UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
WASHINGTON, DC 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 1, 2019

CytoDyn Inc.
(Exact name of registrant as specified in its charter)

Delaware	**000-49908**	**83-1887078**
(State or other jurisdiction of incorporation)	**(SEC File Number)**	**(I.R.S. Employer Identification No.)**

1111 Main Street, Suite 660 **Vancouver, Washington**	**98660**
(Address of principal executive offices)	**(Zip Code)**

Registrant's telephone number, including area code: (360) 980-8524

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 ☐

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. ☐

Item 7.01. Regulation FD Disclosure.

On April 1, 2019, CytoDyn Inc., a Delaware corporation (the "Company") issued a press release announcing data from studies presented at the American Association of Cancer Research Annual Meeting taking place from March 29, 2019 to April 3, 2019 in Atlanta, Georgia. Copies of the press release and the presentation by the Company are furnished as Exhibit 99.1 and 99.2, respectively, to this Form 8-K.

Item 9.01 Financial Statements and Exhibits.

(d)	Exhibit No.	Description
	99.1	Press Release, dated April 1, 2019.
	99.2	Investor Presentation.

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.

CytoDyn Inc.

April 1, 2019

By: /s/ Michael D. Mulholland
Name: Michael D. Mulholland
Title: Chief Financial Officer

CytoDyn Announces Data From Two Studies at the AACR Annual Meeting

Studies Demonstrate the Expression of CCR5 as a Novel Biomarker in Circulating Tumor Cells of HER2 metastatic Breast Cancer (mBC) Patients and the Ability of CytoDyn's Leronlimab (PRO 140), a CCR5 Inhibitor, to Block mBC

VANCOUVER, Washington, April 1, 2019 – CytoDyn Inc. (OTC.QB: CYDY), ("CytoDyn" or the "Company") a late stage biotechnology company developing leronlimab (PRO 140), a CCR5 antagonist with the potential for multiple therapeutic indications, highlighted two studies that will be presented on April 1st at the American Association of Cancer Research (AACR) Annual Meeting taking place March 29 to April 3 in Atlanta, Georgia.

The first study will present data showing the expression of CCR5 in circulating tumor cells (CTCs) of HER2 metastatic breast cancer (mBC) patients and documents the correlation between CCR5 and HER2 expression in CTCs. The second study will highlight murine xenograft data showing that CytoDyn's humanized monoclonal antibody, leronlimab (PRO 140), can effectively block human breast cancer metastases and enhance the cell killing ability of DNA damaging chemotherapy by selectively targeting the CCR5 receptor.

"The key discoveries being announced today potentially contribute to a shift in the treatment paradigm for metastatic breast cancer patients," stated Professor Richard G. Pestell, M.D., Ph.D., M.B.A., F.A.C.P., F.R.A.C.P., Chief Medical Officer and Vice Chairman of CytoDyn. "There are three key findings from today's presentations. First, we now know that CCR5 is overexpressed on CTCs of HER2 metastatic breast cancer patients. Second, we now show in xenograft models that leronlimab (PRO 140) can effectively block CCR5 positive breast cancer metastasis," continued Dr. Pestell. "And, third, we have now shown in murine xenograft models that by reducing the ability of breast cancer cells to metastasize, thereby keeping the tumor more contained, leronlimab can potentially provide standard DNA damaging chemotherapies more time to work. Potentially providing significantly improved efficacy of existing cancer therapies with fewer side effects," concluded Dr. Pestell. CytoDyn has now opened a clinical trial (NCT03838367) that is assessing the efficacy of leronlimab (PRO 140) in metastatic triple negative breast cancer and will measure CTCs in the enrolled patients.

About Leronlimab (PRO 140)

The U.S. Food and Drug Administration (FDA) has granted a "fast track" designation to leronlimab (PRO 140) as a combination therapy with HAART for HIV-infected patients. Leronlimab (PRO 140) is an investigational humanized IgG4 mAb that blocks CCR5, a cellular receptor that appears to play multiple roles with implications in HIV infection, tumor metastases and immune signaling. Leronlimab (PRO 140) has successfully completed nine Phase 1/2/3 clinical trials in over 700 people, including a successful pivotal Phase 3 trial in combination with standard anti-retroviral therapies in HIV-infected treatment-experienced patients.

