THE CANCER LETTER

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Group Sets Prostate Research Agenda; Strategy, Funding Demands To Follow

Before asking for money on Capitol Hill, the National Prostate Cancer Coalition and scientists who advise it met at M.D. Anderson Cancer Center to determine what needs to be done.

As a result of their meeting, the advocates and scientists last week produced a plan for research in prostate cancer. Now, the coalition will proceed to estimate the cost of those initiatives.

"I think it's significant that within 80 days of the formation of the

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In Brief

Issell To Retire As Hawaii Center Director; M.D. Anderson Honors LeMaistre, Faculty

BRIAN ISSELL, director of the University of Hawaii, Cancer Research Center of Hawaii, since 1988, plans to resign from his director position in the summer of 1998. He will continue at the university as professor of medicine and researcher. A national search will be conducted for his successor. Under Issell's directorship, the center was the first to acquire and subsequently convert an NCI P20 Cancer Center Planning Grant to a P30 Cancer Center Support Grant. . . . **CHARLES LEMAISTRE**, president emeritus of M.D. Anderson Cancer Center, was awarded the Board of Visitors Award by the University Cancer Foundation Board of Visitors. The 160-member board consists of prominent volunteers from around the U.S. LeMaistre also was honored recently at a Faculty Honors Covocation. LeMaistre was one of 14 of the cancer center's faculty who received Distinguished Service Awards. In addition, the center named an award in his honor, the Charles A.LeMaistre Outstanding Achievement Award in Cancer. The award was presented to **Frederick Becker**, vice president for research, recognizing Becker's two decades of leadership at M.D. Anderson. Other recipients of the Distinguished Service Awards were: **Mohamed K. Ali, John Batsakis, Richard Black, Vincent Chuang, Luis Delclos, Jack Edeiken, C. Stratton Hill Jr., Dah Hsi Ho, Bruce Mackay, William Murphy, John Murray, Luceil North** and **Sidney Wallace**. Faculty Achievement Awards were presented to **Randy Legerski, Lovell Jones, William Plunkett Jr., Raphael Pollock**, and **Merrick Ross**. **Bruno Calabretta**, professor at Jefferson Cancer Institute and director of the Molecular Biology Program at Kimmel Cancer Institute, received the Distinguished Alumnus Award.

Prostate Cancer Coalition Develops Research Plan

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coalition it was possible to put together a meeting of this magnitude and come up with a research agenda," said Richard Howe, former president of Pennzoil, who was one of the organizers of the Oct. 12-13 conference. "Now this agenda can be quantified."

With a research plan completed, NPCC is formulating a political agenda and making plans to establish a presence in Washington. If these goals are met, it is safe to say that next year's appropriations process will be very different from this year's, as prostate cancer activists join the National Breast Cancer Coalition and AIDS patient advocates in demanding funds for specific diseases.

Funding Infusion or Redistribution?

Regardless of what one may think of earmarking research funds, the efforts of the prostate cancer lobby are likely to lead to an infusion of new money—or a redistribution from existing programs. Research priorities will be affected in either case.

While prostate cancer advocates are yet to exhibit the political savvy of their counterparts at NBCC, they do have substantial clout. Even before the prostate cancer coalition became functional, the Washington lobbyists for CaP CURE, an organization started by the financier and prostate cancer survivor Michael Milken, secured $45 million in earmarks for prostate cancer research in the Department of Defense.

Though Milken's role in the new coalition is yet to be determined, CaP CURE executive director Richard Atkins sits on the NPCC board. "The [research agenda] conference was the next logical step following CaP CURE's work with Congress to increase prostate cancer research funding by $45 million," Atkins said. "It helps identify appropriate directions for these funds and defines how much we are likely to need in the short term, as we attempt to find controls and cures for the disease."

The idea for the research agenda workshop was suggested by Andrew von Eschenbach, chief of urology at M.D. Anderson. In an interview, von Eschenbach said the idea came to him as he attended the NPCC founding meeting near Dallas earlier this summer (**The Cancer Letter**, Aug. 16).

Since the coalition had made a decision to seek funds for prostate cancer research, it became appropriate for cancer centers like M.D. Anderson to help the advocates pinpoint opportunities for investment, von Eschenbach said to **The Cancer Letter**.

"More and more players are stepping into this arena," von Eschenbach said. "I would like this effort to serve as the catalyst for a national action plan on prostate cancer."

While the M.D. Anderson conference focused exclusively on research, subsequent meetings would need to address access to health care and other public health issues, von Eschenbach said.

