

## Review

# Scabies: update on treatment and efforts for prevention and control in highly endemic settings

Sandra Widaty<sup>1</sup>, Eliza Miranda<sup>1</sup>, Emilina Faradila Cornain<sup>1</sup>, Luddwi Achmad Rizky<sup>1</sup>

<sup>1</sup> Department of Dermatology and Venereology, Faculty of Medicine Universitas Indonesia, Dr. Cipto Mangunkusumo Hospital, Jakarta, Indonesia

### Abstract

Scabies is a contagious parasitic skin disease caused by *Sarcoptes scabiei* infestation which can be transmitted through direct or indirect contact. WHO classified scabies as a neglected tropical disease. The prevalence of scabies is high in certain countries ranging from 32.1% to 74%, especially in crowded conditions such as prisons, boarding schools, and orphanages. Indonesia is one of the most heavily affected countries worldwide. Scabies might cause great impact on patients, which includes decreased concentration and academic achievement at school, social stigma, sleep disturbances, and decreased economic productivity in community. Management of scabies with anti-scabies needs to be carried out appropriately, accompanied with treatment for all contacts. Mass treatment with permethrin cream or ivermectin can be given directly to patients. Prevention is conducted by providing medical treatment and breaking the chain of transmission. Source elimination and disinfection of fomites is very important. Participation of non-medical personnel such as teachers, cadres, and parents together with the local health workers (primary health care) is highly recommended. Using checklists or application can aid non-medical personnel to determine suspected cases, thus contributing to scabies elimination. Cooperation between patients, patient's family, health workers and other non-medical personnel will greatly reduce the prevalence of scabies and ultimately improve patient's quality of life. The aim of this review is to provide an update on scabies treatment and efforts for prevention and elimination, with focus on the situation in Indonesia.

**Key words:** Scabies; treatment; elimination; prevention; non-medical personnel.

*J Infect Dev Ctries* 2022; 16(2):244-251. doi:10.3855/jidc.15222

(Received 24 April 2021 – Accepted 11 September 2021)

Copyright © 2022 Widaty *et al.* This is an open-access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

### Introduction

Scabies is a skin infestation of the mite, *Sarcoptes scabiei*, which manifests as a pruritic skin eruption and can be transmitted directly through person-to-person contact and indirectly through bedsheets, clothing, or other fabric material. Scabies infection is particularly prevalent in overcrowded environment such as orphanages and public boarding schools [1]. The World Health Organization (WHO) stated that scabies is one of the most neglected diseases in the world [2]. It is estimated that 200 million people in the world have suffered from a scabies infection at least once in their lifetime. The prevalence of scabies ranges from 0.2 to 71%. A Global Burden of Disease Study in 2015 reported that Indonesia ranks first, among the 195 countries of the world, in scabies infection [3]. The aim of this review is to provide an update on scabies treatment and new efforts for prevention and elimination, with focus on the situation in Indonesia.

### Causative Agent

Scabies is a parasitic skin infection caused by the human-specific mite *Sarcoptes scabiei* var. *hominis*.

Adult mites are approximately 0.4 mm in size, which requires imaging instruments to visualize them [4,5]. The life cycle of scabies infestation starts when a pregnant female mite creates a burrow on the human epidermis, producing 2–3 eggs daily. Most eggs deposited in the burrows hatch into larvae after 48–72 hours. Larvae excavate other burrows and reach the adult stage in 10–14 days. Adult mites then reproduce, ultimately repeating the life cycle. The incubation period is estimated to be between 4–6 weeks after primary infection. Route of transmission includes predominantly via direct skin-to-skin contact [5-7] and rarely via indirect contaminated fomites, particularly in patients with crusted scabies [5,6,8]. Without residing the human host, scabies mites remain to survive around 24–36 hours at room temperature, enabling further infection [6,7].

### Epidemiology and risk factors

An estimated 130 million people worldwide are infected by scabies, at any given time. This estimate is supported by the high number of cases reported throughout the world every year, reaching 300 million

cases [9]. A cross-sectional analysis on the 2015 Global Burden of Disease Study found that Indonesia is one of the top five countries with greatest scabies burden followed by China, Timor-Leste, Vanuatu, and Fiji [3]. The prevalence of scabies varies from 0.2 to 71% in each country.

In Indonesia, a report by the Ministry of Health in 2011 revealed that 2.9% of 69,15,315 people, were infected with scabies. In 2012, the proportion increased to 3.6%. Scabies was frequently observed in children as compared to adults [10]. Reports from community health centres or ‘*Puskesmas*’ across Indonesia found that scabies is the third most commonly found skin disease [11]. The prevalence ranges from 5.6% to 12.9%. In 2012, the number of scabies cases amongst orphanages and religion-affiliated boarding schools in East Jakarta were 51.6% and 68% in South Jakarta the following year [11].

Appropriate treatment *per se* is not adequate to eradicate scabies. A physician must consider environmental factors and patient’s habits, which are pivotal for preventing reinfection. A study conducted in 2017 found that the availability of hot water as well as living conditions in religion-affiliated boarding schools were important factors related to scabies reinfection [12]. While clean water was available in the property, hot water was not available for washing clothes. A water temperature of at least 50°C is essential to ensure mite and egg elimination. This finding was supported by Yasin *et al.* which reveals that personal hygiene consists of bathing frequency, towel, and clothes sharing were not a significant factor for scabies infection, instead clean water availability or room sanitation was correlated with scabies [13].

