Clinical, Cosmetic and Investigational Dermatology

Dovepress
Taylor & Francis Group

Open Access Full Text Article

ORIGINAL RESEARCH

Comparative Profile of Microbiome in Normal Skin and Acne Vulgaris Skin Patients

Irma Bernadette S Sitohang[1], Lili Legiawati[1], Sandra Widaty[1], Hanny Nilasari[1], Triana Agustin[1], Sari Chairunnisa[2], Teguh Hopkop Putera Manurung[1]

[1]Department of Dermatology and Venereology, Faculty of Medicine, Universitas Indonesia – Dr. Cipto Mangunkusumo National Central General Hospital, Jakarta, Indonesia; [2]Paragon Technology and Innovation Ltd., Jakarta, Indonesia

Correspondence: Irma Bernadette S Sitohang, Department of Dermatology and Venereology, Faculty of Medicine, Universitas Indonesia – Dr. Cipto Mangunkusumo National Central General Hospital, Jl. Diponegoro No. 71, Jakarta Pusat, Jakarta, 10430, Indonesia, Tel +62 818 130 761, Email irma_bernadette@yahoo.com

Clinical, Cosmetic and Investigational Dermatology downloaded from https://www.dovepress.com/
For personal use only.

Introduction: Changes in the skin microbiome are associated with acne vulgaris (AV), a condition characterized by comedones, papules, and pustules. While some bacteria have been studied, many others remain unexplored, highlighting the need to understand the microbiome differences between acne-prone and normal skin. This study aimed to compare skin microbiome profiles between AV patients and healthy individuals and to explore associations with microbial diversity and specific bacterial populations involved in AV pathogenesis.

Methods: A total of 144 participants were recruited, comprising 36 AV patients and 108 healthy controls. Skin samples were collected from the left cheek after standardized preconditioning. DNA was extracted using the DNeasy PowerSoil Kit™, and the V3–V4 region of 16S rRNA was amplified and sequenced. Microbial diversity was assessed by the Shannon index, and correlations with sebum levels were analyzed.

Results: The results revealed significant differences in microbial diversity, with AV patients exhibiting a markedly lower Shannon index compared to controls, indicating decreased microbial diversity and potential dysbiosis. While the relative abundance of *Cutibacterium acnes*, a bacterium commonly associated with AV, showed no significant differences between the two groups, the prevalence of *Staphylococcus epidermidis* was notably higher in AV patients. This suggests that *S. epidermidis* may play a complex role in the inflammatory processes associated with AV. Moreover, the study identified a negative correlation between microbial diversity and sebum levels, suggesting that increased sebum production may favor the growth of *S. epidermidis*, potentially exacerbating the condition.

Conclusion: These findings highlight the interaction between host factors and microbial composition. This study emphasizes the role of skin microbiome dysbiosis in acne vulgaris and provides insights for future microbiome-based therapeutic strategies. Further research is needed to clarify microbial mechanisms and potential interventions targeting the microbiome in the management of acne vulgaris.

Keywords: acne vulgaris, skin microbiome, acne grading, *Staphylococcus*, 16SrRNA sequencing

Introduction

The human skin, the body's largest organ, plays a crucial role as a protective barrier against external insults. Resident microorganisms on the skin contribute to its protective function, and any alterations in their composition have been associated with various skin conditions, including acne vulgaris.[1]

Acne vulgaris is a persistent skin condition characterized by specific manifestations like comedones, papules, pustules, and nodules. Its development is linked to factors such as increased sebum production, abnormal keratinization of follicles, bacterial colonization, and inflammation. *Cutibacterium acnes* (*C. acnes*), a common bacterial species in sebaceous gland-rich skin areas, is suggested as a potential acne-causing pathogen.[1] Despite its ability to produce proinflammatory free fatty acids from sebum, studies have presented conflicting results, with *C. acnes* not consistently elevated in acne-afflicted skin compared to normal skin.[2,3]

https://doi.org/10.2147/CCID.S549168
Received: 24 June 2025
Accepted: 30 September 2025
Published: 7 January 2026

© 2026 Sitohang et al. This work is published and licensed by Dove Medical Press Limited. The full terms of this license are available at https://www.dovepress.com/terms.php and incorporate the Creative Commons Attribution – Non Commercial (unported, v4.0) License (http://creativecommons.org/licenses/by-nc/4.0/). By accessing the work you hereby accept the Terms. Non-commercial uses of the work are permitted without any further permission from Dove Medical Press Limited, provided the work is properly attributed. For permission for commercial use of this work, please see paragraphs 4.2 and 5 of our Terms (https://www.dovepress.com/terms.php).

