HOT TOPIC CONFERENCE:

Obesity, Physical Activity & Cancer:
Life course influences and mechanisms
Dear Colleague

We are delighted to welcome you to London for this conference jointly organised by World Cancer Research Fund International and World Obesity Federation. This is a follow-up to the highly successful first OPAC conference we organised together in 2013, and we’re confident you will find this year’s programme equally inspiring and informative.

This year’s topics include several sessions on the mechanisms underpinning the links between obesity and physical activity, and cancer risk and survival. Other sessions look at how early life events are linked to obesity and cancer susceptibility in later life as well as best practice for turning scientific findings into effective policy actions. Future research needs will be shared and discussed throughout.

We know how useful it is for scientific, health and policy professionals working in these areas to come together to share latest findings and take advantage of the excellent networking and collaborative opportunities. We encourage you to take part in the audience and panel discussions, which are always such a prominent feature of this conference.

On behalf of both our organisations we would like to thank all our invited speakers and hope that you enjoy the conference and are also able to enjoy some time in London while you are here.

With best wishes.

World Obesity Federation & World Cancer Research Fund International
**World Obesity**

**Address:**
World Obesity Federation, Charles Darwin 2, 107 Gray’s Inn Road, London, WC1 X8TZ

**Contact:**
Natasha Joyner
enquiries@worldobesity.org

World Obesity Federation represents professional members of the scientific, medical and research communities from over 50 regional and national obesity associations. Through our membership we create a global community of organisations dedicated to solving the problems of obesity.

Our mission is to lead and drive global efforts to reduce, prevent and treat obesity. We collate, conduct and disseminate world-leading research into obesity, its impact, causes, treatment and prevention. We influence policy of academics, government and business at global, regional and national levels. We bring rigour, consistency and credibility to the field through educational programmes, practical training, publications, conferences and accreditation. World Obesity offers an internationally recognised online obesity education programme for health professionals, providing evidence-based content developed by leading obesity experts.

www.worldobesity.org

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**World Cancer Research Fund International**

**Address:**
World Cancer Research Fund International, Second floor, 22 Bedford Square, London WC1B 3HH

**Contact:**
international@wcrf.org

World Cancer Research Fund International is the world’s leading authority on cancer prevention research related to diet, weight and physical activity. Our vision is to live in a world where no one develops a preventable cancer.

We fund high-quality scientific research into cancer prevention and survivorship and apply it in practical ways to empower people all over the world to take steps to reduce their cancer risk.

We lead and unify a network of cancer charities with a global reach. These cancer charities are based in Europe, the Americas and Asia, giving us a global voice to inform people of cancer prevention.

We influence policy at the highest level and are trusted advisers to governments and to other official bodies from around the world.

World Cancer Research Fund International has official relations status with the World Health Organization (WHO).

Visit our Twitter account https://twitter.com/wcrfint and Facebook account https://www.facebook.com/wcrfint to find out more about our work.

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**American Institute for Cancer Research (AICR)**

In 1982, American Institute for Cancer Research (AICR) was founded to advance the simple but then radical idea that cancer could be prevented. AICR has funded over $105 million in research on food, nutrition and physical activity, and the prevention, treatment and survival of cancer. AICR works with its partners in the WCRF network to interpret the results of its research, along with findings from the global scientific community, and craft reliable, evidence-based recommendations for lower cancer risk. For more information on AICR, please visit www.aicr.org.

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**World Cancer Research Fund UK (WCRF UK)**

For the past 25 years, World Cancer Research Fund UK has been the UK’s leading charity dedicated to the prevention of cancer through diet, weight and physical activity. By funding and supporting research, developing policy recommendations and providing health information, we have ensured that people can make informed lifestyle choices to reduce their risk of developing a preventable cancer. As we look forward to our next 25 years, our scientific research ensures that we will continue to have the latest and most authoritative information at our fingertips, all underpinned by independent expert advice.

Our analysis of global research shows that a third of the most common cancers are preventable through a healthy diet, maintaining a healthy weight and regular physical activity.

For more information visit www.wcrf-uk.org
follow us on Twitter at http://twitter.com/wcrf_uk or visit our Facebook page at http://www.facebook.com/wcrfuk

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**Wereld Kanker Onderzoek Fonds (WKOF)**

Wereld Kanker Onderzoek Fonds (World Cancer Research Fund Netherlands) has been since 1994 the leading cancer charity in the Netherlands dedicated to cancer prevention through a healthy diet and lifestyle. As part of the global World Cancer Research Fund network we envision a world where no one develops a preventable cancer.

Wereld Kanker Onderzoek Fonds stimulates and funds research into understanding the links between diet, nutrition, physical activity, body composition and cancer. Through our health information programmes we raise awareness for cancer prevention and help people make healthy choices in their diet and lifestyle.

Our driving force: cancer prevention, together we can.

For more information please visit www.wkof.nl
# SCIENTIFIC PROGRAMME

## Thursday 1 September

<table>
<thead>
<tr>
<th>Activity</th>
<th>Start Time</th>
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<tr>
<td><strong>Registration</strong></td>
<td>07:45</td>
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<tr>
<td><strong>Welcome, introductions and aims – Chair and WCRF International/World Obesity Federation</strong></td>
<td>09:15</td>
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<tr>
<td><strong>Session 1. Overview of obesity, physical activity and cancer risk: what do we know and what don’t we know? Chair: Alan Jackson (UK)</strong></td>
<td>09:30</td>
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<tr>
<td><strong>Topic 1:</strong> The role of adiposity (including weight change) on cancer risk: epidemiology &amp; molecular mechanisms – Marc Gunter (France)</td>
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<td><strong>Topic 2:</strong> The role of physical activity on cancer risk: epidemiology &amp; molecular mechanisms – Michael Leitzmann (Germany)</td>
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<td><strong>Topic 3:</strong> Adiposity (including weight change), physical activity, energy balance and cancer risk: mechanistic evidence from preclinical models – Stephen Hursting (US)</td>
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<td>Facilitated discussion between keynoters and audience.</td>
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<tr>
<td><strong>REFRESHMENTS + Poster session</strong></td>
<td>11:30</td>
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<td><strong>Session 2. Setting out methodological issues and novel approaches</strong></td>
<td>12:00</td>
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<td><strong>Chair: Rudolf Kaaks (Germany)</strong></td>
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<td><strong>Topic 1:</strong> Anthropometry: what can we measure and what does it mean? – Anne McTiernan (US)</td>
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<td><strong>Topic 2:</strong> Novel tools in next-generation epidemiological studies for assessing adiposity and physical activity in cancer research: – Part 1: Imaging approaches – Jimmy Bell (UK)</td>
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<td>– Part 2: Metabolomic biomarker discovery in relation to stratified risk, and personalised medicine – Marc Gunter (France)</td>
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<tr>
<td>Facilitated discussion between keynoters and audience.</td>
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<tr>
<td><strong>LUNCH + Poster session</strong></td>
<td>13:00</td>
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<tr>
<td><strong>Session 3. Reviewing mechanistic evidence on cancer risk and survival</strong></td>
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<td><strong>Chair: Richard Martin (UK)</strong></td>
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<td><strong>Topic 1:</strong> Novel method for reviewing mechanistic evidence on diet, nutrition (including body composition) and physical activity and cancer – Sarah Lewis (UK)</td>
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<td><strong>Topic 2:</strong> Feasibility study on postmenopausal body fatness and breast cancer – Part 1 – Eline van Roekel (The Netherlands)</td>
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<td>– Part 2 – Renée Turzanski Fortner (Germany)</td>
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<td>Facilitated discussion between keynoters and audience.</td>
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**Session 4: Dedicated session for oral abstract presentations** Chair: Martin Wiseman (UK)

**Topic 1:** The effects of caloric restriction on molecular mechanisms related to tumorigenesis in the colon of C57BL/6J mice – Dieuwertje Kok (The Netherlands)

**Topic 2:** The Novel Adipokine Omentin, Obesity and Colorectal Cancer: New Insights from the EPIC - Potsdam Cohort Study – Krasimira Aleksandrova (Germany)

**Topic 3:** A study on effect modification by mTOR/PI3K/Akt gene variants of associations between body size, physical activity, early life energy restriction and colorectal cancer risk – Colinda C. J. M. Simons (The Netherlands)

**Topic 4:** Energy balance-related factors and breast cancer risk in pre- and post-menopausal women – Doris S. M. Chan (UK)

**REFRESHMENTS + Poster session** 16:00

**Session 5: How do early life events impact the adiposity/physical activity-cancer relationship? Co-Chairs: Jennifer Baker (Denmark)**

**Topic 1:** Early life events and obesity risk: epidemiology – Marjo-Riitaa Jarvelin (UK)

**Topic 2:** Early life events and cancer risk: epidemiology – Jennifer Baker (Denmark)

**Topic 3:** The role of height and energy restriction in youth on cancer risk; exploring mechanisms through molecular epidemiology – Matty Weijenberg (The Netherlands)

Facilitated discussion between keynoters and audience.

**FINISH** 17:40

**DRINKS RECEPTION – Sponsored by WKoF** 18:00

**SPEAKER DINNER – Sponsored by WCRF UK** 19:00
Friday 2 September

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<tr>
<th>Start Time</th>
<th>Scientific Programme</th>
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<tr>
<td>09:00</td>
<td>Registration</td>
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<tr>
<td>09:20</td>
<td>Session 6: Obesity, physical activity and cancer survival: what do we know and what don’t we know? – Matty Weijenberg (The Netherlands)</td>
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<tr>
<td>09:30</td>
<td>REFRESHMENTS + Poster session</td>
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<td>11:00</td>
<td>Session 7: International perspectives on obesity and cancer – Matty Weijenberg (The Netherlands)</td>
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<td>11:30</td>
<td>REFRESHMENTS + Poster session</td>
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<tr>
<td>12:30</td>
<td>Session 8: Dedicated session for oral abstract presentations – Matty Weijenberg (The Netherlands)</td>
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<tr>
<td>13:30</td>
<td>REFRESHMENTS + Poster session</td>
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<td>15:00</td>
<td>Session 9: Policy implications for obesity and physical activity – Matty Weijenberg (The Netherlands)</td>
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<tr>
<td>15:00</td>
<td>Session 10: Closing session – Future research directions and concluding remarks – Matty Weijenberg (The Netherlands)</td>
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LEARNING OBJECTIVES

The latest evidence and future research needs will be shared and discussed on topics including:

- The epidemiology and metabolic mechanisms linking obesity, weight change and physical activity to cancer risk and survival.
- Early life events linked to obesity and cancer susceptibility in later life, including discussion on findings on height and cancer risk.
- Issues and new approaches to assess body composition and metabolic markers of cancer risk and progression.
- A novel method for systematically reviewing mechanistic studies, and results from feasibility studies testing this method exploring mechanisms underpinning the links between postmenopausal body fatness and breast cancer.
- Best practice for translating scientific evidence into policy actions for the prevention of obesity as well as cancer and other non-communicable diseases.
**SCIENTIFIC COMMITTEE & ABSTRACT REVIEWERS**

**Prof. Matty Weijenberg**  
(Chair), Maastricht University  
(The Netherlands)

**Dr Marc Gunter**  
International Agency for Research on Cancer (France)

**Prof. Alan Jackson**  
University of Southampton (UK)

**Prof. Rudolf Kaaks**  
German Cancer Research Center (Germany)

**Prof. Jennifer Baker**  
University of Copenhagen (Denmark)

**Prof. Wendy Demark-Wahnefried,**  
University of Alabama at Birmingham (US)

**Prof. Juan Rivera**  
National Institute of Public Health (Mexico)

**Anne McTeirnan**  
Anne McTeirnan, MD, PhD is a Full Member at the Fred Hutchinson Cancer Research Center and Research Professor at the University of Washington Schools of Public Health and Medicine in Seattle, Washington, USA.

Her research focuses on diet, obesity, exercise, and risk for cancer development and prognosis. She has published more than 390 scientific manuscripts, and the book, Breast Fitness (St. Martin’s Press, 2003). Committee work includes the WCRF/AICR expert panel, the 2008 U.S. Physical Activity Guidelines, IARC, and the American Cancer Society. Dr. McTeirnan’s forthcoming memoir is Starved: A Nutrition Doctor’s Journey from Empty to Full (Central Recovery Press).

**Andrew Renehan**  
Professor Andrew Renehan is professor of cancer studies and surgery at the University of Manchester, and is honorary consultant in Colorectal and Peritoneal Surgical Oncology at the Christie NHS Foundation Trust, Manchester. He is currently the theme research lead for obesity, diabetes and cancer for the Manchester Cancer Research Centre and Farr Institute@HarIC. He is the chair of the international Diabetes and Cancer Research Consortium and the EASD Diabetes and Cancer Study Group. He was one of 21 international scientists in the IARC handbook writing group on Weight control and Cancer, April 2016.

**Eline van Roekel**  
Dr. van Roekel is a postdoctoral researcher within cancer and molecular epidemiology at the Department of Epidemiology at Maastricht University, the Netherlands.

During 2011-2015, she conducted her PhD research on associations of physical activity and sedentary behaviour with quality of life in colorectal cancer survivors. For her PhD thesis, she used data from a new observational study in colorectal cancer survivors that she initiated together with her supervisors at Maastricht University: the Energy for life after Colorectal cancer (EnCoRe) study. During her PhD, she obtained a WCRF fellowship to attend the International Course in Nutritional Epidemiology at Imperial College London, and a travel grant from the Dutch Cancer Society to conduct a 2-month work visit to the University of Queensland in Australia.

Since obtaining her PhD degree in December 2015, dr. van Roekel has continued to work on studying associations of lifestyle factors with quality of life of colorectal cancer survivors within the EnCoRe study. In addition, she aims to develop herself as a molecular epidemiologist by studying biological mechanisms linking associations of lifestyle with cancer etiology and quality of life outcomes in cancer survivors. For this purpose, she performed a work visit to the International Agency for Research on Cancer in Lyon in September 2015 to learn about metabolomics and the application in epidemiological research. In November 2015, she started working on a WCRF-funded project aiming to validate a systematic review framework for integrating evidence from human, animal, and other mechanistic studies on biological mechanisms linking lifestyle with cancer.

**Anne May**  
Dr. Anne May is working at the Julius Center for Health Sciences and Primary Care, University Medical Center Utrecht in The Netherlands. She holds a Master’s degree in Epidemiology and in Human Movement Sciences (with distinction). During her PhD studies she was involved in a randomized controlled trial on effects of exercise in patients with breast, colon and esophageal cancer. She is interested in effects of exercise, in the mediators of the effect and also in methodological aspects related to exercise-oncology research. For the latter she received a personal VENI grant for talented and creative researchers to study a novel research design and methodology for exercise-oncology RCTs (The Netherlands Organisation for Health Research (ZonMw)).

**Proff. Andrew Renehan**  
Andrew Renehan is professor of cancer studies and surgery at the University of Manchester, and is honorary consultant in Colorectal and Peritoneal Surgical Oncology at the Christie NHS Foundation Trust, Manchester. He is currently the theme research lead for obesity, diabetes and cancer for the Manchester Cancer Research Centre and Farr Institute@HarIC. He is the chair of the international Diabetes and Cancer Research Consortium and the EASD Diabetes and Cancer Study Group. He was one of 21 international scientists in the IARC handbook writing group on Weight control and Cancer, April 2016.

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Dr Copson is a member of the nCrI Breast Clinical Studies Group. Professor Kampman studied nutrition and health at Wageningen University, the Netherlands. She teaches on the Nutritional Epidemiology Course at Imperial College London and is a Visiting Lecturer for the MSc course in Nutrition at University College London and the University of Reading.

Jennifer Baker is an Associate Professor at the University of Copenhagen and is affiliated with the Institute of Preventive Medicine in Denmark. Jennifer serves on many scientific committees for Danish and European projects and is the co-chair of the Childhood Obesity Task Force for the European Association for the Study of Obesity.

Jimmy Bell joined the MRC (Imperial College London) as a Group Head. He worked extensively on the application of in vivo MR methodologies for the study of disease development, demonstrating for the first time the importance of gene-environment interaction in obesity. Moved to the University of Westminster establishing the Research Centre for Optimal Health. He has published over 200 peer-reviewed papers and book chapters. His research program aims to define the influence of candidate genes and environmental factors on optimal health and chronic diseases, particularly those associated with obesity and accelerated aging.

