



World Cancer
Research Fund
International

Nutrition, Physical Activity & Cancer Prevention: Current Challenges, New Horizons

Abstract Book



12-13 September 2010

**An international two-day Conference
on Science and Policy**

Royal College of Physicians, London, UK

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The conference is approved for Continuous Professional Development by the Royal College of Physicians and the British Dietetic Association.

Welcome and introduction

It is with great pleasure that we welcome you to the first WCRF International scientific conference - *Nutrition, Physical Activity & Cancer Prevention: Current Challenges, New Horizons*. We hope this new conference will be the first of a series that will draw the world's best researchers to help discuss new research in the field of food, nutrition, physical activity and cancer.

In 1997 we published a major report on diet and cancer, which changed the landscape for research in this area. Eight years ago, we started the process that led to the follow up to this, the WCRF/AICR Second Expert Report, *Food, Nutrition, Physical Activity and the Prevention of Cancer: a Global Perspective* in 2007 and its companion *Policy Report, Policy and Action for Cancer Prevention* in 2009. Both reports have had a global impact and have helped us spread the vital message that cancer is preventable. We communicate this message to scientists, policy makers, health professionals and the public through our global network research and education programmes in the UK, USA, The Netherlands, France and Hong Kong.

Since the foundation of our network in 1982, with the American Institute for Cancer Research (AICR), we have funded over £77 million of research. Some of the latest findings will be presented at the conference.

Just as our Expert Reports have galvanised research in the area, we hope that this new conference will “set the bar” even higher as we seek to understand further how the everyday choices that people make can reduce their risk of cancer.

Whatever your interest, we hope you come away from the conference with new knowledge that you can apply in practice, as well as renewing old acquaintances and making new ones. The conference venue, the Royal College of Physicians, is located close to all the amenities of central London and just next to Regent's Park, one of our most beautiful royal parks.

We look forward to seeing you here in September.

With best wishes,



A handwritten signature in black ink that reads "Marilyn Gentry".

Marilyn Gentry

President/CEO WCRF global network



A handwritten signature in black ink that reads "Kathryn Allen".

Kathryn Allen

Director, Science and Communications WCRF International

Scientific Programme and Conference Schedule

Sunday 12 September

9.00 - 10.50 Registration

10.50 - 13.00 OPENING PLENARY SESSION –

Setting the Scene *Wolfson Theatre*

Chair: Prof Martin Wiseman, World Cancer Research Fund International, UK

10.50 - 11.00 Welcome

Marilyn Gentry, President, World Cancer Research Fund Global Network, UK
Dr Kate Allen, World Cancer Research Fund International, UK

11.00 - 11.40 Nutrition & Cancer Around the World – Opportunities and Challenges

Dr Francesco Branca, World Health Organization, Switzerland

11.40 - 12.20 Obesity, Physical Activity and Cancer – Global Perspectives

Prof W Philip T James, International Obesity Task Force, UK

12.20 - 13.00 How Early should we be Concerned with Cancer Prevention?

Prof Ricardo Uauy, London School of Hygiene and Tropical Medicine, UK and University of Chile, Santiago, Chile

13.00 - 14.20 Lunch and Poster Session

Osler/Long Room

14.20 - 15.40 PARALLEL SESSION –

Research Highlights and Directions

A) Preferred Paper Session 1 *Dorchester Library*

Chair: Dr Kate Allen, World Cancer Research Fund International, UK

Oral Presentations from Selected Abstracts:

14.20 - 14.40 Obesity, Inflammatory Markers and Endometrial Cancer Risk: Results from the EPIC Study

Laure Dossus, German Cancer Research Center (DKFZ), Germany

14.40 - 15.00 Cured Meat Promotion of Colon Carcinogenesis is Suppressed by Calcium and α -tocopherol

Dr Fabrice Pierre, ToxAlim ENVT-INRA, France

15.00 - 15.20 Mendelian Randomization to Explore Associations of Folate, Vitamin B12 and Homocysteine with Prostate Cancer Initiation and Progression

Simon Collin, University of Bristol, UK

15.20 - 15.40 The Role of One-Carbon Metabolism in Lung Cancer: Disentangling Metabolic Pathways with Structural Equation Models

Valéria Baltar, University of Sao Paulo, Brazil

B) Workshop 1 – Research

Directions *Wolfson Theatre*

Chair: Prof Massimo Pignatelli, University of Bristol, UK

14.20 - 14.40 WCRF International Research Programme

Dr Panagiota Mitrou, World Cancer Research Fund International, UK

14.40 - 15.00 WCRF/AICR Continuous Update Project

Dr Teresa Norat, Imperial College London, UK

15.00 - 15.20 Research Directions in Asia

Professor TH Lam, University of Hong Kong, Hong Kong

15.20 - 15.40 Panel Discussion

Chair: Prof Massimo Pignatelli, University of Bristol, UK
Prof Ellen Kampman, Wageningen University, The Netherlands
Prof T H Lam, University of Hong Kong, Hong Kong
Dr Panagiota Mitrou, World Cancer Research Fund International, UK
Dr Teresa Norat, Imperial College London, UK

15.40 - 16.00 Coffee Break *Osler/Long Room*

16.00 - 17.20 PARALLEL SESSION – Drawing out Key Priorities

A) Science Hot Topics Session 1 *Wolfson Theatre*

Chair: Prof Hilary Powers, University of Sheffield, UK

16.00 - 16.40 Vitamin D and Cancer

Dr Mazda Jenab, International Agency for Research on Cancer (IARC), France

16.40 - 17.20 Epigenetics, Nutrition and Bowel Cancer Risk

Prof John Mathers, Newcastle University, UK

B) Policy and Practice Hot Topics Session 1

Council Chamber

Chair: Prof Annie Anderson, University of Dundee, UK

16.00 - 16.40 What Evidence do you Need for Public Health Policy?

Prof Mike Kelly, National Institute for Health and Clinical Excellence (NICE), UK

16.40 - 17.20 Folic Acid Fortification, Good or Bad?

Prof Ellen Kampman, Wageningen University, The Netherlands

17.20 - 18.30 Conference Reception *Osler/Long Room*

Drinks reception and walk around Royal College of Physicians gardens (weather permitting)

Monday 13 September

9.30 - 10.00 PLENARY SESSION –

Challenges Overview *Wolfson Theatre*

Chair: Dr Kate Allen, World Cancer Research Fund International, UK

Global Inequalities – Implications for Research, Practice and Policy

Prof Sir Michael Marmot, University College London, UK (pre-recorded)

10.00 - 10.20 Coffee Break *Osler/Long Room*

10.20 - 11.40 PARALLEL SESSION –

Key Priority Challenges and Opportunities

A) Science Hot Topics Session 2 *Wolfson Theatre*

Chair: Prof Kay-Tee Khaw, University of Cambridge, UK

10.20 - 11.00 Cancer Survivors: What we Know, What we Need to Know

i) Asian Perspective

Prof Josette Chor, Chinese University of Hong Kong, Hong Kong

ii) Western Perspective

Dr Michelle Harvie, University of Manchester, UK

11.00 - 11.30 Metabolic Syndrome and Cancer Risk

Prof Pär Stattin, University of Umeå, Sweden

B) Policy and Practice Hot Topics Session 2

Council Chamber

Chair: Dr Susan Jebb, Medical Research Council Human Nutrition Research Unit, Cambridge, UK

10.20 - 10.45 Local Action in Brazil

Fabio Gomes, National Cancer Institute (INCA), Brazil

10.45 - 11.10 Policy and Practice in Scotland

Prof Annie Anderson, University of Dundee, UK

11.10 - 11.35 Nutrition, Physical activity and the Cancer Reform Strategy

Prof Mike Richards, Department of Health, UK

11.40 - 12.40 PANEL DISCUSSION – The

Challenge in Lower-income Countries *Wolfson Theatre*

Chair: Prof Alan Jackson, University of Southampton, UK

11.40 - 12.00 Research Challenges in Lower-income Countries

Dr Isabelle Romieu, International Agency for Research on Cancer (IARC), France

12.00 - 12.20 Challenges for Policy & Practice in Lower-income Countries

Prof Prakash Shetty, University of Southampton, UK

12.20 - 12.40 Panel Discussion

Chair: Prof Alan Jackson, University of Southampton, UK

Cary Adams, International Union Against Cancer (UICC), Switzerland

Dr Mazda Jenab, International Agency for Research on Cancer (IARC), France

Prof Prakash Shetty, University of Southampton, UK

Prof Ricardo Uauy, London School of Hygiene and Tropical Medicine, UK and University of Chile, Santiago, Chile

12.40 - 13.40 Lunch and Poster Session

Osler/Long Room

13.40 - 15.00 PARALLEL SESSION

A) Proffered Paper Session 2 *Wolfson Theatre*

Chair: Dr Rachel Thompson, World Cancer Research Fund International, UK

Oral Presentations from Selected Abstracts:

13.40 - 14.00 Pre-diagnostic Plasma Enterolactone Levels and Survival Among Women with Breast Cancer

Dr Anja Olsen, Danish Cancer Society, Denmark

14.00 - 14.20 Dietary Soy Intake and Changes of Mammographic Density in Premenopausal Chinese Women

Prof Suzanne Ho, Chinese University of Hong Kong, Hong Kong

14.20 - 14.40 Adherence to the WCRF/AICR Cancer Prevention Recommendations in the EPIC study

Dr Dora Romaguera, Imperial College London, UK

14.40 - 15.00 EU Livestock Subsidies Effect on Red Meat Consumption

Robert Pederson, European Public Health and Agriculture Consortium, Belgium

B) Workshop 2 – Media Reporting of Science

Dorchester Library

Chair: Richard Evans, World Cancer Research Fund UK, UK

13.40 - 14.00 Reporting of Public Health and Cancer Risk - Overview

Richard Evans, World Cancer Research Fund UK, UK

14.00 - 14.20 Media Reporting of Cancer Risk

Dr Ben Goldacre, London School of Hygiene and Tropical Medicine, UK

14.20 - 14.40 Media Reporting of Disease Risk: a Scientist's Perspective

Dr Alan Dangour, London School of Hygiene and Tropical Medicine, UK

14.40 - 15.00 Panel Discussion

Chair: Richard Evans, World Cancer Research Fund UK, UK
Dr Ben Goldacre, London School of Hygiene and Tropical Medicine, UK/Independent Science Writer
Dr Alan Dangour, London School of Hygiene and Tropical Medicine, UK

15.00 - 15.20 Coffee Break *Osler/Long Room*

15.20 - 17.15 CLOSING PLENARY SESSION*Wolfson Theatre*

Chair: Prof Martin Wiseman, World Cancer
Research Fund International, UK

15.20 - 16.15 Two-Way Translational Research for Understanding Cancer Aetiology

Dr Chris Wild, International Agency for

Research on Cancer (IARC), France

16.15 - 16.55 Preventability and Potential Public Health Impact

Prof Elio Riboli, Imperial College London, UK

16.55 - 17.15 Closing Remarks

Prof Martin Wiseman, World Cancer Research Fund
International, UK

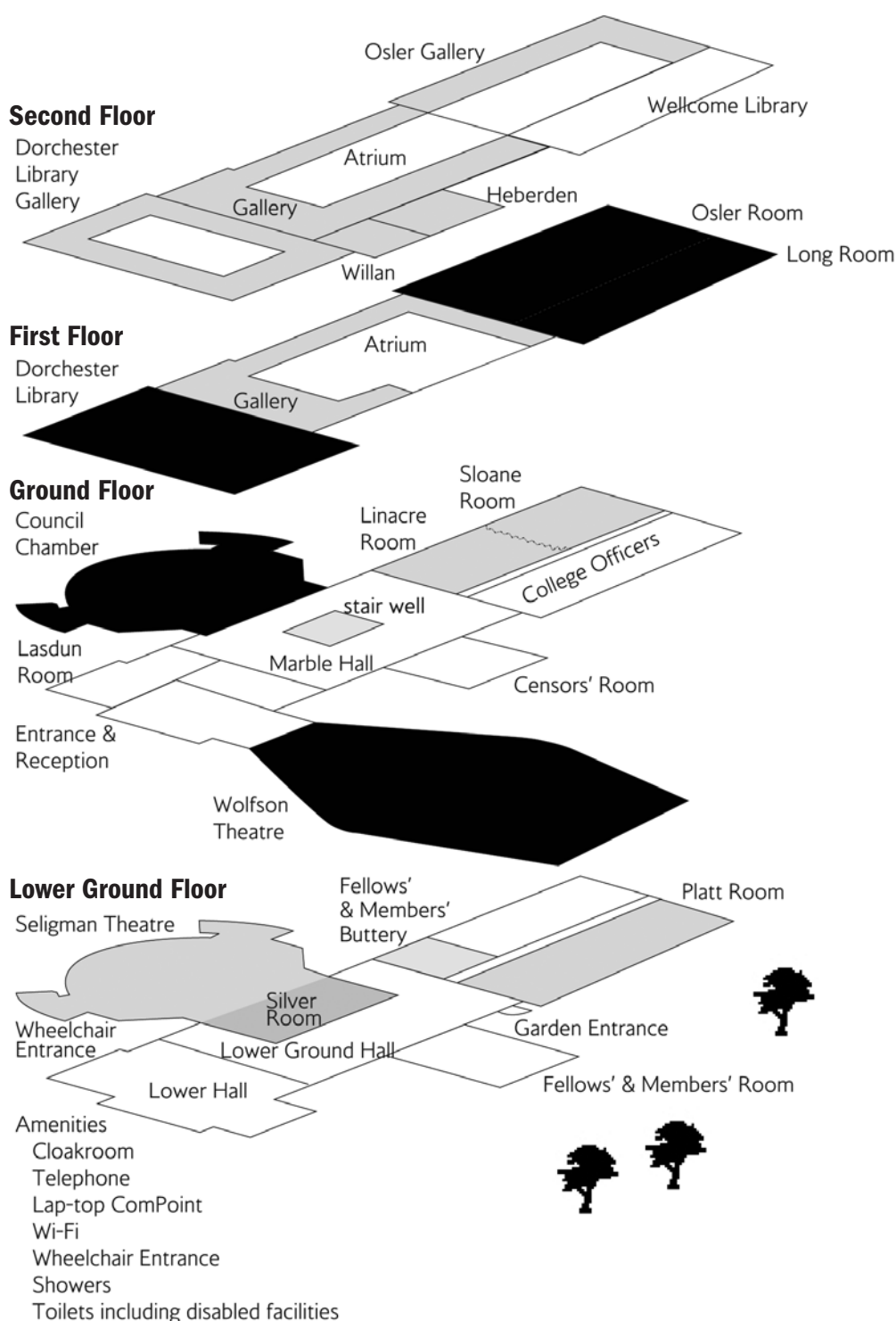
Venue Information

The Royal College of Physicians (RCP) is a convenient and attractive located venue in the centre of London, overlooking Regent's Park and easily accessible by all forms of transport.

The Royal College of Physicians
11 St Andrews Place,
Regent's Park,
London
NW1 4LE

Tel: +44 (0) 20 7935 1174

Web: www.rcplondon.ac.uk



Speaker and Chair Information - in order of appearance in the programme



Professor Martin Wiseman
World Cancer Research Fund International, UK

Professor Martin Wiseman is currently an independent nutrition consultant, and Medical and Scientific Adviser at the World Cancer Research Fund International. He was project director for the 2007 WCRF/AICR expert report Food, Nutrition, Physical Activity, and the Prevention of Cancer: a Global Perspective, and the 2009 companion report, Policy and Action for Cancer Prevention.

In addition, he is a visiting professor at Southampton University. From 1999-2000 he was managing director, food and regulatory affairs, at Burson Marsteller, a global PR company. Prior to that from 1986 he was head of the Nutrition Unit at the Department of Health, where he had responsibility for nutrition science, including the work of COMA, the Committee on Medical Aspects of Food and Nutrition Policy; for nutrition surveys; and for advising on and implementing nutrition policy. He qualified from Guy's Hospital in London in 1975. He became a member of the Royal College of Physicians in 1977 and followed a traditional career path as a general physician until 1981 when he developed an interest in clinical research. He has published papers on diabetes and kidney function and the effects of nutrition on them. He moved to the Department of Health in 1986 but still retains an appointment within the NHS, where he continues with clinical activities in a diabetes clinic.

He was Honorary External Relations Officer of the Nutrition Society from 2001 to 2006. He is Chair of the Management Team of the Intercollegiate Group on Nutrition of the Academy of Medical Royal Colleges, and is a Fellow of the Royal College of Physicians and of the Royal College of Pathologists.



Dr Francesco Branca
World Health Organization (WHO), Geneva

Dr Branca is the Director of the Department of Nutrition for Health and Development at the World Health Organization (WHO), where he has been since 2008. Before that, between 2005 and 2008, he was the Regional Adviser for Nutrition and Food Security for WHO Regional Office for Europe.

Dr Branca studied Medicine and Surgery at the Università Cattolica del Sacro Cuore in Rome, where he later specialised in Diabetology and Metabolic Diseases in 1987. He gained his PhD in Nutrition at Aberdeen University 1995. For more than 10 years, between 1986-1998, he has worked at Istituto Nazionale di Ricerca per gli Alimenti e la Nutrizione (INRAN), in Italy, first as a scientist and later as a senior scientist. During his career he has designed public health nutrition teaching modules for postgraduate medical students and has designed and implemented several studies on the effects of food and nutrients on human health at the different stages of the life cycle. He published 8 books and several original scientific publications.



Professor W. Philip T. James
International Obesity Task Force, UK

Professor James is President of the International Association for the Study of Obesity (IASO) working with WHO, the Commonwealth Secretariat and other organizations to develop strategies for the prevention and management of obesity and its associated diabetes, cardiovascular diseases and cancers. He chaired and wrote the UN commission's report on global issues in nutrition. In 1996 he established the International Obesity TaskForce (IOTF), which was responsible for drafting the first WHO Technical Report (2000) on the prevention and management of obesity. This followed his WHO EURO and WHO global reports on diet and the prevention of chronic diseases and a series of proposals for the UK Department of Health and Royal Colleges of Physicians on the importance of prevention and appropriate management of obesity including more recently childhood obesity. He also produced the Food Standards Agency plan for Tony Blair and the plan for the EU's Food Authority both of which he specified should include nutritional issues as a principal activity.

Professor James qualified in physiology and medicine at University College, London. After various internships and residency posts in London he worked at the UK's MRC Unit in Tropical Metabolism in Jamaica and the Massachusetts General in Boston before taking over nutritional teaching at the London School of Hygiene. He then established the MRC Dunn Clinical Nutrition Centre in Cambridge before becoming Director of the Rowett Research Institute in Aberdeen.



Professor Ricardo Uauy
London School of Hygiene and Tropical Medicine, UK and University of Santiago, Chile

Professor Ricardo Uauy M.D. Ph.D. born Dec/29/1948, Medical Doctor University of Chile/1972, Ph.D. Nutritional Biochemistry MIT/1977. Trained in Pediatrics Harvard Children's Hospital/Boston and Neonatology Yale New-Haven Hospital; Board Certified in Pediatrics and Neonatal-Perinatal Medicine (USA). Past-President of the International Union of Nutrition Sciences (IUNS) 2005-09. Director of INTA University of Chile 1994-2002. Presently, Professor of Public Health Nutrition at Institute of Nutrition (INTA) University of Chile and London School of Hygiene and Tropical Medicine, U.K.

Has participated as expert in multiple WHO/FAO expert committees (Protein Energy 81, Fats and Oils 93, FBDGs 95, Vitamin and Minerals 98, Nutrition in the Elderly 98, Human Energy needs 01, chair WHO/FAO Nutrition Diet and Chronic Disease 02, and Global Strategy WHO/03; WHO/FAO Scientific Update on CHO 06 and on Trans Fats 07, chair FAO/WHO Expert Consultation on Fats and Fatty Acids in Human Nutrition in 08. McCollum Lecture award ASN (USA) 2000, member Chilean Academy of Medicine 2002. Lawton Chiles International Lecturer Award NIH 2003, Spanish Nutrition Society Award and PAHO/WHO Abraham Horwitz award for Leadership in Inter-American Health 2005; in 2006 received the Kellogg's International Nutrition Award from the American Nutrition Society ASN; in 2008 the Rank Lecture Award/UK Nutrition Society and the 2009 British Nutrition Foundation Prize. Member of the Scientific Panel and executive committee of the WCRF World Cancer Research Foundation: Diet Nutrition and Physical Activity Prevention of Cancer Publication in 2007 and the corresponding Policy Report 2008; over 337 scholarly peer reviewed scientific publications.



Dr Kathryn Allen
World Cancer Research Fund International, UK

Dr Kathryn Allen works as Director (Science and Communications) at WCRF International. She is responsible for the scientific programmes of WCRF International and helps facilitate collaboration across the WCRF global network on science-related activities. Kate is also responsible for the development and management of the network websites and is particularly interested in developing and promoting the educational activities of WCRF International, both electronically and via more traditional routes.

Previously Kate worked at The Institute of Cancer Research in London and Sutton, where she set up an Interactive Education Unit developing electronic materials for scientists, healthcare professionals, students, patients and the general public. Prior to that she worked at Medi Cine International, a medical education agency, where she developed educational materials across all media, mainly for specialist physician audiences.

Before joining Medi Cine, Kate worked as a scientist at the Institute of Neurology and the National Hospital for Neurology and Neurosurgery at Queen Square, London, looking at the molecular basis of energy metabolism during stroke. Her PhD studies, for which she was awarded the Queen Square prize, were undertaken at the Royal College of Surgeons of England and involved the use of magnetic resonance spectroscopy and imaging to study brain function after stroke.

In addition to her work at WCRF International, Kate retains an honorary position with the Institute of Cancer Research.



Professor Massimo Pignatelli
University of Bristol, UK

Professor Pignatelli is Full Professor of Histopathology at the University of Bristol (UK). He is Fellow of the Royal College of Pathologists (London, UK) and Council Member and Secretary of the European Association of Pathology Chairs and Programme Directors.

His academic career has focused on research, educational and clinical activities in cell biology and pathology resulting in over 160 original articles, reviews and editorials. He has been a recipient of a Medical Research Council Clinician Scientist Fellowship and other research fellowships. His research programme is directed towards the development of novel tissue bio-markers to predict the behaviour of gastrointestinal tumours and to exploit their use in novel preventative and therapeutic cancer strategies.



Dr Panagiota Mitrou
World Cancer Research Fund International, UK

Dr Panagiota Mitrou received her MSc in Genetic Manipulation and Molecular Biology from the University of Sussex and her Ph.D. in Genetic Epidemiology from Cambridge University. She conducted doctoral research under the supervision of Professor Sheila Rodwell (professionally known as Professor Sheila Bingham) at the Dunn Human Nutrition Unit, Cambridge, where she investigated polymorphisms in xenobiotic and folate metabolism genes in relation to colorectal adenoma and cancer risk. She joined the Division of Cancer Epidemiology and Genetics (DCEG) within the National Cancer Institute (NCI) in the US, as a visiting fellow in 2005. At NCI, she worked on dietary risk factors of cancer, including the role of dietary patterns, within the framework of large, epidemiological studies.

Dr Mitrou is currently the Deputy Head of Science (Development, Strategy and Programmes) at the World Cancer Research Fund International with main responsibilities the strategic development and management of effective and efficient research and education programmes.

Also, she is an Honorary Visiting Research Fellow at the MRC Centre in Nutritional Epidemiology of Cancer Prevention and Survival (CNC), University of Cambridge, with main research interests the role of lifestyle factors including dietary factors in carcinogenesis related pathways.



Dr Teresa Norat
Principal Research Fellow, Department of Epidemiology and Public Health,
Faculty of Medicine, Imperial College London

Dr Norat is Epidemiologist working as Principal Research Fellow in the Department of Epidemiology and Public Health, Faculty of Medicine at Imperial College London since 2007. She worked from 1998 to 2006 in the Unit of Nutrition at the International Agency for Research on Cancer, Lyon. Her research focuses on the role of nutrition, lifestyle, metabolic factors and genetic factors in the aetiology of chronic diseases, in particular cancer. She is the Coordinator of the Continuous Update Project for World Cancer Research Fund at Imperial College. This project aims to provide a comprehensive and up to date depiction of scientific developments on the relationship between diet, physical activity, obesity and cancer. Since 1998 she is collaborating in the large European Prospective Investigation into Nutrition and Cancer (EPIC). Currently she is the Principal Investigator of a project aimed to investigate adherence to dietary recommendations for cancer prevention and cancer risk in EPIC. She also participates in the EPIC-Panacea project, which aims to investigate dietary and lifestyle determinants of weight changes in the population participating in EPIC.



