UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
Washington, D.C. 20549

FORM 8-K

CURRENT REPORT
Pursuant to Section 13 or 15(d)
of the Securities Exchange Act of 1934

Date of Report (Date of Earliest Event Reported): November 13, 2023

NURIX THERAPEUTICS, INC.
(Exact Name of Registrant as Specified in its Charter)

Delaware	**001-39398**	**27-0838048**
(State or Other Jurisdiction of Incorporation or Organization)	**(Commission File Number)**	**(IRS Employer Identification No.)**

1700 Owens Street, Suite 205 **San Francisco, California** **(Address of Principal Executive Offices)**	**94158** **(Zip Code)**

(415) 660-5320
(Registrant's Telephone Number, Including Area Code)

N/A
(Former Name or Former Address, if Changed Since Last Report)

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions:

☐ Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)

☐ Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)

☐ Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))

☐ Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Securities registered pursuant to Section 12(b) of the Act:

Title of each class	Trading symbol(s)	Name of each exchange on which registered
Common Stock, $0.001 par value per share	NRIX	Nasdaq Global Market

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 (§230.405 of this chapter) or Rule 12b-2 of the Securities Exchange Act of 1934 (§240.12b-2 of this chapter).

Emerging growth company ☐

If an emerging growth company, indicate by check mark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act. ☐

Item 7.01 Regulation FD Disclosure.

Nurix Therapeutics, Inc. (the "Company") intends to conduct meetings with securities analysts, investors and others beginning on November 13, 2023. As part of these meetings, the Company intends to utilize an investor presentation (the "Investor Presentation") which includes updated guidance with respect to the Company's clinical programs and additional detail about the Company's Phase 1 clinical trial evaluating NX-2127 for the treatment of various B-cell malignancies.

A copy of the Investor Presentation is attached hereto as Exhibit 99.1 and is incorporated herein by reference.

In accordance with General Instruction B.2 of Form 8-K, the information in Item 7.01 of this Current Report on Form 8-K shall not be deemed to be "filed" for purposes of Section 18 of the Securities Exchange Act of 1934, as amended (the "Exchange Act"), or otherwise subject to the liability of that section, and shall not be incorporated by reference into any registration statement or other document filed under the Securities Act of 1933, as amended, or the Exchange Act, except as shall be expressly set forth by specific reference in such filing. In addition, the information set forth under this Item 7.01, including Exhibit 99.1, shall not be deemed an admission as to the materiality of any information in this Current Report on Form 8-K.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits

99.1	Nurix Therapeutics, Inc. Investor Presentation dated November 13, 2023.
104	Cover Page Interactive Data File (embedded within the Inline XBRL document).

SIGNATURES

Pursuant to the requirements of the Securities Exchange Act of 1934, as amended the Registrant has duly caused this Report to be signed on its behalf by the undersigned hereunto duly authorized.

NURIX THERAPEUTICS, INC.

Date: November 13, 2023

By: /s/ Arthur T. Sands
Arthur T. Sands, M.D, Ph.D.
President and Chief Executive Officer

Exhibit 99.1

nurix

Leader in Targeted Protein Modulation

Nurix Therapeutics

Blazing a New Path in Medicine

Investor Presentation

November 2023

Important notice and disclaimers

This presentation contains statements that relate to future events and expectations and as such constitute forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995. When or if used in this presentation, the words "anticipate," "believe," "could," "estimate," "expect," "intend," "may," "outlook," "plan," "predict," "should," "will," and similar expressions and their variants, as they relate to Nurix Therapeutics, Inc. ("Nurix", the "Company," "we," "us" or "our"), may identify forward-looking statements. All statements that reflect Nurix's expectations, assumptions or projections about the future, other than statements of historical fact, are forward-looking statements, including, without limitation, statements regarding our future financial or business plans; our future performance, prospects and strategies; future conditions, trends, and other financial and business matters; our current and prospective drug candidates; the planned timing and conduct of the clinical trial programs for our drug candidates; the planned timing for the provision of clinical updates and initial findings from our clinical studies; the potential benefits of our collaborations, including potential milestone and sales-related payments; the potential advantages of our DELigase™ platform and drug candidates; the extent to which our scientific approach, our DELigase™ platform, targeted protein modulation, and Degrader-Antibody Conjugates may potentially address a broad range of diseases; the extent animal model data predicts human efficacy; and the timing and success of the development and commercialization of our current and anticipated drug candidates. Forward-looking statements reflect Nurix's current beliefs, expectations, and assumptions. Although Nurix believes the expectations and assumptions reflected in such forward-looking statements are reasonable, Nurix can give no assurance that they will prove to be correct. Forward-looking statements are not guarantees of future performance and are subject to risks, uncertainties and changes in circumstances that are difficult to predict, which could cause Nurix's actual activities and results to differ materially from those expressed in any forward-looking statement. Such risks and uncertainties include, but are not limited to: (i) risks and uncertainties related to Nurix's ability to advance its drug candidates, obtain regulatory approval of and ultimately commercialize its drug candidates; (ii) the timing and results of clinical trials; (iii) Nurix's ability to fund development activities and achieve development goals; (iv) risks and uncertainties relating to the timing and receipt of payments from Nurix's collaboration partners, including milestone payments and royalties on future potential product sales; (v) the impact of macroeconomic events and conditions, including increasing financial market volatility and uncertainty, inflation, increasing interest rates, instability in the global banking system, uncertainty with respect to the federal budget, the impact of war, military or regional conflicts, and global health pandemics, on Nurix's clinical trials and operations; (vi) Nurix's ability to protect intellectual property and (vii) other risks and uncertainties described under the heading "Risk Factors" in Nurix's Quarterly Report on Form 10-Q for the fiscal quarter ended August 31, 2023, and other SEC filings. Accordingly, readers are cautioned not to place undue reliance on these forward-looking statements. The statements in this presentation speak only as of the date of this presentation, even if subsequently made available by Nurix on its website or otherwise. Nurix disclaims any intention or obligation to update publicly any forward-looking statements, whether in response to new information, future events, or otherwise, except as required by applicable law.

Certain information contained in this presentation relates to or is based on studies, publications, surveys and other data obtained from third-party sources and the Company's own internal estimates and research. While the Company believes these third-party sources to be reliable as of the date of this presentation, it has not independently verified, and makes no representation as to the adequacy, fairness, accuracy or completeness of, any information obtained from third-party sources. In addition, all of the market data included in this presentation involves a number of assumptions and limitations, and there can be no guarantee as to the accuracy or reliability of such assumptions. Finally, while we believe our own internal estimates and research are reliable, such estimates and research have not been verified by any independent source.

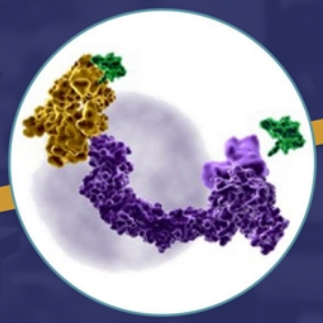
Nurix Drugs Engage Ligases for the Treatment of Cancer

Targeted Protein Modulation: TPM = TPD + TPE

A Powerful Cellular System

Targeted Protein Elevation (TPE)

Harness ligases to decrease specific protein levels



Inhibit ligases to increase specific protein levels

Targeted Protein Degradation (TPD)

Ubiquitin is ligated to target proteins to tag them for degradation by the proteasome

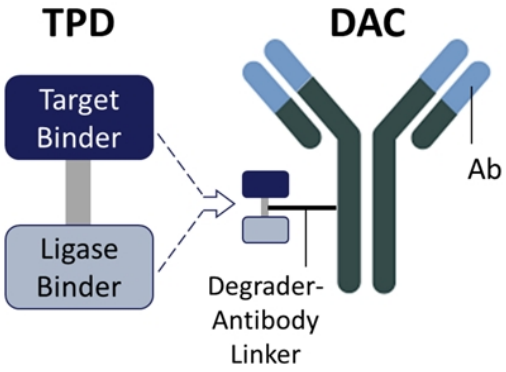
Nurix Is Advancing a Pipeline of Propriety and Partnered Programs in Oncology and Autoimmune/Inflammatory Diseases

MOA	Drug program	Target	Therapeutic area	Discovery	IND enabling	Phase 1a	Phase 1b
TPD	NX-2127	BTK-IKZF	B-cell malignancies				
	NX-5948	BTK	B-cell malignancies				
	NX-0479 / GS-6791	IRAK4	Rheumatoid arthritis and other inflammatory diseases				GILEAD
	Multiple	Undisclosed	Oncology / autoimmune disease				
	Multiple	Undisclosed	Undisclosed				GILEAD
	Multiple	Undisclosed	Undisclosed				sanofi
TPE	NX-1607	CBL-B	Immuno-Oncology				
DAC	Multiple	Undisclosed	Oncology				Seagen

Advancing a New Therapeutic Class

Degrader-Antibody Conjugates (DACs)

