Nurix Therapeutics

Blazing a New Path in Medicine

Investor Presentation

November 2020

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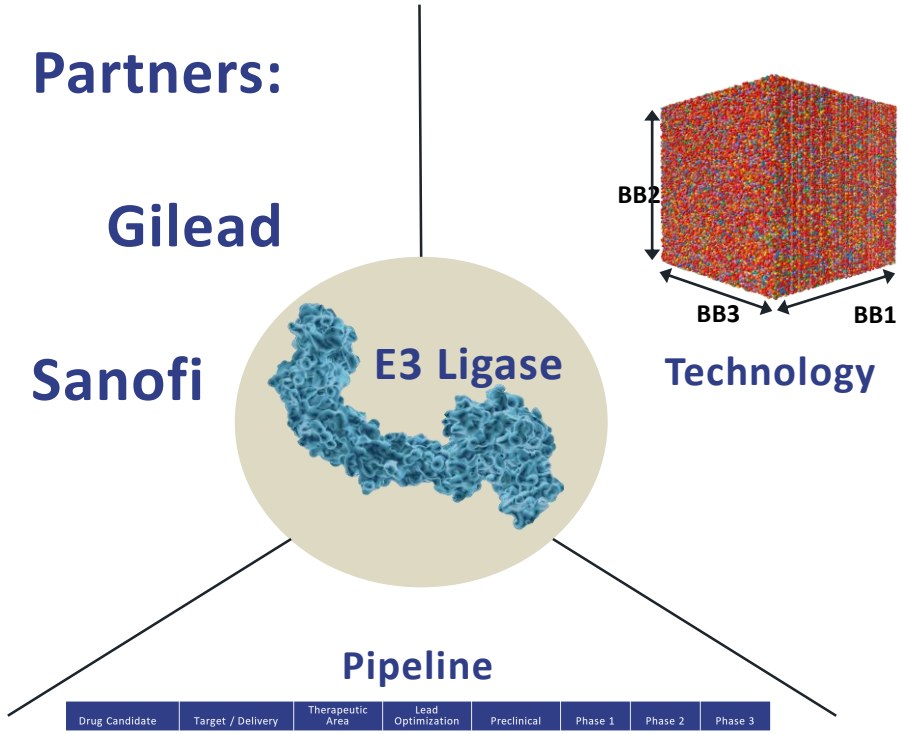
This presentation contains forward-looking statements and information relating to Nurix Therapeutics, Inc. (the “Company,” “we," "us" or "our"). Forward-looking statements are neither historical facts nor assurances of future performance. Instead, they are based on our current beliefs, expectations and assumptions regarding the future of our business, future plans and strategies, our development plans, our preclinical results and other future conditions. All statements, other than statements of historical facts, contained in this presentation are forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995, including statements regarding our future financial or business performance, conditions, plans, prospects, trends or strategies and other financial and business matters; our current and prospective product candidates; the timing of our planned IND submissions for our product candidates; the potential advantages of our DELigase™ platform and product candidates, including NX-1607 and NX-0255; the extent animal model data predicts human efficacy; and the success and timing of our development and commercialization of our product candidates, including our DeTIL and DeCART opportunities. In addition, when or if used in this presentation, the words “may,” “could,” “should,” “anticipate,” “believe,” “estimate,” “expect,” “intend,” “plan,” “predict” and similar expressions and their variants, as they relate to the Company may identify forward-looking statements. Although we believe the expectations reflected in such forward-looking statements are reasonable, we can give no assurance that such expectations will prove to be correct. Readers are cautioned that actual results, levels of activity, performance or events and circumstances could differ materially from those expressed or implied in our forward-looking statements due to a variety of factors, including risks and uncertainties related to our ability to advance our product candidates; obtain regulatory approval of and ultimately commercialize our product candidates; the timing and results of preclinical and clinical trials; our ability to fund development activities and achieve development goals; the impact of the COVID-19 pandemic on our business; our ability to protect intellectual property; and other risks and uncertainties described under the heading “Risk Factors” in our final prospectus pursuant to Rule 424(b)(4) filed with the Securities and Exchange Commission (the “SEC”) on July 24, 2020 and in our Quarterly Report on Form 10-Q for the quarter ended August 31, 2020, as well as other SEC filings. Accordingly, readers are cautioned not to place undue reliance on these forward-looking statements. Except as required by applicable law, we do not plan to publicly update or revise any forward-looking statements contained herein.

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 is reliable, such estimates and research have not been verified by any independent source.

Nurix: A Targeted Protein Modulation Company

- Targeting E3 ligases to develop small molecule targeted protein modulation drug candidates that can *increase* or *decrease* protein levels
- Four wholly-owned oncology and immunology drug candidates with first clinical trial expected to commence in H1 2021
- Applying targeted protein modulation to create new adoptive cell therapies for cancer and to discover anti viral drugs
- DELigase™: a versatile drug discovery platform comprised of massive DNA-encoded libraries to screen an expanded universe of E3 ligases
- Revenue generating drug discovery partnerships with Sanofi and Gilead



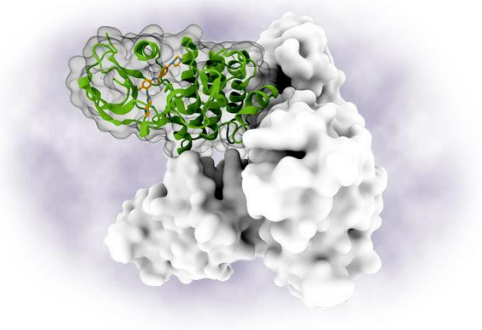
Pipeline

Drug Candidate	Target / Delivery	Therapeutic Area	Lead Optimization	Preclinical	Phase 1	Phase 2	Phase 3
Protein Degradation Chimeric Targeting Molecule (CTM) Portfolio							
NX-2127	BTK + IMiD activity *Oral*	B-cell Malignancies	*Late Q4 '20 – Q1 '21				
NX-5948	BTK *Oral*	B-cell Malignancies And GVHD		*H2 '21			
Ligase Inhibition Portfolio							
NX-1607	CBL-B *Oral*	Immuno-oncology		*Q3 '21			
DeTIL-0255	CBL-B *ex vivo*	Adoptive Cell Therapy (ACT)		*H2 '21			

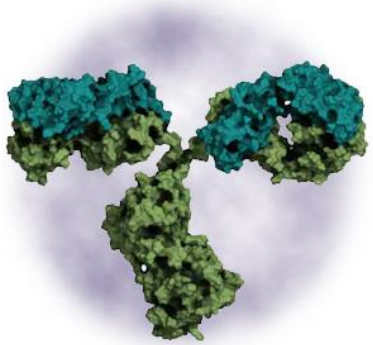
*Expected IND submission timing based on calendar year quarters

Working to Create a New Category of Medicine

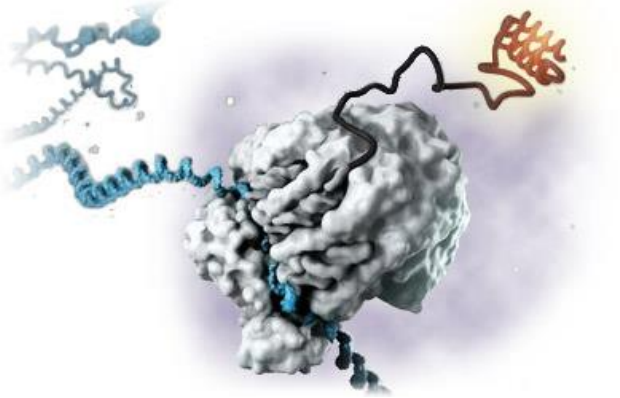
Evolution of new therapeutic modalities



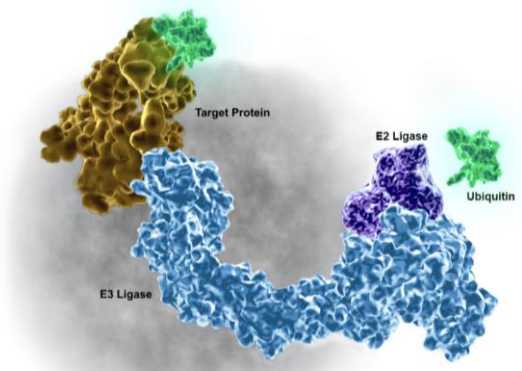
Small Molecule Inhibitors



Antibodies



Nucleic Acid-Based Therapies:
Antisense, RNAi
Gene Therapy
CRISPR



Nurix Protein Modulation Drugs to *Increase* or *Decrease* Specific Protein Levels

Nurix's Wholly-Owned Targeted Protein Modulation Drug Pipeline: Multiple Clinical Programs Expected Next Year

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Nurix's Wholly Owned Research Pipeline

Drug Candidate	Target	Therapeutic Area	DEL Discovery	Lead Optimization	Preclinical
Protein Degradation					
KINASE-CTM3	Undisclosed	T-cell Malignancies and Autoimmune disease			
COVID-CTM 1	SARs CoV2	Anti-viral			
COVID-CTM 2	SARs CoV2	Anti-viral			
COVID-CTM 3	SARs CoV2	Anti-viral			
Ligase Inhibition					
LIGASE-INH2	Undisclosed	Immuno-oncology			

Accomplished Leadership Team

Leadership Team

Arthur T. Sands, M.D., Ph.D.
Chief Executive Officer; Member of the Board

Hans van Houte
Chief Financial Officer

Pierre Beaurang, Ph.D.
Chief Business Officer

Gwenn Hansen, Ph.D.
Chief Scientific Officer

Michael T. Lotze, M.D.
Chief Cellular Therapy Officer

Robert J. Brown, M.D.
Vice President of Clinical Development

Christine Ring, Ph.D., J.D.
General Counsel

Jason Kantor, Ph.D.
SVP Finance and Investment Strategy

Founders

Michael Rapé, Ph.D.
UC Berkeley, HHMI

John Kuriyan, Ph.D.
UC Berkeley, HHMI

Arthur Weiss, M.D., Ph.D.
UCSF, HHMI

Board

David Lacey, M.D.
Chairman, Independent

Leon Chen, Ph.D.
Independent

Julia P. Gregory
Independent

Lori A. Kunkel, M.D.
Independent

Jeff Tong, Ph.D.
Independent

Significant Strategic Collaborations for an Extensive Early Stage Pipeline of Targeted Protein Degradation Candidates

Nurix's collaborations with premier pharmaceutical companies designed to provide non-dilutive capital, expand our drug discovery platform, generate future pipeline, and retain rights to our internal pipeline

Gilead Sciences

June 2019

- Option to license up to 5 drug candidate programs identified via DELigase™ proprietary platform
- Upfront payment of $45M; Up to $2.3B in additional payments, including early discovery milestones
- Nurix retains U.S. rights for 2 product candidates under a co/co structure
- Nurix internal or third party programs excluded

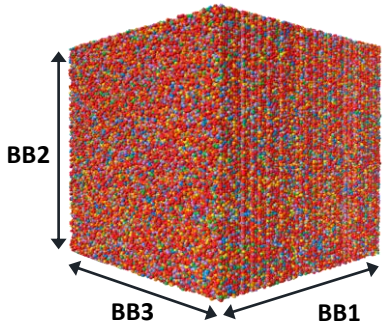
Sanofi

December 2019

- License for 3 programs with potential expansion for 2 additional programs identified via DELigase™ proprietary platform
- Upfront payment of $55M; Up to $2.5B in additional payments, including early discovery milestones
- Nurix option to retain U.S. rights for up to two product candidates under a co/co structure
- Nurix internal or third party programs excluded

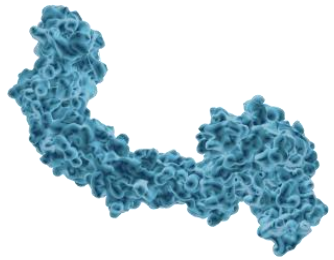
DELigase™: Platform Enables Two Complementary Protein Modulation Approaches for Drug Discovery

