A COMMON HOPE, AN UNCOMMON BOND

The Quarterly Journal of the International Pemphigus Foundation

the quarterly

Fall 2005 • Issue #42

CRP & Vascular Disease

Is there a connection? IPF founder Janet Segall shares her personal story of loss and awareness.

See page 5

Fundraising For Everyone

Outside of traditional fundraising options, JustGiving.com offers a modern way of supporting the IPF.

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In this Issue

PEMPHIGUS 2005 HAILED A SUCCESS

Because of the scientific focus, it was difficult to summarize the meeting in non-scientific terms. We would like to thank Dr. John Stanley for his assistance in editing this article for this publication.

On June 16 & 17, 2005, the IPF and the American Autoimmune Related Diseases Association (AARDA) sponsored a two day meeting, "Pemphigus 2005: Progress and Future Directions." With the help of several sponsors, and a generous grant from the NIH, experts from around the world gathered to discuss the mechanisms of causation and progress of treatment.

The IPF would like to thank the NIH, especially **Drs. Stephen Katz** and **Alan Moshell** for their continued support of the IPF and these scientific meetings.

Event Organizers (l-r): Dr. Sergei Grando, Dr. Luis Diaz, Ms. Janet Segall, Dr. Jean-Claude Bystryn, and Dr. John Stanley

We also would like to thank **Centocor**, **Aspreva**, **AMGEN**, **Peptimmune**, and **ZLB Bering** for their contribution to the meeting and their roles in helping patients with pemphigus and pemphigoid with new treatment options.

The meeting opened with remarks from Dr. Stephen Katz, Director of NIAMS, and **Dr. Jean-Claude Bystryn**, NYU Medical Center and Head of the IPF Medical Advisory Board. They both spoke on the importance of having a meeting such as this, and the importance of the IPF to patients and doctors.

After Drs. Katz and Bystryn, **Janet D. Segall**, IPF Director of Patient Services, welcomed everyone and talked about pemphigus from a patient's point of view *(See her speech on page 2)*.

Talks on Day 1 from doctors and researchers focused on the science and advances in understanding the causes of pemphigus. Drs. Yasuo Kitajima, Yorum Milner, Carlos Pincelli, and Eliane Mueller discussed mechanisms of why cells adhere to one another, and why they then fall apart in pemphigus. They discussed whether the death of a cell causes the cells to separate, or does the separation cause cell death. The consequences of autoantibodies (antibodies that are produced against one's own cells) binding to skin cells and then causing the separation of those cells were the subject of several studies. The studies of Dr. **Yoram Milner** of Hebrew University in Jerusalem (Israel) and **Carlo Pincelli** of the Univesity of Modena (Italy) emphasized the importance of cell death as an early event in the disease process of pemphigus, although others questioned the presence of cell death in the mouse model and in human skin.

Several speakers showed data that implicated "signaling" in pemphigus. Signaling occurs

continued on page 4...

THE PATIENT'S PERSPECTIVE

PATIENT CARE IS THE FOCUS

Janet Segall, Director of Patient Services

It has been a busy summer. Scott Leigh is no longer employed by the IPF and our scientific meeting was a success! This is my speech given at **PEMPHIGUS 2005: Progress and Future Directions** held in Bethesda MD June 16-17, 2005. These were my opening remarks to the doctors and researchers in attendance. I spoke about how everything the IPF does is for the patients and how they as medical professionals are the ones we look to for the answers and the cures.

"As a patient with PV for over 20 years, and the Founder of the IPF, I have learned a lot about the disease. In a broader sense, patients are all looking for one of two things: a cure, or if not a cure, a less dangerous drugs. As a patient, when I look at all of you, the one thing I think about is – You are the people who have my life in your hands. Fix me. For you, today, it is about the science. For me it is about my life.

I know that all of you feel compassion for what a patient goes through. And, we are glad you are on our side. Because of all of you and those who have gone before you, most of us, especially in the U.S., have not succumbed to the disease. I'm not so sure about those in other countries with pemphigus, but, more are alive today than ever before.

