nurix

Leader in Targeted Protein Modulation

Targeted Protein Degraders for the Treatment of Hematologic Malignancies:
Addressing the Mutational Resistance of BTK in the Clinic

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Protein Degradation & Targeting Undruggables Congress Europe
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Nurix Drugs Engage Ligases for the Treatment of Cancer

Targeted Protein Modulation: TPM = TPD + TPE

A Powerful Cellular System

Targeted Protein Elevation (TPE)

Inhibit ligases to increase specific protein levels

Harness ligases to decrease specific protein levels

Targeted Protein Degradation (TPD)

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

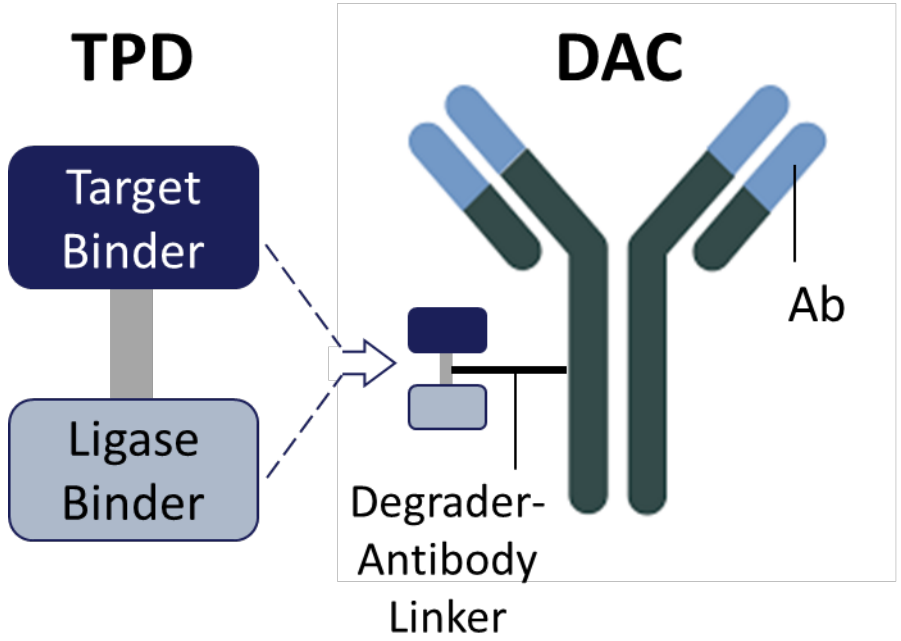
Nurix Is Advancing a Pipeline of Propriety and Partnered Programs in Oncology and Autoimmune/Inflammatory Diseases

MOA	Drug program	Target	Therapeutic area	Discovery	IND enabling	Phase 1a	Phase 1b
TPD	**NX-2127**	BTK-IKZF	B-cell malignancies				
TPD	**NX-5948**	BTK	B-cell malignancies				
TPE	**NX-1607**	CBL-B	Immuno-Oncology				
TPD	**NX-0479 / GS-6791**	IRAK4	Rheumatoid arthritis and other inflammatory diseases				GILEAD
TPM	5 programs	Undisclosed	Oncology / autoimmune disease				
TPD	4 programs	Undisclosed	Undisclosed				GILEAD
TPD	5 programs	Undisclosed	Undisclosed				sanofi
DAC	Multiple programs	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)



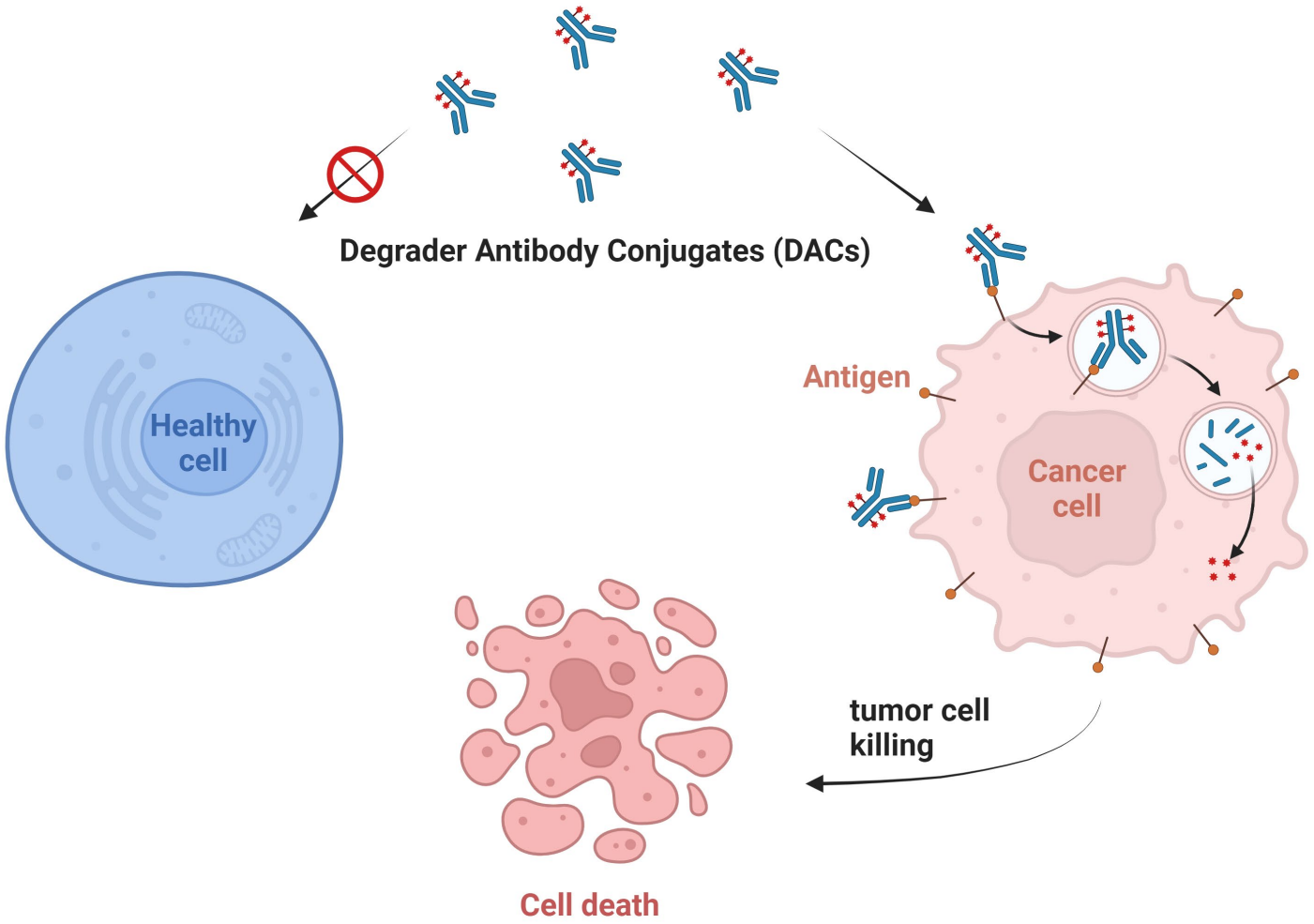
Deal Terms

- $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®

DACs: Tumor-Specific Delivery of Potent Degraders

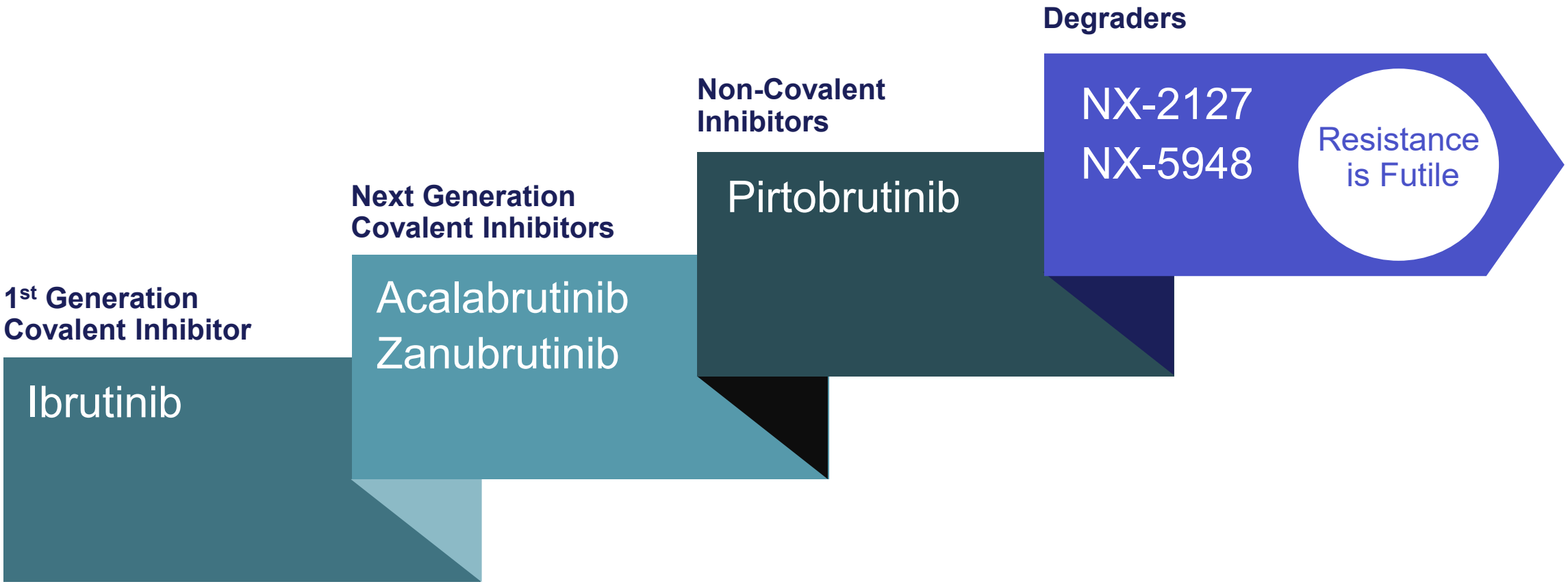
The next generation of ADCs



- Degraders replace highly toxic ADC payloads
- DACs allow for selective delivery of degraders to tumor cells
- Degraders eliminate driver proteins essential for cancer cell growth and survival

Note: Four degraders shown per antibody is illustrative; the actual number may vary.

Evolution of BTK Targeted Therapies

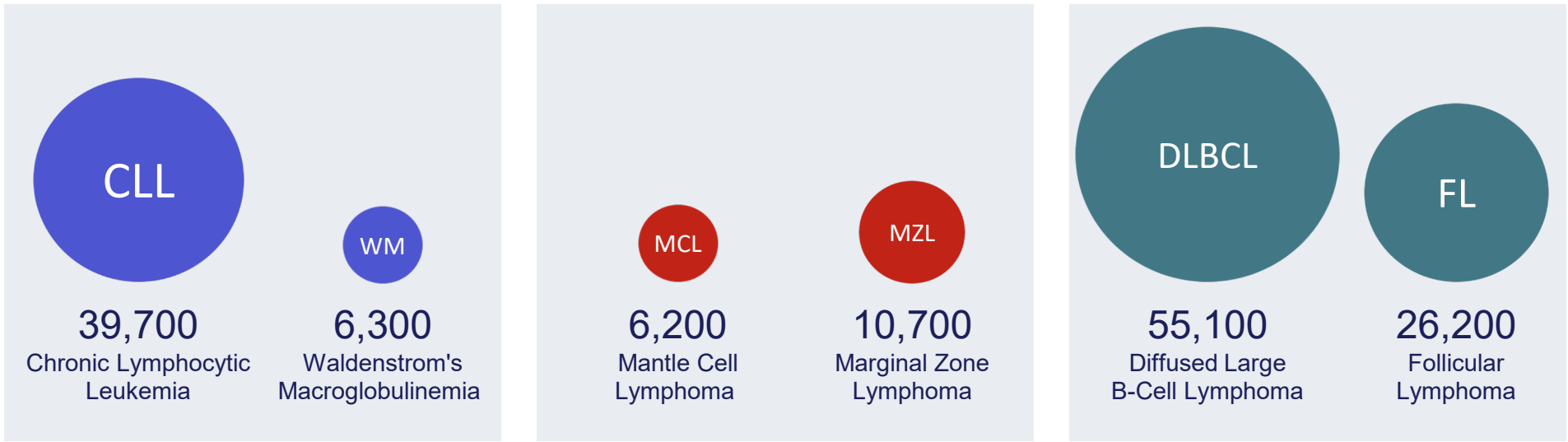


Nurix BTK Degraders: Two BTKs Degraders to Cover the Landscape of B-Cell Malignancies

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

B-Cell Malignancies Annual Incidence (US & EU)



NX-5948

NX-5948 potential in combination

NX-2127 in 3L+

NX-2127

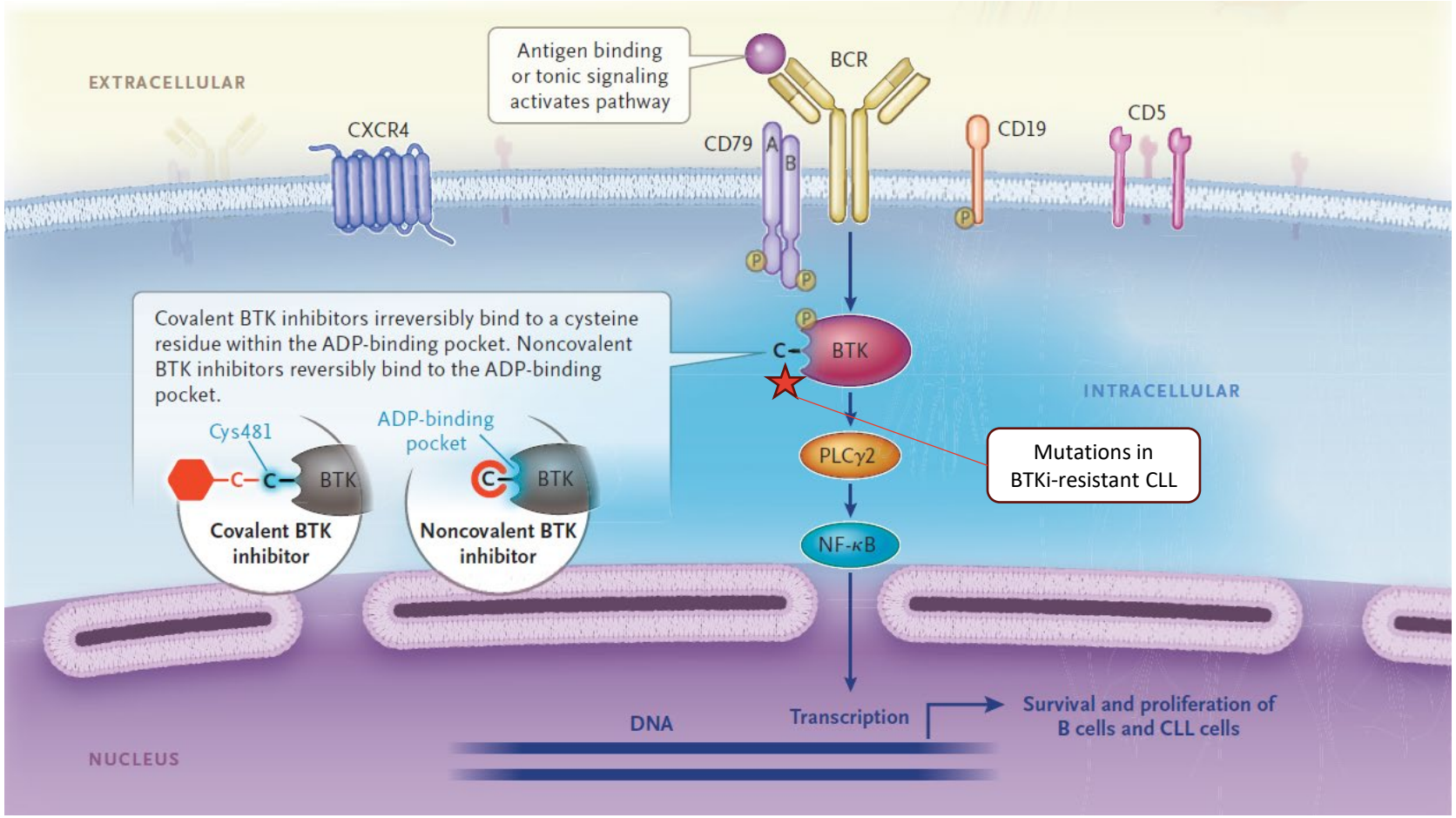
Goal: Displace BTKi in indications where single-agent BTKi is standard of care and address BTKi resistance mutations

Goal: Expand role for BTK target therapy

BTK, Bruton tyrosine kinase; DLBCL, Diffuse large B cell lymphoma; CLL, Chronic lymphocytic leukemia, SLL, small lymphocytic lymphoma; 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