In addition to personal hygiene, bedding conditions was another problem encountered in the study. Since several students occupied beds that were not covered by bedsheets, thus allowing direct skin contact. Mites would drop on the mattress surfaces and since the mattresses are not washed, parasites stayed longer and were able to infect other people promptly [12]. Widuri

*et al.* reported that sleeping together in closed spaces increased the risk of getting scabies by 21 times [14]. Personal habits comprises of towel-sharing, clothes-sharing, and prayer-attire sharing are recurrently noticed in students at religion-affiliated boarding schools [12]. Furthermore, towel-sharing increased the risk of scabies by 3.4 times [14]. Widaty *et al.* found that the practice of clothes-sharing and prayer-attire sharing might be driven by socioeconomic reasons, as study participants admitted to not having spare items [12]. Participants did not frequently wash their attire thus increasing the risk of prolong contact with infectious materials. Akmal *et al.* who conducted a study in Sumatra also reported that poor personal hygiene was strongly correlated to scabies [11]. In addition to behavioural habits, knowledge level was another significant factor correlated to scabies prevalence. However, it was not clearly defined what constituted the knowledge [12].

**Clinical Manifestation and Diagnostic Criteria**

A recently modified Delphi study involving 34 worldwide experts proposed consensus criteria for the diagnosis of scabies, known as The 2020 International Alliance for The Control of Scabies (IACS) criteria. Three levels and eight subcategories of diagnosis are established as per diagnostic certainty, which is confirmed, clinical, and suspected. (Table 1) [5].

A confirmed diagnosis of scabies (level A) is established based on the presence of any stage of scabies’ lifecycle (eggs, larvae, nymphs, or adults) or feces (scybalas). Visualization devices and samples affect the diagnostic subcategory, for instance, A1 if the eggs, mites, or feces are discovered on the skin sample using light microscopy; A2 if the eggs, mites, or feces are visualized *in vivo* using high-powered magnifying devices; A3 if the mites are visualized *in vivo* using dermoscopy [5]. The most recognized and suitable method to confirm scabies diagnosis is by identifying the skin sample using light microscopy. However, the result accuracy is operator-dependent, especially in

**Table 1.** Summary of the 2020 International Alliance for The Control of Scabies (IACS) criteria.

<b>Diagnostic Level and Subcategories of Scabies</b>		
<b>A: Confirmed Scabies</b> <b>Minimum fulfil one criterion</b>	<b>B: Clinical scabies</b> <b>Minimum fulfil one criterion</b>	<b>C: Suspected scabies</b> <b>One criterion</b>
A1: Mites, eggs, or feces depicted through light microscope on skin specimen	B1: Scabies burrows	C1: Typical lesions affecting typical distribution along with one history feature*
A2: Mites, eggs, or feces visualized <i>in vivo</i> through high-powered imaging device e.g. video-dermoscopy	B2: Typical lesions involving male genitalia	C2: Atypical lesions affecting atypical distribution along with two history features*
A3: Mites identified <i>in vivo</i> through dermoscopy	B3: Typical lesions affecting typical distribution along with two history features*	

\*History Features; H1: Itch (pruritus); H2: Positive contact history.

obtaining the burrows and preparing the materials. Although a positive test validates the diagnosis of scabies, a negative test does not eliminate it, given that patients with clinical scabies often have negative results [5,15,16]. High-powered magnifying devices allow the identification of scabies mites, in vivo, in a detailed manner. Magnification using these devices (e.g., reflectance confocal microscopy and video dermoscopy) can achieve more than 70x amplification [5,9]. Identification of scabies mites through dermoscopy in vivo confirms the diagnosis of scabies [5,16-19]. Several hallmarks can be observed through dermoscopy. S-formed burrow demonstrated as a curvilinear trail of scale. Brown triangular V-formed arrangement is analogous to the head and set of legs, commonly mentioned as the triangle sign, delta-wing jet, delta glider, jet plane, or spermatozoid configuration. Visualization of mite at the edge of the burrow is frequently referred to as the jetliner with its trail [17,20,21]. Furthermore, the ovoid forms resting inside the burrows indicated the appearance of scabies eggs [17,18].

Based on the clinical evaluation, such as the patient's history features and dermatological examination, scabies can be diagnosed as clinical scabies (level B) or suspected scabies (level C). If the attributes meet the criteria, then a diagnosis of clinical scabies can be established. However, if the characteristics are less specific, a diagnosis of suspected scabies can be achieved [5].

Itch (pruritus) is frequently observed in infected patients [5,19]. Following primary infection, itch starts to develop [4,5]. Itch intensity can vary between individuals, ranging from extreme-itch impairing quality of life to mild manifestation [5,22-24]. Typically, the itch is often exacerbated at night [5,25,26]. However, numerous pruritic skin diseases demonstrate a similar pattern; hence nocturnal pruritus is inadequate as a diagnostic feature [5,24,27]. Itch can be localized, confined on the noticeable scabies lesions, or generalized, affecting other body regions. Constant scratching on the lesions, which manifests as excoriation, also fulfils the itch criterion, particularly in the pediatric population [5,28].