1



Proprietary DNA-Encoded Libraries, >10^9 compounds

2



Expanded universe of E3 ligases, >30 ligases currently in discovery

Protein Modulation Platform

Inhibitors *of* E3 Ligases

Harnesses *of* E3 Ligases

Ligase Inhibitors *Increase* specific protein levels

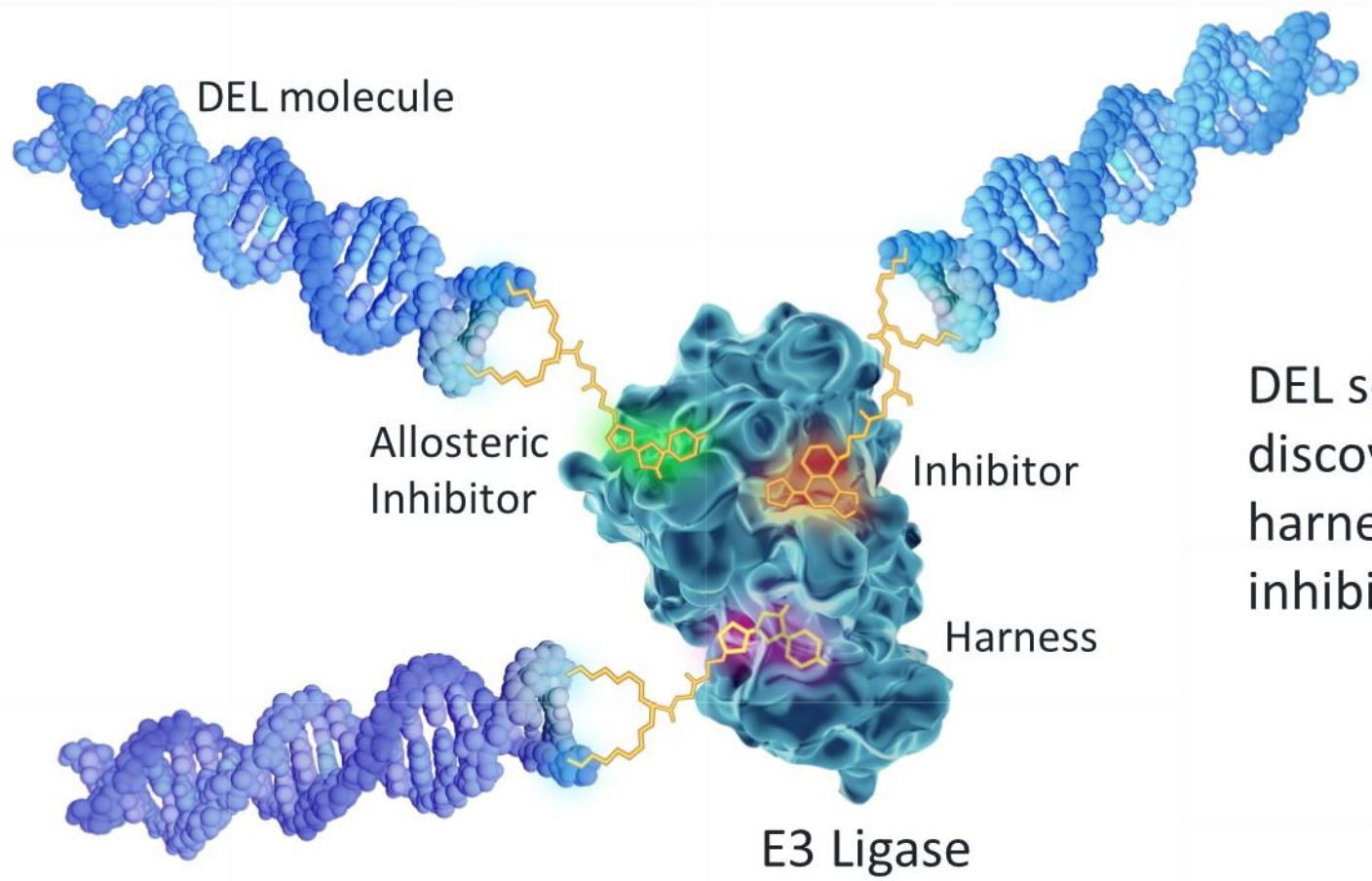
3

Ability to push protein levels in either direction

Chimeric Targeting Molecules (CTMs) *Decrease* specific protein levels

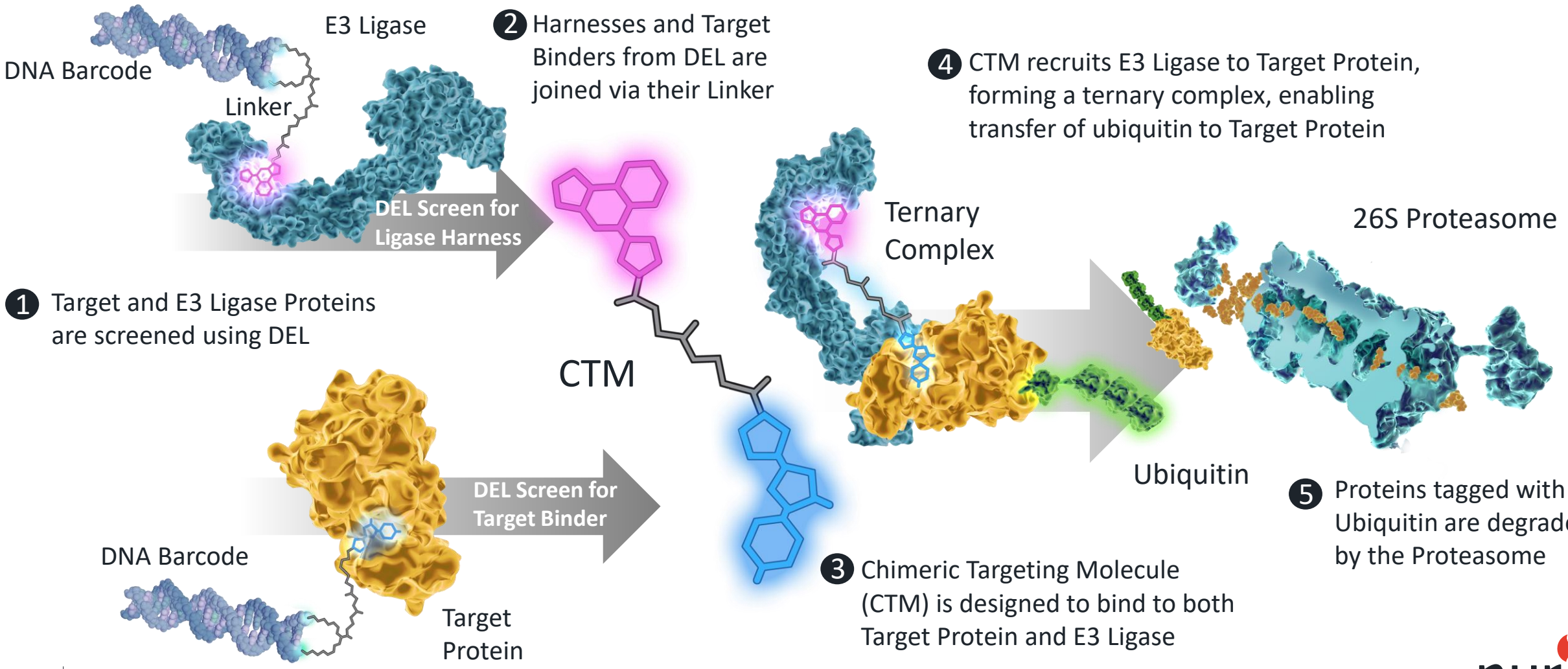
Drug Development

DELigase™ Identifies a Spectrum of Binders Across the Ligase Surface

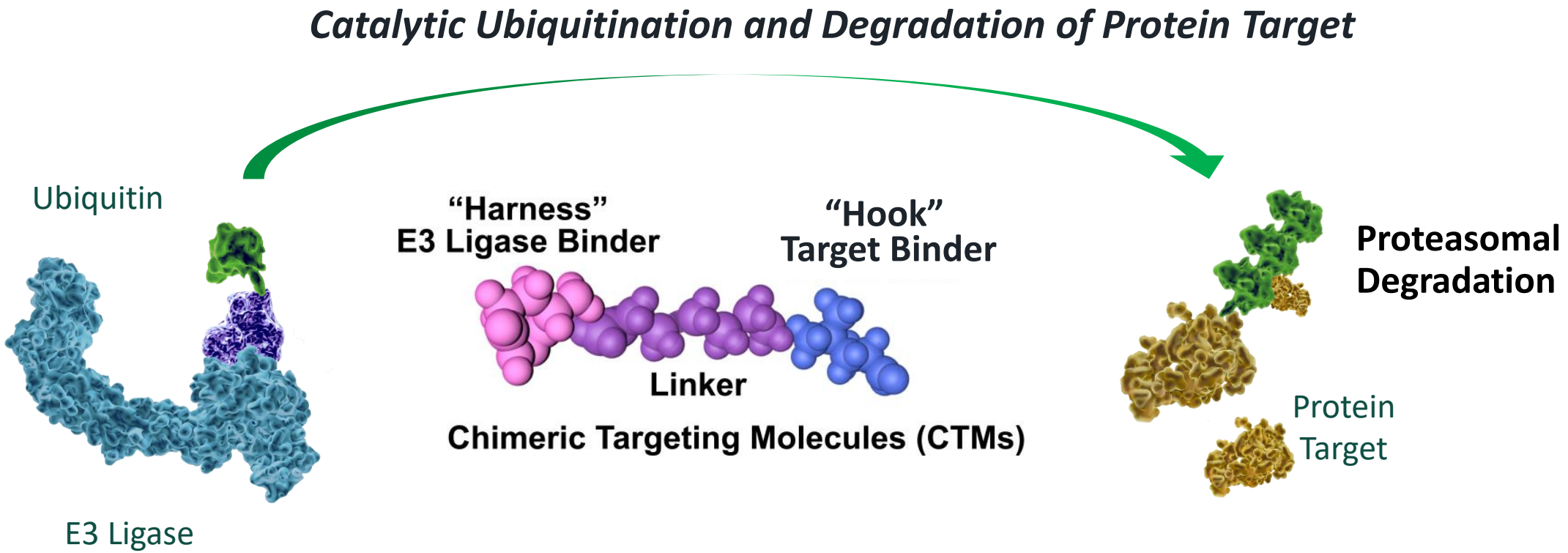


DEL screening enables discovery of both harnesses and inhibitors of E3 ligases

DELigase™ Enables Efficient Chimeric Targeting Molecule (CTM) Component Discovery and Design



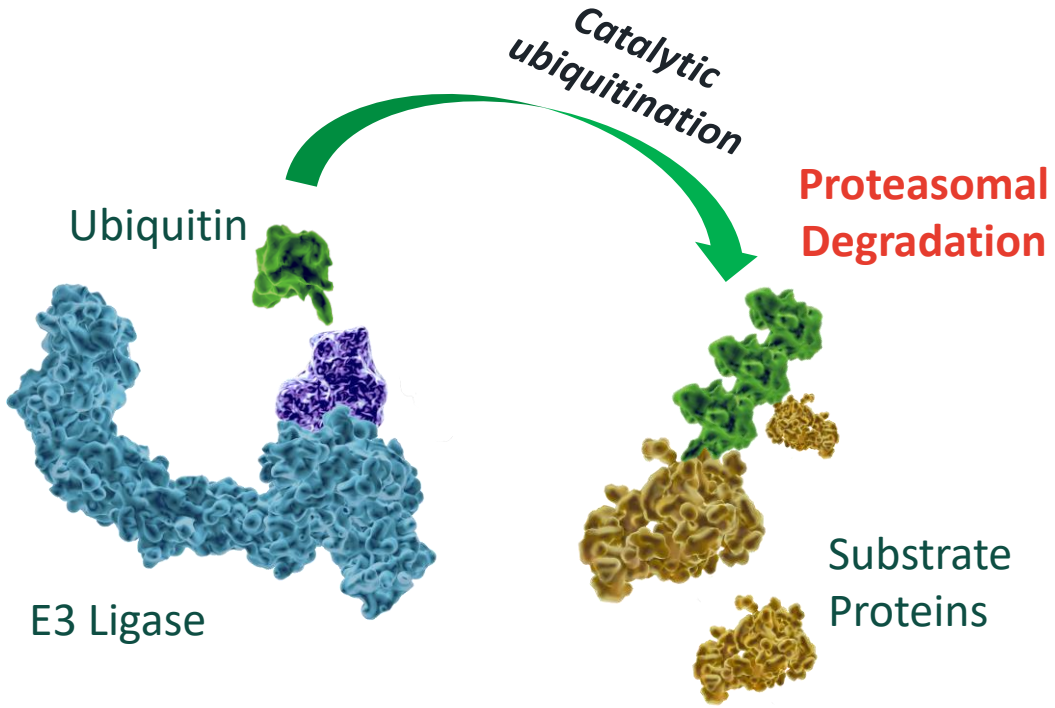
Chimeric Targeting Molecules (CTMs): Induced Degradation Can *Decrease* Levels of Virtually Any Protein Target



- Harnessing E3 ligases to degrade target proteins (neo-substrates)
- One CTM can induce degradation of more than one target protein

