NURIX

Protein Degraders to Outmatch Cancer and Autoimmune Disease

April 2026 Corporate Presentation

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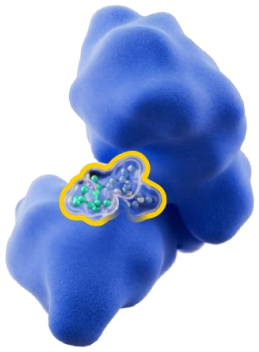
OUR MISSION

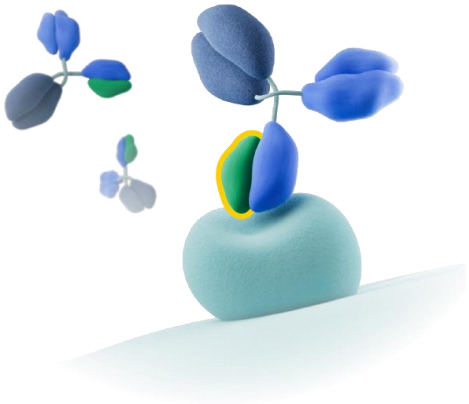
To establish degrader-based medicines at the forefront of patient care

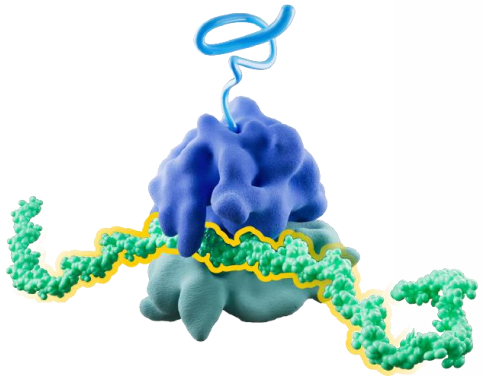
NURIX

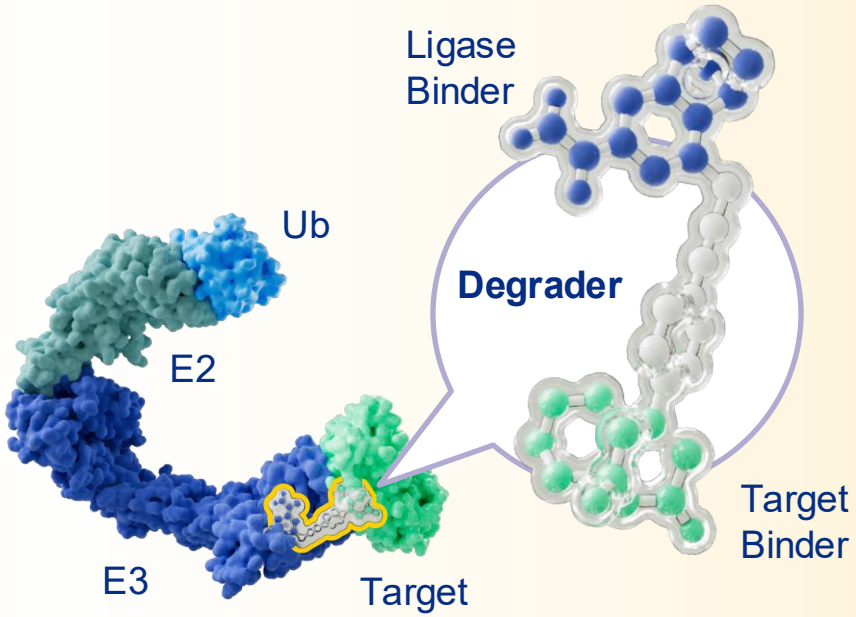
Leading the Next Frontier in Drug Development

Target Protein Degradation (TPD)









Small Molecule Inhibitors

Antibodies

Nucleic Acid-based Therapies:
Antisense • RNAi Gene Therapy • CRISPR

Target Protein Degradation Drugs

Evolution of New Therapeutic Modalities

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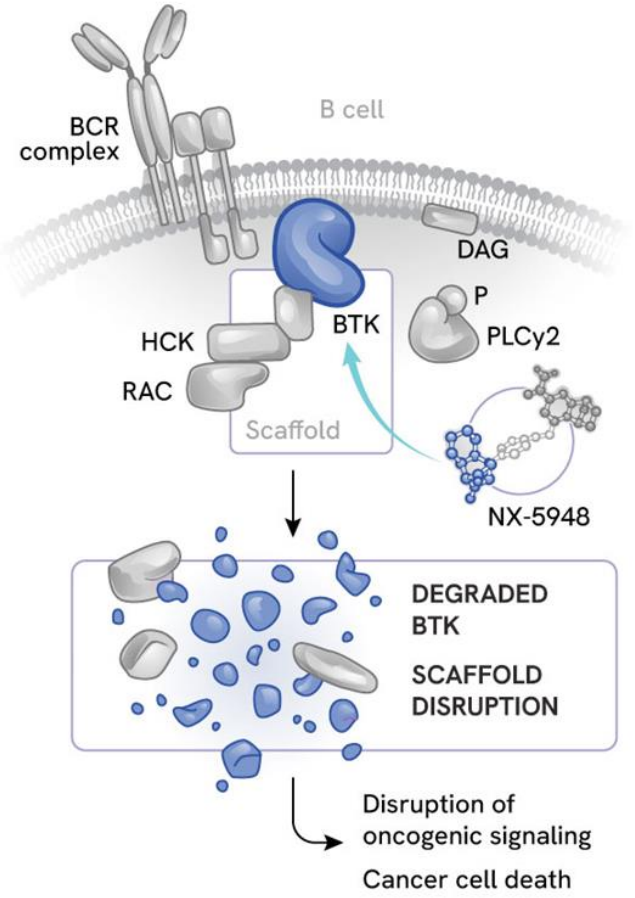
Bexobrutideg – The First "deg" with a Potential Best-in-Class Profile

Novel MOA Against a Clinically and Commercially Proven Target

Removes both BTK enzymatic activity and scaffolding functions



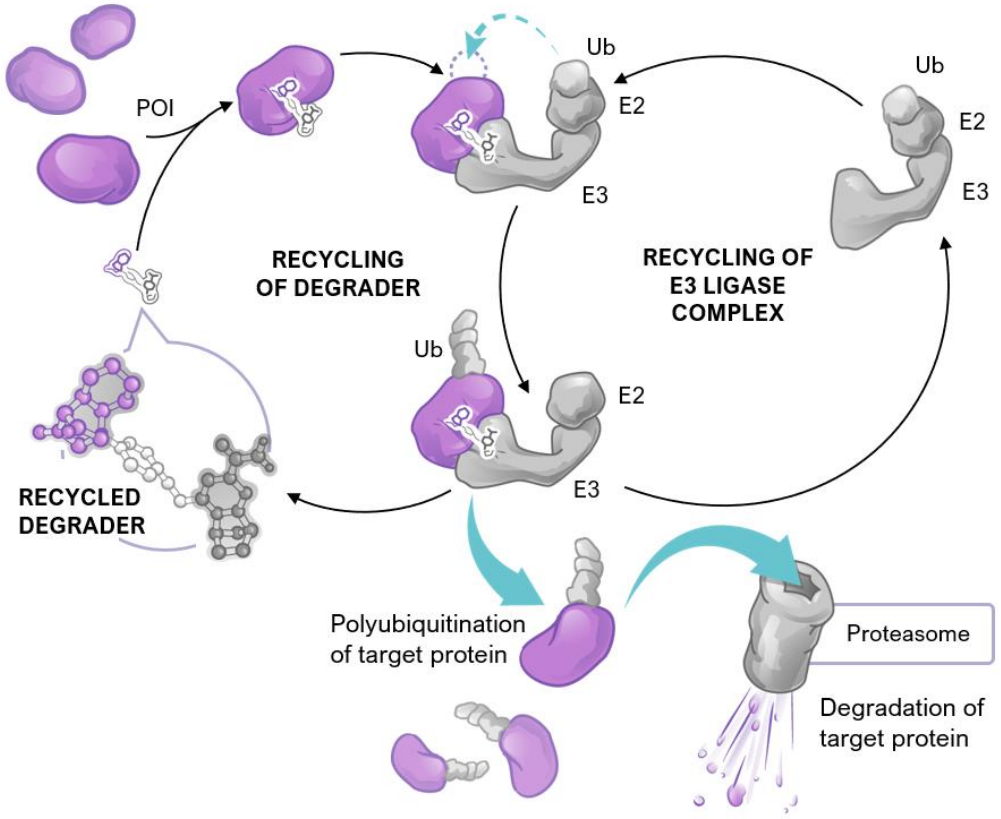
- ✓ Degradation removes all functions of BTK unlike BTK inhibitors
- ✓ Acts catalytically with unprecedented potency
- ✓ Exquisitely selective degrader of BTK
- ✓ Active against wildtype BTK and overcomes BTK inhibitor resistance mutations
- ✓ Crosses the blood brain barrier with clinical responses in patients with advanced CNS disease
- ✓ Demonstrates robust clinical activity in difficult to treat B-cell malignancies

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

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

Novel MOA Against a Clinically and Commercially Proven Target

One molecule of bexobrutideg degrades thousands of BTK proteins per hour



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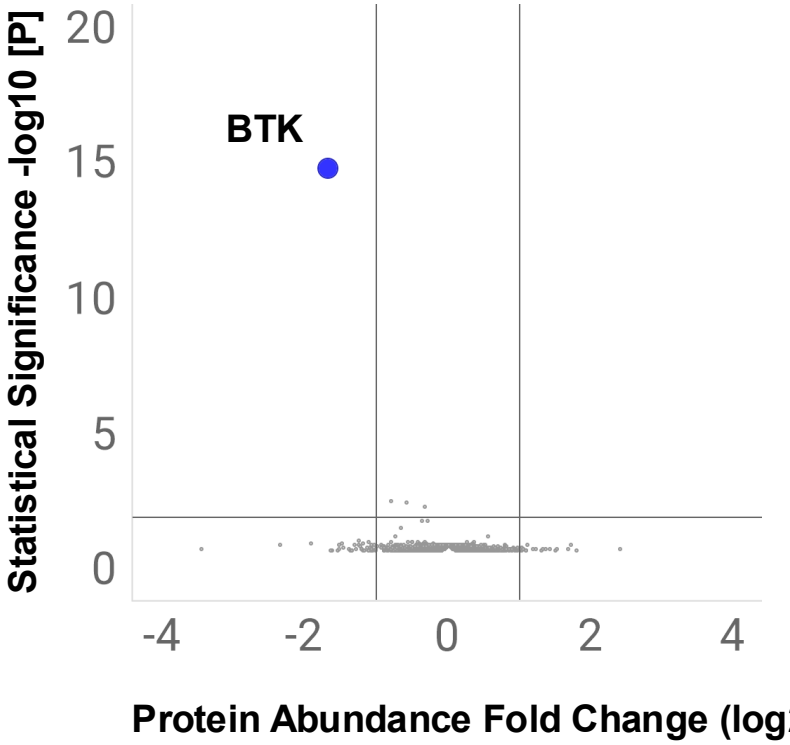
Bexobrutideg – The First "deg" with a Potential Best-in-Class Profile

Novel MOA Against a Clinically and Commercially Proven Target

Global proteomics analysis shows bexdeg selectively degrades BTK with no off-target degradation



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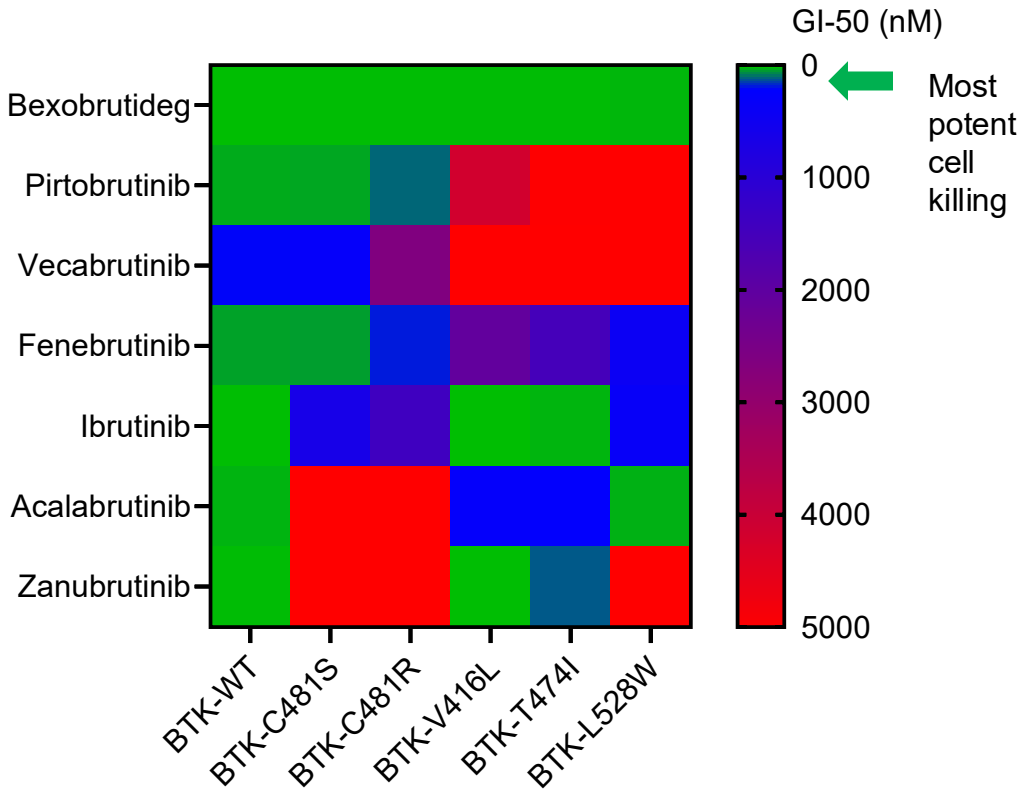
Bexobrutideg – The First "deg" with a Potential Best-in-Class Profile

Novel MOA Against a Clinically and Commercially Proven Target

Bexobrutideg shows superior mutational coverage compared to BTK inhibitors



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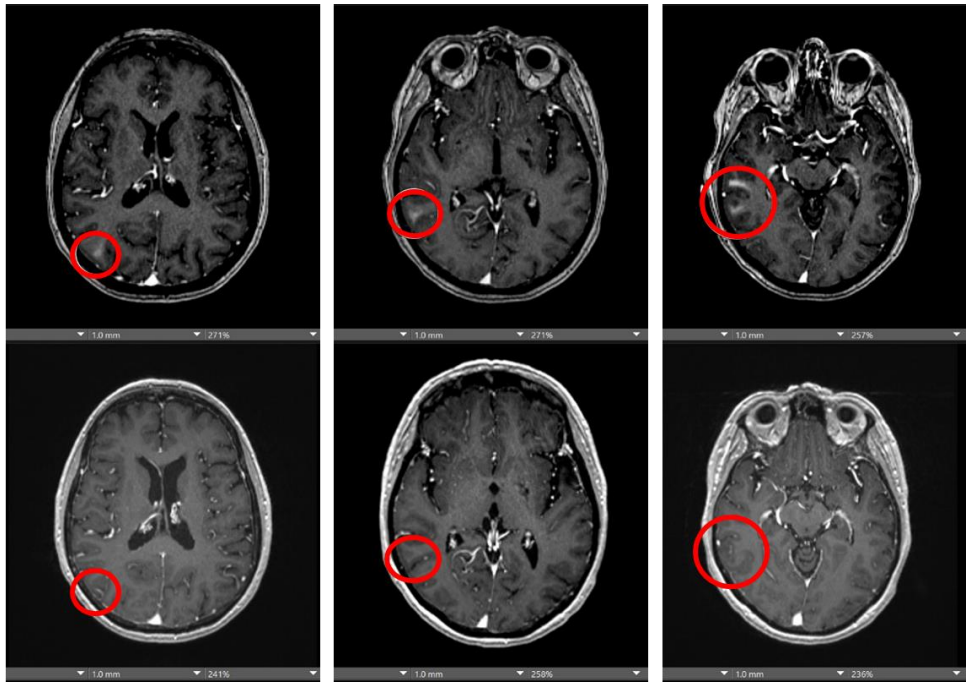
Bexobrutideg – The First "deg" with a Potential Best-in-Class Profile

Novel MOA Against a Clinically and Commercially Proven Target

Only BTK degrader to demonstrate clinical activity in patients with CNS disease including complete responses



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

Novel MOA Against a Clinically and Commercially Proven Target

High objective response rate and prolonged PFS in r/r CLL patients

Efficacy across all doses (50mg – 600mg)	**Phase 1a** (n=47)
Objective response rate (ORR)	**83.0%**
Median progression-free survival (PFS)	22.1 months

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***BTK**, Bruton's tyrosine kinase; **CNS**, central nervous system; **MOA**, mechanism of action; **r/r CLL**, relapsed or refractory chronic lymphocytic leukemia.*

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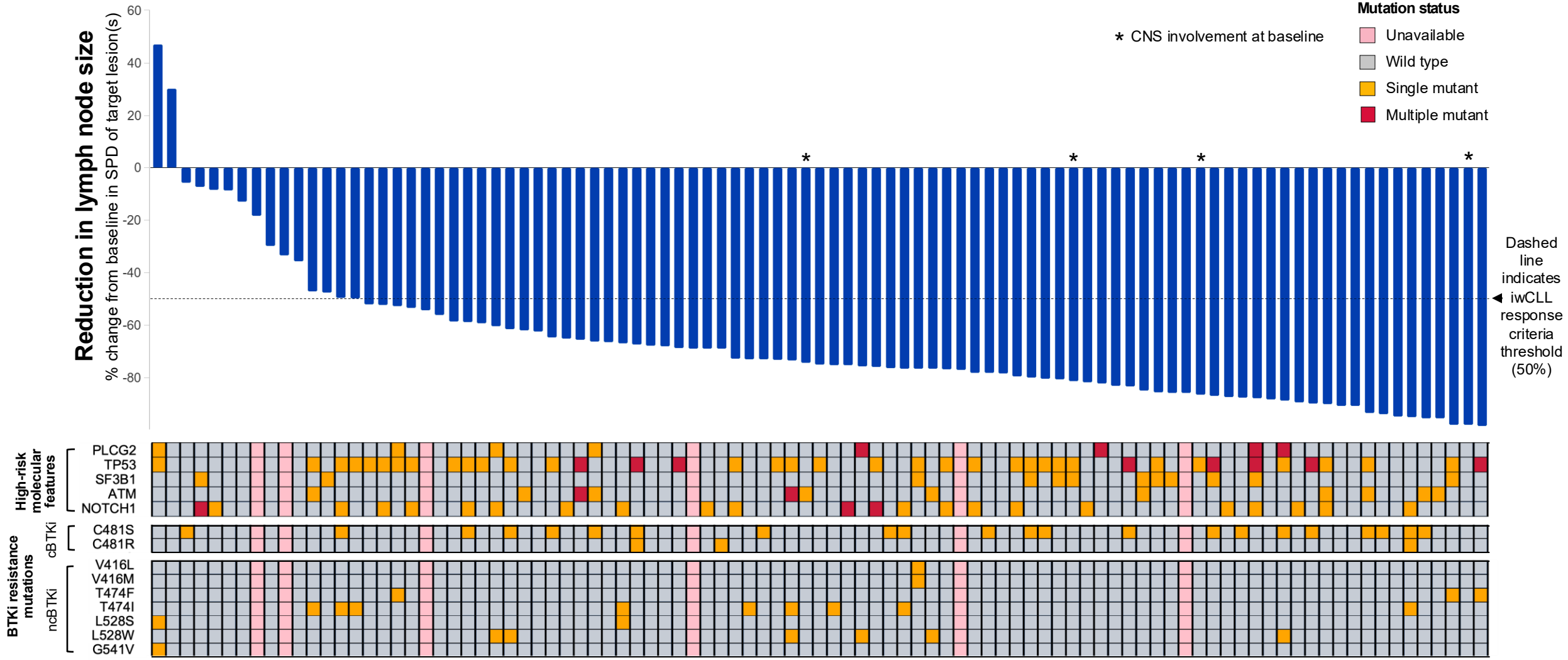
Broad Clinical Activity Across Patients with BTK Mutations, High-Risk Molecular Features and/or CNS Involvement



[a]*Waterfall plot includes patients with measurable lymph node status (n=93); mutations were reported at VAF >5%;* [b]*Patients could have no mutations, a single mutation, or multiple mutations*

Data cutoff: 19 Sep 2025

***ATM**, ataxia-telangiectasia mutated; **BTK**, Bruton's tyrosine kinase; **BTKi**, BTK inhibitor; **cBTKi**, covalent BTKi; **ncBTKi**, non-covalent BTKi; **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*

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Bexobrutideg Demonstrates Deep and Durable Responses in CLL

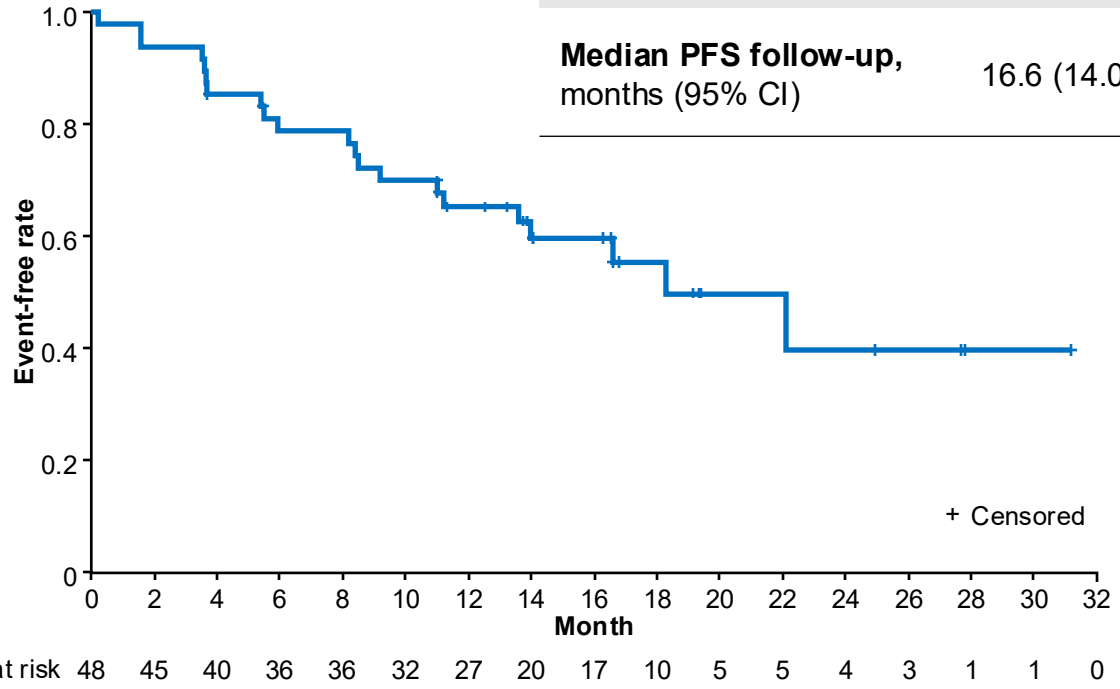
Updated Phase 1a results presented at ASH 2025

Robust Response Rate in Patients with a Median of 4 Prior Lines of Therapy

CLL response-evaluable patients[a]	**Response analysis** (n=47)
Objective response rate (ORR),[b] % (95% CI)	83.0 (69.2–92.4)
Best response, n (%)	
Complete response (CR)	2 (4.3)
Nodal partial response (nPR)	1 (2.1)
Partial response (PR/PR-L)	36 (76.6)
Stable disease (SD)	6 (12.8)
Progressive disease (PD)	2 (4.3)
Median duration of response[c], months (95% CI)	20.1 (12.2–NE) (n=39)

Long-term Disease Control with a Median PFS of 22.1 Months Across All Doses Tested (50mg – 600mg, n=48)

Median PFS, months (95% CI)	22.1 (11.2–NE)
Median PFS follow-up, months (95% CI)	16.6 (14.0–19.3)

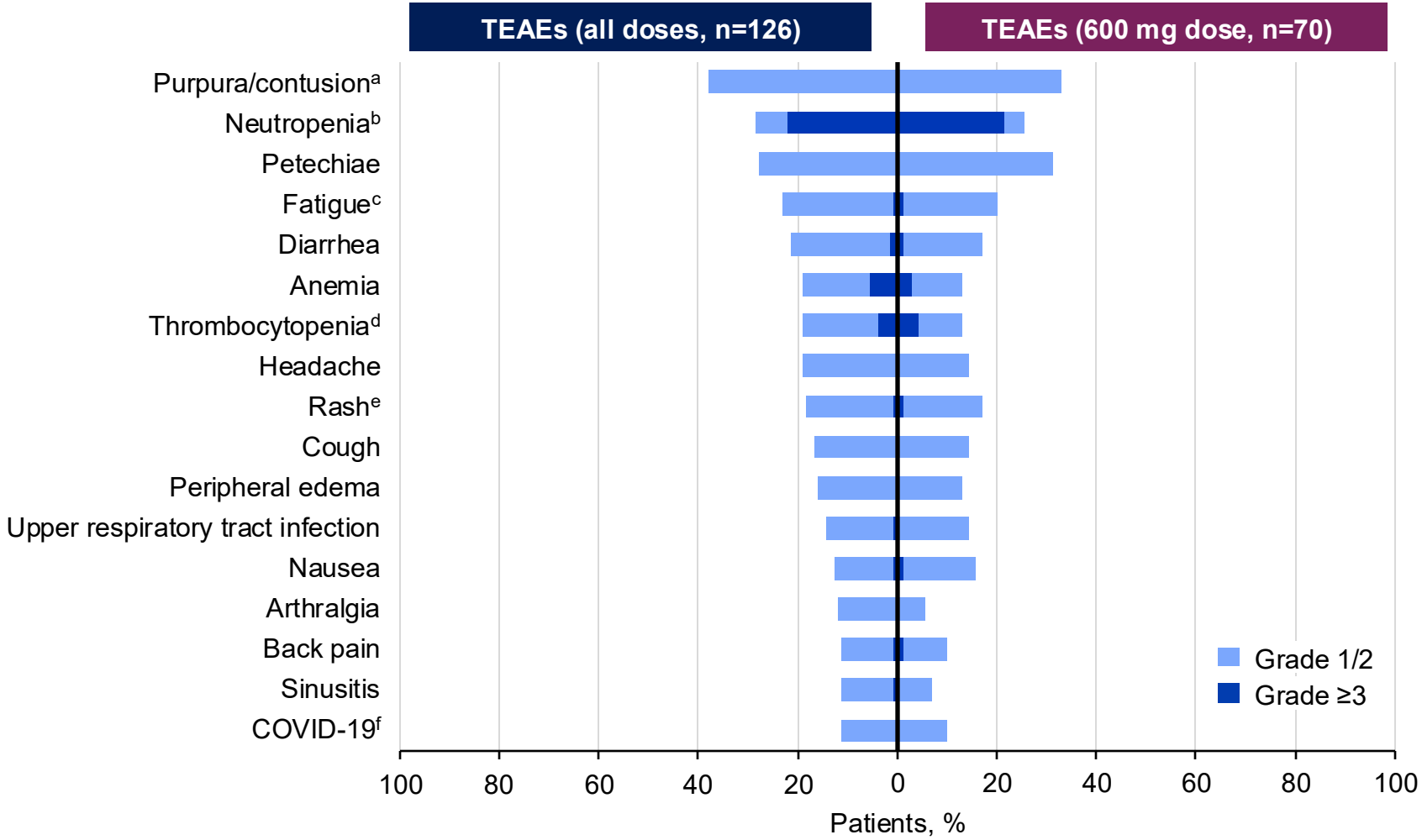


Data cutoff: 19 Sep 2025

[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

Bexobrutideg Is Well Tolerated in Patients with Relapsed/Refractory CLL

Comparable AE profile for patients overall and at the 600 mg dose selected per Project Optimus



- Tolerable safety profile
- No dose-limiting toxicities
- No systemic fungal infections or Grade 4 infections of any kind reported
- Single event of new onset atrial fibrillation consistent with the rate in the age-matched general population
- Three Grade 5 AEs (all deemed not related to bexobrutideg)

[a]*Purpura/contusion includes episodes of contusion or purpura;* [b]*Aggregate of 'neutrophil count decreased' or 'neutropenia';* [c]*Fatigue was transient;* [d]*Aggregate of 'thrombocytopenia' and 'platelet count decreased';* [e]*Aggregate of 'rash' and 'rash maculopapular' and 'rash pustular';* [f]*Aggregate of 'COVID-19' and 'COVID-19 pneumonia'*
***AE**, adverse event;* ***RP2D**, recommended Phase 2 dose;* ***TEAE**, treatment-emergent adverse event*

