RESEARCH AND CLINICAL HIGHLIGHTS OF THE 4th INTERNATIONAL WORKSHOP ON WM, part 2
by Guy Sherwood, M.D.

Part 1, printed in the Fall Torch 2007, covered the papers reporting on the genetics, pathophysiology, and staging system of WM.

In this second article devoted to the 4th International Workshop on Waldenström’s Macroglobulinemia held in June on the island of Kos, Greece, Dr. Guy Sherwood, IWMF Trustee, provides concise summaries of the oral presentations addressing the frontline treatment of WM, novel treatment approaches, and treatment of advanced disease. The final section, Dr. Sherwood’s review of the 30 research posters presented at the Workshop, will appear in the spring 2008 edition of the Torch.

The 4th International Workshop was organized by Dr. Meletios A. Dimopoulos of the Department of Clinical Therapeutics, the Alexandra Hospital, and the Medical School, National and Kapodistrian University of Athens. The presenters represent 8 countries. Whether speaking at the podium or displaying a poster, they are in the forefront of those international researchers who are today rising to the challenge of advancing our understanding of WM in the pursuit of better therapies and a cure. The topics of their presentations present an impressive list of advances made in clinics and laboratories in different corners of the world.

Frontline treatment of WM

IWMF Trustee Dr. Robert A. Kyle (Mayo Clinic, Rochester, MN) led the oral sessions with a review of the “Indications for treatment and the role of alkylating agents in WM.” Dr. Kyle, the world authority in treating plasma cell disorders with alkylating agents (primarily chlorambucil), reminded those present that these agents still have a very favorable and important role to play in the treatment of WM. Dr. Kyle did, however, note a recent paper by Dr. Dimopoulos in 2005 suggesting that prolonged treatment with alkylating agents increases the possibility of myelodysplasia or acute leukemia.

SAVE THE DATE!

The annual IWMF Educational Forum will be held May 16-18, 2008, in Los Angeles CA.

For details concerning registration see the brochure included with this issue of the Torch or the IWMF website (http://www.iwmf.com/Calendar.htm).

Our committee members and many volunteers are excited about this upcoming event and are dedicated to staging another successful Ed Forum in the tradition of those past. We have an outstanding line-up of speakers, a great selection of breakout sessions, and many opportunities for socializing among our WM friends.

We will continue to expand exposure to scientific research by making available articles and posters dealing with WM. (A poster is a printed summary of a research project or a clinical trial; posters are displayed at major medical conferences by doctors and scientists in place of an actual oral presentation.)

An informal Saturday evening poster viewing with cash bar and light snacks will enable Ed Forum participants to mix and mingle. Several of the authors of the posters on display will be at the reception to answer questions about their work.

Register early (the Ed Forums sell out very quickly) and take advantage of affordable airfares. A word of advice: arrive early and spend a few days before or after the event visiting the many interesting attractions that Los Angeles has to offer.

See you there!

The 2008 Educational Forum Committee
Better Neutrophil Activity May Increase Rituximab Effectiveness – A study from the Roswell Park Cancer Institute in Buffalo, NY, has characterized the effect of neutrophils on anti-tumor activity of rituximab. Using mouse models, the researchers discovered that mice with intact neutrophil function responded better to rituximab and had longer mean survival times. They concluded that strategies to improve neutrophil function, such as granulocyte colony-stimulating factor (G-CSF), may increase the effectiveness of rituximab.

New Compound Activates Protein to Control Immune Cell Growth – Scientists at the Vancouver Coastal Health Research Institute and the University of British Columbia have discovered a protein called SHIP, which acts as a control switch for the PI3 kinase pathway essential for growth and survival of immune cells. Persistent activation of this pathway can result in serious immune diseases or blood cancers such as leukemia, lymphoma, and multiple myeloma. These scientists have also identified a compound known as AQX-MN100 that is able to activate the SHIP protein and thus slow down runaway growth of immune cells.

Technology Detects Antigens in Multiple Myeloma Paraproteins – The Boston University School of Medicine has described a new technology called Epitope-Mediated Antigen Prediction (E-MAP) for use in determination of antigens found in the paraproteins of several multiple myeloma patients. The researchers re-constructed antigens found on the myeloma paraproteins and identified human cytomegalovirus as a possible initiating event for development of the malignant clones in these particular patients.

FDA Act Improves Communications on Clinical Trials – In September, President Bush signed into law the Food and Drug Administration Revitalization Act which aims to improve the FDA’s ability to ensure the safety of the nation’s drugs and medical devices. It specifically requires sponsors of clinical trials to register their studies in a public database so that information about the conduct of the trials and their principal results must be made public.

Phase 3 Results to Be Released on BiovaxID Cancer Vaccine – Accentia Biopharmaceuticals will announce results of the fast-tracked Phase 3 clinical trial of BiovaxID, its anti-cancer vaccine for treatment of follicular lymphoma.
CD MARKERS AND LYMPHOMA

by Sue Herms

Not too many years ago, most lymphomas were diagnosed by using a few simple stains and looking at the appearance of cells under the microscope. But there were only a few types of lymphoma that could easily be distinguished in this way, and it often appeared that tumors of the same cell type behaved very differently in different individuals. It was obvious that something else was going on at the molecular level to account for these differences.

As medical diagnosis progressed to the study of molecules, new techniques such as flow cytometry were devised to identify specific molecules, located on the surface of white blood cells. Such molecules are referred to as cluster of differentiation (CD) markers. The system of CD classification was established in 1982; while originally intended only for white blood cell markers, its use has expanded to many other cell types. At last count, over 350 CD markers have been identified. It is now understood that each cell type has its own distinct markers that can serve as “identification tags.” Many of these CD markers have helped physicians to understand why there is so much variation among the different types of lymphoma.

Antibodies have been developed that are specific for each CD surface marker. These antibodies can be tagged with fluorescent stains and allowed to combine with their corresponding markers on the cells. If a particular CD marker is present, the CD-antibody combination will fluoresce; if not, the antibody will not attach and there will be no fluorescence. The presence or absence of fluorescence allows cells to be defined with a + or – symbol to indicate whether they express (that is, exhibit) or lack a particular CD marker. For example, a CD20+, CD5- cell is one that expresses CD20 but does not express CD5. The degree of fluorescence is also important in designating some cells as exhibiting strong expression of markers and others as exhibiting weak expression. It should be noted that, while we commonly think of CD molecules as markers or “identification tags” for specific types of cells, they do function in numerous ways that are important to a cell’s maturation and survival. These functions include acting as receptors for various substances or serving as signals to initiate or alter cell behavior.

As both B- and T-lymphocytes mature, they go through several stages of development. During these stages, lymphocytes will acquire new CD markers, while other CD markers will diminish in their expression or be lost altogether. But if a lymphocyte mutates and forms a clone of lymphoma cells, the mutated cells will express the same CD markers (indicating cell type and stage of development) as the normal cell from which they mutated. When the pathologist makes a lymphoma diagnosis, he first determines the CD pattern of a particular patient’s cells and then compares it with known patterns of cell populations for a whole range of different types of lymphoma. It is the variation in CD expression found among different lymphomas that assists the pathologist in making a lymphoma diagnosis.

