NURIX™

Protein Degraders to Outmatch Cancer and Autoimmune Disease

September 2026 Corporate Presentation



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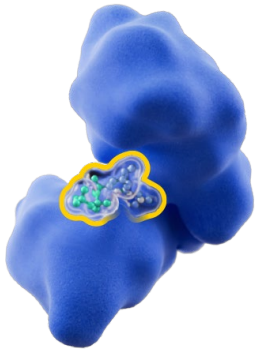
OUR MISSION

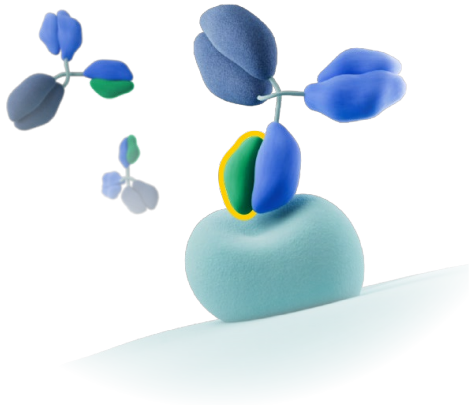
To establish degrader-based medicines at the forefront of patient care

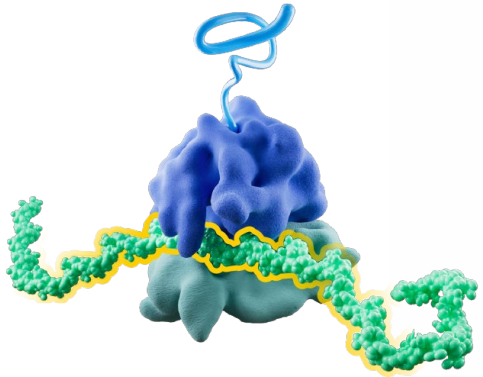
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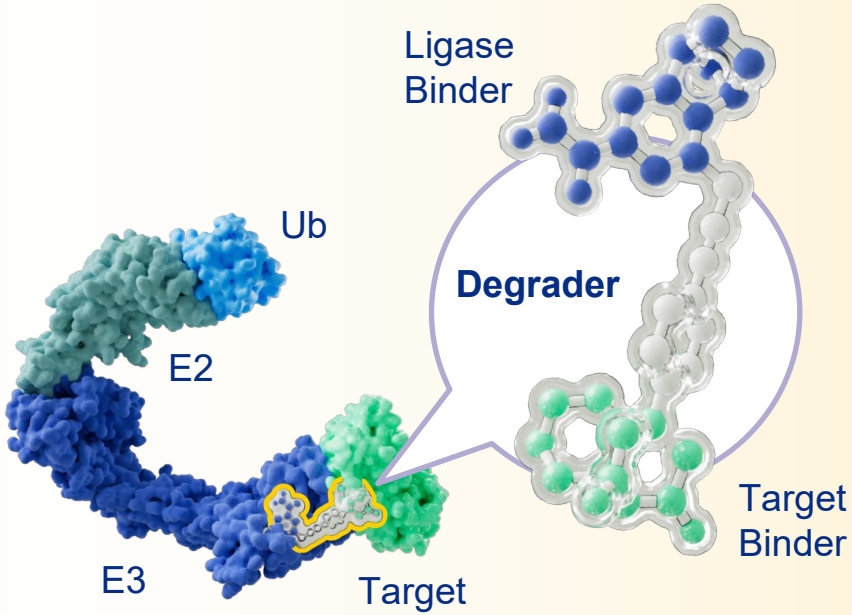
Leading the Next Frontier in Drug Development

Target Protein Degradation (TPD)









Small Molecule Inhibitors

Antibodies

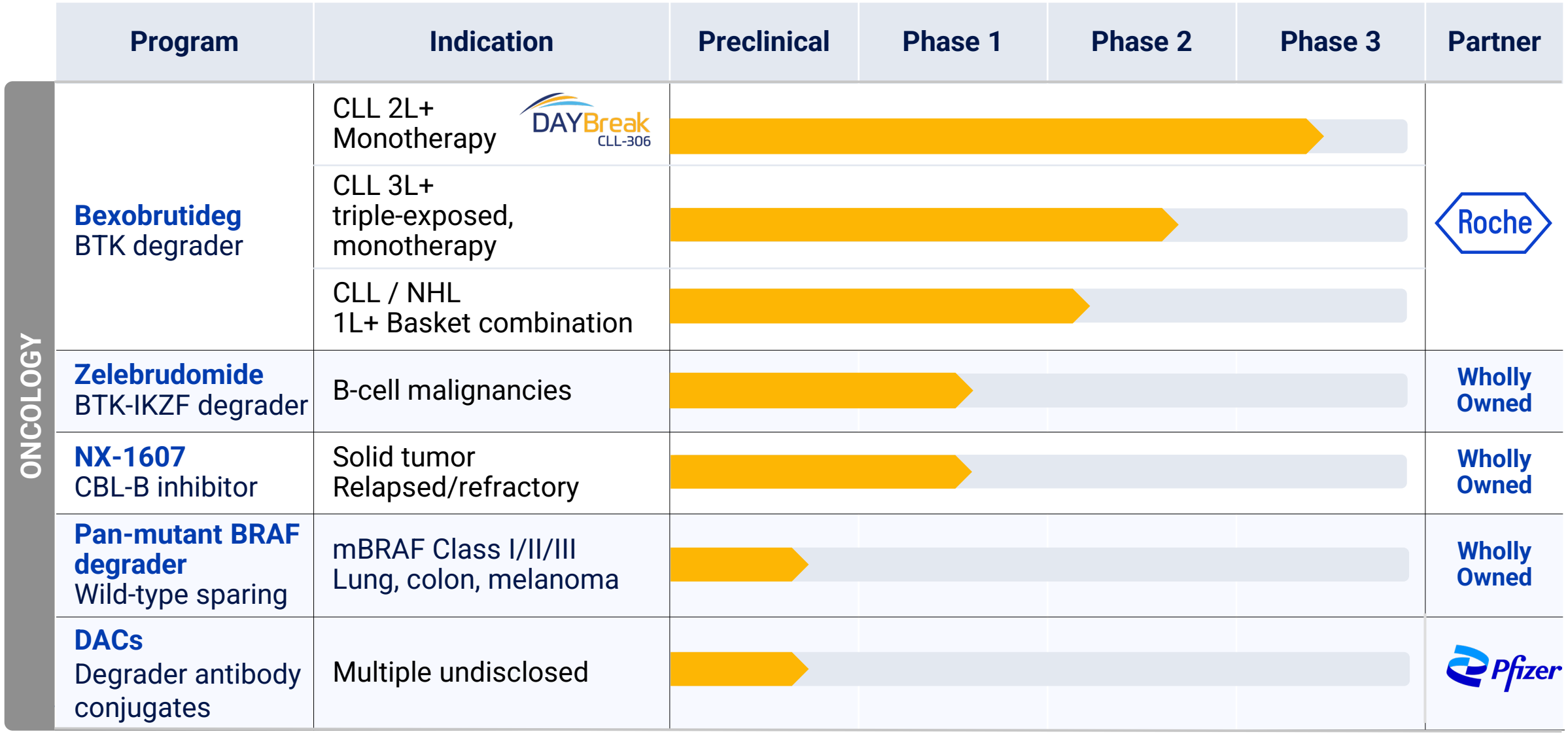
Nucleic Acid-based Therapies:
Antisense • RNAi Gene Therapy • CRISPR

Target Protein Degradation Drugs

Evolution of New Therapeutic Modalities

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Nurix Has Built a Pipeline of Degrader-Based Medicines to Address Unmet Medical Needs in Oncology

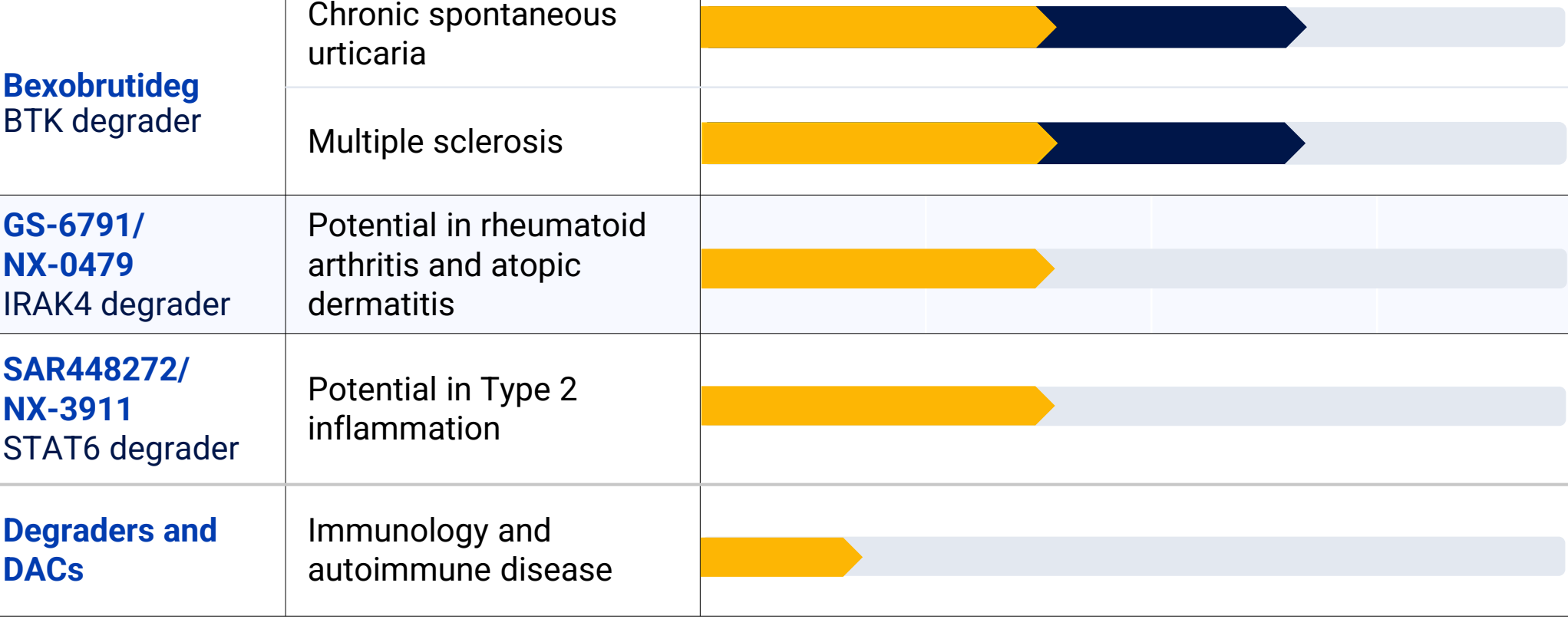


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Nurix Has Expanded Its Pipeline into Immunology and Neurology Through Strategic Partnerships

IMMUNOLOGY & NEUROLOGY

Program	Indication	Preclinical	Phase 1	Phase 2	Phase 3	Partner
Bexobrutideg BTK degrader	Chronic spontaneous urticaria					Roche
	Multiple sclerosis					
GS-6791/ NX-0479 IRAK4 degrader	Potential in rheumatoid arthritis and atopic dermatitis					GILEAD
SAR448272/ NX-3911 STAT6 degrader	Potential in Type 2 inflammation					sanofi
Degraders and DACs	Immunology and autoimmune disease					**Wholly Owned**

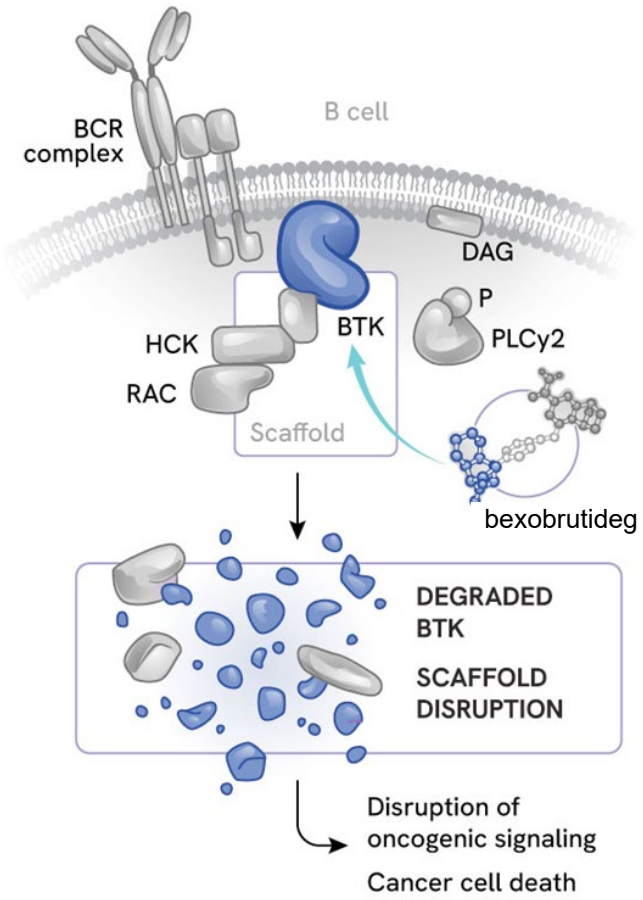


Ongoing Planned

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Bexobrutideg: Targeted Protein Degradation of BTK with Potential Best-in-Class Profile

Eliminates both the enzymatic and scaffolding activities of BTK



- 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
- Demonstrated robust clinical activity in difficult to treat B-cell malignancies

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

A Transformative Partnership to Maximize the Value of Bexobrutideg

Accelerating and expanding the potential of bexobrutideg through a shared ambition to transform patient outcomes across **oncology**, **immunology** and **neurology**

