Press Release

Cerenis Therapeutics featured prominently at the INTERNATIONAL SYMPOSIUM ON ATHEROSCLEROSIS (IAS)

Experimental results demonstrate dose-dependent inhibition of atherosclerotic plaque formation for CER-001 as a novel engineered HDL-mimetic and active role to treat atherosclerosis and non-alcoholic steatohepatitis (NASH) for CER-209 as an agonist of the P2Y13 receptor

Toulouse, FRANCE, Ann Arbor, UNITED STATES, May 26, 2015 – **Cerenis Therapeutics** (**FR0012616852- CEREN**), an international biopharmaceutical company dedicated to the discovery and development of innovative HDL therapies ("good cholesterol") for treating cardiovascular and metabolic diseases, today reported on an oral and poster presentations featuring Cerenis Therapeutics' innovative HDL therapies, CER-001 and CER-209, during the 17th INTERNATIONAL SYMPOSIUM ON ATHEROSCLEROSIS (IAS) held in Amsterdam, May 23-26, 2015.

• CER-001 mimics native HDL as it demonstrates a dose dependent inhibition of atherosclerotic plaque formation modulated by ABCA1 expression

In a first poster presentation ("HDL and CER-001 Inverse-dose dependent inhibition of atherosclerotic plaque formation in apoE-/- mice: Evidence of ABCA1 down-regulation"), Cerenis presented results on natural HDL and CER-001 dose dependent inhibition of atherosclerotic plaque formation in a validated preclinical model modulated by ABCA1 expression.

These results confirm CER-001's efficacy at slowing the progression of atherosclerosis and demonstrate that high doses of HDL and CER-001 are less effective at slowing the progression of atherosclerotic plaque in apoE-/- mice compared to lower doses, following a U-shaped dose-response curve. A potential mechanism for this phenomenon is supported by the observation that high doses of HDL and CER-001 induce a rapid and strong down-regulation of ABCA1 transporter both in vitro and in vivo. Maximally efficient HDL- or CER-001-mediated cholesterol removal from atherosclerotic plaque is achieved by maximizing macrophage-mediated efflux from the plaque while minimizing dose-dependent down-regulation of ABCA1 expression. These observations help support the optimal dose of HDL mimetics for testing in clinical trials of atherosclerotic plaque regression and launching the future Phase II clinical study CARAT of 3mg/kg CER-001 in post-Acute Coronary Syndrome (ACS) patients.

• CER- 209, an agonist of the P2Y13 receptor, is well suited to the treatment of atherosclerosis and non-alcoholic steatohepatitis (NASH)

In a second poster presentation ("P2Y13 receptor agonist CER-209 decreases both atherosclerosis and liver steatosis in vivo"), Cerenis presented results of CER-209, a selective novel agonist of the P2Y13R that caused stimulation of bile acid secretion associated with an increased uptake of HDL-c in the liver. Repeated dose administration stimulated the apoA-I synthesis and formation of small HDL particles, known to be atheroprotective. Agonist-treated plasma samples had high cholesterol efflux capacity for the mobilization of cholesterol in vitro compared to the placebo group. CER-209 induced a decrease in atherosclerotic plaques in aorta and carotids as well as a remarkable decrease in the steatosis in a validated preclinical model.

High-density lipoprotein (HDL) is known to protect against atherosclerosis by promoting the reverse cholesterol transport. A new pathway for the regulation of HDL-cholesterol (HDL-c) removal involving F1-ATPase and P2Y13 receptor (P2Y13R) has been described in vitro, and recently in mice. An increase in the expression of liver mRNA and plasma apoA-I concentration of treated animals was observed. The uptake of large HDL particles by the liver also stimulates de novo synthesis of nascent HDL particles, thereby enhancing the cholesterol efflux capacity of the serum. The overall implication of this increase is not only to allow the removal of cholesterol from atherosclerotic plaques, but

also to regulate lipid homeostasis in the liver.