We feel, like anyone suffering from a life-threatening disease, that our life is just as important to the world as anyone else's living with their disease. Maybe there aren't as many of us with pemphigus as with autoimmune diseases such as Rheumatoid Arthritis or Lupus, or even diseases such as AIDS, Cancer, or Heart Disease. We know that finding treatments and cures for these diseases would save millions of lives. We know that. But, we also know that researching pemphigus might help do that. We worry about all these other diseases because often we can get these diseases too. But, for many pemphigus patients, it is often about the pemphigus first.

I have a friend with pemphigus who developed breast cancer. To her, it's still about the pemphigus more than about the cancer. There's another patient who has been diagnosed with inactive TB, but has PV. Will the drugs she has to take to suppress her immune system turn her inactive TB into active TB, and if so, will the drugs to handle the TB work? For her, it is about the pemphigus.

I talk with patients from all over the world – from Singapore, China, India, and Pakistan. From Serbia and Croatia. From South Africa to South America. In some of these places, patients do not get the treatments they need. Until humanity becomes more important than the dollar, the yen, or the euro, we, the patients will continue to suffer and die from pemphigus, AIDS, cancer, heart disease, malaria, smallpox and whatever crazy disease is out there.

So, we want to thank all of you for your interest in our disease, and in us. We also want to thank you for your continued support of the IPF. We remind you to mention the IPF to your patients, and encourage them to support us financially. Our existence is important to patients. We help them on many levels. We help you too. Help us make sure we continue. Since the dollar is more important right now, we need those dollars to keep us going." ●

CORRECTIONS

The *Quarterly* (SPRING 2005, #41) should have read Summer 2005, Issue #41.
The zip code for the IPF is not 98518. The correct zip code is 95815-4609.
We apologize for these and any other oversights.

The Pemphigus Quarterly

A publication of the International Pemphigus Foundation

THE QUARTERLY STAFF: Editor in Chief - ***Janet D. Segall***; Creative Director - ***Matt Cole;*** Layout/Design - ***William J. Zrnchik II***

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International Pemphigus Foundation is a 501(c)(3) nonprofit organization.

Our goals are to increase awareness of pemphigus and pemphigoid among the public and the medical community; to provide information and emotional support to pemphigus or pemphigoid patients and caregivers; to provide referrals to specialists; and to support research into advanced treatments and a cure.

Founded in 1994.

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The Quarterly is provided free of charge to our donors as a thank you for their support.

The material presented in our journal is not intended as medical advice. Readers are urged to consult their physicians before making any changes in their health regimen.

The opinions of contributors are not necessarily those of the International Pemphigus Foundation.

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BOARD OF DIRECTORS & MEDICAL ADVISORY BOARD ADDITIONS

EXPERIENCE AT WORK FOR YOU

The IPF is proud to announce some recent additions to our Board of Directors and our Medical Advisory Board. We look forward to the experience and dedication each brings to our mission. Dan Goodwill has joined our Board of Directors and Drs. Russell Hall and Robert Swerlick have joined our Medical Advisory Board.

Dan Goodwill is President of *Dan Goodwill & Associates Inc.* He has over 23 years of experience in the logistics and transportation industry in both Canada and the United States. Dan has held many executive level positions in the industry including President of Yellow Transportation's Canada division, President of Clarke Logistics (Canada's largest Intermodal Marketing Company), and General Manager of the Railfast division of TNT.

Dan holds an MBA from McMaster University in Hamilton, Ontario, an Honors B.A. from McGill University in Montreal, Quebec, and a P. Log (Professional Logistician) Certificate from The Logistics Institute, Toronto. He is fluent in French and English and been published in *Logistics Quarterly* and several other publications. Dan has spoken at Council of Supply Chain Management Professionals (CSCMP) and other industry workshops and conferences. He has been active in CSCMP for over ten years, having served as Vice President, Programs for the Toronto Roundtable and hosted the transportation track at the CSCMP Annual Meeting in Kansas City in 2001, the best attended track out of 40 tracks that year.

Dan formed and led the Toronto Pemphigus Support Group for nine years. Working in conjunction with Dr. Daniel Sauder, they developed a checklist for newly diagnosed Pemphigus patients.

