August 2021

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 provide 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 regulatory determinations of leronlimab's efficacy to treat human immunodeficiency virus ("HIV") patients with multiple resistance to current standard of care, COVID-19 patients, and metastatic Triple-Negative Breast Cancer ("mTNBC"), among other indications, by the U.S. Food and Drug Administration 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 obligations; (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) legal proceedings, investigations or inquiries affecting the Company or its products; (xiii) general economic and business conditions; (xiv) changes in foreign, political, and social conditions; (xv) stockholder actions or proposals with regard to the Company, its management, or its board of directors; and (xvi) 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 presentation.

Robust Pipeline

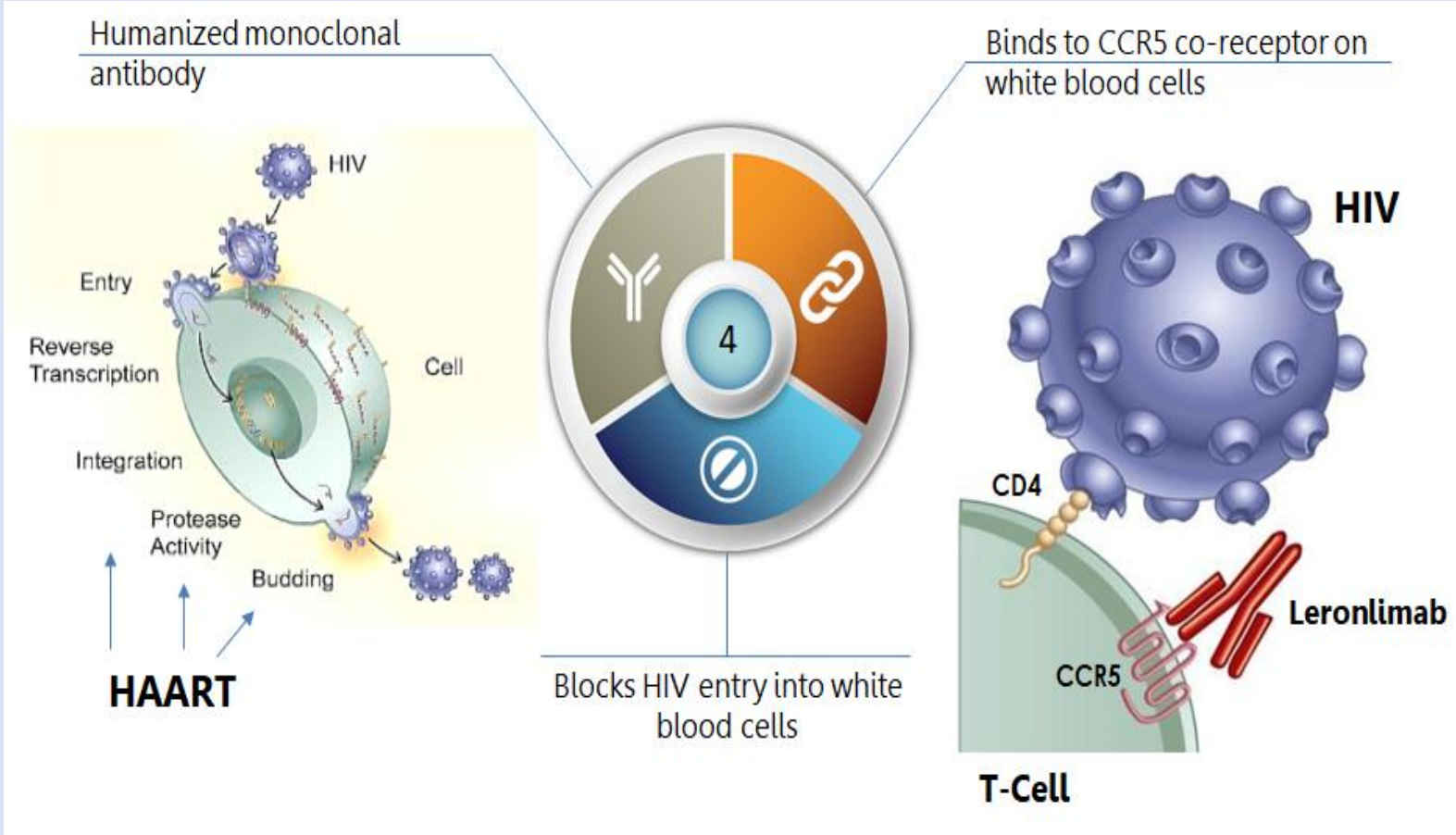
HIV, COVID-19, Cancer, NASH, MS, Stroke, and various Autoimmune Diseases

Indication	Program for 2021
HIV	BLA submission-USA, UK, EU, Canada and Brazil
HIV	Protocol submission for label expansion - Monotherapy (Phase 3)
HIV	Protocol submission - PrEP (Phase 2)
HIV	First patient dose - Cure (Phase 2)
COVID-19	Interim analysis from Severe population in Brazil
COVID-19	Interim analysis from Critical population in Brazil
COVID-19	Long Hauler protocol – Enrollment completion
Cancer	Cancer - mTNBC (Phase 1b/2) – Breakthrough designation application to file
Cancer	Cancer - Basket trial (Phase 2) – Breakthrough designation application to file
NASH	NASH (Phase 2)
MS-Alzheimer-Stroke	Phase 2 – Protocol submission
HIV	Manuscript submission to publication – CD02 (combination therapy)
COVID-19	Manuscript submission to publication - CD10 (mild-to-moderate trial)
COVID-19	Manuscript submission to publication - CD12 (severe-to-critical trial)
COVID-19	Manuscript submission to publication - CD15 (long-haulers trial)

