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): January 9, 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 **94158**
(Address of Principal Executive Offices) **(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 8.01 Other Events.

As previously announced, on January 9, 2023, Nurix Therapeutics, Inc. (the "Company") will present an overview of the Company's performance in 2022 and its major goals for 2023 at the 41st Annual J.P. Morgan Healthcare Conference (the "JPM Conference"). A copy of the Company's presentation materials for the JPM Conference is attached as Exhibit 99.1 hereto and is incorporated herein by reference. Also on January 9, 2023, the Company issued the press release attached as Exhibit 99.2 hereto, which is incorporated herein by reference.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits

The following exhibits are filed herewith and this list is intended to constitute the exhibit index:

99.1	Nurix Therapeutics, Inc. presentation dated January 9, 2023.
99.2	Nurix Therapeutics, Inc. press release dated January 9, 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: January 9, 2023

By: /s/ Christine Ring
Christine Ring, Ph.D., J.D.
General Counsel

Exhibit 99.1

nurix

Leader in Targeted Protein Modulation

Nurix Therapeutics

Blazing a New Path in Medicine

Investor Presentation

January 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 ability to fund our operating activities into the fourth quarter of 2024; 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 advantages of our DELigase™ platform and drug candidates; the extent to which our scientific approach and DELigase™ platform 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) the impact of macroeconomic conditions, including as a result of the COVID-19 pandemic, increasing financial market volatility and uncertainty, inflation and rising interest rates on Nurix's clinical trials and operations; (v) Nurix’s ability to protect intellectual property and (vi) 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, 2022, 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 Nurix’s own internal estimates and research. While Nurix 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.

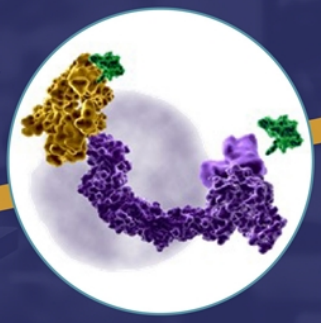
Nurix Drugs Engage Ligases for the Treatment of Cancer

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)

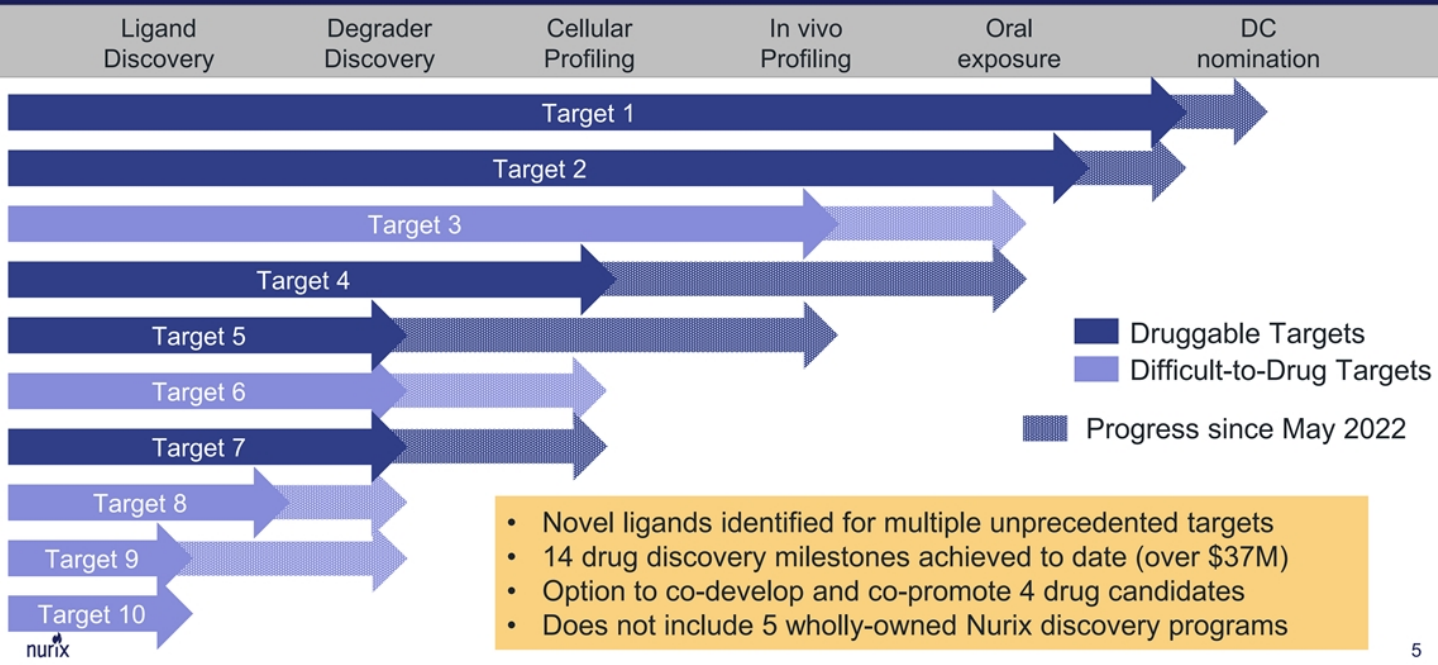
Ubiquitin is ligated to target proteins to tag them for degradation by the proteasome

Nurix Is Advancing Four Wholly Owned Clinical Programs with a Deep Pipeline of Proprietary and Partnered Novel Targets

MOA	Drug program	Target/delivery	Therapeutic area	Preclinical	Phase 1	Phase 2	Phase 3
TPD	**NX-2127** Degrader	BTK-IKZF *Oral*	B-cell malignancies			✓ Advanced to Ph 1b in CLL ✓ Efficacy established in CLL ✓ Single agent CR in DLBCL	
TPD	**NX-5948** Degrader	BTK *Oral*	B-cell malignancies			✓ Dosed first patient in U.K. ✓ Demonstrated BTK degradation ✓ IND cleared for U.S. enrollment	
TPE	**NX-1607** Inhibitor	CBL-B *Oral*	Immuno-Oncology			✓ Demonstration of CBL-B inhibition with novel biomarker ✓ IND cleared for U.S. enrollment	
TPE	**DeTIL-0255** Cell therapy	*Ex vivo CBL-B inhibition*	Gynecologic malignancies			✓ Dosed first patient ✓ Completed safety run-in	
TPM	Wholly owned & partnered	15 targets	Multiple				

Progress since January 2022

Significant Advancement of the Collaboration Pipeline during 2022, Including Targets Considered Undruggable



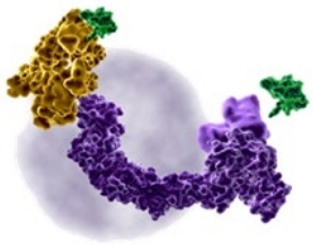
- Novel ligands identified for multiple unprecedented targets
- 14 drug discovery milestones achieved to date (over $37M)
- Option to co-develop and co-promote 4 drug candidates
- Does not include 5 wholly-owned Nurix discovery programs

A First-In-Class Franchise of BTK Degraders:
NX-2127 & NX-5948

NX-2127

BTK DEGRADATION
& IMMUNOMODULATION

- Positive clinical activity in CLL patients, including responses in patients with BTK or BCL2 mutations
- Active in the clinic against multiple BTK inhibitor-resistant mutations
- Complete response observed in a patient with DLBCL
- Phase 1b cohort expansion for CLL patients is ongoing
- Dose exploration is ongoing for patients with NHL



NX-5948

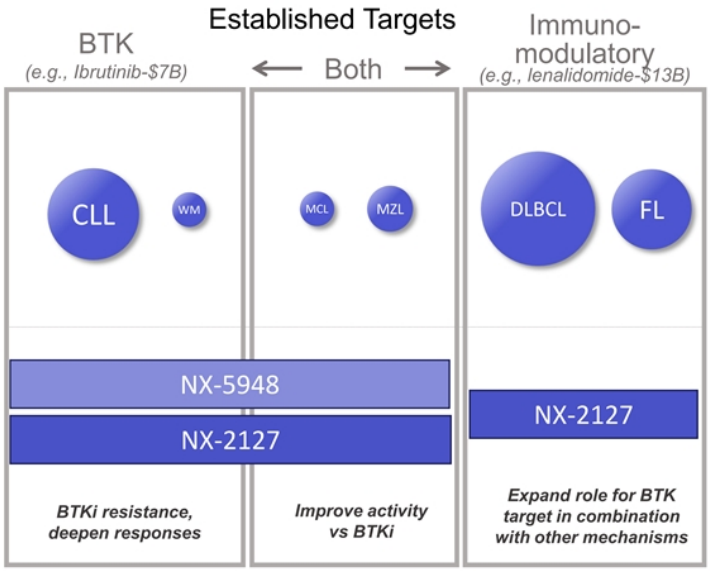
BTK DEGRADATION

- Clinical evidence of potent BTK degradation in all patients tested
- Active in vitro against multiple BTK inhibitor-resistant mutations
- Crosses blood brain barrier and degrades BTK in brain-resident lymphoma cells and microglia in animal models
- Activity in multiple models of autoimmune disease
- Phase 1a dose escalation trial ongoing in U.K. and IND accepted in the U.S.