For example, the typical CD pattern for the majority of WM clonal cells is CD5-, CD10-, CD19+, CD20+, CD23-, CD38-. Although this is the norm, there can be slight variations in the WM pattern as well as in the CD patterns of other B-cell lymphomas. Therefore, a whole range of test results, such as cell morphology, IgM expression, etc., must be considered in the diagnosis.

In another example, the typical pattern for B-cell chronic lymphocytic leukemia (B-cell CLL), is CD5-, CD10-, CD19+, CD20+, CD23+, CD38-. Notice that the major CD differences between the two are usually CD5 and CD23 expression. Also, whereas WM has very strong expression of CD20, B-cell CLL typically shows weaker expression of this particular marker.

Multiple myeloma is generally CD19-, CD20-, CD38+. This occurs because, as B-cells mature into plasma cells, they lose the CD19 and CD20 markers and begin to strongly express the CD38 marker. Consequently, these markers are frequently used in the differential diagnosis of certain B-cell lymphomas vs. multiple myeloma.

T-cell lymphocytes have their own unique CD markers, such as CD3, CD4, and CD8. CD markers are not only important in the diagnosis of lymphoma—they are also useful for predicting the types of treatment that might be successful and for monitoring the effectiveness of treatment. The fact that the CD20 marker is positive for almost all non-Hodgkin’s B-cell lymphomas makes it a good target for the use of rituximab, which is a well-known anti-CD20 antibody treatment. It has been speculated that rituximab treatment is more effective in lymphomas with stronger CD20 expression than in those with weaker expression. Rituximab is not generally used for multiple myeloma because these cells typically no longer express CD20. Interestingly, while CD20 is a very important one, not much is known about its activity in the B-cell. It is suspected to act as a calcium channel in the cell membrane.

The use of CD markers has been a revolution in the diagnosis and treatment of various cancers, including lymphoma. Stem cell transplantation relies on detection of the CD34 marker present on harvested stem cells to determine how successful the harvesting process has been. New CD markers are being discovered, and new antibody therapies against various CD targets are being developed in the hope of improving cancer outcomes.

The author holds degrees in Zoology and in Medical Technology from Ohio University and from the Medical University of South Carolina, respectively. She is currently employed as a medical microbiologist at Roper Hospital in Charleston, South Carolina.

Sue Herms welcomes ideas for future articles from Torch readers. Please forward your ideas to the Torch or send an e-mail with your suggestions to suenchas@bellsouth.net
Dear Friends,

By the time you read this letter, the IWMF's membership and research fundraising campaigns will be well under way. This edition of the Torch contains information on the fund drives for our Research Fund and for our Operating Fund which supports our small office in Sarasota and our many member services.

It occurs to me, based on recent communications with some of you, that the use of funds contributed by members to the IWMF should be clarified to illustrate the frugal management of such funds which keep the IWMF in existence and moving forward.

We have two separate funds and therefore two separate accounts. The Operating Fund pays for the overhead costs of our headquarters in Sarasota, FL, and the wages paid to our full-time office manager, a part-time bookkeeper, and temporary secretarial assistance when needed. It also funds the costs for member services and significant related items such as postage, printing, and supplies. Our basic overhead costs have always been kept quite low, amounting to 10.5% of our annual expenses, and have remained at that level since we organized formally as a private non-profit organization in 1998. The Research Fund is, of course, the larger fund, and these monies are spent solely on direct research.

Those who serve on the IWMF Board of Trustees, including the officers, are volunteers. When accepting a Board appointment, Trustees are asked to commit to serving actively on at least one committee or project. Officers and Committee Chairs spend an average of 20-30 hours per week on their responsibilities. [Please see the list of trustees and committees on which they serve following this article] In addition, there are many members with valuable skills serving on IWMF committees and various project teams, and they are volunteers as well. The only costs associated with those who devote their time and skills to the work of the foundation are travel and hotel expenses, which are limited by our IWMF expense reimbursement policy. When the IWMF was formed there were two important basic tenets that were sworn to: 1) that our information about Waldenstrom's macroglobulinemia would always be free to WM patients; and 2) that travel and hotel expenses would be reimbursed to Board members so that good people, regardless of their personal finances, could become members of the Board.

We limit our meetings of the Board of Trustees to four a year, with one held at the conclusion of the Educational Forum. The face-to-face meetings are important for discussions and decisions on a wide variety of matters and for directing committee work. In between these scheduled meetings we use conference calls and emails for communicating. International travel is limited. Every other year the International Waldenstrom Macroglobulinemia Researcher Workgroup meets, and the location of this four-day meeting changes each time, as determined by the researchers involved. Dr. Steven Treon and Dr. Eva Kimby of the Carolinska Institute in Stockholm are organizing the next meeting. They will solicit funding from pharmaceutical companies for the major costs and from the IWMF for minor expenses.

At the international workshop the IWMF Board is always represented by the president, vice-president for research, and one of the physician/trustees to assist later in the write-up of this meeting. At these workshops we have been very successful in meeting and encouraging researchers, even suggesting, when appropriate, that they apply to the IWMF for funds to continue the studies they report on. We have supported a number of fellowships for young investigators, selected by senior researchers, to attend their first Waldenstrom’s Macroglobulinemia workshop. Most of these young investigators are now, in fact, working in laboratories on WM projects as interns or fellows at various international cancer institutes. We believe this is a very good way to encourage their continued interest in this rare disease when we look down the road at the future need for young researchers to replace those who retire or move to other diseases.

Most of you have heard of Steve Kirsch, the Kirsch Foundation, and his intention to fund WM research. I have just learned that, after extensive personal review, Mr. Kirsch has decided to establish his own Waldenstrom's Research Program and will be developing a Scientific Advisory Board to guide the contribution of funds and the selection of projects. I am happy to report that IWMF has just been named to receive $100,000 for our Research Fund. We are very pleased to see someone with significant resources put money into WM research since, no matter the vehicle used, we all stand to benefit. Bravo Steve!
The vaccine has demonstrated substantial clinical benefit measured in terms of additional years of disease-free survival and, in some cases, long-term complete remissions of over nine years’ duration. The company hopes to gain accelerated conditional approval in 2008 for use in the U.S. and Europe. The vaccine is designed to stimulate the patient’s own immune system to recognize and destroy cancerous B-cells that remain in the body after chemotherapy.

Further News on Velcade Use in United Kingdom – In the United Kingdom, the National Institute for Health and Clinical Excellence has partially reversed its ban on the use of Velcade for cancer treatment. Under the new proposal, patients will be allowed to have the drug the first time they relapse from their initial treatment. Patients who then benefit from Velcade will continue to get the drug fully funded, while those who show minimal or no response will be taken off the drug and the costs refunded by the manufacturer.

