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): December 07, 2022 (December 07, 2022)

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

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

1111 Main Street, Suite 660
Vancouver, Washington 98660
(Address of principal executive offices, including zip code)

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

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:

☐ 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))

Securities registered pursuant to Section 12(b) of the Act:

Title of each class	Trading Symbol(s)	Name of each exchange on which registered
None	**None**	**None**

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 December 7, 2022, CytoDyn Inc. (the "Company") made a presentation during its R&D Update. The presentation has been posted on the Company's website and a copy of the presentation is furnished as Exhibit 99.1 to this report and incorporated herein by reference.

Item 9.01 Financial Statements And Exhibits.
(d) Exhibits.

99.1	Investor Presentation dated December 7, 2022**
Exhibit 104	The cover page from this Current Report on Form 8-K formatted in Inline XBRL.

** Furnished, not filed.

SIGNATURE

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 thereunto duly authorized.

CYTODYN INC.

Date: December 7, 2022

By /s/ Antonio Migliarese
Antonio Migliarese
Chief Financial Officer


CytoDyn
CytoDyn – R&D Investor Update
December 7, 2022
1

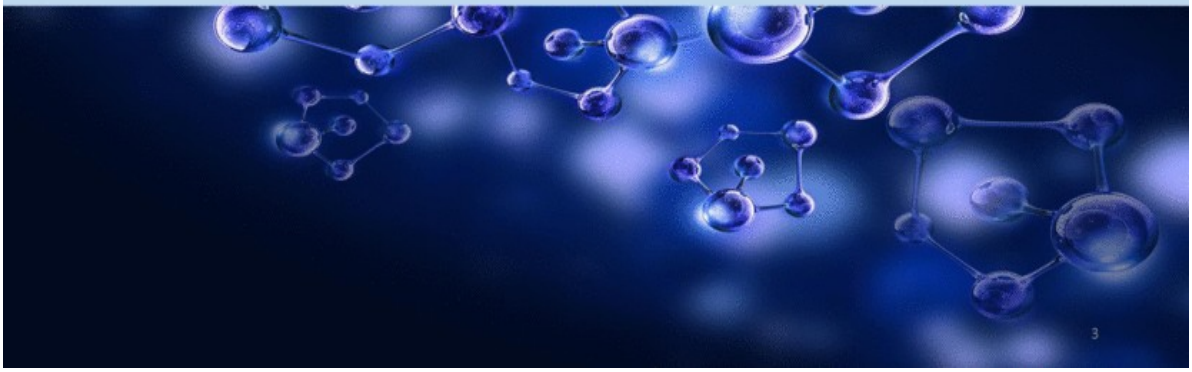
CytoDyn Forward Looking Statements

This presentation 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. Forward-looking statements may include statements about leronlimab, its ability to provide positive health outcomes, the Company's ability to develop a successful operating strategy and thereby create shareholder value, the possible results of clinical trials, studies or other programs or ability to continue those programs, the ability to obtain regulatory approval for commercial sales, and the market for actual commercial sales. 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 regulatory determinations of leronlimab's safety and effectiveness to treat the diseases and conditions for which we are studying the product by the U.S. Food and Drug Administration (FDA) and various drug regulatory agencies in other countries; (ii) the Company's ability to raise additional capital to fund its operations; (iii) the Company's ability to meet its debt and other payment obligations; (iv) the Company's ability to retain other key employees; (v) the Company's ability to enter into partnership or licensing arrangements with third-parties; (vi) the timely and sufficient development, through internal resources or third-party consultants, of analyses of the data generated from the Company's clinical trials required by the FDA or other regulatory agencies in connection with applications for approval of the Company's drug product; (vii) the Company's ability to achieve approval of a marketable product; (viii) the design, implementation and conduct of the Company's clinical trials; (ix) the results of the Company's clinical trials, including the possibility of unfavorable clinical trial results; (x) the market for, and marketability of, any product that is approved; (xi) 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; (xii) regulatory initiatives, compliance with governmental regulations and the regulatory approval process; (xiii) legal proceedings, investigations or inquiries affecting the Company or its products; (xiv) general economic and business conditions; (xv) changes in foreign, political, and social conditions; (xvi) stockholder actions or proposals with regard to the Company, its management, or its board of directors; and (xvii) 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 subsequent Form 10-Qs and Form 8-Ks, 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 presentation.

CytoDyn

Welcome

Cyrus Arman, PhD MBA – President



CytoDyn Agenda & Scientific Advisory Board Speakers

Welcome and Introduction | Cyrus Arman (President)

Company Overview and Background

Clinical Development Programs

Clinical and Preclinical Data in Prioritized Therapeutic Areas | Scientific Advisory Board Members

Dr. Mazen Noureddin

Dr. Stefan Glück

Dr. Jonah Sacha

CytoDyn Scientific Advisory Board Speakers

Dr. Mazen Noureddin

- Director of the Houston Liver Institute and former founding Director of the Fatty Liver Program at Cedars-Sinai Medical Center
- Conducted over 40 investigational clinical studies and published over 180 papers on NASH
- Will present the potential role of leronlimab in treating NASH and non-alcoholic fatty liver disease

Dr. Stefan Glück

- Former Sylvester Professor at the Leonard M. Miller School of Medicine at the University of Miami at Florida and has been a Principal Investigator for over 37 breast cancer clinical studies
- Served in roles for Regeneron and Celgene, focusing on IO in solid tumors and acquiring early assets
- Will discuss the potential role of leronlimab in the tumor microenvironment

Dr. Jonah Sacha

- Professor at the Vaccine and Gene Therapy Institute and Oregon National Primate Research Center, both of OHSU, where he conducts research on infectious diseases
- Published over 85 peer-reviewed research articles and has been awarded 10 patents
- Will share the latest research developments with regard to leronlimab and HIV cure

CytoDyn SAB Disclosures

- **Dr. Mazen Noureddin**
  - **Advisory Board**: Altimmune, BI, BMS, 89BIO, EchoSens, Gilead, CytoDyn, GSK, Merck, Novo Nordisk, OWL, Pfizer, Roche diagnostic and Siemens, Terns and Takeda
  - **Principal Investigator for a Drug Study**: Allergan, Akero, BMS, Gilead, Galectin, Genfit, Conatus,Corcept, Enanta, Madrigal, Novartis, Novo Nordisk, Shire, Terns, Viking and Zydus
  - **Stockholder**: Anaetos, Rivus Pharma, CIMA, ChronWell and Viking

- **Dr. Stefan Glück**
  - **Advisory Board**: CytoDyn

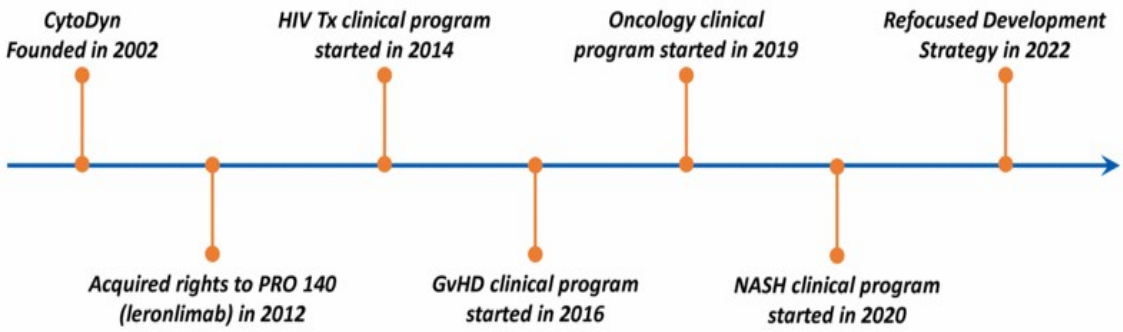
- **Dr. Jonah Sacha**
  - **Advisory Board**: CytoDyn, Vir, Sana, Mabloc

CytoDyn Company Overview

CytoDyn's Mission

CytoDyn Company Overview

- **Headquarters:** Vancouver, WA
- **Fulltime Employees**: 15 (December 2022)
- **Ticker:** CYDY



CytoDyn's Board of Directors

Tanya Urbach

Stephen M. Simes

Ryan Dunlap

Dr. Karen Brunke

Dr. Lishomwa Ndhlovu

CytoDyn's Manufacturing Partners and Publications

Manufacturing Partners

AGC Biologics

SAMSUNG BIOLOGICS

Recent Conference Abstracts

SAN ANTONIO BREAST CANCER SYMPOSIUM

EASL The Home of Hepatology

AASLD American Association for the Study of Liver Diseases

AACR American Association for Cancer Research FINDING CURES TOGETHER

Leronlimab Publications (10+)

biomedicines

nature COMMUNICATIONS

frontiers in Immunology

The Journal of Infectious Diseases

PLOS PATHOGENS

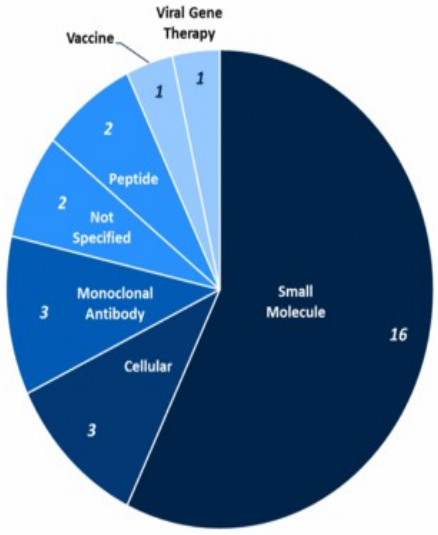
CytoDyn

CCR5 & Leronlimab

CytoDyn The CCR5/CCL5 Axis & Leronlimab

- **Agents directed against the CCL5/CCR5 axis have been studied in many therapeutic areas such as inflammation, cancers, viral infections, and immune responses**
  - In different conditions, the function of the CCL5/CCR5 axis varies, and has implications for infectious diseases, cell proliferation, migration, angiogenesis, metastasis, survival, and autoimmune responses
  - The precise binding location of the molecule on the receptor can also have important downstream effects on immune function and signaling
- **Leronlimab is a humanized IgG4 monoclonal antibody directed against chemokine receptor 5 (CCR5)**
  - Leronlimab was first discovered to bind to the extracellular domains of CCR5 in 1999
  - Leronlimab is a once-a-week subcutaneous injection which may also be administered in IV form
  - Leronlimab received Fast-Track Designation for mTNBC and HIV Treatment
  - Leronlimab is an investigational new drug and is not yet approved or authorized by FDA or any other regulatory authority for any indication

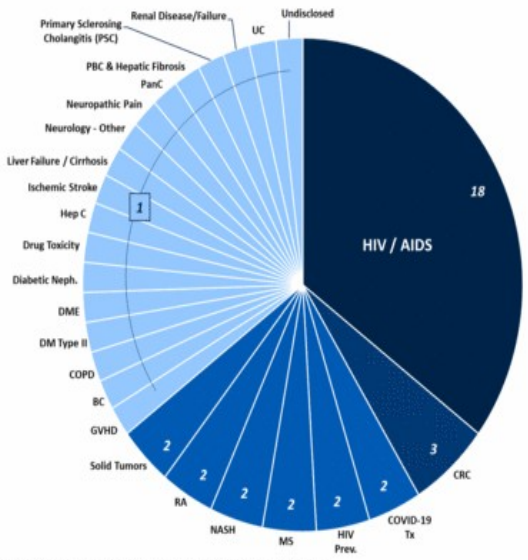
CytoDyn CCR5 Inhibitors – Competitive Landscape



- There are a total of **28** unique assets that target CCR5
- Monoclonal antibodies:
  - Leronlimab (SC)
  - *HGS004 (IV – suspended)*
  - *HGS1025 (IV – suspended)*
- To date only Selzentry (maraviroc) has been approved (for HIV Tx)

***Source**: BioMedTracker (July 2022), CytoDyn Analysis*

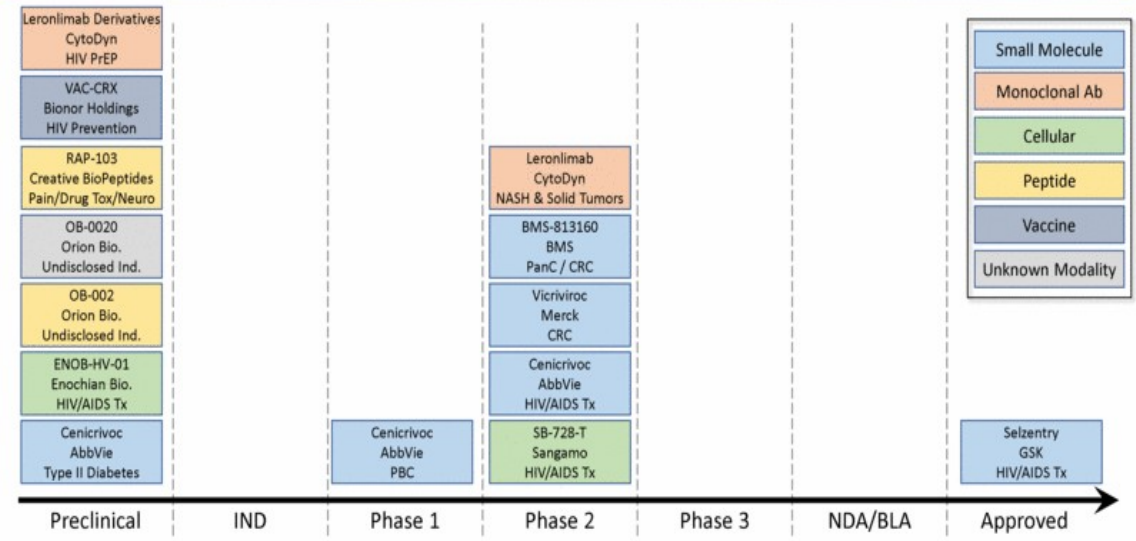
CytoDyn CCR5 Inhibitors – Indications Studied



Source: *BioMedTracker (July 2022), CytoDyn Analysis*

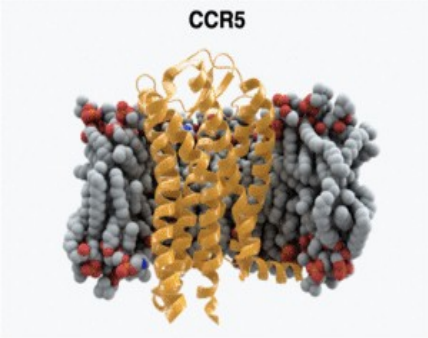
- There are a total of **51** unique disease programs, in ~**25** indications, where CCR5 agents are the primary investigational therapy.
- HIV/AIDs is the largest indication with **18** unique programs.
- Solid tumors (as a group) are the next largest with **8** programs (CRC, BC, PanC, and UC).
- The remaining indications are comprised of neurologic, immunologic, metabolic, and infectious diseases.

