SAVE THE DATE

2017 NCRI Cancer Conference
5th - 8th November 2017
BT Convention Centre, Liverpool, UK
The largest cancer research meeting in the UK

Suggest a session
www.ncri.org.uk/2017

Follow us on Twitter @NCRI_partners for programme updates
On behalf of the Scientific Committee, I am delighted to welcome you all to the 12th NCRI Cancer Conference, hosted for a seventh year in Liverpool.

The pace of cancer research breakthroughs has continued in the past year and this year’s Conference aims to showcase these scientific developments that are already resulting in measurable benefits for patients.

A hallmark of the NCRI Cancer Conference, our programme spans the breadth of cancer research; from basic science to public health. Just some of the highlights of the multi-disciplinary programme we have assembled this year include world-leading experts speaking on early detection and prevention of cancer, tumour initiating cells, regulation of cell proliferation and cell death, cancer genomics, cancer immunology and microbiome, tumour heterogeneity, new developments in cancer treatment, cancer related symptoms in advanced disease and communicating cancer research to the public.

We anticipate attendance at the 12th NCRI Cancer Conference will catalyse ideas and broker new interdisciplinary collaborations. Over the next few days, you will be able to choose between sessions from more than 140 UK and international speakers, read more than 500 abstracts and visit over 70 stands. Our exciting scientific programme will be presented over the course of four days in four session types – plenaries, symposia, parallel sessions and workshops. Use the Conference App to schedule which sessions to attend, note which posters to see and which stands to visit.

Following the success of the past four years, we will again see sessions programmed in association with the Royal College of Radiologists and welcome for the first time BASO ~ The Association for Cancer Surgery who will present embedded sessions. We also welcome back the ever-popular Clinical Trials Showcase which provides updates on practice-changing trials and the Dragons’ Den, led by the NCRI Consumer Forum, which is a great opportunity for researchers to put their study proposals to a panel of patients and carers and gain the benefits of their unique perspectives and experiences. We have reintroduced the proffered papers sessions to encourage younger scientists to present their research orally to a broad audience. And on Wednesday 9th November, for the seventh year in a row, we will welcome around 50 A-level students from local schools to further their understanding of research and inspire the next generation to follow a career in science.

Many sincere thanks to all of the members of the Scientific Committee, and especially to Vice Chair Johann de Bono, for their insight, advice and support and of course, to all the national and international speakers who have agreed to make the 12th NCRI Cancer Conference such a stimulating and informative meeting. None of this would be possible without the excellent support from the NCRI Conference Team for their dedication and enthusiasm for keeping the highest standards of multi-disciplinary cancer research the top priority at our exciting annual NCRI Cancer Conference.

Have a great Conference!

![Signature]

**Professor Caroline Dive**  
Chair, 2016 NCRI Conference Scientific Committee  
Cancer Research UK Manchester Institute, UK

PS: Make sure you share your experience with colleagues back in your institutions and indeed with the wider scientific community. If you are on Twitter, remember to use #NCRI2016 and our Twitter handle @NCRI_partners but please be mindful of not posting pictures of unpublished data!
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Welcome message from the Chair of Trustees and Director of the National Cancer Research Institute (NCRI)

Welcome to the 12th annual NCRI Cancer Conference, the UK's largest cancer research meeting, which brings together the cancer research community to share knowledge and ideas and to develop collaborations.

As always, the NCRI is proud to host the Conference, as it represents the collaborative and inclusive approach we promote. The NCRI brings together a broad range of Partners from across the spectrum of cancer research who collectively spend around £500m on research related to cancer every year.

The unique partnership of NCRI gives us oversight of the entire UK cancer research landscape, identifying areas of need and opportunity as well as reducing duplication of effort. By driving collaboration, we as a Partnership are able to accelerate progress of research relevant to cancer to improve health and wellbeing.

This year gives us a unique opportunity to consult you on the draft Strategic Plan of the NCRI Partnership. The new five year Strategic Plan is due to launch from April 2017 and outlines our purpose, goals, enablers and values. We have an interactive zone at the NCRI Meeting Point on the Lower Floor where all of you will have the chance to make your voice heard. We know that the unique value of the NCRI Partnership is in the diverse range of stakeholders that we work with and the perspectives that they bring, so please come along and tell us what you think.

This year’s Conference showcases a wide range of high quality research from across the UK and internationally, with topics as diverse as epidemiology, healthcare delivery, cancer prevention and living with and beyond cancer. Our carefully compiled scientific programme, for which our utmost gratitude must go to colleagues on the Scientific Committee, features over 50 educational sessions, poster presentations and offers networking opportunities.

You can use our new Conference App to schedule which sessions to attend, which posters to see and which stands to visit. If you are on Twitter, remember to share experiences using #NCRI2016 and our Twitter handle @NCRI_partners.

Sincerest thanks must also go to our speakers, who have travelled to Liverpool from across the UK and from overseas to present their research. We must also, of course, thank our Sponsors and Exhibitors as without their support we would be unable to deliver such a successful event. Finally, we must acknowledge the efforts of the NCRI Conference Team who work hard to ensure its success.

We sincerely hope you enjoy this year’s Conference; that you find it informative, inspiring and engaging. We are always looking at ways to improve the Conference experience so welcome any feedback you may have.

Baroness Delyth Morgan
Chair of Trustees, National Cancer Research Institute

Dr Karen Kennedy
Director, National Cancer Research Institute
The Scientific Committee and Conference Team are grateful to all the NCRI Partners for their continuing commitment to the concept of a multi-disciplinary cancer conference and for their financial support.

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**NCRI Partners**

Biotechnology and Biological Sciences Research Council  
www.bbsrc.ac.uk

HSC Public Health Agency (R&D Division)  
www.research.hscni.net

Bloodwise  
www.bloodwise.org.uk

Macmillan Cancer Support  
www.macmillan.org.uk

Breast Cancer Now  
www.breastcancernow.org

Marie Curie  
www.mariecurie.org.uk

Cancer Research UK  
www.cancerresearchuk.org

Medical Research Council  
www.mrc.ac.uk

Chief Scientist Office, Scottish Government Health Directorates  
www.cso.scot.nhs.uk

Pancreatic Cancer Research Fund  
www.pcrf.org.uk

Children with Cancer UK  
www.childrenwithcancer.org.uk

Prostate Cancer UK  
www.prostate-cancer.org.uk

Department of Health  
www.dh.gov.uk

Roy Castle Lung Cancer Foundation  
www.roycastle.org

Economic and Social Research Council  
www.esrc.ac.uk

Tenvosus Cancer Care  
www.tenvosus.org.uk

Health and Care Research Wales  
www.healthandcareresearch.gov.wales

Wellcome Trust  
www.wellcome.ac.uk

Worldwide Cancer Research  
www.worldwidecancerresearch.org

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Join us next year: 5-8 November 2017  
conference.ncri.org.uk
Scientific Committee

The NCRI is grateful to the Scientific Committee for their support in developing the programme and maintaining the high standards of this Conference.

Caroline Dive
(2016 Chair),
Cancer Research UK
Manchester Institute,
UK

Michelle Garrett
University of Kent,
UK

John Rouse
NCRI Consumer Representative, UK

Johann de Bono
(2016 Vice-Chair),
Royal Marsden Hospital and
The Institute of Cancer Research,
London, UK

Eyal Gottlieb
Cancer Research UK
Beatson Institute,
Glasgow, UK

Manuel Salto-Tellez
Queen's University Belfast, UK

Sue Bailey
Bristol-Myers Squibb Pharmaceuticals Ltd, UK*

Kevin Harrington
The Institute of Cancer Research,
London and the Royal Marsden NHS Foundation Trust,
London, UK

Matt Seymour
NIHR Clinical Research Network: Cancer, Leeds, UK
and National Cancer Research Institute

Linda Bauld
Cancer Research UK and University of Stirling, UK

Fiona Hemsley
The Institute of Cancer Research,
London, UK

Ricky Sharma
University College London Cancer Institute, UK

Jason Carroll
Cancer Research UK
Cambridge Institute,
UK

Miriam Johnson
University of Hull, UK

Charles Swanton
The Francis Crick Institute and
University College London Cancer Institute, UK

Richard Gilbertson
CRUK Major Cancer Centre, University of Cambridge,
UK

Karen Kennedy
National Cancer Research Institute,
UK

Alastair Thompson,
MD Anderson Cancer Centre, USA

Di Gilson
The Royal College of Radiologists, UK

Chris Lord
The Institute of Cancer Research,
London, UK

Lynda Wyld
BASO ~ The Association for Cancer Surgery, UK

Lesley Fallowfield
University of Sussex, UK

Xin Lu
Ludwig Institute for Cancer Research,
University of Oxford, UK

*Chair of Industry Consultation Group – no involvement in presentation or speaker selection
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Join us next year: 5-8 November 2017
2016 Conference supporters

Support grants are provided towards the 2016 programme, which has been designed independently by your peers (see Scientific Committee on page 6).

AbbVie

Astex

AstraZeneca

BASO – The Association for Cancer Surgery

Breast Cancer Now

Bristol-Myers Squibb

British Association for Cancer Research

British Journal of Cancer

Cancer Research UK

Cancer Research UK

Cancer Research UK

Cancer Research UK

Cancer Research UK and Medical Research Council

Cancer Research UK and Medical Research Council

Cancer Research UK and Medical Research Council

Celgene

European Association for Cancer Research

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National Institute for Health Research

Roche

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The Company of Biologists

The Institute of Cancer Research

The Royal College of Radiologists
2016 Media partners

We appreciate the support of all media partners.

Advances in Modern Oncology Research (AMOR)

Bentham Science

BioMed Central Ltd

Cancer World Magazine, European School of Oncology

Cancers – An Open Access Oncology Journal from MDPI

CrowdReviews.com, LLC

eCancer

European Medical Journal

International Society of Paediatric Oncology (SIOP)

Karger Publishers

Nature Reviews Cancer

Nature Reviews Clinical Oncology

Oncology News

Technology Networks

The Lancet Oncology

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Join us next year: 5-8 November 2017

conference.ncri.org.uk
Useful information

Accommodation
For accommodation enquiries, please visit our website at conference.ncri.org.uk/accommodation.

Browsing abstracts
All abstracts are available to view on the Conference App and online at conference.ncri.org.uk/abstracts/2016.

Car parking
Convenient parking is available opposite the BT Convention Centre at The Liverpool Waterfront Car Park at a cost of £15 per 24 hours.

Choose your session
While every attempt has been made to put popular sessions in large rooms, some sessions may be oversubscribed on the day. Entry is on a first come first served basis, so please arrive early at your chosen session.

As a courtesy to speakers and other delegates, please remember to turn off your mobile phone before entering the lecture halls.

Please wear your delegate badge at all times while at the venue and social events.

Cloakroom and lost property
A cloakroom is available at the entrance of the BT Convention Centre. NCRI does not accept any liability for lost property. Please visit the BT Convention Centre helpdesk in the entrance to report or reclaim lost property.

CPD certificates
The 2016 NCRI Cancer Conference has been approved by the Federation of the Royal Colleges of Physicians for 24 category 1 (external) CPD credits.

Please complete the appropriate section of the electronic Conference survey to request your certificate. A link to the survey will be sent to you after the Conference.

Certificates will only be sent electronically and may take up to 10 working days.
**NCRI App Challenge**
The App Challenge is an interactive feature in which attendees can visit trade stands and other designated points in the Exhibition, collecting codes along the way.

There will be daily prize draws for the highest scoring entrants and prizes will include Amazon vouchers, coffee machines, a Fitbit and many more.

Winners will be chosen at the end of each day and contacted via the App.

**Poster sessions**
Posters must be installed and removed according to the below timings:
Session A: Install by 17.25 on Sunday 6 November, remove by 21.00 on Monday 7 November
Session B: Install by 10.25 on Tuesday 8 November, remove by 12.30 on Wednesday 9 November

Posters are available for informal viewing throughout the allocated days of their presentation. Presenters are encouraged to provide handouts for when they are not present to speak to participants.

All posters must be taken down by the designated time on the last day of the allocated session, as posters left up after this time will be removed. The Conference team is not responsible for storing posters that have been removed.

**Press office**
The Conference press office is located in Room 5 on the first floor. To contact the press team, please call +44 (0)151 707 4642/4643/4644/4645/4646.

**Recycling facilities**
The BT Convention Centre is one of the most eco-friendly meeting venues in the UK. Please use the facilities provided to recycle paper. If you wish to recycle your bag, badge and lanyard, pass them to the Conference staff.

**Registration**
Registration is situated on the first floor and is open:
- Sunday 6 November 09.00-18.30
- Monday 7 November 08.00-19.00
- Tuesday 8 November 08.00-18.00
- Wednesday 9 November 09.00-12.00

**Taxis**
A taxi rank is situated on the riverside entrance of the BT Convention Centre. Alternatively, please call +44(0)151 298 2222 or +44(0)151 207 2222.
The trade exhibition and poster presentations, along with all refreshments and networking receptions will take place in Hall 2 on the Lower Floor of the venue.
Choosing the right session

**Plenary lectures**
These feature experts from the UK and overseas, invited by the Scientific Committee to give plenary lectures. All have been briefed to give talks that are accessible to a wider audience. Their talks may address a broad area of work, summarise their own research, or discuss an important area of policy relating to cancer. There are plenary lectures on each of the four days of the Conference.

**Symposia**
Each symposium comprises three talks from speakers of international standing around a broad theme. The aim is to consider one topic from three different angles, with a mix of disciplinary approaches. A symposium may cover basic, translational and clinical research, or it may reach into areas such as social and behavioural studies, as well as approaches to prevention. Symposia should help you to see your own work within a broader context of studies beyond your own expertise.

**Parallel sessions**
These more specialist sessions mainly attract professionals in the areas on which they focus. On each day there will be one parallel session intended to be accessible to a lay audience, though professionals frequently attend these sessions too. Parallel sessions cover a range of themes; see page 14 for further information.

**Workshops**
Workshops are organised on a demand-led basis and vary somewhat in format. Some are educational or commercially-led training sessions, while others debate a hot topic or discuss the availability of research resources such as biosamples or datasets. Workshops are intended to include much more audience participation and are essentially discussion forums.

**Clinical Trials Showcase**
The Scientific Committee select abstracts on clinical trials from those that are submitted. Trials selected for presentation in the Clinical Trials Showcase are often practice-changing, of high quality and/or are presenting new data.
Session themes

**Diagnosis and therapy**
- Screening technologies and diagnostic markers.
- Estimation of prognosis and identification of individuals at increased risk of cancer.
- Factors associated with stage of diagnosis and clinical outcome.
- All types of therapy and all phases of development and testing.

**Epidemiology and prevention**
- Population-based research aimed at understanding causation, incidence, trends, and risk (such as environmental and genetic risk).
- Research on prevention e.g. lifestyle and nutritional factors, including individual and community interventions.

**Healthcare delivery**
- Quality and cost of healthcare and coordination of care.
- Development and testing of healthcare delivery methods.
- Access to healthcare including primary care and screening services.

**Information, patients and the public**
- Public policy issues, ethics and confidentiality.
- Education and communication about cancer.
- Involvement of patients and public in deciding research priorities.
- Patient-led research.

**Supportive, palliative care, survivorship**
- Living with and beyond cancer: physical, psychological and social impacts and their management.
- Research into care at the end of life.

**The cancer cell and model systems**
- Molecular and cellular mechanisms of oncogenesis and tumour suppression.
- The tumour microenvironment.
- Cell biology relevant to cancer.
This year marks a new partnership with BASO ~ The Association for Cancer Surgery which will be holding its annual Scientific Conference alongside the NCRI Cancer Conference. This collaboration is designed to help to support and foster increased surgical research activity.

There will be BASO sessions throughout Sunday 6 November (see separate programme for further details) which are open to all Conference attendees. On Monday 7 November, there will be dedicated surgical sessions across the full day. See below for further information or see the full programme for Monday from page 34.

**08.15 – 09.00, Room 11**
**BASO–ACS Breast session: Breast reconstruction**
Hosted by Lynda Wyld and Tibor Kovacs, BASO–ACS

*The oncological safety of nipple sparing mastectomy: The European INSPIRE Project*
Isabel Rubio, Hospital Universitario Vall d´Hebron, Barcelona, Spain

*The oncological safety of lipomodelling in breast cancer*
Ms Siobhan Laws, Royal Hampshire County Hospital, UK

**09.45 – 10.25, Hall 1A**
**BASO–ACS plenary session: BJS lecture**
Chaired by Lynda Wyld and Michael Douek, BASO–ACS

*Challenges in the management of breast cancer in low and middle income countries*
Cheng-Har Yip, Breast Surgery International, Malaysia

**11.00 – 12.30, Room 11**
**BASO–ACS and BAHNO symposium**
Hosted by Garth Cruickshank, BASO–ACS and Mike Fardy, BAHNO

*HPV vaccines – how to get the maximum protection*
Margaret Stanley OBE, University of Cambridge, UK

*The management of oropharynx cancer in the HPV Era*
Terry Jones, NIHR Clinical Research Network: North West Coast, UK

*Sentinal lymph node biopsy in head and neck cancer: Good ideas need proper trials*
Mark McGurk, King’s College London and Guy’s and St Thomas’ Foundation Trust, UK
14.00 – 16.00, Room 11
BASO~ACS robotic surgery session
Hosted by William Cross, St James’s University Hospital, Leeds, UK

A robotic gynaecological perspective
Simon Butler-Manuel, The Royal Surrey County Hospital, UK

A robotic thoracic perspective
Sasha Stamenkovic, Newcastle Cancer Centre, UK

A robotic urological perspective
Ben Challacombe, Guy’s and St Thomas’ NHS Foundation Trust, London, UK

A robotic colorectal perspective
Mark Gudgeon, Frimley Park Hospital, UK

A robotic head and neck perspective
Vinidh Paleri, Freeman Hospital, Newcastle, UK

We would like to thank Intuitive Surgical for their support in supplying the da Vinci Xi for this session.

17.35 – 18.35, Room 11
BASO~ACS and AUGIS session
Hosted by Hassan Malik, BASO~ACS and Giles Toogood, AUGIS

Advancing outcomes in pancreatic cancer: From bench to bedside
John Neoptolemos, University of Liverpool, UK

Surgical training at a distance
James Garden, University of Edinburgh, UK
The Royal College of Radiologists at the Conference

The Royal College of Radiologists (RCR) is delighted to be working with the NCRI for the fifth consecutive year, delivering sessions by a world-class faculty to the NCRI Cancer Conference on Tuesday 8 November. Join your RCR colleagues for these talks. See the full programme for Tuesday from page 82.

**11.00 – 12.30, Room 3B**

*Where are the gains in technical radiotherapy?*

Hosted by Jeanette Dickson, The Royal College of Radiologists

**Introduction by the host**

**George Edelstyn lecture: Protons**

Steve Hahn, The University of Texas MD Anderson Cancer Center, USA

**Advances in SABR**

Maria Hawkins, Gray Institute for Radiation Oncology and Biology, Oxford University, UK

**Motion management in radiotherapy**

Marcel van Herk, The Christie Hospital, Manchester, UK

**12.30 – 14.00, Hall 2**

*Poster viewing*, including a dedicated radiotherapy and radiobiology section.

These posters will be designated starting with ‘RCR’. Download the Conference App for full details.

**13.00 – 14.00, Room 3B**

*The Royal College of Radiologists proffered paper session*

One of the presentations in this session will be awarded an RCR Ross Prize for the best oral presentation, as judged by an RCR judging panel.

**16.30 – 18.30, Room 3B**

*Improving outcomes with drug/radiotherapy combinations*

Hosted by Geoffrey Higgins, University of Oxford, UK

**Why don’t drug companies provide access to drug for use in combination with radiotherapy?**

Anthony Chalmers, Beatson West of Scotland Cancer Centre, Glasgow, UK

**Immunotherapy and radiotherapy**

Tim Illidge, Cancer Research UK Manchester Institute, UK

**Novel drug/radiotherapy combinations**

Philippe Lambin, University of Maastricht and the MAASTRO Clinic, The Netherlands
Networking

In the past few years 95% of colleagues said they would recommend the Conference to others. And this is not just because of the variety and quality of the science presented: networking – whether planned or serendipitous – is an essential motive for many to come to the Conference year in, year out. Here are some of the key events for you to network with old colleagues, meet fellow participants and speakers and start new collaborations.

**Opening reception**
**Sunday 6 November, 18.00 – 20.00, Hall 2**
Join fellow participants for a welcome drink in the Exhibition Hall. Use this opportunity to visit exhibitors and hear about the latest initiatives, services and products from over 70 organisations.

**Exhibition hall and posters**
**From 15.00 on Sunday 6 November for all refreshments and receptions until 12.00 on Wednesday 9 November, Hall 2**
See the newest initiatives, as well as some of the best products and services available to cancer research professionals in the exhibition hall, and talk to expert staff about your needs. The exhibition hall is the place to meet fellow participants; all refreshments and poster sessions will take place in the hall.

**Drinks reception**
**Monday 7 November, 18.35 – 20.00, Hall 2**

**Pre-Conference dinner drinks reception**
**Tuesday 8 November, 19.30 – 20.00, The Echo Arena, BT Convention Centre**
For Conference dinner ticket holders only

**Conference dinner (ticketed event)**
**20.00 onwards, Tuesday 3 November, The Echo Arena, BT Convention Centre**
This is a chance for you to relax and another opportunity to connect with fellow participants on the last evening of the Conference. At time of print, limited tickets were available for this event – please enquire before noon on Tuesday 8 November at the registration desk as only ticket holders will be admitted.
Prizes at the Conference

ACP McElwain Prize
Tuesday 8 November

12.00 – 12.15  
Hall 1C  
Clonal neoantigens elicit T cell immunoreactivity and sensitivity to immune checkpoint blockade  
Andrew Furness, University College London Cancer Institute, UK

Presentation in ‘Proffered paper session 2’

AstraZeneca Investigator Poster Prize
Tuesday 8 and Wednesday 9 November

Poster 196  
A novel oncogene-selective sensitivity to synchronous ferroptotic cell death following nutrient modulation  
Ioannis Poursaitidis, University of Liverpool, UK

British Association for Cancer Research (BACR)
Gordon Hamilton-Fairley Young Investigator Award
Sunday 6 and Monday 7 November

This prize will be selected from posters BACR1 – BACR7

British Association for Cancer Research (BACR)
AstraZeneca Young Scientist Frank Rose Award
Tuesday 8 November

12.15 – 12.30  
Hall 1C  
Cancer specific non-essential amino acid metabolism – a role for targeted dietary intervention in cancer therapy?  
Oliver Maddocks, University of Glasgow, UK

Presentation in ‘Proffered paper session 2’

British Association for Cancer Research (BACR)
Roger Griffin Prize for Cancer Drug Discovery
Tuesday 8 November

18.05 – 18.20  
Hall 1A  
Engineering potency and selectivity of chemical probes for functional elucidation and target validation  
Matthias Baud, University of Southampton, UK

Presentation in the ‘Advances in cancer drug discovery and development: Small molecule versus antibody-based strategies’ parallel session

Join us next year: 5-8 November 2017  
conference.ncri.org.uk
Prizes at the Conference (continued)

Cancer Research UK Prizes Ceremony
Monday 7 November

16.30 – 16.50
Hall 1A
Winners of the CRUK Research Prizes will be announced

16.50 – 17.30
Hall 1A
The Lifetime Achievement in Cancer Research Award winner:
Drug development and ovarian cancer – Lessons and personal reflections from a 40-year journey
Stan Kaye, Royal Marsden NHS Foundation Trust, UK

Children’s Cancer and Leukaemia Group (CCLG) McElwain Award
Monday 7 November

15.35 – 15.50
Room 3A
Inhibiting HOX-PBX binding in paediatric glioblastoma as a novel therapeutic treatment
William Rogers, University of Surrey, UK

Presentation in the ‘The dark side of the genome – structural catastrophes in paediatric cancers’ parallel session

European Association for Cancer Research Travel Awards
Sunday 6 and Monday 7 November

Poster 207
Axl promotes metastasis of pancreatic cancer by regulating an EMT programme
Lankhanh Trinh, Rechts der Isar Hospital, Germany

Poster 208
Administration-specific migration of Th1 cells modulates the host immune system, tumour microenvironment, and antitumoural efficiency during immunotherapy in pancreatic cancer
Christoph Griessinger, University of Tübingen, Germany

European Association for Cancer Research Travel Awards
Tuesday 8 and Wednesday 9 November

Poster 54
Application of next generation sequencing in liquid biopsy analysis
Eirini Papadopoulou, GeneKor Medical S.A., Greece
National Cancer Research Institute (NCRI) Prize Awards
Sunday 6 and Monday 7 November

Poster 19
Novel serum circulating miRNAs signatures for NSCLC treatment outcome
Julija Fadejeva, Vilnius University Joint Life Sciences Center, Lithuania

Poster 36
A novel NGS based cancer genetic testing solution on high-throughput sequencer BGISEQ-500
Shida Zhu, BGI, China

Poster 190
Endoscopic management of gastrointestinal bleeding in cancer patients with severe thrombocytopenia
Paul Hampel, Mayo School of Graduate Medical Education, USA

National Cancer Research Institute (NCRI) Prize Awards
Tuesday 8 and Wednesday 9 November

Poster 49
Detection of Circulating Tumour Cells for use as prognostic, predictive and pharmacodynamic biomarkers in neuroblastoma
Swathi Merugu, Newcastle University, UK

Poster 110
Metabolism biomarkers measured before prostate cancer diagnosis and second primary tumours: a prospective study in the Swedish AMORIS cohort
Cecilia Bosco, King's College London, UK

Poster 181
Capture of circulating tumour cells with epithelial and mesenchymal features for prostate cancer prognosis
Lei Xu, Barts Cancer Institute, London, UK

National Cancer Research Institute (NCRI) Werth Trust Award
Tuesday 8 November

17.50 – 18.05
Hall 1A
Enhancing responses to melanoma therapy with novel combinations of targeted therapy and immune checkpoint blockade
Robert Szczepaniak Sloane, The University of Texas MD Anderson Cancer Center, USA

Presentation in the ‘Advances in cancer drug discovery and development: Small molecule versus antibody-based strategies’ parallel session
### Prizes at the Conference (continued)

**Royal College of Radiologists (RCR) Ross Awards**  
**Tuesday 8 November**

<table>
<thead>
<tr>
<th>Time</th>
<th>Event</th>
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<tbody>
<tr>
<td>13.00 – 14.00</td>
<td>The RCR Ross Award for the best poster will be selected from posters RCR1 – RCR36</td>
</tr>
</tbody>
</table>

**The Company of Biologists Travel Awards**  
**Sunday 6 and Monday 7 November**

| Poster 32  | TMEPAI, a novel therapeutic target in triple negative breast cancer prevention and therapy  
            | Prajjal Singha, University of Texas Health Science Center, San Antonio, USA |
| Poster 60  | Disulfiram with or without metformin exhibits potent anti-cancer effects toward oesophageal squamous cell carcinoma in vivo  
            | Rupal Jivan, University of the Witwatersrand, Johannesburg, South Africa |
| Poster 124 | Cervical cancer survival among women referred to cancer centres in Ghana  
            | Yvonne Nartey, University of Otago, New Zealand |
| Poster 206 | Dual inhibition of the MEK5 and PI3K pathways synergistically reduces proliferation and viability in triple negative breast cancer cells  
            | Thomas Wright, Duquesne University, Pittsburgh, USA |

**The Company of Biologists Travel Awards**  
**Tuesday 8 and Wednesday 9 November**

| Poster 7   | Outcome of open distal pancreatectomy: An audit at tertiary care hospital  
            | Amyna Jiwani, Aga Khan University Hospital, Karachi, Pakistan |
| Poster 180 | Effect of melatonin treatment on the intratumour heterogeneity in breast cancer model  
            | André de Lima Mota, Faculdade de Medicina de São José do Rio Preto – FAMERP, Brazil |
CALL for PAPERS

We accept original research articles, case reports, comments, correspondence & reviews

Editor-in-Chief: Dr. Omar Abdel-Rahman, M.B.B.Ch., M. Sc., MD, Ain Shams University, Egypt

419 Editorial Board Members, 57 Countries, 1 Common Goal

AMOR: Advances in Modern Oncology Research, is a peer-reviewed, gold Open Access publication that publishes the latest developments in cancer research for a broad audience of medical researchers and clinicians working in the field of oncology.

Our focus is on highlighting cancer research progress made via cross-disciplinary collaborations, and across different geographical areas.

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## Programme at a glance

### Welcome address

| 15.15 – 15.25 | Introduction from the Director of the NCRI |
| Hall 1A | Karen Kennedy, National Cancer Research Institute, UK |

### Plenary lectures

**Chaired by Caroline Dive, Cancer Research UK Manchester Institute, UK**

| 15.25 – 16.05 | State of the art in small cell lung cancer |
| Hall 1A | Charles Rudin, Memorial Sloan Kettering Cancer Center, USA |

| 16.05 – 16.45 | Headlines, hype and hope: How should we talk about cancer research? |
| Hall 1A | Kat Arney, Freelance science writer and science broadcaster, London, UK |

| 16.45 – 17.25 | Yeast cell cycle, what has this taught us? |
| Hall 1A | Iain Hagan, Cancer Research UK Manchester Institute, UK |

### Consumer session

| 17.30 – 18.30 | What is cancer? A 21st century answer |
| Room 3B | Martin Christlieb, University of Oxford, UK |

### Debate

| 17.30 – 19.00 | New tricks for old drugs? This house believes research into repurposing existing medications and optimising use of current breast cancer treatments should be prioritised above research into developing novel agents |
| Room 3A | Hosted by Breast Cancer Now |

### Opening reception, networking, exhibition and poster viewing

| 18.00 – 20.00 | |
| Hall 2 |
Plenary abstracts

State of the art in small cell lung cancer

15.25 - 16.05
Hall 1A

Charles Rudin,
Memorial Sloan Kettering Cancer Center, USA

At the time of printing, this abstract had not been received. Check the Conference App for further details.

Headlines, hype and hope: How should we talk about cancer research?

16.05 - 16.45
Hall 1A

Kat Arney,
Freelance science writer and science broadcaster, London, UK

The media loves a cancer story, good or bad, with bold headlines announcing every possible cause or potential cure. For scientists and doctors these seemingly simplistic statements can be frustrating or even infuriating. For patients and the public they can stoke fear, cause confusion or raise false hopes. But how do these stories get into the public eye in the first place? And how, as a community, can we help to shape them and make them more accurate and engaging? Award-winning science writer, broadcaster and former Science Communications Manager at Cancer Research UK, Kat Arney, shares her thoughts.
The identification of the conserved regulators of yeast cell cycle precipitated a step change in understanding human cell fate decisions through the definition of CDK inhibitors, many of which remain key biomarkers in grading transformation today. More importantly, yeast cell cycle studies produced the concept of coupling unlinked biochemical events through “checkpoint” control. The ensuing characterisation of checkpoint pathways in yeast rapidly led to the realisation that identical checkpoints underpinned the success and failure of many DNA damaging and anti-mitotic therapies. The obvious logic of compromising the checkpoint that maintains survival following insult has pushed checkpoint kinase inhibitors into clinical trials.

Systematic large-scale tissue culture screens, genome editing and expanding catalogues of tumour mutations of this post-genomic era have led to the view that yeast may have told all they have to tell. However, their potential to offer highly effective short cuts in deciphering the architecture of signalling networks is as powerful as ever, particularly when redundancy and feedback controls confound interrogation. Certainly, fission yeast is defining core elements in the coupling of growth and division by TOR signalling and our work on protein phosphatases highlights how these lowly beasts continue to define completely unanticipated, fundamental concepts in cellular regulation.
If our enemy is to be defeated, we must understand our enemy and how it works. During the first decade of this century a more complex and more realistic picture of cancer emerged based on the observed behavior of cancer cells. In this session we will look at what makes a successful cancer cell and how this can lead to a highly variable disease that might need personalised solutions.

The session will be interactive with opportunities for discussion and involvement. The session will assume no prior knowledge and is suitable for anyone with an interest in knowing our enemy better.
Debate

New tricks for old drugs? ‘This house believes research into repurposing existing medications and optimising use of current breast cancer treatments should be prioritised above research into developing novel agents’

Research has uncovered new effective applications for existing treatments in the fight against breast cancer. Bisphosphonates have been shown to prevent metastasis of breast cancer to the bones and reduce breast cancer deaths. Tamoxifen and anastrazole, drugs primarily used in treatment have now been shown to be effective preventive agents. These existing therapies have the advantage that they are already approved with known safety profiles and are cheap due to expired patents.

On the other hand, some of the biggest breakthroughs in treating breast cancer have come from newly discovered drugs acting on novel targets, such as the anti-HER2 drug trastuzumab (Herceptin) which has significantly improved prognosis for thousands of women. Research to predict new therapeutic targets remains a very large part of the UK’s research portfolio and new agents are often seen as the only hope for patients with aggressive subtypes of cancer, or those who develop treatment resistance. As a result, new targeted and immuno-therapies are often hailed as the greatest chance we have in preventing deaths from breast cancer. Experience shows however that many promising agents do not live up to their potential. Historically, the attrition rate for novel therapeutics has been disappointingly high as they fail to result in significant improvements when compared with standard of care treatments in later phase trials. Given the time, costs and risks of developing new drugs, is this the best research strategy to help patients now?

Our question is, given the potential surrounding repurposing and improving available treatments, should we constrain investment in discovering and developing brand new treatments? Or should we focus research into highly innovative drugs with novel mechanisms – with the aspiration that there are potentially lifesaving therapies on the horizon that could hold the key to preventing deaths from breast cancer?

Speakers for:
Robert Coleman, University of Sheffield, UK
David Dodwell, Leeds Teaching Hospitals NHS Trust, UK

Speakers against:
Paul Workman, The Institute of Cancer Research, London, UK
Susan Galbraith, AstraZeneca, UK

17.30 – 19.00
Room 3A

Chaired by
Judith Bliss, The Institute of Cancer Research, London, UK
Current Cancer Drug Targets aims to cover all the latest and outstanding developments on the medicinal chemistry, pharmacology, molecular biology, genomics and biochemistry of contemporary molecular drug targets involved in cancer, e.g. disease specific proteins, receptors, enzymes, genes.
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44 Symposia abstracts
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Key

**Diagnosis and therapy** 1
**Epidemiology and prevention** 2
**Healthcare delivery** 3
**Information, patients and the public** 4
**Supportive, palliative care, survivorship** 5
**The cancer cell and model systems** 6
### Programme at a glance

#### Workshops

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| 08.15 – 09.00 | Room 3B | De-mystifying today’s science  
Hosted by **Elaine Vickers**, Science Communicated, Sheffield, UK |
| 08.15 – 09.00 | Room 3A | BACR educational workshop: Bioinformatics for the uninitiated  
Hosted by **Crispin Miller**, Cancer Research UK Manchester Institute, UK |

#### Scientific symposium

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| 08.15 – 09.00 | Room 11 | BASO–ACS Breast session: Breast reconstruction  
Hosted by **Lynda Wyld** and **Tibor Kovacs**, BASO–ACS |

#### Plenary lecture

Chaired by Richard Gilbertson, CRUK Major Cancer Centre, University of Cambridge, UK

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| 09.05 – 09.45 | Hall 1A | Elucidating lung stem cells and cancer at single cell resolution  
**Mark Krasnow**, Stanford University School of Medicine, USA |

**BASO–ACS plenary session: BJS lecture**

Chaired by Lynda Wyld and Michael Douek, BASO–ACS

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| 09.45 – 10.25 | Hall 1A | Challenges in the management of breast cancer in low and middle  
income countries  
**Cheng-Har Yip**, Breast Surgery International, Malaysia |

#### Networking, exhibition and poster viewing

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#### Symposia

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| 11.00 – 12.30 | Hall 1A | Cancer prevention at the population level: How can we translate  
research evidence into policies that help prevent cancer?  
Hosted by **Linda Bauld**, Cancer Research UK and University of Stirling, UK |
| 11.00 – 12.30 | Room 3B | Stem cells and cancer  
Hosted by **Tariq Enver**, University College London Cancer Institute, UK |
| 11.00 – 12.30 | Hall 1B | Noncoding RNAs in cancer  
Hosted by **Crispin Miller**, Cancer Research UK Manchester Institute, UK |
| 11.00 – 12.30 | Room 11 | BASO–ACS and BAHNO symposium  
Hosted by **Garth Cruickshank**, BASO–ACS and **Mike Fardy**, BAHNO |

#### Workshop

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| 11.00 – 12.30 | Hall 1C | Molecular diagnostics: Lessons being learnt  
Hosted by **Karin Oien**, University of Glasgow, UK |
Lunch, networking, exhibition and poster viewing

12.30 – 14.00
Hall 2

Dragons’ Den workshop

13.00 – 14.00  Hosted by NCRI Consumer Forum
Room 3A

Parallel sessions

14.00 – 16.00  Functional imaging for influencing management: Paradigm shift or pipe dream?
Room 4  Hosted by Nandita deSouza, The Institute of Cancer Research, London, UK

14.00 – 16.00  Early detection and prevention
Hall 1C  Hosted by Sir Harpal Kumar, Cancer Research UK, London, UK

14.00 – 16.00  The dark side of the genome – structural catastrophes in paediatric cancers
Room 3A  Hosted by Richard Gilbertson, CRUK Major Cancer Centre, University of Cambridge, UK

14.00 – 16.00  Liquid biopsy
Hall 1B  Hosted by Nicholas Turner, The Institute of Cancer Research, London, UK

14.00 – 16.00  Models of aneuploidy and centrosomal imbalance
Room 12  Hosted by Iain Hagan, Cancer Research UK Manchester Institute, UK

14.00 – 16.00  Using viruses to treat cancer
Hall 1A  Hosted by Alan Melcher, The Institute of Cancer Research, London, UK

14.00 – 16.00  BASO~ACS robotic surgery session
Room 11  Hosted by William Cross, St James’s University Hospital, Leeds, UK

Proffered paper session 1

14.00 – 16.00  Chaired by Johann de Bono, The Institute of Cancer Research, London, UK
Room 3B

Networking, exhibition and poster viewing

16.00 – 16.30
Hall 2

Cancer Research UK prize ceremony

16.30 – 16.50  Presented by Peter Johnson, Chair of the Cancer Research UK Prizes Selection Panel
Hall 1A
Programme at a glance (continued)

Plenary lecture
Chaired by Peter Johnson, Chair of the Cancer Research UK Prizes Selection Panel

16.50 – 17.30  
**Hall 1A**  
The Lifetime Achievement in Cancer Research Award winner: Drug development and ovarian cancer – Lessons and personal reflections from a 40-year journey  
**Stan Kaye**, Royal Marsden NHS Foundation Trust, UK

Clinical Trials Showcase part 1
Hosted by Matt Seymour, NIHR Clinical Research Network: Cancer, Leeds, UK and National Cancer Research Institute

17.35 – 17.50  
**Hall 1A**  
TACE 2: A randomised placebo-controlled, double-blinded, phase III trial of sorafenib in combination with transarterial chemoembolisation (TACE) in patients with unresectable hepatocellular carcinoma  
**Tim Meyer**, UCL Cancer Institute, UK

17.50 – 18.05  
**Hall 1A**  
Quality of life results of BIG 02-04 MRC EORTC SUPREMO trial of chest wall radiotherapy in patients with intermediate risk stage II breast cancer after mastectomy  
**Galina Velikova**, St James's University Hospital, Leeds, UK

18.05 – 18.20  
**Hall 1A**  
ESPAC-4: An international randomised phase-3 trial of adjuvant combination chemotherapy using gemcitabine (GEM) and capecitabine (CAP) versus mono therapy gemcitabine in resected pancreatic cancer  
**John Neoptolemos**, Cancer Research UK Liverpool Cancer Trials Unit, UK

18.20 – 18.35  
**Hall 1A**  
First Results of ART DECO (CRUK/10/018): A randomised trial of dose escalated intensity modulated radiotherapy (DE-IMRT) versus standard dose IMRT (ST-IMRT) in locally advanced head and neck cancer  
**Chris Nutting**, The Royal Marsden NHS Foundation Trust, London, UK

Scientific symposium

17.35 – 18.35  
**Room 11**  
BASO~ACS and AUGIS session  
Hosted by **Hassan Malik**, BASO~ACS and **Giles Toogood**, AUGIS

Workshop

17.35 – 19.05  
**Room 3A**  
Women in science  
Hosted by **Caroline Dive**, Cancer Research UK Manchester Institute

Drinks reception, networking, exhibition and poster viewing

18.35 – 20.00  
**Hall 2**

Chairs’ evening (by invitation only)

19.00 – 21.00  
**Pullman Hotel**  
Hosted by **Baroness Delyth Morgan**, Chair of Trustees, National Cancer Research Institute and **Caroline Dive**, Chair of the 2016 Scientific Committee

Download the App for the latest updates
Elucidating lung stem cells and cancer at single cell resolution

At the time of printing, this abstract had not been received. Check the Conference App for further details.