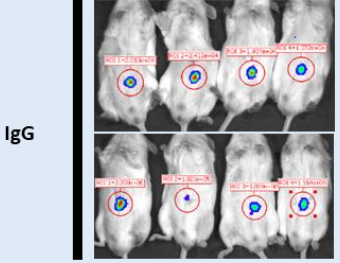
HIV - Mechanism of Action

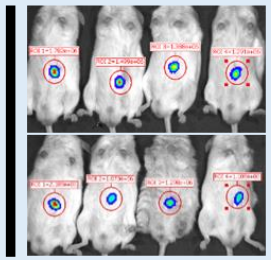
How it Works

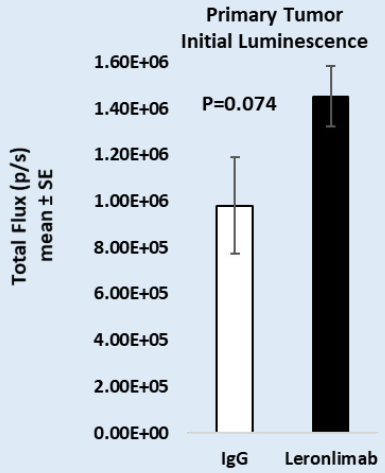
The target of leronlimab (PRO 140) is the important immunologic receptor CCR5. The CCR5 receptor is a protein located on the surface of a variety of cells including white blood cells and cancer cells. On white blood cells, it serves as a receptor for chemical attractants called chemokines. The CCR5 receptor 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.

