Real life efficacy and systemic immune modulation upon tralokinumab treatment in atopic dermatitis

Authors: Meslina Almaci, Philipp Globig, Davender Redhu, Margitta Worm*.

Affiliation: Division of Allergy and Immunology, Department of Dermatology, Venerology and Allergy, Charité Universitätsmedizin Berlin, Germany.

Learning Objective: Understand how IL-13 blockade with tralokinumab impacts peripheral T-cell phenotypes and clinical outcomes in AD.

Take away: Tralokinumab shows real-world efficacy with measurable systemic immunomodulation; a minimal peripheral panel may help evaluate response trajectories.

Conflict of Interest: AbbVie Deutschland GmbH & Co. KG, Aimmune Therapeutics UK Limited, ALK-Abelló Arzneimittel GmbH, Allergopharma GmbH & Co KG, Almirall Hermal GmbH, Amgen GmbH, AstraZenceca GmbH, Bayer AG, Bencard Allergy GmbH, Bioprojet Pharma, Boehringer Ingelheim Pharma GmbH &Co.KG, Bristol Myers Squibb GmbH & Co. KGaA, Galderma Laboratorium GmbH, Glaxo Smith Kline GmbH & Co. KG, Infectopharm Arzneimittel und Consilium GmbH, LEO Pharma GmbH, Lilly Deutschland GmbH, Mylan Germany GmbH (A Viatris Company), Novartis AG, Octapharm AG, Pfizer Pharma GmbH, Sanofi-Aventis Deutschland GmbH/Genzyme Europe B. B. and Stallergenes GmbH.

Contact: Margitta Worm , margitta.worm@charite.de

ISAD — 24 October 2025, Melbourne, Australia

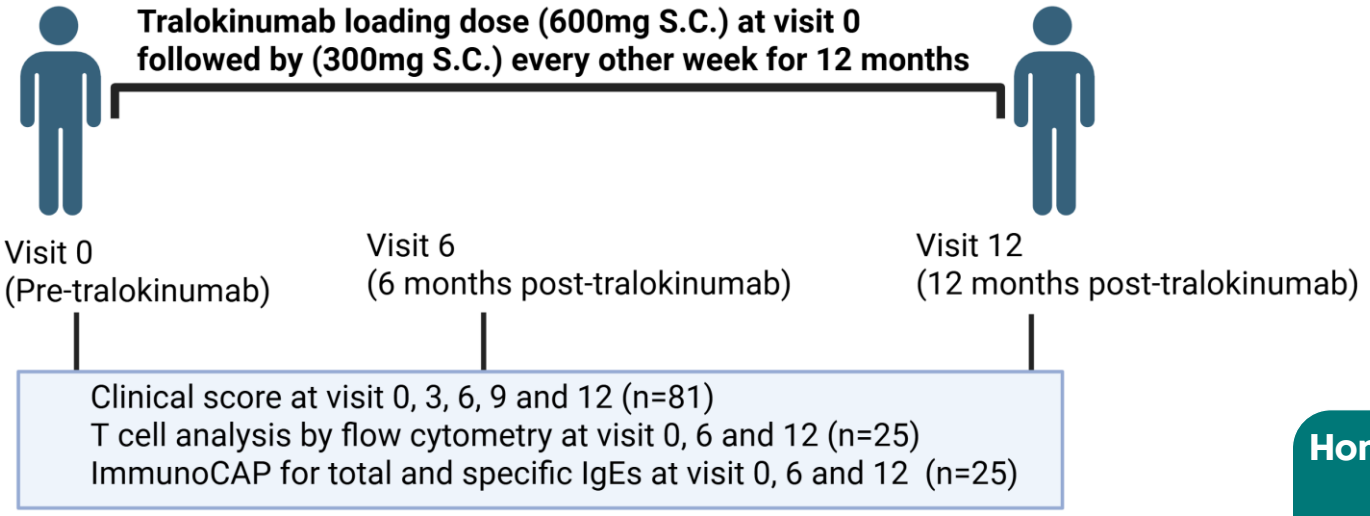
Background

- IL-13 is a key effector cytokine in AD; tralokinumab selectively blocks IL-13.
- Clinical response varies; practical, blood-based markers to anticipate response are limited.

