NURIX™

Medicines to Outmatch Disease

Investor Presentation
January 13, 2025

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The estimated cash and investments amount included in this presentation is a preliminary, unaudited estimate based upon information available to Nurix's management as of the date of this presentation. It is subject to the completion of financial closing procedures, including the completion of audit procedures by Nurix's independent public accounting firm, and therefore is subject to adjustment. The amount does not present all information necessary for a complete understanding of Nurix’s financial condition as of or for the year ended November 30, 2024. Nurix’s audited results as of and for the year ended November 30, 2024, will be included in Nurix’s Annual Report on Form 10-K for the year ended November 30, 2024.

Another Great Year at Nurix

Key accomplishments in 2024

NX-5948 BTK Degrader

Demonstrated clear clinical proof of concept for BTK degradation with NX-5948

- Completed Phase 1a dose escalation
- Demonstrated robust efficacy in CLL and WM with favorable safety profile
- Oral presentations at ASH and EHA

Established unmet medical need with key regulatory agencies

- Fast Track Designation for CLL and WM from the U.S. Food and Drug Administration (FDA)
- PRIME designation from the European Medicines Agency (EMA)

Pipeline

Advanced two other wholly owned assets in the clinic

- NX-2127, a dual degrader of BTK and IKZF1/3
- NX-1607, an inhibitor of CBL-B

Progressed three major partnerships with Sanofi, Gilead, and Pfizer

- Extension of the STAT6 degrader program with Sanofi
- Presented first preclinical data for IRAK4 degrader clinical candidate and advanced toward IND submission
- Achieved potent cell-based activity of degrader antibody conjugates

Ended fiscal year in a strong cash position with $609.6M*

BTK, Bruton's tyrosine kinase; **CLL** chronic lymphocytic leukemia, **CBL-B**, casitas B lymphoma-b; **WM**, Waldenstrom's macroglobulinemia

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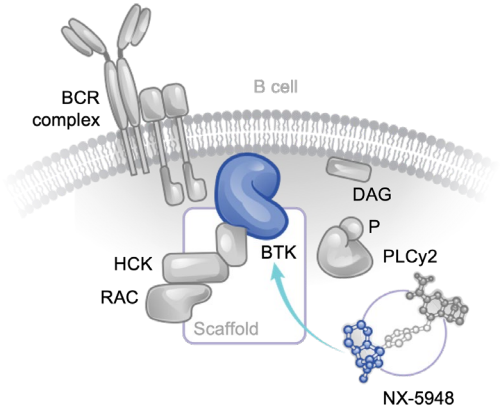
Drug Discovery Pipeline Strategy

Meeting the needs of patients with breakthrough therapies

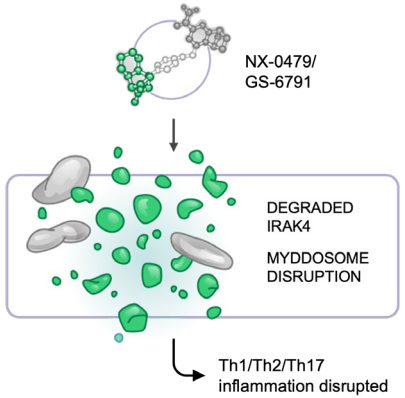
Clinically validated targets where inhibitors fail to address resistance and scaffolding



Kinase targets in cancer

BTK – B-cell malignancies and I&I

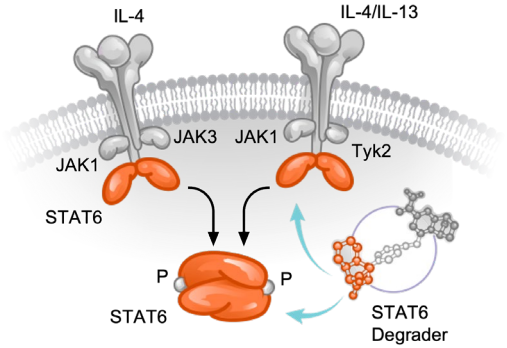
Unmet medical need where inhibitors are not sufficient to drive efficacy



Signaling proteins with scaffolding function

IRAK4 – rheumatoid arthritis

"Undruggable" targets



Transcriptions factors; fusion proteins; E3 ligases

STAT6 – T2 inflammatory diseases

***BTK**, Bruton's tyrosine kinase; **I&I**, inflammation and immunology; **IRAK4**, interleukin-1 receptor-associated kinase 4; **STAT6**, signal transducer and activator of transcription 6*

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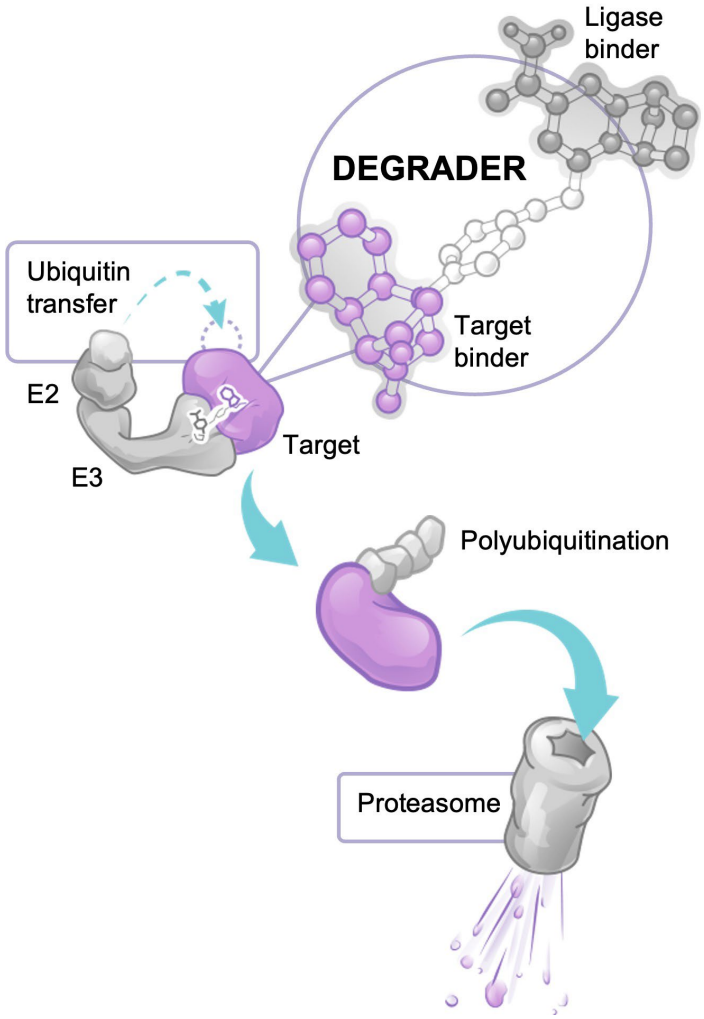
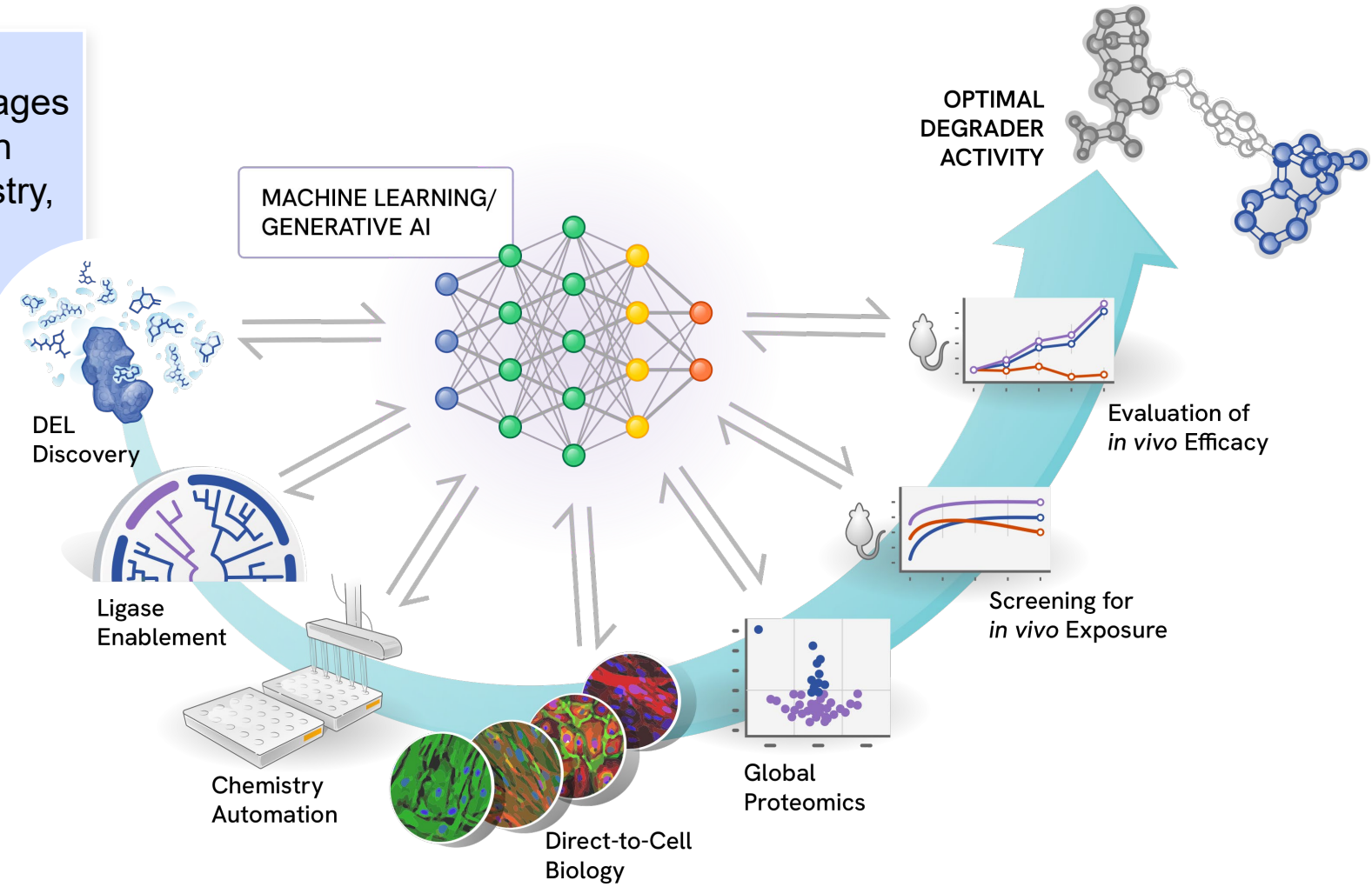
Establish Degrader-Based Medicines at the Forefront of Patient Care