The emergence of acne vulgaris is also associated with other skin microorganisms. The term "dysbiosis," referring to an imbalance in the relative abundance of the entire skin microbiome, may contribute to acne vulgaris by affecting innate and adaptive immune responses within pilosebaceous follicles. The specific roles of individual microorganisms in the pathogenesis of acne vulgaris remain undefined.[1,3] While *Staphylococcus aureus* (*S. aureus*) and *Staphylococcus epidermidis* (*S. epidermidis*) have been well-studied due to their proximity to the skin, many other minor skin microorganisms still lack comprehensive examination.

The microbiome comprises bacteria, viruses, fungi, their genes, metabolites, and surrounding environment, whereas microbiota refers to microorganisms within a specific niche. Microbial cells outnumber human cells by approximately tenfold, and skin and gut microbes are essential for immune, hormonal, and metabolic homeostasis.[3] The bacterial composition varies between individuals and across body sites, influenced by environmental factors such as soaps, cosmetics, antibiotics, occupation, temperature, humidity, and UV exposure.[3]

Consequently, gaining a better understanding of the microbiome profile in acne vulgaris compared to normal skin is crucial. This study highlights the role of skin microbiome dysbiosis in the pathogenesis of acne vulgaris and provides a foundation for future microbiome-based therapeutic strategies. Further research is required to elucidate microbial mechanisms and to develop targeted interventions for the management of acne vulgaris.

Materials and Methods

Subject Recruitment

Thirty-two participants with acne vulgaris and one-hundred-eight controls were recruited consecutively during November 2022 to March 2023. Acne vulgaris diagnosis was made by dermatologists after thorough physical examination of each participant's face. Only participants with no visible acne lesions and acne scars were included as controls. The exclusion criteria were as follows: pregnancy, presence of other skin diseases on the face, presence of psoriasis or atopic eczema lesions in any part of the body, acne conglobata, primary or secondary immunodeficiency diseases, consumption of oral antibiotics, immunosuppressants, immunomodulators, hormones, or retinoids within 1 month before enrolment, history of using any topical acne prescription within 2 weeks before enrolment. Written informed consent following guidance from Declaration of Helsinki was taken from each participant. This study was approved by the ethics committee of Dr. Cipto Mangunkusumo National Central General Hospital, Faculty of Medicine, Universitas Indonesia. The ClinicalTrials.gov registration number of this study is NCT05838534. Informed consent was obtained, and ethical approval was granted by the Health Research Ethics Committee of FKUI-RSCM (No. KET-482/UN2.F1/ETIK/PPM.00.02/2022, dated May 23, 2022).

Sample Collection

Skin microbiome samples were obtained from the left cheek using sterile swabs pre-moistened with DNA/RNA Shield solution. Each swab was applied with uniform pressure in vertical, horizontal, and diagonal strokes across a 4 × 4 cm area for approximately 50 seconds. The swab tip was then aseptically detached and placed into a collection tube containing DNA/RNA Shield. Samples were transported in an insulated container with dry ice (~4°C) and subsequently stored at –80°C within 24 hours of collection.

Prior to sampling, all participants were instructed to cleanse their face exclusively with a standardized mild facial cleanser provided by the investigators for at least one week. On the day of collection, the application of any topical products or cosmetics was prohibited for a minimum of 12 hours.

