Expert Opinion

1. Introduction
2. Overview of the market
3. Indications
4. Chemistry
5. Pharmacokinetics and absorption
6. Formulations
7. Clinical efficacy
8. Safety
9. Expert opinion

informa
healthcare

Desonide: a review of formulations, efficacy and safety

NR Kahanek, CG Gelbard† & AA Hebert
University of Texas-Houston Medical School, 6431 Fannin, Houston, TX 77003, USA

Background: Desonide is a low-potency topical corticosteroid that has been used for decades in the treatment of steroid-responsive dermatoses. The favorable safety profile of this topical agent makes it ideal for patients of all ages. *Objective*: This article provides a review of desonide's history, pharmacodynamic properties, vehicle technology, efficacy and safety. *Methods*: Randomized controlled trials, as well as open-label and non-comparative studies, case series and reports, experimental models, and data from the Galderma pharmacovigiliance program were reviewed in order to address the clinical efficacy and safety of desonide. *Results/conclusion*: Clinical efficacy and safety have been proven in multiple clinical trials. In addition to cream, lotion and ointment formulations, the recently developed hydrogel and foam preparations have increased desonide's versatility and patient tolerability.

Keywords: acne, adverse events, atopic dermatitis, contact dermatitis, desonide, efficacy, mechanism of action, safety, topical corticosteroids

Expert Opin. Investig. Drugs (2008) 17(7):1097-1104

1. Introduction

Topical corticosteroids are now first-line therapy for patients of all ages with a variety of dermatoses, yet their use in dermatology began only 50 years ago. Cortisone was isolated in the 1930s, and cortisol was first synthesized in 1937 [1]. In 1949, Hench found that corticosteroids could be used as treatment for rheumatoid arthritis and rheumatic fever, and he was awarded a Nobel Prize for his work. Shortly after, systemic steroids were used to treat inflammatory dermatoses, and in 1952, Sulzberger and Witten demonstrated the benefits of topical hydrocortisone in the treatment of eczematous dermatitis [2].

Topical corticosteroids have been shown to reduce itching, improve the appearance of skin, and improve quality of life. However, concerns pertaining to the side effects of steroid use contribute to poor patient compliance. Local effects include cutaneous atrophy, striae, glaucoma, contact dermatitis and tolerance. Systemic complications are rare but include Cushing's syndrome, growth retardation and suppression of the hypothalamic-pituitary-adrenal axis [3,4]. The risk of systemic effects is of greater concern in the pediatric population due to children's increased surface area/body mass ratio.

Desonide is a synthetic, nonfluorinated, low-potency corticosteroid that has been used to treat inflammatory, steroid-responsive dermatoses for over 30 years. Traditionally, only creams, ointments and lotions were available in this potency class; recently, however, novel hydrogel and foam formulations of desonide have been developed. These advancements in vehicle technology address the need for effective, well-tolerated treatments, and may enhance patient compliance and acceptability. This article will review the history, pharmacodynamic properties, efficacy and safety of desonide, and the recent therapeutic advancements that have been made in vehicle technology.

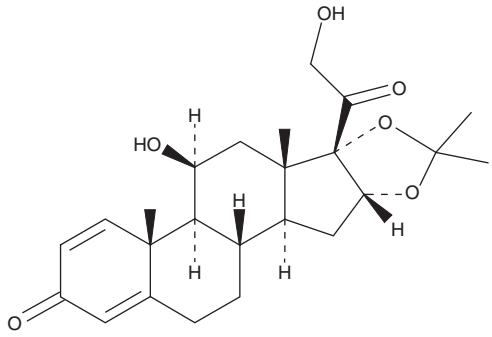

Figure 1. Structural formula of desonide.

2. Overview of the market

There are many topical steroid preparations on the market today, and production of new formulations is ongoing. Choosing the appropriate potency and vehicle is based on the nature and severity of the disease being treated and the product's ease of use.

3. Indications

Desonide is a Class VI, low-potency corticosteroid used for the treatment of atopic dermatitis, seborrheic dermatitis, contact dermatitis, psoriasis, nummular eczema and other steroid-responsive dermatoses. Like most topical corticosteroids, desonide has been shown to have anti-inflammatory, antipruritic and vasoconstrictive properties [5].