In the setting of HIV/AIDS, leronlimab (PRO 140) belongs to a new class of therapeutics called viral-entry inhibitors; it masks CCR5, thus protecting healthy T cells from viral infection by blocking the predominant HIV (R5) subtype from entering those cells. Leronlimab (PRO 140) has been the subject of nine clinical trials, each of which demonstrated that leronlimab can significantly reduce or control HIV viral load in humans. The leronlimab (PRO 140) antibody appears to be a powerful antiviral agent leading to potentially fewer side effects and less frequent dosing requirements compared with daily drug therapies currently in use.

In the setting of cancer, research has shown that CCR5 likely plays a central role in tumor invasion and metastasis and that increased CCR5 expression is an indicator of disease status in several cancers. Moreover, research has shown that drugs that block CCR5 can block tumor metastases in laboratory and animal models of aggressive breast and prostate cancer. CytoDyn is conducting additional research with leronlimab (PRO 140) in the cancer setting and plans to initiate additional Phase 2 human clinical trials, in addition to triple-negative breast cancer, when appropriate.

The CCR5 receptor also appears to play a central role in modulating immune cell trafficking to sites of inflammation and may be crucial for the development of acute graft-versus-host disease (GvHD) and other inflammatory conditions. Clinical studies by others further support the concept that blocking CCR5 using a chemical inhibitor can reduce the clinical impact of acute GvHD without significantly affecting the engraftment of transplanted bone marrow stem cells. CytoDyn is currently conducting a Phase 2 clinical study with leronlimab (PRO 140) to further support the concept that the CCR5 receptor on engrafted cells is critical for the development of acute GvHD and that blocking this receptor from recognizing certain immune signaling molecules is a viable approach to mitigating acute GvHD. The FDA has granted “orphan drug” designation to leronlimab (PRO 140) for the prevention of graft-versus-host disease (GvHD).

About CytoDyn

CytoDyn is a biotechnology company developing innovative treatments for multiple therapeutic indications based on leronlimab (PRO 140), a novel humanized monoclonal antibody targeting the CCR5 receptor. CCR5 appears to play a key role in the ability of HIV to enter and infect healthy T-cells. The CCR5 receptor also appears to be implicated in tumor metastasis and in immune-mediated illnesses, such as graft-vs-host disease (GvHD) and NASH. CytoDyn has successfully completed a Phase 3 pivotal trial with leronlimab (PRO 140) in combination with standard anti-retroviral therapies in HIV-infected treatment-experienced patients. CytoDyn plans to seek FDA approval for leronlimab (PRO 140) in combination therapy and plans to complete the filing of a Biologics License Application (BLA) in 2019 for that indication. CytoDyn is also conducting a Phase 3 investigative trial with leronlimab (PRO 140) as a once-weekly monotherapy for HIV-infected patients and, plans to initiate a registration-directed study of leronlimab monotherapy indication, which if successful, could support a label extension. Clinical results to date from multiple trials have shown that leronlimab (PRO 140) can significantly reduce viral burden in people infected with HIV with no reported drug-related serious adverse events (SAEs). Moreover, results from a Phase 2b clinical trial demonstrated that leronlimab (PRO 140) monotherapy can prevent viral escape in HIV-infected patients, with some patients on leronlimab monotherapy remaining virally suppressed for more than four years. CytoDyn is also conducting a Phase 2 trial to evaluate leronlimab (PRO 140) for the prevention of GvHD and has received clearance to initiate a clinical trial with leronlimab (PRO 140) in metastatic triple-negative breast cancer. More information is at www.cytodyn.com.

Forward-Looking Statements

This press release contains certain forward-looking statements that involve risks, uncertainties and assumptions that are difficult to predict. Words and expressions reflecting optimism, satisfaction or disappointment with current prospects, as well as words such as “believes,” “hopes,” “intends,” “estimates,” “expects,” “projects,” “plans,” “anticipates” and variations thereof, or the use of future tense, identify forward-looking statements, but their absence does not mean that a statement is not forward-looking. The Company’s forward-looking statements are not guarantees of performance, and actual results could vary materially from those contained in or expressed by such statements due to risks and uncertainties including: (i) the sufficiency of the Company’s cash position, (ii) the Company’s ability to raise additional capital to fund its

