Scabies and Pediculosis Pubis: An Update of Treatment Regimens and General Review

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Ectoparasites continue to be a common cause of skin disease throughout the world. The present article dissects the epidemiological profile and treatment of both Sarcoptes scabiei variant hominis and pediculus pubis.

SCABIES

Introduction and methods. Scabies is caused by the mite Sarcoptes scabiei var. hominis. It is estimated that 300 million cases of scabies occur worldwide each year [1], and scabies continues to be a major public health problem in resource-poor areas. A nuisance infection, scabies can cause significant morbidity resulting from secondary bacterial infections. The present article updates previously published treatment-guideline recommendations and will provide a brief overview of the characteristics of the mite, the epidemiological profile of infestation, and the manifestations of disease. To update current approaches to diagnosis and treatment, a search was conducted of the English-language literature published between 1 June 2000 and 1 January 2006. A Medline search was conducted using the terms “scabies,” “Sarcoptes scabiei,” “Norwegian scabies,” “crusted scabies,” “ivermectin,” “benzyl benzoate,” “malathion,” “lindane,” and “permethrin.” AIDSLine was searched, excluding Medline listings, for the terms “scabies,” “Sarcoptes scabiei,” “Norwegian scabies,” and “crusted scabies.” For each study identified, the study population, treatment, outcome measures, findings, and potential biases in study design and analysis were evaluated. This article is an update of an article by Wendel and Rompalo [2] published after the publication of the Centers for Disease Control and Prevention’s sexually transmitted diseases treatment guidelines in 2002.

Etiologic and epidemiological profiles. Sarcoptes scabiei was first identified in the early 1600s but was not recognized as the etiology of the skin disorder scabies until the 1700s [3]. The mite is an obligate human parasite that lives in burrowed tunnels in the stratum corneum of the epidermis. It completes its entire life cycle on humans. Pregnant female mites lay 10–25 eggs in burrows that can be up to 1 cm long and can reach to the boundary of the stratum granulosum [4]. In 3–4 days, the eggs hatch, and the larvae mature on the skin surface. The total length of the life cycle is 30–60 days. On average, ~10–15 female mites live on an infected host, but the number of mites can reach millions in humans with crusted scabies.

The burden of disease is highest in tropical countries where scabies is endemic. In regions other than tropical countries, there is limited evidence of a cyclic prevalence of disease. Epidemiological studies involving Israeli soldiers and regional studies in England and Denmark suggest a 20–28-year pattern of disease in these groups [5–8]. A higher burden of disease appears to be related to crowded living conditions. Some studies suggest higher rates of disease in urban areas and an increased incidence of disease during winter months [6–8]. Scabies disproportionately affects women and children [6, 8].

Such institutions as hospitals, nursing homes, and long-term care facilities are sites of epidemic scabies infestations. In 1992, a study evaluating 130 Canadian long-term health care facilities found that scabies infestations had occurred in 20% of such facilities during a 1-year period [9]. Facilities that are older in age, larger
in size, and have a low ratio of beds to health care workers have a higher risk of scabies infestations [9].

Certain populations are at particularly high risk of developing severe, or crusted scabies. This form of hyperkeratotic scabies infection was first described in Norway in patients with leprosy [10]. Patients receiving systemic or potent topical glucocorticoids, organ transplant recipients, mentally retarded or physically incapacitated individuals, HIV-infected or human T-lymphotropic virus 1–infected individuals, and individuals with various hematologic malignancies are at risk of developing crusted scabies [11]. Of interest, crusted scabies is also seen in Aborigines of rural Australia without identifiable immunocompromise [12]. Compared with normal scabies, crusted scabies is highly infectious and is characterized by a much higher burden of mites in the infected individual.

**Clinical manifestations.** Patients with scabies usually complain of pruritus that is most severe at night. Occasionally, patients are asymptomatic. Skin lesions most commonly involve the interdigital spaces and the flexor surfaces of the wrist, axillae, waist, feet, and ankles [4]. In women, the area around the nipple of the breast may be affected, as can the scrotum and penis in men [4]. The initial infection is asymptomatic, with symptoms developing after 3–6 weeks in association with the development of an immune response to mites and their excrement. Reinfestation is associated with a brisk immune response and a rapid onset of symptoms within 24–72 h [13].