Proposed Decrease in Medicare and Medicaid Reimbursements for Bexxar and Zevalin – The Centers for Medicare and Medicaid Services are proposing to reimburse less than half the cost of treatment with the radioimmunotherapy agents Bexxar and Zevalin. Both agents have shown a high degree of effectiveness against certain lymphomas. The concern is that hospitals will be forced to subsidize the use of these drugs and may opt to discontinue them because of the added expense.

Cord Blood Product Used in Adult Lymphoma Transplant Patient - Gamida Cell announced the use of its new cord blood stem cell product on an adult lymphoma patient. The product, called StemEx, has been developed for use by patients who could benefit from an allogeneic marrow transplant but who cannot be matched with a donor. Umbilical cord blood has fewer donor matching requirements, but because cord blood has a limited number of stem cells, it has previously proven unsuitable for adult transplant patients. StemEx employs a technology that expands the small number of stem cells in cord blood.

New Drug Targets Protein Found in Normal B-Cells and Lymphoma Cells – Doctors at the University of Rochester’s James P. Wilmot Cancer Center are studying a new investigative drug called R788 that targets a common protein, SYK, found in normal B-cells and lymphoma cells. The new therapy is in twice-a-day pill form and offers patients with recurrent disease another choice as their treatment options decrease.

Novel Therapy Centers on BCL2 Gene – ProNAi Therapeutics is pioneering a new oncology drug candidate, PNT2258, which centers on the BCL2 gene’s role in triggering death in cancer cells. In preclinical mouse studies, the drug has demonstrated anti-tumor activity in non-Hodgkin’s lymphoma, prostate cancer, and melanoma.

Impact of PAX5 Protein on Lymphoma Growth – University of Pennsylvania researchers have discovered the process by which a protein called PAX5 stimulates the growth of lymphomas. PAX5 locks the B-cell division switch in the “on” position so that it keeps multiplying, regardless of antigen exposure.

Phase 3 Trial Results of TREANDA Announced – Cephalon, Inc., announced positive results from a Phase 3 clinical trial of TREANDA (bendamustine HCI) in patients with indolent non-Hodgkin’s lymphoma whose cancer is no longer responsive to rituximab. Single-agent TREANDA was evaluated in 100 patients with relapsed, rituximab-refractory NHL. The overall response rate was 75%. The most common side effects were nausea, fatigue, neutropenia, diarrhea, and vomiting. TREANDA is a designed nucleoside purine analog/alkylator hybrid drug. While it prohibits cells from dividing to create new cells, it also damages the DNA.

New Studies for Aranesp, Epogen, and Procrit Underway – Amgen and Johnson & Johnson are planning new studies for Aranesp, Epogen, and Procrit to evaluate risks associated with using these agents to treat anemia in patients with cancer. Four of these studies will involve patients with lymphoid malignancies. The studies are in response to recent rulings by the FDA to limit the use of these drugs in patients with anemia.

Phase 1 Trial for New NHL Therapy Announced – Therapeutics, Inc., has announced the initiation of patient enrollment in a Phase 1 study of its agent RH1 in patients with advanced solid tumors or non-Hodgkin’s lymphoma. RH1 is a small molecule agent activated by the enzyme DTD, which is over-expressed in many tumors. The patients will receive starting doses of a 3-hour intravenous infusion once every 21 days, with dose escalation based on toxicity.

Seattle Genetics Reports on Two New Drug Therapies for Lymphoma – Seattle Genetics has reported preclinical data on SGN-40, a humanized monoclonal antibody that is currently in clinical trials for non-Hodgkin’s lymphoma and multiple myeloma. SGN-40 signals cell death in models of NHL by depleting an important survival signal (BCL-6) for cancer cells and also increases the level of another protein (Tap63a) thought to enhance sensitivity of tumor cells to chemotherapy. Researchers also presented preclinical data on the company’s anti-CD19 antibody-drug conjugate. CD19 is a B-cell marker expressed in many hematological malignancies. Both drugs will be proceeding to clinical trials.

The author gratefully acknowledges the efforts of Howard Prestwich, Bert Visheau, Mike Dewhirst, and Gareth Evans in disseminating news of interest to the WM community.
GOOD NEWS ON THE LASTING LEGACY TO IWMF: THE BEN RUDE HERITAGE SOCIETY

by Dick Weiland

At the December meeting of the IWMF trustees the Board approved policies and procedures for a recognition society of planned giving donors. The official title of this special organization shall be the Ben Rude Heritage Society in honor of Ben Rude, a very early leader of IWMF. Ben’s spouse, Laurie Rude, will serve as the founding honorary chairperson of our Ben Rude Heritage Society.

A corresponding group designed to recognize major IWMF benefactors was also approved by the Board. This entity will be called the Arnold Smokler Circle in tribute to the IWMF founder. We will report more on this Circle in the next edition of the Torch.

In the meantime, if you intend to include the IWMF in your estate planning – or if you know of someone else with this intention – please complete the Inquiry Form below and return it in the enclosed envelope before March 1. Either Dick Weiland (507.645.2633) or Dave Lively will be in touch shortly thereafter and will respond to any questions or concerns you may have. We will also ask permission to recognize you at the Saturday luncheon at the Educational Forum in Los Angeles this spring. Of course, if you prefer to be an anonymous IWMF benefactor, your intentions will be respected. Also note that there are certain restrictions on some planned giving vehicles set by various states and by the IWMF. So be sure to see your attorney or tax adviser as you put your plans on paper.

IWMF RESEARCH GRANT UPDATE:

LINDA M. PILARSKI

edited by Guy Sherwood, M.D.

The following article was written specifically for the Torch by Dr. Pilarski, world-recognized expert in the field of the genetic characteristics of WM. Working at her laboratory at the Cross Cancer Institute, University of Alberta, Edmonton, Canada, her team of talented researchers is elucidating the multiple genetic events that lead to the eventual formation of the malignant WM B-cell. In May 2005 Dr. Pilarski was awarded a three-year research grant from the IWMF for her research proposal “Genetic Characteristics of Waldenström’s Macroglobulinemia”.

Dr. Pilarski and her colleagues are analyzing the genetic contributions to WM. They previously found that a highly abnormal protein is made in WM cells but not in their normal counterparts. This abnormal protein also occurs in other types of cancers where it predicts poor survival, probably by contributing to the emergence of aggressive cancer cells as the disease progresses. The protein abnormality arises during the processing of genetic information that creates a template to direct protein synthesis. Creating the template involves specific cutting and pasting of gene segments. In WM, this cutting and pasting occurs “incorrectly”, leading to an abnormal set of templates and an abnormal set of proteins.

