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***In Vivo* Data on Taligen Therapeutics' Pipeline Candidates Presented at the XXII International Complement Workshop**

CAMBRIDGE, Mass., Oct. 7, 2008 –Taligen Therapeutics Inc. today announced that *in vivo* data on several of the Company's pipeline candidates were presented at the XXII International Complement Workshop in Basel, Switzerland. The data highlighted the candidates' efficacy in several animal models of complement-mediated diseases.

"These highlighted presentations, as well as several *in vitro* studies presented at the conference, reveal the deep expertise and expanding knowledge base within the complement research community," said Michael Holers, M.D., chief scientific officer of Taligen Therapeutics. "The work, not only from our laboratory but from the laboratories of our collaborators, is unlocking the mechanisms of immunomodulation and enabling Taligen to direct the development of new product candidates and test them in a variety of animal models as a precursor to their clinical development."

"The opportunity to further evaluate Taligen's TT30 compound in our research activities has been invaluable in our characterization of the effects of modulation of the complement system on animal models of human disease," said Steve Tomlinson, Ph.D. and principal investigator of several studies presented at the complement meeting. "Our results in the MRL lupus model in collaboration with Dr. Gary Gilkeson's group now show that targeted complement inhibition using TT30 leads to both local control of inflammation as well as a substantial decrease in the autoimmune response *in vivo*. No other type of compound has this dual profile of

activity. I also believe these CR2-targeted compounds will help future research in the lupus area and hopefully provide therapeutic options for patients.”

Key findings from several of the studies testing Taligen’s product candidates are highlighted below:

Data support targeting the alternative complement pathway using TT30 as an immunomodulatory compound for treating complement-mediated diseases

In vivo studies in models for several complement-mediated diseases showed that targeted inhibition of the alternative complement pathway slowed or halted disease progression in these models. Sekine et al. investigated whether TT30 prevented progression of renal disease in a lupus nephritis murine model (Poster #174). TT30 is a fusion protein coupling domains from complement receptor 2 (CR2) with the alternative pathway inhibitor factor H (fH). Treatment with the CR2 domain alone led to decreases in autoantibodies, while treatment with TT30 resulted in additional beneficial effects including significantly reduced renal injury, proteinuria and anti-dsDNA antibody levels compared to controls. Therefore, the dual action of TT30 (CR2 and fH) prevented disease progression in this animal model.

Thurman et al. (Poster #171) compared the effect of systemic administration of TT30 to the effect of CR2-Crry in an airway hyper-responsiveness asthma animal model. CR2-Crry is an inhibitor of the alternative, classical and lectin complement pathways whereas TT30 inhibits only the alternative pathway. The study showed that both TT30 and CR2-Crry significantly reduced airway hyper-responsiveness. This suggests that targeting the alternative complement pathway, while keeping the classical and lectin complement pathways unaffected, may be sufficient to control complement-mediated diseases and thus reduce the risk of infection.

Wet AMD animal model study suggests an alternative to intraocular drug administration

Currently marketed therapies for wet AMD require intraocular injections. In a wet AMD animal model, Rohrer et al. (Poster #202) tested whether TT30 could be

effective in preventing progression of choroidal neovascularization following argon laser photocoagulation if TT30 was injected intraocularly or intravenously. The Rohrer team demonstrated that TT30 prevented progression of choroidal neovascularization regardless of the mode of administration. Besides adding further confirmation to the therapeutic potential of targeting the alternative pathway in complement-mediated diseases, the study showed intravenous administration of TT30 could specifically target a site of complement activation and be an effective alternative to intraocular injections.

Growing pipeline

Additional *In vivo* data were presented for CR2-Crry, a previously undisclosed Taligen pipeline candidate. CR2-Crry is a fusion protein combining CR2 with Crry, a murine analogue of human complement receptor 1 (CR1), which appears to inhibit all three complement pathways. Atkinson et al. showed in a myocardial ischemia/reperfusion injury study (Poster #196) that single doses of CR2-Crry and TT30 were equivalent in protecting heart grafts from ischemia/reperfusion injury. However, when both compounds were tested for their effectiveness in preventing antibody-mediated graft rejection, multiple doses of CR2-Crry were superior to TT30 in increasing graft survival. The study showed the critical importance of the alternative complement pathway in ischemia-reperfusion injury but also highlighted the role of the classical and lectin pathways in antibody-mediated rejection.

About Taligen Therapeutics

Taligen Therapeutics is focused on the discovery and development of novel protein therapeutics that are designed to modulate the alternative pathway of the complement system to treat a wide range of inflammatory conditions and diseases. The Company's lead therapeutic candidates are monoclonal antibodies and recombinant fusion proteins that target key factors in the alternative pathway, which Taligen's founders have validated as an important amplification loop in the inflammation process. Taligen's headquarters and late-stage research and development operations are located in Cambridge, Mass. The company also maintains a drug discovery operation in Denver that is closely aligned with an international complement biology research network organized by Michael Holers,

M.D., one of the Company's founders. For more information, visit www.taligetherapeutics.com.

This press release contains forward-looking statements that involve risks and uncertainties, including statements relating to efficacy of the Company's product candidates and potential advantages of the Company's technology and product candidates. Actual results could differ materially from those projected and the Company cautions readers not to place undue reliance on the forward-looking statements contained in this release.

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