Scabies infestation in humans is a complex interplay between mite, host, and host environment. New techniques for diagnosis, treatment, and eradication are constantly in flux due to varying presentations of scabetic eruptions, a dearth of especially sensitive and specific measures for diagnosis, resistances to pharmacologic therapy, and disparate regional resources. This review will provide an update on the clinical variations, detection methods, and management options.

**Abstract**

Scabies infestation in humans is a complex interplay between mite, host, and host environment. New techniques for diagnosis, treatment, and eradication are constantly in flux due to varying presentations of scabetic eruptions, a dearth of especially sensitive and specific measures for diagnosis, resistances to pharmacologic therapy, and disparate regional resources. This review will provide an update on the clinical variations, detection methods, and management options.

**What’s new**

Protean clinical presentations

Classic clinical features and symptoms of human scabies infestation include well-known descriptors: a particularly nocturnally pruritic patient, often with a history of institutionalization, group living, immunosuppression, or close contacts with these risk factors; presenting with polymorphic excoriated or crusted pink papules, burrows, nodules, or plaques, particularly in skin folds. The clinical variants including classical papular type, Norwegian/crust type in the immunosuppressed, and rarer nodular and vegetilobullous types, often include a broad differential with some-times subtle and divergent features. For instance, the eruption is reported to resemble droves of dermatoses including atopic dermatitis, seborrheic dermatitis, dermatitis herpetiformis, other arthropod assaults, guttate, plaque, palmoplantar and pustular psoriasis, secondary syphilis and other papulosquamous eruptions, hypersensitivity reactions, and exanthema. Some nodular lesions, particularly in children, may mimic histiocytoses (both Langerhans and non-Langerhans cell types), lymphoma, diaper-based eruptions or infections, or even urticaria pigmentosa, replete with Darier sign. Annular pink plaques mimicking tinea and an adult bullous variant resembling pemphigoid are reported, and cases of overlapping dermatoses like granuloma annulare and elastosis perforans serpiginosa in the vicinity of a scabies mite have also recently been described.

Evolving insight into the pathophysiology of the infestation also demonstrates that scabies is a gateway eruption for new infections, particularly *Staphylococcus* and *Streptococcus* species, likely because the skin barrier is vulnerable. Also, scabies mites can inhibit complement and promote bacterial growth; for example, via a mite gut protein type called peritrophin, the mite can express large amounts of the macrophage migration inhibitory factor (MIF), which may aid in evasion of the host immune response. In addition, because some of the affected patient populations are in developing countries and because some patients, no matter the locale, may be immunosuppressed, the secondary infections can lead to rheumatic heart disease and other postinfectious complications (Figure 1). Recent reports also suggest that patients with a history of scabies may become vulnerable to chronic kidney disease and even other skin eruptions, like pemphigoid variants.

**Diagnostics**

Office-based direct diagnostic measures still prevail for scabies mite identification, as the patients’ extreme pruritus and anxiety surrounding their symptomatology mandate expeditious results. Therefore, initial preferred diagnostic measures include mineral oil slide preparation of a skin scraping using a 15-blade or ‘curette prep’ using a disposable curette, adhesive tape stripping, burrow ink test (performed more often in Europe), and dermoscopy. Another novel in-office modality for scabies identification is in vivo reflectance confocal microscopy which displays: refractive mite body parts, eggs, scybala (Figure 2), visible mite movement, and gut pulsation, the presence or absence of which may determine mite viability after scabicide treatment. In addition, there is serologic testing in development for scabies infestation measurement.
ing Immunoglobulin E (IgE) antibody response to recombinant scabies apolipoprotein antigen (Sar s 14) using enzyme-linked immunoabsorbent assay (ELISA). Further, an evolving molecular test using polymerase chain reaction (PCR) amplification of mite DNA from skin scrapings may identify scabies presence but also may be useful for determining if treatment has been effective after 1 month.  

