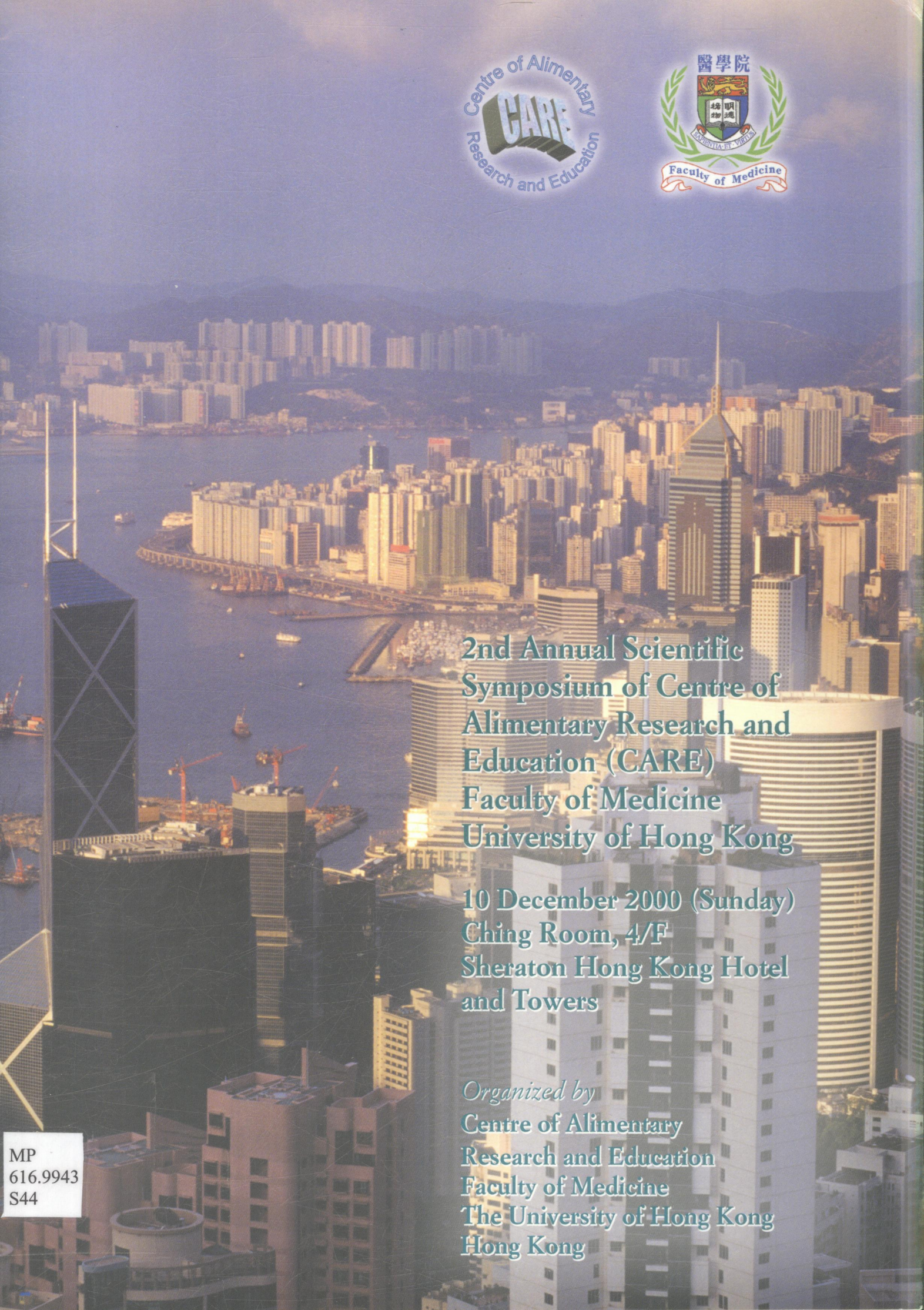

2nd Annual Scientific Symposium of Centre of Alimentary Research and Education (CARE) Faculty of Medicine University of Hong Kong

10 December 2000 (Sunday)
Ching Room, 4/F
Sheraton Hong Kong Hotel and Towers

Organized by
Centre of Alimentary Research and Education
Faculty of Medicine
The University of Hong Kong
Hong Kong

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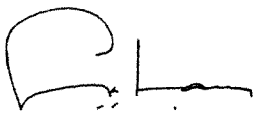
WELCOME MESSAGE

The mission of CARE is to advance gastroenterological science through high quality research and education. Its initial nucleus is a group of scientists and clinicians working in a multitude of disciplines, sharing the same vision of developing an Asian centre of gastroenterological science, and coming together to move towards this ultimate goal.

