

The Effects of Oral Vitamin D Supplement on Atopic Dermatitis: A Clinical Trial with *Staphylococcus aureus* Colonization Determination

Montree Udompataikul MD*,
Sunsern Huajai MD*, Thep Chalermchai MD*,
Malai Taweechotipatr PhD**, Nanticha Kamanamool MD***

* Skin Center, Faculty of Medicine, Srinakharinwirot University, Bangkok, Thailand

** Department of Microbiology, Faculty of Medicine, Srinakharinwirot University, Bangkok, Thailand

*** Department of Preventive Medicine, Faculty of Medicine, Srinakharinwirot University, Bangkok, Thailand

Background: An increase in *Staphylococcus aureus* skin colonization in atopic dermatitis patients resulted from the reduction of cathelicidin production in these patients. Recently, an in vivo study demonstrated that vitamin D could stimulate cathelicidin production. Oral supplements of vitamin D might be beneficial in atopic dermatitis.

Objective: To determine the effects of oral vitamin D supplements on clinical impact including *Staphylococcus aureus* skin colonization evaluation in atopic dermatitis patients.

Material and Method: Twenty-four atopic dermatitis patients were included in this double-blind, placebo-controlled study. They were randomly assigned into 2 groups for oral 2,000 IU/day of vitamin D₂ supplement and placebo. The lesional swab culture for *S. aureus* was done at week 0, 2 and 4. Clinical outcomes were assessed by SCORAD score, mexameter for erythema index and konometer for conductance were done at week 0, 2 and 4. Serum vitamin D levels were also determined at week 0 and 4.

Results: Twenty patients completed the protocol. *S. aureus* skin colonization, SCORAD score and erythema index were significantly reduced from baseline to week 4 for vitamin D treated group comparing with placebo ($p = 0.022$, 0.028 and 0.014 , respectively). There was an inverse correlation between serum vitamin D levels with *S. aureus* skin colonization and SCORAD score ($r = -1.0$, $p < 0.001$).

Conclusion: Oral vitamin D supplement could reduce skin colonization of *S. aureus* and demonstrated the clinical improvement of patients with atopic dermatitis.

Keywords: Atopic dermatitis, Vitamin D supplementation, Cathelicidin, *S. aureus* colonization, SCORAD score

J Med Assoc Thai 2015; 98 (Suppl. 9): S23-S30

Full text. e-Journal: <http://www.jmatonline.com>

Currently there are two upcoming interests concerning the pathogenesis of atopic dermatitis (AD)⁽¹⁾. The first is the dysfunction of skin barrier⁽²⁾. The second is the immune dysregulation process which leads to the depletion of an innate immune response such as antimicrobial peptide molecules^(3,4). Reduction of cathelicidin, one of the innate immune antimicrobial peptide, increases in *Staphylococcus aureus* skin colonization^(3,5). It was reported that 90% of bacterial flora in AD patients were found with this organism⁽⁶⁾. Therefore, an increase in *S. aureus* skin colonization's

the one of the most important aggravating factors of this disease⁽⁷⁾.

Kedzierska et al found that colonization density of *S. aureus* was greater at lesional AD skin than non-lesional area with a statistically significant difference and directly correlated with disease severity⁽⁸⁾. Gong et al reported that the lesional skin was commonly found with bacterial colonization especially *S. aureus*⁽⁹⁾.

Few studies reported that cathelicidin as the main target of vitamin D in this skin disorder^(10,11) increased in lesional skin of moderate to severe AD patients who received oral vitamin D supplement^(12,13). Recently, a few studies have showed that an oral vitamin D supplement could improve the clinical response of AD patients⁽¹⁴⁻¹⁶⁾. However, there was no previous study demonstrating the effect of vitamin D

Correspondence to:

Kamanamool N, Department of Preventive Medicine, Faculty of Medicine, Srinakharinwirot University, Bangkok 10110, Thailand.

Phone: +66-2-2594260

E-mail: pangrum109@gmail.com

on skin colonization with *S. aureus* and on objective measurements in AD patients.

The objective of this study was to evaluate the effect of oral vitamin D supplement on clinical outcome in AD patients and *S. aureus* skin colonization.

