CytoDyn Inc.

OCTOBER 2020

CORPORATE PRESENTATION

CYDY

The pursuit of precision medicine
Humanized Monoclonal Antibody

Forward Looking Information and 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 specifically include statements about leronlimab, its ability to have positive health outcomes, 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 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.

Robust Pipeline

HIV, COVID-19, Cancer, NASH, MS, Stroke, and various Autoimmune Diseases

Designation	Program	Trial Status	Potential Timeline
FTD[1] - RR[2]	**HIV** - USA	BLA submission	2020
	HIV - UK	Pre-BLA meeting	October 26, 2020
	HIV - EU	Requested pre-BLA meeting	2020
	HIV - Canada	Requested pre-BLA meeting	November 9, 2020
	HIV- Monotherapy (Phase 3)	One dose/week	2020
	HIV - PrEP (Phase 2)	One dose/month	2021
	HIV – Cure (Phase 2)	5 patients/Timothy Brown model	2020-2021
	COVID-19 – Severe-to-Critical (Phase 3)	Interim Analysis	October 2020
	COVID-19 – Mild/Moderate(Phase 2)	Completed – Publication	2020
	COVID-19 - Moderate (Phase 3)	FDA discussions underway	Initiate 2020
	COVID-19 - Long Hauler (Phase 2)	File synopsis of the protocol w/FDA	October 2020
FTD[1]	**Cancer** - mTNBC (Phase 1b/2)	Enrolling-BTD discussions	2020
	Cancer - Basket trial (Phase 2)	Enrolling-BTD discussions	2020
ODD[3]	**GvHD** (Phase 2)	Enroll 5 more patients-BTD	2020
	NASH (Phase 2)	First patient enrolled	Nov. 2020
	MS (Phase 2)	Protocol & IND submission	2020
	Stroke (Phase 2)	Protocol & IND submission	2020

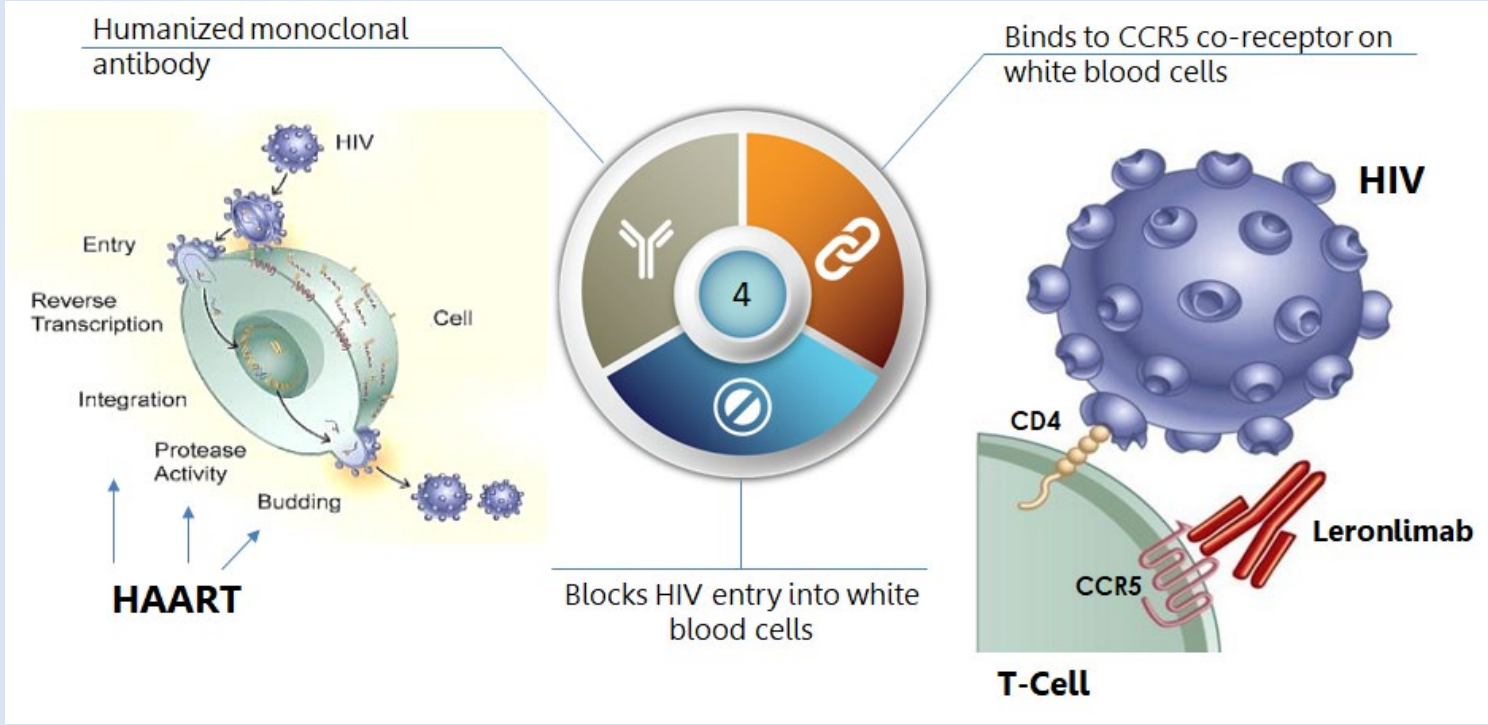
[1]FTD Fast Track Designation [2]RR Rolling Review BLA [3]ODD Orphan Drug Designation