This is the second CARE scientific meeting. We hope that this will arouse the attention of individuals and groups in Asia, who share the same mission and vision, to join us or allow us to join them.

The meeting this year focuses on gastric and colon cancers, which are important issues in both Hong Kong and worldwide. A group of world renowned overseas scholars together with eminent local academics are invited to present areas of basic science research, including the promising cyclooxygenase story, as well as the clinical researches on prevention, diagnosis and treatment. It is hoped that the symposium will provide some of the upfront and state of art research findings and directions in both basic and clinical setting, to benefit the local scientific community.



Prof. Shiu-kum Lam
Director, CARE

CONFERENCE PROGRAMME

10 December 2000, SUNDAY

TIME	ACTIVITY	SPEAKER
10:00 - 19:30	**Registration**	
10:45 - 11:00	**Opening Remarks by** Prof Shiu-Kum Lam, Director, CARE & Dr H Yuen, President, Hong Kong Society of Gastroenterology	
11:00 - 12:00	**State-of-the-Art Lectures** Chairmen: CH Cho & WM Hui	
	Deficiency of COX-1 or COX-2 Reduces Intestinal Cancer in Genetically Predisposed or Carcinogen Treated Mice	Patricia C Chulada
	COX-2 and Colon Cancer: Molecular Mechanisms	Raymond N DuBois
12:00 - 13:00	**Cyclooxygenase (COX) Research Symposium** Chairmen: CH Cho & WM Hui	
	COX and Apoptosis in Gastric Cancer	Xiao-Ming Fan
	COX and Protein Kinase C	Xiao-Hua Jiang
13:00 - 14:30	**Lunch Symposium co-sponsored by Pharmacia and Pfizer** Chairmen: H Yuen & KM Chu	
	New Safety Evidences of COX-2 Specific Inhibitors in Arthritis Management	Kam-Chuen Lai
	Buffet Lunch — Sky Lounge, 18/F	
14:30 - 16:00	**Colon Cancer Symposium** Chairmen: PKH Tam & PCW Fung	
	Gatekeeper, Caretaker, Landscaper — Jobs for the Boys?	Nicholas A Wright
	Anorectal Cancer and Human Papillomavirus	Allan R Ronald
	Hereditary and Early-Onset Colorectal Cancer: Genetic Diagnosis and Implications	Siu-Tsan Yuen
16:00 - 16:30	**Coffee Break**	
16:30 - 18:00	**Gastric Cancer Symposium** Chairmen: J Wong & ST Yuen	
	Ultra Staging in Gastric Cancer: Where Are We Heading?	Martin S Karpeh
	Regional Intra-Arterial Chemotherapy for Advanced Gastric Cancer	Kent-Man Chu
	Genetic and Epigenetic Mechanisms Underlying Mismatch Repair Deficiency	Suet-Yi Leung
18:00 - 19:00	**Cocktails**	
19:00	**Dinner Symposium sponsored by Merck Sharp & Dohme** Chairmen: SK Lam & BCY Wong	
	Chemoprevention of Colorectal Cancer	Raymond N DuBois
20:00	**Chinese Dinner** — Ming Room, 4/F	

CONTINUING MEDICAL EDUCATION (CME)

Accreditation are available from the following Colleges of the Hong Kong Academy of Medicine :

Hong Kong College of Anaesthesiologists

- 6 CME points for the full programme
- FHKCA should sign the Attendance Record for Accreditation at the Registration Desk

Hong Kong College of Community Medicine

- 6 CME points for the full programme

Hong Kong College of Emergency Medicine

- 3 CME points for the full programme
- FHKCEM should sign the Attendance Record for Accreditation at the Registration Desk

