BROADEN STUDY

KT-621, Oral STAT6 Degrader, Phase 1b Results

KYMERA

Agenda

Introduction
Justine Koenigsberg
Vice President, Investor Relations

Revolutionizing Immunology with Oral Medicines
Nello Mainolfi, PhD
Founder, President and Chief Executive Officer

KT-621 BroADen Phase 1b Data
Jared Gollob, MD
Chief Medical Officer

Closing Remarks
Nello Mainolfi, PhD
Founder, President and Chief Executive Officer

Question and Answer Session

Forward Looking Statements

This presentation contains forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995 and other federal securities laws. These statements include, but are not limited to, implied and express statements about our strategy, business plans and objectives for our programs; plans and timelines for the preclinical and clinical development of our product candidates, including the therapeutic potential, clinical benefits and safety profiles of such product candidates; expectations regarding timing, success and data announcements of ongoing preclinical studies and clinical trials; our ability to initiate new clinical programs, including plans to submit investigational new drug (IND) applications; our ability to deliver additional investigational drugs into the clinic; the initiation, timing, progress and results of our current and future preclinical studies and clinical trials of our current and prospective product candidates; our plans to develop and commercialize our current and any future product candidates and the implementation of our business model and strategic plans for our business, current; any future product candidates; our expectations regarding strategy, business plans and objectives on the development of KT-621, including the therapeutic potential, clinical benefits and safety thereof, the initiation of the Phase 2b study of KT-621 in patients with asthma in the first quarter of 2026, the effect of initial parallel development of Phase 2b studies in AD and asthma patients on acceleration of late parallel development and dose selection across multiple indications, Phase 2b data readout of KT-621 in patients with moderate to severe AD expected by mid-2027; and the preliminary cross-study assessments comparing non-head-to-head clinical data of KT-621 to published data for dupilumab, and our financial condition and expected cash runway into the second half of 2028. All statements other than statements of historical facts contained in this presentation, including express or implied statements regarding our strategy, future financial condition, future operations, projected costs, prospects, plans, objectives of management and expected market growth, are forward-looking statements. In some cases, you can identify forward-looking statements by terminology such as "anticipate," "upcoming," "assume," "believe," "could," "estimate," "expect," "goal," "intend," "may," "milestones," "objective," "plan," "predict," "potential," "seek," "should," "target," "will," "would" and other similar expressions that are predictions of or indicate future events and future trends, or the negative of these terms or other comparable terminology. We may not actually achieve the plans, intentions or expectations disclosed in our forward-looking statements, and you should not place undue reliance on our forward-looking statements. You should not rely upon forward-looking statements as predictions of future events and actual results or events could differ materially from the plans, intentions and expectations disclosed herein.

Any forward-looking statements are based on management's current expectations and beliefs and are subject to a number of risks, uncertainties and important factors that may cause actual events or results to differ materially from those expressed or implied by any forward-looking statements including, without limitation, risks associated with: the timing and anticipated results of our current and future preclinical studies and clinical trials, supply chain, strategy and future operations; the delay of any current and future preclinical studies or clinical trials or the development of our drug candidates; the risk that the results of prior preclinical studies and clinical trials may not be predictive of future results in connection with current or future preclinical studies and clinical trials, including those for KT-621; the risk that cross-trial comparisons my not be reliable as no head-to-head trials have been completed KT-621 to dupilumab, and Phase 1 clinical data for KT-621 may not be directly comparable to dupilumab's clinical data due to differences in molecule composition, trial protocols, dosing regiments, and patient populations and characteristics. our ability to successfully demonstrate the safety and efficacy of our drug candidates; the timing and outcome of any interactions with regulatory authorities; obtaining, maintaining and protecting our intellectual property; our relationships with existing and future collaboration partners; the impacts of current macroeconomic and geopolitical events. In addition, any forward-looking statements represent Kymera's views only as of today and should not be relied upon as representing its views as of any subsequent date. Kymera explicitly disclaims any obligation to update any forward-looking statements, except as required by law. No representations or warranties (expressed or implied) are made about the accuracy of any such forward-looking statements. As a result of these risks and others, including those set forth in our filings with the SEC, actual results could vary significantly from those anticipated in this presentation, and our financial condition and results of operations could be materially adversely affected.

Certain information contained in this presentation and statements made orally during this presentation relate to or is based on studies, publications, surveys and other data obtained from third-party sources and the Company's own internal estimates and research. While the Company believes these third-party studies, publications, surveys and other data to be reliable as of the date of the presentation, it has not independently verified, and makes no representation as to the adequacy, fairness, accuracy or completeness of, any information obtained from third-party sources. In addition, no independent sources have evaluated the reasonableness or accuracy of the Company's internal estimates or research, and no reliance should be made on any information or statements made in this presentation relating to or based on such internal estimates and research. This presentation contains trademarks, trade names and service marks of other companies, which are the property of their respective owners.

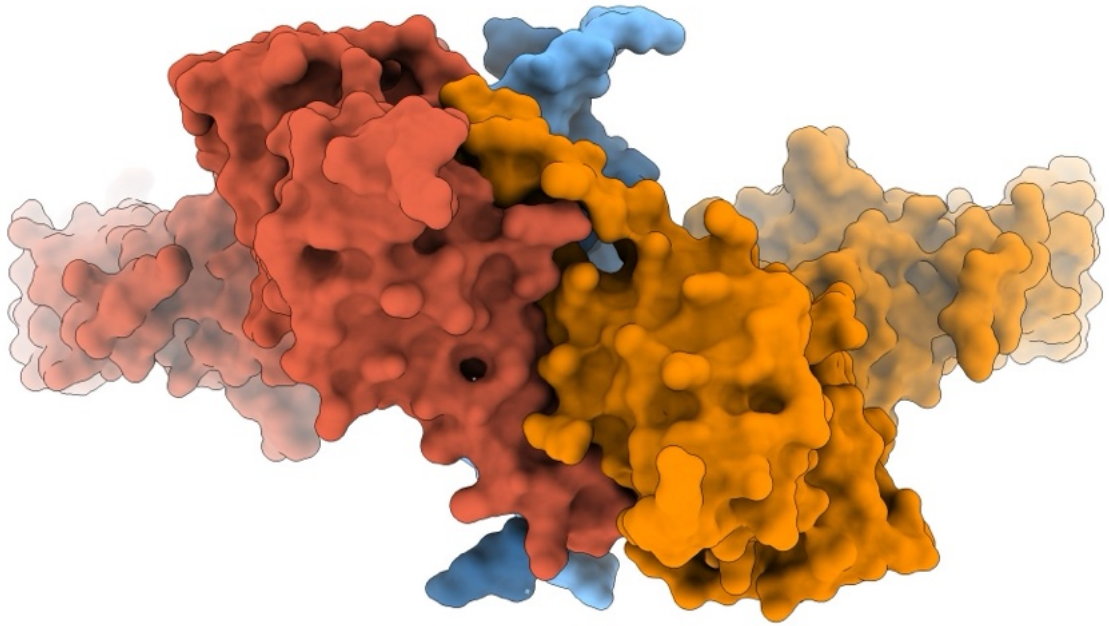
Revolutionizing Immunology with Oral Medicines

Nello Mainolfi, PhD

Founder, President and CEO

Kymera: Revolutionizing Immunology with Oral Medicines

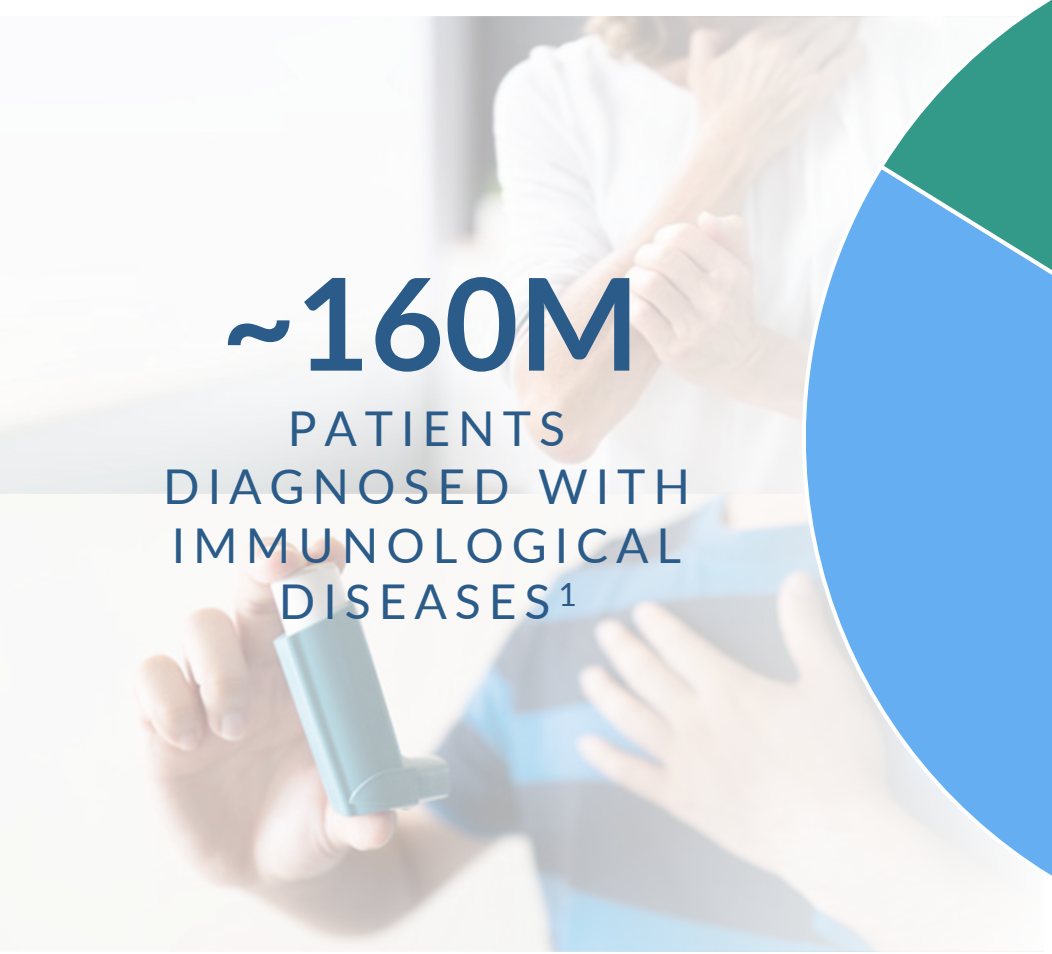
- Leader in Targeted Protein Degradation (TPD)
- Unique target selection strategy pursuing traditionally undrugged targets in highly validated pathways
- Best-in-industry capabilities in hit finding and optimization of oral degraders



Strategically focused on immunology and delivering disruptive oral therapies with biologics-like profiles to transform treatment paradigms

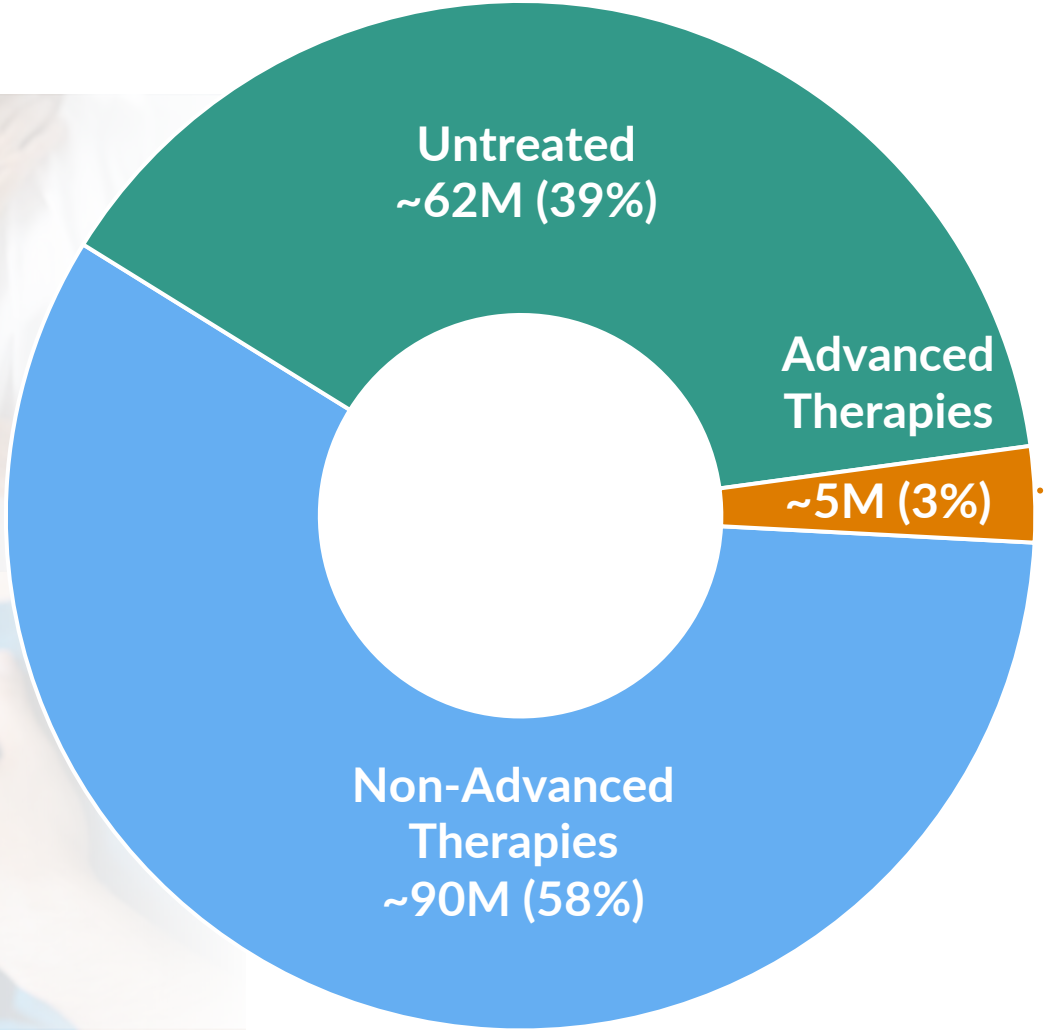
Immunology Remains a Large Underserved Market

Millions of Patients Do Not Have Access to Advanced Systemic Therapies



~160M

PATIENTS DIAGNOSED WITH IMMUNOLOGICAL DISEASES[1]



>$100B

IN ANNUAL SALES FOR ADVANCED THERAPIES[2]

[1]Diagnosed prevalent patient population for US/EU5/JP (GlobalData; 2023); key immunologic diseases includes AD, Asthma, CD, COPD, HS, MS, PsA, PsO, RA, SLE, UC;
[2]Market Forecasts for US/EU5/JP (GlobalData; 2023).

Advanced Systemic Therapies Are Mostly Injectable Biologics

Biologics Have Numerous Limitations, Making Orals Preferred by Most Patients

DUPIXENT® (dupilumab) Injection

Skyrizi™ risankizumab-rzaa

VYVGART® (efgartigimod alfa-fcab)

- Inconvenient, burdensome, and often painful for patients
- Immunogenicity risks
- In-office injection training costs HCPs valuable time and resources
- Expensive to manufacture; cold chain delivery/storage

>90%

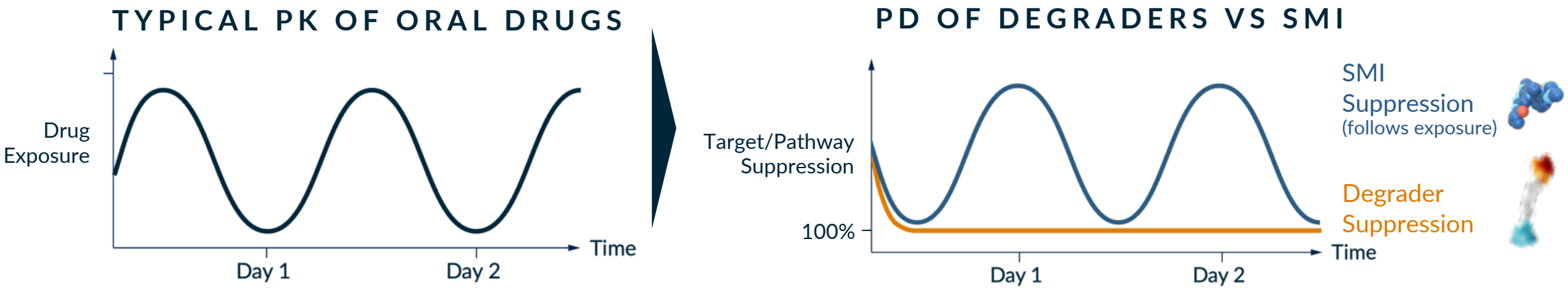
WOULD SWITCH TO AN ORAL OPTION[1]

[1]Stein Gold et al, Fall Clinical Dermatology Conference, 2025. All trademarks and registered trademarks are the property of their respective owners.

Small Molecule Oral Degraders Can Transform Treatment of Diseases

Oral Drugs with Biologics-like Activity

Unlike Traditional Small Molecule Inhibitors (SMI), Degraders Allow for Continuous, Complete Pathway Blockade



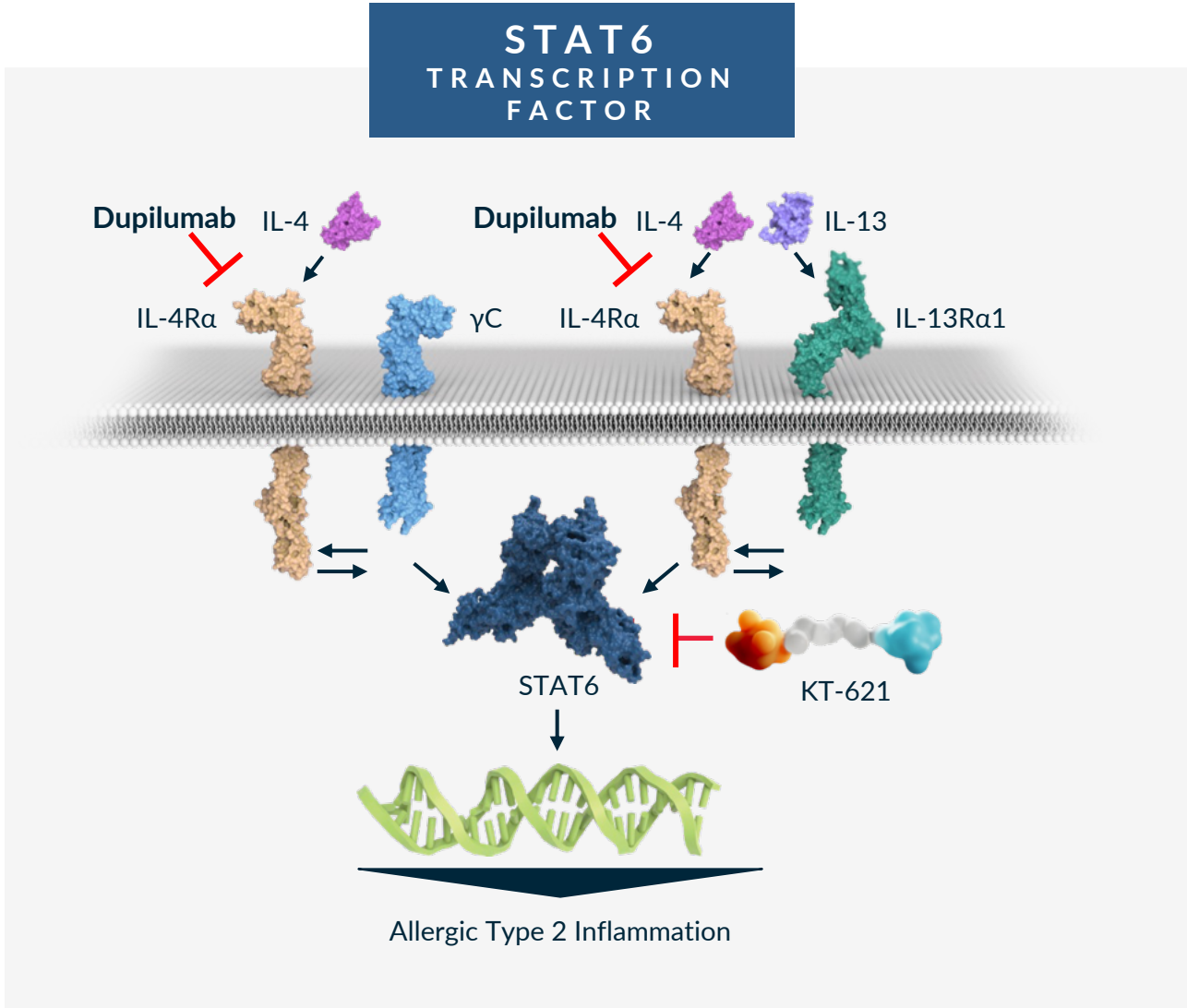
SMI pharmacology (**PD**) is **correlated** to drug exposure (**PK**); **Degraders PD is not correlated to PK** and can lead to complete continuous degradation with its catalytic mechanism[1]

- **Unlocks target classes** unreachable by other modalities
- **High selectivity, strong potency, and catalytic mechanism** enable full and constant target suppression with low doses/concentrations
- **Achieves full pathway blockade**, matching biologics-like depth of immune modulation
- Potential to deliver the **power of biologics in an oral form**

[1]Mainolfi et al, Annual Reports in Medchem, 2017; PD: Pharmacodynamics; PK: Pharmacokinetics; SMI: Small Molecule Inhibitors.

STAT6 Transcription Factor, Highly Validated but Undrugged Target

- STAT6 is the specific transcription factor in the IL-4/IL-13 pathway
- IL-4/IL-13 pathway is clinically validated by dupilumab across multiple Type 2 allergic and atopic diseases:
  - AD, asthma, COPD, EoE, CRSwNP, BP, PN, CSU
- STAT6 is genetically validated by human GoF and heterozygous LoF alleles
- While several therapies target the upstream IL-4/IL-13 receptors, there are no known drugs that selectively target this pathway within the cell with oral delivery potential



Sharma et al, 2023; Takeda et al, 1996; Kaplan et al, 1996; Junttila, 2018; Kolkhir et al, 2023; AD: Atopic Dermatitis; COPD: Chronic Obstructive Pulmonary Disease; EoE: Eosinophilic Esophagitis; CRSwNP: Chronic Rhinosinusitis with Nasal Polyps; BP: Bullous Pemphigoid; PN: Prurigo Nodularis; CSU: Chronic Spontaneous Urticaria; GoF: Gain of Function; LoF: Loss of function.

