This review is a summary of evidence in support of apremilast’s (Otezla™) use in patients with moderate-to-severe plaque psoriasis. Apremilast is registered for therapeutic use in Australia, but it is not yet reimbursed on the Pharmaceutical Benefits Scheme (PBS). At the March, 2015 meeting of the Pharmaceutical Benefits Advisory Committee (PBAC), the committee rejected a major submission from Celgene for subsidy of apremilast 10 mg, 20 mg and 30 mg tablets in moderate-to-severe plaque psoriasis. The committee considered that cost-effectiveness relative to cyclosporin had not been satisfactorily defined, that an incremental efficacy and safety benefit over cyclosporin was not apparent, so that an additional treatment line before starting biologic disease-modifying antirheumatic drugs (bDMARD) therapy was inappropriate; and that the cost-utility analysis approach utilized for economic modelling was not informative.1

Celgene firmly believes that apremilast offers significant clinical advantages for patients with psoriasis and is disappointed with the PBAC outcome. Celgene is committed to continuing to work with the Department of Health and PBAC to make apremilast available to Australian patients.3

**Introduction**

Plaque psoriasis (psoriasis vulgaris) is the most frequent form of psoriasis. It is a non-contagious, chronic, inflammatory skin condition characterized by red, scaly patches and shedding of skin scales.2–4 The areas most commonly affected are the scalp, elbows and knees, although the disorder can manifest anywhere on the body.4,5 Lesions vary in size, but are typically well defined. The condition may be associated with itching and increased susceptibility of the skin to physical and chemical irritation, although many patients do not experience these symptoms.7 In some cases, the plaques may develop into painful skin cracks and fissures.7 Various genes have been linked with susceptibility to psoriasis, and several ‘triggers’ exist in genetically predisposed individuals: infections (e.g. tonsillitis); medications (e.g. β-blockers, lithium, nonsteroidal anti-inflammatory agents); poor diet; lack of exercise; stress; illness; cigarette smoke; and alcohol consumption.1–4

**Prevalence of psoriasis**

Psoriasis is estimated to affect approximately 1–4% of the population,1,7,17 and appears to be distributed equally between men and women.1,7 That is, about 125 million people worldwide are living with psoriasis.2 However, this rate is probably an underestimate because it is based primarily on patient self-reporting.1 About 80–90% of patients with psoriasis have the plaque form of the condition (psoriasis vulgaris).3,5 Although most patients (70–80%) tend to have mild disease that can be effectively managed with topical treatments and phototherapy, 20–30% of patients will have moderate-to-severe disease that typically necessitates systemic treatment.2–4 About 10–40% of patients with psoriasis may also have nail involvement and psoriatic arthritis.2–8 Nevertheless, most studies quote approximately 50% of patients with psoriasis as also having psoriatic arthritis, which can predate skin disease. Psoriatic arthritis may be associated with minimal skin disease, but severe arthritis is often associated with severe skin disease. Nail and scalp involvement are predictors of systemic arthritis.8

Most patients with psoriasis are affected by the condition for most of their lives.2 The peak ages of disease onset seem to be between 30–39 years and 60–69 years, although the disorder may manifest at any age.7 Interestingly, the prevalence of psoriasis appears to be greater at higher latitudes, and in Caucasians rather than other ethnic groups.1,7 Psoriasis is particularly rare in indigenous Australians.19

**Disease burden posed by psoriasis**

Patients with psoriasis have an increased risk of comorbidities such as liver disease, cardiovascular disease, raised lipid levels, stroke, diabetes, obesity, lymphoma, and inflammatory bowel disease. These comorbidities seem to be an additional factor associated with psoriasis per se rather than lifestyle alone. As treatments are becoming more effective in controlling the cutaneous and systemic aspects of psoriasis, long-term management of comorbidities is becoming more important and needs to be addressed. Such management clearly increases overall healthcare costs, and it is believed that the treatment of patients with several chronic conditions costs up to sevenfold more than the treatment of patients with only one long-term disorder. Furthermore, patients with psoriasis, compared with the general population, are considered twice as likely to be hospitalized because of infectious diseases.22

