# Galvokimig, a multispecific, antibody-based IL-13, IL-17A, and IL-17F inhibitor, modulates lesional transcriptome and circulating biomarkers in atopic dermatitis: findings from a 12-week Phase 2a trial

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## Objective

**Report the transcriptomic signature of tissue and levels of select serum biomarkers from patients with moderate-to-severe AD at baseline and treated with galvokimig in the first-in-human proof-of-concept (POC) Phase 2a study.**

## Background

- Inflammatory pathways beyond Th2 play key roles in AD[1-3]
- The synergistic effects of IL-13, IL-17A, and IL-17F have been shown to amplify downstream inflammatory pathways and markers associated with AD pathobiology, including IL-19,[3] a proinflammatory cytokine correlated with AD severity[4]
- Galvokimig is an Fc-free, multispecific, antibody-based therapeutic that inhibits IL-13, IL-17A, and IL-17F, with albumin binding to modulate half-life[3,5]
- In a Phase 2a trial (NCT04643457) of 47 participants with moderate-to-severe AD, the Bayesian-adjusted EASI75 response rate was 64.9% with galvokimig vs 12.3% with placebo[6]
  - Bayesian-adjusted EASI90 response rate was 46.6% with galvokimig vs 3.5% with placebo
- Here, we report cutaneous lesional transcriptome profiles and circulating IL-19 serum levels from participants in the Phase 2a trial to evaluate the molecular effects of galvokimig

## Methods

- Skin biopsies from the Phase 2a study were transcriptomically profiled to evaluate the molecular effects of galvokimig
  - At baseline, 47 lesional skin biopsies (galvokimig arm: 33 participants; placebo arm: 14 participants) and 46 non-lesional skin biopsies were collected
  - At week 12 after treatment initiation, lesional skin biopsies were collected from 31 participants in the galvokimig arm and 13 participants in the placebo arm
  - Bulk mRNA sequencing of lesional and non-lesional biopsies was performed
  - PC analysis was performed on all samples
  - Gene dysregulation at baseline and changes post-treatment were assessed using the *limma* framework[7]
  - MetaBase, Gene Ontology, and GeneRIF were used to analyse Th17-/Th1-/Th2-relevant gene sets[8-10]
  - Normalization of gene expression was defined as percent improvement >75%
- Serum samples (galvokimig, n=30; placebo, n=14) were collected at baseline and at weeks 2 and 12 post-treatment
  - Circulating IL-19 and TARC levels in serum were quantified using a validated ligand-binding immunoassay

## Results

### *AD lesional skin showed marked dysregulation compared with non-lesional skin at baseline, with week 12 galvokimig-treated samples returning to a non-lesional–like state*

- At baseline, there were marked differences between lesional and non-lesional skin (**Figure 1A**)
- Treatment with galvokimig attenuated this dysregulation by week 12 (**Figure 1B**)

## Conclusions

**Dysregulation of multiple inflammatory pathways supports the therapeutic potential of simultaneously targeting IL-17A, IL-17F, and IL-13 in AD. Treatment with galvokimig normalized inflammatory pathways beyond Th2 in people with AD.**

### PossibILity: a Phase 2b dose-ranging clinical trial[11]

This ongoing trial will yield further information on the efficacy, safety, and pharmacokinetics of galvokimig in participants with moderate-to-severe AD (NCT07277660)

## Key takeaways

**Th2/Th1** immune signatures and *IL13* expression signatures are **upregulated** in AD lesional vs non-lesional skin

The **Th17 inflammatory pathway,** including expression of *IL17A* and *IL17F*, is also **upregulated**

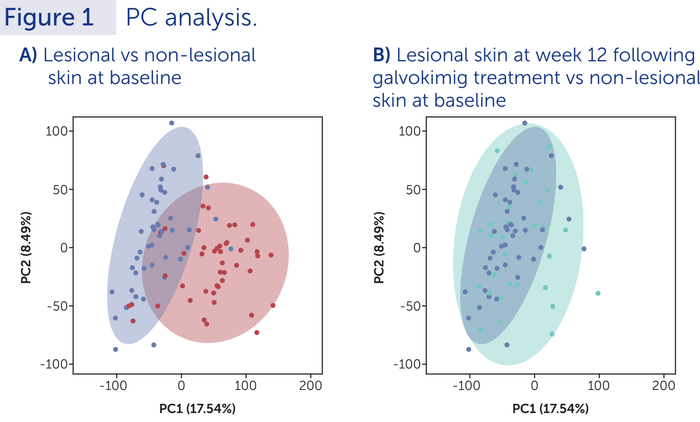
**Galvokimig reduced *IL17A*, *IL17F*, and *IL13* expression and attenuated Th17/Th2/Th1-mediated inflammatory gene expression** compared with lesional skin at baseline

