Developments in Preventive Oncology
Part A

Editor
Herbert E. Nieburgs

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DEVELOPMENTS IN PREVENTIVE ONCOLOGY

PART A

Selected Papers From the Sixth International Symposium on Prevention and Detection of Cancer Held in Vienna, November 26–29, 1984

Editor

HERBERT E. NIEBURGS
Visiting Professor of Pathology
University of Massachusetts Medical School
Berkshire Medical Center
Pittsfield, Massachusetts

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Contributors

Rikiya Abe, Second Department of Surgery, Tohoku University School of Medicine, Sendai, Japan **[311]**

Cyril Abrahams, Department of Pathology, Michael Reese Hospital, University of Chicago, Chicago, IL **[279]**

Caterina Agazzi, Immunohematology Service, Istituto Nazionale per lo Studio e la Cura dei Tumori, Milan, Italy **[459]**

Minoru Akimoto, Second Department of Surgery, Tohoku University School of Medicine, Sendai, Japan **[311]**

E. Alexopoulos, 2nd Prop. Department of Medicine, Aristotelian University of Thessaloniki, Aghia Sophia Hospital, Thessaloniki, Greece **[389]**

Hassan Y. Al Idrissi, College of Medicine and Medical Sciences, King Faisal University, Dammam, Saudi Arabia **[215]**

Abdulaziz Al Quorain, College of Medicine and Medical Sciences, King Faisal University, Dammam, Saudi Arabia **[215]**

Francesca Amelotti, Centro di Studi Biochimici sul Morbo di Cooley, Università degli Studi di Ferrara, Ferrara, Italy **[9]**

Salvatore Andreola, Servizio di Anatomia Patologica, Istituto Nazionale per lo Studio e la Cura dei Tumori, Milano, Italy **[323]**

C. Arciti, National Institute of Cancer Research and Italian League Against Cancer, Genoa, Italy **[421]**

Hartmut Arps, Institute of Pathology and Department of Gynecology and Obstetrics, University of Hamburg, Federal Republic of Germany **[511]**

G. Arrabito, Surgical Clinic, University "La Sapienza," Rome, Italy **[477]**

Riccardo A. Audisio, Clinical Oncology A, Istituto Nazionale per lo Studio e la Cura dei Tumori, Milan, Italy **[459]**

Arthur H. Aufses, Department of Surgery, Mount Sinai Medical Center, New York, NY **[303]**

Merrill S. Babcock, Department of Pathology, Medical College of Ohio, Toledo, OH **[59]**

Paul E. Baldewyns, Cancer Detection Unit and Radio-Immunology Laboratory, Oncology and Nuclear Medicine Department, Jolimont Hospital, La Louvière, Belgium **[495,505]**

P.R. Band, Division of Epidemiology, Biometry and Occupational Oncology, Cancer Control Agency of British Columbia, Vancouver, British Columbia, Canada **[151, 157]**

Peter Bannasch, Abteilung für Cytopathologie, Institut fur Experimentelle Pathologie, Deutsches Krebsforschungzentrum, Heidelberg, Federal Republic of Germany **[243]**

Patrizia Baracchini, Institute of Pathological Anatomy and Histology, Genoa, Italy **[469]**

Rafaella Barbieri, Centro di Studi Biochimici sul Morbo di Cooley, Università degli Studi di Ferrara, Ferrara, Italy **[9]**

Debra Barron, Department of Surgery, The Mount Sinai Medical Center, New York, NY **[359]**

David Bass, Gastroenterology Unit, Kaplan Hospital, Rehovot, Israel **[485]**

Marc P. Beauduin, Cancer Detection Unit and Radio-Immunology Laboratory, Oncology and Nuclear Medicine Department, Jolimont Hospital, La Louvière, Belgium **[495,505]**

Stewart Becker, Gastroenterology Unit, Kaplan Hospital, Rehovot, Israel **[485]**

Zvi Bentwich, Ruth Ben-Ari Institute of Clinical Immunology, Kaplan Hospital, Rehovot, Israel **[485]**

Francesco Bernardi, Centro di Studi Biochimici sul Morbo di Cooley, Università degli Studi di Ferrara, Ferrara, Italy **[9]**

R.B. Bhonsle, Basic Dental Research Unit, Tata Institute of Fundamental Research, Bombay, India **[219]**

P. Bianco, Cytology Service, Institutes of Pathology and Work Medicine, II Faculty of Medicine and Surgery, University of Naples, Naples, Italy **[343]**

Gabriele Binder, Forschungsinstitut Manfred von Ardenne, Dresden, German Democratic Republic **[251]**

Maria Blettner, International Agency for Research on Cancer, Lyon, France **[435]**

R. Boschi, Cytology Service, Institutes of Pathology and Work Medicine, II Faculty of Medicine and Surgery, University of Naples, Naples, Italy **[343]**

Gerald Brauer, Ministry of Health, Vancouver, British Columbia, Canada **[47]**

O. Braun, Institute of Pathology, University of Vienna, Vienna, Austria **[319]**

Klaus D. Brunnemann, Naylor Dana Institute for Disease Prevention, American Health Foundation, Valhalla, NY **[435]**

C. Bulletti, Department of Pathophysiology of Reproduction, University of Bologna, S. Orsola General Hospital, Bologna, Italy **[331]**

The number in brackets is the opening page number of the contributor's article.