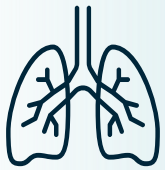
STAT6 Opportunity to Serve Millions of Patients with Type 2 Inflammation

>140M

TOTAL POTENTIAL PATIENT IMPACT[1]

DERMATOLOGY	PATIENTS[1]
AD	43M
CSU	4.4M
PN	305K
BP	166K

GASTROENTEROLOGY	
EoE	1.6M



RESPIRATORY	
Asthma	55M
COPD	34M
CRSwNP	2.8M

<2M

TREATED WITH SYSTEMIC ADVANCED THERAPIES[2]

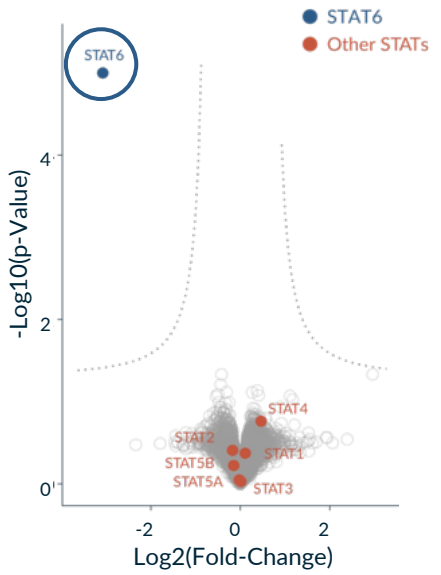
An oral STAT6 degrader has the potential to transform the treatment paradigm for Type 2 diseases and tap into large underserved markets

[1]Diagnosed prevalent patient population for US/EU5/JP (GlobalData; 2023); [2]Estimate based on GlobalData 2023 market forecasts for AD, Asthma, and COPD in US/EU5/JP then extrapolated to remaining Type 2 indications; AD: Atopic Dermatitis; COPD: Chronic Obstructive Pulmonary Disease; EoE: Eosinophilic Esophagitis; CRSwNP: Chronic Rhinosinusitis with Nasal Polyps; BP: Bullous Pemphigoid; PN: Prurigo Nodularis; CSU: Chronic Spontaneous Urticaria.

KT-621: Compelling Preclinical Profile

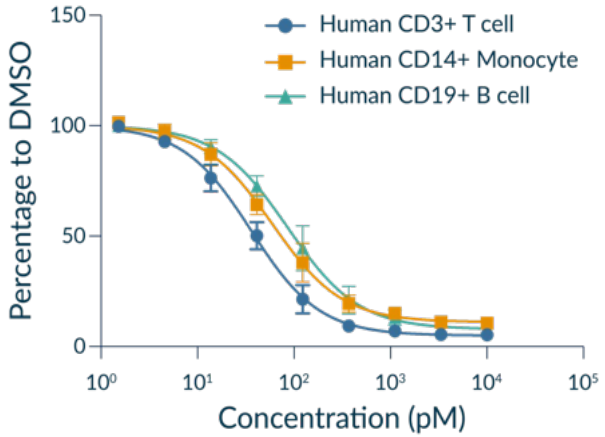
Preclinical Package Suggests Potential for Dupilumab-like Activity in a Pill

Exquisite Selectivity



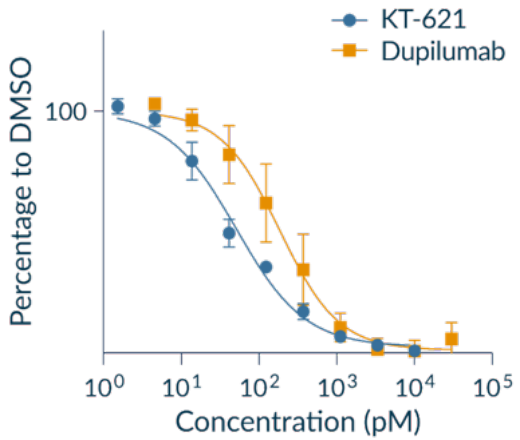
Robust Degradation in Relevant Cell Types

STAT6 Degradation in Immune Cells



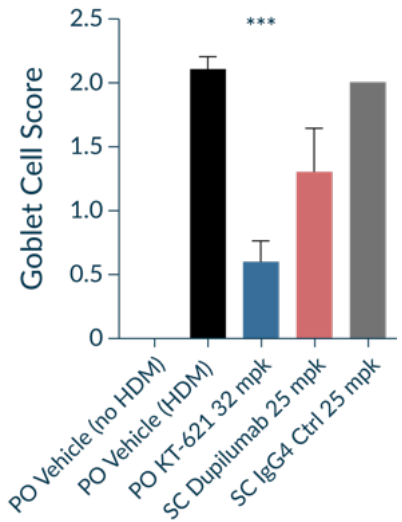
Full Inhibition of IL-4/IL-13 Pathways, More Potent than Dupilumab

IL-4 Induced TARC Release in Human PBMC



Excellent Preclinical Activity Comparable or Superior to Dupilumab

Lung Remodeling Goblet Cell Metaplasia in HDM Model[1]



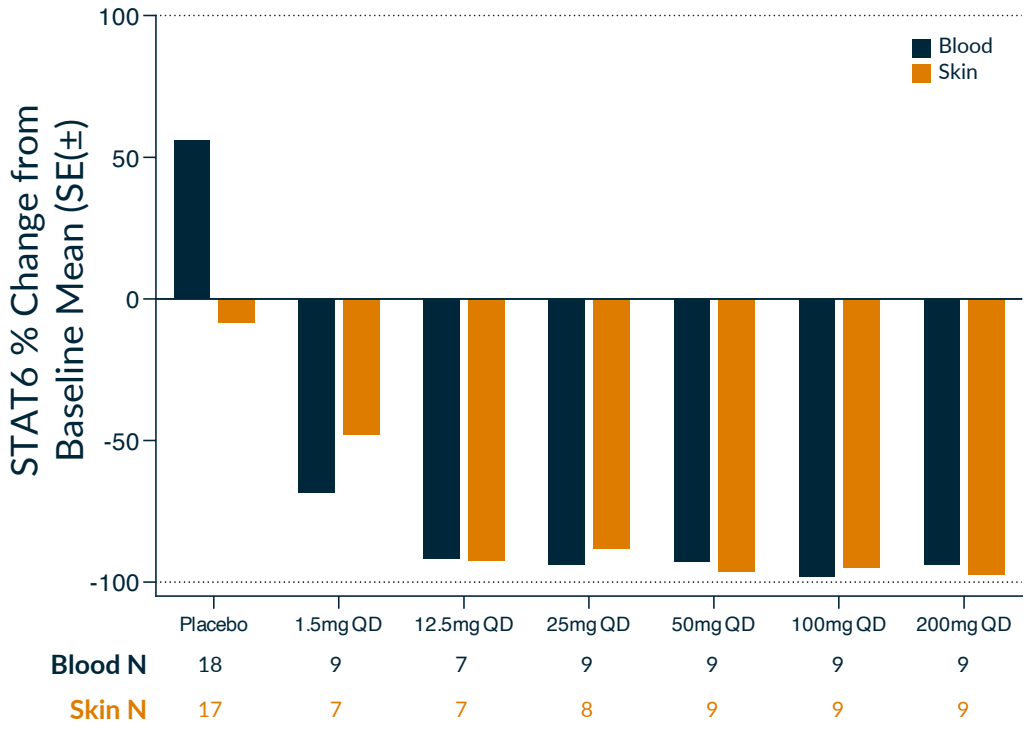
Favorable safety profile: well-tolerated in multiple preclinical species and safety studies at concentrations 40-fold above efficacious dose with up to 4 months of dosing

[1]A lung inflammation model induced by intranasal house dust mite (HDM) administration with dominant Type 2 inflammation in the IL4/IL4RA humanized mice, Le Floc'h et al, Allergy, 2020; *Significance to PO vehicle (HDM); #Significance to SC IgG4 Ctrl 25 mpk; ***p ≤ 0.001.

KT-621: Compelling Clinical Profile

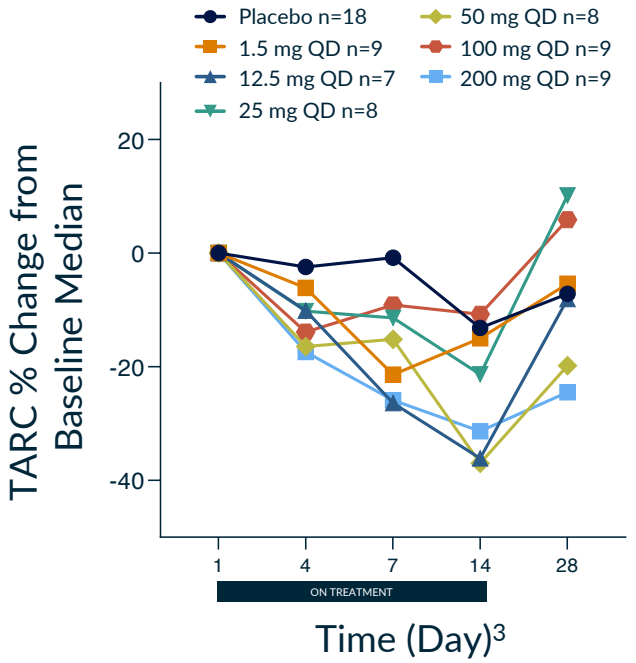
KT-621 Phase 1 Healthy Volunteer Data Suggests Potential for Dupilumab-like Activity in a Pill

Robust STAT6 Degradation in Blood and Skin Following Low Daily Oral Doses

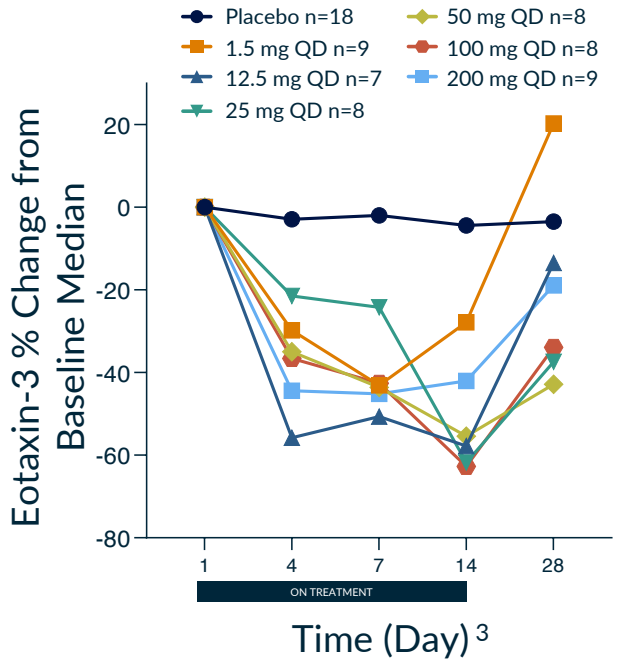


Reductions in Multiple Disease-Relevant Type 2 Biomarkers

Serum TARC[1] with 14-Day KT-621 Dosing



Serum Eotaxin-3[2] with 14-Day KT-621 Dosing



Favorable safety profile: well-tolerated across all dose levels with safety profile undifferentiated from placebo

[1]TARC levels measured in serum using MSD VPLEX; [2]Eotaxin-3 levels measured in serum using MSD VPLEX; [3]Compound dosed on Day 1.

KT-621 Development Plan Enables Efficient Path to Registration Across All Type 2 Diseases

Ph1 HV
SAD-MAD

✓ Complete

Ph1b AD
Single Arm, Open Label

✓ Today's Release

BROADEN2 TRIAL

Ph2b AD
Dose Ranging

Ongoing
Data expected by Mid-2027

Ph3 Dose

Ph3: AD (Initial), EoE, CSU, PN, BP

BREADTH TRIAL

Ph2b Asthma
Dose Ranging

Anticipated Start 1Q26

Ph3 Dose

Ph3: Asthma (Initial), COPD, CRSwNP

Initial parallel Phase 2b trials in moderate to severe AD and asthma have the potential to support subsequent Phase 3 trials across multiple dermatology, GI, and respiratory indications

BroADen KT-621 Phase 1b AD Study Surpassed Objectives

Effects on All Type 2 Biomarkers and Clinical Endpoints Were in Line with or in Some Cases Numerically Exceeded Published Data for Dupilumab at Week 4

Endpoints	BroADen Phase 1b Results Highlights
STAT6 Degradation	✓ Strong fidelity of translation from Phase 1a healthy volunteer study to AD patients with deep STAT6 degradation in blood and skin
Type 2 Biomarkers	✓ Robust reductions in Type 2 biomarkers in blood, skin lesions and lung (FeNO)
Clinical Endpoints	✓ Meaningful improvements of clinical endpoints and patient-reported outcomes in AD, comorbid asthma and allergic rhinitis
Safety	✓ Well-tolerated and favorable safety profile similar to Phase 1a healthy volunteer study

Clinical data continue to support a potential dupilumab-like profile in AD and other Type 2 diseases with a once daily, oral drug

No head-to-head trials have been conducted comparing KT-621 to dupilumab. Phase 1 clinical data for KT-621 may not be directly comparable to dupilumab's clinical data due to differences in molecule composition, trial protocols, dosing regimens, and patient populations and characteristics. Accordingly, cross-trial comparisons may not be reliable.

BROADEN STUDY

KT-621: Phase 1b Atopic Dermatitis Trial Results

Jared Gollob, MD

Chief Medical Officer

Agenda

- Atopic Dermatitis Overview
- Trial Design
- Demographics/Baseline Characteristics
- STAT6 Degradation
- Type 2 Inflammation Biomarkers
- Clinical Endpoints
- Safety

Atopic Dermatitis: Intensely Pruritic, Chronic and Underserved Disease

Opportunity Exists to Transform the Treatment Paradigm

- Chronic inflammatory disease that causes **inflamed and irritated skin**, making it itchy and painful
- Majority of patients have **disease onset as children** and carry the disease burden into **adulthood**
- Can significantly impact a patient's quality of life through **sleep loss**, **depression**, **missed work/school**, **interference** with social activities
- Common comorbidities include **asthma** and **allergic rhinitis**
- Many options only address symptoms, **not underlying Type 2 inflammation**
- Estimated **43 million** adults in US/EU5/JP[1]
- Significant unmet need with **less than 1 million** patients currently on dupilumab[2]

"There remains a clear need for new oral therapies that can address the underlying biology of the disease while potentially offering patients greater convenience."

– Eric Simpson, MD, MCR,
Frances J. Storrs Medical Dermatology Professor and Director of CLEAR Eczema Center, Oregon Health & Science University

[1]GlobalData (2023 diagnosed prevalent patient population for AD in US/EU5/JP); [2]GlobalData.

BROADEN STUDY

Phase 1b in Moderate to Severe Atopic Dermatitis Patients

Baseline entry criteria:

- Adult, moderate to severe AD patients

EASI	**≥16**
vIGA-AD	**≥3**
PPNRS	**≥4**
BSA	**≥10%**

- Documented TCS (Topical Corticosteroid) failure for AD
- Prior biologics allowed, after washout, if patient has responded to treatment
- Concurrent medications for AD not permitted

EASI: Eczema Area and Severity Index; vIGA-AD: Validated Investigator Global Assessment for Atopic Dermatitis; PPNRS: Peak Pruritus Numerical Rating Scale; BSA: Body Surface Area; TCS: Topical Corticosteroid.

Design

- Single arm, open label
- 22 patients
- Oral, once daily dose for 28 days
- 14-day follow-up after dosing completed

Dosing

- Two sequential dose cohorts
  - 100 mg (10 patients)
  - 200 mg (12 patients)

Endpoints

- Safety
- Pharmacokinetics
- STAT6 degradation
- Type 2 biomarkers in blood, skin lesions and lung
- Clinical activity (EASI, PPNRS, vIGA-AD, patient-reported outcomes)

BroADen Phase 1b Demographics

Generally Well-Balanced Across Treatment Cohorts

	100 mg (n=10)	200 mg (n=12)	Overall (n=22)
Gender, n (%)			
Female	6 (60)	7 (58.3)	13 (59.1)
Male	4 (40)	5 (41.7)	9 (40.9)
Age, years, mean (SD)	30.1 (8.5)	33.0 (11.4)	31.7 (10.1)
BMI, kg/m^2, mean (SD)	32.8 (11.5)	30.8 (9.2)	31.7 (10.1)
Ethnicity, n (%)			
Hispanic or Latino	3 (30)	2 (16.7)	5 (22.7)
Non Hispanic or Latino	7 (70)	10 (83.3)	17 (77.3)
Race, n (%)			
White	4 (40)	3 (25)	7 (31.8)
Black or African American	5 (50)	7 (58.3)	12 (54.5)
Asian	0	1 (8.3)	1 (4.5)
Mixed/Other	1 (10)	1 (8.3)	2 (9.1)

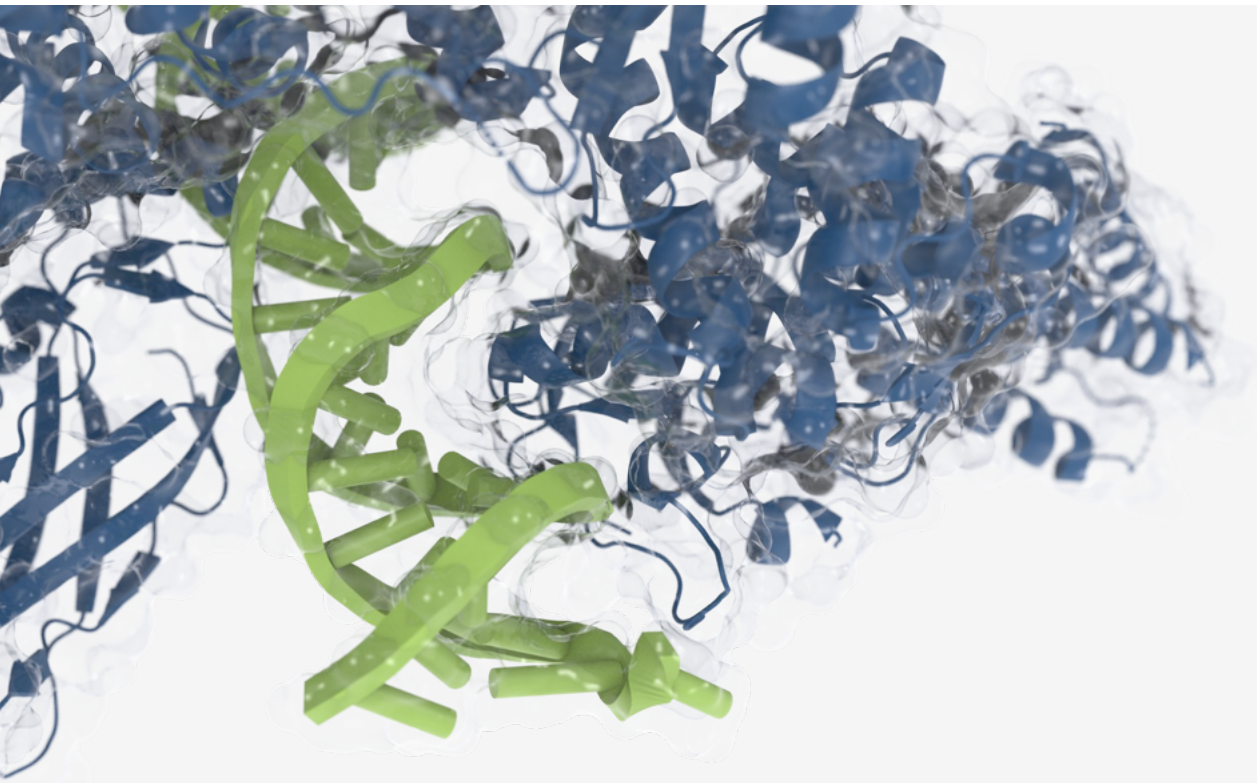
BMI: Body Mass Index; SD: Standard Deviation.

BroADen Phase 1b Baseline Disease Characteristics

Generally Well-Balanced Across Treatment Cohorts

	100 mg (n=10)	200 mg (n=12)	Overall (n=22)
vIGA-AD, n (%)			
Moderate (3)	6 (60)	6 (50)	12 (54.5)
Severe (4)	4 (40)	6 (50)	10 (45.5)
EASI Score, mean (SD)	23.5 (7.5)	26.1 (9)	24.9 (8.3)
Other Disease Characteristics, Mean (SD)			
Peak Pruritus NRS	7.4 (1.2)	7.6 (0.9)	7.5 (1)
SCORAD	55.7 (15.6)	63.8 (13.4)	60.1 (14.7)
BSA (%)	29.1 (9.8)	30.0 (15.1)	29.6 (12.7)
Comorbid Type 2 Diseases, n (%)			
Asthma	1 (10)	3 (25)[3]	4 (18.2)
Allergic Rhinitis	2 (20)	7 (58.3)	9 (40.9)
Prior Systemic Therapy for AD, n (%)	1 (10)[1]	4 (33.3)[2]	5 (22.7)

[1]Patient had prior dupilumab treatment; [2]Two patients had prior dupilumab treatment, one had prior tralokinumab treatment, and one had received both agents; [3]The three patients also have comorbid Allergic Rhinitis; EASI: Eczema Area and Severity Index; vIGA-AD: Validated Investigator Global Assessment for Atopic Dermatitis; PPNRS: Peak Pruritus Numerical Rating Scale; BSA: Body Surface Area; SCORAD: SCORing Atopic Dermatitis; SD: Standard Deviation.