DNA Extraction and Sequencing

DNA extraction followed the DNeasy PowerSoil Kit™ protocol (Qiagen, CA, USA). After extraction, DNA purity and concentration were assessed, with each sample required to exhibit a DNA purity value between 1.6 and 2 and a quantity equal to or below 0.05 ng/mL. The amplification of the V3 and V4 hypervariable regions of the 16SrRNA gene utilized the forward primer 5′-CCTAYGGGRBGCASCAG-3′ and the reverse primer 5′-GGACTACNNGGGTATCTAAT-3′. Post-amplification, DNA concentration in all samples was gauged, and random samples underwent agarose gel testing

to confirm the presence of the 16SrRNA gene. The anticipated 16SrRNA concentration in each sample should be equal to or greater than 2nM. Prior to sequencing, the Nextera XT DNA library preparation kit (Illumina, CA, USA) was used for indexing. Sequencing was conducted on the Illumina MiSeq platform (Illumina, CA, USA).

Bioinformatic Analysis

The analysis was conducted with Quantitative Observation of Microbial Ecology (QIIME) version 1.7.0. Raw data from all samples underwent filtering, trimming, merging, and denoising through the USEARCH plugin. In cases where the quality of one read was subpar (Phred score <20 and duplications <50%), only the higher-quality read was considered for analysis. Annotation of operational taxonomic units (OTUs) followed the Ribosomal Database Project guidelines. OTUs were incorporated into the final analysis if their proportion exceeded 0.1% in each sample, and they had to be present in at least 20% of the samples.

Statistical Analysis

The relative abundance of each microorganism found in samples were presented in percentage. Alpha diversity, measurement of microbial diversity within each sample, was counted using the Shannon index. To compare alpha diversity and the relative abundance of microorganisms across three acne grades, the Kruskal–Wallis test was used. Statistical significance was considered for p-values less than 0.05. If the comparisons were deemed statistically significant, the Mann–Whitney *U*-test was then conducted between two groups.

Results

In this study, a total of 144 participants were recruited. The sociodemographic and clinical characteristics of the participants were provided in Table 1. The mean age of participants was 25.6 ± 3.2 years, and the majority were female. Participants with AV has significantly higher history of AV in first degree family compared to controls ($p < 0.05$). Most participants wash their face two to three times per day with various soaps either moisturizing soap, non-moisturizing soap, or antiseptics. About 5% of controls have been diagnosed and treated for AV in the past. There is no significant difference in the mean face sebum excretion amount measured using sebumeter between AV and controls.

All samples went through DNA extraction and sequencing processes. Sixteen samples from AV participants and five samples from controls had less than 1,000 reads and cannot be analysed further. The average read in each sample is 63,008.

Table 1 Sociodemographic and Clinical Characteristics of the Participants

Acne Grades		**Controls (n = 36)**	**Acne Vulgaris (n = 108)**	**p Value**
Age (years)		25 (19–40)	26 (18–39)	
Genders, n (%)	Male	15 (41.7)	24 (22.2)	
	Female	21 (58.3)	84 (77.8)	
Educations, n (%)	Middle school to college	11 (30.6)	47 (43.5)	
	University or higher	25 (69.4)	61 (56.5)	
Family history of acne, n (%)	Yes	8 (22.2)	46 (42.6)	0.047[‡,*]
	No	28 (77.8)	62 (57.4)	
Daily facial washing frequency, n (%)	<2 times	7 (19.4)	7 (6.5)	0.08[§]
	2–3 times	28 (77.8)	97 (89.8)	
	>3 times	1 (2.8)	4 (3.7)	
Soap type, n (%)	Moisturizing	19 (52.8)	49 (45.4)	
	Non-moisturizing	15 (41.7)	55 (50.9)	
	Antiseptics	1 (2.8)	3 (2.8)	
	None	0 (2.8)	1 (0.9)	
History of acne medications/procedure, n (%)	Yes	2 (5.6)	20 (18.5)	0.11[§]
	No	34 (94.4)	88 (82.5)	
Mean Facial Sebum Excretion (mg/cm)		61.6 (10.4–172)	77.3 (11–338.4)	0.16[¶]

Notes: [‡]Using Chi-square test. [§]Using Fischer's exact test. [¶]Using Mann–Whitney *U*-test. *Statistically significant.

