SAFE HARBOR PROVISION

The presentations for Eli Lilly’s investment community meeting contain forward-looking statements that are based on management’s current expectations, but actual results may differ materially due to various factors. The company’s results may be affected by factors including, but not limited to, the risks and uncertainties in pharmaceutical research and development; competitive developments; regulatory actions; the extent and duration of the effects of the COVID-19 pandemic; litigation and investigations; business development transactions; economic conditions; and changes in laws and regulations, including health care reform.

For additional information about the factors that affect the company’s business, please see the company’s latest Forms 10-K, 10-Q, and any 8-Ks filed with the Securities and Exchange Commission. In addition, certain financial information in this presentation is presented on a non-GAAP basis. Investors should refer to the reconciliations included in these presentations and should consider the company’s non-GAAP measures in addition to, not as a substitute for or superior to, measures prepared in accordance with GAAP.

The company undertakes no duty to update forward-looking statements except as required by applicable law.
JAKE VAN NAARDEN
CEO of Loxo Oncology at Lilly, and President, Lilly Oncology

DAVID HYMAN, M.D.
Chief Medical Officer, Lilly Oncology
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

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

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Analysis landmark | IDFS
--- | ---
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: 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

Verzenio in combination with ET is approved for use in patients with HR+ HER2- high-risk, early breast cancer and a Ki-47 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

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2021 INVESTMENT COMMUNITY MEETING
As expected, high Ki-67 index was prognostic of worse outcome. However, abemaciclib had a similar effect size regardless of Ki-67 index.
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

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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

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PIRTOBRUTINIB
FOUR OF FIVE GLOBAL STUDIES INITIATED IN 2021

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

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PIRTOBRUTINIB
EFFICACY IN BTK PRE-TREATED CLL/SLL PATIENTS

Overall Response Rate Over Time

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 BCL2)

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. **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. 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. 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

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2021 INVESTMENT COMMUNITY MEETING
PIRTOBRUTINIB
EFFICACY IN MANTLE CELL LYMPHOMA (MCL)

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. †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.

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

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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

- 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
- Treatment until progressive disease, unacceptable toxicity, or death

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

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**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

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KRAS G12C (LY3537982)

BIOLOGIC RATIONALE

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 inactivation, 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

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New Important IND: LOXO-783 is a highly potent, mutant-selective, & brain penetrant allosteric PI3Kα H1047R inhibitor (advance from Petra acquisition)

**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

<table>
<thead>
<tr>
<th>Cell AlphaLISA pAKT (pS473) IC₅₀ (nM)</th>
<th>PI3Kα H1047R</th>
<th>T47D</th>
<th>SUM185PE</th>
<th>MDA-MB-453</th>
<th>PI3Kα WT</th>
<th>SK-BR-3</th>
<th>WT Selectivity</th>
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<tbody>
<tr>
<td>LOXO-783</td>
<td>3</td>
<td>64</td>
<td>14</td>
<td>14</td>
<td>14</td>
<td>14</td>
<td>~90x</td>
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<tr>
<td>Alpelisib (GDC-0077)</td>
<td>1.3</td>
<td>43</td>
<td>14</td>
<td>16</td>
<td>30</td>
<td>30</td>
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<tr>
<td>Inavolisib (GDC-0077)</td>
<td>5</td>
<td>49</td>
<td></td>
<td></td>
<td>128</td>
<td>30</td>
<td>2x</td>
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<table>
<thead>
<tr>
<th>Cell proliferation IC₅₀ (nM)</th>
<th>PI3Kα H1047R</th>
<th>T47D</th>
<th>SUM185PE</th>
<th>MDA-MB-453</th>
<th>PI3Kα WT</th>
<th>SK-BR-3</th>
<th>WT Selectivity</th>
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<tr>
<td>LOXO-783</td>
<td>4</td>
<td>396</td>
<td>72</td>
<td></td>
<td>4</td>
<td>356</td>
<td>44</td>
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<tr>
<td>Alpelisib (GDC-0077)</td>
<td>3.2</td>
<td>356</td>
<td>44</td>
<td></td>
<td>3.2</td>
<td>727</td>
<td>67</td>
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<tr>
<td>Inavolisib (GDC-0077)</td>
<td>9</td>
<td>727</td>
<td>67</td>
<td></td>
<td>9</td>
<td>285</td>
<td>104</td>
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</table>

<table>
<thead>
<tr>
<th>PI3Kα WT</th>
<th>SK-BR-3</th>
<th>WT Selectivity</th>
<th>Upper bound of selectivity NE</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt;300</td>
<td>285</td>
<td>1x</td>
<td>1.7x</td>
</tr>
</tbody>
</table>