A recent, large, population-based, cross-sectional study in the UK compared more than 9,000 patients with psoriasis, and aged 25–64 years, with more than 90,000 age- and practice-matched controls without psoriasis.14 After adjustment for age, sex and duration of follow-up, significantly increased risks of the following comorbidities were identified in individuals with versus those without psoriasis (Figure 1): chronic pulmonary disease (+8%, p=0.02); diabetes (+22%, p=0.001); diabetes with complications (+34%, p=0.006); mild liver disease (+41%, p=0.008); myocardial infarction (+34%, p=0.03); peptic ulcer disease (+27%, p=0.04); peripheral vascular disease (+38%, p=0.02); renal disease (+28%, p=0.005); and rheumatological disease (+104%, p<0.001).14
Because of its chronic and highly visible nature, psoriasis is also often linked with psychiatric comorbidity (e.g. depression). Indeed, psoriasis can have a marked detrimental effect on activities of daily living, personal interactions — particularly those requiring skin contact — and work ability and performance. Thus, psoriasis can impose huge costs on patients and healthcare systems, and severe psoriasis is estimated to reduce lifespan by a mean of 3.5–4.5 years. Regarding cost-quantification of disease burden, data from the US in 2004 reveal that more than 3 million people were living with psoriasis. Annual direct costs associated with psoriasis management were estimated at US$1.22 billion, indirect costs due to lost productivity at US$114 million, and intangible costs due to quality-of-life impact at US$2.31 billion. More recently, the huge physical, psychosocial and medical burden of psoriasis was evaluated in two large, multicentre, cross-sectional studies in Australia that were conducted in conjunction with Psoriasis Australia. High proportions of patients reported actively hiding their disease from the public (83%), friends (58%), family (40%) and a spouse or partner (20%). In one of the two trials, a major negative effect on health-related quality of life (HRQoL) was indicated by a mean EQ-5D scale score of only 0.73. This was further reduced, to a mean of only 0.64, in the 75% of patients with comorbidities.

**Current treatment trends**

There is no cure for psoriasis, but the condition can be controlled with effective treatment. For mild psoriasis, topical preparations (e.g. corticosteroids, calcipotriol, coal tar) are at the forefront of treatment. For moderate-to-severe psoriasis, the traditional mainstays of disease management are systemic therapies such as methotrexate, cyclosporin, acitretin, and phototherapy (typically narrow-band ultraviolet B). More recently, biologic agents have revolutionized psoriasis treatment for both cutaneous and arthritic components of the disorder. Biologic agents available on the Australian PBS include adalimumab, etanercept, infliximab, secukinumab and ustekinumab. However, these agents are PBS-funded only as ‘last-line’ therapy for severe psoriasis after several other interventions have been tried and failed or are contraindicated. Some of these agents have been linked with serious infections (e.g. tuberculosis, reactivation of hepatitis B) and non-melanoma skin cancer. Whether the increased risk of non-melanoma skin cancer, and possibly melanoma, is a class effect of these medications or is due to excessive sun exposure in patients with psoriasis remains unclear. The potential for an increased risk of demelinating disorders, leukemia and lymphoma with these agents remains contentious and has not yet been clearly defined.

All traditionally and currently used systemic therapies are associated with treatment-related and idiosyncratic toxicity. Doses and durations of these therapies therefore frequently require adjustment, which can prove problematic for patients. For example, metabolic (i.e. drug interaction) concerns and the potential for hypertension, nephrotoxicity and cutaneous malignancy limit the duration of maintenance cyclosporin treatment to 1 year in the US, and to 2 years in Europe; there is no duration restriction in Australia. Women treated with cyclosporin also often complain of excessive hair production. This can occur as early as 3 months into treatment, and is a frequent cause of treatment cessation.

