KYMERA 10 EST. 2016



Revolutionizing Immunology with Oral Medicines

November 2026

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Kymera: Promise of Unprecedented Innovation, **Now a Reality**



Founded in 2016 with a mission to discover and develop **medicines that address significant unmet patient needs**

Combining **deep small molecule expertise with a discovery engine** built to address difficult-to-drug proteins

Focused on targets that are **highly validated but are undrugged/inadequately drugged** with disease-agnostic platform capabilities

Advancing four clinical-stage programs positioned to address significant **patient demand for effective and safe oral options**

KT-621 BROADEN2 results provide validation of degrader therapies in immunology, supporting differentiated profile with the potential to overcome trade-offs of existing options

Well-capitalized with $1.6B to advance our pipeline through key development milestones

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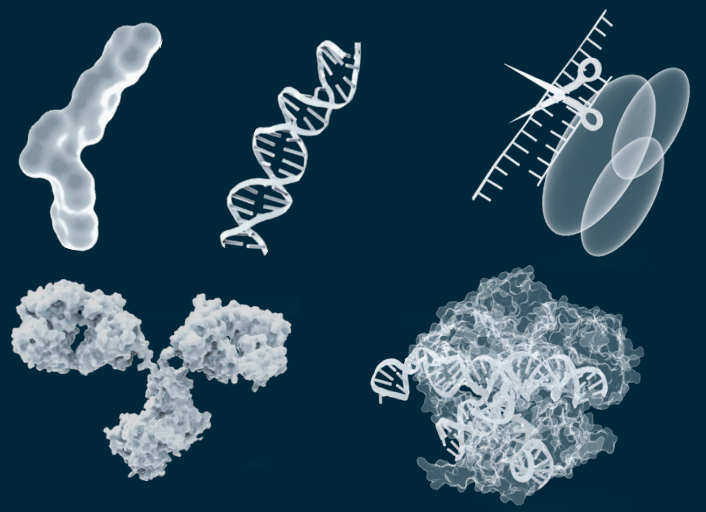
Targeted Protein Degradation: A Bold New Approach

Existing modalities have **functional limitations** (efficacy, safety, convenience) that create gaps in care



This leaves millions of patients **without adequate treatment options**

Targeted protein degradation has the potential to overcome limitations with a unique catalytic mechanism



Small molecule-based modality with gene silencing power

- ✓ High selectivity
- ✓ Picomolar potency
- ✓ Oral administration
- ✓ Disease agnostic

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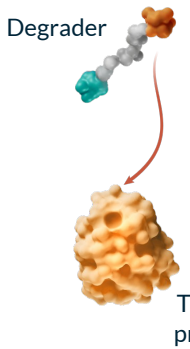
A Catalytic Mechanism That Achieves Full Pathway Suppression with Oral Molecules

Targeted Protein Degradation (TPD) Mechanism of Action

Harnessing the E3 Ubiquitin Proteasome System

1

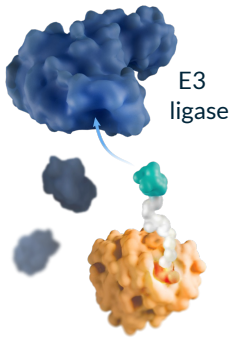
Catching the Target Protein



Degrader molecule engages target protein and E3 ubiquitin ligase to bring them in close proximity

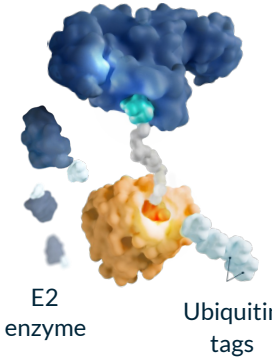
2

Presenting the Target Protein



3

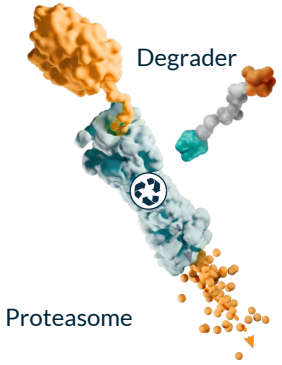
Tagging the Target Protein



E3 ligase places a chain of ubiquitin molecules on the target protein

4

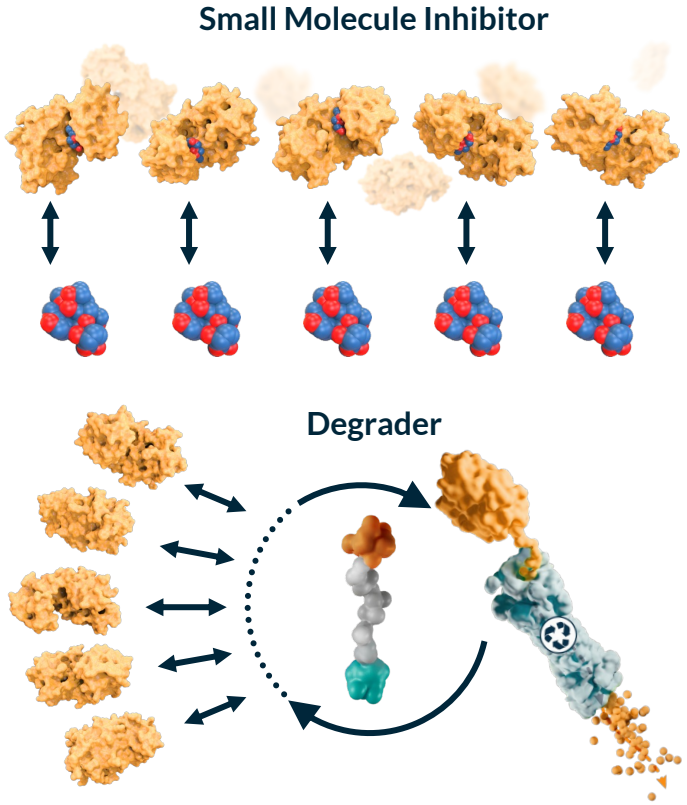
Degrading the Target Protein



Ubiquitin is recognized by the proteasome to selectively degrade the target protein

Degraders Enable Continuous, Complete Pathway Blockade

Superior to Traditional Small Molecule Inhibitors



STOICHIOMETRIC INHIBITION

Pharmacodynamic effect (PD) correlated to drug exposure pharmacokinetics (PK) (stoichiometric) requiring continuous high drug exposures to block protein function[1]

CONTINUOUS DEGRADATION

PD not directly correlated to PK (catalytic), allowing fast, complete protein elimination and continuous pathway blockade with low/short drug exposures

Catalytic activity of degraders enables a single molecule to drive degradation of multiple copies of the target protein, delivering deep and continuous pathway blockade

[1]Mainolfi N, Rasmusson T, *Annual Reports Med Chem*, 2017.

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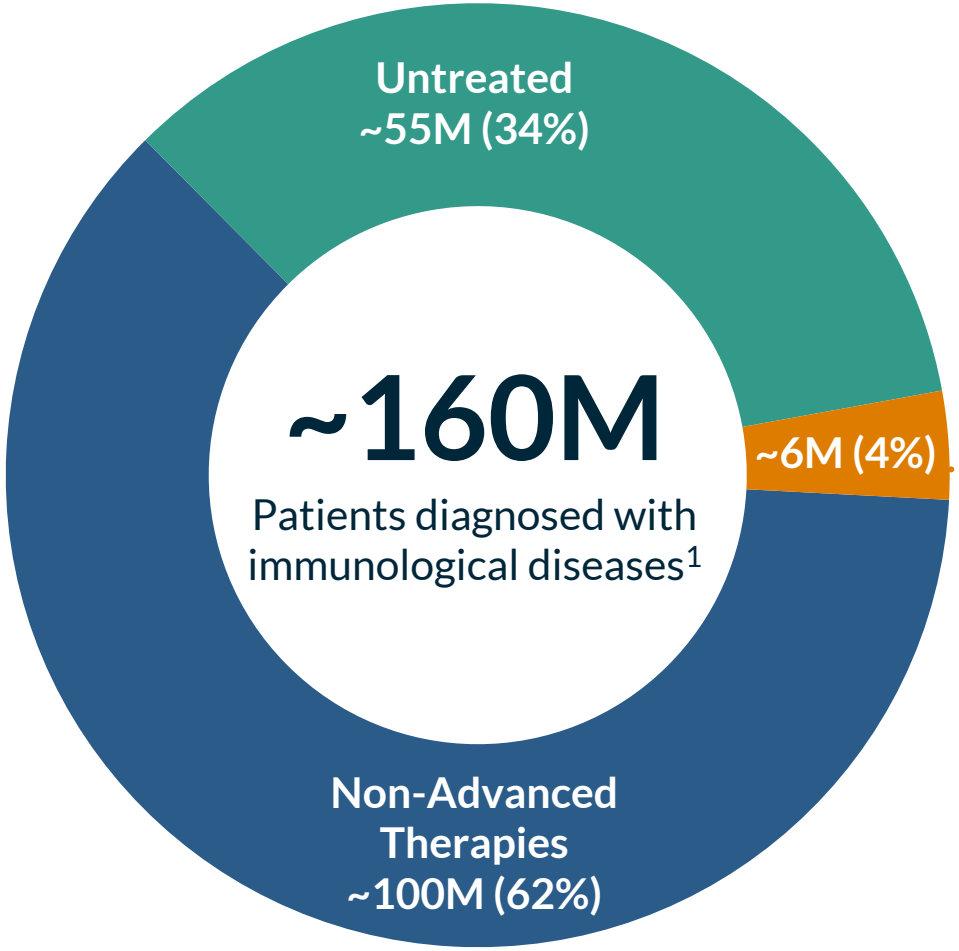
Advanced Therapies Reach a Small Share of Patients Across Major Immunological Diseases



Treated with Advanced Therapies

>$100B

IN ANNUAL SALES FOR ADVANCED THERAPIES[2]



Opportunity to broaden treatment adoption by addressing the trade-offs patients face with existing options

[1]Diagnosed prevalent patient population for US/EU5/JP (GlobalData; 2026); key immunologic diseases includes AD, Asthma, CD, COPD, HS, MS, PsA, PsO, RA, SLE, UC; [2]Market Forecasts for US/EU5/JP (GlobalData; 2026).

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A New Era of Oral Medicines Could Bring More Patients into Treatment

CURRENT TRADE-OFFS	WHAT WE AIM TO DELIVER
Disease control Inadequate disease control & options that don't address root cause	**Meaningful, sustained benefit by targeting disease-driving biology**
Safety and tolerability Concerns about boxed warnings, side effects, monitoring requirements	**Potential for favorable safety and tolerability with long-term use**
Treatment burden Painful injections & complex treatment routines	**Simple oral medicines that fit more easily into patients' lives**



Advancing oral medicines with the potential to be safe, effective and simple to use, expanding treatment possibilities for millions more patients

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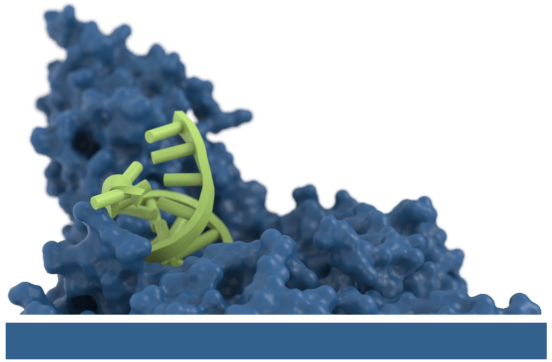
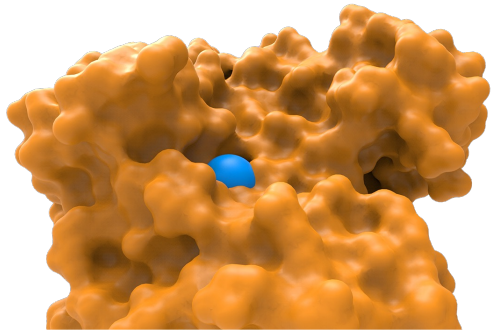
Industry Leader in Developing Oral Small Molecule Medicines

TARGET SELECTION



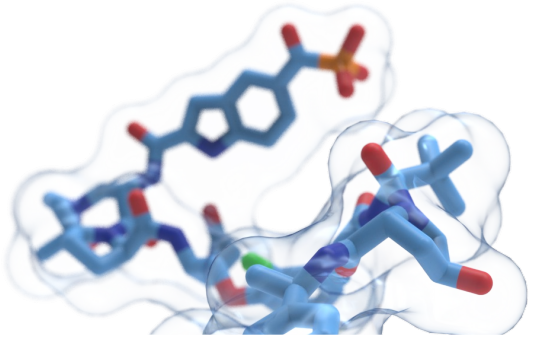
- Historically undrugged/inadequately drugged targets
- Established pathways with genetic/clinical validation
- TPD is the best or only therapeutic solution

HIT FINDING



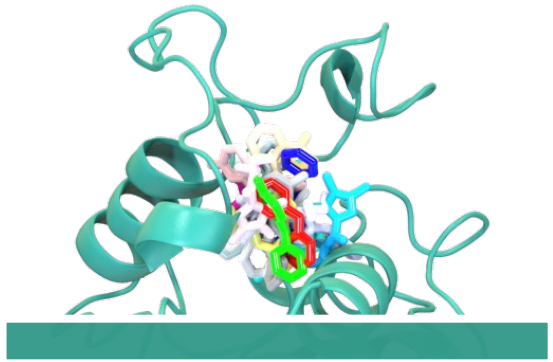
- Multiple orthogonal hit-finding approaches
- Best-in-industry capability to consistently identify ligands for previously undrugged targets

CHEMISTRY



- 10+ years of know-how
- Structurally enabled
- Track record of translating orally bioavailable, potent, selective drugs into the clinic

TECHNOLOGIES



- Expanding the druggable target space through complementary approaches, including heterobifunctional degraders and molecular glues

7 Molecules advanced into clinical studies

ADVANCING A NEW CLASS OF TARGETED ORAL MEDICINES

10+ Discovery programs in areas of unmet need

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Building a Best-in-Industry Oral Immunology Pipeline

Proven Engine for Oral Small Molecule Innovation

	Potential Indications	Patient Opp.[1]	IND-Enabling	Phase 1	Phase 2	Phase 3	Upcoming Milestones
Immunology - Wholly-Owned Oral Small Molecule Degraders							
STAT6	AD, Asthma, COPD, EoE, CRSwNP, CSU, PN, BP, others	>140M	KT-621 – AD				**Ph3 AD Start:** By mid-2027 **Ph2b Asthma Data:** Late 2027
			KT-621 – Asthma				
IRF5	Lupus, Sjögren's, RA, IBD, SSc, DM, others	>10M	KT-579				**Ph1b Lupus Start:** By early 2027
Partnered Programs							
IRAK4[2]	HS, AD, RA, Asthma, IBD, others[3]	>140M	KT-485				sanofi
CDK2[4]	Breast cancer and other solid tumors	>500K	KT-200				**IND:** 2027 GILEAD

[1]GlobalData (2023 diagnosed prevalent patient population in US/EU5/JP; STAT6 estimates include only AD, Asthma, COPD; IRF5 estimates include only SLE, RA, UC, CD; IRAK4 estimates include all noted indications; CDK2 estimates include all HR+/HER2- BC settings); [2]KT-485 (SAR447971) 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; [3]Diseases where IL-1R/TLR pathway has been implicated in pathogenesis; [4]Partnered with Gilead, exclusive option and license agreement to accelerate the development and commercialization of a novel molecular glue degrader program.

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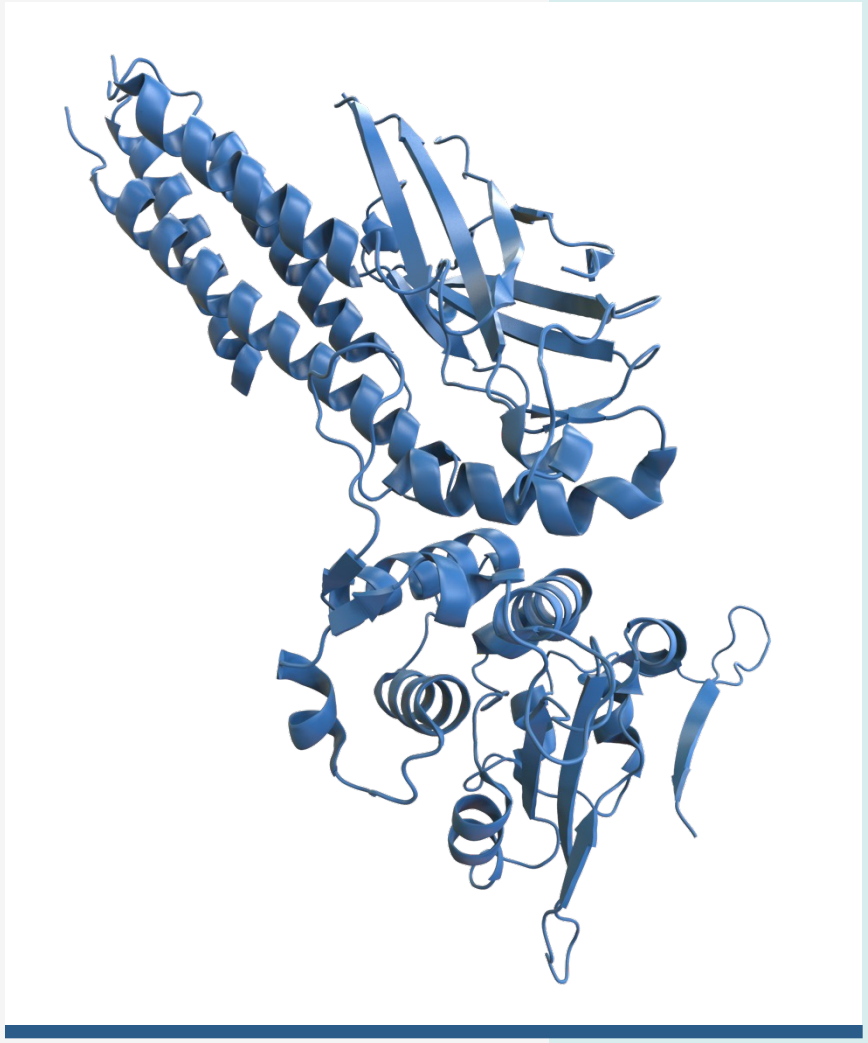

First-in-Class Oral STAT6 Degrader Program

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KT-621: Potentially First Safe, Efficacious, Oral Therapy for Millions of Patients with Type 2 Inflammatory Diseases



VALIDATED BIOLOGY

- Specific transcription factor in the IL-4/IL-13 pathway
- Clinical validated pathway across multiple Type 2 diseases: AD, Asthma, COPD, EoE, CRSwNP, CSU, PN, BP, others

UNIQUE PROFILE

- Compelling preclinical *in vitro/in vivo* potency, efficacy and safety data
- Early Phase 1 data in HVs and AD patients showed deep target/pathway engagement, promising efficacy and favorable safety profile
- Positive Phase 2b data in AD support KT-621's potential to combine strong efficacy and favorable safety in a once-a-day oral therapy

STATUS & MILESTONES

- Phase 3 trials in AD planned to initiate by mid-2027
- BREADTH Phase 2b asthma trial ongoing, with data in late 2027

KT-621 has the potential to expand into an underpenetrated but highly valuable market (>$30B today at ~5% market penetration) **positioning KT-621 as a potential once-in-a-generation drug**

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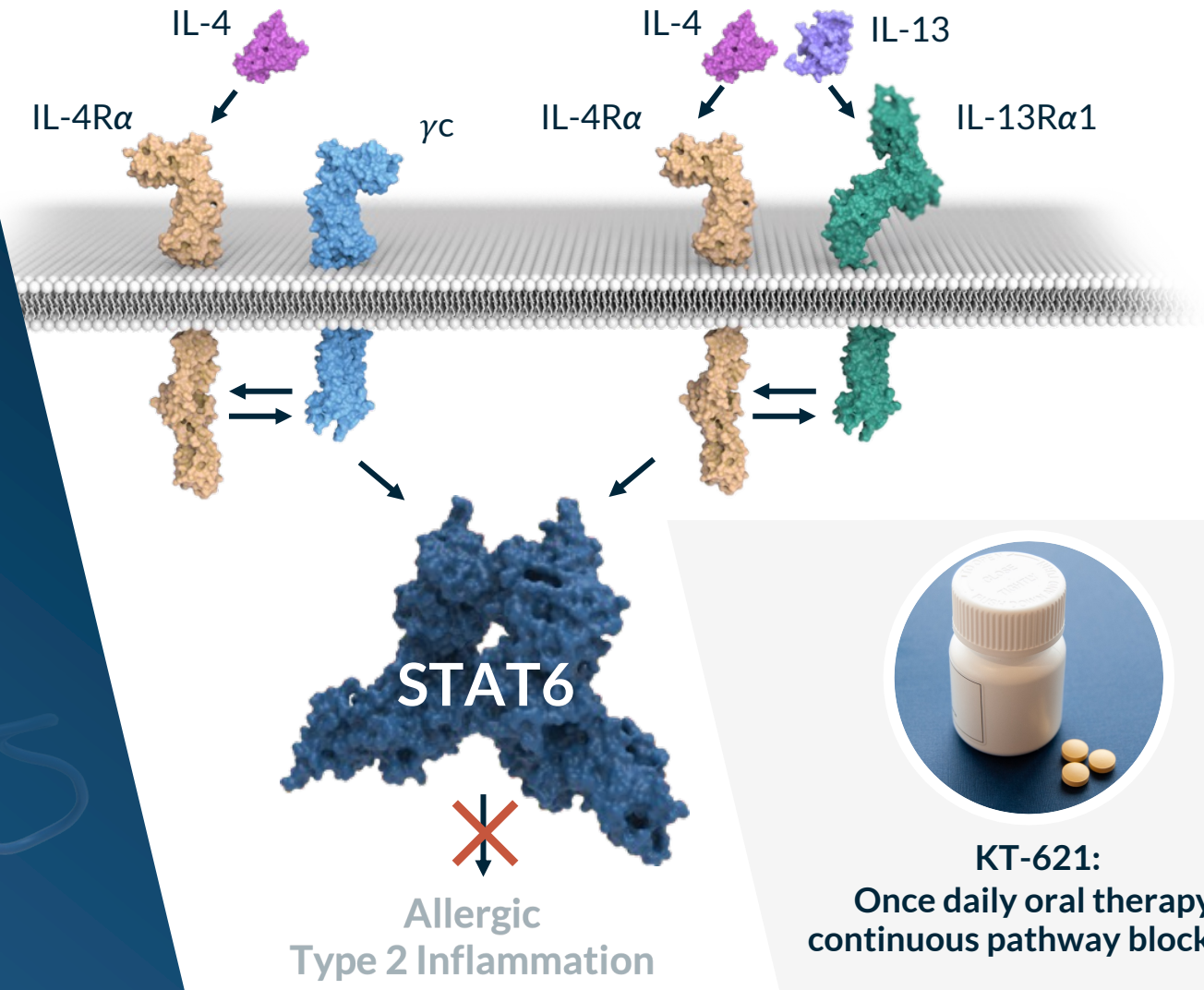
A New Era of Oral Therapy for Type 2 Inflammation

STAT6

FIRST-IN-CLASS
TARGETED MECHANISM

- STAT6 is central to IL-4/IL-13 signaling in Type 2 inflammation
- Dupilumab, an injectable biologic blocking IL-4/IL-13, is the only approved Type 2-targeting drug across 9 indications
- KT-621, potential first-in-industry STAT6 degrader, blocks Type 2 inflammation with a targeted oral small molecule



KT-621:
Once daily oral therapy, continuous pathway blockade

Sharma et al, 2023; Takeda et al, 1996; Kaplan et al, 1996; Junttila, 2018; Kolkhir et al, 2023.

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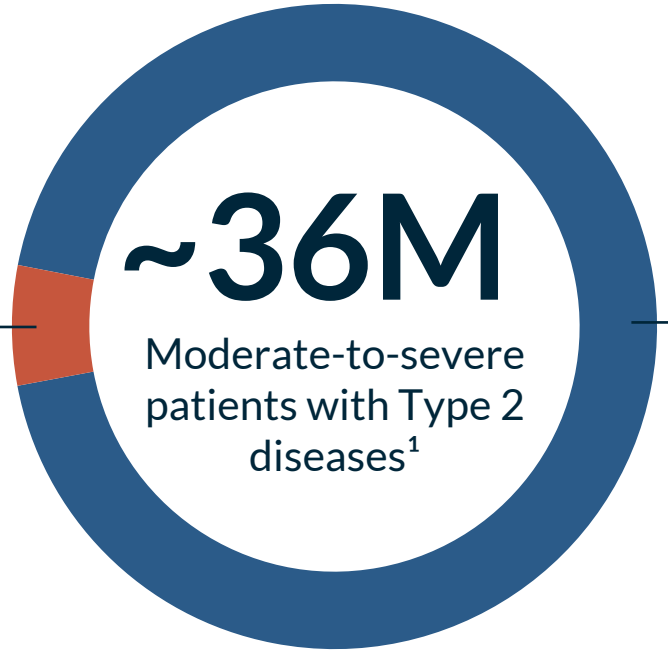
KT-621: Potential to be First Oral Therapy for Millions of Undertreated Patients Across Type 2 Diseases

Limited penetration:

~5% (2M)

on advanced systemic therapies

>$30B market[1]



Opportunity to expand:

~95%

of patients are underserved

DERMATOLOGY				GI	RESPIRATORY		
AD	CSU	PN	BP	EoE	Asthma	CRSwNP	COPD

Substantial market opportunity in immunology

Opportunity to reach millions of patients with a safe, effective, oral option



[1]GlobalData, 2026; AD: Atopic Dermatitis; BP: Bullous Pemphigoid; COPD: Chronic Obstructive Pulmonary Disease; CRSwNP: Chronic Rhinosinusitis with Nasal Polyps; CSU: Chronic Spontaneous Urticaria; EoE: Eosinophilic Esophagitis; PN: Prurigo Nodularis.

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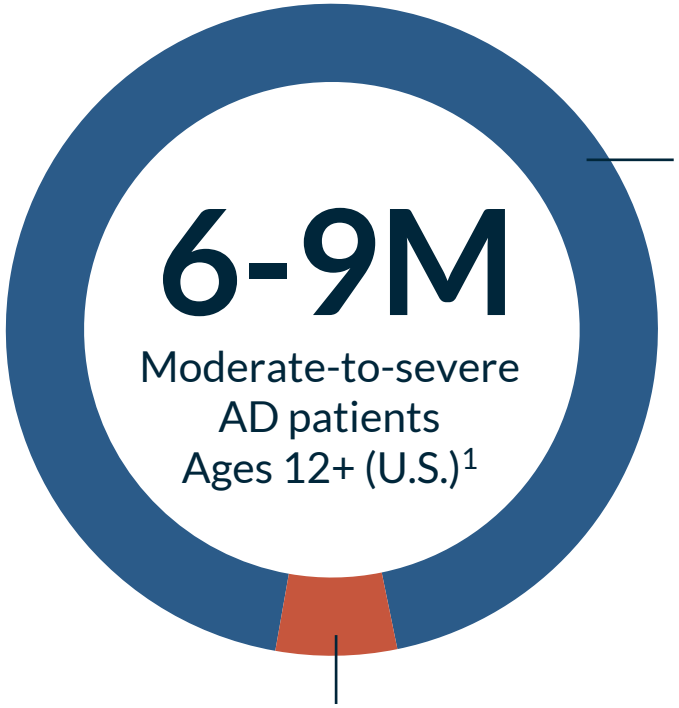
KT-621: Atopic Dermatitis Opportunity



Limited penetration:

~5% (400k)

on advanced systemic therapies

~$11B market[2]

~95% of patients are **underserved**

Many feel their disease state **does not justify the trade-offs** of current advanced therapies[3]

Injection hesitancy: reluctance to initiate injectable biologics and in many cases preference to avoid needles

Safety concerns: boxed warnings and side effects of existing orals



KT-621 has the potential to **expand advanced therapy use**, reaching **millions of AD patients** who are untreated or inadequately controlled

[1]Company estimates based on industry and peer-reviewed research; [2]GlobalData, 2026; [3]Quantitative survey and market research by Trinity, 2026: n = 166 patients and caregivers.

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KT-621 Broad Late-Stage Development in Type 2 Diseases

	DERMATOLOGY/GI	RESPIRATORY
Phase 2	BROADEN2 TRIAL Positive Phase 2b data inform Phase 3 dose selection for dermatology/GI indications	BREADTH TRIAL Late 2027 Phase 2b data will inform/confirm Phase 3 dose selection for respiratory indications
Phase 3	• **Atopic Dermatitis (expected to initiate by mid-2027)**[1] • Eosinophilic Esophagitis • Chronic Spontaneous Urticaria • Prurigo Nodularis • Bullous Pemphigoid	• Asthma • Chronic Rhinosinusitis with Nasal Polyps • Chronic Obstructive Pulmonary Disease

Phase 2b trials in AD and asthma have the potential to support Phase 3 trials across multiple Type 2 indications, where millions of patients remained underserved and a safe, effective, oral option could transform care

[1]Subject to alignment with regulators; GI: Gastroenterology.