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

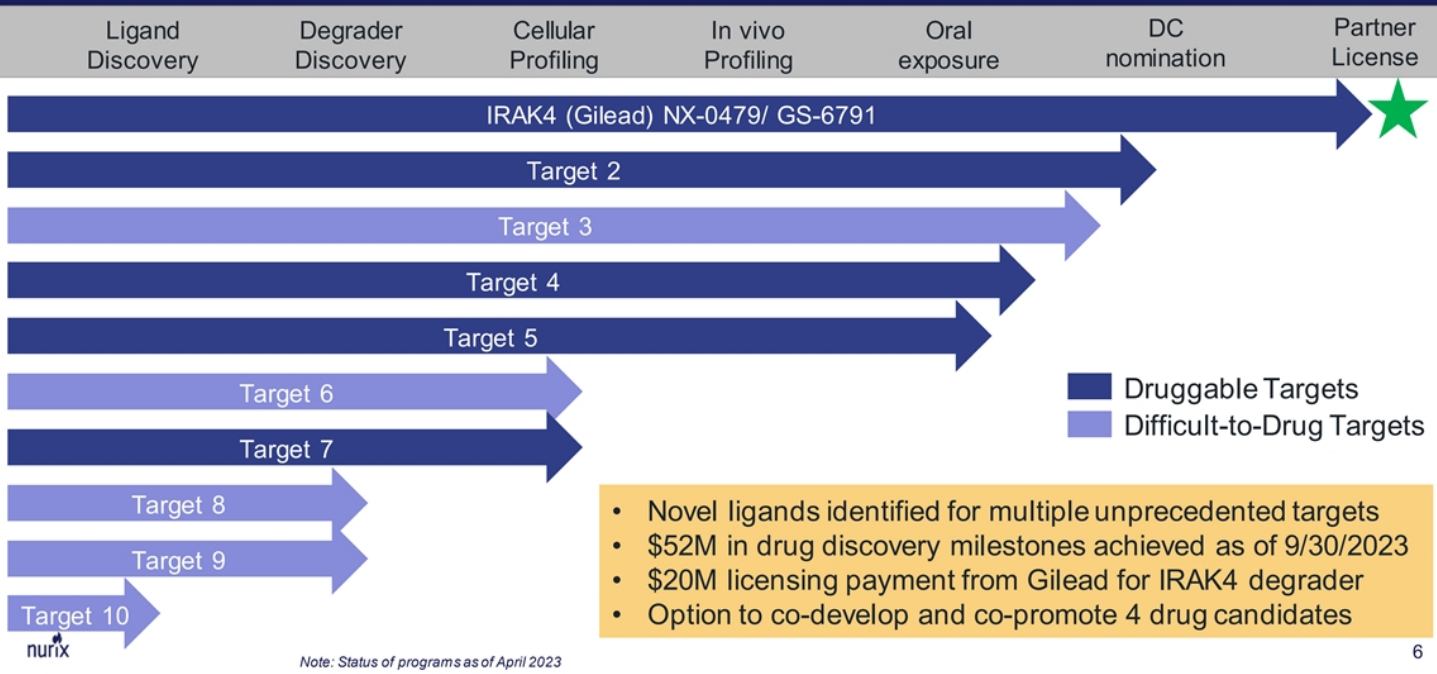


Deal Terms

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

Seagen®

Leveraging Gilead and Sanofi Partnerships To Advance a Broad Pipeline of Targeted Protein Degraders



- Novel ligands identified for multiple unprecedented targets
- $52M in drug discovery milestones achieved as of 9/30/2023
- $20M licensing payment from Gilead for IRAK4 degrader
- Option to co-develop and co-promote 4 drug candidates

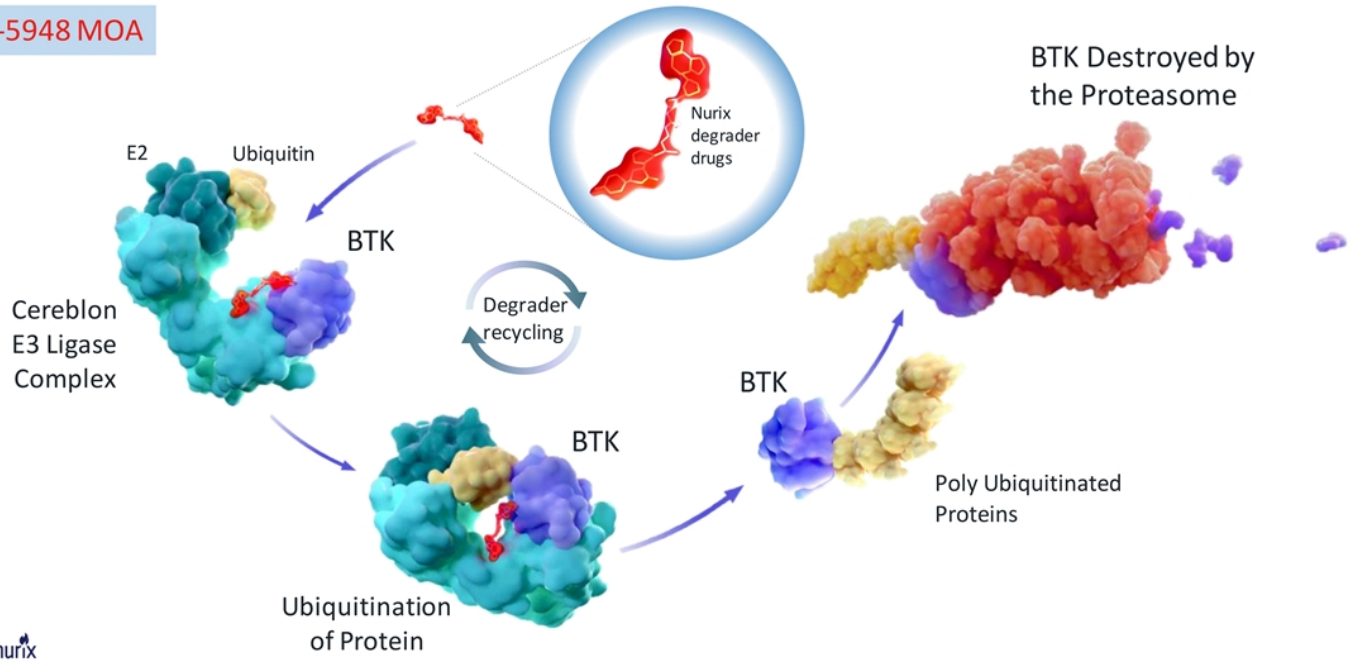
nurix

Note: Status of programs as of April 2023

Targeted Protein Degradation

Harnessing the ubiquitin proteosome system to eliminate disease proteins

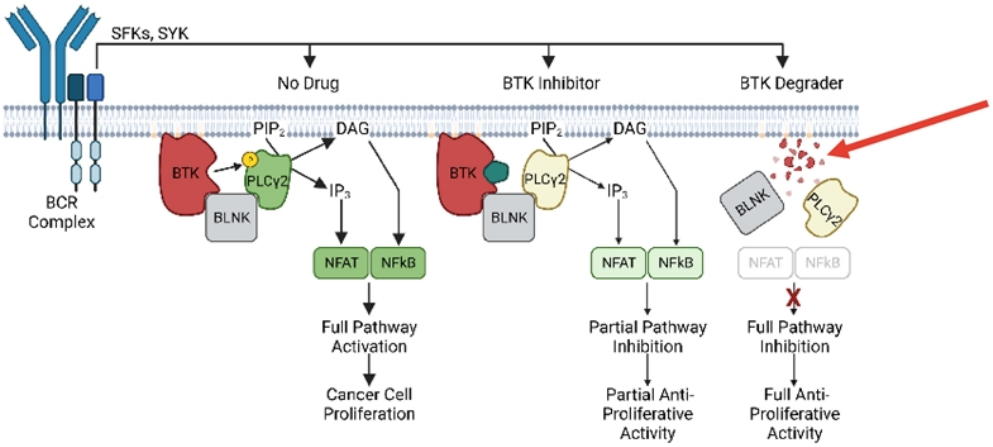
NX-5948 MOA



Degraders More Completely Disrupt BCR Signaling

Nurix Degraders:

1) Are effective against resistance mutations through binding cooperativity between BTK and the ligase complex
2) Eliminate the scaffolding function of BTK oncogenic signals

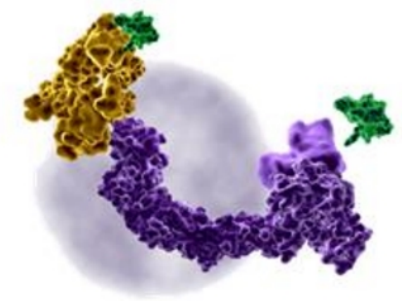


Removal of BTK disrupts the signaling complex, effectively destroying the scaffolding function of the protein

A First-In-Class Franchise of BTK Degraders:
NX-5948 & NX-2127 – The Big Picture

NX-5948
SELECTIVE BTK DEGRADATION

NX-2127
BTK DEGRADATION & IMMUNOMODULATION



BTK degraders have the potential to displace inhibitors in the markets where BTK inhibitors currently dominate (e.g., CLL)

Nurix has demonstrated that BTK degraders can overcome treatment emergent resistance mutations to both covalent and non-covalent inhibitors

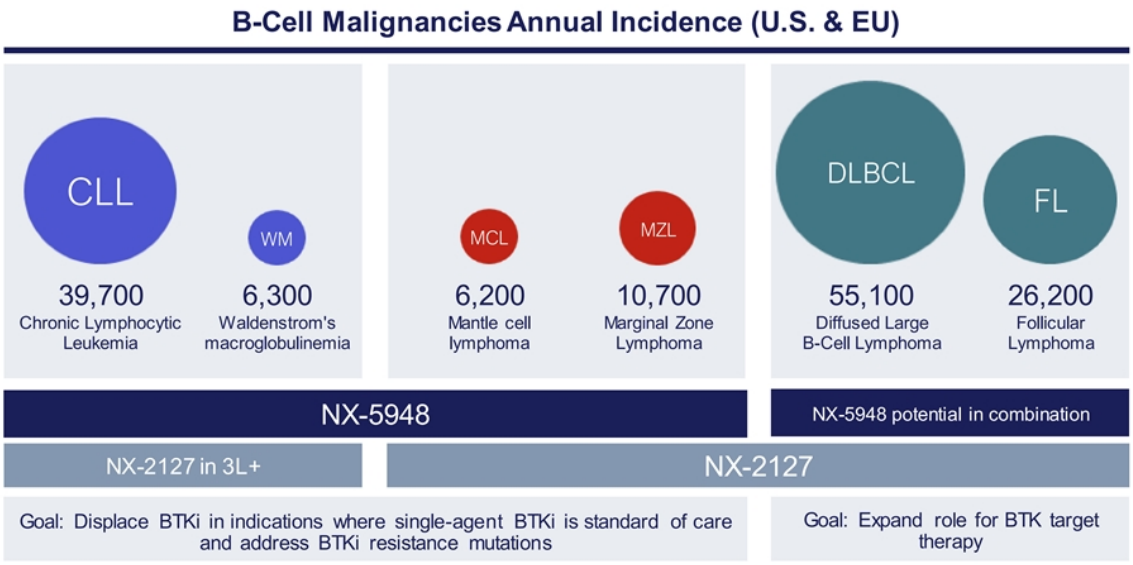
BTK degraders may expand the market for BTK targeted agents into other B-cell malignancies such as DLBCL and potentially into autoimmune diseases