Professor Tai Hing Lam
The University of Hong Kong, Hong Kong

Professor TH Lam graduated from the Faculty of Medicine, The University of Hong Kong with the MBBS degree in 1975. He got an MSc degree in medical sociology and an MSc degree in occupational medicine in 1980 and 81 respectively from The University of London. He obtained his MD degree by research from The University of Hong Kong in 1988. He is Chair Professor and Head of the Department of Community Medicine, The University of Hong Kong since 2000. He is Director of Public Health Research Centre as from July 2004. He was appointed Sir Robert Kotewall Professorship in Public Health, LKS Faculty of Medicine, University of Hong Kong in December 2007. He is Director of the School of Public Health since August 2009.

Professor Lam is a fellow of the Australasian Faculty of Occupational Medicine, the UK Faculty of Public Health, The London Faculty of Occupational Medicine, The Hong Kong Academy of Medicine (Community Medicine) and The Royal College of Physicians of Edinburgh. Professor Lam was President of the Hong Kong College of Community Medicine from 1997 to 2001. He was Vice-Chairman of Hong Kong Council on Smoking and Health and Co-Chair of Grant Review Board, Research Council, Health, Welfare and Food Bureau of the Hong Kong SAR Government. He was appointed Justice of Peace in July 2005 and conferred Honorary Fellowship of Hong Kong College of Community Medicine in September 2008. He has been appointed by the World Health Organization as short-term consultant, temporary advisor or expert participant for about ten occasions.

Professor Lam's research interests include occupational and environmental health, family planning and youth sexuality, adolescent health, the epidemiology of cancer, infectious diseases, cardiovascular and respiratory diseases and their risk factors, molecular epidemiology and health services research, with a major focus on tobacco and other lifestyle related diseases. He has published many papers in high-impact journals and was awarded a Commemorative Certificate and Medal by the World Health Organization in 1998 on achievement worthy of international recognition in promoting the concept of tobacco-free societies.



Professor Ellen Kampman
Wageningen University, The Netherlands

In January 2008, Ellen Kampman was appointed as Professor of Diet and Cancer at the Division of Human Nutrition of Wageningen University. Her research focuses on the role of diet in cancer etiology as well as in cancer progression. She is project leader of many national as well as international projects, and member of several committees and advisory groups on nutrition, epidemiology and public health. She published more than 100 papers in international scientific journals and serves as senior editor of 'Cancer Epidemiology Biomarkers and Prevention' of the American Association of Cancer Research. Professor Kampman was project leader of the SLR on colorectal cancer of the WCRF/AICR Second Expert Report.

She obtained her Master of Science degree in Human Nutrition from the Wageningen University in 1988 and her Doctor of Philosophy degree in cancer epidemiology from the University of Maastricht, the Netherlands in 1993. During her studies, she worked as a visiting scholar at the Departments of Nutrition and Epidemiology of the Harvard School of Public Health, Boston, USA (Prof. Willett). She was awarded a two-year fellowship from the Dutch Cancer Society for a postdoctoral period at the Fred Hutchinson Cancer Research Center in Seattle, USA (Professor Potter). In 1996, she moved back to the Netherlands, to work at the Division of Human Nutrition, Wageningen University.



Professor Hilary Powers
University of Sheffield, UK

Hilary J Powers is Professor of Nutritional Biochemistry and Head of the Human Nutrition Unit at the University of Sheffield, UK. She studied under Professor John Waterlow at the London School of Hygiene and Tropical Medicine, and continued her research career at the Medical Research Council's Dunn Nutrition laboratory in Cambridge. At this time much of her research into the functional effects of B vitamin deficiencies was carried out in the MRC field laboratories in The Gambia, and has had relevance for optimum nutrition in developing countries. She left Cambridge to help establish the Human Nutrition Unit at Sheffield University, which now runs a successful postgraduate teaching programme and a flourishing research program.

Micronutrients still figure prominently in her research portfolio which focuses on the nature of and mechanisms for involvement of B vitamins in the pathophysiology of disease, including cancer and cardiovascular disease. She has served on several Committees for external bodies and continues to work in this capacity with the Biotechnological and Biological Sciences Research Council and the World Cancer Research Fund International and is an advisor for micronutrient analyses for the Food Standards Agency.



Dr Mazda Jenab
International Agency for Cancer Research (IARC), France

Dr Jenab gained a BSc (Hons) in 1992 and an MSc in 1995 at the University of Toronto, Canada, where he specialised in Nutritional Sciences and Human Biology. He later studied for a PhD at the same University to pursue his interest in Nutritional Science. He has since worked as a scientist in the field of nutritional epidemiology, first at the Department of Clinical Research and Regulatory Affairs, in Markham, Ontario, Canada; and later for the Nutrition and Hormones Group at the International Agency for Research on Cancer (IARC) in Lyon, France. Since 2007 he has moved to the Lifestyle, Environment and Cancer Group at IARC. His main research interest is the influence of diet, nutrition and genetics on cancer risk and aetiology.



Professor John C. Mathers
Newcastle University, UK

Professor John Mathers qualified in Agricultural Biochemistry & Nutrition at Newcastle University in 1971 and obtained a Diploma in Nutrition with distinction from the University of Cambridge in 1973. In 1979 he gained a PhD in Nutrition at the University of Cambridge and undertook post-doctoral research in the Universities of Cambridge and Edinburgh.

John Mathers is Professor of Human Nutrition in Newcastle University and Director of the cross-Faculty Human Nutrition Research Centre [HNRC] which was established in 1994. The HNRC was designated as a University Research Centre in November 2008. Currently approximately 75 staff and students work in the HNRC. He is the Principle Investigator for a portfolio of research projects in the areas of nutrition and the prevention of common non-transmissible diseases including colon cancer and cardiovascular disease. This work includes studies on the biological basis of ageing and ranges from molecular and cell biological studies, through investigations in human volunteers to nutritional epidemiology.

Previously, he was President of the Nutrition Society (2001- 2004) and was honoured to receive the British Nutrition Foundation Prize for outstanding contributions to nutrition in 2001. Other professional responsibilities include:

- Advisory Committee on Novel Foods and Processes (ACNFP) (2008 – present)
- MRC Physiological Systems and Clinical Sciences Board (PSCSB) (2004 – 2008)
- BBSRC Healthy Organism Strategy Panel (HOP) (2004 – 2008)
- MRC Lifelong Health & Wellbeing Research Advisory Panel (2008 –present)
- BBSRC DRINC initiative Steering Group (2007 – present)



Professor Annie Anderson
University of Dundee, UK

Professor Annie S. Anderson is Director of the Centre for Public Health Nutrition Research and Co-director of the Centre for Research into Cancer Prevention and Screening at the University of Dundee. Her main research areas focus on theory based, behaviorally focused dietary and obesity interventions aimed at chronic disease risk reduction.

Advisory work includes activities at an international level through the UICC (International Union against Cancer) Taskforce on Cancer Prevention. She was an observer for UICC on the WCRF/AICR Review on Food, Nutrition and Physical Activity and the Prevention of Cancer (2004-2007). At UK level she has been an expert science member of the UK Scientific Advisory Committee on Nutrition (SACN) since 2000, external advisor to the UK Food Standards Agency (Nutrient Profile committee, Low Income Diet and Nutrition Survey, Nutrition Research Programmes) and deputy chairperson of the (MRC) National Prevention Research Initiative scientific committee. She is a director of the Scottish Cancer Foundation and a founder member of the Scottish Cancer Prevention Network.

During 2008/8 she has been responsible for leading work on health and sustainability for the development of the Scottish government Food and Drink policy <http://www.scotland.gov.uk/Publications/2009/06/25143614/0>



Professor Mike Kelly
Director, Centre for Public Health Excellence, National Institute for Health and Clinical Excellence (NICE), UK

Professor Mike Kelly is Director of the Centre of Public Health Excellence at NICE. He originally graduated in Social Science from the University of York, holds a Masters degree in Sociology from the University of Leicester, and undertook his PhD in the Department of Psychiatry in the University of Dundee. Before joining new NICE he was Director of Evidence and Guidance at the Health Development Agency. Professor Kelly has held posts at the Universities of Leicester, Dundee, Glasgow, Greenwich and Abertay. He now has an honorary chair in the Department of Public Health and Policy at the London School of Hygiene and Tropical Medicine, University of London and is a Fellow of the Faculty of Public Health.

Professor Kelly is a medical sociologist with research interests in evidence based approaches to health improvement, methodological problems in public health research, evidence synthesis, coronary heart disease prevention, chronic illness, disability, physical activity, health inequalities, social identity and community involvement in health promotion.



Professor Sir Michael Marmot
University College London, UK

Graduating in Medicine from the University of Sydney, Australia, in 1968, Professor Sir Marmot gained an MPH in 1972 and PhD in 1975 from the University of California, Berkeley. He became a Fellow of the Faculty of Public Health Medicine in 1989, was appointed professor of epidemiology and public health at University College London (UCL) in 1985 and took a joint chair, held at UCL and the London School of Hygiene and Tropical Medicine, in 1990. He became director of the International Centre for Health and Society established at UCL in 1994, was appointed a member of the Royal Commission on Environmental Pollution and awarded an MRC Professorship in 1995. He was elected as a Fellow of the Royal College of Physicians in 1996.

Professor Sir Marmot has been at the forefront of research into health inequalities for the past 20 years, as principal investigator of the Whitehall studies of British civil servants, investigating explanations for the striking inverse social gradient of morbidity and mortality. He chairs the Department of Health Scientific Reference Group on tackling health inequalities and chaired the National Institute for Clinical Excellence (NICE) Research and Development. Internationally acclaimed, Marmot is a vice president of the Academia Europea, a member of the RAND Health Advisory Board, a foreign associate member of the Institute of Medicine, and chairs the WHO Commission on Social Determinants of Health. He was awarded the Balzan Prize 2004 for Epidemiology and gave the RCP Harveian Oration in October 2006. He was knighted by her Majesty The Queen in 2000 for services to epidemiology and understanding health inequalities.

Areas of research conducted by Professor Sir Marmot, other than the Whitehall studies, include: investigating the gap in morbidity and mortality between East and Western European countries; factors that underlie inequalities in health in the elderly; the English Longitudinal Study of Ageing; investigating characteristics of neighbourhood social environment that affect health over and above characteristics of the individuals; and the Japanese and Finish collaborative work on the socio-economic differences in health and disease.



Professor Kay-Tee Khaw
University of Cambridge, UK

Kay-Tee Khaw is Professor of Clinical Gerontology at the University of Cambridge School of Clinical Medicine.

She trained in medicine at Girton College, Cambridge and St. Mary's Hospital, London and in epidemiology at the London School of Tropical Medicine and Hygiene, with subsequent clinical and academic posts in the University of London and University of California San Diego.

Her research interests are the maintenance of health in later life and the causes and prevention of chronic diseases including cardiovascular disease, cancer and osteoporosis with over 400 research publications. She is a principal investigator of the European Investigation into Cancer and Nutrition in Norfolk cohort, <http://www.epic-norfolk.org.uk>, part of a ten country half million participant European collaboration investigating dietary, hormonal and other determinants of cancer and other chronic diseases.



Professor Josette Chor
Chinese University of Hong Kong, Hong Kong

Professor Chor received her medical degree from Faculty of Medicine, The University of Hong Kong. She then obtained Master of Public Health and Doctor of Philosophy in medical sciences at the Chinese University of Hong Kong. She is currently the associate fellow of the Hong Kong College of Community Medicine and the Diplomate member of the Faculty of Public health, UK. She has been granted Professor John Vallance-owen award and Sir Edward Memorial Fellowship during PhD study and Global Scholarship Programme for research Excellence – CNOOC Grant to have further training and research in the University of Oxford, UK. Besides working as a research academics, Prof. Chor is also the academic co-ordinator of the undergraduate (non-medical) programme in Public Health. Her research interests include the role of nutrition in cancer etiology and cancer survivor; cervical and breast cancer screening; HPV epidemiology and vaccination.



Dr Michelle Harvie
University of Manchester, UK

Dr Michelle Harvie is a research dietitian from the Genesis Breast Cancer Prevention Centre University Hospital South Manchester Trust. She qualified as a dietitian in 1991 and was awarded a PhD for studies of weight gain in cancer patients in 2000. Her current programme of work is researching optimum diet and exercise strategies to prevent breast cancer and its recurrence. Her research has been published in many major scientific publications and she was awarded the British Dietetic Association Rose Simmond's Award for best published dietetic research 2005, for her work on weight gain amongst breast cancer patients receiving adjuvant chemotherapy.

Dr Harvie is the principal investigator of the B-AHEAD study Breast - Activity & Healthy Eating After Diagnosis A randomised comparison of 3 weight control programmes during adjuvant treatment for early breast cancer amongst 500 early breast cancer patients.



Professor Pär Stattin
Umeå University, Sweden

Dr Pär Stattin graduated as a Medical doctor at Uppsala University, Sweden in 1984. He did his clinical training and specialised in Urology at Västerås Central Hospital, Sweden 1984-1992. In 1997 he obtained a PhD in Molecular pathology at Umeå University, Sweden and since then he has worked in the Department of Urology and andrology at Umeå University where he became professor in 2009. His current research in cancer epidemiology investigates the association between life-style related hormonal environment and cancer risk. He is chairman of the National Prostate Cancer Register (NPCR) of Sweden and his clinical research is focused on outcomes studies.



Dr Susan Jebb

Medical Research Council Human Nutrition Research Unit, Cambridge, UK

Dr Susan Jebb is Head of Nutrition and Health at the Medical Research Council Human Nutrition Research unit (HNR) in Cambridge, UK. Susan trained in nutrition and dietetics at the University of Surrey prior to her PhD at the University of Cambridge where she studied *in vivo* methods to measure body composition. She moved to HNR in 1998, where her research focuses on the role of dietary factors in the aetiology and treatment of obesity and related metabolic diseases. It includes observational studies on dietary patterns and weight gain, explanatory dietary intervention studies and more pragmatic trials of community based weight management strategies.

Susan also leads the HNR Communication team who focus on the translation of nutrition science into policy and practice, working with policymakers, industry, health professionals and NGOs. She is an advisor to government on a range of issues relating to food and obesity. Currently she is Chair of the cross-government Expert Advisory Group on Obesity in England, Chair of a NICE Programme Development Group developing guidance for the prevention of obesity at a local level and Chair of the UK Association for the Study of Obesity.

As part of HNR's public engagement programme, Susan regularly contributes to media features on food and obesity including several BBC documentaries, she writes a nutrition column for a popular magazine and recently presented a series of Sizzling Science events, working with a chef to look at science in the kitchen by cooking healthy family meals.



Fabio S Gomes

Brazilian National Cancer Institute, Brazil

Fabio Gomes graduated in nutrition at the Rio de Janeiro State University in Brazil. He obtained a MSc. in Population Studies and Social Research in 2007 and is a PhD candidate in Collective Health at the Institute of Social Medicine of the Rio de Janeiro State University.

He has worked as advisor of the United Nations Development Programme for the Brazilian Ministry of Health, supporting the development of strategies to implement nationwide actions for Surveillance of Risk Factors for Non-Communicable Diseases (NCDs) in Schools. Currently he works in the Food, Nutrition and Cancer Division of the National Cancer Institute of Brazil (INCA) as a senior analyst for national cancer control programmes, supporting the development of health promotion strategies in multiple settings, and developing and improving local and nationwide strategies to prevent and control cancer and other NCDs by means of the promotion of healthy eating practices.



Professor Sir Mike Richards
Department of Health, UK

Professor Richards was appointed as the first National Cancer Director in October 1999. In 2000 he led the development of the NHS Cancer Plan, the first comprehensive strategy to tackle cancer in England and was then responsible for overseeing its implementation. More recently he has led the development of the Cancer Reform Strategy (published in December 2007) and the development of the first ever End of Life Care Strategy (published in July 2008). He works closely with ministers, parliamentarians, civil servants, clinicians, managers, patient groups, charities, researchers and industry to achieve the objectives of the plan.

Prior to his appointment to the Department of Health, Mike was a Consultant Medical Oncologist at Guy's Hospital specialising in breast cancer (1986-1995) and Sainsbury Professor of Palliative Medicine at St Thomas' Hospital (1995-1999). He was also Clinical Director of Cancer Services at Guy's and St Thomas' from 1993 to 1999.

Mike was closely involved in the establishment of the National Cancer Research Institute in 2001 and has been a board member since its foundation. Between April 2006 and March 2008 he was Chairman of the NCRI Board in addition to his role as National Cancer Director.

In June 2008 Mike was asked by Alan Johnson, then Secretary of State for Health, to lead a review of policy relating to patients who choose to pay privately for drugs that are not funded on the NHS. His recommendations were accepted and his report was published in November 2008.

Mike was appointed CBE in 2001 and was awarded a Knighthood in the 2010 New Year's Honours.

If he ever has any spare time Mike's hobby is hill walking, particularly in Scotland.



Professor Alan Jackson,
University of Southampton, UK

Professor Jackson is Director of the Institute of Human Nutrition, University of Southampton. He trained in paediatrics at the University of Cambridge and University College Hospital London. He was a Wellcome Research Fellow and subsequently Director of the Tropical Metabolism Research Unit, University of the West Indies, Jamaica, where he helped develop the evidence base for the WHO manual on effective treatment of severe malnutrition. He is an Honorary Member of the Nutrition Society. He is a Fellow of the Royal College of Physicians, a Fellow of the Royal College of Paediatrics and Child Health and a Fellow of the Royal College of Pathologists in London. His current research seeks to determine how poor nutrition of a woman before and during pregnancy limits the ability of a mother to support the development of her baby, and the impact this has on the personal development of the child and in damaging broader society.

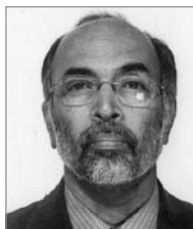
He is the foundation Chairman of the Scientific Advisory Committee on Nutrition to the Department of Health and the Food Standards Agency, UK, a Member of the Scientific Panel on Dietetic Products, Nutrition and Allergies of the European Food Standards Authority, and Member of Council of the Caribbean Health Research Council. He is a member of the main Panel and the Advisory Panel for the second Expert Report on Diet and Cancer of the World Cancer Research Fund.



Dr Isabelle Romieu
International Agency for Cancer Research (IARC), France

Dr. Isabelle Romieu obtained her Medical degree (MD) from the Medical School of Montpellier in France and completed her residency at the Cancer Institute in France. She obtained postgraduate training in biostatistics and nutrition and later obtained a master of Public Health (MPH) and a doctorate of Science (ScD) in Epidemiology with focus on nutritional epidemiology from Harvard University. From 1991-2008 she worked in Mexico and Latin American countries first with the Pan American Health organization, then with the National Institute of Public Health in Mexico where she was professor of epidemiology. She has conducted several studies on the risk of breast cancer and was recently awarded a research prize for her work in the breast cancer field by the AVON foundation.

She is the PI of a large cohort study on the risk of cancer and other chronic diseases in Mexico. She recently joined the IARC as Head of the section on nutrition and metabolism. She is the authors of more than 150 scientific articles and book chapters and widely recognized as an expert in nutritional epidemiology. She has a major interest in early onset breast cancer, life style risk factors and gene environment interaction.



Professor Prakash Shetty
University of Southampton, UK

Professor Shetty is currently Professor of Public Health Nutrition at the Institute of Human Nutrition, University of Southampton, UK and Editor in Chief of the European Journal of Clinical Nutrition. Until 2005 he served as Chief, Nutrition Planning, Assessment & Evaluation Service, in the Food & Nutrition Division of the Food & Agriculture Organisation (FAO) of the United Nations (UN) in Rome, Italy. Before joining the FAO he was Professor of Human Nutrition at the London School of Hygiene & Tropical Medicine (London University). Since his appointment in 1993 to the Chair of Human Nutrition at London University he has been Head of the Public Health Nutrition Unit, Department of Epidemiology & Population Health at the London School. He graduated from Madras University with an MBBS degree (1968) and an MD (1972), both from the Christian Medical College, Vellore, India. He was awarded his PhD in Medicine from Cambridge University, UK while working at the Medical Research Council's Dunn Nutrition Laboratory in Cambridge in 1980. He was elected Fellow of the Faculty of Public Health Medicine (FFPHM) of the Royal College and Fellow of the Royal College of Physicians of London (FRCP). Prior to moving to the UK, he was Professor and Chairman of the Department of Physiology at St Johns Medical College in Bangalore from 1980 to 1993 and established one of Indian Council of Medical Research's (ICMR) Regional Nutrition Research Centre of which he was the Director with research interests in energy and protein metabolism.

He has written over 150 peer-reviewed publications, chapters in textbooks and has edited several books and monographs in nutrition. He has served on several Expert Committees, both nationally and on International Committees and Consultations of the FAO, WHO, UNU and IAEA as well as on scientific taskforces and advisory committees of funding agencies and charities. The Government of UK appointed him as member of the Scientific Advisory Committee on Nutrition (SACN) in 2000.



Cary Adams
International Union Against Cancer (UICC), Switzerland

Born in London, Cary Adams is a 46-year-old, with a BSc Honours degree in Economics, Computing and Statistics from the University of Bath, United Kingdom. He was recruited by Lloyds Bank as a graduate trainee and following 13 years, during which he became a specialist in money transfer systems, Cary began studying part-time for a bank-sponsored MBA at Bath in 1998, receiving a Distinction when he graduated in 2002. He then followed a three-month General Management Program at the Harvard Business School and a Financial Management course at Stanford Business School in 2003.

In 2001 Cary took on his first general management responsibilities in the Business Banking division of the Lloyds TSB Group, which covered 500,000 small businesses in the UK; he became Managing Director in 2004 on the strength of his vision for turning it around. This success led to his appointment, in October that same year, as Managing Director of International Private Banking for Lloyds TSB Group, headquartered in Geneva with offices in 12 other countries and 1,000 staff. In three years, Cary led the business into strong growth and profitability with high morale. This led to his last assignment as Chief Operating Officer of Lloyds TSB Group International Banking in London in 2008. In 2009 he left Lloyds and was appointed Chief Executive Officer of the International Union Against Cancer (UICC) in September 2009.



Dr Rachel Thompson
World Cancer Research Fund International, UK

Dr Rachel Thompson received a BSc Hons in Biochemistry from Leeds University. She worked as a dietitian before moving into nutrition research. She has a PhD in nutrition from the University of Southampton where she worked in research and teaching for 17 years. Whilst at the University of Southampton she was the Review Coordinator for the WCRF Second Expert Report published in 2007.

She is a registered Public Health Nutritionist and Registered Dietitian.

Dr Rachel Thompson is the Deputy Head of Science (Expert Reports and Communications) at the World Cancer Research Fund International. In her current post she manages the Continuous Update Project that builds on the body of data collated during the Second Expert Report. She also works with the Education and Communications Departments in producing information for the public and health professionals.



Richard Evans
World Cancer Research Fund UK, UK

Richard Evans has been Head of Communications at World Cancer Research Fund (WCRF) for three years, regularly securing national media coverage for WCRF's messages about how people can reduce their cancer risk.

In 2007 he managed the UK launch of the WCRF/AICR Second Expert Report, which was one of the biggest health stories of recent years and won the Chartered Institute of Public Relations' annual award for health campaign of the year. He also managed the launch of Policy and Action for Cancer Prevention in February 2009.

Richard has also written articles about media reporting of cancer, including for the BBC and Guardian websites.

Before working at WCRF, Richard worked in local government communications for Lambeth Council and Greenwich Council. He previously spent five years as a journalist, including as health reporter for the Hull Daily Mail.



Dr Ben Goldacre
London School of Hygiene and Tropical Medicine, UK

For a detailed biography please visit <http://www.badscience.net/about-dr-ben-goldacre/>



Dr Alan Dangour
London School of Hygiene and Tropical Medicine, UK

Alan is a senior lecturer and Registered Public Health Nutritionist with a background in biochemistry and biological anthropology. Alan joined LSHTM in 2001 having previously taught at University College London and the University of Cambridge. Alan's primary research focus is nutrition in older age, and he is conducting a series of randomised controlled trials to determine the effectiveness of nutrition interventions for the maintenance of health and function in later life. Alan is also part of an inter-disciplinary team that recently attracted significant funds to set up the Leverhulme Centre for Integrative Research on Agriculture and Health. The Centre conducts inter-sectoral research on: poverty and development; globalisation and food quality; sustainability, environment and climate change; food-borne and zoonotic diseases; and common metrics for agricultural research and evaluation. Alan is a member of the international taskforce on health co-benefits of climate change mitigation policies and has considerable experience in the conduct of systematic reviews.



Dr Christopher Paul Wild
International Agency for Research on Cancer (IARC), France

Christopher Paul Wild obtained his PhD in 1984 from the University of Manchester, UK whilst working on the production of monoclonal antibodies to detect low levels of methylated DNA bases. He was awarded a post-doctoral fellowship from the International Agency for Research on Cancer (IARC) to work in Lyon, France and subsequently a UK Royal Society European Exchange Fellowship to spend a year at the Netherlands Cancer Institute in Amsterdam. In 1987 he rejoined IARC as a staff scientist and later became Chief of the Unit of Environmental Carcinogenesis.