Dr. Russell P. Hall, III, M.D., attended medical school at the University of Missouri's School of Medicine. He completed his internal medicine and dermatology training at the University of Missouri Medical Center after completing an internal medicine internship at St. Louis University. Dr. Hall trained as a fellow at the National Institutes of Health under Dr. Steven Katz (current director of NIAMS). He is currently Chief and the J. Lamar Callaway Professor of Dermatology with the Division of Dermatology at Duke University Medical Center.

Dr. Hall is currently conducting clinical trials on the use of new biologics in the treatment of patients with pemphigus vulgaris and bullous pemphigoid, as well as doing studies aimed at furthering our understanding of the mechanisms of action of these drugs.

Dr. Hall is a member of The Society for Investigative Dermatology, the American Federation for Clinical Research, the American Academy of Dermatology, and the American Society for Clinical Investigation, among others. He has served as a member of the Board of Directors of The Society for Investigative Dermatology and as the President of the National Association of Veterans Affairs Dermatologists. He is currently a Deputy Editor of the Journal of Investigative Dermatology.

Dr. Robert Swerlick is an Associate Professor of Dermatology at Emory University School of Medicine and Chief of Dermatology at the Atlanta Veterans Affairs Hospital. Dr. Swerlick trained in Dermatology at the University of Virginia, completing his residency in 1983. After a brief stint at the University of Oklahoma as junior faculty, he did a fellowship in immunodermatology at the National Institutes of Health in the laboratory of Dr. Thomas Lawley. In 1988, he accepted a faculty position at Emory University in Atlanta, Georgia. Dr. Swerlick's laboratory studies the expression of pro-inflammatory molecules in dermal endothelial cells while his clinical interests focus on the diagnosis and treatment of inflammatory skin diseases, including various forms of blistering skin diseases. ●

*...continued from **PEMPHIGUS 2005**, page 1*

when pemphigus antibodies bind the keratinocyte cell surface and cause various molecules inside the cell to become activated. These activated molecules may contribute to the loss of cell adhesion that causes the pathology in pemphigus. If this view is correct, then blocking some of these signaling pathways might alleviate disease. **Yasuo Kitajima** of Gifu University (Japan) suggested a role for diaceyl glyceraol (DAG) in this process. Using a mouse model of disease, Dr. **David Rubenstein** of the University of North Carolina at Chapel Hill (USA) showed that another signaling pathway involving a molecule called p38MAPK might be involved. Blocking this pathway prevented disease in his model. Finally, Dr. **Elaine Mueller** of the University of Bern (Switzerland), showed that another signaling molecule called myc is activated in patients with pemphigus, and this activation might be important in the pathophysiology of disease.

After the break, immune mechanisms in pemphigus were discussed. The work of Dr. **John Stanley** from the University of Pennsylvania in Philadelphia (USA) on a phage display library was reported in the last issue of the Quarterly. The purpose of this work is to be able to produce essentially unlimited amounts of the monoclonal antibodies that can be characterized as pathogenic (disease causing) or non-pathogenic in cell culture or mouse models. The DNA encoding these antibodies can be sequenced to determine the antibody gene families from which the anti-Dsg antibodies are derived. This research can lead to the development of therapies that more specifically target the pathogenic antibodies than do the currently used immunosuppressive therapies that suppress the entire immune system.

Using a completely different approach, Dr. **Masayuki Amagai** of Keio University in Tokyo (Japan) generated mouse monoclonal antibodies. The monoclonal lines differed in their pathogenic capacities; some bound to mouse skin and produced microscopic disease, some caused more severe blistering, and some bound to mouse skin and did not cause any problems at all; although some individual antibodies were weakly pathogenic, mixtures of these monoclonal antibodies tended to cause more pathology than did the individual antibodies alone.

From Top: Janet Segall, Dr. Michael Hertl, Dr. Yasumo Kitajima, Dr. Masa Amagai, Dr. Luis Diaz, Dr. Eliane Mueller, and Dr. John Stanley

These two rich sets of monoclonal reagents will allow more detailed studies of the immunology of PV; they are also reagents for dissecting the events occurring after antibodies bind to keratinocytes (skin cells).

