

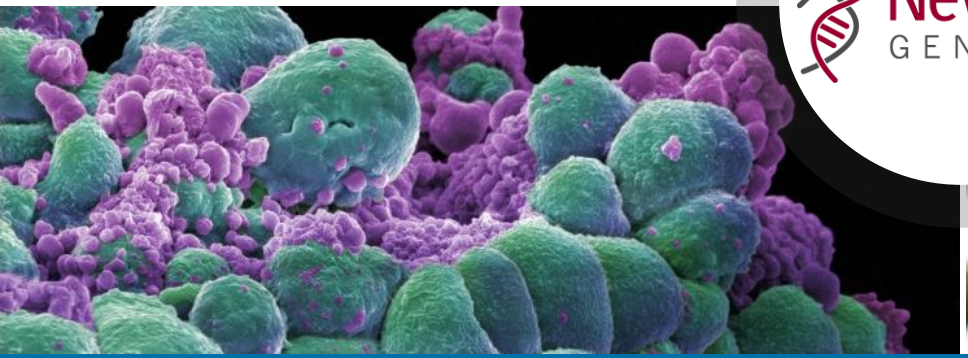


Cantor Fitzgerald Global Healthcare Conference

NewLink Genetics Corporation

NASDAQ: NLNK
September 25, 2017

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This presentation contains forward-looking statements of NewLink that involve substantial risks and uncertainties. All statements, other than statements of historical facts, contained in this presentation are forward-looking statements, within the meaning of The Private Securities Litigation Reform Act of 1995. The words "anticipate," "believe," "estimate," "expect," "intend," "may," "plan," "target," "potential," "will," "could," "should," "seek" or the negative of these terms or other similar expressions are intended to identify forward-looking statements, although not all forward-looking statements contain these identifying words. These forward-looking statements include, among others, statements about NewLink Genetics' financial guidance for 2017; results of its clinical trials for product candidates; its timing of release of data from ongoing clinical studies; its plans related to moving additional indications into clinical development; NewLink Genetics' future financial performance, results of operations, cash position and sufficiency of capital resources to fund its operating requirements; and any other statements other than statements of historical fact. Actual results or events could differ materially from the plans, intentions and expectations disclosed in the forward-looking statements that NewLink makes due to a number of important factors, including those risks discussed in "Risk Factors" and elsewhere in NewLink Genetics' Annual Report on Form 10-K for the year ended December 31, 2016 and other reports filed with the U.S. Securities and Exchange Commission (SEC). The forward-looking statements represent NewLink's views as of the date of this presentation. NewLink anticipates that subsequent events and developments will cause its views to change. However, while it may elect to update these forward-looking statements at some point in the future, it specifically disclaims any obligation to do so. You should, therefore, not rely on these forward-looking statements as representing NewLink Genetics' views as of any date subsequent to the date of this presentation.

NewLink Genetics

Focused on Indoximod, an IDO Pathway Inhibitor

- Indoleamine 2,3-dioxygenase (IDO) pathway is a key immuno-oncology target
- Our IDO pathway inhibitor, indoximod, has a differentiated mechanism of action (MOA)
- Indoximod's unique MOA may allow effectiveness in different combinations and therapeutic settings than direct enzymatic inhibitors
- Clinical data suggest indoximod may be a potent IDO pathway inhibitor in combination with multiple therapies and types of cancer:
  - PD-1 checkpoint inhibitors (advanced melanoma)
  - Cancer vaccine (metastatic prostate cancer)
  - Chemotherapy (pancreatic cancer and acute myeloid leukemia)
- Data support initiation of a pivotal trial of indoximod with PD-1 checkpoint blockade for patients with advanced melanoma, with goal to complete enrollment by the end of 2018

Indoximod has been studied in more than 700 patients

Recent Highlights

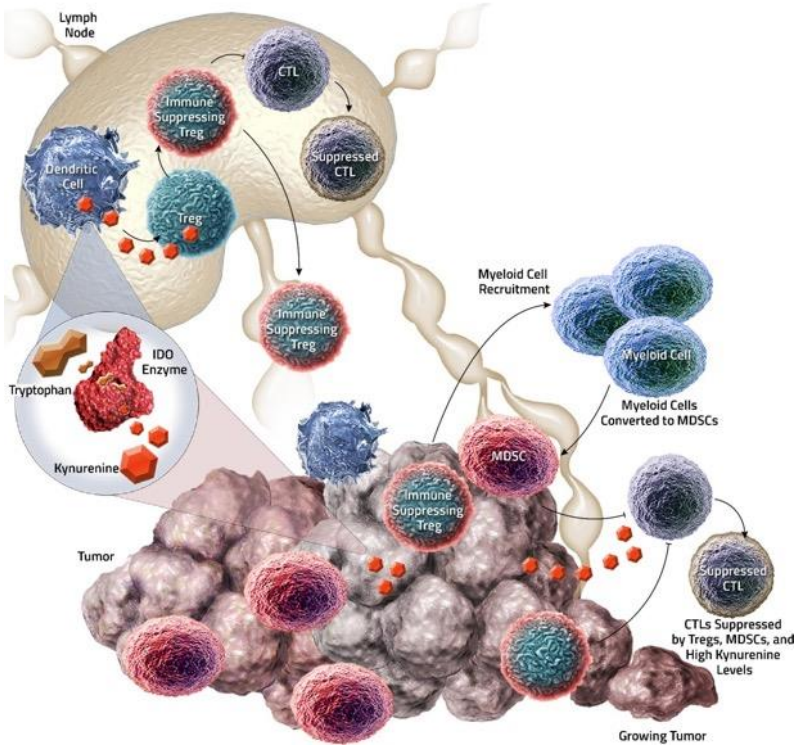
- Phase 2 data for indoximod plus PD-1 checkpoint inhibitor KEYTRUDA® (pembrolizumab) suggest substantial improvement of response rate for patients with advanced melanoma compared to PD-1 inhibitor alone
- Progress toward initiation of a pivotal trial with indoximod for patients with advanced melanoma
  - Pivotal trial of indoximod in advanced melanoma to include both approved PD-1 inhibitors, KEYTRUDA® (pembrolizumab) and OPDIVO® (nivolumab)
  - Pivotal trial has been designed as a large-scale (600 patients) trial in Stage III unresectable and metastatic stage IV melanoma
  - Co-primary endpoints of the study are PFS by RECIST criteria and Overall Survival (OS)
- Clinical collaboration with AstraZeneca in pancreatic cancer announced September 25, 2017
- Improved IP for salt and prodrug formulations of indoximod
  - New US patent issued August 15, 2017; provides exclusivity into 2036
- First patient dosed in the Phase 1 study of NLG802, a novel prodrug of indoximod