operations, (iii) the Company's ability to meet its debt obligations, if any, (iv) the Company's ability to enter into partnership or licensing arrangements with third parties, (v) the Company's ability to identify patients to enroll in its clinical trials in a timely fashion, (vi) the Company's ability to achieve approval of a marketable product, (vii) the design, implementation and conduct of the Company's clinical trials, (viii) the results of the Company's clinical trials, including the possibility of unfavorable clinical trial results, (ix) the market for, and marketability of, any product that is approved, (x) the existence or development of vaccines, drugs, or other treatments that are viewed by medical professionals or patients as superior to the Company's products, (xi) regulatory initiatives, compliance with governmental regulations and the regulatory approval process, (xii) general economic and business conditions, (xiii) changes in foreign, political, and social conditions, and (xiv) various other matters, many of which are beyond the Company's control. The Company urges investors to consider specifically the various risk factors identified in its most recent Form 10-K, and any risk factors or cautionary statements included in any subsequent Form 10-Q or Form 8-K, filed with the Securities and Exchange Commission. Except as required by law, the Company does not undertake any responsibility to update any forward-looking statements to take into account events or circumstances that occur after the date of this press release.

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Leronlimab, a humanized monoclonal antibody to CCR5, blocks breast cancer metastasis and enhances cell death induced by DNA damaging chemotherapy

Exhibit 99.2

Xuanmao Jiao[1], Min Wang[1], Zhiping Li[1], Richard G. Pestell[1, 2, 3, *]

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[2]CytoDyn Inc., 1111 Main St, Suite 660, Vancouver, WA, 98660. [3]Lee Kong Chian School of Medicine, Nanyang Technological University, 637551, Singapore.

Abstract

Purpose of the study. To assess binding and functional interaction of the humanized monoclonal antibody to CCR5 (Leronlimab) with human breast cancer cell lines. The G protein coupled receptor CCR5, is normally expressed on a subset of T cells and serves as a co-receptor for HIV infection. During malignant transformation CCR5 expression is known to increase in a number of cancers (breast cancer (BCa), prostate cancer, colon cancer, melanoma). CCR5 targeted cancer clinical trials using small molecular inhibitors opened to accrual in late 2018. CCR5 is expressed in >50% of human BCa, primarily in triple negative BCa. Its expression in human BCa correlates with poor outcome and CCR5^{+} BCa epithelial cells have characteristics of cancer stem cells, forming mammospheres and initiating tumors with >60-fold greater efficiency in mice. Reintroduction of CCR5 expression into CCR5 negative BCa cells promotes tumor metastases and induces DNA repair gene expression and activity. The CCR5 inhibitor Leronlimab has been used for treatment of >660 patients with HI, including meeting its primary endpoints in a phase III study, without significant adverse events reported. **Results.** Leronlimab bound to CCR5 expressed in human breast cancer cell lines with 98% efficiency. Leronlimab abrogated CCL5 induced Ca^{+2} flux and blocked 3-d matrigel invasion of MDA-MB-231 cells. Leronlimab blocks human breast cancer xenograft metastasis in mice. Leronlimab also augmented cell killing by DNA damage inducing agents including Doxorubicin. **Conclusions.** Leronlimab binds CCR5 in BCa cells, blocking breast cancer cellular invasion and tumor metastasis, and augmenting cell killing by DNA damage inducing chemotherapies. As CCR5 augments DNA repair and is expressed selectively on cancerous but not normal breast epithelial cells, Leronlimab may enhance the tumor specific activities of DDR-based treatments, allowing a reduction in dose of chemotherapy and radiation.

1.The binding of Leronlimab with CCR5 expressed in breast cancer cells

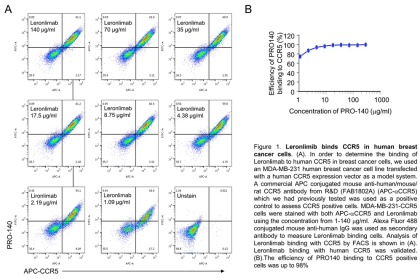

Figure 1. **Leronlimib binds CCR5 in human breast cancer cells.** (A). In order to determine the binding of Leronlimab to human CCR5 in breast cancer cells, we used an MDA-MB-231 human breast cancer cell line transfected with a human CCR5 expression vector as a model system. A commercial APC conjugated mouse anti-human/mouse/ rat CCR5 antibody from R&D (FAB1802A) (APC-αCCR5) which we had previously tested was used as a positive control to assess CCR5 positive cells. MDA-MB-231-CCR5 cells were stained with both APC-αCCR5 and Leronlimab using the concentration from 1-140 µg/ml. Alexa Fluor 488 conjugated mouse anti-human IgG was used as secondary antibody to measure Leronlimab binding cells. Analysis of Leronlimab binding with CCR5 by FACS is shown in (A). Leronlimab binding with human CCR5 was validated. (B).The efficiency of PRO140 binding to CCR5 positive cells was up to 98%