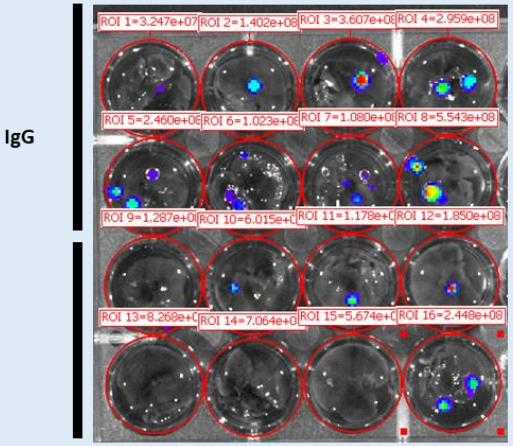


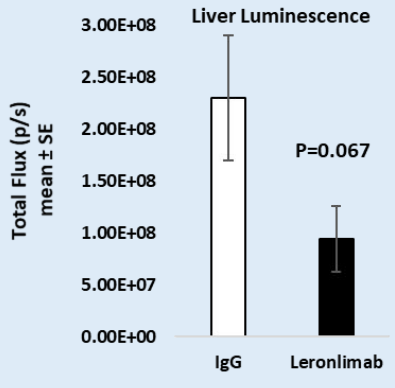
Cancer - Mechanism of Action

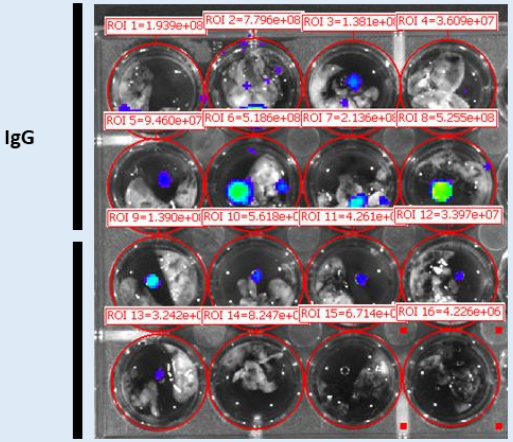


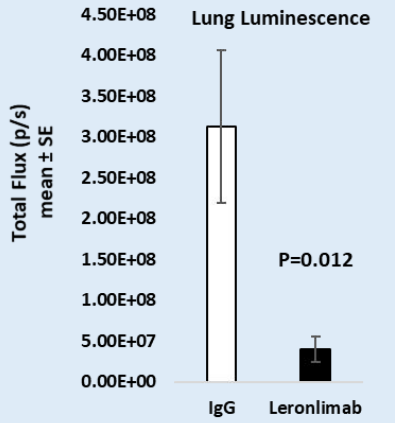


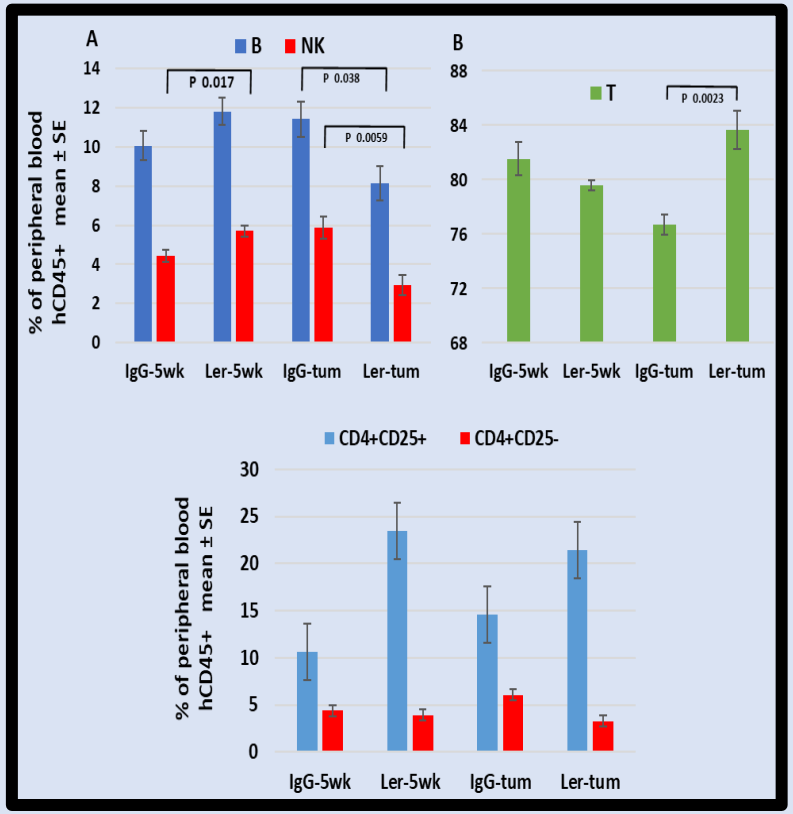


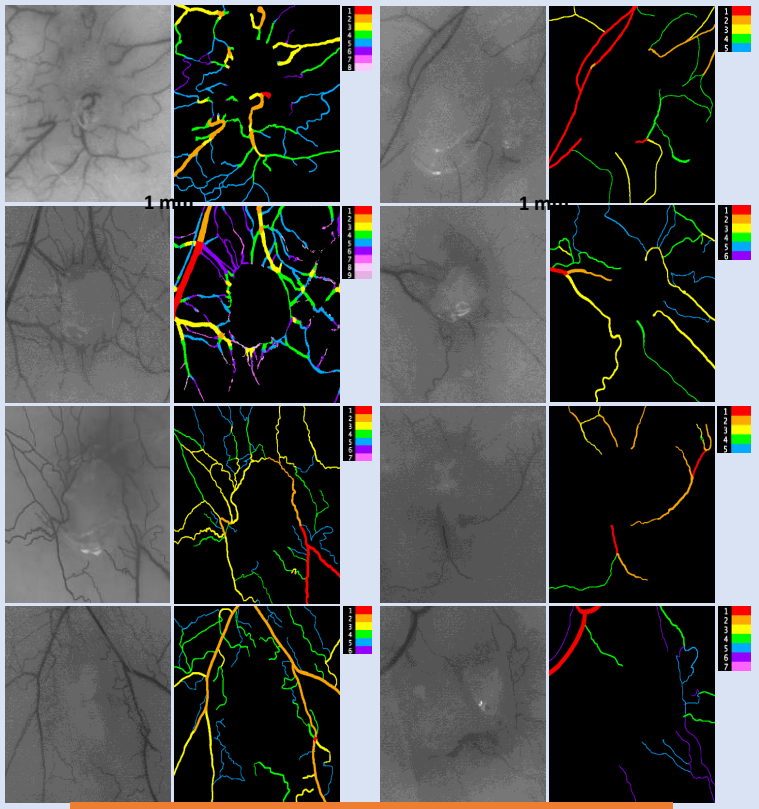












MOA in Cancer anti-tumor – Angiogenesis

Cancer - Mechanism of Action

CCR5's Role in Cancer Metastasis

Research indicates that the CCR5 receptor is the “GPS” system of a cancer cell that promotes metastatic disease. Preclinical studies have shown that Leronlimab blocks the calcium channel signaling of the CCR5 receptor and has the potential to disable the GPS system. CCR5 inhibition may disrupt signaling and ultimately the spread of CCR5+ Circulating Tumor Cells (CTC’s). Current therapies are directed to the primary tumor, rather than the movement or spread of cancer in the bloodstream. Metastatic disease, not the primary tumor, is the cause of death in the vast majority of cancer patients.

Leronlimab, a humanized monoclonal antibody directed to the CCR5 receptor, demonstrated anti-tumor activity in humanized NSG mouse models of both heterotopic and orthotopic SW480 human colon carcinoma. The anti-tumor effect was lost in non-humanized mice, suggesting a critical role for engrafted human leukocytes. Development of xGVHD was delayed in both tumor-bearing and non-tumor-bearing mice that had been treated with leronlimab. In peripheral blood of both tumor-bearing and non-tumor-bearing animals, leronlimab induced immunosuppressive human CD4+CD25+ cells (1.47 fold increase, p=0.016; 2.22-fold increase, p=0.0038, respectively). Leronlimab also caused a 1.84-fold reduction (p=0.033) of GVHD-promoting circulating human CD4+CD25- cells, and caused a 1.84-fold reduction (p=0.033) of GVHD-promoting circulating human CD4+CD25- cells. In the orthotopic model, lung metastatic burden was decreased 87% in leronlimab-treated mice compared to IgG-treated animals (p=0.012). Because of the lack of tumor-infiltrating leukocytes demonstrated by immunohistochemistry, we examined effects upon tumor-induced angiogenesis. Nascent (1 – 2 mm) SW480 tumors inoculated in the dermis were assayed for peri-tumoral vessel formation. The utilization of VESGEN software allowed detailed comparisons between treatment groups and revealed marked reduction in multiple key properties of the vascular network feeding the tumor, including 62% reduction in total vessel area (pixels) (p=0.013). Blockade of CCR5 signaling clearly interfered with host processes required for neo-vessel proliferation surrounding growing tumors.

