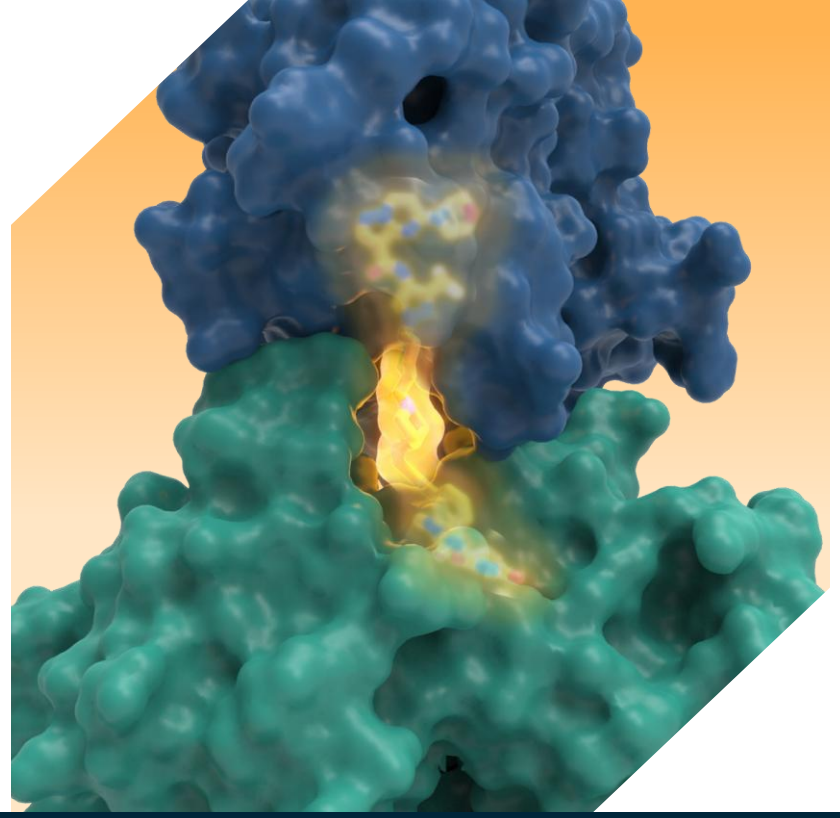


KYMERA

June 2, 2025

KT-621 / STAT6 Degrader Phase 1 Healthy Volunteer Results

Revolutionizing Immunology with Oral Small Molecule Degrader Medicines

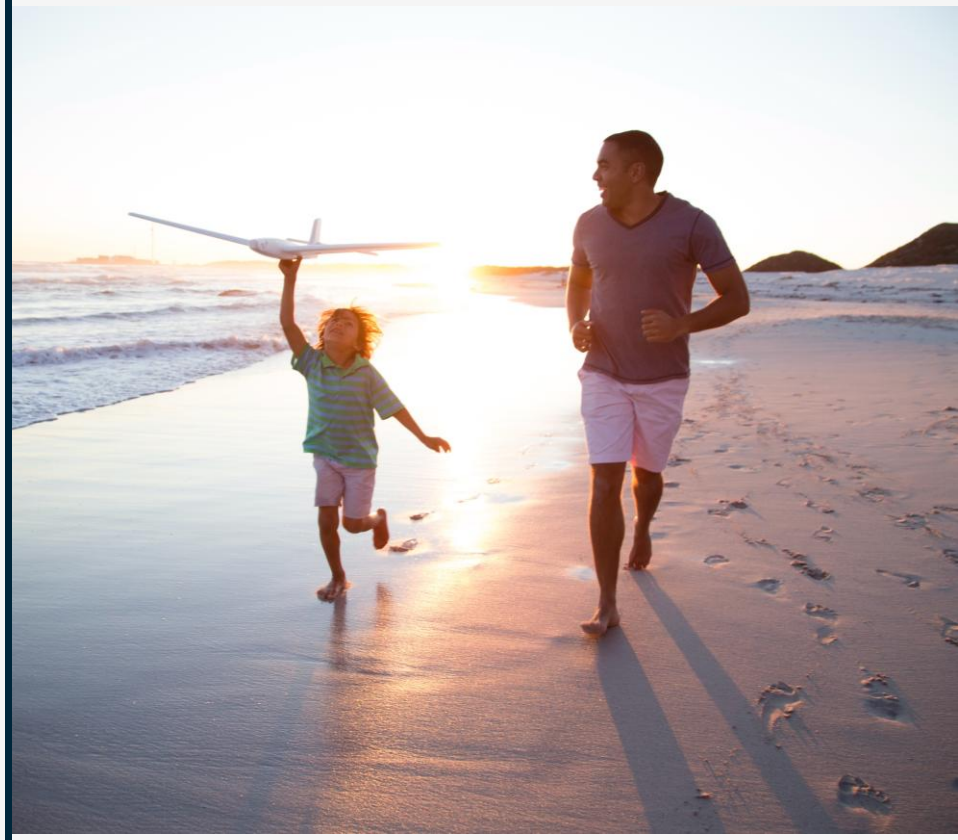
Agenda

Introduction
Justine Koenigsberg
Vice President, Investor Relations

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

KT-621 Phase 1 Healthy Volunteer Data
Jared Gollob, MD
Chief Medical Officer

Question and Answer Session



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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; the preliminary cross-study assessments comparing non-head-to-head clinical data of KT-621 to published data for dupilumab; 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 by 2026; 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; and our financial condition and expected cash runway into the first 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.

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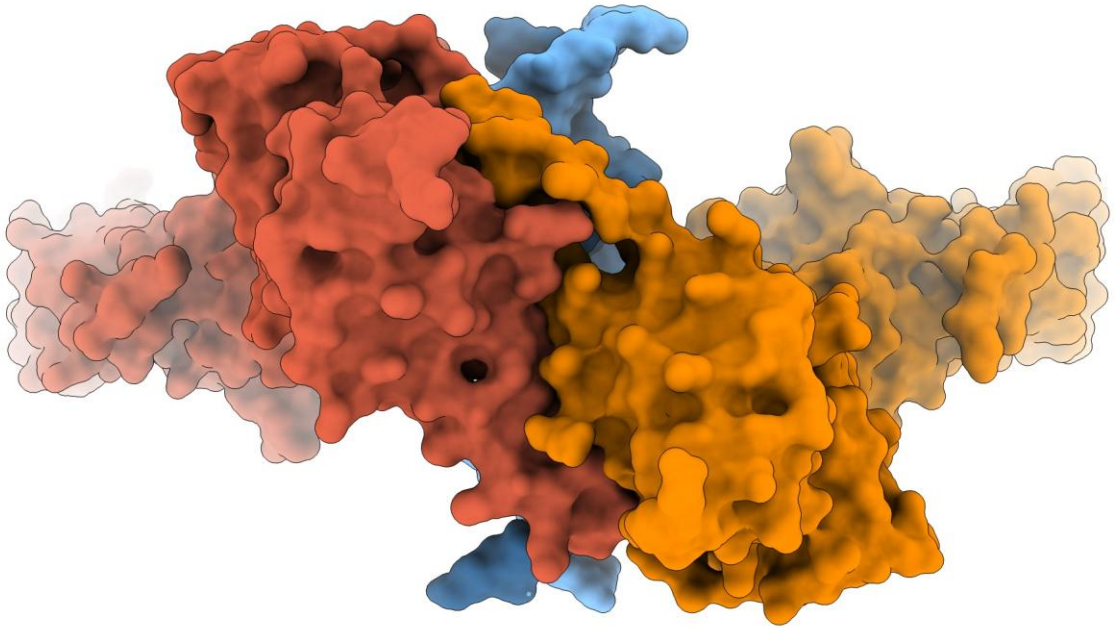
Revolutionizing Immunology with Oral Medicines

Nello Mainolfi, PhD,

Founder, President and CEO

Kymera: Revolutionizing Immunology with Oral Medicines

- Targeted Protein Degradation (TPD) leader
- Best-in-industry capabilities in hit finding and optimization of oral degraders
- Unique target selection strategy pursuing traditionally undrugged targets in highly validated pathways
- Portfolio poised to disrupt conventional treatment paradigms



By combining the **"right target" with the disruptive potential of TPD, Kymera is delivering oral therapies with biologics-like profiles** for the first time in industry with the potential to expand access to millions of patients around the world

A Proven Track Record of Reproducible Innovation

Productive, Reproducible and Scalable Innovation Engine for High Value Immunology Targets

Technology Leader
Capabilities and know-how to develop highly potent/specific oral degraders for undrugged proteins

High Productivity
9 development candidates
5 INDs
Dosed >400 individuals across pipeline

Leading I&I Innovation
First-in-class programs addressing historically undrugged and inadequately drugged targets

Exceptional Translation
>90% target degradation with desired tolerability and strong clinical activity across all clinical programs

Committed to building a fully-integrated, global, commercial biotech company, capitalized to execute with $775M[1] in cash and runway to the first half of 2028

[1]As of March 31, 2025.

Unique Target Selection Strategy Drives Best-In-Class Pipeline

Strong genetic/clinical validation

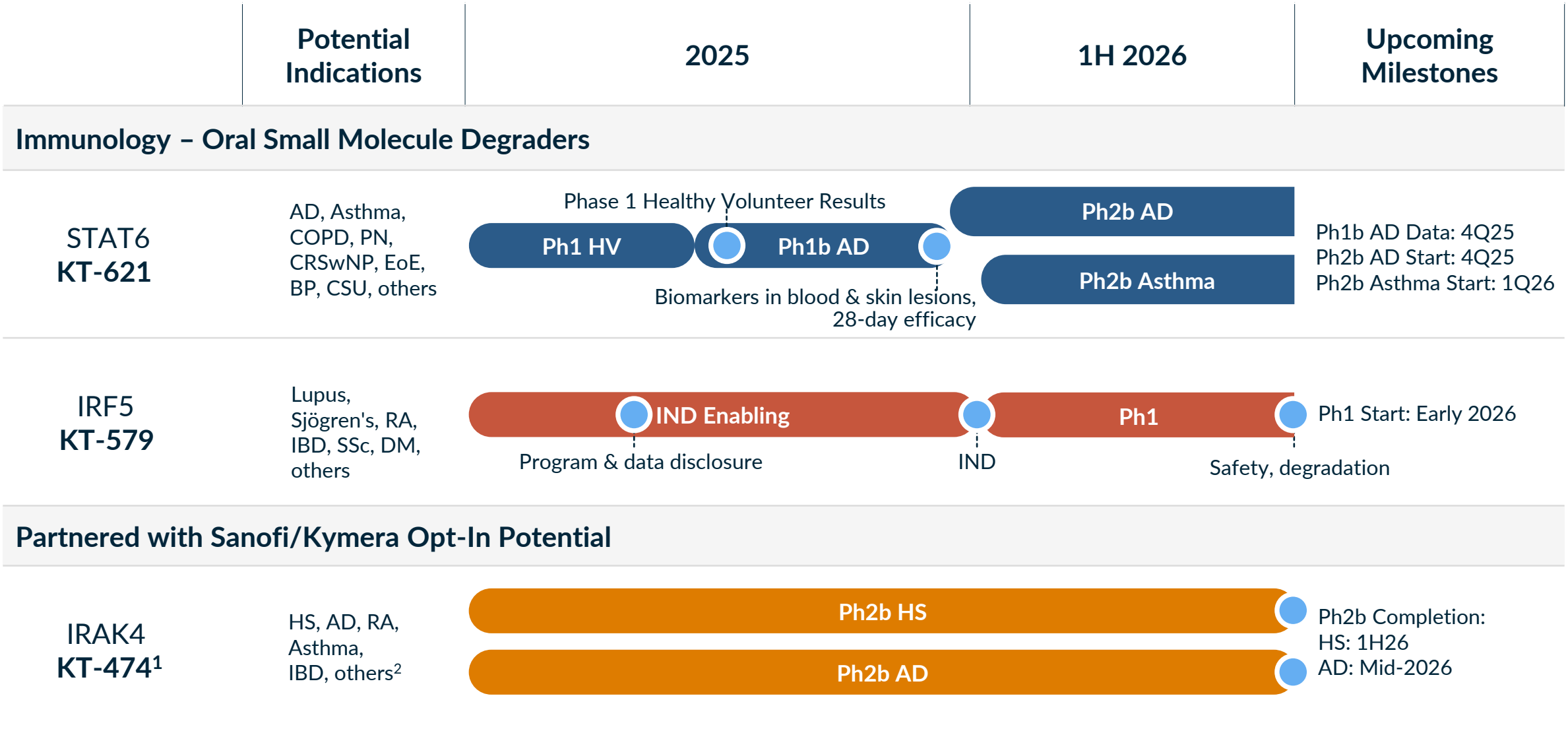
Undrugged or inadequately drugged targets

Large clinical/ commercial opportunities

Clear path to early clinical differentiation

FOCUS ON FIRST- AND BEST-IN-CLASS OPPORTUNITIES

Building a Best-In-Industry Oral Immunology Pipeline



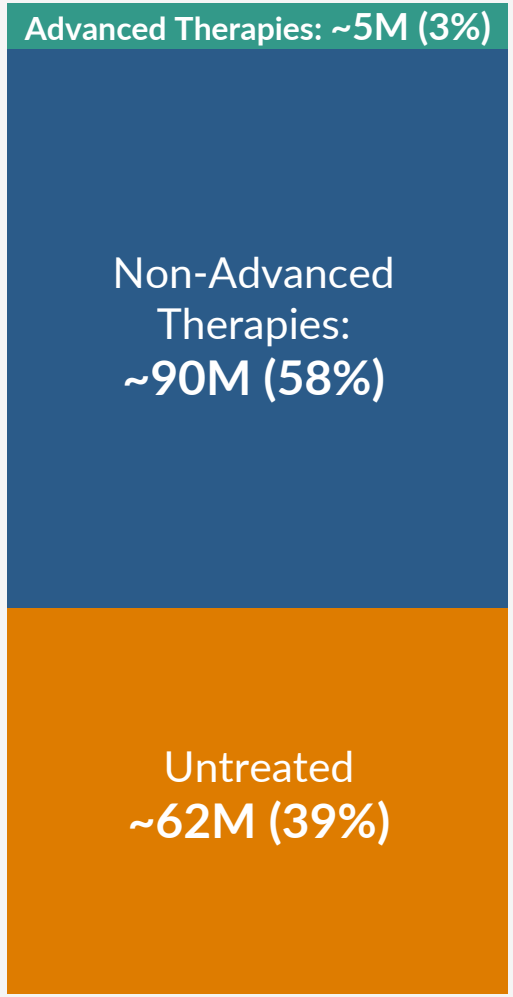
[1]KT-474 (SAR444656) partnered with Sanofi, with Kymera option to participate in the development and commercialization, and 50/50 profit split, in the United States. Double digit tiered royalties in ROW; [2]Current indications: HS and AD. Other diseases shown, where IL-1R/TLR pathway has been implicated in pathogenesis, are additional potential opportunities.