HIV

Mechanism of Action

The Next Generation of Monoclonal Antibody Therapy

CytoDyn is committed to enhancing the lives of patients through target specific medicine. Our team is focused on developing leronlimab, a monoclonal antibody CCR5 receptor antagonist, to be used as a platform drug for a variety of indications.



How it Works

The target of leronlimab (PRO 140) is the important immunologic receptor CCR5. The CCR5 receptor is a protein located on the surface of a variety of cells including white blood cells and cancer cells. On white blood cells, it serves as a receptor for chemical attractants called chemokines. The CCR5 receptor is also the coreceptor needed for HIV to infect healthy T-cells. Recent research has identified the CCR5 receptor as an important target for many disease processes, including cancer metastasis and certain immunological conditions.

HIV

Phase 3 Primary Endpoint Achieved

Leronlimab in combination with HAART

(**H**ighly **A**ctive **A**nti**R**etroviral **T**herapy)

- Leronlimab has received Fast-Track Designation, and "accelerated approval is possible" (FDA)
- Phase 3 pivotal trial completed and hit primary end point ***p=0.0032***
- 24 weeks of safety data. **81%** of patients w/ suppressed viral load compared to 43% (per last approved drug for this population)
- No serious site injection reactions reported in more than 800 patients treated with leronlimab for HIV. No drug-related SAEs reported in patients treated with up to 700mg dose of leronlimab.
- BLA submission potentially in 2020 and has already been granted "**rolling review**" by FDA – Will apply for priority review

Label submission to be requested:

- **One drug resistance in 3 classes; or**
- **One drug resistance in 2 classes with limited treatment options to another class**

