HOT TOPIC CONFERENCE
OBESITY, PHYSICAL ACTIVITY & CANCER
16 –17th April 2013, London UK
## Contents

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HOST ORGANISATIONS

International Association for the Study of Obesity
IASO’s mission statement is “To improve global health by promoting the understanding of obesity and weight-related diseases through scientific research and dialogue, whilst encouraging the development of effective policies for their prevention and management.”

The International Association for the Study of Obesity (IASO) is a not-for-profit organisation linking over 50 regional and national associations with over 10,000 professional members in scientific, medical and research organisations. It is an umbrella organisation for 53 national obesity associations, representing 55 countries.

Over the last decade, IASO has established itself as a dynamic, professionally managed organisation which has become a ‘nerve centre’ for everyone from governments, professionals and media wanting the latest information on prevalence data and new developments in scientific research into the prevention and management of obesity.

World Cancer Research Fund International
WCRF International is the not-for-profit umbrella association that leads and unifies the WCRF network of cancer charities dedicated to funding research and health information programmes into the link between diet, nutrition, physical activity, body weight and cancer. The national charities are based in the US (American Institute of Cancer Research), the UK (WCRF UK), the Netherlands (Wereld Kanker Onderzoek Fonds/WCRF NL) and Hong Kong (WCRF Hong Kong). The American Institute for Cancer Research (AICR) was founded in Washington, DC in 1982 and works side-by-side with WCRF International to prevent and control cancer through healthy food, nutrition, physical activity and body fatness.

WCRF International plays a leading role in directing and managing the research activities and major scientific projects of the WCRF network. These include the WCRF/AICR Continuous Update Project (CUP), the world’s largest ongoing review of the scientific evidence on the link between diet, physical activity, body weight and cancer. The CUP builds on the success of WCRF/AICR’s 2007 Report: Food, Nutrition, Physical Activity, and the Prevention of Cancer: a Global Perspective.

The WCRF network funds innovative research on the role of diet, nutrition, physical activity and body weight in relation to cancer through the WCRF International Research Grant Programme, based in the UK, and the AICR Research Grant Programme, based in the USA.

In addition, WCRF International works to influence policy changes that make it easier for people to make the kind of healthy choices that can help prevent cancer. Working on behalf of all WCRF network charities, we aim to influence governments and key decision-makers to do all they can to achieve a health-promoting environment for all.
MESSAGE FROM THE HOSTS

Dear Delegate,

It is our great pleasure to welcome you to London for this conference jointly organised by WCRF International and IASO. The conference has brought together leading scientists across the disciplines of cancer, obesity and physical activity and promises to be a highly interactive meeting.

This meeting features an exceptional programme that includes the latest findings in the areas of obesity, physical activity and cancer incidence and survival. It offers excellent networking and collaborative opportunities for researchers in a variety of disciplines, including epidemiology, cell biology, nutrition, biochemistry and cancer prevention.

A main focus of the meeting is to compare and contrast mechanisms underpinning the associations between obesity, physical activity, sedentary behaviour, energy metabolism and cancer development and progression. It will also raise awareness about the value of science and research in informing policy on obesity, physical activity and cancer. All sessions include facilitated discussion to encourage debate with the audience.

The conference venue is in the heart of central London and within walking distance of major tourist attractions including the British Museum, the National Gallery and Covent Garden market.

On behalf of all of us working on this programme we would like to say thank you to all our invited speakers and extend a warm welcome to you all in London.

With best wishes,
SCIENTIFIC COMMITTEE MEMBERS

The Scientific Committee contributed their expertise on the areas of obesity, physical activity and cancer to the development of the conference scientific programme and to the selection of the poster abstracts.

**Tim Byers, MD MPH (Chair)**
CMD Associate Dean for Public Health Practice, Colorado School of Public Health
Associate Director for Cancer Prevention and Control, University of Colorado Cancer Center, USA

**Alan Jackson, CBE MD FRCP FRCPath FRCPCH**
Professor of Human Nutrition and Director, Institute of Human Nutrition, University of Southampton
Director, NIHR Southampton Biomedical Research Centre for Nutrition, Southampton University Hospitals NHS Trust, United Kingdom

**Stephen D. Hursting, PhD MPH**
Professor, Academic Chair and McKean-Love Endowed Chair
Department of Nutritional Sciences, University of Texas at Austin
Professor of Molecular Carcinogenesis, UT-MD Anderson Cancer Center, USA

**Michael F Leitzmann, MD MPH DrPH**
Head, Department of Epidemiology and Preventive Medicine, Regensburg University Medical Center, Germany

**Andrew Renehan, PhD FRCS (Hunterian Professor 2011/2012)**
Senior Lecturer in Cancer Studies and Surgery,
Institute of Cancer Sciences, The University of Manchester
Manchester Academic Health Science Centre,
The Christie NHS Foundation Trust, United Kingdom
GENERAL INFORMATION

Venue/Accommodation
The conference venue is on the ground floor at Charles Darwin House, 12 Roger Street, London WC1N 2JU, United Kingdom. The telephone number is +44 (0)207 685 2400. Detailed travel information can be found on the venue website at www.charlesdarwinhouse.co.uk. Information on nearby hotels can also be obtained from the venue website.

Cloakroom
There is limited space for storing coats and bags at the venue. Items are left at own risk.

Toilets
The toilet facilities, including disabled facilities, are on the ground floor.

Security
The delegate badge must be worn at all times when inside the conference venue.

Smoking
Please refrain from smoking anywhere on the premises, including the venue forecourt.

Lunch/Refreshments/Drinks reception
Lunch will be provided both days at 13:00pm in the breakout area. There will also be two refreshment breaks each day. Please refer to the programme for the break times. There will be a drinks reception at 18:00pm on Tuesday 16th for all delegates (breakout area).

Wireless network
A wireless network is available. The password is time2work.

Posters
The conference posters will be displayed in the breakout area and in the lecture theatre.

AV Recording
Some sessions will be video-recorded and photographed. Permission to record and photograph the audience will be assumed. A cameraman, accompanied by a member of WCRF International staff, will also be recording scheduled interviews with speakers throughout the conference. Some of the photographs, video recordings and speaker slides will be available at a later stage on the WCRF International website, and may also be used in future promotional and communications material.

Twitter
The conference Twitter hashtag is #OPAC2013.

WCRF International Online Blog
The WCRF International blog will contain information on some of the conference topics. Visit http://www.wcrf.org/blog/

LEARNING OBJECTIVES

◆ Learn about the latest research advances in the fields of obesity, physical activity and cancer and stimulate discussion about future research directions in this field
◆ Compare and contrast mechanisms underpinning the associations between obesity, physical activity, sedentary behaviour, energy metabolism and cancer development and progression
◆ Explore the impact of body fatness, physical activity and sedentary behaviour in the management and outcome of diagnosed cancer
◆ Raise awareness about the value of science and research in informing policy on cancer

Certificate of Attendance
Upon having attended the Hot Topic Conference Obesity, physical activity and cancer, delegates will receive a certificate of attendance. Certificates can be picked up at the end of the conference. This event is accredited for 4 SCOPE points and 12 CPD points.

More information on SCOPE can be found at the end of this abstract book and online at www.iaso.org/scope

SCOPE
SPECIALIST CERTIFICATION OF OBESITY
PROFESSIONAL EDUCATION
FLOOR PLAN

Presentations will be delivered in the Charles Darwin Lecture Theatre. Lunch and refreshments will be provided in the Breakout Area. The Chatham and James side rooms will be used for interview filming.
**SCHEDULE: Tuesday 16th April**

<table>
<thead>
<tr>
<th>Time</th>
<th>Event</th>
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<tbody>
<tr>
<td>08:00</td>
<td>Registration</td>
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<tr>
<td>09:15</td>
<td>Welcome, introductions and aims</td>
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<td>Chair and IASO/WCRF International</td>
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<tr>
<td>09:30</td>
<td><strong>Session 1: Obesity, physical activity and cancer prevention - Keynote talks</strong></td>
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<td>Chair: Alan Jackson</td>
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<td></td>
<td><strong>Topic 1:</strong> Population science and clinical research - <em>Tim Byers</em></td>
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<td><strong>Topic 2:</strong> Mechanistic insights from basic and preclinical research - <em>Steve Hursting</em></td>
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<td>Facilitated discussion between speakers for all sessions and audience</td>
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<td>11:00</td>
<td><strong>Refreshments</strong></td>
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<tr>
<td>11:30</td>
<td><strong>Session 2: Evidence for the relationship between obesity and cancer incidence and survival</strong></td>
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<td>Chair: Tim Byers</td>
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<td></td>
<td><strong>Topic 1:</strong> Appraisal of evidence for obesity effects - <em>Anne McTiernan</em></td>
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<td><strong>Topic 2:</strong> Racial and ethnic differences in measures and effects of obesity - <em>TH Lam</em></td>
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<td><strong>Topic 3:</strong> Weight loss and cancer risk reduction - <em>Michelle Harvie</em></td>
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<td>Facilitated discussion between speakers for all sessions and audience</td>
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<tr>
<td>13:00</td>
<td><strong>Lunch</strong></td>
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<td>14:00</td>
<td><strong>Session 3: Evidence for the relationship between physical activity and cancer incidence and survival</strong></td>
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<td>Chair: Tim Byers</td>
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<td><strong>Topic 1:</strong> Appraisal of evidence for physical activity effects - <em>Kate Wolin</em></td>
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<td><strong>Topic 2:</strong> Measurement challenges for the various dimensions of physical activity - <em>Ulf Ekelund</em></td>
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<td><strong>Topic 3:</strong> Physical activity change: what biomarkers tell us about cancer mechanisms and effects - <em>Cornelia Ulrich</em></td>
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<td>Facilitated discussion between speakers for all sessions and audience</td>
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<td>15:30</td>
<td><strong>Refreshments</strong></td>
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<td>16:00</td>
<td><strong>Session 4: Energy balance, host related factors and the cancer processes in basic and preclinical services</strong></td>
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<td>Chair: Steve Hursting</td>
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<td><strong>Topic 1:</strong> Energy metabolism within organisms and cells and cancer processes - <em>Henry Thompson</em></td>
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<td><strong>Topic 2:</strong> Obesity, inflammation, and cancer - <em>Catherine Muller-Staumont</em></td>
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<td>Facilitated discussion between speakers for all sessions and audience</td>
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<td>17:10</td>
<td><strong>Session 5: Continuous Update Project (CUP) new developments</strong></td>
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<td>Chair: Steve Hursting</td>
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<td><strong>Topic:</strong> Continuous Update Project RFA: Systematic review of mechanistic literature - <em>Martin Wiseman and Sarah Lewis</em></td>
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<td>Facilitated discussion between speakers for all sessions and audience</td>
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<td>17:50</td>
<td><strong>Finish</strong></td>
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<tr>
<td>18:00</td>
<td><strong>Drinks Reception</strong></td>
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09:00  **Summary of Day 1/Overview of Day 2**  
Tim Byers and Steve Hursting

09:30  **Session 6: Obesity, physical activity and specific cancers (incidence and survival)**  
Chair: Alan Jackson  
**Topic 1:** Energy balance, adiposity, physical activity, and colorectal cancer - Andrew Renehan and Jenifer Fenton  
Facilitated discussion between speakers and audience

10:30  **Refreshments**

11:00  **Session 6 (ctd): Obesity, physical activity and specific cancers (incidence and survival)**  
Chair: Andrew Renehan  
**Topic 2:** Energy balance, adiposity, physical activity, and pancreatic cancer - Rudolf Kaaks and Steve Hursting  
Facilitated discussion between speakers and audience  
**Topic 3:** Energy balance, adiposity, physical activity, and breast cancer - Cheryl Rock and Margo Cleary  
Facilitated discussion between speakers and audience (20 minutes)

13:00  **Lunch**

14:00  **Session 7: Life course factors and cancer (incidence and survival)**  
Chair: Andrew Renehan  
**Topic 1:** Overview of life course factors and cancer - Alan Jackson  
**Topic 2:** Developmental origins of obesity and cancer risk - Susan Ozanne  
**Topic 3:** Epigenetic mechanisms linking early life diet and cancer risk - Karen Lillycrop  
Facilitated discussion between speakers for all sessions and audience

15:45  **Refreshments**

16:15  **Session 8: Closing session**  
Chair: Tim Byers  
**Topic:** Policy on obesity and physical activity: how cancer fits into chronic disease agenda and why - João Breda  
**Panel discussion:** Scientific committee comments on key priorities and opportunities for cancer research and prevention and closing remarks by the Chair

17:30  **Finish**
The speaker biographies are listed in alphabetical order.

**Dr João Breda**

Physical Activity and Obesity, Noncommunicable Diseases and Health Promotion Division, WHO Regional Office for Europe, Denmark

**Title: Policy on obesity and physical activity: how cancer fits into chronic disease agenda and why**

Dr Breda is the programme manager for the Nutrition, Physical Activity and Obesity Noncommunicable diseases and Health Promotion Division at WHO Regional Office for Europe. He is responsible for providing support to the 53 Member States of the WHO European Region on the implementation of the European Charter on Counteracting Obesity and evaluating its progress. His team is responsible for the largest and most comprehensive childhood obesity surveillance mechanisms globally.

He was the Portuguese representative in the WHO-Europe for the area of Nutrition and Physical Activity and in the European Union and also the focal point from Portugal in the European Network on Nutrition and Physical Activity, at the High Level Group on Nutrition & PA and the European Platform on Diet, Nutrition and Physical Activity of the EU.

Dr Breda was the first and former Coordinator of the National Platform against Obesity under the Portuguese Ministry of Health. He worked as a Public Health Nutritionist at the General Health Directorate in the Portuguese Ministry of Health. He has published in scientific journals and presented in national and international congresses, several dozens of papers and also published 14 original books.

**Professor Tim Byers**

Colorado School of Public Health, Associate Director for Cancer Prevention and Control, University of Colorado Cancer Center, USA

**Title: Population science and clinical research**

Since 1995 Tim Byers has held the position of Professor at the University of Colorado. He has served as the Deputy Director and as the Interim Director of the University of Colorado Comprehensive Cancer Center and is now the Associate Director for Cancer Prevention and Control. He is also the Associate Dean for Public Health Practice at the Colorado School of Public Health. He is a member of the Board of Directors of the American Cancer Society, and now serves as the National President Elect. Prior to 1995 he was Chief of the Chronic Disease Prevention Branch of the Nutrition Division at the Centers for Disease Control and Prevention in Atlanta.

