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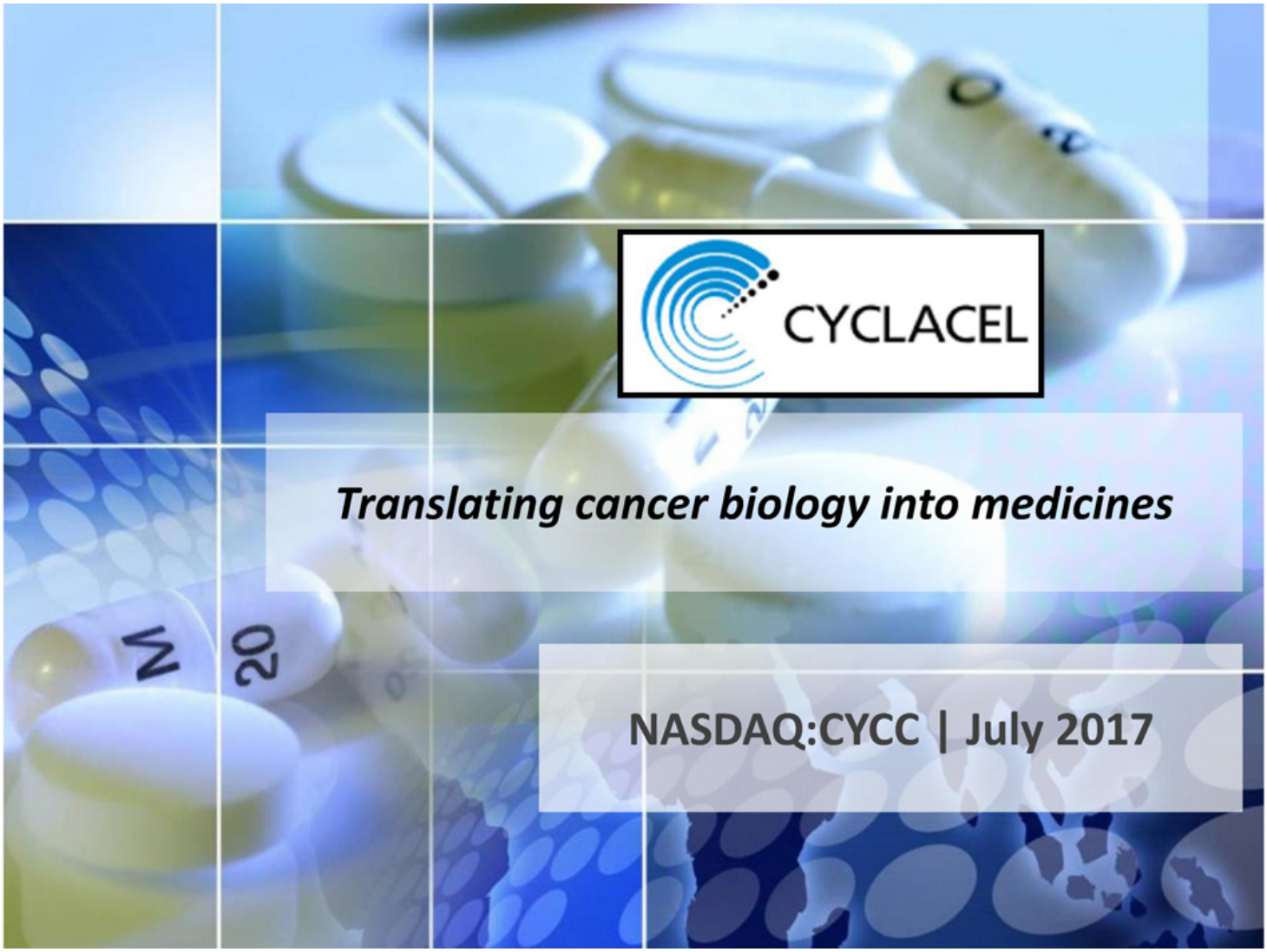

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Statement about Free Writing Prospectus

- This presentation highlights basic information about us and the offering. Because it is a summary that has been prepared solely for informational purposes, it does not contain all of the information that you should consider before investing in our company. Except as otherwise indicated, this presentation speaks only as of the date hereof.
- This presentation does not constitute an offer to sell, nor a solicitation of an offer to buy, any securities by any person in any jurisdiction in which it is unlawful for such person to make such an offering or solicitation.
- Neither the Securities and Exchange Commission (the "SEC") nor any other regulatory body has approved or disapproved of our securities or passed upon the accuracy or adequacy of this presentation. Any representation to the contrary is a criminal offense.
- This presentation includes industry and market data that we obtained from industry publications and journals, third-party studies and surveys, internal company studies and surveys, and other publicly available information. Industry publications and surveys generally state that the information contained therein has been obtained from sources believed to be reliable. Although we believe the industry and market data to be reliable as of the date of this presentation, this information could prove to be inaccurate. Industry and market data could be wrong because of the method by which sources obtained their data and because information cannot always be verified with complete certainty due to the limits on the availability and reliability of raw data, the voluntary nature of the data gathering process and other limitations and uncertainties. In addition, we do not know all of the assumptions that were used in preparing the forecasts from the sources relied upon or cited herein.
- We have filed a Registration Statement on Form S-1 with the SEC, including a preliminary prospectus dated May 26, 2017 (the "Preliminary Prospectus") and subsequent amendments S-1/A dated June 30, 2017 and July 7, 2017, with respect to the offering of our securities to which this communication relates. Before you invest, you should read the Preliminary Prospectus (including the risk factors described therein) and, when available, the final prospectus relating to the offering, and the other documents filed with the SEC and incorporated by reference into the Preliminary Prospectus, for more complete information about us and the offering. You may obtain these documents, including the Preliminary Prospectus, for free by visiting EDGAR on the SEC website at http://sec.gov.
- Alternatively, we or any underwriter participating in the offering will arrange to send you the prospectus if you request it by contacting Ladenburg Thalmann & Co. Inc., 277 Park Ave, 26th Floor, New York, NY 10172 or by email at prospectus@ladenburg.com.