BASO~ACS plenary session: BJS lecture
Challenges in the management of breast cancer in low and middle income countries

The incidence of breast cancer is rising in low- and middle-income countries (LMICs) due to changes in lifestyle which increase risk for developing breast cancer, such as having less children, breast feeding for shorter periods, later age at first childbirth and less physical activity. Lack of early detection programmes as well as sociocultural barriers lead to breast cancer presenting in late stages. Coupled with suboptimal access to effective treatment, breast cancer mortality is high. A multidisciplinary approach to treatment gives the best results. Unfortunately the shortage of almost all specialists involved in the management of breast cancer leads to a lack of timely and appropriate care. Most important is the lack of a reliable pathology service to provide an adequate pathology report with prognostic and predictor information to allow optimal oncological treatment. Stratification of clinical practice guidelines based on resource level can ensure that women will have access to treatment even in a low-resource setting. Advocacy and civil society play a role in galvanizing the political will required to meet the challenge of providing adequate care to women with breast cancer in LMICs. Collaboration between high income countries and LMICs could be a strategy in facing these challenges.
Forty years ago, the treatment of cancer in general, and ovarian cancer in particular, looked very different from the scene today. In this talk, I shall describe my personal perspectives on this 40-year journey, focusing on developments in ovarian cancer as the paradigm for a solid tumour in which rational, mechanism-based early clinical trials have played a key role in the improvements in treatment which we have seen over this period. Our recognition of the important role of PARP inhibitors in treating patients with BRCA mutations is an excellent example of this and I will explain the background and future prospects.

I shall also reflect on some lessons that I have learnt at the three sites where I have been fortunate to work alongside exceptionally talented colleagues, in Glasgow and in London, and at all stages teamwork has been the watchword. Many challenges remain, perhaps the greatest being the evolution of drug resistance, which is a major limitation to most forms of systemic cancer treatment. But the ever-strengthening translational links between the laboratory-based and the clinician scientist give real grounds for optimism for the future, in which CRUK is certain to play a key role.
TACE 2: A randomised placebo-controlled, double-blinded, phase III trial of sorafenib in combination with transarterial chemoembolisation (TACE) in patients with unresectable hepatocellular carcinoma

**Background**

TACE is the standard-of-care for patients with intermediate stage HCC while sorafenib (S) is the current standard for advanced disease. TACE 2 was designed to determine whether TACE + S improves progression free survival (PFS) compared to TACE alone.

**Method**

Patients were randomised 1:1 to continuous S (400mg BD) or placebo (P). Inclusion criteria included; unresectable HCC, ECOG PS ≤1 and Child Pugh A liver score. Study-drug was commenced at randomisation and TACE performed at 2-5 weeks using drug eluting beads (DEB) loaded with 150mg doxorubicin. Further TACE was performed on demand. Primary outcome measure (OM) was PFS. Secondary OM included overall survival (OS), response rate and safety. Target recruitment was 412 to detect a (Hazard Ratio) HR of 0.72 with 2-sided significance α=0.05 and 85% power. A planned interim futility analysis (IFA) was performed at 45% of trial events.

**Results**

At the IFA, 294 had been randomised from 20 UK sites. Median age 67 yrs, 169 (58%) PS 0. In 229 cirrhotic patients, liver disease was: 43% alcohol, 24% HCV; 13% HBV; 38% other. Median PFS for the S and P group was 7.8 (95% CI 5.9, 10.0) and 7.7 (95% CI 5.9, 10.5) months; (HR) of 1.03 (95% CI 0.75, 1.42 p=0.85). For the S and P groups: median OS was 18.8 (95% CI 12.3, 24.0) and 19.6 (95% CI 14.8, 24.0) months; there were 77 and 78 SAEs; 195 and 256 TACE procedures. Median duration of S and P was 5.9 and 7.7 months; median of patient average daily dose was 649mg and 800mg.

**Conclusion**

The TACE 2 trial provides no evidence that addition of S to DEB-TACE improves PFS or OS in European patients with intermediate HCC. Alternative systemic therapies need to be evaluated in combination with TACE to improve outcomes for this patient population.

Quality of life results of BIG 02-04 MRC EORTC SUPREMO trial of chest wall radiotherapy in patients with intermediate risk stage II breast cancer after mastectomy

**Background**

BIG2-04 MRC/EORTC SUPREMO is an international randomised phase III trial assessing chest-wall irradiation in women with intermediate risk breast cancer following mastectomy (ISRCTN61145589). Patients were prospectively randomised to receive chest-wall radiotherapy or not. The primary endpoint is overall survival (OS at 10 years) with secondary endpoints of chest-wall/regional recurrence, disease-free survival, quality of life (QOL) and costeffectiveness. The first OS analysis will be at 300 events (deaths). Here we report QOL results at 2 years.

**Method**

Between April 2007-May 2013 the trial recruited 1688 patients internationally; 990 were
eligible for the QOL study (UK centres only). Patients completed the EORTC QLQ-C30 and BR23 questionnaires, Hospital Anxiety and Depression Scale (HADS) and EQ5D at baseline (pre-randomisation), 1 and 2 years. 947 patients (96%) returned the baseline questionnaires, 85% year 1 and 81% year 2 questionnaires. Repeated mixed effects methods were used to analyse the data, with baseline score and age included as covariates in the model. SAS’s PROC MIXED was utilised due to its ability to deal with data missing at random.

Results
Patients randomised to radiotherapy reported worse chest-wall symptoms (pain, swelling, oversensitivity and skin problems in the “area of the affected breast”) (p=0.013), although an improvement in symptoms was seen between year 1 and 2 post-randomisation (p=0.009). No significant between-group differences were observed for arm symptoms, body image, fatigue, pain, overall QOL, physical functioning or HADS scores. An age effect was found: younger patients reported significantly worse chest-wall (p=0.0004) and arm symptoms (p=0.005), body image problems (p=0.0007) and anxiety (p=0.0015).

Conclusion
Chest wall radiotherapy led to more chest wall symptoms up to 2 years post-randomisation, but reassuringly there was no impact on arm symptoms, body image, overall QOL and patient functioning for this group. However, worse QOL seen in younger women will need further investigation.

ESPAC-4: An international randomised phase-3 trial of adjuvant combination chemotherapy using gemcitabine (GEM) and capecitabine (CAP) versus mono therapy gemcitabine in resected pancreatic cancer

18.05 – 18.20
Hall 1A
John Neoptolemos,
Cancer Research
UK Liverpool Cancer Trials Unit, UK

Background
The ESPAC-3 trial compared adjuvant GEM with 5-fluorouracil/folinic acid for resected pancreatic cancer. GEM is the standard of care based on similar survival and less toxicity. ESPAC-4 aimed to determine whether combination chemotherapy with GEM/CAP improved survival compared to GEM monotherapy.

Method
Patients with pancreatic cancer were randomised to have either six 4wk cycles of IV GEM alone or GEM with oral CAP. The primary endpoint was overall survival; secondary endpoints were toxicity, relapse free survival, 2 and 5 year survival and quality of life. The trial was designed to detect a hazard ratio of 0.74. Using a two-sided alpha level of 0.05, 480 events were required to obtain 90% power.

Results
Between 732 patients were randomised with 730 included in the full analysis set (366 GEM, 364 GEM/CAP). Median age was 65 years, 57% were men. WHO performance status was 0, 1 or 2 in 42% 55% and 3% respectively. Postoperative median CA19-9 was 19 kU/L. Median maximum tumour size was 30 mm, 60% were R1 resections, 80% were node positive and 40% were poorly differentiated. On Dec 11 2015 the Independent Trial Steering Committee requested that the trial proceed to full analysis. Stratified log-rank for overall survival produced an HR=0.82 [95% CI, 0.68 – 0.98]; \( \chi^2 \) (1) = 4.61, P=0.032. Median
survival (months) for patients treated with GEM/CAP was 28.0 (95% CI, 23.5 – 31.5) and 25.5 (22.7 - 27.9) for GEM. The 5 year survival rate for the GEM/CAP was 28.8 (22.9 - 35.2) % and 16.3 (10.2 - 23.7) % for GEM. 196 out of 366 GEM patients in the safety set reported 481 grade 3/4 adverse events, while 226 out of 359 GEM/CAP patients reported 608 grade 3/4 adverse events (P=0.242).

**Conclusion**
Adjuvant GEM/CAP is the standard of care for resected pancreatic cancer.

**First Results of ART DECO (CRUK/10/018): A randomised trial of dose escalated intensity modulated radiotherapy (DE-IMRT) versus standard dose IMRT (ST-IMRT) in locally advanced head and neck cancer**

**Background**
Radical (chemo)radiotherapy offers curative treatment for patients with locally advanced laryngeal or hypopharyngeal cancer. IMRT permits safe dose-escalation and hence potential improved locoregional control given reduction in volume of normal tissue receiving high dose radiation.

**Method**
Patients with histologically squamous cell laryngeal or hypopharyngeal cancer (AJCC III-IVa/b) were randomised to DE-IMRT (primary tumour: 67.2Gray(Gy)/28 fractions(f), at risk nodes: 56Gy/28f) or ST-IMRT (primary tumour: 65Gy/30f, at risk nodes: 54Gy/30f). Patients received 2 cycles concomitant cisplatin, and up to 3 cycles platinum-based induction chemotherapy. Treatment allocation (1:1) used minimisation, balancing for centre, tumour site, nodal status and planned chemotherapy. Primary endpoint was locoregional failure- free rate (LRFFR) i.e. time to locoregional relapse or completion of radiotherapy in patients with persistent disease 3 months after treatment (clinical assessment). Target recruitment was 354 patients; 100 events required to detect improvement in 2-year LRFFR from 60% to 77.5% (two-sided $\alpha=0.05$, 90% power). LRFFR was analysed using competing risks methodology (with death as competing event), compared between groups by Gray’s test. Secondary endpoints ($\alpha=0.01$) included toxicity (CTCAEv4.0, LENT SOMA), patient-reported quality of life and overall survival.

**Results**
276 patients (138 ST-IMRT; 138 DE-IMRT) were randomised (2011-2015) from 32 UK centres. 84% were male, 66% had laryngeal tumours, 98% were NO-2, mean age was 62 years. Planned chemotherapy was 40% induction and concomitant, 48% concomitant only, 13% none. Recruitment stopped early due to planned interim futility analysis (subhazard ratio (SHR)>1 after 50% required events). 32/138 (23%) ST-IMRT and 37/138 (27%) DE-IMRT patients reported LRFFR events, SHR for DE-IMRT 1.22 (95%CI 0.76-1.94), p=0.42. Grade≥2 acute pharyngeal mucositis and 3 month post-RT pharyngeal dysphagia was higher with DE-IMRT (p=0.008, p=0.01 respectively).

**Conclusion**
There was no evidence for a statistically significant improvement in locoregional control with DE-IMRT compared with ST-IMRT. DE-IMRT was associated with worse toxicity profile.
## Cancer prevention at the population level: How can we translate research evidence into policies that help prevent cancer?

Hosted by Linda Bauld, Cancer Research UK and University of Stirling, UK

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<td>11.05 – 11.30</td>
<td>Cancer prevention at the population level: Standardised packaging</td>
<td>Ann McNeill, King's College London and the UK Centre for Tobacco and Alcohol Studies</td>
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<td>11.30 – 11.55</td>
<td>Minimum unit pricing for alcohol: The most cost-effective cancer prevention strategy of all?</td>
<td>Tim Stockwell, University of Vancouver, British Columbia, Canada</td>
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<td>11.55 – 12.20</td>
<td>Cancer prevention at the population level: Policies to prevent and treat obesity</td>
<td>Susan Jebb, University of Oxford, UK</td>
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<td>12.20 – 12.30</td>
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## Stem cells and cancer

Hosted by Tariq Enver, University College London Cancer Institute, UK

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<td>11.05 – 11.30</td>
<td>Therapeutic targeting of childhood medulloblastoma:</td>
<td>Sheila Singh, McMaster University, Canada</td>
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<td>Leukemic stem cell interactions with the microenvironment:</td>
<td>Dominique Bonnet, The Francis Crick Institute, UK</td>
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<td>11.55 – 12.20</td>
<td>Integrating stem and progenitor cell contribution to intestinal self renewal</td>
<td>Doug Winton, Cancer Research UK Cambridge Institute, UK</td>
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### Noncoding RNAs in cancer

Hosted by Crispin Miller, Cancer Research UK Manchester Institute, UK

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<td>11.05 – 11.30</td>
<td>Long non-coding RNAs – Messages from the dark matter of the cancer genome</td>
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<td><strong>Sven Diederichs</strong>, University of Freiburg and German Cancer Research Center (DKFZ), Germany</td>
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<tr>
<td>11.30 – 11.55</td>
<td>Control of cell division by long non-coding RNAs</td>
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<td>Hall 1B</td>
<td><strong>Duncan Odom</strong>, Cancer Research UK Cambridge Institute, UK</td>
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<td>Long non-coding RNAs and the regulation of the immune response</td>
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<td>Hall 1B</td>
<td><strong>Mark Lindsay</strong>, University of Bath, UK</td>
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### BASO~ACS and BAHNO symposium

Hosted by Garth Cruickshank, BASO~ACS and Mike Fardy, BAHNO

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<td>Room 11</td>
<td><strong>Margaret Stanley OBE</strong>, University of Cambridge, UK</td>
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<td>11.30 – 12.00</td>
<td>The management of oropharynx cancer in the HPV era</td>
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<td><strong>Terry Jones</strong>, NIHR Clinical Research Network: North West Coast, UK</td>
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<td><strong>Mark McGurk</strong>, King's College London and Guy's and St Thomas’ Foundation Trust, UK</td>
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Cancer prevention at the population level: How can we translate research evidence into policies that help prevent cancer?

**Introduction**

Over 40% of cancer cases in the UK are attributed to behavioural risk factors such as smoking, being overweight and alcohol consumption. While a better understanding of the mechanisms and links between these risk factors and cancer is valuable, as are interventions that focus on individual behaviour change, the most significant changes are likely to be achieved at the population level. This symposium focuses on new research underpinning policy change for cancer prevention. The speakers will outline how particular studies have influenced the introduction of population level policies, either currently underway or planned. In particular, the speakers will be discussing the evidence underpinning the implementation of standard packaging of tobacco products (and new studies to evaluate the impact), making the case for minimum unit pricing of alcohol and the need for a comprehensive obesity strategy.

**Cancer prevention at the population level: Standardised packaging**

Tobacco is the leading cause of preventable cancers worldwide and accounts for 64,000 new cases of cancer each year in the UK, where smoking is increasingly concentrated on those in more disadvantaged groups in society. It has long been recognised that tobacco advertising promotes smoking and evidence showed that as governments prohibited mainstream channels of advertising, the industry focused its attention on the tobacco pack instead, which has been referred to as the ‘silent salesman’. Standardised or plain tobacco packaging was proposed to counter this. Population level policies of this nature do not however lend themselves to randomised controlled trials, so other research designs are needed. This presentation will outline the range of studies that were developed to test the impact of branded versus standardised packaging which concluded that the latter was less appealing, made health warnings more salient and decreased the likelihood of believing that some tobacco brands were safer than others. Following implementation (Australia was the first country to introduce this policy in 2012 and the UK began implementation in April 2016) other studies have been carried out to explore the impact of the policy on attitudes, beliefs and behaviour. The difficulties of carrying out research in this area in the face of sustained opposition from the tobacco industry will also be discussed and the issues this raises for researchers across other areas of cancer prevention.

**Minimum pricing in theory and practice as a population wide cancer prevention strategy**

Alcohol’s estimated contribution to cancer incidence is likely to increase, as causal relationships with additional cancers (e.g. prostate, melanoma, pancreas) become accepted and downward biases in cohort studies are corrected. Strategies are needed to reduce population exposure to alcohol, the most efficient and cost-effective being strategies that influence the cost of purchasing the carcinogenic ingredient in alcoholic beverages: ethanol. Among pricing policies, those that target the cheapest alcohol appear to be the most efficient as the heaviest drinkers tend to select the cheapest alcohol. Cheap alcohol also has a high price elasticity. Other advantages of minimum pricing include greater public acceptability than across the board price increases, increased revenues for some
governments and support from some sectors of the alcohol industry. Canada is one of a handful of countries where minimum alcohol pricing in some form is evident in all of its 10 provinces. Evaluations of the public health impacts of Canadian minimum pricing indicate delayed effects on chronic alcohol-related diseases two to three years after a price increase. The size of the observed effects on alcohol-related morbidity and mortality tend to be much larger than has been predicted by highly conservative UK modelling studies. Given that minimum pricing is also associated with increased revenues in Canada, a case can be made that this strategies has the potential to be the most cost-effective of all available strategies for cancer prevention. Finally, recommendations are made for implementing minimum pricing so as to maximise public health benefits.

Cancer prevention at the population level: Policies to prevent and treat obesity

Today one in ten children starting school and one in four adults are obese. Excess weight is a major risk factor for diabetes and heart disease through its association with insulin resistance, raised blood pressure and high LDL cholesterol. Obesity is now also recognised as the leading modifiable cause of cancer for non-smokers, primarily through its metabolic effects on sex hormones, adipokines, growth factors and inflammatory pathways. The totality of the evidence from mechanistic studies, prospective cohort studies and following weight loss interventions supports a causal relationship between excess weight and cancers at multiple (though not all) sites. Identifying effective interventions to prevent and treat obesity will be crucial to reducing the incidence of new cancers as well as bringing wider public health benefits.

The causes of the recent rise in obesity are grounded in our modern lives and this can lead to a pessimistic view of the opportunities for change. However the success of public health policies in other areas demonstrates the potential for interventions at the population level to shape the environment to actively enable healthier lifestyles. In addition, there is now good evidence that specific action to support individuals to choose healthier diets and to become more physically active can encourage successful weight loss at a scale to bring population benefits.

This presentation will outline the current policy landscape, including new plans for a soft drink industry levy and reformulation to reduce the sugar content of foods. It will draw on the Government Foresight report on obesity to consider what else needs to be done to create a comprehensive strategy and accelerate progress to prevent and treat obesity.

Discussion

11.55 – 12.20
Hall 1A

Susan Jebb,
University of Oxford,
UK

12.20 – 12.30
Hall 1A

12.20 – 12.30
Hall 1A

Discussion
**Symposia abstracts (continued)**

### Stem cells and cancer

**11.00 – 11.05**

**Room 3B**

**Tariq Enver**, University College London Cancer Institute, UK

**Introduction**

This session will explore the intimate relationship between stem cells and cancer. In part this reflects shared molecular programmes and pathways controlling cell fate decisions, including self-renewal, but also the role of stem and progenitor cell as cellular targets for cancer initiation, maintenance, metastasis and relapse. As such, research into this area could highlight new therapeutic approaches.

**11.05 – 11.30**

**Room 3B**

**Sheila Singh**, McMaster University, Canada

**Therapeutic targeting of childhood medulloblastoma: Strategies for blocking recurrence**

Current clinical trials for recurrent medulloblastoma (MB) patients who no longer respond to risk-adapted therapy are based on genomic profiles of primary, treatment-naïve tumours. These approaches will provide limited clinical benefit for patients since recurrent metastatic MBs are highly genetically divergent from their primary tumour. Treatment for MB patients who present with recurrent metastatic lesions is limited to palliative care, and the development of novel therapeutics for these patients is encumbered by rare clinical opportunities in which specimens may be obtained from relapsed patients. Consequently, we have adapted the existing COG (Children’s Oncology Group) Protocol ACNS0332, for children with newly diagnosed high-risk MB, for treatment of NOD-SCID mice engrafted with human MB xenografts.

Our novel patient-derived xenograft models capture clonal evolution of MB cell populations in response to therapy, allowing for comprehensive, serial profiling of tumour clones by regular time point sampling of tumours throughout the course of our mouse-adapted therapy. Our experimental approach defines several tractable targets, including the epigenetic regulator Bmi1 and strategic Wnt activation, which mitigate recurrent MB. As future clinical oncology trials will most likely begin with relapsed patients, therapeutic targets from comparative analyses in primary and matched-recurrent tumours offer the greatest clinical yield and may be readily translated to the patient bedside.

**11.30 – 11.55**

**Room 3B**

**Dominique Bonnet**, The Francis Crick Institute, London, UK

**Leukemic stem cell interactions with the microenvironment: Friend or foe?**

Acute myeloid leukemia (AML) is a hematologic malignancy, arising within the bone marrow, which is characterised by the uncontrolled proliferation of leukemic blasts, often in association with a disruption of normal hematopoiesis. Like their normal counterparts, AML cells depend upon both cell-intrinsic and -extrinsic regulatory signals generated by their surrounding microenvironment, for their survival and proliferation. AML has long been considered a hematopoietic-cell autonomous disorder in which disease initiation and progression is driven by hematopoietic cell intrinsic genetic events. Recent experimental findings in diverse model systems have challenged this view, implicating different stromal cells of the bone marrow in disease pathogenesis. Thus it is now accepted that leukemic hematopoiesis can turn the BM niche into a “leukemic niche” which promotes leukemic stem cell (LSC) function and impairs the maintenance of normal HSC.

However, much remains to be understood about how different leukemic cells impacts the BM microenvironment and, in turn, how changes in the activity of specific BM niche cells contribute to AML pathogenesis. This talk will highlight some of the current understanding of the alterations of BM niche components and how the dialogue between leukemic and stromal cells participated in leukemogenesis.
**Integrating stem and progenitor cell contribution to intestinal self renewal**

At the time of printing, this abstract had not been not received. Check the Conference App for further details.

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**Discussion**

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**Noncoding RNAs in cancer**

**Introduction**

While only 1.8% of the genome directly encodes amino acids, around 70% is transcribed. Increasing evidence has revealed these transcripts to play a diversity of roles within the cell, and since noncoding loci are as susceptible to mutations as protein coding genes, their potential relevance in cancer is the subject of intense interest. This session will consider both the mechanistic and translational impact of noncoding RNAs on how we think about cancer.

**Long non-coding RNAs – Messages from the dark matter of the cancer genome**

The majority of the human genome is transcribed into non-protein-coding RNA. Hence, RNA is also the primary product of the cancer genome. We have defined the ncRNA expression landscape of lung, breast and liver cancer providing a comprehensive expression map of over 17000 long ncRNAs and discovering new IncRNAs associated with cancer whose molecular and cellular functions we are currently elucidating exploiting our custom siRNA library targeting 638 tumour-associated IncRNAs. The nuclear IncRNA MALAT1 was one of the first IncRNAs associated with cancer: it is associated with metastasis development in lung cancer. However, its high abundance and nuclear localisation have hampered its functional analysis. To uncover its functional importance, we developed a MALAT1 knockout model in human lung tumour cells by genomically integrating RNA destabilising elements site-specifically into the MALAT1 locus. This approach yielded a 1000-fold silencing of MALAT1 providing a unique loss-of-function model. Proposed mechanisms of MALAT1 action include regulation of splicing or gene expression. In lung cancer, MALAT1 does not alter alternative splicing but regulates gene expression inducing a signature of metastasis-associated genes. Consequently, MALAT1-deficient cells are impaired in migration and form fewer tumour nodules in a mouse xenograft model.

Encouraged by this discovery of the essential function of MALAT1 in lung cancer metastasis, we analysed whether MALAT1 could serve as therapeutic target for an Antisense oligonucleotide (ASO). Notably, MALAT1-ASO treatment prevented metastasis formation after tumour implantation. Thus, targeting MALAT1 with ASOs provided a therapeutic approach to prevent lung cancer metastasis with MALAT1 serving as both, predictive marker and therapeutic target. In summary, ten years after the discovery of the IncRNA MALAT1 as a biomarker for lung cancer metastasis, our loss-of-function model unraveled the active function of MALAT1 as a regulator of gene expression governing hallmarks of lung cancer metastasis.
Control of cell division by long non-coding RNAs

Long noncoding RNAs (IncRNAs) regulate many biological processes and have been implicated in development and disease pathogenesis. However of 16,000 IncRNAs identified in humans only ~100 have been functionally characterised. To address whether any of these IncRNAs play a role in cell division, we performed an RNAi screen targeting 2231 human IncRNAs. High-throughput microscopy coupled with automated image analysis uncovered several IncRNAs that are required for normal cell division. We validated and functionally characterised a number of IncRNAs. Depletion of these IncRNAs by RNAi, genome editing and antisense oligonucleotides revealed mitotic defects ranging from aberrant spindle formation, chromosome misalignment, mitotic delay and unfaithful chromosome segregation. Functional genomics combined with proteomics and cell biology will elucidate the molecular mechanisms of how these IncRNAs serve to preserve genome integrity.

Long non-coding RNAs and the regulation of the immune response

The first draft of the human genome uncovered a number of surprises including the observation that exonic regions of protein coding genes represented < 2% of the genome. Whilst some of the remaining DNA plays a crucial role in the maintenance of DNA structure and regulation of mRNA expression (i.e. transcription binding sites, promoter and enhancer regions), subsequent studies have shown that a significant proportion is transcribed into non-coding RNAs (ncRNAs). ncRNAs are broadly classified as either short ncRNAs (< 200 nucleotides) or long ncRNAs (> 200 nucleotides). The microRNA (miRNA) family of short ncRNAs are the best characterised and known to regulate gene expression via RNA interference pathway. In contrast, much less is known about the function and mechanism of action of IncRNAs despite the fact that > 27,000 have now been annotated in human. Cancer is commonly associated with an aberrant immune response include chronic inflammation and immunological tolerance. This presentation will provide an overview of our current understanding of the biology of IncRNAs and describe their role in the regulation of the immune response and inflammation.

Discussion

11.30 – 11.55
Hall 1B

Duncan Odom,
Cancer Research UK
Cambridge Institute,
UK

11.55 – 12.20
Hall 1B

Mark Lindsay,
University of Bath,
UK

12.20 – 12.30
Hall 1B
BASO~ACS and BAHNO symposium

Hosted by Garth Cruickshank, BASO~ACS and Mike Fardy, BAHNO

**11.00 – 11.30**
Room 11

**Margaret Stanley**
OBE, University of Cambridge, UK

**HPV vaccines – how to get the maximum protection**
Viral infections cause at least 15% of all human cancers; one of the most important oncogenic viruses is the human papillomavirus (HPV) a causal agent in 5% of all malignancies. About 13 HPV types are high risk or cancer causing HPVs with HPV 16 and 18 being the most important. Infection with one of these oncogenic HPVs is the cause of carcinoma of the cervix in women, the 3rd commonest cancer in women worldwide. HPV associated cancers are not confined to the cervix and HPV infection is implicated in the development of a proportion of vaginal, vulval, anal penile and head and neck cancers in both men and women. Importantly the incidence of HPV related cancers in these sites, particularly anal carcinomas and tonsillar carcinomas is increasing particularly in men. Prophylactic HPV vaccines are now included in the national immunisation programmes in many countries with young adolescent peripubertal girls the usual cohort for immunisation. Population effectiveness in women is now being demonstrated in those countries with high vaccine coverage. Since HPV associated cancers in men are increasing in incidence an issue of contemporary debate is extending HPV vaccination to adolescent boys.

**11.30 – 12.00**
Room 11

**Terry Jones**, NIHR Clinical Research Network: North West Coast, UK

**The management of oropharynx cancer in the HPV era**
At the time of printing, this abstract had not been not received. Check the Conference App for further details.

**12.00 – 12.30**
Room 11

**Mark McGurk**, King’s College London and Guy’s and St Thomas’ Foundation Trust, UK

**Sentinal lymph node biopsy in head and neck cancer: Good ideas need proper trials**
At the time of printing, this abstract had not been not received. Check the Conference App for further details.
## Parallel sessions

### Functional imaging for influencing management: Paradigm shift or pipe dream?

**Hosted by Nandita deSouza, The Institute of Cancer Research, London, UK**

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<td>14:05 – 14:30</td>
<td>Exploiting functional image information for decision making in clinical practice</td>
<td><strong>Christina Messiou</strong>, The Royal Marsden Hospital, London, UK</td>
<td>Room 4</td>
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<tr>
<td>14:30 – 14:55</td>
<td>Molecular imaging for targeted therapy</td>
<td><strong>Arturo Chiti</strong>, Humanitas University, Italy</td>
<td>Room 4</td>
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<tr>
<td>14:55 – 15:20</td>
<td>Image quantification: Do numbers help treatment stratification?</td>
<td><strong>Alan Jackson</strong>, Wolfson Molecular Imaging Centre, University of Manchester, UK</td>
<td>Room 4</td>
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<td>15:20 – 15:35</td>
<td>Proffered paper: F-18 labelled 3’-deoxy-3’-fluorothymidine (FLT) positron emission tomography (PET) imaging in patients with advanced pancreatic ductal adenocarcinoma: proof-of-concept reproducibility sub-analysis</td>
<td><strong>Angela Lamarca</strong>, The Christie NHS Foundation Trust, Manchester, UK</td>
<td>Room 4</td>
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<td>15:35 – 16:00</td>
<td>Proffered paper: The PROMIS study: Diagnostic accuracy of MRI and TRUS biopsy in prostate cancer</td>
<td><strong>Louise Brown</strong>, MRC Clinical Trials Unit, University College London, UK</td>
<td>Room 4</td>
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### Discussion

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### Early detection and prevention

**Hosted by Sir Harpal Kumar, Cancer Research UK, London, UK**

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<td>Introduction by the host</td>
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<td>14:10 – 14:40</td>
<td>Plasma DNA for early cancer screening</td>
<td><strong>Yuk Ming Dennis Lo</strong>, Chinese University of Hong Kong</td>
<td>Hall 1C</td>
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<td>14:40 – 15:10</td>
<td>Prostate cancer screening as national programme: Benefits at less harm and less cost</td>
<td><strong>Harry de Koning</strong>, Erasmus Medical Center, The Netherlands</td>
<td>Hall 1C</td>
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<td>15:10 – 15:40</td>
<td>Precision early diagnosis for oesophageal cancer</td>
<td><strong>Rebecca Fitzgerald</strong>, MRC Cancer Cell Unit, University of Cambridge, UK</td>
<td>Hall 1C</td>
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<tr>
<td>15:40 – 16:00</td>
<td>Discussion</td>
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The dark side of the genome – structural catastrophes in paediatric cancers

Hosted by Richard Gilbertson, CRUK Major Cancer Centre, University of Cambridge, UK

14.00 – 14.05
Introduction by the host

14.05 – 14.35
Room 3A
Genome-epigenome interplay in medulloblastoma
Paul Northcott, St Jude Children’s Research Hospital, Memphis, USA

14.35 – 15.05
Room 3A
In search for the lacking driver genes of neuroblastoma
Rogier Versteeg, Netherlands Bioinformatics Centre, The Netherlands

15.05 – 15.35
Room 3A
Translocations and catastrophe – chromothriptic disruption of transcription factors in ependymoma
Richard Gilbertson, CRUK Major Cancer Centre, University of Cambridge, UK

15.35 – 15.50
Room 3A
Children’s Cancer and Leukaemia Group (CCLG) McElwain Award winner
Proffered paper: Inhibiting HOX-PBX binding in paediatric glioblastoma as a novel therapeutic treatment
William Rogers, University of Surrey, UK

15.50 – 16.00
Room 3A
Discussion

Liquid biopsy

Hosted by Nicholas Turner, The Institute of Cancer Research, London, UK

14.00 – 14.05
Hall 1B
Introduction by the host

14.05 – 14.30
Hall 1B
Deep sequencing of circulating tumour DNA for personalised cancer detection and monitoring
Maximilian Diehn, Stanford University School of Medicine, USA

14.30 – 14.55
Hall 1B
The versatility of CTCs as a liquid biopsy: Biomarkers, biology and drug development
Caroline Dive, Cancer Research UK Manchester Institute, UK

14.55 – 15.20
Hall 1B
Using circulating tumour DNA to improve the management of metastatic castration-resistant prostate cancer
Gerhardt Attard, The Institute of Cancer Research, London, UK

15.20 – 15.35
Hall 1B
Proffered paper: Molecular profiling of circulating tumour cells (CTCs) in non-small cell lung cancer within the TRACERx study of intratumoural heterogeneity and evolution
Christopher Smith, University of Cambridge, UK
Parallel sessions (continued)

15.35 – 15.50
Hall 1B
Proffered paper: Molecular profiling of circulating tumour cells (CTCs) in non-small cell lung cancer within the TRACERx study of intratumoural heterogeneity and evolution
Sakshi Gulati, Cancer Research UK Manchester Institute, UK

15.50 – 16.00
Hall 1B
Discussion

Models of aneuploidy and centrosomal imbalance
Hosted by Iain Hagan, Cancer Research UK Manchester Institute, UK

14.00 – 14.10
Room 12
Introduction by the host

14.10 – 14.40
Room 12
Loss of primary cilia and tissue hyperplasia associated with centrosome amplification
David Glover, University of Cambridge, UK

14.40 – 15.10
Room 12
The contribution of genomic instability to malignant growth in Drosophila
Cayetano Gonzalez, Institute for Research in Biomedicine and The Barcelona Institute of Science and Technology, Spain

15.10 – 15.40
Room 12
Aneuploidy as the large adaptive mutation: Mechanism and theory
Rong Li, Johns Hopkins University School of Medicine, USA

15.40 – 16.00
Room 12
Discussion

Using viruses to treat cancer
Hosted by Alan Melcher, The Institute of Cancer Research, London, UK

14.00 – 14.05
Hall 1A
Introduction by the host

14.05 – 14.30
Hall 1A
Rhabdovirus based oncolytic virus vaccines
John Bell, Ottawa Hospital Research Institute, Canada

14.30 – 14.55
Hall 1A
Systemic delivery of Enadenotucirev, a group B oncolytic adenovirus for the treatment of cancer
Kerry Fisher, University of Oxford, UK

14.55 – 15.20
Hall 1A
How should we combine oncolytic virotherapy with other agents?
Kevin Harrington, The Institute of Cancer Research, London, UK

15.20 – 15.35
Hall 1A
Proffered paper: PARP trapping enhances oncolytic Reovirus cell killing in melanoma
Joan Kyula-Currie, The Institute of Cancer Research, London, UK
### Proffered paper: HPV drives tumour development throughout the head and neck: Improved prognosis is associated with an immune response largely restricted to the oropharynx

Tim Fenton, University College London, UK

| Time   | Location | Title                                      | Speaker                        | Institution                                |
|--------|----------|--------------------------------------------|--------------------------------|
| 15.35  | Hall 1A  | Proffered paper: HPV drives tumour development throughout the head and neck: Improved prognosis is associated with an immune response largely restricted to the oropharynx | Tim Fenton                    | University College London, UK |

| Time   | Location | Title                                      | Speaker                        | Institution                                |
|--------|----------|--------------------------------------------|--------------------------------|
| 15.50  | Hall 1A  | Discussion                                 |                                |                                          |

### BASO~ACS robotic surgery session

Hosted by William Cross, St James's University Hospital, Leeds, UK

| Time    | Location | Title                                      | Speaker                        | Institution                                |
|---------|----------|--------------------------------------------|--------------------------------|
| 14.00   | Room 11  | A robotic gynaecological perspective       | Simon Butler-Manuel            | The Royal Surrey County Hospital, UK        |
| 14.20   | Room 11  | A robotic thoracic perspective             | Sasha Stamenkovic              | Newcastle Cancer Centre, UK                  |
| 14.40   | Room 11  | A robotic urological perspective           | Ben Challacombe                | Guy's and St Thomas' NHS Foundation Trust, London, UK |
| 15.00   | Room 11  | A robotic colorectal perspective           | Mark Gudgeon                   | Frimley Park Hospital, UK                   |
| 15.20   | Room 11  | A robotic head and neck perspective        | Vinidh Paleri                  | Freeman Hospital, Newcastle, UK             |
| 15.40   | Room 11  | Discussion                                 |                                |                                          |

We would like to thank Intuitive Surgical for their support in supplying the da Vinci Xi for this session.
Parallel session abstracts

**Functional imaging for influencing management: Paradigm shift or pipe dream?**

**14.00 – 14.05**  
Room 4  
Nandita deSouza,  
The Institute of Cancer Research, London, UK

**Introduction**  
This session will appeal to oncologists, surgeons, imagers, nurses and patients and will include review of the role and utility of newer functional and quantitative imaging techniques in determining optimal management strategies for patients.

