
ONCOLOGY
Lilly

SAFE HARBOR PROVISION

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2021 **INVESTMENT COMMUNITY MEETING**

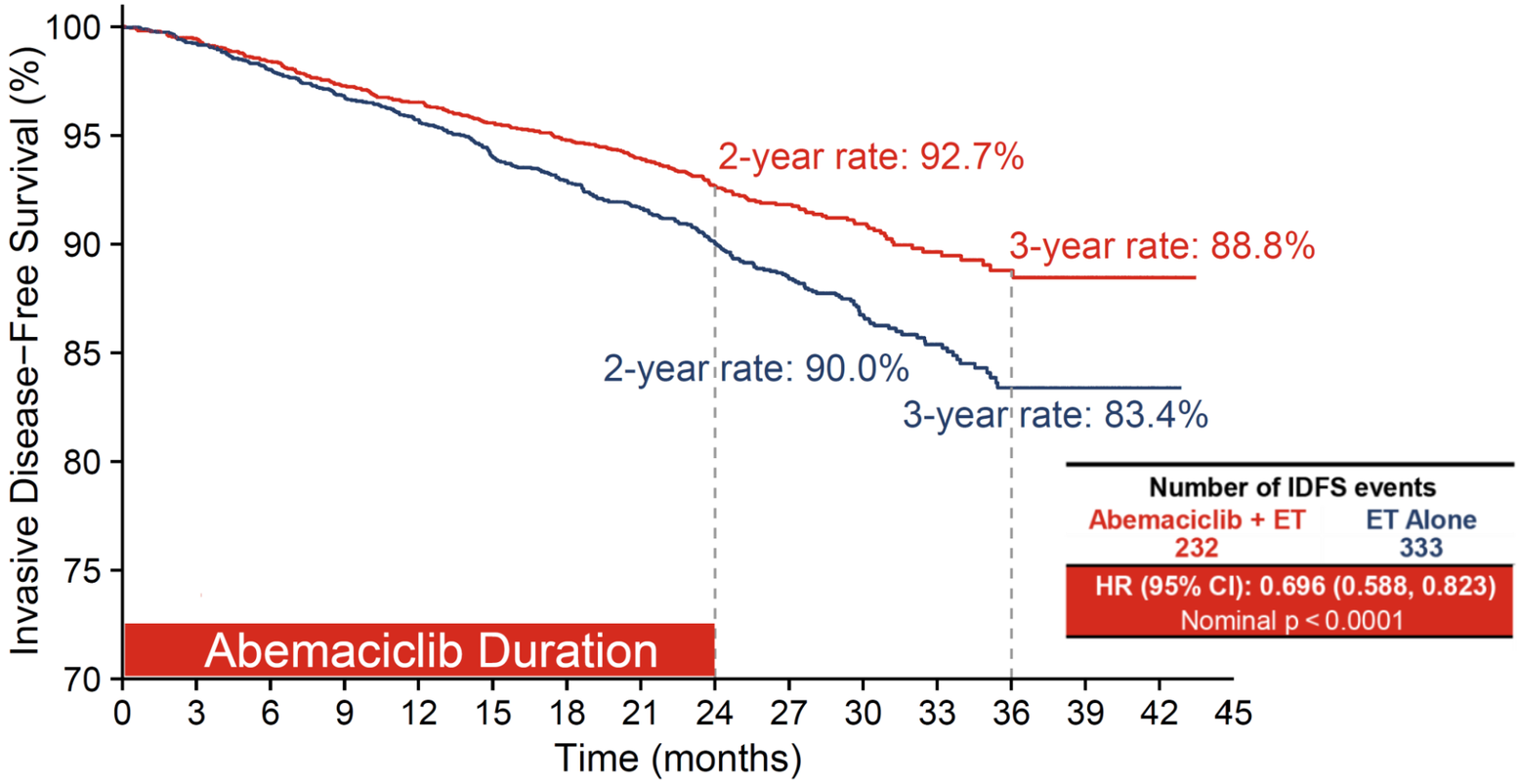
ONCOLOGY STRATEGY

- Focus on drugs and mechanisms that can be derisked early in clinical development
- Quality over quantity
- “Build vs buy” agnosticism
- Biology-driven target selection and development
- Embrace small molecule and biologics-based modalities

VERZENIO: monarchE DATA FROM ESMO VIRTUAL PLENARY

IDFS BENEFIT MAINTAINED WITH ADDITIONAL FOLLOW UP IN ITT POPULATION

VERZENIO REDUCED THE RISK OF CANCER RECURRENCE BY 30.4%



- The absolute difference in IDFS rates between arms was 5.4% at 3 years
- Consistent IDFS treatment benefit observed in prespecified subgroups
- Continued IDFS benefit beyond 2-year Verzenio treatment period
- Median follow-up of 27.1 months

Analysis landmark	IDFS
	Piecewise HR* (95% CI**)
Year 0-1	0.795 (0.589, 1.033)
Year 1-2	0.681 (0.523, 0.869)
Year 2+	0.596 (0.397, 0.855)

Verzenio in combination with ET is approved for use in patients with HR+ HER2- high-risk, early breast cancer and a Ki-67 index >20%. Data presented above are in a population broader than this approved use

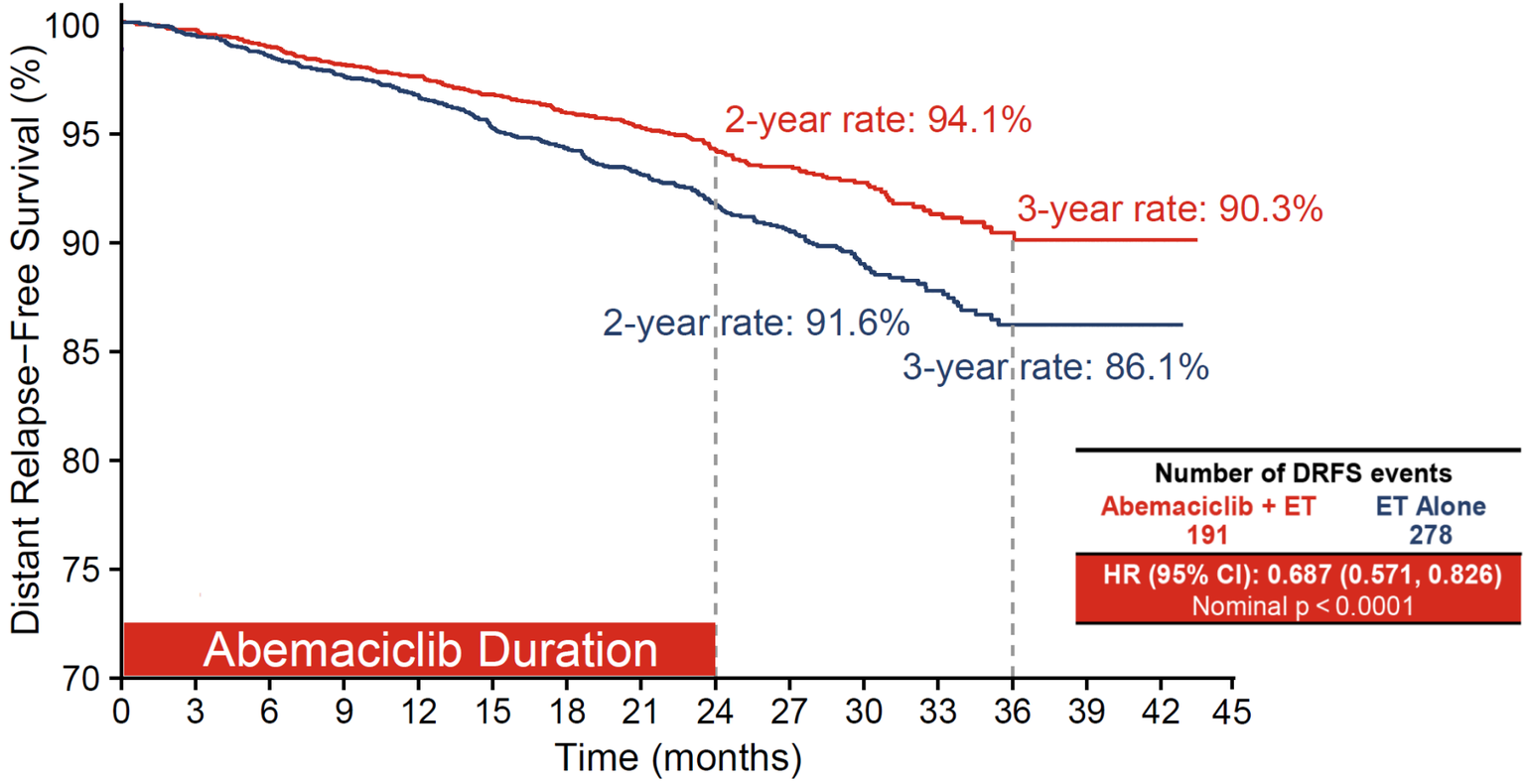
* Piecewise hazard ratio was estimated using piecewise exponential model to assess the yearly treatment effect size ** 95% credible intervals were calculated by equal tails in the posterior samples of Bayesian exponential models