In 1996 he was appointed to the Chair of Molecular Epidemiology at the University of Leeds, was Head of the Centre for Epidemiology and Biostatistics and later became Director of the Leeds Institute of Genetics, Health and Therapeutics in December 2005. He was elected Director of IARC from 1st January 2009.

Dr Wild's main research interest is to understand the interplay between environmental and genetic risk factors in the causation of human cancer. He has particularly sought to apply biomarkers in population-based studies. He has published widely on liver and oesophageal cancer as well as the role of mycotoxins in human disease.

Recently appointed as Director of IARC.



Professor Elio Riboli
Imperial College London, UK

Professor Riboli is Director of the School of Public Health at Imperial College London, rated one of the top two epidemiology and public health submissions to the UK's Research Assessment Exercise (RAE) in 2009. He holds an M.D. degree (1977, State University of Milan), a Master of Public Health (1980, Milan) and a Master of Science in Epidemiology (1982, Harvard University). Professor Riboli is a Registered Physician (General Medical Council, UK, 2005) and an Honorary Fellow of the Royal College of Physicians (2008).

From 1983 to 2005 Professor Riboli was based at Lyon's the International Agency for Research on Cancer (IARC), where he developed new research projects in the areas of nutrition, nutritional status and cancer. In 1989 he initiated the European Prospective Investigation into Cancer and Nutrition (EPIC), which sampled data from 500,000 subjects across 26 centres in ten countries. He was Head of the Nutrition and Hormones Group of IARC from 2004 to 2005.

Professor Riboli has co-authored over 310 peer-reviewed publications and over 100 book chapters and books and serves on editorial boards of major journals on nutrition, cancer and epidemiology.

In 2005 he joined Imperial College London as Professor of Cancer Epidemiology; in 2006 he was appointed Divisional Head of Epidemiology, Public Health and Primary Care, and became Director when the School of Public Health was established in January 2010.

Note:

For all abstracts, conflicts of interest and sources of funding were declared but for reasons of space they were omitted from this abstracts book. This information will be made available on the WCRF International website in due course.

Speaker Abstracts

Nutrition & cancer around the world – opportunities and challenges

Dr Francesco Branca

World Health Organization, Switzerland

This presentation will discuss food, nutrition and the prevention of cancer. It will include a global perspective focussing on the current challenges and future opportunities. Both research and policy aspects of the topic will be discussed.

Obesity, physical activity and cancer – global perspectives

Professor W Philip T James

International Obesity Task Force, UK

The first WCRF/AICR report established new approaches to assessing the value of experimental, clinical and epidemiological evidence which became a forerunner of the new approach to WHO's latest analyses of diet, physical activity and the prevention of chronic diseases. The Second WCRF/AICR report was far more systematic with a separate protocol group setting standard criteria for a series of independent, exhaustive statistical assessments of cohort and case control studies. This was followed by an updated consensus on interpreting convincing and probable causal relationships which coped with the expected difficulty of not having double blind controlled trials of dietary intervention for cancer prevention. The role of physical activity was also assessed. The second report benefited from a remarkable increase in epidemiological studies of cancer but as expected was handicapped not only by the expected errors in diet and physical activity measurements but also by our current inability without intermediate biological markers of mechanistic processes to take account of any substantial inter-individual variation in responsiveness to diet. Thus there is little evidence in cohort studies of a saturated fat -coronary heart disease relationship despite the overwhelming evidence of the casual mechanism relating saturated fats to cholesterol metabolism and atherosclerosis. This means that diet and physical inactivity may be even more important than currently assessed in cancer but the obesity relationship now comes through as the most powerful predictor after tobacco of a series of cancers.

Obesity constitutes not only a biological marker of inappropriate diets and physical inactivity but in itself seems to constitute a major biological mechanism promoting cancer through mechanisms which involve marked endocrinological and immunological changes, substantial inflammatory processes, altered prostanoid metabolism with insulin resistance and substantial metabolic effects which result in and relate to non - alcoholic hepatic steatosis. Physical inactivity has also been underestimated as a profound modifier of metabolism and insulin resistance. The importance of these changes is seen in the major reductions in cancer incidence within 5-7 years of marked weight loss after bariatric surgery. Some of these changes may reflect non -physiological changes in hormonal levels e.g. GLP-1; the dietary consequences of surgery are also profound. Nevertheless the evidence now seems conclusive that preventing or reversing obesity prevents several cancers and may indeed affect cancer survival rates.

Globally the profound obesity epidemic sweeping lower income countries is, with the associated dietary changes and collapse of physical activity, already inducing rising cancer rates. Despite the renewed emphasis on screening and early treatment global cancer rates cannot realistically be combated except by a fundamentally new approach to the role of diet and physical inactivity determining profound changes in population health. Individualistic approaches are welcome and understandable but an economically unrealistic simple option for limiting cancer rates. The UK Foresight obesity analysis, new WCRF policy report and latest US CDC and UK NICE guidelines highlight the political, economic and structural changes needed with new policy initiatives if we are to make major headway with prevention.

How early should we be concerned with cancer prevention?

Professor Ricardo Uauy

London School of Hygiene and Tropical Medicine, UK and University of Chile, Santiago, Chile

The incidence of obesity and hormone-sensitive cancers is increasing worldwide, particularly among women in both industrialised and transitional countries. Age of pubertal maturation is associated with increased risk of breast and testicular cancer, and potentially with prostate cancer. Cross-sectional studies have associated obesity with earlier onset of puberty and age of menarche; however, serial weight and height measurements are rarely available. Thus, it is impossible to assess whether growth and adiposity gain trajectories in early life determine timing and progression of biological/sexual maturation. Infancy weight gain and timing of adiposity rebound might contribute to increased cancer risk based on the effects they have on biological maturation and risk of later obesity. Results of our cohort of Chilean children 0 to 6y of age with serial weight and height measurements and assessments of maturation reveal that critical periods of early growth that are potentially associated with later adiposity, bone age maturation and with timing of puberty and thus potentially risk of breast cancer. We are striving to identify critical periods and potential markers, which may be suitable to assess preventive actions in early life. Additional information from follow up studies of adult weight reduction using various modalities of restricting gastric capacity demonstrate in controlled studies that cancer incidence can be significantly reduced with substantive weight and adipose tissue loss. The potential of effective cancer prevention by reducing risk of obesity in early life offers a unique opportunity for primary prevention of cancer risk.

WCRF International research programme

Dr Panagiota Mitrou

World Cancer Research Fund International, UK

This session will discuss the research directions identified in the 2007 WCRF/AICR Expert Report, and how these are reflected in the WCRF global network Research Grant Programmes, comprising the WCRF International Grant programme and the AICR Grant Programme. These research directions, which aim to increase understanding of the cancer process, including cancer initiation, promotion, progression and metastasis, provide the basis for the research priorities for the WCRF global network programmes. Both Research Programmes are dedicated to funding research on the role of food, nutrition, physical activity and body weight in relation to cancer.

The WCRF International Regular Grant Programme has introduced some changes this year. Applications for the WCRF International research programme will now be accepted from anywhere in the world except the Americas (North America including Canada, Central America including the Caribbean and South America). In addition, in order to reflect the key priority that all research funded by the network should be directly relevant to human cancer, the scope of the WCRF International Grant Programme has been broadened to include experimental designs outside the human and in vivo settings. However, these must be for relevant studies that explore mechanistic pathways of the cancer process. A number of general research principles have also been introduced to reflect this key priority.

The WCRF International Grant Programme focuses on diet, activity and cancer through the lifecourse. Specific priorities include early life exposures, body fatness, physical activity, or patterns of diet and physical activity, in relation not only to cancer development but also to outcome in diagnosed cancer, as well as understanding the impact of genetic and other constitutional factors on the cancer process, and underlying mechanisms. These priorities are often interdisciplinary and address issues that could help translate research into action to prevent cancer. Research into policy and action is a possible topic for future consideration.

WCRF/AICR Continuous Update Project

Dr Teresa Norat

Imperial College London, UK

Following the publication of the Second Expert Report, WCRF/AICR established the Continuous Update project (CUP) in collaboration with Imperial College London (ICL). The CUP will systematically review the science as enough new evidence is accumulated. WCRF/AICR has convened a panel of experts who will consider the evidence produced at ICL, and draw conclusions before making recommendations for cancer prevention.

The CUP initiated its activities in 2007. The data collated during the second Expert Report is being updated in a central database at ICL. A rolling programme has been set up whereby cancers are gradually added to the database. Currently, the database is being updated for cancers of the breast, prostate, colon, rectum, and pancreas. From September 2010 the program will add the evidence on breast cancer survival.

The project benefits with collaboration from universities of the Netherlands (Wageningen), France (Paris 13) and UK (Leeds, Bristol). Two researchers from France and one from the Netherlands have been trained on meta-analytical methods at ICL. In the future, the CUP aims to increase external collaborations.

Periodically, the team of five working in the CUP produces reports with systematic literature reviews. In December 2008 the CU Panel reviewed the updated evidence for breast cancer. The Panel agreed that the current WCRF/AICR recommendations were still up to date for breast cancer. In November 2010, the Continuous Update Panel will meet to review the evidence for colorectal cancer.

The WCRF database is one of, if not the largest resource of existing scientific literature on food, nutrition, physical activity and cancer. Once the central database has been fully updated with more cancer sites, it will be made available to the wider scientific community, enabling researchers working in the field of cancer prevention to have access to the complete body of work relevant on their field.

Research directions in Asia

Professor Tai Hing Lam

School of Public Health, the University of Hong Kong

Asia has very different ethnicity, traditions and cultures. Socio-economic developments and lifestyle changes especially in food, nutrition and physical activity (FNPA) and the stage of epidemiological transition vary widely. Cancer research in Asia on aetiology, gene-environment interaction and epigenetic effects, and lifestyle and life course factors/exposures will contribute unique and new knowledge.

In Asia, apparently simple replication studies on well-known risk factors in the West can have strong local political impacts. These studies are also needed for local research capacity building. If locally relevant or suspected factors, such as some local foods, are examined together, these studies may yield original results.

Case control studies are useful, especially on suspected but unconfirmed risk factors, and on cancers, which are uncommon in the West. Adding genetic and other molecular factors would generate new knowledge, such as using Mendelian randomisation. Pilot studies are needed to select more relevant SNPs and estimate sample size. Pathological specimens can be collected readily. However, whether new knowledge on genetic factors can add to prevention should be considered carefully.

Large cohort studies are scarce in Asia. The collection and storage of bio-specimens, such as DNA, plasma/serum at baseline together with detailed phenotype data will provide the best research platform. A few biobank studies in Asia established with low budget show good potentials and further funding is warranted.

Multi-centre collaborative meta-analysis of individual data is cost effective. The Asia Pacific Cohort Study Collaboration is a successful example. It was started for cardiovascular disease but it has some papers on cancer. New funding is warranted to support expansion and updating of exposure and follow up data.

Good prevention research is rare in Asia. Whether research on FNPA can be considered as fundable cancer research needs to be clarified. Studies on FNPA and their effects on survival and quality of life in cancer survivors are needed as conflicting advices and beliefs are common.

Vitamin D and cancer

Dr Mazda Jenab

International Agency for Research on Cancer (IARC), France

Vitamin D is an important component of the diet but in most populations it is mainly produced endogenously from sun exposure. The main role of Vitamin D is the maintenance of calcium homeostasis and bone metabolism. However, a role for vitamin D has also been proposed in cancer prevention and control, primarily because of its other main functions in cell cycle regulation (promotion of apoptosis and differentiation and inhibition of proliferation), growth factor signalling, and modulation of immune and inflammatory responses. Although a growing body of literature is supportive of an anti-cancer role for vitamin D, the evidence is still lacking for many cancer sites. In addition, information from populations other than North American or European is scarce and more research is necessary. Most of the epidemiologic data available to date are based on dietary vitamin D intake and show mixed findings. Many of these studies do not account for endogenous vitamin D production from sun exposure, and they are limited by the few food sources of vitamin D as well as measurement errors in its dietary assessment. For these reasons, epidemiologic studies measuring circulating blood vitamin D concentration, as an integrative estimate of dietary intakes and endogenous production, are preferred. However, with the exception of colorectal cancer, the epidemiologic evidence from such studies is inconclusive – although many studies are in the pipeline and should be completed in the coming years. Evidence from randomised trials of vitamin D supplementation and cancer risk, regarded as the gold standard of showing a cause and effect relationship, have been largely inconclusive and further evidence to clearly show whether increases in circulating vitamin D can effectively reduce risk of cancers without inducing serious adverse events is necessary. The cumulative evidence to date from in vitro, experimental and epidemiologic studies suggests that vitamin D may have the potential to modulate cancer risk, as well as that of other chronic diseases, and thus further research on this compound is merited.

Epigenetics, nutrition and bowel cancer risk: determinants of bowel cancer risk

Professor John C. Mathers

Human Nutrition Research Centre, Institute for Ageing and Health, Newcastle University

Cancer of the colon and rectum (CRC) is the third most common cancer worldwide. Although genotype contributes to risk, food intake and physical activity are the major determinants of CRC risk either directly or via effects on adiposity. Since unrepaired genomic damage leading to aberrant gene expression is the fundamental cause of all cancer (including CRC), the challenge is to identify the mechanisms through which lifestyle factors contribute to abnormal gene expression. Given their role in the regulation of gene expression, the evidence that aberrant epigenetic marks may precede the development of neoplastic lesions and the hypothesis that epigenetic marks may link environmental exposures with altered gene expression (Mathers 2008), epigenetic mechanisms are increasingly the focus for the development of novel biomarkers of CRC risk.

To identify epigenetically-regulated genes which may be biomarkers of CRC risk, we have undertaken quantitative analysis of promoter methylation in a panel of genes in macroscopically normal mucosa from subjects with colorectal adenomatous polyps and carcinomas in comparison with neoplasia-free subjects (Belshaw et al. 2008). Methylation of APC, HPP1, p16, SFRP4, WIF1 and ESR1 was informative in distinguishing those with carcinoma from non-cancer patients. To avoid the need for colonoscopies, it would be valuable to be able to quantify methylation using a surrogate “tissue” such as blood or stool. Although human DNA can be recovered from stool and promoter methylation quantified (Belshaw et al. 2004), this approach remains poorly developed. To date there is limited information on the functional consequences of altered epigenetic marks in the colonic mucosa. Using a proteomics approach, we have identified a number of proteins, which are differentially expressed in the macroscopically normal mucosa of individuals with colorectal adenomas or carcinomas compared with no neoplasia (Polley et al. 2006). These are potential biomarkers of CRC risk and it will be important to discover the extent to which these proteomic markers are influenced by diet, other lifestyle factors and adiposity and whether their expression is regulated by epigenetic mechanisms.

This work has been carried out in collaboration with Prof. Ian Johnson and colleagues (Institute of Food Research, Norwich).

Belshaw, NJ et al. (2008) Br J Cancer 99, 136-142. Profiling CpG island field-methylation in both morphologically normal and neoplastic human colonic mucosa.

Belshaw, NJ et al. (2004) Cancer Epidemiol. Biomark. & Prev. 13, 1495-1501. Use of DNA from stools to detect aberrant CpG island methylation of genes implicated in colorectal cancer.

Mathers, JC (2008) Proc. Nutr. Soc. 67, 390-394. Epigenomics: a basis for understanding individual differences.

Polley, ACJ et al. (2006) Cancer Research 66, 6553-6562. Proteomic Analysis Reveals Field-Wide Changes in Protein Expression in the Morphologically Normal Mucosa of Patients with Colorectal Neoplasia.

What evidence do you need for public health policy?

Professor Mike Kelly

National Institute for Health and Clinical Excellence (NICE), UK

This paper will examine the relationship between evidence and policy making using particular examples relating to food, nutrition, physical activity and cancer prevention. The paper will begin by considering the hopes of the evidence based policy initiatives of the late 1990s. The chain from evidence, through evidence synthesis or evidence appraisal, to the policy making process and the implementation of policy will be described in schematic form. Problems of taking a linear approach will be highlighted. A number of models of the way that evidence is used in the policy and implementation processes will be presented. These will include: the Mintzberg dilemma; the Jowett dilemma; and the institutional resistance dilemma. Some consideration of the question of knowledge transfer will be examined in the light of the policy of obesity prevention. Policy making as a cultural, social and political process will be considered in contradistinction to it being a scientific process. Some of the common errors that scientists make about the way policy makers operate and their frequent failure to acknowledge the difference between empiricism and rationalism in their own practices will be outlined. The difference between scientific and political judgements will be explored. Several examples from recent policy areas will be considered – the restriction of the advertising of foods high in salt, fat and sugar to children; the use of pedometers to encourage physical activity, and the science of behaviour change and its lack of impact in cancer prevention. Regulation of salt and trans fatty acids across the European Union will also be considered if time permits. The paper will conclude with an assessment of what this means for evidence-based policy.

Folic acid fortification, good or bad?

Professor Ellen Kampman

Wageningen University, The Netherlands

In most European countries, a range of foods is voluntarily fortified with folic acid at variable levels. Mandatory fortification has not been introduced in any European country yet. Although folic acid is good in reducing risk of neural tube defects, discussion is continuing about whether it may be bad for those at risk for certain types of cancer, especially colorectal cancer.

Folic acid is synthetic and does not occur in nature, but can be utilised as a precursor to form biologically active natural folates. However, our capacity to metabolise folic acid is not optimal. Therefore, excess intake may lead to accumulation of folic acid, which has a greater capacity than natural folates to enter cells and stimulate cell growth, potentially setting the stage for carcinogenesis. Randomised control trials designed to test the effect of folic acid on recurrence of colorectal adenomas are however inconsistent.

Last year, a panel of experts convened by EFSA concluded that there was insufficient data to allow a full quantitative risk assessment of folic acid and cancer. They recommended further animal and human studies and continued long term follow up of cancer risk in participants in folic acid supplementation trials. Funded by WCRF, we are currently evaluating the role of folic acid supplementation on DNA methylation in a 3-year intervention trial among healthy elderly with relatively high homocysteine levels.

Since it may lead to some people ingesting significant or even excess amounts of folic acid from their diet on top of the intake through vitamin supplements, folic acid fortification should be treated with caution. First, we need scientific evidence showing that fortification is not only good for pregnant women, but also not bad for the population at large.

Global inequalities – implications for research, practice and policy

Professor Sir Michael Marmot

University College London, UK

Non-communicable diseases, including cancer, are the major causes of death globally, accounting for at least 60% of all deaths annually. Deaths due to non-communicable diseases are predicted to increase in low and middle-income countries over the next 20 years. Evidence from developed countries shows that a number of non-communicable diseases follow a social gradient: the lower the level in the social hierarchy, the higher the risk.

The frequent response is to emphasise individual responsibility based on healthy eating, increased physical activity and not smoking. The social gradient in non-communicable diseases demonstrates that the individual choices people make are influenced by the social circumstances in which they live.

It follows that to make progress in reducing the burden of non-communicable disease we must look beyond the immediate causes and examine the underlying causes of the systematic distribution of these diseases: the social determinants. This came under the remit of the Commission on Social Determinants of Health set up by the World Health Organization (WHO) in 2005 and chaired by Sir Michael.

The Commission on Social Determinants of Health reported to WHO in August 2008, and made its recommendations, based on extensive evidence, across three overarching areas for action: 1) improve the conditions in which people are born, grow, live, work, and age; 2) tackle the structural drivers of those conditions at global, national and local level; 3) monitoring, training and research. Within these areas of action the WHO Commission made recommendations in twelve areas: 1) early child development and education, 2) healthy places - the living environment, 3) fair employment and decent work, 4) social protection across the lifecourse, 5) universal health care, 6) health equity in all policies, 7) fair financing, 8) market responsibility, 9) gender equity, 10) political empowerment, 11) good global governance, 12) knowledge, monitoring and skills.

Sir Michael will discuss how research, practice and policy on the social determinants underpin progress in reducing health inequalities.

Cancer survivors: what we know, what we need to know - an Asian perspective

Professor Josette Chor

Chinese University of Hong Kong, Hong Kong

The incidence rate of cancer closely related to diet including breast and colorectal cancer has been rising sharply for the last two decades in all Asian countries. Westernisation of diet is thought to be one of the key factors in this phenomenon. However little is known in the role of the diet in the prognosis and quality of life in cancer survivors.

The complexity of Asian diet makes the investigation very difficult. People living in the city area and rural area can have very different dietary pattern within the same country for the social disparity and also the living lifestyle. Cultural belief is one of the associated factors, which affect the dietary pattern in cancer survivors. Change in dietary pattern including food avoidance and increased consumption of certain kinds of food is a common practice in cancer survivors because this is thought to be a healing modality for many diseases in traditional belief. The upset of balance causing the disease can be restored by change in diet. Our study showed that there was a mismatch in the demand for dietary and herbal advice and the information given by their health service provider in cancer survivors. The use of herbs is also commonly found in cancer survivors. Herbs are incorporated into the diet and quite often not regarded as medicine. Some small-scale studies have shown the improvement of the quality of life by consumption of particular herbs. However, the small scale and the absence of vigorous methodology led to inconsistent findings. Studies with specific methodology and validated for Asian diet are necessary for reaching conclusion of the role of nutrition in cancer survivors.

Cancer survivors: what we know, what we need to know - a Western perspective

Dr Michelle Harvie

University of Manchester, UK

There are an estimated 2 million cancer survivors in the UK, with over half of these diagnosed with obesity related cancers such as breast (28%), colorectal (12%) and prostate cancer (11%). Weight problems at diagnosis and weight gain, (specifically gains in adiposity and reductions in lean body mass) are common problems amongst these patients. Observational data consistently link excess weight at the time of diagnosis to a poorer prognosis. A key unresolved question is whether weight control and exercise after diagnosis influences outcome and reduces cancer recurrence and survival. Two recent randomised studies amongst early breast cancer patients in the USA (WINS, 2006 and WHEL, 2007) suggest weight control after diagnosis (rather than specific dietary factors) may reduce risk of recurrence. The WHEL study showed no difference in outcome with a low fat diet and increased fruit and vegetable intake (12 vs. 5 portions/ day) with no weight loss, whereas a low fat diet with an average weight loss of 2.7 kg in the WINS study reduced breast cancer recurrence by 24%.

Weight problems may predispose or exacerbate weight related co morbidities such as cardiovascular disease and diabetes amongst cancer patients, which is particularly relevant amongst the increasing ageing Western Cancer population. Observational data show consistent links between excess weight, sedentary behavior and overall mortality. Future research should continue to collect high quality prospective data of the impact of weight control and exercise on cancer related outcomes and overall mortality amongst treatment trial cohorts. Although challenging, further randomised studies are required to determine the unequivocal benefits of weight control and exercise on outcome after cancer diagnosis.

Metabolic syndrome and cancer risk

Professor Pär Stattin

University of Umeå, Sweden

Overeating and little physical activity often lead to development of the metabolic syndrome (MetS), which is increasingly common in the western world. MetS is an established risk factor for cardiovascular disease and diabetes but less is known about the association between MetS and cancer. There is an ongoing debate if there is a synergy between the MetS factors on risk of disease.

In the Collaborative project on MetS and cancer (Me-Can), we pooled data on MetS factors from cohorts in Norway, Austria, and Sweden (1). We investigated the association between MetS and cancer by use of data on height, weight, blood pressure, blood glucose, triglycerides, and cholesterol from 579,000 participants with 37,000 cases of incident cancer and 13,000 fatal cancers after exclusion of events during the first year of follow-up. There were repeated measurements for 134,000 participants that we used to correct for random error in measurements, known to be high for all MetS factors except BMI.

We used Cox proportional hazard models to calculate risk of cancer for the exposures in quintiles, in categories according to WHO, and for a continuous standardised variable (z-score with mean=0 and standard deviation=1) and we also constructed a composite MetS score based on z scores for BMI, blood pressure, glucose, triglycerides, and cholesterol.

To date we have investigated the association between factors in MetS and cancers of the endometrium, breast, pancreas, urinary bladder, kidney, and prostate. For most cancers there was a significant but relatively modest increase in risk for subjects with high levels of MetS, and the association was mostly stronger for fatal than for incident cancer. We did not detect a synergy between the MetS factors on risk for any of these cancers.