Presentations will discuss how to select functional imaging techniques (diffusion-weighted MRI, vascular and hypoxia imaging, metabolic, receptor and cell-signature imaging) when planning management, the evidence base for their use and how to use them to best advantage in the patient pathway. Attendees will also learn about new ways of imaging and image analysis that could be applied in clinical trials.

**14.05 – 14.30**  
Room 4  
Christina Messiou,  
The Royal Marsden Hospital, London, UK

**Exploiting functional image information for decision making in clinical practice**  
Functional MRI is integrated into routine imaging protocols for a number of tumour types. Diffusion weighted MRI is a functional MRI sequence which is common across most protocols. Assessments have traditionally focused on detailed local staging but advances in hardware and software have allowed whole body coverage and whole body diffusion weighted MRI in reasonable time frames. This non-invasive, well tolerated and sensitive whole body imaging technique is providing novel insight particularly into myeloma and metastatic disease. This talk will explore the current status of whole body diffusion weighted MRI and will introduce the impact on clinical decision making in oncology.

**14.30 – 14.55**  
Room 4  
Arturo Chiti,  
Humanitas University, Italy

**Molecular imaging for targeted therapy**  
Molecular imaging has the potentials to become the best imaging modality to predict survival and assess therapy response. The main challenges are related to the identification and standardisation of the best imaging biomarkers to be used in the different clinical and research settings.

**14.55 – 15.20**  
Room 4  
Alan Jackson, Wolfson Molecular Imaging Centre, University of Manchester, UK

**Image quantification: Do numbers help treatment stratification?**  
Advanced imaging modalities, particularly magnetic resonance imaging provide an increasing number of new tumour biomarkers which can be obtained noninvasively and repeatedly. We will review the evidence that such biomarkers are making an impact in clinic or are likely to do in the near future.
Proffered paper: F-18 labelled 3’-deoxy-3’-fluorothymidine (FLT) positron emission tomography (PET) imaging in patients with advanced pancreatic ductal adenocarcinoma: proof-of-concept reproducibility sub-analysis

**Background**
The thymidine analogue FLT is transported and phosphorylated in proliferating cells. FLT tumour uptake correlates with proliferation.

**Method**
Advanced pancreatic ductal adenocarcinoma (PDAC) patients with target lesion >2cm due to start chemotherapy were eligible. Dynamic FLT PET/CT scanning was performed over 60min, before starting chemotherapy (baseline scan, BS). Intra-patient reproducibility was explored by a second FLT scan within 7 days of BS and before chemotherapy (reproducibility scan, RPS). Lesions were manually delineated by two independent radiologists for determination of inter-radiologist concordance. FLT uptake in the primary tumour and metastases was quantified as standardised uptake value (SUV, mean and max) over 45-60min.

**Results**
Of the 21 patients consented 18 were scanned, all with primary tumour in situ and 83% with distant metastases (60% in liver). Thirty-five FLT scans were acquired for the whole study, 21 scans were analysed for this reproducibility sub-analysis (17 BS, 4 RPS) and 27 lesions delineated. At baseline, median SUVmean and SUVmax were 1.9 (95%CI 1.8-2.1) and 5.9 (95%CI 4.6-7.8), respectively, for the primary (n=17) and 4.6 (95%CI 3.7-5.2) and 8.9 (95%CI 8.2-10.5), respectively, for metastatic lesions (n=10; 9 liver, 1 lymph node). Intra-patient reproducibility between BS and RPS was good (all lesions; n=8): mean change and standard deviation (SD) of test-retest differences and Lin’s concordance coefficient (LCC) for SUVmean (mean change -5.4%; SD 9.8%; LCC 0.947, p<0.001) were superior to SUVmax (mean change 7.9%; SD 19.2%; LCC 0.642, p<0.001). The reproducibility achieved by the second radiologist was similar (6 lesions): SUVmean (mean change -10.3%, SD 12.8%; LCC 0.534, p=0.005) and SUVmax (mean change 4.7%, SD 14.5%; LCC 0.826, p<0.001). Inter-radiologist concordance was assessed by comparing 12 lesions (8 scans; 4 BS, 4 RPS): LCC for SUVmean and SUVmax were 0.635 (p<0.001) and 0.489 (p=0.019), respectively.

**Conclusion**
FLT-PET is feasible and reproducible (intra-patient and inter-radiologist) in patients with advanced PDAC.

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Proffered paper: The PROMIS study: Diagnostic accuracy of MRI and TRUS biopsy in prostate cancer

**Background**
Multi-parametric magnetic resonance imaging (MP-MRI) used as a triage test might allow men to avoid unnecessary transrectal ultrasound-guided prostate biopsy (TRUS-biopsy).

**Method**
The PROMIS Study (ClinicalTrials.gov/NCT01292291) was a multicenter, paired-cohort, confirmatory study testing MP-MRI and TRUS-biopsy against an accurate reference test – template prostate mapping biopsy (TPM-biopsy). Men with an elevated PSA up to 15ng/ml with no prior biopsy underwent MP-MRI followed by TRUS-biopsy and TPM-biopsy. The
conduct and reporting of each test was performed blind to other test results. Clinically significant cancer was defined as Gleason \( \geq 4+3 \) and/or a maximum cancer core length \( \geq 6\text{mm} \).

**Results**

Between May 2012 and December 2015, 576 men underwent all 3 tests. On TPM-biopsy 408/576 (71%) had cancer with 230/576 (40%) being clinically significant. For MP-MRI, the sensitivity for clinically significant cancer was 93% [95% CI 88-96], specificity 41% [36-46], positive predictive value 51% [46-56] and negative predictive value 89% [83-94]. TRUS-biopsy was significantly less sensitive than MP-MRI (48% [42-55], \( p < 0.0001 \)). Two alternative scenarios were compared against the current practice standard of TRUS-biopsy. First, using MP-MRI as a triage test would avoid primary biopsy in 158/576 (27%), but miss 14/576 (2%) cases of clinically significant cancer. Second, if targeted biopsy based on the MP-MRI were to match the accuracy of TPM-biopsy, triage with MP-MRI would avoid biopsy in 158/576 (27%) but detect an additional 17/576 (3%) clinically significant cancers.

**Conclusion**

MP-MRI, as a triage test prior to first biopsy, can identify a quarter of men who might safely avoid an unnecessary biopsy and correctly identify over 90% of men with clinically significant cancer.

**Discussion**

Early detection and prevention

**Introduction**

Sir Harpal Kumar,
Cancer Research UK,
London, UK

**Plasma DNA for early cancer screening**

Our group has been interested in the diagnostic applications of plasma DNA since the late 1990s. We started with the development of non-invasive prenatal testing (NIPT) which has now been introduced into over 90 countries and has been used by millions of pregnant women. The success of NIPT has triggered recent intense interest about the use of a similar strategy for cancer detection. Our group has particular interest in the early screening of nasopharyngeal carcinoma (NPC). In this regard, we have previously developed a method based on the measurement of plasma Epstein-Barr virus (EBV) DNA. In 2013, we started a 20,000-person study using plasma EBV DNA to screen for NPC. We expect that this study will be completed within 2016. Our next goal is to attempt to generalise plasma DNA-based cancer screening to other cancer types through the use of genomewide sequencing of plasma DNA.
Prostate cancer screening as national programme: Benefits at less harm and less cost

The European Randomised Study of Screening for Prostate Cancer (ERSPC) showed that Prostate-Specific Antigen (PSA) based screening results in a 27% prostate cancer mortality reduction. Although there are especially concerns on the harms of overdiagnosis and overtreatment, it has been shown that the benefits can outweigh the harms if screening is stopped early to prevent overdiagnosis. The cost-effectiveness of a screening program limited to men aged 55-59, including active surveillance for low-risk men, is comparable to cervical cancer screening in many countries in Europe. In future, further improvements are expected in the use of active surveillance and in discrimination between indolent and significant disease due to new biomarkers and magnetic resonance imaging. However, these future developments are no reason to postpone the implementation of high quality PSA screening and reduce opportunistic PSA testing at old ages. In a next step, the ways to implement a high-quality program should be evaluated.

Precision early diagnosis for oesophageal cancer

Cancer of the oesophagus is a global problem with high mortality due to late diagnosis. The accessibility of the oesophagus makes it possible to collect cells non-endoscopically using the Cytosponge and apply biomarkers on the retrieved material to detect pre-cancerous lesions. The progress in using a variety of genetic and protein biomarkers to diagnose metaplasia and assess the degree of dysplasia in Barrett's and squamous cell dysplasia will be discussed. In addition the path to clinical application for Cytosponge-biomarker testing will be discussed.

Discussion

The dark side of the genome – structural catastrophes in paediatric cancers

Introduction

Next generating sequencing has revealed novel driver mutations of human cancers. Adult malignancies frequently develop with single nucleotide and/or focal copy number variations that activate oncogenes or inactivate tumour suppressor genes. In stark contrast, such genetic events are relatively rare in paediatric cancers. Rather, childhood cancers, and in particular solid tumours, are characterised by large chromosomal copy number changes and gross structural alterations. The apparent rarity of genetic alterations that target specific genes in paediatric cancers has hindered efforts to understand the aberrant biology that underpin these malignancies, and restricted efforts to develop new treatment approaches. But recent studies of paediatric central and peripheral neural tumours have cast light on how structural changes at the chromosomal level can lead to aberrant activation of specific genes and cell pathways. In this session, Dr Paul Northcott (St. Jude Children’s Research Hospital, USA) will discuss how structural chromosomal alterations in medulloblastoma – the most common malignant brain tumour of childhood – lead to enhancer hijacking that activates oncogenic signalling. Professor Rogier Versteeg (Netherlands Bioinformatics Centre) will discuss how structural rearrangements including chromothripsis target and activate TERT expression in high-risk neuroblastoma – the most common extracranial nervous system tumour in children. Finally, Professor Richard Gilbertson (University of Cambridge, England) will discuss how chromothripsis activates aberrant NFκB signalling in ependymoma through a novel fusion protein encoded by the C11orf95-RELA translocation.
Parallel session abstracts (continued)

Genome-epigenome interplay in medulloblastoma

The recent application of genomics to large series of primary human medulloblastomas has dramatically improved our perspective of the genes, pathways, and molecular processes underpinning this highly malignant paediatric brain tumour. Transcriptionally defined molecular subgroups have emerged, changing the fundamental definition of medulloblastoma from a single histologically recognised entity, to four distinct subtypes: WNT, SHH, Group3, and Group4. These subgroups exhibit highly discriminatory genomic landscapes, patient demographics, and clinical phenotypes. Next-generation sequencing has identified and implicated recurrently altered somatic drivers, many of which are affected in a subgroup-restricted manner. In contrast, somatic variants affecting chromatin-modifying genes, including histone ‘writers’, ‘erasers’, and ‘readers’ are prevalent in medulloblastoma and occur across subgroups, suggesting that deregulation of the epigenome is an essential step during medulloblastoma development. Integrative ‘omics’-based strategies have begun to dissect the fascinating genome-epigenome interplay underlying medulloblastoma, revealing previously undisclosed oncogenic mechanisms and drivers of disease. Furthermore, new insights into the biology and putative origins of the poorly understood Group3 and Group4 medulloblastomas have likewise been revealed. This talk will summarise these recent discoveries afforded through genomics and discuss their potential implications, both in the lab and in the clinic, going forward.

In search for the lacking driver genes of neuroblastoma

Like many other childhood tumours, neuroblastoma have a low mutation load and only few recurrently mutated oncogenes and tumour suppressor genes. In contrast, gains and losses of chromosome arms are frequent and show consistent patterns, suggesting a prominent role in oncogenesis. The gained and lost regions are large and include hundreds of genes. We have therefore postulated that gains and losses of many genes in such regions together cause an imbalance in expression that activates cancer driving pathways. However, such a hypothesis is hard to test. We follow two approaches to elucidate the role of genomic gains and losses. Firstly, we pinpoint regions of imbalances by whole genome sequencing of large neuroblastoma series. Secondly, we identify key genes with a potential role in lineage differentiation and oncogenesis in neuroblastoma. We observed that neuroblastoma tumours include two types of tumour cells. They share the same genetic defects, but have highly divergent phenotypes. The two tumour cell types seem to correspond to two stages of normal differentiation of the adrenergic lineage. We identified the master regulators of this differentiation program. Combining these functional studies with the genomic imbalances prioritises candidate genes that may represent the lacking driver genes of neuroblastoma.

Translocations and catastrophe – chromothriptic disruption of transcription factors in ependymoma.

Whole genome sequencing of ependymoma – the third most common brain tumour of childhood – has revealed structural and copy number changes at the chromosomal level, but very few focal alterations of specific genes. These non-random chromosome copy number alterations are large, making it difficult to pinpoint which genes are driving the cancer. This talk describes two approaches to unmask specific genes that drive ependymoma. First, we used a cross-species in vivo screen of 84 candidate oncogenes and 39 candidate tumour suppressor genes, located within recurrent chromosomal alterations to validate eight new ependymoma oncogenes and ten new ependymoma tumour suppressor genes. Second, we show that chromothripsy underpins a series of highly recurrent oncogenic fusions between...
RELA, the principal effector of canonical NFκB signalling, and an uncharacterised gene, C11orf95 in more than two-thirds of supratentorial ependymomas. Together, these data reveal the importance of large chromosomal alterations in driving paediatric ependymoma; mechanisms to identify the genes that are targeted by these events; and thereby potential new therapeutic approaches.

**Children’s Cancer and Leukaemia Group (CCLG) McElwain Award winner**

**Proffered paper: Inhibiting HOX-PBX binding in paediatric glioblastoma as a novel therapeutic treatment**

**Background**
The HOX genes encode a family of transcription factors that play an essential role in embryonic patterning, but are dysregulated in numerous cancers, including glioblastoma. Previous research indicates that all HOX genes are expressed at varying levels in glioblastoma cells, but are highly expressed in glioma stem cells. Previously we reported that disrupting HOX-PBX binding using the HXR9 peptide induced adult glioblastoma cell lines to undergo apoptosis, and that glioma stem cells were the most sensitive to HXR9-mediated death. In this study we assessed whether HXR9 could also kill paediatric glioblastoma cells.

**Method**
HOX and PBX expression in glioma cell lines and health brain tissue was determined using quantitative RT-PCR and IHC staining. Glioma cells were then treated with HXR9, and cell viability was determined by MTS assay. Mode of death was determined via RT-PCR and western blots for AP-1 and Bcl-2 family members.

**Results**
Glioma cell lines showed elevated HOX and PBX expression compared to normal healthy normal paediatric brain tissue. HXR9 showed dose dependent cytotoxicity in all cell lines, with IC50 results below 80uM for all cell lines. Mode of death was determined to be apoptosis mediated by C-Fos up regulation and Bcl-XL down regulation.

**Conclusion**
Initial results show that HOX and PBX genes are aberrantly expressed in paediatric glioma cells, and up regulated compared to healthy normal brain. HXR9 is cytotoxic against glioma cells, which is mediated by an up regulation of C-Fos, and subsequent down regulation of Bcl-XL. Another AP-1 member, C-Jun, was shown to be uneffected as was Bcl-2. Mode of death was determined to be apoptosis.
### Introduction

Liquid biopsies have the potential to transform the treatment of cancer, through more precise monitoring of treatment efficacy and assessment of dynamic changes in tumour as a result of prior therapy. This session will discuss advances in technology for circulating tumour DNA and circulating tumour cell analysis, and how these technologies are being integrated into clinical trials.

### Deep sequencing of circulating tumour DNA for personalised cancer detection and monitoring

At the time of printing, this abstract had not been not received. Check the Conference App for further details.

### The versatility of CTCs as a liquid biopsy: Biomarkers, biology and drug development

The analysis of ctDNA (ctDNA) to stratify and monitor cancer patients is the ‘here and now’, liquid biopsy biomarker, testified by the recent upsurge in the number of biotech companies offering this as a service. However, whilst circulating tumour cells (CTCs) are more technically challenging, they offer more versatility not only as patient management tools but also to explore the biology of metastatic cancers, to identify new drug targets and to support drug development. The increasing number of platforms available to study CTCs and their advantages and disadvantages will be reviewed. I will then focus on the application of CTCs as biomarkers in lung cancer, describing their utility in small cell and non small cell lung cancer trials. The challenges of CTC heterogeneity and how this impacts biomarker development will be discussed. I will overview our development of patient CTC derived explant models (CDX) and how these and cultures developed from them can be used to support drug development. Finally, I will describe how our CTC analysis led to the discovery and interrogation of vasculogenic mimicry in small cell lung cancer.

### Using circulating tumour DNA to improve the management of metastatic castration-resistant prostate cancer

At the time of printing, this abstract had not been not received. Check the Conference App for further details.

### Proffered paper: Comprehensive analysis of cell-free tumour DNA in plasma and urine tracks disease burden and reveals clonal evolution in muscle invasive bladder cancer

**Background**

Muscle Invasive Bladder Cancer (MIBC) is an aggressive disease with a significant risk of metastatic progression. Neo-adjuvant chemotherapy after radical cystectomy can improve overall survival but benefit is limited to ~50% of eligible patients. Presently there is no
accurate and prompt predictor of treatment response. Cell-free tumour DNA (cftDNA) present in plasma (PLS), urine-supernatant (USN) or -cell-pellet (UCP) may represent a non-invasive means to rapidly predict treatment response and track disease progression.

Method
We used a combination of Tagged Amplicon Sequencing (TAm-Seq) and shallow Whole Genome Sequencing (sWGS) to detect and track somatic point-mutations and copy-number abnormalities (SCNA) in 345 longitudinal DNA samples. These were extracted from matched initial endoscopic tumour resection (TUR), PLS, USN or UCP of 20 MIBC patients. Presence and levels of cftDNA were correlated with treatment response and disease course.

Results
TAm-Seq identified 37 mutations (including TP53, KRAS) in TUR, PLS, USN or UCP in 15/20 patients. 81% of these were detected in TUR whilst the remaining 19% were exclusive to peripheral fluids. In all patient TUR, including those where no point-mutations were identified, sWGS revealed genome wide and focal SCNAs, and these were represented to different degrees in matched PLS (27.3%), USN (58.9%) and UCP (58.9%). Indeed, private somatic events were observed in all fluid compartments, suggesting that all are valuable targets for testing. Whilst presence and levels of cftDNA at the first time-point did not significantly correlate with treatment response, longitudinal analysis revealed a general correlation between cftDNA dynamics and ultimate disease course, as well as evidence of clonal evolution.

Conclusion
We demonstrate combined urine and plasma cftDNA analysis as a means of tracking disease course in MIBC. Point-mutation and SCNA levels dynamically changed over time and revealed evidence of clonal evolution. Larger scale analyses are required to confirm predictive utility of cftDNA for treatment response.

Proffered paper: Molecular profiling of circulating tumour cells (CTCs) in non-small cell lung cancer within the TRACERx study of intratumoural heterogeneity and evolution

Background
Circulating tumour cell (CTCs) profiling is gaining momentum as multiple studies demonstrate the potential of 'liquid biopsy' to study tumour biology. The TRACERx study (TRAcking non-small cell lung Cancer Evolution through therapy [Rx], ClinicalTrials.gov number, NCT01888601) is a prospective study in Stage I-III non-small cell lung cancer (NSCLC). Within this study, we are profiling pulmonary CTCs to establish their utility for detection of putative driver mutations and copy number aberrations (CNA) and determine whether number, heterogeneity or genetic status of CTCs isolated from blood just before surgical resection can predict tumour recurrence and aid subsequent therapy selection.

Method
CTCs were enriched, enumerated, isolated, biobanked and analysed using our standard operating procedures. Next generation sequencing (NGS) used for both CNA and mutational profiling (whole exome sequencing WES) of single and pooled CTCs (and WBC controls). CTC genetic profiles are being compared to the genetic profiles of spatially separated resected tumour sections obtained at the same time as pulmonary blood sample used
for CTC enrichment. In addition, metastatic biopsies and peripheral blood samples are being collected from relapsing patients to evaluate which CTC clones were responsible for metastasis. Further, a pipeline to optimise variant calling from this dataset is under development.

**Conclusion**

We have established a robust workflow for enrichment, enumeration, isolation and molecular analysis of CTCs from early stage NSCLC patients in the TRACERx study which can be used to compare to matching tumour. Over 100 patient blood samples have been processed with CTCs isolated from 27 patients. NGS has been applied to >70 CTCs and WBC controls and initial CNA profiling and WES have been used to identify molecular changes shared by tumour and CTCs. On-going analysis will determine how the relationship between CTC and tumour genomic profiles shed light on NSCLC evolution, heterogeneity and recurrence.

**Discussion**

Models of aneuploidy and centrosomal imbalance

**Introduction**

Many tumours are aneuploid and most harbour more than the normal compliment of two centrosomes. Aneuploidy accelerates tumour evolution in the fight with the immune system and therapeutic intervention. Aneuploidy also induces a range of stresses that can be exploited in therapy. Experimentally increasing of centrosome numbers can be tumourigenic. The model system studies in this session address how chromosome/centrosome imbalances arise and the impact of these changes upon cell physiology. The session will be of particular interest to scientists and clinicians whose therapeutic strategies rely upon inhibitors of microtubules or chaperones (e.g. Hsp90) or centrosomal kinases (e.g. polo and Aurora A).

**Loss of primary cilia and tissue hyperplasia associated with centrosome amplification**

To address the relationship between supernumerary centrosomes and cancer, we generated a transgenic mouse that permits inducible expression of the centriole duplication regulator, Polo-like-kinase-4 (Plk4). Over-expression of Plk4 advances tumour formation onset in the absence of p53 and leads to hyperproliferation of cells in the skin. Mice overexpressing Plk4 develop grey hair due to a loss of melanocytes and bald skin associated with thickening epidermis. This reflects increased proliferating cells in the basal epidermis and their expansion cells into suprabasal layers. Expression of markers for hyperplasia is paralleled by a decreased expression of differentiation markers. The proliferating cells show increased centrosomes and a loss of primary cilia, events mirrored in primary cultures of keratinocytes. Pancreatic islets become enlarged following Plk4 over-expression and the α- and β-cells exhibit centrosome amplification. Multiple centrosomes and loss of primary cilia also disrupt organisation of both undifferentiated and differentiated pancreatic organoids overexpressing Plk4 and this is exacerbated by p53 loss. Thus in two different tissues, repeated centriole duplication prevents formation of basal bodies leading to loss of primary cilia, disruption of signalling and aberrant differentiation. The absence of p53 permits these cells to continue dividing setting up a neoplastic state of error prone mitoses.

**14.00 – 14.10**

Room 12

Iain Hagan, Cancer Research UK Manchester Institute, UK

**14.10 – 14.40**

Room 12

David Glover, University of Cambridge, UK
**The contribution of genomic instability to malignant growth in Drosophila**

This talk will summarise data derived from Drosophila tumour models on how unscheduled gene expression impinge on tumour initiation and malignant growth. Most of the malignant tumours that can be experimentally induced in flies are larval brain tumours caused by loss-of-function of any of several genes that control the asymmetric division of neural precursors. However, there is a different type of fly cancer model that derives from the neuroepithelium. In the latter, genes that are normally expressed in germline cells become aberrantly activated. In the human oncology literature, these genes are referred to as cancer-testis, or cancer-germline genes. Direct proof of the functional relevance of such genes in human malignancy is still lacking, but research on the fly models is unveiling unsuspected functions of these genes that may have direct therapeutic implications. Another key aspect of human malignant growth that Drosophila tumour models recapitulate well is genome instability. Through ongoing experiments on this front we are currently addressing the cause-effect relation between genome instability and malignant growth and how the former can be used to inhibit the latter.

**Aneuploidy as the large adaptive mutation: Mechanism and theory**

Aneuploidy, the state of having unequal number of chromosomes, is a hallmark of cancer and pathogenic eukaryotic microbes. As a result of the altered chromosome stoichiometry, the expression of hundreds to thousands of genes is altered in any given aneuploid genome compared to the euploid genome, producing drastic changes in cellular phenotypes under diverse growth conditions. Under stressful conditions, elevated mitotic error rates result in aneuploid cell populations with diverse chromosome stoichiometry. The karyotypic heterogeneity gives rise to phenotypic variability, the degree of which increases with overall fitness decline of the population. The combination of phenotypic variation and selection fuels the rapid emergence of stress-resistant variants. Evidence suggests that this chromosome-based adaptive mechanism is prevalent in cancer and eukaryotic microbes for their pathogenesis and drug resistance. Therapies against these diseases must therefore not only target the fitness of disease-causing cells but also effectively manage these cells’ evolutionary potential due to chromosome instability and aneuploidy.

**Discussion**

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Rhabdovirus based oncolytic virus vaccines
Oncolytic viruses have the capacity to induce anti-tumour immune responses by the direct lysis of cancer cells and liberation of tumour antigens. We have developed strategies to enhance this capacity by creating oncolytic vaccines that encode tumour antigens while retaining their oncolytic activity. I will discuss the development of our Maraba virus based technology and its use in combination therapy approaches.

Systemic delivery of Enadenotucirev, a group B oncolytic adenovirus for the treatment of cancer
Systemic delivery of oncolytic viruses has remained a substantial challenge for the field. Sequestration of virus particles by the reticular endothelial system together with neutralising antibodies and complement take a heavy toll on administered dose. A number of different strategies have been proposed or have been attempted in the clinics or pre-clinically including cell-based delivery, dose scheduling and vector modification. We have focused on using a group B oncolytic virus, Enadenotucirev (formerly called ColoAd1), that retains activity in human blood and can be produced in sufficient quantities to allow repeated administration of a ‘breakthrough dose’. A breakthrough dose is the amount of virus required to saturate neutralisation or unwanted interactions with blood components. Dose scheduling and optimisation of infusion rates can then be used to manipulate clearance by the innate immune system and maximise the number of viable virus particles in the blood stream in order to achieve tumour delivery. In clinical trials we are able to show that a data driven optimisation of virus kinetics can achieve convincing delivery to tumours and expression of virus late proteins. This data validates the utility of Enadenotucirev as an oncolytic product candidate and provides a strong platform for next generation agents expressing therapeutic biologicals.

How should we combine oncolytic virotherapy with other agents?
Talimogene laherparepvec (T-VEC) is a genetically-modified herpes simplex virus type 1-derived injectable oncolytic virus that is selectively replication-competent in tumour cells (especially those with MAPK pathway activation). It expresses human granulocyte-macrophage colony-stimulating factor as an immunostimulatory cytokine. T-VEC is well tolerated. In a phase 3 trial in 436 pts with unresected stage IIIIB-IV melanoma (the OPTIM trial2), intraliteral T-VEC improved durable response rate (continuous partial response [PR] or CR ≥6 months; primary endpoint) from 2% to 16% vs subcutaneous recombinant GM-CSF. T-VEC was approved by the US FDA and EMA in October 2015 and by NICE in 2016. Subsequent phase Ib studies of T-VEC and immuno-oncology (IO) agents (ipiimumab and pembrolizumab) have now completed recruitment. Each of these studies has demonstrated tolerable toxicity profiles (with no dose-limiting toxicities) and impressive anti-tumour efficacy. Detailed data from these studies will be discussed and plans for subsequent phase II evaluations of T-VEC plus pembrolizumab (in melanoma and head and neck cancer) will be presented.

Another oncolytic virotherapy (Coxsackievirus A21 (CVA-21), CAVATAKTM) is also under development and this has achieved impressive responses by intratumoural delivery (Phase II CALM study). In addition, CVA-21 has been administered by intravenous injection in the
Proffered paper: PARP trapping enhances oncolytic Reovirus cell killing in melanoma

Background
Oncolytic viruses have shown good potential in human clinical trials. Reovirus (RT3D), a naturally occurring human double stranded RNA oncolytic virus has shown preclinical efficacy in the treatment of a wide range of tumour types and reached phase III testing in clinical trials. Early clinical studies have shown that this agent has modest monotherapy efficacy and it has been used in combination regimens with cytotoxic chemotherapy. However, not all patients benefit from these treatments therefore highlighting a need to identify therapeutic opportunities for combining oncolytic viruses with other novel anti-cancer drugs.

Method
We carried out a screen containing a range of different targeted therapeutic agents including known cytotoxic drugs and some novel therapeutic agents with the aim of looking for potential "viral sensitizers" that could enhance RT3D tumour killing in the A375 BRAFV600E mutant melanoma cells. Cell Titer Glo was used as an endpoint assay to measure synergistic interaction between RT3D and the therapeutic agents.

Results
We identified talazoparib (BMN-673), a potent PARP trapping poison as a top hit from the screen in A375 cells and validated our findings further in a panel of melanoma cell lines. RT3D infection induced PARP1 activation and this was abrogated in the presence of talazoparib or olaparib, another PARP poison. The combination of RT3D and the PARP poisons correlated with increased levels of cleaved caspase 3, cleaved caspase 8 and PARP cleavage compared to either agent alone. In addition, we found PARP1 to be trapped in the chromatin following RT3D and talazoparib and olaparib. Finally, there was synthetic lethality in cells defective in the DNA binding domain of PARP1 compared to wild type PARP1.

Conclusion
Our pre-clinical data provide a strong rationale for the combination of RT3D with PARP1 trapping agents in melanoma and potentially other tumours.
Proffered paper: HPV drives tumour development throughout the head and neck: Improved prognosis is associated with an immune response largely restricted to the oropharynx

Background
In squamous carcinomas of the head and neck (HNSCC), the increasing incidence of oropharyngeal cancers (OPSCC) is attributable to HPV infection. Despite commonly presenting at late stage, HPV-driven OPSCC are associated with improved prognosis compared with HPV-negative disease. HPV DNA is also detectable in non-oropharyngeal (non-OPSCC), but its pathogenic role and clinical significance are unclear. The objectives of this study were to determine whether HPV plays a causal role in non-OPSCC and to investigate whether HPV confers a survival benefit in these tumours.

Method
Meta analysis was used to build a cross-tissue gene expression signature for HPV-driven cancer. Classifiers trained by machine-learning approaches were used to predict the HPV status of 520 HNSCCs profiled by The Cancer Genome Atlas project. 464 HNSCCs were similarly classified using DNA methylation data and these analyses were integrated with genomic, histopathology and survival data to permit a comprehensive comparison of HPV transcript-positive OPSCC and non-OPSCC.

Results
HPV-driven tumours accounted for 4.1% of non-OPSCC. Regardless of anatomic site, HPV+ HNSCCs shared highly similar gene expression and DNA methylation profiles; non-keratinizing, basaloid histopathological features and lack of TP53 or CDKN2A alterations. Improved overall survival however, was largely restricted to HPV-driven OPSCCs, which were associated with increased levels of tumour infiltrating lymphocytes compared with HPV-driven non-OPSCC.

Conclusion
Our analysis identifies a causal role for HPV in transcript-positive non-OPSCC throughout the head and neck. Notably however, HPV-driven non-OPSCCs display a distinct immune microenvironment and clinical behavior compared with HPV-driven OPSCCs.

Discussion

Introduction
The purpose of this symposium is to highlight the application of robotics within different surgical specialties. Key note speakers, drawn from colorectal, thoracics, ENT, urology, and gynaecology, will provide an overview of robotics within their specialty, outlining the benefits, limitations, and future developments. There will follow an interactive debate to facilitate cross fertilisation of ideas and opportunities for shared learning between the disciplines. This session, which brings together robotic experts from across surgery, will provide a unique opportunity for open discussion and dissemination of knowledge. There will also be an opportunity for participants to practice on the new Xi da Vinci system in a simulated environment.
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<th>Time</th>
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<tr>
<td><strong>14.00 – 14.20</strong></td>
<td>A robotic gynaecological perspective</td>
<td>Simon Butler-Manuel, The Royal Surrey County Hospital, UK</td>
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<td><strong>14.20 – 14.40</strong></td>
<td>A robotic thoracic perspective</td>
<td>Sasha Stamenkovic, Newcastle Cancer Centre, UK</td>
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<td>A robotic urological perspective</td>
<td>Ben Challacombe, Guy’s and St Thomas’ NHS Foundation Trust, London, UK</td>
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<td><strong>15.00 – 15.20</strong></td>
<td>A robotic colorectal perspective</td>
<td>Mark Gudgeon, Frimley Park Hospital, UK</td>
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<td><strong>15.20 – 15.40</strong></td>
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<td>Vinidh Paleri, Freeman Hospital, Newcastle, UK</td>
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*We would like to thank Intuitive Surgical for their support in supplying the da Vinci Xi for this session.*
Proffered paper session 1

Chaired by Johann de Bono, The Institute of Cancer Research, London, UK

14.00 – 14.15
Room 3B
Targeting flap endonuclease 1 (FEN1) for personalisation of ovarian cancer therapy
Reem Ali, University of Nottingham, UK

14.15 – 14.30
Room 3B
Metformin increases 18F-FDG flux and inhibits fatty acid oxidation at clinical doses in breast cancer: Results of a phase 0 clinical trial
Simon Lord, University of Oxford, UK

14.30 – 14.45
Room 3B
Understanding different types of cervical screening non-participant: A population-based survey
Jo Waller, University College London, UK

14.45 – 15.00
Room 3B
PET-PANC: Multi-centre trial of 18Fluorine-2-fluoro-2-deoxy-D-glucose positron emission tomography with computed tomography scanning in the diagnosis and management of pancreatic cancer
Paula Ghaneh, University of Liverpool, UK

15.00 – 15.15
Room 3B
Cancer stigma among ethnic minority women
Laura Marlow, University College London, UK

15.15 – 15.30
Room 3B
Updated results from a phase 3 trial of nivolumab combined with ipilimumab in treatment-naive patients with advanced melanoma (CheckMate 067)
John Wagstaff, South West Wales Cancer Institute, Swansea, UK

15.30 – 15.45
Room 3B
Cell fate imbalance in the oesophageal epithelium: Mutant cell competition
Maria Alcolea, Wellcome Trust-MRC Cambridge Stem Cell Institute, University of Cambridge, UK

15.45 – 16.00
Room 3B
The molecular epidemiology of rare and compound EGFR mutations in 14,304 non-small cell lung carcinomas
Matthew Evans, Queen Elizabeth Hospital Birmingham, UK
Targeting flap endonuclease 1 (FEN1) for personalisation of ovarian cancer therapy

Background
The clinical use of PARP inhibitor (olaparib) in BRCA deficient ovarian cancers suggests that development of alternative synthetic lethality strategies is highly desirable. The flap structure specific endonuclease-1 (FEN1) is critical for DNA long patch base excision repair (LP-BER). FEN1 is a key player in Okazaki fragment maturation during replication, rescue of stalled replication forks and maintenance of telomeres.

Method
We evaluated FEN1 mRNA expression in breast cancer (n=2329), FEN1 protein expression in 1462 breast cancers, 140 gastric cancers and 156 epithelial ovarian cancers. A FEN1 drug discovery program has been developed and a compound library of 391,275 compounds has been screened. We have isolated several novel FEN1 inhibitors. We have tested a prototypical FEN1 inhibitor in ovarian cancer models; A2780 (platinum sensitive), A2780cis (platinum resistant), PEO1 (BRCA2 germ-line deficient, platinum sensitive) and PEO4 (BRCA2 germ-line proficient, platinum resistant) cells.

Results
We whole exome sequenced and DNA repair expression profiled A2780, A2780cis, PEO1 and PEO4 cells to identify differential markers of platinum resistance. Interestingly, cisplatin treatment induced FEN1 expression in ovarian cancer cells. FEN1 knock down resulted in platinum sensitivity in A2780cis cells which was associated with DNA double strand break (DSB) accumulation, G2M cell cycle arrest and apoptosis. Importantly, FEN1 inhibitor treatment reversed platinum resistance in A2780cis cells and also leads to DSB accumulation, G2M cell cycle arrest and apoptosis. In BRCA2 deficient PEO1 cells, FEN1 inhibitor monotherapy induced synthetic lethality as evidence by DNA double strand break (DSB) accumulation, S-phase cell cycle arrest and apoptosis compared to BRCA2 proficient PEO4 cells. An in vivo study in tumour bearing mice is on-going.

Conclusion
FEN1 depletion results in platinum sensitization. FEN1 inhibitor treatment is a platinum sensitizor and also induces synthetic lethality in BRCA2 deficient ovarian cancer cells. We provide the first evidence that FEN1 targeting is a new strategy in ovarian cancers.

Metformin increases 18F-FDG flux and inhibits fatty acid oxidation at clinical doses in breast cancer: Results of a phase 0 clinical trial

Background
Over 100 clinical trials are underway worldwide to investigate the anticancer effects of the diabetes drug, metformin. However, it is still not determined as to whether metformin has significant direct effects on cancer cells or solely indirect effects via modulation of host metabolism.