- Degraders act as molecular matchmakers, bringing together two key players:
  - An E3 ligase (a key part of a cell's protein degradation machinery)
  - A disease-causing target protein
- This process, called induced proximity, enables the E3 ligase to tag the target protein with ubiquitin to mark it for disposal by the proteasome – the cell's protein recycling center
- Given their ability to eliminate target proteins, degraders can achieve effects similar to genetic therapies that silence disease-causing genes



Industry Leading DEL-AI Discovery Engine for TPD and DAC Drug Discovery

Our DEL-AI discovery engine leverages the combined power of our data-rich DEL capabilities, automated chemistry, and advanced machine learning to accelerate drug discovery



TPD, targeted protein degradation; **DAC**, degrader antibody conjugate; **DEL**, DNA encoded library

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
NX-5948	BTK	Degrader	B-cell malignancies				
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
Undisclosed	Undisclosed	Degrader	Undisclosed				sanofi
Multiple	Undisclosed	DAC	Undisclosed				Pfizer

Inflammation & Immunology

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

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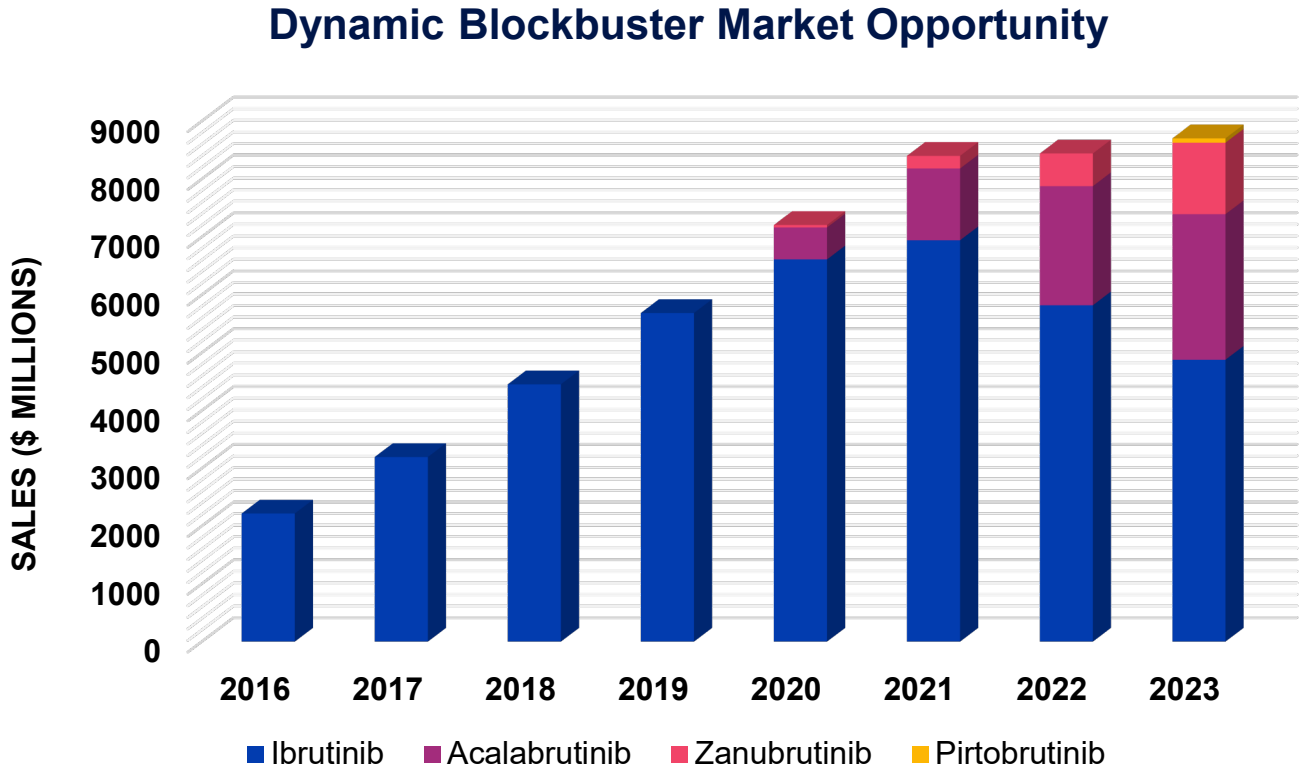
BTK Targeted Agents Are a Major Value Driver Across Biotech and Pharma

Current market for BTK inhibitors annualizing at over $10 billion

- Next generation BTK inhibitors are currently taking market share from Imbruvica
- All BTK inhibitors share resistance mutation vulnerabilities

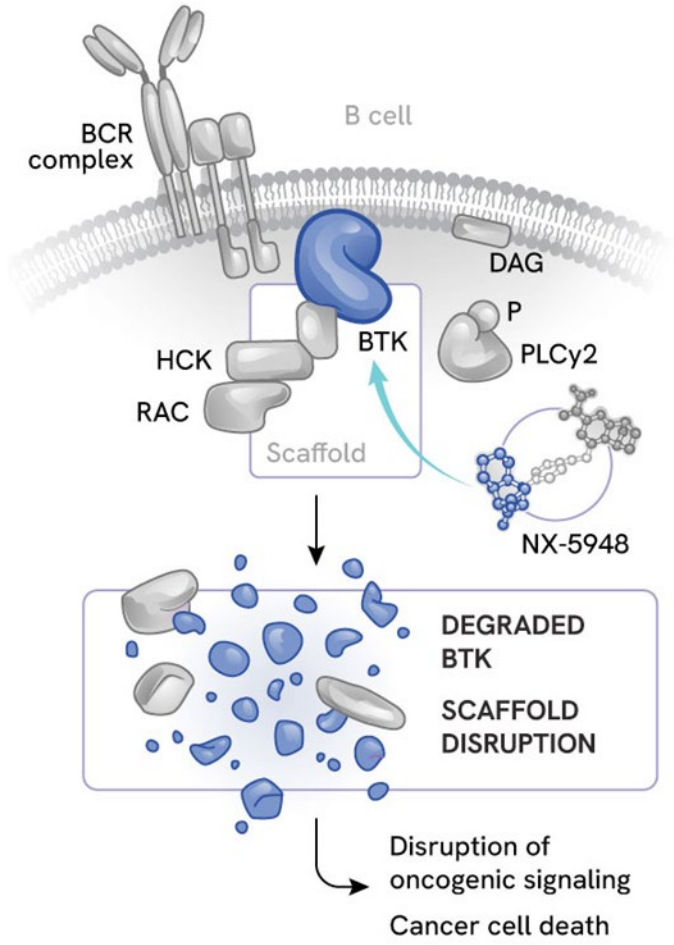
Nurix's Strategy

- Displace inhibitors in key oncology markets
- Expand market for degraders into I&I



Note: $10 billion annualized estimate based on reported Q3 2024 sales
Source: Earnings reports from Johnson & Johnson, Abbvie, AstraZeneca, Beigene, and Eli Lilly

Why Do We Need BTK Degraders?