Indoximod program has made substantial progress in 2017

The IDO Pathway and Cancer

A Key Immuno-Oncology Target

- IDO (indoleamine 2,3-dioxygenase): intracellular enzyme that regulates immune response by degrading tryptophan to kynurenine[1]
- IDO pathway activity results in a shift of the ratio of tryptophan (↓) to kynurenine (↑)[1]
- This shift in ratio signals a suppressive phenotype rather than an activated antitumor phenotype[1]
- Tumors hijack the IDO pathway, a normal part of the immune system, to facilitate immune escape[2]



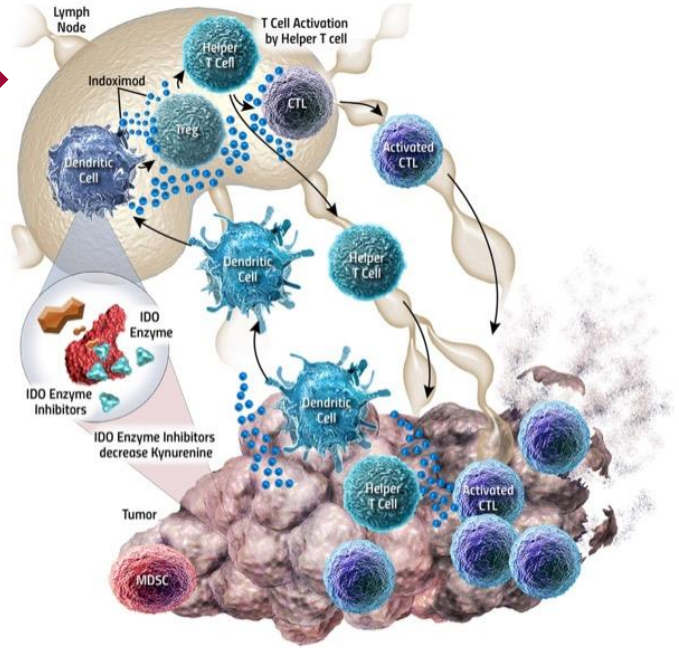
Treg, regulatory T cell; IDO, indoleamine 2,3-dioxygenase;
MDSC, myeloid-derived suppressor cell; CTL, cytotoxic T lymphocyte.

[1]Metz R. *Oncoimmunology*. 2012;1(9):1460-1468. [2]Johnson TS. *Immunol Invest*. 2012;41(6-7):765-797.

Targeting the IDO Pathway

Indoximod - A Unique Approach To Inhibiting the IDO Pathway

- **Indoximod**
  - Acts directly on immune cells to reverse IDO pathway–mediated suppression
- **Other IDO inhibitors**
  - Direct IDO enzymatic inhibitors, block tryptophan metabolism[1,2]



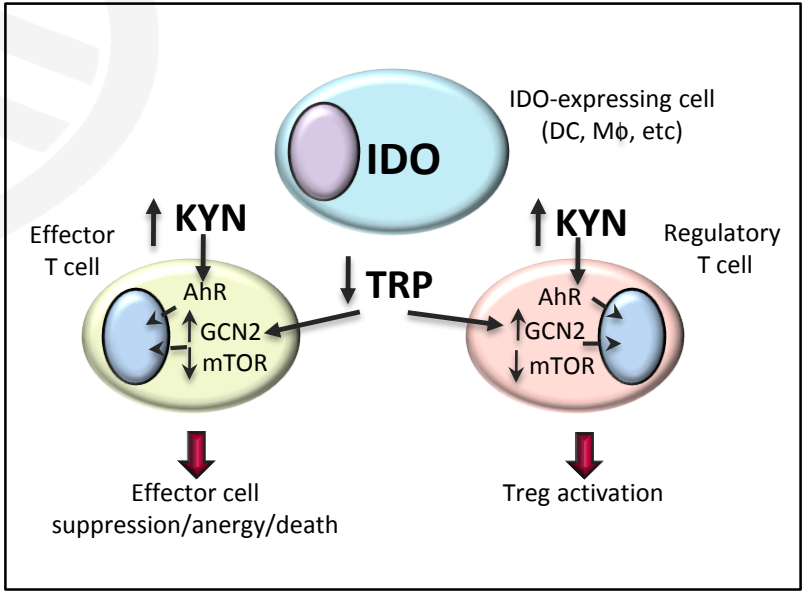
Indoximod has a differentiated mechanism of action

IDO, indoleamine 2,3-dioxygenase; Treg, regulatory T cell; MDSC, myeloid-derived suppressor cell; CTL, cytotoxic T lymphocyte.

