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Demodectic mange in threatened southern sea otters (*Enhydra lutris nereis*)

Nadia Javeed*, Janet Foley*, Arturo Oliver-Guimera†, Verena K. Affolter†, Michael K. Keel†, Angelina Reed‡, Risa Pesapane*, Pádraig J. Duignan§, Michael Murray††, MT Tinker and Melissa A. Miller‡¶**

*Departments of Medicine and Epidemiology, School of Veterinary Medicine, University of California Davis, Davis, CA 95616, USA
†Departments of Pathology, Microbiology, and Immunology, School of Veterinary Medicine, University of California Davis, Davis, CA 95616, USA
‡Marine Wildlife Veterinary Care and Research Center, California Department of Fish and Wildlife, 151 McAllister Way, Santa Cruz, CA 95060, USA
§The Marine Mammal Center, 2000 Bunker Road Fort Cronkhite Sausalito, Sausalito, CA 94965, USA
¶Karen C. Drayer Wildlife Health Center, 1089 Veterinary Medicine Drive, Davis, CA 95616, USA
**Nhydra Ecological Research, 11 Parklea Drive, Head of St Margarets Bay, Davis, NS B3Z 2G6, Canada
††Monterey Bay Aquarium, 886 Cannery Row, Monterey, CA 93940, USA

Correspondence: Janet Foley, 1320 Tupper Hall, Davis, CA 95616, USA. E-mail: jefoley@ucdavis.edu

Background – Southern sea otters (*Enhydra lutris nereis*) rely on intact pelage for thermoregulation, and thus clinically significant demodicosis and associated alopecia can cause morbidity and death.

Hypothesis/Objectives – This study aimed to describe lesions associated with follicular *Demodex* sp. infestation, estimate the prevalence and intensity of infestation, describe mite distribution across key anatomical regions, and assess mite presence or absence in relation to lesions and host risk factors.

Animals – Twenty necropsied, wild southern sea otters that stranded along the central California coast from 2005 to 2018.

Methods and materials – Grossly normal and abnormal integument from the head, perineum, genitals, mamillary papillae and limbs was assessed microscopically for mites and mite-associated pathological findings.

Results – Intrafollicular mites were observed in the integument of 55% of otters and 20% had clinical demodicosis. Demodicosis was considered to be contributory to death or euthanasia in two cases. Although *Demodex* sp. mites often were observed microscopically in grossly normal skin, the presence of multiple densely-packed intrafollicular mites generally was associated with pigmentary incontinence, ectatic follicles, lymphoplasmacytic perifolliculitis, and neutrophilic and lymphoplasmacytic, dermal inflammation. Other findings included epidermal hyperplasia, orthokeratotic hyperkeratosis of epidermis and follicular epithelium, concurrent pyoderma and cell necrosis. Perioral integument, especially of the chin, had the highest prevalence of mites and the highest mite density, suggesting facial contact as a means of mite transmission.

Conclusions and clinical importance – Our research confirmed demodectic mange as a contributor to morbidity and mortality in sea otters, with important implications for clinical care, rehabilitation and conservation.

Introduction

Mites of the genus *Demodex* (Acari: Demodicidae) inhabit the skin of virtually all mammals.[1] Although morphologically similar, these mites are highly host-specific, inhabiting hair follicles or sebaceous glands and feeding on sebum or epithelial cells.[2,3] Local or generalized mite proliferation in the skin of the host has been reported in dogs, cats, cattle and wildlife.[4–9] Associated microscopic lesions include folliculitis, pigmentary incontinence, epidermal hyperplasia, hyperkeratosis, furunculosis and perifollicular granulomas,[10–15] often exacerbated by secondary or concurrent bacterial or fungal infections.[11,16,17] The pathogenesis of clinically significant demodicosis is poorly understood, although it is postulated to be associated with deficient immune responses.[18]

Among aquatic species, *Demodex* spp. infestation and/or lesions have been reported in California sea lions (*Zalophus californianus*),[16,19] harbour seals (*Phoca vitulina*)[14,20] and European otters (*Lutra lutra*).[21] Lesions include scaling, alopecia and deep ulcers.[14,16] Most descriptions are individual case reports, so little is known about the prevalence and population-level impacts of *Demodex* spp. among marine mammals including southern sea otters (*Enhydra lutra nereis*). Southern sea otters are listed as threatened under the US Endangered Species Act, and their population has been slowly recovering from near-extinction with

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disease remaining an important threat.[22] Because sea otters have high energetic requirements[23] and depend on an intact, healthy and dense pelage for thermoregulation,[24] *Demodex*-associated skin pathological consequences may contribute to morbidity and death.

We examined skin samples from 20 stranded southern sea otters in central California from 2005 through 2018. Our goals were to describe *Demodex* sp. distribution and density across key anatomical regions, estimate the prevalence of mite infestation in southern sea otters, and assess mite presence in relation to defined lesions and host risk factors. We also characterized the mites morphologically and using DNA sequencing.

Methods and materials

Sample collection and necropsy

Samples were collected during necropsies of 20 wild southern sea otters that stranded along the central California coast from 2005 through 2018. After combing the pelage to assess the predominant direction of hair growth, approximately 4 cm × 2 cm rectangular strips of haired skin were collected from defined anatomical locations. To facilitate tissue processing and mite detection, the long axis of each sample was orientated in the direction of hair growth; this optimized longitudinal sectioning of the compound hair follicles.

Skin samples were collected from perioral, perinasal, periaural and periocular regions, plantar aspects of the rear flippers, plantar surfaces of the paws, perivaginal and mammary peripapillary skin in females, and peripreputial and scrotal skin in males. Other areas with skin lesions also were sampled. Where possible, paired samples were collected from each site: one frozen at –20°C for PCR and the other fixed in 10% neutral buffered formalin for histopathological evaluation. Formalin-fixed integument was trimmed along the longest axis, paraffin-embedded, sectioned at 5 μm, and stained with hematoxylin and eosin. Section recuts or up to six sequential, 5 μm thickness step-sections were examined in some blocks to assess the value of this technique for facilitating mite detection and enumeration.

Microscopic examination and scoring

Two examiners scored each section independently, with discrepancies between evaluators resolved via joint slide review and discussion. All samples were evaluated for adequate representation and orientation of hair follicles, and presence or absence, number and anatomical location (e.g. follicular lumen, sebaceous gland duct, or both) of *Demodex* mites. Mite presence/absence and approximate density (mites/cm of basement membrane) were recorded in relation to the number of otters sampled for a given anatomical site and the number of sections examined (including recuts and step-sections).

Lesion coding criteria, summarized in Appendix S1 in the supporting information, included: pattern of inflammation (follicular, perivascular, periadnexal, perivascular or diffuse), dominant inflammatory cell type (lymphoplasmacytic or histiocytic) and severity of infiltrate (none, mild, moderate or severe). Additional features recorded were: presence/absence and severity of perifollicular pigmentary incontinence, ectatic follicular luminae, follicular hyperkeratosis, furunculosis with visible dermal keratin debris, erosions or ulcers, acanthosis and hyperkeratosis (orthokeratotic or parakeratotic). Also, presence or absence of bacteria, approximate bacterial number, bacterial morphology (cocci, rods, coccobacilli, mixed) and anatomical location were recorded, excluding microbes that were considered postmortem colonizers.

In order to standardize findings in relation to tissue length, slides were digitally scanned using a VS120 slide scanner (Olympus; Tokyo, Japan), and the length of the basement membrane was estimated for each section using Olympus OlyVIA software (v2.8).

