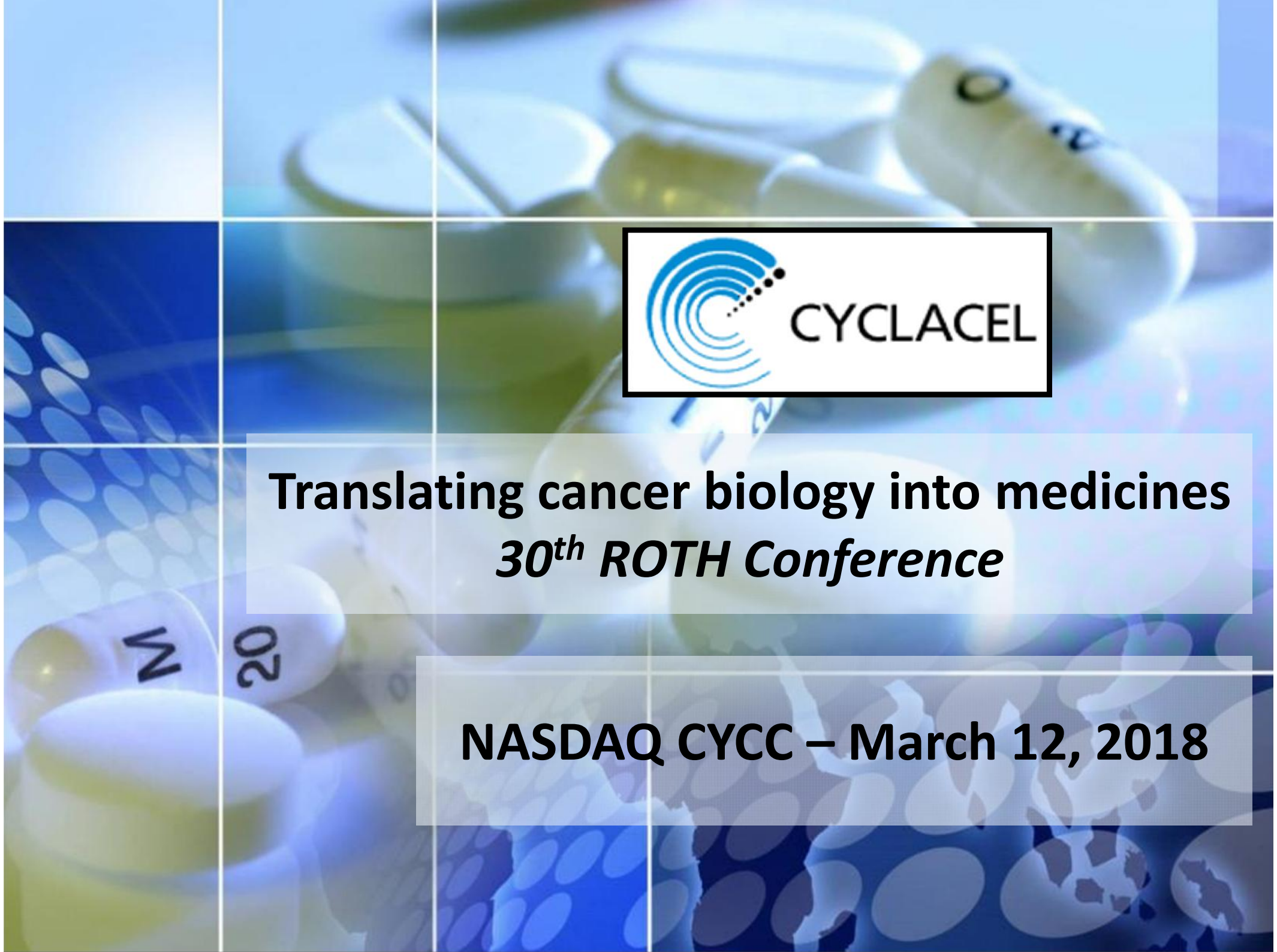


Translating cancer biology into medicines

30th ROTH Conference

NASDAQ CYCC – March 12, 2018

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.

