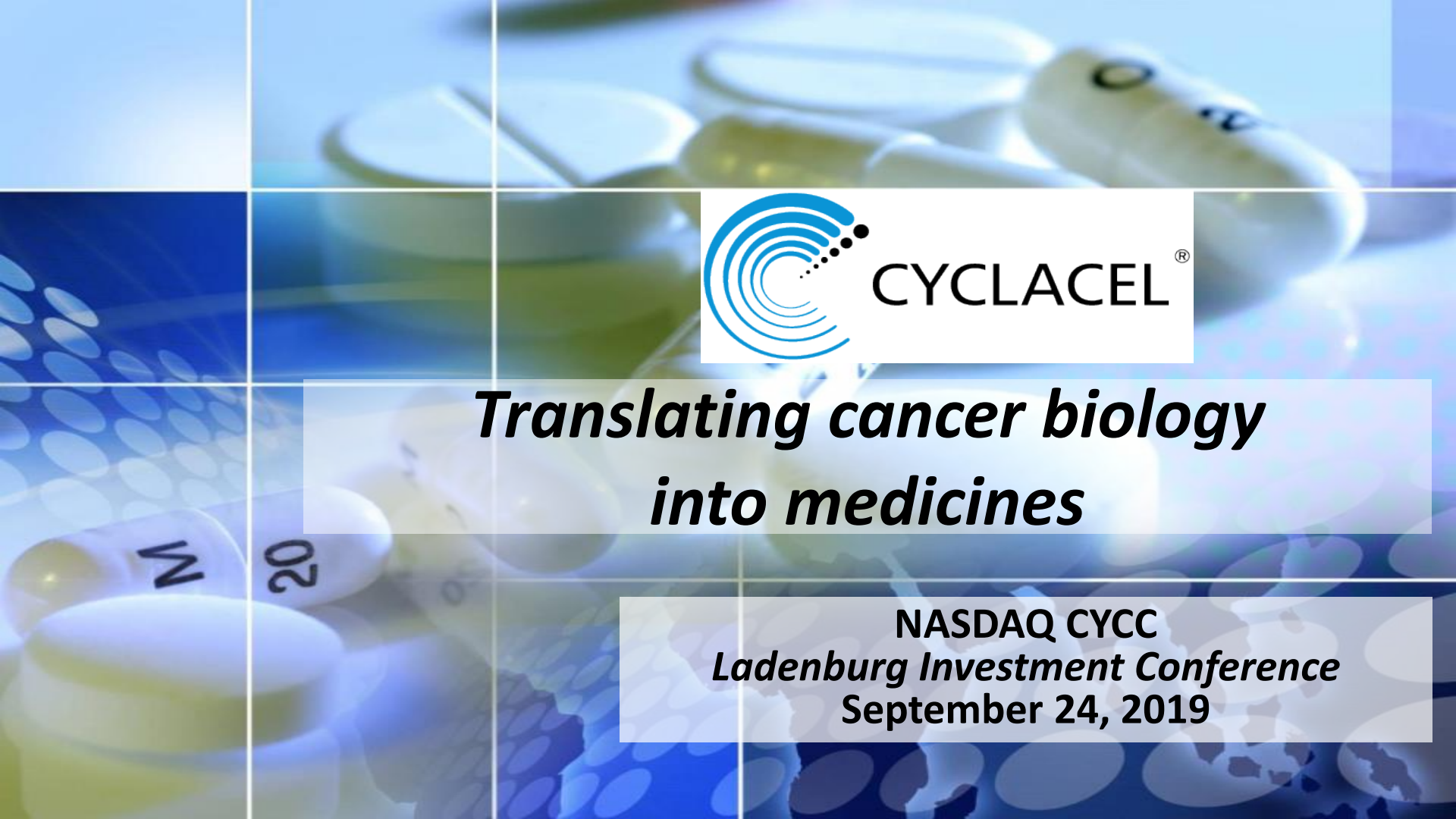


Translating cancer biology into medicines

NASDAQ CYCC
Ladenburg Investment Conference
September 24, 2019

Disclaimer

This presentation contains forward-looking statements within the meaning of the “safe harbor” provisions of the Private Securities Litigation Reform Act of 1995 about financial results and estimates, business strategy, clinical trial plans and research and development programs of Cyclacel Pharmaceuticals, Inc. By their nature, forward-looking statements and forecasts involve risks and uncertainties because they relate to events and depend on circumstances that will occur in the future. For a further list and description of the risks and uncertainties the Company faces, please refer to our most recent Annual Report on Form 10-K and other periodic and current filings that have been filed with the Securities and Exchange Commission and are available at www.sec.gov. The information in this presentation is current as of this date. Cyclacel does not take any responsibility to update such information.