Phototherapy requires visits to a dermatology centre with a phototherapy machine 3 times per week. This is a slow, long-term treatment; typically, a response takes 6–8 weeks to appear. Long-term maintenance therapy requires weekly or fortnightly clinic visits. Excessive dosing leads to erythema (sunburn) and some patients develop pruritus. Psoralen plus ultraviolet A light therapy (PUVA) predates the use of narrow-band ultraviolet B machines and is extremely rarely used these days. PUVA increases the risk of skin cancer and causes nausea in many patients.

Methotrexate is widely used, particularly in the presence of psoriatic arthritis. Nevertheless, significant issues exist with methotrexate use in elderly patients and patients with pre-existing renal toxicity. Methotrexate-induced nausea can be ameliorated by concomitant use of folic acid, which should be taken by all methotrexate recipients, however, nausea may still be a frequent occurrence. Other common disease-management issues with methotrexate comprise mouth ulceration, photosensitivity and drug interactions. Although rare, the potential for long-term hepatotoxicity requires monitoring, especially in patients with diabetes, obesity or fatty liver, and in those who consume large amounts of alcohol (>2 units per day). Acitretin is an oral retinoid. It is contraindicated in women of childbearing potential, as it remains in the system for at least 2 years. It is minimally effective as monotherapy, but is frequently co-administered with phototherapy. However, it has significant mucocutaneous toxicity: dry lips, fragile skin, peeling palms and soles, and hair loss. In women, the latter adverse event, which is also common during methotrexate treatment, is a frequent cause of treatment discontinuation.

Various rotational, sequential, intermittent and combination schedules have been investigated and recommended as a means of reducing the abovementioned toxicities. In general, however, the Australian public is reluctant to switch from one medication to another if an adequate response is perceived.

**Apremilast in Australia**

The Multinational Assessment of Psoriasis and Psoriatic arthritis (MAPP) survey was a large, multinational, population-based, telephone survey conducted in North America and Europe. A total of 3,426 patients with psoriasis and 781 physicians completed the survey, which identified a major need for novel, safe and effective treatments. MAPP revealed that despite the major disease burden of psoriasis, many patients were being undertreated or not receiving prescription therapy from a physician. A total 85% of patients surveyed considered that there was a need for better treatments, and 45% of patients had not seen a physician within the previous year. More than 80% of patients with at least 4% of the body surface area (BSA) covered with psoriasis were not being treated, or were receiving topical therapy only, in addition, 57% of patients given oral therapy and 45% of those given biologic therapy had stopped treatment because of safety concerns or lack of efficacy.

Consideration of statistics such as those outlined above need to be balanced by the fact that, for the same treatments, medical costs in the US are far greater than in Australia; lost productivity in Australia is greater than that in the US because of higher wage costs in Australia. Nevertheless, these statistics are an important step towards defining the overall disease-management costs and disease burden of psoriasis.

In line with MAPP and with the need for new, safe and effective antipsoriatic therapies, much research-and-development interest continues to focus on biologic agents. For example, recently introduced inhibitors of interleukin (IL)-17 and IL-23 continue to be actively investigated. Although some of these ‘forerunning’ inhibitors are now PBS subsidized (e.g. secukinumab, ustekinumab), it will take time to establish long-term safety for these and other novel agents and to build prescriber confidence in use of these agents in the real-world, clinical setting.

Apremilast (Otezla®), however, is an orally active, small molecule that is already approved in several countries, including Australia, for the treatment of moderate-to-severe plaque psoriasis and psoriatic arthritis. It is a selective inhibitor of phosphodiesterase-4 (PDE4), the key enzyme that metabolizes cyclic adenosine monophosphate (cAMP) and that is found in chondrocytes, the dermis, smooth muscle, and the vascular endothelium. Basically, inhibition of PDE4 results in raised intracellular levels of cAMP; in macrophages, this leads to inhibition of pro-inflammatory mediators that have significant pathophysiological roles in psoriasis (e.g. IL-6, IL-12, tumour necrosis factor-α (TNF-α)).