Hong Kong College of Family Physicians

- 4 CME points in Category 6.2 for the full programme
- FHKCFP should sign the Attendance Record for Accreditation at the Registration Desk

Hong Kong College of Obstetricians & Gynaecologists

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Hong Kong College of Radiologists

- 5 CME points for the full programme

The College of Surgeons of Hong Kong

- 6 CME points for the full programme
- FCSHK should sign the Attendance List at the Registration Desk

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	Dr Suet-Yi Leung	*Department of Pathology*
	Professor Paul KH Tam	*Department of Surgery*
	Professor John Wong	*Department of Surgery*

INVITED SPEAKERS

Kent-Man Chu

Associate Professor, Division of Upper Gastrointestinal Surgery, Department of Surgery, The University of Hong Kong Medical Centre, Queen Mary Hospital, Hong Kong

Patricia C Chulada

Health Sciences Administrator, Office of Clinical Research, National Institute of Environmental Health Sciences, National Institute of Health, USA

Raymond N DuBois

Mina C Wallace Professor of Medicine and Cell Biology, Director of Gastroenterology, Hepatology & Nutrition, Department of Medicine & Gastroenterology, Vanderbilt University Medical Center, Nashville, Tennessee, USA

Xiao-Ming Fan

Division of Gastroenterology, Department of Medicine, The University of Hong Kong, Queen Mary Hospital, Hong Kong

Xiao-Hua Jiang

Division of Gastroenterology, Department of Medicine, The University of Hong Kong, Queen Mary Hospital, Hong Kong

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Kam-Chuen Lai

Senior Medical Officer, Division of Gastroenterology, Department of Medicine, Queen Mary Hospital, Hong Kong

Suet-Yi Leung

Associate Professor, Department of Pathology, The University of Hong Kong, Queen Mary Hospital, Hong Kong

Allan R Ronald

Professor, Faculty of Medicine, The University of Manitoba, St-Boniface General Hospital, Winnipeg, Manitoba, Canada

Nicholas A Wright

Professor, Deputy Principal & Vice-Principal for Research, Imperial College School of Medicine, Hammersmith Hospital, London, England, UK

Siu-Tsan Yuen

Senior Medical Officer, Department of Pathology, Queen Mary Hospital, Hong Kong

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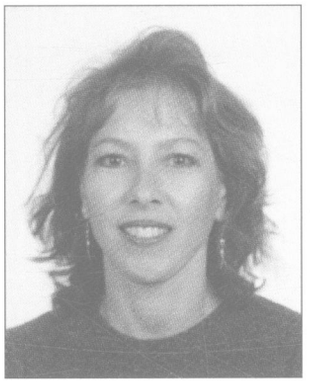
Hon Yuen

Chief of Service and Consultant Physician, Department of Medicine & Geriatrics, Princess Margaret Hospital, Hong Kong

Siu-Tsan Yuen

Senior Medical Officer, Department of Pathology, Queen Mary Hospital, Hong Kong

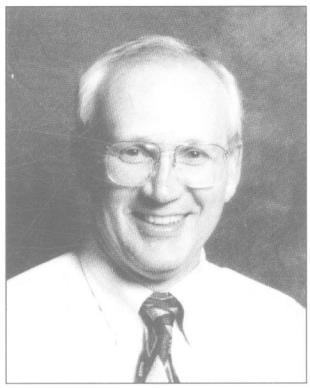
PATRICIA CHULADA



Dr. Patricia Chulada received her Ph.D. in Toxicology/Genetics at North Carolina State University in 1995. As a graduate student and post doctoral candidate under the direction of Dr. Robert Langenbach, Dr. Chulada conducted studies to distinguish the roles of prostaglandin H synthases 1 and 2 in inflammation, gastric toxicity and colon carcinogenesis using mice disrupted for the Ptgs-1 or Ptgs-2 genes.

Currently, Dr. Chulada is a Health Sciences Administrator in the Office of Clinical Research at the National Institute of Environmental Health Sciences where she designs and conducts clinical studies of genetic / environmental interactions as they relate to human disease.