1. T Stocks, et al. Cohort Profile: The Metabolic syndrome and Cancer project (Me-Can). *Int J Epidemiol* 2009.

Local action in Brazil

Fabio S Gomes

National Cancer Institute (INCA) Brazil

The insertion of food, nutrition and physical activity (FN&PA) policy and actions in the Brazilian agenda for cancer prevention is still recent, despite its unquestionable role. In 2007, the National Cancer Institute of Brazil (INCA) created the Food, Nutrition and Cancer Division in order to facilitate the intra- and intersectoral integration of INCA, the Ministry of Health and other Ministries and social actors towards a more favourable context for cancer prevention nationwide. Before that, there were already many policies and actions that contributed and still contribute to cancer prevention, but most policy makers still do not recognise its potential to prevent cancer. The political recognition is indeed a mirror of the overall social recognition, since cancer is still not seen as a preventable disease in Brazil, and much less is FN&PA recognised as key contributor for cancer prevention. Furthermore, policy and actions to address FN&PA issues are still narrowed by a predominantly biomedical perspective, which is expressed as the prioritisation of individual approaches focused on behavioural changes or health assistance. Additionally, nutrition-related problems such as undernutrition, still receive more attention from Brazilian society once the country's history is intimately connected with food privation. For this reason, in Brazil, the main challenges in the field, to cite some, are: 1) to broadcast information on the role of FN&PA to prevent cancer; 2) to build social recognition of cancer as a preventable disease and FN&PA as an important contributor to prevent cancer; 3) identify and articulate multisectoral agendas that favour cancer prevention in a broad sense. Opportunities can be drawn from all these challenges: 1) the media is increasingly interested in this topic, but still needs a push to go further the biological dimension of prevention. In addition to the information broadcast, fostering the discussion about communities' perceptions on the myths and fears related to cancer enables health professionals and people to start talking about it, instead of hiding even from information; 3) there are several other actors and agendas dedicated to achieving goals that meet cancer prevention ones by means of FN&PA policy and actions.

Food, nutrition and physical activity - policy and practice in Scotland

Professor Annie Anderson

University of Dundee, UK

In a country of 5.2 million, > 75% of adults have yet to achieve five a day of fruit and vegetables, > 66% of adults are overweight or obese, >60 % do not yet reach 30 minutes moderate activity most days of the week and >57% men and 37% women report consuming more than 7 units of alcohol weekly. Food and drink play a major part in economy of the country with Scottish whisky being the top export product and Scottish meat marketing supported by government initiatives. Culturally the deep-fried mars bar, pies and sugary drinks are a more dominant part of the everyday foodscape than Scottish soft fruit, seafood and vegetable crops.

In 2008 the Scottish Government initiated work on developing a National Food and Drink Policy for Scotland that would promote economic growth, healthy and sustainable food choices, safeguard Scotland's food reputation and take account of food security. The Policy <http://www.scotland.gov.uk/Publications/2009/06/25133322/0> was launched in June 2009 and provides the framework for policy work on diet. The barriers and opportunities for promoting dietary change within a climate of economic development and growth will be discussed.

Nutrition, physical activity and the Cancer Reform Strategy

Professor Mike Richards

Department of Health, UK

This presentation will discuss food, nutrition, physical activity and the prevention of cancer, within the context of the Cancer Reform Strategy. It will include the current challenges and future opportunities. It will discuss the policy aspects of the topic in terms of i) individual approaches versus upstream approaches; ii) general prevention versus cancer specific prevention – is there a role for cancer-specific primary prevention; iii) the feasibility of different approaches in different parts of the world.

Research challenges in lower-income countries

Dr Isabelle Romieu

International Agency for Research on Cancer (IARC), France

Middle to low income countries are undergoing rapid epidemiological transition with increasing rate of chronic diseases. Diet, physical activity and body composition appears to play an important role in these changes. Dietary intake is shifting toward a diet dominated by higher intakes of caloric sweeteners, animal source foods, and edible oils. Activity patterns are equally shifting rapidly toward reduced energy expenditure. These rapid changes in lifestyle offer a unique opportunity to study risk factors related to cancer. However, the lack of availability of accurate and routinely collected data, the lack of census data, disease registry, the limited literacy of the population, and the lack of well trained researchers are strong limitations to evaluate the health status and to conduct epidemiological studies. Only few developing countries have active cancer registries, and death certificates may suffer from subregistration and inaccuracy in causes of deaths. In addition, while many governments are aware of the urgency to evaluate major risk factors for chronic diseases and implement preventive measures, adequate funding is often not available.

Challenges for policy and practice in lower-income countries

Professor Prakash Shetty

University of Southampton, UK

Low income developing economies confront the double burden of malnutrition and disease. While not having eliminated the overwhelming health burden associated with the existing problem of undernutrition and infectious diseases in their midst, they are now faced with the added burden of emerging nutritional problems and disease patterns that characterise the developed economies. Contributed to by the rapid economic development and globalisation and the increasing urbanisation of communities, they influence the dietary and physical activity patterns of large segments of their populations. The challenges that these countries face are central to how the meagre health budgets are attributed to address old agendas and or tackle the emerging newer problems. The challenges for policy and practice include the need for reliable and representative information and data on the emerging problem and the political will to emphasis the important role for health promotion and for primary prevention as opposed to expensive investment in the treatment of these disorders. The challenges will extend to dissemination and social marketing of preventive and health promotive communication and in investing in and evaluating evidence-based interventions that can be implemented in the local environment. The challenges will also include strengthening the role of regulatory bodies and the health sector to withstand corporate interests that seek markets in developing economies with poor regulatory environments and a treatment oriented private health sector at a time of dwindling markets among the developed and rich consumers and economies – a classic example being tobacco. The foremost challenge in the policy arena is the need to develop a stitched-up food, nutrition and health policy to address the double burden and in practice to ensure the involvement of all sectors to address this issue. International agencies and organisations like the WCRF have an important global role to help facilitate this process to address these challenges faced by the emerging economies.

Reporting of public health and cancer risk – overview

Richard Evans

World Cancer Research Fund UK, UK

In 2009, World Cancer Research Fund published Policy and Action for Cancer Prevention, a Policy Report that made recommendations for changes in society that could help prevent cancer.

One of the groups in society – or “Actors” - it identified was the media. This is because of the large influence the media has over public knowledge, attitudes and beliefs.

The aim of the Media recommendations is to “sustain increased coverage of public health and well-being and prevention of obesity and chronic diseases including cancer” and to provide context to this coverage.

But how well does today’s media meet these recommendations?

While the Report’s definition of the media was broader, this session will look at how the news media in particular communicates scientific messages about how people can reduce their risk of chronic disease in general and cancer in particular.

Does news reporting in this area lead to improvements in public understanding of how people can reduce their risk?
Or does the scientific community interact with the media in a way that increases public cynicism and confusion about health messages?

As well as analysing news reporting of science, and cancer in particular, this session will explore practical difficulties faced by both journalists and scientists in communicating public health messages through the media.

It will also examine whether improvements can be made to the relationship between the science community and the media to make the end product more useful for the people who consume it.

Media reporting of cancer risk

Dr Ben Goldacre

London School of Hygiene and Tropical Medicine, UK

Mainstream media is a key source of information for the public on health. There is good evidence to show that the public act on this information to make health risk behaviour decisions. Sadly mainstream media coverage on cancer is often misleading and built on poor evidence.

Media reporting of disease risk: a scientist's perspective

Dr Alan Dangour

London School of Hygiene and Tropical Medicine, UK

There are many reasons why scientists should be communicating their work, through the media, to the general public. Not least of which is the need for scientists to demonstrate that their work has meaning outside academic research silos and is being conducted to the highest possible standards without external influence or bias. However, scientists still have a lot to learn about how to interact with journalists to ensure that their findings are appropriately reported. I will reflect on the interaction between scientists and journalists drawing on two recent personal experiences of reporting systematic reviews of nutrient content and nutrition-related health benefits of organic food, and the findings of modelling experiments on the potential benefits to health of climate change mitigation policies.

The examples demonstrate that there is still much to learn on how best to communicate the findings of scientific research to the media to ensure that scientific evidence maintains its rightful status in modern debate.

Two-way translational research for understanding cancer aetiology

Dr Christopher Paul Wild

International Agency for Research on Cancer (IARC), France

Environmental exposures, including diet and other lifestyle factors, are a major cause of human cancer. However, the contribution of specific risk factors is difficult to elucidate. One limitation is the inability to accurately measure exposure, particularly past exposure. This is particularly challenging with complex and changing exposures such as nutrition.

Molecular epidemiology promises to improve exposure assessment but this latter area has lagged behind advances in measurement of genetic risk factors. Recently a case has been made to match the genome with an “exposome”, a definition of all exposure events throughout the lifetime of the individual (Wild CP, *Cancer Epid. Bio. Prev.* 14: 1847-1850, 2005; Wild CP, *Mutagenesis*, 24: 117-125, 2009).

Technological advances combined with new understanding of mechanisms of carcinogenesis offer exciting opportunities to the biomarker field. Notably “omics” technologies may provide a step-change in exposure assessment, providing patterns of alterations that reflect specific exposures, including diet. In addition, the increasing recognition of the role of epigenetic changes in carcinogenesis presents a fresh challenge in understanding how exposures influence risk through alterations in DNA methylation, histone modification and microRNA levels for example.

Biomarkers of exposure offer added value over and above their contribution to understanding cancer etiology. For example, these same biomarkers can contribute to establishing the biological plausibility of associations between exposure and disease and may be valuable endpoints in intervention studies; in both instances these approaches hold particular promise in the area of diet and cancer.

The overall importance of this area is highlighted by the large prospective cohort studies which need accurate exposure measurement in order to shed light on the complex gene: environment interactions underlying cancer and other common chronic disorders. A concerted effort is required to develop and validate the required exposure assessment methodology before these cohorts come to maturity.

Preventability and potential public health impact

Professor Elio Riboli

Imperial College London, UK

Preventability can operationally be defined as the proportion of cancer cases that could be prevented in a population by a given public health measure. Previous estimates of the proportion of cancer cases that could potentially be attributed to diet included: The Landmark report by Richard Doll and Richard Peto on “Causes of Cancer” (1981) that estimated that 35 % of cancer cases could be due to diet in the USA (with a range of 10% to 70%). This report considered alcohol separately and did not include physical activity and obesity. In 1989 the National Academy of Sciences of the USA estimated the proportion of cancer attributable to diet at about a third; the 1997 WCRF/AICR report reached a similar conclusion, estimating that, worldwide between 30 and 40% are due to nutritional causes.

In 2009 the WCRF global network published a specialised report “Policy and Action for Cancer Prevention” with the aim of producing recommendations that will help achieve the public health goals set by the 2007 WCRF/AICR Expert Report focused on nutritional causes of cancer. The new updated estimates were elaborated with the aim of convincing policy-makers and decision-takers of the potential impact of following the WCRF report recommendations. Major difficulty for producing up-dated figures of cancer preventability is the scarcity of reliable and representative data on the actual levels of food consumption, physical activity and anthropometric characteristics of different populations around the world.

The WCRF/AICR Policy Report focused on few countries taken as examples of “high income” (UK and USA), middle income (Brazil) and low income (China) areas of the world and analyzed in details twelve cancer sites. Key results indicate that the number of newly diagnosed cancer cases could be reduced by about 20 to 70 percent, depending from the characteristic of the population and the specific cancer site.

A large number of cases of the twelve cancer types examined could be prevented by improving patterns of food, nutrition, physical activity and body fatness. Other cancers may well be prevented by these factors, hence the true figures for all cancers are likely to be higher than the current estimates.

A large number of cases caused by food, nutrition, physical activity and body fatness could be prevented by avoiding overweight and obesity alone. This was particularly true for high income countries. Obesity related cancers are less common in lower income countries but as the number of overweight and obese individuals increase, the preventability estimates for body fatness will also increase with the numbers of preventable cancers.

Poster Abstracts Index

Poster abstracts that will also be presented during the Preferred Paper sessions are highlighted with an asterisk. (*)

Preferred paper abstracts are shown on pages 38 to 41.

The remaining poster abstracts are shown on pages 42 to 70.

A: Nutritional Epidemiology and Cancer

Number	First Author	Title
A1	Aleksandrova, K.	C-reactive Protein and Risk of Colon and Rectal Cancer in the European Prospective Investigation into Cancer and Nutrition
A2	Aune, D.	Fruit and vegetable intakes and colorectal cancer risk: a systematic review and meta-analysis of cohort studies
A3*	Baltar, V.T.	The role of one-carbon metabolism in lung cancer: disentangling metabolic pathways with structural equation models
A4	Botma, A.	Dietary patterns and colorectal adenomas in hereditary colorectal cancer
A5	Brennan, P.	B Vitamins and risk of lung cancer
A6	Carroll, W.D.	Quantitative, Genome-Wide Epigenetic Profiling of CpG Loci identifies Associations with Cord Blood Homocysteine and Birth Weight in Man
A7	Chajès, V.	Plasma phospholipid fatty acid concentrations and risk of gastric adenocarcinomas in the European Prospective Investigation into Cancer and Nutrition (EPIC-EURGAST).
A8	Chan, D.	The WCRF/AICR Continuous Update Project: Folate and colorectal cancer incidence
A9	Chuang, S.	Plasma concentrations of 1-Carbon Metabolite Biomarkers and Pancreatic Cancer Risk in the European Prospective Investigation into Cancer and Nutrition
A10*	Collin, S.	Mendelian randomization: using folate-pathway polymorphisms as instrumental variables to explore associations of folate, vitamin B12 and homocysteine with prostate cancer initiation and progression
A11	Corfe, B.	Butyrate-responsiveness of the human colon at the molecular and cellular level is progressively reversed in colon carcinogenesis
A12	Da Pieve, C.	Screening for colorectal cancer risk biomarkers related to diet
A13	Dahlawi, H.	Pomegranate and its potential for treatment of leukaemia
A14	Egeberg, R.	Intake of wholegrain products and risk of prostate cancer among men in the Danish Diet, Cancer and Health cohort study
A15	Fedirko, V.	Dietary glycemic index and glycemic load, and liver cancer risk within the European Prospective Investigation into Cancer and Nutrition (EPIC)
A16	Gilbert, R.	Associations of circulating and dietary vitamin D with prostate cancer risk: a systematic review and dose-response meta-analysis
A17	Gilsing, A.M.J.	Consumption of dietary fat and meat and risk of ovarian cancer in the Netherlands Cohort Study
A18	Hansen, L.	Metabolite profiling in the Diet, Cancer and Health cohort – a study on colorectal cancer
A19*	Ho, S.C.	Dietary soy intake and changes of mammographic density in premenopausal Chinese women

A20	Hunakova, L.	Modulation of peripheral benzodiazepine receptor and carbonic anhydrase IX expression in human breast and ovarian cell lines by sulforaphane
A21	Hutchinson, J.	High-dose vitamin C supplement use is associated with a personal and family history of cancer in UK women
A22	Jung, A.	Folic acid supplementation and genomic DNA methylation: a double-blind randomised controlled trial
A23	Key, T.	Cancer in vegetarians
A24	K Kolahdooz, F. ey, T.	Intake of antioxidant nutrients and food sources of antioxidants and risk of ovarian cancer
A25	Kuhnle, G.G.C.	GC-MS analysis of urinary biomarkers of alcohol and sugar intake
A26	Kyrø, C.	Intake of whole grains in Scandinavia is associated with "healthy" lifestyle and dietary factors
A27	Lau, R.	The WCRF/AICR Continuous Update Project: Alcohol Intake and Colorectal Cancer Risk
A28	Lee, R.	Dietary westernization and its association with media exposure in two generations of Chinese women
A29	Li, K.	Vitamin and/or Mineral Supplement Use and Total Mortality in a German Cohort
A30	Oliver, S.	Dietary fat, macronutrients, alcohol and screen-detected prostate cancer: results of a population-based case-control study incorporating food diaries
A31*	Olsen, A.	Pre-diagnostic plasma enterolactone levels and survival among women with breast cancer
A32	Outzen, M.	Dietary determinants for haemoglobin-acrylamide and haemoglobin-glycidamide adducts in Danish non-smoking women
A33	Park, J.Y.	Alcohol intake and risk of colorectal cancer: Results from the UK Dietary Cohort Consortium
A34*	Pierre, F.H.F.	Cured meat promotion of colon carcinogenesis in rats is suppressed by calcium and α -tocopherol, which also suppress associated fecal lipid peroxidation in either rats or human volunteers
A35	Rohrmann, S.	Meat and fish consumption and risk of pancreatic cancer
A36*	Romaguera, D.	Adherence to the WCRF/AICR Cancer Prevention Recommendations in the European - Prospective Investigation into Cancer and Nutrition
A37	Roswall, N.	Micronutrient intake and breast cancer characteristics among postmenopausal, Danish women
A38	Travis, R.	Lactase genetic variation, dairy product intake and prostate cancer risk in the European Prospective Investigation into Cancer and Nutrition
A39	Van der Pols, J.C.	Vitamin D and skin cancer risk: an 11-year prospective study in Australia
A40	Velentzis, L.	2007 WCRF/AICRF recommendations and breast cancer progression
A41	Vieira, R.	The WCRF/AICR Continuous Update Project: A Centralised Database System
A42	Wark, P.	Adult height in relation to cancer mortality in the European Prospective Investigation into Cancer and Nutrition (EPIC) cohort
A43	Zaini, R.	Bioactive chemicals from carrot (<i>Daucus carota</i>) juice extracts for the treatment of leukaemia

B: Obesity and/or physical activity and cancer

Number	First Author	Title
B1	Agnoli, C.	Metabolic syndrome and postmenopausal breast cancer according to different receptor status: a case-control study nested in the ORDET cohort
B2	Ambrosini, G.	An energy-dense, high fat, low fibre dietary pattern is prospectively associated with greater adiposity in adolescent girls in the Avon Longitudinal Study of Parents and Children (ALSPAC)
B3	Borena, W.	Serum Triglycerides and Cancer Risk in the Metabolic syndrome and Cancer (Me-Can)
B4	Burton, A.	Adipokines and Prostate Cancer Progression: Nested Case-Control study (Protect)
B5	Lau, R.	The WCRF/AICR Continuous Update Project: Abdominal Fatness and Colorectal Cancer Incidence
B6*	Dossus, L.	Obesity, inflammatory markers and endometrial cancer risk: results from the European Prospective Investigation into Cancer and Nutrition (EPIC)
B7	Douthwaite, W.	The TeesCAKE (Teesside Consumption and Activity for Kids Experience) Project: Study protocol and Year 1 baseline characteristics
B8	Duggan, C.	Weight loss, exercise, and IGF in postmenopausal women
B9	Grote, V.	Glycosylated hemoglobin, serum C-peptide, adiponectin, and risk of pancreatic cancer: a nested-case control study in the European Prospective Investigation into Cancer and Nutrition (EPIC)
B10	Grote, V.	Inflammatory markers as potential risk factors for pancreatic cancer: a prospective casecontrol study nested within the European Prospective Investigation into Cancer and Nutrition (EPIC)
B11	Morois, S.	Lifelong anthropometry and risk of colorectal adenomas: the French E3N-EPIC prospective cohort
B12	Rowlands, M.	Do variations in insulin-like growth factors (IGFs) and their binding proteins (IGFBPs) underlie the associations of adiposity throughout the life course with prostate cancer? Evidence from the ProtecT (Prostate testing for cancer and Treatment) study
B13	Sieri, S.	Obesity, insulin, testosterone and breast cancer risk in the ORDET study
B14	Steindorf, K.	Effects of Physical Activity and Weight-Related Factors on Endogenous Sex Hormones in Postmenopausal Women: Understanding Independent and Joint Roles
B15	Stocks, T.	Metabolic factors and colorectal cancer risk in the Metabolic syndrome and Cancer project (Me-Can)
B16	Yates, M.	The aetiology of Barrett's oesophagus - the effect of BMI, smoking and alcohol in a UK prospective cohort study (EPIC)

C: Other (Behavioural Research, Cancer Survivors, Policy)

Number	First Author	Title
C1*	Doerr, S.	EU livestock subsidies effect on red meat consumption
C2	Strohmaier, S.	Gamma-glutamyltransferase as an indicator of alcohol consumption and its association with cancer incidence
C3	Vrieling, A.	Serum 25-hydroxyvitamin D and postmenopausal breast cancer survival
C4	Yung, T.K.C.	Food avoidance behaviours among Chinese cancer survivors

Proffered Paper Abstracts – in order of appearance in the programme

Session 1

Obesity, inflammatory markers and endometrial cancer risk: Results from the European Prospective Investigation into Cancer and Nutrition (EPIC)

Dossus, L¹, Rinaldi, S², Becker, S¹, Lukanova, A^{1,3}, Kaaks, R¹, on behalf of the EPIC group

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Background: Although a western lifestyle is a well-established risk factor for endometrial cancer, the mechanisms underlying this relationship still have been only partially resolved. Obesity, a major risk factor for endometrial cancer, is a low grade inflammatory state characterised by elevated concentrations of cytokines and acute phase reactants.

Methods: We conducted a case-control study, nested within EPIC, to investigate the associations between endometrial cancer risk and prediagnostic levels of C-reactive protein (CRP), interleukin-6 (IL-6), interleukin-1 receptor antagonist (IL-1Ra), tumor necrosis factor- α (TNF- α), and soluble TNF receptors 1 and 2 (sTNFR1 and sTNFR2). We also examined to which extent these markers can influence the association between obesity and endometrial cancer. This is the first prospective study of its kind to date, comprising 342 incident cases of endometrial cancer, and 645 matched controls.

Results: Conditional logistic regression showed a significant 60% to 90% increased risk of endometrial cancer for women in the highest vs. lowest quartile of each of the measured marker. After adjustment for BMI, the estimates were strongly attenuated and became non-significant for most of the markers. Only for TNF- α and sTNFR1, an increased risk of ~70% persisted, independently of adiposity. The association between BMI and endometrial cancer was also substantially attenuated (~10-20%) after adjustment for inflammatory markers, even when the effects of estrogens had already been taken into account.

Conclusion: We provide direct epidemiologic evidence that chronic inflammation may be a mediator of the association between obesity and endometrial cancer and that endometrial carcinogenesis could be promoted by an inflammatory milieu.

Cured meat promotion of colon carcinogenesis in rats is suppressed by calcium and α -tocopherol, which also suppress associated fecal lipid peroxidation in either rats or human volunteers

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Background: Processed meat intake is associated with colorectal cancer risk. A model cured meat (cooked, nitrite-treated and oxidised high-heme meat) promotes colon carcinogenesis and increases fecal end products of lipid peroxidation in rats (Santarelli et al., Cancer Prevention Research, in press). The aim of the study was to prevent these toxic effects with heme iron binding- or antioxidant-agents, and to validate rodent data in human volunteers.

Methods: Calcium carbonate (150 μ mol/g) or α -tocopherol (0.05%) was added to the model cured meat, and given for 100 days to rats pretreated with dimethylhydrazine. Colons were scored for preneoplastic mucin depleted foci (MDF). In a seven-week cross-over human trial, seventeen volunteers were given the same cured meat (160g/d for 4 days), as such or supplemented with calcium carbonate (1g/d) or α -tocopherol (80mg/d).

Results: In rats, cured meat increased the number of MDF/colon ($p=0.01$) and fecal lipoperoxidation (TBARs). Calcium and α -tocopherol fully normalised the number of MDF/colon ($p=0.01$) and reduced fecal TBARs in cured meat-fed rats. Similarly, TBARs increased in stools of volunteers given cured meat ($42 \pm 5 \mu\text{M MDA equiv.}$, mean $\pm$ SEM) compared with the meat-free period (30 ± 3 , Wilcoxon $P<0.05$). Calcium or α -tocopherol addition fully normalised fecal TBARS in volunteers given cured meat (25 ± 4 and 31 ± 6 respectively, $P<0.05$).

Conclusion: Promotion of colon carcinogenesis by cooked, nitrite-treated and oxidised high-heme cured meat can be suppressed by dietary calcium and α -tocopherol. A WCRF's recommendation is to "avoid processed meat": the identified additives might provide a more accepted alternative to prevent colorectal cancer.

Mendelian randomisation: using folate-pathway polymorphisms as instrumental variables to explore associations of folate, vitamin B12 and homocysteine with prostate cancer initiation and progression

Collin, SM¹, Metcalfe, C¹, Palmer, T^{1,2}, Refsum, H^{3,4}, Smith, AD³, Donovan, J¹, Neal, DE⁵, Hamdy, FC⁶, Martin, RM^{1,2}.

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Background: It has been suggested that low folate concentrations are associated with an increased risk of cancer initiation and high concentrations with more rapid progression. We tested this 'dual-effect' hypothesis in relation to associations of folate, B12 and related metabolites with prostate cancer risk and post-diagnosis prostate-specific antigen velocity (PSAV) using folate-pathway single-nucleotide polymorphisms (SNPs) as instrumental variables (IV).

Methods: Case-control study nested within the UK-wide population-based ProtecT study of prostate cancer in men age 45-69 years. Plasma concentrations of folate, B12, holo-haptocorrin, holo-transcobalamin, total transcobalamin, and total homocysteine (tHcy) were measured in 1,461 cases and 1,507 controls; post-diagnosis PSAV was estimated for 507 men under active monitoring; 13 folate-pathway SNPs were genotyped in 926 cases, 908 controls and 311 actively-monitored men.