HIV

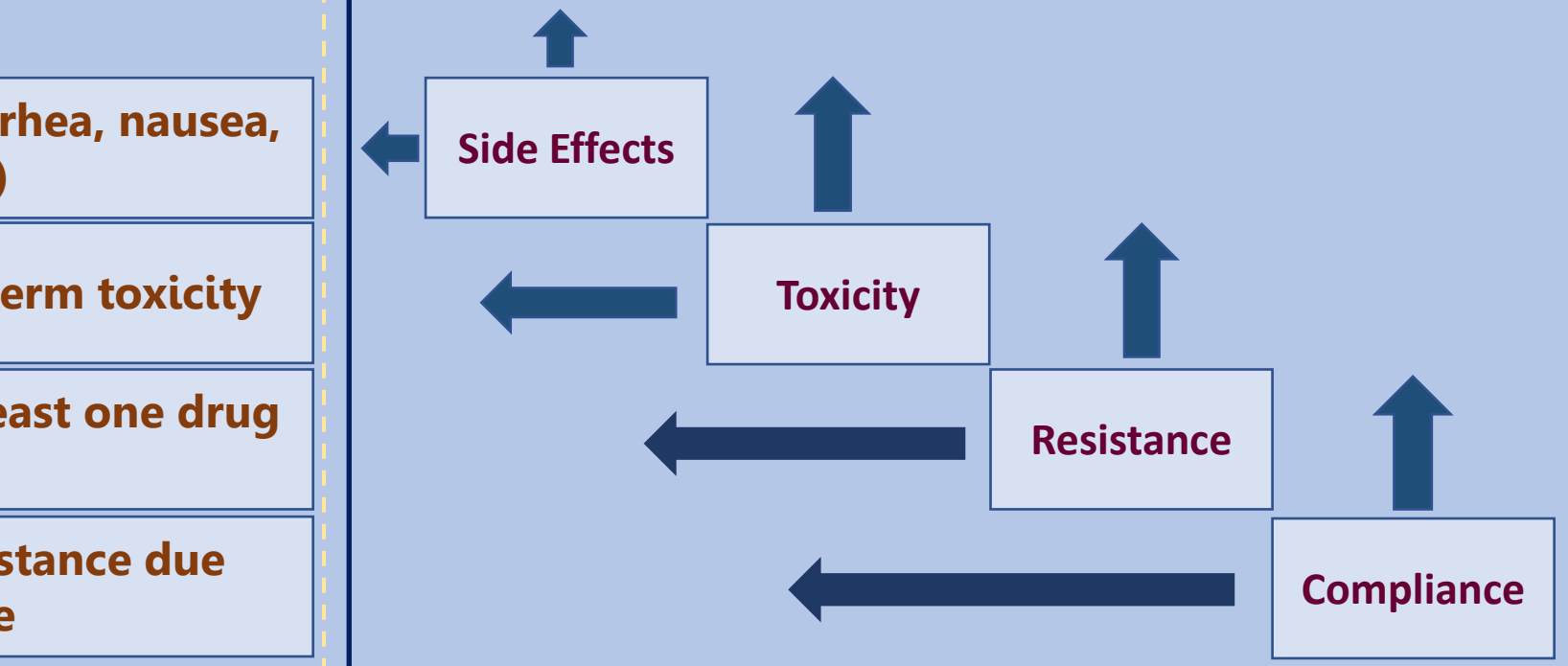
Phase 3 Combination Therapy & Phase 3 Monotherapy

leronlimab

- Hardly any side effects in more than 800 patients
- Negligible toxicity in more than 800 patients
- No drug resistance in patients on monotherapy, a few over 6 years
- Weekly, simple, subcutaneous self administered

HAART

- Ranges from mild to severe (Diarrhea, nausea, lethargy, depression)
- Problems with short- and long-term toxicity
- 76% of HIV patients develop at least one drug resistance
- Daily lifetime dosing with resistance due mainly to compliance



Completed a Phase 3 Investigative Trial – Previously Completed a Successful Phase 2 Trial

- Objective of trial: Assess the subcutaneous use of leronlimab as a long-acting single agent maintenance therapy for 48 weeks in patients with suppressed viral load with CCR5-tropic HIV-1 infection
- Primary Endpoint: Proportion of participants with a suppressed viral load to those who experienced VF
- Secondary Endpoint: Length of time to VF (virologic failure)