- **BTK degraders** can overcome treatment-emergent resistance mutations
- **BTK degraders** address BTK scaffolding function
- **BTK degraders** have demonstrated clinical activity in difficult to treat B-cell malignancies
- **BTK degraders** have the potential to address significant unmet needs in autoimmune and inflammatory disorders

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Phase 1a/b Trial in Adults with Relapsed/Refractory B-cell Malignancies

NX-5948-301 Trial Design

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

NHL/WM

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

Ongoing CLL Phase 1b expansion cohorts

Randomized

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

Fast Track Designation

Ongoing NHL/WM Phase 1b expansion cohorts

- **MZL** Marginal zone lymphoma
- **WM** Waldenstrom's macroglobulinemia
- **FL** Follicular lymphoma

CLL, chronic lymphocytic leukemia; SLL, small lymphocytic lymphoma; NHL, non-Hodgkin's lymphoma; WM, Waldenström macroglobulinemia; BTKi, BTK inhibitor; BCL2i, Bcl-2 inhibitor; QD, once daily

Heavily Pre-Treated Patients With a High Prevalence of Baseline Mutations

Baseline disease characteristics

Characteristics	Patients with CLL/SLL[a] (n=60)
ECOG PS, n (%)	
0	24 (40.0)
1	36 (60.0)
CNS involvement, n (%)	5 (8.3)
Median prior lines of therapy (range)	4.0 (1–12)
Previous treatments[b], n (%)	
BTKi	59 (98.3)
cBTKi	59 (98.3)
ncBTKi[c]	17 (28.3)
BCL2i	50 (83.3)
BTKi and BCL2i	49 (81.7)
CAR-T therapy	3 (5.0)
Bispecific antibody	4 (6.7)
PI3Ki	18 (30.0)
Chemo/chemo-immunotherapies (CIT)	43 (71.7)
Mutation status[d] (n=57), n (%)	
TP53	23 (40.4)
BTK	22 (38.6)
PLCG2	7 (12.3)
BCL2	6 (10.5)

[a]*Baseline disease characteristics in CLL cohort were comparable to those in the overall population;* [b]*Patients could have received multiple prior treatments;* [c]*All patients who received ncBTKi have also previously received cBTKi;* [d]*Mutations presented here were centrally sequenced.*
BCL2, B-cell lymphoma 2; BCL2i, BCL2 inhibitor; BTK, Bruton's tyrosine kinase; BTKi, BTK inhibitor; cBTKi, covalent BTKi; CAR-T, chimeric antigen receptor T-cell; CLL, chronic lymphocytic leukemia; CNS, central nervous system; ECOG PS, Eastern Cooperative Oncology Group (ECOG) performance status; ncBTKi, non-covalent BTKi; PI3Ki, phosphoinositide 3-kinase inhibitor; PLCG2, phospholipase C gamma 2; SLL, small lymphocytic lymphoma

Data cutoff: 10 Oct 2024

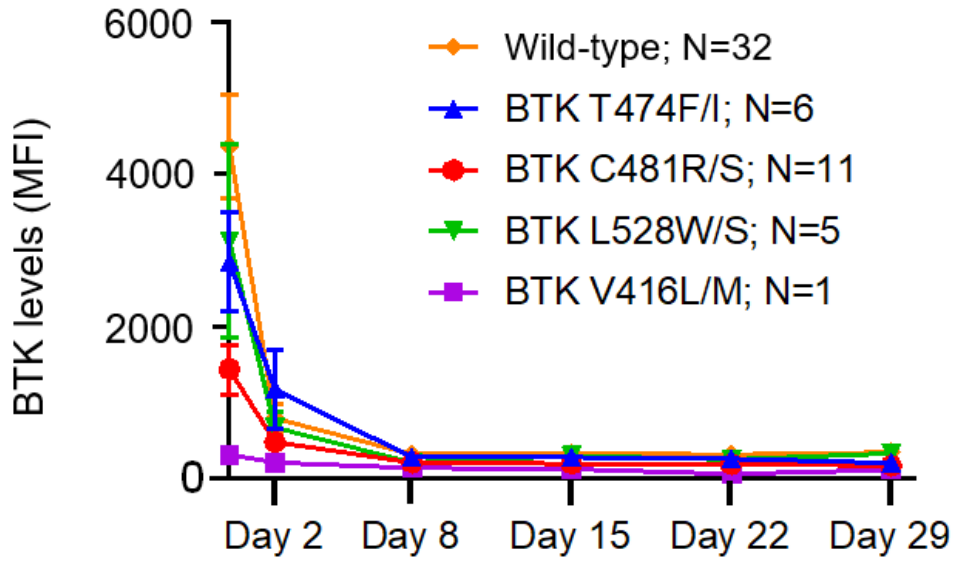
NX-5948 Degrades Wild-Type and Mutated BTK

NX-5948 degrades gatekeeper, kinase-proficient and kinase-dead BTK mutations

	Patients with CLL/SLL (n=57)[c]
Baseline mutation status, n (%)	
BTK mutations[1,a,b]	22 (38.6)
C481S	12 (21.1)
C481R	2 (3.5)
L528W	4 (7.0)
L528S	1 (1.8)
T474I	5 (8.8)
T474F	1 (1.8)
V416M	1 (1.8)
V416L	1 (1.8)
G541V	1 (1.8)

[a]Patients could have multiple prior treatments and BTK mutations; BTK mutations were tested at baseline by next-generation sequencing centrally; ≥5% allelic frequency is reported
[b]Patients can have more than one resistance mutation
[c]Patients with available mutation status

BTK degradation



Note: Some patients have multiple BTK mutations

Reference
1. Montoya et al. Science 2024;383

***BTK**, Bruton's tyrosine kinase; **CLL**, chronic lymphocytic leukemia; **MFI**, mean fluorescence intensity; **SLL**, small lymphocytic lymphoma*

Data cutoff: 10 Oct 2024

Response Rate Deepens with Longer Time on Treatment

NX-5948 overall response assessment

CLL response-evaluable patients	Primary ORR analysis[b] ≥1 response assessment(s) at 8 weeks	Exploratory ORR analysis[b] ≥2 response assessments at 16 weeks
	n=49[c]	n=38[c]
Objective response rate (ORR),[a] % (95% CI)	75.5 (61.1–86.7)	84.2 (68.7–94.0)
Best response, n (%)		
CR	0 (0.0)	0 (0.0)
PR	36 (73.5)	32 (84.2)
PR-L	1 (2.0)	0 (0.0)
SD	10 (20.4)	4 (10.5)
PD	2 (4.1)	2 (5.3)

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

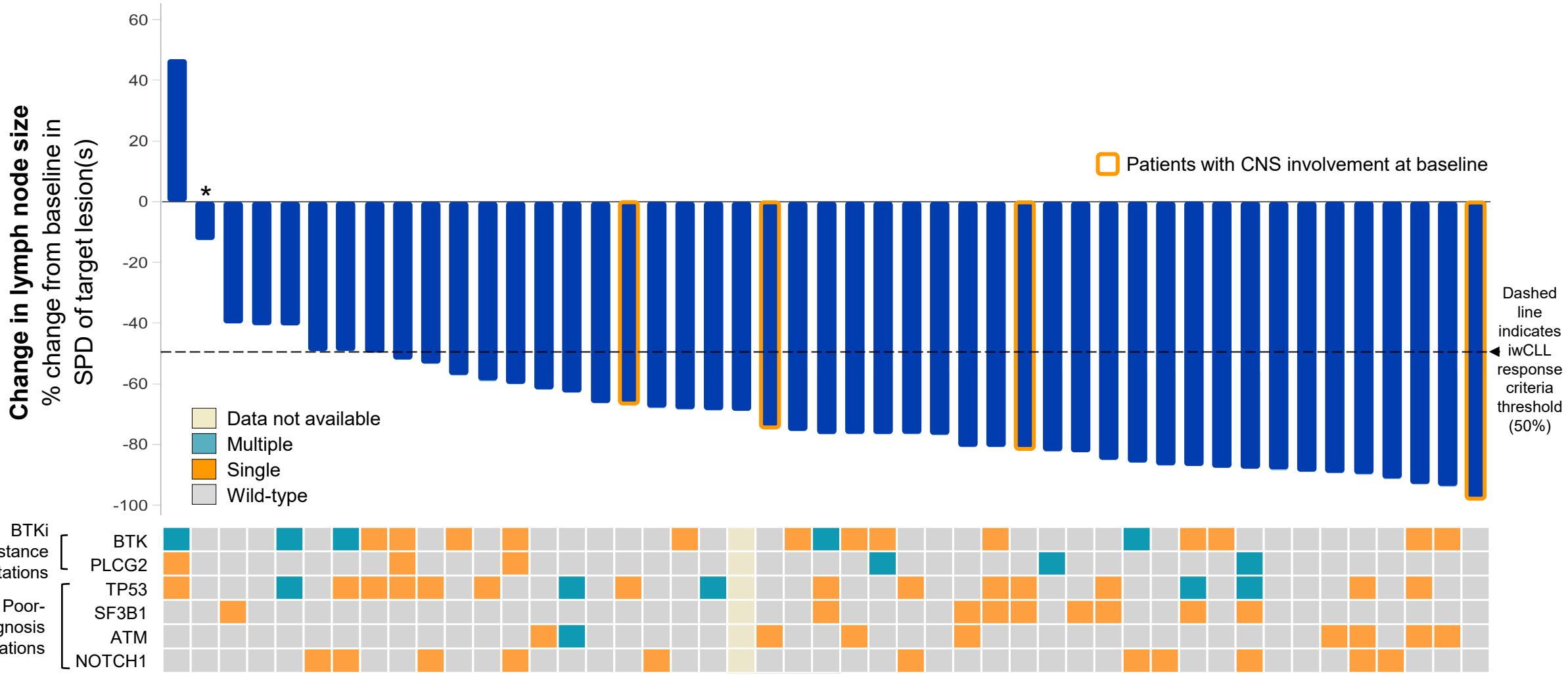
[b]Patients who progressed prior to their first response assessment and patients who discontinued for any reason after their first response assessment are included in the denominators

