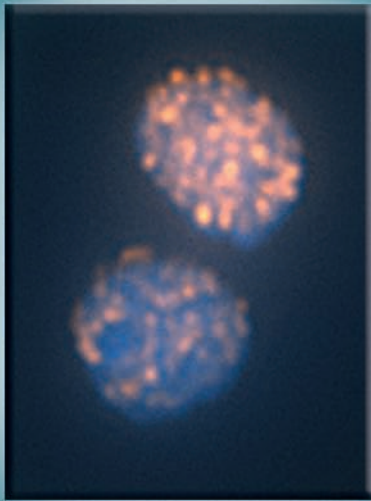


News

• British • Association • for • Cancer • Research •





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Front Cover

Measurement of phosphorylation of the histone variant, H2AX, is a useful biomarker of DNA damage in cells. Phosphorylation of H2AX (H2AX) occurs in response to agents like ionising radiation and chemotherapeutic drugs, in large chromatin domains flanking DNA double strand breaks. The resulting H2AX 'foci' can be detected by immunofluorescence microscopy. Elliott et al (Br J Haem 2011) used this technique to investigate the mechanism by which inhibition of DNA-dependent protein kinase (the enzyme that mediates non homologous end joining) sensitises chronic lymphocytic leukaemia (CLL) cells to the DNA damaging agent, mitoxantrone.

The inset on the cover shows nuclei from two CLL cells with H2AX foci as bright punctate dots visualised by immunofluorescence. The software used to analyse images can generate a 'surface plot' to show fluorescence intensity of the foci, which are shown as peaks in the main picture. This type of image analysis helped to confirm that DNA-PK inhibition enhances drug-induced cytotoxicity in CLL cells by increasing the persistence of drug-induced DNA double strand breaks.



BACR Executive Committee

2010/2011

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British Association for Cancer Research Meeting

‘Cancer Epigenetics’

Thursday 19th May 2011,
Royal Society of Medicine, 1 Wimpole St, London, W1

Topics include

Cancer Initiation, Environmental Exposures and Risk, Novel Epigenetic Technologies, Epigenetic Therapeutic Targets and Epigenome Profiling

Confirmed Speakers include:

Christoph Plass, DKFZ, Germany
Zdenko Herceg, IARC, France
James Flanagan, Imperial College
Stephan Beck, UCL Cancer Institute
Bryan Turner, University of Birmingham
Steve Clifford, NICR, Newcastle University
Nick La Thangue, University of Oxford
Eamonn Maher, University of Birmingham

Registration Opens: 1st January 2011
Abstract deadline: Friday 4th April 2011
Early bird Registration deadline 4th April 2011

Further details available from <http://www.bacr.org.uk>
e-mail: bacr@leeds.ac.uk

Scientific Organisers:
James M. Flanagan, Imperial College London
Adele Murrell, Cambridge Research Institute



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Letter from the Chairman



Dear Colleagues

Welcome to the spring 2011 Newsletter of the BACR.

The BACR in its 51st glorious year and continues to do what it does best: Providing platforms for cancer researchers to interact and to learn. In November 2010 we organised jointly with the RSM a one-day meeting in London on the “Development of cancer medicines: Modelling of disease and preclinical testing”. We had 82 delegates, and the meeting was considered by all attendants a great success.

This year we shall organize a small meeting on a very topical matter, cancer epigenetics, this will happen on 19th May at the RSM in London. More gatherings are planned for 2012 and 2013. In addition, as every year, we will make a substantial contribution to the NCRI meeting in Liverpool in November. The very positive feedback the committee received after our 50th anniversary bash in Edinburgh made us consider setting up a large 2-3-day meeting on a regular, perhaps 5-yearly, basis. Watch this space!

The committee wants to strengthen further the “BACR family feeling”. Thus we would like to encourage members to let us know of awards they received or major career developments they experienced, which may be of interest to the membership at large for inclusion in future newsletters.

Let me also encourage you again to proffer proposals for BACR workshops and meetings and to let us know how you want to be involved with their organisation. More on this topic inside this Newsletter.

Many young members were again able to benefit from BACR support to attend meetings – see inside this issue. We also include synopses of the very useful and informative BACR education workshops which we hosted at NCRI Liverpool in November 2010.

I am exceedingly pleased to be able to direct members’ attention to the “BACR History”, which will be shortly included in the BACR website for download. Special big thanks are due to Professor John Double, who invested a considerable amount of energy into the writing of the history. Several other individuals contributed to this endeavour, and I would like to thank particularly Dr Anne Simmonds-Walder and Professor Herbie Newell for their fantastic input.

I wish you success in your work and look forward to seeing you at some BACR meeting this year.

Best wishes

Andy Gescher
Chairman



We need your help!

The British Association for Cancer Research promotes the advance of research in relation to all aspects of cancer, both laboratory and clinical and to encourage the exchange of information. It achieves this aim by providing educational and training opportunities for all those involved in the cancer field, particularly the next generation of cancer research professionals. As you can see from the following pages one of the ways the BACR achieves its aims is by offering its members a variety of travel, meeting and career bursaries.

We understand with the Government's austerity measures money for travel and meeting attendance will become scarce. In light of this scenario members may help us to promote the BACR and spread the information on membership benefits. To facilitate this we are including in this newsletter a poster to promote the BACR which we would like you to place on your Notice Board. We would also welcome a link between the BACR website and that of your institute/organization. So we can implement this we would be grateful if you could supply the name of a contact in your centre that could facilitate this. It would also be helpful to the Secretariat if members from Universities could forward the names of the relevant Supervisors of students, research assistants and postdoctoral scientists in order that contact can be made and information sent direct.

To help with the exchange of information we would ask you to let us know what type of scientific meetings you would like to see. To do this you can either contact the BACR Secretariat on bacr@leeds.ac.uk with your suggestions or if you want to get more involved then see the following pages.

**Remember the BACR was set up to help you network
but this can only work if you get involved!**

£2 million for research into the causes of childhood cancer

CHILDREN with LEUKAEMIA has recently broadened its objectives to encompass all childhood cancers.

The progress that has been made in advancing understanding and treatment of childhood leukaemia hasn't necessarily been paralleled in other childhood cancers.

In addition, it is likely that different childhood cancers may have aetiological factors in common.

We are now calling for applications for funding for projects investigating the causes of any childhood cancer.

We are interested in the investigation of how mutations arise and in the basic biological mechanisms of childhood cancer development – and the impact of environmental factors on these mechanisms. We are particularly interested in the role of early exposures in the aetiology of childhood cancer including prenatal.

For further information on this grant round and how to apply, please refer to our website www.leukaemia.org

Want to organise a meeting or event with BACR?

1. Why it is attractive to work with BACR?

BACR has a history of facilitating the interchange of ideas among workers in all branches of cancer research and with a membership of over 1000 people you can see why the BACR is an attractive way of ensuring the right audience for an event.

The BACR is a platform in which original clinical and experimental data can be presented not only to members but to anyone who registers for an event. Also having links with other prestigious organisations such as EACR, NCRI and RSM increases the opportunities for networking and establish collaborations with leaders in cancer research from both academia and industry.

Not only does the BACR have great opportunities for networking and targeting colleagues in the cancer field it has a well established Secretariat who can help with the organisation and administration of your event from start to finish, providing you with excellent support and organisational infrastructure.

2. Things to consider when proposing an event

- What are the aims of the meeting
- Will it be a one day or two day event
- What is the anticipated target audience and how big is that audience
- Who will be on the organising committee
- Who do you recommend as Speakers
- What opportunities are there for raising sponsorship
- Should poster presentations or oral presentations from selected abstracts be incorporated
- When and where do you want to hold the meeting? It is worth allowing a minimum of 12 months for organising a one day event / 24 months for residential events, Also, try to avoid clashes with other BACR and major meetings.

3. How to proceed when making your suggestion.

- Complete the BACR Meeting/event proposal form (this can be downloaded from the BACR website at www.bacr.org.uk), taking into consideration points listed above in 2, and send it to the BACR Meeting & Training sub-committee care of Mrs Janet Alexander at the BACR office in LIMM, Leeds. If you are uncertain about some of the points Janet will be happy to advise. Don't forget to include your contact details.
- Your proposal will be reviewed by the BACR Meeting & Training sub-committee. If the committee view the proposal as potentially viable and in keeping with the aims and objectives of the BACR one of the Meeting and Training sub-committee members will contact you to discuss this further and support you and the organising committee in establishing a programme and budget.
- If the proposal is supported by the BACR Executive Officers then the BACR Administrative Secretary will support you and your organising committee throughout helping to promote your event, the search for a suitable venue and any other administration which you require in putting on a successful event.



BACR Special Conference

Development of Cancer Medicines

Royal Society of Medicine – 25th November 2010

Thursday 25th November 2010 saw the third BACR/RSM joint meeting held at the Royal Society of Medicine, London on preclinical cancer models and their role in informing the clinical development of anti-cancer agents.

Following a welcome and introduction from Sue Burchill (BACR Honorary Secretary from the Leeds Institute of Molecular Medicine) Val Brunton from the Edinburgh Cancer Research Centre chaired the morning session. This focused on the importance of the microenvironment and its impact on modelling the initiation and progression of cancer.

The first speaker was **Janine Erler** (Institute of Cancer Research, London) who talked about the ‘Importance of the tumour microenvironment in driving metastasis’. Her work focuses on the role of lysyl oxidase (LOX), which is an amine oxidase involved in crosslinking collagens and elastins in the extracellular matrix. Janine presented evidence that LOX plays an important role in promoting metastasis of solid tumours. In breast cancer and head and neck cancer LOX expression is associated with lower distant metastasis-free survival. Targeting LOX prevented the formation of metastases in mouse models and provides a potential target for preventing and treating metastasis. Manipulation of LOX expression in colorectal tumour cell lines revealed a role for Src kinase in LOX-mediated progression of colorectal cancer and the relationship between LOX expression and Src activation in these tumours may provide a mechanism to identify patients who will benefit from treatment with Src kinase inhibitors such as dasatinib.