Results: Folate was inversely associated with prostate cancer risk in conventional (odds ratio per unit $\log_e(\text{folate})$ (OR)=0.86 (95%CI 0.74, 1.00), P=0.05) and IV (OR=0.20 (0.06, 0.65), P=0.007) analyses. In IV analysis, holo-transcobalamin was inversely (OR=0.44 (0.19, 0.99), P=0.05) and tHcy positively (OR=1.21 (1.05, 1.38), P=0.008) associated with prostate cancer risk, but these associations were not evident in conventional analysis. Folate was positively associated with mean PSAV in conventional (coefficient per unit $\log_e(\text{folate})$ =0.28 (-0.08, 0.65) ng/mL/year, P=0.13) and IV (coefficient=2.66 (-1.70, 7.02) ng/mL/year, P=0.23) analyses.

Conclusion: Folate was associated with a reduced risk of prostate cancer in this case-control study, and may be associated with higher subsequent PSA velocity. These results tend to support the dual-effect hypothesis for opposing roles of folate in prostate cancer initiation and progression.

The role of one-carbon metabolism in lung cancer: disentangling metabolic pathways with structural equation models

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Background: The one-carbon metabolism (OCM), involving B-vitamins and amino acids (homocysteine and methionine), is essential for DNA methylation and synthesis and is likely implicated in carcinogenesis. We propose a pathway analysis between the methylation section of the OCM and lung cancer (LC), in which folate and vitamin B12 are linked to homocysteine levels, while vitamin B6 is linked to methionine levels. Methionine and homocysteine form a methylation-remethylation loop and methionine is linked to LC incidence.

Method: Serum biomarkers were analysed from blood samples taken from 891 LC cases (defined as ICD-10 C34) and 1747 controls matched by country, gender, date of blood collection and date of birth, in a nested study within the European Prospective Investigation into Cancer and Nutrition (EPIC). Statistical analysis was performed using structural equation modelling (SEM), which allows simultaneous regression estimation (whole pathway).

Results: The SEM showed that an increase of 1 $\mu\text{mol/L}$ of methionine reduces lung cancer risk by 4%. Vitamin B12, but not folate, significantly influenced homocysteine levels. Vitamin B6 was significantly related to methionine. The de-methylation-re-methylation loops between homocysteine and methionine were not significant in either direction.

Conclusion: To our knowledge, this is the first study that attempted to associate the B-vitamin levels with lung cancer via components within the OCM. Vitamins B6, B12 and folate all seem to play important protective roles against LC development in the OCM pathway via homocysteine and methionine equilibrium.

Session 2

Pre-diagnostic plasma enterolactone levels and survival among women with breast cancer

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Background: Experimental studies have suggested that the phytoestrogen enterolactone is associated to advantageous mechanisms in breast cancer development. Plant foods such as whole grain cereals, vegetables, and fruits are the primary sources of lignans; lignans are metabolised to enterolactone by intestinal bacteria. Plasma levels of enterolactone have been related to incidence of breast cancer in several studies, but not previously to prognosis after breast cancer diagnosis.

Methods: Between 1993 and 1997, 24,697 postmenopausal women were included in the prospective cohort study Diet, Cancer and Health. Between baseline and year 2000, 434 of the women were diagnosed with breast cancer. Plasma levels of enterolactone were determined by time-resolved fluoroimmunoassay. The 434 cases were followed by record linkage from diagnosis until breast cancer recurrence, mortality (breast cancer or other causes) or December 31 2008. Plasma enterolactone was related to the endpoints by Cox proportional hazard models, adjusted for tumor stage, alcohol intake and use of hormone replacement therapy.

Results: During a median of 10 years, 70 women experienced a breast cancer recurrence and 111 died (80 from breast cancer). When comparing women with baseline enterolactone levels above the median to those with lower levels, decreased hazard rates (HRs) were seen for total mortality (HR: 0.47 (95% CI:0.32-0.68)) and breast cancer specific mortality (HR: 0.56 (95%CI:0.36-0.87)), for recurrence a non-significant tendency towards decreased risk was seen (HR: 0.68 (95% CI:0.42-1.10)).

Conclusion: Higher pre-diagnostic levels of enterolactone were found related to lower mortality among breast cancer patients.

Dietary soy intake and changes of mammographic density in premenopausal Chinese women

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Background: Quantifiable measures of mammographically defined breast composition are related to breast cancer risk. This study aimed to investigate if high soy intake during adolescents predicts low risk breast density pattern and changes overtime.

Methods: This cohort study comprised 817 Chinese premenopausal women aged 35 to 45 years recruited through stratified-cluster sampling from different housing types in Shatin, Hong Kong. Current soy intake (T4) was assessed based on validated soy food frequency questionnaire. Intakes of soy foods during childhood (6-12 y) (T1), adolescents (13-18 y) (T2), and young adulthood (20-34 y) (T3) were also assessed with the assistance of the life-history calendar. Data on potential confounding variables were collected based on standardised measurements and questionnaire. Mammographic films were taken at baseline and at 3-year followup. Percent breast density (BD), based on the digitised mammographic image, was quantitatively assessed using the Cumulus4 software (NB).

Results: Percentage change of BD over the 3-year followup was used as the outcome variable. Univariate analyses showed that baseline body weight, waist circumference, number of live births and soy protein intake at T4 were significant predictors of % BDchange. Soy intake at T2 and T3 were also related to % BDchange. Stepwise multivariate analysis showed that soy intake at T2 was significantly related to a decline in % BD, and together with body weight, age at first birth and baseline BD, accounted for about 15% of the % BD change.

Conclusion: The longitudinal data suggest that adolescent soy intake is protective of high-risk breast density.

Adherence to the WCRF/AICR Cancer Prevention Recommendations in the European Prospective Investigation into Cancer and Nutrition (EPIC)

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Background: In 2007, the WCRF and the AICR issued 8 recommendations (+2 special recommendations) on diet, physical activity and weight management for cancer prevention. We have developed a scoring system to assess the degree of concordance with the WCRF/AICR recommendations in epidemiological studies.

Methods: We have used data from 128,923 men and 257,962 women from 9 European countries, participating in the EPIC study, with dietary, anthropometric, and lifestyle information. We constructed a WCRF/AICR score, incorporating 6 of the WCRF/AICR recommendations for men (regarding body fatness, physical activity, foods and drinks that promote weight gain, plant foods, animal foods, and alcoholic drinks) and 7 (including breastfeeding) for women (score range 0-6 for men; 0-7 for women). Higher scores indicated greater concordance with the WCRF/AICR recommendations. The distribution of the WCRF/AICR scores in the sample, and its association with several socio-demographic and lifestyle variables were examined.

Results: The mean (SD) WCRF/AICR score was 2.84 (1.01) in men and 3.87 (0.98) in women. Participants from Greece and from a UK population of vegetarians and health-conscious people were those with highest scores. Individuals with higher scores showed a healthier dietary profile, characterised by a lower ratio of animal/plant fat and protein. Participants with higher scores were more likely to have a higher educational level, being non-smokers, and having a lower prevalence of chronic diseases.

Conclusion: Independently of the difference in score range, women tended to show higher scores than men. In general, individuals with greater adherence to the WCRF/AICR recommendations were more likely to show a healthier profile.

EU livestock subsidies effect on red meat consumption

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Background: High meat consumption is associated with increased cancer and cardiovascular disease (CVD). Current consumption of red meat in the EU is far above WCRF recommendations, although there is large variation between Member States (MS). Determinants of meat consumption are complex; factors such as price and availability influence consumption of red meat. EU Common Agriculture Policy (CAP) influences what is consumed, but measuring the impact on consumption is complex.

Methods: A systemised review was used to retrieve primary and secondary data.

Results: Data from FAOSTAT suggests that meat consumption is far above WCRF recommendations, up to four-fold in EU Member States, and time trends different trajectories in consumption. A number of studies show a relation between inverse relation between price and consumption, and high price elasticity. However, there is a relative paucity of studies examining the effect of subsidies on consumer prices.

Conclusion: Preliminary results show that price and affordability has a substantial effect on red meat consumption, establishing a clear link between current EU livestock subsidies and consumer prices was not possible in this study. However rechanneling subsidies towards sustainable production of livestock could provide multiple benefits to society in terms of public health, climate change and ensuring global food security.

Poster Abstracts

A: Nutritional Epidemiology and Cancer

C-reactive Protein and Risk of Colon and Rectal Cancer in the European Prospective Investigation into Cancer and Nutrition (EPIC)

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Background: C-reactive protein (CRP) is associated with abdominal adiposity, hyperinsulinemia, dyslipidemia and hyperglycemia and accumulating evidence links these metabolic abnormalities to a higher risk of colorectal cancer. However, the potential effect of these factors on the relationship between CRP and colorectal cancer has not been studied so far.

Methods: We conducted a nested case-control study within the European Prospective Investigation into Cancer and Nutrition (EPIC) among 1,096 incident cases and 1,096 selected with risk-set sampling and matched on various characteristics controls, with the aim to examine the relationship between serum high-sensitivity CRP concentrations and risk of colon and rectal cancer. In particular, we assessed the effect of Body mass index (BMI), waist circumference, C-peptide, HbA1c and HDL-C, on that association.

Results: In conditional logistic regression analysis, after adjustment for smoking, education, alcohol, physical activity, fiber, fruits and vegetables, red and processed meat, fish and shellfish, there was a statistically significant positive association of CRP with risk of colon cancer (RR, highest versus lowest quintile: 1.61; 95%CI: 1.14-2.26; P-trend=0.001), but not of rectal cancer (RR, highest versus lowest quintile: 1.12; 95%CI: 0.71-1.77; P-trend=0.54). When BMI and waist circumference were added to the multivariable model, risk estimates changed slightly (colon cancer: RR, highest versus lowest quintile: 1.42; 95%CI: 0.98-2.05; P-trend=0.01), while further adjustment for C-peptide, HbA1c and HDL-C did not materially alter the results.

Conclusion: These data suggest that CRP is positively associated with risk of colon cancer, but not rectal cancer, independently of general and abdominal adiposity, hyperinsulinemia, dyslipidemia and hyperglycemia.

Fruit and vegetable intakes and colorectal cancer risk: a systematic review and meta-analysis of cohort studies

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Background: The association between fruit and vegetable intakes and colorectal cancer risk has been investigated in numerous studies, but there was only limited suggestive evidence for a reduced risk in the World Cancer Research Fund/American Institute for Cancer Research (WCRF/AICR) report from 2007. In the Continuous Update Project of the WCRF we conducted an updated systematic review and meta-analysis of fruit and vegetable intakes and colorectal cancer incidence.

Methods: We searched the Pubmed database for prospective cohort studies of fruit and vegetable intakes and incident colorectal cancer, up to December 2009. Summary relative risks (RRs) were estimated by use of a random effects model. Details are available at <http://www.dietandcancerreport.org/cu/>.

Results: The summary RR per 100 grams per day of fruit and vegetable intake combined was 0.99 (95% CI: 0.97-1.00) for colorectal cancer (n=7), 0.99 (95% CI: 0.97-1.01) for colon cancer (n=10) and 0.99 (95% CI: 0.96-1.01) for rectal cancer (n=9). The respective summary RRs for fruit intake were 0.97 (95% CI: 0.94-0.99, n=8), 0.98 (95% CI: 0.95-1.01, n=10) and 0.97 (95% CI: 0.92-1.02, n=7) and for vegetable intake were 0.98 (95% CI: 0.96-0.99, n=8), 0.97 (95% CI: 0.95-1.00, n=10) and 1.00 (95% CI: 0.96-1.05, n=7). There was generally little evidence of heterogeneity in the analyses and there was no evidence of publication bias with Egger's test.

Conclusion: This meta-analysis suggests that there is a small, but statistically significant inverse association between fruit and vegetable intakes and colorectal cancer risk.

Dietary patterns and colorectal adenomas in hereditary colorectal cancer

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Background: Little is known about the role of diet in hereditary colorectal cancer. We prospectively investigated associations between dietary patterns and colorectal adenomas in Lynch Syndrome (LS) patients, who have, due to a mutation in one of the mismatch repair (MMR) genes, a very high lifetime colorectal cancer risk.

Methods: Dietary pattern scores were obtained by principal components analysis in 486 LS patients, using an 180-item food frequency questionnaire. Associations were investigated using Cox regression models with robust sandwich estimates controlling for potential confounders.

Results: We identified six dietary patterns: (i) a 'prudent' (high in vegetables, fruits, whole grains, low-fat-dairy, poultry, oils, tea), (ii) 'salad' (high in salad-vegetables, fish, nuts, wine, low in processed meat, pork, potatoes), (iii) 'fried foods' (high in chips, fried snacks, high-fat-dairy, soda), (iv) 'meat' (high in meat, low in whole grains), (v) 'pasta/non-fruit' (low in fruits, non-fat-dairy, high in refined grains), and (vi) 'sweet foods' pattern (high in cookies, cakes, chocolate, low in alcohol). Those within the highest tertile of the 'prudent' pattern had an hazard ratio (HR) of 0.64 (95%CI: 0.33-1.26). The HRs for the 'salad', 'fried foods', 'meat', 'pasta/non-fruit', and 'sweet foods' patterns were 0.78, 1.70, 0.82, 0.98 and 0.40, respectively. Only the HR for the 'sweet foods' pattern was statistically significant (95%CI: 0.17-0.93).

Conclusion: In this cohort of LS patients six dietary patterns were identified. The associations seen for the different dietary patterns with colorectal adenomas were in agreement with those seen in the general Dutch population.

B vitamins and risk of lung cancer

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Background: B-vitamins and factors related to one-carbon metabolism help to maintain DNA integrity and regulate gene expression, and may affect lung cancer risk.

Methods: Within the European Prospective Investigation into Cancer and Nutrition (EPIC) cohort, 899 lung cancer cases and 1,770 matched controls were identified. Serum levels were measured for six one-carbon metabolism factors as well as cotinine.

Results: After accounting for smoking, a lower risk for lung cancer was seen for elevated serum levels of B6 (odds ratio of 4th vs 1st quartile [OR_{4v1}] = 0.44; 95% CI 0.33-0.60; ptrend<0.000001), as well as for serum methionine (OR_{4v1}=0.52; 95% CI, 0.39-0.69; ptrend<0.000001). Similar decreases in risk were observed in never, former and current smokers, indicating that results were not due to confounding by smoking. The magnitude of risk was also constant with increasing length of follow-up, indicating that the associations were not explained by pre-clinical disease. A lower risk was also seen for serum folate, (OR_{4v1}=0.68; 95% CI, 0.51-0.90; ptrend=0.001), although this was apparent only for former and current smokers. Having above median levels of both serum methionine and B6 resulted in a substantially lower lung cancer risk overall (OR=0.41; 95% CI, 0.31-0.54), as well as separately among never (OR=0.36; 95% CI, 0.18-0.71), former (OR=0.51; 95% CI, 0.34-0.76), and current smokers (OR=0.42; 95% CI, 0.27-0.65).

Conclusion: Serum levels of vitamin B6 and methionine were inversely associated with risk of lung cancer.

Quantitative, Genome-Wide epigenetic profiling of CpG Loci identifies associations with cord blood homocysteine and birth weight in man

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Background: Folic acid supplementation during pregnancy is important in the prevention of neural tube defects, promotion of normal development and reduction of the risk of low birth weight. However, there is evidence from animal studies that diets containing high folic acid levels during pregnancy alter the activity of genes involved in cancer and other diseases in the offspring. We recently reported a link between one-carbon supply, changes in genome-wide LINE-1 methylation and fetal outcome in man.

Methods: DNA was extracted from 12 cord-blood DNA samples and genome-wide profiling was used to interrogate 27,000 CpG loci in more than 14,000 genes. Data was analysed by unsupervised hierarchical clustering (UHC) and derived clusters of gene specific methylation were assessed for associations with LINE-1 methylation, one-carbon intermediaries and birth weight.

Results: UHC generated two major clusters; A and B. Compared to cluster A, LINE-1 methylation levels (69.2 vs 74.7; $p=0.028$) and birth weight centile (22.4 vs 57.7; $p=0.019$) were significantly lower in cluster B, while plasma homocysteine concentrations were significantly higher (13.1 vs 7.45; $p=0.038$). CpG sites representing 14 specific genes demonstrated significant correlations with all three parameters.

Conclusion: We show, for the first time in human cord blood, associations between methylation of gene-specific CpGs and the folic acid metabolite homocysteine and birth weight centile. The consequence of these changes to the epigenome in the short or longer-term warrants further investigation. Identified genes may provide useful biomarkers of fetal development and inform policies on supplementation.

Plasma phospholipid fatty acid concentrations and risk of gastric adenocarcinomas in the European Prospective Investigation into Cancer and Nutrition (EPIC-EURGAST)

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Background: Epidemiological data suggested that diet is a predominant factor in the etiology of gastric cancer. However, the role of dietary fatty acids, a modifiable risk factor, remains relatively unexplored. The objective of this study was to determine the association of plasma phospholipid fatty acid concentrations, as biomarkers of exogenous and endogenously produced fatty acids, with the risk of gastric adenocarcinoma in a case-control study nested within the European Prospective Investigation into Cancer and Nutrition (EPIC-EURGAST).

Methods: Fatty acids were measured by gas chromatography in prediagnostic plasma phospholipids from 238 cases matched to 626 controls by age, gender, study center and date of blood donation. Conditional logistic regression models adjusted for *Helicobacter pylori* infection, body mass index, smoking, physical activity, education, and energy intake were used to estimate relative cancer risks.

Results: Positive gastric cancer risk associations were observed in the highest versus the lowest quartiles of plasma oleic acid (odds ratio (OR)=1.72, 95%CI=1.01-2.94, $ptrend=0.047$), elaidic acid (OR=1.63, 95%CI=0.89-2.97, $ptrend=0.122$), di-homo- γ -linolenic (OR=1.92, 95%CI=1.10-3.35, $ptrend=0.030$), α -linolenic acid (OR=3.20, 95%CI=1.70-6.06, $ptrend=0.001$) and the ratio of monounsaturates to saturates, as an indicator of stearoyl-CoA desaturase-1 enzyme activity (OR=1.40, 95%CI=0.81-2.43, $ptrend=0.053$). The positive association with oleic acid was higher in Northern compared to Southern centres, that may reflect a higher meat intake in North.

Conclusion: These data suggest that a specific prediagnostic plasma phospholipid fatty acid profile, presumably reflecting both a complex dietary pattern (possibly high meat intake) and altered fatty acid metabolism, is related to gastric cancer risk.

The WCRF/AICR Continuous Update Project: folate and colorectal cancer incidence

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Background: In their Global Report, the World Cancer Research Fund expert panel concluded that there was limited suggestive evidence that foods containing folate were associated with decreased risk of colorectal cancer. As part of the Continuous Update Project, we conducted an updated systematic review of the associations between dietary folate, total folate intake and plasma/serum folate and colorectal cancer incidence.

Methods: Relevant prospective studies published and identified in PubMed between 2005 and 2009 were reviewed with the previous evidence. Random effect dose-response meta-analysis based on the Der Simonian and Laird method was performed (<http://www.dietandcancerreport.org/cu/>).

Results: The summary relative risk for each 100 mcg/day increase in dietary folate was 0.99 (95% CI = 0.93 – 1.05, seven studies) for colorectal cancer, 0.90 (95% CI = 0.80 – 1.01, six studies) for colon cancer and 1.02 (95% CI = 0.87 – 1.19, four studies) for rectal cancer. The summary RRs for four studies on total folate intake were respectively 0.98, 0.97 and 1.00 and statistically non-significant. For each 5 nmol/L increase in plasma/serum folate, the summary RR was 0.97 (95% CI = 0.93 – 1.01, seven studies), 0.98 (95% CI = 0.83 – 1.15, two studies) and 0.86 (95% CI = 0.67 – 1.09, three studies) respectively. Significant heterogeneity was observed between studies on dietary folate and colon cancer ($I^2 = 60\%$, $P = 0.03$).

Conclusion: There is a possible inverse relation with folate on colorectal cancer, but even with the additional evidence, results remained inconsistent and limited. More studies, especially with dietary and biomarkers data are required to clarify the relations.

Plasma concentrations of 1-Carbon metabolite biomarkers and pancreatic cancer risk in the European Prospective Investigation into Cancer and Nutrition (EPIC) Cohort Study

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Background: We examined the association between plasma concentrations of metabolites and SNPs involved in the one-carbon metabolism pathway and risk of pancreatic cancer in EPIC.

Methods: We conducted a nested case-control study in the EPIC cohort using 463 incident pancreatic cancer cases that occurred during 15 years of follow-up. Controls were selected by incidence density sampling and matched to each case by centre, gender, age, date and time at blood collection, and fasting status. Conditional logistic regression was used to model the associations, considering education, cotinine concentration, alcohol drinking, and BMI as potential confounders.

Results: Weak inverse associations between serum folate and vitamin B6 (pyridoxal phosphate) and pancreatic cancer risk were observed; a U-shape dose-response relationship is apparent. The odds ratios (OR, 95% confidence interval) were 1.00, 0.62 (0.39-1.00), 0.64 (0.39-1.05), 0.53 (0.33-0.86), and 0.83 (0.51-1.35) for each quintile of folate and 1.00, 0.65 (0.42-1.00), 0.49 (0.32-0.77), 0.67 (0.42-1.05), and 0.70 (0.44-1.10) for vitamin B6. However, after stratification by years of follow-up, inverse associations were observed among case-control sets that were followed-up for less than 4 years. Twelve SNPs were found to be associated with pancreatic cancer risk; the BHMT gene, which is involved in the metabolism of homocysteine to methionine, was found to be the most strongly associated to risk (OR=0.44, 0.23-0.84, G/G vs. A/A).

Conclusion: Our results imply that the inverse association between plasma concentrations of folate and vitamin B metabolites may in part be due to reverse causation. The observed associations are worthy of further investigation.

Butyrate-responsiveness of the human colon at the molecular and cellular level is progressively reversed in colon carcinogenesis

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Background: Fibre has been implicated as a chemopreventive for colon cancer, through its fermentation to short-chain fatty acids including butyrate. Butyrate is a potent regulator of cell cycle and cell death in vitro and appears specific against neoplastic cells.

Methods: A cross-sectional study (n=87) was undertaken collecting stool samples and colorectal biopsies from subjects attending GI lists with normal, adenoma or cancer pathologies. Biopsies were collected from the mid-sigmoid in all subjects, and from the lesion and from the contralateral wall in adenoma and cancer subjects. Biopsies were histologically analysed for mitosis, enteroendocrine cell number and for neuropilin-1. Quantitative proteomic analyses were undertaken on duplicate samples.

Results: Correlation between butyrate and mitosis at the mid-sigmoid was positive in normal subjects, weakened in adenoma and a negative in cancer subjects. Correlation of enteroendocrine number and neuropilin-1 with butyrate at each site in the adenoma group was negative at the mid-sigmoid but was lost in the normal tissue adjacent to the adenoma and became positive in the adenoma. Cross-validated proteomic comparison revealed cytoskeletal proteins were progressively altered between sites in the adenoma group.

Conclusion: Our data support models for field cancerisation at level of the whole colon and in the region of neoplasia: there is progressive loss of the normal colon response to butyrate with adenocarcinogenesis and in neoplastic tissue relationships are reversed. This parallels recent understanding of the relationship between folate and colorectal cancer, wherein the nutrient is protective up to formation of the lesion, but a risk factor thereon.

Screening for colorectal cancer risk biomarkers related to diet

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Background: Red and processed meat are associated with high risks of colorectal cancer due to the endogenous formation of O6-carboxymethyl guanine (O6CMG), a potent carcinogen. The aim of our research is to develop liquid chromatography tandem mass spectrometry (LC-MS/MS) analytical Methods: for the measurement of the DNA adducts, such as O6CMG and its nucleoside O6-carboxymethyl deoxyguanosine (O6CMdG), in urine samples and correlate it to different diets.

Methods: Urine samples were collected from volunteers on three different diets (vegetarian as the control, with red and processed meat as the experimental group) over a period of 15 days at the Medical Research Council, Cambridge. Samples were analysed by LC-MS/MS either by direct injection or using a column-switching system with an on-line solid phase extraction (SPE) column.

Results: An LC-MS/MS method was developed and used initially to monitor and quantify O6CMdG and O6CMG using standards in synthetic urine. O6CMG elutes at 4.7 min and have a limit of detection (LOD) of 0.3ng/mL, and O6CMdG elutes at 14.1 min and have a LOD of 0.03ng/mL. The LC-MS/MS direct injection analysis of the clinical samples showed low sensitivity and the need for sample clean-up.

Conclusion: An efficient method for the separation and quantification of O6CMdG and O6CMG was developed. An on-line SPE column system is under development to allow an efficient and rapid processing of a large number of clinical urine samples.

Pomegranate and its potential for treatment of leukaemia

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Background: Studies suggest that pomegranate's contain bioactive chemicals, which show potential for the treatment and prevention of cancers. Pomegranate juice extracts (PJE) have been shown to induce apoptosis, inhibit cellular proliferation and tumor growth in breast, colon, lung, and prostate cell lines. However to date, few studies have investigated the potential of PJE in the treatment of leukaemia.