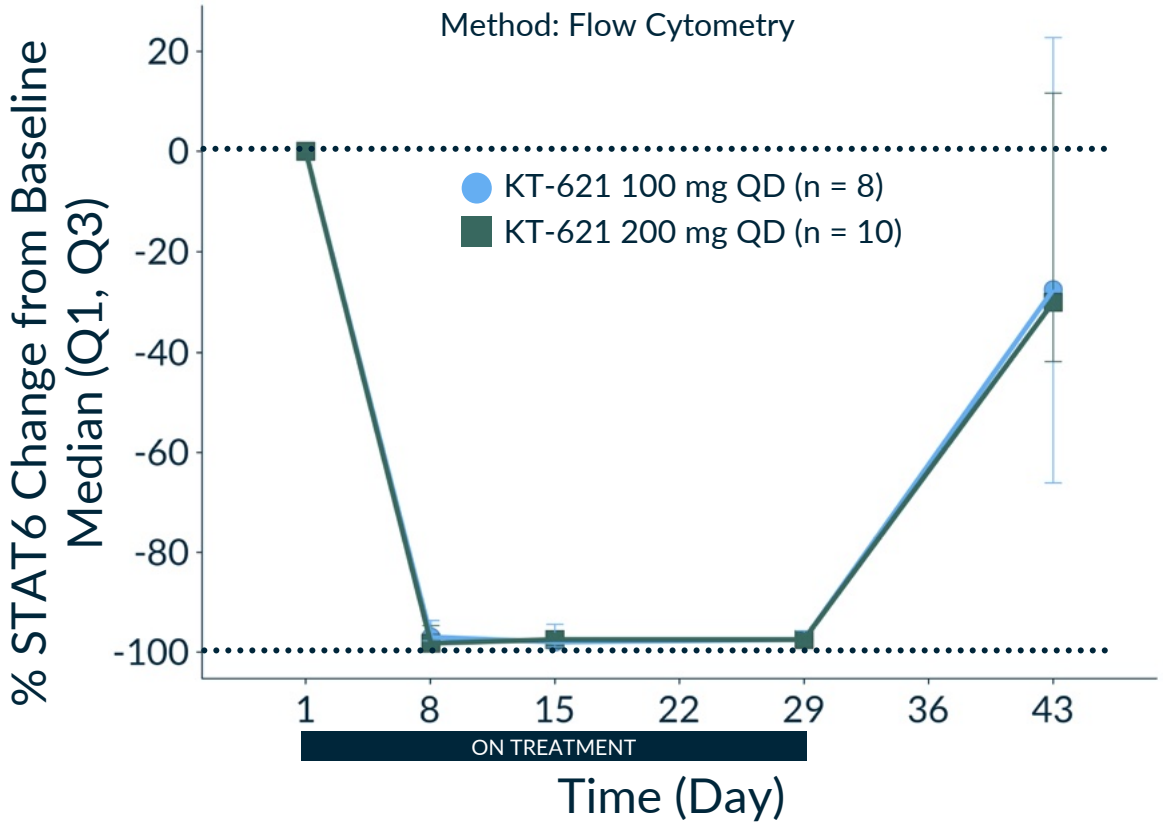
STAT6 Degradation

KT-621 Achieved Deep STAT6 Degradation in Blood

Degradation Maintained for 28 Days Across Both Dose Cohorts

- Median STAT6 degradation of 98% in blood in both dose groups after 4 weeks of treatment
- Deep degradation maintained throughout dosing period
- Plasma PK profile at 100 and 200 mg similar to healthy volunteer study
- Strong translation from healthy volunteer study

Median % Change from Baseline in STAT6

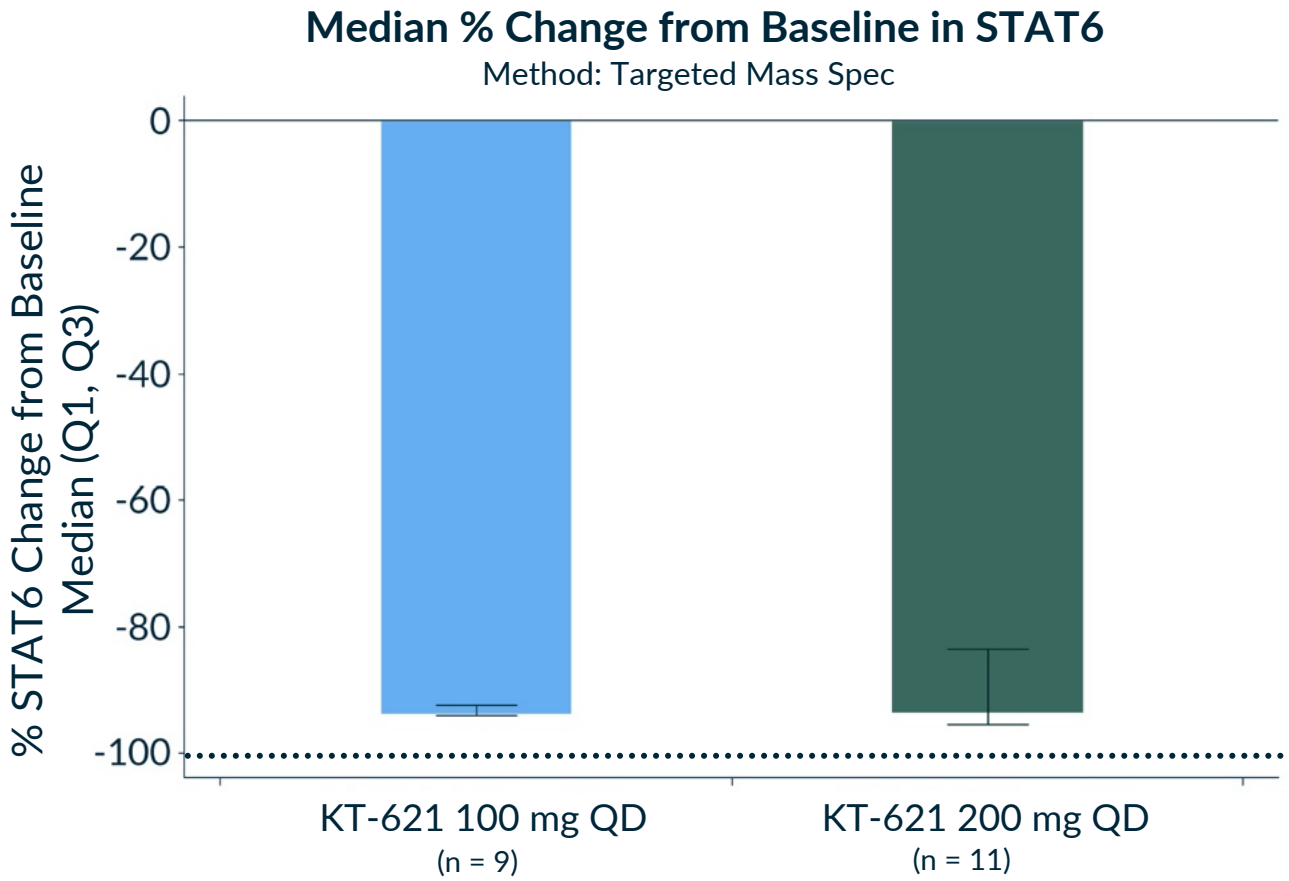


Dose	Median % Change from Baseline at D29
100 mg	-98%
200 mg	-98%

Note: N values reflect the number of participants with available samples at Day 29; Two patients (one each in 100 mg and 200 mg dose groups) did not have baseline samples collected, and two D29 samples were unevaluable due to shipping issues that led to loss of stability; PK: Pharmacokinetics.

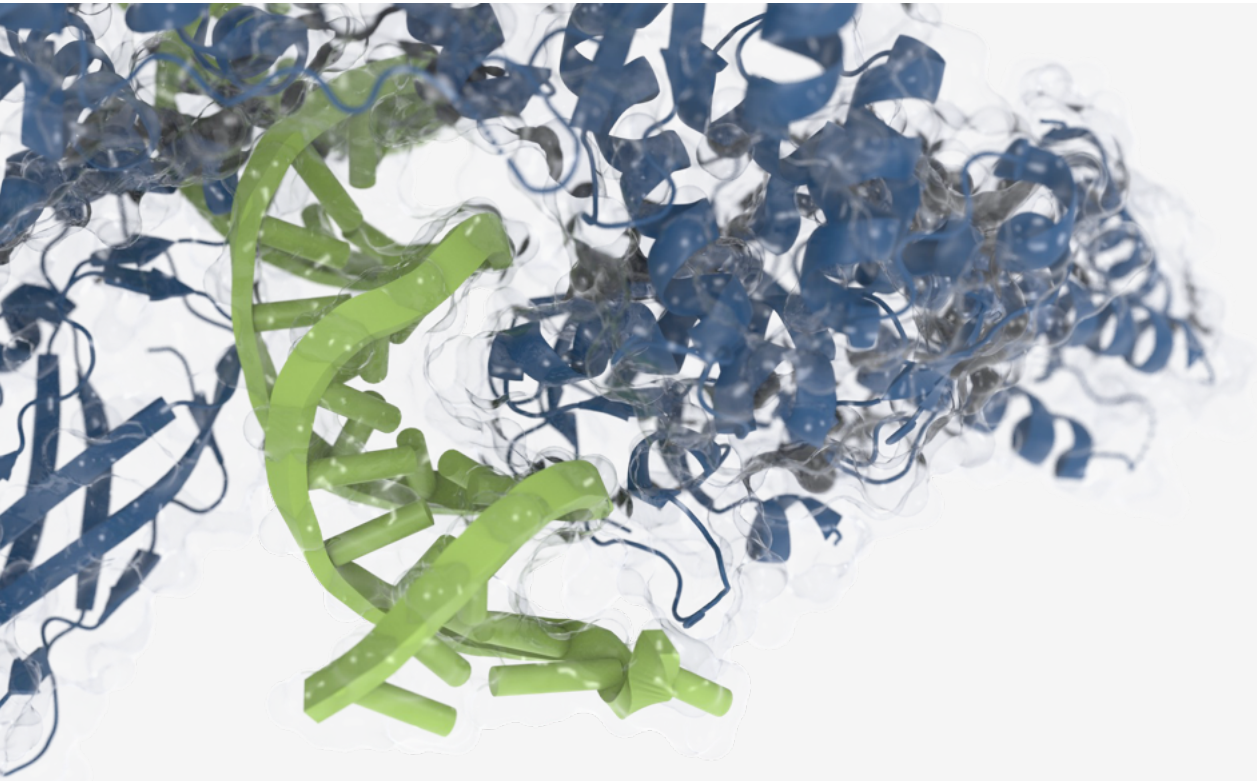
KT-621 Achieved Deep STAT6 Degradation in Skin

- Median STAT6 degradation of 94% in skin in both dose groups after 4 weeks of treatment using targeted Mass Spec
- Deep skin degradation with multiple patients' STAT6 levels below the LLOQ (lower limit of quantification)
- Strong translation from healthy volunteer study



Dose	Median % Change from Baseline at D29
100 mg	-94%
200 mg	-94%

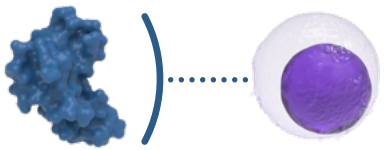
Note: N values reflect the number of participants with available samples at Day 29; two patients did not consent to D29 biopsies.



Type 2 Biomarkers

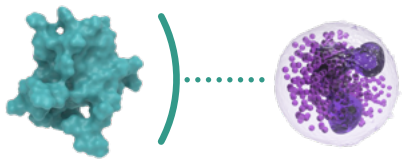
Disease-Relevant Biomarkers of Type 2 Inflammation

TARC (CCL17)



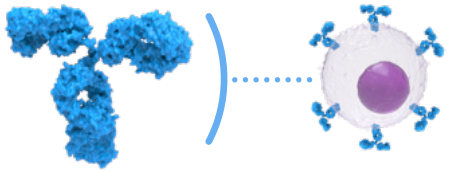
- TARC drives chemotaxis of CCR4-expressed T cells to inflammatory sites
- Validated biomarker of Type 2 inflammation suppression in patients[1]
- Baseline TARC levels typically elevated in AD

Eotaxin-3 (CCL26)



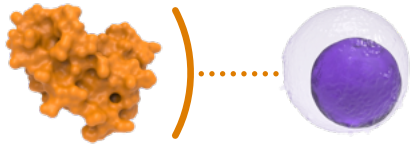
- Eotaxin-3 drives chemotaxis of CCR3-expressed inflammatory cells (e.g., eosinophils) to inflamed sites
- Highly specific downstream cytokine of the IL-4/IL-13 pathway[1]
- Baseline levels typically elevated in AD

IgE



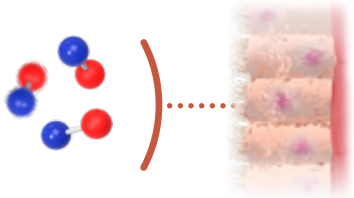
- IgE activates mast cells and basophils to release Type 2 cytokines (e.g., IL-4, IL-13)
- IL-4 promotes B-cell class switching, amplifying IgE production
- IgE half-life ~3 days, but IgE-producing cells persist longer[1]
- Baseline IgE levels typically elevated in AD

IL-31



- IL-31 is a key pruritogenic cytokine produced by activated Type 2 cells[2]
- Signals through the IL-31RA/OSMR complex on neurons, keratinocytes, and immune cells, linking immune activation to itch
- Baseline levels typically elevated in AD

FeNO

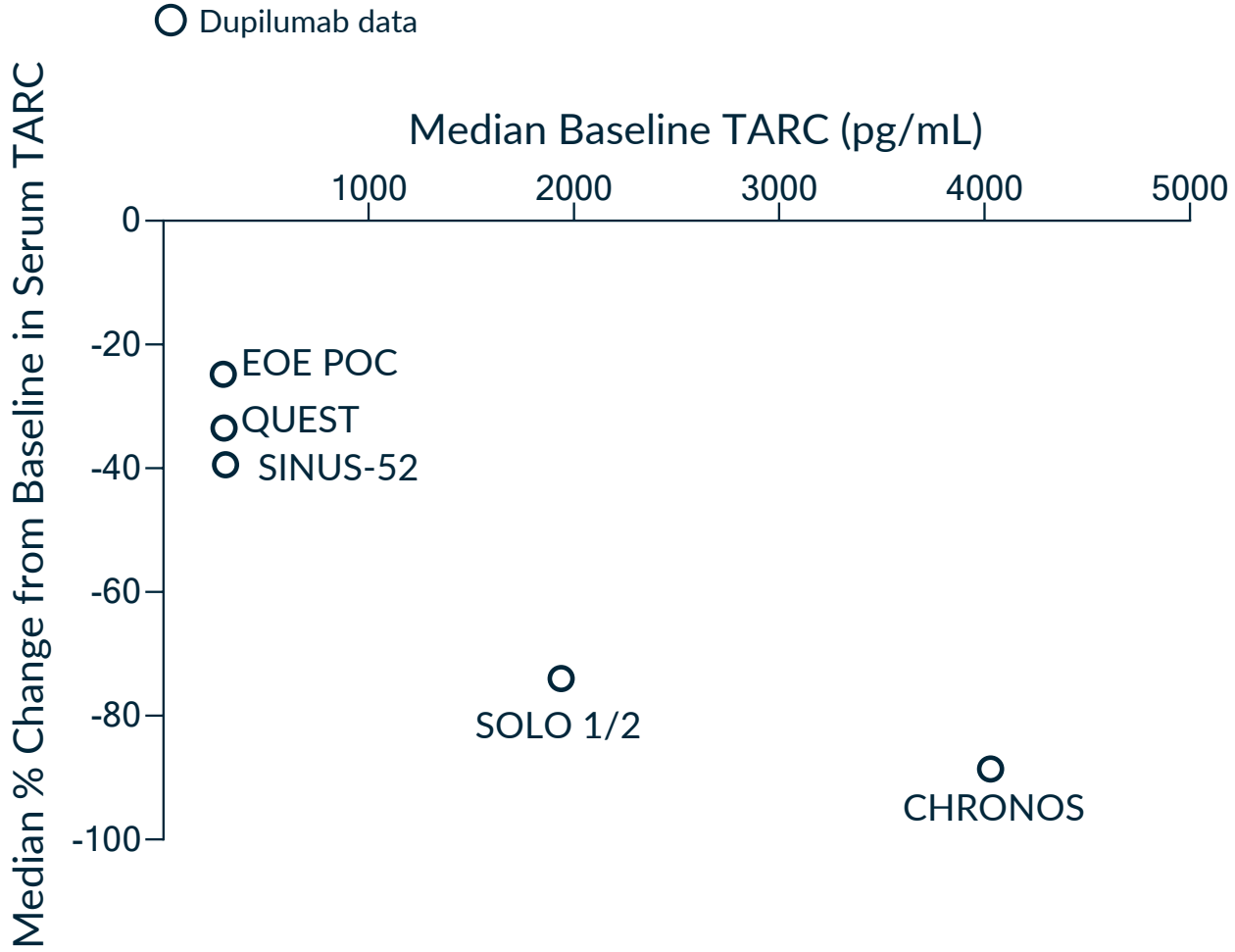


- FeNO (fractional exhaled nitric oxide) reflects airway epithelial iNOS activity driven by IL-4/13 signaling and is an indicator of Type 2 airway inflammation in asthma[3]
- Baseline FeNO is modulated by IL-4Rα–dependent pathways
- Historically not measured in AD patients

[1]Hamilton et al, Clinical & Experimental Allergy, 2021; [2]Raap et al, Journal of Allergy and Clinical Immunology, 2008; [3]Chung et al, Lancet, 2021.

Higher Baseline TARC Levels Associated with Greater TARC Reductions Across Dupilumab Studies

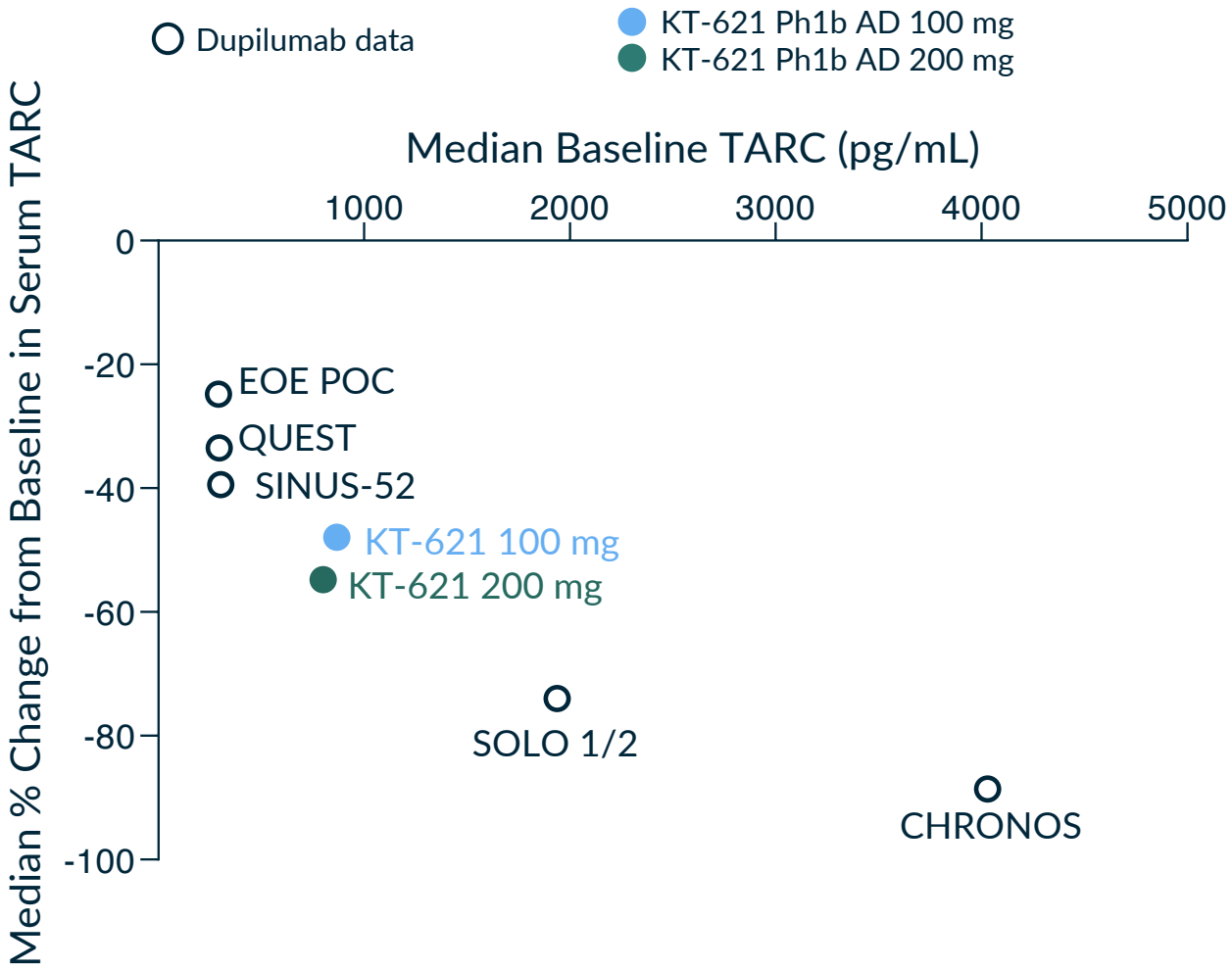
Dupilumab Studies[1]	Median Baseline TARC	Median TARC % Change	Time of Measurement
EOE POC	293	-25%	12 weeks
QUEST	295	-34%	52 weeks
SINUS-52	303	-39%	52 weeks
SOLO 1/2	1,937	-74%[2]	4 weeks
CHRONOS (Dupilumab + TCS)	4,030	-89%	52 weeks



[1]Hamilton et al, Clinical & Experimental Allergy, 2021; EOE POC trial: eosinophilic esophagitis; QUEST trial: asthma; SINUS-52 trial: chronic rhinosinusitis with nasal polyps; SOLO1/2 trials: atopic dermatitis; CHRONOS trial, dupilumab and topical corticosteroids (TCS): atopic dermatitis; [2]4-week measure interpolated from Hamilton et al, Clinical & Experimental Allergy, 2021.

Higher Baseline TARC Levels Associated with Greater TARC Reductions Across Dupilumab and KT-621 Studies

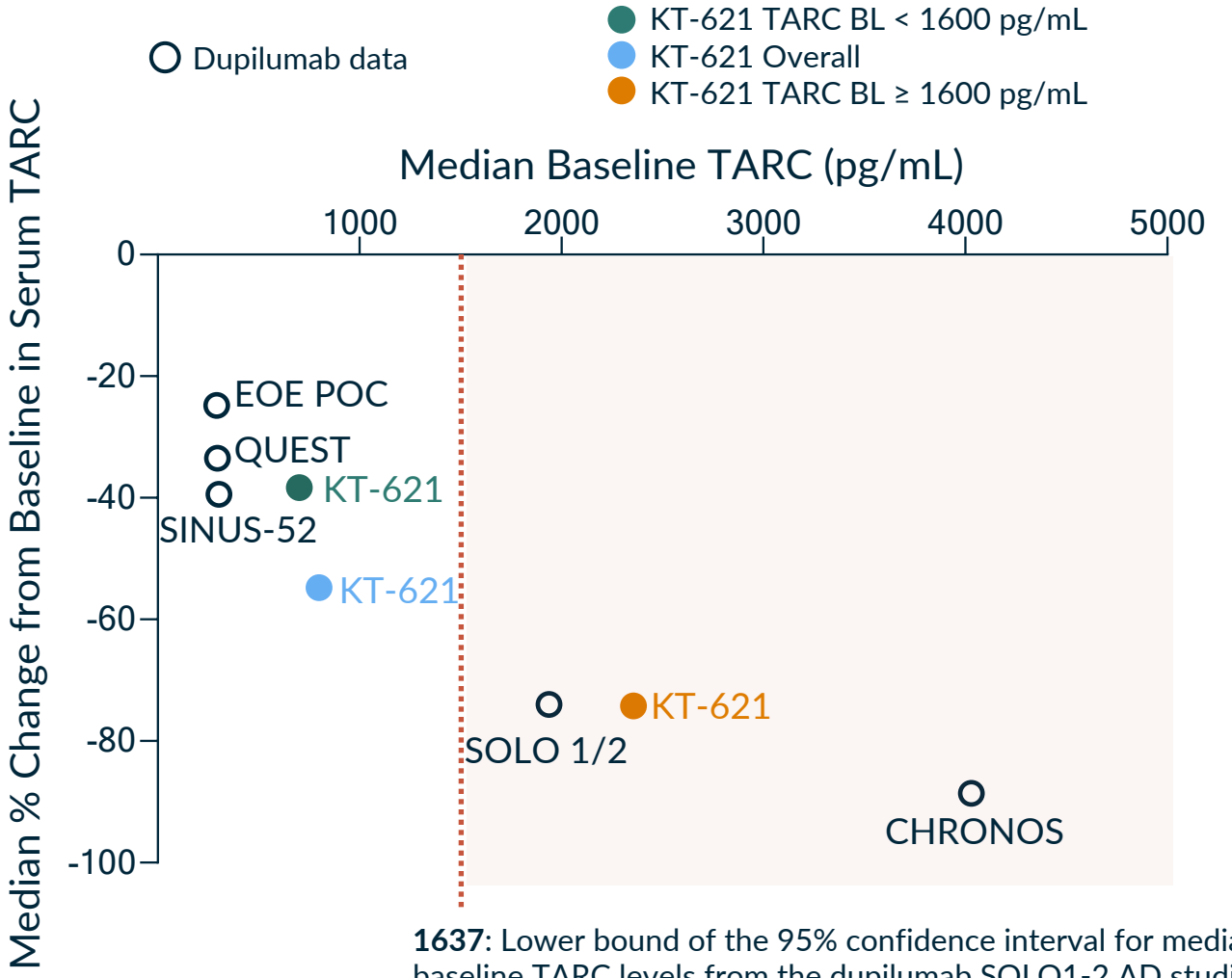
Dupilumab & KT-621 Studies[1,2]	Median Baseline TARC	Median TARC % Change	Time of Measurement
EOE POC	293	-25%	12 weeks
QUEST	295	-34%	52 weeks
SINUS-52	303	-39%	52 weeks
KT-621 100 mg	**868**	**-48%**	**4 weeks**
KT-621 200 mg	**800**	**-55%**	**4 weeks**
SOLO 1/2	1,937	-74%[3]	4 weeks
CHRONOS (Dupilumab + TCS)	4,030	-89%	52 weeks



[1]Hamilton et al, Clinical & Experimental Allergy, 2021; EOE POC trial: eosinophilic esophagitis; QUEST trial: asthma; SINUS-52 trial: chronic rhinosinusitis with nasal polyps; SOLO1/2 trials: atopic dermatitis; CHRONOS trial, dupilumab and topical corticosteroids (TCS): atopic dermatitis; [2]For KT-621, N values reflect the number of participants with available samples at Day 29, KT-621 100 mg n=10, KT-621 200 mg n=12; [3]4-week measure interpolated from Hamilton et al, Clinical & Experimental Allergy, 2021.

