# Dezima Reports Positive Results in its Phase 2b TULIP Trial with CETP Inhibitor TA-8995 in Dyslipidemia

***Combines outstanding efficacy and safety profile with favourable pharmacokinetics***

**Naarden, The Netherlands, 29 August 2014 –** Dezima Pharma ('Dezima'), the biotechnology company developing innovative drugs in the field of dyslipidemia, announced today that it has received very positive results in its Phase 2b TULIP clinical trial, a double blind, placebo controlled, Phase 2b dose ranging study of TA-8995 (DEZ-001), as monotherapy and in combination with statins for treating dyslipidemia.

The TULIP ("***T***A-8995: its ***U***se in patients with mild dys***LIP***idemia") study was conducted in specialized cardiovascular centres across Denmark and the Netherlands. A total of 364 patients were randomised into nine cohorts; a placebo, TA-8995 alone at different doses, or in combination with different statins. The study investigated the effects of TA-8995 on a wide range of established cardiovascular disease (CVD) biomarkers over a three months' dosing period.

The results showed dramatic effects on the primary endpoint, which was a composite of changes in lowering LDL-C and raising HDL-C, as well as strong and clinically relevant effects on other parameters including cholesterol efflux and Lipoprotein(a) (Lp(a)). There were no safety or tolerability issues identified or any pharmacokinetic concerns about potential accumulation of the drug.

"These results are clearly very exciting. Compared to other CETP inhibitors TA-8995 combines the highest levels of efficacy seen on lipid parameters with a 20-fold lower dose," stated Rob de Ree, CEO of Dezima pharma. "Combined with the excellent safety and favorable pharmacokinetic profile this positions TA-8995 as the best-in-class CETP inhibitor as we move towards Phase 3."

John Kastelein, CSO and co-founder of Dezima said: "The results emphasize that TA-8995 robustly lowers all atherogenic lipoproteins, LDL-C, the entity of non HDL-C, apoB as well as Lp(a), compatible with very significant reductions of cardiovascular risk."

Two members of Dezima's Scientific Advisory Board, both world-leading experts in the dyslipidemia field, also commented on the results:

"The TULIP study met all its predefined primary endpoints both for efficacy and safety with impressive reductions of all atherogenic lipoproteins which therefore justifies the initiation of a Phase 3 mortality and morbidity outcome trial", commented Bryan Brewer, MD, Director at Washington Cardiovascular Associates, Washington DC, USA.

Philip Barter, MD, Professor at the Center of Vascular Research, University New South Wales, Sydney, Australia added: "In the TULIP study TA-8995, in combination with statin therapy, achieved reductions of LDL-C that are close to reductions achieved by the PCSK9 inhibitor class. Moreover TA-8995 achieved these results without any apparent sign of accumulation of the compound."

The company plans to publish the full data over the coming months in peer reviewed journals. Meanwhile Dezima will continue to collect data from its ongoing studies (DDI, TQT and a pilot study on Lp(a)), while preparing for the start of pivotal Phase 3 studies in 2015.

--ENDS--

**Notes to editors**:

**About Dezima Pharma B.V.**
Dezima Pharma was founded in 2012 by Prof. John Kastelein, Professor of Medicine at the Department of Vascular Medicine at the Academic Medical Center of the University of Amsterdam, The Netherlands, and financed by Forbion Capital Partners, BioGeneration Ventures and New Science Ventures, to develop novel products to treat dyslipidemic patients suffering from cardiovascular disease (CVD). The company's lead product TA-8995 has been in-licenced from Mitsubishi Tanabe Pharma Corporation and is a potentially best-in-class CETP inhibitor. The company has an outstanding Scientific Advisory Board including world-leading experts in the dyslipidemia space such as Dr Philip Barter, Professor at The Heart Research Institute, Sydney, Australia, and Dr Bryan Brewer, Director, Washington Vascular associates Washington DC, USA.

**About dyslipidemia and CETP inhibitors**
Dyslipidemia is a generally asymptomatic disease in which serum lipid levels deviate from the normal level. It is considered to be a modifiable risk factor for cardiovascular disease due the direct relation with atherosclerosis. The market for dyslipidemic drugs, including statins, fish oils and fibrates, topped $25Bn in 2010. Though current treatment is relatively effective a high unmet need remains: about 60% of treated patients have a considerable chance of experiencing a cardiovascular event, which comes with significant morbidity and mortality.

**The Cholesteryl Ester Transfer Protein (CETP)** facilitates the transfer of cholesterol from HDL to other lipoproteins including LDL, in exchange for triglycerides. The CETP mediated transfer of cholesterol into LDL particles results into maturation of those LDL particles to more atherogenic LDL particles, which contribute to macrophage foam cell, and eventually plaque formation. Large Mendelian Randomization studies, epidemiological as well as preclinical studies have provided evidence for the notion that CETP activity is inversely related to cardiovascular mortality and reduced activity of CETP by pharmaceutical means or by naturally occurring mutations in the CETP gene results in increased HDL and decreased LDL levels. This provides a rationale for inhibition of CETP activity as a therapeutic intervention in dyslipidemic conditions characterized by either low HDL or high LDL cholesterol.

**Lipoprotein(a)** (also called Lp(a)) is a lipoprotein subclass. Genetic studies and numerous epidemiologic studies have identified elevated Lp(a) levels as a risk factor for myocardial infarction, aortic stenosis, coronary heart disease and stroke. Elevated Lp(a) levels predict risk of early atherosclerosis or stenosis independently of other cardiac risk factors, including LDL. Lp(a) concentrations are mainly genetic determined but may be affected by disease states, for example kidney failure. It is only slightly affected by diet, exercise, and other environmental factors. Most commonly prescribed lipid-reducing drugs have little or no effect on Lp(a) concentration.

**For more information, please contact:**

**Dezima Pharma:**
Rob de Ree
rderee@dezimapharma.com
+31 35 699 3000

**For media enquiries on behalf of Dezima Pharma:**
Instinctif Partners
Melanie Toyne Sewell / Dr Robert Mayer
dezima@instinctif.com
+44 20 7457 2029 / +49 89 3090 5189 13