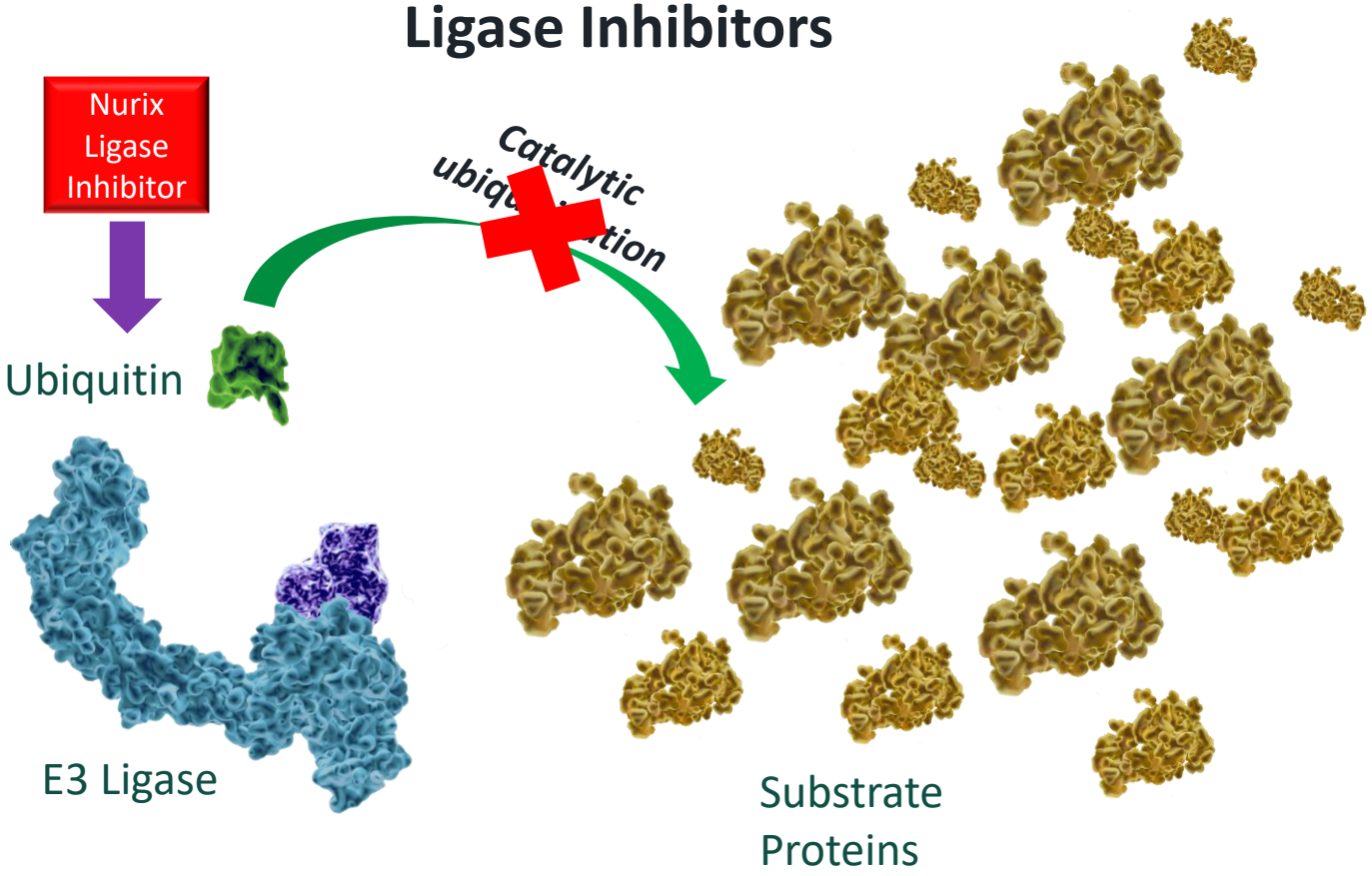
Ligase Inhibitors Can *Increase* Levels of Specific Substrate Proteins

Natural E3 Ligase-Mediated Proteasomal Degradation



E3 Ligase-mediated proteasomal degradation can be highly specific to decrease substrate proteins

Ligase Inhibitors



Substrate protein levels increase

Nurix's Wholly-Owned Targeted Protein Modulation Drug Pipeline: Multiple Clinical Programs Expected Next Year

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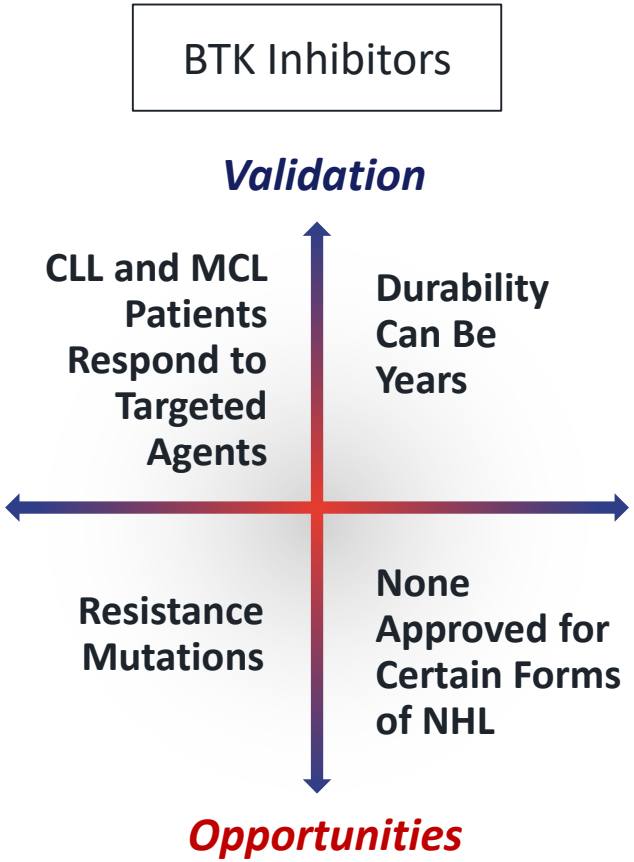
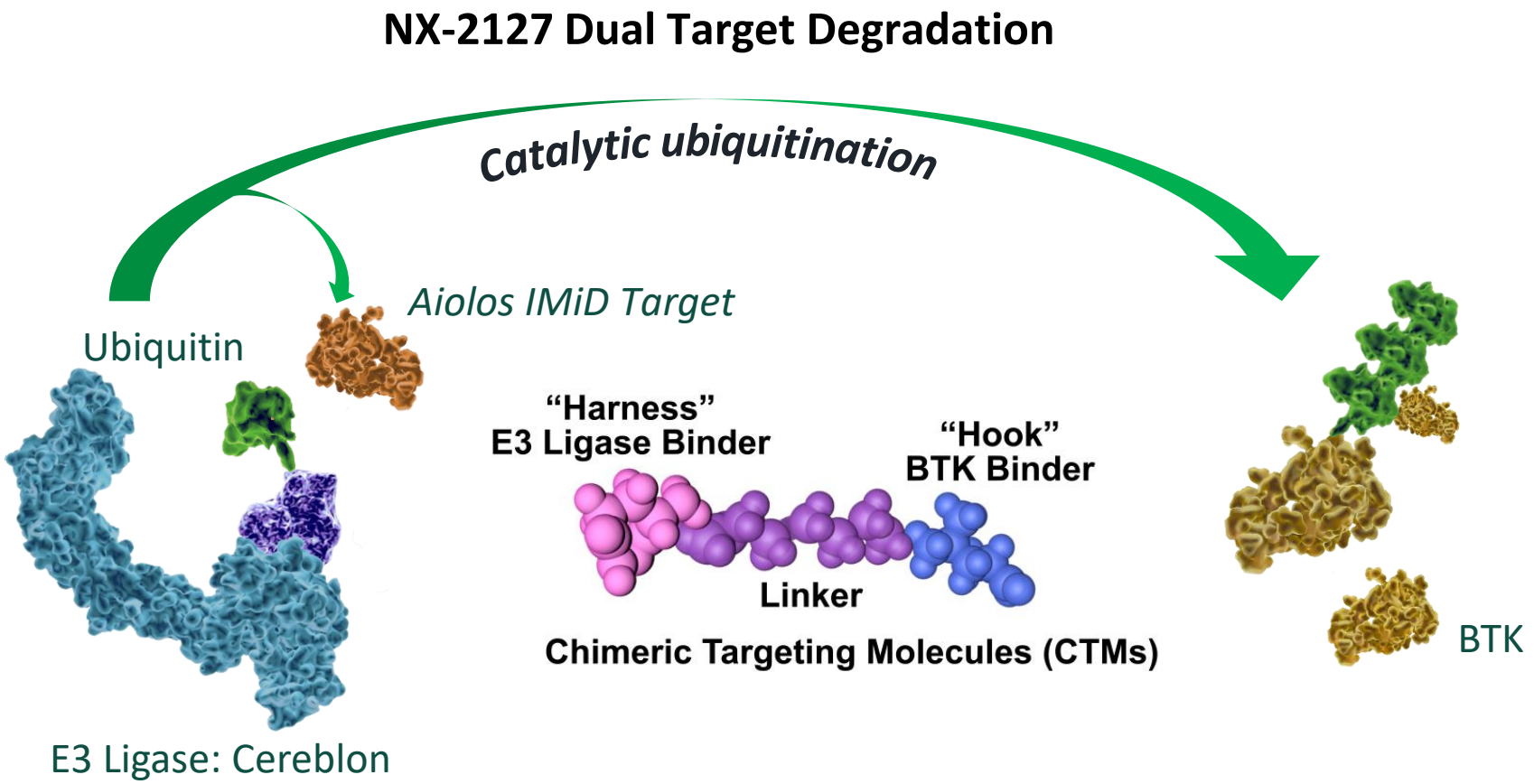
BTK CTMs: A Differentiated Approach to B-Cell Malignancies in BTK Inhibitor Failures

- BTK is a validated target
  - Global sales of BTK inhibitors were $5.8 billion in 2019
  - BTK inhibitors are approved by the FDA for five different diseases across multiple lines of therapy (CLL/SLL, mantle cell lymphoma, Waldenstrom's, marginal zone lymphoma, GVHD)
- Clinical and commercial strategy:
  - Initial focus on fast to market opportunity as a potentially superior treatment for relapsed and resistant chronic lymphocytic leukemia (CLL) and C481S resistance to ibrutinib
  - Expand beyond CLL: 77,000 people in the United States will be diagnosed with Non-Hodgkin's Lymphoma (NHL) in 2020 and 85% of NHLs are a result of B-cell malignancies
  - Opportunities: Follicular lymphoma and diffuse large B-cell lymphoma (DLBCL), areas where BTK inhibitors have not been approved nor proven successful



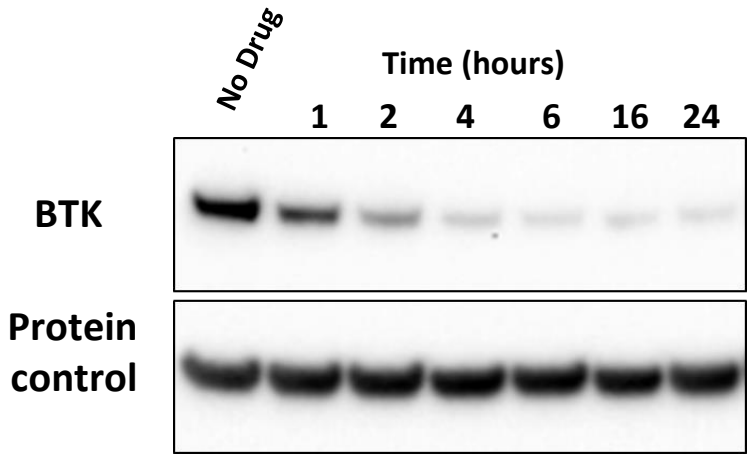
NX-2127 Clinical Candidate Has a Dual Degradation Mechanism of Action for Two Clinically Validated Targets



Nurix CTMs Degrade BTK Levels in Lymphoma Cell Lines and CLL Patient Cells

The precursor compound BTK CTM 1.2 led to the optimization and selection of NX-2127 as a development candidate