NX-5948 and NX-2127 are two distinct drugs with differentiated profiles, each with the potential to be multi-billion dollar B-cell malignancy therapeutic franchises

Nurix BTK Degrader Franchise: Two BTK Degraders To Cover the Landscape of B-Cell Malignancies

NX-5948
for BTK inhibitor resistance mutations in CLL with potential for early lines of therapy

NX-2127
for aggressive NHL and advanced CLL including BTK inhibitor resistance mutations



NX-5948 | NX-5948 potential in combination

NX-2127 in 3L+ | NX-2127

Goal: Displace BTKi in indications where single-agent BTKi is standard of care and address BTKi resistance mutations

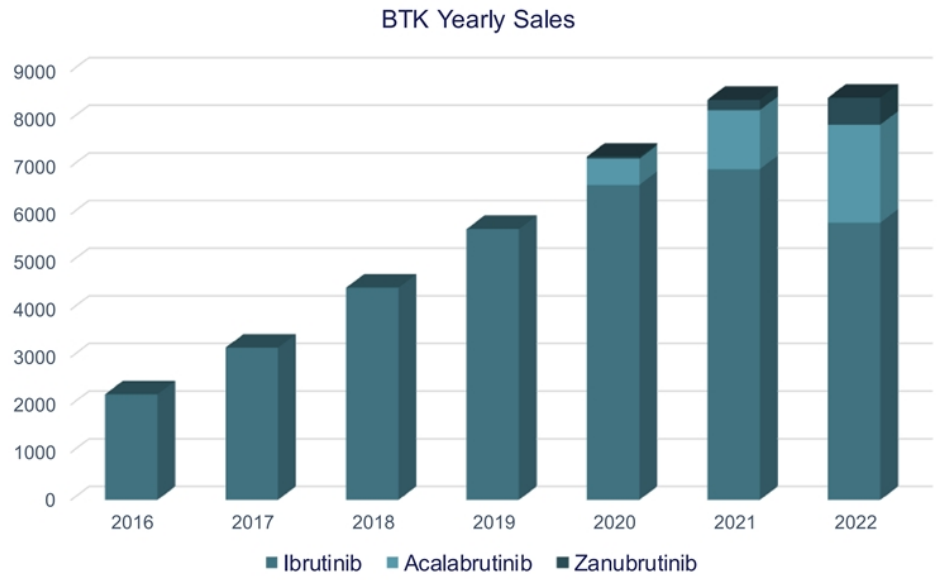
Goal: Expand role for BTK target therapy

BTK, Bruton tyrosine kinase; DLBCL, Diffuse large B cell lymphoma; CLL, Chronic lymphocytic leukemia, SLL, small lymphocyticlymphoma; MCL, Mantle cell lymphoma; WM, Waldenstrom's macroglobulinemia; MZL, Marginal zone lymphoma; FL, Follicular lymphoma; NHL, non-Hodgkin lymphoma

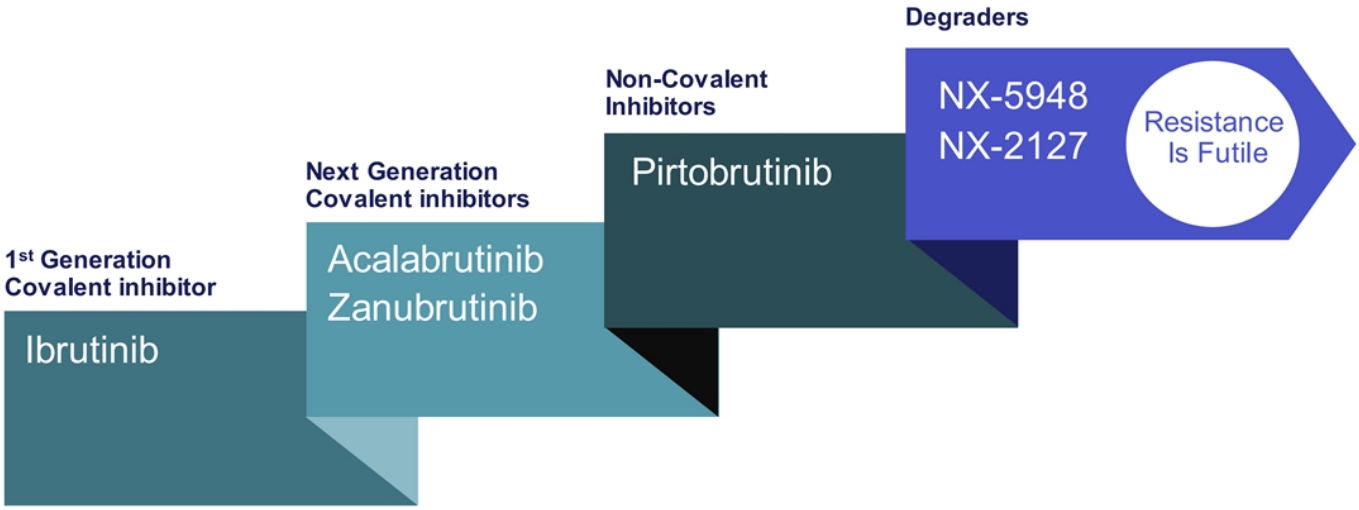
Estimates based on 2020 incidence from DRG, GlobalData and secondary research; EU comprised of France, Germany, Italy, Spain and UK

Blockbuster Opportunity in BTK Market
$8.4 billion in annual sales

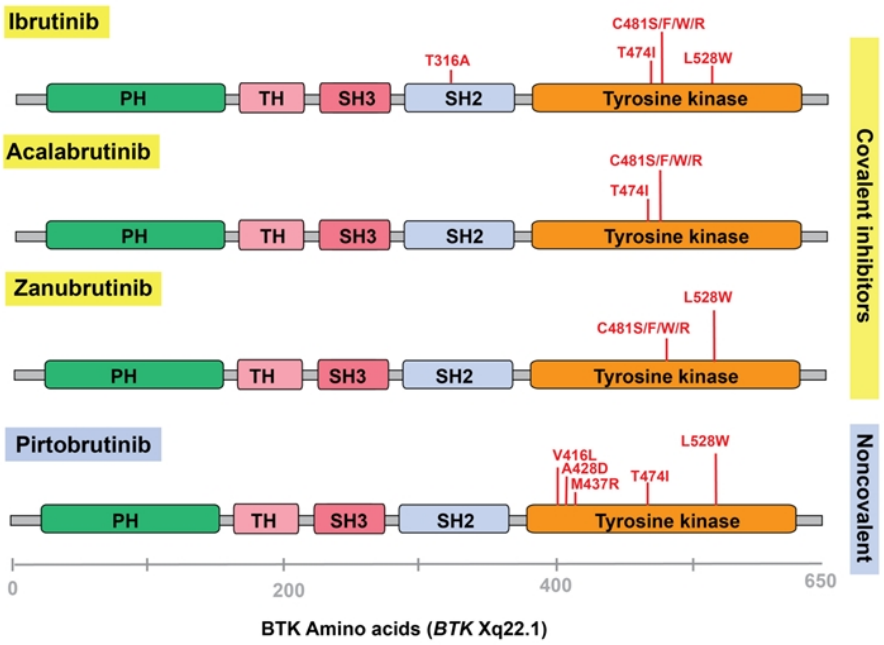
- Next generation BTK inhibitors are currently taking market share from Imbruvica
- Nurix BTK degraders have the potential to be game changing and take shares from the inhibitor market in CLL
- Opportunity for Nurix BTK degraders to expand the market in other B cell malignancies and autoimmune diseases



Evolution of BTK Targeted Therapies

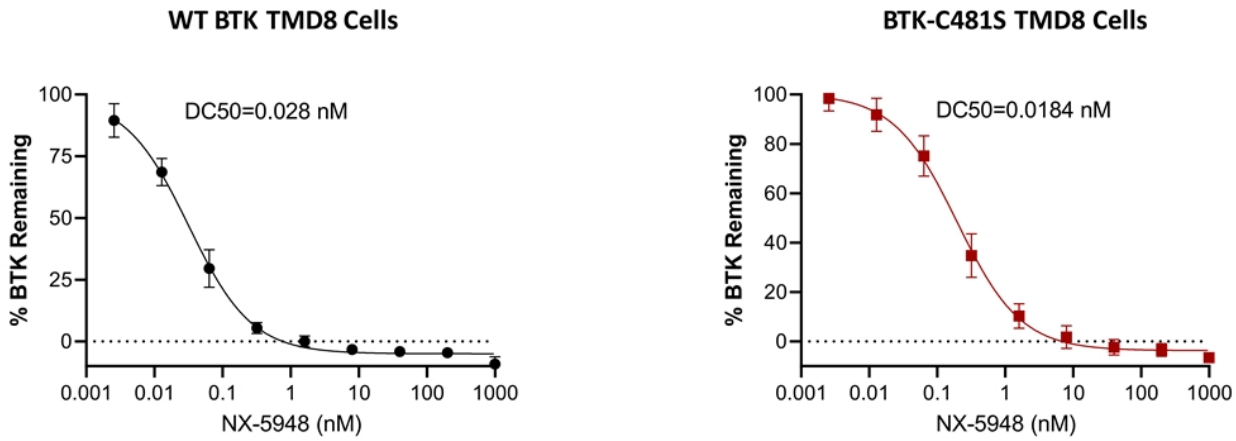


Emerging Unmet Medical Need with Resistance Mutations to Existing BTK Inhibitors



NX-5948 and NX-2127 can degrade all treatment emergent inhibitor mutations identified to date

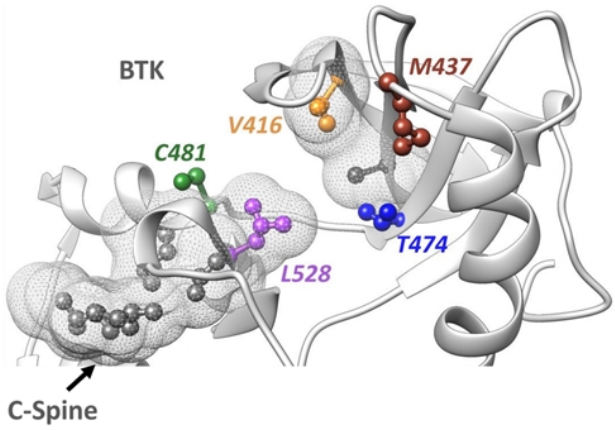
NX-5948 Was Designed for Potent and Rapid Degradation of Wildtype and C481S-Mutated BTK