RAYMOND N. DUBOIS



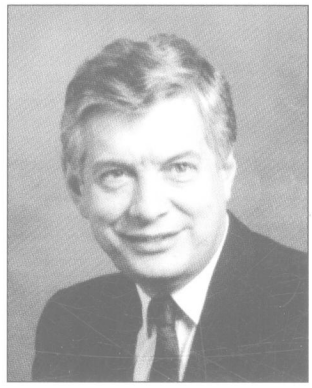
Dr. DuBois is the Director of Gastroenterology at the Vanderbilt University Medical Center in Nashville Tennessee. He also serves as Associate Director of the Vanderbilt-lngram Cancer Center. He received his M.D. and Ph.D. degrees from the University of Texas. He completed residency training and his Gastroenterology fellowship at The Johns Hopkins Hospital in Baltimore, Maryland. He currently holds the Mina Wallace Chair of Gastroenterology and Cancer Prevention at Vanderbilt University.

Dr. DuBois has contributed to many textbooks and is on the editorial board of several biomedical journals including Cancer Research and the Journal of Biological Chemistry. He has published numerous original and review articles on the pathogenesis of colorectal cancer. He is a member of several professional organizations, including the American Society for Clinical Investigation, American Association of Physicians, and was recently inducted as an honorary member of the Royal College of Physicians in London. He serves on the American Gastroenterological Association council as vice-chair of the section on Gastrointestinal Oncology. He currently serves as President of the Gastroenterology Research Group and is President-elect of the Southern Society for Clinical Investigation.

He was awarded the American Gastroenterological Association Young Investigator Award in 1996 and the American Federation for Medical Research Outstanding Investigator Award in April of this year. He was co-chairman of the progress review group committee on colorectal cancer research at the National Cancer Institute and is on the scientific advisory board for the National Colorectal Cancer Research Alliance which was recently founded by Katie Couric to raise public awareness and research support for colorectal cancer.

His research interests are focused on understanding the etiology of colorectal cancer and devising better ways to prevent, detect and treat this disease.

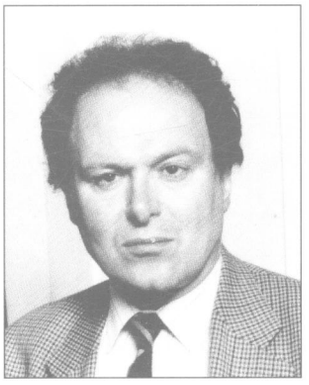
ALLAN R. RONALD



Allan Ronald is Professor Emeritus in the Departments of Internal Medicine and Medical Microbiology at the University of Manitoba. He completed his training in Infectious Diseases, Internal Medicine, and Microbiology in 1968 in Baltimore and Seattle and returned to Winnipeg. Over the next three decades he developed an Infectious Disease Training Program that has trained over 80 clinicians in the discipline, built up a strong Section of Infectious Diseases, and carried out research on urinary infection and sexually transmitted infections.

Since 1980 he has worked part-time in Nairobi, Kenya investigating the epidemiology of HIV and other STDs. He has over 400 publications. He has also served his University as Chair of Internal Medicine, Medical Microbiology, and Associate Dean (Research) for almost 20 years. He has received many recognitions including Distinguished Professor from the University of Manitoba, Officer of The Order of Canada, and Fellowship in the Royal Society of Canada. He is currently active as a consultant within Canada and internationally and continues to see patients as an internist and an Infectious Disease consultant.

NICHOLAS A. WRIGHT



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August 1977 — December 1979
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Royal Postgraduate Medical School;

March 1991 — September 1996
Director of Clinical Research,
Imperial Cancer Research Fund.