Figure 1. *Increased risk of comorbidities in patients with psoriasis*.

*p<0.05, **p≤0.01, †p<0.001 for patients with versus those without psoriasis.

www.researchreview.com.au
Apremilast is generally well tolerated. The most frequent adverse events are: diarrhoea and nausea during the first year of treatment, typically within 2 weeks of the first dose and resolving within 4 weeks; and nasopharyngitis and upper respiratory tract infection during maintenance therapy.23 Weight loss (several kilograms) during the first 6–12 months of treatment may also be a problem for some patients. Altogether, however, the relatively low incidence of adverse events (AEs) and the oral route of administration for apremilast make the compound a particularly pioneering treatment for moderate-to-severe psoriasis.20 The overall place in therapy of the compound is established as monotherapy, but now warrants detailed evaluation in combination regimens (e.g. when used jointly with topical and/or phototherapy).24

In patients with psoriatic arthritis, apremilast can be used in combination with methotrexate. Clinical experience suggests that haematological or other monitoring is not required during apremilast treatment, but it may be clinically prudent to monitor renal function in some settings, since very low urinary outputs will require apremilast dosage reduction.

Although registered for therapeutic use in Australia, apremilast is not yet reimbursed on the PBS. The pharmaceutical company Celgene firmly believes that apremilast offers significant clinical advantages to patients with psoriasis and is committed to continuing to work with the Department of Health and PBAC to make apremilast available to Australian patients.3

### Product ‘In Brief’: Apremilast

#### Pharmacological properties

As an orally active PDE4 inhibitor, apremilast increases intracellular levels of cAMP, leading to downregulation of the inflammatory response because of modulated expression of TNF-α, IL-17, IL-23 and other inflammatory cytokines. cAMP also alters levels of anti-inflammatory cytokines such as IL-10. These pro- and anti-inflammatory cytokines have been implicated in the pathophysiology of psoriasis.25

In clinical trials in patients with psoriasis, apremilast reduced: epidermal thickness in lesional skin; inflammatory cell infiltration; and pro-inflammatory gene expression (e.g. of inducible nitric oxide synthase, IL-12/IL-23p40, IL-17A, IL-22, and IL-8).25

In healthy volunteers, apremilast — at dosages of up to 50 mg twice daily — did not prolong the QT interval.25

Regarding pharmacokinetic properties, apremilast is well absorbed after oral administration, with an absolute bioavailability of about 73%. The time (tmax) to reach peak plasma concentrations (Cmax) is approximately 2.5 hours. Apremilast pharmacokinetics are linear over the dosage range of 10–100 mg daily. When administered twice daily, apremilast accumulation is about 68% in patients with psoriasis. Concurrent food ingestion does not alter apremilast bioavailability; therefore, the compound can be administered with or without food.

Apremilast is approximately 68% bound to plasma proteins and has a mean apparent volume of distribution (Vd) of 87 L, thus indicating extravascular distribution.25

Apremilast is extensively metabolized by cytochrome P450 (CYP) and non-CYP pathways; only 3% of an administered dose is recovered unchanged in urine, and about 7% in faeces. The major circulating metabolite is a glucuronide conjugate that is inactive. Inhibition of a single clearance pathway is not expected to produce major drug-drug interactions. In healthy volunteers, the plasma clearance of apremilast is approximately 10 L/hour; the terminal elimination half-life (t1/2) is approximately 9 hours.25

In patients with mild-to-moderate renal impairment, no apremilast dosage adjustment is necessary. However, in individuals with severe renal impairment (creatinine clearance [CrCl] <30 ml/min), marked increases in apremilast bioavailability and Cmax have been reported. The dosage of apremilast should therefore be halved from 30 mg twice daily to 30 mg once daily.25

