

## Review Article

# Crisaborole in Dermatology

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## ABSTRACT

Phosphodiesterase 4 (PDE4) is crucial in inflammatory skin conditions, increased activity leads to elevated proinflammatory cytokine, contributing to disease pathology like atopic dermatitis (AD). Crisaborole, a topical PDE4 inhibitor, with favorable pharmacokinetics, good skin penetration, minimal systemic absorption, effectively reduces proinflammatory mediators and reactive oxygen species. The ointment is applied topically twice daily and is well-tolerated with minor side effects like application site pain, which typically resolves quickly. In clinical trials, crisaborole has shown significant efficacy in treating AD, chronic plaque psoriasis and vitiligo. At present, the mainstay for management of chronic inflammatory skin diseases is topical corticosteroids and calcineurin inhibitors, but their long-term use is associated with adverse effects such as skin atrophy, telangiectasia, hypertrichosis, hypothalamo-pituitary axis suppression and increased risk of skin cancers, respectively. Crisaborole offers a safer alternative with fewer side effects. Despite promising results, further studies are necessary to expand its indications, particularly for other inflammatory skin diseases such as psoriasis and vitiligo, where the research is limited.

**Keywords:** Atopic dermatitis, Crisaborole, Phosphodiesterase inhibitor, Psoriasis, Vitiligo

## INTRODUCTION

Crisaborole is a benzoxaborole compound with boron as a central atom, due to this central boron, it has the ability to bind bimetal centre of phosphodiesterase 4 (PDE4) enzyme.<sup>[1]</sup> PDE4 is the primarily expressed in immune cells and keratinocytes.<sup>[2]</sup> It is the key regulator of inflammatory mediator production and acts by converting intracellular cyclic adenosine monophosphate (cAMP) to 5' adenosine monophosphate which favours proinflammatory mediators production. In various inflammatory skin conditions, its activity is increased, thereby leading to increased production of proinflammatory cytokines including interleukins (IL) such as IL-4, 13 and 31. Crisaborole is a topical selective PDE4 inhibitor, due to its low molecular weight and favourable skin penetration. Further, it also reduces reactive oxygen species generation which has role in inflammatory skin conditions.<sup>[3,4]</sup>

Refer Table 1 for the pharmacodynamics and pharmacokinetics of crisaborole.<sup>[5-12]</sup>

Figures 1-3 demonstrate the structure of crisaborole and its mechanism of action.

## USES IN DERMATOLOGY

### Atopic dermatitis (AD)

Crisaborole is Food and Drug Administration (FDA) approved for use in patients with AD above 2 years of age. Results from multiple clinical trials have established the effectiveness of crisaborole

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Parameters	Details
Chemical structure	Crisaborole is r 4-((1-Hydroxy-1,3-dihydrobenzo[c] [1,2]oxaborol-5-yl) oxy) benzonitrile (C <sub>14</sub> H <sub>10</sub> BNO <sub>3</sub> ). <sup>[5]</sup> It is a compound made of boron heterocyclic scaffold with wide medical use due to its chemical properties. <sup>[6]</sup> Shown in Figure 1.
Molecular weight	251 Daltons. Thus good skin penetration. <sup>[6]</sup>
Maximum blood concentration (C <sub>max</sub> )	105–111 ng/mL <sup>[4,6]</sup>
Time to Reach C <sub>max</sub> (T <sub>max</sub> )	2.17–3 h <sup>[4,6]</sup>
Steady-state concentration	4–6 days <sup>[6]</sup>
Half-life (t <sub>1/2</sub> )	7.17–11.9 h <sup>[6]</sup>
Distribution	97% plasma protein-bound in the bloodstream. <sup>[6]</sup>
Metabolism	Metabolised into two major metabolites: 1. 5-(4-cyanophenoxy)-2-hydroxybenzyl alcohol (Metabolite 1) through hydrolysis. 2. 5-(4-cyanophenoxy)-2-hydroxybenzoic acid (Metabolite 2) through oxidation. <sup>[7]</sup>
Excretion	Primarily eliminated via renal excretion of metabolites. <sup>[6]</sup>
Mechanism of action	PDE4 is a key enzyme regulating intracellular homeostasis. It is highly expressed in keratinocytes and immune cells, <sup>[2]</sup> and its expression increases in inflammatory skin conditions. <sup>[8]</sup> PDE4 hydrolyses AMP to 5'-AMP, activating NF-κB, leading to the production of proinflammatory cytokines (IL-2, IL-4, IL-12, IL-22, TNF-α and Interferon-gamma). <sup>[9]</sup> Crisaborole is a low molecular weight PDE4 inhibitor with good skin penetration, <sup>[10,11]</sup> that binds selectively to the bimetallic centre of PDE4, replacing boron with carbon or oxygen, thus inhibiting its function. <sup>[12]</sup> This inhibition prevents cAMP degradation, activating protein kinase A, which downregulates NF-κB and NFAT, reducing proinflammatory mediators, ROS and increasing anti-inflammatory cytokines. <sup>[2]</sup> [Figures 2 and 3].
Formulation/ composition	2% white-to-off-white ointment containing petrolatum, propylene glycol, mono- and diglycerides, paraffin, butylated hydroxytoluene and edetate calcium disodium. <sup>[6]</sup>
Usage	Approved in 2016 for atopic dermatitis in children ≥2 years. Applied twice daily as a thin layer. Available only for topical use, not approved for oral, ophthalmic or intravaginal routes. <sup>[6]</sup>
Storage	Stored at 20–25°C. <sup>[6]</sup>
Dose adjustment	No dose adjustments required for hepatic or renal impairment. <sup>[6]</sup>
Pregnancy category	Safety during pregnancy and lactation is unknown. <sup>[6]</sup>
Drug-drug interaction	<i>In vitro</i> studies suggest that crisaborole metabolites are weak inhibitors of CYP 450 1A2, 2B6, 2C8 and 2C9. Due to limited systemic absorption, the likelihood of drug interactions is low. <sup>[6]</sup>