Immunology: A Large, Underserved Market

~160M Total Patients Across Key Immunologic Diseases[1]

>$100B

- **~5M patients (3% of total diagnosed)** are on systemic advanced therapies with **>$100B** in annual sales for key I/I indications[2]
- **2/3 of those therapies are injectable biologics**



>$500B?

- ~ **97%** of the diseased population does not have access to advanced therapies
- Enormous untapped market potential

Oral Degraders with Biologics-Like Profiles Can Disrupt the Immunology Market

- **Displace injectable biologics**
- **Offer a convenient highly effective advanced therapy** to the 97% unserved and underserved patients

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

Small Molecule Oral Degraders Can Transform Immunology

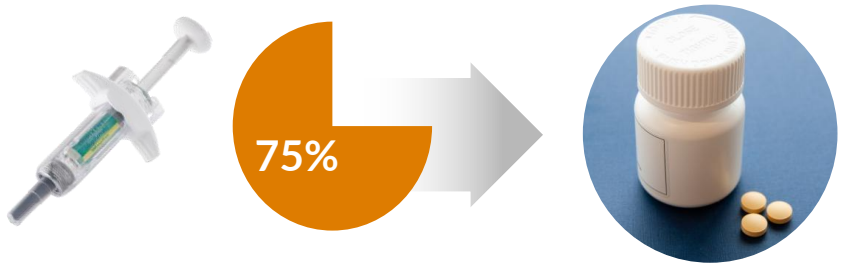
Potentially Superior Profile to Injectable Biologics and Traditional Small Molecule Inhibitors (SMIs)

Biologics can have several limitations, making orals preferred by most patients

DUPIXENT® (dupilumab) Injection

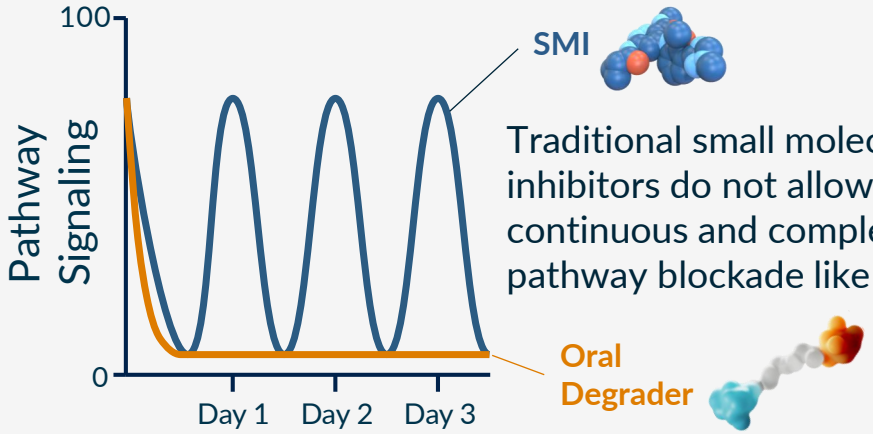
Skyrizi® risankizumab-rzaa

- Expensive, challenging to prescribe/reimburse
- Immunogenicity
- Cold storage
- Inconvenient and/or painful route of administration for patients

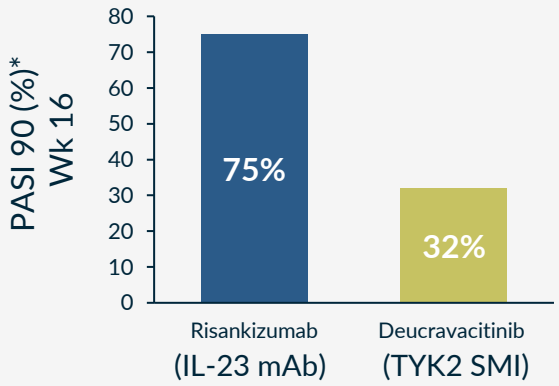


In industry surveys[1], **75%** of patients would switch from injectable biologics to oral with similar profile

Traditional SMIs insufficiently block pathways, which can limit efficacy



Traditional small molecule inhibitors do not allow continuous and complete pathway blockade like degraders

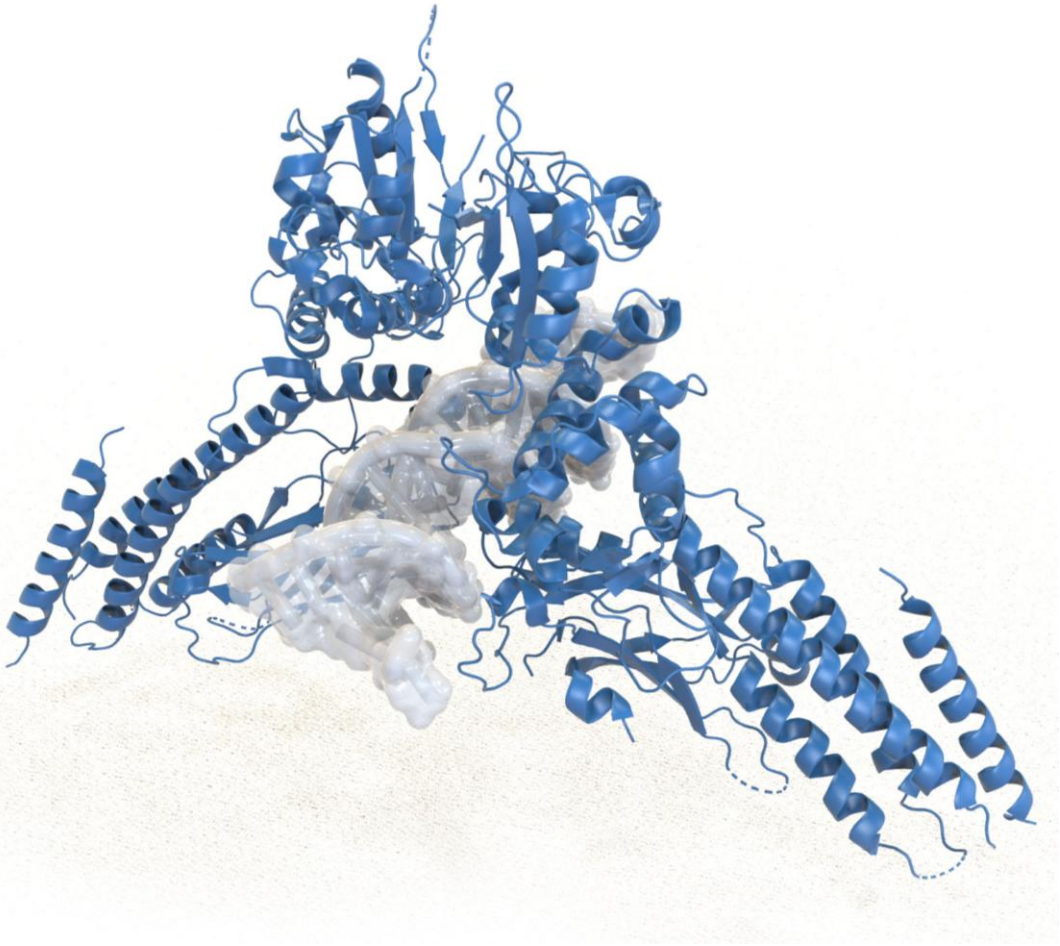


Anti IL-23 biologic significantly more effective than TYK2 SMI in PsO[2]

[1]J&J Business Review Dec '23 (survey of N=395 patients with moderate-to-severe psoriasis); [2]Skyrizi (IL-23 mAb) and Sotyktu (TYK2 SMI) package inserts.

STAT6: An Ideal Immunology Target

STAT6
TRANSCRIPTION FACTOR



- Strong genetic/clinical validation
- Undrugged or inadequately drugged targets
- Large clinical/commercial opportunities
- Clear path to early clinical differentiation

STAT6: Highly Validated Genetically and Clinically

STRONG GENETIC/ CLINICAL VALIDATION

STAT6 is the Key Signaling Node for IL-4 and IL-13

- STAT6 is the obligate and specific transcription factor for IL-4/IL-13, which only signal through STAT6
- STAT6 targeting should phenocopy IL-4/IL-13 targeting

IL-4/IL-13 Pathway Highly Validated by Dupilumab

- Dupilumab is approved in 7 indications
  - AD, Asthma, COPD, PN, CRSwNP, EoE, CSU
- Dupilumab generates >$14B of yearly sales, and is expected to reach $25B by 2030[1]

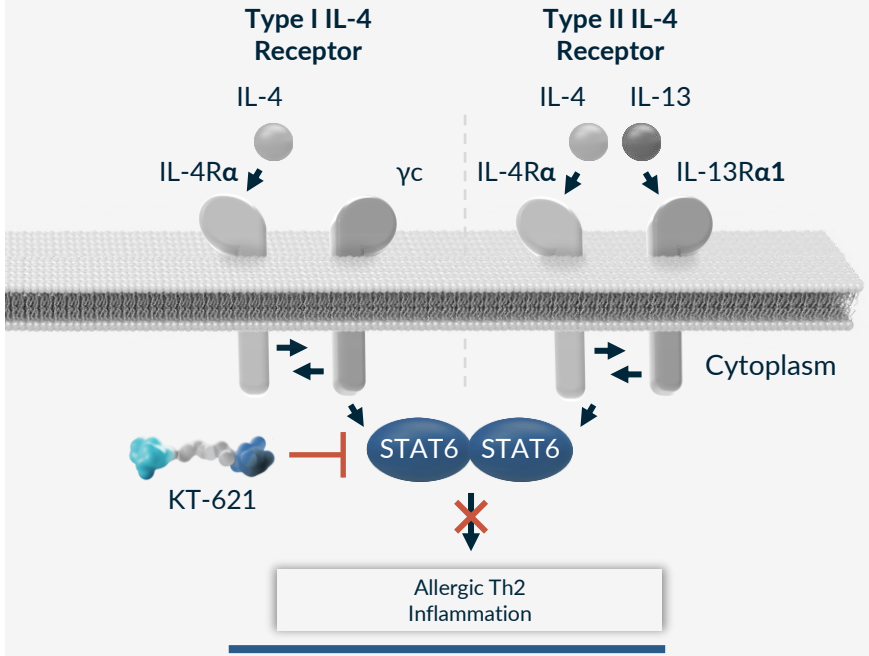
Strong Genetics Support[2]

- Human Gain of Function of STAT6 causes severe allergic disease
- Humans with heterozygous Loss of Function are healthy and protected against Th2 inflammation
- STAT6 knock-out mouse is viable, fertile and healthy

STAT6 degradation should lead to Dupilumab-like efficacy in Th2 diseases with a favorable safety/tolerability profile

STAT6
TRANSCRIPTION FACTOR

IL-4/IL-13 Pathway

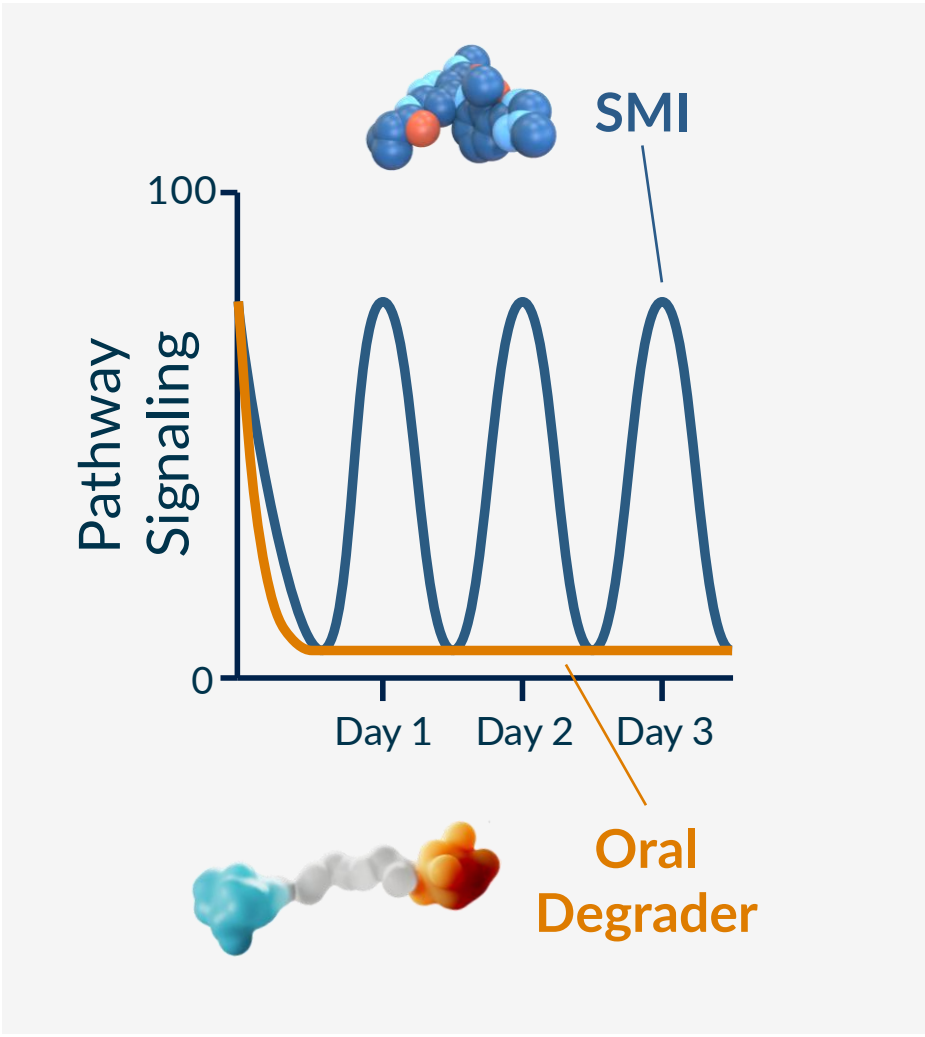


STAT6 is the only specific transcription factor responsible for IL-4/13 signaling

[1]GlobalData Consensus Global Sales Forecast;
[2]Front Immunol. 2018; Sharma et al. J Exp Med. 2023; Suratannon et al. J Allergy Clin. Immunol. 2022; Takeuchi et al. J Allergy Clin Immunol. 2022; Kristjansdottir et al. Journal of Allergy and Clinical Immunology. 2024.

