



# *Era of Hope*

Department of Defense  
Breast Cancer Research  
Program Meeting

September 25-28, 2002  
Orange County Convention Center  
Orlando, Florida

***SUPPLEMENT***

**SPEAKER REPLACEMENTS AND ADDITIONS**

- Thurs., Sept. 26, 2002      *Symposium 3.* Michael Morse will replace Herbert Kim Lyerly as Co-Chair
- IS 4.* The Genomic Revolution: What Is the Impact on Patients?  
\* Co-Chairs: Jeffrey M. Trent and Kathleen Zeitz
- Informed Consent and Patient Perspective  
\* Barbara Bowles Biesecker
- Genomics and the Industry Perspective  
\* Patrick Terry
- Patents and Access: Ethics and Public Policy  
\* Mary R. Anderlik
- Patient Implications/Barriers to Research  
\* Kathleen Zeitz
- Fri., Sept. 27, 2002      *Symposium 13.* Barbara A. Brenner will replace Cathie Ragovin as Co-Chair
- Symposium 13.* Alexey Glazyrin will replace Malcolm S. Mitchell
- Symposium 17.* Judith H. Hariton will replace Alisa Gilbert
- Sat., Sept. 28, 2002      *Symposium 26.* Tyler Curiel will replace Shuang Wei
- Symposium 32.* Dale Porter will replace Ana Merlos-Suarez and will be the first speaker in Symposium 32
- Symposium 32.* Addition. Consumer Advocates as an Integral Part of a Clinical Trials Program. Michele Rakoff

**POSTERS REASSIGNED**

- From P37-9 to P22-46      The Radiation-Induced P53 - 14-3-3 Interaction Is Critical for P53 Function, *E.S. Stravidi et al.*
- From P55-3 and P55-10 (combined) to P3-34      Expression Profiling of Stress-Treated EMT6 Cells Expressing Mutant IκBa Protein Reveals Candidate Genes Involved in the Prevention of Stress-Induced Drug Resistance by Inhibition of NF-κB Activation, *L. M. Brandes et al.*
- and
- TGF-β and the PDGFRα/MAPK Signaling Pathway Are Downstream of NF-κB Activation in the Development of Stress-Induced Resistance to Etoposide, *K. A. Kennedy et al.*
- From P49-11 to P14-11      Synthesis and In Vitro Photosensitizing Efficacy of Fluorinated Porphyrin-Based Photosensitizers for In Vivo MR Studies, *S.K. Pandey et al.*

**LATE POSTERS - SUBMITTED/ASSIGNED**

- P5-19 Proteomic Analysis of Estradiol-Independent Growth of a MCF7 Derivative Cell Line, *S. H. Seeholzer, A. T. Yeung, B. D. O'Connell, and R. C. Clark*
- P7-43 Modulation of Estrogen Receptor Alpha Levels by Estrogen and Antiestrogens, *S. Anghel, V. Perly, M. Lupien, and S. Mader*
- P9-26 Gamma-Synuclein, A Candidate Oncogene, Is Aberrantly Expressed in Breast Cancers and Can Enhance Cancer Cell Motility, Promote Tumor Cell Survival, and Inhibit Stress-Induced Apoptosis, *Z. Pan, W. Bruening, B. I. Giasson, V. M. Lee, and A. K. Godwin*
- P13-15a The Effect of Phytoestrogens on Normal Breast Tissue in Postmenopausal Breast Cancer Survivors, *M. R. Palomares, A. Richardson-Lander, J. R. Gralow*
- P22-47 Cell Proliferation and Chromosomal Changes in DDT-Exposed Rats, *P. T. Uppala, A. Fullwood, R. Sagay, A. Tousson, G. Uppala, S. K. Roy, D. A. Eastmond, and C. A. Lamartiniere*
- P34-1a A Comparison of Massage Therapy, Relaxation, and Standard Care for Women with Breast Cancer, *G. Ironson, M. Hernandez-Reif, and T. Field*
- P43-5a The Genetics of Breast Cancer in Newfoundland, *T-L Young, J. S. Green, and M-C King*