cAMP: Cyclic adenosine monophosphate, AMP: Adenosine monophosphate, PDE4: Phosphodiesterase 4, NF-κB: Nuclear factor kappa B, ROS: Reactive oxygen species, IL: Interleukin, EASI: Eczema area and severity index, POEM: Patient-oriented eczema measure, AD: Atopic dermatitis, ADSI: Atopic dermatitis severity index, TEAE: Treatment-emergent adverse events, NFAT: Nuclear Factor of activated T-cells, CYP: Cytochrome P450

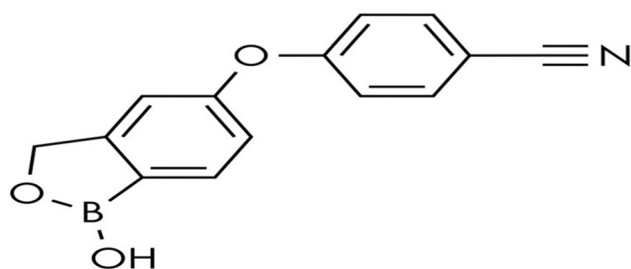
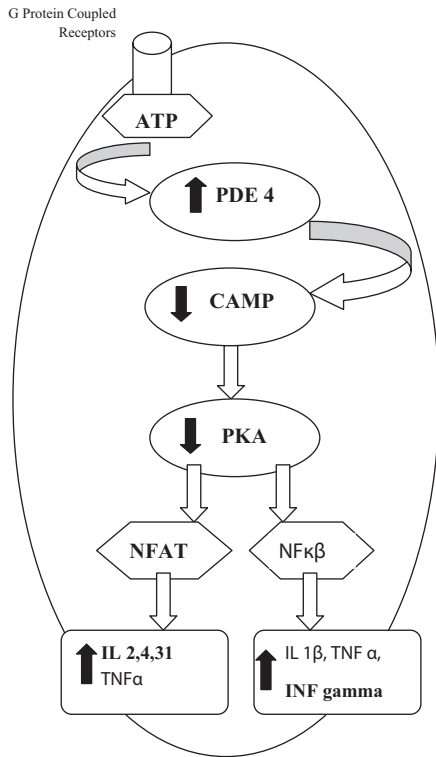


Figure 1: Structure of crisaborole.