Material and Method

Patients and methods

The aim of this study is to compare the efficacy of vitamin D treated group with a control group in atopic dermatitis patients. Paired t-test was used to compare the outcome of interest and the decrease in SCORAD. From data obtained from the Javanbakht MH study⁽¹⁵⁾, the mean difference of the decrease in SCORAD between vitamin D treated group and the control group was 2.4 with the standard deviation of difference in the response of 1.8. The sample size of 20 patients is required with 95% confidence and 80% power. Sample size is calculated using the PS (Power and Sample Size Calculation) program version 3.0.34. With a dropout rate of 20%, samples of 24 patients have to be recruited, 12 in each group. A randomized, double-blind, placebo-controlled trial was conducted. There were 24 patients enrolled into the authors' study. Eligible criteria included patients with mild to moderate atopic dermatitis assessed by SCORAD score (Scoring for Atopic Dermatitis)^(1,17,18), age between 1-18 years old. All patients were advised to take normal, dietary products and pursue their normal activities. They had been advised to discontinue at least 2 weeks topical corticosteroid or topical calcineurin inhibitor applications. The exclusion criteria included, patients with coexisting skin infection on top of AD lesions, known case of primary or secondary immune-compromised host, hepatic or renal disease, oral taking of vitamin D or nutraceutical supplement, continually taking antibiotics, corticosteroids, immunosuppressive agents, anti-epileptic drugs, thiazide diuretics, proton-pump inhibitors or histamine 2-receptor antagonists. Patients who used topical antiseptic or antibiotic products during the study period were also excluded.

All patients and their parents completed an informed consent. The clinical severity was assessed by using the SCORAD score, the objective skin evaluation for erythema by Mexameter (Cologne, Germany) and skin conductance by corneometer measurement (Cologne, Germany). The photographs of the skin lesions were taken by digital camera for pre- and post-treatment at week 0, 2 and 4. All of these measurements were done by dermatologist.

S. aureus culture was done from the skin

lesions at week 0, 2, and 4. Quantitative method to determine total number of *S. aureus* were measured and reported with colony forming units per 1 ml (CFU/ml).

Serum 25-hydroxy vitamin D level (25 (OH) vitamin D) was measured at week 0 and 4. It was tested by chemiluminescence technique at Biochemistry Laboratory Center, Ramathibodi Hospital, Mahidol University, Bangkok, Thailand.

All patients were assigned by computerized blocked randomization (block of 4) method, which was performed by a third person and the encoding was disclosed at the end of the study. Twenty-four subjects were randomly assigned into two groups to receive either 2,000 IUs/day of vitamin D solution or placebo. The dose of 2,000 IUs/day of oral vitamin D₂ supplement used in the study is safe because it is less than the tolerable upper intake level of vitamin D supplement. It is unlikely to cause adverse health effects and is recommended by Food and Nutrition Board at the Institute of Medicine of the National Academies of United State (FNB)⁽¹⁹⁾. The tolerable upper level dose is ranged from 2,500 IUs/day for 1-3 years old, to 4,000 IUs/day for >9 years old. All patients were allowed to take oral antihistamine (2 mg/kg/day, hydroxyzine) if clinically indicated, and providing them with the similar formulation of sunscreen, skin moisturizer and mild gentle cleanser (Physiogel®, Stiefel Laboratory, USA). This research was approved by Ethics Committee for Human Research, Faculty of Medicine, Srinakharinwirot University.

Statistical analysis

The demographic data was reported with proportion and percentage. Regarding the analysis of continuous data, the mean and standard deviation for normal distribution and Wilcoxon Signed Ranks test with data for non-normal distribution were used. Mann-Whitney U test was used to determine and compare between each variable. Spearman's rho test was used to demonstrate the correlation between 25 (OH) vitamin D level with *S. aureus* colonization and SCORAD scores. Statistical significance was reported if *p* less than 0.05. Data software with SPSS program version 19.0 was used to analyze data in this study.

Results

Twenty patients completed the protocol, three of them were lost to follow-up and one patient was excluded due to secondary bacterial skin infection. Mean age was 8.28 years old (range = 1-18 years old).

There were 11 cases with mild AD and 9 cases with moderate AD. Baseline characteristics between groups had no statistically significant difference in term of gender, age, SCORAD score, *S. aureus* colonization, erythema index and skin conductance ($p = 0.64, 0.83, 0.83, 0.95, 0.95$ and 0.08 respectively) (Table 1).