Cyclacel Overview

- Apply deep understanding of cell cycle biology to disrupt cancer
    - **resistance**
    - **DNA repair** or evasion
- Targetable precision medicine strategy:
    - **MCL1** in leukemias (Phase 1)
    - **BRCA1/2** in breast cancer (Phase 1/2)
- Experienced management; estimated capital to end of 2020

Clinical Stage Value Drivers

CYC065

- CDK inhibitor with proof of mechanism (down-regulation of MCL1) in humans
- 2L venetoclax combination in leukemias (CLL, AML)

Sapacitabine

- Oral nucleoside analogue, unique DNA damage response mechanism for BRCA +ve patients
- 2L olaparib combination in BRCA +ve breast cancer

CYC140

- PLK inhibitor with compelling preclinical data in liquid & solid cancers

Addressing Large Markets

CLL 2L

- 21k US incidence; majority on ibrutinib (BTKi)
- venetoclax (1L with ibrutinib or 2L)

CYC065

AML elderly unfit for chemotherapy

- ~16k US incidence; venetoclax+HMA (aza or dec)
- venetoclax combination

CYC065 / sapa

BRCA +ve Breast Cancer

- ~11-15k US incidence; olaparib or other PARPi
- olaparib combination

sapa

Suppressing Resistance Proteins

↑ *protein expression=survival/growth of cancer cells*

- **BCL2** > **venetoclax** approved in 2L CLL & 1L AML

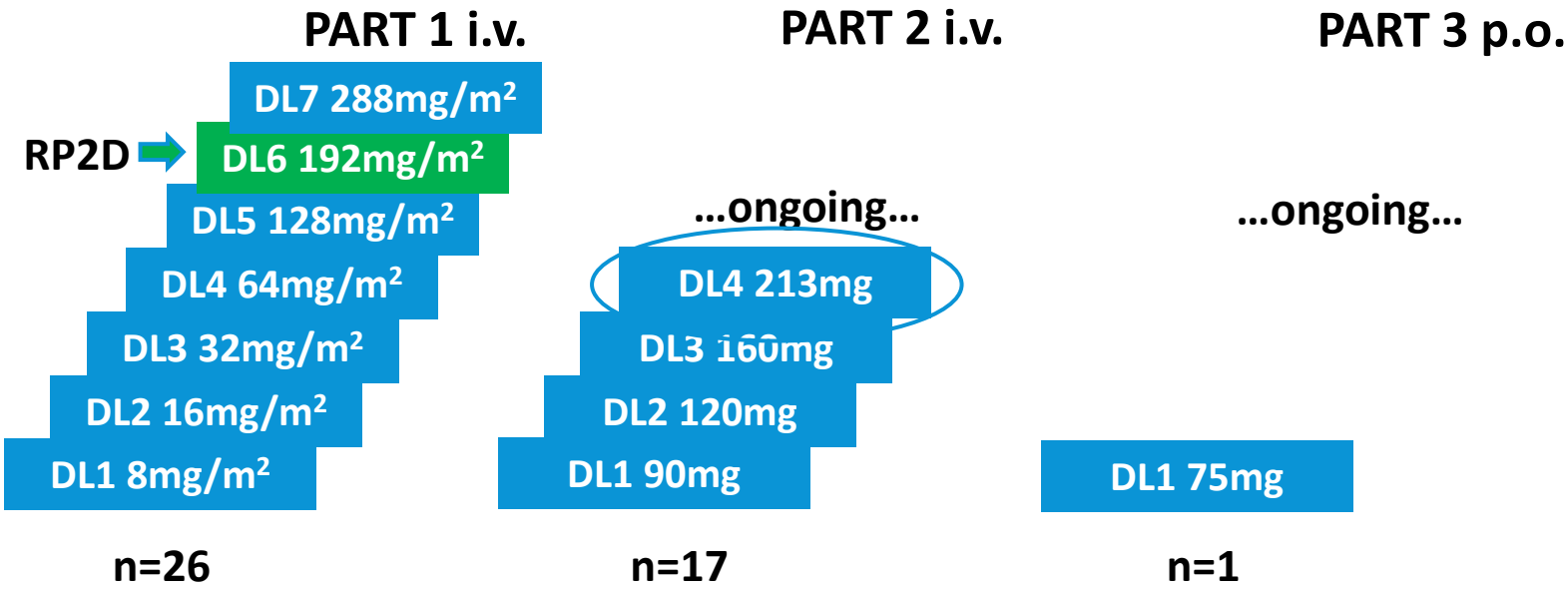
- **MCL1** > transcriptional CDKi, incl. **CYC065**

