Infestivity of *Demodex canis* to Hamster Skin Engrafted onto SCID Mice

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**ABSTRACT.** We demonstrated that *Demodex canis* was transferred to skin xenografts of a dog and a hamster onto severe combined immunodeficiency mice. After the transfer of mites, the number of eggs, larvae, nymphs and adult mites per gram of canine and hamster xenografts increased, whereas no live mites were detected on murine allograft. These results indicate that *D. canis* proliferates in hair follicles of dog and hamster skins but not in murine allograft. Therefore, *D. canis* may have host preference but not strict host-specificity.

**KEY WORDS:** canine, demodicosis, skin graft.

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**NOTE**

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*Demodex* species are found in many kinds of mammals, for example, *D. folliculorum* and *D. brevis* for humans [11], *D. aurati* and *D. ricceti* for hamsters [12], and *D. canis* and *D. injai* for dogs [5, 6, 13]. These species from different hosts exhibit morphologic differences and they have been thought to be physiologically different and host specific. Cross infestivity of *Demodex* mites is important when immunosuppressive human patients keep a pet with demodicosis or when people need to keep their pet with other animals such as the hamster for which naturally occurring generalized demodicosis has been well recognized [19]. The purpose of the present study was to evaluate the infestivity of *Demodex canis* to hamster skin engrafted onto SCID mice.

Clinically normal beagle dogs and hamsters (Djungarian hamster; *Phodopus sungorus*) were used in this study. Canine skin sheets obtained from the lateral thorax under general anesthesia were cut into 10/15 mm pieces and stored at 4°C in sterile physiological saline containing 500 µg/ml gentamycin until engraftment. Subcutaneous fat was completely removed using a dermatome. Half of the skin sheets were confirmed by histologic examination to be free of *Demodex canis*. Hamster skin sheets, obtained from the back, were prepared as well as canine skin described above and were also confirmed to be free of *Demodex spp*. Skin sheets of SCID mice were also prepared in the same way. Five sets of each skin graft were used.

Fox Chase C. B -17 SCID mice were obtained from Clea Japan, Inc. and handled at our laboratory in a pathogen-free environment. All mice used were 7 to 8 weeks old and were confirmed as not “leaky” by the breeder. SCID mice were anesthetized by i.p. injection of 50 mg/kg pentobarbital sodium. The skin was shaved and a skin segment was excised from the back of the animal. The wound was covered with the skin sheet from dogs, hamsters and SCID mice, fixed with suture material, and covered with vaseline gauze and adhesive tape. The dressing material was removed after 10 days. Each mouse was housed separately.

Three sets of skin graft on the SCID mice were inoculated with 200 *D. canis* mites, collected from clinical cases of canine generalized demodicosis, as described previously.
[4]. Briefly, mites were obtained by skin scraping, using a scalpel blade coated in mineral oil and the presence of mites was confirmed by microscopic examination and collected mites were counted. This skin-scraping material was applied directly to the skin grafts on the SCID mice, and the site was bandaged for 10 days. The remaining two sets of skin graft on the SCID mice were used as negative controls, respectively.

Mice were sacrificed 90 days after infection with *Demodex canis* and half of each xenograft was fixed in 10% buffered formalin, embedded in paraffin, sectioned routinely at 5 µm, and stained with hematoxylin and eosin. In cases in which mites were not apparent, multiple histologic sections taken at 100-µm intervals were examined. In addition to the histologic examination, half of the graft was macerated in a solution including 40% dimethyl sulfoxide and 15% KOH. The number of *Demodex* mites (adults, nymphs, larvae, and eggs) per gram of xenograft was counted. Results were expressed as the mean ± standard deviation (SD).

The xenografts were accepted by all mice in this study. Hair was growing from the grafts in all mice 90 days after infection with *D. canis*. Canine epidermis was thicker than the mouse epidermis whereas the thickness of hamster’s epidermis was almost same as those of mice. These engrafts showed normal keratinocyte maturation histologically. Mild to moderate acanthosis was observed in most canine skin grafts, but little was seen in hamsters and mice. Mild infiltration of neutrophils was occasionally observed mainly at the periphery of the graft and they did not target hair follicles. Dermal fibrosis and orthokeratotic hyperplasia were also often prominent, but these skin lesions were seen to a similar degree in uninfected skin grafts.

