KYMERA 10 EST. 2016

Third Quarter 2026 Quarterly Results Call

October 29, 2026

Agenda

Introduction
Justine Koenigsberg
Vice President, Investor Relations

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

KT-579 Phase 1 Healthy Volunteer Data
Terence Rooney, MD
Chief Medical Officer

Third Quarter 2026 Results
Bruce Jacobs, CFA, MBA
Chief Financial Officer

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

Question and Answer Session

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Revolutionizing Immunology with Oral Medicines

Nello Mainolfi, PhD

Founder, President and CEO

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A Long Overdue Paradigm Shift in Immunology Treatment

Existing treatments have functional limitations that create gaps in care

Leaving millions of patients without adequate treatment options

An effective, safe, and convenient oral medication could transform patient care

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KT-621 Accelerated Development Enabled Early De-risking and Efficient Path to Registration Across All Type 2 Diseases

Ph1

Ph1 HV
SAD-MAD

✓ **Jun 2025**
>200 HVs for up to 14 days

BROADEN STUDY

Ph1b AD
Single Arm, Open Label

✓ **Dec 2025**
22 AD patients for 28 days

Ph2

BROADEN2 TRIAL

Ph2b AD
Dose Ranging
>200 AD patients for 16 weeks

✓ **Oct 2026: Last patient last dose; Data expected by year-end 2026**

BREADTH TRIAL

Ph2b Asthma
Dose Ranging

Ongoing; Data expected late-2027

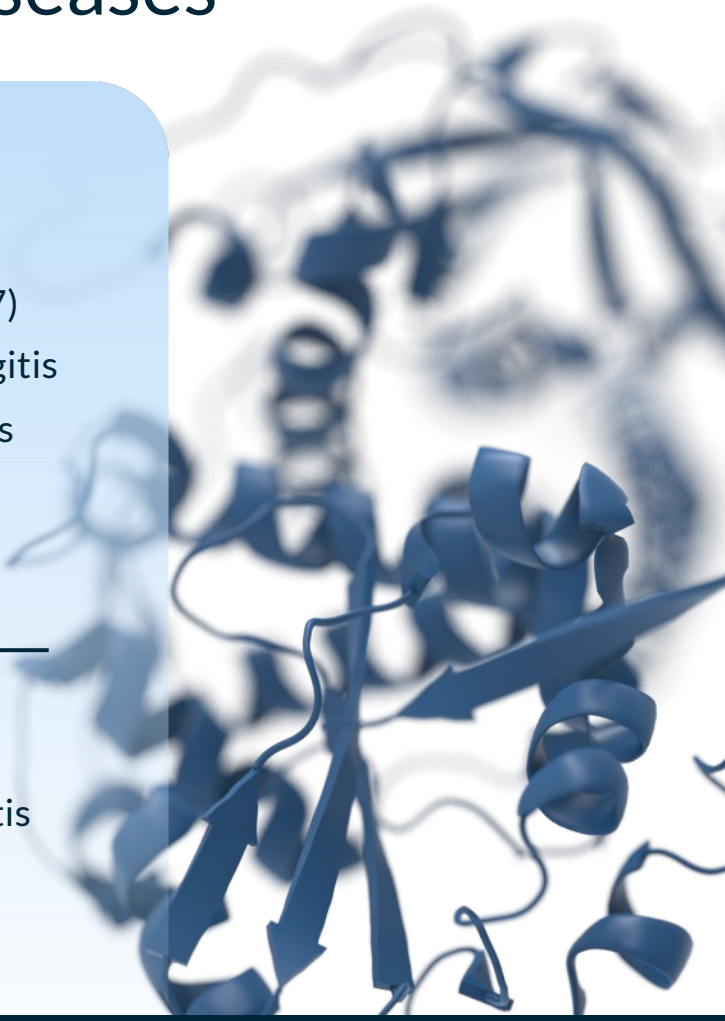
Ph3

Dermatology/GI

- Atopic Dermatitis (initiate by mid-2027)
- Eosinophilic Esophagitis
- Chronic Spontaneous Urticaria
- Prurigo Nodularis
- Bullous Pemphigoid

Respiratory

- Asthma
- Chronic Rhinosinusitis with Nasal Polyps
- Chronic Obstructive Pulmonary Disease



Early Phase 1 data showed deep target/pathway engagement, promising efficacy and favorable safety profile; Phase 2bs in AD and asthma have the potential to support Phase 3 trials across multiple indications