[1]Mautino M. AACR 2013. Abstract 491. [2]Jochems C. *Oncotarget*. 2016;7(25):37762-37772.

IDO Pathway Mediated Immuno-Suppression

Treg Activation and Effector Cell Reduction



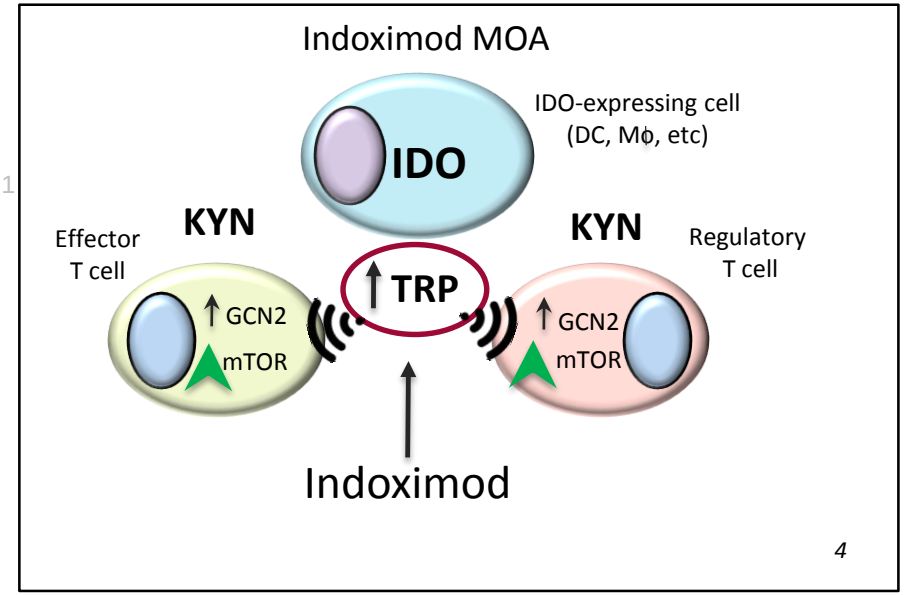
Immuno-suppressive feedback loop dependent on multiple components

Munn, D et al. *Trends in Immunology*. Page 12, Figure 1, March 2012.

Indoximod is an IDO Pathway Inhibitor with a Differentiated MOA

A Proposed Mechanism with a Broad Spectrum of Potential Clinical Utility

- Orally available tryptophan mimetic[1]
- Counteracts immunosuppressive effects of kynurenine[1]
- Activates multiple immune cells (effector cells)[2]
- Prevents activation of regulatory T cells (Tregs)[3]
- Reprograms Tregs into helper T cells[3]



Indoximod interrupts the IDO mediated immuno-suppressive feedback loop

[1]Metz R. *Oncoimmunology*. 2012;1(9):1460-1468; [2]Holmgaard RB, et al. Cell Reports. 2015;13(2):412-424;
[3]Sharma MD, et al. Immunity. 2010;33(6):942-954; [4]Adapted from Munn, D et al. *Trends in Immunology*. Page 12, Figure 1, March 2012.

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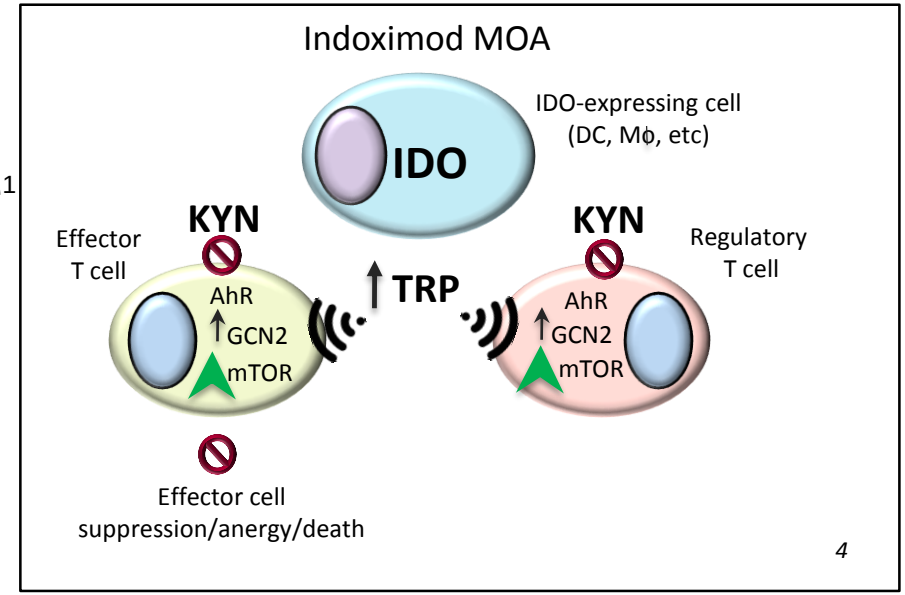

