UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
Washington, D.C. 20549

FORM 8-K

CURRENT REPORT
Pursuant to Section 13 OR 15(d) of
The Securities Exchange Act of 1934

Date of Report (Date of earliest event reported): April 16, 2018 (April 11, 2018)

NewLink Genetics Corporation
(Exact name of registrant as specified in its charter)

Delaware	**001-35342**	**42-1491350**
(State or other jurisdiction of incorporation)	(Commission File Number)	(IRS Employer Identification No.)

2503 South Loop Drive **Ames, IA**	**50010**
(Address of principal executive offices)	(Zip Code)

Registrant's telephone number, including area code: **(515) 296-5555**

Not applicable
(Former name or former address, if changed since last report.)

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions (see General Instruction A.2. below):

[ ] Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)

[ ] Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)

[ ] Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))

[ ] Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 (§ 230.405 of this chapter) or Rule 12b-2 of the Securities Exchange Act of 1934 (§ 240.12b-2 of this chapter).

Emerging growth company o

If an emerging growth company, indicate by check mark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act o

Section 5 - Corporate Governance and Management

Item 5.02. Departure of Directors or Certain Officers; Election of Directors; Appointment of Certain Officers; Compensatory Arrangements of Certain Officers.

On April 11, 2018, Paul Edick notified NewLink Genetics Corporation ("NewLink") that he will resign his position as a member of the board of directors of NewLink effective April 30, 2018. Mr. Edick's professional obligations have increased, and he has determined that it will not be feasible for him to continue as a member of the NewLink board. Mr. Edick served as a director of NewLink since July 2011. His resignation is not due to any disagreement with NewLink regarding any of its operations, policies or practices.

NewLink has identified a candidate for director to fill the vacancy created by Mr. Edick’s resignation.

Section 8 - Other Events

Item 8.01. Other Events.

On April 15, 2018, NewLink issued a press release titled "NewLink Genetics Announces Initial Phase 1 Data with Indoximod Plus Radiation and Chemotherapy for Pediatric Patients with Diffuse Intrinsic Pontine Glioma (DIPG) Presented During AACR Plenary."

The Company has determined that it will not initiate the randomization portion of Indigo301, its study of indoximod in combination with pembrolizumab or nivolumab for patients with advanced melanoma. NewLink's clinical team will evaluate the design, trial size and feasibility of an alternative randomized evaluation of indoximod in melanoma in the context of the failure of a competitor's trial of its enzymatic IDO inhibitor in a similar clinical setting. Our evaluation will include analysis of the full data set from the Company's single-arm Phase 2 melanoma study, the differentiated mechanism of action of indoximod, and the opinions of experts in the field. The company plans to present final results from its Phase 2 melanoma trial and its single-arm Phase 2 pancreatic cancer trial at an upcoming medical meeting in the first half of this year.

Indoximod had demonstrated encouraging clinical data in other cancer indications in combination with chemotherapy, vaccines, radiotherapy, and checkpoint blockade. The company is currently evaluating the most promising indications within the indoximod program to move further into Phase 2 development.

A copy of the press release and the accompanying presentation slides are attached hereto as Exhibits 99.1 and 99.2, respectively, and are incorporated herein by reference.

Section 9 - Financial Statements and Exhibits

Item 9.01. Financial Statements and Exhibits.

(d) Exhibits.

Exhibit Number	Description
99.1	Press Release, dated April 15, 2018, entitled "NewLink Genetics Announces Initial Phase 1 Data with Indoximod Plus Radiation and Chemotherapy for Pediatric Patients with Diffuse Intrinsic Pontine Glioma (DIPG) Presented During AACR Plenary"
99.2	DIPG Presentation Slide Deck

SIGNATURES

Pursuant to the requirements of the Securities Exchange Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned hereunto duly authorized.

Dated: April 16, 2018

NewLink Genetics Corporation

By: /s/ John B. Henneman III
John B. Henneman III
Its: Chief Financial Officer

NewLink Genetics Announces Initial Phase 1 Data with Indoximod Plus Radiation and Chemotherapy for Pediatric Patients with Diffuse Intrinsic Pontine Glioma (DIPG) Presented During AACR Plenary

Early data indicate indoximod has clinical activity when used in combination therapies beyond PD-1 inhibition

AMES, Iowa, April 15, 2018 - NewLink Genetics Corporation (NASDAQ:NLNK), today reported initial data from NLG2105, a Phase 1 study evaluating indoximod, its IDO pathway inhibitor, in combination with radiation and chemotherapy for the treatment of pediatric patients with progressive brain tumors during the "Multimodality Immuno-oncology Approaches" session at the American Association for Cancer Research (AACR) 2018 Annual Meeting in Chicago.

The presentation reviewed NewLink Genetics' trial evaluating the combination of indoximod with radiotherapy and chemotherapy for children with malignant brain tumors. Indoximod has immunostimulatory effects involving multiple immune cell types. Indoximod works by reversing the effects of low tryptophan by increasing proliferation of effector T cells, and directly reprogramming T regulatory cells into helper T cells. Initially, 29 heavily pretreated patients were enrolled in a dose-escalation protocol with initial data presented at the Society for Neuro-Oncology Conference, November 2017. Seventeen of the 29 patients were appropriate candidates for re-irradiation of their tumors and were treated with a combination therapy including indoximod plus conformational radiotherapy followed by maintenance indoximod combined with temozolomide chemotherapy. The other 12 patients were treated with immuno-chemotherapy consisting of indoximod and temozolomide. In aggregate, with further follow-up, the 29 subjects in the dose-escalation phase of the study had a median progression-free survival (mPFS) of 6.2 months and median time on study (time to regimen failure, TTRF) of 11.7 months. The treatment continued to be well tolerated with minimal toxicity attributed to indoximod.