Methods: The anti-cancerous effect of PJE was investigated on eight leukaemia cell lines from lymphoid and myeloid lineages together with non-tumour haematopoietic stem cells. Cells were treated with 3 concentrations of PJE for 24, 48 and 72 hours. The pro-apoptotic actions of PJE were assessed by two assays: Annexin V/Propidium iodide staining with flow cytometric analysis and DAPI morphological assessment. Cell cycle was investigated using propidium iodide staining of DNA content with flow cytometric analysis and live cell counts were also performed with a haemocytometer.

Results: PJE was shown to significantly induce apoptosis in all cells at all time points. The four lymphoid leukaemia cell lines were more sensitive to the effects of PJE than the 4 myeloid leukaemias ($P < 0.05$), and all tumour cells were affected significantly more than non-tumour control cells ($P < 0.05$). PJE induced a dose-dependent arrest of cells in S phase of the cell cycle. Results were confirmed by DAPI analysis and haemocytometer cell counts.

Conclusion: These results provide evidence PJE contains bioactive compounds, which could be used in the treatment of leukaemia.

Intake of wholegrain products and risk of prostate cancer among men in the Danish Diet, Cancer and Health cohort study

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Background: High intake of wholegrain products may protect against prostate cancer (PC), but overall evidence is inconsistent and has only been based on findings from case-control studies. The aim of the present study was to investigate the relationship between intake of wholegrain products and risk of PC in a large prospective cohort.

Methods: 26,775 men aged 50-64 years participated in the Diet, Cancer and Health cohort study and provided information about diet and potential PC risk factors. During a median follow-up of 9.9 years, we identified 714 PC cases. Associations between wholegrain product intake and PC incidence were analysed using Cox's regression model.

Results: There was no association between total intake of wholegrain products and PC risk and no evidence of that the association differed by grade of disease. When investigating individual wholegrain products (rye bread, whole grain bread and oatmeal), a higher intake of rye bread was associated with a significantly lower risk of PC. The adjusted incidence rate ratio (95% confidence interval) was 0.95 (0.90 to 0.99) per each increment in intake of rye bread of 25 grams per day. There was no association between wholegrain bread or oatmeal intake and PC risk.

Conclusion: Our findings suggest that a higher intake of rye bread is associated with a lower risk of PC.

Dietary glycemic load, consumption of carbohydrates and dietary fibre and risk of liver cancers within the European Prospective Investigation into Cancer and Nutrition (EPIC): preliminary results

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Background: Obesity, insulin resistance and type 2 diabetes have been implicated in liver carcinogenesis. The type, quantity and rate of digestion of dietary carbohydrates [quantified by glycemic index (GI) and load (GL)] have significant effects on postprandial blood glucose response and insulin levels. Higher consumption of high-GI foods may increase type 2 diabetes and obesity risks. Thus, it has been hypothesised that the quantity and quality of dietary carbohydrates may be associated with liver cancer.

Methods: The associations of dietary GI and GL, and intakes of total carbohydrates, sugar, starch and fiber with risk of liver cancers (all morphological sub-types) are being investigated in the EPIC cohort (Ncases=170) as well as in a nested case-control study with assessment of hepatitis virus infections status and measurements of hepatic injury biomarkers and a tumour marker, alpha-fetoprotein (Ncases=104, Ncontrols=200). Dietary/lifestyle data were collected from detailed and validated questionnaires. Blood samples were collected at recruitment and stored in the EPIC bio-bank.

Results: In preliminary analyses based on the cohort study, liver cancer cases were more likely to have diets characterised by lower total fibre and starch, and higher sugar intakes; whilst no differences between liver cancer cases and non-cases were observed for dietary GI and GL, and total carbohydrate consumption. In the nested case-control study, similar differences were observed between liver cancer cases and matched controls.

Conclusion: Results of final analyses, including findings by sub-groups and morphological liver cancer sub-types, for both cohort and nested case-control study components will be presented during the meeting.

Associations of circulating and dietary vitamin D with prostate cancer risk: a systematic review and dose-response meta-analysis

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Background: Vitamin D is considered a potentially modifiable exposure for prostate cancer prevention. We systematically reviewed and meta-analysed the published literature examining associations of vitamin D (dietary intake, circulating concentrations of 25-hydroxy-vitamin-D (25(OH)D) and 1,25-dihydroxy-vitamin-D (1,25(OH)2D)) with prostate cancer.

Methods: We searched over 24,000 papers from 7 electronic databases (to December 2009) for exposures related to vitamin D. We carried out dose-response random-effects meta-analyses which pool the log odds ratio (OR) and 95% confidence intervals (CI) per change in natural units of each exposure. The I² statistic quantifies the percentage of between-study variation due to heterogeneity.

Results: Twenty papers were included in meta-analyses. In prospective studies, the OR per 100IU increase in dietary vitamin D intake was 1.02 (5 studies; CI: 1.00,1.03; I²=0%) for total prostate cancer and 1.01 (2 studies; 0.95,1.07; I²=55%) for aggressive prostate cancer. Four case-control studies examined dietary vitamin D intake but there was a high degree of inconsistency between these studies (I²=61%). The OR per 10ng/mL increase in 25(OH)D was 1.04 (11 studies; 0.98,1.11; I²=0%) for total prostate cancer and 0.97 (4 studies; 0.74,1.28; I²=59%) for aggressive prostate cancer. The OR per 10pg/mL increase in 1,25(OH)2D was 1.02 (7 studies; 0.93,1.12; I²=41%) for total prostate cancer and 0.86 (2 studies; 0.72,1.02; I²=0%) for aggressive prostate cancer.

Conclusion: The published literature provides little evidence to support a role of vitamin D in prostate cancer. More high quality large-scale prospective observational studies are required, with repeated measurement of vitamin D exposures and improved control for confounders.

Consumption of dietary fat and meat and risk of ovarian cancer in the Netherlands Cohort Study

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Background: Evidence linking dietary factors to ovarian cancer is conflicting but several epidemiological studies have suggested that consumption of dietary fat and meat may increase ovarian cancer risk.

Methods: We examined the association of intake of total fat, sources and subtypes of fat, fresh meat, processed meat and fish with ovarian cancer risk within the Netherlands Cohort Study (NLCS). The NLCS includes 62,573 postmenopausal women, aged 55-69 yr at baseline, who completed a baseline questionnaire on dietary habits and other risk factors for cancer in 1986. After 16.3 years of follow-up, 340 ovarian cancer cases and 2161 subcohort members were available for case-cohort analysis. Multivariable hazard ratios (HR) were adjusted for age (yr) at baseline, total energy intake (kcal/day), oral contraceptive use (ever versus never), and number of children.

Results: No clear association between intake of total fat, saturated fat, mono- and polyunsaturated fats, animal- or plant based fat, dairy fat, other fat sources, fresh meat, processed meat and fish and ovarian cancer risk was observed. Consumption of trans unsaturated fatty acids (TFA) was positively associated with ovarian cancer risk. The multivariable HR for women in the highest compared to those in the lowest quintile was 1.51 (95% confidence interval, 1.04-2.20; P_{trend}=0.01). Although no statistically significant interactions by oral contraceptive use or parity were found, TFA intake was associated with ovarian cancer risk in never pill users and parous women only.

Conclusion: This prospective study found intake of trans unsaturated fatty acids to be associated with increased ovarian cancer risk.

Metabolite profiling in the Diet, Cancer and Health cohort – a study on colorectal cancer

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Background: It is envisioned that advanced exploratory metabolite screening tools will provide useful insights into biomarkers for recent as well as habitual intakes of various foods and their possible relation to cancer development. The aim of the present study was to investigate metabolite profiling patterns of plasma samples from colorectal cancer cases and references in the Danish Diet, Cancer and Health cohort study.

Methods: The present study includes 350 women (175 colorectal cancer cases and 175 references) from the Diet, Cancer and Health cohort study, which consists in total of 57,053 men and women free of cancer at baseline (1993-1997). Data on lifestyle and dietary factors were collected at recruitment, and non-fasting blood samples (30 ml) were drawn from each participant. All samples were processed and frozen within 2 hours at -20°C and transferred to liquid nitrogen vapor (max. -150°C). For this study, the plasma fraction was used for analysis. Liquid Chromatography-Mass Spectrometry (LC-MS) metabolite profiling was performed on all samples, and data was processed and aligned using Markerlynx software.

Conclusion: Patterns associating the measured metabolites with dietary markers or with disease status are currently being explored by multivariate chemometric methods in Matlab. Data analysis is currently ongoing, and preliminary results are encouraging, indicating several markers that can effectively discriminate between persons in the study population. In fact, tentative markers have been identified that can differentiate according to BMI or smoking status of the participants. Final results are imminent and will be ready in time for the conference.

Modulation of peripheral benzodiazepine receptor and carbonic anhydrase IX expression in human breast and ovarian cell lines by sulforaphane

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Background: Sulforaphane (SFN) is one of naturally occurring cancer chemopreventive isothiocyanates found in cruciferous vegetables, consumption of which has been associated with reduced risk of cancer. We examined its effects on two markers associated with tumour aggressiveness.

Methods: We studied modulation of hypoxic marker CA IX and mitochondrially located peripheral benzodiazepine receptor (PBR) using flow cytometry, HIF-1 α , VEGF and GLUT1 mRNA levels by RT PCR and HIF-1 α protein expression by western blot in human triple-negative breast cancer cell line MDA-MB-231, ovarian A2780 cell line and its resistant variants A2780/ADR and A2780/CP.

Results: SFN downregulated PBR expression in a dose dependent manner in all presented cell lines, statistically significant at 5 microM concentration in A2780 resistant sublines. SFN reduced hypoxia-induced up-regulation of CAIX concomitant with HIF-1 α degradation in human A2780 ovarian tumor cells independently of their resistance type, but significantly affected neither HIF-1 α and CA IX protein expression, nor VEGF and GLUT1 mRNA levels in breast MDA-MB-231 cells.

Conclusion: SFN reduced hypoxic induction of CA IX in human A2780 ovarian tumour cells independently of their resistance type, but did not affect hypoxic pathway in MDA-MB-231 breast cancer cell line where its regulation might be disturbed.

High-dose vitamin C supplement use is associated with a personal and family history of cancer in UK women

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Background: The World Cancer Research Fund does not recommend the use of supplements for cancer survivors and for cancer prevention generally; indeed some high-dose supplements may be harmful and evidence of the effects of high-dose vitamin C on cancer progression has been controversial. Information about the characteristics of UK high-dose vitamin C users and their personal and family history of cancer is limited.

Method: This cross-sectional analysis examined associations between vitamin C supplement use recorded in 4-day diaries and users' characteristics, and self-reported personal and family history of cancer recorded by questionnaire for 12,453 middle-aged women in the UK Women's Cohort Study (UKWCS).

Results: Regular high-dose vitamin C users (≥ 1000 mg) compared to those not taking high doses had higher socioeconomic status, appeared to rely more on alternative practitioners rather than family or private doctors, took more supplement types, had higher fruit and vegetable and lower meat consumption, were most likely to be ex-smokers, and to drink little or no alcohol. Women who self-reported having had cancer (OR=1.33, 95% CI: 1.00, 1.76) or specifically breast cancer (OR=1.70 (95% CI: 1.14, 2.55)), or reported a family history of cancer (OR=1.16, 95% CI: 0.95, 1.41) or breast cancer (OR=1.26 (95% CI: 1.01, 1.58)) had increased odds of being regular high-dose users after adjusting for socio-demographic and health behaviours.

Conclusion: High-dose vitamin C use (≥ 1000 mg) by UK women was associated with healthier behaviours and a personal or family history of breast cancer and total cancer; though the direction of causality cannot be determined.

Folic acid supplementation and genomic DNA methylation: a double-blind randomised controlled trial

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Background: Cancer is a multi-step process caused by changes in normal cellular mechanisms. Normally, genomic DNA is heavily methylated and has been shown to be hypomethylated in leucocytes of individuals with and without cancer. DNA methylation occurs as part of folate-mediated one-carbon metabolism, where folate functions as a donor and acceptor of one-carbon units. The availability of methyl groups for DNA methylation depends largely on diet. We investigated whether daily supplementation of 800 µg folic acid for 3 years would increase global DNA methylation compared to placebo in individuals with elevated homocysteine levels.

Methods: Participants were a subset of Dutch men and post-menopausal women aged 50-70 years with plasma total homocysteine concentrations between 13 µmol/L and 26 µmol/L who participated in the Folic Acid and Carotid Intima-media Thickness trial between November 1999 and April 2001. Two hundred sixteen participants were allocated to treatment of 800 µg folic acid/day or placebo. Participants in the folic acid arm of the trial were randomly selected and individually matched to placebo participants on age, current smoking status, and MTHFR C677T genotype. Quantification of global DNA methylation in leucocytes was measured using liquid chromatography-tandem mass spectrometry.

Results: There was neither a difference in the effect of supplementation after three years between the two groups (difference=0.008, 95%CI=-0.05,0.07, P=0.79), nor a significant change in DNA methylation within groups (folic acid difference=0.02, 95%CI=-0.03,0.08, P=0.39; placebo difference=0.01, 95%CI=-0.04,0.06, P=0.62).

Conclusion: We did not find evidence of changes in genomic DNA methylation after 3 years of folic acid supplementation.

Cancer in vegetarians

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Background: Vegetarian diets may reduce the risk for some types of cancer because they are free from red and processed meat and are also relatively rich in dietary fibre, fruit and vegetables.

Methods: Cancer incidence was examined in two prospective studies in the UK, the Oxford Vegetarian Study and EPIC-Oxford. 40,965 non-vegetarians and 20,601 vegetarians were followed for an average of 13 years. Relative risks were estimated by Cox regression, stratified by study and sex and adjusted for age, smoking, alcohol, body mass index, physical activity and, for women only, parity and oral contraceptive use.

Results: Preliminary findings (to be updated) showed that, compared to meat-eaters, vegetarians had lower risks for the following cancers: stomach, relative risk (RR) =0.36 (95% CI 0.16-0.78); bladder RR=0.47 (0.25-0.89); lymphatic and haematopoietic tissues RR=0.55 (0.39-0.78); and for all cancers combined RR=0.88 (0.81-0.96).

Conclusion: The incidence of some cancers may be lower in vegetarians than in meat-eaters.

Intake of antioxidant nutrients and food sources of antioxidants and risk of ovarian cancer

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Background: We sought to examine the association between intake of fruit and vegetables and associated antioxidant nutrients and ovarian cancer risk in Australian women. We also examined whether the relationships between these nutrients/foods and risk were modified by smoking status.

Methods: Data came from two Australian population-based case-control studies conducted 10 years apart. Analyses included 2012 cases and 2174 controls. Dietary information was obtained via a semi-quantitative food frequency questionnaire. Multivariable-adjusted odds ratios (OR) were estimated separately for each study using logistic regression modelling and results of the two studies were combined using random-effects models.

Results: Women with the highest fruit intake had a significantly decreased risk of ovarian cancer (combined OR=0.73, 95%CI: 0.71-0.76) although no association was seen for vitamin A and C rich fruit. Weak associations were also seen for total vegetables (combined OR=0.83; 95%CI: 0.67-1.03) and green leafy vegetables (combined OR=0.86, 95%CI: 0.70-1.07). While there was no association between intake of vitamins C and E (from food or from food and supplements combined) and ovarian cancer risk, high vitamin C intake was associated with a 40% reduction in risk among current smokers. High beta-carotene intake was associated with a 22% reduction in risk (combined OR=0.78, 95%CI: 0.78-0.86).

Conclusions: Our results suggest that high fruit and vegetable intake may reduce risk of ovarian cancer and individuals who smoke might benefit to a greater extent from a high anti-oxidant diet.

GC-MS analysis of urinary biomarkers of alcohol and sugar intake

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Background: Dietary data obtained from food surveys are often inaccurate because they rely entirely on information supplied by participants. For this reason, nutritional biomarkers that allow objective dietary assessment are important to investigate associations between diet and health. Urinary fructose and sucrose have been developed as biomarker for sugar intake. In a recent population study, self-reported sugar intake was not associated with obesity, though sugar intake measured using these biomarkers was(1). Ethyl-glucuronide is widely used as biomarker for alcohol intake, in particular in forensics, but is not commonly used in epidemiological studies. We have developed a robust and sensitive GC-MS method for the large-scale analysis of urinary biomarkers of sugar and alcohol intake to determine measurement errors and explore associations with self-reported intake and cancer risk.

Methods: EPIC Norfolk is a cohort 25,000 men and women aged 45 to 75 years at recruitment (1993). Detailed dietary, health and lifestyle information as well as serum and urine samples were collected at baseline. EPIC Norfolk spot urines were analysed by GC-MS: after addition of internal standards (13C6-fructose, 13C12-sucrose, D5-ethyl glucuronide), proteins were precipitated, the supernatant dried and derivatised with methoxyamine and MSTFA.

Results: The developed method was robust and within the concentration range of 5 µM to 150 µM for fructose, sucrose and ethyl-glucuronide, the GC-MS method had a precision and accuracy of less than 10%. Preliminary results from the analysis of 2000 samples will be presented at the conference.

Conclusion: The newly developed GC-MS method allows the reliable and accurate determination of biomarkers of alcohol and sugar intake in epidemiological studies.

1.Bingham S. et al., CEBP (2007) 16:1651

Intake of whole grains in Scandinavia is associated with “healthy” lifestyle and dietary factors

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Background: Intake of whole grains (WG) has been associated with a lower risk of developing chronic diseases including some types of cancer. Because the intake of WG has been associated with “healthy” dietary and lifestyle factors, studies on WG/cancer associations need to consider these factors as confounders. In Scandinavia, the intake and the variation of the intake of WG are high, and these countries therefore serve as a good basis for conducting research on WG/cancer associations. The aim of this study was to describe the intake of WG in Norway, Sweden and Denmark and reveal the dietary and lifestyle factors associated with the intake of WG.

Methods: A cross sectional study based on three Scandinavian prospective cohorts using a single 24-hour dietary recall from 8702 persons was used. Multiple linear regression analyses were used for the analyses with the total intake of WG as the dependent variable.

Results: The intake of WG was highest in Norway for women and lowest in Denmark and Sweden for both men and women depending on the assessment method used (WG products vs. calculated total WG content). Fruits, vegetables, dairy products and tea were found to be positively associated with the intake of WG, whereas red meat, white bread and being a smoker were inversely associated with the intake.

Conclusion: The intake of WG is positively associated with “healthy” factors and inversely associated with “less healthy” factors, suggesting that these factors are important to consider as confounders when studying WG/cancer associations.

The WCRF/AICR Continuous Update Project: alcohol intake and colorectal cancer risk

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Background: In 2007, the World Cancer Research Fund and American Institute for Cancer Research (WCRF/AICR) expert panel concluded that alcoholic drinks convincingly increases colorectal cancer (CRC) risk in men. Findings for women appeared to be weaker and inconsistent. Here we update the existing evidence from prospective studies on alcohol intake and CRC risk.

Methods: Relevant prospective studies on alcohol intake and CRC (expressed as grams of ethanol or number of drinks) were identified by performing PubMed search until December 2009. Dose-response meta-analysis was performed by pooling study-specific relative risks (RR) using a random-effects model (<http://www.dietandcancerreport.org/cu/>).

Results: Thirty-one publications were identified on alcohol intake (g/day), of which 15 could be included in the dose-response meta-analysis. In men, the summary RR per 10g/day increase for colorectal, colon and rectal cancer were 1.11 (95%CI=1.08-1.15; n=7studies), 1.10 (95%CI=1.06-1.14; n=10) and 1.10 (95%CI=1.07-1.13; n=9), respectively; in women, the summary RR per 10g/day increase for colorectal, colon and rectal cancer were 1.07 (95%CI=0.98-1.17; n=2), 1.03 (95%CI=0.98-1.09; n=7) and 1.09 (95%CI=1.02-1.16; n=6), respectively. There was substantial heterogeneity for colon cancer in men (I²=62%, p=0.004), but not in women. The summary RR for each drink/day was 1.22 (95%CI=0.94-1.59) for CRC and 1.26 (95%CI=0.99-1.60) for colon cancer for men and women combined. There were too few studies to stratify drinks/day by gender.

Conclusions: In this updated meta-analysis, a suggestive increase in colorectal cancer risk was observed among women with every 10g/day alcohol consumed. The overall positive association, however, remains stronger in men than women.

Dietary westernisation and its association with media exposure in two generations of Chinese women

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Background: Dietary westernisation is suggested to contribute to the increase in colorectal and breast cancer incidences in Asia. However, the western dietary pattern (WDP) practised in the Chinese population and its association with media exposure has not been studied.

Methods: Between May 2008 and August 2009, 103 mother-daughter pairs (aged 18-49y) recruited from an on-going community-based cohort study were interviewed using a validated dietary history questionnaire adopt to assess WDP. WDP was identified by principal components analysis. Media exposure was self-reported by a validated questionnaire. Anthropometric measurements were conducted using standard procedures.

Results: WDP was associated with intake of meats (red, processed, poultry), fast foods (sugar drink, French fries, pizza), seafood, cakes and snacks, high-fat dairy, added oils, cream soup, and egg; frequency of breakfast skipping and food-away-from-home. WDP was also associated with smoking, higher body mass index in mothers and higher prevalence of central obesity in both mothers and daughters. Daughters had a higher WDP score than their mother ($p < 0.001$). Age- and energy-adjusted multivariate analysis indicated mother's WDP score was predicted by years living in Hong Kong ($\beta = 0.201$; $p = 0.009$), weekly frequency of family meals ($\beta = -0.212$; $p = 0.005$), daily hours spent on total media exposure ($\beta = 0.372$; $p < 0.001$) and radio listening [log-transformed] ($\beta = -0.220$; $p = 0.019$); daughter's WDP score was predicted by mother's WDP score ($\beta = 0.107$; $p = 0.019$), weekly frequency of family meals ($\beta = -0.170$; $p = 0.001$), daily hours spent on TV viewing ($\beta = 0.121$; $p = 0.010$), and smoking status ($\beta = 0.144$; $p = 0.004$).

Conclusion: An intergeneration transfer of WDP was apparent in Chinese families. Practice of WDP was associated with media exposure in Chinese women.

Vitamin and/or mineral supplement use and total mortality in a German cohort

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Background: Vitamin and mineral supplements are commonly used by general population in developed countries. From a scientific perspective, however, no consensus has been reached regarding their effects on all-cause mortality due to inconsistent epidemiological findings. All possible scenarios, i.e., null, inverse, and positive associations, have been reported in both observational and experimental studies.

Methods: Between 1994 and 1998, 11,928 men and 13,612 women aged 35-64 years were recruited into the EPIC-Heidelberg cohort. Demographic, lifestyle, dietary, and other health-related information was collected at baseline. Regular vitamin and/or mineral supplement use was assessed at baseline and two follow-up surveys. A follow-up survey was implemented triennially, in which vital status was verified. Subjects with cancer and cardiovascular disease at recruitment and deaths occurred in the first two years of follow-up were excluded from statistical analyses.

Results: During an average follow-up of 11.1 years, 932 deaths occurred. No significant association was observed between baseline supplement use and all-cause mortality when demographic, lifestyle, dietary, and other health-related factors were adjusted for. However, significantly reduced all-cause mortality was observed in users who are men (HR 0.79, 95% CI 0.65-0.96). In comparison to never users, non-users at recruitment who started supplement use on later occasions had a significantly increased risk (1.63, 1.42-2.34).

Conclusion: Regular vitamin and/or mineral supplement use may reduce all-cause mortality in men. The increased risk in non-users at recruitment who started supplement use on later occasions suggests that initiation of supplement use in later life may be in response to beginning health problems.

Dietary fat, macronutrients, alcohol and screen-detected prostate cancer: results of a population-based case-control study incorporating food diaries

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Background: Diet has been suggested as a risk factor for prostate cancer but evidence for diet-disease associations remains weak. Measurement error associated with food frequency questionnaires (FFQs) may contribute to this uncertainty. This study incorporated diet measurement both through FFQ and 7-day food diary in a study of the risk of screen-detected disease.

Methods: A case-control study nested within the detection phase of a RCT of treatment for localised prostate cancer. Measures of energy, macronutrient and alcohol intake were made using both 7-day food diaries and FFQs on 530 men with screen-detected prostate cancer aged 50-70 years and 536 matched controls.

Results: Positive associations were observed between disease risk and intake of total fat (OR highest vs. lowest quartile = 1.64 95% CI 1.10-2.45) and saturated fat (OR highest vs. lowest quartile = 1.56 95% CI 1.02-2.38) based on food diaries, but were not seen for FFQ data. However, associations were sensitive to choice of analytical model, being observed in energy-partition but not energy density models. An inverse association was observed with protein intake estimated by FFQ (OR highest vs. lowest quartile = 0.66 95% CI 0.45-0.96), no associations were observed with carbohydrate or alcohol intake.