Method
We recruited 41 non-diabetic patients with primary breast cancer to a neoadjuvant window trial. Patients received an escalating dose of metformin to 1500mg for 2 weeks with pre- and post-metformin pharmacodynamic assessments including, dynamic 18F-FDG PET-CT scans,
Proffered paper session abstracts (continued)

serum metabolic markers, and tumour biopsies for whole transcriptome RNASeq, tumour metabolomics and immunohistochemistry.

Results
Assessment of tumour FDG kinetics using a classic 2-tissue compartment model with three rate constants showed a 1.3 fold change (FC) post-metformin in the composite 18F-FDG flux constant, $K_{flux}$ ($p = 0.041$, paired t-test). Mass spectrometry analysis revealed a decrease in intratumoural levels of propionylcarnitine (FC -0.50, $p = 0.039$) and acetylcarnitine (FC -0.40, $p = 0.046$) consistent with inhibition of fatty acid oxidation (wilcoxon rank test). This effect on fatty acid oxidation was validated in pre-clinical in vitro and in vivo breast cancer cell line models. RNASeq revealed upregulation of several pathways associated with mitochondrial and lipid metabolism. Immunohistochemical intratumoural nuclear expression of pAMPK increased 1.5 fold ($p = 0.037$, paired t-test). Serum glucose, c-peptide, insulin and IGF-1 levels significantly decreased but did not correlate with change in $K_{flux}$ or short chain acyl-carnitine levels. Peak serum metformin levels correlated with intratumoural metformin levels ($p = 0.012$, Spearman's rank test).

Conclusion
Our data shows that metformin treatment increases 18F-FDG flux, inhibits fatty acid oxidation and leads to altered gene expression in the mitochondrial (nuclear encoded) transcriptome but that these effects do not correlate with changes in host metabolism. This data provides strong evidence that metformin has a direct effect on breast cancer metabolism at clinical doses.

Understanding different types of cervical screening non-participant: A population-based survey

Background
Uptake of cervical screening in England is around 75% and is trending downwards. This study took a novel approach to characterising screening non-participants using the Precaution Adoption Process Model (PAPM), which describes the stages through which people move before engaging in a health protective behaviour. We aimed to establish the prevalence of different non-participant types and to explore demographic differences between types.

Method
Home-based computer assisted interviews were carried out with screening-eligible women in Britain. Survey items assessed PAPM stage for cervical screening uptake and a range of demographic factors.

Results
Of the 3133 women included in analyses, 2,258 women were up-to-date with screening and intended to be screened in the future (‘maintainers’; 73%). Among non-participants ($n=855$), the three most prevalent non-participant types were 1) those who were unaware of screening (30% of non-participants), 2) those who had decided to be screened, but had not yet attended (50%) and 3) those who had decided not to be screened (14%). Compared with ‘maintainers’, the unaware group and those who had decided to act but had not yet done so tended to be younger. Those who had decided against screening were more likely to be older. Non-white British ethnicity and living in an urban area were associated with being
unaware of screening. All three non-participant types were more likely than ‘maintainers’ to be unmarried and from lower socioeconomic status backgrounds.

**Conclusion**
We found that a substantial proportion of cervical screening non-participants are unaware of screening, and another large group are inclined to attend but have not yet done so. These findings challenge the notion that screening non-participants have made an informed choice not to take part, and point to the need for targeted interventions to raise awareness and facilitate participation in women who would like to attend.

**PET-PANC: Multi-centre trial of 18Fluorine-2-fluoro-2-deoxy-D-glucose positron emission tomography with computed tomography scanning in the diagnosis and management of pancreatic cancer**

**Background**
Pancreatic cancer diagnosis and staging can be difficult in 10-20% of patients. The use of 18Fluorine-2-fluoro-2-deoxy-D-glucose positron emission tomography with computed tomography scanning (PET/CT) may add further value to the diagnosis and staging of pancreatic cancer. The aim of this study was to determine the clinical impact of PET/CT in addition to standard diagnostic workup in patients with suspected pancreatic cancer.

**Method**
Eligible patients with suspected pancreatic cancer underwent PET/CT following multi-detector CT (MDCT). Diagnosis and management decisions were recorded before and after PET/CT. Reference standard diagnosis was based on histology or clinical outcome at 12 months. Primary outcome measure was incremental diagnostic value of PET/CT in addition to MDCT. Secondary outcome measures were changes in diagnosis, staging, and management; cost effectiveness was estimated.

**Results**
A total of 18 UK centres, between 2011 and 2013, recruited 550 patients with complete data and in range PET/CT. 261 patients (47%) had pancreatic cancer. The sensitivity and specificity for the diagnosis of pancreatic cancer for MDCT was 88.5%, 70.6% and for PET/CT was 92.7%, 75.8% respectively. PET/CT demonstrated significant improvement in relative sensitivity (p=0.01) and specificity (p=0.023) compared to MDCT. PET/CT correctly changed the staging of PDAC in 56 patients (14%) (p=0.001). PET/CT influenced management in 250 (45%) of patients and stopped resection in 58 patients (20%) due to have surgery. The benefit of PET/CT was limited in patients with chronic pancreatitis or other pancreatic tumours. PET/CT was associated with a QALY gain of 0.0157 (95% CI -0.0101, 0.0430). In the base case model PET/CT was seen to dominate MDCT alone and is likely to be cost effective.

**Conclusion**
PET/CT provided significant additional benefit in the diagnosis, staging and management of pancreatic cancer. PET/CT was cost effective at current reimbursement rates to the UK NHS.

*This project was funded by the NIHR HTA (project number 08/29/02).
Cancer stigma among ethnic minority women

Background
Qualitative research with ethnic minority groups often finds that participants feel cancer is stigmatised or ‘taboo’. Cancer stigma has also been shown to influence engagement with cancer prevention/early detection behaviours particularly in some ethnic minority groups. To date there have been few studies attempting to quantify perceptions of stigma in ethnic minority populations. We aimed to explore differences in cancer stigma in a sample of ethnic minority women and the association between cancer stigma and attendance at cervical screening.

Method
Women aged 30–60 years were recruited from Indian, Pakistani, Bangladeshi, Caribbean, African and white British backgrounds (n=720, response rate = 65%). Participants completed face-to-face interviews with a multi-lingual interviewer. We assessed socio-demographics, self-reported cervical screening attendance, and four dimensions of cancer stigma: personal responsibility, awkwardness, avoidance and community-level stigma.

Results
There were significant differences by ethnic group for each of the four stigma dimensions (p<.001 for each), with White British women scoring lowest on each. Differences on individual items were striking, for example <5% of Bangladeshi and Pakistani women believed cancer was talked about openly in their community compared to 97% of White British women. Across all ethnic groups, personal responsibility, awkwardness and avoidance scores were significantly lower among those born in the UK, but there were no differences in community stigma. Cancer stigma was significantly associated with increased odds of non-attendance for cervical screening. This was the case for community stigma (OR=1.09), personal responsibility (OR=1.12), awkwardness (OR=1.12) and avoidance (OR=1.15). Odds ratios were small but highly significant (p<.001).

Conclusion
While low in the general population, cancer stigma is prevalent in ethnic minority groups, even among the younger generation, and is associated with cervical screening status. Interventions to tackle stigma in ethnic minority populations could be a first step to addressing ethnic inequalities in cancer awareness and preventive behaviours.
by PD-L1 status, BRAF mutation status, and M-stage. Co-primary endpoints were mPFS and overall survival (data remain immature). Secondary endpoints included efficacy by PD-L1 status and safety.

Results
At ≥18 months of follow-up, mPFS continued to be significantly longer for NIVO+IPI (11.5 months) and NIVO (6.9 months) vs. IPI (2.9 months) (p<0.001); hazard ratio for NIVO+IPI vs. NIVO (exploratory endpoint): 0.76 (95% CI: 0.60–0.92). Median duration of response in 181/314 (57.6%) NIVO+IPI responders has not been reached, and was 22.3 and 14.4 months in 138/316 (43.7%) NIVO and 60/315 (19.0%) IPI responders, respectively. For PD-L1 tumour expression ≥5%, mPFS was similar between NIVO+IPI and NIVO, but ORR was numerically higher in the former. For NIVO+IPI, NIVO, and IPI groups, mPFS was 15.5, 5.6, and 4.0 months in BRAF mutation-positive patients and was 11.3, 7.1, and 2.8 months in BRAF wild-type patients, respectively. The frequency and types of drug-related grade 3/4 AEs were consistent with earlier reports (NIVO+IPI, 56.5%; NIVO, 19.8%; IPI, 27.0%).

Conclusion
NIVO+IPI and NIVO continue to demonstrate superior clinical activity vs. IPI. NIVO+IPI resulted in numerically greater PFS and ORR than NIVO alone, including in patients with a BRAF mutation.

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Cell fate imbalance in the oesophageal epithelium: Mutant cell competition

Background
Notch signalling pathway is of significant importance for cell fate determination in several systems, and has been linked to cancer development including squamous oesophageal carcinogenesis. However, the role of Notch in oesophageal progenitor cell behaviour still remains largely unknown. We have previously reported how the squamous epithelium of the mouse oesophagus is maintained and repaired by a functionally equivalent progenitor population.

Method
To investigate how Notch may regulate such cell fate decisions, we expressed a dominant negative form of mastermind-like mutant fused to GFP, which blocks canonical Notch signalling of oesophageal epithelial progenitors at single cell resolution in the mouse oesophagus. Lineage tracing of GFP labelled single cell derived clones was performed to unveil mutant cell dynamics.

Results
Analysis of labelled clone data over one year time course reveals a significant alteration in mutant cell behaviour. The results indicate that mutant clones expand over time due to accelerated cell proliferation, together with a selective block of terminal divisions. Additionally, activation of Notch signalling in adjacent non-mutant cells accelerates their stratification at the clone boundary, promoting clonal expansion. The latter explains how mutant clones progressively take over the entire oesophageal epithelium over one year period.
**Conclusion**

Our observations suggest that loss of function Notch mutations confer clonal dominance, leading to clonal immortalization and field change. As a result, Notch inhibition increases the number and size of tumours that develop following carcinogen treatment. These observations illustrate that, as well as cooperating at the molecular level within cells, oncogenic mutations may also interact at the level of cell dynamics in early cancer evolution.

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**The molecular epidemiology of rare and compound EGFR mutations in 14,304 non-small cell lung carcinomas**

**Background**

The epidemiology of the common Del19 and L858R mutations in non-small cell lung cancer (NSCLC) has been well-characterised. However, few studies have examined the epidemiological characteristics of rarer mutations. We used a large data set from our centre to relate the incidence of these mutations with patient demographics.

**Method**

All NSCLC cases referred to our centre are tested for EGFR mutations; this is not restricted to adenocarcinoma but also includes NSCLC NOS and tumours showing squamous differentiation. We use real time PCR, using the therascreen EGFR RGQ PCR Kit, which detects exon 19 deletions, T790M, L858R, L861Q, G719X, S768I and exon 20 insertions.

14,304 EGFR mutation results from non-small cell lung cancer specimens tested between 2009 and 2015 were retrospectively reviewed. 1,382 specimens harboured mutations (9.7%), of which 270 contained mutations other than Del19 and L858R. Information was collected about mutation type, patient sex and age.

**Results**

19.5% of all mutations were rare mutations:

<table>
<thead>
<tr>
<th>Mutation</th>
<th>Incidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>G719X</td>
<td>64</td>
</tr>
<tr>
<td>L861Q</td>
<td>45</td>
</tr>
<tr>
<td>S768I</td>
<td>14</td>
</tr>
<tr>
<td>Ins20</td>
<td>48</td>
</tr>
<tr>
<td>T790M</td>
<td>3</td>
</tr>
<tr>
<td>Compound mutations</td>
<td>76</td>
</tr>
</tbody>
</table>

Overall, there was no significant difference in the incidence of rare mutations by sex, and although they were more common with age this did not achieve statistical significance. None of the individual mutations showed a significant difference in incidence by sex. L861Q mutations were significantly more common in older patients, becoming as common as Del19 in patients older than 90 years. T790M, S768I and G719X were markedly overrepresented in the compound mutations.

**Conclusion**

Rare EGFR mutations are more common than generally assumed, accounting in our series for one fifth of mutations. L861Q is particularly common, being the joint-second commonest mutation in older patient groups. It is therefore important that molecular testing be sensitive to these mutations to avoid denying significant numbers of patients potentially beneficial therapies.
Scientific symposia

BASO~ACS Breast session: Breast reconstruction
Hosted by Lynda Wyld and Tibor Kovacs, BASO~ACS

08.15 – 08.35
Room 11

Isabel Rubio,
Hospital Universitario Vall d’Hebron,
Barcelona, Spain

The oncological safety of nipple sparing mastectomy:
The European INSPIRE Project
Nipple Sparing Mastectomy (NSM) entails the conservation of the nipple–areola complex (NAC) as well as the skin envelope while performing a complete excision of all the mammary gland. NSM and immediate breast reconstruction has been practiced more and more often in the last decade in treating invasive and in situ breast cancer and for women with an increased risk of developing breast cancer. One significant advantage of the NSM technique is the removal of the whole breast tissue while preserving native breast integrity, the nipple-areola complex as well as the submammary fold, therefore improving the cosmetic outcomes. As rates of NSM continue to increase, it is important to retrieve confirmatory evidence in support of the oncologic safety of the technique. To test the effectiveness and safety of NSM, a large prospective data collection has been set, the INSPIRE project. The INSPIRE project aims to gain insight in treatment strategies for women undergoing NSM and immediate breast reconstruction for breast cancer or for risk reducing purposes. The target is to provide prospective robust evidence on its oncological safety; complications (associated risks of nipple and skin necrosis, infection rates, reconstruction loss, nipple symmetry) and patient reported outcome measures (PROMs).

08.40 – 09.00
Room 11

Ms Siobhan Laws,
Royal Hampshire County Hospital, UK

The oncological safety of lipomodelling in breast cancer
At the time of printing, this abstract had not been not received. Check the Conference App for further details.

BASO~ACS and AUGIS session
Hosted by Hassan Malik, BASO~ACS and Giles Toogood, AUGIS

17.35 – 18.05
Room 11

John Neoptolemos,
University of Liverpool, UK

Advancing outcomes in pancreatic cancer: From bench to bedside

18.05 – 18.35
Room 11

James Garden,
University of Edinburgh, UK

Surgical training at a distance
Workshops

De-mystifying today’s science

08.15 – 09.00
Room 3B

Hosted by Elaine Vickers, Science Communicated, Sheffield, UK

Do words like signal transduction, epigenetics, genomics and biomarkers bring a puzzled frown to your face? Do you plan to attend today’s plenary lectures whilst fearing that you won’t understand a word? Well never fear!

Elaine will explain many of the words, concepts and ideas behind the day’s plenary lectures. Similar to previous years’ workshops, she will use diagrams and illustrations to provide clear, easy-to-understand explanations of complicated biological concepts.

The workshop is geared towards non-scientists, such as doctors, nurses, trials staff and patients who’d like to get the most out of this year’s conference.

BACR educational workshop: Bioinformatics for the uninitiated

08.15 – 09.00
Room 3A

Hosted by Crispin Miller, Cancer Research UK, Manchester Institute, UK

Advances in deep sequencing have have provided enormous insights into the underlying genetic changes that promote tumour growth and maintenance. Central to these discoveries are software tools and analysis pipelines that are used to map these mutations to the genome, and to correlate changes in the underlying genetics of a tumour with changes in phenotype. Cancer research has rapidly become highly multidisciplinary, as software engineers and mathematicians work alongside biologists and clinicians in order to make sense of genomic data; an understanding of how these analysis tools function is critical to the proper interpretation of their output. This workshop, which is aimed at clinical and biological scientists from a non computing background, will provide an overview of the current approaches used to analyse genomics data, a gentle introduction to how they work, as well as some of the key principles that underpin genomic data analysis.

Molecular diagnostics: Lessons being learnt

11.00 – 12.30
Hall 1C

Hosted by Karin Oien, University of Glasgow, UK

Molecular diagnostics are key to the implementation of stratified/precision medicine approaches but their development and particularly their path to clinical application and practice are often complex. Pitfalls, and at least some of their solutions, have been elegantly demonstrated by large-scale initiatives ranging from the CRUK Stratified Medicine Programme and Accelerators, MRC/EPSRC Molecular Pathology Nodes through to Genomics England; and their integration and learning of lessons is being facilitated by NCRI’s CM-Path initiative. Exploring these will be the aim of this workshop: what works, what doesn’t, and what are the barriers and solutions? (Or: what do you wish you’d known at the start?)

Speakers:
John Le Quesne, MRC Toxicology Unit
Emily Shaw, University Hospital Southampton NHS Foundation Trust
Louise Jones, Barts Cancer Institute, Queen Mary University of London
Discover the science behind new targeted treatments that are changing the face of cancer care. Learn from experts and gain a deeper understanding of how these treatments work to better support your patients.

Find out more at the Cancer Research UK Stand (No. 30) on Tuesday 8th November at 10:25-11:00.
Dragons’ Den workshop

The NCRI Consumer Forum is once again hosting a Dragons’ Den session. This session offers researchers the opportunity to meet with small groups of knowledgeable consumers who are already involved in research, as well as other consumers (patients or carers) who are attending the Conference. Researchers are welcome to present new ideas, to discuss problems with studies already running, to seek endorsements for funding, to disseminate findings to patients or to talk about how to create effective consumer involvement in their work.

This session offers practical on-the-spot advice for those who want to engage some consumers to offer long-term support, or just want to talk through an idea at the back of their mind.

Who are the friendly dragons?

Patients and carers who are experienced in cancer research, including NCRI Consumer Forum members who sit on Clinical Studies Groups and other NCRI initiatives, members of the Independent Cancer Patients’ Voice (ICPV), consumers who sit on funding committees, consumers funded by Cancer Research UK, Macmillan, Tenovus or other charities, or those who work with CTUs or other institutions. The session is also open to any Conference attendee who wish to take part, for example, researchers who may have someone in their family affected by cancer, or who simply wish to see some consumer involvement in action.

Women in Science

For the first time at the NCRI Cancer Conference, we will hold a ‘Women in Science workshop’ hosted by this year’s Chair, Professor Caroline Dive.

The workshop will include a motivational talk by Professor Jessie English, whose career encompasses leadership roles in both Academic and Industry sectors, and who has chaired the AACR women in cancer research council, and will be followed by table discussions hosted by leading experts – topics will include Leadership Skills, Mentorship, Clinical Scientist Career, Scientific Journalism, Starting your own lab and many more.
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Information, patients and the public 4
Supportive, palliative care, survivorship 5
The cancer cell and model systems 6
## Programme at a glance

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<th>Location</th>
<th>Title</th>
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<tr>
<td>08.15 – 09.00</td>
<td>Room 3B</td>
<td>De-mystifying today’s science</td>
<td>Elaine Vickers, Science Communicated, Sheffield, UK</td>
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<tr>
<td>08.15 – 09.00</td>
<td>Room 3A</td>
<td>BACR educational workshop: Artificial cells – smart delivery systems for cancer therapy</td>
<td>Oscar Ces, Imperial College London, UK</td>
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### Plenary lectures

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<th>Location</th>
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<tbody>
<tr>
<td>09.05 – 09.45</td>
<td>Hall 1A</td>
<td>Adaptive resistance to tumour immunity: From a hypothesis to anti-PD-1/PD-L1 therapy</td>
<td>Johann de Bono, The Institute of Cancer Research, London, UK</td>
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<tr>
<td>09.45 – 10.25</td>
<td>Hall 1A</td>
<td>Multimodal genomics to improve precision oncology for prostate cancer: Embrace complexity!</td>
<td>Rob Bristow, Ontario Cancer Institute/Princess Margaret Hospital, University of Toronto, Canada</td>
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### Networking, exhibition and poster viewing

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<td>Hall 2</td>
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### Symposia

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<th>Title</th>
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<tbody>
<tr>
<td>11.00 – 12.30</td>
<td>Room 3A</td>
<td>Prostate cancer</td>
<td>Johann de Bono, The Institute of Cancer Research, London, UK</td>
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<tr>
<td>11.00 – 12.30</td>
<td>Hall 1A</td>
<td>Targeting cancer evolution</td>
<td>Charles Swanton, The Francis Crick Institute and University College London Cancer Institute, UK</td>
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<tr>
<td>11.00 – 12.30</td>
<td>Hall 1B</td>
<td>Tumour cell death</td>
<td>Henning Walczak, University College London Cancer Institute, UK</td>
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<tr>
<td>11.00 – 12.30</td>
<td>Room 3B</td>
<td>Where are the gains in technical radiotherapy?</td>
<td>Jeanette Dickson, The Royal College of Radiologists</td>
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### Proffered paper session 2

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<th>Time</th>
<th>Location</th>
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<tbody>
<tr>
<td>11.00 – 12.30</td>
<td>Hall 1C</td>
<td></td>
<td>Caroline Dive, Cancer Research UK Manchester Institute, UK</td>
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</table>

### Workshop

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</tr>
</thead>
<tbody>
<tr>
<td>11.00 – 12.30</td>
<td>Room 11</td>
<td>Accelerating adoption of research findings into NHS clinical practice</td>
<td>Emma Greenwood, Cancer Research UK, London, UK</td>
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Lunch, networking, exhibition and poster viewing

12.30 – 14.00
Hall 2

The Royal College of Radiologists proffered paper session

13.00 – 14.00
Room 3B
One of the presentations in this session will be awarded an RCR Ross Prize for the best oral presentation, as judged by an RCR judging panel.

Scientific symposium

13.00 – 14.00
Room 3A
Immunotherapy for genitourinary cancers and beyond
Hosted by Bristol-Myers Squibb

Plenary lectures
Chaired by Chris Lord, The Institute of Cancer Research, London, UK

14.05 – 14.45
Hall 1A
Targeting tumour evolvability
Reuben Harris, Howard Hughes Medical Institute, University of Minnesota, USA

14.45 – 15.25
Hall 1A
Improving the evidence base for symptom control in advanced cancer: Phase III studies of common interventions
David Currow, Flinders University, Adelaide, Australia

Clinical Trials Showcase part 2
Hosted by Matt Seymour, NIHR Clinical Research Network: Cancer, Leeds, UK and National Cancer Research Institute

15.25 – 15.40
Hall 1A
CONVERT: An international randomised trial of concurrent chemoradiotherapy comparing twice-daily and once-daily radiotherapy schedules in patients with limited stage small cell lung cancer
Corinne Faivre-Finn, University of Manchester, UK

15.40 – 15.55
Hall 1A
First Results of COSTAR (CRUK/08/004): A randomised trial of 3-Dimensional Conformal Radiotherapy (3DCRT) vs Cochlea-Sparing Intensity Modulated Radiotherapy (CS-IMRT) in patients with parotid cancer
Chris Nutting, The Royal Marsden NHS Foundation Trust, London, UK

Networking, exhibition and poster viewing

15.55 – 16.30
Hall 2

Parallel sessions

16.30 – 18.30
Room 11
How we talk about cancer
Hosted by Elena Semino, Lancaster University, UK

Join us next year: 5-8 November 2017  conference.ncri.org.uk 83
### Programme at a glance (continued)

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<tr>
<th>Time</th>
<th>Session</th>
<th>Location</th>
<th>Host</th>
<th>Details</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>16.30 – 18.30</strong></td>
<td>Advances in cancer drug discovery and development: Small molecule versus antibody-based strategies</td>
<td>Hall 1A</td>
<td>Michelle Garrett</td>
<td>University of Kent, UK</td>
</tr>
<tr>
<td></td>
<td>Delivering patient-centred cancer care: Meeting the psychosocial needs of people living with and beyond cancer</td>
<td>Room 3A</td>
<td>Nick Hulbert-Williams</td>
<td>University of Chester, UK</td>
</tr>
<tr>
<td></td>
<td>Epstein-Barr virus: Continuing insights into cancer and its treatment</td>
<td>Room 12</td>
<td>Neil Steven and Graham Taylor</td>
<td>University of Birmingham, UK</td>
</tr>
<tr>
<td></td>
<td>Small cell lung cancer: Genomics, biology and progress with clinical management of this recalcitrant tumour</td>
<td>Hall 1B</td>
<td>Caroline Dive</td>
<td>Cancer Research UK Manchester Institute, UK</td>
</tr>
<tr>
<td><strong>16.30 – 18.00</strong></td>
<td>Novel targeted therapies for cancer cachexia – a new era in cancer treatment</td>
<td>Hall 1C</td>
<td>Miriam Johnson</td>
<td>University of Hull, UK</td>
</tr>
<tr>
<td></td>
<td>Improving outcomes with drug/radiotherapy combinations</td>
<td>Room 3B</td>
<td>Geoffrey Higgins</td>
<td>University of Oxford, UK</td>
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<tr>
<td><strong>Workshop</strong></td>
<td></td>
<td>Room 4</td>
<td>Judith Bliss</td>
<td>The Institute of Cancer Research, London, UK</td>
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<tr>
<td><strong>16.30 – 18.30</strong></td>
<td>The role of CTUs in optimising clinical research – beyond Figure 1</td>
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<tr>
<td><strong>Conference dinner (ticketed event)</strong></td>
<td></td>
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<td></td>
<td>Limited tickets are available for this event. Please go to the registration desk no later than noon on Tuesday 8 November to enquire. Only ticket holders will be admitted.</td>
</tr>
</tbody>
</table>
The Society and College of Radiographers is the professional body and trade union for radiographers and associated professionals with over 27,000 members. We represent the interests of a range of practitioners in the fields of clinical imaging, radiation therapy and oncology.

Whether you are a practitioner or manager, academic or researcher, please come and visit us on **stand 6** to find out more about:

- Our work at local, regional and national level to promote the science and practice of radiotherapy and the interests of our members and their patients.
- Our range of excellent CPD resources, including the acclaimed programmes we have developed in partnership with e-Learning for Healthcare. These include programmes in radiotherapy, clinical image interpretation and radiation protection.
- Our College of Radiographers Industry Partnership Scheme (CoRIPS) which provides awards for research, many of them for first time researchers.
- The wide range of benefits that membership of the Society of Radiographers can offer.
Adaptive resistance to tumour immunity: From a hypothesis to anti-PD-1/PD-L1 therapy

Immune responses are tightly controlled by immunomodulatory pathways which constitute various receptors and ligands and could positively or negatively influence the quality and the direction of inflammatory immune responses. The PD-1/PD-L1 (B7-H1) immune modulatory pathway plays important roles in suppressing antigen-specific immune responses and inflammation. Selective expression of PD-L1/B7-H1 in tumour microenvironment and subsequent interaction with PD-1 on T cells is demonstrated to be a major mechanism of losing T cell immunity in tumour sites in a significant fraction of cancer patients. Monoclonal antibodies blocking this pathway induced tumour regression in more than 15 different types of advanced cancers, especially solid tumours. The treatment is well-tolerated and the clinical responses are long-lasting. Understanding of adaptive resistance mechanism underlying response and resistance to anti-PD-1/PD-L1 therapy is critical for further improvement of cancer therapy in the future. Dr Chen will discuss basic concept of immune modulatory pathways; principles of anti-PD-1/PD-L1 therapy and perspectives of cancer therapy using immune modulation.


This activity has been supported by a grant from Roche Products Limited. Roche Products Limited has had no control over the educational content of this activity.

Multimodal genomics to improve precision oncology for prostate cancer: Embrace complexity!

Prostate cancer is a heterogenous disease with failure rates approaching 30-40% after precision radiotherapy or surgery. Many recurrences are due to pre-existing occult metastases at the time of local treatment. Genomic studies have shown that there exists heterogeneity in mutations, copy number alterations and methylation amongst lesions that have the same Gleason Score and these can have variable aggression. Using a whole genome sequencing approach, we have categorised men into different genetic sub-classes using these indices that are independent prognostic variables that predict for metastatic disease. Adding in information regarding the tumour microenvironment, sub-clonality and sub-pathologies (cribiform-IDC) increases precision of the genomics. These biomarkers can then be used to intensify systemic therapies to those men that harbour occult metastases to improve cure and prevent metastatic castrate-resistant prostate cancer.
Cancers often display incredible genetic heterogeneity characterised by hundreds of gross chromosomal aberrations and tens of thousands of somatic mutations. Tumour evolution is thought to be ongoing and the sum of all exogenous and endogenous mutagenic processes. Many viruses evolve at much higher rates, and virus evolution is thought to be similarly complex with both viral and cellular processes contributing to overall levels of genetic variation. Historically, tumour and virus evolution are thought of as largely independent processes. However, many studies have converged recently on the antiviral enzyme APOBEC3B as a major source of virus and tumour mutagenesis and evolution. This presentation will highlight recent progress in this area including how both virus-dependent and virus-independent mechanisms upregulate APOBEC3B and how starving tumour cells of mutational fuel can suppress evolution of resistance mutations and boost efficacies of current drugs.

At the time of printing, this abstract had not been received. Check the Conference App for further details.
Clinical Trials Showcase part 2

Clinical Trials Showcase
Hosted by Matt Seymour, NIHR Clinical Research Network: Cancer, Leeds, UK and National Cancer Research Institute

CONVERT: An international randomised trial of concurrent chemo-radiotherapy comparing twice-daily and once-daily radiotherapy schedules in patients with limited stage small cell lung cancer Corinne Faivre-Finn, University of Manchester, UK

**Background**
Concurrent chemo-radiotherapy (cCTRT) is the standard of care for good PS LS-SCLC but there is no international consensus on a standard regimen. Our aim was to compare overall survival and toxicity of twice-daily (BD) with once-daily (OD) RT using modern conformal RT techniques given concurrently with chemotherapy.

**Method**
Patients were randomised 1:1 to receive 45Gy in 30 BD fractions over 3 weeks or 66Gy in 33 OD fractions over 6.5 weeks starting on day 22 of cycle 1 chemotherapy (4 to 6 cycles of Cisplatin 25mg/m2 days 1-3 or 75mg/m2 day 1 with Etoposide 100mg/m2 days 1-3), followed by PCI if indicated. RT was planned using 3D conformal or IMRT. Patients were stratified by centre, 4/6 cycles CT and PS 0,1/2. The primary endpoint was 2-year survival and all analyses were by intention to treat.

**Results**
547 patients were recruited between 2008-2013 from 88 centres. Patients’ characteristics were well balanced in both arms. At a median follow up of 45 months for those alive; two-year survival was 56% (95% CI 50-61) vs 51% (95% CI 45-57) and median overall survival was 30 months (95% CI 24-34) versus 25 months (95% CI 21-31) (HR 1.17, 95% CI 0.95-1.45; p=0.15) for BD and OD treatment, respectively. Toxicities were comparable except for significantly more grade 3/4 neutropenia (74% BD vs 65% OD, p=0.03). There was no statistical difference, between BD and OD respectively, in rates of grade 3/4 oesophagitis (19%, 19%), and grade 3/4 radiation pneumonitis was rare (2.5%, 2.2%).

**Conclusion**
OD RT did not result in a superior survival or worse toxicity than BD RT, supporting the use of either regimen for standard of care treatment of LS-SCLC with good PS. The survival for both regimens was higher than previously reported and using modern RT techniques radiation toxicities were lower than expected.

First Results of COSTAR (CRUK/08/004): A randomised trial of 3-Dimensional Conformal Radiotherapy (3DCRT) vs Cochlea-Sparing Intensity Modulated Radiotherapy (CS-IMRT) in patients with parotid cancer

**Background**
30-50% of patients receiving post-operative RT for parotid cancer experience ipsilateral hearing loss. IMRT can reduce radiation dose to cochlea. COSTAR investigated the role of IMRT in reducing hearing loss in these patients.

**Method**
Patients with histologically confirmed carcinoma of the parotid gland (pT1-4, pN0-3, M0) were randomised 1:1 to receive CT-planned 3DCRT or CS-IMRT. 60Gy (R0 resection) or 65Gy (R1-2)
in 30 fractions were delivered over 6 weeks. Treatment allocation used minimisation, balancing for centre and planned RT dose. The primary endpoint was proportion of patients with hearing loss in the ipsilateral cochlea of ≥10dB measured by bone conduction at 4000Hz 12 months (m) after RT; compared between randomized groups by an exact test (α=0.05). Secondary endpoints (α=0.01) included hearing loss at 6 and 24m, vestibular function, acute and late toxicity, patient reported quality of life (including Glasgow Hearing Aid Benefit Profile (GHABP), time to tumour recurrence and survival.

**Results**

110 patients (54 3DCRT; 56 CS-IMRT) were randomised between 2008 and 2013 from 22 UK centres. 99 (90%) patients were R1-2 (47 3DCRT; 52 CS-IMRT). Mean dose to the ipsilateral cochlea was 56.2Gy for 3DCRT and 35.7Gy for CS-IMRT, (p<0.001). 66/110 (60%) patients were evaluable for the primary endpoint. At 12ms, a loss of ≥10dB in ipsilateral bone conduction was observed in 14/35 (40%) 3DCRT and 11/31 (36%) CS-IMRT patients (p=0.80). No statistically significant differences in bone or air conduction were observed at 6 or 24m after RT nor for any GHABP initial disability or handicap subscales, vestibular function, acute or late toxicity, overall quality of life, time to tumour recurrence or survival.

**Conclusion**

IMRT reduced the radiation dose below the accepted tolerance of the cochlea. This did not lead to a statistically significant reduction in the proportion of patients with hearing loss in the ipsilateral ear at 12 months after RT.
Symposia

Prostate cancer
Hosted by Johann de Bono, The Institute of Cancer Research, London, UK

11.00 – 11.05 Introduction by the host

11.05 – 11.30 Targeting tumour-infiltrating myeloid cells for prostate cancer therapy
Andrea Alimonti, Institute of Oncology Research (IOR), Oncology Institute of Southern Switzerland (IOSI), Switzerland

11.30 – 11.55 Molecularly stratified therapeutic strategies for lethal prostate cancer
Johann de Bono, The Institute of Cancer Research, London, UK

11.55 – 12.20 Targeting the androgen receptor in prostate cancer: A resilient foe
Peter Nelson, Fred Hutchinson Cancer Research Center, USA

12.20 – 12.30 Discussion

Targeting cancer evolution
Hosted by Charles Swanton, The Francis Crick Institute and University College London Cancer Institute, UK

11.00 – 11.05 Introduction by the host

11.05 – 11.30 Drug discovery and development to overcome cancer evolution and drug resistance
Paul Workman, The Institute of Cancer Research, London, UK

11.30 – 11.55 DNA replication stress mediates APOBEC3 family mutagenesis in breast cancer
Nnenna Kanu, University College London, UK

11.55 – 12.20 Mutational signatures and cancer evolution
Serena Nik-Zainal, Wellcome Trust Sanger Institute, UK

12.20 – 12.30 Discussion

Tumour cell death
Hosted by Henning Walczak, University College London Cancer Institute, UK

11.00 – 11.05 Introduction by the host

11.05 – 11.30 Which pro-survival BCL-2 family member should be targeted for the treatment of which cancer?
Andreas Strasser, Walter and Eliza Hall Institute of Medical Research, Australia

11.30 – 11.55 Identifying human cancers to target with BCL-2 inhibition
Anthony Letai, Dana-Farber Cancer Institute, USA

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11.55 – 12.20  Harnessing the complex relationship between mitosis and cell death
Pascal Meier, The Institute of Cancer Research, London, UK

12.20 – 12.30  Discussion

Where are the gains in technical radiotherapy?
Hosted by Jeanette Dickson, The Royal College of Radiologists

11.00 – 11.05  Introduction by the host

11.05 – 11.30  George Edelstyn lecture
Protons
Steve Hahn, The University of Texas MD Anderson Cancer Center, USA

11.30 – 11.55  Advances in SABR
Maria Hawkins, Gray Institute for Radiation Oncology and Biology,
Oxford University, UK

11.55 – 12.20  Motion management in radiotherapy
Marcel van Herk, The Christie Hospital, Manchester, UK

12.20 – 12.30  Discussion
Symposia abstracts

Prostate cancer

11.00 – 11.05
Room 3A

Introduction
Our understanding of prostate cancer genomics is burgeoning and giving us an increasing understanding of the inter-patient heterogeneity of this complex disease, distinguishing increasingly lethal cancers from indolent disease. We are also elucidating the genomic diversity of metastatic disease while appreciating the commonest mechanisms of treatment resistance. This session will focus on recent advances in key work that is impacting patient care and cancer risk and will impact understanding of not only the better diagnosis and treatment of advanced disease but also a better understanding of rational strategies to improve outcome of high risk localised disease and direct targeted screening strategies.

11.05 – 11.30
Room 3A

Targeting tumour-infiltrating myeloid cells for prostate cancer therapy
Tumour-infiltrating myeloid cells are a heterogeneous and immunosuppressive cell subset that blocks the proliferation and the activity of T and natural killer (NK) cells and promotes tumour vasculogenesis and progression. Recent evidences demonstrate that the recruitment of myeloid cells in tumours also blocks senescence induced by chemotherapy promoting chemoresistance. I will present novel evidence demonstrating that compounds that interfere with the skewing of tumour-associated macrophages in M2 promote prostate cancer inhibition by enhancing senescence.

11.30 – 11.55
Room 3A

Molecularly stratified therapeutic strategies for lethal prostate cancer
At the time of printing, this abstract had not been received. Check the Conference App for further details.

11.55 – 12.20
Room 3A

Targeting the androgen receptor in prostate cancer: A resilient foe
Nuclear hormone receptors can be considered the first precision oncology targets. To date, therapeutics designed to interfere with androgen receptor (AR) signaling remain the main treatment approach for advanced prostate cancer. While dramatic responses are observed, reactivation of AR signaling at disease progression is a nearly universal occurrence. This presentation will discuss the molecular events that underlie resurrection of the AR program in advanced prostate cancer with an orientation toward new treatment strategies.

12.20 – 12.30
Room 3A

Discussion
**Targeting cancer evolution**

**Introduction**

In this session, an overview of the APOBEC family of cytidine deaminases will be presented. This family of enzymes is an important initiator of mutagenesis in human cancer, and has recently been found to generate branched tumour evolution in several solid tumour types. Targeting APOBEC may provide a mechanism to limit tumour evolution, diversity and adaptation through therapy and drug resistance. This session will provide an update of the role of the APOBEC enzymes in human malignancy, their impact on genetic diversity and insight into how this family might be targeted to improve cancer outcomes.