**Galvokimig significantly downregulated serum IL-19,** a biomarker of combined Th2/Th17 biology, as early as week 2 after treatment initiation ($P$=0.0008), and TARC, a validated marker of severity and Type 2 inflammation ($P$=0.0082; week 12)

×IL-17A
×IL-17F
×IL-13

These findings support the **therapeutic potential of simultaneously targeting IL-17A, IL-17F, and IL-13 in AD**[6]

**Figure 1** PC analysis.

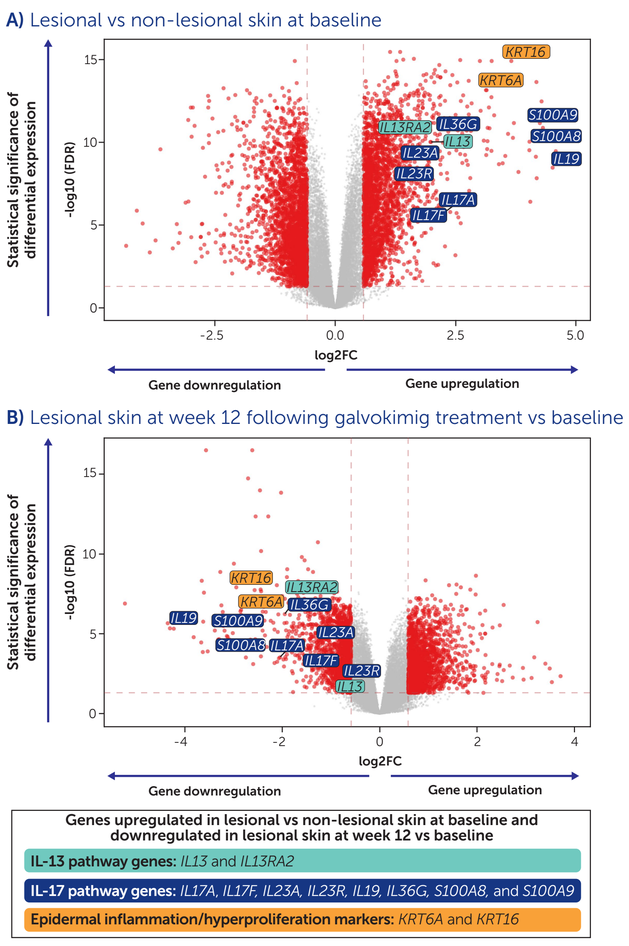



(**A**) Distinct transcriptomic profiles between lesional and non-lesional skin at baseline. (**B**) Attenuated gene dysregulation in AD lesional skin with galvokimig by week 12.

### *Galvokimig treatment reduced the expression of key genes associated with Th2 and Th17 immunity that were upregulated in AD lesional skin at baseline*

- Consistent with known mechanisms of AD pathobiology,[12] key genes associated with Th2 and Th17 immunity were upregulated in lesional vs non-lesional skin at baseline prior to treatment with galvokimig (**Figure 2A**)
- Additionally, genes associated with epidermal inflammation were upregulated
- Galvokimig treatment reduced the expression of key genes associated with Th2 and Th17 immunity and epidermal inflammation at week 12 (**Figure 2B**)