Similar TARC Reductions with KT-621 and Dupilumab When Selecting for Similar Baseline TARC Levels

Dupilumab & KT-621 Studies[1,2]	Median Baseline TARC	Median TARC % Change	Time of Measurement
EOE POC	293	-25%	12 weeks
QUEST	295	-34%	52 weeks
SINUS-52	303	-39%	52 weeks
KT-621 TARC <1600	**701**	**-38%**	**4 weeks**
KT-621 Overall	**800**	**-55%**	**4 weeks**
SOLO 1/2	1,937	-74%[3]	4 weeks
KT-621 TARC ≥ 1600	**2,356**	**-74%**	**4 weeks**
CHRONOS (Dupilumab + TCS)	4,030	-89%	52 weeks

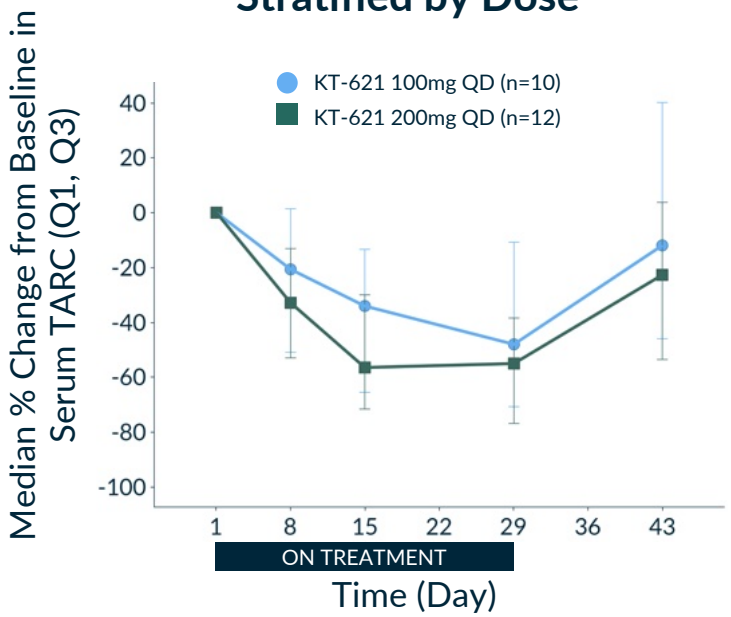


1637: Lower bound of the 95% confidence interval for median baseline TARC levels from the dupilumab SOLO1-2 AD studies

[1]Hamilton et al, Clinical & Experimental Allergy, 2021; EOE POC trial: eosinophilic esophagitis; QUEST trial: asthma; SINUS-52 trial: chronic rhinosinusitis with nasal polyps; SOLO1/2 trials: atopic dermatitis; CHRONOS trial, dupilumab and topical corticosteroids (TCS): atopic dermatitis; [2]KT-621, N values reflect the number of participants with available samples at Day 29, KT-621 100 mg n=10, KT-621 200 mg n=12; [3]4-week measure interpolated from Hamilton et al, Clinical & Experimental Allergy, 2021.

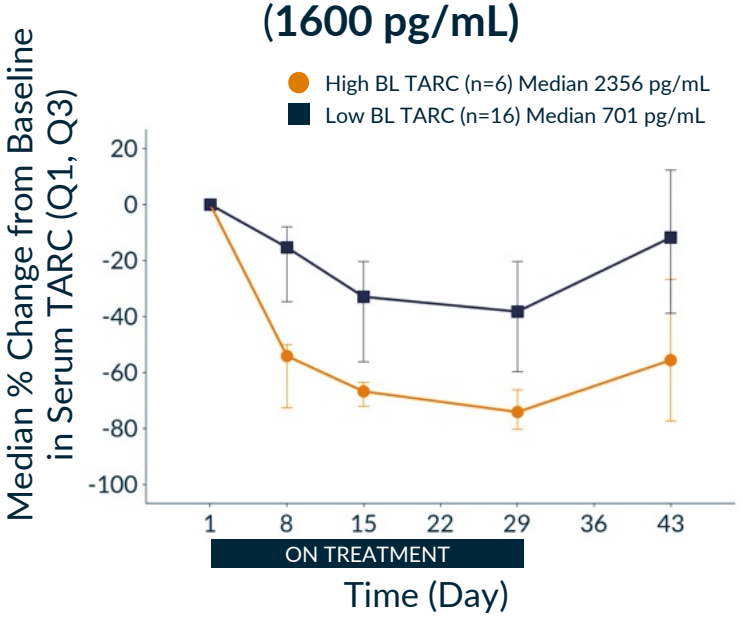
KT-621 Achieved Median TARC Reduction of 74% in Patients with TARC Levels Comparable to Dupilumab AD Studies

Stratified by Dose



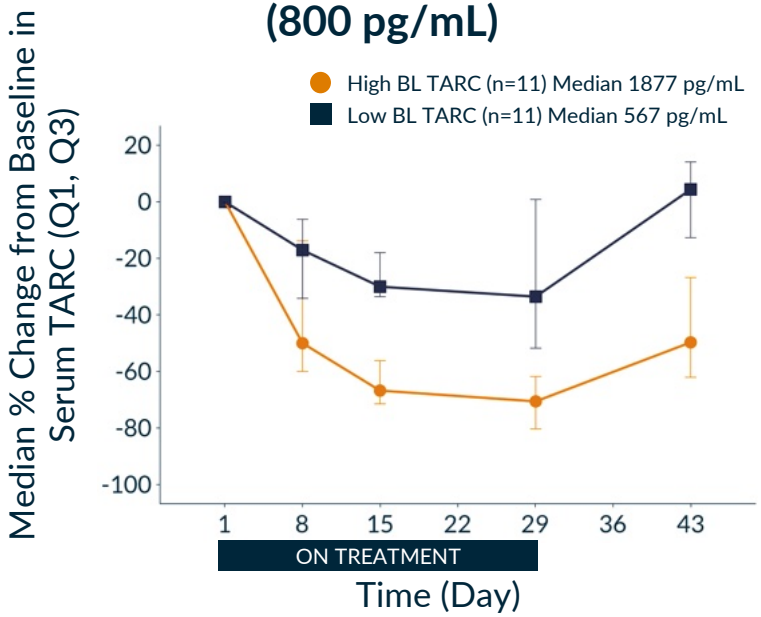
Dose	Median % Change from Baseline at D29
100 mg	-48%
200 mg	-55%

Stratified by Baseline TARC (1600 pg/mL)



Baseline TARC	Median % Change from Baseline at D29
≥ 1600 pg/mL	-74%
< 1600 pg/mL	-38%

Stratified by Median Baseline TARC (800 pg/mL)



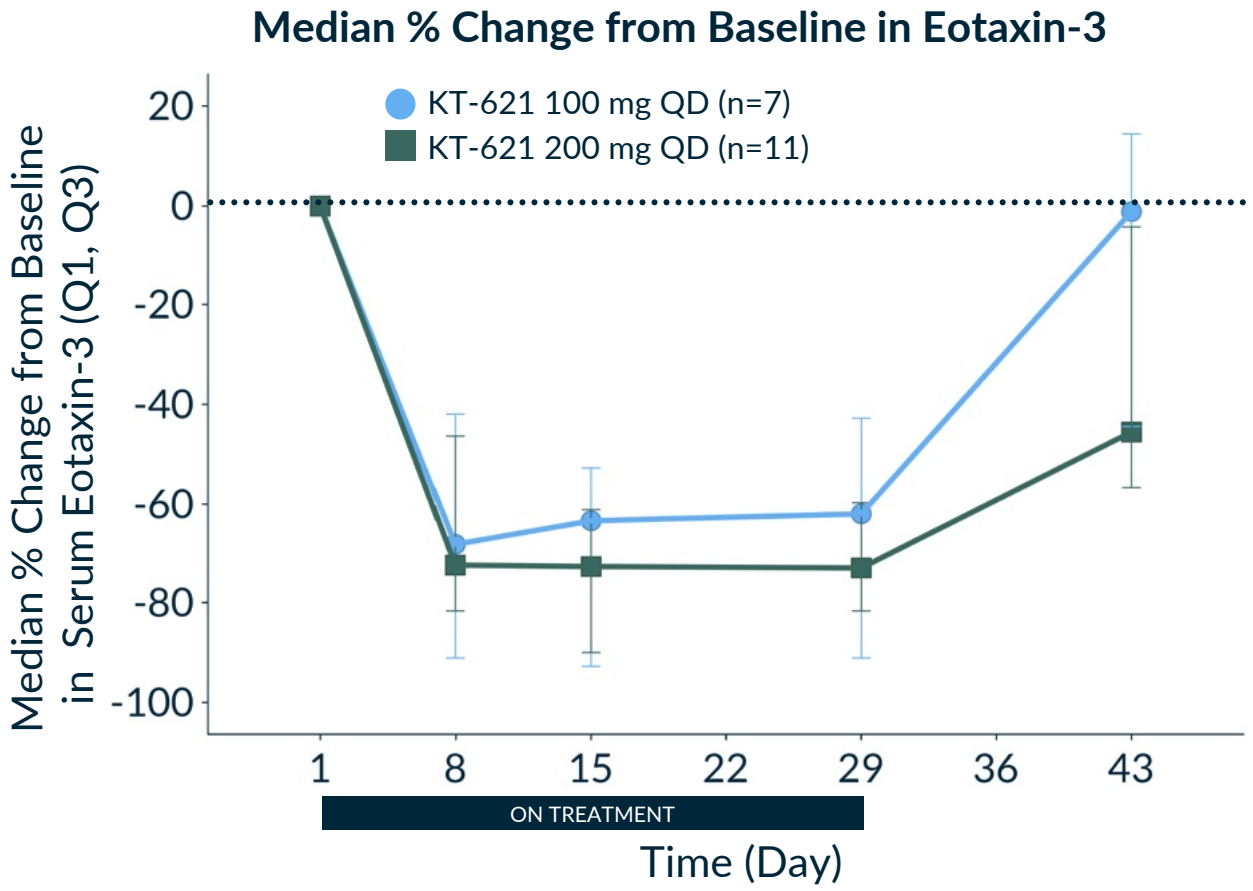
Baseline TARC	Median % Change from Baseline at D29
≥ 800 pg/mL	-71%
< 800 pg/mL	-34%

- Rapid and robust reduction of TARC across both dose cohorts
- 74% median TARC reduction similar to dupilumab at week 4 when stratifying for patients with similar baseline TARC levels (lower bound of the 95% confidence interval for median baseline TARC levels from the dupilumab SOLO1-2 AD studies)
- Even when stratified by BroADen internal median, KT-621 reached dupilumab-like TARC inhibition

Note: N values reflect the number of participants with available samples at Day 29.

KT-621 Achieved Median Serum Eotaxin-3 Reduction of up to 73%

- Rapid and robust median Eotaxin-3 reduction in both dose groups
- Reduction exceeds the dupilumab 52-week results of 51% median reduction in CRSwNP trial and 38% in asthma trial[1]

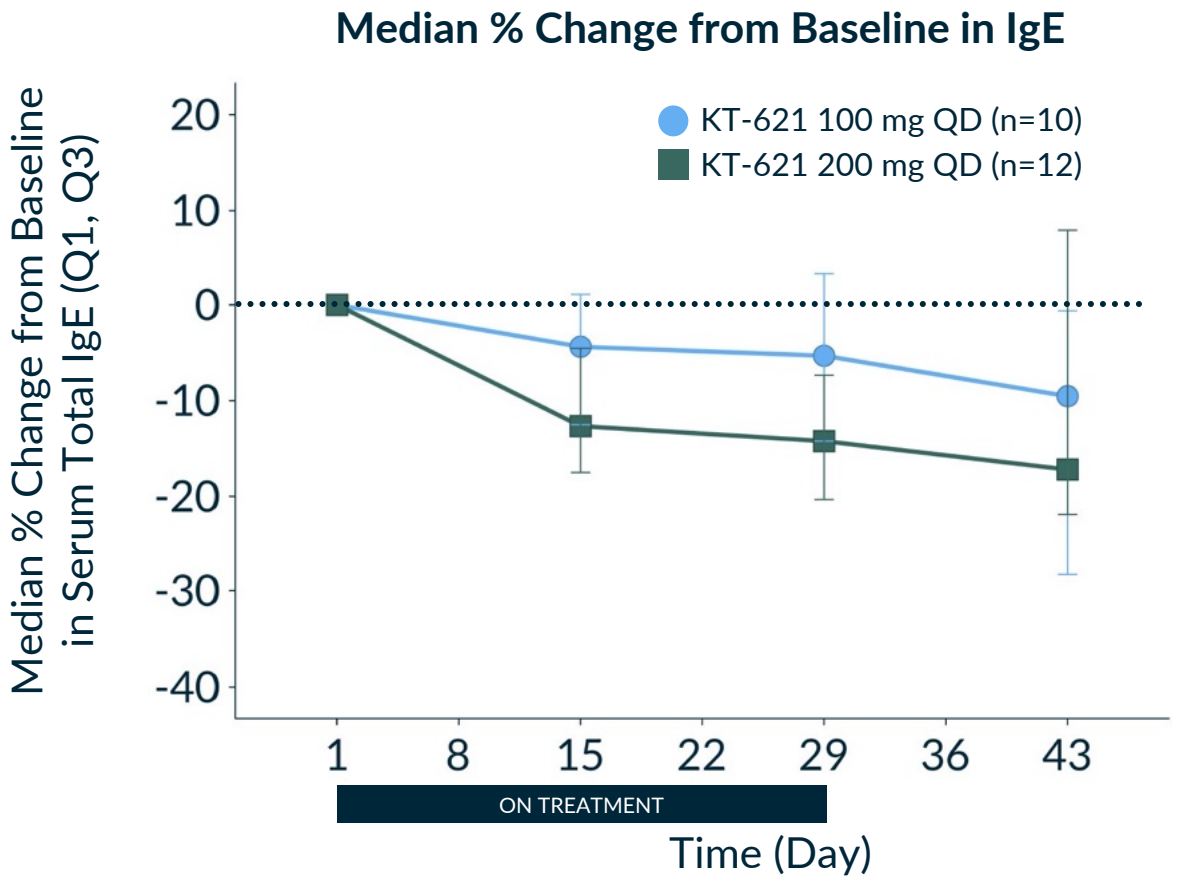


Dose	Median % Change from Baseline at D29
100 mg	-62%
200 mg	-73%

Note: N values reflect the number of participants with available samples at Day 29; Four patients had baseline levels below lower limit of quantification (LLOQ) of assay, hence change could not be calculated at D29; [1]Hamilton et al, Clinical & Experimental Allergy, 2021; CRSwNP: Chronic Rhinosinusitis with Nasal Polyps.

KT-621 Achieved Median Serum IgE Reduction of up to 14%

- IgE reduction comparable to dupilumab data in AD studies at week 4[1]
- More robust IgE reduction often necessitates several months of pathway suppression

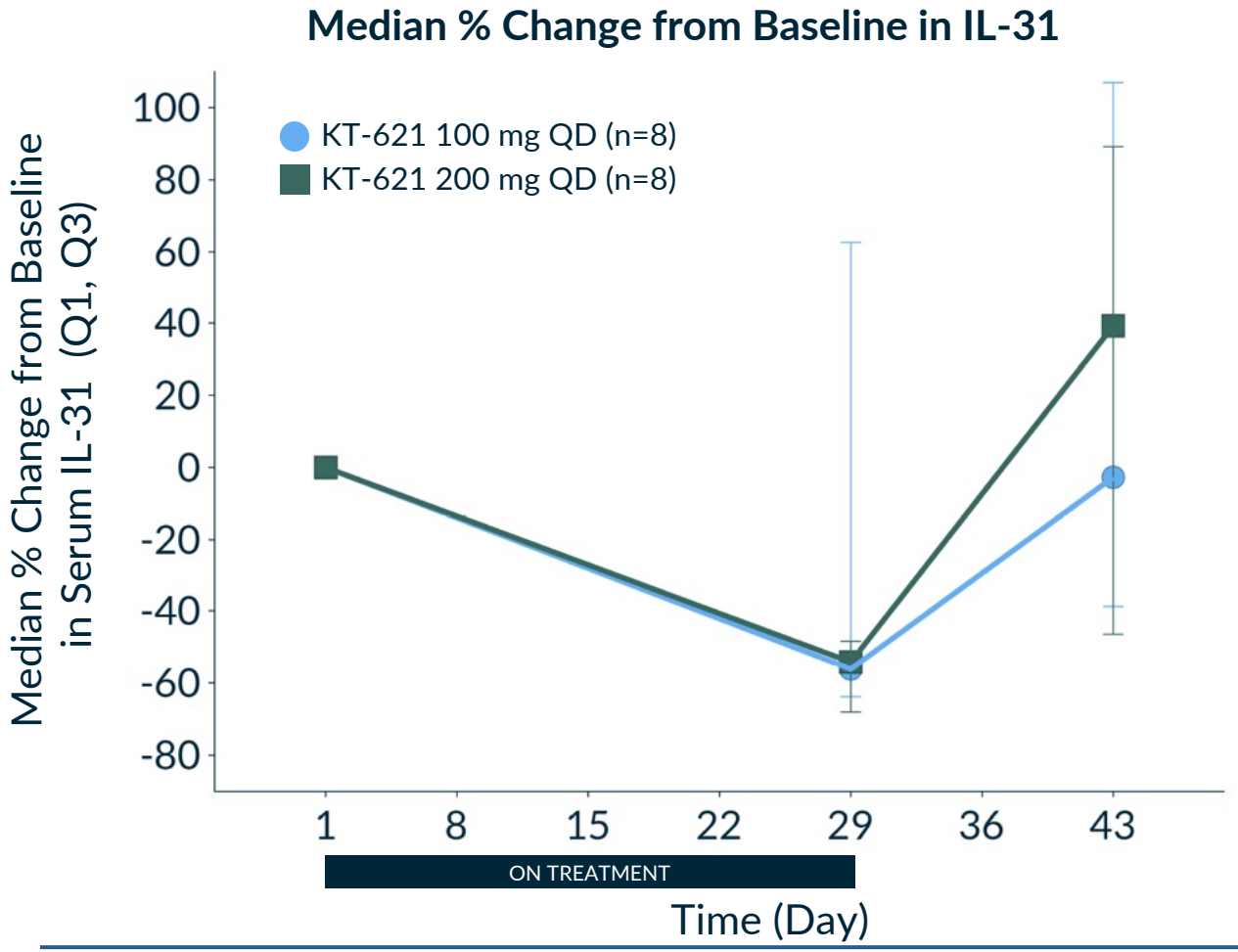


Dose	Median % Change from Baseline at D29
100 mg	-5%
200 mg	-14%

Note: N values reflect the number of participants with available samples at Day 29; [1]Hamilton et al, Clinical & Experimental Allergy, 2021.