The NPCC workshop, chaired by von Eschenbach, and planned by a committee that included Johns Hopkins scientist Donald Coffey and ACS Executive Vice President Harmon Eyre, was attended by 75 people, about a third of them advocates.

Howe, who also served on the planning committee, said he was encouraged by observing scientists, clinicians and patient advocates design a research plan in just 24 hours.

"We didn't have to stretch to come up with an exciting list of projects that are going to help a lot of people," Howe said. "Those ideas were rattling around in the minds of the people who were there. You just needed to get them in the same place."

The excerpted text of the NPCC research agenda follows:

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Etiology and Prevention

1. Establish repositories. A network of repositories is needed for the collection, characterization, and distribution of epidemiologic, clinical, serologic, pathologic, and outcomes data and material. An advisory board would regulate the best use of these data and tissues. The epidemiologic database would be designed to accommodate the development of future factors or methodologies requiring investigation, thus supplying the information to answer questions relating to familial and racial differences, dietary habits, and genetic issues, as they apply to prostate cancer.

2. Develop animal and in vitro models. The NCI efforts in conducting human trials are substantial and their duplication is not warranted. What is needed is the development of models to accelerate the understanding mechanisms of prostate cancer carcinogenesis and developing strategies for prevention. The only known relevant model of prostate cancer carcinogenesis is a dog model, which is not well characterized. Canines are the only species, besides humans, that develop spontaneous prostate cancer. The attractiveness of the dog model is that careful evaluation of the pedigree would have genetic implications, and may address environmental and dietary variables. Initial studies in the development of the model would include epidemiologic surveys of veterinary schools to define the frequency of canine prostate cancer and species most affected by the disease.

3. Evaluate dietary impact on etiology and prevention. Multidisciplinary, multifactorial, prospective and retrospective studies in humans and model systems should be performed to explore dietary factors and identify their role both as a causative and preventive agent in prostate cancer deserves study.

4. Perform collaborative epidemiologic studies. The incidence and virulence of prostate cancer in the US, the Far East, and Africa is widely divergent. Studies would compare epigenetic and dietary influences on prostate carcinogenesis and progression.

5. Establish education programs for prevention. Grants need to be established to teach primary and secondary prevention for physicians, patients, and the public. Also included would be preparative education for health care systems for future discoveries and education regarding ethical issues connected to genetic testing and counseling.

6. Perform aging studies. Longitudinal aging studies have been conducted in at least three centers in the US. Until recently, information collected was not sufficient to perform strong prostate cancer research. These databases are adding all prostate-related information, thus providing a powerful adjuvant to existing epidemiologic and serum collections. These sources should be utilized and studied with respect to the etiology of prostate cancer.

Diagnosis and Prognosis

1. Establish and validate universal practice parameters. The single most significant need in the treatment of prostate cancer is the establishment and validation of practice parameters that can be consistently applied to individual patients. No consensus exists on (1) the specificity and sensitivity of markers and (2) the definition of biochemical or clinical failure after definitive treatment. Even the assays used to measure PSA demonstrate intra-institutional variability. New analytical parameters have been put forth the stratification of risk. These parameters await further study and validation. Example: molecular staging utilizing the reverse transcriptase-polymerase chain reaction (RT-PCR) to detect circulating cancer cells in the peripheral blood. Initial results from studies using this technique are contradictory among different institutions and the biologic relevance of the assay is in question. Another example is the use of neuroendocrine markers in predicting prostate cancer progression. These markers have been reported as markers of virulence, yet their use as a prognostic parameter has not gained widespread utility in the clinic. We need to provide an avenue that allows for the rapid integration of developing technologies that allow rigorous prospective analysis to validate initial observations. We need to conduct multi-institutional, multifactorial, statistically rigorous, flexible studies which would also account for confounding variables including race and age.

2. Develop assays for measuring genetic or serum markers that stratify risk. Identification of high risk populations and risk factors for these groups is currently not feasible. No measurable variables predict who will get prostate cancer, nor what the clinical behavior of that cancer will be. Screening is an issue that pits cost-effective provision of health

care against the realization that prostate cancer can be cured only if it is detected early. Through the development of assays that can detect genetic markers of prostate cancer susceptibility, premalignant change, and organ-specific serum markers of cancer, we can potentially limit the application of screening techniques to clearly delineated high-risk populations.