Dr. Animesh Sinha, Cornell University in New York (USA), discussed the immune process of T-cell response to desmoglein, and **Dr. Takahashi Hashimoto** (Japan) discussed the role of other protein molecules other than desmogleins, such as desmocollins, in pemphigus.

Session II's focus was on what factors are important in initiating the disease, including environmental and genetic factors. The presence of autoantibodies (antibodies that are produced against one's own cells) that bind to the epidermis (the upper layers of the skin) in PV and PF was demonstrated over 40 years ago. Although the triggers that cause this autoantibody production are rarely found, multiple studies have demonstrated that there are certain environmental factors that may explain the susceptibilities of populations in certain areas to PF.

For over a decade, Dr. **Luis Diaz** of the University of North Carolina at Chapel Hill (USA) has intensively studied a population with endemic PF in Western Brazil. Originally several individuals were disease-free, but showed evidence of antibodies to desmoglein 1 (one of the molecules that act like a glue to keep skin cells together). These particular antibodies were not causing disease. However, at some point the individuals developed a different type of antibody to desmoglein 1 that did cause disease. It is unclear exactly why that switch occurred. The origin of the antibodies found during the pre-clinical stage of the disease and this change in reactivity to Dsg1 are under investigation.

continued on page 9. . .

C-REACTIVE PROTEIN (CRP) AS AN EARLY VASCULAR DISEASE INDICATOR? ONE FAMILY'S STORY

FATHER, BROTHER VICTIMS OF HEART DISEASE

Janet Segall
IPF Director of Patient Services

I wanted to write this article because in the last few years I have learned a lot about C-Reactive Protein which could be very important to many of us.

In 1969, when I was in my early 20's, my father woke up one morning and died. This was a very devastating chapter in my life. My father and I were very close and his death shook me to my core. Unfortunately, times being what they were, we never had an autopsy ,but we all assumed he had a typical massive heart attack. Being female, I was not concerned that I would have a heart attack, but my brother did and had every heart test available. He was in his late 20's and, of course, he was fine.

Two years ago, at the age of 61, my brother, Paul, woke up one morning and died. But, with times being what they are now, we knew that what he died from was called an *aortic dissection*. The main artery from his heart to his brain tore. He wasn't in much pain, but he collapsed because the blood from his heart could not flow to his brain.

Several years prior to my brother's death, he developed ulcerative colitis. He was on prednisone and 6MP which is like Imuran. We know that in our family we have the autoimmune gene because of his colitis, my pemphigus, and several other autoimmune diseases in my family. But, we didn't know a lot about cardio-vascular disease. My mother is 85, and in great shape. She even still works part-time. I have 3 brothers (2 now). We thought we were safe. We all watched our cholesterol and HDL and LDL numbers making sure our ratio's were below 4. That is a good score - high HDL, low LDL's. My brother thought, "I'm eating right. I have good score; maybe just a little plaque I have to watch out for."

Josh and Paul Segall, (1945)

At an American Academy of Dermatology meeting, before my brother died, I was talking with a doctor who was head of a prestigious university's Dermatology Department. He had just lived through a heart attack. I started to tell him that my father had died at 56 from a massive heart attack and me and my siblings were a little nervous as we were getting older. The one thing he said that really caught my attention was, "Have your C-Reactive Protein check. That could help tell you whether you might be at risk for a vascular disease."

Many consider elevated CRP to be a positive risk factor for coronary artery disease.

I decided to do as this doctor suggested and had my C-Reactive Protein tested. I tested negative. A sigh of relief. I told my brothers to have themselves tested and both of my older brothers did. My brother Paul tested very high. Within 6 months of that test, he died.

With the last two years, I have heard more and more about C-Reactive Protein being a precursor to cardio-vascular disease. A person who has autoimmune disease and/or is taking prednisone can have a high C-Reactive Protein test because it tests inflammation. My brother said, "I'll go for a cardio test, but I have colitis and I'm on prednisone so that is why the test is high." (He was also a Ph.D. in biology so he knew his science.)