As a rule, skin-to-skin contact facilitates scabies transmission [5-7]. Fomite-mediated transmission hardly occurs in common scabies, except for crusted scabies [5,6,8]. Multiple factors that influence the risk of common scabies transmission are duration, frequency, and body surface area of skin contact [5,29]; hence bed-sharing individuals and children are at higher risk [5]. Previous literature suggested that the minimum

skin-to-skin duration for mites infection is approximately 20 minutes [4,7]; however, it is still undetermined.

Detailed positive contact history and close contact are defined as per the 2020 IACS. A positive contact history with high-risk transmission includes any contact with a patient diagnosed with crusted scabies, close contact with a patient diagnosed with common scabies, close contact with a patient with inexplicable pruritus, and close contact with a patient with typical scabies lesion affecting typical distribution with inexplicable condition. Close contact is described as: persons who sleep together in the same residence; persons who share a bed or sleeping surface, e.g., couples, children in the same teaching space or who play together; and adults with documented skin-to-skin contact, e.g., workplace exposure (health professionals, assisted living caregivers, teachers of schoolchildren) and leisure exposure (contact sports, e.g., wrestling) [5].

Clinical signs of common scabies between each patient are extensive depending on the skin manifestation of individual lesions, grouped or clustered lesions, and various secondary changes including excoriation, lichenification, impetiginisation, and eczematization, which ultimately complicate the primary scabies infestation. If the definite signs of scabies such as burrows or lesions affecting male genitalia are lacking, clinical judgment to determine whether a lesion is considered typical for scabies depends on morphology and number [5].

The most frequent morphological lesions are papules [5,30], which are erythematous or hyperpigmented in darker skin tone [5]. Nodules are more likely to appear in particular body areas (axillae, breasts, groins, genitalia, and torsos in infants) [5,31,32], for few months even after the mites have been completely exterminated [5]. Although less common in adults [5], vesicles and pustules can be observed in infants, particularly in the palmoplantar regions. If the predominant lesions are vesicles, pustules, or blisters, the differential diagnosis should be considered [5,33]. Furthermore, superimposed infection by *S. aureus* or *S. pyogenes* causes impetiginized lesions characterized by demonstrating inflammation along with ulceration and yellowish crusting. Groups of lesions typically emerge on a particular body area, although widespread lesions may occur [5].

Scabies is classified as typical if there are three minimum lesions on a particular body area or contained in diameter of around 10 to 20 cm. If the lesions do not demonstrate typical morphology or the number of

**Table 2.** Comparison of pathophysiologic characteristics between common and crusted scabies.

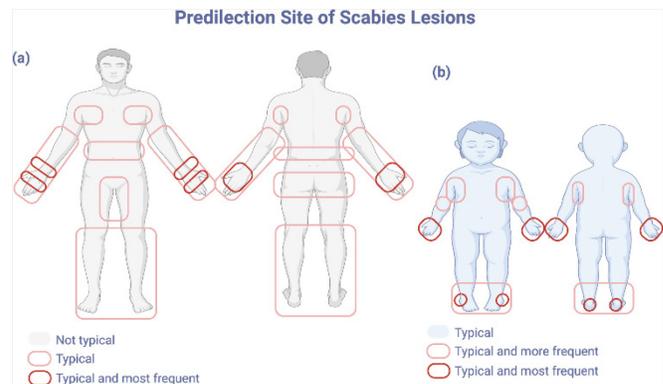
Indicator	Common Scabies	Crusted Scabies
Type of immune response	Th1 lymphocytes	Th2 lymphocytes
Infiltrate along the epidermal junction	Predominantly CD4 <sup>+</sup> T cells	Predominantly CD8 <sup>+</sup> T cells
Consequence	Type IV hypersensitivity reaction	Local IL-17 secretion: induces keratinocyte proliferation, acanthosis, hyperkeratosis, appearance of thick scabs
	Protective response: limitation of the number of mites	Ineffective inflammation: cytotoxic lesions of keratinocytes (by CD8 <sup>+</sup> T cells); proliferation of mites
Histological characteristic	Similar to atopic dermatitis	Similar to psoriasis: acanthosis, parakeratotic hyperkeratosis, keratinocytic apoptosis, dermal lymphocytic infiltrate, basal mitosis

lesions is below 3 in any body area, then the lesions are classified as atypical. Typical distribution of scabies lesions are described as per Figure 1. Lesions of common scabies appear in several body areas [5], while severe infestation results in multiple body areas that can be symmetrical between left and right sides [5,34]. Atypical distribution includes lesions observed on the head, scalp, neck, and tends to be asymmetrical [5,35].

Hyperinfestation of mites in immunodeficient host causes a severe and rare manifestation of scabies, known as crusted scabies [36,37]. It was formerly called ‘Norwegian scabies’, although the term should no longer be applied [36-40]. Comprising up to 4,000 mites per gram of skin, crusted scabies is highly infectious. It is characterized by abundant hyperkeratotic skin crusts and fissures, which have a significant mortality rate compared to common scabies [36,37,41,42]. Contrary to common scabies, the itch is relatively mild or even absent in crusted scabies [38]. Jouret *et al.* [37] proposed a comparison of the pathophysiologic characteristics between common and crusted scabies (Table 2).