Hypothesis

- Change of systemic T-cell immunophenotypes (CLA+, OX40+, Th2/IL-4+, IL-13+, Th1/IFNγ+, Th17/IL-17+ and Treg/IL-10+ T cells) upon treatment with tralokinumab.

Study design and T cell phenotyping



T-cell panel

Lineage
- CD3
- CD4 / CD8

Polarization
- CXCR3 — Th1-like
- CCR6 — Th17-like
- CXCR5 — TFH

Activation
- OX40 (CD134)
- CD25 (IL-2Rα)
- CD127 (IL-7Rα)

Cytokines
- IFNγ (Th1)
- IL-13, IL-4 (Th2)
- IL-17A (Th17)
- IL-10 (regulatory/Tr1)
- IL-9 (Th9)
- IL-2

Homing / Compartment
- CLA — skin-homing
- CXCR5 — TFH-like
- CCR7 — LN-homing

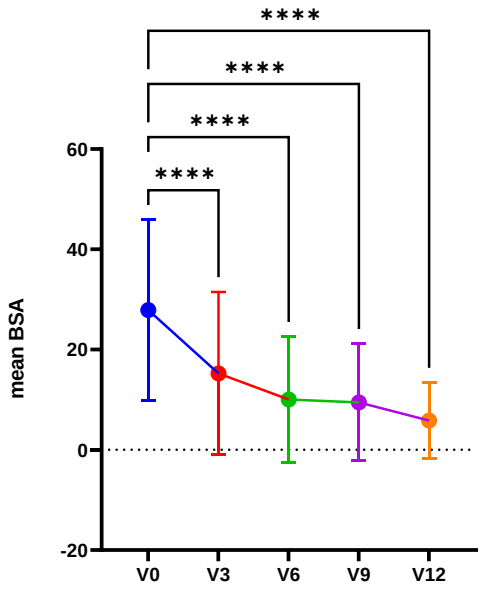
Differentiation
- CD45RA × CCR7

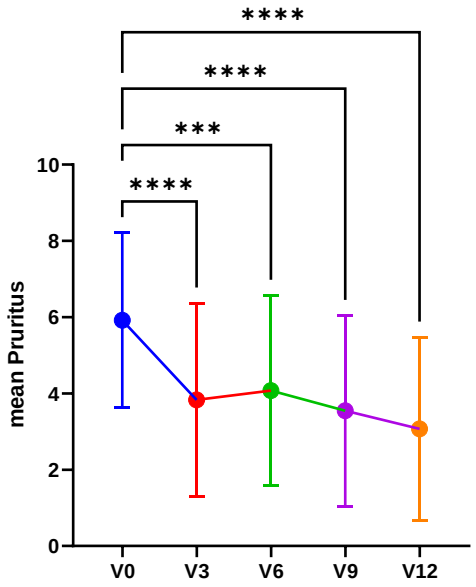
→ Naïve / TCM / TEM / TEMRA

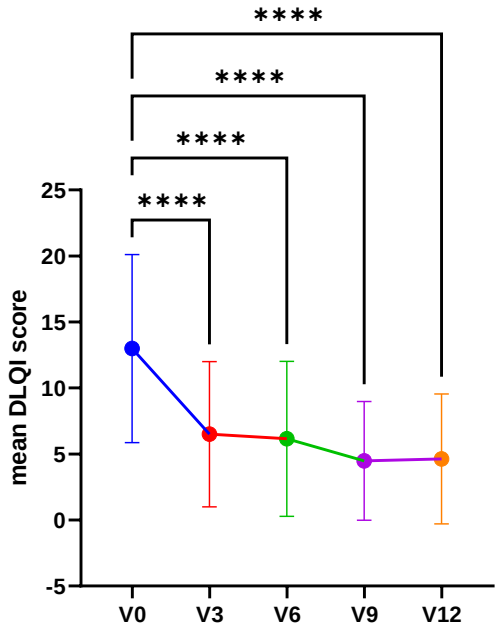
Treg
- CD25^hi CD127^low on CD4+

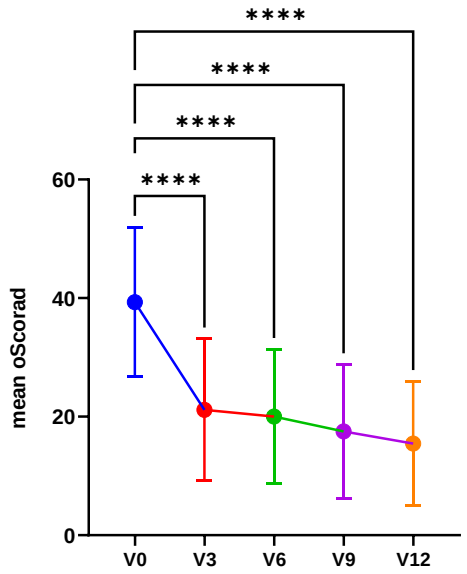
→ Treg-enriched

Clinical response of tralokinumab treated patients

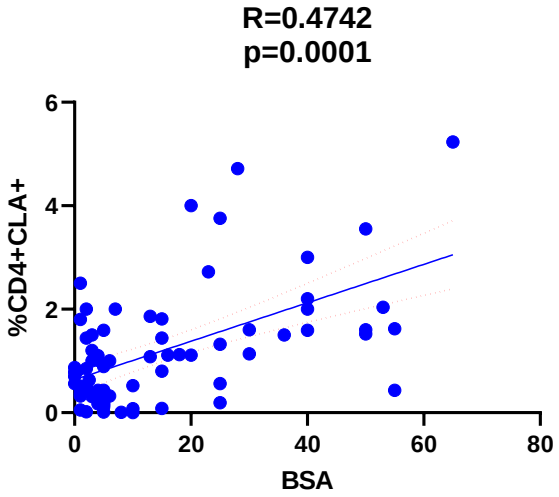