Nurix BTK Degrader Franchise: Two BTK Degraders to Cover the Landscape of B-cell Malignancies

NX-2127 for BTK inhibitor resistance in CLL and for aggressive NHL

NX-5948 may be the degrader of choice for single-target therapy with potential in autoimmunity



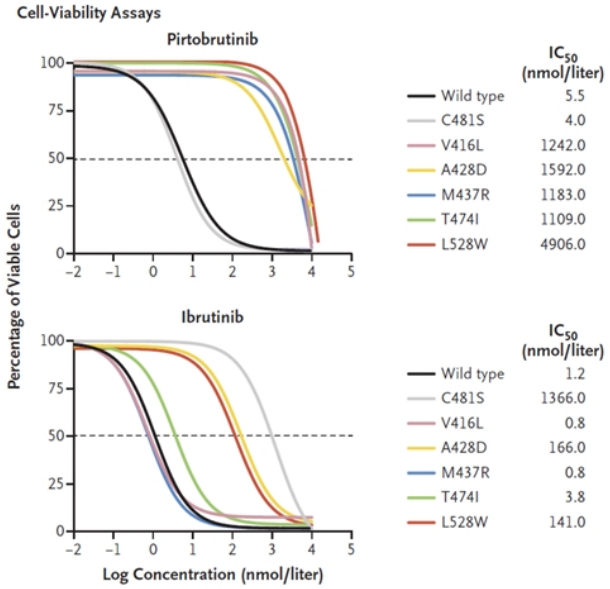
Size of bubble=annual incidence in US and EU

B-CELL MALIGNANCIES ANNUAL INCIDENCE (US & EU)	
Chronic Lymphocytic Leukemia (CLL)	39,700
Diffused Large B-Cell Lymphoma (DLBCL)	55,100
Follicular Lymphoma (FL)	26,200
Mantle cell lymphoma (MCL)	6,200
Marginal Zone Lymphoma (MZL)	10,700
Waldenstrom's macroglobulinemia (WM)	6,300

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

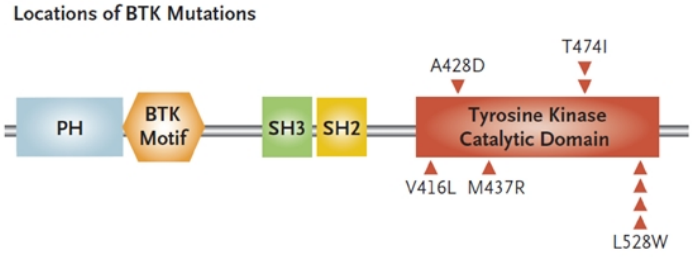
BTK, Bruton tyrosine kinase; DLBCL, Diffuse large B cell lymphoma; CLL, Chronic lymphocytic leukemia, SLL, small lymphocytic lymphoma; MCL, Mantle cell lymphoma; WM, Waldenstrom's macroglobulinemia; MZL, Marginal zone lymphoma; FL, Follicular lymphoma; NHL, non-Hodgkin lymphoma

Resistance to Noncovalent BTK Inhibitors Presents a New and Growing Challenge to Treatment



The NEW ENGLAND JOURNAL of MEDICINE

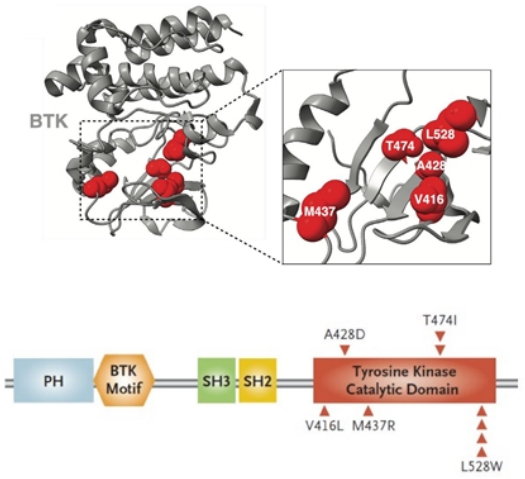
"Our data suggest potential new therapeutic approaches to overcome the newly described BTK inhibitor resistance mechanisms. For example, these data provide a rationale for therapies aimed at addressing the potential scaffold function of BTK rather than inhibiting BTK kinase activity."



Source: Wang et al, N Engl J Med 2022;386:735-43

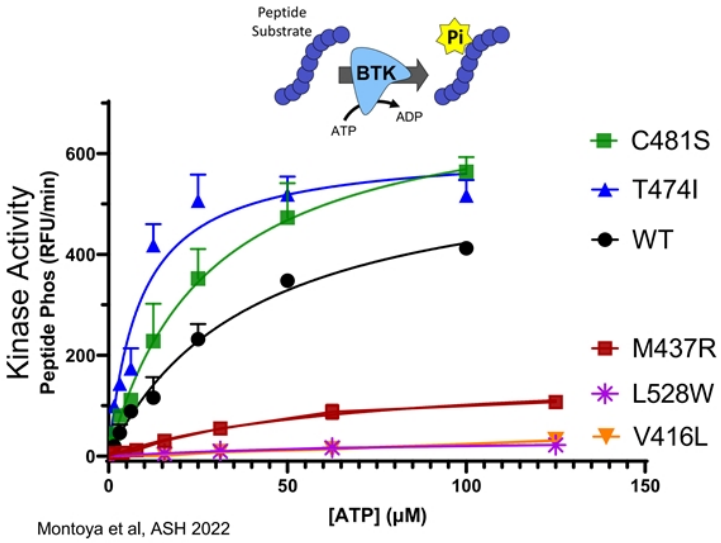
Nurix Degraders Directly Address Emerging Resistance Mutations and BTK Scaffolding Activity

Treatment with BTK inhibitors is changing the resistance landscape



Wang E, et al. NEJM 2022

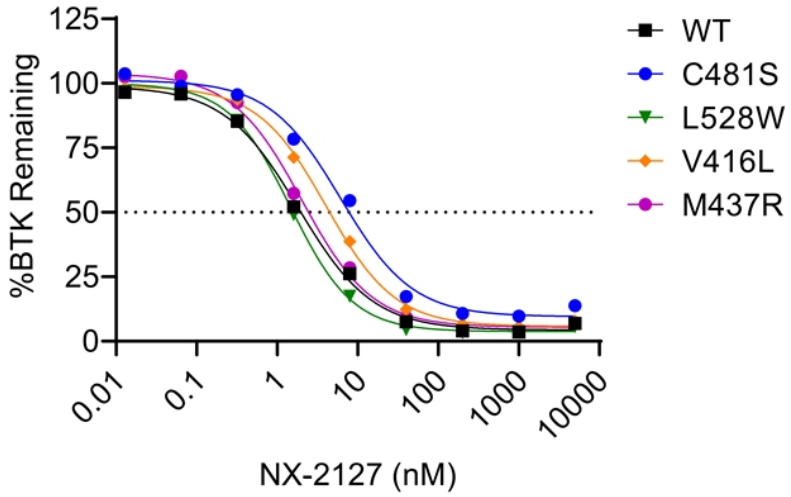
Many of the mutations that confer resistance to BTK inhibitors lack kinase activity



Montoya et al, ASH 2022

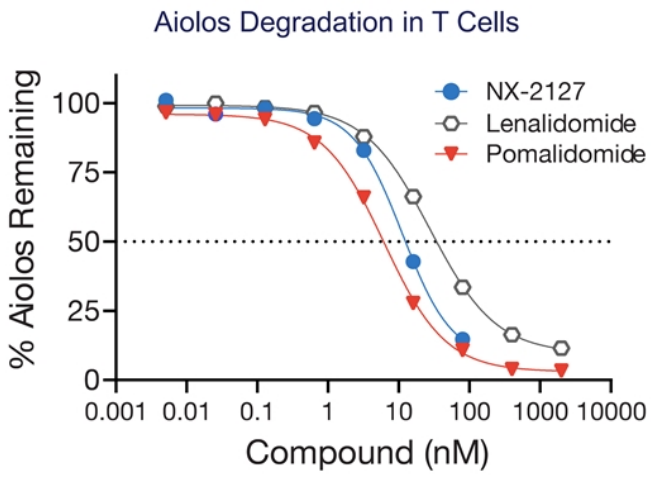
NX-2127 is Active Against Both Wildtype and Mutant BTK

Potential to treat patients who failed both covalent and non-covalent BTK inhibitors



NX-2127 is capable of degrading not only C481S, but also the novel BTK mutations observed post treatment with pirtobrutinib

