Bexobrutideg Investor Update

Investor Presentation
October 2025

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Nurix Is Advancing a Pipeline of Proprietary and Partnered Programs in Oncology and Inflammation & Immunology

Oncology

Program	Target	Modality	Therapeutic area	Discovery	IND-Enabling	Phase 1A	Phase 1B/2	Pivotal
Bexobrutideg (NX-5948)	BTK	Degrader	B-cell malignancies					
Zelebrudomide (NX-2127)	BTK-IKZF	Degrader	B-cell malignancies					
NX-1607	CBL-B	Inhibitor of degradation	Immuno-oncology					
BRAF degrader	Pan-mutant BRAF	Degrader	Solid tumors					
Multiple	Undisclosed	Degrader	Undisclosed					
Multiple	Undisclosed	Degrader	Undisclosed					GILEAD
Multiple	Undisclosed	DAC	Undisclosed					Pfizer

Inflammation & Immunology

Program	Target	Modality	Therapeutic area	Discovery	IND-Enabling	Phase 1A	Phase 1B	Phase 2/3
Bexobrutideg (NX-5948)	BTK	Degrader	Autoimmune cytopenia in CLL patients					
NX-0479 / GS-6791	IRAK4	Degrader	Rheumatoid arthritis and other inflammatory diseases					GILEAD
NX-3911	STAT6	Degrader	Type 2 inflammatory diseases					sanofi
Undisclosed	Undisclosed	Degrader	Inflammation / autoimmune					sanofi
Multiple	Undisclosed	DAC	Inflammation / autoimmune					

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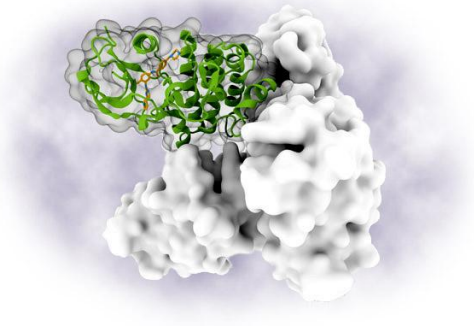
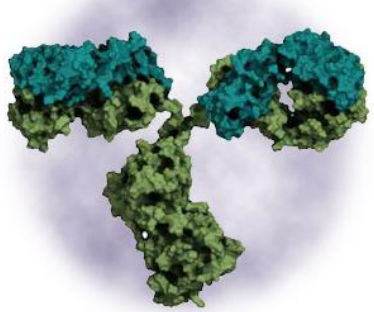
A Leader in the Next Frontier in Drug Development

Targeted protein degradation (TPD)

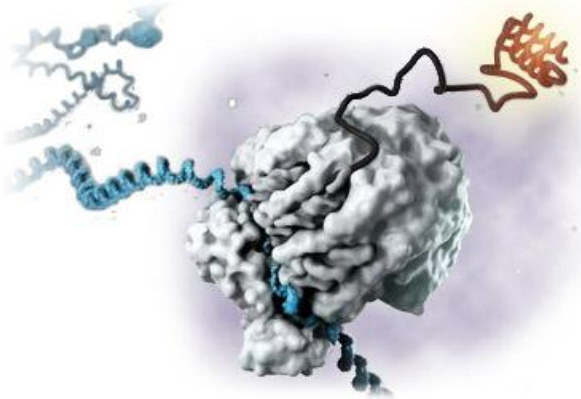
Evolution of new therapeutic modalities



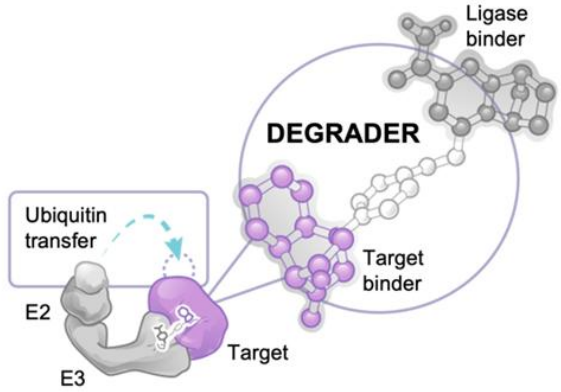
Small molecule inhibitors



Antibodies



Nucleic acid-based therapies
(Antisense, RNAi
Gene Therapy, CRISPR)



Targeted protein degraders

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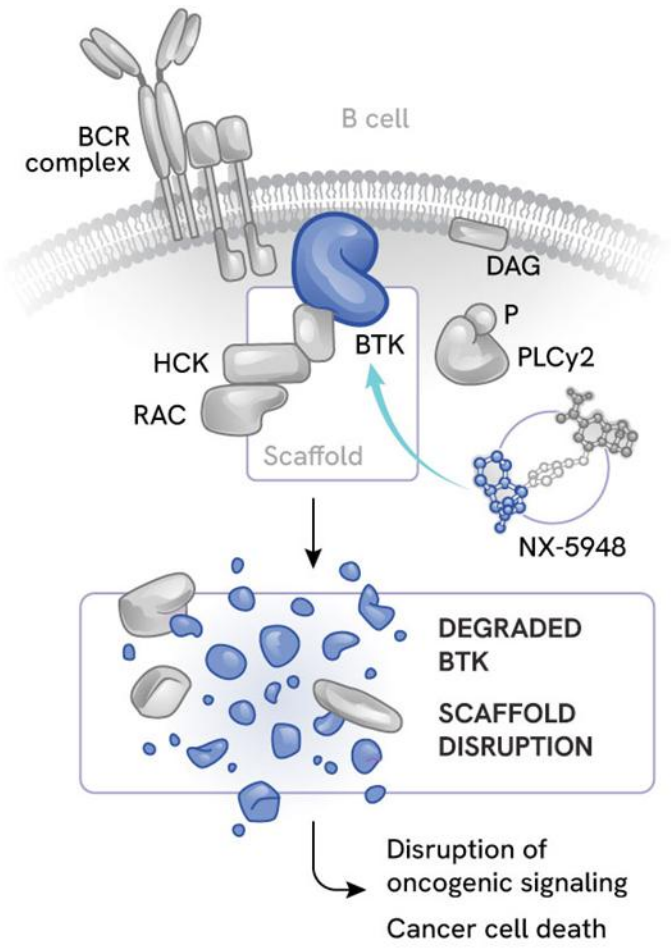
Positioned for Success – Key Program Updates

Advancing bexobrutideg as a potential best-in-class BTK degrader

- ✓ 600 mg dose selected per Project Optimus
  - ✓ Cleared to move ahead globally (FDA, MHRA, EMA)
- ✓ Pivotal Phase 2 trial initiated – DAYBreak CLL-201
- ✓ Confirmatory Phase 3 trial initiation planned for H1 2026
- ✓ New best-in-class *in vitro* potency data
- ✓ New proteomics highlighting best-in-class selectivity
- ✓ Next bexobrutideg clinical update at ASH 2025

Bexobrutideg – The First “deg” with a Potential Best-in-Class Profile

Novel MOA Against a Clinically and Commercially Proven Target



- ✓ Active against wildtype BTK and demonstrated ability to overcome treatment-emergent resistance mutations
- ✓ Addresses BTK scaffolding function unlike current BTK inhibitors
- ✓ Acts catalytically driving degradation at low free-plasma concentrations
- ✓ Crosses the blood brain barrier and demonstrated clinical activity in the CNS
- ✓ Demonstrated robust clinical activity in difficult to treat B-cell malignancies
- ✓ Has potential to address significant unmet needs in autoimmune and inflammatory disorders

***BTK**, Bruton's tyrosine kinase; **CNS**, central nervous system; **MOA**, mechanism of action.*

Agenda

Paula G. O'Connor, M.D.
Chief Medical Officer

Bexobrutideg Clinical Development Update

Gwenn Hansen, Ph.D.
Chief Scientific Officer

Best-in-Class BTK Degrader: Potency, Coverage, and Selectivity

Extending our leadership into I&I

Q&A to follow

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DAYBreak: Registrational Pathway for Bexobrutideg in R/R CLL

Paula G. O’Connor, M.D.
Chief Medical Officer

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Bexobrutideg 600 mg Once Daily Oral Dose Cleared by Global Regulators for Pivotal Monotherapy Trials in Relapsed/Refractory CLL

- **Highest dose tested in Phase 1 cleared by global regulators for pivotal monotherapy studies in r/r CLL**
  - U.S. Food and Drug Administration (FDA), in accordance with Project Optimus
  - U.K Medicines and Healthcare products Regulatory Authority (MHRA)
  - European Medicines Agency (EMA)
- **Global designations in CLL support regulatory interactions**
  - Fast Track Designation with FDA
  - PRIME designation with EMA
- **Pivotal Phase 2 trial underway**
  - First site activated in October 2025
- **Confirmatory Phase 3 trial initiation planned for H1 2026**
  - Key study start up activities underway

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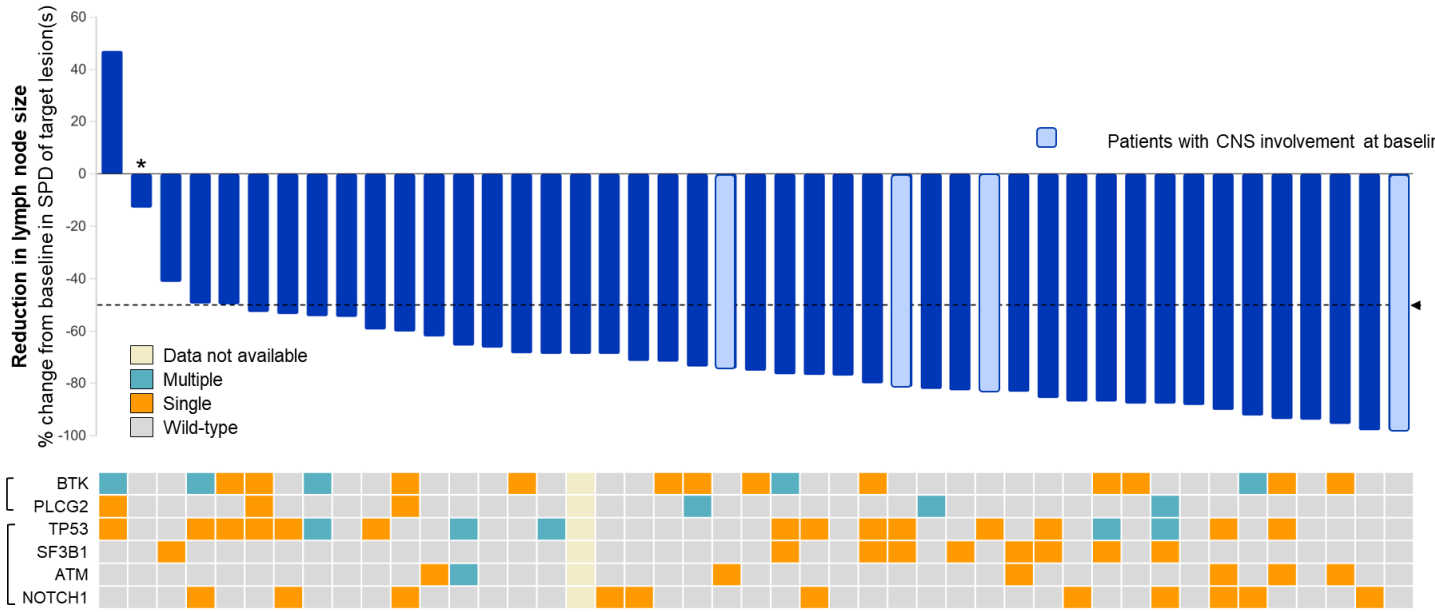
Bexobrutideg Has Consistently Demonstrated Strong Clinical Activity