In WM, Dr. Pilarski’s team has found a set of genetic mutations in the hyaluronan synthase 1 (HAS1) gene that dramatically alters the positions where gene segments are cut and pasted during the assembly process termed “splicing”. HAS1 is an enzyme that makes a large molecule, a sugar polymer, known to be important in many types of cancer. Dr. Pilarski and her colleagues have studied inherited polymorphisms in the HAS1 gene. Polymorphisms are genetic changes that occur in some
Good news: the DVDs from the IWMF 2007 Educational Forum in Atlanta have been shipped. Better late than never! The DVDs should already be in the hands of those of you who have paid in advance. If you still wish to place an order at $35 a set, please contact the IWMF office. For our readers in Europe, please note that we are now offering the DVDs in the European PAL video format at the same price.

We need to give a big round of applause and thanks to our member Ron Draftz who not only was our videographer—along with Michael Luttrell and Mike Cooper—but also edited and produced our 2007 Educational Forum DVDs. We thank Ron doubly for rescuing the DVD footage from the dreaded computer hard drive failure that nearly sank our 2007 Ed Forum production.

By now you will have received a request from the IWMF to renew your membership. In addition to the suggested membership contribution we hope you will consider adding a few extra dollars to help offset our expenses. Like every other organization we are affected by increased postage and other expenditures essential to the services we provide. Even a small additional contribution certainly helps our cause. Of course, no one is ever turned away from membership in the IWMF because of a lack of funds.

One way to help us economize is to receive your Torch on-line. Just inform the IWMF business office that you would like to make the switch. What an easy way to make an extra contribution to the Foundation!

Details of the 2008 Los Angeles Ed Forum to be held May 16-18 in Los Angeles can be viewed on our website, and it is now possible to register electronically and make your hotel reservations directly at the website. The Forum committee is planning exciting programs, discussions, and social gatherings. As you have come to expect, we have an all-star line up of WM doctors who will bring us word of new developments and treatments and will discuss what the future holds in our search for the cure.

Space is limited so please register and make your hotel reservations as soon as possible. Our Ed Forum will surely sell out once again, so do not delay.

This is the first column that I am writing as the Vice President of Administration, a position so ably handled in the past by fellow Trustee and Treasurer, Jim Bunton. Jim will continue to provide good advice and counsel to us all and especially to the ‘rookie’ writing this column.
The effectiveness of single agent rituximab in the primary therapy of WM, concluded Dr. Weber, has been demonstrated in many clinical trials. The addition of rituximab to combinations of chemotherapeutics/novel agents results in high response rates and durable remissions, even after limited therapy. Rituxan is particularly useful in the case of low-level bone marrow infiltration and of projected stem cell collection.

M.A. Gertz (Mayo Clinic, Rochester, MN) presented “Overview in salvage treatment in WM.” Dr. Gertz began with the observation that in the recent past the options for the treatment of relapsed disease included the re-use of a front-line agent, if successful initially, or another front-line agent used as a single agent. More recent options include combination chemotherapy, thalidomide with or without steroids, autologous transplantation, and alemtuzumab (Campath). Dr. Gertz also noted that combination therapies have demonstrated an efficacy as good as, perhaps even better than, single-agent therapies and therefore are at present used more frequently. After reviewing the promising newer agents in WM, Dr. Gertz concluded his talk by noting that “alkylators plus purine nucleoside analogs, nucleoside analogs with rituximab, thalidomide, and stem cell transplantation are all options to be considered in the complex management of patients with WM.”

A. Anagnostopoulos (Department of Clinical Therapeutics, University School of Medicine, Athens, Greece) discussed “The role of autologous transplantation in WM.” At the outset Dr. Anagnostopoulos emphasized that the published experience with high-dose treatment supported by autologous stem cell transplantation (ASCT) for WM consists mostly of retrospective reports from single institutions with small numbers of patients. He added that the fact that only a small number of WM patients have undergone autologous transplants reflects the advanced age of the patients at the time of diagnosis and the relatively indolent nature of the disease, making an aggressive treatment option such as ASCT less attractive to patients and clinicians alike. However, all studies confirm that ASCT is feasible in WM patients, even those who have been heavily pretreated. A non-relapse mortality rate of 6% is noted in the largest series of patients.

Dr. Anagnostopoulos discussed certain practicalities regarding ASCT. The harvest of stem cells should be considered prior to nucleoside analogue exposure; fludarabine in particular is shown to impair the ability to collect stem cells. Harvesting should also be done before extensive bone marrow infiltration occurs. Dr. Anagnostopoulos concluded that ASCT as upfront treatment for WM should be considered only for younger patients with adverse prognostic factors and only within the context of clinical trials. As salvage regimen, however, ASCT should be considered for all fit patients with advanced disease when conventional treatment options have failed.

S.P. Treon (Dana-Farber Cancer Institute, Harvard University) spoke on “Novel agents in the treatment of WM.” Dr. Treon began his presentation on a sobering note: “Despite advances in therapy, WM remains an incurable B-cell disorder with elusive complete remissions (8-10%).” Therefore, novel therapeutic agents and combination strategies are needed, particularly if curative efforts are to be pursued. As such, we have prioritized the development of novel and stem-cell sparing agents in the treatment of WM.

Dr. Treon’s review of novel agents included the following points:

- Bortezomib (Velcade) is a proteasome inhibitor which induces cell death (apoptosis) of WM cells.
- Velcade may also play an important role in modifying the bone marrow micro-environmental support for lymphoplasmacytic cells, making the bone marrow less hospitable for WM cells. Velcade is also a stem cell sparing agent.
- Alemtuzumab (Campath) is a humanized monoclonal antibody targeting CD52, which is widely expressed on bone marrow WM cells and is also present on mast cells.
- Mast cells are increased in the bone marrow of WM patients and provide growth and survival signals to WM cells. The increased number of these mast cells stimulates WM cell growth through several molecular signaling pathways including CD40L, APRIL and BLYS. Inhibition of these signaling pathways may be helpful in the therapy of WM.
- One of the important growth and survival factors for mast cells is stem cell factor (SCF), which signals through CD117. Imatinibmesylate (Gleevec) blocks SCF signaling through CD117, and induces cell death of bone marrow WM cells and mast cells, both of which strongly express CD117.
- Sildenafil citrate (Viagra) works as a phosphodiesterase inhibitor; inhibition of phosphodiesterase leads to apoptosis of malignant lymphoma cells. Viagra induces the death of primary tumor cells in patients with WM and chronic lymphocytic leukemia (CLL).

The concluding words of Dr. Treon sounded a positive note, underscoring the fact that basic scientific research in the biological understanding of WM is yielding newer and more targeted therapies for the treatment of WM and that continued support for basic and clinical research, as well as the establishment of clinical trials, will lead to the continued development of novel agents for the treatment of WM.

**Novel treatment approaches, treatment of advanced disease**

I. Ghobrial (Dana-Farber Cancer Institute, Harvard University) spoke on “Proteomic analysis in WM.” Her research uses antibody-based protein microarrays to compare patterns of protein expression between untreated WM
and normal bone marrow controls in order to understand
the molecular changes that occur in WM. The antibody
microarray detects proteins involved in signal transduction,
cell-cycle regulation, gene transcription, and apoptosis.
Comparing WM cells to control cells, the microarrays
identified many over-expressed proteins in at least 60% of
the WM samples. Once identified, the molecular pathways
that mediate proliferation and resistance to apoptosis in WM
are targets for novel therapies. By reporting the identity
of novel proteins over-expressed in WM, Dr. Ghibrial’s
research enhances our understanding of disease pathogenesis
and reveals targets for new drugs.

