

PRESS RELEASE

Collectis Reports Encouraging Clinical Data from BALLI-01 Study in Relapsed/Refractory B-cell Acute Lymphoblastic Leukemia, and Preclinical Data from TALGlobin01 at the 63rd American Society of Hematology Annual Meeting

- Collectis' Proprietary Product Candidate UCART22 was Administered after Lymphodepletion with Fludarabine, Cyclophosphamide and Alemtuzumab (FCA) with no Dose-Limiting Toxicities in BALLI-01 Phase 1 Study
 - The addition of Alemtuzumab to FC (FCA) Resulted in Prolonged Host-Lymphocyte Suppression, and UCART22 Expansion
 - Preliminary Data Demonstrating Encouraging Anti-Leukemic Activity was Observed in 2 Patients
- BALLI-01 is Currently Enrolling at Dose Level Three with FCA (fludarabine, cyclophosphamide, alemtuzumab) Lymphodepletion Regimen
- Pre-Clinical Data from Collectis' Proprietary Product Candidate Talglobin01 Demonstrates that TALEN[®] Could Be Specific and Efficient at Correcting the Mutated Beta-Globin Gene that Causes Sickle Cell Disease

December 11, 2021 – New York (N.Y.) – Collectis (Euronext Growth: ALCLS - Nasdaq: CLLS), a clinical-stage biotechnology company employing its pioneering TALEN[®] gene-editing platform to develop innovative therapeutics for the treatment of serious diseases, announced today preliminary results from the BALLI-01 Phase 1 study of UCART22, its allogeneic CAR-T cell therapy candidate targeting CD22, in patients with relapsed or refractory B-cell acute lymphoblastic leukemia (r/r B-ALL), and preclinical data on TALGlobin01, its autologous cell therapy product candidate for homozygous SCD patients (HbSS) at the 63rd Annual Meeting of the American Society of Hematology (ASH) in Atlanta, Georgia.

“We are excited by the encouraging preliminary results obtained from patients administered UCART22 after fludarabine, cyclophosphamide and alemtuzumab (FCA) lymphodepletion in the BALLI-01 study. The addition of alemtuzumab to fludarabine and cyclophosphamide (FC) was demonstrated to be safe, improve host-lymphocyte suppression, and promote UCART22 expansion, which was associated with anti-leukemic activity,” said Carrie Brownstein, MD, Chief Medical Officer. “We believe these initial data support our mission to develop UCART22 for patients with r/r B-ALL, who remain in dire need of additional treatment options, particularly those who have failed CD19 therapy.”

BALLI-01 investigating UCART22 product candidate in R/R B-ALL

BALLI-01, a phase 1 open-label dose-escalation study, is designed to assess the safety, maximum tolerated dose (MTD), and preliminary anti-leukemia activity of UCART22 in patients with r/r B-ALL. Additional endpoints include characterization of the expansion, trafficking, and persistence of UCART22 cells.

The poster presentation includes preliminary data from patients who received UCART22 at dose level 2 (DL2) and intermediate dose level 2 (DL2i) after lymphodepletion with FCA. Alemtuzumab

was added to FC to deepen and sustain host lymphocyte suppression and thereby promote UCART22 expansion and persistence.

As of the clinical cut-off date of October 1, 2021, 12 patients received lymphodepletion; 11 were administered UCART22, of which 6 received UCART22 and FCA. Enrolled patients were predominantly male [n=7], young (median age 30 [range 20-61]), and most had recurrent genetic abnormalities including the CRFL2 (cytokine receptor-like factor 2) rearrangement. Additionally, enrolled patients were heavily pretreated with a median of 3 prior lines of therapy [range 2-6]. Three-fourths of patients had received prior blinatumomab, approximately half had received prior inotuzumab, and 3 had received prior CD19 autologous CAR-T therapy.

Safety Data

The FCA lymphodepletion regimen was well tolerated, and most treatment-emergent adverse events (TEAEs) were mild to moderate in intensity and manageable. Importantly, no patients experienced protocol-defined dose limiting toxicities (DLTs), immune effector cell-associated neurotoxicity syndrome (ICANS), nor UCART22-related severe (grade ≥ 3) TEAEs. Three patients experienced mild to moderate cytokine release syndrome (CRS), and one patient reported grade II GvHD with skin involvement only, that required hospitalization.

Activity Data

Encouraging anti-leukemic activity was observed in two (2/6) patients in the FCA cohorts. Both patients, one at DL2 and one at DL2i, achieved blast reductions to $< 5\%$ (0.4% and 0%, respectively) by day 28, accompanied by measurable UCART22 expansion and changes in relevant inflammatory cytokines.

Overall, UCART22 after FCA lymphodepletion regimen demonstrated promising signs of anti-leukemic activity at DL2 and DL2i, without unexpected or significant treatment-related toxicity. The addition of alemtuzumab to the FC lymphodepletion regimen was safe and promoted sustained host T-cell suppression and expansion of UCART22. These data are encouraging and support the further development of UCART22 for patients with r/r B- ALL. BALLI-01 is currently enrolling patients at dose level 3 with FCA lymphodepletion.

