Contents

1319  
E Pluribus Unum: Presidential Address.  
James F. Holland.

1330  
Proliferative Kinetics of Human Hematopoietic  
Cells during Different Growth Phases in Vitro.  
Akio Todo, Annabel Strife, Jerrold Fried, and  
Bayard D. Clarkson.

1341  
Kinetic, Immunoc'iemical, and Physical Studies  
on Purified Rat Liver Adenosine 5'-Phosphate  
Deaminase after Induction with 3'-Methyl-4- 
dimethylaminoazobenzene or Thioacetamide.  
Larry D. Smith and Donald E. Kizer.

1348  
Hormonal Effects on Thymidine Kinase Ac  
tivity in Normal Rat Adrenal and in Hormone- 
dependent Adrenal Carcinoma.  
M. R. Garland, Therese Ng, and J. F. Richards.

1355  
Erythropoietic Effect of Plasma from Patients  
with Advanced Cancer.  
Dincer Firat and Jose Banzon.

1360  
Metabolic Studies on Mammary Tumor MTW9  
following Resection of the Mammosomatotrop  
Tumor MtTw5.  
Shibabrata Biswas and Vincent P. Hollander.

1364  
Identity of Corticosteroid Binder I with the  
Macromolecule Binding 3-Methylcholanthrene  
in Liver Cytosol in Vivo.  
S. Singer and Gerald Litwack.

1369  
The Anionic Nature of Sarcoma 180 Cell Sur- 
faces, and Sensitivity to 4,4'-Diacetyldiphenyl- 
urea-bis(guanylhydrazone).  
L. Weiss and M. T. Hakala.

1373  
Pulmonary Fibroblastomas in a Deer with  
Cutaneous Fibromatosis.  
Loren D. Koller and Carl Olson.

1376  
Enzymatic Phosphorylation of 1-ß-D-Arabinofu- 
ranosylcytosine.  
Yoshio Kozai and Yukio Sugino.

1383  
Treatment of Moloney Virus-induced Leukemia  
with Cyclophosphamide and Specifically Sensi- 
tized Allogeneic Cells.  
John P. Glynn and Meir Kende.

1389  
DNA Synthesis and the Effect of Sucrose in  
Nuclei of Host Liver and Morris Hepatomas.  
P. Ove, M. L. Coetzee, and H. P. Morris.

1396  
Regulation of the Rate of Sterol Synthesis and  
the Level of ß-Hydroxy-ß-methylglutaryl Co-  
enzyme A Reductase Activity in Mouse Liver  
and Hepatomas.  
A. A. Kandutsch and R. L. Hancock.

1402  
Ultrastructural Changes in Friend Erythro-  
leukemia Cells Treated with Dimethyl Sulfoxide.  
Toru Sato, Charlotte Friend, and Etienne de  
Harven.

1418  
A Comparative Study of Some of the Enzymes  
Involved in Glucose Metabolism of Human  
Diploid and SV40-transformed Human Diploid  
Cells.  
George A. Dunaway, Jr., and Eddie C. Smith.

1422  
The Effects of Phleomycin on Mouse L-Cells.  
S. J. Shuve and A. M. Rauth.

1429  
Effects of Priming Dose Schedules in Metho- 
trexate Treatment of Mouse Leukemia L1210.  
Marc J. Straus, Nathan Mantel, and Abraham  
Goldin.

1434  
Effects of Phorbol and Four Diesters of Phorbol  
on the Incorporation of Tritiated Precursors  
into DNA, RNA, and Protein in Mouse  
Epidermis.  
William M. Baird, Jane A. Sedgwick, and R. K.  
Boutwell.

1440  
Comparison of Macromolecular Binding of  
Estradiol in Hormone-dependent and Hormone- 
dependent Rat Mammary Carcinoma.  
William L. McGuire and Jo Anne Julian.

1446  
Depression of Homograft Rejection and Graft- 
versus-Host Reactivity following 7,12-Dime- 
thylbenz(a)anthracene Exposure in the Rat.  
A. Tano Di Marco, Claudio Franceschi, Luigi  
Xerri, and Giorgio Prodi.

1451  
A Study of Leukemic Cell Injury by Physical  
Agents.  
Moriit Miura, Kohei Kawashima, Hiroshi Nishi- 
waki, Masaki Kobayashi, Akimitsu Morita,  
Ryuozo Ohno, Hisami Kakizawa, Tadaaki Uetani,  
Masami Hirano, and Kazumasa Yamada.

1457  
Effect of Dopamine and 6-Hydroxydopamine  
on Mouse Neuroblastoma Cells in Vitro.  
Kedar N. Prasad.

1461  
Carcinogenicity Testing of N-Hydroxy and  
Other Oxidation and Decomposition Products  
of 1- and 2-Naphthylamine.  
J. L. Radomski, E. Brill, W. B. Deichmann,  
and E. M. Glass.