Richard Marais (Institute of Cancer Research, London) then presented an overview of his work on ‘Developing models of melanoma’ and the use of BRAF inhibitors. The oncogenic BRAFV600E mutation is found commonly in a number of solid tumours but most frequently in melanoma. Despite a number of clinical trials with BRAF inhibitors, the clinical results have been disappointing. Richard described a pathway by which inhibition of BRAF in Ras mutant cancer cells leads to MEK hyperactivation through cRAF, which can drive tumorigenesis – thus highlighting the importance of fully understanding the pathways that are altered upon treatment with signalling inhibitors. He described the development of novel inhibitors which selectively target oncogenic BRAFV600E that are showing activity in early clinical trials with melanoma patients.

Last speaker before lunch was **Owen Sansom** (Beatson Institute for Cancer Research, Glasgow) who in his talk entitled ‘Using preclinical models of pancreatic cancer’ outlined the utility of a genetically engineered mouse model of pancreatic ductal adenocarcinoma (PDAC) as a preclinical drug development tool. There is much interest and debate around whether such models will be more predictive of efficacy in the clinical setting than conventional xenograft models. This particular model is driven by pancreatic specific mutations in KrasG12D and p53R172H, and as such mimics the major genetic alterations seen in the human disease. This model has been useful in dissecting the role of mutant p53 in tumour invasion and metastasis. Importantly the histology and disease progression also recapitulates the human disease. As a proof of concept Owen demonstrated that treatment with the Src inhibitor dasatinib could reduce the incidence of metastatic lesions in this model but had no effect on the growth of the primary tumour - suggesting that such agents may have utility in the adjuvant setting.

Lunch was then served, and people had an opportunity to view and discuss the 27 posters that were presented.

The afternoon kicked off with the Poster Discussion Session, chaired by Andreas Schatzlein (School of Pharmacy, London), in which six selected posters were presented and discussed. The first poster was presented by J. Boulton (Institute of Cancer Research, London) who reported on the development of *in vivo* imaging techniques for the characterisation of orthotopic models of high-grade human paediatric glioma. The tumour development was assessed using bioluminescence imaging and, in parallel, MRI imaging: a combination of dynamic contrast enhanced imaging, intravascular imaging agents (USIPOs) and changes in T1 and T2 weighted imaging modes were shown to correlate with a range of histopathological changes, including for example cell density changes and infiltration. The second presentation by E. Tan (Beatson Institute, Glasgow) described how the cooperation of APC and p53 loss may lead to the induction of invasion in a murine model of colorectal carcinoma. By using cre-lox technology the effect of p53 loss was investigated, either alone or in combination with Apc mutations. The group were able to show that only in combination were these mutations able to accelerate tumorigenesis, leading to a more invasive and metastatic phenotype. At the molecular level this suggests that in colorectal cancer Wnt-Myc signaling activation may occur in two stages whereby the first leads to adenomas and is followed by the transition to adenocarcinomas.

The use of three-dimensional invasion assays to predict drug response of breast cancers from individual patients was introduced by A. Leeper (University of Edinburgh). Biopsy material from ER positive tumours was embedded in rat collagen I gels and the response following exposure to tamoxifen monitored. The assay demonstrated a decreased residual tumour burden and reduced proliferation for the treated tumours, suggesting that in the absence of defined biomarkers this empirical approach may yield preliminary information about the response of individual tumours.

The utility of contrast enhanced high-frequency ultrasound (HFUS) for the characterisation of tumour vascularisation in preclinical tumour models was introduced by G. Marston from the University of Leeds. In the study it was possible to monitor changes in tumour blood flow and vascularisation in a model of colorectal cancer using micro-bubbles as contrast agent.

A. Scholz and colleagues from the University of Newcastle reported on the development of a liposomal siRNA delivery system and described how imaging techniques help to characterise its biological behaviour *in vitro* and *in vivo*. Using lipophilic fluorescent dyes that can be incorporated into the liposome and siRNA covalently modified with a second dye, they were able to monitor the distribution of the carriers and siRNA cargo *in vivo*. Their data suggested enrichment of these CD33 targeted (scFv anti-CD33) liposomes in the CD33 positive tumour sites.

The final poster in this session was presented by S. McKeown from the University of Ulster who had studied the effects of Bicalutamide on the microenvironment of androgen dependent prostate cancer. Specifically, it was shown in xenograft models that the antiandrogen treatment over time leads to selection, resulting in a more malignant phenotype, thus replicating the clinical observation of the emergence of more malignant androgen independent cancers after 18-24 months of treatment.

After tea the afternoon continued with a session chaired by Steve Wedge from AstraZeneca. In his talk "Preclinical utility of conditional mouse models of human breast cancer", Jos Jonkers (Netherlands Cancer Institute, Amsterdam) highlighted the power of using genetically modified cells and animal strains to provide translational insights and develop clinically relevant hypotheses. In this presentation he described screening a library of known drugs against a BRCA-2-deficient mouse mammary tumour cell line and its isogenic counterpart with BRCA-2 gene reconstitution. In addition to carboplatin, three bifunctional alkylating agents (chlorambucil, melphalan and nimustine) were found to have selectivity for the killing of BRCA-2-deficient cells. Single agent alkylator activity was confirmed *in vivo* in BRCA-2-deficient tumour cell xenografts, grown orthotopically by cell line inoculation or by implantation of tumour fragments obtained from K14cre;Brca2F11/F11;p53F2-10;F2-10 mice. Historically, chlorambucil and melphalan were tested in breast cancer patients but did not outperform the standard of care in an unselected population. These preclinical studies suggest that bifunctional alkylators could have clinical utility in breast cancer patients with BRCA-2 defects, and potentially also in sporadic breast cancer patients with a homologous recombination deficiency.

Marie-France Poupon (Institut Curie, Paris, France) then presented on her experience with “The development and use of human tumour models derived from fresh surgical specimens”. These have gained recent popularity, because of their ability to provide a more diverse range of in vivo models that may better reflect the range of molecular pathology within a given histological type of tumour. In addition, they generally exhibit more complex structural features, including greater stromal involvement, than xenografts derived from in vitro passaged tumour cell lines. The success rate of establishing such models is dependent upon the tumour type, with pancreatic and colorectal tumours being relatively easy to establish and models from malignancies in hormonally regulated tissues (breast and prostate) being significantly more challenging. Marie-France described work in which 304 surgical samples of breast cancer had been implanted into mice and only 34 specimens found to generate reproducible xenograft models. The majority of these were triple negative. Analysis of tumours by aCGH, mRNA profiling or immunohistochemistry showed individual models to resemble the original corresponding surgical specimen. Encouragingly, a breast tumour model, derived from a patient who was subsequently treated with a number of different cancer therapies, was found to demonstrate a corresponding sensitivity or resistance to treatment.

The final speaker of the day was **Steve Wedge** (Oncology iMed, AstraZeneca, Macclesfield) who gave an overview of the different “Strategies for preclinical testing”, and in doing so aimed to address the predictive value of preclinical models in relation to the clinic. This was largely focussed on the use of human tumour cell line panels and cell-line derived human tumour xenografts. Steve argued that for targets where there is a strong monogenic drive, such as for EGFR mutations in NSCLC or BRAF mutations in melanoma, these preclinical platforms can translate well to the clinic for inhibitors with a defined selectivity profile. The models are also useful for developing therapy responsive biomarkers or transcriptional pathway signatures that can be explored clinically. However, Steve also highlighted some of the challenges in accurately modelling other aspects of human cancer, including the consequences of inhibiting targets within the tumour stroma and recapitulating acquired resistance to therapy. He concluded by emphasising that a more detailed knowledge of human clinical disease is required, in addition to new preclinical models, to optimally position novel therapies. At the end of Steve’s talk, there was a lively discussion session chaired by Steve and Sue.

The programme was designed to maximise opportunities for dialogue and interaction, the poster discussion and end-of-day discussions proving very popular.

Thank you to everyone who helped organise, presented at and attended what was an excellent and informative meeting. Thank you also to the sponsors of the meeting; AstraZeneca, Abcam, Caliper Life Science Ltd, PerkinElmer and Visual Sonics.

We hope to hold a fourth meeting in November 2012. Maybe see you there. ☺.

BACR Educational Workshops

NCRI 2010

BT Conventional Centre, Liverpool

“Opening the vaults of industry” – Discovery Science in Pharma

Hosted by Steve Wedge (Senior Principal Scientist, AstraZeneca, Alderley Park, UK) There is often a lack of appreciation of the breadth of scientific activities conducted within the pharmaceutical sector and what it is like to work there. Discovery scientists in industry have to be innovative, maintain an awareness of emerging science, and frequently work within multi-disciplinary teams or collaboratively with researchers in academia and other companies. The pursuit of new therapeutic strategies that will deliver benefit to cancer patients involves many intellectual and technical challenges that include:

- The identification and preclinical validation of new targets in cancer
- The discovery of small molecule inhibitors against targets with the desired properties to become candidate drugs for testing in man
- The development of novel biomarker strategies to assess target modulation in man and assist early dose-ranging studies
- The prospective molecular characterisation of patients that are most likely to respond to a given treatment
- The identification of novel combination paradigms to augment treatment outcome

This session provided a brief insight into the roles required to address these challenges.

Short presentations were given from scientists working at different stages of the drug discovery process, describing their transition into industry, and their current activities and perspectives.

This was a rare opportunity to gain additional information on the workings of a large Pharma cancer discovery department and to ask questions in an interactive session.

“Drug Metabolism in Cancer Drug Discovery”

Exploiting Drug Metabolism in Cancer Drug Discovery

Hosted by Klaus Pors, Institute of Cancer Therapeutics, Bradford

Objectives of Part I of the BACR educational workshop:

- (i) To provide an overview of CYP450 in cancer drug metabolism
- (ii) To illustrate how CYP450 metabolism potentially can be exploited in Cancer Drug Discovery.

The drug metabolising enzymes (DMEs) are a diverse group of proteins that are responsible for metabolising a vast array of xenobiotic compounds including cancer drugs. This workshop focused on one class of DMEs, the cytochromes P450 (CYPs), a superfamily of mixed function oxidases that are unique in their ability to primarily oxidize xenobiotics but which under stressed conditions such as hypoxia also are capable of reducing certain chemical functionalities. The presence of certain CYPs may reflect a resistance mechanism by diminishing the pharmacological activity of anticancer drugs whilst specific CYPs can also modulate cell proliferation by the formation or conversion of endogenous signalling molecules. The potential for CYP-selective metabolism of xenobiotics coupled to their broad substrate specificity provides a unique opportunity to design drugs whose activity is dependent on a critical functional group that can be unmasked or restored by CYP metabolism selectively in tumour tissue.

