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Human Demodex Mite:
The Versatile Mite of Dermatological Importance

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Abstract
Demodex mite is an obligate human ecto-parasite found in or near the pilo-sebaceous units. *Demodex folliculorum* and *Demodex brevis* are two species typically found on humans. *Demodex* infestation usually remains asymptomatic and may have a pathogenic role only when present in high densities and also because of immune imbalance. All cutaneous diseases caused by *Demodex* mites are clubbed under the term demodicosis or demodicidosis, which can be an etiological factor of or resemble a variety of dermatoses. Therefore, a high index of clinical suspicion about the etiological role of *Demodex* in various dermatoses can help in early diagnosis and appropriate, timely, and cost effective management.

Keywords: *Demodex*, *demodicosis*, *demodicidosis*, *ecto-parasite*

Introduction

What was known?
Demodex mite infestation usually remains asymptomatic, but may be an important causative agent for many dermatological conditions.

Demodex, a genus of tiny parasitic mites that live in or near hair follicles of mammals, are among the smallest of arthropods with two species *Demodex folliculorum* and *Demodex brevis* typically found on humans. Infestation with *Demodex* is common; prevalence in healthy adults varying between 23-100%. [1,2] *Demodex* infestation usually remains asymptomatic, although occasionally some skin diseases can be caused by imbalance in the immune mechanism. In this article, we have described the mite and have highlighted its dermatological importance.

General considerations of Demodex
Demodex mite is an obligatory human ecto-parasite, and it is resident in or near the pilo-sebaceous units.[3] About 65 species of *Demodex* are known. Two species *D. folliculorum* and *Demodex brevis*, collectively referred to as *Demodex*, are typically found on humans, occurring in 10% of skin biopsies and 12% of follicles[4,5] [Figure 1]. Identification of these mites dates back to 1841-42 for *D. folliculorum* by Simon and 1963 for *D. brevis* by Akbulatova. [4,6,7]

Figure 1
Demodex mite, an obligatory human ecto-parasite resides in or near the pilo-sebaceous units

Species/genus identification

Demodex is a saprophytic mite that belongs to family Demodicidae, class Arachnida, and order Acarina. [8]

Morphology

Adult *D. folliculorum* mites are 0.3-0.4 mm in length and that of *D. brevis* are slightly shorter of 0.15-0.2 mm length,[2] with females somewhat shorter and rounder than males [Figure 2]. This makes them invisible to the naked eye, but, under the microscope, their structure is clearly visible. It has a semi-transparent, elongated body that consists of two fused segments. Eight short, segmented legs are attached to the first body segment. The eight legs of this mite move at a rate of 8-16 mm/h and this is mainly done during the night as bright light causes the mite to recede into its follicle. The body is covered with scales for anchoring itself in the hair follicle and the mite has pin-like mouth parts for eating skin cells, hormones, and oils (sebum) accumulating in the hair follicles.[2,4,5]

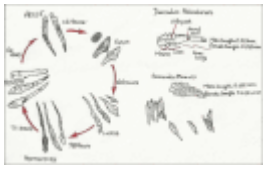

Figure 2
Morphology and life cycle of the *Demodex* mite

Sites of involvement

Demodex is an ecto-parasite of pilo-sebaceous follicle and sebaceous gland, typically found on the face including cheeks, nose, chin, forehead, temples, eye lashes, brows, and also on the balding scalp, neck, ears.[4,5] Other seborrheic regions such as naso-labial folds, peri-orbital areas, and less commonly upper and medial region of chest and back are also infested.[2] They may also be found on penis, mons veneris, buttocks, and in the ectopic sebaceous glands in the buccal mucosa.[2]

D. folliculorum is more commonly localised to the face, while *D. brevis* is more commonly found on the neck and chest.[9] Infestation with *D. folliculorum* is more common than with *D. brevis*, but the latter has wider distribution on the body.[4] *D. folliculorum* is usually found in the upper canal of the pilo-sebaceous unit at a density of ≤ 5/sq cm[4] and uses skin cells and sebum for nourishment.[3,10] Several mites, with heads directed toward the fundus, usually occupy a single follicle.[4,11] *D. brevis*, on the other hand, burrows deeper into the sebaceous glands and ducts and feeds on gland cells.[5] Penetration of *Demodex* into the dermis or, more commonly, an increase in the number of mites in the pilo-sebaceous unit of > 5/sq cm,[4] is believed to cause infestation, which triggers inflammation.[10,12] Some authors consider the density of > 5 mites per follicle as a pathogenic criterion.[10,13]

Life cycle

Female *Demodex* are somewhat shorter and rounder than males. Both male and female *Demodex* mites have a genital opening and fertilisation is internal. Mating takes place in the follicle opening and eggs are laid inside the hair follicles or sebaceous glands. The six-legged larvae hatch after 3-4 days, and the larvae develop into adults in about 7 days. It has a 14-day life cycle [6] [Figure 2]. The total lifespan of a *Demodex* mite is several weeks. The dead mites decompose inside the hair follicles or sebaceous glands.