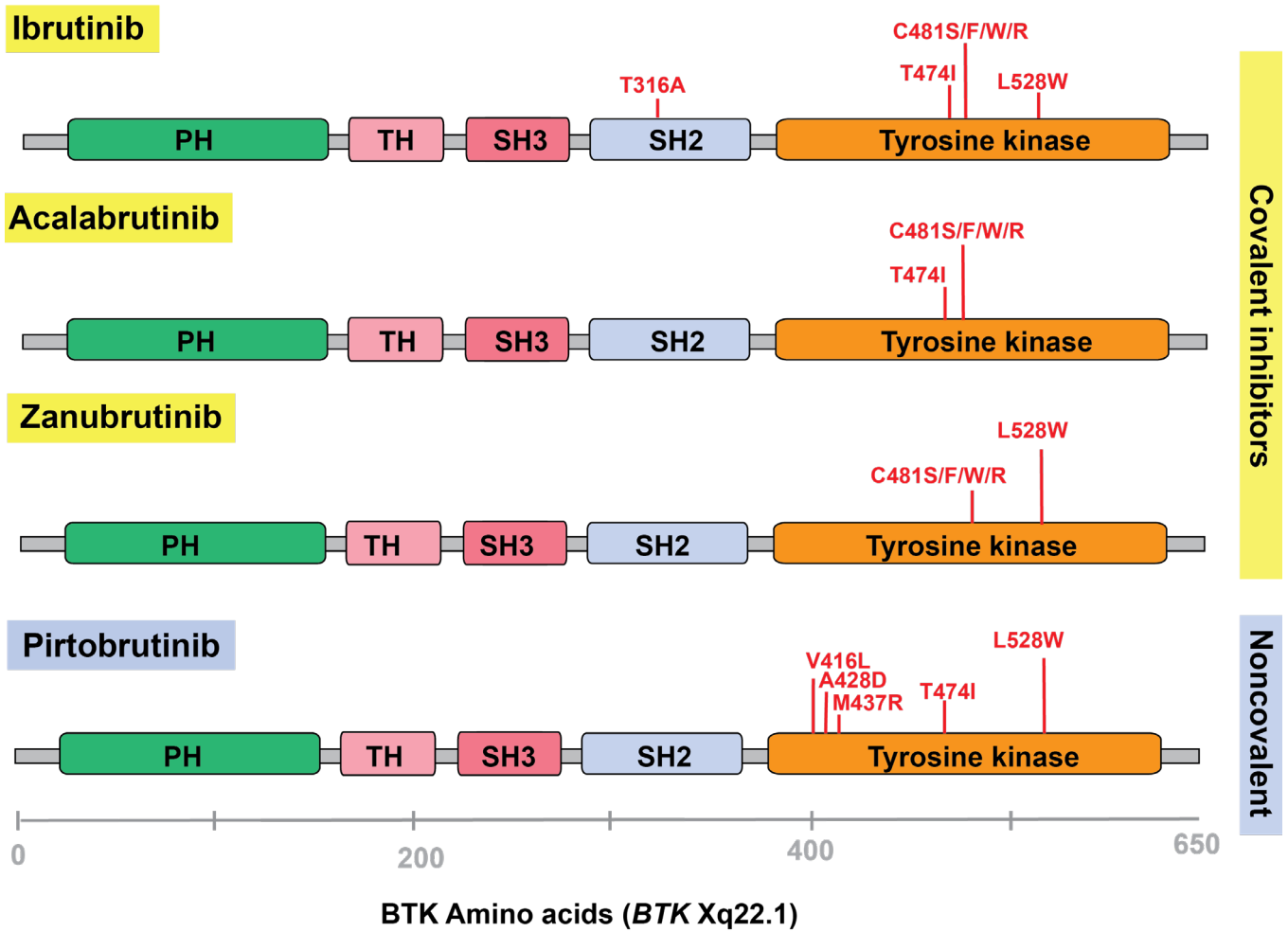
B-cell Malignancies Respond to BTK inhibitors, But Patients Eventually Progress as a Consequence of Inhibitor Resistance

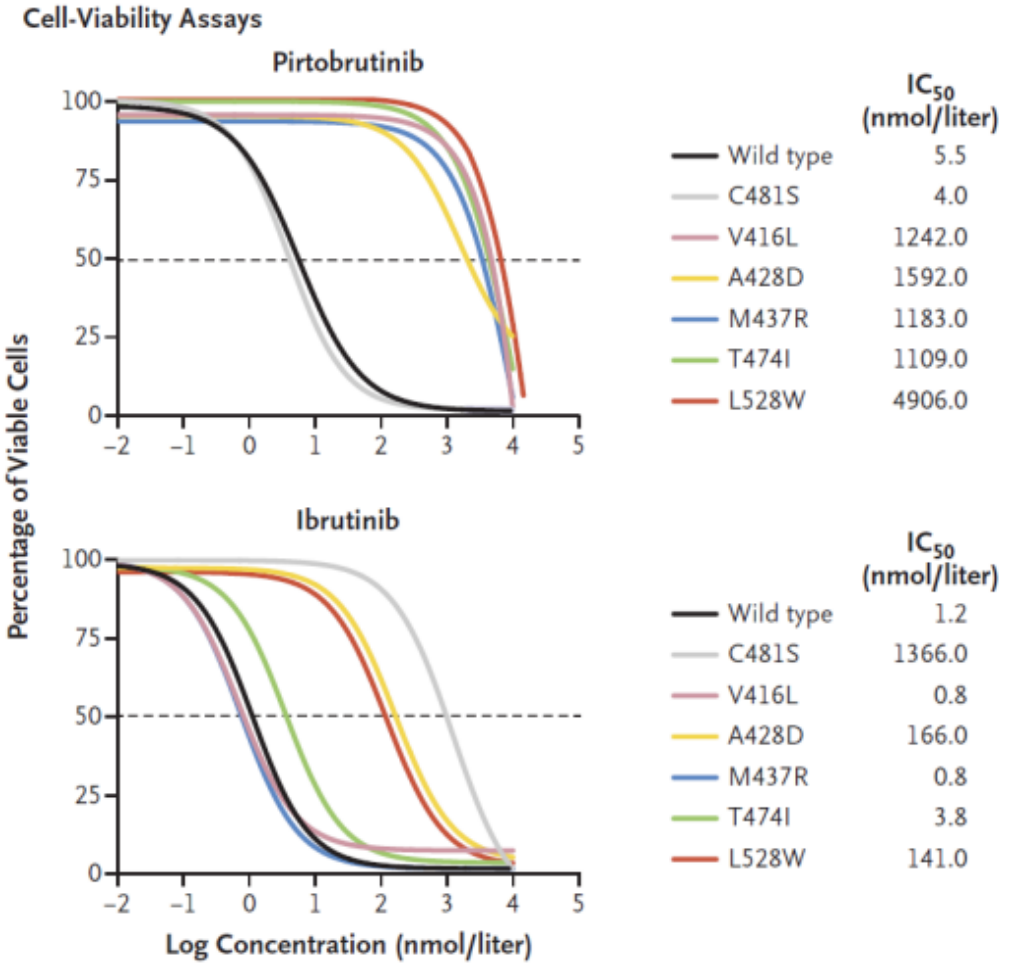


Modified from Kater et al., NEJM 2023 and Nakhoda et al., Br. J. Haematol. 2023

- BTK is a nonreceptor tyrosine kinase and plays a crucial role in the B-cell receptor (BCR) signaling pathway
- Inhibition of BTK enzymatic activity has been established as an effective therapeutic strategy
- All patients eventually progress, and majority carry mutations in *BTK*C481 when treated with ibrutinib; ~53-87% of patients. Rates are similar for acalabrutinib; 69% of patients

Treatment-Emergent Resistance to Inhibitors Is Evolving:
Noncovalent BTK inhibitor Resistance Shows Broader Mutation Profile

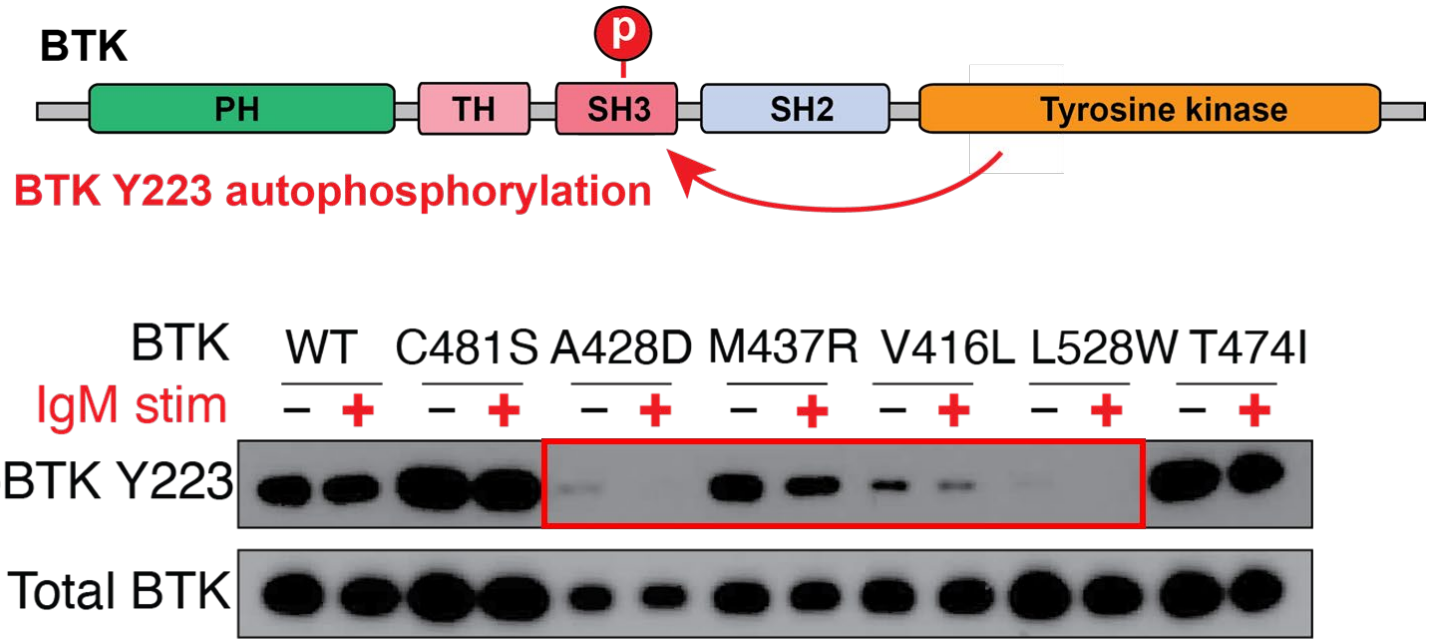




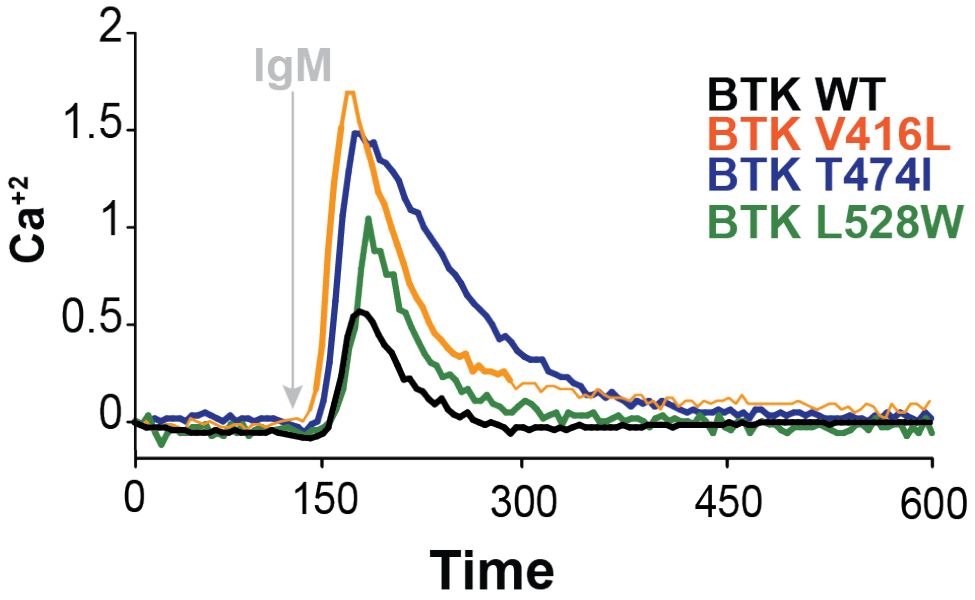
Wang, Mi, Thompson, et al. NEJM 2022

Inhibitor-Induced BTK Mutations Abrogate Phosphorylation Yet Propagate Downstream BCR Signaling: *Mutant BTK is 'Undruggable'*

BTK Y223 Phosphorylation in TMD8 Cell



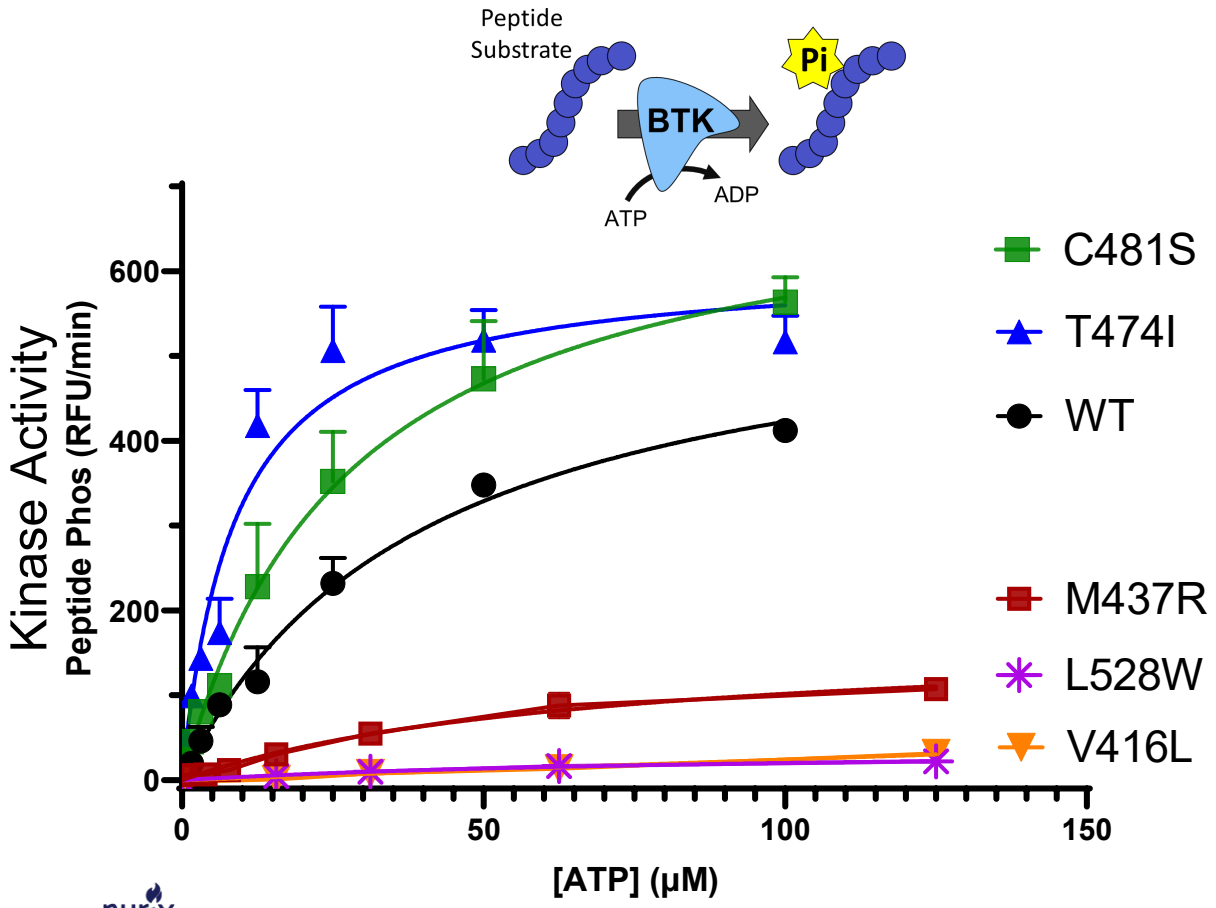
Calcium Ion Flux in TMD8 Cell



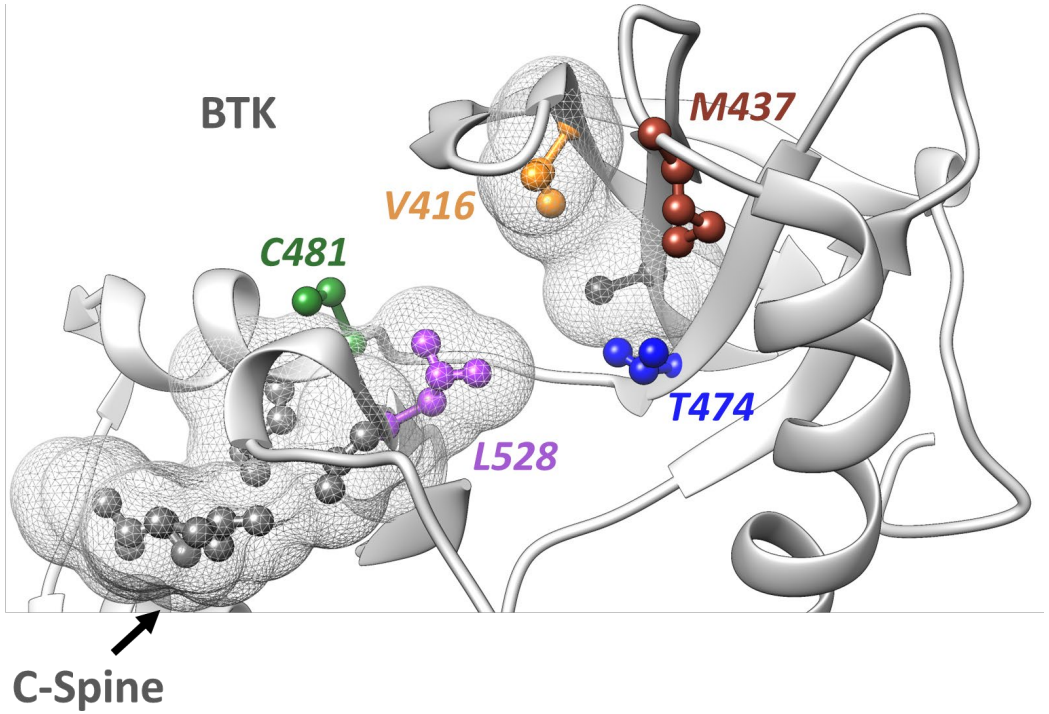
Montoya et al., ASH 2022

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

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



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



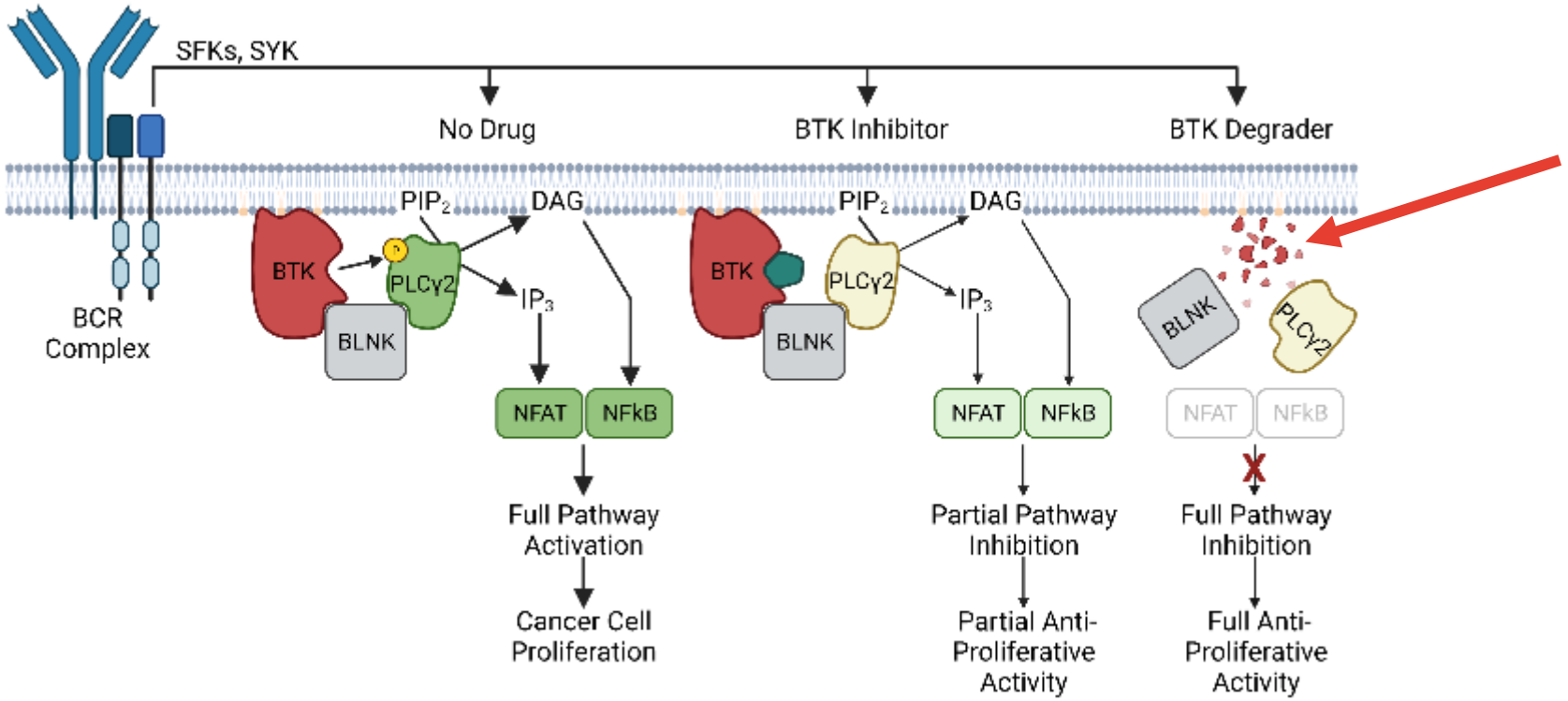
Montoya et al., ASH 2022 and unpublished data

