<table>
<thead>
<tr>
<th>CONTENTS</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Weclome</td>
<td>2</td>
</tr>
<tr>
<td>Map</td>
<td>3-4</td>
</tr>
<tr>
<td>Programme</td>
<td>5-6</td>
</tr>
<tr>
<td>Charity Information</td>
<td>7-8</td>
</tr>
<tr>
<td>Keynote Speakers</td>
<td>9-12</td>
</tr>
<tr>
<td>Oral Presentations</td>
<td>13-32</td>
</tr>
<tr>
<td>Poster Presentations</td>
<td>33-37</td>
</tr>
<tr>
<td>Workshops</td>
<td>38-41</td>
</tr>
<tr>
<td>NEPG 2014 Credits</td>
<td>42</td>
</tr>
</tbody>
</table>
Welcome to the 9th annual North East Postgraduate Conference (NEPG). The NEPG - organised by postgraduates for postgraduates - has become the largest conference for bioscience and medical research postgraduates in the UK. The conference will showcase exceptional research from institutions across the North of the UK. The organising committee would like to take this opportunity to thank everyone that submitted abstracts; these have allowed us to put together an exciting and varied program of talks and posters from a wide range of subject areas.

We are privileged to welcome three inspiring keynote speakers: Dr Mike Addison (BSc, PhD), Dr Mark Prescott (BSc, PhD), and Julia Wilson (B.A. Sc). In addition to the many oral and poster presentations taking place throughout the day, NEPG 2014 will also play host to a range of postgraduate careers and advice workshops.

Finally, we would like to thank our sponsors. Please make time to visit their stalls and talk to their representatives - their kind donations and support has made this conference possible!

We hope you enjoy the day!

**NEPG 2014 Organising Committee**
NEPG 2014 Programme

Please use the colour-coded programme guide and map (pages 3 - 4) to navigate. Don’t hesitate to ask a member of the NEPG 2014 committee if you get lost or encounter any problems - we’re happy to help. Posters are split between the ground and first floor reception areas. We hope you enjoy the day!

**ALL DAY FROM 08:30**

- **REGISTRATION (GROUND FLOOR)**

**09:00-09:30**

- **OPENING REMARKS AND CHARITY SPEECH**

**09:30-10:10**

- **KEYNOTE: DR MIKE ADDISON, NEWCASTLE SCIENCE CITY**

**10:15-11:30**

- **ORAL PRESENTATIONS: LEUKAEMIA AND BEYOND**
- **ORAL PRESENTATIONS: CLINICAL STUDIES**
- **ORAL PRESENTATIONS: HEALTHCARE**
- **WORKSHOP: CAREERS IN INDUSTRY (10:30 - 11:00)**

**11:30-11:45**

- **COFFEE BREAK AND POSTER SESSION**
11:45-13:00
- ORAL PRESENTATIONS: CANCER
- ORAL PRESENTATIONS: MICROBIOLOGY
- ORAL PRESENTATIONS: MITOCHONDRIAL & NEURONAL DYSFUNCTION
- WORKSHOP: STEM AMBASSADORS (12:00 - 12:30)

13:00-14:00
- LUNCH AND POSTER SESSION

14:00-14:40
- KEYNOTE: JULIA WILSON, SENSE ABOUT SCIENCE

14:45-16:00
- ORAL PRESENTATIONS: NEUROSCIENCE AND DEMENTIA
- ORAL PRESENTATIONS: IMMUNOLOGY AND STEM CELLS
- ORAL PRESENTATIONS: ANIMAL MODELS
- WORKSHOP: VIVA AND THESIS SURVIVAL PANEL (15:00 - 15:30)

16:15-16:55
- KEYNOTE: DR MARK PRESCOTT, NC3Rs

16:55-17:30
- CLOSING REMARKS AND RAFFLE
SAVING TINY LIVES

The Tiny Lives Trust is a registered charity that helps to care for premature and sick newborn babies, their mothers and families in the Special Care Baby Unit of the Newcastle Neonatal Service based in the Newcastle Royal Victoria Infirmary (RVI). The trust aims to provide the highest quality of care on an on-going basis and needs to raise at least £150,000 every year to support a range of facilities, staff, and equipment.

“My baby survived because of these fabulous people and this immense charity”

Around 80,000 babies are born each year in the UK needing specialist hospital care. The ward supported by the Tiny Lives Trust is a centre of excellence and cares for babies across the North of the UK. The charity also provides support for families with sick babies on the ward, by providing accommodation nearby, for example. One parent commented: “My baby survived because of these fabulous people and this immense charity”

Other than offering help and support for sick babies and their families, The Tiny Lives Trust has also helped provide equipment to train medical staff - and has even donated to research efforts, by funding and initiating research projects in the health and life sciences sector.

This year, the NEPG 2014 committee are hoping to raise at least £1000 for The Tiny Lives Trust at NEPG 2014! Will you help us? With more than 600 registered delegates, donating just £1-2 would help us reach our goal. Even a small amount will make a huge difference to a baby in need. Why not visit our charity stall, buy a treat at the cake stall or try your luck with a raffle ticket?

Life saving equipment. Medical staff training. A place for family to stay closeby. As a charity that provides all of this and more, NEPG is proud to support the Tiny Lives Trust during our 2014 conference.
CHARITY RAFFLE

Fancy taking a chance for charity? Why not visit our raffle stall?

For just £1 a ticket, you’ll have the chance to win a whole host of amazing prizes, including: tasty treats, pampering sessions, science goodies and vouchers for your very own shopping spree!

Take a chance for charity!

CHARITY CAKES

Fancy a sweet treat?

Come visit our charity bake sale in the banqueting suite! Whether you’re craving a cupcake or bursting for a biscuit, we’re sure to have something tasty to try! A small donation is appreciated with each item, with all proceeds towards the Tiny Lives Trust. The winning baked item will receive a prize!
KEYNOTE SPEAKERS

Do you have an innovative idea? Are you curious about educating the public so they can make sense of scientific and medical claims? Or perhaps you’re interested in alternatives to the use of animal models?

Whatever your ideas, stance or future plans, NEPG is proud to host three outstanding keynote speakers in 2014. Each speaker is from a diverse background, with a wealth of experiences to share. With stories from business, academia and public engagement, why not join us?

There’s even the chance to access up to £10k of business support funding (subject to terms and conditions) from Newcastle Science City, following the 09:30 keynote talk from Dr Mike Addison, an expert in the management of technology and innovation.

09:30 - 10:10 : DR MIKE ADDISON
“Talking innovation”

14:00 - 14:40 : JULIA WILSON
“Science and evidence in the hands of the public”

16:15 - 16:55 : DR MARK PRESCOTT
“The future’s bright - the future’s 3Rs”
Talking innovation

Whether creating new products, seeking to expand into new markets or identifying new problems and addressing them with entirely novel solutions, you already understand the potential for a new concept. Innovation can be fraught with significant risks that, if not carefully managed, can result in much wasted effort, time and money.

Lean innovation methodology was pioneered in the start-up companies of Silicon Valley in the USA. The methodology offers an approach to innovation that facilitates speed but restricts costs when risks are greatest. The benefits of the approach are increasingly recognised by significant numbers of mainstream US companies of all sizes.

Yet the approach remains relatively unknown and underutilised in the UK.

This keynote session will enable you to discover the principles of lean innovation, including key aspects of how the lean approach could potentially be useful to you. You will subsequently have the opportunity to apply the learning to your own innovation and, if the plans are convincing, access up to £10k of business support funding (subject to terms & conditions) from Newcastle Science City.

Biography: Dr Mike Addison, former Procter & Gamble R&D manager, joined Newcastle Science City’s business support team after retiring from his position with the multinational manufacturer following a career spanning three decades.

One of the world’s most experienced open innovation practitioners, Mike has over 31 years’ experience of management of technology and innovation. At Science City Mike’s role is focussed on helping businesses find and develop commercial solutions to societal challenges in ageing, sustainability and medicine.

Website: www.newcastlesciencecity.com/about-nsc
Twitter: @NCLScience
Science and evidence in the hands of the public

Every day, we hear claims about what is good for our health, bad for the environment, how to improve education, cut crime and treat disease. Some are based on reliable evidence and scientific rigour. Many are not. So how can we make companies, politicians, commentators and official bodies accountable for the claims they make? And what role can early career researchers play in responding to bad science?