Spending on Cancer Medicines

$107 bn in 2015 (+12% YoY). ***Est. ~$150 bn in 2020.***

Price hikes, ↑ patient #, **longer duration of therapy**

Avg. annual patient out-of-pocket: **$7k iv, $3k oral Rx**

Major threat to this colossal investment:

RESISTANCE TO CANCER Rx

** Source: Aitken M, Kleinrock, M, IMS Institute for Healthcare Informatics, June 2, 2016.*

Precision Medicine in Oncology

Single Rx targeting mutations: validated approach

↑ response but few cures/long stable disease

EVOLUTION OF RESISTANCE OR

ADDICTION TO CANCER GENES

- Strategy: combine approved Rx that is no longer working with resistance-modifying Rx or
- Rx that breaks addiction

Cyclin Dependent Kinase Inhibitors (CDKi)

2001 Nobel Prize for Physiology & Medicine (*CDKs & cyclins*)

3 approved CDKi:

- IBRANCE® (palbociclib, PFE, approved 2015, ~$3.3bn 2017E)
- 2017: KISQALI® (ribociclib, NVS), VERZENIO® (abemaciclib, LLY)
- CDK4/6 inhibitors → senescence → eventually resistance

*CDK2/9i strategy: overcome **resistance** by lowering killing threshold*

- CYC065 2nd Gen, highly potent, improved Rx profile (Ph 1)

Resistance Biomarkers

In many cancers resistance correlates with:

- ↑ ***pro-survival*** protein expression, such as Bcl-2, Bcl-XL, **Mcl-1**
- addiction to oncogenes, such as **MYC**, **cyclin E**

First Bcl-2 Rx: venetoclax (ABBV, CLL); does not ↓ Mcl-1

Competitive race to develop Rx that suppress Mcl-1

- *CYC065 1st CDK inhibitor Rx: durable ↓ Mcl-1 in patients*

** Source: Cyclacel data on file.*

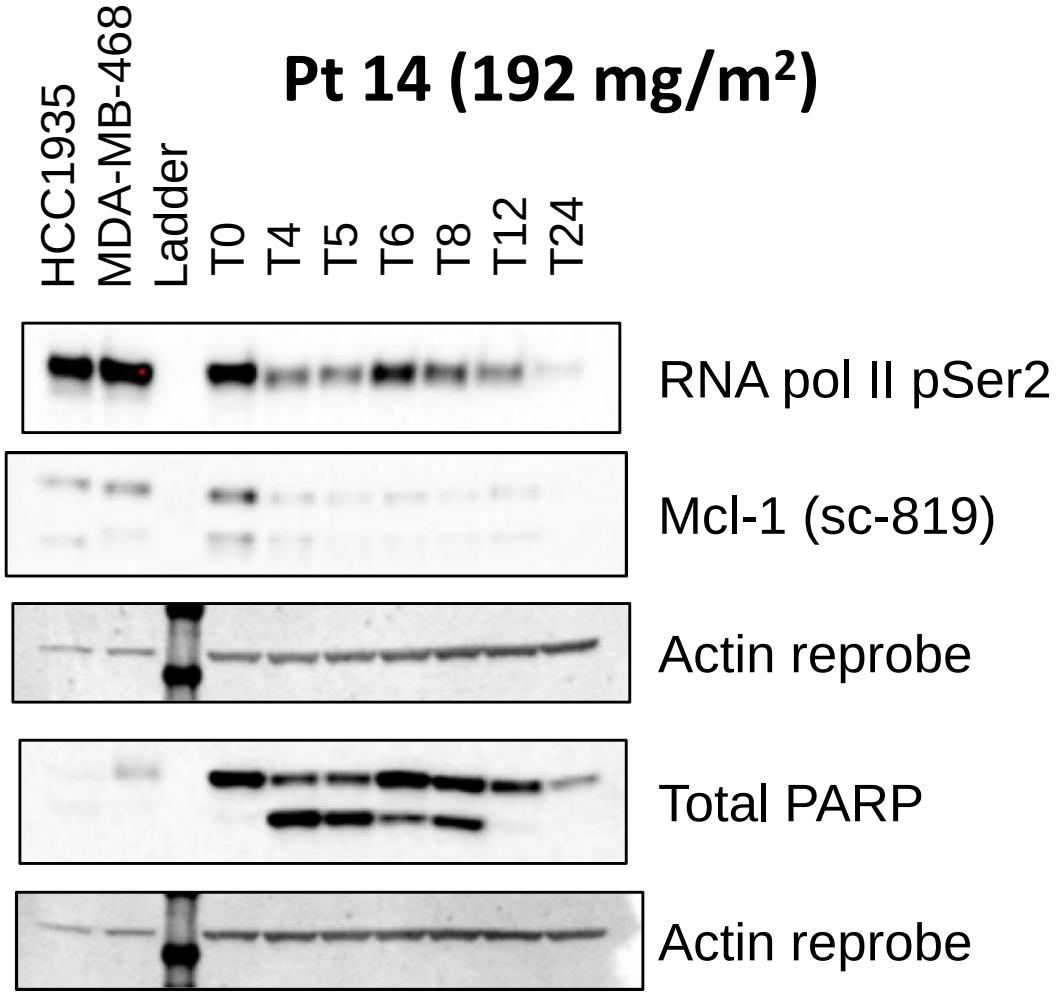
CYC065 First in Human Phase 1 Study (ongoing)