João Breda has a PhD in Nutritional Sciences from Porto University. He has a Master’s Degree in Public Health from the Medical Sciences Faculty of the University Nova de Lisboa and an MBA from the European University in Barcelona. Dr Breda is the Programme Manager: Nutrition, Physical Activity and Obesity at WHO Regional Office for Europe and responsible for providing support to the 53 Member States of the WHO European Region on the implementation of the European Charter on Countering Obesity and the Vienna Declaration on Nutrition and Noncommunicable Diseases as well as evaluating their progress implementation. His team is leading for the largest and most comprehensive childhood obesity surveillance mechanisms globally and developed the new European Food and Nutrition Action Plan 2015-2020 and the first European Physical Activity for Health Strategy. In Portugal, João Breda worked as a Public Health Nutritionist at the General Health Directorate and AFS Centre having launched and led for several years the National Platform Against Obesity. Published in scientific journals and presented in national and international congresses, several dozens of papers and also published several original books. He was Researcher and Professor of Nutrition at Universidade Atlântica and Head of Department of the Nutritional Sciences where he developed and implemented the first Nutritional Sciences Bachelor. He also had academic functions at Algave University, Higher School of Agriculture in Coimbra and the Tourism and Hospitality School of Coimbra.

Linda Bauld holds the Cancer Research/ BUPA Chair in Behavioural Research for Cancer Prevention. This role involves providing leadership for CRUK’s cancer prevention initiative and is combined with her position as Professor of Health Policy and Dean of Research at the University of Stirling. She is also Deputy Director of the UK Centre for Tobacco and Alcohol Studies, a URiCRC Centre for Public Health Excellence that covers 13 Universities. Linda has a background in applied policy research and for the past 17 years her research interests have centred on the evaluation of public health interventions. She has conducted studies on drug and alcohol use, inequalities in health and, most notably, on tobacco control and smoking cessation. She is a former scientific adviser on tobacco control to the UK government and recently led the development of an independent alcohol strategy for the UK, ‘Health First!’ Her CRUK role involves working across health behaviours to build capacity in cancer prevention research in the UK and beyond.

Juan Rivera is founding director of the center for research in nutrition and health at the National Institute of Public Health and Professor of Nutrition at the School of Public Health in Mexico. He is member of the National Academy of Medicine, the Latin American Society of Nutrition (SLAN) and the American Society of Nutrition, the WCRI International Policy Advisory Group, the World Obesity/ Public Prevention Steering Committee, the HPLE on Food Security and Nutrition and is President of SLAN.

Dr Copson and Dr Kampman are particularly interested in genetic and modifiable host factors that influence the outcome of early breast cancer. She is a lead researcher on the POSH study, a large national cohort study of young breast cancer patients and principal investigator of several cohort studies exploring body composition in breast cancer. She is principal investigator for many clinical trials in early and advanced breast cancer.

Jennifer Baker was a visiting postdoctoral fellow at the Division of Cancer Epidemiology and Genetics at the National Cancer Institute, USA and a Research Associate at the MRC Centre in Nutritional Epidemiology for Cancer Prevention and Survival (CNC), Department of Public Health and Primary Care, Cambridge University, UK. Dr Mitrou is currently the Director of Research Funding and Science External Relations at WCRI International with main responsibilities to provide strategic direction for the science activities of the WCRI network (UK, HK, NL) and expand WCRI International’s global positioning and influence through strategic partnerships at national and international level.

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Marc Gunter
Marc Gunter is Head of the Section of Nutrition and Metabolism at the International Agency for Research on Cancer (IARC), the specialized cancer research agency of the World Health Organization.

Dr Gunter holds a PhD in molecular epidemiology from the University of Cambridge and a degree in biochemistry from the University of Oxford. He completed his postdoctoral training at the US National Cancer Institute and has held faculty positions at the Albert Einstein College of Medicine in New York and Imperial College London. His research focuses on the role of nutrition and obesity in the natural history of cancer with an emphasis on metabolic dysfunction and in particular the insulin/IGF/mtTOR pathway.

Dr Gunter is principal investigator of a number of studies applying high dimensional metabolic profiling within the framework of large prospective and clinical cohorts to identify novel biochemical pathways involved in cancer development and prognosis. Recent publications include investigations of obesity subtypes defined by metabolic measurements in relation to breast and colorectal cancer risk, adipose tissue derived factors and breast cancer and the role of endogenous oestrogens in colorectal cancer development.

Martin Wiseman
Professor Martin Wiseman is currently an independent nutrition consultant, and Medical and Scientific Adviser to the World Cancer Research Fund International. In addition, he is a visiting professor in human nutrition at Southampton University. 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 2009 companion Policy and Action for Cancer Prevention.

Until 1999 he was head of the Nutrition Unit at the Department of Health, where he had responsibility for nutrition science, including the work of COMA, the Committee on Medical Aspects of Food and Nutrition Policy, for nutrition surveys, and for advising on and implementing nutrition policy.

He qualified from Guy’s Hospital in London in 1975. He became a member of the Royal College of Physicians in 1977 and followed a traditional career path as a general physician until 1981 when he developed an interest in clinical research. He has published papers on diabetes and kidney function and the effects of nutrition on them. He moved to the Department of Health in 1986 but still retains an appointment within the NHS, where he continues with clinical activities in a diabetes clinic.

He was Honorary External Relations Officer of the Nutrition Society from 2001 to 2006, and was Honorary Treasurer of the Association for Research on Cancer (since 2002), also holding a visiting professorship at Brunel University London, UK and a part-time professorship at the University of Oulu, Finland. She is honorary consultant for Imperial College Healthcare NHS Trust, UK. She has been running large-scale population based studies for over 25 years, working on the genetic and early life environmental origins of multi-factorial diseases and disorders, in close collaboration with many international institutions, groups and networks. She is a Scientific Director of the Northern Finland Birth Cohort (NFBC) research programme (about 20 000 subjects, born in 1966 and 1985-86 and their 35,000 parents), and have an active role in research training as Director of Postgraduate Studies at School of Public Health, Imperial College London. Professor Jarvelin has published over 600 original papers and supervised 29 PhD students until completion and many other postgraduate theses. She has received funding from MRC, UK, Welcome Trust UK, ESRC, UK, the EU (FP5, FP7 and Horizon 2020), NIH USA and Academy of Finland among others. She has been nominated on several prestigious visiting and collaboration awards for e.g. China, Russia, Korea, USA, UK and New Zealand. In 2007-8, she received an award of Excellence in Genetic Epidemiology at Imperial College London. In 2012, she was offered the title, Epidemiologist of the Year in Finland and in 2013 was invited to join, as a member, the Finnish Academy of Sciences.

Marijo-Ritaa Jarvelin
Marijo-Ritaa Jarvelin is Professor and Chair in Life-course Epidemiology at Imperial College London, UK (since 2002), also holding a visiting professorship at Brunel University London, UK and a part-time professorship at the University of Oulu, Finland. She is honorary consultant for Imperial College Healthcare NHS Trust, UK. She has been running large-scale population based studies for over 25 years, working on the genetic and early life environmental origins of multi-factorial diseases and disorders, in close collaboration with many international institutions, groups and networks. She is a Scientific Director of the Northern Finland Birth Cohort (NFBC) research programme (about 20 000 subjects, born in 1966 and 1985-86 and their 35,000 parents), and have an active role in research training as Director of Postgraduate Studies at School of Public Health, Imperial College London. Professor Jarvelin has published over 600 original papers and supervised 29 PhD students until completion and many other postgraduate theses. She has received funding from MRC, UK, Welcome Trust UK, ESRC, UK, the EU (FP5, FP7 and Horizon 2020), NIH USA and Academy of Finland among others. She has been nominated on several prestigious visiting and collaboration awards for e.g. China, Russia, Korea, USA, UK and New Zealand. In 2007-8, she received an award of Excellence in Genetic Epidemiology at Imperial College London. In 2012, she was offered the title, Epidemiologist of the Year in Finland and in 2013 was invited to join, as a member, the Finnish Academy of Sciences.

Marty Weijenberg
Marty Weijenberg is a professor of Molecular Epidemiology of Cancer at the department of Epidemiology and the GROW-School for Oncology and Developmental Biology at Maastricht.

Her field of research evolves around the molecular epidemiology of colorectal cancer, specifically the role of nutrition, physical activity, body composition, early life exposures, gene-environment interaction, epigenetics, in the aetiology of colorectal cancer, and more recently, the quality of life and prognosis of colorectal cancer survivors.

Over the past 20 years she has developed a research line on the molecular epidemiology of colorectal cancer which is embedded within a large prospective cohort study, The Netherlands Cohort Study on diet and cancer, and has resulted in a comprehensive molecular databases of colorectal cancer patients from a prospective study. This enables the study of 1) the aetiology of colorectal cancer according to epigenetic characteristics of tumors, 2) gene-environment interactions and risk of colorectal cancer, 3) epigenetic characteristics in a large unselected sample of colorectal cancer tumors, 4) epigenetic markers of tumors and (long-term) prognosis of colorectal cancer cases.

Dr. Weijenberg has an extensive track record in the field of colorectal cancer research and in 2018 she has been awarded a grant to conduct a large study on colorectal cancer in the Netherlands.

Michael Leitzmann
Michael Leitzmann’s main research interests involve the relations of diet, body size, and physical activity to the development of chronic diseases.

He holds an MD from the University of Berlin and an MPH and a DrPH from the Harvard School of Public Health. He currently serves as chair of the Department of Epidemiology and Preventive Medicine at the University of Regensburg, Germany. He was previously appointed as investigator at the U.S. National Cancer Institute. He serves as a member of several journal editorial boards and as an expert reviewer for numerous international research organisation.

Renee Turzanski Fortner
Renee Turzanski Fortner (Ph.D.) is a scientist at the German Cancer Research Center (DKFZ) in Heidelberg, Germany.

Dr. Fortner’s primary research focus is on risk factors for breast, ovarian, and endometrial cancers and integrating novel biomarkers into epidemiologic research. She completed a postdoctoral fellowship at Harvard University with the Nurses’ Health Study (NHS) and NHSII, and has extended that experience to investigations in the European Investigation into Cancer and Nutrition (EPIC) cohort and the Finnish and Northern Sweden Maternity Cohorts in her current position at the DKFZ.

Richard Martin
Richard Martin is currently Professor of Clinical Epidemiology in the School of Social and Community Medicine, University of Bristol. He qualified in Medicine from the University of Nottingham, trained and worked as a GP, and then built on an interest in epidemiology (that was first sparked during his intercalated BMedSci at Nottingham) by doing the MSc in Epidemiology at the London School of Hygiene and Tropical Medicine. He subsequently moved to Bristol to take up a Lecturer post and then a Wellcome Trust Research Training Fellowship in Clinical Epidemiology, and have worked at the School of Social and Community Medicine ever since.

Rudolf Kaaks
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 epidemiologist at the International Agency for Research on Cancer (IARC) in Lyon (1988-2001) where he contributed to the development of the European EPIC project. In 2001 he became head of the Hormones and Cancer Group at IARC. 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.

From 2009 to 2015 he was member of the Board of Scientific Directors of the German National Cohort (NaKo). In September 2015 he became speaker of Research Program C Cancer Risk Factors and Prevention.
Sarah Lewis
I obtained a BSc in Genetics at the University of Sheffield in 1995 and then went on to complete a PhD in Genetic Epidemiology at the University of Manchester in 1999. I then had a series of short postdoctoral positions including a post at the International Agency for Research on Cancer. I joined the School of Social and Community Medicine in January 2004 as a lecturer in Genetic Epidemiology and was promoted to Senior Lecturer in 2009. My research interests are in using Mendelian Randomisation to understand risk factors for cancer and also to identify key nutrients required for in utero development. I am involved in a large UK wide cohort study of diet Ip and palate, and a large birth cohort in which I am looking at the role of nutrition during pregnancy on childhood IQ and behaviour. I co-led a work package on Mendelian Randomisation for the Integrative Cancer Epidemiology Programme, which is funded by Cancer Research UK. I am a principal investigator on a project to develop a framework for systematic reviews of mechanistic studies of nutrition and cancer in collaboration with the World Cancer Research Fund. I also hold a grant from the WCRF to apply the above framework and Mendelian randomization to understanding the role of diet in prostate cancer.

Theresa Marteau
Professor Theresa Marteau is Director of the Behaviour and Health Research Unit in the Clinical School at the University of Cambridge, and Director of Studies in Psychological and Behavioural Sciences at Christ’s College, Cambridge. She studied psychology at the LSE and the University of Oxford. Her research interests include:
- development and evaluation of interventions to change behaviour (principally diet, physical activity, tobacco and alcohol consumption) to improve population health and reduce health inequalities, with a particular focus on targeting non conscious processes
- risk perception and communication particular of biomarker-derived risks, and their weak links with behaviour change
- acceptability to public and policy makers of government intervention to change behavior
She is a Fellow of the Academy of Medical Sciences and the Academy of Social Sciences.

Stephen Hursting
Dr Stephen Hursting is Professor and Director of the Division of Nutritional Biology in the Department of Nutrition at the University of North Carolina (UNC) at Chapel Hill. He is also Professor at the UNC Nutrition Research Institute and a member of the UNC Lineberger Comprehensive Cancer Center. He earned his PhD in nutritional biochemistry and MPh in nutritional epidemiology from UNC-Chapel Hill, and he completed postdoctoral training in molecular carcinogenesis and cancer prevention at the National Cancer Institute (NCI). Prior to joining the UNC faculty in 2014, Dr. Hursting was Professor and Chair of the Department of Nutritional Sciences at the University of Texas (UT) at Austin, the McKee-Love Endowed Chair of Nutritional, Molecular and Cellular Sciences in the UT College of Natural Sciences, and Professor of Molecular Carcinogenesis at the UT MD Anderson Cancer Center (2005-14). He also previously served as Chief of the NCI’s Nutrition and Molecular Carcinogenesis Laboratory Section (1999-2005). His research interests center on diet-gene interactions relevant to carcinogenesis, particularly the molecular and metabolic mechanisms underlying obesity-cancer associations, and the interplay between obesity, metabolism and cancer. Primarily using preclinical models (including human and mouse cell lines and genetically engineered mouse models of cancer) in parallel with human studies, he is currently focusing on the molecular and metabolic changes occurring in response to lifestyle-based (dietary and physical activity), or pharmacologic manipulation of energy metabolism and cell signaling pathways, with emphasis on the insulin/IGF-1 signaling pathways as well as inflammation.

Wendy Demark-Wahnefried
Wendy Demark-Wahnefried, PhD, RD is Professor and Webb Endowed Chair of Nutrition Sciences at the University of Alabama at Birmingham (UAB). Her career in cancer research began at Duke University where she was on faculty for 17 years, then was recruited to MD Anderson, and then to the UAB Comprehensive Cancer Center in 2010 as Associate Director for Cancer Prevention and Control. Her research in nutrition and cancer control has produced >200 scientific publications, and recognition as a Korean Professor of Survivorship and an American Cancer Society (ACS) Clinical Research Professor. Dr. Demark-Wahnefried serves on the ACS Guidelines Panel for Nutrition and Physical Activity, American Society of Clinical Oncology Committees on Cancer Survivorship and Energy Balance, Institute of Medicine’s National Cancer Policy Forum, and others. She has led several diet and exercise intervention trials in cancer survivors including FRESH START and Reach-out to Enhance Wellness (RENEW) in Older Survivors.

The role of adiposity (including weight change) on cancer risk: epidemiology & molecular mechanisms
– Marc Gunter (France)
Adiposity, assessed either by body mass index (BMI) or waist circumference, is now established as a positive risk factor for cancers of the colon and rectum, gastric cardia, liver (hepatocellular carcinoma), gallbladder, pancreas, kidney (renal cell carcinoma), postmenopausal breast, ovary, thyroid, meningioma, multiple myeloma and adenosarcoma of the oesophagus. Obesity is associated with significant metabolic and endocrine abnormalities including alterations in sex hormone metabolism, insulin signalling, and adipokines/ inflammatory pathways. All three mechanisms influence the balance between cell proliferation and apoptosis and have been linked to cancer development in both experimental and observational studies. There is generally convincing evidence that intentional body-weight loss positively affects these key mechanisms; however, it is likely that other, hitherto unrecognised molecular pathways mediate the adiposity-cancer association. In this presentation I will summarize the existing epidemiologic literature on adiposity and cancer development as well as the underlying molecular mechanisms, highlighting areas of uncertainty that require further investigation.