BLA (HIV) - Combination Therapy with HAART

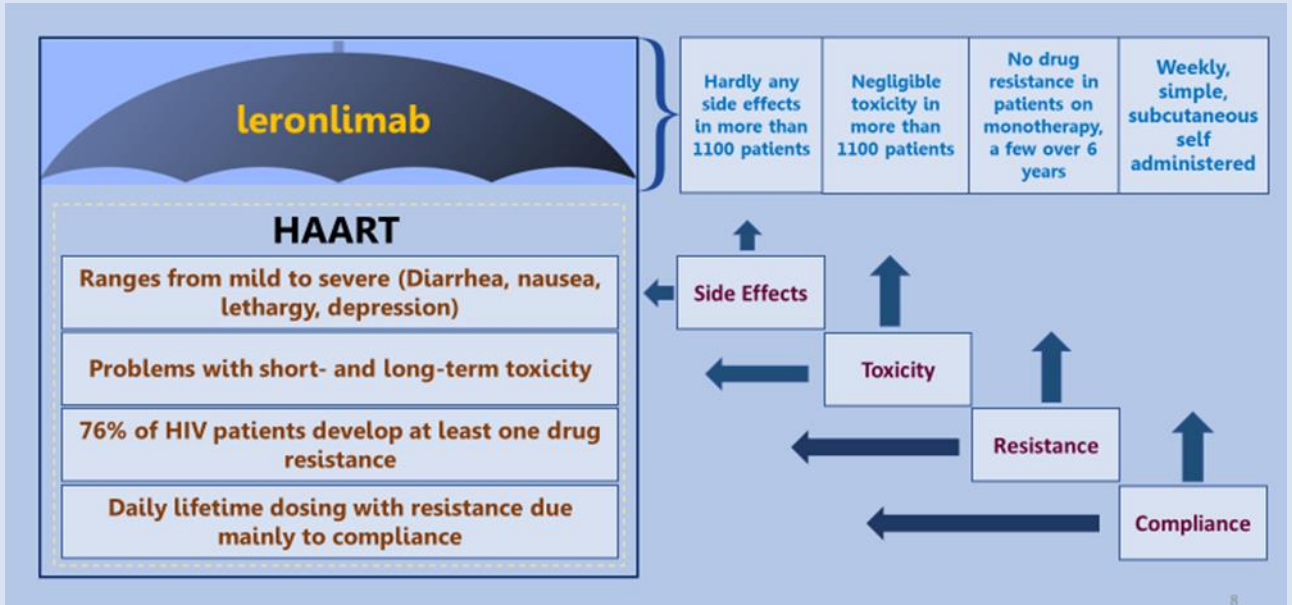
CD02-Phase 3 Primary Endpoint Achieved (*p*=0.0032)

1st potential approval for leronlimab

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**

- Leronlimab has received Fast-Track Designation for HIV and mTNBC
- 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 about 800 patients treated with leronlimab for HIV. Hardly any SAEs reported in patients treated with up to 700mg dose of leronlimab.
- BLA submission in 2021. Some patient in an extension arm for almost 4 years.