KT-621 Achieved Robust Serum IL-31 Reduction of up to 56%

- Robust reduction of pruritogenic cytokine IL-31 in both dose groups
- First known demonstration of IL-31 reduction in blood of AD patients in response to IL-4/13 pathway inhibition

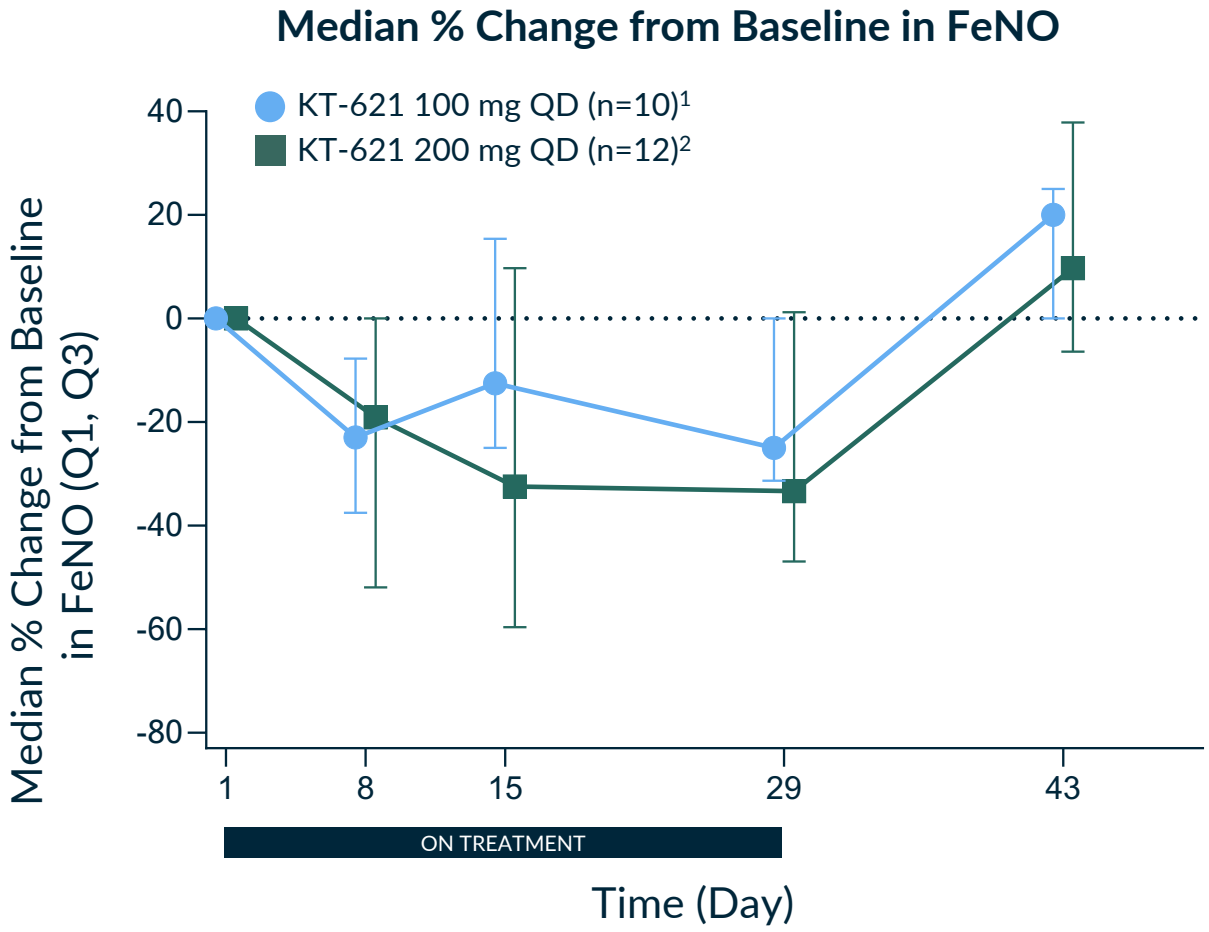


Dose	Median % Change from Baseline at D29
100 mg	-56%
200 mg	-54%

Note: N values reflect the number of participants with available samples at Day 29; In the 100 mg group, 2 patients had baseline levels below the lower limit of quantification (LLOQ) and change could not be calculated; In the 200 mg group, data for 3 patients are not available and 1 had baseline level below LLOQ.

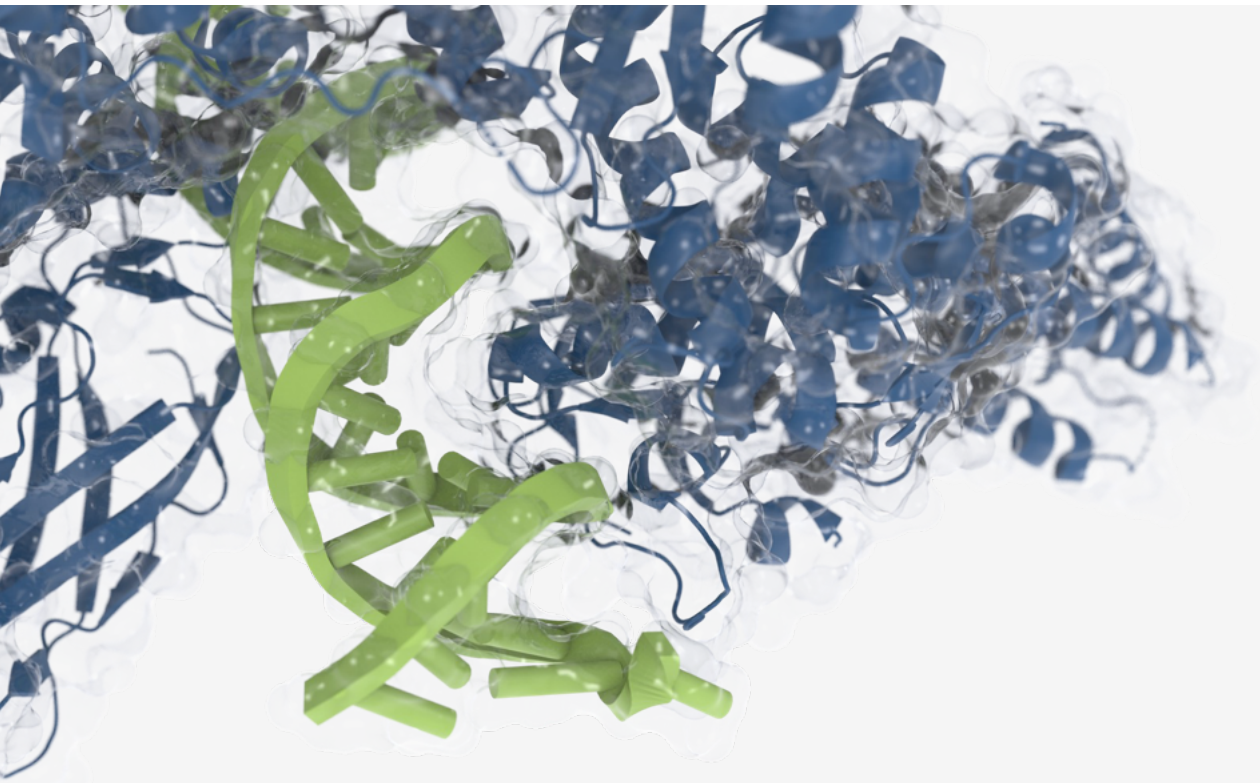
KT-621 Achieved up to 33% Reduction in FeNO in AD Patients

- Rapid and robust median FeNO reduction in both dose groups
- First known demonstration of FeNO reduction in AD patients



Dose	Median % Change from Baseline at D29
100 mg	-25%
200 mg	-33%

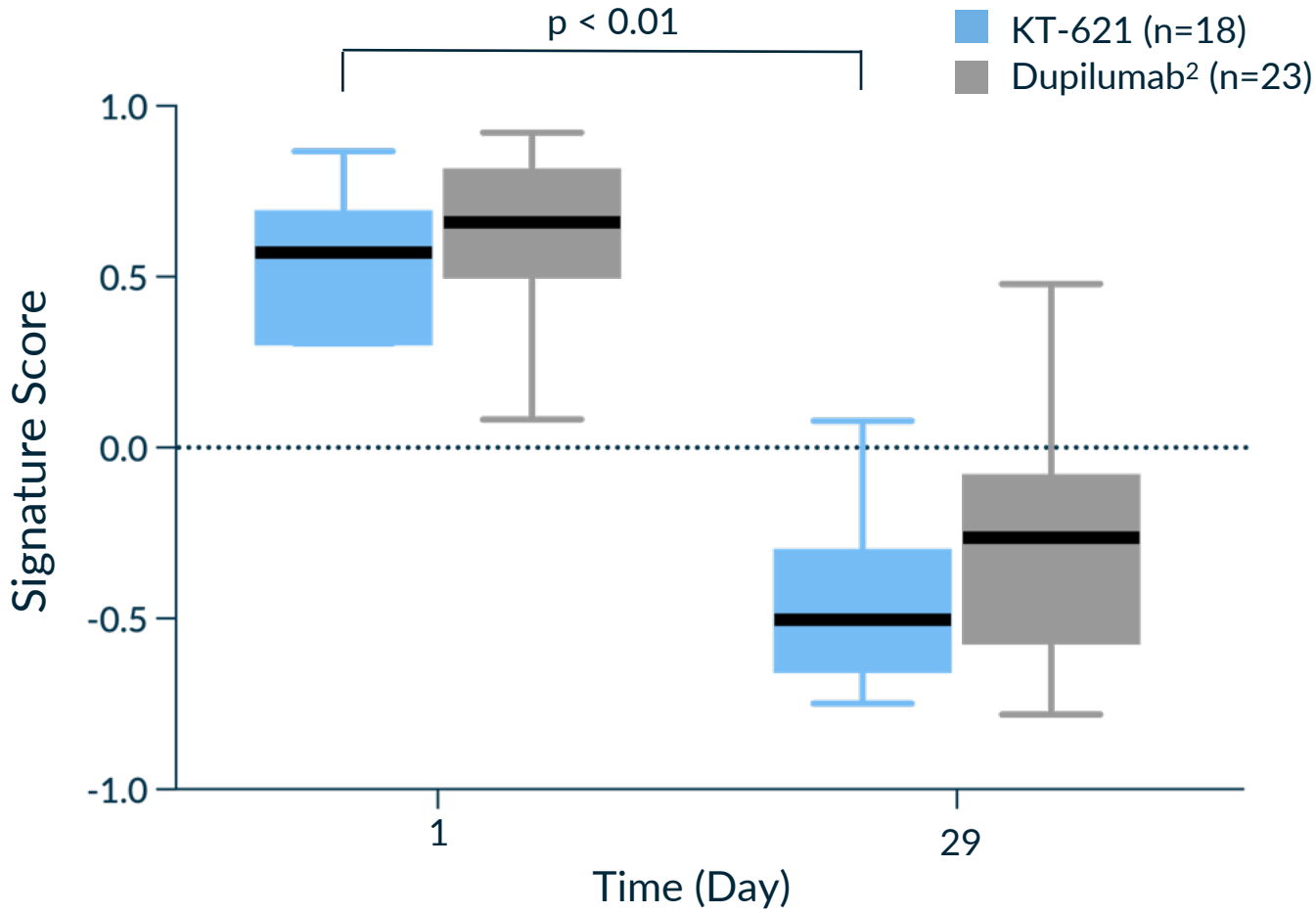
Note: Analysis based on observed cases at each visit; [1]Median FeNO baseline for 100 mg=13 ppb, one patient missed the D29 visit, n=9 at D29; [2]Median FeNO baseline for 200 mg=20 ppb.



Skin Biomarker Changes

KT-621 Significantly Downregulated Core Type 2 Inflammation Gene Set in Skin Lesions of AD Patients

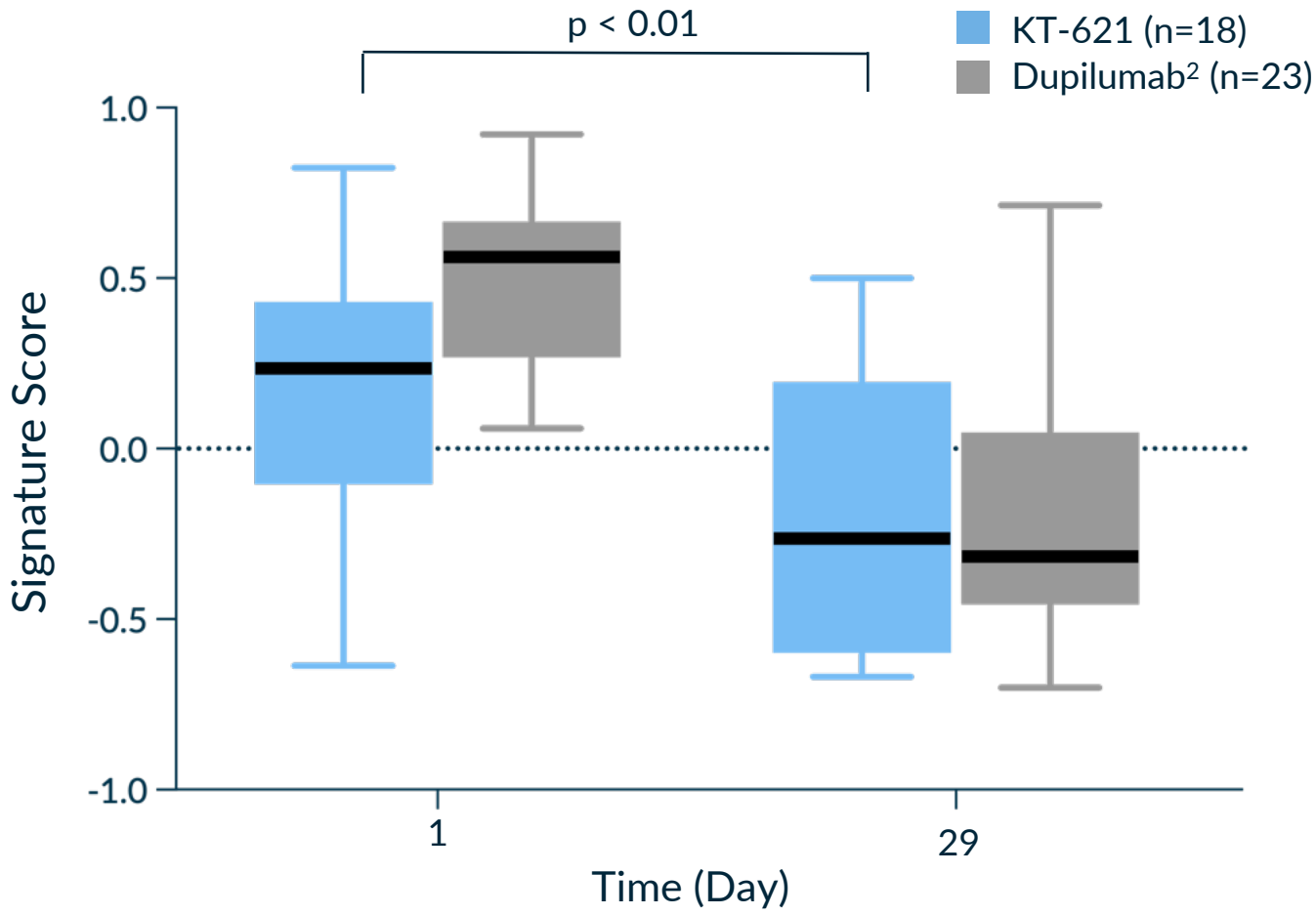
- **Genes in signature[1]:**
  - CCL26/Eotaxin-3
  - CCL17/TARC
  - CCL18/PARC
  - CCL13/MCP-4
- Changes in transcriptome comparable to published data for dupilumab at week 4[2]



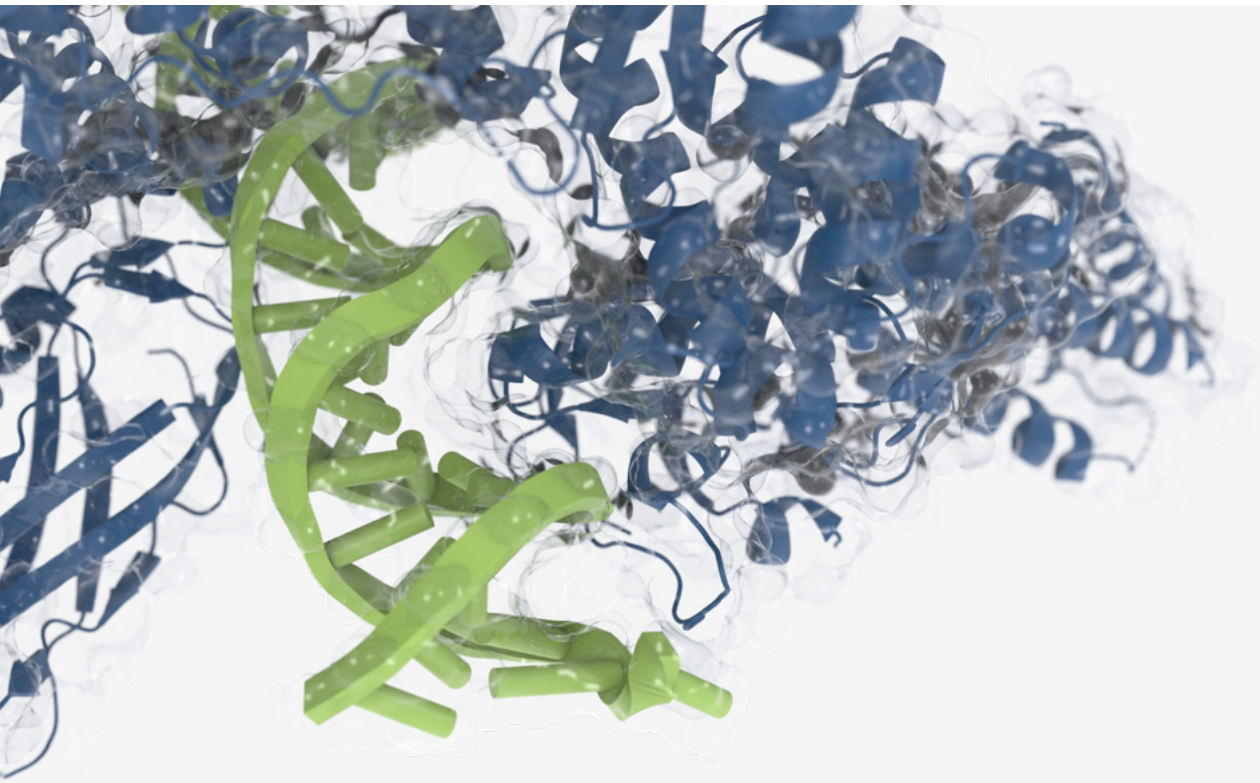
Note: N values reflect the number of participants with available samples at Day 29; In the 100 mg group, one patient did not consent to biopsy; In the 200 mg group, one patient did not consent to D29 biopsy, one patient D29 biopsy was lost, and one patient had poor quality sequencing; [1]Signature scores generated by GSVA (Hänzelmann et al, BMC Bioinformatics, 2013); [2]Dupilumab data from Guttman-Yassky et al, JACI, 2019; P-value < 0.01 for paired t-test between D1 and D29 for both treatments.

KT-621 Significantly Downregulated AD Disease-relevant Gene Set in Skin Lesions of AD Patients

- **Genes in signature[1]:**
  - CCL26/Eotaxin-3 (Type 2)
  - CCL17/TARC (Type 2)
  - CCL18/PARC (Type 2)
  - CCL13/MCP-4 (Type 2)
  - CXCL1/GRO-α (Th17)
  - PI3/Peptidase inhibitor 3 (Th17)
  - KRT16/Keratin 16 (skin hyperplasia)
  - MMP12 (general inflammation)
  - POSTN/Periostin (fibrosis)
  - OSMR/Oncostatin-M receptor (itch)
  - S100A12 (Th17/Th22)
  - TSLP
- Changes in transcriptome comparable to published data for dupilumab at week 4[2]



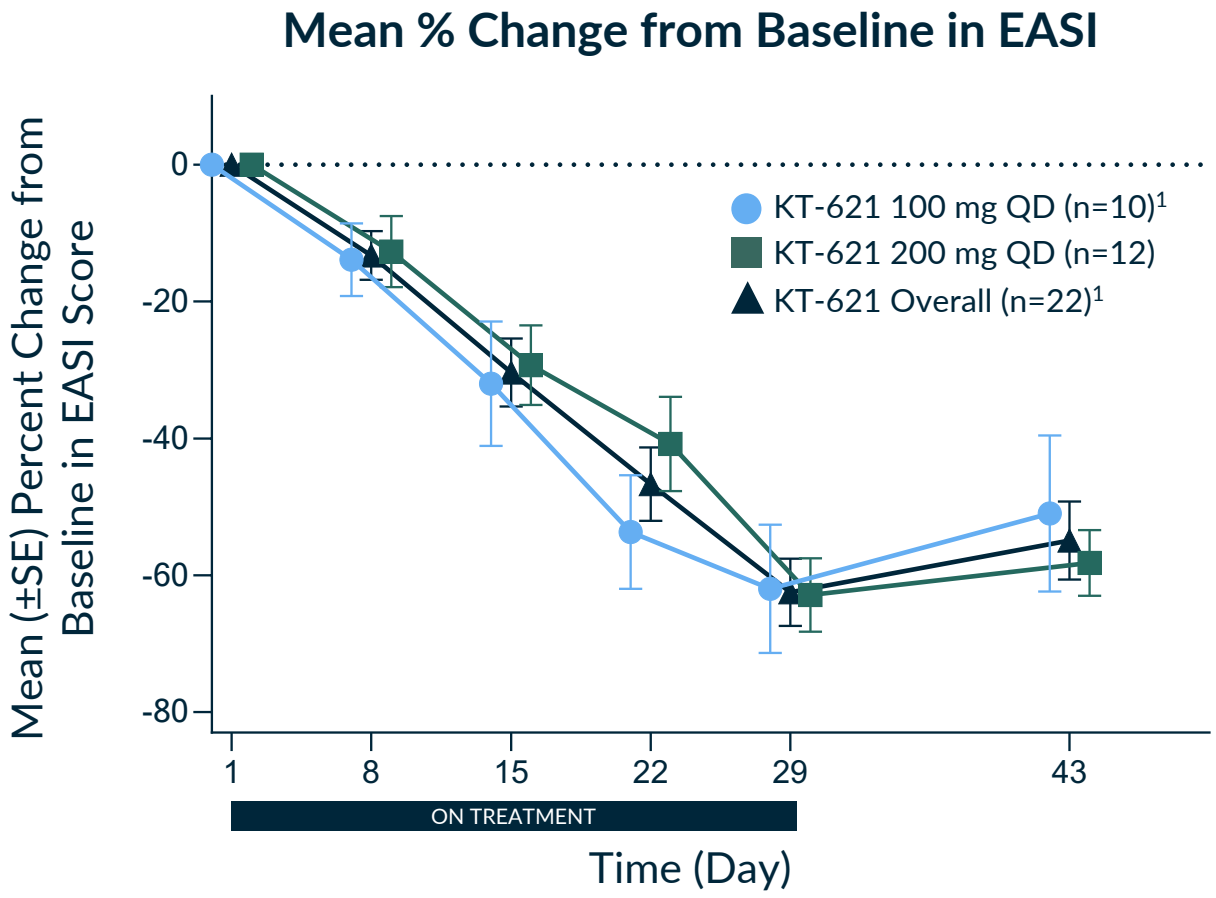
Note: N values reflect the number of participants with available samples at Day 29; In the 100 mg group, one patient did not consent to biopsy; In the 200 mg group, one patient did not consent to D29 biopsy, one patient D29 biopsy was lost, and one patient had poor quality sequencing; [1]Signature scores generated by GSVA (Hänzelmann et al, BMC Bioinformatics, 2013); [2]Dupilumab data from Guttman-Yassky et al, JACI, 2019; P-value < 0.01 for paired t-test between D1 and D29 for both treatments.