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KT-621 Clinical Trial Data

- Phase 1 Healthy Volunteers
- Phase 1b Atopic Dermatitis
- Phase 2b Atopic Dermatitis



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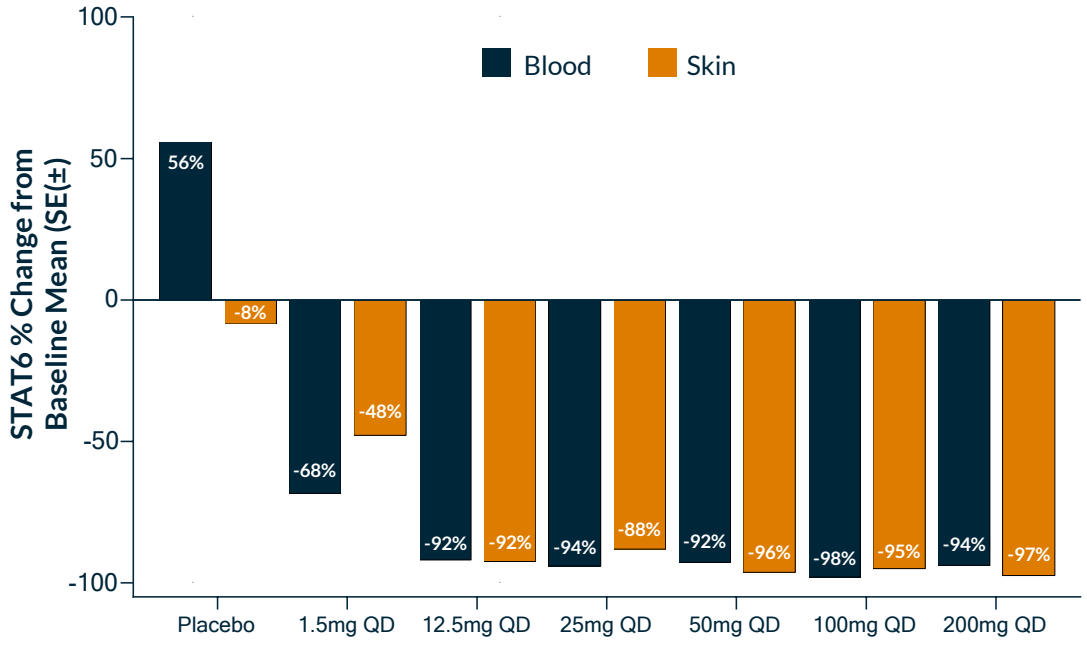
KT-621 Phase 1 Healthy Volunteer Trial

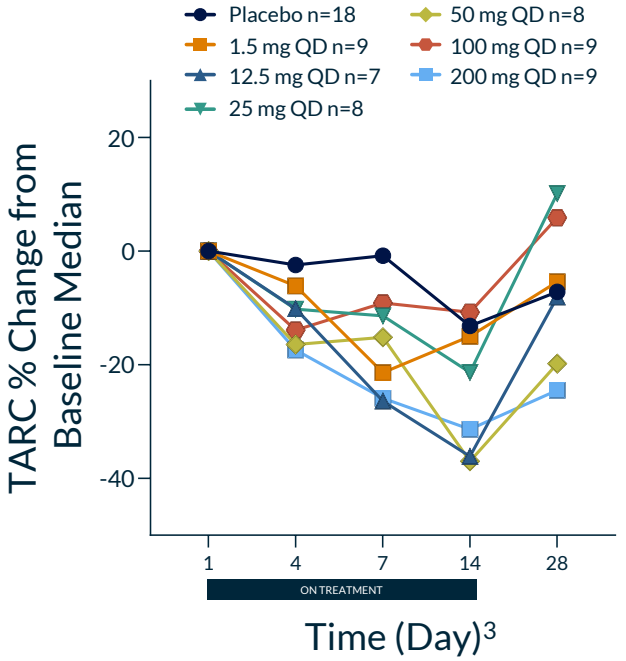
Demonstrated Compelling Clinical Profile

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

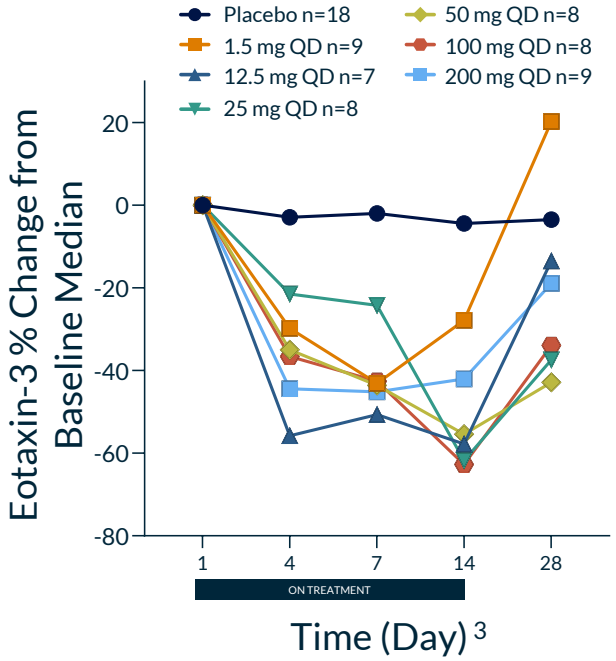


Reductions in Multiple Disease-Relevant Type 2 Biomarkers

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; For more information on KT-621's Phase 1 healthy volunteer clinical trial, visit Kymera's website.

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KT-621 BroADen Phase 1b AD Study Surpassed Objectives

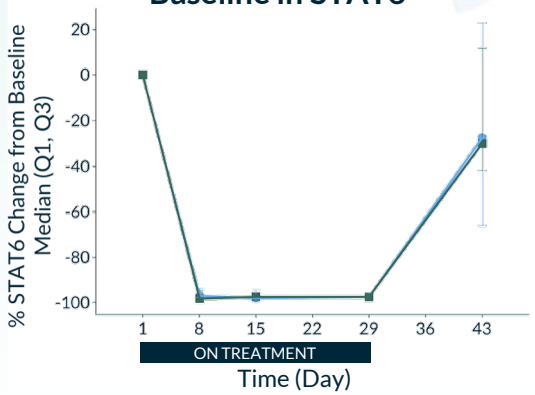
Clinical Data Support the Potential to Deliver an Effective and Well-tolerated Oral Solution to Patients

STAT6 DEGRADATION

Strong fidelity of translation from Phase 1 HV study

- Median 98% STAT6 degradation in blood
- Deep skin degradation of 94%
- STAT6 in multiple pts. below lower limit of quant. (LLOQ)

Median % Change from Baseline in STAT6

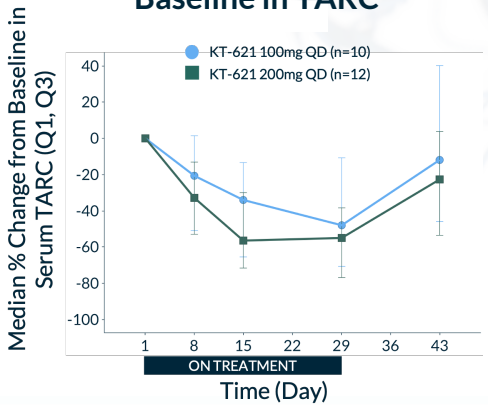


TYPE 2 BIOMARKERS

Robust reductions in Type 2 biomarkers (blood, skin, lung)

- Strong reduction in TARC, a validated Type 2 biomarker
- Robust decreases in other biomarkers: Eotaxin-3 and IL-31
- FeNO reduction in co-morbid asthma patients

Median % Change from Baseline in TARC

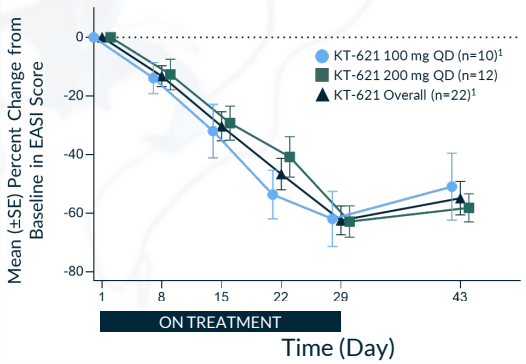


CLINCIAL ENDPOINTS

Meaningful improvements in clinical endpoints and PROs

- Robust reductions in EASI, PPNRS and improvement in PROs
- Results in line with or in some cases numerically exceeded published data for dupilumab at Week 4

Median % Change from Baseline in EASI



SAFETY

Well-tolerated with safety profile similar to Ph1 HVs

- No SAEs or Severe AEs
- No dose-dependent pattern in the TEAEs
- No related TEAEs or TEAEs leading to discontinuation
- No clinically relevant changes in vital signs, lab tests or ECGs

For more information on KT-621's Phase 1b AD clinical trial, visit Kymera's website.

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KT-621 BROADEN2 Phase 2b Trial

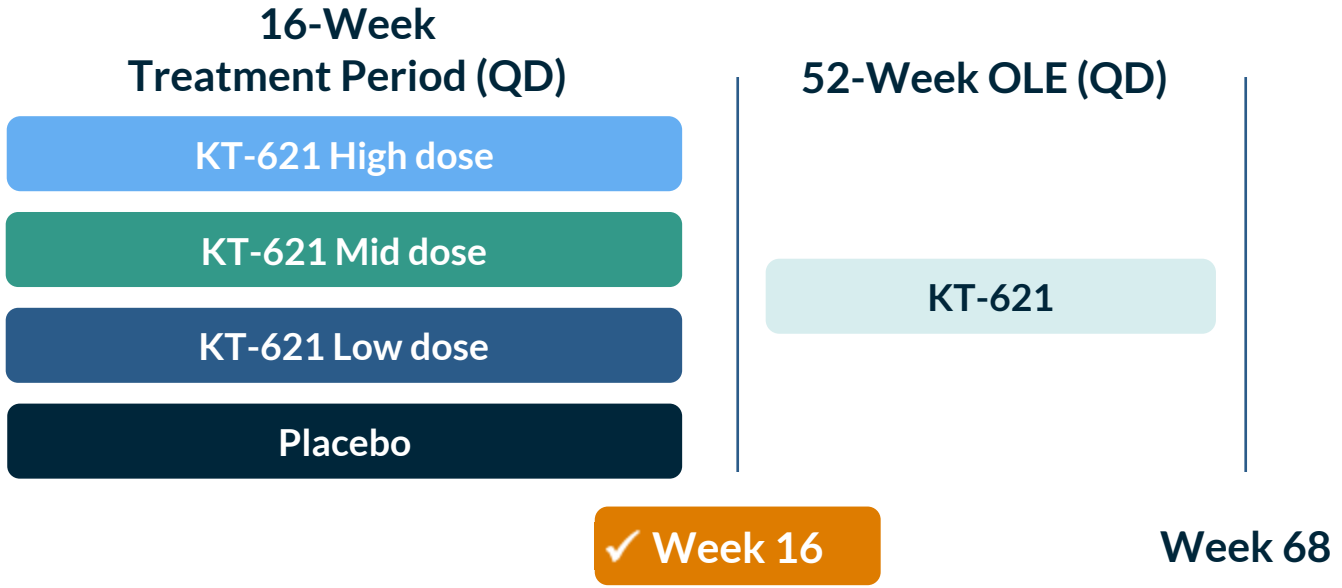
Randomized, Double Blind, Placebo-controlled, Parallel-group, Multicenter, Dose-ranging

BROADEN2 TRIAL

- Adults & adolescents aged ≥12 years with moderate-to-severe AD
- Inadequate topical response
- N=244
- Randomization 1:1:1:1
- 52-week OLE

Baseline Entry Criteria

- EASI score ≥16
- vIGA-AD score ≥3
- Peak Pruritus NRS ≥4
- BSA ≥10%
- Documented TCS failure for AD



Endpoints

- Primary endpoint: percent change from baseline in EASI score at week 16
- Secondary endpoints include:
  - EASI-75, EASI-90, vIGA-AD 0/1
  - Percent change from baseline in PPNRS and proportion of patients achieving a ≥4-point improvement from baseline

Efficacy Analysis: Rescue or discontinuation of study drug for lack of efficacy: non-responder imputation (NRI) for binary endpoints, worst observation carried forward (WOCF) for continuous endpoints; missing data or discontinuation of study drug for reasons other than lack of efficacy: multiple imputation (MI); BSA: Body Surface Area; EASI: Eczema Area and Severity Index; OLE: open-label extension; PPNRS: Peak Pruritus Numerical Rating Scale; QD: once a day; TCS: Topical Corticosteroids; vIGA-AD: Validated Investigator Global Assessment for Atopic Dermatitis.

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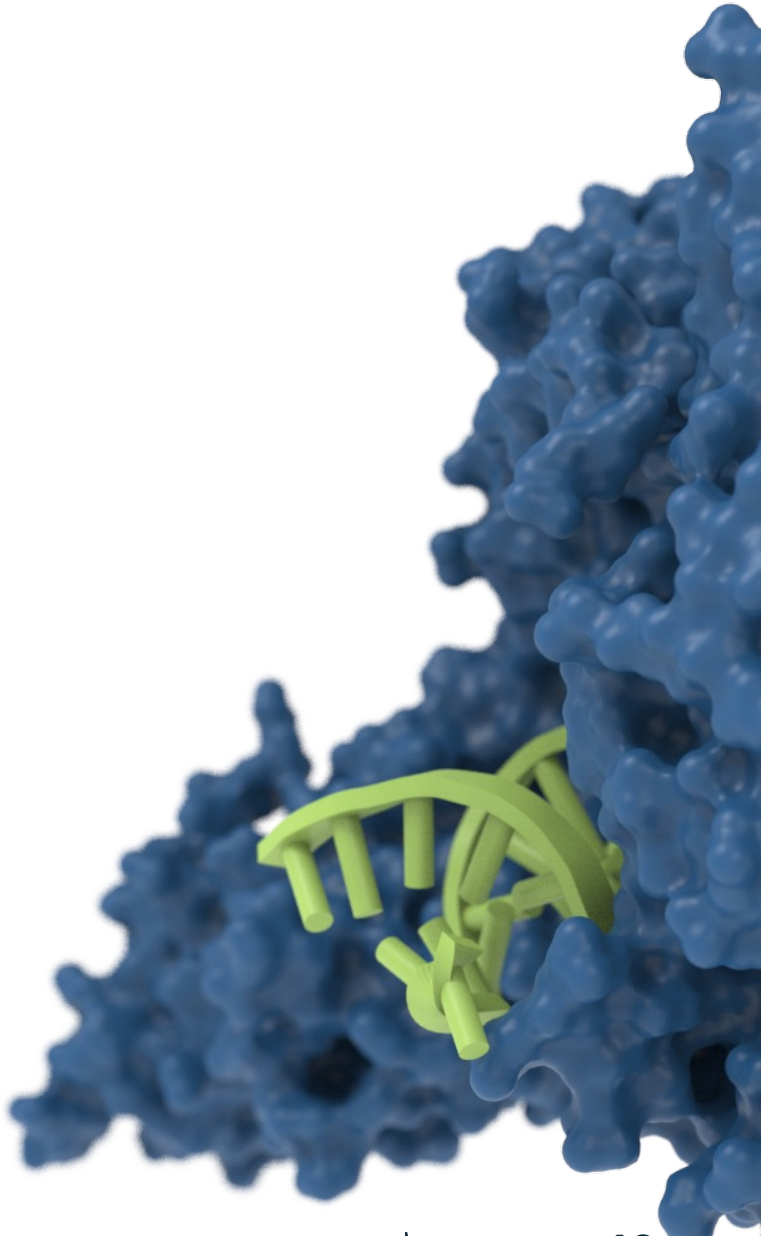
KT-621 BROADEN2 Delivers Compelling Results & Clear Path Forward

- **Well-conducted trial**, with placebo rates within historical ranges
- **Statistically significant results across all doses at Week 16,** with significant onset as early as Week 1 for EASI reductions and itch relief
- **Treatment effect across all doses comparable to marketed biologics,** with mid dose numerically superior
- **Well-tolerated with a favorable safety profile**



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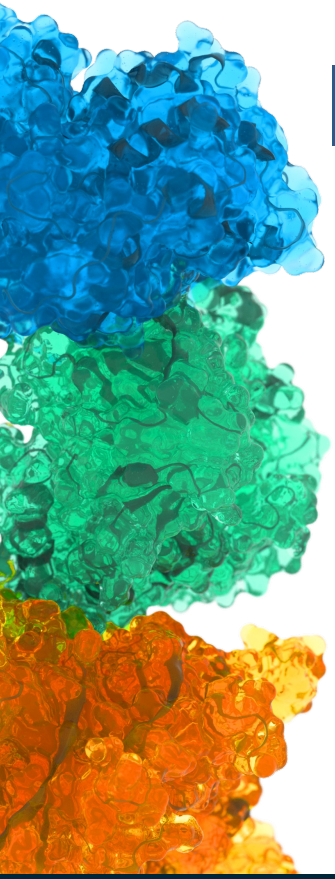
KT-621 BROADEN2 Delivers Compelling Activity Across All Doses

Mid Dose Delivered Strongest Improvements Across Endpoints, Well Positioned to Advance to Phase 3



SKIN IMPROVEMENT[1]

-72%

Mean reduction in EASI

vs. -27% placebo (p<0.0001)

62%

EASI-75

vs. 22% placebo (p<0.0001)

ITCH RELIEF[1]

52%

≥4 PPNRS Improvement

vs. 13% placebo (p<0.0001)

SAFETY

Favorable safety profile

Well-tolerated with a safety and tolerability profile consistent with IL-4/IL-13 biologics

Positive clinical data support a first-in-industry profile:
Potentially transformative safe and effective oral option for patients with Type 2 inflammatory diseases

[1]Data from KT-621 mid dose; EASI: Eczema Area and Severity Index; PPNRS: Peak Pruritus Numerical Rating Scale; vIGA-AD: Validated Investigator Global Assessment for Atopic Dermatitis.

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BROADEN2 Demographics and Baseline Characteristics

	Placebo (n=59)	Low Dose (n=61)	Mid Dose (n=61)	High Dose (n=63)	Overall (n=244)
Female, n (%)	18 (30.5)	31 (50.8)	26 (42.6)	24 (38.1)	99 (40.6)
Age, years, mean (SD)	39.6 (14.5)	39.0 (13.1)	36.9 (14.4)	38.5 (14.2)	38.5 (14.0)
Weight, kg, mean (SD)	82.9 (21.9)	80.7 (23.7)	82.4 (22.2)	80.0 (17.3)	81.5 (21.3)
Duration of AD from diagnosis, year, mean (SD)	30.6 (15.0)	28.0 (14.8)	27.8 (15.1)	26.2 (14.2)	28.1 (14.8)
Race, n (%)					
White	42 (71.2)	37 (60.7)	35 (57.4)	30 (47.6)	144 (59.0)
Black or African American	3 (5.1)	11 (18.0)	5 (8.2)	13 (20.6)	32 (13.1)
Asian	13 (22.0)	12 (19.7)	17 (27.9)	16 (25.4)	58 (23.8)
Other/Unknown	1 (1.7)	1 (1.6)	4 (6.6)	4 (6.3)	10 (4.1)
Baseline Disease Characteristics					
EASI, mean (SD)	28.7 (11.2)	27.7 (10.5)	26.4 (8.2)	28.5 (11.9)	27.8 (10.5)
vIGA-AD 4, n (%)	21 (35.6)	20 (32.8)	26 (42.6)	26 (41.3)	93 (38.1)
PPNRS, weekly mean (SD)	7.3 (1.7)	7.1 (1.6)	6.9 (1.6)	7.1 (1.4)	7.1 (1.6)
BSA affected, mean (SD)	48.4 (20.4)	41.3 (19.7)	41.2 (15.5)	46.3 (21.0)	44.3 (19.4)
Prior Advanced Systemic Therapy, n (%)	19 (32.2)	20 (32.8)	20 (32.8)	21 (33.3)	80 (32.8)
Comorbid asthma, n (%)	23 (39.0)	15 (24.6)	17 (27.9)	16 (25.4)	71 (29.1)

Of 244 randomized patients, 234 completed the 16-week double-blind treatment period; Of these, 230 enrolled in the OLE

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

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KT-621 Efficacy Data

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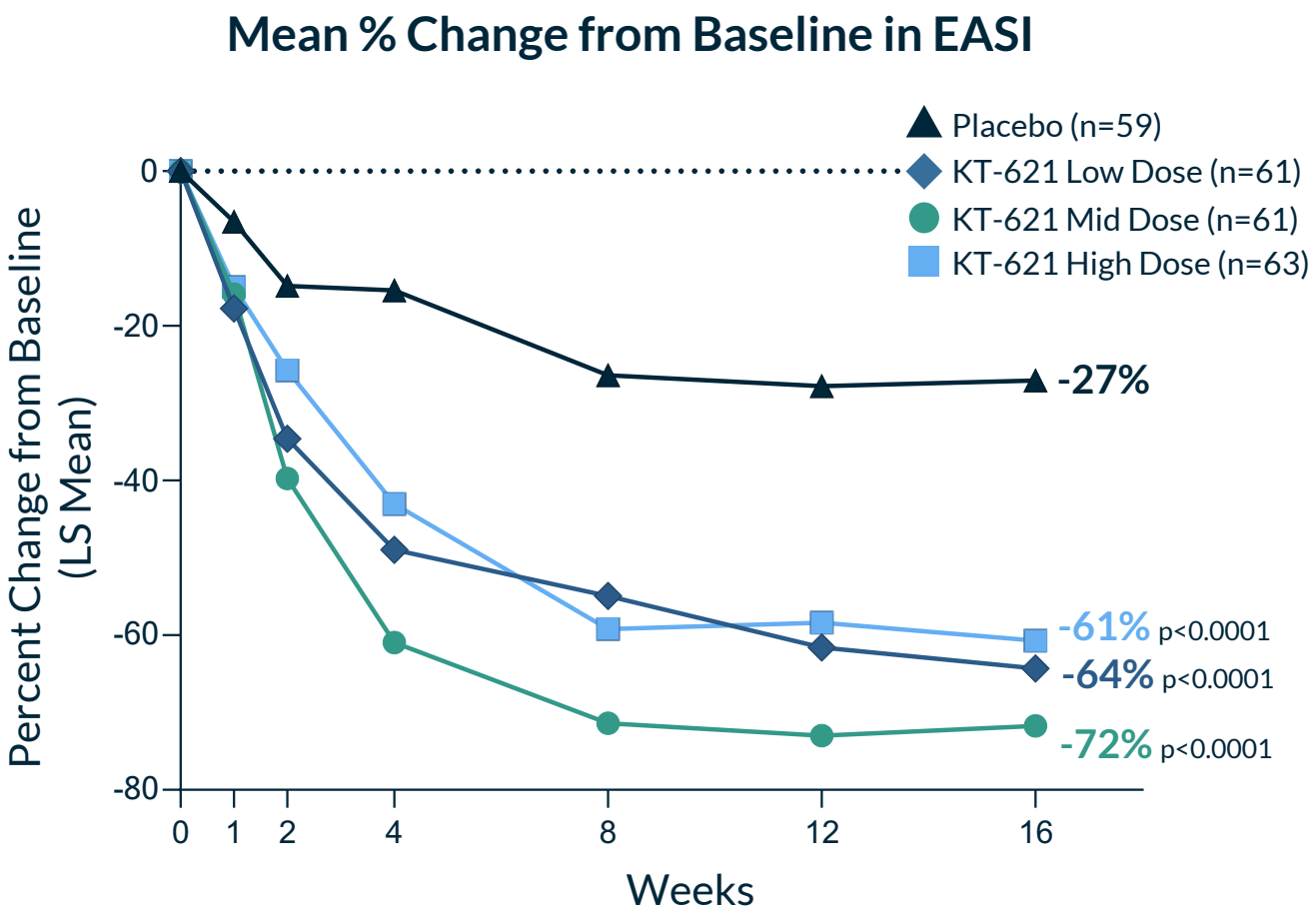
KT-621 Met Primary Endpoint Across All Doses with Rapid Onset



- All three doses demonstrated statistically significant EASI improvement versus placebo
- KT-621 treatment significantly reduced skin lesions as early as Week 1 with continued improvements through Week 16
- At Week 16, mid dose patients achieved a 72% mean reduction versus 27% with placebo
- The mid dose, predicted to achieve the desired STAT6 pharmacology, delivered the largest numerical improvements across all efficacy endpoints

Consistent pharmacology across KT-621 studies:
PK and STAT6 degradation across all doses in BROADEN2 in line with Phase 1 findings

EASI: Eczema Area and Severity Index; LS: Least Squares; PK: Pharmacokinetics.

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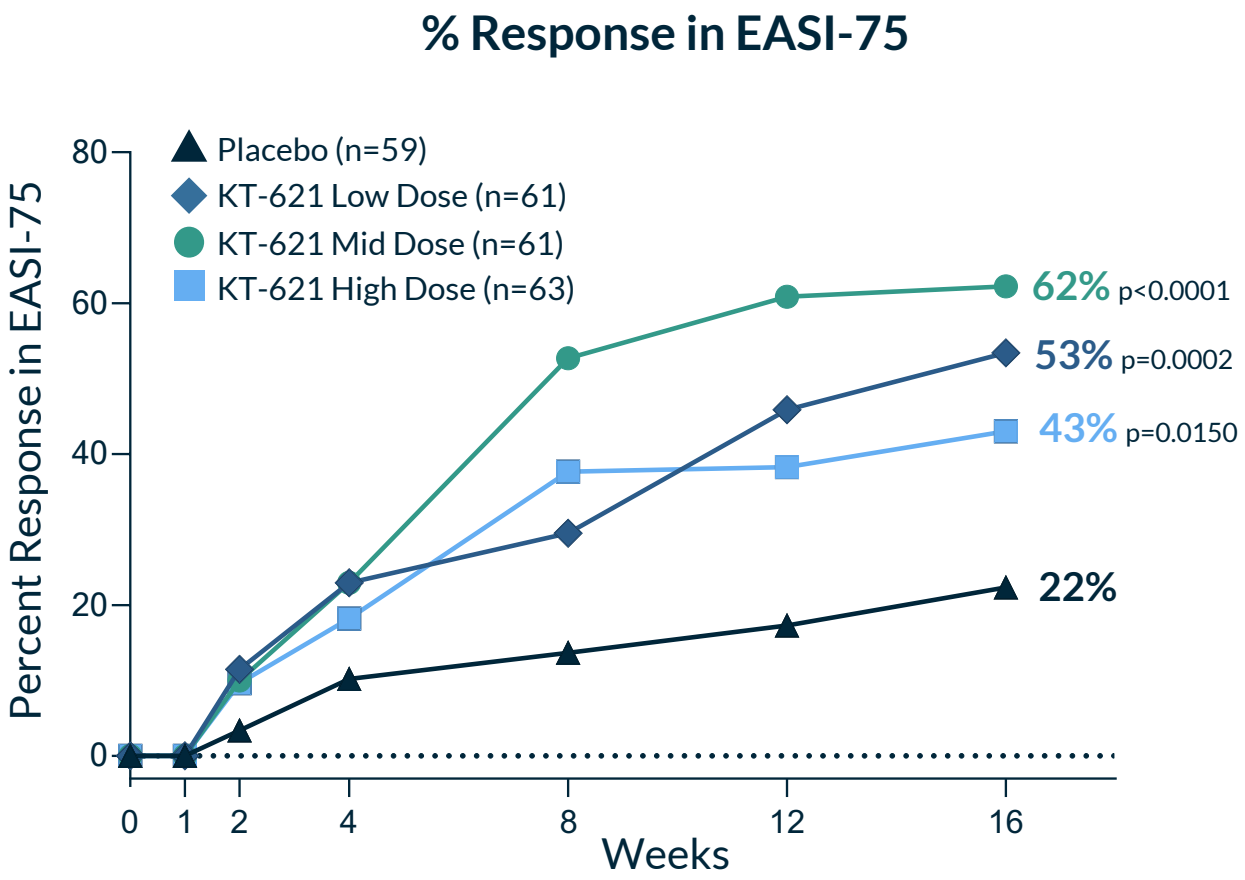
KT-621 Demonstrated Early, Substantial Clinical Improvement



- EASI-75 response rates were statistically significant versus placebo across all three doses
- Responses emerged as early as Week 2, with sustained improvement through Week 16, reflecting early onset and a substantial clinical response
- At Week 16, 62% of mid dose patients achieved at least 75% improvement in EASI versus 22% with placebo

EASI: Eczema Area and Severity Index.

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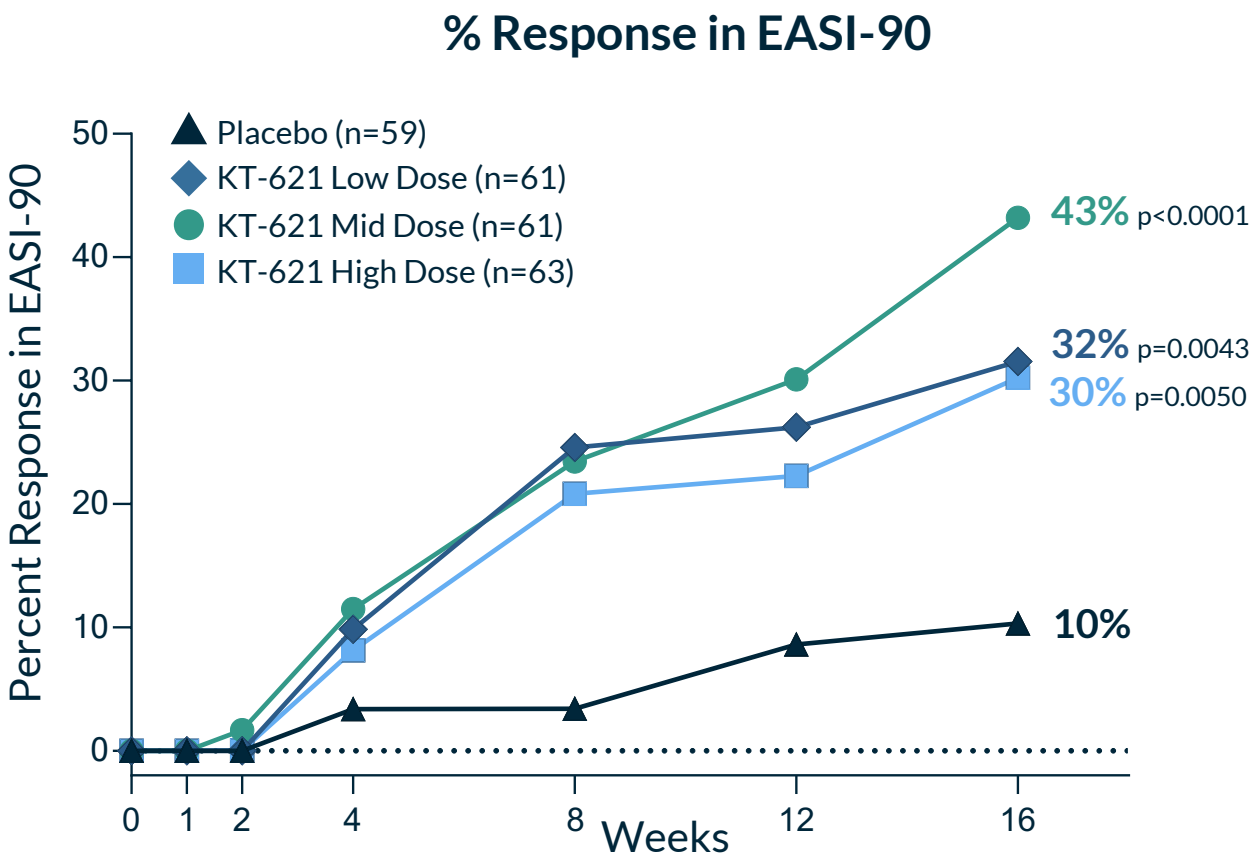
KT-621 Demonstrated Deep Clinical Responses with Continued Improvement



- EASI-90 responses were statistically significant versus placebo across all doses
- Responses emerged by Week 4 with continued improvement through Week 16
- At Week 16, 43% of mid dose patients achieved at least 90% improvement in EASI versus 10% with placebo, and demonstrated pronounced reductions in the extent and intensity of AD lesions

EASI: Eczema Area and Severity Index.