"These early data, though from a small cohort of pediatric patients, demonstrate the potential of the indoximod plus radiochemotherapy combination without an increase in toxicity for these children," said Dr. Theodore S. Johnson, M.D., Ph.D., Associate Professor of Pediatrics at Augusta University, lead investigator for the trial.

Once initial safety data were generated, an additional pilot cohort of newly-diagnosed patients with diffuse intrinsic pontine glioma (DIPG) was opened using indoximod during front-line radiotherapy (RT) followed by maintenance indoximod plus temozolomide. Six newly diagnosed DIPG patients initiated treatment, with all 6 having completed induction radioimmunotherapy. Treatment was well tolerated with symptomatic improvement in all 6 patients. Site-reported radiographic review indicated near resolution of tumor in one patient at the end of radiotherapy and observable improvement in 5 out of 6 patients overall. A seventh patient with progressive DIPG received re-RT combined with indoximod, which was well tolerated with symptomatic improvement and objective tumor reduction per site-reported assessment on post-RT MRI.

“These initial findings further support the potential for indoximod in combination with other agents,” said Charles J. Link, Jr., M.D., Chairman and Chief Executive Officer. “We look forward to working with our investigators toward gathering more data on the effects of indoximod on this deadly disease.”

NewLink will also present during poster session PO.IM02.07, Immunomodulatory Agents and Interventions 1, Abstract 3753, entitled: *Indoximod modulates AhR-driven transcription of genes that control immune function*, from 8:00 AM - 12:00 PM CT on Tuesday, April 17, 2018.

Separately, the Company has determined that it will not initiate the randomization portion of Indigo301, its study of indoximod in combination with pembrolizumab or nivolumab for patients with advanced melanoma. NewLink’s clinical team will evaluate the design, trial size and feasibility of an alternative randomized evaluation of indoximod in melanoma in the context of the failure of a competitor’s trial of its enzymatic IDO inhibitor in a similar clinical setting. The evaluation will include analysis of the full data set from the Company’s single-arm Phase 2 melanoma study, the differentiated mechanism of action of indoximod, and the opinions of experts in the field. The Company will present final results from its Phase 2 trial in melanoma and its single-arm Phase 2 trial in pancreatic cancer at an upcoming medical conference in the first half of 2018.

About Indoximod

Indoximod is an investigational, orally available small molecule targeting the IDO pathway. The IDO pathway is a key immuno-oncology target involved in regulating the tumor microenvironment and immune escape. Indoximod is being evaluated in combination with treatment regimens including anti-PD-1/PD-L1 agents, cancer vaccines, radiation and chemotherapy across multiple indications such as melanoma, pancreatic cancer and other malignancies.

About NewLink Genetics Corporation

NewLink Genetics is a late-stage biopharmaceutical company focusing on discovering, developing and commercializing novel immuno-oncology product candidates to improve the lives of patients with cancer. NewLink Genetics' IDO pathway inhibitors are designed to harness multiple components of the immune system to combat cancer. For more information, please visit www.newlinkgenetics.com and follow us on Twitter @NLNKGenetics.

Cautionary Note Regarding Forward-Looking Statements

This press release contains forward-looking statements of NewLink Genetics that involve substantial risks and uncertainties. All statements, other than statements of historical fact, contained in this press release 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 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; 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, 2017 and other reports filed with the U.S. Securities and Exchange Commission (SEC). The forward-looking statements in this press release represent NewLink Genetics’ views as of the

date of this press release. NewLink Genetics 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 press release.

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Front-line Therapy of DIPG Using IDO Pathway Inhibitor Indoximod in Combination with Radiation and Chemotherapy

American Association of Cancer Research (AACR) 2018

Theodore S. Johnson, MD, PhD

Georgia Cancer Center – Augusta University

April 15, 2018

Cautionary Note Regarding Forward-Looking Statements

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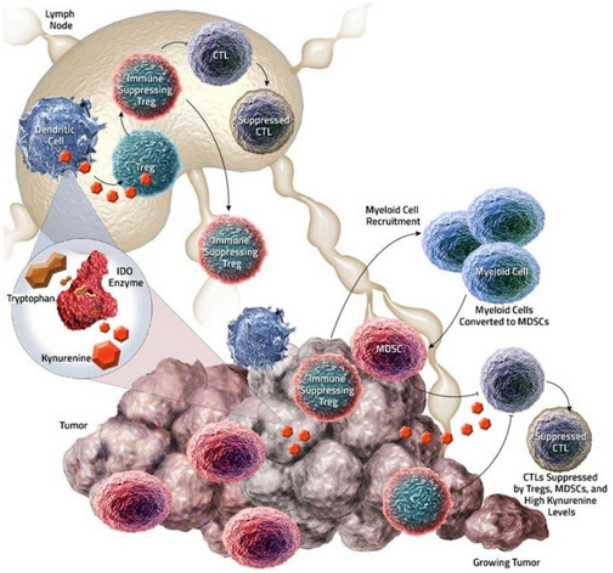
IDO Pathway a Key Immuno-oncology Target

- 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]



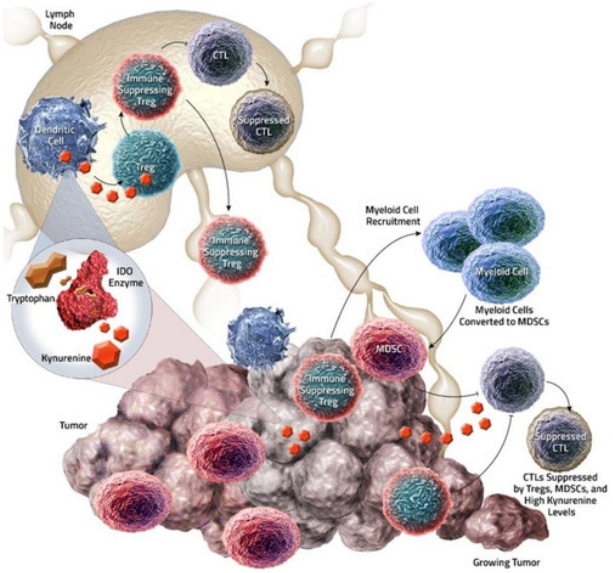
Treg, regulatory T cell; IDO, indoleamine 2,3-dioxygenase; MDSC, myeloid-derived suppressor cell; CTL, cytotoxic T lymphocyte.