NX-2127 is a Dual Acting Agent That Also Degrades Immunomodulatory Cereblon Neosubstrate Aiolos

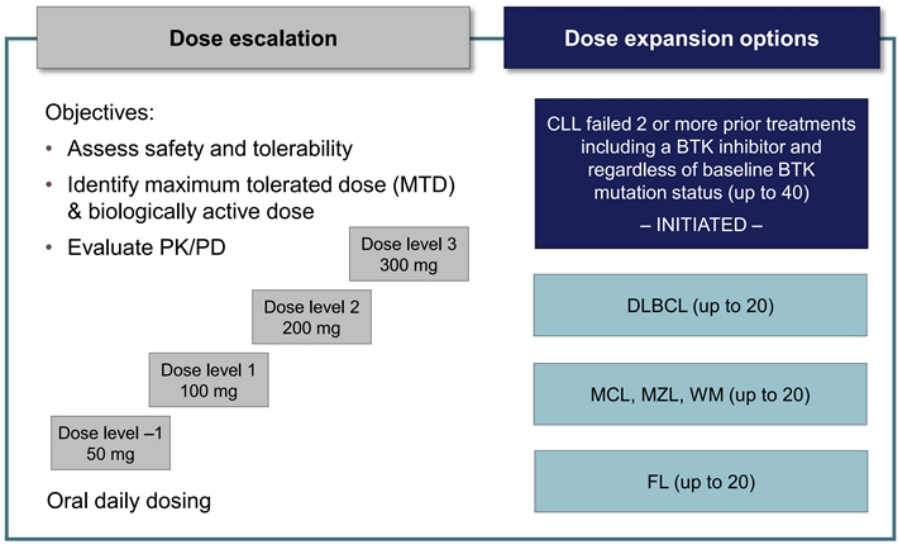


NX-2127 degradation of Aiolos in human T cells occurs at a similar potency to lenalidomide and pomalidomide

- Activity of NX-2127 is pegged to approved agents with well-established efficacy and safety
- Dual activity potentially addresses alternative resistance mechanism in CLL
- Emerging clinical data supports pathway combination approach in ABC-subtype DLBCL
- Dual mechanism shows strong benefit in MCL where both classes of agents are approved single agents

NX-2127-001: Trial Design

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



- CLL Phase 1b expansion cohort ongoing at 100 mg dose
  - MTD not established
  - 100 mg dose chosen as expansion dose based on PD, clinical activity and safety profile
- Phase 1a dose escalation is ongoing at 200 mg and 300 mg doses for patients with NHL (e.g., DLBCL, MCL, MZL, WM, FL)

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; **PD,** pharmacodynamics; **PK,** pharmacokinetics; **WM**, Waldenstrom's macroglobulinemia

Baseline Characteristics

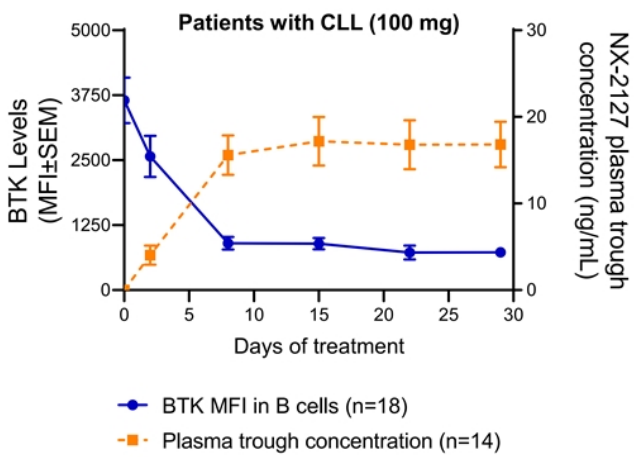
Elderly population with multiple prior lines of targeted therapies and acquired mutations

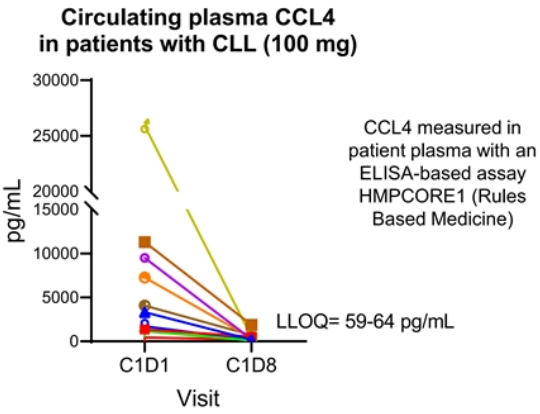
Characteristics	CLL (n=23)	Overall population (N=36)
Median age, years (range)	75 (61–90)	75 (50–92)
Female, n (%)	9 (39.1)	13 (36.1)
Male, n (%)	14 (60.9)	23 (63.9)
Lines of prior therapy, median (range)	5 (2–11)	4 (2–11)
BTKi, n (%)	23 (100)	31 (86.1)
Pirtobrutinib, n (%)	8 (34.8)	11 (30.6)
BTKi and 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





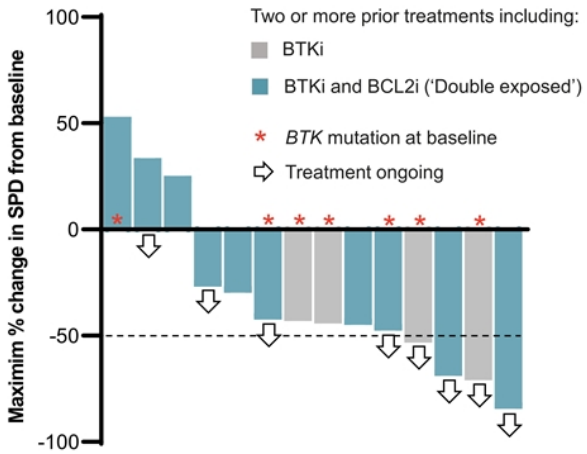
- Daily treatment with NX-2127 resulted in a rapid and sustained suppression of BTK (CD19+) as measured in patient whole blood using a flow cytometry assay. BTK suppression target of 80% reached consistently (data not shown here)
- Robust decrease of plasma CCL4 by Cycle 1 Day 8 and suppression was maintained 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

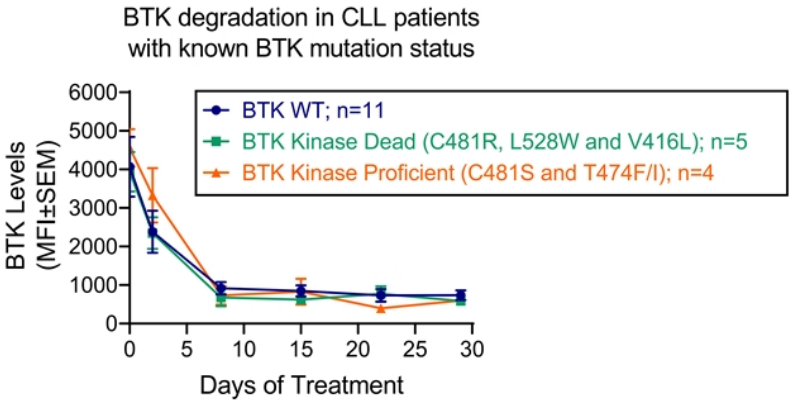


***One patient, not shown above, with prior BTKi and BCL2i treatment and with a *BTK* mutation detected at baseline, had no nodal disease at baseline. Their treatment is ongoing with a PR**

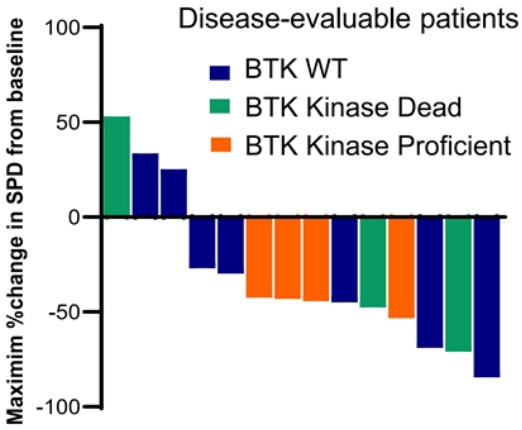
BCL2i, B-cell lymphoma-2 inhibitor; **BTK**, Bruton's tyrosine kinase; **BTKi**, BTK inhibitor; **CR**, complete response; **CRi**, complete response with incomplete count recovery; **NE**, not evaluable; **PD**, progressive disease; **PR**, partial response; **SD**, stable disease

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

Two Heavily Pre-Treated Patients with Non-GCB DLBCL Enrolled in NX-2127 Phase 1 Dose-Escalation