Overview

- Mission: exploit cell cycle biology to disrupt cancer cell immortality
- Pioneer in Cyclin Dependent Kinase inhibitors
- Focus on genetically-defined patient populations
- Rationally designed single agents/combinations in liquid & solid cancers
- CDK inhibitor and DNA Damage Response clinical stage programs
- Experienced management, estimated capital through YE 2018

Cyclin Dependent Kinase inhibitors (CDKi)

2001 Nobel Prize for Physiology & Medicine culminating in approved Rx

Paradigm-shift in breast cancer: CDK4/6i-based combinations with AI

- IBRANCE® (palbociclib, PFE, approved 2015, ~$2.1bn 2016 sales)
- KISQALI® (ribociclib, NVS, approved 2017)

CYCC's CDK2/9i portfolio strategy: address key issue of **resistance**

- Seliciclib 1st Gen, signals of anticancer activity (Ph 2)
- CYC065 2nd Gen, more potent, better profile than seliciclib (Ph 1)

Pipeline

Program	Target/*Indication*	Preclinical	Phase 1/1b	Phase 2	Pivotal	Rights
CYC065 (CDKi)	Solid tumors (FIH)	*RP2D*				Worldwide
	Mcl-1 CLL	*CYC065 + venetoclax RR CLL*				
	Mcl-1 /Cyclin E solid tumors	*CYC065*	*Ovarian*			
	MYCN / Mcl-1 NB / MLL-r leukemias	*CYC065*	*IST*			
	DDR*: HRD+ve Breast, ovarian,pancreatic	*CDKi + sapacitabine*				
	DDR*: HRP+ve Breast, ovarian	*+ PARPi*				
Sapacitabine	AML	Data Analysis				
CYC140 (PLK1 inhibitor)	Solid & liquid cancers	IND-ready	*Ph1 FIH*			Worldwide
Seliciclib (CDKi)	Cushings disease, cystic fibrosis, RA	Investigator Sponsored Trials (IST)				

*DDR=DNA Damage Response

Current status | Future studies | *Data Dependent*

CDK inhibitor landscape

CDK4/6 isoform

- **palbociclib (NYSE:PFE); ribociclib (SWX:NOVN)**
- Approved in combination with letrozole for ER +ve Her2 -ve advanced or met BC
- **abemaciclib (NYSE:LLY)** Ph3
- **trilaciclib (NASDAQ:GTHX)** Ph1/2

CDK2/9 transcriptional isoform

- **CYC065 (CYCC 2Gen)** Ph1
- **seliciclib (CYCC 1Gen)** Ph2
- **dinaciclib (pan CDK, MRK)** Ph3
- **BAY1143572 (CDK9, BAY)** Ph1

** Source: Cyclacel data on file.*

CYC065 CDK2/9 inhibitor summary

Second generation, available by i.v. and oral route

Unique kinase selectivity

- CDK2 (cell cycle control)
- CDK9 (regulation of transcription & survival)

Addressing cancers:
- **dependent on Mcl-1 pro-survival protein**
- **or addicted to oncogenes**

Differentiation vs. CDK4/6 or pan-CDK inhibitors

- Selective pro-apoptotic, p53-independent MoA

Completed Ph 1, FIH study, i.v. in advanced patients with solid tumors

- Established safety, target engagement, RP2D

Robust IP position; exclusivity beyond 2030

CYC065 Genetically-Defined Patient Populations

1. **Mcl-1 or MYC dependent (↑) liquid & solid cancers**
   A. Venetoclax (Bcl-2i) treated CLL* with Mcl-1 ↑
   B. Selected Mcl-1 amplified solid tumors, i.e. ovarian
   C. Cancers addicted to *MYCN*, i.e. neuroblastoma
2. **Cyclin E amplified (↑) solid cancers**
   A. HGSOC *(BRCA mutually exclusive, PARPi not option)*
   B. Selected Cyclin E ↑ solid tumors, i.e. breast HER2+
   C. CDK4/6i resistant breast cancer

** Also MLL-r leukemias, lymphoma, and multiple myeloma.*

CYC065 First in Human Phase 1 Study

n=23 heavily pretreated patients with various advanced solid tumors

- Determined safety, DLT, PK in 7 DL, established RP2D
- Treated n=10 in total at DL6 cohort; DL7 MTD neutropenia
- Demonstrated target engagement and consistent **Mcl-1 suppression** over 24h after single dose in 7/9 DL6 patients
- Anticancer activity observed in patients with:

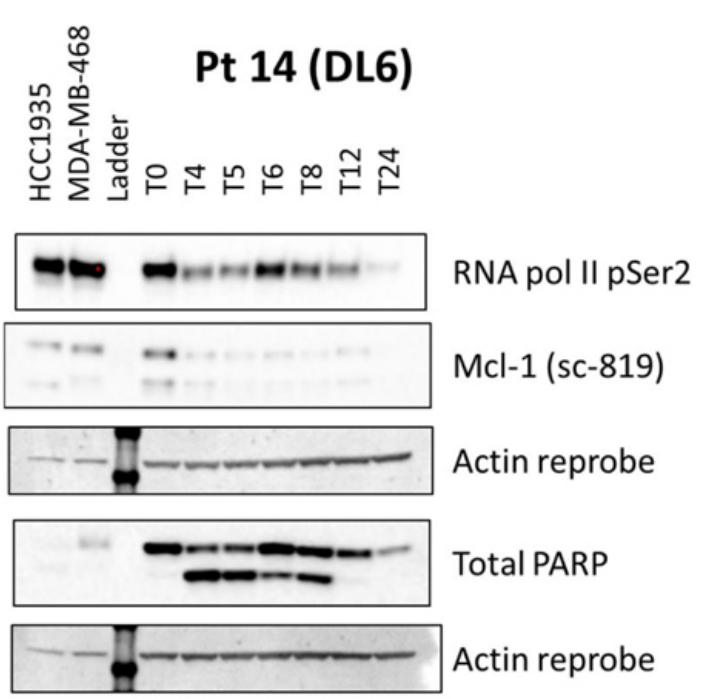
➢ **Mcl-1 ↑ (ovarian),**

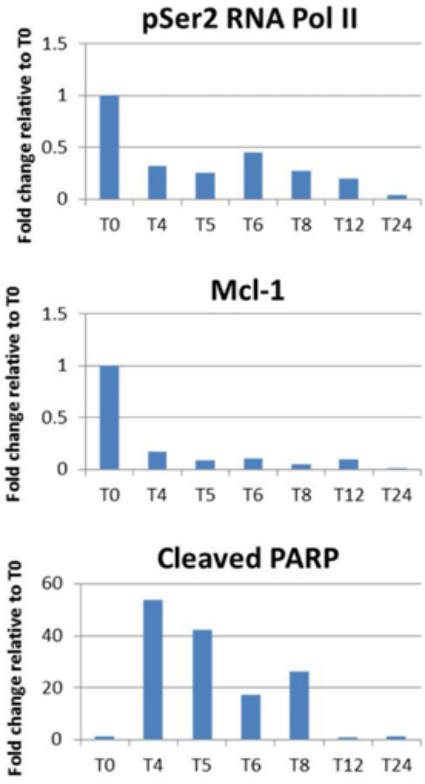
➢ **Myc ↑ (larynx) and**

➢ **cyclin E / Mcl-1 ↑ (ovarian) amplified tumors**

Similar CDK2/9 CYC065 CDK selectivity vs. seliciclib but 40x higher potency & improved pharmaceutical properties.

Phase 1 patient durable target inhibition





Source: Cyclacel data on file. Observations are representative for the cohort.

CYC065 mechanism of action

Inhibits CDK9-mediated oncogenic transcription
- Rapidly induces apoptosis; preferentially kills cancer cells over non-cancerous cells
- ↓ anti-apoptotic proteins (Mcl-1, XIAP) & oncogenes (MYC, cyclins)

→ Mcl-1, MYC, Cyclins

Inhibits CDK2 (cell cycle control)
- Targets cyclin E addicted, drug resistant tumors

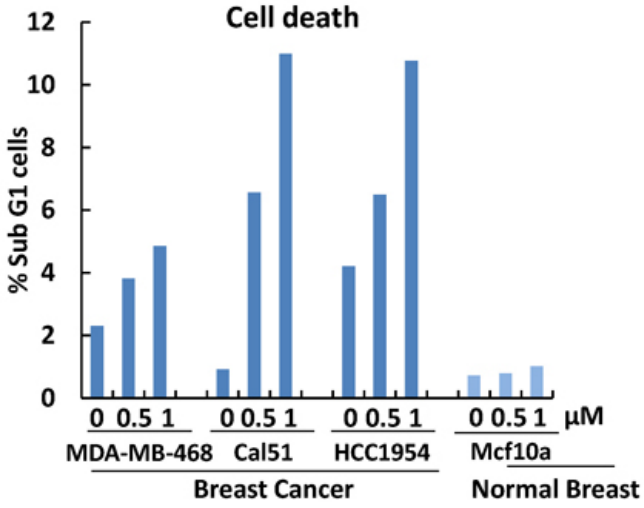
→ Cyclin E

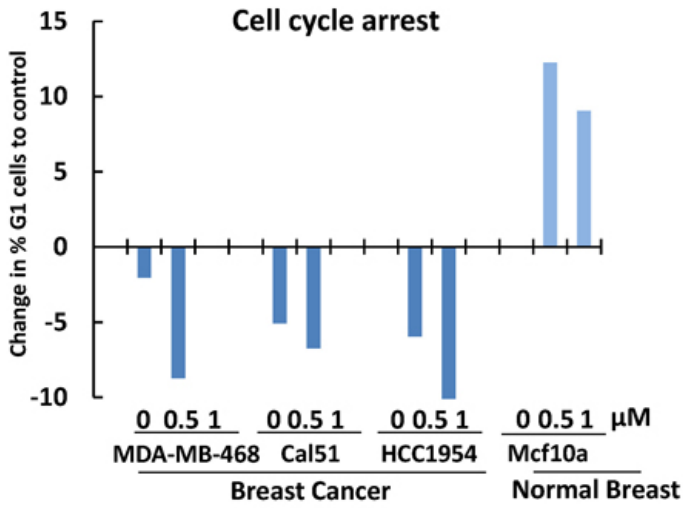
Potentiates effects of DNA damaging agents
- ↓ expression of HR DNA repair genes (BRCA1 and BRCA2)

→ BRCA1, BRCA2

Cyclacel data on file.

CYC065 response in cancer vs. normal cells

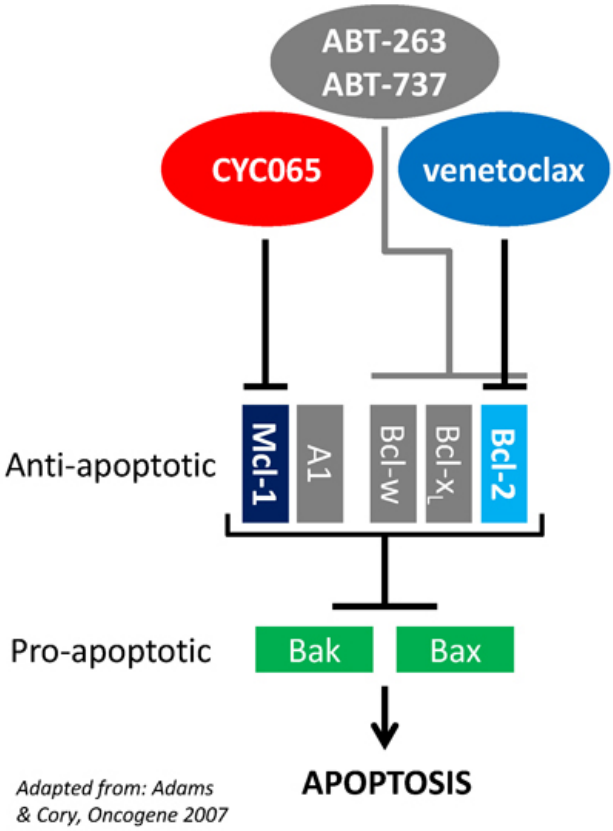




- CYC065-dependent apoptosis induction in cancer cells but not in non-cancer Mcf10a
- CYC065 caused G1 arrest in non-cancer Mcf10a cells
- Similar trend observed in other cancer and non-cancer cell lines