### Dosage and administration

Apremilast therapy should be started by specialists experienced in the management of psoriasis. The recommended dosage is 30 mg orally, taken twice daily approximately 12 hours apart; this is preceded by a 5-day titration schedule (Table 1).25

#### Table 1. Dosage titration schedule for apremilast

<table>
<thead>
<tr>
<th>Treatment day</th>
<th>AM dose</th>
<th>PM dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>10 mg</td>
<td>–</td>
</tr>
<tr>
<td>2</td>
<td>10 mg</td>
<td>10 mg</td>
</tr>
<tr>
<td>3</td>
<td>10 mg</td>
<td>20 mg</td>
</tr>
<tr>
<td>4</td>
<td>20 mg</td>
<td>20 mg</td>
</tr>
<tr>
<td>5</td>
<td>20 mg</td>
<td>30 mg</td>
</tr>
<tr>
<td>6 and thereafter</td>
<td>30 mg</td>
<td>30 mg</td>
</tr>
</tbody>
</table>

Apremilast tablets should be swallowed whole, with or without food; they should not be chewed, crushed or split. No dosage adjustment is needed for elderly patients, those with hepatic impairment, or those with mild-to-moderate renal impairment. However, in patients with severe renal impairment (CrCl <30 ml/min), the dosage should be reduced to 30 mg once daily; the initial titration period in such patients should comprise administration of the morning dose only (Table 1).25

#### Contraindications

Apremilast is contraindicated:25

- During pregnancy and lactation.
- In patients with known hypersensitivity to apremilast or any of the tablet excipients.

#### Drug interactions

Apremilast can be administered concurrently with methotrexate, oral contraceptives, ketoconazole, or drugs metabolized by CYP enzymes. In vitro, apremilast is not an inhibitor or inducer of CYP enzymes. Furthermore, it is only a weak inhibitor of P-glycoprotein, and has minimal inhibitory activity on various other transporter proteins (e.g. several organic anion and cation transporters, breast cancer resistance protein); clinically relevant drug-drug interactions are therefore not expected when apremilast is administered concomitantly with drugs that are substrates or inhibitors of these transporter proteins.25

Systemic exposure to apremilast may be markedly reduced when the compound is administered together with strong inducers of CYP3A4 (e.g. carbamazepine, phenobarbitone, phenytoin, rifampicin and St John’s Wort); this may result in reduced clinical efficacy for apremilast. Apremilast has not yet been evaluated in combination with cyclosporin or biologic therapies.25

#### Adverse events

The safety database for apremilast includes six multicentre, randomized, double-blind, placebo-controlled studies involving a total of 1,184 patients with psoriasis and 1,945 patients with psoriatic arthritis.26 The most frequently reported treatment-emergent AEs were related to the gastrointestinal tract: diarrhoea, 15.7% of patients; nausea, 13.9% (Table 2). The overall incidence of serious AEs was low and similar to that with placebo. The gastrointestinal AEs were mostly mild-to-moderate in severity; only 0.3% of patients had severe diarrhoea, and 0.3% had severe nausea. These events typically occurred during the first 2 weeks of treatment and usually resolved within 4 weeks.25

#### Table 2. Incidence of treatment-emergent AEs (frequency >3%) in clinical trials of apremilast

<table>
<thead>
<tr>
<th>Preferred term</th>
<th>Apremilast 30 mg twice daily n (%)</th>
<th>Placebo n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diarrhoea</td>
<td>186 (15.7)</td>
<td>28 (6.7)</td>
</tr>
<tr>
<td>Nausea</td>
<td>164 (13.9)</td>
<td>28 (6.7)</td>
</tr>
<tr>
<td>Upper respiratory tract infection</td>
<td>100 (8.4)</td>
<td>27 (6.5)</td>
</tr>
<tr>
<td>Headache</td>
<td>77 (6.9)</td>
<td>24 (3.6)</td>
</tr>
<tr>
<td>Nasopharyngitis</td>
<td>89 (7.5)</td>
<td>29 (6.9)</td>
</tr>
<tr>
<td>Tension headache</td>
<td>85 (7.2)</td>
<td>14 (3.3)</td>
</tr>
<tr>
<td>Vomiting</td>
<td>39 (3.3)</td>
<td>7 (1.7)</td>
</tr>
</tbody>
</table>