Data cutoff: 19 Sep 2025

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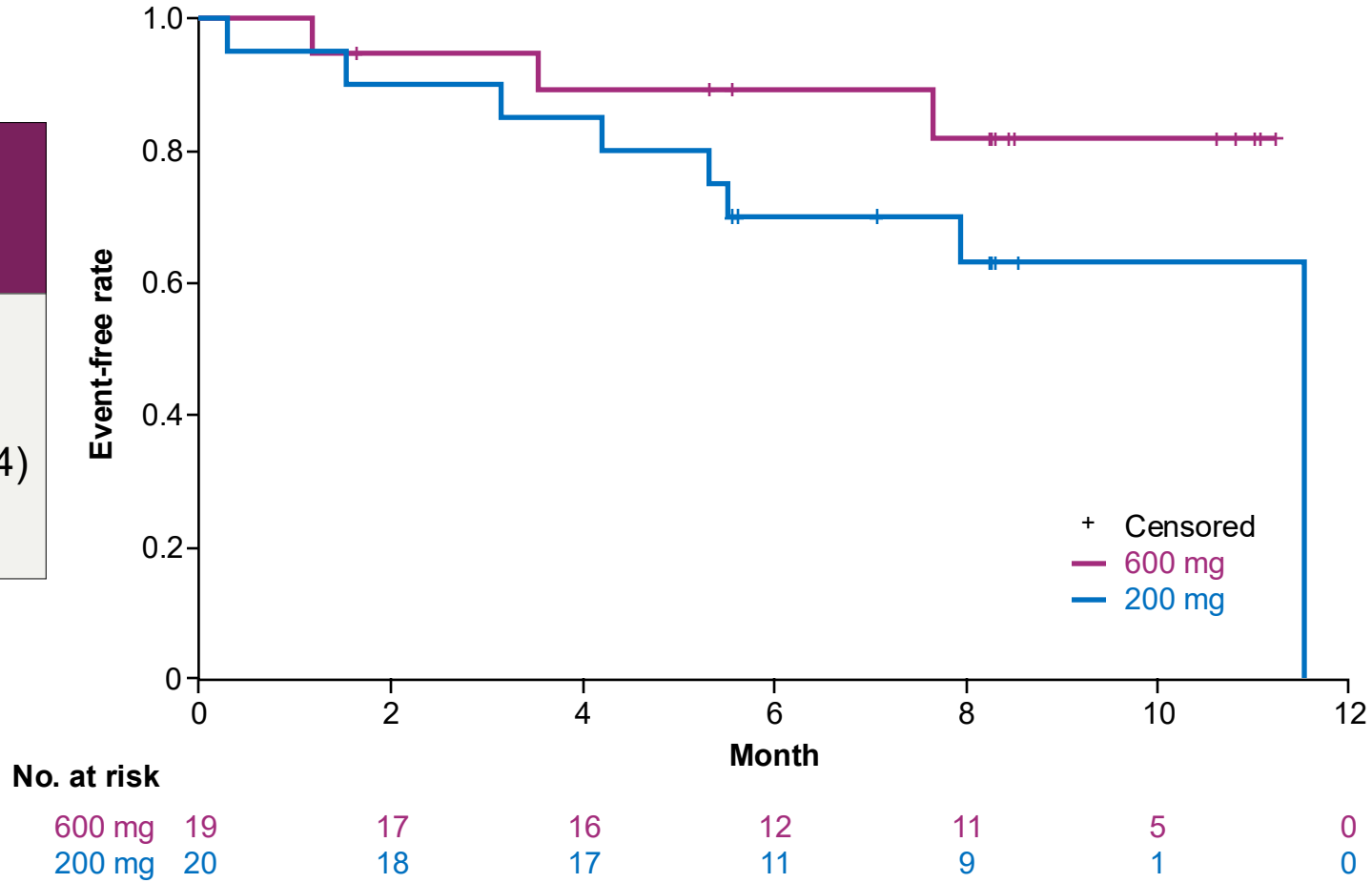
Higher ORR and PFS Observed at 600 mg Dose Selected for Pivotal Trials

Preliminary efficacy in Phase 1b randomized cohort of 200 mg vs 600 mg

Response-evaluable patients	200 mg (n=19)	600 mg (n=18)
Objective response rate,[a] % (95% CI)	73.7 (48.8–90.9)	83.3 (58.6–96.4)



[a]*Objective response rate includes CR + nPR + PR + PR-L*

***CI**, confidence interval; **ORR**, objective response rate; **PFS**, progression-free survival*

Data cutoff: 19 Sep 2025

Latest Bexobrutideg Data Supports a Best-in-Class Pivotal Trial Strategy

Endpoint	Bexobrutideg	Pirtobrutinib (FDA full approval 12/3/2025)
Objective Response Rate (ORR)	**83.0%**	65% (69% investigator)
Median Duration of Response (DOR)	**20.1 months**	13.8 months (13.9 investigator)
Median Progression-Free Survival (PFS)	**22.1 months**	14.0 months
Study	NX-5948-301 (Phase 1a)	BRUIN-321 (vs BR/IR)

Bexobrutideg was evaluated in a more heavily pretreated population than pirtobrutinib:

- Median prior lines of therapy: **4 vs 3**
- ≥4 prior lines of therapy: **56% vs 33%**
- Prior non-covalent BTK inhibitor exposure: **27% vs 0%**
- Prior BCL-2 inhibitor exposure: **83% vs 50%**

Source: NX-5948-301 study data cut off Sept 19, 2025, and Sharman et al, JCO 43: 2538-2549, June 2025

***CLL,** chronic lymphocytic leukemia; **BTK**, Bruton's tyrosine kinase; **BCL-2i**, BCL-2 inhibitor*

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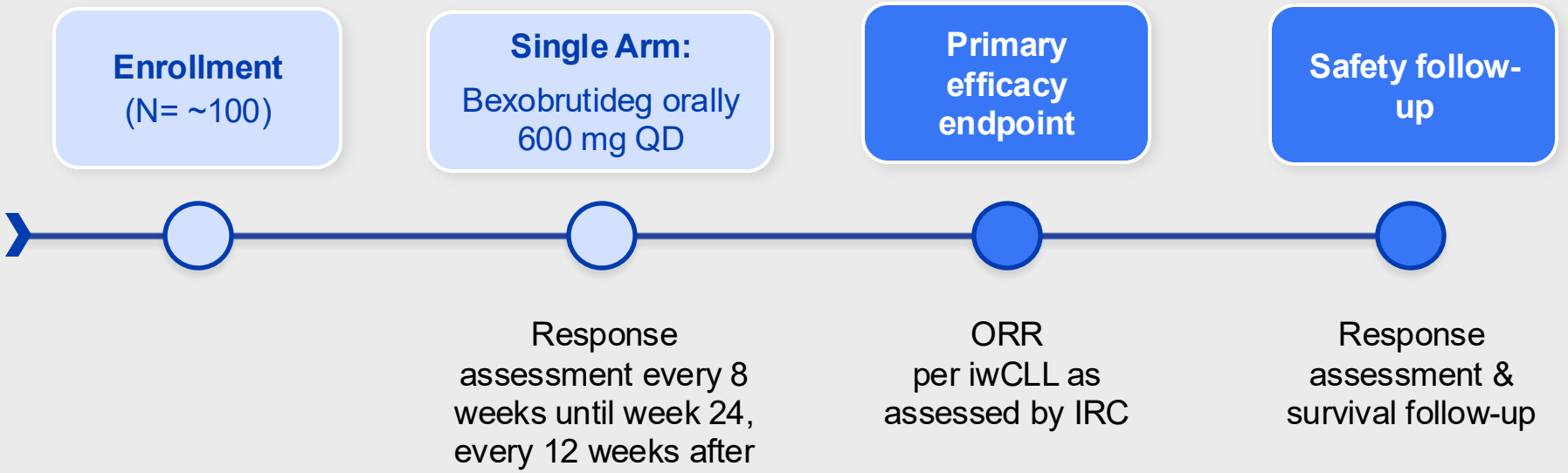
DAYBreak Pivotal Phase 2 Single-Arm Study Designed to Support Bexobrutideg Accelerated Approval

DAYBreak CLL-201

TRIAL DESIGN

Key Eligibility

- R/R CLL/SLL
- Triple-exposed (post-cBTKi, ncBTKi & BCL-2i)



- ✓ First patient dosed in October 2025
- ✓ 600 mg cleared for pivotal studies in R/R CLL

r/r CLL, *relapsed or refractory 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*

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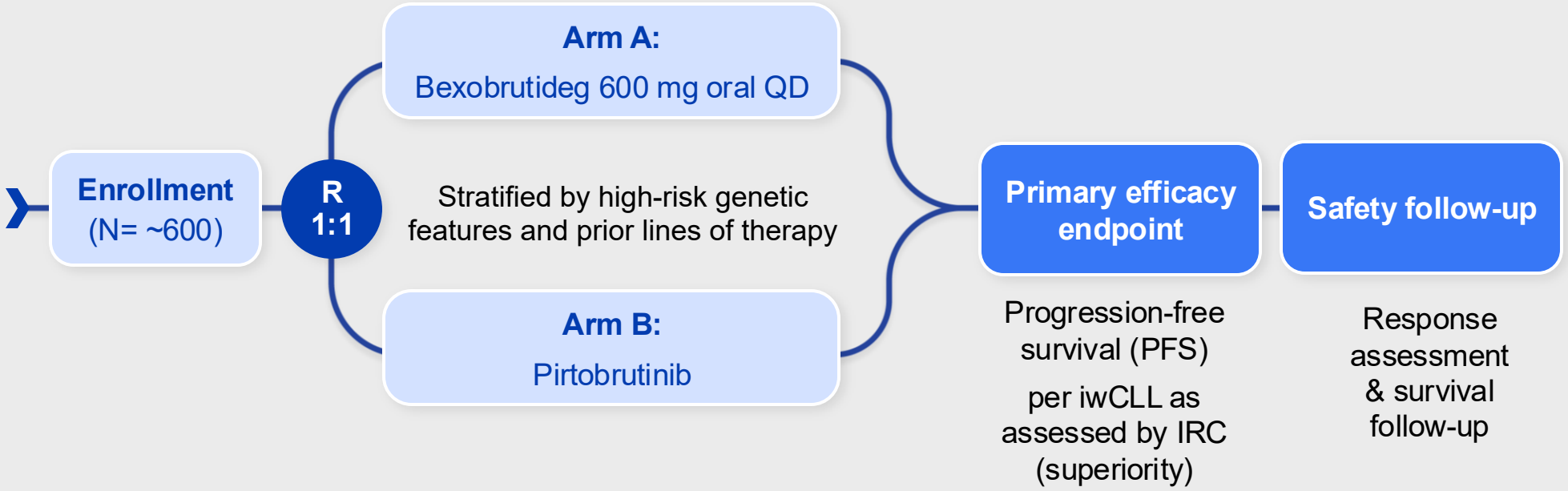
DAYBreak Phase 3 Confirmatory Trial Positions Bexobrutideg for Full Approval

DAYBreak CLL-306

TRIAL DESIGN

Key Eligibility

- 2L+ R/R CLL/SLL
- Prior cBTKi



- ✓ Designed to evaluate superiority to latest approved BTKi, pirtobrutinib
- ✓ Single trial strategy to support global approval and establish superiority of degrader mechanism of action

Note: *Final design pending regulatory feedback*

r/r CLL, *relapsed or refractory chronic lymphocytic leukemia;* ***PFS,*** *progression-free survival;* ***iwCLL,*** *International Workshop on CLL;* ***IRC,*** *Independent Review Committee;* ***QD,*** *once daily;* ***SLL,*** *small lymphocytic lymphoma;* ***cBTKi****, covalent BTK inhibitor*

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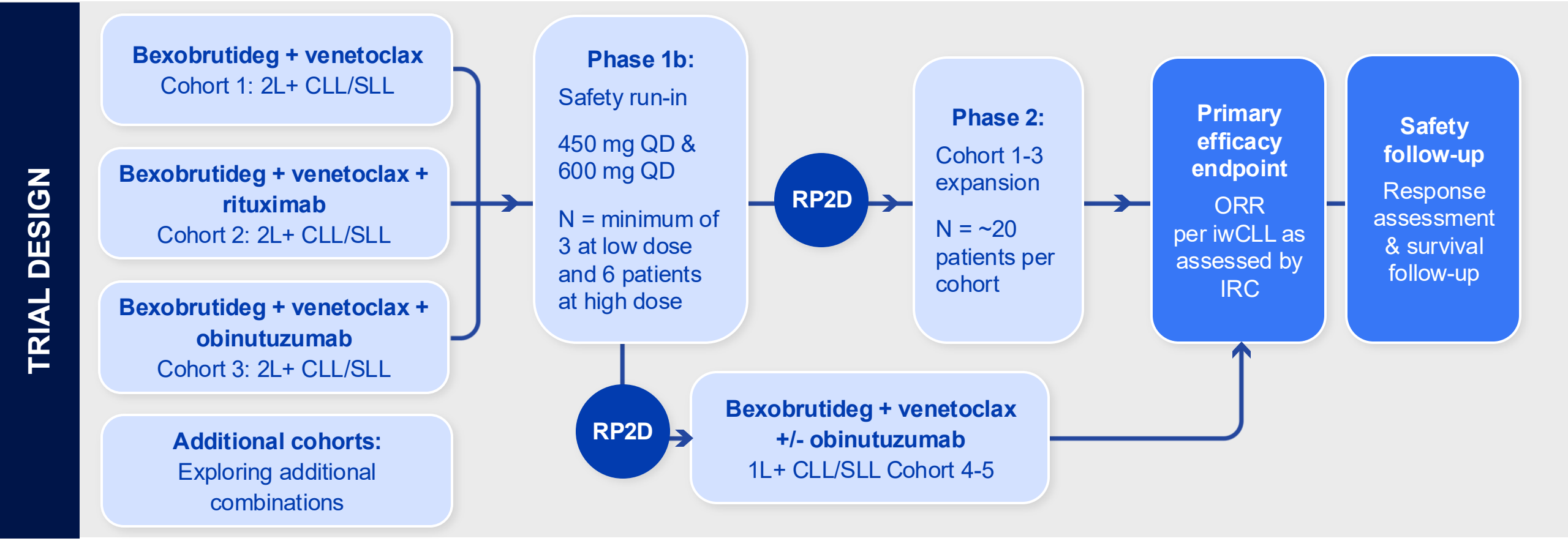
NX-5948-203: Phase 1b/2 Combination Study to Address Emerging Treatment Standards in CLL



- ✓ Combination regimen of bexobrutideg + BCL-2i maximizes 2L market share opportunity
- ✓ Strategy 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*

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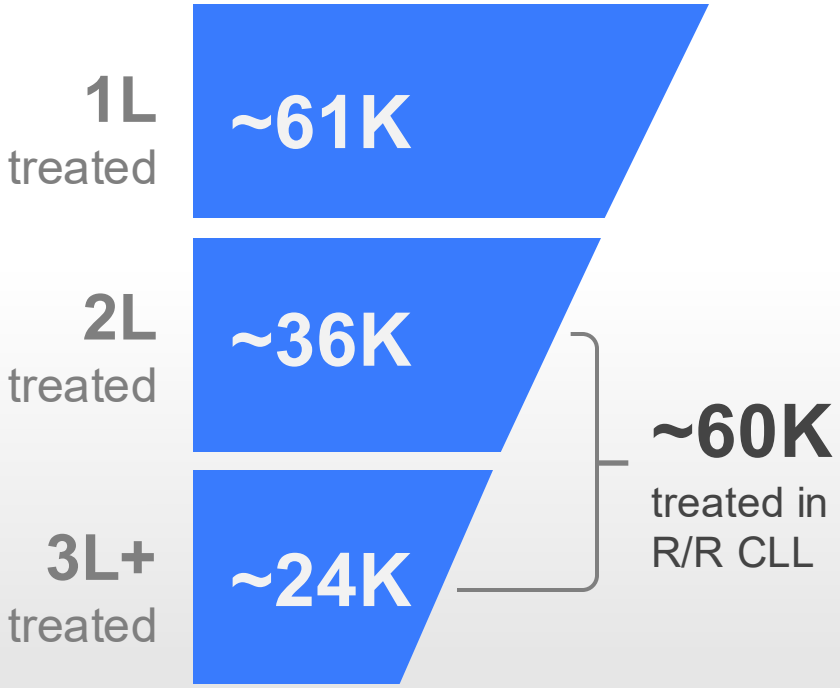
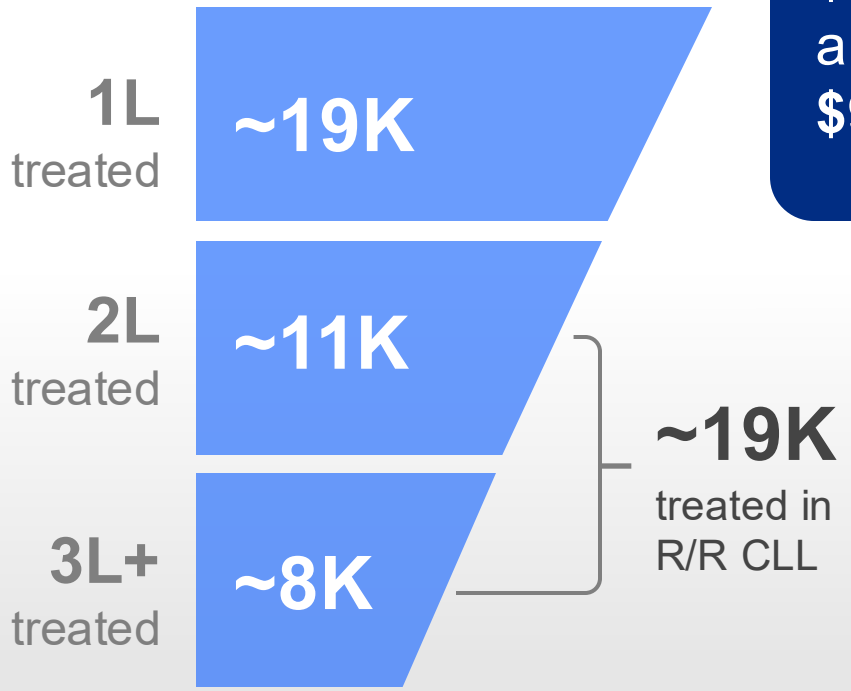
Nurix Has a Clinical Development Plan Addressing Large Segments of the CLL Market as Both a Mono- and Combo- Therapy

Drug-Treated Incidence in Major Markets
US, Canada, Europe, Japan, China



US Drug-Treated Incidence



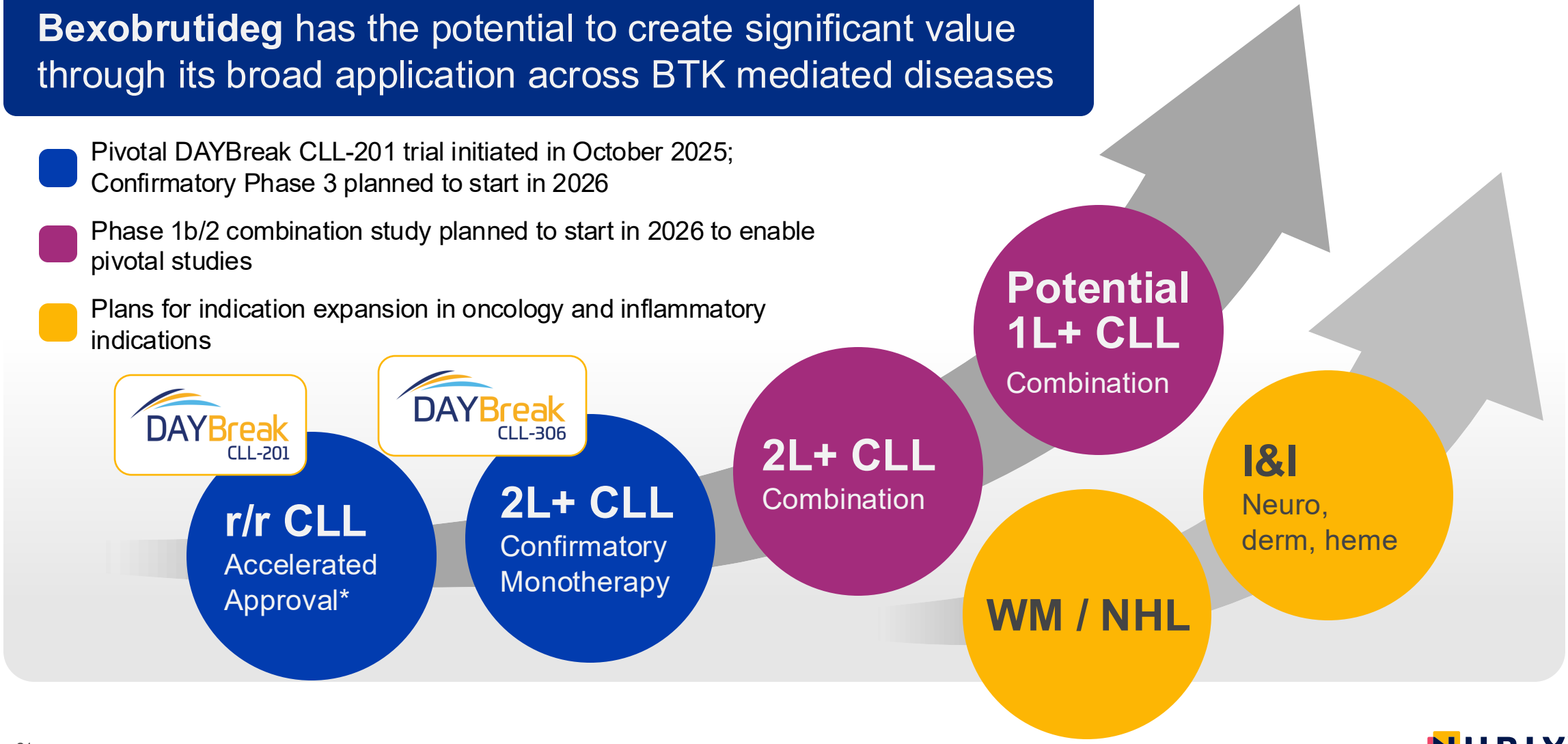
Current BTK inhibitor sales annualizing at **$12.5 billion** with approximately **$9.5 billion** in CLL

Sources for US, EU4+UK, and Japan: [1] Clarivate / DRG Landscape and Forecast Research Report NHL and CLL, April 2023
For Canada, we assume similar drug treated incidence as US and adjust for the smaller Canadian population; For Europe, we adjust EU4+UK drug treated incidence for the total Europe population; For China, we assume lower drug treated incidence based on literature reference: https://ashpublications.org/ashclinicalnews/news/1473/Disease-Detectives-CLL-in-Asia

Unlocking Waves of Clinical Benefit and Value Creation

Bexobrutideg has the potential to create significant value through its broad application across BTK mediated diseases

- Pivotal DAYBreak CLL-201 trial initiated in October 2025; Confirmatory Phase 3 planned to start in 2026
- Phase 1b/2 combination study planned to start in 2026 to enable pivotal studies
- Plans for indication expansion in oncology and inflammatory indications



** Phase 2 single-arm study designed to support Accelerated Approval*

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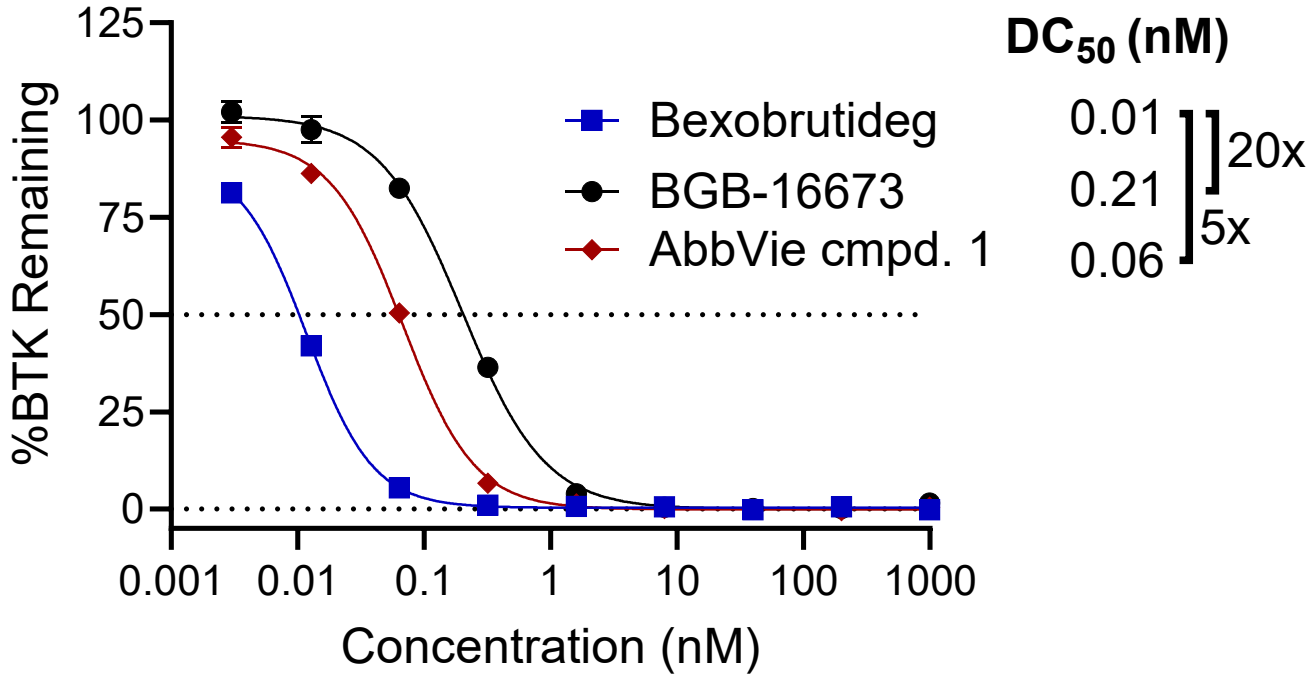
Potential Best-in-Class BTK Degrader: More Potent, Better Coverage, and Superior Selectivity

Bexobrutideg Displays Best-in-Class BTK Degradation Potency

BTK Degradation in Human B Cells



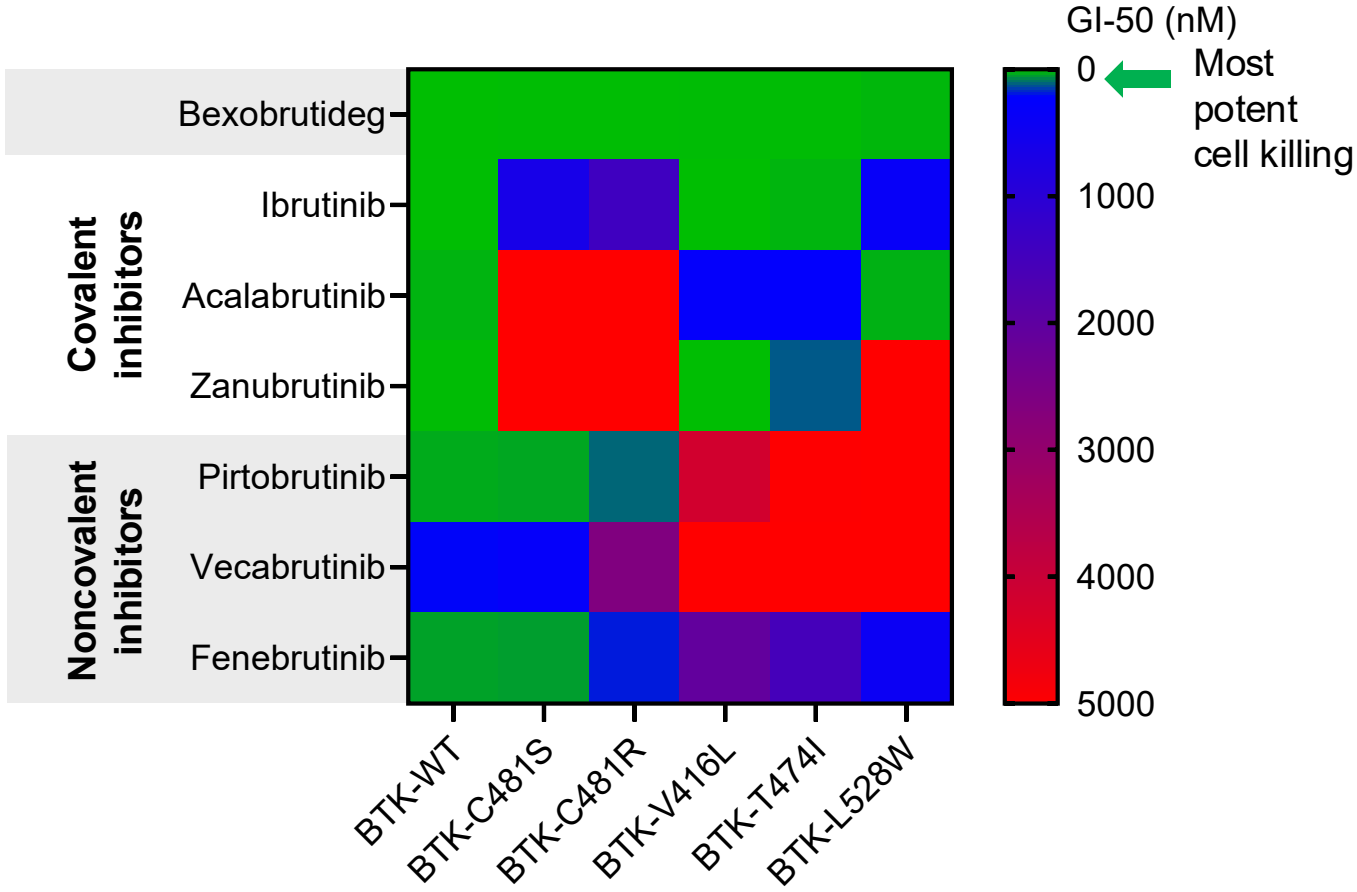
Bexobrutideg is **20x** more potent than BGB-16673 and **5x** more potent than AbbVie cmpd. 1