**POSTERS WITHDRAWN FROM POSTER SESSIONS**

- P7-18 Development of a Mass Spectrometric Protocol to Accurately Evaluate the Functional Effect of Oxidative Stress on the Estrogen Receptor's DNA-Binding Domain, *J. E. Meza et al.*
- P17-7 Monte Carlo Simulations for Modulated Electron Beam Radio-Therapy Treatment Planning, *M. C. Lee et al.*
- P18-4 Evaluation of the Predictive Value of Environmental Pollutant Release Data in Assessing Breast Cancer Risk, *K. Cunningham et al.*
- P22-3 Expression of a Defective Repair Gene, DNA Polymerase B, in Mammary Glands of Transgenic Mice, *S. Banerjee et al.*
- P26-23 SRC and Focal Adhesion Kinase Regulate ACK-1 Tyrosine Kinase Phosphorylation but Are Not Required for Its Downstream Signaling, *K. Modzelewska et al.*
- P34-3 Education and Outreach for Breast Imaging and Breast Cancer Patients, *D. M. Farria et al.*

# **PROTEOMIC ANALYSIS OF ESTRADIOL- INDEPENDENT GROWTH OF A MCF7 DERIVATIVE CELL LINE**

**Steven H. Seeholzer, Anthony T. Yeung,  
Bryan D. O'Connell, and Robert C. Clarke**

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The development and progression of breast cancer is a stepwise process characterized by genetic and epigenetic changes. One common early event in the development of breast cancer is the loss of dependence of a breast epithelial cell on estrogen for growth while still possessing a functional estrogen receptor. The changes are reflected by and, at some level, driven by specific alterations in the patterns of protein expression and subsequent post-translational modification. We have previously derived from MCF7 cells a cell line, LCC1, which grows independently of estradiol (E2). Initial studies showed patterns of protein expression in LCC1 cells grown in the absence of E2 that appeared very similar to those patterns in MCF7 grown in the presence of E2 [1]. The purpose of the work presented here is to complete a more comprehensive description of the influence of E2 on the proteomes of these two cell lines and to describe the changes associated the phenotype switch to E2-independent growth.

MCF7 and LCC1 cells were grown to study the effect of E2 on protein expression patterns. Total cell proteins were analyzed by two dimensional polyacrylamide gel electrophoresis (2D-PAGE). Digital images of the silver or Coomassie stained 2D gels were obtained and quantitative comparisons of the gel images was done with the Progenesis image analysis software. Protein spots showing a consistent two-fold or greater change between different cells/conditions were selected for identification by mass spectrometry.

Among the several hundred proteins in the pI 5 to 8 range considered we find 45 specific proteins increase and 29 proteins decrease under the influence of E2 on MCF7 cells, whereas 13 proteins increase and 47 decrease upon E2 addition to LCC1 cells. Comparing the effect of phenotype switch from MCF7 (E2-dependent) to LCC1 (E2-independent) in the absence of E2, we find 73 proteins increase and 51 decrease. Roughly half of these latter changes are also held in common with the effect of E2 on MCF7 cells. Results of protein identifications suggest a role for apoptosis in controlling the growth characteristics of MCF7 cells that appears to be lost in the LCC1 cells. Other models will be discussed along with their relevance to biomarker discovery.

## **MODULATION OF ESTROGEN RECEPTOR ALPHA LEVELS BY ESTROGEN AND ANTIESTROGENS**

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Mathieu Lupien, and S. Mader**