Investigation of Drug Metabolism by Cytochromes P450 in Phase I Studies

Hosted by Alan V. Boddy, NICR Newcastle

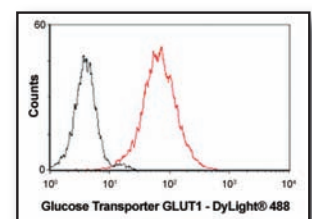
Objectives of Part II of the BACR educational workshop:

- (i) To provide an overview of the technology available to study drug metabolism and pharmacokinetics: i.e. HPLC, mass spectrometry, UV spectroscopy.
- (ii) To illustrate some approaches to the identification of metabolites, including potential problems and pitfalls.

Identification of metabolites at an early stage of drug development can be important, especially if metabolism is extensive, if the metabolites are toxic or active, or if there is the potential for drug interactions or significant interpatient variability. Some information can be obtained from pre-clinical data, and a number of tools are available to extrapolate from in vitro incubations to predict the nature and extent of metabolism. Despite this, the relevant metabolic pathways and degree of metabolism may not become clear before the drug has been administered to patients for the first time. In a phase I study, although the numbers of patients are limited, there is an opportunity to obtain information on drug metabolism, across a wider range of doses than will be experienced in later studies. Using a combination of CYP expression systems, MS-based analysis and careful interpretation of data from the clinical trial it is possible to characterize the metabolic pathways of a new drug.



Shopping for cancer research reagents?



Glucose Transporter GLUT1 antibody (ab40084) - Flow Cytometry
Overlay histogram showing HeLa cells stained with ab40084 (red line). The secondary antibody used was DyLight® 488 goat anti-mouse IgG (H+L) (ab96879). DyLight® is a trademark of Thermo Fisher Scientific Inc. and its subsidiaries.

More information at www.abcam.com/DyLight

Abcam are leaders in protein detection and regulation and are able to deliver a comprehensive portfolio of all the very best and most up to date cancer antibodies and related products. A leading online provider of antibodies and proteins to researchers worldwide, Abcam's diverse catalog of over 70,000 products includes among others cutting edge tools for stem cells, immunology, chromatin and nuclear signaling as well as cancer research.

Visit www.abcam.com/cancer for more information

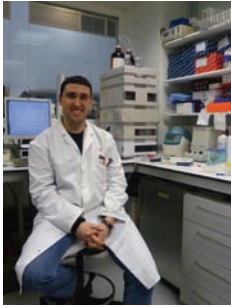


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Travel Bursaries

40th Annual meeting of the European Environmental Mutagen Society (EEMS)

15th – 18th September 2010, Oslo, Norway



Hamza Hamouchene

*Molecular Carcinogenesis.
Institute of Cancer Research.
Sutton.*

I am very grateful to BACR for awarding me a student bursary to attend the 40th Annual meeting of the European Environmental Mutagen Society (EEMS) held in Oslo, Norway between 15th and 18th of September 2010.

My research is in the field of environmental carcinogenesis. I am investigating the influences of cell cycle kinetics on responses of human cells to a widespread environmental pollutant and carcinogen: Benzo[a]pyrene. The project involves different analysis including: gene expression profiling, DNA damage analysis, and DNA repair to mention a few. The EEMS meeting was a natural choice to increase my knowledge in the field and an opportunity to discuss my work with great scientists.

The conference was excellent and had a rich scientific program, which was very relevant to my field of expertise. It covered many topics including: Environment and Genotoxic Stress, Inflammation, nutritional genotoxicology, DNA repair, and DNA damage response. I enjoyed being at all sessions, especially the two later themes, which I am currently investigating in my PhD project. The DNA repair session was a joint symposium with the Society for Free Radical Research (Europe) annual meeting and was an opportunity for members of the two societies to learn from each other.

The conference organisers managed to invite outstanding scientists who are at the forefront of their fields. The talks were scientifically engaging, and so were the poster sessions where I had the chance to present my work and get interesting feedback from other scientists. The conference brought scientists from all over the world and the atmosphere was informal, so I got to know and befriend other people easily. It was really a joy to listen to eminent scientists the like of Eugenia Dogliotti, Penny Jeggo, and Paolo Vineis.

Apart from the exciting learning experience I had during the meeting, I enjoyed exploring the beautiful city of Oslo with all its great attractions. Again, thank you BACR for this tremendous opportunity, and I am sure that I will use the obtained knowledge in my future work.



Julian Laubenthal

University of Bradford

Division of Biomedical Science

(the photo shows Julian in front of the Norwegian parliament in Oslo city centre, 3min from the meeting venue).

The BACR travel fellowship enabled me to attend the 40th Annual meeting of the European Environmental Mutagen Society (EEMS) in Oslo, Norway from the 15th to 18th September 2010. This annual conference is the most important European meeting in the field of environmental carcinogenesis, teratogenesis and mutagenesis and consequently the leading European researchers in this field presented their work.

I currently investigate for my PhD programme the contribution of the male germ line in the development of childhood cancers as well as possible differences in susceptibilities to environmental mutagens/carcinogens between newborns.

This programme is funded by European Union's "NewGeneris: Newborns and Genotoxic exposure risk" project. Hence, the session "Cancer susceptibility with focus on children" in which Jos Kleinjans (Maastricht University), Dan Segerbäck (Karolinska Institute) and Kari Hemminki (German Cancer Center) showed findings of their work within the NewGeneris project was very interesting for me. They presented interesting data on the special vulnerability of children compared to adults as well as novel genetic and environmental risk factors of childhood cancers such as childhood leukaemia. However, I was most impressed of the key lecture given by Yuri Dubrova (Leicester University) "From mutation induction to transgenerational genomic instability in mammals" within the session "Male germ cell mutagenesis". Dr Dubrova is considered as one of the giants in the field of reproductive mutagenesis and I never had the opportunity before to listen to him (and later to ask questions by myself). Dr Dubrova spoke about his hypothesis, that ongoing genomic instability in cancer patients, particularly in chemotherapy survivors, can predispose the offspring of those patients to severe genetic disease, including childhood cancers. Dr Dubrova presented methods to detect these germ-line mutations, such as single-molecule PCR, which he already used successfully for the identification of transgenerational germ-line mutations in children of radioactive exposed parents from the Chernobyl accident.

Overall, the meeting lasted over three days and a total of 80 lectures were given, which were accompanied by many panel discussions, award lectures and two poster sessions.

4th Breast Cancer Symposium

1st – 3rd October 2010, National Harbor, Washington DC, USA



Voralak Vichapat

*Division of Cancer Studies,
Cancer Epidemiology Group
King's College London*

The British Association for Cancer Research gave me a great opportunity to attend the 4th Breast Cancer Symposium from 1-3 October 2010 at National Harbor, Washington DC, United States. This high standard meeting specifically hosted discussion of current information and developments related to breast cancer research and treatments, along with allowing clinicians and researchers from many disciplines to present their ongoing work.

The first day started with a general session on the new modality of breast cancer prevention and risk, chaired by Dr. Swati Kulkarni from Roswell Park Cancer Institute. At the end of this session, I listened to a talk on 'Predictors of contralateral breast cancer in BRCA 1 and BRCA2 mutation carriers' which was a research topic of particular interest to me as it is related to my current PhD project. The whole session gave me an idea of how cancer research in the context of epidemiological studies will develop in the future by taking into account new trends in breast cancer detection and molecular genetic studies. On the same day, I also attended the session specific to breast cancer metastases and was fascinated by the insights given by Dr. Patricia S. Steeg from the National Cancer Institute that, during decade when the treatment of breast cancer has been highly successful and the prognosis of women with breast cancer has been prolonged to more than ten years, brain metastasis is not as uncommon as a recurrent event as it used to be. This piece of information caught my attention as a prompt to investigate more closely the metastatic patterns in a group of women with breast cancer who subsequently developed a second breast cancer, which I am currently involved.

In the morning of the second day, I attended the Networking Breakfast section which gave me an extra opportunity to build up a network of professional contacts and share different experiences with other junior scientists. The later sessions were mostly discussions of current controversial issues regarding the treatment of early breast cancer, for example, controversies in radiation therapy. Issues related to patients' well-being and complications after the treatment of breast cancer, such as physical activity, diet, weight, psycho-social impact, sexual behaviour and breast cancer in the older were also discussed. In the late afternoon, I presented my poster, on behalf of Cancer Epidemiology Group, School of Medicine, King's College London and Cancer Research UK, on the topic of 'Prognosis of metachronous contralateral breast cancer: importance of stage, age and interval time between the two diagnoses' and it was nominated for a merit award. It was also discussed in the poster discussion session later that evening by Dr. Richard L. White, a surgeon from Carolinas Medical Center. My last day at this symposium ended with a forum on the prospective reform of health care policy in the United States. All of these talks gave me not only a chance to hear an expert interpretation and translation of data into a clinical context, but also a whole new perspective on the way clinical practice is going and how cancer research could contribute to better treatment and care.

From my sincere wish that all people who are suffering from cancer might be well again, I hope that the knowledge I have gained from this meeting might contribute towards helping patients with breast cancer through my continuing work on understanding the mechanisms involved in this disease.

5th International Congress of the GRS and IGF Society

3rd – 7th October, New York, USA



Li Zeng

*IGF & Metabolic Endocrinology group,
University of Bristol*

The Fifth International Congress of the GRS and IGF Society was held from 3rd to 7th of October 2010 in New York, USA. My abstract was selected for an oral presentation and was entitled 'Involvement of IGFBP-3 in the growth inhibitory effects of AZA on MDA-MB-231 breast cancer cells'. With £800 awarded from the British Association for Cancer Research (BACR) and other funding from other organisations I was able to afford

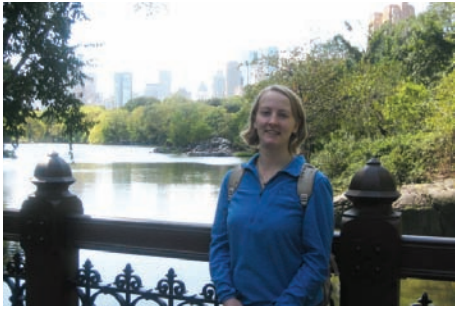
registration, Visa application, travel and accommodation expenses.