Furthermore, a recent clinical grading scale of crusted scabies was established by Davis *et al.* [36], which classified clinical severity into three grades: Grade 1 (mild crusted scabies), Grade 2 (moderate crusted scabies), and Grade 3 (severe crusted scabies) (Table 3).

**Figure 1.** Predilection site of scabies lesions.



(a) Adults and children older than 2 years of age; (b) Infants younger than 2 years of age.

**Table 3.** Clinical grading scale for crusted scabies.

A: Distribution and Extent of Crusting	
1.	Wrists, web spaces, feet only (< 10% Total Body Surface Area)
2.	Point 1 plus forearms, lower legs, buttocks, trunk, or 10-30% TBSA
3.	Point 2 plus scalp or > 30% TBSA
B: Crusting / Shedding	
1.	Mild crusting (< 5 mm depth of crust), minimal skin shedding
2.	Moderate (5-10 mm) crusting, moderate skin shedding
3.	Severe (> 10 mm), profuse skin shedding
C: Past Episodes	
1.	Never had it before
2.	1-3 prior hospitalizations for crusted scabies or depigmentation of elbows, knees
3.	≥ 4 prior hospitalizations for crusted scabies or depigmentation as above plus legs/back or residual skin thickening/ichtyosis
D: Skin Condition	
1.	No cracking or pyoderma
2.	Multiple pustules and/or weeping sore and/or superficial skin cracking
3.	Deep skin cracking with bleeding, widespread purulent exudates

Grade 1: Total score 4-6; Grade 2: Total score 7-9; Grade 3: Total score 10-12.

## Treatment

Various effective scabicides are available, which can be topical or oral, depending on patients' characteristics, disease conditions, treatment cost and accessibility, and physicians' preference. Treatment regimens should be given at every stage of diagnostic levels; not only limited to clinical or confirmed scabies, but also to patients with suspected scabies [6].

A recent network meta-analysis of 52 studies was conducted by Thadanipon *et al.* [43] to determine the relative efficacy and safety of anti-scabies agents. Permethrin, as the first-line of treatment option, demonstrated a statistically significant higher cure rate than sulphur, malathion, lindane, crotamiton, and benzyl benzoate [38,44,45]. A higher cure rate, although insignificant, was obtained when comparing a combination of topical permethrin and oral ivermectin to single topical permethrin [38,46]. Based on the curative perspective, a combination of topical permethrin and oral ivermectin showed the uppermost ranking [46]. Topical ivermectin ranked highest concerning pruritus improvement [47]. However, synergized pyrethrins revealed the most adverse events [48]. Conclusively, there is no single anti-scabies agent that was superior in all aspects such as cure rate, pruritus improvement, and adverse events; hence, physicians' judgment depends on the drug's efficacy-safety and convenience [43].

The latest guideline for the management of scabies was established in 2017, known as the European Guideline for the Management of Scabies. Several new

recommendations were updated since the 2010 edition. A summary of the general principles of treatment are described in Table 4 [38].

Topical agents should be applied to all skin parts nocturnally, including scalp, navel, groin, fingers, interdigital areas, and skin underneath the nails' terminal part. Topical treatment should remain in contact with affected skin for 8 to 12 hours. Skin condition should be cool and dry while on the treatment. Following the application of the topical treatment, clean apparel should be worn. Close contact persons should also be treated concurrently to prevent the spread of infection and the host's reinfection [38,49]. To date, the application of lindane is not suggested due to the possible risk of neurotoxicity [38,50] and production of hazardous pollutants in waste-water [51].

All apparel, bed sheets, towels, and other fomites should be machine washed at a temperature over 50 °C, dry-cleaned, or wrapped in a plastic container for one week (level of evidence: VI; grade C recommendation) [40,52]. Furthermore, a comprehensive written explanation of scabies infestation should also be provided for the patients (level of evidence: IV; grade C recommendation) [38,50]. Successful treatment is indicated by the absence of active scabies manifestation following one week of final treatment, which is the non-appearance of active lesions and convalesced nocturnal itch. However, post-treatment itch may remain until 2 to 4 weeks. Reapplication of emollients, oral antihistamines, and mild-potency topical corticosteroid should be given to alleviate post-treatment itch [38].

**Table 4.** General Principles of Scabies Treatment.

Anti-scabietic Agent*	Instruction	Level of Evidence (LoE); Grade Recommendation
<b>Recommended treatments</b>		
Permethrin 5% cream [32,38]	Apply head-to-toe, repeat after 7-14 days.	Ib; A
Oral ivermectin 200 mcg/kg [32,39]	Consume along with food. Two doses with a one-week interval.	Ib; A
Benzyl benzoate 10-25% lotion [32,46]	Apply once daily by night for two successive days. Reapply at seven days.	IV; C
<b>Alternative treatments</b>		
Malathion 0.5% aqueous lotion [32,39]	Apply once weekly for two weeks, cleanse after 24 hours.	IV; C
Ivermectin 1% lotion [32,41] <sup>†</sup>	Apply once as a single application	Ib; A
Sulphur 6-33% cream, ointment, or lotion [32,43]	Apply once daily for three consecutive days.	Ib; A
Synergized pyrethrin foam [32,44] <sup>†</sup>	Apply once daily for three consecutive days.	IIa; B
<b>Crusted scabies [32,45]</b>		
Topical scabicide (Permethrin 5% cream or benzyl benzoate 25% lotion) and oral ivermectin 200 mcg/kg	Apply topical scabicide once daily for seven days, followed by twice-weekly until healed. Consume oral ivermectin on days 1, 2, and 8. Extra ivermectin might be required in severe cases, given on days 9 and 15 or days 9, 15, 22, and 29.	IV; C