Age/sex consideration of infestation

The number of *Demodex* mites present in the lesion increases with age.[9] The prevalence of infestation with *Demodex* mites is highest in the 20-30 years age group, when the sebum secretion rate is at its highest.[14] Older people are also more likely to carry the mites.[15] Demodicosis is exceptionally seen in children aged <5 years.[16,17] Presumably, *Demodex* passes to newborns through close physical contact after birth; however, due to low sebum production, infants and children lack significant *Demodex* colonisation.[5]

Infestation of both species is more common in males than in females, with males more heavily colonising than females (23% vs 13%) and harbouring more *D. brevis* than females (23% vs 9%).[4]

Mode of transmission
The mites are transferred between hosts through contact of hair, eyebrows, and sebaceous glands on the nose.

Methods of detection on body
Demodex is not easily detected in histological preparations; therefore, skin surface biopsy (SSB) technique with cyanoacrylic adhesion is a commonly used method to measure the density of *Demodex*.[10] It allows the collection of the superficial part of the horny layer and the contents of the pilo-sebaceous follicle;[18] however, it can fail to collect the complete biotope of *D. folliculorum*.[12]

Other sampling methods used in assessing the presence of *Demodex* by microscopy include adhesive bands, skin scrapings, skin impressions, expressed follicular contents, comedone extraction, hair epilation, and punch biopsies.[11,19] The resulting number of mites measured varies greatly depending on the method used.[11] With modern, and more sensitive, assays, the prevalence of *Demodex* in skin samples approaches 100%; therefore, mere presence of *Demodex* does not indicate pathogenesis. Rather, more important in diagnosing *Demodex* pathology is the density of mites or their extra-follicular location.[19]

Predisposing factors
Most people are only carriers of *Demodex* mites and do not develop clinical symptoms. Human demodicosis can be considered as a multi-factorial disease, influenced by external and/or internal factors. [20]
One of the factors for the transition from a clinically unapparent colonisation of mites to dermatoses can be the development of primary or secondary immunodepression.[20,21] Primary immune suppression is most probably based on hereditary defect of T cells, subsequently reinforced by substances that are produced by mites and by bacteria, with intact B cell immunity.[20,22,23] The fact that people and animals with immunodeficiency are prone to infestation with *Demodex* mites has been shown repeatedly.[24,25]

Secondary immune suppression predisposing to demodicosis follows corticosteroid, cytostatic therapy, or due to diseases of an immune-compromised nature such as malignant neoplasia, hepatopathies, lymphosarcoma, and HIV infection.[25,26,27,28] There may, however, be factors other than generalised immunosuppression leading to the development of demodicosis.[29] It has been suggested that infestation may be related to genetic predisposition[30] and also with special types of HLA (Human Leukocyte Antigen), although some HLA types are considered to be resistant to demodicosis.[29]

Immunopathogenesis
Pathogenesis of demodicosis and immune response to mite invasion are poorly understood. [31,32] Many views have been put forth [Figure 3] as follows:

Figure 3
Factors involved in pathogenesis

- Altered immune system, especially in immune-deficient individuals, which eventually causes a skin disorder.
- Hypersensitivity against the mite itself; the evidence being that histopathological examination reveals a dermal infiltrate of lymphocytes, eosinophils, and typical granulomas predominantly composed of CD4+ T helper lymphocytes, often distributed around a *Demodex* body.[33]
- Increased readiness of lymphocytes to undergo apoptosis and increased number of NK cells with Fc receptors is correlated with increased mite density.[34]

- Significant decrease in absolute numbers of lymphocytes and T- cell subsets and significant increase in IgM levels have also been found in patients presenting with *demodex*. *Demodex* proliferation and facial skin lesions.[35]
- Antigenic proteins related to a bacterium isolated from a *D. folliculorum* mite, *Bacillus oleronius*, have the potential to stimulate an inflammatory immune response in patients with papulopustular rosacea by increasing the migration, degranulation, and cytokine production abilities of neutrophils.[36,37]