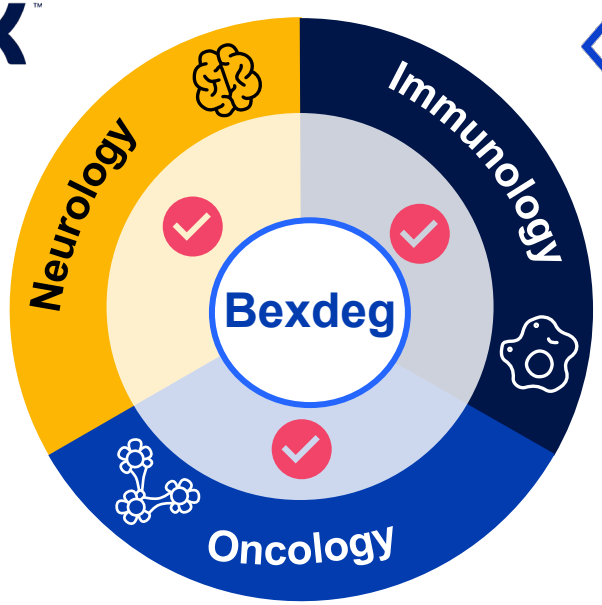
UPFRONT	TOTAL PAYMENTS UP TO	U.S. PROFIT SPLIT	GLOBAL DEV COST SPLIT	EX-U.S. ROYALTIES
$700M	**$2.3B**	**50/50**	**40/60**	**Low–High Teens**
Cash to Nurix at close	Upfront payment plus development, regulatory, and commercial milestones	Equal split of U.S. profits & losses	Nurix 40% Roche 60%	Tiered royalties on ex-U.S. net sales

Co-development and co-commercialization in the U.S. accelerates Nurix's evolution into a fully integrated biopharmaceutical company in major medical markets

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Bexobrutideg: A Complimentary Strategic and Portfolio Fit with Roche

Positions bexobrutideg as a potential **backbone therapy across multiple BTK-dependent diseases**, with accelerated global scale and significantly expanded commercial opportunity



A global leader in oncology, immunology and neurology with expertise and capabilities in **establishing B-cell targeted therapies as a new standard of care** across therapeutic areas

> ***At Roche, our goal is to create new possibilities for patients with challenging diseases. We believe bexobrutideg could represent a major leap forward in the fight against complex blood cancers and other diseases. We are proud to join forces with Nurix to accelerate these potential breakthroughs.***
>
> ***Dr. Levi Garraway, Head of Global Product Development and Chief Medical Officer, Roche***

Bexobrutideg: A Robust Clinical Development Plan Across Oncology, Immunology, and Neurology

Nurix-Roche partnership enables comprehensive bexobrutideg development across **multiple lines of therapy** and **combination settings in B-cell malignancies** and more broadly across **immunology and neurology**

CLL
Chronic lymphocytic leukemia

DAYBreak CLL-306

- DAYBreak CLL-201 | Phase 2 – 3L+ Single Arm
- DAYBreak CLL-306 | Phase 3 – 2L+ vs Pirtobrutinib
- Phase 1b/2 Study – 1L+ Basket Combination Study
  - Planned Phase 3 – 2L Combination
  - Planned Phase 3 – 1L (best regimen to be determined)

MCL
Mantle cell lymphoma

- Planned Phase 3 – Combination

WM
Waldenström macroglobulinemia

- Planned Phase 3 – Combination

CSU
Chronic spontaneous urticaria

- Planned Phase 2

MS
Multiple sclerosis

- Planned Phase 2

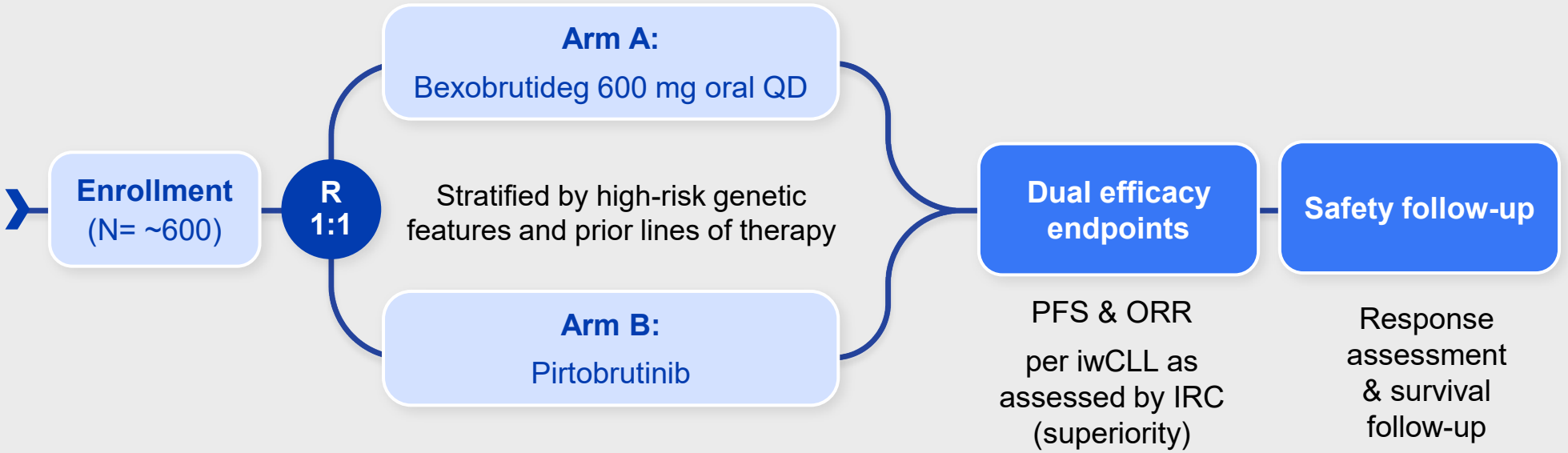
DAYBreak Phase 3 Trial Positions Bexobrutideg for Full Approval

DAYBreak CLL-306

TRIAL DESIGN

Key Eligibility

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



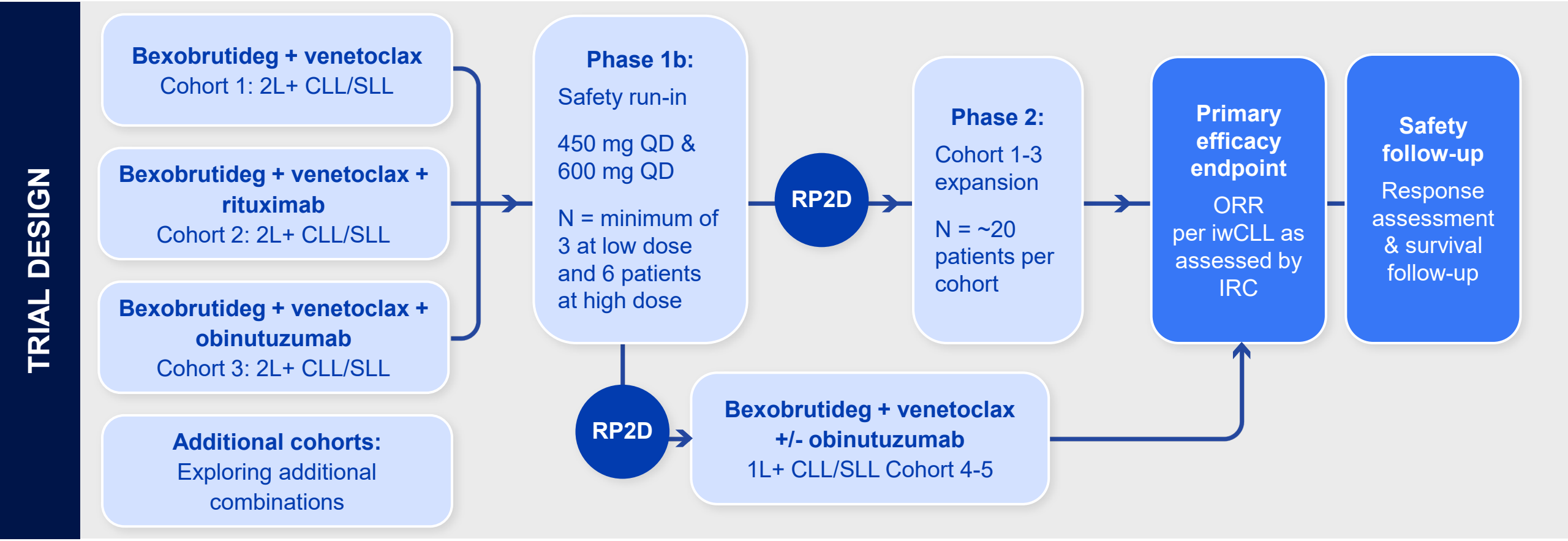
- ✓ Designed to evaluate superiority to latest approved BTKi, pirtobrutinib
- ✓ Single trial strategy to support global approval and establish superiority of degrader mechanism of action

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 and Potentially Earlier Lines 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*

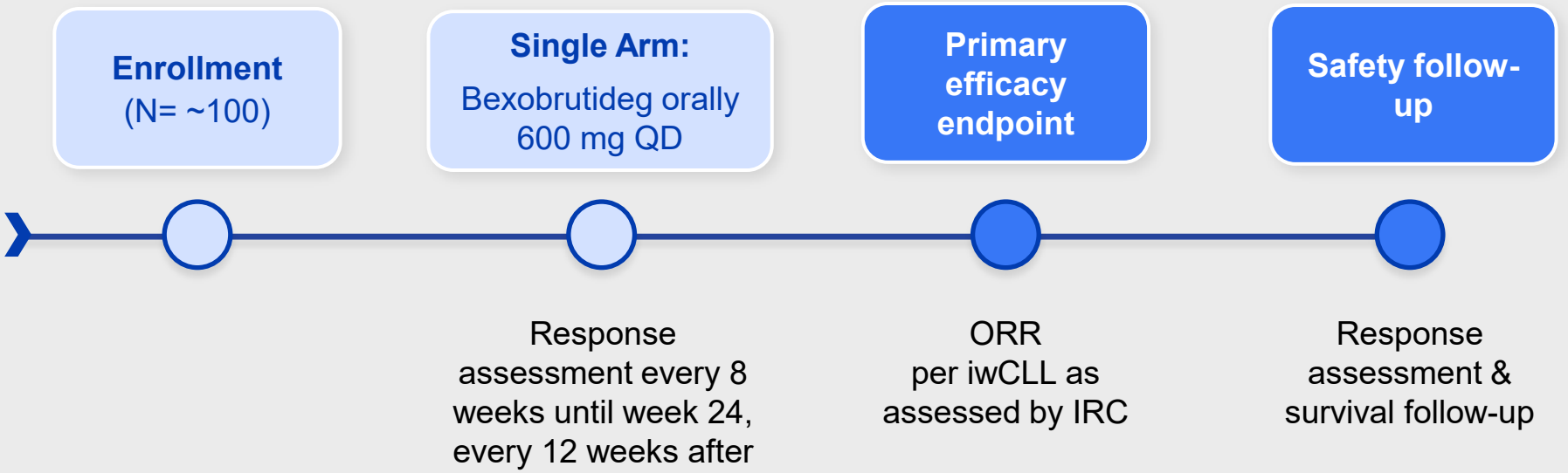
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DAYBreak Pivotal Phase 2 Single-Arm Study Designed to Support Bexobrutideg Accelerated Approval

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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Bexobrutideg Phase 1a/b Clinical Data in CLL Supports Potential Best-in-Class Profile



Bexobrutideg Phase 1a/b (NX-5948-301) Trial Design

Ongoing Phase 1a/b clinical trial in adults with relapsed/refractory B-cell malignancies

Phase 1a dose escalation (fully enrolled)

CLL/SLL

- 600 mg QD
- 450 mg QD
- 300 mg QD
- 200 mg QD
- 100 mg QD
- 50 mg QD

CLL Phase 1b randomized cohort 1 (fully enrolled; 200 vs 600 mg)

- **CLL/SLL 200 mg QD Prior BTKi and BCL2i**
- **CLL/SLL 600 mg QD Prior BTKi and BCL2i**

CLL Phase 1b expansion, other cohorts (ongoing; all 600 mg)

- Non-C481S BTK mutations, prior BTKi and BCL2i
- Prior non-covalent BTKi, no BCL2i
- TP53 or 17p deletion, 2L, prior BTKi, no BCL2i
- 2L+, prior BTKi, no BCL2i
- BTKi-naïve
- With wAIHA, prior BTKi
- With CNS involvement, prior BTKi