The role of physical activity on cancer risk: epidemiology & molecular mechanisms
– Michael Leitzmann (Germany)
There is strong evidence that physical activity is related to decreased risks of colon, breast, and endometrial cancers, whereas the evidence for a protective effect of physical activity on other cancer sites is limited. There are only sparse data regarding the proportion of cancer occurrences that can be attributed to lack of physical activity, but insufficient physical activity levels have been estimated to cause 9% of breast cancer cases and 10% of colon cancer cases in Europe. Distinguishing the preventability estimates of physical activity from those related to weight control is challenging because body mass is related to both physical activity and to cancer. Physical activity may influence cancer risk through beneficial effects on insulin resistance, sex hormone metabolism, inflammatory cytokines, immune function, prevention of oxidative damage, and upregulation of DNA repair mechanisms. By comparison, effects of physical activity on growth factor concentrations and their binding proteins are less clear. The primary challenges in deriving recommendations for physical activity lie in our lack of knowledge regarding which specific type, intensity, frequency, and duration of physical activity is necessary to reduce cancer risk. Future research should be directed at clarifying the etiologic pathways underlying the relation of physical activity to carcinogenesis, gaining a better understanding of the etiologically relevant time period of exposure to physical activity during life that potentially counters cancer risk; improving methods for assessing physical activity in large-scale population studies; and increasing our knowledge concerning a possible link between physical activity and improved cancer survival.

Energy Balance, Adiposity and Cancer: Mechanistic Evidence from Preclinical Models – Stephen D. Hursting, PhD, MPH (US)
The prevalence of obesity, an established risk factor for many cancers, has increased dramatically over the past 40 years in the US and many other countries. Relative to normoweight cancer patients, obese cancer patients often have poorer prognoses, resistance to chemotherapies and are more likely to develop distant metastases. Recent progress on elucidating the mechanisms underlying the obesity-cancer connection suggests that obesity exerts pleomorphic effects on pathways related to tumor development and progression, and thus there are multiple opportunities for primary to tertiary prevention of obesity-associated cancer. We know from preclinical studies that obesity can impact each of the well-established hallmarks of cancer, and obesity-associated perturbations in systemic metabolism and inflammation, and the interactions of these perturbations with cancer cell energetics, are emerging as the primary drivers of obesity-associated cancer development and progression. Several obesity-related host factors, including components of the stromal and structural components of the tumor microenvironment, are extinct to, and interact with, the intrinsic molecular characteristics of cancer cells (including cancer stem cells), and each will be considered in the context of potential preventive and therapeutic strategies to reduce the burden of obesity-related cancers. Moreover, the impact and underlying mechanisms of obesity reversal via calorie restricted diets, physical activity or surgical interventions in a preclinical breast cancer model will be discussed.

Anthropometry: what can we measure and what does it mean? – Anne McTiernan (US)
Increased body size and adiposity are associated with increased risk for several cancers, and for reduced survival in those diagnosed with cancer. While body size in adults has been operationally defined by the World Health Organization by level of body mass index (BMI, kg/m²), the underlying issue for
This talk will review several measures of overall body composition and specific depots of adipose tissue including: weight, height, BMI, body circumferences, dual-energy X-ray absorptiometry (DXA), computed tomography (CT), and magnetic resonance imaging (MRI). Measurement properties will be discussed, as well as systematic error from self-reported measures. The utility of standard cut-points for measures vs. study-specific categorizations will be presented. Examples from observational studies and clinical trials will be provided.

Adipose tissue can be sampled in humans to examine histology, and to measure cancer-related biomarkers such as adipokines and gene expression. Subcutaneous adipose tissue can be sampled from most areas of the body, while visceral fat has been obtained in conjunction with surgical procedures, and fat within and around muscle and organs is less accessible. Examples from our studies of weight loss procedures, and fat within and around muscle and organs will be given.

Metabolomics is the measurement of small metabolites in biological samples that represent and products of a variety of metabolic and cellular pathways. It is a powerful new approach to capture biomarker information on a range of disease processes as well as host responses to exogenous and endogenous exposures. As an emerging approach used in epidemiologic and clinical studies, metabolomics has the potential to improve cancer risk assessment, screening, diagnosis, prognosis and predictive response to therapy, as well as to provide mechanistic insights. In this presentation I will outline the main concepts underlying the application of metabolomics to population-based study designs with specific reference to risk stratification and personalized interventions and will provide examples from recent and ongoing studies from our group.

Methods: A multidisciplinary team with expertise in informatics, statistics, epidemiology, systematic reviews, cancer biology and nutrition was assembled. A series of 5 one-day workshops took place involving presentations, group work and discussions, along with smaller meetings and research being carried out in the intervening periods. Results: We have developed a two stage framework, the first stage of which is a mechanisms discovery stage, followed by a targeted systematic review of studies on a specific mechanism. In doing this we overcame many challenges including developing an automation tool to deal with the vast amount of data generated in the first stage, developing strategies for assessing the quality, relevance and likelihood of publication bias among animal and cell studies, displaying disparate data and integrating data from a variety of study types. Conclusion: The above template will be available to researchers in the future who wish to conduct or host systematic reviews of the mechanisms which underpin associations between exposures and cancer. Funding: World Cancer Research Fund (WCRF)

Applying and evaluating a novel systematic review framework to integrate mechanistic studies linking body fatness with (postmenopausal) breast cancer: Results from the Maastricht Team – Eline van Roekel (The Netherlands)

Although several mechanisms have been proposed to underpin the body fatness-postmenopausal breast cancer association, no systematic review of mechanistic studies has been performed to date. In this study, we have used the systematic review framework that was developed by WCRF International/University of Bristol to summarize the literature on potential mechanisms linking body fatness to breast cancer (stage 1), and conduct a systematic review of existing mechanistic evidence on the role of the insulin-like growth factor 1 receptor (IGF1R) in linking body fatness to (postmenopausal) breast cancer (stage 2). For stage 1, we performed an extensive literature search to identify studies linking body fatness and/or breast cancer to (breast cancer) mechanisms. The Text Mining for Mechanism Prioritization (TeMMMPa) tool was used to generate a comprehensive overview of potential mechanisms and scores were calculated for the amount of evidence for specific mechanisms. For stage 2, we searched the literature for mechanistic studies linking body fatness to IGF1R, and IGF1R to breast cancer. Two independent reviewers selected articles based on predefined inclusion and exclusion criteria. A total of 105 articles were identified, which were screened for quality, relevance, and likelihood of publication bias. The quality of the included studies (n=35) was assessed by study type-specific risk-of-bias tools and data from different types of studies were synthesized and integrated. Results showed that the current evidence on IGF1R as a potential mechanism linking body fatness to breast cancer is weak and that more mechanistic studies are necessary. We think the framework is the start of a discussion on how to integrate evidence from different research streams and can provide guidance on this process.
Early life events and obesity risk: epidemiology – Marjo-Ritaa Jarvelin (UK)

Childhood and adolescent overweight and obesity, which are leading causes of early type 2 diabetes and cardiovascular disease, have become major public health concerns both in westernized and, more recently, in developing countries. Traditional approaches for the management of obesity have had poor long-term efficacy. Therefore prevention is currently the most promising strategy for controlling the obesity epidemic. However, much research is needed in order to understand better the critical life-course “windows” for effective prevention. Several socio-demographic and anthropometric predictors, as well as several common genetic variants, have been associated with obesity development. Some longitudinal studies have shown a strong association between early infancy weight gain rate or adiposity and childhood as well as adult body weight, fat mass and body mass index (BMI). In Northern Finland Birth Cohort studies we have aimed to build models for the early identification of clinical, socio-demographic and genetic risk factors that predict obesity development. Our extensive studies on childhood overweight/obesity show for the first time that a set of readily available risk factors relating to maternal/child welfare care at birth-paternal BMI, birthweight, gestational weight gain, health behaviour and social indicators are good predictors of child and adolescent overweight/obesity, while currently known genetic variants make a smaller contribution. Results of this research and the related online obesity calculator could easily be implemented after further testing into clinical practice (http://files-good.ibl.fr/childhood-obesity/). An obesity calculator could easily be implemented after further testing into clinical practice (http://files-good.ibl.fr/childhood-obesity/).

The role of height and energy restriction in youth on cancer risk; exploring mechanisms through molecular epidemiology – Matty Weijenberg (The Netherlands)

Adult attained height is an established risk factor for cancer risk at several sites. The most convincing evidence has been reported for postmenopausal breast cancer and colorectal cancer risk. For every 5 cm increase in height, postmenopausal breast cancer risk is reported to be increased by 7% to 11% and colorectal cancer risk is increased by 6% to 11% in women and 4% to 9% in men. Height in itself is probably not causally related to cancer, but rather a consistent marker for one or more factors and/or mechanisms that influence growth in relation to both height and cancer risk. Height is determined in the first 20 years of life by aggregated genetic and environmental components, which determine growth in childhood and adolescence but may also spur neoplastic growth later in life. Although adult attained height is found not to be a likely target for intervention to reduce cancer risk, understanding how height is associated to cancer is essential to expand our knowledge concerning the pathways that lead to cancer development later in life. The similarity of the height-associated risk factors for different cancer sites in different populations suggests that common mechanisms are at play in part environmental mechanisms involved in early life and in part genetically determined mechanisms. Both mechanisms will be discussed in more detail. Specifically, energy restriction in early life and the role of genetic variants in further unravelling the underlying mechanisms linking height to cancer risk.

Impact of weight change on cancer survival: what is the role of body composition pre and post diagnosis? – Ellen Copson (UK)

High body mass index (BMI) is associated with an increased risk of breast cancer in post-menopausal women but poorer outcomes in all age groups. The underlying mechanism is likely to be multi-factorial. Patients with a high BMI tend to present later due to their body habitus. Some studies have also indicated an increased indication of biologically adverse features, including ER negative tumours, in obese patients. Surgical complications occur with a higher incidence in obese patients, potentially delaying systemic therapies, and reports suggest that both chemotherapy and endocrine therapy are less effective in patients with BMIs of 30 kg/m2 or greater. High BMI is generally interpreted as excess adiposity and a World Cancer Research Fund report judged that the associations between BMI and incidence of breast cancer were due to body fatness. However, BMI cannot distinguish lean mass from fat mass, or characterise body fat distribution, and so individuals with the same BMI can have different body composition. Most chemotherapy drugs are dosed according to calculated body surface area (BSA). Patients with a similar BSA or BMI may have wide variations in their distribution of adipose tissue and skeletal muscle (body composition); however few studies have looked at the effect of this on chemotherapy tolerance. Finally, adjunct treatments for breast cancer can themselves result in body composition changes. In this presentation I will discuss data from the Southampton based POSH and CAnDo-2 studies to illustrate the complex effects of body composition on the presentation, treatment and outcome of early breast cancer.

Physical exercise interventions in cancer survivors: effects and methodological issues? – Anne May (The Netherlands)

For many patients, cancer and its treatment are associated with physical and psychological side-effects, including reduced physical fitness and quality of life. Much interest has been devoted to exercise interventions on cancer patients’ quality of life, fatigue, physical functioning and fitness during and after completion of treatment. Recently the Predicting Optimal Cancer Rehabilitation and Supportive care therapy (POCReS) consortium has been established: 34 exercise oncology RCTs are pooled in one database in order to perform meta-analyses based on individual patient data. First results for quality of life will be presented.

Exercise is an intervention that cannot be blinded, which has the possible disadvantage of differential accrual, dropout and randomization to control, and contamination between study groups (mainly non-compliance in controls). In the second part of the presentation, the set-up of the UMBRELLA Fit study will be presented. In this study a new trial design, the cohort multiple Randomized Controlled Trial (mRCT), is applied, which has the potential to overcome the here mentioned disadvantages. In the UMBRELLA Fit study the short- and long-term effects of exercise on quality of life in 146 breast cancer patients participating in the UMBRELLA cohort (University Medical Center Utrecht, NL) are investigated as well as feasibility of the mRCT design in the field of exercise-oncology. *Burriss MA et al. Predicting Optimal, Cancer Rehabilitation and Supportive care (POLARIS): rationale and design for meta-analyses of individual patient data of randomized controlled trials that evaluate the effect of physical activity and psychosocial interventions on health-related quality of life in cancer survivors. J Vet Res 2013 Sep 12:75.

Background: Weight management is recommended after diagnosis in patients with obesity-related cancers. Rationale include that excess body weight is (i) associated with poor prognosis, (ii) increased non-cancer mortality, (iii) prevention of second primary cancers, and (iv) reduced quality of life (QoL). Here, the focus is on prognosis.

Methods: A critical appraisal of the evidence in the prognosis domain was performed, mainly drawing on systematic reviews (search: Pubmed since 2010) in breast, colorectal, prostate and kidney cancers. This was supplemented by a narrative review of the available evidence in other cancers. The findings were interpreted against criteria used by the World Cancer Research Fund (WCRF) to grade evidence, particularly evaluating for susceptibility to confounding where weight exposure was measured, and for potential for bias.

Results: There is a large volume of data, especially for breast cancer, linking elevated body mass index (BMI), measured either pre-diagnosis or < 12 months post-treatment, with increased overall mortality and reduced breast cancer-specific survival. Findings for the other cancers were mixed. There was a paucity of studies where BMI was measured during survival (< 12 months post-treatment). In many studies, the possibilities of confounding could not be excluded – for example, many studies lacked data on stage and treatment. Except for ER breast tumours, few analyses demonstrated specificity of association.

Conclusions: Much of the evidence underpinning the rationale for weight management after cancer diagnosis is WCRF grade ‘limited suggestive’. This interpretation challenges many contemporary commentaries. Long-term oncological outcomes are awaited from a small number of cancer-specific trials assessing the impact of weight management.
Weight loss interventions in cancer survivors – Wendy Demark-Wahnefried (US)

Consensus now exists that obesity is a risk factor for nine different cancers, therefore, many cancer patients present at the time of diagnosis and often gain even more weight with various cancer therapies. Given that obesity is a poor prognostic factor for roughly 15 different cancers and also increases the risk for common comorbidities, such as cardiovascular disease and diabetes, the American Society of Clinical Oncology (the largest organization of oncologists in the world) issued a position statement on obesity, encouraging practitioners to reinforce weight management in this patient population.

While some of the lessons learned in conducting weight loss interventions in the general population can be applied to cancer survivors, survivors have several issues (many related to cancer and its treatment) which call for modified approaches to weight management. Moreover, some of the more compelling research questions call for focused energy balance research among cancer survivors. Examples of such research questions are as follows: Will negative energy balance (either caused by caloric restriction, increased physical activity or both) result in improved cancer control, as well as other key outcomes? Does weight control exert similar effects across various neoplasms and are there optimal times to intervene? And, what are key elements of optimal interventions in cancer survivors? Information from previous reviews and findings of more recent clinical trials will be presented to begin to answer these research questions and show where challenges and gaps remain.

IARC Handbook – Weight management and cancer prevention – Rudolf Kaaks (Germany)

In 1995, the IARC Handbooks of Cancer Prevention were launched to complement the IARC Monographs’ evaluations of carcinogenic hazards with evaluations on the scientific evidence on preventive agents and primary and secondary interventions. The working procedures and the evaluation scheme of the IARC Handbooks of Cancer Prevention closely mirror those of the IARC Monographs. Interdisciplinary working groups of expert scientists review the published studies and evaluate the weight of the evidence that an agent or activity can act as cancer-preventive.

In 2002, a first working group to assess the preventive effects of weight control on cancer risk concluded that there was sufficient evidence in humans for a cancer-preventive effect of avoidance of weight gain for cancers of the colon, esophagus (adenocarcinoma), kidney (renal cell), breast (postmenopausal), and corpus-uteri. In April 2016, based on the review of more than 1000 epidemiological studies on cancer risk and excess body fatness, a new working group reaffirmed that absence of excess body fatness lowers cancer risk at the sites previously identified, and additionally identified eight further cancers (gastric cancer, liver, gall bladder, pancreas, ovary, and thyroid, multiple myeloma, mervingoma) for which there is now also sufficient evidence that absence of body fatness lowers cancer risk. For cancer of the breast, endometrium and ovary, the association with excess body fatness was reduced or absent among women using postmenopausal hormone replacement therapy.