Conclusion: The study provides limited support for a positive association between risk of screen-detected prostate cancer and intake of total fat and saturated fat in particular.

Dietary determinants for haemoglobin-acrylamide and haemoglobin-glycidamide adducts in Danish non-smoking women

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Background: Acrylamide (AA) is a probable human carcinogen that is formed in heat treated carbohydrate rich foods. The validity of food frequency questionnaires (FFQs) to assess AA exposure has been questioned. The aim of this cross-sectional study was to investigate dietary determinants of haemoglobin-acrylamide adduct (Hb-AA) and haemoglobin-glycidamide adduct (Hb-GA) concentrations.

Methods: This study included 537 non-smoking postmenopausal women aged 50-65 years who participated in the Diet, Cancer, and Health cohort (1993-1997). Blood samples and information on dietary and lifestyle variables obtained from self-administrated questionnaires were collected at entry into the cohort. From the blood samples Hb-AA and Hb-GA concentrations in erythrocytes were analysed by LC/MS/MS. Dietary determinants were evaluated by multiple linear regression analyses adjusted for age and smoking behaviour among ex-smokers.

Results: The median (5%, 95% percentile) for Hb-AA was 35 pmol/g globin (17, 89) and for Hb-GA 21 pmol/g globin (8, 49). Intakes of coffee and chips were statistically significantly associated with a 4% per 200g/d ($P < 0.0001$) and 18% per 5g/d ($P = 0.002$) higher Hb-AA concentration, respectively. This model explained 17% of the variation in Hb-AA concentration. Intakes of coffee and biscuits/crackers were statistically significantly associated with a 3% per 200g/d ($P = 0.005$) and 12% per 10g/d ($P = 0.01$) higher Hb-GA concentration, respectively. This model explained 12% of the variation in Hb-GA concentration.

Conclusion: Only few dietary determinants of Hb-AA and Hb-GA were identified. Dietary intake measured by a FFQ explains only to a limited extent the variation in Hb-AA and Hb-GA concentrations.

Alcohol intake and risk of colorectal cancer: results from the UK Dietary Cohort Consortium

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Background: Epidemiological studies suggest that excessive alcohol intake increases colorectal cancer (CRC) risk. However, findings regarding tumour subsites and sex differences have been inconsistent.

Methods: We investigated prospective associations between alcohol intake on overall and site- and sex- specific CRC risk. Analyses were conducted on 579 CRC cases and 1,996 matched controls nested within the UK Dietary Cohort Consortium which comprises seven established UK cohorts using standardised data from food diaries as a main nutritional method and repeated using data from food frequency questionnaire (FFQ).

Results: Compared with individuals in the lightest category of drinkers (>0 to <5 g/day), the multivariable odds ratios of CRC were 1.16 (95% CI: 0.88, 1.53) for non-drinkers, 0.91 (95% CI: 0.67, 1.24) for drinkers with 5 to <15 g/day, 0.90 (95% CI: 0.65, 1.25) for drinkers with 15 to <30 g/day, 1.02 (95% CI: 0.66, 1.58) for drinkers with 30 to <45 g/day, and 1.19 (95% CI: 0.75, 1.91) for drinkers with ≥45 g/day (P trend=0.82). No clear associations were observed between site-specific CRC risk and alcohol intake in either sex. Analyses using FFQ showed similar results.

Conclusion: Within a moderate range of alcohol intake, there was no evidence for an increased risk of colorectal cancer in the UK Dietary Cohort Consortium.

Meat and fish consumption and risk of pancreatic cancer – results from the European Prospective Investigation into Cancer and Nutrition

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Background: Pancreatic cancer is the fourth most common cause of cancer death worldwide with large geographical variation, which implies the contribution of diet and lifestyle in its etiology. We examined the association between meat consumption and risk of pancreatic cancer in the European Prospective Investigation into Cancer and Nutrition (EPIC).

Methods: Included in our analysis were 410,411 EPIC participants from 10 European countries aged 35-65 (females) and 40-65 (males) at recruitment between 1992 and 2000. Until 2007, 555 non-endocrine pancreatic cancer cases have been observed. Relative risks (RR) and 95% confidence intervals (CI) were computed using multivariately adjusted Cox Hazard Regression models.

Results: The consumption of red meat was associated with an increased risk of pancreatic cancer in women (RR=1.46, 95% CI 0.98-2.16, 80+ vs. <20 g/day), but not in men (RR=0.76, 95% CI 0.47-1.25; p-interaction=0.07). After calibration, a 50 gram increase in red meat statistically significantly increased the risk in women by 75%. The consumption of processed meat, fish, and of poultry were not related to pancreatic cancer risk. Including only microscopically confirmed cases strengthened the observed association between red meat consumption and pancreatic cancer risk in women.

Conclusion: Our results partly support the conclusion of the World Cancer Research Fund that red meat consumption may increase the risk of pancreatic cancer, although it is unclear why this association should be restricted to women.

Micronutrient intake and breast cancer characteristics among postmenopausal, Danish women

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Background: Few studies on micronutrients and postmenopausal breast cancer have examined the association with breast cancer characteristics. The aim of the present study was to investigate associations between vitamin C, E, folate and beta-carotene from diet and supplements and risk of postmenopausal breast cancer subtypes defined by histology (ductal/lobular), estrogen receptor (ER) and progesterone receptor (PGR) status.

Methods: In a prospective cohort study of 26.224 postmenopausal women information on diet, supplements and lifestyle was collected through questionnaires. 1072 cases were identified during follow-up. Incidence rate ratios of total breast cancers and breast cancer subtypes related to micronutrient-intake were calculated using Cox Proportional Hazard analyses.

Results: The present study found no association between overall breast cancer and any micronutrients, while some effects were shown when stratifying by breast cancer subtypes: Dietary but not supplemental beta-carotene showed a protective effect against lobular breast cancer (IRR: 0.72; 95% CI: 0.57-0.91). Dietary vitamin E was associated with decreased risk of ER and PGR positive breast cancer (IRR: 0.50, 95% CI: 0.25-0.98) and dietary folate was associated with increased risk of ER and PGR positive breast cancer (IRR: 1.27; 95% CI: 1.03-1.95).

Conclusion: The present study found no effect of micronutrients on overall risk of postmenopausal breast cancer, but indicated possible effects of micronutrients in subgroups of breast cancer, with a potential beneficial effect of dietary beta-carotene in lobular breast cancer and dietary vitamin E in ER+PGR+ breast cancer and a potential harmful effect of dietary folate in ER+PGR+ breast cancer.

Lactase genetic variation, dairy product intake and prostate cancer risk in the European Prospective Investigation into Cancer and Nutrition

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Background: We have previously found that a high intake of dairy protein is associated with an increased risk for prostate cancer in the European Prospective Investigation into Cancer and Nutrition (EPIC). Findings from other prospective studies are similar but not conclusive. We examined the hypothesis that individuals carrying the rs4988235 T allele (lactase-persistence variant) will have a higher intake of lactose-rich dairy products and an increased risk for prostate cancer.

Methods: A nested case-control study was conducted in EPIC with 630 cases of prostate cancer and 873 matched controls. The C/T-13910 lactase variant (rs4988235) was genotyped. Relative risks for prostate cancer in relation to lactase genotype were estimated by conditional logistic regression.

Results: Lactase genotype frequency varied strikingly between countries, with frequencies of the T allele ranging from 7% and 17% in Greece and Italy, respectively, to 75% in Sweden and 79% in Denmark. Intake of milk and total dairy products varied significantly by lactase genotype after adjustment for recruitment centre, with the highest intake being observed among men homozygous for the T (lactase-persistence) allele. A similar variation in dairy intake was seen within countries. Although the lactase variant was not significantly associated with risk for prostate cancer, numbers were small and the confidence intervals were wide.

Conclusion: Lactase genotype frequencies vary substantially across Europe and lactase genotype is strongly associated with milk intake, even within countries. There is no significant association, however, between lactase genotype and prostate cancer risk, although confidence intervals are wide.

Vitamin D and skin cancer risk: an 11-year prospective study in Australia

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Background: Vitamin D is thought to play a role in prevention of cancer. Molecular work shows that vitamin D is part of an intrinsic protective mechanism that protects skin cells against UV damage. Whether vitamin D is protective against skin cancer risk in the population is not known.

Methods: We carried out an 11-year prospective study in a subtropical community-based sample of Australian adults to study the association between vitamin D status and skin cancer risk. Serum 25(OH)-vitamin D concentrations were ascertained at baseline in 1996. All incident basal cell (BCC) and squamous cell carcinomas (SCC) were recorded and histologically confirmed between 1997 and 2007. Relative risk of BCC and SCC by quantiles of serum 25(OH)-vitamin D concentrations will be ascertained using negative binomial modelling (skin cancer counts) and Poisson regression (number of persons affected) adjusting for confounders.

Results: Of 931 participants, 209 participants (22%) had serum 25(OH)-vitamin D concentrations below 50 nmol/L indicating vitamin D insufficiency, including 13 vitamin D deficient persons (1%) who had concentrations below 25 nmol/L. Associations between serum 25(OH)-vitamin D concentrations and risk of BCC and SCC will be presented.

Conclusion: Vitamin D insufficiency is common in this Australian adult subtropical population. An association between serum 25(OH)-vitamin D concentrations and skin cancer risk would have major implications for skin cancer prevention efforts.

2007 WCRF/AICRF recommendations and breast cancer progression

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Background: Research on diet and breast cancer progression is limited, but as the number of survivors increases there is a need for lifestyle recommendations. In its 2007 report, one of the WCRF/AICR recommendations was that guidelines provided for cancer prevention could be used by cancer survivors. We investigated the association between adherence to these recommendations and breast cancer recurrence in a cohort of 1228 survivors.

Methods: An index was constructed based on the 2007 WCRF/AICR recommendations, excluding breastfeeding, but with smoking history included. Dietary, anthropometric and lifestyle data collected from 1228 DietCompLyf participants one year after diagnosis were assessed and one point allocated for meeting each recommendation, with a maximum score of 11. Cox proportional hazard models were used to evaluate the relationship between the scores and time to recurrence after diagnosis.

Results: During 4.4 years of follow-up, 122 women were diagnosed with recurrence. Overall, poor compliance to the recommendations was observed, with the median score of the cohort being 4. The lowest compliance was for frequent physical activity, followed by avoidance of processed meat. Although no association was observed between higher lifestyle scores and recurrence for the whole cohort, postmenopausal women who adhered to 6 or more recommendations were significantly less likely to recur [HR 0.28 (0.10, 0.78), P_{trend}=0.005] than women who adhered to 3 recommendations or less.

Conclusion: Adherence to a combination of health behaviours as recommended by the 2007 WCRF/AICR guidelines was found to be protective against breast cancer recurrence in postmenopausal women.

The WCRF/AICR Continuous Update Project: a centralised database system

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Background: As a part of the WCRF Continuous Update Project a new system has been setup to allow the extraction of data for systematic literature reviews and subsequent consultation and analysis. The nature of the information to be captured is complex, especially with the addition of new research areas such as gene-diet interactions. An improved system facilitating data extraction and consultation without the need of technical database knowledge was needed.

Methods: The database server consists of a MySQL engine, hosted in an Imperial College facility. In order to provide wider usability, the application runs on the Java platform, allowing it to be used on most operating systems without modification.

The system enables data extraction using features such as field validation and automatic retrieval of paper information from PubMed (<http://eutils.ncbi.nlm.nih.gov/>).

Results: Currently the database contains over 6600 papers and over 114,000 models, reporting on more than 3700 exposures. The possibility to perform queries independently of detailed data organisation knowledge has allowed quick and easy access to this information by all researchers working on the Continuous Update Project, facilitating data extraction. Moreover, the application permits users to choose the format in which data is exported, allowing its use in other software packages for analysis. Interface control and a query builder features further enhance usability of the system.

Conclusion: Basing the system on a server allows data to be accessible globally for authorised users. Additional features implemented in the client application permit access to the database in a more user-friendly environment which improves data extraction and retrievability.

Adult height in relation to cancer mortality in the European Prospective Investigation into Cancer and Nutrition (EPIC) cohort

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Background: Adult height is a marker for genetic factors as well as for environmental, hormonal and nutritional factors occurring early in life. Evidence so far suggests that taller people are more likely to be diagnosed and die from cancer than shorter people, which we verify in a large multicentre prospective cohort study.

Methods: Within the European Prospective Investigation into Cancer and Nutrition (EPIC), standing height was measured in adults (216,280 women and 131,544 men) from nine countries between 1991 and 1999. Within the follow-up period that comprised 9.8 years on average, 2,716 men and 2,692 women died of cancer. Hazard ratios (HR) of cancer mortality according to height were estimated from Cox proportional hazard models adjusted for smoking status, educational level, alcohol consumption, physical activity, weight and waist circumference.

Results: Preliminary analyses showed that cancer mortality rates were higher among taller than among shorter men and women. Among men, a 6% increase in the hazard rate was observed for every 5 cm increase in height (HR=1.06, 95% confidence interval=1.03-1.10). A very similar increase was seen in women (HR=1.06, 95% confidence interval=1.02-1.10).

Conclusion: These initial findings suggest that factors leading to higher attained adult height or its consequences affect cancer mortality rates in Europeans. Further work will include analyses on cancer incidence and site-specific risks. Our observations do not have direct implications for cancer prevention but could point to underlying mechanisms and thereby trigger further research. The latter may lead to public health interventions on the long term.

Bioactive chemicals from carrot (*Daucus carota*) juice extracts for the treatment of leukaemia

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Background: Overwhelming evidence indicates that the consumption of fruits and vegetables with strong antioxidant properties correlate with reduced risk of a number of cancers include leukaemia. Carrots contain beneficial agents including beta-carotene and polyacetylene, which could be effective in the treatment of leukaemia. This study investigated the effect of organic carrot juice extracts (CJE) on four myeloid, four lymphoid together with non-tumour haematopoietic stem cells (HSC).

Methods: Annexin V and Propidium iodide (PI) staining followed by flow cytometric analysis and DAPI (4'-6-Diamidino-2-phenylindole) morphological analysis used to investigate the induction of apoptosis. Moreover, PI cell cycle analysis and cell counts used to investigate the inhibition of cell proliferation.

Results: This study, demonstrated that treatment of leukaemia cell lines with CJE showed a decrease in the number of live cells and an increase in both apoptotic and necrotic cells in a dose responsive manner with significant decrease in cell number with increasing concentration of carrot juice especially following 72 hour treatments. We demonstrated that the lymphoid cell lines were affected to a greater extent than myeloid leukaemia cell lines. Additionally, the non-tumour haematopoietic stem cells were less sensitive to CJE treatment than the majority of the leukaemia cell lines.

Conclusion: This study has shown that extracts from carrots can induce cell death and cause cell cycle arrest in leukaemia cell lines which suggests carrots could be an excellent source of bioactive chemicals which could be used in the treatment and or prevention of leukaemia.

B: Obesity and/or physical activity and cancer

Metabolic syndrome and postmenopausal breast cancer according to different receptor status: a case-control study nested in the ORDET cohort

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Background: Metabolic syndrome, a cluster of metabolic abnormalities and cardiovascular risk factors (i.e. high serum glucose, high triglycerides, low HDL-cholesterol, high blood pressure, and abdominal obesity) that occur in subjects with impaired insulin sensitivity, is an emerging risk factor for breast cancer, especially among postmenopausal women. However, no prospective study investigated the role of metabolic syndrome in relation to different receptor status.

Methods: Association between metabolic syndrome and breast cancer risk defined by receptor status was assessed in a nested case-control study on postmenopausal women of the ORDET cohort who gave blood samples in 1987-1992. Relative risks (RRs) were estimated by polytomous logistic regression.

Results: After a median follow-up of 13.5 years, 166 women developed breast cancer; of these cases, 124 were ER-positive, 93 were PR-positive and 25 were HER2-positive. Metabolic syndrome (i.e. presence of three or more components) was significantly associated with increased risk of ER-positive (RR 1.67, 95%CI 1.08-2.58), PR-positive (RR 1.74, 95%CI 1.07-2.84) and HER2-negative cancer (RR 1.70, 95%CI 1.11-2.62), with a significant risk increase for increasing number of components (P for trend 0.004 for ER-positive and PR-positive cancer, and 0.002 for HER2-negative cancer). No association was found between ER-negative, PR-negative and HER2-positive breast cancer.

Conclusion: This is the first prospective study evaluating the effect of metabolic syndrome on postmenopausal breast cancer according to different receptor status. We found that metabolic syndrome was associated with a significant increase of ER-positive, PR-positive and HER2-negative postmenopausal breast cancer, but not in ER-negative, PR-negative and HER2-positive breast cancer.

An energy-dense, high fat, low fibre dietary pattern is prospectively associated with greater adiposity in adolescent girls in the Avon Longitudinal Study of Parents and Children (ALSPAC)

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Background: Specific dietary risk factors for weight gain in young people are poorly understood. We explored prospective relationships between an energy dense, high fat, low fibre dietary pattern (DP) and fat mass index (FMI) in a childhood cohort.

Methods: A 3d food diary was completed by 5,550 children at 10 and 13 y of age. The DP was derived at each age using reduced rank regression on 46 food groups, with dietary energy density, fat and fibre as response variables. Fat mass (FM) was measured using DXA at 9, 13 and 15 y and FMI calculated as FM/Htx. The odds of being in the highest quintile of FMI were examined using multinomial logistic regression, adjusting for age, gender, baseline FMI, pubertal stage and physical activity.

Results: The DP at 10 and 13 y was high in confectionery, low fibre bread, cakes and biscuits, and low in fruits and vegetables. Among girls, a 1 SD increase in DP z score at 10 y was associated with an OR of 1.43 (95% CI, 1.12 1.83) for the top quintile of FMI at 13 y. A 1 SD increase in DP z score at 13 y was associated with an OR of 1.30 (95% CI, 1.05 1.62) for the top quintile of FMI at 15 y. No associations were observed for boys.

Conclusion: Among girls, an energy dense, high fat, low fibre dietary pattern during pre adolescence is associated with an increased risk of excess adiposity 3-5 years later, confirming similar associations seen at 5 and 7 y in this cohort.

Serum triglycerides and cancer risk in the metabolic syndrome and cancer (Me-Can) collaborative study

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Background: High levels of serum triglycerides are related to increased risk of cardiovascular diseases; however, little is known about their role in cancer risk. We examined the association between serum triglycerides and cancer risk in a large prospective study of 514,097 participants among seven European cohorts.

Methods: The metabolic syndrome and cancer project (Me-Can) includes cohorts from Norway, Austria, and Sweden. The current study included data on 257,585 men and 256,512 women. Mean age at baseline was 44 years and mean follow-up time was 10.4 years. A total of 38,746 men and women were diagnosed with cancer. Cox regression models were used to estimate relative risk (RR) of cancer for triglyceride levels in quintiles and as a continuous variable. All risk estimates were corrected for random error in exposure measurement by use of regression dilution ratio.

Results: Relative risk for the top versus bottom quintiles of triglycerides of overall cancer was 1.16 (95% confidence interval (CI) 1.06-1.26) in men and 1.15 (1.05-1.27) in women. For specific cancers, significant increases were found for cancers of the colon (RR: 1.96, 95% CI, 1.44-2.67), respiratory tract (RR: 1.42; 95% CI, 1.12-1.80) and the kidney (RR: 1.85, 95% CI, 1.14-3.02) among men and for respiratory (RR: 2.10; 95% CI, 1.41-3.12) and cervical cancers (RR: 1.88; 95% CI, 1.07-3.30) among women.

Conclusion: Data from our study provided evidence for a possible role of serum triglycerides in cancer development.

Adipokines and prostate cancer progression: nested case-control study (ProtecT)

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Background: Obesity has been associated with increased risk of aggressive of prostate cancer and with poor disease outcome. Adipokines, small signalling molecules produced by adipocytes, may mediate this association and be useful markers of prognosis. We investigated the association of two adipokines, leptin and adiponectin, with prostate cancer stage in a nested case-control study.

Methods: 146 men with localised (TNM stage ≤2) prostate cancer and 146 with advanced stage (T≥3) prostate cancer were selected from the prostate-specific antigen (PSA) case finding phase of the UK population-based Prostate testing for cancer and Treatment (ProtecT) study. Adiponectin and leptin concentrations were established in sera taken at enrolment by enzyme-linked immunosorbent assay.

Results: Statistical analyses are ongoing, but preliminary odd ratios for adiponectin, leptin and leptin:adiponectin ratio (4th quartile versus 1st quartile of logged values), adjusted for age at blood draw, are 0.84 (95% confidence interval (CI) 0.44-1.61), 0.75 (95% CI 0.39-1.44) and 0.80 (95% CI 0.42-1.54) respectively. These estimates were not altered following adjustment for body mass index.

Conclusion: This study found little evidence that adiponectin and leptin separately, and as a ratio, are very strongly related to risk of advanced prostate cancer in PSA-detected cases, but the study was small and we cannot rule out modest effects.

The WCRF/AICR Continuous Update Project: abdominal fatness and colorectal cancer incidence

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Background: In 2007, the World Cancer Research Fund and American Institute for Cancer Research (WCRF/AICR) expert panel concluded that abdominal fatness convincingly increases colorectal cancer. It is not known whether waist circumference (WC) and waist-hip ratio (WHR) are risk factors independent of overall body mass (BMI). Here we update the existing evidence from prospective studies on the association of abdominal fatness and examine specifically if the association is independent of BMI.

Methods: Relevant prospective studies were identified by performing PubMed search until December 2009. Dose-response meta-analysis was performed by pooling study-specific relative risks (RR) using a random-effects model (<http://www.dietandcancerreport.org/cu/>).

Results: Results from seven prospective studies were included. For WC, the summary RR per inch increase for colorectal, colon and rectal cancer were 1.03 (95%CI=1.02-1.04), 1.05 (95%CI=1.03-1.06) and 1.03 (95%CI=1.01-1.04), respectively. Four studies adjusted for BMI and the summary RRs per inch increase for colorectal, colon and rectal cancer were 1.02 (95%CI=1.01-1.04), 1.04 (95%CI=1.01-1.07) and 1.01 (95%CI=0.98-1.05), respectively. Significant heterogeneity was observed for colon cancer. For WHR, the summary RR per 0.1 increment increase for colorectal, colon and rectal cancer were 1.17 (95%CI=1.09-1.25), 1.23 (95%CI=1.14-1.32) and 1.20 (95%CI=1.07-1.34), respectively. Only two studies reported results adjusted for BMI therefore no meta-analysis was performed.

Conclusion: The evidence accumulated after the publication of the WCRF/AICR report confirms that abdominal fatness is associated with an increased colorectal cancer risk. Results from a subset of the studies indicate that abdominal obesity might be a risk factor independent of BMI.

The TeesCAKE (Teesside Consumption and Activity for Kids Experience) Project: study protocol and Year 1 baseline characteristics

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Purpose: To test whether a 26-week health promotion programme (TeesCAKE) can reduce obesity in children aged 9-10 in schools in the Tees Valley area.

Methods: All schools in the Tees Valley area were invited to participate; the first four appropriate schools to sign up were selected; two schools were randomly assigned to participate in the study for three consecutive years; the other two were assigned as control groups for the same period. The intervention involves children taking part in a combined health promotion programme called TeesCAKE between January and July each year for three years. The study is built on the concept of partnership working (including parental involvement) and involves several key local groups including sports development teams, and the intervention includes dance sessions, football training and food preparation sessions. Outcome measures will be taken at 0, 6, 12 and 18 months. Overall around 680 children will be invited to take part (340 in the intervention and 340 in the control group).

Results: 187 participants provided baseline data for the Year 1 cohort (intervention group=88). There were no significant differences in mean BMI or waist circumference between the intervention and control schools. Overweight and obesity levels in the control schools were 20% and 6% respectively compared with 23% and 9% in the intervention schools.

Conclusion: This study will provide evidence on the efficacy of an evidence-based health promotion programme (TeesCAKE) which aims to improve eating behaviours and activity levels amongst socially deprived children aged 9-10 years and their families.

Weight loss, exercise, and IGF in postmenopausal women

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Background: Obesity, sedentary lifestyle, and elevated insulin-like growth factor (IGF) are associated with risk for several cancers. We investigated the effects of 1-year exercise (E) and/or caloric restriction (CR) on serum IGF1 and its binding protein IGFBP3 in overweight/obese post-menopausal women.

Methods: Women were randomized to: E (N=117), CR (N=118), E+CR (N=116), or control (C, N=87); attrition was 9%. The CR was a group-based modification of the Diabetes Prevention Program. The E intervention was 45 min/day, 5 days/week moderate-intensity facility and home aerobic exercise. Mean 1-year weight change was -8.7% in CR, -2.4% in E, and -10.8% in E+CR ($p < 0.001$, 0.03, & < 0.001) vs. -0.6% in C. IGF-1 and IGFBP-3 were measured at the Pollack Oncology Laboratory at baseline and 1-year using ELISA (Diagnostic Systems Laboratories, Beckman Coulter). Mean changes were compared between groups (intent-to-treat) using generalised estimating equations.