DEFICIENCY OF COX-1 OR COX-2 REDUCES INTESTINAL CANCER IN GENETICALLY PREDISPOSED OR CARCINOGEN TREATED MICE

Patricia Chulada

Two isoforms of cyclooxygenase(COX) are known and most studies have implicated COX-2, rather than COX-1, as the isoform involved in colon carcinogenesis. In the present study, we show that homozygous disruption of Ptgs-1, as well as of Ptgs-2 (genes coding for COX-1 or COX-2), reduced polyp formation in Min/+ mice by 74% and 85%, respectively. A statistically significant gene dosage effect was observed for Ptgs-1, but not for Ptgs-2. Using immunohistochemistry, only COX-1 protein was detected in normal intestinal tissue of Min/+ mice. In polyps, both COX-1 and variable levels of COX-2 protein, were detected. However the COX-2 was detectable in interstitial cells of the polyp rather than epithelial cells. Intestinal PGE2 levels were reduced by 99% in COX-1(-/-) mice, which identified COX-1 as the source of PGE2 in normal intestinal tissue. PGE2 levels were increased in polyps compared to normal tissue, and data from Ptgs-2 deficient mice indicated that the increased PGE2 was attributable to COX-2. The life spans of both COX-1(-/-) and COX-2(-/-)Min/+ mice were increased compared to Min/+ mice. Thus, the results indicate that COX-1, as well as COX-2, plays a key role in intestinal tumorigenesis and that COX-1 may also be a target for the chemotherapeutic effects of NSAIDs.

COX-2 AND COLON CANCER: MOLECULAR MECHANISMS

Raymond N. DuBois

Several groups have demonstrated an increase in prostaglandin endoperoxide synthase-2 or cyclooxygenase-2 (COX-2) expression in a variety of cancers (colon, skin, lung, breast, bladder, prostate). The presence of COX-2 in human lung and colon cancers has been associated with a negative clinical prognosis. Chronic use of nonsteroidal anti-inflammatory drugs (NSAIDs) results in a 40-50% reduction in risk for colorectal cancer. Nonselective NSAIDs inhibit both COX-1 and COX-2. COX-2 has been shown to play a role in rodent models of intestinal carcinogenesis and treatment with selective COX-2 inhibitors, such as celecoxib, blocks the growth of a variety of cancers such as colon, skin, head/neck and bladder. We evaluated the contribution of host-derived COX-1 and COX-2 in tumor growth using both genetic and pharmacologic approaches. We have reported a decrease in vascular density in tumors grafted onto COX-2 -/- mice when compared to those grown in wild type or COX-1 -/- animals.

Recently, we have focused our research on obtaining a better understanding of the role of prostaglandins generated by the cyclooxygenase enzymes. The mechanism(s) by which nonsteroidal anti-inflammatory drugs (NSAIDs) exert their anti-neoplastic effects are not completely understood. We found that prostaglandin E2 (PGE2) affects the morphology and growth of colorectal carcinoma cells. Treatment of colorectal carcinoma cells with PGE2 resulted in rapid cell spreading, increased motility and increased invasiveness.

The prostaglandin EP4 receptor signaling pathway was found to play an important role in regulating these morphogenic effects. PGE2 treatment also induced the activation of extracellular signal-regulated kinase (ERK) and protein kinase B (Akt/PKB) that were essential for the stimulatory effect of PGE2. Our results suggest that PGE2 may act in both a paracrine and/or autocrine manner to enhance the malignant potential of colorectal carcinoma cells via activation of major signal transduction pathways. We have also found that endogenously produced PGI2 serves as an excellent ligand for PPAR, a nuclear receptor which regulates the expression of several genes.

GATEKEEPER, CARETAKER, LANDSCAPER — JOBS FOR THE BOYS?