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

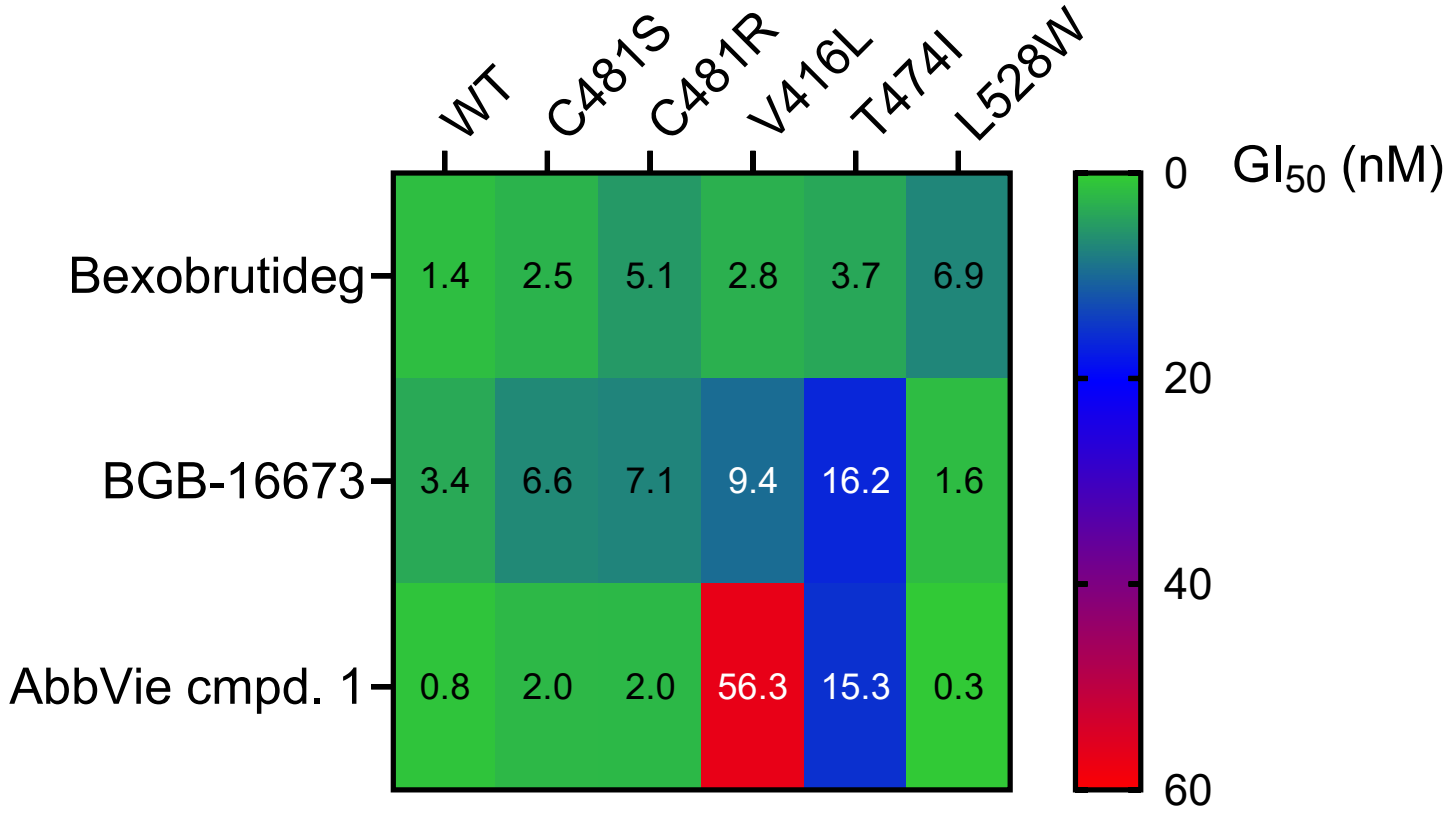


***BTK**, Bruton's tyrosine kinase; **GI-50** growth inhibition 50%; **WT** wildtype*

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

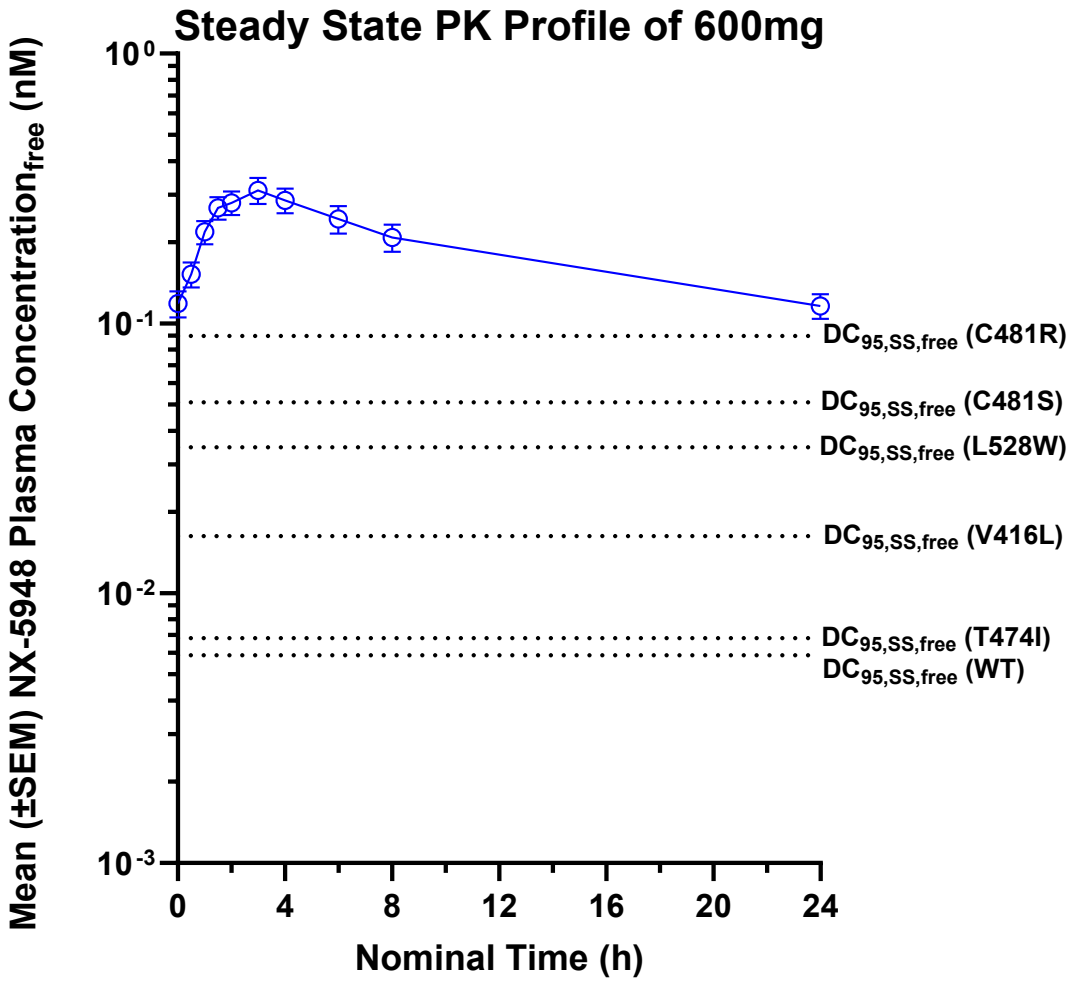
Bexobrutideg demonstrates GI_{50} values of <10 nM across relevant mutations, while BGB-16673 and AbbVie cmpd. 1 display potential liabilities

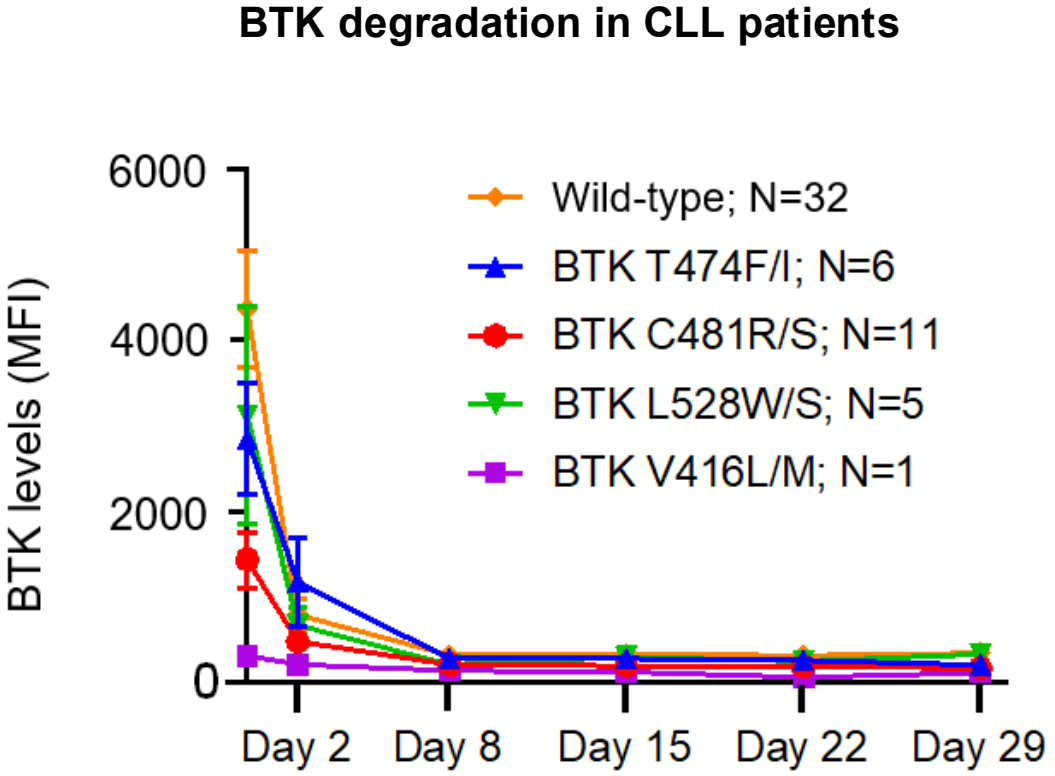
Cell Killing Activity Across Clinically Relevant Mutations



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



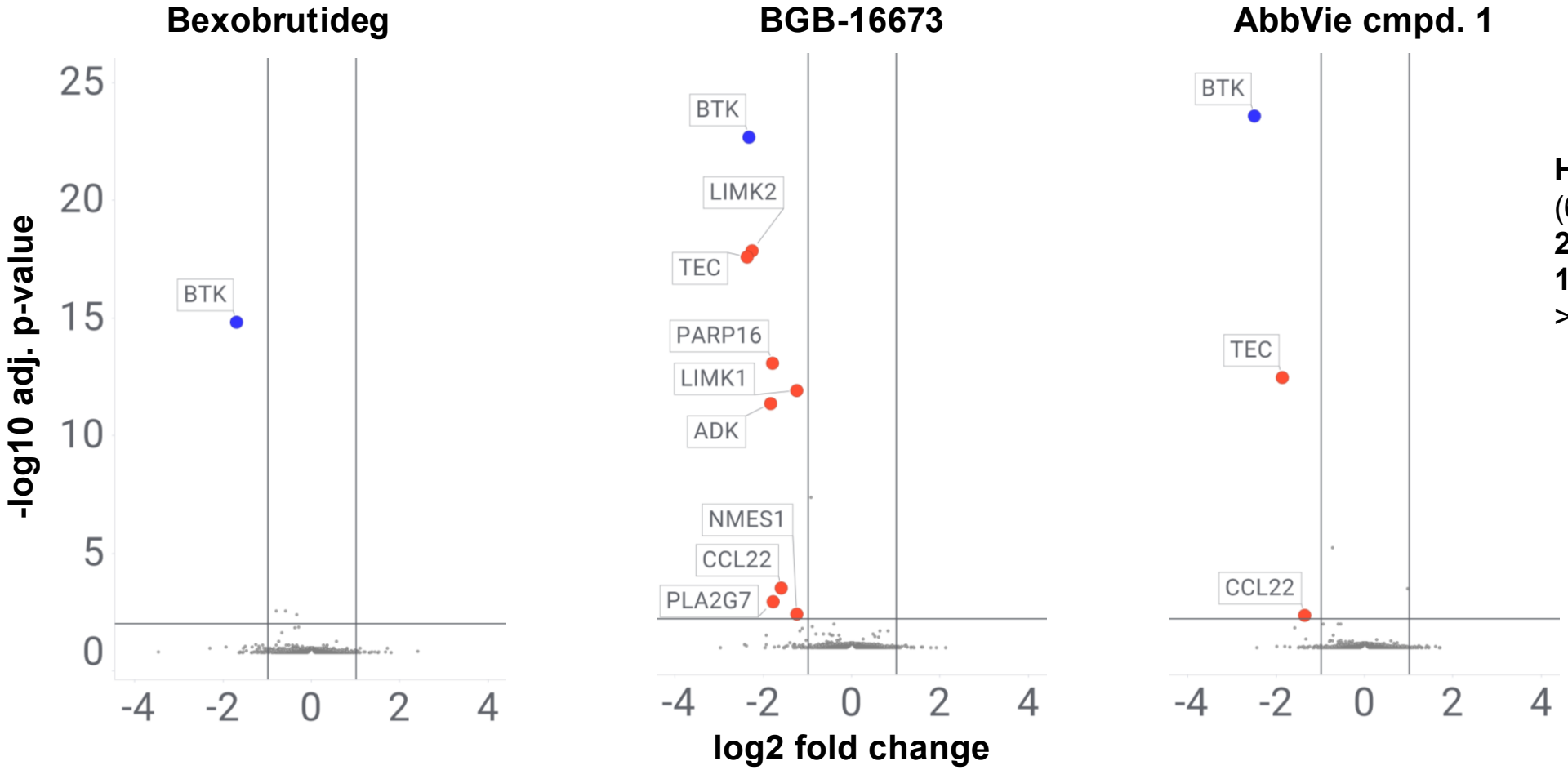


Note: Some patients have multiple BTK mutations

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

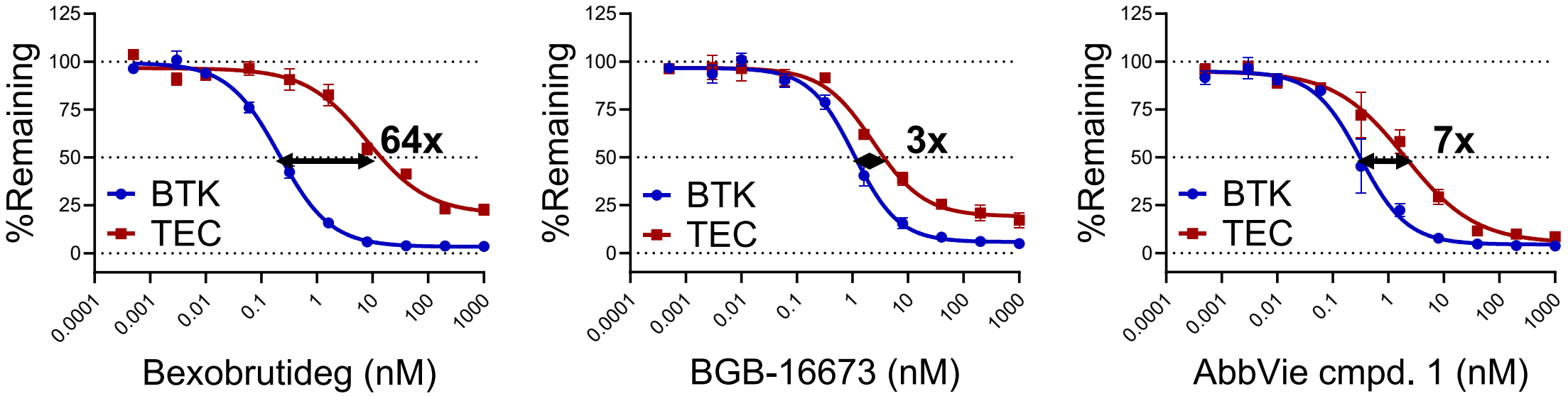
***PBMC**, peripheral blood mononuclear cell*

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]

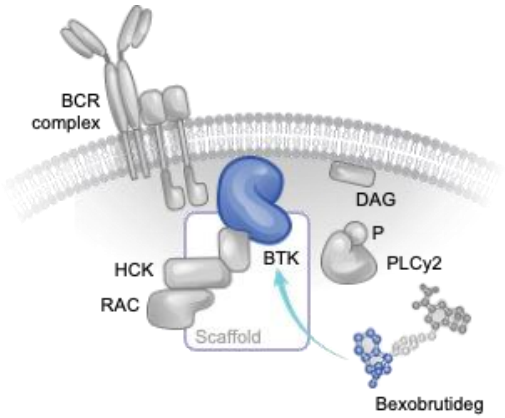
BTK degradation assessed by flow cytometry in human PBMCs (hPBMCs), gated on CD20+ B cells. LCK/CSK/ADK DC_{50} values are compared to BTK DC_{50} in primary B cells, TEC DC_{50} values are compared to BTK DC_{50} in K562 cells. LCK degradation by bexobrutideg assessed by Jess SimpleWestern in bulk hPBMCs. 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

Nurix's Industry-Leading Wholly Owned and Partnered Degrader Portfolio in Inflammation and Autoimmune Diseases

BTK

B-cell & myeloid cell-driven inflammation



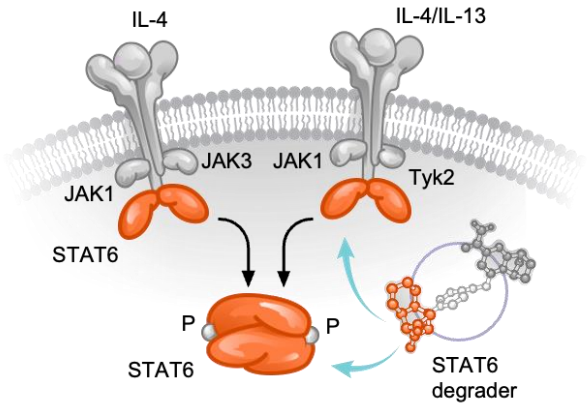
Bexobrutideg opportunity
(wholly owned)

>6M patients[1]
Including CSU, HS, MS, wAIHA

>$18B annual sales[2]

STAT6

Type 2 inflammation



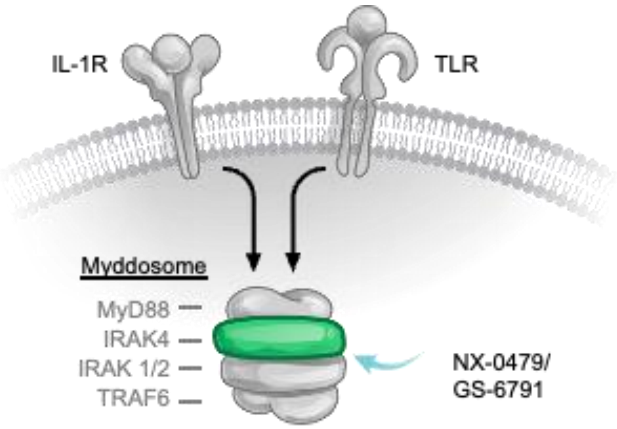
NX-3911 opportunity
(partnered with Sanofi)

>125M patients[1]
Including asthma, AD, BP, CSU, COPD, CRwNP, EoE, PN

>$34B annual sales[2]

IRAK4

IL-1R/TLR-driven inflammation



NX-0479/GS-6791 opportunity
(partnered with Gilead)

>110M patients[1]
Including asthma, AD, HS, PsO, PsA, RA, SLE, CLE, CD, UC

>$100B annual sales[2]

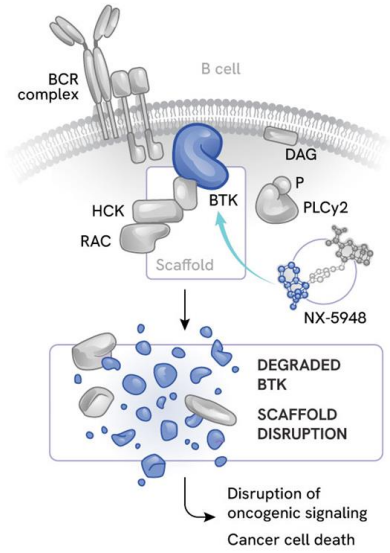
[1]Patient estimate across key indications in U.S., EU5 and JP (2023); [2]Annual sales across modalities (2023)
Sources: GlobalData, Lumanity analysis, Evaluate Pharma, IQVIA Institute for Human Data Science, Global Use of Medicines: Outlook to 2028, January 2024.
Indication abbreviations defined in notes section in the back of the deck.

Three Blockbuster Targets Addressing Different Critical Pathways in Inflammation

Program	Target	Modality	Therapeutic area	Discovery	IND-Enabling	Phase 1A	Phase 1B/2	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

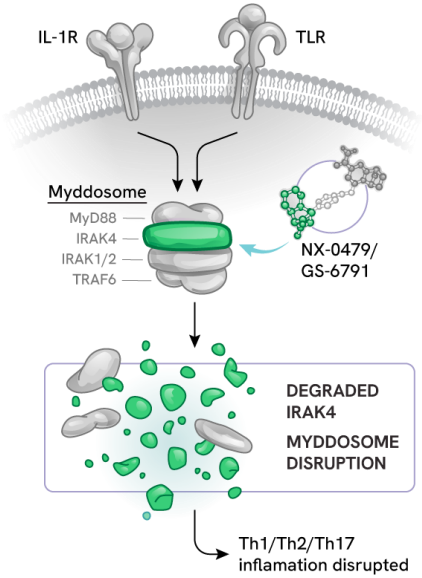
BTK

A critical node in controlling signaling through the B cell receptor and Fc receptors.



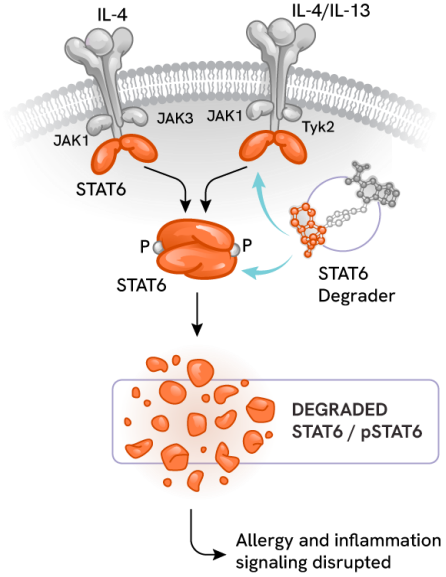
IRAK4

A master regulator of the Toll-like receptor and interleukin-1 receptor signaling pathways



STAT6

A transcription factor that mediates type 2 inflammation

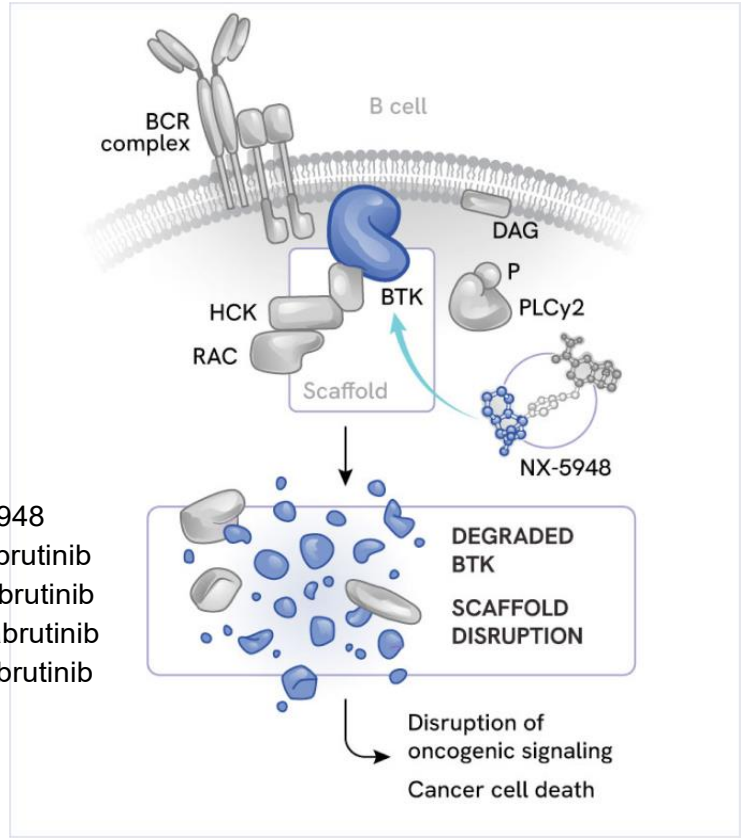


NURIX

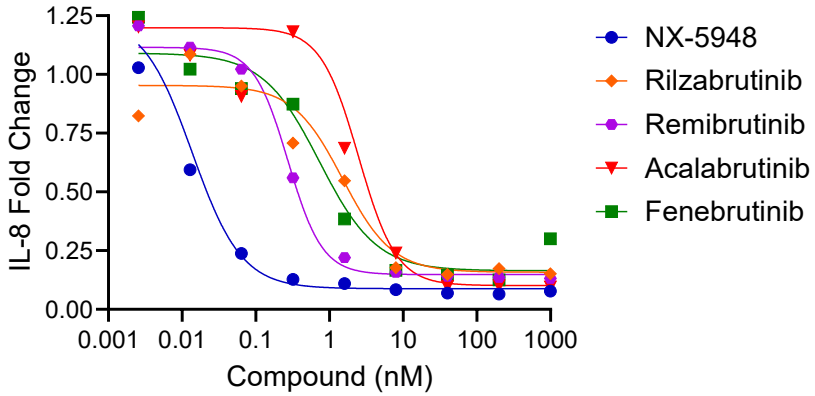
Advancing Bexobrutideg in Immunology and Inflammation

Key observations underpinning Nurix's bexobrutideg I&I strategy

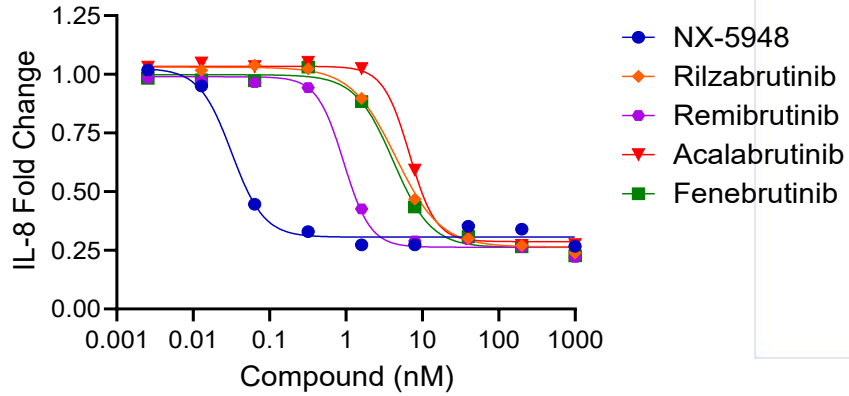
- Human and mouse knockouts are associated with reduced immune function yet have otherwise normal physiology
- BTK inhibitors have shown positive clinical results across a wide range of I&I diseases in hematology, dermatology, and neurology
- The same scaffolding functions that limit efficacy of inhibitors in oncology may also be limiting their efficacy in autoimmune disease settings