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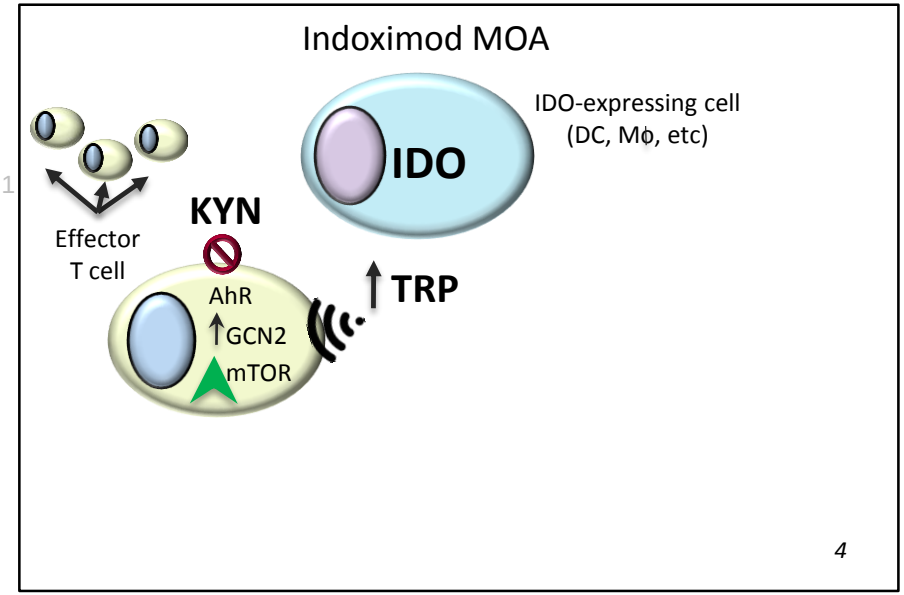

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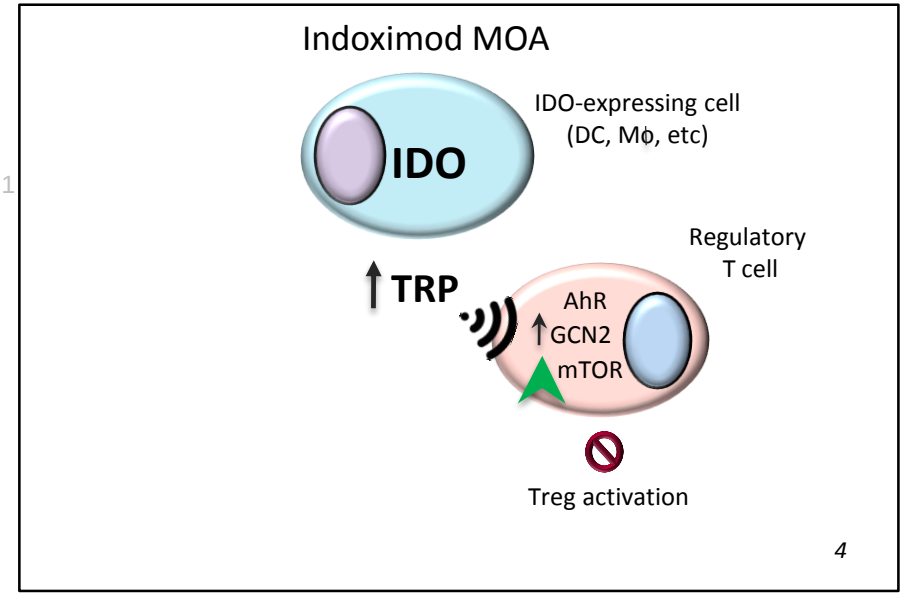

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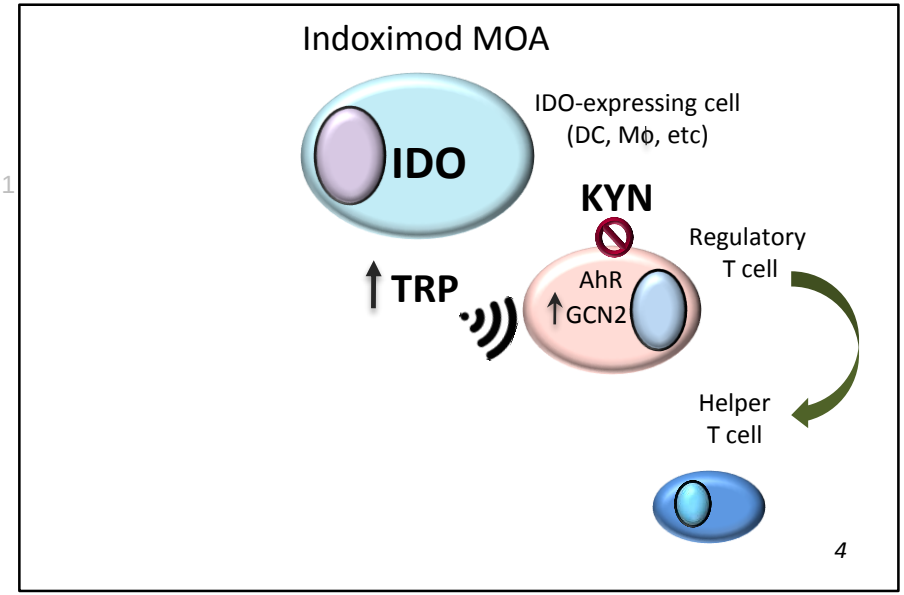

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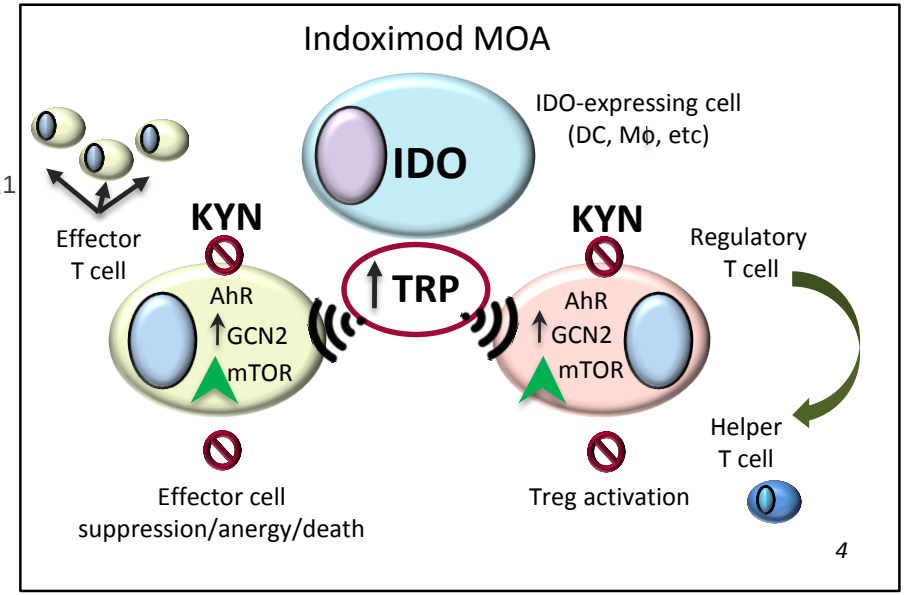