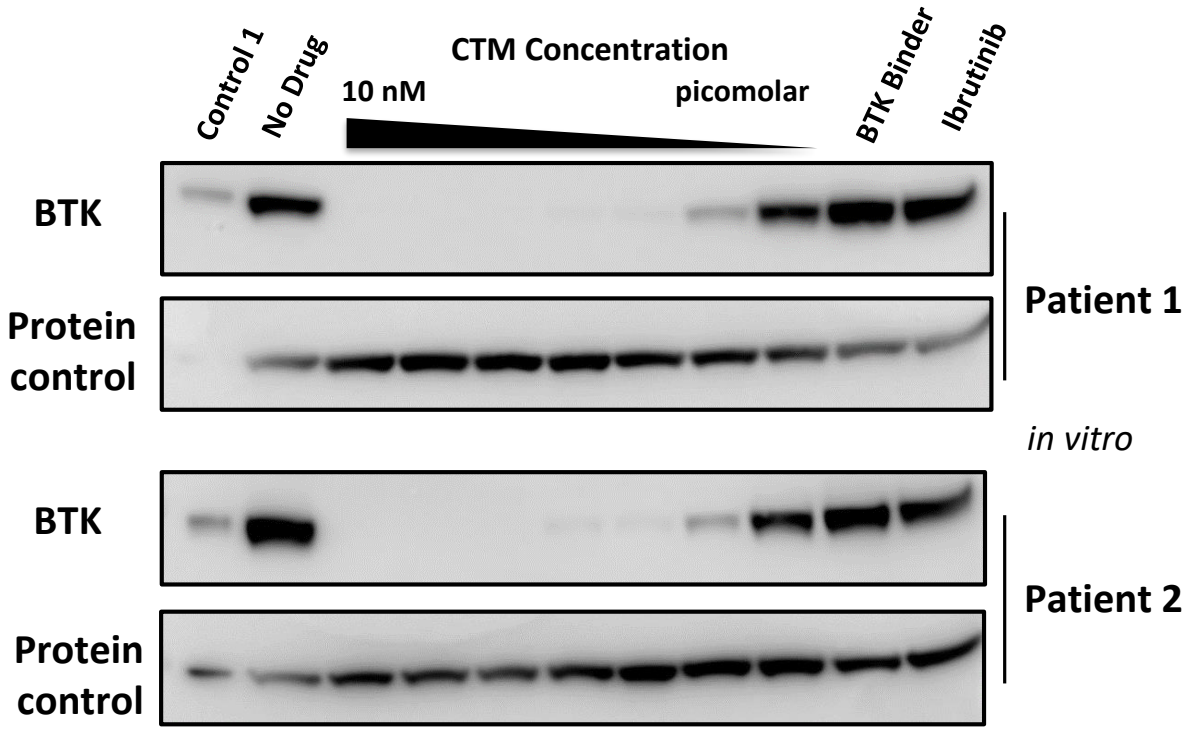
Rapid Degradation of BTK Protein in Lymphoma Cell Line - BTK CTM 1.1



in vitro, 100 nM CTM

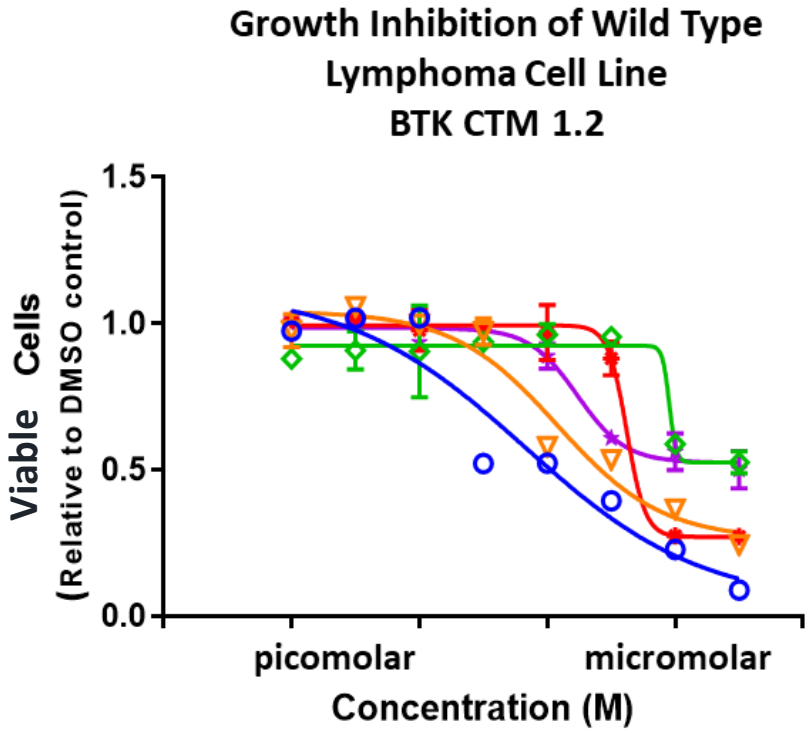
- Potent degradation of BTK shown in primary CLL patient cells
- Degradation is both time and concentration dependent

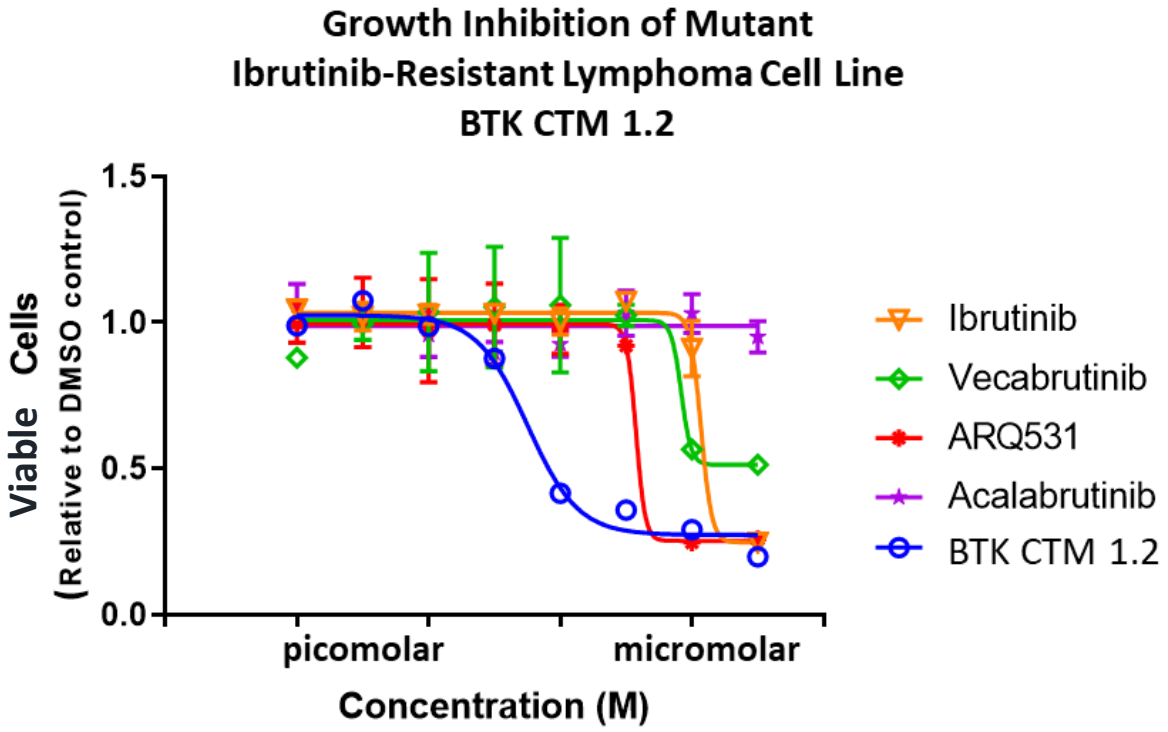
Potent Degradation of BTK Protein in CLL Patient Cells - BTK CTM 1.2



Note: Control 1 lane has one-tenth the total protein loaded compared to other lanes

Nurix CTM Effectively Inhibits Growth of Ibrutinib-Resistant Tumor Cell Lines



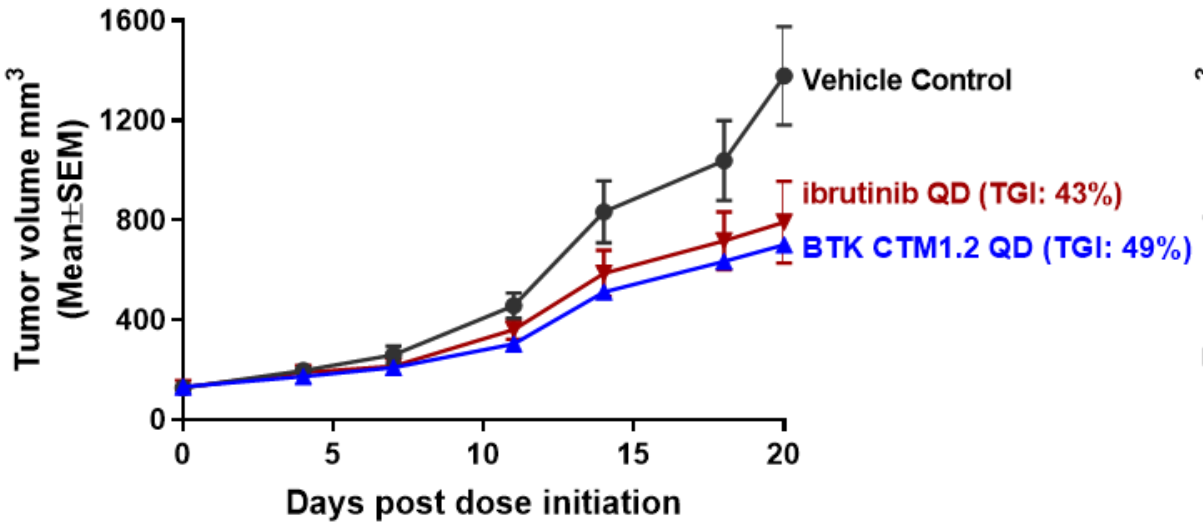


- BTK CTM 1.2 demonstrates comparable growth inhibition as ibrutinib of a tumor cell line with a wild type (normal) BTK target protein and more potent effects compared to other BTK inhibitors

- BTK CTM 1.2 retains potent growth inhibition activity relative to BTK inhibitors in a tumor cell line carrying the C481S mutation, one of the most common known human resistance mutations in the BTK target protein

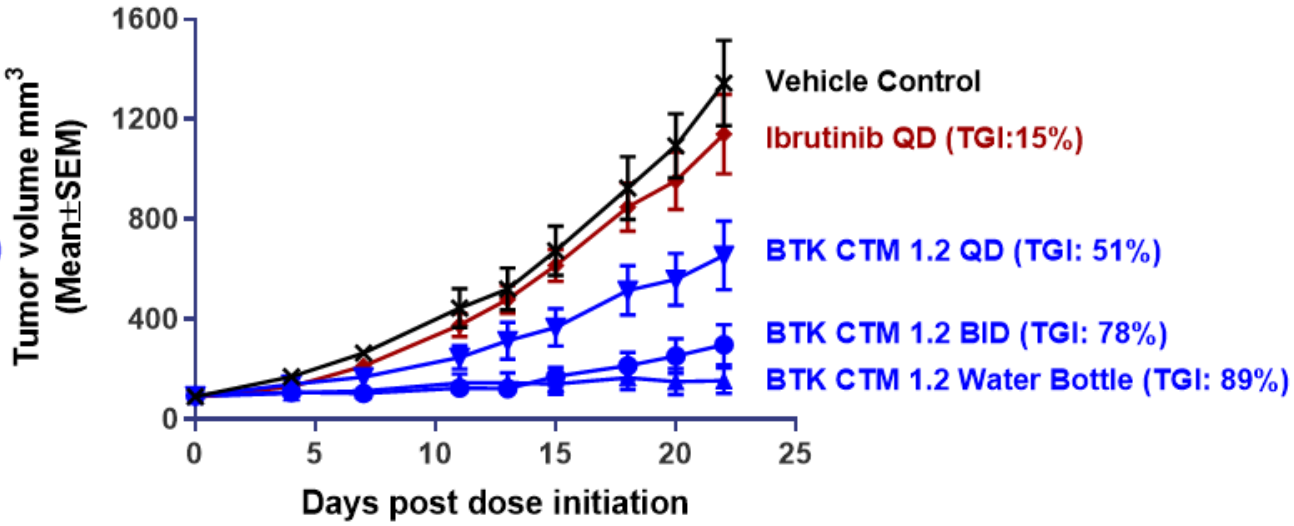
Oral Administration of BTK CTM Demonstrates Cancer Growth Inhibition in Mouse Xenograft Tumor Model

Tumor Growth Inhibition in Xenograft Model of Wild Type Lymphoma



- BTK CTM 1.2 demonstrates comparable growth inhibition as ibrutinib in a xenograft mouse model containing tumors with a wild type (normal) BTK target protein

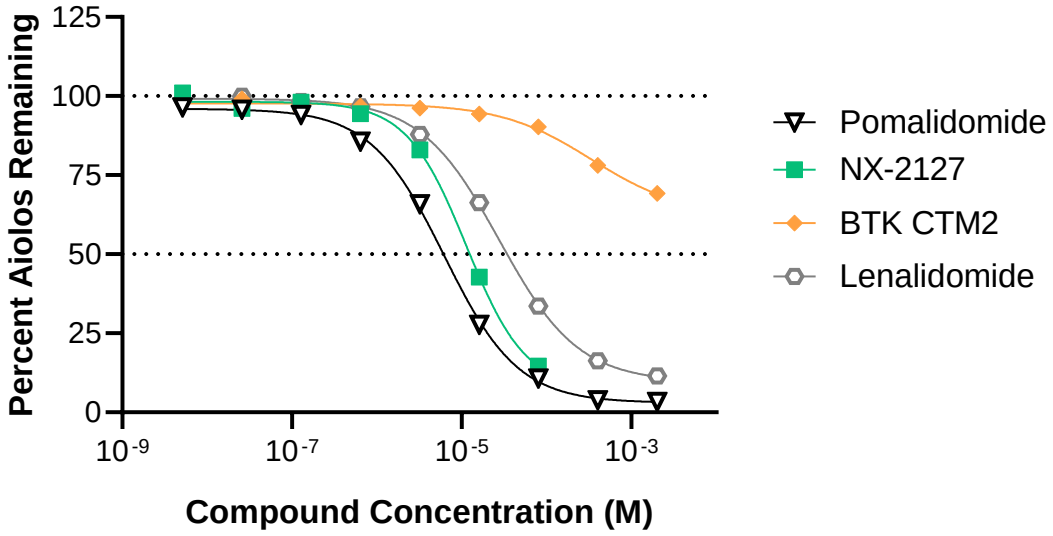
Tumor Growth Inhibition in Xenograft Model of Mutant Ibrutinib-Resistant Lymphoma