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

IDO Pathway a Key Immuno-oncology Target

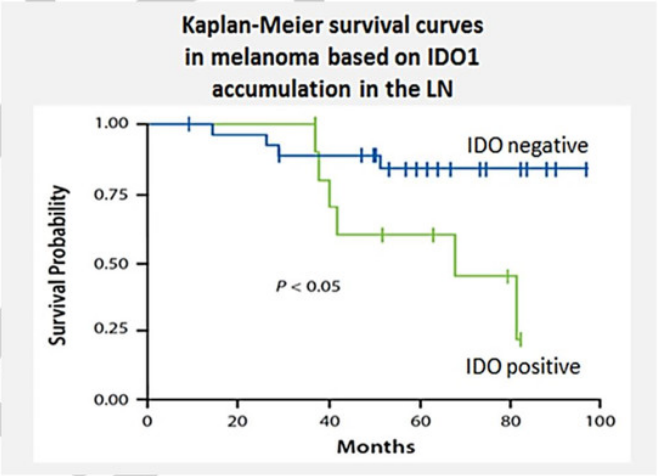
Key points:

- IDO is a natural mechanism of immunosuppression and tolerance in the immune system involved in
  - Acquired peripheral tolerance (pregnancy, mucosal tolerance)
  - Maintenance of tolerance to apoptotic cells (including apoptotic tumor cells)
- We hypothesize that the effect on tolerance to apoptotic cells may be critical for synergy with chemotherapy and radiation



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

IDO Expression in Certain Tumors is Associated with Poor Patient Outcomes

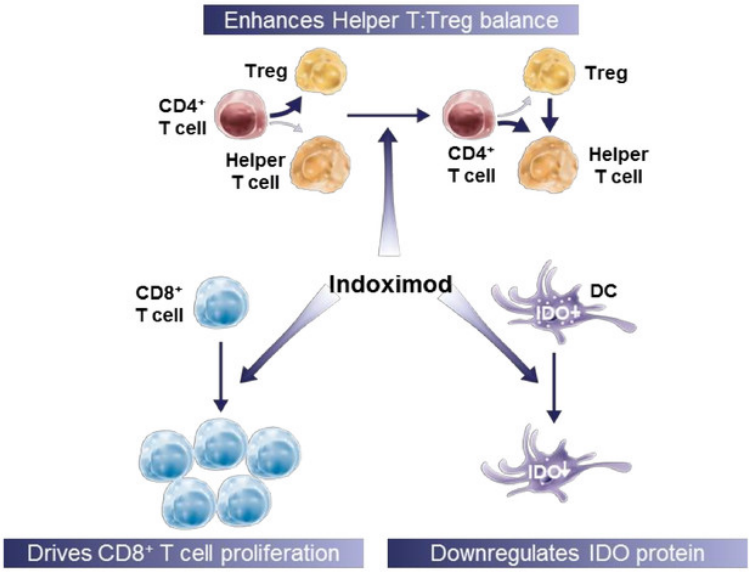


- IDO1 is highly expressed in multiple tumor types
  - Melanoma
  - NSCLC
  - Ovarian cancer
  - Pancreatic cancer
  - Colorectal cancer
  - Glioblastoma
  - Squamous cell carcinoma
  - Endometrial carcinoma
  - DLBCL
  - RCC
  - TCC
  - TNBC

IDO, indoleamine 2,3-dioxygenase; LN, lymph node; NSCLC, non-small cell lung cancer; DLBCL, diffuse large B-cell lymphoma; RCC, renal cell carcinoma; TCC, transitional cell carcinoma; TNBC, triple-negative breast cancer. Munn DH, et al. J Clin Invest. 2004;114(2):280-290.

Indoximod Differentiated Mechanism of Action

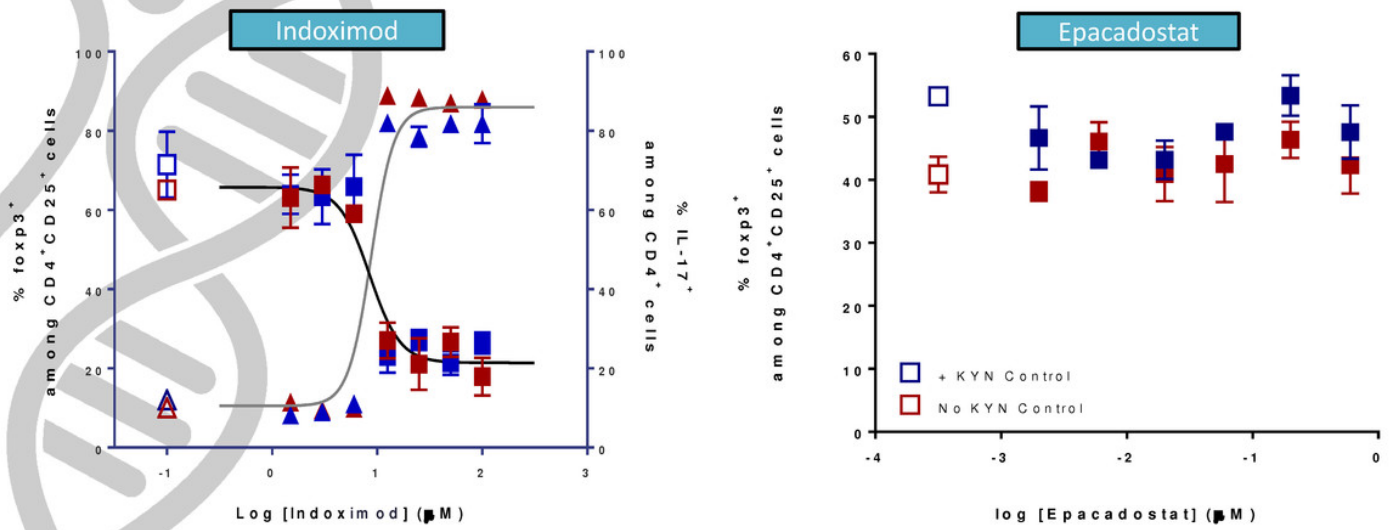
- Orally administered, small-molecule IDO pathway inhibitor that reverses the immunosuppressive effects of low tryptophan and high kynurenine that result from IDO activity
- Immunostimulatory effects involving 3 main cell types: CD8+ T cells, T regulatory cells, and dendritic cells[1]
  - Reverses effects of low tryptophan by increasing proliferation of effector T cells
  - Directly reprograms T regulatory cells to helper T cells
  - Downregulates IDO expression in dendritic cells
- Potential synergy has been shown with checkpoint blockade, chemotherapy, radiation and vaccines