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The turn-over of estrogen receptors is regulated in breast and uterine cells in a ligand-specific manner. Both estradiol and the full antiestrogen ICI182,780 induce degradation of ER $\alpha$  in MCF7 cells, the effect of ICI182,780 being more dramatic. On the other hand, 4-hydroxy-tamoxifen (Tam) stabilizes ER $\alpha$  against degradation. These effects can also be observed in transiently transfected HeLa cells. Here we report that contrary to Tam, raloxifene (Ral) reduces ER $\alpha$  steady state levels in transfected HeLa cells. Transfection of ER $\alpha$  deletion mutants and point mutants in MCF7 or HeLa cells indicated that the ligand binding domain is crucial for the effect of ligands on ER $\alpha$  stability, and that the position of helix 12 (H12) is an important determinant for the effects of both estrogen and antiestrogens. Several mutations in the ligand binding domain in the vicinity of H12 were found to stabilize ER $\alpha$  in the presence of Ral and/or reduce ER $\alpha$  levels in the presence of Tam. In addition, mutations at Asp 351, which is involved in a hydrogen bond interaction with the tertiary amine in the side chain of Ral, abolished the capacity of this antiestrogen to reduce ER $\alpha$  levels. Together, these results suggest that differences in the binding mode of Ral and Tam and resulting differences the tertiary structure of ER $\alpha$  in the presence of these two SERMs may be responsible for their opposite effects on ER $\alpha$  steady state levels.

# GAMMA-SYNUCLEIN, A CANDIDATE ONCOGENE, IS ABERRANTLY EXPRESSED IN BREAST CANCERS AND CAN ENHANCE CANCER CELL MOTILITY, PROMOTE TUMOR CELL SURVIVAL, AND INHIBIT STRESS-INDUCED APOPTOSIS

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Synucleins are a family of small, highly conserved proteins predominantly expressed in neurons. Although the normal functions of the synucleins are not fully understood,  $\alpha$ - and  $\gamma$ -synuclein have been implicated in the pathogenesis of several neurodegenerative diseases and cancer, respectively. Recently we and others have found that  $\gamma$ -synuclein, a candidate proto-oncogene also known as *BCSG1* or persyn, is dramatically up-regulated in the vast majority of late-stage breast (>70%) and ovarian (>85%) cancers and that  $\gamma$ -synuclein over-expression can enhance tumorigenicity. To address the biological function of  $\gamma$ -synuclein and its role in the malignancy of breast cancer, we ectopically over-expressed  $\gamma$ -synuclein in several cancer cell lines. We have observed that exogenous expression of  $\gamma$ -synuclein in tumor cells can induce stress fiber formation, and enhance cell motility and invasion as analyzed by Boyden chamber assay. Consistent with their role in cell migration, at least one of the Rho family members in its activated (GTP-bound) form is constitutively elevated in cells that over-express  $\gamma$ -synuclein. The enhanced cell migration was completely inhibited when the cells were treated with *C. difficile* Toxin, a bacterial toxin that can inactivate Rho family members. We have also found that  $\gamma$ -synuclein is associated with two major mitogen-activated kinases (MAPK), i.e., extracellular signal-regulated protein kinases (ERK1/2) and c-Jun N-terminal kinase 1 (JNK1). Over-expression of  $\gamma$ -synuclein lead to constitutive activation of ERK1/2, and inhibition of ERK using U0126, a MEK1/2 inhibitor, blocked  $\gamma$ -synuclein enhanced cell migration. Over-expression of  $\gamma$ -synuclein also lead to down-regulation of JNK1 in response to a host of environmental stress signals, including UV light, heat shock, sodium arsenate, nitric oxide, and chemotherapeutic drugs. For example,  $\gamma$ -synuclein over-expressing cells were found to be (4 to 5-fold) more resistant to paclitaxel as compared to the parental cells. This resistance could be partially restored when ERK activity was inhibited using U0126. In addition, activation of the mitochondrial apoptotic pathway by paclitaxel was blocked by ectopic expression of  $\gamma$ -synuclein, suggesting that  $\gamma$ -synuclein may be a novel negative regulator of breast cancer apoptosis. Taken together, our data indicate that  $\gamma$ -synuclein can enhance cell motility and invasion and that Rho and ERK pathways are required for the enhanced cell migration. Furthermore,  $\gamma$ -synuclein is likely to be involved in the pathogenesis of breast and ovarian cancer by promoting tumor cell survival under adverse conditions and by providing resistance to certain anti-cancer drugs. Because of its high frequency of expression in late-stage breast and ovarian cancers,  $\gamma$ -synuclein may be a promising target for cancer therapy.