Of course, traditional skin biopsy may also display classic diagnostic features, such as scabies mites, eggs (or eggshell ‘pigtails’), or scybala in the stratum corneum and granulosum, spongiosis near a burrowed female mite, and an eosinophilic and lymphocytic dermal infiltrate. In addition, there are instances where skin biopsy with direct immunofluorescence of perilesional skin for eruptions resembling autoimmune blistering disease may also be falsely positive in scabies infestations, perhaps relating to an overlapping immune response and the aforementioned risk of pemphigoid development in some patients. Polarized light microscopy may also be helpful for these biopsy specimens, in that the outer sheath of scabies spines, scybala, and at times the mite gut are all polarizable.

Therapeutics

There is no consensus regarding which topical, systemic, and environmental eradication modalities are most efficacious for scabies management. The range of treatment strategies reflect geographic, economic, and feasibility concerns as well as patient age, comorbidities, and medication resistance and toxicities. Most consider permethrin 5% as the most safe and effective first-line therapy, with 2 topical applications one week apart as the most-utilized regimen. Oral ivermectin 200 µg/kg is often employed in crusted scabies and in mass treatment efforts, but it is also being increasingly used for typical presentations. One or two weekly dosages of ivermectin in some studies, however, is considered less effective than permethrin topical, but others conclude that although ivermectin’s response is delayed, its long-term cure rates are superior. Table 1 summarizes the use of these and other current therapies, and Table 2 provides levels of evidence.

**FIGURE 1. Complications of scabies infestation.** Reprinted with permission from Dr. Andrew Steer.

**FIGURE 2. Reflectance confocal microscopy image of scabies—Sarcoptes scabiei mites (asterisks), with their eggs (thin arrows) and droppings (thick arrows).** This material is reproduced with permission of John Wiley & Sons, Inc.
Ivermectin may not be available or licensed for scabies in some countries, and it is not preferred in pregnant or lactating women (in which precipitated sulfur or permethrin topicals are now recommended) or in patients under 15 kg, although recent evidence suggests that it may be safe for smaller children.\(^3^4,3^5\) In recalcitrant crusted scabies patients, it has recently been shown that weekly dosage regimens (in one case 7 doses) of ivermectin until clinical clearance is effective, as is pretreatment of crusted plaques with surgical debridement.\(^3^6,3^7\)

Recently elucidated resistance mechanisms to these common therapies are likely mediated by sodium channel mutations for permethrin, P-glycoprotein-mediated efflux for ivermectin, and increased activity of metabolic enzymes like cytochrome P450 and glutathione S-transferases for both of these medications. However, some studies suggest that combined topical and/or oral therapy regimens may synergistically surmount these pathways. Rates of resistance vary, but this is likely because clinical and laboratory
definition and timeline for cure are not consistent across studies.\(^3^8,3^9\)

Other evolving therapies include moxidectin, a veterinary oral acaricide with a longer half-life than ivermectin (4 days vs 18 hours), which may provide more effective single-dosage therapy.\(^4^0\) Herbal newcomers for scabies management include tea tree oil, clove oil, neem oil, and aloe vera, which all show some in vitro acaricidal activity but no convincing safety and efficacy data, demarcating their use as adjunctive.\(^3^1\) In recent reports, topical ivermectin may be used as an alternative to permethrin. Also, topical calcineurin inhibitors may be effective in nodular scabies, although these agents may perhaps promote local cutaneous immunosuppression that could incite crusted scabies in vulnerable patients.\(^4^1-4^4\)

The future
The International Alliance for the Control of Scabies was founded in November 2012 and is committed to worldwide scabies eradication,

