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CytoDyn Presents New Leronlimab Data in Metastatic Triple-Negative Breast Cancer at AACR Annual Meeting 2026

Translational study in metastatic triple-negative breast cancer (mTNBC) shows leronlimab-mediated CCR5 inhibition induces PD-L1 expression, modulates immune checkpoint signaling, and is associated with long-term survival in heavily pretreated patients

VANCOUVER, Washington, April 20, 2026 (GLOBE NEWSWIRE) -- **CytoDyn Inc. (OTCQB: CYDY)** ("CytoDyn" or the "Company"), a clinical-stage oncology company advancing leronlimab, a first-in-class humanized monoclonal antibody targeting the CCR5 receptor with therapeutic potential across multiple indications, including metastatic triple-negative breast cancer (“mTNBC”) and colorectal cancer (“mCRC”), today announced that new clinical and translational data in metastatic triple-negative breast cancer (mTNBC) were presented at the **AACR Annual Meeting 2026**, taking place April 17–22, 2026, at the **San Diego Convention Center**.

The presentation highlighted emerging evidence supporting CCR5 inhibition with leronlimab as a strategy to modulate the tumor microenvironment, enhance immune responsiveness, and improve outcomes in metastatic triple-negative breast cancer (mTNBC).

Metastatic triple-negative breast cancer remains an aggressive disease with limited treatment options and poor long-term survival. While immune checkpoint inhibitors (ICIs) have demonstrated benefit in select patients, many tumors exhibit low PD-L1 expression and resistance to immunotherapy. Preclinical and clinical findings presented at AACR show that CCR5 inhibition with leronlimab modulates immune checkpoint signaling, potentially sensitizing tumors to immune checkpoint inhibitor therapy.

“Our findings suggest that CCR5 plays a key role in immune exhaustion and therapy resistance pathways in TNBC,” said Richard G. Pestell, M.D., Ph.D., FRCP, AO, Lead Consultant in Preclinical and Clinical Oncology at CytoDyn. “Induction of PD-L1 predicts response to immune checkpoint therapy. Leronlimab-mediated CCR5 inhibition induced PD-L1 expression and reduced key mediators of immune suppression, including sB7-H3 and sTyro3 signaling. These data support the hypothesis that leronlimab may help prime tumors for immune checkpoint therapy and improve clinical outcomes in patients with otherwise limited therapeutic options.”

Key findings from baseline tumor biology and leronlimab treatment analysis in TNBC include:

- Across breast cancer cohorts (N=1,096), CCR5 expression correlated with gene signatures of T-cell immune exhaustion and immune infiltration.
- CCR5 expression was enriched in TNBC subtypes associated with immune modulation, including mesenchymal-like immune-altered (MLIA) and

immunomodulatory (IM) subtypes.

- In TNBC cell models, CCR5 inhibition with leronlimab increased PD-L1 expression, suggesting a potential mechanism to enhance responsiveness to PD-L1-targeted therapies.
- Proteomic analyses demonstrated that CCR5 activity promotes expression of immune checkpoint mediators, including sB7-H3 (CD276) and Tyro3 signaling, both associated with resistance to ICIs; these effects were attenuated with leronlimab.
- In a retrospective analysis of 28 patients with mTNBC, leronlimab treatment was associated with induction of PD-L1 in circulating tumor cells (CTCs) and cancer-associated macrophage-like cells (CAMLs).
- Higher leronlimab dose, PD-L1 induction, and use in combination or sequence with ICIs were associated with improved patient survival outcomes.
- Notably, 17.9% (5/28) of heavily pretreated patients remain alive after more than 60 months of follow-up.

"These results reinforce the potential role of CCR5 as a critical regulator of the tumor microenvironment in TNBC," said Jacob P. Lalezari, M.D., Chief Executive Officer of CytoDyn. "The observed induction of PD-L1 and association with long-term survival in mTNBC support continued clinical development of leronlimab in combination approaches designed to enhance immune response and overcome treatment resistance."

Dr Pestell is the first author of the poster presentation titled *"Leronlimab induces PD-L1 expression and is associated with long term survival with an ICI in PD-L1 low metastatic TNBC"* on April 19, 2026, from 2:00 p.m. – 5:00 p.m. PT (Poster #1033). A copy of the poster will be made available on CytoDyn's website under the Publications & Posters section.

About CytoDyn

CytoDyn is a clinical-stage oncology company dedicated to advancing leronlimab, a first-in-class humanized monoclonal antibody that targets the CCR5 receptor, a key regulator of immune function implicated in cancer, infectious diseases, and autoimmune disorders. Guided by a mission to improve patients' quality of life through therapeutic innovation, CytoDyn is committed to integrity, responsibility, and service as it works to bring transformative treatments to patients worldwide.

For more information, please visit www.cytodyn.com and follow us on LinkedIn.

Note Regarding Forward-Looking Statements

This news release may contain forward-looking statements relating to, among other things, the mechanism of action of leronlimab, clinical trial results, product development, market position, future operating and financial performance, and business strategy. The reader is cautioned not to rely on these statements, which are based on current expectations of future events. For important information about these statements and our Company, including the risks, uncertainties and other factors that could cause actual results to vary materially from the assumptions, expectations and projections expressed in any forward-looking statements, the reader should review our Annual Report on Form 10-K for the fiscal year ended May 31, 2025, including the section captioned "Forward-Looking Statements" and in Item 1A, as well as subsequent reports filed with the Securities and Exchange Commission. CytoDyn Inc. does not undertake to update any forward-looking statement as a result of new information

or future events or developments except as required by applicable law.

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