Dr. Byers is an expert in cancer prevention research. He has worked in many settings in clinical medicine, public health, and academic medicine. He has a particular interest in epidemiologic studies of the role of early detection, diet, and nutrition in the prevention of cancer, and in the application of disease prevention in community settings. He has published over 350 papers in peer-reviewed scientific journals. His current research includes epidemiologic and clinical studies of nutrients as protective factors in colon, breast, and lung cancer, studies of cancer treatment decision-making by patients and physicians, studies of cancer genetics, and studies and programs to promote the early detection of lung and colorectal cancer. He is now the Director of the Center for Public Health Practice at the Colorado School of Public Health, developing training and programs to improve the effectiveness of public health workers, agencies, and programs.
SPEAKERS

Professor Margot Cleary
Hormel Institute, University of Minnesota, Austin, USA
Title: Preclinical Insights into the Relationship of Obesity and Breast Cancer

Margot P. Cleary is a professor at the Hormel Institute, at the University of Minnesota. She received her doctoral degree from Columbia University. Her early studies focused on adipose tissue development and the impact of interventions on the prevention/treatment of obesity. Presently, her laboratory’s research efforts are on the impact of obesity and/or calorie restriction on the development of mammary cancer. Effects of metformin treatment on mammary tumor development in both lean and obese mice are being investigated. Complementary in vitro studies have also been done. Overall results of her studies suggest that changes in two adipose tissue produced proteins, leptin and adiponectin, and their ratio and also IGF-I play import roles on how body weight affects tumorigenesis.

Dr Ulf Ekelund
Department of Sport Medicine, Norwegian School of Sport Sciences, Oslo, Norway and MRC Epidemiology Unit, Cambridge, UK
Title: Measurement Challenges for Various Dimensions of Physical Activity

Ulf Ekelund is professor of physical activity epidemiology at the Norwegian School of Sport Sciences, Oslo, Norway and investigator scientist at the MRC Epidemiology Unit, Cambridge, UK. He obtained his PhD in 2002 at the Karolinska Institutet, Sweden with post-graduate training in epidemiology at the University of Cambridge.

His research interests are broadly divided into three areas;

- Developing precise methods to assess physical activity in populations
- Understanding the dose-response associations between physical activity and sedentary behaviour with metabolic and chronic diseases across the life span
- Understanding the biological determinants of physical activity and how these variables may modify the associations between physical activity and chronic diseases.

Ulf has published more than 200 original research papers, review articles, and book chapters, and serves on the editorial board for four international journals. He was awarded the new investigator scientist award of the American College of Sports Medicine in 2007.
**Dr Jenifer Fenton**  
Department of Food Science and Human Nutrition, Michigan State University, USA  
**Title: Energy balance, adiposity, physical activity, and colorectal cancer**

Jenifer Fenton is an Assistant Professor in the Department of Food Science and Human Nutrition at Michigan State University. She earned her PhD in Nutrition from MSU and a MPH in Epidemiology from University of Michigan. She completed postdoctoral training in cancer prevention at the National Cancer Institute. Dr. Fenton utilizes in vitro cell culture models, mouse models and human clinical studies to understand how proinflammatory cytokines associated with metabolic obesity increase colon cancer risk, with emphasis on leptin and IL-6 and Nf-kapaB and STAT signaling pathways. She also has expertise on how diet influences host immune response to bacterially-induced colitis and colon cancer progression.

**Dr Michelle Harvie**  
Nightingale and Genesis Prevention Centre, Wythenshawe Hospital, Manchester, United Kingdom  
**Title: Weight loss and cancer risk reduction**

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. Her current research covers optimum diet and exercise strategies to prevent breast cancer and its recurrence. She has over 30 peer reviewed publications and was awarded the British Dietetic Association Rose Simmond’s Award 2005 for her paper on weight gain amongst breast cancer patients receiving adjuvant chemotherapy. Most recently she received the Association Study Obesity National Best practice award 2011 for her work on intermittent energy restricted diets.
**Professor Stephen D. Hursting**  
Department of Nutritional Sciences, The University of Texas at Austin and Department of Molecular Carcinogenesis, the University of Texas MD Anderson Cancer Center  

**Title 1: Mechanistic insights from basic and preclinical research**  
**Title 2: Energy balance, adiposity, physical activity, and pancreatic cancer**  

Stephen D. Hursting is Professor and Chair of the Department of Nutritional Sciences at the University of Texas at Austin. He is also the McKean-Love Chair of Nutrition, Molecular and Cellular Sciences at the University of Texas and is Professor of Molecular Carcinogenesis at the UT-MD Anderson Cancer Center. Dr. Hursting earned his PhD in nutritional biochemistry and MPH in nutritional epidemiology from the University of North Carolina, and he completed postdoctoral training in molecular carcinogenesis and cancer prevention at the NCI. Prior to joining the University of Texas in 2005, Dr. Hursting was Deputy Director of the NCI’s Office of Preventive Oncology and Chief of the NCI’s Nutrition and Molecular Carcinogenesis Laboratory Section. His research, which has resulted in over 150 publications, centers on diet-gene interactions relevant to cancer prevention, particularly the molecular, metabolic and inflammatory mechanisms underlying obesity-cancer associations.

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**Professor Alan Jackson**  
Institute of Human Nutrition, University of Southampton and Director, NIHR Southampton Biomedical Research Centre for Nutrition, Southampton University Hospitals NHS Trust, United Kingdom  

**Title: Overview of life course factors and cancer**  

Professor Jackson was appointed Professor of Human Nutrition in the University of Southampton in 1985; the first Chair in the discipline in an undergraduate medical school in the UK. He is Director of the National Institute of Health Research Southampton Biomedical Centre in Nutrition. 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 was foundation Chair of the Scientific Advisory Committee on Nutrition to the Departments of Health and Food Standards Agency and a member of the nutrition panel of the European Food Safety Authority. 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, and how to take nutrition interventions that are known to be efficacious to scale effectively.

He is currently President of the Association for Nutrition, a Council Member of the Caribbean Health Research Council, convenor for the International Malnutrition Task Force and Chairman for the Continuous Update Panel for the evidence linking diet, nutrition and physical activity to cancer for the World Cancer Research Fund/American Institute for Cancer Research.
**Professor Rudolf Kaaks**  
Deutsches Krebsforschungszentrum (DKFZ), Division of Cancer Epidemiology, Heidelberg, Germany  
**Title: Energy balance, adiposity, physical activity, and pancreatic cancer**  

Rudolf Kaaks received his Master of Science in Human Nutrition & Epidemiology at the University of Wageningen in 1987 and his PhD in Nutritional Epidemiology in 1994. Within his scientific career he worked as a junior researcher at the Utrecht University (1987-1988), as Epidemiologist at IARC, Lyon (1998-2001) and in 2001 became head of the Hormones and Cancer Group at this institution. In 2006 Rudolf Kaaks was appointed Professor (Chair, Cancer Epidemiology) at the University of Heidelberg and head of the Division of Cancer Epidemiology at the German Cancer Research Center (DKFZ), Heidelberg, Germany.

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**Dr Sarah Lewis**  
School of Social and Community Medicine, University of Bristol, UK  
**Title: Continuous Update Project RFA. Developing a template to carry out systematic reviews of mechanistic studies of diet and cancer**  

Dr Sarah Lewis is a Senior Lecturer in Genetic Epidemiology, School of Social and Community Medicine, University of Bristol. Her main area of research is genetic epidemiology and in particular using genetic variants involved in the uptake, metabolism and transport of nutrients to elucidate pathways between nutrition and disease, with a focus on prostate cancer as well as other outcomes. She has experience in conducting systematic reviews of genetic association studies.
**Dr Karen A Lillycrop**  
Institute of Developmental Sciences, University of Southampton, United Kingdom  
**Title: Epigenetic mechanisms linking early life diet and cancer risk**

Dr Karen Lillycrop is a Reader in Developmental Epigenetics at the Institute of Developmental Sciences at the University of Southampton. Karen obtained her first degree in Chemistry and Biochemistry at Imperial College, London followed by her doctorate in Biochemistry at the University of Leicester. Karen’s current research is focused on how early life environment influences the epigenome and the development of human disease. She was the first to demonstrate that maternal diet can alter the epigenetic regulation of key transcription factors within the fetus. Her group is funded by the WCRF, BBSRC and EU and the Epigen Research Consortium.

**Professor Anne McTiernan**  
Fred Hutchinson Cancer Research Center, USA  
**Title: Appraisal of evidence for obesity effects**

Anne McTiernan, MD, PhD is a Full Member/Professor at the Fred Hutchinson Cancer Research Center and the University of Washington in Seattle, USA. She is Principal Investigator of several clinical trial and cohort studies of obesity, exercise, chemoprevention, and cancer risk and prognosis. Anne McTiernan is a Komen Scholar and received a 2012 American College of Sports Medicine Citation Award. She has published over 350 scientific manuscripts and written or edited several books. She has advised the International Agency for Research on Cancer, the American Cancer Society, the U.S. Department of Health and Human Services Physical Activity Guidelines Advisory Committee, and the World Cancer Research Fund.

**Professor Catherine Muller-Staumont**  
Institute of Pharmacology and Structural Biology/University of Toulouse, Toulouse, France  
**Title: Obesity, inflammation and cancer**

Catherine Muller-Staumont (MD, PhD) is full-time professor at the University of Toulouse. She is the Head of the Cancer Biology Department of the Institute of Pharmacology and Structural Biology in Toulouse and the group leader of the “Microenvironment, Cancer and Adipocytes” group.

Her work is focused on the paracrine role of adipose tissue in cancer progression in both lean and obese conditions.
Dr Susan Ozanne
Institute of Metabolic Science (IMS), University of Cambridge Metabolic Research Laboratories, United Kingdom

Title: Developmental Origins of Obesity and Cancer Risk

Dr Ozanne is a Reader in Developmental Endocrinology and BHF Senior Fellow at the University of Cambridge as well as a Fellow of Churchill College. She obtained a first class honours degree in Biochemistry from the University of Edinburgh (1990) and her PhD (1994) from the University of Cambridge. Her research is focused on understanding the mechanistic basis of the relationship between suboptimal early nutrition and health in later life. She has authored over 100 papers on the early origins of health and disease and is a council member of the Society for the Developmental Origins of Health and Disease.

Dr Andrew G Renehan
Institute of Cancer Sciences, University of Manchester, United Kingdom

Title: Energy balance, adiposity, physical activity, and colorectal cancer: clinical perspectives

Dr Andrew Renehan is a senior lecturer in cancer studies and surgery at the University of Manchester, and is honorary consultant at the Christie NHS Foundation Trust, Manchester. His PhD was on “Insulin-like growth factors (IGFs) and their binding proteins in colorectal carcinogenesis” undertaken at the Paterson Institute for Cancer Research, Manchester.

He is currently the lead for the ManDOC (Manchester Diabetes, Obesity and Cancer) project covering a number of related themes:

- Complex modelling of anthropometric measures with time
- Associations between anthropometric measures and colorectal cancer
- Influence of body mass index at cancer diagnosis on survival
- Inter-relationship between BMI, diabetes therapies and cancer risk
- Cancer screening utilisation in patients with type 2 diabetes
- Exploration of novel measures of excess adiposity (proton MR spectroscopy) and cancer risk and progression

He is a fellow of the Royal College of Surgeons of England; held the British Travelling Fellowship (for Coloproctology) for 2009; and is the Hunterian Professor for 2011/2012. He is chair of the international Diabetes and Cancer Research Consortium.
Professor Cheryl L. Rock
Department of Family and Preventive Medicine, School of Medicine, University of California, USA
Title: Energy Balance, Adiposity, Physical Activity, and Breast Cancer

Cheryl Rock, PhD, RD, is a professor in the School of Medicine, University of California, San Diego. She completed undergraduate training in nutrition and dietetics at Michigan State University, and achieved a Masters degree in clinical nutrition at Emory University and a PhD in nutritional sciences at UCLA. Her research is focused on the role of nutritional factors in the development and progression of cancer, and healthy weight management. Dr. Rock has served on numerous scientific review panels and editorial boards for peer-reviewed journals. To date, Dr Rock is the author of more than 235 scientific papers and book chapters.

Professor Tai Hing Lam
Department School of Public Health, Department of Community Medicine University of Hong Kong, Hong Kong, China
Title: Racial and ethnic differences in measures and effects of obesity

Professor Lam has been Chair Professor and Head of the Department of Community Medicine at the University of Hong Kong since 2000, Director of the Public Health Research Centre since 2004, and Director of School of Public Health since 2009.

He 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. He was President of the Hong Kong College of Community Medicine from 1997 to 2001 and is currently Vice-Chairman of the Hong Kong Council on Smoking and Health and Co-Chair of the Grant Review Board, Research Council, Health, Welfare and Food Bureau of the Hong Kong SAR Government.

Professor Lam’s research interests include 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 Organisation in, the China Tobacco Control Award in, a University Teaching Fellowship 1999-2000, Outstanding Research Student Supervisor Award 2000-01, and Outstanding Researcher Award 2001-02 by the University of Hong Kong.
**Professor Henry J. Thompson**  
Cancer Prevention Laboratory, Colorado State University, USA

**Title: Energy metabolism within organisms and cells and cancer processes**

Henry J. Thompson, Ph.D is professor in the College of Agricultural Sciences and director of the Cancer Prevention Laboratory at Colorado State University in Fort Collins, Colorado. Thompson is a member of the American Association for Cancer Research and the American Society for Nutritional Sciences. Dr. Thompson has published more than 165 journal articles and book chapters. Thompson has a long standing interest in the prevention of, and prognosis in, breast cancer and he maintains an active program of clinical and laboratory research that addresses this topic. Since 1988, Thompson’s laboratory has been investigating the mechanisms underlying the cancer inhibitory activity of energy restriction and exercise. The focus of these studies using preclinical models has been on identifying the cellular processes, molecular machinery, and chemical mediators by which these interventions regulate tissue size homeostasis and inhibit the carcinogenic process. Currently, his laboratory is investigating the effects of energy restriction mimetic agents such as 2-deoxyglucose and metformin on cancer initiated cell deletion from the breast. Since 1993, Henry Thompson has been working with a team of medical oncologists specializing in breast cancer in order to translate his preclinical findings into effective clinical weight control interventions.