There was a decrease in SCORAD score from baseline to week 2 and 4 in both groups. At week 4, the decreased score of vitamin D group was statistically significantly difference from the placebo ($p = 0.02$) (Fig. 1). It was shown that the vitamin D-treated group had lower colony count of *S. aureus* than those taking placebos at week 4 with statistically significant differences ($p = 0.03$) (Fig. 2).

Erythema index was decreased. At week 4, it was found that the erythema index of those with vitamin D supplement was statistically significant lower than those with placebo ($p = 0.01$) (Fig. 3). Skin conductance was not statistically significant difference between groups at each visit ($p = 0.08, 0.65$ and 0.55 respectively) (Fig. 4).

Concerning the SCORAD score, there was a statistical significant group difference for erythema, edema, excoriation and pruritus at week 4; however, there was not statistically significant difference for skin

lichenification, dryness and sleeplessness (Fig. 5).

Eight patients (4 for each group) were measured for serum 25(OH) vitamin D level in the serum. At baseline, the average mean of 25 (OH) vitamin D level of vitamin D treated group and placebo group was 18.55 ± 2.68 and 17.02 ± 2.66 ng/ml, respectively, which were lower than normal value (cut off point of normal value was 20 ng/ml). There was no significant group difference in vitamin D level at baseline ($p = 0.45$). At the beginning of the study, the 25 (OH) vitamin D level of the vitamin D treated group was range from 13.70-20.20 ng/ml (the mean was 17.03 ng/ml), by the end of week 4, it was range from 24.40-27.60 ng/ml (the

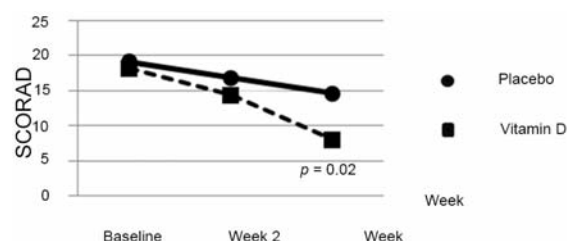


Fig. 1 To demonstrate the difference of SCORAD score between vitamin D and placebo group.

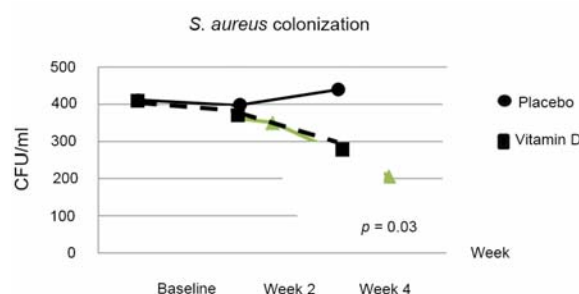


Fig. 2 To demonstrate *S. aureus* colonization between vitamin D and placebo group.

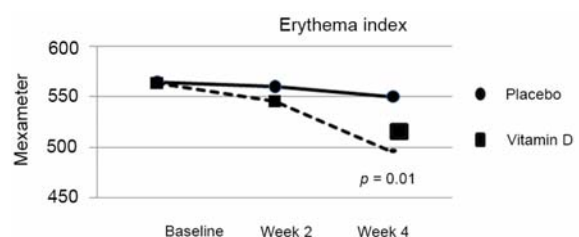


Fig. 3 To demonstrate erythema index between vitamin D and placebo group.

Table 1. Demographic data with SCORAD score and *S. aureus* colonization, erythema index and conductance at baseline

Variables	Vitamin D (n = 10)	Placebo (n = 10)	p-value
Gender (male: female)	4:6	3:7	0.64
Age (year) (mean, SD)	8.59 (5.93)	7.90 (6.95)	0.83
SCORAD score (mean, SD)	18.23 (8.89)	19.21 (10.61)	0.83
<i>S. aureus</i> colonization (CFU/ml) (mean, SD)	387.50 (246.22)	395.50 (276.91)	0.95
Erythema index (mean, SD)	563.40 (31.57)	564.40 (35.31)	0.95
Skin conductance (mean, SD)	18.49 (9.05)	26.77 (10.68)	0.08

SD = standard deviation

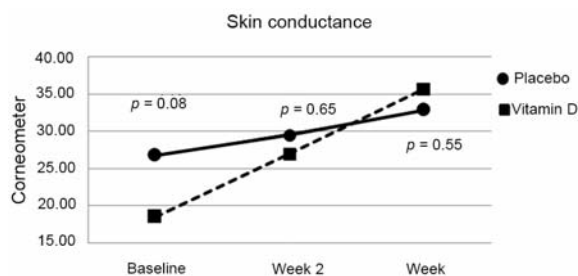


Fig. 4 To demonstrate skin conductance between vitamin D and placebo.