DNA sequencing

Transverse 25 mg slices were collected from frozen skin with a clean razor blade for DNA extraction using the DNeasy Blood and Tissue kit (Qiagen; Redwood City, CA, USA). DNA also was extracted from 5–10 μm thickness scrolls from five paraffin blocks with high mite density (≥10 mites per tissue section on histopathological evaluation) using a QIAamp DNA FFPE kit (Qiagen). An internally transcribed spacer (ITS)1–2 fragment of rDNA was amplified using previously published primers: forward 5′-AGAGGAAGTAAAAGTCGTAACAAG-3′ and reverse 5′-ATATGCTTAAATTCAGGGGG-3′[25] in a 25 uL PCR reaction containing GoTaq Green Master Mix (Promega; Madison, WI, USA). Cycling conditions were: 95°C for 2 min followed by 35 cycles of 94°C for 30 s, 54°C for 30 s and 72°C for 2 min, with a final extension step of 7 min at 72°C. PCR products were visualized on a 1% agarose gel and purified using ExoSAP-IT (ThermoFisher; West Sacramento, CA, USA) before sequencing on an ABI 3730 sequencer (Davis Sequencing; Davis, CA, USA). Sequenced amplicons were evaluated by Blast (NCBI; http://blast.ncbi.nlm.nih.gov/Blast.cgi).

Statistical methods

Summary data were compiled in Excel (Microsoft; Redmond, WA, USA) and analysed using R.[26] Fisher's exact tests were used to examine associations between mite presence/absence and sea otter sex, county of stranding and age class (pups, 0–6 months; subadults, 1–4 years; adults, >4–10 years; and aged adults, >10 years). The mean numbers of mites in mite-positive male and female otters of any age class were compared using a two-tailed Student's *t*-test. Fisher's exact tests also were used to assess whether mite presence/absence was associated with the lesions described above. Tests were considered significant if the *P*-value was <0.05.

Results

Clinical, gross and histopathological changes

Twenty southern sea otters that stranded from 2005 to 2018 in Marin, San Luis Obispo, Monterey or Santa Barbara counties on the central California coast were included (Table 1). Otters were found dead, died in care, or were humanely euthanized. The sample population consisted of 13 females and seven males, aged one week to 14 years, including one pup, four subadults, nine adults and six aged adults. One otter (7927-18) had been treated once with intranasal ivermectin (0.3 mg/kg) for nasopulmonary mite infestation 26 weeks before death; other animals had no history of acaricide therapy.

We assessed 173 skin sections across all anatomical sites from the 20 otters of which 53 were discarded owing to inadequate section quality or poor follicle orientation. A mean of five paraffin blocks were examined from each otter, most containing samples from multiple skin regions (Table 2, range one to 12 paraffin blocks/otter). Otters were not classified as mite-negative unless five to seven different anatomical sites were examined microscopically, often including more than one section/site.

Eleven otters (55%), including three subadults, four adults and four aged adults, were infested with *Demodex* (Table 1). Intrafollicular mites were observed in five males (71.43%) and six females (46.16%) with no sex predilection ($P = 0.37$). Approximately 64% of infested individuals were from Monterey County, and 34% were aged adults. The single pup was mite-free on histopathological evaluation. Neither age class ($P = 0.63$) nor the county of stranding ($P = 1.0$) were predictors of mite infestation.

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Table 1. Demographic data for 20 necropsied southern sea otters (*Enhydra lutris nereis*) that were examined microscopically for *Demodex* sp. mite infestation

Age class	Estimated age	Sex	County	Mite number
Pup	1–2 weeks	Male	Monterey	None
Subadult	3–4 years	Male	Marin	High†
Subadult	1–3 years	Female	Monterey	High
Subadult	Unknown	Female	Monterey	High
Subadult	Unknown	Female	San Luis Obispo	None
Adult	4 years	Male	San Luis Obispo	Low*
Adult	5–6 years	Female	Monterey	None
Adult	7–8 years	Female	Monterey	None
Adult	7–8 years	Female	Monterey	None
Adult	7–8 years	Female	Monterey	Low
Adult	8 years	Male	San Luis Obispo	None
Adult	4–6 years	Male	San Luis Obispo	High
Adult	5–7 years	Female	Monterey	None
Adult	Unknown	Female	Monterey	Low
Aged adult	12–14 years	Male	Monterey	High
Aged adult	14 years	Male	Santa Barbara	Low
Aged adult	10+ years	Female	Monterey	High
Aged adult	13–14 years	Female	Monterey	None
Aged adult	12–14 years	Female	Santa Barbara	None
Aged adult	12–14 years	Female	Monterey	Low

*Small number of mites, 0.44–1.2 mites/cm basement membrane.
†Large number of mites, >1.2 mites/cm basement membrane.

Table 2. Anatomical distribution and density of *Demodex* sp. mite infestation in integument from southern sea otters (*Enhydra lutris nereis*)

Anatomical site of sampled integument	Number of sections examined	Number of blocks examined	Number of otters examined	Number of otters positive for mites	Proportion of mite-positive otters	Mean mite density/cm basement membrane of mite-positive sections	Mean mite density for each mite-positive section	Maximum mites/ section
Perioral*	26	15	14	7	50%†	0.84	7.2	60
Perinasal	7	7	7	0	0%†	0	0	0
Periocular	11	8	8	0	0%	0	0	0
Periaural	15	12	10	1	10%†	4.1	22	30
Top of head	0	0	0	0	0%†	0	0	0
Front paw	14	12	9	0	0%†	0	0	0
Rear limb/ Flipper	12	11	10	1	10%†	8.5	47	47
Main body	2	1	1	1	50%	1	3	3
Mammary	1	1	1	0	0%	0	0	0
Perivulvar/ Perineal/ Periscrotal/ Preputial	7	7	4	1	25%	5.8	6	6
Tail	0	0	0	0	0%	0	0	0
Unknown anatomic location ‡	25	16	14	3	21%†	1.8	2.7	6

*Six of seven sections of integument from the chin were mite-positive, with a mean of 10.9 mites/section.
†Includes samples of grossly apparent skin lesions (alopecia, dermatitis, dermal hyperplasia and/or excoriation).
‡Some unknown sections may be from the ventral abdominal mid-line (a common sample site for sampling sea otter integument during pelting).

All intrafollicular mites were elongate and fusiform, with an average length of 164 ± 3.67 μm (Figure 1b). The scaled, semi-transparent bodies were composed of two fused segments with four pairs of short, stout appendages arising from the anterior body segment. Mites were orientated with their head towards the hair bulb. They were located in the follicular infundibulum near the opening of the sebaceous gland ducts (79%), within the ducts or the sebaceous gland (20%), or in the follicular isthmus (1%). In otters with a high mite density, up to seven *Demodex* mites were visible within each follicle, often clustered in adjacent follicles.

The most commonly infested locations were the face and head (73% of all mites seen), followed by the hind flippers (22%), prepuce (3%) and shoulders (2%). Perioral skin had the highest proportion of mite-positive sections and highest mite density occurred in chin with ≤60 mites/section (Table 2). For slides that lacked *Demodex* mites, subsequent tissue sections of the same paraffin blocks also were mite-negative (data not shown). The number of mites varied between 0.44 and 8.5 mites/cm of basement membrane with no significant difference ($P = 0.61$) between males [average 3.17 mites/cm; standard deviation (SD) 1.56] and females (average 3.80 mites/cm; SD 3.50).

Gross lesions were most common on the face and head, especially around the mouth, and were characterized by brownish-orange exudate, matted hair, orange discolouration of perilesional hair shafts (interpreted as salivary staining), alopecia and lichenification. A small number of papules and excoriations also were evident (Figures 2a–d, 3a–d).