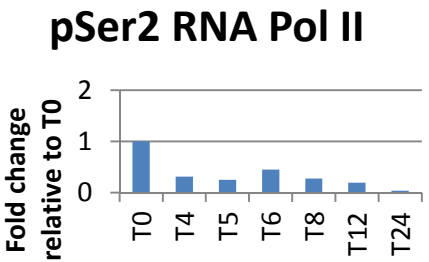
n=26 heavily pretreated patients with advanced solid tumors (13 in DL6 cohort RP2D)

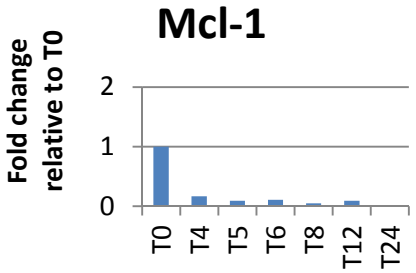
- Durable **Mcl-1 suppression** >24h after single dose in 11/13 evaluable DL6 patients
- Anticancer activity in 6/13 patients (5 at RP2D)

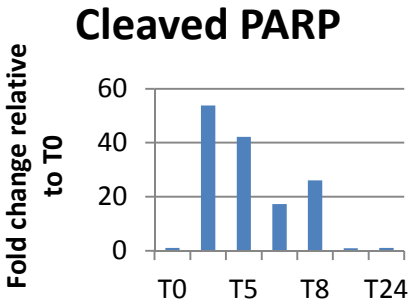
** Source: Cyclacel data on file.*

CYC065 First in Human Phase 1 Study (b)









Observations are representative for the cohort.

Source: Cyclacel data on file

CYC065: Clinical Development Priorities

Molecularly-defined patient populations

Hematological malignancies:

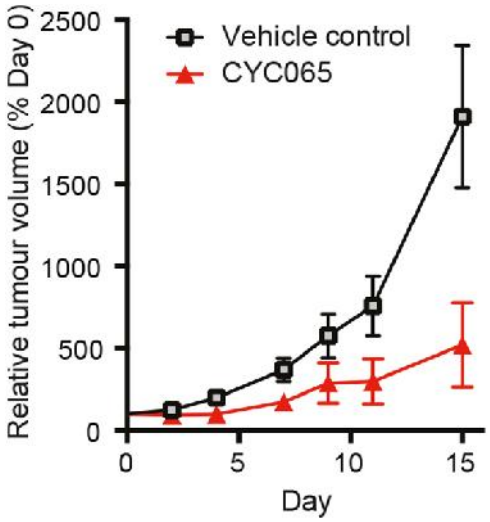
1. Combination with **venetoclax**, i.e. relapsed/ refractory CLL (incl. Mcl-1 ↑)

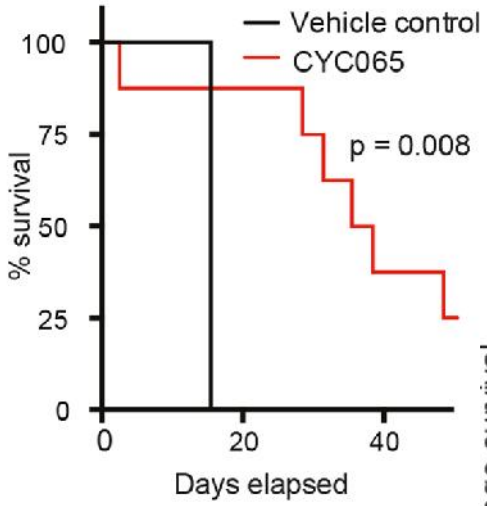
Solid tumors:

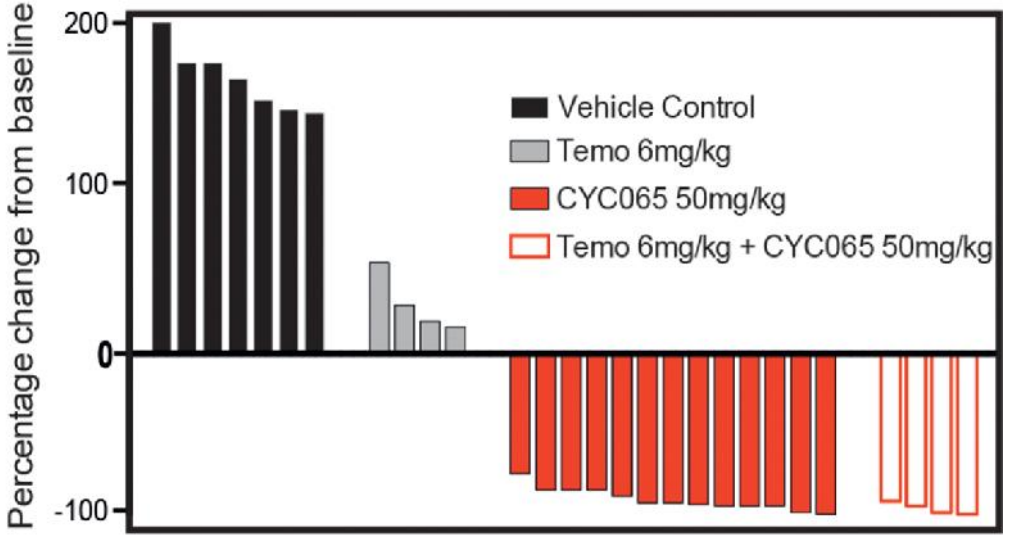
2. Selected Mcl-1 ↑ or MYC ↑ solid tumors, i.e. **neuroblastoma**, ovarian, etc.
3. Selected Cyclin E ↑ solid tumors, i.e. breast, uterine (USC)

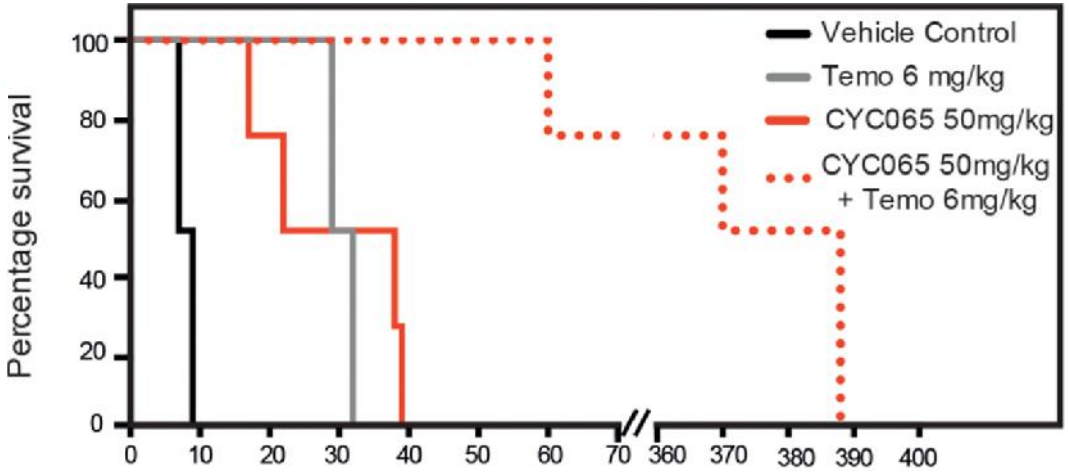
CYC065 in MYCN↑ NB Extended Survival; Tumor Regression

Kelly (MYCN amplified)









** Source: Poon et al. 4th Neuroblastoma Society Symposium 2015, 25-27 Nov, Newcastle. Cyclacel data on file. CYC065 – po, dq x 5/week x2.*