It was a very good chance for me to meet top researchers in this field from the USA, Germany, Australia, Israel, Sweden, Denmark, France, Japan, Spain and China, etc.

The meeting was started with an opening lecture given by Prof. Sinclair from Harvard Medical School, Cambridge, USA. His title is 'Longevity genes: parlor trick or path to drugs that revolutionize medicine'. In this interesting talk, he suggested SIRT3 as a key regulator of the mitochondrial transition pore (MTP) and an important factor in aging related diseases.

The following days were arranged into plenary sessions, oral sessions and poster sessions. Each session on different days also had different topics, they were roughly divided into growth hormone actions or IGF related research works, since people attending the meeting had relatively different specificities and interest. As my PhD study is about nutrition epigenetic changes in relation to breast cancer, especially how IGF axis plays a role between nutrition and cancers, I'm very interested in talks and posters related to IGFs and human metabolism, IGFs in physiology and disease, IGF binding proteins and cancer. There were many good talks from which I learned a lot of new aspects of cancer and IGFs. For example, Dr. Lee from University of Pittsburgh held a talk 'Targeting the IGF pathway in breast cancer'. He introduced IGFI receptor (IGFIR) could be predictive marker for breast cancer prognosis and targeting IGFIR using IGF blocking antibodies or IGFIR tyrosine kinase inhibitors could be useful tool in breast cancer treatment; the talk of Dr. Korbons from the UK introduced a unique hormone called ghrelin. As a hunger signal and a signal of presence of dietary calories, it has effects on cardiac function, liver metabolism, adipose tissue, gut motility, Langerhans islet cell regulation, memory, cognitive ability, sleep, anxiety, thermoregulation, immune function as well as cell proliferation; in Dr. Lazzarino's talk, he indicated that IGF signalling is required for the maintenance and renewal of stem cells in the mammary gland development. There were also many interesting works in the postern presentations. Dr Durfort from France showed that silencing of IGFIR and IGFI by RNA interference may offer a new clinical approach for treatment of breast tumours expressing IGFIR or IGFI; Dr. Foulstone from our group showed a poster 'IGFBP-2 acts as a survival factor breast cancer cells' which showed addition of IGFBP-2 decreased paclitaxel induced cell death in MCF-7 and T47D cells and conversely silencing IGFBP-2 would significantly enhance the chemotherapy-induced apoptosis.

The meeting was very successful and very informative for me. It gave me the opportunity to learn about all the most recent findings in the field. It also gave me chance to talk to many researchers from around the world about our work, which provided me with useful suggestions for the future studies. Following my presentation, I thanked you for your sponsorship, both verbally and by showing your logo on my final slide. I'd like to thank the BACR again for supporting my attendance at this meeting.



Claire Worrall

*Karolinska Institutet and University Hospital
Dept of Oncology & Pathology
Stockholm*

As my first conference as a post-doctoral researcher, the Fifth International Congress of the GRS and IGF Society in New York in October 2010 was a lovely introduction into the field of IGF-1 biology, in which I am now studying cancer. My PhD project focused on a small family of secreted proteins dysregulated in gastric cancer, so I had made a substantial change

in field to IGF-1R signaling in cancer! This meant that the broad range of talks introduced many new aspects of the area to me, which were not directly linked to my new group's research into the cross-talk between IGF-1R and GPCR pathways, but painted a bigger picture. For example, although the structure of the related Insulin Receptor is known, the structure of the IGF-1R, a protein critical in the progression of so many cancer types, is not yet fully resolved, although it was clear a lot of progress is being made on certain domains. As we are studying the involvement of specific residues on the interaction with different proteins this was helpful to put in context our work and fuel our debates about the relative interactions of proteins.

At the opposite end of the basic to translational research spectrum, there were talks about the progress of clinical trials on drugs that target the IGF-1R pathway in cancer. These showed me that progress was being made but there was still room for further understanding of how they actually affect the pathway and how cross reactivity to the Insulin Receptor is being minimized. It stimulated our thoughts on how combinations of drugs with different target proteins that we are investigating could improve the efficacy of treatments and how we could investigate this further within our research.

The time outside of talks gave me the chance to meet and get to know many of our group's collaborators, and allowed us to develop our ideas about work to be done during the planned exchange visits, the first of which will happen in spring this year and I have no doubt we made more progress than a week of e-mails would have! It also gave us the valuable opportunity to suggest a new collaboration with a group that has made a very similar discovery to us but in a slightly different context, ideally so that through communication in the next few months we can exchange ideas and work on complementary areas.

I enjoyed the opportunity to design and present a poster for the meeting and got some useful feedback from other researchers in the field and valuable practice at explaining our work to others. Compared to my experience during my PhD of the 100th AACR meeting in Denver, this meeting was relatively small, appropriate to the size of field and clearly a very welcoming place for early stage researchers to have the opportunity to talk. This means that my aim for the next meeting, planned for Munich in 2012, will be to give a talk on my current work on the mechanisms and effects of ubiquitination on the IGF-1R and I am looking forward to it.

Of course, while I was in New York, I could not pass up the opportunity to see a little of such a fascinating city, including some relaxing time walking in the sun along the High Line, trying some of a colleague's favourite home food in China town and a refreshingly cool evening visit with my group to see the Manhattan views from the top of the Empire State building! For all of this I am thankful the BACR travel grant allowed me to attend.

15th International p53 Workshop

8th – 12th October, Philadelphia PA, USA



Ee Hong Tan

*Beatson Institute for Cancer Research
Glasgow*

The 15th International p53 Workshop was held in Philadelphia in Oct 2010 with the specific aim of bringing together premier researchers in the fields of p53, cell death, oncology and drug discovery. I was very fortunate to be awarded the BACR travel fellowship which enabled me to attend this exciting meeting.

The meeting was span over 5 days, with the discoverers of the p53 suppressor gene, Dr. Arnold Levine and Sir David Lane presenting the opening keynote addresses. The talks that followed were split into themes which included:

- p53 biology
- Drug discovery
- Protein modifications
- Cell death pathways
- Animal models of Cancer

I am currently a post doctoral scientist at the Beatson Institute for Cancer Research working on the novel colorectal mouse models of APC and p53 mutations. I had the privilege of presenting my research findings in the poster session. Besides the high quality talks at the meeting, this meeting has also allowed me to discuss my research with many fellow scientists, providing me with valuable criticism and feedback on my research. As this meeting was attended by more than 300 participants, it has also allowed me to network and form useful collaborations.

In summary, the new and important unpublished basic, translation and clinical research findings presented at the meeting has inspired me with new ideas to bring my research forward. I thank the BACR once again for awarding me the travel fellowship which covered part of the cost for attending this meeting.

AACR Special Conference – Colorectal Cancer Biology to Therapy

27th – 30th October 2010, Philadelphia, USA



Helena Smart

*CRUK Colorectal Tumour Biology Group
University of Bristol*

The BACR Meeting Bursary that I was awarded last summer enabled me to attend the AACR Special Conference entitled: “Colorectal Cancer: Biology to Therapy” in Philadelphia, 27-30th October 2010.

This was an exceptional gathering of leaders in the field of colorectal cancer research and a fantastic opportunity to get up-to-date on the latest developments, both in basic research and in the clinical field. In particular it was invaluable to hear the two main leaders in my field of research describe their latest, unpublished work concerning the roles of prostaglandins in colorectal neoplasia, a particularly topical area considering recent media attention on the anti-cancer effects of aspirin, and great to finally have a chance to question them in person about their findings.

Moreover, this meeting provided a very timely opportunity to present my new data, providing useful feedback prior to publication and allowing me to establish new and useful contacts within our field of interest which are likely to lead to at least one new collaboration in the exciting area of organoid culture.

Finally this meeting was also a great opportunity to learn the latest developments within the field in general, including a novel approach to targeting dormant, drug-resistant cancer stem cells and a new stool DNA test (for methylated genes TPFI2, BMP3, NDRG4 & total DNA) which could help many people avoid colonoscopies and subsequently generated considerable press interest. In addition to enhancing my research, this opportunity is likely to benefit the rest of the lab as well through passing on both the latest findings in the field but also through the new methodologies we plan to introduce to the lab through our new collaborations.

BT Convention Centre, Liverpool, UK



cancer conference

ncri

national cancer research institute



NCRI Cancer Conference 6 - 9 November 2011

Plenary speakers

- Michael Hall (Switzerland)
- Michael Stratton (UK)
- Hans Clevers (The Netherlands)
- Maria Blasco (Spain)
- Harald zur Hausen (Germany)
- Jeffrey Settleman (USA)
- Murray Brennan (USA)
- Sir Mike Richards (UK)
- Eva Grunfeld (Canada)
- John Potter (USA)



The Conference is the major forum in the UK for showcasing the **best British and international cancer research**, bringing together the **leading experts across all disciplines** with a compelling mix of **high-quality plenary speakers, symposia and parallel sessions**, including **focused satellite meetings and workshops**.

Important dates for the 2011 NCRI Cancer Conference

- Abstract submission opens: **Monday 21 March**
- Abstract submission deadline: **Friday 13 May**
- Registration opens: **Wednesday 1 June**
- Late breaking abstract submission opens: **Monday 11 July**
- Earlybird registration deadline: **Monday 1 August**
- Late breaking abstract deadline: **Wednesday 31 August**
- Online registration deadline: **Friday 30 September**
- NCRI Cancer Conference commences: **Sunday 6 November**

Also featuring symposia on

<ul style="list-style-type: none"> Cancer screening and prevention Hosted by Robert Steele Epithelial mesenchymal transition Hosted by Nicholas Hastie Living with and beyond cancer Hosted by Peter Selby / Julia Brown Predictive models of human cancer Hosted by David Tuveson 	<ul style="list-style-type: none"> Metabolism and cancer Hosted by Eyal Gottlieb The diagnostic and therapeutic potential of the tumour microenvironment Hosted by Thorsten Hagemann Epigenetics and cancer Hosted by Peter Adams Stratified medicine Hosted by Alan Ashworth
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National Cancer Research Institute (NCRI) 2010

7th – 9th November, BT Convention Centre, Liverpool

Lynne Howells

*Research Fellow,
Department of Cancer Studies and Molecular Medicine,
University of Leicester, UK.*

This award allowed me to attend the National Cancer Research Institute (NCRI) 2010 annual meeting in Liverpool, the true home of football (alas, now somewhat ailing, but as I write this the season is yet young!!). The NCRI is the foremost UK-based cancer conference, which allows dissemination of research findings for researchers, physicians and the lay public across a wide variety of cancer sites, from basic science through to clinical endpoints.