[c]Patients without identified target lesion(s) at baseline are evaluated as disease-evaluable per iwCLL criteria, while they may not be represented in waterfall plot

Data cutoff: 10 Oct 2024

__CLL__, chronic lymphocytic leukemia; __CR__, complete response; __iwCLL__, International Workshop on CLL; __ORR__, objective response rate; __PD__, progressive disease; __PR__, partial response; __PR-L__, partial response with rebound lymphocytosis; __SD__, stable disease

Clinical Activity in CLL Patients Including Those with Baseline Mutations and CNS Involvement



*Patient with Richter's transformation to Hodgkin's on biopsy

Note: patients without identified target lesion(s) at baseline are evaluated as disease-evaluable per iwCLL criteria, while they may not be represented in waterfall plot

Data cutoff: 10 Oct 2024

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

NX-5948 Safety Profile

TEAEs in ≥10% of overall population or Grade ≥3 TEAEs or SAEs in >1 patient

TEAEs, n (%)	Patients with CLL/SLL (n=60)			Overall population (N=125)		
	Any grade	Grade ≥3	SAEs	Any grade	Grade ≥3	SAEs
Purpura/contusion[a]	22 (36.7)	–	–	42 (33.6)	–	–
Fatigue[b]	16 (26.7)	–	–	29 (23.2)	2 (1.6)	–
Petechiae	16 (26.7)	–	–	28 (22.4)	–	–
Thrombocytopenia[c]	10 (16.7)	1 (1.7)	–	26 (20.8)	7 (5.6)	–
Rash[d]	14 (23.3)	1 (1.7)	1 (1.7)	24 (19.2)	2 (1.6)	1 (0.8)
Neutropenia[e]	14 (23.3)	11 (18.3)	–	23 (18.4)	18 (14.4)	–
Anemia	11 (18.3)	4 (6.7)	–	21 (16.8)	10 (8.0)	–
Headache	10 (16.7)	–	–	21 (16.8)	1 (0.8)	1 (0.8)
COVID-19[f]	10 (16.7)	–	–	19 (15.2)	2 (1.6)	2 (1.6)
Diarrhea	12 (20.0)	1 (1.7)	–	18 (14.4)	1 (0.8)	–
Cough	9 (15.0)	–	–	16 (12.8)	1 (0.8)	–
Pneumonia[g]	4 (6.7)	2 (3.3)	2 (3.3)	10 (8.0)	6 (4.8)	6 (4.8)
Lower respiratory tract infection	3 (5.0)	1 (1.7)	1 (1.7)	9 (7.2)	3 (2.4)	2 (1.6)
Fall	1 (1.7)	1 (1.7)	1 (1.7)	8 (6.4)	2 (1.6)	2 (1.6)
Hypertension	2 (3.3)	1 (1.7)	–	7 (5.6)	5 (4.0)	–
Hyponatremia	–	–	–	3 (2.4)	2 (1.6)	–
Pulmonary embolism	1 (1.7)	1 (1.7)	1 (1.7)	2 (1.6)	2 (1.6)	2 (1.6)
Subdural hematoma	1 (1.7)	–	1 (1.7)	2 (1.6)	1 (0.8)	2 (1.6)

- Tolerable safety profile consistent with prior disclosures
- 1 case of Grade 1 AFib in a CLL patient with pre-existing AFib
- 6 TEAEs resulted in drug discontinuation (1 CLL; 5 NHL)
- 2 Grade 5 AEs (1 pulmonary embolism; 1 case pending) deemed not related to NX-5948

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

***AE,** adverse event; **AFib**, atrial fibrillation; **CLL**, chronic lymphocytic leukemia; **NHL**, non-Hodgkin's lymphoma; **SAE**, serious adverse event; **SLL**, small lymphocytic lymphoma; **TEAE**, treatment emergent AE*

Data cutoff: 10 Oct 2024

NX-5948 High Preliminary Overall Response in Patients with Waldenström Macroglobulinemia

Preliminary efficacy presented at the International Workshop on Waldenström Macroglobulinemia

WM response-evaluable patients	**Primary ORR analysis[b]** ≥1 response assessment(s) at 8 weeks
	n=9
Objective response rate (ORR),[a] %	77.8
Best response, n (%)	
CR	0 (0.0)
PR / MR	7 (77.8)
SD	2 (22.2)
PD	0 (0.0)

[a]Objective response rate includes CR + PR + MR

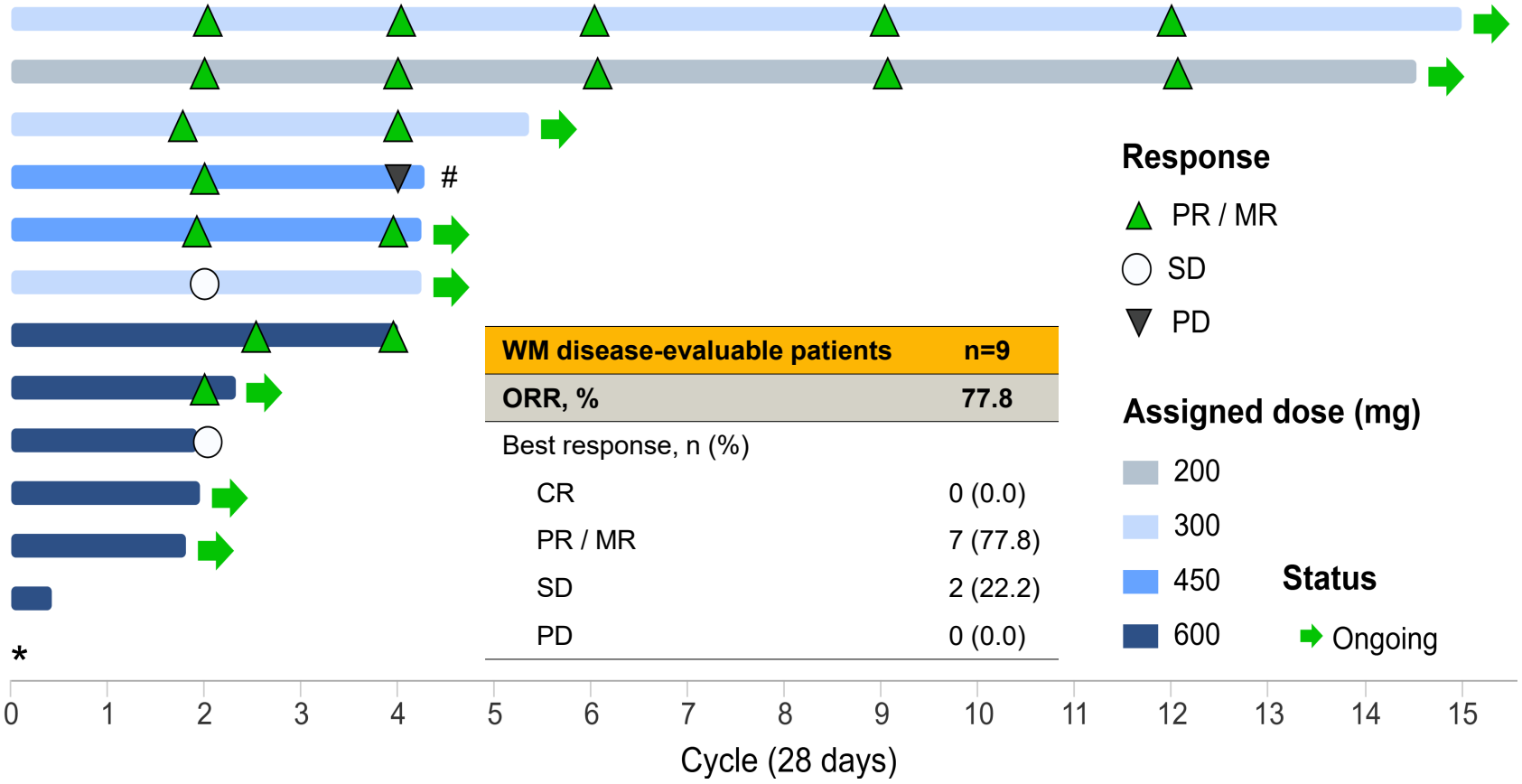
[b]Patients who progressed prior to their first response assessment and patients who discontinued for any reason after their first response assessment are included in the denominators