CDK Inhibitor Landscape

CDK4/6 isoform

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

trilaciclib (GTHX) Ph1/2

CDK2/9 transcriptional isoform

CYC065 (CYCC 2G) Ph1

seliciclib (CYCC 1G) Ph2

dinaciclib (pan CDK, MRK) Ph3

BAY1143572 (CDK9, BAY) Ph1

Other *(pan CDK or selective):*

SY1365 (CDK7, Syros);

voruciclib (CDK4/6/9, MEI Pharma)

** Source: Cyclacel data on file.*

DNA Damage Response (DDR):

Overcoming Cancer DNA Repair & Evasion

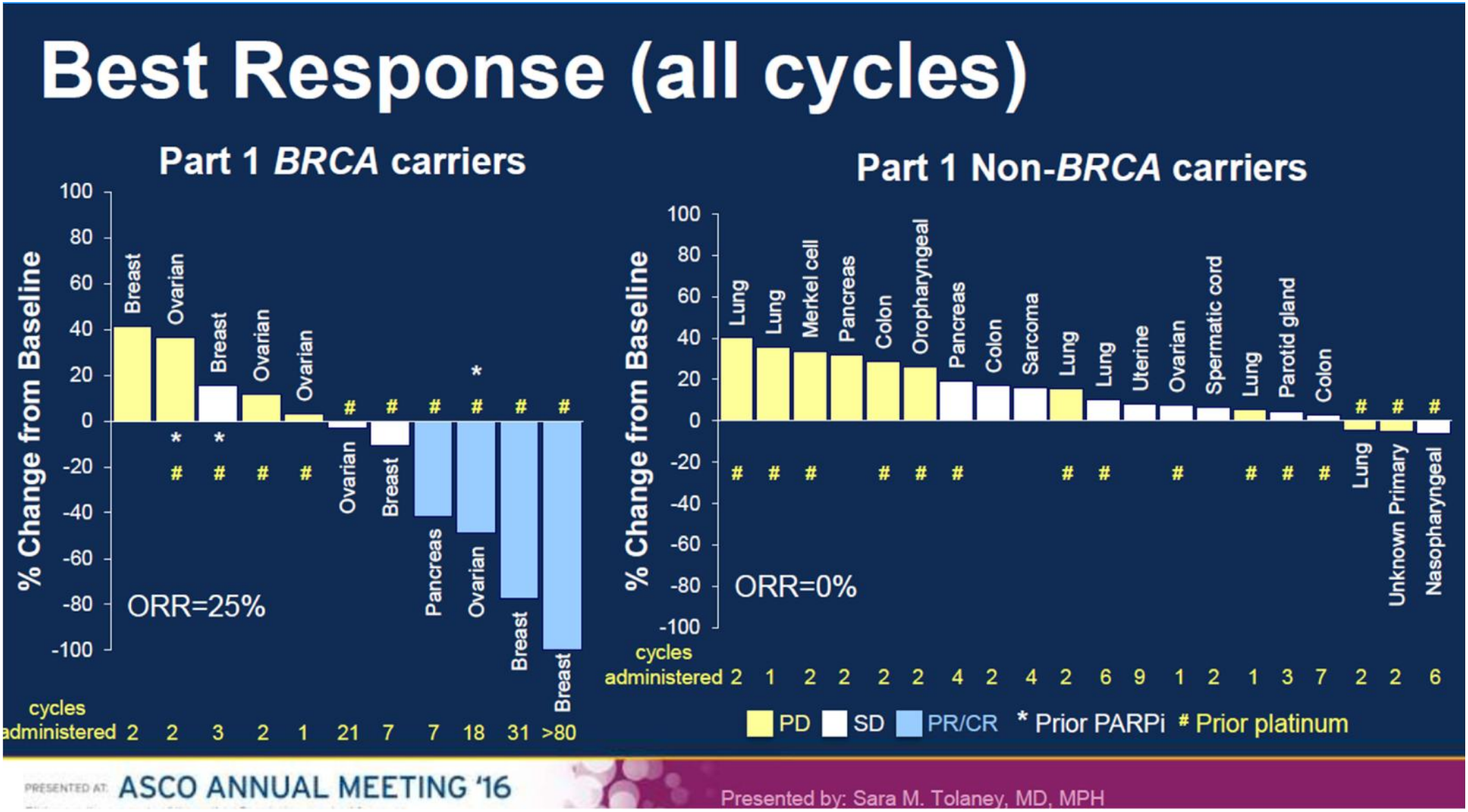
Cancer cells evade Rx; block DNA repair; ultimately become immortal