Sense About Science helps people to make sense of science and evidence and promotes the use of evidence in public life. This takes them from responding to outlandish diet claims by celebrities to helping parents understand vaccines, from working with people with chronic diseases to beat misleading ‘cure’ claims on the Internet to pressing for sound use of statistics in media reporting. Sense About Science’s development manager Julia Wilson will share some of these experiences which can be funny, emotional and inspiring, and talk about how you can get involved.

Biography: Julia is Development Manager at Sense About Science. She is responsible for Sense About Science’s fundraising activities and partnerships as well as the international programme of work and communications. Julia launched the Ask for Evidence campaign in Boston in February 2013 and oversaw the launch of the US version of our public guide to peer review. She previously coordinated the Voice of Young Science programme where she headed a campaign that pressured the W.H.O. to respond to the promotion of homeopathy for serious diseases in Africa, and has held many Standing up for Science media workshops in the UK and internationally including South Africa. Julia has a degree in Biology from the University of Manchester and joined the Sense About Science team in April 2009.

Website: http://www.senseaboutscience.org/pages/our-work.html
Twitter: @senseaboutsci
The future’s bright – the future’s 3Rs

There are many reasons for implementing the 3Rs – the replacement, reduction and refinement of animal use – principles developed over 50 years ago as a framework for humane in vivo research. The 3Rs are embedded in legislation regulating the use of animals in scientific procedures, and opinion polls consistently show that in the UK public support for animal research is conditional on application of the 3Rs. Perhaps more compelling is the growing recognition within academia and industry of the 3Rs as a framework for conducting high quality science. There is now much more focus on developing alternative approaches that avoid the use of animals and provide better tools for modelling human biology and predicting the efficacy and safety of medicines. The need to improve the design, conduct and analysis of in vivo research is also gathering momentum, with greater emphasis on minimising animal use and improving animal welfare. Knowledge about animals’ physical and behavioural requirements is expanding rapidly and being translated into practical information to minimise pain and distress and improve the robustness and reproducibility of animal experiments. The UK’s national centre on the 3Rs has been central to these changes within the scientific community, by funding research and early career development, supporting open innovation and the commercialisation of 3Rs technologies, and stimulating changes in policy, regulations and practice.

Biography: Dr Mark Prescott is Head of Research and Policy at the NC3Rs, an independent scientific organisation established by the UK Government to lead the discovery and application of new technologies and approaches which reduce reliance on in vivo research and improve animal welfare. Mark provides oversight on research funding and relationships with the academic community, including other research funders, and has primary responsibility for animal welfare issues. He trained as a zoologist and primatologist, and has 20 years research experience in primatology and animal welfare science. He serves on a number of institutional ethics committees and advisory bodies concerned with animal research and primatology.

Website: www.nc3rs.org.uk    Twitter: @NC3Rs
Vessels, Surfaces, and now... Media.

Corning alone manufactures the combination of advanced surfaces, innovative vessels, and both classic and custom media. We are uniquely positioned to help you develop a scalable, integrated cell culture environment.

Call us today at +31 (0) 20 659 60 51 or email cceu.nl@corning.com to request your free Corning samples.

For scientific support Ask the scientist! Email ScientificSupportEMEA@corning.com

We’ve got your cells covered.
ORAL PRESENTATIONS

SESSION 1 10:15 - 11:30

Leukaemia and Beyond
Pandon Room, 1st Floor
15 - 16

Clinical Studies
Collingwood Room, 1st Floor
17 - 18

Healthcare
Swan-Parsons Room, 1st Floor
19 - 20

Workshop: “How science works in a commercial environment”
Banqueting Hall, Ground Floor (10:00 - 10:30)
39

SESSION 2 11:45 - 13:00

Cancer
Pandon Room, 1st Floor
21 - 22

Microbiology
Collingwood Room, 1st Floor
23 - 24

Mitochondria and Neuronal Dysfunction
Swan-Parsons Room, 1st Floor
25 - 26

Workshop: STEM Ambassadors
Banqueting Hall, Ground Floor (12:00 - 12:30)
40

SESSION 3 14:45 - 16:00

Neuroscience and Dementia
Pandon Room, 1st Floor
27 - 28

Immunology and Stem Cells
Collingwood Room, 1st Floor
29 - 30

Animal Models
Swan-Parsons Room, 1st Floor
31 - 32

Workshop: Thesis and viva survival panel
Banqueting Hall, Ground Floor (15:00 - 15:30)
41
**LEUKAEMIA AND BEYOND**

http://ne-pg.co.uk/abstracts/leukaemia

**MARTINA FINETTI**
**NEWCASTLE UNIVERSITY**  
**10:15-10:30**

*Next-Generation Sequencing Identifies the Mechanism of Tumourigenesis Caused by Loss of SMARCB1 in Malignant Rhabdoid Tumours*

**Introduction:** Malignant Rhabdoid Tumours (MRT) are unique malignancies caused by bi-allelic inactivation of a single gene (SMARCB1). SMARCB1 encodes a protein that is part of the SWI/SNF chromatin remodelling complex, responsible for the regulation of hundreds of downstream genes/pathways. The understanding of downstream effects is essential to identifying therapeutic targets that can improve the outcome of MRT patients.

**Methods:** RNA-seq and 450k-methylation analyses and Chip-seq have been performed in MRT human primary malignancies (n>30) and in 4 MRT cell lines in which lentivirus was used to re-express SMARCB1 (G401, A204, CHLA-266, and STA-WT1). The MRT cell lines were treated with 5-aza-2’-deoxycytidine followed by RNA-seq and 450k-methylation.

**Aim:** To combine primary tumours and *in vitro* models in order to understand downstream effects of SMARCB1 loss as well as to identify key tumourigenic pathways which may be targetable therapeutically in the future.

**Results:** We show that primary MRTs have defined expression/methylation profiles which confirm that MRT broadly constitute a single paediatric tumour type. We observed that re-expression of SMARCB1 in MRT cell lines cause changes in expression and methylation status, determines activation/inactivation of specific downstream pathways (IL-6/TGF-beta), promotes expression of aberrant isoforms and novel transcripts and causes genome-wide changes in SWI/SNF binding.

**Conclusion:** The next generation analysis in primary MRT and in functional model provides detailed downstream effect of SMARCB1 loss in MRTs. Here we show how a single deletion of SMARCB1 is responsible for deregulation of expression, methylation status and binding at promoter regions of potent tumour-suppressor genes.

Genes, pathways and biological mechanisms implicated in tumour development will lead to better treatments for one of the most lethal paediatric cancers.

**HARRIET YOUNG**
**NEWCASTLE UNIVERSITY**  
**10:35 -10:50**

*The Role of Angiopoietin-1 in the Leukaemic Niche of Acute Lymphoblastic Leukaemia Harbouring the MLL/AF4 Fusion Gene*

**Introduction:** The MLL/AF4 fusion gene (t(4;11)(q21;q23)), commonly found in infant acute lymphoblastic leukaemia, confers a dismal prognosis. Its effects are not well understood, but gene analysis has shown angiopoietin-1 to be upregulated in MLL/AF4 positive cells. In vivo, leukaemic cells reside in a cell niche, which is thought to influence their growth.

**Aims:** To confirm a relationship between the MLL/AF4 fusion gene and angiopoietin-1, and to explore the effects of angiopoietin-1 on a leukaemic cell niche model *in vitro*.

**Methods:** The MLL/AF4 positive cell line SEM, transduced with siRNAs targeted towards MLL/AF4 and angiopoietin-1, were assessed by qRT-PCR and ELISA. Gene knockdown effects were evaluated in lone and co-culture with human umbilical vein endothelial cells (HUVEC) to mimic the stem cell niche. Cultures were treated with the chemotherapeutic drug doxorubicin to investigate the role of angiopoietin-1 in protection from cytotoxic stress.

**Results:** Knockdown of MLL/AF4 caused a reduction in angiopoietin-1 at an RNA and protein level. Angiopoietin-1 knockdown had no effect on cell growth of SEMs or HUVECs in lone or co-culture. Co-culture reduced SEM cell growth (p<0.01) but increased SEM survival against Doxorubicin by 10% (p>0.05). Conversely, HUVEC conditioned media increased SEM cell growth (p=0.01).