STATUS

- Enrollment closed after reaching 565 patients
- Trial was also used to provide safety data for BLA filing for leronlimab as a combination therapy
- Higher responder rate prompted us to file a Phase 3 pivotal trial protocol with the FDA for leronlimab monotherapy as “switch therapy”
- In discussion with FDA to finalize protocol, after which we expect to initiate the Phase 3 trial if the FDA gives green light to initiate

215 patients approached 1 year of suppressed viral load and over 40 patients average of 2.5 years, with 5 patients nearly 6 years

HIV

Monotherapy, PrEP and Cure

Program	Status
HIV- Monotherapy	Phase 3, label expansion, protocol submitted to the US FDA; will also be presenting the protocol to EMA, Canada, UK regulatory agencies and pursue approvals.
HIV - PrEP	One dose/month – Phase 2 – Animal study was very successful for use of leronlimab in PrEP for one dose per month. Publication has been submitted to journal and potential Phase 2 is to initiate in early 2021.
HIV - Cure	5 patients/Timothy Brown model – Phase 2 – This trial is currently searching for potential HIV patient who is in need of bone marrow transplant similar to Timothy Brown. If leronlimab can mimic the delta-32 during a bone marrow transplant by covering all the CCR5 receptor, then the result could be a cure. First patient injection could be 2020 or early 2021.

COVID-19

Phase 2 (Mild-to-Moderate) – Completed

86 patients were randomized, but only 84 received treatment
Two populations for all the analysis (mITT[1] and PP[2])
mITT = 84 patients: 56 leronlimab vs 28 placebo
PP = 69 patients: 46 leronlimab vs 23 placebo

Mild-to-Moderate CD10 Trial	n	Control	Leronlimab
Modified Intent to Treat population	84	28	56
Baseline Total Symptom Score ≥4	45	15	30
Baseline Total Symptom Score <4 to ≥1	31	10	21
Baseline Total Symptom Score = 0	8	3	5

Leronlimab	Placebo
5 patients had 8 SAEs	6 patients had 11 SAEs
5 patients out of 56 ~ 9%	6 patients out of 28 ~ 21%
8 SAE among 56 ~ 14%	11 SAEs among 28 ~ 39%
96 AEs events in both arm ~ 33.9%	96 AEs events in both arm ~ 50%

Leronlimab	Placebo
At Day 3 - mITT Patients had improvement ~ 63%	At Day 3 - mITT Patients had improvement ~ 56%
At Day 3 – PP Patients with Total Symptom score ≥ 4 Improved ~ 90%	At Day 3 – PP Patients with Total Symptom score ≥ 4 Improved ~ 71%
At Day 14 – PP Patients with Total Symptom score ≥ 4 Improved ~ 96.3%	At Day 14 – PP Patients with Total Symptom score ≥ 4 Improved ~ 92.9%

Leronlimab	Placebo
At Day 3 - mITT Patients had improvement ~ 63%	At Day 3 - mITT Patients had improvement ~ 56%
At Day 3 – PP Patients with Total Symptom score ≥ 4 Improved ~ 90%	At Day 3 – PP Patients with Total Symptom score ≥ 4 Improved ~ 71%
At Day 14 – PP Patients with Total Symptom score ≥ 4 Improved ~ 96.3%	At Day 14 – PP Patients with Total Symptom score ≥ 4 Improved ~ 92.9%

NEWS2

National Early Warning Score 2

Based on: Respiration rate, oxygen saturation, any supplemental oxygen, temperature, systolic blood pressure, heart rate, level of consciousness

mITT[1] –	Day 3	Leronlimab ~ 38%	vs Placebo ~ 16%	$p = 0.0675$
mITT –	Day 14	Leronlimab ~ 50%	vs Placebo ~ 21%	$p = 0.0223$
PP[2] –	Day 3	Leronlimab ~ 42%	vs Placebo ~ 14%	$p = 0.0282$
PP –	Day 14	Leronlimab ~ 55%	vs Placebo ~ 23%	$p = 0.0185$