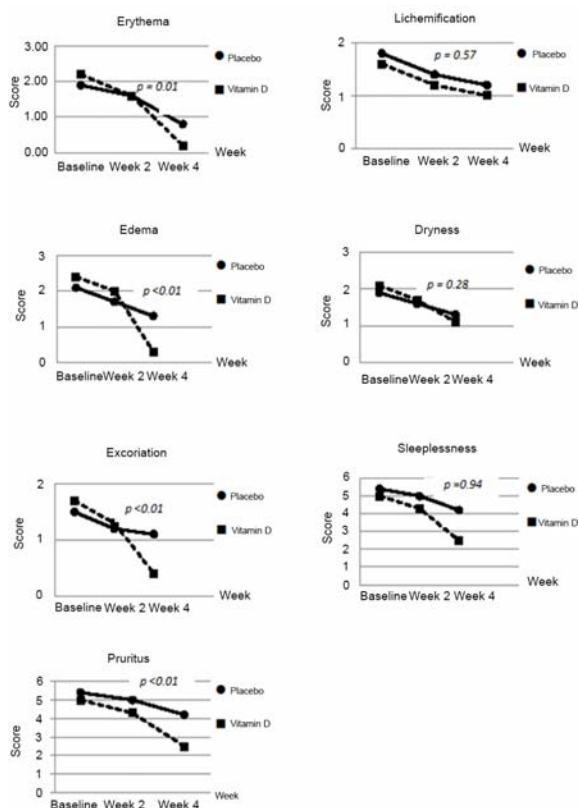


Fig. 5 To demonstrate the difference of sub-SCORAD score between vitamin D and placebo group.

mean was 25.06 ng/ml). The increment of 25 (OH) vitamin D level of the vitamin D treated group was significantly different from the placebo group, 8.03 ng/ml vs. 0.75 ng/ml ($p < 0.01$). The correlation between serum vitamin D level of the patients with SCORAD score and *S. aureus* colonization were analyzed. It was discovered that the serum vitamin D level was inversely correlated with SCORAD score and *S. aureus* colonization with statistically significant differences ($r = -1.0$, $p < 0.01$). No adverse effects were reported during the study

period.

Discussion

Atopic dermatitis is a common chronic relapsing dermatitis with a complex of etiology. The standard management composes of anti-inflammatory agents, moisturizer and avoidance of aggravating factors. The role of vitamin D in the pathogenesis and treatment of AD was first elucidated by Schaubert et al who demonstrated that vitamin D induced toll-like receptor 2 recognition of keratinocytes to microbes and led to cathelicidin upregulation⁽²⁰⁾. Later, the study of Hata et al included patients with moderate to severe AD and volunteers with normal skin. All of them were orally given 4,000 IUs/day of vitamin D for three weeks. Then cathelicidin expression from the patients' skin was done on week 0 and week 3. It was found that patients with AD had statistically significant increase in cathelicidin expression when compared with the baseline. Furthermore, volunteers with normal skin also had modest increased expressions of cathelicidin. The researchers suggested that oral vitamin D supplement could significantly induce cathelicidin expression in the skin of AD patients⁽¹²⁾. Then the work of Peroni et al suggested that 25 (OH) vitamin D level of AD patients was inversely correlated with disease severity⁽¹³⁾.

Recently, three studies demonstrated the beneficial effect of oral vitamin D supplement on the clinical manifestation of AD. Sidbury et al firstly conducted a clinical study of eleven AD patients with oral 1,000 IUs/day of vitamin D supplement⁽¹⁶⁾. They reported 80% improvement of IGA (Investigator's Global Assessment); however, there was no significant difference for EASI (Eczema area and severity index scale)⁽¹⁶⁾. The second study of Javanbakht et al was done to evaluate the effect of vitamin D and vitamin E supplements on the clinical outcome. The clinical result of SCORAD score with 34.80% improvement in vitamin D treated group was detected after 60 days of treatment⁽¹⁵⁾. The third study by Amestajani et al also demonstrated 38.30% improvement of SCORAD score with 1,600 IUs/day of oral vitamin D supplement⁽¹⁴⁾. Moreover, the authors suggested that further studies with different doses of vitamin D would be better suited for approving this important issue⁽¹⁴⁾. Since the dose of vitamin D used in all previous clinical studies was range from 1,000-1,600 IUs/day. Therefore, this study designed with higher doses of vitamin D (2,000 IUs/day) which was safe and lower than the tolerable upper intake level recommended by FNB⁽¹⁹⁾.