Only STAT6 Degradation Has the Potential to Lead to Dupilumab-Like Pathway Inhibition

- **STAT6,** long pursued immunology “holy-grail” target, **has remained undrugged before KT-621**
- Most oral approaches centered around small molecule inhibitors (SMIs), which have significant limitations
- SMIs do not allow continuous and complete pathway blockade because of the direct PK-PD relationship
- Only degradation is expected to match dupilumab’s pathway blockade with once-a-day oral delivery



Building a STAT6 Franchise That Will Serve All Patients with Th2 Inflammation

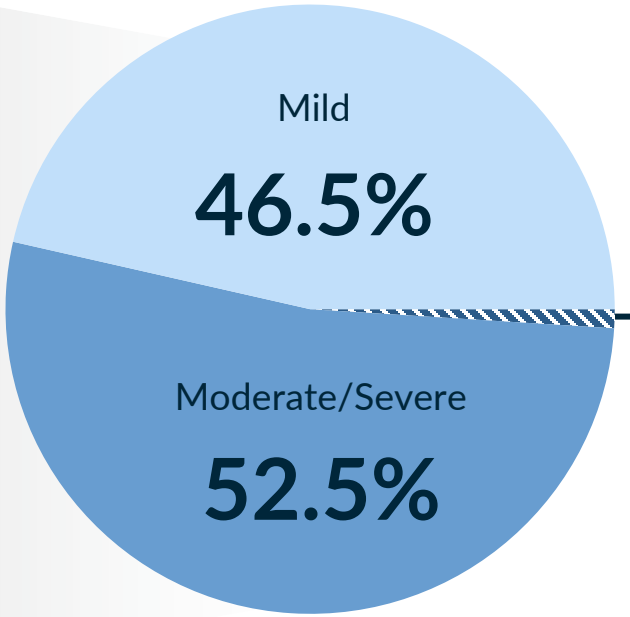
LARGE CLINICAL &COMMERCIAL OPPORTUNITIES

	Patient Numbers[1]
DERMATOLOGY	
AD	42M
CSU	4.5M
PN	305K
BP	166K
GASTROENTEROLOGY	
EoE	1.6M
RESPIRATORY	
Asthma	55M
COPD	34M
CRSwNP	2.8M

Total Potential Patient Impact:

>130M patients[1]

Numerous indications/ therapeutic areas that are de-risked by dupilumab



Only 1% Treated with Systemic Advanced Therapies

>$14B market[1]

An oral STAT6 degrader has the potential of transforming the treatment paradigm for Th2 diseases

[1]GlobalData (2023 diagnosed prevalent patient population and forecasted sales for approved systemic advanced therapies for AD, Asthma, and COPD only in US/EU5/JP).

KT-621 Development Plan Enables Efficient Path to Registration Across All Th2 Diseases

CLEAR PATH TO EARLY CLINICAL DIFFERENTIATION

Initial Parallel Phase 2b Trials in Moderate/Severe Atopic Dermatitis (AD) and Asthma are Expected to Support Subsequent Phase 3 Trials Across Multiple Dermatology, GI and Respiratory Indications

Ph1 HV
SAD-MAD

✓*June 2, 2025 Data Release*

- Safety and tolerability
- STAT6 degradation
- Th2 biomarkers

Ph1b AD
Single Arm, Open Label

Ongoing

- Dosing for 28 days
- Biomarkers in blood and skin lesions
- Clinical activity

Ph2b AD
Dose Ranging

Start in Q4 2025

Ph3 dose

Ph3 AD, PN, EoE, BP, CSU

Ph2b Asthma
Dose Ranging

Start in Q1 2026

Ph3 dose

Ph3 Asthma, COPD, CSRwNP

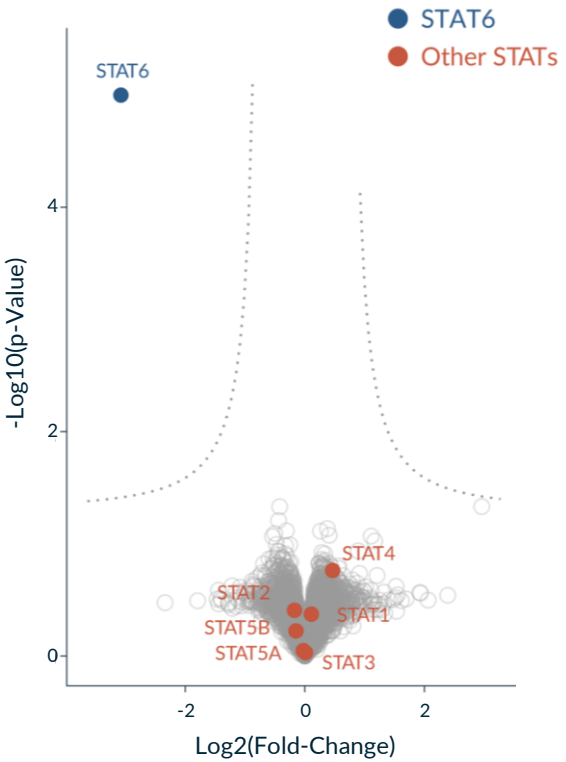
- Efficacy and safety in two initial Th2 diseases
- Ph3 dose selection for franchise

- Ph3 dose selected for AD has potential to be used for other derm/GI indications, such as PN, EOE, BP, and CSU
- Ph3 dose selected for asthma has potential to be used for other respiratory indications, such as COPD and CRSwNP

KT-621: Compelling Preclinical Package

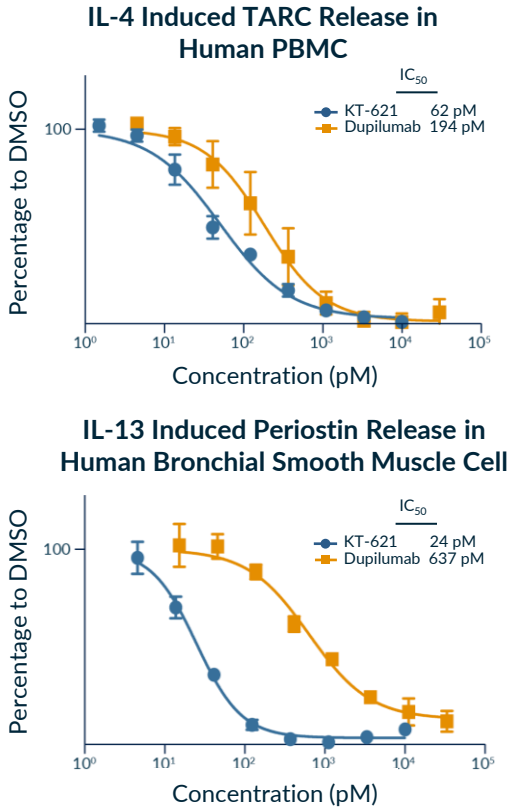
Exquisite Selectivity

Complete STAT6 degradation selectivity in human PBMC at 100xDC$_{90}$



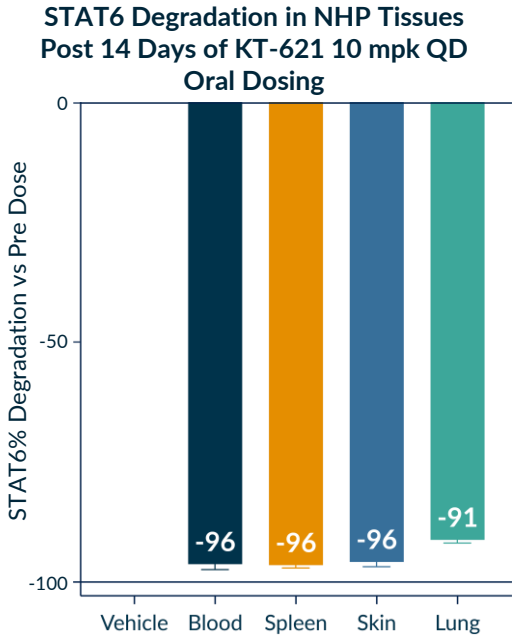
Highly Potent

Full inhibition of IL-4/IL-13 pathways more potent than dupilumab



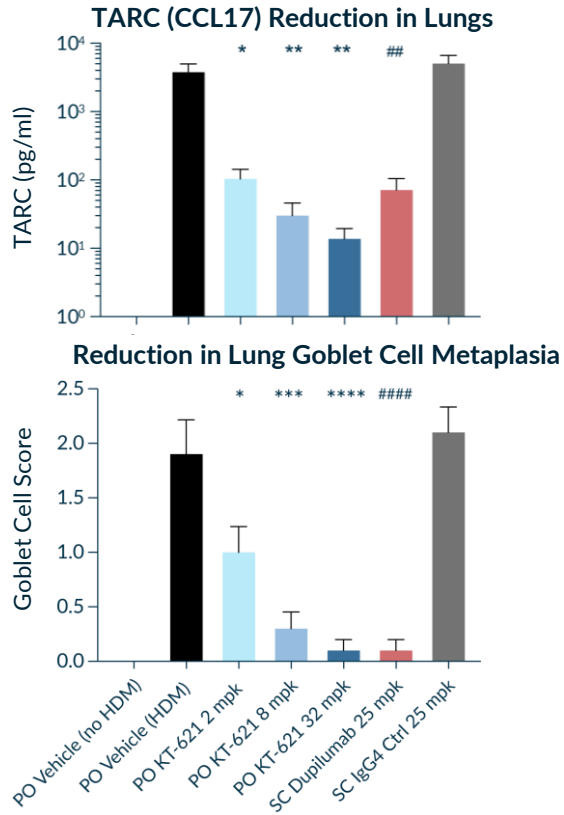
Robust Degradation

KT-621 can degrade STAT6 in all relevant tissues in non-human primates (NHP)



Excellent Preclinical Efficacy

90% STAT6 degradation leads to comparable or superior *in vivo* efficacy to dupilumab in asthma models



Safety: Well tolerated in multiple preclinical species and safety studies at concentrations 40-fold above efficacious dose

*Significance to PO vehicle (HDM); # Significance to SC IgG4 Ctrl 25 mpk.

Phase 1 Healthy Volunteer Exceeded Study Objectives

Clinical Data Continues to Support a Dupilumab-like Profile

Endpoint	KT-621 Healthy Volunteer Objectives	KT-621 Healthy Volunteer Data
STAT6 Degradation in Blood	**90%+**	**Complete**
STAT6 Degradation in Skin	**90%+**	**Complete**
Safety	**Well tolerated at 90%+ degradation**	**Well tolerated 16-fold above the dose with >90% degradation; No SAEs; No TRAEs >1 subject**
Th2 Biomarkers	**Dupi-like profile (non dose-dependent TARC reduction)**	**Robust inhibition of several biomarkers, comparable/superior to published dupilumab data**

Note: Complete degradation within a cohort is defined as either a mean reduction of ≥95% or when most subjects' STAT6 levels are reduced below the LLOQ, or both.
SAE: Serious Adverse Event; TRAE: Treatment Related Adverse Event.