In addition to the cancer sites for which there was sufficient evidence, the working group concluded that there is limited evidence for an association between obesity and breast cancer, diffuse large B-cell lymphoma, and male breast cancer. For eight other cancers (lung, esophagus [squamous cell carcinoma], gastric non-cardia, extra-hepatic biliary tract, skin [cutaneous melanoma], testis, uterine bladder, and glioma) the evidence for an association was considered inadequate, due to limited data, inconsistent results, or no data suggesting an association.

Experimental animal studies confirmed that obesity in rodents promotes tumorgrowth and increases the incidence of cancers of the mammary gland, colon, liver, pancreas, prostate (advanced stage), and skin, and to a lesser extent, leukemia. Furthermore, a large number of studies in rodent models showed evidence for a cancer-preventive effect of limitation of body-weight gain by caloric/dietary restriction, for cancers of the mammary gland, colon, liver, pancreas, skin, and pituitary gland, whereas an inverse association was observed between caloric or dietary restriction and cancer of the prostate, lymphoma and leukemia. Regarding mechanisms, the working group identified strong evidence for obesity-related alterations in sex hormone metabolism chronic inflammation in mediating the obesity-cancer relation, and moderate evidence for a role of insulin-like growth factor signaling.

Studies on the effects of controlled weight-loss were sparse. Nonetheless, data on body-weight loss, either from observational studies or from follow-up of patients undergoing bariatric surgery, did suggest that intentional weight loss may reduce cancer risk, notably for breast and endometrial cancer, although the number and quality of these studies was judged insufficient for a formal evaluation.

Increasing Public Acceptability of Policies tackling Obesity – Theresa Marteau (UK)

The causes of obesity are complex but overconsumption of food and sugary drinks is a critical proximal determinant. Evidence is accumulating to show the potential effectiveness of Choice Architecture interventions (Nudging) such as reducing size and availability of less healthy products, and price-based interventions such as increasing the price of sugary drinks. Many of these interventions will require regulation for which public acceptability will be a critical consideration for policy-makers.

Paradoxically, public acceptability is high for the least effective interventions, which are information-based, and lower for Choice Architecture and price-based interventions, which are more effective.

This presentation will outline a possible resolution to this paradox arising from findings from several studies showing that public perception of intervention effectiveness is one of the most reliable predictors of intervention acceptability. This leads to the testable hypothesis that presenting evidence of an intervention’s effectiveness at changing behaviour to tackle obesity increases its acceptability to the public, and in turn, policy-makers.

Implementing science into actions for obesity prevention – Juan Rivera (Mexico)

Mexico is facing an obesity epidemic with prevalences of excess BMI (overweight and obesity) of 70% in adults and about 33% in school-age children and adolescents. The National Public Health Institute in Mexico (IPH) has generated a body of scientific evidence aimed at designing the policy for its prevention and control. Monitoring through periodic National Health and Nutrition Surveys (NHNS) since 1988 provided information about the magnitude, distribution and trends of excess BMI during the last three decades. In less than a quarter of a century, obesity in adult women increased from 9.5% to 35.2%. This dramatic rise alerted the government about the urgent need for interventions. The IPH has conducted the following set of studies aimed at generating information to inform policymaking: a) Monitoring changes in key drivers of obesity through NHNS have been useful to identify shifts in the diet that may be responsible for the increase of obesity b) Formative research allowed recognition of obesogenic elements in different environments and barriers for the adoption of healthy eating and physical activity. c) Randomized efficacy trials have tested effects of specific interventions. d) Estimation of own- and cross-price elasticities of SSB provided justification for a tax on these products. e) The evaluation of policy actions has provided feedback for redesign or improvements. The evidence from these studies led to recommend a comprehensive strategy for the prevention of obesity, which started in 2010 and we are currently evaluating.

Facilitating the uptake of policies to promote healthy lifestyles and estimating their impact – Linda Bauld (UK)

More than four in ten cancers in the United Kingdom could be prevented by modifying behavioural risk factors, a pattern that is also observed in other countries. Using the UK as an example, this presentation will examine the relationship between overweight and obesity and cancer risk and outline how policy interventions could reduce the number of obesity-related cancers, particularly if the actions taken affect children and families.

Evidence strongly suggests that children with excess body weight are more likely to become overweight or obese as adults, increasing their risk of at least ten types of cancer. In the UK, one in five girls are overweight or obese when they enter primary school (aged 4–5 years) and by age 11, this rises to one in three. If these rates continue, almost three in four adults could be overweight or obese by 2035.
Individual behavioural interventions have a role to play, but the most significant gains are likely to be made at the population level if policies are introduced that address some of the main drivers. These include price, promotion, the product and place-based measures. Working with the Obesity Health Alliance, a multi-agency national partnership, CRUK has recently invested in research that makes the case for policy change on both price and promotion. This presentation will focus in particular on the changes needed to address the promotion of foods high in salt, sugar and fat to children in the UK and how these types of marketing restrictions are likely to contribute to cancer prevention in the future.

Cancer and Nutrition NIHR infrastructure collaboration: future research directions – Alan Jackson (UK)

The Cancer and Nutrition NIHR infrastructure collaboration was established in early 2014 against the need to better align research activities. The UK has world leading research both in cancer and in nutrition but historically the research in the two areas has not been brought together sufficiently to make full use of the potential opportunities.

Cancer patients want guidance on how to achieve the best quality of life, to slow the development or progression of their cancer, and to improve their survival. Unfortunately the lack of reliable evidence often means that health professionals are not able to provide evidence-based advice, and hence patients find unreliable information from unaccredited sources. Clinicians need evidence to make informed decisions and advise patients appropriately. However, they have told us that the lack of reliable evidence on how nutrition and related factors affect those living with cancer makes giving such advice problematic.

Patients, charities, researchers and clinicians have a key role to play in enabling and supporting the generation of evidence that both patients and healthcare professionals need, and are asking for, to ensure cancer patients get the best possible care.

To bridge this gap the Collaboration has developed five work streams to better enable collaborative and mutually supportive endeavours (http://cancerandnutrition.nihr.ac.uk/).

The effects of caloric restriction on molecular mechanisms related to tumorigenesis in the colon of C57BL/6J mice.

Drewerjite Kok1, Fanni Rusli2, Carolyn Lute3, Ellen Kampman1, Michael Müller1, Wilma Steegenga1

1. Division of Human Nutrition, Wageningen University, Wageningen, the Netherlands; 2. NUTRISCIENCE and Sysytems Nutrition, Norwich Medical School, University of East Anglia, Norwich, UK

Obesity is linked to an increased risk of multiple types of cancer, including colon cancer. From this perspective, caloric restriction (CR) has been considered a strategy to restore the energy balance and prevent tumorigenesis, although the underlying mechanisms are poorly understood. The aim of this study was to provide a comprehensive overview of the impact of CR on colonic health.

Male C57BL/6J mice received a life-long CR diet with 30% energy reduction, or a control (C) diet or medium-fat (MF) diet ad libitum. Mice (n=8-18/group) were sacrificed at the age of 6 or 28 months and colon, blood and urine samples were collected. Colonic gene expression profiles, levels of short chain fatty acids, microbiota composition, serum cytokine levels and urinary metabolites were determined.

After CR, expression of genes related to inflammatory and immune response pathways was down-regulated compared to the C-MF diet. Also serum levels of the pro-inflammatory cytokine interleukin 6 were lower in mice on CR as compared to the MF diet (p<0.05). Microbial profiling showed that CR resulted in a higher abundance of the Bifidobacteria and Lactobacillus genera, whereas Clostridium s. tertium was less abundant compared to the other diet groups. Furthermore, microbial richness was correlated with body weight at sacrifice (r=-0.613, p<0.001).

These findings indicate that prolonged CR is associated with complex and integrated molecular phenomena related to metabolic, anti-inflammatory and immune responses. Detailed insight into effects of CR may facilitate development of feasible strategies to reduce energy intake and thereby prevent obesity and cancer.

Omentin is a novel pleiotropic adipokine linked to obesity, immune response and metabolic dysfunction, thereby it could play a role in the development of colorectal cancer (CRC).

However, the association between omentin and CRC risk has not been evaluated in prospective cohort studies. We explored the association between circulating plasma omentin concentrations and risk of CRC in a case-cohort study comprising 251 incident CRC cases diagnosed over a mean follow-up time of 10.4 years and 2,295 persons who remained free of cancer in the European Prospective Investigation into Cancer Nutrition (EPIC)-Potsdam. Hazard ratios as a measure of relative risk (RR) and 95% confidence intervals (CI) were computed using a Prentice modified Cox regression. In a multivariable model adjusted for age, sex, education, dietary and lifestyle factors, body mass index (BMI) and waist circumference, higher omentin concentrations were associated with a higher CRC risk (RR adjusted for age, sex, education, dietary and lifestyle factors, BMI and waist circumference =1.98, 95% CI: 1.45-2.73).

Additional adjustment for metabolic biomarkers, including glycedated hemoglobin, high-density lipoprotein cholesterol and C-reactive protein, did not alter the results. In stratified analyses, the positive association between omentin and CRC risk was observed in participants with BMI < 30 (RR=2.26, 95% CI: 1.57-3.27), whereas among participants with BMI ≥ 30 no association was revealed (RR = 1.07, 95%CI: 0.63-1.83; Pinteraction = 0.009). These data suggest for the first time an independent association between circulating omentin concentrations and CRC risk. Furthermore, the data revealed a potential interaction with the adiposity state of an individual that requires further evaluation in future research.
A study on effect modification by mTOR/PI3K/Akt gene variants of associations between body size, physical activity, early life energy restriction and colorectal cancer risk

Colinda C.J.M. Simons1, Leo J. Schouten2, Rietger W.L. Godschalk3, Frederik-Jan van Schooten4, Monika Stoll5,6, Kristel van Steen5,6, Piet A. van den Brandt5, Mathijs P. Weijenberg1

1. Department of Epidemiology, Groningen University Medical Center, Groningen, the Netherlands; 2. Department of Pharmacology & Toxicology, Maastricht University, Maastricht, the Netherlands; 3. Institute of Human Genetics, Genomic Epidemiology, University of Münster, Münster, Germany; 4. Department of Genetics, Maastricht University, Maastricht, the Netherlands; 5. Systems and Modeling Unit, Monائر Institute, University of Utrecht, Utrecht, Utrecht, Germany.

Introduction: The influence of body size, physical activity and early life energy restriction on colorectal cancer (CRC) risk may be modified by mTOR/PI3K-Akt gene variants. These genes integrate signals from nutrients and growth factors and affect (cancer) growth.

Methods: The Netherlands Cohort Study includes 120,862 men and women aged 55–69 years old, who were administered a questionnaire in 1986. Follow-up was 20.3 years. In a total of 24,145 events, we investigated the association between body size related genes, early life energy restriction and colorectal cancer risk.

Results: Early adulthood (age 18–<30 years) body mass index (BMI) was significantly inversely associated with pre- and post-menopausal CRC (summary RRs per 5 kg/m² = 0.82, 95% confidence interval [CI] 0.76–0.89 and 0.82 [0.76–0.88], respectively). Adult weight gain significantly increased the risk of post-menopausal CRC, but not pre-menopausal CRC (summary RRs per 5 kg gain = 1.06, 95% CI = 1.05–1.08 and 0.99 [0.96–1.03], respectively). In pre-menopausal women, greater BMI and higher vigorous physical activity was significantly inversely associated with CRC risk, when BMI was accounted for in the study, increased risk was observed with greater waist circumference (WC) (summary RR per 1 cm = 1.14, 95% CI = 1.04–1.26) and similarly with waist-to-hip ratio (WHR).

Conclusions: Greater abdominal obesity (WC/WHR) throughout life increases pre- and post-menopausal CRC risk. After menopause, the effect of excessive general obesity (BMI) is more relevant.

ORAL PRESENTATION ABSTRACTS
DAY 1

Energy balance-related factors and breast cancer risk in pre- and post-menopausal women

Doris S.M. Chan1, Snigdha Vingelienė1, Dagfinn Aune2,3, Elii Polomaa1, Leila Abar1, Rita Vieira1, Darren C. Greenwood1, Teresa Norat1

1. Department of Epidemiology and Biostatistics, School of Public Health, Imperial College London, London, United Kingdom; 2. Department of Public Health and General Practice, Norwegian University of Science and Technology, Trondheim, Norway; 3. Leeds Institute of Cardiovascular and Metabolic Medicine, University of Leeds, Leeds, United Kingdom.

Aim: To systematically review the findings from prospective studies of energy balance-related factors (physical activity, obesity, and adult weight gain) and breast cancer (BC) risk in pre- and post-menopausal women.

Methods: Relevant publications on the associations of interest were searched in PubMed up to April 2015. In our meta-analysis, data were from 16 prospective studies (8 pre-menopausal and 8 post-menopausal women) and odds ratios (ORs) were calculated. We calculated an overall (fixed-effects model) estimate of the ORs for each outcome.

Results: Pre-menopausal women had a lower risk of BC (summary OR 0.90; 95% CI = 0.80–1.00) and post-menopausal women had a higher risk of BC (summary OR 1.27; 95% CI = 1.10–1.47). Higher adult weight gain was associated with a higher risk of BC in both pre- and post-menopausal women (summary OR 1.10; 95% CI = 1.00–1.21).

Conclusions: Physical activity and obesity at pre-menopause may protect against BC. However, obesity and weight gain during adult life can lead to a higher BC risk in both pre- and post-menopausal women.
ORAL PRESENTATION ABSTRACTS

Here, we assessed the association between levels of serum glucose, cholesterol, and triglycerides and PCa deaths.

Methods and material: From the Swedish AMORIS cohort, we selected all men diagnosed with PCa between 1996 and 2011 who had baseline measurements of serum glucose, triglycerides and total cholesterol and whose tumour information (i.e. TNM stage, Gleason score, and PSA) was registered in the National Prostate Cancer Register (%14,150). In addition, we used information on sociodemographic characteristics, comorbidities and cause of death. Multivariable Cox proportional models were used to determine the hazard ratios (HR) for PCa-specific and all-cause mortality by quartiles of glucose, triglycerides and total cholesterol.

Results: Glucose had an independent positive association with all-cause mortality (HR for highest versus lowest quartile: 1.18; 95% CI: 1.07-1.31). Similarly, risk of PCa deaths increased with increasing glucose levels (HR: 1.20; 95% CI: 1.03-1.39). Risk of death from all-causes was higher among hypertriglycemic men than those with normal triglycerides levels (HR: 1.14; 95% CI: 1.03-1.26 and 1.16 (1.05-1.30) for third and fourth quartiles versus first), but PCa-specific death rates did not vary. Survival did not differ by total cholesterol levels.

Conclusion: Our results suggest that glucose metabolism influences PCa survival. However, more research on the link between glucose and PCa survival is needed to validate our findings as management of glucose levels may have therapeutic importance in helping to control PCa progression.

Randomised controlled trial of continuous versus intermittent energy restriction during adjuvant chemotherapy (The B-AHEAD2 Trial)

Michelle Harvie1, Mary Pegington1, Anne Armstrong2, Nigel Bundred1, Julie Morris1, Judith Adams1, Lee Graves1, Louise Gorman1, Alastair Greystone2, Sacha Howell1,2, Anthony Howell1,2

1. Ninewells Hospital and Medical School, University of Dundee; 2. Department of Medical Oncology, The Christie NHS Foundation Trust. 3. Department of Medical Statistics, UHSM; 4. Institute of Population Health University of Manchester; 5. Research Institute for Sport and Exercise Sciences, Liverpool John Moores University; 6. Northern Centre for Cancer Care, Freeman Hospital, Newcastle upon Tyne.

Background: Observational data indicate that excess weight at breast cancer diagnosis and weight gain during adjuvant chemotherapy increases risk of recurrence and death. We have reported that continuous energy restriction (CER) and exercise is only partially effective for weight control during chemotherapy. Randomised trials in healthy subjects indicate that intermittent energy restriction (IER) may be equivalent or superior to CER for weight control, and animal data suggest reduced chemotherapy toxicity with IER. Here we will report the results of a randomised comparison of IER vs. CER amongst 172 women receiving adjuvant /neoadjuvant chemotherapy.