The most characteristic lesion of scabies infestation is the burrow, the excavated tunnel in which the mite lives. These burrows are usually thin, curvy, elevated tracts that measure 1–10 mm [4]. Other skin manifestations include papules, blisters, eczematous changes, and nodules [8, 14]. In patients with crusted scabies, the lesions are psoriasiform or warty and can be accompanied by nail hyperkeratosis. The head and neck can be involved, and there may be only mild pruritus. On occasion, there is accompanying eosinophilia and lymphadenopathy [11].

**Complications.** Scabies is a common dermatosis that usually results in a mild-to-moderate rash with pruritus. However, significant morbidity occasionally is associated with scabies infestation. The extensive lesions of crusted or bullous scabies can be debilitating, with pain occurring on movement and a significant breakdown in skin integrity developing. In northern Australia, a mortality rate of up to 50% over 5 years was reported, with deaths primarily resulting from secondary sepsis [15]. Secondary bacterial infection is most commonly due to *Staphylococcus aureus*, group A *β-*hemolytic streptococci, or peptostreptococci [16]. Several case reports have documented leukocytoclastic vasculitis complicating scabies, and one report also noted the presence of lupus anti-coagulant [17, 18]. Glomerulonephritis has also been reported to complicate scabies [19]. A pediatric service in Dakar, Senegal, observed 114 cases in 2 years, with only 1 death occurring and with complete resolution of glomerulonephritis occurring in 97% of cases.

**Diagnosis.** A presumptive diagnosis of scabies is based on the clinical presentation of pruritus with skin lesions and identification of a characteristic burrow. Burrows can be best identified by mineral oil or ink enhancement or by tetracycline fluorescence tests [4]. Definitive diagnosis requires microscopic identification of mites or their eggs or feces. This is usually achieved by obtaining skin scrapings at the site of a burrow or under the fingernails. Other possible strategies for diagnosis include skin biopsy, videodermatoscopy, and epiluminescence microscopy. Videodermatoscopy is performed using a video microscope system [20]. In one study, this technique was evaluated and compared with regular skin scraping. Both procedures were performed twice by 2 independent observers. This test can be performed at a magnification of ×1000, and it takes ~5 min to perform. The results of scraping and videodermatoscopy were similar, but 2 cases were only evident on scraping. Thirty-eight patients were evaluated, and 16 had demonstrable microscopic proof of infection [20]. Epiluminescence microscopy allows examination of the skin to the level of the superficial papillary dermis [21]. For 65 of 70 cases of scabies, this technique showed dark triangular structures at the sites of infection. The technique requires ~5 min and has a low false-positive rate [21]. However, the special diagnostic equipment required for videodermatoscopy and epiluminescence microscopy is unlikely to be available at most sites performing primary evaluation, and these methods have not been shown to be more sensitive than routine skin scraping.

**Transmission.** Transmission is by direct skin-to-skin contact. The average host has only 5–10 mites. A patient with crusted scabies can be infected with millions of mites and is therefore far more contagious than a patient with normal scabies [3]. Live mites have been documented in dust samples obtained from patients with normal scabies and have been found on floors, furniture, and bedding [22]. For this reason, fomite transmission of disease is considered to be possible. Some studies have documented survival of mites for >3 days [22–25]. After initial infection, symptoms can take several weeks to develop. In recurrences, symptoms of pruritus may arise within 24 h [11].

**Treatment.** Regimens for the treatment of scabies are presented in table 1. Although not studied in randomized controlled trials, treatment of all close contacts and housemates of persons with scabies and washing bedding, towels, and clothing in warm to hot water are generally recommended. Items that cannot be washed should be isolated from use for ≥3 days. Since 1996, 5 randomized, controlled clinical trials of scabies treatment have been reported in the English-language literature [31–34]. Usha et al. [31] performed an important study addressing the relative efficacy of topical permethrin and oral
Table 1. Scabies treatment options.