Other properties

- CNS penetration: Predicted to achieve meaningful brain exposure
- Target occupancy: 1-3 h, <10 mins

GI = gastrointestinal; IND = investigational new drug
Breast cancer cell lines used - T47D: PI3Kα H1047R/WT [ER+, HER2-]; MDA-MB-453: PI3Kα H1047R/WT [ER+, HER2-]; SUM185PE: PI3Kα H1047R/WT [ER-, HER2-]; SK-BR-3: PI3Kα H1047R/WT [ER-, HER2+]; NE = not estimable

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2021 INVESTMENT COMMUNITY MEETING
**PI3Ka (LOXO-783)**

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

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

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

*<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

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# FGFR3 (LOXO-435)
**FGFR1 AND FGFR2 INHIBITION DRIVE IMPORTANT, DOSE-LIMITING TOXICITIES**

<table>
<thead>
<tr>
<th>Inhibitor classification</th>
<th>Erdafitinib&lt;sup&gt;1&lt;/sup&gt;</th>
<th>Pemigatinib&lt;sup&gt;2&lt;/sup&gt;</th>
<th>Infigratinib&lt;sup&gt;3&lt;/sup&gt;</th>
<th>Bemarituzumab&lt;sup&gt;5&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>Indication</td>
<td>Pan-FGFR</td>
<td>Pan-FGFR</td>
<td>Pan-FGFR</td>
<td>FGFR2b mAb</td>
</tr>
<tr>
<td>Dose and schedule</td>
<td>FGFR2/3-altered UC</td>
<td>FGFR2-altered CCA</td>
<td>FGFR2-altered CCA</td>
<td>Investigational</td>
</tr>
<tr>
<td>8×1 mg/day</td>
<td>13.5 mg/day</td>
<td>125 mg/day</td>
<td>15 mg/kg every 2 weeks (plus 7.5 mg/kg on day 8 of first cycle only)</td>
<td></td>
</tr>
<tr>
<td>ORR</td>
<td>32.2%</td>
<td>36%</td>
<td>30.8%</td>
<td>--</td>
</tr>
<tr>
<td>Hyperphosphatemia</td>
<td>76%</td>
<td>60%</td>
<td>46%</td>
<td>--</td>
</tr>
<tr>
<td>Common adverse events</td>
<td>Hyperphosphatemia, ocular disorders, embryofetal toxicity</td>
<td>Hyperphosphatemia, ocular disorders, embryofetal toxicity</td>
<td>Hyperphosphatemia, ocular disorders, embryofetal toxicity</td>
<td>stomatitis, ocular disorders</td>
</tr>
<tr>
<td>Cell IC&lt;sub&gt;50&lt;/sub&gt; (nM)</td>
<td>1.7 [FGFR1]&lt;br&gt;1.0 [FGFR2]&lt;br&gt;3.3 [FGFR3 S249C]</td>
<td>0.9 [FGFR1]&lt;br&gt;1.5 [FGFR2]&lt;br&gt;2.0 [FGFR3 S249C]</td>
<td>4.3 [FGFR1]&lt;br&gt;4.9 [FGFR2]&lt;br&gt;8.4 [FGFR3 S249C]</td>
<td>--</td>
</tr>
<tr>
<td>Selectivity (target) vs FGFR3 S249C</td>
<td>3.3x [FGFR2], 1.0x [FGFR3]</td>
<td>1.4x [FGFR2]</td>
<td>1.7x [FGFR2]</td>
<td>--</td>
</tr>
</tbody>
</table>

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FGFR1-mediated hyperphosphatemia is a dose-limiting toxicity of pan-FGFR inhibitors<sup>1-4</sup>

FGFR2-mediated cutaneous/nail, ocular, and periorial toxicities drive chronic intolerance of pan-FGFR inhibitors<sup>5</sup>

---

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FGFR3 (LOX0-435)
A POTENT AND HIGHLY ISOFORM-SELECTIVE FGFR3 INHIBITOR WITH GATEKEEPER ACTIVITY

Cellular signaling assays best exemplify LOX0-435’s potential to avoid FGFR1/2-mediated toxicities

<table>
<thead>
<tr>
<th>Cellular Phospho Target Inhibition (HEK293)</th>
<th>Fold Selectivity</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>pFGFR1 IC₅₀ (nM)</td>
</tr>
<tr>
<td>Erdafitinib</td>
<td>1.7</td>
</tr>
<tr>
<td>Pemigatinib</td>
<td>0.9</td>
</tr>
<tr>
<td>Infigratinib</td>
<td>4.3</td>
</tr>
<tr>
<td>Futibatinib</td>
<td>1.0</td>
</tr>
<tr>
<td>LOX0-435</td>
<td>207.8</td>
</tr>
</tbody>
</table>

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

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2021 INVESTMENT COMMUNITY MEETING
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 mm³. 