ESMO = European Society for Medical Oncology; ITT = intention-to-treat; IDFS = invasive disease-free survival; ET = endocrine therapy; HR = hazard ratio

VERZENIO: monarchE DATA FROM ESMO VIRTUAL PLENARY

DRFS BENEFIT MAINTAINED WITH ADDITIONAL FOLLOW UP IN ITT POPULATION

VERZENIO REDUCED THE RISK OF DISTANT METASTASES BY 31.3%



- The absolute difference in DRFS rates between arms was 4.2% at 3 years
- Consistent DRFS treatment benefit observed in prespecified subgroups
- Continued DRFS benefit beyond 2-year Verzenio treatment period
- Median follow-up of 27.1 months

Analysis landmark	DRFS
	Piecewise HR* (95% CI**)
Year 0-1	0.732 (0.520, 0.987)
Year 1-2	0.675 (0.507, 0.875)
Year 2+	0.692 (0.448, 1.032)

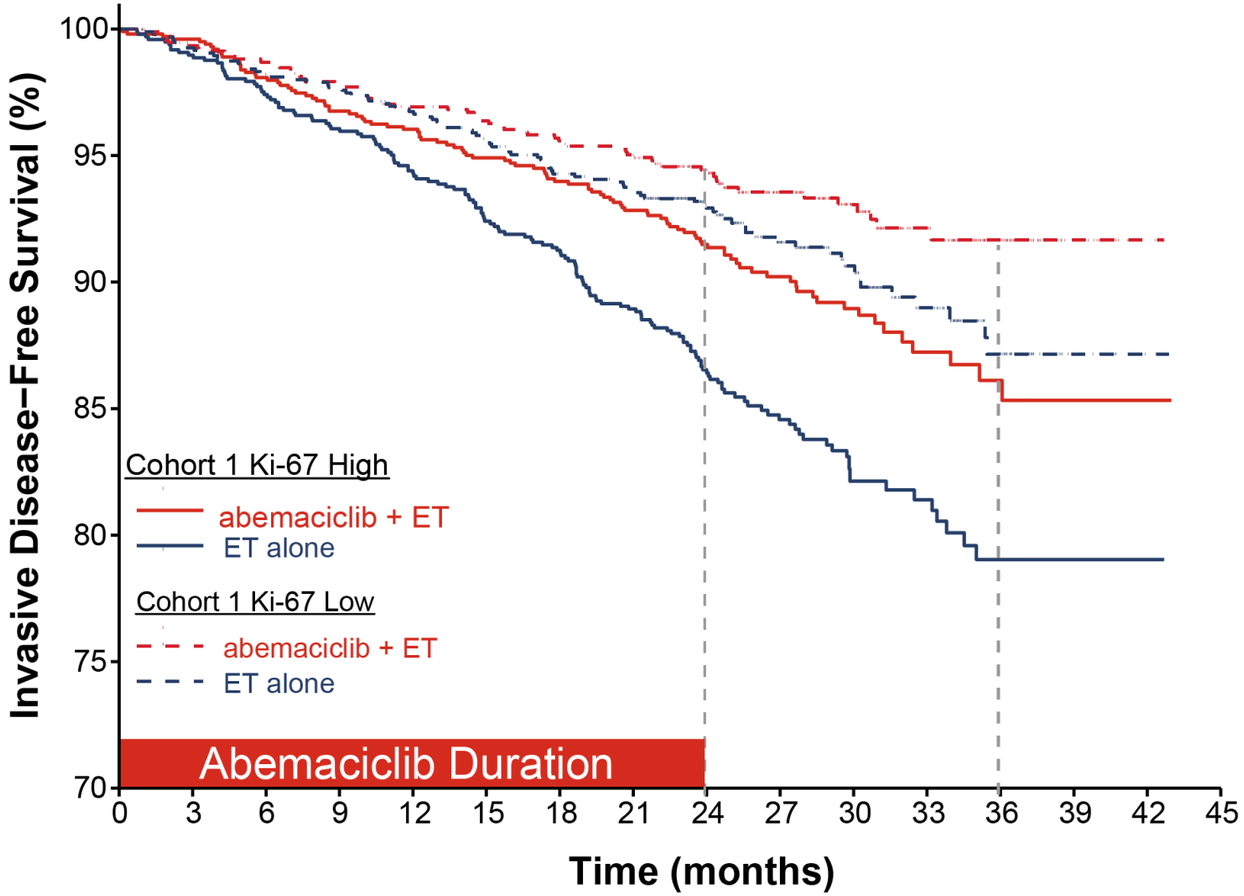
Verzenio in combination with ET is approved for use in patients with HR+ HER2- high-risk, early breast cancer and a Ki-67 index >20%. Data presented above are in a population broader than this approved use

* Piecewise hazard ratio was estimated using piecewise exponential model to assess the yearly treatment effect size ** 95% credible intervals were calculated by equal tails in the posterior samples of Bayesian exponential models

ESMO = European Society for Medical Oncology; ITT = intention-to-treat; DRFS = distant recurrence free survival; ET = endocrine therapy; HR = hazard ratio

VERZENIO: monarchE DATA FROM ESMO VIRTUAL PLENARY

Ki-67 WAS PROGNOSTIC OF RECURRENCE RISK BUT NOT PREDICTIVE OF ABEMACICLIB BENEFIT



	Abemaciclib + ET	ET alone	HR (95% CI)
Cohort 1 Ki-67 High, N = 2003			
Patients, N	1017	986	0.626 (0.488, 0.803)
Events, n	104	158	
3-Year Rates	86.1%	79.0%	
Cohort 1 Ki-67 Low, N = 1914			
Patients, N	946	968	0.704 (0.506, 0.979)
Events, n	62	86	
3-Year Rates	91.7%	87.2%	

As expected, high Ki-67 index was prognostic of worse outcome.
However, abemaciclib had a similar effect size regardless of Ki-67 index.