Results: At baseline, mean IGF1 and IGFBP3 were 166.4 ng/mL and 5042 ng/mL, respectively. IGF-1 correlated inversely and significantly with body mass index ($\rho = -0.25$, $P < 0.0001$), total fat mass ($\rho = -0.22$, $P < 0.0001$) and percent fat mass ($\rho = -0.19$, $P < 0.0001$). IGFBP-3 correlated inversely, but weakly with percent body fat ($\rho = -0.10$, $P = 0.03$). IGF1 increased 1.6% and 1.9% in CR and E+CR groups ($p = 0.05$ and 0.04 , respectively, vs. decrease of 2.8% in C). IGFBP3 did not change in any intervention group vs. controls.

Conclusion: Weight loss and exercise do not lower IGF1 levels. Therefore the associations between obesity, physical activity, and cancer risk are not likely due to alterations in IGF or its binding protein in humans.

Glycosylated hemoglobin, serum C-peptide, adiponectin, and risk of pancreatic cancer: a nested-case control study in the European Prospective Investigation into Cancer and Nutrition (EPIC)

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Background: Excess body weight and type-2 diabetes mellitus, risk factors of pancreatic cancer, are characterized by metabolic disturbances, particularly increased glucose and insulin concentrations, and by decreased levels of adiponectin. The aim of this study was to investigate the associations of prediagnostic levels of glycosylated hemoglobin (HbA1c), C-peptide, and adiponectin with pancreatic cancer risk.

Methods: We conducted a case-control study nested within EPIC. Blood samples of 466 pancreatic cancer cases and 466 individually matched controls were analyzed by immunoassays. Multivariate conditional logistic regression was used to estimate odds ratios (OR).

Results: We observed a significant increase in pancreatic cancer risk with increasing levels of HbA1c (OR 1.45, 95% CI 1.16-1.81, for 1 unit increase in HbA1c concentrations). A stronger association was observed among subjects with a shorter follow-up time (< 2 years, OR 2.21, 95% CI 1.26-3.86), for former smokers (OR 1.92, 95% CI 1.29-2.87), and for non-diabetics (OR 1.68, 95% CI 1.00-2.81).

Elevated adiponectin levels reduced pancreatic risk only among never smokers (OR, 0.94; 95% CI, 0.89-0.99), but increased the risk in current smokers (OR, 1.08; 95% CI, 0.99-1.81). Excluding diabetic subjects strengthened the risk association of adiponectin with pancreatic cancer. C-peptide was not significantly associated with pancreatic cancer risk.

Conclusions: Our study supports the relationship of increased HbA1c levels with pancreatic cancer risk. In addition, higher adiponectin concentrations may be associated with the development of pancreatic cancer, with the direction of risk depending on the smoking status.

Inflammatory markers as potential risk factors for pancreatic cancer: a prospective case-control study nested within the European Prospective Investigation into Cancer and Nutrition (EPIC)

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Background: Central obesity and diabetes are associated with increased risk of pancreatic cancer, and both are considered to be states of low-grade chronic inflammation. Whether obesity or diabetes related inflammation plays an independent role in pancreatic cancer development is not clear and we, therefore, aim to examine prediagnostic levels of C-reactive protein (CRP), interleukin 6 (IL-6), and tumor necrosis factor receptor 2 (TNF-R2) with pancreatic cancer risk.

Methods: A nested case-control study was conducted within EPIC. Blood samples of 466 pancreatic cancer cases and 466 individually matched controls were analysed by immunoassays. Multivariate conditional logistic regression was used to estimate odds ratios (OR).

Results: We observed a significant increase in pancreatic cancer risk with increasing levels of TNF-R2 (OR 1.35, 95% CI 1.04-1.75; for 1 ng/mL increase) and a borderline significant increase with CRP concentrations (OR 1.08, 95% CI 0.99-1.18); for 1 mg/L increase). Adjustment for BMI, diabetes or smoking status attenuated the risk association of TNF-R2 and CRP to non-significance. Tests for heterogeneity resulted in significant differences by smoking and diabetes status for TNF-R2 (p interaction 0.001, 0.002; respectively) and CRP (p interaction 0.010, 0.011; respectively), but risk estimates were not significant. IL-6 was not significantly associated with pancreatic cancer risk.

Conclusion: Prediagnostic CRP and TNF-R2 levels do not seem to be associated with pancreatic cancer risk independently from excess body weight. However, since this is the first study investigating the association of inflammatory markers with pancreatic cancer risk, further prospective studies are needed to clarify their role.

Lifelong anthropometry and risk of colorectal adenomas: the French E3N-EPIC prospective cohort

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Background: Anthropometric factors have been associated with colorectal adenomas, but results are sparse concerning anthropometry throughout life and its consequences in adulthood. We aimed to explore associations between colorectal adenomas and anthropometric factors in childhood and early adulthood, including birth weight and height and body silhouettes at different ages, in a large prospective cohort of French women.

Methods: We analysed the 18,178 women of the French E3N-EPIC cohort who underwent a colonoscopy during follow-up (1993-2002), including 1,445 who developed a first colorectal adenoma.

Results: In Cox multivariate proportional hazard regression models, weight and height at birth, and silhouettes in childhood and adulthood were not associated with overall colorectal adenoma risk. However, both a large birth weight and a large body shape in adulthood were associated with an increased risk of left colon adenomas (Hazard Ratio (HR) = 1.28; 95% Confidence Interval (CI): 1.01, 1.63 for high versus medium birth weight; and HR = 1.28; 95% CI: 1.05, 1.57 for high versus low body shape at baseline). At age 8 years, the largest silhouettes were associated with a decreased risk of rectal adenomas compared with the lowest silhouette (HR = 0.70; 95% CI: 0.51, 0.97; p homogeneity colon versus rectum = 0.01). Associations did not differ between advanced and non-advanced adenomas.

Conclusion: In our findings add evidence for a differential role of anthropometry at different ages and on different sites of the large bowel, and suggest that early life events may influence colorectal adenoma genesis.

Do variations in insulin-like growth factors (IGFs) and their binding proteins (IGFBPs) underlie the associations of adiposity throughout the life course with prostate cancer? Evidence from the ProtecT (Prostate testing for cancer and Treatment) study

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Background: Increased adiposity is associated with prostate cancer risk and progression, and obesity-related variations in insulin-like growth factors (IGF-I and -II) and their binding proteins (IGFBPs) could underlie these associations.

Methods: We investigated associations of adiposity throughout the life course (determined retrospectively) with serum levels of IGFs and IGFBPs in a population-based study of 1,106 healthy men.

Results: IGF-I and IGF-II showed inverted U-shaped associations with adult body mass index (BMI) (p quadratic model = 0.04 and 0.06, respectively), although differences between quartiles with the highest and lowest IGF-I levels were small (no more than 5ng/ml). IGFBP-2 was strongly inversely related to adult BMI (-22% change per SD increase in BMI; 95% confidence interval (CI) -24%,-19%) and waist circumference (-18% change per SD increase in waist circumference; 95% CI -20%,-15%) (p<0.001). IGFBP-3 was positively related to BMI (63.5ng/ml increase per SD increase in BMI; 95% CI -2.69,129.8, p=0.06). IGFBP-2 and IGFBP-3 were strongly related to body shape change from childhood to adulthood, with men who gained the most weight having the lowest IGFBP-2 (9% lower per category body shape change; 95% CI -11%,-7%, p<0.001) and the highest IGFBP-3 (50ng/ml increase per category; 95% CI 8, 92, p=0.02).

Conclusion: We provide evidence that adiposity and change in body shape through the life course are related to the IGF system, with the largest effect being on IGFBP-2. The results suggest that circulating IGF-I levels may not be important mediators of the association of adiposity with aggressive prostate cancer, but the role of IGFBP-2 deserves further investigation.

Obesity, insulin, testosterone and breast cancer risk in the ORDET

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Background: There is considerable evidence that obesity, metabolic syndrome and insulin resistance are associated with increased risk of breast and other cancers. We sought further insight into the relation of these variables to breast cancer risk.

Methods: In a nested-design case-control study on women recruited to the ORDET cohort who gave blood samples in 1987-1992, we assessed associations of breast cancer risk with serum testosterone, serum insulin, and obesity. After a median 13.5 years of follow-up, 371 women developed breast cancer. Relative risks (RRs) of developing breast cancer were estimated by conditional logistic regression.

Results: Overweight women (body mass index >25) with high (above median) levels of both testosterone and insulin had increased breast cancer risk compared to lean women with low testosterone and insulin (RR of 1.68, 95% CI=1.13-2.49). No associations were found for other categories (high and low levels of insulin and testosterone in lean and obese women). On stratifying by menopausal status at baseline, high total testosterone plus high insulin were associated with increased breast cancer risk in both pre- and post-menopausal overweight women (RR 1.75, 95% CI=0.99-3.12 in pre-menopause; 1.64, 95% CI=0.89-3.01 in post-menopause) compared to lean women with low insulin and testosterone.

Conclusion: This is the first study to provide evidence that three variables together (high circulating testosterone, high insulin increase and body mass index >25) increase the risk of developing breast cancer. The risk was significantly higher than for lean women with low testosterone and low high insulin, and was independent of menopausal status.

Effects of physical activity and weight-related factors on endogenous sex hormones in postmenopausal women: understanding independent and joint roles

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Background: Physical activity is hypothesised to lower postmenopausal breast cancer risk at least in part via hormonal pathways. We investigated whether recent physical activity was associated with endogenous sex hormone levels after menopause and to what extent associations were accounted for by weight-related factors.

Methods: A cross-sectional study in 1260 postmenopausal women was conducted. Associations between serum concentrations of sex hormones and sex-hormone-binding-globulin (SHBG) and different physical activity variables (leisure-time physical activity, exercise, bicycling, walking) were investigated. Generalised linear models were adjusted for potential confounders with a special focus on confounding and effect modification by weight-related factors (BMI, body weight, waist and hip circumference, waist-to-hip ratio).

Results: Higher levels of exercise were significantly associated with lower levels of estrone and total and free testosterone in adjusted models. All associations remained significant after further adjustment for weight-related factors, however, associations with estrone and free testosterone were attenuated. None of the physical activity variables were significantly related to total and free estradiol, androstenedione, or SHBG. Additionally, we did not observe effect modification by weight-related factors.

Conclusion: Our data suggest that exercise may lead to lower circulating levels of estrone and total and free testosterone in postmenopausal women. Effects on estrone and free testosterone might be partly due to body weight and body composition changes, while effects on testosterone appear to be largely independent of weight-related factors. These independent effects could be one explanation for the frequently observed reductions in postmenopausal breast cancer risk through physical activity, even after accounting for BMI.

Metabolic factors and colorectal cancer risk in the Metabolic syndrome and Cancer project (Me-Can)

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Background: The metabolic syndrome (MetS) has been related to an increased risk of colorectal cancer, but the modest size of previous studies precluded detailed characterisation of the association. The present study included nearly ten times as many cases compared to the largest previous prospective study.

Methods: In the Metabolic syndrome and Cancer project (Me-Can), data on body mass index (BMI), blood pressure, and blood levels of glucose, cholesterol, and triglycerides were available for 578,700 men and women. Mean age at baseline was 44.0 years and mean follow-up time was 12.0 years. Relative risks (RR) of colorectal cancer by continuous Z scores (mean = 0, standard deviation = 1) of factors, and for a combined MetS score, were calculated from Cox regression models. All risk estimates were corrected for random error in exposure measurement based on data from repeated examinations in 133,820 individuals.

Results: During follow-up, 2,834 men and 1,861 women were diagnosed with colorectal cancer. The RR of colorectal cancer for the MetS score was 1.25 (95% confidence interval, 1.18-1.32) in men, and 1.14 (1.06-1.22) in women. Significant associations were also found in men for BMI (RR = 1.07; 1.02-1.13), blood pressure (RR = 1.10; 1.02-1.18), and triglycerides (RR = 1.17; 1.06-1.28), and in women for BMI (RR = 1.08; 1.01-1.15). There was no significant positive interaction between the metabolic factors on risk.

Conclusion: The combination of metabolic factors, and some separate factors, was related to an increased risk of colorectal cancer but there was no interaction between metabolic factors.

The aetiology of Barrett's oesophagus - the effect of BMI, smoking and alcohol in a UK prospective cohort study (EPIC)

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Background: Determining the aetiology of Barrett's oesophagus, a major risk factor for oesophageal adenocarcinoma, is important to prevent the dramatic increase in incidence of this cancer. The aim of this study was to investigate the effects of body mass index (BMI), smoking and alcohol in the aetiology of Barrett's oesophagus, for the first time in a prospective cohort epidemiological study.

Methods: A total of 23750 healthy men and women, aged 45-74 years, were recruited between 1993 and 1997 into the EPIC-Norfolk Study (European Prospective Investigation into Cancer). Participants provided information on height, weight, smoking and alcohol intake at baseline health checks and from validated questionnaires. The cohort was then monitored until December 2008, and using a hospital histopathology databases identified, those who developed Barrett's oesophagus. A full cohort analysis was performed using cox regression, to calculate hazard ratios (HR).

Results: In the cohort, 105 patients (80% men) developed Barrett's oesophagus at a median age of 60.4 years (range 40.1-75.4 years). Of these, 93% have metaplasia, without dysplasia. BMI was positively associated with developing Barrett's oesophagus (BMI >25 HR=1.58, 95% CI= 1.01-2.47 p=<0.05; BMI > 35 HR= 4.86, 95% CI= 1.09-21.74 p=0.04). The HR for trend across standard categories of BMI was 1.28 (95% CI=1.04-1.59). Adjusting for social class does not affect risk.

Conclusion: BMI was associated with an increased risk of developing Barrett's in a dose-dependent manner. If future work supports a causal association, then encouraging a reduction in the population's BMI may help prevent oesophageal adenocarcinoma.

C: Other (Behavioural Research, Cancer Survivors, Policy)

Gamma-glutamyltransferase as an indicator of alcohol consumption and its association with cancer incidence

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Background: Recent evidence suggests that elevated levels of gamma-glutamyltransferase (GGT) are associated with both incidence and mortality of cardiovascular disease and cancer. Although GGT is regarded as a marker of liver function which may in turn reflect alcohol consumption, to date, no study has investigated the relationship of GGT with cancer sites known to be alcohol-related or non-related.

Methods: First visit measurements in 94,628 adult women and 80,224 men screened for metabolic risk factors as part of the Vorarlberg Health Monitoring & Promotion Programme (VHM&PP). During a median follow-up of 13 years, a total of 5136 incident cancers were diagnosed in men and 4665 in women. Sex-specific Cox proportional hazards models, adjusted for age, body-mass index and smoking were performed to estimate hazard ratios (HR) and 95% confidence intervals (95%CI) per quintiles of GGT.

Results: In males, the highest GGT-quintile revealed a high risk of alcohol-related cancer incidence (HR=2.20, 95%CI 1.74-2.78). In females, there was a less pronounced effect of GGT with cancer sites known to be related with alcohol consumption (HR=1.16, 1.02-1.32). Additionally, elevated GGT was found to be significantly related to cancer sites with weak or no evidence of alcohol consumption as a risk factor.

Conclusion: Although elevated GGT levels were associated with incidence of alcohol-related cancers, most markedly in men, there were still effects of GGT in non-alcohol related cancer sites. This suggests that alcohol consumption explains the relationship between GGT and cancer outcomes only in part.

Serum 25-hydroxyvitamin D and postmenopausal breast cancer survival

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Background: Vitamin D has been inconsistently associated with breast cancer risk. Thus far, only one study has investigated its association with survival after breast cancer diagnosis.

Methods: Follow-up information from 1,376 women aged 50-74 years with incident postmenopausal breast cancer, diagnosed between 2002 and 2005 and interviewed at recruitment into a German population-based case-control study (MARIE), was used to assess the effect of post-diagnostic serum 25-hydroxyvitamin D [25(OH)D] concentrations on mortality. Serum samples were collected a median of 83 days after diagnosis, and all deaths were verified by death certificates. Hazard ratios for all-cause and breast cancer-specific mortality were calculated using Cox proportional hazards models, stratified by age at diagnosis and season of blood draw and adjusted for other prognostic factors.

Results: Median follow-up time was 5.8 years; 199 deaths occurred, 149 due to breast cancer. Serum 25(OH)D concentrations were inversely associated with all-cause mortality. Compared with the lowest category (<30 nmol/L), univariate hazard ratios and 95% confidence intervals for the higher categories of 25(OH)D (30-45, 45-60, 60-75 and ≥75 nmol/L) were 0.47 (0.31-0.69), 0.39 (0.25-0.61), 0.44 (0.27-0.71), and 0.34 (0.20-0.58), respectively (p-trend<0.0001). The association was attenuated but remained significant after multivariate adjustment for tumor stage, personal history of other cancers, and self-reported diabetes (p-trend<0.01). Further adjustment for therapy, ER/PR status, and lifestyle factors did not substantially change the risk estimates. Similar results were obtained for breast cancer-specific mortality (p-trend<0.05).

Conclusion: Higher serum 25(OH)D concentrations may be associated with improved overall and breast cancer-specific survival in postmenopausal breast cancer patients.

Food avoidance behaviours among Chinese cancer survivors

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Background: It is extremely common among Chinese cancer survivors to practice drastic diet restriction. Yet there is no study investigates the significance of over-restriction on food consumption based on a traditional Chinese medicine perspective.

Methods: Cross-sectional survey by face-to-face interview to a target of 150 cancer survivors in two cancer clinics in Hong Kong.

Results: Preliminary results based on the first 83 subjects reveal a substantial proportion of them consider they should avoid beef (65.1%), poultry (71.08%), fish without scale (60.25%), seafood (74.7%), fruit of sweeter flavour (60.2%), and food that is high in protein (79.5%). Majority of them agree that avoidance of these specific foods item suppresses the growth of cancer cells (60.2%); improves the medical treatment effects (71.0%); alleviates the cancer patients' discomfort (57.8%); strengthens the cancer patients' immunity (51.8%) and reduces the chance of cancer recurrence (66.3%). Reducing the intake of food that is high in nutritive value is considered to be beneficial because of suppression of cancer cell growth (32.5%), improvement in medical treatment effects (31.3%), improvement of immunity (18.07%) and reduction of likelihood of recurrence (25.3%).

Conclusion: The preliminary findings reveal the subjects have distorted perception about food and its association with cancer prognosis. Upon the completion of the study, the association of their food avoidance attitude and the actual dietary intake will also be presented in WCRF conference in London.

WCRF International and the WCRF Global Network

World Cancer Research Fund Global Network

Since its foundation in 1982, the World Cancer Research Fund (WCRF) global network has been dedicated to the prevention of cancer through food, nutrition, physical activity and body weight. All members of the global network share the same vision, heritage and mission.

WCRF International is the umbrella association for the WCRF global network and plays a leading role in directing and supporting the research activities of the network. Working with researchers, health professionals, policy-makers and other health organisations throughout the world, we provide people with the information they need to make choices that can reduce their chances of developing cancer.

For more information please visit www.wcrf.org

WCRF International – Our work

Second Expert and Policy Reports

In 2007 WCRF/American Institute for Cancer Research (AICR) published the Second Expert Report: *Food, Nutrition, Physical Activity and the Prevention of Cancer: a Global Perspective*. It is the most comprehensive analysis of the literature on diet, physical activity, obesity and cancer. The Second Expert Report features personal recommendations for people as well as goals for the population as a whole. But setting these targets is just one step. Equally important is understanding how to achieve them successfully.

A companion publication to the Second Expert Report addresses why people practise particular eating and physical activity behaviours over a lifetime. *Policy and Action for Cancer Prevention* examines policies and actions that may change those behaviours. It also provides advice and guidance on what can be done to influence and change the lifestyle choices that people make, as they relate to their risk of cancer. For more information please visit www.dietandcancerreport.org

Continuous Update Project

To keep the evidence current and updated into the future, WCRF and AICR are undertaking the Continuous Update Project, in collaboration with Imperial College London (ICL), who will conduct regular systematic reviews of the published literature.

The Continuous Update Project will provide the scientific community with a comprehensive and up-to-date depiction of scientific developments on the relationship between diet, physical activity, obesity and cancer. It will also provide an impartial analysis and interpretation of the data as a basis for reviewing and where necessary revising WCRF/AICR's cancer prevention recommendations which were based on the 2007 Second Expert Report.

Grant Programmes

WCRF International manages and administers the International Research Grant Programme (consisting of the Regular Grant Programme and the Request for Application (RFA) Programme) on behalf of its member organisations in the UK, the Netherlands, Hong Kong and France. WCRF International's Research Grant Programme is dedicated to funding research on the role of food, nutrition, physical activity and body weight in relation to cancer.

Applications for the WCRF International Grant Programme will be accepted from anywhere in the world except the Americas (North America including the USA and Canada, Central America, the Caribbean and South America).

Our US member organisation, AICR, operates its own Grant Programme. Applications for the AICR Grant Programme will be accepted from the Americas. For more information on the AICR Grant Programme please visit www.aicr.org

WCRF International Academy

The WCRF International Academy is a new educational initiative. The main objective is to provide educational materials and activities for a broad range of audiences, ranging from scientists to the general public, about the importance and impact of food, nutrition, physical activity and body weight in cancer development, progression and survival.

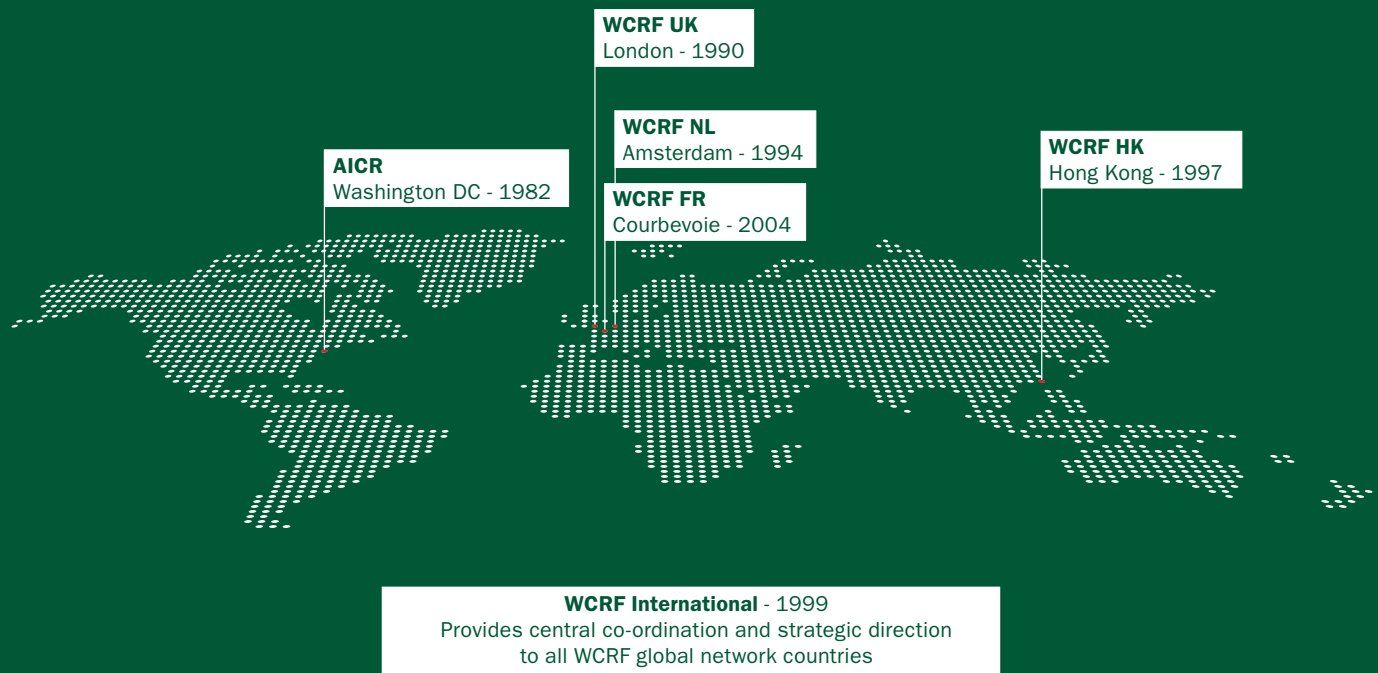
As part of the WCRF International Academy activities, six fellowships were awarded to outstanding applicants for the two-week International Course in Nutritional Epidemiology at Imperial College London, 31 August to 10 September 2010.

AICR Annual Research Conference

The 2010 Annual Research Conference will take place on 21-22 October 2010 in Washington DC. For more information on the AICR annual conference please visit www.aicr.org/conference

Notes

The WCRF global network



American Institute
for Cancer Research

www.aicr.org



World Cancer
Research Fund

www.wcrf-uk.org



Wereld Kanker
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contre le Cancer

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