Efficacy Data Across All Patients

CLL response-evaluable patients[a]	Response analysis (n=47)
Objective response rate (ORR),[b] % (95% CI)	80.9 (66.7–90.9)
Best response, n (%)	
Complete response (CR)	1 (2.1)
Partial response (PR)	37 (78.7)
PR with rebound lymphocytosis (PR-L)	0 (0.0)
Stable disease (SD)	7 (14.9)
Progressive disease (PD)	2 (4.3)
Median follow-up, months[c] (range)[d]	9.0 (1.6–26.1)

Efficacy Data Per Patient Regardless of Mutations or High Risk Cytogenetic Features

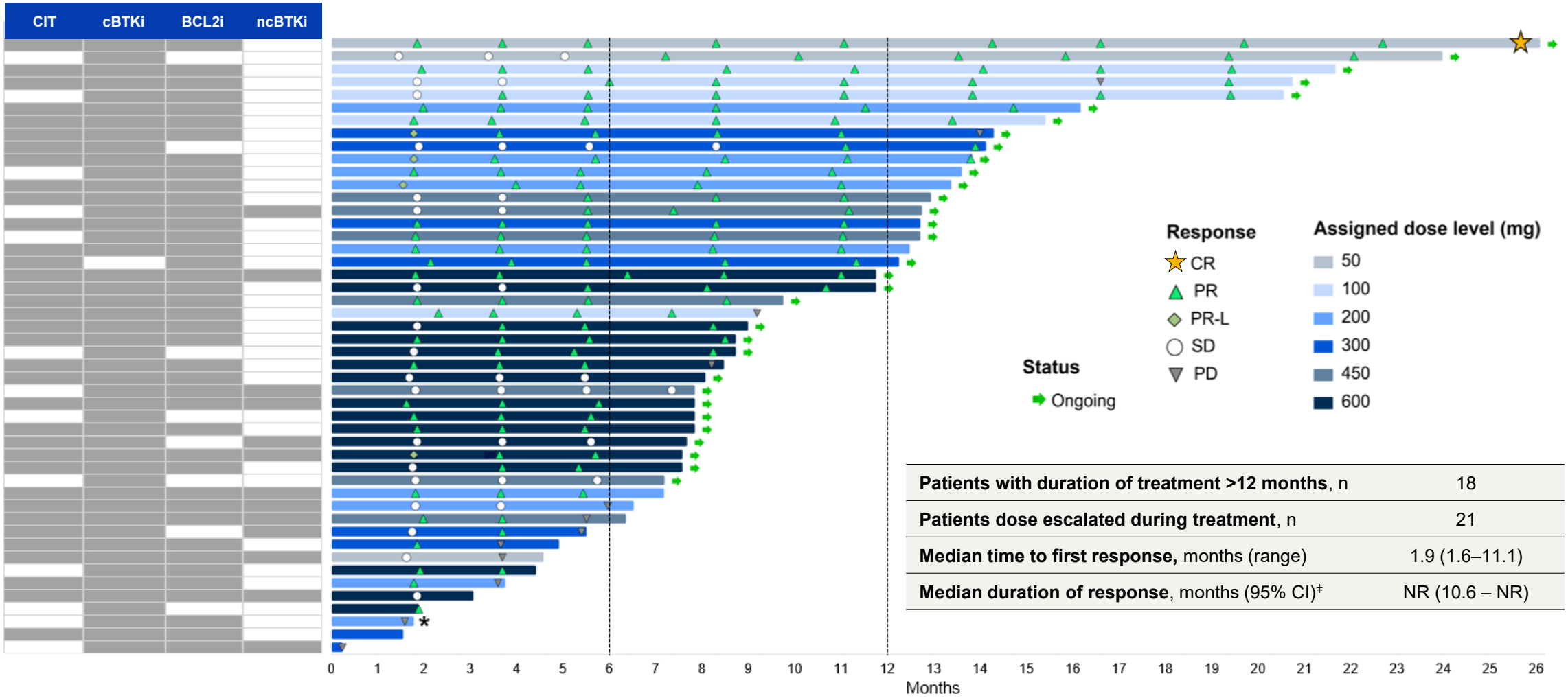


[a]Patients who were treated with bexobrutideg having ≥1 post-baseline disease assessment or documented clinical PD. [b]Objective response rate was evaluated using iwCLL criteria and included CR + PR + PR-L. [c]Kaplan-Meier estimate; [d]Observed values

***ATM**, Ataxia-telangiectasia mutated; **BTK**, Bruton's tyrosine kinase; **BTKi**, BTK inhibitor; **CLL**, chronic lymphocytic leukemia; **CNS**, central nervous system; **iwCLL**, International Workshop on CLL; **NOTCH1**, neurologic locus notch homolog protein 1; **PLCG2**, phospholipase C gamma 2; **SPD**, sum of products diameters*

Data cutoff: 12 Mar 2025

Bexobrutideg Demonstrates Durable Responses in Patients with Relapsed/Refractory CLL (n=48)



Patients with duration of treatment >12 months, n	18
Patients dose escalated during treatment, n	21
Median time to first response, months (range)	1.9 (1.6–11.1)
Median duration of response, months (95% CI)‡	NR (10.6 – NR)

**Patient with Richter's transformation to Hodgkin's on biopsy;* ‡Kaplan-Meier estimate

Data cutoff: 12 Mar 2025

***BCL2i**, BCL2 inhibitor; **BTKi**, BTK inhibitor; **cBTKi**, covalent BTKi; **CI**, confidence interval; **CIT**, chemo/chemo-immunotherapies; **CR**, complete response; **ncBTKi**, non-covalent BTKi; **NE**, not evaluable; **NR**, not reached; **PD**, progressive disease; **PR**, partial response; **PR-L**, PR with rebound lymphocytosis; **SD**, stable disease*

Bexobrutideg Safety Profile: Well Tolerated in Patients with Relapsed/Refractory CLL

TEAEs, n (%)	Patients with CLL/SLL (n=48)		
	Any grade	Grade ≥3	SAEs
Purpura/contusion[a]	22 (45.8)	–	–
Diarrhea	15 (31.3)	2 (4.2)	–
Fatigue[b]	15 (31.3)	–	–
Neutropenia[c]	14 (29.2)	11 (22.9)	–
Rash[d]	13 (27.1)	1 (2.1)	1 (2.1)
Petechiae	12 (25.0)	–	–
Headache	12 (25.0)	–	–
Thrombocytopenia[e]	11 (22.9)	1 (2.1)	–
Anemia	9 (18.8)	2 (4.2)	–
COVID-19[f]	9 (18.8)	–	–
Peripheral edema	9 (18.8)	–	–
Cough	8 (16.7)	–	–
Lower respiratory tract infection	7 (14.6)	1 (2.1)	1 (2.1)
Nausea	7 (14.6)	–	–
Pneumonia[g]	6 (12.5)	2 (4.2)	2 (4.2)
Arthralgia	6 (12.5)	–	–
Upper respiratory tract infection	5 (10.4)	–	–
Vomiting	5 (10.4)	1 (2.1)	–
Respiratory syncytial virus infection	2 (4.2)	1 (2.1)	2 (4.2)

- No dose-limiting toxicities
- No new atrial fibrillation
- No new ventricular arrhythmias
- No systemic fungal infections

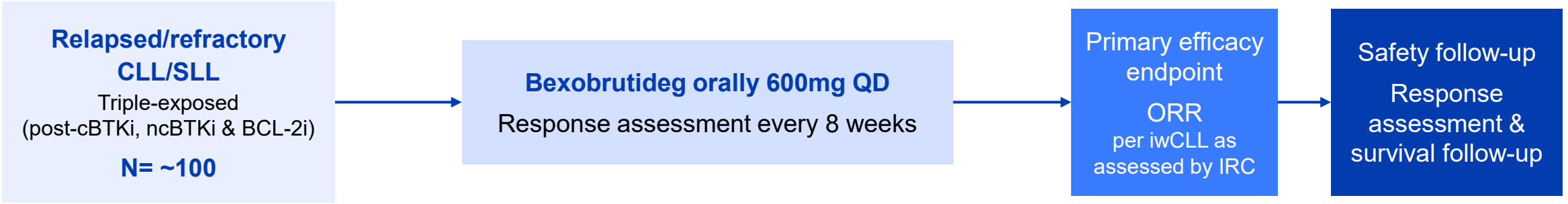
[a]Purpura/contusion includes episodes of purpura or contusion; [b]Fatigue was transient; [c]Aggregate of 'neutrophil count decreased' or 'neutropenia'; [d]Aggregate of 'rash' and 'rash maculopapular' and 'rash pustular'; [e]Aggregate of 'thrombocytopenia' and 'platelet count decreased'; [f]Aggregate of 'COVID-19' and 'COVID-19 pneumonia'; [g]Aggregate of 'pneumonia' and 'pneumonia klebsiella'

***CLL**, chronic lymphocytic leukemia; **SAE**, serious adverse event; **SLL**, small lymphocytic lymphoma; **TEAE**, treatment-emergent AE*

Data cutoff: 12 Mar 2025

Phase 2 Single-Arm Study for Potential Accelerated Approval

Triple-exposed CLL patients who progressed on or did not respond to prior therapy

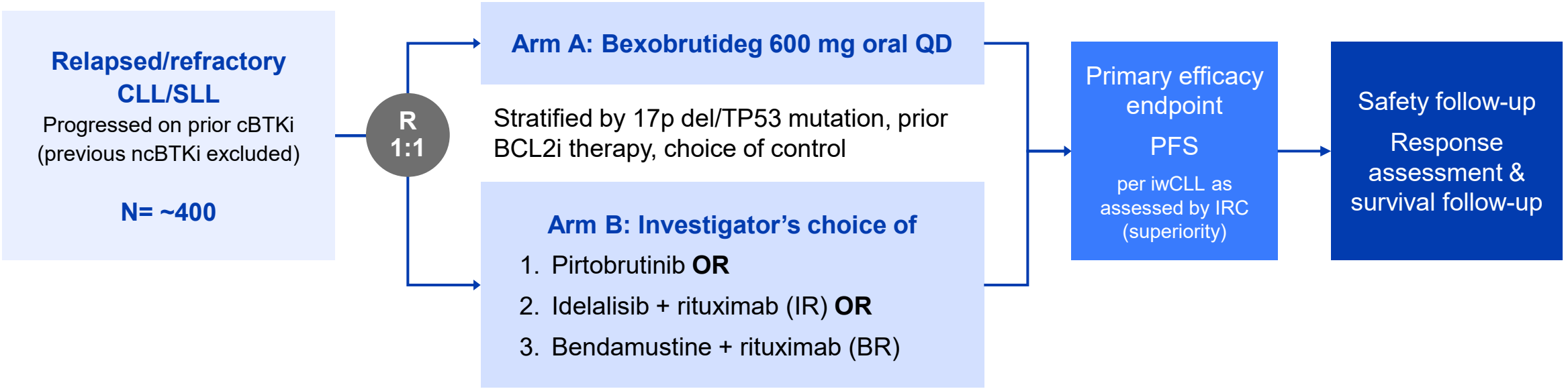


- Accelerated approval strategy depends on:
  - FDA's determination of unmet need at time of regulatory review
  - Adequate enrollment in confirmatory Phase 3 trial
- Trial designed to support potential accelerated approval in a high unmet need treatment setting
  - Post-cBTKi, post-ncBTKi, and post-BCL-2i (triple exposed)
- First site activated October 2025
  - 600 mg cleared for initiation of pivotal studies