Verzenio in combination with ET is approved for use in patients with HR+ HER2- high-risk, early breast cancer and a Ki-67 index >20%. Data presented above are in a population broader than this approved use; Data from Cohort 1 = high-risk based on clinical pathological features (≥4 ALN OR 1-3 ALN and at least 1 of the following: grade 3 disease, tumor size ≥5 cm); ESMO = European Society for Medical Oncology; ET = endocrine therapy; HR = hazard ratio

VERZENIO: PROSTATE CANCER

PHASE 2/3 TRIAL: CYCLONE 2

- Evaluating metastatic castrate-resistant prostate cancer; a second-line population by today's standards
- Intervention: Abiraterone/prednisone +/- Verzenio
- ~350 patients
- Blinded to IDMC analysis, but high efficacy bar for rPFS was set for Phase 3 trigger
- Historic duration of therapy ~16 months
- Primary outcome data expected 2024

NEW PHASE 3 TRIAL: CYCLONE 3

- Study start planned for mid-2022
- Evaluating an earlier line treatment population in prostate cancer
- Intervention: Abiraterone/prednisone +/- Verzenio
- Historic duration of therapy ~40 months

IDMC = independent data monitoring committee; rPFS = Radiographic progression-free survival

RETEVMO
FIRST-IN-CLASS RET INHIBITOR

PERFORMANCE

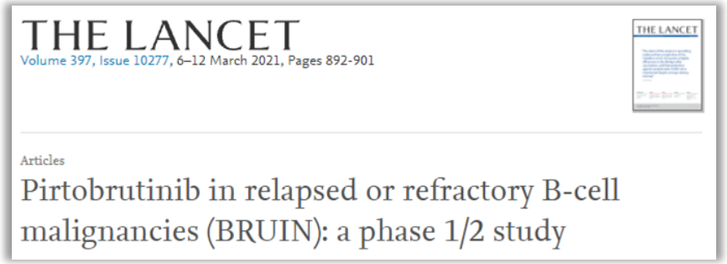
- First RET inhibitor approved for certain lung and thyroid cancers with RET fusions and mutations
- Rapid development timeline: 3 years from first human dose to approval
- In the largest and most mature set of clinical data for a RET inhibitor, Retevmo has shown robust, durable objective response rates
- Market leading performance with continued focus on diagnostics utilization

CLINICAL & REGULATORY UPDATES

- CCR manuscript supported Retevmo as a new standard of care for treatment of brain metastases for advanced RET-fusion positive NSCLC
- Randomized first-line lung study expected to read out in early 2023
- sNDA for full U.S. approval for lung cancer submitted with regulatory action expected in 2022
- Recently submitted data to the EMA to expand the lung indication to be line agnostic

CCR = Clinical Cancer Research; NSCLC = non-small cell lung cancer; sNDA = supplemental new drug application; EMA = European Medicines Agency

PIRTOBRUTINIB
FOUR OF FIVE GLOBAL STUDIES INITIATED IN 2021


THE LANCET
Volume 397, Issue 10277, 6–12 March 2021, Pages 892-901

Articles

Pirtobrutinib in relapsed or refractory B-cell malignancies (BRUIN): a phase 1/2 study

1 ✓
Monotherapy in post-BTK CLL patients
250 patients

2 ✓
Monotherapy in Tx naïve CLL
250 patients

3 ✓
H2H vs. cBTKs in BTK naïve MCL
500 patients

4 ✓
Combo with Venclexta+Ritux in CLL
600 patients

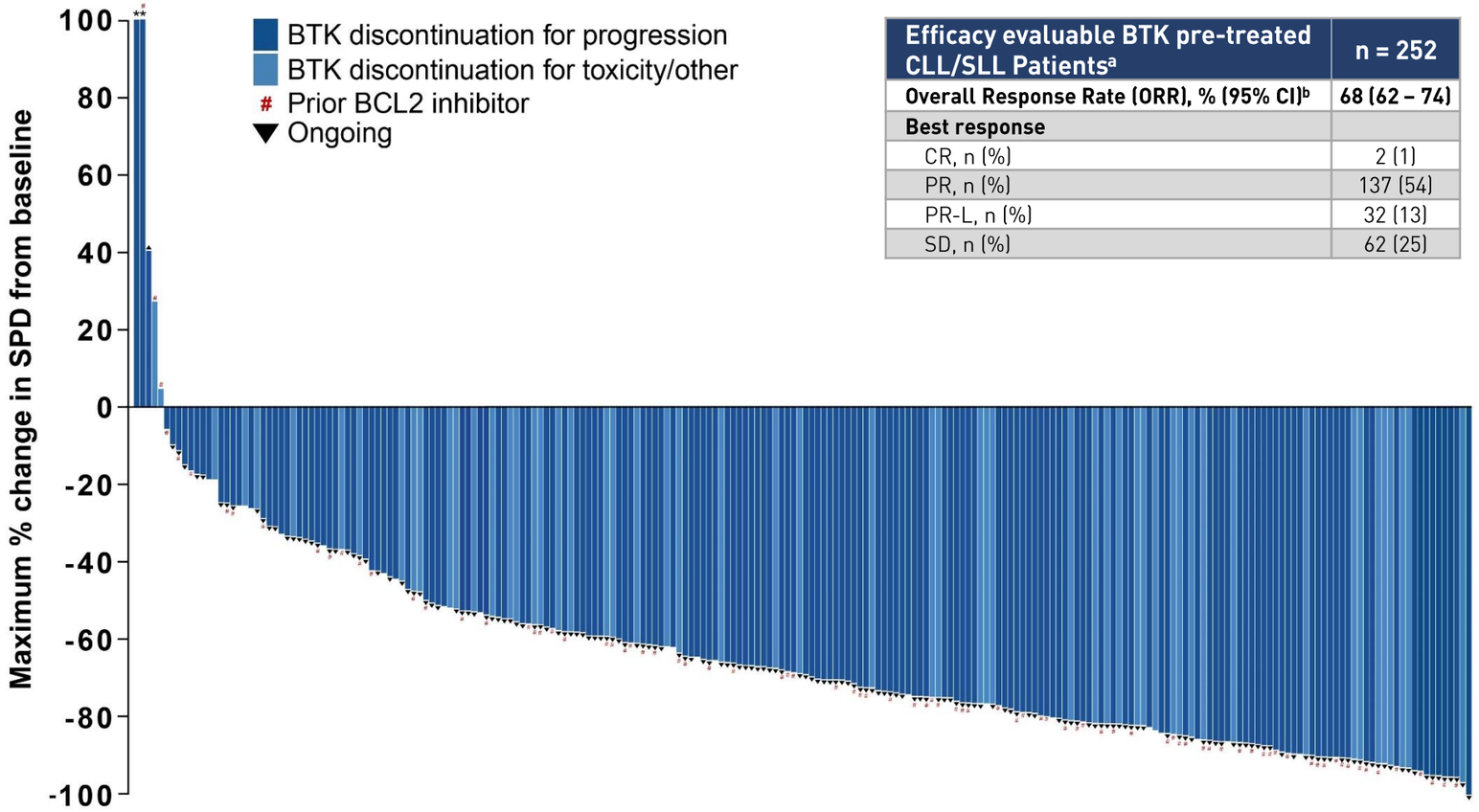
5
Expected start in 1H 2022
H2H vs. ibrutinib in BTK naïve CLL
650 patients

✓ Denotes study has started

BTK = Bruton Tyrosine Kinase; cBTK = covalent Bruton Tyrosine Kinase; CLL = Chronic lymphocytic leukemia; MCL = mantle cell lymphoma; H2H = head-to-head