### TABLE 1. Drugs commonly used to treat scabies

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Dosage</th>
<th>Treatment Regimen</th>
<th>Contraindication</th>
<th>Advantages</th>
<th>Disadvantages</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Permethrin</td>
<td>5% cream</td>
<td>Rinsed off after 8-12 hrs</td>
<td>Effective, well tolerated, safe</td>
<td>Itching and stinging on application</td>
<td>Second application often routinely prescribed 1 week after the first application</td>
<td></td>
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<tr>
<td>Lindane</td>
<td>1% lotion or cream</td>
<td>Rinsed off after 6 hrs</td>
<td>Pregnant women, infants, seizure disorders</td>
<td>Effective, inexpensive</td>
<td>Cramps, dizziness, seizures in children</td>
<td>Withdrawn in the European Union because of neurotoxicity concerns</td>
</tr>
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<td></td>
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</tr>
<tr>
<td>Benzyl benzoate</td>
<td>25% ointment</td>
<td>Rinsed off after 24 hrs (once or several times)</td>
<td>Pregnant women and infants (limit duration of use to 12 hrs)</td>
<td>Effective, inexpensive</td>
<td>Can cause severe skin irritation</td>
<td>Not currently available in Canada, approved in Europe</td>
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<tr>
<td>Esdépallétrine (bioallethrin)</td>
<td>0.6% aerosol</td>
<td>Rinsed off after 12 hrs</td>
<td>People with asthma</td>
<td>Well tolerated, safe for infants</td>
<td>Questionable efficacy</td>
<td>Not available in Canada, often used on scabies nodules in children</td>
</tr>
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<tr>
<td>Crotamiton</td>
<td>10% ointment</td>
<td>Rinsed off after 24 hrs and then reapplied for an additional 24 hrs</td>
<td></td>
<td></td>
<td>Safe for infants, pregnant and breastfeeding women</td>
<td>Questionable efficacy, skin irritation</td>
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<tr>
<td>Precipitated sulfur</td>
<td>2%-10% precipitate in petroleum base</td>
<td>Rinsed off after 24 hrs and then reapplied every 24 hrs for the next 2 days (with a bath taken between each application)</td>
<td></td>
<td></td>
<td>Safe for infants, pregnant and breastfeeding women</td>
<td>Questionable efficacy, skin irritation</td>
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<td></td>
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<td></td>
</tr>
<tr>
<td>Ivermectin</td>
<td>Pills</td>
<td>200 µg/kg repeated on day 14</td>
<td>Children &lt;15 kg; pregnant or breastfeeding women</td>
<td>Good patient compliance</td>
<td>Expensive</td>
<td>Not approved in many countries</td>
</tr>
</tbody>
</table>

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advocacy, and research. It is comprised of clinicians from high-prevalence areas, including public health physicians, policy makers, and researchers. In recent years, a group of 167 parasitologists from 88 countries called the Sarcoptes World Molecular Network was formed which aims to integrate and optimize molecular and clinical research, epidemiology, and control efforts for this disease in humans and animals. In the throes of a decade where many genomes, including that of Sarcoptes scabiei, are being sequenced, these collaborations and innovations help to mount a robust, molecularly specific, and geography-germane response to a global public health burden that enters any clinician’s office on any given week.

References

Dermatoses associated with mites other than Sarcoptes

Kimberly M. Ken, BA; Solomon C. Shockman, MD; Melissa Sirichotiratana, MD; Megan P. Lent, MD; and Morgan L. Wilson, DVM, MD

Abstract

Mites are arthropods of the subclass Acari (Acarina). Although Sarcoptes is the mite most commonly recognized as a cause of human skin disease in the United States, numerous other mite-associated dermatoses have been described, and merit familiarity on the part of physicians treating skin disease. This review discusses several non-scabies mites and their associated diseases, including Demodex, chiggers, Cheyletiella, bird mites, grain itch, oak leaf itch, grocer’s itch, tropical rat mite, snake mite, and Psoroptes.

Demodex

There are over 100 species within the genus Demodex, with many residing as commensals or ectoparasites in the skin and pilosebaceous units of a variety of mammalian species. Demodex is the name given to disease related to Demodex mites. The most well-known example is demodectic mange, which occurs in dogs in association with increased numbers of Demodex canis, often in the setting of impaired immunity, and presents with alopecia, scale, and dermatitis. This and other forms of demodicosis in animals are not known to be transmissible to humans. Although well-established in dogs, the role of Demodex in human skin disease has been difficult to clearly define.