BTK Degraders Offer an Approach to Better Control BCR Signaling

Scaffolding Function of BTK was Previously Unrecognized as a Signaling Driver

Degraders Have the Potential to Eliminate Both the Enzymatic and Scaffolding Function of BTK

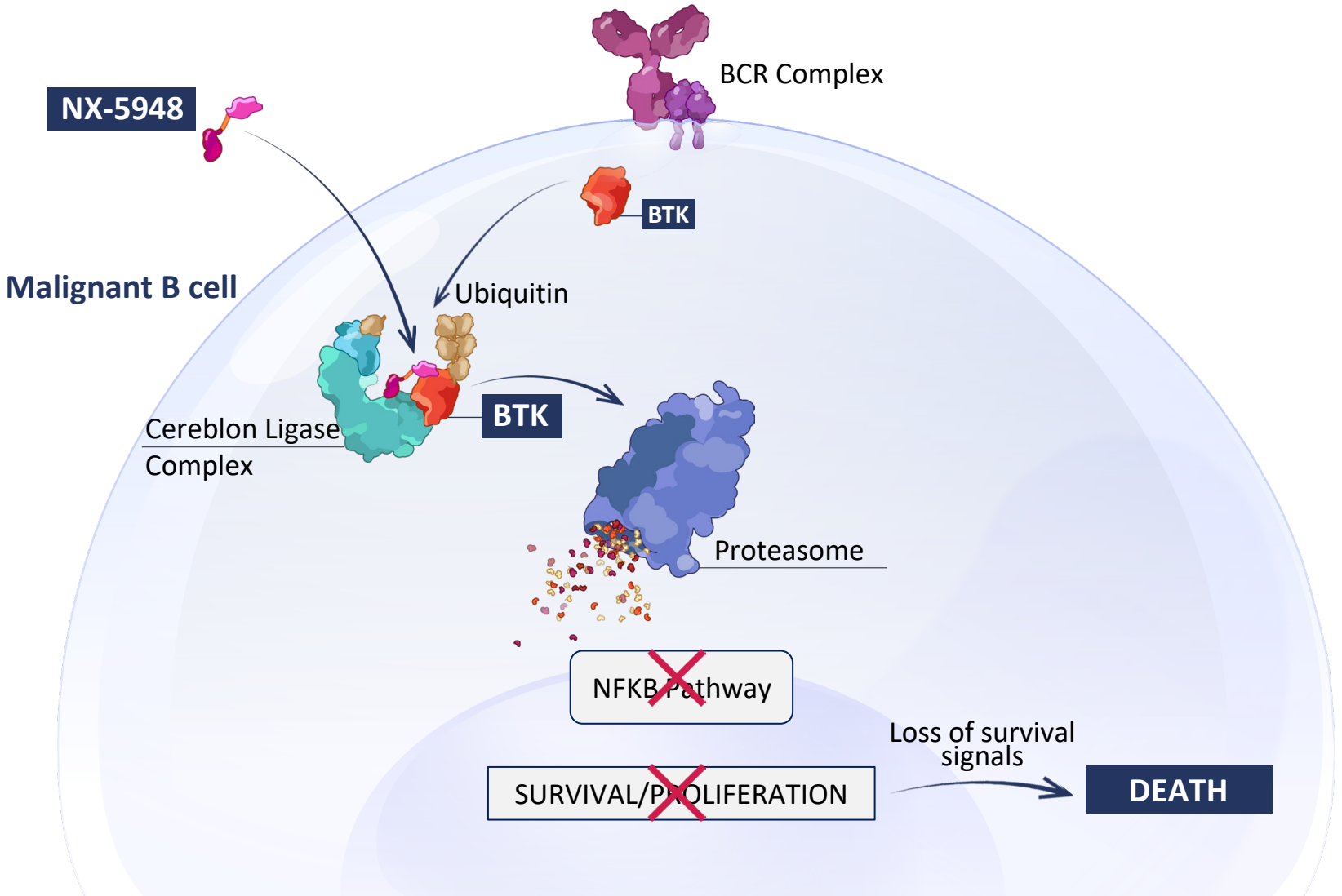
Degraders May be More Effective for Treating BCR-signaling Driven Disease



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

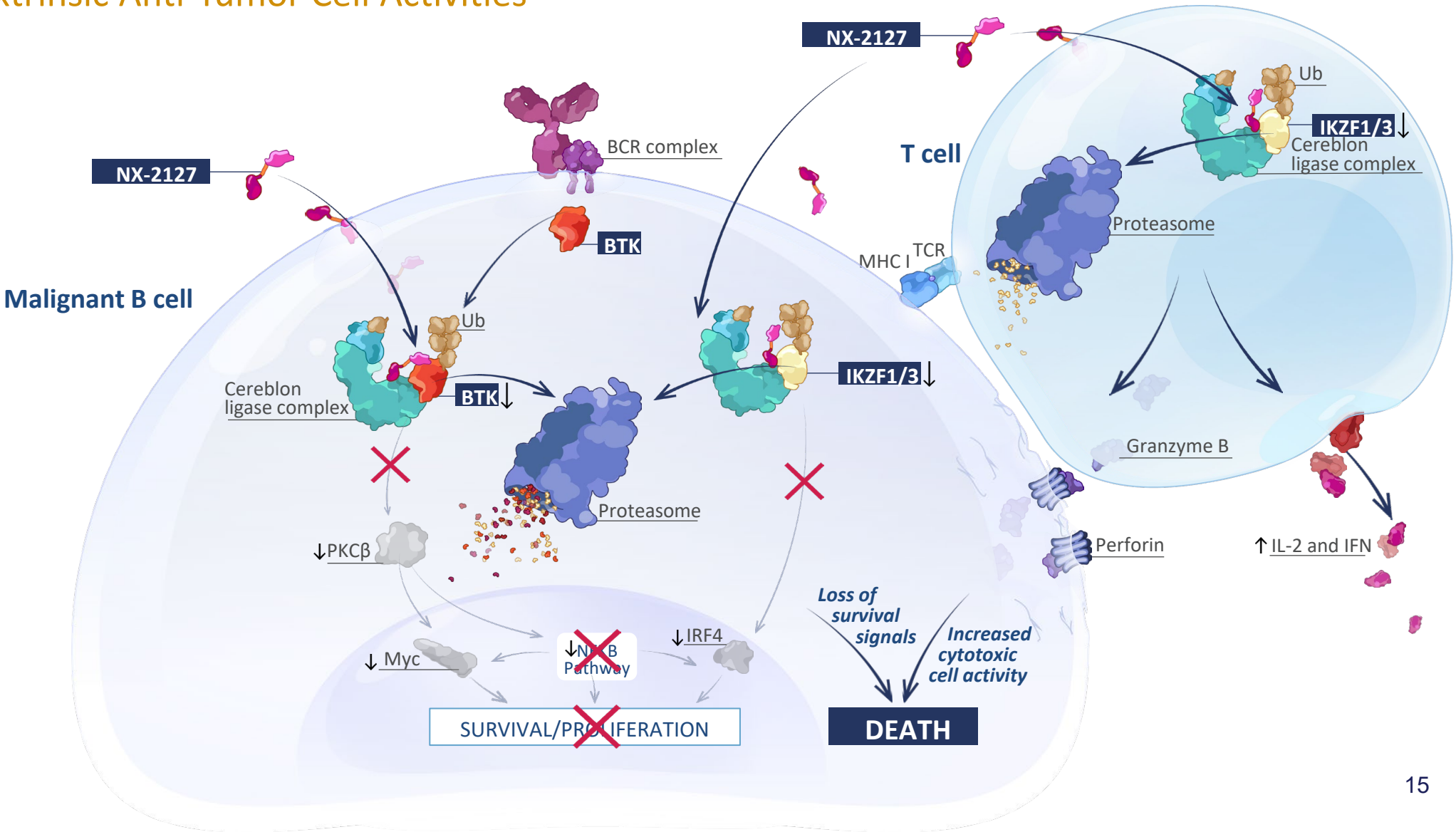
NX-5948 is a Potent and Selective Degrader of BTK

Targeted Degradation of Bruton's Tyrosine Kinase Can Address BTKi Resistance



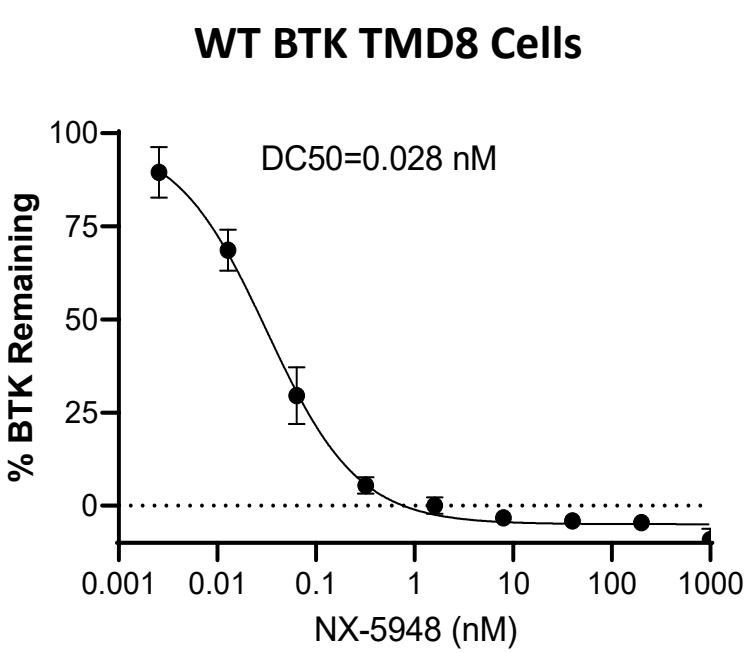
NX-2127 Dual Mechanism of Action

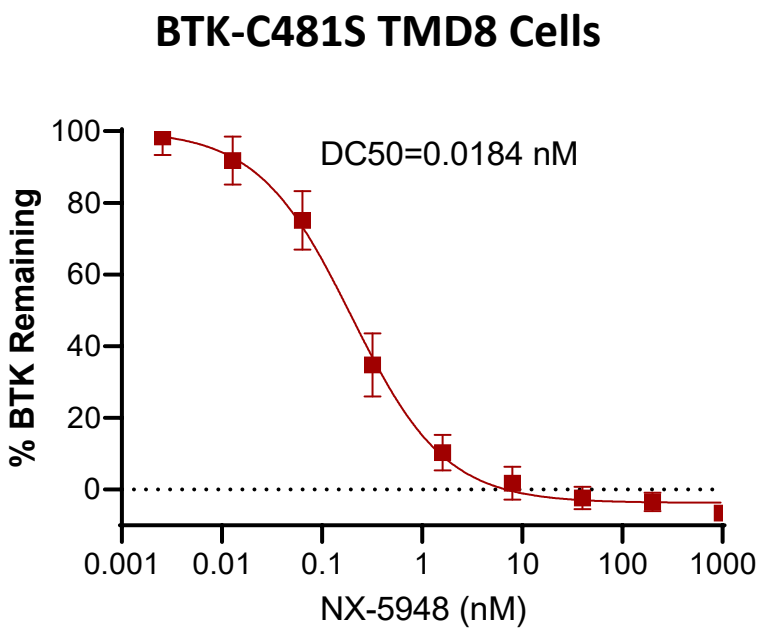
Targeted Degradation of BTK and IKZF1/3 Provides Both Intrinsic and Extrinsic Anti-Tumor Cell Activities

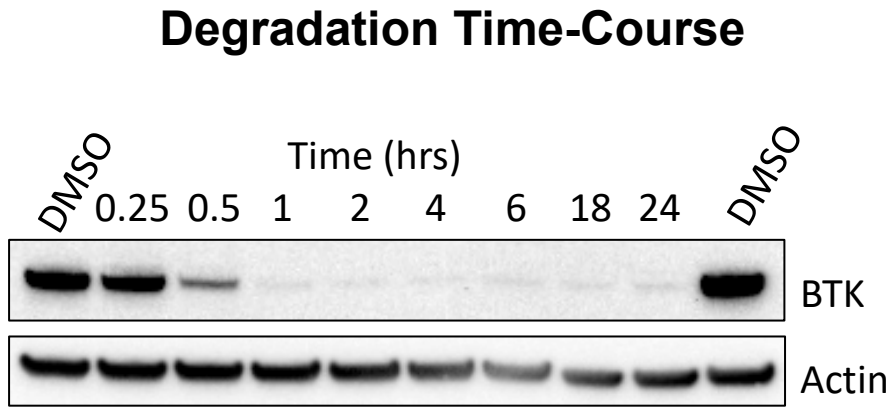


Nurix BTK Degraders Were Designed for Potent and Rapid Degradation of Wildtype and C481S-Mutant BTK

- NX-5948 degrades WT and mutant forms of BTK with sub-nanomolar potency in ABC DLBCL TMD8 cells
- BTK degradation is observed within 1 hour and is complete within 2 hours in Ramos cells





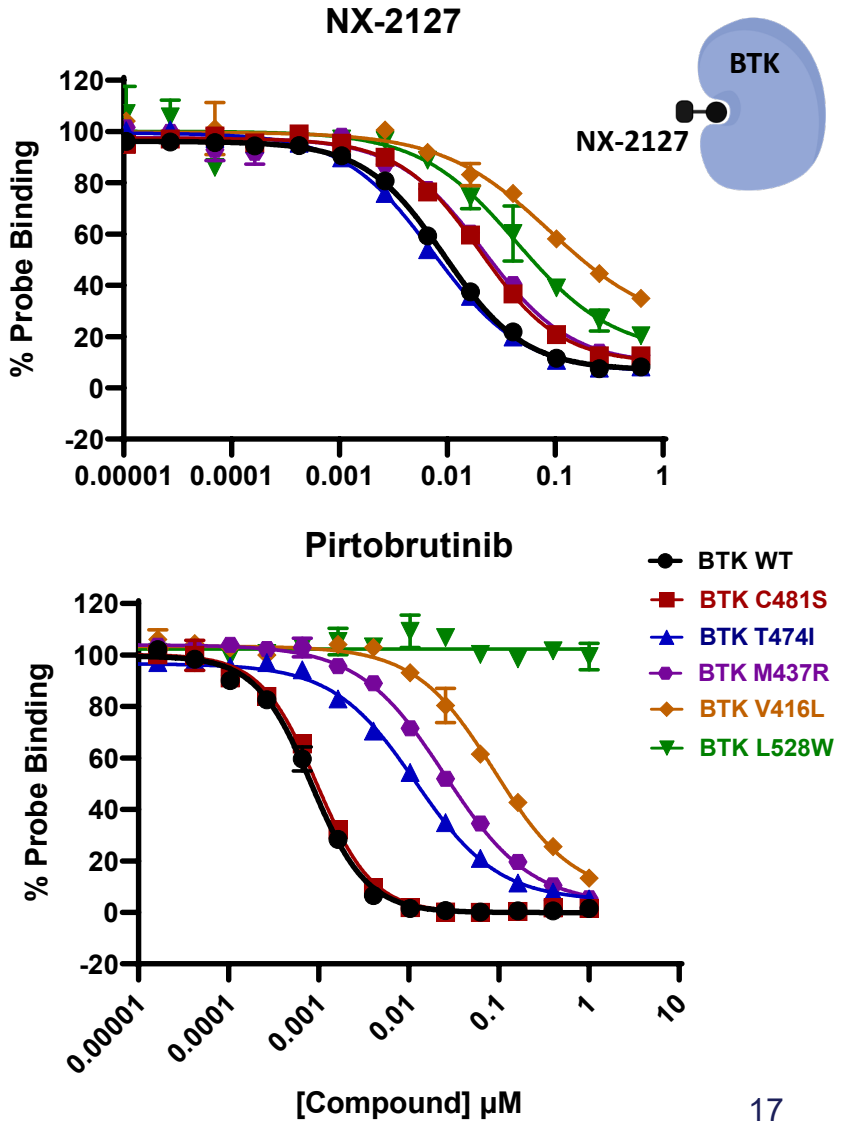


TMD8 cells harboring WT BTK or a knock-in BTK mutation (C481S) were incubated with NX-5948 for 24 hours, and BTK degradation was assessed by flow cytometry. Ramos human Burkitt's lymphoma B cells incubated with 10 nM NX-5948 and assessed by western blot