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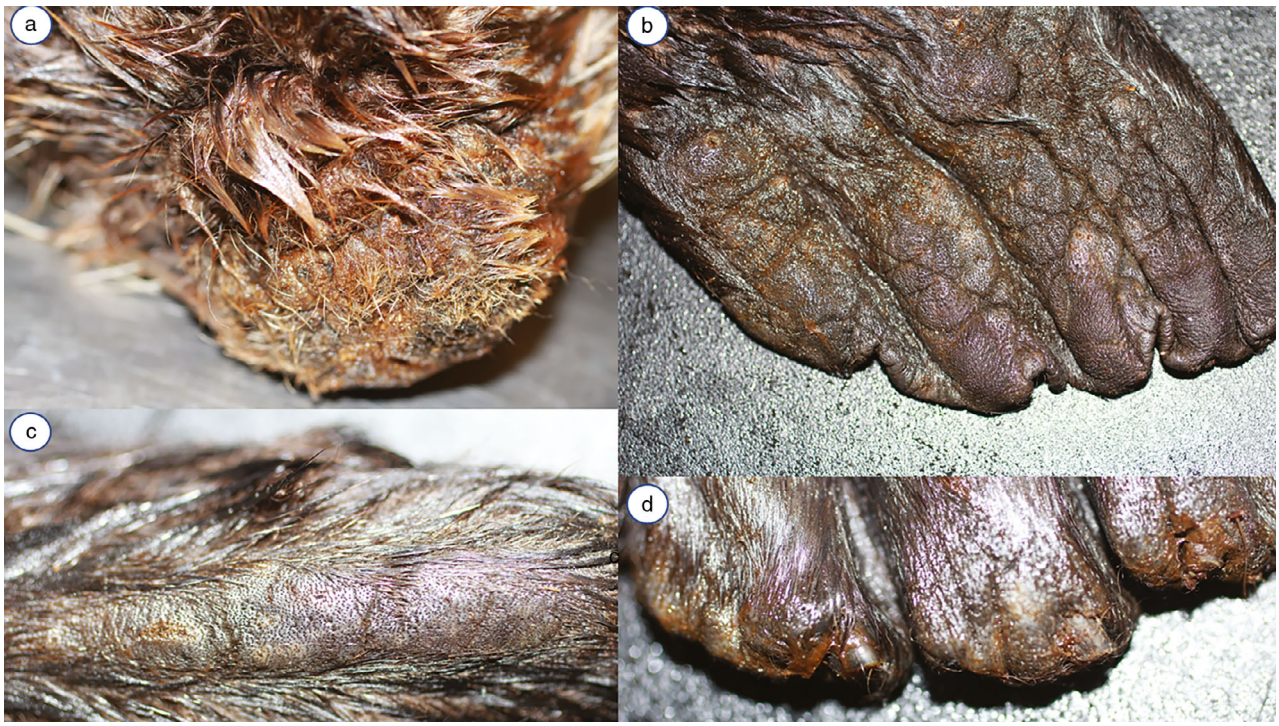

Figure 1. Demodicosis and bacterial pyoderma in an aged adult female southern sea otter (*Enhydra lutris nereis*: case 6734-13) (euthanized due to severe chronic dermatitis).
(a) Dorsal surface of the nose, showing marked dermal hyperplasia characterized by prominent ridges and deep tissue folds, plus moderate alopecia and patchy hypo/hyperpigmentation. The skin and surrounding hair are lightly coated with orange-brown viscous material (sebum, salivary staining and/or exudate from concurrent bacterial dermatitis). (b) Ventral flipper, showing chronic severe proliferative dermatitis, lichenification, alopecia, patchy hypo/hyperpigmentation and mild excoriation. Scant viscous orange-brown material is visible on the skin surface. (c) Dorsal tail, showing lesions similar to those on the flipper, with proliferative dermatitis, alopecia, patchy hypo/hyperpigmentation and surface excoriation. (d) Dorsal flipper, showing marked tissue thickening and blunting visible at the edge of each digit, along with patchy hypo/hyperpigmentation, excoriation and alopecia. The skin and hair are coated with moderate orange-brown viscous material.

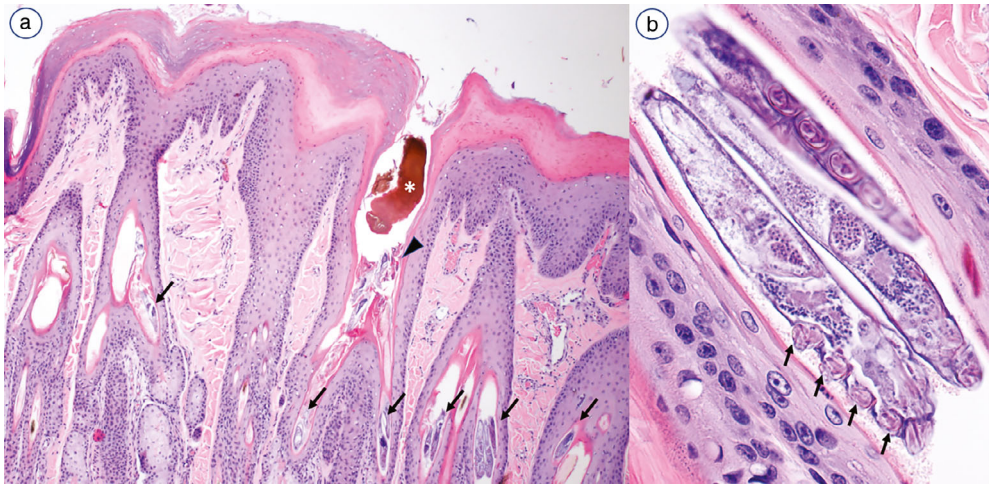

Figure 2. Follicular and surface epithelial histopathological findings for an aged adult female southern sea otter (*Enhydra lutris nereis*: case 6734-13) (euthanized due to severe chronic dermatitis).
(a) Haired skin from the left hind flipper, showing superficial and follicular hyperkeratosis. The follicular lumenae are variably dilated, and the ostium of one follicle is plugged with dark brown material (sebum: asterisk) and keratin debris (arrowhead). Follicles and associated sebaceous gland ducts contain numerous *Demodex* sp. mites (arrows). (b) Higher magnification view of a single follicle (the second follicle from the right in Figure 2a), showing longitudinal profiles of three cigar-shaped mites orientated head-down in the lumen. Four short, stout legs are visible on the anteroventral body surface of the lefthand mite (arrows). Haematoxylin and eosin.

Histological changes associated with intrafollicular mite presence included perifollicular perivascular dermatitis ($P = 0.03$), pigmentary incontinence (19 of 20 sections; $P = 0.0003$), follicular ectasia ($P = 0.0009$), superficial and follicular hyperkeratosis, and dermal fibrosis and plugging of follicular ostia with admixed sebum and keratin