The American Institute for Cancer Research (AICR) kindly sponsored Professor Henry Thompson’s attendance at the conference. For more information on AICR, please visit: www.aicr.org

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**Professor Cornelia Ulrich**  
National Center for Tumor Diseases (NCT) Heidelberg, Germany

**Title: Physical activity change: what biomarkers tell us about cancer mechanisms and effects**

Cornelia (Neli) Ulrich is since 2009 Head of the Department of Preventive Oncology at the German Cancer Research Center and Director of the National Center for Tumor Diseases (NCT) in Heidelberg, Germany. She holds a Master’s degree in Nutrition and PhD in Epidemiology. From 1999 to 2012, she was Professor at the Fred Hutchinson Cancer Research Center and University of Washington in Seattle. Professor Ulrich has authored more than 200 publications and is an elected member to the European Academy of Cancer Sciences. She serves on many national and international advisory boards, committees, and editorial boards. Cornelia Ulrich is an expert in cancer prevention and epidemiology. She is best known for her studies on the molecular epidemiology of colorectal cancer, with emphasis on the biologic pathways related to one-carbon metabolism, inflammation and non-steroidal anti-inflammatory drugs (NSAIDs). Additional research includes pharmacogenetic, prognostic, and cancer survivorship studies, including leadership of the international ColoCare Consortium, a colorectal cancer patient cohort. Finally, Professor Ulrich oversees the area ‘Exercise and Cancer’ in Heidelberg and investigates links between obesity, physical activity, cancer risk and clinical outcomes.
**Professor Martin Wiseman**  
World Cancer Research Fund International, United Kingdom  

**Title: Continuous Update Project RFA. Developing a template to carry out systematic reviews of mechanistic studies of diet and cancer**

Professor Martin Wiseman is Medical and Scientific Adviser with 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 its companion report, Policy and Action for Cancer Prevention, published in 2009. In addition, he is a Visiting Professor in human nutrition at Southampton University.

**Dr Kathleen Y. Wolin**  
Stritch School of Medicine, Loyola University Chicago, USA  

**Title: Appraisal of evidence for physical activity effects**

Dr. Kate Wolin is a behavioural epidemiologist whose research focuses on the role of lifestyle in cancer prevention and control. She joined the faculty as an Associate Professor at Loyola University Chicago in April 2013. Her current research investigates the role of physical activity and obesity in cancer etiology among high-risk individuals and in cancer treatment sequelae. Prior to leaving Washington University in St Louis this spring, Dr. Wolin was the PI of a study of bone health outcomes among breast cancer survivors engaging in weight loss, the PI of an observational study of energy balance and post-prostatectomy urinary and sexual function and the site PI of a vanguard trial of weight loss and quality of life and disease free survival in breast cancer. She also researches the implementation of cancer prevention and control knowledge in cancer survivorship care. Dr. Wolin blogs at drkatewolin.com and can be found on Twitter @drkatewolin. Dr. Wolin has a BA in anthropology from Tufts University and a ScD in epidemiology from the Harvard School of Public Health where she was an NCI-funded Cancer Prevention and Epidemiology fellow. She completed her postdoctoral training as an NCI-funded fellow in cancer epidemiology at Northwestern University. Dr. Wolin is a fellow in the American College of Sports Medicine.

*The American Institute for Cancer Research (ACIR) kindly sponsored Professor Kathleen Wolin’s attendance at the conference. For more information on ACIR, please visit: www.aicr.org*
Title: Population science and clinical research

Speaker: Tim Byers

Although the concept that nutrition might influence cancer risk is centuries old, direct evidence for this relationship has emerged in only the past 50 years. Early ecological and case-control studies suggested very strong relationships, but in the past 25 years substantially greater evidence, in both quality and quantity, has firm ed-up our understandings of the relative magnitude of the various nutritional factors on cancer risk. Although specific types of foods do influence cancer risk, it is the balance between the total amount of food consumed and physical activity that has the greatest influence on cancer risk. Obesity and physical inactivity are now pandemic problems that are increasing risk for many different types of cancer. This keynote talk will discuss the current status of both observational research and clinical trials studying the relationship between nutrition and cancer across the world.

Title: Obesity, energy balance and cancer prevention: Mechanistic insights from transdisciplinary studies

Speaker: Stephen D. Hursting

The prevalence of obesity, an established risk factor for many cancers, has risen steadily for the past several decades in the US and many other countries. Unfortunately, the mechanisms underlying the obesity and cancer connection are not well understood, and new targets and strategies for offsetting the impact of obesity on cancer risk and/or progression are urgently needed. We have established that calorie restriction (CR), the most commonly recommended dietary strategy for preventing or reversing obesity, inhibits tumor development and progression in a variety of animal models. In contrast, diet-induced obesity enhances tumorigenesis in many of these same models. We have shown in a series of transgenic model systems and microarray studies that the insulin/insulin-like growth factor (IGF)-1 pathway appears central to many of the anti-cancer effects of CR and pro-cancer effects of obesity. Using AZIP/F1 transgenic mice (which lack white adipose tissue but have high levels of insulin and IGF-1), and liver-specific IGF-1-deficient mice, we have reported that elevated IGF-1/insulin resistance/inflammation (which typically accompany obesity), independent of the adipose tissue per se, appear to be the important targets for disrupting the obesity-cancer link. Also, genetic and pharmacologic approaches suggest the Akt/mammalian target of rapamycin (mTOR) pathway (downstream of insulin and IGF-1 receptors) provides an important target for disrupting the obesity-cancer link. A better understanding of the mechanisms underlying the energy balance-cancer link will facilitate the development of novel prevention and treatment strategies for offsetting the effects of obesity on cancer.

Title: Appraisal of evidence for obesity effects

Speaker: Anne McTiernan

The International Agency for Research on Cancer estimates that 25% of cancer cases worldwide are due to overweight/obesity and a sedentary lifestyle, and a United States Institute of Medicine workshop recently concluded that obesity at diagnosis is a poor prognostic factor for several types of cancer. This talk will review human data on the associations of overweight and obesity with cancer incidence, survival, and potential mechanisms. Overweight and obesity (body mass index > 25.0 kg/m2) increase risk for several cancers including colon, postmenopausal breast, endometrium, lower esophagus, kidney, liver, and other cancers. Overweight or obesity has been associated with reduced survival in persons with breast, colon, prostate, and other cancers, and with increased risk for co-morbidities. Overweight and obesity may affect cancer risk and prognosis by several mechanisms including increased amount and availability of sex hormones, insulin, other adipokines, and inflammation. Randomised clinical trial data have found significant reductions in estrogens, insulin and inflammation, and changes in adipokines with weight loss with or without exercise. Few studies have assessed weight loss effects on target tissue markers, however. To determine the cancer-relevant effects of reductions in adiposity, human intervention studies are vital, because animal models are not always applicable to humans, and because it is difficult for epidemiologic observational studies to determine effects of significant change in adiposity. Studies are needed on cancer risk and prognosis biomarker effects of various methods of weight loss in persons with and without cancer, including behavior change, specific weight-loss diets, medications, and bariatric surgery.
Title: Racial and ethnic differences in measures and effects of obesity
Speaker: Professor Tai Hing Lam
Epidemiologically, obesity is classified into general and central obesity. Body mass index (BMI) is commonly used because data on central obesity are scarce. For the same BMI, Asians have 3-5 percentage points higher in body fat and larger waist circumference (WC) than Caucasians. Although obesity and its indices are all continuous variables, cutoff points are needed to define overweight and obesity. The cutoff levels are lower for Asians but % body fat/BMI ratio varies within Asians, Caucasians and the same ethnic group (e.g. Chinese in different regions).

Obesity increases the risks of cardiovascular diseases, type II diabetes and some cancers. Most of the evidence has come from Caucasian populations. Studies, especially prospective studies, designed to examine ethnic differences are scarce. Evidence predominantly based on BMI and western populations cannot show clear regional or ethnic differences for colorectal, breast and prostate cancer.

Measures of central obesity (WC and WHR) may be more strongly associated with type II diabetes than BMI, but the relationships for hypertension and dyslipidaemia were similar. The relationships of BMI, WC and WHR with cardiovascular outcomes are broadly similar. The differences are unlikely to be of clinical and public health significance. The Asia Pacific Cohort Studies Collaboration (APCSC), a meta-analysis based on individual data, showed no significant regional (Asian and Australasian) differences. But regional differences are not racial/ethnic differences.

Baseline indices may not reflect lifelong obesity status. Changes of body weight/fat before baseline and during follow up can have major impacts on disease burden and mortality. The growing obesity epidemic in the West has started a few decades ago and may take a few more decades to show its full impacts. In the East, the epidemic is at an earlier stage. Results on racial/ethnic/regional differences must be interpreted cautiously.

Title: Weight loss and cancer risk reduction
Speaker: Michelle Harvie
Energy restriction and weight loss decrease development and progression of breast and other common cancers. This was first shown in animal models over 100 years ago and more recently within observational studies. This presentation will summarise the evidence for weight loss and cancer incidence and survival. It will address key questions of 1. Who we should target with weight control for prevention and after diagnosis? When should we intervene? What are optimum weight loss interventions, i.e. the optimum composition of weight loss diets or the use of intermittent or continuous patterns of energy restriction? Weight control and energy restriction has a number of potential cancer protective mechanisms which require further study. The presentation will discuss key mechanistic pathways, which opens the possibility of using energy restriction mimetics for cancer prevention.

Title: Appraisal of evidence for physical activity effects
Speaker: Kathleen Y. Wolin
Data consistently shows individuals who are more active are less likely to be diagnosed with, and die from, numerous forms of cancer. Most of this research has focused on aerobic activity, but evidence is emerging for the benefits of resistance training and the risk of sedentary time, independent of aerobic physical activity participation.

Over 50 studies of physical activity and colon cancer have been published and most found a 20-25% lower risk among the most versus least active. Convincing evidence also shows that physical activity reduces the risk of breast cancer by about 25%. Support for the benefits of physical activity in prostate cancer are less strong, but suggests a likely benefit, which may be limited to participation in vigorous physical activity for reducing the risk of lethal prostate cancer. Evidence is also growing for an inverse association of physical activity and other cancers. Physical activity has also been associated with decreased risk of cancer mortality in colon, breast and prostate cancers.

Healthy lifestyle behaviors do cluster together, but physical activity is not merely a marker of healthier lifestyle, it exerts an independent protective effect. Many of the studies that report an association between physical activity and cancer have controlled for other lifestyle factors (e.g., body weight, alcohol intake, diet) that may be associated with physical activity. Studies have also examined whether obesity modifies the effect of physical activity and found the activity benefit is present in all BMI classes. Evidence for the harmful effects of sedentary behavior, independent of physical activity, is limited, but growing. The equivocal findings may result from the challenges of accurately measuring sedentary time.
Title: Measurement challenges for various dimensions of physical activity

Speaker: Ulf Ekelund

Physical activity is a complex and multidimensional behaviour including the type, frequency, intensity, and duration of physical activity and the context in which physical activity is performed. Physical activity is clearly distinguished from exercise which is a sub-dimension of overall physical activity usually aimed at enhancing or maintaining components of physical fitness. Physical activity can broadly take place in four different domains; occupation, transportation, leisure time and domestic activity.

Measuring the multiple dimensions of physical activity requires different measurement methods, and in epidemiological settings, the choice of method is usually a trade-off between precision and feasibility. Until recently, physical activity measures in population based studies have been obtained by self-report methods such as questionnaires. Although they are prone to bias and error when estimating the intensity and duration of activity, these instruments may categorise and rank individuals reasonably well.

In contrast, objective assessment methods, e.g. accelerometry, are often more precise in determining the amount of time spent sedentary and in different intensity levels and when estimating physical activity energy expenditure. Increased precision obtained by objective methods will enhance our understanding of; the detailed dose-response associations between activity and health outcomes; time trends in activity levels; the effect of interventions; and the effect of habitual physical activity in exercise training trials. Further, a more precise assessment of activity will reduce sample size regardless whether activity is measured as the exposure or outcome.

No single method is currently available that can assess all different dimensions of PA. Therefore, a combination of methods is preferable.

Title: Physical activity change: what biomarkers tell us about cancer mechanisms and effects

Speaker: Professor Cornelia Ulrich

Both obesity and sedentary behavior have been linked to an increased risk of various cancer types and initial data suggest also that these components of energy balance can also positively impact survival among cancer patients. There are a variety of biological mechanisms that can link energy balance to cancer risk and prognosis, many of which are now well supported both by observational studies and randomized controlled trials with biomarker endpoints. This talk will give an overview of biologic mechanisms and highlight some key findings that help discern the role of weight loss versus exercise in affecting cancer risk. Major emphasis will be on inflammation, immune function and recently published work on adipose-tissue gene expression. Other mechanisms, such as effects on sex-steroid hormones, the insulin/IGF axis, DNA repair function, vitamin D and the gut microbiome will also be summarised briefly. An outlook will highlight new research of exercise training among cancer patients and the challenges in measuring biomarkers in that setting.

Title: Energy metabolism within organisms and cells and cancer processes

Speaker: Henry Thompson

While strong evidence from animal models indicates that limiting energy availability in the presence of adequate nutrition is protective against cancer at many organ sites, emerging findings indicate that limiting specific energy sources may exert additional protective effects, with a reduction in the accumulation of gain of function mutants of p53 by glucose restriction serving as a provocative example of new insights. This presentation will summarize the cellular and molecular data linking energy availability to the development of cancer. Specific consideration will be given not only to effects on cell proliferation and cell death via apoptosis, but also to the intracellular energy messenger/energy sensor detection system and its connection to the signalling network in which AMP-activated protein kinase (AMPK), mammalian target of rapamycin (mTOR), and protein kinase B (Akt) are key regulatory components. The role of histone deacetylases such as sirtuins and of autophagy will also be considered from the viewpoint of whether they enhance or diminish the protective effects of energy restriction against cancer. The effects of candidate energy restriction mimetic agents such as dichloroacetate and biguanides that target mitochondrial energy metabolism will be presented. The importance of matching animal model selection to the question being addressed will be considered and both challenges and opportunities for future research will be highlighted.
Title: Obesity, inflammation and cancer
Speaker: Catherine Muller-Staumont

Obesity is associated with an increased incidence of cancer and is also an independent negative prognosis factor for cancers such as breast and prostate. While investigating the paracrine role of adipose tissue (AT) in breast cancer progression, we have found both in vitro and in vivo that: i) invasive cancer cells impact dramatically on surrounding adipocytes; ii) peri-tumoural adipocytes exhibit a modified phenotype and specific biological features. We therefore named these adipocytes “Cancer-Associated Adipocytes (CAAs)”; iii) CAAs promote the aggressive behaviour of cancer cells, by secreting pro-inflammatory cytokines and by modulating cancer cell metabolism. In obese conditions, CAAs and adipocytes share several common traits including inflammation. Therefore, it is tempting to speculate that the deleterious crosstalk between mature adipocytes and breast cancer is amplified in obese conditions and contributes, at least in part, to the poor prognosis of breast cancer observed in obese patients. This hypothesis is reinforced by recent data obtained in humans and animals suggesting that mammary adipose tissue exhibits, much like visceral adipose tissue, a sub-inflammatory state in obesity. Increased inflammation due to the modification of local surrounding AT has also been proposed in the case of prostate cancer. Finally, chronic inflammation observed in obese conditions could also contribute to cancer progression via endocrine mechanisms. In conclusion, there is ample evidence to suggest that AT plays a critical role in shaping the local tumour microenvironment as well as the systemic metabolic parameters that tumour cells are exposed to, through the secretion of inflammatory mediators. Such results might lead to the proposition of new therapeutic approaches targeting this subset of patients with aggressive diseases.

