Treatment of Seborrhoeic Dermatitis in Asia: A Consensus Guide

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Asia at a meeting held in Singapore. The consensus group developed a comprehensive algorithm to aid clinicians to recommend appropriate treatment of SD in both adults and children. In most cases, satisfactory therapeutic results can be accomplished with topical antifungal agents or topical corticosteroids. Non-steroidal anti-inflammatory agents with antifungal properties have been shown to be a viable option for both acute and maintenance therapy.

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Key Words
Seborrhoeic dermatitis · Asia · Treatment · Diagnosis · Consensus

Abstract
Seborrhoeic dermatitis (SD) is common in Asia. Its prevalence is estimated to be 1–5% in adults. However, larger population-based studies into the epidemiology of SD in Asia are lacking, and the aetiology of SD may differ widely from Western countries and in different parts of Asia. In addition, clinically significant differences between Asian and Caucasian skin have been reported. There is a need to define standardized clinical diagnostic criteria and/or a grading system to help determine appropriate treatments for SD within Asia. With this in mind, experts from India, South Korea, Taiwan, Malaysia, Vietnam, Singapore, Thailand, the Philippines, Indonesia, and Italy convened to define the landscape of SD in Asia at a meeting held in Singapore. The consensus group developed a comprehensive algorithm to aid clinicians to recommend appropriate treatment of SD in both adults and children. In most cases, satisfactory therapeutic results can be accomplished with topical antifungal agents or topical corticosteroids. Non-steroidal anti-inflammatory agents with antifungal properties have been shown to be a viable option for both acute and maintenance therapy.

Introduction
Seborrhoeic dermatitis (SD) is a common chronic inflammatory disease of the skin, which manifests as scaly reddish-brown itchy patches in sebaceous, gland-rich regions of the scalp, face, and trunk [1]. It affects both children and adults, with a higher incidence in infancy and...
mid-adulthood (age 30–60 years) [2–4]. In infants, SD frequently follows an uneventful course, and generally clears by 6–12 months of age (3 months in most Asian infants) [5]. It may appear again in individuals in their teens and twenties and may then generally follow a waxing and waning course throughout adulthood.

The distribution of SD is typically symmetrical and lesions range from being mild, patchy, and scaling to being widespread, thick, and with adherent crusts [1, 2]. The lesions may have a red, smooth, glazed appearance in skin folds. SD of the trunk may appear in the presternal area and in body folds, including the axillae, navel, groin, and inframammary and anogenital areas. Common sites of involvement are hairy areas of the head, including the scalp, the scalp margin, eyebrows, eyelashes, the moustache, the beard, along with the forehead, nasolabial folds, external ear canals, and postauricular creases [2, 5].

**Epidemiology of SD in Asia**

SD has been reported to affect approximately 1–5% of the population worldwide, depending on the country studied [1, 6–8]. A limited number of studies have investigated the epidemiology of SD in Asia [9–14]. In a Korean cross-sectional study of military personnel, SD was ranked as the third most troublesome skin disease after atopic dermatitis and tinea cruris; the prevalence of SD was 2.1% [9]. A study in India reported that 13.4% of children aged <5 years had SD, with the prevalence peaking during infancy and decreasing steadily with age [10]. In Indian adults with scalp dermatoses, 18.7% of cases were attributed to SD [11]. Furthermore, data from Singapore revealed an SD prevalence of 3.2% in children but of 7.0% in adults [12]. In Asian individuals aged 12–20 years, the prevalence of SD varied widely between tropical cities and countries (i.e., Macao 2.7%, Guangzhou 2.9%, Malaysia 17.2%, and Indonesia 26.5%) [13]. A cross-sectional study conducted in Japan found that the prevalence of SD among 67,448 patients attending hospital dermatology clinics was 3.28% [14]. The wide variation in prevalence rates obtained in these studies is likely to be a reflection of the high variability of the SD expression [15].

Similar to Western countries, in Asia there is an increased trend for SD in patients with immunosuppression (e.g., organ transplant recipients, AIDS patients), neurological or psychiatric conditions (e.g., Parkinson’s disease, tardive dyskinesia, depression), or genetic disorders (e.g., Down’s syndrome, cardiofaciocutaneous syndrome) [16]. The prevalence of SD in HIV patients was reported as 47.0% in Thailand, 19.2% in Malaysia, and 17.0% in Korea [17–19].