O. Buonomo, Surgical Clinic, University "La Sapienza," Rome, Italy **[477]**

Jocelyne A. Burion, Cancer Detection Unit and Radio-Immunology Laboratory, Oncology and Nuclear Medicine Department, Jolimont Hospital, La Louvière, Belgium **[495,505]**

Daniele Buzzoni, Centro di Studi Biochimici sul Morbo di Cooley, Università degli Studi di Ferrara, Ferrara, Italy **[9]**

Vera Cappelletti, Oncologia Sperimentale C, Istituto Nazionale per lo Studio e la Cura dei Tumori, Milan, Italy **[323]**

Brian I. Carr, Department of Medical Oncology, City of Hope National Medical Center, Duarte, CA **[195]**

Yu-Qing Chen, Cancer Institute, Chinese Academy of Medical Sciences, Beijing, China **[145]**

Georgia Christodoulopoulou, Department of Biology, Nuclear Research Center "Democritos" Aghia Paraskevi, Attiki and Metaxas Memorial Cancer Hospital, Piraeus, Greece **[385]**

John Christodoulopoulos, Department of Biology, Nuclear Research Center "Democritos" Aghia Paraskevi, Attiki and Metaxas Memorial Cancer Hospital, Piraeus, Greece **[385]**

P. Ciotti, Department of Pathophysiology of Reproduction, University of Bologna, S. Orsola General Hospital, Bologna, Italy **[331]**

Mieczysław Cisło, Department of Pathological Anatomy, and Clinic of Gynecological Surgery, School of Medicine, Wrocław, Poland **[347]**

Andrew J. Coldman, Division of Epidemiology, Biometry and Occupational Oncology, Cancer Control Agency of British Columbia, Vancouver, British Columbia, Canada **[47,151]**

G. Concolino, Medical Clinic, University "La Sapienza," Rome, Italy **[477]**

Francesco Conconi, Centro di Studi Biochimici sul Morbo di Cooley, Università degli Studi di Ferrara, Ferrara, Italy **[9]**

C. Conti, Medical Clinic, University "La Sapienza," Rome, Italy **[477]**

Danila Coradini, Oncologia Sperimentale C, Istituto Nazionale per lo Studio e la Cura dei Tumori, Milan, Italy **[323]**

Joseph W. Cullen, Division of Cancer Prevention and Control, National Cancer Institute, National Institutes of Health, Bethesda, MD **[125]**

A. Curt, Biochemical and Experimental Division of the IInd Department of Surgery, University of Cologne, Köln, Federal Republic of Germany **[17]**

D.K. Daftary, Basic Dental Research Unit, Tata Institute of Fundamental Research, Bombay, India **[219]**

Patrizia Dell'Orto, Institute of Pathology, University of Milan, Milan, Italy **[511]**

Laura del Senno, Centro di Studi Biochimici sul Morbo di Cooley, Università degli Studi di Ferrara, Ferrara, Italy **[9]**

M.G. Deo, Cancer Research Institute, Tata Memorial Centre, Parel, Bombay, India **[207]**

M. Deschamps, Division of Epidemiology, Biometry and Occupational Oncology, Cancer Agency of British Columbia, Vancouver, British Columbia, Canada **[151]**

Manfred Dietel, Institute of Pathology and Department of Gynecology and Obstetrics, University of Hamburg, Hamburg, Federal Republic of Germany **[511]**

Giovanni Di Fronzo, Oncologia Sperimentale C, Istituto Nazionale per lo Studio e la Cure dei Tumori, Milan, Italy **[323]**

Christian Dittrich, Department of Chemotherapy, University of Vienna, Vienna, Austria **[521]**

Rakesh Dixit, Department of Pathology, Medical College of Ohio, Toledo, OH **[59]**

Roberto Doci, Clinical Oncology A, Istituto Nazionale per lo Studio e la Cura dei Tumori, Milan, Italy **[459]**

William D. Dupont, Department of Preventive Medicine, Vanderbilt University School of Medicine, Nashville, TN **[441]**

