

CytoDyn Presents Novel CCR5 Inhibition Mechanisms and Long-Term Survival Signals for Leronlimab in Metastatic Breast Cancer at AACR Immuno-Oncology Conference

Leronlimab-mediated CCR5 blockade modulates T-Cell exhaustion pathways and PD-L1 Biology relevant to ICI response

Durable Clinical Observations and Favorable Safety Profile Observed in Heavily Pretreated mTNBC Patients

VANCOUVER, Washington, Feb. 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 preclinical, translational, and clinical data supporting leronlimab's proposed role in the treatment of metastatic triple-negative breast cancer was presented at the [AACR Immuno-Oncology Conference \(AACR IO\)](#), held February 18-21, 2026 in Los Angeles, California.

Leronlimab is being evaluated for its ability to modulate the tumor immune microenvironment by targeting the CCR5 receptor, a key regulator of immune function implicated in cancer progression and immune resistance. The data presented explore mechanisms of immune checkpoint resistance in TNBC and offer insight into how CCR5 blockade may enhance responses to immune checkpoint inhibitors ("ICIs").

"In this study, we continued to explore how CCR5 signaling may contribute to immune checkpoint resistance in metastatic triple-negative breast cancer by integrating patient-derived datasets with molecular, cellular, and translational analyses involving leronlimab," said Professor Richard Pestell, M.D., Ph.D., FRCP, AO, Lead Consultant in Preclinical and Clinical Oncology at CytoDyn. "Mechanistic findings suggest that CCR5 blockade with leronlimab may modulate pathways associated with T-cell exhaustion and PD-L1/PD1 regulation, providing a mechanistic rationale for combination strategies with immune checkpoint inhibitors."

Key findings:

- Across multiple TNBC patient gene expression datasets, CCR5 expression correlated with elevated cytotoxic T-lymphocyte signatures and T-cell exhaustion profiles, identifying immune states potentially amenable to CCR5 inhibition.
- PD-L1 and PD1, the abundance of which correlates with improved response to

immune checkpoint therapies, was increased by leronlimab.

- In cell culture and histology analyses, CCR5 inhibition with leronlimab increased the abundance of PD-L1 in breast cancer cells.
- In Rhesus monkeys, leronlimab induced PD1 in T cells.
- CCR5 activity was associated with the secretion of immunosuppressive mediators from triple-negative breast cancer cells, [sB7-H3 (CD276), BAFF (sTNFSF13B), and sTyro3], which were significantly reduced following leronlimab treatment.
- In a pooled retrospective clinical analysis of 28 heavily pretreated patients with metastatic triple-negative breast cancer from three clinical trials, leronlimab demonstrated a favorable safety profile with no therapy-limiting toxicities, and 5 of 28 patients (17.9%) remain alive after a median follow-up of more than 63 months.

“These data strengthen the clinical case for leronlimab in metastatic TNBC by aligning mechanistic insights with encouraging safety and long-term survival observations, while adding important clinical perspective to the emerging role of CCR5 inhibition in this disease,” said Dr. Jacob Lalezari, M.D., Chief Executive Officer of CytoDyn. “The consistency of the mechanistic and clinical findings supports our decision to continue the clinical development and investigation of leronlimab, including further evaluation in combination immunotherapy settings.”

Associate Professor Xuanmao Jiao of the Pennsylvania Cancer and Regenerative Medicine Center at the Baruch S. Blumberg Institute presented the poster, titled “**Leronlimab is associated with long-term survival in metastatic TNBC: Enhancing PD-L1 expression, ICI response, and the role of T cell exhaustion,**” on February 19, 2026, from 12:15 p.m. – 3:25 p.m. PST. 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.

Corporate Contact

CytoDyn Inc.

ir@cytodyn.com

Media Contacts

David Schull or Ignacio Guerrero-Ros, Ph.D.

Russo Partners, LLC

CytoDyn@russopartnersllc.com



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