***CLL**, chronic lymphocytic leukemia; **ORR,** objective response rate; **iwCLL,** International Workshop on CLL; **IRC,** Independent Review Committee; **QD,** once daily; **SLL,** small lymphocytic lymphoma; **cBTKi,** covalent BTK inhibitor; **ncBTKi,** non-covalent BTK inhibitor; **BCL-2i,** BCL-2 inhibitor*

Confirmatory Phase 3 Trial for Full Approval

2L+ CLL patients who progressed on prior covalent BTK inhibitor

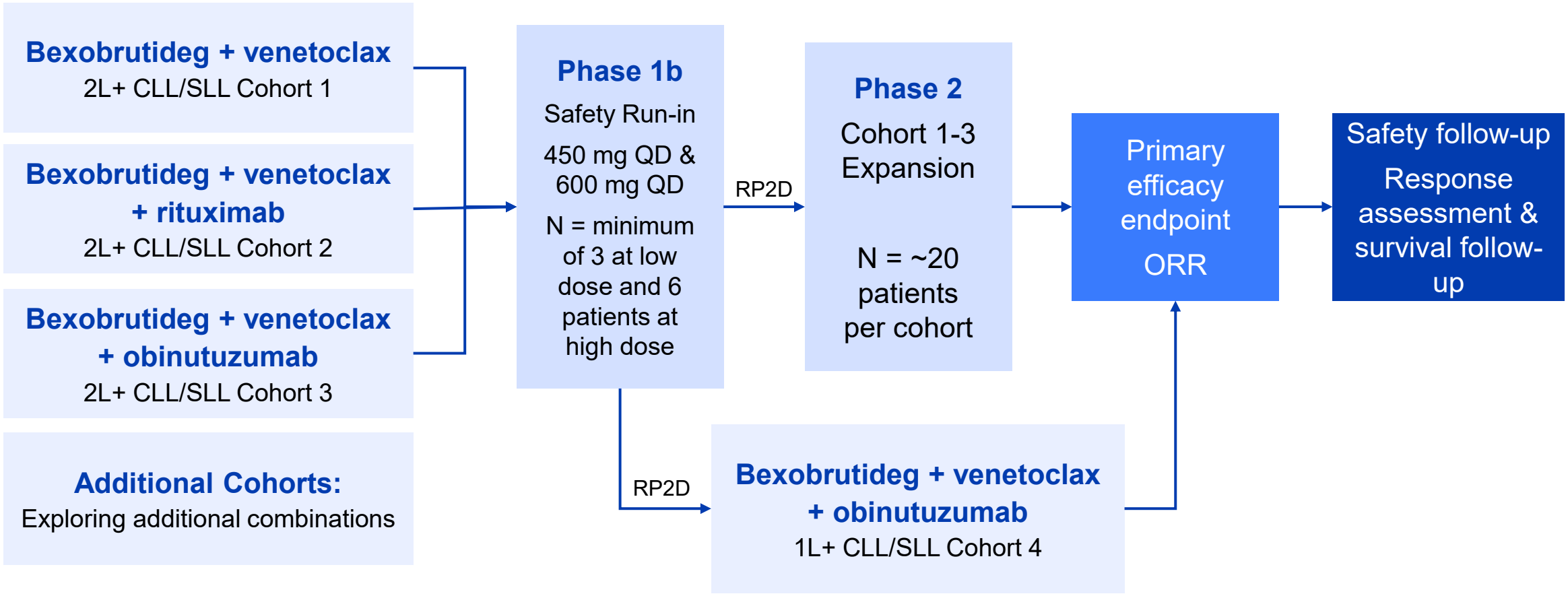


- Single trial strategy to support global approval
- Investigator's choice control arm:
  - Provides clinical relevance across geographies
  - Addresses current and emerging standards of care
  - Maximizes enrollment opportunities
  - Provides option for cross over to bexobrutideg upon documented progression

__CLL__, chronic lymphocytic leukemia; __PFS__, progression-free survival; __iwCLL__, International Workshop on CLL; __IRC__, Independent Review Committee; __QD__, once daily; __SLL__, small lymphocytic lymphoma; __ncBTKi__, non-covalent BTK inhibitor

Phase 1b/2 Combination Study to Address Emerging Treatment Standards in CLL

Combination regimen of bexobrutideg + BCL-2i maximizes 2L market share opportunity and provides potential path to 1L CLL



***CLL**, chronic lymphocytic leukemia; **SLL**, small lymphocytic lymphoma; **ORR**, objective response rate; **QD**, once daily; **RP2D**, recommended Phase 2 dose*

Building a World Class Hematology/Oncology Franchise

We aim to establish bexobrutideg as a cornerstone therapy across the CLL landscape

- **Phase 2 DAYBreak trial underway for monotherapy in r/r CLL**
- **Confirmatory Phase 3 for monotherapy in r/r CLL planned to start in H1 2026**
- **Combination trial planned to start H1 2026 to expand clinical opportunity across lines of therapy in CLL**
  - Initial focus on combinations with standards of care in CLL (BCL-2i & anti-CD20)
- **Maturing data from the ongoing Phase 1a/1b study to inform further development and potential paths for additional accelerated approvals**
  - Multiple bexobrutideg abstracts accepted for presentation at the upcoming American Society of Hematology (ASH) Annual Meeting in December 2025

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Potential Best-in-Class BTK Degrader: Potency, Coverage, and Selectivity

Gwenn Hansen, Ph.D.
Chief Scientific Officer

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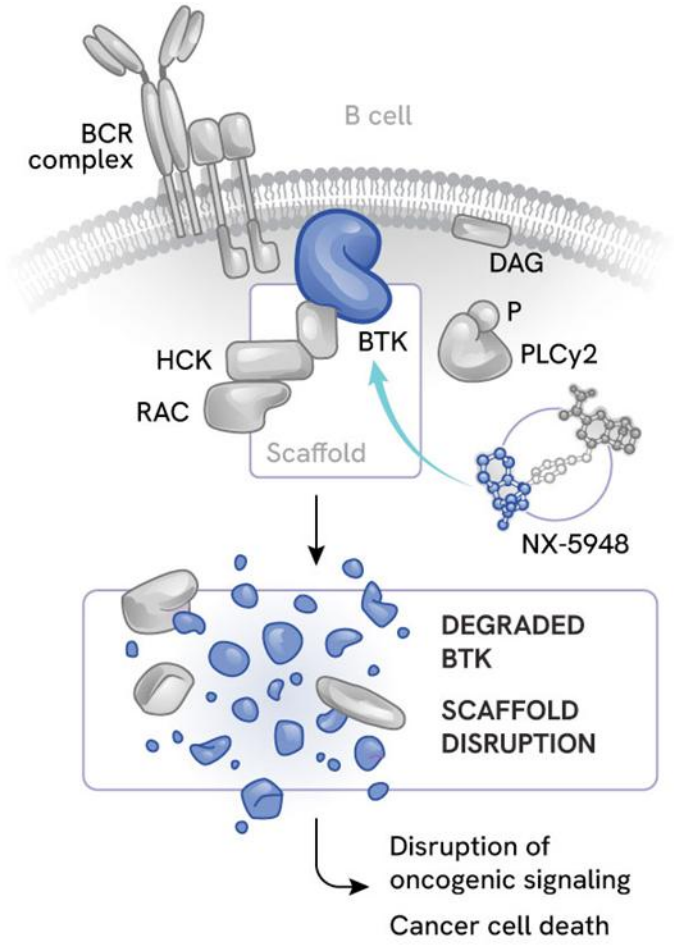
Bexobrutideg – Defining a Best-in-Class Profile

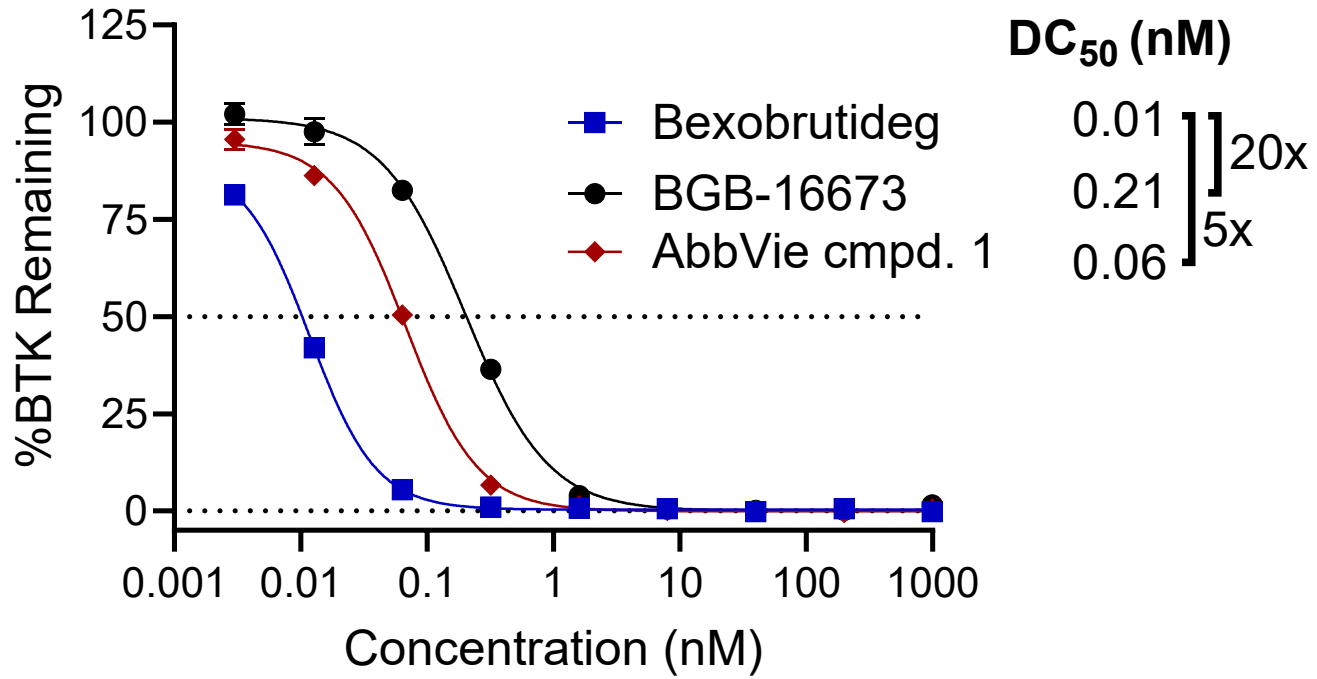


- ✓ Best-in-class degrader potency
- ✓ Best-in-class mutational coverage
- ✓ Best-in-class degrader selectivity
- ✓ Clear evidence of clinical activity in the CNS
- ✓ FDA clearance to proceed with highest tested dose