**Conclusions:** Angiopoietin-1 did not affect the growth of SEMs in lone or co-culture in vitro. However, co-culture reduced SEM growth suggesting a close contact effect of endothelial cells and leukaemic cells which could represent an interaction between cells in the leukaemic niche *in vivo*.  

**PANDON ROOM, 10:15 - 11:30**
Prognostic biomarkers for relapsed acute lymphoblastic leukaemia

Introduction: Acute lymphoblastic leukaemia (ALL) is the most common childhood cancer. Despite excellent cure rates, it remains a significant cause of morbidity and mortality due to unfavourable prognosis in relapsed patients. Poor outcomes have been associated with inadequate response to glucocorticoids, which facilitate leukaemic cell apoptosis via the glucocorticoid receptor (GR). The mechanisms responsible for glucocorticoid-resistance are unknown but one hypothesis is that deletions of NR3C1, the GR gene, play a vital role. Recent studies have demonstrated NR3C1 deletions exclusively at relapse in several cases, however, only small cohorts have been investigated to date.

Aims: This study aimed to determine the incidence of NR3C1 deletions in relapsed childhood ALL and to discern their prognostic relevance.

Methods: A real-time PCR copy number assay was validated and used to discern the NR3C1 copy number of 232 marrow relapse patients on the ALLR3 trial. Deletions were confirmed using Fluorescence In-Situ Hybridisation.

Results: The incidences of heterozygous and homozygous deletions were 7% and 1%, respectively. Investigation of matched presentation samples revealed NR3C1 deletions can be present at diagnosis or acquired at relapse. Analysis of clinical data evidenced that children with NR3C1 deletions have a statistically significant reduced likelihood of achieving second complete remission following first relapse \((p=0.03)\) and a borderline significant reduced overall survival \((p=0.08)\).

Conclusions: This study describes a reliable screening method for NR3C1 deletions and suggests its use as a predictor of response to induction therapy in relapsed ALL patients. Recommended future research includes trialling increased glucocorticoids or alternative drugs in children with NR3C1 deletions.

Second malignancies after childhood cancer – what are the likely reasons?

Introduction: With improving long-term survival rates for cancer in childhood and young adulthood, second malignancies are an increasing concern.

Aims: The present study describes the incidence of second malignancies in a cohort of children (0-14 years) and young adults (15-24 years) and assesses associations between the type of treatment received for first malignancies and the development of second malignancies.

Methods: 3,499 children and 3,462 young adults on the Northern Region Young Person's Malignant Disease Registry were included in the cohort. In three nested-case control studies of primary Hodgkin disease, brain tumours and sarcomas, 50 cases were matched to 100 controls by type of first malignancy, age at diagnosis, sex and follow-up. Treatment details were extracted from patient notes and conditional logistic regression used to determine associations between treatment type and second malignancy.

Results: Overall incidence of second malignancy was 1.4 per 1,000 person-years (95% CI 1.1, 1.6) (total 90,141 person-years follow-up ranging from 0-46.1 years). Children and young adults with primary Hodgkin disease, brain tumours or sarcomas had the highest rates of second malignancy. Odds of second malignancy decreased by a factor of 0.82 (95% CI 0.69, 0.98), 0.61 (95% CI 0.36, 1.05) and 0.86 (95% CI 0.74, 0.99) per year for primary Hodgkin disease, brain tumours and sarcomas respectively. Chemotherapy and/or radiotherapy increased odds of second malignancy non-significantly.

Conclusions: The significant reduction in rate of second malignancies over time reflects improvements in treatments, particularly radiotherapy. Future work should replicate this study in a larger population and include full treatment details.
High intensity intermittent exercise reverses abnormal cardiac function in people with Type 2 diabetes: an MRI/S study

Introduction and aims: Cardiovascular disease is the leading cause of morbidity and mortality in Type 2 diabetes but evidence based therapies to improve cardiac function are limited. This study investigated high intensity intermittent exercise as a potential therapeutic tool to moderate cardiac risk in this patient group.

Methods: Twenty-three patients with Type 2 diabetes (age 60±9 years) were randomised to 12 weeks of high intensity intermittent exercise (treatment, n=12) or standard care (controls, n=11). Cardiac structure, function and energetics were measured by 3.0 T magnetic resonance imaging and 2 dimensional tagging. Liver fat was determined by 1H-magnetic resonance spectroscopy.

Results: Compared to controls, high intensity intermittent exercise improved cardiac structure (Left ventricular Mass-104 ± 17 to 116 ± 20 vs 107 ± 25 to 105 ± 25g p<0.05) and systolic function (stroke volume-76 ± 16 to 87 ± 19 vs. 79 ± 14 to 75 ± 15ml, p<0.05). Early diastolic filling rates increased (241 ± 84 to 299 ± 89 vs. 250 ± 44 to 251 ± 47ml/s, p<0.05) and peak torsion decreased (8.1 ± 1.8 to 6.9 ± 1.6 vs. 7.1 ± 2.2 to 7.6 ± 1.9°, p=0.05). High intensity intermittent exercise also delivered benefits to liver fat (7 ± 7 to 4 ± 4 vs. 7 ± 7 to 8 ± 7%; p<0.05).

Conclusions: High intensity intermittent exercise has positive effects on cardiac structure and function alongside reductions in liver fat. Clinical care teams should consider high intensity intermittent exercise as a therapeutic strategy to moderate cardiac dysfunction in people with Type 2 diabetes.

The role of T-cells in IgA nephropathy-driven tubulointerstitial fibrosis

Background: IgA nephropathy is a major contributor to the global burden of chronic kidney disease. Tubulointerstitial fibrosis, the final common pathway for all progressive non-cystic kidney diseases, is an independent predictor of poor prognosis in IgA nephropathy. Experimental animal studies have suggested that T-cells infiltrating the renal cortex may promote tubulointerstitial fibrosis, although their role in human chronic kidney disease has not previously been investigated.

Aim: To quantify and phenotype the T-cell infiltrate in renal biopsies from patients with IgA nephropathy and, using this data, explore the relationship between T-cells, tubulointerstitial fibrosis and renal function.

Methods: Formalin-fixed paraffin-embedded renal biopsies underwent immunohistochemical dual-staining for CD4/Ki67 (proliferating CD4+ T-cells), CD4/Tbet (TH1), CD4/GATA3 (TH2), CD4/IL17 (TH17), CD4/FOXP3 (T-regs) and CD8/Ki67 (proliferating CD8+ T-cells). The number and location of dual-positive cells was analysed with clinical data and severity of fibrosis at biopsy.

Results: An increase across all T-cell subsets infiltrating the interstitium was associated with a greater degree of fibrosis (P<0.05). An increase in CD4+ and CD4+/GATA3+ T-cells infiltrating the tubular epithelium was associated with a reduced eGFR at biopsy and, along with CD4+/FOXP3+ T-cells, increased fibrosis (P<0.01). Extracellular IL17 staining within the tubular epithelium, an unanticipated finding, was also associated with reduced eGFR and increased fibrosis (P<0.05).

Conclusion: In agreement with previous research these results suggest an important role for interstitial T-cells in tubulointerstitial fibrosis. Extracellular IL17, TH2 cells and T-regs infiltrating the tubular epithelium may represent a previously unreported pathological process contributing to renal injury and fibrosis in chronic kidney disease.
KATHERINE JOHNSON
NEWCASTLE UNIVERSITY 10:55 - 11:10
Functional analysis of the osteoarthritis susceptibility locus marked by the polymorphism rs10492367

Introduction: The 2012 arcOGEN genome-wide association study reported that the rs10492367 G to T single nucleotide polymorphism (SNP) marks a region on chromosome 12p that is associated with hip osteoarthritis (OA) in Europeans. rs10492367 is intergenic and resides in a putative enhancer region 59 kb downstream of KLHDC5 and 96 kb downstream of PTHLH.

Aims: i) to identify the SNPs in high linkage disequilibrium (LD; ≥0.8) with rs10492367 that regulate enhancer activity of this region and ii) to determine the identity of the transcription factors that bind the SNPs.

Methods: Luciferase reporter assays were used to assess if any of the alleles of the SNPs in high LD with rs10492367 influenced enhancer activity in chondrosarcoma and osteosarcoma cell lines. Transcription factor binding was assessed using electrophoretic mobility shift assays (EMSAs).

