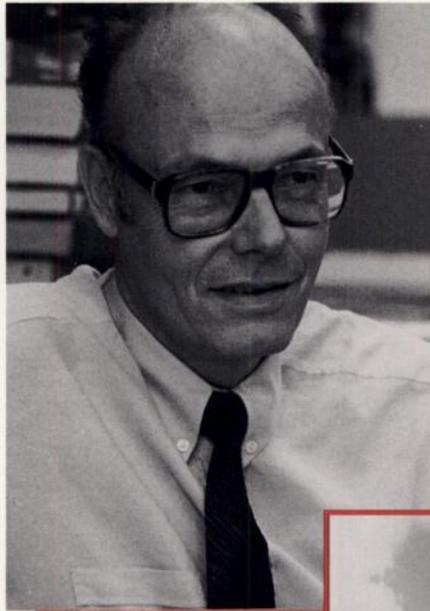


Cancer Research

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AMERICAN ASSOCIATION FOR CANCER RESEARCH SCIENTIFIC CONFERENCES

MARCH 7-12, 1997 ***Basic and Clinical Aspects of Breast Cancer***

Chairpersons: J. Carl Barrett, Research Triangle
Park, NC; Karen S.H. Antman, New York, NY;
Mary-Claire King, Seattle, WA
Keystone Resort, Keystone, CO

APRIL 12-16, 1997 ***88th Annual Meeting***

Chairperson: Frederick P. Li, Boston, MA
Co-Chairpersons: Clara Derber Bloomfield,
Buffalo, NY; Stephen H. Friend, Seattle, WA
San Diego Convention Center, San Diego, CA

JUNE 7-11, 1997 ***Cancer of the Central Nervous System***

Conference with Neurosurgery Joint Section on
Tumors
Chairpersons: Peter McL. Black, Boston, MA;
Webster K. Cavenee, La Jolla, CA
Loew's Coronado Bay Resort, San Diego, CA

SEPTEMBER 9-13, 1997 ***Molecular Genetics of Cancer***

Conference with the European Association for
Cancer Research
Chairpersons: Eric J. Stanbridge, Irvine, CA;
Walter Bodmer, Oxford, England
Hertford College, Oxford, England

SEPTEMBER 26-30, 1997 ***Tumor Suppressor Genes***

Chairpersons: Stephen H. Friend, Seattle, WA;
Philip Branton, Montreal, Quebec, Canada
Victoria Conference Centre, Victoria, BC, Canada

OCTOBER 17-21, 1997 ***Gene Regulation in Differentiation and Development***

Chairpersons: Robert Eisenman, Seattle, WA;
Elaine V. Fuchs, Chicago, IL
The Sagamore Resort, Bolton Landing (Lake
George), NY

DECEMBER 12-16, 1997 ***Gene Silencing in Cancer: The Epigenetics of Neoplasia***

Chairperson: Peter A. Jones, Los Angeles, CA
El Conquistador Resort and Country Club, Las
Croabas, PR

JANUARY 1998 ***Angiogenesis and Cancer***

Chairpersons: Judah Folkman, Boston, MA;
Michael Klagsbrun, Boston, MA
Location in Orlando, FL to be Announced

FEBRUARY 16-21, 1998 ***Innovative Molecular Biology Approaches to the Prevention, Diagnosis, and Therapy of Cancer***

Joint Meeting with the Japanese Cancer
Association
Chairpersons: Edward Bresnick, Worcester, MA;
Kaoru Abe, Tokyo, Japan
Maui Marriott Resort, Maui, HI

AACR members will receive brochures on the
above conferences as soon as they are available.
Nonmembers should call or write:

American Association for Cancer Research
Public Ledger Building, Suite 816
150 South Independence Mall West
Philadelphia, PA 19106-3483
215-440-9300 • 215-440-9313 (FAX)
E-Mail: aacr@aacr.org

For regular updates to this list visit the AACR's
Website, <http://www.aacr.org>



**LATE-BREAKING RESEARCH SESSION
AT THE AACR ANNUAL MEETING
Tuesday, April 15, 1997**

Time has been set aside for the presentation of 4-5 definitive reports of highly significant and timely findings in the field. Criteria for the selection of these presentations and instructions for submission of abstracts are as follows:

INSTRUCTIONS FOR SUBMISSION OF LATE-BREAKING ABSTRACTS

1. The work to be presented must be of major novelty and significance, *e.g.*, the characterization of a new gene in familial cancer or the discovery of a new diagnostic marker, and should not have been previously published in a peer-reviewed scientific journal or presented at a national meeting.
2. The abstract must be sponsored by an AACR member in good standing (dues paid for 1997).
3. Each member in good standing may sponsor only **one** abstract for this session whether or not he or she sponsored an abstract last November for the regular annual meeting program. If an associate member is the **sponsor**, the abstract must also be **endorsed** by an active or corresponding member in good standing. In this case, the **endorser** does **not** forfeit the opportunity to **sponsor** a late-breaking abstract.
4. Abstracts must be typed on **one** side of **one** sheet of white paper.
5. All text on the page must fit within an area 6 1/2" wide and 9" high (16.5 cm X 22.9 cm) with margins of at least 1" (2.5 cm) on the top, bottom, and sides of the page.
6. Each abstract must be accompanied by a covering letter from the sponsor explaining why the work is novel and significant enough to be considered for this late-breaking research session and certifying that the findings became available **after** the annual meeting abstract deadline of November 12, 1996. This letter must contain the sponsor's complete mailing address, FAX number, and E-mail address (if available) so that we can communicate the scheduling decision of the Program Committee.
7. Abstracts and covering letters must be received in the AACR Office by 5:00 p.m. Eastern Time on **March 7, 1997**. FAX transmissions are **not** acceptable. Carrying envelopes should be clearly marked "Late-Breaking Abstract," and should be addressed to American Association for Cancer Research, Public Ledger Building, Suite 816, 150 South Independence Mall West, Philadelphia, PA 19106-3483. If you wish to receive acknowledgment of receipt of your abstract, enclose a self-addressed post card with appropriate postage affixed. Accepted abstracts will not be published since they will be received after the *Proceedings of the American Association for Cancer Research* has been printed; however, they will be distributed at the session in San Diego.
8. A special subcommittee of the Program Committee appointed by President Louise C. Strong will select the papers to be presented. Presenters of accepted papers will be notified via FAX no later than **March 24, 1997**.

SOCIETÀ ITALIANA DEI TUMORI
SECONDA UNIVERSITÀ DEGLI STUDI DI NAPOLI
KIMMEL CANCER INSTITUTE, JEFFERSON MEDICAL COLLEGE
SBARRO INSTITUTE FOR CANCER RESEARCH AND MOLECULAR MEDICINE

present:

First Joint International Conference

“GENE TARGETS FOR CANCER TREATMENT”

June 3-6, 1997 in Capri, Italy

Chairpersons

GIOVAN GIACOMO GIORDANO, M.D., Seconda Università degli Studi di Napoli
CARLO M. CROCE, M.D., Kimmel Cancer Institute, Jefferson Medical College

Environmental and Genetic Factors in Human Cancer
D.E. FISHER, B. HENDERSON, L.A. LOEB, M. TUCKER,
G. THOMPSON, I.B. WEINSTEIN

Oncogenes, Tumor Suppression Genes, The Cell Cycle and Cancer
C. BASILICO, C.M. CROCE, A. GIORDANO, J. SCHLESSINGER, S.I. REED,
G. STEIN, J. SHAY

Molecular Epidemiology
P.J. LANDRIGAN, C. HARRIS, L.L. ADAMS-CAMPBELL

New Trends for Therapeutic Interventions
W.K. HONG, H. VITETTA, R. HERBERMAN

Concluding Round Table Discussion:
Present and Future Strategies in Preclinical and Clinical Treatment

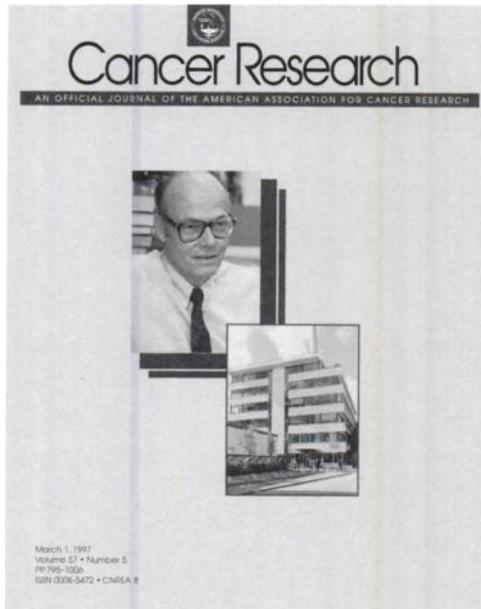
Moderators:
C.M. CROCE and F. CRUCITTI

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Sbarro  **Institute**
Cancer Research Molecular Medicine



In the 1960s, the loss of contact inhibition of the growth and movement of transformed cells was already interpreted as a potential sign of cell surface alteration. However, none of today's sophisticated analytical approaches were available in those days to characterize and pinpoint such membrane changes. Max M. Burger (*cover*), Director of the Friedrich Miescher-Institute (FMI) in Basel, Switzerland, was the first to show that a carbohydrate-specific plant lectin, wheat germ agglutinin (WGA), was able to distinguish poorly agglutinating untransformed cells from well agglutinating transformed or tumor cells (Proc. Natl. Acad. Sci. USA, 57: 359, 1967). Furthermore, a simple proteolytic treatment of normal cells led not only to the same lectin agglutinability as displayed by tumor cells but also to unexpected growth initiation and loss of growth control in tissue culture. Thus, the concept was brought forward that there are not only differences in composition but also differences in the architecture of the surface membrane of normal and virally transformed cells and that these differences lead to changes in growth control (Proc. Natl. Acad. Sci. USA, 62: 994, 1969). The *PNAS* article became a citation classic and led to the discovery of proteases as growth-inducing and invasion factors and later on to the concept of the fluid lipid plasma membrane (G. L. Nicolson and S. J. Singer) and its alteration in tumor cells.