	Patient #1	Patient #2
Subtype	Non-GCB (ABC subtype) Double-hit, BCL2/BCL6	Non-GCB (ABC subtype)
Dose	100 mg	300 mg
Time on Study	3.5 months	5 months and ongoing
Priors	4	4
Response(s)	Stable Disease (SD) at 8w → Progressive Disease (PD)	Complete Response (CR)* at 8w confirmed at 16w

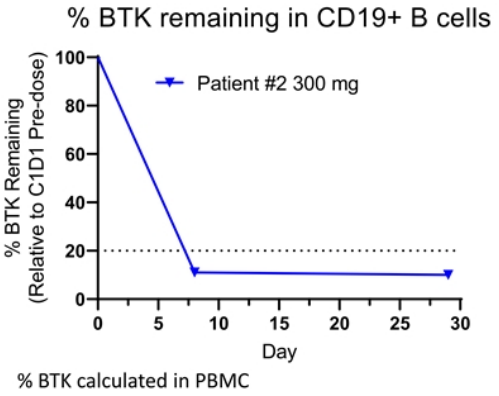
Patient #2	Baseline demographic and disease characteristics
Age; Relevant medical history	84; aortic regurgitation, diastolic dysfunction, aspergillosis sinus infection
Cancer Diagnosis	1988: Waldenstrom's macroglobulinemia (WM) 2015: Diffuse large B-cell lymphoma (DLBCL) ABC subtype
Prior treatments for DLBCL	2015: Rituximab + CHOP followed by focal axillary irradiation 2017: Rituximab + ICE 2018: Rituximab, mogamulizumab (anti-CCR4), and magrolimab (anti-CD47) 2019: Rituximab, ibrutinib, and lenalidomide (RIL)
Disease features at study entry	Stage IV, MYD88 mutated and CXCR4 mutated
Time on study	Ongoing, Cycle #6 (5 months)

nurix

Data as reported October 26, 2022

*CR per Lugano criteria

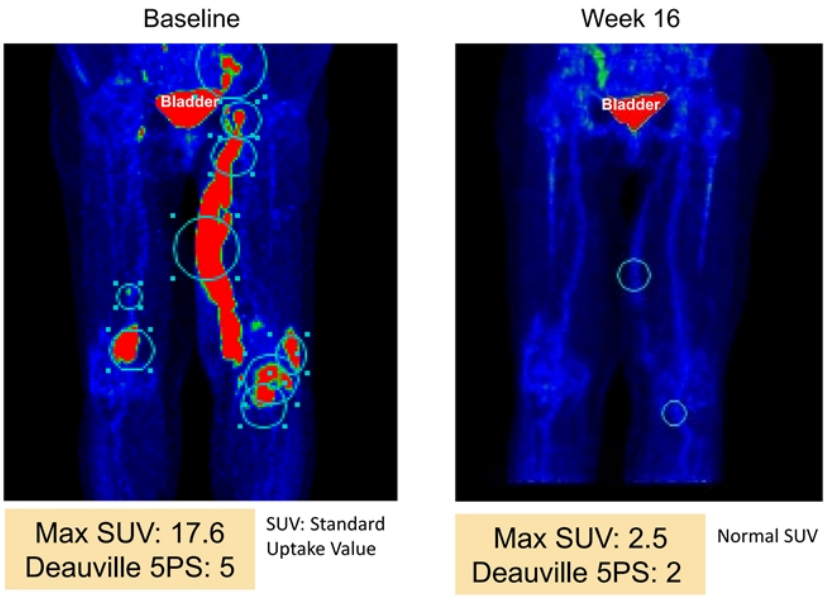
Rapid BTK Degradation and Confirmed Complete Response Following NX-2127 Therapy



% BTK calculated in PBMC

Significant Ikaros and Aiolos degradation also confirmed by day 8

FDG-PET CT Scan Disease Assessment



Max SUV: 17.6
Deauville 5PS: 5

SUV: Standard Uptake Value

Max SUV: 2.5
Deauville 5PS: 2

Normal SUV

- Complete response at first assessment (Week 8) and confirmed at subsequent assessment (Week 16)
- Safety: No DLT or SAE. Grade 3 neutropenia without infection, resolved with G-CSF. No Rx interruptions.

Data as reported October 26, 2022

NX-2127: First-in-Class BTK Degrader Demonstrates Early Signs of Meaningful Clinical Activity in Both CLL and NHL

Chronic lymphocytic leukemia (CLL)

- Objective responses observed in CLL patients who failed a median of 6 prior lines of therapy including patients who failed BTK inhibitors and BCL2 inhibitors
- Objective responses observed in patients whose tumors harbor BTK mutations known to cause resistance to both covalent and non-covalent BTK inhibitors

Next steps: Enrollment in Phase 1b is ongoing with clinical update planned for H2 2023

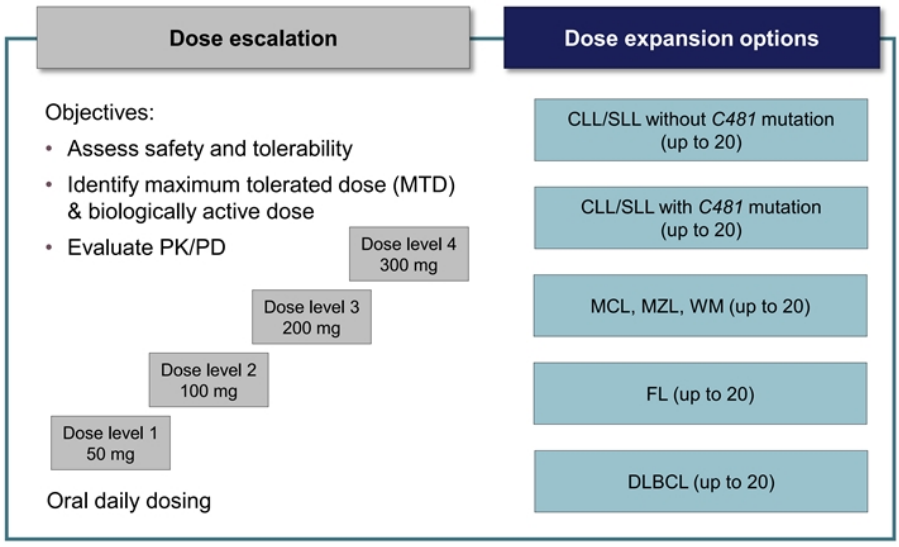
Non-Hodgkin lymphoma (NHL)

- Rapid and complete response in a patient with advanced relapsed/refractory non-GCB DLBCL following four prior lines of therapy

Next steps: Enrollment in Phase 1a is ongoing at the 200 mg and 300 mg doses in patients with NHL with clinical update planned for H2 2023.

NX-5948-301: Trial Design

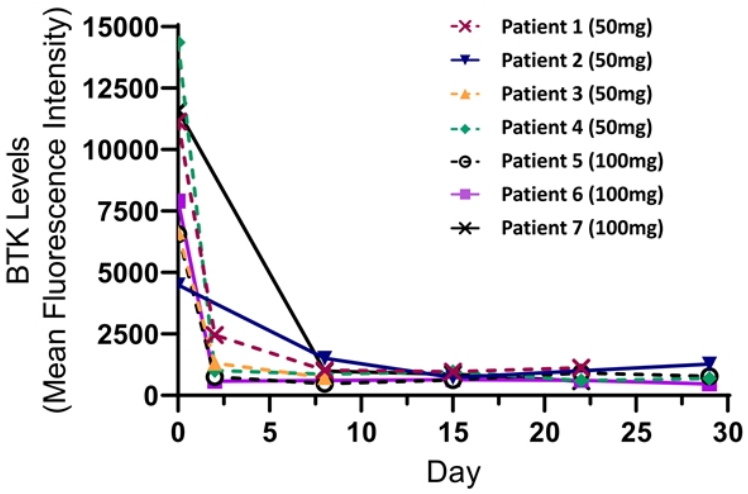
Phase 1 trial in adults with relapsed/refractory B-cell malignancies



- Phase 1a dose escalation is ongoing at clinical sites in the U.K.
- Plans to initiate U.S. sites in early 2023

CLL, chronic lymphocytic leukemia; **DLBCL**, diffuse large B-cell lymphoma; **FL**, follicular lymphoma; **MCL**, mantle cell lymphoma; **MZL**, marginal zone 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