TALGlobin01; an autologous *ex vivo* TALEN®-edited hematopoietic stem and progenitor cell gene therapy for the treatment of Sickle Cell Disease

Initial pre-clinical data from Collectis' .HEAL platform's product candidate, TALGlobin01 demonstrates that TALEN® is specific and efficient in correcting the mutated beta-globin gene, the underlying cause of sickle cell disease.

The data, presented in a poster, demonstrate that TALEN®-based engineering could be used to correct the beta-globin gene mutation in HbSS patient-derived hematopoietic stem and progenitor cells. The data show up to 70% of *HBB* allelic correction, with only 9% of *HBB* biallelic inactivation and a low level of TALEN® off-target cleavage. Genetic correction of *HBB* translates into high level of hemoglobin A expression (up to 47% HbA detected among total hemoglobin) and reversion of the sickling phenotype in differentiated red blood cells. Preclinical data show the capacity of TALGlobin01 edited cells to engraft *in vivo* using an NSG mouse model.

Collectively, the preclinical data demonstrate high efficiency and safety of TALEN® treatment in HbSS patient-derived hematopoietic stem and progenitor cells.

A copy of each poster presentation is available on Collectis' website, linked [here](#).

About UCART22

UCART22 is an allogeneic T-cell product manufactured from healthy donor cells. T-cells are transduced using a lentiviral vector to express the anti-CD22 chimeric antigen receptor (CAR) and are genetically modified using TALEN® gene-editing technology to disrupt the T-cell receptor alpha constant (*TRAC*) gene to minimize risk of graft-vs-host disease (GvHD) and the *CD52* gene to eliminate sensitivity to anti-CD52–directed agents used in lymphodepletion regimens. UCART22 is being developed for the treatment of R/R B-cell ALL.

About TALGlobin01

TALGlobin01, is an autologous *ex vivo* TALEN®-edited CD34+ HSC gene therapy for the treatment of SCD. TALGlobin01 is developed using both TALEN® technology to induce a double strand DNA break in the SCD-causing hemoglobin subunit beta (*HBB*) gene and adeno-associated virus (AAV) particles containing a DNA repair template designed to correct the faulty *HBB* gene via endogenous homology directed repair.

About Collectis

Collectis is a gene editing company, developing first of its kind therapeutic products. Collectis utilizes an allogeneic approach for CAR-T immunotherapies in oncology, pioneering the concept of off-the-shelf and ready-to-use gene-edited CAR T-cells to treat cancer patients, and a platform to make therapeutic gene editing in hemopoietic stem cells for various diseases. As a clinical-stage biopharmaceutical company with over 21 years of expertise in gene editing, Collectis is developing life-changing product candidates utilizing TALEN®, its gene editing technology, and PulseAgile, its pioneering electroporation system to harness the power of the immune system in order to treat diseases with unmet medical needs.

As part of its commitment to a cure, Collectis remains dedicated to its goal of providing lifesaving UCART product candidates for multiple cancers including acute myeloid leukemia (AML), B-cell acute lymphoblastic leukemia (B-ALL) and multiple myeloma (MM). .HEAL is a platform focusing on hemopoietic stem cells to treat blood disorders, immunodeficiencies and lysosomal storage diseases.

Collectis headquarters are in Paris, France, with locations in New York, New York and Raleigh, North Carolina. Collectis is listed on the Nasdaq Global Market (ticker: CLLS) and on Euronext Growth (ticker: ALCLS).

For more information, visit www.collectis.com

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Forward-looking Statements

This press release contains “forward-looking” statements within the meaning of applicable securities laws, including the Private Securities Litigation Reform Act of 1995. Forward-looking statements may be identified by words such as “anticipate,” “believe,” “expect,” “plan,” “scheduled,” “could” and “will,” or the negative of these and similar expressions. These forward-looking statements, which are based on our management’s current expectations and assumptions and on information currently available to management, include statements about our research and development projects and priorities, our pre-clinical project development efforts and the timing of our presentation of data. These forward-looking statements are made in light of information currently available to us and are subject to numerous risks and uncertainties, including with respect to the numerous risks associated with biopharmaceutical product candidate development as well as the duration and severity of the COVID-19 pandemic and governmental and regulatory measures implemented in response to the evolving situation. With respect to our cash runway, our operating plans, including product development plans, may change as a result of various factors, including factors currently unknown to us. Furthermore, many other important factors, including those described in our Annual Report on Form 20-F and the financial report (including the management report) for the year ended December 31, 2020 and subsequent filings Collectis makes with the Securities Exchange Commission from time to time, as well as other known and unknown risks and uncertainties may adversely affect such forward-looking statements and cause our actual results, performance or achievements to be materially different from those expressed or implied by the forward-looking statements. Except as required by law, we assume no obligation to update these forward-looking statements publicly, or to update the reasons why actual results could differ materially from those anticipated in the forward-looking statements, even if new information becomes available in the future.