Because sugar-directed lectins in high doses can also kill receptor-positive cells, WGA-resistant B-16 mouse melanoma cells could be selected and, as expected, were found to have a defect in terminal sialic acid of their glycoproteins. Such surface carbohydrate mutant cells turned out to lose their metastatic capabilities. Other lectins which were selected for the reversion of this terminal sialic acid defect restored the metastatic ability of these cells. From thereon, such lectin selections became a powerful tool to study surface carbohydrate function (cell adhesion, migration, receptor function, and signaling), and sialic acid has been increasingly implicated in the metastatic behavior of tumor cells ever since. Surface carbohydrate changes in general have received more attention in the last few years since the discovery of carbohydrate recognizing animal proteins, the so-called selectins, which may not only guide leukocytes to inflammation zones in the vascular system but also tumor cells to target organs.

In the 1980s, Dr. Burger contributed significantly to the concept that the cytoskeleton plays an important role in metastasis with a simple approach. He isolated more deformable cells which were

missing their stiff actin bundles by selecting them through nucleopore filters. Such cells were highly metastatic.

Organ-specific metastasis was traditionally ascribed to homing, i.e., recognition of the target organ by the metastasizing cell. There were indeed good examples of brain- or lung-specific cells which were seeding into the brain or the lung due to specific surface adhesions to brain endothelial or lung cells. Dr. Burger's group was the first to select liver-specific cells from parental lung-specific cells, but they could not find selective absorption by liver. In a series of studies they could, however, demonstrate that after i.v. application these cells spread to all organs of the body but survived and grew preferentially in the liver only (Proc. Natl. Acad. Sci. USA, 85: 7251, 1988). This was confirmed by coculture *in vitro* where contact with liver, i.e., the target organ cell, was necessary to pass on the growth signal (a membrane protein). Thereafter, it was shown by many others that organ-targeted metastasis is either due to organ-specific growth factor or to organ adhesion or to both.

Recently, Dr. Burger and his group have been interested in the possible relevance of the proto-oncogene c-met receptor for metastatic promotion by its HGF/SF ligand to the liver in particular, and they have isolated and cloned a 150 kD protein that may turn out to be a marker for undifferentiated mouse as well as human tumor cells.

Dr. Burger received his M.D. from the University of Zurich in 1959 and his Ph.D. in Biochemistry from Washington University, St. Louis, MO, in 1964. He began his postdoctoral research under Arthur Pardee in 1965 and advanced to the position of Full Professor in the Department of Biochemical Sciences at Princeton University in 1971. From 1972–86, he was Professor of Biochemistry at the University of Basel, where he served as Chairman of the Biocenter from 1972–79. In 1986, he became Director of FMI. Dr. Burger has been a member of the UICC Council since 1978 and of its Executive Council since 1990. And, since 1982, he has been the organizer of the UICC Study Group meetings that take place in Woods Hall, MA. Among his honors are the Selman A. Waksman Medal (1971) and the Otto Naegeli Prize (1975) in addition to his selection as a foreign honorary member of the American Academy of Arts and Sciences in 1988.

FMI (*bottom photo*) is a basic research institute associated with the University of Basel. In 1995, the institute celebrated its 25th anniversary and commemorated the death of Friedrich Miescher, a native son of Basel and the discoverer of DNA, who died in 1895. Primarily supported by Novartis (formerly Ciba), FMI is dedicated not only to cancer research but also to molecular neurobiology, plant molecular biology, and gene regulation. At FMI, there has always been an interest in growth control, particularly signaling, as exhibited in the work carried out there by 100 scientists and 90 graduate students. George Thomas found ribosomal S-6 phosphorylation when protein synthesis is initiated, and he unraveled its signal pathway step by step. Brian Hemmings cloned some of the most relevant protein-phosphatases, found RAC, and codiscovered the pleckstrin homology domain. Extracellular matrix tenascin was discovered by Ruth Chiquet, and Nancy E. Hynes is working on membrane receptor-induced differentiation and growth control of mammary cells. Other groups are studying plant (Thomas Hohn) and animal tumor viruses (polyoma, Kurt Ballmer-Hofer), as well as cell cycle control (Wilhelm Krek).

Finally, the American Association for Cancer Research (AACR) is indebted to Dr. Burger for his role as a major driving force in the development of the very successful March 1994 AACR Special Conference, "Growth Factors, Development, and Cancer." This meeting, which took place in Interlaken, Switzerland, was a joint conference of the AACR and FMI and was chaired by Harold L. Moses and Bernd Groner.