Dr. Jean-Louis Dasseux, Founder and CEO of Cerenis comments*: "These results are key to properly understanding the mechanism of action of CER-001 for it to be effective at slowing the progression of atherosclerotic plaque. We could define the optimal dose of HDL mimetics for testing in clinical trials of atherosclerotic plaque regression. In addition, the second poster presentation reinforces the fact that CER- 209 has the potential to be another effective treatment for both atherosclerosis and non-alcoholic steatohepatitis (NASH). All these mechanistic understandings will support the preparation of the phase II study (CARAT) for the post-ACS indication and the phase III study for the treatment of patients affected by Familial Hypoalphalipoproteinemia (FPHA) orphan disease. The next step for us is to initiate the first patient enrolments for both studies by the end of 2015."*

Dr. Richard Pasternak, Chairman of the Board of Cerenis comments: *"The results demonstrate the ability of the Cerenis scientists to critically advance our understanding of HDL metabolism pointing to new clarity regarding therapeutic dosing of natural HDL, and to a new mechanism for HDL regulation, which could lead to an entirely new class of therapeutics. These presentations, coming soon after the Company's very successful IPO, further confirm the excellence of our scientific and management teams."*

Notes to editors

Atherosclerosis is a disease arising from formation of plaque, so-called atherosclerotic plaque, caused by deposits of lipids especially cholesterol in the vessel wall, which leads to the manifestation of cardiovascular diseases including angina pectoris and myocardial infarction ("heart attack") all designated by the term acute coronary syndrome (ACS). Atherosclerosis affects the entire vascular system and also leads to several other complications, including ischemic stroke, renal failure and arteriopathy of the lower limbs.

The major carriers for cholesterol in the blood are lipoproteins, including the low-density lipoprotein (or LDL) particles, and the high-density lipoprotein (or HDL) particles. In a healthy human body, there is a balance between the delivery and removal of cholesterol. The LDL particles deliver cholesterol to organs, where it can be used to produce hormones, maintain healthy cells, and be transformed into natural products that assist in the digestion of lipids. The HDL particles remove cholesterol from arteries and tissues to transport it back to the liver for storage, recycling, and elimination through a pathway called "Reverse Lipid Transport (RLT)".

Epidemiological studies have historically demonstrated that the risk of developing cardiovascular disease appeared to be higher in patients with low HDL-cholesterol independent of the level of LDL-cholesterol, even when patients are treated with the best available standard of care. This observation can be explained by the role the HDL particle plays in the Reverse Lipid Transport (RLT) pathway, the only natural mechanism capable of removing cholesterol from peripheral tissues and delivering it back to the liver for elimination. HDL particles mediate the flux of cholesterol through the RLT and therefore act to counterbalance the delivery of cholesterol to the vessel wall by the LDL particles. The RLT is a pathway that may protect against atherosclerosis and cardiovascular disease by clearing excess cholesterol from the arterial wall. The ATP-binding cassette transporter called ABCA1 is a protein that mediates the first step of RLT and acts as a gatekeeper for eliminating excess tissue cholesterol.

Next financial press release: Cash position and revenue for H1 2015, on August 27, 2015.

About Cerenis Therapeutics: www.cerenis.com

Cerenis Therapeutics is an international biopharmaceutical company dedicated to the discovery and development of innovative HDL therapies for the treatment of cardiovascular and metabolic diseases. HDL is the primary mediator of the reverse lipid transport, or RLT, the only natural pathway by which excess cholesterol is removed from arteries and is transported to the liver for elimination from the body.
Cerenis is developing a portfolio of HDL therapies, including HDL mimetics for the rapid regression of atherosclerotic plaque in high-risk patients such as post-ACS patients and patients with HDL deficiency, and drugs which increase HDL for patients with low number of HDL particles to treat atherosclerosis and associated metabolic diseases.

Cerenis is well-positioned to become one of the leaders in the HDL therapeutic market, with a broad portfolio of programs being developed.

Since its inception in 2005, the company has been funded by top tier investors: Sofinnova Partners, HealthCap, Alta Partners, EDF Ventures, Daiwa Corporate Investment, TVM Capital, Orbimed, IRDI/IXO Private Equity and Bpifrance (Fund for Strategic Investment) and last March successfully completed an IPO on Euronext raising €53.4m.

About CER-001:

CER-001 is an engineered complex of recombinant human apoA-I, the major structural protein of HDL, and phospholipids. It has been designed to mimic the structure and function of natural, nascent HDL, also known as pre-beta HDL. Its mechanism of action is to increase apoA-I and the number of HDL particles transiently, to stimulate the removal of excess cholesterol and other lipids from tissues including the arterial wall and to transport them to the liver for elimination through a process called Reverse Lipid Transport.

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