WM/NHL

- 600 mg QD
- 450 mg QD
- 300 mg QD
- 200 mg QD
- 100 mg QD
- 50 mg QD

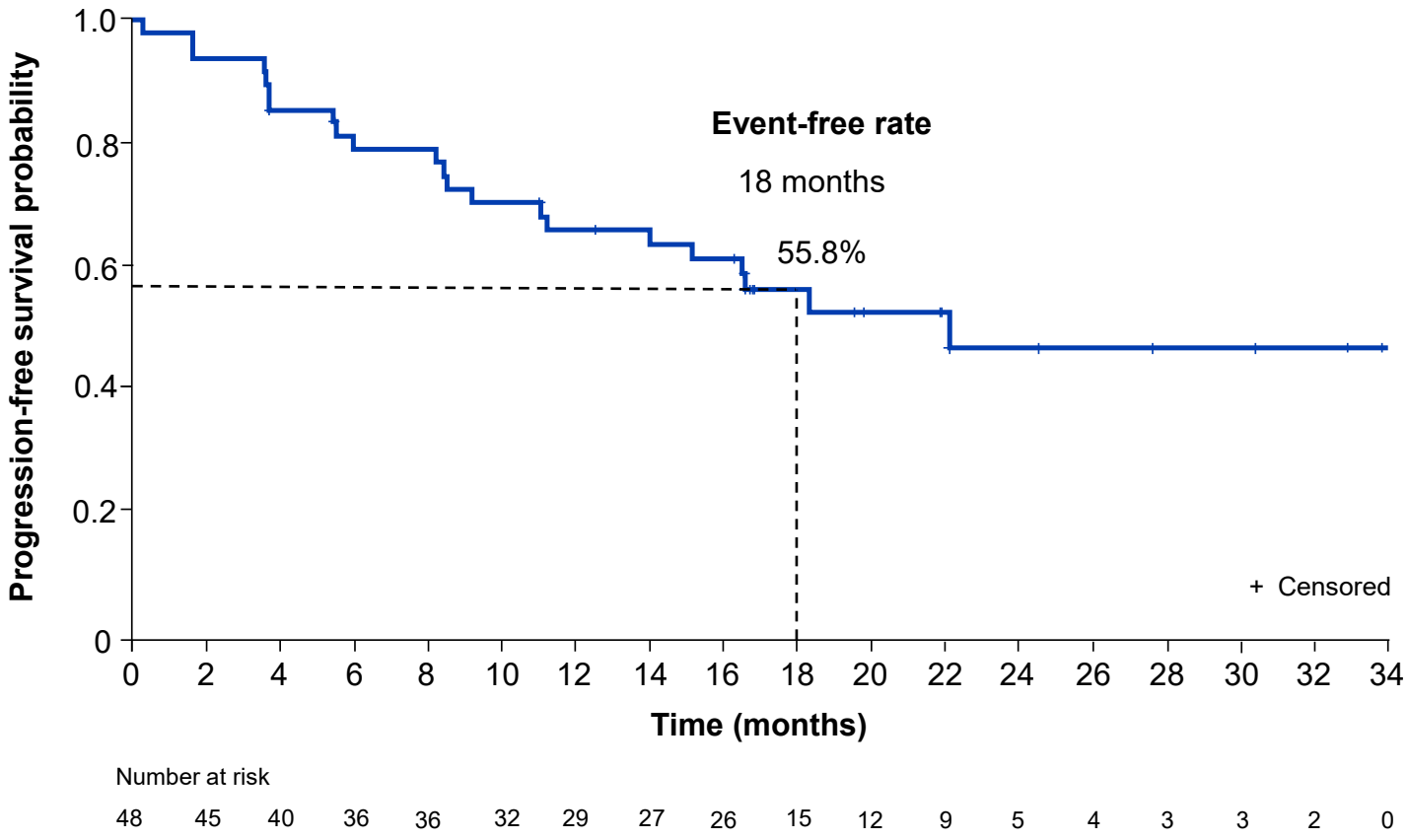
NHL/WM Phase 1b expansion cohorts (600 mg)

- **MZL** Marginal zone lymphoma
- **FL** Follicular lymphoma
- **WM** Waldenström macroglobulinemia
- **MCL** Mantle cell lymphoma
- **DLBCL** Diffuse large B-cell lymphoma
- **PCNSL** Primary CNS lymphoma

***2L+**, second line +; **BCL2i**, B-cell lymphoma 2 inhibitor; **BTKi**, Bruton's tyrosine kinase inhibitor; **CLL**, chronic lymphocytic leukemia; **CNS**, central nervous system; **NHL**, non-Hodgkin's lymphoma; **QD**, once daily; **SLL**, small lymphocytic lymphoma; **wAIHA**, warm autoimmune hemolytic anemia; **WM**, Waldenström macroglobulinemia*

Bexobrutideg PFS and ORR in Relapsed/Refractory Patients (Phase 1a)

PFS remains durable with a median of 22.1 months



Relapsed/refractory CLL/SLL	
PFS summary	
	n=48
Median PFS[a], months (95% CI)	**22.1** (14.0–NR)
ORR summary	
	n=47[b]
ORR, % (95% CI)[c]	**83.0** (69.2–92.4)
CR	2 (4.3)
nPR	1 (2.1)
PR	36 (76.6)
SD	6 (12.8)
PD	2 (4.3)
Median follow-up,[d] months (range)	22.4 (16.9–35.7)

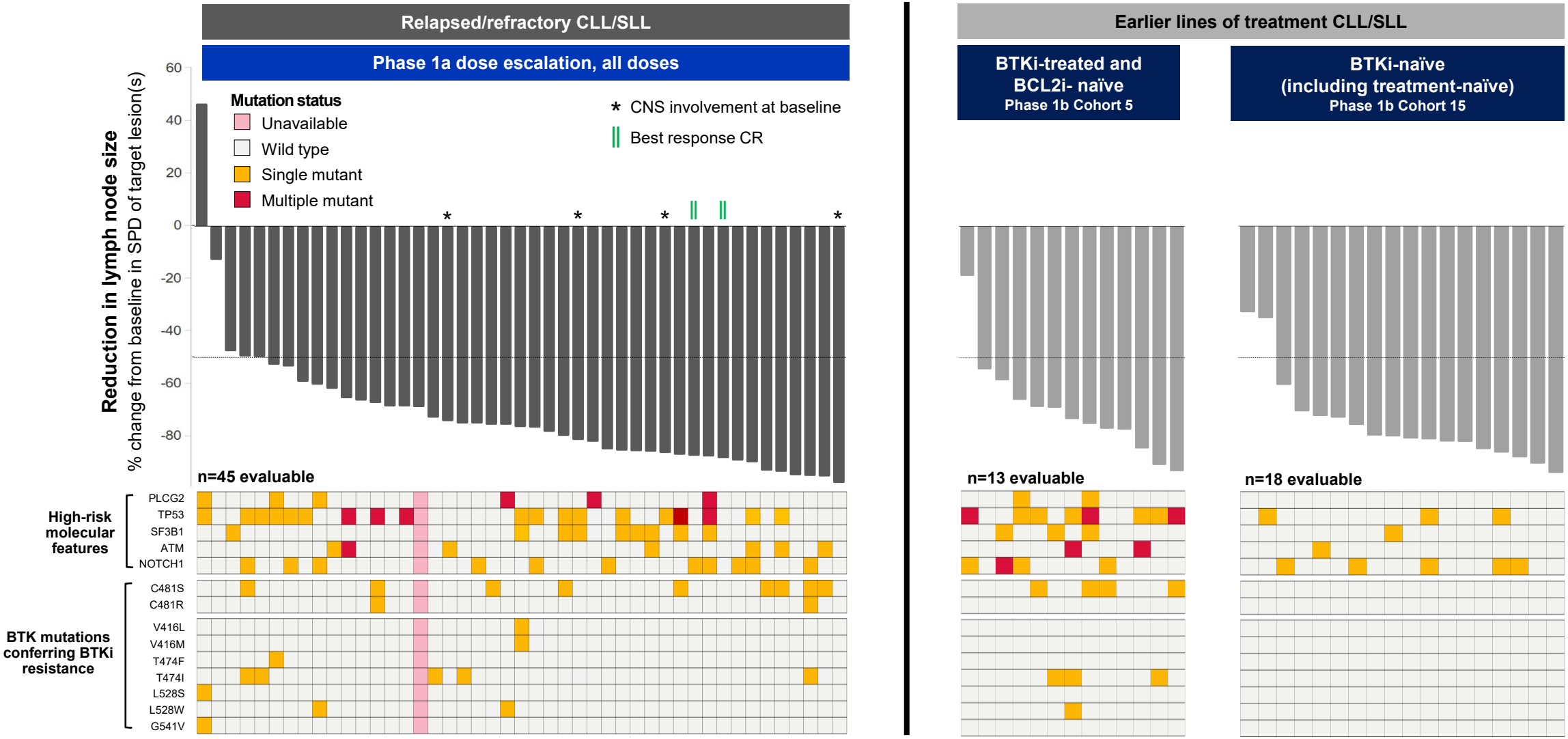
***CI**, confidence interval; **CLL**, chronic lymphocytic leukemia; **CR**, complete response; **nPR**, nodal partial response; **NR**, not reached; **ORR**, objective response rate; **PD**, progressive disease; **PFS**, progression-free survival; **PR**, partial response; **PR-L**, partial response with lymphocytosis; **SD**, stable disease; **SLL**, small lymphocytic lymphoma*

[a]Median for PFS by Kaplan–Meier method; [b]Excludes 1 patient who was not evaluable for response; [c]Objective response rate includes CR + nPR + PR + PR-L; [d]Time from treatment start to data cutoff

Data cutoff: 1 Jan 2026

Reduction in Lymph Node Size in Patients Treated with Bexobrutideg

Bexobrutideg demonstrates broad clinical activity including in high-risk patients



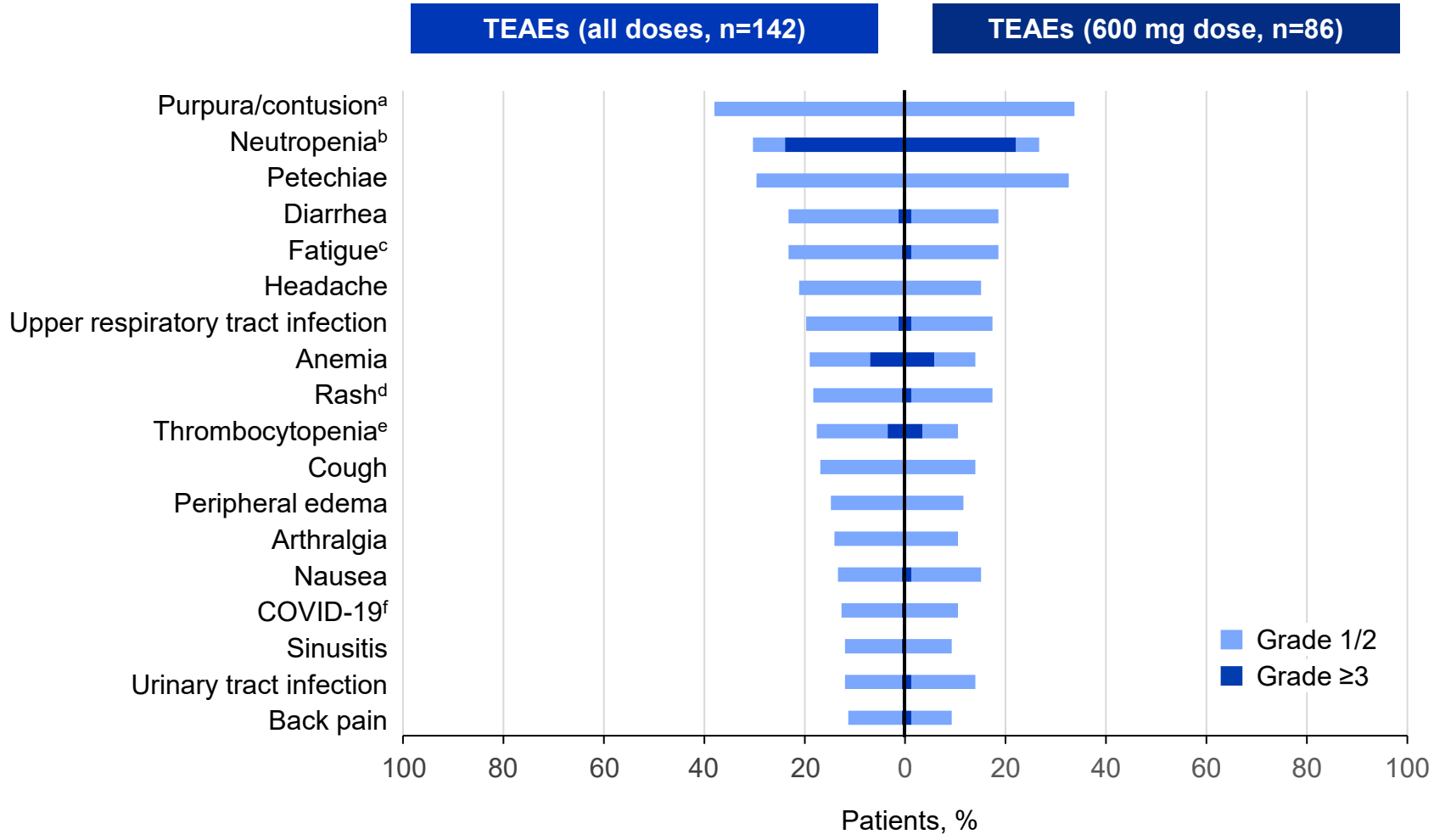
***ATM**, ataxia-telangiectasia mutated; **BTK**, Bruton's tyrosine kinase; **BTKi**, BTK inhibitor; **CLL**, chronic lymphocytic leukemia; **CNS**, central nervous system; **CR**, complete response; **NOTCH1**, neurologic locus notch homolog protein 1; **PLCG2**, phospholipase C gamma 2; **SLL**, small lymphocytic lymphoma; **SPD**, sum of products diameters*

Waterfall plot includes patients with measurable lymph node status; mutations were reported at VAF >5%; Patients could have no mutations, a single mutation, or multiple mutations

Data cutoff: 1 Jan 2026

TEAEs in ≥10% of Patient Population (Phase 1a/b)

Tolerable AE profile: comparable between overall population and patients receiving 600 mg dose



- Tolerable safety profile consistent with prior disclosures
- Safety at 600mg RP2D equal to or better than overall population across doses
- No dose-limiting toxicities
- Low 5.6% discontinuation rate due to AEs
- Three Grade 5 AE, all deemed not related (pneumonia, pulmonary embolism, NOS)

***AE**, adverse event; **CLL**, chronic lymphocytic leukemia; **NOS**, not otherwise specified; **RP2D**, recommended Phase 2 dose; **SLL**, small lymphocytic lymphoma; **TEAE**, treatment-emergent adverse event*

[a]Purpura/contusion includes episodes of contusion or purpura; [b]Aggregate of 'neutrophil count decreased' or 'neutropenia'; [c]Fatigue was transient; [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'

Data cutoff: 1 Jan 2026

Bexobrutideg Data Supports a Potential Best-in-Class Profile Relative to the Latest Approved BTK Inhibitor Pirtobrutinib