\*All topical agents should be applied to all skin parts nocturnally and remain in contact with the skin for 8 to 12 hours [32]; <sup>†</sup>Demonstrated equal efficacy to permethrin cream 5% [32].

Following treatment completion, a follow-up visit within two weeks is essential to evaluate the affected skin through microscopic examination (level of evidence: IV; grade C recommendation) [38,50].

Permethrin [38,49], benzyl benzoate, and sulphur [38,50,53,54] are considered safe during pregnancy (level of evidence III; grade B recommendation). Furthermore, permethrin is permitted for children above two months of age [38,55]. For pregnancy, lactation, and children below two years of age, permethrin application should be restricted to no more than two hours between two doses with a one-week interval [56]. Ivermectin should not be given to pregnant women and children weighing below 15 kg [38,57].

Failure to scabies treatment has recently been reported [58]. Sunderkotter *et al.* proposed several reasons underlying the failure of scabies treatment such as improper application of permethrin, reinfection because of the partial management of environs, including contact with individuals and fomites, and resistance of mites toward permethrin. Several circumstances might affect the improper application of permethrin, including a short exposure period to permethrin, not trimming the fingernails which may hide the subungual mites following scratching, ineffective treatment of hyperkeratotic skin, or failure to employ permethrin in the head area in toddlers [58,59]. Reinfestation because of incomplete eradication of mites in surroundings can result in treatment failure. Unrecognized or incompletely treated contacts are commonly encountered in a paediatric population who share the same surroundings [58,60]. Furthermore, several plausible explanations might address the resistance to permethrin and ivermectin [58]. Mounsey *et al.* reported an extended survival of scabies after exposure to permethrin and associated with greater glutathione-S-transferases' transcription [61]. Several studies report resistance of mites to ivermectin, due to genetic changes of the mites structure that regulate the glutamate-directed chloride gate [62,63] or a P-glycoprotein membrane transport protein [64]. However, it is improbable that scabies mites are insensitive to both permethrin and ivermectin [58].

### Scabies prevention

Although scabies is not regarded as a deadly disease, it largely affects the patient's quality of life [65]; therefore, elimination and prevention efforts are important. A way to eliminate scabies is by increasing community awareness and knowledge regarding the

diseases and the preventive measures, for instance proper handling of contaminated materials (bedsheets, clothing, towel). To ensure mite elimination, contaminated materials must be washed in hot water and dried with a hot dryer. If hot water is not available, sterilization can be performed by keeping infected materials in a plastic bag for 7 days, as the survivability of mites only last 3 days outside of the host. As scabies are frequently encountered in boarding schools, participation of non-medical personnel in scabies screening might be beneficial for early detection of cases. The utilization of a screening checklist for scabies' signs and symptoms might be practical for early detection and thus promoting prompt treatment [66].

A screening checklist, known as “*Deskab*” in Indonesia, comprises of simplified history taking and physical examination that can be performed by a non-medical personnel. This checklist was based on the established criteria for diagnosing scabies such as, nocturnal pruritus, history of itchiness in exposed individuals, and presence of lesions suggestive of scabies. We investigated the application of this screening tool and the results showed that there was no statistically significant difference between students who were examined by dermatologists and those who were assessed by non-medical personnel using the *Deskab* form [66]. *Deskab* has been used in a number of boarding schools throughout Indonesia. We found that by training non-pharmacological intervention such as, involvement of non-medical personnel, contributes to the significant decrease of scabies incidence rate [67].

### Conclusions

Scabies is a skin disease that affects an individual's quality of life. Diagnosis is established clinically or through additional supporting examinations deemed necessary. It is important that all members of the affected household are treated and their surroundings are sterilized properly to ensure mite elimination and ultimately preventing further transmission. The role of non-medical personnel in performing screening for scabies and providing surveillance in over-crowded communities will aid scabies eradication.

