Press Release

COTI-2 MOVES INTO HUMAN TESTING

Critical Outcome Technologies Signs Phase 1 Clinical Trial Agreement

London, Ontario (December 10, 2015): Critical Outcome Technologies Inc. ("COTI" or the "Company") (TSX Venture: COT; OTCQB: COTQF), announced today the signing of a clinical trial agreement ("CTA") to conduct a Phase 1 clinical trial to evaluate COTI-2, the Company's activator of mutant p53 protein, in patients with advanced gynecologic cancers at The University of Texas MD Anderson Cancer Center ("MD Anderson").

"This is a significant milestone for our Company, our lead oncology COTI-2 program, and potentially for the future of patients afflicted with gynecologic and other cancers," said Dr. Wayne Danter, President and CEO. "The CTA provides many benefits to our Company, and to the advancement of COTI-2, not the least of which is the caliber of the team that will help to guide this first-in-humans trial. MD Anderson is world-renowned for its state-of-the-art facilities, technical capabilities, and the expertise to successfully execute our Phase 1 clinical trial." Principal Investigator for the COTI-2 Phase 1 clinical trial, Shannon Westin, MD, MPH, stated, "An agent, such as COTI-2, that successfully leverages the most common molecular abnormality in solid tumors, a p53 mutation, has the potential to advance the current state of gynecologic cancer care."

With the CTA in place, the Company can now commence the clinical trial with COTI-2 that is planned to include up to 46 women with advanced gynecologic cancers who have failed conventional therapy. The trial will focus on determining the maximum tolerated-dose, pharmacokinetics, and safety of COTI-2. Patient enrollment is anticipated to begin in mid-December following a site visit to MD Anderson with treatment to commence shortly thereafter. The study includes an expansion arm of ten women specifically with ovarian cancer whose test results will be examined for early signals of treatment efficacy once the maximum tolerated dose is determined.

For the purposes of the Phase 1 clinical trial, the term "gynecologic" cancer refers to cancers of the ovary, endometrium, and cervix. Dr. Westin, an Assistant Professor in the Department of Gynecologic Oncology and Reproductive Medicine at MD Anderson will work in association with the lab of Gordon B. Mills, MD, Chairman, Department of Systems Biology, Wiess Distinguished University Chair in Cancer Medicine, Co-Director, Institute for Personalized Cancer Therapy at MD Anderson in conducting the trial.

About COTI-2

COTI-2 is a small molecule activator of misfolded mutant p53 protein approved for clinical development. Extensive studies have demonstrated COTI-2's ability to restore mutant p53 function and thus induce cancer cell death in many common p53 mutations. Mutations of the p53 gene are the most common genetic alterations in human cancers, occurring in a wide range of cancers, including ovarian, lung, colorectal, breast, liver, bladder and other cancers. COTI-2's specific protein target, low toxicity, combination effectiveness with standard agents, and potential for longer term outpatient therapy as an oral agent, supports a dramatic change in the treatment of susceptible cancers.

About Critical Outcome Technologies Inc.

COTI is a biopharmaceutical company advancing the treatment of cancer through targeted therapeutics. The Company's lead compound, COTI-2, has a novel p53-dependent mechanism of action with selective and potent anti-cancer activity. The initial indication is in gynecologic cancers (ovarian, cervical and endometrial) that will begin patient dosing in a Phase 1 clinical trial at MD Anderson Cancer Center in late 2015/early 2016. The Company has secured orphan drug status for the ovarian indication in the U.S. and is planning additional studies in other indications such as head & neck, AML and Li-Fraumeni as well as combination therapies with other leading cancer drugs. Pre-clinical data provides evidence to suggest a potentially dramatic change in the treatment of cancers with mutations of the p53 gene.

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