**Drug discovery and development to overcome cancer evolution and drug resistance**

Over the last two decades we have progressed from traditional one-size-fits-all cytotoxic chemotherapy to discovery and successful use of innovative precision drugs that counteract effects of particular cancer genes that drive individual cancers — known as ‘oncogene addiction’. These include a number of drugs discovered in our Unit and progressed to the clinic, including the antihormonal agent abiraterone and inhibitors of oncogenic protein kinases and PI3 kinase. Despite substantial progress made, we and others have discovered many different ways by which resistance develops to both targeted and cytotoxic cancer drugs — including biochemical rewiring and genetic instability, cellular heterogeneity and Darwinian selection allowing evolution of resistant clones. Overcoming drug resistance is the greatest challenge that we now face in the cancer clinic — analogous to antibiotic resistance and resistance to HIV drugs. Achieving substantial increases in survival and cure rates will require diverse innovative approaches including: 1) discovering drugs acting across a wider coverage of cancer targets; 2) targeting non-oncogene addiction mechanisms including adaptive stress and proteostasis pathways; 3) exploiting genetic instability, DNA repair pathways and other evolutionary mechanisms; 4) activating a wider range of immune responses; and 5) creative combinations of the above.

**DNA replication stress mediates APOBEC3 family mutagenesis in breast cancer**

The APOBEC3 family of cytidine deaminases mutate the cancer genome in a range of cancer types. Although many studies have documented the downstream effects of APOBEC3 activity through next-generation sequencing, less is known about their upstream regulation. We sought to identify a molecular basis for APOBEC3 expression and activation. We identified that HER2 amplification and PTEN loss promote DNA replication stress and APOBEC3 activity in vitro and correlates with APOBEC3 mutagenesis in vivo. HER2-enriched breast carcinomas, display evidence of elevated levels of replication stress associated DNA damage in vivo. Chemical and cytotoxic induction of replication stress, through aphidicolin, gemcitabine, camptothecin or hydroxyurea exposure, activate transcription of APOBEC3B via an ATR/Chk1-dependent pathway in vitro. APOBEC3 activation can be attenuated through repression of oncogenic signalling, small molecule inhibition of receptor tyrosine kinase signalling and alleviation of replication stress through nucleoside supplementation. Our data link oncogene, loss of tumour suppressor gene and drug-induced replication stress with APOBEC3 activity, providing new insights into how cytidine deaminase-induced mutagenesis might be activated in tumourigenesis and limited therapeutically.
Mutational signatures and cancer evolution

Mutational signatures are the imprints of the biological processes that have gone awry in human cells. We previously outlined the methods for identifying and quantifying base substitution mutational signatures present in primary human cancers (http://cancer.sanger.ac.uk/cosmic/signatures). Here, using a highly-curated cohort of 560 whole genome sequenced breast cancers, we extend the understanding of mutational signatures to include six novel rearrangement signatures. We further demonstrate how mutational signatures can report on subclonal architecture of the cancer genome distinguishing clinically relevant subpopulations in breast cancer.

Introduction

For cancer researchers and patients alike, probably the only good cancer cell is a dead cancer cell. Unfortunately, cancer cells have acquired resistance to the normal physiological mechanisms by which non-transformed cells die on a regular basis in virtually all tissues in the body, thereby allowing for normal tissue homeostasis to take place as they are replaced by new cells derived from division of normal tissue stem cells. Thus, in many cases cancer is the consequence of loss of the capacity to die rather than that of an increased ability to grow. After approximately 25 years of research in the field of cell death around the globe we have come to understand the mechanisms of cell death in normal versus cancerous cells to an extent that has enabled the development of several different new classes of drugs to treat cancer that work by directly targeting the cell death pathways uncovered by this research. In addition, this research has led to new ways of molecularly identifying the cause of cancer cell resistance to therapy. Both themes will be covered in the talks in this session on tumour cell death.

Which pro-survival BCL-2 family member should be targeted for the treatment of which cancer?

Impaired apoptosis is considered one of the prerequisites for the development of most, if not all, cancers, but the mechanisms that guarantee the sustained survival of most cancer cells remain unknown. Members of the BCL-2 family of proteins are key regulators of apoptosis and include proteins essential for cell survival and those required to initiate cell death. Studies with transgenic mice have shown that over-expression of BCL-2 or related pro-survival family proteins, such as BCL-XL or MCL-1, can promote tumourgenesis, particularly in conjunction with mutations that deregulate cell cycle control, such as deregulated c-MYC expression. It is, however, not known whether expression of pro-survival BCL-2 family members under endogenous control is required to maintain the survival of cells undergoing neoplastic transformation. Using Eµ-Myc transgenic mice, a well-characterised model of human Burkitt’s lymphoma, and other murine cancer models, we investigated the role of BCL-2 pro-survival proteins when expressed under endogenous control in lymphoma development. BCL-2 was found to be dispensable for the development of Eµ-myc pre-B/B lymphoma. In contrast, loss of BCL-XL and even more remarkable, loss of a single allele of Mcl-1 greatly impaired lymphoma development. Experiments with inducible knockout mice
demonstrated that MCL-1 but not BCL-2 or BCL-XL is essential for the sustained survival and expansion of c-MYC-driven malignant pre-B/B lymphoma, AML driven by various oncogenes and T cell lymphoma driven by loss of p53. Remarkably, even loss of one allele of Mcl-1 greatly impaired lymphoma growth. These findings were translated into human lymphoid malignancies by using inducible expression of guide RNAs that target different Bcl-2 family members. Such studies showed that MCL-1 is also critical for the sustained survival and expansion of Burkitt Lymphoma, a c-MYC-driven malignancy. These observations indicate that even relatively weak targeting of MCL-1 may be an attractive strategy for the treatment of c-MYC-driven hematological malignancies and possibly also other cancers driven by different oncogenic lesions.

**Identifying human cancers to target with BCL-2 inhibition**

Selectively targeting the apoptotic pathway in human cancers has recently become a clinical reality with the FDA approval of the BCL-2 inhibitor, venetoclax, in chronic lymphocytic leukemia (CLL). How can we determine what cancers are best targeted with this type of therapy? In this talk, Dr Letai will review how BH3 profiling, a functional assay of mitochondrial dependence on anti-apoptotic proteins, identified the BCL-2 dependence of CLL. He will discuss other cancers, including acute myeloid leukemia (AML), acute lymphoblastic leukemia (ALL), multiple myeloma and blastic plasmacytoid dendritic cell neoplasm (BPDCN) where BCL-2 inhibition shows promise. He will also outline how to identify drugs that enhance BCL-2 dependence as a strategy for rationally choosing combinations with drugs like venetoclax. While there is considerable attention paid to genomic means for matching cancers and individual patients to the right drugs, it is important to recognise the many examples, as here, of cancers that are sensitive to a targeted therapy without any indicative genetic lesion. In these cases, a functional precision medicine strategy is required.

**Harnessing the complex relationship between mitosis and cell death**

Cell death and inflammation are ancient processes of fundamental biological importance in both normal physiology and cancer. The recent observation that cell death regulatory components have dual roles in cell death and inflammation suggests that these proteins function, not primarily to kill, but to coordinate tissue repair and remodelling. This perspective unifies cell death components as positive regulators of tissue repair that replaces malfunctioning or damaged tissues and enhances the resilience of epithelia to insult. Much remains to be learned about whether therapy induced cell death, even if decreasing tumour size, causes the production of signals that contribute to relapse or metastasis by stimulating the survival and expansion of small numbers of cells capable of seeding a new tumour. Pascal Meier will discuss the complex relationship between cell death and inflammation, and elaborate on its impact on tissue health and disease. In particular he will focus on unexpected new insights into how components of the cell death machinery intersect with the mitotic checkpoint to ensure accurate chromosome segregation and prevent the establishment of aberrant phenotypes that would otherwise lead to evolvability (chromosomal instability), favouring tumour evolution, heterogeneity, acquisition of drug resistance and heighten risk for tumour relapse.
Symposia abstracts (continued)

Where are the gains in technical radiotherapy?

11.00 – 11.05
Room 3B

**Introduction**
At the time of printing, these abstracts had not been received. Check the Conference App for further details.

Hosted by Jeanette Dickson, The Royal College of Radiologists

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11.05 – 11.30
Room 3B

**George Edelstyn lecture**
Protons

Steve Hahn, The University of Texas MD Anderson Cancer Center, USA

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11.30 – 11.55
Room 3B

**Advances in SABR**

Maria Hawkins, Gray Institute for Radiation Oncology and Biology, Oxford University, UK

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11.55 – 12.20
Room 3B

**Motion management in radiotherapy**

Marcel van Herk, The Christie Hospital, Manchester, UK

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12.20 – 12.30
Room 3B

**Discussion**

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Parallel sessions

**How we talk about cancer**

Hosted by Elena Semino, Lancaster University, UK

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<td>Introduction by the host</td>
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<td>16.40</td>
<td>The war on prevention: Bellicose cancer metaphors undermine prevention behaviours</td>
<td>David Hauser</td>
<td>University of Michigan, USA</td>
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<td>16.40</td>
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<td>The war on cancer: Reflections from a conscientious objector</td>
<td>Andrew Graystone</td>
<td>Media Futures, UK</td>
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<td>Proffered paper: Keeping the Customer Satisfied? #5 – Who’s Talking?</td>
<td>Richard Stephens</td>
<td>Chair, NCRI Consumer Forum</td>
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<td>17.30</td>
<td>’Searching for the new normal’: Exploring the role of language and</td>
<td>Lynda Appleton</td>
<td>Clatterbridge Cancer Centre, UK</td>
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<td>metaphor in becoming a cancer survivor</td>
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**Advances in cancer drug discovery and development: Small molecule versus antibody-based strategies**

Hosted by Michelle Garrett, University of Kent, UK

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<td>16.35</td>
<td>Title to be confirmed</td>
<td>Jessie English</td>
<td>IONC TIP, EMD Serono, Merck KGaA, USA</td>
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<td>17.00</td>
<td>Small molecule therapeutics in oncology: Challenges and</td>
<td>Olivia Rossanese</td>
<td>The Institute of Cancer Research, London, UK</td>
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<td>17.00</td>
<td>opportunities</td>
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<tr>
<td>17.25</td>
<td>Title to be confirmed</td>
<td>Danielle Carroll</td>
<td>MedImmune, Cambridge, UK</td>
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<td>17.50</td>
<td>Proffered paper: Enhancing responses to melanoma therapy with novel combinations of targeted therapy and immune checkpoint blockade</td>
<td>Robert Szczepaniak Sloane</td>
<td>The University of Texas MD Anderson Cancer Center, USA</td>
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Parallel sessions (continued)

18.05 – 18.20
Hall 1A
BACR Roger Griffin Prize for Cancer Drug Discovery
Engineering potency and selectivity of chemical probes for functional elucidation and target validation
Matthias Baud, University of Southampton, UK

18.20 – 18.30
Hall 1A
Discussion

Delivering patient-centred cancer care: Meeting the psychosocial needs of people living with and beyond cancer

Hosted by Nick Hulbert-Williams, University of Chester, UK

16.30 – 16.40
Room 3A
Introduction by the host

16.40 – 17.05
Room 3A
Screening for distress in cancer: Does it influence uptake of care?
Alex Mitchell, University of Leicester, UK

17.05 – 17.30
Room 3A
Enhancing patient-centred care: Assessment and management of patients’ unmet needs
Afaf Girgis, University of New South Wales, Australia

17.30 – 17.55
Room 3A
Proffered paper: REPORT-UK (Real-time Electronic Patient Outcome RePoRtIng of adverse events in UK cancer trials) – a feasibility pilot study in a UK oncology setting
Galina Velikova, St James’s University Hospital, Leeds, UK

17.55 – 18.20
Room 3A
Fear of cancer recurrence: Current state and future directions for research and clinical practice
Gozde Ozakinci, University of St Andrews, UK

18.20 – 18.30
Room 3A
Discussion

Epstein-Barr virus: Continuing insights into cancer and its treatment

Hosted by Neil Steven and Graham Taylor, University of Birmingham, UK

16.30 – 16.40
Room 12
Introduction by the hosts

16.40 – 17.10
Room 12
Future prospects for novel therapeutics for EBV-associated diseases
Lawrence Young, University of Warwick, UK
Human tumour virus infection and immune control in vivo
Christian Münz, University of Zurich, Switzerland

T-cell Therapy for EBV-positive Malignancies: Lessons learned from the clinic
Stephen Gottschalk, Baylor College of Medicine, Houston, USA

Discussion

Small cell lung cancer: Genomics, biology and progress with clinical management of this recalcitrant tumour
Hosted by Caroline Dive, Cancer Research UK Manchester Institute, UK

Introduction by the host

Title to be confirmed
Roman Thomas, University of Cologne, Germany

Pre-clinical mouse models of SCLC to identify and test novel therapies
Julien Sage, Stanford University, USA

Advances in the radiotherapy treatment of SCLC
Corinne Faivre-Finn, University of Manchester, UK

Discussion

Novel targeted therapies for cancer cachexia – a new era in cancer treatment
Hosted by Miriam Johnson, University of Hull, UK

Introduction by the host

Cancer cachexia: A personal perspective
Debbie Keatley, NCRI Consumer Forum

Classification, mechanisms and evidence for current treatments
Richard Skipworth, University of Edinburgh, UK

Clinical trials of pharmacological interventions for cancer cachexia
David Currow, Flinders University, Adelaide, Australia

Summary
### Improving outcomes with drug/radiotherapy combinations

Hosted by Geoffrey Higgins, University of Oxford, UK

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<td>16.40 – 17.10</td>
<td>Why don’t drug companies provide access to drugs for use in combination with radiotherapy?</td>
<td>Anthony Chalmers, Beatson West of Scotland Cancer Centre, Glasgow, UK</td>
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<tr>
<td>17.10 – 17.40</td>
<td>Immunotherapy and radiotherapy</td>
<td>Tim Illidge, Cancer Research UK Manchester Institute, UK</td>
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<td>17.40 – 18.10</td>
<td>Novel drug/radiotherapy combinations</td>
<td>Philippe Lambin, University of Maastricht and the MAASTRO Clinic, The Netherlands</td>
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<tr>
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Parallel session abstracts

How we talk about cancer

Introduction
This session is concerned with how cancer is talked about, and the problems, challenges and opportunities that arise in communication about cancer. Topics may include: language and cancer prevention; language and living with cancer and cancer treatment; language and cancer survivorship; communication between people with cancer and healthcare professionals; media reports about cancer; the discourse of cancer charities. The session is aimed at anyone who is interested in understanding and improving communication about cancer.

The war on prevention: Bellicose cancer metaphors undermine prevention behaviors
Cancer health information is dominated by enemy and war metaphors. However, these metaphors may influence understanding of, and responses to, cancer. Encountering a metaphorically-framed concept induces people to conceptualize the concept in terms of the metaphor. As such, framing cancer as an “enemy” leads people to map their knowledge of enemies (such as how to deal with them) onto how they think about cancer. Cancer prevention benefits from avoiding risk-increasing behaviors, yet self-limitation is not closely associated with fighting enemies. If so, the metaphor may hurt prevention intentions involving self-limitation.

In two studies, participants read messages with minute wording variations that established different metaphoric frames (randomly assigned). Results show that metaphorically framing cancer as an enemy lessens how often participants think of limiting cancer risk-increasing behaviors (study 1) and lessens how much participants intend to limit risk-increasing behaviors (study 2). Additionally, enemy framing does not increase intentions for more active behaviors like electing for cancer screening procedures. Overall, these results suggest that enemy metaphors in cancer information reduce some prevention intentions without increasing others, making their use potentially harmful for public health.

The war on cancer: Reflections from a conscientious objector
As a writer, my personal experience of cancer caused me to reflect on the language we use to talk about it. The vocabulary we choose (or are offered) to talk about illness expresses what we believe about our lives, and in particular our bodies. Metaphors from warfare serve to objectify cancer. But for those like me for whom it is a personal experience, they may create a paradigm of alienation and failure. So, between my frequent visits to the loo, I set out to explore and experiment with fresh imagery for life with cancer.

Proffered paper: Keeping the Customer Satisfied? #5 – Who’s Talking?
Findings On Research Conversations From The National (English) Cancer Patient Experience Survey 2015

Background
The annual Cancer Patient Experience Survey includes the question, ‘Since your diagnosis, has anyone discussed with you whether you would like to take part in cancer research?’ The new (2015) national Cancer Strategy, Achieving World Class Outcomes, highlighted the
importance of patient experience. The Cancer Dashboard (2016) has a key CPES-derived metric for patient experience. Analysis of the 2013 CPES data showed that research participation is associated with higher levels of patient satisfaction with their overall experience.

Method

All in-patient and day case cancer patients treated in the 148 acute and specialist Trusts in England between April and June 2015 were offered the opportunity to complete the 2015 NCPES questionnaire. Over 71,000 (66%) responded, with national results published in June 2016, Trust- and CCG-level reports in July 2016.

Results

28% of patients reported having had a discussion about research participation. This compares with 33% (2012), 32% (2013), and 31% (2014). Having a discussion about research participation continues to vary significantly by Trust and cancer type. By Trust the variation extends from 11.7% of patients reporting a discussion to 55.2% of patients. Past years were 14%-56% (2012), 11%-62% (2013), 10%-61% (2014) and 11%-55% (2015). By cancer type the variation extends from 13.3% of patients reporting a discussion to 34.8% of patients, consistent with previous years; 15%-39% (2012), 16%-38% (2013), 14%-37% (2014), 13%-35% (2015).

Conclusion

Improving the patient experience and encouraging participation in research are both national policy objectives. The continued fall in the proportion of patients reporting a research discussion is thus doubly disappointing. It may be explained by the changing ecology of cancer research, but the wide variations in performance by Trust suggest otherwise. Significant inequalities still exist for patients in gaining access to opportunities to participate in research and potentially to an improved patient experience.

References

2 http://www.ncpes.co.uk/index.php

‘Searching for the new normal’: Exploring the role of language and metaphor in becoming a cancer survivor

The language of cancer is known to influence the personal and social adjustment of the patient following the completion of a course of treatment. Language can aid understanding but can also give rise to false hopes and misunderstanding. This qualitative study aimed to investigate the impact of words and metaphors on the identity of a person living with cancer.

The study employed a focus group design, in which eighteen people, recruited through regional networks and support groups, participated. Data were analysed thematically and organised into descriptive categories concerned with the interpretation of common words and phrases in the cancer lexicon.
Themes identified were grounded in personal, relational and social identity: ‘managing identity and emotions’, ‘relationships’ and ‘public perceptions’, demonstrating positive and negative consequences for adjustment and subtle, but important differences in the way healthcare professionals and lay people use the language and metaphors of cancer.

The language health professionals’ use plays an important part in shaping peoples’ cancer experience and suggests a need for professionals to elaborate their broad understanding of communication skills and move toward a common language based on mutual understanding and meaningful partnership with the patient.

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**Advances in cancer drug discovery and development: Small molecule versus antibody-based strategies**

**Introduction**

The aim of this session is to give both clinicians and researchers (basic and translational) an update on recent advances in cancer drug discovery, from concept to clinic and to and discuss where the field is going. Participants will gain an up-to-date international perspective on small molecule and antibody-based cancer drug discovery and development as well as an understanding of the advantages/disadvantages of both approaches to cancer drug discovery and development.

**16.30 – 16.35**

Hall 1A

Michelle Garrett,
University of Kent, UK

**16.35 – 17.00**

Hall 1A

Jessie English,
IONC TIP, EMD Serono,
Merck KGaA, USA

**17.00 – 17.25**

Hall 1A

Olivia Rossanese,
The Institute of Cancer Research, London, UK

**17.25 – 17.50**

Hall 1A

Danielle Carroll,
MedImmune,
Cambridge, UK

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**Title to be confirmed**

At the time of printing, this abstract had not been not received. Check the Conference App for further details.

**Small molecule therapeutics in oncology: Challenges and opportunities**

At the time of printing, this abstract had not been not received. Check the Conference App for further details.

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For more information, visit conference.ncri.org.uk 103
**Proffered paper: Enhancing responses to melanoma therapy with novel combinations of targeted therapy and immune checkpoint blockade**

**Background**
Treatment with targeted therapy and immunotherapy have revolutionized melanoma treatment. However, only a subset of patients respond durably, and combination strategies are emerging as a clear choice. We now have strong data to support the use of combined targeted therapy and immunotherapy, and sought to investigate different combination regimens in a BRAF-mutant murine melanoma model to help rationally guide novel combination strategies.

**Method**
C57BL/6 mice were implanted with melanoma tumours (BRAFV600E/PTEN-/-) and were treated with immunotherapy combinations (α-CTLA-4, α-OX40, α-PD-1, α-CTLA-4/α-PD-1 and α-OX40/α-PD-1) with or without BRAF/MEK inhibitors (dabrafenib and trametinib – DT). Total treatment duration was limited to 19 days to test the hypothesis that a short burst of therapy could result in long-term control. Tumour growth and survival were monitored, and immune profiling and gene expression profiling were performed in tumours.

**Results**
In these studies, treatment with aPD-1 and aCTLA-4 monotherapy was relatively ineffective in controlling tumour growth, while aOX40 had a modest impact. Combination immunotherapy was more effective, with the best control observed in the α-OX40/α-PD-1 group (with 5/12 mice showing no evidence of disease). The addition of DT to immunotherapy improved tumour control in all groups. Immune profiling demonstrated increases in CD8 T cell number and function, as well as lower expression of TIM-3 and LAG-3 in all DT-treated groups. Gene expression corroborated these findings, with significant increases in immune-related genes in presence of DT, suggesting immune mechanisms to the enhanced responses.

**Conclusion**
We have shown that DT treatment can be used to effectively shape the tumour immune microenvironment and enhance responses to immunotherapy – even in the setting of a short burst of therapy. These studies provide rationale for clinical trials investigating these combination strategies in melanoma, and results may support further studies combining molecularly targeted therapy and immunotherapy for other cancer types.

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**BACR Roger Griffin Prize for Cancer Drug Discovery**

**Engineering potency and selectivity of chemical probes for functional elucidation and target validation**

Designing selective chemical probes is crucial for accurate functional elucidation and target validation in disease. However, this can become a daunting task when the target is part of a family of structurally related proteins. One example of such a family of proteins includes Bromo and Extra-Terminal (BET) proteins brd2, brd3, brd4 and brdt, which are important transcriptional co-regulators modulating gene expression in the nucleus. Key to their activity are small modular domains called bromodomains that recognise and interact with acetylated lysine residues from chromatin. The eight BET bromodomains (two per BET protein) have attracted particular attention lately for drug development. Disregulation of their transcriptional activity has been linked to a number of aggressive cancers that are usually associated with relatively poor prognosis, such as NUT midline carcinoma, multiple myeloma,
mixed-lineage leukemia and myeloid leukaemia. These findings have fuelled the interest of medicinal chemists for developing new routes towards synthetic and potent small molecules modulating the activity of BET bromodomains by disrupting their interaction with their chromatin substrates. This eventually resulted in the development of a range of small molecules showing efficacy against several cancers. A number of such compounds are currently evaluated in the clinic as anticancer agents. Despite their promising pharmacologies, these molecules bind to all BET bromodomains rather unspecifically in cells and all past attempts at modulating an individual BET bromodomain with a small molecule proved largely unsuccessful. This in turn hampered accurate functional elucidation of individual BET bromodomains, making it unclear which of these domains should be the ideal target of further medicinal chemistry efforts.

We recently developed a chemical genetics approach called “bump-and-hole”, aimed at delivering a new tool that would allow us to selectively modulate an individual BET bromodomain with a small molecule. This approach is based on the generation of orthogonal and high-affinity protein/ligand pairs and involves introducing a single point mutation (the “hole”) onto the BET bromodomain of interest together with making a synthetic modification (the “bump”) onto the parent BET bromodomain binder to complement a newly created protein subpocket. We discovered that compound “ET”, a derivative of I-BET762, was able to target engineered bromodomains bearing a leucine to alanine (L/A) mutation in their Z/A loop with very high potency (< 100nM) and selectivity (up to 500 fold) both in vitro and in cells. This orthogonal protein/ligand pair provided the first chemical tool that allows controlling a single BET bromodomain while leaving the remaining seven domains unaffected. Following-up on these results, we are currently using our approach to gain deeper understanding of the biological function of BET proteins, which we expect will further shed light on their potential as targets in oncology. We also anticipate that this approach will be of benefit to address a wide range of other protein targets where chemical probes development has been hampered by selectivity and potency issues.

Discussion

Delivering patient-centred cancer care: Meeting the psychosocial needs of people living with and beyond cancer

Introduction

Cancer can be psychologically distressing for both patients, and their families. This has profound effects on quality of life, emotional wellbeing, and engagement with medical care. It is only by addressing psychological aspects of cancer that we provide adequate patient-centred care. This session covers the latest evidence and application in distress screening in clinical settings, identifying and intervening to improve unmet psychosocial care needs, and anxiety and fear of recurrence in cancer survivorship. This session is aimed at all those providing cancer care and support, including those working in primary care and palliative settings.
Screening for distress in cancer: Does it influence uptake of care?

Screening for distress is recommended by several organisations following cancer but the evidence base is unclear. We reviewed 33 publications involving a total of about 17,000 cancer patients. Screening with follow-up enhanced psychosocial care by 9.1% (risk difference). 8 implementation studies measured receipt of psychosocial referral before and after distress screening and referrals increased by 11.6%. Six implementation studies examined the effects of screening for quality of life on communication. Screening increased clinician-patient communication of emotional issues by only 5.8%. Unfortunately even when offered help only 36.5% of distressed cancer patients were immediately willing to accept professional psychosocial help although 51.6% would consider it now or in the future. Screening can influence uptake of care but the effect is modest and barriers must be addressed for screening to be a success.

Enhancing patient-centred care: Assessment and management of patients’ unmet needs

Patient reported outcomes are increasingly important to providing high quality and targeted patient-centred care. Systematic assessment and management of cancer patients’ unmet needs is an essential component of health care for people with cancer. Over the past two decades or so, psychometrically sound and clinically useful needs assessment tools have been developed and tested. These tools have informed both the prevalence as well as the predictors of unmet need in cancer patient populations as well as their caregivers. Information about the prevalence and types of unmet needs being reported can inform service planning and redesign, as well as patient-centred individual care. This presentation will provide an overview of the prevalence of unmet needs in various cancer patient and caregiver populations and provide examples of how the assessment of unmet needs informs patient care and self-management.

Proffered paper: REPORT-UK (Real-time Electronic Patient Outcome ReporTing of adverse events in UK cancer trials) – a feasibility pilot study in a UK oncology setting

Background

Adverse events (AEs) reporting is essential in clinical trials. The current system for reporting (Common Toxicity Criteria and Adverse Events, CTCAE) relies on clinicians’ interpretation of symptoms. The value of patient self-reports of AEs and Patient Reported Outcome Measures (PROMs) is recognised but robust data collection methods are needed. REPORT-UK was a proof-of-principle study to develop and evaluate an electronic (internet/telephone) system for self-reporting AEs and PROMs during trials. A feasibility pilot study to assess compliance with the systems in a general oncology setting ran between August 2014 and October 2015.

Method

249 varied diagnosis cancer patients undergoing treatment (chemotherapy/targeted agents/hormone therapy/radiotherapy/surgery, and an ECOG group with performance status ≥2) were recruited. For 12 weeks patients were reminded (text/email) to complete weekly AEs (NCI PRO-CTCAE) and monthly PROMs questionnaires (EORTC QLQ-C30) on their preferred system. Acceptability was measured by recruitment rates, attrition, compliance, and patient and staff feedback at end-of-study (EOS).
Results
Overall, the consent rate was 48%. System preference was 82% internet/17% IVR. Only 13 participants withdrew and 6 died whilst on study. 192 returned EOS questionnaires. Overall patient compliance was good for both weekly AE and monthly PROMs reporting, but differed between treatment groups, and dropped over time, but comparable to clinical trials. Both systems were perceived as easy-to-use. Time to complete was perceived by patients to be acceptable, although actual times show the internet is quicker (median time 9 minutes vs. 21.5 minutes). Baseline comparisons between patient vs. clinician-reporting of some AEs differed substantially.

Conclusion
The study demonstrates a user-friendly electronic data collection system, which provides information on patient compliance in a general oncology setting but we recognise this is different to a real trial setting. The system could be implemented in practice in clinical trials alongside traditional approaches to improve data quality and safety.

Fear of cancer recurrence: Current state and future directions for research and clinical practice
Fear of cancer recurrence, defined as “Fear, worry, or concern about cancer returning or progressing” is one of the most frequently experienced unmet need by survivors. It has an impact on quality of life of those living with and beyond cancer and also on the carers. These fears do not necessarily dissipate over time and tend to be triggered by various mechanisms such as follow-up appointments and news on cancer in the media. In this session, I will summarise the latest research evidence on the prevalence and impact of fear of cancer recurrence on those living with and beyond cancer and their carers. I will also talk about the latest psychological support programmes that are being developed and tested and current gaps in our knowledge and what future directions we need to take to assist those who may be struggling with these fears.

Discussion

Epstein-Barr virus: Continuing insights into cancer and its treatment

Introduction
Epstein Barr virus (EBV) co-evolved with humans. We mostly catch it in childhood and it persists lifelong, apparently a harmless passenger. Yet EBV is found in some lymphomas, stomach cancer and nasopharyngeal carcinoma. EBV-positive cancers are important worldwide. The virus offers a window into the complex changes in cell regulation and signalling, inflammation and immunity that play key roles in driving cancers. We aim to inform a wide audience about our species’ commonest cancer virus. We will discuss how the virus promotes cancers and explore how pre-clinical models and clinical trials have driven our understanding of immune therapy for malignancy.
Future prospects for novel therapeutics for EBV-associated diseases

Given the significant burden of EBV-associated tumours worldwide, an important priority is to design novel therapeutic approaches that specifically target viral proteins or otherwise exploit the presence of the virus in malignant cells. This is particularly relevant in the context of nasopharyngeal carcinoma (NPC) where 30% of patients with locoregionally advanced disease will subsequently succumb to distant metastases. The development of novel therapeutic approaches using targeted drugs, gene therapy, or immunotherapy is essential to effectively target the clinically challenging aspects of EBV-associated malignancy. Alongside these therapies, the advent of personalised medicine raises the possibility of using molecular classification to sub-divide virus-associated tumours thereby improving patient management and outcomes. This talk will review the current status of novel therapeutics and consider how our growing understanding of EBV-associated oncogenesis is continuing to provide paradigms for the development of targeted cancer therapies relevant to the more general management of malignant disease.

Human tumour virus infection and immune control in vivo

Primary infection with the human oncogenic Epstein Barr virus (EBV) can result in infectious mononucleosis (IM), a self-limiting disease caused by massive lymphocyte expansion, which predisposes for the development of distinct EBV-associated lymphomas. It remains unclear why some individuals experience this symptomatic primary EBV infection, while the majority acquires the virus asymptomatically. Using a mouse model with reconstituted human immune system components, we could show that depletion of human natural killer (NK) cells enhances IM symptoms and promotes EBV-associated tumourigenesis, mainly due to loss of immune control over lytic EBV infection.

These data suggest that failure of innate immune control by human NK cells augments symptomatic lytic EBV infection, which drives lymphocyte expansion and predisposes for EBV-associated malignancies. These human NK cells carry inhibitory killer cell immunoglobulin-like receptors (KIRs), recognizing distinct HLA molecules. NK cells with KIRs for self-HLA molecules acquire superior cytotoxicity against HLA negative tumour cells during education for improved missing-self recognition. We could show that co-reconstitution of two KIR-ligand mismatched human immune system compartments in mice does not alter the frequency of KIR expressing NK cells, but their education. NK cell education is diminished for KIRs, whose cognate HLA molecules are lacking on leucocytes that reconstitute in parallel in the same mice. This change in NK cell education in mixed human donor reconstituted mice is functionally relevant, because it improves NK cell mediated immune control of Epstein Barr virus infection. Thus, leucocytes lacking cognate HLA ligands can disarm KIR positive NK cells for improved immune control of a human gamma-herpesvirus.

T-cell Therapy for EBV-positive Malignancies: Lessons learned from the clinic

Epstein-Barr virus (EBV) is associated with a range of malignancies. All of these are associated with the latent life cycles of EBV, but the pattern of latency-associated viral antigens expressed in tumour cells depends on the type of tumour. EBV-specific T cells (EBVSTs) have been explored as prophylaxis and therapy for EBV-associated malignancies for more than two decades. EBVSTs have been most successful as prophylaxis and therapy for post-transplant lymphoproliferative disease (PTLD), which expresses the full array of latent EBV antigens (type 3 latency), in hematopoietic stem cell transplant recipients. While less effective, clinical studies have also demonstrated their therapeutic potential for PTLD...
post solid organ transplant, and for EBV-associated malignancies such as Hodgkin’s Lymphoma, Non-Hodgkin’s Lymphoma, and nasopharyngeal carcinoma that express a limited array of latent EBV antigens (type 2 latency). Several approaches are actively being pursued to improve the antitumour activity of EBVSTs including activation and expansion of T-cells specific for the EBV antigens expressed in type 2 latency, genetic approaches to render EBVSTs resistant to the immunosuppressive tumour environment and combination approaches with other immune-modulating modalities. In my talk I will review the current status and future directions of EBVST-Therapy for EBV-positive malignancies.

Discussion

Small cell lung cancer: Genomics, biology and progress with clinical management of this recalcitrant tumour

16.30 – 16.40
Hall 1B

Caroline Dive,
Cancer Research UK
Manchester Institute,
UK

Introduction

Small Cell Lung Cancer (SCLC) usually presents late and it is highly metastatic. SCLC prognosis is dismal (<5% survival for 5 years) and the paucity of tumour biopsies for translational research has been a constant obstacle to progress. SCLC is treated with platinum based chemotherapy with or without radiotherapy. Most patients respond initially, but relapse is swift with minimal treatment options thereafter. This session follows the plenary lecture on new treatment approaches for SCLC (Charles Rudin) that gives hope that targeted and immunotherapies may extend the range of SCLC treatments and extend progression free survival. Here, we will showcase the significant progress made recently in understanding the biology of SCLC, with a comprehensive look at the genomic landscape (Roman Thomas). The application of patient relevant genetically engineered mouse models to explore the multiple levels of SCLC heterogeneity, drug sensitivity and resistance pathways and function-test new hypotheses will be described by (Julien Sage). The important role of thoracic and cranial radiotherapy and recent advances will be described for limited and extensive stage SCLC patients (Corinne Faivre-Finn). SCLC remains a recalcitrant tumour, but the current concerted efforts worldwide aim to improve outcomes after 30 years of little progress.

16.40 – 17.10
Hall 1B

Roman Thomas,
University of Cologne,
Germany

Title to be confirmed

At the time of printing, this abstract had not been not received. Check the Conference App for further details.
Pre-clinical mouse models of SCLC to identify and test novel therapies

Small cell lung cancer (SCLC) is a neuroendocrine subtype of lung cancer characterised by a fast growth rate, extensive dissemination, and rapid resistance to chemotherapy. Survival rates are dismal and have not significantly improved in the past few decades. Sequencing the genomes of over 100 human SCLC demonstrates universal inactivation of p53 and RB and identified inactivating recurrent mutations in NOTCH family genes (George, Lim, et al., Nature, 2015). Accordingly, we found that activation of Notch signaling in a pre-clinical SCLC mouse model dramatically reduces the number of tumours and extends the survival of the mutant mice. Thus, Notch plays a key tumour suppressive role in SCLC and strategies to re-activate Notch in SCLC tumours may be beneficial to patients. We will present new studies focusing on mouse models on the possible role of Notch-negative and Notch-positive cells in SCLC tumours during cancer progression and in response to chemotherapy. At the histological level, SCLC tumour cells are often viewed as homogeneous. These studies and previous studies (e.g. Calbo et al., Cancer Cell, 2011 – Berns lab) identify several levels of intra-tumour heterogeneity in SCLC, which may contribute significantly to SCLC aggressive nature and resistance to therapy.

Advances in the radiotherapy treatment of SCLC

Major advances in SCLC have come from improvements in RT techniques. The role of thoracic radiotherapy is well established in the management of stage I-III SCLC. There is robust evidence in favour of early concurrent chemoradiotherapy, and a standard of care for patients with a good performance status is twice-daily thoracic radiotherapy with concurrent cisplatin and etoposide. The CONVERT study (twice-daily versus once daily radiotherapy given concurrently with chemotherapy in stage I-III SCLC) was presented at ASCO 2016. It showed that once-daily radiotherapy did not result in a superior survival or worse toxicity than twice-daily radiotherapy, supporting the use of either regimen for standard of care treatment.

The survival for both regimens was higher than previously reported and using modern RT techniques radiation toxicities were lower than expected. In the extensive-stage setting the CREST trial as shown that the addition on thoracic RT to chemotherapy and PCI leads to a significant reduction in intrathoracic recurrence and a significant improvement in overall survival at 2 years. PCI reduces the incidence of brain metastases and improves survival in all stages of SCLC It is crucial that patients with SCLC are given the opportunity to participate in clinical research in order to continue to improve the survival of this disease.

Discussion

Novel targeted therapies for cancer cachexia – a new era in cancer treatment

Introduction

To date the only way to reverse the metabolic syndrome of cancer-related anorexia-cachexia has been to treat the cancer successfully with chemotherapy. For many patients that is not an option, leaving this debilitating aspect of the disease unalleviated. Attempts with agents such as steroids and progestogens have been met with limited net benefit, if any at all. The
advent of novel agents, for the first time, open a vista of well tolerated treatments which have the potential to improve quality of life, and increase survival.

In this session, the results of 5 phase III RCTs will be presented in the context of current understanding of the mechanisms and classification of cachexia, and evidence for treatments in common use.

