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): October 13, 2015

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))

Section 8 - Other Events

Item 8.01. Other Events.

On October 13, 2015, NewLink Genetics Corporation (the "Company") announced that a peer-reviewed article in *Cell Reports* offers new insight demonstrating that the tumor indoleamine 2,3-dioxygenase (IDO) pathway is a central regulator of both local and systemic immunosuppression and resistance to immunotherapy in melanoma.

The press release is attached hereto as Exhibit 99.1 and 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 October 13, 2015, entitled “New Study Published in *Cell Reports* Highlights Indoximod, Demonstrates the Key Role of IDO in Both Local and Systemic Immunosuppression”

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: October 13, 2015

NewLink Genetics Corporation

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

INDEX TO EXHIBITS

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New Study Published in *Cell Reports* Highlights Indoximod, Demonstrates the Key Role of IDO in Both Local and Systemic Immunosuppression

Data show NewLink Genetics' IDO inhibitor indoximod reverses tumor-associated suppression in pre-clinical models

Ames, IOWA - October 13, 2015 - NewLink Genetics Corporation (NASDAQ:NLNK), a biopharmaceutical company at the forefront of developing and commercializing novel immuno-oncology product candidates to improve the lives of patients with cancer, announced today that a peer-reviewed article in *Cell Reports* offers new insight demonstrating that the tumor indoleamine 2,3-dioxygenase (IDO) pathway is a central regulator of both local and systemic immunosuppression and resistance to immunotherapy in melanoma.

The paper also reports that indoximod, NewLink Genetics' wholly owned IDO pathway inhibitor, reversed tumor-associated immunosuppression in pre-clinical melanoma models and that there is a "strong rationale" for therapeutic targeting of IDO as one of the central regulators of immune suppression. The paper, titled *Tumor-Expressed IDO Recruits and Activates MDSCs in a Treg-Dependent Manner* and authored by Rikke B. Holmgaard, Dmitriy Zamarin, Yanyun Li, Billel Gasmi, David H. Munn, James P. Allison, Taha Merghoub, and Jedd D. Wolchok, was published on line and appears today in the October 13, 2015 issue of *Cell Reports*.

Rikke B. Holmgaard, Ph.D., lead author of the paper and Research Associate, Memorial Sloan Kettering Cancer Center, said, "We learned that IDO mediates local and systemic immune suppression in murine tumor models by means of other immunosuppressive cells, such as MDSCs and Tregs, and by upregulating other immunosuppressive mechanisms mediated by MDSCs, such as arginase 1, iNOS, and immunosuppressive cytokines. In so doing, IDO makes tumors grow faster and become less responsive to immune checkpoint therapy, such as PD1/CTLA4. Thus, adding IDO inhibition may be one of the key elements of combination therapies for strong, durable anti-tumor responses."

Jedd D. Wolchok, M.D., Ph.D., Chief of Melanoma and Immunotherapeutics Service at Memorial Sloan Kettering Cancer Center, Associate Professor of Immunology and Microbial Pathogenesis at Weill Cornell Medical College, and co-author of the article, added, "This data further demonstrate IDO as one of the key immune checkpoint targets and show that indoximod can block the expansion of suppressive myeloid cells and recruitment of MDSCs to the tumor microenvironment that mediate local immune suppression. It prevents activation of Tregs that mediate systemic immune suppression in a mouse model and enhances the antitumor activity of combination anti-CTLA4 and anti-PD1."

David H. Munn, M.D., Professor of Pediatric Hematology and Oncology at Georgia Regents University and co-author of the article in *Cell Reports*, added, "This research not only sheds additional light on the mechanism of IDO expression in tumors but also shows how indoximod can enhance anti-tumor responses when combined with other checkpoint inhibitors."

"This important work, both in animal studies and in analyses of human melanoma samples, by leading scientists in immuno-oncology validates our view of the central role IDO inhibition and indoximod may play in tumor immunosuppression," said Charles Link, M.D., CEO and Chief Scientific Officer of

NewLink Genetics. “It also points to the potential for IDO inhibitors like indoximod to overcome that immunosuppression and enhance anti-tumor responses.”

Indoximod is in Phase 2 clinical trials for breast cancer, prostate cancer, melanoma and glioblastoma multiforme as well as in Phase 1b/2 clinical trials for pancreatic cancer.

The authors’ analysis of tumor samples from 36 melanoma patients found that myeloid-derived suppressor cells (MDSCs) were increased in IDO+ tumors. This suggests that IDO recruits and activates such cells through activation of regulatory T cells (Tregs). The authors found greater tumor growth, as well as resistance to immune checkpoint blockade, in the tumors overexpressing IDO. Treatment of the mice bearing tumors overexpressing IDO with the IDO pathway inhibitor indoximod reversed this tumor-associated immunosuppression by decreasing the number of MDSCs and Tregs and stopping their suppressive capability.

About NewLink Genetics Corporation

NewLink is a biopharmaceutical company focused on discovering, developing and commercializing novel immuno-oncology products to improve treatment options for patients with cancer. NewLink Genetics’ portfolio includes biologic and small molecule immunotherapy product candidates intended to treat a wide range of oncology indications. NewLink Genetics’ product candidates are designed to harness multiple components of the immune system to combat cancer without significant incremental toxicity, either as a monotherapy or in combination with other treatment regimens. For more information, please visit http://www.newlinkgenetics.com.

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 facts, 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 NewLink Genetics’ financial guidance for 2015; enrollment in or 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 Genetics 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, 2014 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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