IDO, indoleamine 2,3-dioxygenase; Treg, T regulatory cell; DC, dendritic cell.
1. Brincks EL, et al. Poster presented at the AACR Annual Meeting. April 14-18, 2018. Abstract 3753.

Indoximod vs Epacadostat: A Differentiated Mechanism of Action

Indoximod Directly Reprograms T Regulatory Cells Helper T Cells



AACR Poster #3753 (Brincks, EL, *et al*, 2018)

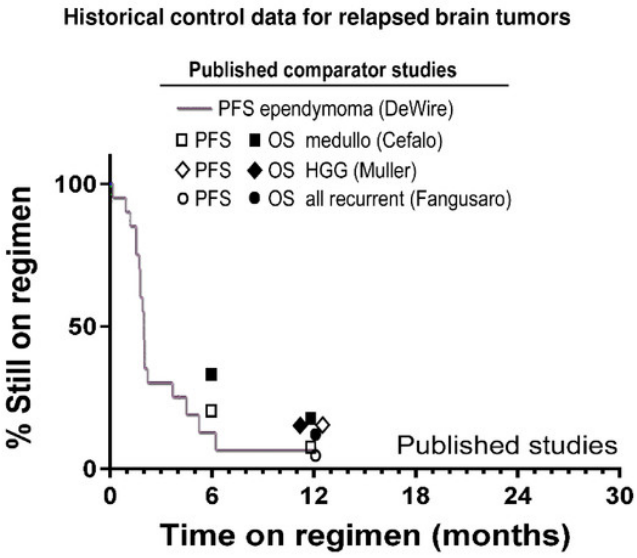
Designing Multimodal Chemo-radio-immunotherapy

- Hypothesis
  - Immune activation (immunotherapy) can allow responsiveness to chemotherapy and radiation in patients who would otherwise be refractory
- However, this synergy with chemotherapy/radiation requires targeting the antigen-presenting step and creating a pro-inflammatory (immunogenic) tumor milieu
  - Essentially, it must break tolerance to the dying/apoptotic tumor cells
  - This antigen cross-presentation step lies upstream of the conventional T-cell checkpoints

Recurrent/Refractory Pediatric Brain Tumors

Recurrent/refractory brain tumors represent the greatest single cause of mortality in pediatric cancer

- Cannot be cured by current standard treatments (treatment-refractory)
- Standard of care is largely palliative



PFS, progression-free survival; OS, overall survival; HGG, high grade glioma. Historical controls adapted from: DeWire M, et al. J Neurooncol. 2015;123:85; Cefalo G, et al. Neuro Oncol. 2014;16:748; Muller K, et al. Radiat Oncol. 2014;9:177; Fangusaro JR, et al. J Clin Oncol. 2017;35(suppl): abstract 10543

First-in-children Phase 1 Trial of Indoximod-based Multimodal Chemo-radio-immunotherapy

- Relapsed or refractory primary brain tumor patients
- Primary endpoints
  - Regimen limiting toxicities of indoximod + temozolomide
  - Objective response rate
  - Regimen-limiting toxicities of indoximod + radiation
  - Safety
- Key eligibility criteria
  - 3-21 years of age
  - Histologically proven initial diagnosis of primary malignant brain tumor, with no known curative treatment options
  - MRI confirmation of tumor progression
- Multimodal management is a key feature of the regimen
- Radiographic evidence of progression (escape lesions) can be managed with continued indoximod and:
  - Surgical resection (regain local control)
  - Targeted radiation (regain local control)
  - Crossover to 2nd-line chemotherapy (cyclophosphamide/etoposide)

MRI, magnetic resonance imaging.
Clinicaltrials.gov (NCT02502708).

First-in-children Phase 1 Trial of Indoximod-based Multimodal Chemo-radio-immunotherapy

Group 1
- **Indoximod dose escalation (study dose, PO, twice daily on days 1-28)**
- **Temozolomide (200 mg/m^2/day, PO, once daily on days 1-5 of 28-day cycles)**

Group 2 (expansion cohort of Group 1)
- RP2D of indoximod
- Temozolomide (200 mg/m^2/day, PO, once daily on days 1-5 of 28-day cycles)

Group 3
- **Indoximod dose escalation (study dose, PO, twice daily on days 1-28)**
- **Individualized radiation plan**
- **Followed by indoximod combined with cyclic temozolomide**

Group 4 (progressive disease on indoximod + temozolomide)
- Indoximod (32 mg/kg/dose PO, twice daily on days 1-28)
- Cyclophosphamide (2.5 mg/kg/dose PO, once daily)
- Etoposide (50 mg/m^2/dose PO, once daily)