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%**

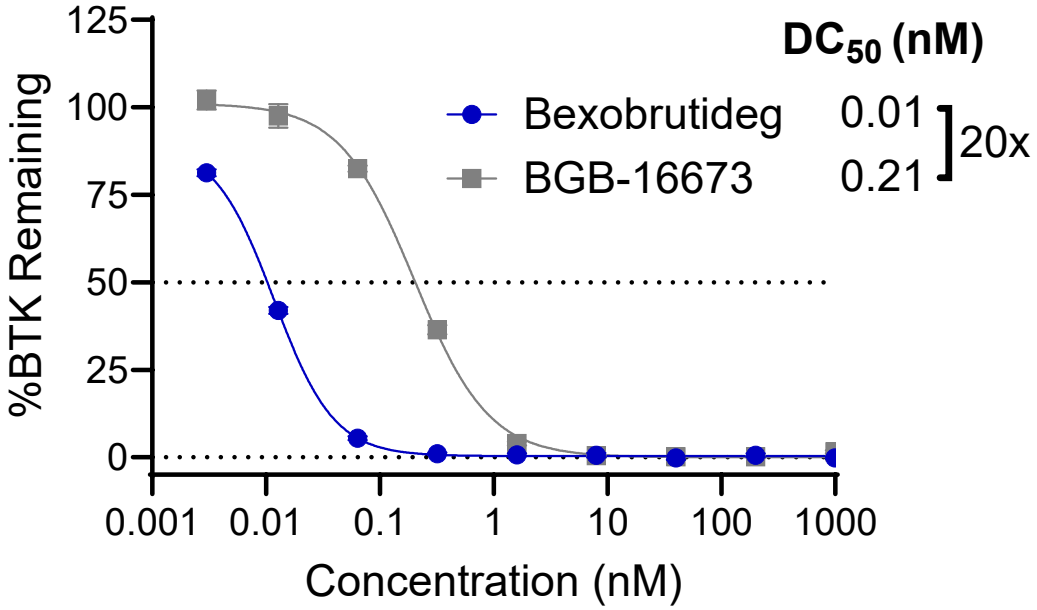
Source: NX-5948-301 study data cut off Sept 19, 2025, and Sharman et al, JCO 43: 2538-2549, June 2025

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

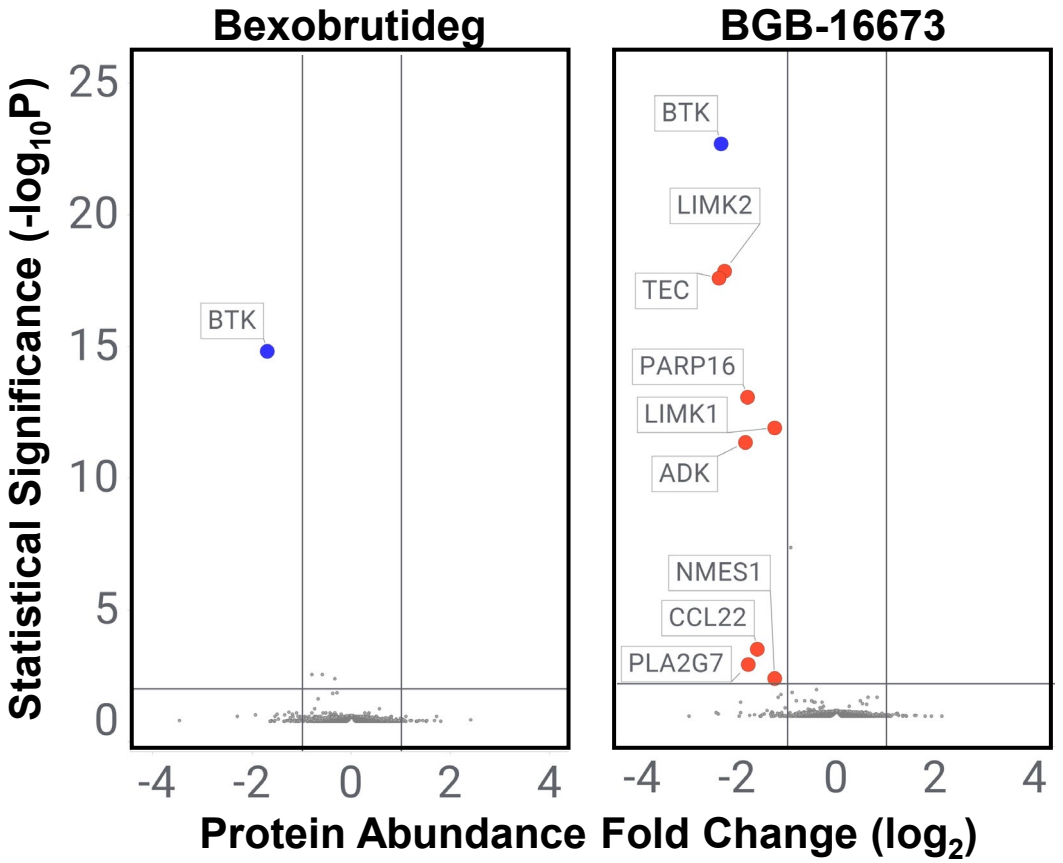
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Bexobrutideg Displays Best-in-Class Potency and Selectivity Relative to BeOne's BTK Degrader

BTK Degradation in Human B Cells



Selectivity of BTK Degraders by Global Proteomics



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

Degradation assessed at 24 hours by flow cytometry in human peripheral blood mononuclear cells, gated on CD20+ B cells

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.

Nurix's Leadership Extends Into I&I with Potential Best-in-Class Agents

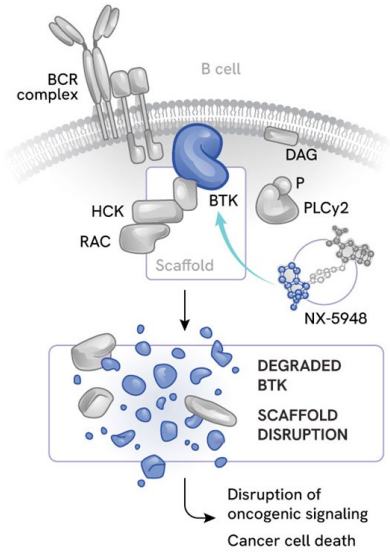


Three Blockbuster Targets Addressing Different Critical Pathways in Immunology and Neurology

Program	Target	Modality	Therapeutic area	Preclinical	Phase 1	Phase 2	Phase 3	Partner
Bexobrutideg (NX-5948)	BTK	Degrader	Chronic spontaneous urticaria			Planned		Roche
			Multiple sclerosis			Planned		
GS-6791/ NX-0479	IRAK4	Degrader	Rheumatoid arthritis and other inflammatory diseases					GILEAD
SAR448272/ NX-3911	STAT6	Degrader	Type 2 inflammatory diseases					sanofi

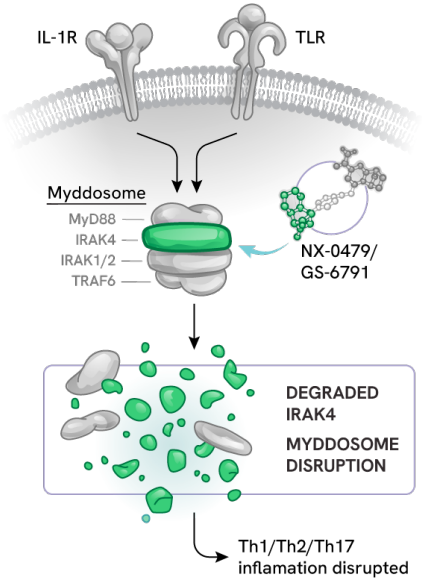
BTK

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



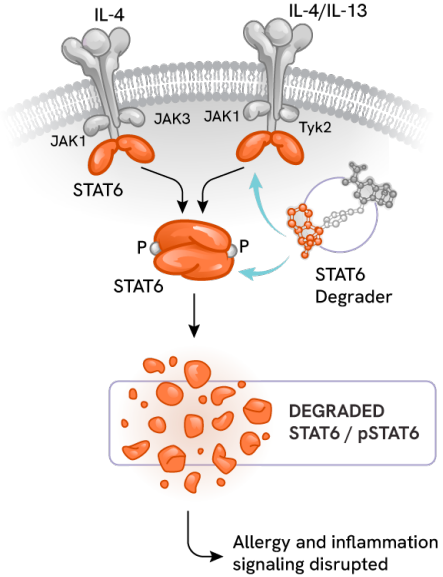
IRAK4

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



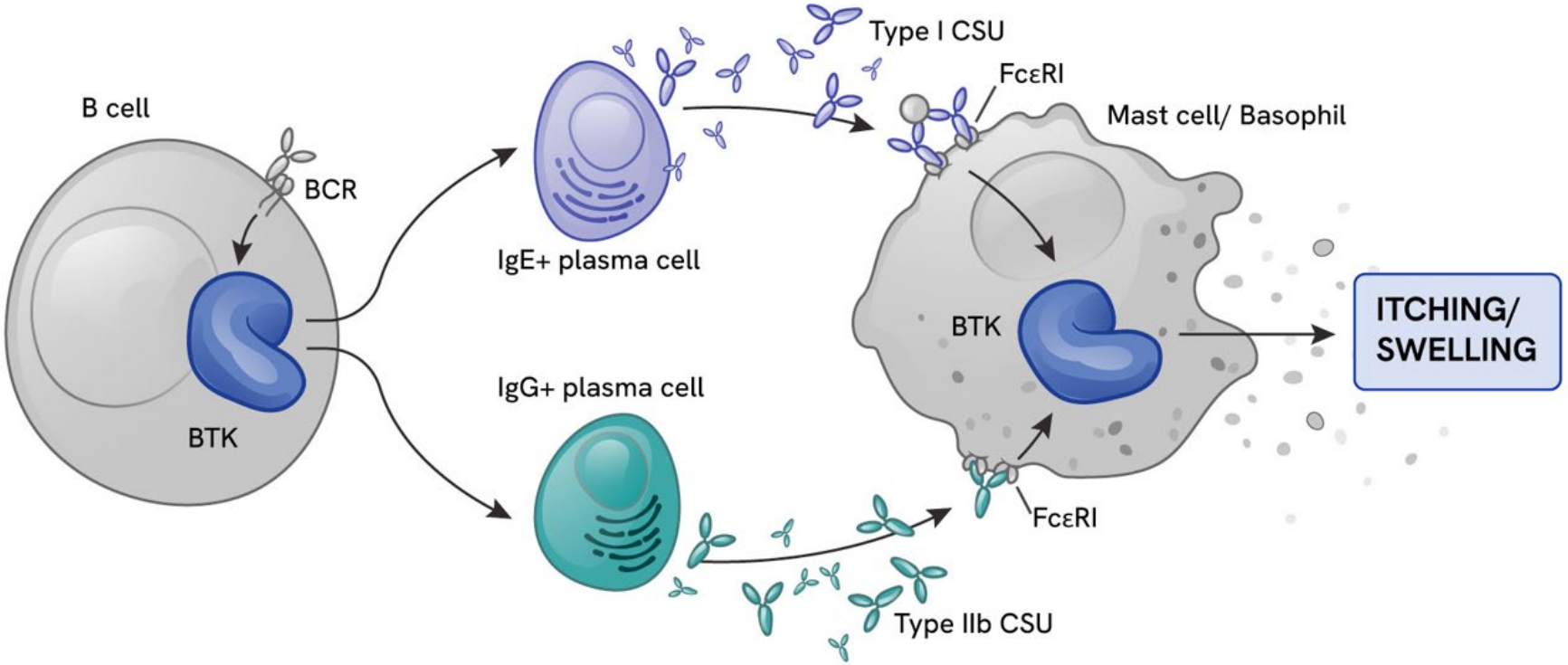
STAT6

A transcription factor that mediates type 2 inflammation



BTK Is a Clinically-Validated Target in Chronic Spontaneous Urticaria (CSU)

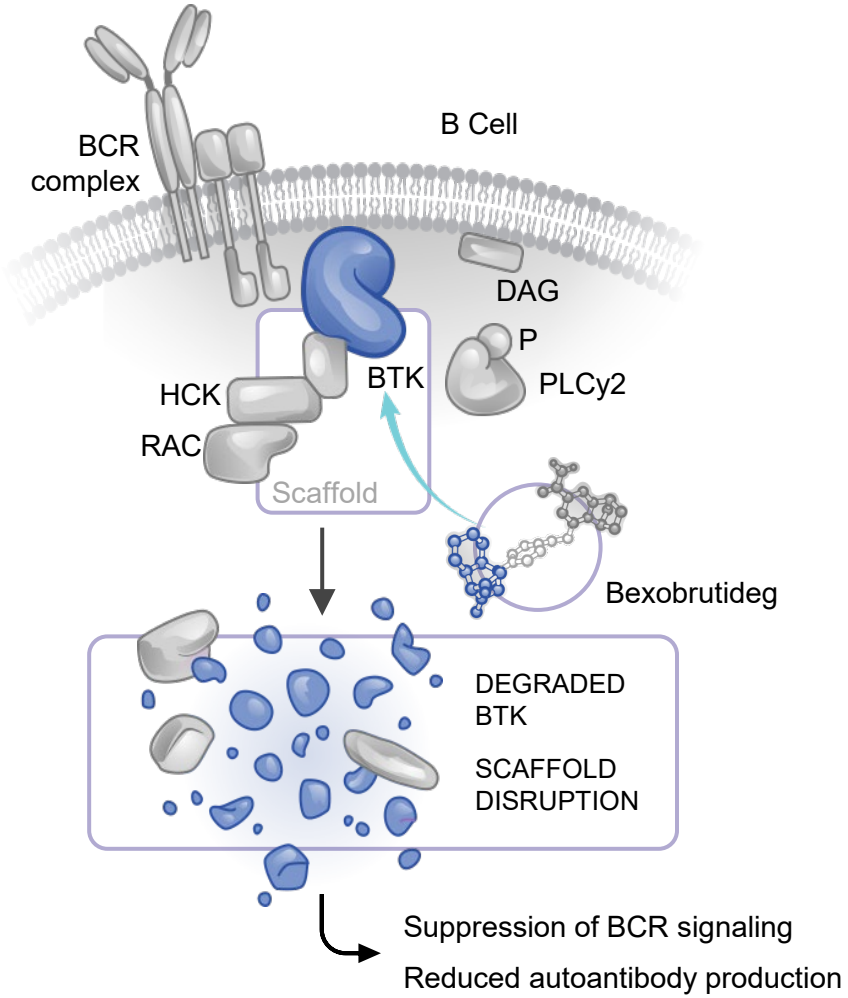
B cell activation and mast cell/basophil degranulation are BTK-dependent processes



