

March 1, 1991

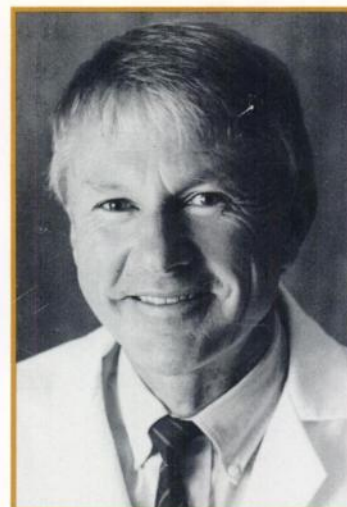
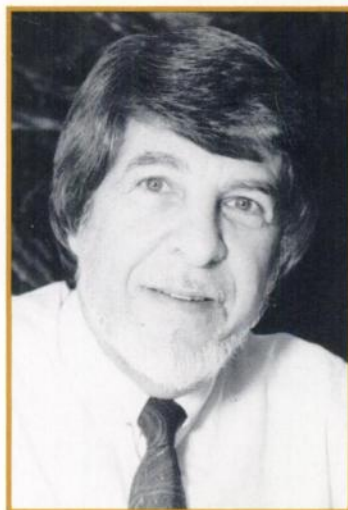
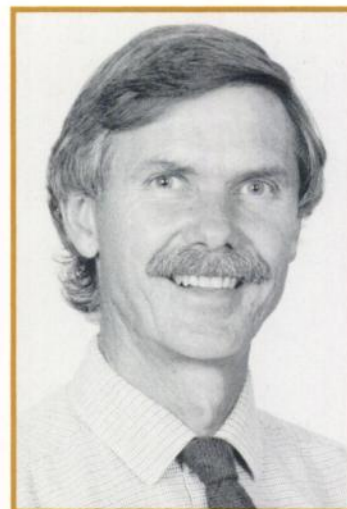
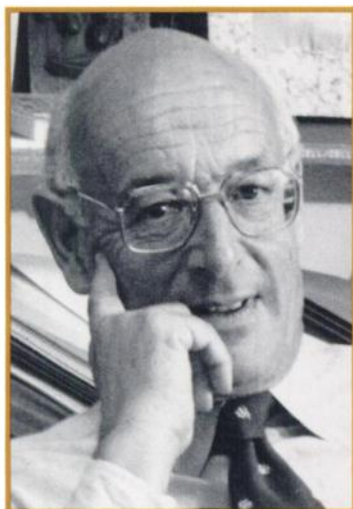


Cancer Research

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50
Years of Publication

**THE NATIONAL
NEUROFIBROMATOSIS
FOUNDATION**

announces the availability of

**YOUNG INVESTIGATOR
AWARDS**

which will provide salary support not
to exceed \$35,000 annually for
period up to two years

and

RESEARCH GRANTS

which will provide up to \$50,000 for
up to two years for research on the
cause and treatment of
neurofibromatosis 1 and 2

The deadline for filing applications
for funding beginning July 1, 1991,
is April 2, 1991.

For information on applications
please contact:

The National
Neurofibromatosis Foundation
141 Fifth Avenue, Suite 7-S
New York, New York 10010

212 - 460 - 8980

1 - 800 - 323 - 7938

Fax # 212 - 529 - 6094

**The Hayashibara International
Cancer Research Fellowship**

The Hayashibara Foundation, a nonprofit-making organization within the Hayashibara Group, announces a fellowship program.

Fellowship will be taken up at the Fujisaki Cell Center which is devoted to basic and applied research related to the problems in human cancer. The fellowship is normally made for 1 year and is renewable for up to 5 years. The fellowship will be expected to commence within 6 months of the announcement of the awards.

No fixed deadline for application is set throughout the year. The awards are for high quality research work in one of the three categories described below.

1. Fundamental Leukemia-Lymphoma Research
2. Cytokine-Lymphokine Research
3. Hematopoietic Cell Lines

Qualified person who is holding a Ph.D., M.D., or equivalent qualifications should make inquiry for additional information and application forms from:

Jun Minowada, M.D.
Director, Fujisaki Cell Center
675-1
Fujisaki, Okayama 702
JAPAN

STAFF SCIENTIST

Experimental Therapeutics Division

**THE ONTARIO CANCER INSTITUTE/
THE PRINCESS MARGARET HOSPITAL**

Applications are invited for the position of Staff Scientist to join a group of investigators who are involved in experimental studies relating to cancer treatment including experimental radiotherapeutics, experimental chemotherapy, hyperthermia, radiobiology, metastases and DNA repair. Experimental Therapeutics is a Division of the Research Department of OC/PMH which currently has 30 full-time staff scientists in five research divisions and has a graduate student training program involving about 75 students associated with the Department of Medical Biophysics of the University of Toronto.

The successful candidate will have an interest in the above experimental studies and possess expertise in molecular biology. The candidate will be expected to establish a research program within the Experimental Therapeutics Division and attract peer-reviewed research funds. This position will involve appointment to the Senior Scientific Staff of the Institute and candidates should be eligible for appointment to the Department of Medical Biophysics at the level of Assistant Professor.

Persons interested in this position should apply in writing to: Dr. G.F. Whitmore, Head, Experimental Therapeutics Division, Research Department, The Ontario Cancer Institute, 500 Sherbourne Street, Toronto, Ontario M4X 1K9, enclosing curriculum vitae, a statement of their expertise, current interests, proposed research program, and the names of three referees willing to provide references.