4. Chemistry

Hydrocortisone was the first corticosteroid to be used topically, and it contains the structural backbone of most topical corticosteroid molecules. Desonide is a synthetic nonfluorinated corticosteroid with the chemical formula $C_{24}H_{32}O_6$ and the structural formula depicted in **Figure 1**.

4.1 Vasoconstriction

Vasoconstriction has long been used as an indicator of topical corticosteroid potency, and skin-blanching assays are often used as surrogates for corticosteroid efficacy. The vasoconstrictive effect is attributed to decreased sensitivity of vascular smooth muscle to histamine and bradykinin, as well as an increase in the vascular response to catecholamines [4].

4.2 Anti-inflammatory

The effects of glucocorticoids on the inflammatory response include vasoconstriction, stabilization of lysosomal membranes and inhibition of inflammatory mediators from mast cells. These effects are thought to be due to both indirect and direct mechanisms. Indirect effects inhibit the inflammatory response by altering protein synthesis; this occurs via protein–protein interactions between the activated glucocorticoid receptor complex and transcription factors, or by interactions with the cell membrane [1,6].

Direct effects are a result of interactions between the activated glucocorticoid receptor and cellular DNA. Glucocorticoids are steroid hormones, and their lipophilic nature allows them to diffuse through cell membranes. Once inside a cell, steroids bind to a cytoplasmic glucocorticoid receptor [6]. A conformational change in this steroid-receptor complex allows entry into the nucleus, where the complex can bind to DNA. This binding alters gene transcription in order to regulate the inflammatory response.

Corticosteroids upregulate the transcription of the inhibitory protein lipocortin 1, which prevents phospholipase A2 from releasing arachidonic acid from membrane phospholipids. Arachidonic acid is a precursor of inflammatory mediators such as prostaglandins and leukotrienes, and it is postulated that some of the anti-inflammatory effects of corticosteroids are due to the inhibition of phospholipase A2 release and the resulting decreased production of inflammatory mediators [4,6,7]. In addition, glucocorticoids inhibit the synthesis of inflammatory cytokines such as IL-1, IL-2, TNF-α and IFN-γ [1].

4.3 Antiproliferative

Topical glucocorticoids also have antimitotic effects. Decreased epidermal mitosis results in thinning of the stratum granulosum and stratum corneum. In addition, decreased fibroblast synthesis and vasoconstriction can lead to dermal atrophy. The antiproliferative and atrophogenic effects of glucocorticoids can be an advantage when treating proliferative dermatoses such as psoriasis but can lead to unwanted skin atrophy when used to treat other disease processes [4,6].

4.4 Antipruritic

Recent studies have demonstrated a rapid improvement of pruritus in pediatric patients using desonide. Two randomized, vehicle-controlled studies involving 582 children with atopic dermatitis demonstrated marked improvement in pruritus after 4 weeks of twice-daily desonide hydrogel application. Pruritus scores in the desonide group decreased from 76% at baseline to 12% following 4 weeks of treatment. The vehicle group only showed a decrease from 69% at baseline to 41% after treatment [8].

5. Pharmacokinetics and absorption

Several factors affect the pharmacokinetics of topical corticosteroids. The molecular structure, the vehicle used for delivery and the state of the patient's skin at the site of application all play important roles in the overall efficacy of the medication.

Most topical steroids share a hydrocortisone backbone, and their differences lie in the addition or alteration of various functional groups or double bonds [9]. These subtle changes can have significant impact on the absorption and activity of the molecules. The vehicles used to deliver topical

Table 1. Desonide formulations.