- BTK CTM 1.2 shows potent growth inhibition compared to ibrutinib in a xenograft mouse model containing tumors with the most common human resistance mutation (C481S) in BTK target protein

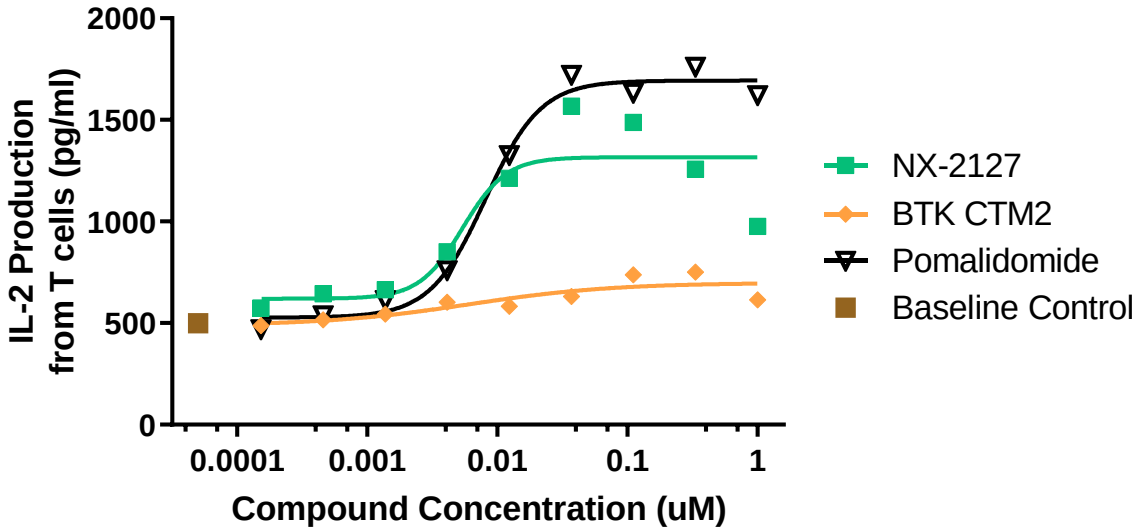
NX-2127 Demonstrated Dual Target Activity Against BTK and Aiolos May Improve Anti-Tumor Activity

IMiD Activity: Aiolos Degradation



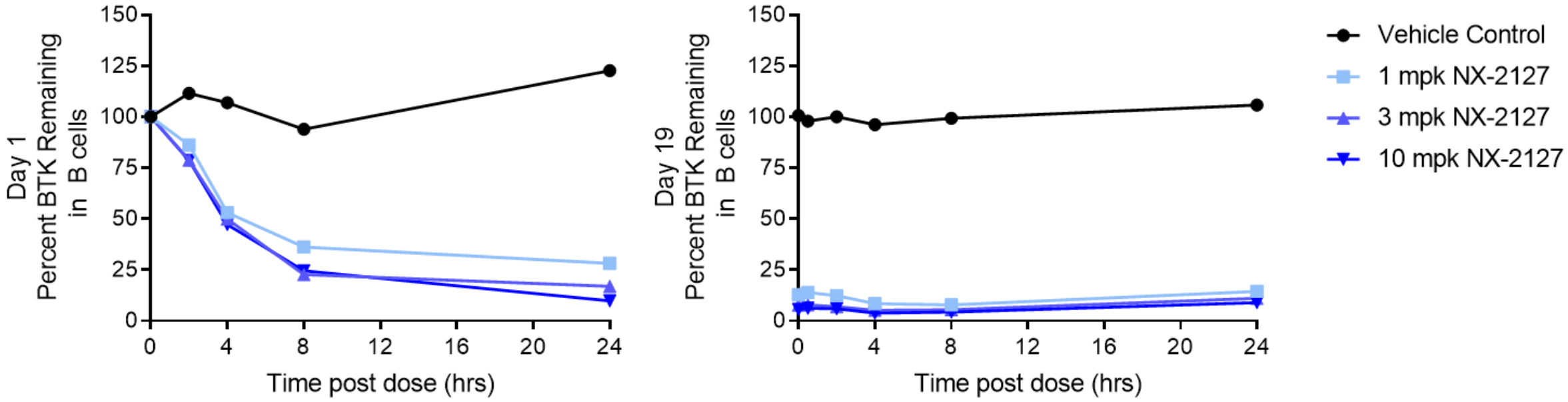
- NX-2127 shows comparable Aiolos degradation as pomalidomide and lenalidomide

IMiD Activity: T Cell Activation



- NX-2127 IMiD activity corresponds with T cell activation
- NX-5948 is within the BTK CTM 2 series which shows little or no pharmacologically meaningful IMiD activity

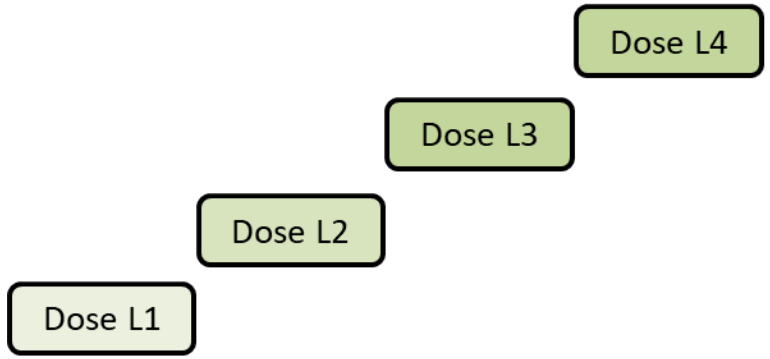
Oral Dosing of NX-2127 Degrades BTK in NHPs



- Significant degradation of BTK in 4 hours and more than 90% degradation through 24 hours post dosing at the highest dose level
- Once daily, oral dosing of NX-2127 maintains suppression of BTK protein levels throughout the 19-day duration of the study

NX-2127: Phase 1 Clinical Development Plan

Phase 1a - Monotherapy Dose Escalation
(n= ~24)



Patients: relapsed/refractory B-cell malignancies
- Oral dosing once per day
- 4 to 6 cohorts
- Total projected patients, n = ~24

Phase 1b Monotherapy Expansion Recommended Phase 2 Dose
(n= ~100)

- Chronic Lymphocytic Leukemia (CLL) with C481 mutation (n=~20)
- CLL – unselected (n=~20)
- Mantle Cell Lymphoma (MCL), Marginal Zone Lymphoma (MZL), Waldenstrom's Macroglobulinemia (WM) (n=~20)
- Follicular Lymphoma (FL) (n=~20)
- Diffuse Large B-Cell Lymphoma (DLBCL) (n=~20)

- Establish proof of concept in relapsed and refractory B-cell malignancies including those in which have shown ibrutinib resistance or intolerance
- Planning a two-part Phase 1 monotherapy trial in relapsed or refractory NHL and CLL
  - Phase 1a:
    - Assess safety and tolerability
    - Identify maximum tolerated dose
  - Phase 1b:
    - 5 cohorts of up to 20 patients each
    - Patients with CLL, CLL + C481 mutation, MCL, MZL or WM, FL and DLBCL

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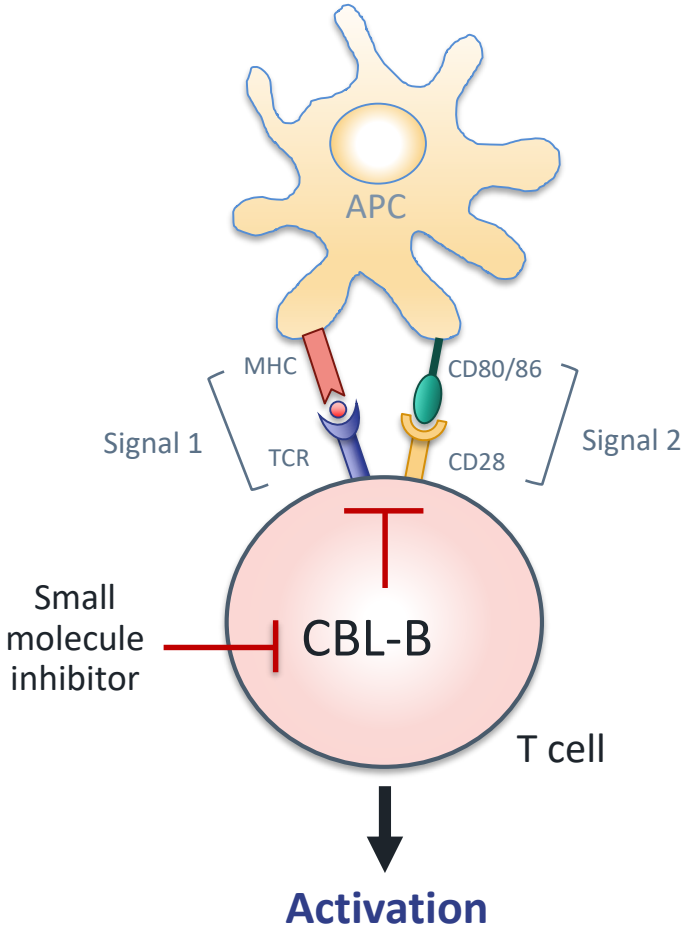
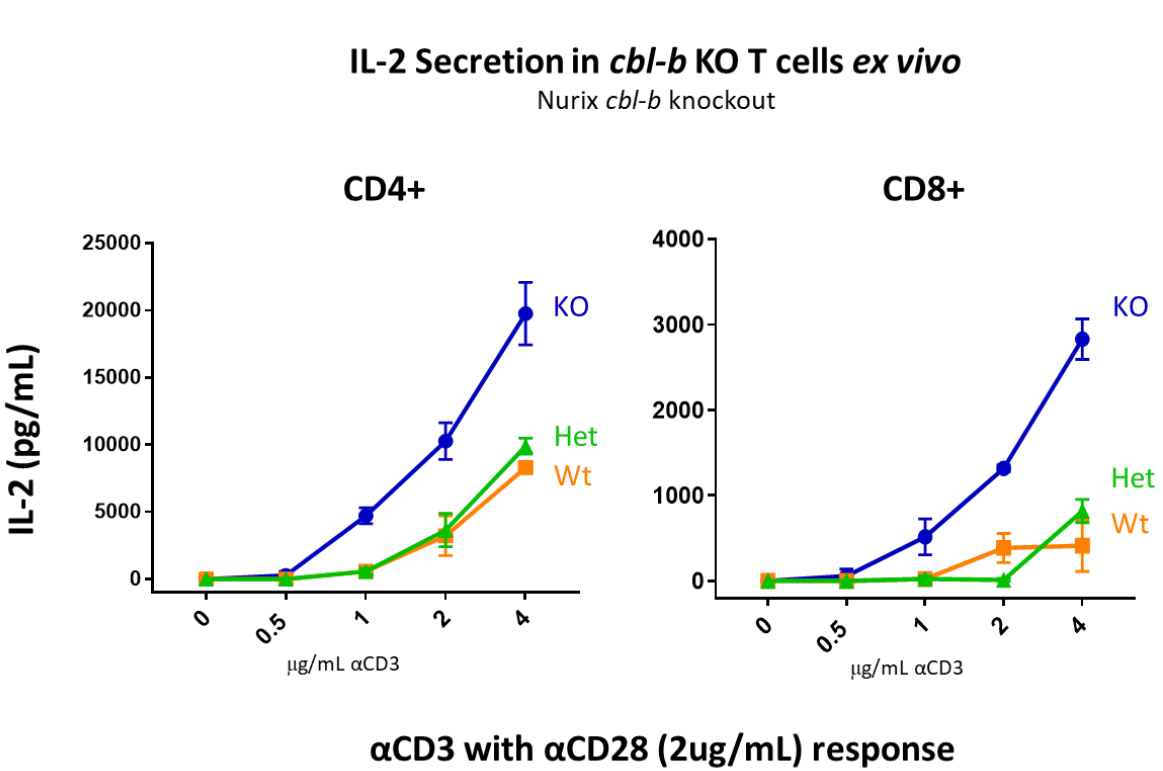
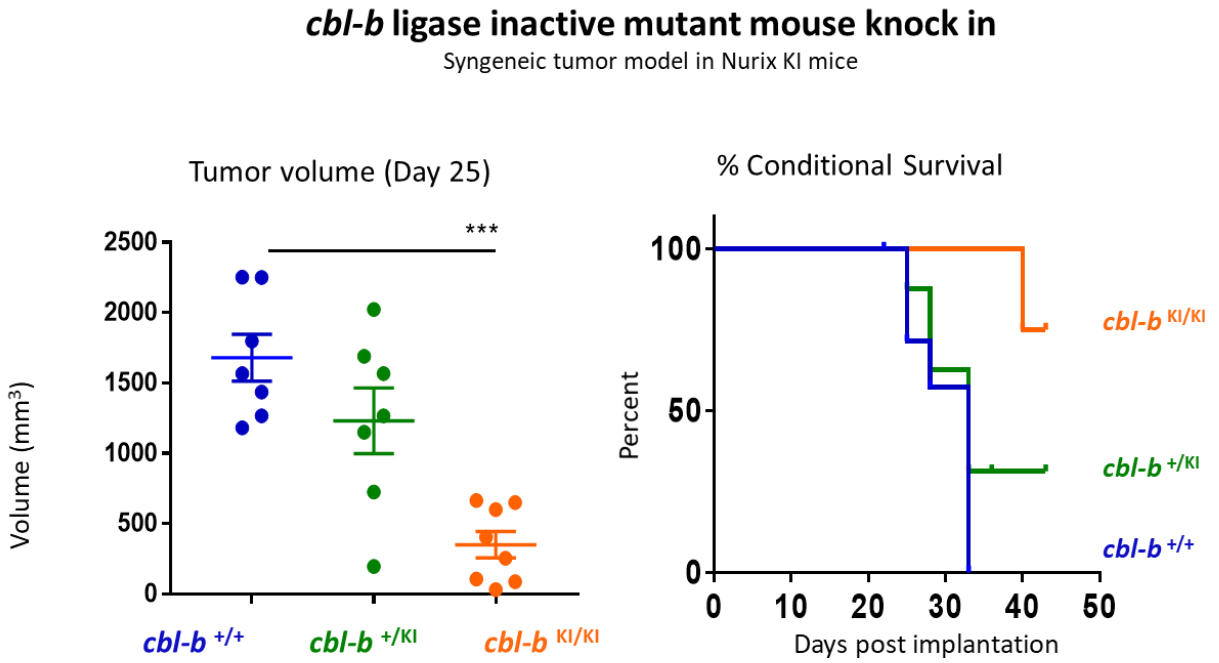
CBL-B: A Modulator of T Cell Activation for Tumor Immunotherapy