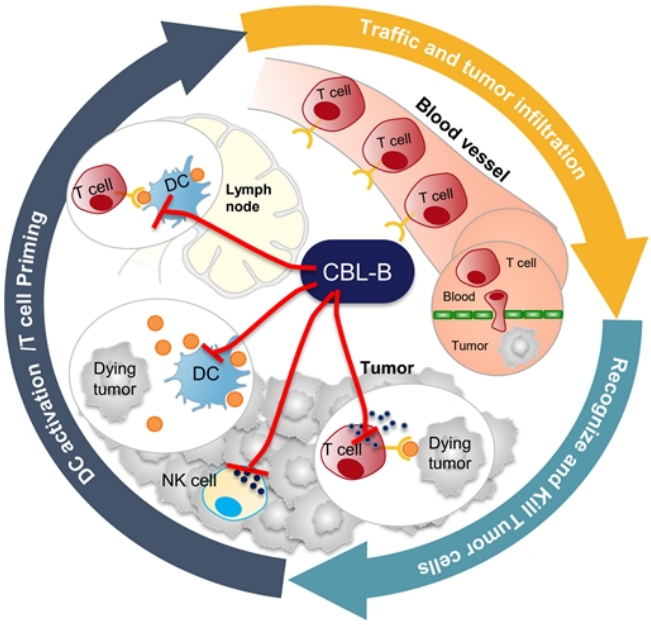
Targeting CBL-B Enhances Antitumor Response

A Master Orchestrator of the Immune System

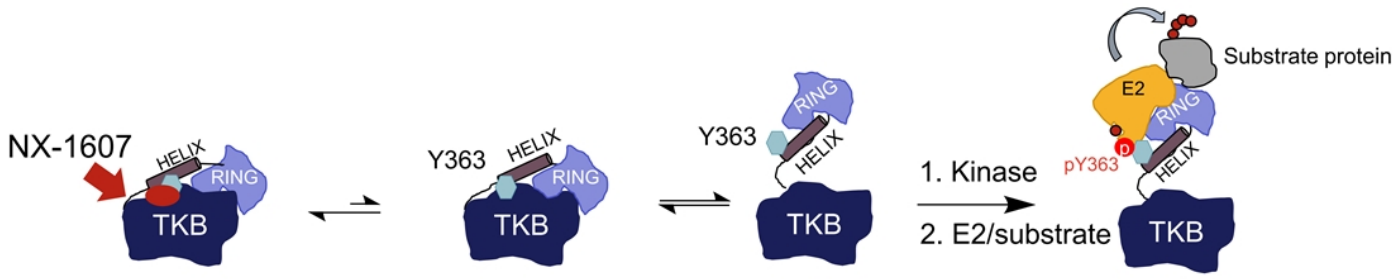
CBL-B mediated mechanisms strongly restrains a productive anti-tumor response

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**

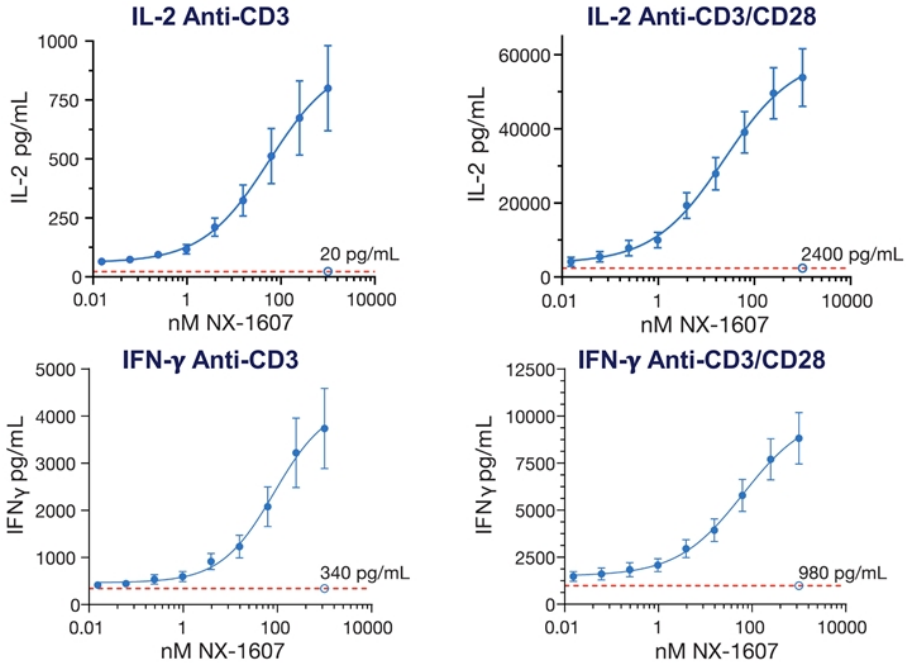
Opened State

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



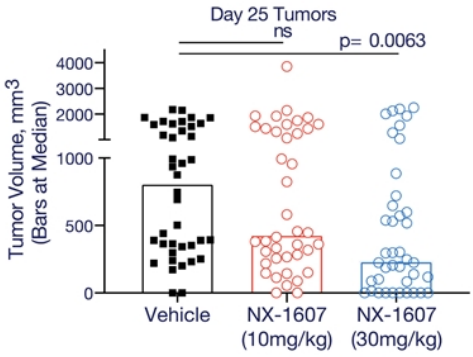
NX-1607 increases TCR stimulation-dependent production of IL-2 and IFN-γ in primary human T cells

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

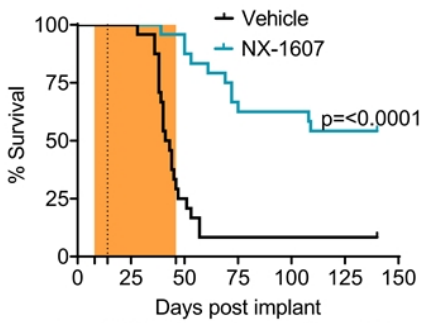
Cytokine Response
Baseline Response

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

NX-1607
Reduced Tumor Volume
Colorectal

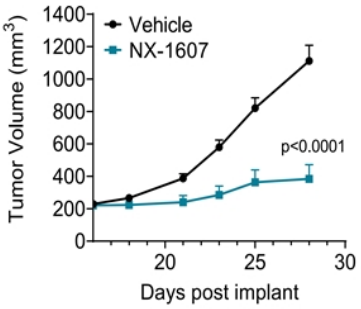


NX-1607
Prolonged Survival
Triple-Negative Breast



NX-1607 30 mg/kg day 7 to 46

NX-1607
Reduced Tumor Volume
B Cell Lymphoma

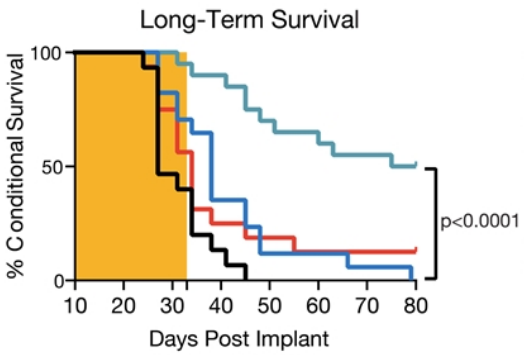


NX-1607 30 mg/kg day 16 to 28

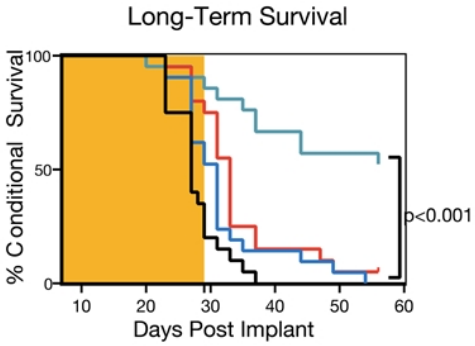
Shaded area indicates dosing period

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

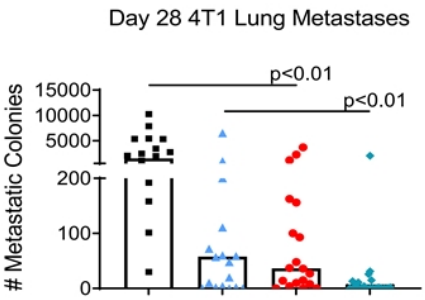
Colorectal (CT26)



Colorectal (MC38)



Triple-Negative Breast (4T1)