Fc Receptor Pathway



Inflammatory Signaling Pathway

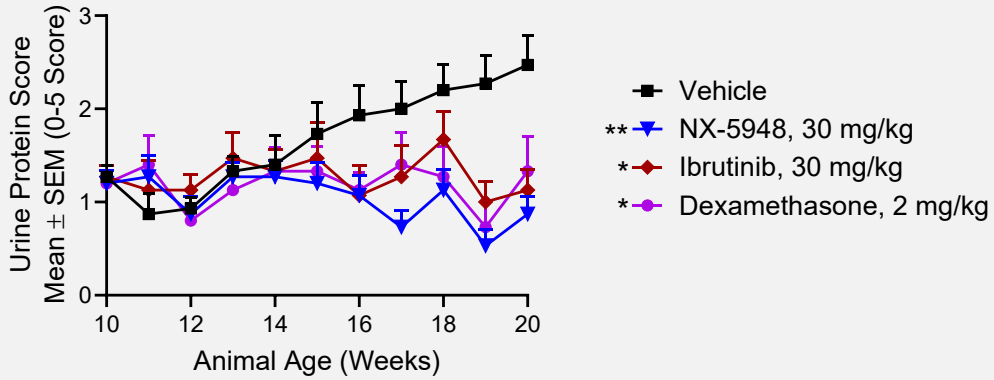


IL-8 is a chemokine that recruits neutrophils into inflamed tissues

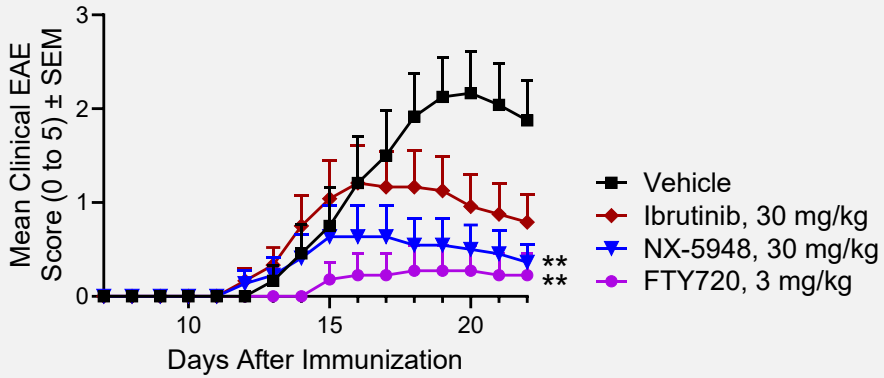
***BTK**, Bruton's tyrosine kinase; **I&I**, inflammation and immunology; **LPS**, lipopolysaccharide; **TLR**, toll-like receptor; **BCR**, B cell receptor; **ssRNA/DNA**, single-stranded RNA/DNA*

Bexobrutideg Is Active Across a Range of Autoimmune and Inflammation Models

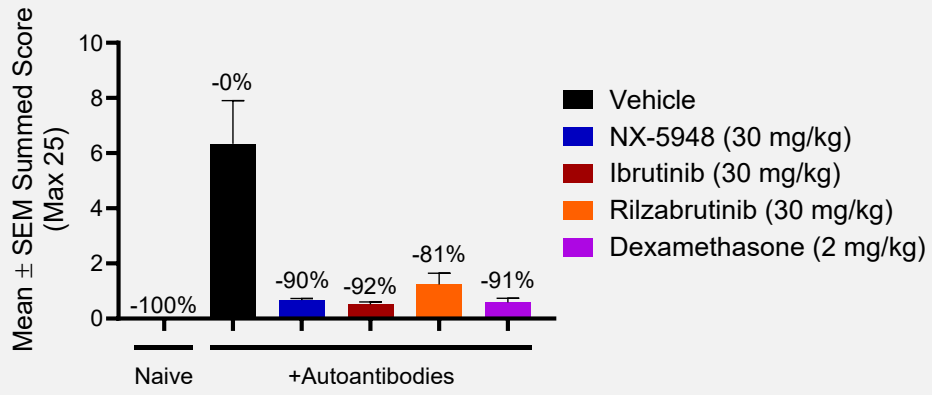
Systemic Lupus Erythematosus (MRL/lpr Model)



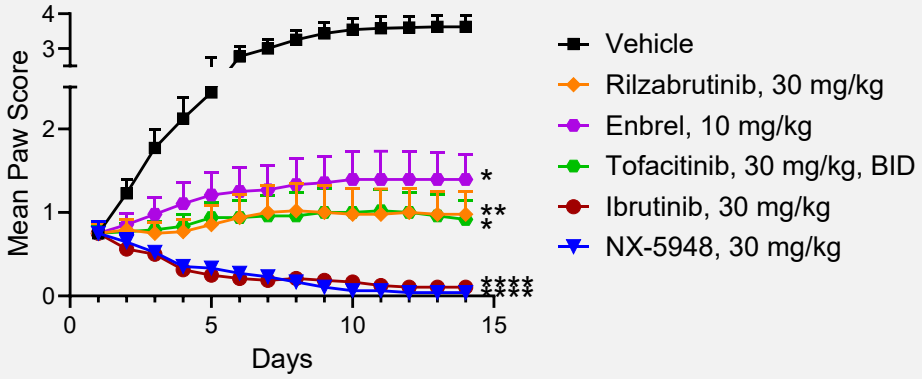
Multiple Sclerosis (EAE Model)



Lupus Nephritis (AIG Model)



Rheumatoid Arthritis (Established CIA Model)



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

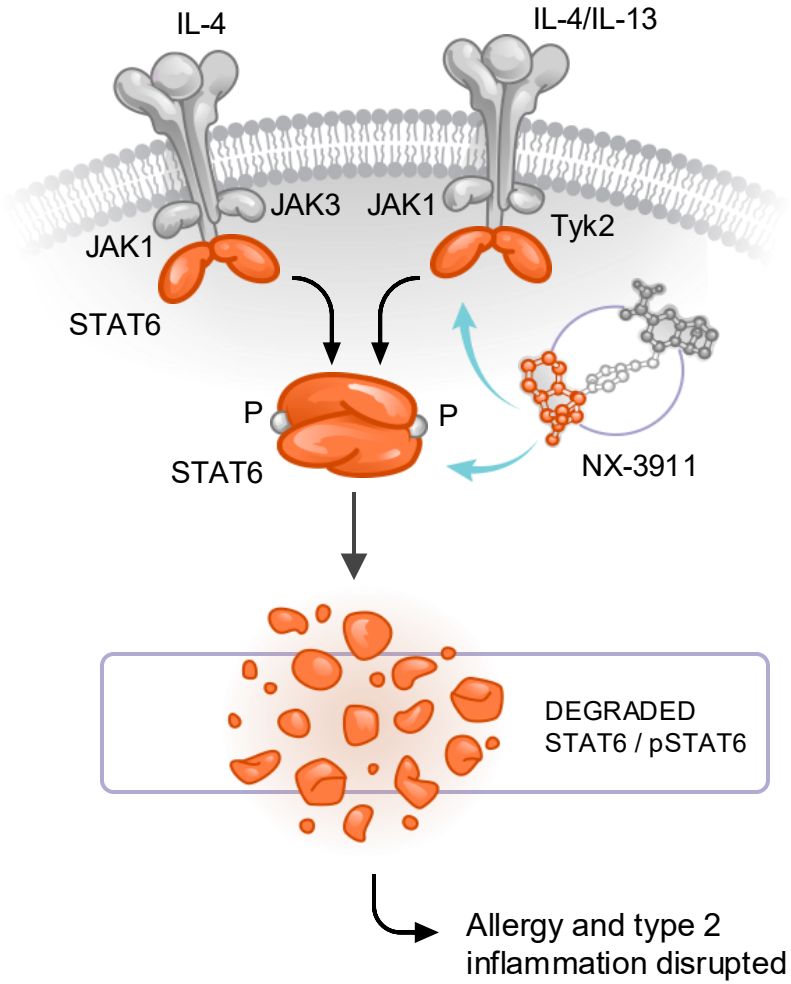
NX-3911: A Potential Best-in-Class STAT6 Degrader in Collaboration with Sanofi that Achieves Complete STAT6 Pathway Blockade

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

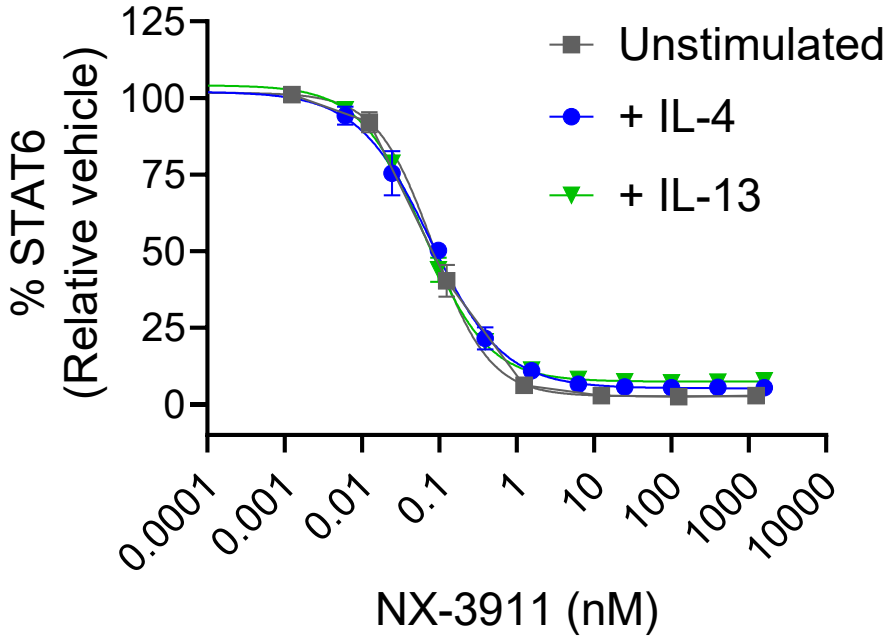
***JAK**, Janus kinase; **STAT**, Signal Transducer and Activator of Transcription*

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*

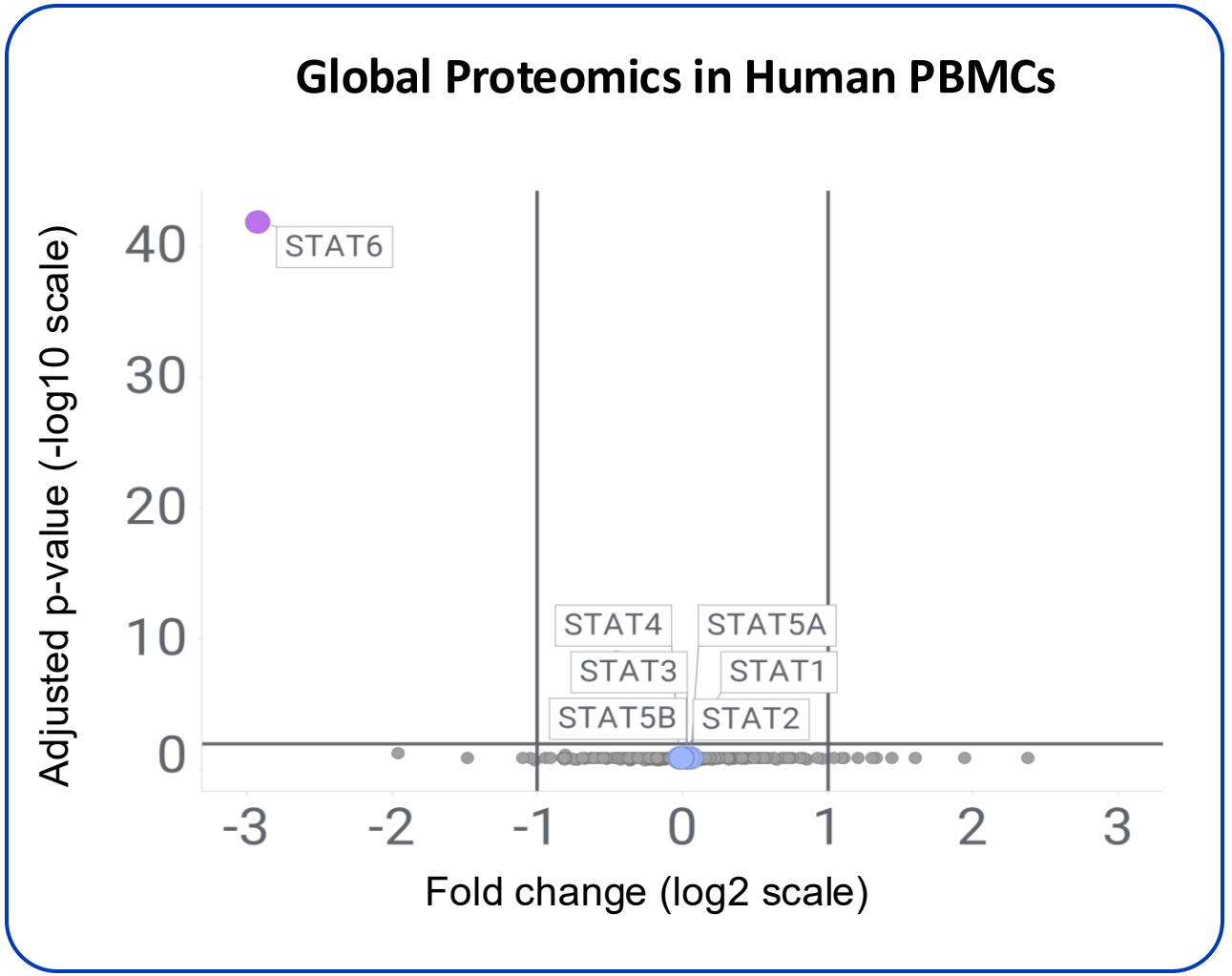
NX-3911 Degrades STAT6 With Exquisite Selectivity

- 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)



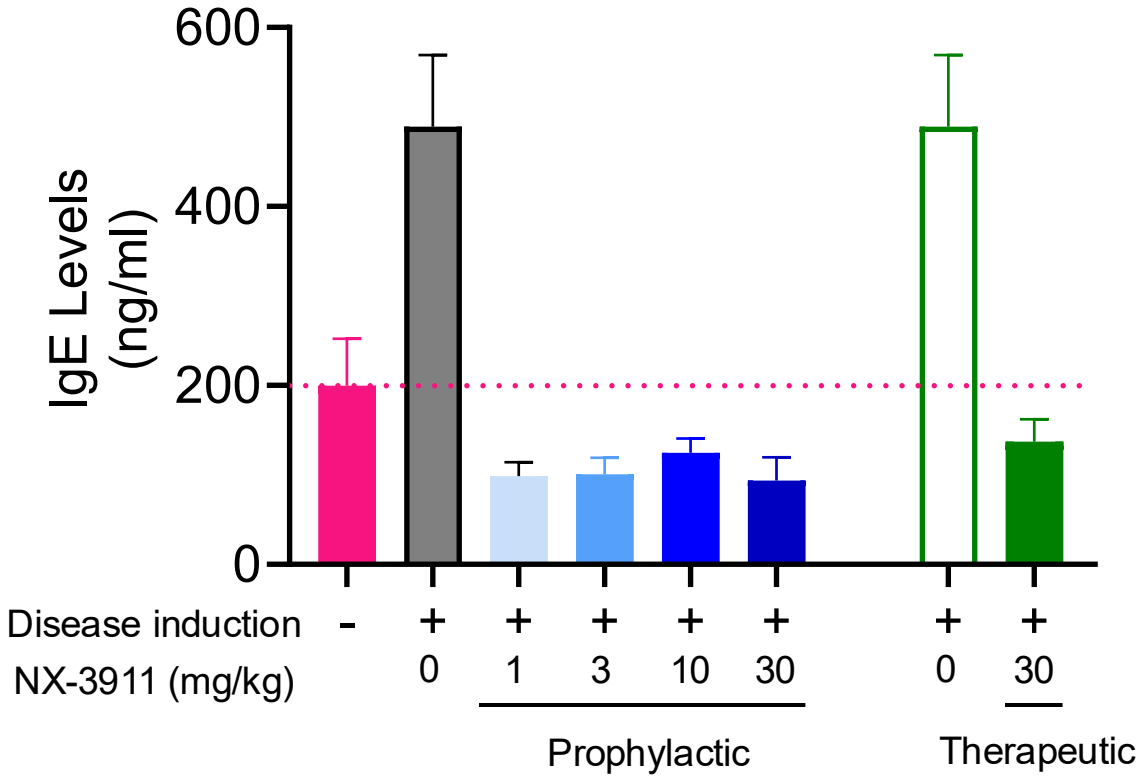
***PBMC**, peripheral blood mononuclear cell*

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

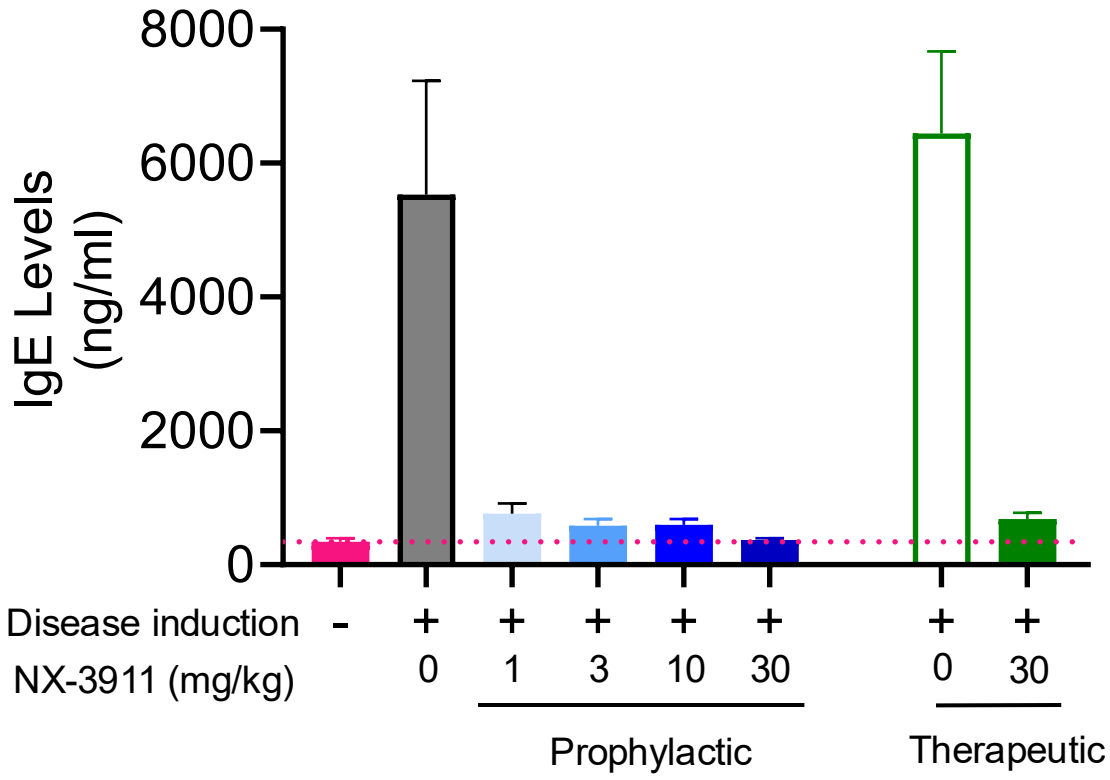
Atopic Dermatitis Model

Suppression of IgE production



Asthma Model

Suppression of IgE production



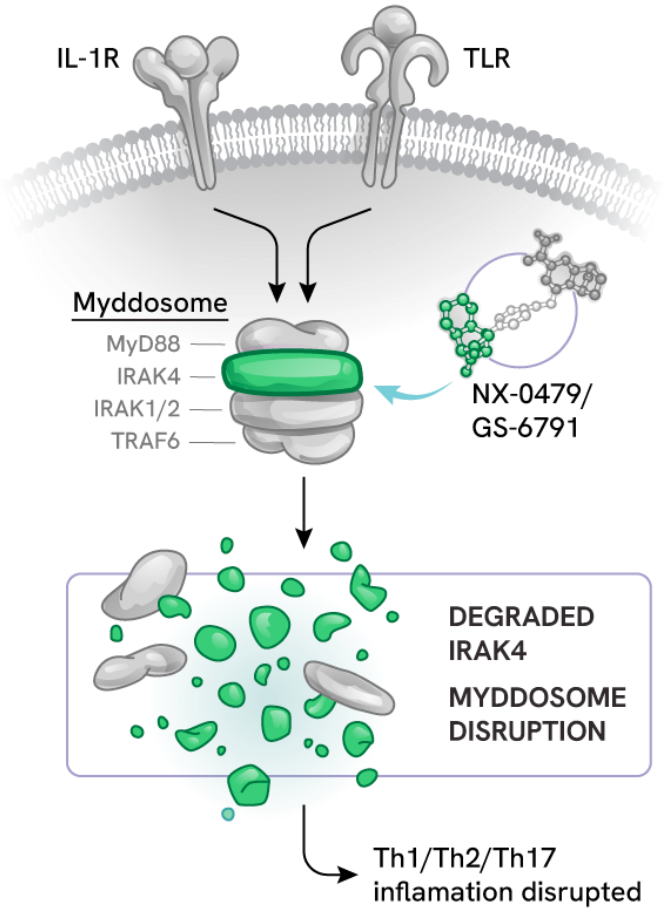
Disease induced by MC903 in Atopic Dermatitis and House Dust Mite in Asthma models in hIL-4/IL-4Rα/hIL-13 mice (C57BL/6 background)
Red dotted line represents the average IgE levels observed in naïve mice.
All treated groups are statistically significant ($P<0.05$) compared to vehicle control (0 mg/kg)

NURIX

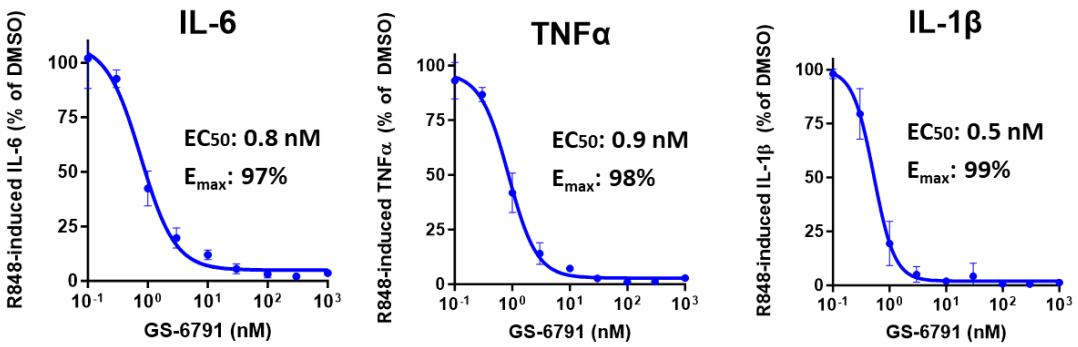
IRAK4 Degrader NX-0479/GS-6791: Potential Treatment for Rheumatoid Arthritis and Other Inflammatory Diseases

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

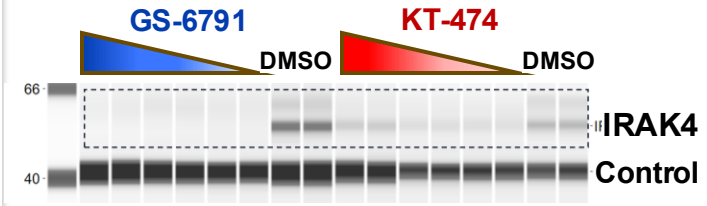


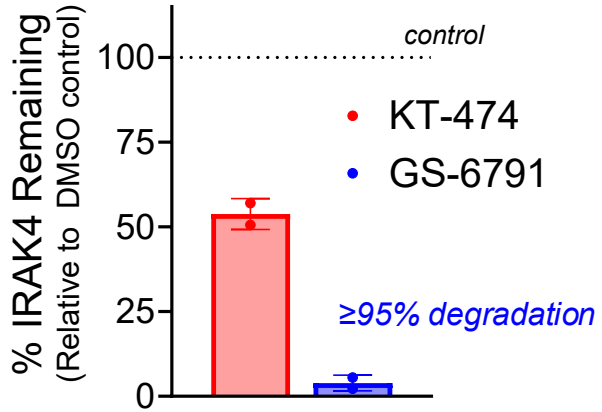
Source: Teng et al., ACR 2024, November 2024 .

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

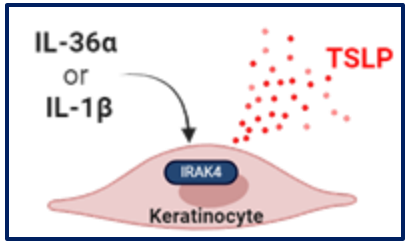
NX-0479/GS-6791 Elicits Best-in-Class IRAK4 Degradation and Inhibition of Functional Responses in Skin Epithelial Systems

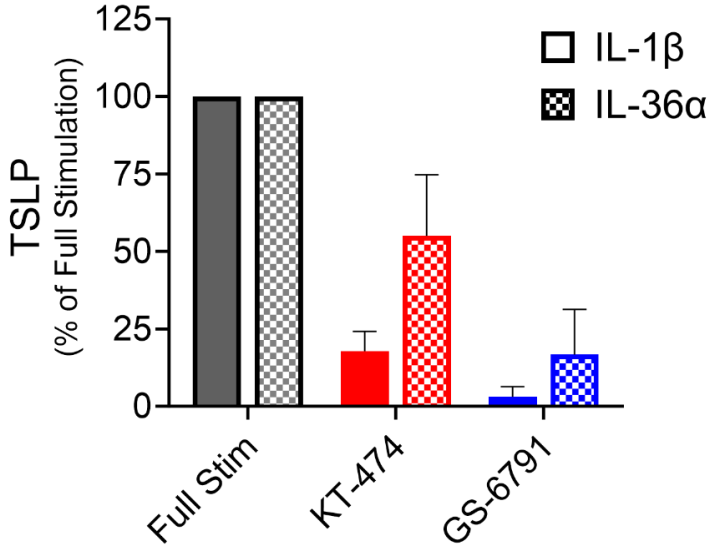
Deeper IRAK4 degradation in basal human keratinocytes at 24 h



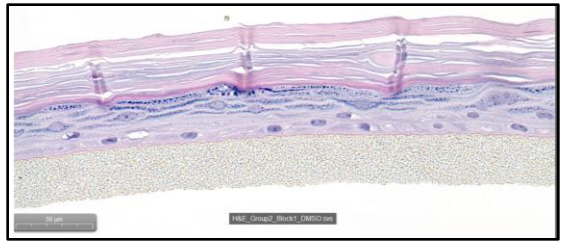


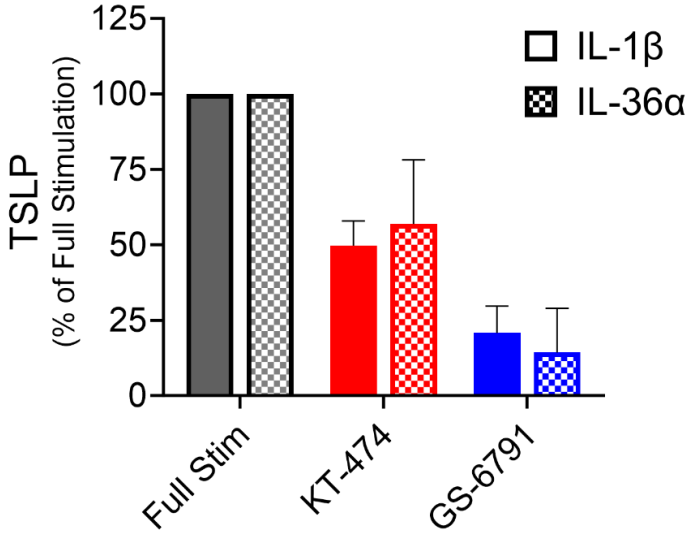
Superior inhibition in differentiated human keratinocytes





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

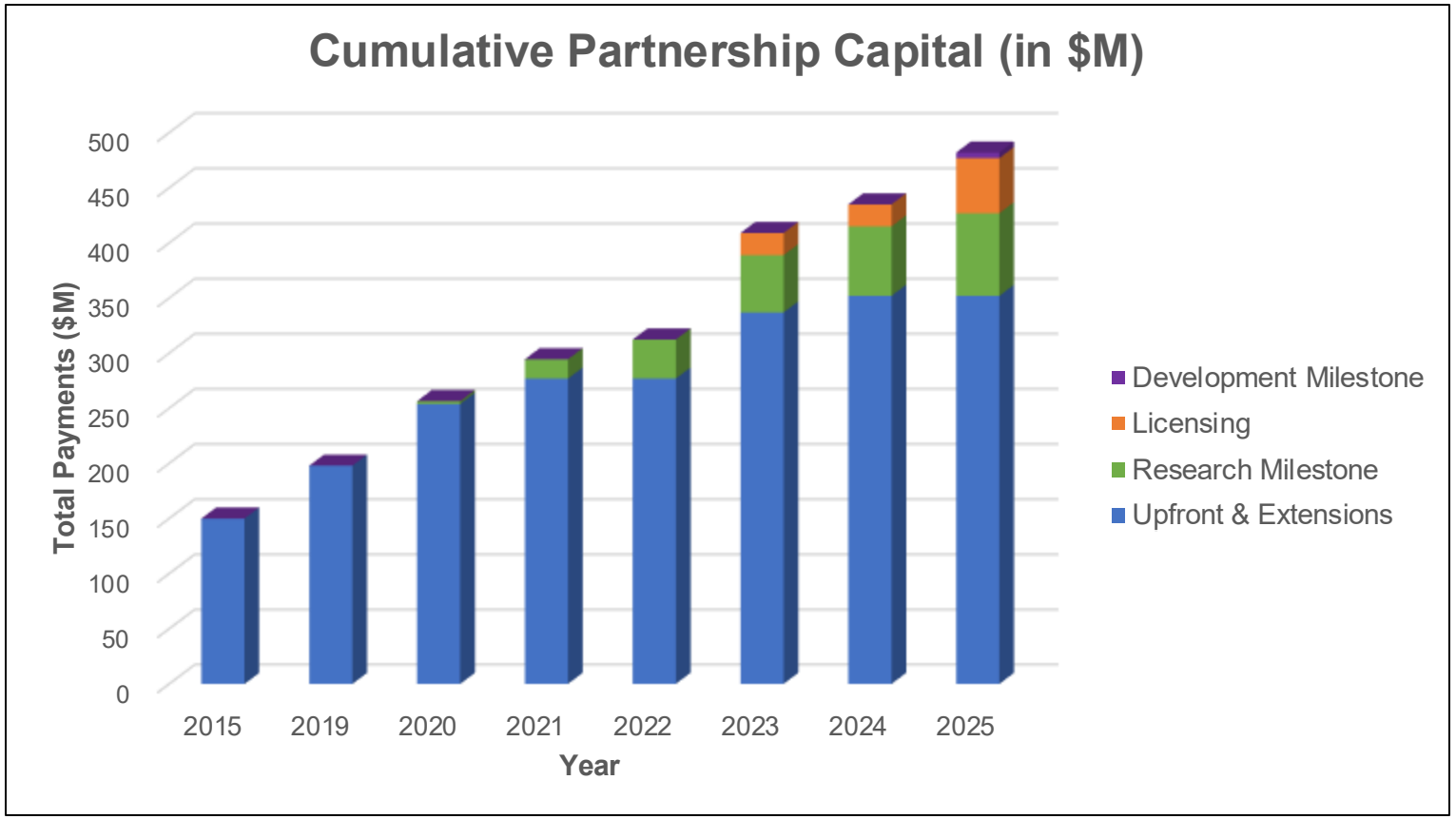
NURIX

Driving Value with Wholly Owned and Partnered Programs

Partnered Degrader Programs Expand Our Pipeline with Opt-Ins and Generate Non-Dilutive Cash Flow

Well capitalized with cash and marketable securities of $540.7 million* providing expected runway into 2028

GILEAD sanofi Pfizer



R&D collaboration cashflow:

Gilead: $135M in total payments to date with the potential for $1.8B in future milestones and fees.