The Department of Cancer Studies and Molecular Medicine at the University of Leicester is part of the CRUK/ Department of health-sponsored Experimental Cancer Medicine Network, which is responsible for the delivery of early-phase clinical trials for new drugs and drug combinations to our local populace for a variety of malignancies. The NCRI provides an excellent platform by which to showcase every aspect of the ECMC, and provides useful and informative sessions allowing dissemination of research, ECMC aspirations and best practice.

One area of my specific research interests was particularly well represented this year. Within our group, we aim to develop cancer stem cell assays and translate them to clinically relevant applications, including tumour characterisation with respect to stem cell-like composition, and assays that elucidate affects of therapeutic or chemopreventive intervention on these stem-like populations. Attending the meeting allowed me to interact with my peers within the field, and to discuss trials and tribulations of this particular area of research in addition to identifying potential collaborators and new avenues of research.

Research seminars in the cancer stem cell field were of excellent quality (particularly the keynote speakers), and provided valuable insight into the advancements made within this field internationally. Aside from these specific research interests, there were very informative presentations from those involved in the MRC COIN trial, as well as on subjects diverse from my normal sphere of interest. Particularly pertinent were those at the ‘patient orientated’ end of the spectrum, including end of life care issues. I believe that as a scientist, it is sometimes all too easy to become devolved from the real reasons behind our research. Conferences such as the NCRI present the opportunity for the scientific community to engage with both clinicians and consumers to focus strategies that may have a real chance to ultimately improve or increase the therapeutic options available where they are needed most.

Now on to the alternative ‘networking’ opportunities provided during the conference dinner in the setting of the magnificent St Georges Hall – if only the dancing matched the setting! Anyway, a good time was had by the younger student representatives, the more ‘mature’ professorial complement and, ahem, middle-aged research fellows alike... Thank you BACR.

22nd EORTC-NCI-AACR Symposium on Molecular Targets and Cancer Therapeutics

16th-19th November 2010, Berlin



Chris Morrow

*Clinical and Experimental Pharmacology,
Paterson Institute for Cancer Research,
University of Manchester,*

The 22nd EORTC-NCI-AACR symposium on Molecular Targets and Cancer Therapeutics took place in Berlin on the 16th-19th November 2010. Thanks to the kind funding of the BACR I was able to attend and present my recent work investigating the effect of PI3 kinase inhibition on the ability of the BH-3 mimetic drug ABT-737 to induce apoptosis in colorectal cell lines. The conference attracted a mixed audience of clinicians, academic scientists and pharmaceutical companies from around the world to discuss and present the latest advances in the field of oncology drug discovery.

The main focus of the meeting was the progress novel anti-cancer agents are making, both preclinically and clinically, potential patient stratification options and rational drug combinations. Numerous early stage clinical trials with PI3 kinase inhibitors were reported and, whilst toxicity appeared to be acceptable there was little evidence of consistent tumour response when used as a single agent (although the trials reported were not designed to detect this). Other novel agents reported which were only at the preclinical stages of development included acid sphingomyelinase inhibitors, which Marja Jäättelä reported destabilised lysosomes leading to cancer cell death, and the AMPK activator metformin, with Pam Goodwin summarising the evidence for the use of this diabetic drug in the cancer setting. An intriguing different take on the same phenomena came from David Lane and Michael Kastan. Both reported that the p53 wild-type status of normal tissue in patients with p53 mutant tumours could be harnessed to reduce toxic side effect of cytotoxic agents. Prof. Lane demonstrated that activating p53 in the normal tissue lead to cell cycle arrest and reduced toxicity from cytotoxic agents that only target proliferating cells. Conversely, Prof. Kastan presented data supporting the argument that inhibition of p53 in normal tissue would protect from toxicity associated with p53 activation, although the two possibilities are certainly not mutually exclusive and both offer interesting rational drug combination strategies for future development. The topic of rational drug combinations was also covered in a session hosted by James Doroshow and Maurizio Scaltriti, with the overwhelming conclusion that for a drug combination to have a good chance at making it in the clinical setting there must be a strong rational for combining the two agents, ideally with preclinical work backing it up. All in all, this was an excellent meeting that really highlighted the relevant issues involved in modern oncology drug development.

Androgens 2010

25th-26th November 2010, Leuven, Belgium



Greg Brooke

*Department of Surgery and Cancer
Division of Oncology
Imperial College London*

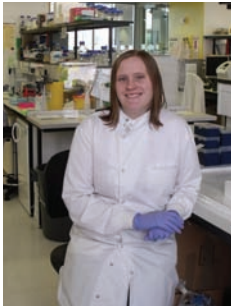
I am very thankful to the British Association of Cancer Research for enabling me to attend and present my work at the Androgens 2010 conference, held in the beautiful city of Leuven, Belgium. The biennial Androgens conferences are a highlight in the calendar for all researchers in the field of androgen signalling and have been running for 10 years. We arrived in Leuven the day before the conference and checked in to our accommodation, the aptly named Hotel Professor! We popped out briefly to sample some of the culinary delights of Belgium before retiring for the evening to get in some much needed rest before the early start of the conference the next day.

The androgens conference covered a wide range of androgen receptor functions, including its roles in development and cancer. The talks were of a very high standard and hence it is difficult to pick out a few highlights here. For me, however, Professor Karen Knudsen's (Thomas Jefferson University, USA) talk was excellent. Professor Knudsen's work beautifully demonstrates the role of Rb in the control of androgen signalling and prostate cancer progression. Also of note, was the talk by Professor Michael Stallcup (University of Southern California) who described the work that his team has been doing to define the order of protein complex formation at the regulation regions of target genes. Finally I would also like to mention the work of Professor Adriaan Houtsmuller (Erasmus, Holland) who has eloquently used FRET and FRAP technology to define the order of intra- and inter-molecular events leading to the androgen receptor binding to DNA and promoting transcription.

I was honoured to have been picked by the organizing committee to present my recent findings on the role of the RNA binding protein FUS in androgen receptor signalling and prostate cancer growth. Presenting my work was extremely useful as I came away from the conference with some useful suggestions and potential collaborations. I left Androgens 2010 inspired and with a hunger to return to my bench and test out new hypotheses that I had formulated during the conference.

51st Annual Meeting of the American Society of Haematology

4th – 7th December, Orlando, Florida, USA



Victoria Forster

*Northern Institute for Cancer Research,
Newcastle University.*

I would like to convey my gratitude and thanks to the BACR for providing me with a travel award to attend the 51st annual meeting of the American Society of Haematology in Orlando, Florida in early December 2010. I am currently a third-year PhD student at the Northern Institute of Cancer Research, Newcastle University working under the supervision of Dr. James Allan and Dr. Olaf Heidenreich and my project focuses on the leukaemia fusion gene AML1/ETO and how it mediates cellular response to mutagenic agents.

Originally I was destined to fly out from Gatwick airport, but after two days of snow delays, I finally managed to get on a flight from Manchester to Orlando, after a 500 mile tour of the UK from Newcastle-London-Manchester! As the first major international conference I had attended, it was quite an experience being one of around 20,000 haematologists, clinicians and scientists in one conference centre and some of the lecture theatres and exhibition spaces were gigantic.

The conference focuses on a very wide range of haematological disorders from cancers such as leukaemias and myeloma, to thalassaemias and even therapeutic strategies for combating HIV infection. Needless to say there was plenty to keep me busy and I was fortunate enough to attend some excellent presentations; many on areas that I was previously unfamiliar with such as microRNA regulation in leukaemias, which gave me new ideas for my project.

The best part of the conference for me personally was attending the poster sessions. Being a haematology-specific event, I found that there were a lot of posters relevant to my project, almost too many to read in the time available! Being able to speak to the presenters in such a relaxed and informal setting was outstanding for meeting new people and trading ideas and thoughts on new and in-progress research. I also presented a poster at the meeting and it was a very rewarding experience speaking to people from all over the world about my work. I made some excellent contacts and received some very productive suggestions and feedback on how to proceed with my PhD project.



Dr Evan Mulligan

*Newcastle Cancer Centre at the Northern Institute for Cancer Research
Newcastle University*

Thanks to being awarded a travel bursary from the British Association of Cancer Research I was able to attend the 52nd Annual meeting and exposition of the American Society of Haematology at the Orange County Convention Centre December 4-7th 2010.

The conference was an excellent meeting and brought together thousands of scientists and clinicians from across the world. I gave my presentation in one of five poster sessions dedicated to novel aspects of therapy for Chronic Lymphocytic Leukaemia (CLL). My work highlights the importance of all five of the subunits of the stress inducible transcription factor NF- κ B in the prognosis and progression of CLL. Through the use of small molecule inhibitors of DNA repair enzymes we have shown that improved chemosensitivity to agents such as fludarabine and mitoxantrone is due not only to increased DNA damage, but through an inhibition of the protective NF- κ B response.

The meeting provided excellent opportunities to attend “meet the expert sessions” and to receive the latest updates on clinical trials and new areas of CLL therapy. Many of the sessions that I attended focused upon the

importance of the tumour microenvironment to the development and therapy resistance of CLL. I was also particularly interested in the work of Asish Ghosh from the Mayo Clinic on the receptor tyrosine kinase Axl. Their work showed the specificity of activated Axl expression to CLL cells and that inhibition of the receptor provided a unique specific target for CLL therapy.

I would like to thank the BACR for assisting me to attend the meeting and I greatly valued the opportunity to meet and discuss my work with a wide range of scientists and clinicians, the trip provided me with many new ideas to take forward into my next section of research.



Dr Joanna Zabkiewicz

*Experimental Cancer Medicine Centre,
Department of Haematology,
University of Wales Hospital Cardiff*

I would first like to give my thanks to the BACR for funding me to attend this prestigious annual meeting for haematologists. Set in a stadium sized arena and attracting more than 20,000 delegates of clinical, scientific and business backgrounds, the scale of this conference is vast and requires comfortable walking shoes, or as some delegates preferred, a segway to zip between talks.