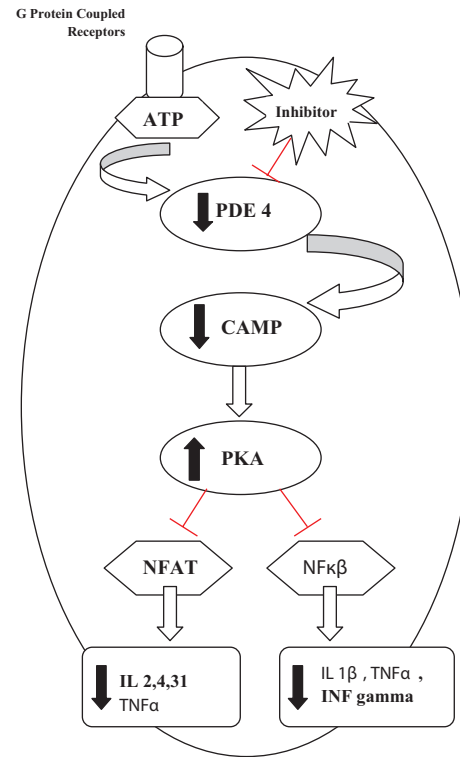
in AD, with 31–47% patients treated successfully, while 49–65% patients achieved investigator's static global assessment (ISGA) score of almost clear to clear.<sup>[13]</sup> Further, significantly improved sleep outcome and quality of life were noted in patients as well as their parents who received crisaborole for AD.<sup>[6]</sup> Topical steroid and calcineurin inhibitors – the first line of agents are associated with adverse effects such as telangiectasia, skin atrophy, striae and risk of hypothalamo-pituitary axis suppression on prolonged use with former and burning, stinging and a black box warning of increased risk of



**Figure 2:** Role of phosphodiesterase 4 in inflammatory skin conditions. ATP: Adenosine triphosphate, PDE: Phosphodiesterase, cAMP: Cyclic adenosine monophosphate, PKA: Protein kinase A, NFAT: Nuclear factor of activated T cells, NF-κB: Nuclear factor kappa B, TNF-α: Tumour necrosis factor-alpha.

malignancy on prolonged use with later.<sup>[14]</sup> Crisaborole makes an appealing option for management for mild-moderate AD due to fewer side effects, limited systemic absorption and long-term safety on continuous use; drawback is high cost and limited available studies. Further crisaborole was compared with 1% pimecrolimus and 0.1% tacrolimus in patients aged  $\geq 2$  years with mild-to-moderate AD using a matching-adjusted indirect comparison. Data from two phase III randomised controlled trials (RCTs) of crisaborole were reweighted against published RCTs of calcineurin inhibitors. Results showed higher odds of ISGA improvement with crisaborole compared to pimecrolimus (odds ratio [OR] = 2.03) and tacrolimus 0.03% (OR= 1.50).<sup>[15]</sup>

Crisaborole in children aged <2 years: A phase IV open-label study (CrisADe CARE 1) assessed the safety, effectiveness and pharmacokinetics of crisaborole 2% in 137 infants aged 3–<24 months with mild-to-moderate AD. Crisaborole was applied twice daily for 28 days, with 21 infants in a pharmacokinetic cohort. Treatment-emergent adverse events occurred in 64.2%, mostly mild/moderate and treatment-related in 16.1%, the most common being application site pain, discomfort and erythema. By day 29, 30.2% achieved ISGA clear/almost clear with  $\geq 2$ -grade improvement, alongside



**Figure 3:** Mechanism of action – Phosphodiesterase 4 inhibitor increases cyclic adenosine monophosphate levels, thereby increasing protein kinase A activity which inhibits nuclear factor-kappa beta and nuclear factor of activated T cells which, in turn, inhibit the production of proinflammatory cytokines such as Interleukin 1 beta, 2, 4, 23, 26 31, tumour necrosis factor-alpha and interferon gamma thus help reduce inflammation in chronic inflammatory skin condition. ATP: Adenosine triphosphate, PDE: Phosphodiesterase, cAMP: Cyclic adenosine monophosphate, PKA: Protein kinase A, NFAT: Nuclear factor of activated T cells, NF-κB: Nuclear factor kappa B, TNF-α: Tumour necrosis factor-alpha.

significant reductions in Eczema area and severity index (EASI) (–57.5%) and Patient-oriented eczema measure (POEM) scores (–8.5). Pharmacokinetic analysis showed systemic exposure in infants was comparable to that in children  $\geq 2$  years. Overall, crisaborole was well tolerated, safe and effective in this age group.<sup>[16]</sup> Table 2 further summarizes major finding in various trials showing role crisaborole in AD.<sup>[17,18]</sup>