2nd potential approval

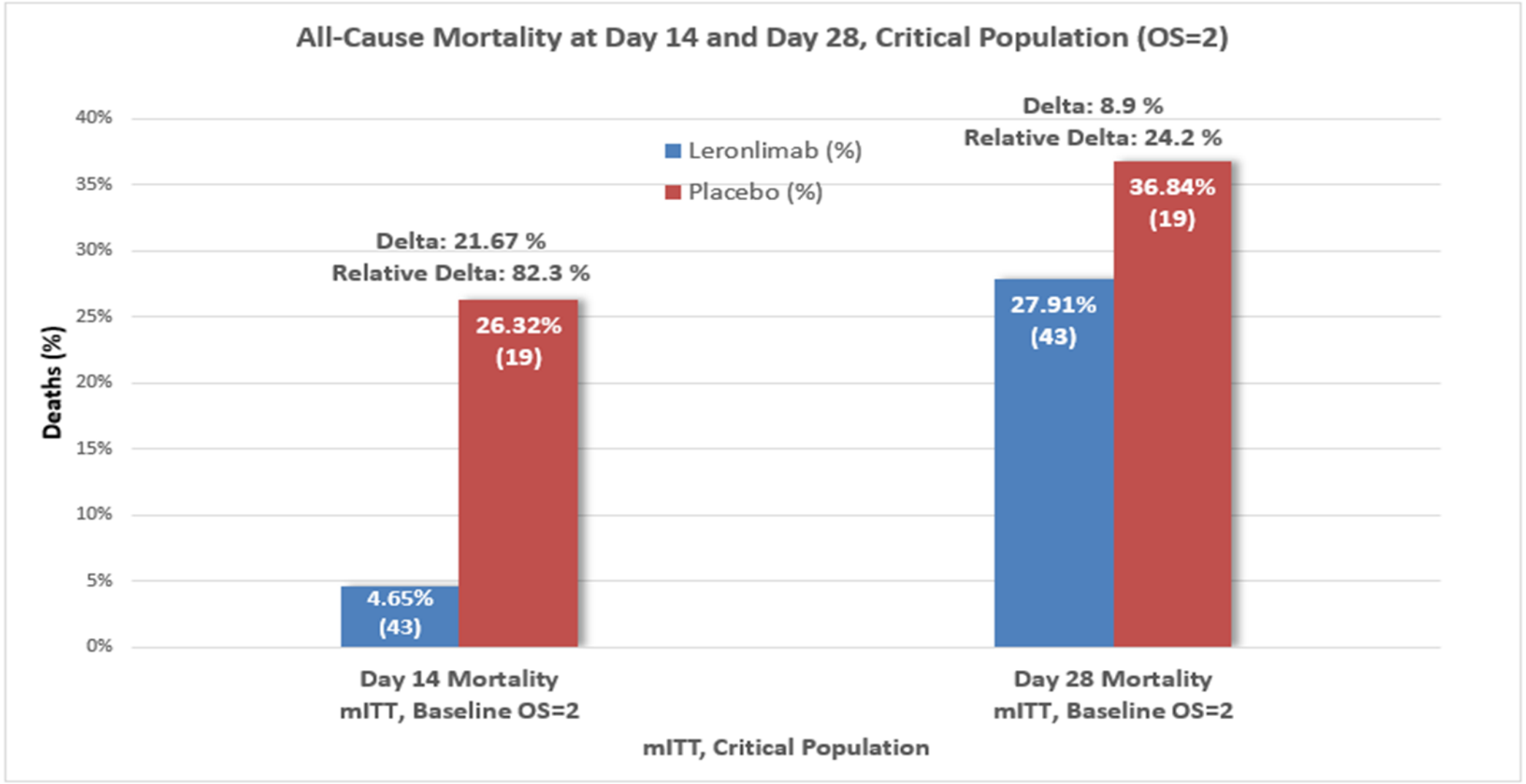
2nd potential approval as label expansion for Monotherapy in HIV

- 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 for label expansion
- **215 patients reached almost 1 year of suppressed viral load and over 40 patients average of 3 years, with 5 patients almost 7 years**

COVID-19
Five Trials and eIND patients

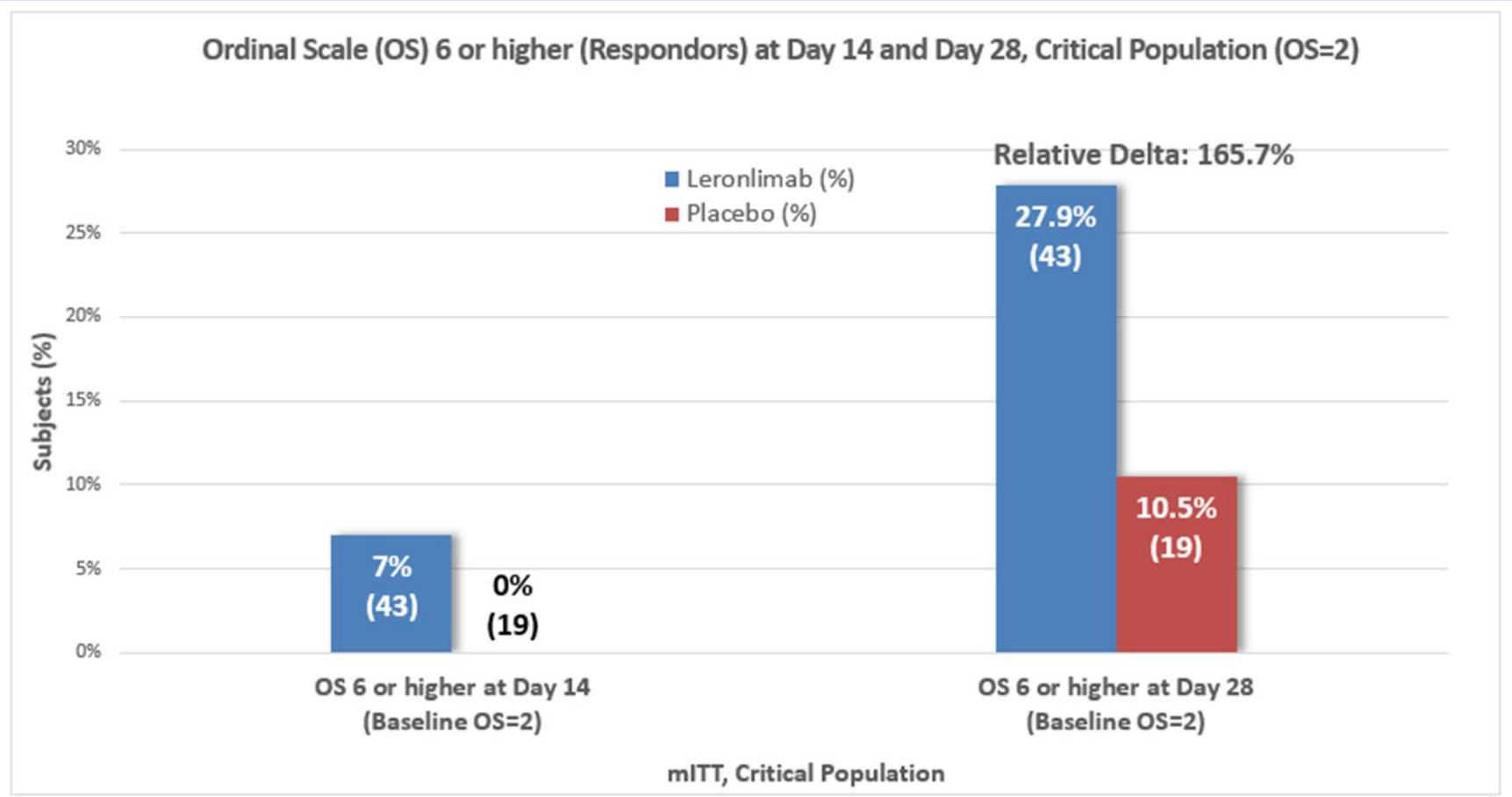
Program	Status
CD10 – Mild-to-moderate	Completed
CD12 – Severe-to-Critical	Completed
CD15 – Long-haulers	Completed
eIND	Over 70 approvals
CD16 – Critical ill (Brazil trial)	Pending 1st patient's enrollment
CD17 – Severe ill (Brazil trial)	Pending 1st patient's enrollment