*Demodex canis* mites were present in the histologic sections of the xenografts of a dog (Fig. 1) and a hamster (Fig. 2) in all mice infected with *D. canis*. Numerous mites were observed in each infected hair follicle of canine xenografts, whereas several mites were observed in each hair follicle of hamster xenografts. No live mites were seen in the hair follicles of the surrounding murine skin and murine allograft. No mites were observed either on the surface of the epidermis or in the dermis.

In microscopic examination of macerated skin grafts, all four stages of *D. canis* were observed in canine and hamster xenografts. The number of eggs, larvae, nymphs and adult mites per gram of canine xenograft were 489 ± 102, 867 ± 306, 1933 ± 200, 7178 ± 1140, respectively. The total of number mites of canine xenograft was 10,467 ± 1058. The number of eggs, larvae, nymphs and adult mites per gram of hamster’s xenograft were 611 ± 192, 2,416 ± 363, 2,444 ± 1,503, 20,417 ± 8830, respectively. The total of number mites from hamster xenograft was 24,778 ± 8360. *Demodex* mites of hamster xenografts were morphologically the same as *D. canis* on canine xenografts.

In this study, we have provided direct evidence that *D. canis* may not be host-specific. *D. canis* survived not only when applied to canine xenograft onto SCID mice but also when applied to hamster xenograft. The presence of intact eggs, larvae and nymphs, detected by microscopic examina-

![Fig. 1. Photomicrograph showing canine xenograft and adjacent SCID mouse skin. (a) The arrow marks the junction of canine and murine skin. *Demodex* mites are present in hair follicles (arrow head). H&E. × 55, (b) *Demodex* mites are present in extended hair follicles. H&E × 220.](image-url)
tion of macerated skin grafts, indicates that the mites proliferated in both the transplanted dog and hamster skins. This is the first report that experimental cross-infestivity with Demodex canis was confirmed using the SCID mice model. Canine skin engrafted onto SCID mice did not prevent the proliferation of D. canis. Dogs that are experimentally infected with large numbers of D. canis rarely develop cutaneous lesions [7]. However, puppies treated with anti-lymphocyte serum for the first 16 weeks of life are acceptable to experimental infection with D. canis [7]. These results reported by Healey and Giafar [7] indicate that host lymphocytes protect mite infestation. Caswell et al. [4] suggested that reconstitution of infected SCID mice with canine leukocytes might provide the importance of specific subsets of lymphocytes in controlling the proliferation of D. canis. Using this model, Linder et al. [9] reported that systemic dog factors were important for controlling the proliferation of mites. Therefore, skin barrier systems of dogs may not prevent the proliferation of D. canis, although the natural skin environment of dogs may differ from canine skin grafts in aspects of hormones, temperature and other factors [4]. The number of total mites from xenograft of hamster per gram of skin was larger than that of canine xenograft, although the number of mites per hair follicle of xenograft of hamster was smaller than that of canine xenograft. This phenomenon could reflect the different thickness of skins, and also the space of hair follicles per gram of skin might be wider in xenograft of hamster.

D. canis may not be host-specific but may have host preference. Sarcoptes scabiei, ectoparasites that are morphologically the same among species, but not host-specific and possess some degree of host preference [1]. At present, host species play a key role in identifying Demodex spp. Thus it is possible that some different morphological Demodex species may be the same species or mutants. Cutaneous diseases associated with demodicosis of immunosuppressive patients have been reported [8, 14, 16], although the pathogenicity of D. folliculorum remains controversial. Also, natural transfer of Demodex mites between human and dogs has been reported [10]. The SCID mouse model using humans and other mammals’ skin may provide further knowledge of cross-infestivity of Demodex mites. New technology including DNA fingerprinting may help to identify Demodex species as well as Sarcoptes scabiei [3, 20]. Although we have described only 90 days after infection with D. canis, long-term observation is necessary to identify morphological differences in Demodex mites.

In conclusion, D. canis may not be host-specific but may have host preference, since D. canis survived when applied to xenograft of hamster on SCID mice, but not to murine allograft.

REFERENCES