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EFFECTS AND TREATMENT OF SARCOPTIC MANGE IN SOUTHERN HAIRY-NOSED WOMBATS (*LASIORHINUS LATIFRONS*)

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ABSTRACT: We examined the clinical and cellular effects of sarcoptic mange on southern hairy-nosed wombats (SHNW, *Lasiorhinus latifrons*) and the effectiveness of a single dose of ivermectin as a treatment for captive and wild animals. Wambats were caught at three sites in South Australia between April and August 2005 and blood and skin samples were collected. Hematology, biochemistry, and protein electrophoresis reference intervals were determined for healthy and diseased SHNW. Diseased SHNW had significantly higher white blood cell counts, neutrophils, lymphocytes, and total protein but lower red blood cell counts, hemoglobin, hematocrit, and creatinine. Microscopic investigation indicated substantial hyperplasia, hyperkeratosis, and fluid infiltration into the dermis and epidermis of diseased animals. Conclusions on the efficacy of a single dose of ivermectin were limited by low sample size ($n=5$, two captive and three wild SHNW) and are preliminary. However, ivermectin effectively treated mild, but not severe, mange in wild SHNW and severe mange in captive animals. This study has implications for the conservation and management of SHNW and the broader Vombatidae family.

Key words: Ivermectin, *Lasiorhinus latifrons*, mange, *Sarcoptes scabiei*, wombat.

INTRODUCTION

Sarcoptic mange, a cosmopolitan disease of mammals caused by infestation of the pathogenic mite *Sarcoptes scabiei* (Acari: Sarcoptidae), is prevalent in humans, domestic animals, and an increasing range of wildlife species (Daszak et al., 2000). Among Australian wildlife, it is best known from the family Vombatidae, in which it is the major infectious disease (Hartley and English, 2005). In common wombats (*Vombatus ursinus*), mange is both enzootic and epizootic, and outbreaks can cause significant mortality and substantial decreases in local abundance (Skerratt, 2003b). Studies in this species have examined the behavioral, hematologic, clinical, and epidemiologic effects of mange and briefly explored treatment options (Skerratt et al., 1999, 2004; Skerratt, 2003a, b). In contrast, mange in southern hairy-nosed wombats (SHNW, *Lasiorhinus latifrons*) is poorly studied, most likely because until recently the disease was considered relatively uncommon (Martin et al., 1998; Skerratt et al., 1998). However, both anecdotal and scientific evidence now suggest that mange can cause significant morbidity and mortality in SHNW (Ruykys et al., 2009). The disease is of conservation concern given that SHNWs have a patchier distribution than do common wombats (Van Dyck and Strahan, 2008). Mange has not been confirmed in the critically endangered northern hairy-nosed wombat (*Lasiorhinus krefftii*, NHNW, Bryant and Reiss, 2008).

To better manage mange in SHNW and prevent its spread to NHNW, managers require an understanding of the disease at the host genus level as well as information on treatment options. Ideally, outbreaks would be prevented by curtailing mite transmission, which most likely occurs when wombats frequent burrows that have previously been used by infected red foxes (*Vulpes vulpes*), dogs (*Canis familiaris*), or other wombats (Skerratt et al., 2002). However, because there is currently no method for fumigating burrows, treating wombats with an acaricide is the most viable alternative (Skerratt, 2001).

One option is ivermectin, which has been successfully used to treat mange in a range of domestic and wild species (Pence and Ueckermann, 2002). The drug can be applied orally, subcutaneously (SC), or topically and works by interrupting parasites' nerve impulses, causing paralysis and death (Victoria and Trujillo, 2001). A course of treatment (two to three treatments 10 days apart) is preferable because it ensures that the larvae that emerge from the relatively drug-resistant ova are also killed (Curtis, 2004). However, difficulties in both recapturing and holding wild individuals in captivity usually make such courses impractical. It was thus necessary to establish the efficacy of one treatment of ivermectin in SHNW.

The life-cycle of *S. scabiei* is completed within 10–14 days (Mellanby, 1944), so if a single dose provides acaricidal blood levels for this length of time, it could clear the infection. Single doses of ivermectin have been effective when administered orally to humans (Meinking et al., 1995) and administered SC to camels (*Camelus dromedaries*), pigs (*Sus scrofa*), dogs, and cattle (*Bos taurus*; Burgess, 1994). However, a single dose failed to treat mange in red foxes (Newman et al., 2002) and gorillas (*Gorilla gorilla beringei*; Graczyk et al., 2001). In captive common wombats, Skerratt et al. (2004) found that one injection (0.4 mg/kg) cleared mild mange but that retreatment was required for the most-severely diseased animals. A pharmacokinetics study of another macrocyclic lactone, moxidectin, suggested that a single injection at 0.2 mg/kg would not clear mange in SHNW (Death et al., 2011). To further our understanding, our aims were to establish the clinical and cellular effects of mange in SHNW and determine the effectiveness of a single dose of ivermectin for treating infected wild and captive animals.