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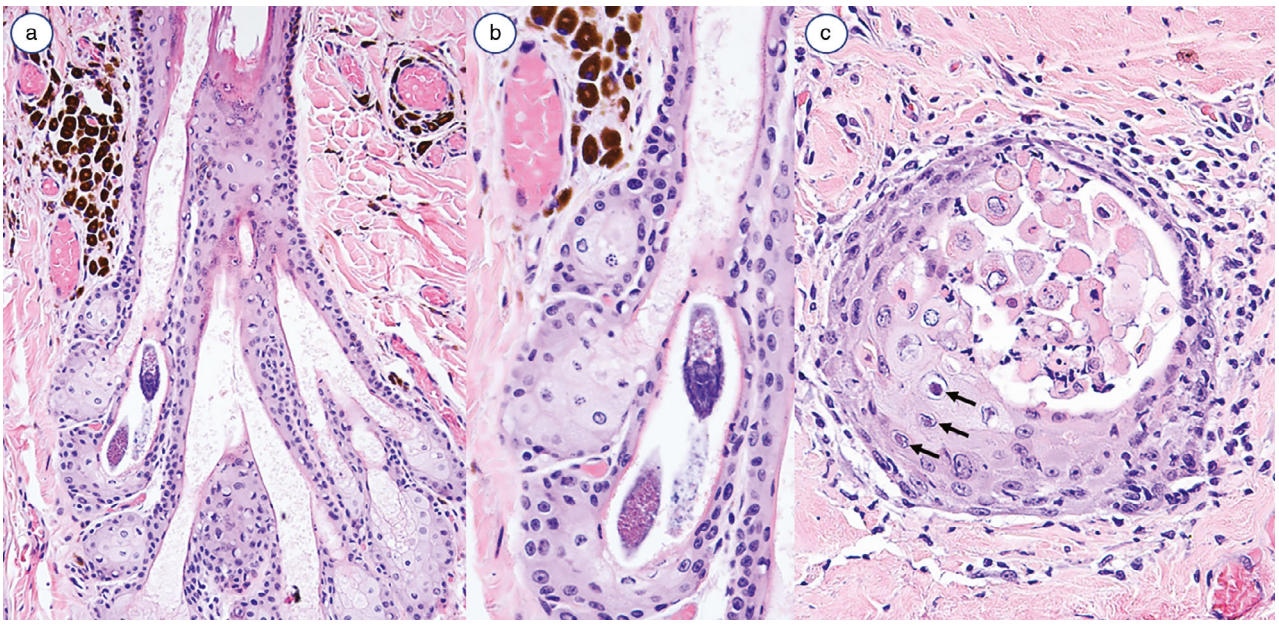

Figure 3. Follicular mural and perifollicular histopathological findings for an aged adult female southern sea otter (*Enhydra lutris nereis*: case 6734-13) (euthanized due to severe chronic dermatitis).
(a) Integument from the chin: longitudinal section through a hair follicle, showing partial profiles of mites within a sebaceous gland. The sebaceous gland ducts and follicular lumen are moderately dilated. The adjacent dermis contains abundant perifollicular and perivascular macrophages with abundant dark brown cytoplasmic granules (melanin; pigmentary incontinence: top left and top right). (b) Higher magnification detail of a portion of the previous image to show the mites and dermal pigmentary incontinence. (c) Hind flipper, transverse section of a hair follicle at the level of the infundibulum: some epithelial cells contain swollen, pale nuclei with central amphophilic intranuclear inclusions (presumptive herpesvirus: arrows) accompanied by epithelial apoptosis and mild perifollicular and follicular mural suppurative and lymphoplasmacytic dermatitis. All photos: Haematoxylin and eosin.

debris (10 of 20 sections) (Table 3; Figure 1a,b). These lesions were not present in nine mite-negative otters, and lesions suggestive of infestation were significantly less common and less severe in mite-negative tissue sections (Table 3). The perifollicular infiltrate was lymphoplasmacytic ($P = 0.02$), whereas interstitial and perivascular dermal infiltrates consisted of lymphocytes, plasma cells and neutrophils ($P = 0.03$). Mural folliculitis ($P = 0.39$) and furunculosis ($P = 0.42$) were not significantly associated with mite presence. Marked epidermal hyperplasia and compact orthokeratotic hyperkeratosis indicated chronic irritation. Epithelium of heavily affected follicles and associated sebaceous glands had apoptotic keratinocytes, some distorted follicles and some cell necrosis. Furunculosis was attributed to the effects of secondary bacterial folliculitis.

Two otters (10%) had chronic, severe generalized dermatitis and alopecia that contributed to death or euthanasia. Severe chronic skin lesions in an aged adult female (6734-13) necessitated humane euthanasia. In addition to demodicosis, she had epidermal ulceration associated with herpesvirus-like intranuclear inclusions in keratinocytes (Figures 2a–d, 3a–c). Bacterial cocci were present on the skin surface and within some affected follicles. Aerobic bacterial culture of affected skin and axillary and inguinal lymph nodes yielded *Staphylococcus intermedius*, *S. pseudintermedius*, *Streptoccocus phocae* and *Archanobacterim phocae*. This animal also had chronic cerebral lymphoplasmacytic meningoencephalitis with intralesional tissue cysts compatible with *Toxoplasma gondii*. The intestines were heavily infected with *Corynosoma enhydri* acanthocephalans.

The second case with severe demodicosis, an aged adult male (6397-12), died from dilated cardiomyopathy, with secondary acute hepatic vein thrombosis and massive hepatic parenchymal infarction. Chronic severe alopecia and dermatitis of the shoulder, head, extremities and thorax associated with demodicosis and secondary

Table 3. Univariate associations between histologically confirmed *Demodex* sp. mite infestation in skin of necropsied southern sea otters (*Enhydra lutris nereis*) and potential mite-associated skin lesions

Lesion	*P*-value	Number of mite-positive sections with lesions	Number of mite-negative sections with lesions	Number of mite-positive sections without lesions	Number of mite-negative sections without lesions
Pigmentary incontinence	0.0003*	10	12	10	88
Ectatic follicles	0.0009*	10	14	10	86
Perifolliculitis	0.02*	9	19	11	81
Dermal inflammation	0.03*	8	17	12	83
Mural folliculitis	0.39	3	8	17	92
Furunculosis	0.42	1	2	19	98
Perivascular inflammation	0.46	10	61	10	39

P-values obtained via Fisher's exact test: *indicates statistically significant difference between mite-positive and mite-negative integument.

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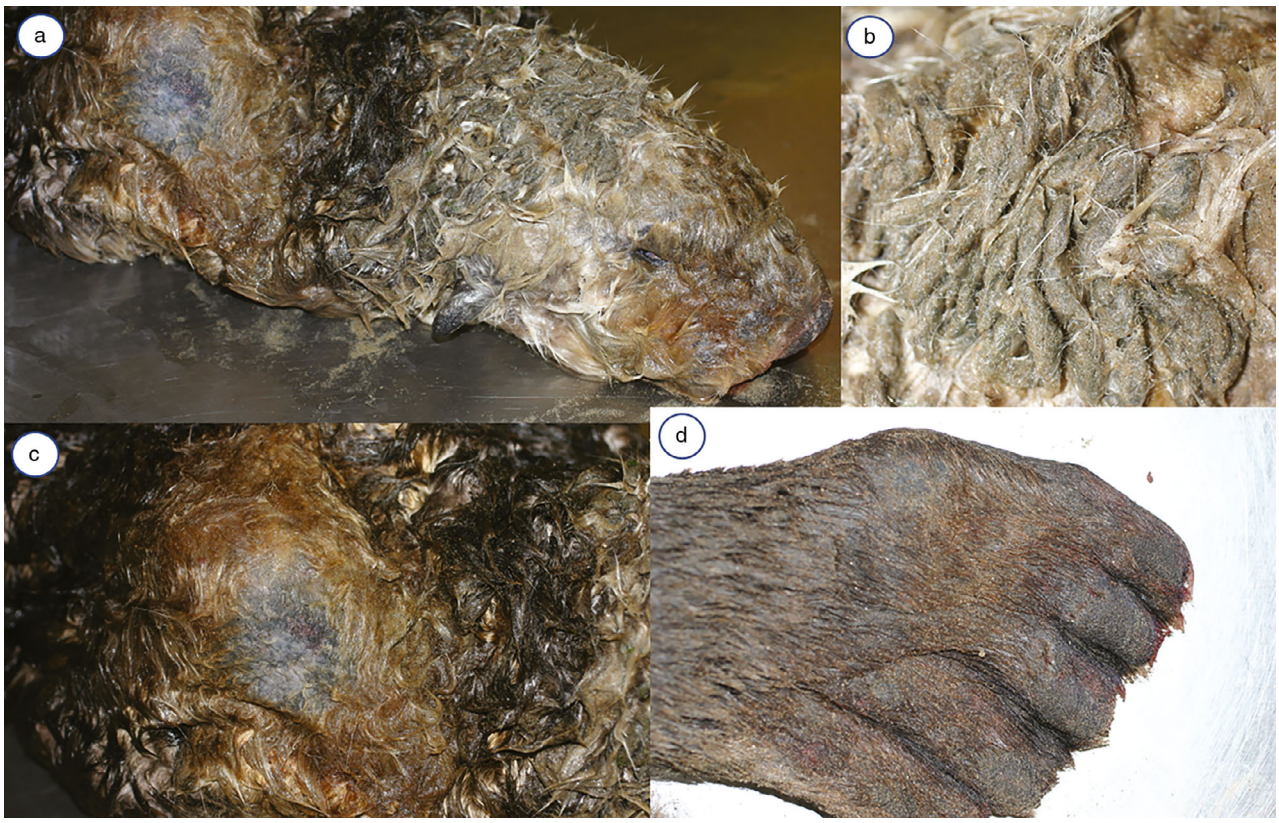