Two species of Demodex mites, Demodex folliculorum and Demodex brevis, are saprophytes found on normal human skin, and colonize nearly 100% of humans by adulthood. Demodex folliculorum is 0.3-0.4 mm in length, and resides in the superficial part of the follicular infundibulum, where its head faces the deeper portion of the hair follicle. Demodex brevis is slightly shorter, has a pointed posterior segment, and is found in sebaceous and meibomian glands.

Proposed mechanisms of Demodex pathogenesis include mite blockage of hair follicles and sebaceous ducts leading to reactive hyperkeratosis, stimulation of host immune reactions in response to the mites and their waste, foreign body granulomatous reaction to the chitinous skeleton of the mite, and mites serving as a vector for bacteria. Since Demodex is part of the normal human skin fauna, it can be difficult to determine its significance when identified in the setting of skin disease. Potassium hydroxide (KOH) preparations can be used to visualize mites (Figure 1), but the number expected in normal skin relative to diseased skin is unclear. Histopathologically, reported cases of demodicosis have shown perifollicular and perivascular lymphohistiocytic infiltrates, neutrophils, and numerous Demodex mites in follicular infundibulum and within infundibular pustules. Dermatoscopic findings of demodicosis include whitish Demodex tails projecting from follicular ostia and brown plugs surrounded by erythematous halos in dilated follicular openings.

A noninvasive standardized skin-surface biopsy (SSSB) technique has been used to assess the density of mites in demodicosis compared with normal skin. A density of greater than 5 mites/cm² in SSSB is considered indicative of demodicosis. In clinical practice, the pathogenic role of Demodex is most convincingly supported when patients with disease in Demodex-colonized areas and a high density of Demodex mites are cured with acaricidal treatments.
Demodex has been implicated in multiple dermatoses. It has been suggested that there is an increased risk in patients older than 50 years, a history of topical steroid use, immunosuppression, or debilitation; however, most reported patients with demodicosis are in good general health.3,4,9,10

Pityriasis folliculorum presents with diffuse faint facial erythema, burning and itching sensations, and a dry “nutmeg grater” appearance resulting from follicular plugging by scales.11 Primarily affecting women, pityriasis folliculorum is often preceded by a history of infrequent use of soap for facial washing together with the use of heavy creams and make-up.9

Rosacea-like demodicosis presents very similarly to conventional rosacea, with erythematous papulopustular lesions.7,9 Other findings include infundibular mite infestation and superficial scale, as well as resistance to usual rosacea therapies.9 Granulomatous rosacea-like eruption, also known as demodicosis gravis, presents with dome-shaped papules without observable pustules.7 Histological, dermal caseating granulomas with phagocytized mite remnants are seen.9

Demodex-associated pustular folliculitis has been described as antibiotic-unresponsive folliculitis, with numerous mites in KOH skin scrapings.12 Perioral dermatitis associated with Demodex infestation presents as papulopustular lesions limited to the perioral areas.9 Demodex abscess has been described as a protracted course of confluent erythematous papules, pustules, and deep nodules on the face, with many Demodex mites seen on skin scrapings.13 Other skin diseases that have been described in association with Demodex are blepharitis, alopecia, and external otitis with chronic pruritus.4,15

There is no standard therapy for human demodicosis. Medications used successfully in case reports have included ivermectin and topical permethrin, selenium sulfide, crotamiton, and metronidazole.4,7,14

Harvest mites (chiggers)

Chigger (Figure 2) is an alternate name for larval mites of the family Trombiculidae. Globally, this family comprises many species, which are also known by common names, such as harvest mites, mower’s mites, red bugs, berry bugs, and scrub itch mites.16,17 In North America, Trombicula is the primary genus of medical interest.

Bites are often sustained during the summer or early autumn. Chiggers abound in moist, grassy or bushy terrain, and may be concentrated in short to long grass transition zones or at the edges of forests.18-20 Female mites lay clusters of eggs on low vegetation or damp ground. Subsequent to hatching, the tiny yellow to red 6-legged larval mite crawls onto vegetation, where it waits for a host to pass by. A variety of mammals, birds, and reptiles are the preferred hosts, however, humans are also readily infested.17,19 Measuring less than 1 millimeter, the larvae often go unnoticed by affected humans. The mite becomes engorged after 1-4 days of feeding19 and drops off to continue maturation into an 8-legged free-living nymph and then an adult.