N.C. Gutierrez (Hospital Universitario de Salamanca,
Spain) spoke on “The gene expression signature of clonal
cells from WM: differences and commonalities with the
normal cell counterpart and other related lymphoproliferative
disorders.” Reporting on the results of his research, Dr.
Gutierrez indicated that the genes discriminating the WM
plasma cells from MM cells are those involved in plasma cell
differentiation: PAX5 (over-expressed in WM); and IRF4
and BLIMP1 (under-expressed in WM). The IL10 receptor
gene is one of the more important over-expressed genes in
both WM and CLL. Dr. Gutierrez concluded that the distinct
transcription signature of WM is genetically different from
its MM and CLL cell counterparts. These distinct genes are
implicated in B-cell differentiation and oncogenesis.

E. Hatjiharissi (Dana-Farber Cancer Institute, Harvard
University) addressed the topic of “Gene expression
profiling of WM reveals genes that may be related to disease
pathogenesis.” Gene expression profiling of WM bone
marrow was performed, reported Dr. Hatjiharissi, in order
to determine the molecular biological events implicated in
the pathogenesis, progression and clinical outcome of WM.
Of interest are the two distinguishable tumor populations:
B-lymphocytes (CD19+) and plasma cells (CD138+).
The presence of abnormally regulated genes demonstrates
important differences between WM B-cells and WM plasma
cells, Dr. Hatjiharissi concluded.

In a subsequent lecture entitled “Novel splice variant transcript
of Siva in WM,” Dr. E. Hatjiharissi continued her discussion
of WM genes. Focusing on the transmembrane protein CD27
(a member of the tumor necrosis factor receptor family that
binds to its ligand CD70) she demonstrated that CD27-CD70
interaction supports growth and survival of WM cells. High
levels of soluble CD27 (sCD27) in the serum of patients with
WM were also noted. Variation of certain genes in WM cells
may interfere with the CD27-CD70 signaling pathway in WM.

S. Adamia, (Dana-Farber Cancer Institute, Harvard
University, and formerly with Dr. Linda Pilarski at the
University of Alberta) continued the series of lectures on
genetics with “Aberrant post-transcriptional regulation of
TNF family members and their adaptor molecules essential to
B-cell growth and survival in WM.” Noting that the genetic
basis for WM remains unclear despite the explosion of
genetic studies in WM, she explained the integral role played
by the tumor necrosis factor (TNF) family in malignant, as
well as normal, B-cell growth and survival. TNF receptors
use intracellular adaptor proteins (TRAFs) for signaling.
TRAFs are integral to cell growth and survival. This complex
pathway, which includes activation of the key transcription
factor NF-κB noted in many B-cell malignancies, remains
to be completely understood in WM B-cell survival and
growth. Dr. Adamia’s research suggests that in WM, TNF-
family molecules and their adaptor molecules responsible
for normal B-cell growth and survival are regulated by
aberrations in post-transcriptional processes. Further studies
are under way in these fascinating and complex genetic
mechanisms.

B.T. Ciccarelli (Dana-Farber Cancer Institute, Harvard
University) spoke on “The functional role of CD27-CD70
interactions in WM.” Mast cells are often present in excess
in the bone marrow of WM patients and provide a support
and some measure of protection to the WM cells through
several TNF-family ligands (CD40L, APRIL, BLYS). The
interactions between CD27 and CD-70 were evaluated. There
appears to be a functional role for soluble CD27 (sCD27) in
the development of WM. Serum levels of sCD27 may also
be used in the future in as a marker of disease and as a target
in the treatment of WM.

A. Anagnostopoulos (Department of Clinical Therapeutics,
University of Athens School of Medicine, Athens, Greece)
reported on “Serum concentrations of angiogenic cytokines in
WM.” Carefully defining tumor angiogenesis (the induction
of growth of blood vessels in order to supply the tumor in
question) and angiogenic cytokines (chemical messenger
molecules released by tumor cells to promote angiogenesis),
Dr. Anagnostopoulos explained that the ratio of the cytokines
angiopoietin-1 to angiopoietin-2 and angiogenin correlate
with disease severity. Blood vessel density is increased in
30% of patients with WM, yet there is little information for
the role of angiogenic cytokines in WM. The results of Dr.
Anagnostopoulos’ research may lead to the use of angiogenin
in the evaluation and follow-up of WM patients and possibly
to the development of new agents in the treatment of WM
that target angiogenic molecules.

J. Soumerai (Dana-Farber Cancer Institute, Harvard
University) presented a lecture on “Use of the
immunomodulators thalidomide and lenalidomide to
augment rituximab clinical activity in WM.” Rituximab is
used extensively in WM, producing response rates of 30-
40%. Lower response rates are observed in patients with the
polymorphism FcγRIIIA-158 FF; in patients with high
β2M (>3.0 mg/dL); and in patients with high IgM levels
(>6,000 mg/dL). Thalidomide and its analogue lenalidomide
(Revlimid) are two immunomodulators used frequently in MM. In previous studies, these two immunomodulators used in combination with rituximab demonstrated increased antibody-dependant cellular cytotoxicity activity against WM lymphoplasmacytic cells. Soumerai concluded: “Thalidomide in combination with Rituximab is highly active, produces long-term responses, and may overcome unfavorable prognostic determinants previously reported with rituximab monotherapy in WM. The use of thalidomide along with rituximab appears superior, both in efficacy and with regard to tolerability, versus results observed with lenalidomide (Revlimid) and rituximab in a similar clinical population of patients with WM.”

C. Kyriakou (European Bone Marrow Transplant Registry, United Kingdom) reported on the controversial topic of “Allogenic stem cell transplantation (allo-SCT) in WM.” An analysis of 106 cases from the European bone marrow registry.” The use of allogenic stem cell transplant (allo-SCT) has not been extensively studied and therefore limited data are available. Dr. Kyriakou and his team studied the cases of 106 patients who underwent an allo-SCT for WM prior to December 2005. These patients underwent allo-SCT from HLA-identical (75%) or unrelated donors (25%); the median age at transplant was 49 years (21-65; 49% >50 years); thirty-five (33%) patients died, five (5%) from disease progression and 30 (28%) from non-relapse mortality; progression free survival (PFS) rates were 61%, 50% and 48% at 1, 3 and 5 years and the overall survival 69%, 63% and 63%, respectively. Refractory patients were noted to have a higher relapse risk. The use of total body irradiation in the pre-transplant conditioning regimen was associated with a lower relapse risk and better PFS. Dr. Kyriakou concluded: “This particular study suggests that allo-SCT is a feasible and well tolerated procedure even in this rather old population of patients, and it is followed by a low relapse rate and a promising survival.”