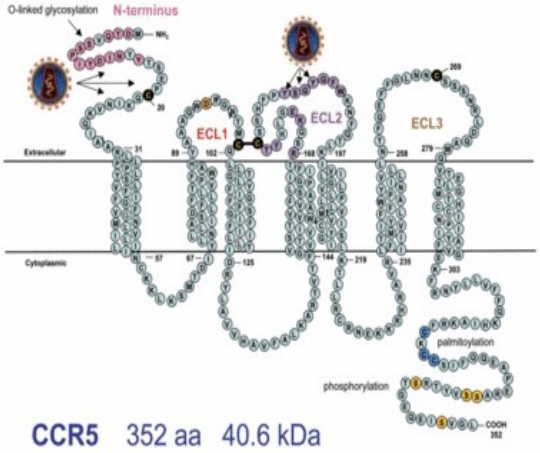
CytoDyn CCR5 Inhibitors – Active Development Pipeline



Source: BioMedTracker (July 2022), CytoDyn Analysis

CytoDyn Unique Binding & Immunomodulating Properties





CCR5 352 aa 40.6 kDa

CytoDyn Leronlimab Clinical Development History

CytoDyn Leronlimab Clinical Development History

CytoDyn Clinical Hold Status with FDA

- **In March of 2022 CytoDyn received a Full Clinical Hold for COVID-19 treatment and a Partial Clinical Hold for HIV Treatment from the FDA**
  - CytoDyn voluntarily withdrew the IND for COVID-19
  - CytoDyn is actively working on resolving the Partial Clinical Hold for HIV

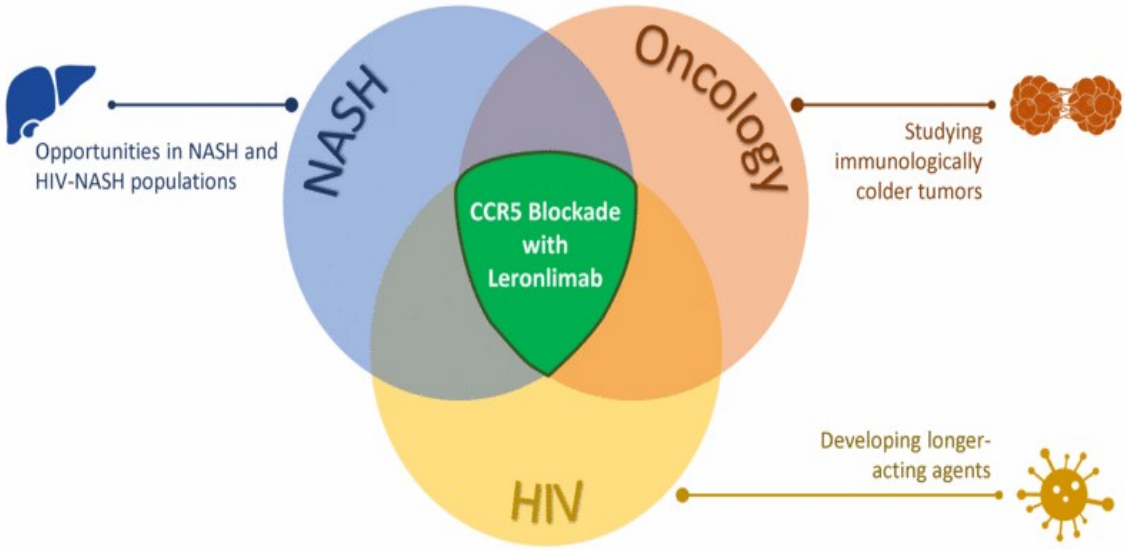
- **5 items requested by the FDA:**
  - **Investigator Brochure –** *Submitted in September*
  - **Development Safety Update Report –** *Submitted in October*
  - **Safety Management Plan –** *Submitted in November*
  - **Aggregate Safety Analysis –** *In progress*
  - **Benefit Risk Analysis –** *In progress*

FDA review ends 30 days following final submission

CytoDyn

Future Development

CytoDyn Refocusing Pipeline on High Value Opportunities



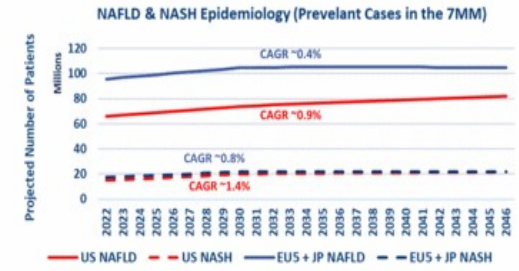
CytoDyn Future Development for Leronlimab

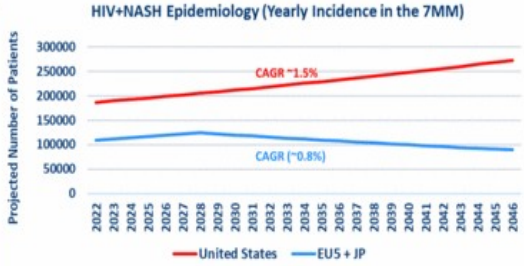
- Going forward CytoDyn is focusing on NASH, oncology, and earlier line HIV indications
- CytoDyn has already generated promising clinical signals in NASH and oncology
- Within NASH, CytoDyn is pursuing the broader NASH population as well as a segment of NASH patients with HIV, where CytoDyn is in a unique position to address both diseases
- Within Oncology, CytoDyn plans to pursue colorectal cancer (MSS population) and breast cancer ($HR^+/HER2^-$ and TNBC population), where the company has already generated promising clinical signals

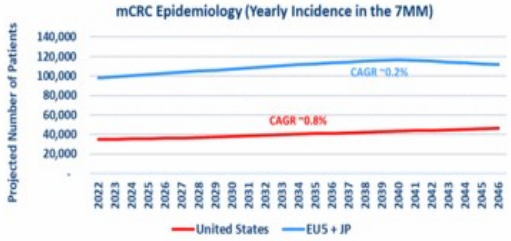
Projected Market Size for NASH & HIV-NASH (2028)		Projected Market Size for BC & CRC (2028)	
General NASH Population	HIV + NASH	Colorectal Cancer (MSS)	Breast Cancer ($HR^+/HER2^-$)
			

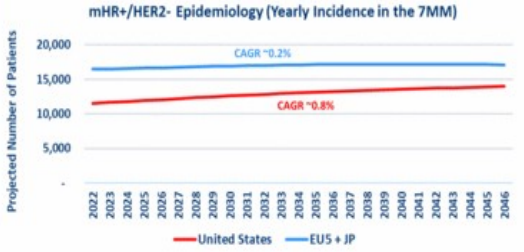
* **Source:** *Average from DataMonitor Healthcare, Insight Partners, Market Growth Reports*
** **Source:** *No 3rd Party Data Available, CytoDyn Projections*
*** **Source:** *Decision Resources Group*

CytoDyn Epidemiology for Future Indications









Source: *DataMonitor (July 2022), CytoDyn Analysis*

CytoDyn NAFLD & NASH – Disease Background

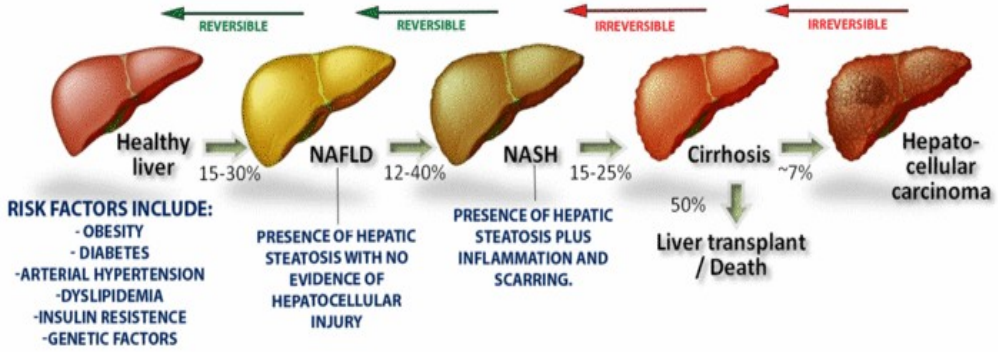
The diagnosis of non-alcoholic fatty liver disease (NAFLD) requires evidence of hepatic steatosis and the lack of secondary causes of liver fat accumulation such as substantial alcohol consumption, long-term use of a steatogenic medicine, or monogenic hereditary disorders.

NASH is a more advanced form of NAFLD where there is liver inflammation with hepatocyte injury, which can lead to hepatic fibrosis. Although the presence of fibrosis is not required for a diagnosis of NASH, fibrosis is present in over 80% of NASH patients. For this reason, NASH patients are often further segmented by their fibrosis stage. Detection of fibrosis is important as it is linked to worse outcomes for patients.

NASH can lead to serious clinical burden due to the high liver-related and non-liver-related mortality rates associated with the disease. Moreover, **advanced NASH patients are at increased risk of developing cirrhosis, which is linked to liver decompensation, end-stage liver disease, and hepatocellular carcinoma**.

CytoDyn NAFLD & NASH – Disease Progression

THE NON-ALCOHOLIC FATTY LIVER DISEASE (NAFLD) SPECTRUM



CytoDyn HIV-NASH – Disease Background

The burden of liver-related morbidity remains high among HIV-infected patients, despite advances in the treatment of HIV and viral hepatitis.

The impact of non-alcoholic fatty liver disease (NAFLD) is significant with a prevalence of up to 50%.

Both HIV infection itself and combination antiretroviral therapy (cART) can contribute to the development of NAFLD/NASH in various ways:

- As ongoing HIV-related immune activation is associated with insulin resistance, mitochondrial dysfunction, and dyslipidemia, early initiation of cART is needed to limit its duration.
- However, the use of early-generation nucleos(t)ide reverse transcriptase inhibitors and protease inhibitors is also associated with the development of NAFLD/NASH.

Patients with HIV are typically excluded from NASH clinical trials, creating an unmet clinical need that is not currently being addressed

CytoDyn Metastatic CRC – Background and Treatment

CRC typically develops through the proliferation of mucosal epithelial cells of the gastrointestinal (GI) wall, eventually forming a polyp or adenoma. As with many other cancers of the GI tract, the vast majority (>95%) are adenocarcinomas.

CRC stands as the second deadliest and third most diagnosed form of cancer worldwide

The most commonly prescribed targeted therapies in front-line mCRC are angiogenesis inhibitors, particularly bevacizumab, which is available for most tumor types and alongside most chemotherapy combinations. Other drugs, such as Erbitux and Braftovi, are limited by gene expression, whereas PD-1 inhibitors Keytruda, Opdivo, and most recently Jemperli may be used in MSI-H tumors (~10-15% of mCRC).

However, these PD-1 inhibitors have failed to show success in micro satellite stable (MSS) tumors to date

CytoDyn Breast Cancer HR$^+$/HER2$^-$ – Disease Background

Hormone receptor-positive (HR+) is the most common breast cancer subtype, **with approximately 70% of breast cancers presenting with overexpression of estrogen receptors, progesterone receptors, or both**.

Overexpression of the hormone receptors allows estrogen and progesterone to drive tumor growth and proliferation. Therefore, endocrine therapy remains the standard treatment for advanced patients with HR+/human epidermal growth factor receptor 2-negative (HER2-) breast cancer.

Worldwide, breast cancer is the most common cancer in terms of new cases and the **fifth-leading cause of cancer-related death regardless of gender.**

In the US, breast cancer is the most common cancer in women **and the fourth-leading cause of cancer-related death regardless of gender**, behind lung cancer, colorectal cancer, and pancreatic cancer.

CytoDyn

NASH & Leronlimab

Mazen Noureddin, M.D., M.H.Sc.
Director of the Houston Liver Institute

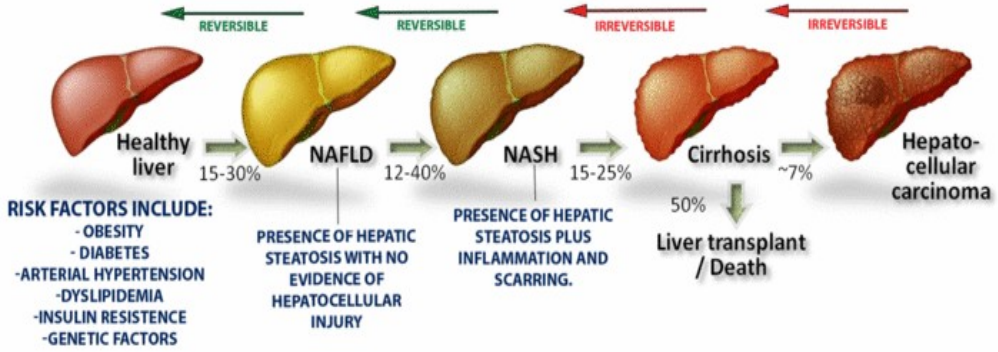
Background - Non-Alcoholic Steatohepatitis (NASH)

- Non-alcoholic steatohepatitis (NASH) is a chronic liver disease characterized by the presence of hepatic inflammation and cell injury due to hepatic fat accumulation (steatosis) in ≥5% of hepatocytes
- Patients with advanced fibrosis due to NASH are at significantly higher risk of liver-related mortality
- Despite its very high burden, there are currently no approved pharmacological therapies for NASH
- Recent studies targeting chemokine-mediated inflammatory signaling pathways that may reduce liver fat and scar tissue formation indicate the great potential of immune modulation as a therapeutic strategy in NASH[1]

1) Lefere S, Puengel T, Hundertmark J, Penners C, Frank AK, Guillot A, de Muynck K, Heymann F, Adarbes V, Defrêne E, Estivalet C, Geerts A, Devisscher L, Weiskirchen R, Tacke F. Differential effects of selective-and pan-PPAR agonists on experimental steatohepatitis and hepatic macrophages. J Hepatol. 2020 Oct;73(4):757-770. doi: 10.1016/j.jhep.2020.04.025. Epub 2020 Apr 29. PMID: 32360434.