Brand name	Form	Desonide concentration	Inactive ingredients
Desocort®	Cream	0.5 mg/g	Citric acid, emulsifying wax, isopropyl palmitate, polysorbate 60, propyl gallate, propylene glycol, purified water, sodium hydroxide, stearic acid, synthetic beeswax
DesOwen®	Cream	0.5 mg/g	Purified water, emulsifying wax, propylene glycol, stearic acid, isopropyl palmitate, synthetic beeswax, polysorbate 60, potassium sorbate, sorbic acid, propyl gallate, citric acid, sodium hydroxide
Desocort®	Ointment	0.5 mg/g	Mineral oil, polyethylene
DesOwen®	Ointment	0.5 mg/g	Mineral oil, polyethylene
Desocort®	Lotion	0.5 mg/g	Sodium lauryl sulfate, light mineral oil, cetyl alcohol, stearyl alcohol, propylene glycol, sorbitan monostearate, glyceryl stearate SE, edetate sodium, citric acid and/or sodium hydroxide (for pH adjustment), methyl paraben, propyl paraben, purified water
DesOwen®	Lotion	0.5 mg/g	Sodium lauryl sulfate, light mineral oil, cetyl alcohol, stearyl alcohol, propylene glycol, methylparaben, propylparaben, sorbitan monostearate, glyceryl stearate SE, edetate sodium and purified water; may contain citric acid and/or sodium hydroxide for pH adjustment
LoKara®	Lotion	0.5 mg/g	Sodium lauryl sulfate, light mineral oil, cetyl alcohol, stearyl alcohol, propylene glycol, methylparaben, propylparaben, sorbitan monostearate, glyceryl stearate SE, edetate sodium, anhydrous citric acid, purified water; may also contain additional citric acid and/or sodium hydroxide for pH adjustment
Verdeso®	Foam	0.5 mg/g	Anhydrous citric acid, cetyl alcohol, cyclomethicone, isopropyl myristate, light mineral oil, white petrolatum, polyoxyl 20 cetostearyl ether, potassium citrate (monohydrate), propylene glycol, purified water, sorbitan monolaurate, phenoxyethanol
Desonate®	Gel	0.5 mg/g	Purified water, glycerin, propylene glycol, edetate disodium dihydrate, methylparaben, propylparaben, sodium hydroxide, Carbopol® 981

steroids can greatly affect the absorption and distribution of corticosteroids, and may offer direct therapeutic effects as well. Penetration of topical corticosteroids is also affected by the condition of the patient's skin. Increased penetration is seen in areas of cutaneous inflammation and impaired barrier function. Well-moisturized skin also demonstrates higher penetration of these topical treatments when compared to xerotic skin [4].

6. Formulations

Desonide comes in ointment, cream, lotion, gel and foam formulations (Table 1). Desonide, the active ingredient, has a molecular weight of 416.52 kD and is a white, odorless powder that is practically insoluble in water, sparingly soluble in ethanol and in acetone, and soluble in methanol and chloroform [10].

6.1 Ointment

Each gram of desonide ointment contains 0.5 mg of desonide microdispersed in a base that typically consists of mineral oil and polyethylene [11-14].

6.2 Lotion

Each gram of desonide lotion contains 0.5 mg of desonide in a base of sodium lauryl sulfate, mineral oil, cetyl alcohol, stearyl alcohol, propylene glycol, sorbitan stearate, glyceryl stearate SE and edetate sodium. Some formulations contain methylparaben, propylparaben, anhydrous citric acid and purified water. Additional citric acid and/or sodium hydroxide may be added for pH adjustment [11-15].

6.3 Cream

Each gram of desonide cream contains 0.5 mg of desonide microdispersed in a base of glycerin, sodium lauryl sulfate, aluminum sulfate, calcium acetate, dextrin, purified water, cetyl stearyl alcohol, synthetic beeswax (B-wax), white petrolatum and light mineral oil. Alternative base formulations contain purified water, emulsifying wax, propylene glycol, stearic acid, isopropyl palmitate, synthetic beeswax, polysorbate 60, potassium sorbate, sorbic acid, propyl gallate, citric acid and sodium hydroxide. Desonide cream is reserved with methylparaben and is buffered to pH 4.2 – 5.0 [11,12].

6.4 Gel

Desonide gel is an alcohol- and surfactant-free carbopol-based polymer containing 0.05% of micronized desonide. Each gram of desonate gel contains 0.5 mg of desonide in an aqueous gel base of purified water, glycerin, propylene glycol, edetate disodium dihydrate, methylparaben, propylparaben, sodium hydroxide and Carbopol 981 [7].

6.5 Foam

Each gram of desonide foam contains 0.5 mg of desonide in a base of anhydrous citric acid, cetyl alcohol, cyclomethicone, isopropyl myristate, light mineral oil, white petrolatum, polyoxyl 20 cetostearyl ether, potassium citrate (monohydrate), propylene glycol, purified water, sorbitan laurate and phenoxyethanol as a preservative. Desonide foam is dispensed from an aluminum can pressurized with a hydrocarbon (propane/butane) propellant [10].