Leronlimab	Placebo
Incidences of hospitalization ~ 1.79%	Incidences of hospitalization ~ 10.71%
Need for mechanical ventilation 1/56 ~ 1.79%	Need for mechanical ventilation 1/28 ~ 3.57%
Did not need oxygen use ~ 83.93%	Did not need oxygen use ~ 78.57%

[1] mITT – Modified Intent To Treat

[2] PP – Per Protocol

COVID-19

Phase 3 (Severe-to-Critical), Phase 2 (Long Hauler) and Phase 3 (Moderate)

Program	Trial Status
COVID-19 – Severe-to-Critical	Interim Analysis – If interim analysis is successful then potential for approval for leronlimab in 2020.
COVID-19 – Mild/Moderate (Phase 2)	Completed – Publication to be submitted to a journal in October 2020.
COVID-19 - Moderate (Phase 3)	Discussions underway with the US FDA.
COVID-19 - Long Hauler (Phase 2)	File synopsis of the protocol w/FDA in October 2020 and plan to initiate trial in 2020 and completion in late 2020 or early 2021.

CANCER

Mechanism of Action

Potential role of leronlimab in Cancer
CCR5 is highly expressed in glioblastoma and is associated with poor prognosis of patients. CCL5/CCR5 is suggested to be an excellent new target for glioblastoma therapy. The molecular mechanisms, by which chemoattractant and receptor respond within the complex tissue microenvironment to promote cancer stem cells and tumour heterogeneity, should be considered in forthcoming studies." https://pubmed.ncbi.nlm.nih.gov/31747383/
"These results indicate that the expression of RANTES is directly correlated with a more advanced stage of disease, suggesting that RANTES may be involved in breast cancer progression. Moreover, it is possible that in patients diagnosed with benign breast disorders, RANTES expression may be indicative of an ongoing, but as yet undetectable, malignant process." https://cancerres.aacrjournals.org/content/59/18/4681.short
"Pathologic expression of CCR5 upon cellular transformation occurs in many types of cancer (**Fig. 1C**). CCR5 expression induced by transformation imbues the cell with dramatic alteration in gene expression, motility, and homing behavior to metastatic sites." https://cancerres.aacrjournals.org/content/79/19/4801
"CCL5 exerts proangiogenic effects by promoting endothelial cell migration, spreading, neovessel formation, and vascular endothelial growth factor (VEGF) secretion. Moreover, tumor cells, upon CCL5 stimulation, can produce VEGF or, by secreting CCL5, may recruit CCR5-expressing TAMs [**19**,**34**]. In turn, by secreting VEGF, TAMs can induce angiogenesis [**18**,**30**,**35**]. Thus, targeting tumor-promoting TAMs, which are now considered to be the major players in the regulation of tumor angiogenesis, may represent an attractive new therapeutic strategy." https://www.mdpi.com/1422-0067/19/5/1477/htm

Cancer

Clinical Trials Update Cancer – Phase 1b/2 and Phase 2 Basket Trial

Basket Trial
11 enrolled - 70 Screened
9 Pending eligibility
1 site (5 sites in selection process)

Compassionate Use – mTNBC
14 enrolled - 66 screened
16 pending eligibility
2 sites (4 sites in selection process)

Phase 1b/2 – mTNBC
3 enrolled – 3 screened
6 sites (8 sites in selection process)

eIND – Any stage 4 cancer
1 patient

Breakthrough Therapy Designation
Need CT scan/MRI results (CTC-CAML)-Currently analysis is being performed

Breakthrough Therapy Designation

mTNBC (6 months with Carboplatin + Leronlimab) – BTD requires at least 5 patients, we have one