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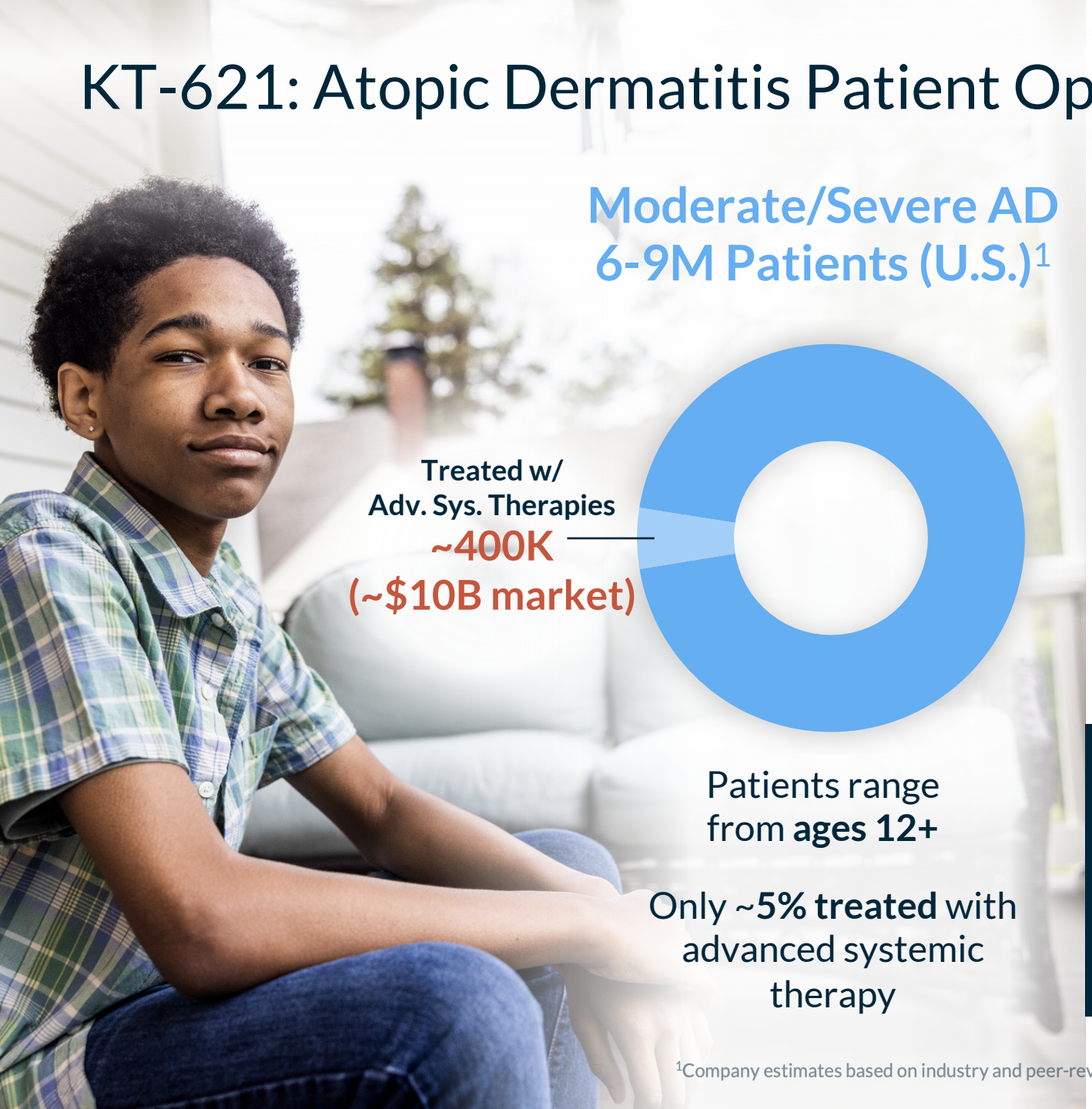
KT-621: Atopic Dermatitis Patient Opportunity



Moderate/Severe AD 6-9M Patients (U.S.)[1]

Treated w/ Adv. Sys. Therapies
~400K
(~$10B market)

Patients range from **ages 12+**

Only ~**5% treated** with advanced systemic therapy

Reasons for Low Penetration

- **Reluctance to use Advanced Systemic Therapies**
  Many patients don't accept their disease state to justify an injectable biologic or oral option with boxed safety warnings
- **High Treatment Burden**
  Multiple injections, blood tests, cold storage, access/reimbursement challenges

KT-621

- Opportunity to be a first line treatment option
- Potentially the first safe, effective, oral for patients across all Type 2 diseases

[1]Company estimates based on industry and peer-reviewed research.

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Building a Best-in-Industry Oral Immunology Pipeline

Combining the Convenience of Oral Drugs and the Activity of Biologics

	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 KT-621 – Asthma				**Ph2b AD Data:** By year-end 2026 **Ph2b Asthma Data:** Late-2027 **Ph3 AD Start:** By mid-2027
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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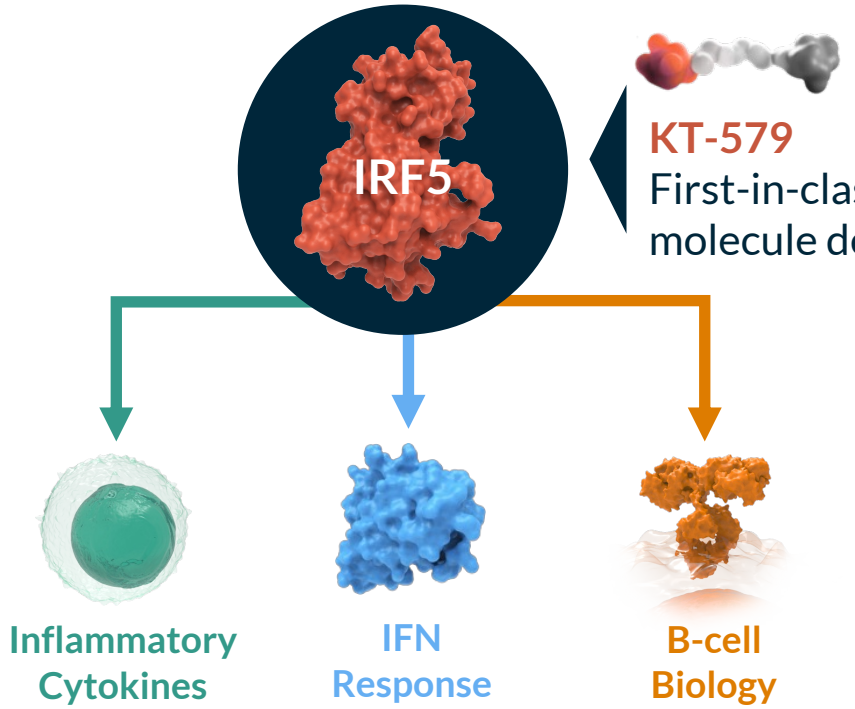
KT-579: A New Treatment Paradigm for Serious Autoimmune Diseases

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 lupus trial

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KT-579/IRF5 Degrader: Phase 1 Healthy Volunteer Data

Terence Rooney, MD

Chief Medical Officer

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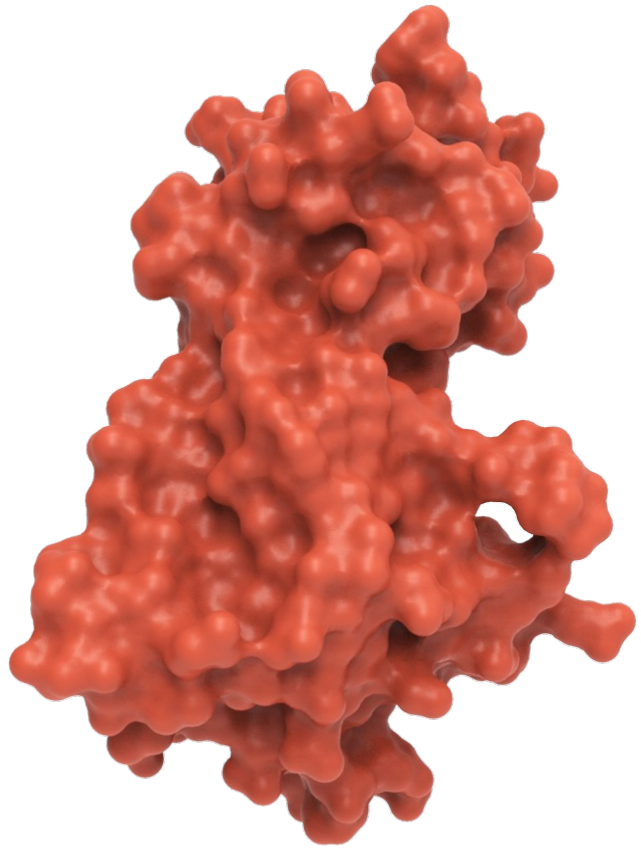
KT-579: Agenda