In accordance with the Canadian Immigration requirements, this advertisement is directed to Canadian citizens and permanent residents. In accordance with the Employment Equity Policy of the University of Toronto and The Ontario Cancer Institute, applications are invited from all qualified women and men.



THE

Ontario Cancer

· INSTITUTE ·



AACR SPECIAL CONFERENCE IN CANCER RESEARCH

Membrane Transport in Multidrug Resistance, Development, and Disease

co-sponsored by the National Cancer Institute of Canada

March 10-14, 1991
Banff Centre, Banff, Alberta, Canada

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Information and Application Forms

American Association for Cancer Research
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Sixth and Chestnut Streets
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215-440-9300 215-440-9313 (FAX)

The Cystic Fibrosis Gene and Its Product

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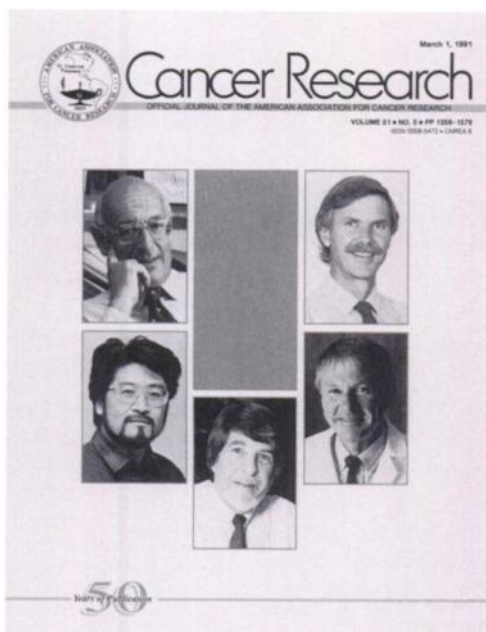
Permease Systems in Bacteria and Eukaryotes

ERNEST M. WRIGHT / Los Angeles, CA
GIOVANNA F.-L. AMES / Berkeley, CA
H. RONALD KABACK / Los Angeles, CA

All places at this conference
have been filled.

The AACR and the National Cancer Institute of Canada acknowledge the generous support of the Alberta Heritage Foundation for Medical Research.

COVER LEGEND



Lowenstein and Kanno discovered that epithelial cells can communicate by transfer of molecules of less than 1200 daltons across junctions of cell membranes [Nature (Lond.), 201: 194, 1964; Science (Washington DC), 143: 959, 1964; J. Cell Biol., 22: 565, 1964; Science (Washington DC), 213: 551, 1981] and proposed that such transfers were involved in growth regulation [Ann. NY Acad. Sci., 137: 441, 1966; Physiol. Rev., 61: 829, 1980]. This hypothesis predicted that disruption of intercellular communication might lead to uncontrolled growth and that normal cells could control growth of neoplastic cells through cytoplasmic signal transfer. Indeed, neoplastic cells deficient in intercellular communication displayed a high degree of malignancy (Cell, 48: 725, 1987).

Murray (Cancer Res., 43: 3614, 1983) and, independently, Trosko (Cell Biol. Toxicol., 5: 27, 1989), through techniques like microinjection of dyes or fluorescence photobleaching, demonstrated that promoters such as phorbol esters could down regulate or

disrupt gap junction function, key knowledge bearing on the mechanism of action of promoters. Bertram (Cancer Res., 45: 1946, 1985) observed growth control through gap junctions in cell culture and noted that retinoids could up regulate signal transfer through gap junctions, partially explaining inhibition of promotion by retinoids. Yamasaki (Cancer Res., 48: 3203–3207, 1988) found selective intercellular communication between transformed and normal fibroblasts and suppression of the transformed phenotype through neighboring normal cells. The regulation of the function of gap junctions appears under the influence of specific oncogenes, in particular *Ha-ras* and *src* (Ann. NY Acad. Sci., 551: 337, 1988; Mol. Carcinog., 2: 131, 1989). Cells treated with carcinogens do not display a neoplastic character when communicating with normal cells [Carcinogenesis (Lond.), 10: 1743, 1989]. A recent book [E.L. Hertzberg and R.L. Johnson (eds.), Gap Junctions. New York: Alan R. Liss, Inc., 1988] provides an overview.

These findings emphasize a key role of intercellular communication via transfer of signal molecules across gap junctions in the regulation of cell growth and the expression of the neoplastic phenotype.

Werner R. Lowenstein, *upper left*, is Chairman of the Department of Physiology and Biophysics, University of Miami School of Medicine; Andrew W. Murray, *upper right*, is Professor of Biological Sciences, The Flinders University at Bedford Park, South Australia; James E. Trosko, *center*, is Professor at the Department of Pediatrics and Human Development, Michigan State University, East Lansing, MI; Hiroshi Yamasaki, *lower left*, is Head, Unit of Mechanisms of Carcinogenesis, International Agency for Research in Carcinogenesis, International Agency for Research in Cancer, Lyon, France; and John S. Bertram, *lower right*, is Director, Basic Science Program, Cancer Research Center of Hawaii, Honolulu.

We are indebted to all mentioned, especially Dr. Trosko, for information and photos.

John H. Weisburger