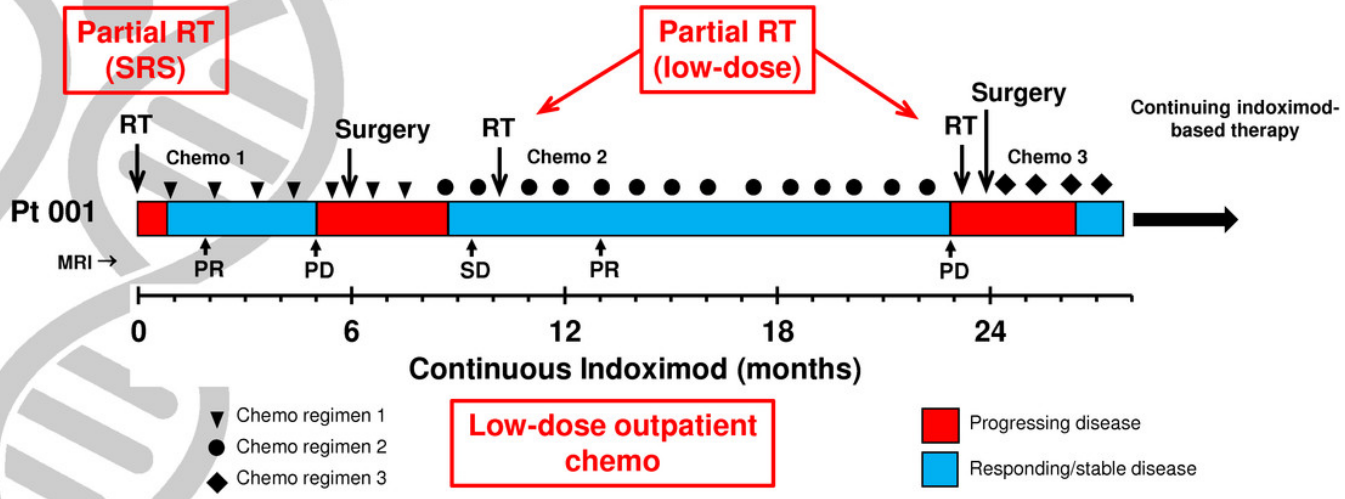
PO, orally; RP2D, recommended phase 2 dose.
Clinicaltrials.gov (NCT02502708

Patient Demographics (Mixed Population)

Total patients enrolled	N = 29
Diagnosis, n (%)	
Ependymoma	14 (48)
Malignant glioma*	9 (31)
Medulloblastoma**	6 (21)
Gender, n (%)	
Female	10 (34)
Male	19 (66)
Race, n (%)	
African American	3 (10)
Caucasian	23 (79)
Hispanic	0
Other	2 (7)
Declined to provide	1 (3)
Age, years	
Median	12.5
Range	3–20

*Includes one each gliosarcoma, bithalamic glioma, and ganglioglioma.
**Includes one previously classified as primitive neuroectodermal tumor.

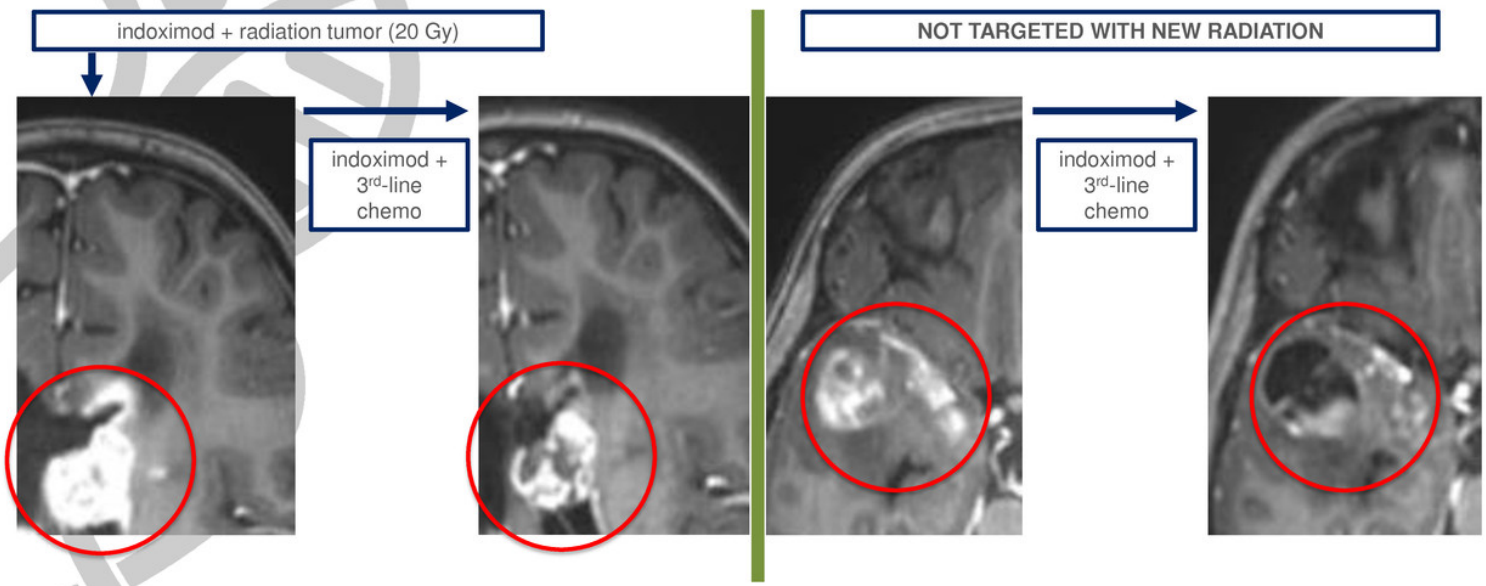
Patient 001: Example of Multimodal Management Chemo-radio-immunotherapy