***BTK**, Bruton's tyrosine kinase; **CNS**, central nervous system*

Bexobrutideg Displays Best-in-Class BTK Degradation Potency

BTK Degradation in Human B Cells



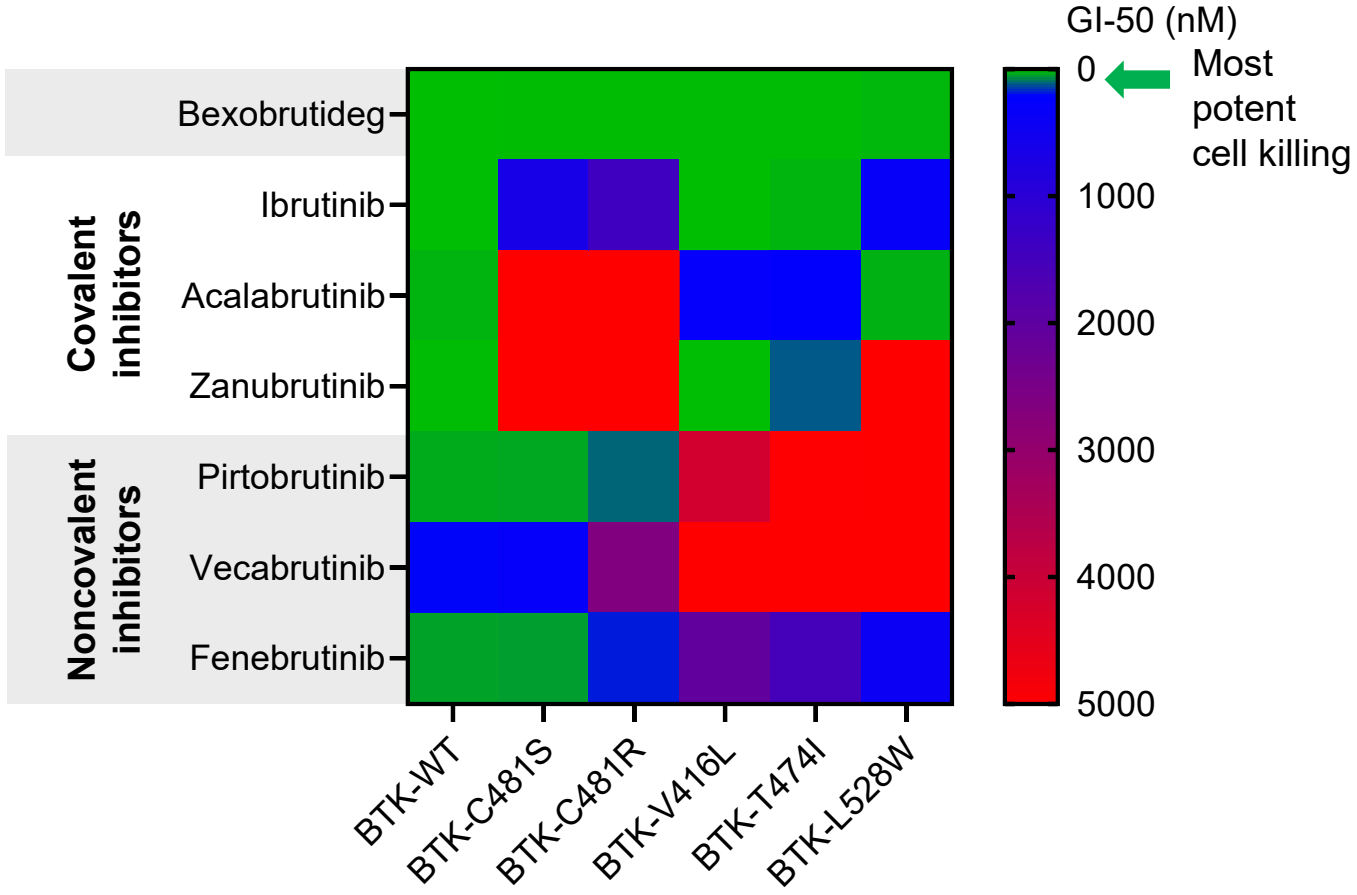
Bexobrutideg is **20x** more potent than BGB-16673 and **5x** more potent than AbbVie cmpd. 1

Degradation assessed at 24 hours by flow cytometry in human peripheral blood mononuclear cells, gated on CD20+ B cells
AbbVie cmpd. 1 is example 1 from WO 2023/183811 A1

Bexobrutideg Degrades Wild-Type and Mutated BTK with Superior Coverage Compared to All BTK Inhibitors

- All inhibitors have resistance mutation liabilities
- Bexobrutideg displays potent cell killing in the context of key resistance mutations
- We have shown that BTK degradation translates into clinical responses across key mutation classes

Bexobrutideg shows superior mutational coverage and cell killing compared to BTK inhibitors

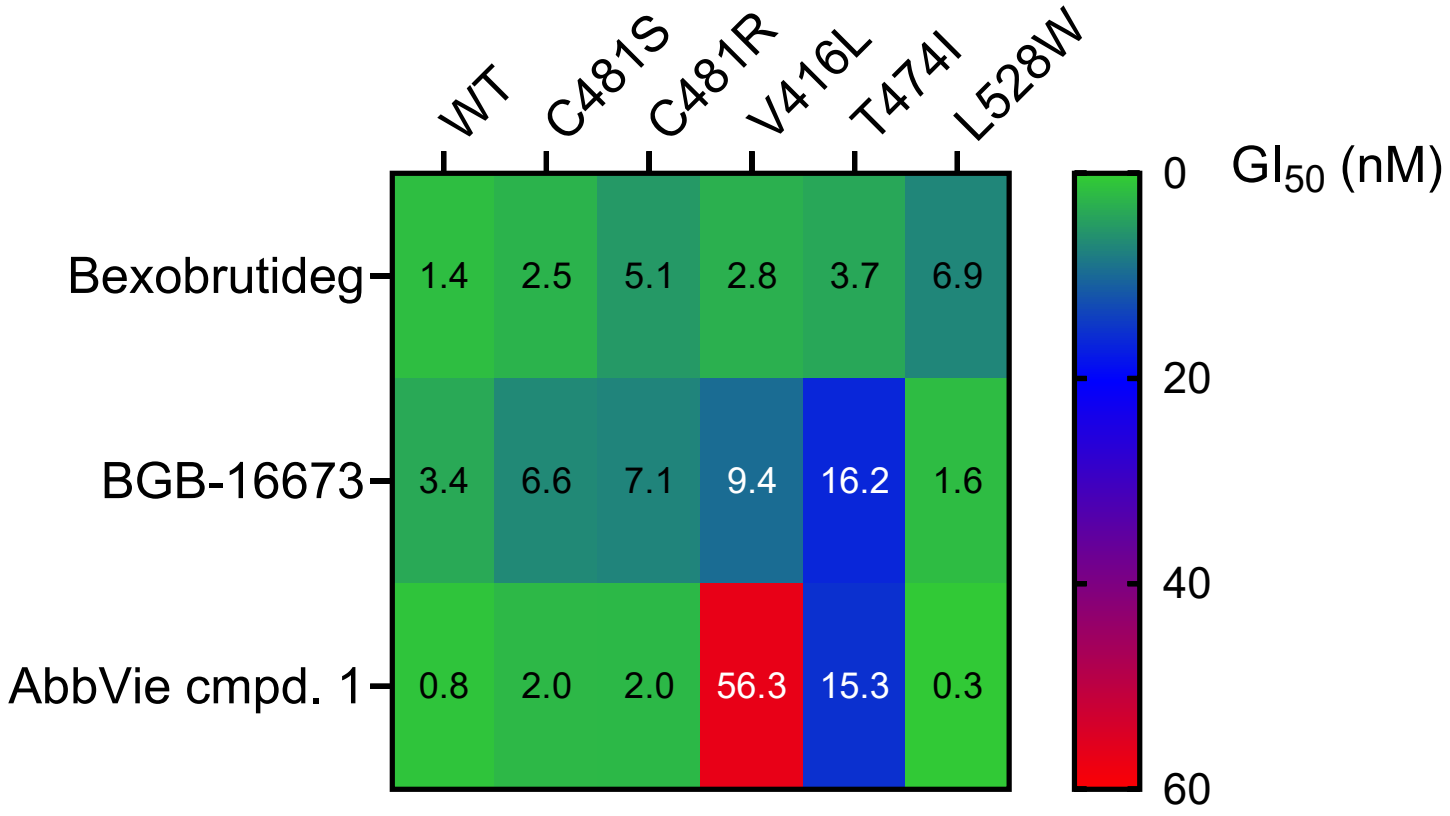


***BTK**, Bruton's tyrosine kinase; **GI-50** growth inhibition 50%; **WT** wildtype*

Bexobrutideg Displays the Most Potent Coverage Across BTK Mutations Compared to Other BTK Degraders

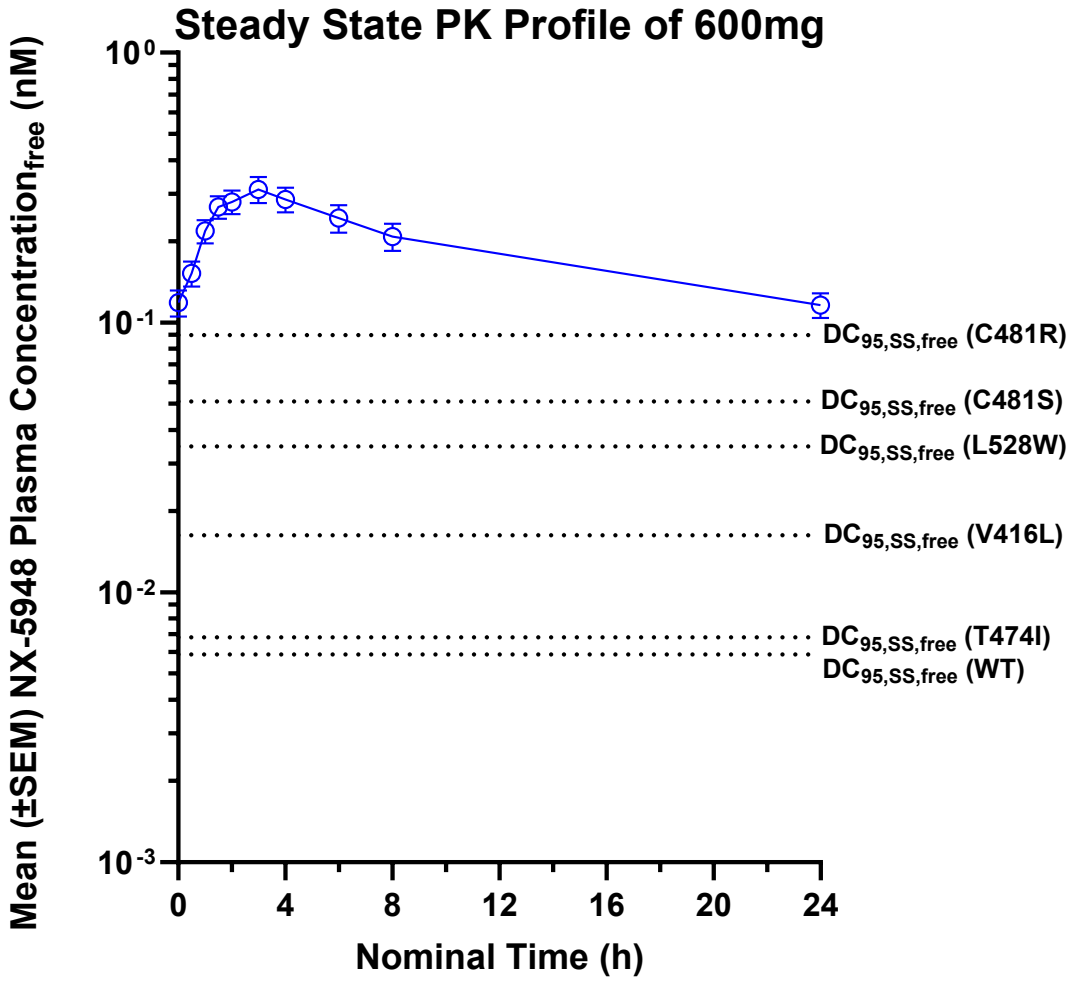
Bexobrutideg demonstrates GI_{50} values of <10 nM across relevant mutations, while BGB-16673 and AbbVie cmpd. 1 display potential liabilities