Sándor Eckhardt, National Institute of Oncology, Budapest, Hungary **[409]**

D. Eddy, WHO Collaborating Center for Research in Cancer Policy, Duke University, Durham, NC **[113]**

Ibrahim El-Desoky, Department of Pathology, Faculty of Medicine, Mansoura University, Egypt **[227]**

Tarek M. El-Diasty, Department of Radiology, Faculty of Medicine, Mansoura University, Egypt **[227]**

Sabry A. El-Mogy, Department of Radiology, Faculty of Medicine, Mansoura University, Egypt **[227]**

Gamal F. El-Wahidy, Department of Radiotherapy, Faculty of Medicine, Mansoura University, Egypt **[227]**

J. Mark Elwood, Department of Community Health, University of Nottingham, Nottingham, England **[47]**

Anders Englund, Saltsjöbaden, Sweden **[413]**

Richard Fagerstrom, Department of Surgery, Mount Sinai Medical Center, New York, NY **[303]**

Zhen-Fu Fan, Cancer Institute, Chinese Academy of Medical Sciences, Beijing, China **[145]**

Farag M. Farag, Department of Medicine, Faculty of Medicine, Mansoura University, Egypt **[227]**

Michael Feldstein, Harvard School of Public Health, Boston, MA **[157]**

Vera Ferlan-Marolt, Institute of Pathology, Medical Faculty, University of Ljubljana, Ljubljana, Yugoslavia **[491]**

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Aharon Fink, Ruth Ben-Ari Institute of Clinical Immunology, Kaplan Hospital, Rehovot, Israel **[485]**

Zvi Fireman, Institute of Gastroenterology, Ichilov Hospital, Tel Aviv, Israel **[485]**

C. Flamigni, Department of Pathophysiology of Reproduction, University of Bologna, S. Orsola General Hospital, Bologna, Italy **[331]**

Arthur L. Frank, Department of Preventive Medicine and Environmental Health, University of Kentucky College of Medicine, Lexington, KY **[337,429]**

Eric Frost, Institut du Cancer de Montréal, Centre Hospitalier Notre-Dame and Département de Médecine, Université de Montréal, Montréal, Québec, Canada **[289]**

Ezio Fulcheri, Institute of Pathological Anatomy and Histology, Genoa, Italy **[469]**

F. Fulciniti, Cytology Service, Institutes of Pathology and Work Medicine, II Faculty of Medicine and Surgery, University of Naples, Naples, Italy **[343]**

H. Gaget, Service des Maladies Sanguines et Tumorales & ICIG (Université Paris–Sud, CNRS LA-149, Association CI. Bernard & ARC), Hôpital Paul-Brousse, Villejuif, France **[79,167]**

Roberto Gambari, Centro di Studi Biochimici sul Morbo di Cooley, Università degli Studi di Ferrara, Ferrara, Italy **[9]**

Andrej Genčík, Laboratory of Human Genetics, Department of Research, University Clinics, Cantonal Hospital, Basel, Switzerland **[455]**

Tarunendu Ghose, Department of Pathology, Dalhousie University, Halifax, Nova Scotia, Canada **[279]**

Tuvia Gilat, Institute of Gastroenterology, Ichilov Hospital, Tel Aviv, Israel **[485]**

Felix Gottesfeld, Gastroenterology Unit, Kaplan Hospital, Rehovot, Israel **[485]**

J. Gouveia, Service des Maladies Sanguines et Tumorales & ICIG (Universitě Paris–Sud, CNRS LA-149, Association CI. Bernard & ARC), Hôpital Paul Brousse, Villejuif, France **[79,167]**

F. Gros, Service des Maladies Sanguines et Tumorales & ICIG (Universitě Paris–Sud, CNRS LA-149, Association CI. Bernard & ARC), Hôpital Paul-Brousse, Villejuif, France **[79]**

Prakash C. Gupta, Basic Dental Research Unit, Tata Institute of Fundamental Research, Bombay, India **[219]**

Matti Hakama, Department of Public Health, University of Tampere, Tampere and Finnish Cancer Registry, Helsinki, Finland **[139]**

Anwar A. Hakim, Department of Internal Medicine, Stritch School of Medicine, and Division of Cellular Immunology, Department of Histology, Loyola University Medical Center, Maywood, IL **[373]**

Aharon Hallak, Institute of Gastroenterology, Ichilov Hospital, Tel Aviv, Israel **[485]**

R.E. Harahap, Palatehan, Kebayoran-Baru, Jakarta, Indonesia **[237]**

Antonina Harłozińska-Szmyrka, Department of Pathological Anatomy, School of Medicine, Worcław, Poland **[347,365]**

Maria Havredaki, Department of Biology, Nuclear Research Center "Democritos" Aghia Paraskevi, Attiki and Metaxas Memorial Cancer Hospital, Piraeus, Greece **[385]**