CytoDyn NAFLD & NASH – Disease Progression

THE NON-ALCOHOLIC FATTY LIVER DISEASE (NAFLD) SPECTRUM



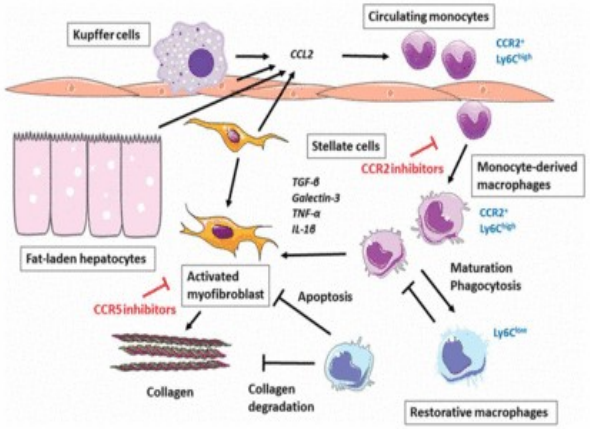
CytoDyn NAFLD & NASH – Molecular Pathways to Fibrosis

CCR2 and CCR5 as targets in NASH and liver fibrosis

The accumulation of hepatic fat causes cellular stress and the release of chemokines, notably CCL2 (= MCP-1), from hepatocytes, Kupffer cells, endothelial and stellate cells.

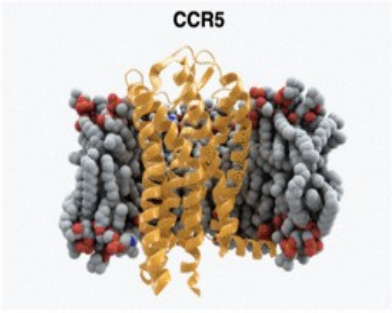
This promotes the infiltration of circulating CCR2+ monocytes, which differentiate into monocyte-derived macrophages. These cells contribute to the progression of NASH by maintaining an inflammatory environment and by activating hepatic stellate cells, promoting collagen deposition. These CCR2+ Ly6Chigh macrophages can mature into Ly6Clow restorative macrophages which boost resolution of inflammation.

CCR5 is expressed by stellate cells and is involved in their profibrogenic activation and proliferation. This concept is based on experimental mouse models of steatohepatitis and fibrosis.



Source: *Lefere et al., Expert Opinion on Investigational Drugs (2020)*

CytoDyn The CCR5 Receptor as a therapeutic target in NASH



CCR5 is G Protein coupled receptor.

Acts as a receptor for chemokines.

Cognate ligands include CCL3, CCL4, and CCL5 (Rantes)

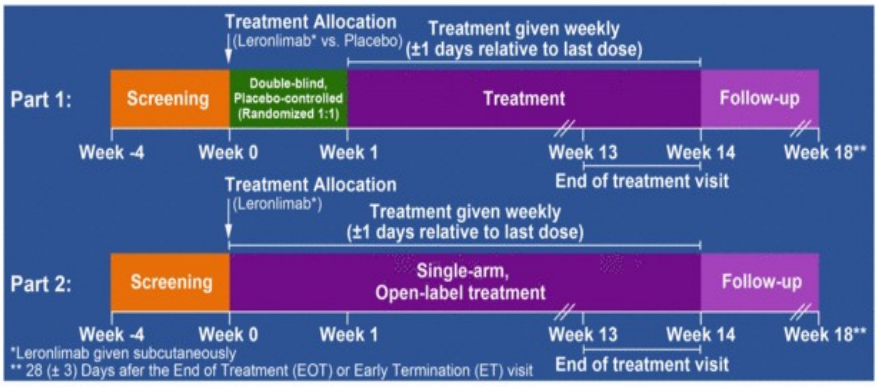
Macrophage-mediated inflammation and hepatic stellate cell activation are key drivers of progression at multiple stages of the disease. CCR5 is present on both.

CytoDyn CCR5 and the Inflammatory Response

- In NASH, liver homeostasis is impaired due to an accumulation of toxic lipids which can activate both Kupffer cells (KCs) and tissue-resident macrophages resulting in the production of fibrogenic cytokines and chemoattractant chemokines such as transforming growth factor-beta(TGF-B) and monocyte chemoattractant protein-1 (MCP-1)
- Not only do these cytokines/chemokines promote transdifferentiation of hepatic stellate cells (HSCs) into myofibroblasts (the primary source for fibrillary collagens), but they also amplify the immune response by recruiting additional cells into the damaged area
- Recruitment of extra-hepatic inflammatory cells to the site of hepatic injury is typically mediated by interactions between cytokines/chemokines and their receptors
- **It has also been shown that patients with NASH also have high levels of C-C chemokine receptor 5 (CCR5) and the associated ligand, CCL5, thus demonstrating a potential role of CCR5 and its ligands in liver fibrosis**
- Furthermore, anti-inflammatory and anti-fibrotic effects have been reported in pre-clinical models of NASH following the use of monoclonal antibodies targeting chemokine signaling pathways
- CCR5 has thus become an attractive target for the clinical development of new antifibrotic therapies[2]

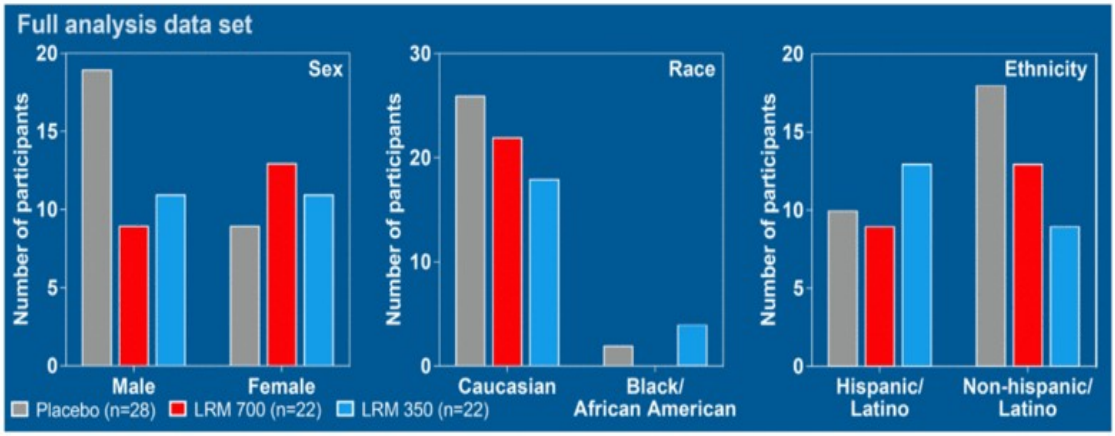
2) *Lefebvre E, M. G. Antifibrotic Effects of the Dual CCR2/CCR5 Antagonist Cenicriviroc in Animal Models of Liver and Kidney Fibrosis. 2016; 11(6), e0158156.*

CDI-NASH-01: Design

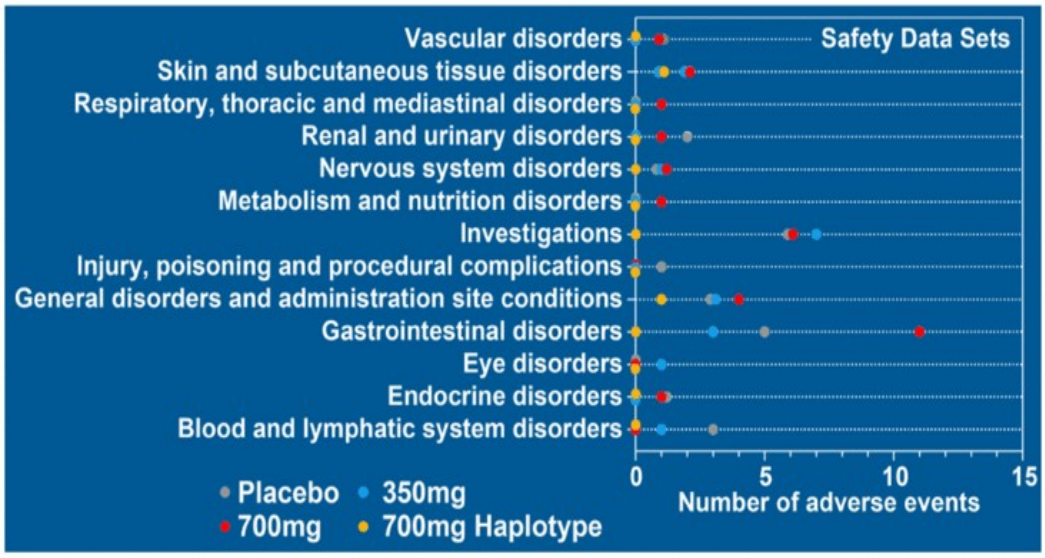


Designed as a multi-center Phase 2a trial, subsequently converted into an exploratory study to evaluate dose, efficacy, and safety of leronlimab 700 mg and 350 mg along with biomarkers to help design future trials and explore mechanisms of action

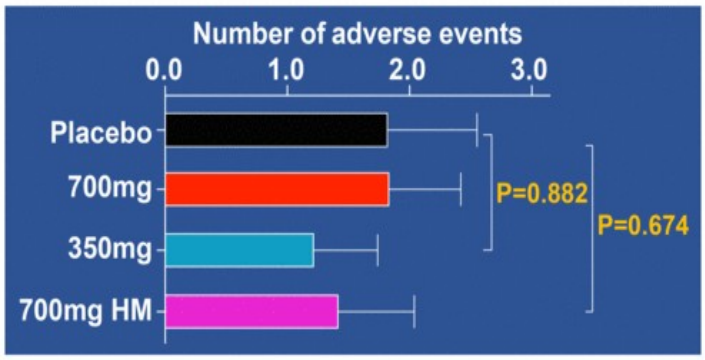
CDI-NASH-01: Results - Demographics



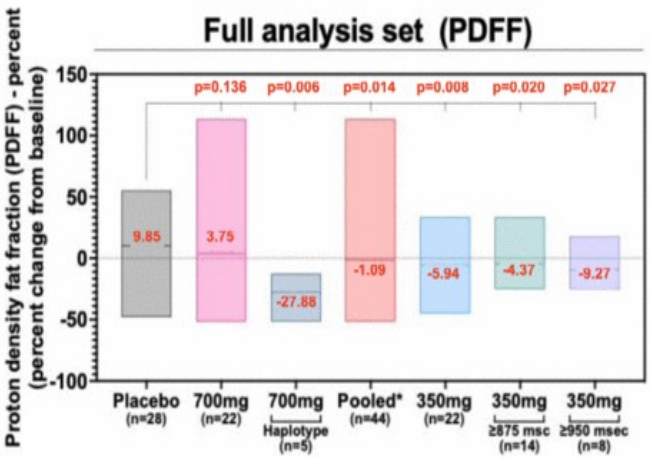
CDI-NASH-01: Results – Adverse Events (Safety)



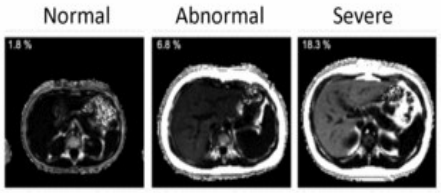
CDI-NASH-01 All Groups: Well Tolerated



CytoDyn CDI-NASH-01: Results – MRI PDFF



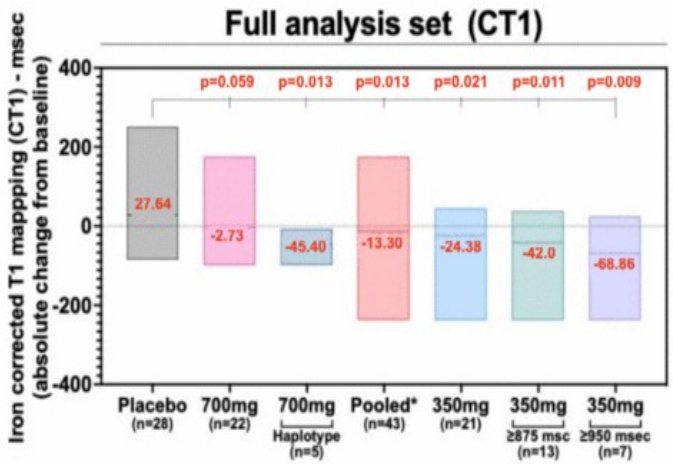
All analyses performed were exploratory.



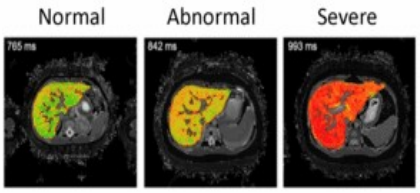
*350 mg + 700 mg
p values are from Wilcoxon Rank Sum test
Not adjusted for multiplicity

MRI-PDFF is being studied as an imaging surrogate endpoint for the fat density in the liver.