**THE EFFECT OF PHYTOESTROGENS ON  
NORMAL BREAST TISSUE IN POSTMENOPAUSAL  
BREAST CANCER SURVIVORS**

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**ABSTRACT:** Phytoestrogens have received recent media attention as a form of breast cancer prevention. Although epidemiologic studies support this claim, there are no prospective clinical trials demonstrating such a protective effect. The proposed project aims to scientifically evaluate the effect of a food-free phytoestrogen intervention, in the form of an isoflavone tablet, on the breast tissue of postmenopausal women with a history of breast cancer. Sixty disease free, post-therapy, postmenopausal women with in-situ or early invasive (St. 0-IIA) breast cancer are to be randomized to either 100mg/d isoflavone tablets or placebo for one year. Biopsies of the uninvolved breast are examined for histologic proliferative changes in response to phytoestrogens, as well as for immunohistochemical breast cancer biomarkers. Mammography is performed to assess changes in breast density in response to phytoestrogens, and for close monitoring for recurrence. As secondary endpoints, menopausal symptoms, vaginal epithelial changes, endometrial histology, and serum steroid hormones are also being measured.

The trial was opened to accrual in June 2001. Since then, 632 breast cancer patients have been screened through the Seattle Cancer Care Alliance to identify 62 potentially eligible candidates. We received 33 additional self or clinician referrals. From both groups, 26 were found to be ineligible after further screening, 41 refused participation, and 11 were eligible and have consented to participation so far. The number one reason for ineligibility is stage (52%). To remedy this, the investigative team has recently approved to open accrual to include women with Stage IIB disease. Reasons for refusal have included: Don't agree to breast biopsy (6), Too invasive for other reasons (3), Fear phytoestrogens will promote recurrence (3), Prefers food sources of phytoestrogens (2), Otherwise refuses to take pills (6), Refuses placebo-controlled randomization (1), Lack of interest (4), Psychosocial reasons (4), Other miscellaneous reasons (6), or Passive refusal (6). In order to increase recruitment yield, a mechanism to see patients who receive their oncologic care outside the sponsoring institution has been developed, and a community outreach campaign begun.

## CELL PROLIFERATION AND CHROMOSOMAL CHANGES IN DDT-EXPOSED RATS

**PT Uppala, A Fullwood, Sagay R, A Tousson, G Uppala,  
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The environmental estrogen DDT and its metabolites have been implicated in breast cancer. The exact mechanisms however, remain to be elucidated. Residents of Triana in Alabama have been exposed for nearly fifty years to the recently banned pesticide DDT. Pilot studies have indicated a breast cancer prevalence of 18% (17 cases among 96 women). This research focused on the role of DDT in cell proliferation and genetic alterations in the rat mammary gland to provide an understanding in the development of breast cancer in the DDT- exposed Triana population in Alabama. Twenty-one day old Sprague-Dawley rats were administered DDT (50 mg/kg b.w.); DMBA (40mg/kg b.w.)+DDT or DDT+DMBA + Genistein (250mg/kg diet) for 14 days. DDT was administered subcutaneously and DMBA by gavage. Two hours before being killed the animals were administered 5-bromo-2-deoxyuridine (BrdU) by I.P. injection. Mammary glands were dissected, embedded in O.C.T medium and frozen in 2-methyl butane pre-chilled in liquid nitrogen. Frozen glands were cut with a Cryostat generating 10µm thick sections that were mounted on glass slides. Sections were processed for either BrdU incorporation or fluorescence *in situ* hybridization (FISH). To determine changes in chromosome number, we used FISH with DNA probes for rat chromosomes 4 and 9. DNA probes were amplified and labeled with digoxigenin-dUTP or biotin-dUTP by nick translation. Previously described methods were used to perform the FISH experiments. Digoxigenin-labelled probe was detected using a FITC-conjugated sheep anti-digoxigenin antibody and biotin-labelled probe was detected with Alexa 555-streptavidin antibody. DAPI was used to counterstain the DNA. One tailed Fisher's exact test was used to determine statistical significance. Significant increases in the incorporation of BrdU were seen in the rats treated with DDT and DMBA together. Certain areas or "hot spots" had numerous intensely stained cells in the DDT+DMBA group. Preliminary studies with FISH assay showed significant increases in hypodiploidy in the DDT+DMBA-treated rats. Increases in hyperdiploid cells were seen in the DMBA and the DDT+DMBA-treated rat mammary tissue. A nonsignificant increase in hyperdiploidy was also seen in the DDT-treated rats. The increases in hypodiploidy and hyperdiploidy in the DDT+DMBA-treated animals were increased relative to those seen in the animals treated only with DMBA. These initial studies suggest that DDT can induce cell proliferation and may enhance chromosomal alterations occurring in the rat mammary gland.