C-reactive protein (CRP) is a special type of protein produced by the liver that is only present during episodes of acute inflammation. Although the role of CRP in coronary artery disease remains unclear, it has been suggested that testing CRP levels in the blood may be a new way to assess cardiovascular disease risk. A high sensitivity test is now widely available. It is not known whether it is merely a marker of disease or whether it actually plays a role in causing atherosclerotic disease. Many consider elevated CRP to be a positive risk factor for coronary artery disease.

My brother was right in thinking that his CRP might be high only because he had autoimmune disease. But, because our father died of heart disease, doing tests to make sure his heart and arteries were okay, seemed reasonable. Unfortunately, it was too late for him.

In the May issue of Prevention Magazine, there is an article on vascular disease. Paul's heart surgeon recommended that the rest of the family have an echo cardiogram performed to test heart arteries. A recent report suggested that vascular disease is more likely in siblings then if a parent only had it. There are several centers in the U.S. that will be giving free sonograms to people potentially at risk for cardiovascular disease.

I am not trying to scare anyone, but I think that this is important. Have your CRP checked. It could just be that a high score could mean autoimmune disease, but check it out. It isn't just because of my brother. A pemphigus patient called me recently who had a high-CRP level, and caught his vascular disease in time. But I had never forgot what the doctor at the AAD meeting had told me, and I wanted to share his concerns and mine with all of you. ●

http://www.nlm.nih.gov/medlineplus/ency/article/003356.htm
http://www.americanheart.org/presenter.jhtml?identifier=4648

2005 IPF PATIENT/DOCTOR MEETING, Washington D.C., September 23 - 25, 2005

CAPITOL HILL DAY/ANNUAL MEETING REGISTRATION FORM

NAME: _____ AGE: _____

ADDRESS _____ # of Previous Conferences Attended _____

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EMAIL _____ ARRIVING _____ DEPARTING _____

*I require special assistance *(please attach a written description for all persons with special needs)* Yes ❑ No ❑

Will you be staying at the Hilton Arlington? Yes ❑ No ❑

If NO, then where will you be staying? _____

You are responsible for making your own hotel reservations by calling (703) 528-6000 or (800) HIL-TONS. Don't delay! Be sure to say you are a part of the International Pemphigus Foundation group to get your special room rate. Reservations are limited, and are on a first-come, first-served basis. Reservations must be made by **Tuesday, August 30, 2005**.

CAPITOL HILL DAY: Please make arrangements for me to meet my legislators or staff on **FRIDAY, SEPTEMBER 23, 2005**. Yes ❑ No ❑

U.S. Senators _____ and _____ ❑ Please find out for me

U.S. Representative _____ ❑ Please find out for me

REGISTRATION - EARLY REGISTRATION IS ENCOURAGED

Registration must be received by the IPF no later than **September 9, 2005**.

	# of people	Price	Total
8th ANNUAL PATIENT/DOCTOR MEETING REGISTRATION FEE		$85.00	$
Saturday Night Dinner		$40.00	$
CAPITOL HILL DAY (includes transportation to/from hotel/capitol and breakfast)		$50.00	$
Optional Scholarship Donation (help someone in need attend this year's meeting)			$
☐ I request scholarship assistance. Please accept this donation to help defray costs. *			$
GRAND TOTAL			$

*** If you are requesting scholarship assistance, please contact the IPF prior to sending in your form.**

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or fax it to us at **(916) 922-1458.** You can call us at **(916) 922-1298** or email at **pemphigus@pemphigus.org.**

Please list names and ages of ***other*** guests who are attending.

Name _____ Age _____ Special assistance? ❑

Name _____ Age _____ Special assistance? ❑

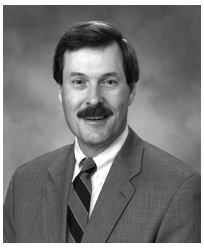
Name of person(s) with Pemphigus/Pemphigoid _____

Cancellations are not refundable after September 16, 2005.