**Differences between Asian and Non-Asian Skin**

Clinically significant differences between Asian and Caucasian skin have been reported, and these differences may impact the management of SD in Asians. The most obvious difference between ethnic groups relates to skin colour, secondary to the presence of melanin [20]. The photo-protective properties of melanin may influence the rate at which skin ages, with Caucasians showing an earlier onset of photo-aging than Asians [20, 21]. Asian skin is more prone to postinflammatory hyperpigmentation than Caucasian skin [20, 22]. Differences have also been observed in the stratum corneum of Asians compared with that of non-Asians [20]. Although evidence regarding transepidermal water loss in Asian skin is contradictory, there are reports of Asians having a lower transepidermal water loss than other races [20]. However, a study in similarly aged Japanese and German women detected no significant differences in transepidermal water loss between the two races [23]. Similarly, there was no difference in transdermal water loss between Japanese and French volunteers whose physiological parameters were investigated at three different skin sites [24]. The interpretation of such data must take into account the ambient humidity, which can dramatically alter observations. Studies generally indicate that Asians have higher stratum corneum water content [25] and higher stratum corneum lipid levels than other races [20, 26, 27]. Investigations involving the removal of the stratum corneum by tape stripping indicated that Asian skin may have a poor barrier function [28–30]. Asians compared with non-Asians have a heightened dermatological response to irritants commonly found in topical, over-the-counter, or cosmetic preparations [23, 31–35]. In a study in Japanese patients with photo-damaged skin, tretinoin caused a higher than anticipated level of irritation than that previously reported in Caucasians [34]. In another study in which participants underwent a skin patch test on the forearm with sodium laurel sulphate, significant subjective sensory differences were found between Japanese and German women [23]. Increased skin reactivity was observed in Asian subjects compared with Caucasian subjects in an analysis of results compiled from 9 acute irritation patch test studies [31]. Similarly, in a study involving
Caucasian and Japanese women, the acute irritant response tended to be greater in the Japanese volunteers, reaching statistical significance with the stronger irritants [35]. Interestingly, 1 study reported no differences in skin pain perception between Chinese, Malay, and Indian participants [36]. Further well-designed studies comparing the structure and physiology of Asian skin with Caucasian skin are warranted.

Pathogenesis of SD

Although the general causes of SD, including interaction of *Malassezia* spp. with sebaceous lipids, seborrhoea, immune dysfunction, neurogenic factors, and emotional stress [1, 5, 37], are considered similar in Asian and Western countries, ethnicity and geography are significant aspects determining the degree of pathogenic association between *Malassezia* spp. and SD. Both *M. globosa* and *M. restricta* are considered the predominant species in Western countries, whereas a relative predominance of *M. restricta* in lesional skin from SD patients is evident in East Asian countries [38–40]. In Korea, for example, analysis of scalp scales from SD patients revealed the presence of *Malassezia* spp. in 85% of cases, *M. restricta* in 47.5%, but *M. globosa* in only 27.5% [39]. Conversely, in Thailand, a study in infants with SD showed a predominance of *M. furfur* [41].

The extent to which *Malassezia* spp. are associated with the presence of dandruff in SD patients also appears to vary markedly throughout Asia. For instance, Iranian researchers reported that only 24.5% of SD patients with dandruff had positive cultures for *Malassezia* spp. [42]. The corresponding percentage in an Indian study was 84%, and *Malassezia* spp. density was significantly associated (p < 0.001) with dandruff severity [43]. In addition, the rate of *Malassezia* spp. isolation from SD patients was significantly greater (p < 0.01) in Southern rather than other regions of India [43]. Regional climatic conditions need to be taken into account when the pathogenesis of SD is being considered [10, 20]. Heat, humidity, and sweat are known to aggravate SD symptoms, especially scalp itch. Sunlight and the high ultraviolet index typical of tropical climates may also exacerbate SD symptoms. Overall, these findings suggest that regional differences in hereditary aspects of host susceptibility (e.g., skin constitution, inflammation) and in climatic conditions facilitating *Malassezia* spp. growth may affect local distribution and pathogenicity of this opportunistic pathogen. To clarify this contention, more specific studies are needed to assess species-specific molecular typing in large patient groups of diverse ethnicity.