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Average wholesale price, US$</th>
<th>Instructions for use</th>
</tr>
</thead>
<tbody>
<tr>
<td>Permethrin 5% (60 g) cream</td>
<td>15.99–40.79</td>
<td>Apply to whole body from the neck down; wash off after 8–14 h</td>
</tr>
<tr>
<td>Lindane 1% (60 mL)</td>
<td></td>
<td>No longer recommended as first-line or alternative therapy because of toxicity; should only be used in clinical situations in which there is an inability to tolerate other therapies; apply thinly to the whole body from the neck down; wash off completely after 8 h</td>
</tr>
<tr>
<td>Lotion</td>
<td>2.75–14.37</td>
<td></td>
</tr>
<tr>
<td>Shampoo</td>
<td>85.99–125.97</td>
<td></td>
</tr>
<tr>
<td>Crotamiton® (10% cream)</td>
<td>62.79</td>
<td>Not an FDA-approved indication; apply to the whole body from the neck down; leave on for 24 h and apply for 2 consecutive days</td>
</tr>
<tr>
<td>Benzyl benzoate (500 mL)</td>
<td>12.70–26.62</td>
<td>Not an FDA-approved indication</td>
</tr>
<tr>
<td>Malathion 0.5% lotion (60 mL)</td>
<td>109.55–143.99</td>
<td>Not an FDA-approved indication</td>
</tr>
<tr>
<td>Sulfur</td>
<td>6.75–27.52</td>
<td>Not an FDA-approved indication; apply thinly to the whole body; leave on 24 h before washing and reapplying for a total of 3 applications</td>
</tr>
<tr>
<td>Ivermectin (3 mg)</td>
<td>103.99–116.99</td>
<td>Not an FDA-approved indication; 200–250 μg/kg given as a single oral dose or 2 doses separated by an interval of 1–2 weeks</td>
</tr>
</tbody>
</table>

**NOTE.** FDA, Food and Drug Administration.

* Data (from 2006) are from Drugstore.com [26], CVS.com [27], and Kerr Drug (North Carolina) [28].

* Data from 1999 Drug Topics Red Book [29].

* 10% cream (croton oil [5 g]) or 10% lotion.

* Data (from 2006) are from GetCanadianDrugs.com [30].

* Not commercially available in the United States.

* Sulfur 6% or precipitated sulfur powder (500 g).

* Twenty tablets.

Ivermectin. They found that a single topical dose of permethrin produced a clinical cure rate for scabies (97.8%) that was superior to that produced by a single dose of ivermectin (70%). However, 2 doses of ivermectin administered at a 2-week interval were as effective as a single dose of topical permethrin. In a comparison of topical lindane and oral ivermectin, 53 patients were enrolled in the study and were randomized to follow a blinded treatment regimen [32]. Only 43 patients completed the study, and final results were determined by as-treated analysis. This study suggested treatment equivalency between these medications after administration of 2 courses of therapy separated by 2 weeks. On clinical evaluation, the rate of cure of signs and symptoms of scabies was 95% for ivermectin and 96% for lindane. This study was limited by low statistical power. Madan et al. [33] compared ivermectin (200 μg/kg as a single dose) with 1% lindane lotion, with >80% of patients who were given ivermectin demonstrating a marked clinical improvement at 4 weeks of therapy, compared with 44% of patients given lindane lotion. Their study suggests that ivermectin may be a better treatment choice than lindane, because of the good clinical response, lower toxicity, and improved adherence associated with ivermectin. A study conducted in Vanuatu compared benzyl benzoate with a single dose of oral ivermectin (200 μg/kg) [35]. There was no statistically significant difference between the findings in the 2 arms in the study. The reported cure rate was low, with a cure rate slightly better than 50% noted 3 weeks after treatment initiation. The study was limited by a 27% rate of failure to return for follow-up, by the single dose of ivermectin administered, and by the short-term follow-up.

Several other clinical studies have been conducted. One retrospective comparative study analyzed the outcomes of 39 HIV-infected inpatients with 60 episodes of scabies treated with benzyl benzoate, ivermectin, or a combination of benzyl benzoate and ivermectin [10]. Benzyl benzoate achieved a cure in 9 (47.4%) of 19 patients; ivermectin, in 10 (62.5%) of 16 patients; and combination treatment, in 4 (100%) of 4 patients. Of interest, a cure was not achieved for any of the 4 patients with crusted scabies who received either treatment alone. All the patients who received combination treatment had been given a diagnosis of crusted scabies, and, for all these patients, a cure was achieved using a combination of topical benzyl benzoate and oral ivermectin. This study provides some preliminary evidence for the use of combination treatment for severe scabies in HIV-infected patients.

