

Emerging Company Profile**Taligen: De-amplifying complement**

By Matthew Mikulski
Staff Writer

Taligen Therapeutics Inc. is taking two approaches to modulating complement for inflammatory diseases. The company thinks its lead antibody, TA-106, can modulate the pro-inflammatory system while still preserving the body's natural immune response. A second program targets complement-mediated inflammation locally to reduce systemic toxicity and the amount of inhibitory protein needed for therapeutic effect.

Complement is an enzymatic cascade that is part of the innate arm of the immune system and leads to inflammation in response to pathogens. The complement cascade has three arms: classical, lectin and alternative. Taligen is focusing on the alternative arm, which amplifies the activity of the other two.

According to Chairman, President and CEO Woodruff Emlen, the alternative pathway is implicated in rheumatoid arthritis (RA), lupus nephritis, asthma and macular degeneration.

Taligen's TA-106 inhibits Factor B, a serine proteinase that is unique to the alternative pathway and exists upstream of complement proteins other companies are targeting for inflammation, including complement 3 (C3) and C5.

Because Factor B is not necessary for signal transduction in either the classical

Taligen Therapeutics Inc.

Aurora, Colo.

Technology: Regulation of the alternative complement pathway

Disease focus: Inflammation

Clinical status: Preclinical

Founded: 2004 by Michael Holers and Woodruff Emlen

University collaborators: University of Colorado at Denver and Medical University of South Carolina

Corporate partners: None

Number of employees: 4

Funds raised: \$3.8 million

Investors: Sanderling Ventures; Tango Ventures; and High Country Ventures

CEO: Woodruff Emlen

Patents: 1 issued covering the structure of complement receptor 2 (CR2), its ligand binding site and related therapeutic uses

or lectin pathways, animals missing the protein can generate a normal immune response to foreign antigens, whereas mice lacking C3, a protein participating in both the classical and lectin pathways, cannot. Thus, Taligen believes inhibiting Factor B can reduce immune hyperactivity without ablating important host defense mecha-

nisms.

TA-106 has shown efficacy in mouse models of several inflammatory diseases, but will initially be developed to treat asthma. Taligen hopes to begin Phase I testing of a nebulizer formulation of the antibody next year.

The company's second program creates fusion proteins consisting of a complement inhibitor and a binding site for a complement activation product, thus targeting the inhibitor to the site of inflammation.

The company's lead candidate from the platform will look to treat age-related macular degeneration (AMD) by delivering Factor H, a down-regulator of the alternative pathway, to sites of complement activation. Taligen hopes to begin clinical testing in 2009.

Because the product will be preferentially distributed to areas of complement activation, Emlen anticipates it will cost less and result in lower systemic side effects than other complement inhibition strategies in AMD (see *BioCentury*, Aug. 14, 2006).

Taligen will look to raise \$15-\$20 million in a series B round by year end, with the expectation that the cash will take its lead program through Phase II and the second program to an IND. The hope for both programs is to find a partner after proof of concept.

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