MacKay et al. SABCS 2015

CYC065-venetoclax rational combination CLL

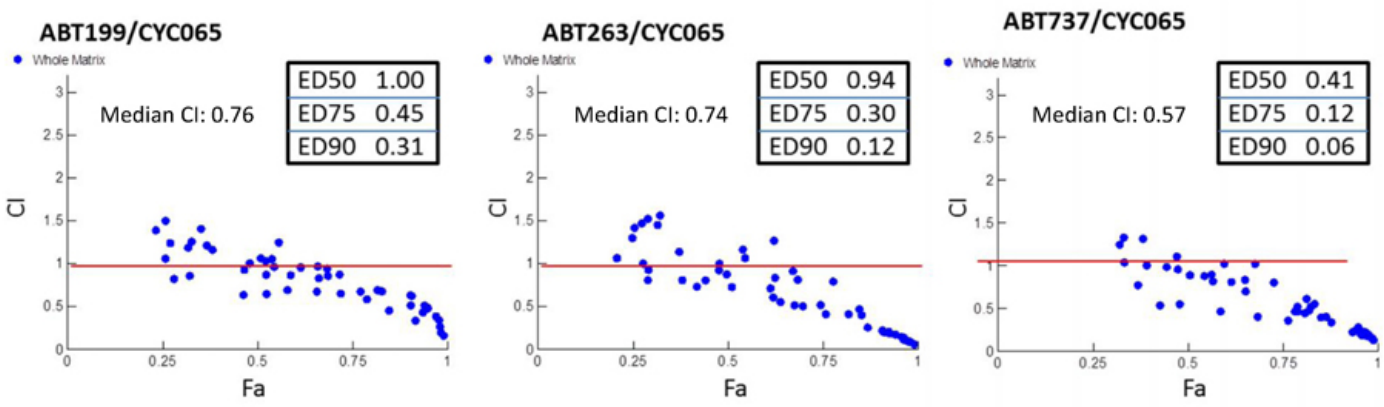


Adapted from: Adams & Cory, Oncogene 2007

- CLL unmet medical need
  - 30% all adult leukemias; 70% mortality rate; 70% aged > 65 years
- Venetoclax ORR: 71-79%; CR: ~20% (R/R CLL with 17p del)[1]
- Tumor microenvironment ↑ Mcl-1 diminishing venetoclax effect[2,3]
- Mcl-1 ↓ overcomes venetoclax resistance (stimulated CLL)[3]
- CYC065 ↓ Mcl-1 effectively & durably (>24h) in Phase 1 patients at well tolerated doses
- CYC065 and venetoclax synergistic in CLL (incl. 17p del), multiple leukemia & lymphoma cell line models[4,5]

1 Roberts et al. 2016. 2 Smith et al. 2007; 3 Oppermann et al. Blood 2016, 4 Frame et al. AACR 2014, 5 Zheleva et al. SOHO 2015.

CYC065 & venetoclax synergy in AML & ALL cells



Example Fa:CI plots & median CI values for THP-1 (AML) cell line. Combination Index (CI) values calculated using Chou & Talalay method. CI of 0.9 to 1.1 indicate additivity, below 0.9 synergy.

- CYC065 synergy with venetoclax (ABT-199) in AML & ALL cell lines
- CYC065 synergy with Bcl-2/Bcl-xL/Bcl-w inhibitors ABT-263 or ABT-737
- CYC065 synergies demonstrated in several acute leukemia cell lines (AML: THP-1, HEL; ALL: Jurkat, SEM)

Source: Frame et al, SOHO, 2014, Abs 209

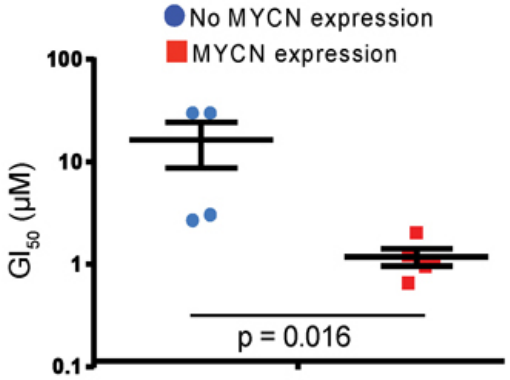
CYC065 transcriptional regulation of MYC

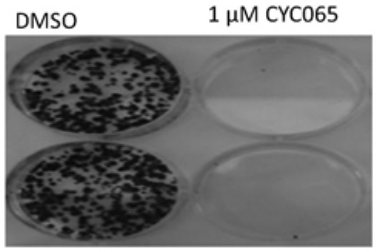
Very common genomic alteration in aggressive tumors

- Amplification of MYC family oncogenes: poor clinical outcome in neuroblastoma (NB), SCLC, breast, prostate
- No therapeutics against *MYCN* or MYC reported
- CDK9 involved in *MYCN* transcriptional regulation
- *MYCN* ↑ NB cells highly sensitive to CYC065
- CYC065 inhibits NB cell proliferation, induces apoptosis and ↓ *MYCN* protein
- CYC065 causes tumor regression & prolongs survival in *MYCN*-amplified NB models

Potent *in vitro* and *in vivo* anti-tumor activity suggest that CYC065 may have therapeutic potential in NB with *MYCN* oncogene amplification.