16.35 – 16.50
Hall 1C
Debbie Keatley,
NCRI Consumer Forum

Cancer cachexia: A personal perspective

Classification, mechanisms and evidence for current treatments

The involuntary loss of weight, accompanied by loss of appetite, in people with cancer has been described since ancient times. However, delineation of the complex underlying metabolic changes responsible for this clinical situation and the definition of the syndrome with an understanding of early and refractory cachexia is relatively recent. Thus the approach to management has focussed on treating the cancer rather than interventions to target the abnormal metabolism. In this session, a consensus definition of cachexia and a review of the underlying pathophysiology will be presented in order to set the context for discussing potential specific therapies for this neglected but distressing area of cancer experience.

16.50 – 17.25
Hall 1C
Richard Skipworth,
University of Edinburgh, UK

Clinical trials of pharmacological interventions for cancer cachexia

At present, there is no consensus on a successful treatment strategy for cachexia. Many previous intervention trials have failed due to either the inclusion of patients with refractory cachexia (too advanced for successful intervention), or the lack of clinically relevant outcome measures. Recently, there have been four Phase III RCTs of anti-cachexia therapies that have failed to demonstrate efficacy as defined by the Food and Drug Administration (FDA) (defined as improvement in both body composition and physical function). This presentation will examine the findings from the following trials:

a) A randomised, double blind control trial of megestrol acetate, dexamethasone and placebo in the management of anorexia in people with cancer. Presentation of data from the recently completed phase III RCT of megestrol acetate vs dexamethasone vs placebo for anorexia.

b) The POWER trials. In these trials, enobosarm, a non-steroidal, oral, selective androgen receptor modulator [SARM] by GTX Inc, was associated with improvements in lean body mass (LBM) in patients with non-small cell lung cancer (NSCLC), but was not associated with any significant improvements in the co-primary endpoint of stair-climb power.

c) The ROMANA 1 and 2 trials. In these trials anamorelin, a ghrelin- receptor agonist by Helsinn, was associated with improvements in LBM, but not handgrip strength, in NSCLC patients.

17.25 – 18.10
Hall 1C
David Currow,
Flinders University, Adelaide, Australia

Summary

18.10 – 18.15
Hall 1C

Join us next year: 5-8 November 2017
conference.ncri.org.uk
Improving outcomes with drug/radiotherapy combinations

16.30 – 16.40
Room 3B

Geoffrey Higgins,
University of Oxford, UK

Introduction

Why don’t drug companies provide access to drugs for use in combination with radiotherapy?
Clinical evaluation and adoption of novel radiotherapy-drug combinations has failed to keep pace with the exciting scientific discoveries in this area, and is lagging behind the uptake of drug-drug combinations. One reason for this has been the reluctance of pharmaceutical companies to make novel compounds available for evaluation in combination with radiotherapy. This presentation will consider the reasons for this situation and some promising solutions.

16.40 – 17.10
Room 3B

Anthony Chalmers,
Beatson West of Scotland Cancer Centre, Glasgow, UK

Immunotherapy and radiotherapy
The translation of our increasing scientific knowledge about the key receptors involved in tumour immunoregulation, from proof of principle with anti-CTLA-4, to effective clinical anti-cancer therapeutics in a large range of cancers eg anti-PD1/ PD-L1 inhibitors has led to huge optimism that immunotherapy will play an increasingly important part in cancer therapy. In contrast radiation treatment (RT) has been established as highly effective cancer therapy for decades albeit the focus in radiobiology has largely been on the direct tumour cell kill. However emerging evidence suggests that RT also has important effects on the tumour microenvironment and can generate local anti-tumour immunity. The generation of systemic immunity and tumour responses outside of the radiation treatment area that leads to the so called “abscopal effect” are however extremely rare and this is thought to be secondary to tumour suppressive adaptive resistance.

The opportunity of combining RT and immunomodulatory agents however offer great potential to reverse the tumour induced resistance to improve outcomes further. In order to increase the number of responses in the majority of patients across many different tumour types, further investigation is required to fully understand the potential underlying mechanisms of resistance. In addition well-designed clinical trials with the appropriate translational research we also be required. In this presentation the interplay of RT with the tumour microenvironment and novel opportunities to overcome adaptive resistance will be discussed alongside clinical translation.

17.10 – 17.40
Room 3B

Tim Illidge,
Cancer Research UK Manchester Institute, UK

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Novel drug/radiotherapy combinations

At the time of printing, this abstract had not been not received. Check the Conference App for further details.

Philippe Lambin,
University of Maastricht and the MAASTRO Clinic, The Netherlands

Discussion
The Royal College of Radiologists proffered paper session

Proffered paper session
Chaired by Jeanette Dickson, The Royal College of Radiologists

13.00 – 13.11
Room 3B
A hypoxia transcriptomic signature predicting benefit from hypoxia-modifying treatment for high risk bladder cancer patients
Lingjian Yang, University of Manchester, UK

13.11 – 13.22
Room 3B
Dosimetric analysis of VMAT for locally advanced pancreatic cancer and a novel duodenal-PTV overlap parameter as a predictor of feasibility of dose escalation
Paul Junni, Leeds Teaching Hospitals Trust, UK

13.22 – 13.33
Room 3B
A higher whole pelvic integral dose is associated with worsening fatigue and functional outcome in prostate cancer patients treated with intensity modulated radiotherapy
Nuradh Joseph, General Hospital Polonnaruwa, Ministry of Health, Sri Lanka

13.33 – 13.44
Room 3B
A novel CBCT-based method for derivation of CTV-PTV margins for prostate and pelvic nodal irradiation
Ciara Lyons, Centre for Cancer Research and Cell Biology, Queen’s University Belfast, UK

13.44 – 13.55
Room 3B
Non surgical treatment of operable rectal cancer: Reducing harm from the standard of care in elderly patients
Arthur Sun Myint, The Clatterbridge Cancer Centre, UK

13.55 – 14.00
Room 3B
Award of prizes
A hypoxia transcriptomic signature predicting benefit from hypoxia-modifying treatment for high risk bladder cancer patients

**Background**
Hypoxia modification improves overall survival (OS) in muscle invasive bladder cancer patients who undergo radical radiotherapy. There is evidence that hypoxic tumours benefit most from hypoxia modification. The study aimed to identify or derive a hypoxia gene signature that predicts benefit from hypoxia-modifying treatment in bladder cancer.

**Method**
Bladder cancer transcriptomic data were available from public datasets and generated for 143 tumour samples from the BCON phase III trial of radiotherapy (RT) alone or with carbogen and nicotinamide (CON) using Affymetrix Human 1.0 Exon ST arrays. Published hypoxia signatures were tested. A novel signature was then derived by identifying candidate hypoxia genes from the literature and evaluating their bladder cancer specificity in the publically available datasets. A gene co-expression network was built and hub genes identified to generate a signature.

**Results**
None of the published hypoxia signatures were prognostic in public datasets or predicted benefit from hypoxia modification in BCON patients. A novel 17-gene signature was derived and cross validated by showing its prognostic significance in a surgical cohort. The signature was then independently validated in BCON patients. Patients categorised as high- versus low-hypoxia by the signature had a poor overall survival following radiotherapy alone (HR 2.80, 95% CI 1.48-5.32, P=0.0016). The signature also predicted benefit from CON with high-hypoxia patients receiving CON having a better overall survival than those receiving radiotherapy alone (HR 0.44, 95% CI 0.24-0.82, P=0.01). Prognostic and predictive significance remained after adjusting for clinicopathological variables (including gender, necrosis, age, stage and carcinoma in situ).

**Conclusion**
A 17-gene hypoxia signature has strong and independent prognostic and predictive value and has potential for individualising the treatment of patients with muscle invasive bladder cancer.

Dosimetric analysis of VMAT for locally advanced pancreatic cancer and a novel duodenal-PTV overlap parameter as a predictor of feasibility of dose escalation

**Background**
This study evaluates the use of conventional and dose escalated volumetric-modulated arc therapy (VMAT) compared with 3D-conformal radiotherapy for locally advanced pancreas cancer, and investigates whether a novel parameter of the overlap volume of the PTV with the duodenum (duoOLV) or stomach (stoOLV) can be used to predict in whom dose escalation is feasible.
Method
21 consecutive patients who had undergone 3D-conformal radiotherapy 54Gy in 30 fractions for locally advanced pancreatic cancer were replanned with VMAT to doses of 54Gy in 30 fractions and 59.4Gy in 33 fractions. Dosimetry to target volumes and organs at risk (OAR) along with irradiated volumes were compared.

Results
Compared with 3D-conformal radiotherapy, VMAT54 provided a significant reduction in dose to stomach, duodenum, small bowel and kidneys. VMAT59 significantly increased mean GTV and PTV doses with no loss of coverage of the 95% isodose from the 54Gy plans. The duodenal constraint of V55<1cm³ was exceeded in 7, 0 and 0 of 3D-conformal, VMAT54 and VMAT59.4 plans respectively. The stomach constraint of V50<16cm³ was exceeded in 7, 6, and 2 of 21 patients with 3D-conformal, VMAT54 and VMAT 59.4 plans respectively. There was no significant difference in irradiated volume comparing VMAT54 and VMAT59.4 plans. For VMAT59.4 mean PTV and GTV dose and coverage of GTV by 95% isodose showed a significant correlation with duoOLV (p<0.001); a duoOLV >21cm³ predicted a failure to achieve 95% prescription dose coverage (V95%Px) for the GTV of >90%. No correlation was found with stoOLV and target volume coverage for VMAT59.4.

Conclusion
Compared with 3D-conformal radiotherapy, VMAT without dose escalation provides significant OAR sparing. Dose escalation to 59.4Gy with VMAT whilst respecting duodenal and stomach constraints is feasible in a subset of patients. A duoOLV of >21cm³ predicted a failure to achieve dose escalation whilst maintaining duodenal doses within tolerance.

A higher whole pelvic integral dose is associated with worsening fatigue and functional outcome in prostate cancer patients treated with intensity modulated radiotherapy

Background
Although intensity modulated radiotherapy (IMRT) permits the delivery of a highly conformal dose to target volumes while sparing dose to identified organs at risk, it results in a higher body integral dose due to irradiation of a larger volume of tissue at lower doses. We hypothesized that a higher integral pelvic dose is associated with worsening fatigue and an adverse functional outcome in patients with localised prostate cancer treated with intensity modulated external beam radiotherapy.

Method
142 patients with localised adenocarcinoma of prostate treated with intensity modulated external beam radiotherapy were included in this analysis. The whole pelvic integral dose was calculated as the product of mean body dose and body volume. To account for differences in fractionation weekly integral dose was used in the analysis. The fatigue, physical functioning and role functioning domains of the EORTC QLQ-C30 questionnaire prior to radiotherapy and upon completion of radiotherapy were assessed. The outcome measure was defined as worsening in any of these three domains. The association with whole pelvic integral dose was analysed as a continuous variable as well as a binary variable dichotomized at median.
Results
The median weekly integral dose was 22.2 litre-Gy (range 8.9 – 48.9). In the whole population 68/142 (48%) had worsening of fatigue, physical or role functioning. A higher whole pelvic integral dose was significantly associated with worsening fatigue when analysed in both the binary variable (p=0.000005) and continuous variable (p=0.000001, Area Under Curve = 0.7322) models.

Conclusion
To our knowledge this is the first study to demonstrate a robust association between whole pelvic integral dose and worsening fatigue and functional outcome. These results need validation in a larger cohort and the relationship between integral dose and acute toxicity merits further investigation.

A novel CBCT-based method for derivation of CTV-PTV margins for prostate and pelvic nodal irradiation

Background
Traditional models of margin derivation for conventional radiotherapy are not applicable in the case of stereotactic ablative radiotherapy (SABR) and/or multiple targets. This study aimed to derive CTV-PTV margins using anatomical information from cone beam CTs (CBCTs) for use in prostate and pelvic nodal (PPN) external beam radiotherapy.

Method
Five CBCTs from 20 patients were selected. Eclipse (v13.5) was used to contour PPN volumes on all CBCTs and to rigidly register the images to the original planning CT. Two different image-registration protocols were investigated (bone/prostate). All contours were transferred to a single structure set; Boolean logic tools were used to create two composite volumes based on each registration method.

Each structure was compared to the original CTV with an isotropic margin applied incrementally to generate PTVs. The percentage overlap of each PTV with the composite structures was used to quantify agreement. A two-sided Wilcoxon signed-rank test was used to evaluate the significance of differences between the paired distributions of percentage overlap values for each match protocol.

Results
Table 1 summarises results obtained for the sample studied, including an estimate of the margins required to achieve 95% overlap with the composite structures for 90% of patients (normal distribution assumed).

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Match</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Average</td>
<td>Bone</td>
<td>Prostate</td>
</tr>
<tr>
<td>pCT CTV Volume (cm³)</td>
<td>360.2</td>
<td>55.2</td>
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<tr>
<td>Composite Volume</td>
<td>1.22</td>
<td>1.33</td>
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<tr>
<td>pCT CTV Volume</td>
<td>0.16</td>
<td>0.22</td>
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<tr>
<td>Margin required to achieve 95% overlap (mm)</td>
<td>3.93</td>
<td>5.55</td>
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<tr>
<td>Margin required to achieve 95% overlap in 90% of population</td>
<td>5.9</td>
<td>7.6</td>
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</tr>
</tbody>
</table>
The Royal College of Radiologists proffered paper session abstracts (continued)

Conclusion
CTV-PTV margins of 5.9 or 7.6mm were calculated for the pelvic ENI volume when matching to bone or prostate respectively. This novel approach is based solely on anatomical information and does not consider dose coverage or delineation error. As five CBCT images per patient were analysed, the results are of particular relevance for SABR.

Non surgical treatment of operable rectal cancer: Reducing harm from the standard of care in elderly patients

Background
The standard of care for early rectal cancer is surgery. However, mortality and morbidity is high following surgery in elderly patients. UK population is ageing and the majority with rectal cancer are over 70 years. The surgical mortality (12-25%) increases with age. We present our single institute data using non-surgical approach for elderly to reduce surgical mortality & morbidity.

Method
Our study reviewed the outcomes between 2003 to 2012. Mean age 74 years (range 32-94). There were 134(67%) males. Histology confirmed in all patients. Stages were T1 21(10.5%); T2 89(44.5%); T3 87(43.5%); T4 3 (1.5%). All patients had contact X-ray brachytherapy (CXB)[Papillon] 90-110 Gy/3-4 fractions over 4-6 weeks. This was followed by EBRT in 184 (92%). Watch and wait policy adopted in all patients who achieved complete clinical response (cCR).

Results
Initial complete clinical response was achieved in 136(68%). Residual abnormality seen in 64(32%). Immediate salvage surgery was carried out in 38(68%) who are fit. Those with cCR 116(85%) maintained complete response. At median follow up of 2.49 years, 16(11.7%) developed local relapse after cCR. Ten patients with loco regional relapse only who are fit had delayed surgery. Distant relapse developed in 17(8.5%). Overall survival (88%) and disease free survival (80%) were better for responders. There were no deaths related to CXB. The main toxicity was bleeding in 30% of cases. At the end of treatment 160 patients (80%) are disease free with good quality of life.

Conclusion
Elderly patients with early rectal cancer not suitable for surgery should be offer CXB (Papillon) to avoid high surgical mortality in elderly. OPERA which is a randomised trial is due to open in the UK to evaluate CXB efficacy. NICE has recommended CXB as safe with acceptable toxicity for patients not suitable for surgery. Data base is set up at Guildford for a prospective audit.

13.44 – 13.55 Room 3B
Arthur Sun Myint, The Clatterbridge Cancer Centre, UK

13.55 – 14.00 Room 3B

Award of prizes
Proffered paper session 2

Chaired by Caroline Dive, Cancer Research UK Manchester Institute, UK

**11.00 – 11.15**
**Hall 1C**
Immune-derived PD-L1 gene expression defines a subgroup of stage II/III colorectal cancer patients with favorable prognosis that may be harmed by adjuvant chemotherapy
*Philip Dunne*, Centre for Cancer Research and Cell Biology, Queen’s University Belfast, UK

**11.15 – 11.30**
**Hall 1C**
Tracing the origin of disseminated tumour cells in breast cancer using single-cell sequencing
*Jonas Demeulemeester*, The Francis Crick Institute, London, UK

**11.30 – 11.45**
**Hall 1C**
Ten year trends of participation of teenagers and young adults (TYA) in selected NIHR National Cancer Research Network trials
*Lorna Fern*, National Cancer Research Institute, UK

**11.45 – 12.00**
**Hall 1C**
Patients and families urge palliative care to prioritise the financial cost of caregiving; reporting from the Palliative and End of life care Priority Setting Partnership (PeolcPSP); A national survey
*Despina Anagnostou*, Cardiff University, UK

**12.00 – 12.15**
**Hall 1C**
ACP McElwain Prize winner
Clonal neoantigens elicit T cell immunoreactivity and sensitivity to immune checkpoint blockade
*Andrew Furness*, UCL Cancer Institute, London, UK

**12.15 – 12.30**
**Hall 1C**
BACR/AstraZeneca Young Scientist Frank Rose Award winner
Cancer specific non-essential amino acid metabolism – a role for targeted dietary intervention in cancer therapy?
*Oliver Maddocks*, University of Glasgow, UK

Join us next year: 5-8 November 2017  conference.ncri.org.uk  119
Immune-derived PD-L1 gene expression defines a subgroup of stage II/III colorectal cancer patients with favorable prognosis that may be harmed by adjuvant chemotherapy

**Background**
A recent phase 2 study of metastatic colorectal carcinoma (CRC) patients showed that mismatch repair gene status was predictive of clinical response to PD-1-targeting immune checkpoint blockade. Further examination revealed strong correlation between PD-L1 protein expression and microsatellite instability (MSI) in stage IV CRC, suggesting that the amount of PD-L1 protein expression could identify late stage patients who may benefit from immunotherapy.

**Method**
To assess whether the clinical associations between PD-L1 gene expression and MSI identified in metastatic CRC are also present in stage II/III CRC, we used in silico analysis methods in a number of clinical datasets to elucidate both the clinical importance of PD-L1 gene expression in stage II/III CRC and the precise cell types expressing the PD-L1 gene.

**Results**
We found a significant association of PD-L1 gene expression with MSI in early stage CRC (P < 0.001) and show that unlike in non-CRC tumours, PD-L1 is derived predominantly from the immune infiltrate. We demonstrate that PD-L1 gene expression has positive prognostic value in the adjuvant disease setting (PD-L1-low v PD-L1-high HR = 9.09; CI, 2.11-39.10). PD-L1 gene expression had predictive value, as patients with high PD-L1 expression appear to be harmed by standard-of-care treatment (HR = 4.95; CI, 1.10-22.35).

**Conclusion**
Building on the promising results from the metastatic CRC PD-1-targeting trial, we provide compelling evidence that PD-L1-high/MSI/immune-high stage II/III CRC patients should not receive standard chemotherapy. This conclusion supports the rationale to clinically evaluate this patient subgroup for PD-1 blockade treatment.

Tracing the origin of disseminated tumour cells in breast cancer using single-cell sequencing

**Background**
Single-cell micro-metastases of solid tumours often occur in bone marrow. These disseminated tumour cells (DTCs) may resist therapy and lay dormant or progress to cause overt bone and visceral metastases. Unfortunately, the molecular nature of DTCs remains elusive, as well as when and from where in the tumour they originate. Here, we apply single-cell sequencing and subclonal reconstruction to identify and trace the origin of DTCs in breast cancer.

**Method**
We sequenced the genomes of 63 single cells isolated by micromanipulation from the bone marrow of six patients using established immunocytochemical markers and morphologic characteristics for epithelial tumour cells. We compared the cells’ DNA copy number aberration (CNA) landscapes with those of the primary tumours and lymph node metastasis, and genotyped somatic mutations called on bulk tumour exomes in the single-cell
sequences. Evolutionary reconstruction analysis of bulk tumour and DTC genomes enabled ordering of CNA events in molecular pseudo-time.

**Results**

CNA landscape analysis revealed that almost half of the cells classified as tumour cells are indeed DTCs disseminating from the observed tumour. The remaining cells represented non-aberrant 'normal' cells and 'aberrant cells of unknown origin' that have CNA profiles discordant from the tumour. Probing somatic mutations confirmed that these cells did not derive from the same lineages as the observed breast cancers. Intriguingly, their prevalence tends to increase with patient age. Evolutionary reconstruction pinpointed the origin of the DTCs to either the main tumour clone, primary tumour subclones, or subclones in an axillary lymph node metastasis.

**Conclusion**

Single-cell sequencing of bone-marrow epithelial-like cells, in parallel with intratumour genetic heterogeneity profiling from bulk DNA, is a powerful approach to identify and study DTCs, yielding insight into metastatic processes. Metastatic potential is acquired relatively late during breast cancer evolution. A heterogeneous population of CNA-positive cells of unknown origin is prominent in bone marrow.

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**Ten year trends of participation of teenagers and young adults (TYA) in selected NIHR National Cancer Research Network trials**

**Background**

We previously reported improvements in recruitment to selected cancer clinical trials for teenagers and young adults (15-24 yrs) during 2005-10. This was related to the ‘5As’ which describes a strategy to maximise opportunities for research participation.

- **Appropriate:** eligibility criteria is permissive of TYA inclusion
- **Availability:** Trials available for rare cancers affecting young people
- **Access:** Study is open where young people are being treated
- **Acceptability:** Trial question and design is acceptable to both professionals and TYA
- **Awareness:** Professional and patient awareness of trials and the importance of offering entry.

We repeated our analysis to determine if accrual rates (AR) for TYA had continued to improve since the last reporting period, 2005-10.

**Method**

We analysed AR by age to NIHR CRN interventional Phase I/II/III trials recruiting newly diagnosed patients to leukaemia; lymphoma; sarcoma; male germ cell; brain and central nervous system. AR were expressed as the ratio of patients entering trials during April 1st 2005-March 31st 2015 and compared to relevant incidence cases for 2005-14.

**Results**

Preliminary analysis demonstrates the upward trend in AR for 0-24 year olds has not been sustained. AR peaked in 2010 for 15-24 year olds, followed by year by year decrease in accrual rates. In 2010, accrual for 15-19 year olds had increased to 37.4%, compared to 24.1% in 2005 and has now fallen to 14.5% in 2014. In 2010 accrual for 20-24 year olds was 18.1%, compared to 13.5% in 2005 depreciating to 6.1% in 2014. The number of available trials in 2010 was 21 compared to 10 in 2015.
Conclusion
The trend for improvements in AR for TYA have not been sustained since 2010. This is related to closure of key trials for TYA and limited availability of trials in rare cancers. The time taken between trials closing and subsequent trials opening also impacted accrual rates.

Patients and families urge palliative care to prioritise the financial cost of caregiving; reporting from the Palliative and End of life care Priority Setting Partnership (PeolcPSP); A national survey

Background
Palliative care is an under researched area. Less than 7% of total spent on cancer research is spent for cancer-related Palliative Care. A Palliative and End of Life Care Priority Setting Partnership (PeolcPSP) set up by Marie Curie, conducted a national survey, asking patients, carers and clinicians to set research priorities for palliative and end of life care. Financial implications of informal caregiving was one of the emergent themes.

Method
A supplementary analysis of the Palliative and End of Life care Priority Setting Partnership' national survey, by performing thematic analysis on the free-text responses. Results related to the financial costs of informal caregiving are the current focus.

Results
They public survey received 1,403 responses. 118 participants (8.41%) provided free-text responses in relation to economic costs and financial support for patients and their families. Participants discussed the financial challenges in looking after a dying family member at home. They argued for a need of financial support for both patients and their families and discussed challenges of accessing the existing support. Equipment and facilities for patients' homes, carer's allowance, employment benefit, after death support, lack of guidance and co-ordination of financial resources, and issues of equity across diseases and geographical areas were the main concerns presented by all participants. The participants confirmed a link between financial constraints and place of care and death, suggesting that palliative care at home is at least partially funded by the patient and the family and hence it might not be viable for individuals who lack personal resources.

Conclusion
Economic burden for the family has a significant impact on the quality of palliative care. Given the policy initiatives to move provision of palliative care from hospital to community settings, the costs of informal caregiving need to be urgently understood and considered in economic evaluations of palliative care services.
ACP McElwain Prize winner
Clonal neoantigens elicit T cell immunoreactivity and sensitivity to immune checkpoint blockade

Regulation of T cell function represents a key obstacle to the successful clinical application of tumour immunotherapy. Antibody-mediated blockade of co-inhibitory immune checkpoint molecules seeks to address such regulation. Durable remissions and even cure has been observed in patients with advanced cancer, however, such responses are limited to a small fraction of treated patients. The identification of biomarkers predictive of response and resistance to therapy therefore remains an area of high scientific priority. Recent studies highlight the impact of the genomic landscape on anti-tumour immunity. Tumour-specific mutations may serve as neoantigens, eliciting T cell responses, and appear to be enriched in patients with advanced melanoma and non-small cell lung cancer (NSCLC) deriving benefit from CTLA-4 and PD-1 blockade respectively.

Genetic heterogeneity within single tumours is well described, however the impact of intra-tumour heterogeneity (ITH) upon the neoantigen landscape and anti-tumour immunity has remained unclear. Through parallel genomic and immunological approaches, we identified neoantigen-reactive CD8+ T cells in patients with early-stage NSCLC. Characterisation of these cellular subsets revealed high expression of co-inhibitory immune checkpoint molecules including PD-1. Analysis of clonal architecture demonstrated that all identified T cell responses were directed against clonal neoantigens, present on every cancer cell. Clonal neoantigens were found to impact positively on overall survival in primary lung adenocarcinoma. Tumours with a high burden of clonal neoantigens displayed an inflamed phenotype, with high levels of CD8A, IFN-γ, STAT-1, PD-1, LAG-3 and PD-L1/2 gene expression. In keeping with these observations, sensitivity to CTLA-4 and PD-1 blockade in patients with advanced melanoma and NSCLC appeared enhanced in tumours enriched for clonal, but not subclonal neoantigens.

Heterogeneity in the neoantigen landscape may therefore influence immune surveillance. Clonal neoantigens, shared by all tumour cells, represent a key substrate for T cell recognition and an attractive target for adoptive cell-based and vaccination strategies which may hold promise to address the challenges of ITH.
# BACR/AstraZeneca Young Scientist Frank Rose Award winner Cancer specific non-essential amino acid metabolism – a role for targeted dietary intervention in cancer therapy?

Oliver D. K. Maddocks1,2, Dimitris Athineos1, Eric C. Cheung1, Pearl Lee1, Tong Zhang2, Julianna Blagih1, Kirsteen Campbell1, Niels van den Broek2, Gillian M. Mackay1, Christiaan F. Labuschagne1, Fatih Ceteci1,3, Owen J. Sansom1, Karen Blyth1 and Karen H. Vousden1

1 Cancer Research UK Beatson Institute, Switchback Road, Glasgow, G61 1BD, UK
2 University of Glasgow Institute of Cancer Sciences, Switchback Road, Glasgow, G61 1QH, UK
3 Institute for Tumour Biology and Experimental Therapy, Georg-Speyer-Haus, Paul-Ehrlich-Strasse 42-44, 60596 Frankfurt am Main, Germany

## Background

Altered cell metabolism is a fundamental hallmark of cancer and tumour cells acquire a range of metabolic perturbations and adaptations that support proliferation and survival. To sustain enhanced growth, cancer cells become dependent on uptake of nutrients such as glucose and amino acids. We have undertaken extensive studies to characterise the dependency of cancer cells on the non-essential amino acids serine and glycine; including the metabolic pathways dependent on these nutrients, and how this phenotype can be exploited for improved cancer therapy.

## Methods

To elucidate metabolic pathways involved in serine and glycine metabolism we have utilised and developed steady state metabolomics and carbon-13 labelled metabolic flux assays. These approaches, combined with conventional protein expression analysis and functional assays, have allowed us to characterise serine and glycine metabolism in cancer cells in vitro and in vivo.

## Results

We have found that many cancer cell lines are sensitive to serine starvation, despite the ability of these cells to synthesise serine de novo. Cancer cells undergo extensive metabolic remodelling in response to serine starvation requiring a balance between nucleotide and glutathione synthesis aided by the tumour suppressor p53. We have also characterised why excess glycine (a direct metabolite of serine) can inhibit cell proliferation when given in excess. In exploring the metabolic links between the methionine cycle and serine dependent one-carbon metabolism we have found that cancer cells do not ordinarily use serine to re-synthesise methionine, but rather use serine to support the methionine cycle – and DNA & RNA methylation – via de novo ATP synthesis. In pre-clinical studies with a range of murine models we show that certain tumours are sensitive to dietary serine restriction, which significantly improves survival as a sole therapeutic intervention.

## Conclusions

This work establishes a strong foundation for the continued investigation of dietary non-essential amino acid restriction as an adjunct to conventional chemo/radiotherapy.
Immunotherapy for genitourinary cancers and beyond

Since it was first proposed that targeting key immunomodulatory pathways may result in an effective anti-tumour immune response, immunotherapy research has progressed substantially. Today a wealth of clinical research is ongoing across a range of tumour types, highlighting a variety immunotherapeutic treatment approaches currently being evaluated.

At this symposium, Professor Hardev Pandha (University of Surrey) will discuss the development of immunotherapy for genitourinary cancers from the preclinical to late-phase clinical trial setting, focusing on the scientific rationale for immunotherapy and the ways in which they differ from conventional therapies. Professor Rob Jones (University of Glasgow) will then review the ongoing immunotherapy clinical trials in renal cell carcinoma, particularly the evaluation of immune checkpoint inhibitors in the metastatic, adjuvant and neoadjuvant stages of this disease. Finally, Professor Jeff Evans (University of Glasgow) will discuss the research of immunotherapeutic treatment approaches employed for other types of cancer, including hepatocellular carcinoma. We hope you will join us at the NCRI Cancer Conference 2016 for this informative event.

For healthcare professionals and scientific researchers only.

ONCUK1601418-01

Date of preparation: August 2016
Workshops

**De-mystifying today’s science**
Do words like signal transduction, epigenetics, genomics and biomarkers bring a puzzled frown to your face? Do you plan to attend today’s plenary lectures whilst fearing that you won’t understand a word? Well never fear!

Elaine will explain many of the words, concepts and ideas behind the day’s plenary lectures. Similar to previous years’ workshops, she will use diagrams and illustrations to provide clear, easy-to-understand explanations of complicated biological concepts.

The workshop is geared towards non-scientists, such as doctors, nurses, trials staff and patients who’d like to get the most out of this year’s conference.

**BACR educational workshop: Artificial cells – smart delivery systems for cancer therapy**

This talk and demonstration session will outline microfluidic strategies for bottom-up synthetic biology that are being used to construct multi-compartment artificial cells where the contents and connectivity of each compartment can be controlled. These compartments are separated by biological functional membranes that can facilitate transport between the compartments themselves and between the compartments and external environment. These technologies have enabled us to engineer multi-step enzymatic signalling cascades into the cells and manufacture cellular bionic systems where real and artificial cells are fused together to generate hybrid systems with enhanced functionality. These artificial systems have the potential to underpin exciting therapeutic applications including smart delivery, stealth shielding, chemical synthesis at the site of action and systems that are capable of sensing and responding to their environment.

**Accelerating adoption of research findings into NHS clinical practice**

This policy-focused session will shine a spotlight on the challenges associated with getting evidence from research adopted into NHS clinical practice in a quick and equitable way. The session will include case studies from the diagnostics, radiotherapy and surgical fields, a panel representing key decision-making bodies, and we hope active audience participation.

The new cancer strategy for England highlights the need to develop a more streamlined process through which research findings are incorporated as quickly as possible into mainstream practice. There are numerous examples where the NHS has been slow to adopt evidence-based interventions, meaning delayed access for patients and variable access across different localities in the UK. This is particularly problem in non-medicinal interventions such as radiotherapy, surgery or diagnostic pathway innovations where pathways of adoption across the UK are less clear.
Speakers:
Ferdia Gallagher, University of Cambridge, UK
Ananya Choudhury, The Christie NHS Foundation Trust, UK
David Phelps, Imperial College London, UK

Panellists:
Lord John Sharkey, Association of Medical Research Charities
Paul Chrisp, NICE
Mahiben Maruthappu, NHS England
Tara Donnelly, Health Innovation Network

The role of CTUs in optimising clinical research – beyond Figure 1

16.30 – 18.30
Room 4

Hosted by
Judith Bliss,
The Institute of Cancer Research,
London, UK

Working with a CTU – this session will provide translational researchers and clinicians with a greater understanding of the scientific opportunities and value provided by developing academic partnerships with NCRI Cancer CTUs to exploit clinical trial data “Beyond Figure 1” and maximise scientific learning.

Areas of contemporary scientific interest for clinical trials methodologists (e.g. use of routine data, determination of intermediate and surrogate endpoints, analysis of biomarker and imaging data) will be presented, each with a view to enhance multi-disciplinary collaborations.

An overview will be provided of the governance and regulatory framework within which clinical trials are conducted to provide a wider understanding of required processes or constraints.

Speakers:
Janet Dunn, University of Warwick, UK
Pamela Kearns, Cancer Research UK Clinical Trials Unit (CRCTU), University of Birmingham, UK
Claire Snowdon, The Institute of Cancer Research, London, UK
130  Programme at a glance
131  Plenary abstracts
132  Parallel sessions
136  Parallel session abstracts

Key

Diagnosis and therapy
Epidemiology and prevention
Healthcare delivery
Information, patients and the public
Supportive, palliative care, survivorship
The cancer cell and model systems
Programme at a glance

Parallel sessions

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<tr>
<th>Time</th>
<th>Session</th>
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<tbody>
<tr>
<td>09.00 – 11.00</td>
<td>An epidemiological overview of obesity and hepatocellular carcinoma</td>
<td>Helen Reeves, Newcastle University, UK</td>
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<tr>
<td>09.00 – 11.00</td>
<td>Strategies to enable more efficient clinical trials</td>
<td>Lucinda Billingham, University of Birmingham, UK</td>
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<tr>
<td>09.00 – 11.00</td>
<td>Towards delivering truly personalised therapy for oesophageal cancer</td>
<td>Tim Underwood, University of Southampton, UK</td>
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<tr>
<td>09.00 – 11.00</td>
<td>Cancer survivorship and late toxicity</td>
<td>John Radford, University of Manchester, UK</td>
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<tr>
<td>09.00 – 11.00</td>
<td>The changing face of cancer follow-up: Supported self-management</td>
<td>Claire Foster, University of Southampton, UK and Elspeth Banks, NCRI Psychosocial Oncology &amp; Survivorship Clinical Studies Group, UK</td>
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<tr>
<td>09.00 – 11.00</td>
<td>Opportunities for step changes in the cancer landscape through health informatics</td>
<td>Andrew Renehan, University of Manchester, UK and David Weller, University of Edinburgh, UK</td>
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NCRI Schools Event

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<tr>
<td>09.30 – 14.30</td>
<td>See the full programme for this year’s Schools Event in Appendix 1 on page 192</td>
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Networking, exhibition and poster viewing

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<th>Time</th>
<th>Event</th>
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<tbody>
<tr>
<td>11.00 – 12.00</td>
<td>Networking, exhibition and poster viewing</td>
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Plenary lectures

Chaired by Eyal Gottlieb, Cancer Research UK Beatson Institute, Glasgow, UK

<table>
<thead>
<tr>
<th>Time</th>
<th>Lecture</th>
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<tbody>
<tr>
<td>12.00 – 12.40</td>
<td>Biology and biomarkers: Molecular correlates of clinical outcomes in patients with metastatic solid tumours treated with anti-PD-(L)1 agents Alexandra Snyder, Memorial Sloan Kettering Cancer Center, USA</td>
</tr>
<tr>
<td>12.40 – 13.20</td>
<td>Understanding responses to cancer therapy: The tissue is the issue but the scoop is in the poop Jennifer Wargo, The University of Texas MD Anderson Cancer Center, USA</td>
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Closing remarks

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<th>Time</th>
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<tr>
<td>13.20 – 13.40</td>
<td>Closing remarks by Johann de Bono, The Institute of Cancer Research, London, UK and Chair of 2017 Scientific Committee</td>
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</table>

130 conference.ncri.org.uk  Download the App for the latest updates
Biology and biomarkers: Molecular correlates of clinical outcomes in patients with metastatic solid tumours treated with anti-PD-(L)1 agents

The anti-programmed cell death 1(PD-1) and anti-programmed death ligand-1 (PD-L1) agents have demonstrated promising activity in patients with multiple solid tumours, particularly melanoma, non-small cell lung, urothelial and renal cell cancers, with lower response rates in other tumour histologies. However, in most settings, only the minority of patients responds. Multiple biomarkers have been examined that associate with response, but a deeper understanding of the biology of response and resistance is needed. The promise and pitfalls of the leading candidate biomarkers, PD-L1 staining and mutation burden, will be discussed. The contribution of evolving technologies, including RNA- and T-cell receptor sequencing will be reviewed.

Finally, recent studies integrating data from the tumour and tumour microenvironment will be presented to demonstrate the complex, heterogeneous and dynamic tumour state. These factors present a challenge to the ongoing development of immunotherapies and bespeak a need for combination therapies to overcome primary and acquired resistance to checkpoint blockade.