CDI-NASH-01: Results – Fibro-Inflammation by MRI cT1



All analyses performed were exploratory.



*350 mg + 700 mg
p values are from Wilcoxon Rank Sum test
Not adjusted for multiplicity

MRI-cT1 is being studied as an imaging surrogate endpoint for hepatic fibro-inflammation. This is a critical unmet need in the NASH space, as many agents have been unable to show reductions in fibro-inflammation despite reductions in hepatic steatosis.

CytoDyn NASH01 350 mg Groups: Improved Liver Function Tests

Baseline ALT ≥50
(n = 8) had reductions in ALT for 350 mg compared to placebo

Elevated ALT 350
ALT mean reduction -29.3 U/L
PDFF mean reduction -18%
cT1 mean reduction -69 ms

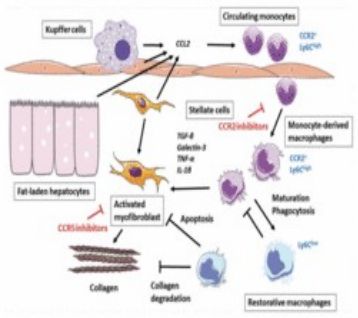
Elevated ALT Placebo
ALT mean reduction -4.2 U/L
PDFF mean increase +6%
cT1 mean increase +5 ms

Biomarker (mean change baseline to week 14)	Placebo (n=28)	350 mg (n=22)	350 mg cT1 ≥ 875 (n=14)	350 mg cT1 ≥ 950 (n=8)
Alkaline Phosphatase U/L	-0.1	-2.82	-3.16	-7.25*
ALT (Alanine Aminotransferase) U/L	-0.11	-2.5	-2.7	-8.3
AST (Aspartate Aminotransferase) U/L	1.29	-5.3	-5.5	-13.0
Gamma Glutamyl Transferase (GGT) U/L	-2.3	-1.3	-2.5	-6.13

34% Subjects at baseline had elevation of one or more Liver function tests
*P values are from ANCOVA or Wilcoxon Rank Sum test as appropriate; *p<0.05, **p<0.01, ***p<0.001 versus placebo (not adjusted for multiplicity)*

Analyses are exploratory. Further trials are needed with larger numbers of patients with elevated liver function at baseline

CytoDyn NASH01 350 mg Groups: Reduced Chemotactic Proteins

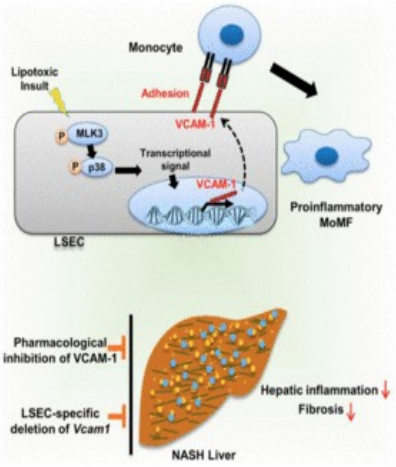


Biomarker (mean change baseline to week 14)	Placebo (n=28)	350 mg (n=22)	350 mg cT1 ≥ 875 (n=14)	350 mg cT1 ≥ 950 (n=8)
Monocyte Chemotactic Protein-1 (CCL2) pg/mL	-49.3	-103.05	-95.53	-179.0
Macrophage Inflammatory Protein-1 Alpha (CCL3) pg/mL	-1.0	-7.82***	-7.14***	-8.0***
RANTES (CCL5) ng/mL	-0.35	-7.0	-7.8	-25.3
Eosinophil Chemotactic Protein (CCL11) pg/mL	-43.75	-22.77	-15.21	-16.25
Macrophage Inflammatory Protein-4 (CCL18) ng/mL	0.46	-31.3**	-32.1**	-48.0**

*P values are from ANCOVA or Wilcoxon Rank Sum test as appropriate; *p<0.05, **p<0.01, ***p<0.001 versus placebo (not adjusted for multiplicity)*

These analyses are exploratory but provide insight into multiple mechanisms of action for leronlimab beyond CCR5

CytoDyn NASH01 350 mg Groups: Reduced VCAM

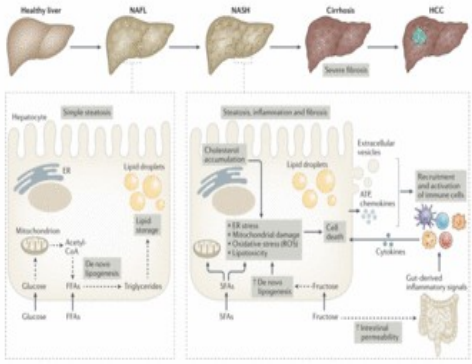


Biomarker (mean change baseline to week 14)	Placebo (n=28)	350 mg (n=22)	350 mg cT1 ≥ 875 (n=14)	350 mg cT1 ≥ 950 (n=8)
VCAM (Vascular Cell Adhesion Molecule) ng/mL	10.2	-93.8***	-94.2***	-81.0*
S100A12 EN RAGE ng/mL	57.75	-80.09**	-115.50**	-169.0*

EN RAGE (extracellular newly identified receptor for advanced glycation end products binding protein). *P values are from ANCOVA or Wilcoxon Rank Sum test as appropriate; *p<0.05, **p<0.01, ***p<0.001 versus placebo (not adjusted for multiplicity)*

These analyses are exploratory but suggest new differentiating mechanisms of action for leronlimab

NASH01 350 mg Groups: Reduction in Key Markers of Inflammation

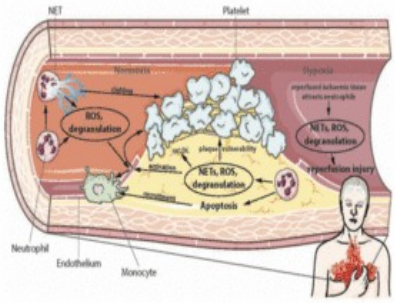


Biomarker (mean change baseline to week 14)	Placebo (n=28)	350 mg (n=22)	350 mg cT1 ≥ 875 (n=14)	350 mg cT1 ≥ 950 (n=8)
IL-1 (Interleukin 1) Beta pg/mL	0.16	0.00	0.00	0.00
IL-1RA (Interleukin 1 Receptor Antagonist) pg/mL	17.04	-28.36*	-34.79**	-26.25
IL-6 (Interleukin 6) pg/mL	0.19	-0.28	-0.44	-0.30
IL-8 (Interleukin 8) pg/mL	-0.071	-2.80**	-1.26***	-4.25**
TNF Receptor 2 (sTNFR-2) ng/mL	0.82	-2.2***	-2.0***	-2.7***

*P values are from ANCOVA or Wilcoxon Rank Sum test as appropriate; *p<0.05, **p<0.01, ***p<0.001 versus placebo (not adjusted for multiplicity)*

These analyses are exploratory but show leronlimab reduces key markers of inflammation involved in NASH

CytoDyn NASH01 350 mg Groups: Improvements in CV Markers



Biomarker (mean change baseline to week 14)	Placebo	350	350 cT1>850	350 cT1>950
Apolipoprotein A1S mg/dL	-3.2	7.4	9.2	2.3
High Density Lipoprotein (HDL) U/L	-1.1	2.05	2.0	0.4
LDL/HDL U/L	0.72	0.09	0.11	0.095
Neutrophil to Lymphocyte Ratio K/uL	0.038	-0.40**	-0.52***	-0.32*
Tissue Inhibitor Metalloproteinases 1 ng/mL	-0.25	-18.27*	-17.26	-25.3
EN RAGE ng/mL	57.75	-80.09**	-115.50**	-169.0*
VCAM (Vascular Cell Adhesion Molecule) ng/mL	10.2	-93.8***	-94.2***	-81.0*

*P values are from ANCOVA or Wilcoxon Rank Sum test as appropriate; *p<0.05, **p<0.01, ***p<0.001 versus placebo (not adjusted for multiplicity)*

These analyses are exploratory but the mechanism of action for leronlimab appears to be multifactorial with implications for systemic reductions in vascular permeability, arterial stiffness, and oxidative stress.

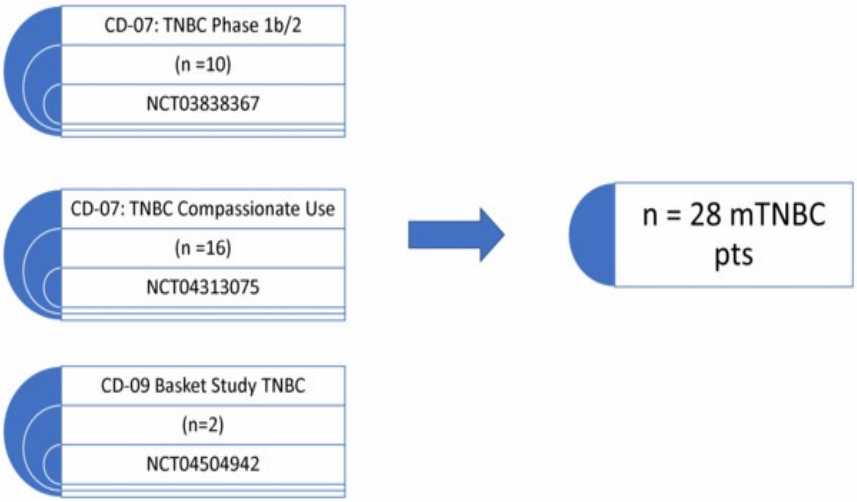
CytoDyn CDI-NASH-01: Conclusions

- All analyses performed were exploratory
- Treatment with leronlimab was generally well tolerated in both Part 1 and Part 2
- Part 1: Leronlimab 700 mg did not reduce mean change in PDFF and cT1 from baseline to week 14 vs. PBO
- Part 2: Leronlimab 350 mg significantly reduced mean change in PDFF and cT1 from baseline to week 14 vs. PBO
- Despite increased fibro-inflammation, in patients with moderate and severe cT1 values at baseline, leronlimab 350 mg still showed significantly reduced cT1 from baseline to week 14 vs. placebo
- Part 1 & Part 2 pooled
  - Pooled 350 mg + 700 mg group also had significant reductions in PDFF and cT1 vs. placebo
  - Although small sample size (n=5) CCR5 haplotype analysis is suggestive that specific haplotypes may be better suited for the 700 mg dose of leronlimab
- **Results warrant additional follow-up in a larger trial**

CytoDyn

Leronlimab in Oncology – Clinical Data

CytoDyn Leronlimab in Patients with mTNBC - Clinical Data



CytoDyn mTNBC Patient Overview

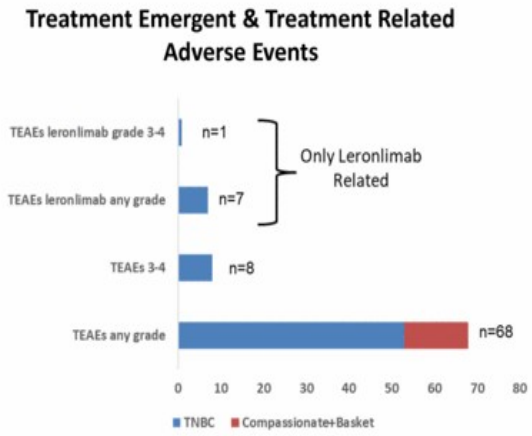
- **Data was available and pooled from three sources (n = 28 patients):**
  - All patients were listed as mTNBC
    - **n=16** were from Compassionate Use
    - **n=10** were from mTNBC trial
    - **n=2** were from Basket trial
  - Patients received ≥1 dose of Leronlimab (range 1 - 33 doses)
  - Doses ranged from 350mg - 700mg
  - 4 patients had dose escalation per study protocol
  - Average age was 52 (range 33 - 75)
  - Data was as reported by August 15, 2021
- **Standard of Care and Sacituzumab Govitecan used as references**
  - Standard of Care (SOC) and Sacituzumab Govitecan (SG) were not evaluated directly in these studies
  - SOC and SG reference lines are for hypothetical reference only from historical controls

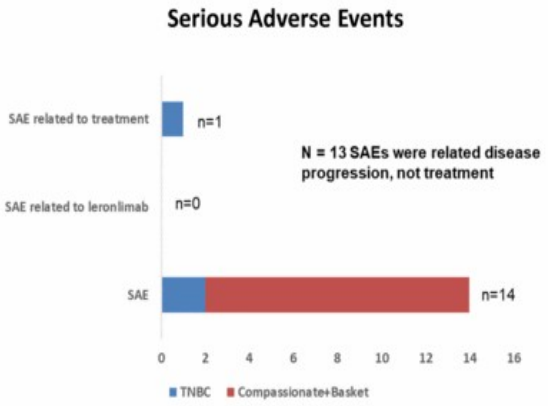
CytoDyn Drugs Used in Combination with Leronlimab

- Carboplatin
- Pacitaxel + Carboplatin
- Gemcitabine + Carboplatin
- Eribulin
- Capecitabine
- Pemetrexed
- Atezolizumab
- nab-paclitaxel + Atezolizumab
- Abraxane + Atezolizumab
- Capecitabine + Atezolizumab