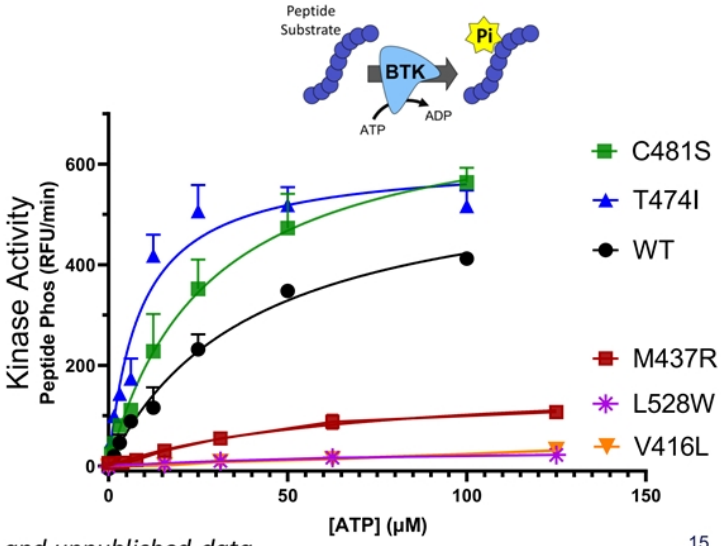
TMD8 cells harboring WT BTK or a knock-in BTK mutation (C481S) were incubated with NX-5948 for 24 hours, and BTK degradation was assessed by flow cytometry.

Structural and Enzymatic Studies of New BTKi-Resistant Mutations Confirms BTK Scaffolding Function

Mutations revealed by non-covalent inhibitors interrupt the catalytic C-spine of kinase domain



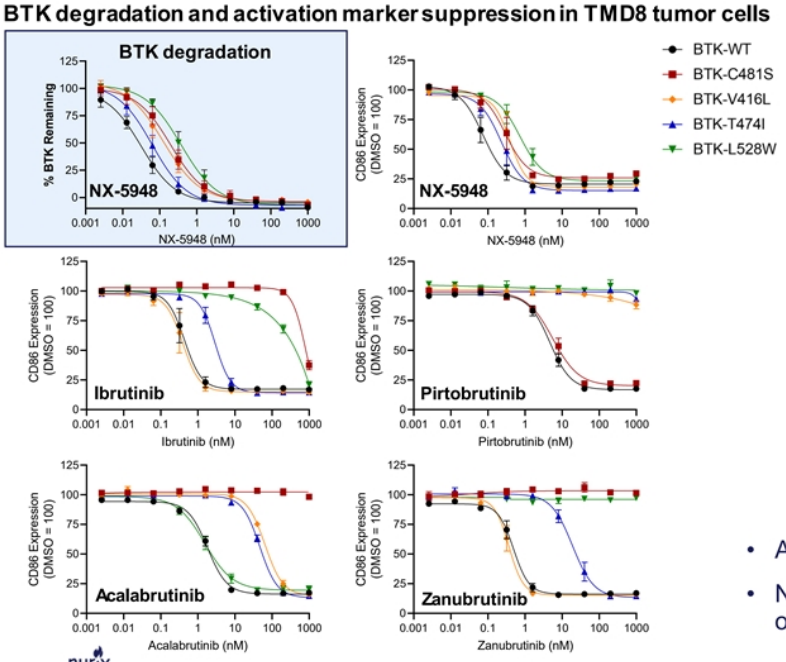
Some mutations that confer resistance to BTK inhibitors lack kinase activity yet still potentiate BCR signaling



Montoya et al., ASH 2022 and unpublished data

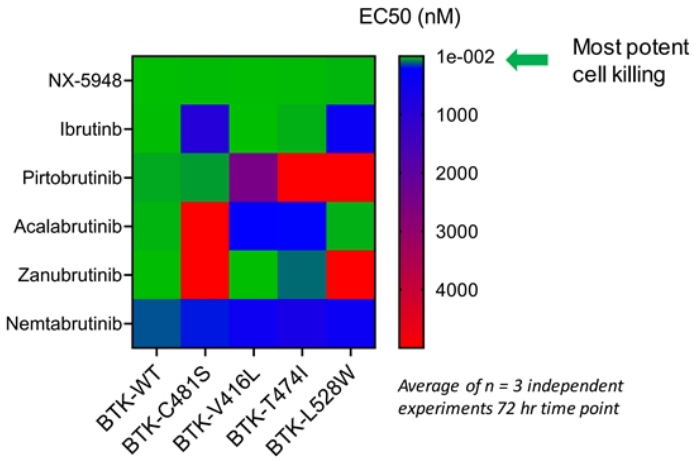
NX-5948 Is More Potent and Broadly Active Than All BTK Inhibitors Tested

BTK degradation and activation marker suppression in TMD8 tumor cells



Average of n = 3 independent experiments +/- SEM

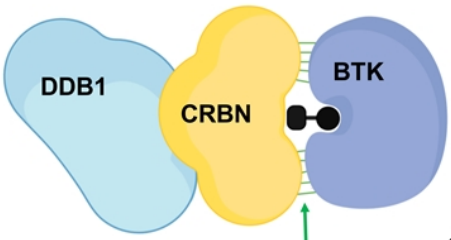
TMD8 tumor cell killing



Average of n = 3 independent experiments 72 hr time point

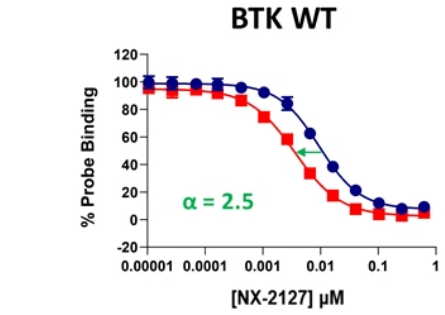
- All inhibitors have resistance mutation liabilities
- NX-5948 displays potent cell killing and maintains suppression of CD86 in the context of key resistance mutations

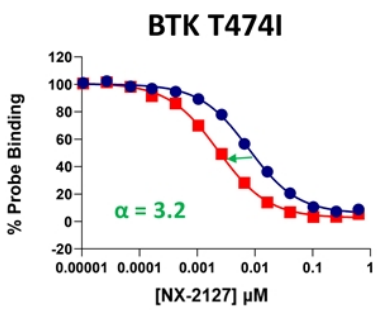
NX-2127 Induces Positive Binding Cooperativity Between BTK and Cereblon

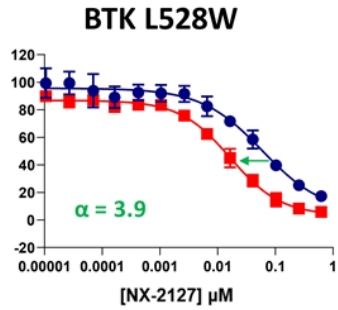


- Positive Cooperativity (α>1)
- Stable ternary complex
- Induced protein-protein interactions
- Greater tolerance for reduced binary affinity

CRBN, cereblon; **DDB1**, DNA damage binding protein 1 (component of the ubiquitin ligase complex)







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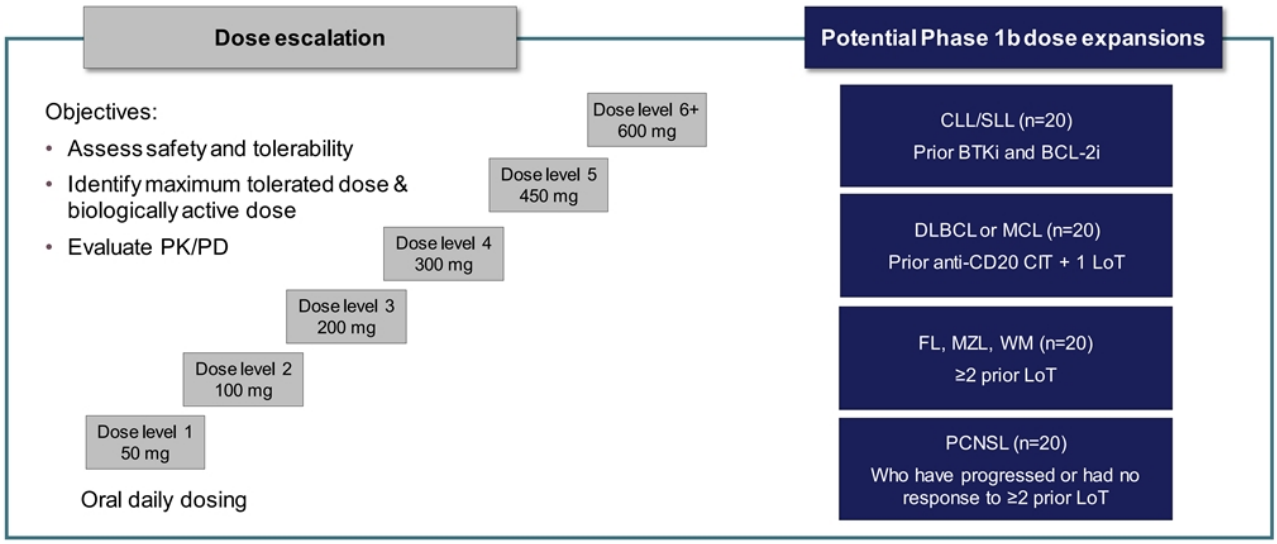
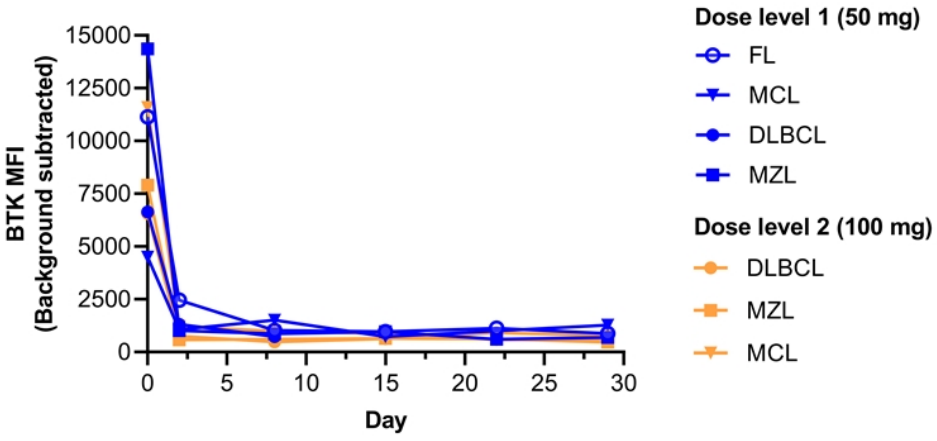
NX-5948-301: Trial Design