NX-2127 Demonstrates Binding to Recurrent Acquired Resistant BTK Mutant Variants

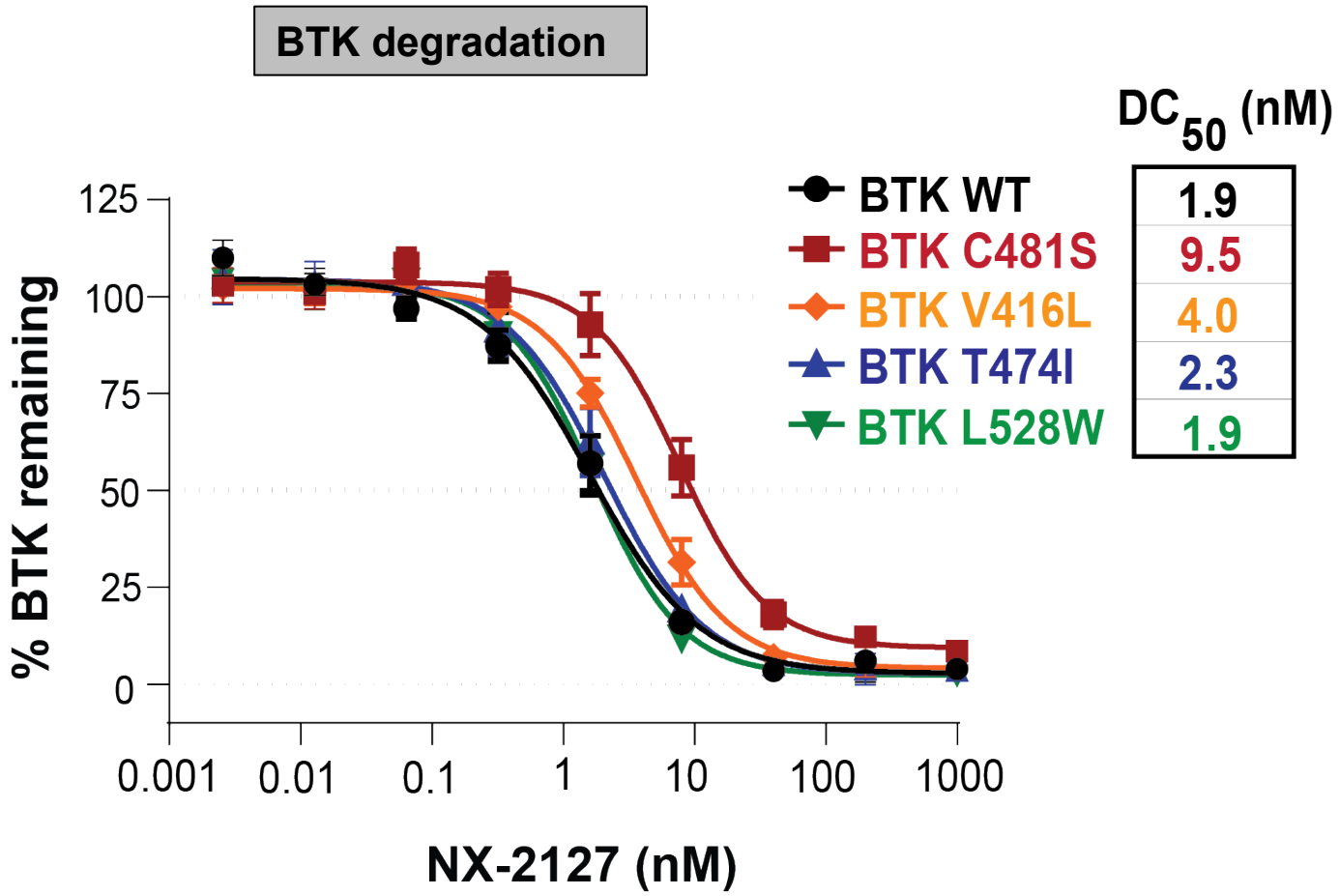
BTK Protein	Binding of **NX-2127** Determined by SPR K_d (nM)	Binding Determined by FRET Displacement Assay IC_{50} (nM)		
		NX-2127	Pirtobrutinib	Ibrutinib
WT	18	10	0.76	1.4
C481S	45	22	0.77	6.2
T474I	18	8.6	12	1.8
M437R	44	23	30	0.28
V416L	97	165	98	3.8
L528W	88	70	>1000	>1000

*IC_{50} determined at 60 min for Ibrutinib
IC_{50}s reported here are mean of at least 3 experiments

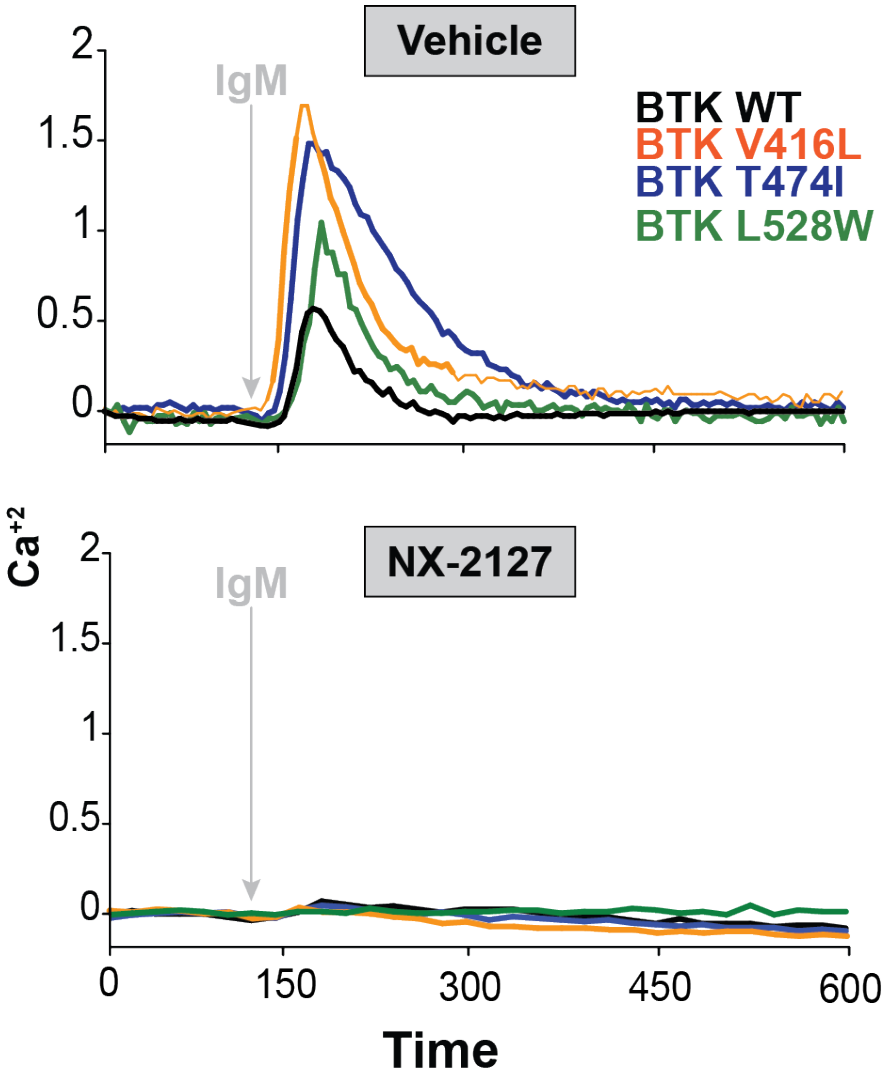


unpublished data

NX-2127 Degrades Both Wild-Type and Kinase Dead BTK and Suppresses Ca^{++} Signaling



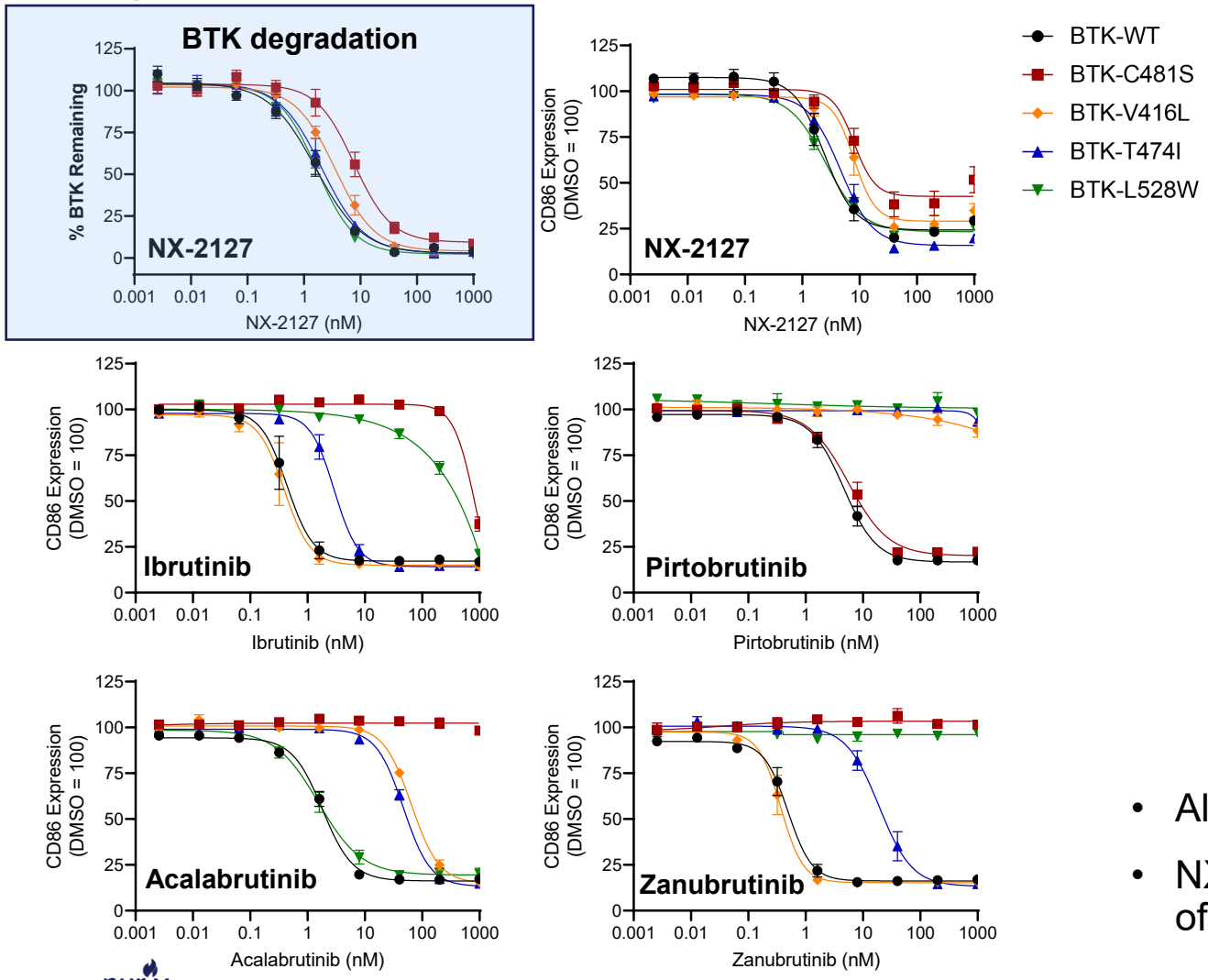
DC_{50} (nM)
1.9
9.5
4.0
2.3
1.9



Montoya et al., ASH 2022

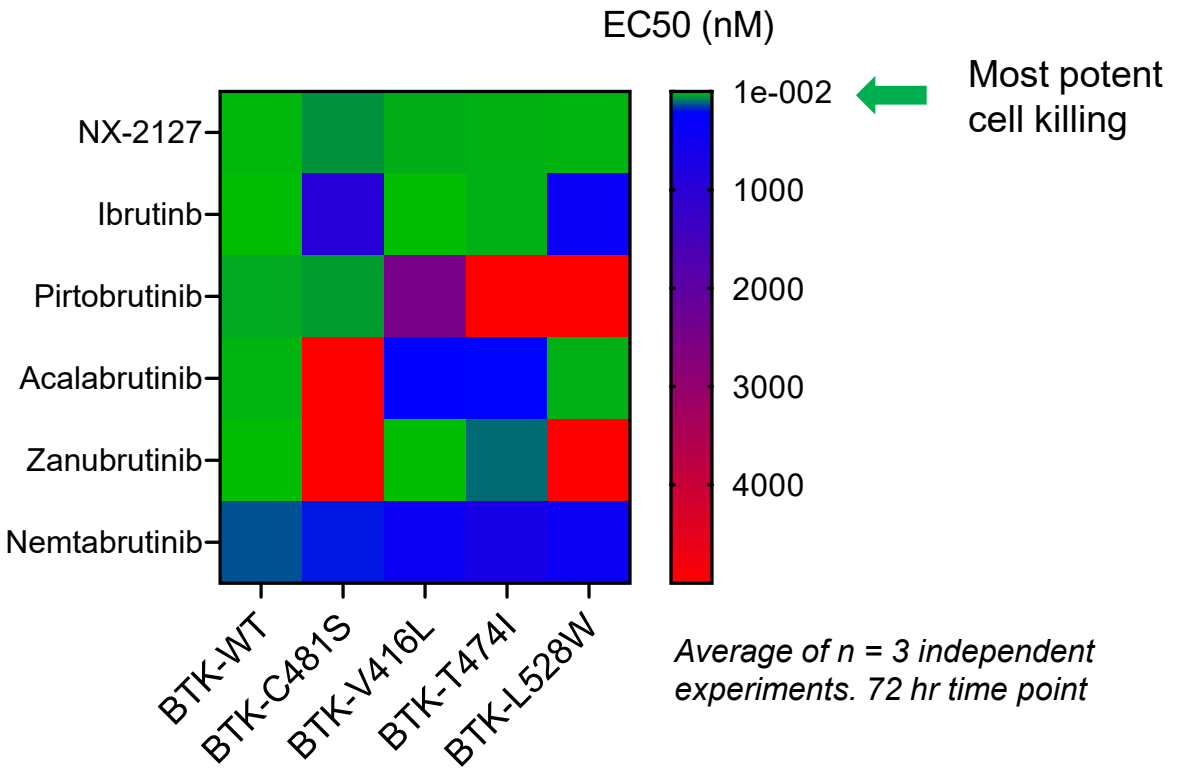
NX-2127 Is Potent and More Broadly Active Than All BTK Inhibitors Tested

BTK degradation and activation marker suppression in TMD8 tumor cells



Average of n = 3 independent experiments +/- SEM

TMD8 tumor cell killing

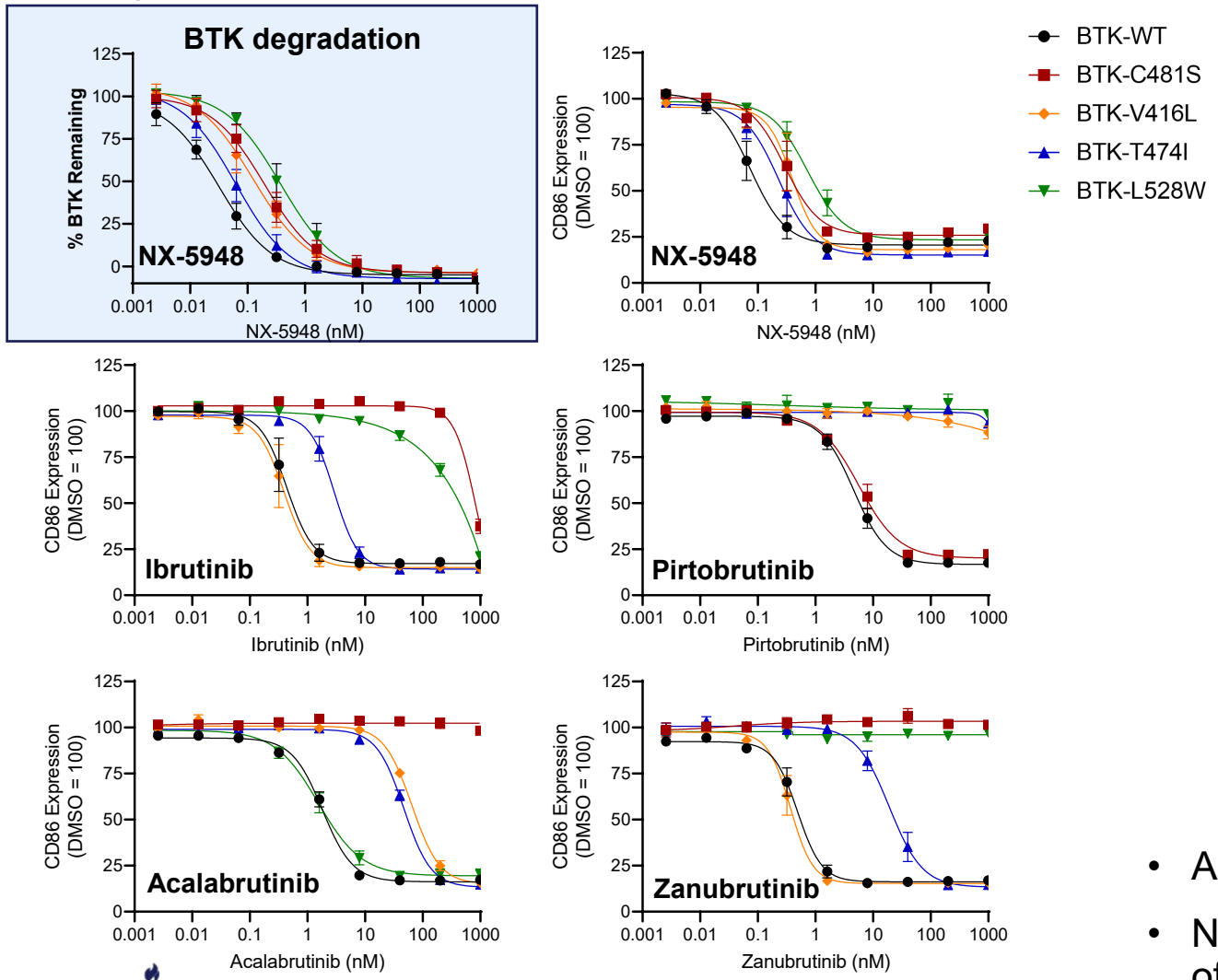


Average of n = 3 independent experiments. 72 hr time point

- All inhibitors have resistance mutation liabilities
- NX-2127 displays potent cell killing and maintains suppression of CD86 in the context of key resistance mutations