3. Establish novel modalities to detect and stage prostate cancer. We need to validate markers that assess the biologic impact of a given tumor, and advocate their routine use as determinants of outcome in the clinical evaluation of patients. Current methods used to evaluate patients are not sensitive or specific enough. PSA has facilitated a significant increase in the detection of prostate cancer. We are finding more cancers, but it is unknown whether therapy has significantly impacted on the natural history of the disease. Even with early detection, staging often underestimates tumor extent and anywhere from 40-60% of patients have pathologic upstaging of their disease at the time of radical prostatectomy. New imaging methodologies are needed to stratify patients for therapy and identify patients most at risk for a poor outcome. New technologies should include development and refinement of static as well as functional imaging of the prostate gland and potential metastatic foci. Technologies that use metabolic or physiologic parameters to exploit differences between cancer cells and normal cells should be evaluated for use in the diagnosis and staging of prostate cancer.

4. Develop markers of cancer virulence. Clinicians primarily use PSA, age, rectal examination finding, Gleason Grade, and patient co-morbidities to stratify patients for therapy. The application of this paradigm has had no impact on overall patient survival in prostate cancer. We need to develop strategies or prognostic indices that will improve our ability to apply an appropriate therapy to an individual patient. Molecular markers of virulence or progression will allow physicians to make those distinctions in a logical fashion. Also, markers could address the issue of what constitutes a clinically insignificant cancer. Several investigators have identified potential markers of progression, such a bcl-2 expression and neuroendocrine differentiation.

5. Define the significance of prostate tumor cell heterogeneity. The concept of tumor cell heterogeneity cannot be completely addressed by prostate biopsy, because it cannot be ascertained whether biopsy specimens truly reflect the spectrum of disease in the entire prostate gland. Prostate cancer is multifocal in origins within the prostate gland. The development of prospective, qualitative, and quantitative assays that can measure and apply relevance to tumor cell heterogeneity should be a major focus of prostate cancer research. These assays could represent a synthesis of functional imaging techniques as well as an assessment of a combination of histologic and serum biomarkers.

6. Study the role of the host response in prostate cancer. Little is known about the role of the immune response, the endocrine milieu, inflammatory responses, stromal factors, or vascular factors in prostate cancer carcinogenesis and progression. These and other markers of host response should be correlated with patient outcome to determine their relevance in prostate cancer biology.

Therapeutics, Local Therapy

1. Defining risk. The development of biomarkers and imaging modalities which allow patients to be stratified by risk of progression, should receive the highest priority in the research agenda. These advances would allow assessment of the biologic potential of disease prior to therapy and could detect early treatment failures.

2. Application of optimum local therapy based on risk. Low risk patients will likely have maximum benefit with minimal associated morbidity from single-agent treatment. For patients in high-risk groups, there are several attractive combined treatment modalities that warrant further investigation. To validate new modalities or multi-modal approaches, we need to establish large scale, multi-institutional clinical trials, that evaluate cancer control as a primary concern, but also address issues of morbidity and quality of life.

3. Define treatment failure. Treatment failure is defined based upon the serologic, pathologic, or radiologic evidence of residual or progressive disease following a given therapeutic regimen. The implications of pathologic evidence of capsular involvement or capsular penetration with tumor as well as a margin positive surgical specimen and specimen confined disease, need to be fully elucidated through multi-institutional longitudinal studies. This will provide for a national consensus on one aspect of treatment associated failures. Multi-institutional studies are needed to delineate

the most efficacious timing of intervention in individuals with evidence of stage and treatment modality specific biochemical failure. The efficacy of new biomarkers capable of detecting residual or recurrent disease will need to be validated prior to implementation into clinical practice.

Therapeutics, Systemic Therapy

1. Prioritize a research strategy for the development of promising systemic therapies. Nine areas of systemic therapy research which show considerable promise are identified. A scoring priority is assigned to each area based on the following parameters: 1) Applicability: defined as the probability of being applied in patient care in the near future. 2) Level of interest: defined as the relative likelihood of the therapy to ultimately impact the outcome of patients with prostate cancer and its worthiness of future development. 3) Development: defined as the need for investment in development. The nine areas include: signal transduction pathways/anti-growth factor therapy, angiogenesis inhibitors, organ-specific (bone) therapy, gene therapy, immunotherapy, anti-invasion/anti-metastatic therapy, androgen receptor/ androgen response pathways, cytotoxic therapy, and differentiation therapy. Within each of these categories of therapeutic intervention, there are a variety of currently available agents which must be meticulously evaluated. Of greater urgency is the need to promote novel and more effective prostate specific reagents.