No cross reactions were observed, but sample sizes are small

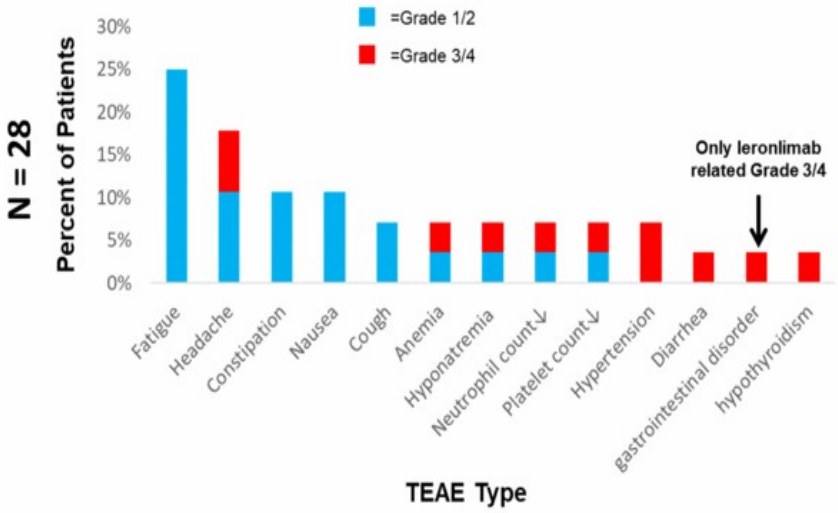
CytoDyn Safety Data: TRAEs, TEAEs, and SAEs (n=28)





Note: One patient had no TEAEs at last visit, but final EOT was not available

CytoDyn Safety Data: Top 13 treatment related TEAEs



KM Plots of Leronlimab Treated Population (n=28)

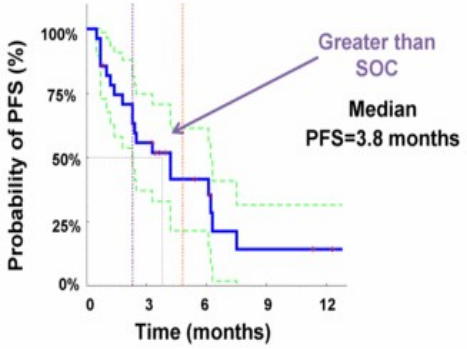
*PFS for standard ≥3rd line therapy = 2.3 months

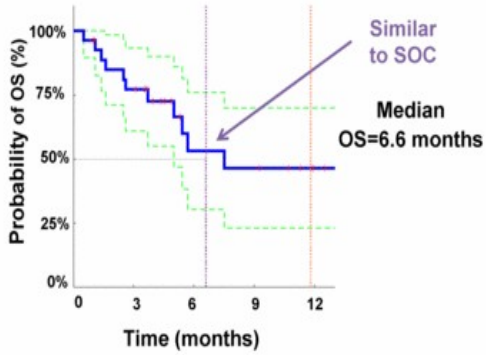
**OS for standard ≥3rd line therapy = 6.5 months

Reference median for Standard of Care
Reference median for SG Trial
95% Confidence Interval
Survival Curve

n=11/28 patients had bone and/or brain mets

SG trial excludes brain or bone mets.





The SOC and SG medians are historical references from data outside of any leronlimab trial; no conclusions about leronlimab should be drawn based on such post-hoc historical comparisons.

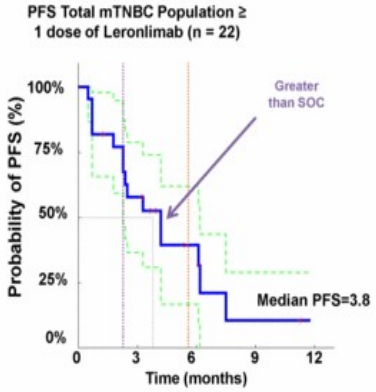
KM Plots of Population without Prior Brain Mets

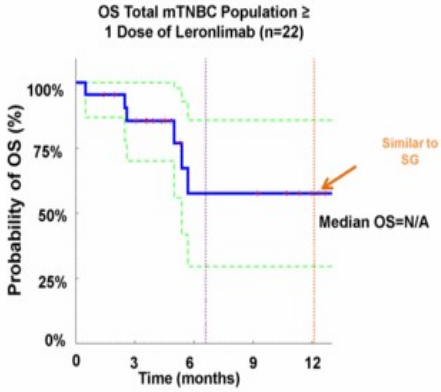
95% Confidence Interval
Survival Curve

Reference median for Standard of Care
Reference median for SG Trial in patients without Brain Metastases

*PFS for standard ≥3rd line therapy=2.3 months

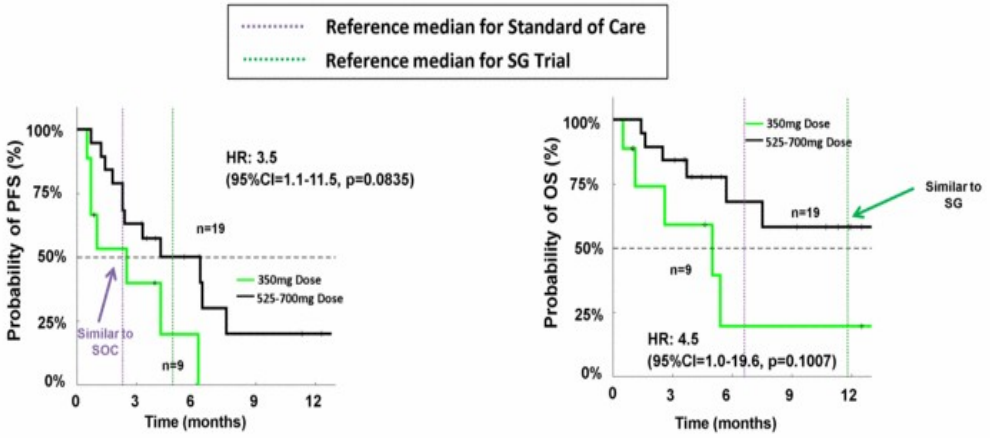
**OS for standard ≥3rd line therapy=6.6 months





The SOC and SG medians are historical references from data outside of any leronlimab trial; no conclusions about leronlimab should be drawn based on such post-hoc historical comparisons.

CytoDyn KM Plots Based on Leronlimab Dose Used (n=28)



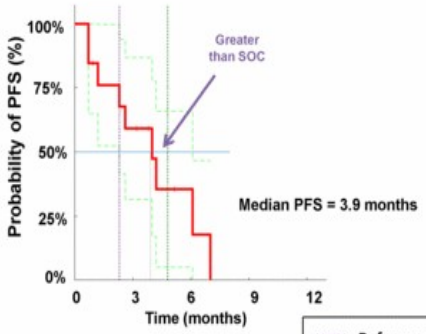
*4 patients increased dose from 350 to 525 and were included in the higher dose group

The SOC and SG medians are historical references from data outside of any leronlimab trial; no conclusions about leronlimab should be drawn based on such post-hoc historical comparisons.

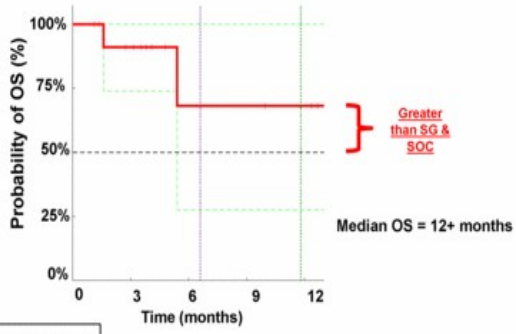
CytoDyn KM Plots of Carboplatin & Leronlimab Population

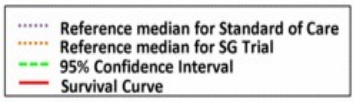
Patients includes mTNBC trial and Compassionate Use Trial

PFS Total mTNBC Population ≥1 dose of Leronlimab (n = 13)



OS Total mTNBC Population ≥1 Dose of Leronlimab (n = 13)

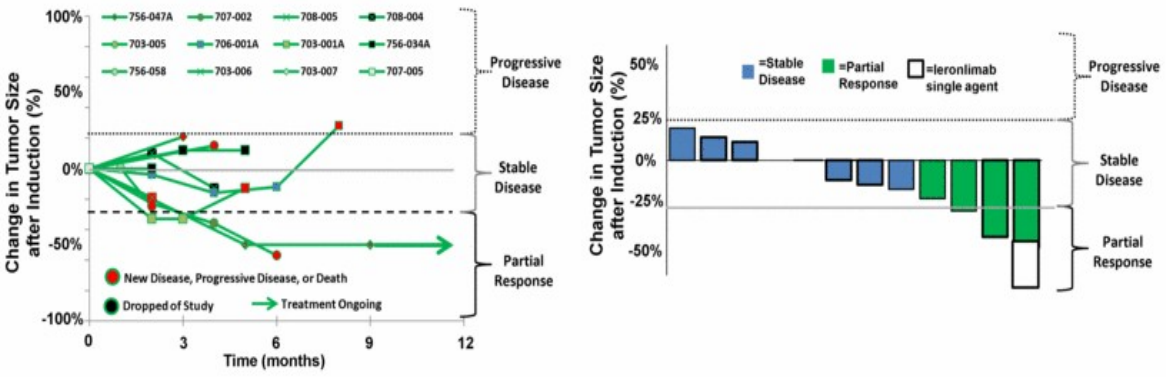




The SOC and SG medians are historical references from data outside of any leronlimab trial; no conclusions about leronlimab should be drawn based on such post-hoc historical comparisons.

CytoDyn Changes in Tumor Growth by PET/CT

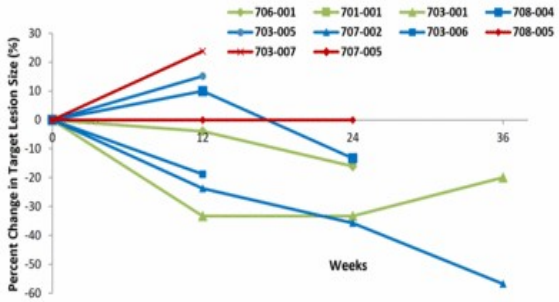
Based on n=12 patients with RECIST Scans



CytoDyn Phase Ib/II mTNBC Changes in Tumors by PET/CT

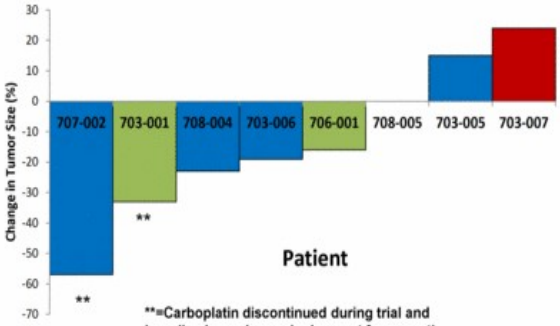
350mg Leronlimab + Carboplatin
525mg Leronlimab + Carboplatin
700mg Leronlimab + Carboplatin

Change in Target Lesion Size at Follow-Up Scan



* 701-001 and 707-005 progressed before follow-up scan

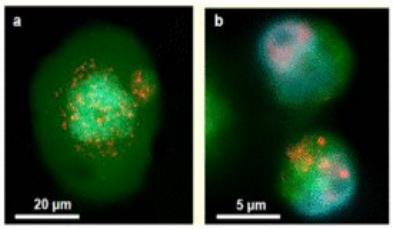
Maximum Change in Tumor Size



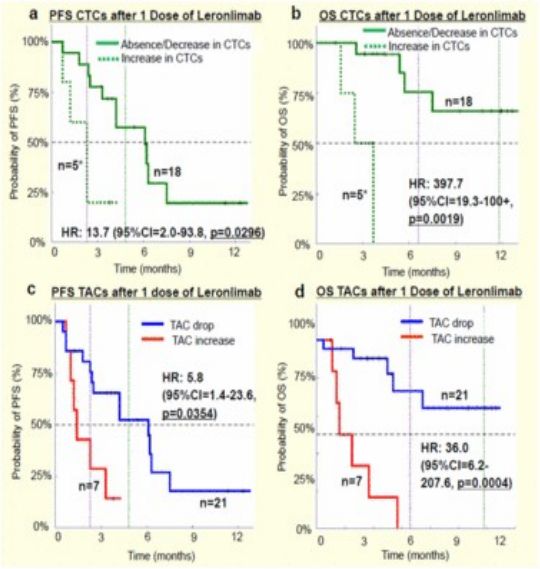
**=Carboplatin discontinued during trial and leronlimab used as a single agent for some time

CytoDyn AACR 2022 Poster Presentation

- **Title**: Changes in Circulating Tumor Associated Cells Predicts Progression Free and Overall Survival in Metastatic TNBC Patients after Induction with the Anti-CCR5 Drug Leronlimab
- Same TNBC patient population pooled from three leronlimab studies described previously

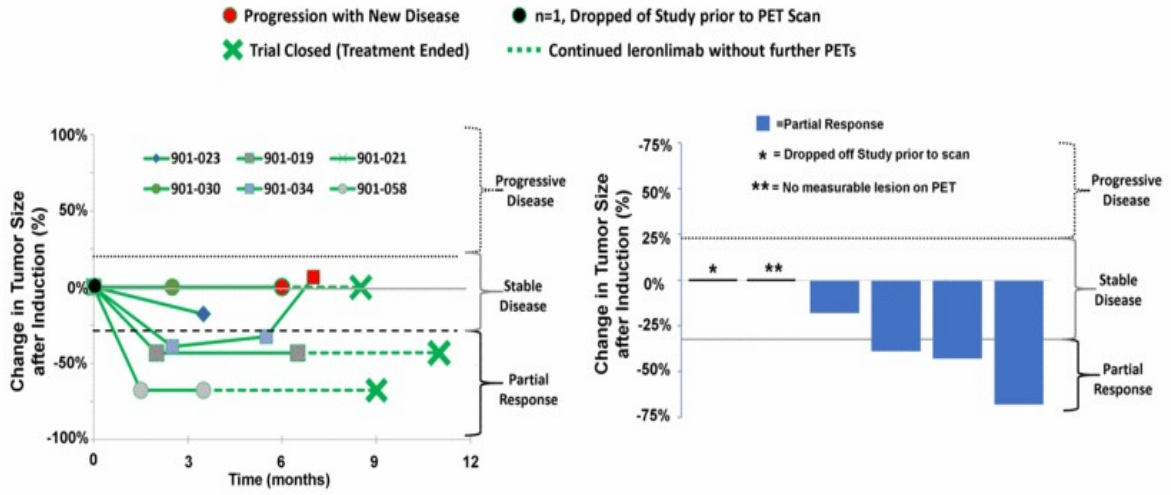


CCR5 expression in a CAML (a) and CTC (b) from mTNBC disease (Red = CCR5, Blue = nucleus, Green = Cytoplasm)