Nicholas A. Wright

We are now used to referring to the Vogelstein and Kindlier classification of the molecular defects in colorectal carcinoma. Thus APC classically acts as the 'gatekeeper', while the DNA mismatch repair (MMR) genes hMSH2 or hMLH1 are the paradigm of the 'caretaker'. The juvenile polyposis syndrome dPS) is a rare Mendelian disorder in which individuals have typical hamartomatous polyps within the gastrointestinal tract. The stromal element of the polyps has classically been thought to be the clonal component, although epithelial malignancies (largely gastrointestinal cancers) occur more frequently than expected in JPS patients. Germ-line mutations in SMAD4 (DPC4) account for about a third of JPS cases. It has been postulated that the apparent paradox of a stromal lesion predisposing to epithelial malignancy is the 'landscaper' effect: an abnormal stromal environment affects the development of adjacent epithelial cells, and the resulting regeneration of damaged epithelium leads to an increased risk of cancer. We have found allele loss at the SMAD4 locus on 18q in polyps from JPS individuals with a germ-line SMAD4 mutation indicating that SMAD4 is acting as a tumour suppressor gene in JPS polyps, as it does in sporadic cancers of the gastrointestinal tract. Interphase fluorescence in situ hybridisation showed deletion of one copy of SMAD4 in the epithelial component of JPS polyps, but not in the inflammatory infiltrate. Fluorescence in situ hybridisation also indicated that a single copy of SMAD4 was present in stromal fibroblasts of JPS polyps. Thus, biallelic inactivation of SMAD4 occurs in both the epithelium and some of the stromal cells in these lesions, indicating a common clonal origin and suggesting that the precursors of these lesions are laid down very early in development, before epithelial: mesenchymal differentiation in the intestinal anlage, with later clonal expansion. Or the mutation could occur later in life as a stem cell with plasticity of a greater degree than is usually considered, as has been recently shown for haemopoietic stem cells, which can differentiate into hepatocytes. Epithelial malignancies almost certainly develop in juvenile polyposis through direct malignant progression of the epithelial component of the hamartomas. SMAD4/DPC4 probably acts as a 'gatekeeper' tumour suppressor in juvenile polyps, and there is no need to invoke a 'landscaper hypothesis.' Thus the 'gatekeeper' and 'caretaker' positions appear safe for the time being, but whether or not openings are available for landscapers in other terrains remains to be

ANORECTAL CANCER AND HUMAN PAPILLOMAVIRUS

Allan R. Ronald

Squamous cell cancers ofthe anal canal account for 1.5% of G.I. cancers. They present with rectal bleeding, anal discomfort or a sensation of an anorectal mass. Risk factors are HPV infection/anal genital warts (50%), receptive anal intercourse; history of more than 10 sexual partners; cervical cancer in women or in partners of heterosexual men; solid organ transplantation; HIV infection.

Of 388 patients with anal cancer, 88% have HPV DNA (73% HPV-16). Renal allografts and other solid organ recipients have a 100-fold increase in anal cancer. Cure of anal cancer occurs in 60-80% of patients with a combination of radiation, fluorouracil, and mitomycin. The use of cisplatin in this combination is under study. Less than 20% of patients require a colostomy; recurrence rates have been 15-25%. Local recurrences are managed by aggressive surgical resection with cure in about 50% of patients; distant metastases should be considered for clinical trials as optimal therapy is not known.

Prevention needs to become the strategy for the future. HPV vaccines are currently in trials for both prophylaxis and treatment. Diagnostic screening programs for HPV and early lesions also need to be further evaluated.

CHEMOPREVENTION OF COLORECTAL CANCER

Raymond N. DuBois

Population-based studies indicate a 40-50% reduction in mortality from colorectal cancer in persons using non-steroidal anti-inflammatory drugs (NSAIDs) on a regular basis (Smalley and DuBois, 1997). These effects are seen when "anti-inflammatory" or even lower doses of NSAIDs are given. Colorectal cancer is a major cause of death from cancer in industrialized countries, claiming more than 55,000 lives in the U.S. this year. Environmental and dietary factors play an important role in the etiology of this disease as well as the known genetic components. Research efforts have been focused on understanding the molecular basis for the chemoprotective effects associated with use of aspirin and other NSAIDs. NSAIDs inhibit both cyclooxygenase-1 (COX-1) and cyclooxygenase-2 (COX-2) activity (Williams et al., 1999). NSAIDs are toxic to cultured cells when given at very high concentrations (>100 M) Since COX-2 levels are increased in a number of solid tumors, this enzyme may serve as a molecular target for cancer prevention (Eberhart et al., 1994; Sheng et al., 1997). Recent clinical studies indicate that presence of COX-2 in human lung and colon cancers is associated with a negative clinical prognosis (Achiwa et al., 1999; Sheehan et al., 1999). Therefore, COX-2 inhibitors are currently being evaluated for the prevention and/or treatment of cancer in humans (Steinbach et al., 2000; Williams et al., 2000). The objectives of my presentation will be to review the data with regard to the effects of NSAIDs and other factors on colorectal cancer risk, discuss the potential mechanisms involved and present clinical studies underway to determine if these agents will be useful in humans. Current recommendations for screening and disease management will also be discussed.