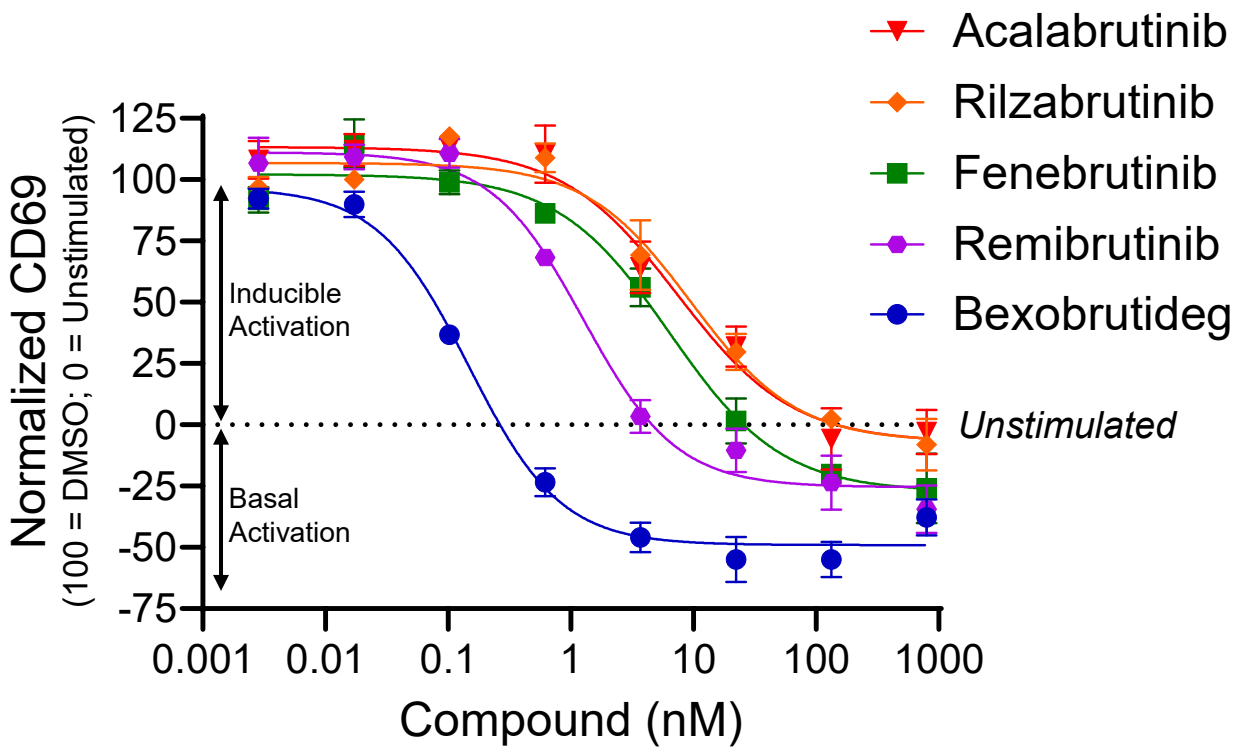
Targeted degradation of BTK better suppresses activation of pathways that contribute to the pathogenesis of CSU

Bexobrutideg Achieves Potent and Deep Suppression of B Cell Activation

Preclinical studies show a BTK scaffold function downstream of particulate antigen stimulation

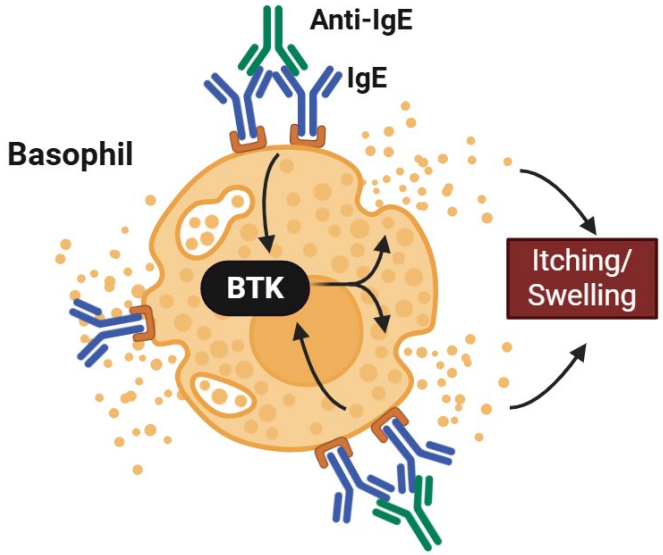


Suppression of B Cell Activation

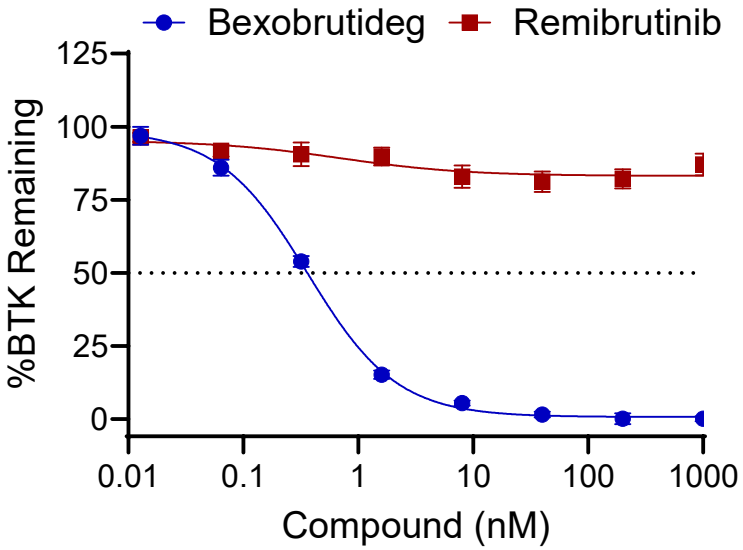


PBMCs were pre-treated with compound for 4 hours and stimulated overnight with bead-bound anti-IgM. CD69 expression on CD20+ B cells was quantified by flow cytometry and normalized; 100 = DMSO-treated cells, 0 = unstimulated cells.

Bexobrutideg Degrades BTK and Potently Suppresses FcεRI Signaling in Basophils



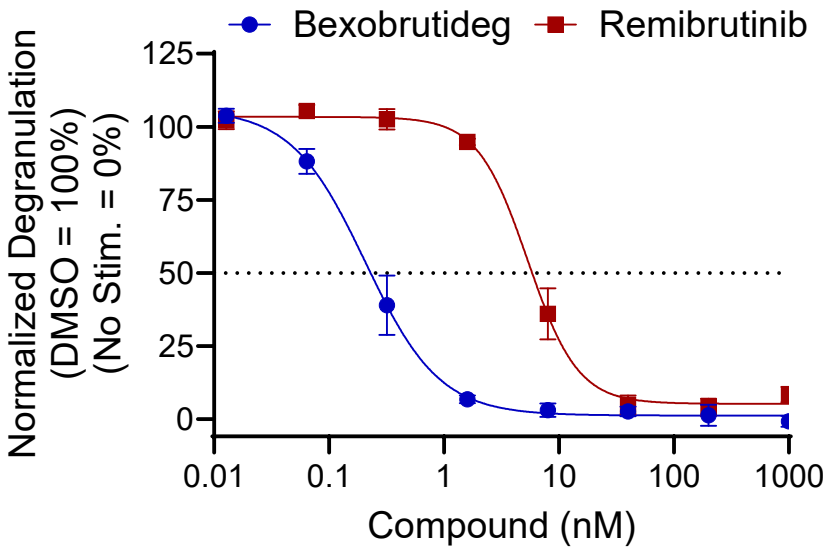
BTK Degradation in Basophils



	DC_{50} ± SEM (nM)
Bexobrutideg	0.18 ± 0.002
Remibrutinib	N/A

Assay performed in whole blood

Basophil Degranulation (Anti-IgE Stimulation)

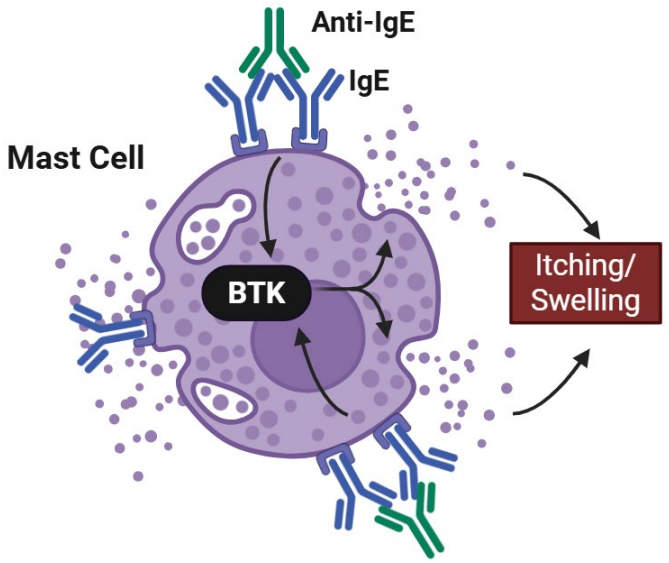


	IC_{50} ± SEM (nM)
Bexobrutideg	0.25 ± 0.07
Remibrutinib	6.4 ± 1

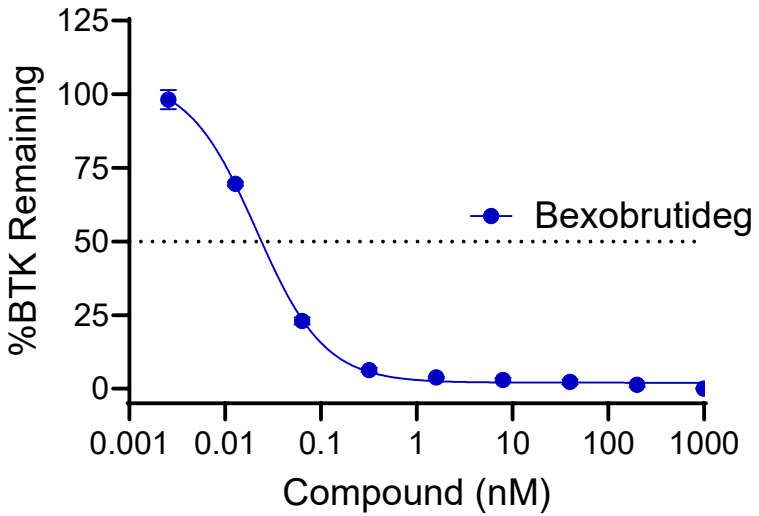
Assay performed in whole blood

Human whole blood from n = 4 donors was pre-treated with compound for 24 hours and stimulated with anti-IgE for 30 minutes. Degradation was quantified by intracellular flow cytometry, and degranulation was quantified by CD203c surface staining. Mean ± standard error (SEM) is displayed.
Graphic created with BioRender

Bexobrutideg Degrades BTK and Potently Suppresses FcεRI Signaling in Mast Cells



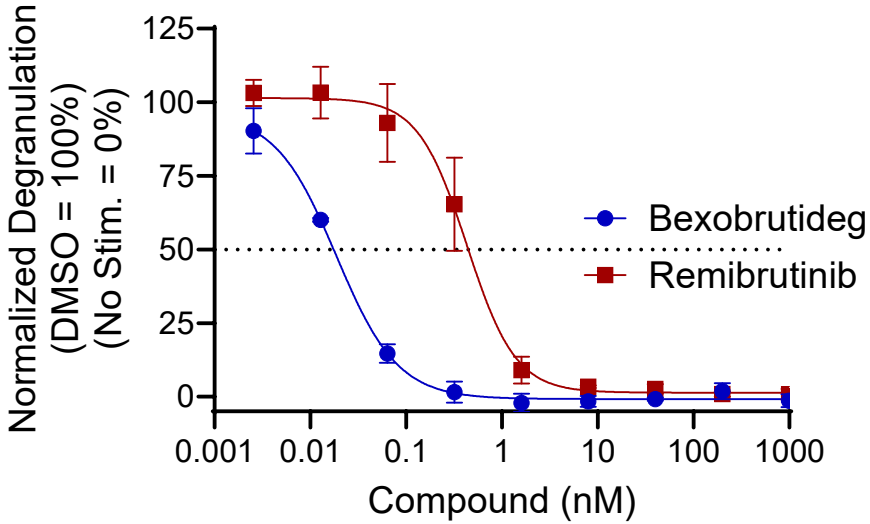
BTK Degradation in Mast Cells



	DC_{50} ± SEM (nM)
Bexobrutideg	0.02 ± 0.001

Assay performed in serum-free X-Vivo 15 media with IL-6 and SCF

Mast Cell Degranulation (Anti-IgE Stimulation)