### Psoriasis

Psoriasis is a chronic immune-mediated inflammatory disorder, involving the complex interplay of numerous proinflammatory cytokines such as IL-12, 17, 22 and 23 in its pathogenesis.<sup>[14]</sup> PDE4 inhibitors, by reducing intracellular cAMP levels, inhibit the generation of cytokines such as IL 2, 12, 23, tumour necrosis factor (TNF) alpha and interferon gamma involved in pathogenesis of psoriasis.<sup>[19]</sup> Studies support the use of systemic PDE4 inhibitor like apremilast, which has been approved for

**Table 2:** Clinical trials of crisaborole.

References	Study design	Patient characteristic	Number of participants	Treatment duration	Primary outcome and measure	Results
Zane et al. <sup>[17]</sup>	Randomised, double-blind, vehicle-controlled, single centre phase I study	Healthy, 18–55 years.	32	3:1 randomisation crisaborole ointment, 2%: vehicle BID to 13 sensitive and nonsensitive skin areas for 21 days	Local tolerability scale	98.8% of all tolerability assessments had a grade of 0 (no signs/symptoms of irritation); 0.85% had a grade of 1 (mild); 0.1% had a grade >1
Zane et al. <sup>[4]</sup>	Multicentre, open-label, maximal use phase 1b study	>12–17 years, diagnosis of AD, minimum AD involvement for treatable percentage of BSA depending on age and ISGA score of 2 (mild) or 3 (moderate)	34	2% Crisaborole ointment, BID to affected areas for 28 days	Pharmacokinetics and safety assessments	Median T <sub>max</sub> for crisaborole was 3 hours on day 1 and day 8. Systemic exposure to crisaborole and metabolite (AN7602) and their accumulation was minimal. 23 patients reported one or more TEAE, 95% of which were mild to moderate.
Gold et al. <sup>[18]</sup>	Multicenter, double-blind, bilateral-comparison, dose-ranging, phase 2 study	12–17 years, clinical diagnosis of mild to moderate AD, ≤35% BSA involved, 2 lesions with similar severity were chosen in each individual.	86	1:1 randomisation with one lesion received crisaborole 0.5% ointment whereas other 2%, for 28 days.	Change from baseline in ADSI score for each lesion	All dosing regimens resulted in improved ADSI scores with the greatest improvement observed with crisaborole ointment, 2%, applied BD.
Murrell et al. <sup>[26]</sup>	Multicenter, randomised, double-blind, vehicle controlled, bilateral evaluation, phase IIa study	18–75 years, diagnosis of mild to moderate AD, 2 comparable lesion were chosen	25	2% Crisaborole ointment or vehicle, each to 1 of the 2 target lesions	Change in ADSI score from baseline to day 28	At day 28, 17 (68%) patients experienced a greater decrease in ADSI scores for the crisaborole-treated lesion compared vehicle treated lesion
Paller et al. <sup>[13]</sup>	Two multicentric randomised double blinded vehicle controlled phase 3 study	2–79 years, clinical diagnosis of AD and baseline ISGA score of 2 (mild) or 3 (moderate)	AD-301: 763; AD302: 764	2:1 randomisation 2% crisaborole ointment : vehicle BD to affected areas (except scalp) for 28 days	ISGA score at day 29 of clear (0) or almost clear (1) with a 2-grade or greater improvement from baseline	Greater number of patients treated with crisaborole 2% ointment achieved ISGA scores success (clear <sup>[0]</sup> or almost clear <sup>[1]</sup> with a 2-grade or greater improvement from baseline) compared with patients treated with vehicle

(Contd...)

**Table 2:** (Continued).

References	Study design	Patient characteristic	Number of participants	Treatment duration	Primary outcome and measure	Results
Eichenfield <i>et al.</i> <sup>[25]</sup>	Randomised, double-blind, vehicle-controlled, phase III clinical trial with an initial open-label run-in phase. (CrisADe CONTROL study)	Patients aged $\geq 3$ months with mild-to-moderate atopic dermatitis involving $\geq 5\%$ treatable BSA who had responded to crisaborole BID in the run-in period.	497 entered the open-label run-in; 270 responders were randomised (135 to crisaborole QD, 135 to vehicle QD).	All patients first received crisaborole 2% ointment twice daily during an open-label run-in phase of up to 8 weeks. Responders, defined by ISGA success and EASI-50 improvement, were then randomised to receive either once-daily crisaborole or vehicle for a 52-week double-blind maintenance phase. Patients experiencing flares (ISGA $\geq 2$ ) could temporarily return to crisaborole twice daily until resolution before resuming their assigned treatment. A total of 497 patients entered the run-in phase and 270 responders were randomised equally between crisaborole and vehicle groups.	Time to onset of first flare (flare defined as ISGA $\geq 2$ ) during the maintenance phase.	Median flare-free maintenance: 111 days with crisaborole versus 30 days with vehicle ( $P=0.0034$ ). Mean flare-free days: 234.0 versus 199.4 days ( $P=0.0346$ ). Mean number of flares: 0.95 versus 1.36 ( $P=0.0042$ ). Well tolerated, with no new or unexpected safety findings.