Sanofi: $127M in total payments to date with the potential for $930M in in future milestones and fees.

Seagen (now part of Pfizer): $70M in total payments to date with the potential for $3.4B in future milestones and fees.

Nurix retains option for U.S. profit share and co-promotion for six drugs across three partnerships

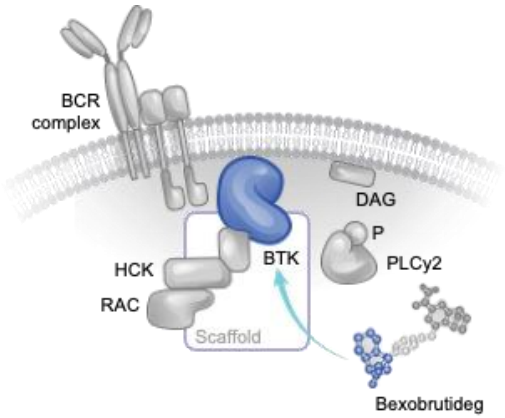
*Cash balance as of February 28, 2026

NURIX

Nurix's Industry-Leading Wholly Owned and Partnered Degrader Portfolio in Inflammation and Autoimmune Diseases

BTK

B-cell & myeloid cell-driven inflammation

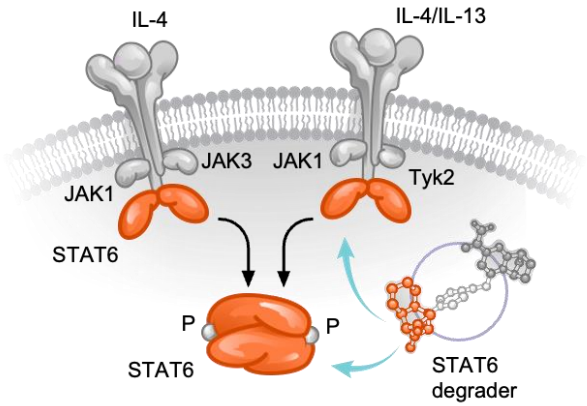


Bexobrutideg
(wholly owned)

New tablet formulation in Phase 1 SAD/MAD study

STAT6

Type 2 inflammation



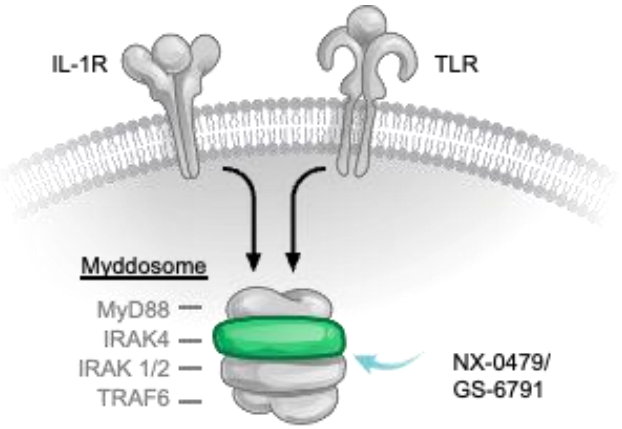
NX-3911
(partnered with Sanofi)

IND-enabling studies ongoing

50/50 U.S. profit share option

IRAK4

IL-1R/TLR-driven inflammation



NX-0479/GS-6791
(partnered with Gilead)

Phase 1 SAD/MAD study ongoing

50/50 U.S. profit share option

NURIX

2026: Building the Future of Protein Degradation

Execute Pivotal Development Pathway in CLL

- Enrollment of Pivotal Phase 2 trial – DAYBreak CLL-201
- Initiation of bexobrutideg confirmatory Phase 3 study in r/r CLL – DAYBreak CLL-306
- Initiation of bexobrutideg combination study in CLL

Advance Degrader Programs in I&I

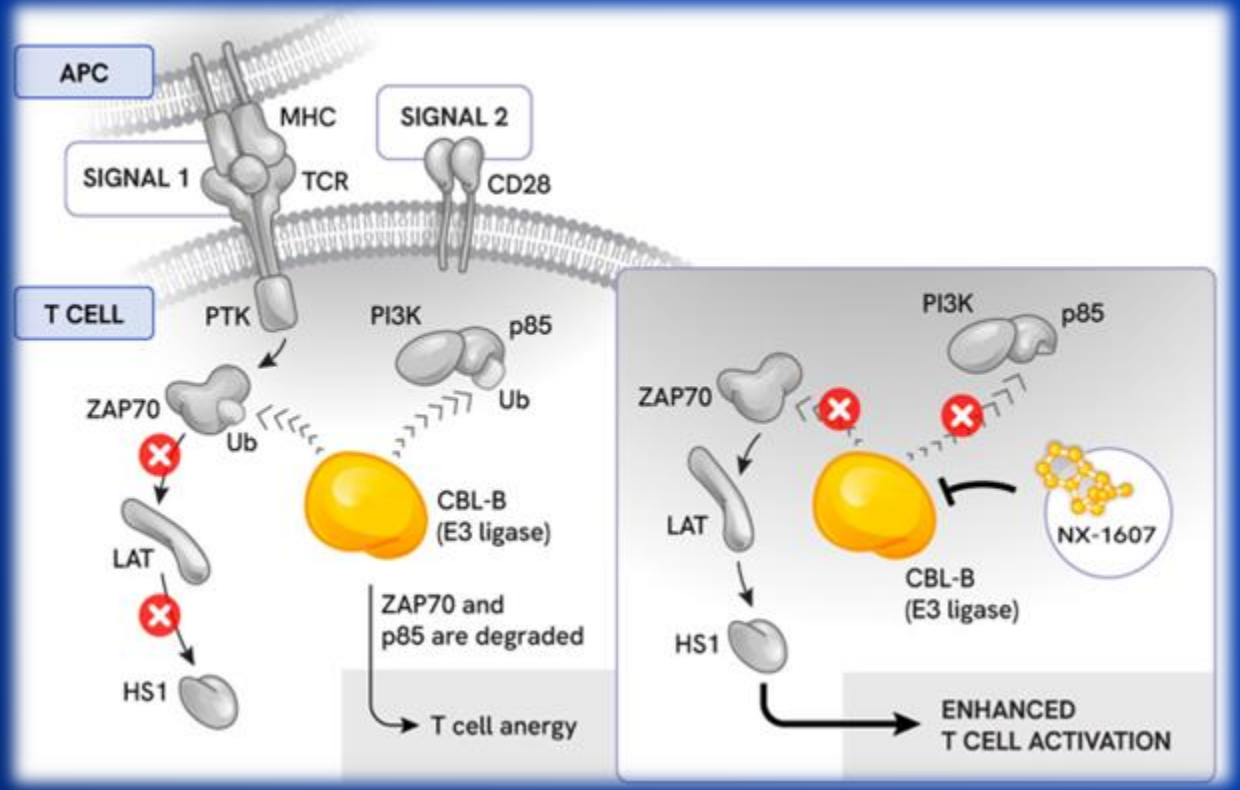
- Potential GS-6791 IRAK4 degrader Phase 1 results from Gilead*
- Potential NX-3911 STAT6 degrader IND filing by Sanofi*
- Bexobrutideg new tablet formulation SAD/MAD study supporting IND in I&I

Clinical Data Updates

- Bexobrutideg Phase 1a/b CLL cohorts
- Bexobrutideg Phase 1a/b NHL cohorts
- Zelebrudomide Phase 1a cohorts

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

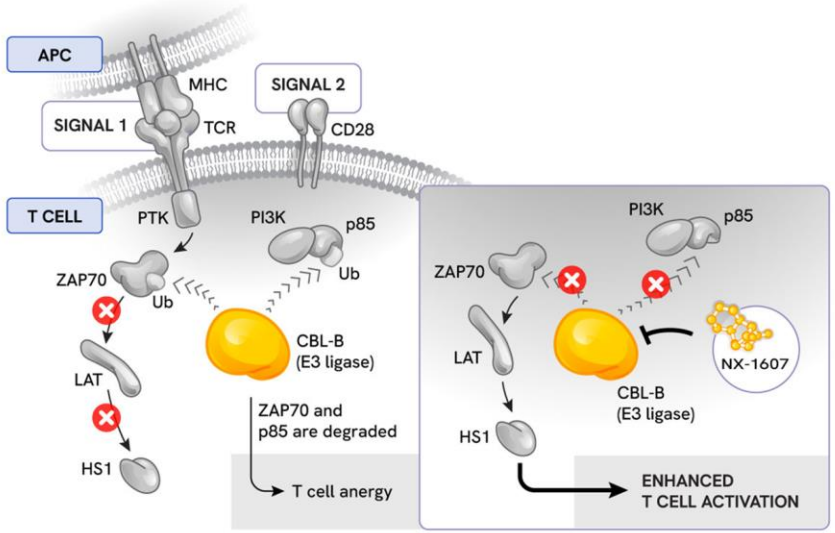
NX-1607: Phase 1a Data in Patients with Advanced Solid Tumors



CBL-B Is a Cytoplasmic E3 Ubiquitin Ligase that Negatively Regulates T Cell Activation, Making It an Attractive Target for Immuno-Oncology

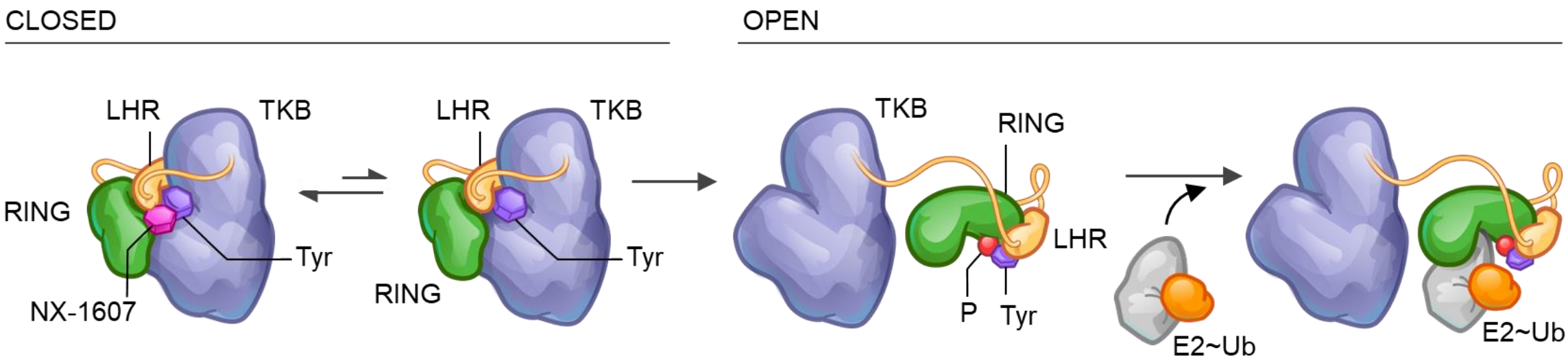
- Inhibition of CBL-B removes an intracellular checkpoint that negatively regulates T cell activation, thereby allowing more robust T cell activation, reversal of T-cell exhaustion, and alleviation of tumor-induced immunosuppression
- NX-1607 is a first-in-class oral inhibitor of CBL-B, offering a novel therapeutic approach to treat solid tumors by targeting a previously unaddressed immune-oncology pathway.
- NX-1607-101 (NCT05107674) is a first-in-human, multicenter, open-label Phase 1a/1b study evaluating safety, pharmacokinetics, pharmacodynamics, and preliminary anti-tumor activity of NX-1607 in patients with relapsed/refractory solid tumors. Results from the NX-1607-101 study monotherapy dose escalation as of 26 July 2025 are reported herein.

NX-1607 Acts as an Intramolecular Glue to Inhibit CBL-B Activity and Enhance T Cell Activation



Source: First-in-Class CBL-B Inhibitor NX-1607: Phase 1a Data in Patients with Advanced Solid Tumors, ESMO 2025

NX-1607 Mechanism of Action: Intramolecular Glue



NX-1607 acts as an intramolecular glue, stabilizing CBL-B in its **inactive** conformation

Immune response

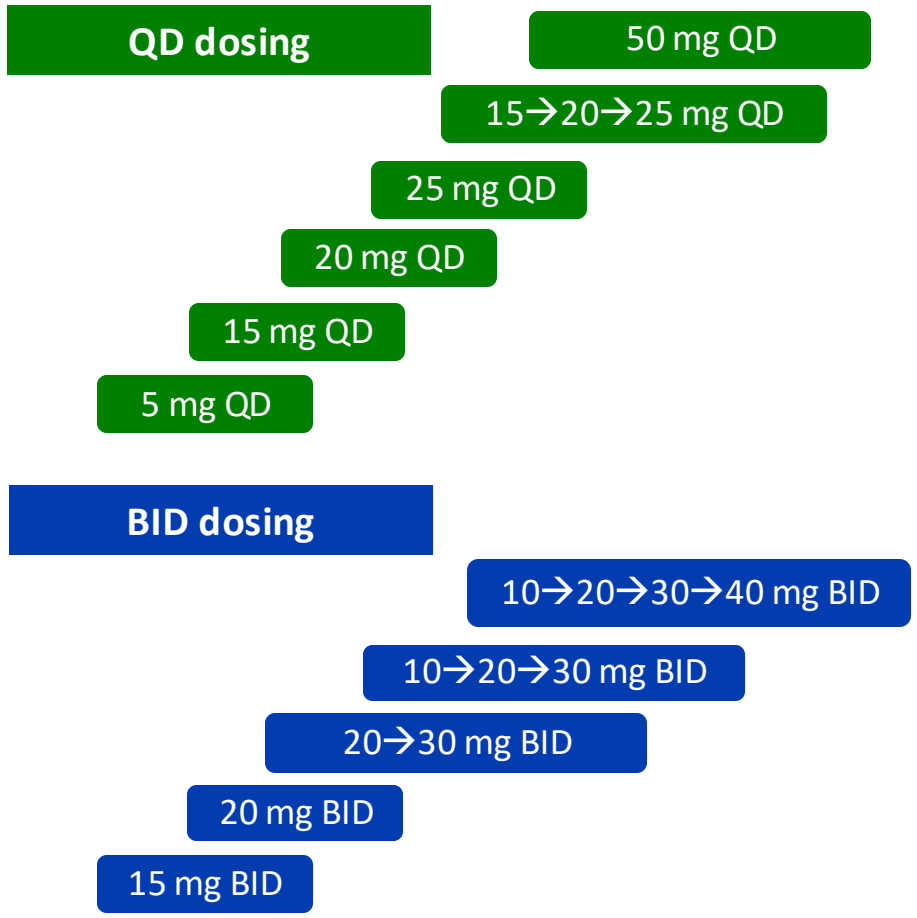
Immune response

NX-1607-101: Phase 1a Study Design (NCT05107674)

Phase 1a/b trial in adults with advanced solid tumor tumors

Key eligibility criteria

- Age ≥18 years
- Metastatic/unresectable disease and exhausted available therapies
- Measurable disease according to applicable response criteria
- ECOG PS 0–1

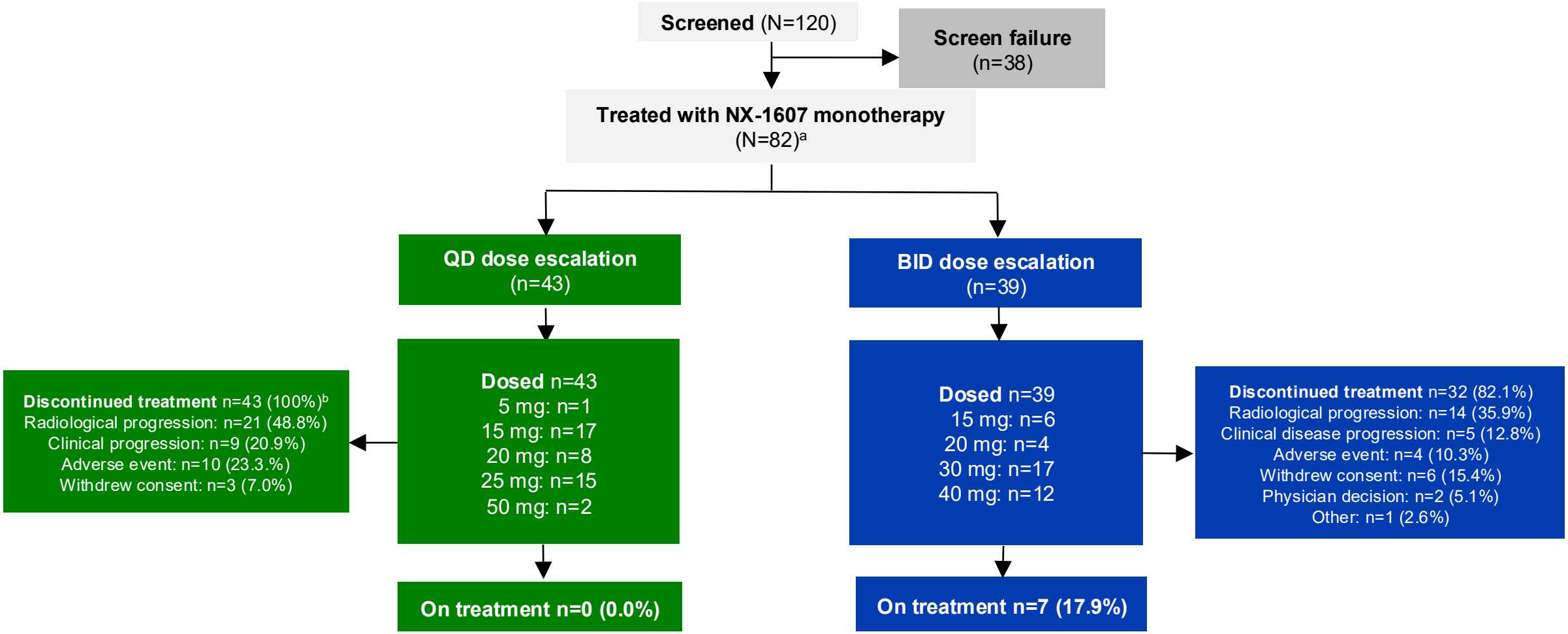


Tumor types enrolled:

- Platinum resistant epithelial ovarian
- Gastroesophageal Junction
- Head and neck squamous cell
- Metastatic Melanoma
- Non-small Cell Lung
- Castration Resistant Prostate
- Malignant Pleural Mesothelioma
- Triple negative breast
- Urothelial
- Cervical
- Microsatellite Stable Colorectal

ECOG PS, Eastern Cooperative Oncology Group (ECOG) performance status

NX-1607-101: Patient Disposition



[a]Excludes patients treated with combinations
[b]Percent of total QD or BID patient population

Data cutoff: 26 Jul 2025

Patient Demographics and Baseline Disease Characteristics

Elderly population with advanced cancer enrolled after multiple lines of prior treatment, including prior I/O therapies

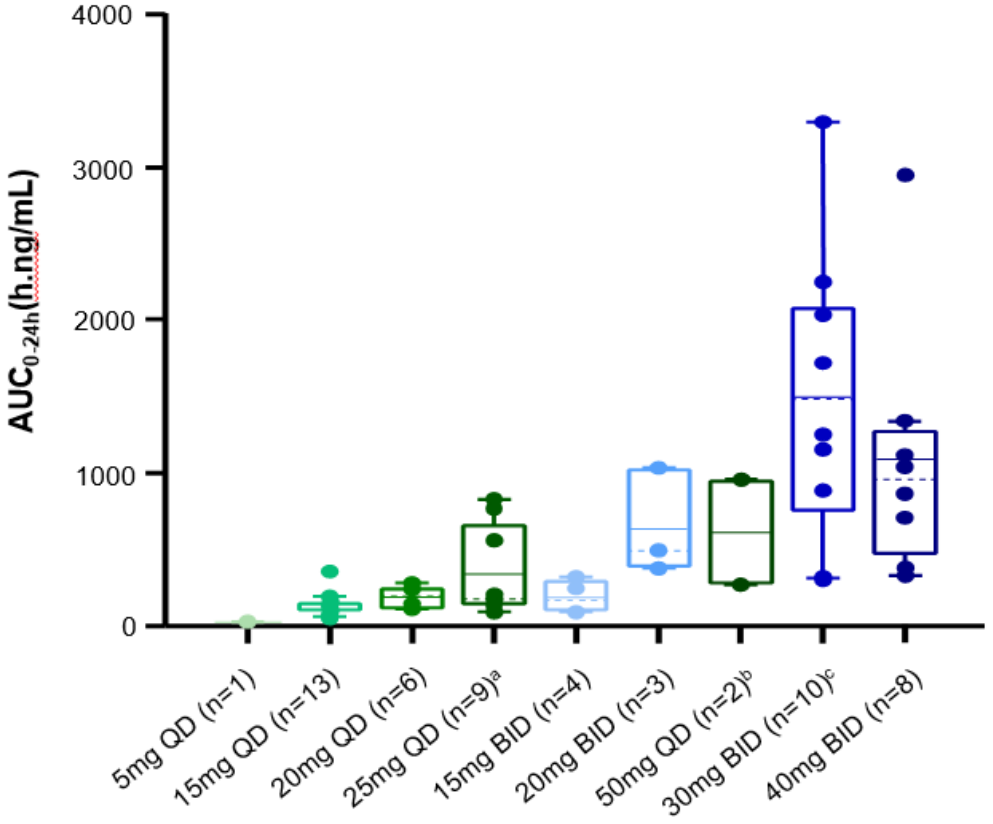
Characteristics	QD dosing (n=43)	BID dosing (n=39)	Overall (N=82)
Median age, years (range)	62 (23-83)	64.0 (35-83)	62 (23-83)
Male, n (%)	26 (60.5)	23 (59.0)	49 (59.8)
Baseline ECOG PS, n (%)			
0	21 (48.8)	17 (43.6)	38 (46.3)
1	22 (51.2)	22 (56.4)	44 (53.7)
Ethnicity , n (%)			
Hispanic or Latino	0	3 (7.7)	3 (3.7)
Not Hispanic or Latino	28 (65.1)	35 (89.7)	63 (76.8)
Not Reported	8 (18.6)	0 (0.0)	8 (9.8)
Unknown	7 (16.3)	1 (2.6)	8 (9.8)
Median prior lines of therapy (range)	3.0 (1–9)	4.0 (2–9)	3.5 (1–9)
Immunotherapies	1.0 (1–3)	1.0 (1–3)	1.0 (1–3)
Anti-Cancer	3.0 (1–9)	4.0 (2–8)	3.0 (1–9)
Anti-Cancer + immunotherapies	1.0 (1–1)	1.5 (1–2)	1.0 (1–2)
Tumor Histology, n (%)			
Platinum Resistant Epithelial Ovarian	1 (2.3)	2 (5.1)	3 (3.7)
Gastroesophageal Junction	1 (2.3)	1 (2.6)	2 (2.4)
Head and Neck Squamous Cell	1 (2.3)	0 (0.0)	1 (1.2)
Metastatic Melanoma	7 (16.3)	1 (2.6)	8 (9.8)
Non-small Cell Lung	2 (4.7)	2 (5.1)	4 (4.9)
Castration Resistant Prostate	9 (20.9)	14 (35.9)	23 (28.0)
Malignant Pleural Mesothelioma	1 (2.3)	3 (7.7)	4 (4.9)
Triple Negative Breast	0 (0.0)	1 (2.6)	1 (1.2)
Urothelial	1 (2.3)	0 (0.0)	1 (1.2)
Cervical	3 (7.0)	4 (10.3)	7 (8.5)
Microsatellite Stable Colorectal	17 (39.5)	11 (28.2)	28 (34.1)

Data cutoff: 26 Jul 2025

Source: First-in-Class CBL-B Inhibitor NX-1607: Phase 1a Data in Patients with Advanced Solid Tumors, ESMO 2025

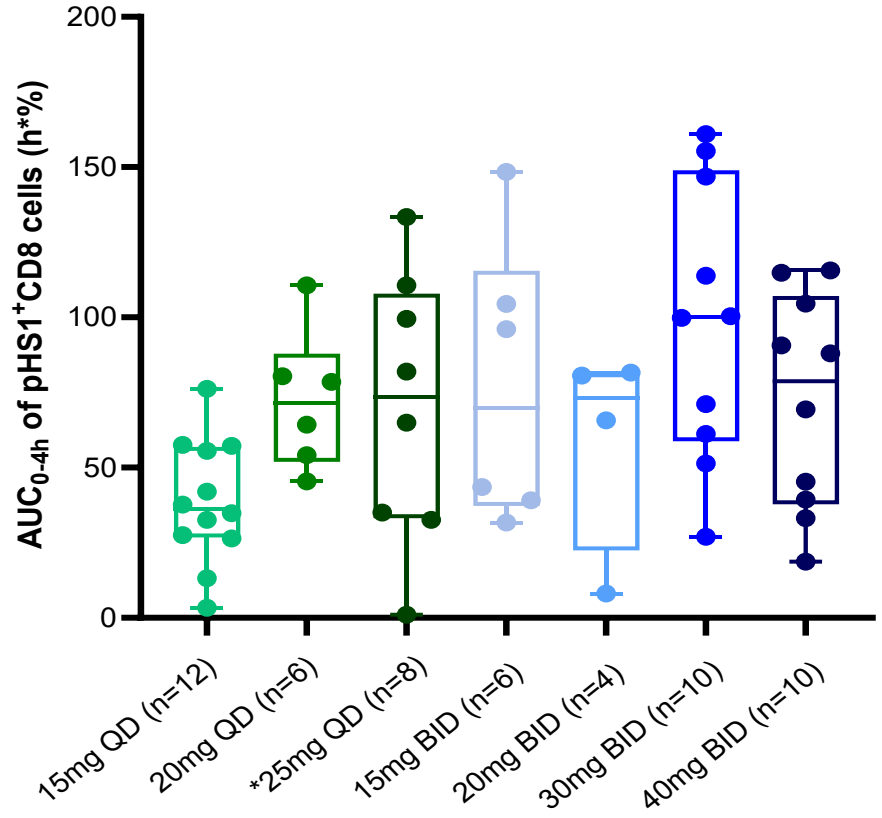
Dose-Dependent Pharmacokinetics with Associated Increases in Pharmacodynamics of the CBL-B Proximal Biomarker pHS1

Steady state pharmacokinetics



[a]Patients enrolled in 25mg QD and 15→20→25mg QD ramp up cohorts
[b]50mg QD – C1D1 $AUC_{0\text{-}24h}$ shown in plot
[c]Patients enrolled in 20→30mg BID and 10→20→30mg BID ramp up cohorts
Solid and dashed lines denote mean and median, respectively. The ends of the box indicate the first and third quartiles. The whiskers show the data values still within 1.5 of the inter-quartile range (IQR), and data values that do not fall between the whiskers are plotted as outliers

Pharmacodynamic activity of pHS1 in CD8 T cells

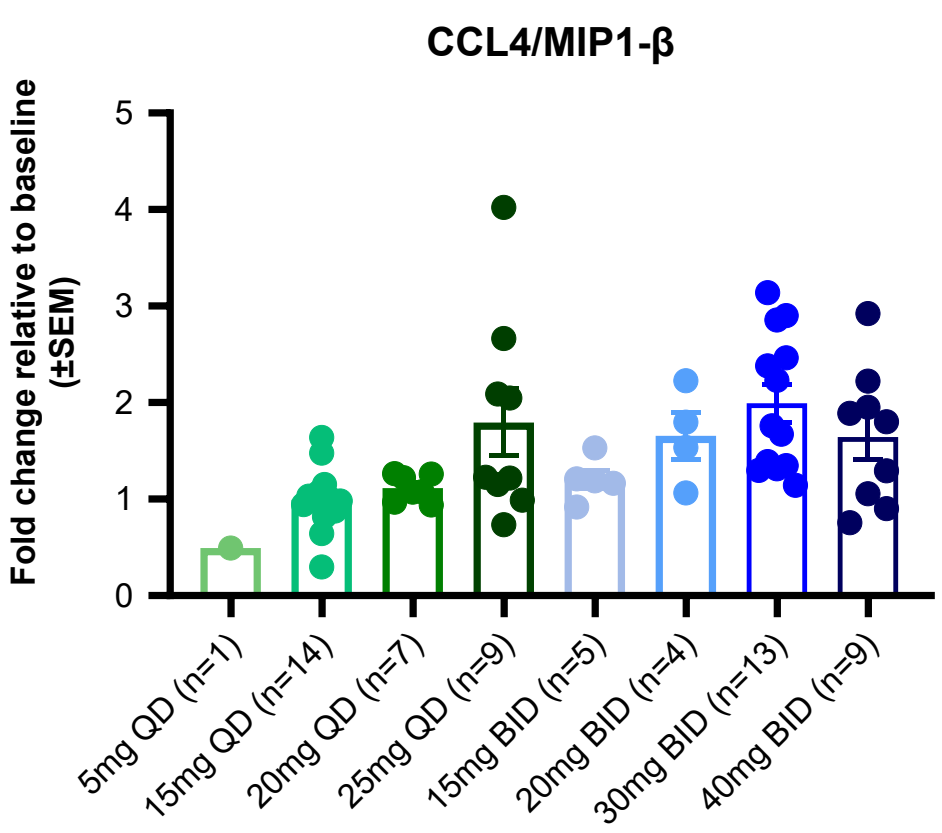


All timepoints were normalized to C1D1 pre-dose. QD AUCs were calculated from $AUC_{0\text{-}4h}$ collected at C1D1 with exception of 2 patients receiving ramp up dosing (*), for which $AUC_{0\text{-}4h}$ represents C2D1. All BID AUCs represent C2D1. The relevance of pHS1 pharmacodynamics for measuring NX-1607 activity has been previously described[3].