Methods: Women were recruited immediately after surgery. IER (n = 86) or CER (n = 86) was administered throughout the 4.5-6 month course of chemotherapy. The primary endpoint was body weight, body fat & lean body mass assessed with DEXA. Secondary endpoints were chemotherapy toxicity (self-report CTCAE and Cytokine 3 ligand serum markers), quality of life (PACT scaled) and serum markers associated with prognosis (insulin sensitivity, adiponectin, leptin).

Results: Uptake was 39%. Twenty-eight women (16%) failed to complete the study, 19 IER and 9 CER. High adherence and adherence effects interest and motivation of women to make positive changes to lifestyle even at the time of diagnosis.

Conclusion: Results are currently being analysed. We will report changes in body weight composition and toxicity, and serum markers associated with prognosis between IER and CER at the September.

POSTER PRESENTATIONS

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Physical exercise in newly diagnosed breast cancer patients may counteract a decrease in cardiovascular capacity during adjuvant treatment.

Anders Hussy1, Tora J Bettum2, Hanne Frydenberg3, Trygve Lofte4, Elisabeth Edvaerden2, Freydis Fjeldheim1, Vidar G Flette1, Gro Bertheussen4, Erik A Wist5, Sigmund A Andersen6, Anne McTiernan6, Inger Thune7,8,9,10

1. The Cancer Center, Oslo University Hospital, Ullevål, Oslo, Norway; 2. Norwegian School of Sport Sciences, Department of Sports Medicine, Oslo, Norway; 3. Oslo University Hospital, Ullevål, Department of Pulmonary Medicine, Oslo, Norway; 4. Department of Physical Medicine and Rehabilitation, St. Olavs University Hospital Trondheim, Trondheim, Norway; 5. Department of Neuroscience, Norwegian University of Science and Technology, Trondheim, Norway; 6. Fred Hutchinson Cancer Research Center, Seattle, WA, USA; 7. Institute of Clinical Medicine, Faculty of Health Sciences, University of Tromsø, Norway.

Methods: The aim was to study the feasibility of completing a 12-month exercise intervention during adjuvant breast cancer treatment, and to evaluate the effect of exercise on cardiovascular capacity. Breast cancer patients were randomized to a control group (N=28) or to a 12-month physical activity intervention (N=31) after surgery. The intervention group participated in supervised exercise 2×60 min/week, combined with at least 60 min/week of home training. The control group received standard care. Cardiorespiratory fitness (VO2 \(_{\text{max}}\)) was assessed during cardiopulmonary exercise testing on a treadmill before surgery, at 6 months and 12 months. Intention-to-treat analysis were performed using a linear mixed model.

Results: The breast cancer patients had the following means at diagnosis: age 55.4 years, body mass index (BMI) 25.2 kg/m\(^2\), VO2 \(_{\text{max}}\) of 32.4 ml·kg\(^{-1}\)·min\(^{-1}\). After 6 months, women in the intervention group had a 0.5 ml·kg\(^{-1}\)·min\(^{-1}\) increase in VO2 \(_{\text{max}}\); while women in the control group had a 4.1 ml·kg\(^{-1}\)·min\(^{-1}\) decline in VO2 \(_{\text{max}}\). These differences in cardiorespiratory fitness were observed overall (p<0.001), and were more pronounced among premenopausal women (p=0.011), and women receiving chemotherapy (p=0.001). Differences were less distinct at 12 months, but did not vary by body composition.

Conclusion: Physical exercise during adjuvant breast cancer treatment is feasible, and may reduce a decline in cardiovascular capacity, regardless of body composition, but may vary by age and type of treatment. These findings need to be replicated in larger clinical trials.

Physical Activity during Cancer Treatment (PACT) study: randomized clinical trial of physical exercise during cancer treatment – long-term effects on physical activity levels

Anouk Hienisch1, Miranda J Velthuis2, Jonna K van Vulpen3, Elsken van der Wall3, Petra HM Peeters3,4, Anne M May5

1. Julius Center for Health Sciences and Primary Care, University Medical Center Utrecht, Utrecht, the Netherlands; 2. Netherlands Comprehensive Cancer Organisation, Utrecht, the Netherlands; 3. Department of Medical Oncology, Cancer Center, University Medical Center Utrecht, Utrecht, The Netherlands; 4. School of Public Health, Imperial College London, London, United Kingdom.

Background: A recent randomized trial, the PACT study, showed beneficial effects on fatigue and physical fitness after an 18-week supervised exercise program in breast and colon cancer patients undergoing adjuvant treatment. Yet, maintaining a physically active lifestyle after cancer treatment is reported as challenging, even though it is important to preserve achieved beneficial effects. This study aims to assess long-term effects of the PACT exercise intervention on physical activity (PA).

Methods: 128 out of 237 breast and colon cancer patients participated in the 4-year follow-up physical activity questionnaire (SQUASH, n=70 exercise; n=58 usual care control). We investigated minutes per week moderate-to-vigorous leisure and sport PA and total moderate-to-vigorous PA, which was assessed at baseline (within 10 weeks after diagnosis), 18-week (post-intervention), and 36-week and 4-years post-baseline.

Results: Intention-to-treat mixed linear model analyses showed that both moderate-to-vigorous leisure and sport PA and total moderate-to-vigorous PA decreased significantly from baseline to 18-weeks in both groups (i.e. during adjuvant treatment). Four-years post-baseline, patients returned to their baseline PA levels, and moderate-to-vigorous leisure and sport PA in the intervention group was significantly higher (85 (31;148) minutes/week). Also, compared to control, patients in the intervention group reported 142 (2,282) minutes/week more total moderate-to-vigorous PA 4-years post-intervention, whereas moderate-to-vigorous leisure and sport PA did not differ (22 (44;88) minutes/week).

Conclusions: Breast and colon cancer patients who participated in an exercise intervention during adjuvant treatment reported higher total PA levels four years later, emphasizing the benefits of exercising during this period.

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Prizes will be awarded to the best poster presentation and the best oral presentation. Voting slips for the poster presentation award are included in your delegate pack.
Inflammation and breast cancer risk: a case-control study nested in the epic-Varese cohort

Agnoli C1, Grioni S1, Krogh V1, Pala V1, Allione A2,3, Mattullo G2,3, Sieri S1

1. Epidemiology and Prevention Unit, Fondazione IRCCS-Istituto Nazionale dei Tumori, Milan, Italy. 2. Medical Sciences Department, University of Torino, Torino, Italy. 3. Human Genetics Foundation-Torino, Torino, Italy

Breast cancer (BC) is one of the most frequently diagnosed cancers and the leading cause of cancer death among women. Adipokines and inflammation molecules, whose production is influenced by adiposity, may be involved in breast carcinogenesis; however data from prospective studies on their associations with BC risk are not conclusive.

We investigated the association between plasma levels of C-reactive protein, TNF-α, interleukin-6, leptin, and adiponectin in a nested case-control study on women of the EPIC-Varese cohort who gave blood samples in 1992-1997. BC cases identified up to 31 December 2009 were individually matched to control subjects. Rate ratios (RRs) 95% confidence interval (CI) were estimated by conditional logistic regression controlling for BC risk factors.

After a median follow-up of 14.9 years, 351 cases of BC were identified. Among premenopausal women, adiponectin levels reduced the risk (RR 0.40; 95%CI:0.21-0.77), whilst leptin levels increased the risk (P trend=0.012), amongst highest vs lowest tertile). Among premenopausal women, adiponectin levels reduced the risk (RR 0.40; 95%CI:0.21-0.77).

POSTER PRESENTATION ABSTRACTS

Body mass index and cancer risk: a systematic review and dose-response meta-analysis of cohort studies

Dagfinn Aune1,2, Doris S.M. Chan1, Leila Abair1, Sabrina Schlesinger1, Abhijit Sen1, Ana Rita Vieira1, Nievesguervieli1, Christophe Stevens1, Darren C. Greenwood1, Teresa Norat1

1. Department of Epidemiology and Biostatistics, School of Public Health, Imperial College London, London, United Kingdom. 2. Department of Public Health and General Practice, Norwegian University of Science and Technology, Trondheim, Norway. 3. Leeds Institute of Cardiovascular and Metabolic Medicine, University of Leeds, Leeds, United Kingdom

Purpose: To quantitatively summarize the dose-response association of body mass index (BMI) and cancer risk.

Methods: PubMed was searched for relevant cohort studies up to April 2015 as part of the WCRF Continuous Update Project. Random effects models were used to calculate summary relative risks (RRs).

Results: Higher BMI was significantly related to the risk of the following cancers (all summary RRs per 5 kg/m²): total cancer 1.04 (95% CI: 1.01-1.06), esophageal adenocarcinoma 1.48 (1.35-1.62), gastric cancer 1.23 (1.07-1.40), small intestine 1.10 (1.02-1.18), colon 1.07 (1.05-1.09), rectum 1.02 (1.01-1.04), gallbladder 1.23 (1.21-1.37), pancreas 1.10 (1.07-1.14), liver 1.30 (1.16-1.46), prostate-adenocarcinoma 1.32 (1.28-1.36), ovary 1.07 (1.02-1.12), endometrium 1.58 (1.51-1.65), advanced/high grade prostate cancer 1.08 (1.04-1.12), kidney 1.29 (1.24-1.34), bladder 1.05 (1.01-1.09), non-Hodgkin’s lymphoma 1.06 (1.03-1.09), Hodgkin’s lymphoma 1.26 (1.05-1.51), multiple myeloma 1.05 (1.01-1.09), leukemia 1.09 (1.06-1.12), thyroid 1.20 (1.10-1.30) and brain 1.05 (1.01-1.10). Inverse associations were observed for pre-menopausal breast cancer 0.92 (0.88-0.96), nonadvised prostate cancer 0.95 (0.92-0.98) and for two smoking-related cancers: esophageal squamous cell carcinoma 0.64 (0.56-0.73), and lung cancer 0.83 (0.80-0.86). In never smokers, the summary RRs were: total cancer 1.08 (1.03-1.13), oral and pharyngeal cancer 1.02 (0.88-1.19), and lung cancer 0.93 (0.84-1.03).

Conclusions: Higher BMI was associated with increased risk of 20 of 27 types of cancer investigated.

Applying computed tomography-based body composition analysis to assess adipose and muscle tissue parameters at colorectal cancer diagnosis and to investigate associations with long-term health-related quality of life

Eline H. van Roekel1, Martijn J.L. Bours3, Malou E.M. te Molder1, José J.L. Breedveld-Peters2, Steven W.M. Olde Damink3, Leo J. Schouten1, Silvia Sanduleanu1, Gerard L. Beets1 and Matty P. Weijenberg1

1. Department of Epidemiology, GROW School for Oncology and Developmental Biology, Maastricht University, PO Box 616, 6202 MD Maastricht, The Netherlands. 2. Department of Surgery, NijMEd School for Nutrition, Toxicology and Metabolism, Maastricht University Medical Centre+, PO Box 5800, 6202 AZ Maastricht, The Netherlands. 3. Department of Internal Medicine, Division of Gastroenterology and Hepatology, GROW School for Oncology and Developmental Biology, Maastricht University Medical Centre+, PO Box 5800, 6202 AZ Maastricht, The Netherlands

Purpose: Increased visceral adiposity (visceral obesity) and muscle wasting (sarcopenia) at colorectal cancer (CRC) diagnosis, quantified by CT image analysis, have been unfavorably associated with short-term clinical outcomes and survival, but associations with long-term health-related quality of life (HRQoL) are unknown. We studied associations of visceral adiposity, muscle fat infiltration, muscle mass and sarcopenia at CRC diagnosis with HRQoL 2-10 years post-diagnosis.

Methods: A cross-sectional study was conducted in 155 stage I-II CRC survivors, diagnosed at Maastricht University Medical Centre+, the Netherlands (2002-2010). Diagnostic CT images at the level of the third lumbar vertebra of 104 participants were analyzed. Adipose and muscle tissue parameters were measured by eLISA. Percentage change was tested using Wilcoxon Signed rank Test. Correlation analysis was performed using Spearman’s rho.

Results: Twelve participants (mean ±standard deviation age 64 ±11 years) participated. Body composition was maintained throughout the intervention. At baseline, inflammatory status correlated inversely with sedentary behavior (r=-0.74, P=0.009), TNF-α (r=-0.69, p<0.05). IL-8 reduced significantly from pre- to post-intervention (percentage change -11.25%, p<0.05). While energy metabolism did not change, interestingly lactate concentration correlated strongly and inversely with aerobic fitness at post-intervention only (rho=-0.68, p=0.02).

Conclusions: Results suggest that multimodal rehabilitation following CRC treatment reduced inflammatory status and improved lactate metabolism without compromising body composition. Examination of these findings in a larger trial will validate results.
Lifestyle related health factors after cancer treatment

G. Haukur Guðmundsson, physiotherapist B.Sc. - Ljósið Cancer Rehabilitation Centre, Helgi Sigurðsson, Oncologist PhD. University Hospital of Iceland, Erlingur S. Jóhannsson, Physiology professor PhD. University of Iceland

The aim of the study was to (A) evaluate body composition, health related quality of life (HR-QoL) and Personality D amongst cancer survivors who had finished their treatment.

Eighty participants aged 25 – 77 of both genders, who were in remission, answered questionnaires on HR-QoL (SF-36v2 and EQ-5D-3L), Personality D (DS14), general health, lifestyle and cancer treatment. Participants had their blood pressure measured, conducted the 6-minute walk test (6MWT) and had their body composition measured: body mass index (BMI), body fat percentage, waist and hip circumferences. SPSS was used for statistical analysis.

2/3 of participants had body composition over recommended values. Over half of the participants were overweight or obese. 66.3% had waist circumference over recommended values and 45.0% had waist:hip ratio over recommended values. Significant correlation (p<0.05) was found between 6MWT and body composition as well as most categories of HR-QoL. Increased fitness correlated with better body composition and higher HR-QoL. Only 13.8% of participants measured with distressed personality according to DS14. Fitness evaluated with 6MWT seems to be able to indicate HR-QoL and body composition and therefore its use in clinical setting is supported.

Irregular intake of energy and alcohol is not associated with an increased risk of breast cancer in UK women

GK Potsi1, MAH Lemij3, AM Stephen1, TJ Key2, EJ Brunner4, and KT Khaw1

1. Wiye University Amsterdam, Section Health and Life, Faculty of Earth and Life Sciences, the Netherlands; 2. Kings College London, Division of Diabetes and Nutritional Sciences, Franklin-Wilkins Building, London, UK; 3. University of Cambridge, Department of Public Health and Primary Care, Cambridge, UK; 4. Surrey University, Department of Nutritional Sciences, Faculty of Health and Medical Sciences, Surrey, Guildford, UK; 5. Cancer Epidemiology Unit, University of Oxford, Oxford UK; 6. Department of Epidemiology and Public Health Unit, University College London, London UK

Background: Irregular eating could be a potential risk factor for breast cancer; however, research on this topic is scarce.

Objective: To study associations of irregular intake of energy and alcohol in relation to breast cancer risk.

Design: This case-control study included dietary intake data from 7-day estimated diet diaries of 629 UK women with breast cancer and 1,798 controls. Intakes of energy and alcohol during breakfast, lunch, dinner, and between meals were analysed using a score for irregularity based on the deviation from the 7 day mean. Conditional logistic regression models were used to estimate odds ratios (ORs) for associations between meal regularity scores across tertiles and breast cancer risk adjusting for relevant covariates. A separate model was run for post-menopausal women only.

Results: No associations between breast cancer risk and irregular intake of energy during breakfast (OR 0.93 95% CI 0.68-1.30, p trend 0.11), lunch (OR 0.89 95% CI 0.65-1.23, p trend 0.55), evening meal (OR 1.07, 95% CI 0.79-1.45, p trend 0.49), between meals (OR 0.95, 95% CI 0.69-1.32, p trend 0.92), and daily total (OR 0.92, 95% CI 0.65-1.96, p trend 0.51) were observed. Daily irregular intake of alcohol was also not associated with an increased risk of breast cancer (OR 1.11, 95% CI 0.79-1.55, p trend 0.57).

Conclusions: Irregular intakes of energy and alcohol were not associated with an increased breast cancer risk in women in this UK case-control study. Further research is needed to investigate whether irregular meal patterns are associated with other types of cancer.

