What's Eating You? *Demodex folliculorum*

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Identifying Features

The prostigmata mite *Demodex folliculorum* (Figure, A) is characterized by a long wormlike body devoid of setae (hairs). Its body is annulate (composed of rings). Children become colonized by the mite early in life, presumably during breast-feeding and other close physical contact.

Adverse Reactions

Demodex mites are commonly associated with an inflammatory response.[1] Their role in rosacea has been a matter of controversy, but *Demodex* mites are common in lesions of rosacea. They can be found in great numbers in smears from papulovesicular lesions and have been associated with dramatic eruptions that mimic rosacea in immunosuppressed patients.[2,3] *Demodex*-associated folliculitis is a common finding in histologic specimens from the face. *Demodex* folliculitis may present as a solitary papular lesion, which may clinically suggest basal cell carcinoma. Sulfur, permethrin, crotamiton, and metronidazole have been used to treat *Demodex*-associated folliculitis.[4-7]

Demodex mites have been implicated as a cause of blepharitis, responsive to lindane, pilocarpine, and mercury ointment.[8,9] Such cases are best managed with the help of an ophthalmologist.

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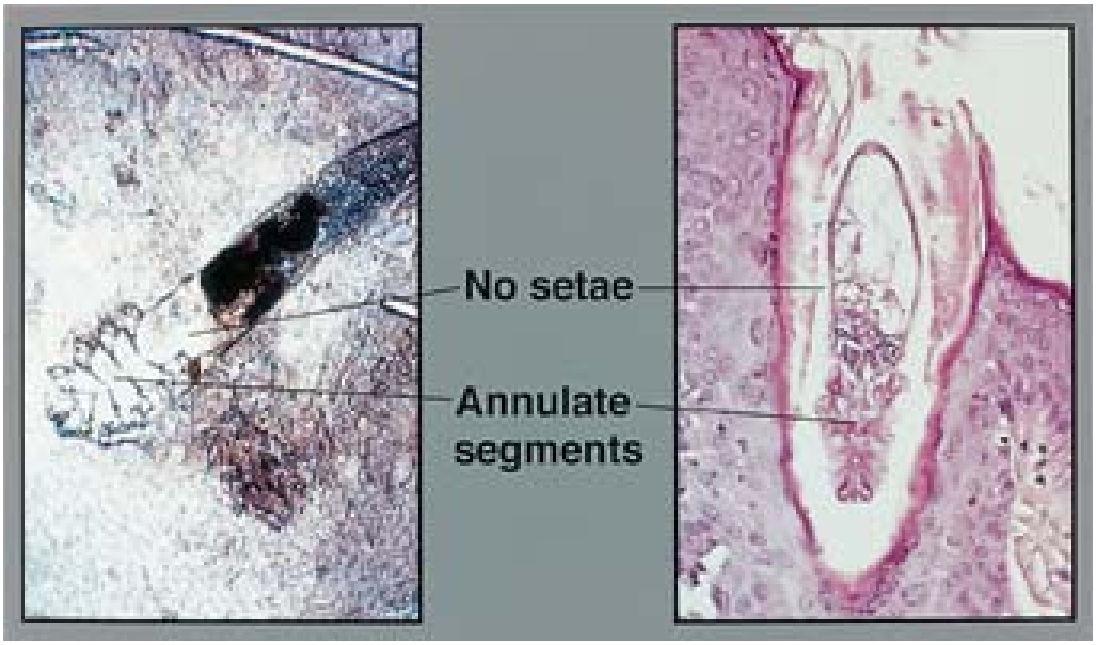
Demodex canis

Canine demodicosis, also referred to as red mange or demodectic mange, is caused by *Demodex canis*, a mite present in small numbers on most healthy dogs. Demodicosis occurs when large numbers of *D canis* colonize hair follicles resulting in localized or generalized demodicosis. Localized demodicosis is generally self-limited and affects dogs 3 to 6 months of age. This form of demodicosis presents as erythematous patches and plaques with alopecia and fine scale, most commonly involving the periorbital area and commissures of the mouth. Generalized demodicosis is not self-limited; it affects dogs 2 to 5 years of age and may be fatal if untreated. Lesions are widespread, involving the head, trunk, and legs, with partial sparing of the abdomen. Lesions are initially characterized by erythematous coalescing patches and plaques with alopecia, folliculitis, and follicular hyperkeratosis. As the disease progresses, secondary bacterial invasion occurs, resulting in crusted pyogenic plaques and generalized lymphadenopathy.

Demodicosis occurs more often in purebred dogs, especially Great Danes and Scottish Terriers. Because most dogs harbor *D canis* but never manifest demodicosis, the obvious question is why. Immunosuppression is a possible answer. This initially appears quite logical because it has been long recognized that dogs with cancer, severe metabolic diseases, or those being treated with immunosuppressive medications have a greater risk of developing disease. However, immunosuppression in the usual sense does not adequately explain why some dogs develop demodicosis and others do not. It seems more likely that demodicosis results from a mite-specific immunosuppression induced by *D canis*. It also is possible that a hereditary



(A) *Demodex folliculorum* mites. (B) Numerous mites in a scalp scraping from a patient with demodectic alopecia (H&E, original magnification ×100).

D canis–specific T-cell defect is involved in some cases. The reason some dogs manifest demodicosis and others do not is no doubt related to the interaction of host immune function with numerous other host and parasite factors.

Human Demodectic Alopecia

Human demodectic alopecia appears to be a real entity. This condition bears a striking resemblance to canine demodectic mange, combining features of alopecia, erythema, and scaling. Large numbers of *Demodex* mites are noted in biopsies or smears from affected follicles (Figure, B). Permethrin 5% has been successful in treating some patients with this condition.

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