INVENTING NEW MEDICINES

WITH TARGETED PROTEIN DEGRADATION

UBS Global Healthcare Virtual Conference

May 25, 2021

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Targeted Protein Degradation

What if you could remove disease causing proteins…

…with a small molecule-based technology?

What We Are Building

Vision

A fully integrated **degrader medicines company** that discovers, develops, and commercializes transformative medicines while leading the evolution of targeted protein degradation (TPD)

Opportunity

- Potential to **expand the druggable proteome dramatically**

Platform

- Advancing **TPD beyond current opportunities**

Strategy

- Focusing on undrugged targets and clinical indications with **high unmet medical need and franchise potential**

Team

- Driven by a **culture of scientific innovation**

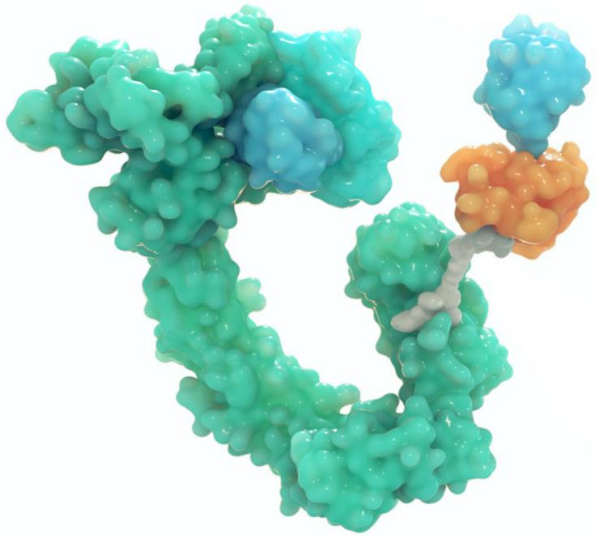
Kymera: A Leading TPD Company

Fierce 15 WINNER FierceBiotech

BOSTON BUSINESS JOURNAL BPtW 2021 BEST PLACES TO WORK

Founded:	2016
Stage:	Clinical
NASDAQ:	KYMR
Employees:	>100
Cash balance at Q1'21*:	$435M

KYMERA



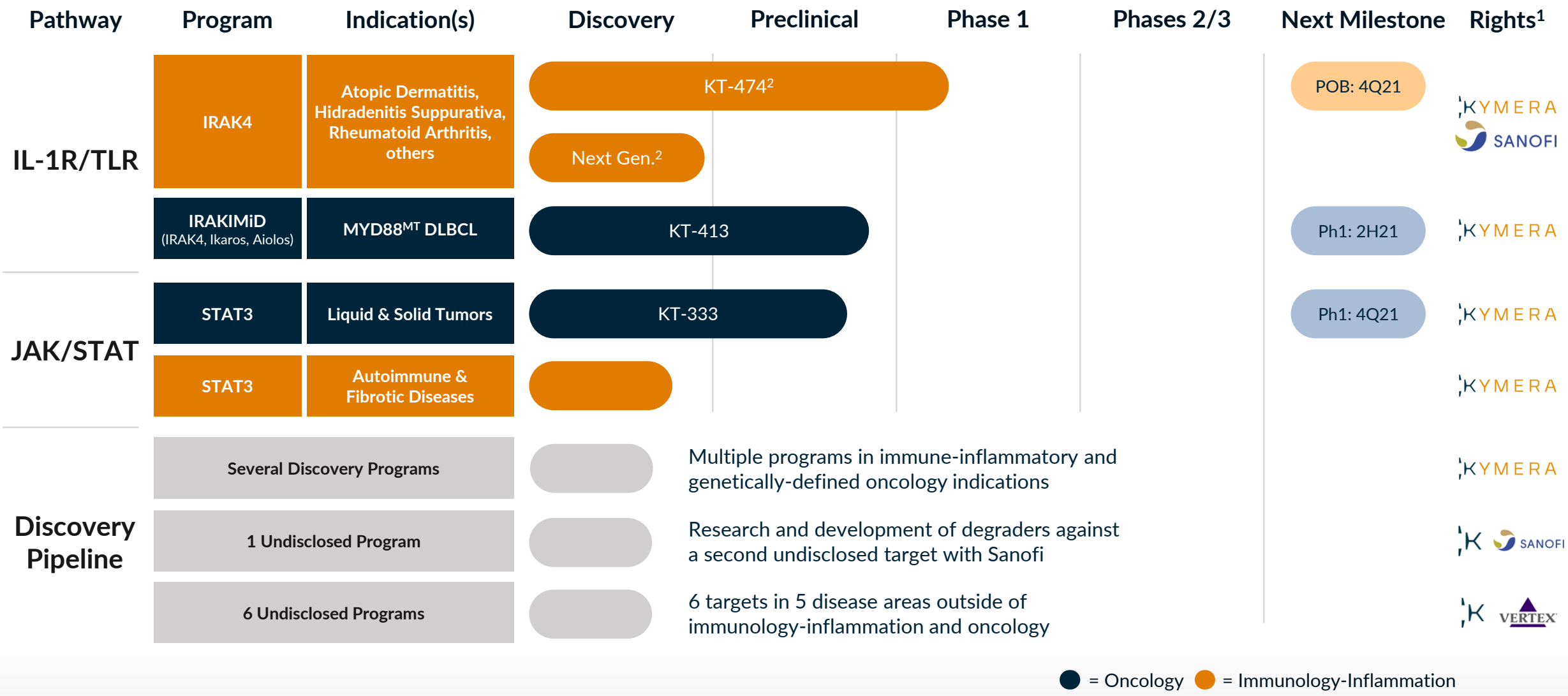
- Premier, **disease agnostic** protein degrader discovery platform
- Key **enabling partnerships**:

SANOFI VERTEX gsk

- Initial focus in **immune-inflammation (I/I) and oncology**
- First company set to dose degrader to **healthy volunteers and I/I patients**
- Expect **3 INDs** and clinical initiations by end of **2021**
- First proof-of-biology established in humans in **2021**

* Kymera expects that its cash, cash equivalents, and investments as of 3/31/2021, excluding any future potential milestones from collaborations, will enable the Company to fund its operational plans into 2025.

Kymera's Pipeline of Novel Protein Degraders



1. Option to participate equally in the development and commercialization of Sanofi-partnered programs in the US.
2. Sanofi collaboration to develop IRAK4 degrader candidates, including KT-474 (SAR444656), outside of oncology and immuno-oncology fields.

Pegasus™ Platform and R&D Approach

KYMERA

Targeted Protein Degradation

Biology

Co-opting a Naturally Occurring Process to Regulate Protein Levels

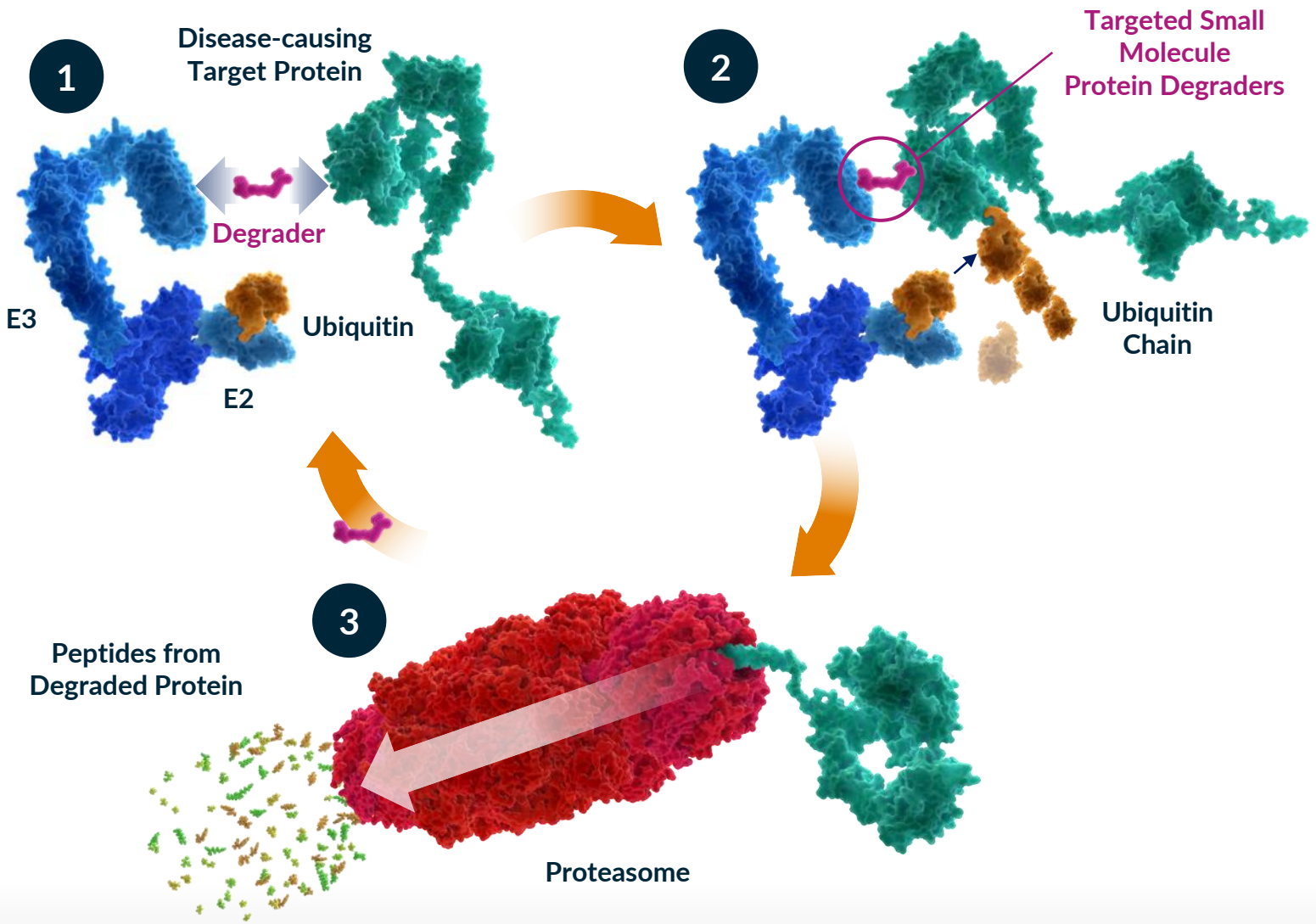
1. E3 ligase recognizes protein
2. Ubiquitin chain transferred
3. Protein is marked for elimination

KYMERA

Broad Opportunity
Only Binding Site Required

Efficient
Catalytic

Prolonged Impact
Targeted Protein Degradation



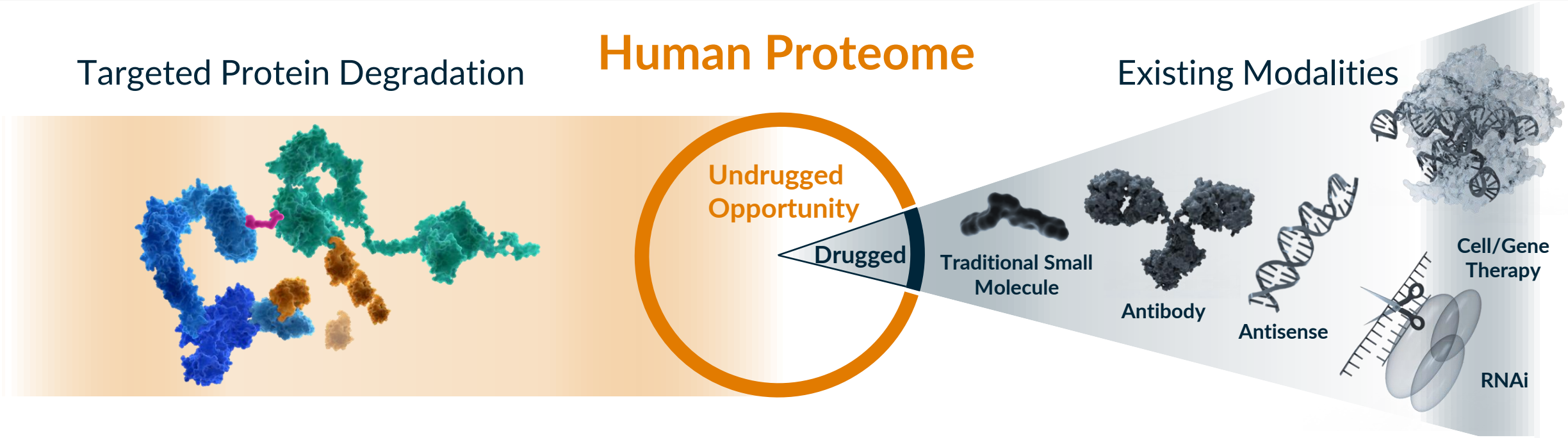
Targeted Protein Degradation

Next Potential Breakthrough Modality to Expand Drugged Proteome

Targeted Protein Degradation

Human Proteome

Existing Modalities



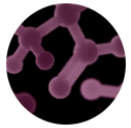
✓	**Undruggable Targets** Scaffold, transcript factor, multiple functions	✓
✓	**Efficient** Development / Manufacturing	X
✓	**Systemic** Exposure	X
✓	**Oral** Bioavailability	X