- 7-year-old with ependymoma: prolonged disease responsiveness
- Indoximod-based multimodal regimen is well tolerated

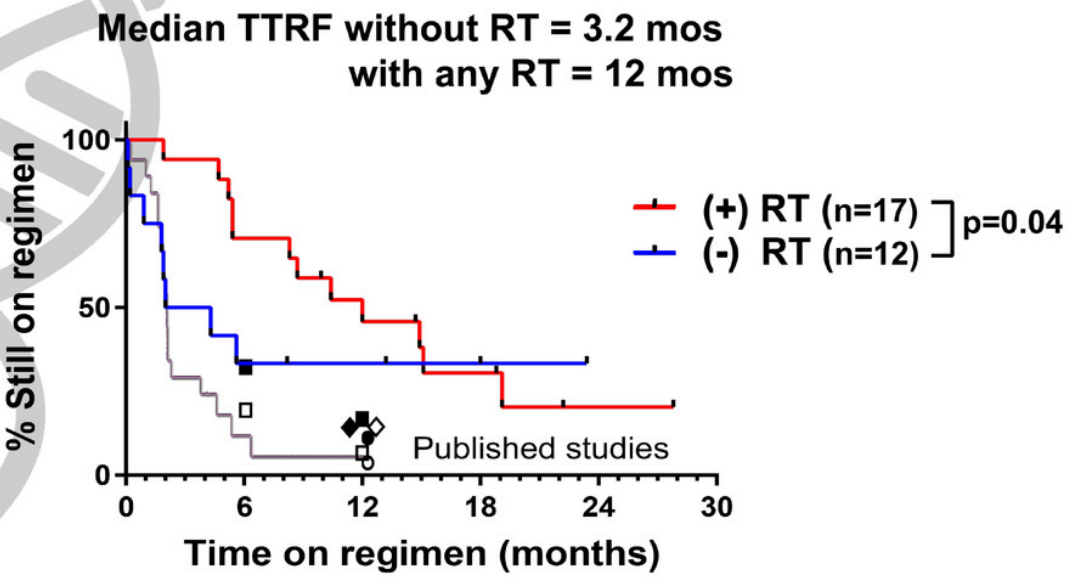
SRS, stereotactic radiosurgery; RT, radiation therapy; PR, partial response; PD, progressive disease; SD, stable disease.

Patient 001: Continued Responsiveness Using Indoximod-based Multimodal Management



Johnson, T, *et al*, AACR 2018

Radio-Immunotherapy Improves Time to Regimen Failure (TTRF)



Historical controls adapted from: DeWire M, et al. J Neurooncol 2015;123:85.
Cefalo G, et al. Neuro Oncol 2014;16:748.
Muller K, et al. Radiat Oncol 2014;9:177.

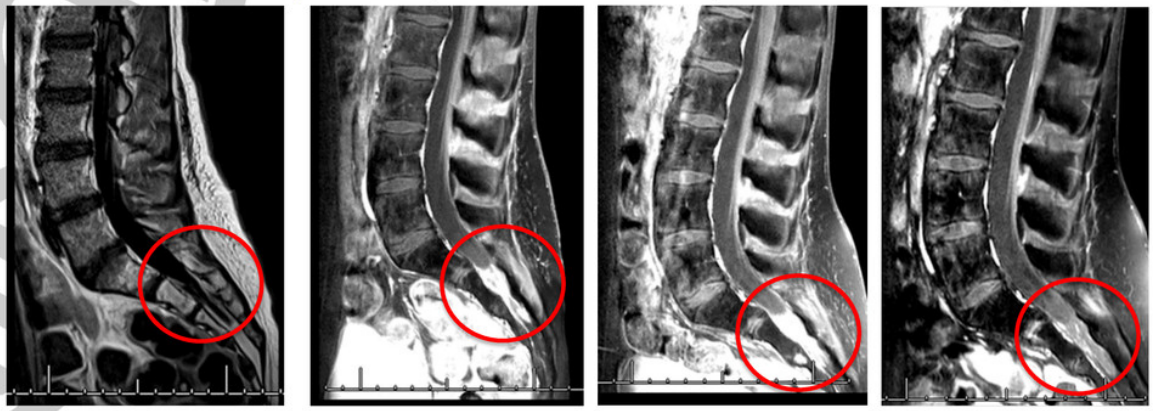
RT, radiation therapy

Fangusaro JR, et al. J Clin Oncol, 2017;35(suppl): abstract 10543.

New Metastatic Tumor Arising While on Therapy Later Regresses

14 yo with CSF relapse of medulloblastoma

Begin indoximod + temozolomide



Potential for late responses makes TTRF an important outcome metric

CSF, cerebrospinal fluid; TTRF, time to regimen failure.

Indoximod-based Multimodal Regimen is Well Tolerated

- In the 29 patients included in the study, SAEs possibly related to indoximod included 1 case each of:
  - Febrile neutropenia
  - Hemiparesis
  - Hydrocephalus
  - Spinal cord compression
  - Status epilepticus
  - Urinary tract infection
- Overall, indoximod did not worsen the toxicity of the base treatment

Pilot Cohort in Diffuse Intrinsic Pontine Glioma (DIPG)

Group 1
- **Indoximod dose escalation (study dose, PO, twice daily on days 1-28)**
- **Temozolomide (200 mg/m^2/day, PO, once daily on days 1-5 of 28-day cycles)**

Group 2 (expansion cohort of Group 1)
- RP2D of indoximod
- Temozolomide (200 mg/m^2/day, PO, once daily on days 1-5 of 28-day cycles)

Group 3
- **Indoximod dose escalation (study dose, PO, twice daily on days 1-28)**
- **Individualized radiation plan**
- **Followed by indoximod combined with cyclic temozolomide**

→ **Pilot cohort**
- **Patients with radiographic diagnosis or histologically proven DIPG**

Group 4 (progressive disease on indoximod + temozolomide)
- Indoximod (32 mg/kg/dose PO, twice daily on days 1-28)
- Cyclophosphamide (2.5 mg/kg/dose PO, once daily)
- Etoposide (50 mg/m^2/dose PO, once daily)

DIPG Is Rapidly Fatal

- DIPG has the worst prognosis of any pediatric cancer
- Median time to progression after radiation is ~6 months[1]
- At progression, patients follow a rapidly declining course
  - Median OS is 10-12 months[2]
  - Uniformly fatal

DIPG, diffuse intrinsic pontine glioma; OS, overall survival.

