



FOR IMMEDIATE RELEASE

## **THROMBOGENICS PRESENTS POSITIVE POOLED RESULTS FROM THE MIVI-TRUST PHASE III PROGRAM**

*Data Presented at Royal Hawaiian Eye Meeting Confirms the Potential of Ocriplasmin (Microplasmin) as a Treatment Option for Symptomatic Vitreomacular Adhesion (sVMA)*

**MAUI, USA and LEUVEN, BELGIUM** – 20 January, 2011 – ThromboGenics NV (Euronext Brussels: THR), a biopharmaceutical company focused on the discovery and development of innovative treatments for eye disease, cardiovascular disease, and cancer, announces that the pooled results from the successful ocriplasmin (microplasmin) MIVI-TRUST phase III program were presented today at the Royal Hawaiian Eye (RHE) Meeting in Maui, Hawaii. The MIVI-TRUST program is the largest interventional clinical program ever performed to specifically evaluate the pharmacological treatment of symptomatic vitreomacular adhesion (sVMA) recruiting a total of 652 patients at 90 centers across the United States and Europe.

The pooled results of the TG-MV-006 and TG-MV-007 phase III trials were presented by Dr. Steven D. Schwartz (Jules Stein Eye Institute, UCLA, CA). These results demonstrate the potential of ocriplasmin to transform the treatment of sVMA.

The phase III program showed that both the TG-MV-006 and TG-MV-007 trials of ocriplasmin vs placebo:

- Met the primary end point: Achieving a statistically and clinically significant improvement in the resolution of VMA
  - 26.5% of subjects had pharmacological resolution of VMA at day 28 ( $P < .001$ )
- Demonstrated pharmacological resolution of VMA in 34.5% of subjects without an epiretinal membrane (ERM) ( $P < .001$ )
- Met the secondary end points: (1) pharmacological closure of full-thickness macular hole (FTMH) and (2) total posterior vitreous detachment (PVD)

- 40.6% of subjects had pharmacological closure of FTMH at day 28, which was maintained at month 6 ( $P < .001$ )
- 13.4% of subjects had induction of total PVD ( $P < .001$ )
- Visual acuity and visual function questionnaire outcomes favored ocriplasmin
- Ocriplasmin was generally well tolerated

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Regarding the presentation and trial results, Steven D. Schwartz, MD, commented, “Based on the phase III results and my experience with ocriplasmin, I am extremely hopeful and confident that it will become a key therapy in the treatment of a wide spectrum of common sight threatening retinal diseases whose common pathologic feature is symptomatic vitreomacular adhesion or sVMA. In this trial macular hole, macular pucker and vitreomacular traction syndrome were addressed. The treatment works rapidly and the effect is durable because relief of sVMA is a binary event that does not recur. This new treatment has the potential to provide an early, relatively safe and effective pharmacologic option for patients who often do not have treatment option until they progress to surgery. Release of VMA in the operating room may also benefit from pretreatment with this agent”

In addition to the data presented at the RHE meeting, microplasmin data is anticipated to be presented at:

- *Macula meeting*  
Julia Haller, MD, “Microplasmin for Vitreomacular Traction”  
Saturday, January 29, 3:40 PM
- *Angiogenesis 2011*  
Andrew A. Moshfeghi, MD, MBA, “Microplasmin in Clinical Practice”  
Saturday, February 12, 10:30 AM
- *Retinal Physician Symposium 2011*  
Pravin Dugel, MD, “Pharmacologic PVD: Microplasmin MIVI-TRUST Trial Results”  
Friday, February 25, 12:50 PM

## Notes to Editors

**About Symptomatic Vitreomacular Adhesion** Symptomatic Vitreomacular adhesion (sVMA) is a disease of the eye that primarily involves the vitreous, macula, and retina—all structures important for visual acuity needed to read, drive, and perform other activities. Patients that have asymptomatic VMA are usually followed up without further intervention, however, the usual trigger for further intervention is the presence of symptomatic VMA. The vitreous is a clear jelly-like substance within the

eye that takes up the space behind the lens and in front of the retina, the light-sensitive layer at the back of the eye responsible for central vision. .

With age, the vitreous begins to liquefy, creating fluid-filled areas that can coalesce or combine to form pockets of vitreous that are mostly liquid with very small concentrations of collagen. If these areas are close to the interface between the vitreous and the retina, they can cause complete separation of the vitreous from the retina in a normally occurring process called posterior vitreous detachment (PVD). If the separation of the vitreous from the retina is not complete, areas of focal attachment or vitreomacular adhesion (VMA) can occur. The incomplete separation of the vitreous from the retina is called pathologic PVD.