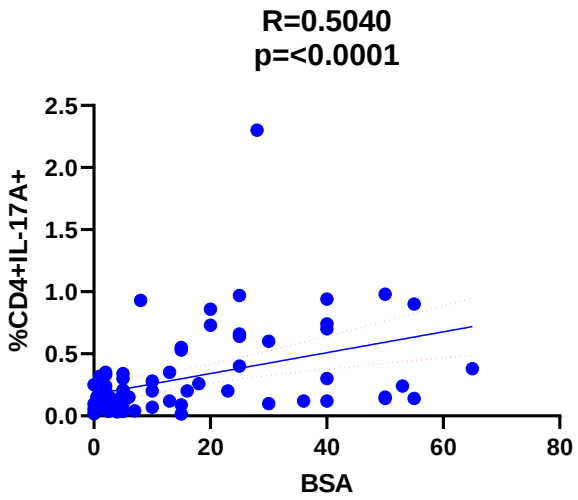


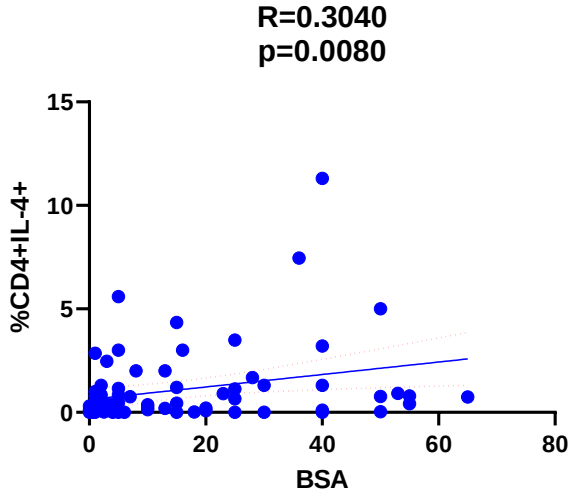


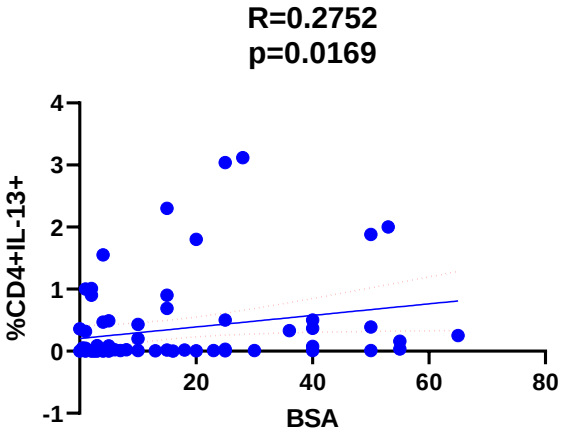


Association of T cell immune cell subsets with AD severity



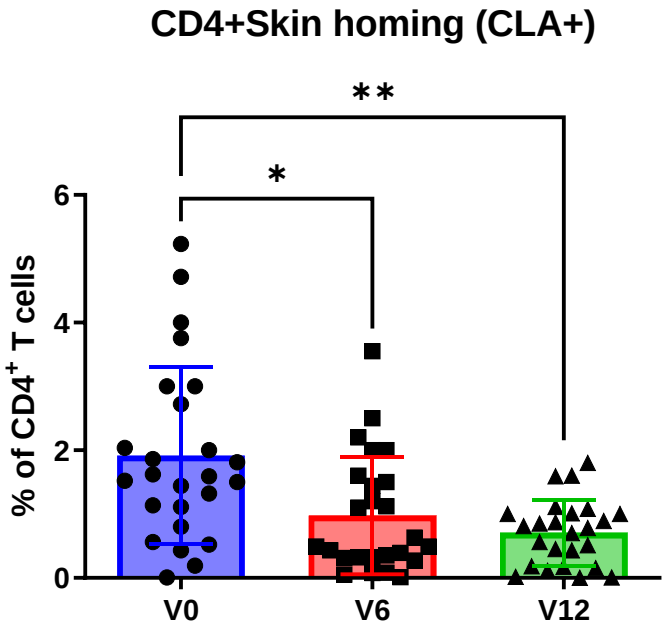


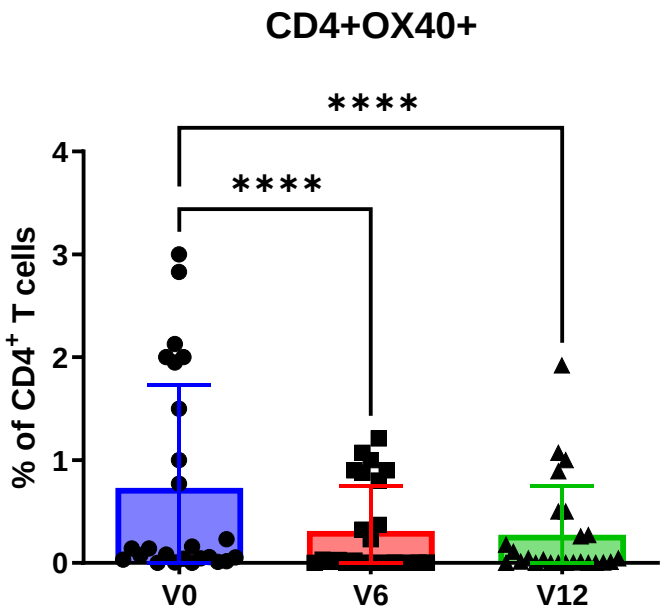




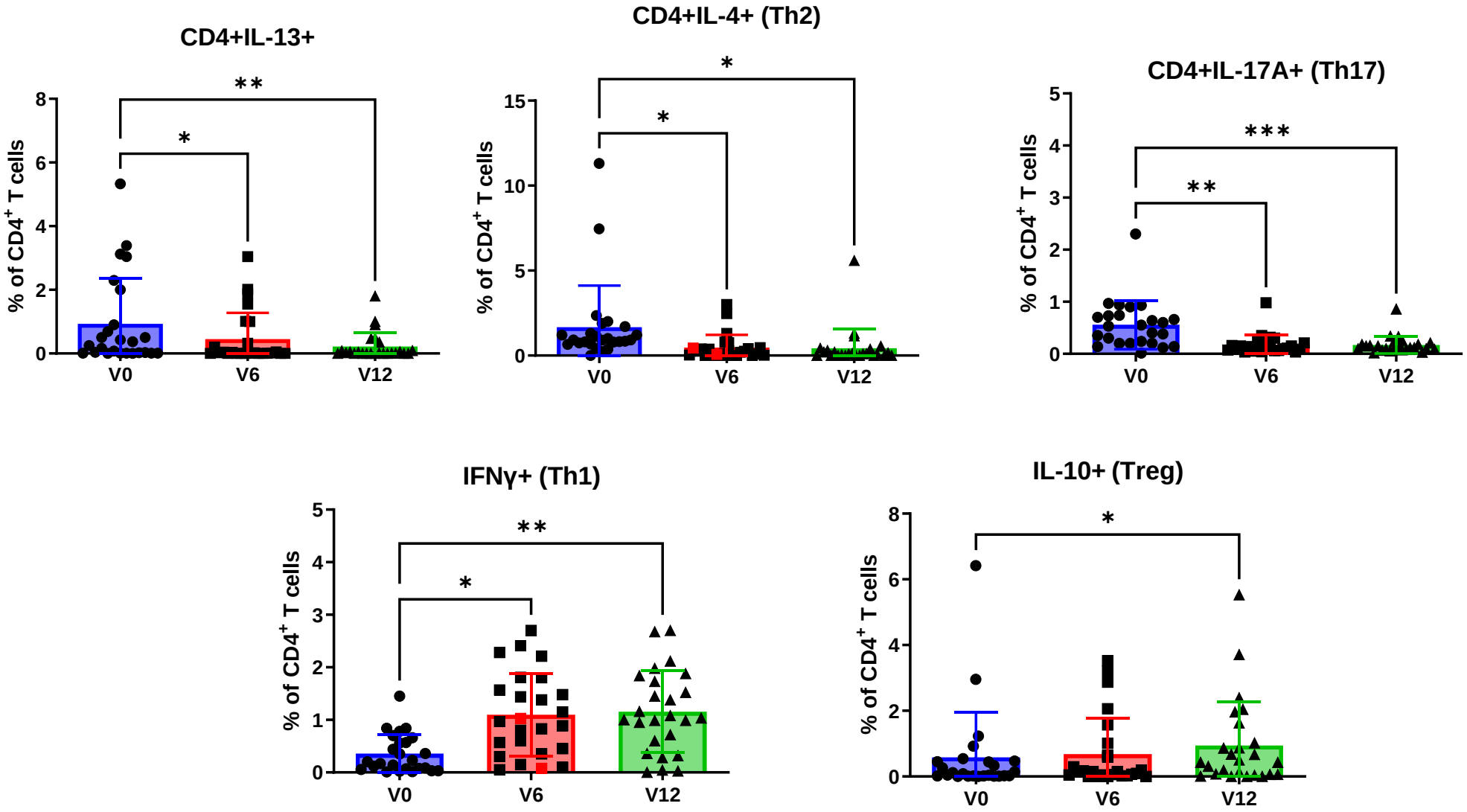
CHARITÉ

Reduced frequencies of CLA and OX40 positive CD4+ cell subsets over 12 months of treatment





Course of Cytokine-producing T cell subsets during treatment



Summary and Conclusions

- Tralokinumab induced clinical improvement over 12 months
- CLA+, IL-17A+, IL-4+ and IL-13+ T cell subsets positively associated with AD severity
- Systemic skin-homing (CLA+) and activated (OX40+) T cells decreased over time
- CD4 cytokine profiles shifted from Th2/Th17 (IL-13/IL-4/IL-17A) toward regulatory/Th1 (IL-10/IFNγ)

Systemic anti-T2-inflammatory immune modulation


EDMUND LESSER-HAUS
Klinik für Dermatologie,
Venerologie und Allergologie