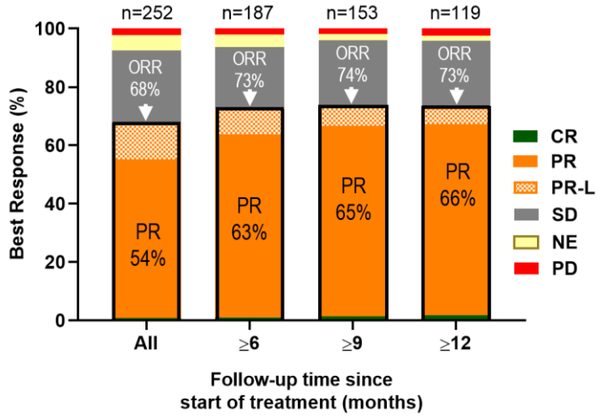
PIRTOBRUTINIB

EFFICACY IN BTK PRE-TREATED CLL/SLL PATIENTS



Efficacy evaluable BTK pre-treated CLL/SLL Patients[a]	n = 252
Overall Response Rate (ORR), % (95% CI)[b]	**68 (62 – 74)**
Best response	
CR, n (%)	2 (1)
PR, n (%)	137 (54)
PR-L, n (%)	32 (13)
SD, n (%)	62 (25)

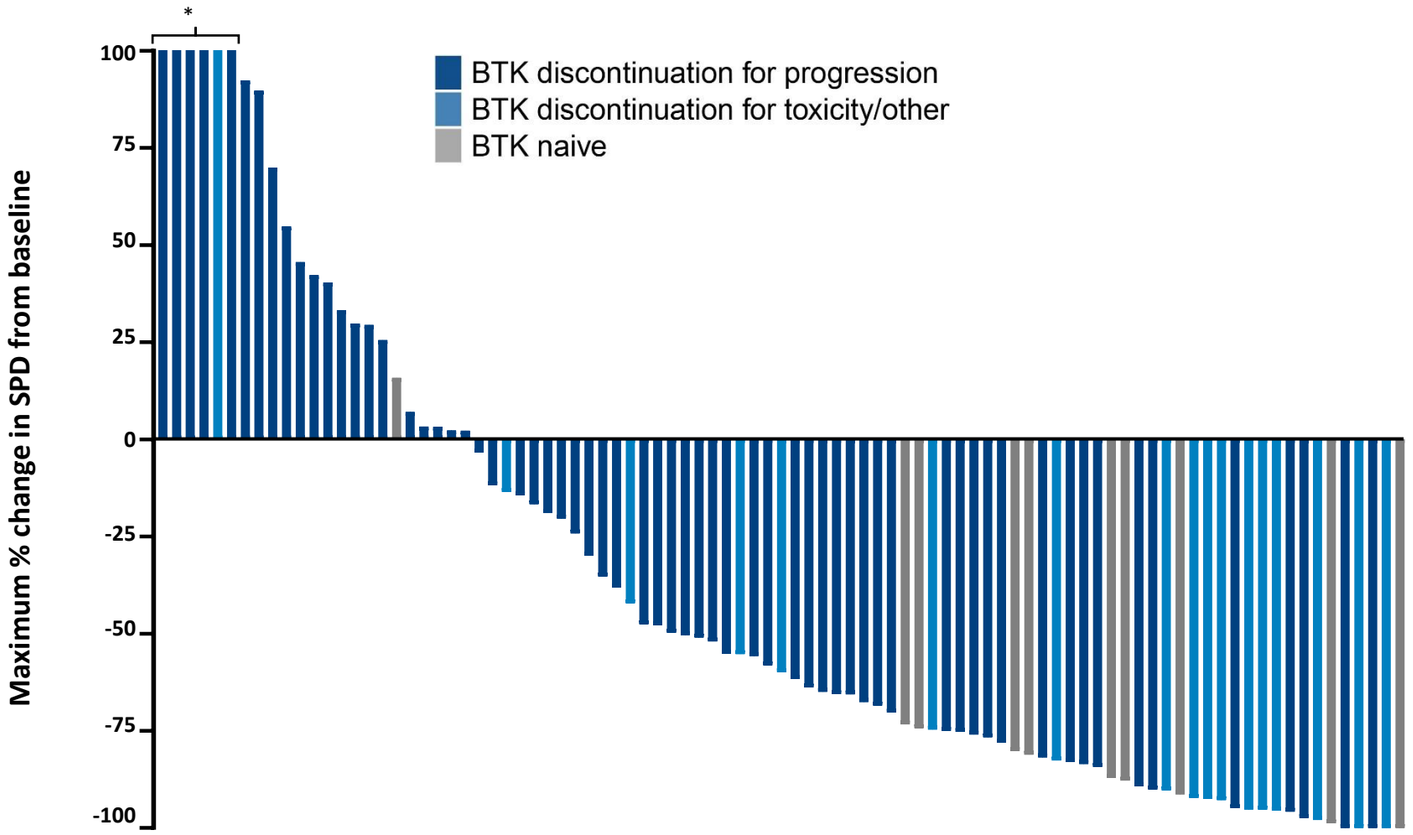
Overall Response Rate Over Time[c]



- 68% ORR in CLL/SLL patients rising to 73% in patients followed for 12 or more months
- Efficacy was independent of BTK C481 mutation status, the reason for prior BTK discontinuation, or other classes of prior therapy received (including covalent BTK and BCL2i)
- 74% of BTK pre-treated patients remain on therapy

Data cutoff date of 16 July 2021. *Patients with >100% increase in SPD. Data for 30 patients are not shown in the waterfall plot due to no measurable target lesions identified by CT at baseline, discontinuation prior to first response assessment, or lack of adequate imaging in follow-up. [a]Efficacy evaluable patients are those who had at least one post-baseline response assessment or had discontinued treatment prior to first post-baseline response assessment. [b]ORR includes patients with a best response of CR, PR, and PR-L. Response status per iwCLL 2018 according to investigator assessment. Total % may be different than the sum of the individual components due to rounding. [c]Includes the BTK pre-treated efficacy-evaluable CLL/SLL patients at the time of data cutoff. Data at each timepoint includes the BTK pre-treated efficacy-evaluable CLL/SLL patients who had the opportunity to be followed for at least the indicated amount of time. CLL = Chronic lymphocytic leukemia; SLL = small lymphocytic lymphoma; CR = complete response; PR = partial response; SD = stable disease

PIRTOBRUTINIB
EFFICACY IN MANTLE CELL LYMPHOMA (MCL)