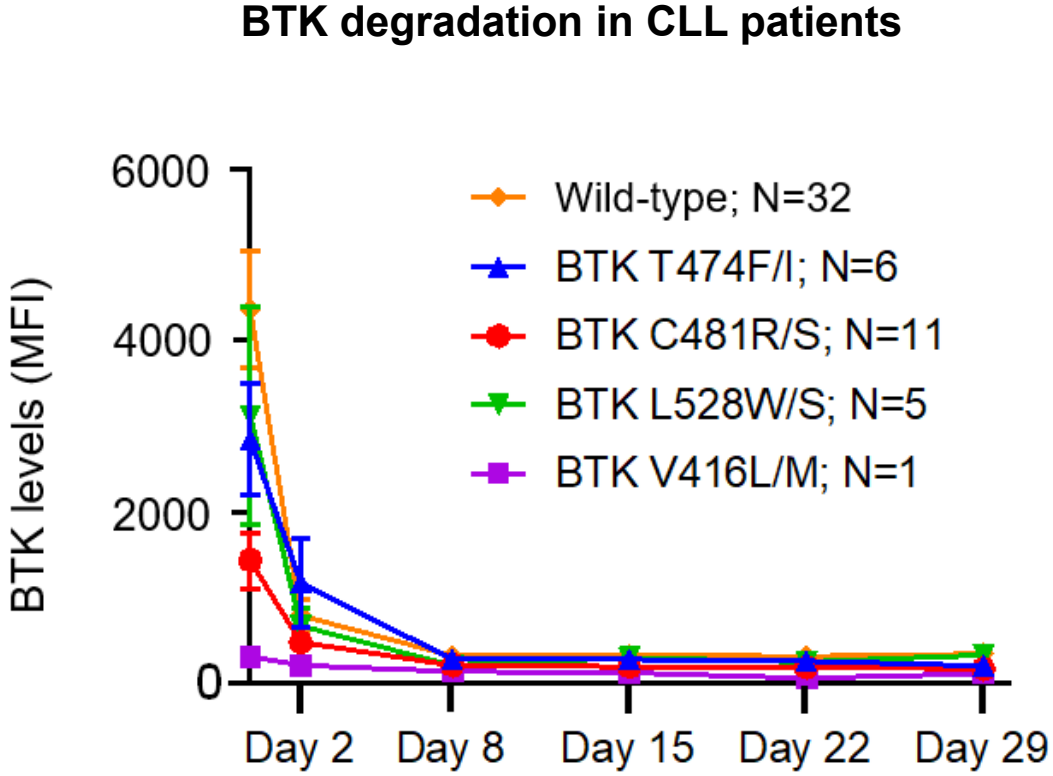
Cell Killing Activity Across Clinically Relevant Mutations



Viability assessed at 72 hours in TMD8 cells harboring CRISPR knock-ins of each BTK mutant. AbbVie cmpd. 1 is example 1 from WO 2023/183811 A1

Once a Day 600 mg Oral Dose of Bexobrutideg Achieves Optimal Coverage of Wild Type and Mutant BTK in CLL



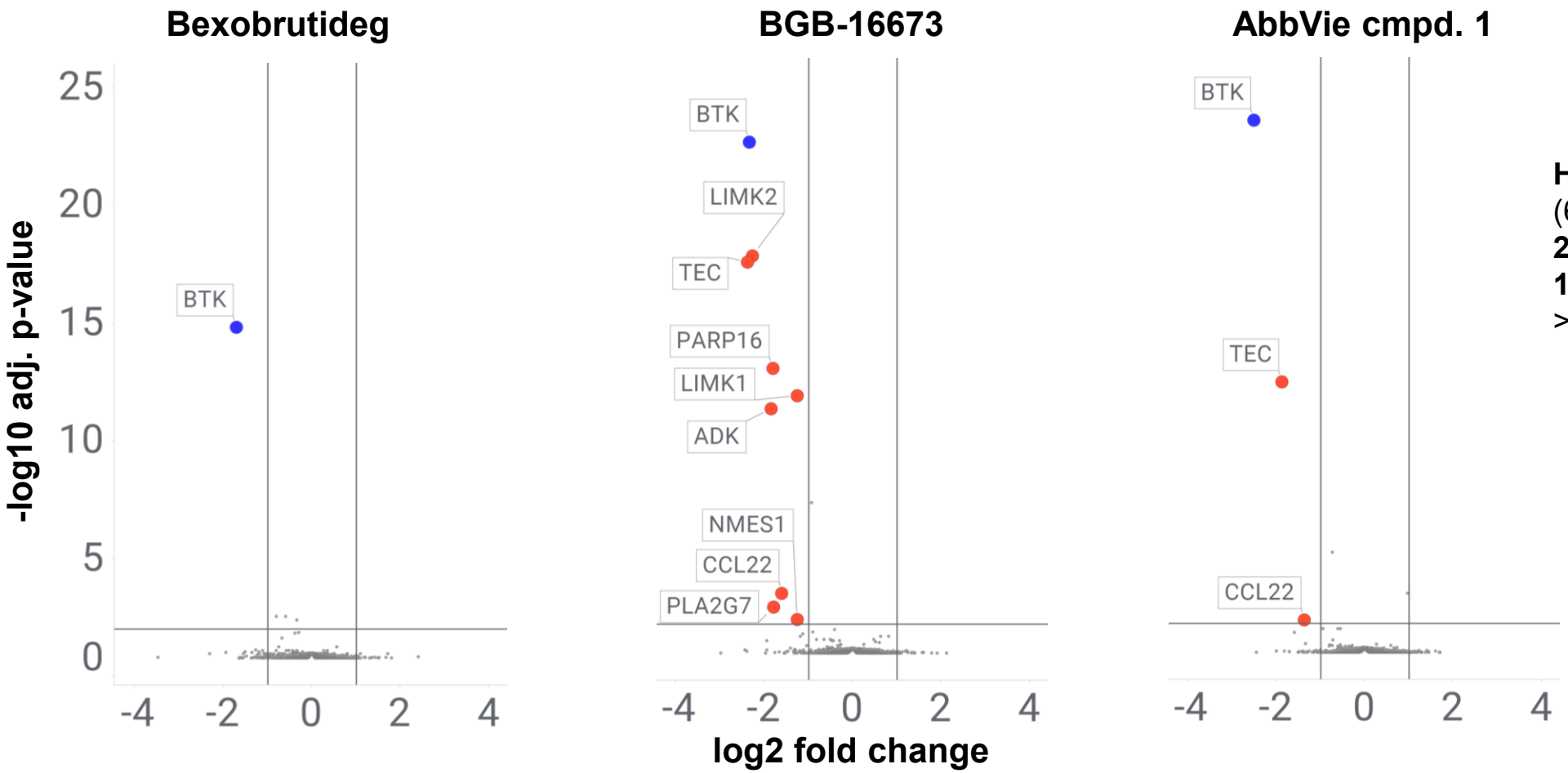


Note: Some patients have multiple BTK mutations

Arithmetic Mean (SEM) of n=90 are plotted for PK concentrations; PK Datacut: 27 May 2025
Avg = average; BTK = Bruton's tyrosine kinase; DC95 = concentration resulting in 95% degradation of target; SS = steady state; WT = wild type;
Horizontal dashed lines represent the free DC95 potency values at SS for WT BTK and mutants of interest (C481R, C481S, L528W, V416L, and T474I), as well as the avg value across these mutants of interest. Potency parameter (DC) was adjusted for rate of resynthesis using methodology published by Haid et.al, Clinical Pharmacology & Therapeutics, Vol 116 (3), September 2024.
PD Datacut: 10 Oct 2024

Bexobrutideg Is an Exquisitely Selective BTK Degrader

Global Proteomics in human PBMCs at clinically relevant exposures



Human PBMCs
(6 healthy donors)
24-hour treatment
10xDC90
>8700 proteins detected

- Global proteomics analysis reveals **bexobrutideg selectively degrades BTK, displaying no off-target degradation**
- **BGB-16673** and **AbbVie cmpd. 1** exhibit off-target liabilities including **TEC and ADK**

AbbVie cmpd. 1 is example 1 from WO 2023/183811 A1

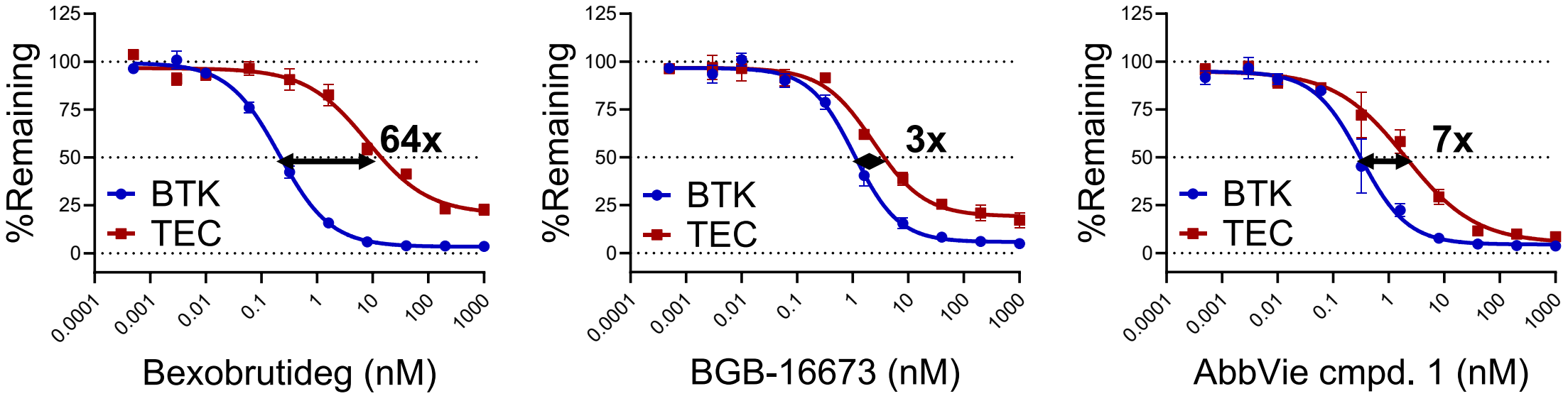
***PBMC**, peripheral blood mononuclear cell*

Bexobrutideg Has Best-In-Class Selectivity of BTK Over TEC

Selectivity of BTK over TEC is anticipated to provide safety advantage from lower cardiovascular side effects[a]

	Bexdeg	BGB-16673	AbbVie cmpd. 1[b]	Acala.	Zanu.	Ibrutinib
BTK/TEC Selectivity[c,d]	64x	3x	7x	25x	7x	7x

AbbVie and BeOne BTK degraders potently degrade TEC in K562 cells



a. Chen et al. 2024. Heart, Lung and Circulation 33: S481. *b. AbbVie cmpd. 1 is example 1 from WO 2023/183811 A1*
c. Degradation selectivity assessed in K562 cells at 24 hours; Mean ± SEM from n = 3 independent experiments is displayed
d. Inhibitor selectivity is the ratio of BTK and TEC IC_{50} values from O'Brien et al. 2021. Front Oncol 11: 720704.