Phase 1 Healthy Volunteer Data Surpass Target Product Profile (TPP) and Underscore Significant Potential of KT-621

Phase 1 HV Goals Exceeded

✓ **Potent Degradation**

- >90% STAT6 degradation in blood in all doses above 1.5 mg
- Complete STAT6 degradation achieved in both blood and skin in all doses ≥50 mg

✓ **Encouraging Th2 Biomarker Impact**

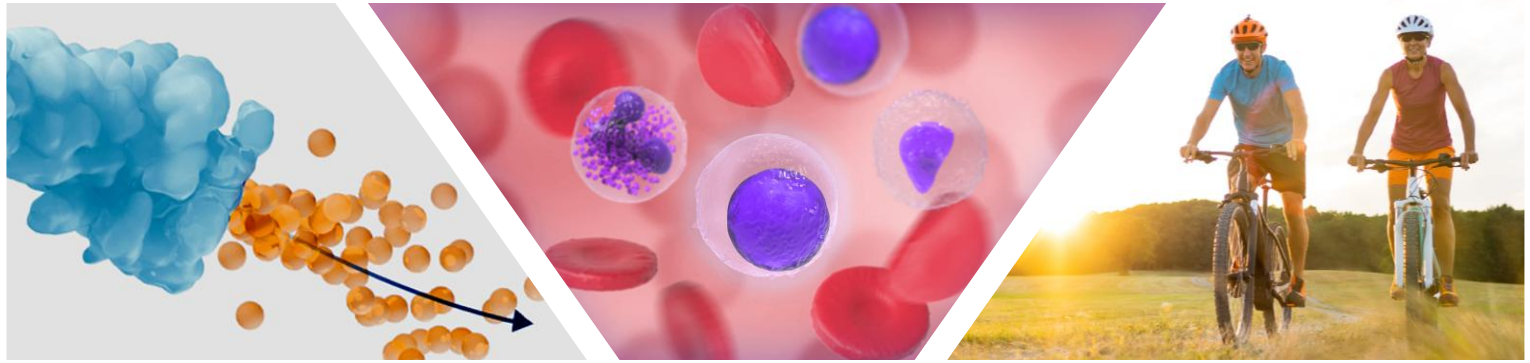
- Results in line or superior to published data for dupilumab, with strong TARC and Eotaxin-3 reductions

✓ **Excellent Tolerability**

- KT-621 was well-tolerated with a safety profile undifferentiated from placebo

Conclusions / Next Steps

- KT-621 profile surpasses Kymera's TPP with compelling preclinical profile human translation above expectations
- We believe the results derisk program and continue to validate KT-621's oral, biologics-like profile
- Proven biology of IL-4/IL-13 pathway, and clearly defined market development path, create a unique and compelling opportunity
- Study informs continued development, including ongoing Phase 1b in AD and upcoming Phase 2b studies in AD and Asthma



KT-621/STAT6 Degrader: Dupilumab-like Activity in a Pill

Phase 1 Healthy Volunteer Data

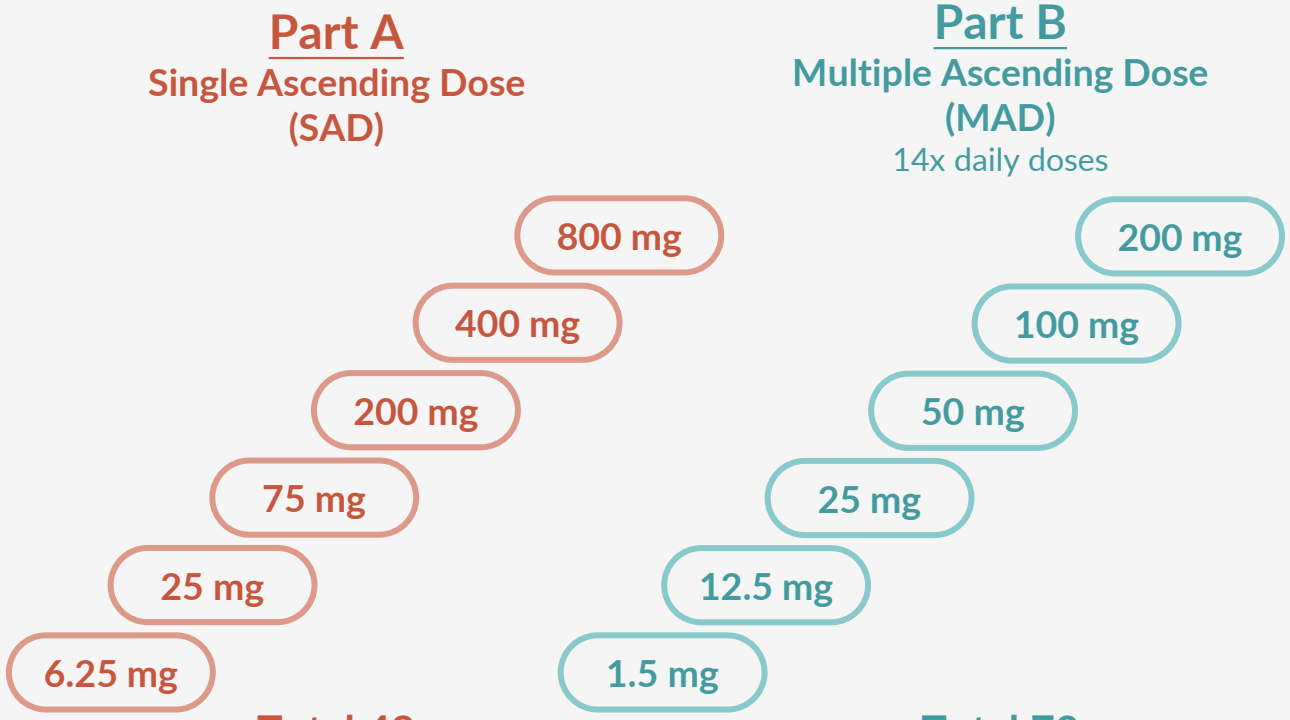
Jared Gollob, MD,
Chief Medical Officer

Agenda

- Trial Description
- Demographics
- Pharmacokinetics (PK)
- SAD Degradation
- MAD Degradation
- Th2 Biomarkers
- Safety

KT-621: Phase 1a SAD and MAD Healthy Volunteer Trial

Randomized, Double-blind, Placebo-controlled SAD and MAD in 118 Healthy Volunteers



Primary

- Safety & tolerability of escalating single and multiple doses of KT-621

Secondary

- Pharmacokinetic measures

Exploratory

- STAT6 protein levels in blood (SAD/MAD) and skin (MAD)
- Th2 biomarkers (MAD)

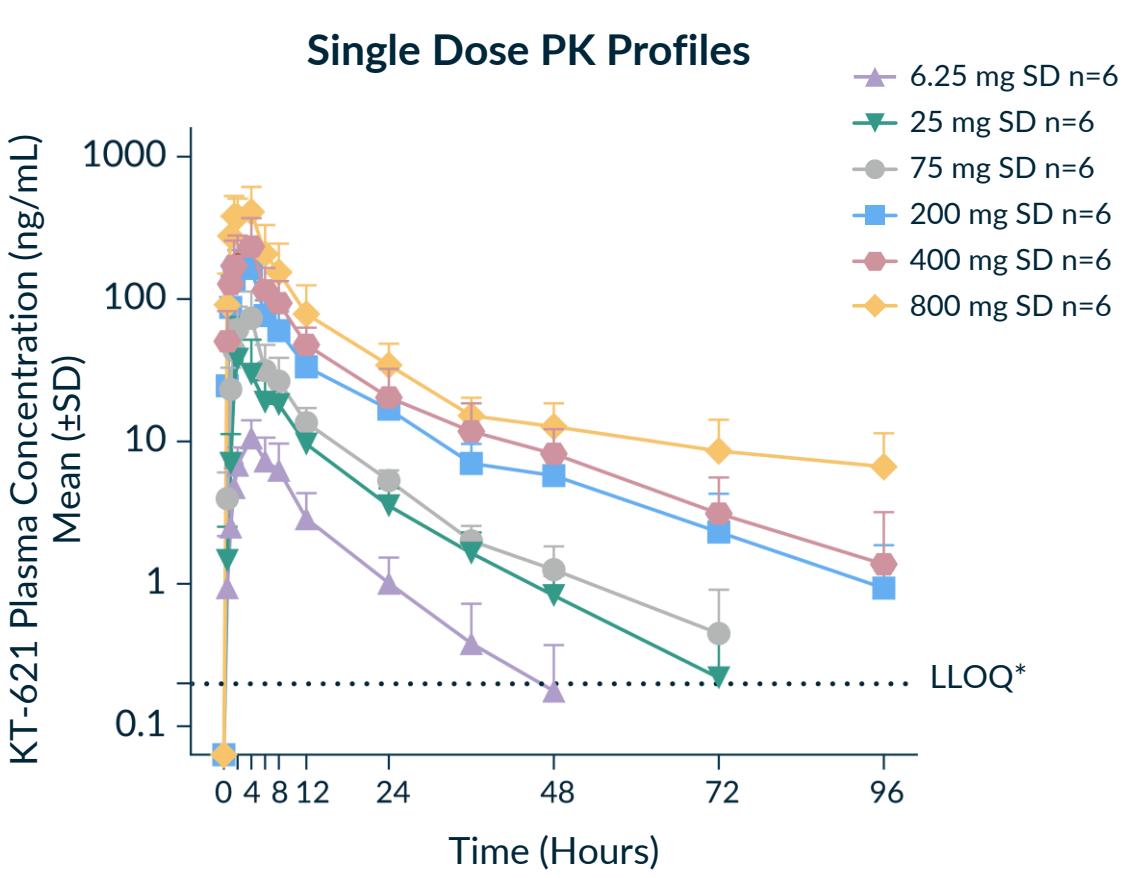
*Part B: 10 subjects were enrolled onto 12.5 mg (7 drug and 3 pbo).

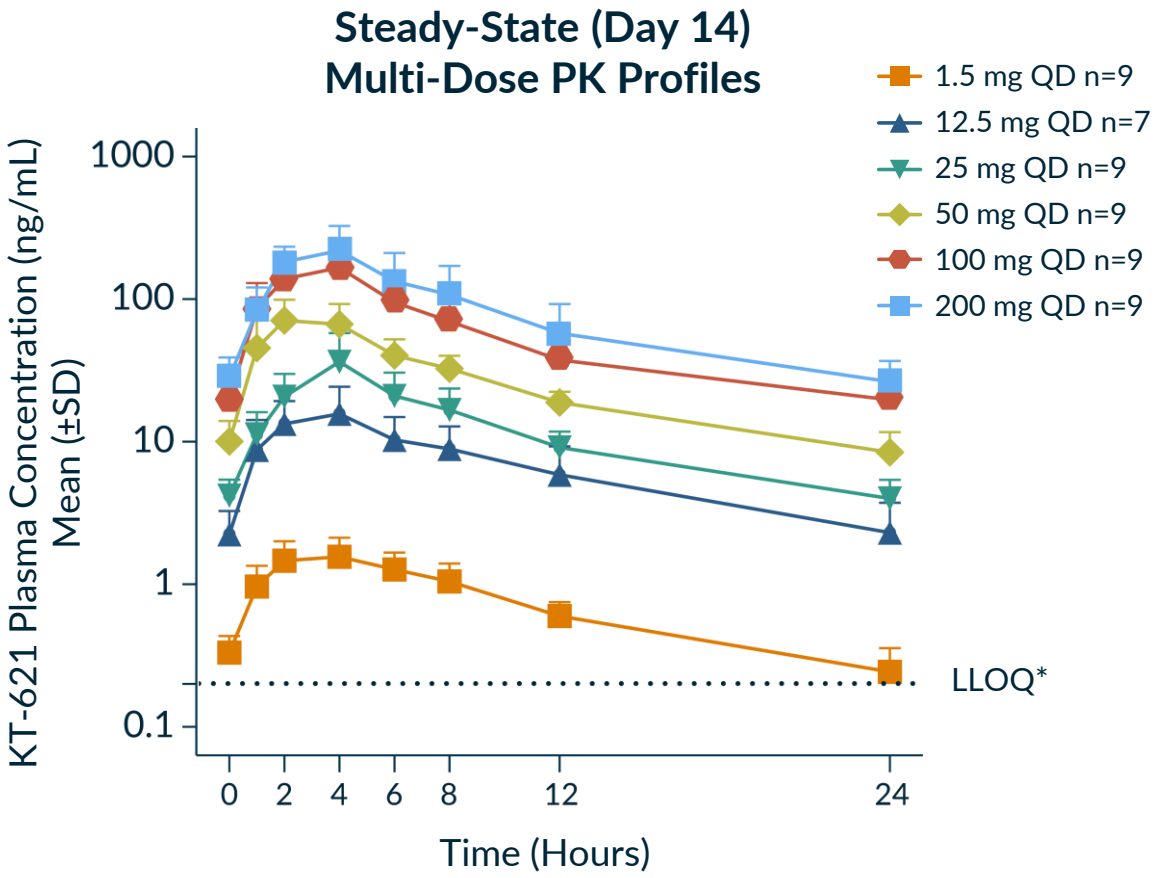
SAD/MAD Demographics

Generally Well-Balanced Across Placebo and Treatment Arms

	SAD 1-6: PBO (n=12)	SAD 1-6: KT-621 (n=36)	MAD 1-6: PBO (n=18)	MAD 1-6: KT-621 (n=52)
Gender, n (%)				
Female	7 (58.3)	11 (30.6)	0	5 (9.6)
Male	5 (41.7)	25 (69.4)	18 (100)	47 (90.4)
Age, years, median (range)	34.5 (20, 50)	32.0 (21, 43)	34.0 (23, 53)	32.5 (19, 52)
Ethnicity, n (%)				
Hispanic or Latino	1 (8.3)	9 (25.0)	4 (22.2)	14 (26.9)
Non-Hispanic or Latino	11 (91.7)	27 (75.0)	14 (77.8)	38 (73.1)
Race, n (%)				
White	9 (75.0)	18 (50.0)	10 (55.6)	29 (55.8)
Black or African American	2 (16.7)	12 (33.3)	6 (33.3)	17 (32.7)
Asian	0	0	2 (11.1)	3 (5.8)
Native Hawaiian or Pacific Islander	0	1 (2.8)	0	0
American Indian or Alaska Native	0	0	0	1 (1.9)
Mixed/Other	1 (8.3)	5 (13.9)	0	2 (3.8)