(one of ten most frequently overexpressed cancer genes)

Competitive race to develop drugs that suppress MCL1

CYC065 1st Rx to show durable MCL1 suppression in humans

CYC065-01 Phase 1 Escalation Schema



Source: Cyclacel data on file.

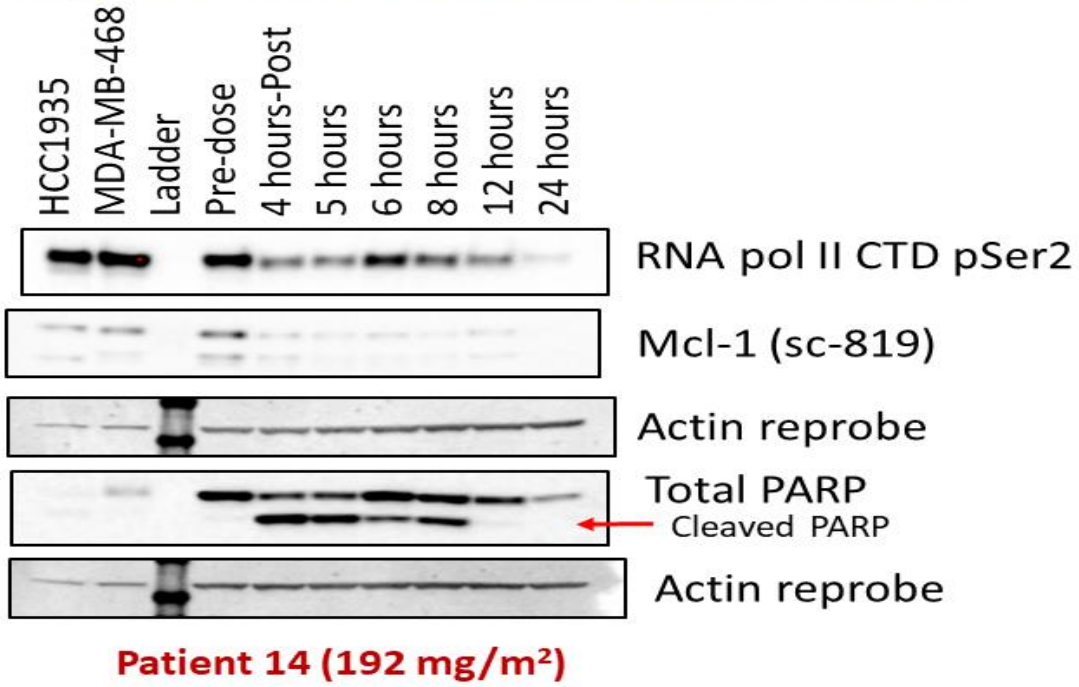
CYC065-01 First in Human Ph 1 part 1 (n=26)

- Heavily pretreated patients with advanced solid tumors
- Durable **MCL1 suppression** after single dose observed at RP2D
- Anticancer activity in 6/13 patients