GvHD

Mechanism of Action – Phase 2 Update

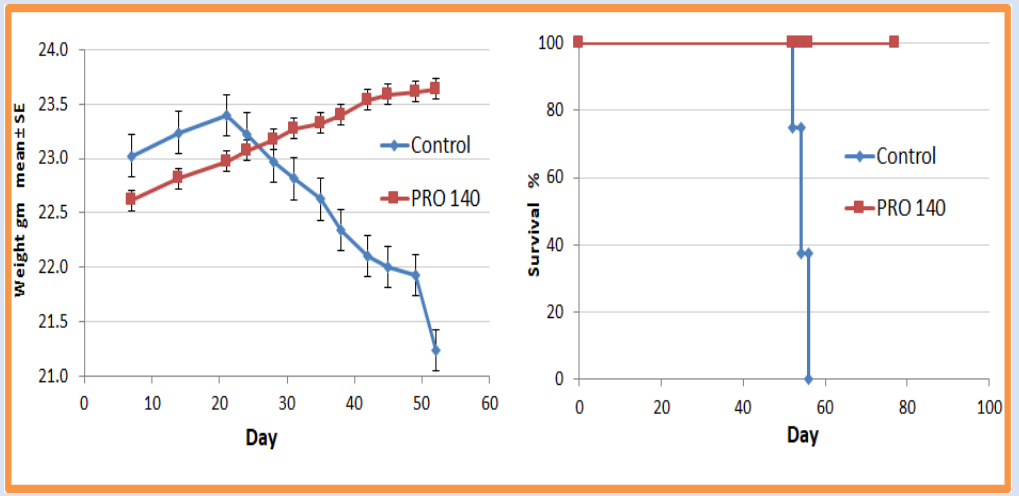
Potential role of leronlimab in GvHD
"Longer follow-up reveals a sustained reduction in acute GVHD incidence in maraviroc-treated patients compared with the control cohort, with a stronger effect on visceral vs skin GVHD and importantly no adverse impact on disease relapse, infections, or immune recovery. Thus, these data add further support that CCR5 blockade protects against GVHD." https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5314813/
"Importantly, although CCR5 deficiency affects lymphocyte trafficking to target tissues, T cells would still be able to recognize pathogen-derived antigens.[5] Furthermore, humans with CCR5 deficiency are not grossly susceptible to infections, and in fact, we observed no increase in infection rate with maraviroc in our study. This suggests that maraviroc can dampen alloreactive T-cell responses while not impairing immunity against infections." https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5314813/
"CCR5 is a marker for GVHD effector cells and that $CCR5^+$ T cells are active participants in the pathogenesis of human acute GVHD." https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3182111/

GvHD (Graft vs. Host Disease)

- FDA granted Orphan Drug Designation (ODD)
- Xeno-GvHD Human BM transplanted into Immuno-Deficient mice

  Study was published in peer-reviewed publication:
  "Biology of Blood and Marrow Transplantation"

- Phase 2 trial initiated
- Amended protocol for Phase 2 trial received IRB approval
- Patient enrollment underway



Designation	Program
Orphan Drug Designation	GvHD (Phase 2) – Need 5 patients in this open label trial. With very positive results we can apply for breakthrough designation

NASH

Mechanism of Action

Potential role of leronlimab in NASH
"CCR5 plays a central role in all the events related to liver matrix remodeling and it has been observed that patients with chronic liver disease present high levels of CCR5 and CCL5." "Our result suggests that in early NASH, HSCs secrete Ccl5 which contributes to a broad array of mechanisms by which hepatic steatosis and inflammation are achieved." "Our data indicate that chemokine (C-C motif) ligand 5 (Ccl5, a.k.a. Rantes) is one of the HSC-secreted mediators in NASH that directly induce steatosis and pro-inflammatory factors in initially healthy hepatocytes." **From Dr. Ken Sherman:** "It is possible that someday all patients with HIV may be treated with a blocking agent as part of their HIV drug cocktail designed to protect the liver and regain and maintain liver health," Dr. Ken Sherman suggests. https://www.nature.com/articles/s41598-018-25699-9