MAYO CLINIC REPORTS ON WORK SUPPORTED BY IPF GRANT

EPIDEMIOLOGICAL RESEARCH

Mark R. Pittelkow, M.D.
Mayo Clinic, Rochester MN



The research, headed by **Dr. Mark Pittelkow**, shows that the epidemiology of pemphigus and pemphigoid has been variably investigated worldwide. Incidence rates for pemphigus range from 0.08 (Finland) to 1.6 (Jerusalem) per 100,000/yr. The only US study (CT) calculated a crude incidence of 0.42 per 100,000/yr. Incidence rates for pemphigoid are very limited, ranging from 0.6-0.9 per 100,000/yr for Germany and France, respectively. No detailed US study of pemphigoid incidence has been reported.

The Rochester Epidemiology Project (REP), utilizing the well defined population demographics and healthcare record systems of Olmsted County, MN, provides a unique resource to simultaneously determine the overall and age-adjusted incidence rates and further compare the epidemiology of these two distinct autoimmune blistering diseases. Over 5 decades (1950-2000) incident cases of pemphigus and pemphigoid were identified and confirmed by histology and immunopathology.

The overall age- and sex-adjusted incidence rates for pemphigus and pemphigoid were 0.35 and 2.4 per 100,000/yr, respectively. Whereas the rates were similar for increasing age (by decade) for pemphigus, the rates increased dramatically for pemphigoid, from <1 (40-49 yrs) to 76 (90-99 yrs) per 100,000/yr. More woman than men (1.4:1) developed pemphigus or pemphigoid. For the Olmsted County population, the overall incidence of pemphigoid was 7-fold that of pemphigus. There was no marked increase in incidence with age for pemphigus, but pemphigoid dramatically increased over 100-fold for the oldest vs. youngest age groups. Potential risk factors of development as well as mortality, associated disease, treatment and outcomes are under investigation. ●

IS THERE A NEW TREATMENT FOR CICATRICIAL PEMPHIGOID?

INITIAL RESULTS LOOK GOOD

This information is from the National Institutes of Health.

Researchers at the University of Cologne's Department of Dermatology in Germany have uncovered what could prove to be an alternative treatment for cicatricial pemphigoid. While the initial results look good, with only one person given the treatment it is far too early to tell if it will be effective or not. Consequently, doctors in the United States began giving Enbrel® to CP patinets. The results have yet to be published.

The treatment of cicatricial pemphigoid is generally regarded as difficult and usually relies on individual clinical experience. Corticosteroids, as drugs of first choice, often have to be combined with steroid-sparing agents to prevent hazardous, long-term side effects. The team in Germany describes a 72-year-old woman with long-standing cicatricial pemphigoid.

They had trouble establishing treatment regimens, but, nevertheles she responded rapidly - and more importantly lastingly, to therapy with the ***tumor necrosis factor alpha antagonist etanercept***. To our knowledge, this is the first report of its use in the treatment of a bullous autoimmune disease. ●

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www.idi.it/amb/csmba/index.htm
Philippines - Dr. Benjamin Bince
Jose Reyes Memorial Medical Center
(632) 711-6740; Email: bbin@runbox.com
Online/Website
www.pemphigus.org/support.html

In France, the Association Pemphigus France is a newly formed support organization providing information and support for people living with pemphigus, their families, and caregivers. Please contact Peter Foldes: peterfoldes@tiscali.fr (01 47 32 42 05); Josée de Felice: felice@paris7.jussieu.fr (01 60 72 18 73), or Isabelle Gentile: ifgentile@free.fr (02 23 96 39 21)

In the UK, the PV Network is a patient support group providing information and support for people living with pemphigus, their families and caregivers. For information and support call 020-8690-6462 or send a self-addressed, stamped envelope to:
PV Network
Flat C, 26 St. Germans Rd., SE23 1RJ
www.pemphigus.org.uk

PEMPHIGUS EUROPEAN MEETING 2005

ExCel London International Exhibition Center

October 15, 2005, Noon until 8:00 p.m. *(or sooner)*

Connect with others!

Exchange Ideas!

Joining us will be:

Dr. Karen Harman, *UK*
Dr. Dedee Murrell, *Australia*
Dr. Daniel Mimouni, *Israel*
Dr. Michael Hertl, *Germany*
Dr. Marcel Jonkman, *Netherlands*
Dr. Sergei Grando, *USA*
...and others...