X. Leleu (formerly of the Dana-Farber Cancer Institute, Harvard University, and now recently returned to the Laboratoire d’Immunologie et Service des Maladies du Sang, CHRU, Lille, France) spoke on the “Increased incidence of disease transformation and development of MDS/AML in WM patients treated with nucleoside analogues.” Dr. Leleu and his colleagues at Dr. Steven Treon’s laboratory noted an increased incidence of disease transformation and development of myelodysplasia/acute myeloid leukemia (MDS/AML) among WM patients receiving fludarabine. Among nucleoside analog treated patients, 4% transformed to disease transformation and development of MDS/AML among WM patients treated with nucleoside analogs.

A.D. Wechalekar (Royal Free & University College Medical School, London, United Kingdom) reported on the “Clinical profile and treatment outcome in 103 patients with AL amyloidosis associated with IgM paraproteinaemia.” Patients who have WM and amyloidosis are rare. The clinical features of IgM-associated amyloidosis are similar to other forms of amyloidosis, but lymph node involvement is more common. Response to treatment is generally poor, and no complete responses were noted among the patients in this study. Patients who were given intermediate dose chemotherapy appeared to have higher responses. Dr. Wechalekar’s study confirms the difficulty in effectively treating WM patients with IgM-associated amyloidosis. More effective treatment regimens are desperately needed.

IWMF Research Grants, cont. from page 6

individuals within the total population and, in the absence of other events, probably have no impact on the health of the individual carrying them. HAS1 polymorphisms are very frequent in WM patients, suggesting that they predispose individuals to develop WM. Most people carrying these polymorphisms will not develop cancer. However, for a few this inherited predisposition leads to WM when additional HAS1 mutations accumulate in an individual.

Dr. Pilarski speculates that when the combination of inherited polymorphisms and acquired mutations is sufficient to direct the abnormal splicing of HAS1, cancer becomes a virtual certainty. It is likely that inherited and acquired genetic abnormalities in HAS1 act in concert with other cancer-causing events, but HAS1 does appear to be a very early participant in the malignant transformation that leads to WM.

To directly prove that genetic abnormalities in WM are responsible for the abnormal assembly of HAS1, the team cloned the HAS1 gene from a WM patient to make a “splicing construct” that could be tested for its ability to direct the abnormal assembly of HAS1. When the abnormal DNA of the cloned HAS1 gene (DNA taken from the gene cloned from a WM patient) is incorporated in the test cells, these cells acquire the ability to carry out abnormal splicing. Dr. Pilarski’s work thus confirms that the genetic abnormalities are directly responsible for the abnormal proteins that are made in WM. Her results also suggest that the HAS1 abnormalities and the HAS1 splicing process itself are likely to be important targets for the development of new and possibly more effective molecular therapies.
Since the last Torch was published, Talk has been the locus of its usual wide range of topics and emotions related to our condition, augmented by the arrival of at least one new and unusual personality.

Let’s start our selected summary by raising the topic of Rituxan and plasmapheresis (PP for short). Rob Selden wrote to Rob and others that he’d taken solo wonders why more WM patients do not use says that some chemo drugs can added that, like in the art of medicine. 2007. Bob puts his trust in the intuition of his oncologist and where it continued to drop until it reached 249 in October ping-ponged from 4,000 to 7,000 and then down to 1,000, to tell how his IgM, through various courses of treatments, avoid what he calls the “complication” of PP and went on Bob Bent he suffered a flare, by the way) Four months later Pete’s IgM had dropped to 4,300 and has continued to drop ever since. (Pete does not believe numbers. Four months later Pete’s IgM had dropped to 4,300 but, as he says, “that’s another story,” possibly for another Talk summary in the future.

Ron Draftz wrote to Rob and others that he’d taken solo Rituxan at a time when his IgM was above 6,000 and did so without PP as a conservative first step. Ron wondered why doctors of other patients recommended PP—was it because of “flare fear”? Because of worries about potential for increased serum viscosity? Because of the potential for Rituxan inefficacy when IgM climbs above 6,000? Or some weighted combination of factors? Pete DeNardis responded that in late 2004, when his IgM was just short of the 6,000 demarcation, he began treatment with fludarabine and Cytoxan plus Rituxan and did not intervene with PP beforehand. Pete’s doctor was aware of the potential for a spike or flare but was focusing more on symptoms than the numbers. Four months later Pete’s IgM had dropped to 4,300 and has continued to drop ever since. (Pete does not believe he suffered a flare, by the way)

Bob Bent added that, like Joan Kopernik, he wanted to avoid what he calls the “complication” of PP and went on to tell how his IgM, through various courses of treatments, ping-ponged from 4,000 to 7,000 and then down to 1,000, where it continued to drop until it reached 249 in October 2007. Bob puts his trust in the intuition of his oncologist and in the art of medicine.

David Heiser told his story, having received eight weekly Rituxan infusions, the first one when his IgM measured 7,000. David flared to 9,000, at which point his IgM started its fall back to the starting point of 7,000. David and his doctor together concluded that Rituxan was ineffective, and proceeded to the next treatment.

Alice Riggins, whose major symptom is anemia, added to the discussion that she’d had single-agent Rituxan twice since diagnosis—both times without an intervening step of PP. And both times, Alice’s IgM fell from over 9,000 to about 4,800--minor responses (in her words) that left her asymptomatic and pleased to have done so well for four years without recourse to PP and more aggressive treatment.

Ray Patti wondered why more WM patients do not use PP as their main or only treatment, since it is such a safe procedure. Ron Draftz responded that, for all the apparent good PP does, it of course does not reduce tumor burden, that repeated PP destroys blood cells and removes platelets, and, finally, that PP usually includes the addition of albumin, a natural product, to replace the albumin discarded with our IgM. Some reaction may be experienced with repeated addition of donor albumin.

Tinnitus, a persistent loud ringing or tinkling noise within the ear, like that of a bell, proved to be another popular Talk topic over the past few months. Some WM patients seem to experience this aggravation as an offshoot of our condition. Ray Patti reported at one point that his “was blaring like crazy.” Thomas Keyes replied that he visited an ear-nose-and-throat specialist for his tinnitus who told Thomas that as many as 40 million Americans may suffer from it, that there is no medical cure, that it is not traceable to the structure of the ear or brain, and that tinnitus probably has nothing to do with WM.