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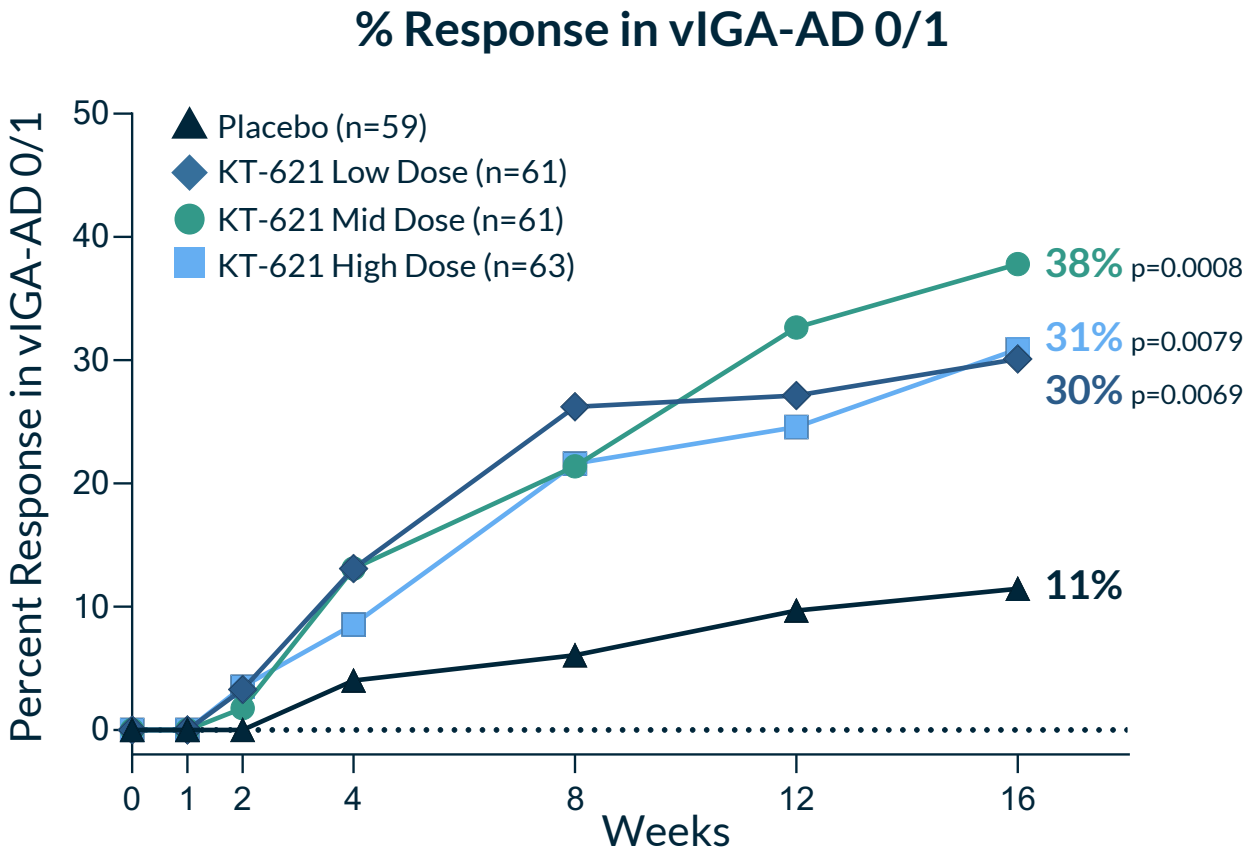
KT-621 Achieved Clear or Almost Clear Skin Through Week 16 in More Than a Third of Patients



- vIGA-AD 0/1 responses were statistically significant across all doses compared with placebo
- Clear or almost clear skin responses emerged as early as Week 2 and increased through Week 16
- At Week 16, 38% of mid dose patients achieved clear or almost clear skin with at least a 2-point improvement from baseline versus 11% with placebo

vIGA-AD: Validated Investigator Global Assessment for Atopic Dermatitis.

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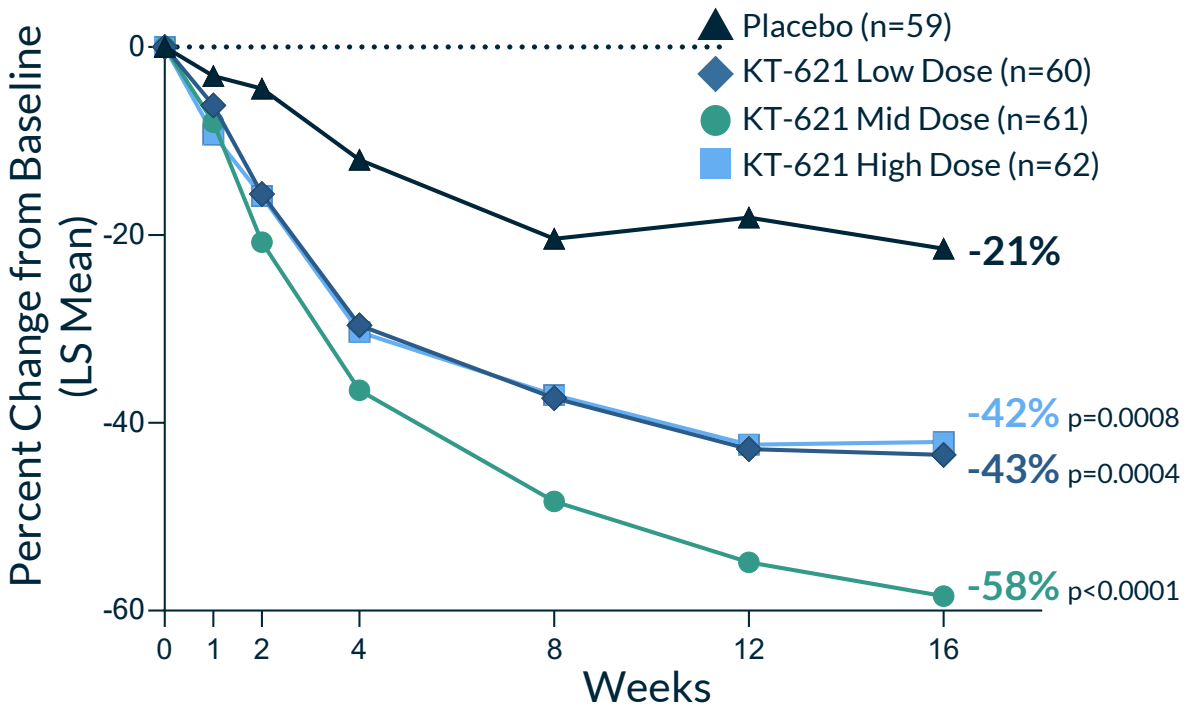
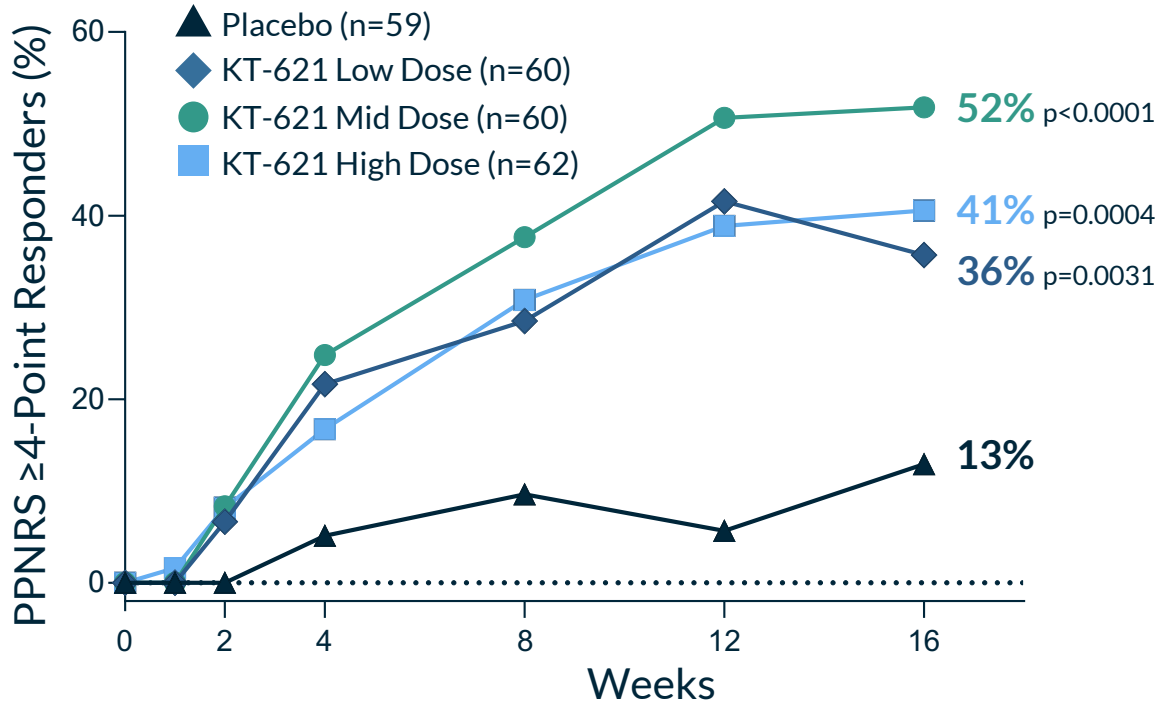
KT-621 Delivered Rapid and Substantial Itch Relief

Mean % Change from Baseline Peak Pruritus NRS



Peak Pruritus NRS ≥4-Point Improvement



- Itch reductions were significant as early as Week 1, with continued improvement through Week 16
- At Week 16, mid dose patients achieved a 58% mean reduction in itch versus 21% with placebo. More than half of mid dose patients achieved a ≥4-point improvement versus 13% with placebo

LS: Least Squares; PPNRS: Peak Pruritus Numerical Rating Scale.

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Example of Clinical Response to KT-621 in BROADEN2 Patient

Significant Improvements in Disease Severity and Quality of Life

Before and After Treatment for EASI-90 Responder

Baseline



Week 16



- Patient with severe AD inadequately controlled with prior topical therapies, including TCS, TCI and an AhR agonist
- EASI score decreased from almost 50 at baseline to less than 5 at Week 16, representing a >90% reduction
- vIGA-AD improved from 4 (severe) at baseline to 1 (almost clear) at Week 16
- PPNRS decreased from 7 at baseline to 1 at Week 16, reflecting substantial itch relief

AhR: Aryl Hydrocarbon Receptor; EASI: Eczema Area and Severity Index; PPNRS: Peak Pruritus Numerical Rating Scale; TCI: Topical Calcineurin Inhibitors; TCS: Topical Corticosteroids; vIGA-AD: Validated Investigator Global Assessment for Atopic Dermatitis.

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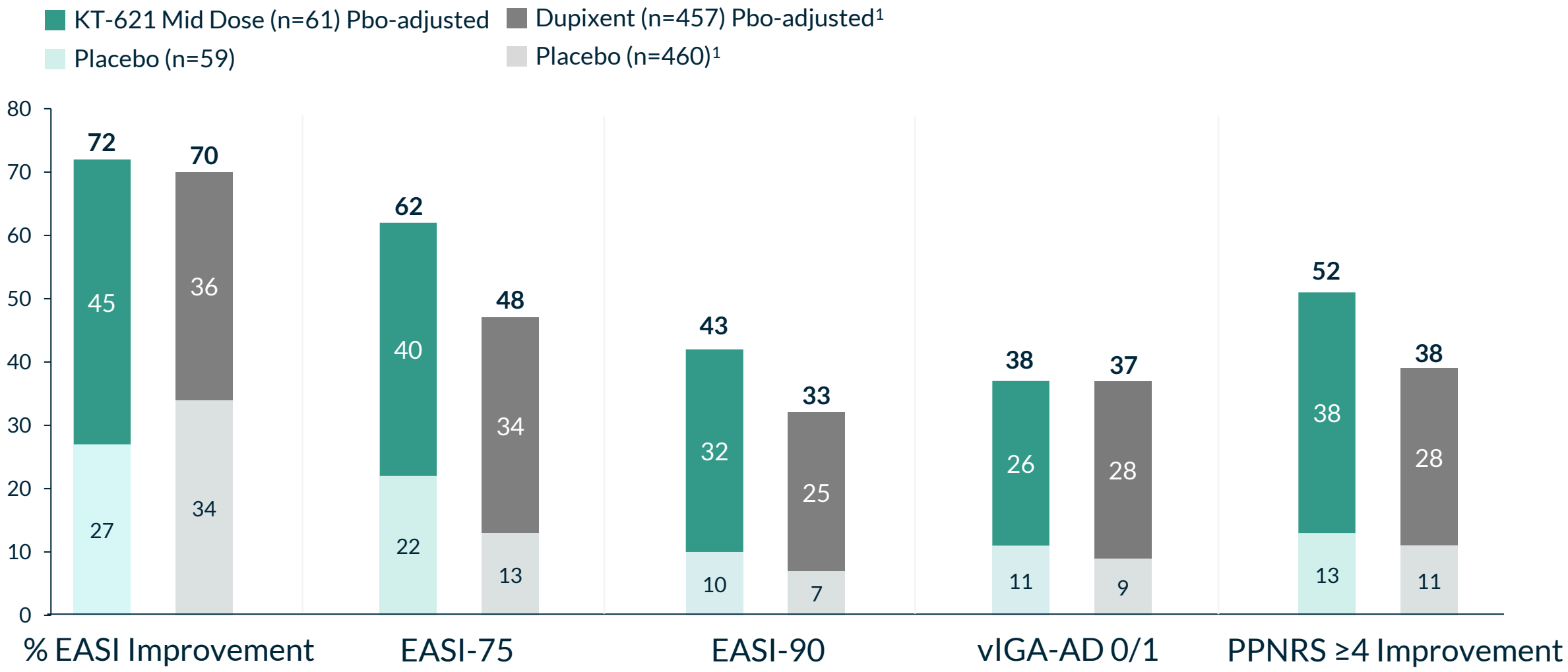
KT-621 BROADEN2 Clinical Efficacy Demonstrated Competitive Profile



[1]Dupilumab data from SOLO1 and SOLO2 studies (Thaci et al, Dermatological Science, 2019); No head-to-head trials have been conducted, cross-trial comparisons may not be reliable due to differences in molecule composition, trial protocols, dosing regimens, and patient populations and characteristics; EASI: Eczema Area and Severity Index; PBO: Placebo; PPNRS: Peak Pruritus Numerical Rating Scale; vIGA-AD: Validated Investigator Global Assessment for Atopic Dermatitis.

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KT-621 Safety Data

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BROADEN2 Phase 2b Safety Summary

Well-tolerated with a Safety and Tolerability Profile Consistent with Prior Clinical Experience of IL-4/IL-13 Biologics

	Placebo (n=59)	Low Dose (n=61)	Mid Dose (n=61)	High Dose (n=63)	All KT-621 (n=185)
Safety summary through Week 16, n (%)					
Patients reporting ≥1 TEAE	30 (50.8)	31 (50.8)	37 (60.7)	28 (44.4)	96 (51.9)
Patients reporting ≥1 severe TEAE[1]	4 (6.8)	1 (1.6)	0	1 (1.6)	2 (1.1)
Patients reporting ≥1 serious TEAE[1]	2 (3.4)	1 (1.6)	0	1 (1.6)	2 (1.1)
Patients who discontinued study drug due to TEAE	0	0	1 (1.6)	1 (1.6)	2 (1.1)
Most frequent TEAEs through Week 16 (≥5% of patients in any KT-621 arm), n (%)					
Nasopharyngitis	5 (8.5)	8 (13.1)	4 (6.6)	2 (3.2)	14 (7.6)
Conjunctivitis	0	5 (8.2)	3 (4.9)	3 (4.8)	11 (5.9)
Conjunctivitis allergic	0	4 (6.6)	5 (8.2)	2 (3.2)	11 (5.9)
Upper respiratory tract infection	5 (8.5)	2 (3.3)	4 (6.6)	2 (3.2)	8 (4.3)
Dermatitis atopic	6 (10.2)	4 (6.6)	0	0	4 (2.2)

- No dose response or meaningful imbalances in placebo versus treatment arm TEAEs except for conjunctivitis
- Pooled conjunctivitis rate[2] (all terms) of 19.7% for low dose, 13.1% for mid dose and 11.1% for high dose; all events were mild to moderate and none led to treatment discontinuation

[1]7 severe TEAEs occurred in 6 patients across all arms (atopic dermatitis 4, impetigo 1, hand fracture 1, schizoaffective disorder 1, in a participant with a prior history); of which 4 were SAEs (atopic dermatitis 2, hand fracture 1, schizoaffective disorder 1); All were deemed unrelated to study drug; [2]Pooled conjunctivitis rate includes the following preferred terms: conjunctivitis allergic, conjunctivitis, conjunctivitis bacterial, conjunctivitis viral, noninfective conjunctivitis; SAE: Serious Adverse Event; TEAE: Treatment Emergent Adverse Event.

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KT-621 Data Support Late-Stage Development & Unique Profile

BROADEN2 TRIAL

- Met primary and secondary endpoints, with statistical significance, rapid onset, and sustained or increasing responses across all doses through Week 16
- KT-621 efficacy profile is at least comparable to current standard of care
- Demonstrated favorable safety and tolerability profile
- Mid dose that delivered efficacy numerically superior to approved biologics is well positioned to advance towards Phase 3
- 96% of patients completed treatment through Week 16; 98% of these enrolled in the OLE
- Continuing to build evidence through longer-term follow-up in BROADEN2 OLE and BREADTH Phase 2b asthma trial



KT-621 has demonstrated consistent translation from STAT6 biology to clinical activity, supporting continued development in AD and beyond

OLE: Open Label Extension.

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KT-621: BREADTH Phase 2b Trial

Randomized, Double Blind, Placebo-controlled, Parallel-group, Multicenter Dose-ranging

BREADTH TRIAL

Adult, Moderate to Severe Eosinophilic Asthma Patients

Baseline entry criteria:
Blood eosinophils ≥300 cells/uL
FeNO ≥25 ppb
Pre-bronchodilator FEV1 40-80% of predicted normal

Design

- Randomized, double-blind, placebo-controlled
- ~264 patients
- Daily dose for 12-weeks
- 52-week open label extension

Dosing

- Three KT-621 doses + one placebo (1:1:1:1)

Endpoints

- Primary endpoint: Change from baseline in pre-bronchodilator FEV1 at week 12
- Secondary endpoints include:
  - Change from baseline in ACQ-5, AQLQ

Key Trial Aim

Establish clinical activity and safety in **asthma to select Phase 3 dose to support registrational studies** in multiple respiratory indications

Status update:
Ongoing;
Data expected late 2027

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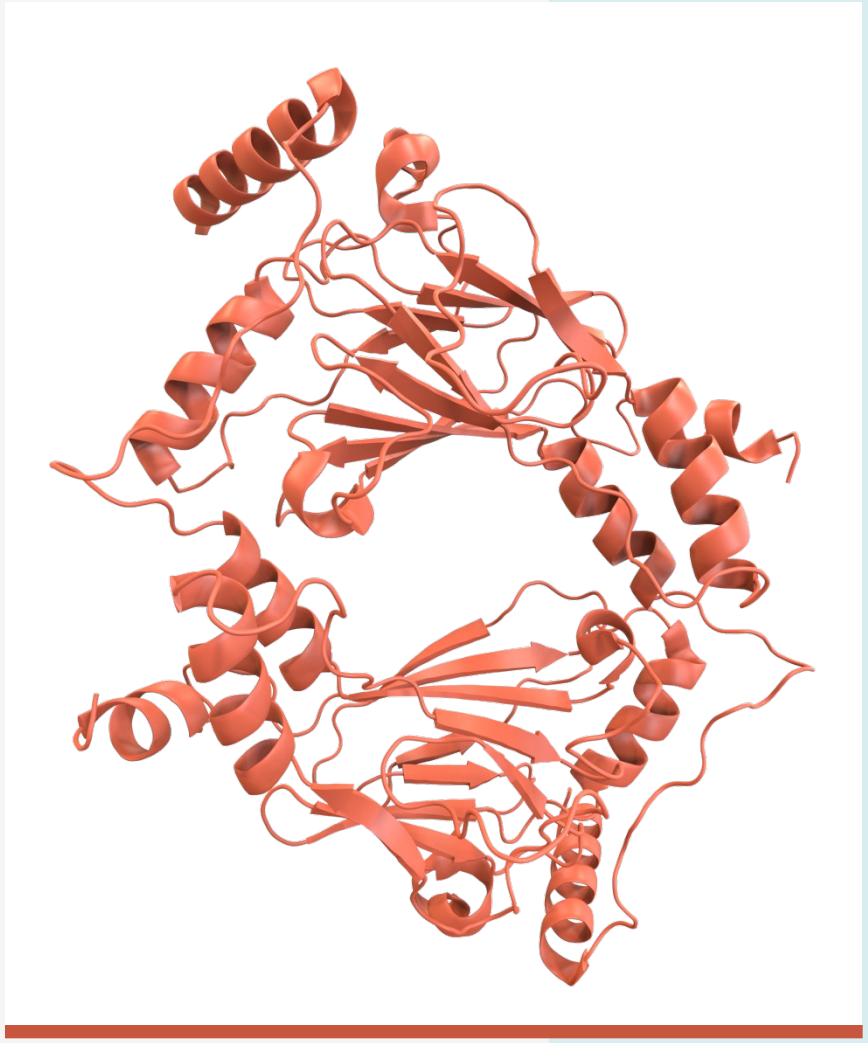

First-in-Class Oral IRF5 Degrader Program

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KT-579: First-in-Class Oral IRF5 Degrader



VALIDATED BIOLOGY

- Genetically validated transcription factor in autoimmunity
- Regulates inflammatory cytokines, IFN responses and B-cell biology

UNIQUE PROFILE

- Potent, selective oral degrader with robust preclinical activity in autoimmune disease models
- Phase 1 showed ≥90% median blood IRF5 degradation across multiple MAD doses and robust biomarker suppression
- Favorable safety and tolerability profile in healthy volunteers

STATUS & MILESTONES

- LUMORA Phase 1b lupus trial planned to initiate by early 2027

KT-579 has the potential to address multiple drivers of autoimmunity with a single oral medicine

IFN: interferon; MAD: multiple ascending dose.

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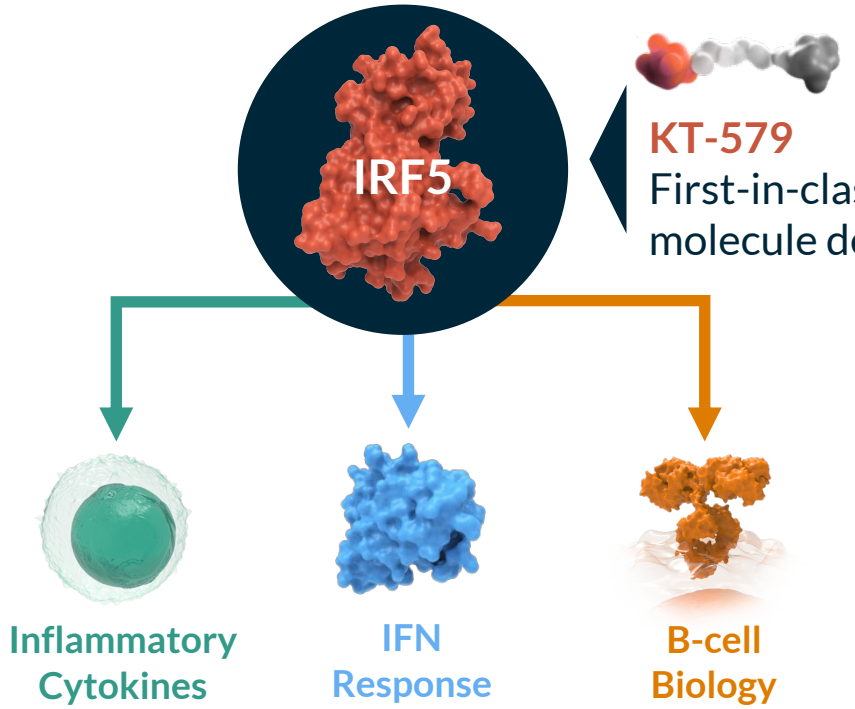
A New Treatment Paradigm for Serious Autoimmune Diseases

IRF5 hyperactivation impacts three validated disease-defining pathways



Historically Undrugged Target

IRF5 is a genetically validated transcription factor with impact on multiple clinically validated pathways

Compelling Preclinical Profile

KT-579 demonstrated robust preclinical activity in primary human cells, patient donor cells and *in vivo* models of autoimmune diseases with a favorable safety profile

Positive Early Clinical Data

In healthy volunteers, KT-579 showed strong clinical translation with deep degradation, robust reductions of inflammatory biomarkers, and a favorable safety profile

Broad Patient Impact

Phase 1 data unlocks multiple clinical opportunities starting from upcoming LUMORA lupus trial

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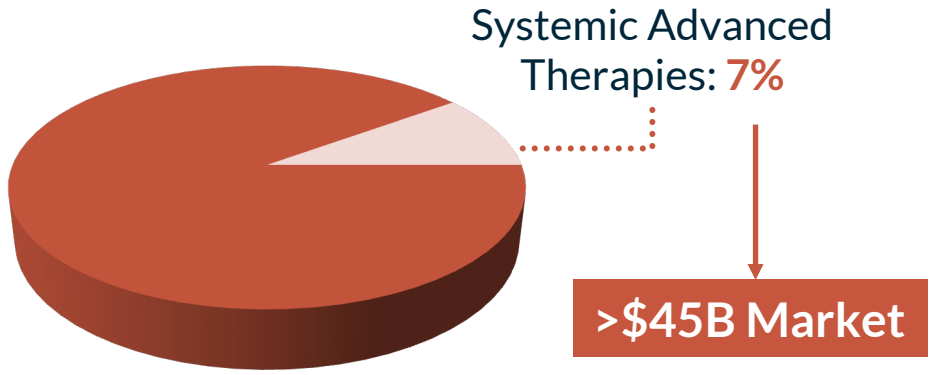
IRF5: A Novel Mechanism in High Unmet Need Indications in I&I

>10M



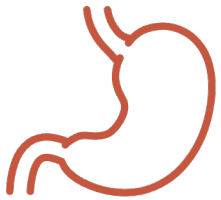
TOTAL POTENTIAL PATIENT IMPACT[1]

IRF5 functional risk variants associate with increased susceptibility to multiple diseases, and IRF5 regulated pathways have been clinically validated by multiple drugs



RHEUMATOLOGY

	PATIENTS[1]
RA	4.7M
Lupus[2]	1.2M
Sjögren's	765K
SSc	200K
DM	>100K



GASTROENTEROLOGY

UC	1.9M
CD	1.5M

[1]GlobalData (2023 diagnosed prevalent patient population US/EU5/JP); [2]Lupus includes SLE, CLE, LN.