**Figure 2** Differential gene expression in the transcriptome of AD skin.

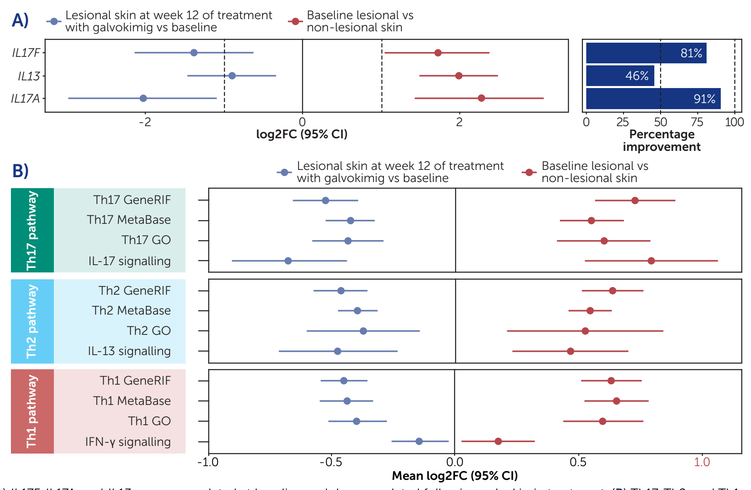



(**A**) Genes associated with Th17/Th2 inflammation and skin barrier integrity were differentially expressed (defined as having an absolute log2FC ≥1 and an FDR-adjusted $P$-value <0.05) in AD lesional (n=47) vs non-lesional (n=46) skin. (**B**) Genes associated with Th17/Th2 inflammation and skin barrier integrity were attenuated in AD lesional skin at week 12 following galvokimig treatment (n=31) vs baseline (n=47).

### *Dysregulation of IL-17A and IL-17F expression, and of Th17, Th2, and Th1 pathway signalling, in AD lesional skin was attenuated with galvokimig*

- *IL17A*, *IL17F*, and *IL13* expression (**Figure 3A**), as well as Th17/Th2/Th1 immune pathways (**Figure 3B**), were dysregulated at baseline
- At week 12, galvokimig normalized the expression of inflammatory genes *IL17A* and *IL17F* (percent improvement >75%), reduced the expression of *IL13*, and attenuated downstream Th2/Th17 pathways
- Additionally, galvokimig attenuated the Th1 immune pathway

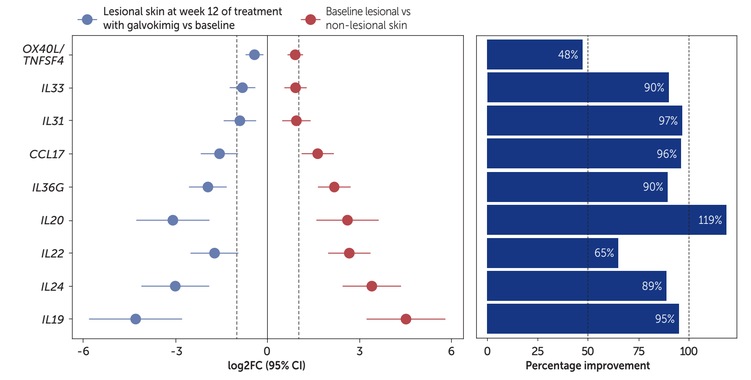
**Figure 3** *IL17A*, *IL17F*, and *IL13* expression and Th17/Th2/Th1 immune pathway signalling in AD skin.




(**A**) *IL17F*, *IL17A*, and *IL13* were upregulated at baseline and downregulated following galvokimig treatment. (**B**) Th17, Th2, and Th1 pathways were upregulated in AD lesional skin at baseline and downregulated following galvokimig treatment.

### *Galvokimig reduced the expression of additional genes associated with AD pathophysiology (Figure 4)*

**Figure 4** Expression of additional genes associated with AD pathophysiology.

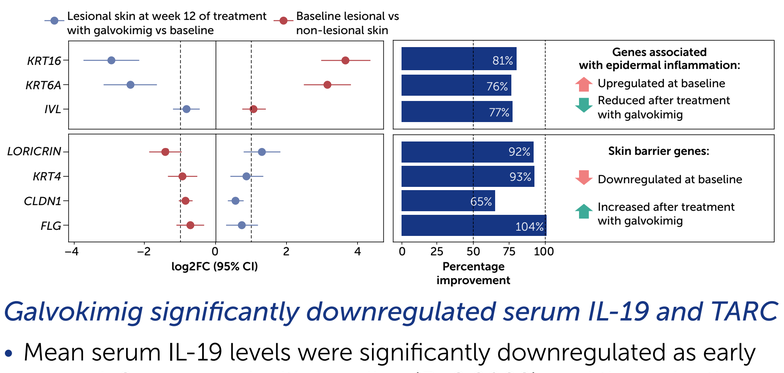



Genes associated with AD inflammation were upregulated at baseline and downregulated following treatment with galvokimig.

