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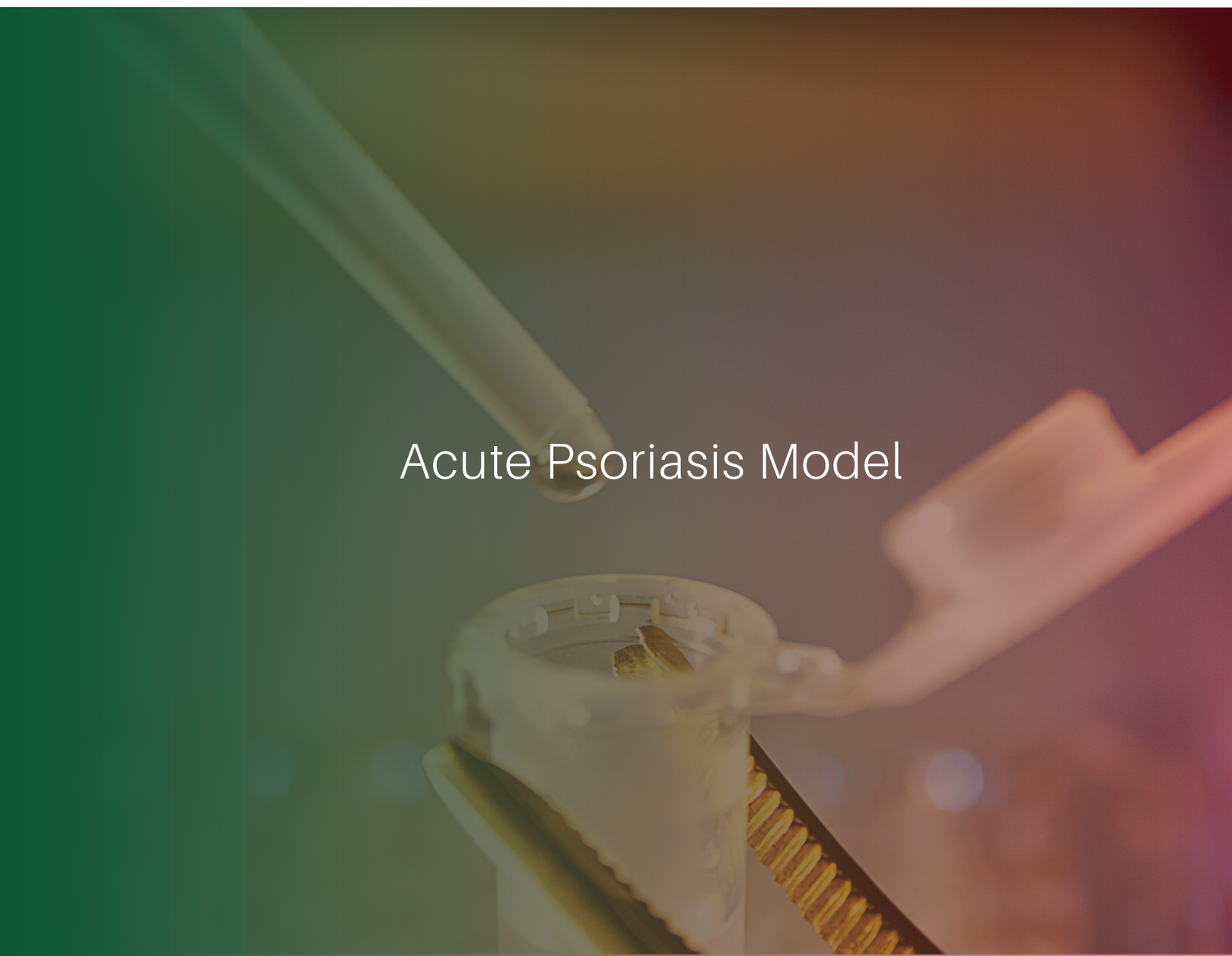
CASE STUDY



Acute Psoriasis Model

Case Study

Background

Modelling the pathophysiology of psoriasis in animal models remains challenging because psoriasis does not occur naturally in laboratory animals. However, multiple studies have shown that the IL-23 injection model is the closest to acutely mimicking the human disease[1,2].

Aim

We aim to confirm that the acute IL-23 induced psoriasis model is a suitable preclinical model to assess the inflammatory response following a putative psoriasis treatment.

Study design

	IL-23 induced model of psoriasis
Experimental model:	C57BL6/J mice (8 weeks of age)
Study duration:	2 weeks animal acclimatization, 10 days in-life phase 2-4 weeks to generate a full study report, in-life phase results provision upon study completion
Groups:	Vehicle (n=8), treatment (n=8), and positive control group (n=8)
Psoriasis induction:	Injection of recombinant IL-23 in right ear every two days for 8 days in total, PBS (control) in left ear
Dosing schedules:	Once or twice daily via oral gavage
Standard assessments:	Body weight determination, clinical observations, ear thickness (assessment of swelling), terminal bleeding
Histopathology evaluation:	Key psoriasis hallmarks to evaluate the extent and presence of acanthosis, inflammatory cell infiltration, spongiosis, perivascular lymphocyte infiltration, vesicles and pustule (Munro's abscess) formation, and hypergranulosis (H&E staining), optional IHC (e.g., FoxP3, CD4)
Complimentary readout choice:	Immune cell profiling (MSD, flow cytometry, qPCR, ELISA)

Results

Injection of IL-23 induces psoriasis-like ear dermatitis (not shown here). Ear thickness, the main readout useful to indicate swelling due to inflammation in the ear, was increased in response to IL-23 treatment (**Figure 1**).