KT-621: Favorable PK Profile After Single and Multiple Dosing



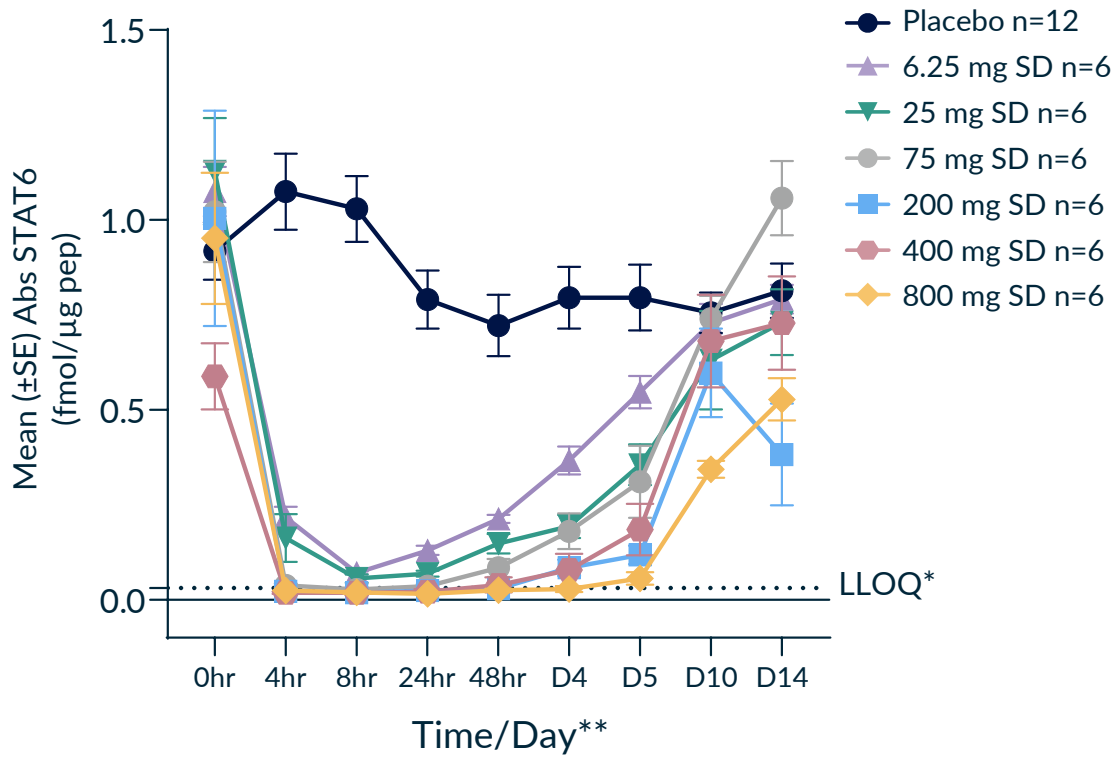


- Rapid absorption with median t_{max} of 2-4 hours and mean half-life of 9-36 hours
- Generally dose-proportional increase in exposure after single and multiple doses with low-moderate variability
- Steady-state achieved by Day 4 of once daily dosing

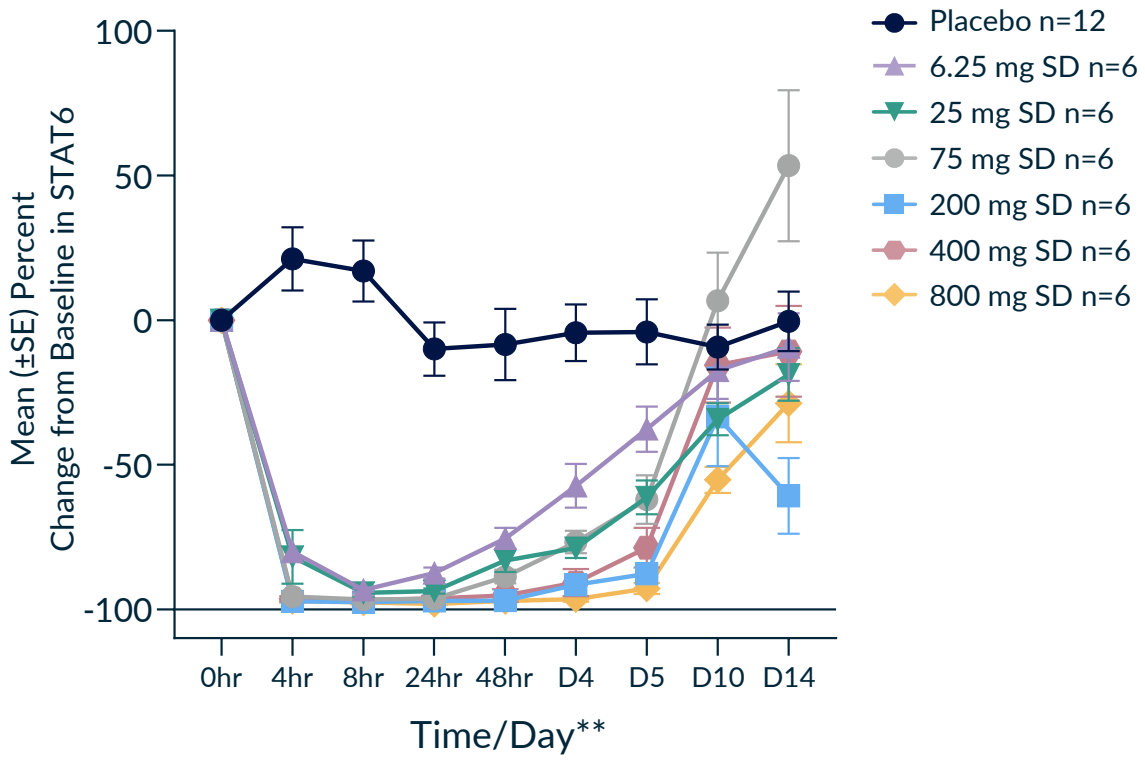
*Lower Limit of Quantification (LLOQ): Below this level, value is assumed to be zero for calculation of summary statistics.

Single Doses of KT-621 Achieved Rapid, Deep and Prolonged STAT6 Degradation in Blood

Absolute STAT6 Levels[1]



Mean Percent Change from Baseline in STAT6

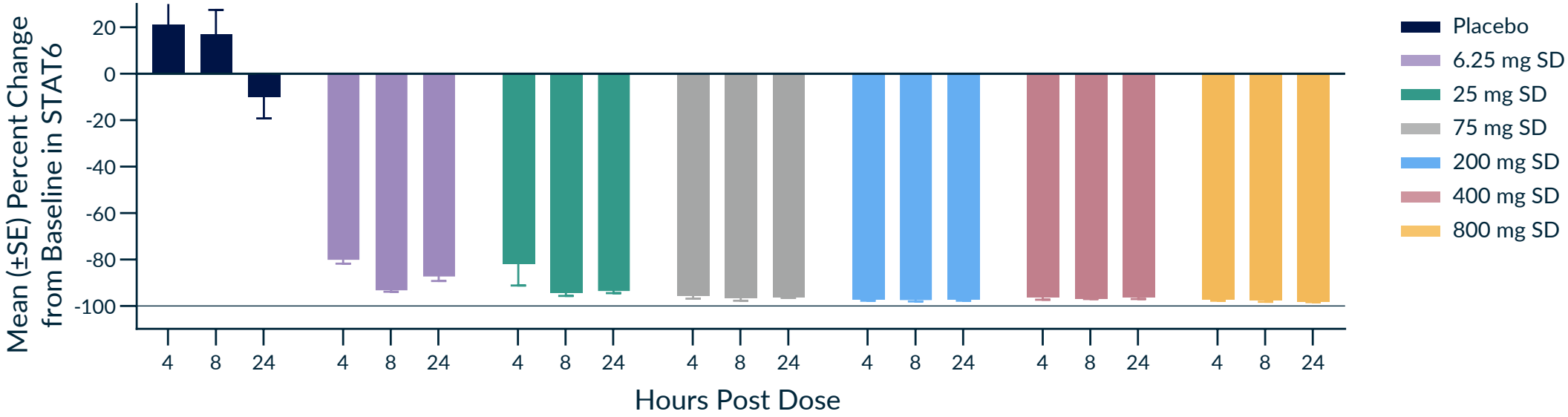


- Maximal degradation seen as quickly as 4 hours after single dose
- Robust degradation maintained as long as 4 days after single dose with recovery by Day 14
- Multiple subjects with STAT6 levels below LLOQ at doses of 75 mg or greater

[1]STAT6 levels measured in isolated PBMC using targeted mass spectrometry;
*Lower Limit of Quantification (LLOQ): Below this level, only estimated value and imputed LLOQ to ½ the value;
**Compound dosed on Day 1.

Single Dose Administration of KT-621 Achieved >90% STAT6 Degradation in Blood Across All Dose Levels

>95% Mean STAT6 Degradation Achieved at Doses of 75 mg or Greater



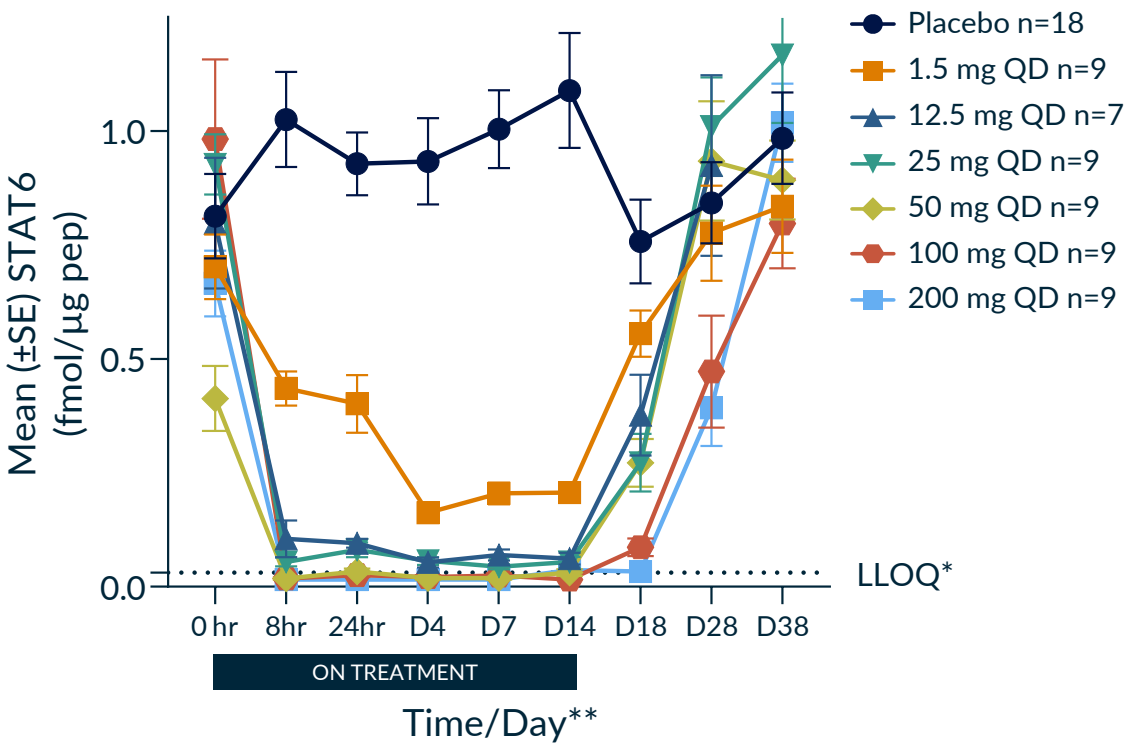
Mean % STAT6 Change	N	4hr	8hr	24hr
Placebo	12	21%	17%	-10%
6.25 mg	6	**-80%**	**-93%**	**-87%**
25 mg	6	**-82%**	**-94%**	**-94%**
75 mg	6	**-96%**	**-97%**	**-96%**
200 mg	6	**-97%**	**-98%**	**-97%**
400 mg	6	**-96%**	**-97%**	**-96%**
800 mg	6	**-97%**	**-98%**	**-98%**

- At doses ≥75 mg, >95% mean STAT6 degradation was achieved with STAT6 levels below LLOQ in multiple subjects
- $p < 0.0001$ for all comparisons (KT-621 vs. Placebo)

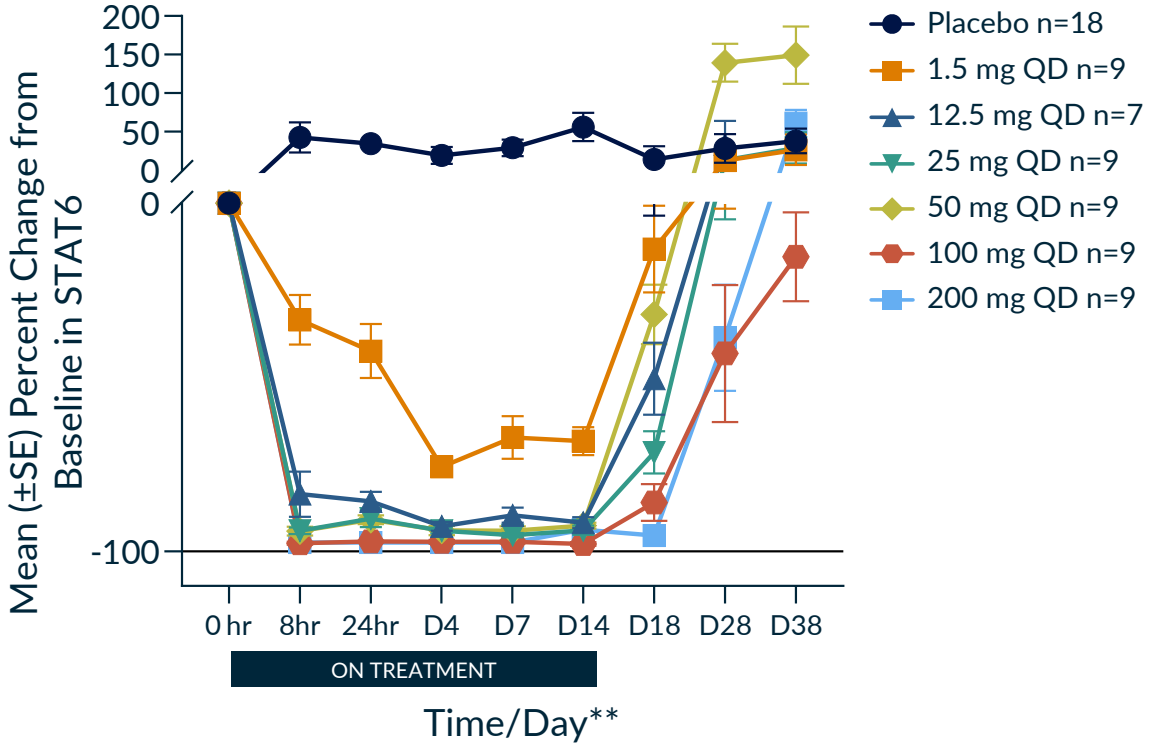
Note: Not all time points have the same N.