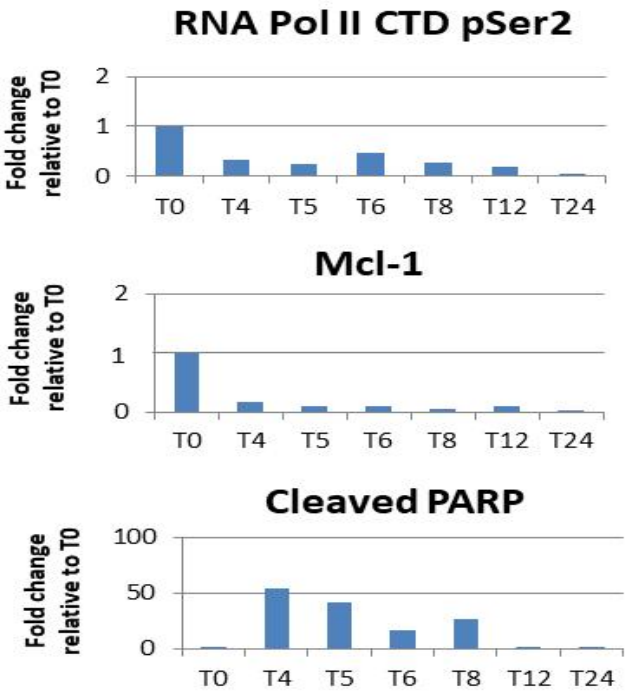
Source: Cyclacel data on file.

CYC065-01 Phase 1 part 1 Proof of Mechanism

Target inhibition detectable at 24 hours



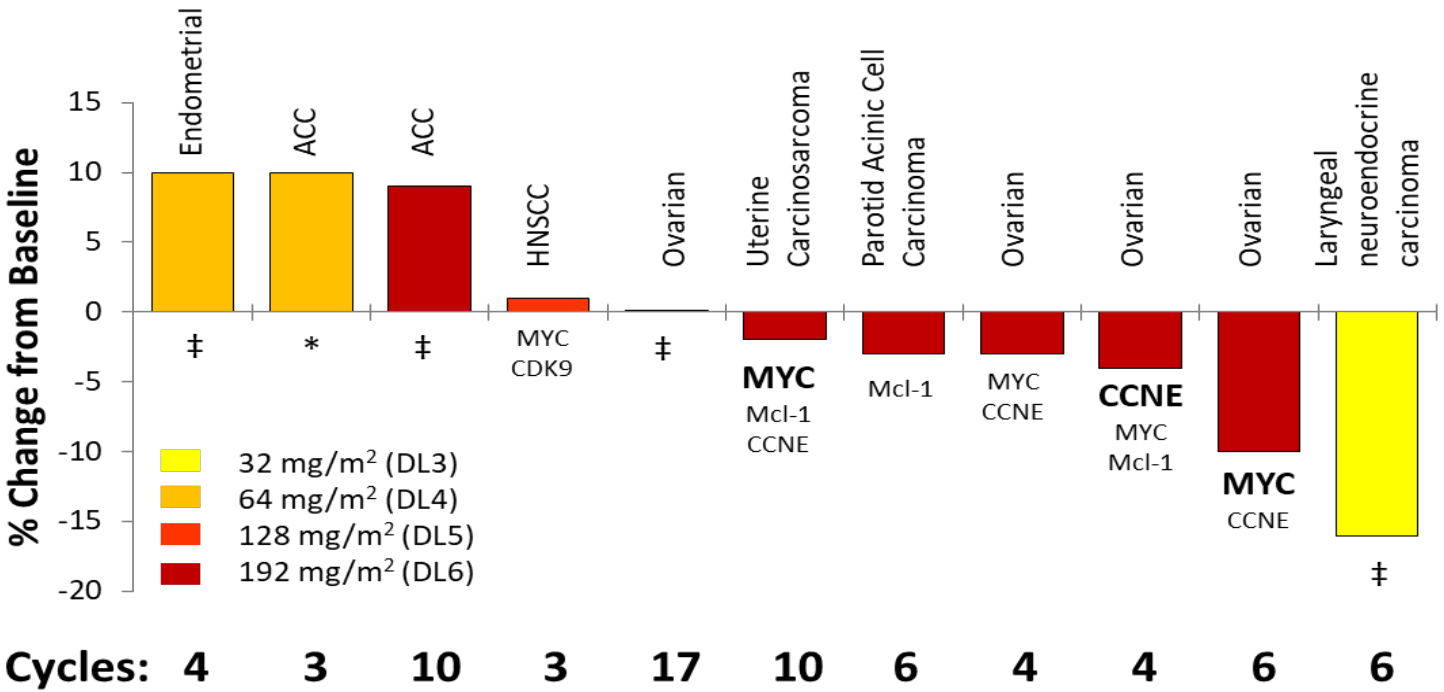
Patient 14 (192 mg/m²)



Source: Do, Khanh T., et al, AACR Annual Meeting 2018.