Program	Trial Status
NASH (Phase 2)	First patient enrollment expected November 2020

MS

Mechanism of Action – Status of Trial

Potential role of leronlimab in MULTIPLE SCLEROSIS (MS)
"Thus, chemokines appear to be associated with MS and an increased chemokine expression may further enhance disease progression by attracting more leukocytes into the brain parenchyma and by activation of effector functions of astrocytes and microglial cells." https://onlinelibrary.wiley.com/doi/full/10.1046/j.1365-2249.2000.01334.x
"Individuals homozygous for a polymorphism in the CCR5 gene (CCR5D32) do not express a functional receptor, and although they are not protected from MS, they do exhibit a later age of disease onset and a lower risk of clinical recurrent disease activity." https://www.rndsystems.com/resources/articles/chemokine-receptors-and-multiple-sclerosis-pathogenesis
"Both MIP-1β as well as RANTES were found to be significantly elevated in brain tissue of MS patients." https://onlinelibrary.wiley.com/doi/full/10.1046/j.1365-2249.2000.01334.x
"CCR5 expression was increased during relapse, compared with control individuals. During remission, CCR5 values decreased, suggesting an association of $CCR5^+$ T cells with disease activity." https://jamanetwork.com/journals/jamaneurology/fullarticle/780942
"The cerebrospinal fluid (CSF) of patients with relapsing-remitting MS has $CCR2^+CCR5^+$ T_H1 cells during a relapse; $CCR5^+CD8^+$ T cells and $CCR5^+$ monocytes are higher in the CSF than in the blood of patients with the disease, and CCR5 is expressed in inflammatory cells infiltrating the CNS *in vivo* (17, 18). CCR5 is also expressed on immune cells within inflammatory lesions in MS and may contribute to recruitment of these cells to the inflamed tissue or to their activation. Finally, the expression of CCR5 ligands has been shown at sites of inflammation in MS (19). Interestingly, MS can develop in people who are homozygous for the CCR5Δ32 mutation. The CCR5Δ32 allele is not associated with MS risk (20, 21), but the disease seems to be less severe in carriers of the allele (22), suggesting that CCR5 antagonists might diminish disease activity." https://www.frontiersin.org/articles/10.3389/fimmu.2017.01981/full

Program	Trial Status
MS (Phase 2)	Protocol & IND submission to the FDA (US) by end of 2020. First patient enrollment in early 2021

Stroke

Mechanism of Action

Potential role of leronlimab in Stroke and Traumatic Brain Injury
"CCR5 is uniquely expressed in cortical neurons after stroke." "Post-stroke neuronal knockdown of CCR5 in premotor cortex leads to early recovery of motor control." "In a large clinical cohort of stroke patients, carriers for a naturally occurring loss-of function mutation in CCR5 (CCR5-D32) exhibited greater recovery of neurological impairments and cognitive function." "CCR5 is a translational target for neural repair in stroke and TBI and the first reported gene associated with enhanced recovery in human stroke." "Stroke and traumatic brain injury (TBI) are the leading causes of adult disability due to limited neurological recovery. Approximately 50%–60% of patients continue to experience motor impairments after stroke (Schaechter, 2004). 43% of those hospitalized for TBI suffer long-term disability (Ma et al., 2014)." "There have been no medical therapies developed to promote recovery in these conditions." https://www.sciencedirect.com/science/article/pii/S0301008204000565
CCR5 is differentially upregulated in neurons after stroke. • Knockdown of CCR5-induces motor recovery after stroke and improves cognition after TBI • Treatment with an FDA-approved drug, maraviroc induces recovery after stroke and TBI • Human carriers for CCR5delta32 have better outcomes after stroke • There have been no medical therapies to promote recovery in TBI and stroke.