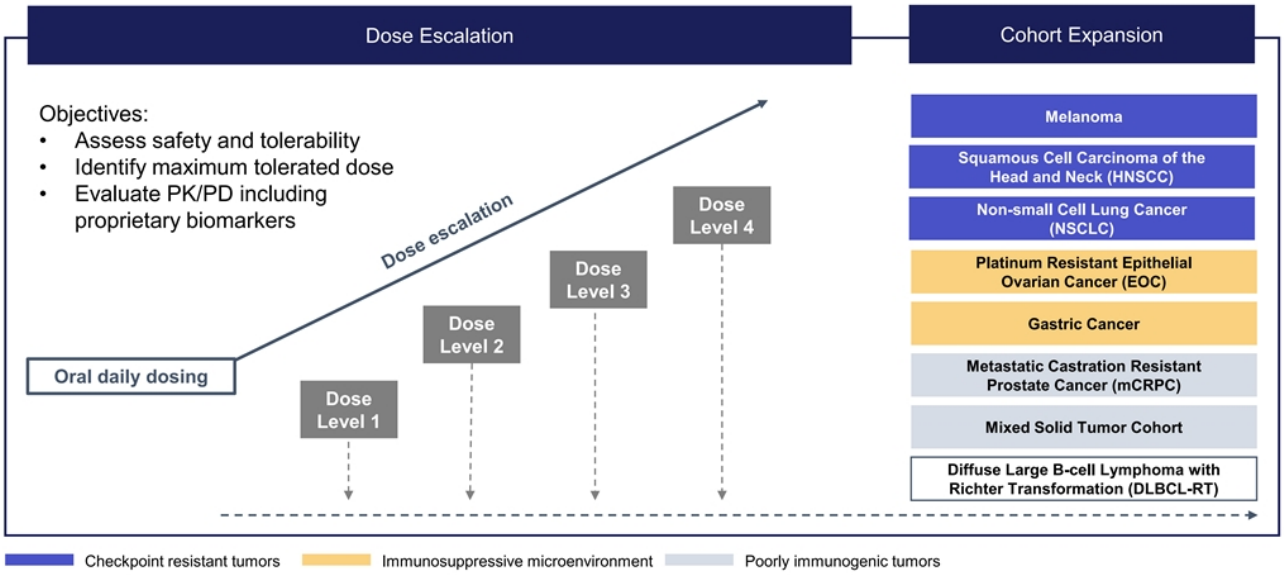


Vehicle NX-1607 anti-PD-1 NX-1607+anti-PD-1

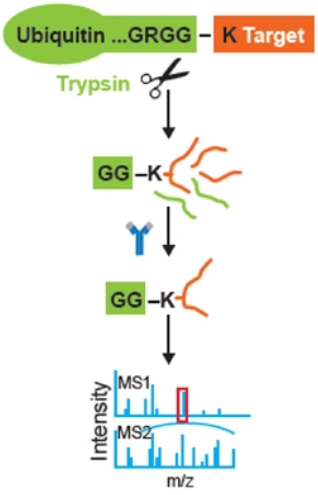
Shaded area indicates dosing period: NX-1607 (30 mg/kg, PO daily) and anti-PD-1 twice a week at 10 mg/kg dosing period

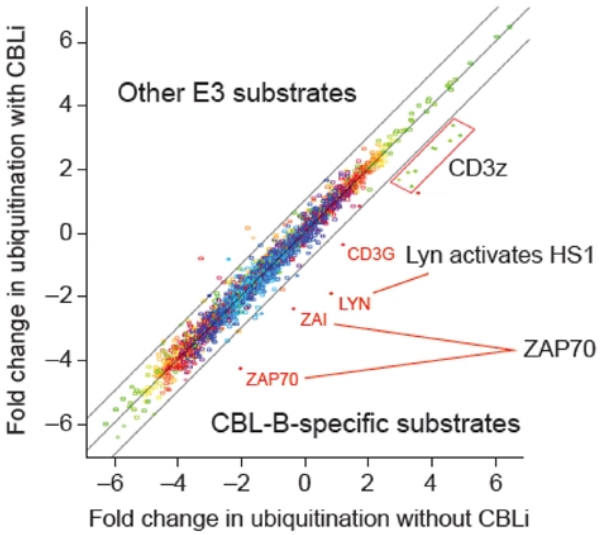
NX-1607-101: Phase 1 first-in-human clinical trial design

Two-Part Phase 1 Monotherapy Trial of NX-1607 in Relapsed or Refractory Tumors



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

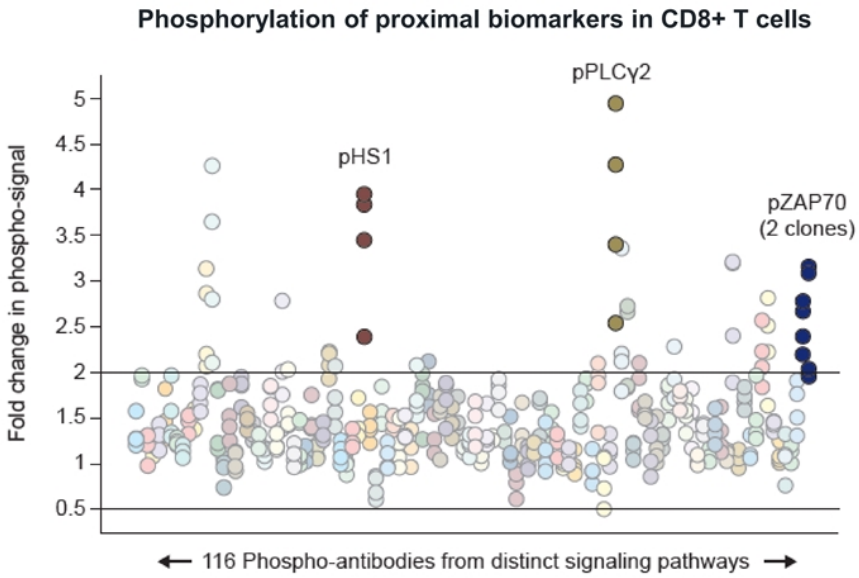




Decreased signal represents direct substrates ubiquitinated by CBL-B ligase activity

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

Phospho-Protein Flow Cytometry Assay Identified Proximal Biomarkers



- Stimulated human PBMCs with or without CBL-B inhibition
- Cells were stained with a panel of phospho-specific antibodies for proteins downstream the TCR signaling
- Expression levels were assessed by flow cytometry
- Overlapping results from orthogonal assays (Ubiscan) provided confidence in proximal biomarker signals

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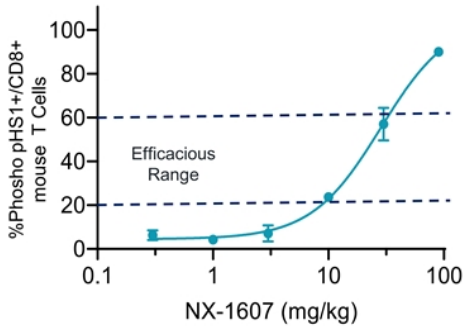
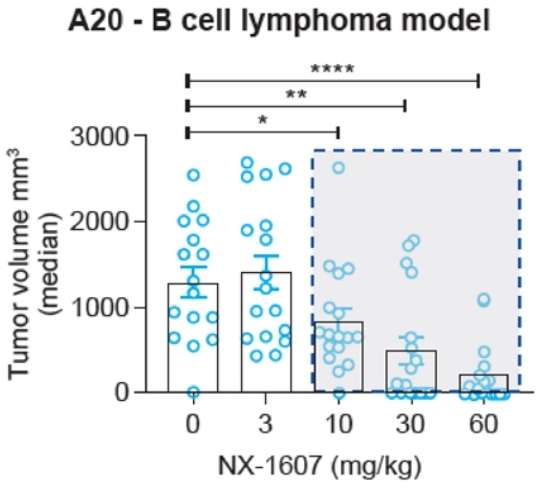
Dose Dependent Increases of CBL-B Proximal Biomarker Correlates with Antitumor Effects of NX-1607

Pharmacodynamic relationship in mice following NX-1607 dosing



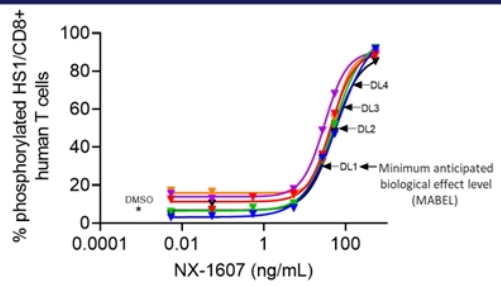
In vivo efficacy observed between 10-60 mpk which corresponds to ~20-60% pHS1+ CD8+ T Cells

NX-1607 reduced tumor volume



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

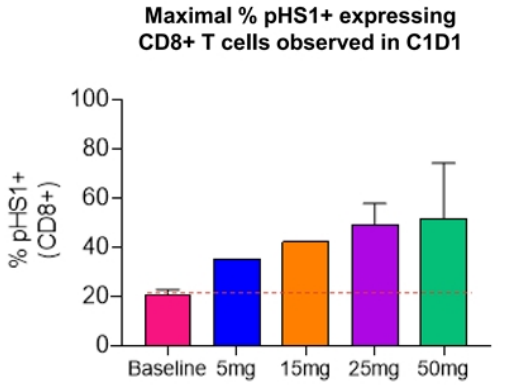
Human whole blood and dose projection modeling