Title: Continuous Update Project RFA. Developing a template to carry out systematic reviews of mechanistic studies of diet and cancer
Speaker: Martin Wiseman/Sarah Lewis

Systematic reviews are acknowledged as the most rigorous way to synthesise data which have addressed a common question. Methods for conducting and reporting rigorous systematic reviews of clinical and epidemiological studies have been developed, but remain lacking for mechanistic studies. Given the plethora of published mechanistic studies in the field of nutrition and cancer, some way of synthesising, and judging the quality and reproducibility of, these studies is needed.

This project is funded by World Cancer Research Fund UK (WCRF UK), as part of the Continuous Update Project (CUP), which builds on the 2007 Expert Report Food, Nutrition, Physical Activity, and the Prevention of Cancer: a Global Perspective, and the systematic reviews on which it was based. Mechanistic evidence is one important component that the Expert Panel uses to infer causality among observed associations between nutritional and physical activity exposures and cancer incidence. WCRF identified the need to strengthen the evidence base for mechanistic data as part of the CUP.

The aim of this project is to draw on the expertise (in systematic reviews of epidemiological studies and in experimental studies of cancer) in our group in order to develop and publish a template for carrying out rigorous systematic reviews of mechanistic studies. The key objectives which will be addressed are: to design a comprehensive search strategy that will identify the diverse study types of relevance, to determine inclusion/exclusion criteria based on a hierarchy of evidence and quality criteria which will arise from this project, to identify and quantify publication bias, which is likely to be substantial, and to test our methodology in a feasibility study designed to examine the hypothesis that milk intake increases prostate cancer risk.

This study will be based within the University of Bristol, but with contributions from scientists from the University of Cambridge and the International Agency for Research on Cancer. We have selected a large multi-disciplinary team to carry out this project with expertise in informatics, statistics, cancer biology, epidemiology, genetic epidemiology, nutrition and systematic reviews. In order to achieve our objectives we will hold a series of four one day workshops in which we will brainstorm the expertise within the group, which spans many different cancer sites and disciplines of cancer research. The workshops will be a mixture of presentations, small-group discussions and open discussions. Between meetings we will carry out literature reviews, assess our methodology and ask each member of the team to comment on draft documents arising from the meetings and related research, and in doing this we will arrive at a consensus template for systematic reviews of mechanistic studies of diet and cancer. We will also carry out a systematic review to summarize the evidence from mechanistic studies potentially underlying the association between milk and prostate cancer. This will feed into and be carried out in parallel with developing the systematic review methodology above.

Developing a template will allow researchers to assimilate the overall evidence in order to test any hypothesis in this area. In doing this it may help to determine whether there is a causal link between an exposure and cancer and could also highlight areas where further research is necessary. This will ultimately contribute towards preventing cancer via dietary advice and interventions.
Title: Energy balance, adiposity, physical activity, and colorectal cancer: clinical perspectives

Speaker: Andrew G Renehan

A large volume of epidemiology demonstrates associations between increased body mass index (BMI), as an approximation of general adiposity, and increased risk of colorectal cancer (CRC). These associations are sex-specific (men >> women) and site-specific (colon >> rectum). Weight gain in adulthood is also associated with increased CRC risk in a sex- and site-specific manner. A smaller volume of epidemiology links measures of abdominal adiposity — namely waist circumference and waist-hip-ratio (as better approximates of metabolic dysfunction) — with increased risk of CRC. However, it is unclear whether these measures are more informative of risk, than BMI. For cancer mortality, a distinction is made between two study designs: (i) inception cohorts, evaluating the effect of baseline excess adiposity (pre-diagnosis) on cancer-related mortality; and (ii) cancer patient cohorts evaluating adiposity measures at or shortly after (post)-diagnosis and cancer outcome. For pre-diagnosis cohorts, baseline BMI negatively impacted on CRC-related mortality, in part, conditional on the increased incident risk. For post-diagnosis survival studies, there are many inherent biases, failure to take address confounding, and hence, susceptibility to misinterpretation. In general, the established link between excess adiposity and increased incident CRC risk does not necessarily extrapolate to inferior post-diagnosis survival. Indeed, there is emerging trial-based data suggesting that an overweight state is associated with improved survival in patients with colon cancer undergoing adjuvant chemotherapy — the ‘obesity paradox’. Consequently, it is unclear whether weight-control interventions in CRC patients will improve disease outcome.

Title: Energy balance, adiposity, physical activity, and colorectal cancer

Speaker: Jenifer Fenton

Obesity can result in increased systemic biomarkers of inflammation. Elevated hormones, growth factors and cytokines produced by adipocytes (adipokines) are associated with colon cancer risk and progression. We suggest that chronically elevated proinflammatory adipokines as observed in the obese state may have profound effects locally on colon epithelial cells through growth promotion effects, induction of autocrine signaling, angiogenesis and immune cross-talk. This dysregulation of the local environment via a systemic signal may lead to the promotion of colon cancer via a novel set of mechanisms. Elucidating the mechanisms by which obesity may increase cancer risk may lead to the identification of treatment/prevention targets. The use of models of this multistage process should allow for mechanism-based approaches to block phenotypes associated with the process of carcinogenesis. The review highlights using in vitro model systems of these various stages to understand the molecular mechanisms of obesity, adipokines and colon cancer risk. The advantage in using these systems is that the response of cells possessing various transformations can be compared to “normal cells”. An important key is to identify targets that are aberrant from normal to perturb for cancer prevention strategies. The long-term goal of this research is to understand how obesity increases colon cancer risk and identify and test dietary approaches to reduce the risk. Reducing this systemic inflammation associated with obesity is a viable target for prevention of chronic diseases and cancer.

Title: Energy balance, adiposity, physical activity, and pancreatic cancer

Speaker: Rudolf Kaaks

This presentation will discuss the epidemiological evidence of obesity, physical activity and pancreatic cancer and will discuss the underlying mechanisms. It will comment on methodological issues, challenges and opportunities for future research.
Title: Dietary energy balance modulation of murine pancreatic cancer: The role of insulin-like growth factor-1  

Speaker: Stephen D. Hursting  

New molecular targets and intervention strategies for breaking the obesity-pancreatic cancer link are urgently needed. Using relevant murine spontaneous and orthotopically transplanted models of pancreatic cancer, we tested the hypothesis that dietary energy balance modulation impacts pancreatic cancer development and progression through an insulin-like growth factor (IGF) 1–dependent mechanism. In LSL-KrasG12D/Pdx-1-Cre/Ink4a/Arflox/+mice and K5-cyclooxygenase (COX)-2 transgenic mice, calorie restriction, relative to overweight- or obesity-inducing diet regimens, decreased serum IGF-1, tumoral Akt/mammalian target of rapamycin (mTOR) signaling, pancreatic desmoplasia, and progression to pancreatic ductal adenocarcinoma (PDAC); and increased pancreatic tumor-free survival. Serum IGF-1, Akt/mTOR signaling, and orthotopically transplanted PDAC growth (multiple models) were decreased in liver-specific IGF-1−deficient mice (versus wild-type mice), and rescued with IGF-1 infusion. In addition, rapamycin and metformin inhibited mTOR and mimicked many of the anticancer effects of calorie restriction. Thus, dietary energy balance modulation impacts spontaneous pancreatic tumorigenesis induced either by mutant Kras and Ink4a deficiency (the most common genetic alterations in human pancreatic cancer) or overexpression of COX-2 (associated with human pancreatitis, which increases PDAC risk by up to 55-fold). Furthermore, IGF 1 and components of its downstream signaling pathway are promising mechanistic targets for breaking the obesity-pancreatic cancer link.

Title: Energy balance, adiposity, physical activity, and breast cancer  

Speaker: Cheryl L. Rock  

Breast cancer is the most common cancer worldwide in women, accounting for 23% of the total number of new cancers diagnosed. Death rates from breast cancer have been steadily declining, due to earlier diagnosis and more effective initial therapies, and there are now an estimated 2.9 million women in the U.S. alone with a history of breast cancer. Obesity is an established risk factor for postmenopausal breast cancer incidence and is a major risk factor for breast cancer recurrence and morbidity in both pre- and postmenopausal women. Both obesity and low levels of physical activity are associated with an increased risk of recurrence and all-cause mortality in breast cancer survivors. Even a modest degree of intentional weight loss favorably affects many breast cancer-relevant risk factors and potential mediators of progression (reproductive steroid hormones, Insulin and other growth factors, and inflammatory cytokines). Weight loss interventions for breast cancer survivors need to address issues specific to this population, including issues related to cancer and cancer treatments, enduring psychosocial symptoms, and changes in family dynamics. Increased physical activity is particularly important, due to effects of treatments on body composition. Results of several weight loss and exercise interventions in this target population have been reported, and more studies are ongoing. Individualised counseling (in person or telephone), group sessions, and mailed material have been shown to promote recommended behavioral changes. Evidence suggests that women at risk for breast cancer or breast cancer recurrence can make modifications in diet and physical activity to promote weight management.
SPEAKER ABSTRACTS CONTINUED

Title: Preclinical insights into the relationship of obesity and breast cancer
Speaker: Margot Cleary

Obesity is a risk factor for postmenopausal breast cancer but specific mechanisms of action remain unknown. In agreement, elevated body weight has been reported to affect latency and/or incidence of chemically-induced and spontaneous mammary tumors (MTs) in rodents, but these initial studies did not focus on mechanisms of action. Towards this goal more recently we found that neither genetically obese Lep^{ob}Lep^{ob} (0/59) (leptin deficient) nor Lepr^{db}Lepr^{db} (0/42) (leptin receptor deficient) mice developed transgene induced (MMTV-TGF-alpha) MTs, while lean mice had a MT incidence of 59% (44/74). These findings suggested an intact leptin-signaling axis is important in MT development. This is supported by the finding that MMTV-TGF-alpha mice with diet-induced obesity (DIO) had shortened MT latency compared to Obesity-Resistant mice that consumed the high-fat diet but weighed the same as Low-Fat mice. Serum leptin was elevated in DIO compared to Obesity-Resistant and Low-Fat mice, 10.6, 3.6 and 1.6 ng/ml, respectively. Other MT models have also been utilized by ourselves and others to address the role of obesity on MT development focusing on leptin as well as adiponectin. In addition to adipokines the insulin/IGF-1 axis may also be important in the obesity/breast cancer relationship. Of recent interest, epidemiological/observational studies indicate that treatment of diabetes with the insulin lowering drug, metformin, is associated with reduced breast cancer incidence. In addition, in vitro and preclinical studies support that metformin has cancer preventive properties. Presently, we are evaluating the impact of metformin versus weight loss on the development of MT in mice with diet-induced obesity.

Title: Overview of lifecourse factors and cancer
Speaker: Alan Jackson

Veterinary science has known for decades that early life exposures determine a fixed later phenotype. Furthermore, there is now a large and consistent body of research on early life events, including fetal and child growth and maturation, as determinants of cardiometabolic risk in humans. More recently evidence has emerged, as systematically explored and displayed in the WCRF /AICR Second Expert Report and the Continuous Update Project, that markers of early life events, growth and maturation predict risk of several cancers. Animal studies have identified plausible mechanisms through which such effects might be mediated in humans. However, the direction of effect differs, with greater growth increasing cancer risk but reducing cardiometabolic risk. It is particularly important to understand the mechanisms associated with these phenomena in order to determine optimal growth trajectories and safe and effective childcare and public health approaches to management.
Title: Developmental origins of obesity and cancer risk

Speaker: Susan Ozanne

It is well established, through studies in humans and animal models, that events in very early life can influence the risk of an individual developing metabolic conditions such as obesity, type 2 diabetes and insulin resistance. More recently it has been demonstrated that the early environment can also influence the risk of other conditions such as certain forms of cancer and mental health. This has been termed the Developmental Origins of Health and Disease. Exposures during critical periods of development that can influence disease risk include suboptimal maternal nutrition, maternal stress, maternal smoking and fetal/maternal hypoxia. To date much focus has been directed towards the effects of maternal under-nutrition followed by accelerated postnatal growth on the long-term health of the offspring. However in light of the growing epidemic of obesity, including in women of child-bearing age, attention has now also been directed towards understanding the effects of maternal over-nutrition during pregnancy and lactation on the offspring. To address the mechanisms underlying these associations we have used rodent models of maternal protein restriction and maternal diet-induced obesity (where dams are fed a diet rich in saturated fats and simple sugars reflective of a human westernized diet). These studies have demonstrated that key programming mechanisms include permanent changes in organ structure, accelerated cellular ageing and epigenetic changes. Understanding further these mechanisms could lead to the identification of novel targets for therapeutic intervention as well as markers of disease risk.

Title: Epigenetic mechanisms linking early life diet and cancer risk

Speaker: Karen A Lillycrop

Epigenetic processes play a central role in regulating gene expression and alterations in these processes have been implicated in the pathogenesis of many human diseases including human cancer. However there is now growing evidence that the environment particularly variations in diet, during specific developmental periods can induce changes in the epigenome, which are then stably maintained throughout life influencing cancer susceptibility. This talk will review the evidence that alterations in early life nutritional exposure can alter the epigenome and subsequent cancer risk and discuss how detection of such altered epigenetic marks in early life may provide biomarkers to detect individuals at increased risk of disease.

Title: Policy on obesity and physical activity: how cancer fits into chronic disease agenda and why

Speaker: João Joaquim Rodrigues Silva Breda

The burden of diseases due to poor diet remains high and in many countries within the WHO European Region it is actually increasing. Chronic undernutrition is declining but continues to have a marked impact on health in some member states, while overweight, obesity and excess consumption of saturated and trans fats, sugars and salt, and low consumption of fresh vegetables and fruits, have become the leading risk factors for many noncommunicable diseases. Policies to tackle this disease burden have been developed through a series of strategic initiatives globally and at the European level, including the European Charter on Counteracting Obesity, the Action Plan for the Implementation of the European Strategy for the Prevention and Control of Noncommunicable Disease, and the Health 2020 framework. An overview of measures and initiatives to implement nutrition health policies, developed within the WHO European Region is presented.

Note: Conflicts of interest and sources of funding will be declared in the speaker presentations and/or made available on the WCRF International website in due course.
## POSTER ABSTRACTS

The poster abstracts are ordered by alphabetical order of first author. The presented posters will be displayed in the Lecture Theatre and the Breakout Area.

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POSTER ABSTRACTS

Childhood body mass index and the risk of prostate cancer in adult men
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Background: High body mass index (BMI; kg/m²) among adult men is potentially associated with an increased risk of prostate cancer, although evidence is inconsistent. It is largely unknown whether any of this potential risk originates in childhood.