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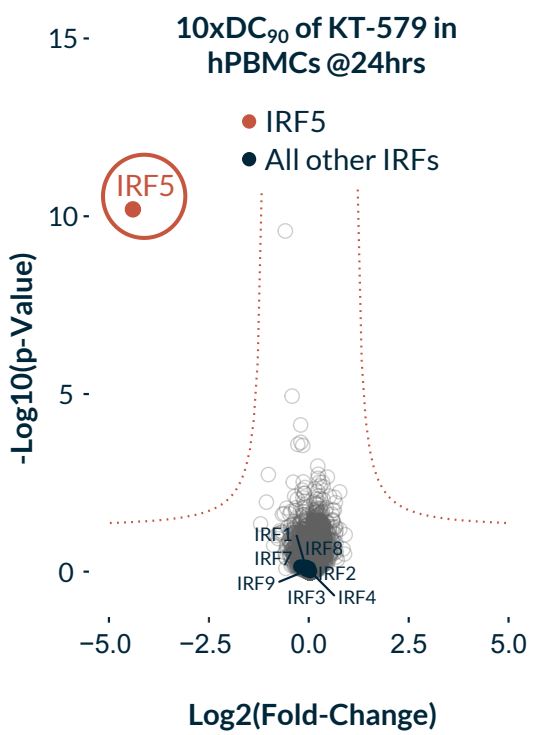
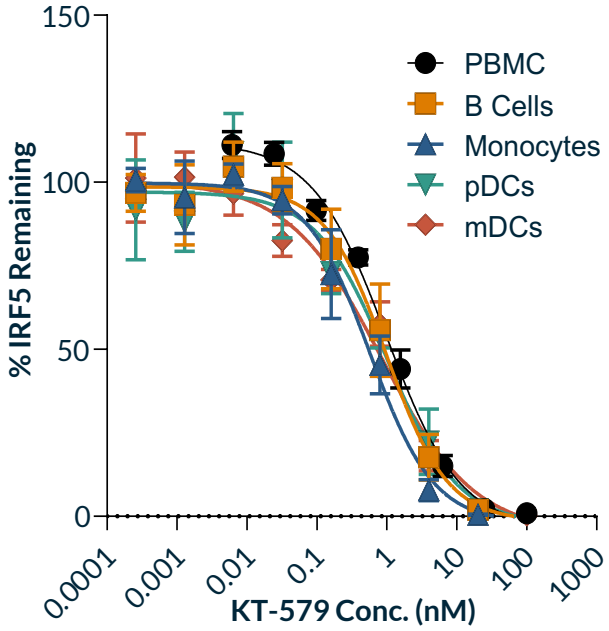
KT-579: Compelling Preclinical Profile

Novel Oral Mechanism to Block Multiple Validated Disease-defining Pathways

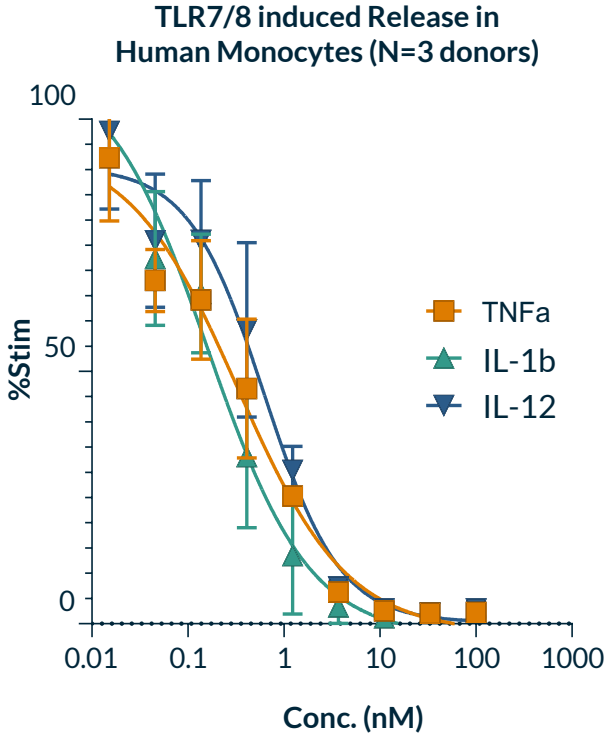
Exquisite Selectivity



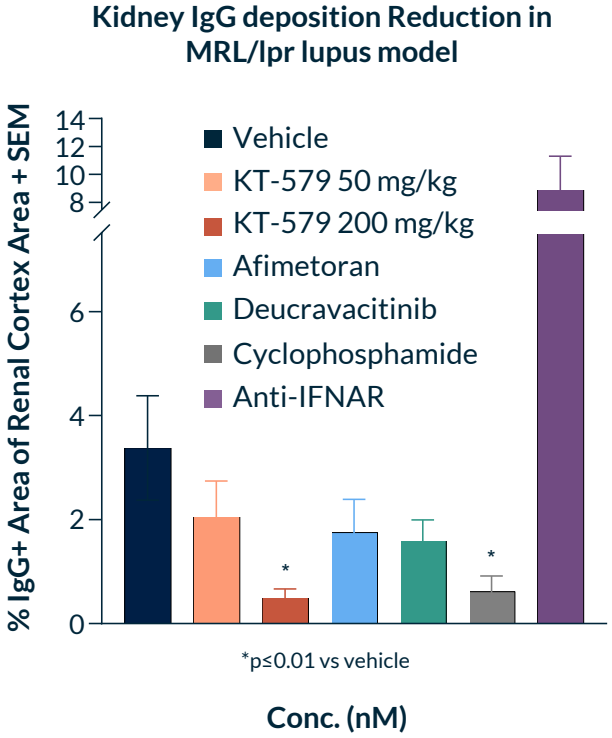
Robust Degradation in Relevant Cell Types



Full Inhibition of IRF5 Regulated Cytokines



Excellent Preclinical Activity Superior to Targeted Agent Comparators



For more information on KT-579's preclinical profile, visit Kymera's website.

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KT-579 Phase 1 Healthy Volunteer Clinical Trial Data



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KT-579: First IRF5 Agent to Enter Clinical Evaluation

Phase 1 Trial: Double-blind, Placebo-controlled SAD and MAD in Healthy Volunteers

PART A
Single Ascending Dose (SAD)

Total 48 Healthy Volunteers
8 per cohort
(6:2 KT-579:Placebo)

PART B
Multiple Ascending Dose (MAD)

14x daily doses

Total 72 Healthy Volunteers
12 per cohort
(9:3 KT-579:Placebo)

Dosing
- Evaluated a range up to 600 mg with multiple daily doses for up to 14 days

Primary
- Safety & tolerability

Secondary
- Pharmacokinetic measures

Exploratory
- IRF5 protein levels in blood
- *Ex vivo* blood biomarkers and pathway gene transcripts:
  - Inflammatory cytokines
  - IFN response
  - B-cell biology



Key Trial Aim

Key trial aim to show that **KT-579 can robustly degrade IRF5 in blood** at doses that are safe and well-tolerated

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Phase 1 SAD/MAD Demographics

	SAD 1-6: PBO (n=12)	SAD 1-6: KT-579 (n=36)	MAD 1-6: PBO (n=18)	MAD 1-6: KT-579 (n=54)
Gender, n (%)				
Female	3 (25.0%)	10 (27.8%)	7 (38.9%)	7 (13.0%)
Male	9 (75.0%)	26 (72.2%)	11 (61.1%)	47 (87.0%)
Age, years, median (range)	38.0 (19, 53)	37.5 (22, 55)	38 (21, 51)	35.5 (20, 54)
Ethnicity, n (%)				
Hispanic or Latino	3 (25.0%)	7 (19.4%)	4 (22.2%)	18 (33.3%)
Non-Hispanic or Latino	9 (75.0%)	29 (80.6%)	14 (77.8%)	36 (66.7%)
Race, n (%)				
White	9 (75.0%)	25 (69.4%)	15 (83.3%)	35 (64.8%)
Black or African American	1 (8.3%)	8 (22.2%)	1 (5.6%)	15 (27.8%)
Asian	1 (8.3%)	1 (2.8%)	1 (5.6%)	2 (3.7%)
Multiple	1 (8.3%)	2 (5.6%)	1 (5.6%)	2 (3.7%)

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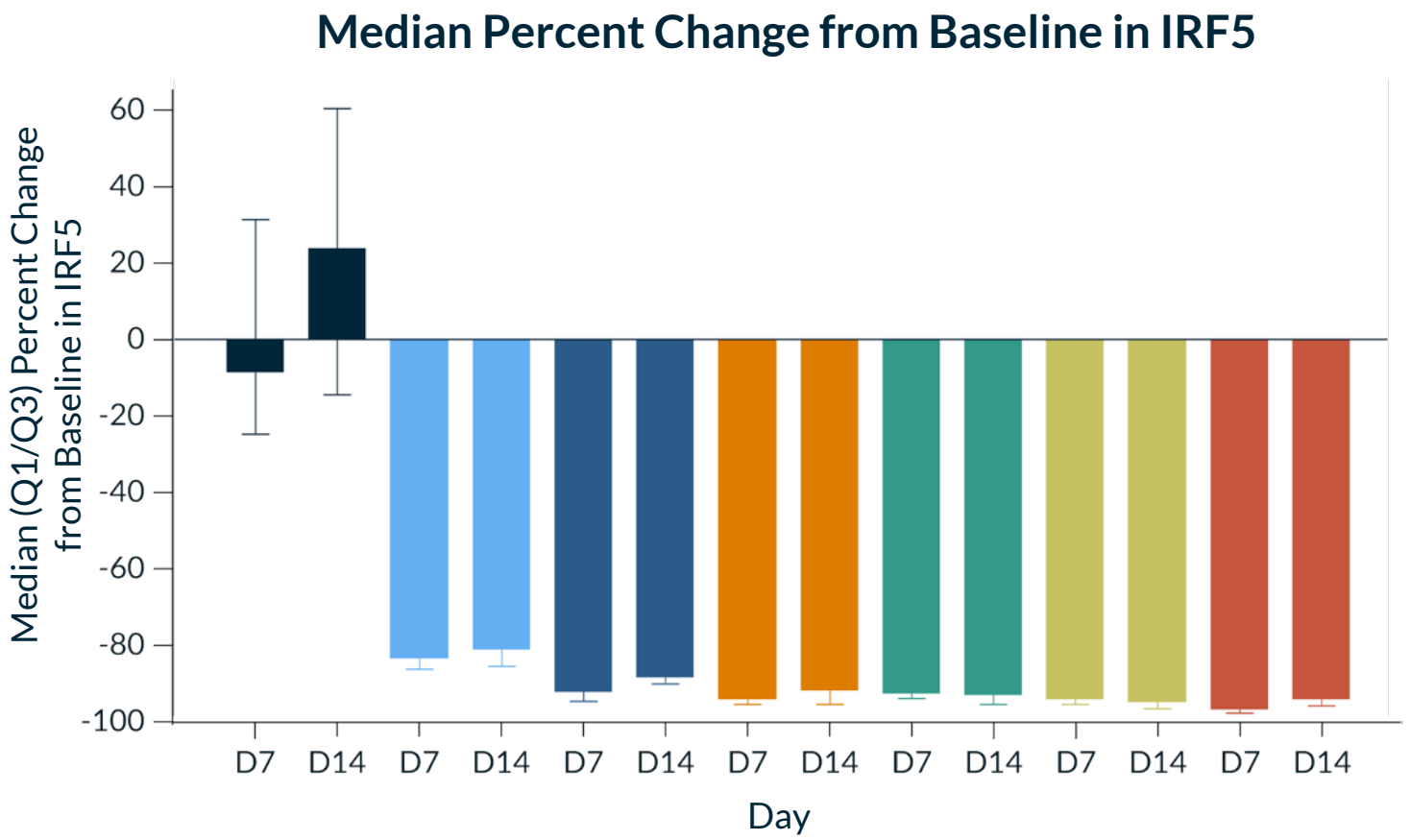
Multiple Daily Doses of KT-579 Achieved >90% IRF5 Degradation in Blood Across Almost All Dose Levels



	Median % IRF5 Change	n	D7	D14
	Placebo	18	-9%	24%
	Dose 1	**9**	**-83%**	**-81%**
	Dose 2	**9**	**-92%**	**-89%**
	Dose 3	**9**	**-94%**	**-92%**
	Dose 4	**9**	**-93%**	**-93%**
	Dose 5	**9**	**-94%**	**-95%**
	Dose 6	**9**	**-97%**	**-94%**

- KT-579 dosed daily for 14 days showed dose dependent exposure and degradation
- Degradation at every dose by Day 14 was statistically significant ($p \leq 0.0001$ for all comparisons of KT-579 vs. Placebo)

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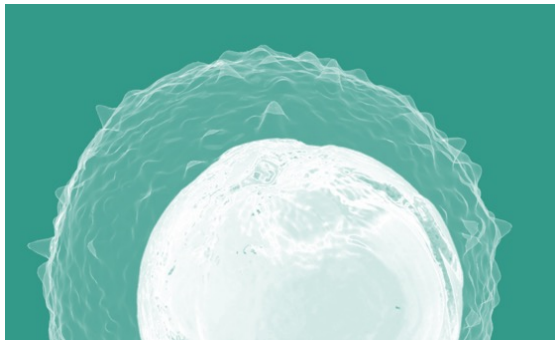
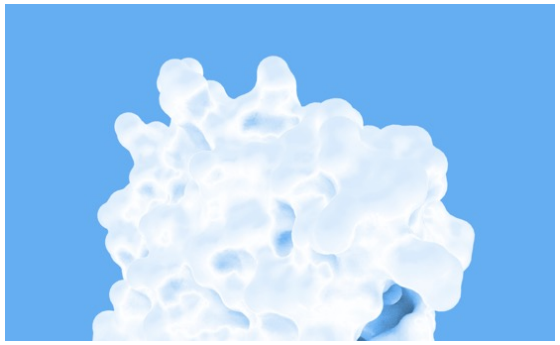
Complementary Ex Vivo Biomarker Assays Capture the Breadth of IRF5 Biology Across Three Key Immune Pathways[1]

INFLAMMATORY CYTOKINES[2]



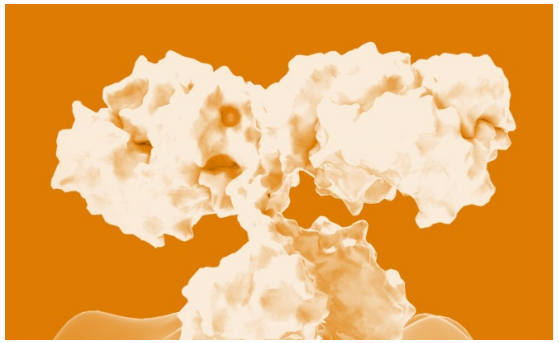
- IRF5 promotes production of key inflammatory cytokines in myeloid cells
- When dysregulated, these responses can sustain chronic inflammation, relevant to several autoimmune diseases

INTERFERON RESPONSE[3]



- IRF5 connects nucleic-acid sensing with broader interferon-associated transcriptional responses
- Interferon dysregulation is a central feature of several autoimmune diseases, where it amplifies immune activation and contributes to inflammation

B-CELL BIOLOGY[4]



- IRF5 contributes to immune responses linked to B-cell activation and autoantibody-related biology
- This axis is particularly relevant in several autoantibody driven autoimmune diseases

[1]Wang et al., *Signal Transduction and Targeted Therapy*, 2024; [2]Krausgruber et al., *Nature Immunology*, 2011; [3]Barnes et al., *J Biol Chem*, 2001; [4]De et al., *Frontiers in Immunology*, 2018.

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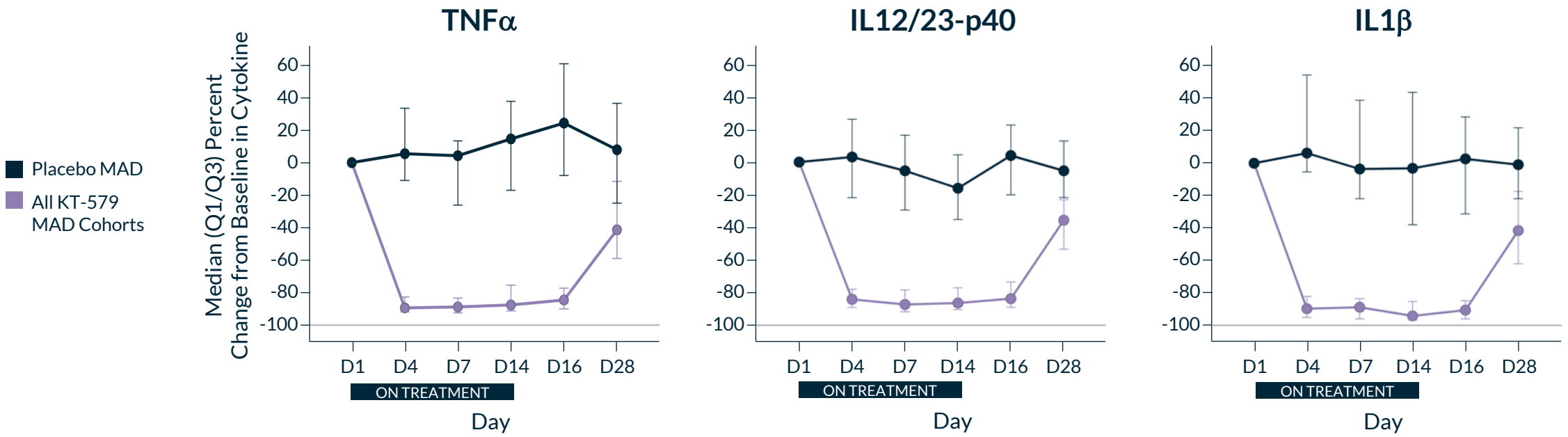
Multiple Daily Doses of KT-579 Achieved Deep Inhibition of Clinically Validated Inflammatory Cytokines



Median Cytokine Inhibition Day 14 - R848 (TLR7/8) Ex Vivo Stimulation

Median Inhibition Day 14[1]	TNFα	IL12/23-p40	IL1β
Placebo	15%	-16%	-3%
All KT-579 MAD Cohorts	**-88%**	**-87%**	**-94%**

[1]Placebo, n=18, and all KT-579 MAD cohorts, n=52, two subjects were excluded due to a processing error.

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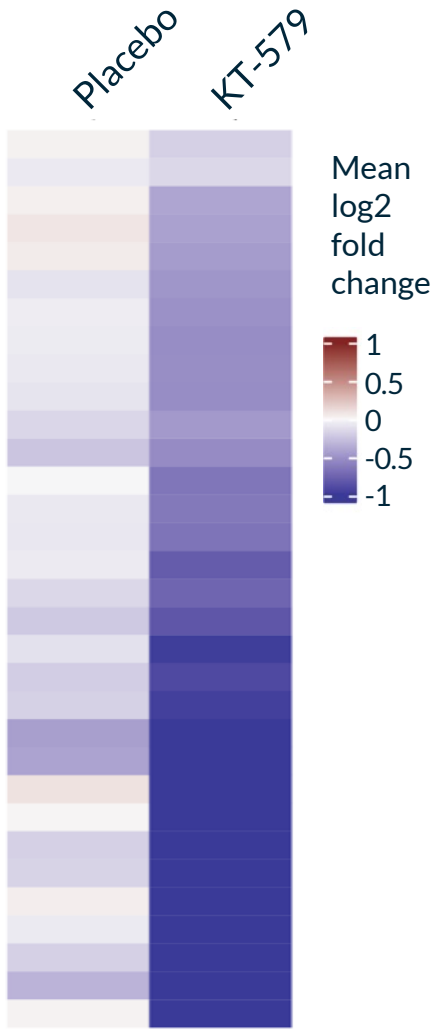
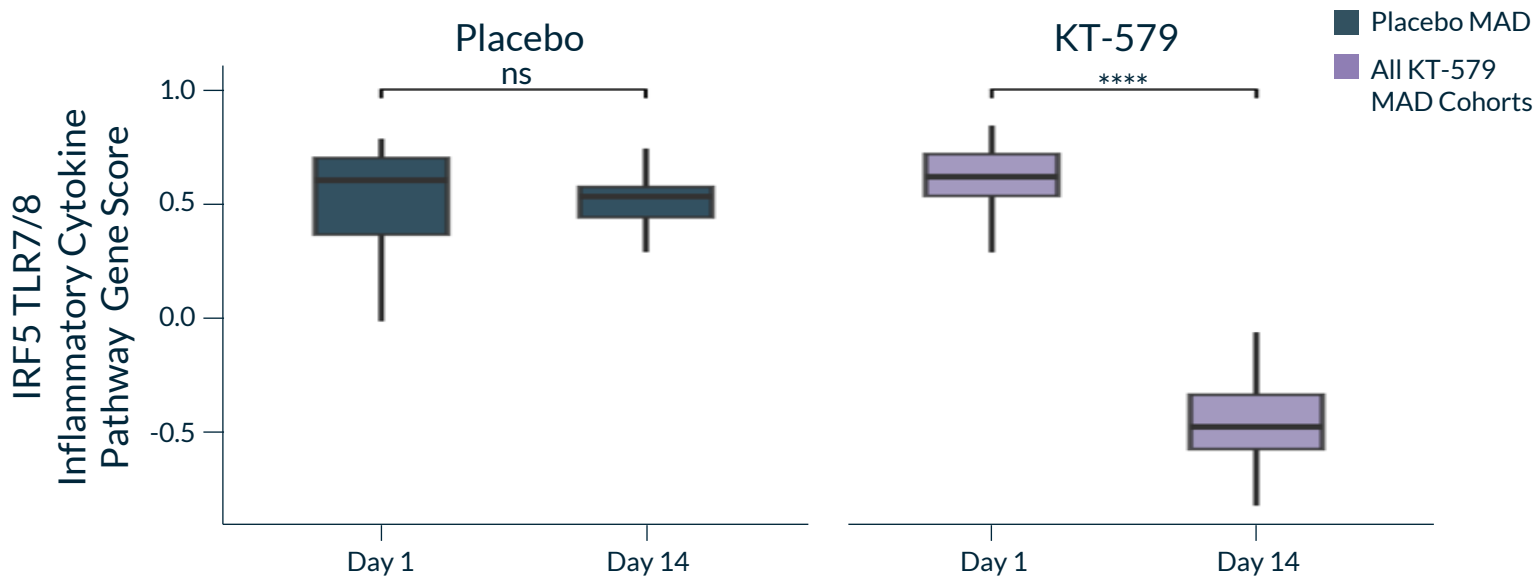
KT-579 Downregulates Inflammatory Cytokine Pathway Genes



Genes Significantly Decreased Across MAD Cohorts[1]

Expression at Day 14 compared to pre-dose

Inflammatory Cytokine Gene Score



- Significant reductions in multiple genes, selected from a core IRF5 mediated TLR7/8 signature focused on inflammatory pathways
- Strong concordance between KT-579 effects on gene and protein reductions in inflammatory cytokines such as IL12β and TNF

[1]Genes from pre-defined gene sets from preclinical KT-579 data across ex vivo stimulations; Names of genes have been removed; Significance defined as downregulated ≥ 1.5x with adj. P value ≤ 0.05; All KT-579 MAD cohorts n=52, Placebo n=18; Signature scores were calculated using GSVA; Paired t-test on GSVA scores; P-values are shown as *** for p < 0.001, **** for p < 0.0001.

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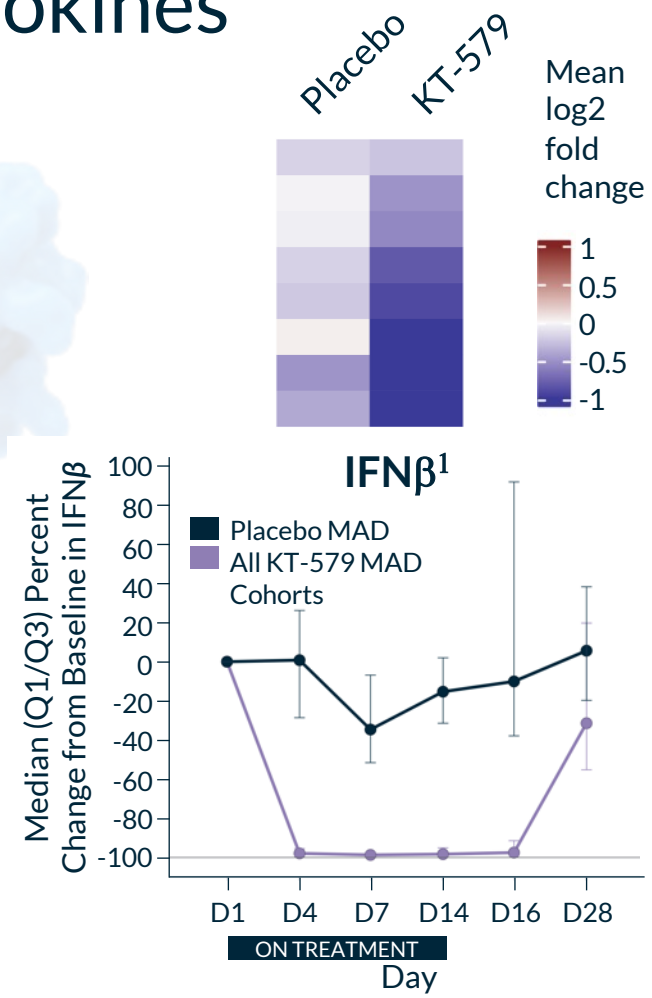
KT-579 Downregulates Interferon Pathway Genes and Cytokines

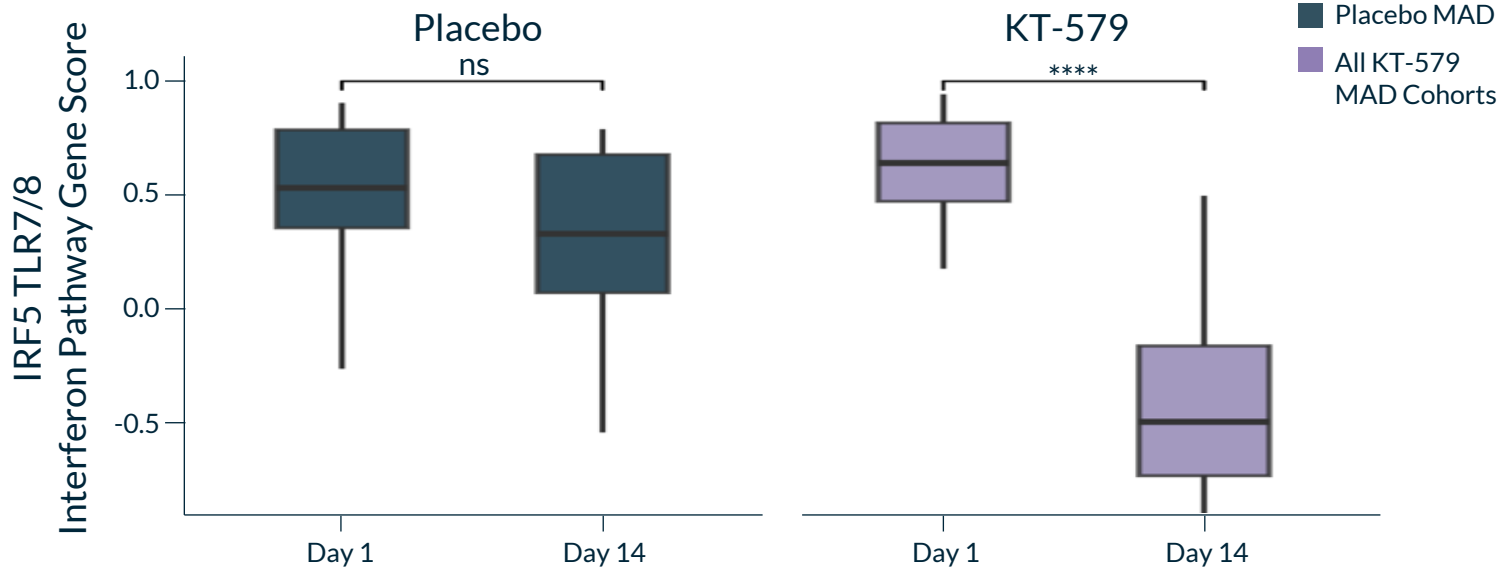


Median Inhibition Day 14[1]	IFNβ
Placebo	-15%
All KT-579 MAD Cohorts	**-98%**

Genes Significantly Decreased Across MAD Cohorts[2]

Expression at Day 14 compared to Pre-dose

Interferon Gene Score



- Significant reductions in multiple genes selected from a core IRF5 mediated TLR7/8 signature focused on the interferon pathway

[1]All KT-579 MAD cohorts, n=54, Placebo, n=18; [2]Genes from pre-defined gene sets from preclinical KT-579 data across ex vivo stimulations; Names of genes have been removed; Significance defined as downregulated ≥ 1.5x with adj. P value ≤ 0.05; All KT-579 MAD cohorts n=52, Placebo n=18; Signature scores were calculated using GSVA; Paired t-test on GSVA scores; P-values are shown as *** for p < 0.001, **** for p < 0.0001.