Results: rs11049206 and rs58649696 independently resulted in altered enhancer activity in both cell lines in the luciferase reporter assays. Enhancer activity of rs10492367 only differed between alleles in the chondrosarcoma cell line. Competition EMSAs have identified allele-specific transcription factor binding to the three SNPs; including C/EBPβ binding to rs10492367; NFIC to rs11049206; and XBP1 to rs58649696. Supershift EMSAs are currently being used to confirm these results.

Conclusions: We have shown differential enhancer activity between alleles of intergenic SNPs in high LD with the OA association SNP, and have subsequently putatively identified transcription factor binding. Our findings provide the basis for further functional dissection and understanding of the OA associated region marked by rs10492367.

HOLLY STANDING
NEWCASTLE UNIVERSITY 11:15 - 11:30
‘We’re like a gang, we stick together’: Accounts of community between VAD patients

Introduction: Until recently heart transplants were often the only treatment available to patients with advanced heart failure. However, technological advances coupled with a decline in the number of donor hearts available for transplantation has resulted in increased use of ventricular assist devices (VADs). VADs are mechanical circulatory devices which support or replace the function of a failing heart.

Aims: The purpose of this study is to provide a deeper understanding of what it means to live with a VAD, specifically focusing on issues around communities within the VAD patient group.

Methods: This on-going qualitative study adopts a phenomenological methodology, using semi-structured interviews (n=20) with VAD patients. Purposive sampling was used to include patients with a range of experiences of VAD support.

Results: Several key aspects of community appeared in the data. Firstly, patients compared themselves to other patients to ascertain how well they were coping with the device. Secondly, patients offered support to each other and shared information around the realities of living with the device. Thirdly, death or deterioration of another patient often resulted in two levels of distress: grief at the death of a friend and concerns they would suffer the same fate. Fourthly, the manifestation and importance of the community varied across the trajectory of getting and living with a VAD.

Conclusions: The role and impact of community is multi-faceted with both positive and negative impacts on patients. Whilst patients valued exposure and contact to others who could empathise and offer support, exposure to those faring worse could result in distress.
Do we see in size or depth?

Introduction: Stereoscopic three-dimensional (S3D) technology is a rapidly advancing area in both entertainment and the medical field. As with all fast developing technology however, problems are found which need solving. One important issue regarding S3D is cue conflicts: the brain receiving different depth information from different signals. One such conflict: differing familial size and stereo information provided by S3D, may lead to the belief that something is incorrectly shown. In the industry this issue is widely referred to as miniaturisation. Although the conflict is well known in the commercial world of S3D production, it has not been studied much in research.

Methods: We consider whether humans have a preference for a familial size cue or a stereo depth cue. Using a standard credit card displayed in S3D, we varied the size and disparity information to test which cue was preferred. We tested both absolute and relative disparity. The subjects were required to make a decision as to whether the card appeared bigger or smaller than a standard, ‘real life’ credit card, given the size and depth information available.

Results and discussion: Analysis using probability heat maps revealed humans prefer familial size cues, and often ignored disparity when the card was presented. Mathematical modelling verified the heavier weighting we have toward familial size cue, which was reflected in the weaker reliability of the disparity cue. This could have repercussions for medical operations that use S3D technology, such as 3D laparoscopy, if the image shown has distortions in size.

Exploring individual perceptions of compassion in nursing

Introduction: Concerns regarding a plethora of negative patient experiences have led to the re-endorsement of compassion as a core professional nursing value. However, the notion of compassion requires further investigation due to the subjective and complex nature of this intangible concept, as it is perceived and understood uniquely by each individual who experiences it.

Aims: This doctoral study aimed to explore individual perceptions of compassion and address the research question: “what does compassion mean to the individual and how do they perceive this as a result of their unique experiences of nursing care?”

Methods: A constructivist grounded theory methodology was implemented. Participants were sampled from an existing group affiliated with Northumbria University in a role to share experiences with undergraduate health students. Semi structured individual interviews giving rise to rich and detailed data were audio recorded, transcribed verbatim and analysed using initial, focused and theoretical coding techniques.

Results: Two overarching themes emerged following data analysis:

- Individual perceptions of compassion- characterising what compassion means to participants, incorporating elements related to personal and professional dimensions of compassion.

- Enablers and inhibitors of compassion- highlighting factors impacting compassion, involving issues related to development of the mindful self, the influence of structure and the biology of selection.

Conclusions: Data analysis suggests that compassion involves a highly complex network of concepts which are interwoven and interdependent, all of which interact to influence individual perceptions of compassion in nursing. This results in significant emerging implications for practice in terms of issues such as recruitment, education, leadership and organisational culture.
The efficacy of the PPH shelf to facilitate uterine compression using a mannequin model: a randomised cross-over study

Introduction: Postpartum haemorrhage (PPH) remains the major cause of maternal mortality worldwide. The treatment of PPH has for many years focused on the provision of uterotonics. However, there are problems not only with the provision of the drugs to low resource community settings, but also in the escalation of care for those women who continue to bleed despite oxytocics. For treatment of atonic PPH bimanual uterine compression (BMC) is an appropriate procedure to initiate the management. However, the technique of BMC requires the insertion of a fist into the vagina, an act that is both painful and has overtones of gender-based violence. If, however, it could be performed in a less invasive manner, then it could act as a low-cost complete treatment for PPH. The development of a simple, low cost, treatment for PPH that can be easily used by low-level providers is therefore crucial.

The PPH shelf is a new device designed to make uterine compression available at a much earlier stage in the PPH process. This study tested the use of the PPH shelf in a mannequin model by delivery suite staff.

Method: Both experienced obstetricians (expert at BMC) and midwives (no previous BMC experience) took part. Each participant conducted two forms of uterine compression: bimanually and using the PPH shelf. The mannequin was supplied with an atonic uterus containing a pressure sensor. The sensor assessed the amount of intrauterine pressure produced by compression of each participant.

Preliminary results: 41 participants were included. There was no difference between the two groups in the amount of uterine pressures produced by the two methods and in the percentage of time over average pressure over 5 minutes.

Conclusion: PPH Shelf produces equivalent pressure to BMC. Experience has no effect on performing uterine compression on manikin model.

Does utilising simulation technology facilitate professional capability in undergraduate student nurses?

Introduction: High-fidelity simulation (HFS) has great potential to improve decision-making in clinical practice. In previous research, students reported an increase of confidence following HFS, but its effectiveness in clinical practice has not been established.

Aim: To establish to what extent exposing students to simulated clinical scenarios through HFS facilitates learning and informs decision-making skills in clinical practice.

Method: Multiple-criteria decision-making theory (MCDTM) allows the measurement of human judgement and decision-making under uncertainty. In this study, MCDTM is used to measure the quality of second-year pre-registration nursing students’ learning experience and the usefulness of HFS for clinical decision-making. A repeated-measures design is used to take two measurements: the first one after the first simulation experience and again after clinical placement. Baseline measurements were obtained from academics. Data were analysed utilising the MCDTM tool.

Results: The results demonstrate that immediately following their initial teaching students believe that simulation supports all aspects of clinical decision-making (83%) and that HFS supports high-quality learning experience (87%). Following clinical practice the level of support for clinical decision-making fell to 71%, suggesting that students’ perception of transferability of knowledge is limited.

Conclusion: Overall, the levels of support from simulation on the education experiences of undergraduate student’s nurses are high. However, there are no comparative data available from classroom teaching of similar content so it cannot be established if these levels are due to simulation alone. There is still a need for an objective, valid and reliable evaluation tool for student’s clinical performance following simulation education.
CANCER

http://ne-pg.co.uk/abstracts/cancer

KATIE BEST
NEWCASTLE UNIVERSITY 11:45-12:00
Cross-priming of the melanoma antigen, Melan-A, by human dendritic cells

Introduction: Dendritic cells (DCs) constitute a heterogeneous population of professional antigen presenting cells. DCs are capable of inducing de novo antigen-specific cytotoxic T lymphocyte responses by the phenomenon of cross-priming. Melanoma is the most aggressive form of skin cancer with a rising incidence worldwide. Current treatment options are associated with resistance and poor response. Demonstrating the ability of human DCs to elicit a cytotoxic anti-melanoma response will facilitate the development of novel anti-melanoma immunotherapies.