Aim: To investigate if childhood BMI at each age from 7-13 years is associated with the risk of prostate cancer in adult men.

Methods: Subjects were 125,209 men from the Copenhagen School Health Records Register born from 1930-1969 with data on measured heights and weights at 7-13 years. BMI was transformed into age- and sex-specific z-scores using an internal reference. Follow-up occurred by linkage via a personal identification number to the Danish Cancer Registry and the vital statistics register. Cox proportional hazards regressions were performed at each age and stratified by birth cohort. Subjects were followed from age 40 until a diagnosis of prostate cancer, death, emigration, loss-to-follow-up, or December 31, 2010; whichever came first.

Results: 2,987 men were diagnosed with prostate cancer. At age 7 the risk of prostate cancer in adulthood was 1.06 (95%CI: 1.02-1.11) per BMI z-score increase, and at 13 years the risk was 1.06 (95%CI: 1.01-1.11) per BMI z-score increase. The estimates were similar at all other ages.

Conclusions: Higher BMI during childhood is associated with an increased risk of prostate cancer in adulthood. The identification of childhood BMI as a potential risk factor for prostate cancer is of public health interest due to its modifiable nature and contributes to the limited knowledge on identified risk factors.

Prediagnostic circulating adipokine concentrations and risk of colorectal cancer in the European Prospective Investigation into Cancer and Nutrition (EPIC)
Aleksandrova K.1, Boeing H.1, Jenab M.2, Bueno-de-Mesquita H.B.3,4, Jansen E.3, van Duijnhoven F.J.B.3,5, Rinaldi S.2, Fedirko V.2,6,7, Romieu I.2, Gunter M.J.8, Riboli E.8, and Pischon T.1,9
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Background: Adipose-tissue-derived proteins collectively named “adipokines” may provide a molecular link between adiposity and colorectal cancer; however evidence from large prospective studies on these associations is limited.

Methods: We studied prospectively the association of prediagnostic circulating concentrations of adipokines (total adiponectin, high-molecular-weight (HMW), non-HMW-adiponectin, leptin and soluble leptin receptor (sOB-R)) with risk of colorectal cancer in a nested case-control study within the European Prospective Investigation into Cancer and Nutrition (EPIC) cohort. A total of 1,206 incident cases with measured adiponectin concentrations and 1,129 cases with measured leptin and sOB-R concentrations were matched within risk sets to equal numbers of respective controls using incidence density sampling. Conditional logistic regression was used to calculate relative risks (RRs) and 95% confidence intervals (95% CIs).

Results: After multivariable adjustment for dietary and lifestyle factors, body mass index and waist circumference, non-HMW-adiponectin and sOB-R were inversely associated with colorectal cancer risk (RR comparing top versus bottom quintile = 0.39; 95%CI= 0.26-0.60, \( P_{\text{trend}} = 0.001 \); and \( RR = 0.61; 95\%\text{CI} = 0.45-0.83; P_{\text{trend}} = 0.002 \), respectively). After the same multivariable adjustment, no associations were seen for total adiponectin (RR = 0.81; 95%CI= 0.60-1.09; \( P_{\text{trend}} = 0.23 \)), HMW-adiponectin (RR = 1.05; 95%CI= 0.77-1.43; \( P_{\text{trend}} = 0.11 \)), and leptin (RR = 0.85; 95%CI= 0.56-1.29; \( P_{\text{trend}} = 0.23 \)).

Conclusion: These findings suggest that selected adipokines, including non-HMW-adiponectin and sOB-R, may play an important role in the pathogenesis of colorectal cancer. More research is warranted to confirm these results and to elucidate underlying mechanisms.
Metabolic biomarkers as mediators of the association between adiposity and risk of colon cancer: findings from the European Prospective Investigation into Cancer and Nutrition (EPIC)

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Background: Adiposity is a risk factor for colon cancer but underlying mechanisms are not well understood. We evaluated the extent to which circulating biomarkers, having inflammatory and metabolic potential, mediate the association of adiposity measures, waist circumference and body mass index (BMI), with colon cancer.

Methods: We analysed data from a nested case-control study in the European Prospective Investigation into Cancer and Nutrition (EPIC) among 662 incident colon cancer cases matched within risk-sets to 662 controls. Relative risks (RRs) and 95% confidence intervals (CIs) were calculated using conditional logistic regression. Percent effect change and corresponding CI-s were estimated after adjustment for metabolic biomarkers.

Results: After multivariable adjustment, waist circumference was associated with colon cancer risk in men (top vs bottom tertile RR = 1.77; 95%CI = 1.14-2.77; P trend = 0.006) and in women (RR=1.64; 95%CI = 1.08-2.48; P trend = 0.03). BMI was associated with risk only in men. The association of colon cancer with waist circumference was mostly accounted for by three biomarkers, high-density lipoprotein cholesterol, non-high-molecular-weight adiponectin and soluble leptin receptor, which in combination explained 45% (95%CI=20% to 95%) of the association in men and 51% (95%CI=23% to 128%) of the association in women. Similar results were observed for the association with BMI in men.

Conclusions: These data suggests that alterations in the levels of metabolic biomarkers may represent a primary mechanism of action in the relation of adiposity with colon cancer. Further studies are warranted to determine whether altering the concentrations of these biomarkers reduces colon cancer risk.

Anthropometric measures and risk of ovarian cancer: a systematic review and meta-analysis of prospective studies

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Background: The World Cancer Research Fund/American Institute for Cancer Research report “Food, Nutrition, Physical Activity, and the Prevention of Cancer: A Global Perspective” from 2007 stated that the evidence relating body fatness and abdominal fatness to ovarian cancer risk was inconclusive, but the evidence was based on a limited number of studies. Greater height was probably associated with an increased risk of ovarian cancer. As part of the Continuous Update Project of the World Cancer Research Fund we conducted an updated systematic review and meta-analysis of anthropometric measures and risk of ovarian cancer.

Methods: PubMed and several other databases were searched up to December 2012. Summary relative risks were calculated using a random effects model.

Results: Twenty-three publications (19 prospective studies) have been published since the WCRF/AICR report. The summary RR was 1.06 (95% CI: 1.02-1.11, P=54%, n=22) per 5 units increase in body mass index (BMI), 1.05 (95% CI: 1.02-1.07, P=0%, n=3) per 5 kg increase in weight, 1.03 (95% CI: 0.97-1.10, P=0%, n=4) per 10 cm increase waist circumference, 1.01 (95% CI: 0.75-1.36, P=81%, n=3) per 10 cm increase in hip circumference, and 0.99 (95% CI: 0.92-1.06, P=0%, n=4) per 0.1 unit increase in waist-to-hip ratio. The summary RR per 5 cm increase in height was
1.08 (95% CI: 1.05-1.10, I²=35%, n=14). In the analysis of BMI and ovarian cancer, there was indication of publication bias with Egger, p=0.05, but this appeared to be explained by the largest study, and when excluded Egger’s test was no longer significant, p=0.93 and heterogeneity was reduced (I²=21%), but the summary estimate was not substantially altered, RR = 1.07 (95% CI: 1.04-1.11). There was evidence of a nonlinear association between BMI and ovarian cancer (pnonlinearity<0.0001), with a steeper association at higher levels of BMI (statistically significant from BMI>28.4), but not for height, (pnonlinearity=0.09).

Conclusion: Our results suggest that greater body fatness and height increases ovarian cancer risk. Further studies are needed to clarify the association between abdominal fatness and ovarian cancer risk.

Role of leptin as a biomarker in prostate cancer

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Epidemiological and preclinical data suggest that leptin, an adipokine produced prevalently by adipose tissue, is associated with prostate cancer (PCa) and can directly modulate PCa cell behaviour. In an observational study, we evaluated the role of leptin as cancer biomarker in cohorts represented by PCa patients and patients with urologic non-tumoural diseases, considering as covariables the age and the anthropometric measures of adiposity (body mass index, BMI, and waist to hip ratio, WHR). Our data confirmed the association of leptin levels with PCa diagnosis: in absence of significant differences in BMI classification, PCa patients had higher leptin values respect to control patients. Further analysis revealed a stronger predictive value of serum leptin in older PCa subjects, as demonstrated by a better ROC curve. Recombinant leptin stimulated cancer cell survival and proliferation in PCa cell lines but these effects were statistically appreciable mostly in glucose-restricted culture conditions. Metabolic switch induced by leptin in PCa cells was associated with upregulation of sirtuin 3 (SIRT3) and activation of AMPK. In conclusion, visceral adiposity (WHR) appeared a more appropriate measure of obesity in elderly subjects with respect to BMI categories, and may explain the elevated leptin values seen in elderly PCa subjects with higher WHR. The expression of leptin receptor mainly observed in invasive prostate carcinoma tissue and in aggressive prostate cancer cell lines in parallel with in vitro evidence suggests a possible molecular link between persistently high leptin levels, mainly seen in aged obese subjects, and PCa progression.

The EnCoRe study: design of a cohort study on diet, physical activity and health-related quality of life in colorectal cancer survivors based on a bio-psychosocial framework

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Background & Aim: Although the number of people surviving colorectal cancer (CRC survivors) continues to grow, individually tailored lifestyle interventions targeting diet and physical activity to safeguard or improve their health-related quality of life (HRQoL) are lacking. The EnCoRe (Energy for life after ColoRectal cancer) study aims to investigate how diet and physical activity influence HRQoL in CRC survivors, providing lifestyle intervention targets. WHO’s International Classification of Functioning, Disability and Health (ICF) was used as bio-psychosocial framework to design the EnCoRe study. The ICF systematically classifies human functioning in three domains (body functions/structures, activities, participation) that interact with individuals’ health condition and the environmental/personal context in which functioning takes place.
**Methods:** The EnCoRe study comprises two parts. First, a prospective study in which stage I-III CRC patients (N=~240) are enrolled at diagnosis and followed up until 2 years after initial anti-cancer treatment. Second, a cross-sectional study with 2-10-year post-treatment stage I-III CRC survivors (N=~220). HRQoL is measured by the European Organization for Research and Treatment of Cancer Quality of Life Questionnaire (EORTC QLQ-C30), including CRC-specific module CR29. Dietary intake is assessed using 7-day food diaries, and physical activity by 7-day accelerometry and the Short QUestionnaire to ASsess Health-enhancing physical activity (SQUASH). Additional health-related outcome measures include fatigue, physical functioning, anxiety and depression.

**Results & Conclusion:** Participant enrolment is ongoing. The EnCoRe study will increase understanding of complex interrelations between diet, physical activity and HRQoL, and identify targets for development of novel lifestyle interventions to improve the HRQoL of CRC survivors.

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**Path Model of Early Childhood Obesity: parenting stress, feeding, eating and socio-demographics in Hong Kong**

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**Aims:** The aims of this study were to explore the interrelationship between young children’s weight outcomes and parenting stress, parental feeding and children’s eating behaviour in Hong Kong in order to provide information for the intervention of rapid increased childhood obesity worldwide.

**Methods:** A number of 336 Chinese parents of young children aged 2-7 years from 31 kindergartens with different SES in Hong Kong were recruited for a cross sectional study. Young children’s BMI were classified by the IOTF. The Parental Feeding Behaviour Questionnaire (PFBQ) and Child Eating Behaviour Questionnaires (CEBQ), Parenting Stress Index (PSI-SF) were employed.

**Data Analysis:** Path analyses were used to compare and to investigate associations between variables.

**Results:** The first model indicated that higher parenting stress significantly predicted higher children food responsiveness (Standardized beta-coefficient = 0.195, p < 0.001), which predicted higher weight status (Standardized beta-coefficient = 0.249, p < 0.001). Higher parenting stress also predicted higher instrumental feeding (Standardized beta-coefficient = 0.294, p < 0.001) which predicted lower weight status (Standardized beta-coefficient = -0.204, p < 0.001). The standardized indirect effects of parenting stress on children’s weight status via food responsiveness (Standardized Sobel’s Z = 2.799, p < 0.01) and instrumental feeding (Standardized Sobel’s Z = -3.015, p < 0.01) were significant. The second model showed higher parenting stress predicted higher emotional feeding (Standardized beta-coefficient = 0.242, p < 0.001), which predicted higher weight status (Standardized beta-coefficient= 0.249, p < 0.001).

**Conclusions:** These findings could suggest directions to early childhood obesity interventions.
Educational workshops in Brazil: contextualising obesity related cancer prevention recommendations

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The National Cancer Institute (INCA), Ministry of Health, Brazil, works towards tackling the burden of cancer, the second highest cause of death nationally. Within INCA, the Food, Nutrition and Cancer Division focuses on raising the social recognition of cancer preventability and the role of obesity, nutrition and physical activity therein. Additionally it works towards developing, fostering, integrating and implementing policies and actions to promote healthy eating practices that contribute to the prevention of obesity and cancer. The main information sources supporting these activities are WCRF and AICR’s 2007 Diet and Cancer Report Summary translated by INCA, and INCA’s 2009 Brazilian Policy Report Executive Summary. Since 2009 educational workshops have been performed with primary health professionals in five of the 27 Brazilian states promoting dialogue between scientific and popular knowledge. Approximately 300 participants have been involved, including health system managers, dieticians, nurses, community health agents, physicians and physical activity professionals. Participatory methods have been used to discuss the recommendations for cancer prevention and translate them into contextually appropriate messages and interventions within local communities. In the Amazonian state of Pará, for example, fish is still mostly salt preserved and beans are commonly prepared with processed meats. Recommendations are also included in ongoing local activities. In the north-eastern state of Sergipe participants suggested inclusion of the topic in the national strategy for breastfeeding and complementary feeding practices. Other initiatives resulting from the workshops include a leaflet and a recipe book by the Rio de Janeiro and Pará groups respectively. In 2013 eight workshops are planned to continue this work.

Impact of body mass index, physical activity and three other lifestyle factors on cancer risk: results from the E3N cohort

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Aims: To quantify the impact of five lifestyle characteristics on all-sites and site-specific cancer risk in middle-aged women.

Material: The study included 64,732 women from the French E3N prospective cohort, aged 40 to 65 at recruitment in 1990. During 19 years of follow-up, 6,938 cases of invasive cancer were diagnosed.

Methods: The studied lifestyle characteristics (physical activity, body mass index (BMI), smoking, alcohol drinking, and fruits and vegetables consumption) were aggregated into a healthy index, scored from 0 (unhealthy) to 5 (healthy). Population attributable fractions (PAF) were estimated to quantify the proportion of cancer cases that would have been prevented if all women had followed a healthy lifestyle.