The purpose of this annual meeting is to allow the exchange of scientific and clinical results in all aspects of haematology and as such the meeting was timetabled into educational, scientific, career development and networking sessions.

I am currently involved in a translational project establishing new therapeutic targets in Acute Myeloid Leukaemia (AML). Despite gradual improvements in treatment outcomes, most patients with AML still die from their disease. Treatment with conventional cytotoxic agents seems to have reached its limits and advances in the understanding of the complex and highly heterogeneous molecular mechanisms underlying AML have fuelled a drive towards targeted therapy. I presented a poster on my recent work regarding the role and therapeutic potential of survival kinase PDK1 in AML. Although the initial scale of this conference is daunting, it was particularly useful for me to get a wider overview of the hot topics within the field. The educational programme gave a good grounding in basic haematology and extended my knowledge of other haematological disorders.

I was particularly impressed with the lecture of Tsvee Lapidot (Weizmann institute, Israel). His special lecture to a capacity audience was televised on 5 huge screens, yet he delivered an engaging and fascinating overview into recent progress in the dynamic regulation of haemopoietic stem cell migration and homing. He highlighted the importance of preclinical transplantation NOD/SCID models in determining the interplay of neurotransmitters, cytokines, adhesion molecules and stromal progenitors in mediating stress signals. This knowledge will be beneficial in providing better success rates with transplantation or leukaemic stem cell mobilization therapies.

Another key topic discussed was the contribution of oxidative stress to leukaemogenesis. Several interesting talks implicated reactive oxygen species (ROS) in cell migration and drug responsiveness; although it is clear further investigation is required to elucidate these complex signalling pathways. Of note was Steve Baylin's lecture on polycomb markers in cancer and the contribution of ROS to epigenetic modifications.

Finally, an interesting talk by Elaine Willmore described the success of DNA-PK inhibitors in targeting overactive DNA repair mechanisms in drug resistant leukaemia patients. This conference provided an excellent opportunity to expand my knowledge and gather inspiration for future grant applications. I presented my findings prior to publication and gained valuable feedback from peers and potential collaborators. I am grateful to BACR for making this possible.



Sarah Fordham

*Northern Institute for Cancer Research
Newcastle University*

With the valuable help of a BACR Travel Award, I was able to attend and present my work at the 52nd Annual Meeting of the American Society of Haematology (ASH) held in Orlando, Florida in December 2010. This 4-day international conference is the largest in the field of haematology and aims to facilitate the exchange of current scientific research and clinical findings in leukaemia, lymphoma and myeloma, as well as other non-malignant haematological disorders. I am a PhD student working in the field of acute myeloid

leukaemia, and my work is aimed at understanding the role DNA repair defects play in the aetiology and pathology of this disease. The opportunity to be involved in such a prestigious conference, attended by many leading researchers and clinicians from my research area was invaluable, especially as DNA repair defects in haematological malignancy was one of the key themes.

During the first poster session of the conference, I presented work in which I identified a role for DNA mismatch repair in the response to certain key anti-leukaemic drugs. This is a novel finding and as such I received a lot of interest and gained valuable feedback from many peers. I am currently in the writing-up stage of my PhD, and the opportunity to present and discuss my work at this point in my studies was especially valuable as it generated a number of ideas for my thesis and publications. Following my poster presentation, I was fortunate to attend an intimate Education Spotlight Session specifically focussed on DNA repair and how it can be exploited by novel therapeutic approaches. This session was chaired by two worldrenowned DNA repair experts and constituted a significant highlight of the meeting for me.

As well as the many talks, lectures, symposia and poster presentations which formed the main backbone of the meeting, ASH also included a Trainee Day specifically for researchers at the early stages of their career. The day involved several presentations covering effective research communication, job negotiation and how to secure funding, as well as careerdevelopment lunch sessions which allowed smaller groups of trainees to meet with leaders in the haematology field. I found this opportunity to speak with experienced researchers very rewarding in terms of the encouragement and support I was given for my future professional development. In addition to the Trainee Day, a trainee lounge was available throughout the meeting, providing an informal environment (with free tea and coffee!) in which to meet and share experiences with people at a similar stage as I am. Through this, I was able to establish work-related connections, but also make some new friends whom I hope to stay in touch with.

Following completion of my PhD, I intend to continue my research in the field of DNA repair and its deregulation in leukaemia. I would like to relocate to the USA during the early stages of my post-doctoral career, which was another reason I was keen to attend the ASH meeting this year. Through my attendance, I was privileged to be able to meet with group members from major US research laboratories, as well as other labs around the world, and have established some contacts which I hope will lead to future job opportunities!

All in all, I gained a lot from the ASH annual meeting and wouldn't hesitate to recommend it to anyone working in the field of haematology, especially students and early career researchers. I would like to thank the BACR for making my attendance possible.



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33rd Annual San Antonio Breast Cancer Symposium

8th – 12th December, Texas, USA



Dr Ciara O'Brien

*Paterson Institute for Cancer Research
Manchester University*

I am a medical oncology trainee in the final year of a PhD in Oncology based at the Paterson Institute of Research, University of Manchester. Becoming a recipient of a BACR travel fellowship enabled me to present some of the key data from my PhD in the international setting of the San Antonio Breast Cancer Symposium, Texas, USA, in December 2010.

My PhD investigates the role of breast cancer initiating cells (BCICs), also known as cancer stem cells in the response of estrogen receptor positive (ER+) breast cancers to tamoxifen treatment and in the acquisition of tamoxifen resistance. My data demonstrates that tamoxifen preferentially targets more differentiated ER+ cells within ER+ breast cancers while estrogen receptor negative (ER-) BCICs appear relatively insensitive to tamoxifen therapy. Furthermore the acquisition of tamoxifen resistance leads to an enrichment of BCIC activity. Therefore my work implicates BCICs in a novel mechanism of tamoxifen resistance.

For this work I have used a range of established cell lines and importantly a significant number of early and metastatic ER+ breast cancer samples with the aim of accurately reflecting the clinical spectrum of ER+ breast cancer. To carry out BCIC assays in the gold standard in vivo setting, a necessary part of my work was to develop a novel, reproducible xenograft model of ER+ breast cancer derived from patient samples, which had been historically difficult to achieve.

As a medical doctor with a strong interest in ongoing breast cancer research the opportunity to travel to the San Antonio Breast Cancer Symposium using a BACR Travel Fellowship was invaluable. This conference melds cutting edge basic, translational and clinical science research in a format that promotes learning, discussion and collaboration between research groups. The opportunity to participate in small seminars with leading figures in breast cancer research fields is rare and has really helped me as I now prepare my PhD thesis and revise for my viva exam. Furthermore the facility to attend poster sessions and discuss methodology and early data with fellow researchers was hugely helpful.

The selection of my submitted abstract for an oral presentation during the general session of an international breast cancer conference was a surprise and may indeed be a once in a lifetime experience! The considerable expense of travelling to Texas and finding accommodation for the 6 days of the conference was significantly assisted by the kind allocation of a BACR Travel Fellowship. Nothing can really prepare you for the surreal experience of presenting your work to 8000 people and seeing your face and research data being amplified on huge screens dotted around a conference facility the size of an aircraft hangar. The nerves were overwhelming but constant refinement of my talk and meticulous preparation allowed it to go ahead without any significant hitches. Furthermore the opportunity to discuss and defend my work in the question and answer session which followed my talk in which I was quizzed by international experts in the field of my research was invaluable. Not only will this experience become a useful addition to my CV but it will also stand me in good stead in many aspects of my future career.

Genes and Cancer Conference

13th to 15th December 2010, Warwick University



Luisa Robbez-Masson

*Supervisor: Dr Richard Grose
Bart's Cancer Institute, Centre for Tumour Biology,
Queen Mary University*

This year the 27th edition of the Genes and Cancer meeting took place in Warwick University from the 13th to the 15th of December 2010. This meeting allowed some 140 delegates to hear about the latest advances in cell biology and cancer research from an outstanding international line-up of speakers. As a second-year PhD student, this was a really valuable opportunity to hear about some of the latest research and meet some great speakers.

Following opening remarks from Richard Marais, chair of the organising committee, the three days were divided into four parts; Genome Integrity; Cell growth and proliferation; Cancer frontiers and Signalling. The first one, Genome Integrity, covered of telomere biology, DNA repair and cell cycle and was my favourite session. The keynote lecture, sponsored by Breakthrough Breast Cancer, was given by Prof Gregg Semenza (Baltimore), a pioneer in the study of Hypoxia. He emphasised how adaptation of cancer cells to their microenvironment is an important driving force of clonal selection and has an impact on invasive and metastatic tumours. In this scenario, the Hypoxia-inducible factor 1 (HIF-1) that he discovered is a key player. The second day saw many people, me included, present their work at a stimulating poster session, complete with plenty of refreshment to encourage scientific debate! The opportunity to discuss my research allowed me to receive lots of helpful feedback and insight from fellow attendees.

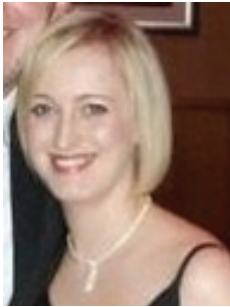
The highlight of the second day for me was Gerard Evan's presentation on Myc and how important targeting this key transcription factor could be for cancer therapeutics (Cancer frontiers session). Alongside talks about current research in cancer cell biology, there was a captivating talk about immunology by Dr Facundo Batista (CRUK) that concluded the meeting. He described how new high resolution imaging methods developed in his laboratory could help investigate the molecular events that occur during B-cell activation and identify the role of macrophages in adaptative immunity as antigen presenting cells.

The conference dinner, on the second evening, was followed by the infamous Genes and Cancer Ceilidh, where we were entertained by the same band that has been playing at this meeting for over 20 years now. It was hugely enjoyable although very demanding physically!

This was the first Genes and Cancer meeting I have attended and I was thrilled by the range of speakers and topics. The cross-disciplinary nature of the conference gave me the opportunity to learn more about several aspects of cancer research that are quite far from the main focus of my PhD. This, I think, helps to give perspective on my research project, something that is easy to lose. I am indebted to the BACR for their generous award of a Travel grant, which allowed me to attend this meeting.