Mohamed M. Higazi, College of Medicine and Medical Sciences, King Faisal University, Dammam, Saudi Arabia **[215]**

T. Gregory Hislop, Division of Epidemiology, Biometry, and Occupational Oncology, Cancer Control Agency of British Columbia, Vancouver, British Columbia, Canada **[47,151]**

Ilsetraut Hoepfner, Institute of Pathology, University of Hamburg, Hamburg, Federal Republic of Germany **[293]**

Dietrich Hoffmann, Naylor Dana Institute for Disease Prevention, American Health Foundation, Valhalla, NY **[435]**

Fritz Hölzel, Institute of Pathology and Department of Gynecology and Obstetrics, University of Hamburg, Federal Republic of Germany **[511]**

J.P. Homasson, Service des Maladies Sanguines et Tumorales & ICIG (Université Paris–Sud, CNRS LA-149, Association CI. Bernard & ARC), Hôpital Paul-Brousse, Villejuif, France **[79,167]**

John Horton, Division of Oncology, Department of Medicine, Albany Medical College, Albany, NY **[417]**

Ezzeldin M. Ibrahim, College of Medicine and Medical Sciences, King Faisal University, Dammam, Saudi Arabia **[215]**

Maria Teresa Illeni, Immunohematology Service, Istituto Nazionale per lo Studio e la Cura dei Tumori, Milan, Italy **[459]**

Hiroshi Ishii, Department of Surgery, Sanuma Public Hospital, Sanuma, Japan **[311]**

Hideyasu Iwasaki, Second Department of Surgery, Tohoku University School of Medicine, Sendai, Japan **[311]**

J.R. Izbicki, IInd Department of Surgery of the University of Cologne, Köln, Federal Republic of Germany **[17]**

Birger Jansson, Department of Biomathematics, University of Texas, Houston, TX **[171]**

V.M. Jasonni, Department of Pathophysiology of Reproduction, University of Bologna, S. Orsola General Hospital, Bologna, Italy **[331]**

Kasturi Jayant, Cancer Research Institute, Tata Memorial Centre, Parel, Bombay, India **[207]**

Lisa Kan, Department of Epidemiology, Biometry and Occupational Oncology, Cancer Control Agency of British Columbia, Vancouver, British Columbia, Canada **[47]**

Morio Kasai, Second Department of Surgery, Tohoku University School of Medicine, Sendai, Japan **[311]**

B. Kim, Service des Maladies Sanguines et Tumorales & ICIG (Université Paris–Sud, CNRS LA-149, Association CI. Bernard & ARC), Hôpital Paul-Brousse, Villejuif, France **[79]**

Kitai Kim, Department of Pathology, Medical College of Ohio, Toledo, OH **[59]**

I. Koza, Department of Haematology, Institute of Clinical Oncology, Bratislava, Czechoslovakia **[113]**

Andrea Kröger, Institute of Pathology and Department of Gynecology and Obstetrics, University of Hamburg, Federal Republic of Germany **[511]**

Winfried Krüger, Forschungsinstitut Manfred von Ardenne, Dresden, German Democratic Republic **[251]**

J. Kusche, Biochemical and Experimental Division of the IInd Department of Surgery, University of Cologne, Köln, Federal Republic of Germany **[17]**

L. La Marca, Department of Pathophysiology of Reproduction, University of Bologna, S. Orsola Hospital, Bologna, Italy **[331]**

Gabriella Lapertosa, Institute of Pathological Anatomy and Histology, Genoa, Italy **[469]**

G. Lemaigre, Service des Maladies Sanguines et Tumorales & ICIG (Université Paris–Sud, CNRS LA-149, Association CI. Bernard & ARC), Hôpital Paul-Brousse, Villejuif, France **[79]**

Gerson Lesnick, Department of Surgery, Mount Sinai Medical Center, New York, NY **[303]**

Emilia Levy, Oncology Unit, Rehovot, Israel **[485]**

Frederick P. Li, Clinical Epidemiology Branch, National Cancer Institute **[41]**

Shu-Zhong Liu, Cancer Institute, Chinese Academy of Medical Sciences, Beijing, China **[145]**

Claudia Lombardo, Immunohematology Service, Istituto Nazionale per lo Studio e la Cura dei Tumori, Milan, Italy **[459]**

Thomas Löning, Institute of Pathology, University of Hamburg, Hamburg, Federal Republic of Germany **[293]**

Luciano Luciani, Oncologia Clinica B, Istituto Nazionale per lo Studio e la Cura dei Tumori, Milan, Italy **[323]**

Gadi M. Magbool, College of Medicine and Medical Sciences, King Faisal University, Dammam, Saudi Arabia **[215]**