American Association for Cancer Research

CYC065-01 Phase 1 part 1 Activity



*‡ no information; * complex deletions/gains. High copy gains shown in bold.*

Source: Do, Khanh T., et al, AACR Annual Meeting 2018.

Summary:

- **20/26 patients evaluable for response per RECIST 1.1**
- **11/20 patients achieved stable disease (SD)**
- **6/11 patients achieved SD for 4+ cycles**

American Association for Cancer Research

CYC065-01 Phase 1 part 2 (n=17 ongoing)

PART 2 i.v.

…ongoing…

- Endometrial cancer patient with MCL1 amplification
- Tumor shrinkage of 15% after 2 cycles
- Treatment ongoing

Source: Cyclacel data on file.

CYC065-01 Phase 1 part 3 p.o. (n=1 ongoing)

- High solubility & permeability
- Oral route beneficial (esp. when combining with other oral agents)
- Aim to establish bioavailability
- Analyze PK/PD of oral form

Source: Cyclacel data on file.

Indication Rationale: 2L CLL (post BTKi)

- 1L US incidence 21,000; nearly all survivors receive 2L
- Venetoclax does not ↓ MCL1
- “Double-Hit” strategy to suppress BCL2 + MCL1
- Preclinical evidence of synergy for venetoclax + CYC065*
- CYC065 1st CDKi to durably suppress ↓ MCL1 in patients
- **CYC065 + venetoclax Phase 1b study ongoing**

*Source: * Chen et al AACR 2018 Abs 5095; Cyclacel data on file.*

CYC065-02 Phase 1 CLL (n=2, ongoing)

PART 1 i.v.

…ongoing…

DL1 64mg/m²

- 2nd pat.; ibrutinib failure; lymphoadenopathy
- PR on venetoclax ramp-up
- Lymph node shrinkage after 5 cycles of 065+venetoclax
- Treatment ongoing

Source: Cyclacel data on file.

CYC065-03 Phase 1 AML/MDS (n=4, ongoing)

PART 1 i.v.

…ongoing…

DL2 85mg/m²

DL1 64mg/m²

- MCL1 plays prominent role in AML
- Aim to suppress apoptotic pathways
- Combination with venetoclax post ramp-up

Source: Cyclacel data on file.

CYC065 Future Clinical Indications

1. ***Cancers addicted to cyclin E***
   - Breast
   - Ovarian
   - Uterine serous carcinoma (USC), etc.

2. ***Cancers addicted to MYC***
   - Lymphomas
   - Neuroblastoma
   - Ovarian, etc.

CDK Inhibitor Landscape

CDK4/6 isoform

palbociclib (PFE), ribociclib (NVS), abemaciclib (LLY) Approved in combination with hormone therapies for ER +ve Her2 -ve advanced or metastatic BC

trilaciclib (GTHX) Ph3

CDK2/9 transcriptional isoforms

CYC065 (CDK2/9, CYCC) Ph1 data

BAY1251152; *atuveciclib BAY1143572* **(CDK9, BAY)** Ph1 data

AZD4573 (CDK9, AZN) Ph1 ongoing

Other (pan CDK or selective):

flavopiridol/alvocidib (pan CDK, SUM) Ph2

dinaciclib (pan CDK, MRK) Ph3 terminated

voruciclib (CDK4/6/9, MEIP) Ph1 data

SY1365 (CDK7, SYRS) Ph1 data

MCL1 inhibitors: S64315 (Ph1b ven combo AML); AMG176 i.v./AMG397 oral (FiH); AZD5991 (FiH).

Source: Cyclacel data on file.

