Switching treatments in psoriasis: a targeted literature review

CO132

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Findings

- Switching treatments can enhance psoriasis disease control, but it is unclear if this is because of desensitization to the initial treatment or the introduction of newer, more effective biologic treatments.
- To address this research gap, studies that swap between parallel treatment arms should be conducted to determine the most effective treatment strategy.

Introduction

Psoriasis is an immune-mediated disease manifesting in patches of inflammation on the skin.[1] Long-term treatment for the condition often requires adjustments in medication, including switching between different biologic treatments and routes of administration, to ensure a continued or sustained improvement in Psoriasis Area Severity Index (PASI) score.[1] However, it is currently unclear if some treatment-switching pathways offer better efficacy outcomes than others for patients with psoriasis.

Methods

A targeted literature review was conducted to investigate how treatment switching in psoriasis impacts treatment outcomes. The MEDLINE database was searched for randomized controlled trials between April 2014 and April 2024, in which participants with psoriasis (≥12 years old) were switched between different treatments in the following routes of administration: oral to injectable, injectable to oral, and injectable to injectable. The search strategy (Table S1 in the Supplementary Material) used a combination of keywords, such as 'moderate-to-severe psoriasis', 'plaque psoriasis', 'treatment switching' and 'switching therapies' to identify studies in which participants experienced a psoriasis treatment switch, ensuring comparable populations prior to and after treatment switching. Abstracts and full-text articles were assessed for eligibility by two reviewers, and key outcomes were extracted from the included articles. Reason for switching, PASI scores, Dermatology Life Quality Index (DLQI) scores and safety outcomes were assessed before versus after switching or continuation of the original treatment.

Objective: To evaluate clinical trial evidence for the efficacy of treatment switching in psoriasis.

Results and interpretation

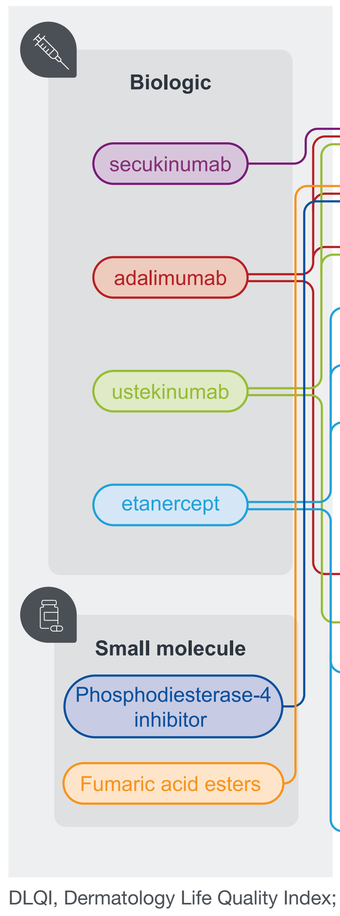
Thirteen research articles encompassing 15 trials were analysed (Figure 1).[2–14] The reason for the treatment switch was examined:

- In five trials, all participants were switched as part of the methodology[2–6]
- In five trials, all participants were re-randomized depending on severity scores[7–9]
- In five trials, all participants were switched when they experienced an inadequate response[10–14]

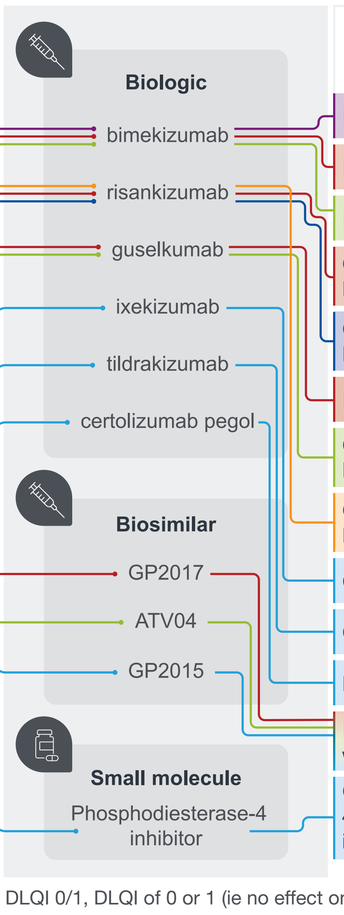
A different sequence of drugs was investigated in each trial (Figure 1). Nine trials switched between different biologic treatments,[5,7,9–13] three switched from a biologic reference product to a biosimilar,[2–4] two switched from small molecule to biologic treatments,[8,14] and one trial switched from a biologic to a small molecule treatment.[6]

Figure 1: Matrix illustrating psoriasis treatment switching and findings provided in the literature.