Proprietary Pegasus™ TPD Platform

Key capabilities

Expanded E3 ligase toolbox

- **E3 ligase Whole-Body Atlas**: Identification of the **expression profiles of ~600 unique E3 ligases**
- Match target protein with appropriate E3 ligase based on expression, distribution, intracellular localization, and biology
- **Toolbox of proprietary ligands** leverages the E3 Ligase Whole-Body Atlas



Understanding degradation (PK/PD) across tissue types

- **Ternary complex modeling tool optimizes the development** of highly efficient and selective degrader therapeutics
- **Quantitative System Pharmacology Model** measures and predicts diverse sets of parameters that impact protein levels
- Based on understanding of PK/PD, both *in vitro* and *in vivo*, and across different tissues and cell types

Proprietary Chemistry

- **Comprehensive hit finding technologies toolbox**: chemoproteomics, DEL, fragment screens, *in silico*
- **Proprietary chemistry expertise** enables the design and optimization of both E3 ligases and target protein binders
- Ability to convert into degraders with optimal pharmaceutical properties tailored to specific patient populations

Leading the Evolution of Targeted Protein Degradation

What if you could remove disease causing proteins…

…only where it matters?

Pegasus: E3 Ligase Whole-Body Atlas

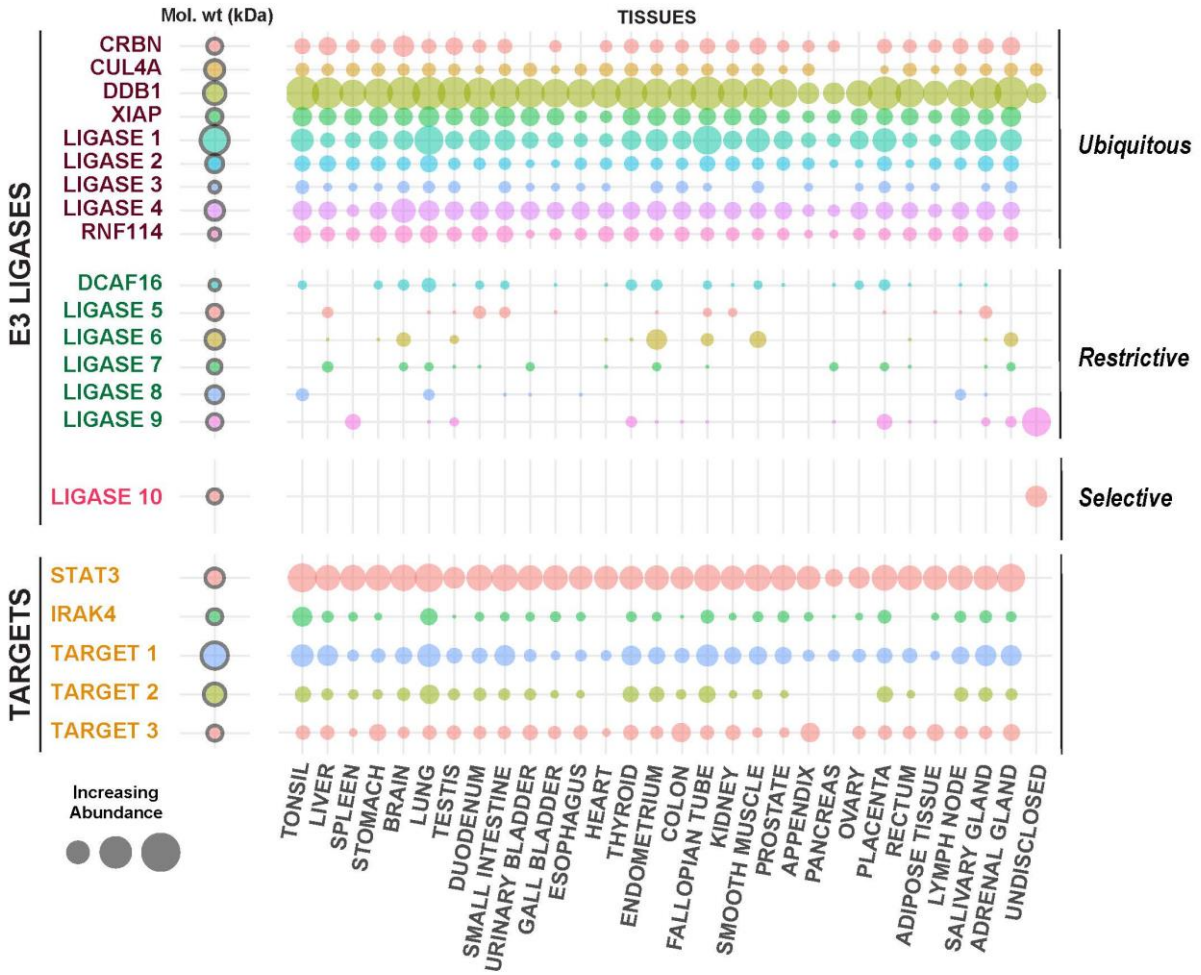
Different expression profiles of E3's provide opportunity for tissue selective/restrictive degradation

Expanded E3 ligase toolbox

Understanding degradation (PK/PD) across tissue types

Proprietary Chemistry

- Focused on determining the expression profiles of ~600 unique E3 ligases
- Patterns mapped in both disease and healthy contexts
- Ability to match a target protein with appropriate E3 ligase based on expression and biology
- Vision to develop tissue-selective or tissue-restrictive degraders to enable novel therapeutic opportunities



Pegasus: E3 Ligase Whole-Body Atlas

A Bone Marrow Sparing E3 Ligase

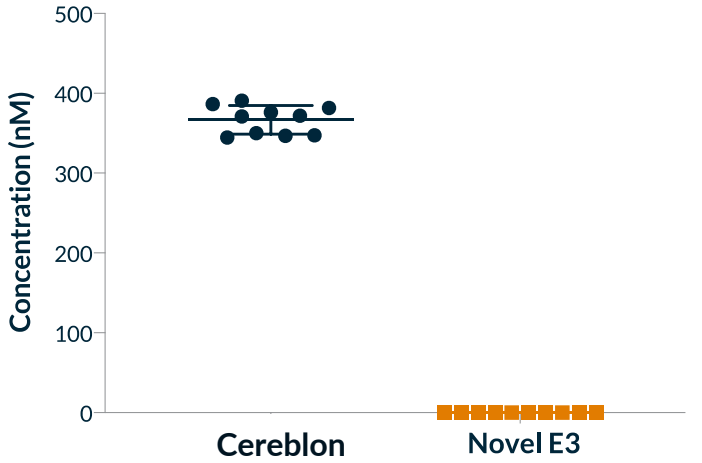
Expanded E3 ligase toolbox

Understanding degradation (PK/PD) across tissue types

Proprietary Chemistry

- E3 Ligase Whole-Body Atlas queried to identify a tissue sparing E3 ligase based on target protein unwanted pharmacology (i.e. bone marrow for a particular target of interest)
- A bone marrow sparing E3 ligase identified
- Screening and optimization lead to a novel binder to a previously unliganded E3 ligase (E3 ligase binders toolbox)
- A novel degrader based on a bone marrow sparing E3 ligase demonstrated target degradation

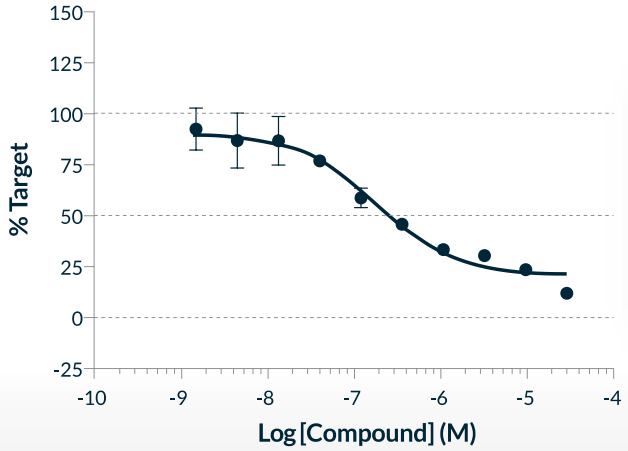
This E3 Ligase is Not Expressed in Bone Marrow



Bone marrow: CD34+ from five donors

Ligand Identification

TPD with Bone Marrow Sparing Novel E3 Ligase



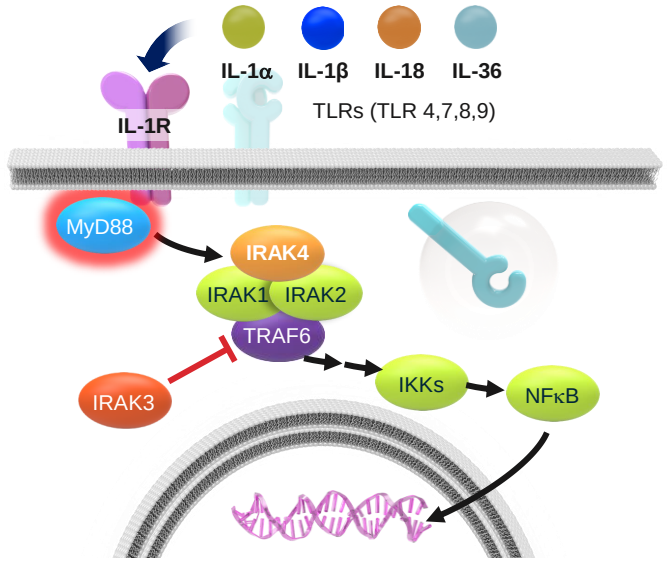
Target	Target Protein
DC_{50} (nM)	206
Dmax (%)	88

Kymera Drug Development Principles

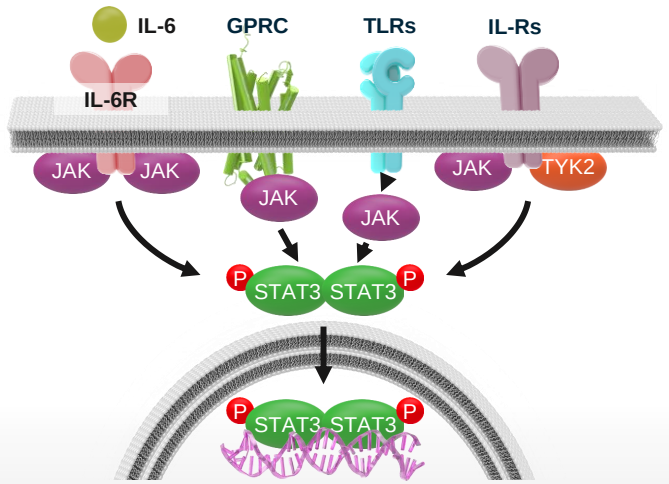
Initial focus on pathways that have been clinically and commercially validated with undrugged nodes