Data cutoff: 10 Oct 2024

*CR, complete response; **MR minor** response; **ORR**, objective response rate; **PD**, progressive disease; **PR**, partial response; **PR-L**, partial response with rebound lymphocytosis; **SD**, stable disease; **WM**, Waldenström macroglobulinemia*

Rapid and Durable Responses to NX-5948 in Patients with Waldenström Macroglobulinemia

Select prior therapies			Select mutations	
BTKi	BCL2i	Pirto	CXCR4	MYD88



WM disease-evaluable patients	n=9
ORR, %	77.8
Best response, n (%)	
CR	0 (0.0)
PR / MR	7 (77.8)
SD	2 (22.2)
PD	0 (0.0)

*Ineligible, identified post 1st dose
#Transformed to DLBCL

Data cutoff: 10 Oct 2024

***BCL2i**, B-cell leukemia/lymphoma 2 inhibitor; **BTKi**, Bruton's tyrosine kinase inhibitor; **CR**, complete response; **DLBCL**, diffuse large B-cell lymphoma; **MR**, minor response; **PD**, progressive disease; **Pirto**, pirtobrutinib; **PR**, partial response; **SD**, stable disease*

NX-5948 Regulatory Milestones

Advancing NX-5948 program globally in CLL and WM

CLL

- U.S. Fast Track Designation from the FDA in January 2024
- Type B End of Phase 1 meeting held with the FDA, key takeaways:
  - Reviewed dose levels of 200 mg QD and 600 mg QD in the context of Project Optimus
  - Feedback on principles of pivotal trial designs including Fast Track population and considerations for randomized controlled trials
  - Nurix plans future interactions in 2025 as sufficient data is accumulated
- EU expansion of enrollment into France, Poland, Italy and Spain approved in Q3 2024
- EU PRIME designation from EMA in November 2024

WM

- U.S. Fast Track Designation from the FDA in December 2024

***CLL**, chronic lymphocytic leukemia; **WM**, Waldenström macroglobulinemia*

Nurix Is Accelerating Development of NX-5948 in CLL with First Pivotal Study To Be Initiated in 2025

Current status in CLL

- Clear demonstration of clinical activity in difficult to treat CLL population
- Phase 1b cohorts enrolling rapidly with post-BTKi/post-BCL2i CLL patients randomized between 200mg QD and 600mg QD
- Planning for a broad Phase 3 program across lines of therapy as monotherapy and in combination with other approved agents

Outline of potential pivotal plans in CLL*

Potential path for accelerated approval

1. Single-arm monotherapy trial in post-BTKi/post-BCL2i patients (Fast Track population)

Confirmatory study in 2L+

2. Randomized head-to-head trial vs. comparator(s)* in the post-BTKi, 2L+ population

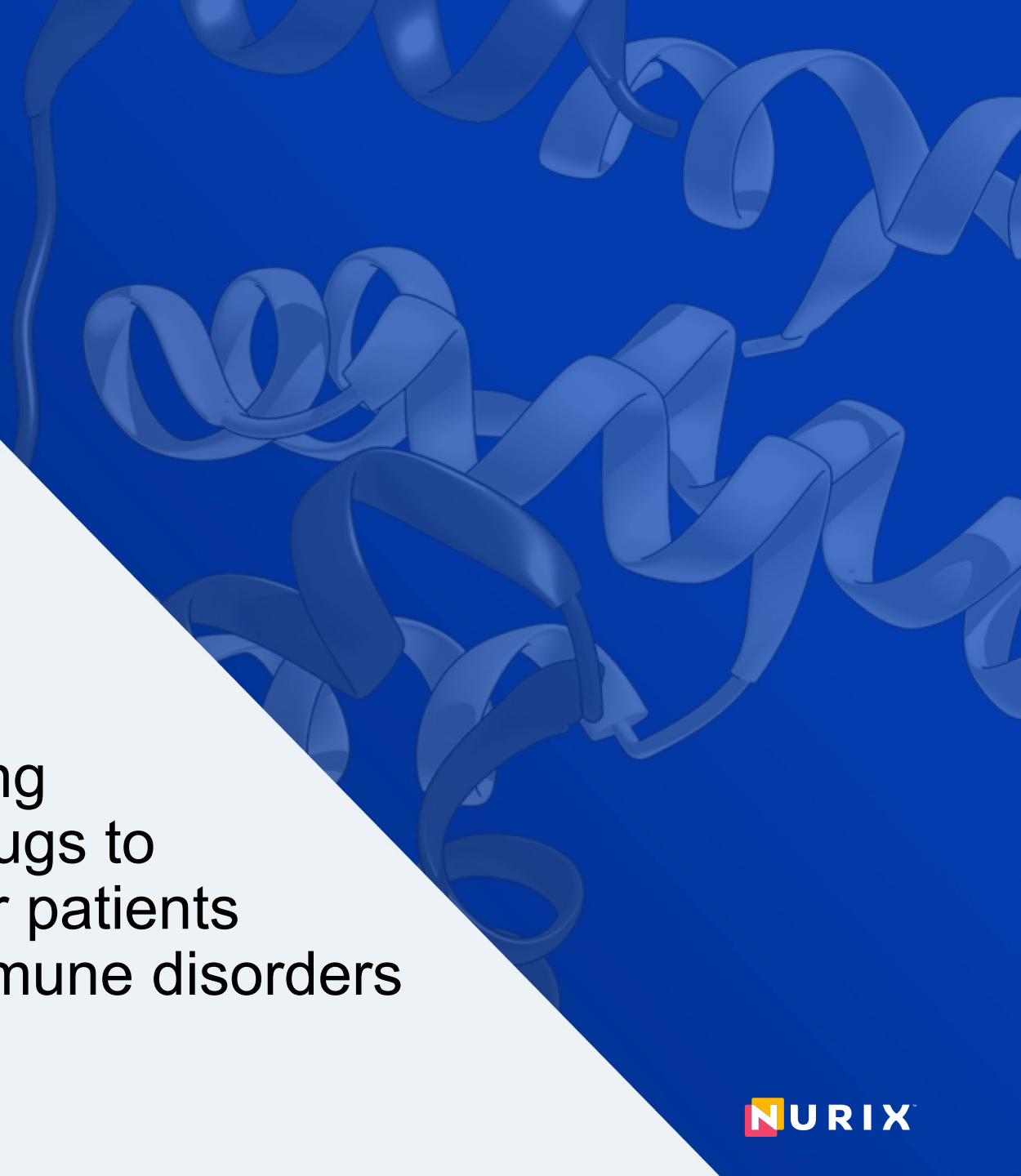
Expansion to 1L+

3. Monotherapy head-to-head vs. investigator choice BTKi* including BTKi treatment naïve patients
4. NX-5948 in combination with BCL2i head-to-head vs. standard of care*

**All plans subject to change based on regulatory feedback including definition of control arms of all studies*

***BTKi**, Bruton's tyrosine kinase inhibitor; **BCL2i**, B-cell leukemia/lymphoma 2 inhibitor; **CLL**, chronic lymphocytic leukemia; **QD**, once daily*

Inflammation & Immunology



Leveraging our expertise in
B- and T-cell biology, Nurix is building
a pipeline of innovative degrader drugs to
address the unmet medical need for patients
living with inflammation and autoimmune disorders