Other frequent AEs comprised upper respiratory tract infection (8.4% of patients), headache (7.9%), and tension headache (7.2%).

www.researchreview.com.au

a RESEARCH REVIEW publication
Clinical trials of apremilast
Apremilast, an oral phosphodiesterase 4 (PDE4) inhibitor, in patients with moderate to severe plaque psoriasis

Authors: Papp K et al.

Background: In clinical trials, apremilast, among many other things, reduced numbers of T cells and myeloid dendritic cells in the dermis and epidermis in psoriatic lesions. The phase III Efficacy and Safety Trial Evaluating the Effects of apremilast in psoriasis (ESTEEM) program consists of two multicentre, randomized, placebo-controlled trials in patients with moderate-to-severe plaque psoriasis. Results from ESTEEM-1, described here, were published recently.

Methods: A total of 844 adults with a Psoriasis Area Severity Index (PASI) score ≥12, BSA ≥10%, and a static Physician Assessment Global (sPGA) score of ≥3, and who were eligible for phototherapy or systemic therapy, were randomized to 16 weeks’ administration of: apremilast 30 mg twice daily (n=562) or placebo (282) for 16 weeks. For a subsequent maintenance phase (weeks 16–32), placebo recipients switched to apremilast, and apremilast-treated patients continued active treatment. A withdrawal phase (weeks 32–52) comprised a switch to placebo for initial apremilast recipients who had a ≥75% decrease in PASI score (PASI-75) from baseline to week 32. The primary endpoint efficacy was the proportion of patients with a PASI-75 response at week 16.

Results: At week 16, significantly more apremilast-treated patients than placebo recipients attained a PASI-75 response (33.1% vs 5.3%; the 95% confidence interval [CI] for the apremilast–placebo difference of 27.8% was 23.1–32.5% [p<0.0001; Figure 2]. The principal secondary endpoint — the proportion of patients with an sPGA score of 0 (clear) or 1 (almost clear), and a decrease of 2 points from baseline to week 16 — also revealed significantly greater improvement in the apremilast group (21.7% vs 3.9% of patients; 95% CI for difference 17.8–21.9% [p<0.0001; Figure 3]. At the end of the withdrawal phase (week 52), 61.0% of apremilast-treated patients compared with 11.7% of placebo recipients had attained a PASI-75 response. During the initial 16-week phase, 69.3% vs 55.7% of patients in the apremilast vs placebo group had at least one AE. AEs were generally mild or moderate in severity; severe AEs occurred in 3.6% vs 3.2% of patients. The most frequent AEs in the apremilast group were diarrhoea (18.8%) and nausea (15.7%). Overall, approximately 75% of episodes of diarrhoea and nausea occurred within 2 weeks after the first apremilast dose, and about 75% were mild in severity; approximately 60–65% of cases resolved within 1 month of onset.

Conclusions: Apremilast is the first oral PDE4 inhibitor to demonstrate efficacy as an antipsoriatic therapy. The severity of psoriasis was significantly reduced over 16 weeks, responses were sustained for up to 1 year, and the drug was generally well tolerated. Thus, “… apremilast provides a novel therapeutic option for the treatment of patients with moderate to severe plaque psoriasis.”

Comment: This paper summarizes the pivotal studies for apremilast. Diarrhoea and nausea occurred frequently within the first 2 weeks of the first dose; however, most of these events were very mild. These complaints also usually settled within a month of onset. Systemic monitoring of this medication is not required, except in patients with severe renal impairment. Apremilast is a novel oral therapy that has been shown to be helpful for both cutaneous and ankylosing spondylitis.