Keystone Symposia: Histone code: fact or fiction

10th – 15th January 2011, Midway, Utah, USA



Kelly Armstrong

*Northern Institute for Cancer Research
Newcastle University*

In January of this year, a travel fellowship from the BACR allowed me to attend the Keystone Symposia entitled 'Histone code: Fact or Fiction' at Zermatt resort, Midway, Utah. The aim of this meeting was to bring together a variety of researchers from different fields who are interested in the posttranslational modification (PTM) of histone proteins to highlight the many viewpoints currently held relating to the effect of PTMs on the structure and reading of the genome.

My research is based on histone modifying enzymes and their potential activities towards non-histone proteins specifically in prostate cancer development and progression. I presented a poster at this meeting which was well received by other attendees. I gained some useful insights and suggestions to help my research including several pertinent ideas from non-cancer related fields. In particular, I was exposed to lots of novel, unpublished techniques that are being established in the histone field.

The meeting was initiated with a keynote session where Bryan Turner and David Allis presented their key findings in this field, highlighting the many important interactions and downstream consequences of histone modifications and chromatin structure. This review perfectly set the scene for the rest of the conference.

At this meeting there were 7 sessions of lectures and 2 workshops where 'hot topics' were presented and discussed. I particularly enjoyed Karolin Luger's talk where she discussed models of nucleosome structure highlighting that there are several structural states which may be in flux with one another. In addition I found the work of Michael Poirier to be very interesting. He discussed his research into nucleosome dynamics and how PTMs at the DNA-histone interface can influence the amount of energy required to unwrap DNA from the nucleosome. He also highlighted how this process plays a role in many functional processes, including replication and DNA repair.

In total, 149 posters were presented over 3 evening sessions, some of which were also presented as short talks. As these sessions were dominated by unpublished findings they were very interactive and thought provoking. During one of the sessions I viewed a poster presented by Alejandra Loyola discussing the functions and shuttling of the cytosolic histone H3/H4 complex. I had very little understanding in this area and Dr. Loyola was very happy to explain at length exactly what her findings were. These poster sessions provided a fantastic platform to interact with and learn about the research of other laboratories all over the world; an excellent forum to develop new collaborations to help you in your research and future career.

This meeting was rounded off with a debate relating to whether the histone code actually exists. A very lively and passionate discussion was held with many varying issues and points raised by many individuals at every level including internationally renowned field leaders to PhD students who had just begun their studies. This is one of the most beneficial aspects of attending any Keystone meeting; attendees, regardless of experience are always encouraged to share their opinions and theories and interact with each other at every level. Attending this conference also gave me the opportunity to meet with a collaborator, Judd Rice, from USC. Judd and I have been collaborating for about a year via email prior to this meeting so it was very beneficial to our working relationship to actually meet face to face for the first time.

In summary, I would highly recommend a Keystone Symposia meeting to any scientist regardless of experience. During my 10 years in science I have found that these meeting are definitely the most beneficial and exciting ones to attend!

BACR Mid-Career Fellowship

Visit to China



Dr. Andrew D. Westwell

Welsh School of Pharmacy, Cardiff University

Positron Emission Tomography (PET) is a leading non-invasive imaging technique for cancer diagnosis, staging and monitoring of response to therapeutic intervention, combining high resolution with exquisite sensitivity. One of the major new areas of research interest within my group concerns the chemical synthesis of radiolabelled cancer drugs/biomarkers for PET imaging. The relatively short half-lives of positron-emitting radionuclides (e.g. ^{18}F , 110 minutes) impose considerable restrictions on

the preparative route for PET biomarkers, and present a major challenge to synthetic medicinal chemists.

We have established new projects aimed at developing novel routes to fluorinated nucleoside-based biomarkers, using chemistries that can be adapted to the preparation of ^{18}F -radiolabelled molecules for PET imaging. This work is being carried out in collaboration with Cardiff colleagues with expertise in nucleoside chemistry (Prof. Chris McGuigan), microwave chemistry (Dr. Mark Bagley) and the newly established PET imaging centre at Cardiff University (www.cardiff.ac.uk/petic; scientific director Dr. Stephen Daniels). During November 2010, I undertook a research visit to China, supported by a mid-career fellowship from BACR. My trip involved visits to two research groups with chemical expertise in this area, in order to forge new collaborations, facilitate knowledge exchange and provide a means to allow excellent Chinese students to come to the UK to further their knowledge and experience.

I first visited the research group of Prof. Shende Jiang at the Tianjin University School of Pharmaceutical Sciences and Technology. Here I was able to spend time meeting with Prof. Jiang and his research team (see picture), discussing science and giving lectures to students within the University on aspects of medicinal chemistry and drug discovery. Importantly I had the opportunity to tour the clinical PET Centre at Tianjin University Medical Centre, which gave me a useful perspective on the practice of PET imaging in China, and the enormous impact that this relatively new technology is having worldwide.

Following my time in Tianjin, I took an overnight train to Shanghai, to visit the research group of Prof. Feng-Ling Qing and colleagues at the Organofluorine Research Centre of the Shanghai Institute of Organic Chemistry. The scale of activity at the Shanghai Institute was truly impressive, and it is clear that this highly productive research institute will go from strength to strength in contributing to the development of chemistry-based research in China. I am immensely grateful to BACR for giving me this opportunity to visit this fascinating and diverse country, to take our science to a new audience and see first hand the enormous growth in scientific research within China. The scale of investment, development and growth in both the cities I visited (Tianjin and Shanghai) was breathtaking, and the kind hospitality I received from my hosts was second to none. This was hopefully the first step of a memorable collaborative journey that I intend to continue in the future.

The BACR Hamilton-Fairley Poster Prize

Presented at the NCRI Liverpool 2010



Tine S Mantoni

*Grays Institute
Oxford*

Tumour-stellate cell interaction and radiation resistance in pancreatic cancer

Status: Pending

Tine Mantoni, Serena Lunardi, Thomas Brunner

Background: Pancreatic ductal adenocarcinoma (PDAC) is characterised by a strong desmoplastic reaction, which is believed to be a contributing factor to the poor therapeutic response of PDAC. A central mediator of desmoplasia is the pancreatic stellate cells (PSC). Signalling between PSC and tumour cells stimulates proliferation and migration of both cell types. We aim to gain a better understanding of the crosstalk between PSC and tumour cells and how this affects tumour cell response to ionising radiation.

Method: We have set up a clonogenic survival assay for tumour cells directly cocultured with PSC in order to assess the radiosensitivity of mono- versus cocultured tumour cells. We also use in vivo mouse models to study the effects of PSC on tumour proliferation and tumour regrowth in response to radiation.

Results: We demonstrate that PSC promote radioprotection and stimulate proliferation in pancreatic tumour cells in direct coculture. This is a PSC specific response as fibroblasts do not have a radioprotective effect on the tumour cells. Our in vivo studies of xenograft tumours in mice likewise demonstrate PSC dependent radioprotection and increased tumour proliferation. The kinetics of DNA repair in tumour cells showed no changes in the presence of PSC and was therefore concluded not to form part of this radioprotective response. We show that abrogating β 1-integrin and/or FAK signalling results in a significant decrease in the PSC mediated radioresistance in tumour cells.

Conclusion: We here demonstrate for the first time that PSC promote radioprotection of pancreatic tumour cells in a β 1-integrin dependent manner.

BACR Award Presentation at NCRI 2010

BACR/AstraZeneca Award presentation at NCRI Cancer Conference 2010



Dr. Grant Stewart

*Lister Fellow
School of Cancer Sciences,
University of Birmingham*

HNRNPUL-1 promotes DNA double-strand break end resection

Background: The cellular genome is under constant attack from a multitude of both intrinsic and extrinsic agents, which may occur directly, such as exposure to ionizing (IR) or ultraviolet radiation (UV), or indirectly through the generation of reactive oxygen species. While the cell has evolved a complex network of repair pathways that are specialised to recognise and respond to different deleterious DNA lesions, many of the proteins involved are capable of functioning in multiple repair processes. DNA double strand breaks (DSBs) are the most deleterious lesion induced by IR and these are repaired predominantly by two pathways: non-homologous end-joining (NHEJ) and homologous recombination repair (HRR). Single-stranded DNA (ssDNA) generated by DNA end resection at the DSB is the principle factor that stimulates both ATR-dependent cell cycle checkpoint activation and HRR. DNA end-resection is strictly controlled by Cyclin-dependent kinase activity and is mediated by the hMre11/Nbs1/hRad50 (MRN) complex, CtIP, Exo1 and the RECQ helicase, BLM. Despite our understanding of the cellular components governing end-resection, the exact mechanistic details remain unclear.

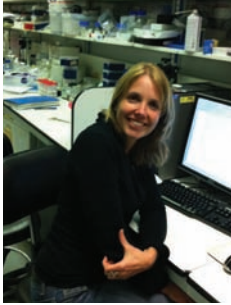
Results: Here we describe a novel role for the hnRNP protein, hnRNPUL-1 in regulating DNA end resection. Loss of hnRNPUL-1 results in cellular hypersensitivity to DNA damaging agents and an inability to activate ATR-dependent cell cycle checkpoints, mostly likely caused by a failure to stimulate BLM-dependent DNA end-resection and RPA recruitment to sites of DSBs. As a consequence, cells lacking hnRNPUL-1 exhibit a defect in HRR.

Conclusions: We have identified a new component of the DNA end-resection pathway that functionally regulates the recruitment of BLM.

Acknowledgments: Andrew N. Blackford, Sophie Polo, Anoushka Thomas, Rachel Blundred, Andrew S. Turnell, A. Malcolm R. Taylor, Roger A. J. Grand and Stephen P. Jackson

BACR Award Presentation at NCRI 2010

BACR Translational Award presentation at NCRI Cancer Conference 2010



Britta Weigelt

*Cancer Research UK
London Research Institute
London*

Molecular taxonomy of breast cancer: is there a gold standard?