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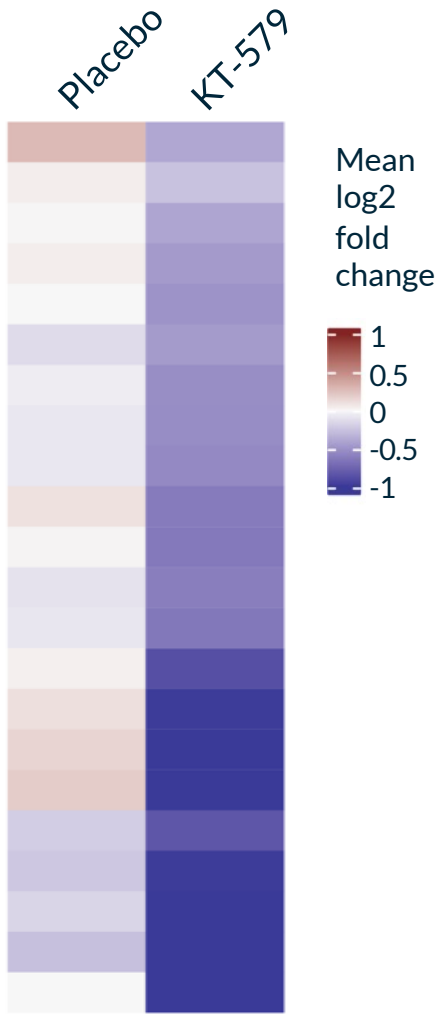
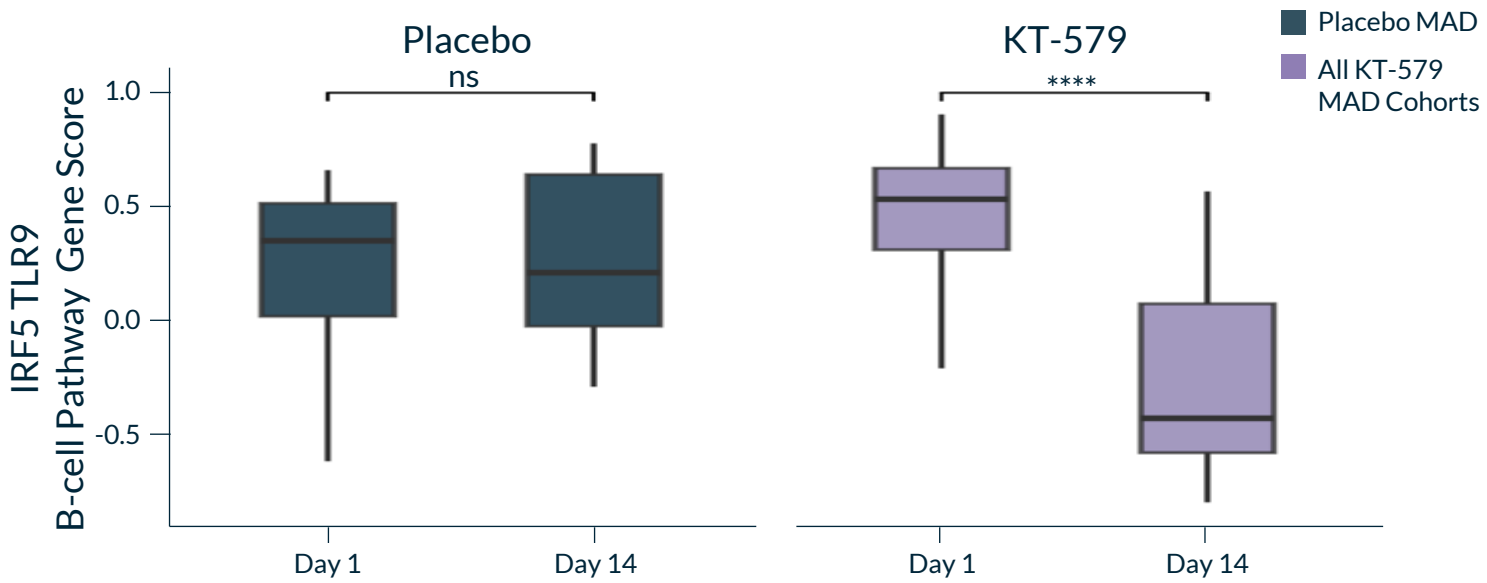
KT-579 Downregulates B-cell Pathway Genes



Genes Significantly Decreased Across MAD Cohorts[1]

Expression at Day 14 compared to Pre-dose



- Significant reductions in multiple genes selected from a core IRF5 mediated TLR9 signature focused on DNA-sensing and B-cell activation pathways

[1]Genes from pre-defined gene sets from preclinical KT-579 data across ex vivo stimulations; Names of genes have been removed; Significance defined as downregulated ≥ 1.5x with adj. P value ≤ 0.05; All KT-579 MAD cohorts n=52, Placebo n=18; Signature scores were calculated using GSVA; Paired t-test on GSVA scores; P-values are shown as *** for p < 0.001, **** for p < 0.0001.

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Healthy Volunteer SAD/MAD Safety Summary

KT-579 Well Tolerated Across All Doses Evaluated with Safety Profile Comparable to Placebo

- No SAEs
- No Severe AEs
- No AEs leading to study drug discontinuation
- No dose-dependent pattern in the TEAEs
- No imbalances in the rates of infections
- No clinically relevant changes in vital signs, laboratory tests, or ECGs

TRAEs that Occurred in >1 Participant (MAD/SAD* Cohort)

AE Term (*severity*)	MAD 1-6 Placebo (n=18)	MAD 1-6 KT-579 (n=54)
Headache (*mild*)	4 (22.2%)	3 (5.6%)
Myalgia (*mild*)	0 (0.0%)	2 (3.7%)
Constipation (*mild*)	2 (11.1%)	1 (1.9%)

*No TRAEs occurred in more than one participant in the SAD cohorts

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

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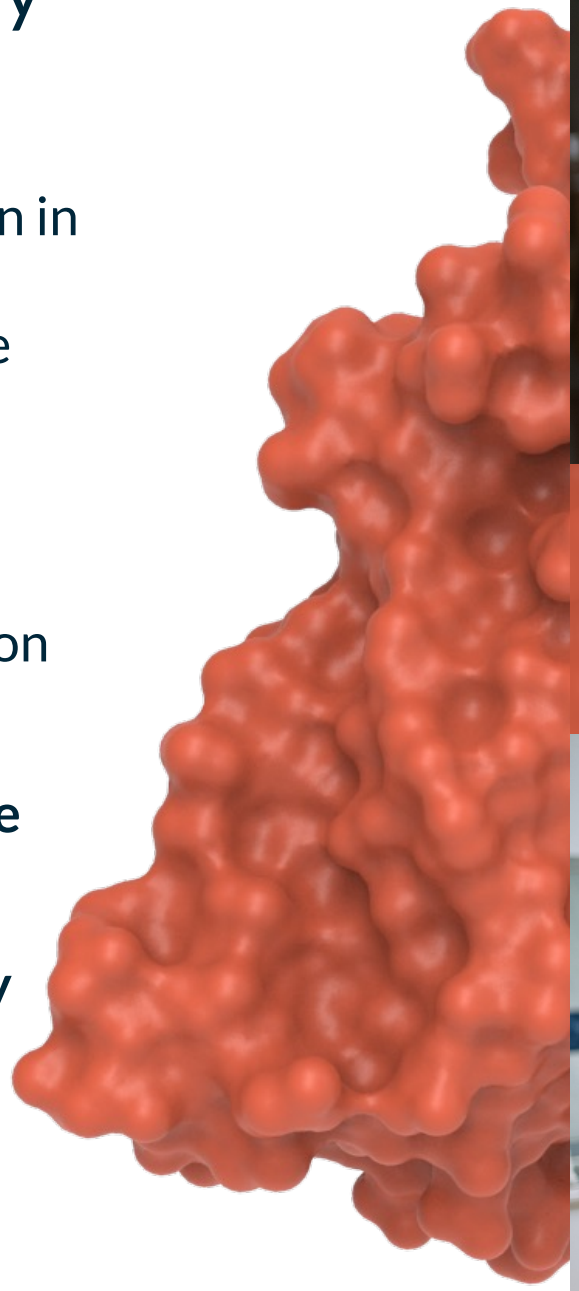
KT-579 Phase 1 Summary

- Deep, sustained IRF5 degradation in blood, with **≥90% median IRF5 degradation** across multiple dose levels in MAD
- Robust **suppression of disease-relevant blood biomarkers** for inflammatory cytokines, interferon response, and B-cell biology
- Downregulation of **pathway gene transcripts**
- **Favorable safety and tolerability profile in healthy volunteers**





A Novel Oral Mechanism for Serious Autoimmune Diseases



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KT-579: LUMORA Phase 1b Trial

Biomarker Study for First-in-Class Mechanism in Lupus Patients

lumora TRIAL

Adult Patients with Systemic Lupus Erythematosus

Baseline entry criteria:

- ANA ≥1:80
- BILAG-2004 score A or B[1]
- Oral corticosteroid background therapy permitted

Design

- Randomized, double-blind, placebo-controlled
- ~30 patients

Dosing

- KT-579 or placebo (2:1)
- Dosing for 16-weeks

Endpoints

- Safety and tolerability
- Pharmacokinetics
- IRF5 protein levels in blood
- Biomarkers
  - Inflammatory cytokines
  - IFN response
  - B-cell biology
- Exploratory disease endpoints

Key Trial Aim

Biomarker study to demonstrate that **KT-579 can modulate underlying disease biology** with a favorable safety and tolerability profile

Status update:
Trial start by early 2027

[1]In mucocutaneous and/or musculoskeletal domains.

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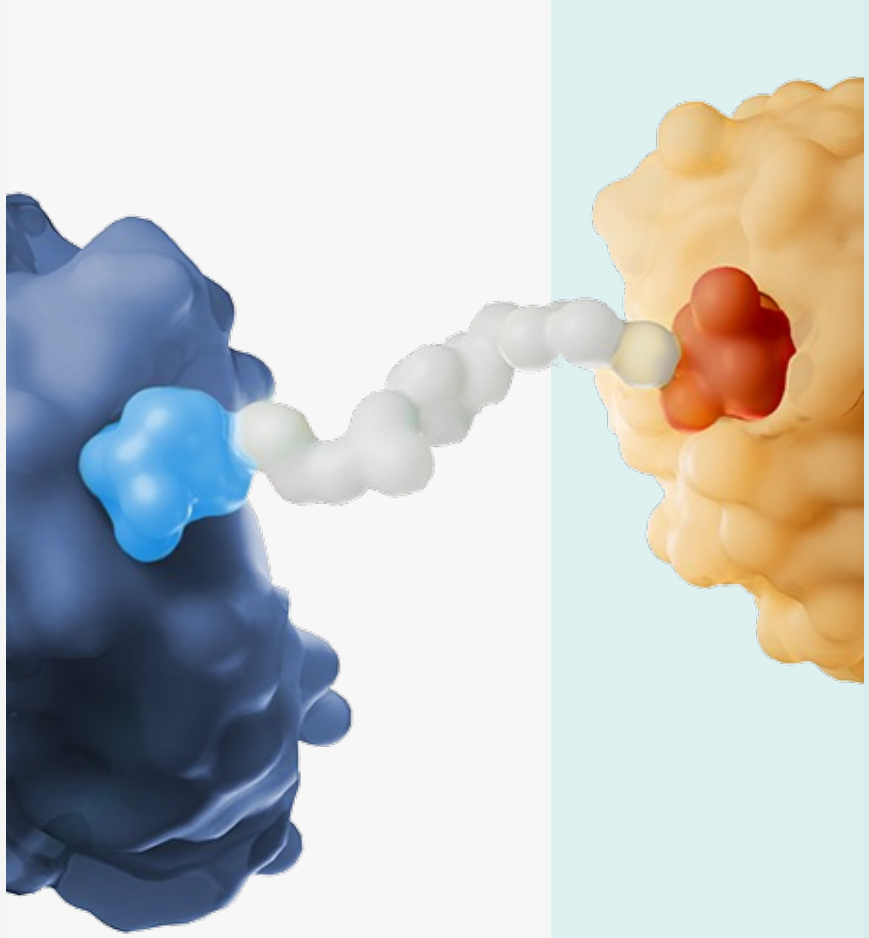

Partnered Oral Degrader Programs

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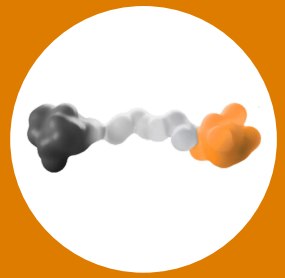
IRAK4 Overview

Combined Activity of Upstream Biologics (anti-IL-1/18/33/36) in a Pill



KT-485, a second generation IRAK4 degrader, is a highly selective, potent, **oral IRAK4 degrader**

- KT-485 has increased selectivity and potency over KT-474 including absence of any QTc signal and has been prioritized for clinical development by Sanofi
- Learnings from the clinical studies of KT-474 will be applied to accelerate development of KT-485

OPPORTUNITY

- Over **140M** potential patient impact[1]
- **>$55B** in combined global drug sales[2] opportunity
- Large potential for **oral degraders with best-in-pathway efficacy** across Th1-Th17 and Th2 Diseases

Potential to deliver the combined activity of upstream biologics in an oral drug for multiple diseases

STATUS & UPCOMING MILESTONES

- Partnered with Sanofi
- KT-485 Phase 1 SAD/MAD study in healthy volunteers and hidradenitis suppurativa patients initiated

[1]GlobalData (2023 diagnosed prevalent patient population for US/EU5/JP); [2]GlobalData (2023 sales for AD, HS, Asthma, COPD, UC, CD, RA, SLE).

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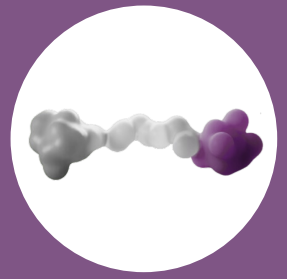
CDK2 Overview

Novel Oral Molecular Glue (MG) Degrader



KT-200 is a first-in-class, oral CDK2 molecular glue degrader

- CDK2 is a master regulator of the cell cycle with strong genetic/ clinical validation and broad oncology treatment potential
- Unlike inhibitors, CDK2 molecular glue degraders selectively degrade CDK2 while sparing closely related kinases like CDK1
- In preclinical testing, KT-200 demonstrated low-nanomolar degradation of CDK2, robust activity in CCNE1 amplified, overexpressed cell lines and in vivo tumor models, brain penetrant potential, and a favorable safety profile

OPPORTUNITY

- **~500k** potential patient impact in multiple tumor types[1]
- CDK4/6 inhibitor class ~$13B in WW revenue in 2024; projected to grow to $18B by 2030[2]

.....►

Potential to provide more precise, safe, effective, oral treatments for breast cancer and other solid tumors

STATUS & UPCOMING MILESTONES

- Partnered with Gilead to accelerate development and commercialization, with up to $750 million in total payments, including $85 million in upfront and option exercise payments
- Gilead exercised option to license KT-200, and will progress the program into IND-enabling studies to support an IND filing in 2027

[1]GlobalData (2025 diagnosed incident patient population for US/EU5/JP); [2]CDK4/6 inhibitor class includes palbociclib, abemaciclib, and ribociclib; Future revenue projection reflects GlobalData's consensus forecast.

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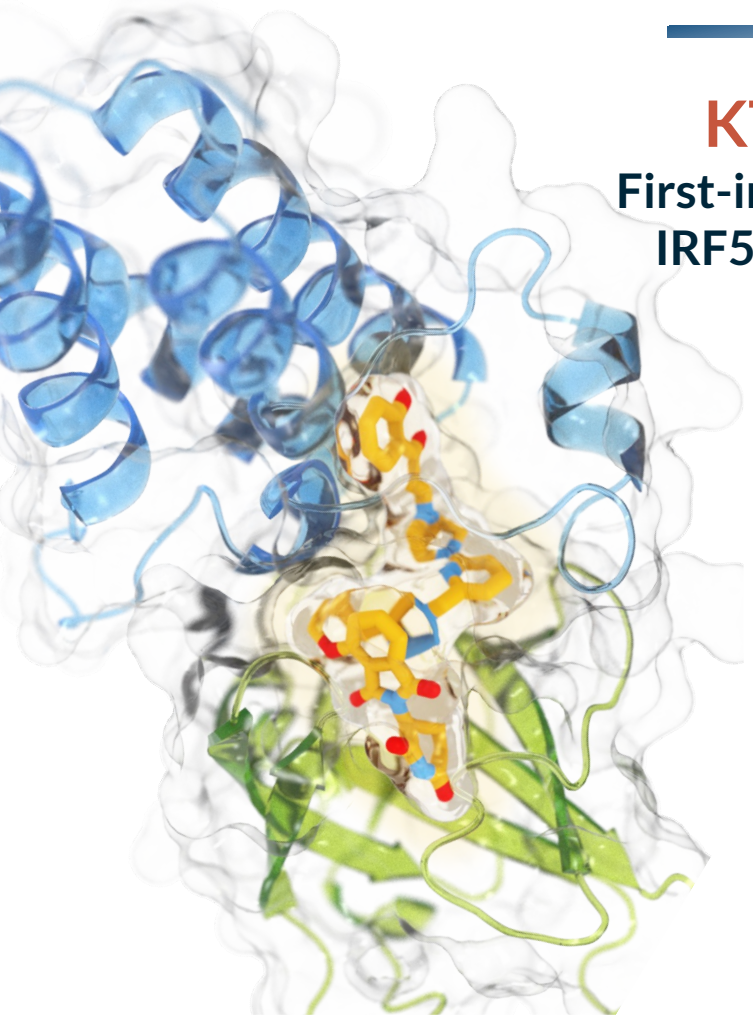
Catalysts for Growth

KT-621
First-in-Class Oral STAT6 Degrader

- Initiate Phase 3 trials in AD by mid-2027
- Advance BREADTH Phase 2b asthma study; report data in late 2027
- Announce the next indication, broadening development across Type 2 diseases



KT-579
First-in-Class Oral IRF5 Degrader

- Start LUMORA Phase 1b lupus trial by early 2027

Discovery Engine

- Advance an early pipeline of novel programs with a goal to deliver at least one new DC per year

Partners

- Sanofi advancing KT-485, oral IRAK4 degrader, in Phase 1
- Gilead advancing KT-200, oral CDK2 molecular glue degrader, toward a 2027 IND filing

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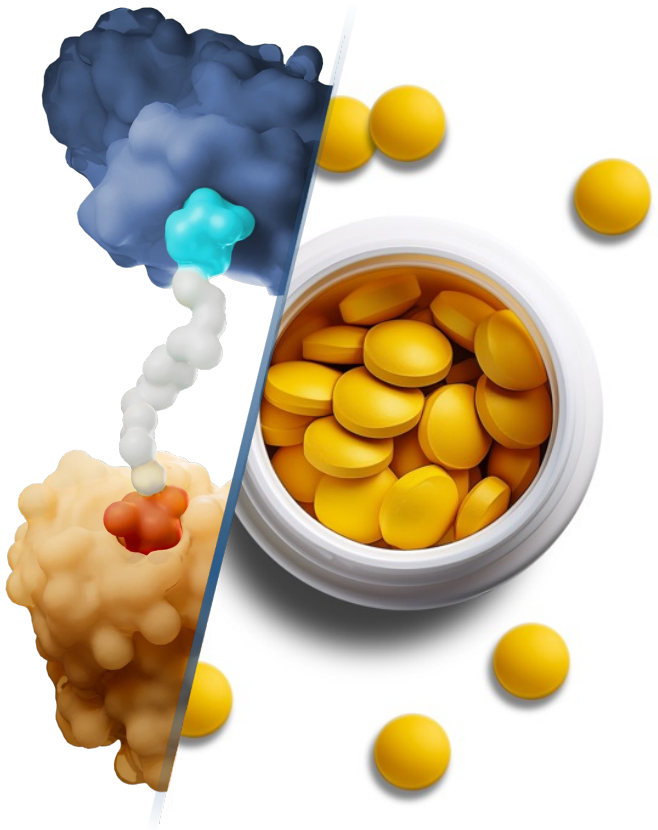
Turning Scientific Innovation into New Treatment Possibilities



Addressing historically undrugged targets

Combine disciplined target selection with deep expertise in ligand discovery and small molecule design to expand the reach of oral medicines

Advancing an innovative pipeline with transformative potential

KT-621 and KT-579 lead a growing portfolio targeting complementary immunoregulatory mechanisms, with the potential to build a broad immunology franchise

Established clinical validation of TPD in immunology

KT-621 BROADEN2 results reinforce the potential of oral degraders to deliver differentiated profiles that could overcome conventional treatment trade-offs

Expanding treatment options for patients

Potentially safe, effective oral medicines could broaden advanced therapy use and reach more patients who remain untreated or inadequately controlled

Kymera is working to redefine the standard of care in immunology and
deliver novel oral therapies to patients around the world

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Thank You

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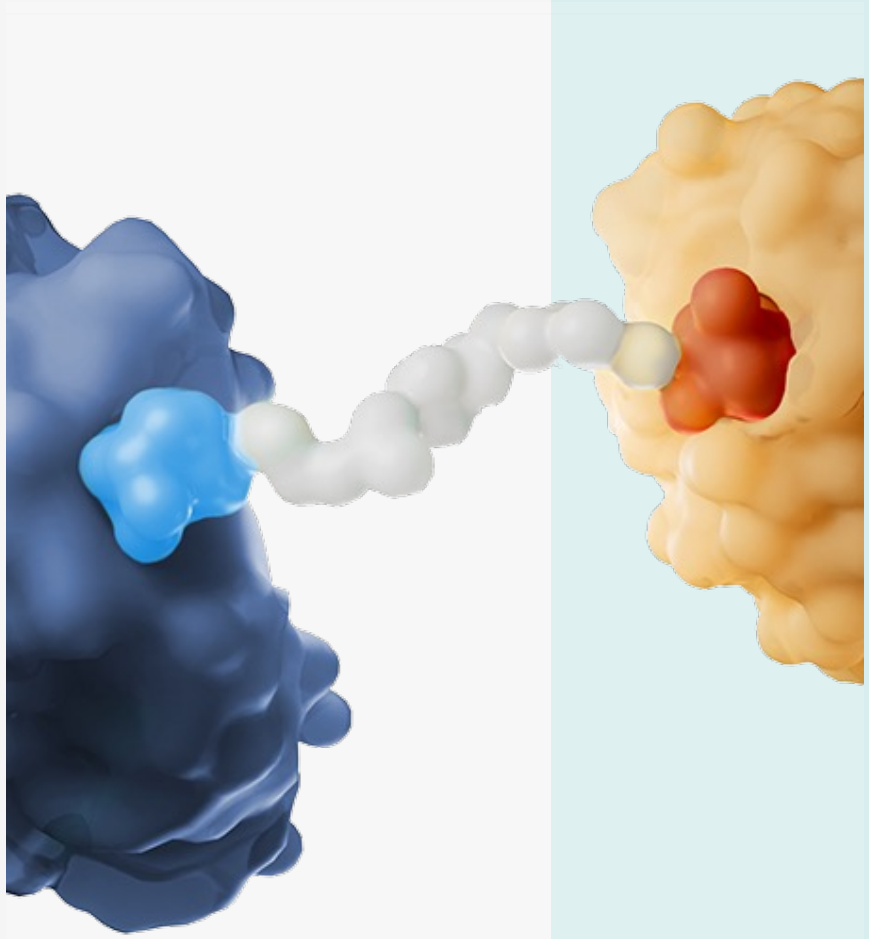

Appendix: First-in-Class Oral STAT6 Degrader Program

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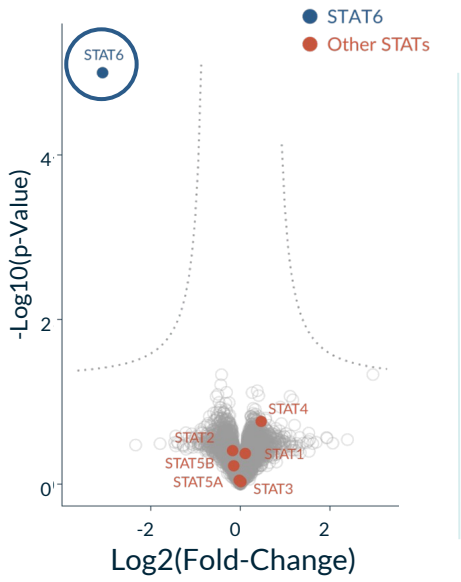
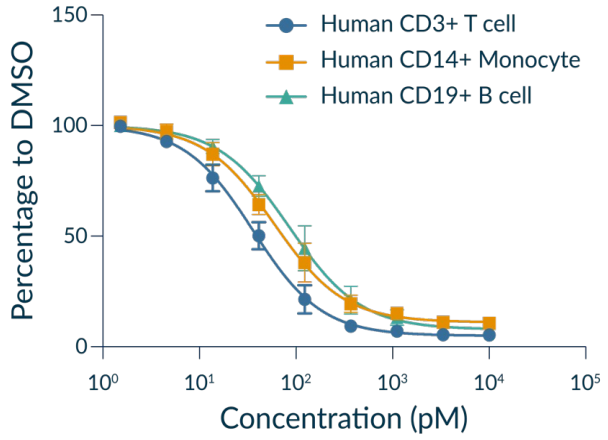
KT-621: Compelling Preclinical Profile

Exquisite Selectivity



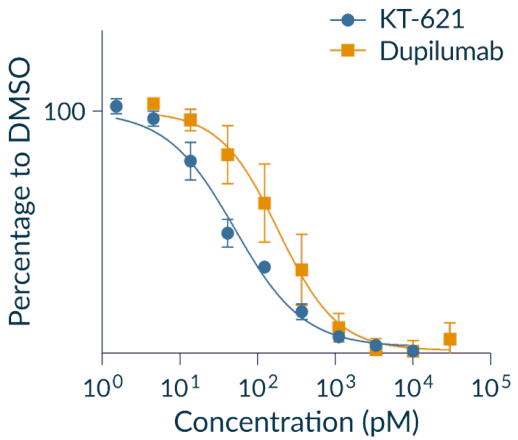
Robust Degradation in Relevant Cell Types

STAT6 Degradation in Immune Cells



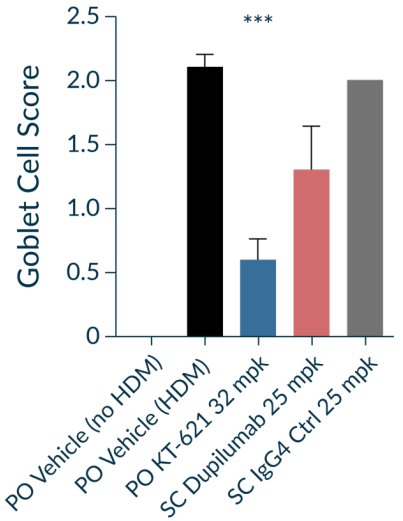
Full Inhibition of IL-4/IL-13 Pathways, More Potent than Dupilumab

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]



[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. For more information on KT-621's preclinical profile, visit Kymera's website.

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KT-621 Data Provide Validation and Derisk Future Clinical Trials

Preclinical

POTENCY

- <100 pM DC90 in all relevant human cell types

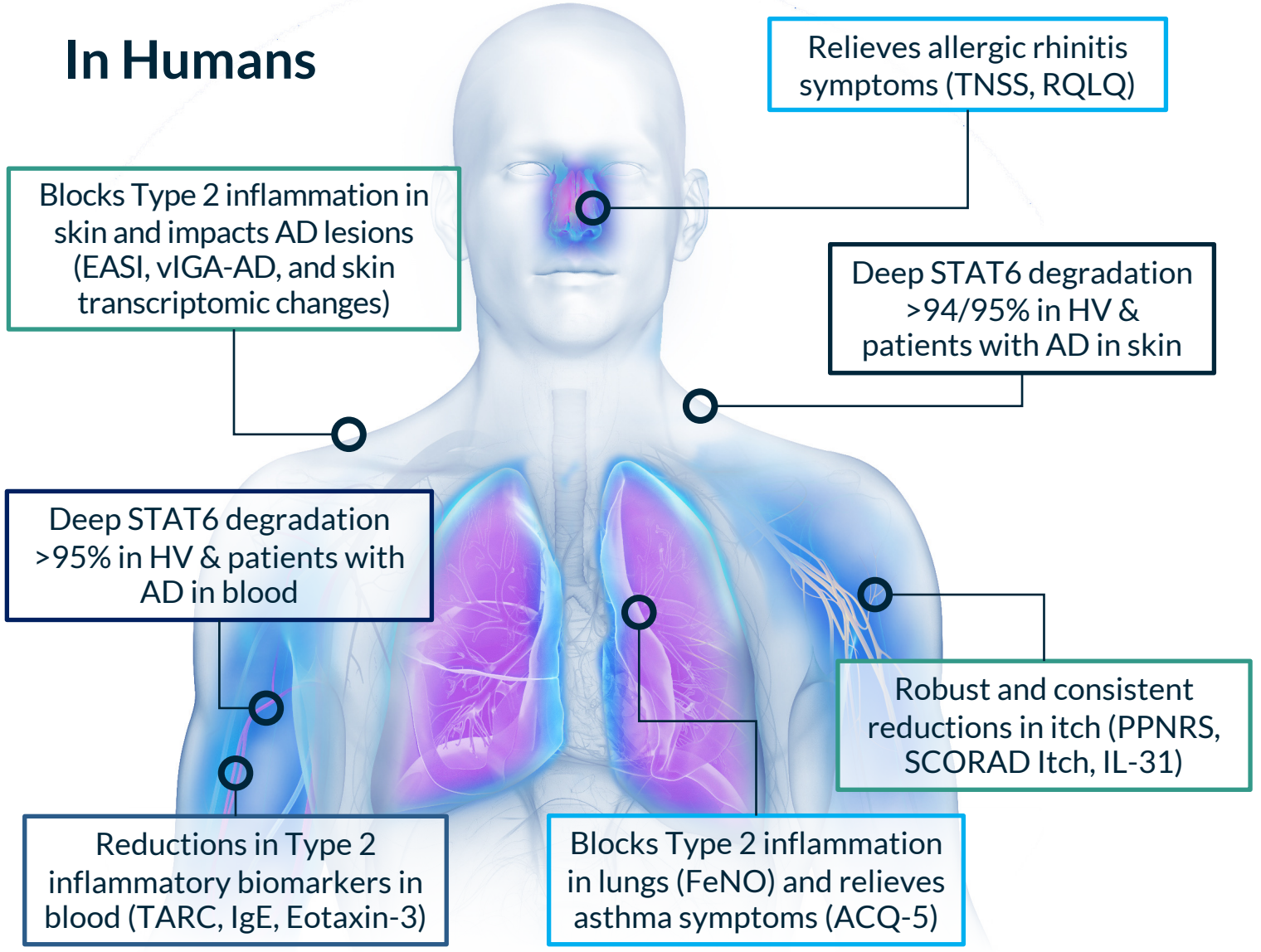
EFFICACY

- Blocked IL-4 and IL-13 signaling in human cells and *in vivo* systems equally or more potently than dupilumab
- Robust activity in asthma and AD in mouse models

SAFETY

- No AEs at any doses in:
  - 4 weeks rat and NHP GLP tox
  - 4 months rat and NHP GLP tox
  - 6/9 months rat and NHP GLP chronic tox
  - Embryofetal development tox in rat, rabbit and NHP