Improvements in patient-reported outcomes with apremilast, an oral phosphodiesterase 4 inhibitor, in the treatment of moderate to severe psoriasis

Authors: Strand V et al.

Background: Patients with psoriasis have impaired HRQoL. This may include physical distress, restrictions in activities of daily living, psychosocial difficulties and emotional disturbances. Symptom severity in psoriasis has also been linked with the extent of HRQoL impairment. Consequently, this phase IIb, multicentre, randomized, placebo-controlled study evaluated the effects of apremilast on patient-reported outcomes (PROs) over 16 weeks.

Methods: A total of 352 adults with stable, chronic, moderate-to-severe plaque psoriasis (PASI ≥12 and BSA involvement ≥10% for ≥6 months), and who were eligible for systemic therapy or phototherapy, were randomized to 16 weeks’ administration of: apremilast 10, 20 or 30 mg twice daily, or placebo. At week 16, placebo recipients were re-randomized to 8 weeks’ treatment with apremilast 20 or 30 mg twice daily; initial apremilast-treated patients continued their apremilast schedule. Minimum clinically important differences (MCIDs) in PROs were recorded for Dermatology Life Quality Index (DLQI), pruritus visual analogue scale (VAS) score, and Short Form-36 (SF-36) score.

Results: At week 16, mean DLQI and pruritus VAS scores were significantly lower in the apremilast 20 and 30 mg twice daily groups than in the placebo group (p<0.005). Approximately 45–50% of patients in these two apremilast groups, compared with 25% of placebo recipients, had DLQI improvements greater than or equal to the MCID (p<0.011 vs placebo). Corresponding percentages for pruritus VAS scores were 61–64% versus 44% (p=0.034). In the apremilast 30 mg twice daily group, four SF-36 domains showed statistically significant improvement: bodily pain (p=0.023), social functioning (p=0.028), role-emotional (p=0.006), and mental health (p=0.013).

Conclusions: Moderate-to-severe plaque psoriasis has a detrimental effect on HRQoL. In this trial, apremilast 20 or 30 mg twice daily produced reliable, statistically significant and clinically meaningful improvements in various PRO measures of HRQoL. “Findings indicate that the benefit:risk profile of apremilast results in a net improvement in HRQOL for the patient.”

Comment: It goes without saying that any medication that improves a patient’s disease will invariably also improve quality of life. This paper summarizes various disease assessment tools recorded during the study; it is relatively self-evident.


Full article
Research Review™ PRODUCT REVIEW
Otezla® (Apremilast) use in Moderate-to-Severe Plaque Psoriasis

Figure 3. Proportion of patients with an sPGA score of 0 or 1, or with a ≥2-point reduction from baseline, at week 16 in the ESTEEM-1 trial. A

References

This review was commissioned by Celgene Pty Ltd. Otezla® is a registered trade mark. The content is entirely independent and based on published studies and the presenter’s opinions. It may not reflect the views of Celgene.

Product Reviews are prepared with an independent commentary from relevant specialists. To become a reviewer please email geoff@researchreview.com.au

Research Review Australia Pty Ltd is an independent Australian publisher. Research Review receives funding from a variety of sources including Government depts., health product companies, insurers and other organisations with an interest in health. Journal content is created independently of sponsor companies with assistance from leading local specialists.

Privacy Policy: Research Review will record your email details on a secure database and will not release them to anyone without your prior approval. Research Review and you have the right to inspect, update or delete your details at any time. Disclaimer: This publication is not intended as a replacement for regular medical education but to assist in the process. The reviews are a summarised interpretation of the published study and reflect the opinion of the writer rather than those of the research group or scientific journal. It is suggested readers review the full trial data before forming a final conclusion on its merits.

Research Review publications are intended for Australian health professionals.