SoC HR deficient cancers (incl. BRCA): PARP inhibitors in ~ 50% of patients

CYCC DDR strategy: combine CDKi + sapacitabine

- Sapacitabine active in patients with BRCA +ve (HR def) cancers
- CDKi modulate DNA repair via HR, NHEJ; ↓ expression of HR DNA repair genes incl. BRCA; disrupts cyclin E amplification
- Encouraging clinical data: durable CR, PR, SD (n=76, ASCO 2016)

Sapacitabine & Seliciclib Phase 1 BRCA+ve Benefit*



** Source: Tolaney S et al, JCO 34, 2016 (suppl; abs. 2503).*

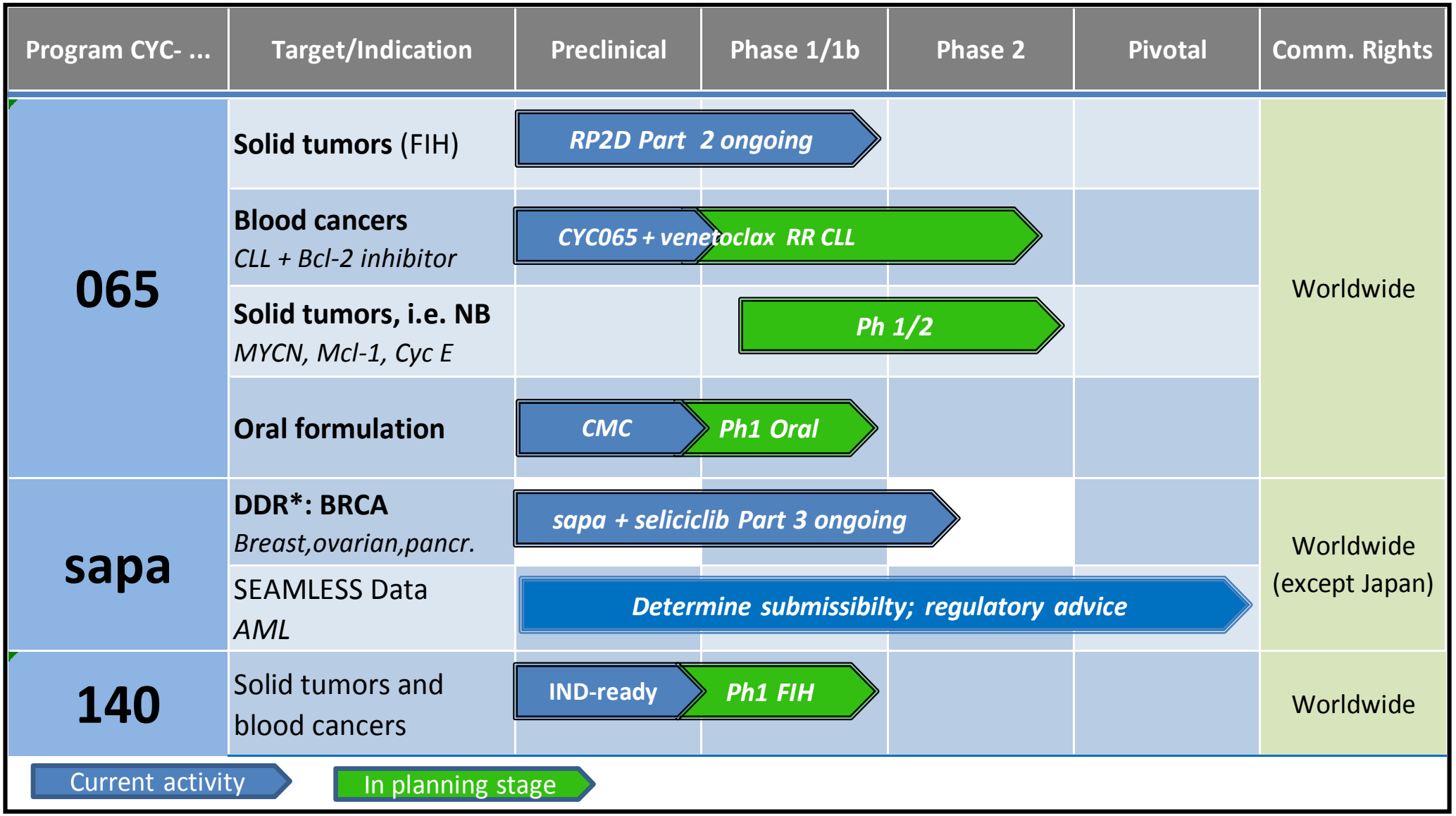
Sapacitabine in AML (SEAMLESS Ph 3 data)