Advancing NX-5948 in Immunology and Inflammation

Key observations underpinning Nurix's NX-5948 I&I strategy

The genetics of BTK are compelling: highly specific with potent biology

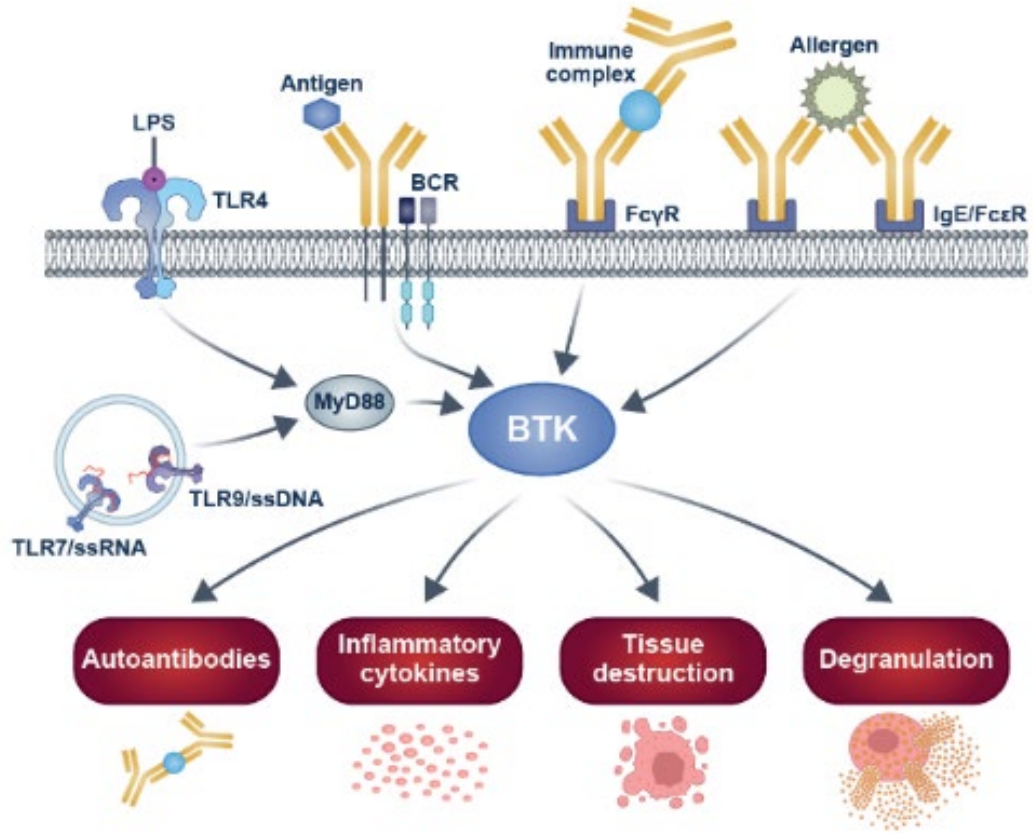
- Human and mouse knockouts are associated with reduced immune function yet have otherwise normal physiology

Positive clinical experience

- BTK inhibitors have shown positive clinical results across a wide range of I&I diseases in hematology, dermatology, and neurology

Inhibitors leave room for improvement

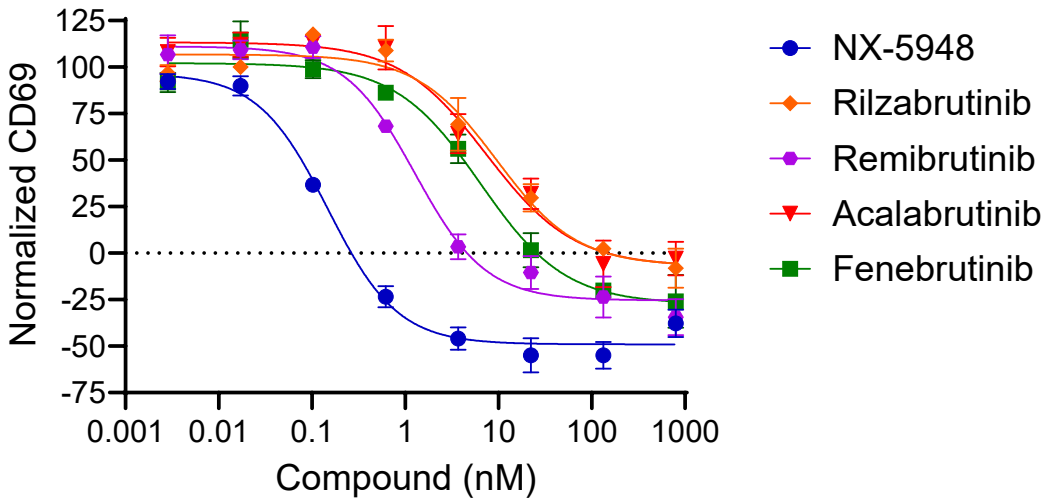
- The same scaffolding functions that limit efficacy of inhibitors in oncology may also be limiting their efficacy in autoimmune disease settings



***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*

NX-5948 More Potently Suppresses Activation of Stimulated B Cells and Myeloid Cells Compared to a Range of BTK Inhibitors

Potent Suppression of B Cell Stimulation



Bead-bound anti-IgM stimulation

Potent Suppression of Myeloid Cell Stimulation

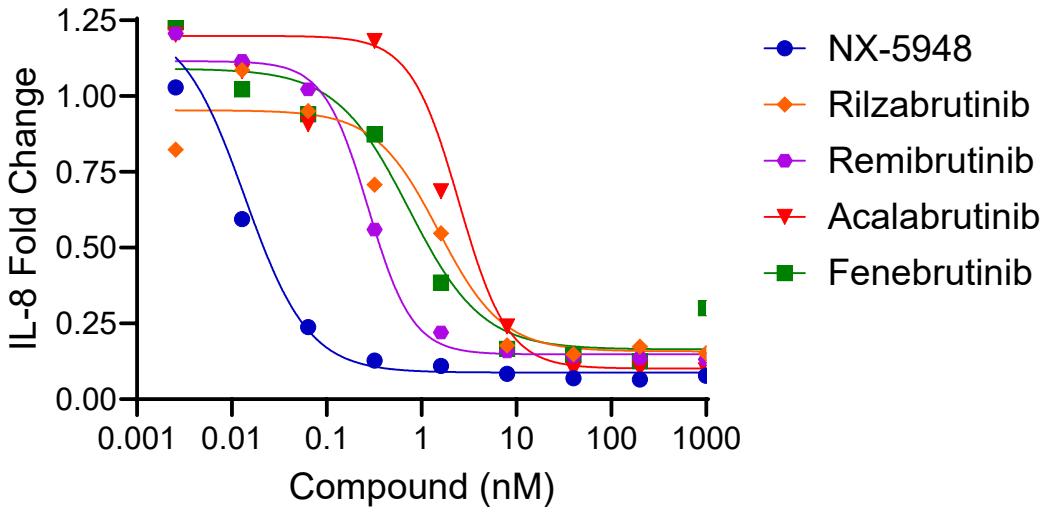
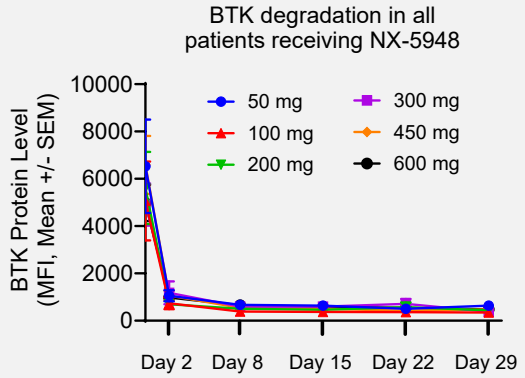


Plate-bound IgG2 stimulation

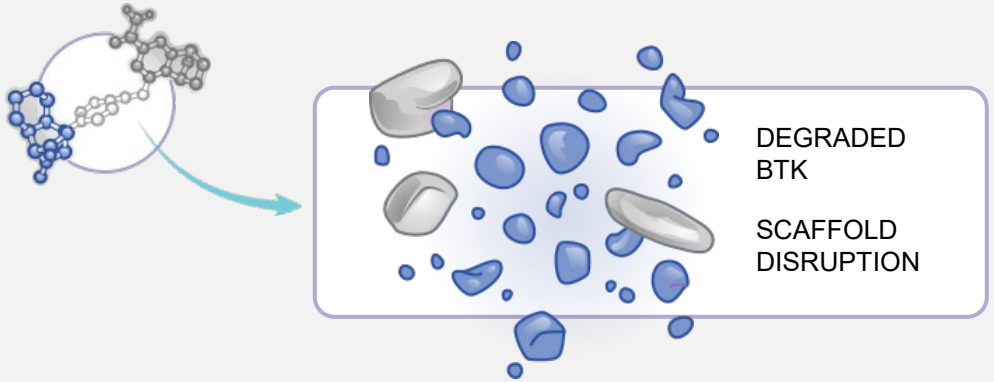
Source: Noviski et al., ACR 2024, November 2024

NX-5948 Has the Right Clinical Profile To Address Unmet Clinical Needs in Both Oncology and I&I

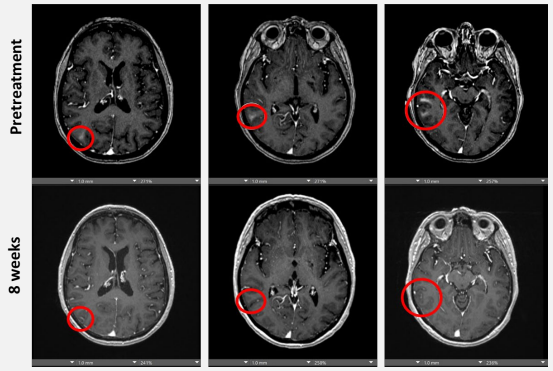
Wide dose range produces robust BTK degradation



Elimination of BTK's scaffolding function



Demonstrated ability to cross the blood-brain barrier



Favorable safety profile in oncology clinical trials

Nurix's Systematic Approach To Expand Development of NX-5948 Across Multiple I&I Indications