Physical activity and quality of life in men living with advanced prostate cancer

Gráinne Sheill1, Lauren Brady2, Emer Guinan3, Juliette Hussey1, David Hevey1, Tatjana Vlajnic1, Orla Casey1, Anne-Marie Baird1, Fidelma Cahill1, Mieke Van Hemelrijck1, Nicola Peat1, Sarah Rudman1, Thomas Lynch1, Rustom P. Manecksha1, Brian Hayes1, Moya Cunningham1, Liam Grogan1, John McCaffrey1, Dearbhail M O’Donnell1, Ray Mc Dermott1, John O’Leary1, Stephen Finn1

1. Discipline of Physiotherapy, School of Medicine, Trinity College Dublin, Ireland; 2. Department of Histopathology and Medical Anatomy, School of Medicine, Trinity College Dublin, Ireland; 3. School of Psychology, Trinity College Dublin; 4. Institute of Haematology, University Hospital Basel, Switzerland; 5. All Ireland Co-operative Oncology Research Group, Dublin, Ireland; 6. Cancer Epidemiology Group, Division of Cancer Studies, Kings College London, UK; 7. Guy’s and St Thomas’ NHS Trust Foundation, London, UK; 8. Department of Urology, St James’s Hospital, Dublin, Ireland; 9. Department of Haematopathology, Cork University Hospital, Cork, Ireland; 10. Department of Radiation Oncology, St Luke’s Hospital, Dublin, Ireland; 11. Department of Oncology, Beaumont Hospital, Dublin, Ireland; 12. Department of Oncology, Meath hospital, Dublin, Ireland; 13. HOMD Directories, St James’s Hospital, Dublin, Ireland; 14. Department of Oncology, AMNH, Dublin, Ireland; 15. Department of Haematopathology, St James’s Hospital, Dublin, Ireland

Background: Patients with metastatic prostate cancer live with a considerable disease burden that may have a profound impact on everyday physical function and quality of life. Little is known about physical activity levels and other lifestyle factors of patients with advanced prostate cancer.

Methods: EuFACT (Exercising, Prostate Cancer and Circulating Tumour Cells), is a multi-centre clinical trial for patients with metastatic prostate cancer. Questionnaires examining quality of life (FACT-P), sleep (Pittsburgh Sleep Index), depression (PHQ-9) and physical activity (Harvard Health Professionals Study Questionnaire) are completed at baseline, three and six months.

Result: An interim analysis of the baseline outcome measures of 25 patients (mean age 71.62 (SD 8.08) years) was completed. Mean BMI was 29.4 (SD 7.7) kg/m2. The majority (76% (n=19) of participants had sleep scores indicative of poor sleep quality. Sleep scores correlated negatively with global quality of life (r =-0.54, p=0.009). Patients scored lowest in the ‘functional’ and ‘additional prostate cancer related concerns’ domains of quality of life. Preliminary data demonstrate that 32% (n=8) of participants failed to reach a threshold physical activity of 9 MET h/wk, previously associated with reductions in all-cause mortality in this population. Physical activity levels were not associated with quality of life (p=0.98), sleep (p=0.42) or depression (p=0.10) scores.

Discussion: Findings highlight the association between poor quality of life and sleep in this population. In addition, initial data suggest a high prevalence of suboptimal physical activity levels among older men. Lifestyle interventions involving exercise may provide a mechanism to increase physical activity levels in patients with metastatic disease.

Disturbed NK cell function in obesity is associated with an increased colon tumor risk

Bahr I, Gonitz V, Dobberstein H, Berreis T, Rosenstock P, Kielstein H

Department of Anatomy and Cell Biology, Faculty of Medicine, Martin Luther University Halle-Wittenberg, Germany

Background: Natural killer (NK) immune cells control tumor progression and metastases. Previous studies showed altered NK cell functions in obese individuals. Obesity is associated with a higher colon cancer incidence, but underlying mechanisms remained unclear. We investigated the impact of an altered NK cell functionality on the increased colon cancer risk in obesity.

Methods: The cytotoxicity of leptin-activated NK cells against colon tumor cells was determined using Europium release assay. Cytolysis of NK cells was measured by luminescence immunoassay. Moreover, in vivo experiments in normal weight and obese rats were performed. Colon cancer growth was induced in half of the animals by injection of azoxymethane (AOM). Body weight and visceral fat mass were determined, colon tumors were characterized and tumor, splenic and hepatic NK cells were analyzed by real-time RT-PCR and immunohistochemistry.

Results: The cytotoxicity of NK cells against colon tumor cells was significantly reduced after leptin incubation. Additionally, the reduced NK cell cytotoxicity by leptin was associated with a decreased IFNγ secretion. In obese rats, body weight and visceral fat mass were significantly increased compared to the normal weight animals. AOM-treated obese rats showed an increased quantity, size and weight of colon tumors compared to the normal weight AOM group. Molecular and immunohistological analyses indicate tissue specific changes in NK cell numbers as well as expression levels of activating NK cell receptors in obese animals.

Conclusion: The results showed that the impaired NK cell function may be one cause for the elevated colon cancer risk in obesity.
Inflammatory Potential of Diet and the Risk of Colorectal Tumours in Persons with Lynch Syndrome

JGM Brouwer1, M Makama2, GJ van Woudenbergh1,2, HFA Vos3, FM Nagengast4, JH Kleibeuker2, E Kampman2, FJB vDuijnhoven1

1. Wageningen University, Wageningen, the Netherlands; 2. Christelijke Hogeschool, Ede, the Netherlands; 3. Foundation for the Detection of Hereditary Tumour Syndromes (GOET), Leiden, the Netherlands; 4. Radboud University Medical Centre, Nijmegen, the Netherlands; 5. University Medical Centre Groningen, Groningen, the Netherlands

Background: Persons with Lynch syndrome (LS) have a lifetime risk of developing colorectal tumours (CRTs) due to mutations in mismatch repair genes. An important process in the development of CRTs is inflammation, which has been shown to be modulated by diet. We aimed to investigate the association between the inflammatory potential of diet and the risk of CRTs in persons with LS.

Methods: We used dietary intake of 466 persons with LS from a prospective cohort study to calculate the adapted dietary inflammatory index (ADI). Cox proportional hazards models, with robust sandwich variance estimates to adjust for dependency within families, were used to calculate hazard ratios (HR) of ADI tertiles. The adjusted model included age, smoking status and sex. Stratified analyses were performed by mutated gene and non-steroidal anti-inflammatory drugs (NSAIDs) use.

Results: During a median person-time of 60.9 months, 198 (42.5%) participants developed CRTs. The ADI was not associated with risk of CRTs in the adjusted model (HRhighest vs. lowest tertile: 1.12 [95%CI 0.77-1.62], p=0.68). The adjusted hazard ratios were higher for participants with an MLH1 mutation (HRhighest vs. lowest tertile: 1.68 [95%CI 0.92-3.09], p=0.23) but lower among those with an MSH2 MLH1 mutation (Hrhighest vs. lowest tertile: 1.68 [95%CI 0.92-3.09], p=0.23) than for patients without the recommendations for PA attenuated the association less fatigue (-3.5; -6.9,-0.1). Associations of an adherence score with HrQoL was analyzed by confounder-adjusted linear regression models. The overall WCrf/AICr score adherence (continuous) with HrQoL was analyzed by confounder-adjusted linear regression models. The mean score adherence was 4.9 (SD: 1.4, range 1.5-8.5). A higher score adherence was significantly associated with better physical functioning (β 2.9, 95%CI 0.5, 5.3) and less fatigue (β 3.5, 9-10). Associations of an adherence score without the recommendations for PA attenuated the association with physical functioning but the association for fatigue was unchanged (β 3.5, -7, 7.0). Discussion: Higher adherence of CrC survivors to WCrf/AICr lifestyle recommendations are for cancer prevention. In the absence of specific recommendations for cancer survivors, we investigated how colorectal cancer (CRC) survivors adhere to the WCRF/AICR lifestyle recommendations and how this is associated with their health-related quality of life (HRQol). Results: Body-weight, visceral fat amount, plasma leptin and IL-6 levels were significantly increased in diet-induced obese BALB/c mice. Mice burden was increased in the obese animals as compared to their lean/littermates. Interestingly, the functionality of NK cells was altered in the obese mice with breast cancer.

Conclusion: The present study on a postmenopausal breast cancer model helps to understand basic molecular mechanisms regulating NK cell functionality in obese individuals and the association of the elevated breast cancer risk in obesity.

Natural killer cell functionality is impaired by diet-induced obesity in a postmenopausal breast cancer mouse model

Laura Matthes, Juliane-Susanne Schmidt, Heike Kielstein, Julia Spielmann

Department of Anatomy and Cell Biology, Faculty of Medicine, Martin-Luther University Halle-Wittenberg

Aim: Obesity is a widespread disease and was identified as a major risk factor for malignant diseases, including postmenopausal breast cancer. Although the underlying mechanisms are poorly understood, it is known from in-vitro studies that essential functions of natural killer (NK) cells such as targeting tumor cells are disturbed in obese individuals. Thus, the aim of the present study is the investigation of NK cell functionality of obese mice in a postmenopausal breast cancer model.

Methods: To induce obesity, female mice (BALB/c) received either a standard diet (4% fat) or a high-fat diet (34% fat) for up to 13 weeks. Thereafter, mice were ovariectomized and after 3 weeks of recovery, syngeneic 4T1-Luc2 mouse mammary tumor cells were injected orthotopically into the fat pad of the mammary gland. Twenty hours or 3 weeks after tumor cell challenging blood, tissues and tumors were collected and analyzed. Different techniques such as flow cytometry, luminescence and real-time PCR aimed to analyze numbers, activity and physiological properties of NK cells.

Results: Body-weight, visceral fat amount, plasma leptin and IL-6 levels were significantly increased in diet-induced obese BALB/c mice. Mice burden was increased in the obese animals as compared to their lean/littermates. Interestingly, the functionality of NK cells was altered in the obese mice with breast cancer.

Conclusion: The present study on a postmenopausal breast cancer model helps to understand basic molecular mechanisms regulating NK cell functionality in obese individuals and the association of the elevated breast cancer risk in obesity.

Colorectal cancer survivors' adherence to the cancer prevention recommendations of the WCRF/AICR and cross-sectional associations with health-related quality of life

Jose J.L. Breedveld-Peters1, Eloise Müller-Schulte2, Bernadette W.A. van der Linden3, Martijn J.L. Bours1, Eline H. van Roekel1, Matty P. Weijenberg1

1. Department of Epidemiology, GroW – School for Oncology and Developmental Biology, Maastricht University, PO Box 416, 6200 MD Maastricht, The Netherlands; 2. Institute of Medical Microbiology and Hygiene, Saxion University Medical Center, Homburg, Germany

Background: The World Cancer Research Fund (WCRF) and American Institute for Cancer Research (AICR) lifestyle recommendations are for cancer prevention. In the absence of specific recommendations for cancer survivors, we investigated how colorectal cancer (CRC) survivors adhere to the WCRF/AICR lifestyle recommendations and how this is associated with their health-related quality of life (HRQol).

Methods: The cross-sectional part of the Energy for Life after Colorectal Cancer (EnColle) study includes 155 CRC survivors (stage I-III) 2-10 years post-diagnosis. Dietary intake, physical activity (PA) and body composition were measured by seven-day food diaries, the SQUASH questionnaire, accelerometry, and physiological properties of NK cells.

Results: Body-weight, visceral fat amount, plasma leptin and IL-6 levels were significantly increased in diet-induced obese BALB/c mice. Mice burden was increased in the obese animals as compared to their lean/littermates. Interestingly, the functionality of NK cells was altered in the obese mice with breast cancer.

Conclusion: The present study on a postmenopausal breast cancer model helps to understand basic molecular mechanisms regulating NK cell functionality in obese individuals and the association of the elevated breast cancer risk in obesity.

HOT TOPIC CONFERENCE: Obesity, Physical Activity & Cancer: Life course influences and mechanisms

POSTER PRESENTATION ABSTRACTS
Mediating Factors of the Association between Adult Weight Gain and Colorectal Cancer: Data from the European Prospective Investigation into Cancer and Nutrition (EPIC) Cohort

Krasimira Aleksandrova1, Sabrina Schlesinger1, Veronika Fedorova2,3, Mazda Jenabi4, Bas Bueno-de-Mesquita5,6, Tobias Pischon5,6, Marc J. Gunter3, Christina Botta1,2, Elisabeth2,1, Heiner Boeing2,1 on behalf of the EPIC Group

1. Nutrition, Immunity and Metabolism Start-up Lab, Department of Epidemiology, German Institute of Human Nutrition Potsdam-Rathenow, Nuthe-Nikolas, Germany; 2. Institute of Epidemiology, Christian-Albrechts University of Kiel, Kiel, Germany; 3. Department of Epidemiology, Rikker School of Public Health, Emory University, Atlanta, GA, USA; 4. Winship Cancer Institute, Emory University, Atlanta, GA, USA; 5. International Agency for Research on Cancer (IARC-WHO), Lyon, France; 6. National Institute for Public Health and the Environment (RIVM), Bilthoven, the Netherlands; 7. Department of Gastronutrition and Public Health, University Medical Center, Utrecht, The Netherlands; 8. Division of Epidemiology, Public Health and Primary Care, Imperial College, London, United Kingdom; 9. Department of Social and Preventive Medicine, Faculty of Medicine, University of Malaya, Kuala Lumpur, Malaysia; 10. Molecular Epidemiology Group, Max Delbrueck Center for Molecular Medicine (MDC), Berlin-Buch, Germany; 11. WHO Collaborating Center for Maternal and Child Health, Department of Haematology, Epidemiology and Medical Statistics, University of Athens Medical School, Athens, Greece; 12. Hallenic Health Foundation, Athens, Greece; 13. Bureau of Epidemiology and Research, Academy of Athens, Athens, Greece; 14. Department of Epidemiology, German Institute of Human Nutrition Potsdam-Rathenow, Nuthe-Nikolas, Germany.

A growing body of evidence has emerged consistently suggesting that high body weight gain during adulthood is associated with a higher risk of colorectal cancer (CRC), whether weight gain per se promotes CRC risk or weight gain is a marker for other risk factors. However, exact mechanisms by which weight gain during adulthood is associated with CRC risk are not completely understood.

Methods:

We simulated a random effects model of colon cancer risk: an update of the WCRC-AICr systematic review of published prospective studies

1. Department of Epidemiology and Biostatistics, Imperial College, London, United Kingdom; 2. Biostatistics Unit, Centre for Epidemiology and Biostatistics, University of Leeds, Leeds, United Kingdom (D.C.G); 3. Biostatistics Unit, Centre for Epidemiology and Biostatistics, University of Leeds, Leeds, United Kingdom (D.C.G); 4. Department of Urology, St James’s Hospital, Dublin, Ireland; 5. Department of Histopathology and Morbid Anatomy, School of Medicine, University of Helsinki, Helsinki, Finland; 6. Department of Oncology, Geneva University Hospitals, Geneva, Switzerland; 7. Department of Epidemiology and Biostatistics, Imperial College, London, United Kingdom; 8. Department of Biostatistics, King’s College London, London, UK; 9. Department of Biostatistics and Technology, Imperial College, London, UK; 10. Institute of Experimental Pathology, National Research Council, Canada; 11. Department of Histopathology, St James’s Hospital, Dublin, Ireland; 12. Department of Pathology, St James’s Hospital, Dublin, Ireland; 13. Department of Pathology, St James’s Hospital, Dublin, Ireland; 14. Department of Pathology, St James’s Hospital, Dublin, Ireland.

Background: Obesity is a global healthcare burden which is associated with lifestyle choices. Obesity, metabolic syndrome and inflammation play key roles in prostate cancer disease progression. While obesity is not predictive of prevalence, it is associated with a more aggressive metastatic disease state. We therefore sought to examine exercise as an addition to treatment in metastatic prostate cancer and determine its effectiveness through the assessment of obesity related markers.

Methods: ExPeCT (Exercise, Prostate Cancer and Circulating Tumour Cells, ICRG 15-21) is a multi-centre clinical trial for patients with metastatic prostate cancer participants are randomised into exercise and control arms and further stratified within groups based on clinical measurements. Patients in the exercise arm complete six months of aerobic exercise at a moderate-to-vigorous intensity. Patient serum samples were assessed for adipokine, leptin and resistin using the MS- SCALE Discovery platform at baseline, three and six months.