Treatment of SD

The goal of SD treatment is not only to alleviate signs and symptoms of the condition but also to promote normalization of skin structure and function [44]. SD has been found to significantly impact a patient’s quality of life [16], and treatment should be addressed to improve skin symptoms as well as quality of life.

In Western countries, topical treatments with antifungals and anti-inflammatory drugs have been extensively studied in patients with SD [8, 45, 46]. Although guidelines for the treatment of SD are generally lacking [8], recent evidence-based Danish guidelines have recommended antifungal azoles as first-line treatment [47]. The same paper indicates that a short course of topical corticosteroid or topical calcineurin inhibitors, both having an anti-inflammatory effect, may be considered beneficial [47]; systemic treatment of SD with oral antifungals may be advised in selected patients [47, 48].

**Treatment of SD in Asia: Critical Issues**

When treating Asian SD patients, the physician needs to consider not only the possible differences in aetiology between Asian and Western skin, but also a number of other sociological, economic, and cultural differences [49]. For example, the ratio of dermatologists to the overall population is very low in many Asian countries [49], meaning that most patients with SD are generally not treated by dermatologists. In addition, there is a wide availability of over-the-counter medications, cosmetics, and generics as well as a variety of unproven and unorthodox treatments within Asia [49]. This may result in more Asian SD patients self-treating or seeking treatment from beauticians and other non-health-care personnel, therefore increasing the risk of irritating or inappropriate treatments [49]. In addition, significant differences in the acceptance, availability, and insurance support for treatment modalities for dermatological conditions vary from country to country within Asia [49].

In the light of the impact of these various factors, there is a need to have common treatment strategies for SD patients within Asian. Therefore, an expert consensus panel of twelve dermatologists from India, South Korea, Tai-
wan, Malaysia, Vietnam, Singapore, Thailand, the Philippines, Indonesia, and Italy was convened in Singapore on the 26–27 September 2014.

**Consensus Recommendations for SD in Asia**

The specific practice recommendations identified by this consensus group for the treatment of SD in Asian adults and infants are outlined in the subsequent subsections. The panel used a consensus approach to determine recommendations about each clinical aspect addressed; this approach was based on grade levels in the Strength-of-Recommendation Taxonomy (SORT) scheme [50]. Each recommendation was also graded by the level of evidence according to the March 2009 Oxford Centre for Evidence-Based Medicine levels of evidence [51].

**Treatment**

**SD of the Scalp and Hairy Areas**

In adults, SD is a chronic condition that is likely to recur after treatment (category A, level 2b). Hence, patients should be counselled about the need for proper skin care [5]. Treatment selection should consider drug efficacy,
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potential for side effects as well as cosmetic acceptability (category B, level 4). Wherever possible, patient self-treatment should be avoided, to minimize the likelihood of inappropriate treatment, SD symptom exacerbation, and variability in treatment response.

For SD of the scalp and hairy areas, the panel recommends the treatments summarized in table 1 (category A, level 1b). For mild forms, a topical approach is recommended starting with ketoconazole or ciclopirox, or alternatively selenium sulphide/zinc pyrithione, or keratolytic shampoos [8, 52]. Similarly to non-scalp SD [53–56], non-steroidal and anti-inflammatory with antifungal properties (AIAFp) shampoo may represent a viable option, as reported in a recent randomized, single-blind clinical trial [57]. In case of failure, add a 4-week course with a weak-to-moderately potent corticosteroid [class I and II according to the Anatomical Therapeutic Chemical (ATC) classification by the World Health Organization (WHO)] followed by its gradual discontinuation [52].

For moderate-to-severe forms, especially if itchy, a combination of antifungal or AIAFp shampoo with weak-to-moderately potent (class I–II) topical corticosteroids for up to 4 weeks is recommended [52]; in case of no improvement, consider to include in the weekly routine treatment with antifungal or AIAFp shampoo, 2 days of potent-to-very potent topical corticosteroid shampoo (class III and IV), containing fluocinolone acetonide 0.01% (class III) or clobetasol propionate 0.05% (class IV) for up to 2 weeks [45, 58–60] (category A, level 1b). In case of more resistant disease, systemic antifungals may be added [47, 48]. For long-term maintenance, antifungal, AIAFp, or other shampoos active on SD may be used once or twice weekly (category B, level 5).