Table 2 Comparison of Shannon Index and Relative Abundance of Microbiome Between Study Participants

Acne Grades	Controls (n = 31)	Acne Vulgaris (n = 92)	p Value
Shannon Index	2.96 (1.1–4.2)	2.4 (1.2–4.7)	<0.001†,*
Relative abundance of *C. acnes* (%)	29.5 (0.2–60)	31.8 (0.3–85.4)	0.83†
Relative abundance of *S. epidermidis* (%)	0.1 (0–0.5)	0.2 (0–2)	0.02†,*
Others (%)	70 (39.9–99.3)	68.2 (14.4–99.7)	0.78†

Notes: †Presented in median (range), using Mann–Whitney test. *Statistically significant.

Table 3 Correlation Between Mean Facial Sebum Excretion with Shannon Index and Relative Abundance of Microbiome Among Study Participants

Mean Facial Sebum Excretion (n = 123)	Pearson's Correlation Coefficient	p Value
Shannon Index	−0.285	<0.001*
Relative abundance of *C. acnes*	0.046	0.613
Relative abundance of *S. epidermidis*	0.233	0.01*
Relative abundance of other bacteria	−0.05	0.583

Note: *Statistically significant.

The average length of each read is 191 bp. The total reads in all samples were 9,073,415. There were 1,451 unique OTUs in total.

The comparison of the relative abundance of observed species and Shannon diversity index between groups was shown in Table 2. The Shannon index was higher in controls compared to AV participants and statistically significant ($p < 0.001$). The value is below 3 in almost all samples which may signify low bacterial diversity in all samples. The comparison of *C. acnes'* relative abundance was not remarkable. None of our samples showed the presence of *S. aureus*. The relative abundance of *S. epidermidis* is higher in AV participants and statistically significant. Species with relative abundance above 0.1% and can be detected in at least 20% samples were taken into final analysis. Other lesser species constitute more than 50% of the microbiota found in each sample, yet there is no difference in the proportion between both samples. We also did correlation analysis of MFSE and the previous parameters in Table 3 and found significant correlation between MFSE and Shannon Index and relative abundance of *S. epidermidis*.

Discussion

Several individual factors might affect the outcome in a study comparing microbiome profile between AV patients and controls. Fortunately, most of those factors are comparable between groups in this study, except for family history of AV. A family history can increase the risk of AV by up to three times, especially if the history comes from parents.[4] Family members may share similar skin microbiomes due to genetic factors, environmental exposures, and lifestyle choices. Song reported family members living under the same roof shared similar skin microbiota. However, this finding is heavily affected by the frequent skin-to-skin contact between individuals.[5] Furthermore, it is not possible to segregate individuals based on family relation between participants in this study.

The Shannon index is used to assess alpha diversity, indicating the number and balance of species in a habitat, such as skin samples. A lower index suggests fewer species and dysbiosis.[6] While previous studies found no significant differences in dominant microbiota between acne vulgaris (AV) patients and healthy individuals, this study noted an increase in rare bacteria in AV. The Shannon index was lower in AV patients compared to controls, contrasting with some studies that reported no significant difference. Methodological variations in sample collection may account for these discrepancies (pore stripping vs skin swabbing).[2,7] The decrease in diversity in AV may lead to an increase in pathogenic microorganisms. Furthermore, commensal microorganisms have a specific role in educating both innate and adaptive immunity. Disruption of this microbial composition may alter skin conditions to become more pro-inflammatory.

 https://doi.org/10.2147/CCID.S549168

Several microorganisms, namely *C. acnes* and various *Staphylococcus* species, are the most abundant inhabitants of human skin. *C. acnes* is considered a causative bacterium of AV because it can enhance sebaceous gland lipogenesis, keratinocyte differentiation, and local inflammatory.[8] In this study, no significant differences were found in the proportion of *C. acnes* among AV patients and normal. This finding supports the research by Barnard et al, which reported that AV is more related to the presence of increased proportions of certain strains of *C. acnes*, specifically RT4, RT5, and RT8 strains.[2] We did not perform whole genome sequencing in this study, therefore we could not analyze the differences in *C. acnes* strains.