1. Wolff JE, et al. J Neurooncol. 2012;106(2):391-397. 2. Cohen KJ, et al. Neuro Oncol. 2011;13(4):410-416.

Effective Treatments for DIPG are Lacking

- Standard-of-care treatment is palliative radiation (usually 54 Gy)
- Chemotherapy has no proven benefit
- Thus far, trials have not shown clinical benefit from currently available chemotherapy, radiosensitizing drugs, or biologics
- Due to their location in the brainstem, DIPGs cannot be surgically removed

DIPG, diffuse intrinsic pontine glioma.

Multimodal Chemo-radio-immunotherapy for DIPG Pilot Cohort

- First question: Could DIPG patients tolerate the indoximod immunotherapy regimen?
  - DIPG patients are often highly symptomatic
- Pilot cohort of 6 newly diagnosed DIPG patients
  - All 6 patients have finished upfront radiation combined with indoximod
  - All 6 patients showed initial improvement in symptoms
  - 3/6 later developed inflammatory symptoms (eg, waxing/waning, migratory)
    - 2 of these occurred during first cycle of temozolomide with indoximod

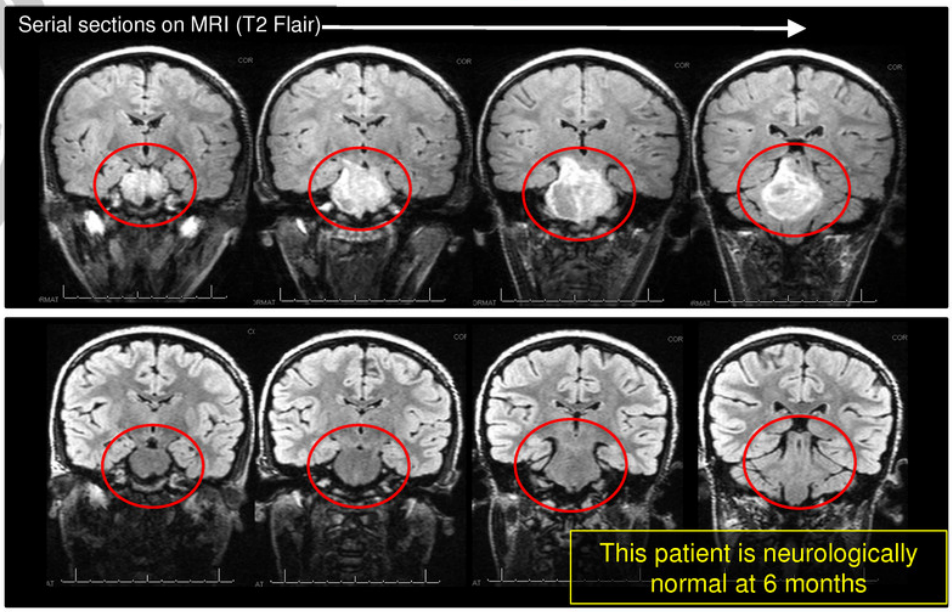
NLG2105-037: 9.4-Year-Old Male with Newly Diagnosed DIPG

Baseline (pretreatment)

DIPG scans reviewed by Tina Young-Poussaint, M.D., Boston Children's Hospital

Patient 037 classified as: "Significant response"

After 6 weeks of indoximod + radiation (54 Gy)

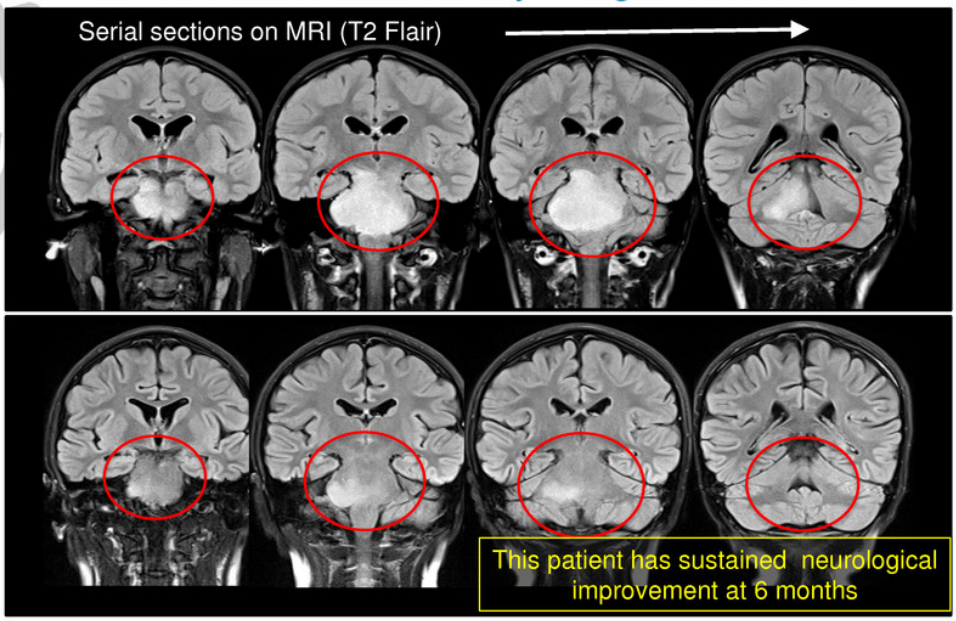


DIPG, diffuse intrinsic pontine glioma; MRI, magnetic resonance imaging..