Multiple Daily Doses of KT-621 Rapidly (8hr) Achieved Complete STAT6 Degradation in Blood

Mean Absolute STAT6 Levels[1]



Mean Percent Change from Baseline in STAT6

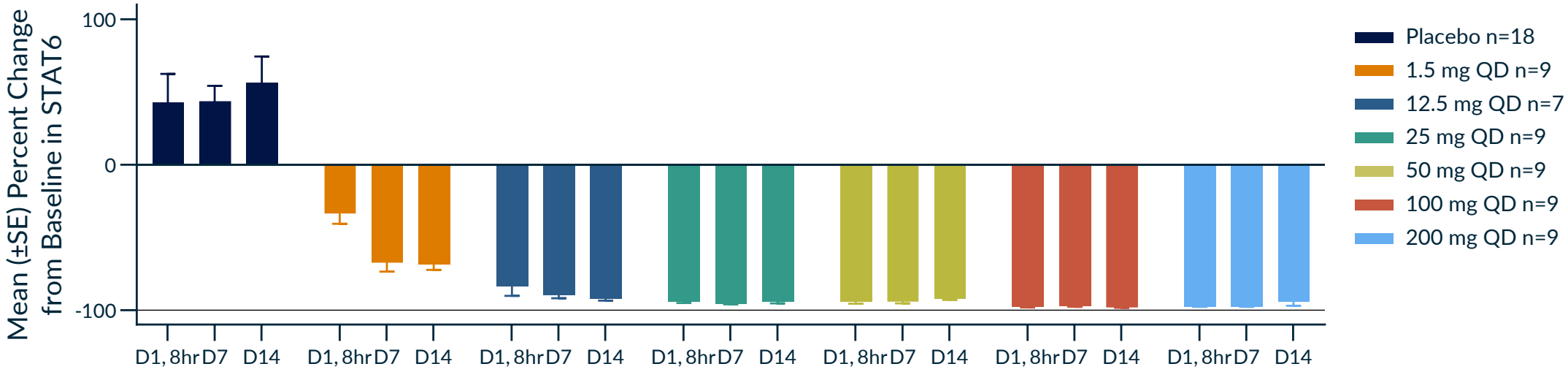


- Robust STAT6 degradation observed at first time point measured (8hr) for all doses above 1.5 mg
- Steady-state maximum degradation achieved as early as 8hr post-first dose with recovery starting at 4 days post-last dose
- Most subjects with STAT6 levels below LLOQ at doses of ≥50 mg

[1]STAT6 levels measured in isolated PBMC using targeted mass spectrometry;
*Lower Limit of Quantification (LLOQ): Below this level, only estimated value and imputed LLOQ to ½ the value;
**Compound dosed on Day 1.

Multiple Daily Doses of KT-621 Achieved >90% STAT6 Degradation in Blood at All Dose Levels Above 1.5 mg

Complete Degradation Achieved at Doses of 50 mg or Greater



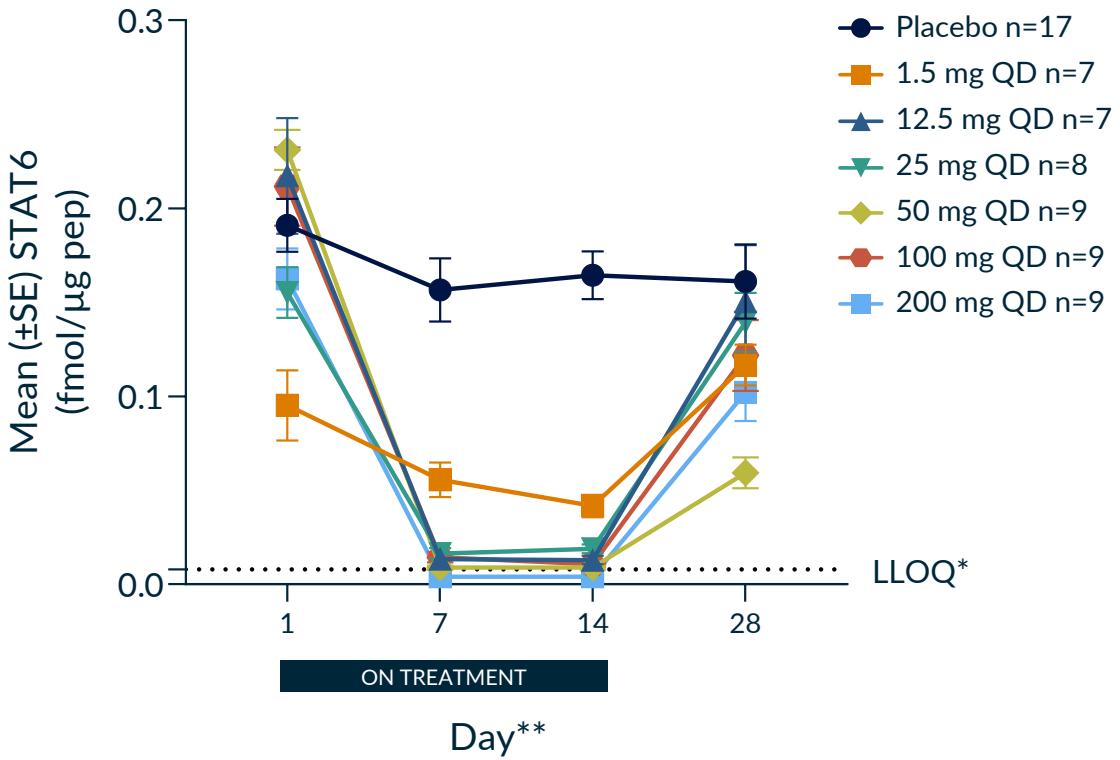
	Mean % STAT6 Change Day 1, 8hr	Mean % STAT6 Change Day 7	Mean % STAT6 Change Day 14
Placebo	43%	44%	56%
1.5 mg	**-34%**	**-67%**	**-68%**
12.5 mg	**-84%**	**-90%**	**-92%**
25 mg	**-94%**	**-95%**	**-94%**
50 mg	**-94%**	**-94%**	**-92%**
100 mg	**-98%**	**-97%**	**-98%**
200 mg	**-97%**	**-97%**	**-94%**

- At doses ≥50 mg STAT6 Levels are below LLOQ for the majority subjects at day 14
- $p < 0.0001$ for all comparisons (KT-621 vs. Placebo)

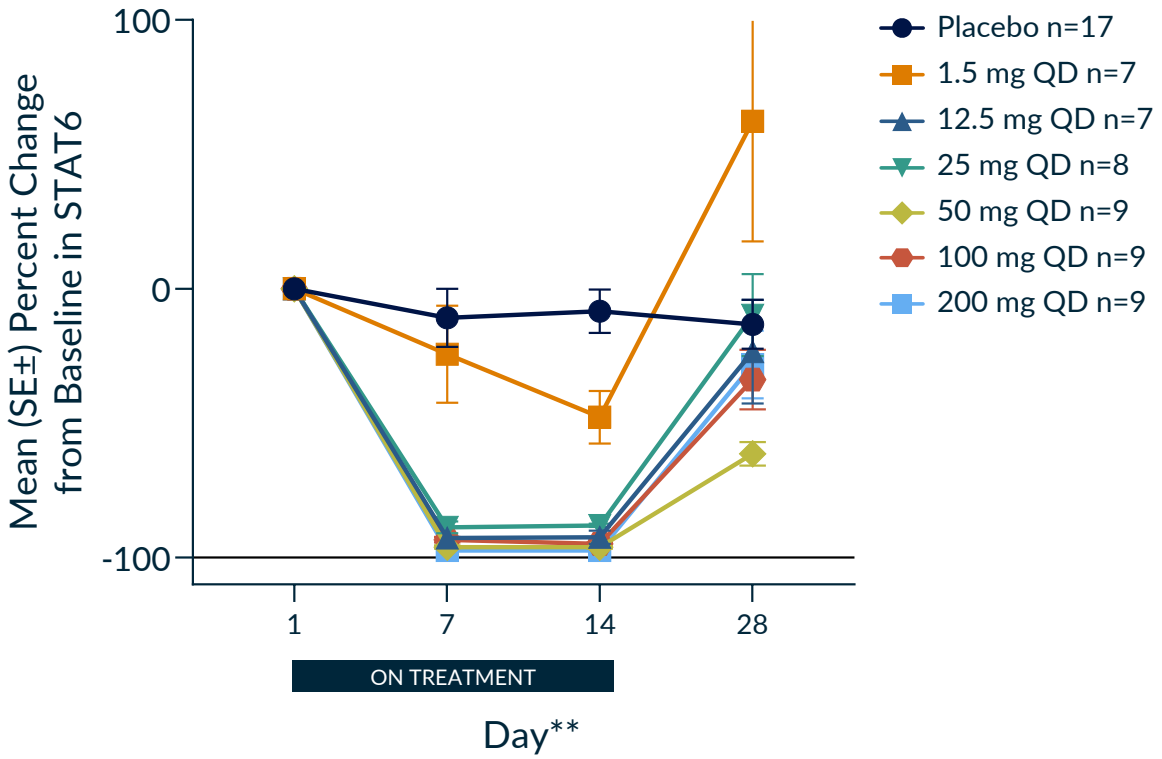
Note: Not all time points have the same N.

Multiple Daily Doses of KT-621 Achieved Complete STAT6 Degradation in Skin

Mean Absolute STAT6 Levels[1]



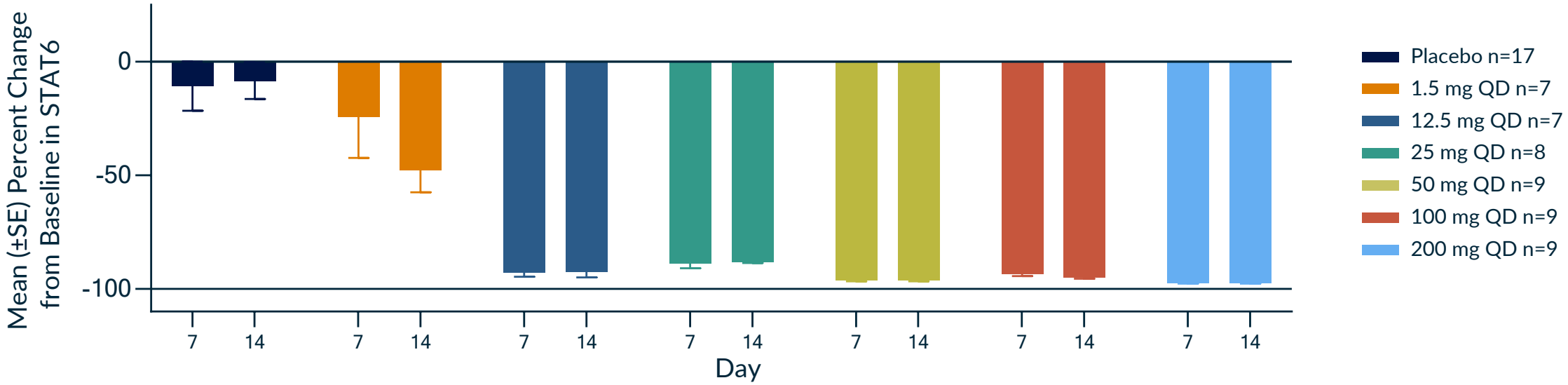
Mean Percent Change from Baseline in STAT6



- Baseline skin STAT6 levels 5-fold lower than peripheral blood mononuclear cells (PBMC)
- Steady-state maximum STAT6 degradation achieved by Day 7 at doses >1.5 mg with recovery observed 14 days post-last dose

[1]STAT6 levels measured in skin biopsies using targeted mass spectrometry;
*Lower Limit of Quantification (LLOQ): Below this level, only estimated value and imputed LLOQ to ½ the value;
**Compound dosed on Day 1.