These findings suggest that colonisation of the skin with *Demodex* could be a reflection of immune response of the host to organism. [34]

Clinical manifestation

Demodex mites are present in healthy individuals and may have a pathogenic role when present in high densities.[13] The infestation may be clinically inapparent, but, under favorable circumstances, these mites may multiply rapidly, leading to the development of different pathogenic conditions.[30,38]

All cutaneous diseases caused by *Demodex* mites are clubbed under the term demodicosis or demodicidosis. It remains unknown if *Demodex* is the underlying cause of these conditions or if *Demodex* mite density increases due to inflammation of affected follicles.[39] It is possible that by blocking the hair follicles, it can cause inflammation or allergic reaction or act as vector for other microorganisms.[40]

These conditions are briefly described below:

Rosacea and Demodex rosacea

Demodex may have a direct role in rosacea or may manifest as rosacea like dermatitis [Figure 4a]. Numerous studies have reported elevated *Demodex* density in patients with rosacea. [4,10,11,41,42]

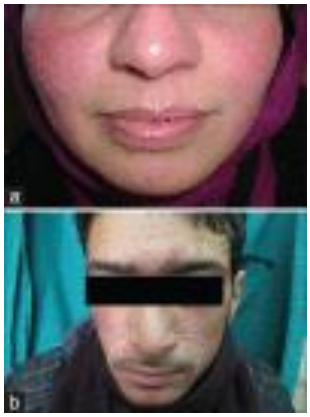

Figure 4
Clinical photograph showing rosacea (a) and steroid induced rosacea (b)

Human demodicosis may manifest as a dry type of rosacea, termed rosacea-like demodicidosis. [43] Rosacea of demodicosis needs to be differentiated from the common rosacea. *Demodex* type rosacea is characterised by dryness, follicular scaling, superficial vesicles, and pustules, while common rosacea is characterised by oily skin, absent follicular scaling, and being more deeply seated. [44]

Another useful feature is the complete resolution of demodicosis on treatment with scabicide crotamiton or lindane. It has been proposed that failure to wash the face and overuse of oily or creamy preparations supplies the *Demodex* mites with extra lipid nourishment, which promotes reproduction of mites in large numbers, which plugs the pilo-sebaceous ducts and leads to appearance of rosacea-like facial eruption. [45]

Non specific facial dermatitis

Patients presenting with nonspecific facial symptoms such as facial pruritus with or without erythema, a seborrheic dermatitis-like eruption, perioral dermatitis-like lesions and papulopustular, and/or acneiform lesions without telangiectasia, flushing, or comedones have been found to have significantly higher median mite density[39,46] [Figure 5].

Demodex dermatitis may in fact be distinct from rosacea and seborrheic dermatitis, as reported by one group [47] and the presence of facial erythema, dryness, scaling, and roughness with or without papules/pustules may be a result of *D. folliculorum* proliferation. [48]

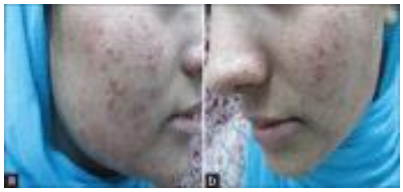

Figure 5
Clinical photograph of *Demodex* induced non specific facial dermatitis

Demodex dermatitis may in fact be distinct from rosacea and seborrheic dermatitis, as reported by one group [47] and the presence of facial erythema, dryness, scaling, and roughness with or without papules/pustules may be a result of *D. folliculorum* proliferation.[48]

Steroid rosacea

The role of *D. folliculorum* in the pathogenesis of topical corticosteroid-induced rosacea is controversial.[49,50] It has been reported that the population of *Demodex* mites is increased in these patients [5,11,51,52] [Figure 4b].