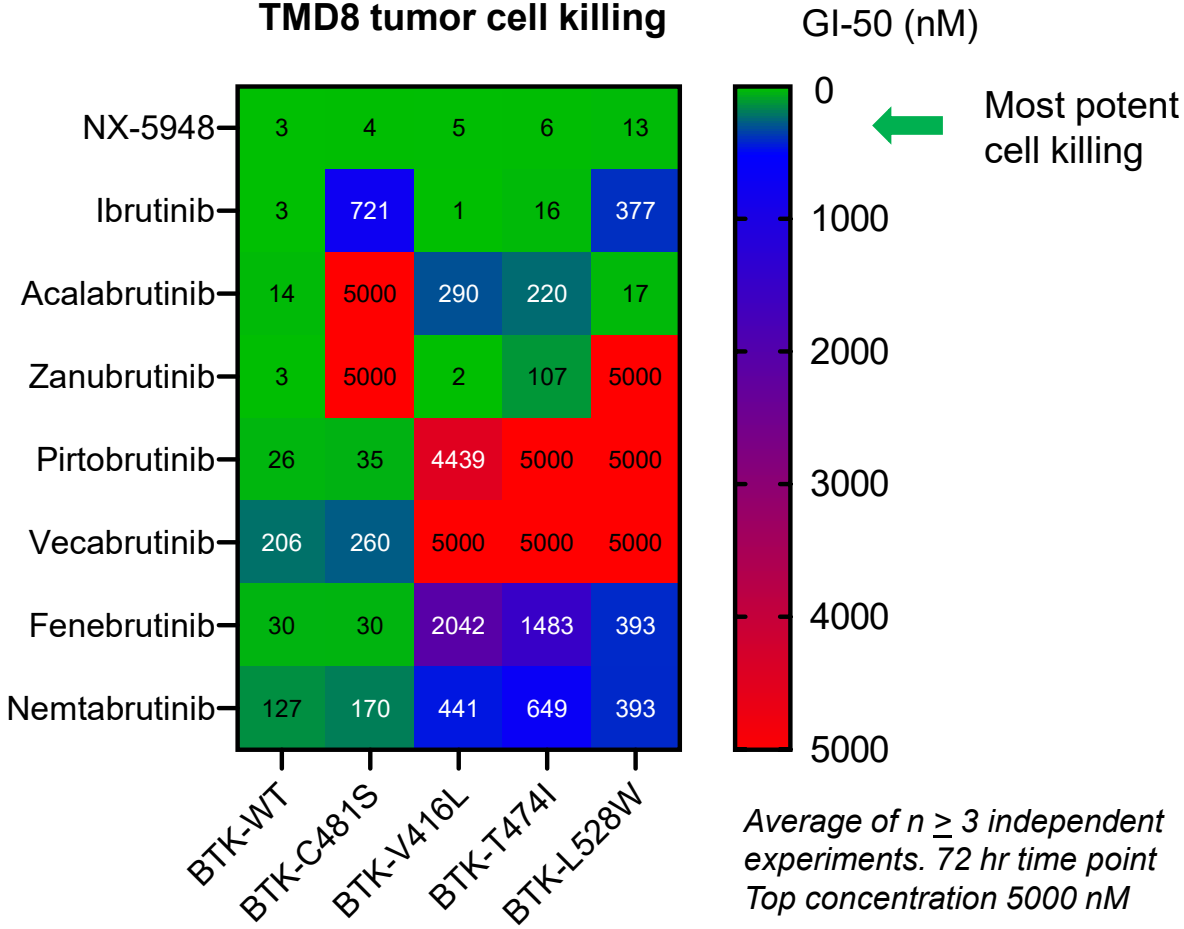
NX-5948 Is More Potent and Broadly Active Than All BTK Inhibitors Tested

BTK degradation and activation marker suppression in TMD8 tumor cells



Average of n = 3 independent experiments +/- SEM

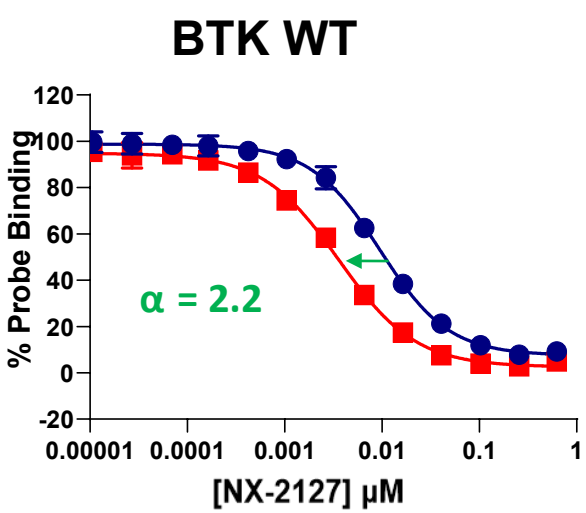
TMD8 tumor cell killing

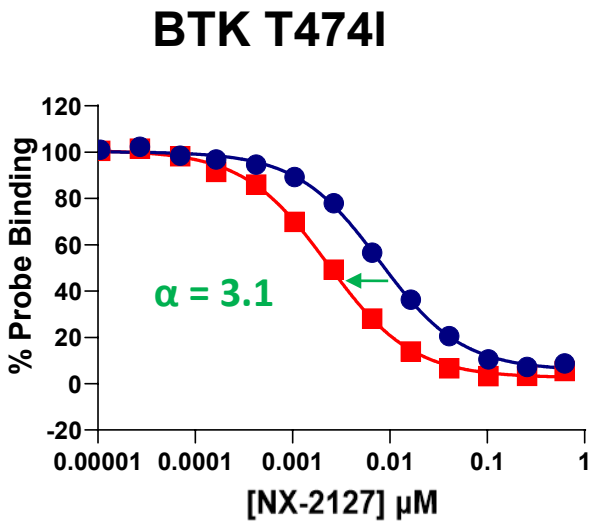


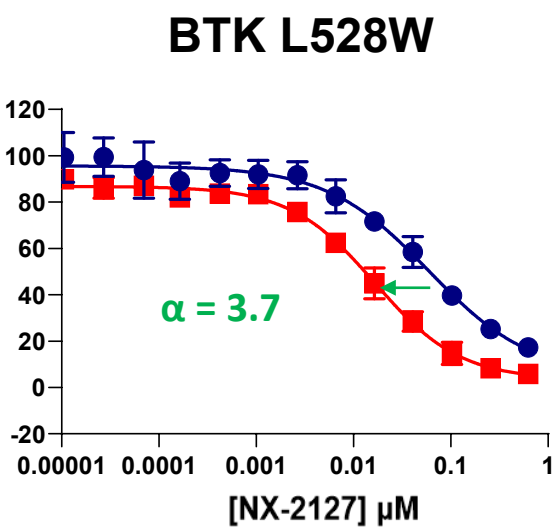
Average of n ≥ 3 independent experiments. 72 hr time point Top concentration 5000 nM

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

Nurix BTK Degraders Form Stable Ternary Complexes Between BTK and CRBN Irrespective of Mutation Status



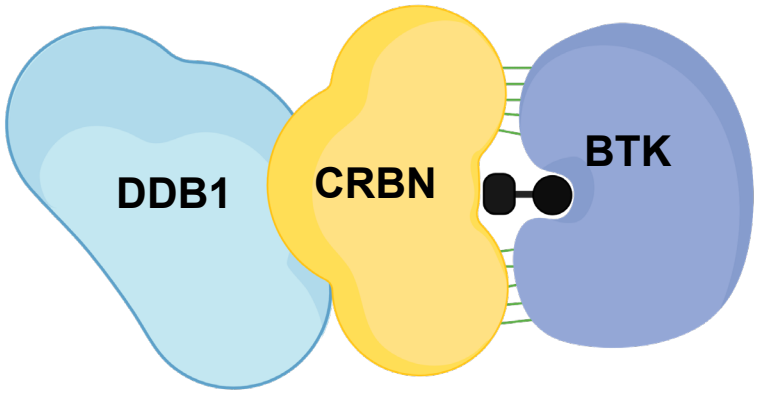




No CRBN

⊕ CRBN (1 μM)

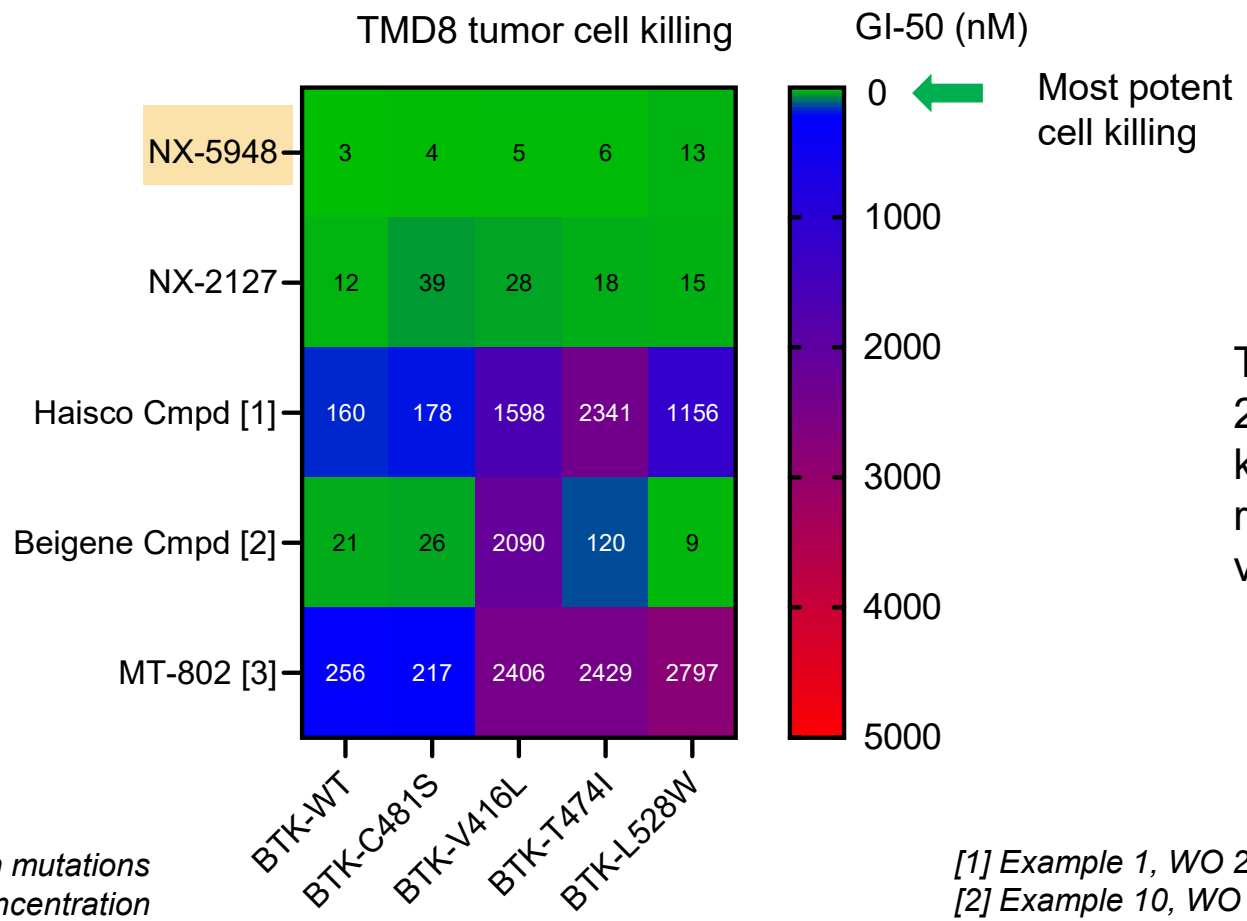
$$\alpha = \frac{IC_{50}\ \text{No CRBN}}{IC_{50}\ \text{with CRBN (1 μM)}}$$



- 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.

Not All BTK Degraders Are Created Equal



The ability of NX-5948 and NX-2127 to induce TMD8 tumor-cell killing was compared to other reported degraders in a 72-hour viability assay

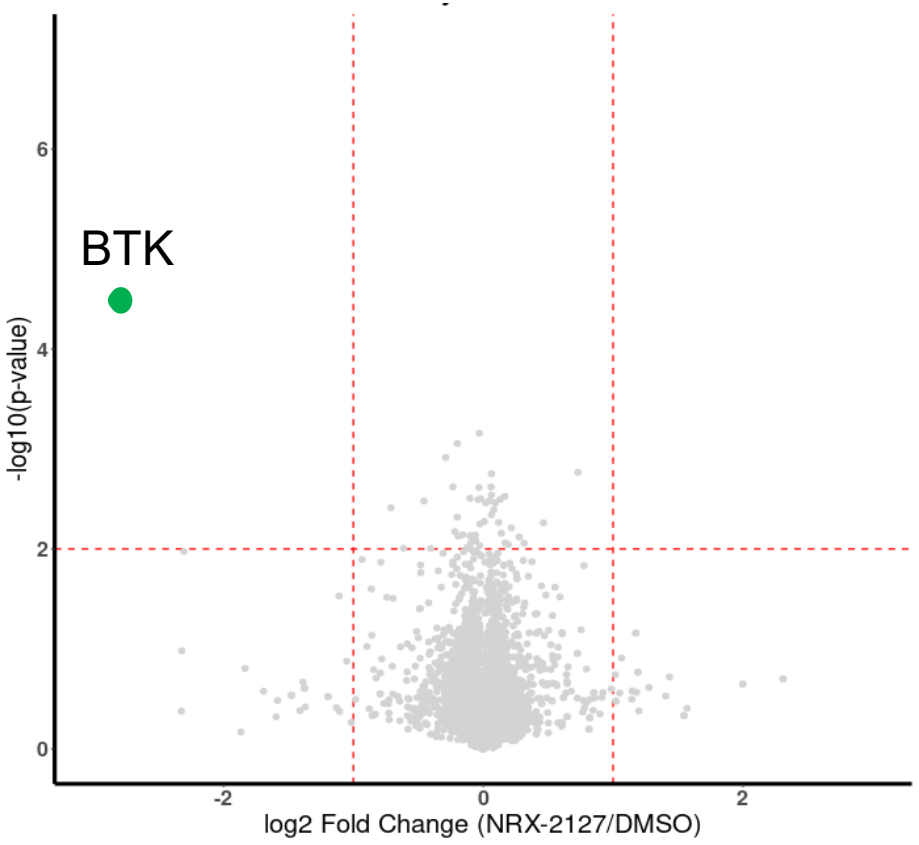
TMD8 cells with knock-in mutations
72 hr time point, 5000 nM top concentration
Average of n ≥ 4 independent experiments

[1] Example 1, WO 2022/111449 (Haisco)
[2] Example 10, WO 2021/219070 (BeiGene)
[3] Buhimschi et al. 2018. Biochemistry 57(26): 3564-3575.

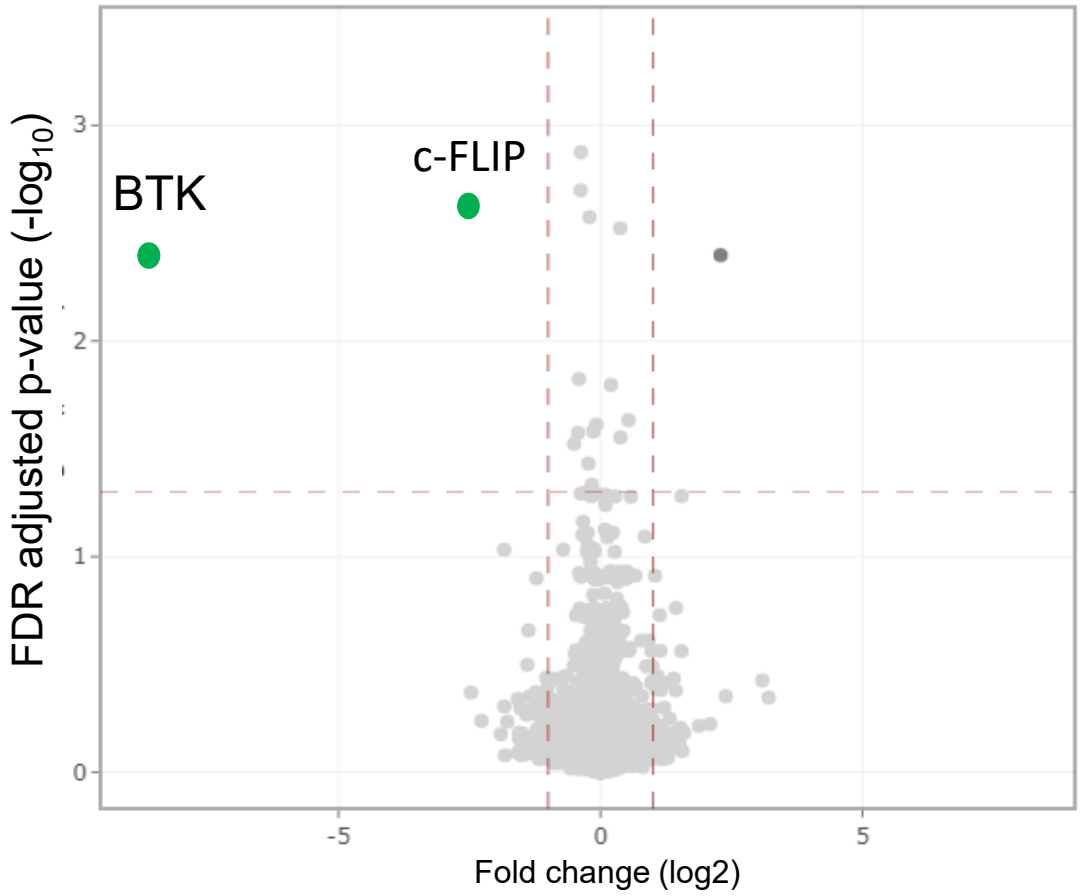
Targeted Protein Degraders Can Display Exquisite Selectivity