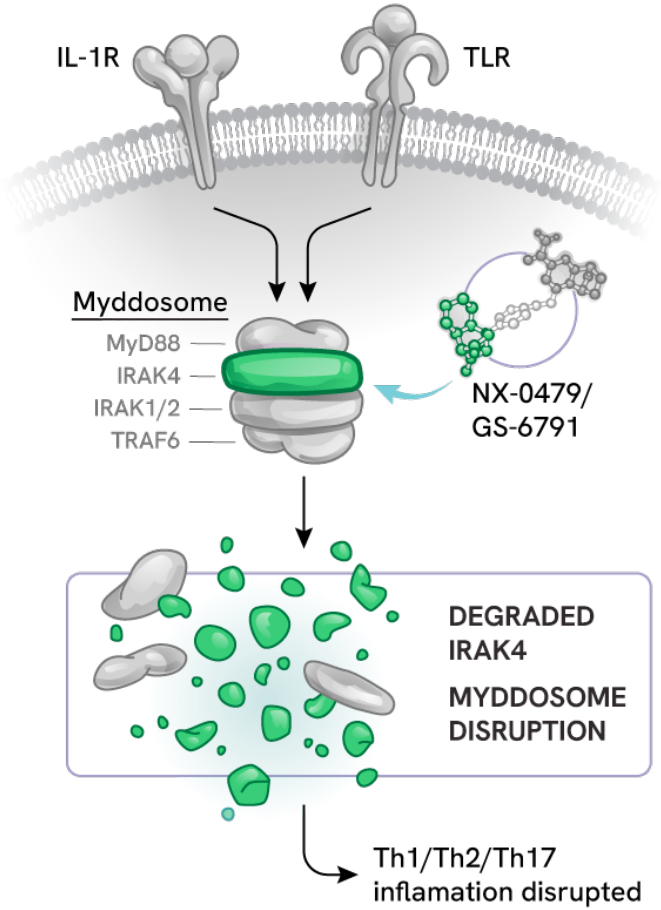
Next Steps:

1. Plan to open a new Phase 1b cohort for patients with CLL and associated autoimmune hemolytic anemia in H1 2025
2. Plan non-malignant hematology IND in 2025 for autoimmune cytopenias (e.g., wAIHA)
3. Conduct a healthy volunteer study of a new formulation to address potential need for broader range of doses and dose regimens for I&I indications (study underway)
4. Explore potential for additional indications in other organ systems based on evolving data (e.g., dermatology and neurology)

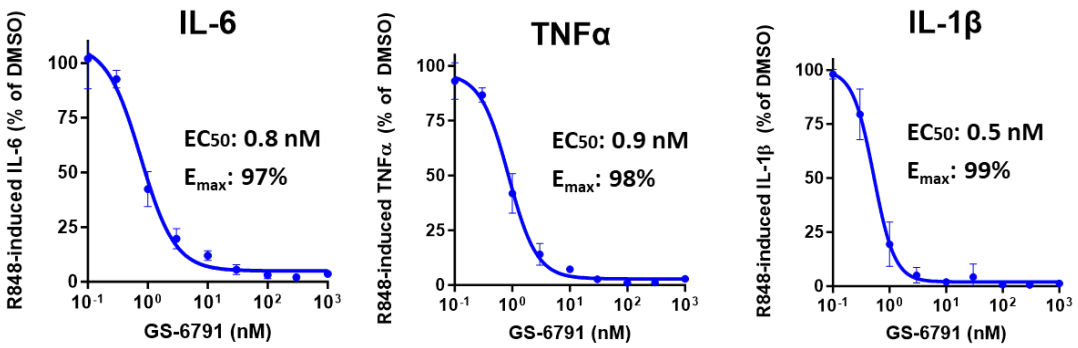
IRAK4 Degrader NX-0479/GS-6791 for the Potential Treatment of Rheumatoid Arthritis and Other Inflammatory Diseases

IND anticipated in 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-14 signaling pathways and yields broader anti-inflammatory effects than inhibition alone



Functional Inhibition of TLR responses in human PBMC



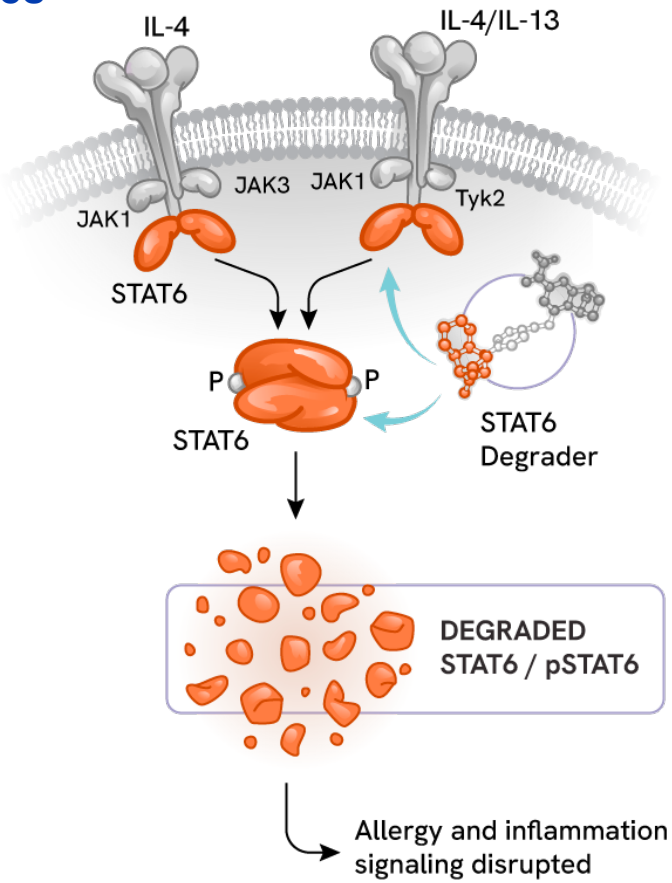
Source: Teng et al., ACR 2024, November 2024 .

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

STAT6 Degrader for the Potential Treatment of Th2-Mediated Inflammatory Disorders Such as Allergies, Asthma, and Eczema

Development candidate nomination anticipated in H1 2025
Nurix has a co-development and 50/50 profit share option in the United States

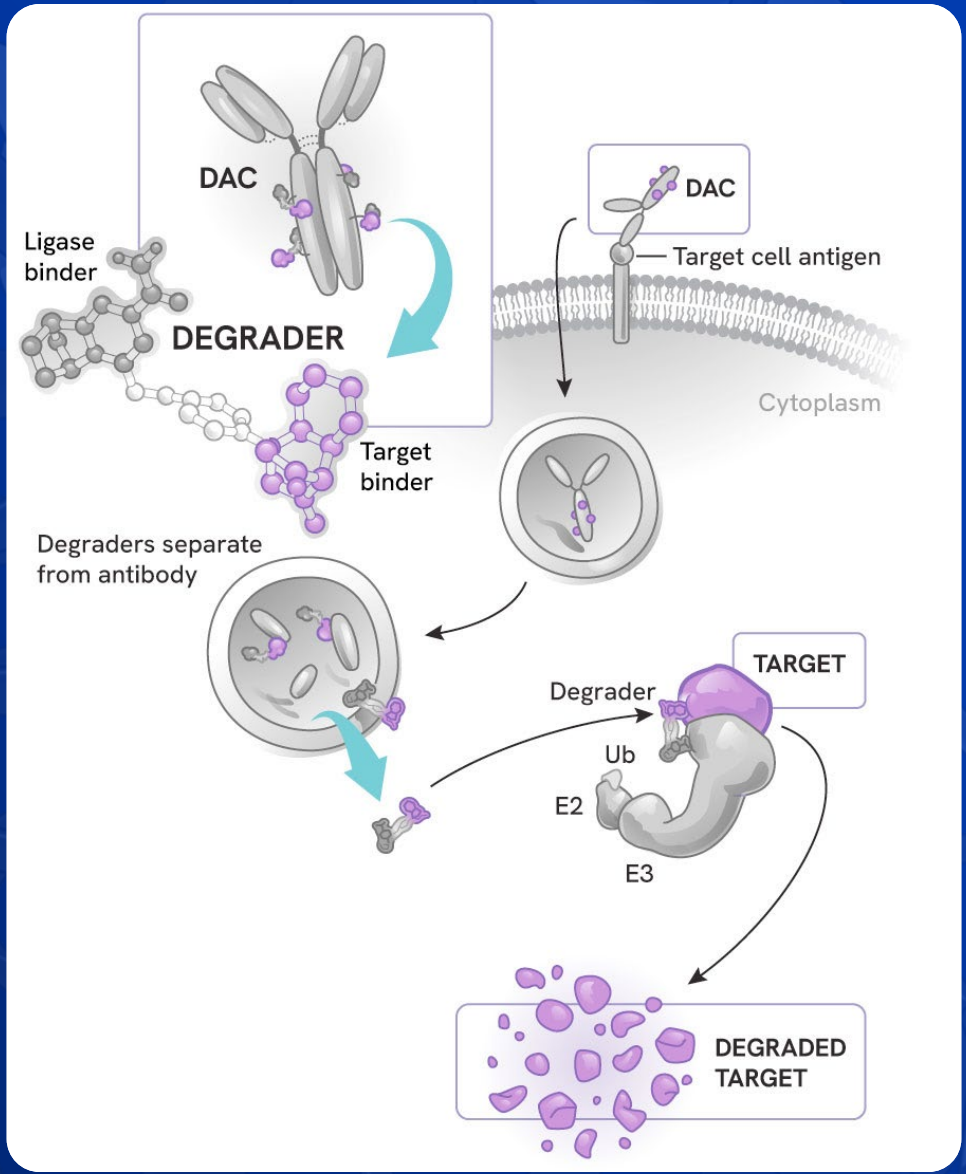
- Signal Transducer and Activator of Transcription 6 (STAT6) is a key transcription factor in the JAK/STAT signaling pathway
- STAT6 acts downstream of the inflammatory cytokines IL-4 and IL-13, driving Th2-mediated inflammatory disorders such as allergies, asthma, and eczema
- STAT6 is considered undruggable by standard inhibitors
- Degradation of STAT6 offers a compelling new modality to target this clinically validated pathway