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Indoximod plus Pembrolizumab in Advanced Melanoma

Phase 2 (n=123 as of Aug 2017, Including Biopsy Cohort Still Enrolling)

N = 88	Cutaneous, Mucosal, or Melanoma Unknown Primary
51	**Evaluable reported AACR plenary session in April 2017 and updated September 2017**
21	Data pending
8	Received indoximod in combination with either ipilimumab (4) or nivolumab (4)
8	Not evaluable per protocol
N = 14	**Uveal Melanoma**
9	**Evaluable reported AACR plenary session in April 2017**
5	Data pending

Biopsy expansion cohort currently at 21 patients

Evaluable defined as having ≥ 1 on treatment image as of data cut-off

Baseline Demographic and Clinical Characteristics

Indoximod + Pembrolizumab for Advanced Melanoma

Characteristic	n = 51*
Median age (range), yr	62.9 (27–88)
Male, n (%)	34 (67)
Race/Ethnicity, n (%)	
White, non-Hispanic†	50 (98)
LDH above ULN, n (%)	12 (24)
Disease stage, n (%)	
III	8 (16)
IV	43 (84)
M1a	9 (18)
M1b	13 (25)
M1c	21 (41)

Characteristic	n = 51*
ECOG PS, n (%)	
0	38 (75)
1	13 (25)
Primary site, n (%)	
Cutaneous	40 (78)
Mucosal or primary unknown	11 (22)
Prior therapy, n (%)	
Radiation	9 (18)
Systemic therapy	14 (27)
None	28 (55)

ECOG PS, Eastern Cooperative Oncology Group performance status; LDH, lactate dehydrogenase; ULN, upper limit of normal.
*Excludes uveal melanoma patients.
†One patient declined to answer.

Updated Phase 2 Melanoma Patients – Key Findings

Cohort of 51 Patients Evaluable in April Continued to Improve

	Interim Phase 2 Data n (%) April 2017 (data as of January 2017)	Updated Phase 2 Data n (%) September 2017 (data as of August 2017)
ORR	**30 (59)**	**31 (61)**
CR	6 (12)	10 (20)
PR	24 (47)	21 (41)
SD	11 (22)	10 (20)
DCR	**41 (80)**	**41 (80)**
PD	10 (20)	10 (20)
Median PFS	N/R	**12.9 months**
PFS at 12 months	N/R	**56%**

ORR, overall response rate; CR, complete response; PR, partial response; SD, stable disease;
DCR, disease control rate; PD, progressive disease. Includes cutaneous, mucosal and metastatic of unknown primary

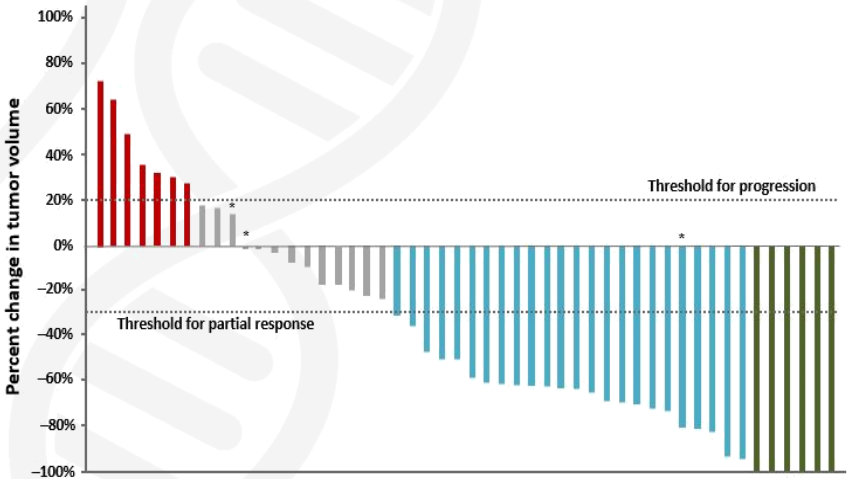
Based on Response Evaluation Criteria in Solid Tumors (RECIST) guidelines version 1.1 by site report.

Zakharia Y, et al. Oral presentation at Third International Cancer Immunotherapy Conference; Sep 6-9, 2017; Frankfurt, Germany.