S. aureus was not detected in any skin samples, aligning with findings from other studies that report low proportions (less than 0.1%) in AV lesions. This bacterium is believed to exacerbate inflammation in AV but is typically identified in throat or nasal swabs where it is more abundant.[9,10] In contrast, *S. epidermidis* was found in greater numbers in AV participants compared to controls. However, the role of *S. epidermidis* in AV is still unclear. While it could potentially inhibit pathogenic bacteria like *C. acnes* through competition or antimicrobial peptide production, this study found no reduction in *C. acnes* levels in AV patients. Another hypothesis suggests that the increased *S. epidermidis* may be related to its biofilm formation, which could block sweat glands and provoke inflammation.[11,12] Further research, including whole genome sequencing and biofilm analysis, is needed to clarify these findings.

Various other microorganisms were also identified from skin samples in this study, including *S. cohnii, C. macginleyi, S. caprae*, and *S. hominis*. There were no overall differences in the number of these microorganisms between AV samples and controls, nor across different severity levels of AV. These microorganisms are commensal bacteria on human skin, but *C. macginleyi, S. caprae*, and *S. capitis* can develop into opportunistic pathogens.[13,14] The role of these microorganisms in AV has not been extensively explored, but these findings indicate an imbalance in composition.

The composition of skin microorganisms is influenced by physiological factors like sebum and moisture. In this study, samples were taken from the cheeks, which are less seborrheic. A negative correlation was found between skin microbial diversity, measured by the Shannon index, and sebum levels, mirroring findings from a similar study in India.[15] Sebum provides substrates for lipophilic bacteria like *C. acnes*, which can convert oleic acid into propionic and acetic acids that inhibit other bacteria, leading to decreased microbial diversity as sebum levels rise. However, this study found no correlation between sebum levels and the proportion of *C. acnes*, likely due to limitations in sampling. Conversely, *S. epidermidis* showed a positive correlation with sebum levels. While previous research has focused on the genus level, findings indicate that *S. epidermidis* can metabolize fatty acids in sebum and produce succinic acid, enabling it to thrive in seborrheic environments. This is supported by reports that *S. epidermidis* can survive across varying sebum levels.[16] Additionally, the significant increase of *S. epidermidis* relative to *C. acnes* suggests that the more seborrheic conditions on the cheeks, which come with increased moisture, favor *S. epidermidis*.[15]

Acne vulgaris (AV) is a chronic disorder of the pilosebaceous unit that predominantly affects adolescents and is characterized by polymorphic lesions, including comedones, papules, pustules, cysts, nodules, scarring, and dyspigmentation. An acneiform presentation of cutaneous lupus erythematosus (CLE) is extremely rare but may clinically mimic acne.[17]

The severity of AV may also be associated with dysbiosis of commensal skin flora, particularly *Staphylococcus caprae*and *S. epidermidis*.[18] The predominant bacterial isolates identified were *S. epidermidis* (50.5%), *C. acnes* (11.0%), and *S. aureus* (7.7%). Among available antibiotics, doxycycline and minocycline demonstrated the highest efficacy, whereas resistance to erythromycin and clindamycin was frequently observed.[19]

Standard therapy for acne vulgaris in Indonesia, as recommended by the national clinical practice guidelines, was evaluated in a retrospective observational study. We found at baseline, 63.4% of patients presented with moderate acne.[20] Most patients received a combination of retinoic acid, benzoyl peroxide, and either topical or oral antibiotics, with several adjuvant such as comedone extraction,[21] chemical peeling, pulse dye laser and others. Observing after 12 weeks of treatment, a significant reduction in the median number of non-inflammatory lesions, inflammatory lesions and total lesions significantly reduction ($p < 0.001$).[20] These findings support the implementation of national guidelines and the promotion of antimicrobial stewardship in Indonesia.[20]

Isotretinoin remains the most effective therapy, targeting all major pathogenic factors of acne, although it requires close monitoring. In contrast, topical tretinoin is safe, well tolerated, and effective not only for AV but also for photoaging.[22,23]

As adjunctive therapy, nicotinamide combined with zinc and antibacterial adhesive (ABA) has shown efficacy in reducing non-inflammatory lesions within two weeks, suggesting a potential role in improving early treatment

outcomes.[24] Meanwhile, although serum vitamin D levels in the study population were low (mean 17.29 ng/mL), no significant correlation was observed with acne severity.[25]