Several open-label studies of ivermectin for the treatment of patients with uncomplicated scabies have been published since 1996 [36–39]. The studies evaluated 1–2 doses of ivermectin (200 μg/kg separated by 7 days). In the largest study, which included 120 patients, the clinical response rate, as determined by as-treated analysis, was 88% after administration of 1 dose of ivermectin. After 4 weeks and administration of 2 doses of ivermectin, the cure rate increased to 100% [38]. There have been reports of several other open-label studies that involved...
smaller patient cohorts treated with ivermectin (200 µg/kg) given in 1–2 doses, with cure rates of 76%–100% noted [36, 37, 39]. These studies included few cases of crusted scabies. In a cohort study, 20 patients with crusted scabies were treated with 1–3 doses of ivermectin (200 µg/kg) combined with a topical scabicide and a keratolytic agent. Eight of 20 patients had an initial complete response, and 8 of 10 patients had a response to 3 doses of ivermectin [40]. Finally, in a study of 10 patients with uncomplicated scabies, a complete response was seen in all 10 patients after receipt of 3–4 treatments with topical 1.8% ivermectin cream [41]. In vitro testing of 6 different scabicides (neem, permethrin, benzyl benzoate, ivermectin, lindane, and tea tree oil) showed that all reduced mite survival, with the exception of neem [42]. However, of the 5 treatments that reduced mite survival, the duration of survival was found to be longest with permethrin exposure.

A clear challenge facing scabies treatment in the future is the optimal management of populations with high rates of endemic scabies. In one recent study, community-wide permethrin treatment was followed by treatment of patients with newly diagnosed scabies at all follow-up visits [43]. At 25 months of follow-up, the prevalence of scabies had decreased from ~28% to 7% (P = .002). In Papua, New Guinea, inhabitants of 2 villages were evaluated for filariasis and scabies [44]. The inhabitants of one village were selected to receive ivermectin (400 µg/kg) as a single dose. The inhabitants of the treated village experienced a decrease in the prevalence of scabies (from 87% to 26%) at 5 months of follow-up. These studies suggest a role for community-wide treatment in controlling disease in communities where scabies is endemic; however, additional studies will be needed to clarify the long-term influence of these treatments and their economic feasibility.

Scabies epidemics in long-term care facilities were the focus of several additional cohort studies, which demonstrated good control of the epidemic with the use of 1–2 doses of ivermectin [45, 46]. One of these studies reported that ivermectin treatment was successful after treatment with several different topical scabicides failed [46].

Treatment toxicity. Topical agents have been the most extensively used type of scabies treatment, and, therefore, their toxicity profiles are fairly well defined. Both permethrin and benzyl benzoate have been reported to cause rash and diarrhea [47]. More worrisome are the reports of convulsions and aplastic anemia occurring in association with benzyl benzoate and lindane [48, 49]. According to the World Health Organization 1998 Collaborating Centre for International Drug Monitoring, convulsions have been associated with the use of benzyl benzoate, crotamiton, lindane, malathion, and permethrin. Deaths were reported to occur in association with crotamiton, lindane, and permethrin treatment [49]. In 2003, the US Food and Drug Administration issued a health advisory that lindane should be used only as an alternative therapy if other treatments are not tolerated or if other approved therapies have failed. It is thought that warm baths and extensive dermatitis may increase systemic absorption of lindane. Lindane is not recommended for pregnant or lactating women, for children <2 years of age, or for patients with extensive dermatitis. Given the warnings and concerns about toxicities, lindane is not recommended as a first-line or alternative therapy for scabies. The use of lindane would best be reserved for patients for whom the inability to tolerate other therapies leaves this as a last option.

In a randomized controlled trial of lindane versus ivermectin treatment, all reported adverse effects were mild and transient [32]. Patients treated with lindane more frequently complained of headache, but some patients treated with ivermectin noted abdominal pain and vomiting. One patient treated with lindane developed hypotension. Ivermectin has been well tolerated in all but one study. This study described an outbreak of scabies in a long-term care facility for elderly individuals; in the study, patients received multiple applications of topical scabicides before finally receiving treatment with a single dose of ivermectin [50]. Over the following 6 months, there was an increased incidence of death among these 47 patients, compared with age- and sex-matched control subjects. These patients experienced lethargy and anorexia before death. Other investigators argue with the outcomes of the study, pointing out that patients were not matched for comorbidities, such as severity of dementia [51]. No other studies have demonstrated increased mortality occurring in association with a history of ivermectin therapy. In another study, involving ~220 patients in a psychogeriatric nursing home who were treated with ivermectin for a scabies outbreak, the mortality rate in the 6 months after treatment was compared with that occurring in the 30 months before treatment; no significant difference was found [46]. Of note, a rash and a transient increase in pruritus have been reported in the first 3 days after scabetic patients receive ivermectin [37].