Liane Cochran-Stafira says that some chemo drugs can cause tinnitus, especially those containing platinum. Her impression was reinforced by her own specialists who said that platinum in chemo drugs does induce the worst tinnitus. In Liane’s case the ringing or hissing (which is how she experiences it) is caused by damage to tiny hair cells in the inner ear, often killed by chemo, resulting in faulty electric signals sent to the nerves and brain. These are interpreted as

**FROM IWMF-TALK**

*by Mitch Orfuss*

From IWMF-Talk, cont. on page 12

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**HOW TO JOIN THE IWMF-TALK**

Here are two ways to join:

1. Send a blank e-mail to: iwmf-talk-subscribe-request@lists.psu.edu
   Make sure to enter the word subscribe as your subject, and do not sign or put anything in the message area (make sure you do not have any signature information in there). Also, do not put a “period” after “edu” or it will reject. Once approved you can post by sending e-mail to iwmf-talk@lists.psu.edu
2. Contact Peter DeNardis at pdenardis@comcast.net and provide your full name
More than 80 people attended the most recent meeting on October 6. A contingent from the newly reactivated Los Angeles support group attended as well. The meeting began with an informal exercise: all those with a WM diagnosis were asked to stand. They then remained standing until the number of years since their diagnosis was called out. It was a useful way for the most recently diagnosed patients to see that there was a good representation of long-time survivors. Dr. Irene Ghobrial of Dana-Farber Cancer Institute made a presentation that provided an education on WM and on the progress of research efforts pursued by herself and her colleagues. Everyone attending was given the opportunity to submit a written question, all of which she answered. Then Dr. Ghobrial took questions from the floor and continued until everyone was satisfied. She also gave out her contact information and invited participants to contact her if they had more questions. The meeting was a very positive experience for the group who so appreciated Dr. Ghobrial’s time and effort to make the meeting successful. Minutes of the meeting are available. Email a request to Marty Glassman at mglassman@cox.net.

Sacramento and Bay Area

Three long-time WMers (each more than ten years) spoke on the subject “Life with WM after Diagnosis” during our last meeting. One had been diagnosed before the IWMF was formed. Luckily, his wife researched his options and discovered Fludara as a treatment option. Now it is so much easier to learn about WM. All three spoke movingly about what gave them strength and inspiration. Often discussions at meetings focus on the mechanics of WM, its treatment and management, while the emotional, spiritual, and personal are overlooked. The group felt grateful for this intimate and generous sharing. The next meeting will be in February or March 2008.

Support Group News, cont. on page 13
COLORADO & WYOMING
The Rocky Mountain support group had a great meeting in September with Dr. Irene Ghobrial in conjunction with the local chapter of the Leukemia & Lymphoma Society. The group plans to host Dr. Steven Treon on Saturday, February 9, 2008, at the Presbyterian/St. Luke’s Medical Center. The meeting will begin at 9:30 am. Roy Parker, the long-time support group leader, has moved up to higher positions within IWMF; the co-facilitators of the group are now Bill Bass and Cindy Furst.

FLORIDA
Ft. Lauderdale Area
The next meeting is scheduled for 1 pm on Saturday, March 22 at Memorial Hospital West in Pembroke Pines. Drs. Grossman and Treon will present, and lunch will be provided. Further details to follow. Contact Charlie Koch at bonnie143@bellsouth.net

Southwest Florida
The next meeting of the SW Florida support group will be held on Saturday, March 1, 2008, from 1 to 4 pm at the Hampton Inn in Sarasota. Dr. Steven Treon will again be our speaker. He will update us on new developments in WM research. There will also be a question and answer session. If you have any questions contact Herb Kallman at 239-466-6911 or margerina@aol.com.

Tallahassee
The Tallahassee Area group enjoyed a great turnout for a lunch meeting at Marie Livingston’s Steak House in Tallahassee in October. Jim O’Neill will be the support leader for 2008 and he can be reached at 850-656-5586. The plans are to meet January 25, April 25, June 27, and October 31, 2008. “The group feels blessed,” says Doris Mathis, “all of us are enjoying good health at the moment.”

MINNESOTA & WESTERN WISCONSIN
Approximately 30 people gathered on September 8 to reconnect with one another and to discuss plans for 2008. The following dates have been reserved for 2008 meetings: January 12; May 17, Dr. Alice Shapiro will discuss nutrition; July 12, Summer Picnic; September 20, Dr. Irene Ghobrial will speak.

MISSOURI
The lucky Missouri group eats well when they meet. Lorraine Wynn provided lunch for the 11 members, including two new members, when they met in Kansas City in October at St. Luke’s East. Dinner was served before the meeting with Dr. Deauna at the January 2008 meeting.

NEW YORK
New York City
In conjunction with the Lymphoma Research Foundation Educational Forum in Brooklyn on October 13, the IWMF coordinated an all-day session on Waldenstrom’s. The day began with a Patient Panel moderated by Guy Sherwood. Panelists were members of the New York City support group, Barbara Bacher, Joseph Dunn, Neil Massoth and Robert Selden. The days’ speakers were Dr. Irene Ghobrial who spoke on Novel Therapeutic Options in Waldenstrom’s, Dr. Morton Coleman, whose topic was The Basics of Waldenstrom’s and More, and Dr. Gwen Nichols. Her topic was Rare Diseases and the Pharmaceutical Industry—How Can We Bring These Worlds Together? At the end of the day all the doctors participated in an Ask the Doctors session moderated by Neil Massoth. The 70-plus participants came from NY, NJ, PA, CT, and NC. There was much enthusiasm and agreement that the program had covered a broad spectrum of WM issues.

Recordings of the sessions from the LRF Annual Educational Forum, including the WM meeting, are available at no cost. Please contact the LRF Helpline directly at 1-800-500-9976 to request a copy.

EASTERN OHIO, WESTERN PENNSYLVANIA, & WEST VIRGINIA
Optimism was the feeling of the day as Dr. Irene Ghobrial spoke to 20 members of the group on September 29 in Pittsburgh, PA. Dr. Ghobrial gave the latest clinical trial updates on perifosine and RAD001 and discussed numerous promising agents at various stages of research. Dr. Ghobrial foresees a hopeful future for the management of WM with synergistic combinations of targeted therapies. All in attendance felt very fortunate to be able to interact with Dr. Ghobrial in a personal setting where questions and concerns could be freely addressed. Thanks to Pete DeNardis, a DVD of the meeting is in the works. Information on availability will be posted on the IWMF Talk List. Group members, including several new members, enjoyed the group sharing and health-conscious lunch by Whole Foods followed by some “unhealthy” desserts. Former support group co-leader, Bob Shaffrey, was honored for his vision and dedication in ten years of service to the group. Fond memories of Julia Wesmiller, who died recently, were shared by Shariann Hall.

OREGON & SOUTHWEST WASHINGTON
The tentative 2008 support group meeting schedule is: February 2, 2008; May 3, 2008; July 26, 2008; and October 25, 2008.
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If you can’t get to a local support meeting, use our IWMF Telephone Lifeline to call a WM veteran.

The lifeline is seeking volunteers who speak a language other than English. If you would like to volunteer, please contact the IWMF business office at 941-927-4963 or info@iwmf.com.