CytoDyn Leronlimab in Patients with CRC - Clinical Data

CytoDyn Colorectal Patients with Available PET/CT

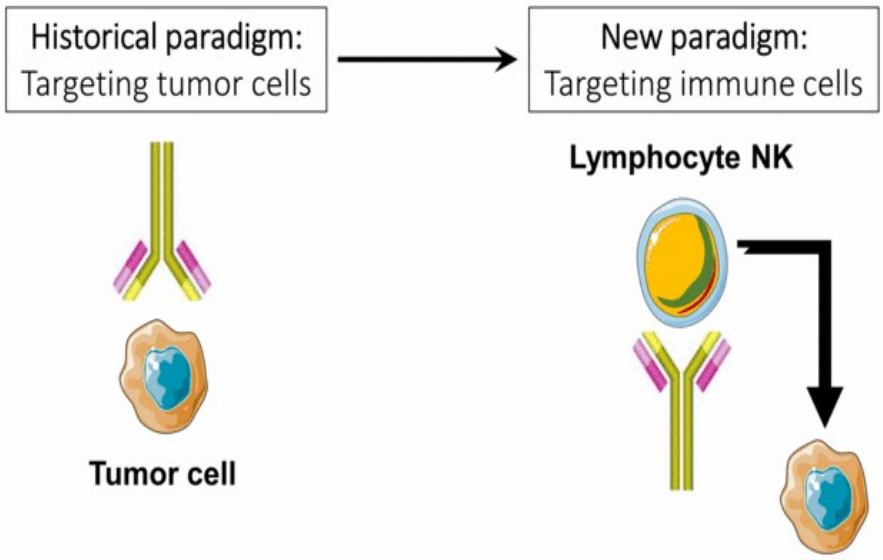


CytoDyn

Microenvironment and IO in Cancer Therapy

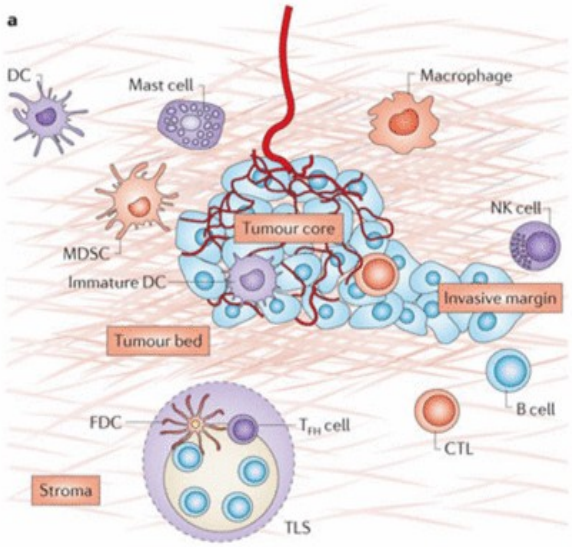
Stefan Glück MD PhD FRCPC
Professor em. of Medicine
Oncology Hematology Immunology Biotech

CytoDyn Paradigm shift in cancer therapy



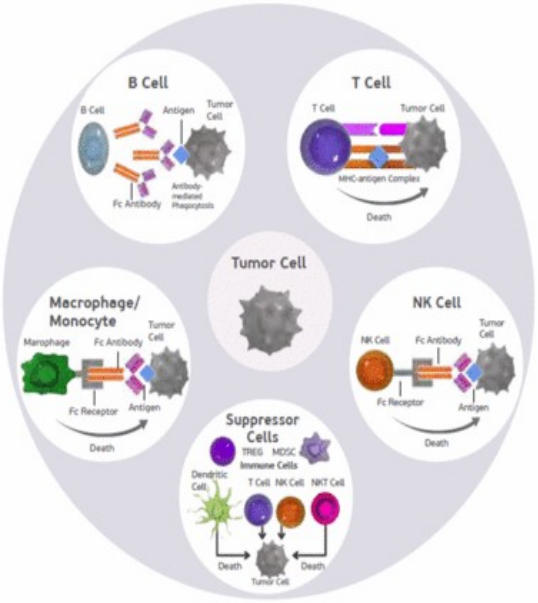
W H Fridman et al., Nature Rev. Cancer, 12, 298-306, 2012

The Tumor Microenvironment (TME)

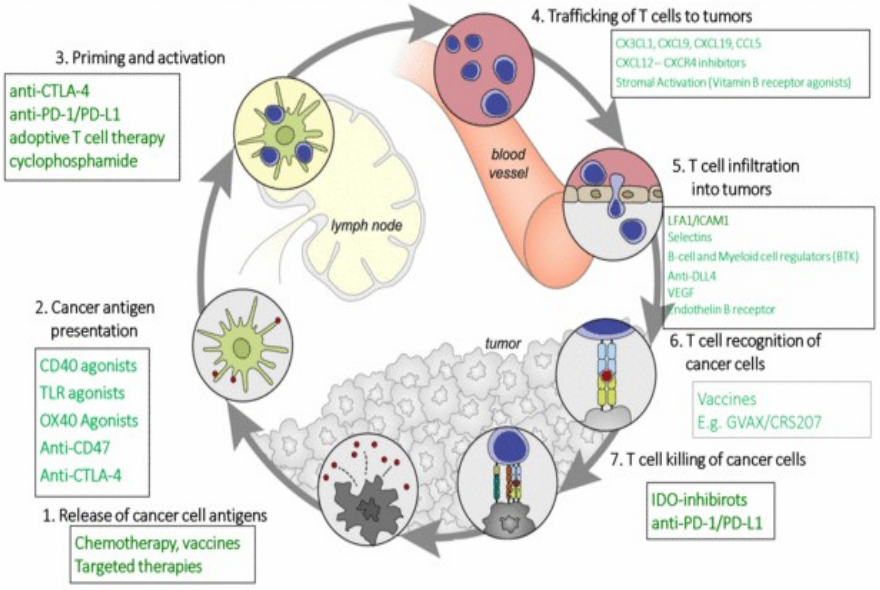


W H Fridman et al., Nature Rev. Cancer, 12, 298-306, 2012

CytoDyn Multiple Aspects of the Anti-Tumor Immune Response

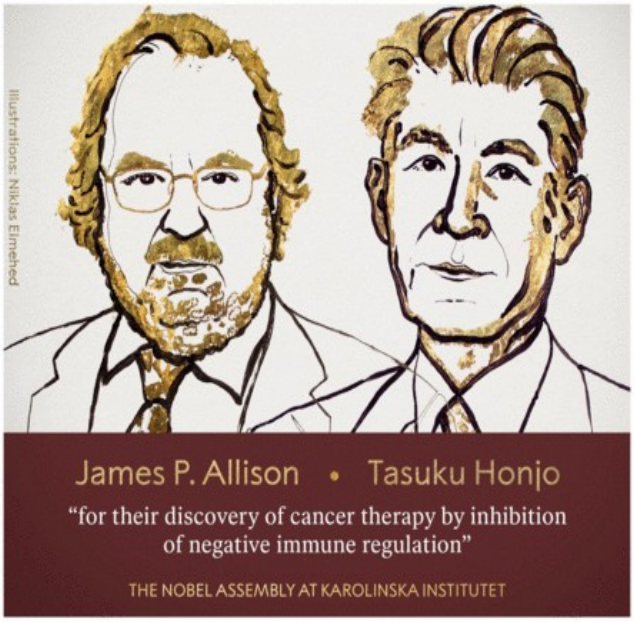


CytoDyn The Cancer Immune Cycle

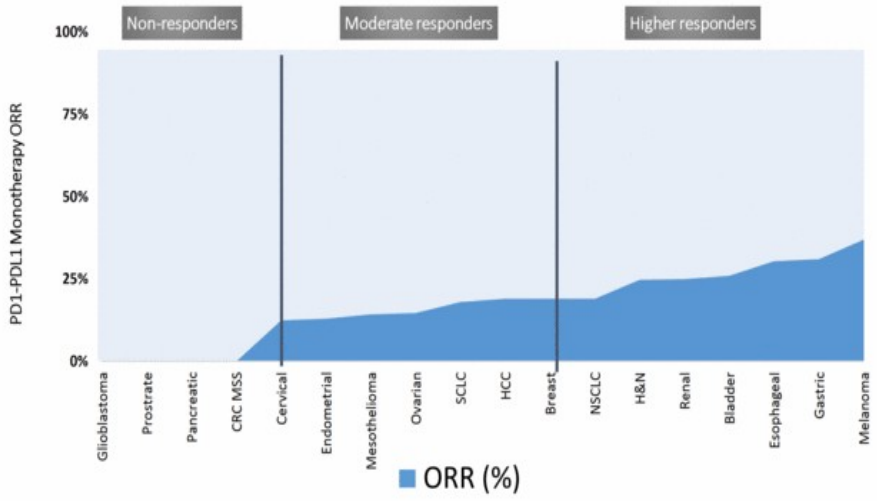


Adapted from Immunity 39:1 2013

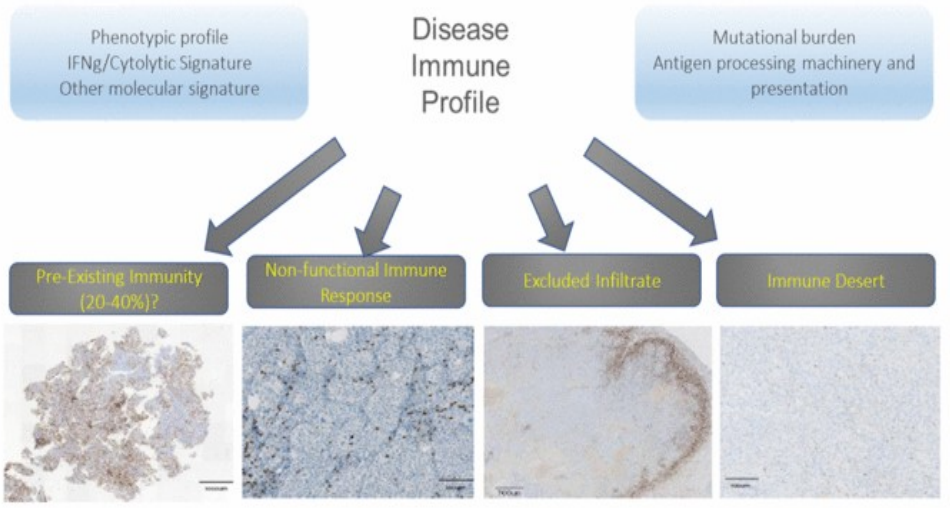
The Nobel Prize in Physiology or Medicine 2018



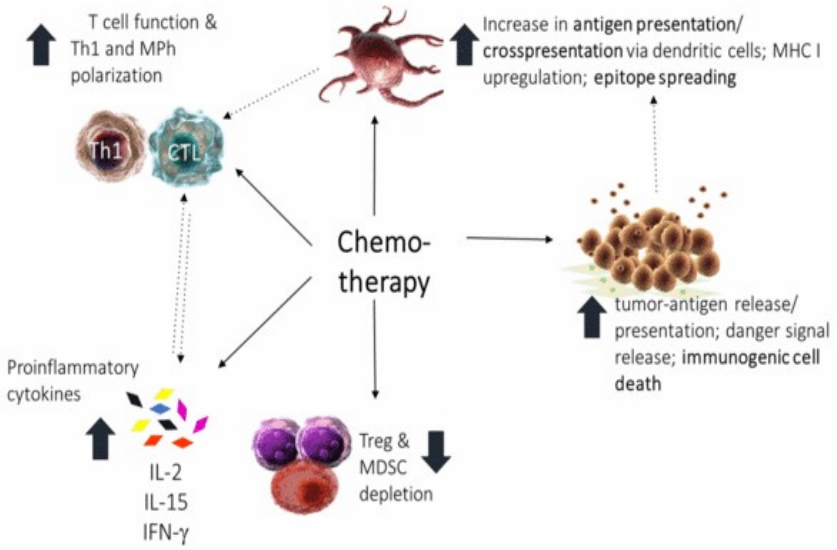
CytoDyn Three Categories of Response to Anti-PD-1/PD-L1



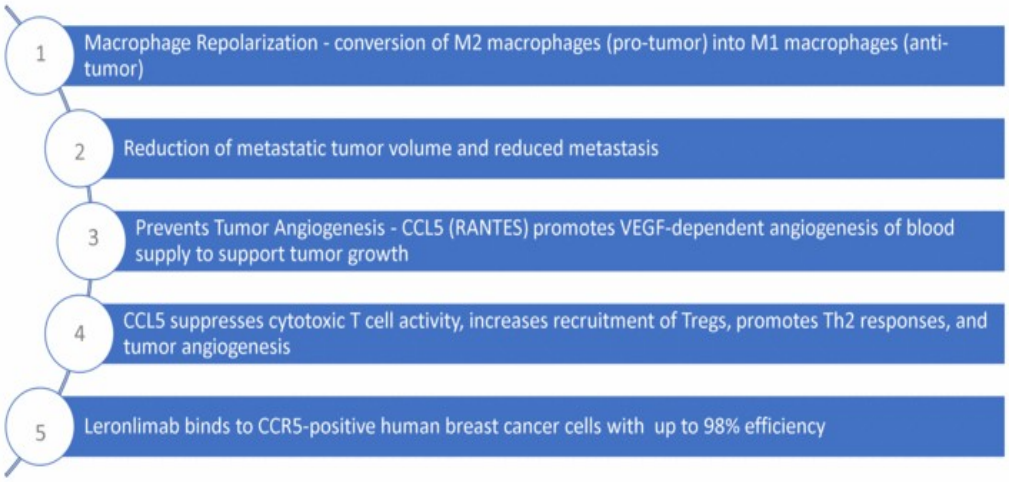
Indication Selection and Patient Segmentation Based on Categories of Tumor Immune Profile