- Phase 1 Trial Design
- Demographics
- IRF5 Degradation
- *Ex Vivo* Blood Biomarkers
- Safety
- Next Steps



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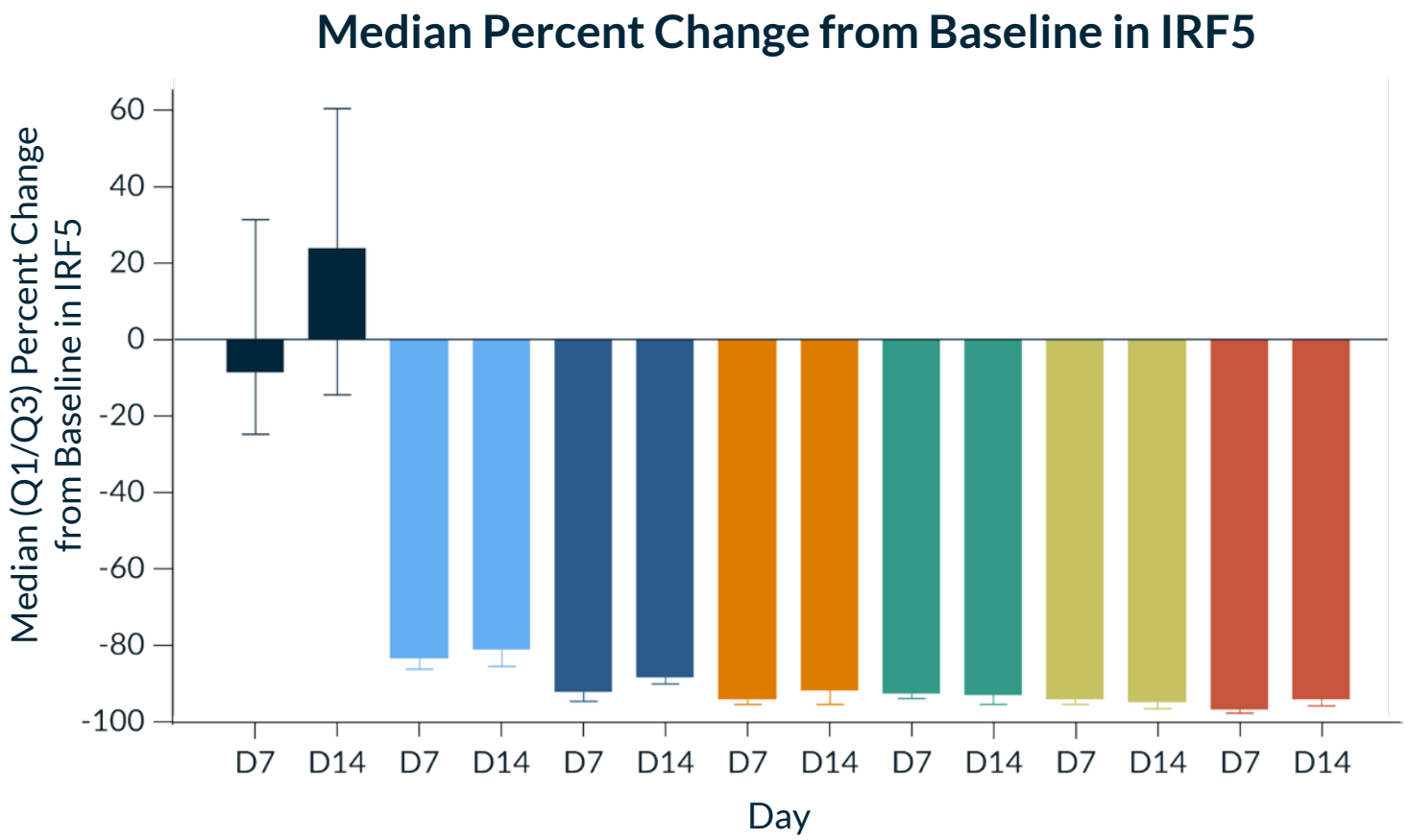
Multiple Daily Doses of KT-579 Achieved >90% IRF5 Degradation in Blood Across Almost All Dose Levels



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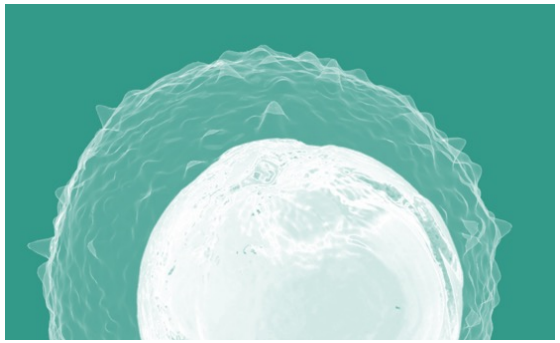
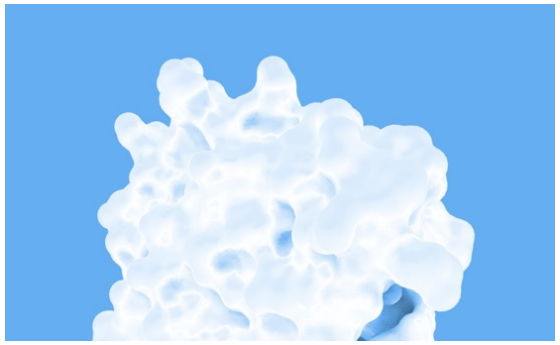
Complementary Ex Vivo Biomarker Assays Capture the Breadth of IRF5 Biology Across Three Key Immune Pathways[1]