The **International Pemphigus Foundation** invites you to join us on October 15, 2005 at the ExCeL London International Exhibition Centre *(London Docklands, London E16 1XL, United Kingdom)* when some of Europe's leading physicians discuss pemphigus and answer questions from patients and caregivers.

We will meet at noon and go until we have finished, or 8:00 pm whichever comes first. Don't miss this exciting opportunity as we gather to open doors and break down myths about pemphigus, its treatment, and the future directions we are heading.

Complete Event Costs: **$75.00USD**
Registration Fee: **$35.00USD**
Dinner (per person): **$40.00USD**
Approximately €62 or £42 for the complete event
(meeting: €29/£20 and dinner: €33/£22)

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*I require special assistance *(please attach a written description for all persons with special needs)* Yes ❑ No ❑

Everyone is encouraged to attend! We look forward to seeing you there!

REGISTRATION - EARLY REGISTRATION IS ENCOURAGED

Registration must be received by the IPF no later than **September 15, 2005.**

	# of people	Price	Total
EUROPEAN MEETING REGISTRATION FEE		$35.00, €29, £20	$
Dinner		$40.00, €33, £22	$
Optional Scholarship Donation (help someone in need attend this year's meeting)			$
❑ I request scholarship assistance. Please accept this donation to help defray costs.			$
GRAND TOTAL			$

Check ❑ **Money Order ❑** **Visa ❑** **Master Card ❑**

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*...continued from **PEMPHIGUS 2005**, page 4*

The role of environmental factors associated with endemic PF seen in Tunisia was reported in the poster session. **Dr. Sarah Brenner** (Israel) led a spirited discussion on the role of drugs, especially sulfhydryl-containing drugs like penicillamine on pemphigus acantholysis, and she discussed the possibility of pesticides and other environmental factors such as stress, hormones, and UV radiation as possible factors in the disease process.

Dr. Michele Mignogna (Italy) presented the possibility of drugs such as Captopril being triggers for pemphigus. The consensus was that drugs may be triggering agents but that the relative importance of the effect of the drug on the immune system as opposed to directly on the epidermis is unclear.

Dr. Don Siegel, University of Pennsylvania, Philadelphia talked about the relationship between pemphigus and another autoimmune disease, idiopathic thrombocytopenic purpura (ITP), in which antibodies are produced against platelets. Platelets play a crucial part in the blood clotting process. The lessons learned and the tools being used in the creation of novel therapies for diseases such as ITP, can now be applied toward developing new drugs for pemphigus as well.

Dr. Mark Pittelkow, Mayo Clinic, reported on his ongoing study on the epidemiology of pemphigus vulgaris and bullous pemphigoid. The IPF helped fund this study with a generous donation to our reseach fund, and its current progress is detailed on page 7.

Dr. Grant Anhalt of Johns Hopkins University in Baltimore, Maryland (USA) presented his extensive experience with paraneoplastic pemphigus, which is associated with malignancies, usually lymphomas, thymic tumors or Castleman's disease. Dr. Anhalt found that PNP patients developed autoantibodies to not only desmoglein 3 but also other molecules associated with adhesion structures in the epidermis.

Corticosteroids have been the mainstay of therapy for PV for over fifty years, however the potentially severe adverse consequences of long-term high dose steroid use has led to the use of immunosuppressives as adjunctive medications. The paucity of evidence-based studies in PV and the challenges inherent in performing them was presented by Dr. **Marcel Jonkman** of the University of Groningen (The Netherlands), who performed a double-blind clinical trial that did not show pulse oral dexamethasone to be useful.

New studies with agents to reduce steroid dosage, such as dapsone and mycophenolate, were presented by Dr. **Victoria Werth** of the University of Pennsylvania in Philadelphia (USA) and Dr. **Daniel Mimouni** of Rabin Medical Center in Petah-Tiqva (Israel), respectively.