In Humans



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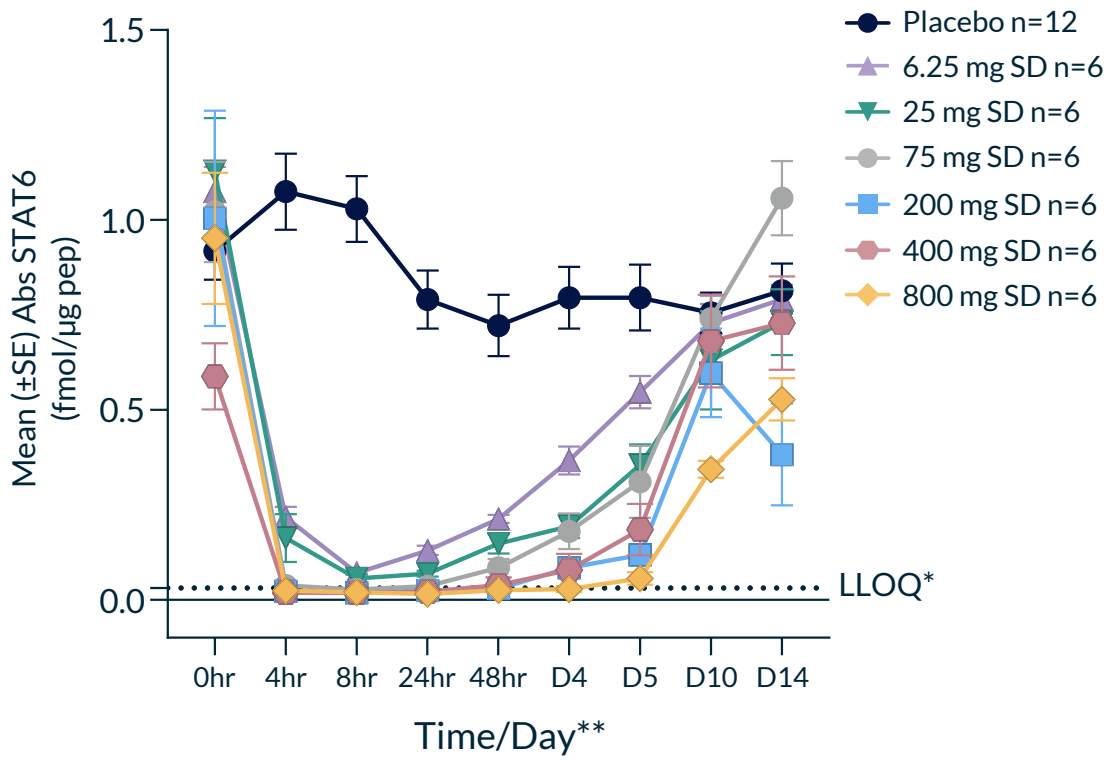
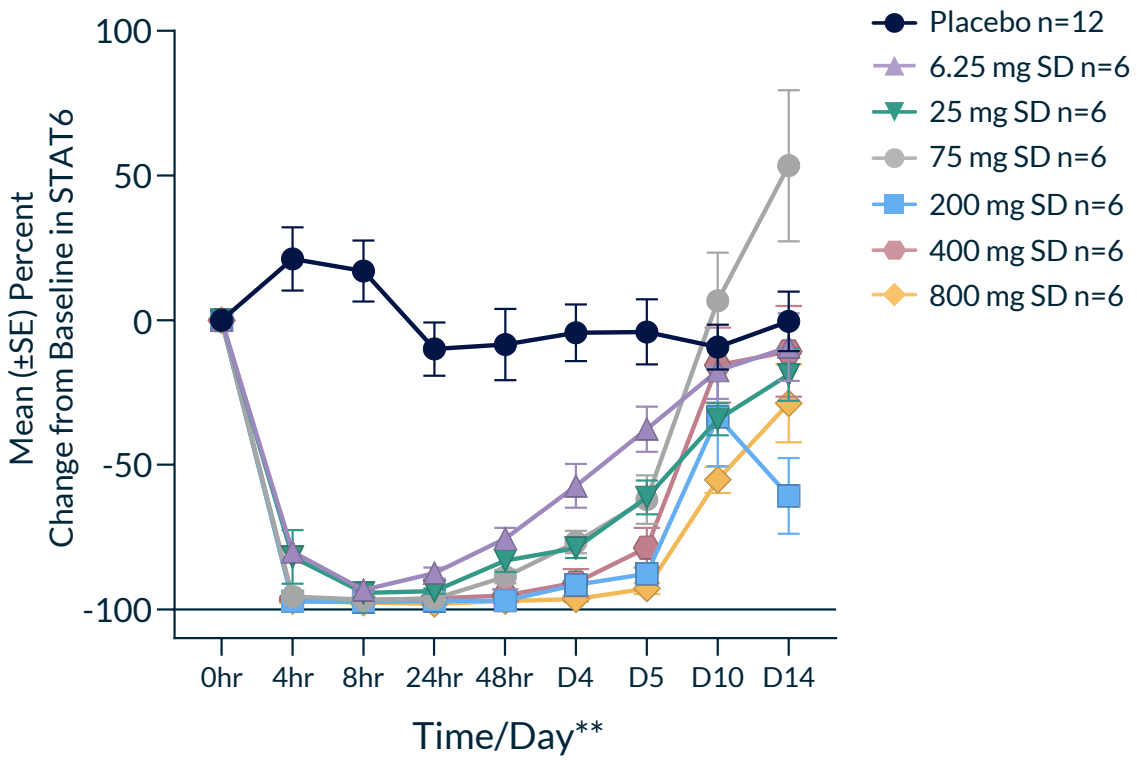
Single Doses of KT-621 Achieved Rapid, Deep and Prolonged STAT6 Degradation in Blood

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; For more information on KT-621's Phase 1 healthy volunteer clinical trial, visit Kymera's website.

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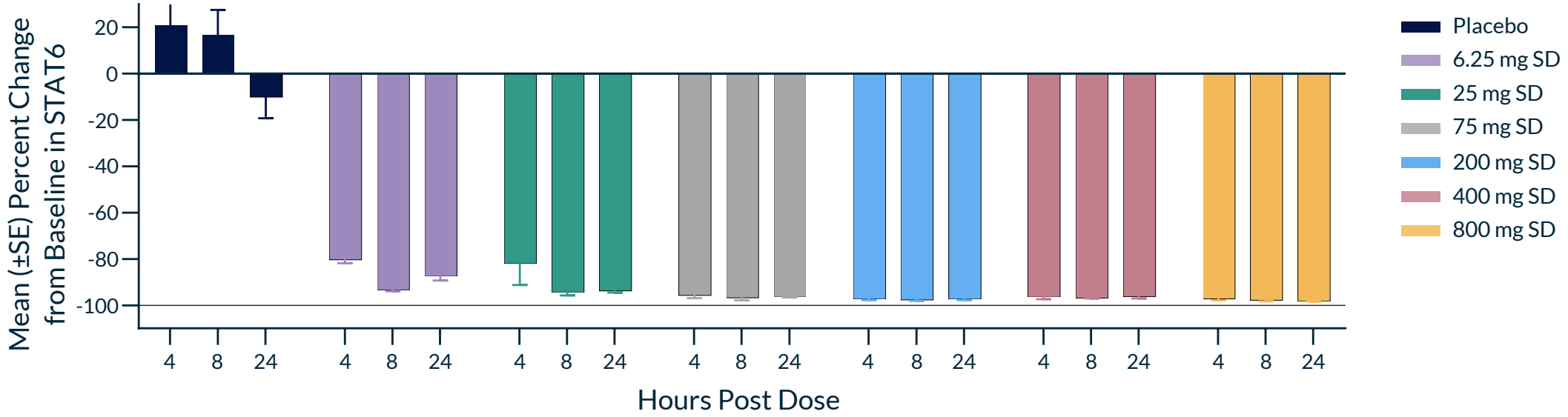
Single Dose Administration of KT-621 Achieved >90% STAT6 Degradation in Blood Across All Dose Levels

>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; For more information on KT-621's Phase 1 healthy volunteer clinical trial, visit Kymera's website.

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KT-621 Phase 1a Healthy Volunteer Trial: Safety Summary

Well Tolerated Across All Doses Evaluated and Safety Profile Undifferentiated from Placebo

- No Serious Adverse Events
- No Severe Adverse Events
- No dose dependent pattern in Treatment Emergent Adverse Events (TEAEs)
- No Treatment Related AE (TRAE) 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 Cohorts

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

TRAEs by Preferred Term: MAD Cohorts

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

For more information on KT-621's Phase 1 Healthy Volunteer trial, visit clinicaltrials.gov (NCT06673667); ECG: electrocardiogram.

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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

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)

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.

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KT-621 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)

For more information on KT-621's BroADen Phase 1b atopic dermatitis trial, visit clinicaltrials.gov (NCT06945458); BMI: Body Mass Index; SD: Standard Deviation.

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KT-621 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; For more information on KT-621's BroADen Phase 1b atopic dermatitis trial, visit clinicaltrials.gov (NCT06945458); 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.

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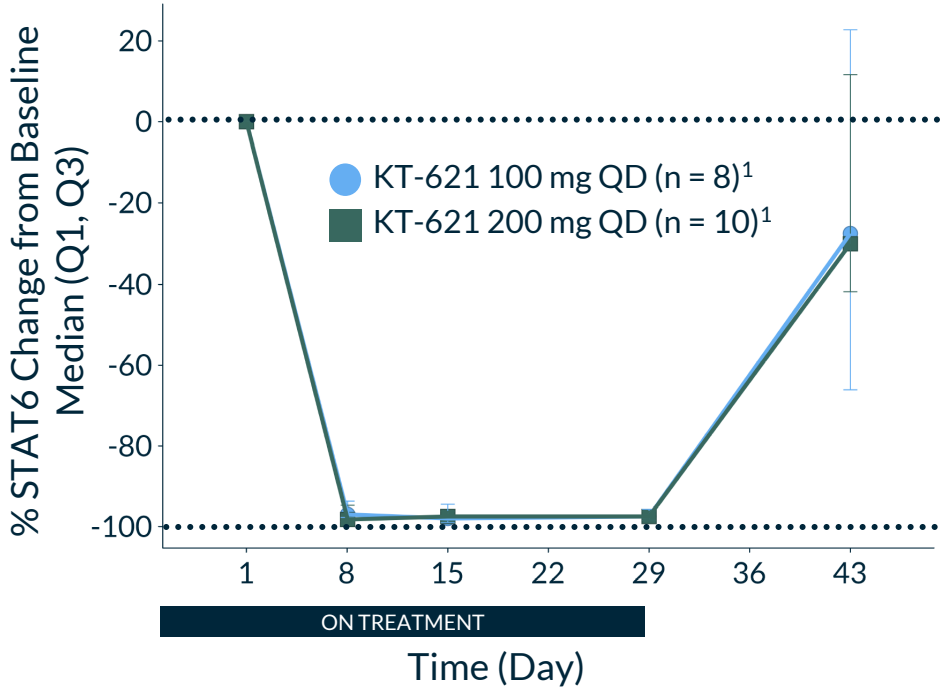
KT-621 Achieved Deep STAT6 Degradation in Blood and Skin

Degradation Maintained for 28 Days Across Both Dose Cohorts

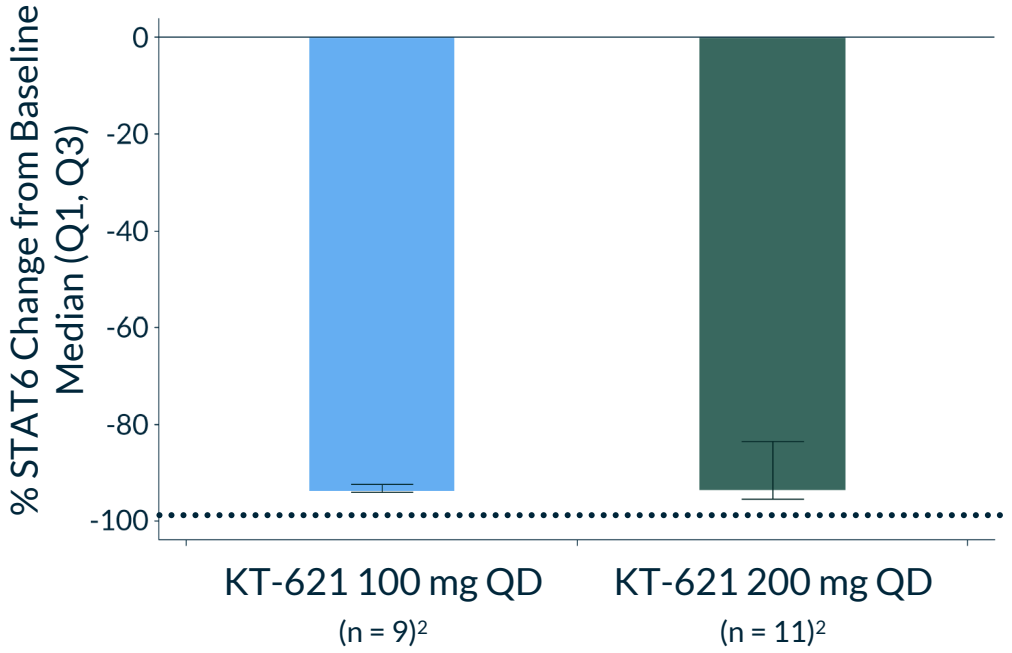
Median STAT6 Degradation in Blood

Method: Flow Cytometry



Median STAT6 Degradation in Skin

Method: Targeted Mass Spec



- Median STAT6 degradation of 98% in blood in both dose groups maintained throughout the treatment period
- Deep skin degradation of 94% in both dose groups with multiple patients' STAT6 levels below the LLOQ (lower limit of quantification)

Note: N values reflect the number of participants with available samples at Day 29; [1]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; [2]Two patients did not consent to D29 biopsies; PK: Pharmacokinetics.

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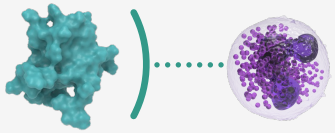
Robust Impact on All Disease-Relevant Biomarkers of Type 2 Inflammation in BroADen Phase 1b AD Trial

	TARC (CCL17)	Eotaxin-3 (CCL26)	IgE	IL-31	FeNO
					
	Validated biomarker of Type 2 inflammation suppression in patients Drives chemotaxis of CCR4-expressed T cells to inflammatory sites	Highly specific downstream cytokine of the IL-4/13 pathway Drives chemotaxis of CCR3-expressed inflammatory cells to inflamed sites	IL-4 promotes B-cell class switching, amplifying IgE production IgE activates mast cells and basophils to release Type 2 cytokines	Key pruritogenic cytokine produced by activated Type 2 cells[1] Signals through the IL-31RA/OSMR complex on neurons, keratinocytes, and immune cells, linking immune activation to itch	Marker of Type 2 airway inflammation in asthma[4] FeNO reflects airway epithelial iNOS activity driven by IL-4/13 signaling Historically not measured in AD patients
Median % Inhibition at Day 29					
KT-621	**74%[5]**	**73%**	**14%**	**54%**	**33%**
Dupilumab[6]	74%	51% (in Asthma)	~15%	No change[2,3]	Not measured

[1]Raap et al, *Journal of Allergy and Clinical Immunology*, 2008; [2]Kishi et al, *Cells*, 2023; [3]Suleiman et al, *Acta Derm Venereol*, 2026; [4]Chung et al, *Lancet*, 2021; [5]Represents TARC reduction in patients with elevated baseline TARC levels, defined as the lower bound of the 95% confidence interval for median baseline TARC levels from the dupilumab SOLO1-2 AD studies; [6]Hamilton et al, *Clinical & Experimental Allergy*, 2021.

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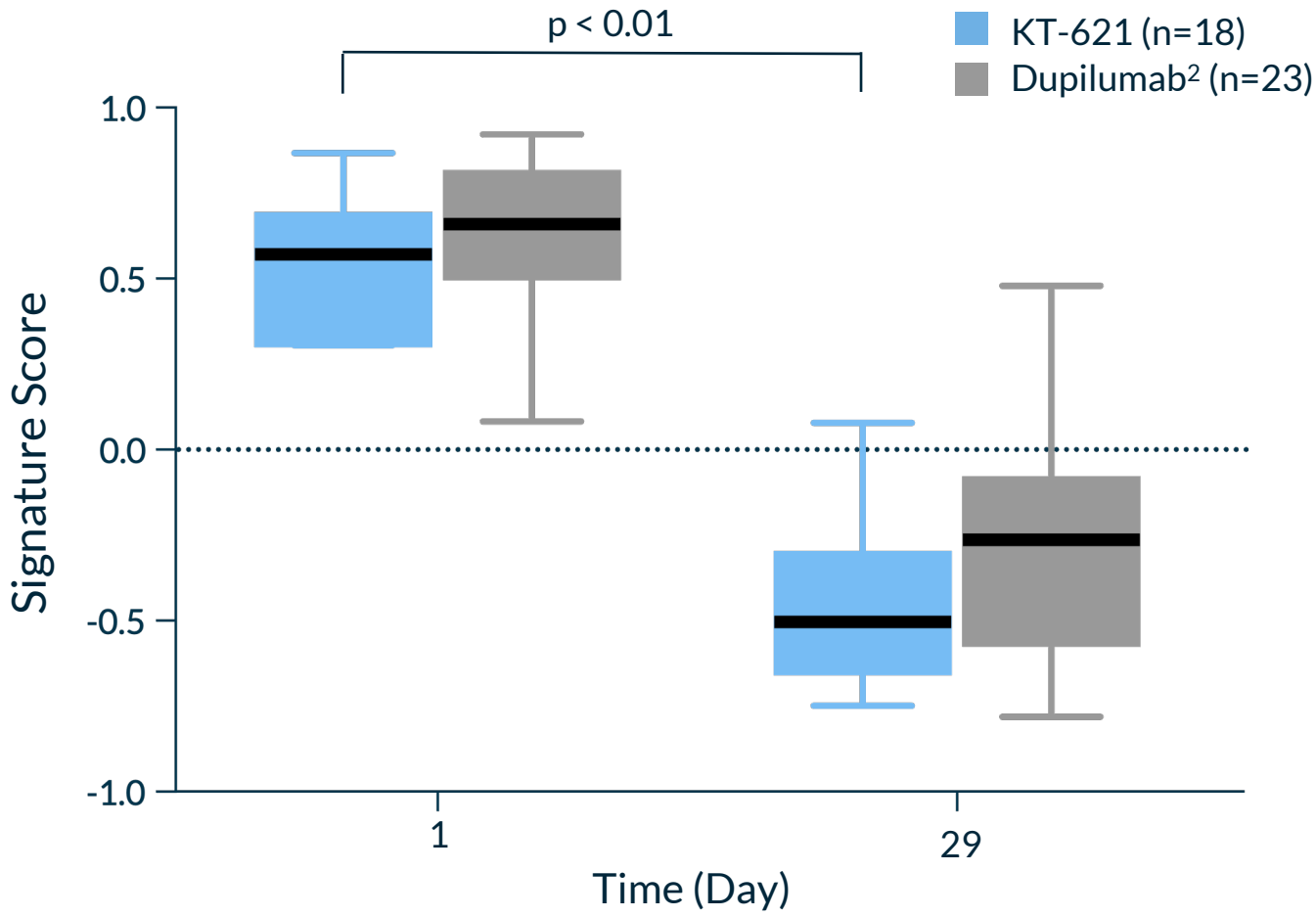
KT-621 Significantly Downregulated Core Type 2 Inflammation Gene Set in Skin Lesions of AD Patients

- **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.

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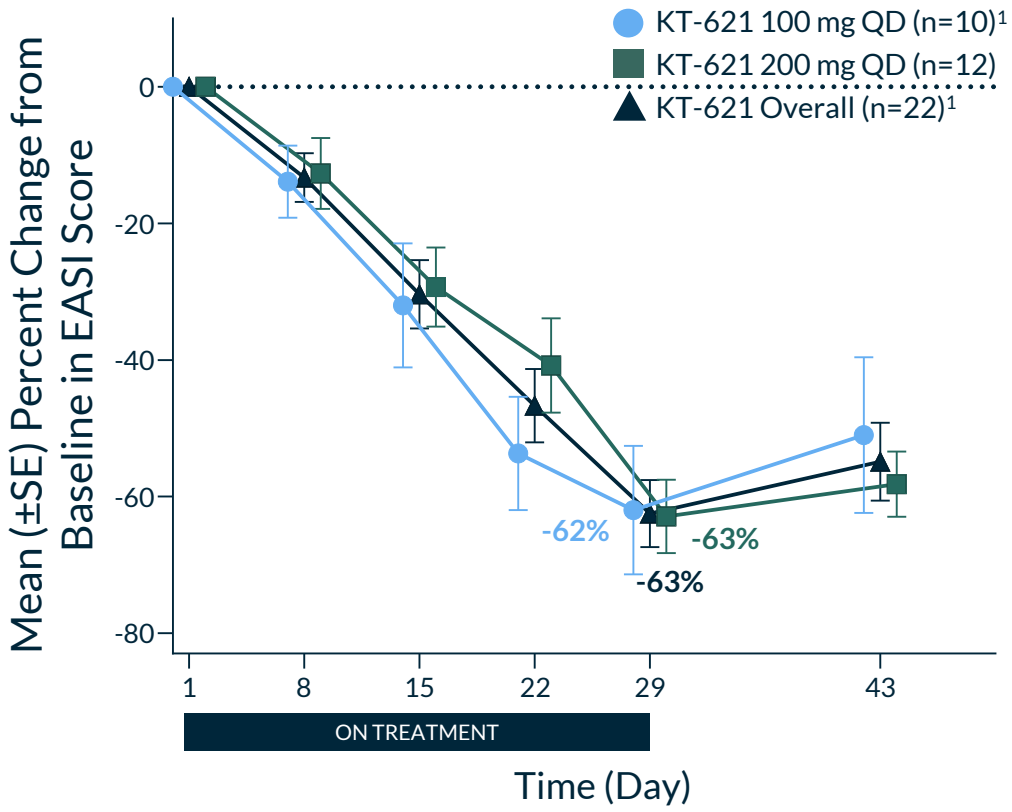
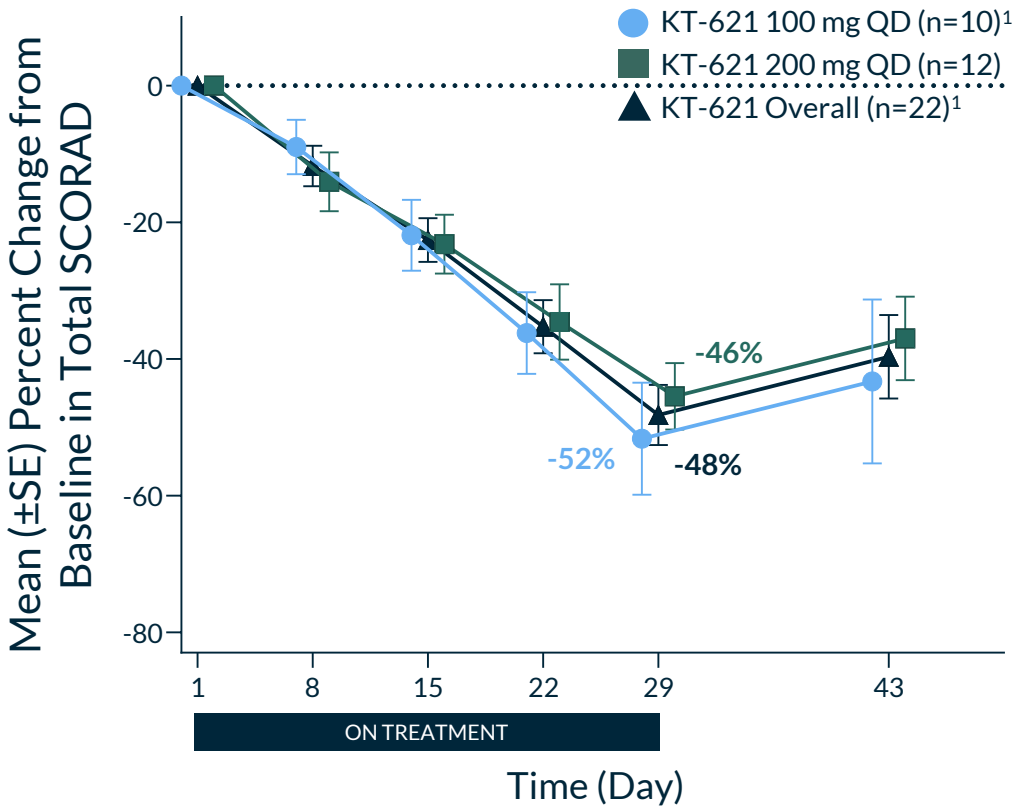
KT-621 Achieved Rapid and Robust Reductions in EASI and SCORAD Across All Patients

Mean % Change from Baseline in EASI



Mean % Change in Total SCORAD



- Reductions seen as early as Day 8 without apparent plateau during treatment duration
- Achieved robust clinical improvement across EASI-50 (76% overall), EASI-75 (29% overall) and vIGA-AD (19% overall)

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; SCORAD: SCORing Atopic Dermatitis.

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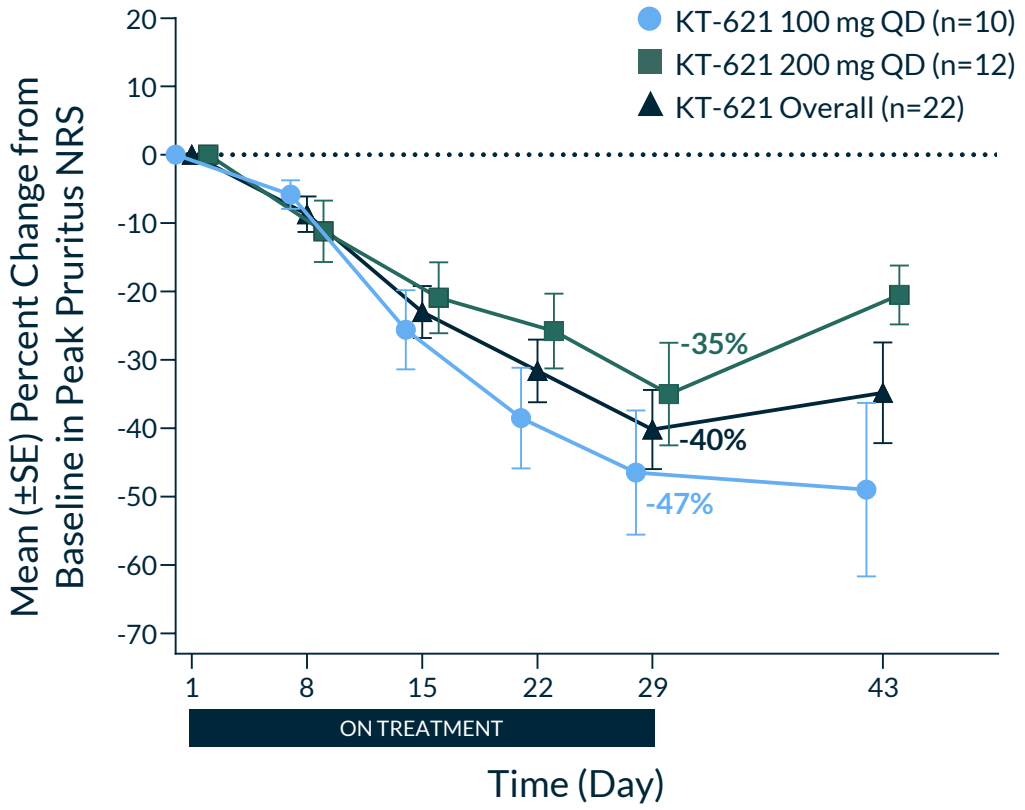
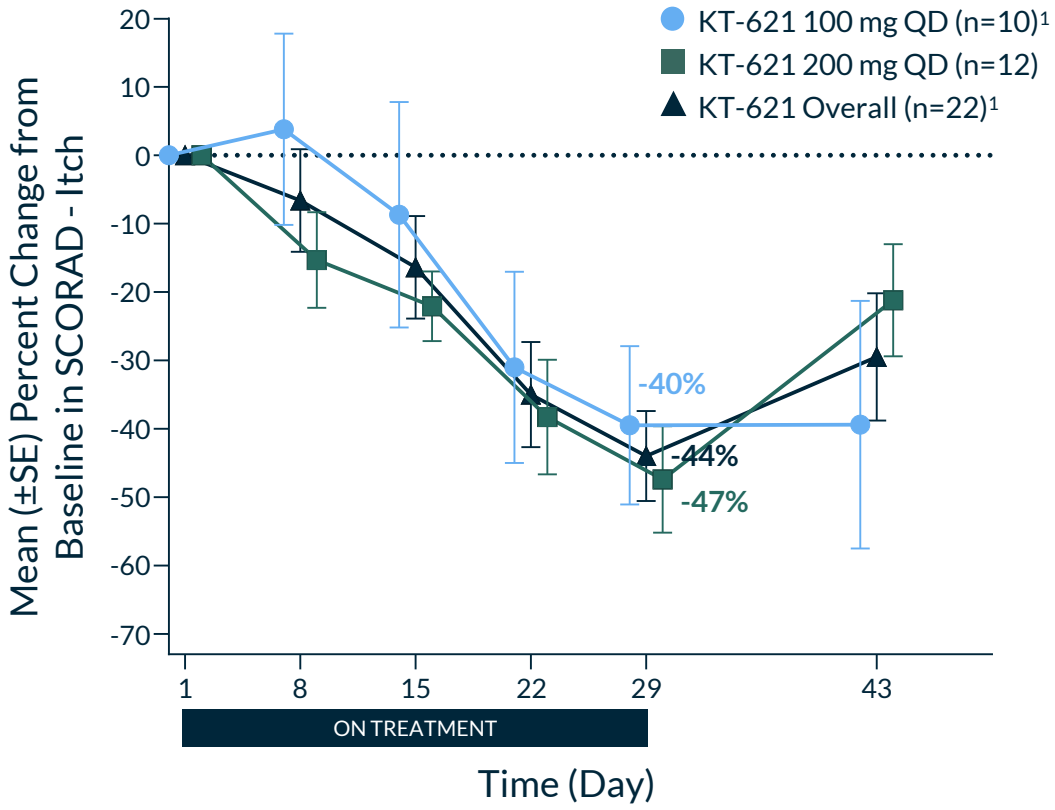
KT-621 Achieved Robust and Consistent Reductions in Itch Across Independent Clinical Measures

Mean % Change in Peak Pruritus NRS



Mean % Change in SCORAD Itch



- KT-621 achieved rapid and robust mean Peak Pruritus NRS and SCORAD-Itch reduction without apparent plateau during treatment duration

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.