NLG2105-035: 9.3-Year-Old Male with Newly Diagnosed DIPG

Baseline (pretreatment)

After 6 weeks of indoximod + radiation (54 Gy)

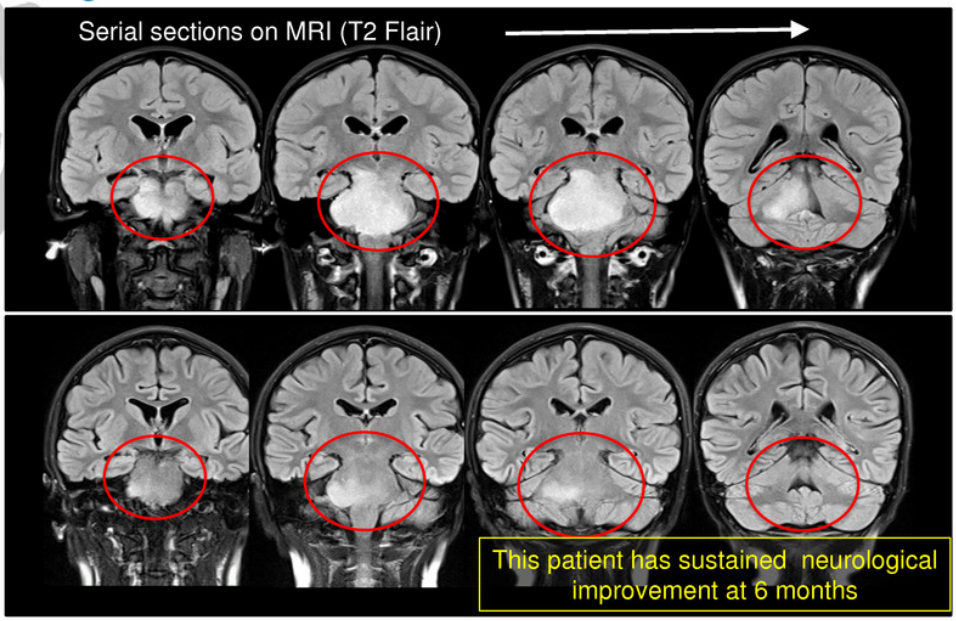


DIPG, diffuse intrinsic pontine glioma; MRI, magnetic resonance imaging..

Additional Newly Diagnosed DIPG Patients

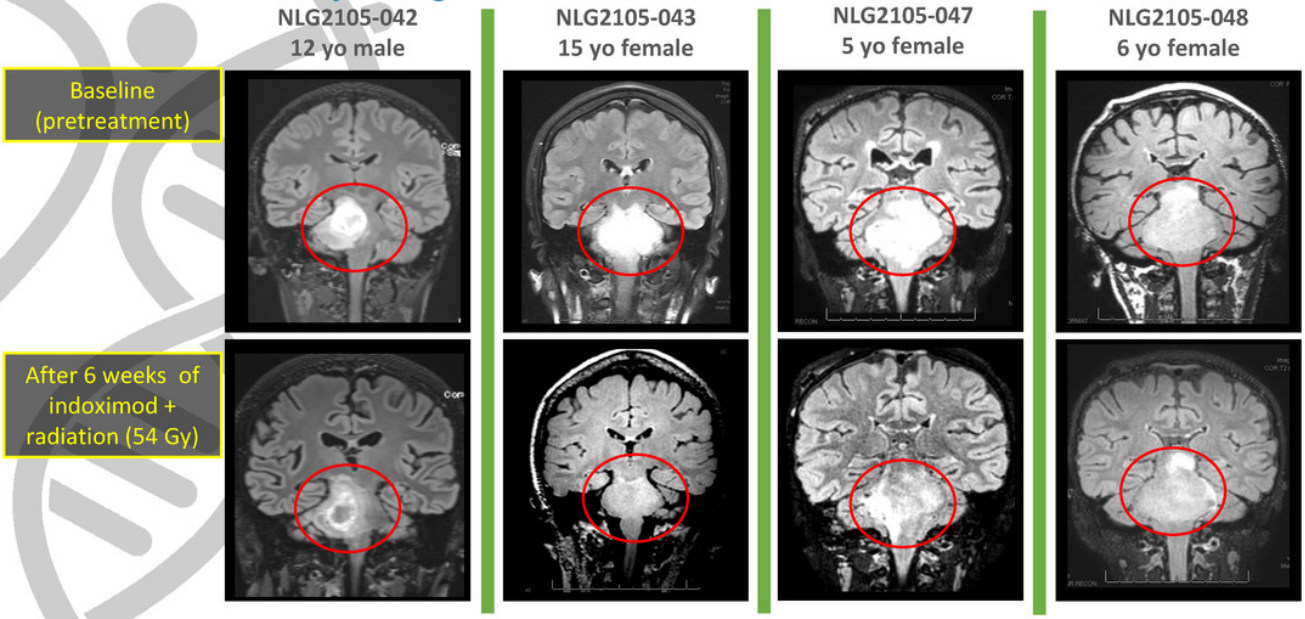
Baseline (pretreatment)

After 6 weeks of indoximod + radiation (54 Gy)



DIPG, diffuse intrinsic pontine glioma

Additional Newly Diagnosed DIPG Patients



DIPG, diffuse intrinsic pontine glioma

Conclusions and Future Directions

- Phase 1 data suggest that indoximod-based immunotherapy can allow disease responsiveness to conventional therapy (radiation, chemotherapy)
- Pilot cohort is under way applying this approach to newly diagnosed DIPG patients
- Phase 2 trial is planned