- CBL-B is an E3 ligase that regulates the immune system by specifically degrading proteins involved in shutting off T-cell signaling
- Blocking CBL-B removes a brake on the immune system
- CBL-B function is supported by mouse and human genetics
- CBL-B inhibitors have remarkable effects on T cells
  - CBL-B inhibitors induce immune cells to secrete IL-2
  - Skewing T cells to a central memory phenotype
  - *Ex vivo* and *in vivo* administration of CBL-B inhibitors demonstrate anti-tumor effects in animal models of cancer

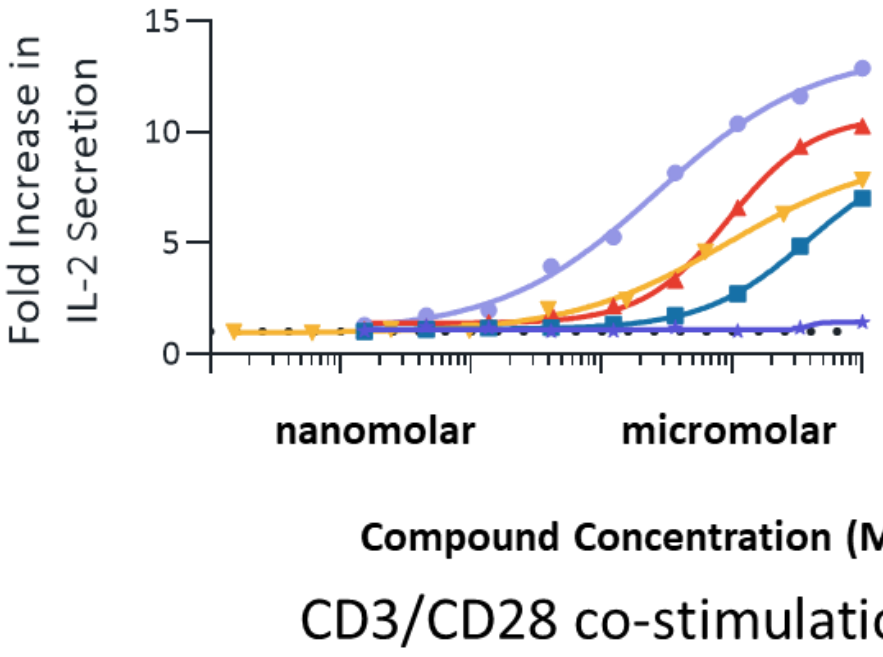


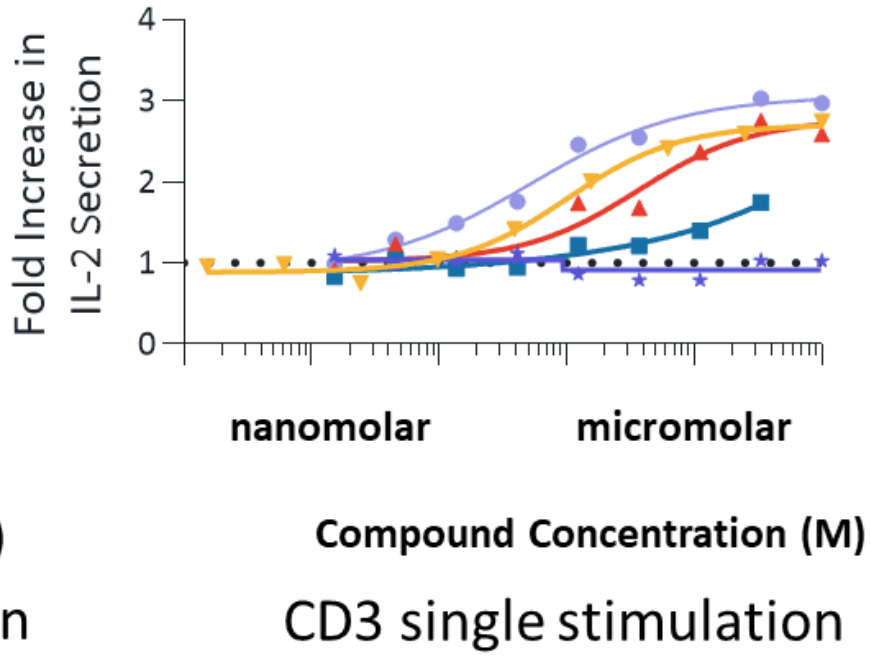
CBL-B Knockout Validation: Enhanced IL-2 Secretion, Tumor Growth Inhibition, and Survival





Nurix CBL-B Inhibitors Elevate IL-2 Levels *ex vivo* in Human Donor T Cells





Biochemical Activity

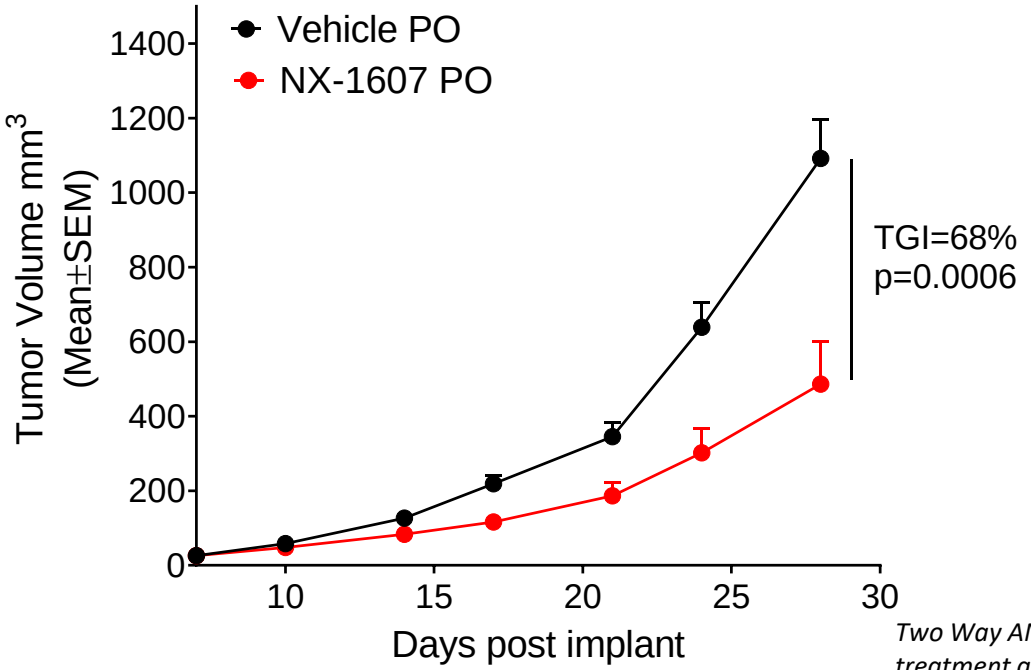
Compound	IC_{50} nM
NRX-5	5
NRX-4	15
NRX-3	26
NRX-2	112
NRX-1 (inactive enantiomer of NRX-4)	1,191

T cell activity ranks orders with biochemical activity

- Several fold increase in IL-2 production corresponds with increasing biochemical activity of CBL-B inhibitors
- CBL inhibition results in increased T cell activation in the absence of co-stimulation with CD3 and CD28, a potential advantage in a suppressive tumor microenvironment

Once Daily Oral Dosing of NX-1607 Recapitulates Anti-Tumor Effects of CBL-B, Ligase Inactive, Knock-in Mouse Model

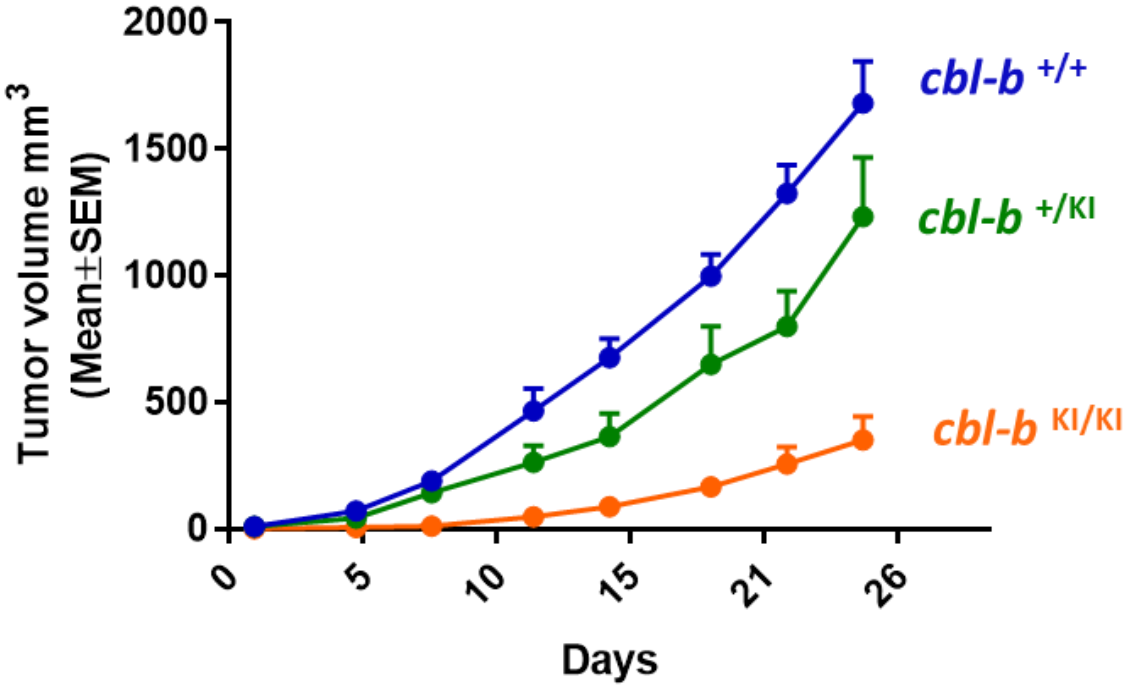
Oral daily dosing of NX-1607



Two Way ANOVA of treatment group vs vehicle control
Average tumor volumes from both flanks are depicted

Tumor growth inhibition (TGI) with NX-1607 treatment; tumors implanted at Day 0; once daily oral dosing of NX-1607 was given from Day 9 to Day 28