1468  
Influence of 3-Methylcholanthrene and Diet  
on the Binding of 2-Acetylaminofluorene and  
Its N-Hydroxy Metabolite to Rat Liver Nucleic  
Acids.  
Charles C. Irving, Thelma C. Peeler, Richard  
A. Veazey, and Ralph Wiseman, Jr.

1473  
Strain Specificity in Mouse Mammary Tumor  
Virus Virion Antigens.  
Phyllis B. Blair.

1478  
Irreversible Change of the Pattern of Carcino- 
genic Aminoazo Dye-binding Proteins in Rat  
Liver during Continuous Feeding of 3'-Methyl-
4-dimethylaminoazobenzene.

Tsutomu Sugimoto and Hiroshi Terayama.

Histology and Ultrastructure of Cultured Human Tumor Cells Exposed to Antiserum to the Nerve Growth Factor.

H. Pinkerton, B. Bhagat, M. W. Rana, and S. Holtwick.

Immunization with Chemically Modified Lymphoma Cells.

Morton D. Prager, Ina Derr, Alan Swann, and Joseph Cotropia.

Isolation, Identification, and Biological Study of Compounds Derived from 3-Methylcholanthrene by Irradiation in Dimethyl Sulfoxide.

Thomas L. Dao, Charles King, and Takeshi Tominaga.

Morphogenesis of Epithelial Neoplasms Induced in the Rat Kidney by Dimethylnitrosamine.


Reduction and Enhancement by Phenobarbital of Hepatocarcinogenesis Induced in the Rat by 2-Acetylaminofluorene.

Carl Peraino, R. J. Michael Fry, and Everett Staffeldt.

Correspondence.

I. Brodsky.

Christopher A. Reilly, Jr., and Gerd T. Schloss.

American Association for Cancer Research: Honorary Certificates of Award for 1971.

Books Received.

Special Announcement: Annual Meeting of the American Association for Cancer Research, Inc.

Announcements.

COVER LEGEND

Observations, from 1950 to 1953, of an unusually high incidence of amyotrophic lateral sclerosis and other neurological disorders endemic to Guam were subsequently (1963) correlated with the ingestion of cycad nut meal. This discovery was accomplished by epidemiological investigations, especially those of Leonard T. Kurland and Majorie G. Whiting. The cycad (Cycas circinalis) is a plant indigenous to the Mariana Islands, and cycad varieties occur widely from the Japanese Archipelago to the subcontinent of India. The plant is of localized economic importance as a source of foodstuffs, fiber, and medicinal products. The studies of Kurland and Whiting suggested the possibility of the existence of a neurotoxic agent in the nut meal and edible starch extracted from cycad roots, stems, and leaves.

In 1963 Gert L. Laqueur (b. 1912, Strasbourg, France), Chief, Laboratory of Experimental Pathology at the National Institute of Arthritis and Metabolic Diseases, and his associates uncovered a carcinogenic property in the cycad. Crude nut meal from C. circinalis fed to rats failed to elicit neurological symptoms but induced cancers of the liver, kidney, and intestinal tract (G. L. Laqueur, O. Mickelsen, M. Whiting, and L. T. Kurland, Carcinogenic Properties of Nuts from Cycas circinalis Indigenous to Guam. J. Natl. Cancer Inst., 31: 919—951, 1963). This work indicated that a glycoside isolated from cycads, and known as cycasin, might yield in its metabolic breakdown a compound with a carcinogenic potential similar to that of dimethylnitrosamine (DMN). This inference was supported by comparable pathological alterations in rats fed toxic cycad nut meal and those reported for rats treated with DMN. Collateral investigations revealed that cycasin was ineffective as a hepatotoxin and a hepatocarcinogen when administered to germfree rats.

A later report by Laqueur and his coworkers showed that a metabolic degradation via β-glucosidase of bacterial origin in the intestinal tracts of rats released the aglycone, a potent carcinogen (G. L. Laqueur, E. G. McDaniel, and H. Matsumoto, Tumor Induction in Germfree Rats With Methylazoxymethanol (MAM) and Synthetic MAM Acetate. J. Natl. Cancer Inst., 39: 355—371, 1967). The aglycone of cycasin (MAM) and the synthetic aglycone acetate ester produced tumors in germfree animals, thus establishing MAM as the proximate carcinogen.

These studies were greatly assisted by the collaboration of Hiromu Matsumoto, whose group prepared synthetic derivatives of MAM (H. Matsumoto, T. Nagahama, and H. O. Larson, Studies on Methylazoxymethanol, the Aglycone of Cycasin: A Synthesis of Methylazoxymethanol. Arch. Biochem. Biophys., 110: 373—380, 1965).

The cover illustrates a mature cycad plant; upper right, recent photograph of Dr. Laqueur (courtesy of the Information Office of the National Institute of Arthritis and Metabolic Diseases. NIH, and Mrs. Frances W. Davis, Editor, NIH Record). Professor Matsumoto is shown at lower right in a photograph supplied by Dr. Laqueur.
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