INFLAMMATORY CYTOKINES[2]



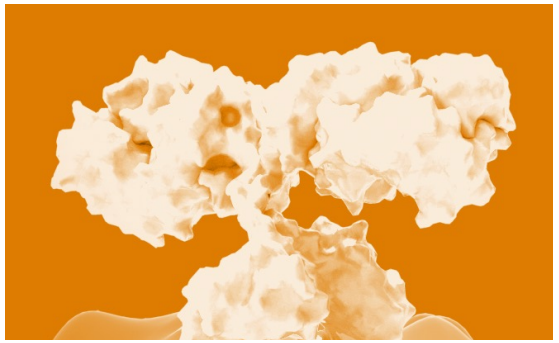
- IRF5 promotes production of key inflammatory cytokines in myeloid cells
- When dysregulated, these responses can sustain chronic inflammation, relevant to several autoimmune diseases

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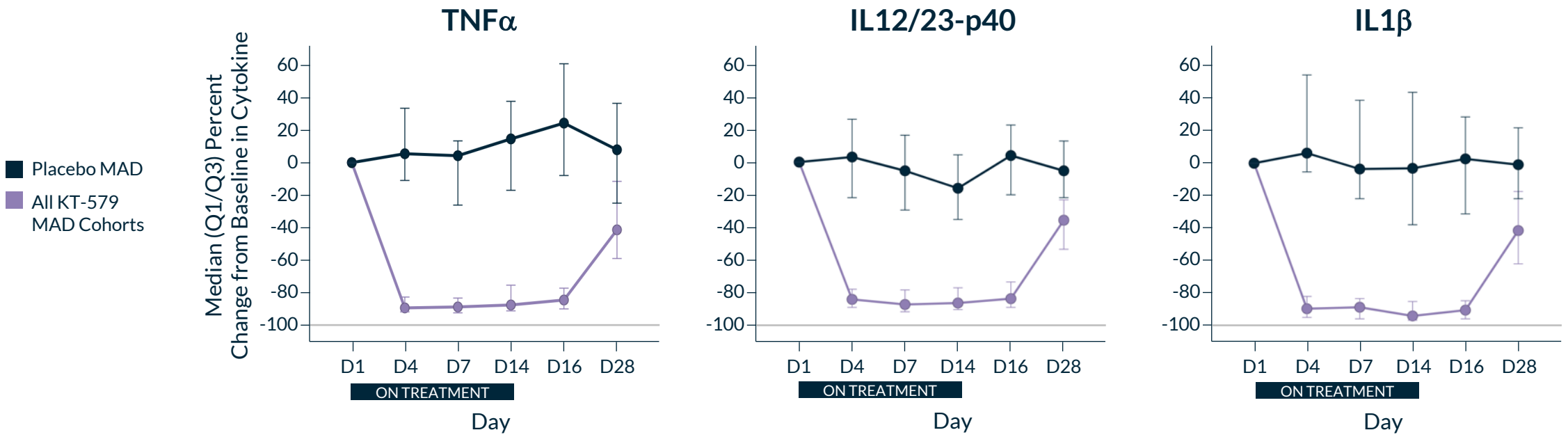
Multiple Daily Doses of KT-579 Achieved Deep Inhibition of Clinically Validated Inflammatory Cytokines



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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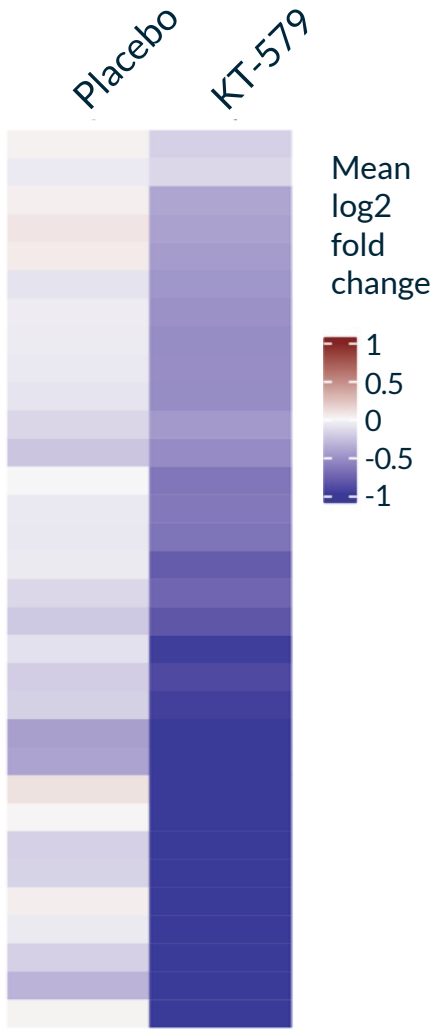
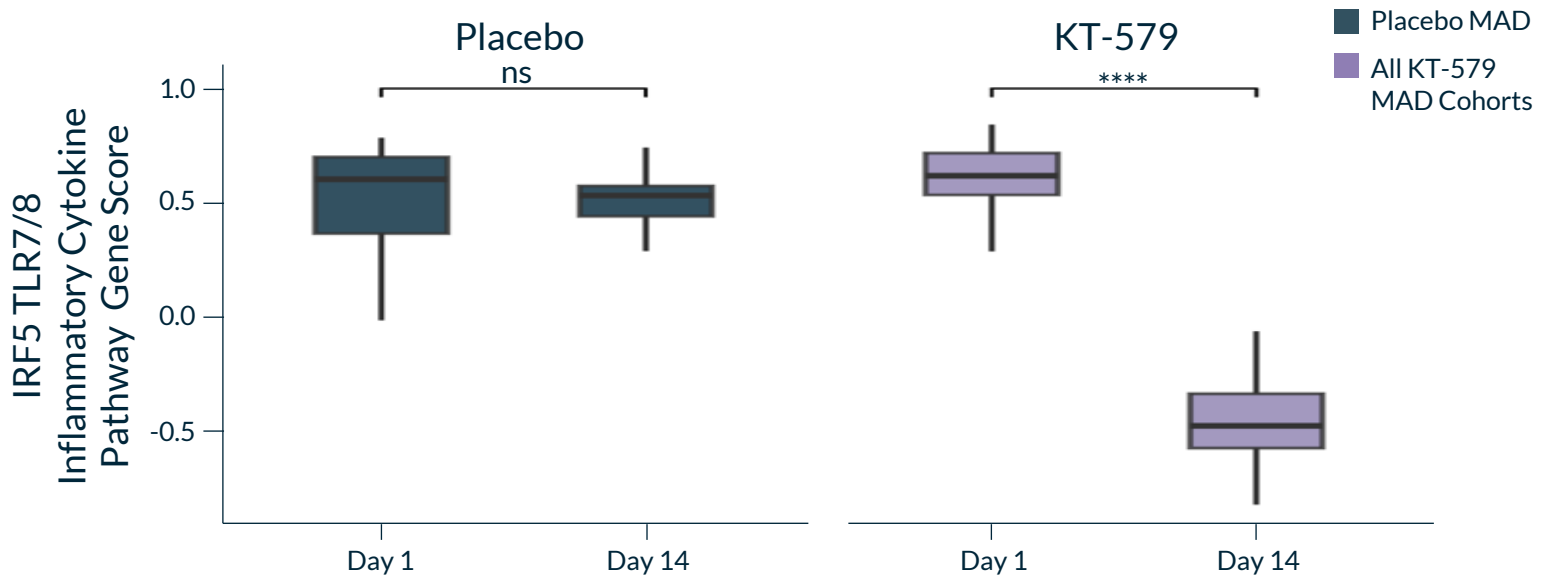
KT-579 Downregulates Inflammatory Cytokine Pathway Genes