SD of Non-Scalp Areas

For SD of the non-scalp areas, the panel recommends the treatments summarized in table 2 (category A, level 1b). For the treatment of mild, non-scalp SD in adults, especially on the face, the use of antifungal creams (e.g., ketoconazole 2% cream, ciclopirox 1% cream) or AIAFp cream is preferred [52]. Topical antifungals represent the most common approach, and in the past years AIAFp cream has clearly demonstrated efficacy and tolerability in the treatment of mild-to-moderate SD of the face [53–56, 61] (category A, level 1b). In case of failure, a combination of both antifungals and AIAFp agents may be considered. If no improvement is seen or in case a more rapid control of SD signs and symptoms is desired, a weakly potent topical corticosteroid (class I according to the

### Table 2. Treatment products for non-scalp SD

<table>
<thead>
<tr>
<th>Class of product</th>
<th>Formulation</th>
<th>Instructions for use</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild SD</td>
<td></td>
<td>Have to be continued until a clear improvement is achieved</td>
</tr>
<tr>
<td>Topical antifungals</td>
<td>Ciclopirox 1% cream</td>
<td>Twice daily for 4 weeks</td>
</tr>
<tr>
<td></td>
<td>Ketoconazole 2% cream</td>
<td>Twice daily for 4 weeks</td>
</tr>
<tr>
<td></td>
<td>e.g. Piroctone olamine/algycera/bisabolol cream</td>
<td>Twice daily for 4 weeks</td>
</tr>
<tr>
<td>AIAFp</td>
<td>Piroctone olamine/algycera/bisabolol cream</td>
<td>Twice daily for 4 weeks</td>
</tr>
<tr>
<td></td>
<td>Pimecrolimus 1% cream</td>
<td>Twice daily for 4 weeks</td>
</tr>
<tr>
<td></td>
<td>Tacrolimus 0.1% ointment</td>
<td>Twice daily for 4 weeks</td>
</tr>
<tr>
<td>Topical corticosteroids (class I)</td>
<td>Desonide 0.05% cream</td>
<td>First month: 200 mg/day for 1 week, then 200 mg/day for 2 days/month up to 11 months</td>
</tr>
<tr>
<td></td>
<td>Hydrocortisone 1% cream and ointment</td>
<td>Continuous regimen: 250 mg/day for 4–6 weeks</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Intermittent regimen: 250 mg/day for 12 days per month for 3 months</td>
</tr>
<tr>
<td></td>
<td></td>
<td>50 mg/day for 2 weeks or 200–300 mg weekly for 2–4 weeks</td>
</tr>
<tr>
<td>Topical calcineurin inhibitors*</td>
<td></td>
<td>First month: 200 mg/day for 1 week, then 200 mg/day for 2 days/month up to 11 months</td>
</tr>
<tr>
<td>Moderate-to-severe SD</td>
<td></td>
<td>Continuous regimen: 250 mg/day for 4–6 weeks</td>
</tr>
<tr>
<td>Topical corticosteroids</td>
<td>Alclometasone 0–05% ointment</td>
<td>First month: 200 mg/day for 1 week, then 200 mg/day for 2 days/month up to 11 months</td>
</tr>
<tr>
<td>(class II)</td>
<td>Desonide 0.05% cream</td>
<td>Continuous regimen: 250 mg/day for 4–6 weeks</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Intermittent regimen: 250 mg/day for 12 days per month for 3 months</td>
</tr>
<tr>
<td></td>
<td></td>
<td>50 mg/day for 2 weeks or 200–300 mg weekly for 2–4 weeks</td>
</tr>
<tr>
<td>Systemic antifungals</td>
<td>Itraconazole 100-mg caps</td>
<td>First month: 200 mg/day for 1 week, then 200 mg/day for 2 days/month up to 11 months</td>
</tr>
<tr>
<td></td>
<td>Terbinafine 250-mg caps</td>
<td>Continuous regimen: 250 mg/day for 4–6 weeks</td>
</tr>
<tr>
<td></td>
<td>Fluconazole 50-mg caps</td>
<td>Continuous regimen: 250 mg/day for 4–6 weeks</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Intermittent regimen: 250 mg/day for 12 days per month for 3 months</td>
</tr>
</tbody>
</table>

* Off-label use.
ATC by WHO) once or twice daily for up to 2 weeks may be added [4]. If successful, the use of a topical corticosteroid may be extended for an additional 2 weeks. The use of AIAFp cream may be considered for maintenance treatment.