Figure 4. Demodicosis and bacterial pyoderma in an aged adult male southern sea otter (*Enhydra lutris nereis*: case 6397-12) (dermatitis as a contributing cause of death).
(a) Head and right shoulder, showing chronic dermatitis and alopecia. Adjacent hairs exhibit orange-brown discoloration (sebum, salivary staining and/or exudate from concurrent bacterial dermatitis). (b) Dorsal head, showing severe, regional matting of the pelage. (c) Right shoulder, higher magnification, showing central alopecia, lichenification and excoriation, and peripheral salivary staining. (d) Dorsal flipper, showing diffuse hyperpigmentation and lichenification, patchy alopecia and multifocal surface excoriation.

bacterial septicemia was a contributing cause of death (Figure 4a–d). This otter also had mild intestinal *C. enhydri* infection and sparse sarcocysts (*Sarcocystis* sp.) in skeletal muscle.

DNA sequencing

Amplification of mite DNA from cryopreserved integument was unsuccessful using the generic mite PCR protocol. PCR of paraffin scrolls from formalin-fixed, mite-positive sea otter integument yielded numerous ambiguous DNA bases, plus longer sequences of sufficient quality to perform a BLAST search that yielded 94% homology with *D. folliculorum* (GenBank accession HQ728000).

Discussion and conclusions

By contrast with other mustelids, sea otters have evolved a life history with extremely high energetic demands[22] yet lack the subcutaneous blubber layer found in other marine mammals.[27] Their ability to survive in the cold marine environment is instead entirely dependent on insulating air trapped within a highly specialized fur coat.[23,24,28] Sea otters have the densest pelage of any mammal with ≥150,000 hairs/cm^2.[28] Their compound hair follicles have central primary follicles forming long, thick guard hairs surrounded by many smaller follicles producing a shorter and finer undercoat.[24,27] Thermal insulation is achieved through interlocking of adjacent hairs which trap a layer of air next to the skin.[23,27] Even small patches of hair loss or wetting can substantially alter thermoregulation and cause death.[22] Here we document intrafollicular *Demodex* sp. mite infestation in southern sea otters with alopecia and dermatitis – lesions that can contribute to morbidity and mortality.

The pelage characteristics of these otters posed challenges to achieving longitudinal sections of follicles and sebaceous glands, an important requirement to assess *Demodex* mites microscopically. Techniques were developed during this study to facilitate sampling in relation to hair follicle orientation. Sand contamination presented an additional challenge as it contributed to poor-quality tissue sections.

A quarter of the sea otters had grossly apparent demodicosis, and chronic, mite-associated dermatitis was a primary or contributing cause of death in 10%. Half had *Demodex* sp. mite infestation on histological evaluation, including otters with and without skin lesions. We acknowledge sample bias because the sampled otters were obtained opportunistically and were found sick or dead. The population prevalence may exceed our sample prevalence because only a small portion of integument was evaluated microscopically. The single ivermectin-treated otter could have reduced prevalence as well.

Demodex mites were observed primarily within hair follicles, most prominently in the infundibulum near sebaceous gland ducts or within these ducts. Different

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Demodex spp. infest specific locations of their hosts: *D. canis* infests canine hair follicles and occasionally sebaceous glands, and *D. cati* infests feline hair follicles, whereas *D. injai* and *D. phocidi* are found in sebaceous glands of dogs and harbour seals, respectively.[29] *Demodex cornei* (possibly a strain of *D. canis*)[30] and *D. gatoi* inhabit the stratum corneum of dogs and cats, respectively.[24,31,32] *Demodex zalophi* is found in the sebaceous ducts and occasionally the hair follicles of California sea lions (PD, unpublished observation).

Morphological features were consistent with prior descriptions of *Demodex* spp.[21,33] and DNA sequencing confirmed that the sea otter mite is in the *Demodex* genus. As *Demodex* mites tend to be monoxenous,[34] this parasite is likely unique to sea otters. The length of sea otter mites (164 μm) is comparable to *Demodex* sp. from European otters (*L. lutra*: 170–209 μm)[21] and California sea lions (202–258 μm).[19] Our measurements may underestimate total length because they were taken from histological sections.

Histological features of sea otter mite infestation included ectatic follicles with perifollicular inflammation and associated pigmentary incontinence, similar to lesions in dogs and other mammals.[4,12,31,34-36] As reported for dogs with demodicosis, inflammatory lesions were occasionally seen in the absence of *Demodex* mites.[31,37] Although this could represent suboptimal tissue collection, sampling error or the presence of mite-infested follicles outside the plane of tissue section, deeper serial sections did not improve mite detection. Lymphocytic mural folliculitis (seen in dogs with uncomplicated demodicosis) and furunculosis (a common feature in canine demodicosis with secondary bacterial infections) were rare in sea otters.[12] Fewer mites were observed in sea otter skin samples, when compared to dogs with clinical demodicosis.[7,31]

Often *Demodex* infestations in mammals are localized and asymptomatic,[38] with more severe lesions being associated with complicating secondary infections.[1,12,17,37] Demodicosis in sea otters appears to be extensive and prolonged, with associated alopecia and erythema, as is characteristic of animals with immune deficits or genetic predispositions, such as in young animals, certain breeds, older dogs, animals with concomitant disease, animals on immune-suppressive therapies or animals in oestrus or nursing.[31,39,40] Two of the aged otters had severe chronic skin lesions with associated alopecia and evidence of additional bacterial, viral or protozoal infections. In dogs, severe clinical lesions with pustules, luminal folliculitis and furunculosis are the result of demodicosis with secondary bacterial infection,[12] further suggesting immune compromise.

Although grossly apparent demodicosis in sea otters was associated with higher follicular mite burdens, high mite burdens were not always predictive of more severe lesions. This may be a consequence of the small sample size and the limited number of sections examined from each individual, as mite numbers vary across locations. Sex and age were not significant predictors of severe disease, although both otters with demodicosis as primary or contributing causes of death were aged adults. Inbreeding depression has been documented in most sea otter subpopulations as a consequence of massive harvest during the 19th Century, and immune suppression has been hypothesized as an impediment to southern sea otter population recovery.[35,41] Potential added stressors include concurrent disease (including polyparasitism), nutritional deficiencies, high nutritional demands, hormonal fluctuations due to reproduction, seasonal weather and oceanographic changes, intraspecific aggression, stress of captivity, and exposure to oil or other environmental toxins.[15,16,36,41]

The most highly infested areas were chin and perioral skin, followed by hind flippers, torso and prepuce. The high parasite density on the chin suggests that facial contact, including affiliative, aggressive and grooming behaviours, could be an important means of mite transmission. The larger number of mites from the genital area could indicate that sexual transmission is part of sea otter *Demodex* ecology. Even if the high mite density in these regions is the result of tissue factors (e.g. the quality of sebaceous exudate), the larger numbers of mites could facilitate contact-dependent transmission.