2. Develop accurate markers of minimal residual disease. The development of markers to measure minimal residual disease and disease progression could lead to early intervention and potential cure. Markers should convey a measure of tumor burden and virulence. It may also be possible to identify patient subsets with clinically localized disease but at risk for progression.

3. Identify and evaluate alternative therapies. Alternative therapies hold great promise for discovery, as anecdotal experiences with diet, herbal therapies, etc., suggest biologically active natural substances which require rigorous scientific exploration. Large numbers of patients are turning to alternative therapies. The use of such agents in an uncontrolled manner may confound the results of standardized clinical trials and result in the loss of untested therapeutic leads. A registry to catalogue the use of such agents should be instituted to assess both efficacy and toxicity.

Tumor Cell Biology and Progression

1. Develop studies that are prostate cancer specific. Since prostate cancer is unique in many ways, we must resolve tumor specificity at a tissue and cellular level. Studies show that the growth factor receptors on the cell surface are unique to different organs, including the prostate, by the mechanism of alternate gene splicing. Molecular specificity for the prostate is an ideal target for therapy. We need to understand how this alternate splicing is regulated in the prostate. With cancer cell invasion and metastasis, prostate cancer cells are able to detach from extracellular matrix integrins and separate from neighboring cells in the prostate. As a consequence, the cells become free to migrate out of the prostate and to form invasive and metastatic lesions. We need further studies to understand the molecular biology of cancer cell interaction with cell adhesion and integrin molecules in the prostate.

2. Metastasis: the relationship between tumor cells and the bone environment. The primary landing sites for prostate cancer metastases are the pelvic lymph nodes and the bones of the axial skeleton. To understand this ÒhomingÓ specificity, several investigators have demonstrated the importance of a synergistic interaction established between tumor cells and bone cells. Tumor cells release paracrine factors that stimulate bone growth to form osteoblastic lesions. Conversely, the bone cells release growth factors that stimulate tumor cell growth. Many of the secretions released by the prostate tumor cells are thought to play a role in producing the pain associated with boney metastasis, which is characteristically difficult to alleviate by conventional means. We urgently need more studies at the biological, cellular, and molecular levels regarding tumor cell-bone interaction. Delineation of this complex relationship will allow the development of novel therapies that specifically target the control of the osseous environment as a metastatic site, and may also provide insights into novel forms of pain management for bone metastases.

3. The role of angiogenesis in prostate cancer. Preliminary studies show that the endothelial cells lining the blood vessels in the normal prostate are

themselves tissue specific. When prostate cancer develops, the endothelial cell becomes tumor-specific. We need to understand the mechanisms of angiogenesis in prostate cancer biology. If understood sufficiently, these angiogenesis pathways could be exploited as novel therapeutic targets.

4. Develop models of prostate cancer progression. To understand prostate cancer cell carcinogenesis and progression, we need to develop and study relevant models that accurately reflect the natural history of the process in humans. Recently, a new animal transgenic model has been developed. By placing a prostate tissue specific gene promoter in front of a cancer gene and introducing it into a transgenic animal, it is possible to follow the early stages of tumor development and to study what genes might activate or retard specific steps in tumor progression. The specific cell fate in tumor progression can be resolved in this model system. In addition, powerful new molecular techniques of studying differential gene expression between normal tissues and tumors with different metastatic potential or site specificity should provide new insight into the specific molecular steps in progression. The identified genes associated with metastasis in human prostate cancer could then be tested in the transgenic model to elucidate their biological mechanisms and properties.

Genomic instability of prostate cancer is responsible for the production and selection of virulent and highly resistant clones that demonstrate a high propensity for invasion and metastasis. Alteration in DNA methylation patterns is an early and common event seen in prostate carcinogenesis that deserves further study. The role of telomerase and telomere shortening is an exciting area of prostate cancer research that may provide clues to understanding cell transformation and metastatic progression.

5. The biology of the androgen receptor. Androgen activity has long been observed to be a requirement for the development of prostate cancer. New molecular insights are now being made into androgen receptor biology. A section of the human androgen receptor has a domain of variable repeats. Recent experiments show that the shorter the glutamine repeats are in length, the more androgenic is each receptor molecule in its biology. Racial studies of the length of these glutamine repeats reveal that African-American males, who have the highest incidence of prostate cancer, have the shortest repeats; Caucasians rank next; and Orientals have the longest glutamine repeats and the lowest incidence of prostate cancer. Such molecular modulation of the androgen receptor may permit it to serve as an enhanced tumor promoter. These data highlight our need to further study the modulation of the androgen receptor in prostate cancer, as it appears to play a critical and central role that is unique to prostate cancer biology.

