ChemoCentryx Initiates Phase II Clinical Trial for CCX168, a Novel Small Molecule C5aR Antagonist for the Treatment of Vasculitis

Mountain View, CA., October 17, 2011 – ChemoCentryx, Inc., today announced the initiation of a Phase II clinical trial for CCX168, an orally-administered small molecule for the treatment of anti-neutrophil cytoplasmic antibody (ANCA)-associated vasculitis (AAV), a disease which can lead to renal and pulmonary failure. CCX168 specifically targets the C5a receptor (C5aR), a potent pro-inflammatory mediator that is involved in AAV as well as several other autoimmune diseases. This randomized, double-blind, placebo-controlled trial will evaluate the safety and tolerability of CCX168 in patients with ANCA-associated renal vasculitis while assessing the potential for reducing or eliminating the use of corticosteroids in these patients.

"Having advanced six orally active drugs into the clinic, we believe that we have successfully demonstrated the capabilities of our drug discovery platform and have shown the promise of blocking specific chemokines and chemoattractant cytokines in the treatment of autoimmune and inflammatory diseases," stated Thomas J. Schall, Ph.D., President and CEO, ChemoCentryx. "Today's announcement is another example of how we are harnessing our drug discovery and development capabilities to address inflammatory and autoimmune diseases that currently have inadequate treatment options."

About C5aR and CCX168

The complement system consists of a set of proteins that regulate certain types of inflammatory responses. Fragments of complement proteins, such as the chemoattractant complement fragment known as C5a, work to recruit immune system cells, including neutrophils, to sites of inflammation by means of its receptor (C5aR). This system is active in many diseases, thus making C5aR an attractive target for human therapeutics. Given molecular structure similarities between the C5aR and chemokine receptors, ChemoCentryx researchers successfully applied the Company's proprietary drug discovery technologies to the design of small molecule C5aR antagonists and selected CCX168 based on its potency, selectivity and favorable pharmacokinetics. CCX168 is one of four drug candidates discovered and developed by ChemoCentryx under the Company's alliance with GlaxoSmithKline (GSK) to identify molecules targeting four specific chemokine and chemoattractant receptors.

About ANCA-Associated Vasculitis

Granulomatous polyangiitis and microscopic polyangiitis vasculitides are associated with anti-neutrophil cytoplasmic antibodies (ANCA). These auto-antibodies lead to the activation and increased adhesiveness of neutrophils to endothelial cells that line the blood vessels. These accumulating adhering neutrophils initiate an inflammatory cascade in the small blood vessels by secreting pro-inflammatory cytokines and chemoattractants.

If left untreated, AAV may lead to renal and pulmonary failure and is often fatal. AAV is currently treated with pulses of cyclophosphamide and high-dose systemic corticosteroids. Little advance in the treatment of these diseases has been made since the 1970's when these treatments were first introduced. Cyclophosphamide is a toxic alkylating agent. Systemic corticosteroid use is associated with increased

risk of infection, osteoporosis, etc. Rituximab, azathioprine, methotrexate, or mycophenolate mofetil therapy are also used, but these drugs are associated with an increased risk of infection, bone marrow suppression, certain types of cancer, serious and sometimes fatal infusion reactions and progressive multifocal leukoencephalopathy (rituximab). Therefore, there is clearly still a substantial unmet medical need in treatment of patients with AAV.

About ChemoCentryx

ChemoCentryx, Inc. is a clinical-stage biopharmaceutical company focused on discovering, developing and commercializing orally-administered therapeutics that target the chemokine and chemoattractant systems in order to treat autoimmune diseases, inflammatory disorders and cancer. The chemokine system is a biological network that regulates inflammation via a collection of secreted chemokine molecules, or ligands, and their specific cell surface receptors. Based on its proprietary drug discovery and drug development platform, ChemoCentryx has generated multiple clinical and preclinical-stage programs, each targeting distinct chemokine and chemoattractant receptors with different small molecule compounds. ChemoCentryx's lead compound, CCX282-B (Traficet-EN, now designated GSK1605786, also known as GSK'786), a specific CCR9 antagonist, completed a multi-national clinical trial, called PROTECT-1, in patients with moderate-to-severe Crohn's disease, where it demonstrated the ability to induce a clinical response and to maintain clinical remission, and is now in Phase III clinical development. ChemoCentryx's lead independent drug candidate, CCX140-B, a CCR2 antagonist, has been shown to be safe and well tolerated while demonstrating clinical activity on glycemic indices in a Phase II clinical trial in type 2 diabetes. CCX140-B is expected to enter Phase II clinical development for the treatment of diabetic nephropathy in 2011. Other clinical programs include CCX354, a CCR1 antagonist which recently completed a Phase II clinical trial for the treatment of rheumatoid arthritis; CCX168, a C5aR antagonist, in Phase II clinical development for the treatment of vasculitis; and CCX832, a ChemR23 antagonist in Phase I clinical development. ChemoCentryx also has several programs in advanced preclinical development.

Certain statements in this press release may constitute "forward-looking statements". These statements are made on the basis of current expectations, forecasts and assumptions that involve risks and uncertainties, including, but not limited to, economic, competitive, governmental and technological factors outside of our control, that may cause our business, strategy or actual results to differ materially from those expressed or implied. We do not intend, and undertake no obligation, to update any forward-looking statements, whether as a result of new information, future events or otherwise.