There was interest in the experience of Dr. Jean-Claude Bystryn of New York University in New York City (USA) and Dr. A. **Razzaque Ahmed** of Harvard School of Dental Medicine in Boston, Massachusetts (USA), who used IVIg for the therapy of pemphigus. IVIg was often effective in difficult patients who did not respond to other agents or as a way to reduce systemic steroid use. The mechanism(s) of IVIg effectiveness in PV and PF is not fully understood but is being investigated (see below).

While most studies report that PF and PV are related to antibodies against Dsg1 and Dsg3, respectively, other studies, such as those reported by Dr. **Sergei Grando** of the University of California-Davis Medical Center in Sacramento California (USA), implicate other membrane proteins (e.g., cholinergic receptors). Dr. Grando also reported that certain drugs used to treat myasthenia gravis, such as pyridostigmine bromide (Mestinon) might be useful in the therapy of pemphigus vulgaris. However, almost all investigators focused on antibodies to Dsg1 and Dsg3 and their consequences on cell and tissue function. Blood from patients showing autoantibodies to Dsg1 and/or Dsg3 were commonly employed for studies. Two groups reported new monoclonal (single cell, identically genetic) antibodies to Dsg1 and Dsg3, as discussed above.

Zhi Liu of the University of North Carolina at Chapel Hill (USA) presented data using the neonatal mouse pemphigus model. His studies suggested that IVIg was associated with the breakdown of the anti-Dsg1 or anti-Dsg3 antibodies.

Dr. Grant Anhalt of Johns Hopkins University in Baltimore, Maryland (USA) presented the results of a Phase I dose-escalation safety trial in a group of patients treated with a 19 amino acid peptide derived from a Dsg3 epitope recognized by T cells from PV patients. The long-term aim of this treatment is to induce tolerance in pemphigus by decreasing autoreactive T cells in the patients. The peptide does not induce flares of the disease, and further testing in a trial will go forward.

The high cost of drugs (e.g., mycophenolate) and the ultra-high cost of IVIg led to discussions of the need for controlled studies of the treatments of PV. To accomplish this goal, an international committee was formed, chaired by **Victoria Werth** of the University of Pennsylvania in Philadelphia (USA). The committee is charged with developing standard nomenclature for patient diagnosis and the results of pemphigus trials, so the description of their results can be compared.

The complete program is available online www.pemphigus.org/2005Meeting.htm, and abstracts from the meeting will be published October 2005 Issue of the The Journal of Investigative Dermatology.

We would like to thank **Dr. Lowell Goldsmith**, University of North Carolina at Chapel Hill (USA) for writing a summary of the meeting. ●

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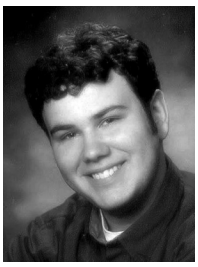

Todd, 24 now, has suffered with PV since he turned 16 when he suffered from an outbreak of painful, ugly lesions on his gums and inner cheeks. His parents took him to 21 doctors – dermatologists, dentists, oral surgeons, immunologists, et al - from coast to coast, over the period of about a year.

It was finally diagnosed correctly in his hometown of Louisville, KY, by Dr. Jeffrey Callen. Over the next seven years, Todd was on Imuran® and prednisone daily. This past year Todd decided on his own to completely drop the Imuran® and use only prednisone -- and that is when, and if, he experiences bad outbreaks. Fortunately, those have been few and far between.

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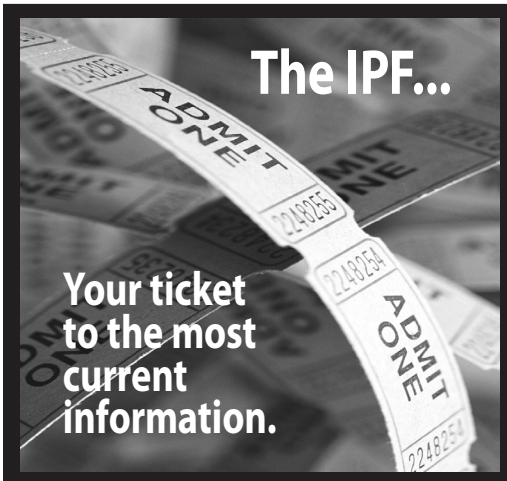

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