



## A SYNOPSIS OF THE FIRST TUTZING (MUNICH) ANTIPHOSPHOLIPID CONFERENCE

This conference was held in Tutzing, Germany (south of Munich) on April 22nd through the 25th, 2002 and provided a unique opportunity for researchers to present and discuss the most recent advances in basic science and clinical developments related to the antiphospholipid syndrome (APS). Meeting participation was limited to those presenting abstracts in a workshop setting which enhanced the exchange of information and views on this rapidly evolving field. The program and list of participants with post event updates can be found at [www.tac2002.de](http://www.tac2002.de). In addition, abstracts of the presentations have been published in *Immunobiology 205:133-192(2002)*. A brief summary of some of the most relevant topics discussed is presented below.

To open the conference, some of the leading researchers in the APS field presented their perspectives on the pathogenesis of APS. G. Hughes (London) reviewed the history of antiphospholipid antibodies and commented that future testing to assess the risk of thrombosis may involve the determination of antibodies to Protein S-phospholipid complexes, probably to mimic some *in vivo* biological membrane events. Y. Shoenfeld (Tel Aviv) discussed the association of APS with SLE and speculated that the presence of different antiphospholipid antibodies may determine the nature of the clinical manifestations presented by patients. In addition, he pointed out the importance of testing for IgA antiphospholipid antibodies for an adequate serologic evaluation of APS. Colleagues of A. Gharavi (Atlanta) presented his work on the induction of pathogenic antiphospholipid antibodies in mice immunized with CMV (cytomegalovirus) peptides. These results generated discussions about viral infections as possible triggers for antiphospholipid antibodies.

On the topic of protein cofactors in APS, T. Koike (Sapporo) tested the binding of anti-beta-2 glycoprotein 1 (anti-B2GP1) antibodies in Asian and

Caucasian populations and found more binding to domain IV than to other domains of the B2GP1 molecule in the Asian population. Studies conducted by M. Iverson (San Diego), however, demonstrated that the binding occurred only to domain I with his patient population. These results may indicate a difference in epitope specificity related to ethnic background. Interestingly, Iverson's group has developed a tolerogenic drug for the treatment of APS which targets antibodies to domain I of the B2GP1 molecule. This drug is currently in Phase I clinical trials. E. Matsuura (Okayama) presented his studies on the ligand (oxLig-1) responsible for the binding of B2GP1 with oxidized LDL (oxiLDL). Elevated serum levels of oxiLDL and antibodies to oxLig-1 showed a better correlation with arterial than venous thrombosis in APS patients. These results suggested a possible mechanism for antiphospholipid antibodies in the development of atherosclerosis. This topic generated active discussions as these serologic markers may help to distinguish various clinical features of APS and may represent new directions in APS research. E. Gromnica-Ihle (Berlin) tested for oxiLDL in autoimmune patients and failed to see a correlation with atherosclerosis. This may be due to the absence of B2GPI in Gromnica-Ihle's system.

Several groups presented data on the importance of testing for multiple antiphospholipid antibodies when screening for APS. D. Wagenknecht and J. antiphospholipid antibodies (antiphosphatidylserine [aPS], antiphosphatidylethanolamine [aPE], etc.) allows the detection of more and probably clinically relevant, positive reactors. The addition of anti-B2GPI and aPS to anticardiolipin (aCL) testing significantly increased the number of positive reactors in patients with a history of stroke (S. Levine, New York). P. von Landenberg (Regensburg) and R. Wöhrle (Munich) independently concluded that the presence of more than one of the different antiphospholipid antibodies in the sera of patients with autoimmune

(see Tutzing Conference, pg. 2)

## READER ANNOUNCEMENTS

- The 48<sup>th</sup> **Scientific and Standardization Committee (SCC) Meeting** of the International Society on Thrombosis and Haemostasis will be held in Boston, MA, July 18 – 20, 2002. **Corgenix** will participate in the standardization discussions related to the **Corgenix** antiphospholipid and hemostasis products. Registration information and a meeting schedule can be found online at: [www.med.unc.edu/isth/bostonssc.htm](http://www.med.unc.edu/isth/bostonssc.htm).

- **Mayo Clinic's annual Coagulation Conference, "Bleeding and Thrombosing Diseases: the Basics and Beyond,"** and associated Coagulation Wet Workshop will be held July 25-27, 2002, at Mayo Medical Center, Rochester, MN. **Corgenix** will participate as a co-sponsor for the Conference, which will examine basic and clinical aspects of hemostasis and blood coagulation in relation to common laboratory tests. For more information, call (800) 323-2688 or visit their website at: [www.mayo.edu/cme](http://www.mayo.edu/cme).

- The 54<sup>th</sup> **AACC Annual Meeting and Clinical Lab Exposition** will be held July 28 – August 1, 2002, in Orlando, FL. We encourage you to visit the **Corgenix** booth (#3037) during the Exposition, which will be open daily from Tuesday, July 30 through Thursday, August 1. The following **Corgenix** abstracts have been accepted for presentation during the poster sessions at 10:00 am on August 1, 2002 and published in *Clinical Chemistry*:

**Evaluation of 3 monoclonal antibodies to functional epitopes of vWF for the diagnosis of type II vWD by ELISA.** Lopez D, et al. Abstract #E22, Hematology/ Coagulation Session.

**Performance of 3 commercial anti-B2GP1 antibody ELISA test kits on selected thrombosis samples.** Dier K, et al. Abstract #E42, Immunology Session.

- In preparation for CE-Marking, **Corgenix** has adopted the dating format in the European standard BS EN375 - Information Supplied by the manufacturer with in vitro diagnostic reagents for professional use. Effective June 1, 2002, expiration dates will be formatted CCYY-MM-DD. For example, July 15, 2002 will be expressed as 2002-07-15.

## READER PRODUCT FEATURE

### **REAADS Anti-Phosphatidylserine (aPS) Semi-Quantitative Test Kits**

For *In Vitro* Diagnostic Use

Assay format -	96-well microtiter plate (8 x 12 strips) with breakaway wells
Sample matrix -	Human serum or plasma collected in 3.2% sodium citrate
Sample dilution -	1:50
Antigen -	Phosphatidylserine
Conjugate -	Horseradish peroxidase (HRP) / goat anti-human IgG, IgM or IgA
Chromogenic substrate -	TMB (single component)
Stopping solution -	0.36 N Sulfuric acid
Assay incubations	
Sample -	15 min @ room temperature
Conjugate -	15 min @ room temperature
Substrate -	10 min @ room temperature
Wavelength -	450 nm
Clinical specificity -	IgG 96%; IgM 96%; IgA 95%
Clinical sensitivity -	Primary APS: IgG 84%; IgM 60%; IgA 36%; Secondary APS: IgG 75%; IgM 16%; IgA 40%
Product numbers:	030-001: 96 well aPS IgG/IgM 030-002: 288 well aPS IgG/IgM 10206: 96 well aPS IgA 10497: 288 well aPS IgA

### **Tutzing Conference** (cont. from pg. 1):

diseases was a strong predictor and represented an additional risk factor for vascular disease and thrombosis.

This very successful meeting represented an open forum for active and fruitful discussions among leading antiphospholipid researchers. The second Tutzing meeting was proposed to take place in year 2004.

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