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

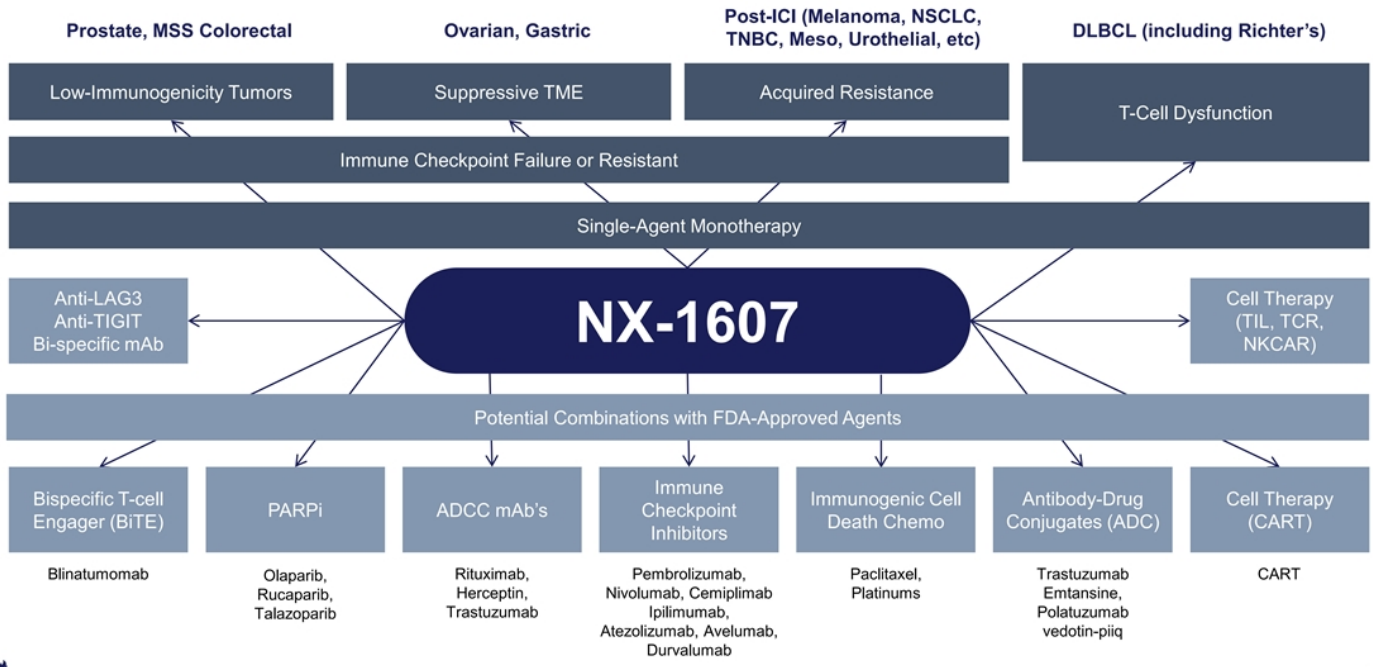
[a]Dose levels in NX-1607-101.
[b]Minimum anticipated biological effect level (MABEL).

Clinical data

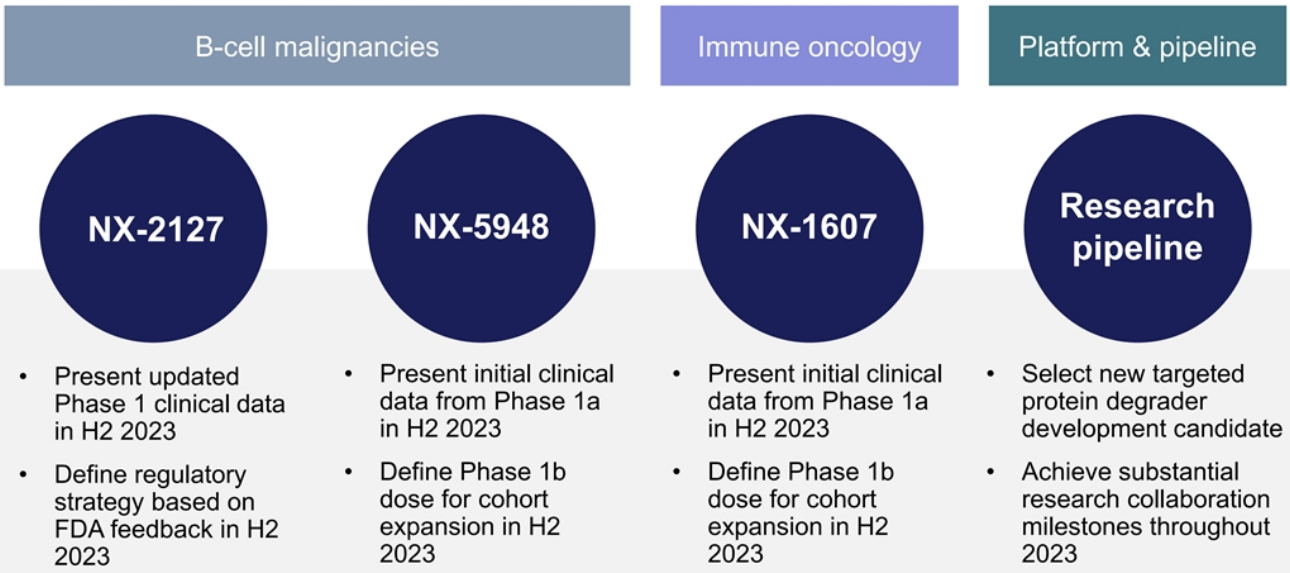


	Dose level 1 5mg	Dose level 2 15mg	Dose level 3 25mg	Dose level 4 50mg
Cycle 1, N:	1	1	6	2

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



Defining Success in 2023

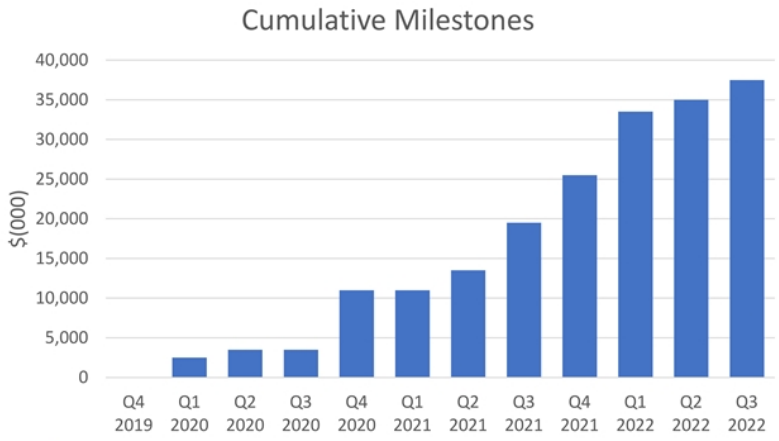


Note: All anticipated timing is based on calendar-year periods

Strong Financial Position

$414M in cash and investments as of August 31, 2022

- Funded through key readouts for all clinical programs
- Cash runway into Q4 2024 excluding any future potential milestones from collaborations



R&D collaboration details:

- Gilead $45M upfront and up to $2.3B in development, regulatory and sales milestones plus royalties
- Sanofi $77M upfront and expansion payments and up to $2.5B in development, regulatory and sales milestones plus royalties
- Nurix option for 50/50 U.S. co-development for two drug candidates per partner

Thank you

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Nurix Therapeutics Advances Promising Targeted Protein Modulation Pipeline and Outlines 2023 Strategic Priorities

Nurix leads targeted protein modulation field with its wholly owned clinical stage programs and two strategic collaborations fueled by its DELigase platform

Clinical data updates planned for Nurix's two first-in-class BTK degraders in 2023

Clinical data update planned for Nurix's first-in-class CBL-B inhibitor in 2023

Significant progress made across deep pipeline of preclinical candidates

San Francisco, CA, January 9, 2023 — Nurix Therapeutics, Inc. (Nasdaq: NRIX), a clinical-stage biopharmaceutical company developing targeted protein modulation drugs designed to treat patients with hematologic malignancies and solid tumors, today outlined key objectives and anticipated milestones for 2023 and provided an overview of recent progress in a presentation at the 41st Annual J.P. Morgan Healthcare Conference.

"2022 was a landmark year for Nurix as we progressed our wholly owned pipeline and presented clinical data from our lead program, demonstrating the promise of targeted protein modulation drugs for patients with advanced B cell malignancies," said Arthur T. Sands, M.D., Ph.D., president and chief executive officer of Nurix. "Most importantly, we have shown that targeted protein degradation can overcome resistance mutations in patients with chronic lymphocytic leukemia and also shows promise in non-Hodgkin lymphoma. We are well-positioned to maintain the momentum of the past year into 2023 and beyond."

2023 Goals and Catalysts

Pipeline

- **Planned updates to Nurix's wholly owned clinical programs as described below:**
  - **NX-2127**: Nurix's lead drug candidate from its protein degradation portfolio, NX-2127, is a novel orally bioavailable bifunctional molecule that degrades Bruton's tyrosine kinase (BTK) and cereblon neosubstrates Ikaros (IKZF1) and Aiolos (IKZF3). Nurix expects to provide a clinical update in H2 2023 from its ongoing Phase 1a/1b clinical trial of NX-2127 in adults with relapsed or refractory B cell malignancies. Nurix also anticipates defining a regulatory strategy for NX-2127 in H2 2023 based on emerging clinical data and feedback from the U.S. Food and Drug Administration (FDA). Additional information on the clinical trial can be accessed at www.clinicaltrials.gov (NCT04830137).