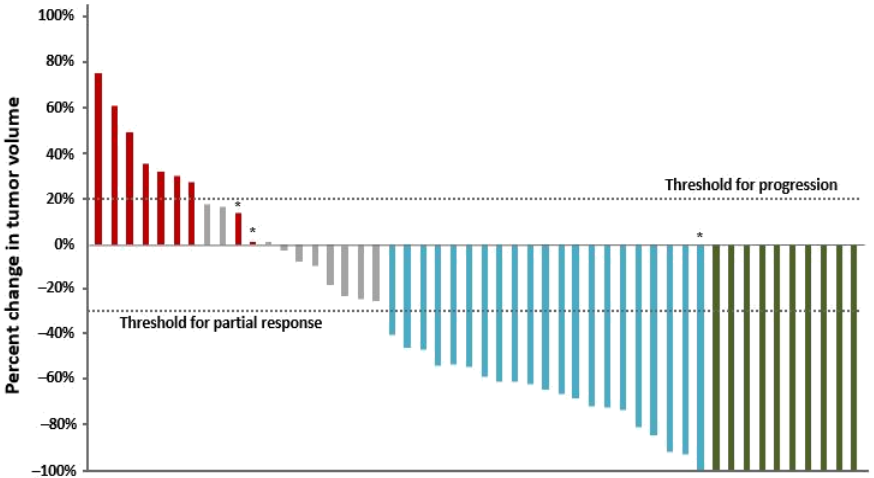
Best Response by Patient with Advanced Melanoma

Significant Depth of Response

Interim Phase 2 Melanoma Data
April 2017[1]



Same Patients Updated
September 2017[2]



Progressive Disease | Stable Disease | Partial Response | Complete Response

*Patients that progressed due to new non-target lesions.

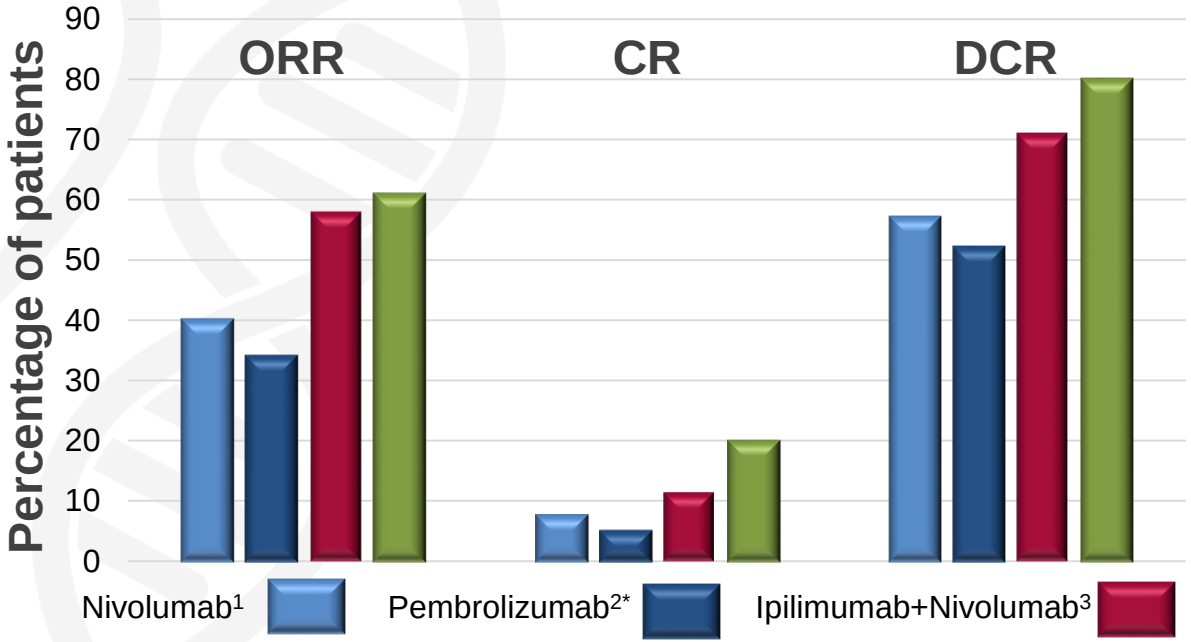
Note: 1 patient was unevaluable for response due to pleural effusion/collapsed left lung; the patient progressed based on several new non-target lesions.

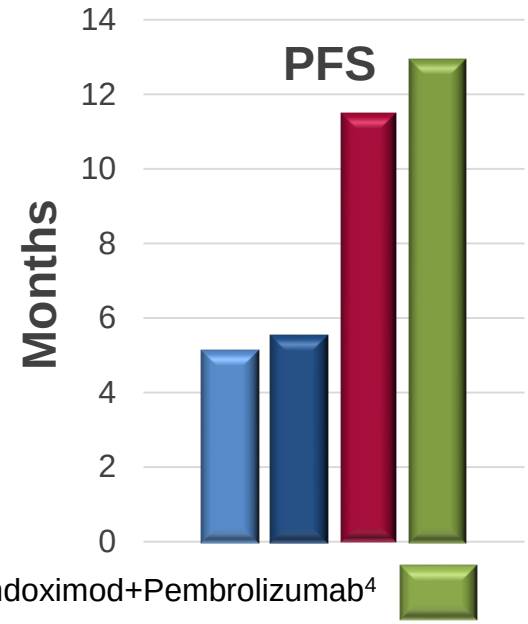
[1]Zakharia Y, et al. Oral presentation at: 107th Annual Meeting of the American Association of Cancer Research (AACR); April 1-5, 2017; Washington, DC. Abstract CT117.

[2]Zakharia Y, et al. Oral presentation at: Third International Cancer Immunology Conference; September 6-9, 2017; Frankfurt, Germany.

Indoximod Plus PD-1 Response and Survival in Advanced Melanoma

Potential to Improve Outcomes Without Added Toxicity of Ipilimumab + Nivolumab





Comparative anti-PD-1 monotherapy & anti-PD-1 + anti-PD-1 combination data provided for illustrative purposes only; no head-to-head trials conducted.

*Data are for Q2W regimen. ORR, overall response rate; CR, complete response; DCR, disease control rate; PFS, progression-free survival.