Global Proteomics Analysis in Human PBMCs or TMD8 DLBCL Cells

NX-2127, 50 nM, 4h, human donor PBMCs

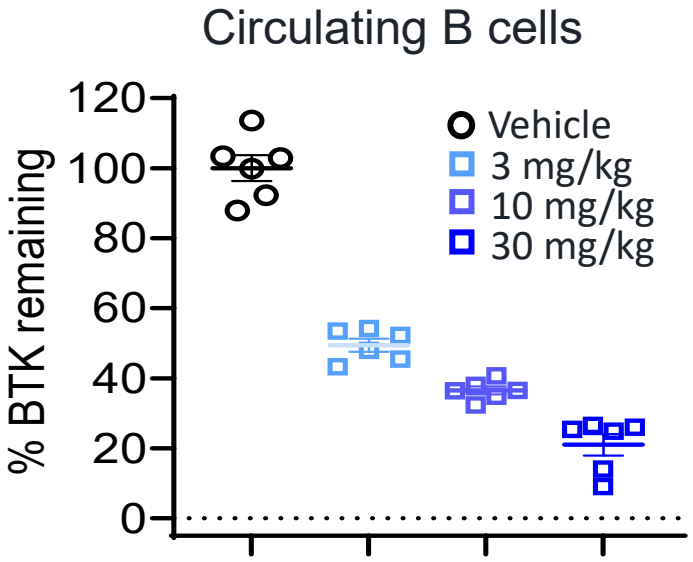


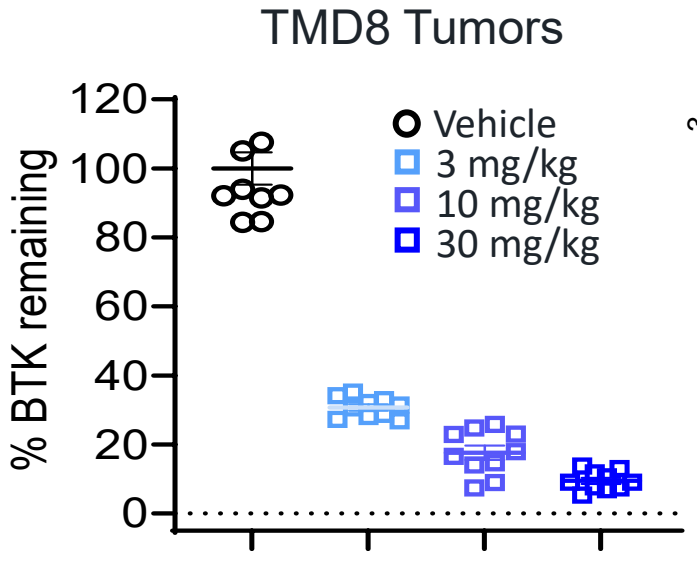
NX-5948, 50nM, 6h, TMD8 DLBCL cells

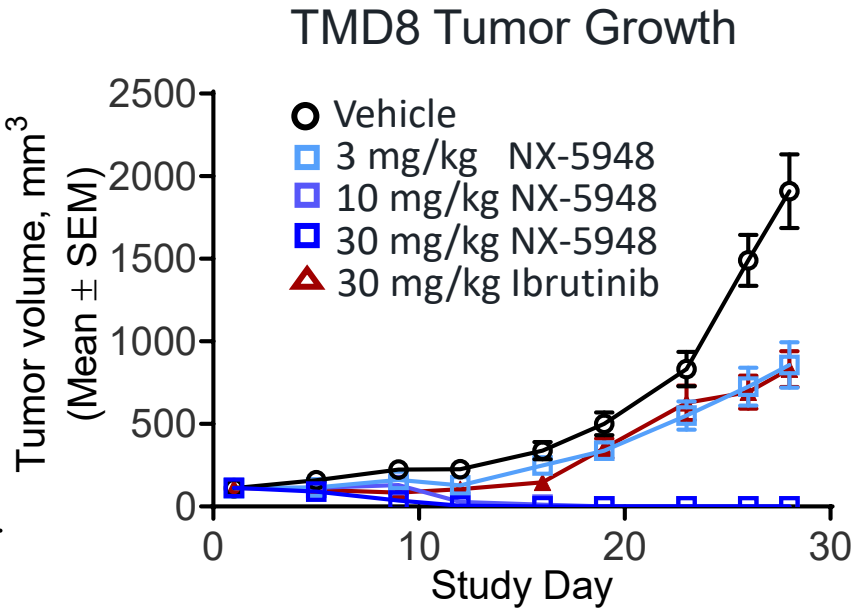


c-FLIP is an anti-apoptotic protein required to maintain survival of ABC DLBCL cells that is regulated by the BCR/NF-κB signaling axis. BTK inhibition by ibrutinib also downregulates c-FLIP in TMD8 cells (*Nagel; Onco Target; 2015*) c-FLIP reduction is believed to be a secondary effect of BCR/BTK signaling loss.

Degradation of BTK by NX-5948 Correlates with Significant Tumor Growth Inhibition







Treatment	Oral gavage dose (mg/kg)	% BTK degradation in circulating B cells	% BTK degradation in TMD8 tumor tissue	% TGI vs Vehicle (Day 26)	*P* value vs Vehicle
Vehicle	0	0.0±3.7	0.0±4.7	N/A	N/A
NX-5948	3	50.5±1.9	69.2±0.9	54%	0.0025
	10	63.5±1.1	82.4±2.1	100%	<0.0001
	30	79.0±3.1	90.5±0.5	100%	<0.0001
Ibrutinib	30	N/A	N/A	57%	0.0015

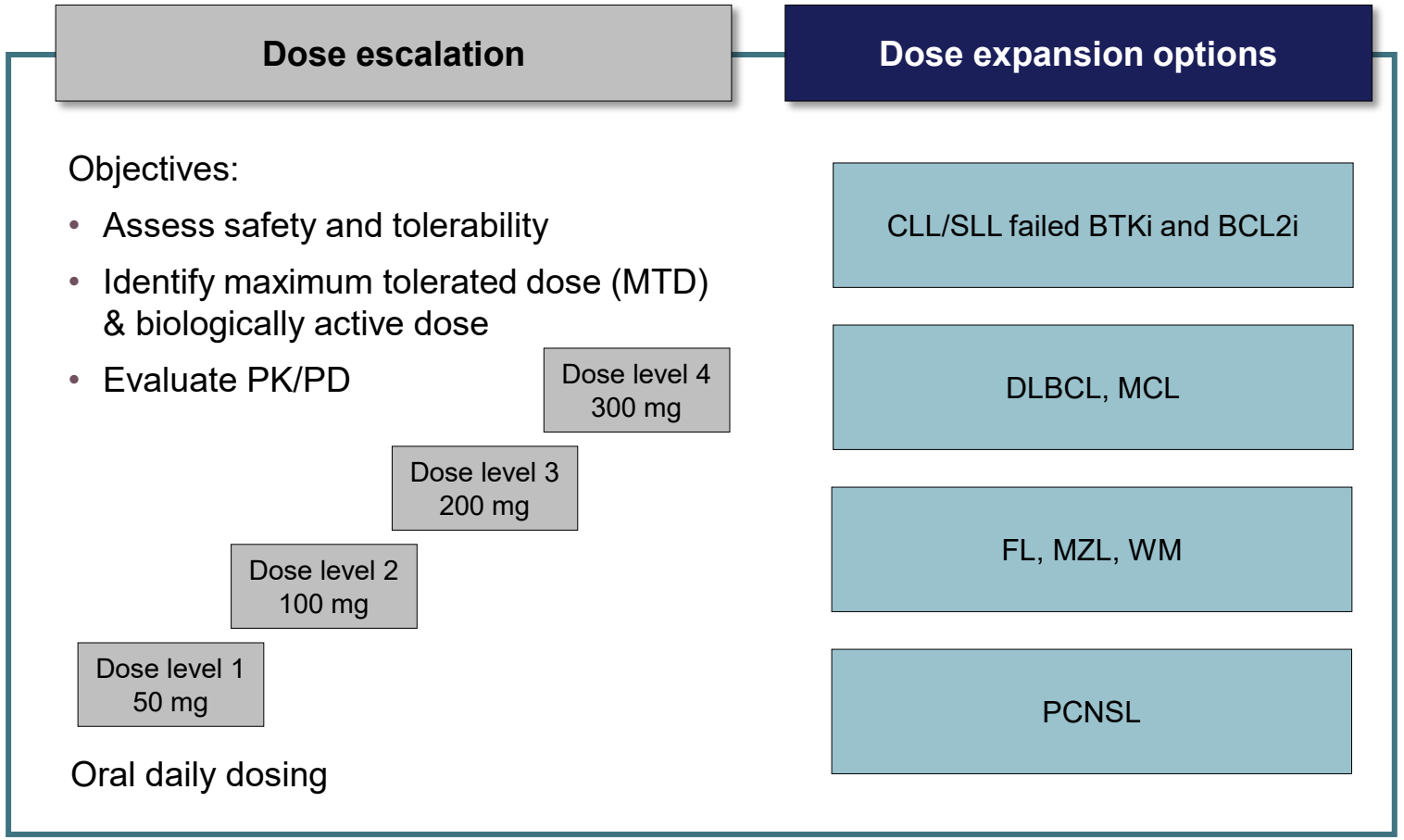
N/A: Not applicable; TGI: tumor growth inhibition.
P values determined on tumor volume by mixed-effect analysis with Dunnett's multiple comparisons test

nurix

Rountree et al., TPD 2022

NX-5948-301: Trial Design

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

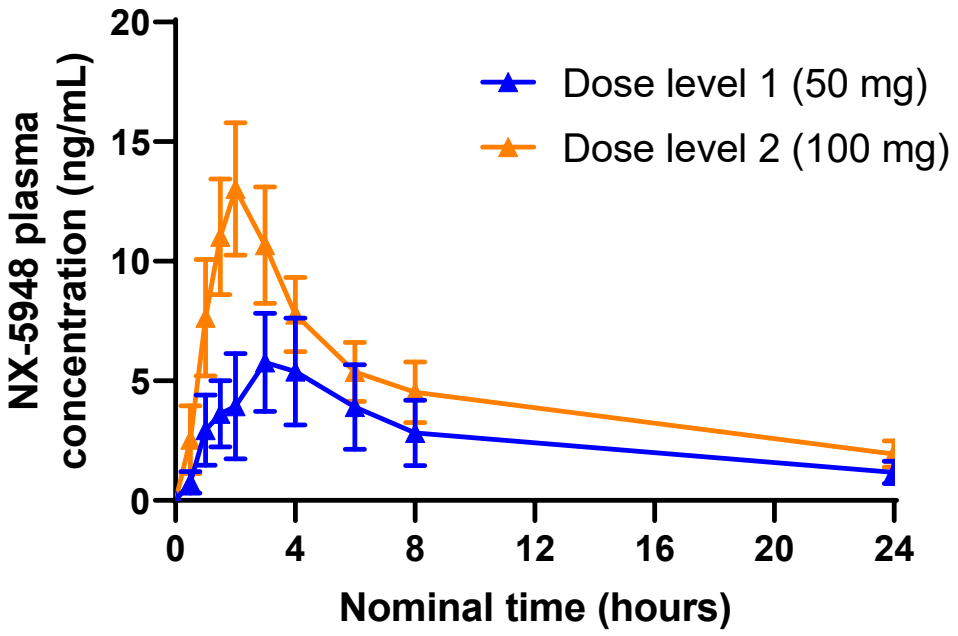


- Phase 1a dose escalation is ongoing at clinical sites in the U.S. and U.K.
- Anticipate initiating expansion cohort(s) in H2 2023

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; **PCNSL**, primary CNS lymphoma; **PD,** pharmacodynamics; **PK,** pharmacokinetics; **WM**, Waldenstrom's macroglobulinemia

Preliminary Data Suggest that NX-5948 Exhibits Dose Proportional Pharmacokinetics

Mean (± SEM) Cycle 1 Day 1 pharmacokinetic profile of patients treated with NX-5948



Data cutoff: December 1, 2022

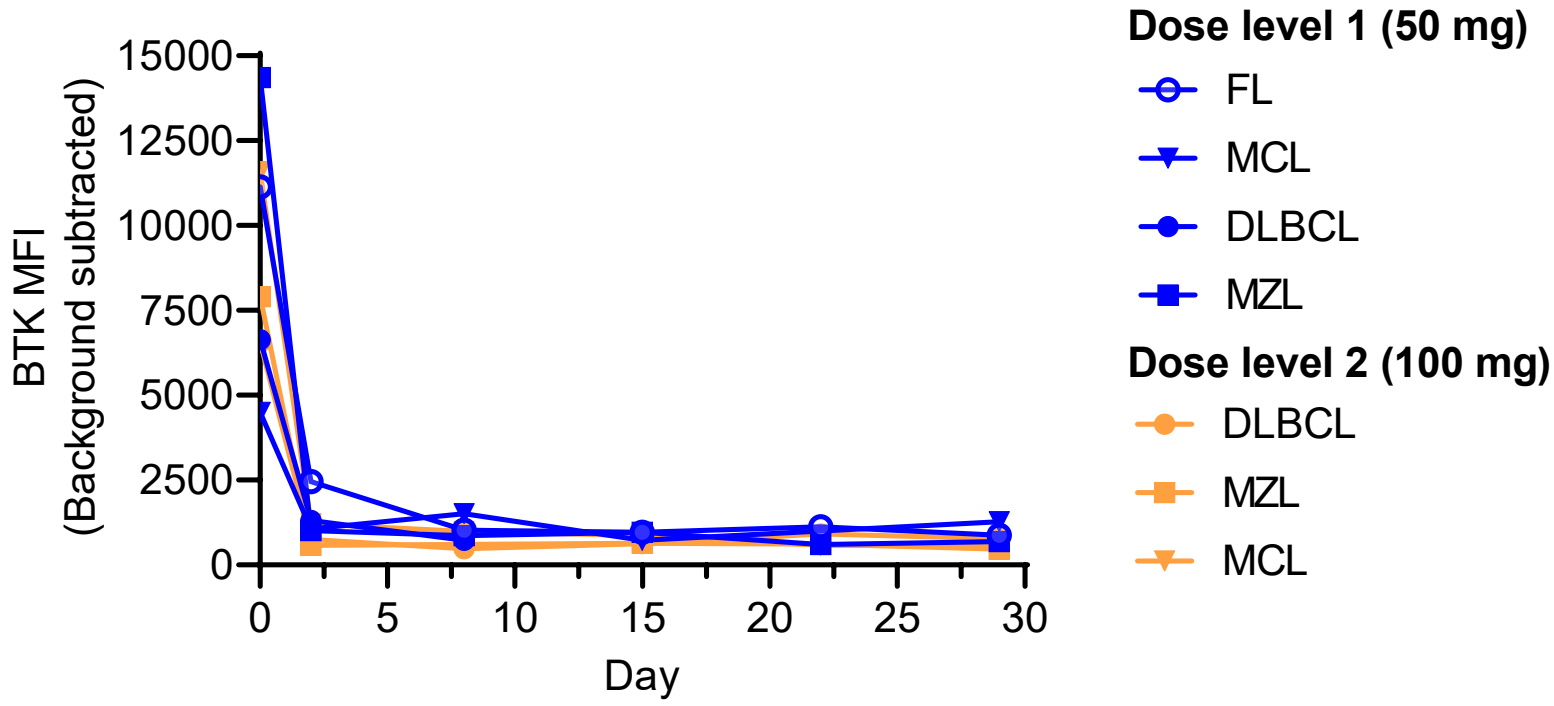
	Cycle 1, Day 1			
Dose	C_{max} (ng/mL)	AUC_{0-last} (h*ng/mL)	T_{max} (hours)	$t_{1/2}$ * (hours)
50 mg n=4	4.52 (102)	42.1 (152)	3.0	12.8 (19.9)
100 mg n=3	12.3 (45.3)	99.6 (50.2)	2.0	12.4 (9.39)

C_{max} and AUC_{0-last} are presented as geometric mean (geometric %CV); T_{max} is presented as median; $t_{1/2}$ is presented as mean (%CV); *AUC extrapolation >20%

- The half life of ~12.6 hours supports once daily dosing.
- The T_{max} of 2–3 hours suggests fast absorption.
- Exposures (both AUC and C_{max}) increase linearly with dose.

NX-5948 Resulted in Rapid, Robust and Sustained BTK Degradation in all Patients Dosed

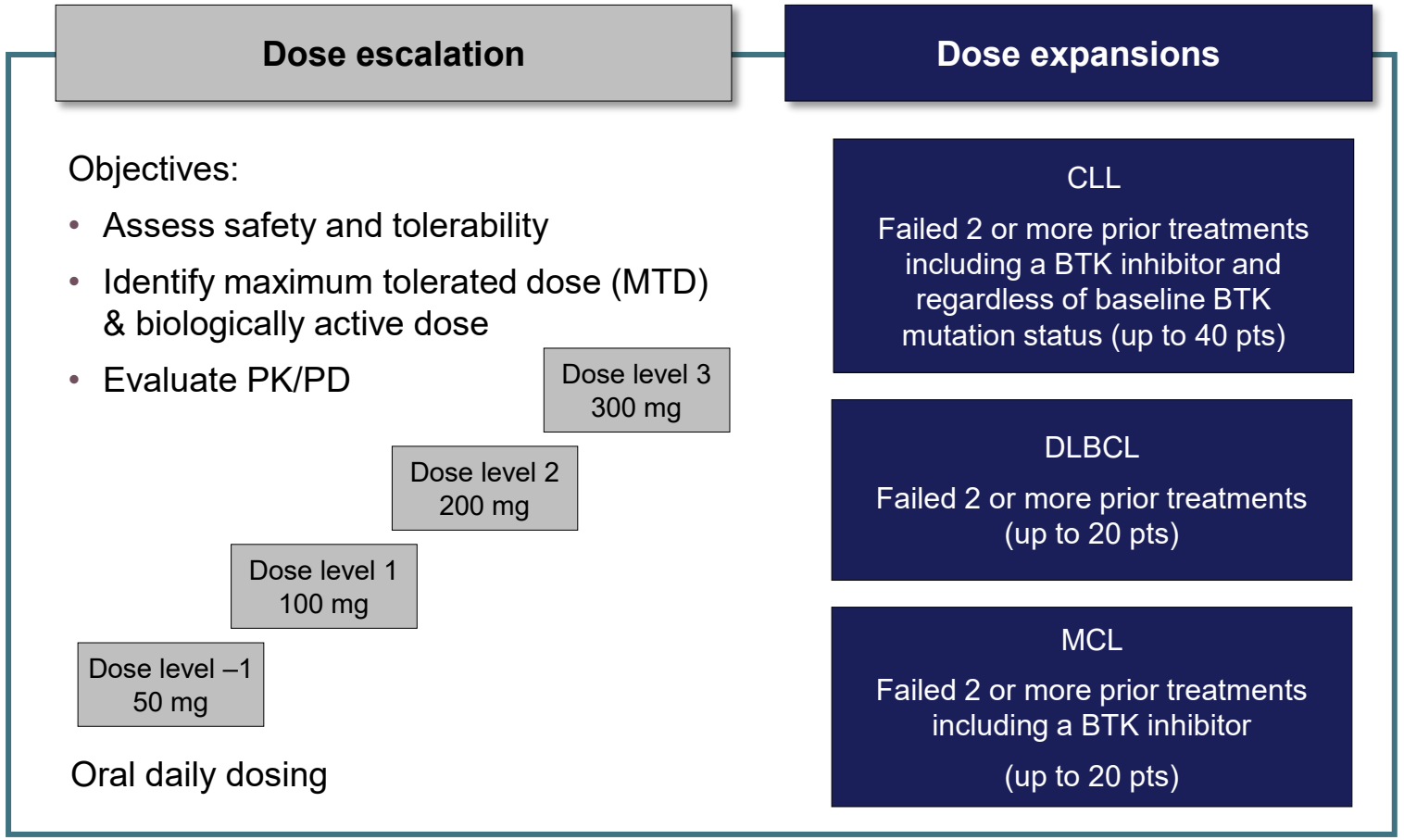
- NX-5948 induced sustained BTK degradation of 89±4% at Cycle 2 Day 1 across dose levels



