This presentation contains forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995. These statements are often, but not always, made through the use of words or phrases such as “anticipates”, “expects”, “plans”, “believes”, “intends”, and similar words or phrases. Such statements involve risks and uncertainties that could cause Checkpoint Therapeutics’ actual results to differ materially from the anticipated results and expectations expressed in these forward-looking statements. These statements are only predictions based on current information and expectations and involve a number of risks and uncertainties. Actual events or results may differ materially from those projected in any such statements due to various factors, including the risks and uncertainties inherent in clinical trials, drug development, and commercialization. You are cautioned not to place undue reliance on these forward-looking statements, which speak only as of the date hereof. All forward-looking statements are qualified in their entirety by this cautionary statement and Checkpoint Therapeutics undertakes no obligation to update these statements, except as required by law.
Corporate Overview

• Immuno-oncology biopharmaceutical company focused on the acquisition, development and commercialization of immune-enhanced combination treatments for patients with solid tumor cancers
  – Two immuno-oncology “I/O” antibodies
  – Four targeted anti-cancer agents

• Majority-controlled subsidiary of Fortress Biotech (Nasdaq:FBIO)
  – Form 10 filed to become a separate publicly-reporting company
  – 4Q 2015: Raised $58 million in gross proceeds (common stock/warrants)

• Collaboration with TG Therapeutics (Nasdaq:TGTX) to develop I/O antibodies in combination with TG’s targeted therapies for liquid tumors (e.g. Non-Hodgkin’s Lymphoma and CLL)
Mission Statement

Develop first-in-class or best-in-class combination treatments in targeted solid tumor cancers

...and liquid tumors in collaboration with TGTX
<table>
<thead>
<tr>
<th>Compound &amp; Indication</th>
<th>Preclinical</th>
<th>Phase I</th>
<th>Phase II</th>
<th>Phase III</th>
<th>Originator</th>
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<tbody>
<tr>
<td><strong>Immuno-Oncology Agents</strong></td>
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<tr>
<td>Anti-PD-L1 (Multiple forms of Cancer)</td>
<td></td>
<td>Target IND: 1H 2017</td>
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<td>Dana Farber</td>
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<tr>
<td>Anti-GITR (Multiple forms of Cancer)</td>
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<td>Target IND: 2H 2017</td>
<td></td>
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<td>Dana Farber</td>
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<tr>
<td><strong>Targeted Anti-Cancer Agents</strong></td>
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</tr>
<tr>
<td>CK-101 EGFR Inhibitor (Lung Cancer)</td>
<td></td>
<td>Phase 1 pending</td>
<td></td>
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<td>NeuPharma</td>
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<td>CK-102 PARP Inhibitor (Multiple forms of Cancer)</td>
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<td>Phase 1b: 2017</td>
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<td>Teva/Cephalon</td>
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<tr>
<td>CK-103 BET Inhibitor (Multiple forms of Cancer)</td>
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<td>Jubilant</td>
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<tr>
<td>Anti-CAIX (Renal cell Carcinoma, CAIX+ solid tumors)</td>
<td></td>
<td>Target IND: 2H 2017</td>
<td></td>
<td></td>
<td>Dana Farber</td>
</tr>
</tbody>
</table>
To acquire and develop novel immuno-oncology and targeted anti-cancer agents alone and in combination to treat patients with solid tumor cancers.

**Targeted Anti-Cancer Agents**

- Anti-EGFR small molecule for Lung Ca
- PARP-inhibitor for multiple cancers
- BET inhibitor for multiple cancers
- Anti-CAIX monoclonal antibody for RCC

...and from TG collaboration:
- PI3K Delta small molecule
- Anti-CD20 monoclonal antibody

**Immuno-Oncology Agents**

- Anti-PD-L1

**Additional I/O Agents** to augment immune system engagement
- Anti-GITR monoclonal antibody
- Others under consideration
Goal of I/O therapy:

To engage the immune system (particularly Killer T-Cells) to kill cancer cells.
Immuno-Oncology Agents
Current Pipeline

• **Anti-PD-L1 mAb**
  – A fully human antagonistic antibody that binds PD-L1 and opens up cancer cells to immune system attack
  – Retained native Fc-region could engage the innate immune system and induce antibody-dependent cell-mediated cytotoxicity (ADCC).
  – Applicability across many different cancers

• **Anti-GITR mAb**
  – A fully human agonistic antibody that binds and triggers signaling in GITR expressing T-reg cells permitting immune attack of cancer cells
  – Designed to be synergistic with anti-PD-L1 to engage the immune system to attack cancer cells
PD-L1 and Effect of Anti-PD-L1 Antibodies

PD-L1 Ligand protects cancer cells by de-activating tumor specific T-cell responses.

Anti-PD-L1 monoclonal antibody

PD-L1 ligand on tumor cells

Anti-PD-L1 monoclonal antibodies block PD-L1 signaling allowing the immune system’s T-cells to attack the cancer.
In addition to the tumor’s defense, the immune system may also block T-Cells from attacking the cancer.