Phase 1 trial in adults with relapsed/refractory B-cell malignancies



BTK, Bruton tyrosine kinase; **CLL**, chronic lymphocytic leukemia; **DLBCL**, diffuse large B-cell lymphoma; **FL**, follicular lymphoma; **LoT**, line of therapy; **MCL**, mantle cell lymphoma; **MZL**, marginal zone lymphoma; **PCNSL**, primary CNS lymphoma; **PD,** pharmacodynamics; **PK,** pharmacokinetics; **WM**, Waldenstrom's macroglobulinemia

First Report of BTK Degradation with NX-5948 in Patients with B Cell Malignancies



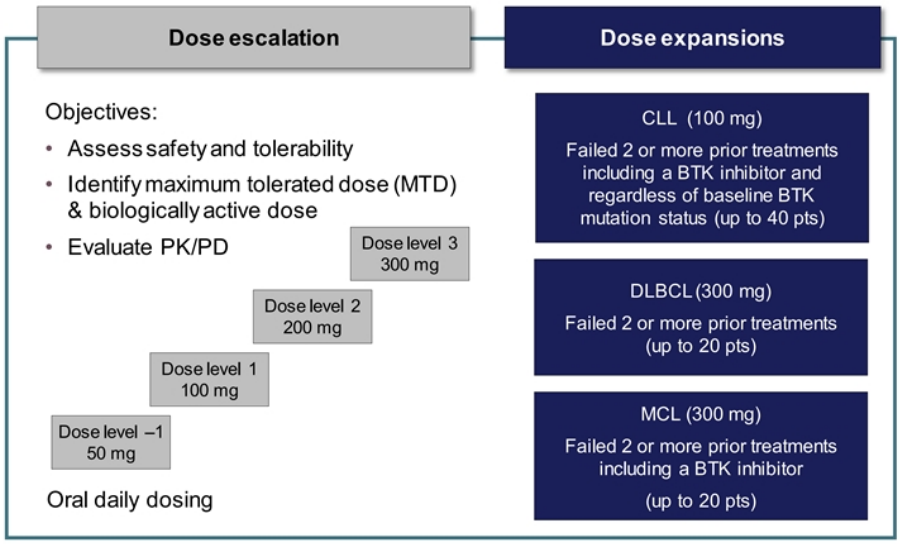
BTK levels are evaluated in real time in a FACS-based assay on whole blood from patients treated with NX-2127

Initial proof of mechanism

- Rapid and sustained degradation of BTK
- Robust BTK degradation observed in all patients tested to date
- Dose escalation ongoing in patients with relapsed/refractory B cell malignancies

NX-2127-001: Trial Design

Phase 1 trial in adults with relapsed/refractory B-cell malignancies



- Enrollment of new patients paused due to partial clinical hold, pending alignment with FDA on introduction of new chirally controlled drug product
- CLL Phase 1b expansion cohort ongoing for patients currently on study
- DLBCL Phase 1b expansion cohort ongoing for patients currently on study
- MCL Phase 1b expansion cohort ongoing for patients currently on study
- Phase 1a dose escalation is ongoing for patients currently on study with NHL

BTK, Bruton tyrosine kinase; **CLL**, chronic lymphocytic leukemia; **DLBCL**, diffuse large B-cell lymphoma; **FL**, follicular lymphoma; **MCL**, mantle cell lymphoma; **MZL**, marginal zone lymphoma; **NHL**, non-Hodgkin lymphoma; **PCNSL**, primary CNS lymphoma; **PD,** pharmacodynamics; **PK,** pharmacokinetics; **WM**, Waldenstrom's macroglobulinemia

Baseline Characteristics

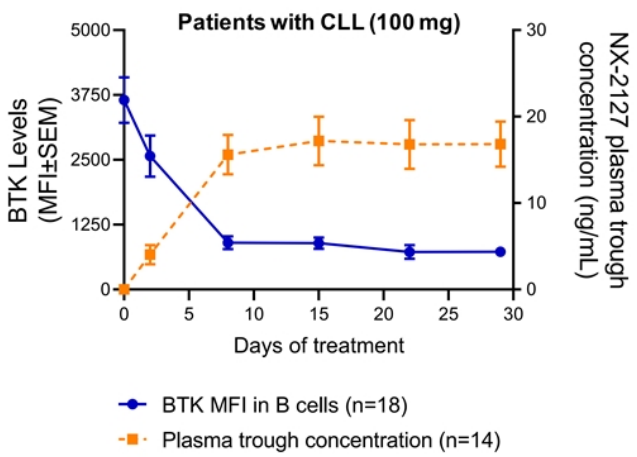
Elderly population with multiple prior lines of targeted therapies and acquired mutations

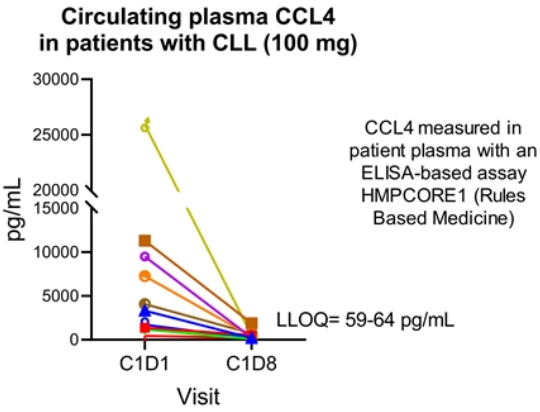
Characteristics	CLL (n=23)	Overall population (N=36)
Median age, years (range)	75 (61–90)	75 (50–92)
Female, n (%)	9 (39.1)	13 (36.1)
Male, n (%)	14 (60.9)	23 (63.9)
Lines of prior therapy, median (range)	5 (2–11)	4 (2–11)
BTKi, n (%)	23 (100)	31 (86.1)
Pirtobrutinib, n (%)	8 (34.8)	11 (30.6)
BTKi and BCL2i, n (%)	18 (78.3)	19 (52.8)
cBTKi, ncBTKi, and BCL2i, n (%)	7 (30.4)	7 (19.4)
***BTK* mutation present**[a], n (%)	10 (48)	11 (35)
C481	5 (24)	5 (16)
L528W	4 (19)	4 (13)
T474	3 (14)	4 (13)
V416L	1 (5)	1 (3)
***BCL2* mutation present**[a], n (%)	4 (19)	4 (13)
***PLCG2* mutation present**[a], n (%)	0 (0)	1 (3.2)

[a]Specific mutations are not additive as some patients have multiple *BTK* mutations
Mutations were tested by NGS centrally in those patients with available samples (n=31 in total population; n=21 in CLL population)

Data cutoff: September 21, 2022

NX-2127 Leads to Robust BTK Degradation and Decrease in B-cell Activation





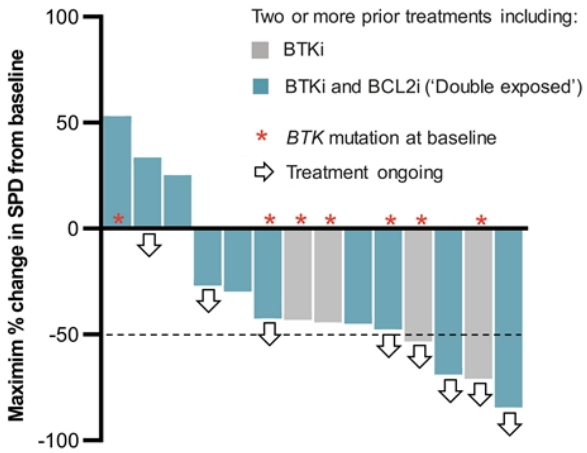
- Daily treatment with NX-2127 resulted in a rapid and sustained suppression of BTK (CD19+) as measured in patient whole blood using a flow cytometry assay. BTK suppression target of 80% reached consistently (data not shown here)
- Robust decrease of plasma CCL4 by Cycle 1 Day 8 and suppression was maintained through Cycle 2 Day 1, consistent with clinically observed lymphocytosis occurring in majority of patients with nodal disease by Cycle 1 Day 8
- NX-2127 treatment also resulted in degradation of cereblon neo-substrate Ikaros

BTK, Bruton's tyrosine kinase; **CCL4**, C–C motif ligand 4; **LLOQ**, lower limit of quantification

NX-2127 Preliminary Efficacy
Positive Initial Findings in CLL

Disease-evaluable patients	n=15
Objective response rate,[a] % (95% CI)	33 (12–62)
Best response, n (%)	
CR	0 (0)
PR	5 (33.3)
SD	5 (33.3)
PD	2 (13.3)
NE[b]	3 (20)

[a]Objective response rate includes CR + CRi + nPR + PR-L + PR
[b]Patients who discontinued after a single assessment of SD are considered as NE



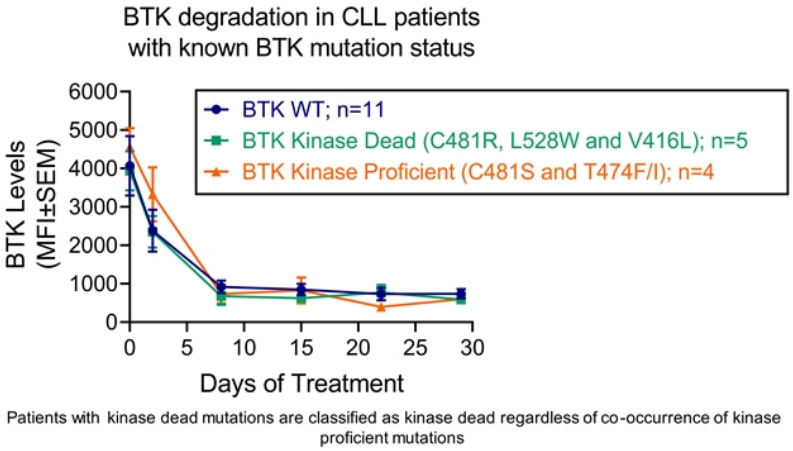
***One patient, not shown above, with prior BTKi and BCL2i treatment and with a *BTK* mutation detected at baseline, had no nodal disease at baseline. Their treatment is ongoing with a PR.**