Bexobrutideg Has Best-In-Class Selectivity

In vitro dose-dependent degradation assays used to confirm off target liabilities predicted by global proteomics

Target		Parameter	Bexobrutideg	BGB-16673	AbbVie cmpd. 1
BTK	Bruton's tyrosine kinase	DC_{50}	0.010 nM	0.206 nM	0.063 nM
LCK	Lymphocyte-specific kinase	Fold Selectivity (ratio of DC_{50} at 24h)	2,300x	49x	>10,000x
CSK	C-terminal Src kinase		4,200x	39x	6,000x
ADK	Adenosine kinase		>10,000x	60x	>10,000x
TEC	Tyrosine kinase expressed in hepatocellular carcinoma		64x	3x	7x

- **LCK** humans with loss of LCK have combined immune deficiency syndrome with severely defective T cell signaling and suffer from opportunistic infections[1]
- **CSK** human genetics shows low expression is associated with hypertension; knockdown in animal models causes hypertension[2]
- **ADK** is an important metabolic enzyme. ADK deficiency in humans has been shown to cause abnormal liver function, hypermethionemia and encephalopathy.[3] ADK-deficient mice are not viable and have abnormal liver function.[4]
- **TEC** is a tyrosine kinase related to BTK. Combined loss of BTK and TEC leads to cardiac hypertrophy and ventricular fibrosis in mice.[5]

BTK degradation assessed by flow cytometry in human PBMCs (hPBMCs), gated on CD20+ B cells. LCK/CSK/ADK DC_{50} values are compared to BTK DC_{50} in primary B cells, TEC DC_{50} values are compared to BTK DC_{50} in K562 cells. LCK degradation by bexobrutideg assessed by Jess SimpleWestern in bulk hPBMCs. CSK degradation by bexobrutideg assessed by flow cytometry in hPBMCs, gated on CD4+ T cells. LCK and CSK degradation by BGB-16673 and AbbVie cmpd. 1 assessed by flow cytometry in hPBMCs, gated on CD3+ CD8- (LCK) and CD3+ (CSK) T cells. Bexobrutideg did not significantly degrade ADK in global proteomics in hPBMCs (top conc. = 1000 nM). ADK degradation by BGB-16673 and AbbVie cmpd.1 assessed by Jess SimpleWestern in HepG2 cells. TEC degradation assessed by Jess SimpleWestern in K562 cells. AbbVie cmpd. 1 is example 1 from WO 2023/183811 A1 [1] Keller et al. 2024. J Clin Immunol. **44**(4). [2] Hyon-Ju Lee et al. 2016. PLOS One **11**(1): e0146841. [3] Bjursell et al. 2011. Am J Hum Gen **89**(4): 507–515. [4] Boison et al. 2002. Proc Nat Acad Sci **99**(10): 6985-6990. [5] Chen et al. 2024. Heart, Lung and Circulation **33**: S481.

Extending our leadership into I&I with potential best-in-class agents

Building a High-Value I&I Franchise with Best-in-Class Mechanisms for Validated Targets

Inflammation & Immunology

Program	Target	Modality	Therapeutic area	Discovery	IND-Enabling	Phase 1A	Phase 1B	Phase 2/3
Bexobrutideg (NX-5948)	BTK	Degrader	Autoimmune cytopenia in CLL patients					
NX-0479 / GS-6791	IRAK4	Degrader	Rheumatoid arthritis and other inflammatory diseases					GILEAD
NX-3911	STAT6	Degrader	Type 2 inflammatory diseases					sanofi
Undisclosed	Undisclosed	Degrader	Inflammation / autoimmune					sanofi
Multiple	Undisclosed	DAC	Inflammation / autoimmune					

NURIX

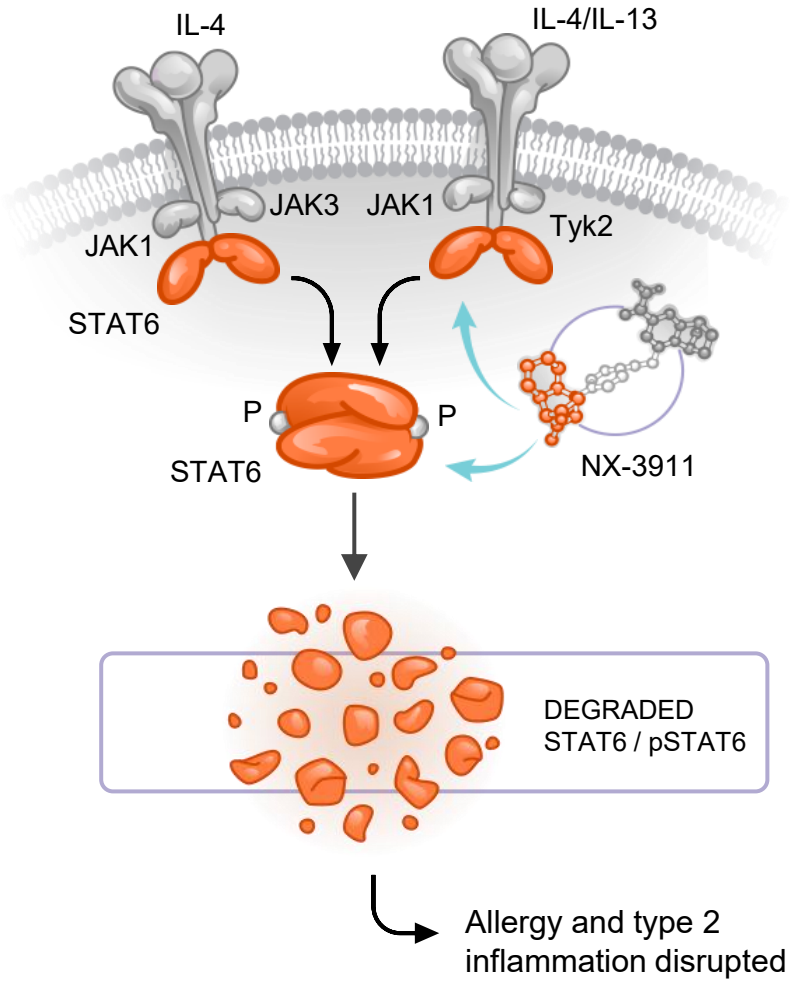
NX-3911: A Potential Best-in-Class STAT6 Degrader in Collaboration with Sanofi that Achieves Complete STAT6 Pathway Blockade

STAT6 plays a central role in type 2 inflammation, driving diseases such as atopic dermatitis & asthma

NX-3911 Potential for Biologic-like Efficacy in a Pill

KEY ADVANTAGES:

- **Powerful efficacy:** Delivers complete STAT6 pathway blockade in disease-relevant cells
- **Exquisite selectivity:** Designed to avoid off target effects
- **Patient-friendly**: Convenient oral dosing
- **Greater accessibility**: Potentially expands treatment to previously unreachable populations



The STAT6 Transcription Factor:

- Acts as a key regulator of the JAK/STAT signaling pathway selectively downstream of the inflammatory cytokines IL-4 and IL-13
- Drives Th2-mediated inflammatory disorders including allergies, asthma, atopic dermatitis, and eosinophilic esophagitis
- Pathway is clinically validated:
  - anti-IL4Ra and anti-IL13 monoclonal antibodies
  - JAK inhibitors

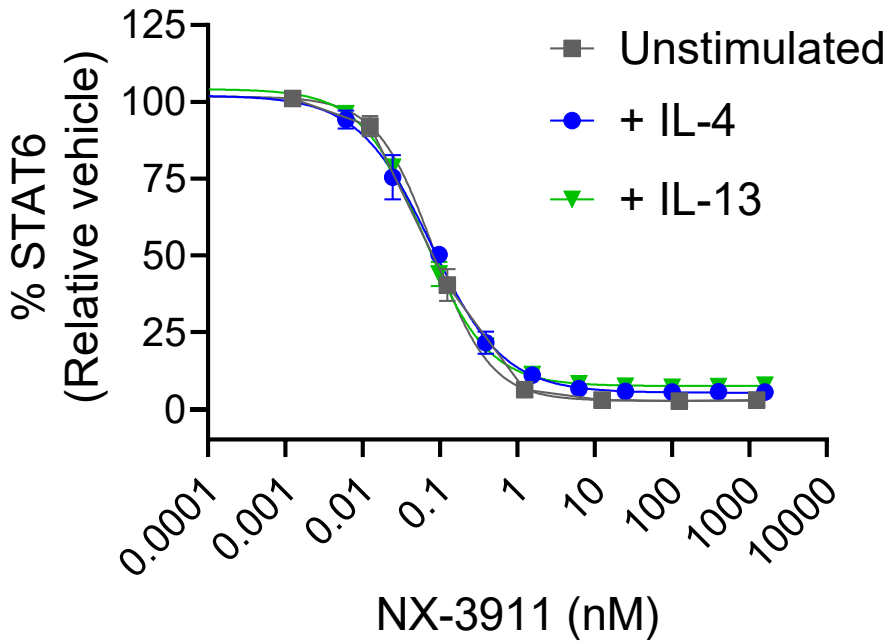
***JAK**, Janus kinase; **STAT**, Signal Transducer and Activator of Transcription*

NX-3911 Degrades STAT6 With Picomolar Potency in Disease Relevant Cells

- Potent STAT6 degradation in multiple primary human disease relevant cell types: immune cells, epidermal keratinocytes and dermal fibroblasts
- NX-3911 fully degrades STAT6 in resting as well as TH2-pathway stimulated PBMCs

Primary Human Cell Type	DC_{50} (nM)
Human PBMCs	0.09
Human IL-4 stimulated PBMCs	0.09
Human IL-13 stimulated PBMCs	0.08