Understanding responses to cancer therapy: The tissue is the issue but the scoop is in the poop

At the time of printing, this abstract had not been received. Check the Conference App for further details.
# Parallel sessions

## An epidemiological overview of obesity and hepatocellular carcinoma

**Hosted by Helen Reeves, Newcastle University, UK**

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<thead>
<tr>
<th>Time</th>
<th>Session</th>
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<tbody>
<tr>
<td>09.00 – 09.05</td>
<td>Introduction by the host</td>
<td>12</td>
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<tr>
<td>09.05 – 09.30</td>
<td>Understanding molecular and cellular mechanisms triggering NASH and subsequent HCC development</td>
<td>12</td>
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<tr>
<td></td>
<td><strong>Mathias Heikenwälder</strong>, German Cancer Research Center (DKFZ), Heidelberg, Germany</td>
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<tr>
<td>09.30 – 09.55</td>
<td>Advances in immunotherapy for patients with hepatocellular carcinoma</td>
<td>12</td>
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<td></td>
<td><strong>Tim Meyer</strong>, University College London, UK</td>
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<tr>
<td>09.55 – 10.20</td>
<td>Genetic factors in NAFLD: Disease progression and hepatocellular carcinoma</td>
<td>12</td>
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<tr>
<td></td>
<td><strong>Quentin Anstee</strong>, Newcastle University, UK</td>
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<tr>
<td>10.20 – 10.35</td>
<td>Proffered paper: Building a weight of evidence to prevent cancer in later life</td>
<td>12</td>
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<tr>
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<td><strong>Gillian Rosenberg</strong>, Cancer Research UK, London, UK</td>
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<tr>
<td>10.35 – 10.50</td>
<td>Proffered paper: Association between metabolic syndrome components and the risk of primary liver cancer and cirrhosis</td>
<td>12</td>
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<tr>
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<td><strong>Mieke Van Hemelrijck</strong>, King's College London, UK</td>
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<tr>
<td>10.50 – 11.00</td>
<td>Discussion</td>
<td>12</td>
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## Strategies to enable more efficient clinical trials

**Hosted by Lucinda Billingham, University of Birmingham, UK**

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<tbody>
<tr>
<td>09.00 – 09.05</td>
<td>Introduction by the host</td>
<td>11</td>
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<tr>
<td>09.05 – 09.30</td>
<td>Improving efficiency of phase II oncology trials using RECIST endpoints</td>
<td>11</td>
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<td><strong>James Wason</strong>, University of Cambridge, UK</td>
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<td><strong>Richard Simon</strong>, National Cancer Institute, USA</td>
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<td>Tumour-size as a surrogate for overall survival in advanced colorectal cancer</td>
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<td><strong>Tomasz Burzykowski</strong>, Hasselt University, Belgium</td>
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<td><strong>Dominic Rothwell</strong>, Cancer Research UK Manchester Institute, UK</td>
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09.00 – 09.10  
**Room 3A** 
**Introduction by the host**

09.10 – 09.40  
**Room 3A** 
Development of biomarkers to aid delivery of stratified trials for oesophageal cancer  
**Fergus Noble**, University of Southampton, UK

09.40 – 10.10  
**Room 3A** 
A mouse model of Barrett Esophagus - translational opportunities in GI cancer  
**Michael Quante**, Technical University of Munich, Germany

10.10 – 10.40  
**Room 3A** 
Mutational signatures reveal subgroups of distinct aetiology and new therapeutic opportunities in oesophageal adenocarcinoma  
**Maria Secrier**, University of Cambridge, UK

09.00 – 09.10  
**Hall 1B** 
**Introduction by the host**

09.10 – 09.40  
**Hall 1B** 
Second malignancy risk following treatment for lymphoma  
**Flora van Leeuwen**, Netherlands Cancer Institute, The Netherlands

09.40 – 10.10  
**Hall 1B** 
Cardiac toxicity among cancer survivors: Is everything old new again?  
**David Hodgson**, Princess Margaret Cancer Centre, University of Toronto, Canada

10.10 – 10.40  
**Hall 1B** 
Approaches to patient-experience and patient-centredness research  
**Janelle Yorke**, The Christie NHS Foundation Trust, UK

10.40 – 11.00  
**Hall 1B** 
Discussion
### Parallel sessions (continued)

#### The changing face of cancer follow-up: Supported self-management

Hosted by Claire Foster, University of Southampton, UK and Elspeth Banks, NCRI Psychosocial Oncology & Survivorship Clinical Studies Group, UK

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<td>The changing face of cancer aftercare, what this means to patients and clinicians&lt;br&gt;<strong>Dame Jessica Corner</strong>, University of Nottingham, UK</td>
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<td>Development and testing of evidence-based psycho-educational interventions that can be readily adopted into clinical practice&lt;br&gt;<strong>Penny Schofield</strong>, Swinburne University of Technology, Australia</td>
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<td>Facilitating self-management and personalised access to supportive care&lt;br&gt;<strong>Irma Verdonck-de Leeuw</strong>, VU University Medical Center, The Netherlands</td>
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<td>Proffered paper: The impact of co-morbidities on recovery from colorectal cancer within first 2 years after surgery: results from the UK Colorectal Wellbeing (CREW) cohort study&lt;br&gt;<strong>Amanda Cummings</strong>, University of Southampton, UK</td>
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<td>10.35 – 10.50</td>
<td>Proffered paper: Impact of Dementia on treatment of older patients with breast cancer: An interim analysis of the Bridging the Age Gap in Breast Cancer Study&lt;br&gt;<strong>Osama Zaman</strong>, University of Sheffield Medical School, UK</td>
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<td>10.50 – 11.00</td>
<td>Discussion&lt;br&gt;Chaired by <strong>Elspeth Banks</strong>, NCRI Psychosocial Oncology &amp; Survivorship Clinical Studies Group</td>
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Opportunities for step changes in the cancer landscape through health informatics

Hosted by Andrew Renehan, University of Manchester, UK and David Weller, University of Edinburgh, UK

09.00 – 09.10  Introduction by the hosts
Room 3B

09.10 – 09.40  Cancer data management and analysis using a pre-planned multi-database approach across Nordic countries and the United Kingdom
Morten Andersen, Karolinska Institute, Stockholm, Sweden
Room 3B

09.40 – 10.10  The informatics challenges facing biobanks: A perspective from a United Kingdom biobanking network
Philip Quinlan, University of Nottingham, UK
Room 3B

10.10 – 10.40  Understanding outcomes of whole pathways of care: The national cancer vanguard approach to utilising cancer intelligence
Kathy Pritchard-Jones, University College London Hospitals (UCLH)
Cancer Collaborative, UK
Room 3B

10.40 – 11.00  Discussion
Room 3B
**An epidemiological overview of obesity and hepatocellular carcinoma**

**Introduction**

Obesity is increasingly common. Its consequences include adipose dysfunction, low grade inflammation, impaired lipid and glucose handling, insulin resistance and non-alcoholic fatty liver disease, all of which increase cancer risk. Epidemiological data indicating that mortality attributed to obesity related hepatocellular carcinoma in particular is rising dramatically in the UK will be presented, before addressing some of the underlying potential mechanisms. This will be followed by an overview of advances in medical therapy for patients with hepatocellular carcinoma. The final talk will cover clinically relevant genetics of these diseases, discussing how these may – alongside behavioural and lifestyle changes – prevent disease progression, as well as potentially aiding earlier detection and treatment stratification.

**Understanding molecular and cellular mechanisms triggering NASH and subsequent HCC development**

Enhanced fat uptake by hepatocytes in combination with a sedentary life style leads to non-alcoholic fatty liver disease (NAFLD), comprising a spectrum of liver disorders ranging from fatty liver (steatosis) to nonalcoholic steatohepatitis (NASH) which can proceed to fibrosis, cirrhosis and HCC. Currently, 90 million Americans and 30 million Europeans suffer from NAFLD. At the same time there is no established pharmaceutical to treat NASH and established standard of care therapy for HCC is limited. A “two-hit hypothesis” has been proposed for NASH progression from NAFLD: Lipid accumulation in the cytoplasm of hepatocytes is considered the first step in NASH development; however, a second hit promoting oxidative stress, inflammation, DNA damage, hepatocyte cell death and fibrosis is needed. In C57BL/6 mice, NASH can be induced by methionine/choline-deficient diet (MCD) or choline-deficient diet (CD) but not by high fat diet (HFD) alone. However, C57BL/6 mice fed with MCD or CD do not develop obesity, metabolic syndrome or HCC and the diet has to be discontinued after a few months due to weight loss (up to 40%) or cachexia. Hence, these short-term approaches fail to recapitulate NASH-induced long-term consequences found in the human liver and possibly other metabolic organs. Deficiency of the essential nutrient choline was described in NAFLD patients to exacerbate NAFLD and NASH. Moreover, humans with insufficient choline-uptake have defects in hepatic lipoprotein secretion, oxidative damage caused by mitochondrial dysfunction and ER stress. We combined choline deficiency with a high fat diet (CD-HFD), which we hypothesized may lead to metabolic syndrome, steatosis, liver damage and NASH, thus delivering the “second hit” that promotes dietary-induced liver carcinogenesis - similar to the human situation. This approach enabled us to study a chronic mouse model of NASH in the context of metabolic syndrome, triggering subsequent HCC in C57BL/6 mice, in the absence of chemical carcinogens or genetic mutations predisposing to NASH or HCC development. CD-HFD fed mice display several pathologies for a long time frame: abdominal obesity, overweight, insulin resistance, liver damage, fibrosis, hepatic mitochondrial damage, dyslipidemia and NASH as observed in human patients. Moreover, HCC developed 12 months post CD-HFD start resembling human HCC. Using this mouse model we demonstrated that CD8+ T-cells and NKT-cells become activated during metabolic syndrome, interact with hepatocytes and alter hepatic lipid metabolism causing NASH and HCC. Here I will report on novels findings in regards to the use of this and other NASH models.
Advances in immunotherapy for patients with hepatocellular carcinoma

At the time of printing, this abstract had not been not received. Check the Conference App for further details.

Genetic factors in NAFLD: Disease progression and hepatocellular carcinoma

Hepatocellular carcinoma (HCC) incidence is rising in the UK and globally. Due to rising worldwide obesity, non-alcoholic fatty liver disease (NAFLD) is an increasingly frequent underlying aetiology. NAFLD encompasses a spectrum that spans simple steatosis, through steatohepatitis (NASH) to fibrosis and ultimately cirrhosis and HCC. NAFLD is considered a complex disease trait that occurs when environmental exposures act upon a susceptible polygenic background composed of multiple independent modifiers. Genome-wide association studies and candidate-gene studies have informed our understanding of factors contributing to the well-recognised inter-individual variation in disease progression and outcomes in NAFLD. This presentation will discuss the role of genetic modifiers across the full spectrum from steatosis to HCC; in particular, functional coding variants in modifier genes including PNPLA3 and TM6SF2, as well as recent intriguing data suggesting an epigenetic contribution to disease pathogenesis. Future areas of genetic research, and the potential for these advances to influence clinical management will be discussed.

Proffered paper: Building a weight of evidence to prevent cancer in later life

Background
Obesity is the largest preventable risk factor for cancer after smoking. Being overweight as an adult is linked to 10 cancer types, and overweight children are more likely to become overweight adults. Recent data shows that around one in three children leave primary school overweight or obese, with the most deprived children twice as likely to be so. A comprehensive evidence-based childhood obesity strategy is vital in order to prevent obesity related cancers later in life.

Method
A multidisciplinary research strategy was developed at Cancer Research UK to build a body of evidence that can directly influence government policy making in obesity. This included a quantitative study to investigate obesity and cancer awareness in the general population; a modelling study to predict future obesity related cancer cases; and qualitative studies to explore obesity-linked behaviours in children.

Results
Cancer was not at the forefront of people’s minds when thinking about obesity, with only 26% showing an unprompted awareness of the link. However the projected impact of obesity on cancer is high: if current trends continue it will lead to a further 670,000 cases over the next 20 years. When looking specifically at childhood obesity, it was found that junk food marketing was associated with parental pester power and can override nutritional knowledge.

Conclusion
These research studies formed an integral part of the current Cancer Research UK campaign on obesity. The first stage addressed the poor knowledge of obesity and cancer in the general population. The modelling results were a key part of this, alongside existing data on the...
mechanisms behind the causal relationship of obesity and cancer. Following this, during the development of the government’s childhood obesity strategy, the childhood obesity studies demonstrated to policy makers the importance of taking action to limit advertising exposure in children.

**Proffered paper: Association between metabolic syndrome components and the risk of primary liver cancer and cirrhosis**

**Background**
Hepatocellular carcinoma (HCC) accounts for the majority of malignant primary liver cancers (PLCs). Metabolic syndrome is associated with non-alcoholic fatty liver disease, progression of which may result in cirrhosis, a significant risk factor of HCC. We therefore aimed to investigate the association between metabolic syndrome components (lipids, raised glucose, diabetes and obesity), PLC and cirrhosis.

**Method**
A total of 509,436 participants from the Swedish AMORIS cohort, recruited January 1985 to December 1996 (study end-date December 2011), aged ≥20 with baseline triglycerides (TG), total cholesterol (TC) and glucose were included. Those with benign or metastatic liver cancer or cirrhosis at baseline were excluded. Lipids were categorised using clinical cut-offs and quartiles. Multivariate Cox proportional hazards, adjusted for age, gender, socio-economic status, liver disease and metabolic factors were used to estimate the association with PLC and cirrhosis.

**Results**
There were 766 PLC and 2,775 cirrhosis cases (mean follow-up 13 years). Raised TG, low TC, raised glucose, diabetes and low HDL (according to clinical cut-offs and quartiles) were associated with an increased risk of developing cirrhosis and PLC (e.g. TG≥1.71mmol/l vs <1.71mmol/l, HR: 1.38 (95% CI:1.17-1.63)). Raised ApoB and ApoB/ApoA-I were also associated with an increased risk of PLC whilst low LDL quartiles, raised TG/HDL and low ApoA-I were associated with an increased risk of cirrhosis (e.g. ApoA-I<1.05g/L vs ≥1.05g/L, HR: 1.78 (95% CI:1.31-1.63)). Obesity (BMI >30kg/m2) was not independently associated with PLC or cirrhosis, but raised glucose and diabetes remained significant risk factors after additional BMI adjustment. Raised TG, low TC, raised glucose and diabetes showed stronger associations with PLC in patients with a history of cirrhosis. Many patients developed PLC without a history of cirrhosis.

**Conclusion**
Metabolic serum biomarkers (triglycerides, cholesterol, apolipoproteins B and A-1, and glucose) were independently associated with an increased risk of developing PLC and cirrhosis, but obesity was not.

**Discussion**
Introduction
The session will present and discuss emerging trial methodology that aims to make cancer clinical trials more efficient. The efficiency of phase II trials can be improved by using continuous tumour shrinkage endpoints rather than the conventional overall response rate. In stratified medicine, master protocols implementing basket and umbrella designs are being used to provide an efficient approach to evaluate targeted therapy. The efficiency of phase III trials can be improved by incorporating prior evidence in a Bayesian design or through the use of valid surrogate outcome measures.

Improving efficiency of phase II oncology trials using RECIST endpoints
In phase II trials in solid tumours, endpoints are generally based on the RECIST criteria. This categorises patients based on their percentage change in tumour size and other reasons for non-response such as whether a patient develops new lesions. Normally these endpoints are analysed in a binary way – i.e. estimating the proportion of patients who are responders. However, this ignores the information contained in the change in tumour size. Dichotomising continuous outcomes loses a lot of information and means higher sample sizes are needed for the same power. Analysing the continuous change in tumour size, although statistically efficient, is not always clinically meaningful as it ignores the other reasons for non-response.

This talk will discuss a method to analyse the clinically relevant response endpoint, but using the information contained in the change in tumour size. This uses a statistical model of the different components of the responder-based outcome. The method results in considerably higher power without any additional cost. Generally, the increase in power is equivalent to increasing the sample size by around 35%. I will also discuss current work that aims to extend this method to progression-free survival.

Personalised oncology in 2016: New paradigms in clinical trial methodology
Cancers are diseases of DNA dis-function and new diagnostic classification systems based on somatic genomic alterations are rapidly replacing traditional systems based on primary site and histology. A large proportion of the cancer drugs that have been approved by regulatory authorities in the past decade have an intended use for a restricted subset of patients. The intended use subset is often characterised by genomic de-regulation of a gene related to the molecular target of the drug. Much current drug development in oncology involves co-development of a companion in-vitro diagnostic test for selecting the subset of patients who are likely to benefit from the drug. The companion diagnostics are often based on DNA sequencing of patients’ tumours.

Progress in the development of effective drugs has increased in oncology. The progress has been based on use of non-traditional clinical trial designs such as enrichment designs in which a relatively narrow subset of patients is selected for randomization instead of the usual broad eligibility trials. Adaptive enrichment designs and run-in designs have been developed for settings where a single candidate predictive biomarker is not known a-priori. There is also considerable interest in basket clinical trials are phase II development studies which accrue patients with a common genomic alteration but a range of histologic types of tumours.

I will review some of the new phase II and III designs for biomarker driven clinical trials that have been developed and used in oncology.
Tumour-size as a surrogate for overall survival in advanced colorectal cancer

It has been shown (for instance, in metastatic breast cancer or advanced colorectal cancer) that tumour response is not a valid surrogate for overall survival. However, tumour response is a binary endpoint and it ignores information contained in the change in tumour size, upon which it is based. Thus, one can ask question whether change in tumour size might be a better candidate for a surrogate for overall survival. In the presentation we will present results of an analysis aiming at answering this question using a database of clinical trials in colorectal cancer.

Proffered paper: The TARGET trial: molecular profiling of circulating tumour DNA to stratify patients to early phase clinical trials

Background

The Tumour chARacterisation to Guide Experimental Targeted Therapy Trial (TARGET) tests the hypothesis that molecular profiling of both archival/fresh tumour and circulating tumour DNA (ctDNA) can be used to stratify patients to early phase trials of targeted therapies to maximise patient benefit.

Method

Patients were consented for molecular analysis of tumour and blood. Tumour was analysed by Sequenom OncoCarta using a 19 gene panel. ctDNA was subjected to next generation sequencing (NGS) and bioinformatic analysis of a panel of >600 genes known to be frequently mutated in cancer. Clinical reports from tumour and blood were discussed in a monthly Molecular Tumour Board (MTB) to identify possible driver aberrations and to aid clinicians in selection of relevant experimental medicine trials.

Results

The initial stages of the trial have focused on process development, optimisation of ctDNA sequencing, bioinformatic analysis and establishing the MTB. The current ctDNA pipeline identified at least one mutation within ctDNA from 87.5% (35/40) samples. For the first 20 samples concordance between tumour and ctDNA was 90%. Eight patients had clinically relevant mutations, confirmed in ctDNA by droplet digital PCR and/or repeat NGS. The MTB has been optimized to review and interpret tumour and ctDNA reports within 3-4 weeks of consent and has identified relevant clinical trials for individual patients.

Conclusion

Our results support the use of ctDNA for routine molecular characterisation. The success of the overall approach has led to scale up of patient recruitment to ~350 patients over the next 2-3 years. The focus of ongoing work will be to allocate patients to clinical trials based on ctDNA and/or tumour profiling and facilitate monitoring of treatment response and emerging resistance mechanisms using serial blood samples. Outcome measures will include numbers of patients allocated and recruited to matched experimental medicines, response rates and survival outcomes.
**Proffered paper: National Lung Matrix Trial: successful implementation of a phase II umbrella trial testing multiple genetic-marker-directed drugs in advanced non-small cell lung cancer**

**Background**
The National Lung Matrix Trial (NLMT) is a flagship trial in the United Kingdom being the first to combine the development of a technology platform that screens for multiple genetic aberrations in tumours (provided by the Cancer Research UK Stratified Medicine Programme 2) with testing of multiple novel genetic-marker-directed drugs. The trial is focused on patients with advanced non-small cell lung cancer (NSCLC) and currently includes 7 different drugs targeting 20 molecular markers. In addition, patients with no actionable genetic change (NA) are included and will be treated with a sequential pipeline of drugs. This paper summarises the progress with the implementation of this umbrella trial.

**Method**
Eligible patients are recruited to treatment arms depending on molecular targets identified by SMP2. The protocol currently includes targeted drugs that inhibit FGFR (AZD4547), mTORC-1/2 (vistusertib), CDK-4/6 (palbociclib), ALK (crizotinib), MEK (selumetinib) combined with docetaxel, AKT (AZD5363) and EGFR mutation positive T790M+ (osimertinib). Current treatment for NA is a PD-L1 Inhibitor (durvalumab). The trial aims to recruit from the 18 Experimental Cancer Medicine Centres (ECMCs) with each treating additional patients referred from feeder sites. Target recruitment for each drug-biomarker cohort is 30 patients to determine whether there is sufficient signal of activity in any drug-biomarker combination to warrant further investigation with interim analyses after 15 patients.

**Results**
The first participating centre opened to recruitment in March 2015 and by June 2016, 12 ECMC sites were open. At that time, 61 patients had been recruited covering all treatment arms (range 1 to 16 with 18 on the NA arm) and only 3 drug-biomarker cohorts with no patients.

**Conclusion**
NLMT has been successfully implemented with two further treatment arms in the pipeline. Continued recruitment will ensure the trial produces important results in the stratified treatment of advanced NSCLC.

**Discussion**

**Towards delivering truly personalised therapy for oesophageal cancer**

**Introduction**
Current therapies for oesophageal cancer are limited with surgery the mainstay of curative treatment. In this session the speakers will discuss how our understanding of the origins and development of oesophageal cancer will underpin the next generation of targeted therapies.
A mouse model of Barrett Esophagus – translational opportunities in GI cancer

Esophageal adenocarcinoma (EAC) is the most rapidly increasing cancer in the Western World and has a very poor prognosis with a median survival of less than one year. Although the reasons for this dramatic increase at a rate of up to 10% annually are mostly unknown, there have been a relatively small number of basic research studies or preclinical models that have been able to address important questions in the field. Barrett Esophagus (BE) is a known precursor for EAC and is a premalignant, inflammation-dependent condition of the esophagus, characterised by a metaplastic change from squamous to columnar intestinal-type epithelium. Substantial resources are expended on surveillance of BE, with the goal of early detection of HGD or EAC. Unfortunately, endoscopic surveillance of patients with BE has largely failed as a strategy to prevent cancer since we cannot predict the development of EAC based on histological findings from metaplastic BE biopsies without signs of dysplasia. Molecular biomarkers are urgently needed as prognostic markers to identify those patients with endoscopically diagnosed and histologically confirmed BE who have increase risk to develop EAC from BE. Good prediction of neoplastic progression would allow focusing surveillance on patients with a high risk of malignant transformation. Thus, there is a critical need to develop preventive strategies and therapies for a growing population of BE patients. However, the development of surveillance strategies and chemoprevention therapies for BE has been severely restricted by the absence of a tractable pre-clinical model for BE and EAC. Validated preclinical models could assist in the search for such biomarkers of risk and consequently help to design prevention trials by providing new insight into the biology of an inflammation-driven metaplasia to dysplasia sequence, and the factors that drive inflammation induced carcinogenesis in general and specifically the progression of BE to EAC. To address this need we have developed the L2-IL-1β transgenic mouse model of BE that recapitulates the histologic progression to EAC while inducing chronic inflammation in the esophagus and in my lab we use this model to translate our understanding of pathology to human disease. We analyse the importance and function of the microenvironment during the development of BE and EAC and the impact of these changes on stem cells and cancer initiating cells. We will utilize the first and unique inflammatory transgenic L2-IL-1b mouse model of BE/EAC, a novel 3D organotypic in vitro culture system of BE, and biopsies of human patients at different time points during progression from BE to EAC.

Mutational signatures reveal subgroups of distinct aetiology and new therapeutic opportunities in oesophageal adenocarcinoma

Oesophageal adenocarcinoma has increased rapidly in the western world, but the underlying causes and the mutational processes that contribute to this cancer are still unclear. Due to the lack of robust classification methods, targeted therapy trials have so far been disappointing. We sought to investigate the aetiology and sub-classification of oesophageal adenocarcinoma by characterising the mutational patterns in the genomes of 129 chemonaive patients for which whole-genome sequencing data was available. Mutational signatures reveal three distinct molecular subtypes with potential therapeutic relevance, verified in

Further analysis and experimental validation suggests that PARP inhibitors may be effective in the HR-scarred tumours of the ‘DDR impaired’ subgroup, while combination therapy targeting ERBB2/MET could be further investigated in the ‘C>A/T dominant’ subgroup. The ‘mutagenic’ subgroup appears to be linked to gastric acid exposure, and its genomic features, as well as higher CD8+ T-cell densities in these tumours suggest an increased immunogenic potential. In summary, mutational signatures have not only uncovered three subgroups of oesophageal adenocarcinoma with clear aetiological differences, but could also be used as a high-throughput, spatially unbiased stratification strategy in the clinic, to inform treatment options for this cancer.

Discussion

Cancer survivorship and late toxicity

Introduction

Many more patients are surviving cancer than in previous decades. This is clearly a cause for celebration but unfortunately these improvements come at the price of late treatment toxicity which undermine long term quality of life and survival. These late effects of radiotherapy and chemotherapy include increased risks of second cancers, cardiovascular disease, infertility, hormonal disturbances and psychosocial problems.

In this session international experts in the field will review the effects of treatment for a first cancer on the development of second cancers and cardiovascular disease and how these and other late effects impact on the survivor experience. The session will also consider how we can best mitigate the effects of late toxicity for those already treated for cancer and develop new, less harmful approaches to treatment for those yet to be diagnosed.

Second malignancy risk following treatment for lymphoma

At the time of printing, this abstract had not been not received. Check the Conference App for further details.
Cardiac toxicity among cancer survivors: Is everything old new again?

Epidemiologic studies of delayed cardiac toxicity require prolonged follow-up to produce reliable estimates of long-term risk. By definition, then, these estimates often apply to obsolete treatments, creating a challenge for clinicians to infer the risks associated with modern treatment and optimize the management of contemporary patients.

This presentation will describe how the development of novel systemic agents, better understanding of anthracycline and radiation toxicity, and more sensitive measures of cardiac function can be expected to influence the risks of cardiac toxicity among cancer survivors. With emphasis on lymphoma, participants will gain insight into how to extrapolate toxicity data from historic treatment to modern clinical decision making, and apply the evidence for secondary prevention of cardiac toxicity. In addition, insights into the genetic correlates of cardiac toxicity, and some unsolved methodologic issues relevant to studies of late cardiac toxicity will be addressed.

Approaches to patient-experience and patient-centredness research

At the time of printing, this abstract had not been not received. Check the Conference App for further details.

Discussion

The changing face of cancer aftercare, what this means to patients and clinicians

The renewed focus on health and wellbeing after cancer diagnosis and treatment has brought to the fore the ongoing needs for support of cancer survivors and the need to develop new models of aftercare. Until recently priorities for the design of new approaches had not been identified, though understanding of how support might be tailored to meet the needs of different individuals on the basis of risk factors is emerging. The importance of preparation for managing the effects of treatment and the process of recovery has recently been identified from patient feedback through the National Colorectal PROMS study. Evaluation of a model of supported self-management aftercare in a cancer centre will be presented showing how implementation of new models of care is a complex process as is understanding the outcomes of this for patients, clinicians and services. A key finding is that
defining the individual dimensions of supportive care required by cancer survivors is an essential pre-cursor to risk stratification in follow-up care. Individuals receiving appropriately tailored self-managed follow-up report high levels of satisfaction with their care, the model also released clinical capacity enabling resources to be diverted to support patients with complex needs.

**Development and testing of evidence-based psycho-educational interventions that can be readily adopted into clinical practice**

As the global burden of cancer increases, health care services face mounting challenges in meeting the complex needs of patients where direct clinical contact may be constrained or not readily available. Patients and families require resources and skills to manage their illness within their own communities. A framework for the development and delivery of psycho-educational interventions drawing on theoretical principles of behaviour change and evidence-based interventions will be presented. At the core of this framework are considerations of efficiency: interventions are designed to cater for individuals' unique needs; to place minimal demands on the health system infrastructure and to be rapidly disseminated into usual care if successful. There are seven key features: (1) Targeting cancer type and stage; (2) Tailoring to unique individual needs; (3) Promotion of patient self-management of their disease and treatment side-effects; (4) Efficient delivery of the intervention; (5) Training and adherence to protocol; (6) Ensuring the intervention is evidence-based; (7) Confirming stakeholder acceptability of the intervention. Case studies of a randomised controlled trial which tested psycho-educational oncology interventions using this framework will be presented. This framework, which is driven by theory, evidence, and experience, is designed to ensure that interventions are effective, clinically feasible and sustainable.

**Facilitating self-management and personalised access to supportive care**

The number of cancer survivors will continue to grow, and many cancer survivors have unique care needs. Barriers to supportive care are a lack of screening instruments in oncology settings, and that traditional models of supportive care do not meet current demands. Self-management and e-health can be used to enhance the efficiency of supportive care. For example, in the Netherlands, cancer survivors can use the Oncokompas2.0 to fill in questionnaires at home on their quality of life, existential issues, and lifestyle, and view their personal well-being profile. Supported by an evidence-based knowledge and decision support algorithm, advices are generated automatically, and based on the individual well-being profile, cancer survivors can be directed towards guided self-help treatments or professional care providers, according to a matched care model. When systems such as the Oncokompas2.0 are implemented in regular care, big data analyses will provide better insight into the needs of cancer survivors for supportive cancer care. Also, more understanding will be obtained into the possible determinants of supportive cancer care and the success of interventions. Eventually, this will lead to a “personalised” instead of a “one size fits all” approach of supportive care in the future.
Proffered paper: The impact of co-morbidities on recovery from colorectal cancer within first 2 years after surgery: results from the UK Colorectal Wellbeing (CREW) cohort study

**Background**
As the number of cancer survivors increases more people have to deal with the consequences of multiple morbidities. We describe frequencies of co-morbidities in a UK colorectal cancer cohort treated with curative intent and associations with health-related quality of life (HRQoL) outcomes up to two years following surgery.

**Method**
Cohort study of 872 UK colorectal cancer patients (Duke's stage A-C) recruited November 2010-March 2012 from 29 centres, awaiting curative intent treatment and consenting to follow-up. Questionnaires were administered at baseline (pre-surgery), 3, 9, 15, 24 months. Co-morbidities were self-reported by participants from 3 months. The EORTC QLQ-C30 and QLQ-CR29 assessed global health/QoL, symptoms and functioning from 3 months onwards. Longitudinal analyses investigated associations between co-morbidities and HRQoL outcomes.

**Results**
The mean age of participants was 68 years, with 60% male, 65% colon and 35% rectal cancer. Of the 658 participants who completed the co-morbidities questions at 3 months, 28% had none, 32% had one, 23% had two and 17% had 3+ co-morbidities. The most common were high blood pressure (44%), arthritis/rheumatism (33%), anxiety/depression (18%), diabetes/high blood sugar (16%) and asthma (16%). 28% reported that co-morbidities limited their daily activities. An increasing number of co-morbidities was associated with poorer global health/QoL, worse symptoms and poorer functioning on many domains, regardless of cancer site, stage or treatment. Co-morbidities strongly associated with poorer global health/QoL were arthritis/rheumatism, asthma, heart failure, chest pain and anxiety/depression.

**Conclusion**
Pre-existing co-morbidities are an important determinant of recovery of QoL following colorectal cancer, regardless of cancer stage and treatment. Clinical assessment should prioritise the presence of comorbidities to help identify those patients at risk of reduced QoL. Tailored follow-up, shared-care management of comorbidities and enhancing supported self-management could aid recovery of health and wellbeing in these patients.

Proffered paper: Impact of Dementia on treatment of older patients with breast cancer: An interim analysis of the Bridging the Age Gap in Breast Cancer Study

**Background**
One third of all breast cancer cases occur in women over 70 in whom co-morbidity rates may be higher than younger patients. Seven percent of women aged over 65 have dementia and so the two conditions often co-exist. Dementia is associated with a reduced life expectancy and increases the risk of acute or chronic post-operative cognitive dysfunction after surgery. This study has examined the impact of dementia on women with early breast cancer in terms of treatment decisions and post-operative complications.
Method
The Age Gap study is a large, multicentre, prospective cohort study of older women with early breast cancer. Interim comparison of treatment allocation and surgical complications in women with and without a clinical or MMSE confirmed diagnosis of dementia, was performed. Comparison of rates of surgery in women with and without dementia was assessed via simple logistic regression. Surgically treated patients with or without dementia were compared using Chi2 to detect any association between the presence or absence of dementia and incidence of local and systemic complications.

Results
Data was available for 1965 patients recruited between April 2013 and December 2015, from 51 UK hospitals. Of these 207 (10.5%) had dementia. Median age of the patient cohort was 77 years (70 - 101). Patients with dementia were 66% less likely to receive surgery than those without dementia (OR 0.344, 95% CI 0.249 – 0.473, p < 0.001). In patients undergoing surgery, dementia was not a significant predictor of local (p<0.16) or systemic (p<0.84) complications.

Conclusion
This study suggests that whilst older patients with dementia seem to tolerate surgery well, they are significantly less likely to undergo breast cancer surgery. Whether this will translate into higher breast cancer specific mortality in dementia patients is unknown, although overall survival in dementia patients is known to be reduced.

Discussion

Opportunities for step changes in the cancer landscape through health informatics

Introduction
Health informatics research offers critical opportunities to refine knowledge about cancer and how application of that knowledge can positively impact patient care. Cancer research requires large, multi-centre, deep-phenotype studies at local, regional and, national levels, where the focus is the alignment and linkage of all available biomedical data (improving data quality) per individual, the ability to link health (care) history, genetic, other biological information, environments and lifestyles in a trustworthy way. This session shall discuss how new cancer research collaborations can leverage the scale of the UK’s diverse populations and multiple Universities, The NHS, companies and other stakeholder organisations. The session will illustrate how networked analytics can assist earlier cancer diagnosis and population-level treatment optimizations.
Cancer data management and analysis using a pre-planned multi-database approach across Nordic countries and the United Kingdom

Combining data from multiple countries is valuable when studying rare exposures and outcomes. In the CARING (CAncer Risk and INsulin analoGues) project, data from national health registers in the Nordic countries Denmark, Finland, Norway and Sweden and the United Kingdom Clinical Practice Research Datalink were combined with the purpose of investigating cancer risks associated with specific insulins.

A major challenge in multi-country studies is how to combine databases with different structure and different drug and diagnosis classification systems. One approach is to analyse country-specific data separately and combine the results using aggregate data (AD) meta-analysis. Another approach is to collect all data centrally at one site and perform a combined individual patient data (IPD) meta-analysis.

The implementation of a common data model (CDM) to harmonise databases has several advantages. Less resources are needed both for generating analysis datasets and for the statistical analysis when data are in a uniform structure. Furthermore, the use of an integrated concept dictionary enables the mapping of exposure, confounder and outcomes concepts used in the study to country-specific code systems of drugs and diagnoses in an efficient and transparent way.

New insulin user cohorts were formed and Poisson regression used to estimate incidence rate ratios of colorectal cancer, breast cancer and prostate cancer, comparing exposure to different insulins. Analyses were performed both as an IPD meta-analysis on a common dataset from all countries (adjusted for common covariates), and on separate datasets for each country (adjusted for all available covariates), subsequently pooling results using AD meta-analyses with fixed and random effects. No consistent differences in risk of the cancers investigated between different insulins were found. Low power in individual cohorts and uniform distribution of available covariates between cohorts favoured the use of IPD over AD meta-analysis in this specific case.

The informatics challenges facing biobanks: A perspective from a United Kingdom biobanking network

At the time of printing, this abstract had not been not received. Check the Conference App for further details.
Understanding outcomes of whole pathways of care: The national cancer vanguard approach to utilising cancer intelligence

Cancer outcomes for an individual and for populations are determined by many factors, all of which can be variable. These may be related to organisation of healthcare service and how patients interact with these, diagnostic processes, treatment planning and delivery, as well as biological characteristics of the individual and their tumour. Until now, information about each of these aspects of a patient’s pathway has often been as fragmented as their care pathway itself. Improvement efforts have tended to focus on service elements, such as the advantages of redesigning complex cancer surgery for a large population or new models of direct access to diagnostics or a definitive diagnosis for general practitioners or stratified follow up that support patients to self-manage in the community. Providing high quality intelligence on the use and outcome of such services is helping to drive improvement and provide evidence for the long term patient outcome and health service benefits of these new models.

The national cancer vanguard comprises the UCLH Cancer Collaborative, RM Partners and Greater Manchester, serving a population of 10.8 million. Our informatics expertise is working closely with the expertise and datasets of Public Health England (PHE) and NHS England to augment the value of investment in data and clinical outcomes and avoid duplication. We aim to improve understanding of the whole patient pathway, both at an individual and population level for defined cohorts of patients, using existing clinical data in ways that enable the provider clinical teams and each footprint’s cancer system-leadership role to monitor and evaluate outcomes across the whole patient pathway, with rapid feedback to clinical teams and services. Working with local digital roadmaps in our respective Sustainability and Transformation Planning footprints, we will integrate data on ‘real life’ routes to diagnosis with actual treatment and holistic care that patients receive.

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Contact us
For further details about this role please contact:
Dr Matt Sephton, Consultant Medical Oncologist, on 01935 384869
or via email: matthew.sephoton@ydh.nhs.uk

To Apply
Please contact Carol Gill, Medical Recruitment Officer, on 01935 384502
or via email: carol.gill@ydh.nhs.uk.

We welcome you to submit a copy of your CV to apply for the post. Alternatively please contact Carol to request a copy of the job description and enhanced benefits package associated to this vacancy or to arrange a visit to meet our Oncology team.
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Exhibition stand: 37

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Exhibition stand: 29

The Advanced Data Analysis Centre (ADAC) was established in 2012 in the University of Nottingham to provide a vital infrastructure to undertake advanced data analysis in multi-disciplinary contexts. ADAC offers skilled data analysis expertise, across a range of subject areas and application contexts.

www.nottingham.ac.uk/adac

Contact: Philip Quinlan, Chief Technical Officer
Email: adac@nottingham.ac.uk
Phone: +44 (0)115 951 6359
School of Veterinary Medicine and Science, University of Nottingham, Sutton Bonington Campus, College Road, Sutton Bonington, LE12 5RD

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AstraZeneca is a global, innovation-driven biopharmaceutical business that focuses on the discovery, development and commercialisation of prescription medicines, primarily for the treatment of cardiovascular, metabolic, respiratory, inflammation, autoimmune, oncology, infection and neuroscience diseases. Medimmune is the worldwide biologics research and development arm of AstraZeneca. AstraZeneca’s innovative medicines are used by millions of patients worldwide.