Demodex sp. infestation appears to be common in stranded southern sea otters, which augments the relatively sparse literature on parasitic arthropods of southern sea otters,[29,42] and reports of infestation across all mustelids and marine mammals.[21] Careful evaluation with repetitive deep skin scrapings or molecular assays is warranted for sea otters with alopecia and dermatitis, including pups given the possibility of infestation during parturition. Given the likely contribution of immune-suppression to disease severity, treatment options must be explored. Ivermectin, doramectin and amitraz have shown efficacy for demodicosis treatment in wildlife, including a captive koala (*Phascolarctos cinereu*) and captive harbour seals [20,43–45]. However, amitraz must be applied in a contained environment due to its toxicity to other marine life.[46]

By compromising integrity of the specialized pelage, *Demodex* infestation may contribute to sea otter mortality. This charismatic threatened species has struggled to regain population stability after surviving impacts of hunting, shark bite, trauma, habitat change, disease, inbreeding and anthropogenic pollution. Studies employing a large sample size and assessing other sea otter populations will help to further characterize the scope and severity of this newly identified threat.

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References

1. Fisher WF. Recent advances in psoroptic acariasis and demodectic mange of domestic animals and sarcoptic scabies of humans. *Int J Dermatol* 1981; 20: 585–588.
2. Nutting WB, Satterfield LC, Cosgrove GE. *Demodex* sp. infesting tongue, esophagus, and oral cavity of *Onychomys leucogaster*, the grasshopper mouse. *J Parasitol* 1973; 59: 893–896.

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3. Spickett SG. Studies on *Demodex folliculorum* Simon (1842). I. Life history. *Parasitology* 1961; 51: 181–192.
4. Chesney CJ. Demodicosis in the cat. *J Small Anim Pract* 1989; 30: 689–695.
5. Forrester DJ, Spalding MG, Wooding JB. Demodicosis in black bears (*Ursus americanus*) from Florida. *J Wildl Dis* 1993; 29: 136–138.
6. Gentes M-L, Proctor H, Wobeser G. Demodicosis in a mule deer (*Odocoileus hemionus hemionus*) from Saskatchewan, Canada. *J Wildl Dis* 2007; 43: 758–761.
7. Gortel K. Update on canine demodicosis. *Vet Clin North Am Small Anim Pract* 2006; 36: 229–241.
8. Hamir AN, Snyder DE, Hanlon CA, et al. First report of a *Demodex* sp. in raccoons (*Procyon lotor*). *J Wildl Dis* 1993; 29: 139–141.
9. Matthes H. Investigations of pathogenesis of cattle demodicosis: sites of predilection, habitat and dynamics of demodectic nodules. *Vet Parasitol* 1994; 53: 283–291.
10. Baker KP. Hyperpigmentation of the skin in canine demodicosis. *Vet Parasitol* 1975; 1: 193–197.
11. Cayatte SM, Scott DW, Miller WH. Perifollicular melanosis in the dog. *Vet Dermatol* 1992; 3: 165–170.
12. Caswell JL, Yager JA, Ferrer L, et al. Canine demodicosis: a reexamination of the histopathologic lesions and description of the immunophenotype of infiltrating cells. *Vet Dermatol* 1995; 6: 9–19.
13. Day M. An immunohistochemical study of the lesions of demodicosis in the dog. *J Comp Pathol* 1997; 116: 203–216.
14. Desch CE, Dailey MD, Tuomi P. Description of a hair follicle mite (Acari: Demodecidae) parasitic in the earless seal family Phocidae (Mammalia: Carnivora) from the harbor seal *Phoca vitulina* Linnaeus, 1758. *Int J Acarol* 2003; 29: 231–235.
15. Salvadori C, Formenti N, Trogu T, et al. Demodicosis in Chamois (Rupicapra rupicapra subsp. rupicapra) in the Italian Alps, 2013–14. *J Wildl Dis* 2016; 52: 433–435.
16. Nutting WB, Dailey MD. Demodicosis (Acari: Demodicidae) in the California sea lion, Zalophus Californianus. *J Med Entomol* 1980; 17: 344–347.
17. Tarallo VD, Lia RP, Sasanelli M, et al. Efficacy of Amitraz plus Metaflumizone for the treatment of canine demodicosis associated with *Malassezia pachydermatis*. *Parasit Vect* 2009; 2: 13.
18. Singh SK, Dimri U. The immuno-pathological conversions of canine demodicosis. *Vet Parasitol* 2014; 203: 1–5.
19. Dailey M, Nutting W. *Demodex zalophi* sp. nov. (Acari: Demodicidae) from *Zalophus californianu*s, the California sea lion. *Acarologia* 1980; 21: 423–428.
20. Kim K-T, Lee S-H, Kwak D. Treatment of naturally acquired demodectic mange with amitraz in two harbour seals (*Phoca vitulina*). *Acta Vet Hung* 2015; 63: 352–357.
21. Izdebska JN, Rolbiecki L. *Demodex lutrae* n. sp. (Acari) in European otter *Lutra lutr* a (Carnivora: Mustelidae) with data from other demodecid mites in carnivores. *J Parasitol* 2014; 100: 784–789.
22. Murray MJ. Veterinary medicine and sea otter conservation. In: Larson SE, Bodkin JL, VanBlaricom GR eds. *Sea Otter Conservation, chapter 7*. Amsterdam: Elsevier, 2015;, 159–195.
23. Yeates LC, Williams TM, Fink TL. Diving and foraging energetics of the smallest marine mammal, the sea otter (*Enhydra lutris*). *J Exper Biol* 2007; 210: 1,960–1,970.
24. Williams TD, Allen DD, Groff JM, et al. An analysis of California sea otter (*Enhydra lutris*) pelage and integument. *Mar Mamm Sci* 1992; 8: 1–18.
25. de Rojas M, Riazzo C, Callejón R, et al. Molecular study on three morphotypes of *Demodex* mites (Acarina: Demodicidae) from dogs. *Parasitol Res* 2012; 111: 2,165–2,172.
26. R Core Team. *R: A language and environment for statistical computing*. Vienna, Austria: R Foundation for Statistical Computing, 2015; http://www.R-project.org/.
27. Uhen MD. Evolution of marine mammals: back to the sea after 300 million years. *Anatom Rec* 2007; 290: 514–522.
28. Nickerson R. *Sea otters: a natural history and guide*: San Francisco. USA: Chronicle Books, 1989.
29. Izdebska JN, Rolbiecki L. Parasitic arthropods as the cause of parasitoses in aquatic animals. In: Buczek A, Błaszak C, eds. *Arthropods. Ecological and pathological aspects of parasite–host relationships*. Lublin, Poland: Akapin, 2010; 125–135.
30. Sastre N, Ravera I, Villanueva S, et al. Phylogenetic relationships in three species of canine *Demodex* mite based on partial sequences of mitochondrial 16S rDNA. *Vet Dermatol* 2012; 23: 509-e101.
31. Izdebska JN. *Demodex* sp.(acari, demodecidae) and demodecosis in dogs: characteristics, symptoms, occurrence. *Bull Vet Inst Pulawy* 2010; 54: 335–338.
32. Izdebska JN, Fryderyk S. Diversity of three species of the genus *Demodex* (Acari, Demodecidae) parasitizing dogs in Poland. *Pol J Environ Stud* 2011; 20: 565–569.
33. Nutting WB, Desch CE. *Demodex canis*: redescription and reevaluation. *Cornell Vet* 1978; 68: 139–149.
34. Walter DE, Proctor HC, eds. Animals as habitats. In: *Mites: Ecology, Evolution & Behaviour, chapter 9*. Dordrecht: Springer, 2013; 341–422.
35. Larson S, Jameson R, Etnier M, et al. Loss of genetic diversity in sea otters (*Enhydra lutris*) associated with the fur trade of the 18th and 19th centuries. *Mol Ecol* 2002; 11: 1,899–1,903.
36. De Bosschere H, Casaer J, Neukermans A, et al. Severe alopecia due to demodicosis in roe deer (*Capreolus capreolus*) in Belgium. *Vet J* 2007; 174: 665–668.
37. Grandi F, Pasternak A, Beserra HEO. Digit loss due to *Demodex* spp. infestation in a dog: clinical and pathological features. *Open Vet J* 2013; 3: 53–55.
38. Lacey N, Raghallaigh SN, Powell FC. Demodex mites–commensals, parasites or mutualistic organisms. *Dermatology* 2011; 222: 128–130.
39. Scott D, Miller W, Griffin C. *Muller and Kirk's Small Animal Dermatology*, 7th edition. Maryland Heights, MO: Saunders Elsevier, 2012; 304–310.
40. Shipstone M. Generalised demodicosis in dogs, clinical perspective. *Aust Vet J* 2000; 78: 240–242.
41. Kannan K, Perrotta E, Thomas NJ, et al. A comparative analysis of polybrominated diphenyl ethers and polychlorinated biphenyls in southern sea otters that died of infectious diseases and noninfectious causes. *Arch Environ Contam Toxicol* 2007; 53: 293–302.
42. Pesapane R, Dodd E, Javeed NN, et al. Infestation with the nasopulmonary mite *Halarachne halichoeri* in threatened southern sea otters (*Enhydra lutris nereis*). *Int J Parasitol Parasites Wildl* 2018; 7: 386–390.
43. Kuznetsova E, Vysokikh A, Bourdeau P. First description of demodicosis in 12 galagos (*Galago senegalensis*). *Vet Dermatol* 2012; 23: 61–e14.
44. Takle GL, Suedmeyer WK, Mertins JW, et al. Generalized demodecosis in three sibling, juvenile rock hyraxes (*Procavia capensis*). *J Zoo Wildl Med* 2010; 41: 496–502.
45. Vogelnest LJ, Vogelnest L, Mueller RS. An undescribed *Demodex* sp. and demodicosis in a captive koala (*Phascolarctos cinereus*). *J Zoo Wildl Med* 2000; 31: 100–106.
46. Sanchez-Bayo F. Insecticides mode of action in relation to their toxicity to non-target organisms. *J Env Analyt Toxicol* 2011; S4: 1–9.