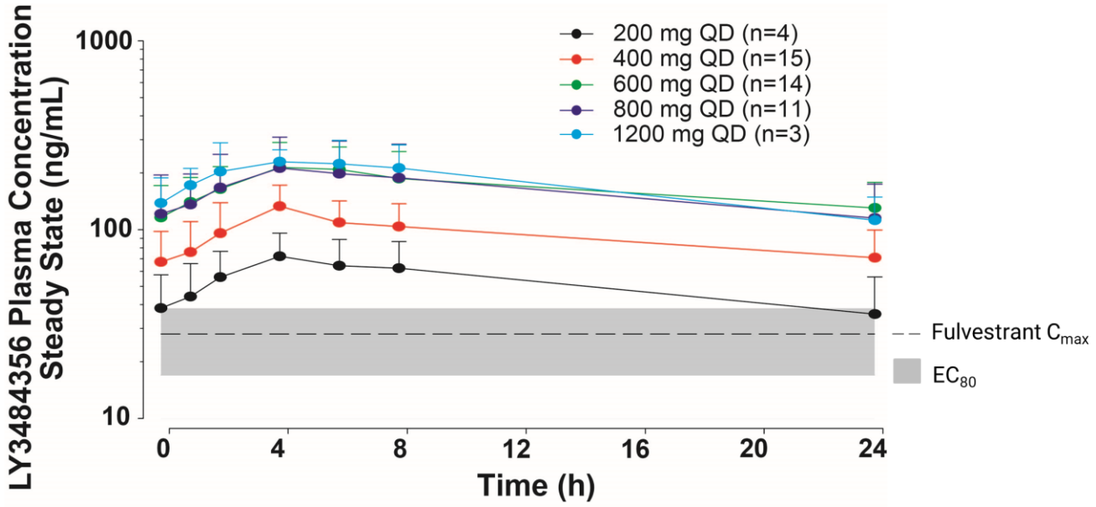
BTK Pre-Treated MCL Patients[a]	n=100
Overall Response Rate (ORR) [b], % (95% CI)	**51% (41-61)**
Best Response	
CR, n (%)	25 (25)
PR, n (%)	26 (26)
SD, n (%)	16 (16)
BTK Naive MCL Patients[a]	**n=11**
Overall Response Rate[b], % (95% CI)	**82% (48-98)**
Best Response	
CR, n (%)	2 (18)
PR, n (%)	7 (64)
SD, n (%)	1 (9)

- 51% ORR in BTK pre-treated MCL patients
- Median duration of response was 18 months at a median follow-up of 8.2 months (range, 1.0-27.9 months)
- 60% of responding patients are ongoing
- Favorable safety and tolerability are consistent with the design of pirtobrutinib as a highly selective and non-covalent (reversible) BTK inhibitor
- **Rolling submission for MCL initiated in December with potential regulatory action date in early 2023**

Data cutoff date of 16 July 2021. Data for 20 MCL patients are not shown in the waterfall plot due to no measurable target lesions identified by CT at baseline, discontinuation prior to first response assessment, or lack of adequate imaging in follow-up. *Indicates patients with >100% increase in SPD. [a]Efficacy evaluable patients are those who had at least one post-baseline response assessment or had discontinued treatment prior to first post-baseline response assessment. [b]ORR includes patients with a best response of CR and PR. Response status per Lugano 2014 criteria based on investigator assessment. Total % may be different than the sum of the individual components due to rounding; CR = complete response; PR = partial response; SD = stable disease

IMLUNESTRANT (ORAL SERD)

PHASE 1 PK DATA

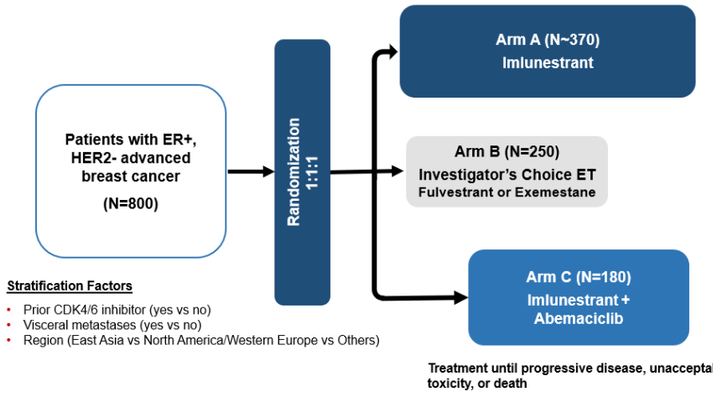


- Early efficacy in heavily pretreated advanced breast cancer patients at recommended Phase 2 dose (400mg)
- Potential for a favorable safety profile versus competition (no class cardiac/ophthalmic signal)
- No dose limiting toxicities were observed

PHASE 3 DESIGN

ember-3 CLINICAL TRIAL

Imlunestrant vs. Investigator's Choice of Endocrine Therapy, and Imlunestrant plus Abemaciclib in Patients with ER+/HER2- Locally Advanced or Metastatic Breast Cancer Previously Treated with Endocrine Therapy



Primary Endpoint
- PFS (Arm A vs Arm B; Arm C vs Arm A), as assessed by investigator

Secondary Endpoints
- PFS, as assessed by blinded Independent Review Committee
- Overall survival
- ORR, DoR, CBR, as assessed by investigator
- PFS, according to circulating *ESR1*[a] mutation status
- Safety and tolerability
- PROs: Time to worsening of the "worst pain" as measured by Worst Pain NRS
- PK: Plasma concentrations of imlunestrant and abemaciclib

[a] *ESR1* mutation status will be centrally determined in plasma by ctDNA assay from a blood draw at baseline

- Phase 3 2L metastatic breast cancer results expected in 2023
- Disclosure of adjuvant plans expected in 2022

ET = endocrine therapy; ER = estrogen receptor; HER2 = human epidermal growth factor receptor 2; QD = daily dosing; PFS = progression free survival; ORR = overall response rate; DOR = duration of response; CBR = clinical benefit rate; PROs = patient reported outcomes; NRS = numerical rating scale; PK = pharmacokinetics; 2L = second line

IDH1/2 (LY3410738)

BIOLOGIC RATIONALE

- Clinically active dual IDH1 and IDH2 inhibitor
- Binding mode/site preserves activity against second-site resistance mutations
- This unique profile has the potential for longer disease control relative to other IDH inhibitors
- No known IDH inhibitors with this profile in development

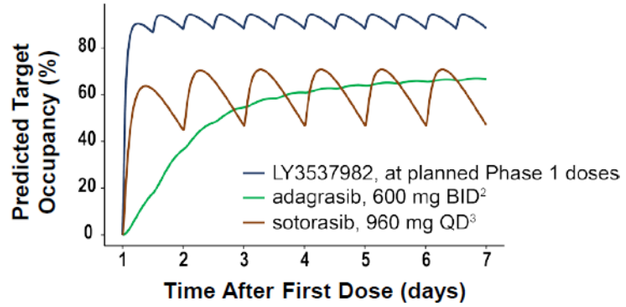
OPPORTUNITY & NEXT STEPS

- ~20% of acute myeloid leukemia
  - U.S.: 4,200 patients per year
  - Global: 13,800 patients per year
- ~20% of cholangiocarcinoma
  - U.S.: 600 new patients per year
  - Global: 3,800 new patients per year
- Robust Phase 1 dataset (heme and solid tumors) in 2022 expected to inform next steps

IDH = isocitrate dehydrogenase

KRAS G12C (LY3537982)

BIOLOGIC RATIONALE



	LY3537982	Adagrasib	Sotorasib
pERK H358 IC_{50} (nM)	0.65 (2h, n=5)	14 (3h)[4]	13.5 (2h, n=2)
Active RAS H358 IC_{50} (2h, nM)	3.35 (n=6)	89.9 (n=1)	47.9 (n=3)
Kinact/Ki ($M^{-1}s^{-1}$)	522,000	35,000[4]	9,900[5]
Predicted Target Occupancy Range	>90% trough*	60%*	45–70%*

- Highly potent covalent inhibitor with potential for >90% clinical target occupancy, which may translate to greater single agent efficacy
- Better pharmacologic properties could allow for less toxicity in combination with other targeted therapy combos in NSCLC & EGFR monoclonal antibodies in CRC