Conclusion

In conclusion, this study highlights significant differences in the skin microbiome of AV patients compared to healthy individuals, particularly reflected in the Shannon index, which indicates decreased microbial diversity in AV. Despite no notable changes in the proportion of *C. acnes*, the increase in rare bacteria and the higher prevalence of *S. epidermidis* in AV patients, suggests complex interactions that may influence inflammation and skin health. The findings underscore the need for further investigation into specific strains of *C. acnes*, the role of *S. epidermidis* and its implications for AV pathogenesis. Additionally, the study reveals how physiological factors like sebum levels can affect microbial composition, with increased sebum potentially favoring *S. epidermidis*. Overall, these insights contribute to understanding the dysbiosis associated with AV and serve as foundation for future research and therapeutic approaches.

Acknowledgments

We acknowledge the Faculty of Medicine, Universitas Indonesia–Dr. Cipto Mangunkusumo Hospital, as well as the health personnel of the Department of Dermatology and Venereology and the Clinical Research and Supporting Unit, Faculty of Medicine, Universitas Indonesia. We also acknowledge the financial support provided by Paragon Technology and Innovation, Ltd.

Funding

This research was fully funded by Paragon Technology and Innovation, Ltd. No. Grant: HK.02.03/2.2/37409/2022.

Disclosure

The authors report no conflicts of interest in this work.

References

1. Ramasamy S, Barnard E, Dawson TL, Li H. The role of the skin microbiota in acne pathophysiology. *Br J Dermatol*. 2019;181(4):691–699. doi:10.1111/bjd.18230
2. Barnard E, Shi B, Kang D, Craft N, Li H. The balance of metagenomic elements shapes the skin microbiome in acne and health. *Sci Rep*. 2016;6:39491. doi:10.1038/srep39491
3. Lee YB, Byun EJ, Kim HS. Potential role of the microbiome in acne: a comprehensive review. *J Clin Med*. 2019;8(7):987. doi:10.3390/jcm8070987
4. Heng AHS, Chew FT. Systematic review of the epidemiology of acne vulgaris. *Sci Rep*. 2020;10(1):5754. doi:10.1038/s41598-020-62715-3
5. Song SJ, Lauber C, Costello EK, et al. Cohabiting family members share microbiota with one another and with their dogs. *eLife*. 2013;2:e00458. doi:10.7554/eLife.00458
6. Finotello F, Mastrorilli E, Di Camillo B. Measuring the diversity of the human microbiota with targeted next-generation sequencing. *Brief Bioinform*. 2018;19(4):679–692. doi:10.1093/bib/bbw119
7. Shi J, Cheng JW, Zhang Q, Hua ZX, Miao X. Comparison of the skin microbiota of patients with acne vulgaris and healthy controls. *Ann Palliat Med*. 2021;10:7933–7941. doi:10.21037/apm-21-1482
8. Xu H, Li H. Acne, the skin microbiome, and antibiotic treatment. *Am J Clin Dermatol*. 2020;20(3):335–344. doi:10.1007/s40257-018-00417-3
9. Khorvash F, Abdi F, Kashani HH, Naeini FF, Narimani T. *Staphylococcus* aureus in acne pathogenesis: a case-control study. *N Am J Med Sci*. 2012;4(11):573–576. doi:10.4103/1947-2714.103317
10. Numata S, Akamatsu H, Akaza N, et al. Analysis of facial skin-resident microbiota in Japanese acne patients. *Dermatology*. 2014;228(1):86–92. doi:10.1159/000356777
11. Claudel JP, Auffret N, Leccia MT, Poli F, Corvec S, Dréno B. *Staphylococcus epidermidis*: a potential new player in the physiopathology of acne? *Dermatology*. 2019;235(4):287–294. doi:10.1159/000499858
12. Allen HB, Vaze ND, Choi C, et al. The presence and impact of biofilm-producing staphylococci in atopic dermatitis. *JAMA Dermatol*. 2014;150(3):260–265. doi:10.1001/jamadermatol.2013.8627
13. Severn MM, Williams MR, Shahbandi A, et al. The ubiquitous human skin commensal *Staphylococcus hominis*protects against opportunistic pathogens. *mBio*. 2022;13(3):e0093022. doi:10.1128/mbio.00930-22
14. Ito Y, Sasaki T, Li Y, et al. *Staphylococcus cohnii* is a potentially biotherapeutic skin commensal alleviating skin inflammation. *Cell Rep*. 2021;35(4):109052. doi:10.1016/j.celrep.2021.109052
15. Mukherjee S, Mitra R, Maitra A, et al. Sebum and hydration levels in specific regions of human face significantly predict the nature and diversity of facial skin microbiome. *Sci Rep*. 2016;6:36062. doi:10.1038/srep36062
16. Swaney MH, Nelsen A, Sandstrom S, Kalan LR. Sweat and sebum preferences of the human skin microbiota. *Microbiol Spectr*. 2023;11(1):e0418022. doi:10.1128/spectrum.04180-22