2. The effects of PRO140 on CCL5 induced Ca^{2+} responses in MDA-MB-231-CCR5 cells

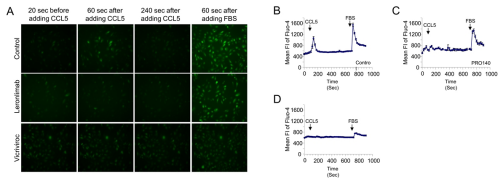

Figure 2. **PRO140 blocks human CCR5-mediated signaling in human breast cancer cells.** CCR5 activation induces calcium flux (Mueller et al., 2002; Petkovic et al., 2004). To assess the effects of Leronlimab on CCR5 function, we measured the calcium responses induced by CCL5 in MDA-MB-231-CCR5 cells with or without Leronlimab by living cell image (Figure 2A-C). Fluo-4 was used as calcium concentration indicator. The CCR5 antagonist, Vicriviroc, was used as positive control (Figure 2A, D). The results showed that Leronlimab can block CCL5 induced calcium responses in MDA-MB-231-CCR5 cells (1.23±0.10, N=10 for control cells and 0.54±0.13 N=12 for PRO140 treated cells. P<0.001 at calcium peak induce by CCL5).

3. Leronlimab blocks breast cancer cell 3D-matrigel invasion

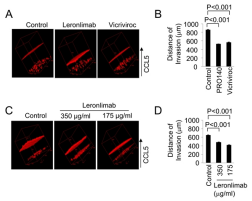

Figure 3. **Leronlimab blocks CCR5 mediated invasion of human breast cancer cells into extracellular matrix.** The ability of breast cancer cells to invade extra-cellular matrix is distinguishable from but an important step in tumor metastasis (Zetter, 1990). To test the ability of PRO140 to block 3D-matrigel invasion assay, MDA-MB-231 cells were used. CCL5 was used as chemoattractant to induce invasion. The small molecule inhibitor of CCR5, Vicriviroc, was used as a form of positive control. Leronlimab reduced CCL5-induced MDA-MB-231 breast cancer cell invasion with similar efficacy as Vicriviroc (A, B) (855±9, N=8 for control vs 855±9, N=9 for Leronlimab, P <0.001). We also tested the effects of different dose of Leronlimab on breast cancer cell invasion and the results showed that both 175 and 350 µg/ml of Leronlimab can effectively block MDA-MB-231 cell invasion (C, D).

4. Leronlimab blocks breast cancer cell metastasis in a mouse lung metastasis model

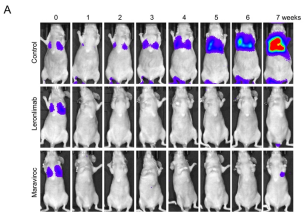

Figure 4. Leronlimab block breast cancer metastasis in mice. The mice were divided into 4 groups (control, Leronlimab, Maraviroc and vicriviroc) randomly. MDA-MB-231 cells stable transfected with Luc2-GFP was injected into the mice through tail-vein. The mice in each group were treated one day before injection. The metastasis tumor formed in the lung was determined by bioluminescence imaging. The bioluminescence images of the representative mice from control, Leronlimab and Maraviroc group were showed in (A). The quantitative analysis of tumor size in each group was showed in (B). The size of tumors defined by photon flux (x10^{5} p/sec/cm^{2}/sr). The data was showed as Mean ± SE. Leronlimab dramatically decreased breast cancer tumor metastasis to the lung.

5. *Leronlimab enhances cell death induced by Doxorubicin*

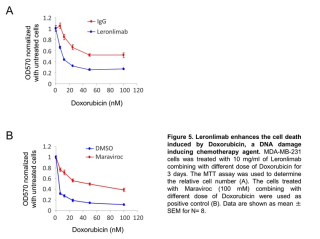

Figure 5. Leronlimab enhances the cell death induced by Doxorubicin, a DNA damage inducing chemotherapy agent. MDA-MB-231 cells was treated with 10 mg/ml of Leronlimab combining with different dose of Doxorubicin for 3 days. The MTT assay was used to determine the relative cell number (A). The cells treated with Maraviroc (100 nM) combining with different dose of Doxorubicin were used as positive control (B). Data are shown as mean ± SEM for N= 8.