



Kineta Partner Awarded UC Discovery Grant to Advance Preclinical Studies on Lead Autoimmune Therapeutic

University of California, Irvine to Conduct IND Enabling Studies

SEATTLE, WA, Dec. 10, 2009, – Kineta, Inc. today announced its development partner, K. George Chandy, M.D., Ph.D. of the University of California, Irvine has been awarded a grant from the University of California Discovery Fund for preclinical studies on its lead autoimmune drug, a Kv1.3 potassium channel blocker. Kineta is participating as Dr. Chandy's industry sponsor. The supported studies will further the characterization of ShK-186 in preparation for first-in-human clinical trials commencing in mid-2010.

“Kineta is very pleased to continue its work with Dr. Chandy and his outstanding team at UC Irvine. Dr. Chandy possesses extraordinary experience as lead developer of ShK-186, our peptidic Kv1.3 blocker. We are also grateful to the State of California for its recognition of the importance of public / private partnerships in advancing breakthrough science,” said Shawn P. Iadonato, Ph.D., Executive Vice President and Chief Scientific Officer at Kineta.

Dr. Chandy's scientific team will conduct animal studies to determine optimal dosing and to gain enhanced understanding of the mechanism of action of ShK-186. These studies will compliment non-clinical work and advance Kineta's IND (investigational new drug) application to the FDA.

ShK-186 is designed to suppress activation of effector memory T cells, which are important mediators of inflammation and tissue damage in multiple sclerosis, type 1 diabetes and other autoimmune diseases. These compounds have been shown to significantly reverse disease in animal models of multiple sclerosis and rheumatoid arthritis, and also have potential against a number of other autoimmune diseases mediated by effector memory T cells. Animal models have demonstrated that ShK-186 efficacy is achieved without the generalized immunosuppression that occurs with competing therapies.

Dr. Chandy is a professor of physiology and biophysics at the University of California, Irvine and an internationally recognized authority on mechanisms of autoimmunity and the role of ion channels in autoimmune disease. He is the principal investigator responsible for the discovery of more than 300 ShK analogs licensed by Kineta.

For more information on today's announcement or to schedule interviews contact Meg O'Connor moconor@kinetabio.com 206-251- 8638

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Kineta, Inc. is a Seattle-based privately held biotechnology company specializing in clinical advancement of drugs that modulate and enhance the human immune system. Our world class scientists are pioneers, with an aggressive strategy to develop life-changing classes of drugs that harness the power of the immune system to fight disease. Kineta seeks to improve the lives of millions of people suffering from autoimmune and viral diseases. Our progressive business model focuses on targeting unmet medical needs, rapid achievement of important clinical milestones and a strong return for investors. For more information on Kineta Inc. visit our website, www.Kinetabio.com

This press release contains forward-looking statements, including without limitation, all statements related to plans, future events and financial performance. Forward-looking statements involve risks and uncertainties, which could cause actual results to vary materially from those expressed in or indicated by the forward-looking statements. The companies' actual results and timing of events could differ materially, including

efficacy and demand for our products, our ability to manage our costs, competition from other drug development companies, and our ability to recruit and maintain senior management and other key personnel. The companies' stakeholders are cautioned not to place undue reliance on these forward-looking statements, which reflect only an analysis and speak only as of the date of this press release. The companies undertake no obligation to revise or update any forward-looking statements to reflect events or circumstances after the date hereof.