### References

- Centers for Disease Control and Prevention (2010) Scabies. Available: <https://www.cdc.gov/parasites/scabies/>. Accessed: 20 January 2020.
- World Health Organization Neglected tropical diseases: Treating more than one billion people for the fifth consecutive year (2020) Available from:

- <https://www.who.int/news/item/16-07-2020-neglected-tropical-diseases-treating-more-than-one-billion-people-for-the-fifth-consecutive-year>. Accessed: 19 January 2020.
3. Karimkhani C, Colombara DV, Drucker AM, Norton SA, Hay R, Engelman D, Steer A, Whitfeld M, Naghavi M, Dellavalle RP (2017) The global burden of scabies: a cross-sectional analysis from the Global Burden of Disease Study 2015. *Lancet Infect Dis* 17: 1247–1254.
  4. Mellanby K (1944) The development of symptoms, parasitic infection and immunity in human scabies. *Parasitology* 35: 197–206.
  5. Engelman D, Yoshizumi J, Hay RJ, Osti M, Micali G, Norton S, Walton S, Boralevi F, Bernigaud C, Bowen AC, Chang AY, Chosidow O, Estrada-Chavez G, Feldmeier H, Ishii N, Lacarrubba F, Mahé A, Maurer T, Mahdi MMA, Murdoch ME, Pariser D, Nair PA, Rehmus W, Romani L, Tilakaratne D, Tuicakau M, Walker SL, Wanat KA, Whitfeld MJ, Yotsu RR, Steer AC, Fuller LC (2020) The 2020 international alliance for the control of scabies consensus criteria for the diagnosis of scabies. *Br J Dermatol* 183: 808–820.
  6. Chandler DJ, Fuller LC (2019) A review of scabies: an infestation more than skin deep. *Dermatology* 235: 79–90.
  7. Arlian LG, Runyan RA, Achar S, Estes SA (1984) Survival and infestivity of *Sarcoptes scabiei* var. *canis* and var. *hominis*. *J Am Acad Dermatol* 11: 210–215.
  8. Mellanby K (1941) Transmission of scabies. *Br Med J* 2: 405–406.
  9. Micali G, Lacarrubba F, Verzi AE, Chosidow O, Schwartz RA (2016) Scabies: advances in noninvasive diagnosis. *PLoS Negl Trop Dis* 10: e0004691.
  10. Indonesian Ministry of Health (2012) Indonesian health profile year 2012 [Article in Indonesian] Available: <https://pusdatin.kemkes.go.id/folder/view/01/structure-publikasi-pusdatin-profil-kesehatan.html>. Accessed 17 January 2020.
  11. Akmal S, Semiarty R (2013) The association of personal hygiene and incidence of scabies at Darul Ulum Islamic education center, Palarik Air Pacah, Koto Tengah Padang region. *Jurnal Kesehatan Andalas* 164–167 [Article in Indonesian].
  12. Widaty S, Rihatmadja R, Miranda E, Wicaksono MM (2019) Why are they hard to treat? A preliminary survey to predict important factors causing persistent scabies among students of religion-affiliated boarding schools in Indonesia. *Dermatol Reports* 11: 41–43.
  13. Yasin (2009) Scabies prevalence and related factors among students of Darul Mujahadah boarding school, Tegal Regency, Central Java Province. PhD Thesis [Thesis in Indonesian].
  14. Widuri N, Candrawati E, Masluhiya S (2017) Analysis of risk factor for scabies among students of Nurul Hikmah boarding school, Kebonagung Village, Kecamatan Pakisaji, Malang Regency. *Nursing News* 2: 622–633. [Article in Indonesian].
  15. Thompson MJ, Engelman D, Gholam K, Fuller LC, Steer AC (2017) Systematic review of the diagnosis of scabies in therapeutic trials. *Clin Exp Dermatol* 42: 481–487.
  16. Walter B, Heukelbach J, Fengler G, Worth C, Hengge U, Feldmeier H (2011) Comparison of dermoscopy, skin scraping, and the adhesive tape test for the diagnosis of scabies in a resource-poor setting. *Arch Dermatol* 147: 468–473.
  17. Grover C, Jakhar, D (2017) Dermoscopy in the diagnosis of scabies. *Int J Dermatol* 1: 67–68.