Findings are colour-coded, matching the colour of the treatment the participants were switched from. —• Treatment switched to



Treatment administered by injection



Treatment administered orally

Switched from	Switched to	Findings: Efficacy outcomes	Efficacy improvement when switched	DLQI score outcomes	DLQI score improvement when switched
secukinumab	bimekizumab	In PASI<90 participants (n=53), 79.0% achieved PASI≥90[7]	Yes	DLQI 0/1 increased from 51.0% to 86.0%[7]	Yes
secukinumab	risankizumab	In PASI<90 participants (n=54), 91.0% achieved PASI≥90[7]	Yes	DLQI 0/1 increased from 30.0% to 81.0%[7]	Yes
secukinumab	guselkumab	In PASI<90 participants (n=44), 90.0% achieved PASI≥90[7]	Yes	DLQI 0/1 increased from 49.0% to 81.0%[7]	Yes
adalimumab	ixekizumab	Of the PASI50 to <90 participants who switched (n=53), 66.0% achieved PASI≥90 vs 21.0% who continued adalimumab (n=56)[13]	Yes	DLQI 0/1 66.0% vs 16.0% for participants who switched and who continued adalimumab, respectively[13]	Yes
adalimumab	tildrakizumab	Of the PASI<75 participants who switched (n=83), 72.3% achieved PASI≥90 vs 2.6% who continued phosphodiesterase-4 inhibitor (n=78)[8]	Yes	DLQI 0/1 62.7% vs 9.0% for participants who switched and who continued phosphodiesterase-4 inhibitor, respectively[8]	Yes
ustekinumab	certolizumab pegol	In PASI<90 participants (n=112), 66.1% achieved PASI≥90[5]	Yes	DLQI 0/1 increased from 0% to 57.6%[5]	Yes
ustekinumab	guselkumab	Of the IGA≥2 participants who switched (n=135), 50.0% achieved PASI≥90 vs 24.0% who continued ustekinumab (n=133)[12]	Yes	DLQI 0/1 38.8% vs 19.0% for participants who switched and who continued ustekinumab, respectively[12]	Yes
etanercept	risankizumab	Of the PASI<75 participants who switched (n=20), 80.0% achieved PASI≥90 vs 50.0% who continued fumaric acid esters (n=14)[14]	Yes	DLQI 0/1 45.0% vs 29.0% for participants who switched and who continued fumaric acid esters, respectively[14]	Yes
etanercept	GP2017	Of the PASI<75 participants (n=165), 77.6% achieved PASI≥75[10]	Yes	–	*Not reported*
etanercept	ATV04	Of the PASI<75 participants (n=120), 53.8% achieved PASI≥75[11]	Yes	–	*Not reported*
etanercept	GP2015	Prior etanercept treatment did not impact certolizumab pegol efficacy[9]	Yes	–	*Not reported*
Phosphodiesterase-4 inhibitor	bimekizumab, risankizumab, guselkumab, ixekizumab, tildrakizumab, certolizumab pegol, GP2017, ATV04, GP2015	Improvements in PASI scores were comparable between participants who underwent multiple switches and those with continued treatments[2–4]	No	DLQI scores were similar between participants who were switched to biosimilars and participants who remained on biologic treatments[3,4]	No
Fumaric acid esters	Phosphodiesterase-4 inhibitor	Of switched participants (n=79), 51.9% achieved PASI≥75 vs 49.5% of participants who were treated with phosphodiesterase-4 inhibitor throughout the study (n=74)[6]	Yes	DLQI 0/1 26.0% vs 30.0% for participants who switched and who were treated with phosphodiesterase-4 inhibitor throughout the study, respectively[6]	No

DLQI, Dermatology Life Quality Index; DLQI 0/1, DLQI of 0 or 1 (ie no effect on the patient's life) out of a total score of 30; IGA, Investigator Global Assessment; n, number of participants included in the study treatment arm; PASI, Psoriasis Area Severity Index

PASI scores were used to investigate the switch efficacy. Participants who were switched due to an inadequate response generally had lower PASI scores at the data cut-off than those responding to the initial treatment. However, participants consistently benefited from switching between biologic treatments, regardless of response to the initial treatment.[5,7–14] Furthermore, switching between biologic treatments consistently improved DLQI scores.[5,7,12,13] The one biologic to small molecule switch captured (etanercept to phosphodiesterase-4 inhibitor) showed minor efficacy benefits but no DLQI improvement.[6] The improvements in PASI and DLQI scores may be attributed to treatment switches consistently being from older to newer drugs. Switching from biologic treatments to biosimilars demonstrated similar efficacy and safety outcomes to continuing biologic treatments and did not impact DLQI scores, confirming no difference between these therapies.[2–4] No switching-related safety concerns were identified in any of the studies included in this review, thus it appears that there are no large safety differences between therapies, regardless of switch methodology.

The data gathered during this review support the view that switching treatments can improve or sustain outcomes and suggests switching to newer biologic treatments could be the optimal approach for treating psoriasis. It was unclear how much outcomes could be attributed to the mode of action versus the route of administration of the treatments, as only three trials reported on such switches, while all biologic treatments were administered via injection and all small molecules were given orally. It was not possible to ascertain one optimal switching methodology or therapy due to the substantially different treatment durations before and after switching between the captured trials (Table S2 in the Supplementary Material).

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