**A COMPARISON OF MASSAGE THERAPY,  
RELAXATION, AND STANDARD CARE FOR  
WOMEN WITH BREAST CANCER**

**G. Ironson, M. Hernandez-Reif, and T. Field**

University of Miami Touch Research Institute

Women with stage 1 or 2 breast cancer were randomly assigned to one of three group conditions: (1) Receive three massage therapy sessions per week for 5 weeks, (2) practice three Progressive Relaxation (PMR) therapy sessions per week for 5 weeks, or (3) a standard care control group. Massage therapy effects over 5 weeks included reduced anxiety, depressed mood and anger, increased urinary dopamine and serotonin levels; and increased plasma natural killer cell number and lymphocytes when compared with the standard care control group. After the first session, women reported reduced pain on the first day of the study and showed an increase in percent lymphocytes by the last day of the study. These findings suggest that massage, and to a lesser extent PMR can be effective adjunct therapies for attenuating psychological, physical and immunological symptoms associated with breast cancer.

## THE GENETICS OF BREAST CANCER IN NEWFOUNDLAND

Terry-Lynn Young,<sup>1,2</sup> Jane S. Green,<sup>2</sup>  
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A genetic susceptibility to breast cancer was confirmed with the discovery of two highly penetrant breast cancer genes, BRCA1 (17q21) and BRCA2 (13p21). However, our ability to predict the lifetime risk and age of onset of cancer in high-risk families is limited. The majority of linked families have “private” mutations, ie. disease-associated changes in BRCA1 or BRCA2 that are not found in other women with breast cancer. Furthermore, many high-risk families have no detectable mutation in either BRCA1 or BRCA2 (“mystery families”) and probably have perturbations in as yet undiscovered breast cancer genes. A population-based study of familial breast cancer in extended Newfoundland families will allow us to: (1) describe the natural history of every cancer susceptibility allele we find and (2) identify extended “mystery families” to use in the search for novel breast cancer genes.

Gene discovery in complex diseases is easier in isolated populations. In Newfoundland, most of its 530,000 residents are the product of natural expansion from ~20,000 English and Irish fisher folk who founded many coastal fishing villages (outports) between 1700 and 1830. More than 50% of this population still lead a traditional lifestyle and live close to their extended families in outports of < 2000 people. The relative homogeneity of the genetic and environmental background, the availability of large families, and centralized public health records greatly facilitate gene discovery. The Newfoundland population has already made major contributions to the genetics of human disease, including inherited predisposition to cancer. The role of mismatch repair genes in hereditary colon cancer was first discovered in an extended Newfoundland family.

We have screened 157 women (proband) with breast cancer for mutations in BRCA1 and BRCA2. We use conventional methods including single-stranded confirmation polymorphism (SSCP), protein truncation testing (PTT) and direct sequencing to search for disease-associated mutations. We have identified 5 probands with truncating mutations in BRCA1 and 4 probands with mutations in BRCA2. We also report that 3.0% of probands carry the recently identified 1100delC mutation in the cell cycle CHK2 gene. In families with mutations, we are collecting DNA and medical records from all available family members to assess the age of onset and lifetime risk of breast and other associated cancers. So far, we have also excluded BRCA1 and BRCA2 as the cause of breast cancer in 3 probands. In these families we are collecting DNA samples from informative relatives to possibly exclude linkage to 17q21 and 13p12.

The study of the genetics of breast cancer in Newfoundland will provide information to develop screening protocols for families with known mutations in cancer genes and may lead to the discovery of novel breast cancer susceptibility genes.