Résumé

Contexte – La loutre de mer de Californie (*Enhydra lutris nereis*) utilise son pelage intact pour sa thermorégulation et ainsi, une démodécie cliniquement significative et l'alopécie associée peuvent causer une morbidité et la mort.

Hypothèses/Objectifs – Cette étude a pour but de décrire les lésions associées à une infestation folliculaire de *Demodex* sp., estimer la prévalence et l'intensité de l'infestation, décrire la distribution des

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acariens à l'aide de régions anatomiques clés et déterminer la présence ou l'absence d'acariens et les facteurs de risque des hôtes.
Sujets – Vingt loutres de mer de Californie sauvages ont été autopsiées après s'être échouées le long de la côte californienne de 2005 à 2018.
Matériels et méthodes – La peau macroscopiquement normale et anomale de la tête, du périnée, des zones génitales, des papilles mammaires et des membres a été évaluée microscopiquement pour les acariens et les lésions pathologiques liées aux acariens.
Résultats – Les acariens intrafolliculaires ont été observés dans la peau de 55% des loutres et 20% avaient une démodécie clinique. La démodécie a été considérée comme contribuant à la mort ou à l'euthanasie dans deux cas. Bien que les acariens *Demodex* sp. soient souvent observées dans la peau macroscopiquement normale, la présence multiple d'acariens intrafolliculaires regroupés en amas denses était généralement associée avec de l'incontinence pigmentaire, des follicules ectatiques, une périfolliculite lymphoplasmocytaire et une inflammation dermique neutrophilique et lymphoplasmocytaire. Les autres données regroupent une hyperplasie épidermique, une hyperkératose orthokératosique de l'épiderme et de l'épithélium folliculaire, une pyodermite concomitante et une nécrose cellulaire. La peau péri-orale, en particulier le menton, avait la prévalence la plus élevée d'acariens et la densité en acarien la plus élevée suggérant un contact facial comme mode de transmission.
Conclusions et importance clinique – Nos recherches confirment que la démodécie participe à la morbidité et à la mortalité des loutres de mer avec implications importantes pour les soins de cliniques, de réhabilitation et de conservation.

RESUMEN

Introducción – las nutrias marinas del sur (*Enhydra lutris nereis*) dependen de un pelaje en buenas condiciones para la termorregulación y, por lo tanto, la demodicosis clínicamente significativa y la alopecia asociada pueden causar morbilidad y muerte.
Hipótesis/Objetivos – Este estudio tuvo como objetivo describir las lesiones asociadas con la infestación folicular por *Demodex sp.*, estimar la prevalencia e intensidad de la infestación, describir la distribución de ácaros en regiones anatómicas clave y evaluar la presencia o ausencia de ácaros en relación con las lesiones y los factores de riesgo del huésped.
Animales – Veinte nutrias marinas salvajes del sur necropsiadas que quedaron varadas a lo largo de la costa central de California entre 2005 y 2018.
Métodos y materiales – Se evaluó microscópicamente el tegumento macroscópicamente normal y anormal de la cabeza, el perineo, los genitales, las papilas mamilares y las extremidades para detectar ácaros y hallazgos patológicos asociados a los ácaros.
Resultados – Se observaron ácaros intrafoliculares en el tegumento del 55% de las nutrias y el 20% presentó demodicosis clínica. Se consideró que la demodicosis contribuyó a la muerte o la eutanasia en dos casos. Aunque ácaros de *Demodex sp.* a menudo se observaron microscópicamente en piel macroscópica normal, la presencia de múltiples ácaros intrafoliculares densamente empaquetados generalmente se asoció con incontinencia pigmentaria, folículos ectásicos, perifoliculitis linfoplasmocítica e inflamación dérmica neutrofílica y linfoplasmacítica. Otros hallazgos incluyeron hiperplasia epidérmica, hiperqueratosis ortoqueratósica de epidermis y epitelio folicular, pioderma concurrente y necrosis celular. El tegumento perioral, especialmente del mentón, tuvo la mayor prevalencia de ácaros y la mayor densidad de ácaros, lo que sugiere el contacto facial como medio de transmisión de ácaros.
Conclusiones e importancia clínica – Nuestra investigación confirmó que la sarna demodécica contribuye a la morbilidad y mortalidad en las nutrias marinas, con importantes implicaciones para la atención clínica, la rehabilitación y la conservación.