U. Maier, University Clinic for Urology, University of Vienna, Vienna, Austria **[319]**

Giovanna Marchetti, Centro di Studi Biochimici sul Morbo di Cooley, Università degli Studi di Ferrara, Ferrara, Italy **[9]**

G. Marino, Cytology Service, Institutes of Pathology and Work Medicine, II Faculty of Medicine and Surgery, University of Naples, Naples, Italy **[343]**

M. Marino, Cytology Service, Institutes of Pathology and Work Medicine, II Faculty of Medicine and Surgery, University of Naples, Naples, Italy **[343]**

Saša Markovič, Institute of Pathology, Medical Faculty, University of Ljubljana, Ljubljana, Yugoslavia **[491]**

Giorgio Martinelli, Department of Surgery, The Mount Sinai Medical Center, New York, NY **[359]**

Eliana Mascheretti, Immunohematology Service, Istituto Nazionale per lo Studio e la Cura dei Tumori, Milan, Italy **[459]**

G. Mathé, Service des Maladies Sanguines et Tumorales & ICIG (Université Paris–Sud, CNRS LA-149, Association CI. Bernard & ARC), Hôpital Paul-Brousse, Villejuif, France **[79,167]**

Eugene G. Matiakin, All Union Cancer Research Center of the USSR Academy of Medical Sciences, Moscow, USSR **[435]**

Fali S. Mehta, Basic Dental Research Unit, Tata Institute of Fundamental Research, Bombay, India **[219]**

D. Memmos, 2nd Prop. Department of Medicine, Aristotelian University of Thessaloniki, Aghia Sophia Hospital, Thessaloniki, Greece **[389]**

R. Mennigen, IInd Department of Surgery of the University of Cologne, Köln, Federal Republic of Germany **[17]**

Seth R. Miller, Department of Surgery, Mount Sinai Medical Center, New York, NY **[303]**

Patrizia Miodini, Oncologia Sperimentale C, Istituto Nazionale per lo Studio e la Cura dei Tumori, Milan, Italy **[323]**

Y. Mishima, Second Department of Surgery, School of Medicine, Tokyo Medical and Dental University, Bunkyo-Ku, Tokyo, Japan **[449]**

J.L. Misset, Service des Maladies Sanguines et Tumorales & ICIG (Université Paris–Sud, CNRS LA-149, Association CI. Bernard & ARC), Hôpital Paul-Brousse, Villejuif, France **[79,167]**

Felix Mitelman, Department of Clinical Genetics, University Hospital, Lund, Sweden **[31]**

Hansjakob Müller, Laboratory of Human Genetics, Department of Research, University Clinics, Cantonal Hospital, Basel, Switzerland **[455]**

Y. Murayama, Second Department of Surgery, School of Medicine, Tokyo Medical and Dental University, Bunkyo-Ku, Tokyo, Japan **[449]**

P.R. Murti, Basic Dental Research Unit, Tata Institute of Fundamental Research, Bombay, India **[219]**

Yoshimichi Nakajima, Second Department of Surgery, Tohoku University School of Medicine, Sendai, Japan **[311]**

S. Naldi, Department of Pathophysiology of Reproduction, University of Bologna, S. Orsola Hospital, Bologna, Italy **[331]**

N.P. Napalkov, N.N. Petrov Research Institute of Oncology, Leningrad, USSR **[1]**

Axel Niendorf, Institute of Pathology and Department of Gynecology and Obstetrics, University of Hamburg, Federal Republic of Germany **[511]**

Cordula Nitsch, Max-Planck-Institute for Brain Research, Frankfurt am Main, Federal Republic of Germany **[259]**

K.H. Notaney, World Health Organization, Programme Coordinator and Representative, Colombo, Sri Lanka **[113]**

Anna Noworolska, Department of Pathological Anatomy, School of Medicine, Wrocław, Poland **[365]**

H. Ogimura, Japan Computer Center, Toranomon, Tokyo, Japan **[449]**

Saro Oriana, Oncologia Clinica B, Istituto Nazionale per lo Studio e la Cura dei Tumori, Milan, Italy **[323]**

P. Paesani, Medical Clinic, University "La Sapienza," Rome, Italy **[477]**

David L. Page, Departments of Pathology and Preventive Medicine, Vanderbilt University School of Medicine, Nashville, TN **[441]**

L. Palombini, Cytology Service, Institutes of Pathology and Work Medicine, II Faculty of Medicine and Surgery, University of Naples, Naples, Italy **[343]**

M. Papadimitriou, 2nd Prop. Department of Medicine, Aristotelian University of Thessaloniki, Aghia Sophia Hospital, Thessaloniki, Greece **[389]**

Angelos E. Papatestas, Department of Surgery, Mount Sinai Medical Center, New York, NY **[303]**