Clinical Endpoints

KT-621 Achieved 63% Overall Mean Reduction in EASI

- KT-621 achieved rapid and robust mean EASI reduction in both dose cohorts
- Reductions seen as early as Day 8 and were similar across both dose groups

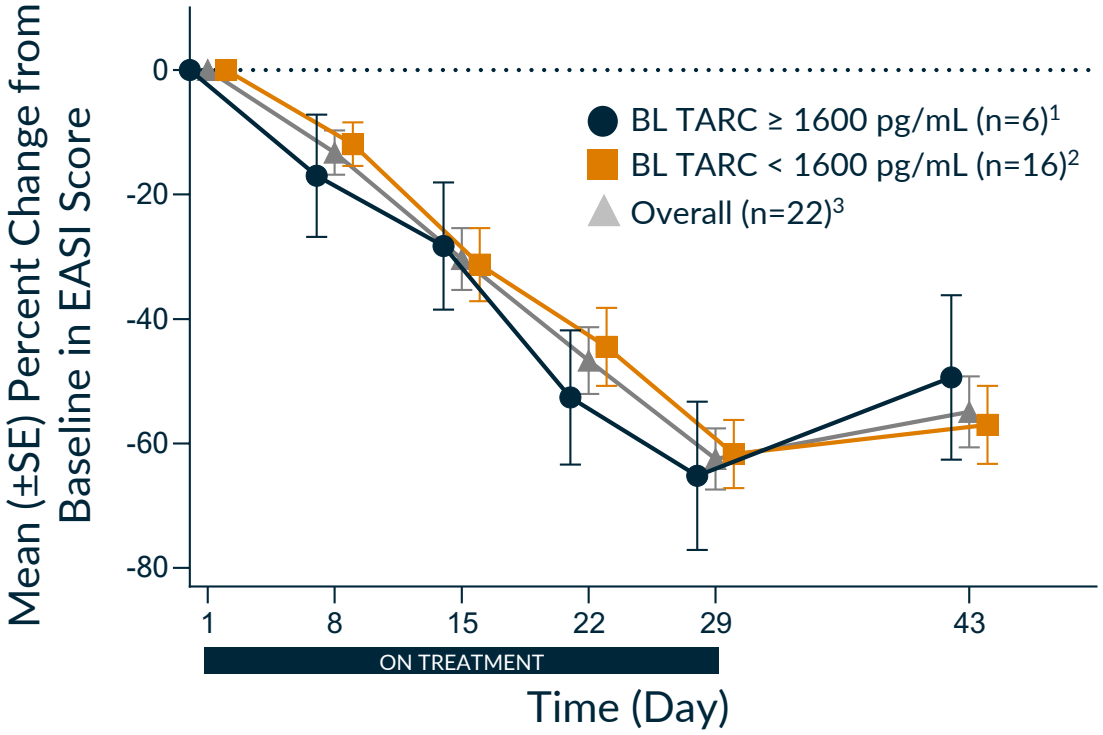


Dose	Mean % Change from Baseline at D29
100 mg	-62%
200 mg	-63%
Overall	-63%

Note: Analysis based on observed cases at each visit; [1]One patient in the 100 mg cohort missed the D29 visit (n=9 for 100 mg and n=21 for Overall at D29); EASI: Eczema Area and Severity Index.

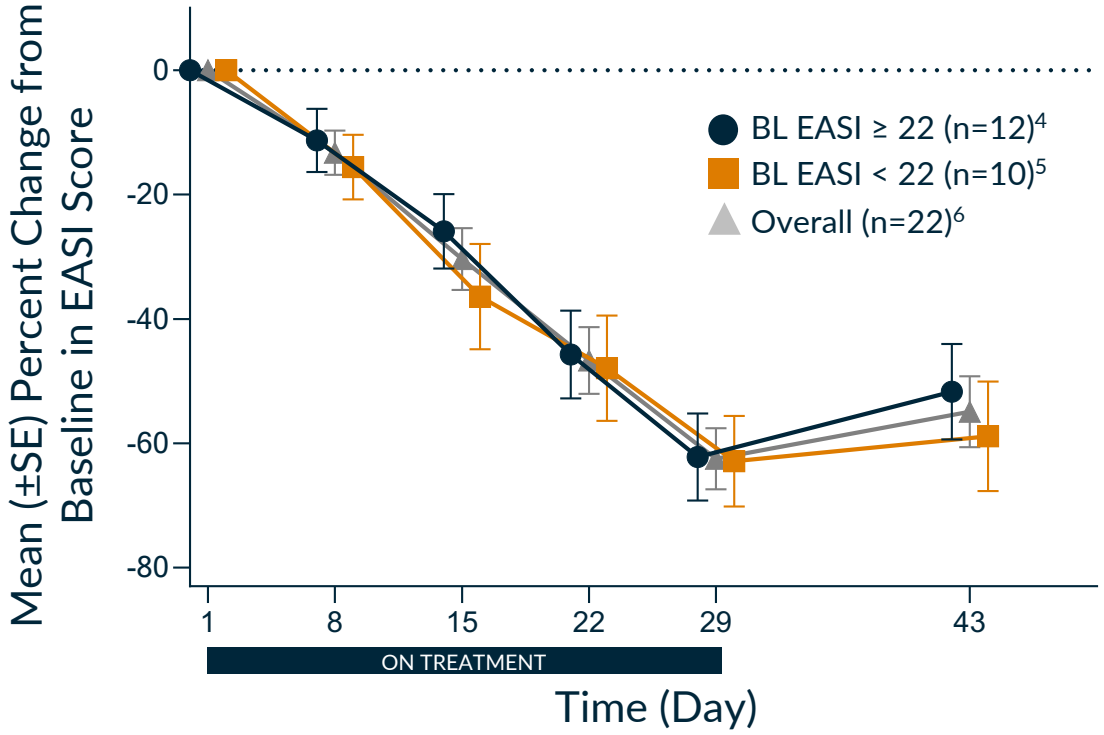
KT-621 Achieved Consistent EASI Reduction Across All Patient Subgroups, Regardless of Baseline TARC/EASI

Mean % Change in EASI: Stratified by Baseline TARC



Baseline TARC	Mean % Change from Baseline in EASI at D29
≥ 1600	-65%
< 1600	-62%
Overall	-63%

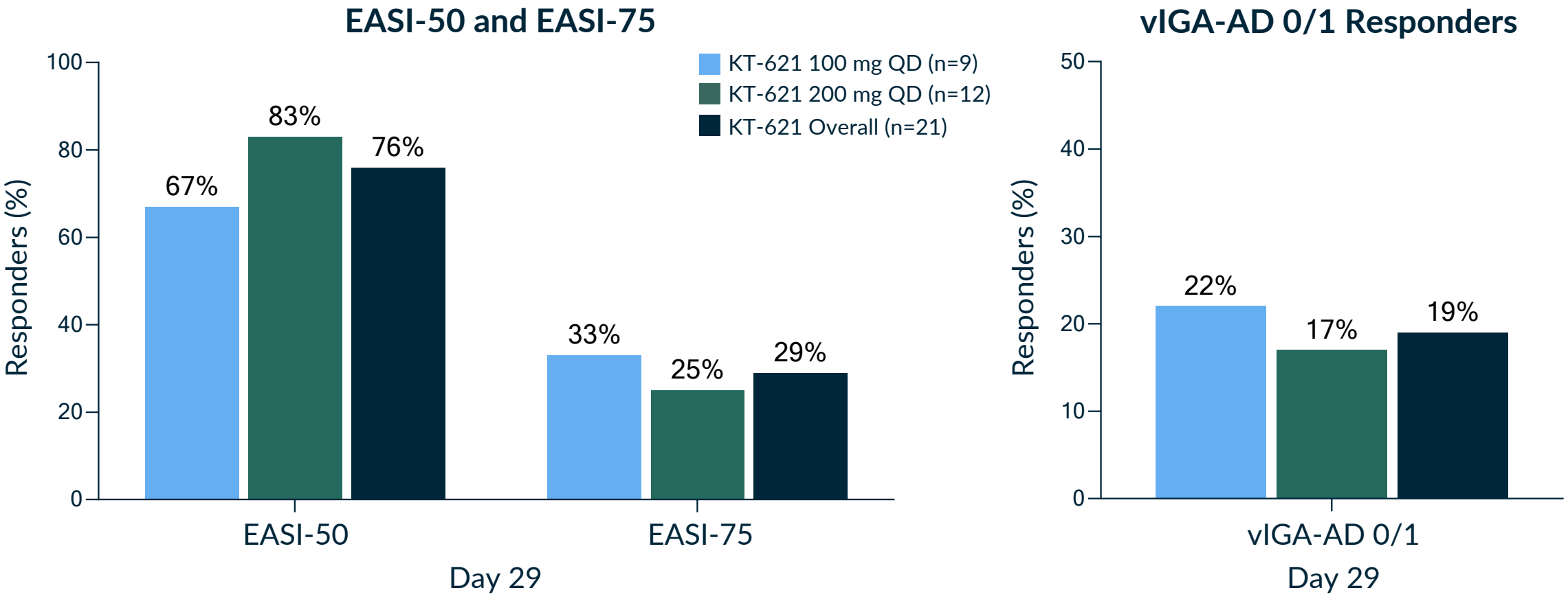
Mean % Change in EASI: Stratified by Baseline EASI



Baseline EASI	Mean % Change from Baseline in EASI at D29
≥ 22	-62%
< 22	-63%
Overall	-63%

Note: Analysis based on observed cases at each visit; [1]Mean baseline EASI=33, one patient missed the D29 visit (n=5 at D29); [2]Mean baseline EASI=22; [3]Mean baseline EASI=25, n=21 at D29; [4]Mean baseline EASI=31, n=11 at D29; [5]Mean baseline EASI=18; [6]Mean baseline EASI=25, n=21 at D29; EASI: Eczema Area and Severity Index.

KT-621 Achieved Robust Clinical Improvement Across EASI-50, EASI-75, and vIGA-AD 0/1 Measures

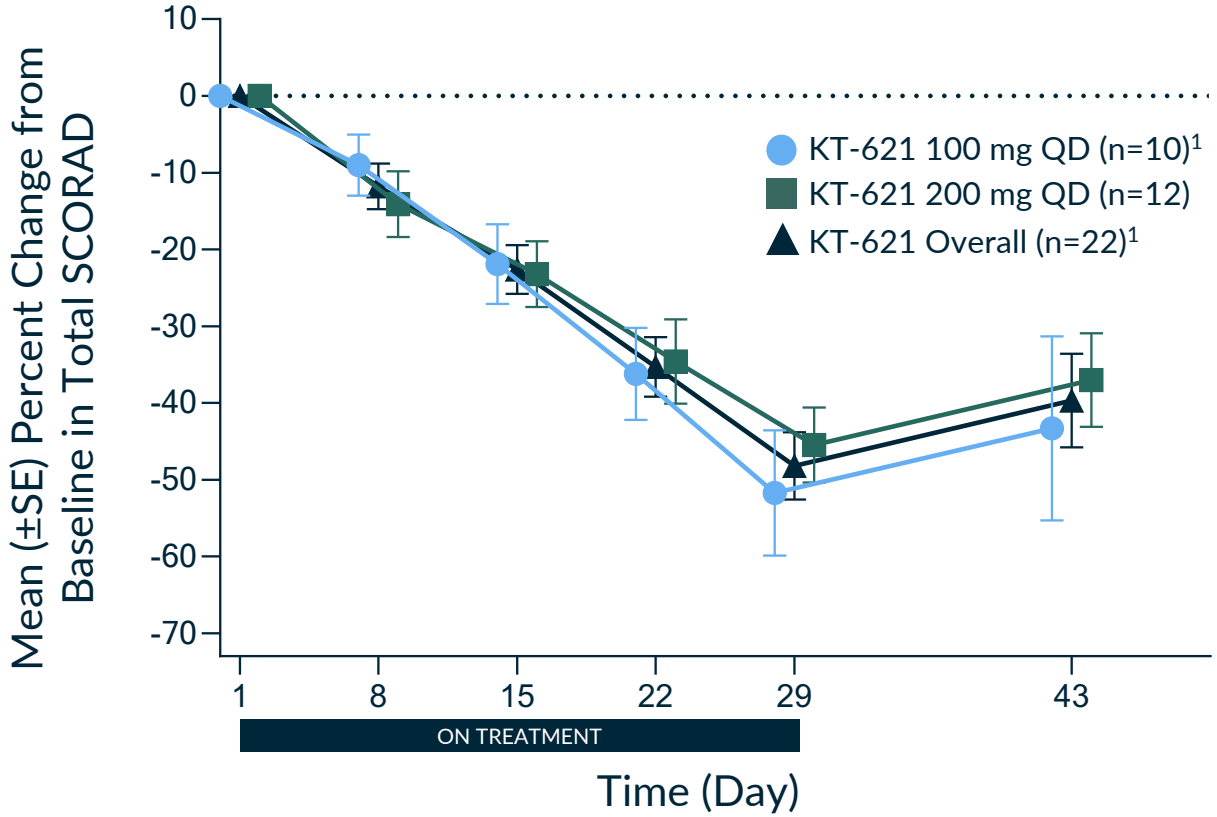


Note: Analysis based on observed cases at D29; EASI: Eczema Area and Severity Index; vIGA-AD: Validated Investigator Global Assessment for Atopic Dermatitis.

KT-621 Achieved 48% Overall Mean Reduction in SCORAD

- SCORAD is a validated composite measure of AD severity, integrating extent, intensity, and patient-reported impact
- KT-621 achieved rapid and robust mean SCORAD reductions in both dose cohorts

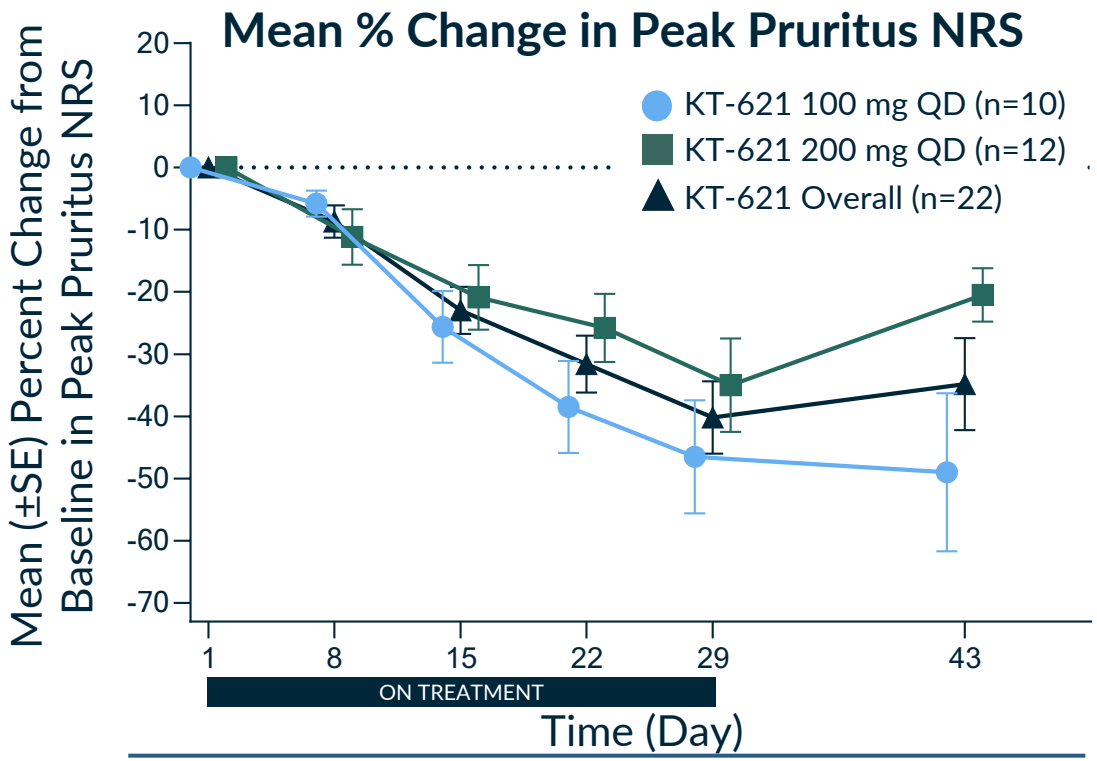
Mean % Change in Total SCORAD



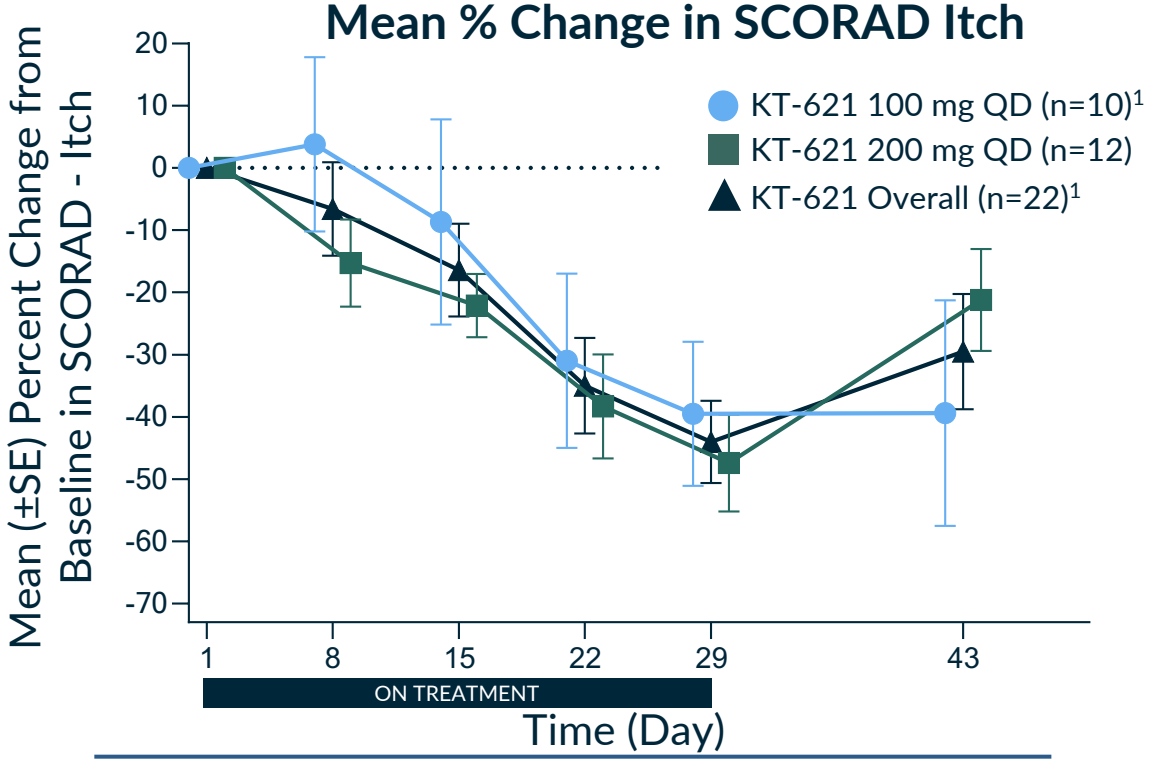
Dose	Mean % Change from Baseline at D29
100 mg	-52%
200 mg	-46%
Overall	-48%

Note: Analysis based on observed cases at each visit; [1]One patient in the 100 mg cohort missed the D29 visit (n=9 for 100 mg and n=21 for Overall at D29); SCORAD: SCORing Atopic Dermatitis.

KT-621 Achieved Robust and Consistent Reductions in Itch Across Independent Clinical Measures



Dose	Mean % Change from Baseline at D29
100 mg	-47%
200 mg	-35%
Overall	-40%



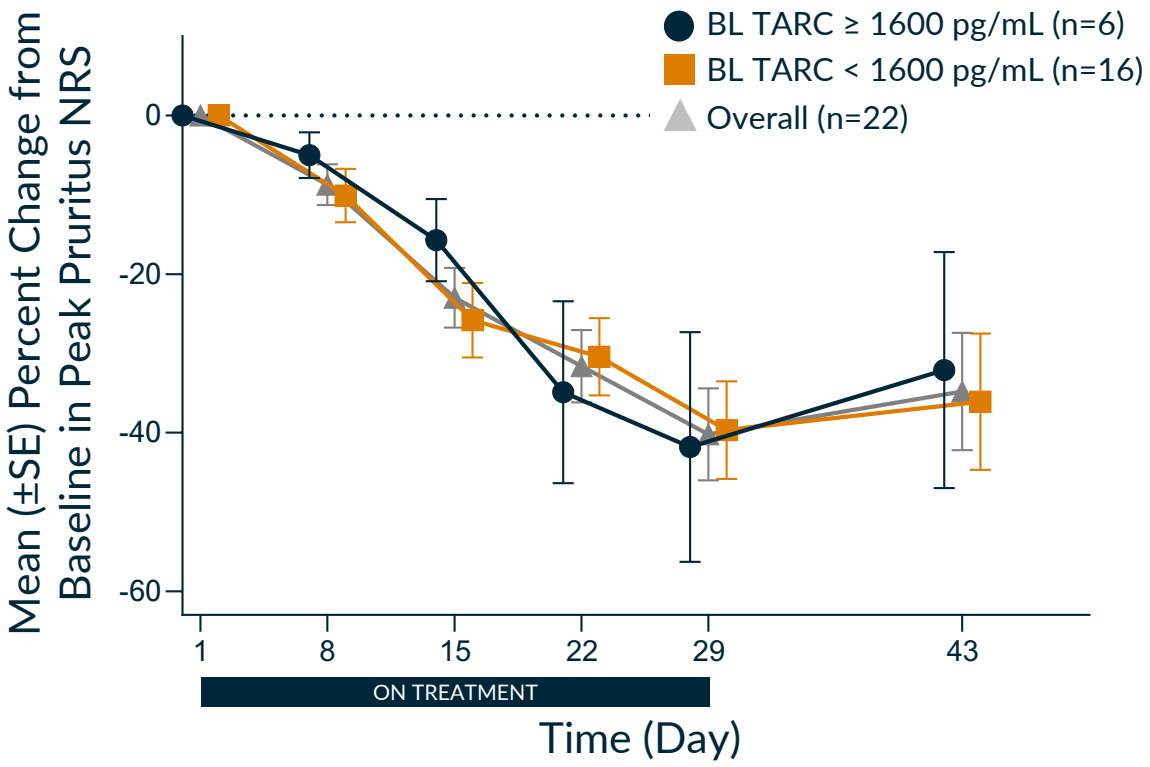
Dose	Mean % Change from Baseline at D29
100 mg	-40%
200 mg	-47%
Overall	-44%

- KT-621 achieved rapid and robust mean Peak Pruritus NRS and SCORAD-Itch reduction in both dose cohorts

Note: Analysis based on observed cases at each visit; [1]One patient in the 100 mg cohort missed the D29 visit (n=9 for 100 mg and n=21 for Overall at D29); PPNRS: Peak Pruritus Numerical Rating Scale; SCORAD: SCORing Atopic Dermatitis.