17. Sitohang IBS, Fitri EM, Mohamad Rheza L, Suseno S, Sirait SP. Acne vulgaris mimicking cutaneous lupus erythematosus in an adolescent: report of a rare case. *Case Rep Dermatol*. 2021;13:69–74. doi:10.1159/000511530
18. Manurung THP, Sitohang IBS, Agustin T. *Staphylococcus* caprae and *Staphylococcus* epidermidis define the skin microbiome among different grades of acne vulgaris. *Arch Dermatol Res*. 2024;317(1):156. PMID: 39738874; PMCID: PMC11685260. doi:10.1007/s00403-024-03581-1
19. Sitohang IBS, Fathan H, Effendi E, Wahid M. The susceptibility of pathogens associated with acne vulgaris to antibiotics. *Med J Indones*. 2019;28 (1):21–27. doi:10.13181/mji.v28i1.2735
20. Wiraputranto MC, Sitohang IBS, Sampurna AT, Ilyas M. Effectiveness of standard therapy for acne vulgaris based on clinical practice guidelines in Indonesia. *Clin Cosmet Invest Dermatol*. 2024;17:2165–2175. doi:10.2147/CCID.S469143
21. Sitohang IBS, Soebaryo RW, Kanoko M. Acne lesion extraction versus oral doxycycline for moderate acne vulgaris: a randomized clinical trial. *J Clin Aesthetic Dermatol*. 2021;14(6):E61–E65.
22. Sitohang IBS. Isotretinoin for treating acne vulgaris. *Int J Appl Pharm*. 2021;13(2):19–24. doi:10.22159/ijap.2021v13i2.40045
23. Sitohang IBS, Makes WI, Sandora N, Suryanegara J. Topical tretinoin for treating photoaging: a systematic review of randomized controlled trials. *Int J Womens Dermatol*. 2022;8:e003. doi:10.1097/JW9.0000000000000003
24. Sitohang IBS, Yahya YF, Simanungkalit R, Adi Winarni DR, Madjid A. Efficacy and tolerability of topical nicotinamide plus antibacterial adhesive agents and zinc-pyrrolidone carboxylic acid versus placebo as an adjuvant treatment for moderate acne vulgaris in Indonesia: a multicenter, double-blind, randomized, controlled trial. *J Clin Aesthetic Dermatol*. 2020;13(7):27–31.
25. Saptarini D, Witjaksono F, Sitohang IBS. Correlation study of acne vulgaris and serum vitamin D levels in adolescents. *Int J Appl Pharm*. 2020;12 (Special Issue 3):69–73. doi:10.22159/ijap.2020.v12s3.39462

Clinical, Cosmetic and Investigational Dermatology

Dovepress
Taylor & Francis Group

Publish your work in this journal

Clinical, Cosmetic and Investigational Dermatology is an international, peer-reviewed, open access, online journal that focuses on the latest clinical and experimental research in all aspects of skin disease and cosmetic interventions. This journal is indexed on CAS. The manuscript management system is completely online and includes a very quick and fair peer-review system, which is all easy to use. Visit http://www.dovepress.com/testimonials.php to read real quotes from published authors.

Submit your manuscript here: https://www.dovepress.com/clinical-cosmetic-and-investigational-dermatology-journal