For moderate-to-severe non-scalp SD, topical moderately potent corticosteroids (class II according to the ATC by WHO) may be used up to a maximum of 2 weeks in combination with AIAFp or topical antifungals. This approach will achieve the most rapid control of SD signs and symptoms. In case of clinical improvement, the use of a topical corticosteroid may be considered for an additional 2 weeks. If the response is not satisfactory, the use of systemic antifungals should be considered. Finally, topical calcineurin inhibitor agents may represent an alternative for mild-to-severe SD refractory cases (category A, level 1b).

The above recommendations are summarized in figures 1 and 2.

**Treatment in Infants**

SD of the Scalp and Hairy Areas

SD management in infants involves advising simple measures, such as regular washing of the scalp with baby shampoo and gentle brushing to loosen scales [62]. The daily use of white petrolatum may help to soften scales. If these measures are not effective, ketoconazole 2% shampoo could be used until the condition resolves [62, 63] (category A, level 1b). The clinical efficacy of AIAFp cream in infants has been demonstrated in a multicentre,
double-blind, placebo-controlled, parallel-group study in infants with cradle cap, in whom there was a significant difference in the reduction of scaling between the treatment and placebo groups [64].

**SD of Non-Scalp Areas**

The use of ketoconazole 2% cream is advised alone or together with a weakly potent topical corticosteroid (class I according to the ATC by WHO) [65] (category A, level 5). All therapeutic options are listed in table 3.

**Conclusions**

Because the skin of Asians compared with non-Asians is more reactive to irritants, topical agents with irritant potential, and therefore with the likelihood to complicate SD lesions [31], should be avoided. In particular, cosmetic products containing alcohol, soap and shaving cream, greasy emollients, and any known trigger factors, if they cause irritation, should be changed for more gentle products. Finally, the presence of dry or damp conditions in...
the workplace/living place, a facilitating factor for SD common in many Asia countries, should also be considered [66] (category B, level 5).

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### Statement of Ethics

Published research complies with the guidelines for human studies and animal welfare regulations.

### Disclosure Statement

The authors have received honoraria for their participation in the Asia Pacific Seborrhoeic Dermatitis Leaders’ Summit 2014. Dr. Wai Kwong Cheong is a speaker for Galderma and for L’Oréal and a member of the Advisory Board for P & G Olay Pro-X Global Dermatologist Alliance.

### Table 3. Treatment products for infantile SD

<table>
<thead>
<tr>
<th>Class of product</th>
<th>Formulation</th>
<th>Instructions for use</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Scalp and hairy areas</strong></td>
<td><strong>Topical antifungals</strong></td>
<td>Ketoconazole 2% shampoo</td>
<td>Shampoo: twice a week for 4 weeks</td>
</tr>
<tr>
<td><strong>Emollients</strong></td>
<td>White petrolatum ointment</td>
<td>Daily use</td>
<td>Softens scales to help ease manual removal (e.g. with a soft brush)</td>
</tr>
<tr>
<td><strong>AIAFp</strong></td>
<td>e.g. Piroctone olamine/alglycerol/ bisabolol cream</td>
<td>Every 12 h</td>
<td>Effective for cradle cap</td>
</tr>
<tr>
<td><strong>Non-scalp areas</strong></td>
<td><strong>Topical antifungals</strong></td>
<td>Ketoconazole 2% cream</td>
<td>Once daily for up to 7 days</td>
</tr>
<tr>
<td></td>
<td><strong>Topical corticosteroids (class I)</strong></td>
<td>Hydrocortisone 1% cream</td>
<td>Once daily for up to 7 days</td>
</tr>
</tbody>
</table>

### References

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