MATERIALS AND METHODS

Southern hairy-nosed wombats were caught on three pastoral properties in the Murraylands region (34°55′S, 139°28′E) of South Australia (SA), between April and August 2005, using the methods of Ruykys et al. (2009). Briefly, animals were captured using a "stunning" technique (Taggart et al., 2003) and anesthetized with zolazepam/tiletamine (3 mg/kg intramuscular, Zoletil® 100H; Virbac Australia Pty. Ltd., Peakhurst, New South Wales, Australia). Wombats' sex, body condition, and morphometrics were recorded and microchips (Texas Instruments Pty. Ltd., Dallas, Texas, USA) implanted at first capture. To establish if animals had mange, skin scrapings were examined for *S. scabiei* mites, larvae, or eggs. This involved moistening a wombat's skin with water or paraffin oil and, using a scalpel, scraping the epidermis deeply enough to just bring blood. Scrapings were macerated in 5% potassium hydroxide and examined under 40× magnification (Skerratt, 1998). For infected animals, erythema, parakeratosis, and alopecia were each quantified for the head, neck, abdomen, flank, and back according to the categories of none, slight (1–33%), moderate (34–66%), or severe (>66%). The average from the five body sites was used to allocate wombats to an overall disease category of mild, moderate, or severe.

Blood samples were collected from adult, infected SHNW that were caught for the first time, those that were recaptured, and from a sample of healthy individuals. Repeat samples from individuals were excluded from the analyses and a maximum of 22 individuals were sampled for any one parameter. Blood was collected from the femoral vein using a 22-gauge needle and placed in a 1-mL ethylenediaminetetraacetic acid (EDTA) tube and 4 mL plain blood tube. The EDTA tubes were centrifuged at 1,200 × G for 15 min and the plasma separated into blood tubes for protein electrophoresis and biochemical analyses. All samples, including the plain blood tubes for hematology, were refrigerated for a maximum of 3 days before being analyzed at a commercial laboratory (Gribbles Pathology Pty. Ltd., Adelaide, Australia). Comparisons between healthy and diseased wombats were completed using SPSS (ver. 19, IBM Corporation, Armonk, New York, USA). Student's *t*-tests (Blalock, 1972) were used for parametric data and Mann-Whitney *U*-tests (Dineen and Blakesley, 1973) for nonnormal or heteroscedastic data. Assessing differences between animals of different sexes and mange severity was beyond the context of the study; however, these factors may have affected the results. Reference intervals were generated using the technique recommended by Solberg (1987). If significant differences existed between healthy and diseased animals, intervals were generated

for both groups; otherwise, results were combined.

The cellular response of the dermis to mange was determined by light microscopy. Using a leather hole-puncher, skin samples were taken from the ears of both infected and healthy wombats and stored in ethanol. These samples, together with pieces of parakeratotic crust stored in 10% formalin, were dehydrated, embedded in paraffin wax, sectioned at about 7 μm, and stained with hematoxylin and eosin (Skerratt, 2001). Photographs were taken using an Olympus Camedia F1.8 C-5050 zoom camera (Olympus, Mt. Waverly, Victoria, Australia).

In September 2004, a trial with a single dose of ivermectin (Ivomec, Merck Sharp & Dohme, South Granville, New South Wales, Australia) was conducted on a captive SHNW with severe mange. The female was caught at the study site, taken to Adelaide Zoo, SA, treated SC (0.2 mg/kg), and anesthetized irregularly over 7 wk for examination (body weight and skin scrapings). Upon full recovery, the animal was rereleased to the wild. We describe this initial trial as well as results from June 2005, when a male SHNW with severe mange was brought into Adelaide Zoo and treated SC with ivermectin (0.2 mg/kg). This animal was anesthetized every 7 days for 10 wk for weighing and skin scrapings. The scrapings were examined for mite presence or absence and alive or dead status.