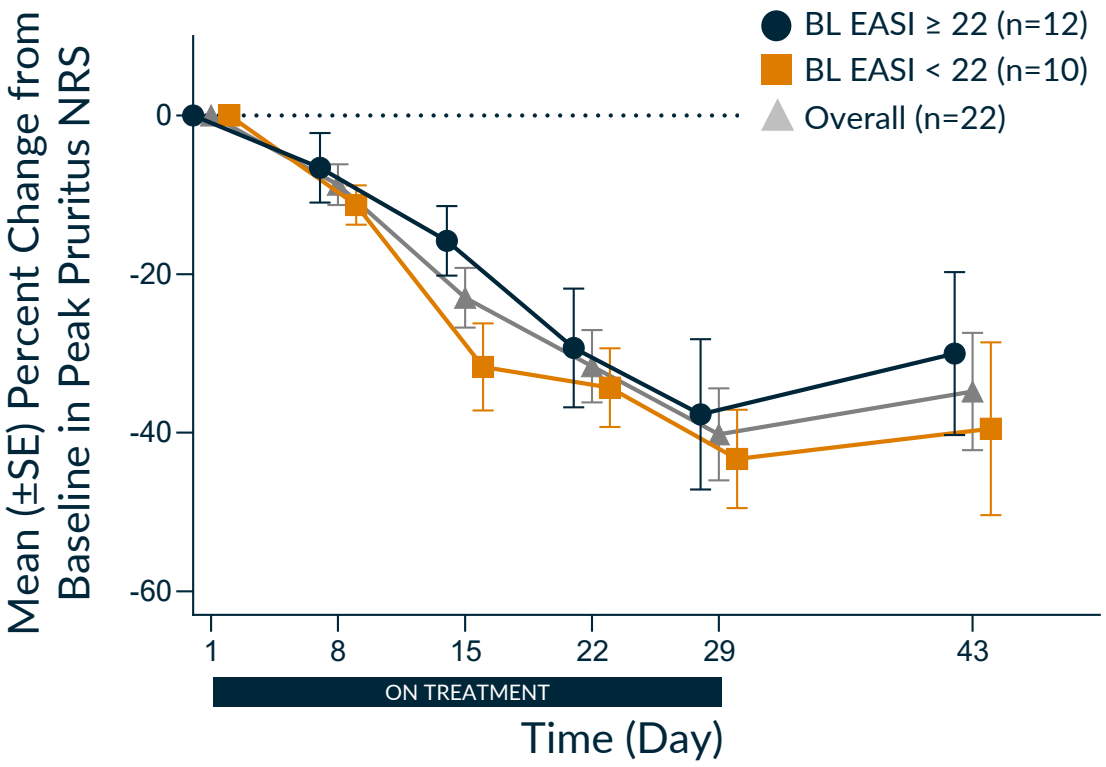
KT-621 Achieved Consistent Reduction of Peak Pruritus NRS Across All Patient Subgroups, Regardless of Baseline TARC/EASI

Mean % Change in PPNRS: Stratified by Baseline TARC



Baseline TARC	Mean % Change from Baseline in PPNRS at D29
≥ 1600	-42%
< 1600	-40%
Overall	-40%

Mean % Change in PPNRS: Stratified by Baseline EASI



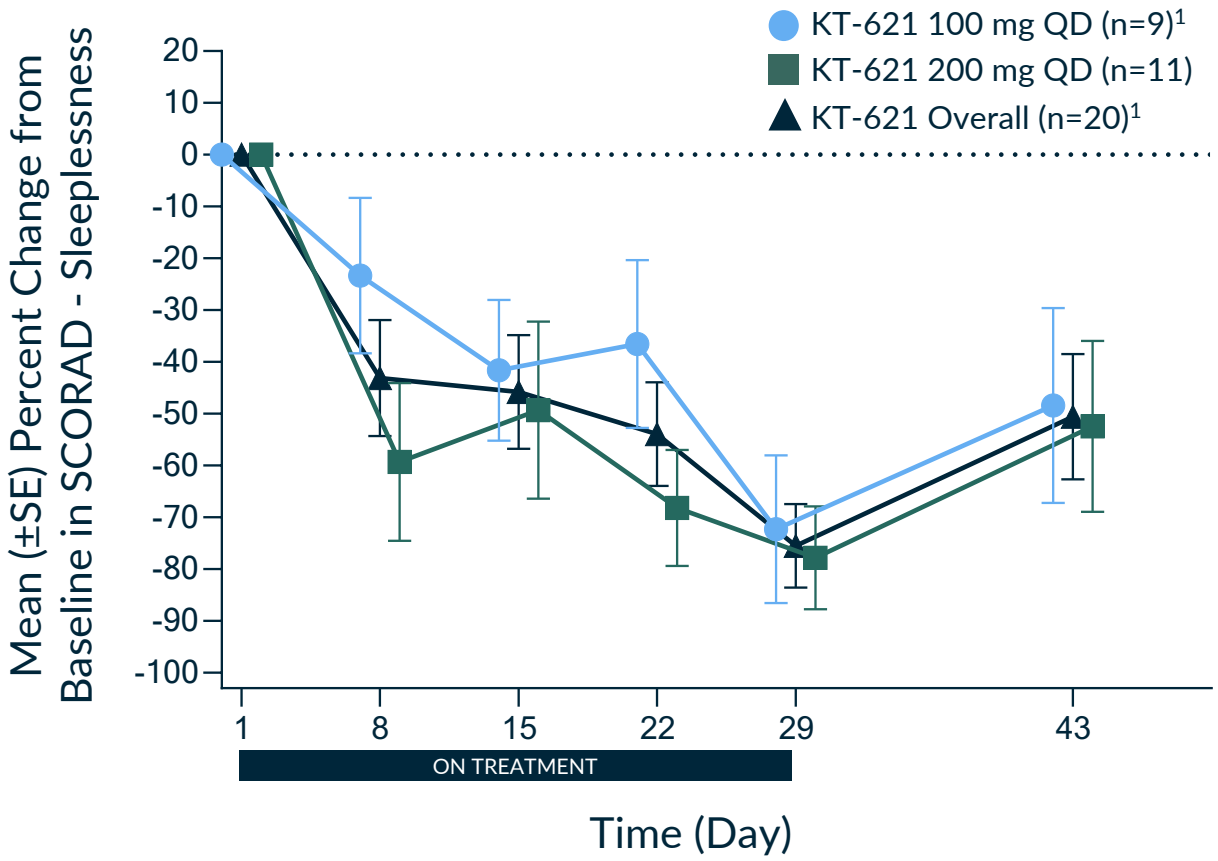
Baseline EASI	Mean % Change from Baseline in PPNRS at D29
≥ 22	-38%
< 22	-43%
Overall	-40%

Note: Analysis based on observed cases at each visit. PPNRS: Peak Pruritus Numerical Rating Scale; EASI: Eczema Area and Severity Index.

KT-621 Achieved 76% Overall Mean Reduction in SCORAD-Sleeplessness

- SCORAD-Sleeplessness is a validated measure of AD severity, capturing associated quality-of-life impact
- KT-621 achieved rapid and robust mean SCORAD-Sleeplessness reduction in both dose cohorts

Mean % Change in SCORAD - Sleeplessness

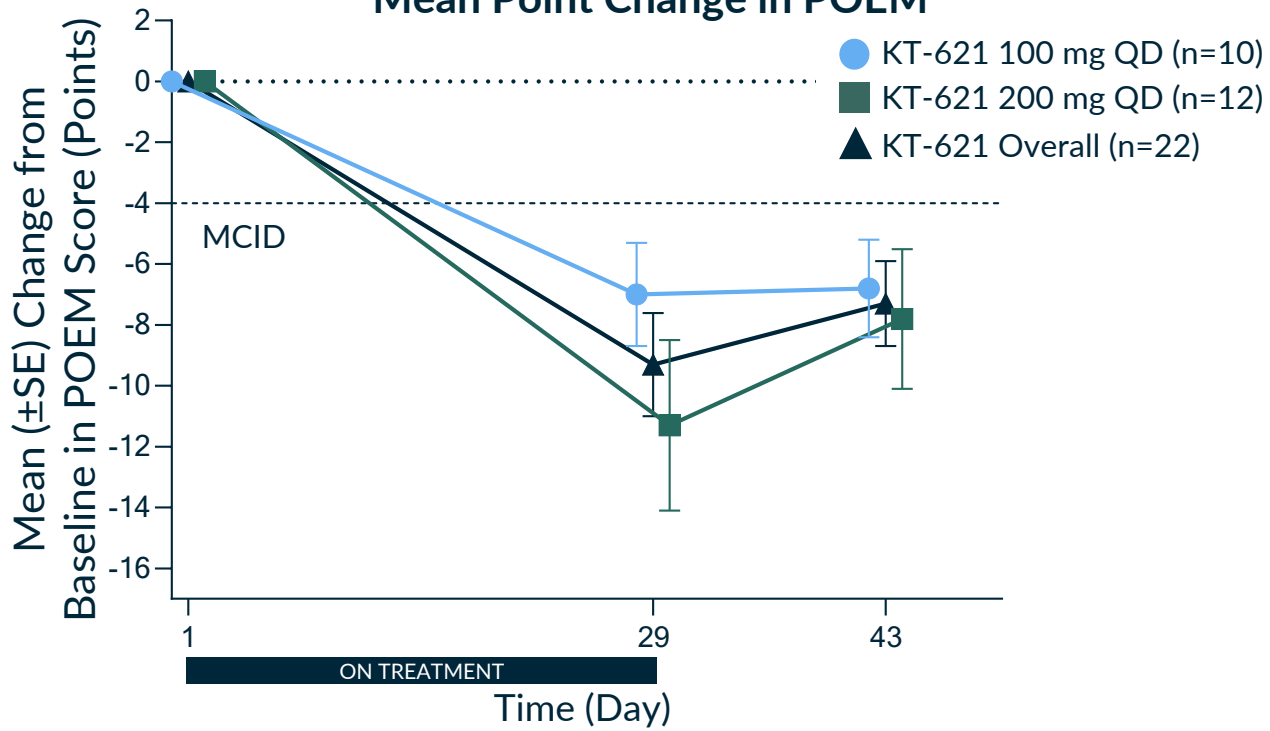


Dose	Mean % Change from Baseline at D29
100 mg	-72%
200 mg	-78%
Overall	-76%

Note: Analysis based on observed cases at each visit, only patients with non-zero baseline sleeplessness are included; [1]One patient in the 100 mg cohort missed the D29 visit (n=8 for 100 mg and n=19 for Overall at D29); SCORAD: SCORing Atopic Dermatitis.

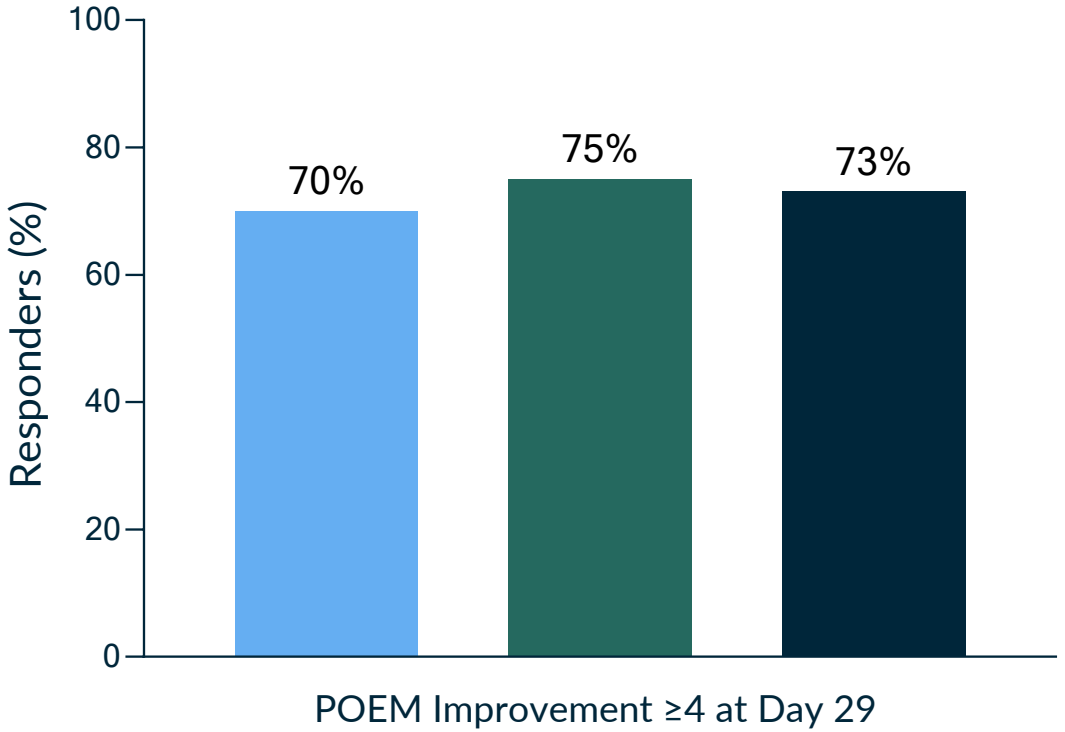
KT-621 Achieved Robust Improvement in Patient Quality of Life (POEM)

Mean Point Change in POEM



Dose	Mean Point Change from Baseline at D29
100 mg	-7
200 mg	-11
Overall	-9

POEM Responders (Improvement ≥4 Points)

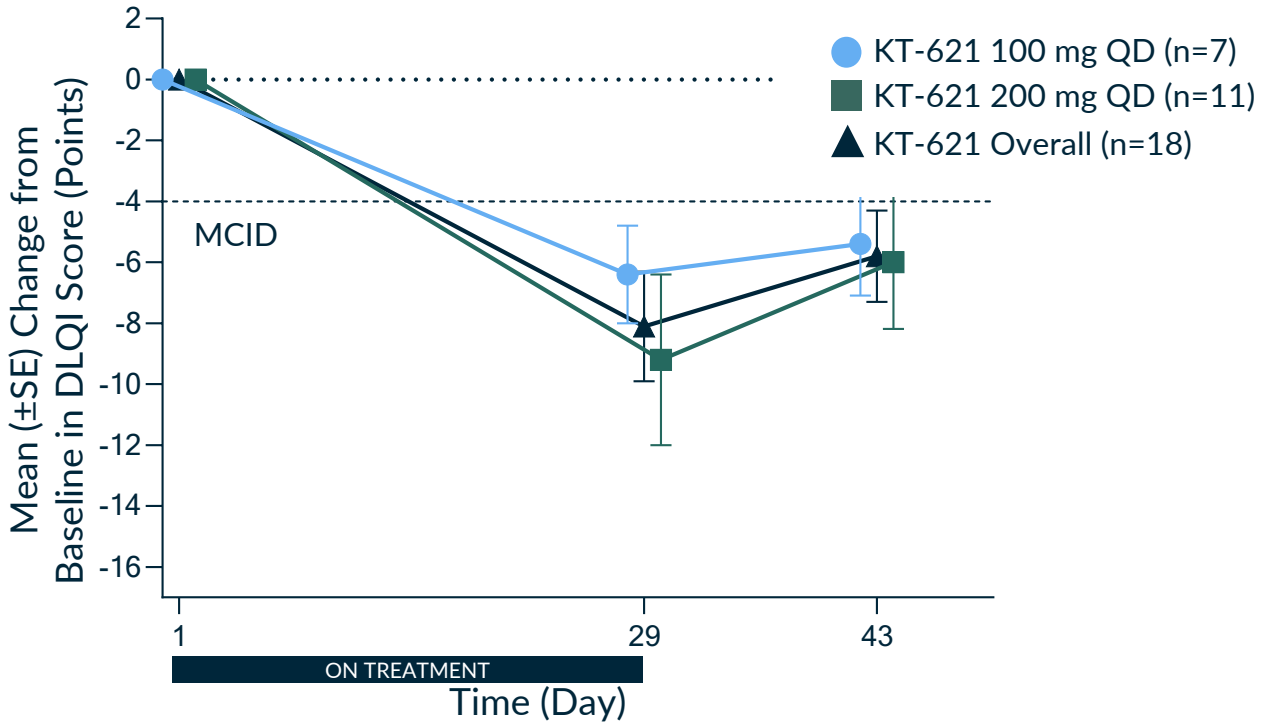


- POEM is a measure evaluating patient experience and impact on quality of life
- Demonstrated improvements are greater than the minimum clinically important difference (MCID)

Note: Analysis based on observed cases at each visit; POEM: Patient-Oriented Eczema Measure.

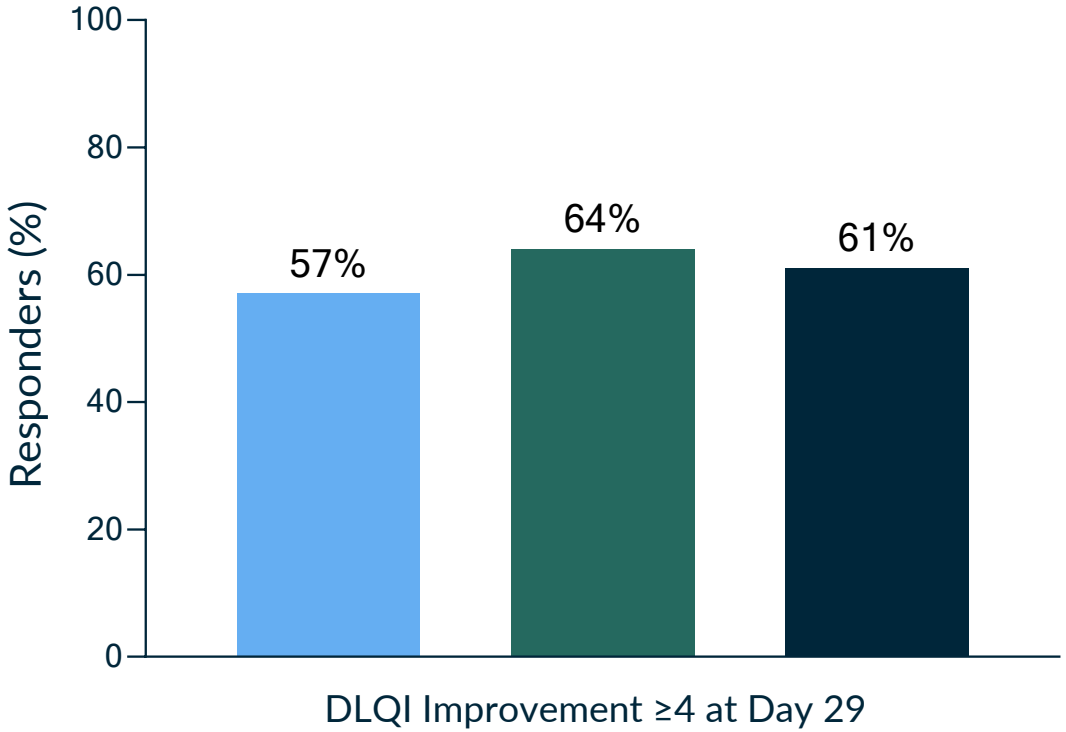
KT-621 Achieved Robust Improvement in Patient Quality of Life (DLQI)

Mean Point Change in DLQI



Dose	Mean Point Change from Baseline at D29
100 mg	-6
200 mg	-9
Overall	-8

DLQI Responders (Improvement ≥ 4 points)



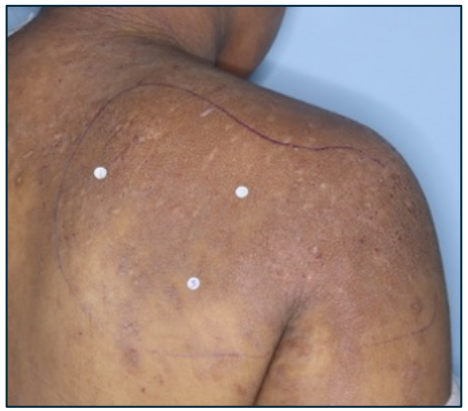
- DLQI is a measure of quality of life validated in AD
- Demonstrated improvements are greater than the minimum clinically important difference (MCID)

Note: Analysis based on observed cases at each visit, only patients with baseline DLQI score ≥ 4 included; DLQI: Dermatology Life Quality Index.

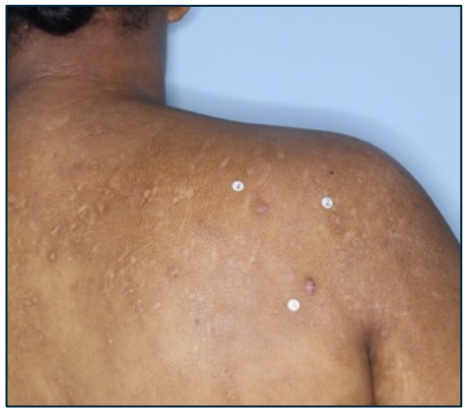
Example of Clinical and Biomarker Response to KT-621 in BroADen Patient with Severe AD and Prior Dupilumab Treatment

Pre and Day 29 Photos for EASI-75 Responder

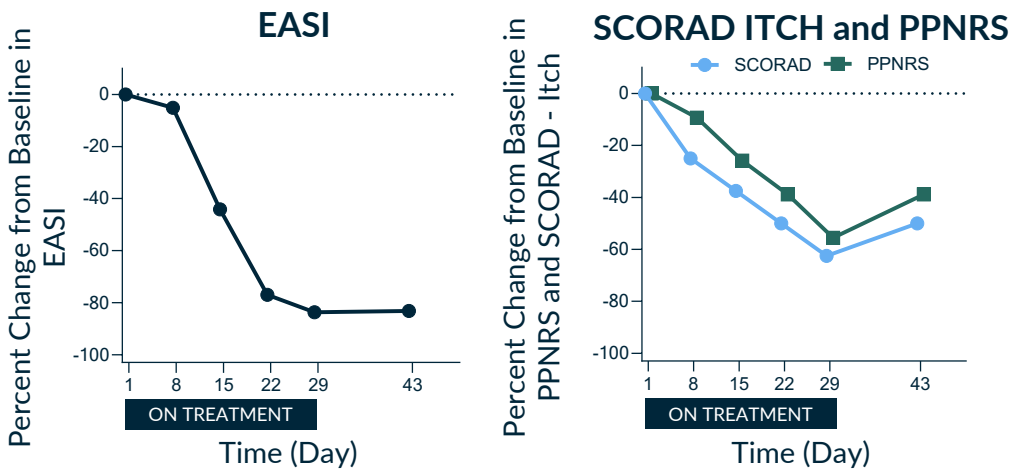
Baseline



Day 29



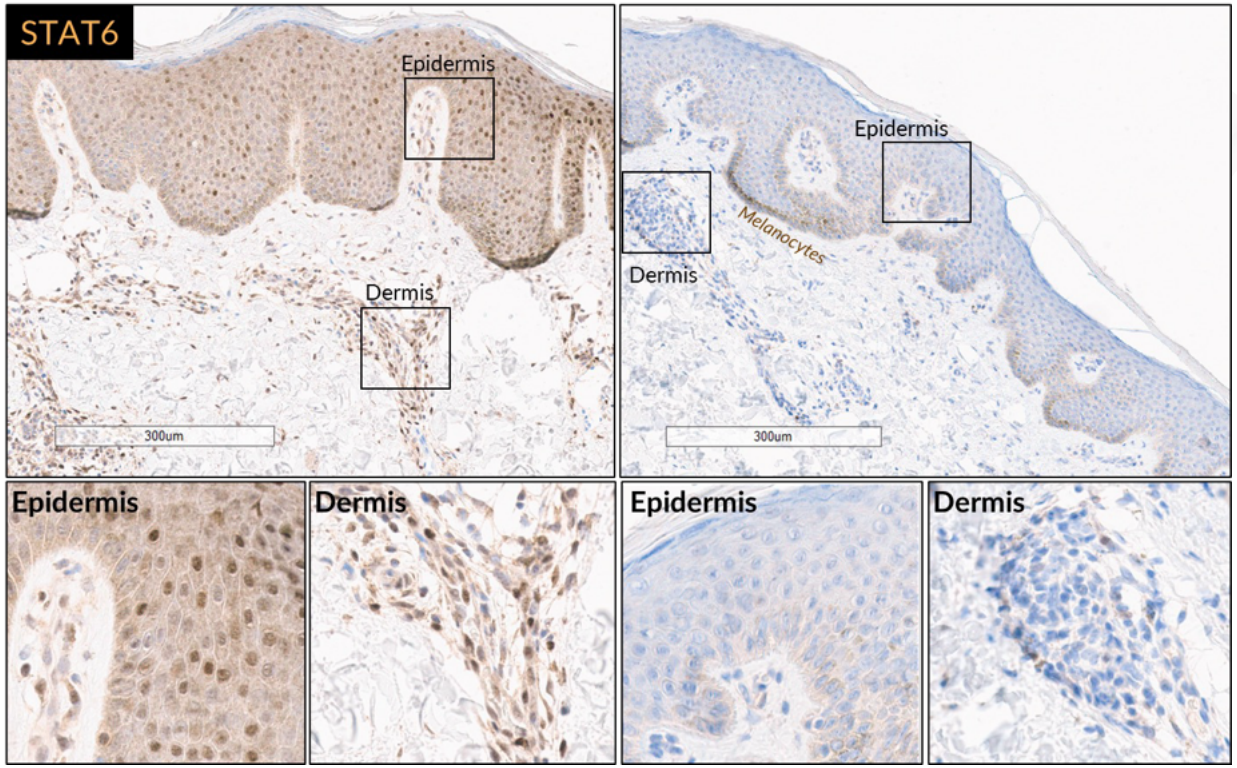
% Change in EASI and Itch[1]