BCL2i, B-cell lymphoma-2 inhibitor; **BTK**, Bruton's tyrosine kinase; **BTKi**, BTK inhibitor; **CR**, complete response; **CRi**, complete response with incomplete count recovery; **NE**, not evaluable; **PD**, progressive disease; **PR**, partial response; **SD**, stable disease

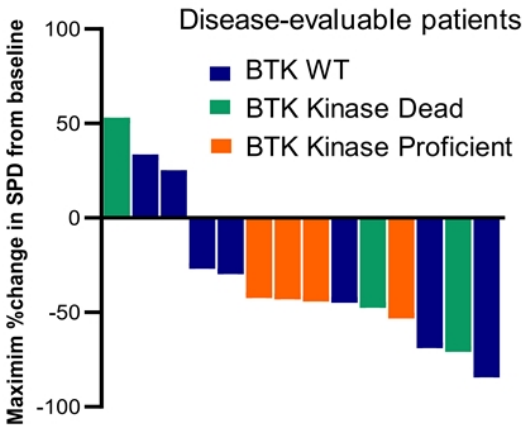
Data cutoff: September 21, 2022

First Demonstration of Clinical Activity of a Degrader Against a Range of BTK Mutations

NX-2127 Preliminary Efficacy in Patients with CLL



Patients with kinase dead mutations are classified as kinase dead regardless of co-occurrence of kinase proficient mutations



- BTK degradation of 80% achieved in CLL patients including those harboring BTK C481, T474, L528, and V416 resistance mutations

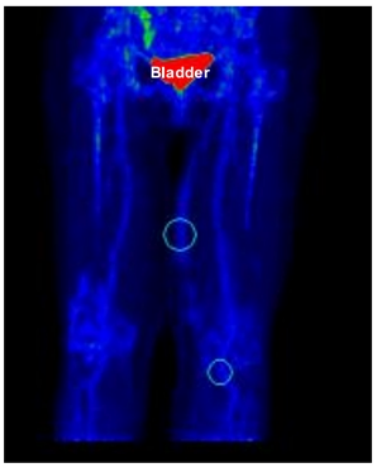
Rapid and Sustained Complete Response on Single-Agent NX-2127

FDG-PET CT Scan Disease Assessment

Baseline

Deauville score: 5

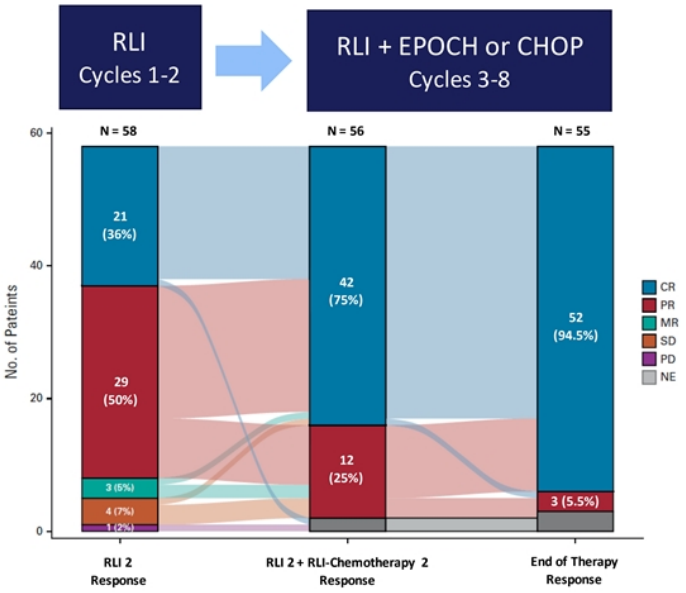
Confirmatory Week 16 Scan



Deauville score: 2

- 84-year-old woman with multiply relapsed ABC-DLBCL following 4 lines of aggressive therapy (including combination of rituximab, ibrutinib, and lenalidomide).
- Complete response at first assessment (week 8), confirmed at week 16, and ongoing through week 24.
- As of June 14, 2023, this patient remains on treatment with over 12 months of follow up

Phase 2 Smart Start: Ibrutinib, Lenalidomide, and Rituxan + Chemo in Newly Diagnosed Non-GCB DLBCL



Journal of Clinical Oncology

Smart Start: Rituximab, Lenalidomide, and Ibrutinib in Patients With Newly Diagnosed Large B-Cell Lymphoma

"The combination of RLI alone and with chemotherapy resulted in high response rates and promising survival outcomes in patients with newly diagnosed DLBCL."

"Smart Start resulted in PFS and OS rates at 2 years, of 91.3% and 96.6%, respectively. R-CHOP with and without ibrutinib resulted in a 3-year PFS rates of 70.8% and 68.1%, respectively. R-CHOP with and without lenalidomide resulted in a 2-year PFS rates of 67% and 64%, respectively."

Source: Westin et al; Journal of Clinical Oncology, published online August 11, 2022

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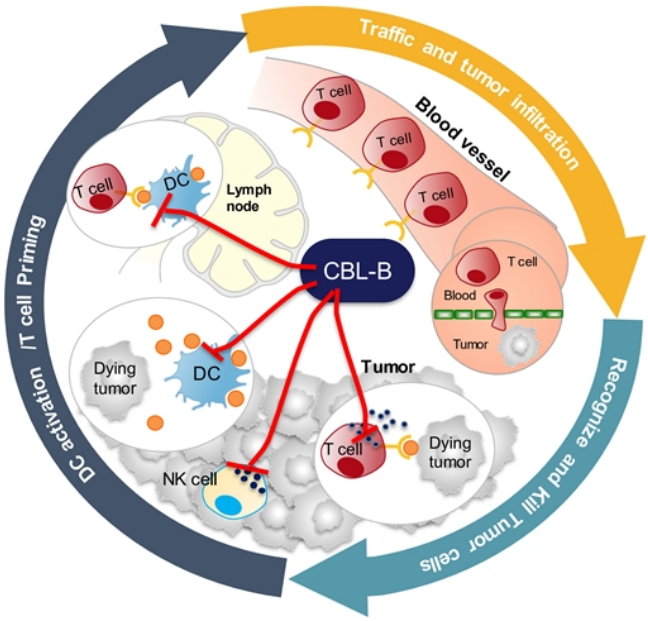
Targeting CBL-B Enhances Antitumor Response

A Master Orchestrator of the Immune System

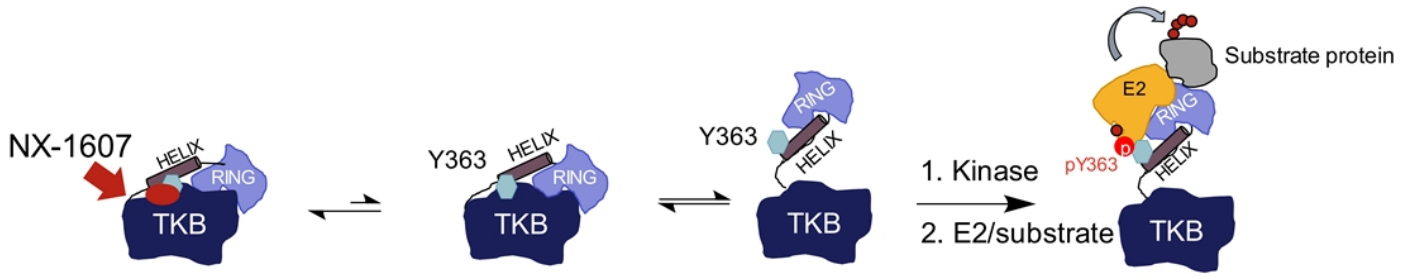
CBL-B mediated mechanisms strongly restrains a productive anti-tumor response

CBL-B inhibition increases:

- DC and NK infiltration and function
- T cell priming
- Cytotoxic T cells function
- Ability of T cells to resist tumor immunosuppressive mechanisms: Treg, MDSC, and TGF-β



NX-1607 Mechanism of Action: Intramolecular Glue



NX-1607 acts as an intramolecular glue forcing CBL-B in its folded **INACTIVE** state

Closed State **INACTIVE**

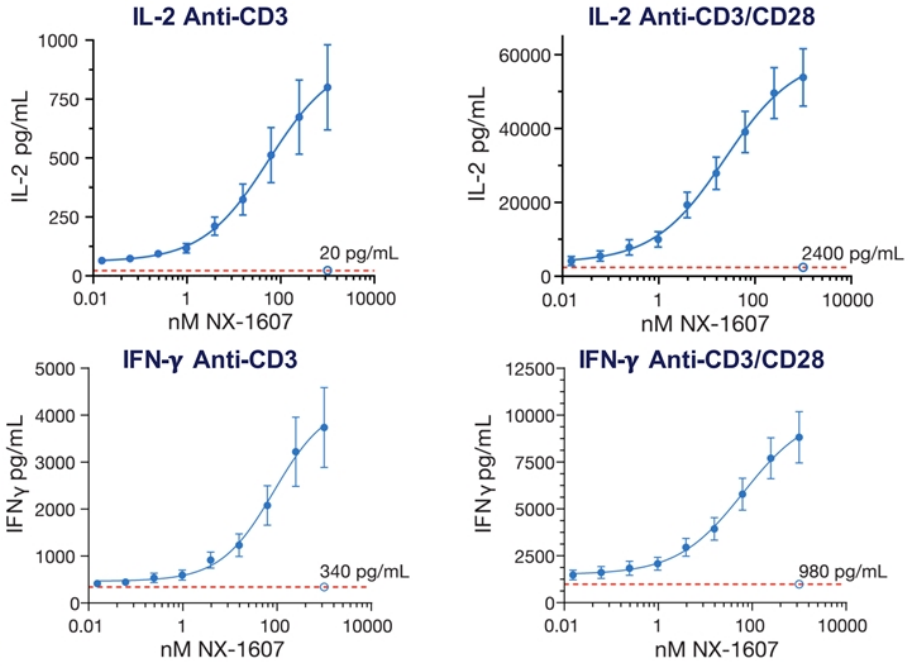
Opened State

Phosphorylation locks CBL-B in the **ACTIVE** Conformation

Immune Response

Immune Response

NX-1607 Increases IL-2 and IFN-γ Secretion in TCR Stimulated Primary Human T cells