Results: An interim analysis of eleven patients has suggested an increase in resistin levels over time in the control group compared with a decrease in the exercise group. Preliminary data from adiponectin and leptin assays demonstrated negligible changes, with adiponectin marginally decreased post-exercise. Accrual to this trial is ongoing and the link between obesity and inflammation, a number of inflammatory mediators are being investigated.

Discussion: Initial data suggests that resistin levels are decreased in our exercise cohort after three months. Therefore exercise may provide valuable clinical benefit to patients with metastatic disease, given the link between high levels of resistin and prostate cancer disease progression.

Conclusion: The positive association with height suggests that early life nutrition might play a role in colorectal cancer risk in adulthood. Higher total and abdominal body fatness during adulthood are risk factors of colorectal cancer.

Survival after breast cancer among women with anorexia nervosa

Lene Meldemkjer1, Fotios C Papadopoulos2, Eero Pulkka2,3, Anders Ekbom4, Mikko Gissler5, Jorgen H. Olsen1

1. Danish Cancer Society Research Center, Copenhagen, Denmark; 2. Department of Neuroscience, Psychiatry, Uppsala University, Uppsala, Sweden; 3. Finnish Cancer Registry, Institute for Statistical and Epidemiological Cancer Research, Helsinki, Finland; 4. School of Health Sciences, University of Tampere, Tampere, Finland; 5. Department of Medicine Solna, Karolinska University Hospital, Karolinska Institutet Stockholm, Sweden; 6. National Institute for Health and Welfare, Helsinki, Finland.

Aim: The hypothesis concerning dietary energy restriction and reduction of cancer risk is primarily based on animal experiments. Anorexia nervosa is an indicator of prolonged energy restriction in humans. We previously found that breast cancer incidence among anorexia nervosa patients was 39% lower than among population comparisons. We investigated overall survival after breast cancer among women with anorexia nervosa versus population comparisons.

Methods: Among 22,654 women with anorexia nervosa in National Hospital Registers in Sweden, Denmark and Finland during 1988-2010, we identified 76 breast cancer cases while 1,468 cases were observed in randomly selected comparisons from population registers following survival after breast cancer among women with anorexia nervosa was compared to that of population comparisons by estimating hazard ratios (HR) in Cox regression analyses with adjustment for age and calendar period. Further adjustment for extent of disease (local vs. non-localized) was performed in a subset of breast cancers.

Results: During follow-up after breast cancer, 23 women in the anorexia nervosa group died while 247 women in the comparison group died. The HR for overall survival was 2.1 (95% CI = 1.4-3.2) after adjustment for age and calendar period. When restricting to the subset with information on extent of disease and further adjusting for this variable, the HR was 2.6 (95% CI = 1.5-4.5).

Conclusion: With a prior diagnosis of anorexia nervosa have a worse survival after breast cancer than other women.

The Impact of a Structured Exercise Programme on Adipokine Status in Patients with Metastatic Prostate Cancer

Lauren Brady1, Graineen Shed1, Tatjana Vlajcic1, John Greene1, Orla Casey1, Anne-Marie Baird1, Emer Guinan1, Juliette Hussey1, Fidelma Cahill1, Mieke Van Hemelrijk1, Nicola Peat2, Sarah Ruddman3, Thomas Lynch1, Rustom P. Maneksha1, Brian Hayes1, Moys Cunningham1, Liam Grogan1, John McCaffrey1, Dearbhail O’Donnell1, Ray Mc Dermott1, John O’Leary1, Stephen Finn1,4

1. Department of Haematology and Medical Oncology, School of Medicine, Trinity College Dublin, Ireland; 2. Discipline of Physiotherapy, School of Medicine, Trinity College Dublin, Ireland; 3. Institute of Pathology, University Hospital Basel, Switzerland; 4. All Ireland Co-operative Oncology Research Group, Dublin, Ireland; 5. Cancer Epidemiology Group, Division of Cancer Studies, King’s College London, London, UK; 6. Institute of Biomedical Sciences, Norwegian University of Science and Technology, Trondheim, Norway (DA); 7. Biostatistics Unit, Centre for Epidemiology and Biostatistics, University of Leeds, Leeds, United Kingdom (D.C.G).

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Discussion: Initial data suggests that resistin levels are decreased in our exercise cohort after three months. Therefore exercise may provide valuable clinical benefit to patients with metastatic disease, given the link between high levels of resistin and prostate cancer disease progression.

Conclusion: The positive association with height suggests that early life nutrition might play a role in colorectal cancer risk in adulthood. Higher total and abdominal body fatness during adulthood are risk factors of colorectal cancer.

Survival after breast cancer among women with anorexia nervosa

Lene Meldemkjer1, Fotios C Papadopoulos2, Eero Pulkka2,3, Anders Ekbom4, Mikko Gissler5, Jorgen H. Olsen1

1. Danish Cancer Society Research Center, Copenhagen, Denmark; 2. Department of Neuroscience, Psychiatry, Uppsala University, Uppsala, Sweden; 3. Finnish Cancer Registry, Institute for Statistical and Epidemiological Cancer Research, Helsinki, Finland; 4. School of Health Sciences, University of Tampere, Tampere, Finland; 5. Department of Medicine Solna, Karolinska University Hospital, Karolinska Institutet Stockholm, Sweden; 6. National Institute for Health and Welfare, Helsinki, Finland.

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Conclusion: With a prior diagnosis of anorexia nervosa have a worse survival after breast cancer than other women.
ReStOre - Rehabilitation Strategies following Oesophageal Cancer: Achieving exercise guidelines in a nutritionally vulnerable group without undesirable weight loss: A Feasibility Study

L O’Neill1, SL Doyle2, E Guman3, J O’Sullivan4, JV Reynolds5, J Hussey6

1. Discipline of Physiotherapy, Trinity College, Dublin; 2. School of Biomedical Sciences, Dublin Institute of Technology, Dublin; 3. Department of Surgery, Trinity College Dublin and St James’s Hospital, Dublin

Background: Malnutrition is a long-term side effect of oesophageal cancer (OC) treatment. Whilst exercise programmes have the potential to improve physical function, quality of life, and survival, the increase in energy expenditure as a result of exercise participation may result in undesirable weight loss if not managed correctly. This feasibility study aimed to demonstrate that OC survivors can achieve recommended levels of exercise through a structured diet and exercise programme without compromising body composition.

Methods: Patients who were ≥12 months post OC curative treatment completion, including oesophagectomy, were recruited. The 12-week programme consisted of exercise rehabilitation, dietary counselling to ensure adequate energy and protein intake, and multi-disciplinary education. Body composition was assessed pre- and post-intervention using bio-impedance analysis (Seca mBCA machine), key variables and protein intake, and multi-disciplinary education. Body composition was assessed pre- and post-intervention using bio-impedance analysis (Seca mBCA machine), key variables were weight(kg), fat mass(kg) and skeletal muscle mass(kg). Activity levels were monitored with Actigraph activity monitors.

Results: Twelve participants (mean, standard deviation (SD) age 61.4 (7.29) years, 8 male, BMI 24.01(5.02) kg/m2) completed the programme. Post-intervention all were achieving the programme. This activity induced increase in energy expenditure was recommended 150 minutes of moderate intensity exercise per week. This activity increased in energy expenditure was not associated with loss of weight from pre to post intervention (10.19(19.95)kg vs 10.29(19.4)kg p=0.169), or fat mass (19.488(40)kg vs 19.148(34)kg, p=0.599) or skeletal muscle mass (25.448.72)kg vs 24.868.7)kg, p=0.093.

Conclusion: Under the guidance of a physiotherapist and dietitian, survivors of OC may achieve exercise guidelines without compromising body composition. The results of this study will be further verified in a randomised controlled trial.

Adiposity and cancer: an umbrella review of the literature.

Maria Kyriiou1, Ilka Kallilas2, George Markozannes3, Marc J Gunter4, Evangelos Paraskevaidis4, Hani Gabra2, Pierre Martin-Hirsch2, Konstantinos K Tsididis6

1. Department of Surgery & Cancer, Hammersmith, Imperial College, London, W12 ONN, UK; 2. School of Nursing, Epidemiology & Ophthalmology, University of Ioannina School of Medicine, 41110, Ioannina, Greece; 3. Department of Obstetrics and Gynaecology, University of Ioannina, 45110, Ioannina, Greece; 4. Department of Epidemiology and Biostatistics, School of Public Health, Imperial College London, London, UK; 5. Department of Obstetrics and Gynaecology, University of Ioannina, 45110, Ioannina, Greece; 6. Department of Gynaecologic Oncology, Lancaster Teaching Hospitals, Preston, PR2 9HT, UK

Background: To evaluate strength and validity of the literature evidence on the associations between adiposity and risk of cancer.

Methods: An umbrella review of meta-analyses of observational studies investigating adiposity and cancer risk was conducted after searching in PubMed, EMBASE and the Cochrane library. The primary analysis focused on cohort studies exploring associations for continuous contrasts of adiposity. The literature evidence was graded into strong, highly suggestive, suggestive or weak categories after applying various criteria, such as the statistical significance of the random effects summary estimate and of the largest study in a meta-analysis, the number of cancer cases, between-study heterogeneity, 95% prediction intervals, small study effects, excess significance bias and sensitivity analysis with credible ellipsoids.

Findings: A total of 157 meta-analyses were identified, which investigated associations of seven indices of adiposity with the development or death from 32 cancers. Of the 76 meta-analyses that included cohort studies and used a continuous scale to measure adiposity, only 11 (14%) associations for nine cancers demonstrated strong evidence. BMI was associated with a higher risk of developing oesophageal adenocarcinoma, colorectal cancer in men, biliary tract, pancreatic and kidney cancer and multiple myeloma. Weight gain and weight to hip circumference ratio were strongly associated with a higher risk of postmenopausal breast and endometrial cancer, respectively.

Interpretation: The association of adiposity with cancer risk has been extensively studied, but only associations for nine cancers were graded with strong evidence.

Tumor expression of Fatty Acid Synthase and Its Ligand in colorectal cancer and its impact on prognosis; A Systematic Review

Rosin O’Neill1 and Patrick Hickland1, Maurice Loughrey2, Ruth Wein3, Jayne V Woodside4, Marie M Cantwell1

1. School of Medical Dentistry and Biomedical Sciences, Queen’s University Belfast, Northern Ireland; 2. University of Ulster, Coleraine, Northern Ireland

Background: Binding of fatty acid synthase (FAS), a cell surface receptor from the TNF receptor superfamily to its ligand (FASL) results in a death-inducing signalling complex which induces apoptosis. Clinical studies have demonstrated aberrant expression of FAS / FASL in colorectal cancer cells. The aim of this study was to systematically review studies published to date which have examined the association between colorectal tumour FAS and/or FASL expression and subsequent prognosis.

Methods: Using terms for FAS and FASL, Medline, Embase, and Web of Science databases were systematically searched from inception to September 2015 using appropriate MESH terms and keywords.

Results: Nine studies were identified for inclusion. The association between tumour FAS expression and prognosis was inconclusive in these studies which suggested improved prognosis used monocular antibodies, had a large sample size, included a greater proportion of low grade tumours and did not include rectal samples. The association between FAS expression and prognosis may also be dependent on obesity, tumour FAS overexpression was associated with improved survival in non-obese patients, but predicted a worse outcome in moderately overweight/obese patients. The association between tumour FAS expression and prognosis was inconsistent but in the studies which suggested a poorer prognosis, FASL expression was increased with advanced tumour stage, and was associated with an increase in tumour depth, lymph node involvement and presence of metastases.

Discussion: There is insufficient evidence to recommend FAS/ FASL expression as a prognostic marker in colorectal cancer patients. Additional studies which adjust or stratify by obesity status are warranted.

Lower handgrip strength is associated with worse quality of life and more fatigue and disability in long-term colorectal cancer survivors

M.J.L. Bours1, E.H. van Roekel1, J.J.L. Breedveld2, Peters1, and M.P. Weijenberg3

1. Department of Epidemiology, Gronow School for Oncology and Developmental Biology, Maastricht University, PO. Box 616, 6200 MD Maastricht, The Netherlands

Purpose: An unhealthy body composition may contribute to persisting treatment-related complaints and decreased quality of life (QoL) and functioning reported by many colorectal cancer (CRC) survivors. Therefore, we explored associations of body composition with QoL, fatigue, and disability in CRC survivors.

Design: Cross-sectional study in 155 stage I–III CRC survivors, 2.10 years post-diagnosis. Body composition was assessed by anthropometric measures, including body mass index (BMI), waist circumference, waist-to-hip ratio, four-site skinfold measurements to determine body fat percentage, and maximum handgrip strength. Validated questionnaires were used to measure QoL (EORTC-QLQ C30; 100-point scale), fatigue (Checklist Individual Strength; 120-point scale) and disability (WHODAS-II; 100-point scale). Associations of anthropometric measures with QoL, fatigue and disability were analyzed by confounder-adjusted linear regression models.

Results: CRC survivors (38% females) were on average 70.1 (SD 8.9) years of age, 57/ 18 (3.8 years since diagnosis, and 74.7% were overweight or obese (BMI ≥ 25 kg/m2). Multivariable regression analyses showed that greater handgrip strength was significantly associated with better global QoL (β per 5 kg increment: 2.3; 95% CI: 0.3, 4.3) and physical functioning (2.9; 9.9, 5.0), and with less fatigue (5.9; -8.7, -3.0) and disability (2.5; -4.1, -0.9). Gender-stratified analyses showed similar results for handgrip strength in both men and women. Additionally, among men only, higher body fat percentage was significantly associated with worse physical functioning (β per 5% increment: -5.0; -9.1, -0.8) and more fatigue (5.9; 0.0, 11.7).

Conclusion: Lower handgrip strength was cross-sectionally associated with worse QoL, fatigue and disability in long-term CRC survivors.
An increase in physical activity after colorectal cancer surgery is associated with improved recovery of physical functioning

Moniek van Zutphen1, Renate M. Winkels2, Frainzel J.B. van Duijnhoven3, A. Suzanne van Harten-Gemists4, Diereuvertje E.G. Koop5, Peter van Duijvendijk6, Henk van Halteren6, Bibi M.E. Hansson7, Flip M. Kruijf8, Ernst J. Spillenlaar Bilgen9, Johannes H.W. de Wit10, Jaap J. Dronkers11, Ellen Kampman12

1. Division of Human Nutrition, Wageningen University, Wageningen, The Netherlands; 2. Department of Surgery, Galje Hospital, Apeldoorn, the Netherlands; 3. Department of Internal Medicine, Admiraal de Ruyter Hospital, Goes/Vlissingen, The Netherlands; 4. Department of Surgery, Canisius Wilhelmina Hospital, Nijmegen, The Netherlands; 5. Department of Surgery, Rijnstate Hospital, Arnhem, The Netherlands; 6. Department of Surgery, Radboud University Nijmegen Medical Centre, Nijmegen, The Netherlands; 7. Department of Physical Therapy, Galje Hospital, Goes, The Netherlands.

Background: The influence of physical activity on patient-reported recovery of physical functioning after colorectal cancer (CRC) surgery is unknown. Therefore, we studied recovery of physical functioning after hospital discharge by (a) an increase in physical activity and (b) absolute activity levels before and after surgery.

Methods: We included 327 incident CRC patients (stages I-II) from a prospective cohort study. Patients completed questionnaires that assessed physical functioning and moderate-to-vigorous physical activity both before and after surgery. Cox regression models were used to calculate prevalence ratios (PRs) of no recovery of physical functioning. All PRs were adjusted for age, sex, physical functioning before surgery, stage of disease, tumour and body mass index.

Results: At six months post-diagnosis 54% of CRC patients had not recovered to preoperative functioning. An increase in physical activity after CRC surgery was associated with improved recovery of physical functioning. This benefit was seen regardless of physical activity level before surgery. These associations provide new leads to explore connections between physical activity and recovery from CRC surgery after discharge from the hospital.

Body mass index, total cancer incidence and colorectal cancer in young and middle-aged men and women followed over 28 years: The Tromso Study

Sunday Oluwafemi Oyeyemi, Bjarne Koster Jacobsen, and Kristin Benjamannen Borch, Department of Community Medicine, UiT-The Arctic University of Norway

A re-audit of a Weight Management after Cancer Treatment Programme

Bracegirdle R and Kegey O, Guys and St Thomas NHS Foundation Trust


Background: The “Weight Management after Cancer Treatment” programme is open to overweight cancer survivors who have completed radical oncological treatment. It is a sunworkshop initiative involving teaching on healthy eating to support weight loss. (“The Cancer Strategy (2015-2020)” recognises the need for healthy lifestyle advice to reduce risk of recurrence.