***JAK**, Janus kinase; **pSTAT**, phosphorylated Janus kinase*

Degrader Antibody Conjugates (DACs)

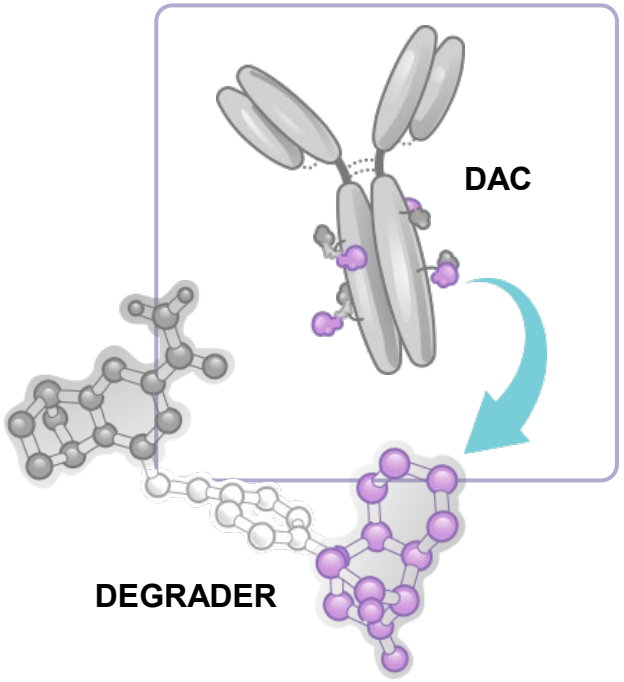
DACs represent the next evolution in targeted protein degradation, combining the highly potent and catalytic activity of degraders with the cell and tissue specificity of antibodies



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

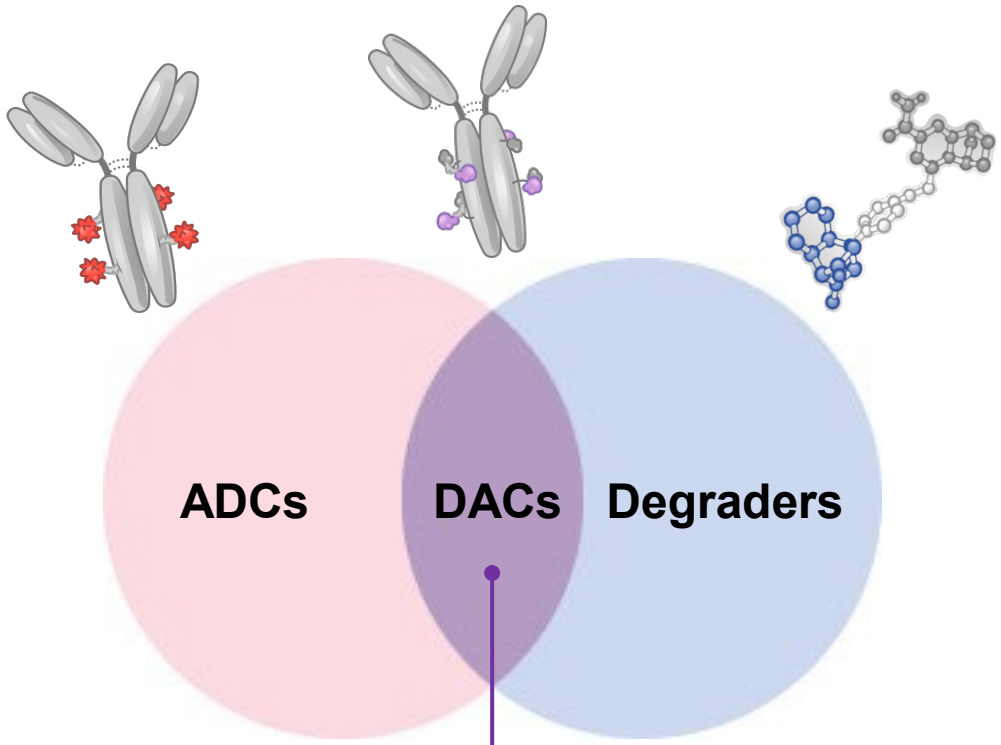
- $60 million upfront cash payment
- $3.4 billion in potential research, development, regulatory and commercial milestone payments
- Mid-single to low double-digit percentage tiered royalties on future product sales
- Option for U.S. profit sharing and co-promotion on up to two products arising from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

Pfizer

** Seagen is now part of Pfizer*

NURIX

DACs Provide Exquisite Selectivity

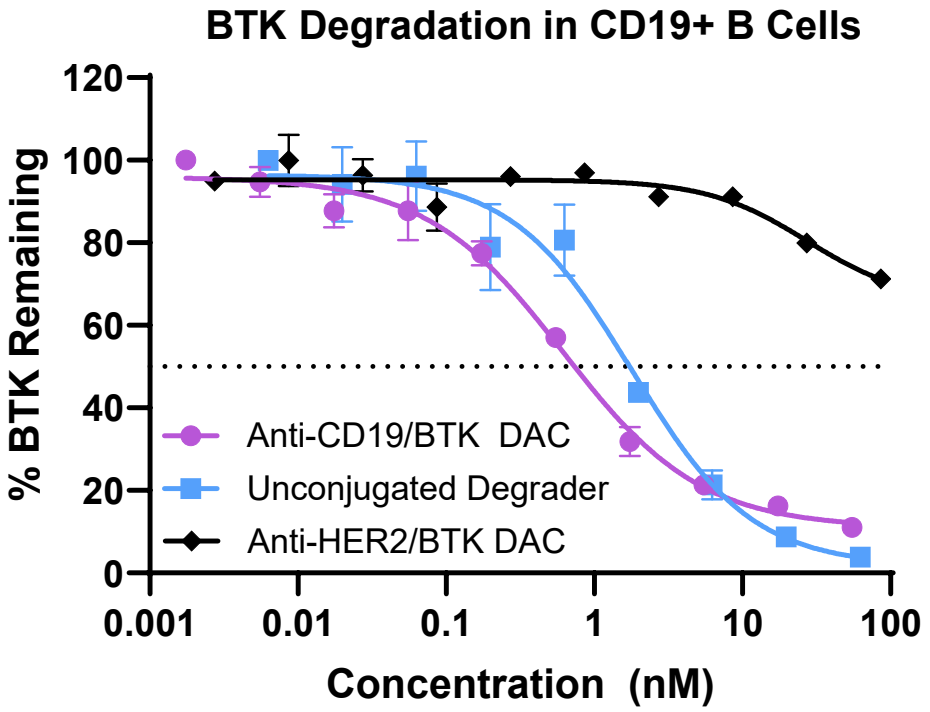


Antigen provides cell and tissue selectivity

Degrader target and ligase expression provide cell and tissue selectivity

DAC provides multi-layer selectivity

DACs Degrade Target Protein Only in Cells Expressing a Select Antigen



Well Funded to Execute on Our Strategy

Estimated $609.6 million in cash and investments at fiscal year-end 2024, November 30, 2024*

Cash runway to fund operations into H1 2027**

Key initiatives for 2025

- Initiate a suite of clinical trials in 2025 intended to support global registration of NX-5948 for the treatment of patients with chronic lymphocytic leukemia
- Expand the development of NX-5948 in additional cancer indications and inflammatory diseases
- Advance our portfolio of partnered programs in inflammation and immunology, including degraders of IRAK4 and STAT6
- Advance Nurix's two other wholly owned clinical-stage assets, NX-2127 and NX-1607
- Invest in our highly productive DEL-AI discovery engine to create and advance novel degrader-based treatments in our wholly owned and partnered portfolios
- Maintain a strong cash position with the ability to fund and control our most valuable programs and co-development options

* The estimated cash and investment amount is a preliminary, unaudited estimate based upon information available to Nurix's management as of the date of this presentation. The amount is subject to the completion of financial closing procedures, including the completion of audit procedures by Nurix's independent registered public accounting firm, and therefore is subject to adjustment.
** Cash runway guidance is based on Nurix's current operating plan

Thank you