- **Unmet Medical Need**
- **Validated Biology**
- **Undrugged Node**
- **Precision Medicine Approach**

IL-1R/TLR Pathway



JAK-STAT Pathway



Clinical Pathway Validation

IL1-Rα/IL-1β : Rheumatologic Diseases
IL-1α: Atopic Dermatitis
IL-1β: CANTOS Data, Atherosclerosis, Lung Cancer
IL-18: Macrophage Activation Syndrome
IL-36: Generalized Pustular Psoriasis
IRAK4 SMI: Rheumatoid Arthritis

IL-6R: Rheumatoid Arthritis
IL-6: Multicentric Castleman's Disease
JAK1/2: Myelofibrosis
JAK3: Alopecia Areata
TYK2: Autoimmune Diseases
STAT3 ASO: AZD9150 in Oncology

IRAK4

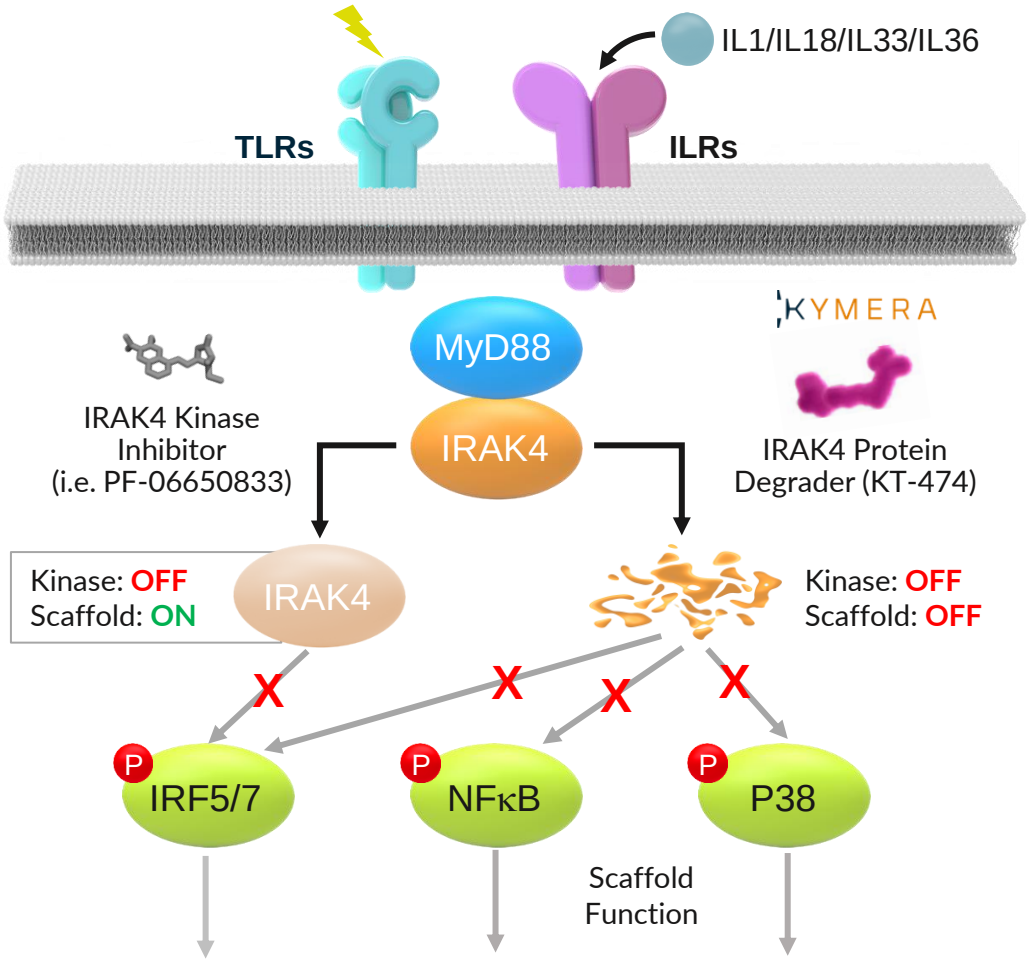
KYMERA

IRAK4 Biology and Degrader Rationale

- IRAK4 is a key component of the myddosome protein complex
- Myddosome is involved in innate immunity that mediates signals through IL-1R and TLRs
- IL-1R/TLR signaling through the myddosome complex is dependent on IRAK4 kinase and scaffolding functions
- Believe degrading IRAK4 can provide a single oral small molecule solution to many diseases impacted by this pathway
- Sanofi collaboration on development of degraders targeting IRAK4 outside oncology and immuno-oncology

Indications/Expected Timeline

AD, HS, RA, others
Phase 1 SAD initiation: 1Q 2021 ✔
Phase 1 MAD enrollment: 2H 2021*
Phase 1 proof-of-biology in healthy volunteers: 4Q 2021



IFNα/β, Inflammatory Cytokines (IL-6, TNF) & Mediators

* Initiation of MAD portion of Phase 1 trial is on partial clinical hold pending FDA review of interim SAD data.

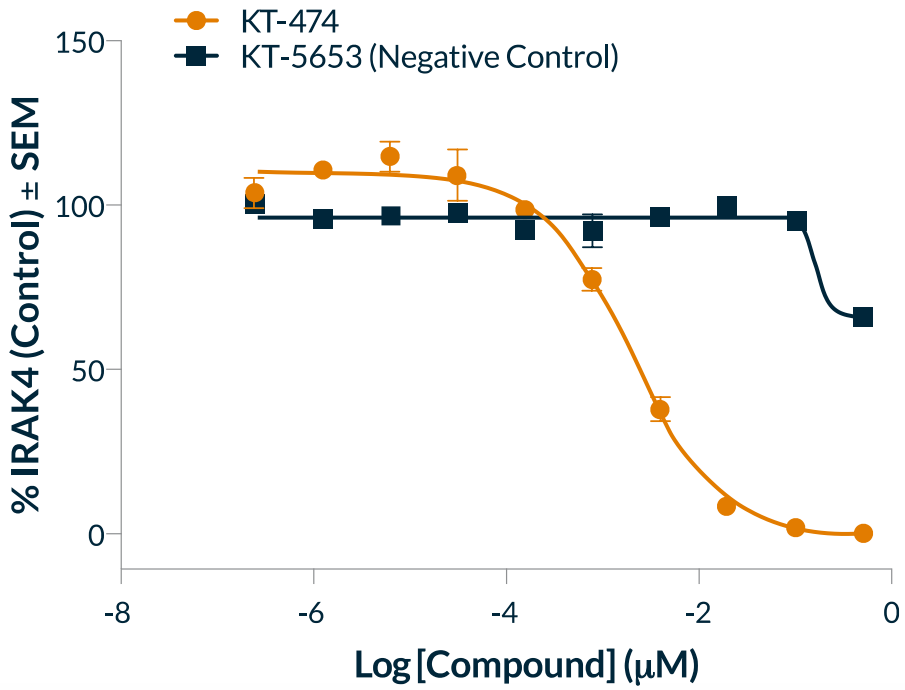
KT-474 Opportunity

Immune-inflammatory disorders collectively impacting millions of patients in the U.S.

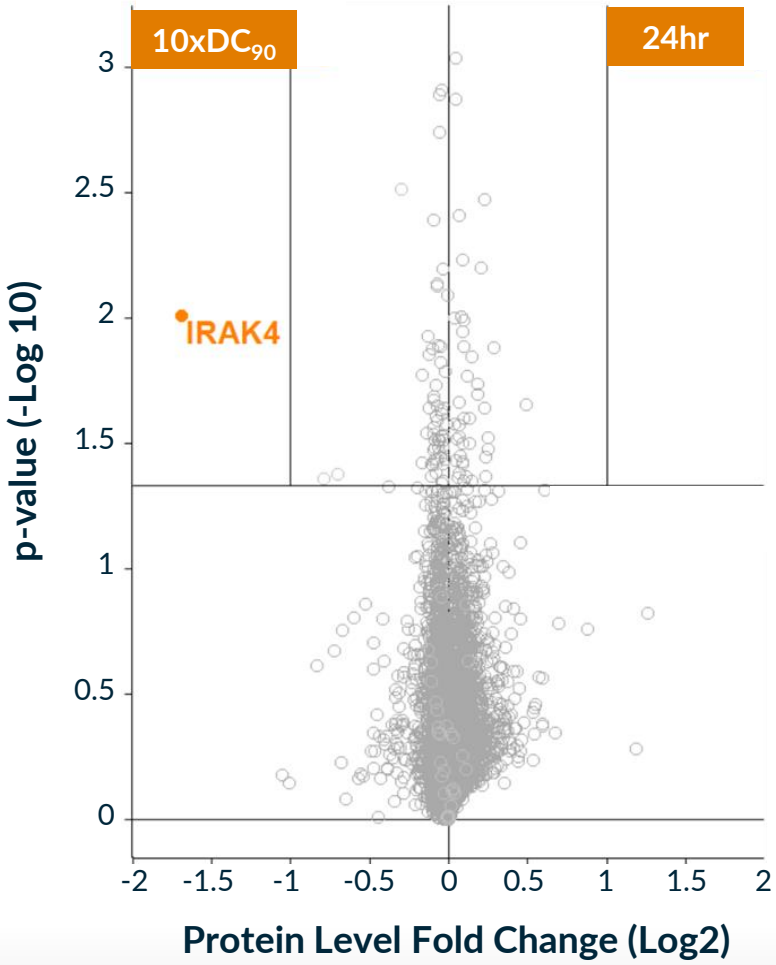
	Total Prevalence (U.S.)	
Hidradenitis Suppurativa (HS)	>325K	• Chronic and debilitating inflammatory skin disease • **~25%** of patients with moderate-to-severe disease • Adalimumab (anti-TNF antibody) is approved, which provides some benefit to **~50% of patients** with moderate-to-severe disease
Atopic Dermatitis (AD)	>11.0M	• Chronic, pruritic **inflammatory skin disease** • Large unmet need for safe and effective oral agents for patients with AD
Rheumatoid Arthritis (RA)	>1.3M	• Chronic, systemic **autoimmune disease** that can cause irreversible joint damage • Multiple therapies targeting the **IL-1R/TLR pathway** are approved
Additional Opportunities	→	• Immune-inflammatory diseases impacted by **IL-1R/TLR pathway**

KT-474: Specific IRAK4 Degradation

Degradation in Human Monocytes



Selectivity in Human PBMC

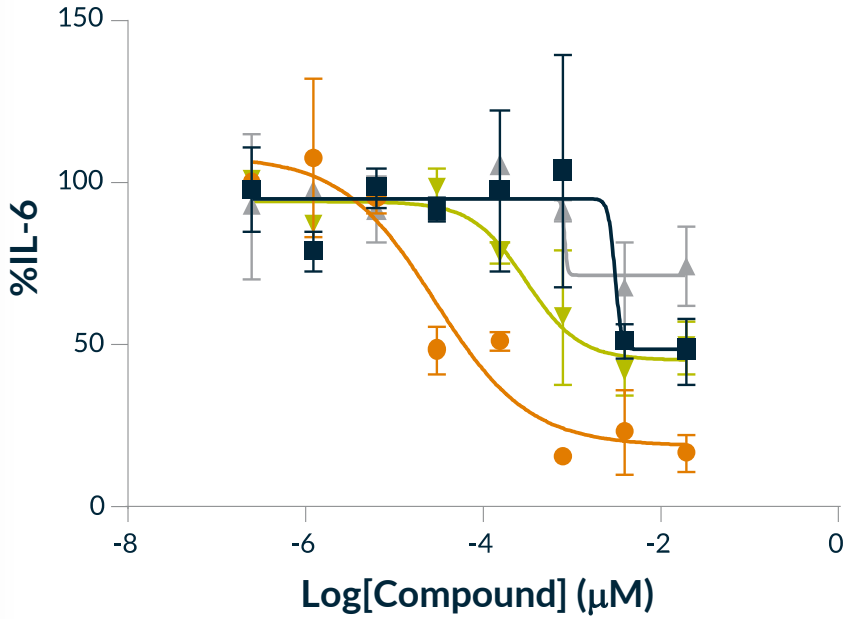


- Calculated DC_{50} of 2.1 nM and E3 ligase dependent degradation of IRAK4 in human immune cells
- IRAK4 was only protein of over 10,000 to be degraded by KT-474 in human immune cells at concentration 10-fold above the DC_{90}