***MYCN* ↑ neuroblastoma highly sensitive**



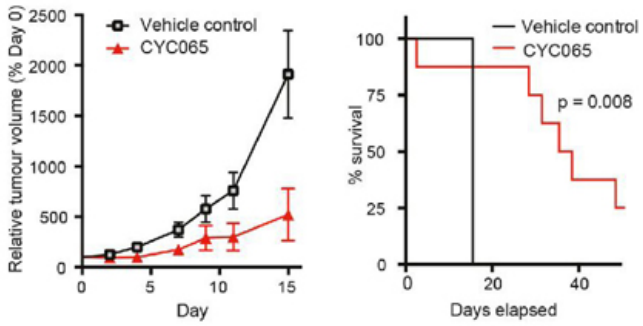


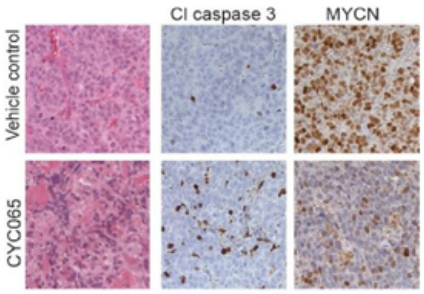
ICR The Institute of Cancer Research

Poon et al. 4th Neuroblastoma Society Symposium 2015. 8 h treatment. SRB colony formation assay after 72 h.

CYC065 in *MYCN*↑ NB: regression & ↑ survival

Kelly xenograft (MYCN amplified)



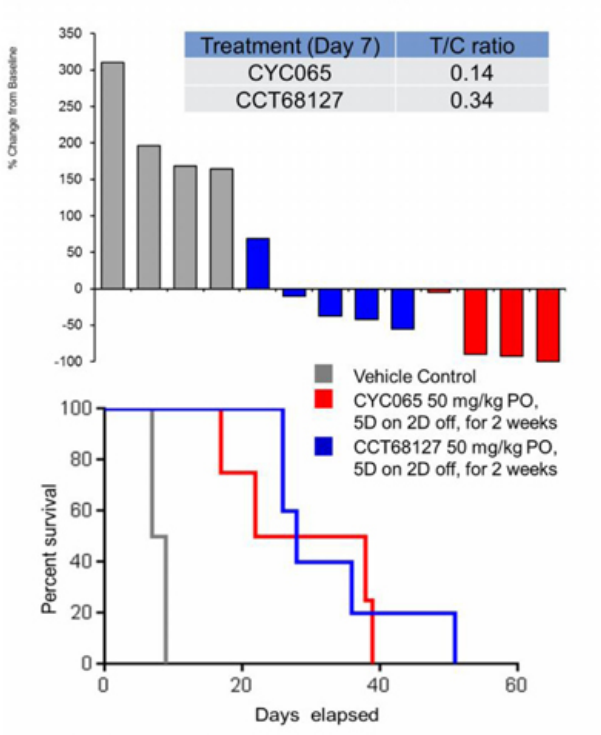


Treatment regimen:
CYC065 – po, dq x 5/week x2

Poon et al. 4th Neuroblastoma Society Symposium 2015, 25-27 Nov, Newcastle.

ICR The Institute of Cancer Research

Th-MYCN GEMM of NB

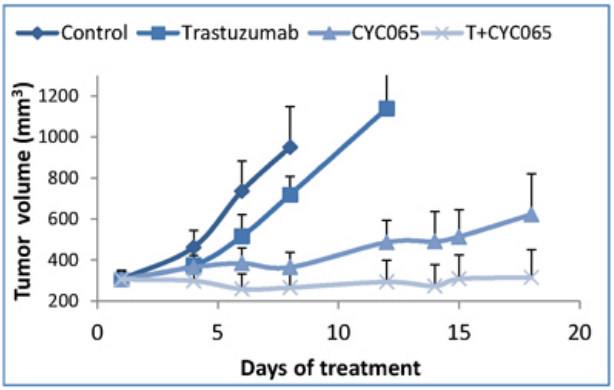


Treatment (Day 7)	T/C ratio
CYC065	0.14
CCT68127	0.34

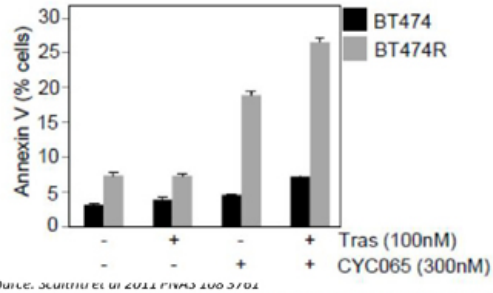
Cyclacel data on file.

Overcoming resistance in cyclin E amplified tumors

Trastuzumab resistant Her2+ breast cancer

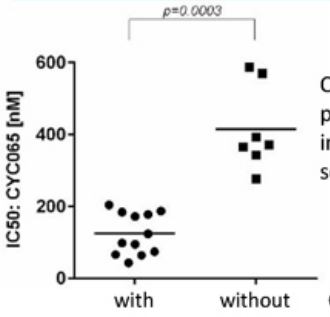


CYC065 alone or in combination with trastuzumab causes very effective *in vivo* tumor growth inhibition of BT474R trastuzumab resistant HER2⁺ BC cell line, and increased apoptosis *in vitro*.