Field trials of ivermectin were also conducted. Specifically, seven of the infected SHNW that were captured were treated (SC, 0.2 mg/kg) and six were untreated (controls). Recaptured wombats that were part of the ivermectin trial had the severity of erythema, parakeratosis, and alopecia reassessed across their five body parts. On the final fieldtrip, all captured SHNW were treated.

RESULTS

Sixty-seven individual SHNW were caught and 17 (25.4%) had mange: eight mildly, one moderately, and eight severely (excludes recaptures).

Clinical pathology

There were significant differences between healthy and diseased SHNW in 12 parameters (Table 1). Diseased wombats had lower hemoglobin, hematocrit, bicarbonate, creatinine, red blood cell counts, and albumin:globulin ratios but higher white blood cell (WBC) counts, neutrophil, lymphocyte, alanine aminotransferase, total protein, and globulin values. Healthy SHNW occasionally had hypersegmented but otherwise normal neutrophils and their platelets were aggregated and adequate. In contrast, SHNW with mild mange had no abnormality or a mild increase in polyclonal gamma globulins, while severely infected animals had marked increases in polyclonal gamma globulins and mild to moderate neutrophilia. Nucleated red blood cells, elevated platelets, and mild hemoconcentration were other findings for severely infected animals.

Cellular response

The cellular response was characterized by inflammation in the dermis and epidermis, with the most obvious changes occurring at the stratum corneum. In healthy SHNW, the structure of the stratum corneum was smooth, with no hyperplasia or disruption to several layers of flat, keratinized, a-nucleated cells. In contrast, the stratum cornea of diseased SHNW exhibited significant hyperplasia, hyperkeratosis, and edema. Thick parakeratotic scabs of keratin, neutrophilic debris, bacteria, *S. scabiei*, and their debris also occurred. Parakeratosis presented as both an increased number of keratinocytes around burrowing mites and the presence of nucleated keratinocytes. Atrophy of hair follicles and sebaceous glands was common.

The stratum granulosum of healthy SHNW was also continuous and smooth and the stratum spinosum had only small dermal papillae. In contrast, diseased wombats had substantial increases in the number and depth of connective tissue papillae at the dermal-epidermal interface. The cells involved were mainly lymphocytes, eosinophils, neutrophils, and mast and plasma cells.

Treatment trials

Two severely infected SHNW (one female in 2004, one male in 2005) were

TABLE 1. Comparison of clinical pathology parameters (means±SD and reference intervals) for healthy and mange-infected southern hairy-nosed wombats (*Lasiorhinus latifrons*) in the Murraylands, South Australia. If differences between the two groups were not significant, data were combined.