IRAK4 Degradation Superior to Kinase Inhibition in Cytokine Production

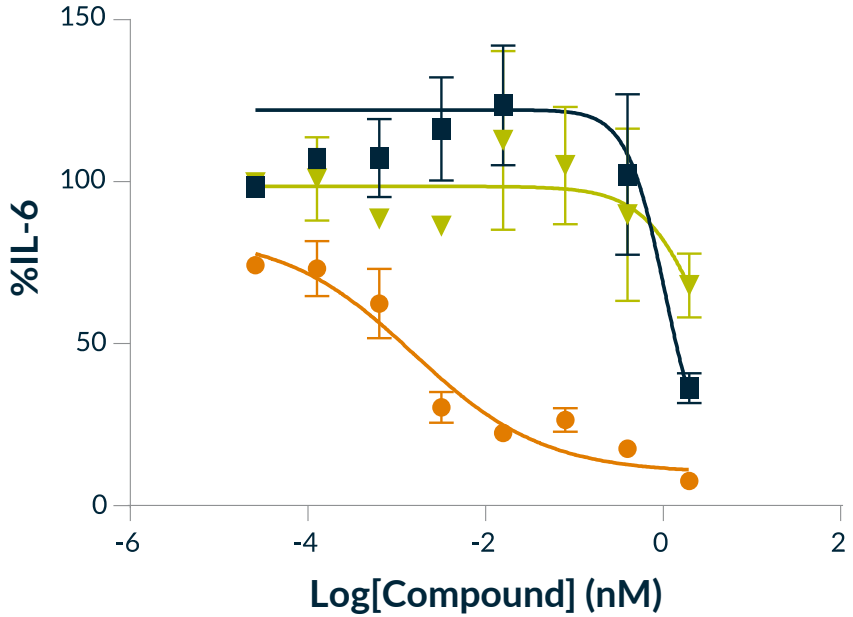
- Functional activity of KT-474 assessed by measuring pro-inflammatory cytokine levels upon activation
- Cells pre-treated with KT-474, a negative control, and small molecule IRAK4 kinase inhibitors
- KT-474 better able to inhibit IL-6 under both LPS and LPS + IL-1B than clinically active IRAK4 SM kinase inhibitor PF-06550833

LPS → IL-6



Legend	Compound	IL-6 IC_{50} (nM)
	KT-474	3
	Negative control	335
	IRAK4 SMI *(PF-06550833)*	N/A
	IRAK4 SMI *(other)*	N/A

LPS + IL-1B → IL-6



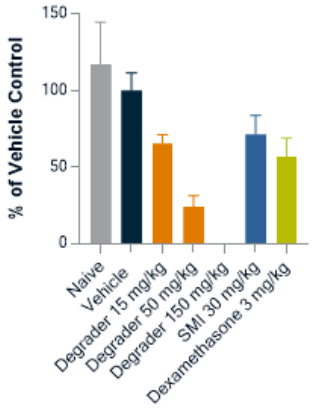
Legend	Compound	IL-6 IC_{50} (nM)
	IRAK4 Degrader	0.8
	Negative control	450
	IRAK4 SMI *(PF-06550833)*	N/A

KT-474 is Superior to IRAK4 Kinase Inhibitors Across Multiple Preclinical Immune-inflammatory *In Vivo* Models

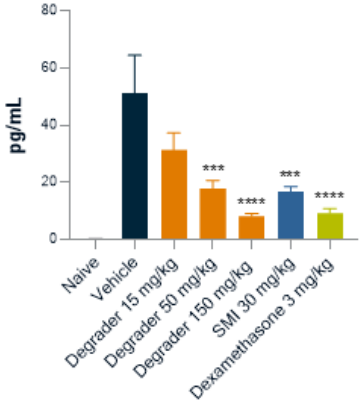
- KT-474's efficacy and superiority to IRAK4 small molecule inhibitors in models of IL-33, IL-36 and Th17-mediated inflammation
- In IL-33 and IL-36 models, KT-474 dose-dependently reduced IRAK4 levels in blood cells and inhibited skin inflammation and/or systemic as well as local cytokine production to the same extent as a potent corticosteroid (dexamethasone) and more potently than an IRAK4 small molecule inhibitor
- In a mouse model of Th17-mediated multiple sclerosis, KT-474 was superior to IRAK4 kinase inhibition and similar to FDA-approved fingolimod (FTY720) in significantly reducing clinical disease scores

rmIL-33 Intradermal Challenge Model

***In vivo* IRAK4 Degradation in Whole Blood**

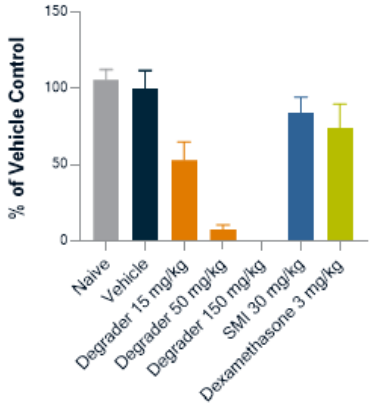


IL-5 in Ear Tissues

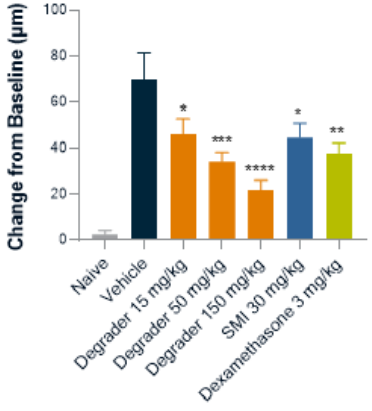


rhIL-36αβγ Intradermal Challenge Model

***In vivo* IRAK4 Degradation in Whole Blood**

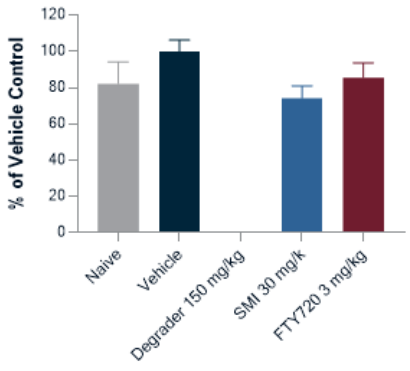


Ear Thickness

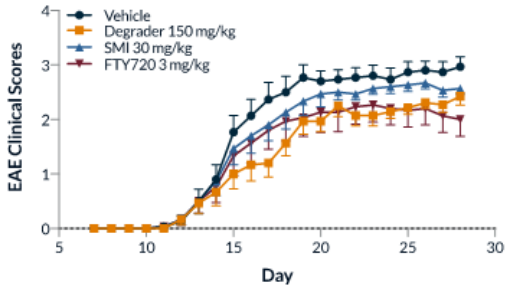


Th17-mediated Multiple Sclerosis Model

***In vivo* IRAK4 Degradation in Whole Blood**



MOG-EAR[1]

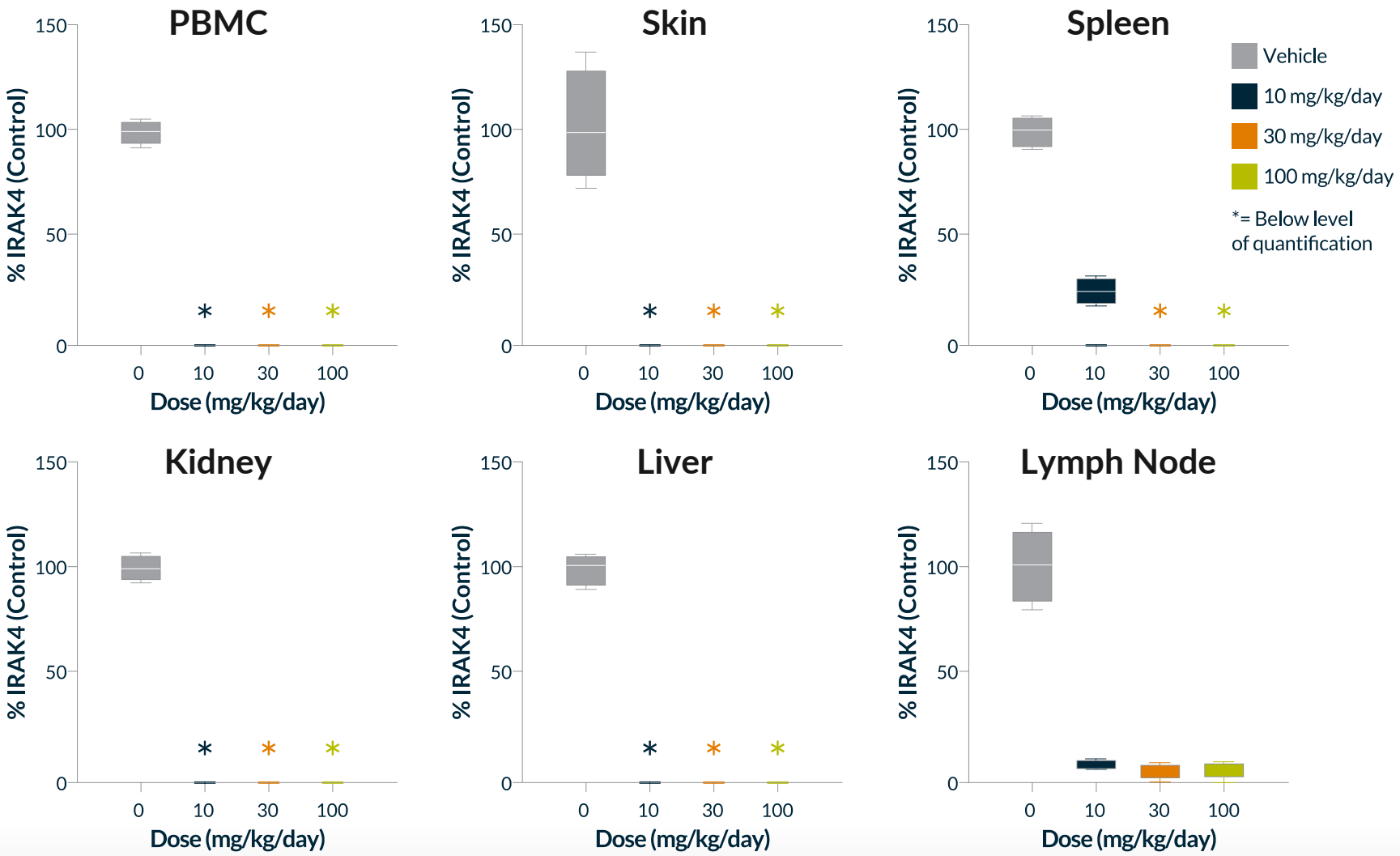


Treatment	Mean Max Score +/- SD	p value
Vehicle	3.40 +/- 0.54	-
Degrader 150 mg/kg	**2.69 +/- 0.52**	0.0018
SMI 30 mg/kg	3.07 +/- 0.42	0.0822
FTY720 3 mg/kg	**2.70 +/- 1.28**	0.0271

1. Myelin Oligodendrocyte Glycoprotein-induced Experimental Autoimmune Encephalomyelitis (MOG-EAR) Model

KT-474: Close to Complete IRAK4 Degradation and Well Tolerated in Preclinical Non-rodent Model