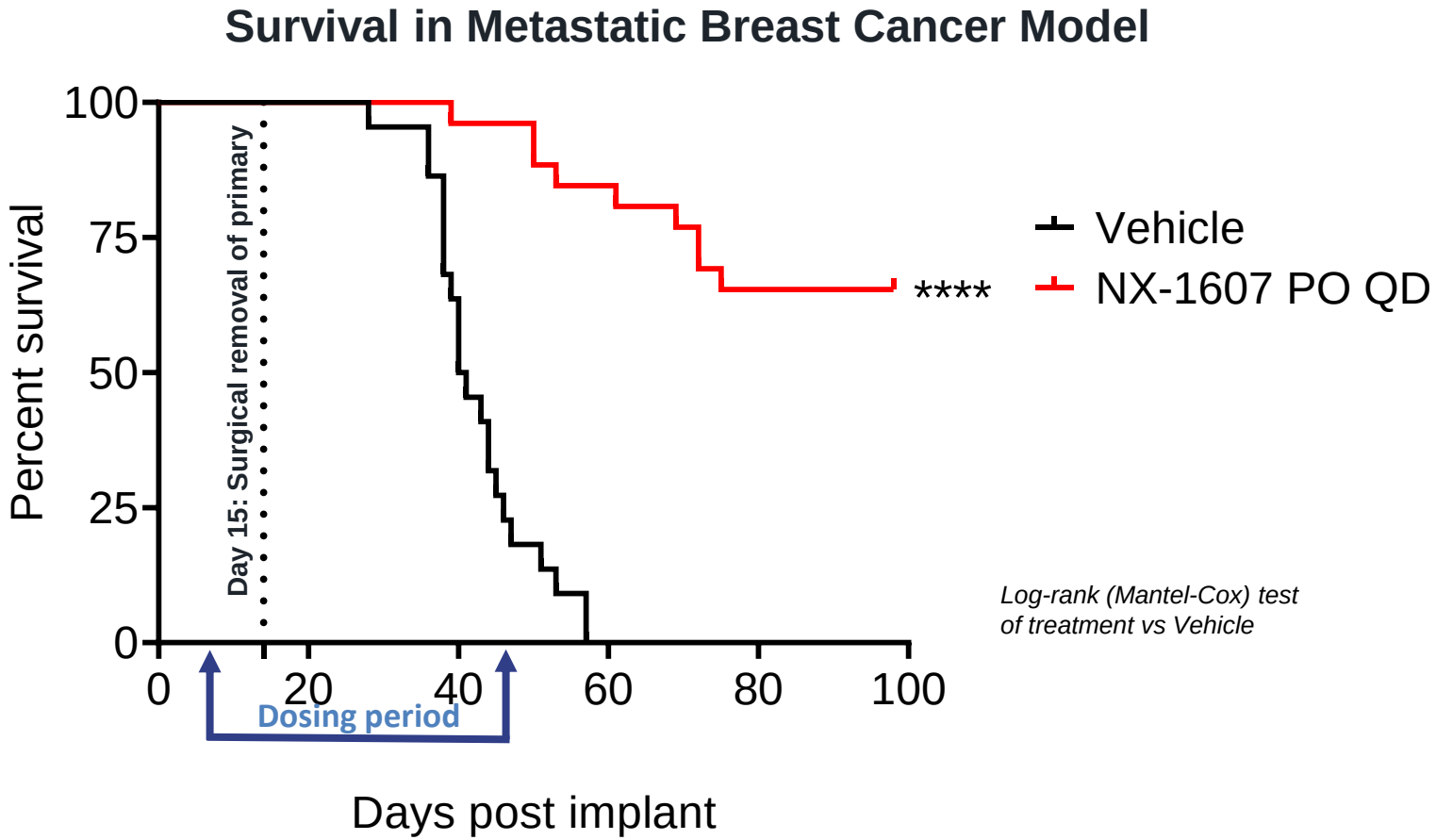
CBL-B ligase-dead, knock-in (KI) model



Anti-tumor effects in ligase-inactive, knock-in (KI) mutation model

NX-1607 Induces Long Term Survival in Metastatic, Triple Negative, Breast Cancer Model

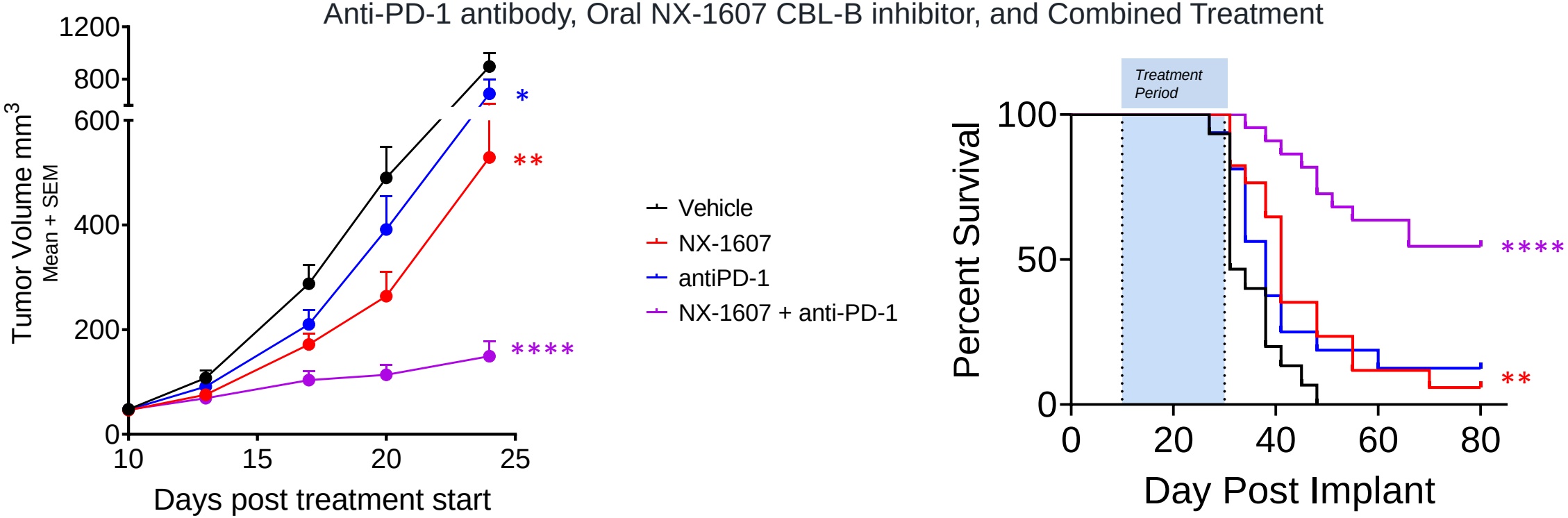
- Once daily oral dosing of NX-1607
- Tumors implanted at Day 0
- Surgical removal of primary tumor at Day 15
- NX-1607 was given before the surgery from day 7 to day 15 (neo-adjuvant phase) and continued after surgery until day 46.



Triple negative breast carcinoma cells metastasize from subcutaneous space to distant sites

NX-1607 and Anti-PD-1 Synergize to Improve Anti-Tumor Effects and Survival of Tumor-bearing Mice

Anti-PD-1 antibody, Oral NX-1607 CBL-B inhibitor, and Combined Treatment



Combination of NX-1607 and anti-PD-1 treatment significantly improves anti-tumor response and survival in mice bearing two tumors relative to vehicle or anti-PD-1 alone

Tumors from both flanks plotted
Two-way ANOVA of treatment group vs vehicle control

Log-rank (Mantel-Cox) test of vehicle vs treatment

Developing Small Molecule CBL-B Inhibitors as Intracellular Checkpoint Inhibitors to Treat Solid Tumors

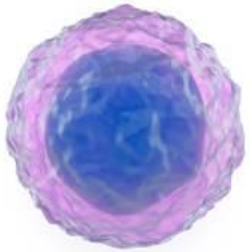
- Solid tumors represent approximately 90% of adult human cancers; current checkpoint inhibitors have benefited patients with a growing list of tumor types
- CBL-B inhibitors may induce immune cell localized IL-2 secretion in addition to other immune activation effects that may enhance anti-tumor responses
- Clinical strategy: develop CBL-B inhibitors to treat patients who fail to respond or experience only transient responses to current therapies
- Potential for both monotherapy and combination therapy strategies for NX-1607

Solid Tumor Indication	2020 Estimated Deaths	2020 Estimated New Cases
Melanoma	6,850	100,350
Ovarian	13,940	21,750
Breast	42,170	276,480
Cervix Uteri	4,290	13,800
Lung and Bronchus	135,720	228,820
Bladder	17,980	81,400
Pancreatic	47,050	57,600
Oral Cavity, Pharynx, Larynx	10,750	53,260
Brain and other Nervous System	18,020	23,890

NX-1607 Clinical Development Plan

- Preclinical characterization and IND-enabling activities ongoing
- Phase 1 study design:
  - Single agent, dose-escalation study
  - Patients with solid tumors resistant to standard of care including checkpoint inhibitors
- Study objectives:
  - Primary: safety and tolerability, identification of a maximum tolerated dose for further evaluation
  - Secondary: pharmacokinetic and pharmacodynamic profile, exploratory assessment of anti-tumor activity

Introducing Pharmacologic Control of Adoptive Cell Therapy with Targeted Protein Modulation



Adoptive Cell Therapy

Convergence

Small molecule targeted protein modulation:
CBL-B Inhibitors

DeCART™

Drug-**E**nhanced
Chimeric **A**ntigen **R**eceptor **T** Cell Therapy

NxACT™

Nurix **A**doptive **C**ell **T**herapy Program

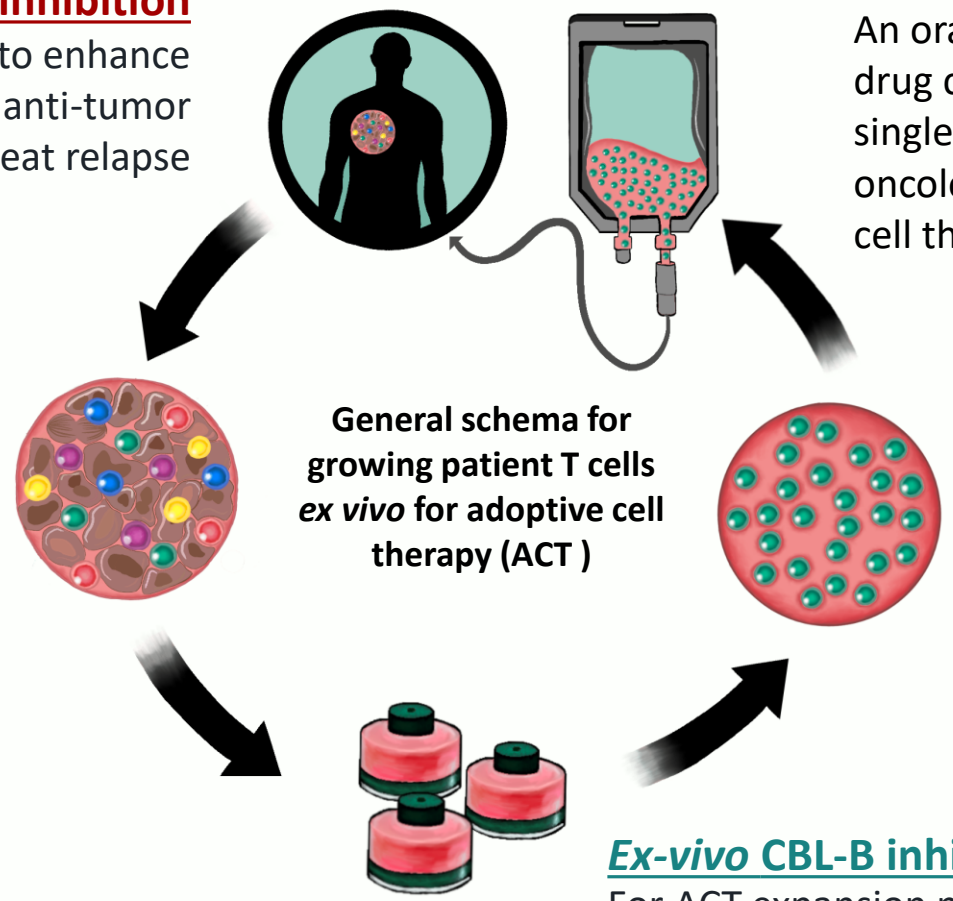
DeTIL™

Drug-**E**nhanced
Tumor **I**nfiltrating **L**ymphocytes

CBL Inhibitors to Enhance Adoptive Cell Therapy: DeTIL™ and DeCART™

Oral CBL-B inhibition
For co-administration to enhance engraftment to improve anti-tumor activity or to treat relapse

Oral NX-1607
An oral small molecule immunotherapy drug candidate in development as a single agent or in combination with other oncology therapies including adoptive cell therapy



General schema for growing patient T cells *ex vivo* for adoptive cell therapy (ACT)