Persistent symptoms. All patients should be informed that the rash and pruritus associated with scabies may persist for up to 2 weeks after treatment completion [11]. When symptoms and signs persist for >2 weeks, there are several possible explanations, including treatment resistance, treatment failure, reinfection from family members or fomites, drug allergy, or worsening rash due to cross-reactivity with antigens from other household mites [12, 52].

Poor response and presumed resistance to lindane have been reported elsewhere [48, 53–57]. Treatment failure not related to resistance can be due to faulty application of topical scabicides. Patients with crusted scabies may have poor penetration of scabicides into thick scaly skin and may harbor mites in these difficult-to-penetrable layers. Particular attention must be given to the hyperkeratotic fingernails of such infested pa-
tients. Certainly, to avoid reinfection, it is recommended that all close contacts of patients with scabies be empirically treated. All linens, bedding, and clothing should be washed, if possible, during the time of application of the topical scabicide. Even when treatment is successful and reinfection is avoided, symptoms may worsen secondary to allergic dermatitis. This complication has been seen with several of the topical scabicides. Finally, ordinary household mites may drive persistent symptoms resulting from cross-reactivity between antigens. In a study of 25 patients, scabetic patients had higher skin-prick test results, as determined by the extent of wheal development in response to extracts of mice antigen, and higher levels of IgE to house dust mite and storage mite antigen than did control subjects [52].

**PEDICULOSIS PUBIS**

*Introduction and methods.* Lice are blood-sucking insects that have no free-living stage in their life cycle [58]. Lice primarily infest the head (*Pediculus humanus* var. *capitis*), the body (*Pediculus humanus* var. *corporis*), or the pubic region (*Phthirus pubis*). Head lice are the most common lice. In the present article, we will summarize key characteristics of pediculosis pubis and update the current approaches to diagnosis and treatment. A search was conducted of the English-language literature published between 1 June 2000 and 1 January 2006. A Medline search was conducted using the terms “pubic lice,” “*Phthirus pubis*,” “pediculosis,” “pediculosis pubis,” “permethrin,” and “lindane.” AIDSline was searched, omitting Medline listings, by use of the terms “*Phthirus pubis*,” “pediculosis,” and “pediculosis pubis.” Abstracts from meetings of the Infectious Diseases Society of America and the International Society of Sexually Transmitted Diseases Research, as well as from the joint meetings of the American Sexually Transmitted Diseases Association and the Medical Society of Venereal Diseases, were reviewed for contributory work. For each study, the following characteristics were defined: study dosing, study population, treatment, outcome measures, findings, and potential biases in study design and analysis. Head lice treatment trials were included in our findings.

**Characteristics of lice.** *P. pubis* is 1–3 mm long and has 3 pairs of legs [11]. The life cycle of the female insect is 1–3 months. The adult female lays up to 300 eggs adhering to the hairs at the skin-hair junction. Eggs or nits hatch in 6–10 days. These nymphs then mature to adults within 10 days.

*P. pubis* infests hairs in the pubic area and, occasionally, in areas heavily covered with body hair. They rarely infest eyebrows and eyelashes [59]. The most common symptom of infection is pruritus that is thought to be due to hypersensitivity to feeding lice. Physical findings include visible opalescent nits or live lice and blue macules (maculae ceruleae) at feeding sites.

**Diagnosis and transmission.** Pediculosis pubis is diagnosed by the identification of live lice and/or viable nits. All patients with pediculosis pubis should be thoroughly investigated for other sexually transmitted diseases. Transmission occurs through direct intimate contact [11]. Fomite transmission is unlikely to play a significant role. Condoms do not prevent transmission of *P. pubis*.

**Treatment.** Recommended treatment regimens are listed in Table 2. In addition, all bedding, towels, and clothing should be washed. Patients should be instructed to avoid contact with their sex partner until they have been treated and both individuals have been seen in follow-up. Some patients may require a second application of topical therapy 3–7 days after the initial treatment application [59]. Infestation of the eyelashes should be treated by application of an occlusive ointment, such as petroleum jelly, twice daily for 10 days. Other agents that may be effective in the treatment of *P. pubis* are 0.5% malathion, 0.5%–1% carbaryl, and 0.2% phenothrin [59].