7. Clinical efficacy

Several large studies have examined the efficacy (**Table 2**) and safety of desonide in the treatment of steroid-responsive dermatoses. In 1995, a randomized trial compared 0.05% desonide ointment to 1.0% hydrocortisone ointment in 113 children aged 10 months to 12 years with mild to moderate atopic dermatitis. Patients were randomized to receive treatment with desonide ointment or hydrocortisone ointment twice daily for 5 weeks, and 36 patients continued treatment for an additional 20 weeks. The physician's overall global assessment of improvement was the primary efficacy variable and included evaluation of erythema, lichenification, excoriations, oozing/crusting, induration/papules and pruritus. Efficacy scores at all evaluations (weeks 1, 3 and 5, and months 2 to 6) showed greater efficacy and more rapid improvement with 0.05% desonide ointment than with 1.0% hydrocortisone ointment (69% desonide vs 41% hydrocortisone; $p = 0.003$). Neither treatment group showed signs of cutaneous atrophy, and any stinging or burning reported was slight, demonstrating equal topical safety profiles [16].

Two studies with a total of 582 patients, aged 3 months to 18 years, with mild to moderate atopic dermatitis demonstrated the safety and efficacy of 0.05% desonide hydrogel. In these studies 425 subjects were randomized to the desonide hydrogel arm, and 157 to the hydrogel vehicle arm. The primary efficacy end point was determined by the Investigator's Global Severity Score (IGSS). Other efficacy assessments included erythema, induration, oozing/crusting, induration/papulation, lichenification, scaling and pruritus. Total success rate was 39% for the hydrogel group and 11% for the vehicle group ($p < 0.001$), and severity scores for secondary end points were markedly improved from baseline to week 4 in the desonide hydrogel group when compared to the hydrogel vehicle group. Adverse events occurred in 20% of the desonide hydrogel group, compared with 29% in the hydrogel vehicle group. Adverse events occurred at the application site in 3% of subjects treated with desonide hydrogel, and included application-site burning, rash and pruritus. Application-site adverse events were not higher when compared to the hydrogel vehicle group, and there were no reports of skin atrophy in the desonide hydrogel group. The remainder of the adverse events were considered to be unlikely or definitely not related to the study medication. Desonide is officially indicated for use in patients aged 3 months and older as a result of its proven efficacy and safety [8].

In 2003, a noncomparative Indian study of 1789 patients with mild to moderate steroid-responsive dermatosis evaluated the efficacy and safety of desonide 0.05% cream and lotion. Of the 1483 subjects who completed the treatment according to the protocol, 98.5% showed at least some improvement in their dermatitis, and over half showed > 75% improvement when compared with baseline. No adverse events were noted in any of the patients [17].

In an Australian study, 81 patients with atopic or seborrheic dermatitis affecting the face were evaluated in order to determine the efficacy, cutaneous tolerance and cosmetic acceptability of desonide 0.05% lotion. Subjects were randomized to receive twice-daily treatment over 3 weeks with either desonide lotion or vehicle. In the group treated with desonide, 88% were considered clear or almost clear of disease at the completion of the study, compared to 42% in the vehicle-only group. Desonide lotion was well tolerated, with only 2 occurences of adverse effects (rash in non-treatment location and pruritus) seen in the desonide group, versus 17 in the vehicle-only group. There were no severe adverse events in the desonide group but 4 patients in the vehicle group had a serious adverse event (rash). In addition, a patient survey showed that 97% of the patients in the desonide group found the lotion pleasant to use, and 95% would use it again [18].

A French trial compared the efficacy of 0.1% micronized desonide cream and 0.05% betamethasone dipropionate (BMDP) cream in children with atopic dermatitis. The authors found that both treatments resulted in significant improvement in percentage of body surface involved and lesion score but that there was no statistically significant difference between the two groups [19].

8. Safety

Adverse effects that have been associated with topical corticosteroids include stinging and burning, irritation, contact dermatitis, worsening of the condition, peeling of skin, itching, erythema, dryness, folliculitis, acneiform eruptions, hypopigmentation, perioral dermatitis, secondary infection, skin atrophy, striae and miliaria [13]. Systemic effects include Cushing's syndrome, growth retardation, and suppression of the hypothalamic-pituitary-adrenal axis [3,4]. Despite these concerns regarding local and systemic adverse effects of topical corticosteroids, desonide has been shown to have a low side-effect profile in multiple studies.