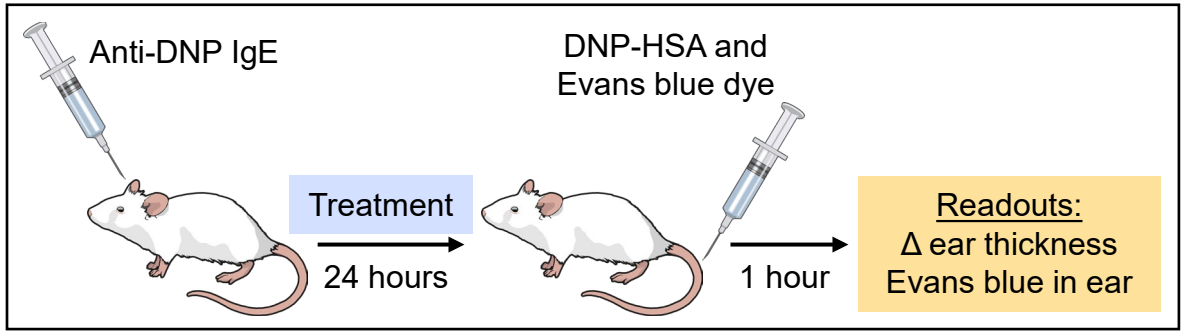


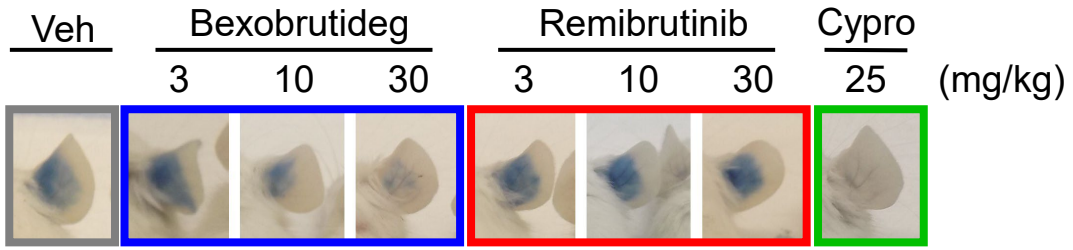
	IC_{50} ± SEM (nM)
Bexobrutideg	0.02 ± 0.0002
Remibrutinib	0.44 ± 0.2

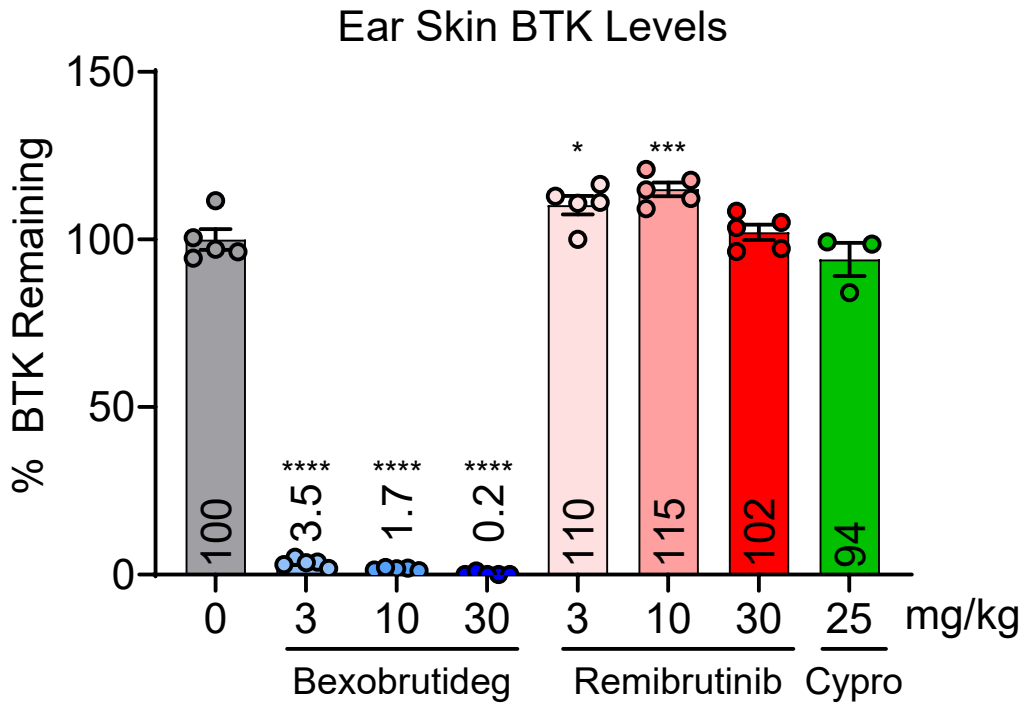
Assay performed in serum-free X-Vivo 15 media with IL-6 and SCF

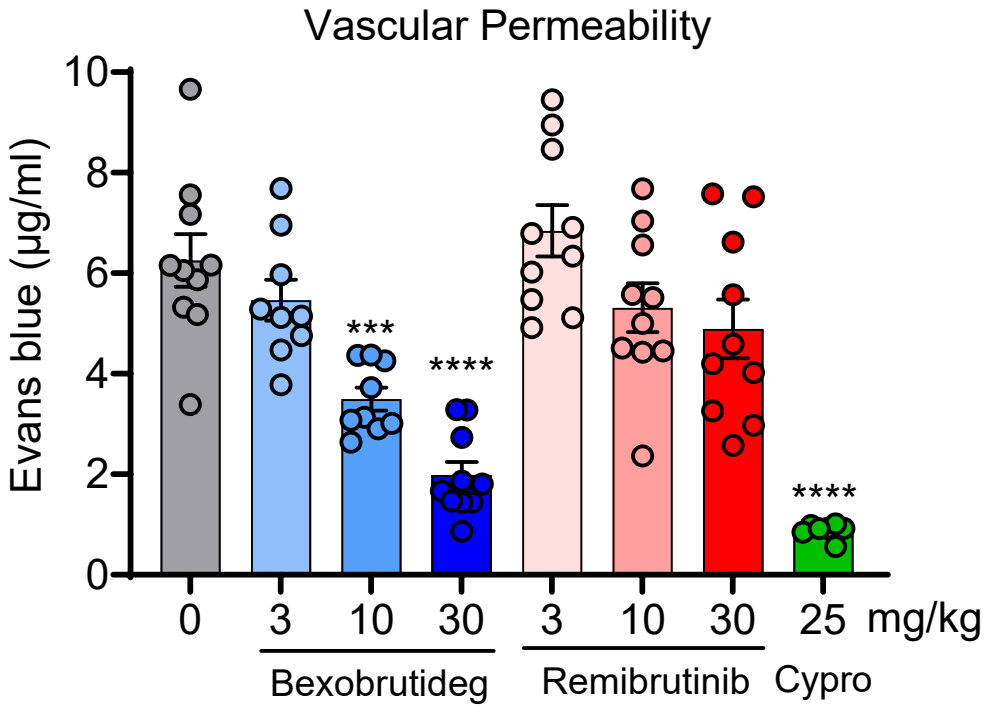
Mast cells differentiated from human CD34+ stem cells were pre-treated with compound and loaded with IgE for 24 hours, then stimulated with anti-IgE for 90 minutes. Degradation was quantified by intracellular flow cytometry, and degranulation was quantified by CD63 surface staining.
Graphic created with BioRender

Bexobrutideg Degrades BTK and Suppresses Vascular Permeability in Passive Cutaneous Anaphylaxis Model





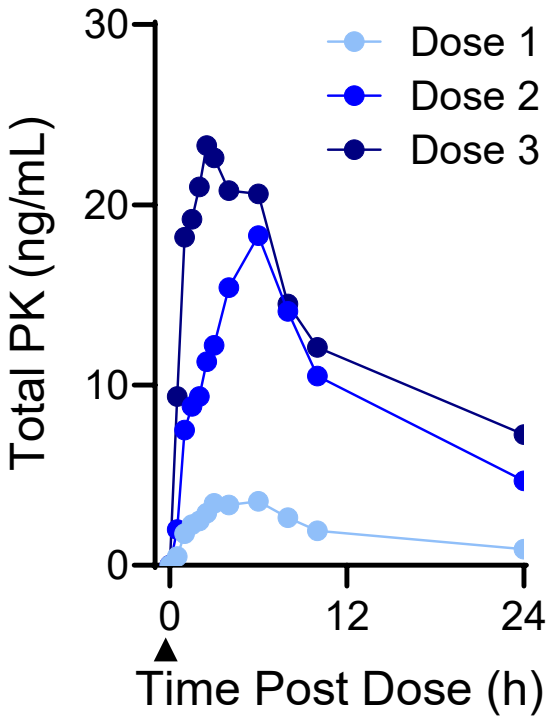




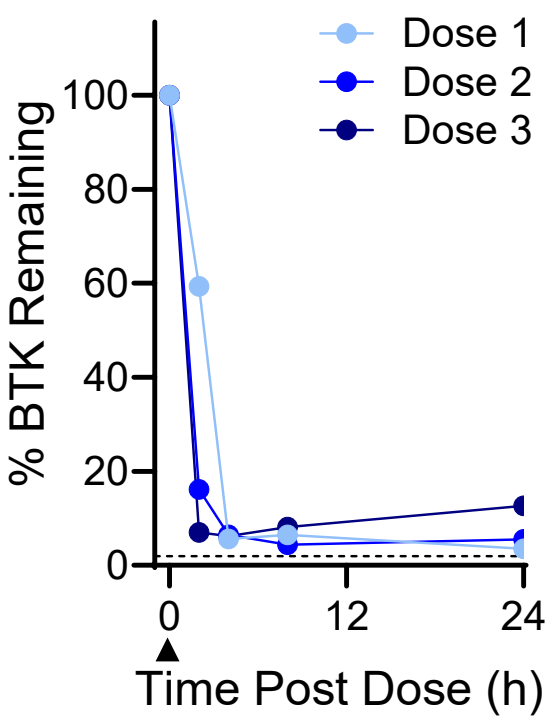
*Bexobrutideg was administered orally two times on a QD schedule, remibrutinib was administered orally three times on a BID schedule. Cyproheptadine was administered intraperitoneally two times on a QD schedule. Final dose was administered 4 hours prior to antigen challenge, and dye extravasation was assessed 1 hour after antigen challenge. One animal from bexobrutideg 3 mg/kg group excluded due to excess antigen + Evans blue dosage during IV challenge. Data are represented as mean ± SEM. *p < 0.05, **p < 0.01, ***p < 0.001, ****p < 0.0001. One-way ANOVA, Dunnett's multiple comparison test vs. vehicle.*

Bexobrutideg Demonstrates Deep BTK Degradation in Both Blood Cells and Skin of Healthy Volunteers

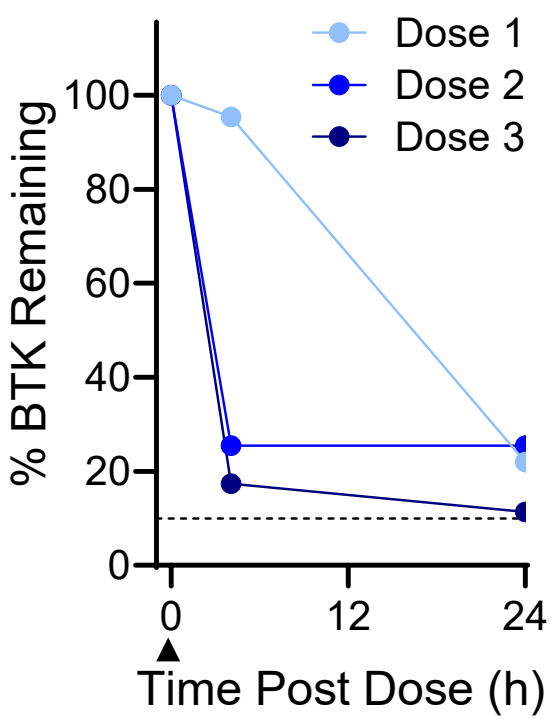
SAD Plasma PK



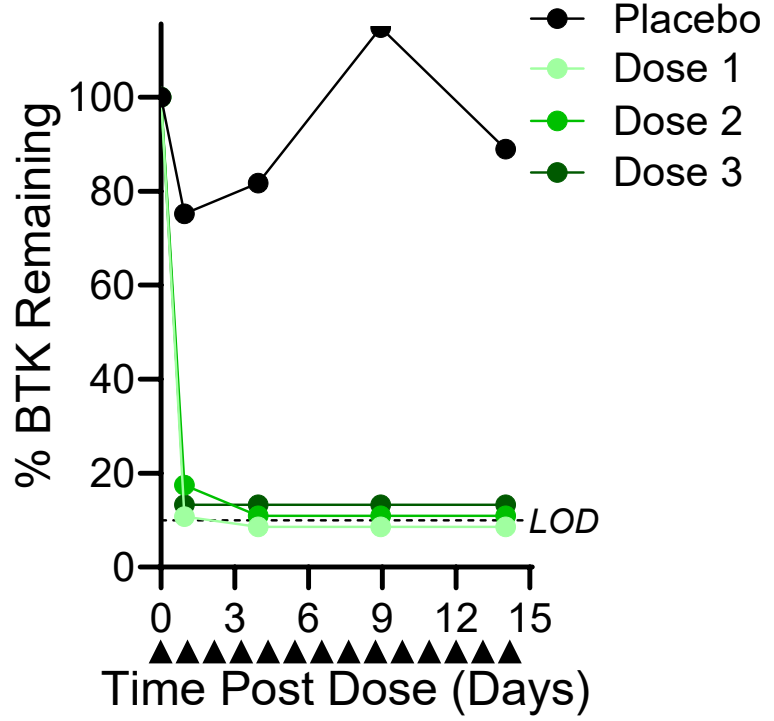
SAD BTK Degradation in PBMCs



SAD BTK Degradation in Skin



MAD BTK Degradation in Skin



Bexobrutideg was safe and well tolerated at all doses tested[a]

▲ Dose

Data as of 07 May 2026

SAD, single ascending dose; MAD, multiple ascending dose; LC-MS, liquid chromatography–mass spectrometry; PBMCs, peripheral blood mononuclear cells. BTK was quantified by LC-MS, DNA-normalized, and expressed as % remaining relative to average baseline. Dashed lines indicate the approximate effective limit of detection after DNA normalization. Values below the assay lower limit were imputed as one-half the lower limit. Arrows indicate dose administration. [a]*Data not shown* (NCT06717269)