Since 2000, to our knowledge, there have been no new treatment trials published concerning the treatment of pediculosis pubis. There were, however, 3 randomized clinical treatment trials assessing the treatment of head lice. In one comparative

### Table 2. Treatment regimens for pediculosis pubis.

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Average wholesale price, US$</th>
<th>Instructions for use</th>
</tr>
</thead>
<tbody>
<tr>
<td>Perm 1% crème rinse (2 oz)</td>
<td>7.67&lt;sup&gt;a&lt;/sup&gt;</td>
<td>Apply to affected areas and wash off after 10 min</td>
</tr>
<tr>
<td>Lindane 1% shampoo</td>
<td>3.00–14.72&lt;sup&gt;b&lt;/sup&gt;</td>
<td>No longer recommended as first-line therapy because of toxicity; should only be used as alternative therapy because of an inability to tolerate other therapies; apply to affected areas and wash off thoroughly after 4 min</td>
</tr>
<tr>
<td>Pyrethrins with piperonyl butoxide</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2 oz (shampoo)</td>
<td>1.67&lt;sup&gt;c&lt;/sup&gt;</td>
<td>Apply to affected area and wash off after 10 min</td>
</tr>
<tr>
<td>5.5 oz (mousse)</td>
<td>9.20&lt;sup&gt;c&lt;/sup&gt;</td>
<td></td>
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</tbody>
</table>

<sup>a</sup> Warner-Lambert product information.

<sup>b</sup> Data from [29].

<sup>c</sup> Bayer product information.
trial conducted in Florida, comparison of 0.5% malathion and 1% permethrin demonstrated a clearance rate of 98% with malathion, compared with 55% with permethrin [60]. Permethrin coupled with oral trimethoprim-sulfamethoxazole enhanced the efficacy of therapy with permethrin [61]. Combing did not improve clearance in response to therapy with permethrin [62]. One study assessed both the in vitro susceptibility of lice and the in vivo response to topical treatment of lice in children in Bath and Bristol, England. Both cohorts of children had lice that demonstrated a decreased mortality rate in response to permethrin and malathion [63]. Children in Bath were treated with malathion, with a cure rate of 36% noted at 48–72 h. Children in Bristol were treated with permethrin, with a cure rate of 13% noted at 48–72 h. The sample size of this study was only 30 children, but it suggests the presence of dual resistance to permethrin and malathion in these cohorts.

Additional in vitro evaluations were performed by Pollack et al. [64], who sampled head lice from children in Massachusetts, Idaho, and Malaysian Borneo. They found that, among lice in the United States, the mortality rate was ~50% in response to treatment with permethrin. The mortality rate was unaltered by increasing concentrations of drug. Conversely, the mortality rate for lice from Malaysia was 37% when the lowest concentrations of permethrin were used. Their mortality rate increased linearly with increasing permethrin concentrations, to a maximum mortality rate of ~95%. This study used different evaluation times for the US and Malaysian lice, because of baseline differences in survival times. Regardless, this study suggests the possible emergence of drug resistance in head lice in the United States. The concerns raised by these results are amplified by another in vitro analysis demonstrating that lice with resistance to permethrin also have resistance to sumithrin and a newer agent for treatment, deltamethrin [65]. The emergence of drug resistance in head lice throughout the world is of concern, but the implications for treatment of pediculosis pubis are unclear. A case of pubic lice resistant to pyrethrins in vitro was reported, but the patient demonstrated clinical clearance with 5% permethrin [66]. The current recommendations for treatment of pediculosis pubis are listed in table 2. Since 1996, there have not been any English-language studies documenting significant treatment failure in the management of pediculosis pubis.

**DISCUSSION**

Ectoparasites continue to be a common cause of skin disease throughout the world. Topical agents are generally effective in the management of extoparasites but are accompanied by sometimes-serious adverse effects, and to several agents has been documented. Ivermectin is a new oral mode of treatment for scabies and may hold particular promise in the treatment of epidemic or severe scabies. Lindane is no longer recommended as a first-line or alternative treatment for scabies. Combination treatment with ivermectin and topical scabicides may prove to be the best treatment for crusted scabies, but this requires further study. Pediculosis pubis is usually effectively treated with topical agents, but increasing rates of resistance among head lice warrant concern regarding future efficacy of current topical agents in the treatment of pediculosis pubis.

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