Zusammenfassung

Hintergrund – Der Seeotter des Südpazifiks (*Enhydra lutris nereis*) benötigt zur Thermoregulierung zwingend ein intaktes Haarkleid und daher kann eine klinisch signifikante Demodikose und eine damit einhergehende Alopezie Erkrankung und Tod verursachen.
Hypothese/Ziele – Diese Studie zielte darauf ab, Veränderungen, die mit einer follikulären *Demodex* sp. Infektion einhergehen, zu beschreiben, die Prävalenz und die Intensität des Befalls abzuschätzen, die Milbenverteilung an den anatomischen Schlüsselregionen zu beschreiben, und die Milbenpräsenz oder deren Fehlen in Relation zu den Veränderungen und den Risikofaktoren des Wirtes zu beurteilen.
Tiere – Es wurden zwanzig wilde südliche Seeotter, die entlang der Küste Zentralkaliforniens zwischen 2005 und 2018 gestrandet waren, autopsiert.
Methoden und Materialien – Makroskopisch normale und abnormale Haut von Kopf, Perineum, den Genitalien, der Brustwarzen und der Extremitäten wurden mikroskopisch auf Milben und auf pathologische Befunde im Zusammenhang mit Milben untersucht.
Ergebnisse – Es wurden intrafollikuläre Milben in der Haut von 55% der Otter gefunden, wobei 20% von ihnen eine klinische Demodikose aufwiesen. Die Demodikose wurde als ursächlich für den Tod und die

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Euthanasie in zwei Fällen angesehen. Obwohl *Demodex* sp. Milben oft mikroskopisch in mit freiem Auge normaler Haut gefunden wurden, wurde das Vorkommen von multiplen dicht gepackten intrafollikulären Milben generell mit Pigmentinkontinenz, ektatischen Follikeln, lymphoplasmazytischer Perifollikulitis, und neutrophiler und lymphoplasmazytischer, dermaler Entzündung gesehen. Die Haut rundum den Mund, vor allem am Kinn, zeigte die höchste Prävalenz von Milben und die höchste Milbendichte, was auf einen Gesichtskontakt als Übertragungsweg für die Milben hinweist.
Schlussfolgerungen und klinische Bedeutung – Unsere Forschung bestätigte die Demodexmilbe als Beitragende zur Morbidität und Mortalität der Seeotter, was wichtige Implikationen für ihre klinische Versorgung, Rehabilitation und Erhaltung darstellt.

要約
背景 – 南方ラッコ (Enhydra lutris nereis)の体温調節は無傷の毛に依存するため、臨床的に著しいニキビダニ症およびそれに伴う脱毛症は、病的疾患や死亡の原因となることがある。
仮説/目的 – 本研究の目的は、毛包寄生性ニキビダニ属に関連する病変の記述、寄生の有病率および強度の推定、主要な解剖学的領域におけるダニの分布の記述、病変および宿主の危険因子との関連におけるダニの有無を評価することであった。
被験動物 – 2005年から2018年までカリフォルニア中央海岸で座礁した野生の南方ラッコ20頭を剖検した。
材料と方法 – 頭部、会陰部、生殖器、乳頭および四肢の総じて正常または異常な皮膚を顕微鏡的に評価し、ダニおよびダニに関連した病理学的所見を調査した。
結果 – ラッコの55%の皮膚には毛包内にダニが観察され、20%には臨床的にニキビダニ症が認められた。そのうち2例は死亡または安楽死の原因と考えられた。正常な皮膚においてしばしばニキビダニが顕微鏡下で観察されたが、毛包内に密に詰まった複数のダニの存在は、一般的に色素脱、拡張性毛包、リンパ球形質細胞性毛包周囲炎、好中球性およびリンパ球形質細胞性の真皮の炎症と関連していた。その他の所見としては、表皮過形成、表皮および毛包漏斗部の正角化性角化亢進、膿皮症および細胞壊死を併発していた。口周囲の皮膚、特に顎部でダニの有病率が最も高く、ダニ密度も最も高かったことから、顔面接触がダニの感染手段であることが示唆された。
結論と臨床上の重要性 – 本研究は、ラッコの罹患率および死亡率の一因であるニキビダニを確認し、臨床ケア、リハビリテーション、管理に重要な意味を持つことを示した。

摘要
背景 — 方海獭依赖于被毛完整来进行体温调节，因此具有临床意义的蠕形螨病和造成的脱毛可导致其发病和死亡。
假设/目的 — 本研究旨在描述毛囊蠕形螨侵染造成的病变，评估侵染的流行率和严重程度，描述关键解剖区域的螨虫分布，并评估是否存在造成病变的螨虫，以及宿主风险因素。
动物 — 2005年至2018年，在加利福尼亚中部海岸搁浅并进行尸检的20只野生南方海獭。
方法和材料 — 采自外观正常和异常的头部、会阴、生殖器、乳头和四肢皮肤，评估显微镜下螨虫及螨虫相关的病理学结果。
结果 — 在55%的水獭皮肤中观察到毛囊内螨虫，20%患有临床蠕形螨病。在2个病例中，蠕形螨病导致了死亡或因此而人道处死。尽管在外观正常的皮肤中，通过显微镜经常能观察到蠕形螨虫体，多处毛囊内出现的密集螨虫，通常造成色素失禁、毛囊扩张、淋巴浆细胞性毛囊周炎以及中性粒细胞和淋巴浆细胞性真皮炎症。其他结果包括表皮增生、表皮和毛囊上皮的正角化性过度角化、并发脓皮病和细胞坏死。口周皮肤，尤其是颏部，螨虫侵染率最高，螨虫密度最高，这提示面部接触是螨虫传播的一种途径。
结论和临床重要性 — 我们的研究证实蠕形螨是海獭发病和死亡的因素，临床护理、康复和保护具有重要意义。

Resumo
Contexto – As lontras do mar do Sul (*Enhydra lutris nereis)* dependem da pelagem intacta para termorregulação e, portanto, demodiciose clinicamente significativa e alopecia associada podem causar morbidade e morte.
Hipótese/Objetivos – Este estudo teve como objetivo descrever lesões associadas à infestação folicular por *Demodex sp.*, estimar a prevalência e intensidade da infestação, descrever a distribuição de ácaros nas principais regiões anatômicas e avaliar a presença ou ausência de ácaros em relação a lesões e fatores de risco do hospedeiro.
Animais – Vinte lontras do mar do Sul selvangens necropsiadas que ficaram presas ao longo da costa central da Califórnia de 2005 a 2018.
Métodos e materiais – Tegumento grosseiramente normal e anormal da cabeça, períneo, genitais, papilas mamilares e membros foram avaliados microscopicamente para pesquisa de ácaros ácaros e achados patológicos associados aos ácaros.
Resultados – Observou-se ácaros intrafoliculares no tegumento de 55% das lontras e 20% apresentaram demodiciose clínica. A demodiciose foi considerada como contribuinte para a morte ou eutanásia em dois casos. Embora os ácaros *Demodex sp.* tenham sido frequentemente observados microscopicamente na

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pele grosseiramente normal, a presença de múltiplos ácaros intrafoliculares densamente compactados geralmente estava associada a incontinência pigmentar, folículos ectáticos, perifoliculite linfoplasmocitária e inflamação dérmica neutrofílica e linfoplasmocitária. Outros achados incluíram hiperplasia epidérmica, hiperceratose ortoqueratótica da epiderme e epitélio folicular, pioderma concomitante e necrose celular. O tegumento perioral, principalmente do mento, apresentou a maior prevalência de ácaros e a maior densidade de ácaros, sugerindo o contato facial como meio de transmissão dos ácaros.

Conclusões e importância clínica – Nossa pesquisa confirmou a sarna demodécica como contribuinte para a morbidade e mortalidade em lontras marinhas, com implicações importantes para o atendimento clínico, reabilitação e conservação.

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