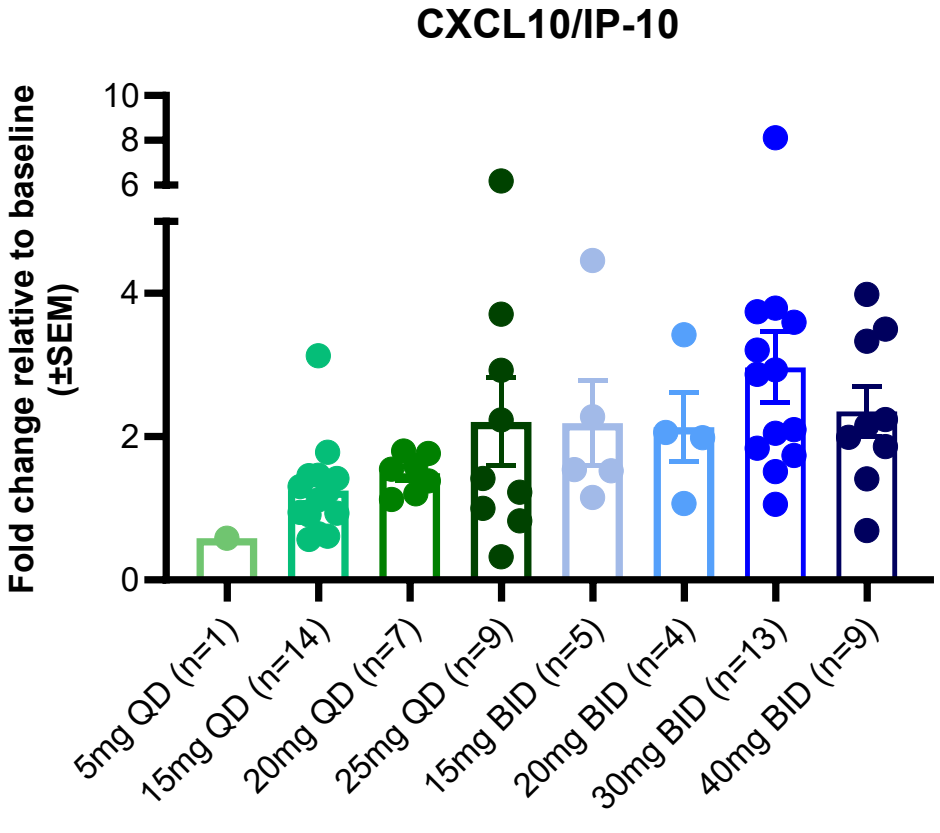
Source: First-in-Class CBL-B Inhibitor NX-1607: Phase 1a Data in Patients with Advanced Solid Tumors, ESMO 2025

NX-1607 Demonstrates Dose-Responsive Peripheral Immune Activation via Increases in Distal Biomarkers: Chemokines CXCL10 and CCL4

NX-1607 led to an increase in the peripheral chemokines, CXCL10 and CCL4, at Cycle 2 Day 15, suggesting the upregulation of pro-inflammatory signaling and corresponding immune cell recruitment



Chemoattractant for dendritic cell recruitment



IFN-γ inducible chemokine recruiting effector T cells & NK cells

Source: First-in-Class CBL-B Inhibitor NX-1607: Phase 1a Data in Patients with Advanced Solid Tumors, ESMO 2025

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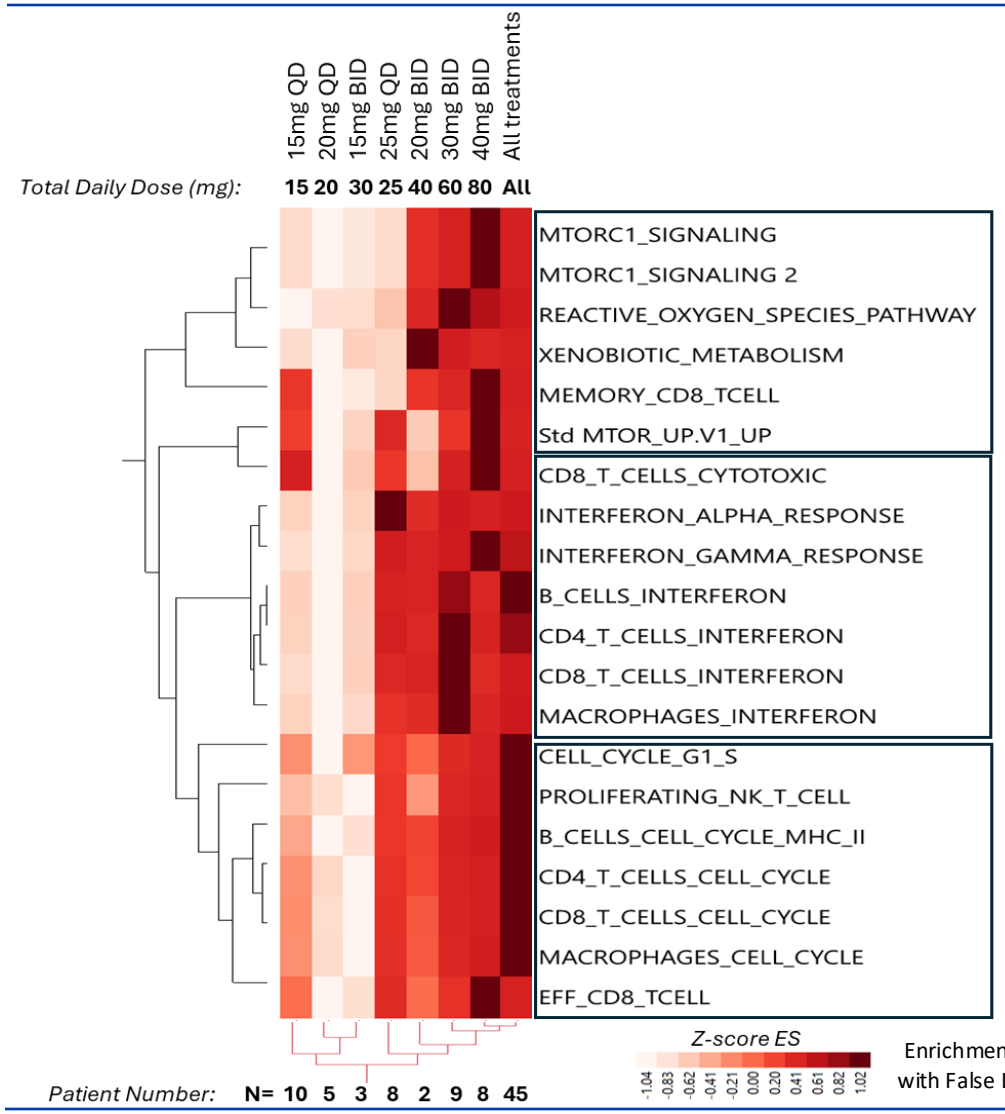
Gene Expression in PBMCs Demonstrates Dose-Responsive Modulation of Immune Pathway Activation by NX-1607



Immune Cell Metabolic Support

IFN-α/IFN-γ Response & Induced Immune Cell Activities

Immune Cell Proliferation

- Transcriptomic profiling demonstrated dose-responsive enrichment of key immune signaling pathways
- Pathways included enhanced immune cell metabolic support, progressive induction of IFN-α/IFN-γ response and downstream immune cell activities, and upregulation of inflamed immune cell proliferation programs
- These data support a mechanistic relationship between dose and coordinated anti-tumor immune pathway engagement

Enrichment scores (ES) of the 20 significant pathways selected for each dose cohort were generated by Gene Set Enrichment Analysis (GSEA) with False Discovery Rate (FDR, adjusted p-value) < 0.1 cutoff. Normalized ES by z-score was used for the unsupervised 2D hierarchical cluster

 Source: First-in-Class CBL-B Inhibitor NX-1607: Phase 1a Data in Patients with Advanced Solid Tumors, ESMO , SITC 2025

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NX-1607 Has a Safety Profile Comparable to Approved Immuno-oncology Agents in Early Development

Most adverse events were ≤Grade 2 in severity; Active doses of ≥30mg are tolerable.

Adverse event	Doses ≥30 mg BID[a] (n=24)	Overall (N=82)
Dose-limiting toxicities[b,c]	0 (0%)	9 (11.0%)
TRAEs all grades TRAEs ≥Grade 3	23 (95.8%) 4 (16.7%)	70 (85.4%) 20 (24.4%)
Treatment-related SAEs	0 (0%)	7 (8.5%)
Discontinuations due to TRAEs	1 (4.2%)	12 (14.6%)
Immune-related AEs[d]	2 (8.3%)	6 (7.3%)
Nausea TRAEs all grades	13 (54.2%)	39 (47.6%)
Vomiting TRAEs all grades	6 (25.0%)	26 (31.7%)
Nausea TRAEs ≥Grade 3	0 (0%)	2 (2.4%)
Vomiting TRAEs ≥Grade 3	0 (0%)	2 (2.4%)

[a]≥30mg BID includes 10–20–30mg BID and 10–20–30–40mg BID dose regimens, 20-30mg BID are not included
[b]DLTs observed at lower dose levels were managed by changing the dose regimens and adding anti-emetics, leading to improved tolerability of higher doses
[c]DLTs were as follows: acute kidney injury/increased creatine (n=2), hypotension (n=2), decreased albumin (n=1), syncope (n=1), vomiting (n=1), headache (n=1), dehydration (n=1)
[d]Immune-related adverse events were as follows: acute kidney injury/increased creatine (n=2); hypothyroidism (n=1); increased alkaline phosphatase/ALT (n=1); fatigue (n=1); arthralgia (n=1); rash (n=2)

Source: First-in-Class CBL-B Inhibitor NX-1607: Phase 1a Data in Patients with Advanced Solid Tumors, ESMO 2025

NX-1607 Demonstrates a Disease Control Rate of 49.3% Across Doses and Tumor Types

All response-evaluable patients	(n=71)
Disease control rate (DCR), n (%)	35 (49.3)
Objective response rate (ORR), n (%)	1 (1.4)
Best response, n (%)	
Complete response (CR)	0 (0.0)
Partial response (PR)	1 (1.4)
Stable disease (SD)	34 (47.9)
Progressive disease (PD)	36 (50.7)

Data cutoff: 26 Jul 2025

NX-1607 Treatment Demonstrates Preliminary Signals of Clinical Benefit

Patients with Advanced Metastatic Cancer Who Have ≥ Stable Disease After Multiple Prior Lines of Therapy

Tumor type	Dose/schedule	Duration of treatment (months)[a]	Response (tumor volume change)	Biomarker reduction
MSS CRC	15mg QD	1.7	SD (–3.7%)	NA
MSS CRC	25mg QD	27.1	**PR (–37.5%)**	NA
MSS CRC	15mg BID	3.3	SD (–23.6%)	NA
MSS CRC[b]	10–20–30–40mg BID	5.3	SD (–23.9%)	CEA reduction of 26%
CRPC	5mg QD	8.5	SD (–10.5%)	PSA reduction of 30%
CRPC	15–20–25mg QD	3.1	SD (–11.1%)	NA
CRPC	20–30mg BID	6.6	SD[c]	PSA reduction of 90%; CTC from 12→0
CRPC	10–20–30mg BID	2.0	PD[c]	PSA reduction of 71%
CRPC	10–20–30mg BID	4.7	SD (–14.3%)	PSA reduction of 65%
CRPC	10–20–30mg BID	0.7	PD (+40.9%)	PSA reduction of 73%
Melanoma	15mg QD	0.9	SD (–10.9%)	NA
Melanoma	20mg QD	4.2	SD (–27.1%)	NA
NSCLC	15mg QD	9.8	SD (–4.8%)	NA
NSCLC	15mg BID	24.3	SD (–13.1%)	NA
Cervical[b]	10–20–30mg BID	8.2	SD (–5.7%)	NA
Gastroesophageal	10–20–30–40mg BID	4.8	SD (–18.5%)	NA

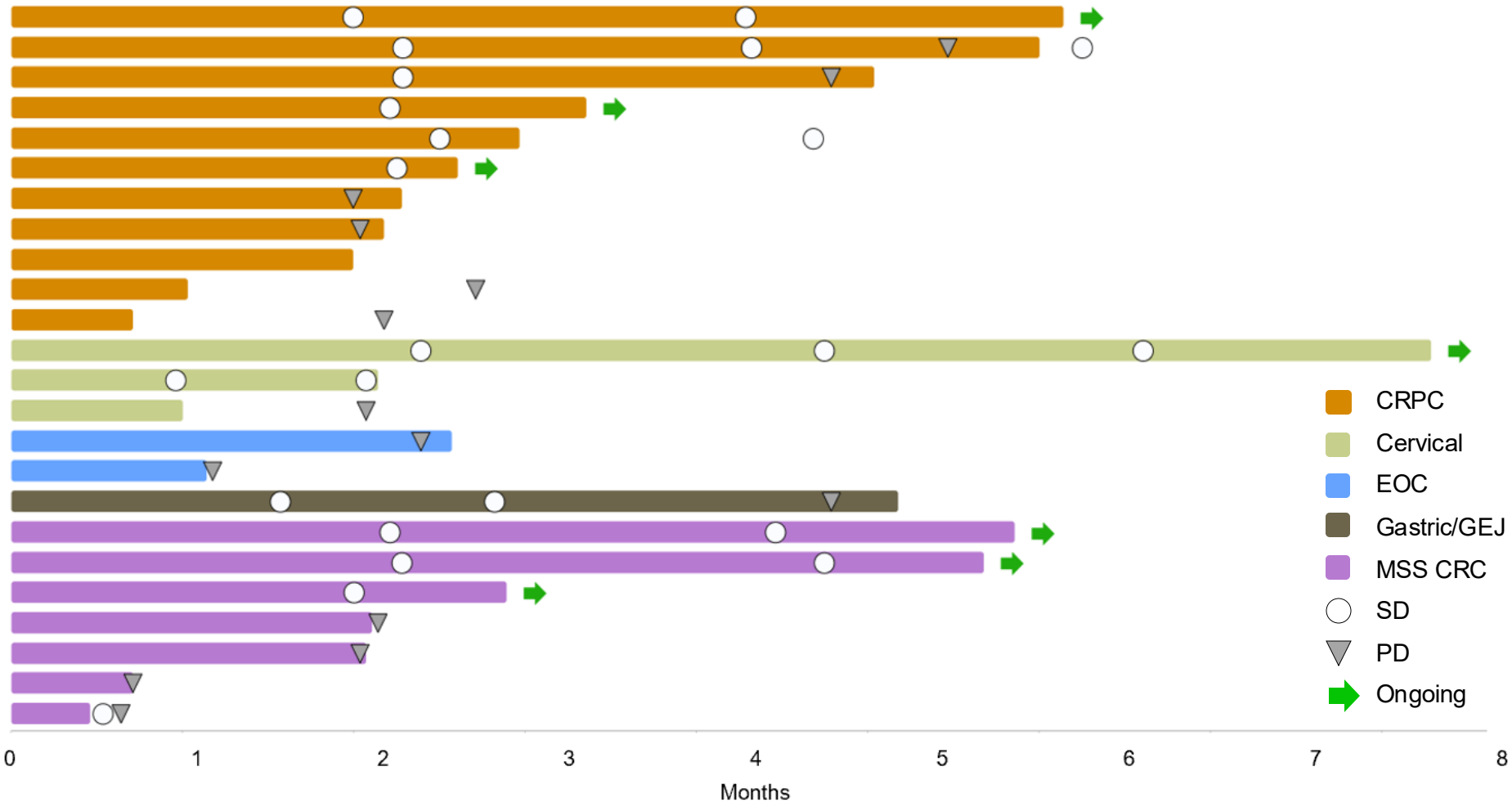
- NX-1607 provided a high disease control rate (CR+PR+SD) at 49.3% overall and demonstrated meaningful clinical activity (tumor volume/biomarker reductions) across a broad range of indications
- 7 patients achieved disease control (SD or PR) for ≥5 months on treatment; 1 patient reached 27 months on treatment with a best overall response of PR (CRC, **bolded**)

[a]Days (months) of clinical benefit calculated from C1D1 until clinical/radiological progression, adverse event or withdrew consent
[b]Patients are ongoing as of the data cut
[c]Bone-only disease and thus no corresponding tumor volume change information
Source: First-in-Class CBL-B Inhibitor NX-1607: Phase 1a Data in Patients with Advanced Solid Tumors, ESMO 2025

Data cutoff: 26 Jul 2025

Duration of Treatment and Response by Tumor Type in Patients Receiving Doses ≥30mg BID

Dose level	Discontinuation
10→20→30→40mg BID	
10→20→30mg BID	Physician decision
10→20→30mg BID	Radiological PD
10→20→30→40mg BID	
10→20→30mg BID	Withdrew consent
10→20→30→40mg BID	
10→20→30→40mg BID	Radiological PD
10→20→30mg BID	Radiological PD
10→20→30→40mg BID	Physician decision
10→20→30→40mg BID	Adverse event
10→20→30mg BID	Withdrew consent
10→20→30mg BID	
10→20→30mg BID	Withdrew consent
10→20→30mg BID	Withdrew consent
10→20→30→40mg BID	Radiological PD
10→20→30mg BID	Radiological PD
10→20→30→40mg BID	Radiological PD
10→20→30mg BID	
10→20→30→40mg BID	
10→20→30→40mg BID	
10→20→30→40mg BID	Radiological PD
10→20→30mg BID	Clinical PD
10→20→30mg BID	Clinical PD
10→20→30→40mg BID	Clinical PD



Data cutoff: 26 Jul 2025

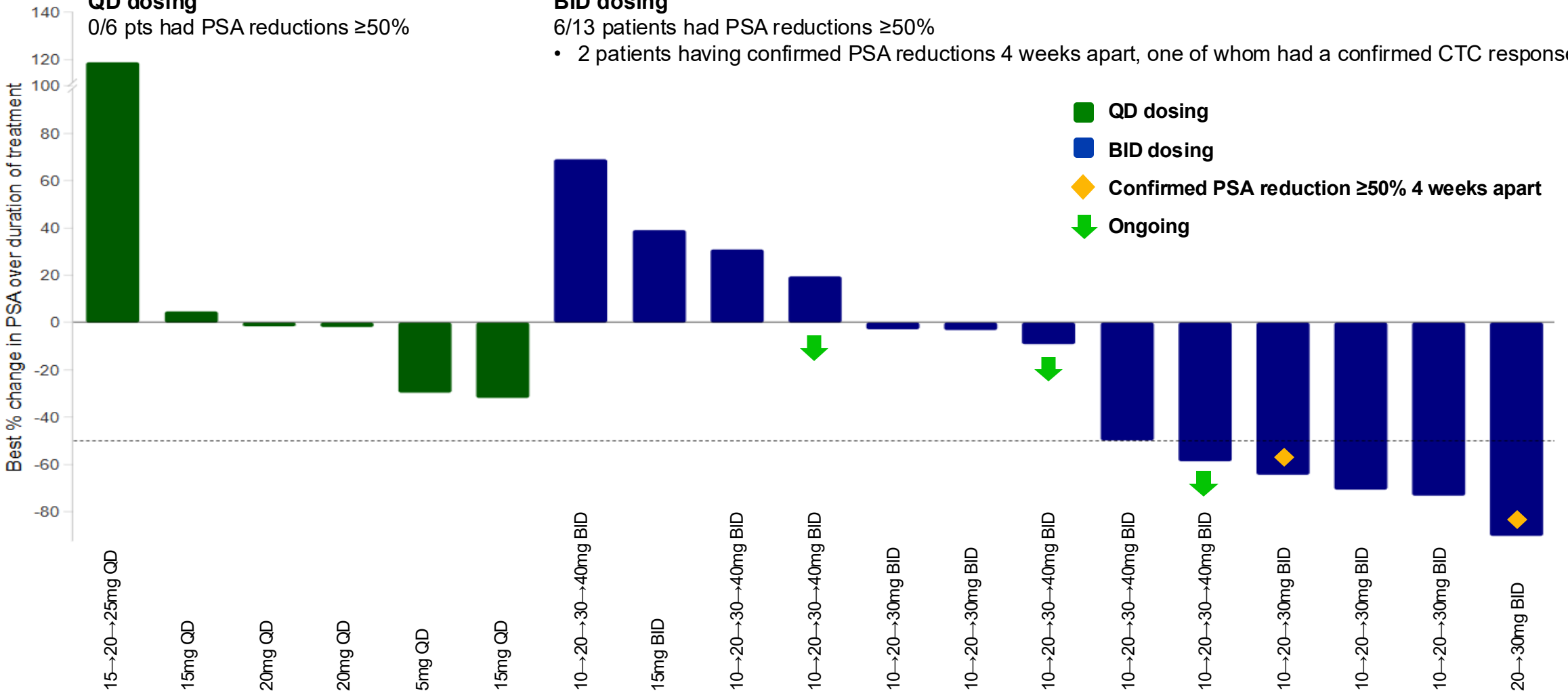
AE, adverse event; BID, twice daily; Disc, discontinued, DLT, dose-limiting toxicity; QD, once daily; PD, disease progression; SD, stable disease

Clinical Activity in Patients with CRPC: ≥50% Reduction of PSA

BID dosing shows promising and meaningful reductions in PSA in patients with CRPC

QD dosing
0/6 pts had PSA reductions ≥50%

BID dosing
6/13 patients had PSA reductions ≥50%
- 2 patients having confirmed PSA reductions 4 weeks apart, one of whom had a confirmed CTC response



QD daily dosing**; BID**, twice daily; CTC, circulating tumor cells ; **PSA**, prostate specific antigen

Source: First-in-Class CBL-B Inhibitor NX-1607: Phase 1a Data in Patients with Advanced Solid Tumors, ESMO 2025

***CRPC**, Castration-Resistant Prostate Cancer; **PSA**, prostate specific antigen; **BID**, twice daily; **QD**, once daily*

Data cutoff: 26 Jul 2025

Case Study 1: Patient with MSS Colorectal Cancer

Patient profile

- 59-year-old male with CRC
- 7 prior lines of therapy: FOLFOX, 5-FU+panitumumab +irinotecan, capecitabine, tipiracil/trifluridine (x2), 5-FU+irinotecan, nivolumab
- 8th line of therapy NX-1607, dosed at 25mg QD

Pharmacodynamic profile

- Proximal biomarker: the maximum %pHS1+CD8+ increased to ~80% from baseline
- Distal biomarkers: the maximum CXCL10 and CCL4 increased 2- and 6-fold over baseline

Anti-tumor profile

- PR at Week 27, confirmed at week 39
- Target lesion 28 x 24mm decreased to 28 x 15mm (reduction of 37.5%)
- Patient developed PD at Week 87 but continued post progression to derive clinical benefit until Week 123

***MSS**, micro-satellite stable*

Source: First-in-Class CBL-B Inhibitor NX-1607: Phase 1a Data in Patients with Advanced Solid Tumors, ESMO 2025

Case Study 2: Patient with Castration-Resistant Prostate Cancer (CRPC)

Patient profile

- 72-year-old male with CRPC
- 2 prior lines of therapy: docetaxel and cabazitaxel
- 3rd line of therapy NX-1607, dosed with a ramp-up regimen at 20–30mg BID
- Bone-only disease

Pharmacodynamic profile

- Proximal biomarker: the maximum %pHS1+CD8+ increased to 62.6% from baseline
- Distal biomarker: the max CCL4 increased 3-fold over baseline. No data available for CXCL10

Anti-tumor profile

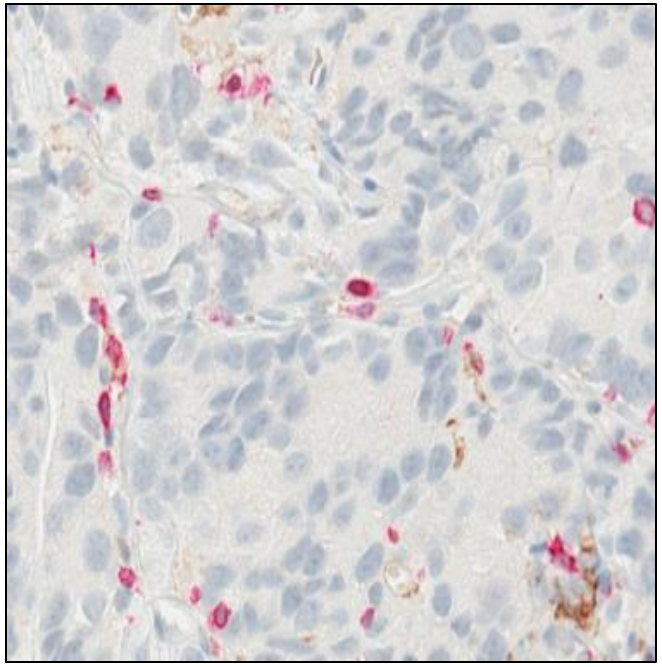
- Baseline PSA 592µg/L reduced by 56.3% at week 9 to 259µg/L, by 83.9% at week 27 to 95.1µg/L, and by 90.3% at week 51 to 57.4µg/L.
- Baseline CTC 12/7.5mL reduced to 0 at Week 9 and confirmed at week 27
- No new bone lesions

Source: First-in-Class CBL-B Inhibitor NX-1607: Phase 1a Data in Patients with Advanced Solid Tumors, ESMO 2025

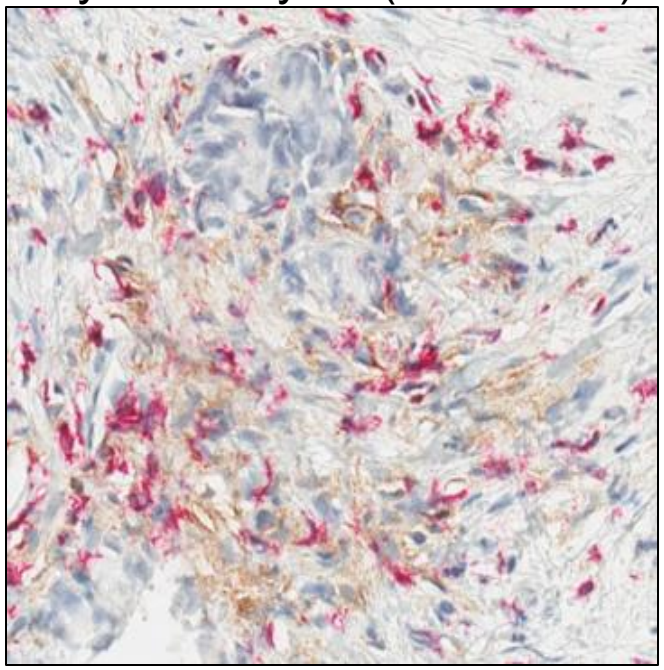
Case Study: NX-1607 Drives Robust Immune Activation and Durable Disease Control in a Highly Refractory mCRPC Patient