[1]Robert C, et al. *N Engl J Med*. 2015;372(4): 320-330. [2]Robert C, et al. *N Engl J Med*. 2015;372(26): 2521-2532. [3]Larkin J, et al. *N Engl J Med*. 2015;373(1):23-34.

[4]Zakharia Y. Oral presentation at: Third International Cancer Immunotherapy Conference, September 6-9, 2017; Frankfurt, Germany.

Indoximod plus PD-1 (Pembrolizumab)

Adverse Event Profile Appears Comparable to Pembrolizumab Alone

- Combination was generally well tolerated
- No treatment related Grade 4/5 adverse events reported to date
- Serious Adverse Events (SAEs) led to discontinuation in only 3 patients
- SAEs labelled as possibly related to indoximod reported in 4 patients
- Limited immune-mediated adverse events reported regardless of attribution to treatment
- No treatment related deaths were reported

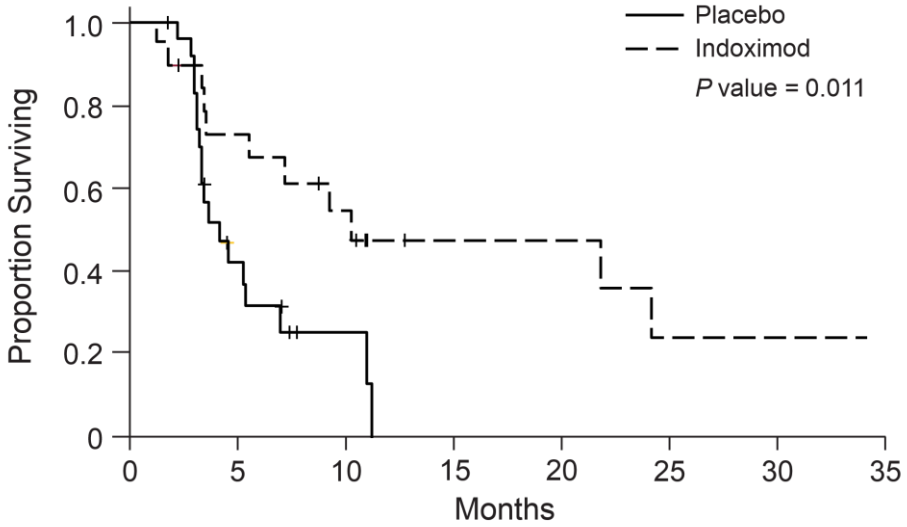
Full dose combination generally well tolerated

Zakharia Y, et al. Oral presentation at: 107th Annual Meeting of the American Association for Cancer Research (AACR); April 1-5, 2017; Washington, DC. Abstract CT117.

Indoximod plus Vaccine (Provenge [sipuleucel-T])

Randomized, Double-blind, Placebo-controlled Phase 2 Study

- 46 patients: indoximod (22) vs placebo (24)
- PA2024 ELISPOT response data showed no statistically significant difference*
- Statistically significant improvement in radiographic progression-free survival (rPFS)
  - Median rPFS of 10.3 months for indoximod vs 4.1** months in placebo (p=0.011)
  - Median OS has not yet been reached
- Combination treatment was well tolerated



Randomized data suggesting indoximod can improve responses beyond PD-1

*PA2024 response was evaluable in 35 patients.

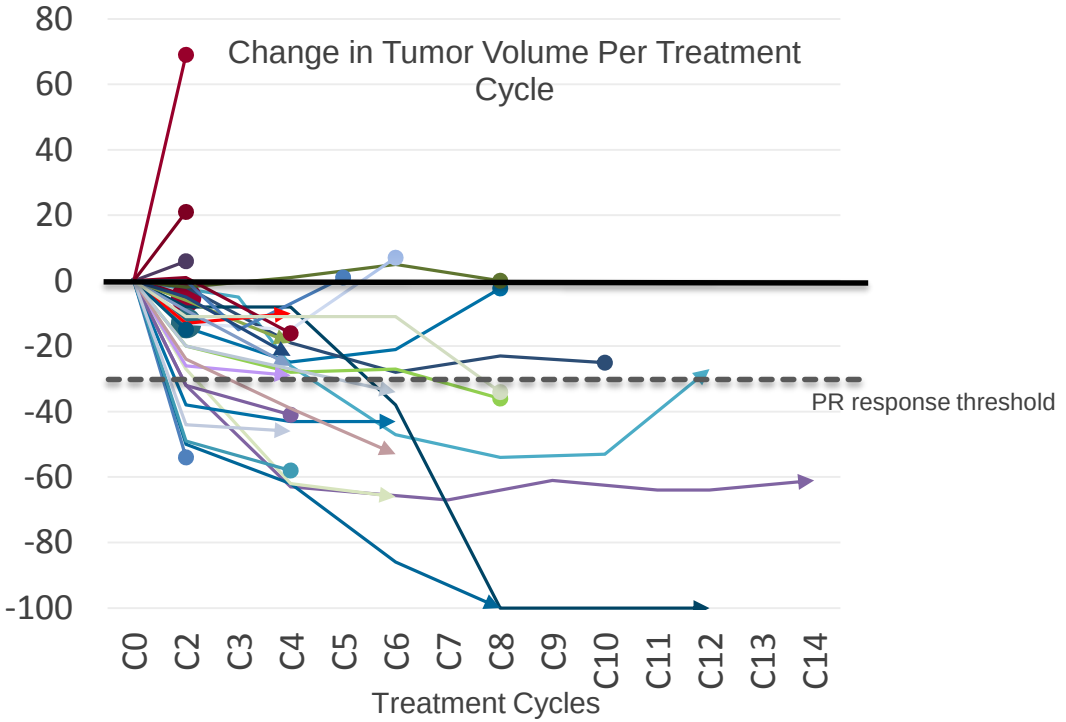
**Median time to objective progression for pivotal IMPACT trial of sipuleucel-T was 3.7 mo.