Britta Weigelt¹, Alan Mackay², Roger A'hern³, Rachael Natrajan², David SP Tan², Mitch Dowsett^{2,4}, Alan Ashworth², and Jorge S Reis-Filho²

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Background: Microarray gene expression profiling has led to a working model for a molecular taxonomy of breast cancer comprising five molecular subtypes: luminal A, luminal B, basal-like, HER2 and normal breast-like. In the past decade, three distinct microarraybased single sample predictors (SSPs) have been described to determine the molecular subtype class of individual samples. It has been assumed that assignments made by different SSPs are synonymous. We analysed the agreement between these three SSPs in the identification of the molecular subtypes, and investigated whether each SSP identifies molecular subtypes with similar associations with outcome.

Methods: Breast cancers derived from four microarray datasets (total n=832) were assigned to the molecular subtype classes using the three previously described SSPs. We used Kappa score analysis to determine the agreement between SSPs for the whole classification system and for each molecular subtype separately. Associations with outcome were assessed by Kaplan-Meier analysis.

Results: The agreement between each pair of SSPs in each cohort was modest (Kappa scores 0.238 to 0.740). The assignment of samples to the luminal A, luminal B, HER2 and normal breast-like subtype classes was strongly dependent on the SSP employed, whereas basal-like cancers were robustly identified irrespective of the SSP (Kappa scores >0.810). This was not altered by different microarray-centring methodologies. Different SSPs produced similar survival curves, however the number and identity of cases assigned to the five molecular subtypes in each cohort differed depending on the SSP used, with the exception of the basal-like subtype. Analysis of each molecular subtype individually revealed that the associations with outcome of each molecular subtype other than basal-like and luminal A varied by the SSP employed.

Conclusion: Before introduction of the molecular subtypes into clinical practice, clear definitions and standardised methods for the identification of the molecular breast cancer subtypes are required.

Cambridge Research Institute
Annual International Symposium
**Unanswered Questions in
Transcription**

4–5 November 2011

Talks and panel discussions on:

Transcriptional regulation in mammalian cells

Shelley Berger University of Pennsylvania

Ronald Evans Salk Institute for Biological Studies

Tony Kouzarides Wellcome Trust/Cancer Research UK
Gurdon Institute

Bing Ren University of California, San Diego

Paolo Sassone-Corsi University of California, Irvine

Cancer epigenetics

Stephen Baylin Johns Hopkins University

Kristian Helin Biotech Research and Innovation Centre,
University of Copenhagen

Jean-Pierre Issa MD Anderson Cancer Center

Peter Jones University of Southern California

Keith Robertson Georgia Health Sciences University

Regulatory networks

Daphne Koller Stanford University

Jan Korbel European Molecular Biology Laboratory

Shirley Liu Harvard University

Dana Pe'er Columbia University

RNA regulators of gene expression

David Baulcombe University of Cambridge

Eric Miska Wellcome Trust/Cancer Research UK Gurdon
Institute

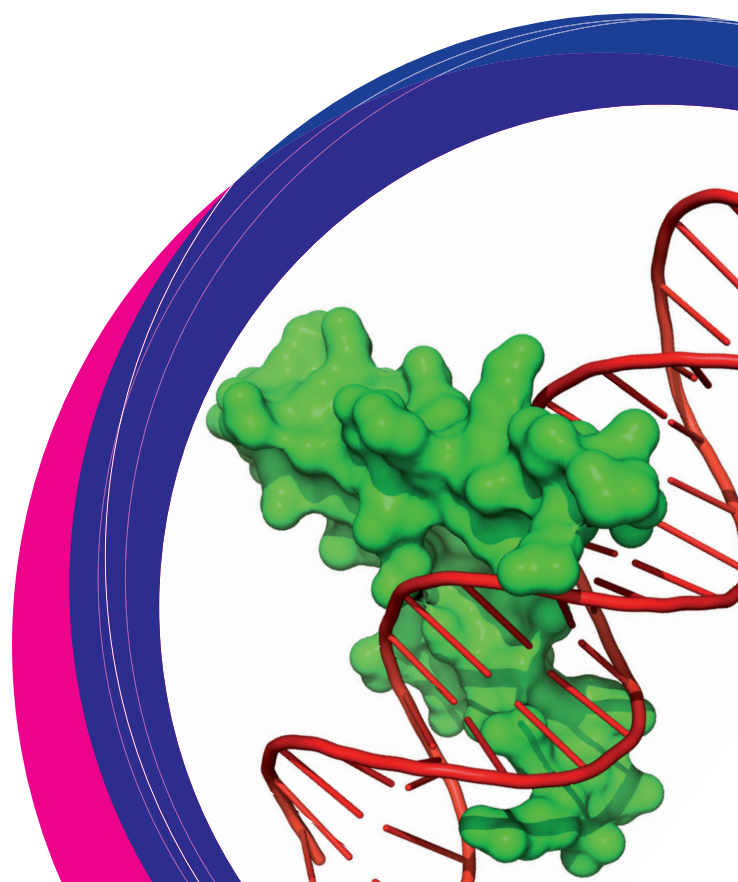
Nikolaus Rajewsky Max Delbrück Center for Molecular
Medicine, Berlin

Phillip Sharp Koch Institute for Integrative Cancer Research

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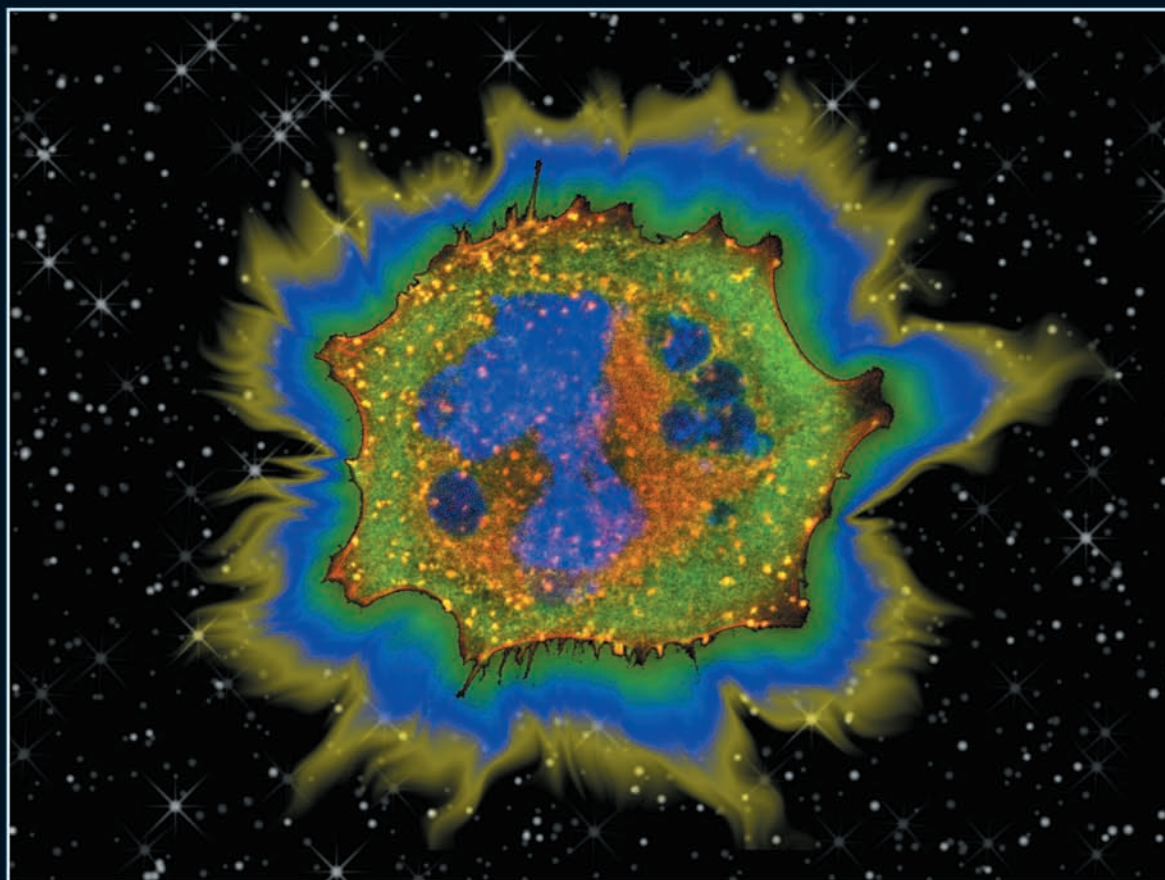
www.cambridgecancer.org.uk/cri_symposium

Registration deadline: 31 August 2011



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Cancer Models and Novel Therapies

Sunday July 3 – Wednesday July 6 2011 Glasgow, Scotland

Speakers and Sessions:

Keynote Address: Suzanne Cory (AU)

Signalling and Cancer I: Boris Bastian (US), Gideon Bollag (US), Lionel Larue (FR), Richard Marais (UK),
Inflammation and Cancer Stem Cells: Frances Balkwill (UK), Mariano Barbacid (ES), Tessa Holyoake (UK),
Rob Nibbs (UK), Luis Parada (US), Marcos Vidal (UK)

Angiogenesis and Invasion: Federico Bussolino (IT), Peter Carmeliet (BE), Kairbaan Hodivala-Dilke (UK),
Jim Norman (UK), Michael Olson (UK), Steve Wedge (UK)

Targeting Protein/Protein Interactions: Alan Fersht (UK), Paul Polakis (US), Saul Rosenberg (US), Dale Porter (US)

Signalling and Cancer II: Gerard Evan (UK), Margaret Frame (UK), Frank McCormick (US), Norbert Perrimon (US),
Catrin Pritchard (UK), Owen Sansom (UK)

Aims of the Conference

This conference will focus on the use of biological models of human cancer that may be used to provide insight into the causes and processes of this disease. The study of these models will facilitate the discovery, development and testing of novel therapies.

Short talks will be granted to the authors of outstanding abstracts. Some financial assistance will be available to the presenters of these talks through sponsorship from the Association for International Cancer Research.

Website, on-line registration, payment and abstract submission instructions: <http://www.beatson.gla.ac.uk/conf>

For additional information please contact:

Conference Administrator, Beatson Institute for Cancer Research, Garscube Estate,
Switchback Road, Bearsden, Glasgow, G61 1BD, UK

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E mail: s.price@beatson.gla.ac.uk

Deadline for registration payment and abstract submission May 6 2011