Current trial status with leronlimab

One patient treated– Very strong anecdotal data

Autoimmune Diseases

Mechanism of Action

Potential role of leronlimab in AUTOIMMUNE DISEASES
"CCR5 may also have a role in autoimmune diseases. In rheumatoid arthritis, increased levels of CCR5 ligands CCL3, CCL4, and CCL5 are found in the synovial fluid (37, 38), and the CCR5Δ32 variant seems to protect from the disease (39). https://www.frontiersin.org/articles/10.3389/fimmu.2017.01981/full
"The predominance of CCR5-positive mononuclear cells in the synovial effusions of patients with arthritis suggests an important role for CCR5 in the process of joint inflammation, and identifies CCR5 as a possible new target for therapeutic intervention." https://onlinelibrary.wiley.com/doi/abs/10.1002/1529-0131(199905)42:5%3C981::AID-ANR17%3E3.0.CO;2-4
"CCL5 expression is increased in inflammatory bowel disease (IBD), likely pointing to a contribution by CCL5 in the progressive tissue destruction during the inflammatory processes (45). A recent investigation provided evidence that blocking CCR5 either by genetic ablation or by pharmacological inhibition with maraviroc rescued mice from colitis in both acute and chronic models (46)." https://www.frontiersin.org/articles/10.3389/fimmu.2017.01981/full
"In summary, CCR5 regulates recruitment of blood leukocytes into the colon indicating that targeting CCR5 may offer therapeutic options in IBDs." https://www.nature.com/articles/srep30802

Sepsis and Seizures

Mechanism of Action

Potential role of leronlimab in SEPSIS

CCR5-deficient mice are largely resistant to lethal *S. aureus* infection, highlighting the importance of CCR5 targeting in *S. aureus* pathogenesis. Thus, depletion of $CCR5^+$ leukocytes by LukED suggests a new immune evasion mechanism of *S. aureus* that can be therapeutically targeted.
https://www.nature.com/articles/nature11724

Potential role of leronlimab in SEIZURES

"Decrease in CCR5 in circulating cells strongly protected from excitotoxin-induced seizures, BBB leakage, CNS injury, and inflammation, and facilitated neurogenic repair."
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3023386/

Leronlimab Manufacturing

Samsung BioLogics (Commercial Partnership)

- Deal signed April 2019
- Build-out in process
- Targeted minimum capacity commitment will be reached by end of 2020, with additional capacity as needed. First delivery of clinical grade leronlimab is targeted early 3Q20

Management Team

Board of Directors

Executive Management Team

Dr. Nader Z. Pourhassan, Ph.D.

President, CEO and Director - 12 years at CytoDyn, founded leronlimab opportunity, purchased the whole program, changed its development path and initiated all its new potentials. Ph.D. in Mechanical Engineering with exceptional background in business development.

Dr. Scott A. Kelly, M.D.

Chief Medical Officer, Head of Business Development and Chairman of the Board

Michael D. Mulholland

Chief Financial Officer- Over 30 years of senior financial leadership roles with public companies across several industries.

Dr. Nitya G. Ray, Ph.D.

Chief Technology Officer, Head of Process Sciences, Mfg & Supply Chain- Over 30 years experience in biologics manufacturing, including engineered tissue therapeutics, antibody drug conjugates, and small molecule drugs.

Dr. Brendan Rae, Ph.D., J.D.

Senior Vice President of Business Development- Accomplished licensing and business development executive. Experience as an attorney focused on biopharma intellectual property law.

Arian Colachis, J.D.

Vice President, General Counsel, Corporate Secretary – Over 30 years of experience in regulatory, compliance, litigation and corporate matters.

Board of Directors

Dr. Scott A. Kelly, M.D.

Chairman of the Board, Chief Medical Officer and Head of Business Development

Dr. Nader Z. Pourhassan, Ph.D.

Director, President and CEO

Jordan G. Naydenov

Director

Alan P. Timmins

Director

Dr. Samir R. Patel, M.D.

Director