A pharmacovigilance program initiated by Galderma in 1992 collected reports of adverse events associated with topical desonide over 9 years. The program collected 62 reports, 37 of which were provided by consumers and were not medically confirmed. There were no serious reactions directly attributed to desonide treatment and the

Table 2. Trials of desonide efficacy.

Ref.	Sample size	Patient age	Design	Formulation	Comparison	Measure of effect	Results
[16]	113	10 months – 12 years	Multicenter, randomized, investigator-masked, parallel-group study	0.05% Desonide ointment	1.0% Hydrocortisone ointment	Physician's global assessment	Greater efficacy and more rapid improvement was seen with desonide compared to hydrocortisone (69% vs 41%)
[8]	582	3 months – 18 years	Two multicenter, randomized, blinded, vehicle-controlled studies	0.05% Desonide hydrogel	Hydrogel vehicle	Investigator's Global Severity Score	Two independent studies showed total success rate of 39% in hydrogel group, compared to 11% in vehicle group. Hydrogel also demonstrated markedly improved severity scores for secondary end points
[17]	1483	Adults and children	Multicenter, open-label, noncomparative	0.05% Desonide cream or lotion	None	Total clinical severity score	98.5% showed an improvement in dermatitis, and over half showed greater than 75% improvement when compared with baseline
[18]	81	18 – 66 years	Double-blind, multicenter, parallel-group comparison	0.05% Desonide lotion	Vehicle	Clinical evaluation of disease status Global assessment of improvement	In the group treated with desonide, 88% were considered clear or almost clear of disease after twice-daily application for 3 weeks, compared to only 42% in the vehicle-only group
[19]	29	< 8 years	Randomized, double-blind, parallel-group comparison	0.1% Micronized desonide cream	0.05% Betamethasone dipropionate cream	Involved body surface Lesion score	Both groups showed a significant improvement in percentage of body surface involved and lesion score, but there was no statistically significant difference between the two groups

Table 3. Evaluations of hypothalamic-pituitary-adrenal axis suppression in patients treated with desonide.

Ref.	Sample size	Design	Formulation	Comparison	Measure of systemic effect	Results
[3]	40	Multicenter, open-label	0.05% Desonide hydrogel	None	Measurement of cortisol levels pre- and post-administration of Cortrosyn at baseline and week 4	No adrenal suppression was seen in patients completing study without protocol violations (37/40)
[25]	20	Randomized, parallel-group, open-label	0.05% Desonide ointment	2.5% Hydrocortisone ointment	Early-morning serum cortisol measured on days 0, 14, and 28; ACTH-stimulated cortisol level measured on days 0 and 28	No effect was seen on baseline cortisol levels or ACTH-stimulated values after treatment with desonide or hydrocortisone ointment
[19]	29	Randomized, double-blind parallel-group	0.1% Micronized desonide cream	0.05% BMDP cream	Plasma cortisol levels on days 0, 5, 20 and 30	Decreased plasma cortisol levels were seen between day 0 and day 5 in both groups. Level remained depressed on days 20 and 30 only in the BMDP group. This study utilized 0.1% desonide formulation (only 0.05% is approved in the US)

ACTH: Adrenocorticotropic hormone; BMDP: Betamethasone dipropionate.

majority of events reported were classified as expected local reactions. Skin irritation was the most common complaint and included burning, stinging, pruritus, erythema, swelling, sensitive skin, dry skin and blisters. Additional adverse events were rare, and included scars on the face, bleaching of the skin around the treatment area, cataract formation, tingling feet, difficulty sleeping and topical corticosteroid-induced acne. Medical confirmation was available for only 40% of all reports. Reports of generally mild adverse events in only 62 cases over a 9-year period underscores the safety of desonide in the treatment of atopic dermatitis. Specific side effects are detailed in Sections 7.1 to 7.9 below.

