

## FOR IMMEDIATE RELEASE

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### **Data from *In Vivo* Disease Models on Taligen Therapeutics' Pipeline Candidates to be Presented at the XXII International Complement Workshop**

#### ***Presentations Include Proof-of-Concept Animal Data on Previously Disclosed Fusion Protein TT30 and New Molecules TT32 and mAb171***

CAMBRIDGE, Mass., Sept. 25, 2008 –Taligen Therapeutics Inc. today announced that data from *in vivo* disease models on several of the company's pipeline candidates will be presented next week at the XXII International Complement Workshop in Basel, Switzerland.

The data, which will be presented in eight oral and poster presentations, are the results of research conducted by the laboratories of Michael Holers, M.D., chief scientific officer of Taligen, and the company's academic collaborators in the complement research community. Five studies testing the company's fusion protein TT30 and two new compounds, a fusion protein TT32 and a monoclonal antibody mAb171, in animal models are highlighted below. These data further support Taligen's approach that targeting the alternative complement pathway can impact complement-mediated inflammatory disease processes.

#### *Poster Presentations:*

#### **TT30**

#### **Targeted inhibition of the alternative pathway by CR2-fH fusion protein ameliorates progression of renal disease in MRL/lpr mice**

Hideharu Sekine\*, Gary Gilkeson, Stephen Tomlinson

Oct. 1, 2008, Posters - 10:30 a.m. - noon, Session 2, Poster 174

**Alternative complement pathway signaling in a mouse model of choroidal neovascularisation**

Bärbel Rohrer\*, Qin Long, Brooks Wilson, Beth Coughlin, Yuxiang Huang, Fei Qiao, Peter Tang, Kannan Kunchithapautham, Gary Gilkeson, Stephen Tomlinson

Oct. 1, 2008, Posters - 10:30 a.m. - noon, Session 2, Poster 202

**TT30 and TT32**

**Targeted complement-inhibitory proteins CR2-Crry and CR2-fH ameliorate allergen-induced airway hyperresponsiveness and inflammation**

Joshua Thurman\*, Katsuyuki Takeda, Stephen Tomlinson, Erwin Gelfand, V. Michael Holers

Oct. 1, 2008, Posters - 10:30 a.m. - noon, Session 2, Poster 171

**The role of complement in myocardial ischemia/reperfusion injury and antibody-mediated rejection in a mouse heterotopic heart transplant model**

Carl Atkinson\*, Yongquan Gong, Yuxiang Huang, Emily Pauling, Martin Goddard, Stephen Tomlinson

Oct. 1, 2008, Posters - 10:30 a.m. - noon, Session 2, Poster 196

*Oral Presentation:*

**mAb171**

**Treatment with a highly inhibitory monoclonal antibody directed to the C3d ligand binding site on human CR2/CD21 suppresses antigen-specific immune responses in vivo**

Liudmila Kulik\*, Jonathan P. Hannan, Brigitte T. Huber, V. Michael Holers

Oct. 1, 2008, Animal models (Room: Montreal), 9:30 a.m. - 9:45 a.m., Oral 49

**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 under Taligen's chief scientific officer Michael Holers, M.D., which closely collaborates with an international complement biology research community. For more information, visit [www.taligetherapeutics.com](http://www.taligetherapeutics.com).

*This press release contains forward-looking statements that involve risks and uncertainties, including statements relating to development 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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