Aims: To test the ability of human monocyte-derived DCs, blood DCs, monocytes and skin antigen presenting cells to cross-prime naïve CD8+ T cells against the melanoma-associated antigen, Melan-A, in vitro.

Methods: Monocyte-derived DCs were generated from CD14+ monocytes. Naïve CD8+ T cells, blood DCs and monocytes were isolated from peripheral blood, and skin antigen presenting cells were isolated from digested whole skin, by fluorescence activated cell sorting. Monocyte-derived DCs, blood DCs, monocytes and skin antigen presenting cells were pulsed with Melan-A protein. Monocyte-derived DCs, blood DCs and monocytes were subsequently co-cultured with autologous naïve CD8+ T cells and skin antigen presenting cells were co-cultured with allogeneic naïve CD8+ T cells, for nine days. The induction of Melan-A-specific cytotoxic T lymphocytes was determined by flow cytometry analysis of MHC class I/Melan-A tetramer binding.

Results: Human monocyte-derived DCs, blood plasmacytoid DCs, blood CD1c+ myeloid DCs and epidermal Langerhans cells effectively cross-prime naïve CD8+ T cells against Melan-A, in vitro.

Conclusions: Definitive confirmation of the cross-priming status of these subsets will facilitate the development of rational, DC targeted anti-melanoma vaccination strategies.

GESA JUNGE
NEWCASTLE UNIVERSITY 12:05 -12:20
DNA-dependent protein kinase (DNA-PK) as a therapeutic target in the treatment of cancer

Introduction: DNA double strand breaks (DSBs) are toxic DNA lesions cells sustained due to environmental stress. There are two major repair pathways to cope with these; non-homologous end joining (NHEJ) and homologous recombination (HR). While mutations of key proteins in these pathways, for example Ataxia Telangiectasia Mutated (ATM) or Breast Cancer Early Onset (BRCA1), is associated with increased risk of cancer, DNA repair deficiency also provides a therapeutic opportunity, as inhibition of a second repair pathway can lead to cell death (“synthetic lethality”) as seen with PARP inhibitors in BRCA-deficient breast cancer.

Aims: Here, we aim to determine whether cancers deficient in ATM (a HR kinase) are hypersensitive to inhibition of DNA-PK, a key player in NHEJ.

Methods: Cell lines and primary samples from chronic lymphocytic leukaemia (CLL) patients were used to assess cell viability, apoptosis (Caspase 3/7 assay) and DNA damage (H2AX immunofluorescence).

Results: Two DNA-PK inhibitors, NU7441 and a novel Compound A chemo-sensitised CLL cells ex vivo 80-fold (range 1-500-fold) and 330-fold (1-2100-fold), respectively. The degree of chemosensitisation was not related to disease stage or previous treatment, and loss of p53 or ATM only accounted for some of the variation seen. However, DNA-PK inhibitors prevented repair of chemotherapy-induced DSBs and increased apoptosis in CLL cells ex vivo.

Conclusions: We have shown that DNA-PK inhibitors have promising activity in primary CLL samples, but have not found any relationship with loss of ATM. Current work is focussed on identifying biomarkers that allow patient stratification to determine which patients will likely benefit from treatment with DNA-PK inhibitors.
Plasma and the prostate: a new way to treat cancer?

Introduction: Low temperature plasmas (LTPs) have great potential for biomedical applications in bacterial sterilisation, wound healing, and cancer therapy. Adverse cellular effects such as DNA damage are induced through the formation of reactive oxygen and nitrogen species (RONS) delivered by the plasma to the target.

Aims:
- To assess the efficacy of LTP treatment on normal and cancerous prostate primary epithelial cells, by mapping the immediate cellular responses through to cell death at the molecular level.
- To evaluate the potential of LTP for the treatment of localised prostate cancer as an alternative to, or in conjunction with, existing therapeutic options.

Methods: Primary prostate cells from patient tissue were treated with LTP. DNA damage was analysed using the comet assay and by immunofluorescence for γ-H2AX foci. Cell viability and recovery were quantified by alamar blue and clonogenic assays. Western blots were performed for apoptosis and autophagy. Staurosporine (1 µM) and H2O2 (1 mM) were used as positive controls.

Results: We observed high levels of DNA damage from 30 seconds LTP exposure, similar to H2O2 control. Cell viability was significantly reduced following 600 seconds of LTP treatment, which lead to an 80% reduction in colony forming efficiency, versus untreated control. Western blotting showed that the cells do not die through apoptosis, indicating cell death must occur through other mechanisms.

Conclusions: LTP could prove an effective future treatment for localised prostate cancer; however, more work is required to elucidate the mechanism of cell death. In addition, a thorough understanding of the key RONS responsible for cellular response is necessary.

Metastasis Inducing Proteins are Overexpressed in High Grades Human Endometrial cancer

Introduction: Endometrial cancer (EC) is the commonest gynaecological tumour in the Western world. 50% of the aggressive type and advanced stages subsequently develop recurrence with gloomy outcome, yet reliable markers for follow up are lacking. Metastasis inducing proteins (MIPs): AGR2, S100P and S100A4 are described in other hormonally regulated malignancy; however, their expression in human EC is not fully defined.

Aims: Investigate the expression profile of MIPs (protein and transcript) in human endometrial cancer and their contribution to patients’ outcome and whether they are hormonally regulated.

Methods: 100 human EC samples, 15 postmenopausal controls (PM) were immuno-stained with AGR2, S100P, S100A4, steroid receptors (ERα, ERβ, PR and AR). mRNA levels were investigated using qRT-PCR.

Results: Preliminary results showed up-regulation of AGR2 in low grades ECs that significantly correlated with ERα. Nuclear S100P tend to be overexpressed in high grades EC compared to low grades and both showed significant up-regulation of S100P compared to PM control. Further, S100P expression inversely correlated to PR. S100A4 showed significant translocation to malignant epithelium, compared to predominant stromal expression in PM, which tend to increase with higher grades. S100A4 didn’t show any correlation with steroid receptors. MIPs transcripts showed a pattern similar to their proteins expression. Survival data to be presented in the conference.

Conclusion: The expression of AGR2 and S100P in EC might be regulated by steroid hormone and overexpression of MIPs in high grades EC suggests a possible role in tumour progression and metastasis. We propose that they may be potential therapeutic targets and prognostic indicators in ECs.
Preterm Gut Microbiome in Health and Disease

Introduction and aims: The gut microbiome in preterm infants is significantly associated with the development of NEC and sepsis. We aimed to extensively explore the differential community development and metabolic function in patients with necrotising enterocolitis and sepsis, matched to controls.

Methods: In total, 42 preterm infants were enrolled contributing a total of 747 stool samples. Patients were split into two groups consisting of 21 patients where 7 patients developed proven NEC and/or sepsis matched to 14 controls. Next generation sequencing was carried using the MiSeq platform and metabolomics profiles were generated using liquid chromatography mass spectrometry.

Results: The gut microbiota was relatively unstable in the initial weeks of life whereas the metabolic profiles were more comparable. The core microbiome consisted of Klebsiella Oxytoca, Escherichia coli, Staphylococcus, Enterococcus, and Veillonella. Increases in the abundance of certain operational taxonomic units (OTUs) were observed prior to NEC diagnosis, particularly with Escherichia coli. Organisms isolated in blood culture for the diagnosis of sepsis were typically abundant in the gut. Metabolites associated with both health and disease have also been identified.

Conclusion: The preterm gut microbiome is a complex and dynamic community with a multitude of factors influencing its development. Gestational age had important influences on the community. E. coli was prevalent prior to NEC onset in the most severe cases and dominant organisms in the gut were associated with sepsis. Metabolites were comparatively more stable, but important compounds have been identified and offer potential as biomarkers in health and disease.

Detection of staphylococcal enterotoxin fragments in urine

Introduction: Bacterial toxins cause a variety of conditions and can lead to serious complications such as toxic shock and sepsis. A proposed link exists between bacteria and the development of chronic, autoimmune conditions like rheumatoid arthritis (RA), relating to the hygiene hypothesis and molecular mimicry. It is hypothesised that staphylococcus aureus could play a role in the initiation and development of RA. A methodology for the rapid detection of common staphylococcal enterotoxin fragments in urine needs to be developed.