Results: If all women had been in the healthy BMI category (18.5-25 kg/m²), whatever the other 4 dimensions, 1.1% of any-site, 0.3% of postmenopausal breast and 1.4% of colorectal cancer cases would have been prevented. If all women had been highly physically active (i.e. exercising more than 35 Metabolic Equivalents of Task (METs)-hour/week), 1.2% of any-site, 2.2% of postmenopausal breast and 1.4% of colorectal cancer cases would have been prevented. Combining all five healthy lifestyle characteristics would have prevented 7.9% of any-site cancer cases, 16.5% and 6.1% of post-menopausal breast and colorectal cancer cases, respectively.

Conclusion: Although adhering to one healthy behavior only seemed to have a moderate impact on the prevention of any-site and site-specific cancer, adhering to a whole healthy lifestyle may substantially decrease the number of diagnosed cancer cases.
POSTER ABSTRACTS CONTINUED

Weight change later in life in relation to breast cancer risk: results from the European Prospective Investigation Into Cancer (EPIC)

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**Background:** Long-term weight gain has been related to higher risk of postmenopausal breast cancer, whereas it has a protective effect against premenopausal breast cancer. The effect of weight change later in life, especially around menopause, is unclear. Here we investigate the association between weight change later in life and the risk of breast cancer with an emphasis on timing of weight change.

**Methods:** The association between prospectively assessed weight change (median follow-up of 4.3 years) and breast cancer risk was examined in 205,723 women participating in EPIC. Annual weight change was categorised using quintiles taking quintile 2+3 as the reference (range: -0.44 to 0.36 kg change/year). Multivariable Cox regression analysis was used to examine the association between weight change and breast cancer risk.

**Results:** During a median follow-up of 7.5 years 4,663 incident breast cancer cases were diagnosed. Highest quintile weight gain (1.55±0.7 kg/year) was related to a slightly, but significantly higher breast cancer risk (HRQ5-Q2/3 1.09, 95% CI: 1.01−1.18). The association was most pronounced for weight gain before age 50 in relation to breast cancer diagnosed before or at age 50 (HRQ5-Q2/3 1.37, 95% CI: 1.02−1.85). Weight loss was not associated with breast cancer risk (HRQ1-Q2/3 : 1.03, 95% CI: 0.95−1.12). There was no evidence for heterogeneity of the association according to tumour hormone receptor status.

**Conclusion:** High weight gain later in life was associated with increase breast cancer risk, especially of cancer diagnosed before or at age 50. Our results illustrate the importance of avoiding weight gain.

Insulin resistance, IGF-1 and tumor cell proliferation (Ki67) among newly diagnosed breast cancer patients

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**Background:** High levels of insulin and Insulin Growth Factor-1 (IGF-1) have been associated with breast cancer prognosis, but less is known about these factors in relation to tumour cell proliferation (Ki67 index).

**Methods:** Among women with newly diagnosed stage I-II invasive breast cancer, aged 35-75 years, body mass index [BMI, kg/m²] and waist circumference [WC, cm] were measured and fasting serum concentrations of glucose, insulin, insulin resistance [Homeostasis Model Assessment (HOMA)-score] and IGF-1 were assessed before surgery. Tumor cell proliferation, Ki67 index was determined according to international guidelines. Linear regression models were used to test for associations between selected patient characteristics and Ki67 index in breast tumours.

**Results:** Among 45 breast cancer patients, mean age at diagnosis was 54.9 years, mean BMI was 24.8 kg/m², and mean WC was 87.0 cm. We observed a negative association between estrogen receptor (ER)-positive tumours and Ki67 index (beta = -0.45 [95% CI: -0.63, -0.27], p<0.0001) and progesterone receptor (PR)-positive tumours and Ki67 index (beta = -0.29 [95% CI: -0.45, -0.12], p=0.001), and a positive association between histologic grade 3 versus grade 1 (beta =43.4 [95% CI: 32.0, 54.9], p <0.0001) and Ki67 index. Among premenopausal breast cancer patients IGF-1 and insulin resistance was positively associated with Ki67 index, respectively (betaIGF-1 =1.73 [95% CI: 0.25, 3.21], p=0.025) (betainsulinresistance =41.9 [95% CI: -8.75, 92.6], p=0.097).

**Conclusion:** Our results hypothesize an association between IGF-1, insulin resistance and Ki67 index, but these associations need to be replicated in larger studies.
Modification of the nutritional status and physical activity level in French breast cancer patients starting chemotherapy

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In breast cancer (BC) patients, a lack of physical activity (PA) is a probable factor of weight gain during adjuvant treatment. A moderate-intensity PA could reduce recurrence and improve survival. A randomised controlled trial is ongoing to study the feasibility and benefits of a programme promoting healthy diet and adapted PA in BC patients starting adjuvant chemotherapy. Baseline anthropometrics and PA level are presented.

At first chemotherapy, weight, height and waist circumference (WC) were measured. Body fat (BF) was assessed by bioelectrical impedancemetry. Aerobic capacity (VO$_{2\text{max}}$), daily energy expenditure (DEE), time spent in moderate-intensity PA (≥4METs) and screen time were estimated by the PAQAP© questionnaire during the month preceding chemotherapy and retrospectively during the pre-diagnostic period. Medians were compared using the Wilcoxon signed-rank test.

Among the first 37 patients enrolled (median age, 53y), median body mass index (BMI) was 23.4kg/m$^2$. While 16 (43%) women were overweight (BMI>25kg/m$^2$), 33 (89%) presented excess BF (median BF, 35%). Median WC was 85cm and 23 (62%) patients had excess visceral adiposity (WC>80cm). Between the pre-diagnosis and pre-chemotherapy periods, DEE (~591kJ/d), VO$_{2\text{max}}$ (~1.7ml/min/kg) and moderate-intensity PA (~8min/d) decreased and screen time increased (~42min/d) (p<0.001 for all).

While the proportion of overweight patients was similar to that of French women of same age, a large majority had excess BF and a WC reflecting metabolic risks. Moreover, a significant decrease in PA level early after diagnosis supports the necessity of engaging patients in PA soon after BC diagnosis to prevent physical deconditioning and co-morbidities.

Attenuation of brown adipose tissue activity during chemotherapy treatment: a new mechanism of weight gain in breast cancer women?

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**Background and Aims:** Weight gain has been reported in 17 to 34% of early stage breast cancer patients during chemotherapy. This gain has become a major concern because it has been suggested that weight variation was associated to a poor prognosis. However the mechanisms involved remain unclear. Alteration in thermogenesis induced by a decrease of brown adipose tissue (BAT) activity after chemotherapy, may partly contribute to weight gain in these patients.

**Methods:** A PET/CT scan was performed at baseline and after one course of docetaxel + trastuzumab treatment in 26 breast cancer women. Variation of 18F-FDG uptake in BAT between the two measures was assessed retrospectively according to weight changes.

**Results:** Percentage of patients who gained weight was 35%, 25% lost weight and 40% remained stable. Women who gained weight during chemotherapy experienced a significant decrease of 18F-FDG uptake in BAT (p=0.0048).

**Conclusion:** These original data suggest for the first time that BAT modulation by chemotherapy would be a potential contributor to body weight gain through blunted thermogenesis in breast cancer patients.
Feasibility and preliminary results about using a Telerehabilitation system (E-CUIDATE) in quality of life for breast cancer survivors


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Objective: This pilot study trialled a telerehabilitation web system to determine the feasibility of delivering rehabilitation services based on remotely therapeutic exercise for breast cancer survivors.

Methods: Patients were prospectively recruited to a randomised clinical trial (NCT01801527) using E-CUIDATE telerehabilitation system (cancer related symptoms application of assessment, personal physical training adapted to their symptoms using HD-image sequence and videoconferencing system to monitor the physical exercise programme). Quality of life is the primary outcome of this study. It was assessed using the EORTC QLQ-C30 and QLQ-Br23 questionnaires. These are both validated and gold standard measures for the assessment of overall health-related quality of life for cancer patients (QLQ-C30) and specifically for breast cancer patients (QLQ-Br23). Quality of life was assessed at baseline and 4 weeks after the intervention.

Results: A total of 56 breast cancer survivors participated and 32 of them received the telerehabilitation intervention with adequate acceptability for the participants. The analysis of the study results revealed significant differences in quality of life between measurement phases in one of the symptoms domains: fatigue (P=0.001) and a tendency to significant improvement (P=0.05) in physical, cognitive and role functioning symptom domains. An improvement in body image subscale of breast module was found in the intervention respect usual care group (P=0.002). The rest of subscales and overall quality of life were not significantly different between groups (P>0.05).

Conclusion: A 4-week period of telerehabilitation intervention in breast cancer survivors is feasible and could improve specific cancer related symptoms but not overall quality of life.

Physical activity and nutritional interventions for the development and progression of prostate cancer: a systematic review

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Background: Prostate cancer is the most common male cancer in the Western world. It is often slow growing; however in some men the cancer may have more rapid progression. Physical activity and nutritional interventions have demonstrated positive outcomes in colon and breast cancer patients, such as improved cancer survival, prevention of progression and reduced cancer recurrence. Nutritional and physical activity interventions could have similar positive outcomes for men with prostate cancer.

Aim: This review aims to systematically and critically evaluate physical activity and nutritional interventions in men with prostate cancer, which aimed to impact the development or progression of the disease.

Methods: Five databases (Embase, Medline, AMED, Cinahl, The Cochrane Library) were searched from inception to present day, identifying all physical activity or nutritional intervention studies in prostate cancer populations. No language restrictions were imposed. Two reviewers will independently determine the eligibility of studies for inclusion using predefined criteria. Data will be extracted, assessed for methodological quality and synthesised.

Results: The search identified 9257 papers, equating to 4057 (Embase), 4000 (Medline), 10 (AMED), 432 (Cinahl), 758 (The Cochrane Library). Duplicates will be removed and those not meeting pre-defined inclusion criteria will be excluded. From scoping the literature it is anticipated that physical activity interventions will include brisk walking and resistance training, and that nutritional interventions will include lycopene and selenium.

Conclusions: Data extraction and analysis is on-going. A feasibility trial of a physical activity and nutritional intervention for men with prostate cancer will be developed, based on the outcomes from this systematic review.
Physical effectors of body composition and weight gain among BRCA mutation cancer survivors

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Aims: Post-diagnosis weight gain is a risk factor for breast cancer recurrence. Mechanisms underlying post-diagnosis weight gain have not been clearly delineated but may involve adverse changes in body composition. The purpose of this study was: 1) to examine the association between anthropometrics and post-diagnosis weight gain among BRCA mutation cancer survivors and 2) to identify potential effectors.

Methods: The study population included 37 breast and ovarian cancer survivors with BRCA mutations, 45 BRCA previvors, and 26 controls. Anthropometric measurements included weight, body mass index (BMI), and body composition (bioelectric impedance). Potential effectors of anthropometrics included physical activity (General Practice Questionnaire), sleep, diet, quality of life (QoL; Rand-36), and anxiety (Beck).

Results: We found no significant differences in BMI between survivors, previvors and controls (26±2, 25±1, 25±1, respectively). However, lean body mass was significantly lower and body fat mass significantly higher for the survivors as compared to the previvors or controls (p=0.0005). Body composition (lean/fat mass) was an independent predictor of post-diagnosis weight gain (r=0.86, p<0.001). Body composition measures were not significantly (p>0.05) associated with psychological status, diet or sleep but were significantly (p<0.001) correlated to levels of physical activity and physical QoL (pain, functioning, limitations).

Conclusions: BRCA cancer survivors have adverse post-diagnosis changes in body composition in association with low physical activity and reduced physical QoL. Based upon these findings, the post-diagnosis survivorship program for BRCA survivors may need to include a regimen of regular physical activity, healthcare attention to physical QoL, and routine anthropometric assessments, including body composition.

Obesity promotes the development of multiple myeloma in vivo

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Multiple myeloma is a fatal haematological malignancy associated with tumour growth within the bone marrow and the development of an osteolytic bone disease. There is increasing evidence to support an association between obesity and myeloma, however the mechanisms that mediate this remain unknown. To determine whether obesity promotes myeloma development, we have used the well-characterised 5TGM1 murine model of myeloma, where myeloma will only develop when 5TGM1 myeloma cells are inoculated into immunocompetent C57Bl/KaLwRij mice (KaLwRij) mice and not in closely related C57Bl6 mice. The aim of this study was to determine whether (i) whether myeloma-permissive KaLwRij mice were obese and (ii) whether diet-induced obesity can promote myeloma development in otherwise non-permissive C57Bl6 mice. Using body composition analysis, myeloma-permissive KaLwRij mice had a significant increase in body weight, total fat and percentage fat mass as compared with age- and sex-matched C57Bl6 mice. In addition, KaLwRij mice had a significant reduction in trabecular bone volume and an increase in bone marrow adiposity. C57Bl6 mice were fed a high fat diet or control diet for 4 weeks, when a significant increase in percentage body fat was detected. At this point, mice were inoculated with 5TGM1 myeloma cells. The high fat diet promoted myeloma growth within the bone marrow and trabecular bone loss. Removal of the diet upon detection of myeloma reduced tumour burden, but had no effect on trabecular bone volume. Taken together, these studies demonstrate that an increase in adiposity creates a permissive host microenvironment for the development of multiple myeloma.
Objectively-assessed physical activity and sedentary behaviour amongst colon cancer survivors

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Aim: Moderate-vigorous physical activity is associated with prolonged survival, diminished treatment side effects and enhanced quality of life for cancer survivors. Emerging evidence suggests that sedentary behaviour (sitting time) may also affect health outcomes. Our understanding of these health behaviours in cancer survivor populations is limited, and based on self-reported data. Our study aim was to objectively examine the prevalence of physical activity and sedentary behaviour amongst colon cancer survivors, using accelerometers.

Methods: Two hundred and eighty-five potentially eligible Stages I – III colon cancer survivors from the Western Australian Cancer Registry and 380 cases from the Southern Alberta Cancer Registry were identified. In total, 203 individuals (31%) agreed to participate. An Actigraph® GT3X+ accelerometer and self-administered questionnaire were posted to participants. Participants wore the accelerometer for seven consecutive days, and the resultant data were summarised using Freedson cutpoints.

Results: The accelerometers were worn, on average, for 14 hours and 20 minutes each day. Sedentary behaviour comprised 62% of the recorded day; light-intensity physical activity comprised 35% and moderate-intensity physical activity made up 3%. Vigorous-intensity physical activity was negligible; 80% of participants recorded no vigorous activity on any day. Sedentary and activity levels varied significantly by age (sedentary p<0.001; light p=0.006; moderate p<0.001) and body mass index (sedentary p<0.001; light p=0.014; moderate p<0.001).

Conclusions: Colon cancer survivors are highly sedentary. The objective-assessment of physical activity and sedentary behaviour may better inform behavioural change interventions and translational research that aim to enhance quantity and quality of life for cancer survivors.