On biting, a chigger penetrates the host’s upper dermis with cheleterae before injecting saliva. The saliva solidifies into a stylostome, or feeding tube, attached to the mouthparts, through which salivary enzymes digest host tissues.21 Initial lesions appear as small papules or wheals which may progress into pustules, vesicles, or bullae. The pruritus is severe, and excoriations are often prominent. Lesions have a characteristic distribution, following a circumferential pattern in areas where clothing is constrictive, such as sock bands and waistbands. Penile and scrotal lesions are also common. Boys may experience “summer penile syndrome” with seasonal penile swelling, pruritus, and dysuria.22

In Asia and islands of the south Pacific and Indian Oceans, members of the genus Leptotrombidium may act as vectors for scrub typhus (Tsutsugamushi fever) when the causative bacterium Orientia tsutsugamushi is passed into the host via the mite’s salivary secretions. At the site of the larval bite, an eschar forms in the majority of primary cases. Other manifestations may include fever, chills, headache, lymphadenopathy, rash, pneumonitis, and encephalitis.23,24 Laboratory data have raised questions as to whether these mites may also serve as vectors for other viral and bacterial pathogens; however, the clinical significance of these findings remains uncertain.25-27

Prevention is the optimal defense against chigger bites. Using DEET (N, N diethyl-meta-toluamide) repellent and/or treating clothing with permethrin is effective against various arthropods, including chiggers.28,29 Wearing clothing treated with permethrin reduces chigger attachments,29 and the repellent effect can last for multiple days and through multiple washings. Products containing citronella oil, jojoba oil, tea tree oil, geranium oil, lemon grass oil, and clove oil have also demonstrated efficacy as chigger repellents.30,31 Treatment for cutaneous eruptions is aimed at symptomatic relief with topical antipruritics or topical anesthetics. Potent topical corticosteroids may be helpful, but their benefits are delayed and may require occlusion. If topical therapy is unsuccessful, intralesional corticosteroids may be effective.

Cheyletiellosis

Cheyletiella yasguri, C. blakei, and C. parasitovorax are non-burrowing mites that parasitize dogs, cats, and rabbits, respectively,32,33 and may cause dermatitis in humans who have contact with

![FIGURE 2. Harvest mite (chigger). Photo courtesy of Dirk Elston, MD.](image-url)
Dermatoses associated with mites other than Sarcoptes

Cheyletiella live in the keratin layer of the epidermis and consume surface debris and tissue fluids. The ova attach to the host animal’s hair, and are smaller than the average louse nit. The adult mites measure approximately 0.4 mm, and are barely visible to the naked eye as small white specks on the host animal’s hair or skin, leading to their description as “walking dandruff”. Microscopically, they are similar in size to scabies mites, but have a somewhat more elongated body and characteristic hook-shaped accessory mouthparts (Figure 3).

A history of a recently introduced pet in the household may be a clue to the diagnosis. Infested pets may be asymptomatic or have a mild dermatitis, often with dry, white scales on the dorsum of the back. Affected humans may develop a more prominent dermatitis, with grouped erythematous, pruritic papules. Occasionally, apical vesicles, bullae or urticarial wheals can be noted. The rash is commonly found in areas which have been in direct contact with the source animal, such as the chest, abdomen and upper extremities. Systemic hypersensitivity to Cheyletiella blakei has been reported, with associated peripheral blood eosinophilia and joint pain.

Diagnosis involves examination of the source animal by a veterinarian, with identification of Cheyletiella in brushings, scrapings, or acetate tape preparations. Treatment of Cheyletiella dermatitis primarily involves acaricidal treatment of the source animal(s) by a veterinarian. As the mites do not establish a sustained infestation in humans, mites are not found on scrapings from affected humans, and acaricidal treatment is typically not necessary, with spontaneous resolution expected within a few weeks of cessation of exposure. Topical anti-pruritic or anti-inflammatory agents can be used symptomatically if needed.