ISGA: Investigator's static global assessment score, TEAE: Treatment emergent adverse effects, AD: Atopic dermatitis, BSA: Body surface area, NEAT: Nuclear Factor of activated T-cells, CYP: Cytochrome P450, BD and BID: Bis in die (twice daily), QID: Quater in die (four times daily)

use in psoriasis and psoriatic arthritis.<sup>[19,20]</sup> Systemic PDE4 inhibitors are associated with adverse effects such as nausea, vomiting, bloating, flatulence, headache and depression which limit their use.<sup>[20]</sup> Crisaborole has been evaluated in chronic plaque psoriasis in a phase 2b randomised, double-blinded, vehicle-controlled and bilateral comparison 12-week study, which concluded that 2% crisaborole ointment twice daily regimen was effective and well tolerated.<sup>[21]</sup> More recently

crisaborole has been evaluated for use in sensitive areas such as intertriginous, anogenital and facial lesions, which was found to be effective and well tolerated.<sup>[22]</sup>

### Vitiligo

Vitiligo is an autoimmune disease resulting from the loss of epidermal melanocytes. Inflammatory and immune

responses are the essential mechanisms inducing dysfunction and death of melanocytes. PDE-4 inhibitors inhibit type-2 cytokines such as IL-4 and IL-13 and also reduce cytokines such as TNF- $\alpha$  and IL-1, which have a role in vitiligo. Three case reports showed crisaborole being successfully used for patients who did not respond to conventional treatment approach.<sup>[23]</sup>

### Seborrheic dermatitis

A trial in 30 adults (18–80 years) with mild-moderate facial seborrheic dermatitis (SD) showed that twice-daily 2% crisaborole for 4 weeks led to clearance or near-clearance in majority of cases. Clinical improvements included reduction in erythema, scaling, dryness and pruritus. Only one participant discontinued due to headache and facial pain.<sup>[24]</sup>

### Inflammatory linear verrucous epidermal nevus (ILVEN)

Case reports highlight benefit in refractory ILVEN. A 9-year-old girl unresponsive to steroids, calcineurin inhibitors and Vitamin D analogues improved with crisaborole. A 5-year-old boy also achieved control with twice-daily application. Although limited to reports, the safety and long-term usability make it promising.<sup>[24]</sup>

### Morphea

A pilot, open-label study in seven adults with active morphea (<20% body surface area involvement) treated with crisaborole for 12 weeks showed: Histologic reduction of dermal fibrosis in five patients. Clinical improvement (reduction in induration and plaque size) in 6 patients suggested as an option in steroid-refractory or intolerant cases.<sup>[24]</sup>

### Stasis dermatitis

A randomised study ( $n = 65$ ) compared crisaborole with vehicle for 6 weeks. Crisaborole significantly reduced total clinical sign scores compared to vehicle. Both groups had a few dropouts due to adverse effects.<sup>[24]</sup>

### Alopecia areata

PDE-4 activity is elevated in alopecia areata lesions, making it a potential therapeutic target. Evidence is still theoretical and exploratory, which suggested mainly for limited patches.<sup>[23]</sup>

### Rosacea

A 68-year-old woman with steroid-unresponsive rosacea improved markedly within 2 weeks of crisaborole plus ceramide cream highlights benefit in inflammatory facial dermatoses when standard treatments fail.