CD12 (1st Phase 3 – COVID19 Severe-to-Critical Population)
All-Cause Mortality, Day 14 and Day 28



Note: Data label on the inside end of bar in the parenthesis reflect number of subjects in the population/treatment group (N).

CD12 (1st Phase 3 – COVID19 Severe-to-Critical Population)
Ordinal Scale 6 or Higher (Discharged Alive), Day 14 and Day 28

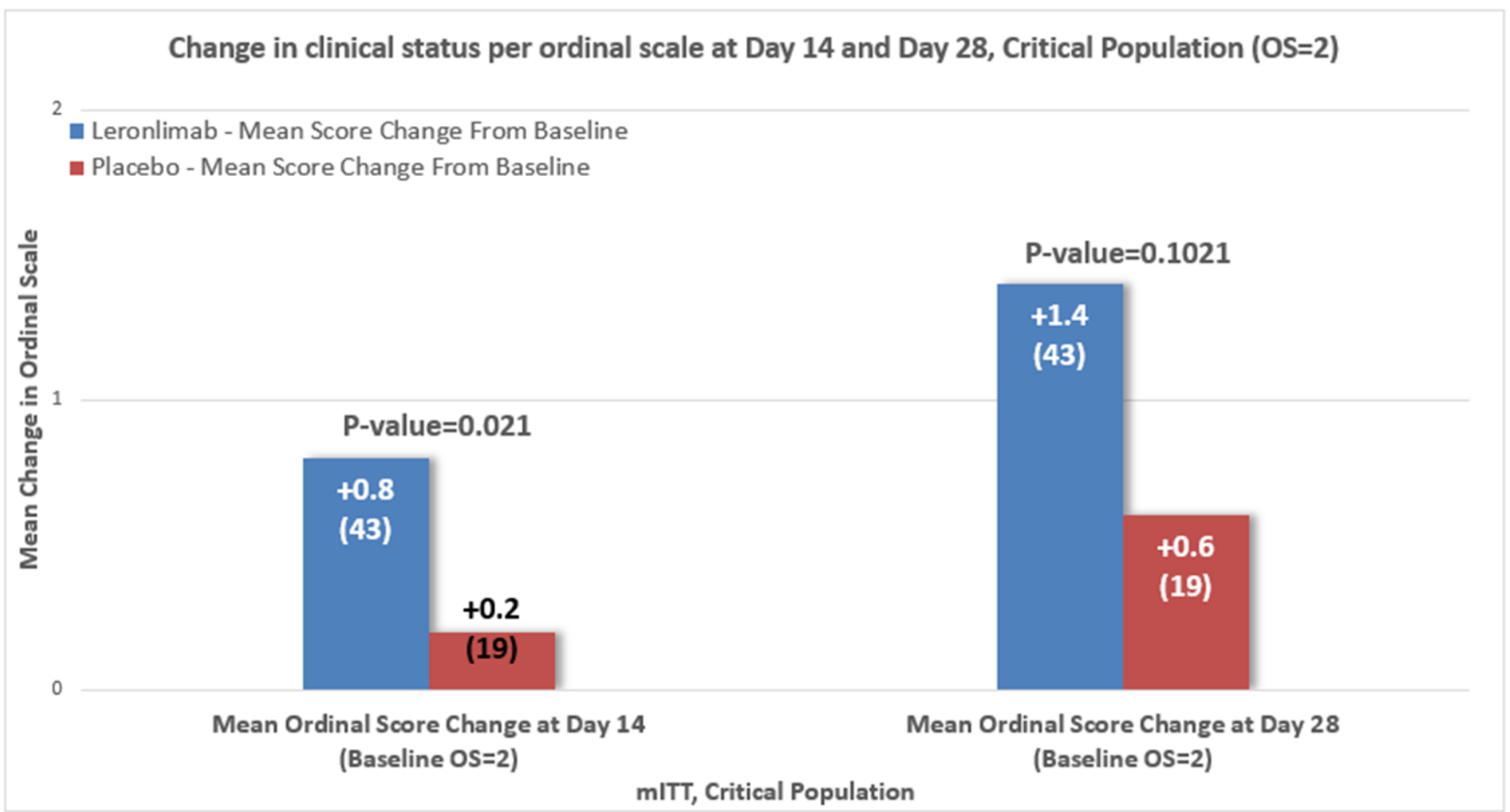


Note: Data label on the inside end of bar in the parenthesis reflect number of subjects in the population/treatment group (N).