IHC of paired metastatic lymph node biopsies show increased CD8$^+$ (brown) and PD-L1$^+$ (red) Tumor Infiltrating Lymphocytes (TIL) following NX-1607 treatment

Cycle 1 Day 1 (Predose)



Cycle 2 Day 15 (Post Dose)

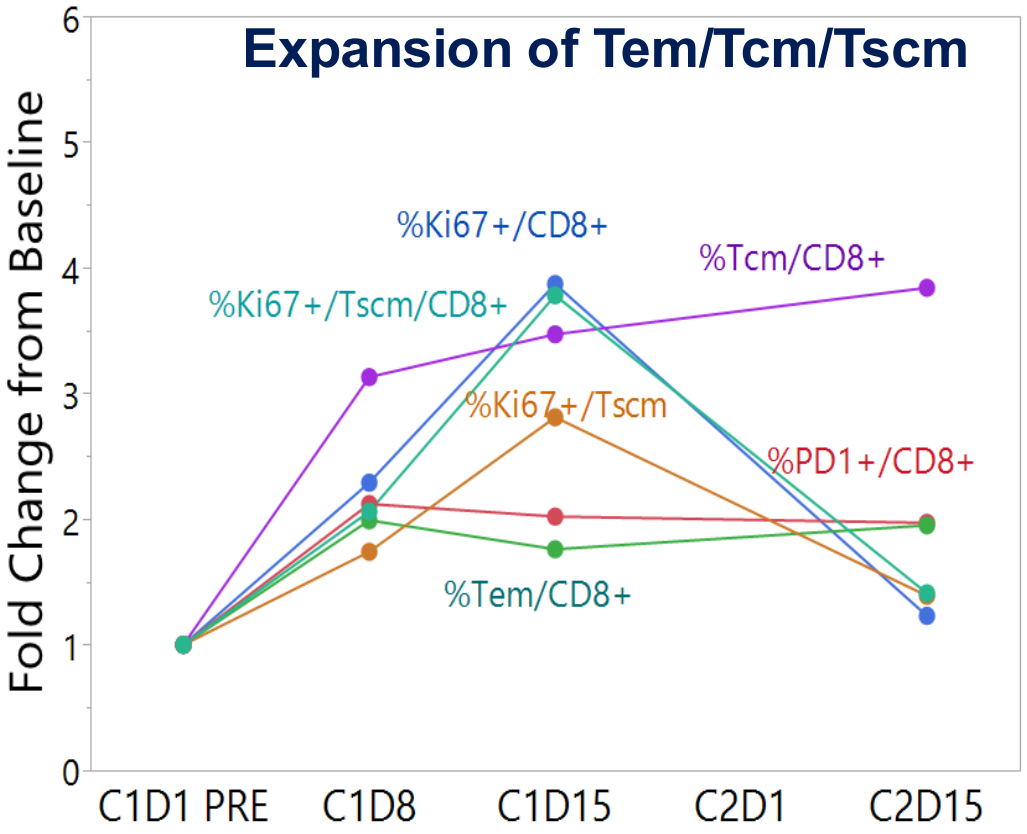


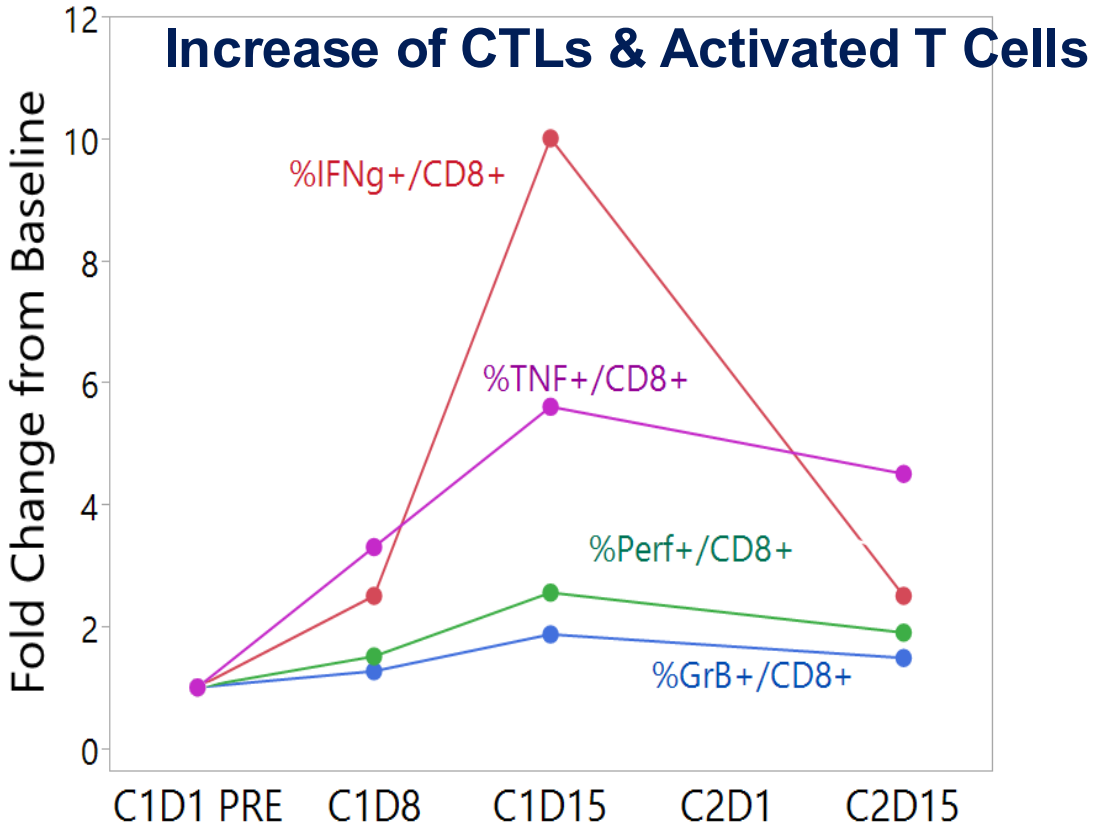
- Patient Overview: 5 prior lines; initiated NX-1607 (30 mg BID) as 6th-line therapy
- Best Response: Durable Stable Disease (27 weeks)
- Key Immunologic Findings:
  - IHC demonstrates a strong increase in intratumoral CD8$^+$ & PD-L1$^+$ TIL, confirming on mechanism immune engagement
  - RNA-seq and peripheral immune data (*not shown*) show increased cytotoxic, NK, APC, and DC signatures, reduced Treg suppression, & systemic expansion of cytolytic effector memory CD8$^+$ T cells

Source: First-in-Class CBL-B Inhibitor NX-1607: Phase 1a Data in Patients with Advanced Solid Tumors, ESMO , SITC 2025

Case Study: NX-1607 Enhances Expansion & Activation of T cell Subsets in the Periphery of a Highly Refractory mCRPC Patient

Peripheral flow cytometry reveals robust expansion of proliferating, cytotoxic, and memory CD8⁺ T cell subsets, including PD-1⁺ antigen-experienced cells indicating enhanced antitumor immunity

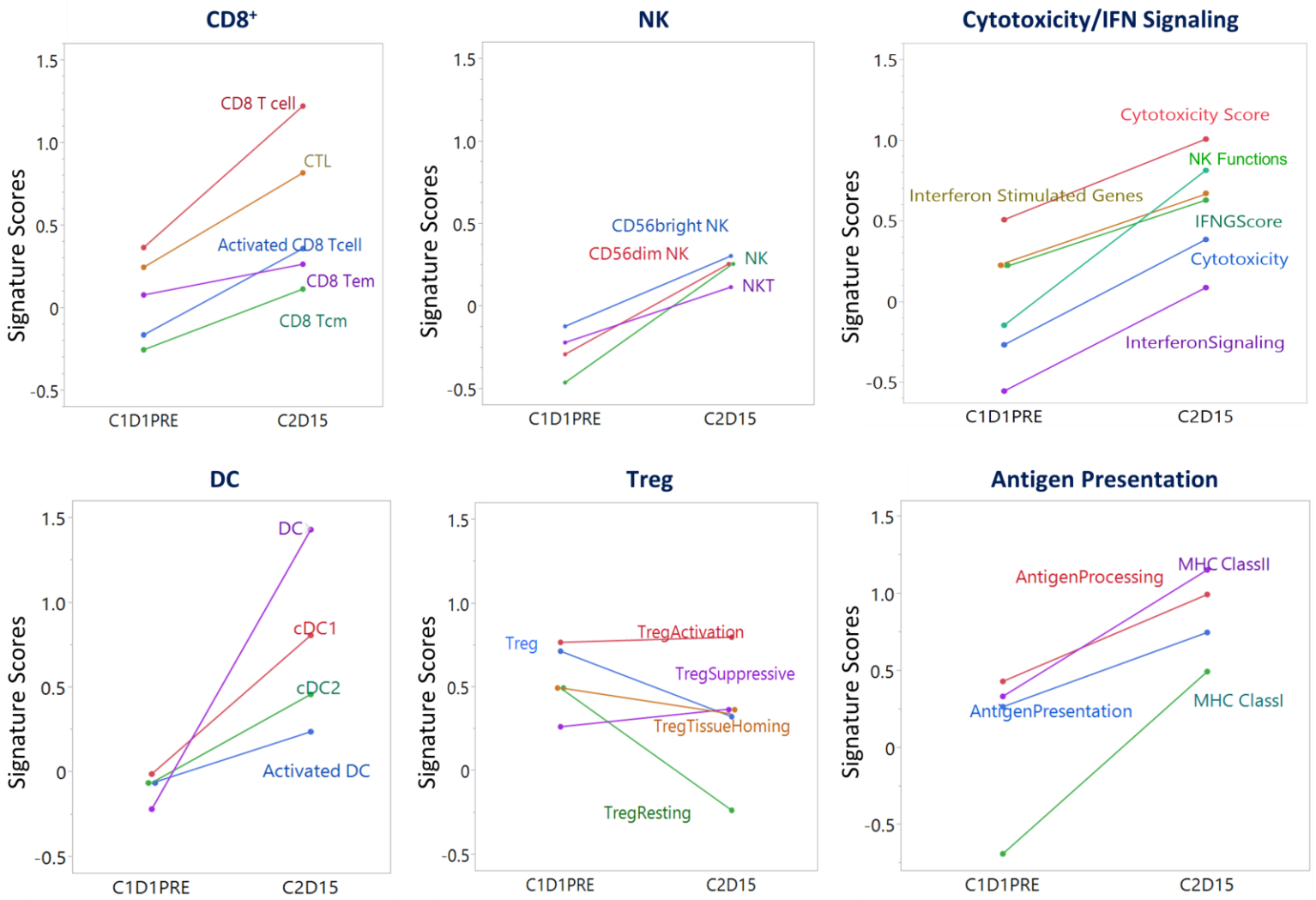




Abbreviations: C = Cycle; D = Day; PRE = Predose; Tem = Effector Memory T cells; Tcm = Central Memory T cells; Tscm = Stem-like Memory T cells; PD-1 = Programmed cell-death receptor-1; Perf = Perforin; GrB = Granzyme B, IFNg = Interferon gamma; TNF = Tumor necrosis factor

Source: First-in-Class CBL-B Inhibitor NX-1607: Phase 1a Data in Patients with Advanced Solid Tumors, ESMO , SITC 2025

Case Study: NX-1607 Promotes Gene Signatures of Immune Response Within a Metastatic Lymph Node of a Highly Refractory mCRPC Patient



RNA-seq reveals broad immune activation in the TME with NX-1607

- Increased cytotoxic CD8+ and NK cell signatures, interferon-stimulated pathways, and antigen-presentation signatures (MHC-I/II, DC-related)

- Decreased Treg-associated suppressive signatures

These data support NX-1607 mediated remodeling of the TME toward enhanced antitumor immunity

Abbreviations: C = Cycle; D = Day; PRE = Predose;Tem = Effector Memory T cells; Tcm = Central Memory T cells; Tscm = Stem-like Memory T cells; PD-1 = Programmed cell-death receptor-1; CTL = Cytotoxic T Lymphocyte; NK = Natural Killer; NKT = Natural Killer T cell; DC = Dendritic Cell; cDC1/cDC2 = Conventional Dendritic Cell subsets; MHC-I/II = Major Histocompatibility Complex class I/II; IFNγ = Interferon gamma; TNF = Tumor necrosis factor; Perf = Perforin; GrB = Granzyme B.

Source: First-in-Class CBL-B Inhibitor NX-1607: Phase 1a Data in Patients with Advanced Solid Tumors, ESMO , SITC 2025

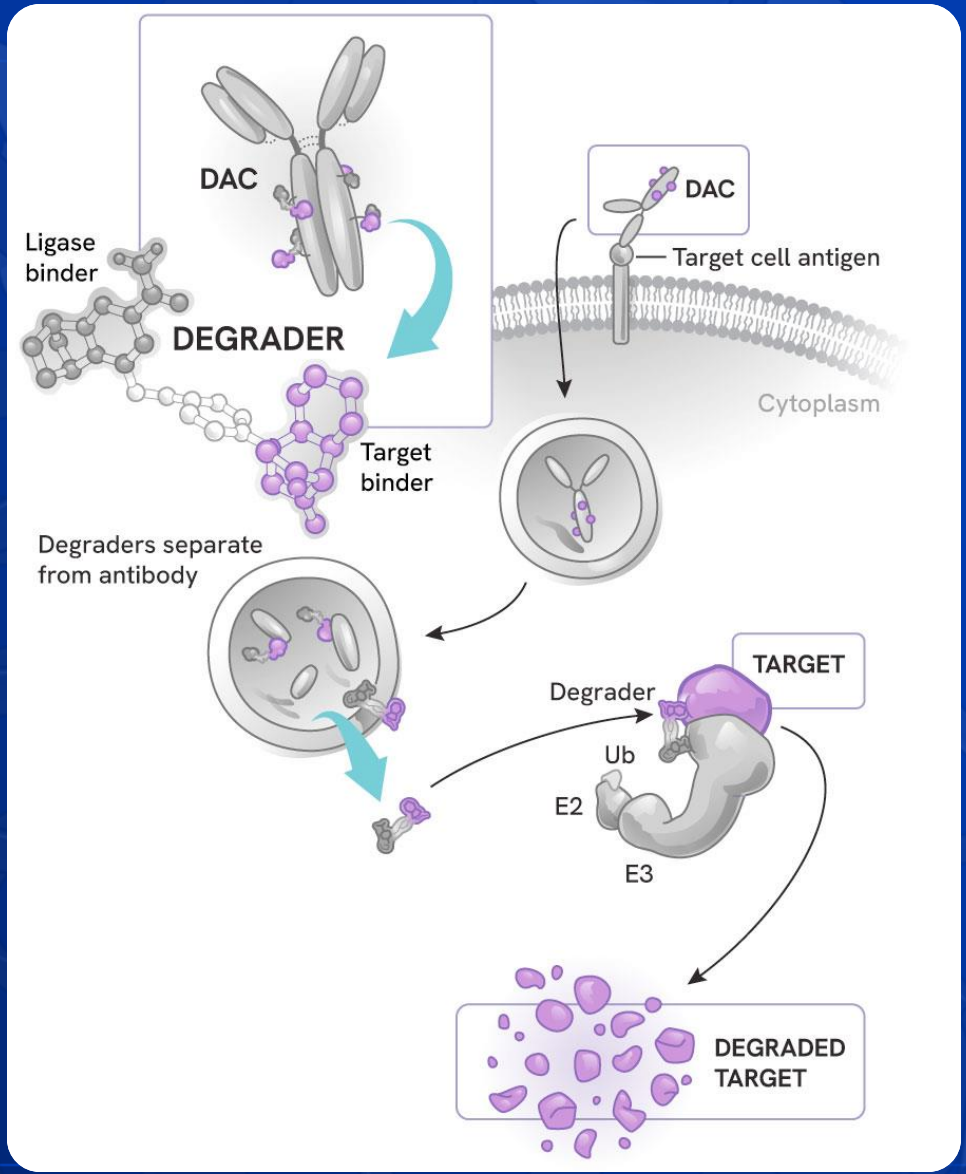
Conclusions: Encouraging Data From NX-1607 Phase 1 Single Agent Therapy

NX-1607 monotherapy demonstrates the characteristics of an active immuno-oncology agent

- NX-1607 is a first-in-class oral CBL-B inhibitor demonstrating a novel immune checkpoint mechanism distinct from PD-1/PD-L1
- NX-1607 is tolerable at pharmacologically active doses
- Oral dosing of NX-1607 demonstrated dose-dependent exposure, increases in proximal and distal biomarkers, evidence of peripheral immune activation and reductions in tumor volume and cancer biomarkers, which together provide clinical proof that CBL-B inhibition can confer anti-tumor activity
- NX-1607 monotherapy showed a high disease control rate of 49.3% with encouraging signals of clinical activity observed across multiple tumor types in heavily pretreated patients as with other successful immuno-oncology agents during early development, such as anti-PD12 and anti-CTLA43
- Data support the continued development of NX-1607 as monotherapy or in combination with other agents for the treatment of advanced solid tumors

Degrader Antibody Conjugates (DACs)

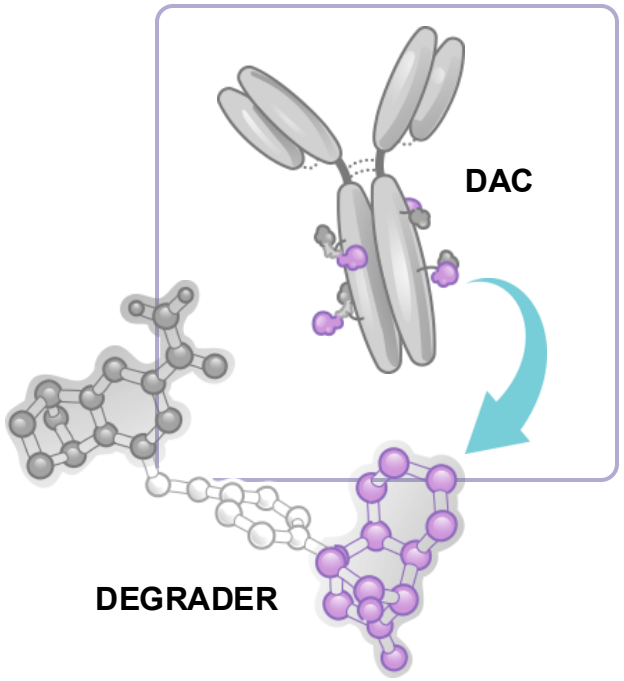
DACs represent the next evolution in targeted protein degradation, combining the highly potent and catalytic activity of degraders with the cell and tissue specificity of antibodies



Advancing a New Therapeutic Class

Degrader Antibody Conjugates (DACs)

- DACs combine the catalytic activity of a degrader with the specificity of an antibody
- DACs represent the next generation of antibody drug conjugates (ADCs)



Seagen* Deal Terms

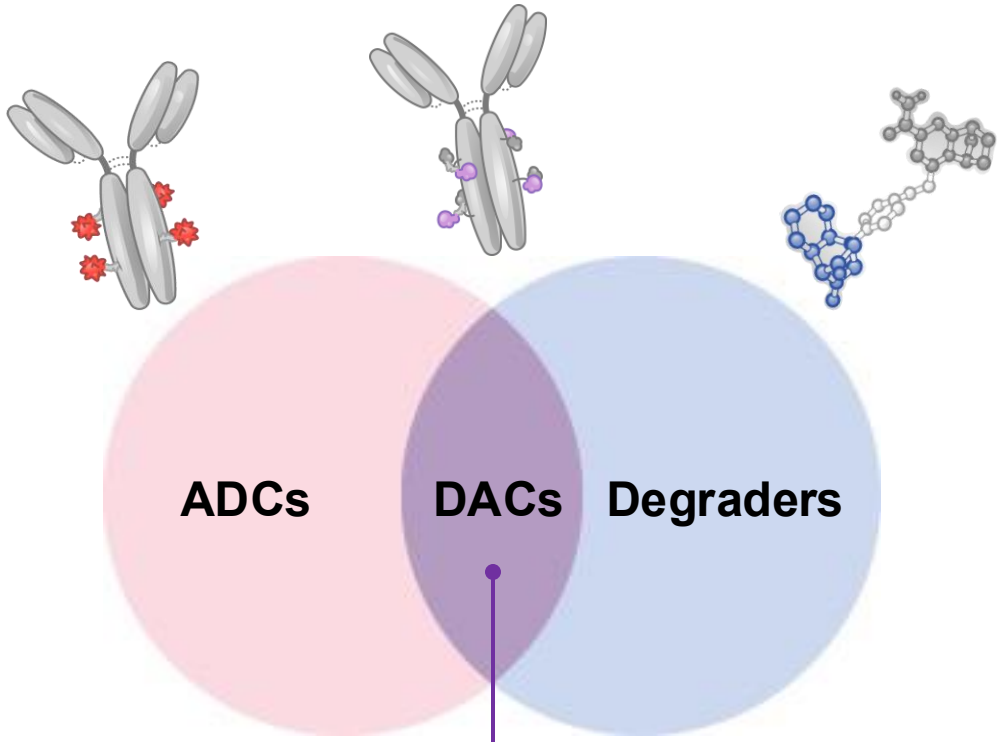
- $60 million upfront cash payment
- $3.4 billion in potential research, development, regulatory and commercial milestone payments
- Mid-single to low double-digit percentage tiered royalties on future product sales
- Option for U.S. profit sharing and co-promotion on up to two products arising from the collaboration

Pfizer

** Seagen is now part of Pfizer*

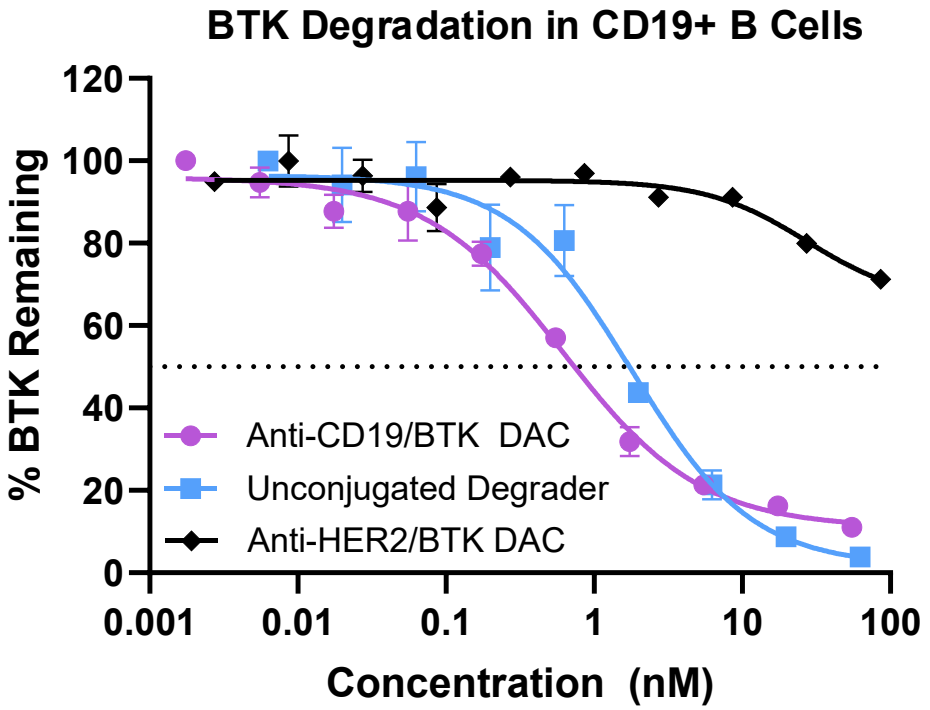
NURIX

DACs Provide Exquisite Selectivity



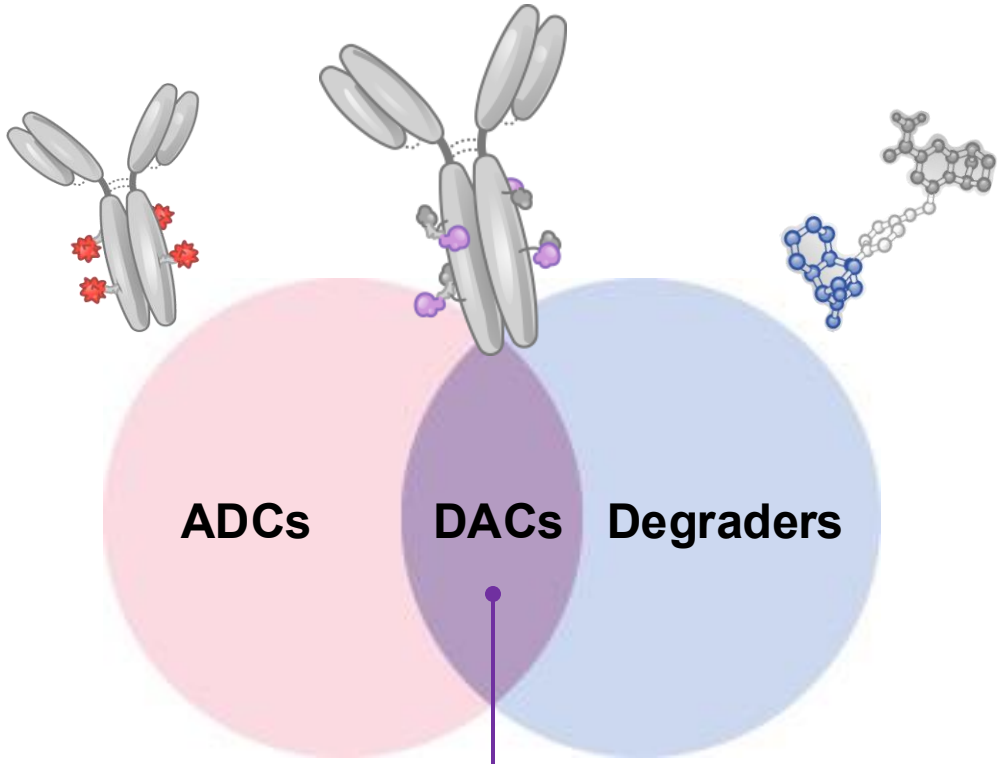
DAC provides multi-layer selectivity

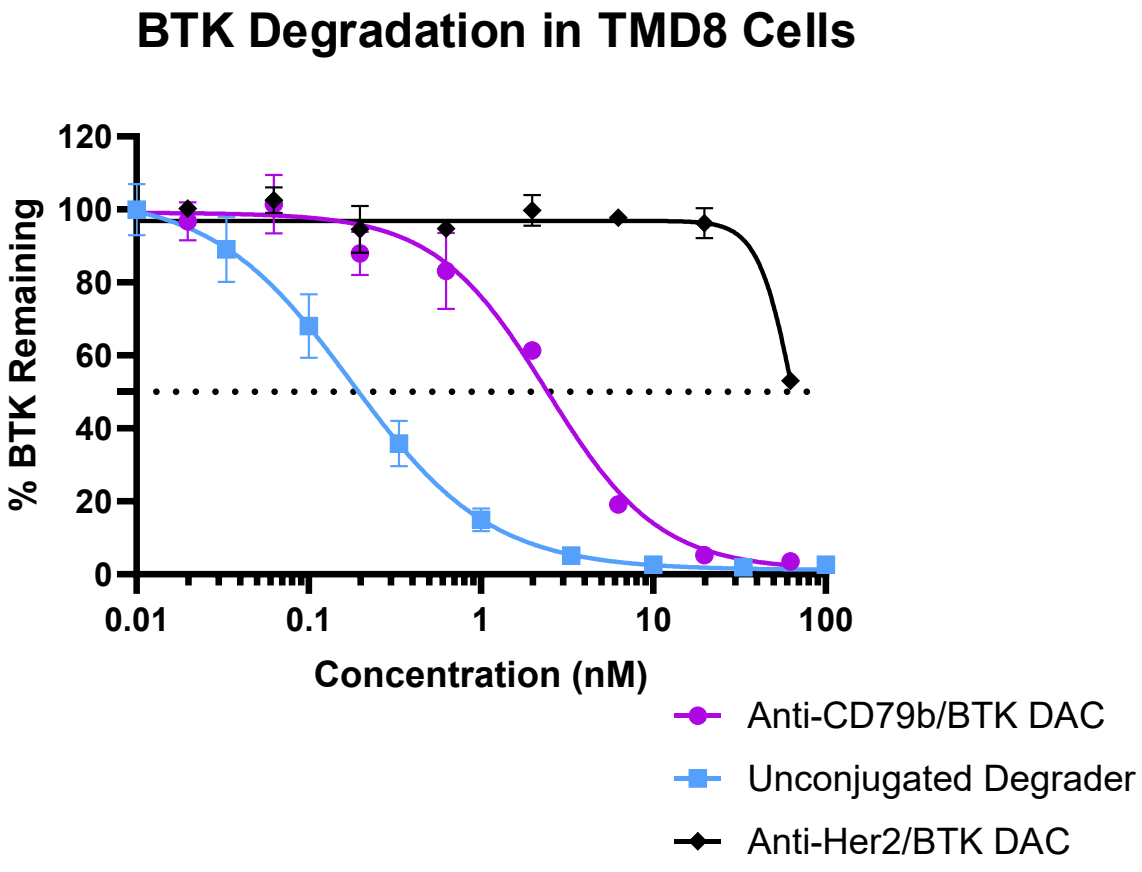
DACs Degrade Target Protein Only in Cells Expressing a Select Antigen