DNA Damage Response

Exploiting DDR to Overcome Cancer DNA Repair & Evasion

Homologous recombination deficient (HRD), incl. BRCA mutant, cancers have an Achilles heel:

- Synthetic lethality: accumulation of SSBs converted to DSBs; cannot repair DNA by HR (i.e. inhibition of PARP enzymes)
- Approved indications: breast, ovarian, pancreatic
- Future: prostate, hematological malignancies
- Significant unmet medical need remains

** SSB=single strand breaks; DSB=double strand breaks.*

Sapacitabine Oral Capsules

- Metabolizes into CNDAC; induces SSBs via β-elimination reaction; converted into DSBs that cannot be repaired by HR
- Multi-year treatment achieved in solid and blood cancers
- Durable CR, PR, SD in patients with BRCA mutant breast, ovarian and pancreatic cancers
- CR, CRp, PR and major HI in AML or MDS R/R to SoC

Sapacitabine in AML (SEAMLESS Phase 3 data)

- ✓ Increase in median OS (primary endpoint) did not reach stat. sig.
- ✓ Doubling of CR rate (secondary endpoint)
- ✓ Improved median OS in large (2/3 of study) prospectively defined subgroup based on WBC level
- ✓ National regulatory consultations in various EU countries

Source: Cyclacel press releases and data on file.

CYC682-11 sapa+venetoclax (n=3 ongoing)

- AML SoC: HMA (decitabine or aza)+ venetoclax
- HMA administered by i.v. or s.c. route
- Hypothesis generating SEAMLESS data
- Convenience of oral regimen to elderly patients
- Enrolling in AML or MDS to SoC

Source: Cyclacel data on file.

MD Anderson-Cyclacel Alliance

- Up to 170 patients with single agent or combinations of:

  CYC065, CYC140, sapacitabine

- Risk Sharing: MD Anderson assumes patient costs; Cyclacel supplies drugs

  and limited support

- Payments to MD Anderson upon First Commercial Sale in indications studied

Development Pipeline

CYC- Rx Candidate	Phase 1	Phase 2	Phase 3	MoA / Rights
065 i.v.	065-01 parts 1/2 solid tumors			CDK2/9; W/W
065 oral	065-01 part 3 solid tumors			CDK2/9; W/W
065 i.v.	065-02 + venetoclax R/R CLL [M]			CDK2/9; W/W
065 i.v.	065-03 + venetoclax R/R AML/MDS [M]			CDK2/9; W/W
sapacitabine oral	682-11 sapacitabine + venetoclax all oral R/R AML/MDS [M]			W/W exc. Japan
sapacitabine oral	682-12 SEAMLESS sapacitabine alternating i.v. decitabine 1L AML >70 y.o. *(EU scientific advice – submissibility)*			W/W exc. Japan
sapacitabine oral	IST sapacitabine + olaparib all oral BRCA mutant breast cancer			W/W exc. Japan
140 i.v.	140-01 part 1 R/R AML/MDS [M]			PLK1; W/W

[M] *MD Anderson alliance programs. W/W = worldwide.*

Financial Position & Capitalization

June 30, 2019 cash & cash equivalents: $15.2m [1]

Operating cash burn (annual; excludes non-cash items)

- ✓ 2016: ~ $10.1m [2]
- ✓ 2017: ~ $ 7.5m [2]
- ✓ 2018: ~ $ 6.7m [2]
- ▪ 2019: ~ $10.0m [3]

Fully diluted shares: ~27.1 million [1,4]

No debt

1. *10 Q*
2. *10 K*
3. *Company estimate*
4. *Common stock outstanding 17.2 million*

Key Milestones

- Report initial data from CYC065+venetoclax Phase 1 in R/R leukemias
- Report initial data from sapacitabine+venetoclax Phase 1 in R/R AML or MDS
- Report initial data from CYC140 Phase 1 First-in-Human study
- Report bioavailability from Phase 1 of oral CYC065
- Report updated CYC065 Phase 1 data in patients with advanced solid cancers
- Report data from sapacitabine-olaparib combination Phase 1b/2 IST in BRCA mutant metastatic breast cancer patients when reported by investigators
- Determine regulatory pathway/submissibility of sapacitabine in elderly AML

Investment Thesis

- Clinical stage, state-of the-art oncology programs
- Targeting molecularly-defined patient populations
- Overcome cancer cell resistance & DNA repair
- CDK inhibitors: validated drug class
- Competitively positioned
- Significant market opportunities

THANK YOU

Cyclacel Pharmaceuticals, Inc.

200 Connell Drive #1500
Berkeley Heights, NJ 07922
+1 (908) 517 7330

Contact: ir@cyclacel.com