6. Pathways of androgen independent growth. Following androgen ablation, hormone resistant clones are selected which ultimately progress and can kill the patient. The fact that there currently is no effective therapy for androgen independent prostate cancer highlights how little is known about the emergence of the androgen independent phenotype. It is essential to understand this process of androgen resistance. Recently, it was discovered that a gene product bcl-2 is expressed in androgen resistant cells. Bcl-2 is a protein regulatory factor that protects the prostate cancer cells in an androgen deprived state from undergoing cell death or apoptosis. The androgen receptor itself might also support prostate cancer growth even in the absence of androgens. The androgen receptor appears to dimerize and form an additional regulatory protein complex through interaction with a co-activator protein. Changes in the co-activator protein can produce aberrant activation of the androgen receptor and the receptor can even function in the absence of androgens, or more importantly, may be activated rather than blocked by anti-androgens. These and other preliminary observations deserve further study as possible mechanisms of androgen resistant cell growth.

7. Define the importance of the host-tumor cell interaction. There are numerous host-tumor interactions of tremendous potential that are now starting to be resolved, and warrant further investigation. For example, it is possible to activate or enhance the host immune system to attack tumor cells.

Transfecting genes for cytokines or antigens into tumor cells has produced a genetically engineered vaccine approach. The lymphocytes from these treated patients are now being used to identify and isolate tumor specific prostate antigens.

Prostate immunology is ripe for important studies, in the normal, tumor and metastatic settings.

The role of the Major Histocompatability Complex Type 1 (MHC-1) expression, dendritic cells, and anergy are all important immunological frontiers that need to be resolved in prostate cancer. Immunotherapy, especially through the use of cancer ÒvaccinesÓ, could be developed as a therapeutic strategy to "mop up" residual cancer cells after therapy, or eliminate the "unneeded prostate" altogether.

Outcomes Research

1. Outcomes research in prostate cancer screening. Existing databases are representative of patients diagnosed with prostate cancer. Thus, they are of limited usefulness in dealing with the question of outcomes research in screening. A survey of the current practice of physicians to determine their compliance with screening guidelines and the patient awareness of these guidelines is needed. In addition, studies are needed to understand the impact of geography, physician bias, payer mix and managed care on the decision to screen patients.

2. Outcomes research in therapy for clinically localized prostate cancer. Existing databases must be probed and information applied to the question of outcome of local treatment and treatment failure. It is critical to develop a consensus on definitions of outcome to eliminate disparities in studies. One can then utilize these definitions and databases in studies to assess the comprehensive impact of treatment failure. Further, we need to assess the impact of managed care in selection and allocation of treatment options and patient follow-up protocols.

3. Outcomes research in advanced prostate cancer. To understand the effect of end-stage disease on patients, studies must be performed that address several questions: What is the burden of care in advanced prostate cancer relative to perceived patient benefit? What is the impact of advanced disease on lost productivity for both the patient and his family? What are the financial costs to the patient, family, and society, in the treatment of advanced disease? What compromises in quality of life are made with and without treatment intervention? What are the barriers to therapy for advanced disease in terms of access, expense, and the role of managed care?

4. Outcomes research in prostate cancer education. We need to investigate the role of prostate cancer education in overcoming racial, ethnic, educational, and cultural biases that interfere with prostate cancer diagnosis and therapy. Also, we need to evaluate methods of education and distribution of information and identify effective media that convey important information to high risk target groups as well as to assess the impact of new technologies such as the Internet in providing patient and physician education. Finally, as patient advocacy and support groups become more prominent, we need to evaluate their optimal methodologies and their effectiveness to assist patients and their families in coping with the financial, social, and psychological impact of prostate cancer.

Behavioral, Psychosocial, Quality of Life Issues

1. Define and evaluate quality of life issues that impact on diagnostic/treatment choices. We do not have an understanding of what decision making processes are involved in deciding whether or not to participate in prostate cancer screening, or how men choose between the options of different local and systemic therapies for their disease. Physician bias has a role, but there are other patient derived issues at play that are poorly understood. With the development of deferred therapy or Òwatchful waitingÓ as a potential therapy option, we need to assess the impact of Òdoing nothingÓ on the patientÕs level of stress, coping mechanisms, and risk for depression.