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Merck Sharp & Dohme (Asia) Ltd — Dinner Symposium

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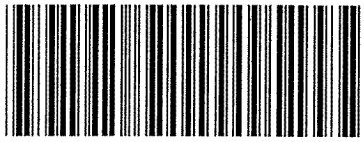
Last but not least, we would like to thank all speakers, chairmen and delegates for their participation and contribution.

NOTES

NOTES



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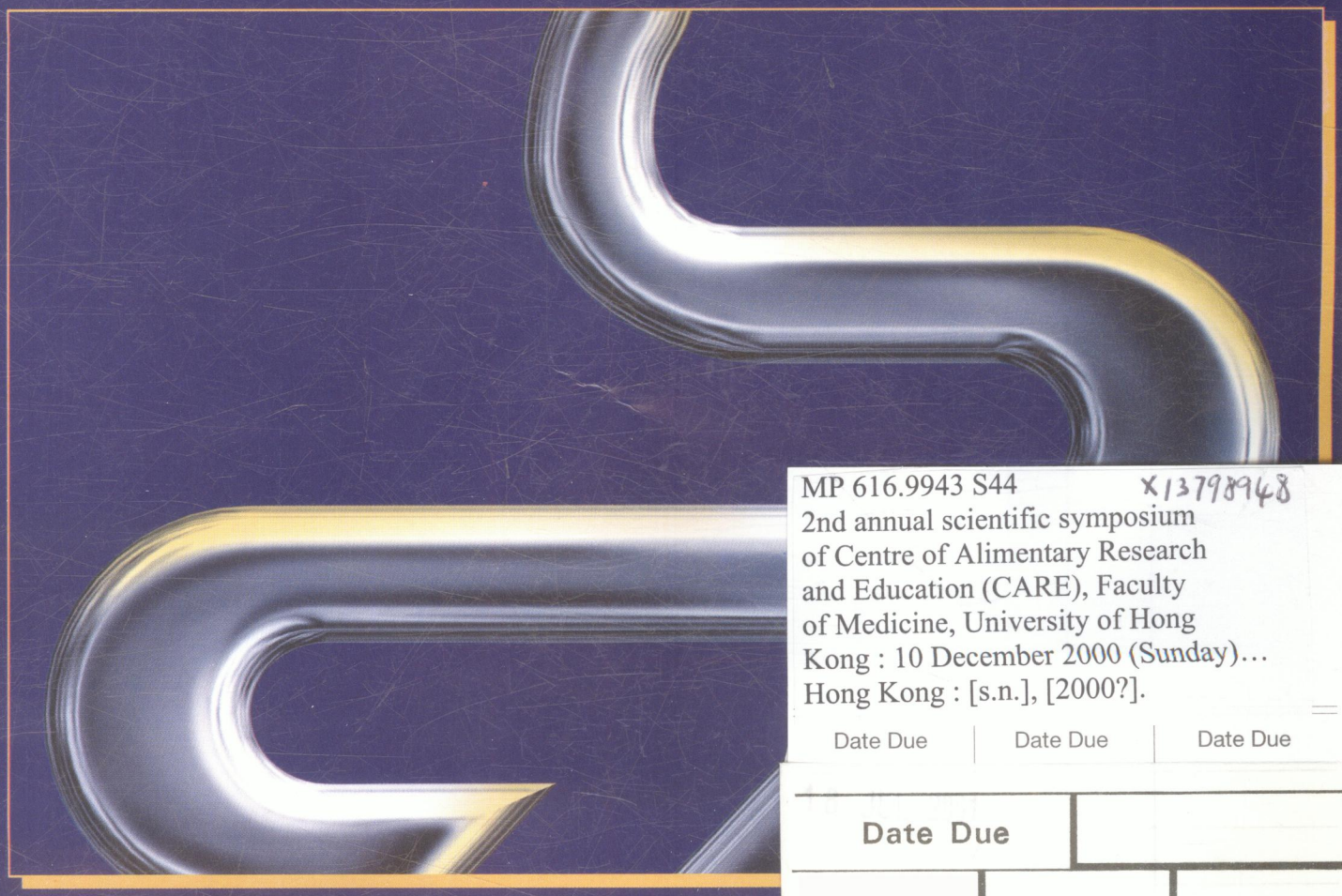