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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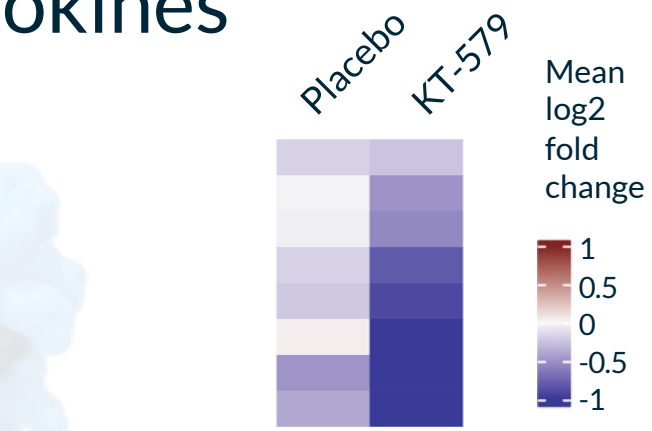
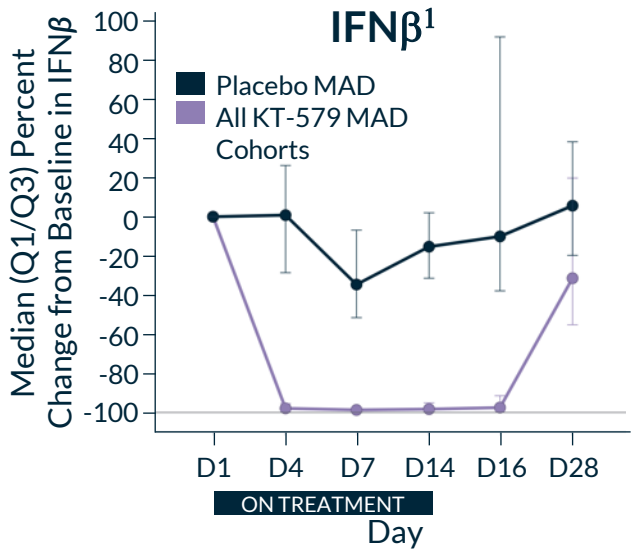
KT-579 Downregulates Interferon Pathway Genes and Cytokines