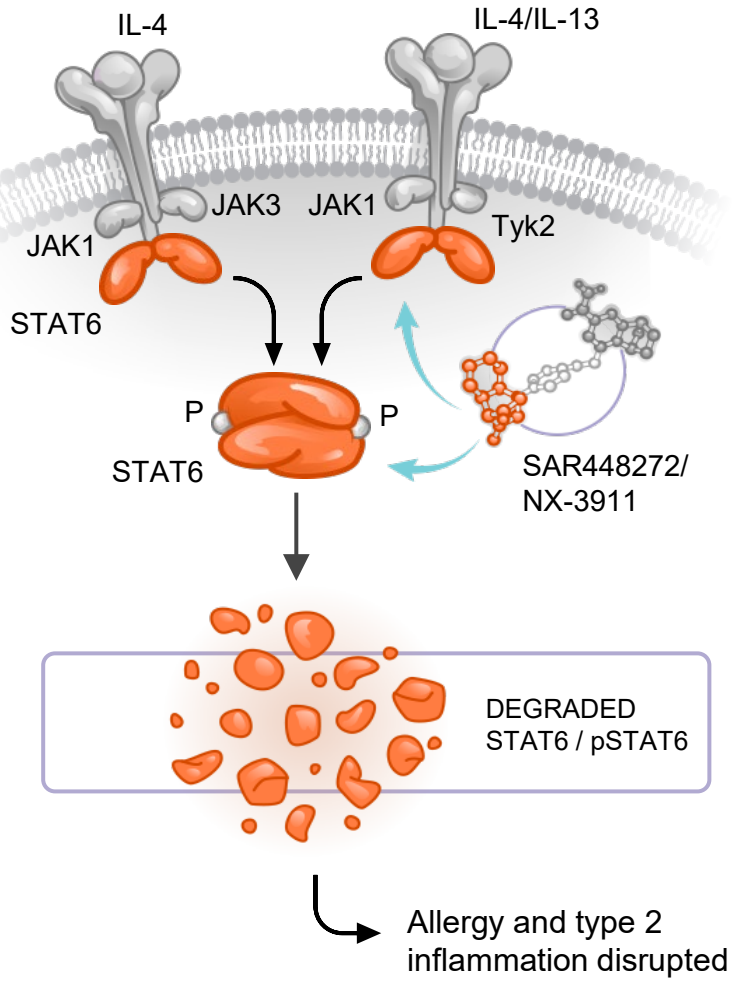
SAR448272/NX-3911: A Potential Best-in-Class STAT6 Degrader in Collaboration with Sanofi that Achieves Complete STAT6 Pathway Blockade

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

SAR448272 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

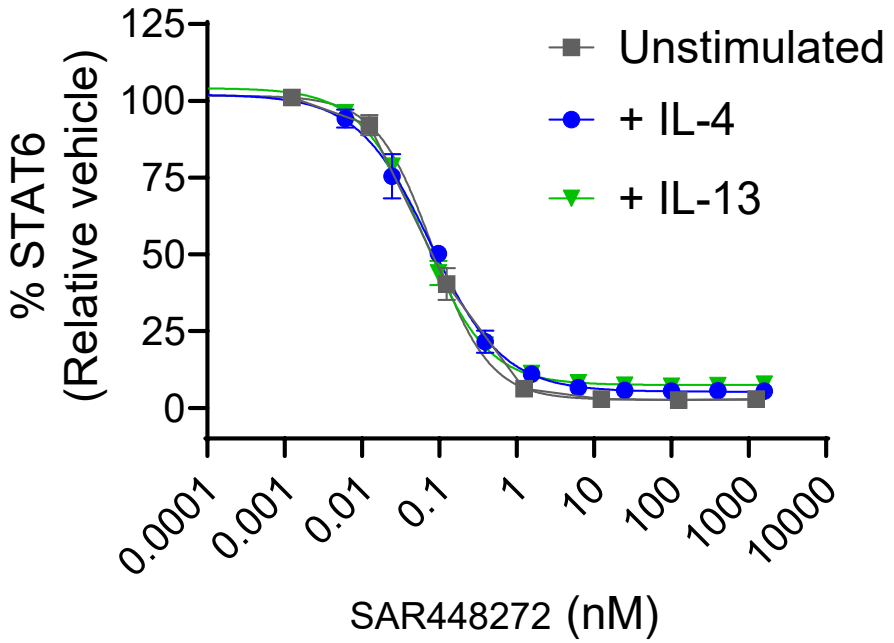
***JAK**, Janus kinase; **STAT**, Signal Transducer and Activator of Transcription*

SAR448272 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
- SAR448272 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*

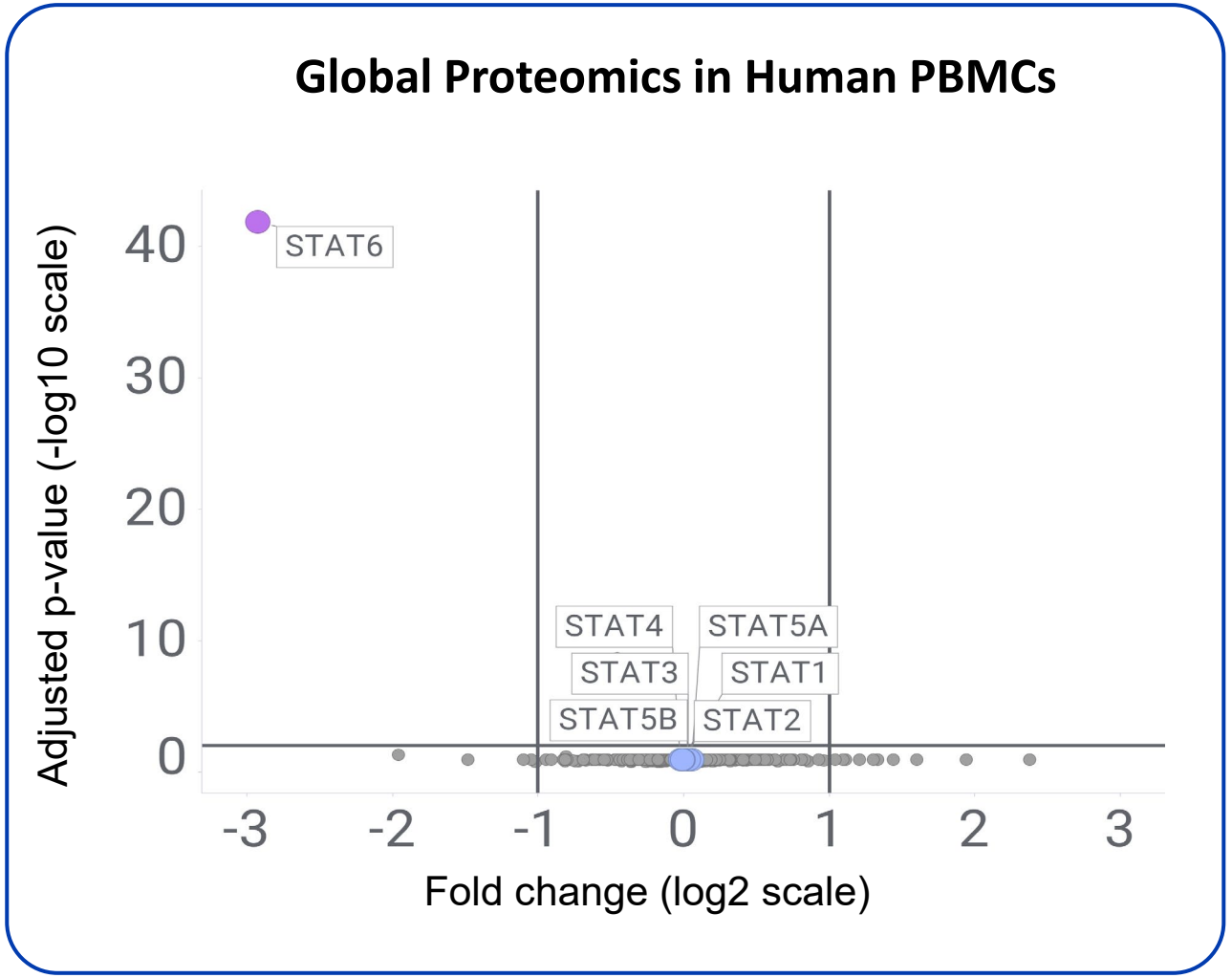
SAR448272 Degrades STAT6 With Exquisite Selectivity

- 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

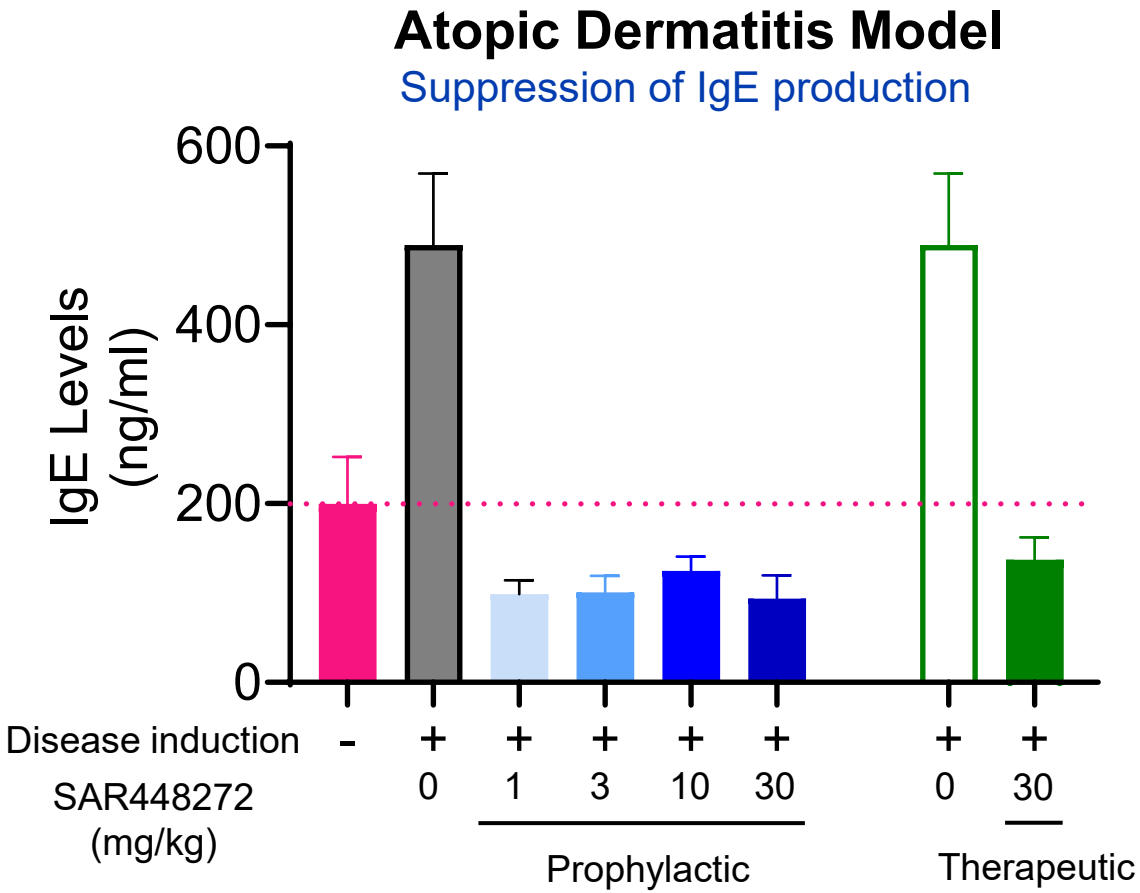
  (9500 total proteins measured)

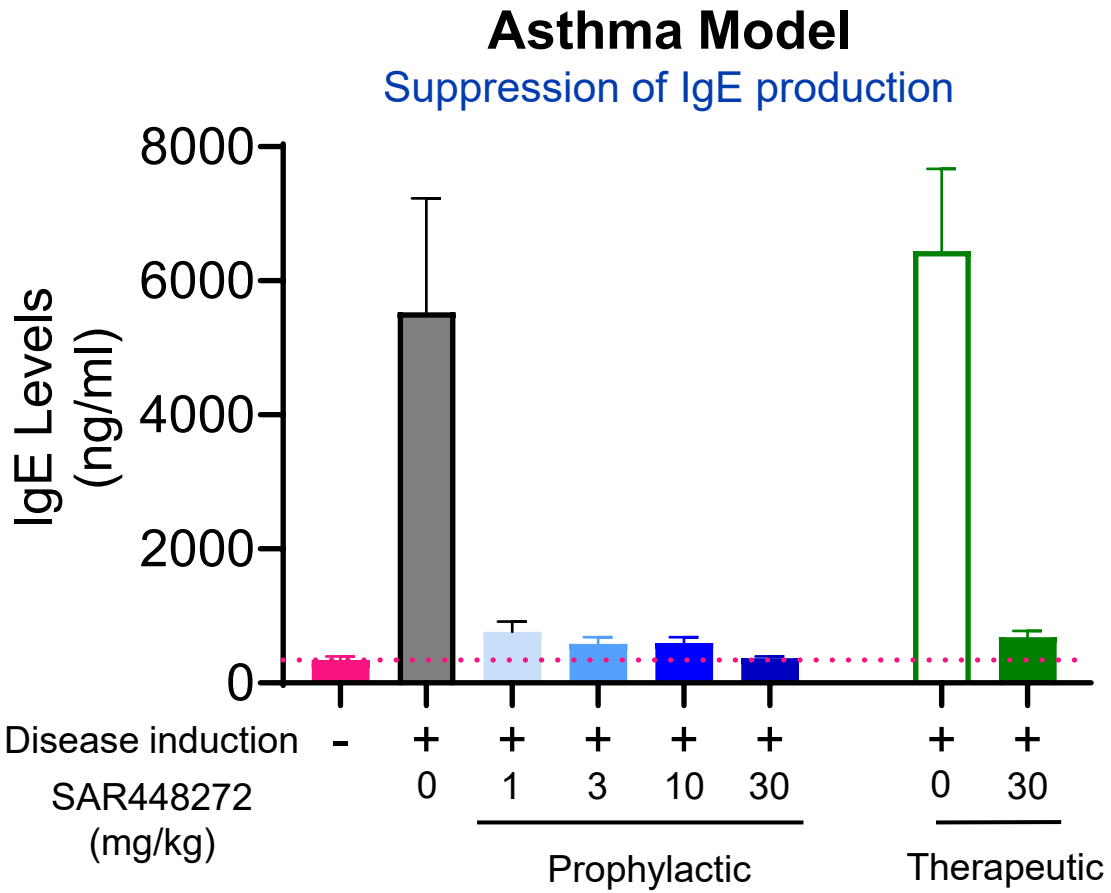


***PBMC**, peripheral blood mononuclear cell*

SAR448272 Has Demonstrated Activity in Inflammatory Disease Models of Atopic Dermatitis and Asthma