Anti-GITR antibodies are believed to be able to block T-reg function, permitting T-cells to attack the cancer.
• Anti-PD-L1 antibody is in IND-enabling studies
  – IND targeted for 1H2017

• Anti-GITR antibody is in lead selection
  – IND targeted for 2H2017

• Other I/O targeted agents in exploratory stages
Targeted Anti-Cancer Therapies

Goal of targeted anti-cancer therapy:

To apply direct anti-cancer killing by targeting known (in humans) active cancer pathways or driver mutations
Targeted Anti-Cancer Therapies
Current Pipeline

- CK-101, oral, third-generation EGFR inhibitor for lung cancer with T790M mutation
- CK-102, oral, small molecule selective inhibitor of PARP-1 and PARP-2 enzymes
- CK-103, oral, small molecule inhibitor of the BET protein, BRD4
- Anti-CAIX fully human antibody for CAIX+ RCC and other CAIX+ solid tumors
CK-101, 3rd Generation EGFR Rationale

• EGFR Mutations – Validated Target
  – Success of 1st generation EGFR’s have led to acquired resistance through further mutations to EGFR (T790M)
  – One 3rd generation EGFR inhibitor (Tagrisso™) is approved for patients with lung cancer with T790M mutation

• CK-101 has potential safety advantages
  – Tagrisso™ has significant skin tox due to also targeting wild-type EGFR
  – CK-101 has limited targeting of wild-type
CK-101, 3rd Generation EGFR Pre-Clinical Efficacy

CK-101:
• Strong efficacy for T790M and Del19 mutations
• Good selectivity for mutant:
  – A431/H1975 ratio ~ 100 fold
• No efficacy for EGFR wt cell line (A431)

<table>
<thead>
<tr>
<th>Cell Line</th>
<th>A431</th>
<th>H1975</th>
<th>HCC827</th>
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<tr>
<td>Mutation</td>
<td>EGFR WT</td>
<td>L858R/T790M</td>
<td>Exon 19 del</td>
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<tr>
<td>Afatinib</td>
<td>34</td>
<td>23</td>
<td>1</td>
</tr>
<tr>
<td>Tagrisso</td>
<td>280</td>
<td>2</td>
<td>3</td>
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<tr>
<td>CK-101</td>
<td>689</td>
<td>5</td>
<td>10</td>
</tr>
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</table>

H1975 NSCLC xenograft model

Checkerpoint, data on file.
CK-101, Phase 1/2 Study

- IND approved; Phase 1 to commence in September
  - Approx. 30 advanced solid tumor patients
  - Dose escalation study to determine MTD and RP2D

- Phase 2 – Approx. 60 NSCLC patients with T790M mutation
  - Primary endpoint: Objective response rate (ORR)

- Plan to develop as a monotherapy and in combination with synergistic I/O agents
### Annual Incidence of NSCLC with T790M Mutation

<table>
<thead>
<tr>
<th>Type</th>
<th>U.S.</th>
<th>E.U.</th>
<th>Japan</th>
</tr>
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<tbody>
<tr>
<td>NSCLC Annual Incidence</td>
<td>190,000</td>
<td>260,000</td>
<td>80,000</td>
</tr>
<tr>
<td>EGFR Mutations L858R, del(19)</td>
<td>19,000</td>
<td>26,000</td>
<td>24,000</td>
</tr>
<tr>
<td>T790M Resistant</td>
<td>9,000</td>
<td>12,000</td>
<td>10,000</td>
</tr>
</tbody>
</table>

**Annual Incidence: 31,000 patients worldwide**

CK-102, PARP-Inhibitor Rationale and Status

• PARP enzymes are involved in normal cellular homeostasis, such as DNA transcription, cell cycle regulation, and DNA repair
  – PARP activity and expression is up-regulated in certain tumor cells and believed to contribute to resistance
  – By inhibiting PARP, certain cancer cells may be unable to repair single strand DNA breaks, which in turn causes double strand DNA breaks, leading to cancer cell death

• Promising activity for PARP inhibitors seen across multiple tumor types
  – Breast, ovarian, prostate cancer
  – Particularly in tumors with existing DNA repair defects: BRCA1 and BRCA2

• Evaluating alternative drug product formulations. Planning to commence a Phase 1b clinical study in next 6-12 months
• BET proteins, such as BRD4, play a pivotal role in regulating the transcription of key regulators of cancer cell growth and survival, including the c-Myc oncogene
  – BRD4 is often required for expression of c-Myc
  – Inhibition of BRD4 may lead to selective killing of tumor cells across a broad range of hematologic malignancies and certain targeted solid tumors, such as those associated with elevated c-Myc expression.

• Targeting IND filing in 1H 2017
Anti-CAIX mAb
Rationale and Status

• CAIX is highly expressed on Renal Cell Carcinoma (RCC)
  – Limited expression on healthy tissue
  – CAIX plays an important role in tumor progression and metastasis

• Anti-CAIX monoclonal antibody elicits strong ADCC and CDC mediated cell killing and inhibits human RCC tumors in mice

• Targeting IND filing in 2H 2017
Targeted Anti-Cancer Therapies Collaboration with TG Therapeutics

- Joint development of anti-PD-L1 and anti-GITR antibodies, and BET inhibitor program
- Checkpoint to focus on solid tumor indications and TGTX to focus on liquid tumors
- Checkpoint is eligible for royalties and milestones
Key Takeaways

• Building a platform to combine targeted agents with immuno-oncology agents to maximize anti-cancer effect
  – CK-101 (EGFR) IND approved; Phase 1/2 initiation planned for September
  – Anti-PD-L1 IND expected in 1H17
  – CK-103, anti-GITR and anti-CAIX INDs expected in 2017
  – CK-102 (PARP) Phase 1b planned in 6-12 months
  – Developing each as a monotherapy, followed by combinations for enhanced activity

• Funded with approximately two years of cash to support development programs