  18. Fox G (2009) Diagnosis of scabies by dermoscopy. *BMJ Case Rep* 2009: bcr0620080279.
  19. Dupuy A, Dehen L, Bourrat E, Lacroix C, Benderdouche M, Dubertret L, Morel P, Feuillade de Chauvin M, Petit A (2007) Accuracy of standard dermoscopy for diagnosing scabies. *J Am Acad Dermatol* 56: 53–62.
  20. Park JH, Kim CW, Kim SS (2012) The diagnostic accuracy of dermoscopy for scabies. *Ann Dermatol* 24: 194–199.
  21. Prins C, Stucki L, French L, Saurat J-H, Braun RP (2004) Dermoscopy for the in vivo detection of *Sarcoptes scabiei*. *Dermatology* 208: 241–243.
  22. Worth C, Heukelbach J, Fengler G, Walter B, Liesenfeld O, Feldmeier H (2012) Impaired quality of life in adults and children with scabies from an impoverished community in Brazil. *Int J Dermatol* 51: 275–282.
  23. Puza CJ, Suresh V (2018) Scabies and pruritus—A historical review. *JAMA Dermatol* 154: 536.
  24. Jannic A, Bernigaud C, Brenaut E, Chosidow O (2018) Scabies itch. *Dermatol Clin* 36: 301–308.
  25. Jackson A, Heukelbach J, Filho AF da S, Campelo Júnior E de B, Feldmeier H (2007) Clinical features and associated morbidity of scabies in a rural community in Alagoas, Brazil. *Trop Med Int Health* 12: 493–502.
  26. Shin K, Jin H, You HS, Kim JM, Shim WH, Kim GW, Kim HS, Ko HC, Kim MB, Kim BS (2017) Clinical characteristics of pruritus in scabies. *Indian J Dermatol Venereol Leprol* 83: 492–493.
  27. Brenaut E, Garlantezec R, Talour K, Misery L (2013) Itch characteristics in five dermatoses: non-atopic eczema, atopic dermatitis, urticaria, psoriasis and scabies. *Acta Derm Venereol* 93: 573–574.
  28. Bernardeschi C, Le Cleach L, Delaunay P, Chosidow O (2013) Bed bug infestation. *Br Med J* 346: f138.
  29. Ministry of Health Malaysia. (2015) Guideline for management of scabies in adults and children Malaysia. Available: <http://moh.gov.my/>. Accessed 17 January 2020.
  30. Nast A, Griffiths CEM, Hay R, Sterry W, Bologna JL (2016) The 2016 international league of dermatological societies' revised glossary for the description of cutaneous lesions. *Br J Dermatol* 174: 1351–1358.
  31. Suh KS, Han SH, Lee KH, Park JB, Jung SM, Kim ST, Jang MS (2014) Mites and burrows are frequently found in nodular scabies by dermoscopy and histopathology. *J Am Acad Dermatol* 71: 1022–1023.
  32. Orkin M (1977) Special forms of scabies. In Orkin M, Maibach H, Parish L, Schwartzman R, Editors. *Scabies and Pediculosis*. Philadelphia: J.B. Lippincot. 23–30.
  33. Prendiville J (2011) Scabies and lice. In Irvine A, Hoeger P, Yan A, editors. *Harper's Textbook of Pediatric Dermatology*. Chichester: Wiley-Blackwell.
  34. Chosidow O (2006) Scabies. *N Engl J Med* 354: 1718–1727.
  35. Cassell JA, Middleton J, Nalabanda A, Lanza S, Head MG, Bostock J, Hewitt K, Jones CI, Darley C, Karir S, Walker SL (2018) Scabies outbreaks in ten care homes for elderly people: a prospective study of clinical features, epidemiology, and treatment outcomes. *Lancet Infect Dis* 18: 894–902.
  36. Davis JS, McGloughlin S, Tong SYC, Walton SF, Currie BJ (2013) A novel clinical grading scale to guide the management of crusted scabies. *PLoS Negl Trop Dis* 7: e2387.
  37. Jouret G, Bounemour R, Presle A, Takin R (2016) Hyperkeratotic scabies. *Ann Dermatol Venereol* 143: 251–6. [Article in French].
  38. Salavastru CM, Chosidow O, Boffa MJ, Janier M, Tiplica GS (2017) European guideline for the management of scabies. *J Eur Acad Dermatol Venereol* 31: 1248–1253.