Bird mites

Gamasoidosis is the term used for human skin disease caused by bird mites. The avian mites most commonly affecting humans are the chicken mite or red mite (Dermanyssus gallinae), the northern fowl mite (Ornithonyssus sylviarum; Figure 4), and the tropical fowl mite (Ornithonyssus bursa). These mites parasitize chickens and other domestic fowl, as well as wild and pet birds, including pigeons, starlings, finches, ducks, turkeys, wrens, parakeets, and canaries. When the preferred avian hosts are absent, humans and other mammals may be infested. D. gallinae mites are less than 1 mm in diameter, and are clinically barely visible as tiny brown to red dots. They hide in nests, cracks, and crevices during the day, and attach temporarily to the host bird at night to feed. O. sylviarum, in contrast, spends essentially its entire life on the host bird. It is likewise barely visible to the naked eye, and has been described by patients as tiny “black dots” when observed in the household environment.

Disease in humans occurs in several situations. Commonly, agricultural workers handling infested poultry are affected. Secondly, wild birds often nest on or near window sills, balconies, eaves, rooftops, and attics. When young birds leave the nest, the mites remaining in the nest will then seek a new host, and may enter homes through windows, air conditioners, or ventilation ducts. Humans who collect or otherwise intentionally handle bird nests may be directly infested. Bird mites may also parasitize small mammals, and gerbils have served as a source for human infestation.

The dermatitis is non-specific, consisting of widespread pruritic papules, often on uncovered areas. In contrast to scabies, the eruption often spares the interdigital spaces and genitalia. Mites have been identified directly on humans in rare cases; however, it is more typical that they take a blood meal and then leave the human, such that mites are not found during skin examination. Investigation for an environmental reservoir, such as a bird, abandoned nest, or pet rodent, is critical. It is noted that Ornithonyssus spp. are typically found on the host animal, while examination of the host animal’s environment (cages, bedding, nests) is necessary in order to identify D. gallinae. Eradication of nests and treatment of the environment with an appropriate acaricide is recommended for control of Dermanyssus, whereas treatment of the affected birds and/or elimination of exposure to the source birds is necessary for Ornithonyssus spp. When mites are found on humans, topical permethrin may be of benefit. Topical corticosteroids are of variable benefit for symptomatic relief.

**FIGURE 3.** Cheyletiella. Photo courtesy of Dirk Elston, MD

**FIGURE 4.** Northern fowl mite (Ornithonyssus sylviarum). Photo courtesy of Dirk Elston, MD.
Grain itch/straw itch

*Pyemotes ventricosus* is responsible for most cases of “grain itch” or “straw itch”. This mite is small (0.16-0.22 mm), and is barely visible to the naked eye as a white speck. It is a primarily a parasite of insects found on wheat and other grain-producing plants; however, it will attack humans if its normal hosts are in short supply.\(^4^2\)

It has been found in most of the United States, with most cases of grain itch reported in midwestern or southern states. *P. tritici* has been reported to cause similar outbreaks in the Middle East.\(^4^3\)

Cases of human disease typically occur in agricultural workers or others in direct contact with wheat straw or a variety of grains. Within hours of exposure, affected individuals develop pink to red wheals surmounted by vesicles, which evolve to pustules.\(^4^4\) The lesions are intensely pruritic, and often excoriated. Some patients are severely affected, with 200-300 bites. Associated symptoms can include fever, headache, vomiting, and lymphadenopathy. Other arthropod bites, rickettsialpox, and varicella are among the clinical differentials of dermatitis.\(^5^2\) as well as other rodent hosts. \(^5^2,5^3\)

Affected individuals again lived in proximity to pin oak trees with marginal leaf fold galls containing *P. herfsi* (Figure 6).\(^4^7\)

The oak leaf itch mite, approximately 0.2 mm in length, is difficult to see with the naked eye. Due to its small size, the mites can be carried by the wind, and can pass through window screens. Although oak leaf itch mites may bite humans, they do not establish an ongoing infestation, and spontaneous resolution of lesions is expected. In areas where this mite is recognized as a problem, preventative strategies include use of light-colored, tight-fitting clothing, permethrin-treated clothing, and an application of DEET (N,N-diethyl-meta-toluamide).