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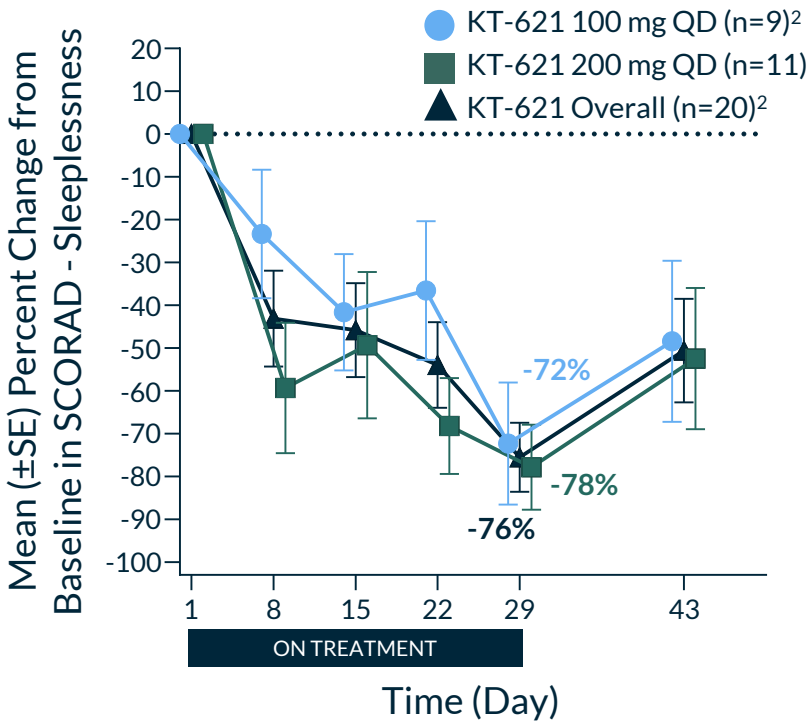
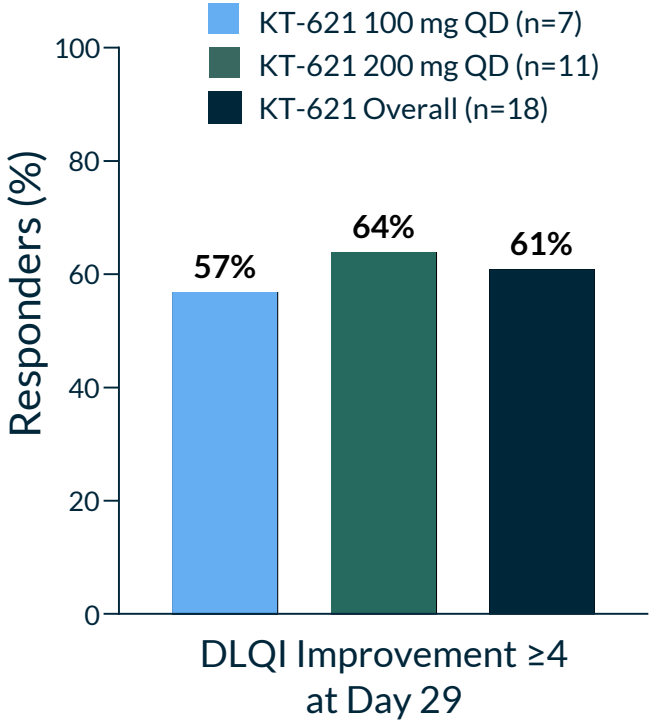
KT-621 Achieved Robust Improvement in Patient Reported Outcomes and Quality of Life Measures

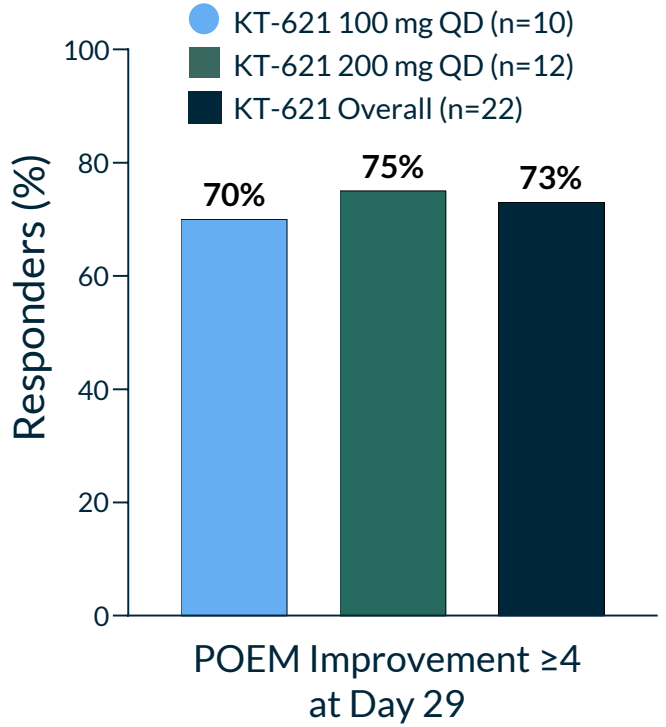
Mean % Change in SCORAD - Sleeplessness[1]



DLQI Responders (Improvement ≥ 4 points)[3]



POEM Responders (Improvement ≥ 4 Points)



- KT-621 achieved rapid and robust mean SCORAD-Sleeplessness reduction in both dose cohorts
- POEM and DLQI are patient-reported measures evaluating severity, experience, and quality of life; demonstrated improvements are greater than the minimum clinically important difference (MCID)

Note: Analysis based on observed cases at each visit. [1]Only patients with non-zero baseline sleeplessness are included; [2]One patient in the 100 mg cohort missed the D29 visit (n=8 for 100 mg and n=19 for Overall at D29); [3]Only patients with baseline DLQI score ≥ 4 included; SCORAD: SCORing Atopic Dermatitis. DLQI: Dermatology Life Quality Index; POEM: Patient-Oriented Eczema Measure.

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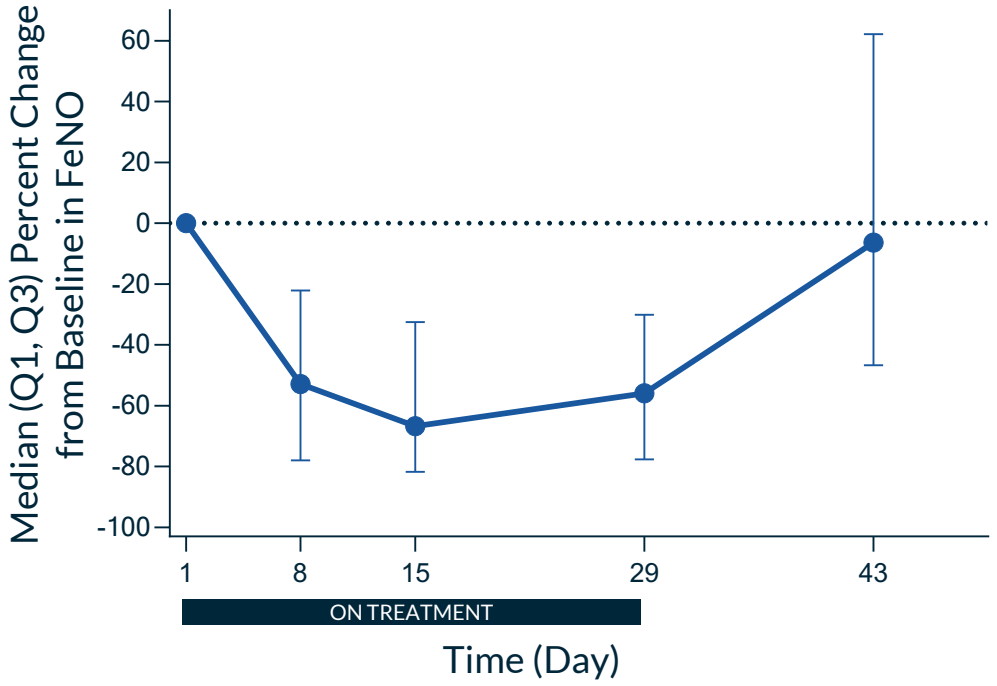
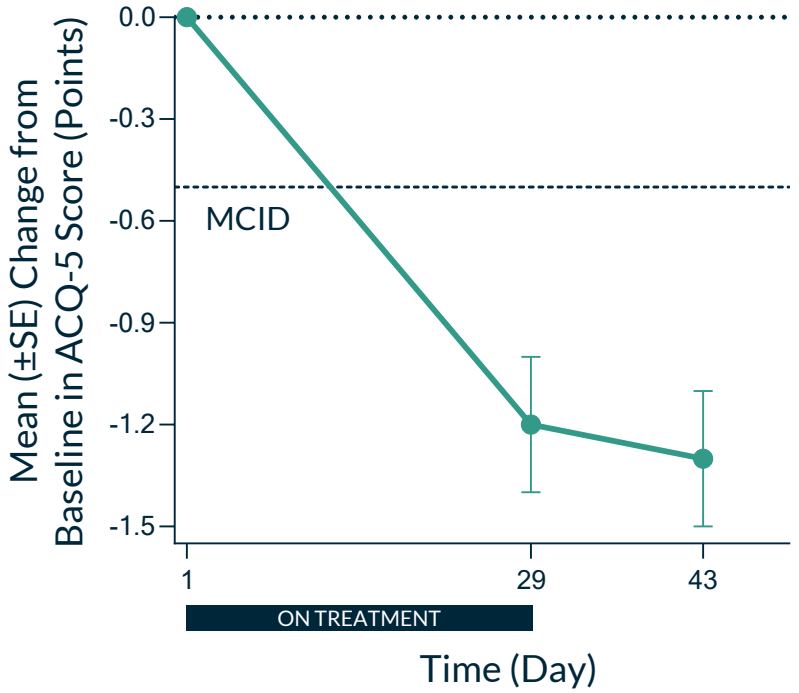
KT-621 Achieved Robust Impact on FeNO and ACQ-5 in AD Patients with Comorbid Asthma

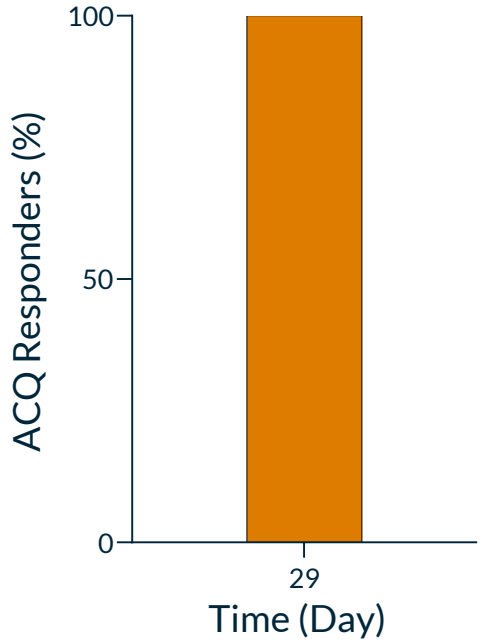
Median % Change from Baseline in FeNO (n=4)



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.

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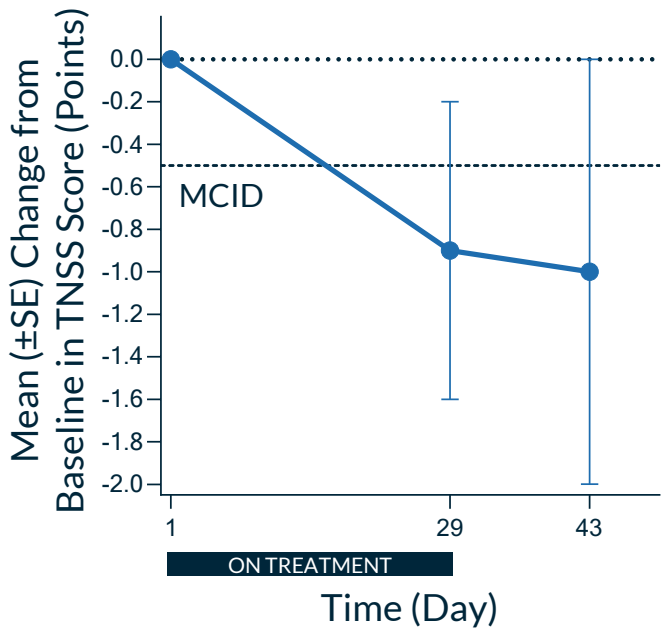
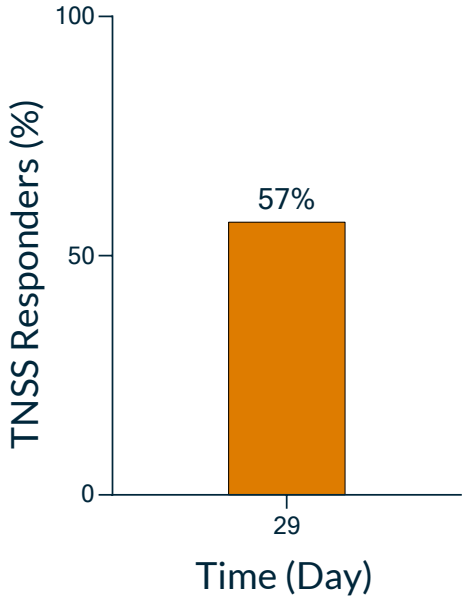
KT-621 Achieved Robust Impact on TNSS and RQLQ in AD Patients with Comorbid Allergic Rhinitis

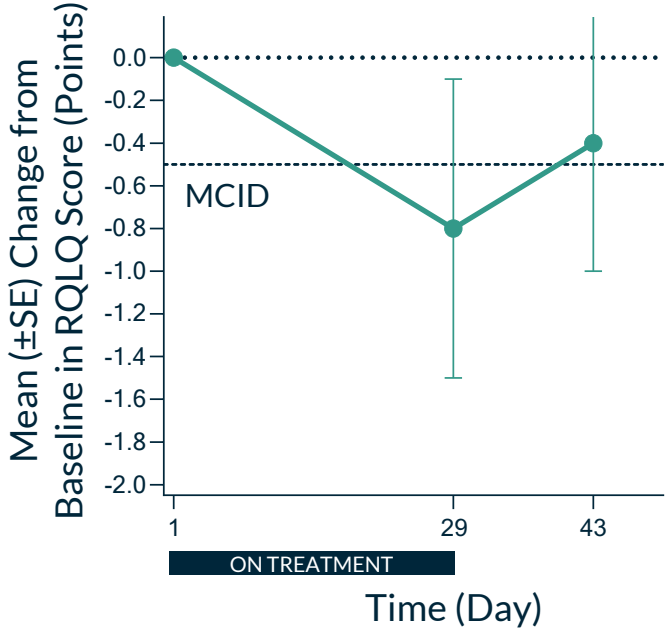
Mean Point Change in TNSS (n=7)[1]



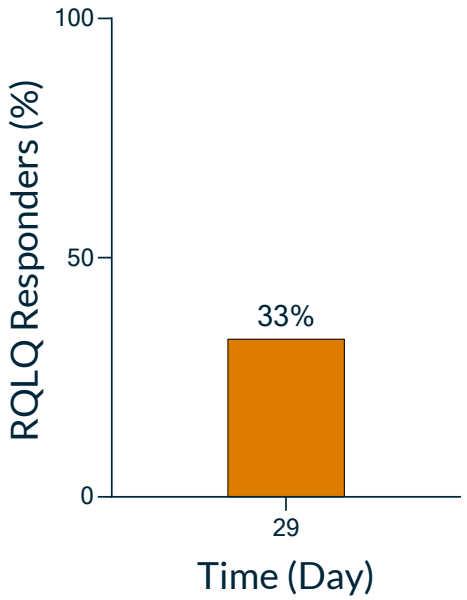
TNSS Responders (Improvement ≥ 0.55, n=7)[1]



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.

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KT-621 BroADDen 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.

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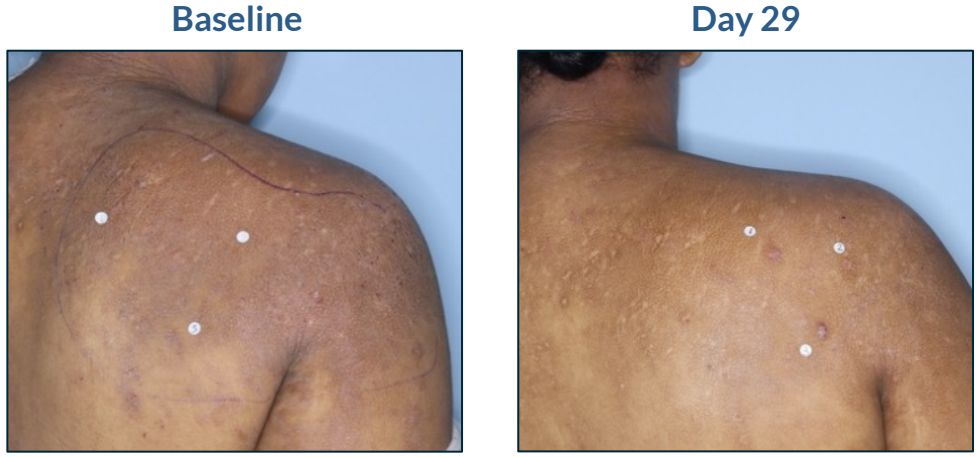
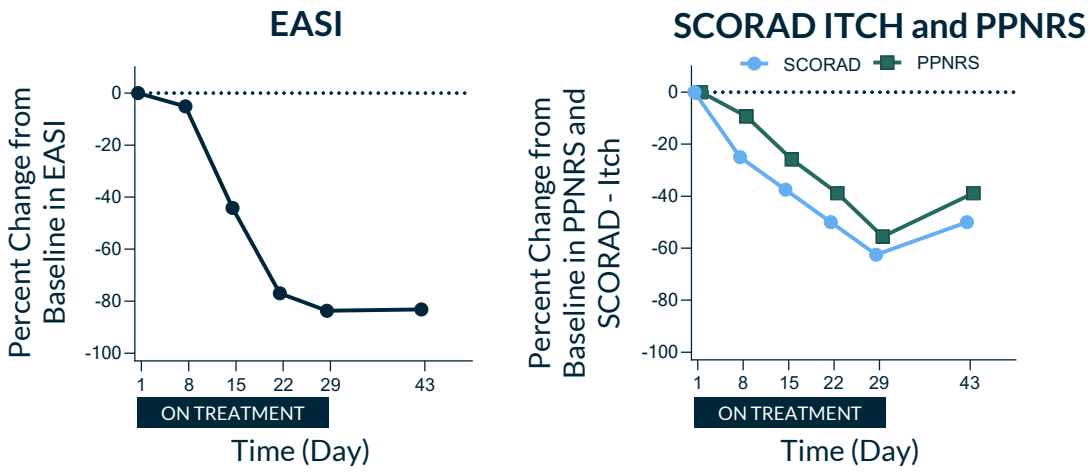
Example of Clinical and Biomarker Response to KT-621 in BroADen Patient with Severe AD and Prior Dupilumab Treatment

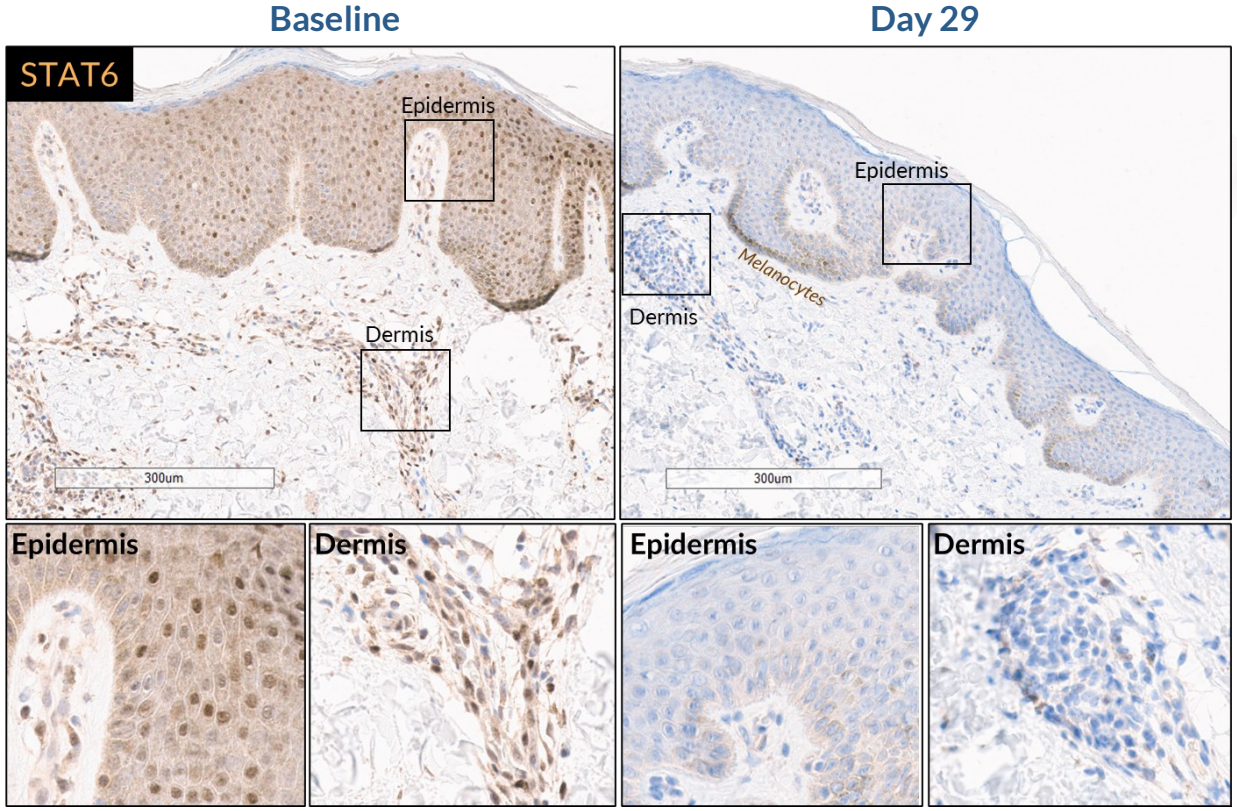
Pre and Day 29 Photos for EASI-75 Responder



% Change in EASI and Itch[1]



STAT6 Staining IHC



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; For more information on KT-621's BroADen Phase 1b atopic dermatitis trial, visit clinicaltrials.gov (NCT06945458); EASI: Eczema Area and Severity Index; SCORAD: SCORing Atopic Dermatitis; PPNRS: Peak Pruritus Numerical Rating Scale; IHC: Immunohistochemistry.

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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,** disrupting daily activities
- Common comorbidities include **asthma**
- Many options only address symptoms, **not underlying Type 2 inflammation**
- Estimated **~40 million** adults in US/EU5/JP[1]
- **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 (2026 diagnosed prevalent patient population for AD in US/EU5/JP); [2]GlobalData.

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Asthma: Chronic, Inflammatory Lung Disease

Opportunity Exists to Transform the Treatment Paradigm

- The airways in lungs to become swollen and inflamed, causing coughing, shortness of breath, chest tightness or pain, and **making it harder to breathe**
- **Symptoms may resemble other lung conditions,** called asthma mimics, and can be misdiagnosed as a result
- Can significantly impact a **patient's quality of life** and lead to permanent lung damage
- Many options only address symptoms, **not underlying Type 2 inflammation**
- Estimated **55 million** adults in US/EU5/JP[1]
- **Less than 1 million** patients currently on dupilumab[2]



Participant expressed hope "for future treatments that are easier to use…and better routes of administration than inhalers or injections due to fear of needles."

– Little Airways, Big Voices[3]
Patient Focused Drug Development: Asthma

[1]Diagnosed prevalent patient population for US/EU5/JP (GlobalData; 2023); [2]GlobalData; [3]Little Airways, Big Voices, Patient Focused Drug Development, 2021.

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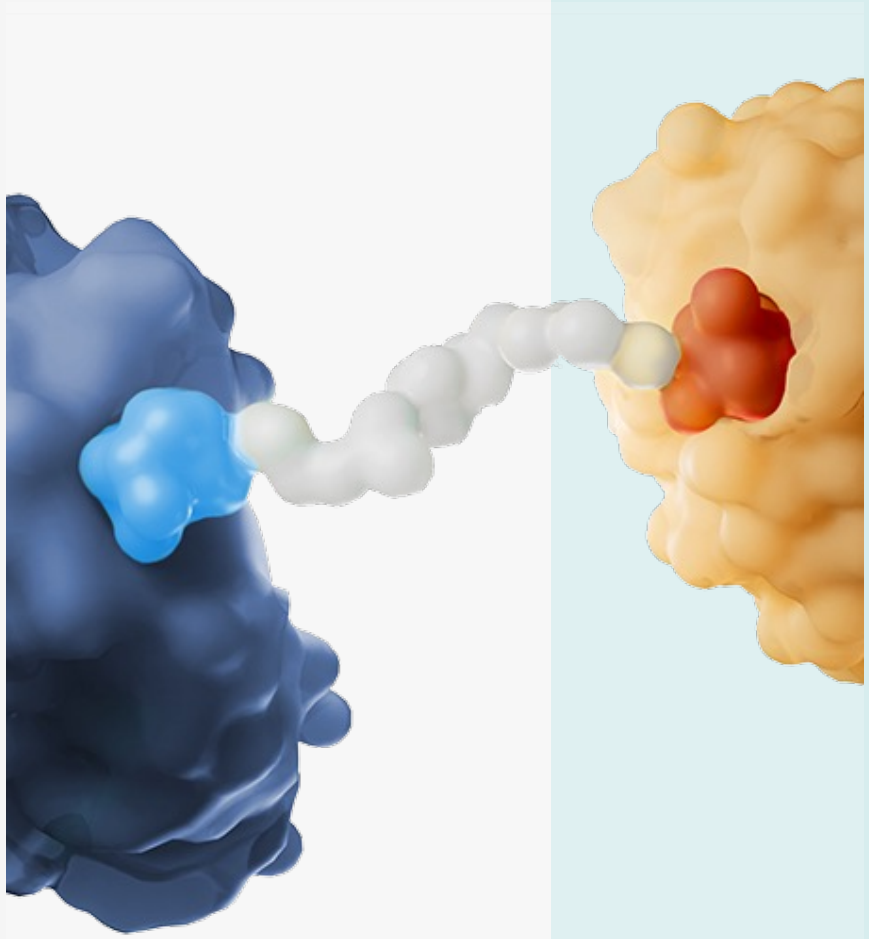

Appendix: First-in-Class Oral IRF5 Degrader Program

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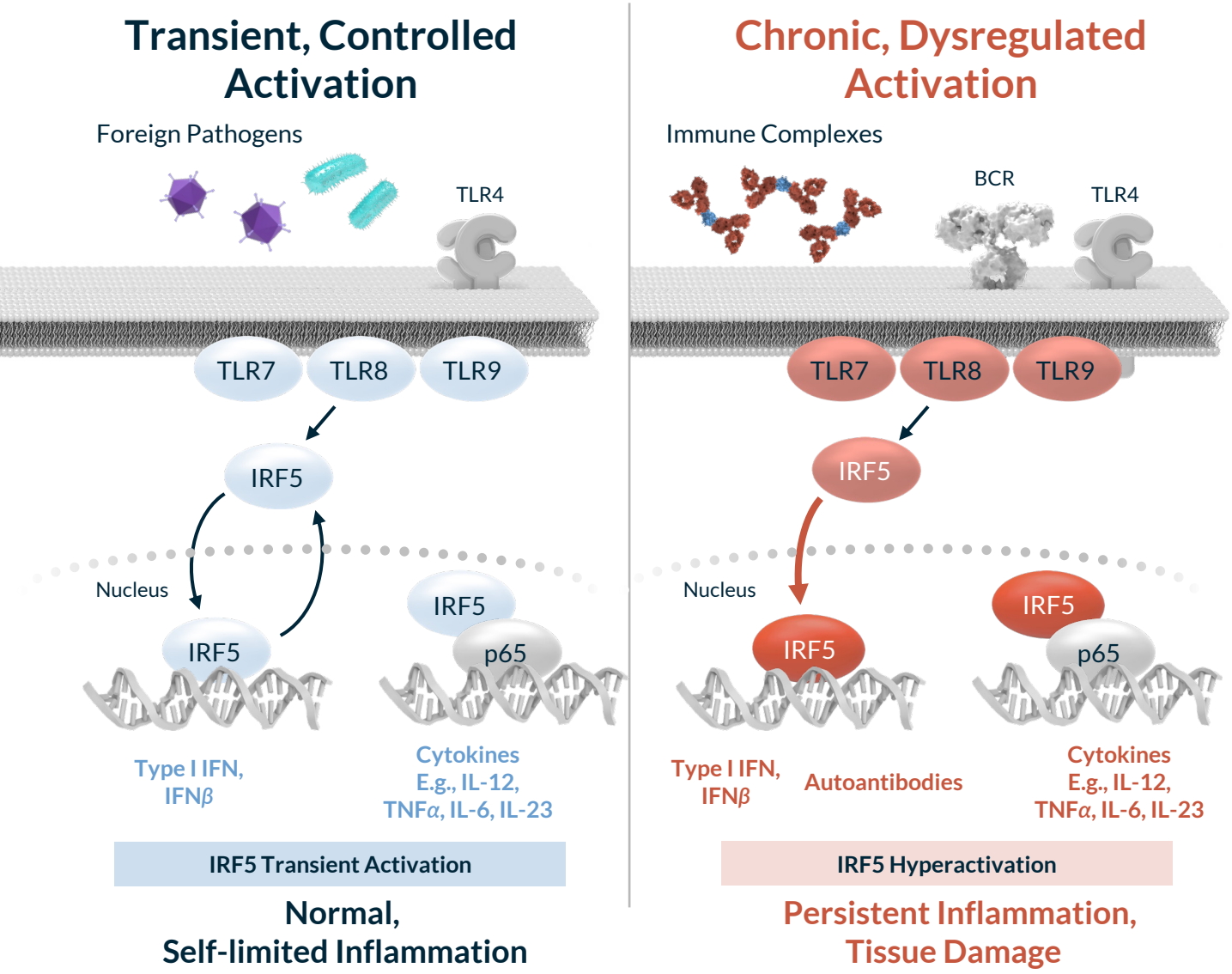
Central Driver of Immune Dysregulation in Autoimmune Diseases