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1 Francis Crick Avenue, Cambridge Biomedical Campus, Cambridge, CB2 0AA

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**Bayer Plc**

Exhibition stand: 32

Bayer is committed to delivering science for a better life. The oncology franchise includes three products and several other compounds in various stages of clinical development. These products reflect the company’s approach to research, which prioritises targets and pathways with the potential to impact the way that cancer is treated.

[www.bayer.co.uk](http://www.bayer.co.uk)
**Contact:** Jason Thompson
**Email:** Jason.thompson@bayer.com
**Phone:** +44 (0)7341 075144
Bayer House, Strawberry Hill, Newbury, Berkshire, RG14 1JA

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**Email:** Research@bodystat.com  
**Phone:** +44 (0)1624 629571  
PO Box 50, Douglas, Isle of Man, IM99 1DQ

**Bone Cancer Research Trust**

Exhibition Stand: 61

The Bone Cancer Research Trust is dedicated to saving lives and improving outcomes for people affected by primary bone cancer.

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www.bcrt.org.uk  
**Contact:** Zoe Davison  
**Email:** zoe.davison@bcrt.org.uk  
**Phone:** +44 (0)7876200945  
10 Feast Field, Horsforth, Leeds, LS18 4TJ

**Breast Cancer Now**

Exhibition Stand: 16

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www.breastcancernow.org  
**Contact:** Cheryl Lenny, Tissue Bank Liaison Manager  
**Email:** tissuebank@breastcancernow.org  
**Phone:** +44 (0)333 20 70 300  
5th Floor Ibex House, 42–47 Minories, London, EC3N 1DY
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www.b-ms.co.uk
Contact: Christina Cockley, Senior Medical Education Manager
Email: christina.cockley@bms.com
Phone: +44 (0)7753 976 705
Uxbridge Business Park, Sanderson Road, Uxbridge, UB8 1DH

British Association for Cancer Research (BACR)

Exhibition Stand: 11
The aim of the BACR is to promote the advance of research in relation to all aspects of cancer, both laboratory and clinical, and to encourage the exchange of information. Its functions are to organise scientific meetings and workshops; fund exchanges between laboratories to encourage knowledge transfer; provide opportunities for senior investigators to undergo further training; and to provide opportunities for junior investigators and research students to present their work at meetings and conferences.

www.bacr.org.uk
Contact: Janet Alexander, Administrative Secretary
Email: bacr@leeds.ac.uk
c/o Leeds Institute for Molecular Medicine, Clinical Sciences Building, St James’s University Hospital, Leeds, LS9 7TF

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Exhibition Stand: 22
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www.bioscience.co.uk
Contact: Mike Kerins, Managing Director
Email: support@bioscience.co.uk
Phone: +44 (0)1223 316 855
2-3 Munro House, Trafalgar Way, Bar Hill, Cambridge, CB23 8SQ
Cancer Clinical Trials Unit Scotland (CaCTUS)

Cancer Clinical Trials Unit Scotland (CaCTUS) is a partnership between the Cancer Research UK Clinical Trials Unit in Glasgow and the Scottish Clinical Trials Research Unit in Edinburgh. CaCTUS is a member of the NCRI Cancer CTU group and is a registered UK Clinical Research Collaboration CTU.

CaCTUS offers support for all aspects of management of clinical trials and is committed to working with Investigators to develop and manage clinical trials.

- Website: www.cactusonline.org.uk
- Contact: Julie Uttridge, Service Manager
- Email: NSS.SCTRU@nhs.net
- Phone: +44 (0)131 275 7061
- Address: SCTRU, Area 159e, 1st Floor Gyle Square, 1 South Gyle Crescent, Edinburgh, EH12 9EB

Cancer Research Technology

Cancer Research Technology is dedicated to advancing discoveries to beat cancer. We develop and commercialise exciting new discoveries in cancer research, working closely with leading clinical and academic institutions, pharmaceutical companies and biotechs worldwide. We’re uniquely placed to capitalise on the research and connections of our parent organisation Cancer Research UK.

- Website: www.cancertechnology.co.uk
- Contact: Rebecca Webb, Conference and Events Coordinator
- Email: enquiries@cancertechnology.com
- Phone: +44 (0)20 3469 6300
- Address: Angel Building, 407 St John Street, London, EC1V 4AD

Cancer Research UK

Cancer Research UK is the world's largest independent funder of cancer research, investing around £350 million every year across the entire research pipeline. We partner with a range of organisations, and support over 4,000 scientists, clinicians and nurses to meet our ambition of 3/4 people surviving cancer by 2034.

- Website: www.cancerresearchuk.org
- Email: Researcher.comments@cancer.org.uk
- Phone: +44 (0)20 7242 0200
- Address: Angel Building, 407 St John Street, London, EC1V 4AD
Cancer Research UK Cambridge Centre

Exhibition Stand: 33

The CRUK Cambridge Centre unites more than 600 laboratory and healthcare professionals around a common mission to end death and disease caused by cancer, through research, treatment and education. Our vision is to be the world leader in the development of ways to detect, monitor and cure cancer. As a CRUK Major Centre we serve as a national and international resource for patients with cancer and their families; researchers and health care providers; and cancer professionals in training.

www.cambridgecancercentre.org.uk
Contact: Katie Edwards, Communications Manager
Email: Katie.edwards@cruk.cam.ac.uk
Phone: +44 (0)1223 769 702

Cancer Research UK Cambridge Institute, University of Cambridge, Li Ka Shing Centre, Robinson Way, Cambridge, CB2 0RE

Cancer Research UK Glasgow Centre

Exhibition Stand: 49

The CRUK Glasgow Centre, a partnership between the University of Glasgow, CRUK Beatson Institute, NHS Greater Glasgow & Clyde and the University of Strathclyde, brings together scientists and clinicians to develop the very best in precision medicine, drug discovery and patient care.

www.glasgowcancer.org
Contact: Dr Jackie Beesley, Centre Manager, CRUK Glasgow Centre. Email: j.beesley@beatson.gla.ac.uk
Contact: Dr Liz Musgrove, Synergy Manager, CRUK Glasgow Centre. Email: Liz.Musgrove@glasgow.ac.uk
Phone: +44 (0)141 330 8722

Cancer Research UK Beatson Institute, Garscube Estate, Switchback Road, Glasgow, G61 1BD

Cancer Research UK Liverpool CTU

Exhibition Stand: 74

The Cancer Research UK (CR-UK) Liverpool Cancer Trials Unit (LCTU) was established in 1996. CR-UK funding commenced in 2006 to enable the LCTU to deliver high quality cancer trials to international regulatory standards aiming to change clinical practice.

The LCTU achieved full UK Clinical Research Collaboration (UKCRC) in 2007 and full re-registration in 2012 as part of the Liverpool Trials Collaborative.

www.lctu.org.uk
Contact: Lindy Martin
Email: lc当地@liv.ac.uk
Phone: +44 (0)151 7955293

Block C, Waterhouse Building, 1-3 Brownlow Street, Liverpool, L69 3GL
Cancer Research UK Manchester Centre

Exhibition Stand: 53

The Cancer Research UK Manchester Centre benefits from the strong partnership with The University of Manchester, including the Cancer Research UK Manchester Institute, and The Christie NHS Foundation Trust.

The Centre brings together the expertise, ambition and resources of its partner organisations in order to translate basic research into better diagnosis and treatment for cancer patients.

www.crukcentre.manchester.ac.uk
Contact: Dr Katy Holliday, Science Communications Officer
Email: crukcentre@mcrc.man.ac.uk
Phone: +44 (0)161 306 0800
The University of Manchester, Wilmslow Road, Manchester, M20 4QL

Cancer Research UK Oxford Centre

Exhibition Stand: 19

The Cancer Research UK Oxford Centre is a network and partnership between Oxford University, Oxford University Hospitals NHS Foundation Trust, and Cancer Research UK. As a CRUK Major Centre, Oxford pioneers comprehensive precision medicine; underpinned by interdisciplinary collaboration. We harness and translate our world-leading research to enhance cancer cure rates.

www.cancercentre.ox.ac.uk
Contact: Dr Michael Youdell, Centre Manager
Email: cancercentre@oncology.ox.ac.uk
Phone: +44 (0)1865 617043
Department of Oncology, Old Road Campus Research Building, Roosevelt Drive, Oxford, OX3 7DQ

Cancer Research UK-UCL Centre

Exhibition Stand: 85

The Cancer Research UK-UCL Centre brings together researchers and clinicians from UCL and its partner hospital trusts: UCLH NHS Foundation Trust, Great Ormond Street Hospital, Royal Free Hospital, Moorfields Eye Hospital, Royal National Orthopaedic Hospital and the National Hospital for Neurology and Neurosurgery.

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www.ucl.ac.uk/cancer/research/research-centres/cancer-research-uk-ucl-centre
Contact: Ieva Songailaite
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Phone: +44 (0)20 7679 6452
UCL Cancer Institute, Paul O’Gorman Building, 72 Huntley Street, London, WC1E 6DD
ChemoMetec

Exhibition Stand: 36

ChemoMetec develops high quality automated cell Counters and Analysers. Including aggregated cells, cells growing on microcarriers, Adipose Derived Stem Cells. Advanced analyses include plug-and-play assays for Apoptosis, Cell Cycle, GFP and more. Streamlining R&D and production work-flows for maximum efficiency. Used in fields from Cancer and Immunology to Cell Therapy.

www.chemometec.com
Contact: Ben Mantle
Email: bma@chemometec.com
Phone: +44 (0)7956 774 390
Sheraton House, Cambridge, Castle Park, CB3 0AX

Clearbridge BioMedics

Exhibition Stand: 82

Clearbridge BioMedics is a clinical stage oncology research and diagnostics company that enables real-time liquid biopsy using a label-free Circulating Tumour Cell (CTC) enrichment platform.

Headquartered in Singapore, Clearbridge BioMedics has customers spanning Asia, Europe and North America. The company has won numerous awards and garnered global recognition for the ClearCell® FX System.

www.clearbridgebiomedics.com
Contact: Ms Stacy Yu, Products Specialist
Email: contactus@clearbridgebiomedics.com
Phone: +65 6482 0668
81 Science Park Drive #02-03 The Chadwick, Singapore Science Park II, Singapore 118257

Colonis Pharma Limited

Exhibition Stand: 76

Mucodis products are used to help prevent and/or treat the side effects associated with cancer treatment. Improve patient experience through cancer treatment with Mucodis.

www.colonis.co.uk
Contact: Michael Dobson
Email: info@quantumpharma.co.uk
Phone: +44 (0)1892 739400
Niche Pharmaceutical Division of Quantum Pharma, Pinewood Chineham Business Park, Crockford Lane, Basingstoke, Hampshire, RG24 8AL
Dendrite Clinical Systems

Exhibition Stand: 56

Dendrite Clinical Systems is a specialist supplier of clinical databases, analysis software, consultancy and publishing services for the international healthcare sector. With over 22 years’ experience, the company’s global user base extends across hundreds of hospitals, with over 150 national and international databases, across more than 50 major countries.

[Contact Information]

Eurogentec

Exhibition Stand: 1

Eurogentec is a world-wide provider at the service of the scientists. The company supplies efficient qPCR kits and other genomics and proteomics related products. Eurogentec also manufactures GMP components intended to diagnostic and preclinical use. It also offers the dispensing of any in-house developed assay and commercial kit.

[Contact Information]

European Association for Cancer Research

Exhibition Stand: 38

The European Association for Cancer Research is Europe’s professional member association for cancer researchers with over 10,000 members in 101 countries. We provide a wide variety of services to our community of members, organise scientific meetings and courses of the highest quality, and facilitate communication and collaboration within the cancer research community.

[Contact Information]
Experimental Cancer Medicine Centre (ECMC) Network

The Experimental Cancer Medicine Centre (ECMC) Network is funded in partnership by Cancer Research UK (CRUK) and the Health Departments for England, Scotland, Wales and Northern Ireland. Through collaboration across the experimental cancer medicine community, the vision is to bring together laboratory and patient-based clinical research to speed up the development of better treatments for cancer patients.

www.ecmcnetwork.org.uk
Contact: Caitlin Hamilton
Email: Caitlin.hamilton@cancer.org.uk
Phone: +44 (0)203 469 6210
Angel Building, 407 St John Street, London, EC1V 4AD

FluidX

FluidX are Sample Storage and Tracking Specialists, with a focus on 2D barcoded storage. In October 2014, FluidX was acquired by Brooks Automation to help form a new Consumables and Instruments group within Brooks Life Science Systems, a Global leader in automated cold-chain management for drug discovery and biostorage applications.

www.fluidx.eu
Contact: James Singer
Email: James.singer@fluidx.eu
Phone: +44 (0)161 777 2000
Brooks Life Science Systems, Northbank, Irlam, Manchester, M44 5AY

GATC Biotech

GATC Biotech provides leading DNA and RNA sequencing services to more than 10,000 customers worldwide. The company’s innovative portfolio combines use of leading sequencing platforms with profound scientific expertise. GATC Biotech offers a large portfolio of oncology-related products, such as the world’s only research liquid biopsy service line, GATCLIQUID.

www.gatc-biotech.com
Contact: Customer Service
Email: customerservice@gatc-biotech.com
Phone: +44 (0)207 69 14 921
The London BioScience Innovation Centre, 2 Royal College Street, London, NW1 0NH
Exhibitor information (continued)

Greiner Bio-One

Exhibition Stand: 23 & 24
Greiner Bio-One is a leading manufacturer and direct supplier of high quality laboratory products to the scientific research community. Focusing on the evolving needs of our customers in the development of innovative solutions our unsurpassed range includes products for cell and tissue culture, including 3D cell culture solutions, biobanking, high throughput screening, immunology, microbiology, liquid handling, molecular biology and clinical sample collection.

www.gbo.com
Contact: Gary Kennerley, Sales Manager
Email: info@uk.gbo.com
Phone: +44 (0)1453 825255
Greiner Bio-One, Brunel Way, Stroudwater Business Park, Stonehouse, GL10 3SX

Horizon Discovery Ltd

Exhibition Stand: 9
Horizon Discovery combines deep scientific experience in translational research with a precision gene-editing platform incorporating rAAV, CRISPR and ZFN technologies. Horizon supplies genetically-defined cell lines, in vivo models, custom cell line generation, molecular reference standards, and contract research services to over 1,000 academic, clinical and biopharmaceutical organisations.

www.horizondiscovery.com
Contact: Amy Cowan, Events & Production Manager
Email: Amy.Cowan@horizondiscovery.com
Phone: +44 (0)1223 976 126
Cambridge Research Park, Waterbeach, Cambridge, CB25 9TL

Illumina

Exhibition Stand: 84
Illumina is improving human health by unlocking the power of the genome. Our focus on innovation has established us as the global leader in DNA sequencing and array-based technologies, serving customers in research, clinical and applied markets. Our products are used for applications in the life sciences, oncology, reproductive health, agriculture and other emerging segments.

www.illumina.com
Email: info@illumina.com
Chesterford Research Park, Little Chesterford, Saffron Walden, Essex CB10 1XL
King's Health Partners Comprehensive Cancer Centre

Exhibition Stand: 5

Research is embedded within King’s Health Partners cancer services, delivering the highest standard of care to our patients and collaborating with research partners to improve outcomes for future patients.

The Cancer Centre at Guy’s brings together radiotherapy, chemotherapy, outpatient clinics and support services under one roof for the first time.

www.gstt.nhs.uk/OHCT
Contact: Nick Gomm
Email: Nicholas.Gomm@gstt.nhs.uk
Phone: +44 (0)20 7188 1430
Guys and St Thomas’ NHS Foundation Trust, Guys Hospital, Great Maze Pond, London, SE1 9RT

Labcyte, Inc.

Exhibition Stand: 62

Labcyte Echo® liquid handling systems use sound to precisely transfer liquids without contact, eliminating the use of pipettes. Labcyte instruments are used throughout the pharmaceutical industry, biotechnology, contract research organisations, and academic institutions. Our customers work across a wide spectrum of scientific research, including drug discovery, genomics, proteomics, diagnostics and personalised medicine.

www.labcyte.com
Contact: Devin Donnelly, Account Manager, Ireland/UK
Email: info-europe@labcyte.com
Phone: +353 87 2140406
1190 Borregas Avenue, Sunnyvale, CA 94089, USA

LI-COR Bioscience

Exhibition Stand: 10

LI-COR provides the complete solution for quantitative and chemiluminescent Western blot imaging, and a variety of other applications including in vivo imaging with its Odyssey® Infrared Imaging Systems, C-DiGit® Blot Scanner, Pearl® Trilogy Imaging System, Image Studio® analysis software and IRDye® Infrared Dye-based antibodies and reagents.

www.licor.com/bio
Email: bio-eu@licor.com
Phone: +44 (0)1223 422104
St. John’s Innovation Centre, Cowley Road, Cambridge, CB4 0WS
Exhibitor information (continued)

Macmillan Cancer Support

Exhibition Stand: 20

Macmillan is a leading charity that seeks to reach and improve the lives of everyone living with cancer and to inspire others to do the same. Our research helps us to understand the numbers, needs and experiences of people affected by cancer.

www.macmillan.org.uk
Contact: Dr Hannah Pimperton, Academic Research Officer
Email: evidence@macmillan.org.uk
Phone: +44 (0)20 78407840
89 Albert Embankment, London, SE1 7UQ

Mallinckrodt Pharmaceuticals

Exhibition Stand: 45

Mallinckrodt Pharmaceuticals is focused on safely providing patients with nuclear medicine agents and other specialty pharmaceutical products, as we manage complexity and help to improve lives. Our products, including Octreoscan™ (Kit for the Preparation of Indium In-111 Pentetreotide) and Ultra-Technekow™ Tc-99m generators, offer significant benefits to nuclear medicine departments.

www.mallinckrodt.com
Contact: Lesa Carmichael, Sales & Operations Analyst
Email: lesa.carmichael@mallinckrodt.com
Phone: +44 (0)2392 704289
Perth House, Millennium Way, Chesterfield, Derbyshire, S41 8ND

Mammotome

Exhibition Stand: 2

Mammotome provides technology to help clinicians accurately diagnose breast disease, such as breast cancer, through minimally invasive procedures. As the global market leader in vacuum-assisted breast biopsy, Mammotome offers a wide range of products to help radiologists, surgeons and clinicians using stereotactic, ultrasound, magnetic resonance imaging, or molecular imaging guidance.

www.mammotome.com
Contact: Aude Kunick, Marketing Coordinator EMEA
Email: info.uk@mammotome.com
Phone: +44 (0)800 862 0587
Mammotome
Devicor Medical UK Lt, 20-22 Bedford Row, London, WC1R 4JS
Marie Curie

Exhibition Stand: 17
Marie Curie is the largest charitable funder of research in palliative and end-of-life care.
Visit our stand to learn more about what we do, and how we fund research to improve the quality and experience of care for people living with terminal illness, their carers and families.

www.maricurie.org.uk/research
Contact: Shefali Shah, Senior Research Information Officer
Email: Research.Info@mariecurie.org.uk
Phone: +44 (0)20 7091 4154
89 Albert Embankment, London SE1 7TP

MP Biomedicals

Exhibition Stand: 3
MP Biomedicals offers a line of more than 55,000 life science research and diagnostic products (in the field of Molecular Biology, Cell Biology, Immunology, Biochemicals, Rapid Diagnostic, EIA/RIA Diagnostic, etc...) that support academic and government research institutions as well as pharmaceutical and biotechnology companies.

www.mpbio.com
Contact: Dr Douglas Betts, Business Development Manager
Email: dbetts@mpbio.com
Phone: +44 (0)77 11 051 275
Rue Geiler de Kaysersberg, 67402 Illkirch, France

NanoString Technologies

Exhibition Stand: 54
NanoString Technologies provides life science tools for translational research and molecular diagnostic products. The company’s nCounter® Analysis System, which has been employed in basic and translational research, has also now been applied to diagnostic use as the nCounter DX Analysis System. The nCounter-based Prosigna™ Breast Cancer Prognostic Gene Signature Assay is FDA 510(k) cleared.

www.nanostring.com
www.prosigna.com
Contact: Claire McDonnell, Marketing Manager
Email: cmcdonnell@nanostring.com
Phone: +44 (0)7538 301 168
St Mary's Court, The Broadway, Amersham, HP7 0UT
National Cancer Registration and Analysis Service (NCRAS)

Exhibition Stand: 41

Through near real-time, comprehensive data collection, quality assurance and analysis, Public Health England’s National Cancer Registration and Analysis Service is building detailed picture of the cancer pathway. This rich data supports patient care, treatment and clinical research into the factors that affect cancer risk, early detection and effective treatment.

www.gov.uk/government/organisations/public-health-england

Contact: Dr Jem Rashbass, National Director for Disease Registration

Email: NCRASenquires@phe.gov.uk

Phone: +44 (0)20 7654 8158

NCRAS, Skipton House, 80 London Road, London, SE1 6LH

Newcastle University

Exhibition Stand: 72

Alongside campus-based degrees, Newcastle University offers online part-time postgraduate programmes for healthcare professionals. 30+ modules which include cancer related and molecular pathology subjects are ideal for CPD and revalidation. Combine modules for a PG Certificate, PG Diploma or MSc. Our long-established, interactive e-learning oncology and palliative care courses attract students from across the world.

www.ncl.ac.uk/medicalsciences-online/

Email: pgclinhealth@ncl.ac.uk, ncpall@ncl.ac.uk

Phone: +44 (0)191 208 7032

The Graduate School, Ridley Building 1 Level 3, Newcastle University, Newcastle Upon Tyne, NE1 7RU

Nexcelom BioScience Ltd

Exhibition Stand: 46

Nexcelom Bioscience is a designer, developer and manufacturer of innovative instruments for cell-based assays used in cancer research and drug discovery. With researchers critical input, Nexcelom's solutions automate time-consuming counting procedures for hundreds of different cell types, enabling scientists to focus more on the results and not on the process.

www.nexcelom.co.uk

Contact: Lori Fitton, EU Sales Director

Email: info@nexcelom.co.uk

Phone: +44 (0)161 232 4592

Unit 5, Greenheys Building, 10 Pencroft Way, MSP, Manchester, M15 6JJ
NIHR Clinical Research Network: Cancer

The NIHR Clinical Research Network provides the infrastructure necessary to undertake high quality clinical research in the NHS. We help researchers set up clinical studies efficiently, support the life-sciences industry to deliver research, provide research training, and work with patients to ensure their needs are integral to all research activity.

www.crn.nihr.ac.uk/cancer

Contact: Shamaila Anwar, Theme Manager for Cancer Surgery and Oral and Dental Health
Email: shamaila.anwar@nihr.ac.uk
Phone: +44 (0)113 343 6551
University of Leeds, Fairbairn House, 71-75 Clarendon Road, Leeds, LS2 9PH

North West Cancer Research Centre – University of Liverpool

The North West Cancer Research Centre – University of Liverpool (NWCR Centre-UoL) conducts innovative world-class research, aiming to reduce the impact cancers have across the Region. It strives to bring together cancer research teams across Liverpool, Bangor and Lancaster Universities, to better understand cancer biology, discover new therapies and improve patients’ survivorship.

www.liverpool.ac.uk/nwcrce/

Contact: Debbie Horne, horned@liverpool.ac.uk, 0151 794 9814
Contact: Dominique Hare, dominique@nwcr.org, 0151 709 2919
North West Cancer Research Centre, University of Liverpool, 200 London Road, Liverpool, L3 9TA

Oncology News

Oncology News is a high quality source of information covering as wide a spectrum of topics as possible. There are regular sections such as Conference reviews and previews, journal and book reviews, diary listings, web reviews, product information and most importantly lead articles covering the latest issues for the oncology profession.

www.oncologynews.biz

Contact: Patricia McDonnell, Publisher
Email: patricia@oncologynews.biz
Phone: +44 (0)288 289 7023
88 Camden Road, Dromore, Co Tyrone, BT78 3AT
Owise

Px HealthCare has developed the only validated, cancer patient-empowerment platform, which has demonstrated to improve the patient-physician relationship.

With the award-winning, mobile app OWise breast cancer, patients have access to a range of personalised medical tools to support them during the treatment and to regularly track & trace their wellbeing and side effects to treatments.

Contact: Anne Bruinvels  
Email: info@pxhealthcare.com  
Phone: +44 (0)20 7691 4926, +44 (0)77 6780 7865  
Px HealthCare, London Bioscience Innovation Centre, 2 Royal College Street, London, PNW1 0NH

Oxford Nanopore Technologies

Oxford Nanopore Technologies is developing and commercialising a new generation of nanopore-based electronic systems for analysis of single molecules, including DNA, RNA and proteins. The handheld MinION™ device and the high-throughput PromethION™ are designed to provide simplicity of workflows and real-time data streaming. Publications from MinION users can be found at https://publications.nanoporetech.com

www.nanoporetech.com  
Contact: Kim Cowan, Events Coordinator  
Email: kim.cowan@nanoporetech.com  
Phone: +44 (0)7557 430899  
Edmund Cartwright House, 4 Robert Robinson Avenue, Oxford Science Park, Oxford, OX4 4GA

Pancreatic Cancer UK

Pancreatic cancer is a tough one but we’re taking it on.

We are supporting those affected by the disease, investing in ground breaking research, lobbying for greater recognition of pancreatic cancer and being a voice for everyone involved in the fight.

Together we are taking on pancreatic cancer.

www.pancreaticcancer.org.uk  
Contact: Leanne Reynolds, Head of Research  
Email: enquiries@pancreaticcancer.org.uk  
Phone: +44 (0)20 7820 6705  
6th Floor Westminster Tower, 3 Albert Embankment, London, SE1 7SP
**Patient Journey App**

Exhibition Stand: 83

Our award-winning platform brings paper care plans into the 21st century!

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www.patientjourneyapp.com  
**Contact:** Guusje van der Heijden, Head of Customer Success  
**Email:** info@patientjourneyapp.com  
**Phone:** +31 20 26 16 429  
Singel 542, 1017 AZ Amsterdam, The Netherlands

**Promega UK Ltd**

Exhibition Stand: 39

With over 3,500 products in genomics, protein analysis and expression, cellular analysis, drug discovery and genetic identity, Promega is a global leader in providing innovative solutions and technical support to life scientists.

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www.promega.com  
**Email:** ukmarketing@promega.com  
**Phone:** +44 (0)23 8076 0225 Freephone: 0800 378994  
Delta House, Southampton Science Park, Enterprise Road, Southampton, SO16 7NS

**QIAGEN**

Exhibition Stand: 81

QIAGEN is the leading global provider of Sample to Insight solutions to transform biological materials into valuable molecular insights. Our mission is to make improvements in life possible by enabling our customers to achieve outstanding success and breakthroughs in life sciences research, applied testing, pharma and molecular diagnostics.

www.qiagen.com  
**Contact:** Customer Care  
**Email:** customercare-uk@qiagen.com  
**Phone:** +44 (0) 808 234 3665  
Skelton House, Lloyd Street North, Manchester, M15 6SH
Royal College of Pathologists and NCRI’s CM-Path Initiative

Exhibition Stand: 75

The Royal College of Pathologists (RCPath) is a professional membership organisation overseeing the training of pathologists and scientists. As part of National Pathology week (7-11 Nov), RCPath is promoting cellular molecular pathology (CMP) alongside the new NCRI initiative, CM-Path. RCPath has also invited CMP trainees with academic promise to attend and experience this major conference.

www.rcpath.org and www.ncri.org.uk/initiatives/pathology
Contact: Jessica Lee, CM-Path Programme Manager
Email: jessica.lee@ncri.org.uk
Phone: +44 (0)20 3469 8802
Royal College of Pathologists, 21 Prescot St, 4th Floor, London, E1 8BB

Servier Laboratories Ltd

Exhibition Stand: 57

Servier is an international pharmaceutical company, governed by a non-profit Foundation and headquartered in France. Servier's research is focused in five areas: oncology, cardiology, metabolism, neuropsychiatry and rheumatology. Being completely independent, Servier reinvests 25% of Servier's products turnover in Research and Development, and all its profits in its growth.

www.servier.co.uk
Email: info-uk@servier.com
Phone: +44 (0)1753 662744
Rowley, Wexham Springs, Framewood Road, Wexham, SL3 6PJ

St. Jude Children’s Research Hospital

Exhibition Stand: 15

A non-profit biomedical research institution where cutting-edge basic research is rapidly translated into ground-breaking treatments for cancer and other life-threatening diseases. We consistently rank on FORTUNE magazine’s “100 Best Companies to Work For” list. Visit our exhibition stand to discuss postdoctoral research opportunities.

www.stjude.org/pwostdoc
Email: postdoc@stjude.org
262 Danny Thomas Place Memphis, TN 38105 USA
**STRATEC Molecular GmbH**

Exhibition Stand: 7

STRATEC Molecular, established in 1992, develops and manufactures reagents and kits for DNA/RNA stabilisation and purification using manual and automated systems. The latest products are kits for automated extraction of cell-free DNA from liquid biopsy samples, quantification of the extracted DNA using qPCR and highly robust bisulfite conversion on cfDNA.

- **Website:** [www.stratec.com](http://www.stratec.com)
- **Contact:** Sonja Farhangi, Marketing Manager
- **Email:** info.berlin@stratec.com
- **Phone:** +49 (0)30 9489 2901
- Robert-Rössle-Str. 10, 13125 Berlin, Germany

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**Stratech Scientific Ltd**

Exhibition Stand: 80

Stratech supply over 1.8 million specialist life science research tools for researchers who need consistent, reproducible results. They have built an excellent reputation over 34 years for supplying high quality, competitively priced, reliable products. They are a family run business dedicated to delivering exceptional product quality with unbeatable technical support.

- **Website:** [www.stratech.co.uk](http://www.stratech.co.uk)
- **Contact:** Mark Aspinall-O’Dea
- **Email:** mark@stratech.co.uk
- **Phone:** +44 (0)1638 782600
- Unit 7, Acorn Business Centre, Oaks Drive, Newmarket, Suffolk, CB8 7SY

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**The Brain Tumour Charity**

Exhibition Stand: 51

The Brain Tumour Charity is at the forefront of the fight to defeat brain tumours, making a difference every day to the lives of people with a brain tumour and their families. We fund pioneering research to increase survival, raise awareness of the symptoms and effects of brain tumours and provide support for everyone affected to improve quality of life.

- **Website:** [www.thebraintumourcharity.org](http://www.thebraintumourcharity.org)
- **Contact:** Erica Moyes
- **Email:** erica.moyes@thebraintumourcharity.org
- **Phone:** +44 (0)1252 749 044
- Hartshead House, 61-65 Victoria Road, Farnborough, GU14 7PA
The College of Radiographers

Exhibition Stand: 6

The College of Radiographers is committed to developing the science and practice of radiography, radiotherapy and clinical imaging, including ultrasound, nuclear medicine and magnetic resonance imaging. We are keen to ensure that these services are centred around patients’ needs and very keen to engage with patients and the public. We promote study and research and make major contributions to health policy development in these fields.

www.sor.org
Contact: Spencer Goodman, Professional Officer
Email: info@sor.org
Phone: +44 (0)20 7740 7200
207 Providence Square, Mill Street, London, SE1 2EW

The Francis Crick Institute

Exhibition Stand: 21

The Francis Crick Institute is a biomedical discovery institute dedicated to understanding the scientific mechanisms of living things. Its work is helping to understand why disease develops and to find new ways to treat, diagnose and prevent illnesses such as cancer, heart disease, stroke, infections, and neurodegenerative diseases.

Contact: Esther Walker
Email: info@crick.ac.uk
Phone: +44 (0)20 3796 000
The Francis Crick Institute, 1 Midland Road, London, NW1 1AT

The Institute of Cancer Research

Exhibition Stand: 40

The Institute of Cancer Research, London, is one of the world’s most influential cancer research organisations. We are world leaders in isolating cancer-related genes and discovering new targeted drugs for personalised cancer treatment.

The ICR is a college of the University of London and ranks as the top academic research centre in the UK. We have charitable status and rely on support from partner organisations, charities, major donors and the public.

www.icr.ac.uk
Contact: Sophia McCully
Email: sophia.mccully@icr.ac.uk
Phone: +44 (0)20 7153 5136
123 Old Brompton Road, London, SW7 3RP
The Royal College of Radiologists

Through its Fellows and members, The Royal College of Radiologists maintains and develops the standards of education and practice in clinical oncology supporting doctors throughout their careers. Increasing the number of clinical oncology trainees is essential to the care of cancer patients and to build the next generation of doctors.

www.rcr.ac.uk
Email: enquiries@rcr.ac.uk
Phone: +44 (0)20 7405 1282
63 Lincoln’s Inn Fields, London, WC2A 3JW

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Email: uvp@uvp.co.uk
Phone: +44 (0)1223 420022
Trinity Hall Farm Estate, Nuffield Road, Cambridge, CB4 1TG

Univadis Oncology

Univadis is a free online medical reference trusted by over 3 million healthcare professionals worldwide

The Univadis Oncology program offers a wide range of evidence-based oncology information and decision-support tools, along with daily publications from leading publishers, such as The Lancet Oncology, JAMA Oncology, NEJM, JCO and more.

www.univadis.co.uk/oncology
Contact: Mr. Anthony Possuelos, Univadis Oncology Project Manager
Email: Anthony.Possuelos@univadis.com
Phone: +33 (0)1 7314 0074
Aptus Health, 5 Place de la Pyramide, 92800 Puteaux, France
Wales Cancer Partnership

Exhibition Stand: 52

The Wales Cancer Partnership brings together all cancer-focused organisations in Wales for the benefit of patients. Five of the member organisations are represented at the NCRI: European Cancer Stem Cell Research Institute, Cancer Research UK Cardiff Centre, the Wales Cancer Bank, the Wales Cancer Research Centre and the Wales Cancer Trials Unit.

www.walescancerpartnership.com/WCP

Contact: Jodie Bond
Email: WCP@Cardiff.ac.uk
Phone: +44 (0)2921 845850

Institute of Cancer and Genetics, Room 1TB2 31, Main Building, University Hospital of Wales, Cardiff, CF14 4XN

Warwick Clinical Trials Unit

Exhibition Stand: 35

Warwick Clinical Trials Unit (CTU) is a member of the NCRI Cancer CTU Group.

We specialise in the development and delivery of large multicentre clinical trials to inform clinical practice in the UK and throughout the world in a variety of clinical fields.

All cancer trials are NIHR adopted and seek to change clinical practice.

www.warwick.ac.uk/wms

Contact: Professor Janet Dunn
Email: j.a.dunn@warwick.ac.uk
Phone: +44 (0)2476 15117

Warwick Medical School, University of Warwick, Gibbet Hill Road, Coventry, CV4 7AL

Worldwide Cancer Research

Exhibition Stand: 27

Worldwide Cancer Research funds some of the most ambitious early-stage cancer research around the world and in 35 years has awarded almost £200 million in research grants to support some of the world’s best scientists across 34 different countries, supporting 1800 research projects.

Our horizons are broad and our vision bold but we believe we will, with the right research, see no life cut short by cancer.

www.worldwidecancerresearch.org

Contact: Debbie Wheelans, Grants & Information Manager (Post-Awards)
Email: debbiw@worldwidecancerresearch.org
Phone: +44 (0)1334 477910

Madras House, South Street, St Andrews, Fife, KY16 9EH
Adverse Event Management in Anaplastic Lymphoma Kinase-positive Non-small Cell Lung Cancer

Christian Rolfo, Ignacio Gil-Bazo

Antibody–Drug Conjugates in Relapsed/Refractory CD30-positive Lymphomas

Ulrich Jager and Martin Hutchings

Pomalidomide – An Appraisal of Its Clinical Development and Role in the Treatment of Relapsed/Refractory Multiple Myeloma

Paul G Richardson, Antonio Palumbo, Stephen A Schey, Meletios A Dimopoulos, Thierry Facon, Katja C Weisel, Peter O’Gorman, Xavier Leleu, Martha Q Lacy, Matthew J Streetly, Joseph R Mikhael, David S Siegel, Jesus F San Miguel and Kenneth C Anderson
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Acknowledgements

The NCRI Cancer Conference is a team effort involving many staff from the NCRI, NCRI Partners and contractors.

We would like to extend our thanks to all our suppliers whose involvement has been invaluable for the successful delivery of the Conference.

Thank you to all the NCRI Executive for their input and support throughout the year.
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Appendix 1 – NCRI Schools Event

Chair: Paula Chadwick, CEO, Roy Castle Lung Cancer Foundation

09.30 – 10.00
Registration and refreshments

10.00 – 10.05
Welcome address
Baroness Delyth Morgan, Chair of Trustees, National Cancer Research Institute

10.05 – 10.30
Setting the scene: What is cancer research? An overview
Sarah Blagden, Associate Professor of Experimental Cancer Medicine, University of Oxford Department of Oncology

10.30 – 10.45
Why we do it: The patient’s experience
Laura Haddad, Cancer patient

10.45 – 11.00
Q&A

11.00 – 11.15
Refreshment break

11.15 – 12.15
Quick-fire table discussions
Table hosts:
Lorna Fern, Health Services Researcher and Patient Engagement, National Cancer Research Institute
Angus McNair, Colorectal Surgeon, University of Bristol
Iain Phillips, Clinical Research Fellow, Royal Surrey County Hospital
Eleanor Gregson, PhD Student, MRC Cancer Unit, University of Cambridge
Christina Saville, PhD Student, University of Southampton

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12.15 – 12.30
Sharing thoughts

12.30 – 13.00
Lunch

13.00 – 13.30
How to prepare your statement for...
Tips and common pitfalls
Helen Brooks, Admissions and Access Administrator, Mansfield College, University of Oxford

13.30 – 14.00
Get your thoughts on paper
An exercise to get you going

14.00 – 14.15
Your ‘inspiration moment’ from today (in 140 characters)

14.15
Final remarks and close
Paula Chadwick, CEO, Roy Castle Lung Cancer Foundation
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