Variable[a]	Category	*n*	Mean±SD	Reference	*P* value
General hematology					
Red blood cells ($\times 10^{12}$/L)	Healthy	8	5.2±0.5	4.7–6.3	0.02
	Diseased	10	4.3±0.8	3.0–5.4	
Hemoglobin (g/L)	Healthy	8	136.2±14	120–161	0.01
	Diseased	10	113.2±20	87–144	
Hematocrit (%)	Healthy	8	43±5	0.4–0.5	0.01
	Diseased	10	0.35±0.07	0.3–0.4	
MCV (fl)[a,b]	Combined	18	82.0±2.8	79–88	0.3
MCH (pg)[a]	Combined	18	26.5±1.2	25–31	0.6
MCHC (g/L)[a]	Combined	18	321.6±11.4	302–349	0.2
White blood cells ($\times 10^9$/L)	Healthy	8	6.2±6.4	0.6–20.8	0.03
	Diseased	10	14.2±7.6	4.4–27.1	
Neutrophils ($\times 10^9$/L)[b]	Healthy	8	4.7±5.7	0.3–17.7	0.02
	Diseased	10	10.6±6.5	3.2–21.1	
Lymphocytes ($\times 10^9$/L)[b]	Healthy	8	1±1.2	0.2–3.7	<0.01
	Diseased	10	2.6±1.7	1–5.1	
Monocytes ($\times 10^9$/L)	Combined	18	0.5±0.4	0–1.5	0.5
Eosinophils ($\times 10^9$/L)[b]	Combined	13	0.32±0.58	0–2.2	0.1
General biochemistry					
Sodium (mmol/L)[b]	Combined	22	137.5±2.8	134–146	0.3
Potassium (mmol/L)	Combined	22	7.5±1.7	4.2–10.4	0.8
Chloride (mmol/L)[b]	Combined	11	94.8±3.6	88–99	0.2
Bicarbonate (mmol/L)[b]	Healthy	5	42.8±6	33–49	0.03
	Diseased	6	34.3±4.6	28–40	
Na/K	Combined	22	19.4±5.1	13.1–34.8	0.9
Urea (mmol/L)[b]	Combined	22	10.1±3.4	4.3–20.0	0.051
Creatinine (mmol/L)[b]	Healthy	11	0.19±0.06	0.07–0.29	<0.01
	Diseased	11	0.097±0.03	0.06–0.17	
Calcium (mmol/L)	Combined	11	2.5±0.1	2.3–2.8	0.4
Phosphate (mmol/L)	Combined	11	1.9±0.7	0.8–3.0	0.5
Total bilirubin (umol/L)[b]	Combined	11	2.4±1.1	1–5	0.3
Alkaline phosphatase (U/L)[b]	Combined	22	214.4±144.4	60–628	0.3
Alanine aminotransferase (U/L)[b]	Healthy	11	46.7±23.6	24–106	<0.01
	Diseased	11	125.4±74	37–260	
Gamma glutamyl transferase (U/L)	Combined	11	27.4±23.6	4–74	0.6
Aspartate aminotransferase (U/L)	Combined	22	38.9±17.8	17–68	0.2
Creatine phosphokinase (U/L)	Combined	11	482±368	53–1197	0.4
Lactate dehydrogenase (U/L)	Combined	11	443.1±104.6	275–610	0.8
Cholesterol (mmol/L)	Combined	11	3.0±0.6	2.3–4.4	0.1
Protein electrophoresis					
Total protein (g/L)	Healthy	11	63.5±4.6	52–69	<0.01
	Diseased	11	71.2±6.8	60–85	
Albumin (g/L)	Combined	22	34.7±5.9	22–43	0.3
Globulins (g/L)	Healthy	11	27.4±5.3	20–37	<0.01
	Diseased	11	37.9±9	24–53	
A:G ratio[a]	Healthy	11	1.4±0.3	0.96–1.9	0.02
	Diseased	11	1.1±0.3	0.59–1.5	

[a] MCV = mean corpuscular volume; MCH = mean corpuscular hemoglobin; MCHC = mean cell hemoglobin concentration; A:G ratio = albumin:globulin ratio.

[b] Compared using Mann-Whitney *U*-test.

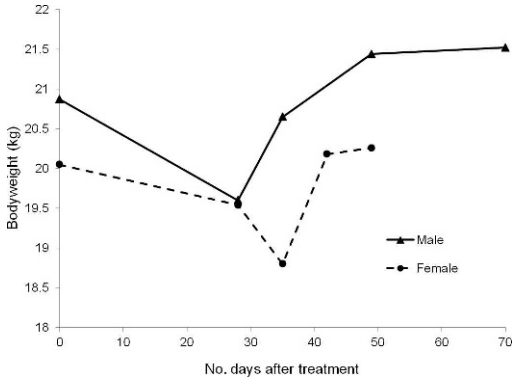

FIGURE 1. Body weight changes for two southern hairy-nosed wombats (*Lasiorhinus latifrons*) from the Murraylands of South Australia that had severe mange and which were treated with a single dose of ivermectin at time 0.

involved in the captive trials. A single dose of ivermectin cleared each animal's infection and both exhibited a similar pattern of recovery. Although both animals initially had large numbers of live *S. scabiei*, by 14 days after treatment (DAT), there was a 75–90% reduction in mite abundance. Live mites only persisted up to 28 DAT, by which time parakeratotic scales were lifting from the wombats' heads and flanks and hair regrowth was evident over the whole body. By 42–70 DAT, there was fur regrowth of up to 2 cm, no erythema or parakeratosis, and only 0–2% of the original abundance of *S. scabiei*, all of which were dead. Both individuals also showed similar changes in body weight (Fig. 1); initial decreases culminating in minimums by 28–35 DAT, followed by an increase.

In the field trials, two treated and one control SHNW were recaptured. Both treated individuals, one of each sex, initially presented with mild mange. By 70–98 DAT, both had fully recovered with no alopecia, parakeratosis, or erythema and either no, or no live, mites. The female's infection did not manifest hematologically, with normal values reported at both the original capture and the recapture. In contrast, at original capture, the male presented with an increase in polyclonal gamma globulins. By recapture at 98 DAT, gamma globulins had fallen to within the species' reference intervals and red blood cell counts and platelets were normal.