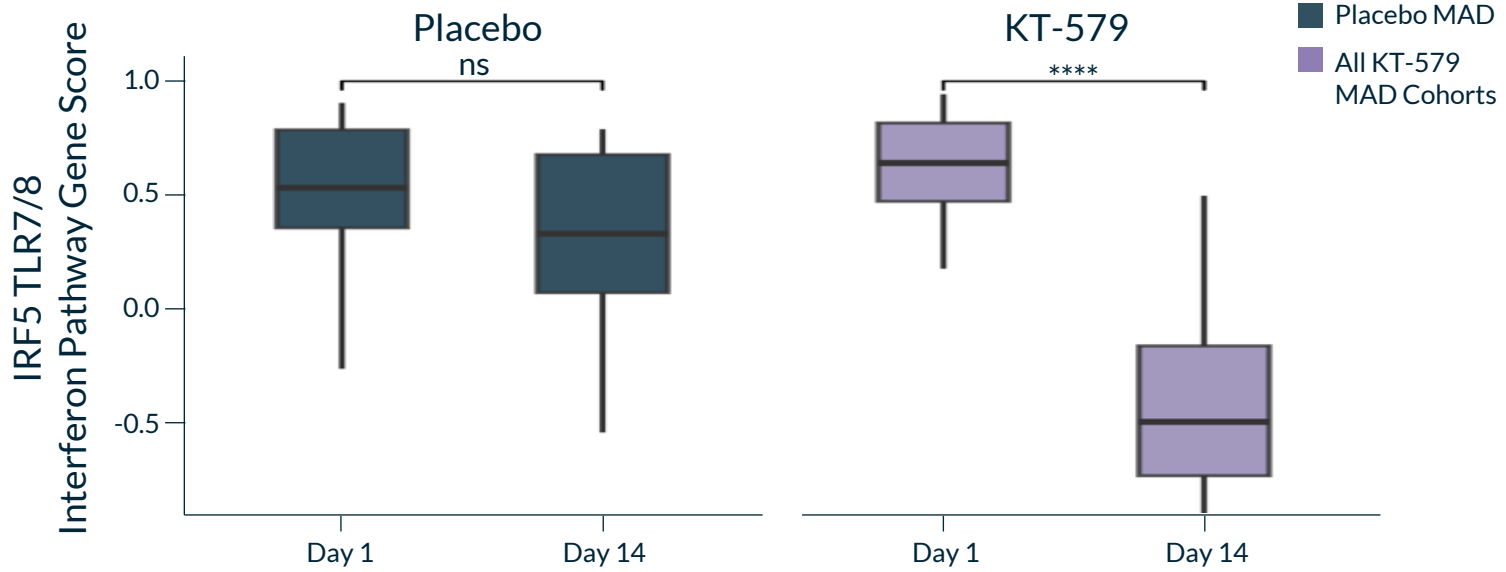


Median Inhibition Day 14[1]	IFNβ
Placebo	-15%
All KT-579 MAD Cohorts	**-98%**

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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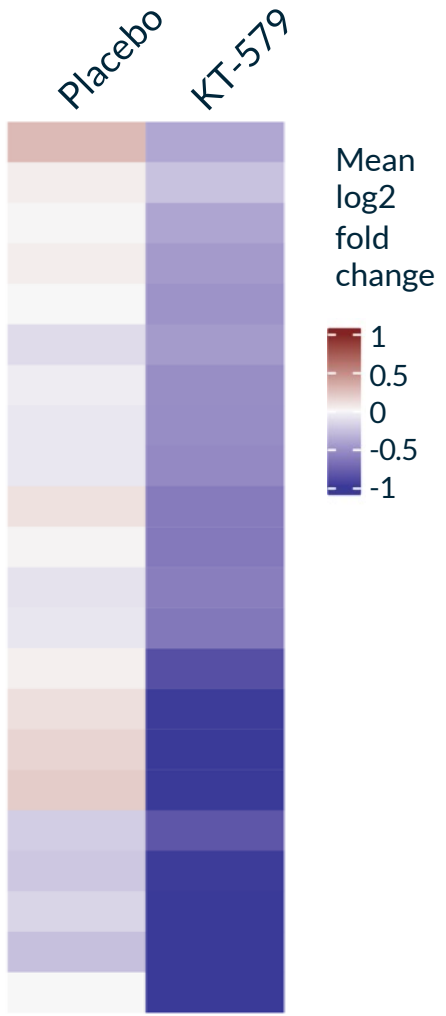
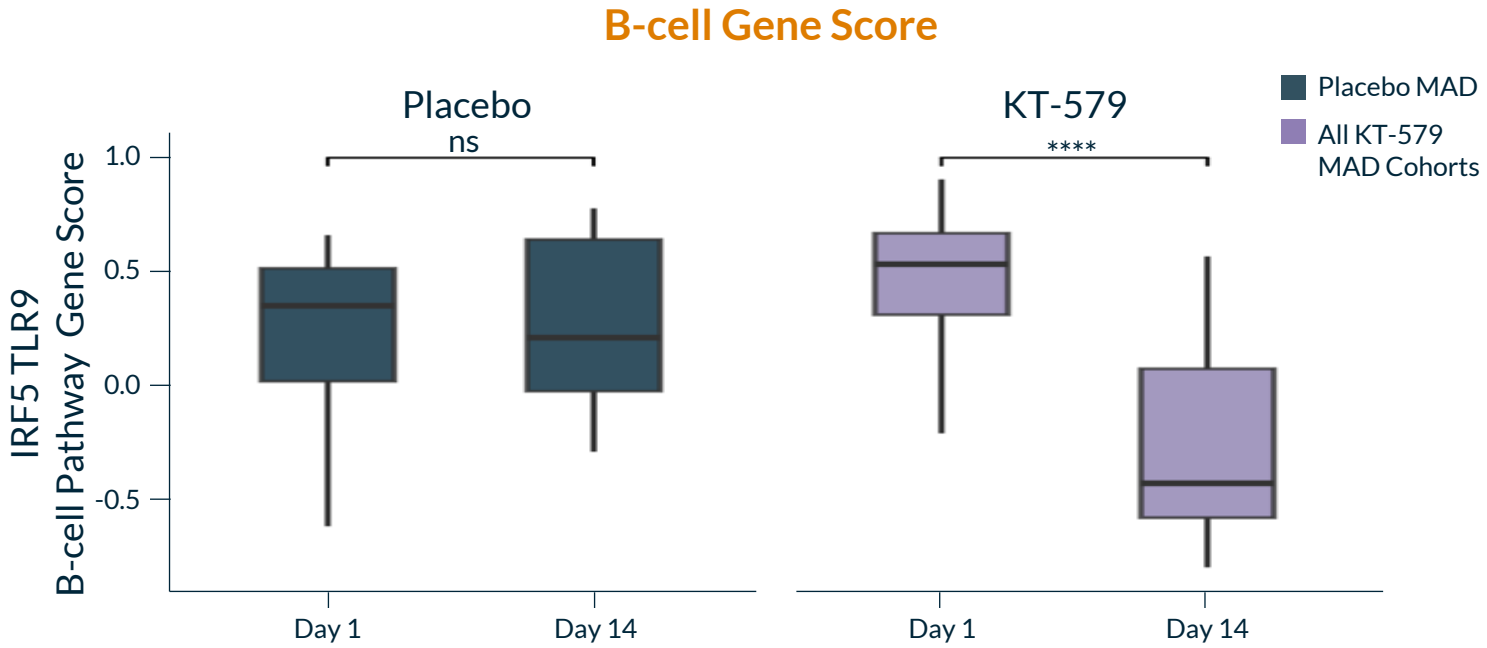
KT-579 Downregulates B-cell Pathway Genes