39. Scott G (2017) United Kingdom national guideline on the management of scabies. Available: <https://www.bashhguidelines.org/media/1137/scabies-2016.pdf>. Accessed 15 January 2020.
40. Schlesinger I, Oelrich DM, Tying SK (1994) Crusted (Norwegian) scabies in patients with AIDS: the range of clinical presentations. *South Med J* 87: 352–356.
41. Walton SF (2010) The immunology of susceptibility and resistance to scabies. *Parasite Immunol* 32: 532–540.
42. Roberts LJ, Huffam SE, Walton SF, Currie BJ (2005) Crusted scabies: clinical and immunological findings in seventy-eight patients and a review of the literature. *J Infect* 50: 375–381.
43. Thadanipon K, Anothaisintawee T, Rattanasiri S, Thakkinstian A, Attia J (2019) Efficacy and safety of antiscabietic agents: a systematic review and network meta-analysis of randomized controlled trials. *J Am Acad Dermatol* 80: 1435–1444.
44. Schultz MW, Gomez M, Hansen RC, Mills J, Menter A, Rodgers H, Judson FN, Mertz G, Handsfield HH (1990) Comparative study of 5% permethrin cream and 1% lindane lotion for the treatment of scabies. *Arch Dermatol* 126:167–170.
45. Joint Formulary Committee (2015) Malathion. *British National Formulary* 70: 1015.
46. Chouela EN, Abeldaño AM, Pellerano G, La Forgia M, Papale RM, Garsd A, del Carmen Balian M, Battista V, Poggio N (1999) Equivalent therapeutic efficacy and safety of ivermectin and lindane in the treatment of human scabies. *Arch Dermatol* 135: 651–655.
47. Chhaiya SB, Patel VJ, Dave JN, Mehta DS, Shah HA (2012) Comparative efficacy and safety of topical permethrin, topical ivermectin, and oral ivermectin in patients of uncomplicated scabies. *Indian J Dermatol Venereol Leprol* 78: 605–10.
48. Amerio PL, Capizzi R, Milani M (2003) Efficacy and tolerability of natural synergised pyrethrins in a new thermo labile foam formulation in topical treatment of scabies: a prospective, randomised, investigator-blinded, comparative trial vs. permethrin cream. *Eur J Dermatol* 13: 69–71.
49. Mytton OT, McGready R, Lee SJ, Roberts CH, Ashley EA, Carrara VI, Thwai KL, Jay MP, Wiangambun T, Singhasivanon P, Nosten F (2007) Safety of benzyl benzoate lotion and permethrin in pregnancy: a retrospective matched cohort study. *BJOG: Int J Obstet Gynaecol* 114: 582–587.
50. Scott GR, Chosidow O (2011) European guideline for the management of scabies, 2010. *Int J STD AIDS* 22:301–303.
51. Humphreys EH, Janssen S, Heil A, Hiatt P, Solomon G, Miller MD (2008) Outcomes of the California ban on pharmaceutical lindane: clinical and ecologic impacts. *Environ Health Perspect* 116: 297–302.
52. Carslaw RW, Dobson RM, Hood AJK, Taylor RN (1975) Mites in the environment of cases of Norwegian scabies. *Br J Dermatol* 92: 333–337.
53. Chosidow O (2000) Scabies and pediculosis. *Lancet* 355: 819–826.
54. Singalavanija S, Limpongsanurak W, Soponsakunkul S (2003) A comparative study between 10 per cent sulfur ointment and 0.3 per cent gamma benzene hexachloride gel in the treatment of scabies in children. *J Med Assoc Thai* 86: S531-6.
55. Centers for Disease Control and Prevention (2019) Classic scabies. In: *CDC Resources for Health Professionals*. Available: [http://www.cdc.gov/parasites/scabies/health\\_professionals/mes.html](http://www.cdc.gov/parasites/scabies/health_professionals/mes.html). Accessed: 20 January 2020.
56. Wheat CM, Burkhart CN, Burkhart CG, Cohen BA (2019) Scabies, other mites, and pediculosis. In Kang S, Amagai M, Bruckner AL, Enk AH, Margolis DJ, McMichael AJ, Orringer JS, editors. *Fitzpatrick’s Dermatology in General Medicine*. New York: McGraw Hill. 3274–3286.
57. Workowski KA, Bolan GA (2015) Sexually transmitted diseases treatment guidelines, 2015. *CDC Recommendations and Reports* 64: 1–137.
58. Sunderkötter C, Aebischer A, Neufeld M, Löser C, Kreuter A, Bialek R, Hamm H, Feldmeier H (2019) Increase of scabies in Germany and development of resistant mites? Evidence and consequences. *J Dtsch Dermatol Ges* 17: 15–23.
59. Sunderkötter C, Feldmeier H, Fölster-Holst R, Geisel B, Klinke-Rehbein S, Nast A, Philipp S, Sachs B, Stingl J, Stoevesandt J, Hamm H (2016) S1 guidelines on the diagnosis and treatment of scabies - short version. *J Dtsch Dermatol Ges* 14: 1155–1167.
60. De Sainte Marie B, Mallet S, Gaudy-Marqueste C, Baumstarck K, Bentaleb N, Loundou A, Hesse S, Monestier S, Grob J-J, Richard M-A (2016) Therapeutic failure in scabies: an observational study. *Ann Dermatol Venereol* 143: 9–15. [Article in French].
61. Mounsey KE, Pasay CJ, Arlian LG, Morgan MS, Holt DC, Currie BJ, Walton SF, McCarthy JS (2010) Increased transcription of glutathione s-transferases in acaricide exposed scabies mites. *Parasit Vectors* 3: 43.
62. Currie BJ, Harumal P, McKinnon M, Walton SF (2004) First documentation of in vivo and in vitro ivermectin resistance in *Sarcoptes scabiei*. *Clin Infect Dis* 39: e8-12.
63. Mounsey KE, Holt DC, McCarthy J, Currie BJ, Walton SF (2008) Scabies: molecular perspectives and therapeutic implications in the face of emerging drug resistance. *Future Microbiol* 3: 57–66.
64. Xu M, Molento M, Blackhall W, Ribeiro P, Beech R, Prichard R (1998) Ivermectin resistance in nematodes may be caused by alteration of p-glycoprotein homolog. *Mol Biochem Parasitol* 91: 327–335.
65. Menaldi SL, Marissa M, Surya D, The VV (2021) Impact of scabies on Indonesian public boarding school students’ quality of life: A mixed-method analysis. *J Gen Proced Dermatol Venereol Indones* 5: 74–78.
66. Widaty S, Krisanti RIA, Rihatmadja R, Miranda E, Marissa M, Arsy M, Surya D, Priyanto M, Menaldi SL (2019) Development of “Deskab” as an instrument to detect scabies for non-medical personnel in Indonesia. *Dermatol Reports* 11: 8023.
67. Widaty S, Menaldi SL, Rihatmadja R, Miranda E, Marissa M, Aria K, Friska D, Oktarina C, Surya D (2021) Involvement of non-medical personnel in management of scabies at a boarding school in Indonesia. *Southeast Asian J Trop Med Public Health* 52: 527–537.

### Corresponding author

Sandra Widaty, MD, PhD

Department of Dermatology and Venereology, Faculty of Medicine Universitas Indonesia, Dr. Cipto Mangunkusumo Hospital.

Jl. Pangeran Diponegoro No.71, Kenari, Kec. Senen, Kota Jakarta Pusat, Daerah Khusus Ibukota Jakarta 10430

Email: [sandra.widaty@gmail.com](mailto:sandra.widaty@gmail.com); [sanwidaty@ui.ac.id](mailto:sanwidaty@ui.ac.id)

**Conflict of interests:** No conflict of interests is declared.