Malaysian breast cancer survivorship cohort: body mass index and body fat percentages of the newly diagnosed patients

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Background: The incidence of breast cancer in Malaysia is increasing and it may be due to adopting the lifestyle and dietary pattern of the developed countries. Breast cancer (BC) is the commonest cancer among women and has good survivorship rate if detected early. A study has shown that Malaysian women with BC have 58% of overall 5 year survival. Several studies have shown positive association high body mass index (BMI) and BC risk among postmenopausal women but little is known from the developing Asian countries. The aim of this study was to investigate the BMI and body fat percentage of the newly diagnosed BC patients.

Method: Hundred and thirty-one patients (> 18 years old) were recruited and weighed in this study. Height was measured using stadiometer SECA 217 (Hamburg, Germany). Weight and body composition were measured using TANITA BC 418 (Illinois, USA). Measurements were taken from patients prior to having their breakfast.

Results: The mean age of presentation was 55 years old (range 18-80) with 85% above 50 years old. Sixty four percent of the newly diagnosed BC patients were either overweight or obese with the mean BMI of 26.5 kg/m² (range 16.1-43.7). Only 40% of the BC patients have their body fat composition within the normal range, the mean fat% was 34.7  (range 17.7 – 49.4).

Conclusion: Malaysian newly diagnosed BC patients were either overweight/obese. This information may be useful for public health interventions in designing future therapeutic and lifestyle interventions.
Supplementation with tocotrienols and whey protein isolate improves exercise capacity in diet-induced obese rats

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Obesity-induced impairments in metabolic health are strongly associated with reductions in exercise capacity and an increased risk of cancer. Recent research shows that both whey protein isolates (WPIs) and vitamin E tocotrienols (TCTs) exert favorable effects on obesity-related metabolic parameters. This research sought to test the effects of dietary supplementation with TCTs and WPIs, alone and in combination, on metabolic and exercise capacity parameters in diet-induced obese Sprague-Dawley (SD) rats. Male SD rats (6 weeks old) were placed on a high-fat diet (40% of energy from fat) for 10 weeks to induce obesity. They were then separated into 4 groups: control (n=8), TCT (n=9), WPI (n=7) and TCT+ WPI (n=7) and maintained for a further 8 weeks. Peak running speed on a treadmill was determined for each rat 2 weeks before a timed test to exhaustion (TTE) at 65% of their peak running speed. The TCT (110 ± 7mins), WPI (110 ± 12mins), and TCT+WPI (98 ± 5mins) groups were able to run significantly longer than the control group (67 ± 6mins) in the TTE (P<0.05). Additionally, WPI increased the maximum in vitro activity of beta-hydroxyacyl-CoA in the soleus muscle of animals (P<0.05 vs. Control). The results show that dietary TCTs and WPI increase exercise capacity in obese rats, and WPIs may exert their effect through enhancing fatty acid oxidation in skeletal muscles. By increasing capacity for exercise, dietary supplementation with TCTs and WPI may reduce obesity and associated cancer risk.

Physical activity and breast cancer in women: the modifying role of menopausal status and obesity

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Aim: The aim of this study is to investigate the association between physical activity and breast cancer, as well as the potential modifying role of menopausal status and obesity.

Methods: Two hundred and fifty breast cancer patients with first diagnosis of the disease (56 ± 12 years old) and 250 age-matched, population-based healthy controls were recruited in Athens, Greece, from November 2010 to July 2012. Socio-demographic, dietary, psychological, lifestyle as well as environmental characteristics were recorded through face-to-face interviews with the participants. Physical activity was assessed using the International Physical Activity Questionnaire (IPAQ) index that has been validated for the Greek population. Multivariate logistic regression analysis was performed, including as potential confounders age, body mass index (BMI), menopausal status, current smoking and Mediterranean Diet Score.

Results: Even moderately physically active women (i.e. IPAQ score=600-3000 MET-minutes/week) had lower likelihood of having breast cancer (OR=0.41; 95%CI: 0.27-0.62). Engagement to more intense exercise (i.e. IPAQ score>3000 MET-minutes/week) did not seem to confer further protection (OR=0.50; 95%CI: 0.26-0.98). Menopausal status modified the aforementioned results; moderate exercise (OR=0.36; 95%CI: 0.21-0.60) as well as intense exercise (OR=0.46; 95%CI: 0.21-1.006; p=0.05) maintained their protective role in postmenopausal women, whereas no significant association was observed for premenopausal women. Moreover, obesity (BMI>30 kg/m²) seemed to mask the protective effect of intense physical activity since no significant relationship was observed (p=0.24).

Conclusions: Moderate and intense physical activity seems to provide a similar magnitude of protection in terms of breast cancer risk in postmenopausal women.
Association between C-reactive protein and colorectal cancer risk: a Mendelian Randomisation study using multiple genetic variants


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Background: Circulating C-reactive protein (CRP), an acute phase marker of chronic low-grade inflammation, has been associated with a moderately increased risk of colorectal cancer in several prospective studies including the European Prospective Investigation into Cancer and Nutrition (EPIC). To improve causal inference, we conducted a Mendelian Randomisation study using multiple genetic variants as relatively unbiased proxies for CRP concentration.

Methods: A prospective nested case-control study within EPIC included 727 cases diagnosed between 1992 and 2003 and 727 matched controls selected according to an incidence-density sampling protocol. Baseline CRP concentrations were measured in plasma samples by a high sensitivity assay. Single nucleotide polymorphisms (SNPs) in the CRP gene (rs1205, rs1800947, rs1130864, rs2808630, rs3093077) were identified via HapMap.

Results: The SNPs rs1205, rs1800947, rs1130864 and rs3093077 were significantly associated with CRP concentrations and incorporated in a CRP allele score that was associated with 13% higher CRP per allele count. Using the CRP-score as instrumental variable, a genetic increase in CRP concentrations by the factor 2 was statistically significantly associated with higher risk of colorectal cancer (relative risk 1.62; 95% confidence interval: 1.04, 2.53). The SNPs rs1205 and rs1130864 were also individually statistically significantly associated with risk of colorectal cancer in the direction expected from their association with CRP-levels.

Conclusion: In the EPIC study, both measured and genetically raised CRP levels were associated with higher risk of colorectal cancer, supporting the hypothesis that CRP is a risk factor for colorectal cancer.
A randomised controlled trial to evaluate the efficacy of a 6-month dietary and physical activity intervention in prostate cancer patients receiving androgen deprivation therapy

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Androgen Deprivation Therapy (ADT) use in prostate cancer patients is associated with adverse side effects including: changes in body composition; an increase in fat mass and a decrease in muscle mass, increased fatigue and a reduced quality of life (QoL). The aim of this study was to test the efficacy of a combined 6-month diet and physical activity intervention to reduce the side effects associated with ADT.

Prostate cancer patients on ADT were randomly assigned to either an intervention arm to receive individualised healthy eating advice plus physical activity advice (to achieve 30 minutes of brisk walking per day, 5 or more days per week) (n=47) or a standard care (n=47). Baseline, 3 month and 6 month assessments included body mass index (BMI), percentage fat mass, waist circumference, functional capacity (6 minute walk test), fatigue, QoL, and perceived stress scores.

At 6 months, weight, BMI and percentage fat mass decreased significantly (p<0.001) in the intervention arm compared with the control arm; the between group differences (adjusted for baseline values) were -3.3kg (95% CI -4.5, -2.1), -1.1kg/m² (95% CI -1.5, -0.7) and -2.1% (95% CI -2.8, -1) respectively. Waist circumference decreased and functional capacity increased in the intervention group compared to the control group; the between group differences were -3.3cm (95% CI -4.6, -1.9) (p=0.009), +36.5m (95% CI 14.5, 58.4) (p <0.001). Improvements in fatigue, QoL and stress scores were also shown but were not statistically significant. This intervention was beneficial in terms of reducing the adverse body composition changes and functional decline associated with ADT.

Randomised controlled trial of diet, physical activity and breast cancer recurrences: the DIANA-5 study.

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Background: Western diet and low levels of physical activity are associated with metabolic syndrome, insulin resistance and high serum levels of sex hormones and growth factors. These factors are strongly related with breast cancer (BC) risk and prognosis. Previous trials showed that an insulin lowering diet significantly decreases body weight, serum testosterone, insulin, and the bioavailability of IGF-I, in both healthy and BC women. Consistently, observations suggested that physical activity and weight reduction may help preventing both BC and BC recurrences.

Methods: The DIANA-5 study is a multicenter randomized dietary and physical activity intervention trial to reduce BC recurrences in BC patients at high risk because of high serum testosterone (>0.4 ng/ml), or insulin (>50 pmol/L), or the presence of metabolic syndrome.

Results: Among 2256 BC patients recruited, 1605 were randomized into an intervention (804), and a control group (801). The control group received general recommendation for the dietary prevention of cancer (the 2007 WCRF Decalogue), while the intervention group received an active support, through kitchen courses, physical activity classes, and common meals. Preliminary comparisons on the 1091 women who have already completed the one year intervention (574 in the intervention and 517 in the control group), showed a significant difference for waist circumference (-2.9 cm of reduction in the intervention vs -1.2 cm in the control, p<0.01), body weight (-2.5 Kg vs-1.0 Kg, p<0.01), total cholesterol (-8.2 mg/dl vs -4.8 mg/dl, p=0.04) and triglycerides (-16.9 mg/dl vs -8.8 mg/dl, p=0.03).

Conclusions: Preliminary results suggest the effectiveness of the intervention.
Physical activity and gastric cancer risk: a meta-analysis.
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**Aim:** This meta-analysis aims to evaluate the association between physical activity and risk for gastric cancer.

**Methods:** Potentially eligible articles were sought in PubMed with a predefined search algorithm; end of search date was set at February 08, 2013 and no language restrictions were adopted. Two authors working independently performed the selection of studies and data extraction. The highest versus lowest exposure categories of physical activity were extracted from each study. Random-effects models (DerSimonian-Laird) were implemented for the calculation of the pooled relative risk (RR) estimates. Separate analyses were performed by gender, type of physical activity (leisure; occupational; overall) and location of gastric cancer (cardia, noncardia). Analysis was performed with STATA 11.1 statistical software.

**Results:** Among 188 screened abstracts, 17 studies were deemed eligible (nine case-control, eight cohort studies). Physical activity exerted a protective role (pooled RR=0.80, 95%CI: 0.72-0.89). At the gender-specific analyses, the effect was prominent among females (pooled RR=0.69, 95%CI: 0.50-0.95), whereas the protective trend did not reach significance among males (pooled RR=0.91, 95%CI: 0.81-1.03). Subanalyses by type of exercise were hampered by low statistical power and did not point to statistical significant associations. The protective effect regarding non-cardia carcinomas was particularly sizeable (pooled RR=0.62, 95%CI: 0.52-0.75), whereas the effect concerning cardia cancers was marginal (pooled RR=0.80, 95%CI: 0.64-1.01).

**Conclusions:** The protection offered by physical activity in terms of gastric cancer risk seems sizeable, particularly among females and non-cardia lesions.

Obesity and risk for gliomas and meningiomas: a meta-analysis
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**Aim:** This meta-analysis aims to evaluate the association between overweight as well as obesity and risk for gliomas and meningiomas, taking into account the modifying role of gender.

**Methods:** Potentially eligible articles were sought in PubMed with a predefined search algorithm; end of search date was set at February 08, 2013 and no language restrictions were adopted. Two authors working independently performed the selection of studies and data extraction. Overweight and obesity were defined as body mass index (BMI) 25.0–29.9, and >30 kg/m², respectively. Random-effects models (DerSimonian-Laird) were implemented for the calculation of the pooled relative risk (RR) estimates; separate analyses were performed by gender. Analysis was performed with STATA 11.1 statistical software.

**Results:** Among 359 screened abstracts; nine studies were deemed eligible (four case-control and five cohort studies). Increased risk for meningioma was observed among obese females (pooled RR=1.49, 95%CI: 1.25-1.77) but not males. Overweight was not associated with increased meningioma risk in either gender (pooled RR=1.09, 95%CI: 0.96-1.23 for females and pooled RR=1.65, 95%CI: 0.54-5.03 for males). Regarding glioma, there was scarcity of data at the gender-specific analyses. Nevertheless, overweight seemed to be associated with increased glioma risk among females (pooled RR=1.25, 95%CI: 1.02-1.53, data stemming only from two studies); however, the respective finding was not reproduced upon obese status or males.

**Conclusions:** Obesity in females represents a risk factor for meningiomas, whereas further studies seem to be needed regarding gliomas.
Impact of resistance training on muscle strength, body composition, and fatigue in cancer: a meta-analysis

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Purpose: Current evidence suggests many health benefits from physical activity during and post cancer treatment. However, the optimal exercise programme for cancer survivors has not yet been established. The purpose of this meta-analysis is to summarise evidence for the efficacy of a resistance training (RT) intervention to improve muscle strength, body composition, and fatigue among adult cancer survivors.

Methods: A systematic literature review of the Cochrane Trial Register, MEDLINE, and EMBASE literature databases was undertaken. Studies were included if they were randomised controlled trials (RCT) comparing RT with an exercise or non-exercise control group in cancer survivors during and post treatment. 15 RCTs met our inclusion criteria. We performed a random-effects meta-analysis to determine weighted-mean differences (WMD) with 95% confidence intervals. A random-effects meta-regression model was performed in order to examine dose-response relationships between intensity, duration and frequency of RT and assessed outcomes.

Results: Quantitative evidence shows a large effect of RT on lower-limb and upper-limb muscle strength (WMD: +15.52 kg, p=0.0002 & +7.34 kg, p<0.0001, respectively) and moderate effects on lean body mass and percent body fat (WMD: +1.07 kg, p<0.0001 & -2.08%, p=0.003, respectively). A small positive effect of RT was noted in FACT-fatigue (p=0.05). Upper-limb muscle strength and percent body fat improved to a greater extent when RT interventions were of low to moderate-intensity (<70%1RM, p=0.02) or the RT intervention was of shorter duration (p=0.053).