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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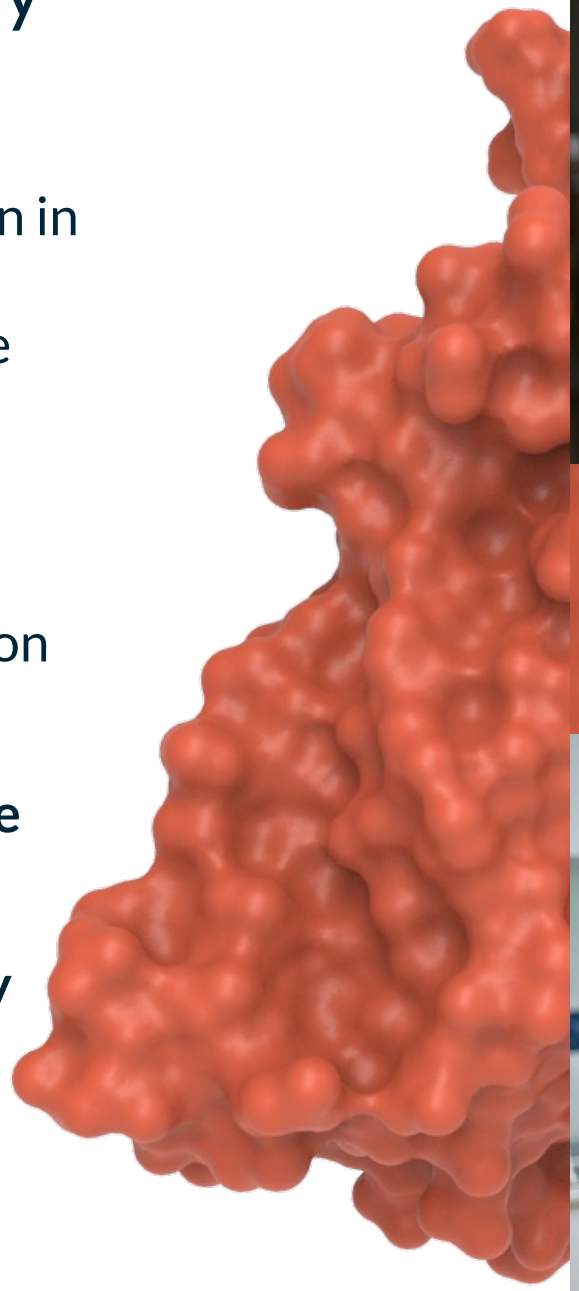
KT-579 Phase 1 Summary

- 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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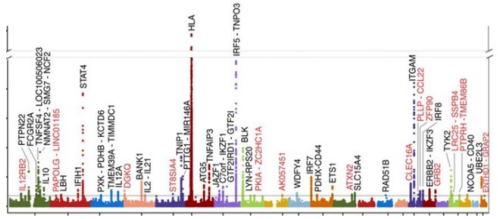
KT-579: First-in-Industry Opportunity in Lupus

Novel Oral Mechanism to Address Underlying Biology for High-Burden Disease

GENETICS/PRECLINICAL VALIDATION

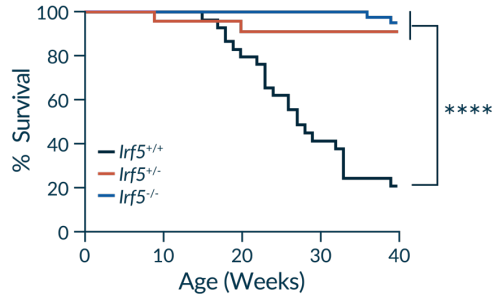
Meta Analysis

Meta-analyses identified IRF5 as a risk locus for lupus[1]



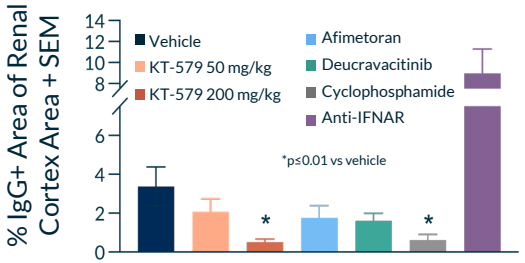
Survival

IRF5 KO increases survival in mice and has essential role in lupus[2]



Disease-modifying Activity

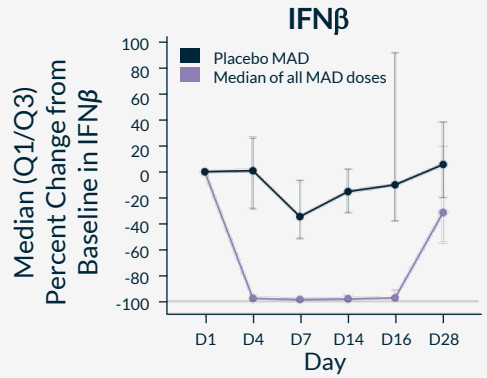
Activity comparable or superior to existing agents in preclinical lupus models



CLINICAL VALIDATION

KT-579 Phase 1 Data

Modulation of key pathway biology in Phase 1 HV trial



Pathway Validation

Pathways clinically validated in lupus (mAbs/depletors)

Saphnelo® | Benlysta® (belimumab) | GAZYVA® (obinutuzumab)

[1]Santana-de Anda et al., *Autoimmunity Reviews*, 2011; [2]Richez et al., *J Immunol*, 2010; All trademarks and registered trademarks are the property of their respective owners.

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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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Third Quarter 2026 Financial Results

Bruce Jacobs, CFA, MBA

Chief Financial Officer

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Third Quarter 2026 Income Statement

	Three Months Ended September 30,		Nine Months Ended September 30,	
	2026	2025	2026	2025
Collaboration revenue	$ —	$ 2,764	$ 99,365	$ 36,341
Operating expenses:				
Research and development	$ 128,509	$ 74,094	$ 346,152	$ 232,737
General and administrative	18,723	17,336	60,207	51,252
Impairment of long-lived assets	—	3,855	—	3,855
Total operating expenses	147,232	95,285	406,359	287,844
Loss from operations	(147,232)	(92,521)	(306,994)	(251,503)
Total other income, net	15,233	10,346	44,550	27,133
Net loss	$ (131,999)	$ (82,175)	$ (262,444)	$ (224,370)

Balance Sheet	September 30, 2026	December 31, 2025
Cash, cash equivalents & marketable securities	$1,645,883	$1,619,434

Unaudited consolidated statements of operations and comprehensive loss; in thousands.

