# Chronic Nasolabial Fold Seborrheic Dermatitis Successfully Controlled With Crisaborole

Dede Liu MD,[a] Peter Chow BS,[b] Sarah Strawn MD,[a] Anand Rajpara MD,[a] Ting Wang MD,[a] and Daniel Aires MD JD[a]

[a]University of Kansas Medical Center, Kansas City, KS
[b]University of Kansas School of Medicine, Kansas City, KS

Seborrheic dermatitis is a chronic inflammatory dermatosis characterized by redness and scaling and is typically seen in the brows, nasolabial folds, and chest.[1] Topical antifungals such as ketoconazole can be helpful, but resistant seborrheic dermatitis often requires topical steroids or steroid sparing agents such as tacrolimus.[2] The exact cause and pathogenesis of seborrheic dermatitis is not known. However, inflammation is known to play a role. The disease has a preference for body sites with increased or larger sebaceous glands (ie, face, scalp). Sebaceous glands create an environment favorable for growth of lipid dependent fungi such as *Malassezia*. Multiple seborrheic dermatitis treatment studies have demonstrated a connection with *Malassezia*, but mechanisms remain elusive.[6] Because it is unclear that seborrheic dermatitis patients have higher *Malassezia* counts than controls, an alternative explanation posits that anti-fungals exert a local anti-inflammatory effect. [7]

Seborrheic dermatitis might result from a host's immune response to *Malassezia* or its by-products such as lipase, free fatty acids, or reactive oxygen species.[8] *Malassezia* or its byproducts have been hypothesized to induce inflammatory cytokine production in keratinocytes.[10] A factor that can cause localized inflammation is the lipase activity of *Malassezia*, which can generate fatty acids from skin lipids.[9] Additionally, seborrheic dermatitis skin biopsies demonstrate upregulation of inflammatory interleukins, natural killer cells, as well as complement activation.[8]

Phosphodiesterase 4 (PDE4) is involved in the regulation of pro-inflammatory cytokines through its degradation of cAMP. PDE4 activity has been previously discovered to be increased in the inflammatory cells of atopic dermatitis and thus targeting and inhibiting PDE4 reduces the local production of inflammatory mediators.[5] This has led to the creation of Crisaborole 2% ointment, a novel PDE4 inhibitor that reduces local inflammation, and is newly approved for treatment of atopic dermatitis.[3,4]

We hypothesized that Crisaborole may show utility in seborrheic dermatitis via reducing the inflammatory component. We report a case of chronic bilateral nasolabial fold seborrheic dermatitis in a 50-year-old man who was previously treating it with over-the-counter CeraVe moisturizer BID. During the treatment period CeraVe was continued BID to both nasolabial folds,

**FIGURE 1.** Seborrheic dermatitis of left nasolabial fold treated with over-the-counter CeraVe moisturizer BID for 4 weeks without treatment of Crisaborole.

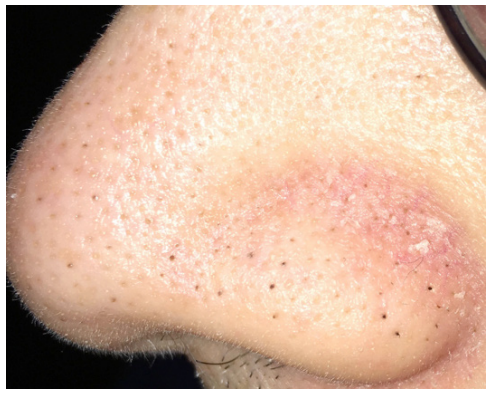
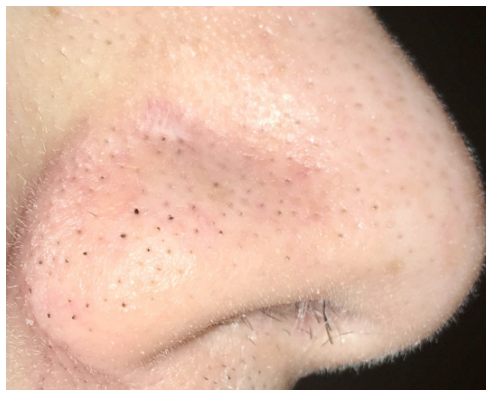


**FIGURE 2.** Seborrheic dermatitis of right nasolabial fold treated with 4 weeks of twice per week application of Crisaborole, along with over-the-counter CeraVe moisturizer BID.



while Crisaborole was applied twice per week to the right nasolabial fold only. After four weeks the scale and erythema was notably reduced on the right nasolabial fold treated with Crisaborole (Figure 2) versus the untreated left (Figure 1) nasolabial fold. Larger trials will be needed to replicate this.

## DISCLOSURES

The authors have no conflicts of interest to declare.

## REFERENCES

1. Reider N, Fritsch P. Other Eczematous Eruptions. In: Bolognia J, Schaffer J, Cerroni L, eds. Dermatology. 4th ed. Philadelphia: Elsevier. 2017;1:228-241.
2. Gupta AK, Versteeg SG. Topical Treatment of Facial Seborrheic Dermatitis: A Systematic Review. *Am J Clin Dermatol.* 2017;18:193.
3. Stein Gold LF, Spelman L, Spellman MC, et al. A Phase 2, Randomized, Controlled, Dose-Ranging Study Evaluating Crisaborole Topical Ointment, 0.5% and 2% in Adolescents With Mild to Moderate Atopic Dermatitis. *J Drugs Dermatol.* 2015;14:1394.
4. Murrell DF, Gebauer K, Spelman L, Zane LT. Crisaborole Topical Ointment, 2% in Adults With Atopic Dermatitis: A Phase 2a, Vehicle-Controlled, Proof-of-Concept Study. *J Drugs Dermatol.* 2015;14:1108.
5. Zebda R, Paller AS. Phosphodiesterase 4 (PDE4) Inhibitors. *J Am Acad Dermatol.* https://www.ncbi.nlm.nih.gov/pubmed/29248522. Accessed December 19, 2017.
6. Valia R. Etiopathogenesis of seborrheic dermatitis. *Indian J Dermatol Venereol Leprol.* 2006;72(4):253.
7. McGinley KJ, Leyden JJ, Marples RR, Kligman AM. Quantitative microbiology of the scalp in non-dandruff, dandruff, and seborrheic dermatitis. *J Invest Dermatol.* https://www.ncbi.nlm.nih.gov/pubmed/237965?dopt=Abstract. Published June 1975. Accessed December 19, 2017.
8. Faergemann J, Bergbrant I-M, Dohsé M, Scott A, Westgate G. Seborrhoeic dermatitis andPityrosporum(Malassezia) folliculitis: characterization of inflammatory cells and mediators in the skin by immunohistochemistry. *Br J Dermatol.* 2001;144(3):549-556.
9. Bergbrant IM. Seborrhoeic dermatitis and Pityrosporum yeasts. *Curr Top Med Mycol.* https://www.ncbi.nlm.nih.gov/pubmed/8724243?dopt=Abstract. Accessed December 19, 2017.
10. Watanabe S, Kano R, Sato H, Nakamura Y, Hasegawa A. The effects of Malassezia yeasts on cytokine production by human keratinocytes. *J Invest Dermatol.* https://www.ncbi.nlm.nih.gov/pubmed/11348468. Published May 2001. Accessed December 19, 2017.

## AUTHOR CORRESPONDENCE

**Daniel Aires MD JD**
E-mail:..... daires@kumc.edu.