Aim: To enzymatically digest staphylococcal enterotoxin B and C (SEB/SEC), toxic shock syndrome toxin-1 (TSST-1) and alpha haemolysin (AH).

Methods: Enzymatic digestion of the toxins, using trypsin, pepsin and papain; confirmed by enzyme-linked immunosorbent assay (ELISA) and western blot.

Results: Trypsin digests SEB and SEC however TSST-1 needs to be incubated with pepsin and AH with papain. Optimal temperatures and timings for each enzyme and toxin were also established.

Conclusion: We have shown complete digestion of the staphylococcal toxins. There is currently no published method for the detection and identification of s.aureus fragments. The toxins may be broken down in the human body, which means that the detection of peptide fragments, rather than whole toxin, may lead to more effective identification of a bacterial presence. The application of mass spectrometry analysis of urine for the detection of staphylococcal enterotoxins is non-intrusive and could be extended to a range of autoimmune and inflammatory illness. This could also reach to the food industry as a more sensitive test for contaminated food.
**Modulation of E. coli phagocytosis by murine macrophages in presence of probiotic conditioned medium is mediated by NADPH-oxidase activity**

**Introduction:** *Lactobacillus rhamnosus* GG (LGG) is a well-studied probiotic bacterium demonstrating health beneficial effects. However, the physiological mechanisms by which the probiotics confer their health benefits are unclear. In this study a cell free LGG conditioned medium (LGG-CM) was used as it has been reported that LGG releases a number of soluble factors with beneficial health effects.

**Aims:** To investigate the role of LGG-CM on the phagocytic function of macrophages and elucidate the physiological mechanisms contributing to their functional attributes.

**Methods:** Phagocytosis of *E. coli* HfrC by murine macrophage J774 cells (both bacterial ingestion and digestion phases of phagocytosis) was monitored by gentamicin protection assays. Nitric-oxide (NO) production was measured by griess assays. Reactive oxygen species (ROS) and NO production were also monitored by fluorescence studies. Expression of NADPH oxidase proteins were confirmed by Western blot.

**Results:** *E. coli* uptake by macrophages was significantly reduced by LGG-CM. However, the digestion rate was significantly accelerated by LGG-CM. The accelerated *E. coli* digestion was found to be mediated by NADPH oxidase dependent ROS production. Inhibition of Nitric-oxide production had little effect on LGG-CM mediated enhanced bacterial digestion.

**Conclusion:** LGG-CM modulated production of free radicals is necessary for the normal phagocytic function of macrophages. This ability of LGG-CM to modulate production of NO and ROS could be a novel approach in improving the intestinal homeostasis and immunity.

---

**Influence of chromosomal location on heterogeneous gene expression in Bacillus subtilis**

**Introduction:** Highly expressed genes including 70% of identified tRNA operons in 68 species are reported to lie near the origin of replication. In bacteria such as *B. subtilis* and *E. coli*, essential genes also tend to cluster close to the origin. Such genes benefit from a gene dosage effect since multiple rounds of replication can be initiated in the same cell at high growth rates. The Bacillus industrial community uses a variety of integration vectors for locating genes encoding commercial products at ectopic chromosomal locations with consideration of the potential benefits of placing such genes close to the origin.

**Aim:** The aim of the present study was to determine the influence of location and orientation on the expression of the same reporter gene construct.

**Methods:** For this purpose two common reporters, superfolder GFP and β-galactosidase, were integrated randomly into the chromosome at various loci using the marine transposon. The reporters were cloned on opposite DNA strands to determine additionally the influence of gene orientation. To investigate the activities of both reporters, samples were analysed in exponential and stationary phase during growth in LB-medium.

**Results:** The results strongly suggest that there is indeed a bias in the expression of a gene relative to its location. The activities of superfolder GFP and β-galactosidase were higher when their genes were located close to the origin of replication. Hence the data strongly indicates that to improve productivity in large-scale applications in industrial environments, it is an advantage to use integration sites close to the origin of replication.
MITOCHONDRIA AND NEURONAL DYSFUNCTION
http://ne-pg.co.uk/abstracts/mito

ALEXIA CHRYSOSTOMOU
NEWCASTLE UNIVERSITY 11:45-12:00
Communication breakdown: the impact of mitochondrial dysfunction on the synapse

Introduction: Mutations in mitochondrial DNA lead to a heterogeneous group of disorders, known as mitochondrial diseases. These are defined by multi-system involvement with characteristic neuromuscular features. Approximately 70% of patients present with cerebellar ataxia, a neurological condition where motor co-ordination and balance are impaired. Previous neuropathological studies show severe neuronal loss, respiratory chain deficiency in remaining neurons and synaptic pathology throughout the olivocerebellar pathway. However, very little is known about the early mechanisms of degeneration in the pathway.

Aims: Since synapses constitute the most energetically costly part of a neuron, this study aims to quantify mitochondrial protein expression in Purkinje cells and their inhibitory projections on to the dentate nucleus. This should provide clues as to whether synaptic pathology precedes neuronal degeneration and cell loss.

Methods: A quantitative quadruple immunofluorescence technique was developed to measure respiratory chain protein (complex I-19kDa) expression in neuronal cell bodies and GABAergic presynaptic terminals in cerebellum sections from 12 patients with mitochondrial disease and 10 age-matched controls. Confocal microscopy was employed for neuronal sampling and enabled three-dimensional reconstruction of synapses.

Results: Significant reduction of complex I expression was observed in Purkinje cells and inhibitory presynaptic terminals formed on dentate nucleus neurons in patients. The severity of deficiency between the two sub-neuronal compartments is positively correlated. Patients have reduced synaptic numbers but when present, complex I deficient synapses are smaller in volume.

Discussion: This work suggests altered intracerebellar wiring in patients with mitochondrial disease and provides important insights into the neurodegenerative processes taking place.

JOHN G. F. CLELAND
NEWCASTLE UNIVERSITY 12:05 -12:20
Understanding the mitochondrial DNA defect in substantia nigra neurons

Introduction: The substantia nigra (SN) is an area of the midbrain which deals with the input and output from the basal ganglia. Previous studies have shown that in both Parkinson’s disease (PD) and old age neurons of the SN harbour high levels of mitochondrial DNA (mtDNA) deletions and respiratory deficiency.

Aim: To determine whether one species of mitochondrial DNA deletion or many species of deletion are present within single neurons in the substantia nigra.

Methods: This study used midbrain sections from a cohort of 10 PD patients and 10 age-matched controls. Following tandem cytochrome c oxidase (COX)/succinate dehydrogenase histochemistry, laser micro-dissection was used to isolate single COX positive neurons. Long range PCR was used to identify the presence of mtDNA deletions in single neurons. Next-generation sequencing was used to confirm that these were deletions and whether there were different deletion species present. Real-time PCR was used to quantify the heteroplasmy level of deletions within these neurons.

Results: The results show that individual substantia nigra neurons from PD patients and age-matched controls contain both single and multiple deletion species. Sequencing confirmed that there were multiple deletions within single neurons. Quantification showed that the level of deletions were not high enough to cause mitochondrial dysfunction, however, this is due to our method of cell selection.

Conclusion: The presence of multiple mtDNA deletion species within a single neuron would suggest that clonal expansion of more than one species of deletion can occur, which has implications for how clonal expansion is understood.
Investigating the contribution of axonal torpedo formation to Purkinje cell vulnerability in patients with mitochondrial disease

**Background:** The central nervous system is highly dependent on mitochondria for ATP generation, via oxidative phosphorylation. This dependence is exemplified in that patients with mitochondrial disease frequently develop neurological symptoms. The cerebellum is commonly affected in mitochondrial disease, with axonal torpedoes being a common neuropathological feature. Axonal torpedoes are fusiform swellings of the proximal axonal segments of Purkinje cells and contain a high concentration of disorganised neurofilaments. Our understanding of the mechanisms leading to formation of torpedoes and the consequence on Purkinje cell function remains undetermined.

**Aim:** The aim of this study is to quantify the expression of Complex I 19kDa (CI 19kDa) in Purkinje cells, axons and axonal torpedoes and to determine the consequences of axonal torpedo formation.

**Methods:** Using a quadruple immunofluorescent assay and confocal microscopy, the level of CI 19kDa expression was quantified in 10 mitochondrial DNA patients and 13 control subjects. In addition, immunofluorescence was used to determine how axonal torpedo formation alters the level of myelination.