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Closing Remarks

Nello Mainolfi, PhD

Founder, President and CEO

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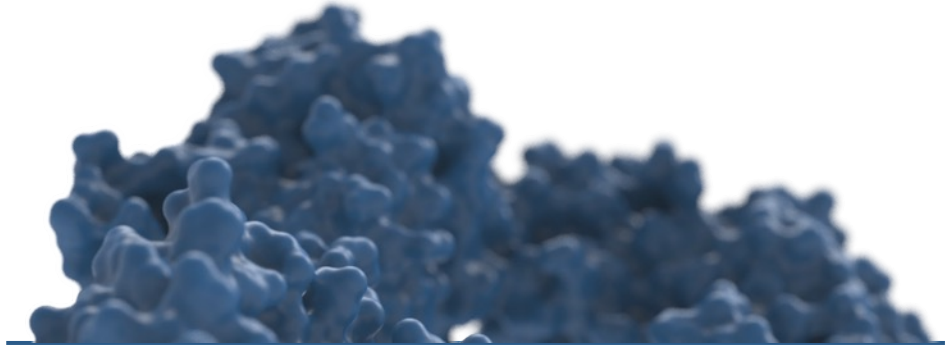
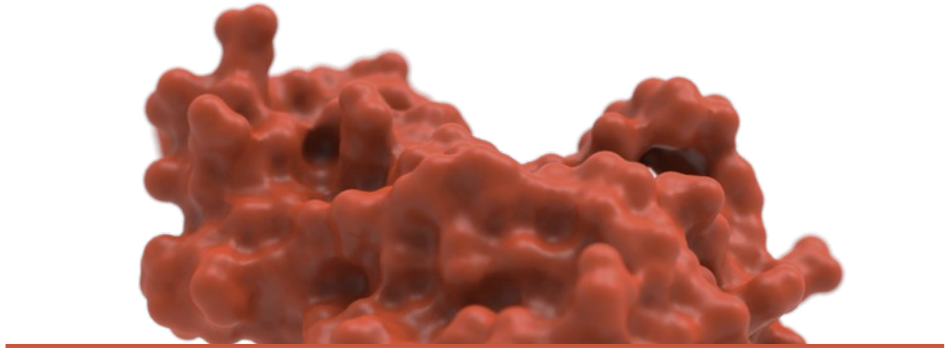
Kymera Wholly Owned Immunology Programs

Fundamental Immuno-inflammatory Pathways with Large Market Potential





	KT-621/STAT6	KT-579/IRF5
Innovation	**Potential to be the first and only oral medicine for Type 2 diseases**	**Kymera's seventh first-in-class molecule to enter the clinic, demonstrating strong human translation of PK/PD and safety**
Early Validation	Compelling profile in Phase 1 HV and AD trials showing blockade of IL-4/IL-13 signaling and early efficacy, with favorable safety profile	In Phase 1 HV, demonstrated robust degradation, safety, and modulation of three disease-driving pathways: inflammatory cytokines, IFN response and B-cell biology
Opportunity	A once-daily pill could transform/expand the market for common diseases: AD, asthma, EoE, COPD, others	Genetically validated across multiple serious autoimmune diseases: lupus, Sjögren's, RA, IBD, others
Status	• **BROADEN Ph2b AD** data by year-end 2026 • **BREADTH Ph2b asthma** data by late-2027 • **Phase 3 AD** start by mid-2027	• **LUMORA Ph1b lupus** start by early 2027

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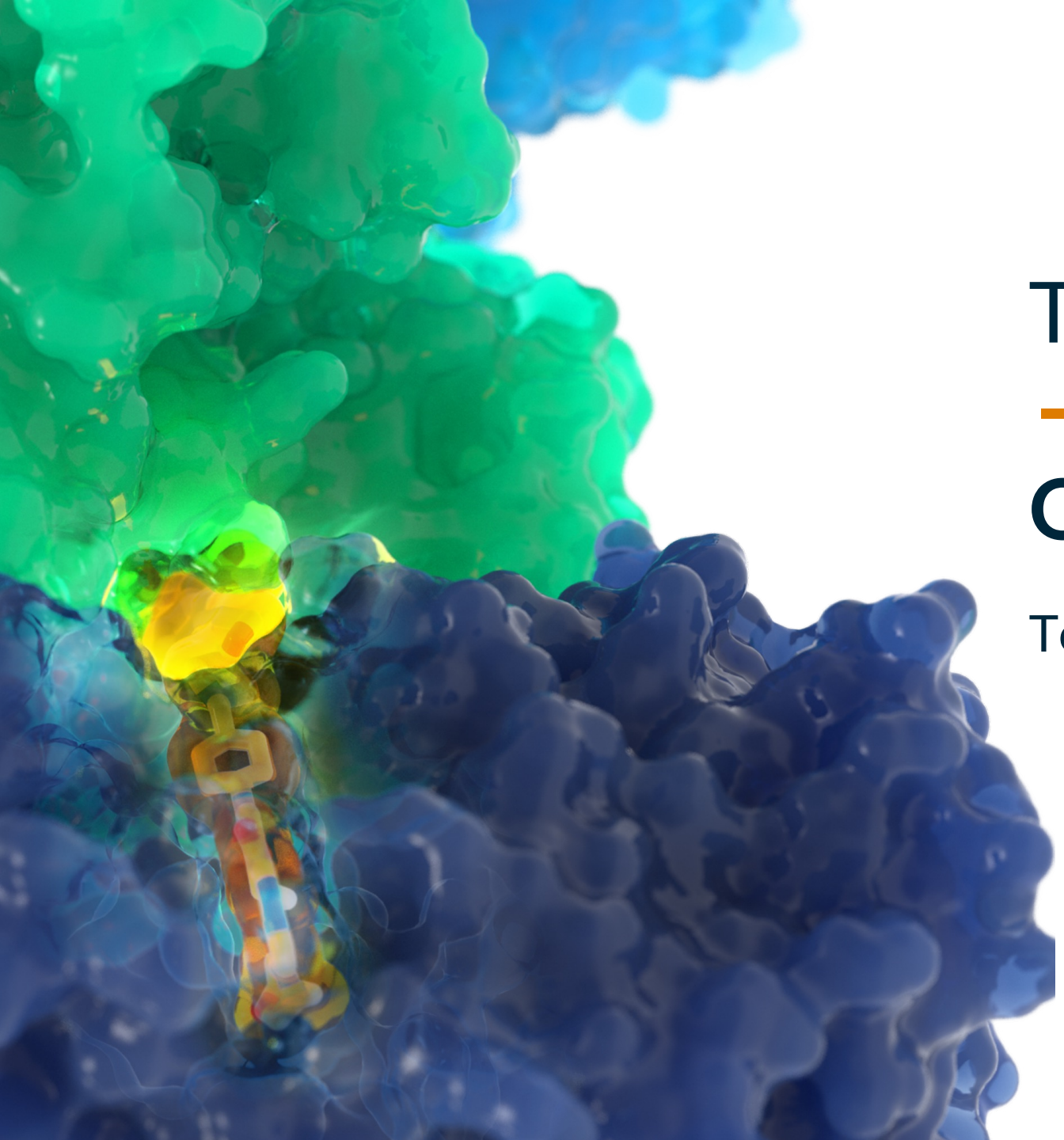

Thank You

Q&A

To ask a question, raise your virtual hand

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