STAT6 Staining IHC

Baseline

Day 29



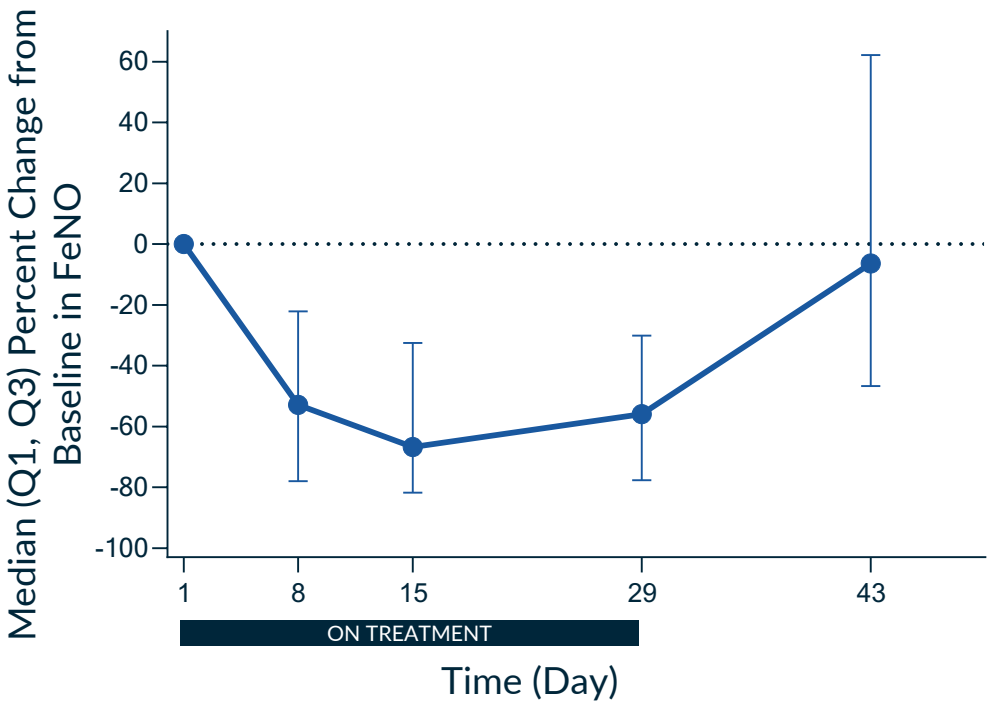
Biomarker Changes at D29

	Blood STAT6	Skin STAT6	Serum TARC	Serum Eotaxin-3	Serum IgE	Skin Eotaxin-3	Skin KRT16
Change from Baseline	-95%	-94%	-78%	-96%	-19%	-92%	-93%

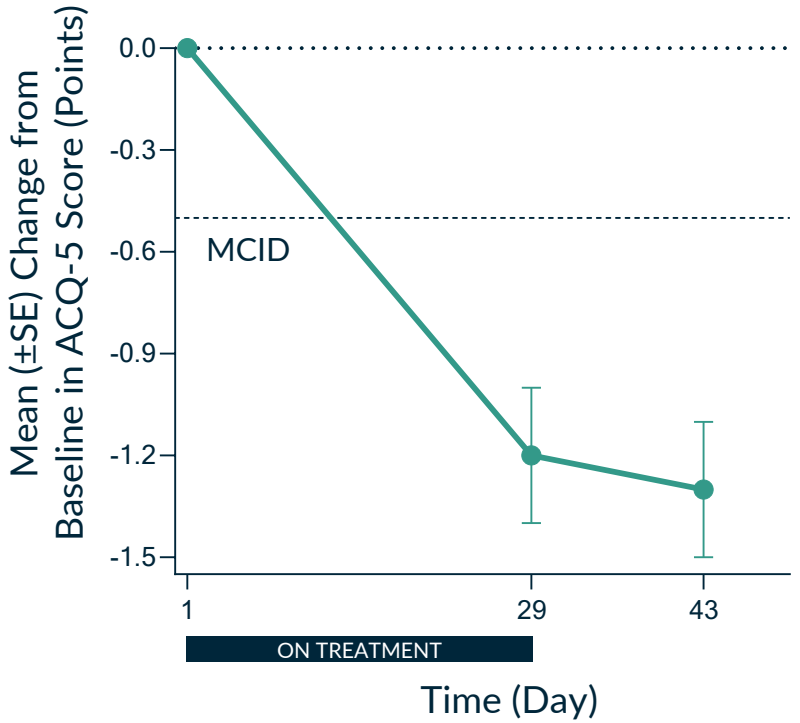
[1]Baseline EASI = 37.4, Baseline Pruritus NRS = 7.7; EASI: Eczema Area and Severity Index; SCORAD: SCORing Atopic Dermatitis; PPNRS: Peak Pruritus Numerical Rating Scale; IHC: Immunohistochemistry.

KT-621 Achieved Robust Impact on FeNO and ACQ-5 in AD Patients with Comorbid Asthma

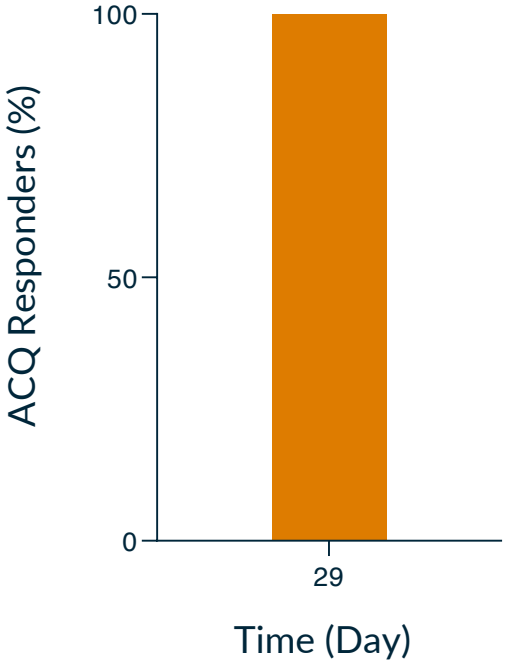
Median % Change from Baseline in FeNO (n=4)



Mean Point Change in ACQ-5 (n=4)



ACQ-5 Responders (Improvement ≥ 0.5, n=4)

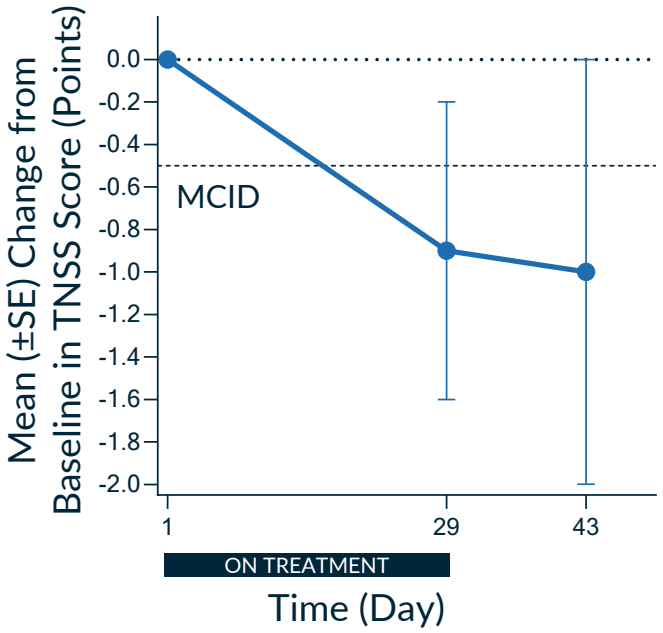


- KT-621 achieved 56% median FeNO reduction at Day 29, exceeding dupilumab (31%) in asthma studies at week 4[1]
- All 4 patients had clinically meaningful reduction in ACQ-5 (mean change of -1.2 points) and a 100% responder rate

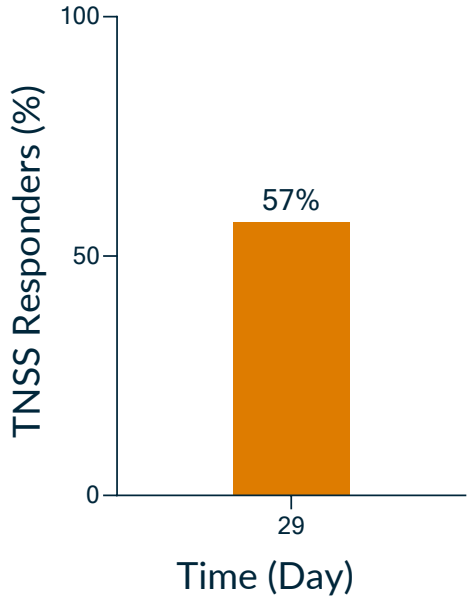
Note: Analysis based on observed cases at each visit; Mean baseline FeNO of 49 ppb and ACQ-5 of 2.5; [1]Pavord et al. JACI. 2024; FeNO: Fractional Exhaled Nitric Oxide; ACQ-5: Asthma Control Questionnaire-5; MCID: Minimum Clinically Important Difference.

KT-621 Achieved Robust Impact on TNSS and RQLQ in AD Patients with Comorbid Allergic Rhinitis

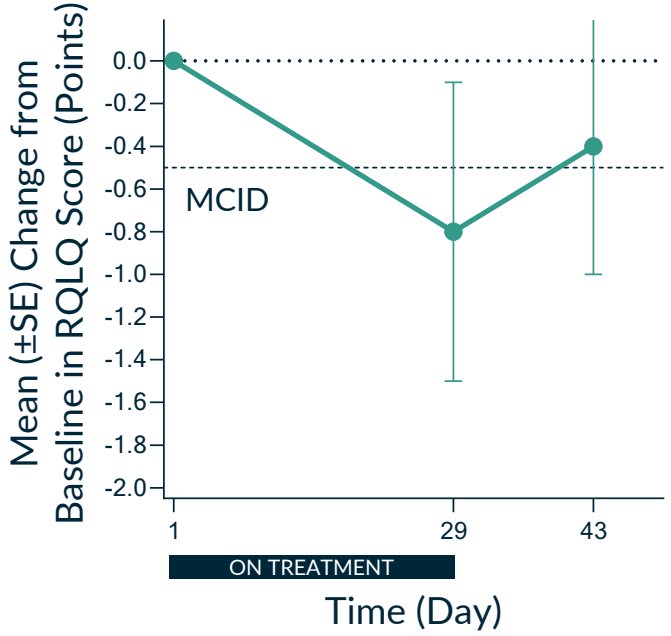
Mean Point Change in TNSS (n=7)[1]



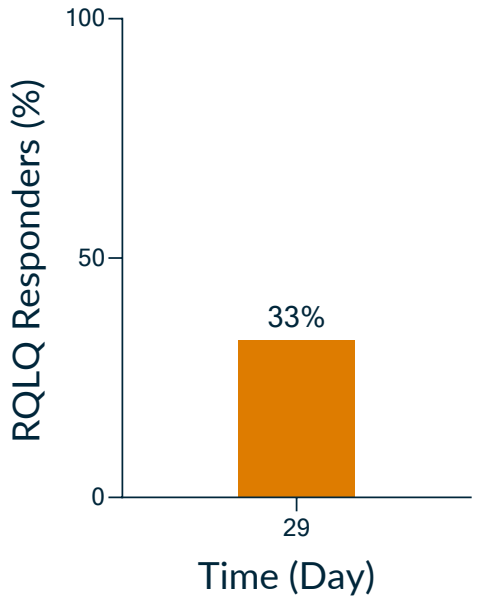
TNSS Responders (Improvement ≥ 0.55, n=7)[1]



Mean Point Change in RQLQ (n=6)[2]



RQLQ Responders (Improvement ≥ 0.5, n=6)[2]



- At Day 29, KT-621 achieved mean changes of –0.9 and -0.8 points in TNSS and RQLQ, respectively
- TNSS and RQLQ responder rates were 57% and 33%, respectively

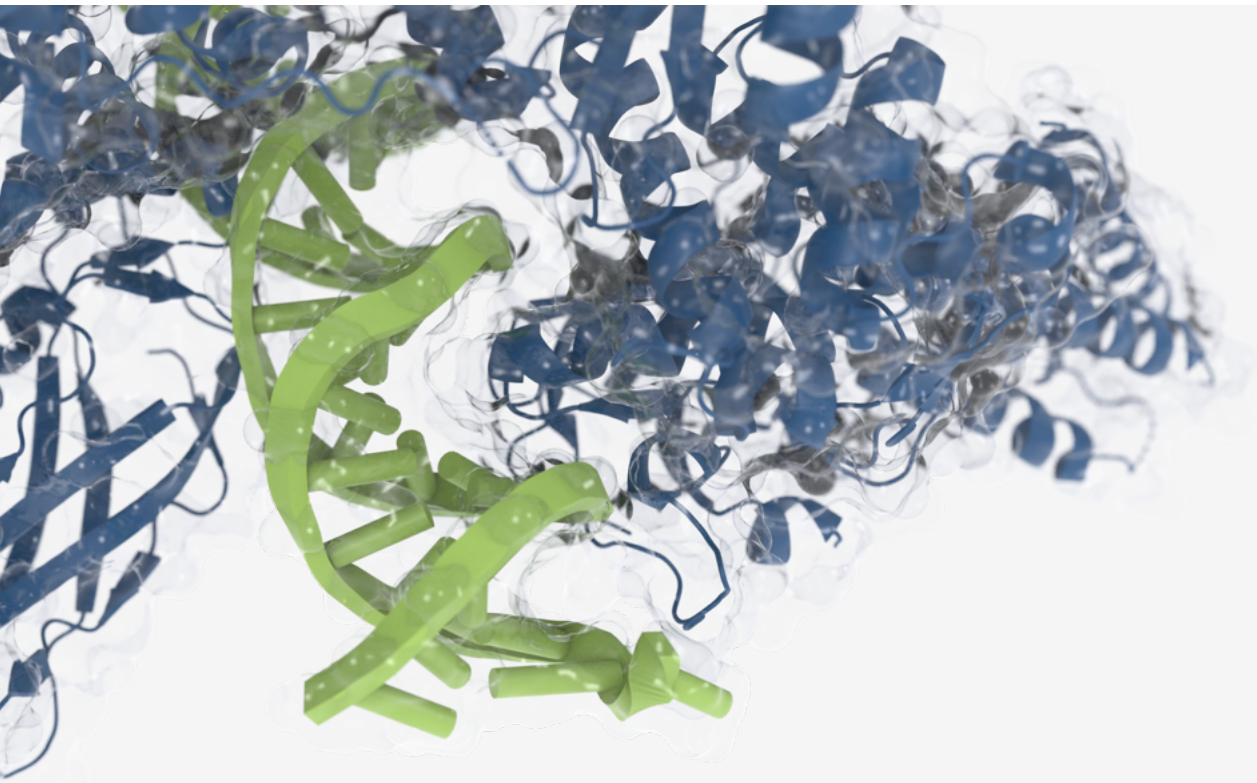
Note: Analysis based on observed cases at each visit; [1]Only patients with baseline TNSS score ≥ 0.55 are included; [2]Only patients with baseline RQLQ score ≥ 0.5 are included; Mean baseline TNSS and RQLQ of 4.4 and 2.4, respectively; MCID: Minimum Clinically Important Difference; TNSS: Total Nasal Symptom Score; RQLQ: Rhinoconjunctivitis Quality of Life Questionnaire.

KT-621 Demonstrated Robust Improvements Across All Key Clinical Efficacy Endpoints

Results in Line With or in Some Cases Numerically Exceeded Published Data for Dupilumab at Week 4

	KT-621 100 mg QD Day 29 (n=10)	KT-621 200 mg QD Day 29 (n=12)	KT-621 Overall Day 29 (n=22)	Dupilumab 300 mg Q2W D28 Ph3 (n=457)[1]
Mean % Change in EASI	-62%	-63%	**-63%**	**-52%**
EASI-50	67%	83%	**76%**	**57%**
EASI-75	33%	25%	**29%**	**28%**
Mean % Change in PPNRS	-47%	-35%	**-40%**	**-33%**
Mean % Change in SCORAD	-52%	-46%	**-48%**	**-41%[2]**
vIGA-AD 0 and 1	22%	17%	**19%**	**12%**
Mean % Change in % Body Surface Area	-55%	-44%	**-49%**	**-36%[2]**
Mean % Change in SCORAD - Sleeplessness	-72%	-78%	**-76%**	**NR**
Mean % Change in SCORAD - Itch	-40%	-47%	**-44%**	**NR**
POEM Responders	70%	75%	**73%**	**69% at Week 16**
DLQI Responders	57%	64%	**61%**	**69% at Week 16**

[1]Dupilumab Phase 3 data derived or digitized from the pooled data of SOLO1 and SOLO2 studies (Thaci et al, Dermatological Science, 2019; Cather et al, Dermatol Ther, 2022); [2]Data from 64 patients in dupilumab Phase 2b study (Thaci et al, Lancet, 2016); EASI: Eczema Area and Severity Index; vIGA-AD: Validated Investigator Global Assessment for Atopic Dermatitis; PPNRS: Peak Pruritus Numerical Rating Scale; SCORAD: SCORing Atopic Dermatitis; NR: Not Reported.



Safety

KT-621 BroADen Phase 1b Safety Summary

- Well-tolerated with favorable safety at both 100 mg and 200 mg with a profile similar to what was observed in the Phase 1a healthy volunteer trial
- No SAEs or Severe AEs
- No dose-dependent pattern in the TEAEs
- No related TEAEs or TEAEs leading to discontinuation
- No AEs of conjunctivitis (or of any ocular disorder), herpes infections, or arthralgias
- No clinically relevant changes in vital signs, laboratory tests or ECGs

AE: Adverse Event; SAE: Serious Adverse Event; TEAE: Treatment Emergent Adverse Event; ECG: Electrocardiogram.

Positive BroADen Phase 1b Data Underscore Significant Potential of KT-621 in AD and Other Type 2 Allergic Diseases

✓ **Once-a-day, Oral Dosing of KT-621 Achieved Deep STAT6 Degradation in Blood and Skin with Strong Translation from Healthy Volunteers**

✓ **Strong Effect on Disease-relevant Type 2 Inflammation Biomarkers Demonstrating IL-4/IL-13 Pathway Inhibition**

- Dupilumab-like reductions in TARC, Eotaxin-3 and IgE
- Reduction of IL-31, key cytokine elevated in AD driving pruritus
- Dupilumab-like reductions in core Type 2 inflammation and AD disease-relevant gene sets in skin lesions
- First known demonstration of FeNO reduction in AD patients, providing POC for KT-621 inhibition of Type 2 inflammation in lungs in addition to blood and skin

✓ **Robust Clinical Endpoint Impact**

- Dupilumab-like impact on EASI, SCORAD and multiple measures of pruritus as well as on sleeplessness and quality of life; no rescue therapy required during treatment period
- Similar effect of KT-621 in patients with low or high baseline EASI/TARC and in patients with or without prior biologics
- Early evidence of activity in asthma and allergic rhinitis based on improvement in biomarkers and/or patient-reported outcomes in AD patients with these comorbid conditions

✓ **Excellent Tolerability**

- KT-621 was well tolerated, with safety profile similar to results in the Phase 1a healthy volunteer trial

EASI: Eczema Area and Severity Index; Scale; SCORAD: SCORing Atopic Dermatitis.

BroADen Phase 1b Summary

Nello Mainolfi, PhD

Founder, President and CEO

Kymera Completes the Clinical Translation of STAT6 Degradation

Study	Data	Potential for "Dupilumab-in-a-pill" Profile
Human Genetics	STAT6 is a key driver of Type 2 inflammation	✓
Preclinical	KT-621 degraded STAT6 and blocked IL-4/13 Type 2-driven inflammation *in vitro/in vivo* as effectively as dupilumab	✓
Phase 1a Healthy Volunteers	KT-621 safely and deeply degraded STAT6, blocking IL-4/13 biomarkers equally or numerically better than dupilumab	✓
BroADen Phase 1b AD Patients	KT-621 safely and deeply degraded STAT6 and demonstrated meaningful improvements on: - Type 2 biomarkers in blood and skin - FeNO in AD and comorbid asthma patients - Clinical endpoints in patients with AD, comorbid asthma and allergic rhinitis **Results in line with or in some cases numerically exceeded published data for dupilumab at week 4**	✓

KT-621 clinical data continues to support STAT6 degradation as a potentially transformative approach for Type 2-driven inflammatory diseases, with a once-a-day, oral drug

BroADen Phase 1b Data Achieved Objectives and Underscore Significant Potential of KT-621

Summary & Upcoming Milestones

- KT-621 BroADen AD patient data represent **strong translation** from Phase 1a healthy volunteer trial results

- Results further validate KT-621's **oral, biologics-like profile** with potential to **broaden clinical access for patients and disrupt a biologics-dominated Type 2 market** in AD, asthma, and beyond

- Study supports continued development, including:
  - **BROADEN2** Phase 2b study in AD (ongoing)
  - **BREADTH** Phase 2b study in asthma (start 1Q26)

Thank You

Q&A

To ask a question, raise your virtual hand

KYMERA