† = 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

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ONCOLOGY BUSINESS DEVELOPMENT

PETRA PHARMA

- Strategic acquisition of a lead optimization preclinical small molecule program (mutant-selective PI3Ka)
- 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
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**LILLY ONCOLOGY PIPELINE**

**SELECT NME AND NILEX PIPELINE AS OF OCTOBER 22, 2021**

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

<table>
<thead>
<tr>
<th>CATEGORY</th>
<th>THERAPEUTIC CATEGORY</th>
<th>DISEASE</th>
<th>STATUS</th>
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</thead>
<tbody>
<tr>
<td>Pre-Clinical</td>
<td>TAU siRNA</td>
<td>Alzheimer’s</td>
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<tr>
<td></td>
<td>SARM1 INHIBITOR</td>
<td>Pain</td>
<td></td>
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<tr>
<td></td>
<td>PI3K σ SELECTIVE</td>
<td>Cancer</td>
<td></td>
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<tr>
<td></td>
<td>FGFR3 INHIBITOR</td>
<td>Cancer</td>
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<tr>
<td></td>
<td>2nd GENERATION RET INHIBITOR</td>
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<td>BCL2 INHIBITOR</td>
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<td>DACRA</td>
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<td></td>
<td>LAARA</td>
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<td>Glucose Sensing Insulin</td>
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<td>CD200R MAB AGONIST</td>
<td>Immunology</td>
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<td>RIPK1 INHIBITOR</td>
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<td>P2X7 INHIBITOR</td>
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<tr>
<td></td>
<td>NRG4 AGONIST</td>
<td>Heart Failure</td>
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<td>PYY ANALOG</td>
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<td>LIPA σ siRNA</td>
<td>Osteoarthritis</td>
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<td>KHK INHIBITOR</td>
<td>Diabetes / MASH</td>
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<td>GIPR AGONIST LA II</td>
<td>Diabetes</td>
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<td>IDH1/2 INHIBITOR</td>
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<td>GIPR AGONIST LA</td>
<td>Diabetes</td>
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<td>ANGPTL3 σ siRNA</td>
<td>CVD</td>
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<td>AUR A KINASE INHIBITOR</td>
<td>Cancer</td>
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**PHASE 1**

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<thead>
<tr>
<th>THERAPEUTIC CATEGORY</th>
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<tbody>
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<td>GIPR AGONIST LAM</td>
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<td>TRPA1 AGONIST</td>
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<tr>
<td>SSTR4 AGONIST</td>
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<td>RACAP33 MAB</td>
<td>Migraine</td>
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<td>IL-2 CONJUGATE</td>
<td>Systemic Lupus Erythematosus</td>
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<td>Parkinson’s Disease</td>
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<td>EPIREG/TGFβ MAB</td>
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<td>BETTELOVIMAB (NX-CG/G4 MAB)</td>
<td>COVID-19</td>
<td></td>
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<td>AUTOMATED INSULIN DELIVERY SYS</td>
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**PHASE 2**

<table>
<thead>
<tr>
<th>THERAPEUTIC CATEGORY</th>
<th>DISEASE</th>
<th>STATUS</th>
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</thead>
<tbody>
<tr>
<td>GIP/GLP COAGONIST PEPTIDE</td>
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<tr>
<td>GIPR AGONIST LA</td>
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<tr>
<td>BTLA MAB AGONIST</td>
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<td>IL-2 CONJUGATE</td>
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<tr>
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<tr>
<td>SSTR4 AGONIST</td>
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</tr>
<tr>
<td>RACAP33 MAB</td>
<td>Migraine</td>
<td></td>
</tr>
<tr>
<td>IL-2 CONJUGATE</td>
<td>Systemic Lupus Erythematosus</td>
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<tr>
<td>GBA1 GENE THERAPY</td>
<td>Parkinson’s Disease</td>
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</tr>
<tr>
<td>EPREG/TGFβ MAB</td>
<td>Chronic Pain</td>
<td></td>
</tr>
<tr>
<td>BETTELOVIMAB</td>
<td>COVID-19</td>
<td></td>
</tr>
<tr>
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**PHASE 3**

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<td>B-Cell Malignancies</td>
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<tr>
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<td>Ulcerative Colitis</td>
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<tr>
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<td>Pain</td>
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<tr>
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</tr>
</tbody>
</table>

**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

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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.