**Grocer’s itch**

Mites of several genera are common contaminants of stored cereals, fruits, cheeses, and meat. Dermatitis occurring in individuals handling such items has been referred to as “grocer’s itch”. Specifically, *Carpoglyphus passularum* has been reported to cause an eruption of pruritic red papules in a dock worker exposed to infested figs.\(^4^8\) An irritant contact-like dermatitis has been observed in workers handling cheese contaminated with *Tyroglyphus* (Tyrophagus) mites,\(^4^9\) which are likewise the cause of copra itch,\(^5^0\) and have resulted in dermatitis after contact with cured ham.\(^5^1\)

**Tropical rat mite**

The tropical rat mite (TRM), *Ornithonyssus bacoti*, is a hematophagous external parasite of rodents. The term “tropical” is misleading, as it is found in both tropical and temperate climates, and has been identified on all continents other than Antarctica\(^5^2\) as well as in the majority of the United States.\(^5^3\) The principle hosts are the wild brown rat and black rat, but other rodents will also suffice, such as mice, hamsters, and gerbils.\(^5^2,5^3\) When the availability of these hosts decreases, as may occur during rodent extermination programs, humans can become an alternative host, leading to epidemics of dermatitis.\(^5^2\)
The adult mite measures 0.6 mm to 1.1 mm. The mites feed briefly at night, and hide in dark cracks and crevices during the day. The bite is not immediately recognized by the host, and can lead to dermatitis, presumably due to a reaction to the saliva and mouth parts. The rash typically consists of pruritic, erythematous papules or wheals, often on the back, extremities, and waistline. Diagnosis of TRM dermatitis requires a thorough inspection of the environment for host rodents and mites. Since the mites spend the majority of their time in the environment, they will not necessarily be found on the host animal, and are rarely found on affected humans. Treatment involves extermination of the mites, as well as control of the associated rodent host population. Any small mammalian pets should be examined by a veterinarian and treated appropriately. Since the mites do not establish a sustained infestation in humans, acarical treatment of humans is typically not necessary. Topical corticosteroids may be used for symptomatic treatment of the dermatitis.

Snake mite

The snake mite (Ophionyssus natricis) is a 0.6-1.3 mm hematophagous parasite of snakes and lizards, and has been reported to cause a papular and vesicular eruption in a family with an infested pet python. Dusting of the snake and its immediate environment with pyrethrum powder was effective in ending the infestation.

Psoroptes

Psoroptes ovis is a non-burrowing ectoparasite of sheep, which causes psoroptic mange (sheep scab), characterized by crusts, alopecia, and emaciation in affected animals. The disease is believed to be eradicated in some nations, including the US, Canada, Australia, and New Zealand. There are rare reports of human infestation in individuals who have contact with sheep, often presenting as dermatitis on the upper or lower extremities. Source animals should be treated by a veterinarian. The optimal therapy for humans is uncertain, although application of a topical acaricide such as 5% permethrin cream has been recommended.

Conclusion

Patients with papular urticaria are a common clinical challenge, particularly when the history does not reveal a known source of arthropod bites. In addition to common culprits such as fleas, mosquitoes, and bed bugs, it is useful to consider infestation with non-scabies mites. Inquiry regarding occupational and environmental factors is critical, and in many cases, the responsible mite is found not on the human patient, but rather in the environment or on domestic or wild animals in the patient’s surroundings. When a source of exposure can be identified, elimination or reduction of exposure is often the key step in management of the clinical disease. Finally, when patients present with papulopustular facial eruptions unresponsive to conventional rosacea therapy, consideration of demodiosis may be warranted.

References