Aim: Re-audit the clinical effectiveness of the weight management programme against NICE standards and ascertain areas for improvement.

Methods: Weight measures were prospectively collected for participants attending the programme between February 2014-July 2015. Paired t-test analysis was used to determine statistical difference (p<0.05) between baseline and final weight measurements, after nine weeks active intervention. Data on participant satisfaction was gathered from 28 anonymous questionnaires.

Results: An average of 8 participants attended each programme over the data collection period, giving a total sample size of 40. Average weight loss was 3.4kg (3.7%) over 9 weeks duration of programme (p<0.01). 30%(n=12) of participants achieved ≥5%. 11% clinically significant weight loss. 27.5% (n=11) of participants achieved a weight change of ≥3–5% over 9 weeks. 42.3% (n=17) of participants achieved ≥2% weight loss. All respondents had increased knowledge of a healthy lifestyle and 94.6% made lifestyle changes. 78.5% of respondents were ‘quite’ to ‘very’ pleased with weight loss progress.

Conclusion: Overall, weight loss outcomes achieved compared favourably to NICE standards. The weight management after cancer treatment programme is effective in increasing knowledge and promoting healthy lifestyle changes in cancer survivors. Development of annual follow-up sessions was identified as an area for improvement.

Height, genetic variants in the insulin-like growth factor pathway and post-menopausal breast cancer risk according to estrogen receptor status

Rachel Elsands1, Colinda C Simons1, Leo J Schouten2, Janneke G Hogervorst3, Bas A Verhage4, Piet A van den Brand1, Matty P Weijenberg1

1. Department of Epidemiology, GROW School for Oncology and Developmental Biology, Maastricht University, Maastricht, the Netherlands; 2. Department of Environmental Biology, Centre for Environmental Sciences (CML), Wageningen University, Wageningen, The Netherlands; 3. Department of Internal Medicine, Admiraal de Ruyter Hospital, Goes, The Netherlands; 4. Department of surgery, Gelre Hospital, Apeldoorn, The Netherlands.

Introduction: Height is a risk factor for post-menopausal breast cancer (BC). To understand how growth is associated with BC, height and single nucleotide polymorphisms (SNPs) in insulin-like growth factor (IGF) pathway genes were investigated in relation to BC estrogen receptor (ER) subtypes.

Methods: Women (n=42,573) from The Netherlands Cohort Study self-reported height at baseline (aged 55-69). Follow-up was 20.3 years (case-cohort design). Twenty-four SNPs in 9 IGF pathway genes were genotyped in total DNA. Multivariable-adjusted hazard ratios (HR) and 95% confidence intervals (CI) were calculated for overall BC (n=2,387), ER+ (n=1,161) and ER- (n=1,249) subtypes (subcohort n=1,170). Mediation by height was indicated when additionally adjusting for height introduced a >10% change of HR. Genotype-height interactions were tested using the Wald-test.

Results: Height was associated with overall BC (HR per 5 cm=1.07, 95%CI=1.01-1.13), and specifically ER+BC (HR=1.09, 95%CI=1.02-1.17), but not with ER-BC (HR=1.02, 95%CI=0.90-1.15). IRF5 rs1801278 and ADORA1 rs76539542 were associated with overall BC risk (dominant model). Three SNPs were (borderline) significantly associated with ER+BC, but not with ER-BC risk (ADORA1 rs76539542: Hr(ER+)=0.79, 95%CI=0.67-0.92 and Hr(ER-)=1.03, 95%CI=0.95-1.10; IRS1 rs4773802: Hr(ER+)=0.84, 95%CI=0.69-1.02 and Hr(ER-)=1.04, 95%CI=0.71-1.52; ADORA2 rs167870: Hr(ER+)=1.15, 95%CI=0.97-1.36 and Hr(ER-)=0.91, 95%CI=0.67-1.28). Additional adjustment for height did not alter the HRs. There were no significant height-SNP interactions.

Conclusion: Although height and ADORA1, ADORA2 and IRS1 SNPs were specifically associated with ER+BC, suggesting a role for growth and energy metabolism in ER+BC development, the results do not support that these SNPs explain part of the height ER+BC association nor modify this association.

POSTER PRESENTATION ABSTRACTS
Background: Observational research has shown that higher levels of physical activity (PA) are associated with improved prognosis in breast cancer patients. Hence, promotion of being physically active is important. We investigate PA levels of Dutch breast cancer patients and compare these with the Dutch female population as reference population.

Method: Breast cancer patients were included when planned for radiotherapy and PA levels of an average week in the last two months were measured (SQUASH questionnaire). The same questionnaire was used in the Dutch female population. Hours per week performing PA and adherence to Dutch PA guidelines were compared between patients and the Dutch female population. Standardized Incidence Ratios (SIR) were calculated for patients who meet the guidelines, with the Dutch female population as reference population.

Results: Breast cancer patients (N=424, mean age 58.5±10.6 years) were on average 34.4 hours per week physically active (including household activities). This was 8.7 hours per week less compared to the Dutch female population (n=3076, mean age 49.8±17.7 years; 95%CI -11.0;-6.4). no difference was found between patients and the Dutch female population.

Aim: To systematically review the evidence from prospective studies on the association of BMI with risk of oesophageal and stomach cancers and examine if the association differs by histological or anatomical cancer types.

Methods: This review has been conducted as part of the WCRF Continuous Update Project. PubMed has been searched for relevant prospective cohort studies until 28th February 2014. Random-effects dose-response meta-analyses were conducted to calculate summary relative risks (RR) for an increment of 5 kg/m² in BMI.

Results: Nineteen studies (28 916 cases) were included in the dose-response meta-analysis on BMI and gastric cancer and 16 studies (10 342 cases) in the analysis of oesophageal cancer. Significantly positive association was observed for oesophageal adenocarcinoma (RR=1.18 (95% CI: 1.15-1.21, I² = 37%, P = 0.13, 9 studies) and gastric cardia cancer (RR=1.23 (95% CI: 1.07-1.40, I² = 56%, P = 0.004, 7 studies). Significant inverse association was found for oesophageal squamous cell carcinoma (RR=0.64 (95% CI: 0.56-0.73, I² = 72%, P = 0.001, 8 studies). High heterogeneity for squamous cell carcinoma was not explained in stratified analysis by sex, geographic location, self-reported or measured BMI, and adjustment for smoking.

Conclusion: The evidence from cohort studies confirms the positive association between BMI and oesophageal adenocarcinoma and gastric cancer. The inverse association with SCC may be influenced by residual confounding by smoking which is a major risk factor for SCC.

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The WCRF/AICR continuous update project: bmi and incidence of oesophageal and gastric cancer subtypes

Snigdha Vingelii1, Doris S. M. Chan1, Dagfinn Aune1,2, Ana R. Vieira1, Christophe Stevens1, Elii Polemi1, Leila Aba1, Deborah Navarro Rosenblatt1, Darren C. Greenwood1 and Teresa Norat1

1. Department of Epidemiology and Biostatistics, School of Public Health, Imperial College London, London, United Kingdom; 2. Department of Public Health and General Practice, Faculty of Medicine, Norwegian University of Science and Technology, Trondheim, Norway; 3. Division of Biostatistics, University of Leeds, United Kingdom

Evolution of lean body mass (LBM) during palliative systemic treatment in metastatic colorectal cancer (mCRC) patients participating in the randomized phase 3 CAIRO3 study

Sophie A. Kurk, MD, Department of Medical Oncology, Utrecht University Medical Center Utrecht, Utrecht University, Heidelberglaan 100, 3584 CX, Utrecht, the Netherlands. E-mail: s.a.kurk@umcutrecht.nl, Petra H.M. Peeters, MD PhD. 1. Julius Center for Health Sciences and Primary Care, University Medical Center Utrecht, Utrecht University, Heidelberglaan 100, 3584 CX, Utrecht, the Netherlands. 2) School of Public Health, Imperial College London, South Kensington Campus, London SW7 2AZ, United Kingdom. E-mail: p.h.m.peeters@umcutrecht.nl, Bram Doresteijn, PhD. Nutricia Research, Uppsala, 12, 3584 CT, Utrecht, The Netherlands. Email: bram.doresteijn@danone.com, Marion Jourdan, PhD. Nutricia Research, Uppsala, 12, 3584 CT, Utrecht, The Netherlands. Email: marion.jourdan@nutricia.com, Hugo Kuijf, PhD. Image Sciences Institute, University Medical Center Utrecht, Heidelberglaan 100, 3584 CX Utrecht, the Netherlands. E-mail: h.kuijf@isi.uu.nl, Cornelis J.A. Punt, MD PhD. Department of Medical Oncology, Academic Medical Center, University of Amsterdam, Meibergdreef 9, 1105 AZ Amsterdam, the Netherlands. E-mail: c.punt@amc.uva.nl, Miriam Koopman*, MD PhD. Department of Medical Oncology, University Medical Center Utrecht, Utrecht University, Heidelberglaan 100, 3584 CX, Utrecht, the Netherlands. E-mail: m.koopman6@umcutrecht.nl, Anne M. May*, PhD. Julius Center for Health Sciences and Primary Care, University Medical Center Utrecht, Utrecht University, Heidelberglaan 100, 3584 CX, Utrecht, the Netherlands. E-mail: a.m.may@umcutrecht.nl, *authors contributed equally

Background: Observational studies suggest that low LBM is associated with chemotherapy-related toxicity and poor survival in mCRC patients. Little is known about patterns of LBM during palliative systemic therapy. Here we use data of the CAIRO3 study in which mCRC patients with stable disease or better after 6 cycles CAPOX-B were randomized between maintenance treatment (CAP-B) and observation. Disease progression in both groups was followed by systemic reinduction therapy (CAPOX-B or other treatment) until second disease progression. We used repeated scan data of the first 62 consecutive patients to investigate LBM during treatment.

Methods: 1% CT-scans of 62 patients (63.7±9.4 years, n=31 maintenance, n=31 observation) were analyzed for LBM at four occasions during palliative systemic treatment using single slice evaluation at L3. Paired-sample t-tests were conducted to assess changes over time.

Results: Before study-randomization during CAPOX-B induction treatment, LBM decreased significantly in all patients (0.7kg, 95% CI 1.2; 0.3). After randomization, LBM increased significantly during maintenance treatment (0.8kg, 0.03; 1.7, median treatment time 8.8 months) compared to the observation arm (0.2kg, 0.4; 0.8, median observation time 4.8 months). Upon first progression, during reinduction with CAPOX-B/other treatment, LBM again decreased significantly in both groups (1.1kg, 1.7; -0.5). Updated results will be presented at the conference.

Conclusion: Our preliminary data suggests muscle loss in mCRC patients during palliative chemotherapy is reversible and depends on treatment regimen. Although studies have shown prognostic capacity for LBM, the effect of subsequent changes in LBM are unknown and may be clues for new future therapeutic interventions.

*Simkovic et al. Lancet 2015

** Results will be updated with data of more CAIRO3 participants.
**POSTER PRESENTATION ABSTRACTS**

**Mendelian randomization study of triglycerides, low- and high-density lipoprotein, and the statin-targeted 3-hydroxy-3-methylglutaryl-coenzyme A reductase (HMGCR) gene, in relation to cancer risk**

Tanja Stocks, George Hindy, and Marju Orho-Melander

**Department of Clinical Sciences in Malmö, Diabetes and Cardiovascular Diseases – Genetic Epidemiology, Lund University, Sweden**

**Background:** Mendelian randomization analysis makes a strong case for causality, but it has rarely been used to study blood lipids and cancer risk.

**Methods:** We included 26,904 participants from the Malmö Diet and Cancer Study. In logistic regression analysis, we used a Mendelian randomization approach with instrumental variables comprising 26-41 single nucleotide polymorphisms (SNPs) for triglycerides, low-density lipoprotein, LDL, or high-density lipoprotein, to investigate their associations with cancer risk. We also used pleiotropy-adjusted analysis to reveal associations adjusting for SNP effects on several lipid traits, which indicates the adjusted direction of association, but not the effect size. We also investigated cancer risk by HMGCR rs12916, which affects LDL levels and is targeted in statin treatment.

**Results:** Incident cancers were 6607 in total, 1322 prostate, 1181 breast, 591 lung, 497 colon, and 400 bladder. Triglycerides were inversely related to total cancer, odds ratio per standard deviation genetic triglyceride level was 0.91 (95% CI: 0.80–1.03, P=0.1), and the association was significant in pleiotropy-adjusted analysis (P=0.01). HMGCR rs12916 per T-allele association was 1.09 (1.01–1.18, P=0.03) for prostate cancer and 0.89 (0.82–0.96, P=0.004) for breast cancer. In pleiotropy-adjusted analyses, LDL was suggestively, inversely related to prostate cancer (P=0.09, and 0.05 for prevalent vs incident cases), but was not related to breast cancer (P=0.6).

**Conclusion:** Elevated triglycerides may reduce the risk of cancer overall, but the LDL-lowering effect of statins may increase prostate cancer risk, but statins may decrease breast cancer risk through non-targeted pathways. Replication in other populations, and investigation of potential mechanisms, are needed.

**Correlations of Physical Activity and Obesity with All Cause Cancer Mortality**

Soh, Yih Chau, Soh, Chin Yun

This is a study pooling and aggregating the dataset from the World Health Organisation and the World Bank. The study was set in the year 2010. All member countries of the World Health Organisation were included in the analysis. Missing data of these member states were imputed by Markov Chain Monte Carlo and linear regression.

There is no correlation for the proportion of physical inactivity in the countries with the all cause cancer mortality rate for both gender at -0.056 (95% confidence interval, -0.168 to 0.068). The correlation for male is -0.073 (95% confidence interval, -0.172 to -0.027) and for female is -0.007 (95% confidence interval, -0.126 to -0.121).

There are positive correlations of the proportion of Body Mass Index (BMI) greater than 25 among nations with all cause cancer mortality for both genders at 0.207 (95% confidence interval, 0.166 to 0.337). However, the correlation is stronger for male at 0.327 (95% confidence interval, 0.237 to 0.484) and non-significant findings for female at 0.027 (95% confidence interval, -0.102 to 0.149).

The pooled data on the mean BMI of all WHo member states were imputed by Markov Chain Monte Carlo and linear regression.

**2007** Second Expert Report on Food, Nutrition, Physical Activity, and the Prevention of Cancer is launched. Developed and managed by WCRF International, it is the most comprehensive study ever published showing the scientific links between food, nutrition, physical activity and cancer prevention.

**2009** Policy and Action for Cancer Prevention report launched to support the scientific research in the Second Expert Report, setting out recommendations for how policy can help encourage people to adopt WCRF’s Recommendations for Cancer Prevention.

**2009** WCRF International becomes Vanguard partner to Union for International Cancer Control, working together to effect change in global cancer control.

**2011** WCRF International attends UN High Level Meeting on Non-Communicable Diseases in New York, along with other major cancer leaders from around the globe.

**2016** World Cancer Research Fund’s Continuous Update Project (CUP) is a trusted, authoritative scientific resource, which informs current guidelines and policy for cancer prevention. Reports on thirteen cancer types have now been published with a comprehensive report due in late 2017.

**2016** World Cancer Research Fund for WCRF network charities.

**2008** World Cancer Research Fund network committed to investing in a long term project - the Continuous Update Project (CUP). The CUP, overseen by WCRF International was established to maintain a central database of the accumulated evidence related to food, physical activity, weight and cancer.

**2010** WCRF International grant programme introduces geographical split between the Americas and rest of world. Programme funds research on the effects of diet, nutrition, body composition and physical activity on cancer prevention and survival.
For further information about World Obesity activities please contact:

World Obesity Federation
Charles Darwin House 2, 107 Gray's Inn Road,
London, WC1X 8TZ
Tel +44 (0)20 7685 2580
enquiries@worldobesity.org
www.worldobesity.org
@WorldObesity
Registered Charity No. 1076981

For further information about World Cancer Research Fund International please contact:

World Cancer Research Fund International
Second Floor, 22 Bedford Square, London WC1B 3HH
Phone: +44 (0)20 7343 4200
international@wcrf.org
www.wcrf.org
@wcrfin