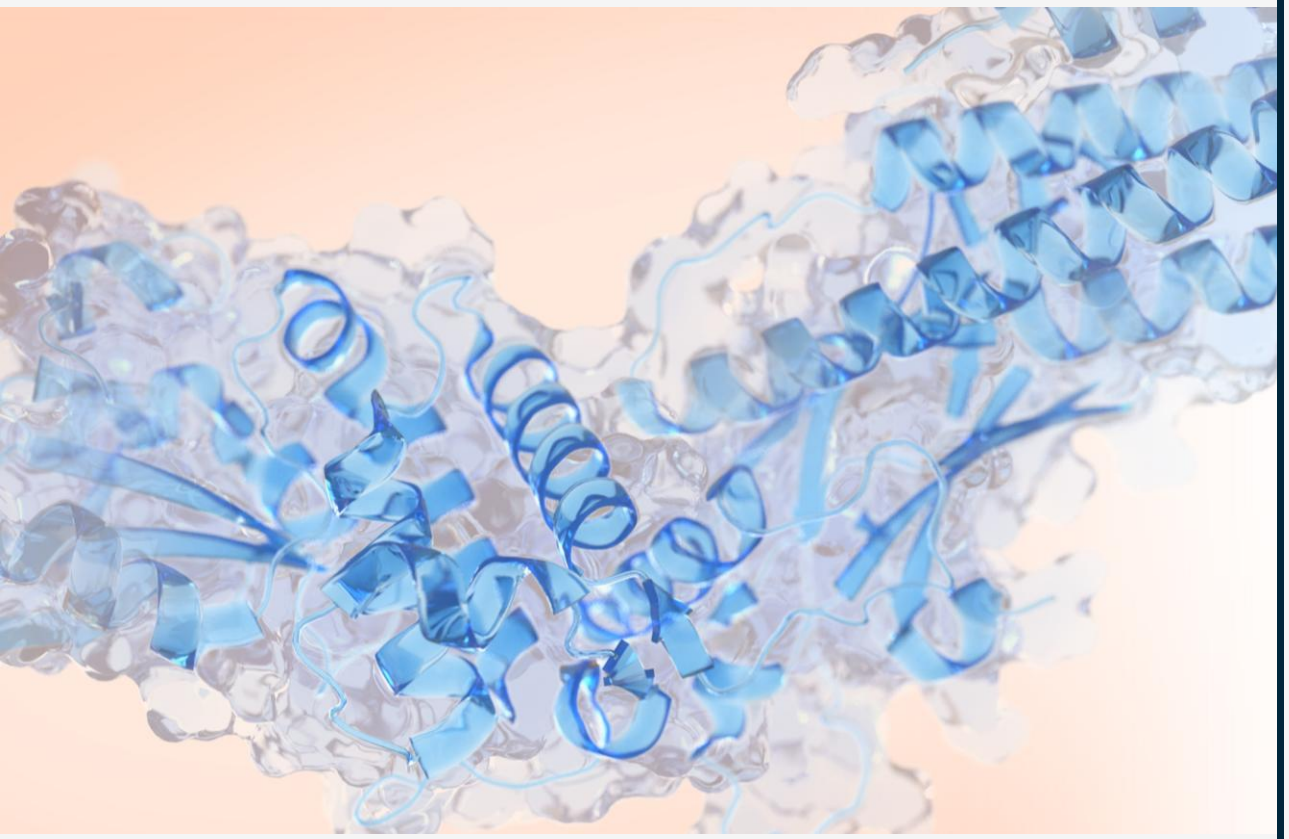
Multiple Daily Doses of KT-621 Achieved Complete STAT6 Degradation in Skin at All Doses ≥50 mg



	Mean% Change in STAT6 Day 7	Mean % Change in STAT6 Day 14
Placebo	-11%	-8%
1.5 mg	**-24%**	**-48%**
12.5 mg	**-93%**	**-92%**
25 mg	**-89%**	**-88%**
50 mg	**-96%**	**-96%**
100 mg	**-93%**	**-95%**
200 mg	**-97%**	**-97%**

- At doses ≥50 mg, ≥95% mean STAT6 degradation was achieved with STAT6 levels below LLOQ in multiple subjects
  - $p < 0.0001$ for all comparisons (KT-621 vs. Placebo)

Note: Not all time points have the same N.

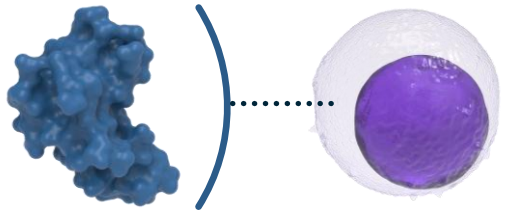


Th2 Biomarkers

TARC, IgE, and Eotaxin-3: Validated Biomarkers of Th2 Biology

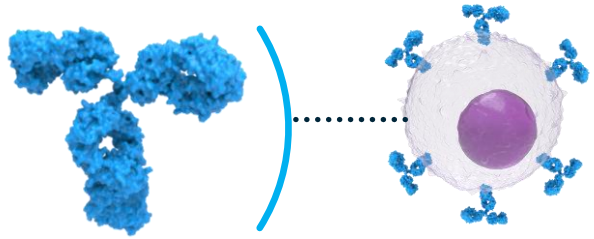
Not Typically Elevated in HV Subjects Without Th2-Driven Disease[1]

TARC (CCL17)



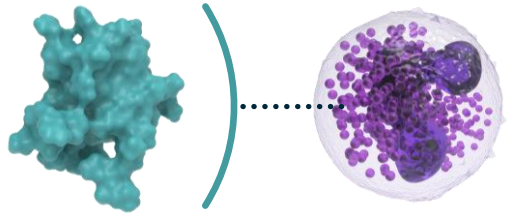
- TARC is the chemokine responsible for chemotaxis of CCR4-expressing T cells (e.g., Th2) to sites of inflammation
- **TARC is a validated biomarker in patients** for suppression of Th2 driven inflammatory responses
- Constitutive **normal levels of TARC** might be partially dependent on IL-4Rα
- Assessed in dupilumab healthy volunteer studies

Immunoglobulin E (IgE)



- IgE activates mast and basophil cells to produce allergic inflammatory mediators (e.g., IL-4 and IL-13)
- **IL-4 promotes B cell class switching which produces more IgE**
- IgE half-life is estimated to be 3 days; **however, turnover of IgE producing B cells and long-lived plasma cells is longer**
- Assessed in dupilumab healthy volunteer studies

Eotaxin-3 (CCL26)

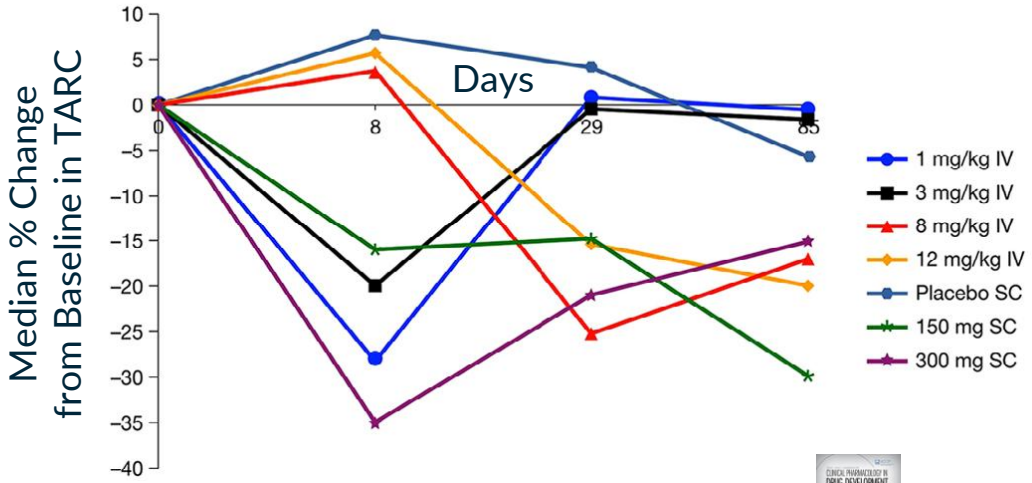


- The chemokine responsible for chemotaxis of CCR3-expressing inflammatory cells (e.g., eosinophils) to sites of inflammation
- **Eotaxin-3 is a highly specific downstream cytokine of IL-4/IL-13 pathway**
- Only assessed in dupilumab patient studies

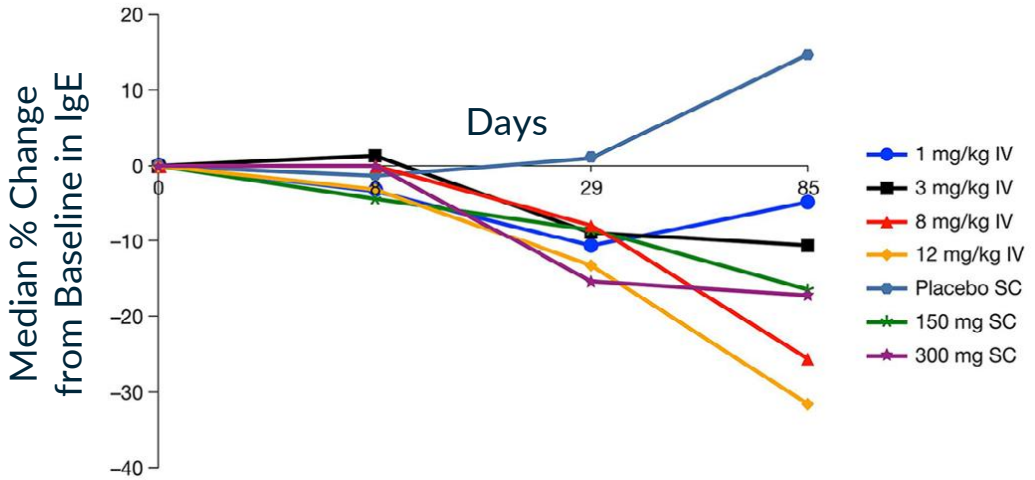
[1]Hamilton et al. Clinical & Experimental Allergy. 2021.

TARC and IgE Assessment in Dupilumab Healthy Volunteer Study

Dupilumab Decreased TARC up to 35% in a Non-Dose Dependent Manner in Healthy Volunteers



High Variability and Minimal Changes in IgE after 1-4 Week



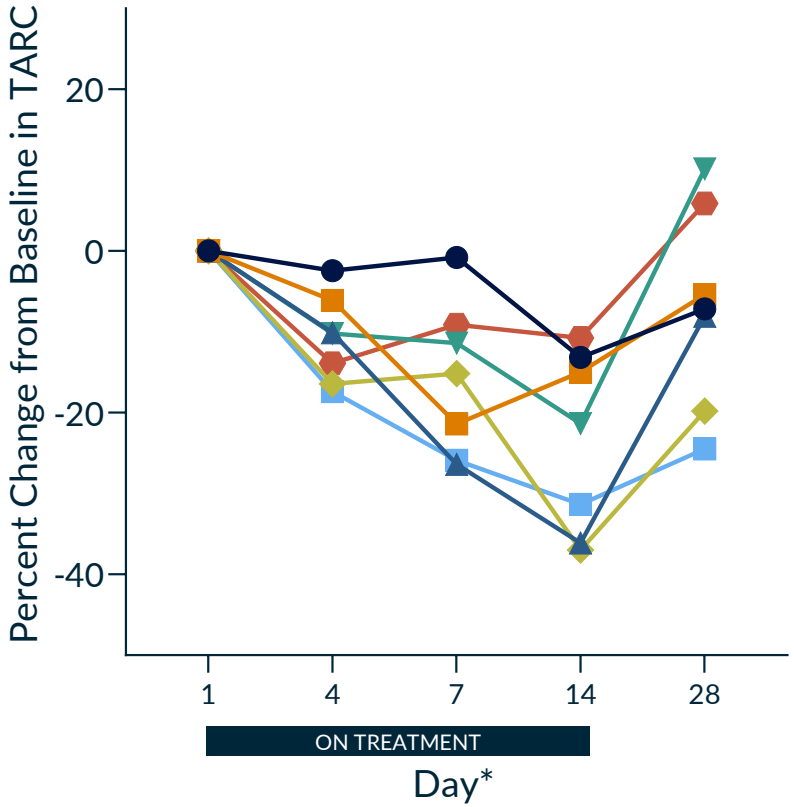
Li et al. *Clinical Pharmacology Drug Development* (2020)

- Non-dose dependent TARC reduction was observed in the study in some but not all doses
- 300 mg SC dose gave max median reduction of ~35%
- Assay window likely very small given that baseline is within normal range

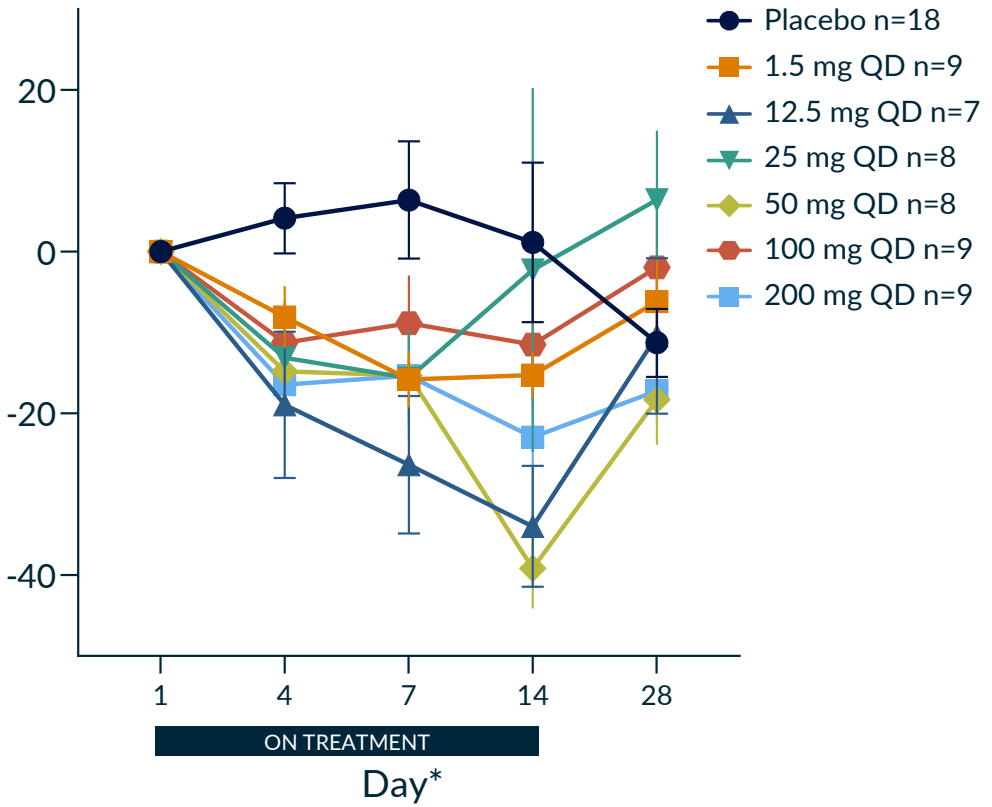
- No meaningful changes of IgE in HV within first week with modest maximal effect of ~15% after 1 month
- In patients where baseline levels of IgE are higher, IgE reduction is minimal within first 2-4 weeks; months are often required to see robust reduction of IgE in patients with Th2 diseases