8.1 Contact dermatitis

Although reactions to topical corticosteroids often represent sensitivity to a vehicle ingredient, and testing can be difficult [20,21], there have been rare reports of contact dermatitis reactions to desonide [5,20,22]. In a series of 29 patients with contact dermatitis to topical corticosteroids, 3 patients demonstrated reactivity to desonide on patch testing [22].

Despite the structural similarities between desonide and budesonide, evidence supporting cross-reactivity between these two medications is lacking. In a small study of 20 patients with known sensitivity to budesonide, patch testing was done to determine desonide sensitivity. Patch testing was positive in one patient but the patient had used multiple topical steroids in the past, including desonide, and the reaction was thought to be the result of concomitant sensitization rather than cross-reactivity [5].

Nine reports of allergic reactions were noted in the Galderma surveillance program, seven of which were presumed cases of contact dermatitis. Of these, five cases were confirmed by either dechallenge and rechallenge, or by epicutaneous testing [23].

8.2 Atrophy

Jorizzo *et al.* found that children using desonide 0.05% ointment twice daily showed no signs of cutaneous atrophy after 5 (5 – 25) weeks of treatment [16]. In a study by Hebert, there were no reported cases of skin atrophy in 425 subjects treated with desonide hydrogel [8].

8.3 Tingling/burning

Jorizzo *et al.* report that any stinging or burning sensations noted in their study were slight [16]. In the study by Hebert, the incidence of application-site burning was 1% in the desonide hydrogel group, which was not higher than that reported in the vehicle group [8].

8.4 Telangiectasia

A single case in which telangiectasias appeared at the application site was reported in the study by Hebert [8].

8.5 Acne

One case report of acne associated with desonide use was identified. A 2-year-old girl developed topical corticosteroid-induced acne (perioral dermatitis) after using a regimen of clotrimazole 1%, betamethasone 0.05% cream and desonide 0.05% cream [24].

8.6 Bleaching

No reports of desonide-induced skin bleaching have appeared in the literature.

8.7 Non-improvement/worsening

The Galderma surveillance program reported five cases in which patients experienced exacerbation of disease, described as worsening redness of the skin.

8.8 Ophthalmologic

Three cases of eye irritation were reported in the Galderma surveillance program. One patient experienced eye swelling and facial edema following application of desonide to the chest. A second patient developed iritis after application of desonide cream under the eyes, and the third developed burning, irritated eyes after application of desonide cream to an unspecified site [23]. Cataracts have been reported in association with topical corticosteroid use but none have been reported in association with desonide specifically.

8.9 Systemic effects and hypothalamic-pituitary-adrenal axis suppression

Systemic effects of topical corticosteroids are related to their percutaneous absorption. Although rare, there have been reports of growth retardation, intracranial hypertension, Cushing's syndrome, osteoporosis and hypothalamic-pituitary-adrenal (HPA) axis suppression (**Table 3**) [4]. Children have an increased body surface area to weight ratio compared to adults, making systemic absorption a greater concern in the pediatric population. Factors that increase the likelihood of systemic side effects include high daily dosage, longer duration of administration of the treatment, greater body surface covered by the treatment, and increased potency of the corticosteroid [25].

The systemic absorption of topical corticosteroids can be measured indirectly by evaluating endogenous cortisol production [19]. Early-morning cortisol levels and ACTH-stimulated cortisol levels measured before, during and after the completion of treatment with topical corticosteroids are used to quantify adrenal suppression.

A randomized, parallel-group, open-label study of 20 patients aged 11 months to 11 years with atopic dermatitis evaluated the effect of 0.05% desonide ointment and 2.5% hydrocortisone ointment on the HPA axis. The patients had a minimum affected body surface area of 20%, with an average of 38.1% in the desonide group. Patients were treated with either desonide or hydrocortisone ointment twice daily for 4 weeks. Baseline morning serum cortisol was measured on days 0, 14 and 28, and ACTH-stimulated cortisol level was measured on days 0 and 28. Neither the baseline cortisol levels nor the ACTH-stimulated values were affected by treatment with desonide or hydrocortisone ointment. The absence of adrenal suppression when using an ointment preparation predicts a similar outcome when other, less occlusive vehicles are used [25].