STAT6 Degradation in Human PBMCs



***PBMC**, peripheral blood mononuclear cell*

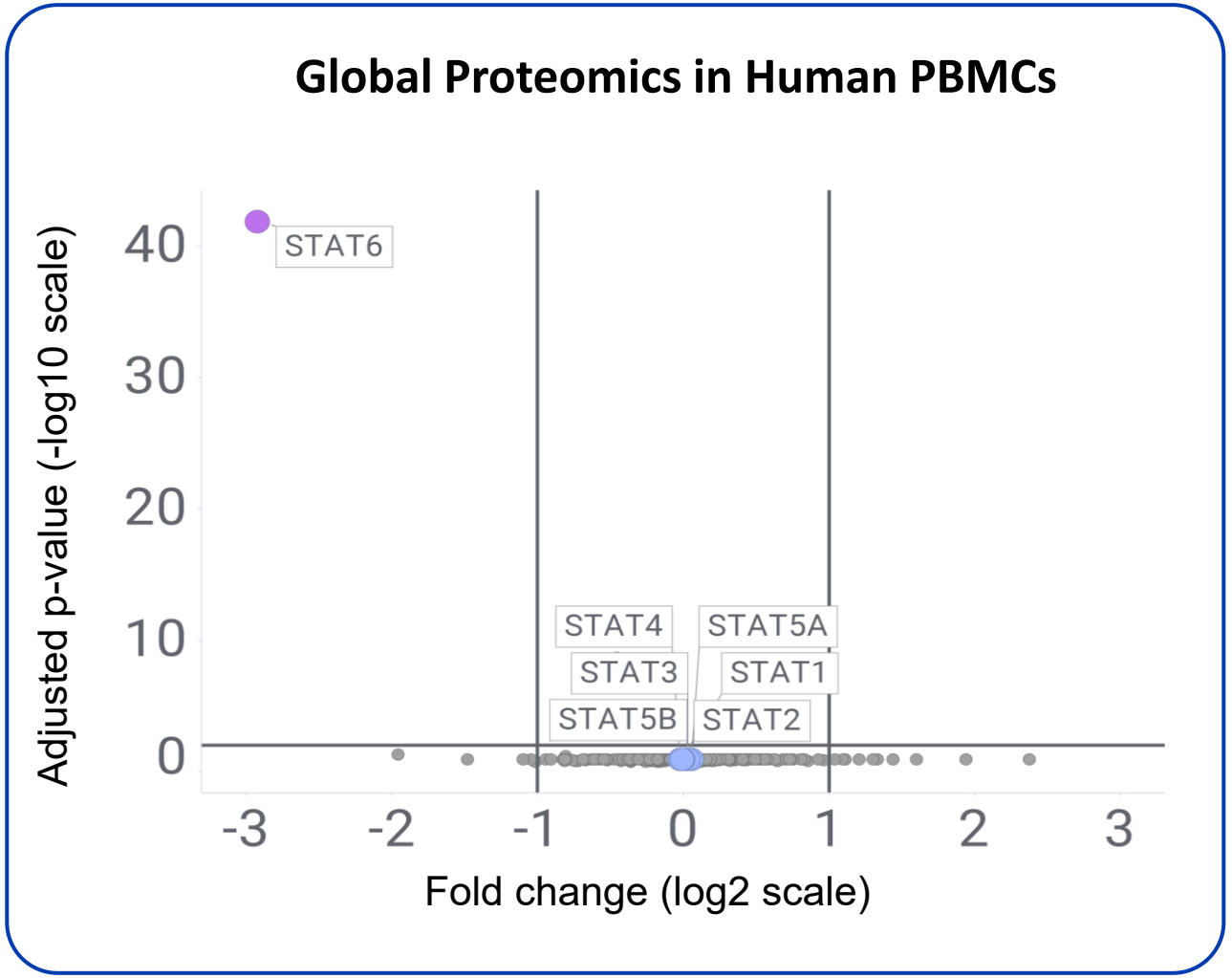
NX-3911 Degrades STAT6 With Exquisite Selectivity

- Highly selective degradation of STAT6 in human PBMCs

  (global proteomics assessed at 24h at 50x DC_{50})

- No significant change observed for any other protein, including STAT family members

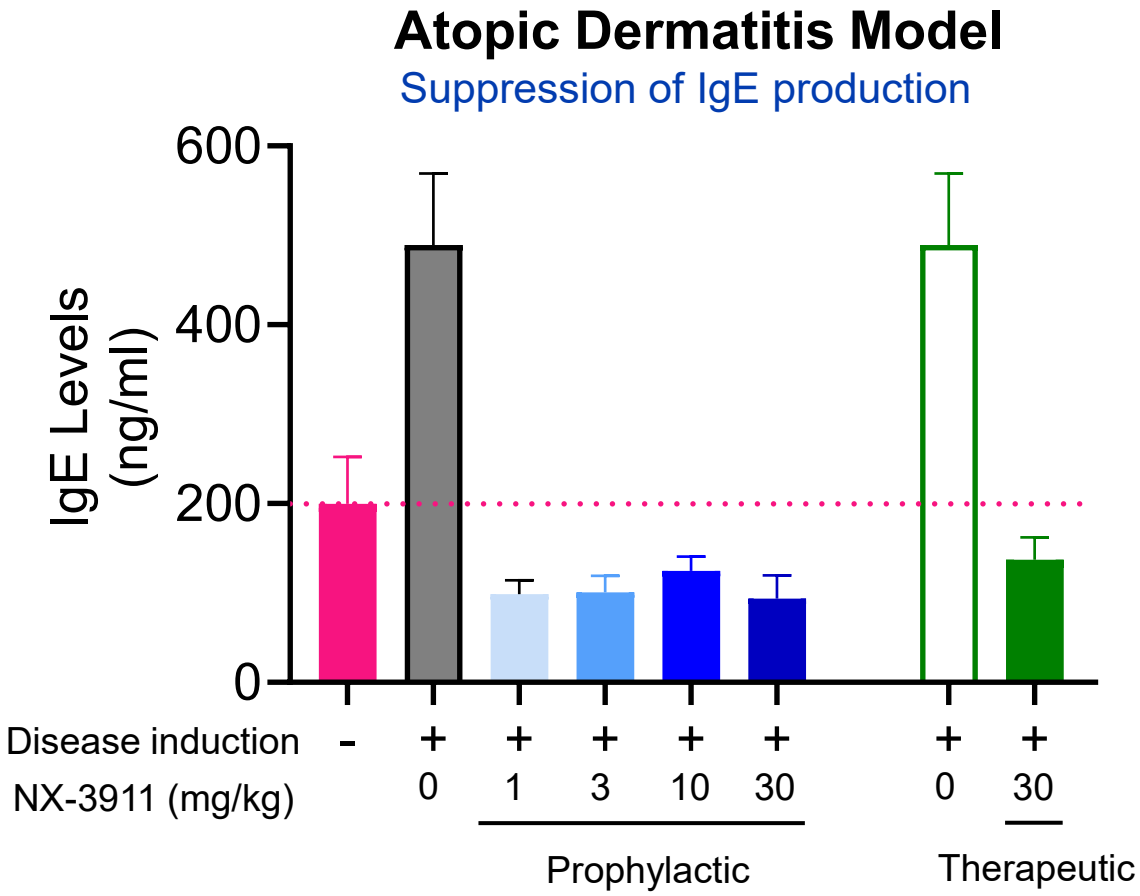
  (9500 total proteins measured)

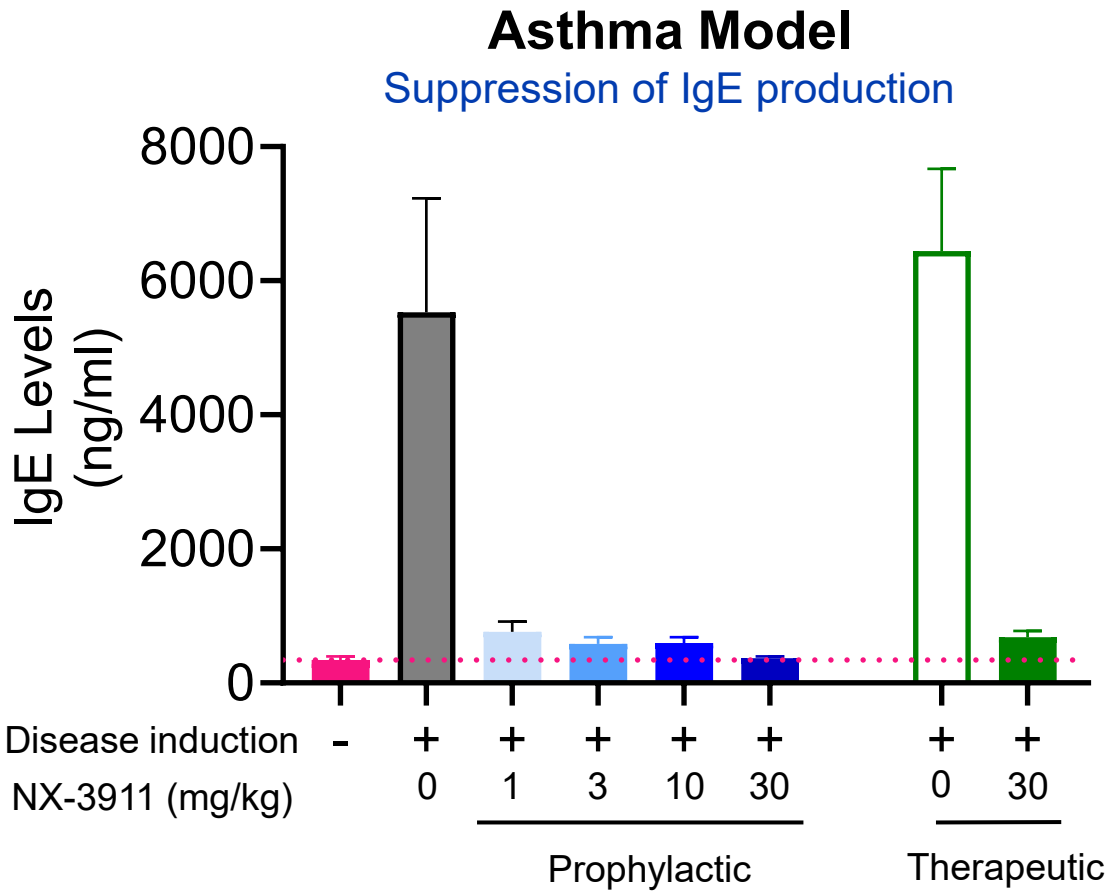


***PBMC**, peripheral blood mononuclear cell*

NX-3911 Has Demonstrated Activity in Inflammatory Disease Models of Atopic Dermatitis and Asthma

Achieves control of inflammatory activity in both prophylactic and therapeutic settings





Disease induced by MC903 in Atopic Dermatitis and House Dust Mite in Asthma models in hIL-4/IL-4Rα/hIL-13 mice (C57BL/6 background)
Red dotted line represents the average IgE levels observed in naïve mice.
All treated groups are statistically significant (P<0.05) compared to vehicle control (0 mg/kg)