OPPORTUNITY & NEXT STEPS

- KRAS G12C is 14% of mNSCLC (adenocarcinoma)
  - U.S.: 8,000 patients per year
  - Global: 32,000 patients per year
- KRAS G12C is 3% of mCRC
  - U.S.: 650 patients per year
  - Global: 4,000 patients per year
- Initial clinical data expected in 2022

Target occupancy (TO) predicted by mechanistic PK/PD model using mouse xenograft and cell-based studies that account for KRAS turnover, KRAS-GTP hydrolysis, GDP to GTP exchange, and KRAS-GDP binding to drug and inactiviation, relative to human free exposures; For adagrasib and sotorasib, PK of the RP2D and relative Koff values were used to predict TO; mNSCLC = metastatic non-small cell lung cancer; mCRC = metastatic colorectal cancer

PI3Kα (LOXO-783)

A POTENT, HIGHLY MUTANT-SELECTIVE, & BRAIN PENETRANT ALLOSTERIC PI3Kα H1047R INHIBITOR

SELECTIVITY DRIVES POTENTIAL FOR GREATER EFFICACY WITH IMPROVED TOLERABILITY

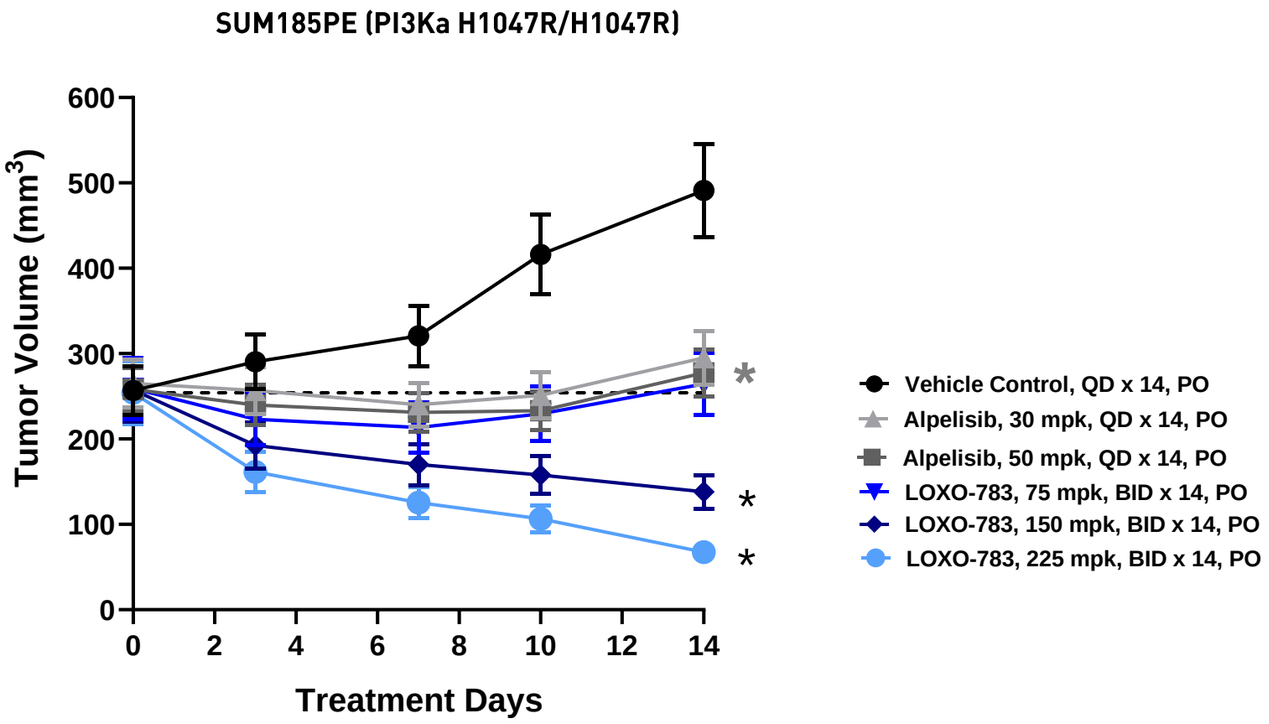
- PI3Kα H1047R are activating oncogenic events that occur in ~15% of breast cancers and less commonly in other cancers
- All approved PI3Kα inhibitors inhibit both wild-type and mutated PI3Kα with approximate equal potency resulting in potentially limited efficacy by on-target wild-type PI3Kα mediated toxicity including dose-limiting hyperglycemia as well as cutaneous and GI toxicity
- Human studies expected to begin in H1 2022

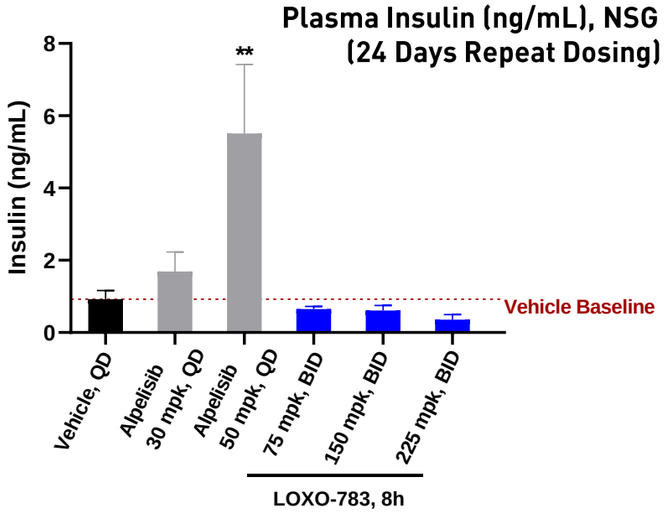
			LOXO-783	Alpelisib	Inavolisib (GDC-0077)
Cell AlphaLISA pAKT (pS473) IC_{50} (nM)	PI3Kα H1047R	T47D	3	64	14
		SUM185PE	1.3	43	14
		MDA-MB-453	5	49	16
	PI3Kα WT	SK-BR-3	286	128	30
	WT Selectivity		~90x	2.4x	2x
Cell proliferation IC_{50} (nM)	PI3Kα H1047R	T47D	4	396	72
		SUM185PE	3.2	356	44
		MDA-MB-453	9	727	57
	PI3Kα WT	SK-BR-3	>300	285	104
	WT Selectivity		Upper bound of selectivity NE	1x	1.7x
Other properties	CNS penetrance		Predicted to achieve meaningful brain exposure		
	Target occupancy		1-3 h	<10 mins	<10 mins

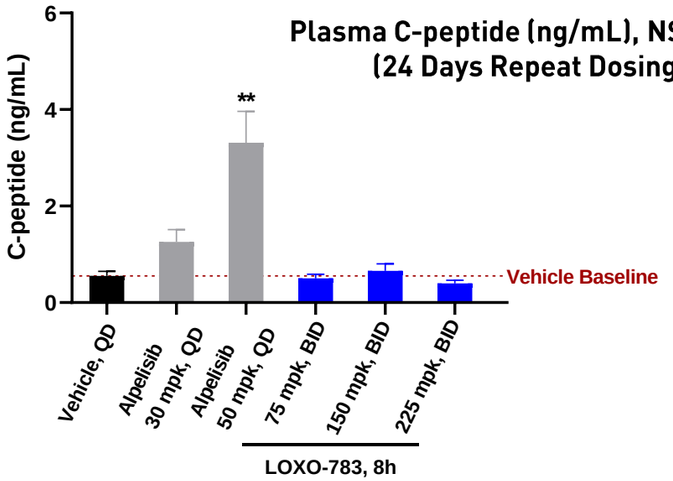
GI = gastrointestinal; IND = investigational new drug
Breast cancer cell lines used - T47D: $PI3K\alpha^{H1047R/WT}$ (ER+, HER2-); MDA-MB-453: $PI3K\alpha^{H1047R/WT}$ (ER-, HER2-); SUM185PE: $PI3K\alpha^{H1047R/H1047R}$ (ER-, HER2-); SK-BR-3: $PI3K\alpha^{WT/WT}$ (ER-, HER2+); NE = not estimable