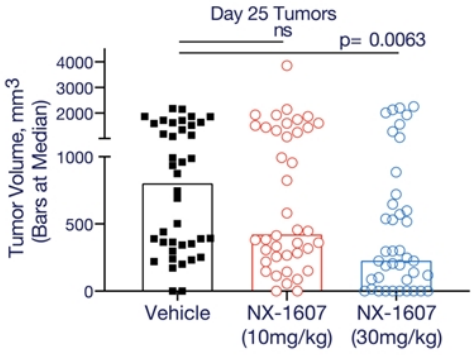
NX-1607 increases TCR stimulation-dependent production of IL-2 and IFN-γ in primary human T cells

NX-1607 has no impact in the absence of T cell stimulation as measured by proliferation, activation, or cytokine release

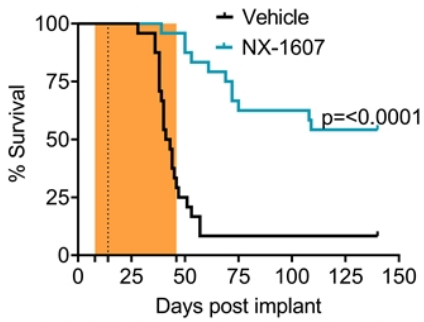
Cytokine Response
Baseline Response

Single-Agent NX-1607 Induces Antitumor Response in Multiple Models

NX-1607
Reduced Tumor Volume
Colorectal

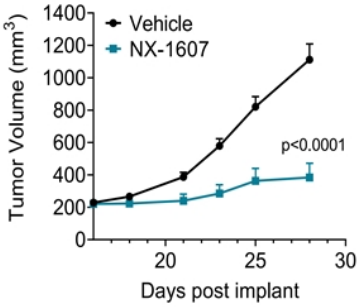


NX-1607
Prolonged Survival
Triple-Negative Breast



NX-1607 30 mg/kg day 7 to 46

NX-1607
Reduced Tumor Volume
B Cell Lymphoma

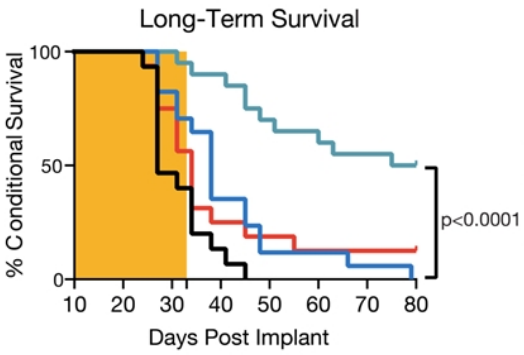


NX-1607 30 mg/kg day 16 to 28

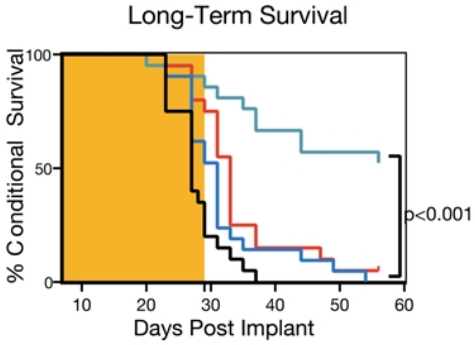
Shaded area indicates dosing period

NX-1607 and Anti-PD-1 Synergize to Enhance Anti-Tumor Effects and Survival of Mice in Multiple Tumor Models

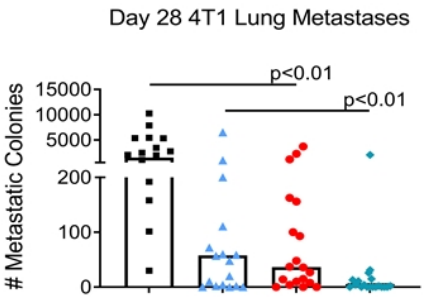
Colorectal (CT26)



Colorectal (MC38)



Triple-Negative Breast (4T1)

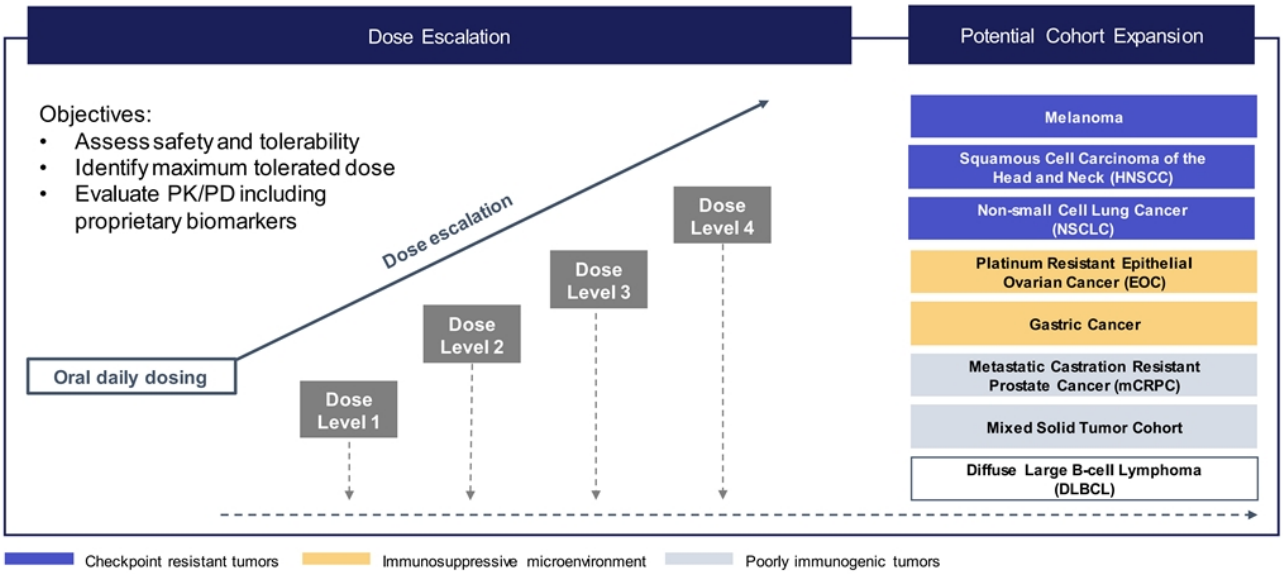


Vehicle NX-1607 anti-PD-1 NX-1607+anti-PD-1

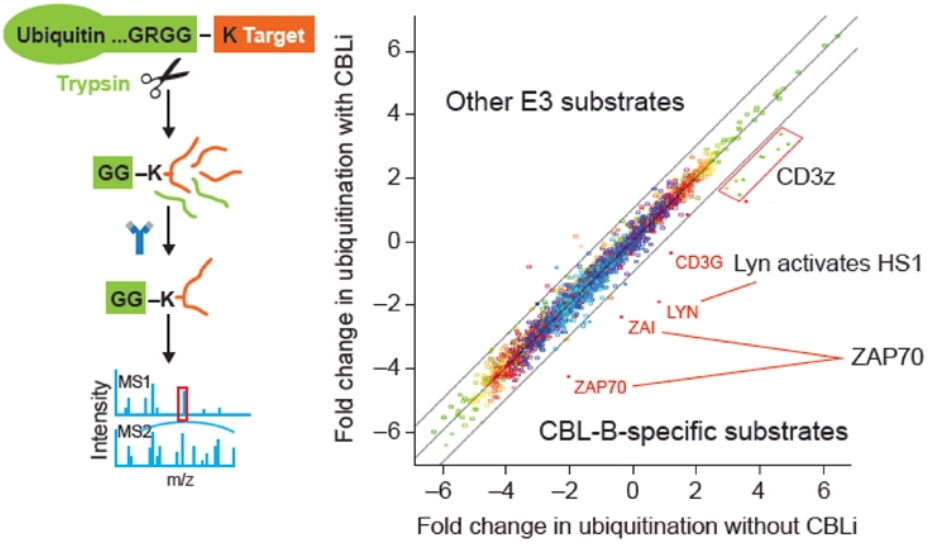
Shaded area indicates dosing period: NX-1607 (30 mg/kg, PO daily) and anti-PD-1 twice a week at 10 mg/kg dosing period

NX-1607-101: Phase 1 First-in-Human Clinical Trial Design

Phase 1 trial testing both monotherapy and combination with paclitaxel in relapsed or refractory tumors



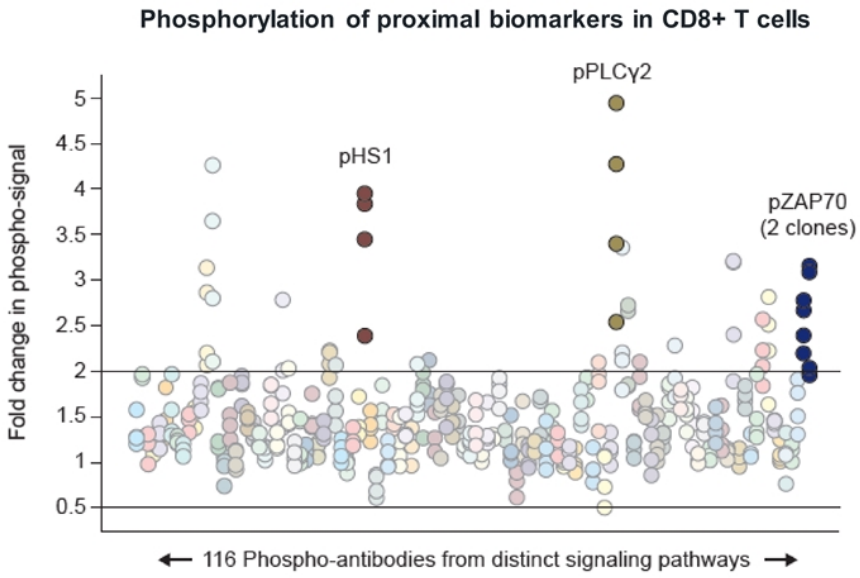
UbiScan Identified Direct CBL Substrates Within the T Cell Receptor (TCR) Signaling Cascade