The results of this study showed the highest

Table 2. Comparison of this study with the others

	Sidbury et al ⁽¹⁶⁾	Javanbakht et al ⁽¹⁵⁾	Amestejani et al ⁽¹⁴⁾	This study
Number	11	45	60	20
Age (year)	2-13 (median, 7)	13-45 (mean, 25.95)	mean = 23.34 (SD = 2.10)	1-18 (mean, 8.28)
Severity of disease	Mild (EASI, range; 10 to 18.60)	Mild to severe (Mean SCORAD = 34.20)	Mild to severe (Mean SCORAD = 24.80)	Mild to moderate (Mean SCORAD = 19.50)
Dose of vitamin (D/day)	1,000 IU	1,600 IU	1,600 IU	2,000 IU
Average follow-up (days)	28	60	60	28
Clinical outcome:				
Subjective assessment				
SCORAD		34.8% improvement ($p < 0.01$)	38.3% improvement ($p = 0.01$)	55.8% improvement ($p = 0.02$)
EASI	EASI score ($p = 0.40$)			
TIS			45.7% improvement ($p = 0.03$)	
IGA	80% improvement ($p = 0.04$)			
Objective assessment:				
Mexameter				12.2% improvement ($p = 0.01$)
Corneometer				48.2% improvement ($p = 0.08$)
Laboratory assessment:				
<i>S. aureus</i> skin colonization				46% reduction ($p = 0.03$)

SCORAD = scoring for atopic dermatitis; EASI = eczema area and severity index scale; IGA = investigators' global assessment; TIS = three item severity score

improvement of SCORAD score (58%) compared with the other studies (Table 2). Nevertheless, this study can considerably compare only with Sidbury et al study because of the same age group of patients (pediatrics) and the same range of severity of atopic dermatitis (mild to moderate). Taken all together, the results implied that a higher dose of vitamin D usage might yield a higher clinical response. Regarding outcome assessment, not only the SCORAD score was used in our study, the objective measurements such as erythema index and skin conductance that represented the skin inflammation and moisturizing levels respectively were used. It was found that erythema index also improved significantly. However, the lichenification, dryness, sleeplessness and skin conductance score were not significant different from placebo. As a result, vitamin D had no effect on skin moisturizing property.

Regarding the role of vitamin D on the pathogenesis of AD, it was hypothesized that an oral

vitamin D supplement increased cathelicidin expression on AD patient skin, which in turn could inhibit the growth of *S. aureus*. This study demonstrated that oral vitamin D supplement could cause statistically significant reduction of *S. aureus* skin colonization in AD patients and lead to the improvement of subjective and objective clinical outcomes. Finally, it also determined the inverse correlation between serum 25 (OH) vitamin D level and *S. aureus* colonization.

Concerning the side effects of oral vitamin D supplement, the FNB suggested that serum 25 (OH) vitamin D level above 50 ng/ml should be avoided. Since it could be associated with an increase in side effects such as cancer in some areas like the pancreas, greater risk of cardiovascular events, and more fractures in the elderly. It was found from the researches that vitamin D intakes of 5,000 IUs/day achieved serum 25 (OH) vitamin D levels between 40-60 ng/ml. Utilizing an uncertainty factor of 20% to this intake dose gave a

4,000 IUs/day, which is the lower FNB recommended dose for younger children aged 9 or older^(13,19). The dose used in the previous studies was ranging from 1,000-1,600 IUs/day and the maximum dose of 4,000 IUs was used to determine the correlation between vitamin D supplement and cathelicidin expression. The dose of 2,000 IUs/day used in the present study was safe because it was lower than the recommended dose and the serum 25 (OH) vitamin D level in this study was in the range of 22.40 to 27.60 ng/ml (mean = 26.53 ng/ml), which was much lower than the critical serum level (40-60 ng/ml).

In conclusion, the results of this study supported that oral daily vitamin D supplement of 2,000 IUs might decrease in *S. aureus* skin colonization and provide clinical improvement especially the inflammatory parameters, but not for a moisturizing effect in mild to moderate pediatric AD patients. However, concerning the side effects of vitamin D supplement, further studies on different doses, not to exceed 2,000 IUs/day in long-term follow-up and an increase in sample size should be conducted.