Conclusions: RT was shown to be associated with clinically important positive effects on muscular function and body composition in patients during treatment or in long-term follow-up.

miRNome analysis in high-fat diet-Induced hepatocarcinoma

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Obesity is one of the main public health problems, with increasing prevalence in both adults and children, even in developing countries. Epidemiological studies reveal a strong link between obesity and development of various tumours, including liver cancer. Obesity is associated to liver diseases such as non-alcoholic fatty liver disease (NAFLD) and the more severe non-alcoholic steatohepatitis. MicroRNAs (miRNAs) are endogenous small non-coding molecules able to regulate the expression of target genes at the post-transcriptional level. Accumulating evidences reveal the important role of the disturbance of miRNAs expression and functions in liver cancer. In this study, we used a mouse model predisposed to diet-induced obesity and NAFLD to investigate the effects of a high-fat (HF) diet in the transition from steatosis to hepatitis, cirrhosis and then hepatocarcinoma initiation and progression. C57BL6/J mice were fed with a HF diet for different time ranges (3, 6, 12, 18 months). Animals were sacrificed and the hepatic tissues collected. After conventional histological analysis, RNA was extracted and the miRNome was analyzed. Several miRNAs resulted differentially expressed in hepatic tissues from HF-fed mice. Among these, miRNAs 125a-5p and 199a-3p were down-regulated whereas miRNAs 411 and 139-3p were up-regulated after 3 and 6 months of treatment. In addition, some miRNAs (miRNA 143, 182, 195, 200, 214, 497) resulted differentially expressed (i.e. up-regulated in 3 months and down-regulated in 6 months-treated mice, or inversely). Further experiments are in progress to complete the evaluation of the effects of currently ongoing time points on hepatic injury progression and hepatocarcinoma onset.
Lifestyle during and after prostatic radiotherapy influences the risk of late toxicity

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Background: More men are surviving prostate cancer yet living with the late adverse effects of treatment. Although improvements in radiotherapy techniques are the reducing risks of late side effects, little is known about the additional benefits of lifestyle factors particularly physical activity and obesity.

Methods: This retrospective study evaluated the entire cohort of alive men treated with radical radiotherapy between 2000-2010 at Addenbrooke’s Hospital, referred via the Bedford Hospital pathway (n=470). 440 (94%) completed a questionnaire consisting of the Vaizey Rectal Toxicity score, the NCI common toxicity scores for rectal bleeding, erectile function and urinary incontinence; A General Practical Physical Activity Questionnaire. The effect of each lifestyle criteria on late toxicity was investigated using a non-parametric ANOVA (Kruskal-Wallis) test.

Results: 7.5% men smoked during their radiotherapy. At the time of the survey, 63% were overweight or obese (BMI >25); 58% were inactive, 27% moderately inactive and 15% active. Active men had lower rectal toxicity and better erectile (p<0.001) and urinary function (p<0.001). Men smoking >5/day had worse rectal toxicity (p<0.01) as did overweight men (p<0.05). There was no correlation between PSA relapse and lifestyle.

Conclusions: This is the first comprehensive evaluation of lifestyle habits during and after radical radiotherapy for prostate cancer. In this large cohort, most men were inactive and overweight although few smoked. Although a retrospective analysis, the data strongly suggests lower late toxicity among non-smokers, individuals who exercise regularly and who were not overweight. We recommend that men should receive written information, lifestyle counseling before and after radiotherapy and if necessary referral to smoking cessation clinics, local gyms and dieticians.

Participation, adherence and weight loss: results of a feasibility study involving a diet and physical activity intervention in breast cancer survivors

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1Unit of Nutrition, Environment and Cancer; Catalan Institute of Oncology (ICO-IDIBELL), Barcelona, Spain, 2Department of Physiological Sciences II, School of Medicine; University of Barcelona, Barcelona, Spain, 3Clinical Nutrition Unit, Catalan Institute of Oncology (ICO-IDIBELL), Barcelona, Spain

Background and Objectives: Observational and experimental evidence suggests that energy restriction from a low-calorie diet and increased energy expenditure induced by physical activity (PA) could promote weight loss/maintenance and be a determinant of breast cancer (BC) prognosis. The aim of the present study was to assess the feasibility of a diet and PA intervention designed to induce weight loss in BC survivors.

Methods: The intervention of this 12-week single-arm pre-post feasibility study involved group-based sessions: one-hour weekly diet sessions and 75-minute bi-weekly PA sessions of moderate-to-high intensity. The study targeted overweight and obese women, aged 18 to 75, who had recently completed chemotherapy and/or radiotherapy for a non-metastatic BC. Before and after the intervention, anthropometry, dietary information, quality of life (QoL) and cardiorespiratory fitness (CRF) were collected.

Results: A total of 113 BC survivors were identified and invited to participate and 42 of them started the intervention. The 37 participants who completed the intervention attended more than 90% of the sessions offered, and showed a significant weight loss of 5.62±2.04kg as well as significant decreases in fat mass and waist and hip circumferences. At the end of the intervention, participants had significantly decreased their total energy intake, reducing in particular fat and saturated fat intakes and increased their QoL and CRF.

Conclusions: This study demonstrated the success of a short-term intervention based on diet and PA to induce weight loss and promote healthful changes in BC survivors. Further study is needed to determine the impact of this lifestyle change on BC prognosis.
Qualitative analysis of breast cancer survivors’ perspectives on a diet and physical activity-based weight loss intervention

van Patten C.L.¹, Balneaves L.G.², Truant T.L.O.², Neil S.E.³, and Campbell K.L.⁴

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Purpose: To explore the experiences of overweight/obese breast cancer survivors who participated in a weight loss intervention and identify areas of improvement and future research.

Methods: Participants (n=14; BMI=30.1; age=54.6 years) completing a 24-week group-based weight loss intervention were invited to participate. Six women participated in a 1-hour focus group and three women participated in individual 1-hour interviews. Interviews were transcribed verbatim and a thematic analysis was conducted.

Results: Women valued the camaraderie of other cancer survivors. Participants praised the knowledgeable facilitators and developed new skills and knowledge related to adopting a healthier lifestyle for both themselves and family. The nutrition component challenged women’s dietary beliefs and habits, and shifted perspective of being on a diet to a healthy “way of eating”. Women reported increased strength and stamina related to the physical activity component regardless of whether they met prescribed weight loss targets. Women identified several positive psychological benefits: feeling more in control, less anxious and distressed and a renewed sense of confidence and independence. Challenges included logistics of attendance, fatigue and negative responses from their social network. Women found the length of the intervention suitable but expressed uncertainty of how to independently maintain their progress.

Conclusion: Women valued the group-based intervention that validated their cancer experience and provided a safe and supportive environment. The intervention was unanimously recommended to all breast cancer survivors post-treatment as part of standard care. Changes recommended included the involvement of patient’s partners as well as ongoing follow up for sustainability of positive lifestyle changes.

Is a cancer diagnosis a trigger for health behaviour change? Findings from a prospective, population-based study

Williams K.¹, Steptoe A.², and Wardle J.¹

¹Health Behaviour Research Centre, Department of Epidemiology and Public Health, University College London, London, UK ²Psychobiology Group, Department of Epidemiology and Public Health, University College London, London, UK

Background: Health behaviour change following cancer can improve long-term outcomes. No studies have examined health behaviour change among cancer survivors in the UK, or tracked behaviours over multiple time-points in survivors and controls. We assessed changes in smoking, alcohol and physical activity at three times (0-2 years before a cancer diagnosis, 0-2 years post-diagnosis and 2-4 years post-diagnosis) and at matched times in a comparison group.

Methods: Data were from waves 1-5 of the English Longitudinal Study of Ageing; a cohort of older adults in England. Smoking, alcohol and physical activity measures were taken at each wave. Generalised estimating equations were used to examine differences by diagnosis, change over time, and group-by-time interactions.

Results: Of the 5146 adults included in the analyses, 433 (8.4%) were diagnosed with cancer. The latter were less likely to be physically active (p<0.01) and more likely to be sedentary (p<0.001). There were no differences in alcohol or smoking. Interactions were not significant for smoking (p=0.17), alcohol (p=0.20), physical activity (p=0.17) or sedentary behaviour (p=0.86), suggesting that cancer survivors were no more likely to make changes than the comparison group.

Conclusion: Our findings provide little evidence that a cancer diagnosis motivates health-protective changes. Given that healthier lifestyles are important for long-term outcomes, effective support for behaviour change in cancer survivors needs to be identified.
Concordance with WCRF/AICR cancer prevention recommendations and colorectal tumors in Lynch Syndrome (hereditary colorectal cancer)


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Background: In 2007, WCRF/AICR published public health recommendations for cancer prevention. The role of lifestyle in hereditary cancer prevention has only sparsely been studied. Individuals with Lynch Syndrome (hereditary colorectal cancer) have mutations in specific mismatch repair genes. Approximately 1-3% of all colorectal cancer cases are caused by Lynch Syndrome.

Aim: The aim of our study was to assess the association between concordance with WCRF/AICR-recommendations and colorectal tumours in Lynch Syndrome carriers.

Methods: Within the GeoLynch study - a prospective cohort of 449 persons with confirmed Lynch Syndrome - data on diet and lifestyle were obtained from food frequency and general questionnaires between 2006 and 2008. From those data, a combined score was constructed for concordance with WCRF/AICR recommendations on weight management, level of physical activity, consumption of energy-dense foods, plant foods, red and processed meat, alcohol, and use of supplements. The total score ranged from 0 (no concordance) to 7 (full concordance). During a median follow-up of 3.2 years, 128 colorectal tumours occurred. Cox regression was used to calculate hazard ratios.

Results: Higher concordance with the recommendations was associated with lower risk of colorectal tumours.

Conclusion: A healthy lifestyle may also be beneficial for persons with an inherited high risk of colorectal cancer, as it may decrease their risk of colorectal tumours.

Excessive energy intake and cancer risk


1Epidemiology Unit, Centro di Riferimento Oncologico Aviano (Italy) 2University of Udine (Italy) 3Istituto di Ricerche Farmacologiche Mario Negri, Milan (Italy)

Excessive energy intake is related to obesity and to free-radicals formation, two conditions that have been shown to increase cancer risk. We evaluated the association between excessive energy intake and cancer risk in an Italian network of case-control studies on 12 cancer types. Cases were 10,753 patients with incident, histologically confirmed, cancer. Controls were 24,245 cancer-free patients matched to cases by sex, age, and place of living. Daily Energy Requirement (DER) was estimated from height, ideal weight, sex, age, and self-reported physical activity. Caloric Balance (CB) was calculated as the difference between daily energy intake (estimated from dietary habits in the years preceding enrolment, assessed through a food-frequency questionnaire) and DER. Odds ratios (OR) and confidence intervals (CI) for excessive energy intake (i.e., CB≥500 kcal/day) compared to proper one (i.e., -150≤CB<150 kcal/day) were calculated using logistic regression model, adjusted for relevant confounders.

Cancer cases reported significantly higher CB than controls (median=243 and 97 kcal/day, respectively; Wilcoxon p<0.01). Excessive energy intake was associated to increased risk of cancers of the larynx (OR=1.83; 95% CI: 1.27-2.63), colon (OR=1.33; 95% CI: 1.12-1.58), rectum (OR=1.32; 95% CI: 1.06-1.63), postmenopausal breast (OR=1.21; 95% CI: 1.01-1.49), pancreas (OR=1.52; 95% CI: 1.01-2.38), and prostate (OR=1.39; 95% CI: 1.07-1.82). No significant associations emerged for cancers of the head and neck, oesophagus, nasopharynx, endometrium, ovary, and kidney.

Study results highlight the importance of an appropriate CB in the prevention of several cancers. Therefore, monitoring daily energy intake and increasing physical activity should be strongly recommended.
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BODY FATNESS
Be as lean as possible within the normal range of body weight.

PHYSICAL ACTIVITY
Be physically active as part of everyday life.

FOODS AND DRINKS THAT PROMOTE WEIGHT GAIN
Limit consumption of energy-dense foods and avoid sugary drinks.

PLANT FOODS
Eat mostly foods of plant origin.

ANIMAL FOODS
Limit intake of red meat and avoid processed meat.

ALCOHOLIC DRINKS
Limit alcoholic drinks.

PRESERVATION, PROCESSING, PREPARATION
Limit consumption of salt and avoid mouldy cereals (grains) or pulses (legumes).

DIETARY SUPPLEMENTS
Aim to meet nutritional needs through diet alone.

BREASTFEEDING
Mothers to breastfeed; children to be breastfed.

CANCER SURVIVORS
Follow the recommendations for cancer prevention.

AND, ALWAYS REMEMBER...
Do not smoke or use tobacco in any form.

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The aim of the Stock Conferences is to bring together world leading experts to discuss a focused topic related to obesity.

The 12th Stock Conference will focus on the topic of Functional Body Composition and Related Aspects in Research on Obesity and Cachexia.

Attendance for this event is limited and places are allocated on the basis of competitive entry.

Application submission deadline:
Friday 5th July 2013

View the scientific programme overleaf

For more information please contact:
stock@iaso.org  www.iaso.org/12th-stock-conference-2013
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<td>08:45</td>
<td>Introduction and Welcome</td>
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<td>Conference Chairs:</td>
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<td>Manfred J. Müller</td>
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<td>Steven Heymsfield</td>
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<td>09:00</td>
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<td>Kevin D Hall</td>
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<td>A model of functional body composition in humans</td>
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<td>Abdul Duloo</td>
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<td>Regulation of body composition. Body components - brain feedbacks in weight control</td>
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<td>Steven Heymsfield</td>
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<td>What is an appropriate energy expenditure for body composition?</td>
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<td>12:30</td>
<td>Lunch</td>
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<td>Session II: Functional Aspects of adipose tissue and muscle</td>
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<td>John Speakman</td>
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<td>Effects of over- and under-nutrition on body composition and metabolism of the mouse – what goes up doesn’t necessarily come down</td>
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<td>14:30</td>
<td>Anja Bosy-Westphal</td>
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<td>Functional correlates of fat mass-fat free mass relationships during under- and refeeding</td>
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<td>16:00</td>
<td>Jürgen Eckel</td>
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<td>Skeletal muscle crosstalks in response to exercise and nutrition.</td>
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<td>Session III: Obesity</td>
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<td>Jim D Bell</td>
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<td>Phenotyping adiposity - the metabolically healthy obese patient</td>
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Track 3 – From healthy weight to weight-related pathologies
Body composition, fat distribution (including ectopic), imaging, regional and ethnic differences, diabetes, cancer, NAFLD, OSA/sleep, cardiovascular, osteoarthritis, lifespan issues, gender differences, obesity assessment (BMI for age), biomarkers

Track 4 – From nutrition, exercise and psychology to lifestyle
Food environment (food availability, affordability, safety), macronutrients, intake (regional, gender), CBAs, physical activity environment (availability, affordability, safety), exercise (type and duration, metabolism), psychology and behaviour issues (depression, microstructures of feeding), measurement and patterns of diet and physical activity, sedentary behaviour, energy balance

Track 5 – From lifestyle intervention to drugs and surgery
Diet, exercise, behaviour, psychology, sleep, VLEDs, novel pharmacotherapy, multidisciplinary, state and role of bariatric surgery, lifespan issues, new surgical approaches and devices, e-technology, biomarkers, health economics

Track 6 – From home environment to society: causes and consequences
Home environment and equity, prenatal and early nutrition, epidemiology, inequalities, habitat, marketing, workplace, school, role of industry, health economics

Track 7 – From individual choice to population prevention: solutions and interventions
Primary prevention, interventions (community, workplace, school), disease prevention programmes, personalised nutrition, health services, e-technology

Track 8 – From evidence to policy
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