7% (3/43) of the critically-ill population (i.e., on invasive mechanical ventilation/intubated at baseline) who received leronlimab + SoC were discharged from the hospital within 14 days compared to none (**0%,** 0/19) of the patients in the placebo + SoC group.

28% (12/43) of the critically-ill population (i.e., on invasive mechanical ventilation/intubated at baseline) who received leronlimab + SoC were discharged from the hospital within 28 days compared to only **11%** (2/19) patients in the placebo + SoC group (**p-value of 0.0824**).

CD12 (1st Phase 3 – COVID19 Severe-to-Critical Population)
Change in Clinical Status of Subject at Day 14 and Day 28 (on a 7-point O.S.)



Note: Data label on the inside end of bar in the parenthesis reflect number of subjects in the population/treatment group (N).

Note: For the critically ill population, the improvement in clinical status was **4 times higher at Day 14** and **more than 2 times higher at Day 28** in the leronlimab + SoC group compared to the placebo + SoC group.

CD12 (1st Phase 3 – COVID19 Severe-to-Critical Population) Results Overview

- **Shortened time to recovery**: The average length of hospital stay was lower in leronlimab group compared to placebo/SoC group in the critically ill population with a statistically significant **p-value of 0.0050** using the Rank-ANCOVA model.

	Days		P-value
	Leronlimab	Placebo	
Mean Length of hospital stay (days), mITT, Baseline OS=2	33.0	38.5	**0.005**

Note: p-value is from the rank-ANCOVA model adjusted for stratification factor and age

CD12 (1st Phase 3 – COVID19 Severe-to-Critical Population) Leronlimab Safety Profile

- The overall incidence rate, frequency, and severity of adverse events and serious adverse events (SAEs) were similar between the leronlimab and placebo group in the CD12_COVID-19 study.

	Leronlimab 700 mg		Placebo	
	N=259		N=125	
Parameter	**n(%)**	**Events**	**n(%)**	**Events**
Subjects with ≥ 1 AE	**142 (54.8)**	**555**	**77 (61.6)**	**337**
Subjects with ≥ 1 SAE	**99 (38.2)**	**197**	**47 (37.6)**	**98**
By Severity				
Subjects with ≥ 1 AE Leading to Death	59 (22.8)	59	31 (24.8)	31
Subjects with ≥ 1 Life-Threatening AE	17 (6.6)	83	8 (6.4)	41
Subjects with ≥ 1 Severe AE	24 (9.3)	158	11 (8.8)	93
By Causality				
Subjects with ≥ 1 Probably Related AE	1 (0.4)	1	0 (0.0)	0
Subjects with ≥ 1 Possibly Related AE	8 (3.1)	8	9 (7.2)	16
By Outcome				
Subjects with ≥ 1 AE Leading to Drug Withdrawal	1 (0.4)	1	1 (0.8)	1

Note: All percentages are based on the number of subjects in the safety population and treatment group (N).

Note: A subject is counted only once within each category, using the event with the worst-case intensity (by severity) or relationship (by causality).

Based on data snapshot as of 11-Feb-2021.

- The most commonly reported AEs across treatment groups, by preferred term (PT), were respiratory failure, anaemia, hypotension, and septic shock. Remaining PTs were reported at incidences <5%.

CD12 (1st Phase 3 – COVID19 Severe-to-Critical Population) Leronlimab Safety Profile

Parameter	PRO 140 (700 mg) N=56		Placebo N=28		Total N=84	
	n (%)	Events	n (%)	Events	n (%)	Events
Subjects with ≥ 1 AE	**19 (33.9)**	**43**	**14 (50.0)**	**53**	**33 (39.3)**	**96**
Subjects with ≥ 1 SAE	**5 (8.9)**	**8**	**6 (21.4)**	**11**	**11 (13.1)**	**19**
By Severity						
Subjects with ≥ 1 Severe AE [1]	5 (8.9)	6	3 (10.7)	3	8 (9.5)	9
By Causality						
Subjects with ≥ 1 Probably Related AE	1 (1.8)	1	0 (0.0)	0	1 (1.2)	1
Subjects with ≥ 1 Possibly Related AE	2 (3.6)	4	3 (10.7)	6	5 (6.0)	10

[1] Severe AEs are those adverse events that were considered severe or life-threatening or causing death.

Note1: All percentages are based on the number of subjects in the safety population and treatment group (N).

Note2: A subjects is counted only once within each category.

Based on data snapshot as of 21-Jul-2020.

- The most commonly reported AEs across treatment groups, by preferred term (PT), were dyspnoea, urinary tract infection, C-reactive protein increased, and muscular weakness. Remaining PTs were reported at incidences <4%.