A multicenter, open-label study of 40 children aged 6 months to 6 years with moderate to severe atopic dermatitis evaluated HPA axis suppression potential, tolerability and efficacy of 0.05% desonide hydrogel applied twice daily for 4 weeks. No suppression of adrenal function was observed in subjects who completed the study according to protocol. Of the subjects who completed the study with complications in cortisol testing, there was one subject (1/37 = 3%) who had a low post-stimulation cortisol level at week 4. This low cortisol level may have been due to technical problems in administering the cosyntropin injection and a late blood draw for cortisol-level testing. Therefore, the investigator deemed this laboratory result an unreliable indicator of adrenal suppression. In addition, desonide hydrogel 0.05% exhibited marked efficacy in all parameters examined, including erythema, induration, oozing/crusting and percentage of body surface area affected. By week 4, 55% of subjects were deemed to show treatment success on the Physician's Global Severity Score (PGSS) [3].

In general, the therapeutic effects of topical corticosteroids have been assumed to correlate with their local and systemic side effects. However, a French trial comparing the effect of 0.1% micronized desonide cream and 0.05% BMDP cream on the adrenal cortex demonstrated that desonide has a less suppressive effect than BMDP, despite their similar therapeutic effects. The dissociation between the therapeutic effect and the effect on the adrenal cortex remain unclear. Pharmacokinetic hypotheses include epidermal metabolism of desonide, yielding an inactive metabolite for systemic absorption, or rapid metabolism and inactivation of the drug in the circulation [19]. Although adrenal suppression was observed in both the desonide and the BMDP groups, the desonide concentration was twice as high in this clinical trial as that approved for use in the United States.

A study using mouse and rat experimental models demonstrated that desonide was more potent than hydrocortisone and had no thymolytic effects at the highest cutaneous doses: 'Desonide is a glucocorticoid having a high local anti-inflammatory/systemic side-effect ratio' [26].

9. Expert opinion

Desonide is a nonfluorinated, low-potency topical steroid used to treat atopic dermatitis, seborrheic dermatitis, contact dermatitis, psoriasis and nummular eczema. The efficacy of desonide is attributed to its vasoconstrictive, anti-inflammatory and antipruritic properties. Multiple studies have demonstrated the efficacy and safety of desonide in the treatment of steroid-responsive dermatoses. The incidence of adverse effects is low, and multiple trials including trials on children have shown an absence of adrenal suppression after treatment with desonide ointment, hydrogel and cream formulations. In addition to exhibiting remarkable efficacy and safety profiles, desonide is also exceedingly versatile, with currently

available ointment, cream, lotion, gel and foam preparations. The more recently developed gel and foam formulations address the need for improved patient tolerability, and should enhance patient adherence to steroid treatment regimens.

Declaration of interest

AA Hebert is a researcher, lecturer and consultant for Stiefel Laboratories.

Bibliography

Papers of special note have been highlighted as either of interest (•) or of considerable interest (••) to readers.