2. Evaluate the impact of disease and treatment related morbidity on quality of life. Standardized national surveys of incontinence and impotence following therapy will help define the true scope of these treatment related complications. Also, we need to assess the impact of available modalities used to treat these complications. With increasing utilization of androgen ablation in younger patients, at earlier points in the course of the disease, the long term biologic consequences of this intervention require investigation. Long term effects of osteoporosis, anemia, loss of muscle mass, increased body fat, and ÒfeminizationÓ psychologic changes require investigation and intervention.

3. Pain control and quality of life. The most important issue in the quality of life of a cancer patient is the concept of pain control. A better understanding of the biology of prostate cancer pain, particularly the physiology of bone pain, will enable us to direct effective therapies that may include a combination of conventional analgesics and holistic

approaches. We need to explore novel approaches to pain control in patients with advanced disease.

4. Evaluate the impact of prostate cancer on the quality of life of patient families. The family members of men with prostate cancer often act as primary caregivers and are intimately associated with the consequences of prostate cancer progression. Their needs and concerns should be identified and addressed.

Voluntary Organizations

Myles Cunningham Elected President Of Cancer Society

Myles Cunningham, clinical associate professor of surgery, University of Illinois College of Medicine, was elected president of the American Cancer Society at the annual meeting of the society's board of directors, held Nov. 3 in Orlando, FL.

Cunningham served as chairman of the ACS Medical Affairs Committee and is a past president of the Illinois Division. He succeeds Raymond Lenhard Jr., professor of oncology and medicine, Johns Hopkins University School of Medicine.

George Dessart, professor and deputy chairman for graduate studies in television and radio at Brooklyn College of the City University of New York, was re-elected chairman of the board.

David Rosenthal, director of Harvard University Health Services and professor of medicine at Brigham & Women's Hospital, was elected vice president and president-elect.

Jennie Cook, an accountant from Larkspur, CA, was re-elected as chair-elect.

Francis Coolidge, an attorney from Boston, MA, was re-elected as vice-chair.

Charles McDonald, chairman of the department of dermatology, Brown University, was elected chairman of the medical affairs committee.

Gerald Woolman, clinical professor, Department of Surgery, Texas Tech. University School of Medicine, was elected vice-chair of the medical affairs committee.

Lay officers include treasurer John Baity, of New York City, and secretary John Kelly, of Gulfport, MS.

ACS Awards Presented

ACS presented its Medal of Honor Awards to FDA Commissioner David Kessler, medical oncology pioneer B.J. Kennedy, and scientist Janet Rowley.

Kessler was honored for "his courageous and historic efforts to protect today's children and those in future generations from the dangers of tobacco;" and for his work on issues including the public health consequences of nicotine addiction, nutrition labels on food products, faster approval of cancer drugs, and quality standards for mammography equipment, facilities and personnel.

Kennedy, Regent's Professor of Medicine Emeritus and Emeritus Masonic Professor of Oncology, University of Minnesota Medical School, received the Clinical Research Award for "his pioneering efforts in establishing medical oncology as a subspecialty of internal medicine," as well as his clinical research, teaching and patient care.

Rowley, Blum-Riese Distinguished Service Professor, Department of Medicine and Department of Molecular Genetics and Cell Biology, University of Chicago, received the Basic Research Award for "conducting groundbreaking research that established her as a world leader in cancer genetics."

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ACS presented its Humanitarian Award to Susan Mellette, of the Medical College of Virginia, for her significant contributions to the society's service and rehabilitation program. Mellette established a model cancer rehabilitation program at MCV and developed home hospice care programs.

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The society presented its Distinguished Service Award to three leaders in cancer control: Marion Morra, John Lewis Young Jr., and Michael Moore.

Morra, a volunteer for the Connecticut Division for more than 20 years, was cited for her contributions to cancer patients through her writing and production of booklets for health professionals and the public on cancer topics.

Young, internationally known in the field of epidemiology, was cited for his role in development of standards for population-based registries.

Moore, the Mississippi attorney general, was honored for his anti-tobacco leadership. Under his direction, Mississippi was the first state to sue 22 tobacco companies and distributors on behalf of taxpayers to recover Medicaid expenses used to treat patients who developed illnesses caused by tobacco.

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The ACS Volunteer Leadership Award was presented to Edwina Thorn, of Hudson, NY, and John Lynch, of Washington, DC.