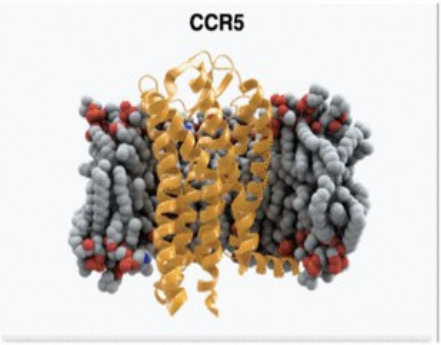
Chemotherapy: Pleiotropic Stimulatory Effects on the Immune System

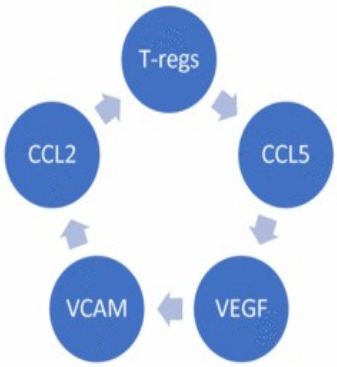


Unique MoA of Leronlimab in Immuno-Oncology



CytoDyn The Potential Role of CCR5 in the TME

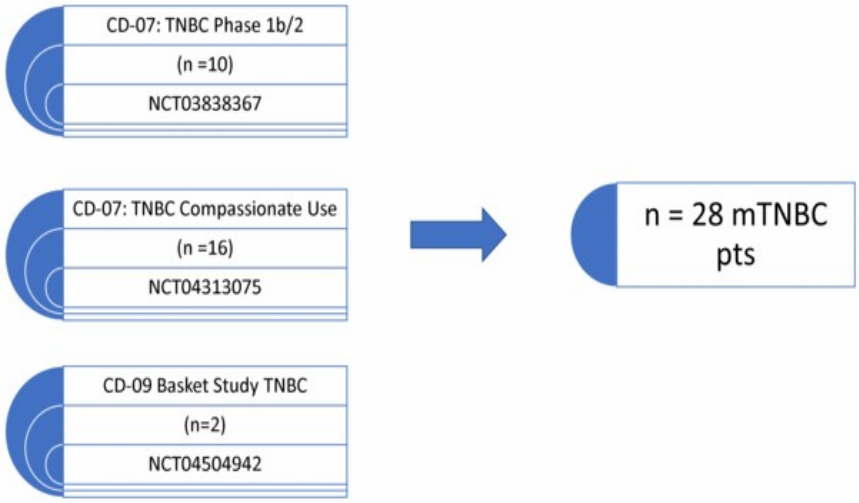




Immune Surveillance – T-regs - MDSC's, CCL5

Ward ST, Li KK, Hepburn E, Weston CJ, Curbishley SM, Reynolds GM, Hejmadi RK, Bicknell R, Eksteen B, Ismail T, Rot A, Adams DH. The effects of CCR5 inhibition on regulatory T-cell recruitment to colorectal cancer. Br J Cancer. 2015 Jan 20;112(2):319-28. doi: 10.1038/bjc.2014.572. Epub 2014 Nov 18. PMID: 25405854; PMCID: PMC4301825.

CytoDyn Leronlimab in Patients with mTNBC - Clinical Data



CytoDyn Results from CTC and CAML Studies in mTNBC

Changes in Circulating Tumor Associated Cells Predicts Progression Free and Overall Survival in Metastatic TNBC Patients after Induction with the Anti-CCR5 Drug Leronlimab

1. CCR5 quantification via CAMLs (Cancer Associated Macrophage-Like Cells) or CTCs (Circulating Tumor Cells) is possible in 100% of the patient population
2. The absence or drop in CTCs at T1 predicts improved survival.
3. Increases in TACs (Tumor-Associated Cells) at T1 significantly predicts for patients with worse survival.
4. Treatment with Leronlimab resulted in rapid decreases in TACs in 75% of patients which correlated with improved survival.

CytoDyn Results from CD-07: TNBC Phase 1b/2

A phase 1b/2 study of leronlimab combined with carboplatin in patients with CCR5+ metastatic Triple-Negative Breast Cancer (mTNBC)

1. Leronlimab, in combination with carboplatin, has been well-tolerated in all 3 dose levels
2. Leronlimab shows early signs of anti-tumor activity in patients with CCR5+ mTNBC.
3. The RP2D is weekly leronlimab 700 mg and 3-weekly carboplatin AUC5

CytoDyn Overall Conclusions for mTNBC Studies

Based on the analysis of 28 mTNBC patients treated with leronlimab who had failed ≥1 line of previous therapy and its historical comparison with SOC chemotherapy treatment or Sacituzumab Govitecan (SG) treatment, including those with brain or bone metastases:

1. The median overall survival for patients that received leronlimab + carboplatin was greater than 12 months, regardless of brain or bone metastases, and with more than one line of previous failed therapy, which is superior to standard of care chemotherapy (6.6 months) or SG (11.8 months)

2. The median overall survival for patients that received higher doses (≥525 mg) of leronlimab was 12+ months, regardless of brain or bone metastases, and with more than one line of previous failed therapy, which is also superior to standard of care chemotherapy and SG

3. The median progression-free survival for patients that received higher doses of leronlimab (≥525 mg) have significantly longer mPFS of 6.2 months. (95% CI: 2.6 months - 7.5 months) compared to standard of care chemotherapy in third line setting of 2.3 months (95% CI: 2.3 – 2.5 months) or compared to SG treatment (including patients with brain metastases) after first line setting of 4.8 months (95% CI: 4.1 months – 5.8 months).

4. 92% of the patients that had measurable lesions prior to start of leronlimab had stable disease or partial response in target legions after the 1st dose of leronlimab.

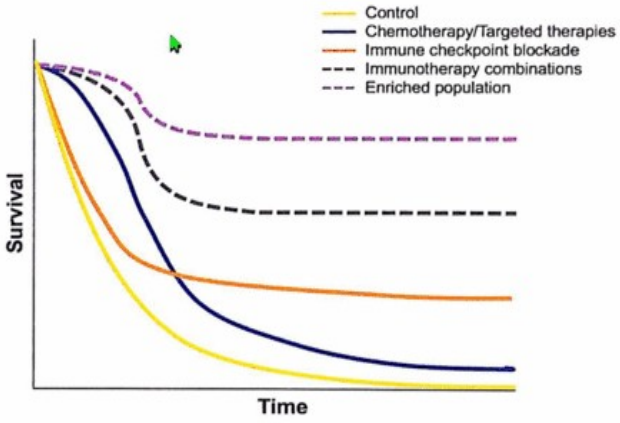
CytoDyn Hallmarks of Cancer: New Dimensions (Review)

***Source**: Hanahan: Cancer Discovery 2022; Hanahan Weinberg: Cell 2011; Hanahan Weinberg: Cell 2000*

Figure 6. Hallmarks of Cancer—new additions. Depicted are the canonical and prospective new additions to the "Hallmarks of Cancer." This treatise raises the possibility, aiming to stimulate debate, discussion, and experimental elaboration, that some or all of the four new parameters will come to be appreciated as generic to multiple forms of human cancer and hence appropriate to incorporate into the core conceptualization of the hallmarks of cancer.

CytoDyn Thank You!

"The Promise"



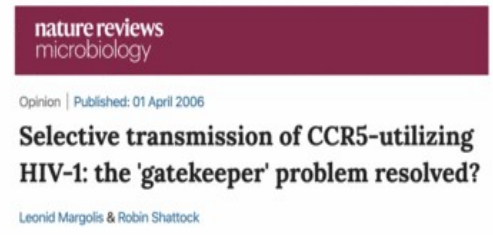
Ribas A. Presented at: BIT's 3rd Annual World Congress of Microbes (WCM); 30 July - 1 August 2013: Wuhan, China. Ribas A, et al. *Clin Cancer Res.* 2012;18(2):336-341. Drake CG. *Ann Oncol.* 2012;23(suppl 8):viii41–viii46.

CytoDyn

CCR5 in HIV Prevention and Cure

Jonah B. Sacha, Ph.D.
Oregon Health & Science University

CytoDyn CCR5 is Critical in Sexual Transmission of HIV

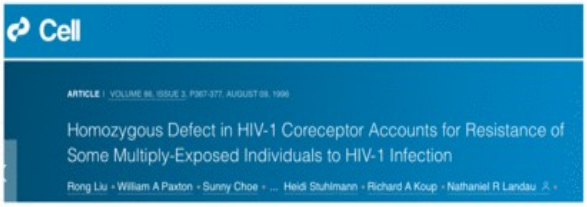


nature reviews microbiology

Opinion | Published: 01 April 2006

Selective transmission of CCR5-utilizing HIV-1: the 'gatekeeper' problem resolved?

Leonid Margolis & Robin Shattock



Cell

ARTICLE | VOLUME 86, ISSUE 3, P367-377, AUGUST 09, 1996

Homozygous Defect in HIV-1 Coreceptor Accounts for Resistance of Some Multiply-Exposed Individuals to HIV-1 Infection

Rong Liu • William A Paxton • Sunny Choe • ... Heidi Stuhlmann • Richard A Koup • Nathaniel R Landau

LETTERS TO NATURE

Resistance to HIV-1 infection in caucasian individuals bearing mutant alleles of the CCR-5 chemokine receptor gene

Michel Samson*, Frédérick Libert*, Benjamin J. Doranz†, Joseph Rucker†, Corinne Liesnard‡, Claire-Michèle Farber§, Sentob Saragosti‖, Claudine Lapouméroulie¶, Jacqueline Cognaux#, Christine Forceille#, Gaetan Muyldermans#, Chris Verhofstede#, Guy Burtonboy#, Michel Georges☆, Tsuneo Imai**, Shalini Rana††, Yanji Yi††, Robert J. Smyth††, Ronald G. Collman††, Robert W. Doms†, Gilbert Vassart*‡‡ & Marc Parmentier*

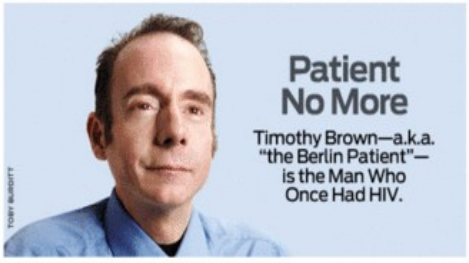
REPORTS

Genetic Restriction of HIV-1 Infection and Progression to AIDS by a Deletion Allele of the *CKR5* Structural Gene

Michael Dean*, Mary Carrington*, Cheryl Winkler, Gavin A. Huttley, Michael W. Smith, Rando Allikmets, James J. Goedert, ...

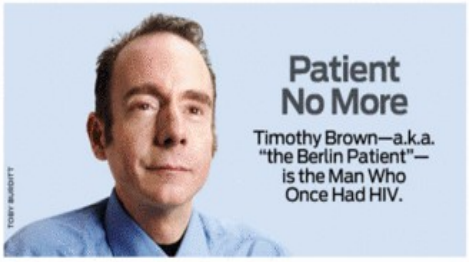
Hemophilia Growth and Development Study, Multicenter AIDS Cohort Study, Multicenter Hemophilia Cohort Study, San Franci

CytoDyn CCR5 in HIV Cure



"Berlin patient"
Timothy Brown
HIV remission 13 years

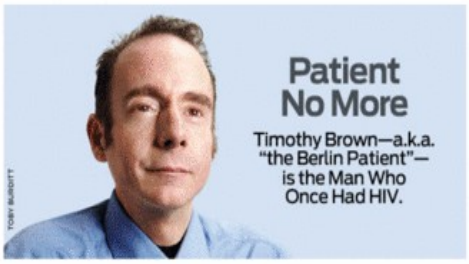
CytoDyn CCR5 in HIV Cure



"Berlin patient"
Timothy Brown
HIV remission 13 years

"London patient"
Adam Castillejo
HIV remission >4 years

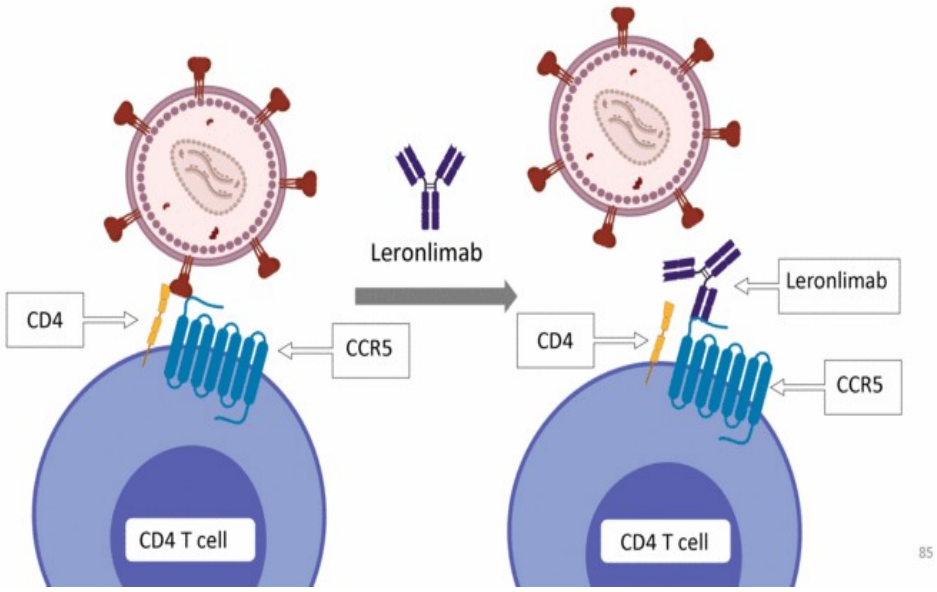
CytoDyn CCR5 in HIV Cure



IMPAACT
International Maternal Pediatric Adolescent AIDS Clinical Trials Network

IMPAACT P1107 Team Presents First Known Case of a Woman with HIV Remission

How to Mimic CCR5$^{\Delta 32/\Delta 32}$ Phenotype?