PI3Kα (LOXO-783)

ACHIEVED SIGNIFICANT TUMOR REGRESSION IN PI3Kα H1047R BREAST CANCER XENOGRAFTS WITHOUT ANY INCREASES IN INSULIN OR C-PEPTIDE







*Alpelisib exposure at 50 mpk dose in NSG mice is ~2x higher than human exposure at approved dose

LOXO-783 showed greater activity than alpelisib at doses that did not cause any increase in plasma insulin or C-peptide

*p<0.05 compared to alpelisib on day 14. All treatments significantly (p<0.05) reduced tumor volume compared to the vehicle control on day 14. **p<0.05 compared to vehicle. Data are mean ± SEM

FGFR3 (LOXO-435)

FGFR1 AND FGFR2 INHIBITION DRIVE IMPORTANT, DOSE-LIMITING TOXICITIES

	Erdafitinib[1]	Pemigatinib[2]	Infigratinib[3]	Bemarituzumab[5]
Inhibitor classification	Pan-FGFR	Pan-FGFR	Pan-FGFR	FGFR2b mAb
Indication	*FGFR2/3*-altered UC	*FGFR2*-altered CCA	*FGFR2*-altered CCA	Investigational
Dose and schedule	8+1 mg/day (dose changes based on phosphate levels)	13.5 mg/day 14 days on, 7 days off	125 mg/day 21 days on, 7 days off	15 mg/kg every 2 weeks (plus 7.5 mg/kg on day 8 of first cycle only)
ORR	32.2%	36%	30.8%[4]	–
Hyperphosphatemia	76%	60%	46%[4]	–
Common adverse events	Hyperphosphatemia, ocular disorders, embryofetal toxicity	Hyperphosphatemia, ocular disorders, embryofetal toxicity	Hyperphosphatemia, ocular disorders, embryofetal toxicity	stomatitis, ocular disorders
Cell IC_{50} (nM)	1.7 (FGFR1) 1.0 (FGFR2) 3.3 (FGFR3 S249C)	0.9 (FGFR1) 1.5 (FGFR2) 2.0 (FGFR3 S249C)	4.3 (FGFR1) 4.9 (FGFR2) 8.4 (FGFR3 S249C)	–
Selectivity (target) vs FGFR3 S249C	3.3x (FGFR2), 1.0x (FGFR3)	1.4x (FGFR2)	1.7x (FGFR2)	–

- *FGFR1*-mediated hyperphosphatemia is a dose-limiting toxicity of pan-FGFR inhibitors[1-4]
- *FGFR2*-mediated cutaneous/nail, ocular, and perioral toxicities drive chronic intolerance of pan-FGFR inhibitors[5]

[1]Balversa. Prescribing information. Janssen Pharmaceutical Companies; 2020. [2]Pemazyre. Prescribing information. Incyte corporation; 2021. [3]Truseltiq. Prescribing information. QED Therapeutics; 2021. [4]Lyou Y et al. 2020 *JCO*, 38:15_suppl, 5038. [5]Catenacci DVT et al. 2021 *JCO*, 39:15_suppl (May 20, 2021) 4010. ORR = overall response rate; mAb = monoclonal antibody

FGFR3 (LOXO-435)

A POTENT AND HIGHLY ISOFORM-SELECTIVE FGFR3 INHIBITOR WITH GATEKEEPER ACTIVITY

Cellular signaling assays best exemplify LOXO-435's potential to avoid FGFR1/2-mediated toxicities

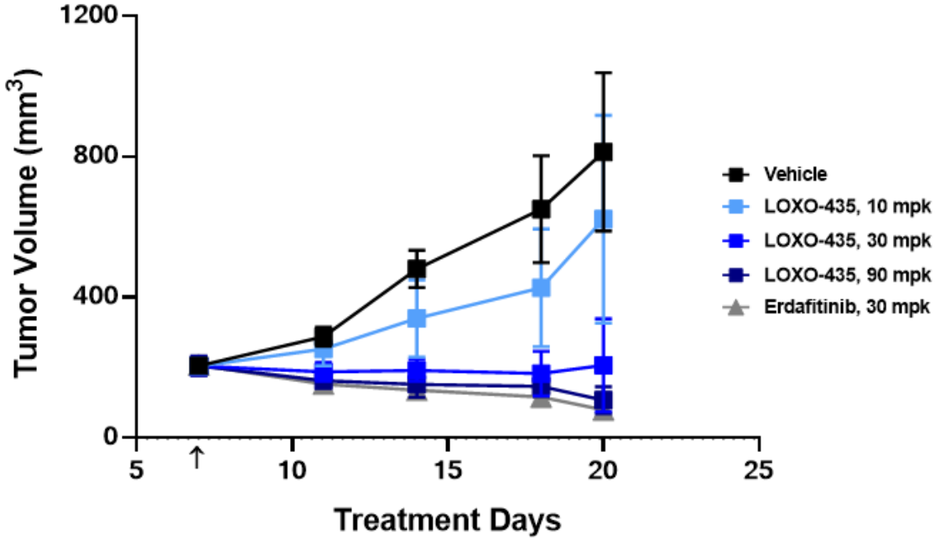
	Cellular Phospho Target Inhibition (HEK293)				Fold Selectivity	
	pFGFR1 IC_{50} (nM)	pFGFR2 IC_{50} (nM)	pFGFR3 S249C IC_{50} (nM)	pFGFR3 S249C, V555M IC_{50} (nM)	pFGFR3 S249C over pFGFR1	pFGFR3 S249C over pFGFR2
Erdafitinib	1.7	1.0	3.3	132.4	0.5x	0.3x
Pemigatinib	0.9	1.5	2.0	1451.7	0.5x	0.8x
Infigratinib	4.3	4.9	8.4	244.9	0.5x	0.6x
Futibatinib	1.0	1.0	2.7	63.4	0.4x	0.4x
LOXO-435	207.8	112.2	3.4	9.7	61x	33x

- Activating FGFR3 gene alterations are found in ~15-20% of advanced bladder cancers
- All approved and investigational FGFR small molecule inhibitors are similarly potent against FGFR1-3 resulting in potentially limited efficacy by toxicities driven by inhibition of both FGFR1 and FGFR2
- Existing drugs lose potency in the setting of FGFR3 gatekeeper mutations, which have been reported as mechanisms of acquired resistance to existing pan-FGFR inhibitors
- Human studies expected to begin in H2 2022