Histopathology assessment showed epidermal thickening, proliferation and differentiation of keratinocytes, dilation of blood vessels and infiltration of inflammatory cells (**Figures 2-4**).

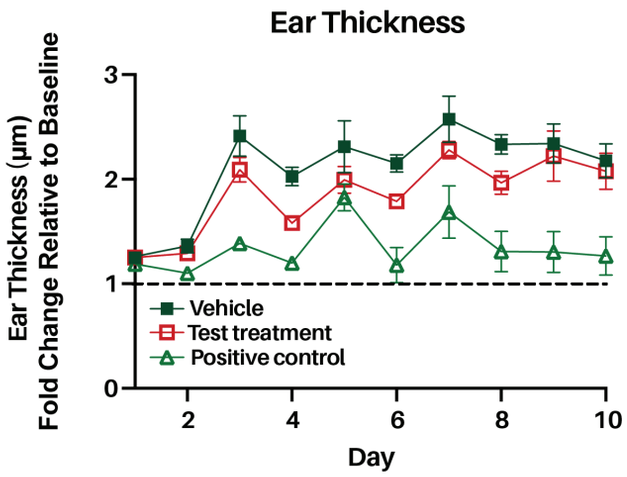


FIGURE 1. Ear thickness increases in response to IL-23 treatment. Ear thickness increased in the IL-23 control (vehicle) group following IL-23 injection. A slight decrease in ear thickness was observed on days 4 and 6 in the positive control-treated group. M ± SEM.

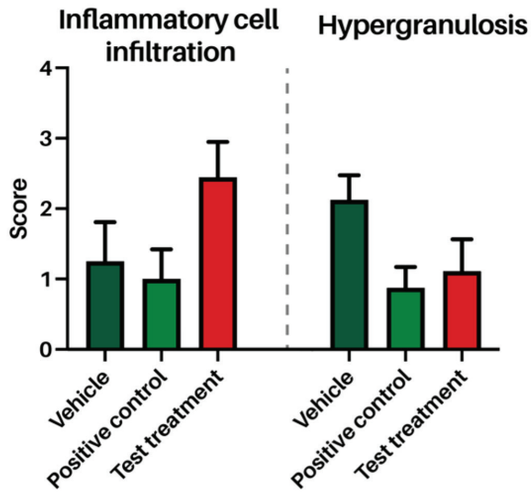


FIGURE 2. Scoring of psoriasis-like lesions reveals increase in inflammatory cell infiltration and hypergranulosis in response to IL-23 treatment. Score 0 indicates presence of such features in normal limits, while scores 2 and 3 indicate slight to moderate presence of psoriasis-like features. Data shown as M ± SEM.

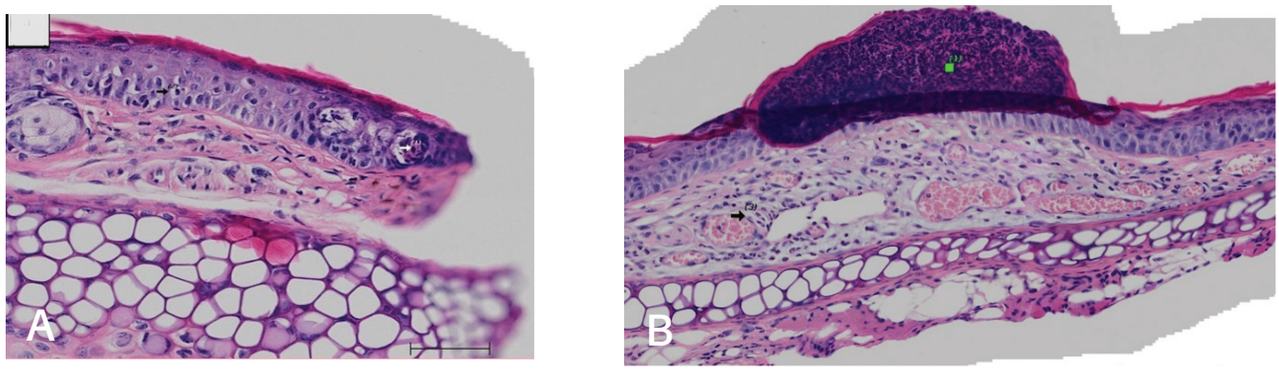


FIGURE 3. H&E-stained sections of ear skin confirm psoriasis-like lesions in the IL-23 induced model. (A) Vehicle-treated ear sections show spongiosis. **(B)** Test treatment ear sections shows Munro's abscess in the surface of epidermis.

Conclusion

In this case study we confirmed the suitability of this model, as recombinant IL-23 injection resulted in acute induction of psoriasis, confirmed by ear thickness measurement and histopathology evaluation. The complimentary readout options targeted to immune cell profiling can provide more insight into the mechanism of action of the putative treatment.

References

1. Singh TP, Zhang HH, Hwang ST, Farber JM. IL 23 and imiquimod induced models of experimental psoriasis in mice. Current protocols in immunology. 2019 Jun;125(1):e71.

2. Zheng Y, Danilenko DM, Valdez P, Kasman I, Eastham-Anderson J, Wu J, Ouyang W. Interleukin-22, a TH17 cytokine, mediates IL-23-induced dermal inflammation and acanthosis. Nature. 2007 Feb 8;445(7128):648-51.

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