J.V. Parkin, IInd Department of Surgery of the University of Cologne, Köln, Federal Republic of Germany **[17]**

Norbert Pateisky, 1st Department of Gynecology and Obstetrics, University of Vienna, Vienna, Austria **[521]**

Patrizia Patracchini, Centro di Studi Biochimici sul Morbo di Cooley, Università degli Studi di Ferrara, Ferrara, Italy **[9]**

Demetrios Pertsemlidis, Department of Surgery, Mount Sinai Medical Center, New York, NY **[303]**

C. Picardi, Department of Surgery, University "Tor Vergata," Rome, Italy **[477]**

Jens J. Pindborg, Department of Oral Pathology, Royal Dental College and Dental Department, Rigshospitalet, Copenhagen, Denmark **[219]**

Roberta Piva, Centro di Studi Biochimici sul Morbo di Cooley, Università degli Studi di Ferrara, Ferrara, Italy **[9]**

Eric P. Pluygers, Cancer Detection Unit and Radio-Immunology Laboratory, Oncology and Nuclear Medicine Department, Jolimont Hospital, La Louvière, Belgium **[495,505]**

Boris P. Poljakov, All Union Cancer Research Center of the USSR Academy of Medical Sciences, Moscow, USSR **[435]**

Jerzy Rabczyński, Department of Pathological Anatomy, and Clinic of Gynecological Surgery, School of Medicine, Wrocław, Poland **[347]**

M.A. Rahim, Cancer Institute, Dacca, Bangladesh **[203]**

K.V.N. Rao, Department of Radiology, University of Chicago, Chicago, IL **[279]**

Roman Richter, Department of Pathological Anatomy, and Clinic of Gynecological Surgery, School of Medicine, Wrocław, Poland **[347,365]**

Koshilya S. Rijhsinghani, Department of Pathology, Michael Reese Hospital, Chicago, IL **[279]**

Ruth Roemer, School of Public Health, University of California, Los Angeles, CA **[99]**

Lowell W. Rogers, Department of Pathology, Vanderbilt University School of Medicine, Nashville, TN, and Department of Pathology, Carraway Methodist Medical Center, Birmingham, AL **[441]**

Enrico Ronchi, Oncologia Sperimentale C, Istituto Nazionale per lo Studio e la Cura dei Tumori, Milan, Italy **[323]**

Miriam P. Rosin, Environmental Carcinogenesis Unit, British Columbia Cancer Research Centre, Vancouver, British Columbia, Canada **[435]**

Mariella Rossi, Centro di Studi Biochimici sul Morbo di Cooley, Università degli Studi di Ferrara, Ferrara, Italy **[9]**

Paul Rozen, Institute of Gastroenterology, Ichilov Hospital, Tel Aviv, Israel **[485]**

Geno Saccomanno, St. Mary's Hospital, Grand Junction, CO **[157]**

G. Sakellariou, 2nd Prop. Department of Medicine, Aristotelian University of Thessaloniki, Aghia Sophia Hospital, Thessaloniki, Greece **[389]**

Hanem M. Sakr, Department of Radiotherapy, Faculty of Medicine, Mansoura University, Egypt **[227]**

Heinrich Salzer, 1st Department of Gynecology and Obstetrics, University of Vienna, Vienna, Austria **[521]**

G. Santelli, Service des Maladies Sanguines et Tumorales & ICIG (Université Paris–Sud, CNRS LA-149, Association CI. Bernard & ARC), Hôpital Paul-Brousse, Villejuif, France **[79,167]**

L. Santi, National Institute of Cancer Research and Italian League Against Cancer, Genoa, Italy **[421]**

Mohamed B. Satti, College of Medicine and Medical Sciences, King Faisal University, Dammam, Saudi Arabia **[215]**

Barbara M. Scher, Department of Microbiology, Chemotherapy Foundation Laboratory, Mount Sinai School of Medicine of the City University of New York, New York, NY **[395]**

William Scher, Department of Medicine, Chemotherapy Foundation Laboratory, Mount Sinai School of Medicine of the City University of New York, New York, NY **[395]**

O.K. Schlappack, University Clinic for Radiotherapy and Radiobiology, University of Vienna, Vienna, Austria **[319]**

Dietrich Schmähl, Institute of Toxicology and Chemotherapy, German Cancer Research Center, Heidelberg, Federal Republic of Germany **[91]**

Paul Sevelda, 1st Department of Gynecology and Obstetrics, University of Vienna, Vienna, Austria **[521]**

Sameh S. Shamaa, Department of Medicine, Faculty of Medicine, Mansoura University, Egypt **[227]**

Adi Shani, Oncology Unit, Kaplan Hospital, Rehovot, Israel **[485]**

Z. Sinakos, 2nd Prop. Department of Medicine, Aristotelian University of Thessaloniki, Aghia Sophia Hospital, Thessaloniki, Greece **[389]**