### *Galvokimig downregulated epidermal inflammation/hyperproliferation genes and increased the expression of skin barrier integrity genes*

- Skin barrier integrity and epidermal inflammatory genes had aberrant expression at baseline (**Figure 5**)
- Galvokimig normalized the expression (defined as percent improvement >75%) of *KRT16*, *KRT6A*, *IVL*, *LORICRIN*, *KRT4*, and *FLG*

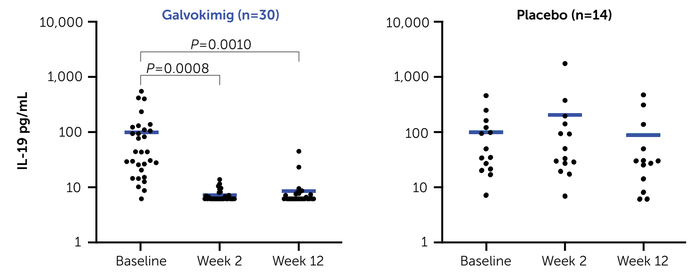
**Figure 5** Genes associated with epidermal inflammation and skin barrier integrity at baseline and after galvokimig treatment.




### *Galvokimig significantly downregulated serum IL-19 and TARC*

- Mean serum IL-19 levels were significantly downregulated as early as week 2 compared with baseline ($P$=0.0008), and the reduction was maintained up to week 12 ($P$=0.0010) (**Figure 6**)

**Figure 6** Serum IL-19 levels in participants treated with galvokimig vs placebo.




Baseline was defined as the day prior to treatment initiation. Data are presented as a scatter plot with the geometric mean indicated by the horizontal line.

- Reduction in mean serum TARC levels was also observed ($P$=0.0082; week 12) (data not shown)

Galvokimig is an investigational biologic currently in clinical development. The safety profile and efficacy have not been established, and it is not approved by any health authority worldwide.

**AD:** atopic dermatitis; **CI:** confidence interval; **EASI75/90:** ≥75%/≥90% reduction in Eczema Area and Severity Index score; **Fc:** fragment crystallizable; **FDR:** false discovery rate; **GeneRIF:** Gene Reference Into Function; **GO:** Gene Ontology; **IFN-γ:** interferon gamma; **IL:** interleukin; **log2FC:** logarithmic base 2 fold change; **PC:** principal component; **POC:** proof-of-concept; **TARC:** thymus and activation-regulated chemokine; **Th:** T helper.

**References:** [1]Savva M *et al. Front Biosci (Landmark Ed).* 2024;29:84; [2]Schuler CF 4th *et al. J Allergy Clin Immunol.* 2023;151:1145-54; [3]Stanyon S et al. Poster presented at ISDS 2025; 12-15 November 2025; New York, USA. Poster 246; [4]Konrad RJ *et al. Sci Rep.* 2019;9:5211; [5]Davé E *et al. MAbs.* 2023;15:2160229; [6]Hunter H *et al.* Abstract presented at ISIS 2025; 12-15 November 2025; New York, USA; [7]Ritchie ME *et al. Nucleic Acids Res.* 2015;43:e47; [8]Clarivate. MetaBase. 2026. Available at: https://clarivate.com/life-sciences-healthcare/research-development/discovery-development/early-research-intelligence-solutions [accessed 17 July 2026]; [9]Ashburner M *et al. Nat Genet.* 2000;25:25-9; [10]Jimeno-Yepes AJ *et al. BMC Bioinformatics.* 2013;14:171; [11]UCB Biopharma SRL. A dose-ranging study to evaluate the efficacy, safety, pharmacokinetics, and pharmacodynamics of galvokimig in adult study participants with atopic dermatitis. 2026. Available at: https://clinicaltrials.gov/study/NCT07277660 [accessed 9 June 2026]; [12]Bieber T *et al. J Eur Acad Dermatol Venereol.* 2022;36:1432-49.

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