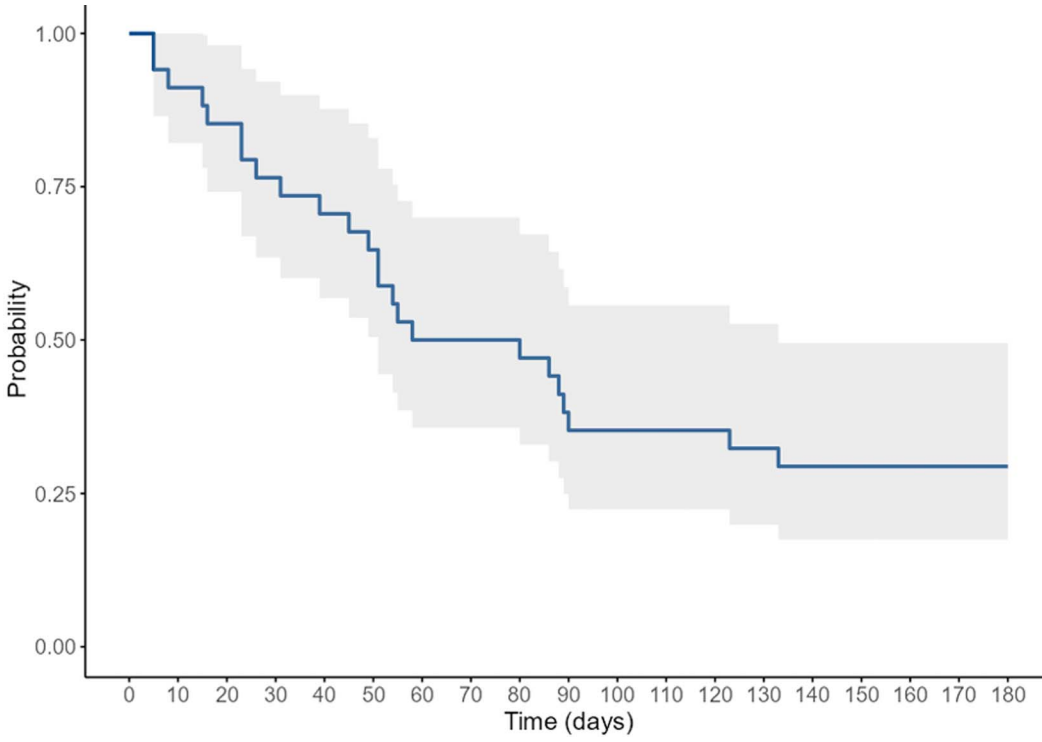

FIGURE 2. Kaplan–Meier estimate single-arm survival curve for the entire eastern gray kangaroo (*Macropus giganteus*) cohort (n=34) with chronic phalaris toxicity studied at Plenty Gorge Parklands (37°37′S, 145°06′E), Bundoora, Victoria, Australia in May–December 2022. The solid line indicates the estimated probability of survival at any given day postcapture. The shaded area is the 95% confidence interval.

(P=0.49; paired analyses between May versus June [P=0.56], May versus July [P=0.47], and June versus July [P=0.28] were also insignificant). Over the course of the study, six animals were sighted on at least one time point grazing the adjacent non-*Phalaris* grasslands north of the study site, having gone through the fence barrier (Fig. 1). This area had broadly the same ecological environment as the study site, but was considered free from any *P. aquatica*. This group showed a trend towards higher survival compared to kangaroos that were sighted only within the study site, but the sample size was too small to determine whether this difference was significant.

Of the 10 animals that remained alive on the final day of the study period, 6 mo after capture, eight were not displaying any clinical signs (full clinical recovery) and two had clinical signs of mild CPT. Three furred pouch young were removed from females that were euthanized during the study because of progression of disease. These animals were sent to rehabilitation centers. There was no evidence of vertical transmission of CPT in these three animals.

All 19 kangaroos euthanized during the study were diagnosed with CPT based on necropsy and histopathological examination. Of these, 17 were assessed to have a body condition score below 2.5/5. Six animals had a pouch infection; one also had a mummified pouch young. Six animals were observed to have high lice burdens, and two animals had subcutaneous ecchymosis in the skull region. None of the animals found dead in the field were in a state that held any diagnostic value.

DISCUSSION

This study measured a survival rate of eastern gray kangaroos affected by CPT of <30% over 180 d of observation, with animals

experiencing up to 4 mo of progressive clinical disease prior to death. The clinical signs observed, and their duration, suggest poor animal health and welfare impacts that should prompt increased attention towards the management of CPT, especially in locations where it occurs frequently, such as on the northern periurban regions of metropolitan Melbourne (Chen et al. 2024).