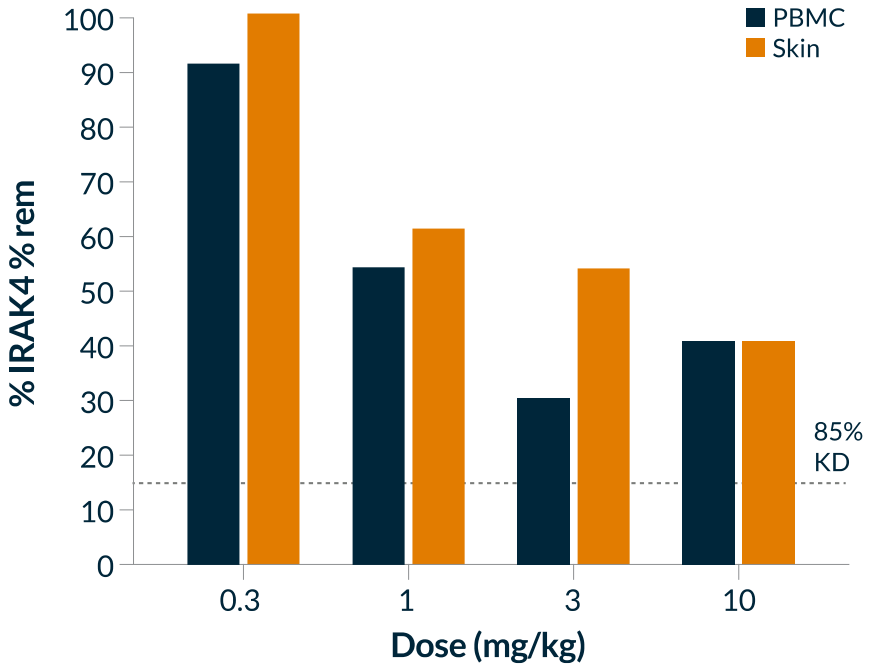
- Orally-administered KT-474 evaluated in a 14-day non-GLP tox and PKPD study in rodent and non-rodents (shown).
- Almost complete knockdown demonstrated across multiple tissues at multiple doses
- Compound well-tolerated at all doses up to 600 mg/kg for rodents and 100 mg/kg for non-rodents



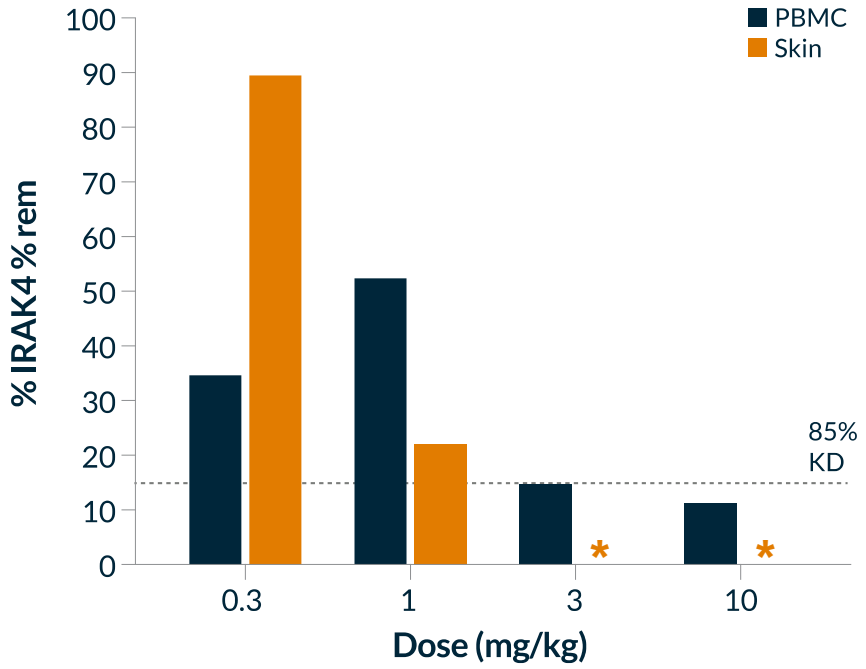
KT-474: Preclinical Single and Multiple Ascending Dose Characterization in Dog Model

- Orally-administered KT-474 achieves >85% knockdown of IRAK4 at Day 7 with repeat dosing in MAD study
- Multiple doses (MAD) lead to optimal degradation profile vs SAD upon reaching steady-state
- Consistency of IRAK4 knockdown observed across peripheral blood mononuclear cell (PBMC) and skin measurements

Single Ascending Dose (SAD)
IRAK4 Knockdown at Day 1



Multiple Ascending Dose (MAD)
IRAK4 Knockdown at Day 7



* = BLQ

KT-474 Phase 1 Trial to Establish Proof-of-Biology

Double-blind, placebo-controlled, Single Ascending Dose (SAD) and Multiple Ascending Dose (MAD) study

	SAD Portion	MAD Portion	
	Healthy Volunteers (HV)	Healthy Volunteers (HV)	Patient Cohort
Target Enrollment	• N = up to 56 adult HV	• N = up to 48 adult HV	• Up to 20 AD or HS patients • Moderate-severe disease

De-risking Milestones:

Oral Bioavailability and Proof-of-Mechanism

- Efficacious plasma exposures that are safe and well-tolerated
- Proof-of-mechanism with IRAK4 knockdown following single KT-474 dose
- Predictable PK/PD supporting oral daily dosing regimen

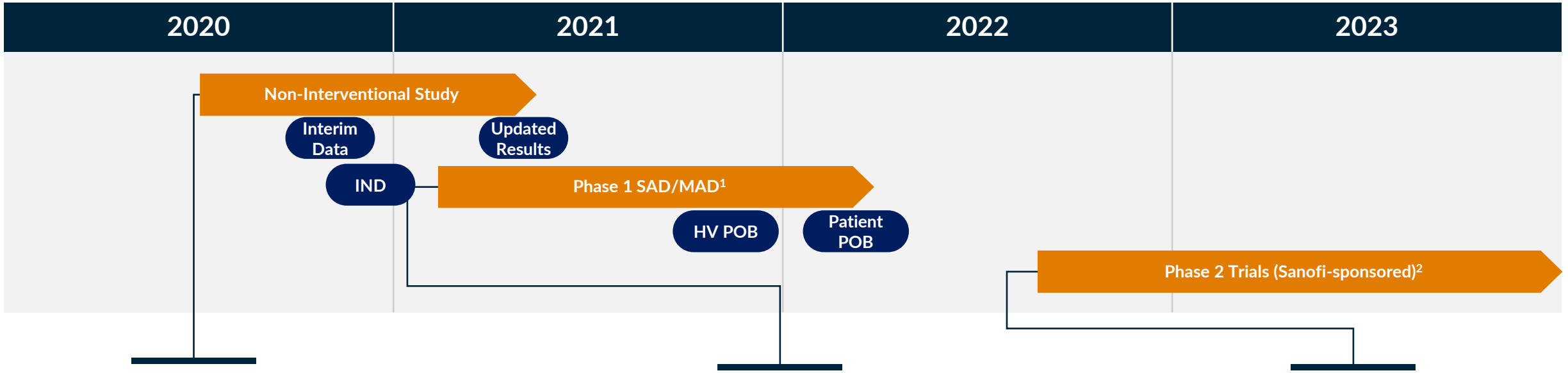
Optimal IRAK4 Reduction and Proof-of-Biology

- ≥85% IRAK4 knockdown in skin and blood with daily dosing x 14 days that is safe and well-tolerated
- Proof-of-biology with systemic anti-inflammatory effect: reduction in plasma hsCRP and inhibition of whole blood *ex vivo* response to TLR agonists and IL-1β
- Establishment of maximum effective dose

Clinical Proof-of-Concept in Patients

- ≥85% IRAK4 knockdown in diseased skin and blood
- Anti-inflammatory effect in diseased skin and reduction of plasma cytokines and hsCRP
- Confirmation of dose for subsequent Phase 2 studies

KT-474 Development Plan



- Evaluating 40 patients (HS n=30; AD n=10)
- Biomarker endpoints in blood and skin: IRAK4, cytokines, acute phase reactants
- Interim data presented Oct 2020
- **Updated data presented in May 2021**

- **Dosing in SAD portion initiated 1Q21**
- SAD and MAD studies in healthy volunteers (HV) and AD/HS patients
- Primary endpoint is safety
- Secondary endpoints to establish proof-of-biology (POB)
- **POB to be presented in 4Q21**

- Randomized, placebo-controlled trials in patients in potential indications such as AD, HS, RA, others

1. Initiation of MAD portion of Phase 1 trial is on partial clinical hold pending FDA review of interim SAD data.
2. Estimated clinical development timeline.


IRAKIMiD
KYMERA

IRAKIMiD

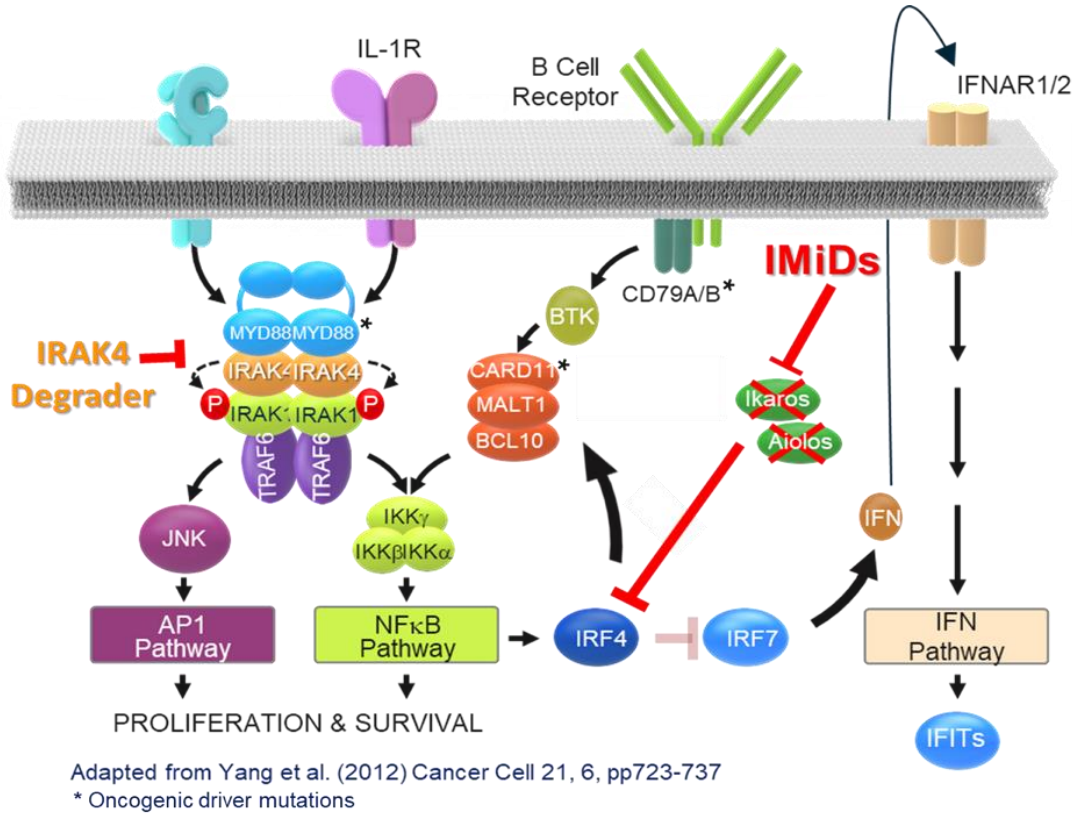
A combo in a single molecule

- MYD88 mutation drives differentiation and proliferation in ~25% of diffuse large B cell lymphoma (DLBCL)
- IMiDs downregulate IRF4, increasing IFN signaling and further suppressing NFkB activation and show activity in lymphoma
- Inhibiting both MYD88 and IRF4-dependent NFkB and activating IFN signaling drive cell death in MYD88-mutant lymphomas and leads to full and durable responses *in vivo*
- Combining two therapeutically relevant pathways in a single molecule has the potential to be first single agent targeted therapy agent in lymphoma (MYD-88 mut)

Indications/Expected Timeline

MYD88-mutant DLBCL
Current: KT-413 in IND-enabling activities
IND/Phase 1 initiation: 2H 2021
Phase 1 proof-of-biology in patients: 2022

Pathway



Adapted from Yang et al. (2012) Cancer Cell 21, 6, pp723-737
* Oncogenic driver mutations