DACs, degrader antibody conjugates; **ADCs**, antibody drug conjugates

A BTK Degrader Conjugated to a CD79b Antibody Demonstrates Antigen-Selective Delivery and Degradation

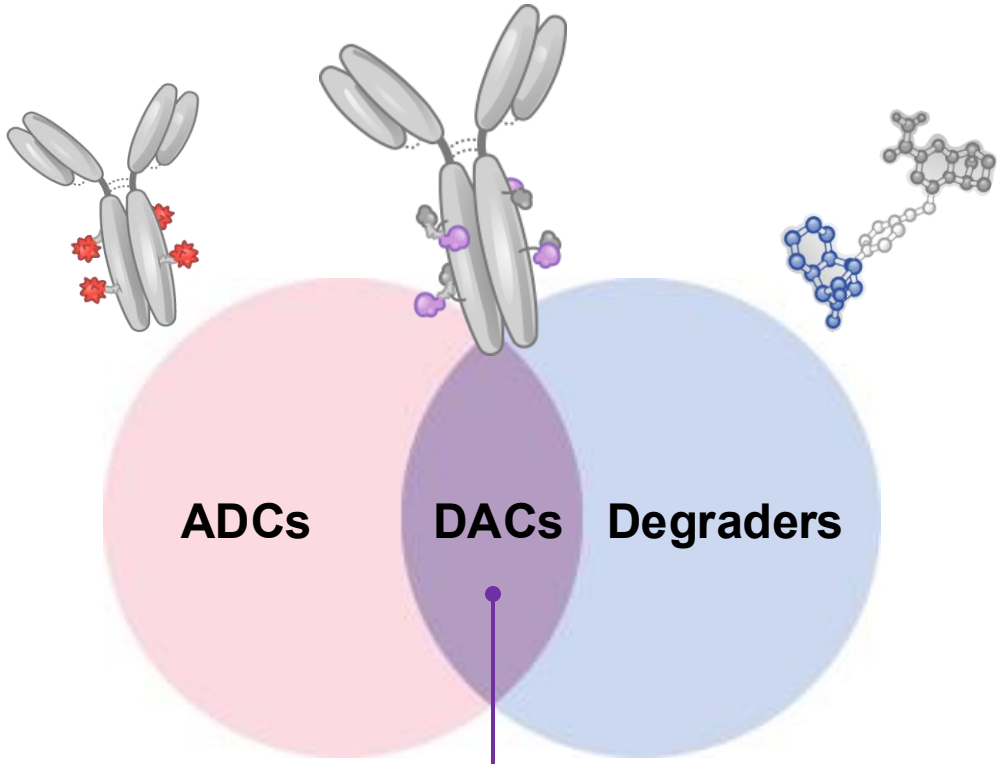


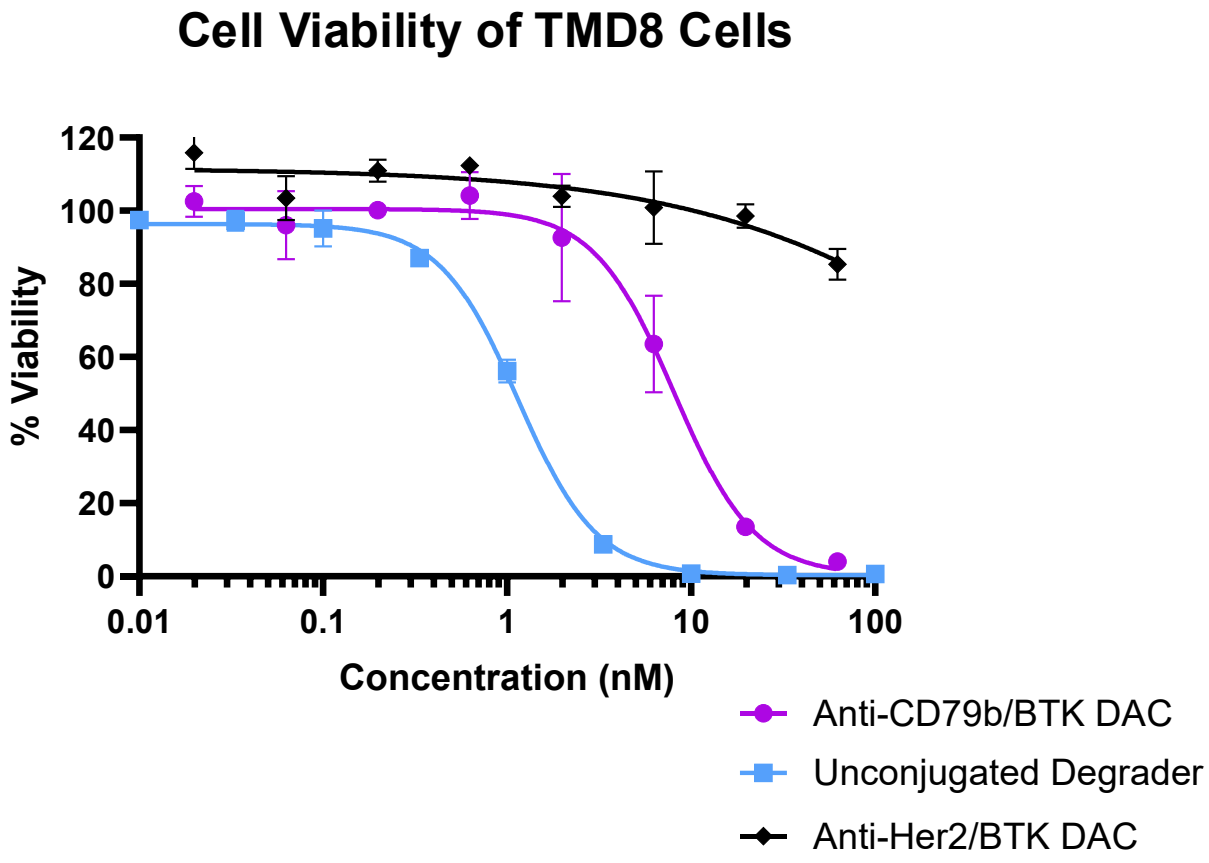


TMD8 cells are CD79b(+) and Her2 (-)

DACs, degrader antibody conjugates; **ADCs**, antibody drug conjugates

A BTK Degrader Conjugated to a CD79b Antibody Demonstrates Potent Antigen-Selective Delivery and Cell Killing





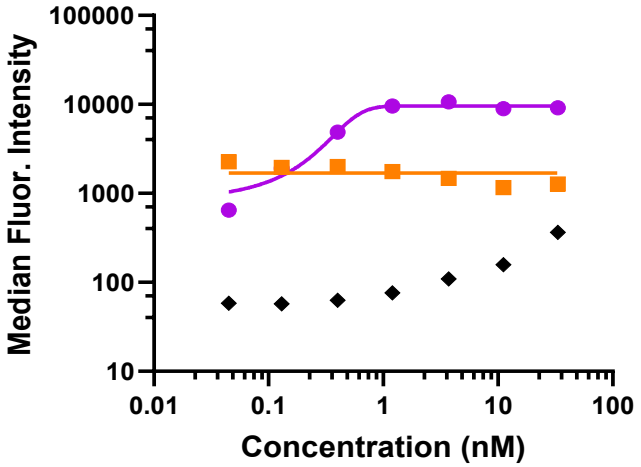
TMD8 cells are CD79b(+) and Her2 (-)

TMD8 cells are CD79b^{+} and Her2

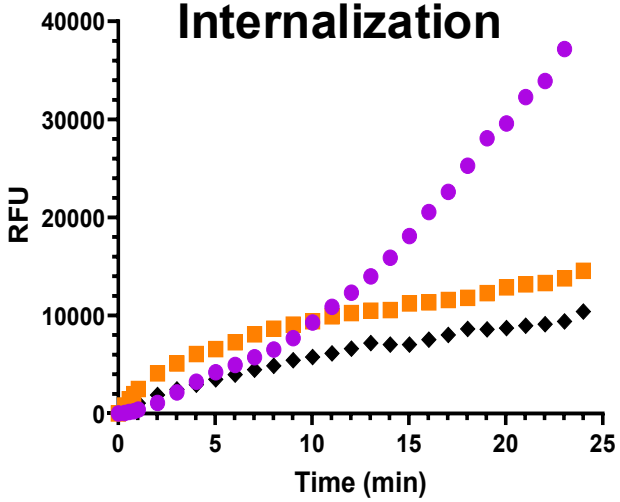
DACs, degrader antibody conjugates; **ADCs**, antibody drug conjugates

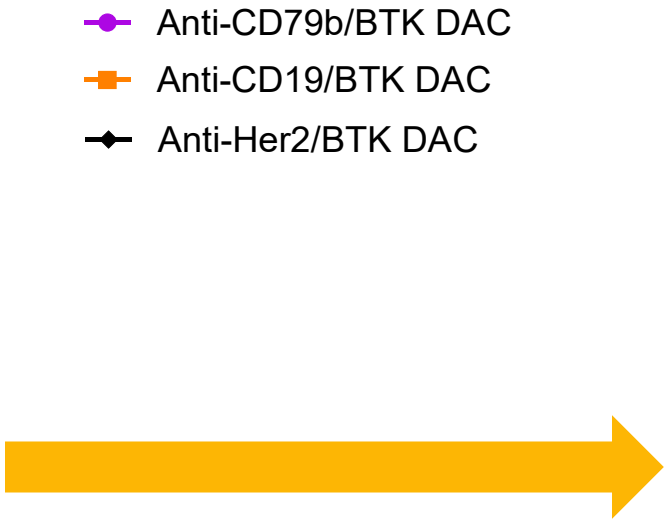
Cell Surface Binding and Internalization Strongly Impacts DAC Activity

Cell Surface Binding



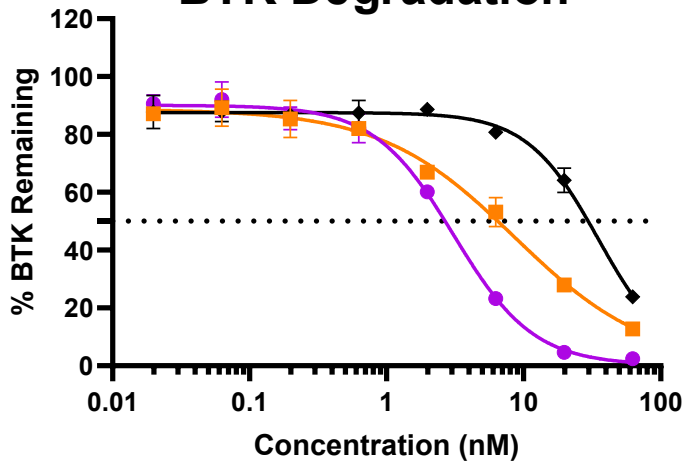
Internalization



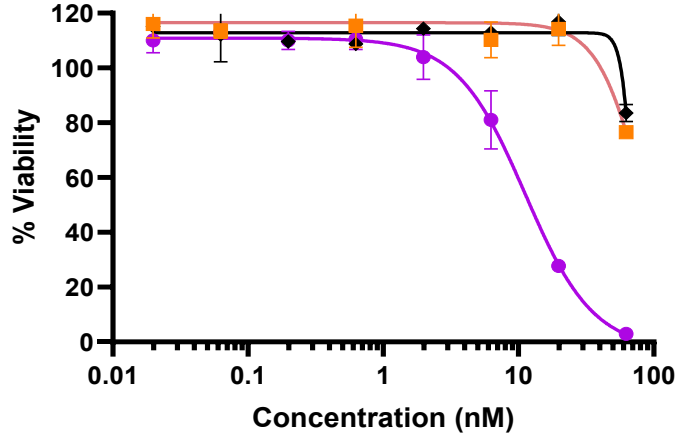


BTK DAC	DAR	% Monomer
Anti-CD79b	4	94
Anti-CD19	4	97
Anti-Her2	4	98

BTK Degradation



Cell Viability



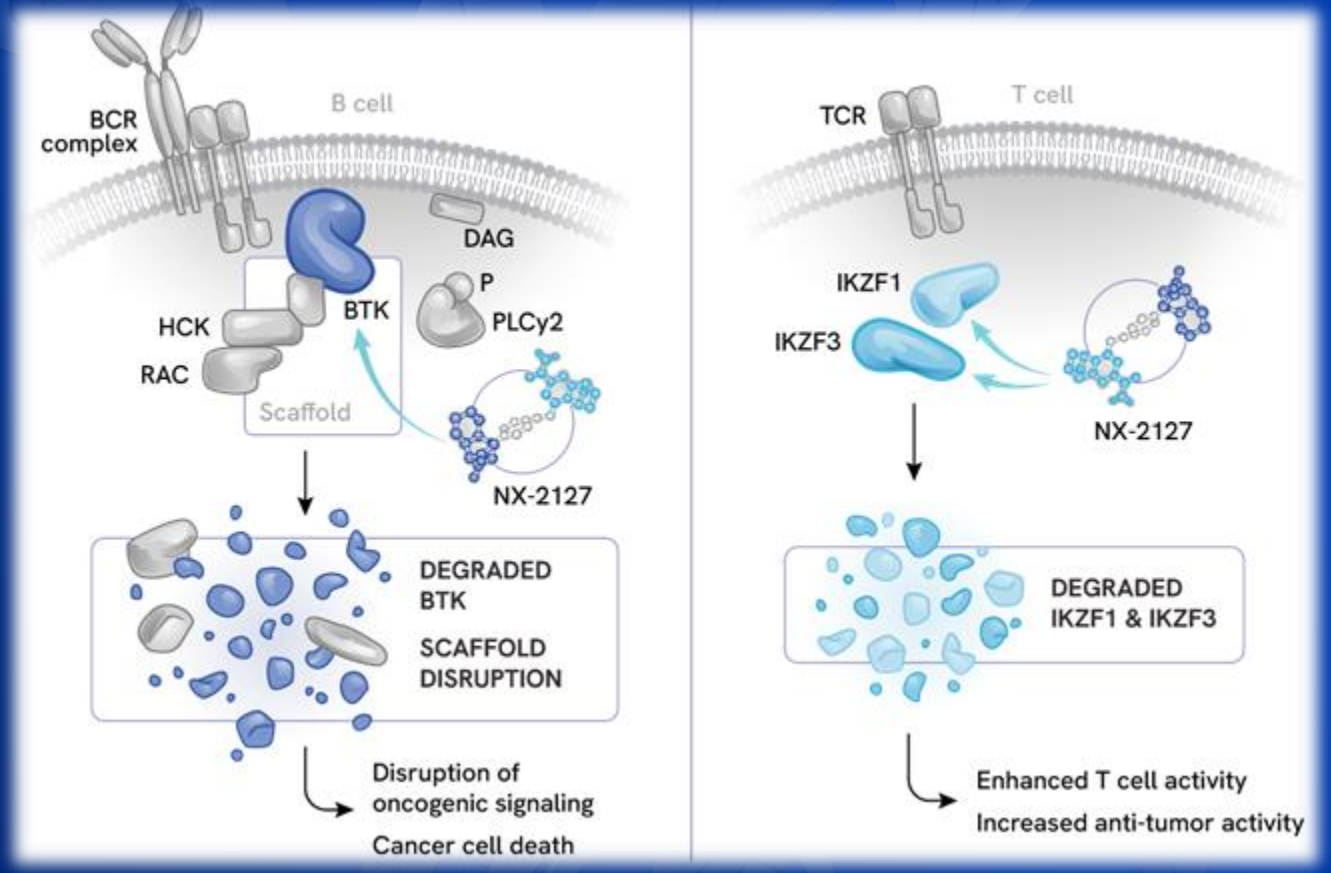
All data measure in TMD8 cells. 50 nM DACs used for the internalization assay

DACs, degrader antibody conjugates; **ADR**, antibody-degrader ratio

NURIX

Zelebrudomide (NX-2127)

Dual acting BTK/IKZF degrader with immunomodulatory activity

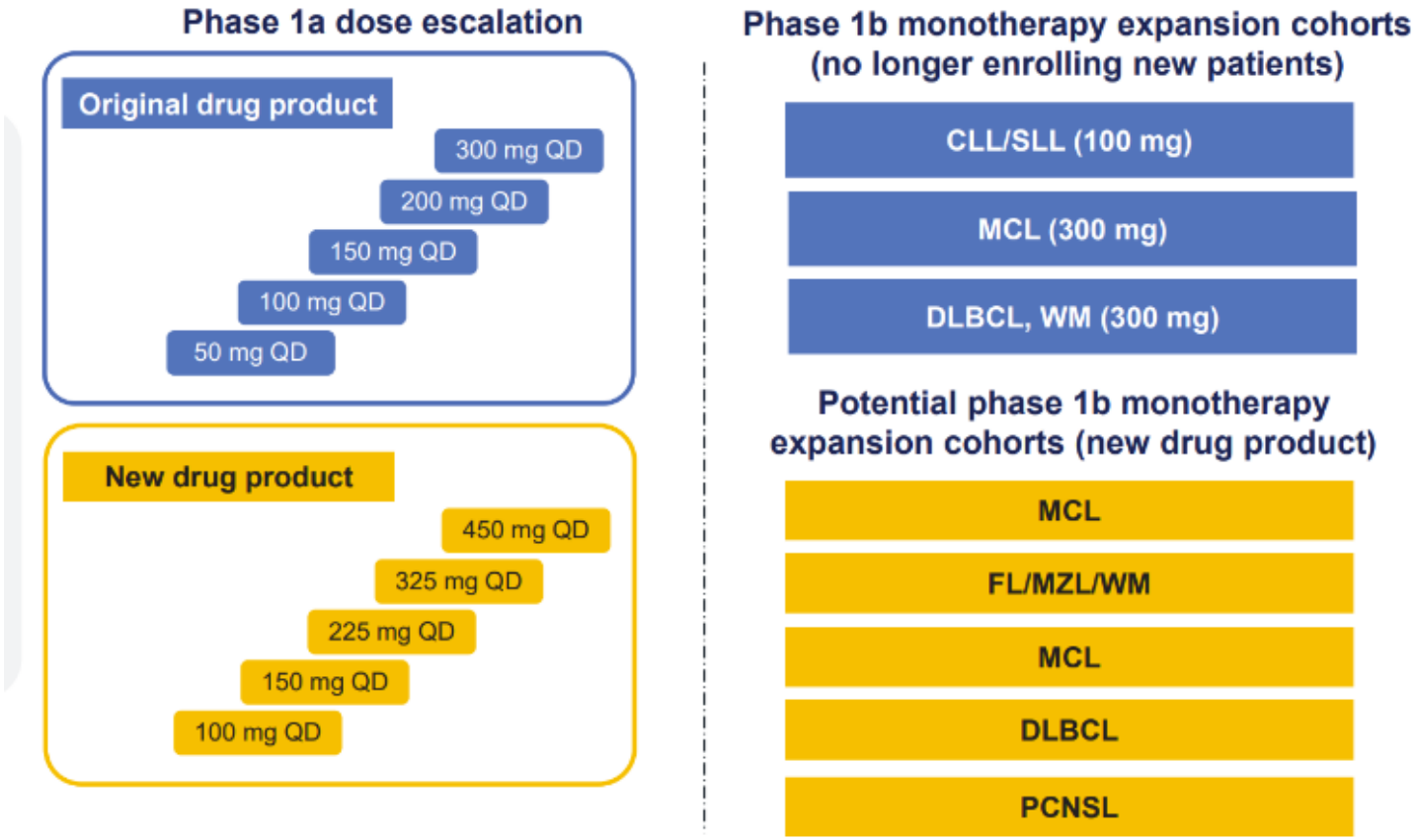


Zelebrudomide: Phase 1a/b Trial in Relapsed/Refractory B-cell Malignancies

Enrollment ongoing in dose escalation with new drug product

Key eligibility criteria

- Age ≥18 years
- Relapsed/Refractory disease
- Ph1a: ≥2 prior lines of therapy (≥1 for WM or PCNSL)
- Ph1b: Prior BTKi for CLL/SLL, MCL, WM



CLL, chronic lymphocytic leukemia; DLBCL, diffuse large B-cell lymphoma; DLT, dose-limiting toxicity; FL, follicular lymphoma; MCL, mantle cell lymphoma; MTD, maximum tolerated dose; MZL, marginal zone lymphoma; PD, pharmacodynamics; PK, pharmacokinetics; PCNSL, primary central nervous system lymphoma; SLL, small lymphocytic lymphoma; WM, Waldenstrom's macroglobulinemia

NURIX

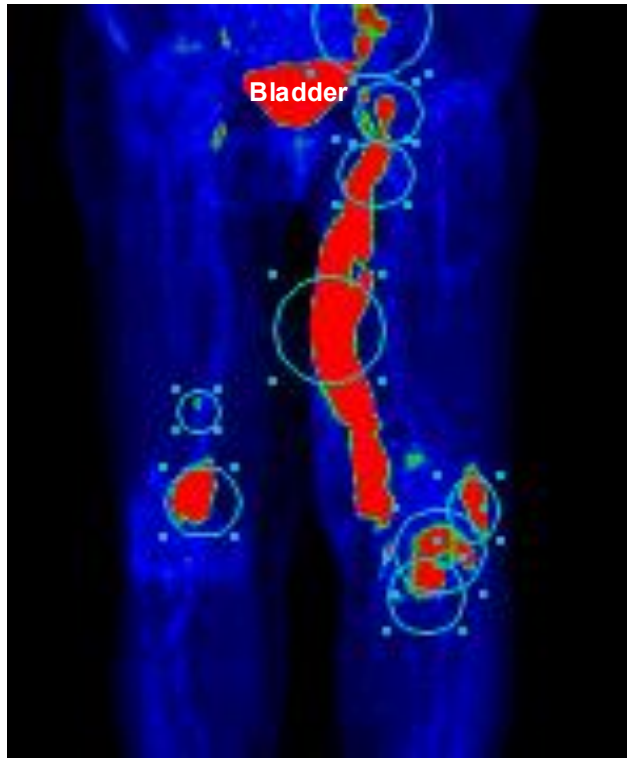
Rapid and Sustained Complete Response with NX-2127 in a Patient with WM Transformed to DLBCL

Case History

- 84-year-old female with WM diagnosed in 2003 with DLBCL (ABC subtype) transformation in 2015
  - MYD88 and CXCR4 mutation
- 4 prior lines of aggressive therapy
  - R-CHOP (CR)
  - R-ICE (PR)
  - Rituximab, mogamulizumab (anti-CCR4), magrolimab (anti-CD47)
  - Rituximab, ibrutinib, and lenalidomide
- Complete response on first assessment at week 8, confirmed at week 16
- **As of November 18, 2024, this patient remains in complete response and on treatment with 33 months of follow up (2.75 years)**

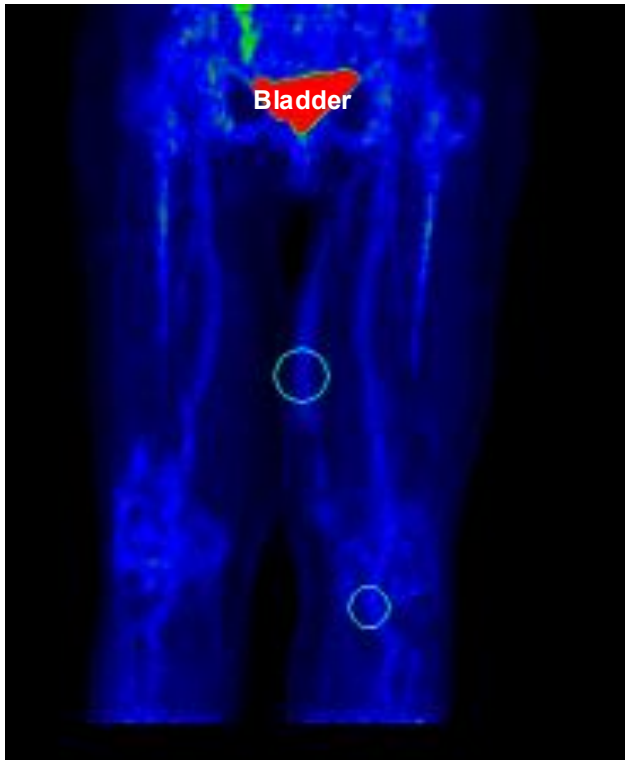
FDG-PET CT Scan Disease Assessment

Baseline



Deauville score: 5

Confirmatory Week 16 Scan



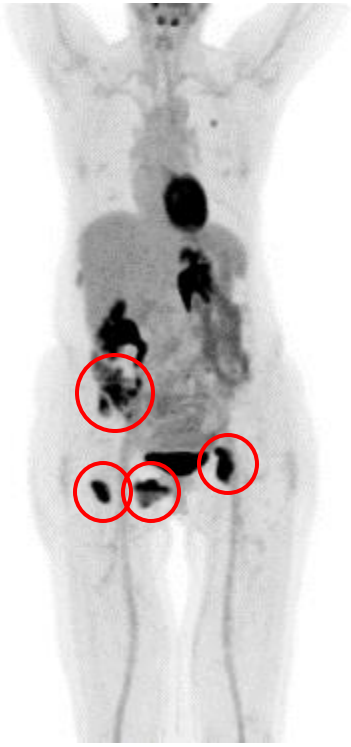
Deauville score: 2

Status update as of November 18, 2024

Rapid and Sustained Complete Response in Relapsed/Refractory MCL With Zelebrudomide

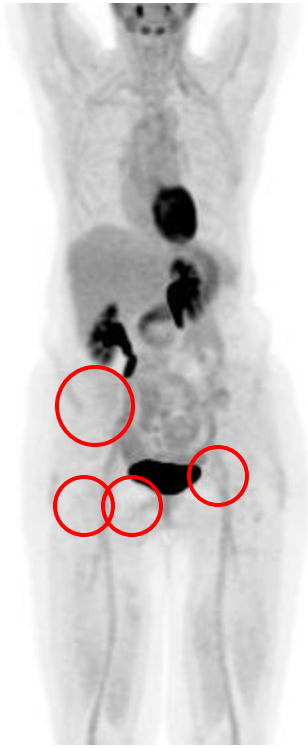
FDG-PET CT Scan Disease Assessment

Baseline



Deauville score: 5

Week 8 Scan



Deauville score: 2

Case History

- 64-year-old woman with multiply relapsed MCL, following:
  - 2016: Rituximab + CHOP; R-hyper-CVAD; cytarabine
  - 2017: Hematopoietic stem cell transplantation (HSCT)
  - 2016-2019: Rituximab, ibrutinib, cytarabine
- Complete response on first assessment at week 8, confirmed at week 16
- She came off therapy on August 28, 2023, after 17 cycles of therapy
- **Approximately 1 year later, as of July 18, 2024, she had no evidence of disease by PET CT and was not on any active treatment for MCL.**

NX-2127 Status and Next Steps

Status

- We have re-initiated enrollment with the new, chirally controlled drug product
- We are focused on the aggressive lymphomas for development of zelebrudomide where the combination of BTK degradation and IKZF1/3 degradation have the potential for synergy and significant therapeutic benefit

Next Steps

- Complete dose escalation with new drug product and select recommended Phase 1b dose for selected indications
- Additional clinical data will be shared after selection of a Phase 1b expansion dose(s) and indication(s)

Thank You

Abbreviations

- AD = Atopic dermatitis
- BP = Bullous pemphigoid
- COPD = Chronic obstructive pulmonary disease
- CD = Crohn’s disease
- CRwNP = Chronic rhinosinusitis with nasal polyps
- CSU = Chronic spontaneous urticaria
- CLE = Cutaneous lupus erythematosus
- EoE = Eosinophilic esophagitis
- wAIHA = Warm autoimmune hemolytic anemia
- HS = Hidradenitis suppurativa
- MS = Multiple sclerosis
- PsA = Psoriatic Arthritis
- PN = Prurigo nodularis
- RA = Rheumatoid arthritis
- SLE = Systemic lupus erythematosus without Lupus Nephritis
- UC = Ulcerative Colitis