Charles M. Siraki, Department of Internal Medicine, Stritch School of Medicine, and Division of Cellular Immunology, Department of Histology, Loyola University Medical Center, Maywood, IL **[373]**

Barbara Ślesak, Department of Pathological Anatomy, and Clinic of Gynecological Surgery, School of Medicine, Wrocław, Poland **[347]**

L. Sobin, Department of Gastrointestinal Pathology and WHO Collaborating Center for the International Histological Classification of Tumors, Armed Forces Institute of Pathology, Washington, DC **[113]**

Jürgen Spona, 1st Department of Gynecology and Obstetrics, University of Vienna, Austria **[521]**

Kenneth E. Stanley, Cancer Unit, World Health Organization, Geneva, Switzerland **[83,113]**

Bryan Steinberg, Department of Surgery, The Mount Sinai Medical Center, New York, NY **[359]**

Hans F. Stitch, Environmental Carcinogenesis Unit, British Columbia Cancer Research Centre, Vancouver, British Columbia, Canada **[435]**

J. Stjernswärd, Cancer Unit, World Health Organization, Geneva, Switzerland **[113]**

Gary D. Stoner, Department of Pathology, Medical College of Ohio, Toledo, OH **[59]**

M.C. Sudre, Service des Maladies Sanguines et Tumorales & ICIG (Université Paris–Sud, CNRS LA-149, Association CI. Bernard & ARC), Hôpital Paul-Brousse, Villejuif, France **[79,167]**

Martha Suh, Institut du Cancer de Montréal, Centre Hospitalier Notre-Dame and Département de Médecine, Université de Montréal, Montréal, Québec, Canada **[289]**

Martin A. Swerdlow, Department of Pathology, Michael Reese Hospital, Chicago, IL **[279]**

Masaki Tan, Second Department of Surgery, Tohoku University School of Medicine, Sendai, Japan **[311]**

Run-Sheng Tan, Cancer Institute, Chinese Academy of Medical Sciences, Beijing, China **[145]**

Rita Tanzi, Institute of Pathological Anatomy and Histology, Genoa, Italy **[469]**

Paul Ian Tartter, Department of Surgery, The Mount Sinai Medical Center, New York, NY **[359]**

Robert W. Teel, Department of Physiology & Pharmacology, Loma Linda University, Loma Linda, California **[59]**

M. Tsechkovski, Cancer Unit, World Health Organization, Geneva, Switzerland **[113]**

T. Tsobanelis, 2nd Prop. Department of Medicine, Aristotelian University of Thessaloniki, Aghia Sophia Hospital, Thessaloniki, Greece **[389]**

A. Vetrani, Cytology Service, Institutes of Pathology and Work Medicine, II Faculty of Medicine and Surgery, University of Naples, Naples, Italy **[343]**

Giuseppe Viale, Institute of Pathology, University of Milano, Milan, Italy **[511]**

A. Vignudelli, Department of Pathophysiology of Reproduction, University of Bologna, S. Orsola General Hospital, Bologna, Italy **[331]**

Alexandra von Metzler, Max-Planck-Institute for Brain Research, Frankfurt am Main, Federal Republic of Gemany **[259]**

Alan B. Watson, Department of Dermatology, Royal Newcastle Hospital, Newcastle, Australia **[161]**

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Walter Weber, Laboratory of Human Genetics, Department of Research, University Clinics, Cantonal Hospital, Basel, Switzerland **[455]**

Ibrahim Yasseen, Department of Surgery, Faculty of Medicine, Mansoura University, Egypt **[227]**

A. Zabatta, Cytology Service, Institutes of Pathology and Work Medicine, II Faculty of Medicine and Surgery, University of Naples, Naples, Italy **[343]**

David G. Zaridze, International Agency for Research on Cancer, Lyon, France **[435]**

W. Jens Zeller, Institute of Toxicology and Chemotherapy, German Cancer Research Center, Heidelberg, Federal Republic of Germany **[91]**

You-Hui Zhang, Cancer Institute, Chinese Academy of Medical Sciences, Beijing, China **[145]**

馆藏专用章

INTRODUCTION

HERBERT E. NIEBURGS, MD

Mt. Sinai School of Medicine of the City University of New York, New York, New York

Symposia and publications of the International Society for Preventive Oncology (ISPO) are designed to foster prevention of cancer morbidity and detection and management of cancer in its earliest curable stages. The 6th International Symposium on Prevention and Detection of Cancer in Vienna, Austria, November 26–29, 1984, was sponsored by ISPO and cosponsored by the World Health Organization and the Austrian Cancer Society-Cancer League. This current volume contains selected contributions updated subsequent to presentation and is offered for assessment and implementation of existing knowledge for the effective management of cancer.