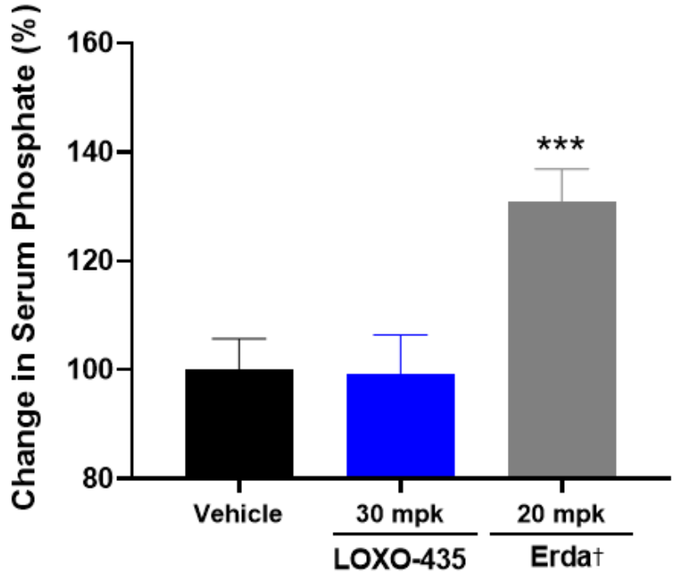
Cell based phosphorylation assays were conducted using HEK293 cell lines engineered to express FGFR1, FGFR2, FGFR3 (S249C) and FGFR3 (S249C, V555M). Assays were developed utilizing In-Cell Western (LICOR) by monitoring FGFR activation loop phosphorylation normalized to GAPDH antibody signal

FGFR3 (LOXO-435)
DISPLAYS A WIDE THERAPEUTIC INDEX IN VIVO

LOXO-435 in UMUC-14 (FGFR3 S249C)



Serum phosphate in rat



LOXO-435 caused tumor regressions without hyperphosphatemia

Rodent efficacy and target inhibition studies were performed by subcutaneous implantation of human urothelial cancer line UMUC-14 in immunocompromised mice. Treatment started when the average tumor size was between 150-200 mm^3; ↑ = Start of treatment; Total serum phosphate changes determined by measuring total serum phosphate pre and post treatment, then compared to vehicle control; ***p≤0.0001 compared to vehicle. †Erdafitinib MTD in rats is 20 mpk; Note: LOXO-435, 90 mpk rat hyperphosphatemia study in progress

ONCOLOGY BUSINESS DEVELOPMENT

PETRA PHARMA

- Strategic acquisition of a lead optimization preclinical small molecule program (mutant-selective PI3Kα)
- Expected to start human studies in the first half of 2022
- Potential to differentiate with unique target profile that could drive greater efficacy and improved tolerability

Merus

- Collaboration to discover novel T-cell re-directing bispecific antibodies
- Differentiated platform in a class with emerging clinical importance

FOGHORN® THERAPEUTICS

- Collaboration for novel oncology targets using proprietary gene traffic control platform and structural biology insights
- Establishes co-development and co-commercialization agreement on selective BRM program and an additional undisclosed program
- Includes three additional discovery programs

BRM = Brahma

LILLY ONCOLOGY PIPELINE

SELECT NME AND NILEX PIPELINE AS OF OCTOBER 22, 2021

Pre-Clinical	PHASE 1			PHASE 2		PHASE 3		REG REVIEW
				TIRZEPATIDE NASH				
				IL-2 CONJUGATE Ulcerative Colitis	PIRTOBRUTINIB ★ B-Cell Malignancies	PIRTOBRUTINIB ★ R/R CLL Combination		
TAU siRNA Alzheimer's				GGG TRI-AGONIST Obesity	GBA1 GENE THERAPY Gaucher Disease Type 2	TIRZEPATIDE CV Outcomes	TIRZEPATIDE Obesity	
SARM1 INHIBITOR Pain	RIPK1 INHIBITOR Immunology			TRPA1 ANTAGONIST Pain	GLP-1R NPA Obesity	SELPERCATINIB ★ 1L NSCLC	TIRZEPATIDE Heart Failure pEF	
PI3Ka SELECTIVE ★ Cancer	P2X7 INHIBITOR Pain	PYY ANALOG Diabetes	RELAXIN-LA Heart Failure	SSTR4 AGONIST Pain	GLP-1R NPA Diabetes	PIRTOBRUTINIB ★ R/R MCL Monotherapy	SELPERCATINIB ★ 1L Med Thyroid Cancer	
FGFR3 INHIBITOR ★ Cancer	NRG4 AGONIST Heart Failure	O-GLCNACASE INH Alzheimer's	OXYNTOMODULIN Diabetes	PACAP38 MAB Migraine	PD-1 MAB AGONIST Rheumatoid Arthritis	EMPAGLIFLOZIN* Post MI	MIRIKIZUMAB Crohn's Disease	
2nd GENERATION RET INHIBITOR Cancer	LP(a) siRNA CVD	N3PG Aβ MAB Alzheimer's	NOT DISCLOSED Diabetes	IL-2 CONJUGATE Systemic Lupus Erythematosus	MEVIDALEN Symptomatic LBD	BARICITINIB Systemic Lupus Erythematosus	EMPAGLIFLOZIN* Chronic Kidney Disease	EMPAGLIFLOZIN* Heart Failure pEF
BCL2 INHIBITOR Cancer	KHK INHIBITOR II Diabetes / NASH	KRAS G12C II ★ Cancer	LP(a) INHIBITOR CVD	GBA1 GENE THERAPY Parkinson's Disease	GRN GENE THERAPY Frontotemporal Dementia	ABEMACICLIB ★ HER2+ Early BC	ABEMACICLIB ★ Prostate Cancer	CONNECTED CARE PREFILLED INSULIN PEN Diabetes
DACRA Obesity / Diabetes	GIPR AGONIST LA II Diabetes	IDH1/2 INHIBITOR ★ Cancer	IL-17A SMALL MOL INHIBITOR Immunology	EPIREG/TGFα MAB Chronic Pain	GGG TRI-AGONIST Diabetes	SOLANEZUMAB Preclinical AD	IMLUNESTRANT ★ ER+ HER2- mBC	BARICITINIB Alopecia Areata
LAARA Obesity / Diabetes	CD200R MAB AGONIST Immunology	GIP/GLP COAGONIST PEPTIDE Diabetes	GIPR AGONIST LA Diabetes	BEBTELOVIMAB (LY-CoV1404 MAB) COVID-19	CXCR1/2L MAB Hidradenitis Suppurativa	MIRIKIZUMAB Ulcerative Colitis	PIRTOBRUTINIB ★ R/R CLL Monotherapy	TIRZEPATIDE Diabetes
Glucose Sensing Insulin Diabetes	ANGPTL3 siRNA CVD	AUR A KINASE INHIBITOR Cancer	BTLA MAB AGONIST Immunology	AUTOMATED INSULIN DELIVERY SYS Diabetes	BASAL INSULIN-FC Diabetes	DONANEMAB^ Early Alzheimer's	LEBRIKIZUMAB Atopic Dermatitis	SINTILIMAB (US)* NonSquam NSCLC 1L

LEGEND

- NME
- NILEX
- * *Commercial Collaboration*
- ^ *Rolling submission in the U.S. initiated*
- ☆ *Discussed in Dec 15 Investment Community Meeting*

Note: select pre-clinical assets listed, most of which were discussed at the Lilly Investment Community meeting on December 15, 2021; NME = new molecular entity; NILEX = new indication or line extension

ONCOLOGY SUMMARY

- Our oncology portfolio is anchored by important medicines including Verzenio, Retevmo and pirtobrutinib which have the potential to deliver meaningful growth over the course of the decade
- Mid-stage portfolio, including IDH 1/2, KRAS G12C, and imlunestrant are poised to deliver new data and potential new trial starts in 2022
- Next year, we expect to initiate human trials for two new assets, LOXO-783 and LOXO-435, that each highlight a core of our philosophy of impacting outcomes through improved target coverage

JAKE VAN NAARDEN

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Lilly Oncology

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Chief Scientific and Medical Officer,
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Lilly

2021 **INVESTMENT COMMUNITY MEETING**