



Halsa Pharmaceuticals Announces ZAG Demonstrates Weight Loss, Rise in Temperature and Reduction of Diabetes Symptoms in Preclinical Studies at Obesity 2009 Annual Scientific Meeting

- *Suggests Increase in Energy Expenditure and Potential as Treatment for Obesity and Type 2 Diabetes –*

HOUSTON, Texas (October 26, 2009) ----- Houston-based Halsa Pharmaceuticals, Inc., a biotechnology company developing therapeutics for the treatment of obesity, diabetes, cachexia and other metabolic diseases, today announced results from preclinical studies demonstrating that recombinant human Zinc- α_2 -glycoprotein (ZAG) induced a progressive loss of body weight in mice of 3.5g in five days, together with a rise in temperature of 0.4°C, suggesting an increase in energy expenditure. In addition, ZAG produced a normalization of the diabetic glucose tolerance curve after three days of treatment, suggesting that ZAG may have a therapeutic application in the treatment of obesity and Type 2 diabetes. The results from this study were presented at Obesity 2009, the 27th Annual Scientific Meeting of The Obesity Society, one of the largest scientific conferences in the field of obesity, being held in Washington, D.C. October 24-28.

“ZAG acts normally in the body to regulate levels of fat, and these new results tell us that ZAG regulates the metabolic hallmarks of Type 2 diabetes as well as obesity,” said Professor Michael J. Tisdale, Aston University, Birmingham, U.K., the inventor of ZAG as a therapeutic. “Certain disease states cause very high over-expression of ZAG, and in those circumstances body fat is depleted, so we know that humans possess a fat-depletion response to ZAG. In addition, there is a low expectation of acute toxicity with ZAG because it occurs at medium prevalence in the human body, and we were pleased that these preclinical studies demonstrated this as well.”

Summary Results:

- ZAG induced a progressive loss of body weight in mice of 3.5g in five days, together with a rise in temperature of 0.4°C.
- Even before major changes in body mass, ZAG induced a decrease in blood glucose and plasma insulin levels, while there was a four-fold increase in pancreatic insulin, and normalization of the diabetic glucose tolerance curve.
- Weight loss was associated with an increase in weight of brown adipose tissue and an increase in expression of uncoupling proteins-1 and -3, which would be expected to channel metabolic substrates into heat as observed.
- ZAG-treated animals showed a significant decrease in plasma levels of triglycerides and non-esterified fatty acids.
- ZAG administration also increased plasma glycerol, which is indicative of an increased lipolysis and a decrease in carcass fat mass.
- ZAG administration increased glucose uptake into adipose tissue and skeletal muscle both in the absence and presence of insulin, associated with an increased expression of glucose transporter 4.

- Due to an increase in protein synthesis and a decrease in protein degradation through the ubiquitin-proteasome pathway, there was an increase in skeletal muscle mass.

“Halsa already has extensive animal proof-of-concept data on ZAG, along with an understood and novel mechanism-of-action and robust patent rights, and we look forward to bringing this compound into clinical testing,” commented Halsa Chief Executive Officer Phil Speros. “These new results demonstrate that the anti-obesity action of ZAG is balanced with metabolic pathways to utilize and dispose of fat breakdown products, pathways that also act to mitigate diabetes.”

ZAG is a recombinant protein (biologic) being developed into a prescription pharmaceutical product which will increase ZAG levels in obese patients to normal levels and reduce body fat to normal levels. ZAG is a natural regulator of fat in humans and other animals. Higher ZAG levels cause fat depletion, and lower levels allow fat accumulation. ZAG utilizes a novel biochemical pathway and acts directly on adipose - it does not impact food intake, water intake, digestion or activity. There is strong scientific evidence that this compound causes fat depletion in humans, as ZAG is an adipokine responsible for loss of adipose tissue in cancer cachexia, and earlier preclinical studies have demonstrated proof-of-concept data in animals. Halsa has exclusive intellectual property rights to the therapeutic.

ABOUT HALSA PHARMACEUTICALS

Halsa Pharmaceuticals, Inc. is a biotechnology company that was founded in 2000 and is developing therapeutics for the treatment of obesity, diabetes, cachexia and other metabolic diseases.

SAFE HARBOR STATEMENT

Under the Private Securities Litigation Reform Act of 1995, a “safe harbor” may be provided to us for forward-looking statements. Words such as “outlook,” “believes,” “expects,” “appears,” “may,” “will,” “should,” “anticipates” or the negative thereof or comparable terminology, are intended to identify these forward-looking statements. These forward-looking statements are estimates reflecting the best judgment of our senior management and are based on our current expectations and projections concerning future events, many of which are outside of our control, and involve a number of risks and uncertainties that could cause actual results to differ materially from those suggested by the forward-looking statements.

CONTACT:

Phil Speros - 832.722.0513

psperos@halsapharma.com

or

Kathryn Morris, PRonCall (media)

845-635-9828

kathryn@proncall.com