FL (follicular lymphoma), DLBCL (diffuse large B cell lymphoma), MCL (mantle cell lymphoma), MZL (marginal zone lymphoma)

NX-2127-001: Trial Design

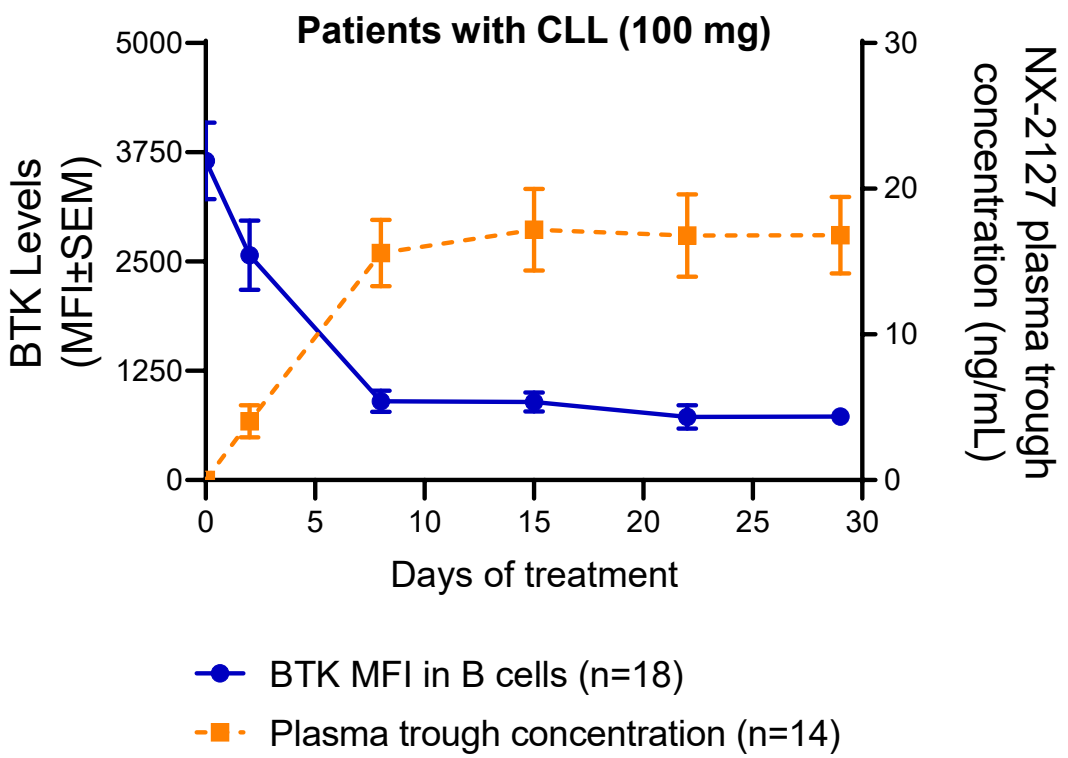
Phase 1 trial in adults with relapsed/refractory B-cell malignancies

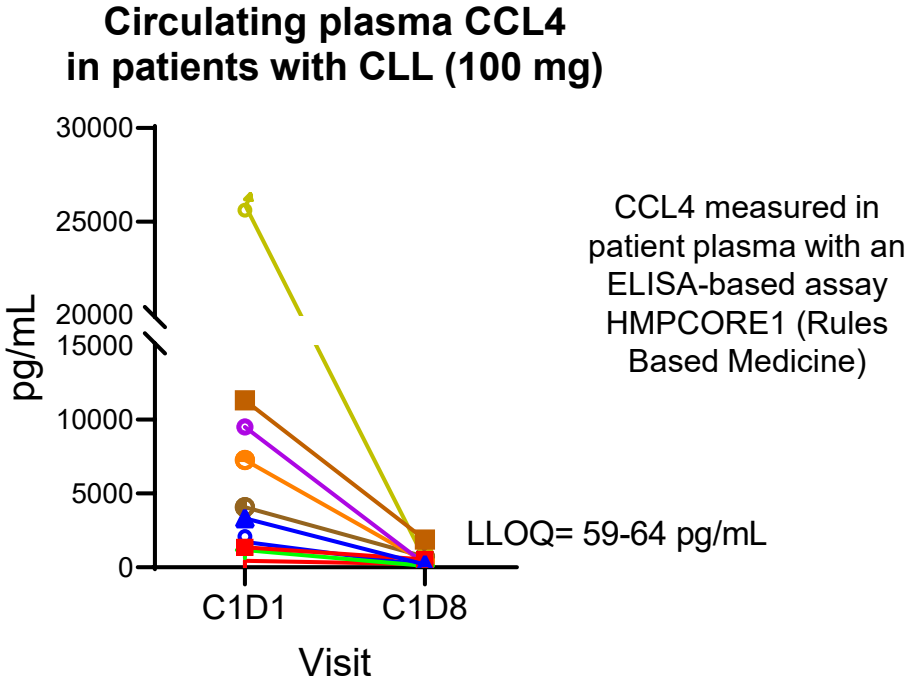


- CLL Phase 1b expansion cohort ongoing at 100 mg dose
- DLBCL Phase 1b expansion cohort ongoing at 300 mg
- MCL Phase 1b expansion cohort ongoing at 300 mg
- Phase 1a dose escalation is ongoing at 200 mg and 300 mg doses for patients with NHL

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

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

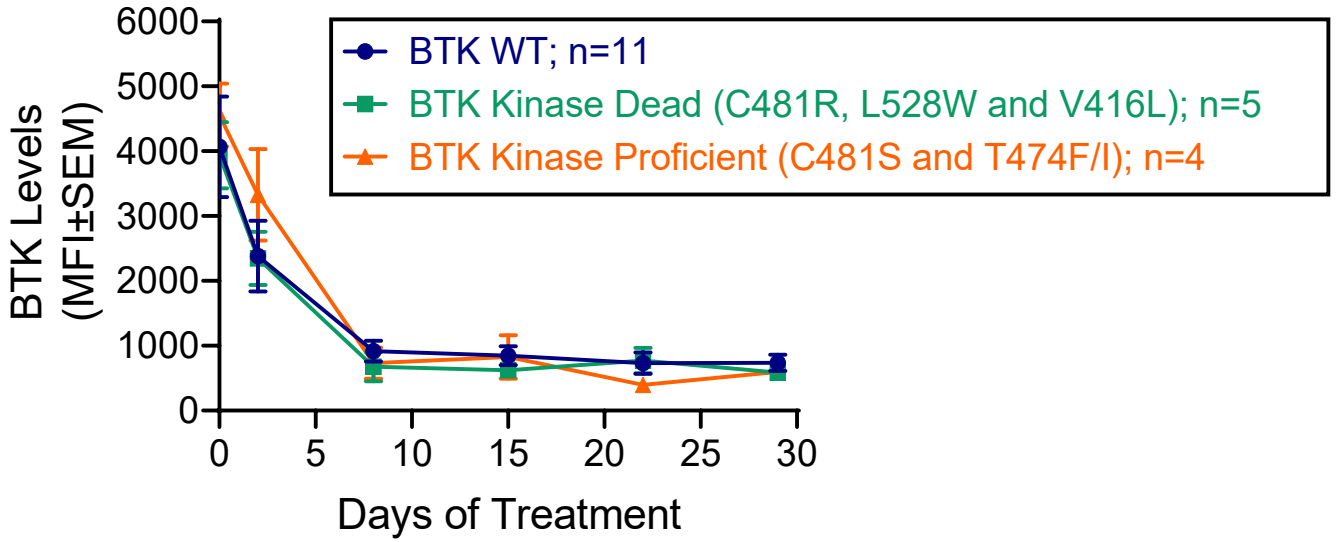
BTK, Bruton's tyrosine kinase; **CCL4**, C–C motif ligand 4; **LLOQ**, lower limit of quantification

Data cutoff: September 21, 2022

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

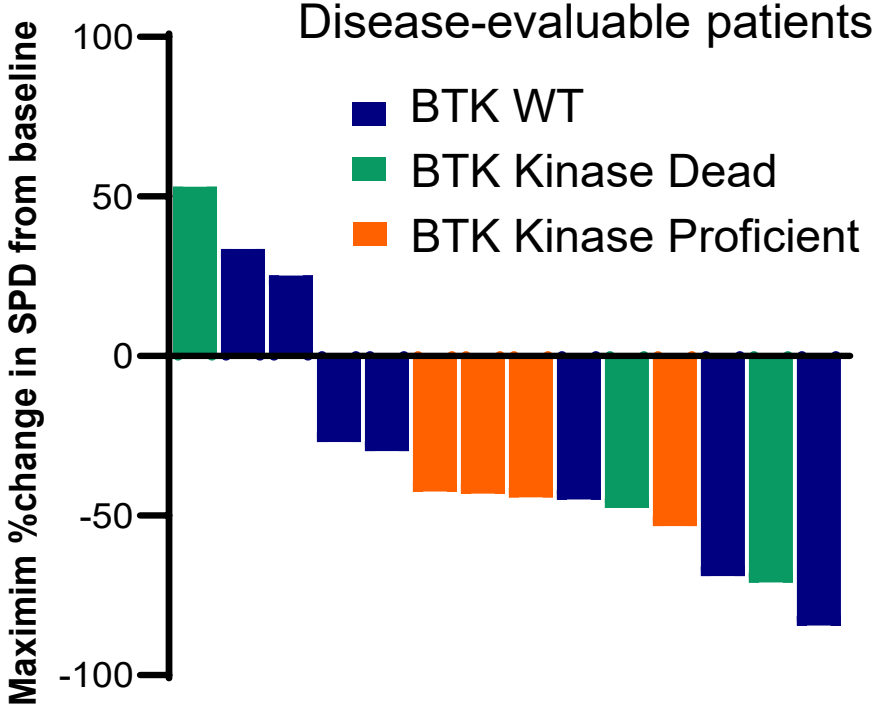
NX-2127 Preliminary Efficacy in Patients with CLL

BTK degradation in CLL patients with known BTK mutation status



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

Disease-evaluable patients



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

Montoya, Dec. 2022 ASH

Mechanistic Rationale for Dual Degrader in DLBCL

CLINICAL TRIALS AND OBSERVATIONS

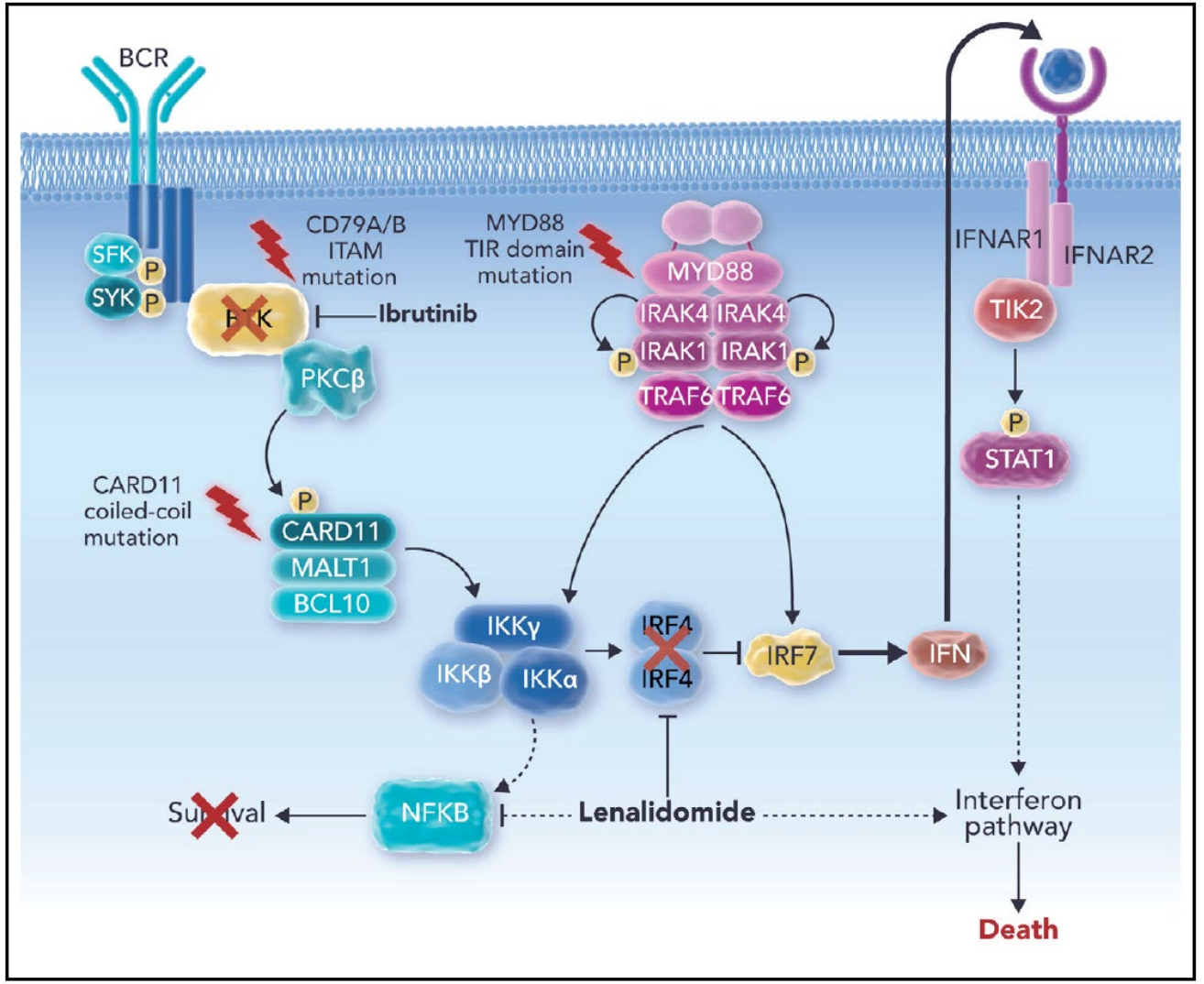
Comment on Goy et al, page 1024

Ibrutinib and lenalidomide: when 1+1 = >2

Jason Westin | MD Anderson Cancer Center

Hyper-activated BCR (CD79b-mut) and TLR (MyD88-mut) signaling are hallmarks of non-GCB DLBCL:

- NX-2127 targets both BCR and TLR signaling through BTK degradation
- NX-2127 targets non-BTK dependent TLR signaling through its immunomodulatory activity

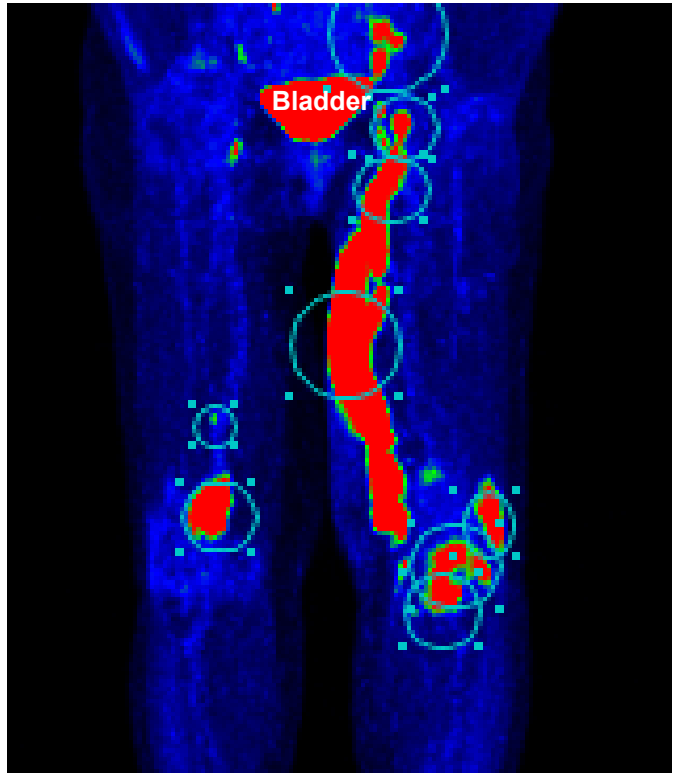


[1] Westin J. Blood 2019;134:996–8

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

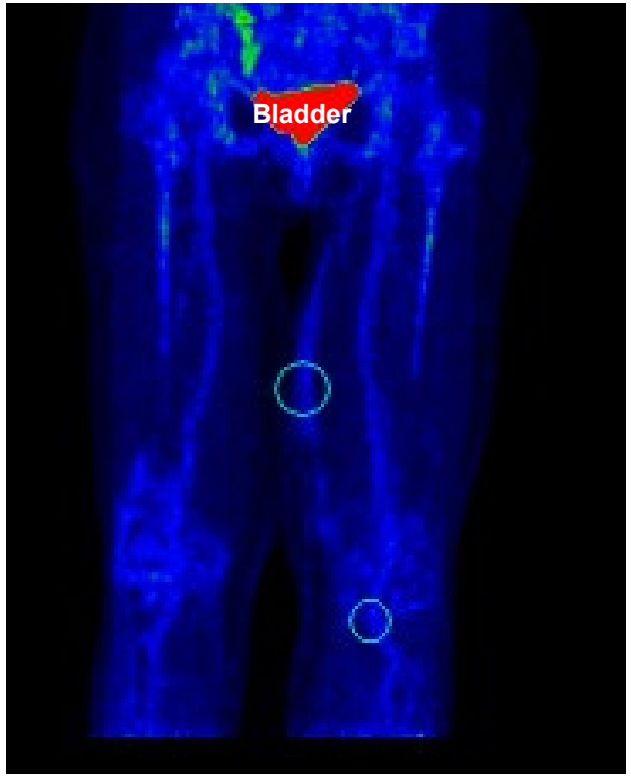
FDG-PET CT Scan Disease Assessment

Baseline



Deauville score: 5

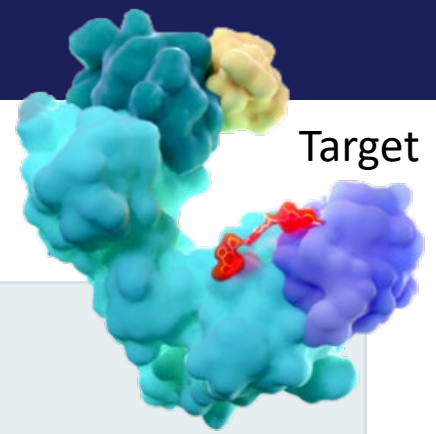
Confirmatory Week 16 Scan



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

Targeted Protein Degradation Holds Promise For Treating Cancer



Emergent BTKi-Resistant Mutations

- The use of BTK inhibition for treating B-cell malignancies has led to the development of acquired mutations that confer resistance to both covalent and noncovalent BTK inhibitors