VMA occurring as a result of pathologic PVD can create pulling forces, or “traction,” on the retinal surface. When this pulling action/traction occurs over the macula (the most sensitive part of the retina, and the area which facilitates central vision) then this can lead to the development of VMA-related complications, such as macular puckers and macular holes, each of which can lead to distorted vision (a condition called metamorphopsia), a loss of visual acuity and/or blindness. Traction can also pull on retinal blood vessels, causing bleeding, or on the optic nerve, causing disruption in the nerve signals being sent to the vision centers in the brain. When VMA causes symptoms such as metamorphopsia, or visual impairment then this is known as Symptomatic Vitreomacular Adhesion (sVMA).

There is evidence that VMA is associated with the development of several common eye disorders that can potentially cause blindness, including macular hole, age-related macular degeneration (AMD), and diabetic macular edema (DME).

### **Diagnosis and Treatment of sVMA**

Careful eye examination by a vitreoretinal specialist is critical for diagnosing VMA. Optical coherence tomography (OCT) has significantly improved the accuracy of diagnosing VMA.

Today, the standard of care in the treatment of sVMA is to either do nothing (“observation”) or perform a vitrectomy, a surgical procedure that induces PVD and removing the vitreous gel, thus releasing the VMA. An estimated 850,000 vitrectomy procedures are performed annually—250,000 in the United States alone. During a vitrectomy, the surgeon uses specialized instruments to remove the aging vitreous and replace it with a saline solution. Although the surgery is efficient at removing the vitreous, vitrectomy is an operative procedure that potentially carries risks for the patients’ eyes, including bleeding, infection and, in rare cases, even blindness.

Recently reported results of the MIVI-TRUST (Microplasmin for IntraVitreous Injection-Traction Release without Surgical Treatment) phase III clinical trial program suggest that a new drug called ocriplasmin (microplasmin) may provide patients with an additional treatment option. Ocriplasmin (microplasmin) is believed to weaken the adhesions associated with pathologic PVD, primarily by targeting the fibronectin, laminin, and collagen fibers that adhere the vitreous to the retina. Ocriplasmin may also target these same fibers in the inner limiting membrane.

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### **The MIVI-TRUST Program**

The ocriplasmin (microplasmin) phase III program, referred to as MIVI-TRUST, consists of 2 multicenter, randomized, placebo-controlled, double-masked trials. These trials were designed to evaluate a single dose of 125 µg ocriplasmin vs placebo (vehicle) administered intravitreally for the treatment of patients with sVMA. The primary end point of both trials was the pharmacological resolution of sVMA 28 days after a single injection of ocriplasmin. This end point was assessed using OCT. The MIVI-TRUST program is the largest interventional clinical program ever performed to specifically evaluate the pharmacological treatment of sVMA. In total, more than 650 patients were enrolled in these trials, which were held across 90 centers in 7 countries.

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**About ThromboGenics**

ThromboGenics is a biopharmaceutical company focused on the discovery and development of innovative medicines for the treatment of eye disease, vascular disease, and cancer. The Company's lead product ocriplasmin (microplasmin) has completed two successful phase III clinical trials for the pharmacological treatment of symptomatic vitreomacular adhesion (sVMA). ThromboGenics will commercialize this product via its own dedicated sales force in the United States and in Europe.

In addition, ThromboGenics is developing novel antibody therapeutics in collaboration with BioInvent International; these include TB-402 (anti-Factor VIII), a long-acting anticoagulant in phase II, and TB-403 (anti-PlGF) in phase Ib/II for cancer in partnership with Roche.

ThromboGenics has currently around 75 employees at its headquarters in Leuven, Belgium, and at its facilities in Ireland and the United States. The Company is listed on Eurolist by Euronext Brussels under the symbol THR.

**Important information about forward-looking statements**

Certain statements in this press release may be considered “forward-looking.” Such forward-looking statements are based on current expectations, and, accordingly, entail and are influenced by various risks and uncertainties. The Company therefore cannot provide any assurance that such forward-looking statements will materialize and does not assume an obligation to update or revise any forward-looking statement, whether as a result of new information, future events, or any other reason. Additional information concerning risks and uncertainties affecting the business and other factors that could cause actual results to differ materially from any forward-looking statement is contained in the Company’s Annual Report.