### Other reported uses

- Vulvar leukoplakia (lichen simplex chronicus, lichen sclerosus): Higher response rate with crisaborole compared to Vitamin E.
- Chronic hand eczema: Retrospective series ( $n = 251$ ) showed improvement in majority cases.
- Irritant contact dermatitis: A nurse with sanitizer-induced hand dermatitis, lesions cleared in 8 weeks.
- Knuckle pads: Thick plaques resistant to steroids improved within 2 weeks.
- Necrobiotic xanthogranuloma with myeloma: Complete response reported with topical crisaborole in a patient with multiple myeloma.<sup>[24]</sup>

### ADVERSE EFFECTS

Crisaborole is well tolerated across various trials, with most common side effect reported being mild to moderate, like application site discomfort, resolving within a day.<sup>[13]</sup> In a study by Eichenfield *et al.*, each patient used a significant amount of ointment (133 g/patient/month) over 52 weeks without any increased risk of side effects, establishing the long-term safety.<sup>[25]</sup> During maximal use study, systemic exposure to crisaborole and its metabolite was found to be limited.<sup>[4]</sup> Moreover, no serious side effects such as increased risk of malignancies,<sup>[25]</sup> significant vital or laboratory abnormalities or death was reported with long-term use.<sup>[26,27]</sup> Ciaravino *et al.*, investigated the increased risk of malignancy on long-term use of high doses of oral and topical crisaborole in mice and found that topical application even in high doses was not associated with increased malignancy risk. However, oral doses >300 mg/kg/day were shown to increase the risk of benign granular cell tumour of reproductive tract in female mice. Such risk was noted at dose of 100 mg/kg/day; researchers consider this benign granular cell tumour of low human relevance.<sup>[28]</sup>

### DISCUSSION

Skin conditions such as AD, psoriasis and vitiligo due to their chronic and recurring course require prolonged treatment. At present, the mainstay of the management of these conditions is topical corticosteroid (TCS). However, prolonged use of TCS is associated with side effects such as telangiectasia, skin atrophy, hypertrichosis, striae and hypothalamo-pituitary-adrenal axis suppression. Further, delicate areas such as face, genitals and intertriginous areas TCS are not advisable. Awareness about these adverse effects has also shown to be a reason for non-compliance. Topical calcineurin inhibitors such as tacrolimus and pimecrolimus, with favourable safety profiles and low systemic absorption, are considered to be second-line agents in the management of inflammatory skin conditions, but their use is limited because they have some

irritant effect and the black box warning of an increased risk of malignancy. Thus, crisaborole, with minor local side effects such as application site discomfort and low systemic absorption with no increased side effects or risk of malignancy on long-term use, makes it an appealing option for the management of various chronic inflammatory skin conditions. However, more trials are needed to expand its scope in dermatology.

## CONCLUSION

Crisaborole is a promising non-steroidal topical PDE4 inhibitor with excellent safety and tolerability profiles. It provides an effective steroid sparing alternative for chronic inflammatory dermatoses like atopic dermatitis, psoriasis, and vitiligo. While current evidence supports its use in mild-to-moderate atopic dermatitis, ongoing research is expected to expand its indications for other inflammatory skin diseases.

## MCQS

- US FDA approved indication of crisaborole in dermatology
  - Psoriasis vulgaris
  - Atopic dermatitis
  - Vitiligo
  - Mycosis Fungoides
- Half life of crisaborole
  - 7–11 h
  - 5–6 h
  - 2–5 days
  - 2–5 h
- Crisaborole is available as
  - 2% ointment
  - 0.05% cream
  - 0.1% gel
  - 2% cream
- Topical crisaborole requires dose adjustment in following conditions
  - Pediatric population
  - Liver diseases
  - Chronic renal diseases
  - None of the above
- Crisaborole was US FDA approved for atopic dermatitis in
  - 2014
  - 2013
  - 2016
  - 2018
- Most common side effect associated with topical crisaborole
  - Pain
  - Redness
  - Skin atrophy
  - Application site discomfort
- Long-term use of crisaborole is associated
  - Skin atrophy
  - Cutaneous malignancy
  - Telangiectasia
  - None of the above
- Molecular weight of crisaborole
  - 150 daltons
  - 500 daltons
  - 251 daltons
  - 268 daltons
- Pregnancy category of crisaborole ointment
  - Category X
  - Category A
  - Category B
  - Not determined
- Mechanism of action of Crisaborole involves:
  - Inhibition of PDE4  $\rightarrow$   $\uparrow$  cAMP  $\rightarrow$   $\downarrow$  proinflammatory cytokines
  - Inhibition of PDE5  $\rightarrow$   $\uparrow$  nitric oxide
  - Activation of calcineurin pathway
  - Direct suppression of melanocyte activity

## ANSWERS

- b;
- a;
- a;
- d;
- c;
- d;
- d;
- c;
- d;
- a;

**Ethical approval:** The Institutional Review Board approval is not required.

**Declaration of patient consent:** Patient's consent was not required as there are no patients in this study.

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