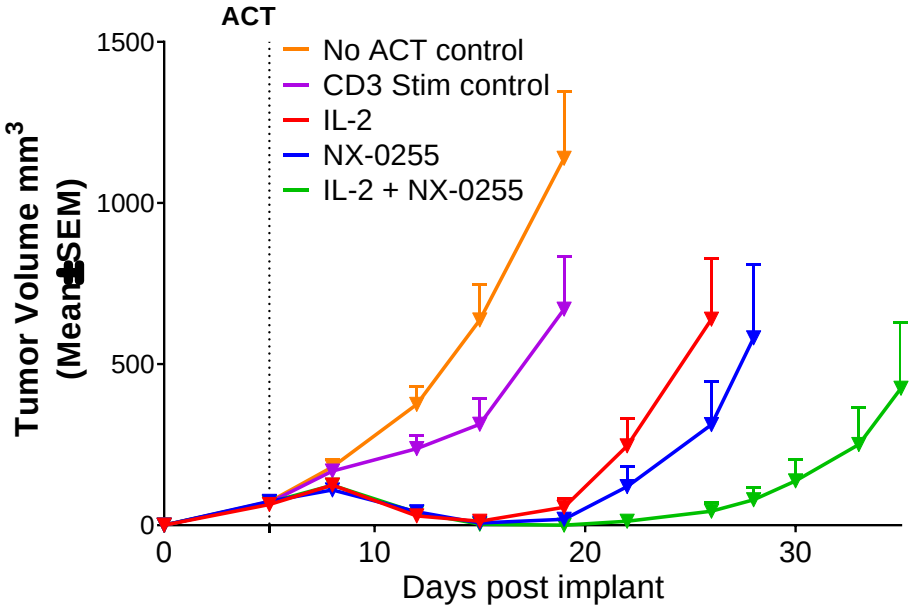
***Ex-vivo* CBL-B inhibition**
For enhanced isolation of T cells for TIL or CAR-T therapy

***Ex vivo* NX-0255**
DeTIL and DeCART created by *ex vivo* CBL inhibition with small molecule NX-0255 producing a TIL and CAR-T cell therapy products with enhanced characteristics

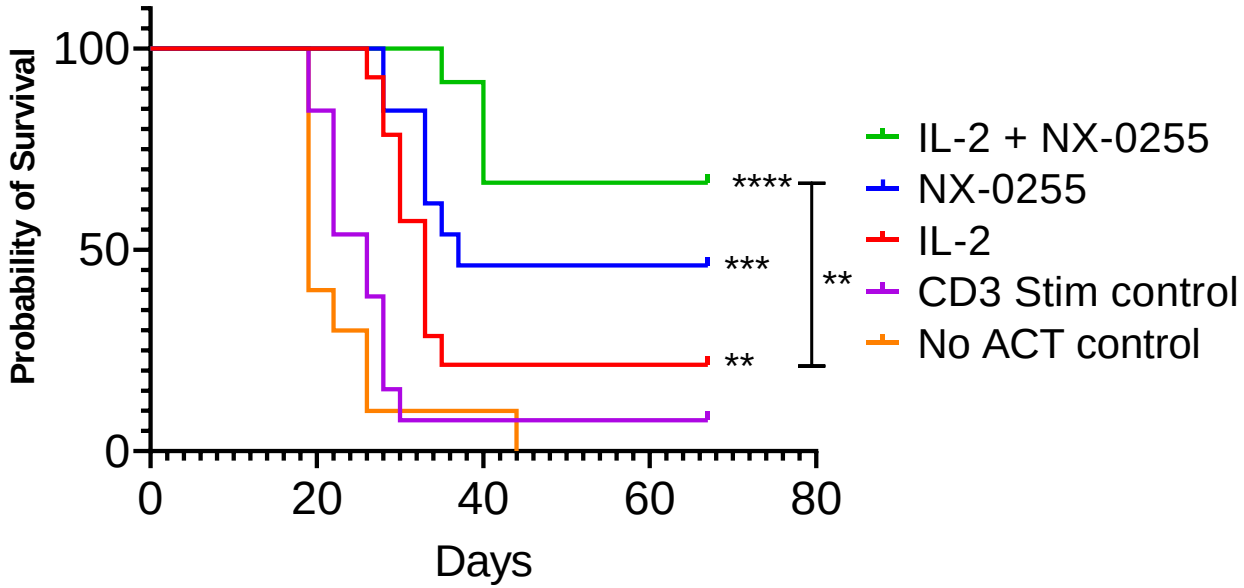
***Ex-vivo* CBL-B inhibition**
For ACT expansion phase to enhance cellular phenotype

NX-0255 *ex vivo* Treatment Provides Robust Anti-Tumor Activity in Mouse Model

Reduction in Tumor Growth in Mouse ACT Tumor Model



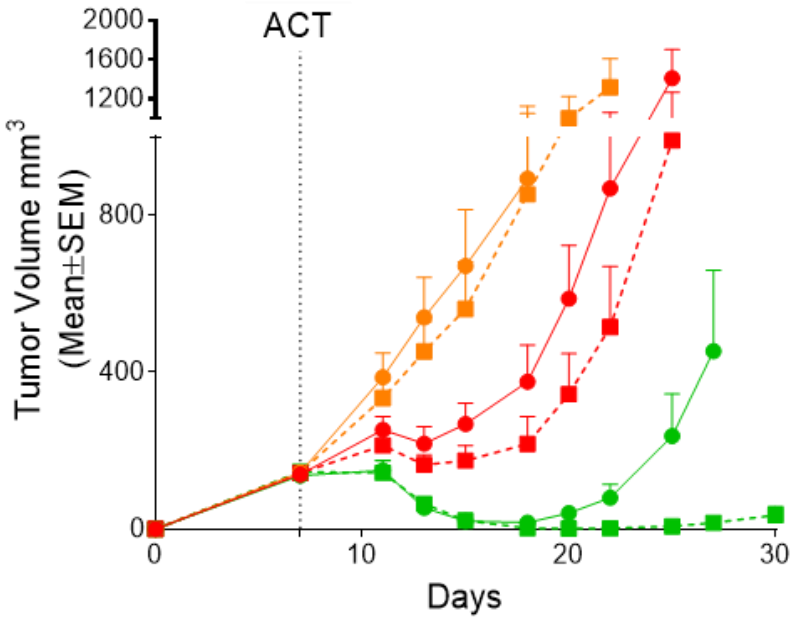
Improvement in Conditional Survival in Mouse ACT Tumor Model



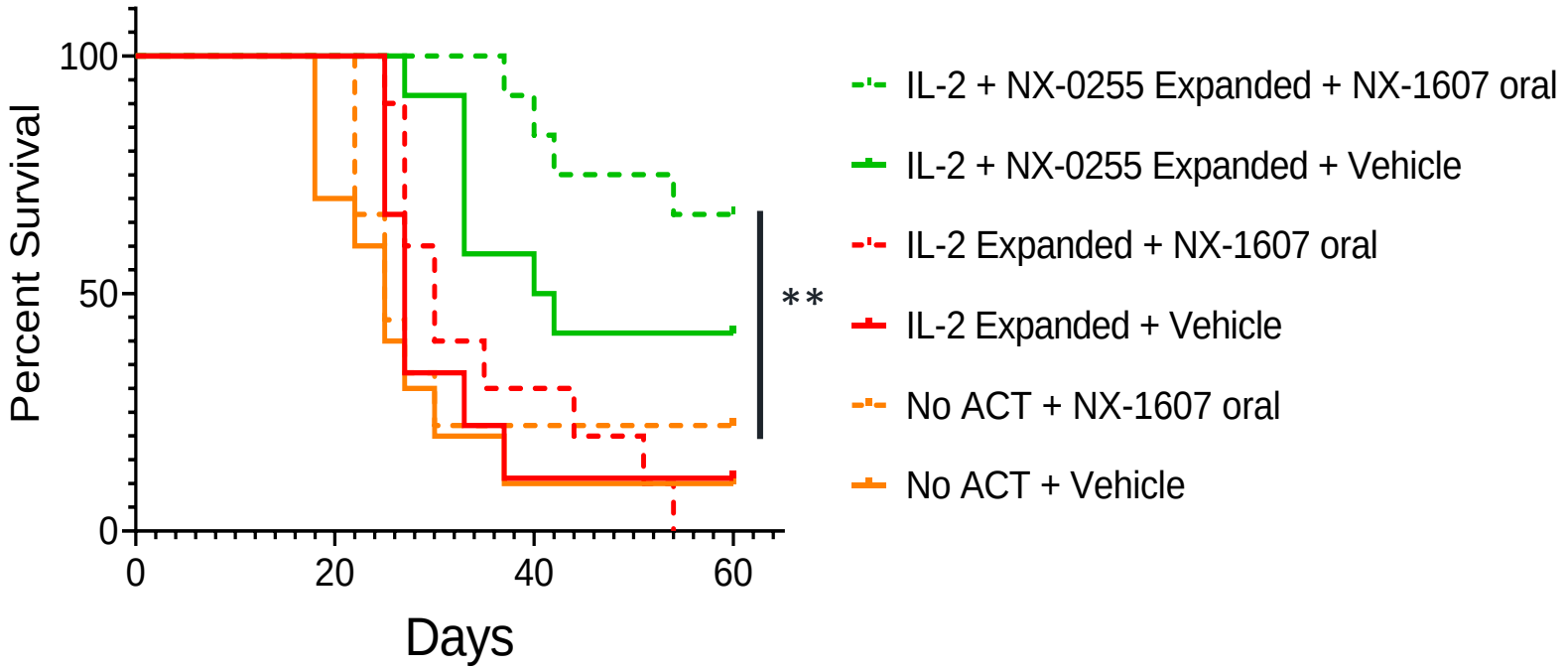
- CD8+ cells exposed to NX-0255 alone *ex vivo* resulted in superior conditional survival compared to using IL-2 alone
- CD8+ cells exposed to NX-0255 and IL-2 combined *ex vivo* exert a deeper anti-tumor response
- NX-0255 *ex vivo* exposure period is only three days, anti-tumor effects persist for over a month after engraftment
- Animals that rejected tumor were rechallenged 80 days post ACT. All animals rejected tumor, demonstrating immunological memory

Oral NX-1607 Augments Anti-Tumor Activity Observed with *ex vivo* NX-0255 Combination in ACT Mouse Model

Reduction in Tumor Growth in Mouse ACT Tumor Model



Improvement in Conditional Survival in Mouse ACT Tumor Model



- Oral NX-1607 treatment once daily further enhances conditional survival and anti-tumor activity of T cells expanded for three days with recombinant IL-2 plus NX-0255 *ex vivo* in adoptive cell therapy mouse model

Matching the Right Business Strategy with Each NxACT Opportunity

DeTIL

Drug-**E**nhanced

Tumor **I**nfiltrating **L**ymphocytes

TIL research and development being built out in Pittsburgh and Philadelphia by industry leading cell therapy experts

Key recruits bring significant cell therapy experience

Michael T. Lotze, M.D.
Chief Cellular Therapy Officer
- *Formerly CSO at Iovance*

Robert J. Brown, M.D.
Vice President of Clinical Development
- *Formerly Allogene and Iovance*

DeCART

Drug-**E**nhanced

Chimeric **A**ntigen **R**eceptor **T** Cell Therapy

- Wholly owned subsidiary seeded with $3M and a license to three Nurix compounds for combination use with CAR-T to enable independent investment
- Industry leader and DeCART founder **Dr. Carl June** to lead Scientific Advisory Board
- Chief Operating Officer, **Dana Hammill**, former director of strategy and business development at the Center for Cellular Immunotherapies University of Pennsylvania where she co-managed Penn-Novartis alliance for commercialization of CART19

DeTIL-0255: Clinical Development Plan

- Establishing efficient DeTIL-0255 process and manufacturing
- Study objectives:
  - Primary: safety and tolerability of DeTIL-0255 autologous cell therapy
  - Secondary: exploratory evaluation of efficacy
  - Exploratory: characterization of DeTIL-0255 phenotypes utilizing several T cell markers, identification of potential mechanisms of response or resistance to DeTIL-0255 including repertoire analysis and persistence of autologous cell therapy with study design and protocol currently under development
- Phase 1 trial in patients with advanced solid tumors who have failed standard of care at multiple sites in the U.S. that have experience with TIL/ACT trials

Nurix is Positioned for Success as We Work to Bring Targeted Protein Modulators to the Clinic

- **Optimized chemical matter** – powerful DEL screening engine and medicinal chemistry
- **Lead CTM program against validated BTK target** with readily accessible blood biomarkers to demonstrate target engagement
- **Oral T-cell activator** (CBL-B inhibitor) with potential broad solid tumor applications and ex-vivo cell therapy applications
- **Four programs** are expected to enter clinical development in 2021
- **Cash position of $395.1M** at end of fiscal Q3 2020

Wholly owned pipeline with multiple programs anticipated to be in clinical development next year

Drug Candidate	Target / Delivery	Therapeutic Area	Lead Optimization	Preclinical	Phase 1	Phase 2	Phase 3
Protein Degradation Chimeric Targeting Molecule (CTM) Portfolio							
NX-2127	BTK + IMiD activity *Oral*	B-cell Malignancies	*Late Q4 '20 – Q1 '21				
NX-5948	BTK *Oral*	B-cell Malignancies And GVHD	*H2 '21				
Ligase Inhibition Portfolio							
NX-1607	CBL-B *Oral*	Immuno-oncology	*Q3 '21				
DeTIL-0255	CBL-B *ex vivo*	Adoptive Cell Therapy (ACT)	*H2 '21				

*Expected IND submission timing based on calendar year quarters

Thank you

Nurix Therapeutics