Despite a high case fatality rate, some kangaroos improved or even appeared to recover completely over the course of our study. These findings share many similarities with cases described in sheep, where Bourke et al. (1987) observed that within the same mob, some animals died over a period of 16 wk, but others survived and had no clinical signs after 20 wk. These results suggest that kangaroos may be similarly susceptible to, and affected by, CPT as sheep, with the potential to cause high mortality rates within affected cohorts.

There was no difference in survival probability between the months of capture, potentially because of the low sample size. Nevertheless, this could also be due to toxins remaining within the plant for extended periods of time during the growth phase of the plant, as described both by Lee et al. (1956) and Read et al. (2020), which causes animals that develop clinical signs during this period to have a similar fate. It is important to note that outcomes may be different during other times of the year, as it has been shown that dried tubers and straws of *P. aquatica* are not toxic when fed to sheep, even when the same paddock produced CPT cases in the same year (Lee et al. 1956). Hence, it is plausible that during months outside of May, June, and July, the progression and outcome of the disease might be less severe because of lower toxin levels within the plants.

The noted trend towards higher survival rates for the six animals that had parts of their diurnal home range outside the *Phalaris*-dominant study suggests that animals that remove themselves partially from the affected pasture may experience reduced impacts associated with CPT. Analogous observations can be seen in sheep, where quick removal from *Phalaris* pasture results in faster and more complete resolution of clinical signs (McDonald 1942). Our ability to obtain consistent data on animal movement and pasture utilization was limited by the VHF collars used. Future studies should implement a better tracking system such as GPS to explore this hypothesis in more detail. Nonetheless, this finding suggests that reducing the cover of *P. aquatica* in the home range of kangaroos, or preventing restriction of populations to areas with a heavy presence of *P. aquatica*, might improve the survival of animals affected by CPT. This may be achieved by direct removal of *P. aquatica*, or by provision of urban wildlife corridors to allow populations to seek alternative feed (Zellmer and Goto 2022). Implementing these management actions, however, is challenging, and may result in further ecological disturbances within the region, such as environmental contamination with herbicides or invasion of other weed species (Kanissery et al. 2019). Landholders should formulate site-specific plans through consultation with ecological professionals based on the risks and limitations of different management approaches.

Our study demonstrated that the clinical impacts of CPT may vary from mild effects on normal behavior, such as a mild ataxia, to severe impacts, such as full body tremors, which are likely to cause high levels of stress for multiple weeks. In addition to these clinical signs, necropsy identified several animals with comorbidities including emaciation, increased ectoparasite burdens, and pouch infection with mummified pouch young. It is possible that these new findings are the result of prolonged exposure to the toxin, which may potentially lead to increased energy expense from muscle tremors and hypermetria, and to chronic disruption of normal behaviors such as grooming and foraging (Loehle 1995; Webster et al. 2014). It is also suspected that CPT may contribute to a higher rate of predation, as severe cases were often seen to be isolated and not seeking shelter (Banks 2001). It would be valuable to conduct a follow-up

study, incorporating a control group free from clinical signs of CPT, to confirm that CPT is indeed associated with these comorbidities within the affected cohort.

Two additional limitations should be considered when interpreting the results of this study. Firstly, the selection criteria of animals for this study were based on clinical signs consistent with the disease rather than definitive diagnosis (which requires euthanasia and histopathological examination). We were therefore unable to rule out differential diagnoses that might overlap in the neurological presentation with CPT, such as toxoplasmosis (Parameswaran et al. 2009) or *Rhododendron* spp. toxicity (Hough 1997), which may contribute to misclassification bias. We also could not rule out differential causes of death or comorbidities for animals that were found dead in the field, such as capture myopathy (Breed et al. 2019), as the carcasses were all too severely damaged to provide any diagnostic value. This was mitigated by ensuring that euthanized kangaroos had histopathological diagnosis confirmed by a pathologist when possible. Secondly, we euthanized a large proportion of animals that were found at the severe stage of disease rather than allowing a natural death; this artificially shortened the survival duration or reduced the survival rate. This was guided by ethical considerations.

It is evident that CPT can be a significant health and welfare concern in some free-ranging kangaroo. Routine surveillance of high-risk populations should be implemented, especially during the high-risk months of April–September in regions such as Victoria, Australia. Euthanasia of individuals showing moderate to severe signs during this time period is justified given the high mortality rate and probable poor welfare associated with the long duration of clinical signs. Animals showing mild clinical signs are at less risk of poor welfare, but should still be closely monitored for deterioration, especially during the early high-risk period of April, May, and June. The animal health impacts of CPT demonstrated by this study highlight the importance of developing disease management strategies to reduce the risk of CPT for wildlife.

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SUPPLEMENTARY MATERIAL

Supplementary material for this article is online at http://dx.doi.org/10.7589/JWD-D-23-00168.

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