Optionality from potential regulatory submission

- ✓ 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
- ✓ Oral presentation at ASH Annual Meeting 2017
- ▪ Determine optimal cut-off for WBC
- ▪ Determine submissibility to regulatory authorities
- ▪ Pre-submission End of Phase 3 meetings

Source: Cyclacel press releases and data on file.

Development Pipeline



Financial Position & Capitalization

Sept 30, 2017 cash & cash equivalents: $26.0m[1]

Current Operating cash burn (excludes non-cash items)

- ✓ 2014: ~ $18.7m annual [2]
- ✓ 2015: ~ $14.5m annual [2]
- ✓ 2016: ~ $10.1m annual [2]
- ▪ 2017: ~ $ 8.0m annual [3]

Fully diluted shares: ~ 20.0 million[1,4]

No debt

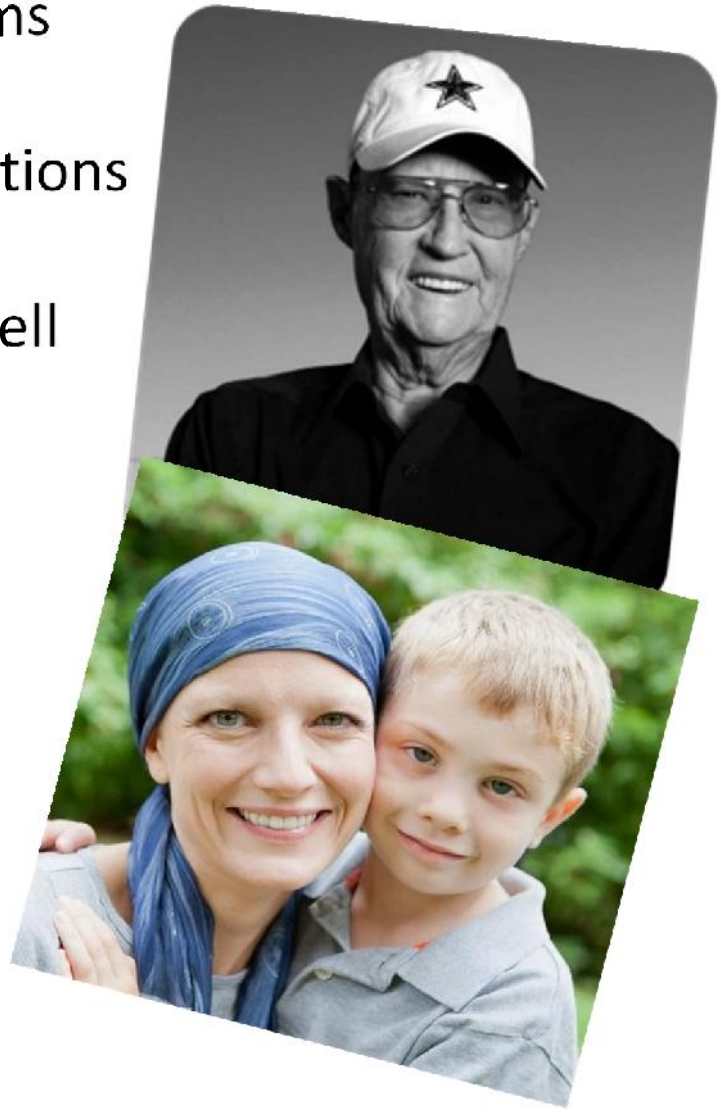
1. *10Q*
2. *10-K*
3. *Company estimate*
4. *Common stock outstanding: 11.9m*

Key Milestones

- Start CYC065 Ph 1b in RR CLL combo with venetoclax
- Start CYC065 Phase 1/2 in solid tumors, incl. NB
- CYC065 Phase 1 data solid tumors
- CYC065 oral formulation development
- Sapacitabine/seliciclib update BRCA +ve breast cancer
- CYC140 (PLKi) IND submission
- Determine submissibility of sapacitabine in eAML