Jha, et al. 2017 ASCO Annual Meeting. Abstract 3066

Indoximod plus Chemotherapy (gemcitabine/nab-paclitaxel)

Encouraging Interim Phase 2 Data

- Overall, combination was well tolerated
- 45 patients enrolled 4 months or longer
- 31 patients evaluable for response
  - 45% (14/31) overall response rate
  - Two complete responses
- Investigator reported response
- Early and delayed durable responses suggest immune mediated mechanism
- Final readout expected in 1H:2018



Bahary, et al. ASCO 2016. Abstract #3020.

Depth and duration of responses suggest immune-mediated mechanism

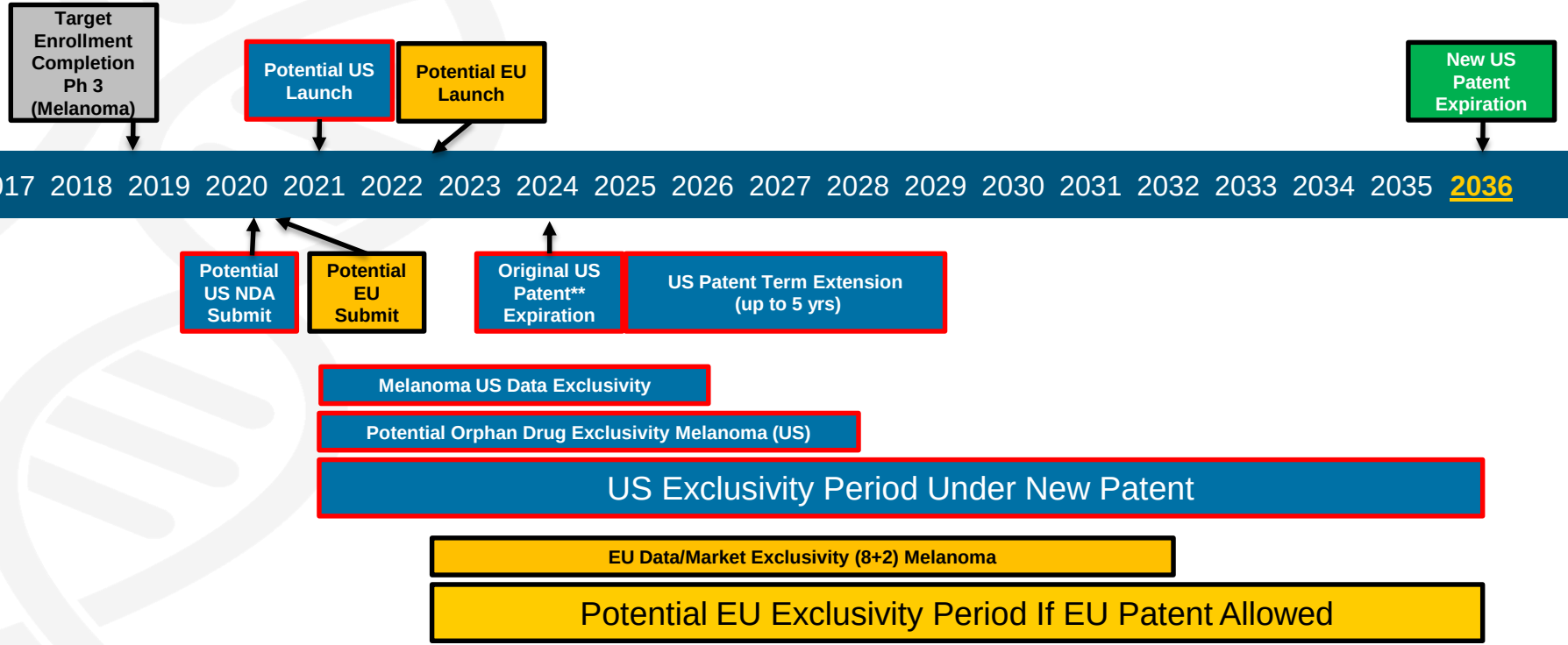
Clinical Collaboration with AstraZeneca

Indoximod and Durvalumab plus Chemotherapy for Pancreatic Cancer

- Phase 2 randomized (2:1:1), double-blinded, placebo-controlled trial
- Chemotherapy-naïve metastatic pancreatic cancer
- Study design includes 3 arms (n= approximately 200):
  A. Indoximod + durvalumab + gemcitabine/nab-paclitaxel
  B. Durvalumab + gemcitabine/nab-paclitaxel
  C. Gemcitabine/nab-paclitaxel
- Primary objectives: Evaluate safety and efficacy of four-drug combination vs gem/nab-paclitaxel
- 50/50 sharing of costs
- NewLink Genetics to serve as the study sponsor
- Plan to begin enrolling patients in Q1 2018

Allowed US Patent for Indoximod Formulation (Salt)

Potential Market Exclusivity In Advanced Melanoma



**Current patent will still apply along with salt patent because it covers inclusion of d-1-methyl-tryptophan (indoximod)

Financial Position

Cash and Equivalents (June 30, 2017)	$107.8 million
Debt (June 30, 2017)	~$0.4 million
YE 2017 Cash (Projected)	~$100 million[1,2]
Quarterly Use of Cash (Projected)	$14 - $16 million[1]
Shares Outstanding	~31 million shares[2]

[1] Excludes any proceeds from future financing or collaborations.
[2] Reflects the sale of approximately 1.9 million shares under the Company's "at the market" offering since June 30, 2017.

~Two years of cash and equivalents on the balance sheet