**Results:** Expression of CI 19kDa was reduced in Purkinje cell bodies, axons and axonal torpedoes of mtDNA patients compared to controls. The formation of axonal torpedoes alters the level of myelination with the majority of axonal torpedoes being unmyelinated.

**Discussion:** Data confirms that axonal torpedoes are not associated with respiratory chain deficiency however the formation of axonal torpedoes disrupts the myelin sheath. Mitochondrial dysfunction combined with axonal torpedo formation is likely to increase the burden on neurons, further disrupting the neuronal circuitry and neurological function.

---

**Regulation of post-transcriptional gene expression in human mitochondria**

**Introduction:** Transduction of energy is essential for the survival of cells. 90% of ATP used by cells is produced by the oxidative phosphorylation (OXPHOS) chain found in the inner membrane of mitochondria (IMM). Thirteen components of the OXPHOS chain are encoded by the mitochondrial genome and translated by mitoribosomes in the organelle itself. A dysfunction in mitochondrial translation will decrease the number of functional OXPHOS complexes and lead to a reduction of ATP synthesis, affecting the health of the cell. The interaction between mitoribosomes and IMM has been claimed to be important for mitochondrial protein translation, but how this association occurs is still not clear.

**Aims:** Study importance and mediators of the interaction of mammalian mitoribosomes with the IMM.

**Methods:** Mitochondria extraction, mitochondrial subfractionation, isokinetic sucrose gradient. Western blot analysis. Knockdown experiment with targeting siRNA. Cloning and overexpression of candidate proteins and their mutants.

**Results:** Our research has been focused on MRPL45, a mitoribosomal specific protein that was proposed as a candidate according to its position and its structural homology with a membrane-interacting protein. The sucrose gradient suggests that MRPL45 is present in the mitoribosome and as a free pool. siRNA depletion of MRPL45 in different cell lines shows a defect in mitochondrial protein synthesis and a reduction in growth rate when compared to a control. The mitochondrial sublocalisation suggests that MRPL45 might associate with the IMM. Wild-type and mutated MRPL45 have been successfully cloned and the effects of their overexpression are under study.

**Conclusions:** The importance of MRPL45 is underlined by the siRNA knockdown experiments. The protein appears to be both associated with the mitoribosome and the IMM. Its role will be further investigated using the wild-type and mutated MRPL45 overexpression cell lines.
Investigation of disease mechanisms in dominant optic atrophy

Introduction: Dominant Optic Atrophy (DOA) is a neurodegenerative disorder which presents primarily with progressive visual loss, usually manifesting in the first decade of life. Most of these patients only develop an isolated optic nerve condition known as Pure Optic Atrophy (OA). However, a subset of patients can also develop additional systemic symptoms later in life, a condition known as Dominant Optic Atrophy Plus (DOA+). Up to 75% of patients with either form of the disease harbour mutations in the OPA1 gene, a regulator of mitochondrial dynamics previously linked to mitochondrial function. Why a subset of patients harbouring OPA1 mutations develop the syndromic form, whilst others do not, remains unknown.

Aims: The aim of my study is to investigate the underlying mitochondrial biochemical pathways discriminating between these two forms of DOA.

Methods: Mitochondrial function, OPA1 protein and gene expression levels were investigated using a range of biochemical, microscopic, molecular and functional assays in patient fibroblasts.

Results: Mitochondrial morphology showed increased fragmentation in DOA+ patients under normal conditions. Whilst OA presented with lower OPA1 protein levels and increased elongation under oxidative phosphorylation (OXPHOS) driven ATP production, OXPHOS functional studies revealed no major differences between both groups of patients.

Conclusion: Data suggest different modes of pathogenicity between the two groups with OA associated with haploinsufficiency and DOA+ associated with an increased susceptibility to stress. However, both groups of patients showed similar mitochondrial biochemical function. This study is the first step towards solving the underlying cause of syndromic DOA.

Prodromal dementia with Lewy bodies

Introduction: Dementia with Lewy bodies (DLB) is the second most common type of degenerative dementia. It is characterised by the presence of parkinsonism, visual hallucinations and fluctuating confusion. 123I-FP-CIT (DaTSCAN) imaging is used to diagnose DLB.

Efforts are being made to identify and sub-type dementia in the prodromal phase — the period between the onset of the earliest symptoms and the development of the full clinical syndrome. Biomarkers such as clinical symptom scales, cognitive tests and blood tests may be helpful with this.

Aims: To identify clinical biomarkers of prodromal DLB.

Methods: Patients with mild cognitive impairment and symptoms suggestive of Lewy body disease were recruited. Each patient had a comprehensive clinical and neuropsychological assessment, followed by DaTSCAN imaging. Those with positive scans were presumed to have prodromal DLB. They were compared to those with negative scans to identify possible markers of prodromal DLB.

Results: The DaTSCAN positive group displayed greater impairments in some cognitive tasks such as attention and set-shifting (Trails B), response control and inhibition (cognitive processing time) and abstract thought (verbal fluency). However, overall there were few differences between the DaTSCAN positive and DaTSCAN negative groups. This may be in part because some of the DaTSCAN negative group also had Lewy body disease.

Conclusions: DaTSCAN may have insufficient sensitivity to reliably identify DLB in the prodromal phase; other markers such as cognitive test scores may be useful. These patients will be followed-up longitudinally in an effort to clarify their diagnosis identify other biomarkers.
Changes in cognition and quality of life in Parkinson’s disease: from diagnosis to 18 month follow up

Background: For the individual living with Parkinson’s disease (PD), the development of dementia has a significant impact upon quality of life (QoL). The impact of mild cognitive impairment in PD (PD-MCI) and QoL is less clear.

Aim: To study the impact of PD-MCI on QoL in a cohort of patients with PD from diagnosis to 18 month follow up.

Methods: Newly diagnosed PD patients (n=219) completed a schedule of neuropsychological tests, together with scales assessing QoL (PDQ-39), neuropsychiatric symptoms and a clinical examination. The Movement Disorder Society criteria were used to classify PD-MCI. These measures were repeated after 18 months.

Results: 186 participants completed follow up assessments (85%). Over 18 months 13% developed PD-MCI, 19% were classified as more severely impaired and five developed dementia. QoL significantly decreased, participants with more severe cognitive impairment (≥2 SD below normative values) had the poorest QoL at baseline and 18 months (p<0.01). More severe PD-MCI was also associated with decreased mobility, activities of daily living, support, cognition, bodily discomfort (all p≤0.01) and communication (p<0.05). Logistic regression analysis showed PD-MCI was a significant predictor of QoL at baseline, but not at 18 months.

Conclusions: MCI negatively affects QoL in newly diagnosed PD with more severe impairment having the greatest effect. Interestingly, regression analysis showed that PD-MCI was not a predictor of QoL at 18 months, although was significant at baseline. This suggests QoL may be initially impaired by PD-MCI but participants may adapt or use coping techniques to minimise the effect of this on QoL over time.

Investigation of the effects of serum and 2-deoxy-D-glucose on transfected HEK293 cells transfected with different APP isoforms

Introduction: Amyloid β peptide (Aβ) is a small insoluble 39–43 amino acid peptide derived from the Amyloid Precursor Protein (APP) by proteolytic cleavage. Different gene splicing produces variant isoforms ranging from 365 to 770 amino acids in length. The main three isoforms are: APP695, APP751 and APP770 and all are potentially sources of Aβ.

Aim: The study aimed to investigate the hypothesis that one of these APP isoforms (APP695, APP751 and APP770) is more likely to be the source of Aβ in Alzheimer’s disease under the stress-induced conditions.

Methods: HEK293 cells stably expressing human APP695, APP751 and APP770 were stressed by: serum alteration (FCS concentration) and energy deprivation (2-deoxy-D-glucose (2DG)). Cell number was recorded using haemocytometre. The culture medium was collected for immunoprecipitation and immunoblotting procedures in order to quantify an amount of APP secretion. The total mRNA was extracted from harvested cell pellets and used to determine gene expression levels using quantitative real-time PCR.

Results: The differences in the concentration of FBS in culture medium did not contribute to cell number, APP production and APP gene expression results of all isoforms of APP. Whereas, the limited glucose utilisation has slowed down cell growth, maintained the amount of secreted APP while increasing the mRNA levels of each isoform of APP.