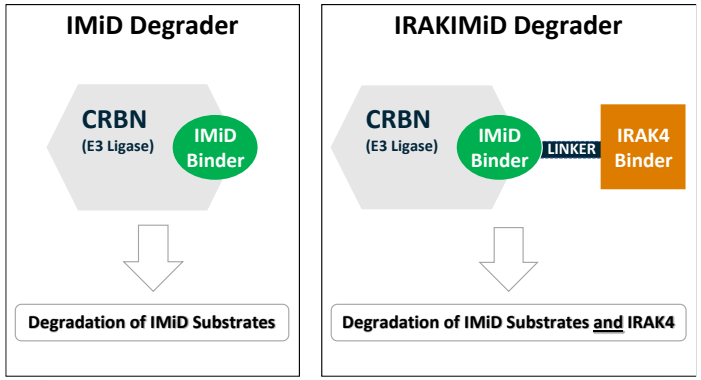
IRAK4 + Ikaros/Aiolos

KT-413 Opportunity

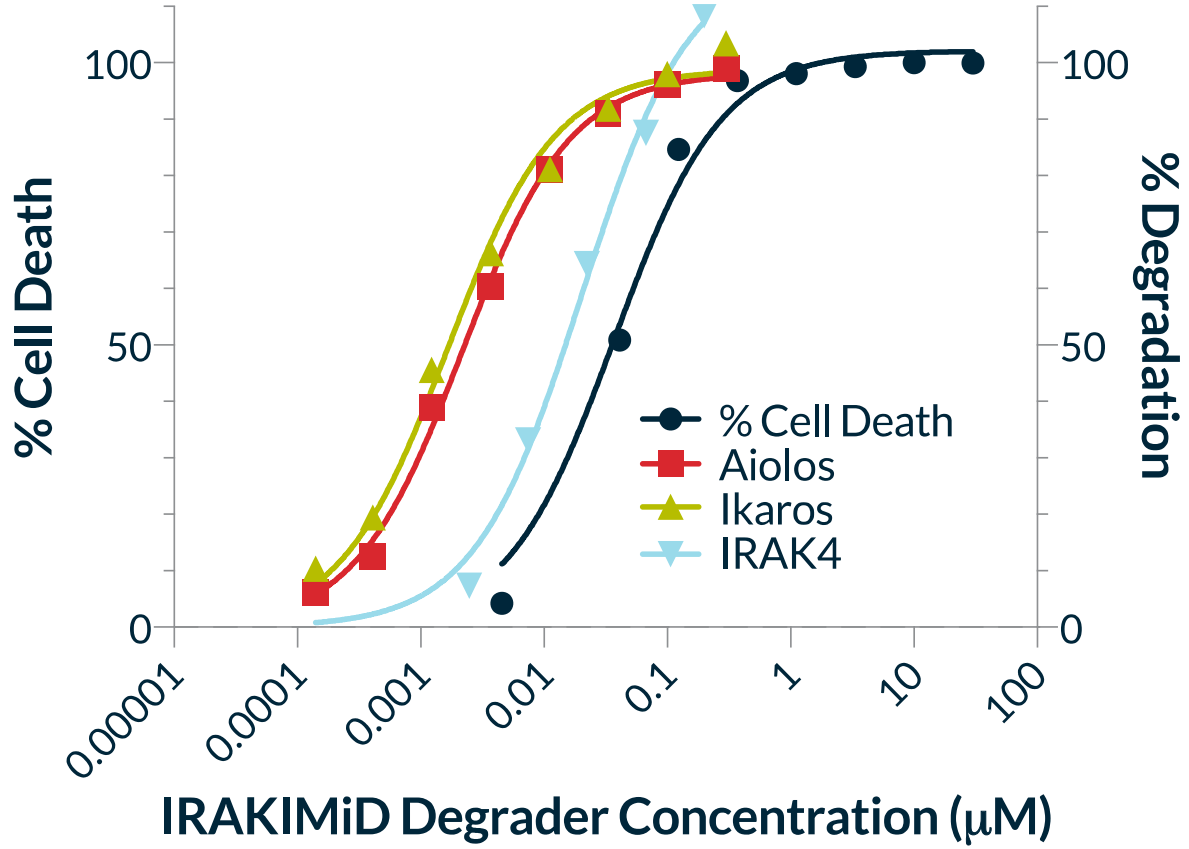
Potential to be first precision medicine in DLBCL to target a genetically defined population (MYD88-mut)

	Patient Impact (U.S.)	
MYD88-mutant DLBCL	**>7.0k** per year	• MYD88 is mutated in at least 25% of DLBCL patients, the most common subtype of non-Hodgkin's lymphoma • Front-line treatment includes **R-CHOP** (chemo/rituximab) • DLBCL **5-year survival rate is ~64%**, and MYD88 mutations in DLBCL are associated with poorer survival following frontline R-CHOP chemotherapy
Other MYD88-mutant B cell Lymphomas	**>1.0k** per year	• MYD88 is mutated in approximately 90% of **Waldenström's macroglobulinemia** cases and 70% of primary central nervous system lymphoma
Additional Cancers	→	• **IL1R/TLR/NFκB**-driven cancers, AML & MDS subsets with long isoform of IRAK4 (IRAK4L)

Degradation of IRAK4, Ikaros and Aiolos Correlates to Cell Killing



- IRAK4, Ikaros and Aiolos degradation measured in MYD-88-mutated OCI-Ly10 cells after 24 h of drug exposure
  - *IRAK4 DC_{50} = 4 nM*
  - *Ikaros/Aiolos DC_{50} = 2/2 nM*
- Degradation correlates with cell killing effects
  - *IC_{50} = 31 nM*



KT-413: Selective for MYD88 Tumors Irrespective of Co-mutations

- KT-413 IRAKIMiD DC is a selective and efficient degrader of both IRAK4 and the IMiD substrates
  - *IRAK4 DC_{50} = 8 nM*
  - *Ikaros/Aiolos DC_{50} = 2 nM*
- Degradation leads to cell viability effects **preferentially in MYD88-mutant lines irrespective of other mutational status**
- Data support potential for broadly targeting tumors harboring MYD88 mutations

Substrate	DC_{50} nM)
IRAK4	8
Ikaros/Aiolos	2/2

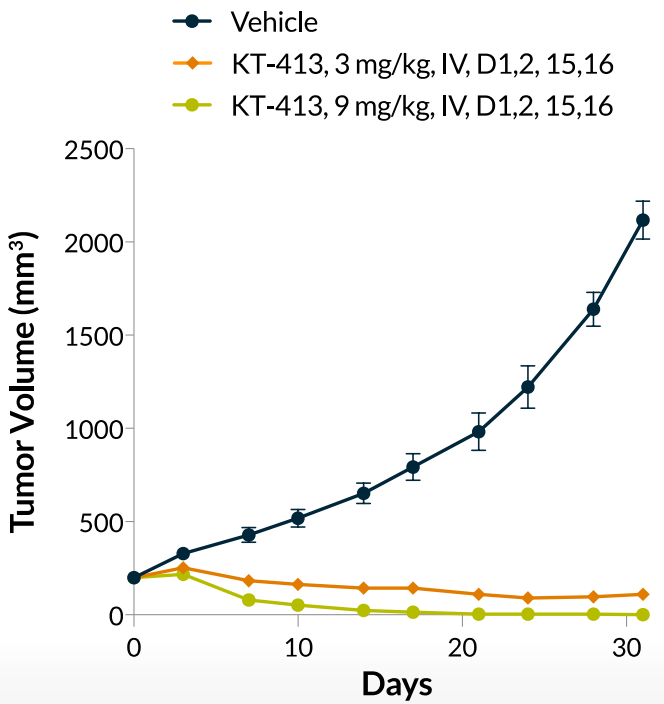
MYD88	Cell Line	Co-mutations	Cell (IC_{50} nM)
mut	OCI-LY10 CTG IC_{50} (nM)	CD79A	7
mut	SU-DHL2 CTG IC_{50} (nM)	TNFAIP3, IRF4, BCL6	14
mut	TMD8 CTG IC_{50} (nM)	CD79A, IRF4	29
Wild type	OCI-LY19 CTG IC_{50} (nM)	None	3,400
Wild type	U2932 CTG IC_{50} (nM)	BCL6	2,600

PK/PD in NHP is Consistent with Exposure and PD Associated with Efficacy

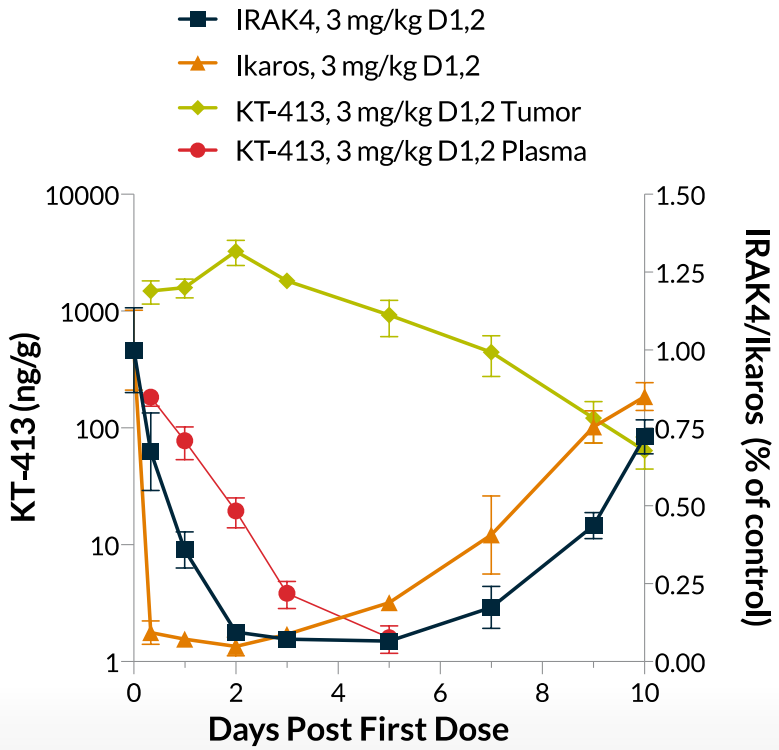
- Efficacy in OCI-Ly10 associated with >75% degradation in IRAK4 and IMiD substrates for >72h on intermittent (Q2W) dosing
- NHP doses on QW and Q2W dosing is associated with almost complete degradation of IRAK4 and IMiD substrates 3 days post dose