CD12 (1st Phase 3 – COVID19 Severe-to-Critical Population)
Leronlimab Safety Profile

	Leronlimab 700 mg N=315		Placebo N=153		Total N=468	
Parameter	**n(%)**	**Events**	**n(%)**	**Events**	**n(%)**	**Events**
Subjects with ≥ 1 AE	169 (53.7)	635	92 (60.1)	403	261 (55.8)	1038
Subjects with ≥ 1 SAE	104 (33.0)	205	54 (35.3)	110	158 (33.8)	315

Note1: All percentages are based on the number of subjects in the safety population and treatment group (N).
Note2: A subject is counted only once within each category.

- The most commonly reported AEs across treatment groups, by preferred term (PT), were respiratory failure, acute kidney injury, anaemia, and hypotension. Remaining PTs were reported at incidences <5%.

Leronlimab Safety History

- Leronlimab has an extensive safety profile available based on data from more than 1000 patients treated across multiple studies and indications. Leronlimab was generally well tolerated with no major safety concerns. Participants have received weekly subcutaneous doses of leronlimab with the longest duration of exposure lasting 5+ years in HIV setting.

Protocol Number	Indication	Phase	Number of Subjects Received Leronlimab
PRO 140 1101	Healthy	1	16
PRO 140 1102	Healthy	1	20
PRO 140 1103	Healthy	1	14
PRO 140 _CD06	Healthy	1	79
PRO 140 1302	HIV	1b	30
PRO 140 2301	HIV	2a	20
PRO 140 2101	HIV	2a	34
PRO 140 _CD01 /CD01 Extn	HIV (as monotherapy)	2b	43
PRO 140 _CD02 /CD02 Extn	HIV (in combination with cART)	2b/3	52
PRO 140 _CD03 /CD03 Extn	HIV (as monotherapy)	2b/3	562
CD10_COVID-19	Mild-to-Moderate COVID-19	2	56
CD12_COVID-19	Severe-to-Critical COVID-19	2b/3	259
		Total	**1185**

COVID-19
Long-Haulers

Program	Status
CD15 – Long-Haulers	Completed 17 symptoms out of 24 improved Manuscript being prepared for submission to a publication
CD18 – Long-Haulers	In discussion with the FDA

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

Summary

- **Goal of Interim Analysis is to compare PFS and OS in relation to Standard of Care (SOC) or Sacituzumab Govitecan (SG) Trial**
  - Goal one: Have a mPFS >2.3 (SOC) or >5.6 (SG)
  - Goal two: Have a mOS >6.7 (SOC) or >12.1 (SG)
- **CCR5 in Tissue Staining**
  - Low CCR5 in tissue partially predicted treatment response
  - Low CCR5 trended for better PFS, BUT had no effect on OS
  - mPFS for patients with <50% CCR5 in tissue was ~5.6
  - No difference was seen in mOS between high and low CCR5 expression
- **CAMLs and CTCs are Baseline (Prior to Induction)**
  - Prior to First dose of Leronlimab CTCs/CAMLs DID NOT predict for response
- **Decrease in CAMLs/CTCs after 1 cycle of Leronlimab (~30 days)**
  - 72% of patients had a decrease in CAMLs after 30 days
  - This decrease was associated with a significant ~300% increase in mPFS
  - Also associated with a significant 450% increase in overall (& median) survival at 12 months

Overall Summary

- High CCR5 may stratify patients likely to progress on Leronlimab
- Decreases in CAMLs/CTCs appear to be related to slower progression and lower mortality
- Leronlimab appears to have efficacy superior to standard of Care in specific populations
- CAMLs appeared to identify populations that are responding to Leronlimab
- Decreases in CAMLs after Leronlimab induction were seen in ~72% of patients which were associated with a significant 450% increase in overall survival at 12 months

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)	Interim Analysis of first 60 patients coming up

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

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.

Two patients 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

Leronlimab Manufacturing

Samsung BioLogics (Commercial Partnership)

SAMSUNG BIOLOGICS

- Deal signed April 2019
- Build-out in process
- Manufacturing underway with multiple batches completed
- 1.2 million vial already manufactured

Robust Pipeline

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

Indication	Program for 2021
HIV	BLA submission-USA, UK, EU, Canada and Brazil
HIV	Protocol submission for label expansion - Monotherapy (Phase 3)
HIV	Protocol submission - PrEP (Phase 2)
HIV	First patient dose - Cure (Phase 2)
COVID-19	Interim analysis from Severe population in Brazil
COVID-19	Interim analysis from Critical population in Brazil
COVID-19	Long Hauler protocol – Enrollment completion
Cancer	Cancer - mTNBC (Phase 1b/2) – Breakthrough designation application to file
Cancer	Cancer - Basket trial (Phase 2) – Breakthrough designation application to file
NASH	NASH (Phase 2)
MS-Alzheimer-Stroke	Phase 2 – Protocol submission
HIV	Manuscript submission to publication – CD02 (combination therapy)
COVID-19	Manuscript submission to publication - CD10 (mild-to-moderate trial)
COVID-19	Manuscript submission to publication - CD12 (severe-to-critical trial)
COVID-19	Manuscript submission to publication - CD15 (long-haulers trial)