The third wild SHNW was initially involved in the trial as a control. The animal was a 25.1-kg male that, at first capture, had moderate mange. Mild macrocytosis and hypersegmented neutrophils were observed in the blood sample from this time. At first recapture, 27 days after initial capture (DAI), the infection had progressed to being severe. By the second recapture (69 DAI), a 13% (3.3 kg) decrease from the original body weight and severe deterioration in clinical signs prompted treatment on welfare grounds. Clinical pathology from this time showed increases in the WBC count, hemoglobin and bicarbonate values, and a progression from mild to moderate cholangiohepatopathy. The third recapture (100 DAI, 31 DAT) occurred by hand after the animal was sighted grazing during the day. The animal died during this last capture. Examination showed a 30% (7.6 kg) loss of body weight since the initial capture and extensive flea infestation. There was also severe parakeratosis on the front of the body including the chest, flanks, head, and anterior back. However, on the posterior back, there was almost full fur and the flaking skin that is symptomatic of mange recovery.

DISCUSSION

Clinical pathology

We present the first published hematologic values of SHNW with mange and provide a critical baseline for management and future research. However, since some results may have been influenced by small sample size, caution is warranted. As Solberg (1987) suggests that at least 40 values be used to make reference intervals, those presented in the current study require verification. Nevertheless, because the results for healthy SHNW generally

correlate well to those previously established for SHNW (Bryant and Reiss, 2008), our intervals are likely to be broadly reliable.

Diseased SHNW had significantly lower red cell mass (red blood cell counts, hemoglobin, and hematocrit) than did healthy animals. If all three parameters are below the range defined for a species, the individual in question is deemed to be anemic (Clark, 2004). Although the values in this study were within the ranges previously reported for SHNW (Gaughwin and Judson, 1980), the decreases do suggest a trend towards anemia, a condition which has been associated with mange in European rabbits (*Oryctolagus cuniculus*), pigs, dogs, and common wombats (Arlian, 1989; Löwenstein et al., 1996; Skerratt et al., 1999).

The fact that diseased SHNW had elevated WBC counts and, more specifically, higher neutrophil and lymphocyte values is explicable by the fact that these cell types control inflammation and initiate tissue defense (Clark, 2004). Elevated neutrophils are indicative of bacterial infection in organs (Skerratt et al., 1999) or secondary infections in skin fissures (Hartley and English, 2005), and rises have been recorded in mangy dogs (Arlian et al., 1996), cattle (Löwenstein et al., 1996), rabbits, foxes, and common wombats (Skerratt et al. 1999). Similarly, lymphocytosis is associated with chronic infection (Clark, 2004), and the rise observed in diseased SHNW concurs with results for mange-infected humans (Morsy and Gaafar, 1989), dogs (Arlian et al., 1997), and cattle (Löwenstein et al., 1996). In contrast, Skerratt et al. (1999) and Hartley and English (2005) found that mangy common wombats had lower lymphocyte values than did healthy animals. Other stressors or variation in infection severity, extent, and duration may explain the discrepancy between the two species. For eosinophils, the finding of no statistical difference between diseased and healthy SHNW requires validation, particularly given that eosinophilia was noted in the current study's written reports and has been observed in mange-infected humans (Arlian, 1989), foxes (Little et al., 1998), and common wombats (Hartley and English, 2005).

Biochemical analyses indicated that diseased SHNW had lower bicarbonate values than did healthy animals. Bicarbonate regulates the pH of blood by buffering carbon dioxide (CO_2); increases in CO_2 thus lead to increases in bicarbonate. Because CO_2 levels are proportional to metabolic rate, bicarbonate can be used as an indicator of metabolism (Burtis and Ashwood, 1998). The depressed value suggests that, on average, diseased SHNW had lower metabolic rates than did healthy animals, perhaps due to reduced metabolic efficiency or energy intake.

Diseased SHNW also had lower creatinine values. This waste molecule is generated in relatively constant amounts during muscle metabolism; thus, depressed values suggest a decrease in muscle mass, a common side-effect of chronic disease (Cantarow and Schepartz, 1962). This result concurs with the fact that severely diseased SHNW at the study site were, on average, 9.9 kg lighter than healthy wombats (Ruykys et al., 2009). Decreases in creatinine have also been noted in mangy coyotes (*Canis latrans*; Pence et al., 1983), European rabbits (Arlian, 1989), and common wombats (Hartley and English, 2005).