Decreased signal represents direct substrates ubiquitinated by CBL-B ligase activity

Inhibiting CBL-B decreases ubiquitination of important T Cell receptor signaling molecules

Phospho-Protein Flow Cytometry Assay Identified Proximal Biomarkers



- Human PBMCs were stimulated with or without CBL-B inhibition
- Expression levels were determined for phospho-proteins downstream the TCR signaling
- Overlapping results from orthogonal assays (Ubiscan) provided confidence in proximal biomarker signals

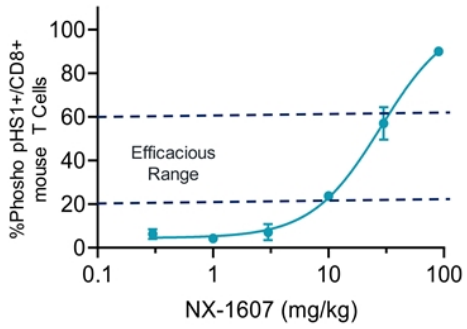
HS1: Substrate of LYN receptor, and an essential adaptor protein at the immune synapse, via VAV1

PLCγ2: Expressed in both T cells and B cells; associates with LAT and SLP-76 & becomes phosphorylated upon TCR stimulation

ZAP70: Key organizer of downstream TCR signaling

Dose Dependent Increases of CBL-B Proximal Biomarker Correlates with Antitumor Effects of NX-1607

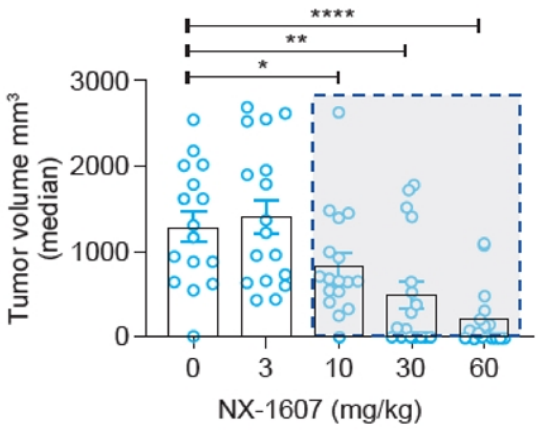
Pharmacodynamic relationship in mice following NX-1607 dosing



In vivo efficacy observed between 10-60 mpk which corresponds to ~20-60% pHS1+ CD8+ T Cells

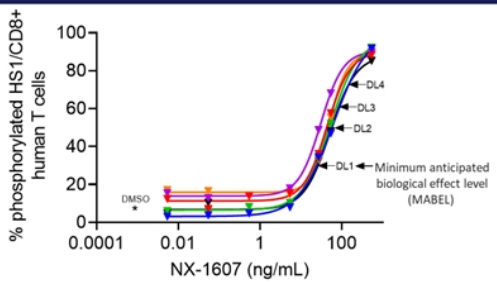
NX-1607 reduced tumor volume

A20 - B cell lymphoma model



Characterization of a Novel Biomarker and First Evidence of Target Engagement for a CBL-B Inhibitor in the Clinic

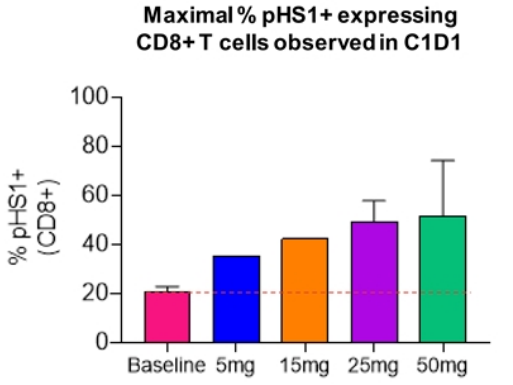
Human whole blood and dose projection modeling



Proposed dose level[a]	NX-1607 dose (mg)	Estimated % HS1+/CD8+ T cells
-1	2.5	22.2
1[b]	5	30.0
2	15	49.7
3	25	60.6
4	50	74.0

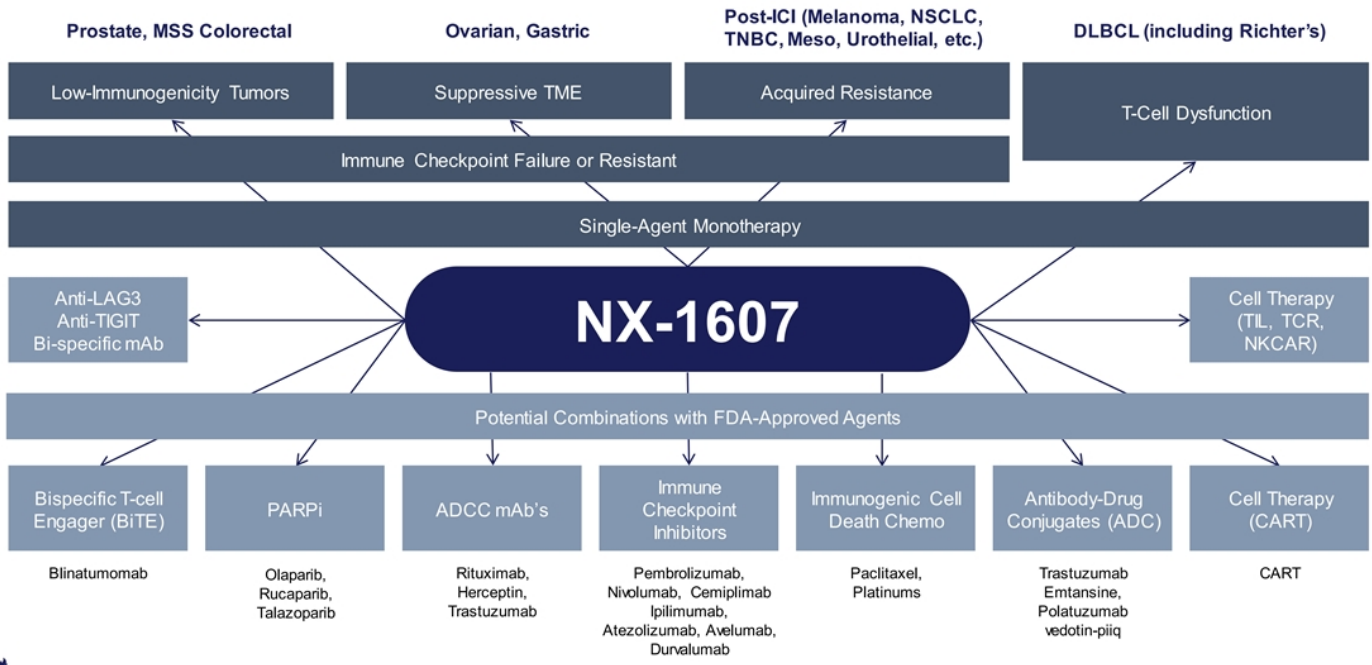
[a]Dose levels in NX-1607-101.
[b]Minimum anticipated biological effect level (MABEL).

Clinical data



	Dose level 1 5mg	Dose level 2 15mg	Dose level 3 25mg	Dose level 4 50mg
Cycle 1, N:	1	1	6	2

CBL-B Inhibition Has the Potential To Be the Small Molecule Centerpiece of Immuno-Oncology Therapy



Anticipated Upcoming Clinical Data Disclosures

Targeted Protein Degradation		Targeted Protein Elevation
NX-5948	NX-2127	NX-1607
• First disclosure of Phase 1a clinical data in patients with CLL and NHL at ASH 2023	• Updated Phase 1 clinical data in patients with CLL at ASH 2023, including more patients and longer follow up • First disclosure of Phase 1a clinical data in patients with NHL at ASH 2023	• First disclosure of Phase 1a clinical data planned for major medical conference in 2024

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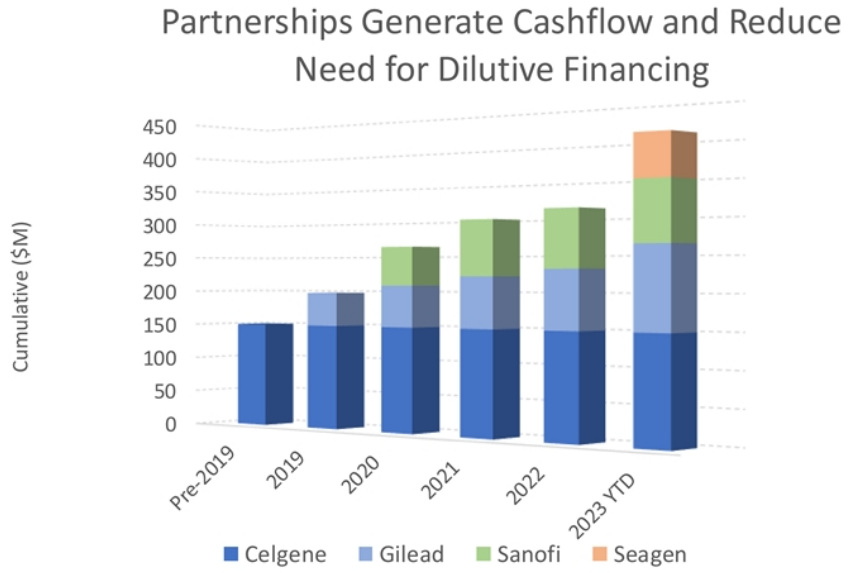
Note: All anticipated timing is based on calendar-year periods

Strong Financial Position

$329M includes funds as of August 31, 2023, plus $60M from Seagen deal



- $409 million generated through discovery partnership payments
- Potential for profit splits on up to 6 programs across three collaboration partners
- $8.2 billion in potential future payments

Thank you

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