Multiple Daily Doses of KT-621 Achieved Up to 37% Median TARC Reduction

Median Percent Change from Baseline in Serum TARC[1]



Mean Percent Change from Baseline in Serum TARC[1]

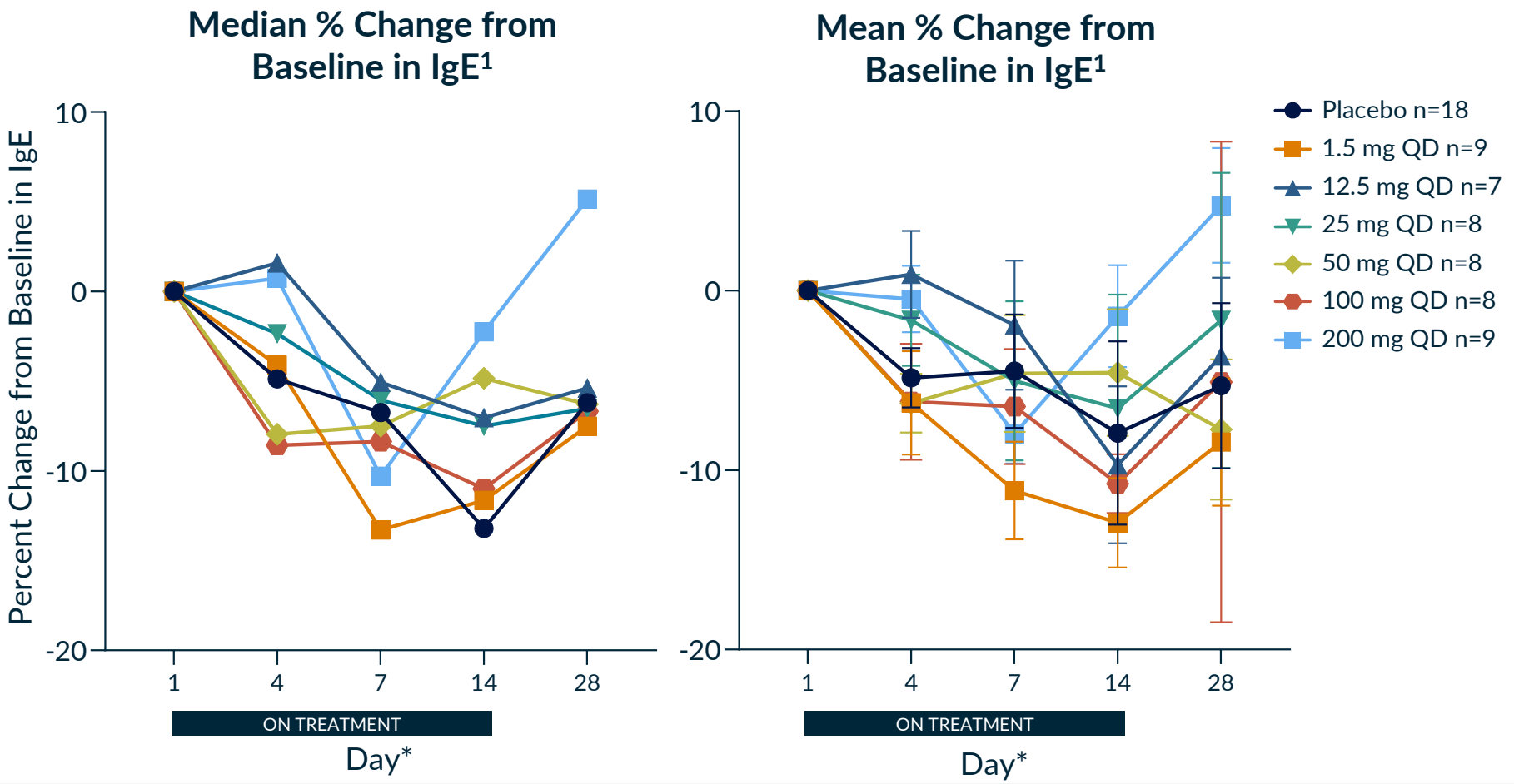


Day 14	Median % Change in TARC	Mean % Change in TARC
Placebo	-13%	1%
1.5 mg	**-15%**	**-15%**
12.5 mg	**-36%**	**-34%**
25 mg	**-21%**	**-2%**
50 mg	**-37%**	**-39%**
100 mg	**-11%**	**-12%**
200 mg	**-31%**	**-23%**

- TARC reduction achieved in all KT-621 doses within the first 14 days
- Baseline TARC levels in line with dupilumab HV study[2]

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

High Variability and Minimal Changes in IgE with Multiple Daily Oral Doses of KT-621

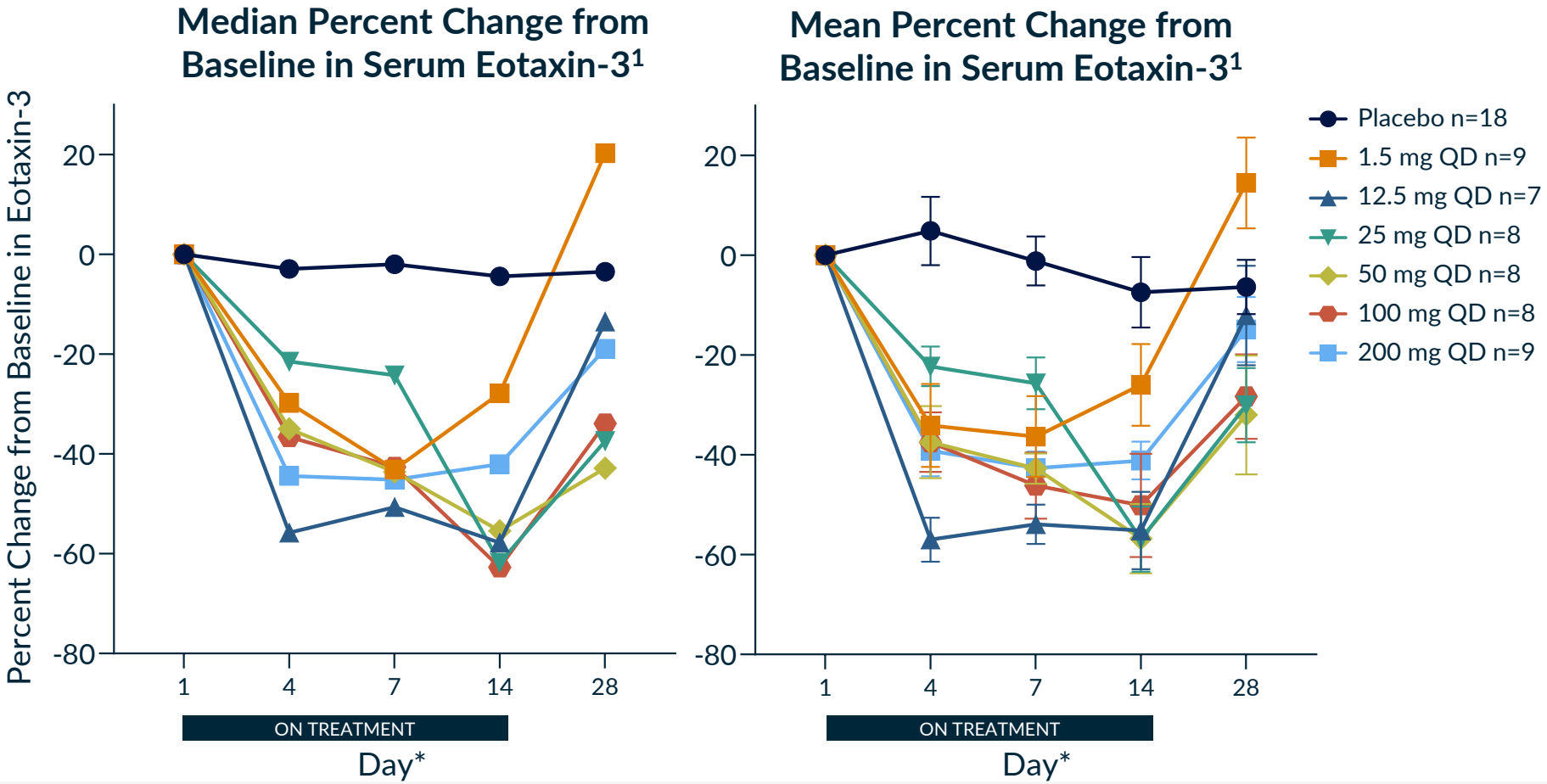


Day 14	Median% Change In IgE	Mean % Change In IgE
Placebo	-13%	-8%
1.5 mg	**-12%**	**-13%**
12.5 mg	**-7%**	**-10%**
25 mg	**-8%**	**-7%**
50 mg	**-5%**	**-5%**
100 mg	**-11%**	**-11%**
200 mg	**-2%**	**-1%**

- Results consistent with dupilumab effect on IgE in healthy volunteers
- Baseline IgE levels in line with dupilumab HV study[2]

[1]IgE levels measured in serum using immunoCAP; [2]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; *Compound dosed on Day 1.

Multiple Daily Doses of KT-621 Achieved Up to 63% Median Eotaxin-3 Reduction



Day 14	Median % Change in Eotaxin-3	Mean % Change in Eotaxin-3
Placebo	-4%	-7%
1.5 mg	**-28%**	**-26%**
12.5 mg	**-58%**	**-55%**
25 mg	**-62%**	**-57%**
50 mg	**-55%**	**-57%**
100 mg	**-63%**	**-50%**
200 mg	**-42%**	**-41%**

- Dupilumab reduced serum Eotaxin-3 by 38% in asthma and 51% in CRSwNP patients at 52 weeks of treatment in Phase 3 studies (no data in healthy volunteers)[2, 3]

[1]Eotaxin-3 levels measured in serum using MSD VPLEX; [2]Hamilton et al. Clinical & Experimental Allergy. 2021; [3]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;
*Compound dosed on Day 1.

Healthy Volunteer SAD/MAD Safety Summary

KT-621 Well Tolerated Across All Doses Evaluated Undifferentiated from Placebo

- No SAEs
- No Severe AEs
- No dose dependent pattern in the TEAEs
- No treatment related AE reported in >1 participant
- No related TEAEs leading to discontinuation
- No clinically relevant changes in vital signs, laboratory tests, and ECGs

TRAEs by Preferred Term: SAD Cohort

AE Term (severity)	SAD 1-6: PBO (n=12)	SAD 1-6: KT-621 (n=36)
Headache (mild)	1 (8.3%)	0

TRAEs by Preferred Term: MAD Cohort

AE Term (severity)	MAD 1-6: PBO (n=18)	MAD 1-6: KT-621 (n=52)
Nausea (mild)	1 (5.6%)	0
Asthenia (mild)	0	1 (1.9%)

AE: Adverse Event; SAE: Serious Adverse Event; TEAE: Treatment Emergent Adverse Event; TRAE: Treatment Related Adverse Event.

Phase 1 Healthy Volunteer Data Surpass Target Product Profile (TPP) and Underscore Significant Potential of KT-621

Phase 1 HV Goals Exceeded

✓ **Potent Degradation**

- >90% STAT6 degradation in blood in all doses above 1.5 mg
- Complete STAT6 degradation achieved in both blood and skin in all doses ≥50 mg

✓ **Encouraging Th2 Biomarker Impact**

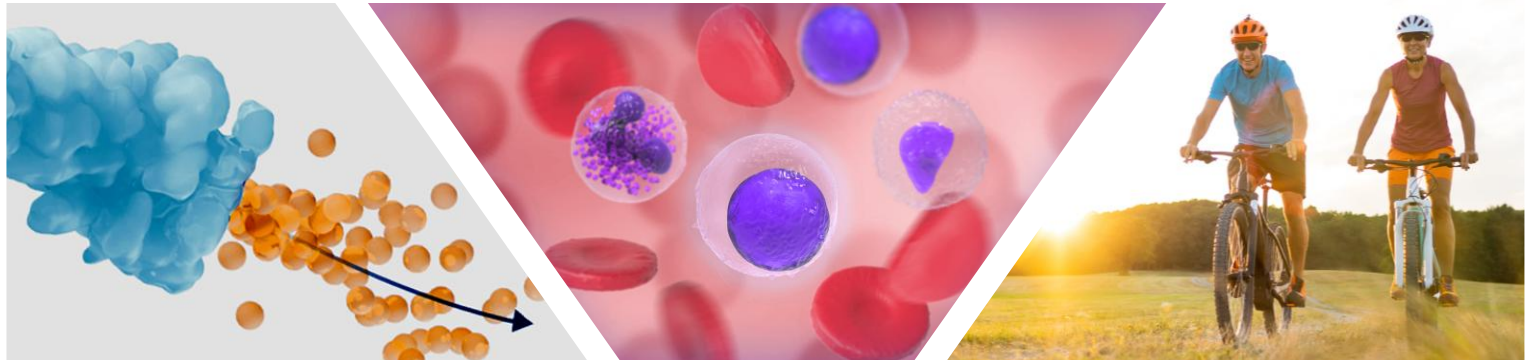
- Results in line or superior to published data for dupilumab, with strong TARC and Eotaxin-3 reductions

✓ **Excellent Tolerability**

- KT-621 was well-tolerated with a safety profile undifferentiated from placebo

Conclusions / Next Steps

- KT-621 profile surpasses Kymera's TPP with compelling preclinical profile human translation above expectations
- We believe the results derisk program and continue to validate KT-621's oral, biologics-like profile
- Proven biology of IL-4/IL-13 pathway, and clearly defined market development path, create a unique and compelling opportunity
- Study informs continued development, including ongoing Phase 1b in AD and upcoming Phase 2b studies in AD and Asthma



Thank You

Q&A

To ask a question, raise your virtual hand

KYMERA