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Brad Alexander  972-529-2002

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FLUDARABINE with cyclophosphamide (Cytoxan)  
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FLUDARABINE with Rituxan  
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Norm Spector  858-454-6313  
Sallie Moore  516-795-3746

ORAL CYTOXAN  
Lou Birenbaum  314-961-5591

PLASMAPHERESIS  
Fred Bickle  805-492-4927

**PENNSYLVANIA**  
Central PA and Northern MD

A larger group than normal gathered to watch the DVD of Dr. Rafat Abonour speaking about WM complications at the 2007 patient education conference in Atlanta. Many said that they were glad they saw this DVD, remembering that if you only learn one thing from any meeting or publication, that one thing could help to save your life. New members discussed their concerns and received encouragement. The group also shared good cheer and delicious snacks provided by Kate and Don Wolgemuth. The next meeting will be on Sunday 10 February, 2008, from 2-4 pm at Messiah Village.

**PENNSYLVANIA (cont.)**  
Philadelphia

The Philadelphia support group met in November at the usual place—a nice comfortable room with large windows and a fireplace at Bryn Mawr Hospital where they have met for 6 1/2 years. Twenty members attended, including two newcomers who were immediately welcomed into the group. After a list of announcements, reminders, and talk about future meetings, the discussion covered the various aspects of shared WM experiences. New members spoke first, had a chance to ask questions, and then learned from what the rest had to say about their experiences. For more details, please contact Heidi, the Pindzola’s little white dog, who made careful notes of everything that was said.

**SOUTH CAROLINA**

The South Carolina WM support group joined forces with the Georgia support group on October 20 in Atlanta to hear Dr. Treon of the Dana-Farber Cancer Institute update us on the latest advancements in WM research and treatments. Mal and Judy Roseman of the Georgia group spearheaded this meeting. Over the past six months the South Carolina group has welcomed four new WM patients. The next meeting of the South Carolina group will be held in March or April 2008.

**TENNESSEE**  
W. Tennessee, E. Arkansas, N. Mississippi

Member Colleen Casey took excellent notes on her laptop during the Atlanta patient education forum. Since the DVDs of the meeting were not available at the time of the support group meeting, her summaries generated many questions and much conversation.

**WASHINGTON D.C. & METROPOLITAN AREA**

In the “What’s New?” department, Dr. Irene Ghobrial was the group’s featured speaker in November 2007.
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James Townsend 352-376-3664
Allen Weinert 603-863-5347
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SOCIAL SECURITY DISABILITY
Howard Prestwich 815-233-0915

SPLENECTOMY
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STEM CELL TRANSPLANT
Howard Donley 307-587-3397
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In memory of Francieta Winter:

In memory of Frank & Jim Berg’s new granddaughter:
Neil & Joyce Massoth

In memory of Dr. John C. Bernloehr:
Ann Lipkowitz and the Grand Ledge Country Club Anniversary Golf Benefit

In memory of Herbert Bionl:
Rick & Iris Park

In memory of Maryellen Bowering:
Michael Sugarman

In memory of Deborah & Kkyle Brunelle:
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In memory of Gayle Clites:
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In memory of Stanley Fisher:
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Washington DC Area Support Group

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Nancy Fisher

In honor of Nancy E. Lambert:
Emily Lambert & Brownie Troop #1462

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Brent & Christine Harmes

In honor of Rita Ziants:
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Since September, 2007, the following contributions to the International Waldenstrom’s Macroglobulinemia Foundation were made in honor of:

From the following Members of the Class of 1959, Our Lady of Nazareth Academy, Wakefield, MA:
COOKS’ HAPPY HOUR

by Penni Wisner and Nancy Lambert

Some years ago, Nancy Lambert and I sat together at an IWMF Patient Education Conference. As happens so frequently among its passionate adherents, the subject soon turned to food. Nancy and her husband Larry had recently started a daily ritual of a Happy Hour. Around 5 pm Nancy would fill a small tray with all the healthy food she could find in the house, glasses of red wine, plus two dog biscuits. Then they all—Nancy, Larry, and Fiona, the dog, would go sit outside to watch the evening advance.

To keep her menu interesting, Nancy was always looking for new ideas for healthy, fast, and easy snacks. We began trading ideas—almond-stuffed dates for roasted, herbed nuts. We do not count calories, carbohydrates, or fats. Any whole food—preferably organic, seasonal, and not packaged (except for the exceptions!)—is fair game. And we want to focus on ideas vs. detailed recipes. Since writing recipes is what I do professionally, I can say with a high degree of confidence that they usually require more space than this column could provide. We invite you to share your ideas with us so that we might, in turn, share them with everyone in this space.

Freshly dried beans hit the market in late fall and early winter. They do not need presoaking and, if you have a pressure cooker, they cook in very little time. But even without, what could be simpler than gently simmering beans in water or stock to cover by a couple of inches with a few flavoring agents including herbs such as a bay leaf; thyme, parsley, or sage sprigs; and a couple of cloves of garlic? I love star anise and add one or two whole ones depending on how many beans I am cooking. You might try ground coriander or cumin to vary the flavor.

Depending on the freshness and size of the beans, they might need an hour of such cooking but they do not need tending meanwhile. Add salt to the cooking water only when the beans are tender. And save that cooking liquid.

Once cooked, beans can be used in soups, as a side dish, as a base for roasted fish, etc. But for a make-ahead snack to serve on toast or crackers or as a dip for raw vegetables, mash the beans by hand or in a food processor with some good-quality extra-virgin olive oil and salt and pepper to taste. Add enough bean water to make a thick puree. To add more flavor and spice, if you like, heat a little olive oil in a pan and add thinly sliced garlic. Cook until pale gold. Add chili flakes, stir, and then fold into the bean puree. Serve at room temperature; refrigerate up to about three days.

And, once the beans are at a simmer, why not add a small bowl of sundried tomato dipping oil to your tray of healthy treats? Blend by hand olive oil, finely chopped sundried tomatoes, thyme or a favorite spice, add salt and pepper to taste. Serve in a shallow bowl with chunks of crusty bread.

Recently, the Lamberts moved to Florida. Nancy wrote: “Tonight, I am having my first Happy Hour in FL. I am serving your herbed nuts, marinated olives, multigrain bread with sundried tomato dipping oil, sardines, and fruit. I am not sure how important food is in our battle against WM, but I like to think that our lifestyle is contributing to our temporary remission.”

Penni (penniw@pacbell.net) and Nancy (llne3@aol.com) look forward to hearing your suggestions for healthy, fast, and easy snacks.

Our motto: Eat Well to Stay Well

President’s Corner, cont. from page 4

On the weekend of December 1-2 the Board of Trustees met in Dallas for our regular board meeting. As we went over the budget and the early results of our campaign for membership dues and research funds, we noticed a slowdown in member dues and in research contributions as of the end of November. From the pattern of donations in the past we are aware that there are more contributions at the end of the year. However, it should be clear to all members and friends of IWMF that this organization functions solely on contributions. I hope that you will keep IWMF in the forefront of your charitable contributions in the coming months. In order to stay on track we need your funds as well as the volunteer energy and talents that have been committed to this cause.

Thank you for everything you do to support IWMF.

Stay Well,

Judith May
President, IWMF

IWMF TRUSTEES AND COMMITTEE LISTING

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