Achieves control of inflammatory activity in both prophylactic and therapeutic settings





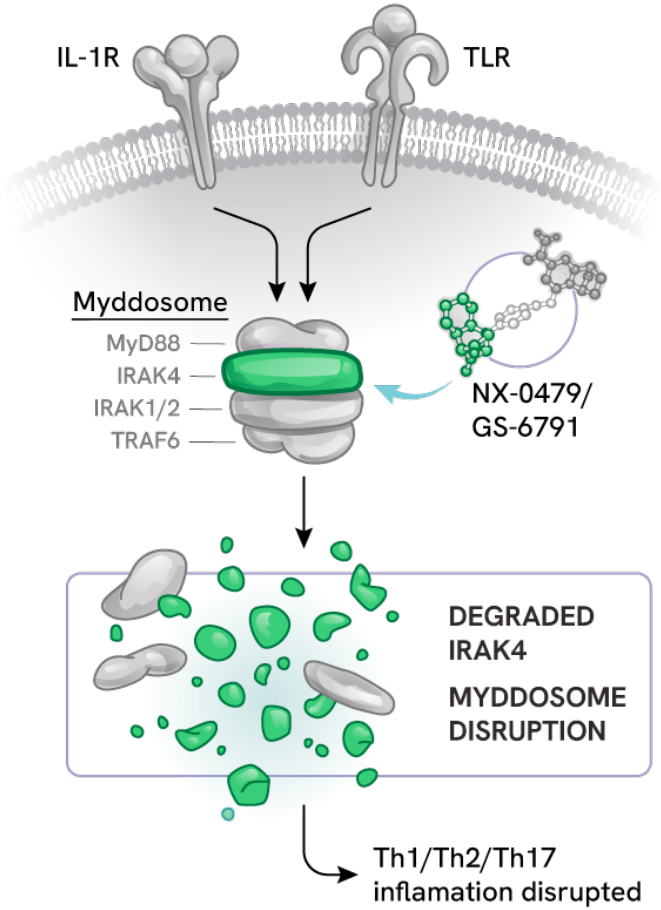
Disease induced by MC903 in Atopic Dermatitis and House Dust Mite in Asthma models in hIL-4/IL-4Rα/hIL-13 mice (C57BL/6 background)
Red dotted line represents the average IgE levels observed in naïve mice.
All treated groups are statistically significant ($P<0.05$) compared to vehicle control (0 mg/kg)

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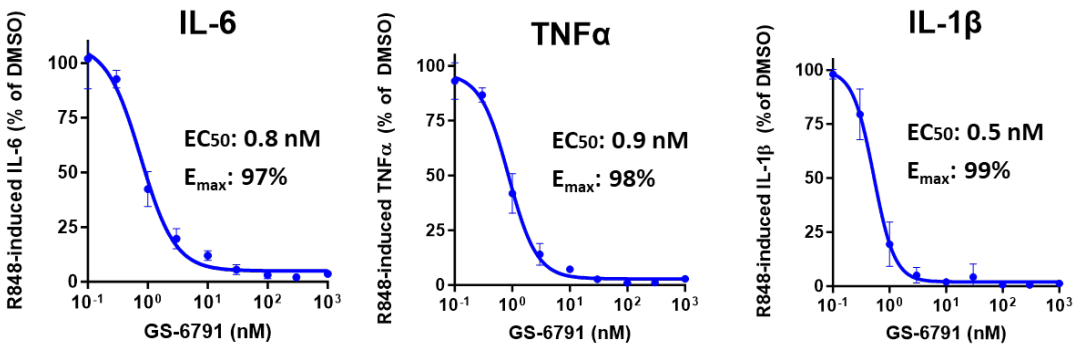
IRAK4 Degrader NX-0479/GS-6791: Potential Treatment for Rheumatoid Arthritis and Other Inflammatory Diseases

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

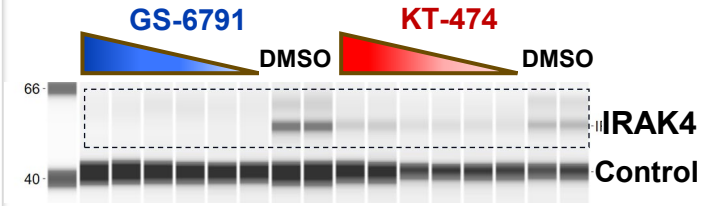


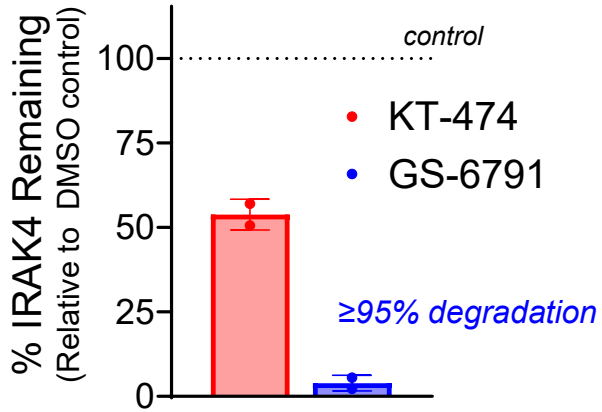
Source: Teng et al., ACR 2024, November 2024 .

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

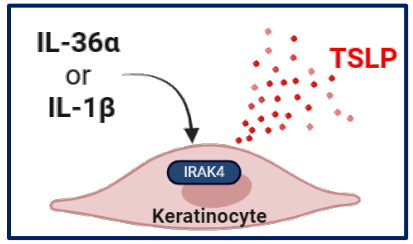
GS-6791 Elicits Best-in-Class IRAK4 Degradation and Inhibition of Functional Responses in Skin Epithelial Systems

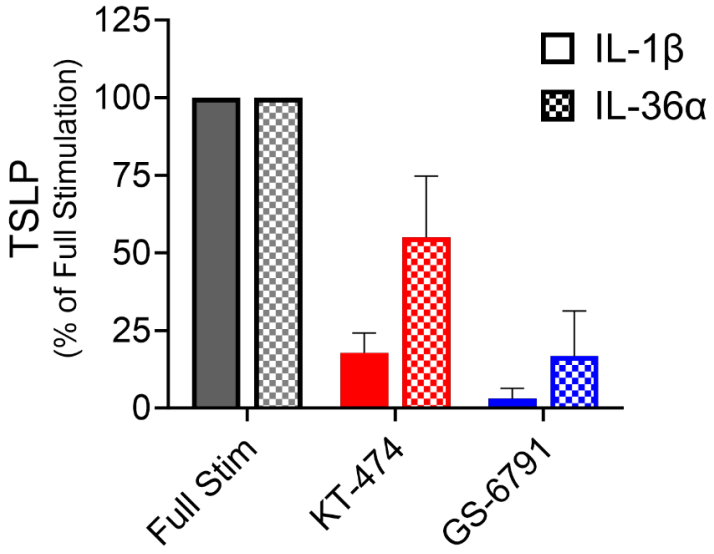
Deeper IRAK4 degradation in basal human keratinocytes at 24 h



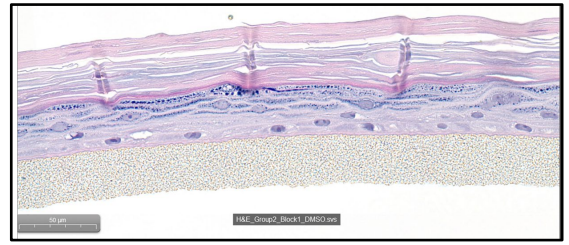


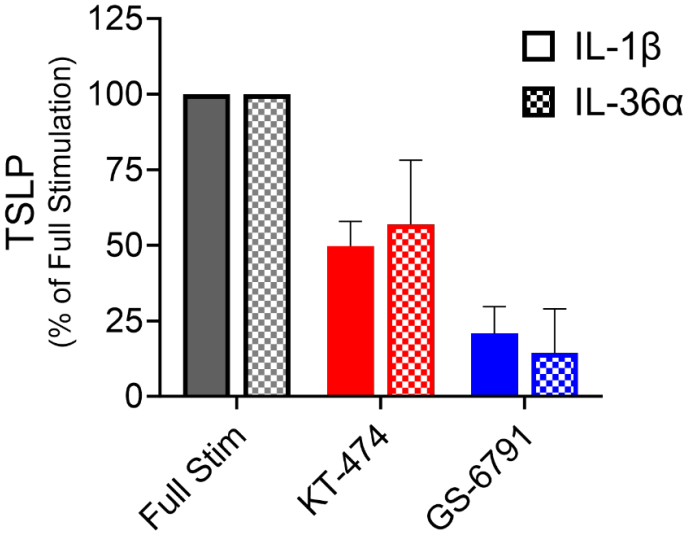
Superior inhibition in differentiated human keratinocytes





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

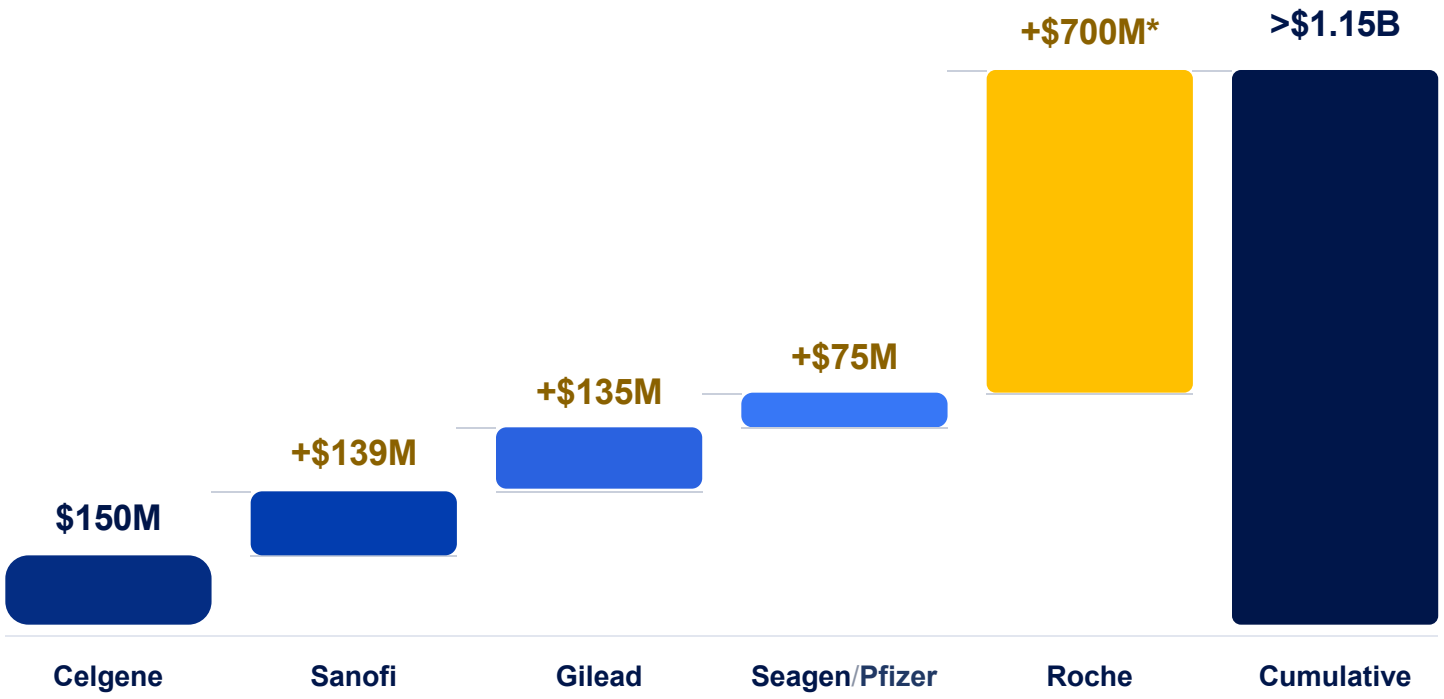
NURIX

Strong Financial Position to Support Execution Across the Nurix Pipeline



Strategic Collaborations Have Generated More Than $1 Billion in Non-Dilutive Capital

CUMULATIVE PARTNERSHIP CAPITAL RECEIVED



PRO FORMA CASH & SECURITIES

~$1.14B*

CUMULATIVE CAPITAL RECEIVED AND EXPECTED

>$1.15B

Non-dilutive, across five partnerships

REMAINING MILESTONE POTENTIAL

Up to $7.7B

Plus U.S. profit share & tiered ex-U.S. royalties

Pro forma cash and marketable securities of approximately **$1.14 billion***, with cumulative non-dilutive partnership capital exceeding **$1.15 billion**, and billions in milestone potential still ahead across the platform.

*Cash, cash equivalents and marketable securities of $443.5 million as of May 31, 2026, and pro-forma cash of approximately $1.14 billion, including the expected $700 million upfront payment from Roche

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Thank You

NURIX