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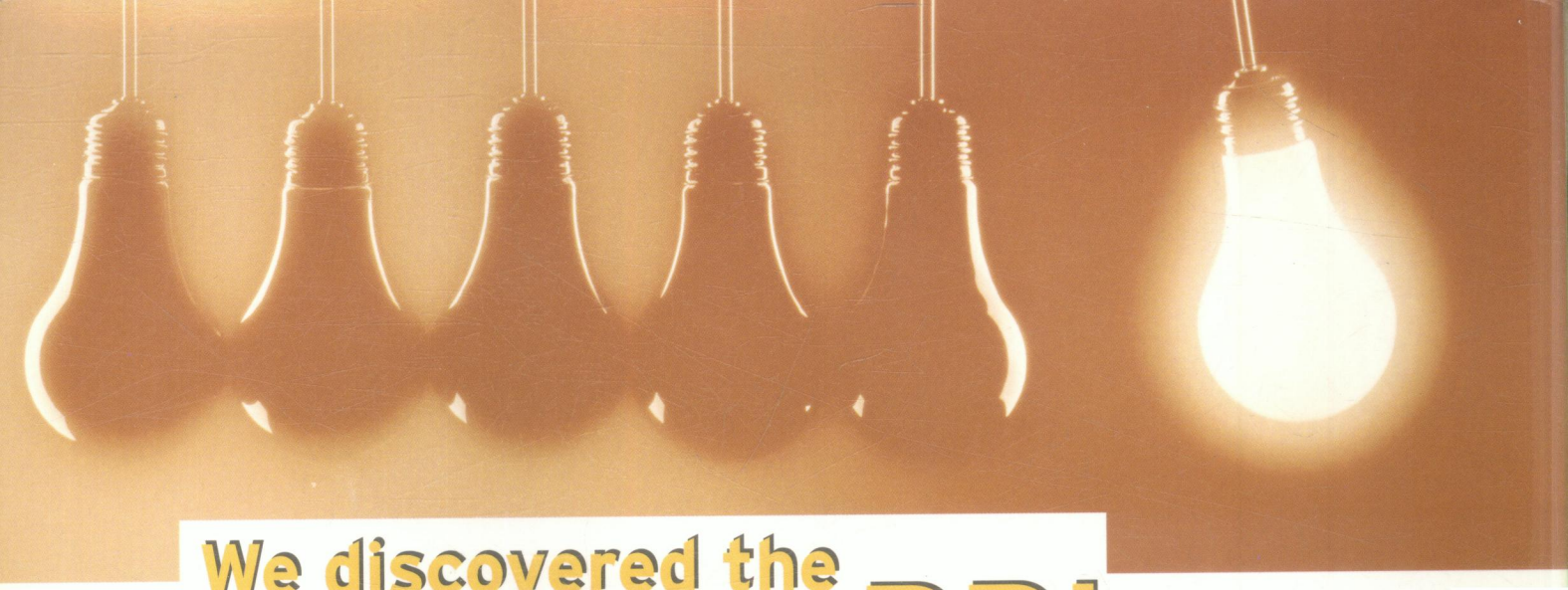

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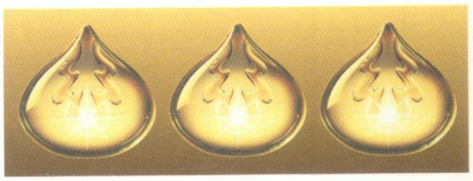

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Reference:

1. Anderberg EK, et al. Bioequivalence between omeprazole MUPS® tablets and omeprazole capsules. *Gastroenterology* 1998;**114**(4:2):A56.

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