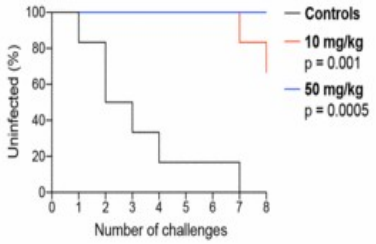


CytoDyn Leronlimab as PrEP for HIV

nature communications

Antibody-based CCR5 blockade protects Macaques from mucosal SHIV transmission

Xiao L. Chang, Gabriela M. Webb, ... Jonah B. Sacha + Show authors



Leronlimab can pharmacologically mimic CCR5$^{\Delta 32/\Delta 32}$ phenotype

CytoDyn Long-Acting Injectables for PrEP

Science

Long-Acting Integrase Inhibitor Protects Macaques from Intrarectal Simian/Human Immunodeficiency Virus

Chasity D. Andrews,[1] William R. Spreen,[2] Hiroshi Mohri,[1] Lee Moss,[2] Susan Ford,[2] Agegnehu Gettie,[1] Kasi Russell-Lodrigue,[3] Rudolf P. Bohm,[3] Cecilia Cheng-Mayer,[1] Zhi Hong,[2] Martin Markowitz,[1] David D. Ho[1]*

FDA NEWS RELEASE

FDA Approves First Injectable Treatment for HIV Pre-Exposure Prevention

Drug Given Every Two Months Rather Than Daily Pill is Important Tool in Effort to End the HIV Epidemic

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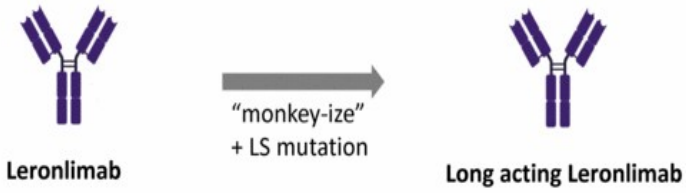
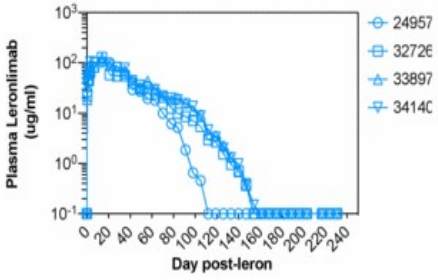


Leronlimab

Long acting Leronlimab

CytoDyn Long-Acting Leronlimab for PrEP?



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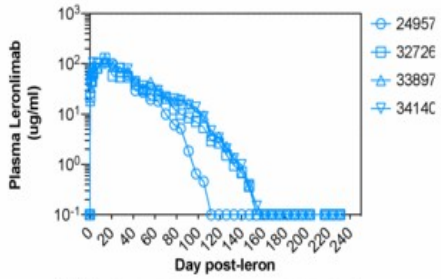
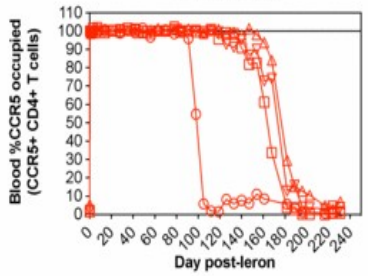
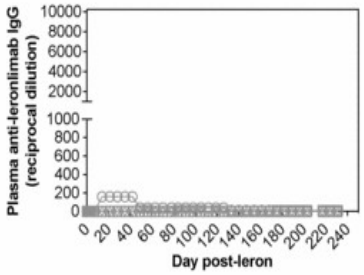
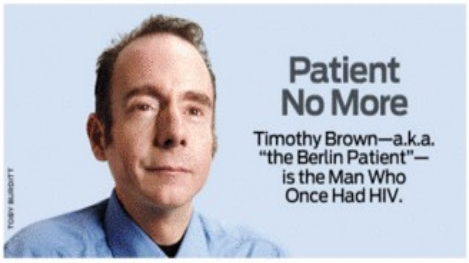






CytoDyn CCR5 in HIV Cure



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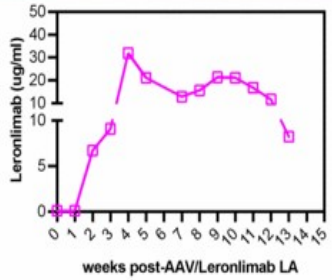
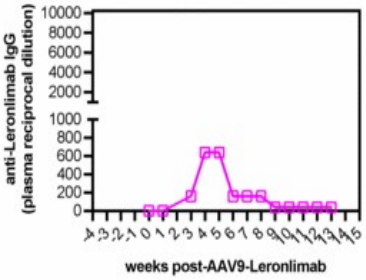
IMPAACT P1107 Team Presents First Known Case of a Woman with HIV Remission

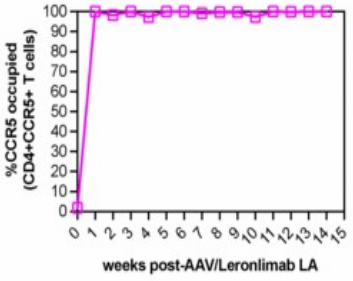
GEN Genetic Engineering & Biotechnology News

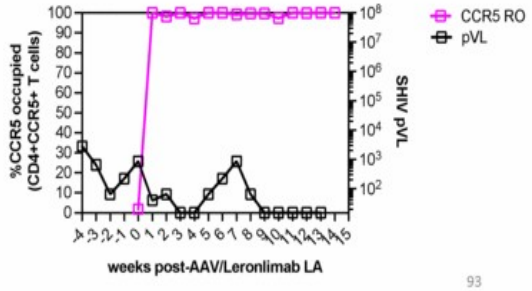
Mary Ann Liebert, Inc. publishers

CytoDyn Long-Acting Leronlimab for Functional Cure?



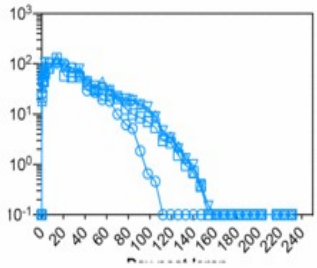




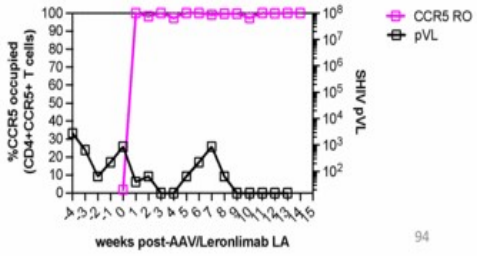


CytoDyn Potential of Long-Acting Leronlimab for HIV

1. Prevention of HIV infection



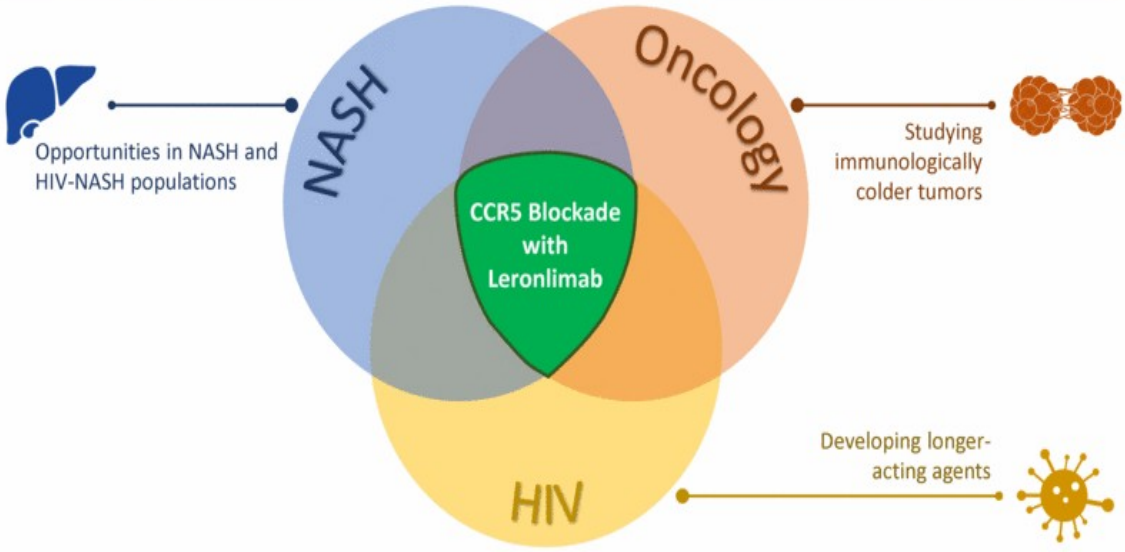
2. Functional cure (control of viremia)



CytoDyn

Closing Remarks

CytoDyn Refocusing Pipeline on High Value Opportunities



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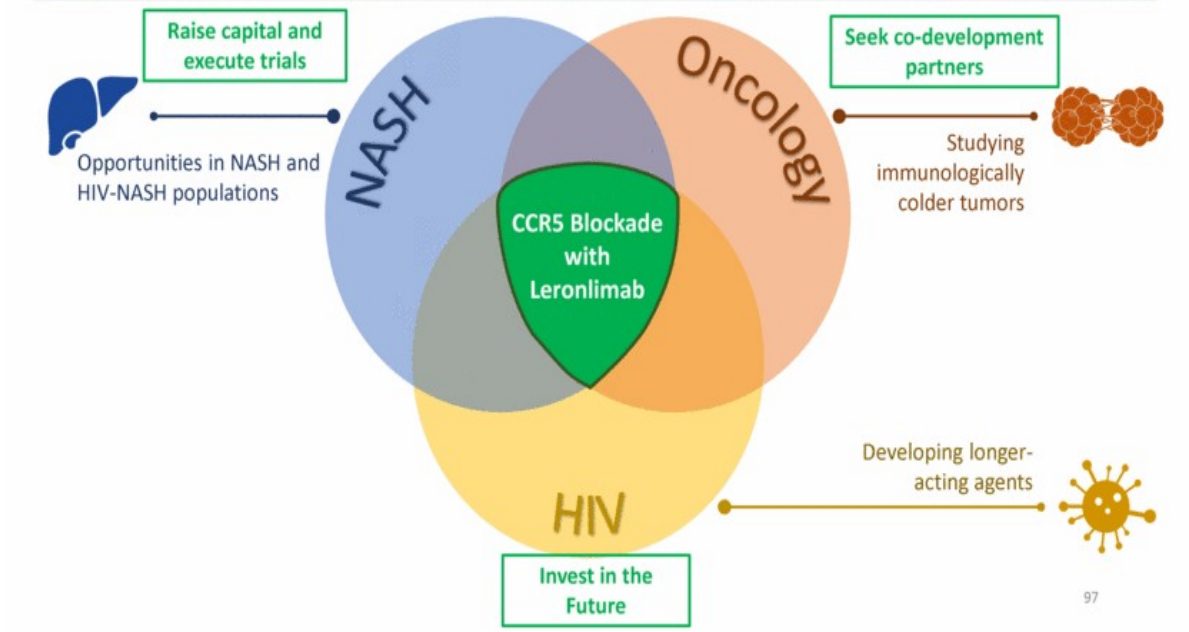
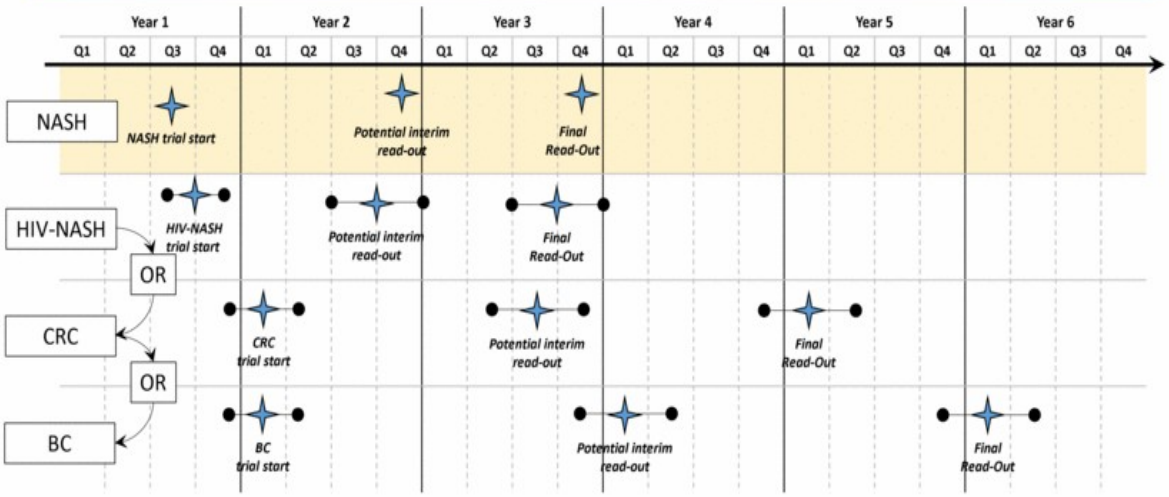


CytoDyn Potential Timeline for Clinical Development*



NASH will be the priority, other indications (NASH-HIV, CRC, and BC) are funding dependent

*** Non-Binding timeline projection, events are based on financing, regulatory, and other considerations***

What We Expect in 2023

- Lifting of the Partial Clinical Hold in HIV
- Financing to fund operations and achieve value inflection
- Initiating a NASH trial
- Invest in and advance a long-acting CCR5 molecule
- Continued contributions in medical meetings and peer-reviewed publications
- Continuing to reshape of our team and capabilities to meet our goals
- Corporate Rebranding

CytoDyn's Mission