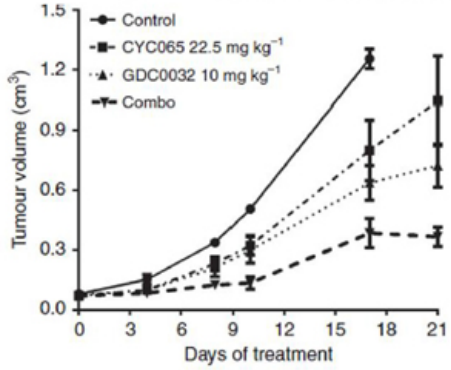


Source: Scaltriti et al 2011 PNAS 108 3761

Drug-resistant Uterine Serous Carcinoma



CCNE1 amplified USC primary cell lines show increased CYC065 sensitivity

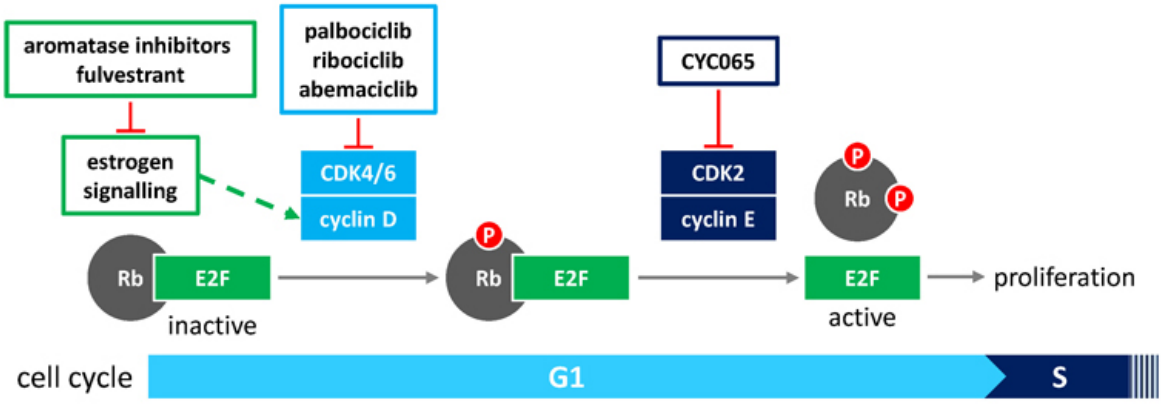


CYC065 active as a single agent and in combination ($p<0.03$) in ARK-1 (trastuzumab-resistant, PI3K mutant) xenograft model

Source: Cocco et al., AACR, 2015, Abs 3103; Cocco et al, BJC, 2016

Overcoming resistance to CDK4/6 inhibitors + AI

CDK4/6i + AI resistance



CDK4/6i+AI inhibit proliferation; but senescence → emergence of resistance, i.e. CDK2/cycE ↑

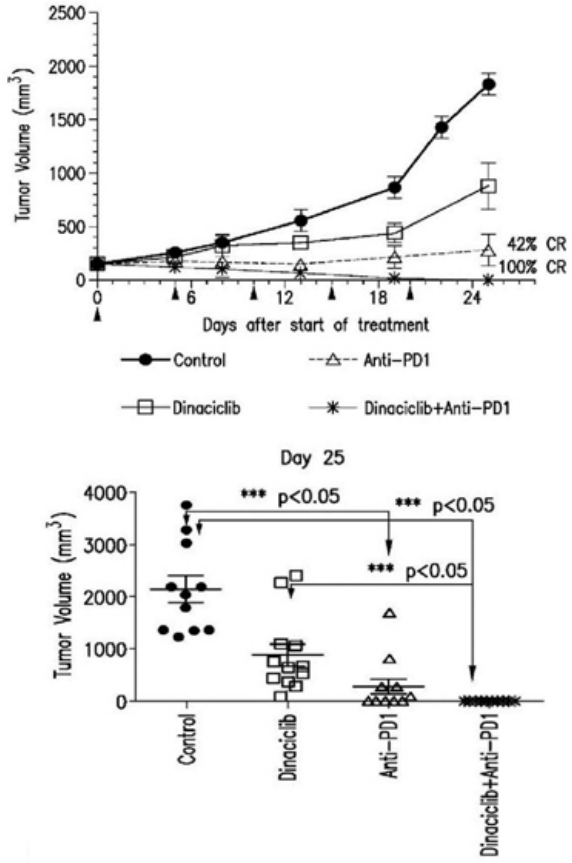
CDK2 activation or cyclin E amplification/overexpression → acquired/intrinsic resistance to CDK4/6i in breast and ovarian cancer and AML models*

Higher CCNE1 gene expression observed in BC resistant to neoadjuvant palbociclib + anastrozole*

Cyclin E mediated resistance to letrozole in HR+ breast cancer is reversed by seliciclib*

**Source: Herrera-Abreu et al. Cancer Res. 2016; Konecny & Slamon Clin. Cancer Res. 2011; Taylor-Harding et al. Oncotarget 2015; Caldon et al. Mol. Cancer Ther. 2012 ; Wang et al. Blood 2007; Ma et al. Clin Cancer Res. 2017; Akli et al, Clin Cancer Res. 2010.*

IO strategy: modulate antitumor immune response



CDK2/9i synergy with IO agents

- Dinaciclib synergistic with anti-PD-1 in mouse syngeneic tumor models
- Dinaciclib induces immunogenic cell death (ICD) with evidence of ↑ T cell infiltration and dendritic cell activation in tumors[1]
- MC38 mouse colorectal carcinoma in C57BL/6 mice, 12 per group, dinaciclib 40 mg/kg, anti-PD-1 5 mg/kg each dosed once daily for 25 days[2]
- Phase 1 open-label study evaluating safety and efficacy of pembrolizumab + dinaciclib in relapsed or refractory CLL, MM, DLBCL[3]

(1) Hossain et al., Cancer Res, 2015 (2) US20160193334A (3) NCT02684617

CYC065 CDK2/9i investment highlights

- CDKis: validated drug class
- Targeting genetically-defined patient populations
- Significant market potential
- Potential to treat difficult cancers and overcome cancer cell resistance
- Single agent and combination opportunity

DNA Damage Response (DDR) Clinical Program

Modulation of DNA Damage Response (DDR)

***Cyclacel's CDK** inhibitor-based DDR strategies:*

- Modulate DNA repair via HR, NHEJ, etc. pathways
- ↓ expression of HR DNA repair genes, i.e. BRCA1 / BRCA2
- ↓ anti-apoptotic survival signalling , i.e. Mcl-1

***Cyclacel's oral sapacitabine** may work best in HR-deficient tumors:*

Clinical utility observed in combination with CDK inhibitor; future options

- Combinations with SoC, i.e. PARP inhibitors
- Single agent treatment in sensitive cancers

DDR sapacitabine/seliciclib in HR-repair deficient tumors*

- Oral combo of complementary mechanisms:

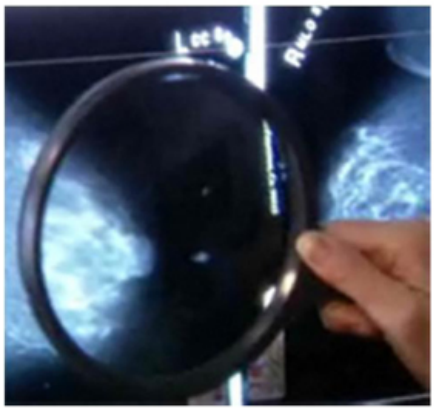
  sapacitabine's unique, MoA (DNA SSBs# & cell cycle arrest) combined with CDKi modulation

- Parts 1 & 2: durable clinical benefit (PRs & prolonged SD) in patients with BRCA +ve:

  breast, ovarian, pancreatic cancers

- Part 3 to start: revised schedule including BRCA +ve ovarian, pancreatic cancer patients

… Plan to substitute seliciclib with CYC065 …



** Source: Tolaney S et al, J Clin Oncol 34, 2016 (suppl; abstr 2503); Shapiro et al, AACR Proceedings, 2013, LB-202. HR=homologous recombination. # single-strand breaks*

ASCO American Society of Clinical Oncology

AACR American Association for Cancer Research

DDR: sapacitabine/seliciclib Ph 1 best responses*

CYCLACEL

RECIST Evaluable BRCA Carriers

Cancer	Best Response	Prior Treatment	Total cycles
Part 1	*(n=16)*		
Breast	CR	adriamycin, cyclophosphamide, paclitaxel, cisplatin	>80
Breast	PR	adriamycin, cytoxan, paclitaxel, carboplatin	31
Ovary	SD	paclitaxel, carboplatin, gemcitabine	21
Ovary	PR	paclitaxel, carboplatin, gemcitabine, topotecan, iniparib	18
Breast	SD	tamoxifen, raloxifene, anastrozole, adriamycin, Cytoxan, paclitaxel, carboplatin, navelbine	7
Pancreas	PR	gemcitabine, 5-FU, oxaliplatin	7
Part 2	*(n=28)*		
Breast	PR	adriamycin, cytoxan, paclitaxel, capecitabine, irinotecan, ABT-888 (PARP inhibitor), MPDL3280A	>19
Ovary	SD	paclitaxel, carboplatin, doxil	22
Breast	SD	adriamycin, cytoxan, capecitabine, faslodex	12
Ovary	SD	paclitaxel, carboplatin, doxil, gemcitabine, topotecan, cytoxan, avastin	11
Ovary	SD	paclitaxel, carboplatin, doxil, olaparib (PARP inhibitor), cediranib	8
Ovary	SD	paclitaxel, carboplatin	4
Ovary	SD	paclitaxel, carboplatin, doxil, gemcitabine, alimta, cytoxan, avastin, olaparib (PARP inhibitor)	4
Pancreas	PR	gemcitabine, abraxane, docetaxel	4
Pancreas	SD	gemcitabine, cisplatin, abraxane, folfox, TH-302	4