What is already known on this topic ?

Staphylococcus aureus plays an important role in atopic dermatitis (AD) exacerbation of the eczema owing to a reduction in the cathelicidin production in the patients. Oral vitamin D supplement in the volunteers demonstrated an increase in the cathelicidin production. A few clinical studies shown that vitamin D supplement had beneficial effects on AD patients.

What this study adds ?

The authors' study demonstrated the additional evidence to support that vitamin D supplement in AD patients might improve the clinical severity and objective measurements. It also decreased the *Staphylococcus aureus* skin colonization. Moreover there was an inverse correlation between serum vitamin D levels with *Staphylococcus aureus* skin colonization and SCORAD score.

Acknowledgement

We would like to thank Faculty of Medicine, Srinakharinwirot University, Thailand, for grant support. We also thank to Mrs. Suchada Boon-itt, Western Languages Department, Faculty of Humanities for English structural audition.

Potential conflicts of interest

None.

References

1. Hanifin J, Saurat JH. Understanding atopic dermatitis: pathophysiology and etiology. Introduction. J Am Acad Dermatol 2001; 45 (1 Suppl): S1.
2. Boguniewicz M, Leung DY. Atopic dermatitis: a disease of altered skin barrier and immune dysregulation. Immunol Rev 2011; 242: 233-46.
3. Ballardini N, Johansson C, Lilja G, Lindh M, Linde Y, Scheynius A, et al. Enhanced expression of the antimicrobial peptide LL-37 in lesional skin of adults with atopic eczema. Br J Dermatol 2009; 161: 40-7.
4. Goo J, Ji JH, Jeon H, Kim MJ, Jeon SY, Cho MY, et al. Expression of antimicrobial peptides such as LL-37 and hBD-2 in nonlesional skin of atopic individuals. Pediatr Dermatol 2010; 27: 341-8.
5. Hata TR, Gallo RL. Antimicrobial peptides, skin infections, and atopic dermatitis. Semin Cutan Med Surg 2008; 27: 144-50.
6. Hauser C, Wuethrich B, Matter L, Wilhelm JA, Sonnabend W, Schopfer K. *Staphylococcus aureus* skin colonization in atopic dermatitis patients. Dermatologica 1985; 170: 35-9.
7. Goh CL, Wong JS, Giam YC. Skin colonization of *Staphylococcus aureus* in atopic dermatitis patients seen at the National Skin Centre, Singapore. Int J Dermatol 1997; 36: 653-7.
8. Kedzierska A, Kapinska-Mrowiecka M, Czubak-Macugowska M, Wojcik K, Kedzierska J. Susceptibility testing and resistance phenotype detection in *Staphylococcus aureus* strains isolated from patients with atopic dermatitis, with apparent and recurrent skin colonization. Br J Dermatol 2008; 159: 1290-9.
9. Gong JQ, Lin L, Lin T, Hao F, Zeng FQ, Bi ZG, et al. Skin colonization by *Staphylococcus aureus* in patients with eczema and atopic dermatitis and relevant combined topical therapy: a double-blind multicentre randomized controlled trial. Br J Dermatol 2006; 155: 680-7.
10. Antal AS, Dombrowski Y, Koglin S, Ruzicka T, Schaub J. Impact of vitamin D3 on cutaneous immunity and antimicrobial peptide expression. Dermatoendocrinol 2011; 3: 18-22.
11. Dombrowski Y, Peric M, Koglin S, Ruzicka T, Schaub J. Control of cutaneous antimicrobial peptides by vitamin D3. Arch Dermatol Res 2010; 302: 401-8.
12. Hata TR, Kotol P, Jackson M, Nguyen M, Paik A, Udall D, et al. Administration of oral vitamin D induces cathelicidin production in atopic