1. Stahn C, Lowenberg M, Hommes DW, Buttgereit F. Molecular mechanisms of glucocorticoid action and selective glucocorticoid receptor agonists. Mol Cell Endocrinol 2007;275:71-8
2. Smith EB, Gregory JF, Bartruff JK. Desonide, a potent nonfluorinated topical steroid, vasoconstriction assay and clinical trial. South Med J 1973;66:325-9
3. Eichenfield LF, Basu S, Calvarese B, Trancik RJ. Effect of desonide hydrogel 0.05% on the hypothalamic-pituitary-adrenal axis in pediatric subjects with moderate to severe atopic dermatitis. Pediatr Dermatol 2007;24:289-95
4. Wolverton S. Comprehensive dermatologic drug therapy. 2nd edtion. Philadelphia: Elsevier; 2007
5. Foti C, Cassano N, Vena GA. Evaluation of cross-reactivity between budesonide and desonide. Contact Dermatitis 2002;47:109-10
6. Ahluwalia A. Topical glucocorticoids and the skin. Mechanisms of action: an update. Mediators Inflamm 1998;7:183-93
7. SkinMedica. Desonate™ (desonide) Gel 0.05% package insert, 2006. Available from: www.fda.gov/cder/foi/label/2006/021844lbl.pdf [last accessed 28 May, 2008]
8. Hebert AA, Cook-Bolden FE, Basu S, et al. Safety and efficacy of desonide hydrogel 0.05% in pediatric subjects with atopic dermatitis. J Drugs Dermatol 2007;6:175-81
9. Thalen A, Brattsand R, Andersson PH. Development of glucocorticosteroids with enhanced ratio between topical and systemic effects. Acta Derm Venereol Suppl (Stockh) 1989;151:11-9; discussion 47-52
10. Verdeso. desonide) Foam, 0.05% package insert, 2006. Available from: www.verdeso.com/pdf/PI.pdf [last accessed 29 May, 2008]
11. Galderma. 2007. Available from: www.galderma.ca/en/prod_sterc.cfm [last accessed 31 March, 2008]
12. Desowen. Website, 2007. Available from: www.rxlist.com/cgi/generic/desonide.htm [last accessed 29 May, 2008]
13. Fougera. Desonide loiton 0.05% package insert, 2007. Available from: www.fougera.com/products/documents/1170.PI.pdf [last accessed 29 May, 2008]
14. Taro. Desonide cream 0.05%, desonide ointment 0.05% package insert, 2007. Available from: www.taro.com/media/oMedia/215_6_300.pdf [last accessed 29 May, 2008]
15. LOKARA. LoKara™ Lotion (desonide lotion 0.05%) package insert, 2005. Available from: www.pharmaderm.com/physician_info/lokara/lokara_lotion.html [last accessed 29 May, 2008]
16. Jorizzo J, Levy M, Lucky A, et al. Multicenter trial for long-term safety and efficacy comparison of 0.05% desonide and 1% hydrocortisone ointments in the treatment of atopic dermatitis in pediatric patients. J Am Acad Dermatol 1995;33:74-7
17. Bhankharia DA, Sanjana PH. Efficacy of desonide 0.05% cream and lotion in steroid-responsive dermatoses in Indian patients: a post-marketing surveillance study. Indian J Dermatol Venereol Leprol 2004;70:288-91
18. Freeman S, Howard A, Foley P, et al. Efficacy, cutaneous tolerance and cosmetic acceptability of desonide 0.05% lotion (DesOwen) versus vehicle in the short-term treatment of facial atopic or seborrhoeic dermatitis. Australas J Dermatol 2002;43:186-9
19. Lebrun-Vignes B, Legrain V, Amoric J, Taieb A. Comparative study of efficacy and effect on plasma cortisol levels of micronised desonide cream 0.1 p. 100 versus betamethasone dipropionate cream 0.05 p. 100 in the treatment of childhood atopic dermatitis. Ann Dermatol Venereol 2000;127:590-5
20. Sturtz RP, Rau RC. Contact dermatitis to desonide. Arch Dermatol 1983;119:1023
21. Hernandez N, Assier-Bonnet H, Terki N, Revuz J. Allergic contact dermatitis from propyl gallate in desonide cream (Locapred). Contact Dermatitis 1997;36:111
22. Rivara G, Tomb RR, Foussereau J. Allergic contact dermatitis from topical corticosteroids. Contact Dermatitis 1989;21:83-91
23. Wong VK, Fuchs B, Lebwohl M. Overview on desonide 0.05%: a clinical safety profile. J Drugs Dermatol 2004;3:393-7
24. Brodell RT, O'Brien MJ Jr. Topical corticosteroid-induced acne. Three treatment strategies to break the 'addiction' cycle. Postgrad Med 1999;106:225-6, 229
25. Lucky AW, Grote GD, Williams JL, et al. Effect of desonide ointment, 0.05%, on the hypothalamic-pituitary-adrenal axis of children with atopic dermatitis. Cutis 1997;59:151-3
26. Tarayre JP, Aliaga M, Barbara M, et al. Comparison of the cutaneous/systemic antiinflammatory activity ratios for desonide and hydrocortisone in various experimental models. Arzneimittelforschung 1988;38:542-5

Affiliation

NR Kahanek[1], CG Gelbard[†,1] & AA Hebert[1,2]
[†]Author for correspondence
[1]Department of Dermatology,
University of Texas-Houston Medical School,
6431 Fannin, Houston,
TX 77003, USA
Tel: +1 713 500 8266;
Fax: +1 713 524 3432;
E-mail: Christina.M.Gelbard@uth.tmc.edu
[2]Department of Pediatrics,
University of Texas-Houston Medical School,
6431 Fannin, Houston,
TX 77003, USA