Androgenetic alopecia

Demodex has been implicated in the etiology of AGA. [53] The role of *Demodex* in AGA has been evaluated to be direct in some studies and indirect in others. The possible mechanisms include the following:

- Induction of inflammation by the presence of an immune-active lipase in *Demodex* mite.[54] Nowadays, inflammation has been considered to be involved in pathogenesis of AGA.[39,55] It has been proposed that inflammation reaction in AGA is confined to the surrounding area of sebaceous glands and infundibulum, and follicular infiltration with activated T cells results in induced synthesis of collagen by dermal sheath fibroblasts and ultimately replacement of hair follicle with fibrosis takes place.[56,57]
- Altering local hormone metabolism by the inflammatory reaction.[58]
- Sebaceous glands of alopecia-affected hair follicles become larger and more active under the influence of dihydrotestosterone, producing oils at a faster rate and, hence, become a more suitable environment for *Demodex*. In fact, *Demodex* infestation is considered to be secondary to AGA and not its cause.
- Exhaustion of the hair bulb and shifting of hair cycle from anagen to telogen through long-term invasion by the parasite.[53]

Madarosis

Infestation of pilo-sebaceous components of the eyelids with *D. folliculorum* can also result in loss of eyelashes. [59] *Demodex* mite causes follicular inflammation that produces edema and subsequent easier epilation of eyelashes. It also affects cilia constriction so that lashes become brittle and fall. [60]

Lupus miliaris disseminatus faciei

Several authors suggest that LMDF is a reaction to *D. folliculorum.* … [61] [Figure 6].

Figure 6
A case of clinically and histopathologically proven LMDF

Dissecting folliculitis

The cause of dissecting folliculitis of scalp is not well understood [Figure 7]. It is generally considered to be an inflammatory reaction to components of the hair follicle, particularly microorganisms like bacteria (especially *Propionibacterium acnes*, *Staphylococcus aureus*), yeasts (M Human *Demodex* Mite: The Versatile Mite of Dermatological Importance. [62]

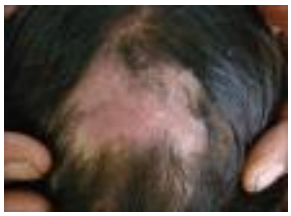

Figure 7
Clinical photograph of dissecting folliculitis leading to cicatricial alopecia

Miscellaneous conditions

Increased number of *Demodex* mites has also been observed in peri-oral dermatitis [Figure 8a], acarica blepharo-conjuctivitis [Figure 8b], grover's disease, eosinophilic folliculitis, papulovesicular facial, papulopustular scalp eruptions, pityriasis folliculorum, pustular folliculitis, *Demodex* abscess, and demodicosis gravis (granulomatous rosacea like demodicosis). [34,39,63]

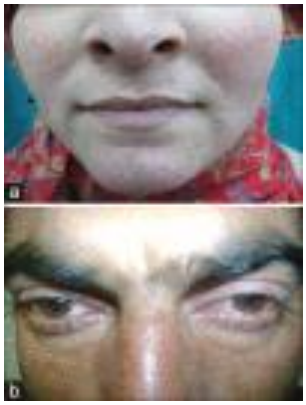

Figure 8
Clinical photograph of peri-oral dermatitis (a) and blepheritis (b)

Other points of importance

As a vector for transmission

Demodex may act as a vector of transmission of various infections from one area of body to another or between individuals by its potential to ingest and transport various microorganisms that are found in its niche, as demonstrated by potassium hydroxide mount of skin scraping from a mycotic plaque, which showed numerous *Demodex* mites containing spores of *Microsporum canis* inside them. [64]

Prevention/treatment of human demodicosis

Demodex can only live in the human hair follicle and, when kept under control, causes no problems. However, to reduce the chance of the mites proliferating excessively, following preventive measures are important:

- Cleanse the face twice daily with non-soap cleanser
- Avoid oil-based cleansers and greasy makeup
- Exfoliate periodically to remove dead skin cells

After clinical manifestations, the mites may be temporarily eradicated with topical insecticides, especially crotamiton cream, permethrin cream, and also with topical or systemic metronidazole. In severe cases, such as those with HIV infection, oral ivermectin may be recommended. [3,48,66] Go to:

Conclusion

Human demodicosis is caused by the clinical manifestation of otherwise asymptomatic infestation of humans by two species of *Demodex* mite, i.e., *D. folliculorum* and *D. brevis*. The etiological role of this versatile mite should be kept in mind as human demodicosis can present as a variety of clinical manifestations mimicking many other dermatoses. This can help in early diagnosis and proper treatment, thereby saving time and at the same time being cost effective.

What is new?

Demodex mite should be considered as an aetiological factor for a number of dermatoses for their early diagnosis and appropriate treatment. Go to:

Footnotes
Source of Support: Nil
Conflict of Interest: Nil. Go to:

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