## **PHYSICIANS: CONTINUING MEDICAL EDUCATION**

### *Accreditation Statement*

The U.S. Army Medical Command is accredited by the Accreditation Council for Continuing Medical Education to provide continuing medical education for physicians.

### *Credit Designation Statement*

The U.S. Army Medical Command designates this educational activity for a maximum of 29.5 credit hours in category 1 credit toward the AMA Physician's Recognition Award. Each Physician should claim only those hours of credit that he/she actually spent in the activity.

### *Statement of Need*

The planning committee for this activity has determined that an important need exists to provide physicians involved in breast cancer prevention, diagnosis, treatment, or quality of life care with important updates to stay informed of new research and the most current technologies and treatments for breast cancer. This activity is being given because breast cancer is a major health concern affecting one in eight women. The ability to cure breast cancer decreases with disease progression and therefore new detection, diagnostic, therapeutic, and quality of life research is needed to find prevention strategies, new cures, or improve survival and life after breast cancer. This activity will provide a forum in which the research coming from the DOD Breast Cancer Research Program will be presented together with symposia, and plenary presentations by leading experts in oncology and breast cancer consumer advocates, providing the medical community with important updates in the progress toward the above goals.

### *Learning Objectives*

At the conclusion of the Era of Hope Meeting, participants should be able to:

- Describe and discuss recent research results and the innovative approaches that are now being used to study the basic biology, prevention, detection, diagnosis, and treatment of breast cancer, and ways to improve patient quality of life.
- Comprehend the latest advancements in the genetics and biology of breast cancer; in particular, the role of signal transduction pathways in breast cancer development and progression.
- Comprehend the latest advancements in the field of breast cancer prevention.
- Comprehend the latest advancements in identification and characterization of molecular targets that can serve as the basis for individualized breast cancer therapy.
- Interact and collaborate with breast cancer researchers who work in different scientific and clinical disciplines and network with consumer advocacy organizations.

### *Intended Audience*

This educational activity is designed for civilian and military physicians; nurses; and researchers, especially oncologists, radiologists, pathologists, general/family practitioners, and osteopaths who might be involved in patient diagnosis, treatment, prevention, or post-cancer care. No special prerequisites are required to attend this educational activity.

*Disclosure of Faculty Relationships*

As a provider accredited by the ACCME, it is the policy of the U.S. Army Medical Command to require the disclosure of the existence of any significant financial interest or any other relationship a faculty member or a sponsor has with the manufacturer(s) of any commercial product(s) discussed in an educational presentation. Disclosure will be made in the handout materials.

*Disclosure of Unlabeled/Unapproved Uses of Drugs or Devices*

NOTICE: In accordance with the ACCME Standards for Commercial Support, the audience is advised that one or more presentations in this continuing medical education activity may contain reference(s) to unlabeled or unapproved uses of drugs or devices. Disclosure will be made in the handout materials.

*Acknowledgment of Commercial Support*

There is no commercial support associated with this educational activity.

**NURSES:  
CONTINUING EDUCATION FOR NURSES WHO ARE ACTIVE DUTY U.S. MILITARY,  
RETIRED U.S. MILITARY, OR ARE CIVILIANS EMPLOYED BY A U.S. ARMY HOSPITAL**

This Educational Design I activity, assigned ANC-CHEP #L162 for 41.4 contact hours, has been approved by the U.S. Army Nurse Corps which is accredited as an approver of continuing education in nursing by the American Nurses Credentialing Center's Commission on Accreditation

**NURSES: CONTINUING EDUCATION FOR ALL U.S. NURSES**



**ANCC (Nursing Credit)** – Approved for 41.4 contact hours of continuing education for RNs, LPNs, LVNs, and NPs. This program is cosponsored with Medical Education Collaborative, Inc. (MEC). MEC is accredited as a provider of continuing nursing education by the American Nurses Credentialing Center's Commission on Accreditation.  
Provider approved by the California BRN provider number: CEP-12990 for 41.4 contact hours.  
Florida BN Provider Number: FBN - 2773