Xenograft Efficacy

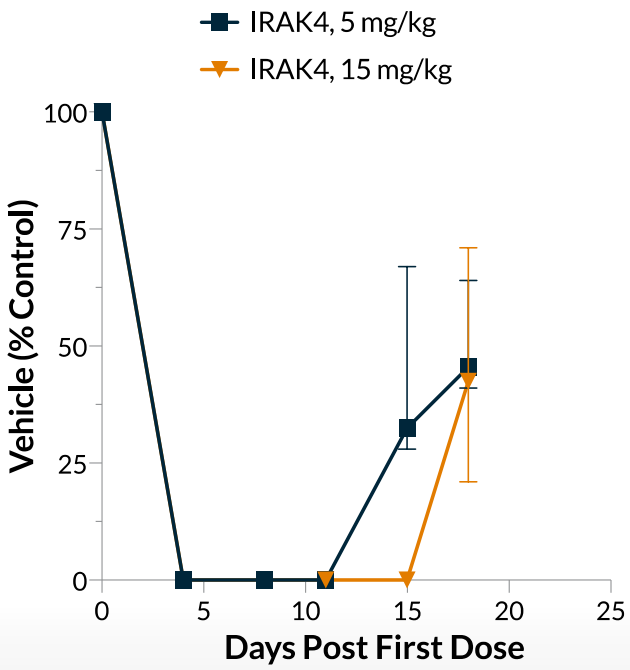
OCI-Ly10



KT-413 IV PK/PD in OCI-Ly10



PBMC PK/PD IRAK4 D1,2,8,9 Schedule



KT-413 Shows Regressions in MYD88^{MT} Patient-Derived Xenograft Models

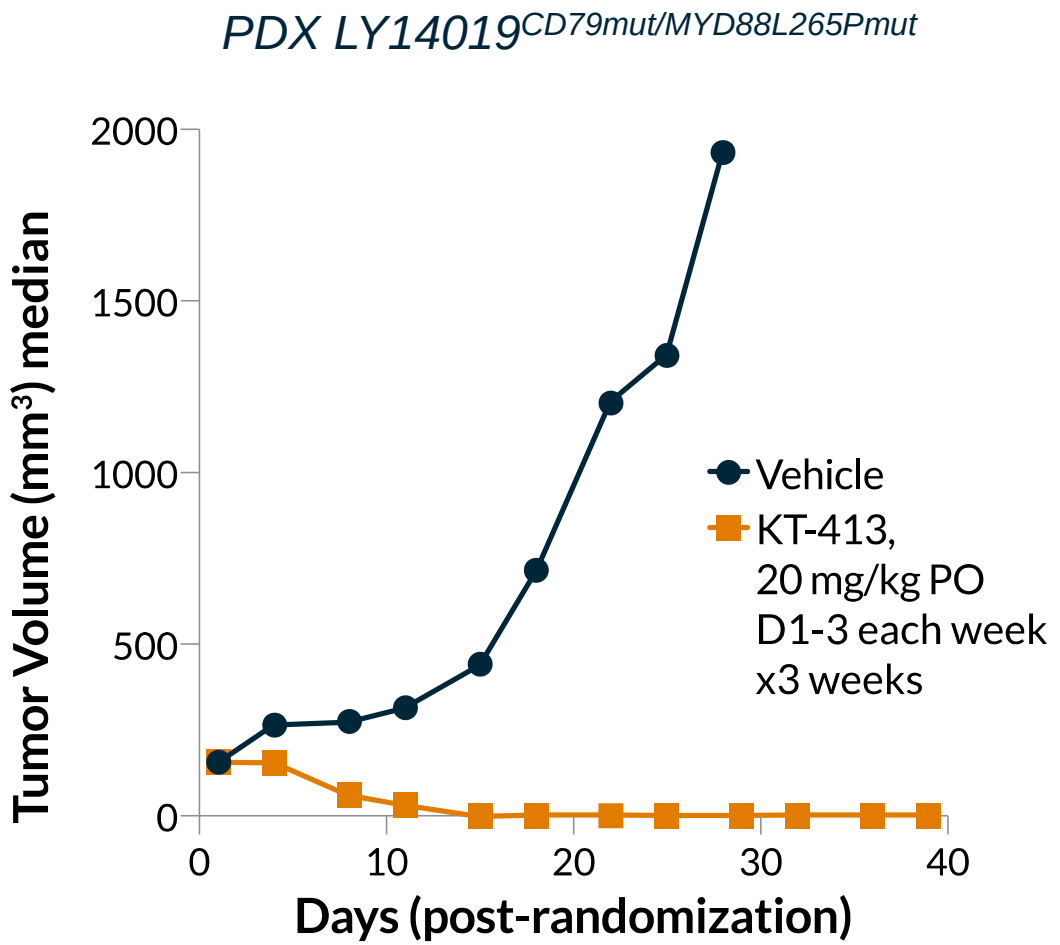
Model	MYD88	CD79B	TNFAIP3	Other	KT-413 (%TGI)
LY14019	L265P	MT	MT		**100**
LY2264	L265P	MT		IRF4	**100**
LY2298	L265P	MT		BCL2/BCL6	**90**
LY12699	L265P	MT			**87**
LY2345	WT		MT		**70**
LY2301	WT				**30**
LY0257	L265P			BCL2/BCL6/IKZF3	**0**

KT-413 dosed orally shows strong tumor growth inhibition (>85% TGI) in 4/5 MYD88-Mutated DLBCL PDX Models

- Activity is observed regardless of co-mutations that activate NFkB and IRF4 pathways
- The non-responsive MYD88^{MT} model LY0257 harbors a mutation in Aiolos and is reported to be insensitive to lenalidomide
- The functional consequence of Aiolos mutations in IRAKIMiD and IMiD response is being investigated

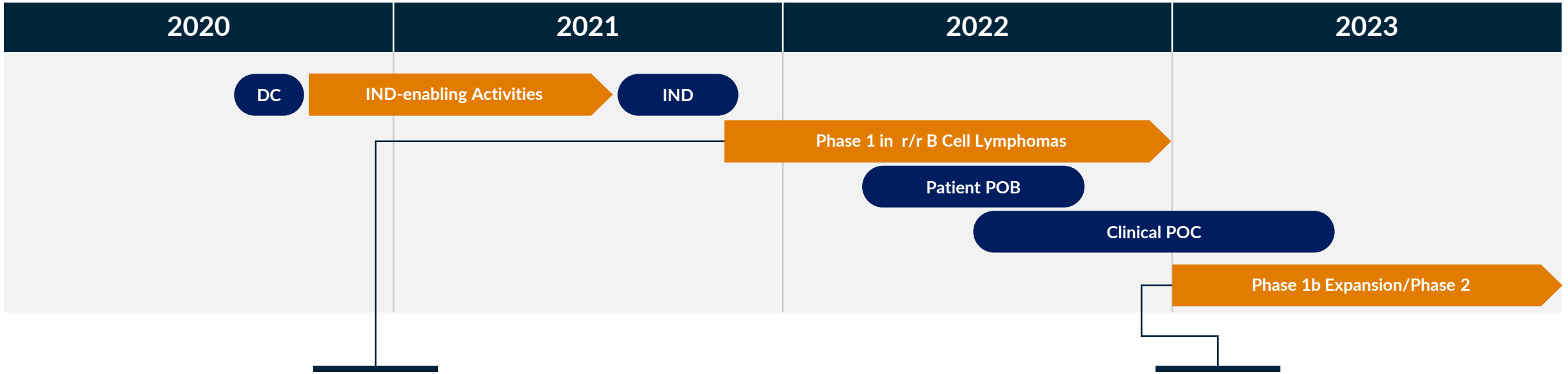
Some level of tumor growth inhibition observed in MYD88-WT PDX

- May be consistent with IMiD activity of KT-413



PDX models run at Crown Biosciences

KT-413 Development Plan



- **Multi-center Phase 1 dose escalation study (US sites) start in 2H21**
- Relapsed/refractory B cell lymphomas, including MYD88-mutant DLBCL
- Objectives include safety, tolerability, PK and PD (proof-of-biology) and preliminary clinical activity
- Clinical and biomarker endpoints
- **POB to be presented in 2022**

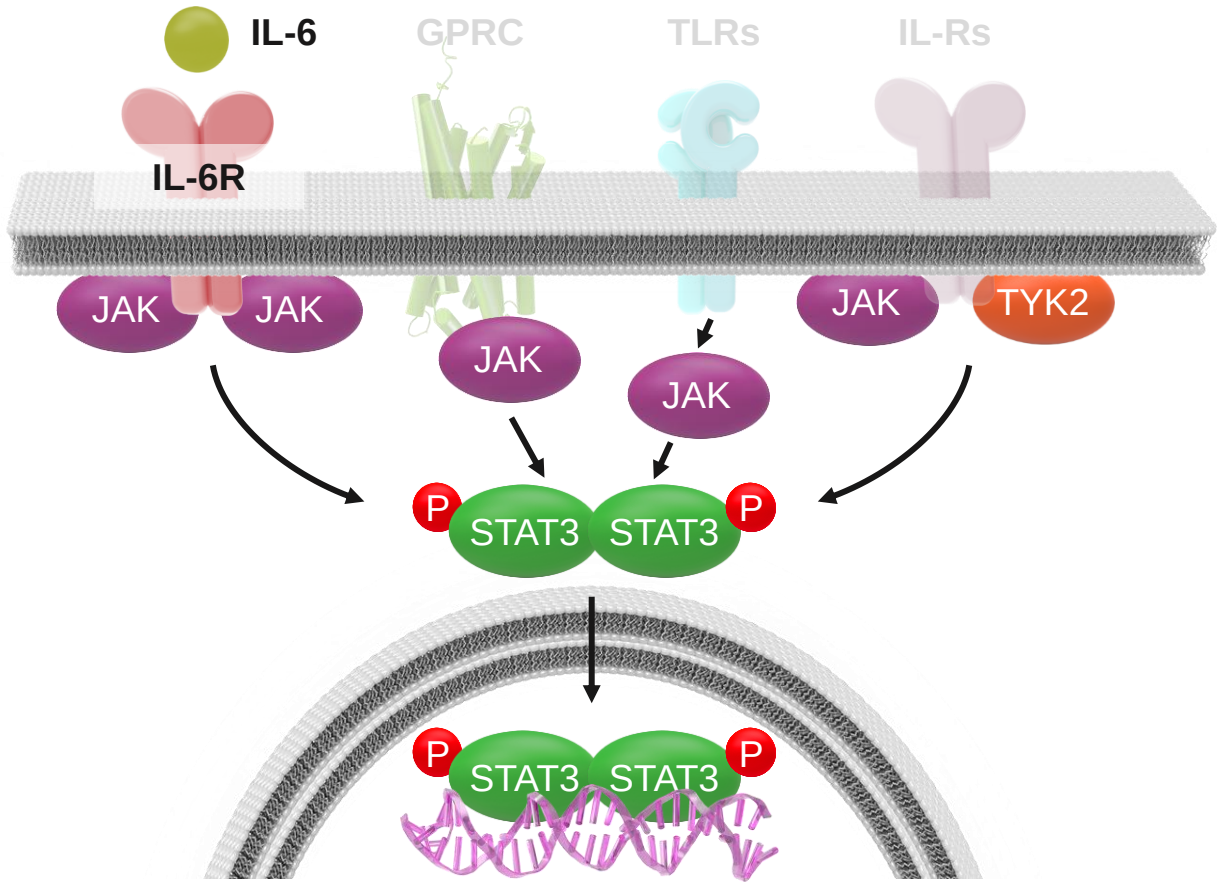
- Phase 1b expansion cohorts in DLBCL (MYD88-mut and –wt) and other MYD88-mut lymphomas, including Waldenstrom's macroglobulinemia and primary central nervous system lymphoma
- Objectives include safety, tolerability and clinical activity of monotherapy and select combinations
- Clinical and biomarker endpoints
- Potential expansion in other indications

STAT3

KYMERA

STAT3 Biology and Degrader Rationale

- STAT3 is a traditionally largely undrugged transcription factor activated through cytokine and growth factor receptors via JAKs and non-JAKs mediated mechanisms
- High degree of validation of JAK-STAT pathway in oncology and immuno-oncology supported also by numerous publications
- STAT3 plays a role in tumor biology, evasion of immune surveillance and inflammation/fibrosis
- No known drugs specifically affect STAT3 broadly across all relevant cell types



Indications/Expected Timeline

Hematological Malignancies/Solid Tumors and Autoimmune/Fibrosis

Nomination of development candidate: 1Q 2021 ✓

IND/Phase 1 initiation: 4Q 2021

Phase 1 proof-of-biology in patients: 2022

STAT3 Opportunity in Oncology & Autoimmunity

First-in-class opportunity to address STAT3-driven pathology across large and diverse indications

Patient Impact (U.S.)