Investment Thesis

- Clinical stage CDKi and DDR oncology programs
- Targeting molecularly-defined patient populations
- Treat difficult cancers and overcome cancer cell resistance & DNA repair
- CDK inhibitors: validated drug class
- Competitively positioned
- Significant market opportunities



THANK YOU

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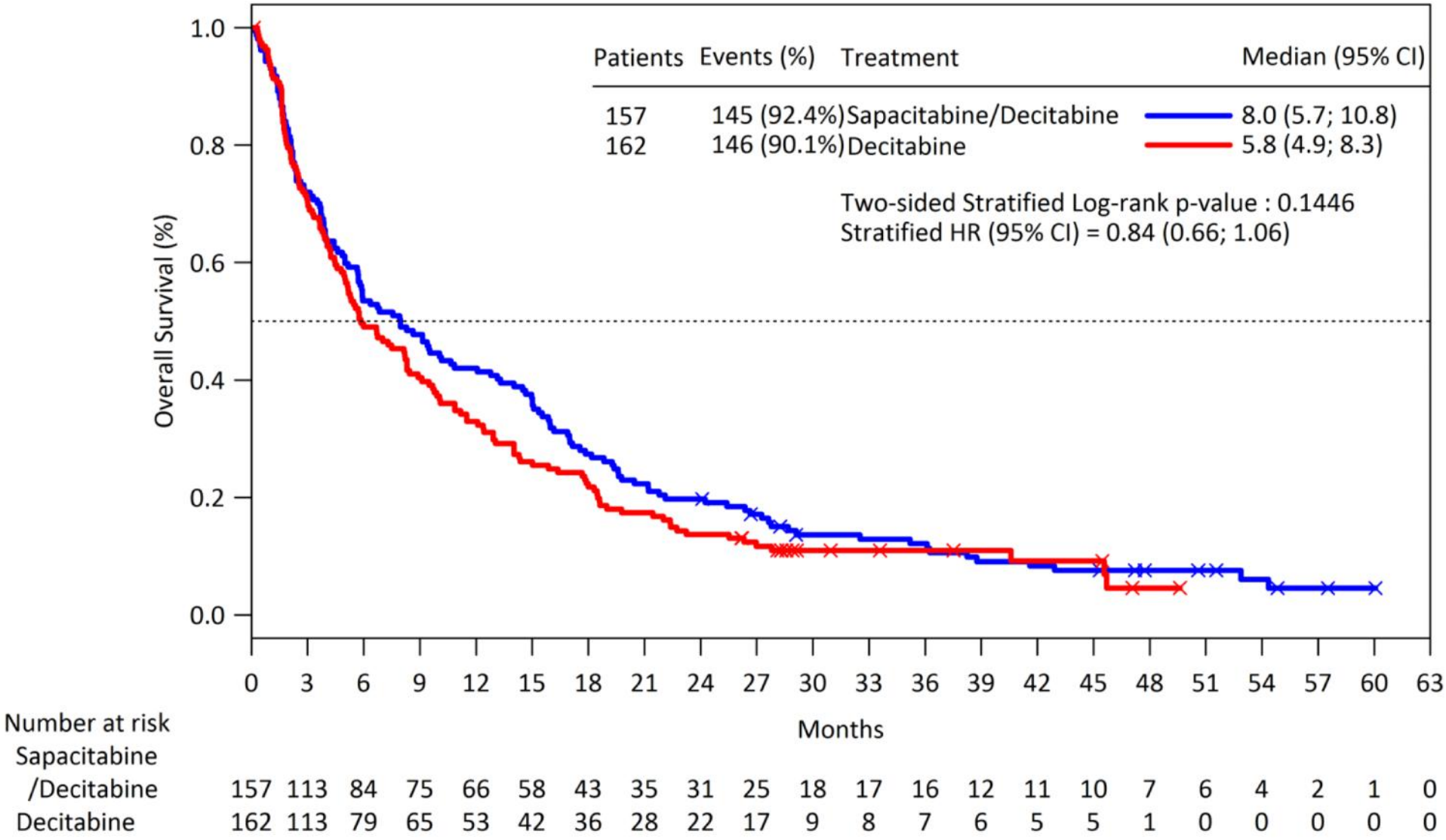
Survival - Baseline WBC <10,000



** Source: Kantarjian H, et al, American Society of Hematology Annual Meeting Dec. 2017, Abstract #891.*