## **PARTNERSHIP PROGRAM INTERACTIVE GROUPS**

As part of the of the BCRP's commitment to foster communication between scientists and consumers, a Partnership Program has been added to the Era of Hope agenda. Teams of symposium Co-Chairs, experts in different fields of research and consumer advocates, will be leading Partnership Program interactive groups for consumers at the beginning of the poster sessions on September 26, 27, and 28, 2002. Interactive groups will be developed around specific research areas, or poster sessions. Partnership Program interactive groups will begin with a 15-minute discussion on the research area, current research in that field, and relevance to breast cancer. Recommended posters will be provided and guided tours of some posters will be offered following the overview.

Consumers and scientists are encouraged to sign-up for Partnership Program interactive groups. Sign-up sheets will be available at the Information Desk in the Registration Area at the Orange County Convention Center. To encourage discussions, space will be limited in individual groups. Meeting times and locations for each Partnership Program interactive group will be posted on the message board.

## **CONSUMER HOSPITALITY ROOM**

A Consumer Hospitality Room is located in *Room 301* in the Orange County Convention Center.

## **PRESS ROOM**

All attending media must register in the Era of Hope Press Room, located in the Orange County Convention Center, Room 202C (telephone: 407/685-4275 fax: 407/685-4276). The Press Room will be open during the following hours:

|                         |                    |
|-------------------------|--------------------|
| Wednesday, September 25 | 2:00 p.m. – 7 p.m. |
| Thursday September 26   | 8:00 a.m. – 5 p.m. |
| Friday, September 27    | 8:30 a.m. – 5 p.m. |
| Saturday, September 28  | 8:30 a.m. – 5 p.m. |

## **ATTENDEES TRAVELING ON INVITATIONAL TRAVEL ORDERS (ITOs)**

The contractor supporting the Congressionally Directed Medical Research Programs (CDMRP) in assisting with ITO travel during the meeting is Science Applications International Corporation (SAIC). SAIC will coordinate all ITO attendee travel changes with Carlson Wagonlit during the course of the Era of Hope 2002 meeting.

Assistance may be obtained at the Travel Desk in the Registration Area during the following hours:

|                         |                        |
|-------------------------|------------------------|
| Wednesday, September 25 | 11:00 a.m. – 8:00 p.m. |
| Thursday, September 26  | 7:00 a.m. – 7:00 p.m.  |
| Friday, September 27    | 7:00 a.m. – 7:00 p.m.  |
| Saturday, September 26  | 7:00 a.m. – 7:00 p.m.  |

## **EXHIBIT DISPLAYS**

Please visit the Congressionally Directed Medical Research Programs and U.S. Army Medical Research Acquisition Activity displays outside of Poster Hall A1, Thursday, September 26, 6:30 p.m. – 8:30 p.m.; Friday, September 27, 12:15 p.m. – 2:15 p.m.; and Saturday, September 28, 6:55 p.m. – 8:55 p.m. The exhibit is concurrent with all Poster Sessions.

## **AUDIO RECORDINGS**

Audiotapes will be available for Session speakers who have agreed to have their presentations audio recorded. Tapes will be available at the meeting; compact disks can be ordered. These recordings will serve to stimulate research that focuses on addressing important breast cancer questions. The audio recordings will assist in communicating the contributions of the Breast Cancer Research Program to the scientific and consumer communities. Audio recordings are available in the registration area from ACTS, Inc.

## **ERA OF HOPE 2002**

**DON'T MISS THE  
SPECIAL LUNCHEON SESSION**

### ***DOD BCRP NEW INVESTIGATORS***

*Please join us for lunch and an opportunity to  
hear about research from new minds!*

**Saturday, September 28, 2002  
Rosen Centre Hotel  
Grand Ballroom C**

**11:50 a.m. – 1:10 p.m.**

*For those who have reserved a space at the luncheon and selected a meal, your luncheon coupon is behind the badge you received when registering.*

*For those who have **not** reserved a space at the luncheon but wish to do so, please contact the Registration Desk no later than Wednesday evening, September 25. This luncheon is included in the registration fee. A head count is necessary. Thank you for your cooperation.*