The control of human oncogenesis is a multidisciplinary task. While tumor growth has a long latency period, the increased life expectancy through the control of health hazards and changing lifestyles has provided the additional number of years needed for tumor development. Improved methods of detection and diagnosis have also influenced the apparent rise in cancer incidence.

Cancer is a disease of great complexity with many inducing and promoting factors usually exerting their action in various combinations. Most neoplastic diseases are systemic with local tumor manifestations. Tumor growth depends upon the actions of multiple etiologic factors: rate of cell proliferation, cell survival time, immunocompetence of the host, and other influences. Because cancer is a multitude of diseases, tumors identified by the same terminology may differ in their response to therapy and from patient to patient at different stages of the disease.

The multifactorial aspects and multidisciplinary approach to the prevention of neoplastic diseases is presented in two major sections. The first section includes reports on oncodevelopmental factors such as geographic oncology, smoking hazards, genetic and dietary influences, xenobiotics, viral causes, immune dysfunctions, and hormones as well as designs and strategies of cancer control studies and the role of education, health organizations and professional societies. The second section is devoted to clinical oncology. It presents much useful information on risk assessment and prognosis, biological markers for detection and diagnosis, therapy-induced changes, and, specifically, the diagnosis and management of tumors in various sites.

Acknowledgement is made to those who gave generously of their time and expertise to make this volume possible: the members of the Program Committee for their help and advice in the preparation of the scientific program, particularly Doctors J Stjernsward, K Stanley, JF Holland, JH Holzner, and VEO Valli; the many contributing and presenting authors; and the reviewers for their editorial collaboration including Doctors JG Bekesi, AL Frank, SA Geller, P Hobbs, HL Ioachim, H Letton, AJ Mastromarino, CJ Mettlin, PM Newberne, J Schlom and coworkers, and P Strax. Thanks are also extended to the devoted staff of ISPO including Ms. S Kay and Ms. J Burgess; the publisher, Mr. Alan R Liss and his staff including Mr. C Robertson and Ms. M Walsh; and many other participants too numerous to name.

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ONCODEVELOPMENTAL FACTORS

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Prenatal and Childhood Exposure to Carcinogenic Factors

N.P. Napalkov, MD, Prof.

N.N. Petrov Research Institute of Oncology, Leningrad, U.S.S.R.

ABSTRACT Transplacental carcinogenic effects have been demonstrated for about 60 chemicals in eight animal species and even in the human. Many carcinogens are much more active in the fetus than in the adult animal. The stage specificity of transplacental carcinogenesis is characterized by the possibility of inducing tumors only at certain stages of embryogenesis (at the end of organogenesis and during the whole period of histogenesis). Risk of transplacental carcinogenesis is owing to the passage of carcinogens or their active metabolites into embryonic tissue and the possibility of metabolic activation of substances within the fetus. In this connection, four main pathways can be hypothesized for the carcinogenic effect of a substance on the fetus. Organotropism with transplacental carcinogenesis is determined by genetic predisposition, differentiation, and proliferative activity in the target tissues. For indirect carcinogens the level of metabolizing enzymes is also important. Teratogenesis and carcinogenesis can be either independent processes or pathogenetically related to each other (eg, DES action). Experimental data can readily be applied to the discussion of prophylaxis of prenatal tumors in the human.

Key words: transplacental carcinogenesis; stage specificity, passage, and metabolic activation of carcinogens in the fetus; organotropism; species and stain specificity; relationship between malformations and tumors; prenatal prophylaxis

The phenomenon of prenatally induced, transplacental carcinogenesis was first described by Larsen [1]. However, almost 20 years passed before an extensive exploration of this problem began in the mid-1960s [2–6]. Since that time, the transplacental carcinogenic effects of more than 60 substances and their different combinations have been demonstrated in animal experiments. It was revealed that virtually all site-specific tumors that could be induced in adult experimental animals were also observed following prenatal exposure to carcinogens. The most frequent findings were tumors of the nervous system, kidney, and lungs.

The possibility of tumor induction by transplacental application of carcinogenic chemicals has already been demonstrated in the following animal species: rat, mouse, hamster, rabbit, pig, guinea pig, dog, and monkey. Similarly, at about the same time, convincing epidemiologic evidence of the risk of transplacental carcinogenesis in the human was reported by Herbst et al [7].

The vast majority of chemicals selected for experiments in prenatal exposure were already known to be highly carcinogenic in adult animals of the same species. However, multiple experiments showed that the ability of these substances to induce tumors by transpla-

Address reprint requests to N.P. Napalkov, N.N. Petrov Research Institute of Oncology, Pesochny-2, Leningrad 188646, U.S.S.R.

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