Cancer

>5.0k per year
Peripheral T-cell Lymphoma

>2.0k per year
Cutaneous T-cell Lymphoma

>200.0k per year
NSCLC

I/I Fibrosis

>40.0k
Systemic Sclerosis

>11.0M
Atopic Dermatitis

>40.0k
Idiopathic Pulmonary Fibrosis

Liquid Tumors

Genetically-defined STAT3 mutation and/or hyperactivation
PTCL, CTCL, T-LGL leukemia

STAT3 activation and dependency
DLBCL, AML, multiple myeloma

Solid Tumors

Cell Intrinsic: STAT3 role in EMT/TKI resistance
Combinations in TKI / chemotherapy resistant settings

Cell Extrinsic: STAT3 role in IO
T-cell infiltrated tumors. Combinations with immune-modulators

Autoimmune

STAT3 GOF syndrome
Genetically-defined population characterized by enteropathy, arthritis, dermatitis, lung disease

Immune-inflammatory
Systemic sclerosis, atopic dermatitis, rheumatoid arthritis, Crohn's disease/ulcerative colitis

Fibrosis

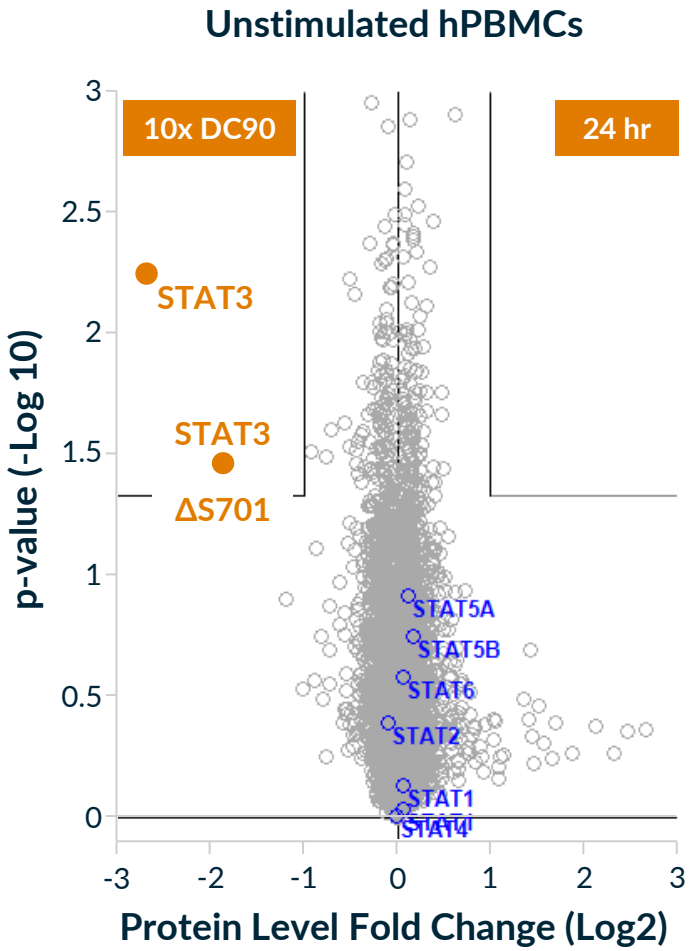
Chronic inflammation / fibrosis
Idiopathic pulmonary fibrosis, CKD/renal fibrosis

KT-333 Highly Specific Degradation of STAT3

CANCER
- Liquid Tumors
- Solid Tumors

I/I FIBROSIS
- Autoimmune
- Fibrosis

- Deep mass spectrometry-based proteomics to assess STAT3 specificity performed
- hPBMC and tumor cells (SU-DHL-1) treated with KT-333 degrader
- STAT3 was the only protein to be degraded with statistical significance
- Data demonstrate highly selective degradation profile



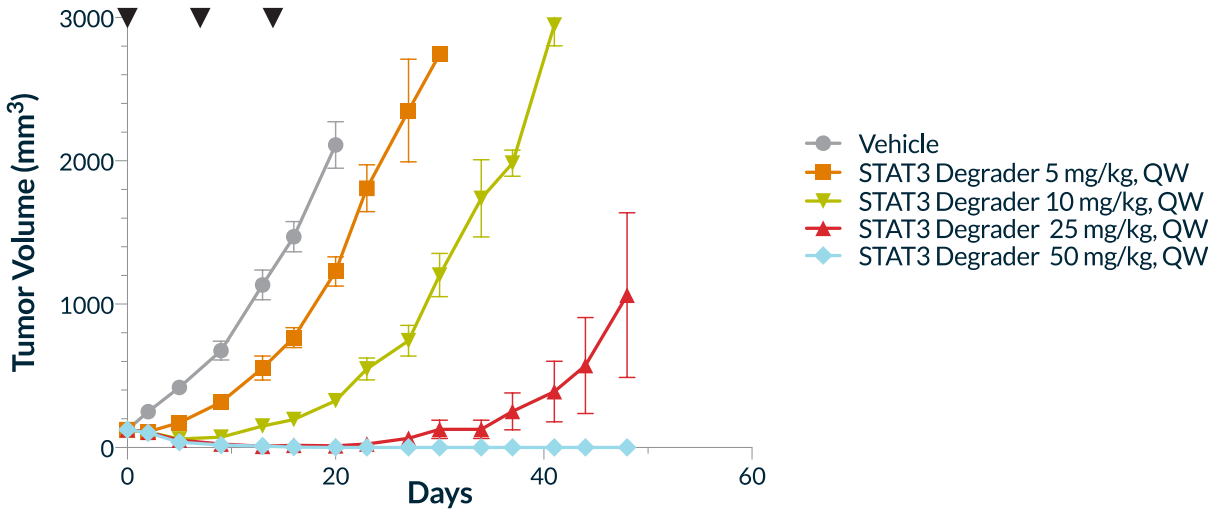
● STAT Family Members: **STAT1, STAT2, STAT3, STAT4, STAT5A, STAT5B, STAT6**

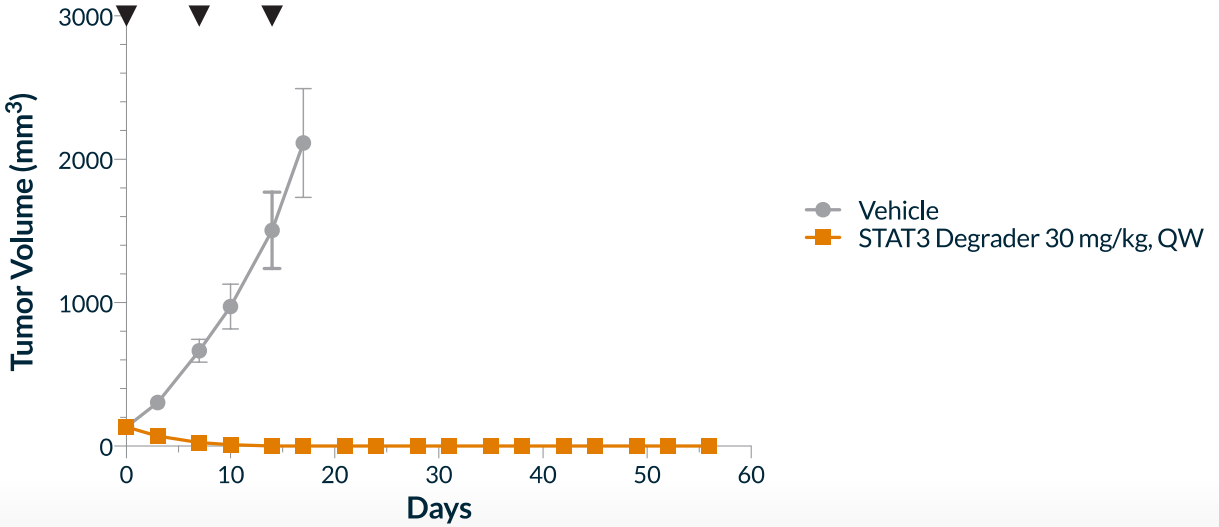
Full and Durable Regressions Across Multiple *in vivo* Preclinical Tumor Models

CANCER
- Liquid Tumors
- Solid Tumors

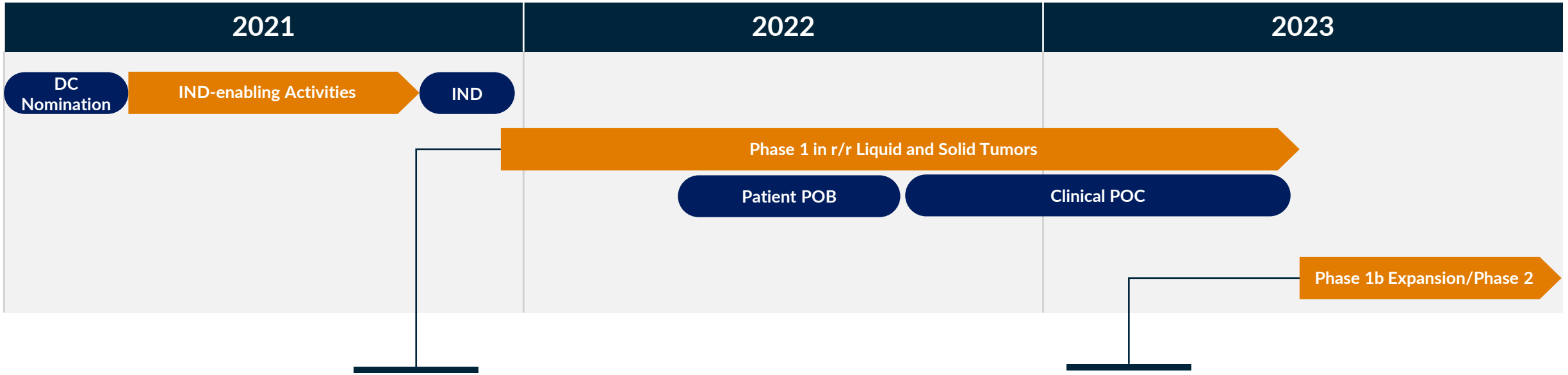
I/I FIBROSIS
- Autoimmune
- Fibrosis

- Mice bearing STAT3-dependent ALK+ ALCL SU-DHL-1 (above) and STAT3-driven ALK+ ALCL xenograft model SUP-M2 (below) tumors dosed with STAT3 degrader
- Dose and degradation dependent tumor growth inhibition observed with once-a-week IV dosing
- 30 mg/kg sufficient to drive full tumor regression that was durable for multiple weeks after the last dose





STAT3 Degrader Development Plan in Liquid & Solid Tumors



- **Multi-center Phase 1 dose escalation study start in 4Q21**
- Safety, tolerability, PK and PD (proof-of-biology) and preliminary clinical activity
- Clinical and biomarker endpoints
- **POB to be presented in 2022**

- Phase 1b expansion cohorts in STAT3-dependent liquid tumors
- Objectives include safety, tolerability and clinical activity of monotherapy and select combinations
- Separate Phase 2 in solid tumors

Near-Term Milestones Provide Significant Opportunity

Program	Compound	Indication(s)	Expected Upcoming Milestones
IRAK4	KT-474	AD, HS, RA, others	✓ Initiate SAD portion of Phase 1 trial in healthy volunteers (1Q 2021) ✓ Present updated Non-Interventional trial results (2Q21) ✓ Present KT-474 preclinical data vs. kinase inhibitors in immune-inflammatory preclinical models (2Q21) • Initiate enrollment in MAD portion of Phase 1 trial in HV, as well as AD and HS patients (2H21)* • Establish Phase 1 proof-of-biology in healthy volunteers (4Q 2021)
IRAKIMiD (IRAK4, Ikaros, Aiolos)	KT-413	$MYD88^{MT}$ DLBCL	✓ Presentation of KT-413 mechanism of action at the AACR Annual Meeting (2Q21) • Submit IND and, if cleared, initiate Phase 1 clinical trial in r/r B cell lymphomas (2H21) • Present additional KT-413 preclinical data and potential expansion strategies (2H21) • Establish Phase 1 proof-of-biology in patients (2022) • Establish Phase 1 initial clinical proof-of-concept in patients (2022)
STAT3	KT-333	Liquid & Solid Tumors	✓ Nominate development candidate for liquid & solid tumor indications (1Q21) • Present additional preclinical data in liquid & solid tumor indications (2021) • Submit IND, and if cleared, initiate Phase 1 clinical trial (4Q21) • Establish Phase 1 proof-of-biology in patients (2022) • Establish Phase 1 initial clinical proof-of-concept in patients (2022)
Discovery Programs & Platform			• Continue pipeline expansion by advancing early-stage discovery programs toward IND-enabling studies • Further expand Pegasus platform to generate novel degrader product candidates • Leverage Whole-Body Atlas to unlock new opportunities across broad therapeutic applications

● = Oncology ● = Immunology-Inflammation

* Initiation of MAD portion of Phase 1 trial is on partial clinical hold pending FDA review of interim SAD data.

THANK YOU

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