- **NX-5948**: Nurix's second drug candidate from its protein degradation portfolio, NX-5948, is an orally bioavailable BTK degrader that, differentiated from NX-2127, has been designed to lack cereblon immunomodulatory activity. Nurix is evaluating NX-5948 in a Phase 1 clinical trial in adults with relapsed or refractory B cell malignancies and expects to present initial clinical data from the Phase 1a portion of the study in H2 2023. In addition, Nurix expects to define a dose for Phase 1b cohort expansion in H2 2023. Additional information on the clinical trial can be accessed at www.clinicaltrials.gov (NCT05131022).
- **NX-1607**: Nurix's lead drug candidate from its E3 ligase inhibitor portfolio, NX-1607, is an orally bioavailable inhibitor of Casitas B-lineage lymphoma proto-oncogene (CBL-B) for immuno-oncology indications including a range of solid tumor types and lymphoma. Nurix is evaluating NX-1607 in an ongoing, Phase 1 trial in adults with a variety of oncology indications and expects to present clinical data from the Phase 1a stage of the study and to define a dose for Phase 1b cohort expansion in H2 2023. Additional information on the clinical trial can be accessed at www.clinicaltrials.gov (NCT05107674).

- **New drug candidate:** Nurix expects to select a new targeted protein degrader development candidate in 2023.
- **Research milestones:** Nurix expects to achieve substantial research collaboration milestones throughout 2023 from its existing collaborations with Gilead Sciences and Sanofi.

2022 Accomplishments and Business Highlights

Pipeline

- **Presented clinical data for and announced progress of four wholly owned programs generated by its proprietary DELigase platform at key oncology-focused medical meetings throughout 2022.**
  - **NX-2127:** Two oral presentations at the American Society of Hematology (ASH) Annual Meeting in December described positive data for NX-2127 in patients with CLL and new scientific findings supporting the rationale for BTK degradation as a novel mechanism of action to address the current and emerging unmet need in patients whose cancer has relapsed following multiple prior lines of therapy. Specifically, data from the ongoing Phase 1 trial of NX-2127 demonstrated clinically meaningful objective responses independent of prior treatments or BTK mutational status. A second presentation described the variety of emerging BTK inhibitor resistance mutations, all of which remain susceptible to BTK degradation.

- **NX-5948:** Initial PK/PD data from the Phase 1 clinical trial of NX-5948 were presented at an analyst event at the ASH Annual Meeting in December, demonstrating early evidence of target engagement with rapid and sustained BTK degradation in all patients and no evidence of immunomodulatory-associated adverse events. An archived webcast of the event can be accessed via the Events and Presentations page of the Investor section of the Nurix website. Nurix subsequently announced clearance from the FDA for its Investigational New Drug (IND) application to expand the ongoing Phase 1 clinical program for NX-5948 into sites in the United States.
- **NX-1607:** At the Society for Immunotherapy of Cancer (SITC) Annual Meeting in November, Nurix presented six posters focused on its CBL-B inhibitor programs, including initial biomarker data demonstrating successful target engagement of CBL-B in its ongoing Phase 1 clinical trial of orally dosed NX-1607. The development and implementation of a novel proprietary biomarker indicate that the doses of NX-1607 currently being tested in the Phase 1 trial are achieving biologic activity anticipated to be within the therapeutic range based on the results from relevant animal models.
- **DeTIL-0255:** In November, Nurix announced the successful completion of the safety run-in portion of the Phase 1 trial of DeTIL-0255 in patients with advanced gynecologic malignancies. Based on the preliminary safety profile of DeTIL-0255 and feedback from the FDA, Nurix plans to explore a potential combination strategy of DeTIL-0255 with NX-1607.

- **Hosted Nurix's first R&D Day:** In May, Nurix provided a deep dive into Nurix's DELigase platform that underpins its current and future targeted protein modulation programs as well as progress on each of Nurix's four wholly owned clinical programs at its first R&D Day. An archived webcast of the event can be accessed via the Events and Presentations page of the Investor section of the Nurix website.
- **Executed on Nurix's regulatory strategy:** In March, the UK Medicines and Healthcare products Regulatory Agency (MHRA) awarded the innovative medicine designation, the Innovation Passport, for NX-1607 for the treatment of patients with advanced solid tumors. The Innovation Passport is the entry point to the Innovative Licensing and Access Pathway (ILAP) which aims to accelerate time to market and facilitate patient access to novel drugs to treat serious and life-threatening diseases. In addition, Nurix also received clearance from the FDA of its IND applications to expand ongoing Phase 1 clinical programs for NX-1607 and NX-5948 into sites in the United States.

- **Presented scientific and preclinical data supporting its DELigase platform and four clinical programs at major scientific and medical meetings throughout the year.** These posters and presentations are archived and can be accessed via the Scientific Resources page of the Nurix website.
- **Advanced preclinical discovery programs with collaboration partners Sanofi and Gilead Sciences.** Nurix is advancing a total of 15 discovery programs, including 10 under its collaboration partnerships with Sanofi and Gilead Sciences. Of its ten partnered programs, Nurix has options to co-develop and co-promote a total of four programs. At its R&D Day in May, Nurix provided visibility into the stage of development of these 10 programs, which have continued to advance significantly in H2 2022.

Corporate

- **Strengthened balance sheet raising gross proceeds of $115 million in 2022.** In July, Nurix entered into separate securities purchase agreements with healthcare-focused investment funds to sell, in registered direct offerings, pre-funded warrants to purchase an aggregate of 6,814,920 shares of Nurix's common stock at a price of $13.939 per pre-funded warrant, cumulatively yielding total gross proceeds of $95 million. In June, Nurix issued and sold 2,000,000 shares of common stock under its Equity Distribution Agreement at a price of $10.0001 per share of common stock for total gross proceeds of approximately $20 million. As of December 31, 2022, Nurix had $130.0 million of common stock remaining available for sale under the Equity Distribution Agreement. As of August 31, 2022, Nurix had cash, cash equivalents and marketable securities of $413.6 million. Nurix expects that its existing cash, cash equivalents and marketable securities, excluding any future potential milestones from collaborations, will be sufficient to fund its operating activities into the fourth quarter of 2024.
- **Enhanced the Nurix leadership team and board with the hiring of chief people officer and the appointment of leading industry strategist to the board of directors:** In August, Nurix announced that Eric Schlezinger, J.D., an industry veteran with extensive experience leading and developing human resources, joined the company as chief people officer. In September, Nurix announced the appointment of leading industry strategist Edward C. Saltzman to its board of directors. Mr. Saltzman has over 30 years of drug strategic development experience and currently serves as Head of Biotech Strategy at Lumanity Inc., a global pharmaceutical and biotechnology advisory firm.

About Nurix

Nurix Therapeutics is a clinical stage biopharmaceutical company focused on the discovery, development and commercialization of small molecule and cell therapies based on the

modulation of cellular protein levels as a novel treatment approach for cancer and other challenging diseases. Leveraging extensive expertise in E3 ligases together with proprietary DNA-encoded libraries, Nurix has built DELigase, an integrated discovery platform to identify and advance novel drug candidates targeting E3 ligases, a broad class of enzymes that can modulate proteins within the cell. Nurix's drug discovery approach is to either harness or inhibit the natural function of E3 ligases within the ubiquitin proteasome system to selectively decrease or increase cellular protein levels. Nurix's wholly owned pipeline includes targeted protein degraders of Bruton's tyrosine kinase, a B-cell signaling protein, and inhibitors of Casitas B-lineage lymphoma proto-oncogene B, an E3 ligase that regulates T cell activation. Nurix is headquartered in San Francisco, California. For additional information visit http://www.nurixtx.com.

Forward Looking Statement

This press release 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 press release, 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, 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 Nurix's future financial or business performance; Nurix's ability to fund its operating activities into the fourth quarter of 2024; Nurix's future plans, prospects and strategies; Nurix's current and prospective drug candidates; the planned timing and conduct of the clinical trials for Nurix's drug candidates; the planned timing for the provision of updates and findings from Nurix's clinical trials; the potential advantages of Nurix's DELigase™ platform and drug candidates; and the extent to which Nurix's scientific approach and DELigase™ platform may potentially address a broad range of diseases. 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) the impact of the COVID-19 pandemic, increasing financial market volatility and uncertainty, rising interest rates and inflation on Nurix's business, clinical trials, financial condition, liquidity and results of operations; (v) Nurix's ability to protect its intellectual property and (vi) 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, 2022, and other SEC filings. Accordingly, readers are cautioned not to place undue reliance on these forward-looking statements. The statements in this press release speak only as of the date of this press release, 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.

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