- individuals. *J Allergy Clin Immunol* 2008; 122: 829-31.
13. Peroni DG, Piacentini GL, Cametti E, Chinellato I, Boner AL. Correlation between serum 25-hydroxy-vitamin D levels and severity of atopic dermatitis in children. *Br J Dermatol* 2011; 164: 1078-82.
 14. Amestejani M, Salehi BS, Vasigh M, Sobhkhiz A, Karami M, Alinia H, et al. Vitamin D supplementation in the treatment of atopic dermatitis: a clinical trial study. *J Drugs Dermatol* 2012; 11: 327-30.
 15. Javanbakht MH, Keshavarz SA, Djalali M, Siassi F, Eshraghian MR, Firooz A, et al. Randomized controlled trial using vitamins E and D supplementation in atopic dermatitis. *J Dermatolog Treat* 2011; 22: 144-50.
 16. Sidbury R, Sullivan AF, Thadhani RI, Camargo CA Jr. Randomized controlled trial of vitamin D supplementation for winter-related atopic dermatitis in Boston: a pilot study. *Br J Dermatol* 2008; 159: 245-7.
 17. Oranje AP. Practical issues on interpretation of scoring atopic dermatitis: SCORAD Index, objective SCORAD, patient-oriented SCORAD and Three-Item Severity score. *Curr Probl Dermatol* 2011; 41: 149-55.
 18. Gu H, Chen XS, Chen K, Yan Y, Jing H, Chen XQ, et al. Evaluation of diagnostic criteria for atopic dermatitis: validity of the criteria of Williams et al. in a hospital-based setting. *Br J Dermatol* 2001; 145: 428-33.
 19. Ross AC, Manson JE, Abrams SA, Aloia JF, Brannon PM, Clinton SK, et al. The 2011 report on dietary reference intakes for calcium and vitamin D from the Institute of Medicine: what clinicians need to know. *J Clin Endocrinol Metab* 2011; 96: 53-8.
 20. Schaubert J, Gallo RL. The vitamin D pathway: a new target for control of the skin's immune response? *Exp Dermatol* 2008; 17: 633-9.

การศึกษาผลของการให้วิตามินดีชนิดรับประทานในผู้ป่วยผื่นภูมิแพ้ผิวหนัง: ผลต่ออาการทางคลินิกและ *Staphylococcus aureus* colonization

มนตรี อุดมเพทยกุล, สรรเสริญ หัวใจ, เทพ เกลิมชัย, มาลัย ทวีโชคภัทร์, นันทิชา คมนามูล

ภูมิหลัง: การเพิ่มของ *Staphylococcus aureus* colonization เป็นปัจจัยหนึ่งในพยาธิกำเนิดของโรค atopic dermatitis โดยพบว่ามีสาเหตุจากการสร้าง cathelicidin ลดลงมีงานวิจัยพบว่าวิตามินดีกระตุ้นการสร้าง cathelicidin ได้ดังนั้นการให้รับประทานวิตามินดีอาจจะช่วยให้อาการของ atopic dermatitis ดีขึ้น

วัตถุประสงค์: เพื่อศึกษาผลของการรับประทานวิตามินดีต่ออาการทางคลินิกรวมทั้ง *S. aureus* colonization ในคนไข้ atopic dermatitis

วัสดุและวิธีการ: เป็นการศึกษาแบบสุ่มที่ต้องควบคุมและปกปิดทั้งสองฝ่าย ผู้ป่วยได้รับวิตามินดีรับประทานวันละ 2,000 IU หรือยาหลอก ทำการส่งเพาะเชื้อที่ผื่น ประเมินอาการทางคลินิกโดยใช้คะแนน SCORAD วัดความแดงของผื่น (erythema index) โดยเครื่อง mexameter วัดความชื้นของผิวโดยค่าการนำไฟฟ้าจากเครื่อง comeometer ที่สัปดาห์ 0, 2 และ 4 ได้เจาะเลือดผู้ป่วยส่งวัดระดับของวิตามินดีที่สัปดาห์ 0, 2 และ 4

ผลการศึกษา: เมื่อสิ้นสุดการศึกษาพบว่า *S. aureus* colonization, คะแนน SCORAD ค่า erythema index ลดลงอย่างมีนัยสำคัญทางสถิติในกลุ่มที่ได้รับวิตามินดีเทียบกับยาหลอก ($p = 0.022, 0.028$ และ 0.014 ตามลำดับ) และนอกจากนั้นพบความสัมพันธ์ระหว่างระดับวิตามินดีในกระแสเลือดกับอาการทางคลินิกและค่าคะแนน SCORAD ($r = -0.1, p < 0.001$)

สรุป: วิตามินดีชนิดรับประทานสามารถลด *S. aureus* colonization และช่วยให้อาการทางคลินิกของผู้ป่วย atopic dermatitis ดีขึ้น