Diseased SHNW also had elevated levels of alanine aminotransferase (ALT). These enzymes occur at high concentrations in liver cells and, if the liver is damaged, leak into the blood stream (Jacobs, 1993). Hartley and English (2005) found no significant difference in ALT between diseased and healthy common wombats. However, mange or secondary infections may lead to liver cirrhosis (Skerratt, 2001) and our results suggest the beginning of such in some of the diseased SHNW. Postmortem histopathology would be useful for confirming liver cirrhosis.

Protein electrophoresis indicated that diseased SHNW had elevated total protein values; this was due to an increase in globulins. Globulins are associated with chronic infection and immune-mediated diseases (Wilkins, 1979) and, together with the observed lymphocytosis, indicate that diseased SHNW were mounting an immune response, possibly to the mites. In coyotes, Pence et al. (1983) found that globulin levels increased commensurate with mange severity.

Cellular response

Infected SHNW had a similar cellular response to that of common wombats (Skerratt, 2005) including epidermal thickening, parakeratosis, and atrophy of hair follicles. In both species, the most pronounced effect was the infiltration of WBC, especially into the rete ridges of the dermis and in the epidermis close to the progressing mite. Although our study was limited to staining with hematoxylin and eosin, results indicated that lymphocytes and eosinophils were the most abundant cells in the infiltrate. The presence of these cell types suggests that, as with common wombats, both Type 1 (immediate) and Type IV (delayed) hypersensitivity reactions develop in SHNW (Skerratt, 2003b). Skerratt (2003a) suggested that this acquired immune response could be responsible for the more limited mite population growth that occurs when common wombats are reinfected with mange. Although we did not conduct reinfestation trials, the delayed hypersensitivity response indicates that this may also be the case for SHNW.

Treatment trials

Because treatment trials were limited by low sample size ($n=5$, consisting of two captive and three wild SHNW), our results are preliminary. However, they suggest that one dose of ivermectin may clear severe (and presumably also mild and moderate) mange in captive SHNW and mild mange in wild SHNW. Given the partial recovery of the severely diseased wild SHNW, further investigations for this group are required. The discrepancy between wild and captive severely diseased SHNW may be related to the fact that captive animals are in a more amenable environment (warmer, with food and water ad libitum) and not consistently re-exposed to mites. In the field trials, mortality may have contributed to the low recapture rate of treated individuals (two recaptured of seven initially treated animals).

The timeline for the resolution of clinical signs in the captive SHNW treated with a single dose of ivermectin correlated closely with that of common wombats treated with three doses (Skerratt, 2003b). In both species, parakeratotic scales started to lift within 7 DAT and skin fissuring had regressed by 20 DAT. There was also substantial lifting of scales by 20 DAT and complete removal by 63–70 DAT, with hair regrowth evident by 28 DAT. The approximately 5% loss in body weight observed 28–35 DAT in the captive SHNW could be attributable to the time required for acclimatization to captivity and recovery from disease. In contrast, Skerratt et al. (2004) found that wild common wombats had an average 0.6 kg weight increase by 37 DAT.

Management implications

Our results provide preliminary support for the efficacy of a single dose of ivermectin for treating mild, moderate, and severe mange in captive SHNW as well as for mild infections in wild animals. Larger sample sizes, more study sites, and pharmacokinetic studies are required to corroborate this finding. Ivermectin is a commonly available drug, and verifying the efficacy of one dose could lead to its use during epizootics in SHNW and common wombats and in areas where the disease is enzootic. This would be particularly critical should mange spread to small, endangered SHNW populations or to NHNW.

While increasing the dose from that used in the current trials is feasible, the

possibility of drug toxicity must be considered. For example, Skerratt (2001) even reported some neurologic effects in common wombats treated at the dose recommended for domestic animals. With severely diseased wild wombats it may, therefore, be safer to combine a single treatment of ivermectin with a topical, pour-on acaricide or removal of parakeratotic scale, although the possibility of adverse drug interactions should be considered (Skerratt, 2001). Washing wombats with an insecticide is also effective (Ruykys et al., 2009); however, this option is only viable when managing individuals and, even then, wetting animals may not be desirable.

Our results and investigations in common wombats (Skerratt, 2001) suggest that infected wombats undergo an acquired immune response. Consequently, the feasibility of vaccinating animals with an antigen from *S. scabiei* should be investigated. Further research into the course of infection and treatment of mange in the Vombatidae is also critical, especially given that wombat populations are already being negatively impacted by a range of threats including habitat loss, drought, and both deliberate and accidental destruction by humans.

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