Scaffolding Functions of BTK

- Multiple mutant variants of BTK are kinase-dead but retain the ability to propagate BCR signaling in TMD8 cells
- Scaffolding functions of BTK in oncogenic setting can pose additional challenges for the application of BTK inhibitors

Targeted BTK Degraders as "Next-Generation" Therapeutics

- Unlike an inhibitor, a degrader can address both the enzymatic and scaffolding functions of a protein
- Degraders that display positive cooperativity are more resilient to resistance mutations
- Nurix's BTK degraders, NX-2127 and NX-5948, are potent against known and novel clinically relevant BTK inhibitor resistance mutations

Thank you

nurix

NX-2127 safety summary (TEAEs >15% in all patients)

Treatment-emergent AEs occurring in >15% of total population, n (%)	Any grade (N=36)	Grade 3+ (N=36)	SAE (N=36)
Fatigue	19 (52.8)	–	-
Neutropenia[a]	14 (38.9)	13 (36.1)	-
Contusion[b]	10 (27.8)	–	1 (2.8)
Thrombocytopenia[c]	9 (25)	3 (8.3)	-
Anemia	8 (22.2)	4 (11.1)	1 (2.8)
Hypertension	9 (25.0)	1 (2.8)	-
Constipation	7 (19.4)	–	-
Dyspnea	7 (19.4)	1 (2.8)	-
Pruritis	7 (19.4)	–	-
Atrial fibrillation/Atrial flutter[d]	6 (16.7)	3 (8.3)	2 (5.6)
Diarrhea	6 (16.7)	–	-
Petechiae	6 (16.7)	–	-
Rash	6 (16.7)	–	-

[a]Aggregate of "neutropenia" and "neutrophil count decreased" [b]Contusion includes episodes of bruising and other similar terms [c]Aggregate of "thrombocytopenia" and "platelet count decreased" [d]Cases were confounded by risk factors such as: age >80 years (4 cases), history of hypertension (4 cases), male sex (3 cases), and history of prior AF on ibrutinib

1 DLT of cognitive disturbance was observed at 300 mg (CLL); MTD not reached

AE, adverse event; **SAE**, serious adverse event; **TEAE**, treatment-emergent adverse event

Data cutoff: September 21, 2022

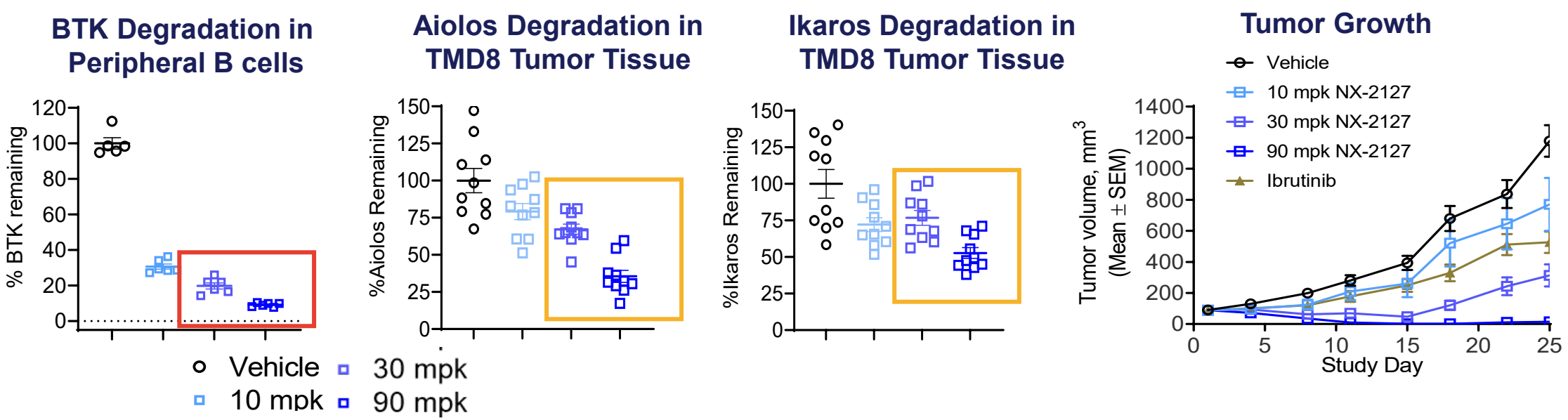
NX-2127 safety summary (all participants) by dose

AEs: all grades, n (%)	All doses (n=36)	100 mg* (n=22)	200 mg (n=8)	300 mg (n=6)
Fatigue	19 (53)	13 (59)	5 (63)	1 (17)
Neutropenia[a]	14 (39)	5 (23)	5 (63)	4 (67)
Contusion[b]	10 (28)	4 (18)	3 (38)	3 (50)
Thrombocytopenia[c]	9 (25)	5 (23)	2 (25)	2 (33)
Hypertension	9 (25)	5 (23)	2 (25)	2 (33)
Anemia	8 (22)	6 (27)	2 (25)	0
Constipation	7 (19)	7 (32)	0	0
Dyspnea	7 (19)	4 (18)	3 (38)	0
Pruritis	7 (19)	5 (23)	1 (13)	1 (17)
Atrial fibrillation/Atrial flutter[d]	6 (17)	3 (14)	2 (25)	1 (17)
Diarrhea	6 (17)	5 (23)	1 (13)	0
Petechiae	6 (17)	4 (18)	1 (13)	1 (17)
Rash	6 (17)	5 (23)	1 (13)	0

[a]Aggregate of "neutropenia" and "neutrophil count decreased" [b] Includes episodes of bruising and other similar verbatim terms [c]Aggregate of "thrombocytopenia" and "platelet count decreased" [d]Cases were confounded by risk factors such as: age >80 years (4 cases), history of hypertension (4 cases), male sex (3 cases), and history of prior AF on ibrutinib (2 cases)
*11 of the 22 patients treated at the 100 mg qd dose had CLL

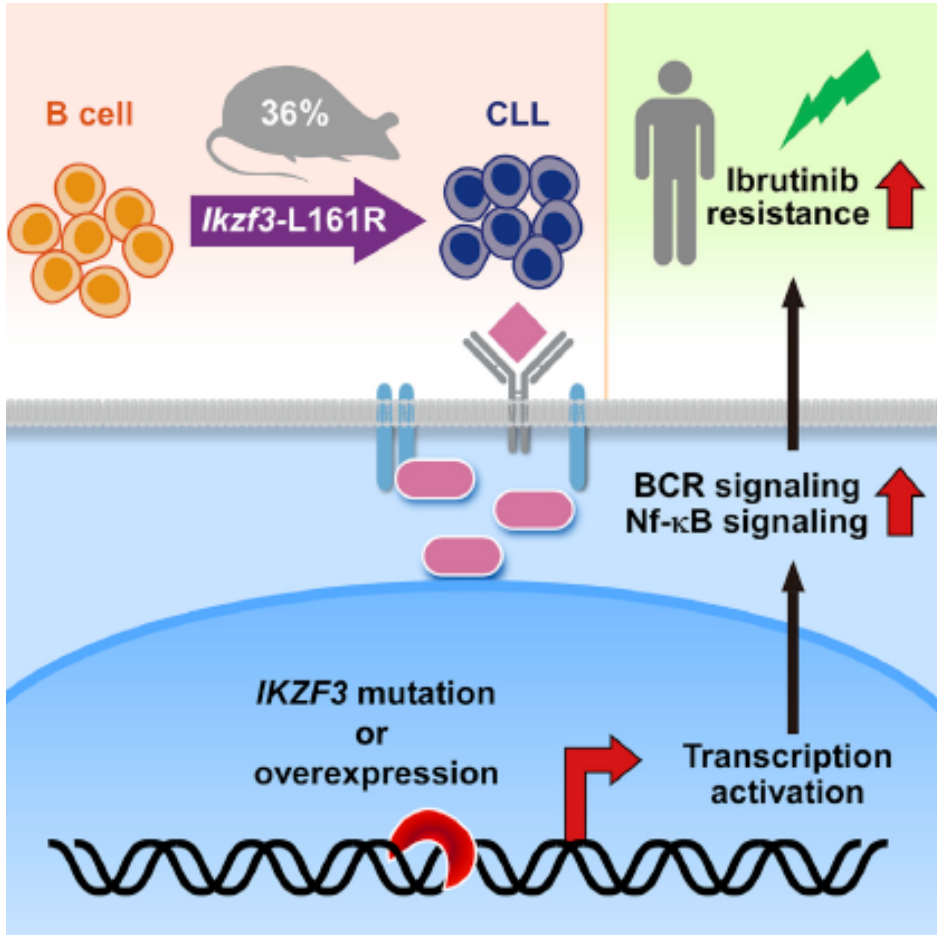
BTK Degradation of 80%+ Drives Potent Anti-tumor Activity in Preclinical Models

Ikaros and Aiolos degradation also achieve target range at therapeutic doses



Oral dose of NX-2127 (mg/kg)	10	30	90
% BTK degradation in peripheral B cells	69%	80%	91%
% Aiolos degradation in tumor tissue	21%	33%	64%
% Ikaros degradation in tumor tissue	28%	23%	47%
% Tumor growth inhibition vs Vehicle (Day 24)	58%	74%	100%

Aiolos (IKZF3) Overexpression Drives BTK Inhibitor Resistance in CLL, a Rationale for a Combination Strategy



Article

Cancer Cell

A hotspot mutation in transcription factor *IKZF3* drives B cell neoplasia via transcriptional dysregulation

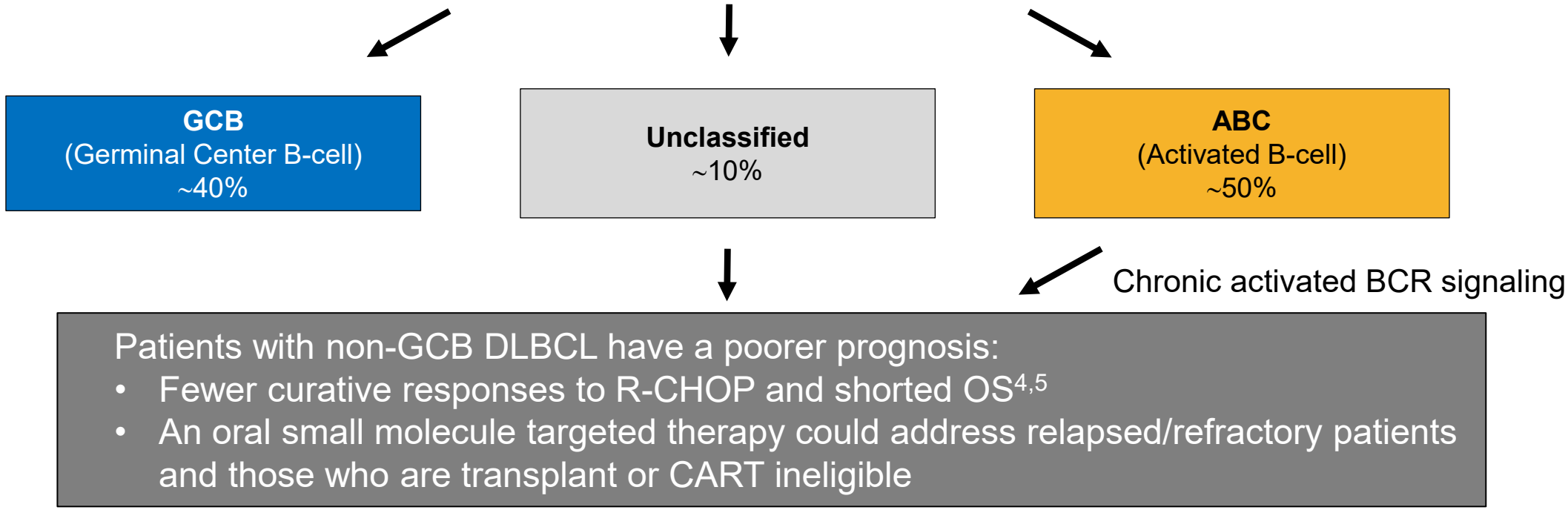
"Our results thus highlight IKZF3 oncogenic function in CLL via transcriptional dysregulation and demonstrate that this pro-survival function can be achieved by either somatic mutation or overexpression of this CLL driver. This emphasizes the need for combinatorial approaches to overcome IKZF3-mediated BCR inhibitor resistance."

Source: Lazarian et al; Cancer Cell 39, 380–393, March 8, 2021

Non-GCB DLBCL Represents an Important Unmet Medical Need

- DLBCL is the most common form of lymphoma, representing ~30% of all NHL diagnoses[1,2]
- ~24,000 people diagnosed in the United States each year, with ~60% 5-year survival[1,2,3]

DLBCL treatments are the same for all patients, even though it is a biologically heterogeneous disease[4]

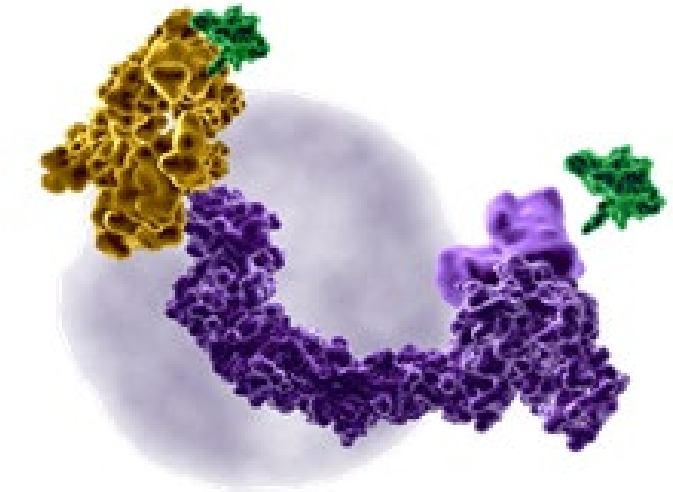


[1]American Cancer Society. Cancer Facts & Figures 2022. Atlanta, Ga: American Cancer Society; 2022. https://www.cancer.org/cancer/non-hodgkin-lymphoma/about/key-statistics.ntml#references
[2]NCCN, B-Cell Lymphomas; April 2021 https://www.nccn.org/professionals/physician_gls/pdf/b-cell.pdf; [3]https://seer.cancer.gov/statfacts/html/dlbcl.html
[4]Mareschal et al. Hematologica 2011;96:1888–90; [5]Schmitz et al. N Engl J Med 2018;378:1396–407

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



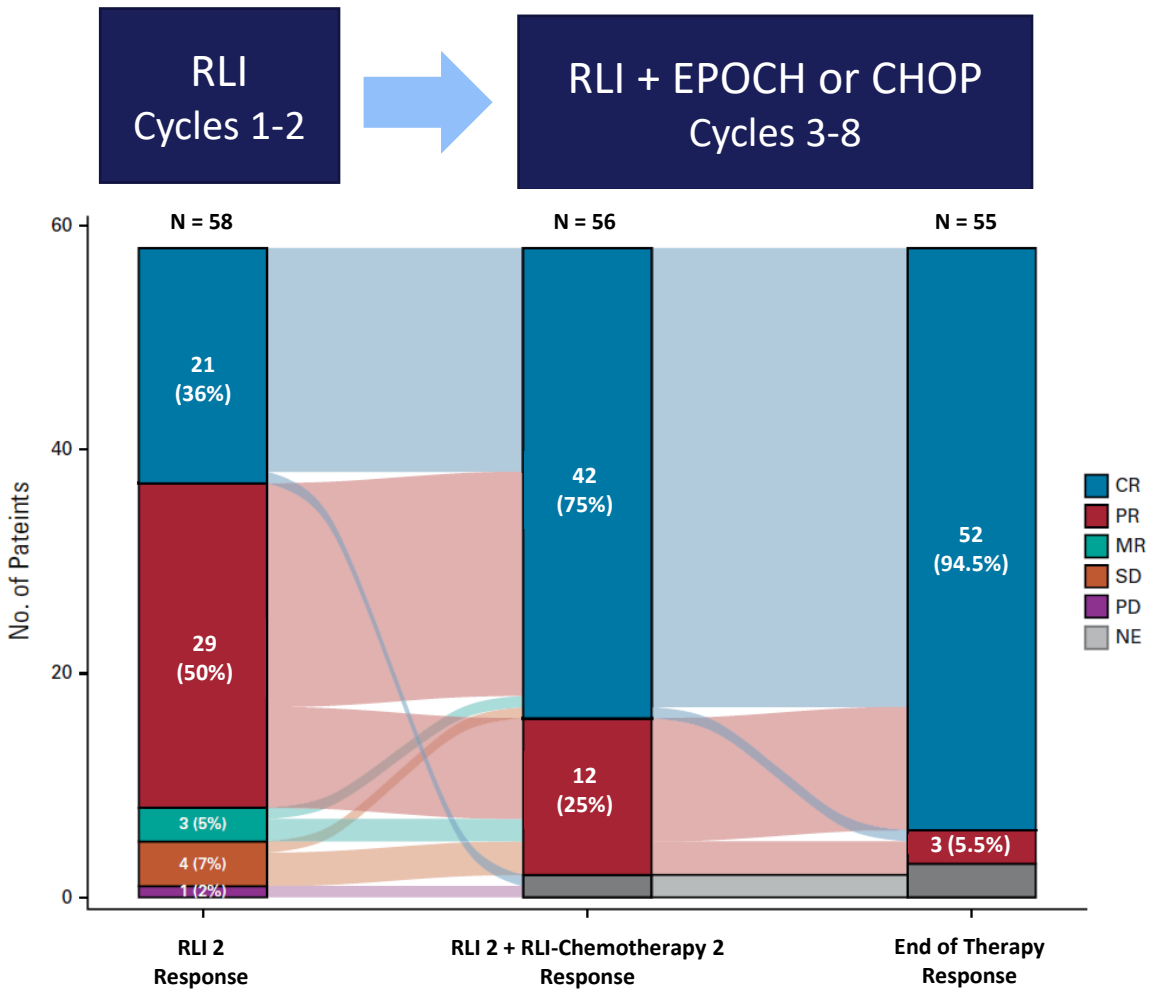
BTK degraders have the potential to displace inhibitors in the markets where BTK inhibitors currently dominate (e.g., CLL)

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

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