IRF5 Expression, Activation and Function

- IRF5 is **predominantly expressed and activated** in immune cell types: Dendritic Cells (DCs), Monocytes, Macrophages, and B cells
- In normal controlled immune responses, IRF5 is transiently activated by pattern recognition receptors (PRRs) with quick inflammation resolution
- In **autoimmune** diseases, **chronic activation** locks IRF5 into a hyperactive state leading to **amplified** and **skewed** upregulation of **pro-inflammatory and disease-defining pathways**
- IRF5 risk variants and functional hyperactivation associate with increased signaling in multiple clinically validated pathways **(Type I IFN, Cytokines, Autoantibodies)**

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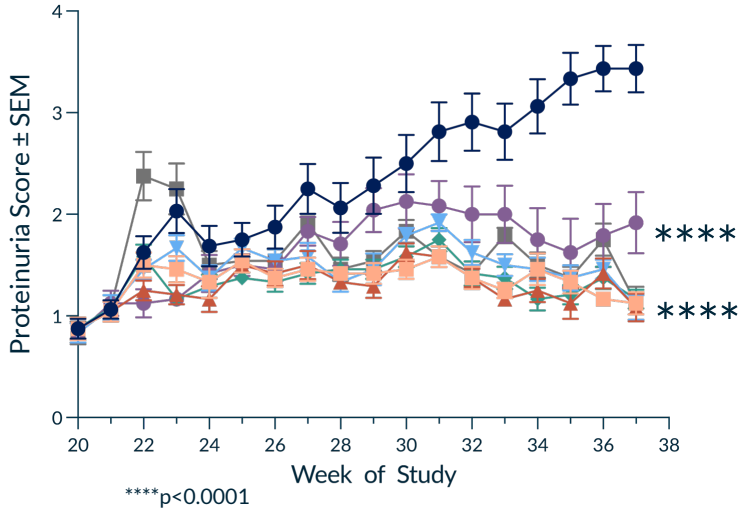
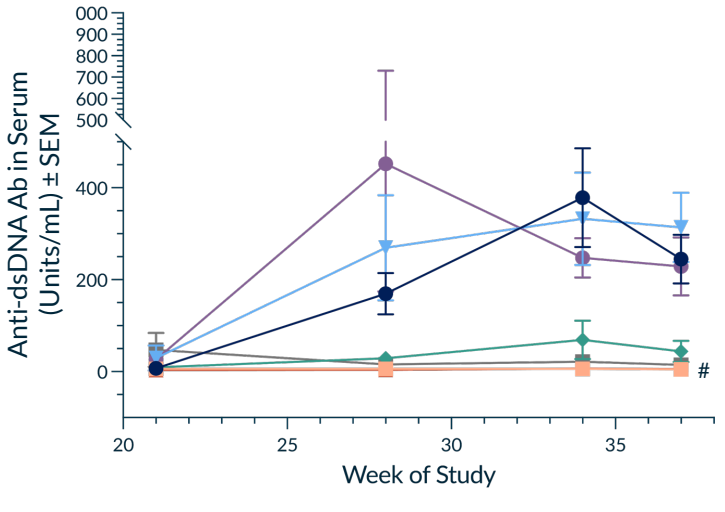
IRF5 Degradation Demonstrated Generally Better Outcomes than Approved or Clinically Active Drugs in NZBW1 Spontaneous[1] Mouse Lupus Model

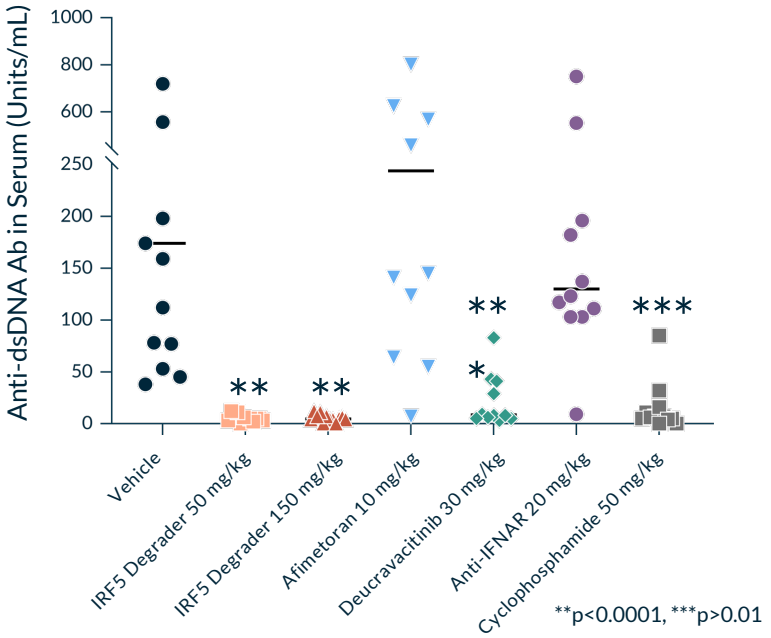
Decreased Proteinuria



Early, Sustained and Complete Reduction in Anti-dsDNA Abs



Superior Activity Reducing Anti-dsDNA at Week 37



Vehicle p.o. QD
IRF5 Degrader 150 mg/kg p.o. QD
Deucravacitinib 30 mg/kg p.o. QD
Cyclophosphamide 50 mg/kg i.p. QW
IRF5 Degrader 50 mg/kg p.o. QD
Afimetoran 10 mg/kg p.o. QD
Anti-IFNAR 20 mg/kg s.c. 2x/week

- **IRF5 degrader, dosed once a day for 107 days, leading to 80% and >90% IRF5 degradation, led to decreased proteinuria and near complete reduction of serum anti-dsDNA Abs superior to standard of care, approved and clinically active agents tested**
- **Significantly reduces key interferon genes (OAS1, IFIT1, IF44) at 150mg/kg in the blood from NZB.W1 mice comparable to other test agents**

[1]NZBW1 mice develop a similar disease to human SLE, characterized by anti-dsDNA ANA production, and immune complex mediated nephritis due to defects in multiple genes; #IRF5 Degrader 50 mg/kg p.o. and 150 mg/kg p.o. lines are overlapping.

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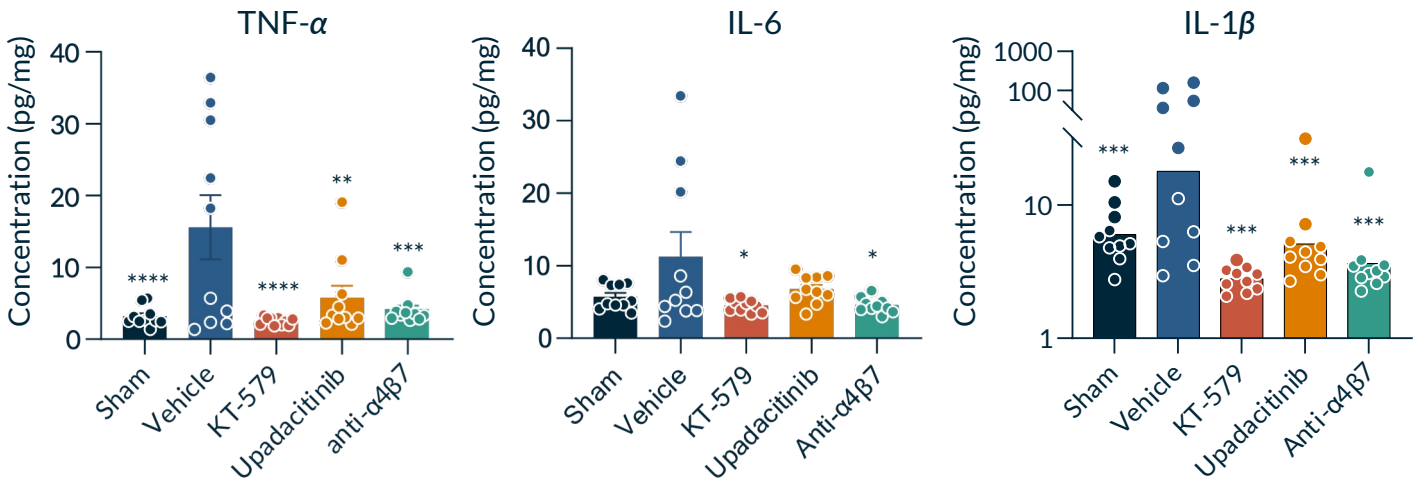
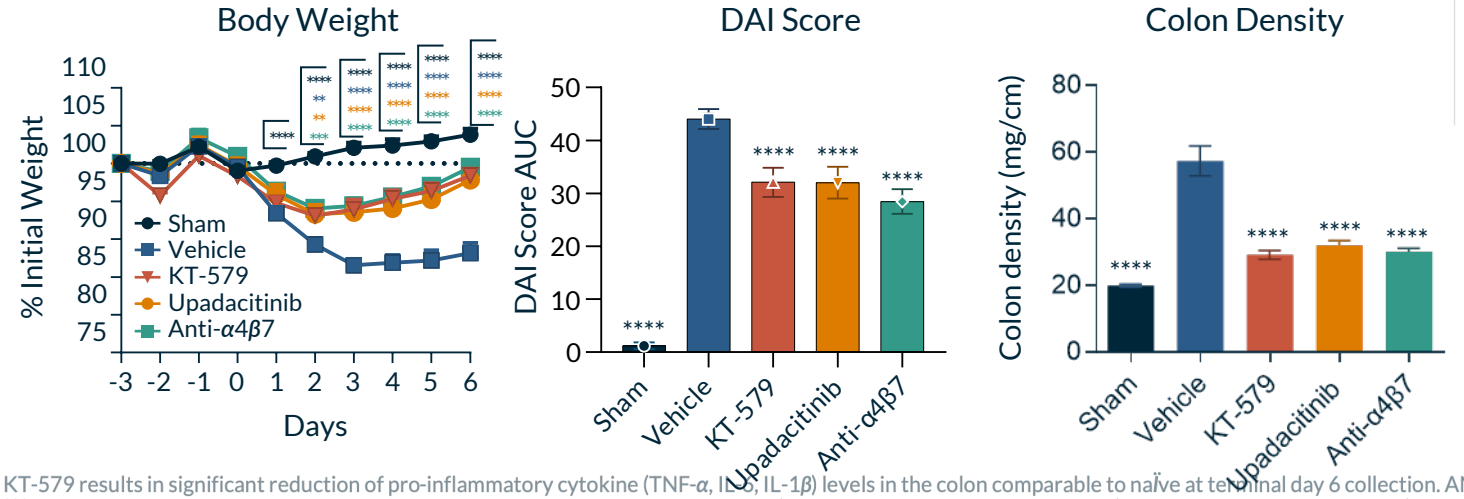
KT-579 Reduces Disease Progression, Fibrosis, and Intestinal Damage in the Preclinical TNBS Mouse Model of IBD

KT-579 Reduces Inflammatory Cytokines in the Colon

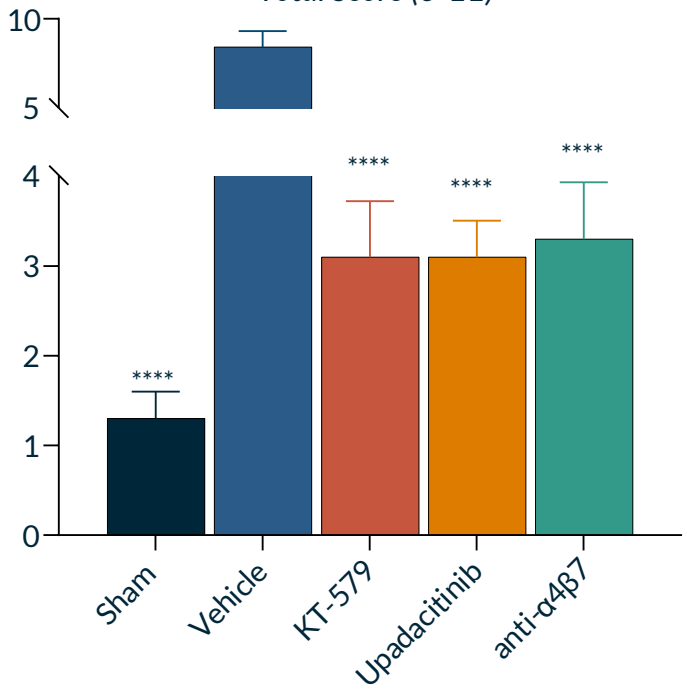


KT-579 Reduces Disease Activity



H&E Pathological Scoring

Total Score (0-11)





KT-579 demonstrated disease-modifying activity comparable or superior to approved and clinically active therapies

KT-579 results in significant reduction of pro-inflammatory cytokine (TNF-α, IL-6, IL-1β) levels in the colon comparable to naïve at terminal day 6 collection. ANOVA, *<0.05, **<0.005, ***<.0001. KT-579 treatment in the TNBS model results in significant reduction of A) body weight loss on Days 2-6 and B) maintenance of colon density (mg/cm) at terminal Day 6 collection. C) Pathological ex vivo analyses of the colons demonstrated KT-579 significantly reduced disease severity resulting in fewer inflammatory infiltrates, improved crypt structure integrity and lack of crypt abscesses. ANOVA, *<0.05, **<0.005, ***<0.001, ****<.0001.

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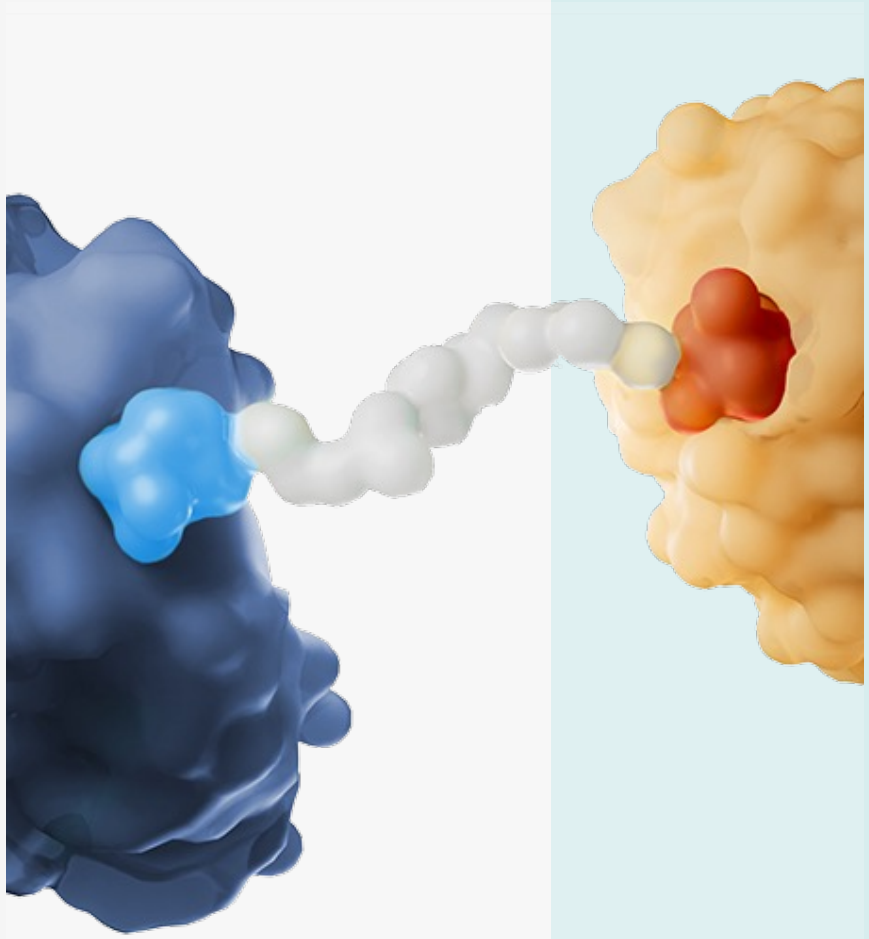

Appendix: Partnered Programs

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IRAK4 Biology and Target Rationale

Target Rationale

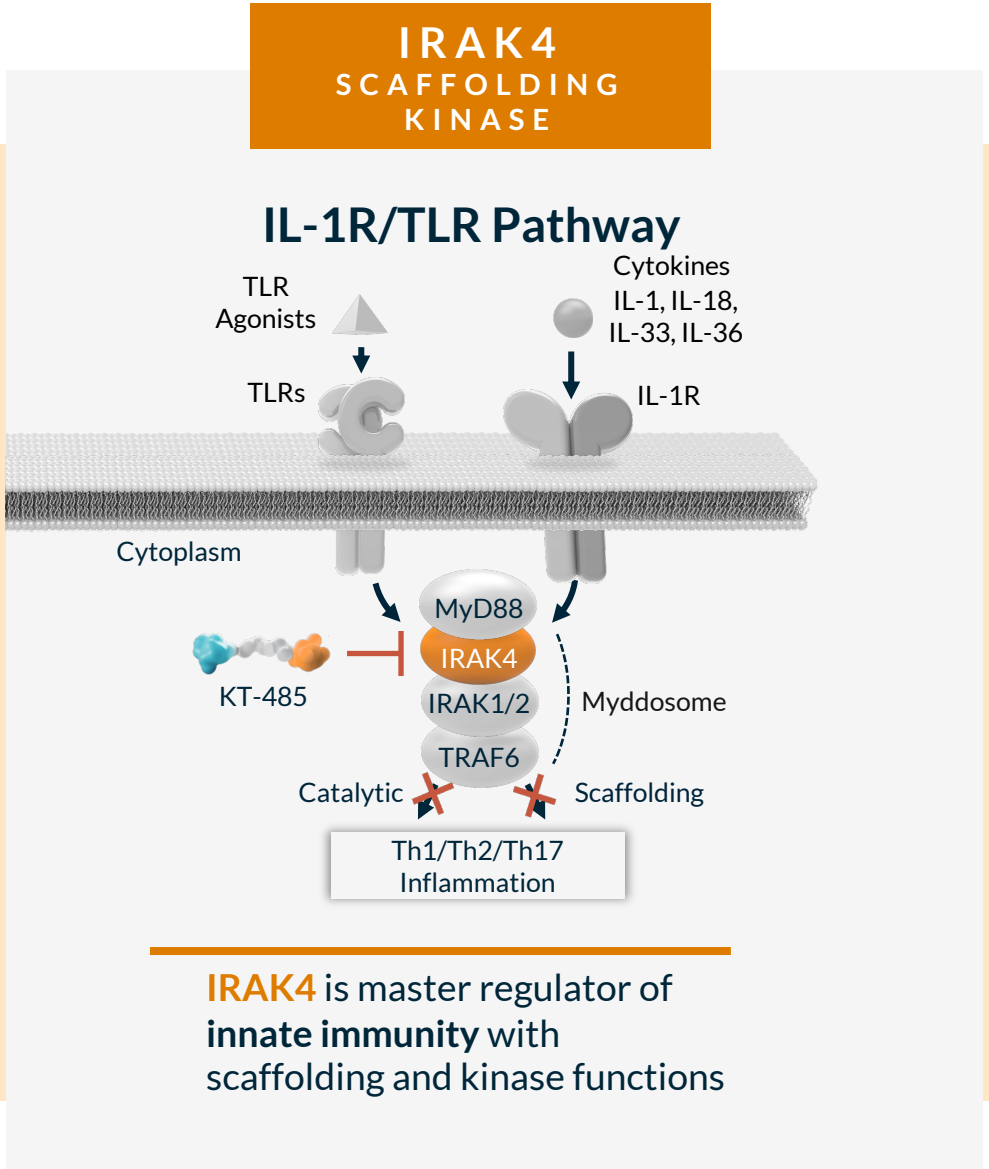
- IRAK4 is an obligate node in IL-1R/TLR signaling, and its degradation is the only approach to fully block the pathway

Human Genetics

- Adult humans with IRAK4 null mutation are healthy

Clinical Pathway Validation

- IRAK4 degradation has the potential to achieve a broad, well-tolerated anti-inflammatory effect
- Multiple development opportunities in immune-inflammatory diseases which signal through MyD88/IRAK4 have been clinically validated[1]:
  - IL-1α/IL-1β: RA, CAPS, HS, AD, RP, Gout
  - IL-18: AD, Macrophage Activation Syndrome
  - IL-36: Generalized Pustular Psoriasis
  - IL-33: Asthma, COPD
  - IRAK4 SMI: RA



[1]Diseases where IL-1R/TLR pathway has been implicated in pathogenesis; Figure adapted from West NT. *Front Immunol* 2019.

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CDK2 Molecular Glue Degrader Advantage

Target Biology and Rationale

- Regulates the G1-S transition, with its partner CCNE
- CCNE is often overexpressed or amplified in HGSOC and CDK4/6i resistant breast cancers, driving uncontrolled cell proliferation

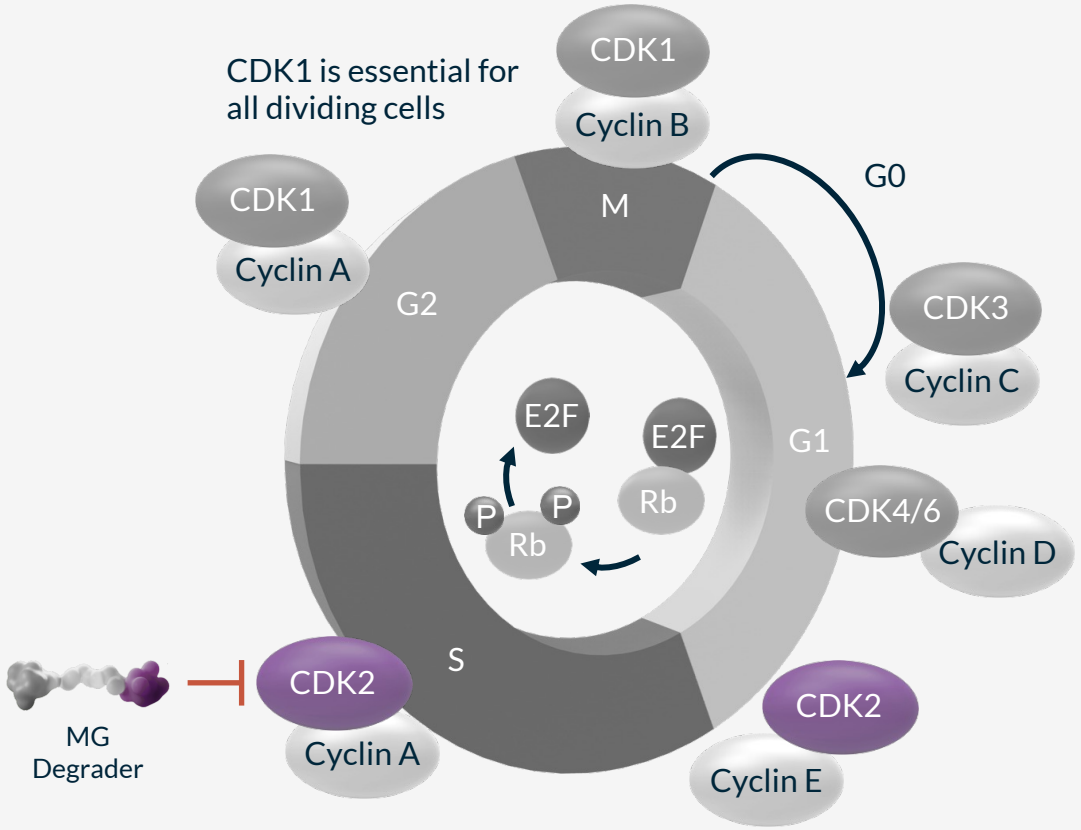
Genetically Validated

- DEPMAP identifies CCNE1 amplification as a biomarker of dependency for CDK2 across cancer cell types
- High CCNE activity is associated to resistance of CDK4/6 therapies

Advantages of MG Degraders

- CDK2 inhibitors face challenges due to similarity with other CDKs, including CDK1, causing toxicities before effective CDK2-pRB pathway inhibition can be achieved
- MG degraders offer greater selectivity, and potentially broader therapeutic index with more complete CDK2 pathway inhibition

CDK2
MASTER CELL CYCLE REGULATOR



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Abbreviations

Ab	Antibody
AD	Atopic Dermatitis
AIA	Antigen-Induced Arthritis
ASMS	Affinity Selection Mass Spectrometry
AN Count	Abscess and Inflammatory Nodule Count
BID	Twice a day
BP	Bullous Pemphigoid
BSA	Body Surface Area
CAPS	Cryopyrin-Associated Periodic Syndrome
CD	Crohn's Disease
CDK2	Cyclin-Dependent Kinase 2
COPD	Chronic Obstructive Pulmonary Disease
CRSwNP	Chronic Rhinosinusitis with Nasal Polyps
Cryo-EM	Cryo-Electron Microscopy
Ctrl	Control
CSU	Chronic Spontaneous Urticaria
$DC_{\#}$	Degradation Concentration
DCs	Dendritic Cells
DEL	DNA-Encoded Library
DLQI	Dermatology Life Quality Index
DM	Dermatomyositis
DMSO	Dimethyl Sulfoxide
dsDNA	Double-Stranded DNA
EASI	Eczema Area and Severity Index
EoE	Eosinophilic Esophagitis
EU	European Union
FDA	Food and Drug Administration
FeNO	Fractional Exhaled Nitric Oxide
FIH	First-in-Human
GI	Gastrointestinal
GLP	Good Laboratory Practice
GOF	Gain of Function
GWAS	Genome-Wide Association Study
HD	High Dose
HDM	House Dust Mite
HiSCR	Hidradenitis Suppurativa Clinical Response
hPBMC	Human Peripheral Blood Mononuclear Cells
HS	Hidradenitis Suppurativa
HTS	High Throughput Screening
HV	Healthy Volunteers
I&I	Immunology and Inflammation
IA	Interim Analysis
IBD	Inflammatory Bowel Disease
$IC_{\#}$	Inhibitory Concentration
ICS	Inhaled Corticosteroid
IFN	Interferon
ISG	Interferon-Stimulated Gene
IGA	Investigator Global Assessment
IgE	Immunoglobulin E
IHS4	International Hidradenitis Suppurativa Severity Score
IL	Interleukin
IND	Investigational New Drug Application
IRAK4	Interleukin 1 Receptor Associated Kinase 4
IRF5	Interferon Regulatory Factor 5
JP	Japan
KO	Knockout
LABA	Long-Acting Beta Agonist
LAMA	Long-Acting Muscarinic Antagonist
LD	Low Dose
LLOQ	Lower Limit of Quantification
LOF	Loss of Function
LPS	Lipopolysaccharide Solution
LS	Least Squares
LTRA	Leukotriene Receptor Antagonist
MAD	Multiple Ascending Dose Study
MOA	Mechanism of Action
NF-kB	Nuclear Factor Kappa B
NHP	Nonhuman Primate
nM	Nanomolar
NRS	Numerical Rating Scale
PBMC	Peripheral Blood Mononuclear Cells
Pbo	Placebo
PGA	Physician Global Assessment

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Abbreviations

Ph	Phase
PK/PD	Pharmacokinetics/Pharmacodynamics
pM	Picomolar
PN	Prurigo Nodularis
POC	Proof-of-Concept
POEM	Patient-Oriented Eczema Measure
PPNRS	Peak Pruritus Numerical Rating Scale
PRRs	Pattern Recognition Receptors
PsO	Psoriasis
pSTAT	Signal Transducer and Activator of Transcription
QD	Once a day
QoL	Quality of Life
R&D	Research and Development
RA	Rheumatoid Arthritis
RNA	Ribonucleic Acid
ROW	Rest of World
RP	Recurrent Pericarditis
RQLQ	Rhinoconjunctivitis Quality of Life Questionnaire
SAD	Single Ascending Dose study
SAE	Serious Adverse Event
SCORAD	SCORing Atopic Dermatitis
SD	Single Dose
SLE	Systemic Lupus Erythematosus
SMI	Small Molecule Inhibitor
SOC	Standard of Care
SSc	Systemic Sclerosis
STAT	Signal Transducer and Activator of Transcription
STAT6	Signal Transducer and Activator of Transcription 6
TARC	Thymus and Activation-Regulated Chemokine
TEAE	Treatment Emergent Adverse Event
Th1	T Helper 1
Th2	T Helper 2
Th17	T Helper 17
TLR	Toll-like Receptors
TNSS	Total Nasal Symptom Score
TPD	Targeted Protein Degradation
TPP	Target Product Profile
TRAE	Treatment Related Adverse Event
TYK2	Tyrosine Kinase 2
UC	Ulcerative Colitis
US	United States
vIGA-AD	Validated Investigator Global Assessment for AD
WW	Worldwide

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