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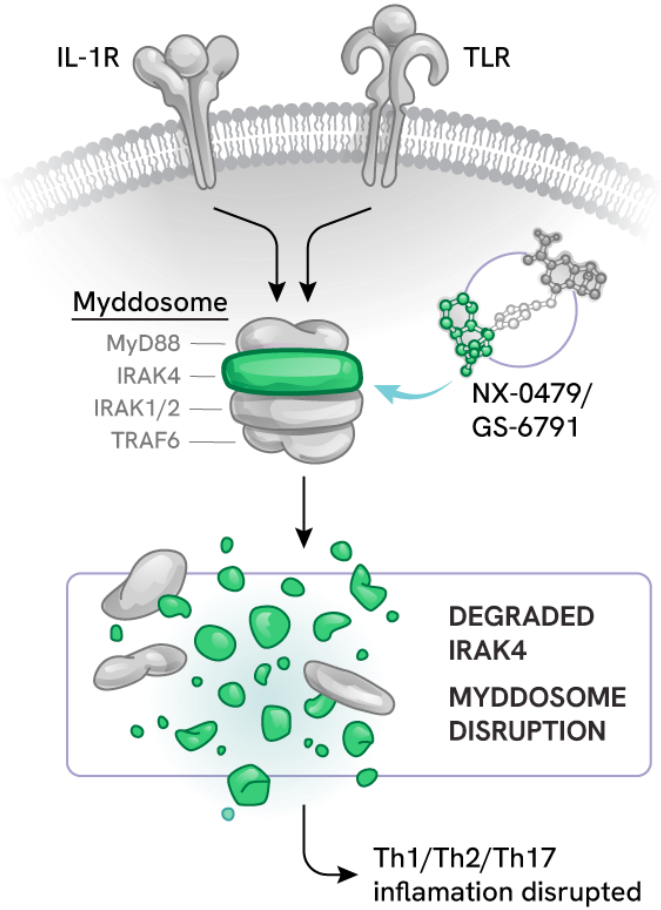
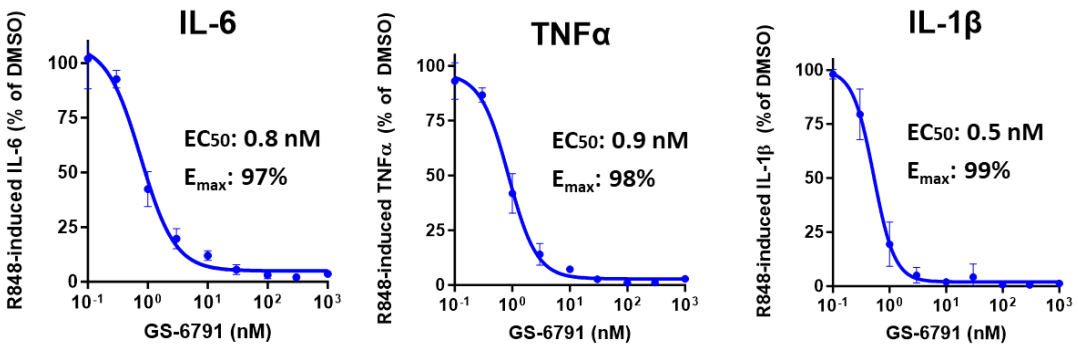
IRAK4 Degrader NX-0479/GS-6791: Potential Treatment for Rheumatoid Arthritis and Other Inflammatory Diseases

Phase 1 initiated by collaboration partner Gilead Sciences in Q2 2025;
Nurix has a co-development and 50/50 profit share option in the United States

- IRAK4 is a master regulator of the Toll-like Receptor (TLR) and Interleukin-1 Receptor (IL-1R) signaling pathways
- Inappropriate activation of these receptors promotes inflammation and autoimmunity through the release of inflammatory cytokines and chemokines
- IRAK4 exhibits both kinase and scaffolding functions
- Degradation of IRAK4 achieves more complete blockade of the TLR/IL-1R signaling pathways and yields broader anti-inflammatory effects than inhibition alone



Functional Inhibition of TLR responses in human PBMC

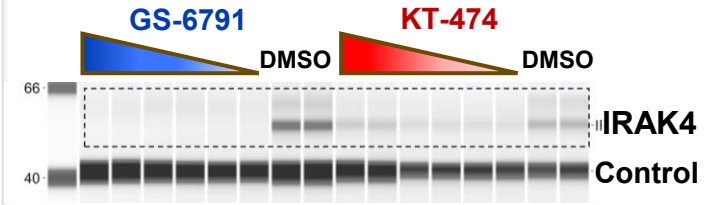


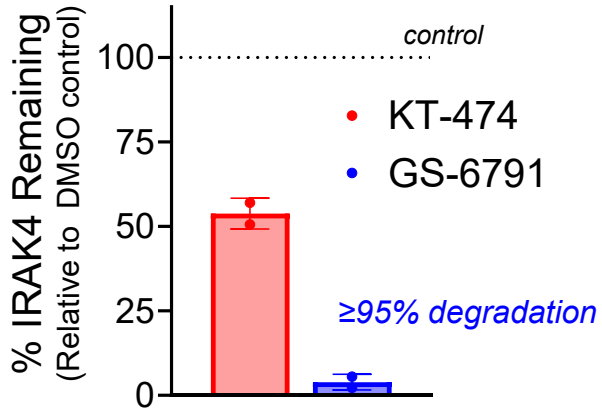
Source: Teng et al., ACR 2024, November 2024 .

***PBMC**, peripheral blood mononuclear cell; **TNF**, tumor necrosis factor*

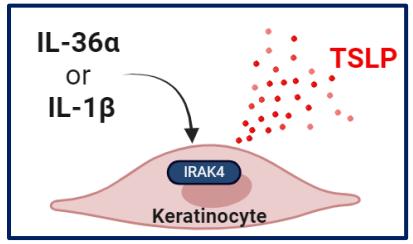
NX-0479/GS-6791 Elicits Best-in-Class IRAK4 Degradation and Inhibition of Functional Responses in Skin Epithelial Systems

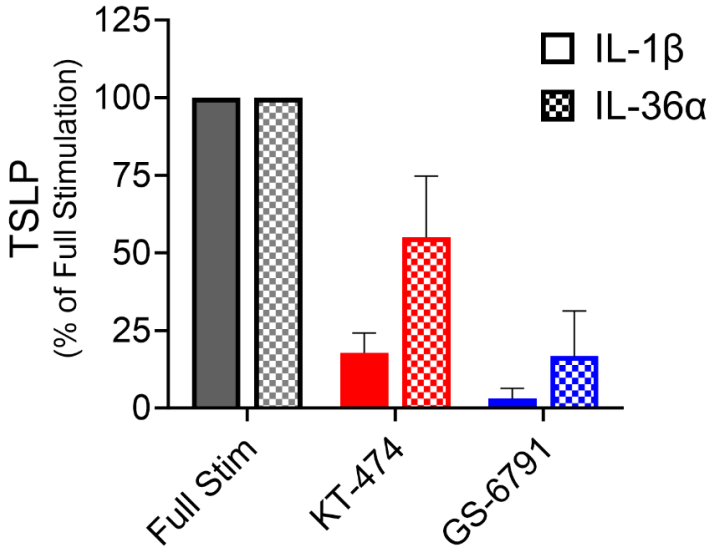
Deeper IRAK4 degradation in basal human keratinocytes at 24 h



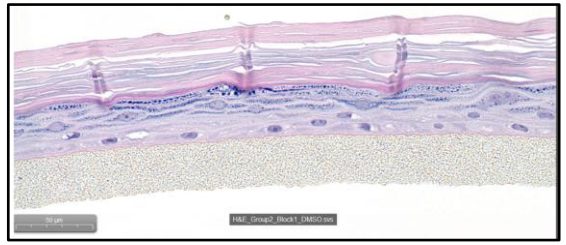


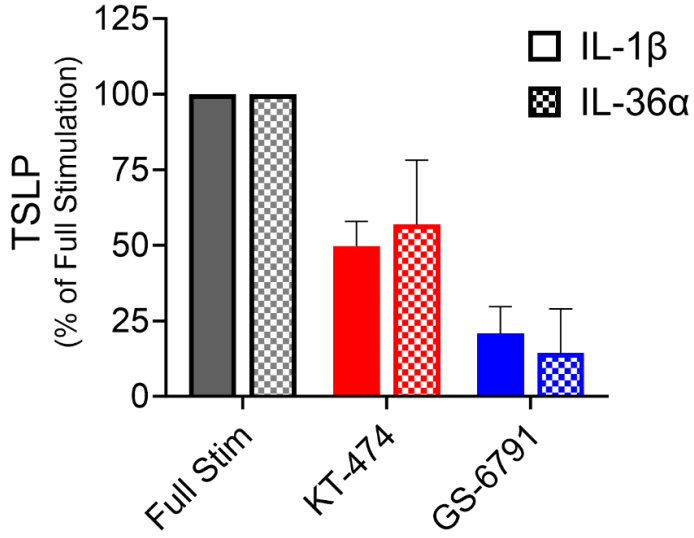
Superior inhibition in differentiated human keratinocytes





Superior inhibition in reconstructed human epidermis





TSLP: Thymic Stromal Lymphopoietin
IRAK4 degradation and functional inhibition assessed at 1 μM test article concentration
Source: Gilead Sciences data

Driving Value with Wholly Owned and Partnered Programs

Arthur T. Sands, M.D., Ph.D.
President and Chief Executive Officer

NURIX

Building an Oncology and Immunology Powerhouse

- **Bexobrutideg has the potential to produce a pipeline of value over the course of its lifecycle**
  - CLL, WM, NHL, and potentially I&I indications

- **Advancing bexobrutideg one step closer to registration and commercialization**
  - ✓ 600 mg dose selected per Project Optimus
  - ✓ Regulatory alignment on dose with FDA, MHRA, EMA
  - ✓ Pivotal Phase 2 trial initiated – DAYBreak CLL-201
  - ✓ Confirmatory Phase 3 trial initiation planned for H1 2026

- **Potential best-in-class profile emerging for bexobrutideg**

- **Partnered programs advancing toward significant regulatory and clinical milestones and have the potential to create substantial value for Nurix shareholders with our 50-50 U.S. profit share opt in rights**
  - STAT6 and IRAK4

- **Upon completion of our registered direct offering, pro forma cash is anticipated to be $678.8 million, providing expected runway into 2028***

***EMA**, European Medicines Agency; **FDA**, U.S. Food and Drug Administration; **MHRA**, U.K. Medicines and Healthcare products Regulatory Agency*

** Pro forma cash reflects expected net proceeds from the October 2025 offering added to Nurix's cash, cash equivalents, and marketable securities balance as of August 31, 2025.*

NURIX

Paths to Value Creation

Forecast milestones for bexobrutideg and select I&I programs

	2025	2026
Bexobrutideg: potential best-in-class leadership in CLL	✓ Establish 600 mg dose ✓ Initiate DAYBreak study ▪ Present clinical update at ASH	▪ Ph1a/b NHL results and Ph1b CLL cohorts ▪ Initiate confirmatory Ph3 study in r/r CLL (H1) ▪ Initiate bexobrutideg combination study in CLL
Extending leadership into I&I	✓ Initiate GS-6791 Ph1 by Gilead ✓ Secure license with Sanofi for 2 degrader programs including STAT6 ✓ Initiate healthy volunteer studies for bexobrutideg to support I&I IND	▪ Potential GS-6791 opt-in based on Gilead's Ph1 results* ▪ Bexobrutideg Ph1 SAD/MAD data ▪ Potential NX-3911 STAT6 degrader IND filed by Sanofi*

** Nurix estimate for partnered programs using industry standard timelines based on current stage of development (not official guidance of partners).*

Q&A

Thank you