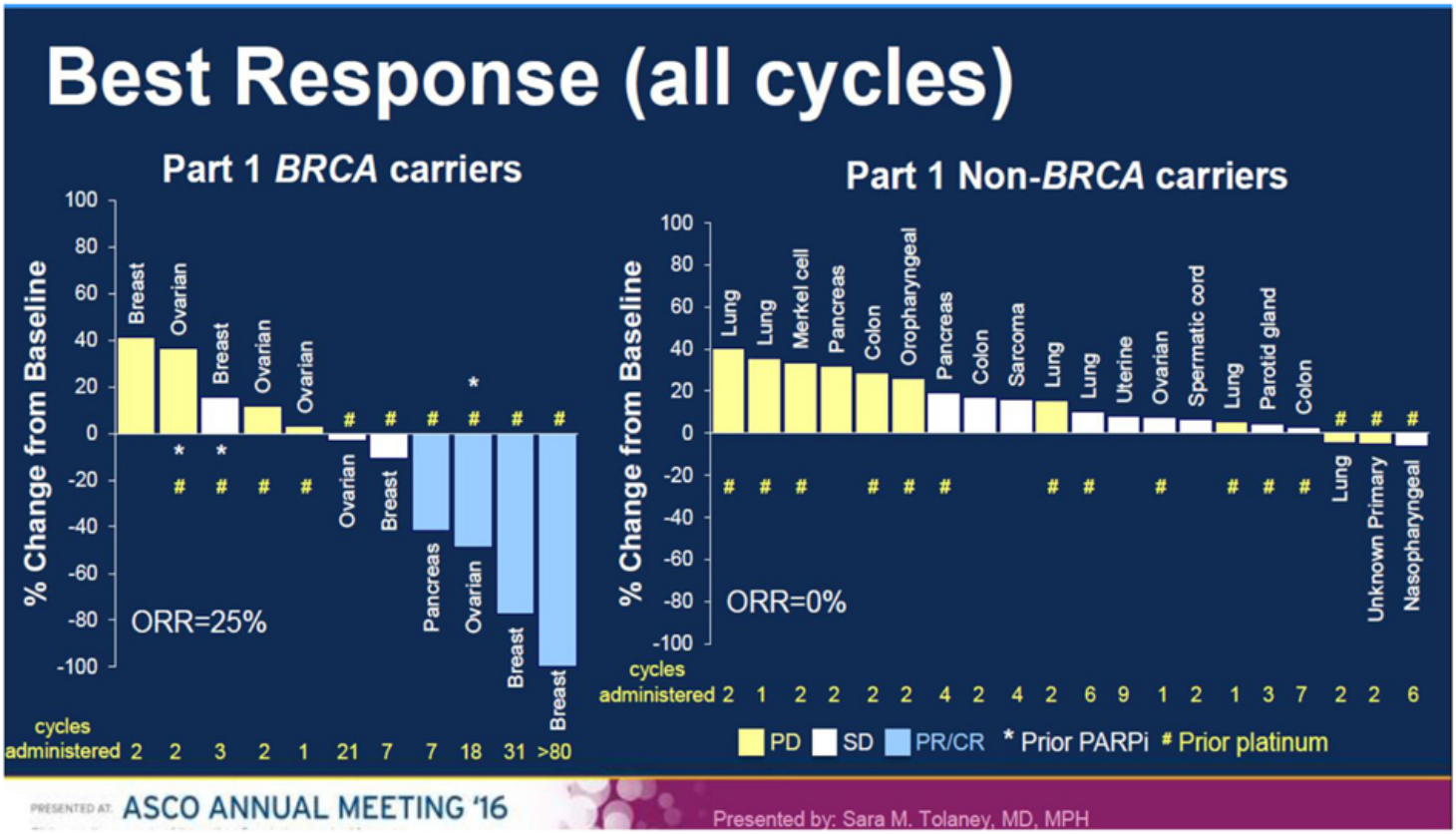
ED AT: ASCO ANNUAL MEETING '16

Presented by: Sara M. Tolaney, MD, MPH

** Source: Tolaney S et al, J Clin Oncol 34, 2016 (suppl; abstr 2503).*

ASCO American Society of Clinical Oncology

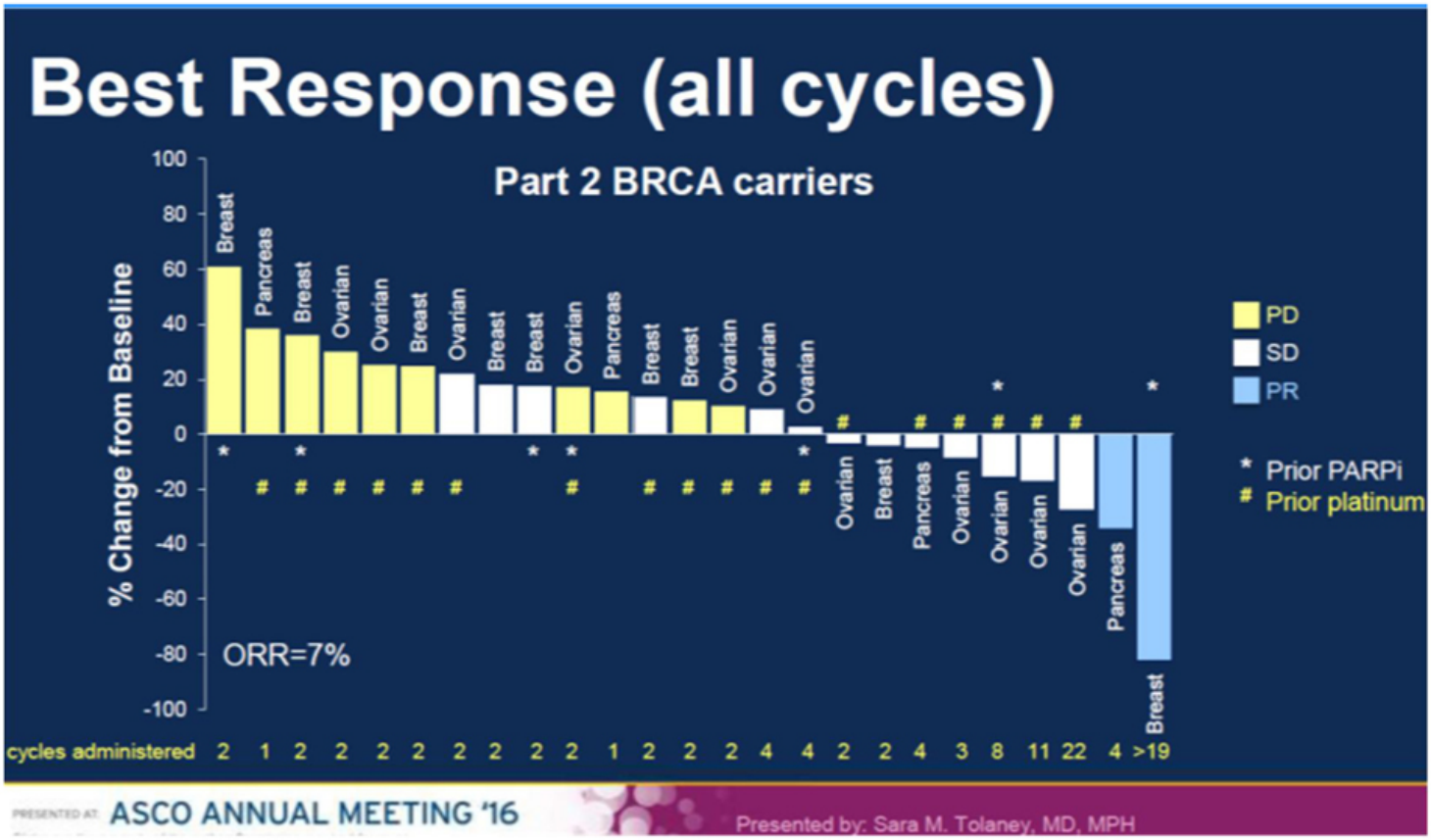
Sapacitabine/seliciclib Phase 1 BRCA +ve benefit* A



** Source: Tolaney S et al, J Clin Oncol 34, 2016 (suppl; abstr 2503).*

American Society of Clinical Oncology

Sapacitabine/seliciclib Phase 1 BRCA +ve benefit* B



** Source: Tolaney S et al, J Clin Oncol 34, 2016 (suppl; abstr 2503).*

American Society of Clinical Oncology

Financials

Financial position & capitalization

Mar. 31 2017 *Pro Forma* Cash & cash equivalents: $15.7m[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: ~ 4.6 million[4,5]

No debt

1. *Cyclacel press release*
2. *10-K.*
3. *Company estimate*
4. *10Q - 31 March 2017*
5. *Common stock outstanding: 4.3m.*

Key milestones

- CYC065 First-in-Human, Phase 1 data solid tumors
- Start CYC065 Phase 1, Part 2 in solid tumors
- Start CYC065 Ph 1b in R/R CLL combo with venetoclax
- Sapacitabine/seliciclib update BRCA+ve breast cancer
- CYC140 (PLKi) IND submission

Cyclacel Highlights

- Transcriptional CDKi and DNA Damage Response clinical stage oncology programs
- Treat difficult cancers and overcome resistance
- Competitive positioning
- Large markets

Contact: ir@cyclacel.com. Thank you.