Conclusion: No significant effects of short term stress on APP gene expression and secretion was found when comparing the three main isoforms of APP: APP695, APP751 and APP770. Nevertheless, the slight changes in cell number could be the indication of some relevant process of stress since the pathological changes in AD has taken place over several years.
Metal-on-metal joints: cobalt can cause an inflammatory response through human TLR4

Introduction: Joint replacement is used to treat conditions such as osteoarthritis. Materials used for joint implants include ceramic, polyethylene and metal. Metal-on-metal joints, often fabricated from a cobalt-chromium alloy, are associated with adverse reactions including inflammatory pseudotumours and bone breakdown. The molecular mechanisms underlying these reactions are unknown.

Cobalt ions activate human toll-like receptor 4 (TLR4), a cell surface immune receptor that usually recognises bacterial lipopolysaccharide and induces an inflammatory response.

Aims: This study aims to determine the inflammatory consequences of cobalt-mediated TLR4 activation using an in vitro approach.

Methods: MonoMac 6 (macrophage) and U2OS (osteoblast or bone-forming) cell lines were selected for this study because of their importance in innate immunity and relevance to joint replacement, respectively. Secretion and expression of pro-inflammatory cytokines were measured by ELISA and real-time PCR. Monocyte or neutrophil migration was assessed by trans-filter chemotaxis assay.

Results: In TLR4-expressing macrophages and osteoblasts, cobalt ions were found to increase expression and secretion of the pro-inflammatory chemokine interleukin-8 (IL-8), and of pro-inflammatory CXCL10 in macrophages. Cobalt-treated macrophages were found to increase migration of monocytes and neutrophils, an essential aspect of an inflammatory response. These effects were inhibited using a TLR4-specific antagonist, showing they are a direct consequence of cobalt-mediated TLR4 activation.

Conclusions: These findings suggest that cobalt ions induce a complex inflammatory response through TLR4 activation, leading to migration of immune cells. As well as joint implants, cobalt is used in coronary artery stents, dental implants and spinal rods. Consequently, further work is required to better understand and prevent cobalt's inflammatory effects.
**Characterisation of an induced pluripotent stem cell model of prostate development**

**Introduction:** Studies into prostatic diseases such as prostate cancer are limited by the lack of a relevant model. Prostate specific induced pluripotent stem cells (ProiPSCs) have recently been generated and represent a novel method to study human prostate development and the transition from normal to malignant tissue. ProiPSCs have been shown to differentiate into cells expressing prostate markers however further characterisation is required before these cells can be used for pre-clinical research.

**Aims:** To optimise a 3D cell culture model for the differentiation of ProiPSCs and analysis of prostate marker expression.

**Methods:** A protocol for in situ immunofluorescent analysis was adapted for use with 3D prostate structures within a matrix. ProiPSCs were differentiated in 2D or 3D cultures and marker expression analysed by both in immunofluorescence and RT-PCR.

**Results:** ProiPSCs can be differentiated within a 3D cell culture system and early analysis shows significantly decreased expression of the stem cell marker OCT4 and enhanced expression of prostate specific markers including NKX3.1, AR and TMPRSS2.

**Conclusions:** This study has optimised a 3D cell culture system for the differentiation of ProiPSCs and analysis of expression markers in situ. The full spectrum of ProiPSC differentiation can now be interrogated further.

**Manufacturing mesenchymal stromal cells to Good Manufacturing Practice standard for the treatment of graft-versus-host disease**

**Introduction:** Mesenchymal stromal cells (MSCs) are multipotent stem cells with immunomodulatory properties, defined by their adherence to plastic, antigen expression (>95% CD73, CD90, CD105; <2% CD14, CD19, CD34, CD45, HLA-DR), and differentiation potential. Acute graft-versus-host disease (aGVHD) is a complication of haematopoietic stem cell transplantation causing T-cell-mediated damage to skin, gut and liver. MSCs can be expanded to treat aGVHD by following Good Manufacturing Practice (GMP), a European quality and safety assurance system for medicinal products.

**Aims:** To validate an MSC expansion protocol under GMP conditions and establish its feasibility as future aGVHD therapy.

**Methods:** Two MSC samples were harvested from bone marrow, cultured under either non-GMP or GMP conditions, phenotyped by flow cytometry (passages P2 and P3), and differentiated. Immunomodulatory properties were tested in the skin explant model, an in vitro predictor of aGVHD: allogeneic activated T-cells were co-cultured at a 20:1 ratio with MSCs primed with TNF-α and IFN-γ, then co-cultured with a volunteer’s skin biopsy, which was graded for aGVHD damage (I-IV).

**Results:** All work conducted under GMP conditions was compliant with recommendations. Both samples displayed normal morphology, proliferation, and differentiation capacity. Antigen expression followed definition criteria in both samples except for HLA-DR in GMP-MSCs, which was 7.42% (P2) and 5.32% (P3). aGVHD grade was reduced from a positive control in skin co-cultured with non-GMP- and GMP-MSC-modulated T-cells.

**Conclusions:** MSCs can be successfully cultured under GMP conditions, and have immunomodulatory potential for the treatment of aGVHD in Newcastle-upon-Tyne, UK.
The role of centrosomal protein 104 (cep104) in congenital anomalies of the kidney and urinary tract

**Introduction:** Congenital anomalies of the kidneys and urinary tract (CAKUT) account for ~50% of chronic kidney disease in children. CAKUT includes a wide range of phenotypes including cystic kidney disease. Individual CAKUT phenotypes are often attributable to single gene mutations. Mutations in genes whose protein products are expressed in centrosomes and cilia have been linked to cystic kidney diseases. Zebrafish have emerged as an informative animal model in which to study congenital renal diseases. Centrosomal protein 104 (cep104) is a candidate cystogene /cilipathy gene, but its role in renal development is unknown.

**Aim:** Using zebrafish as a model organism we will determine the role of cep104 in embryogenesis, with emphasis on renal development.

**Methods:** Splice-blocking morpholino oligonucleotides (MOs) targeted to cep104 were designed and used for microinjection of zebrafish embryos. Specificity of gene knockdown was assessed using RT-PCR. Mortality rates in MO injected and uninjected embryos were determined. Light and fluorescent microscopy at 48 and 72 hours post fertilisation was used to identify developmental abnormalities.

**Results:** cep104 morphants displayed developmental defects resulting in higher mortality rates than uninjected embryos in a dose dependent manner. cep104 morphants displayed a consistent set of abnormal phenotypes including heart, tail, ear and pronephros (renal) defects. Abnormal splicing of cep104 was not confirmed by RT-PCR.

**Conclusion:** cep104 morphants displayed phenotypes consistent with it being a cilipathy. Morphants exhibited renal phenotypes including cloacal abnormalities. Further work is needed to confirm the specificity of cep104 knockdown and its functional effects on primary cilia during development.

The variant Histone H2AZ influences developmental DNA methylation and is necessary for correct embryogenesis in Zebrafish

**Introduction:** H2AZ is a variant form of Histone H2A, a member of the histone protein family involved in DNA organisation. Structural differences in variant histones enable roles beyond chromatin organisation and H2AZ has been shown as inversely correlated with methylation levels in the plant A. thaliana. In a study of heritable adaptation to the fibrotic response we recently demonstrated increased incorporation of H2AZ at the PPARy promoter in the sperm of injured rats followed by decreased methylation of PPARy in offspring. This suggests that alterations in H2AZ expression early in development may influence methylation status in adults.

**Aims:** To investigate whether H2AZ influences methylation during early embryonic development using Zebrafish as a model organism.

**Methods:** H2AZ expression was effectively depleted via morpholino (MO) injection at the one to four cell stages. Global methylation levels, protein and gene expression were analysed by immunoblotting and qRT-PCR.

**Results:** H2AZ depletion resulted in significant mortality and phenotypic abnormality. MO injected fish were smaller than control fish, presenting with curvature of the body, malformation of the head, deformation of organs and defective movement. Defects were accompanied by genome wide hypermethylation until 24 hours post fertilisation and alteration in expression of key developmental pathways such as Hedgehog signalling.

**Conclusions:** H2AZ therefore appears critical during embryonic development as morphants suffer significant morphological defects. Furthermore, hypermethylation in morphants supports the role for H2AZ influencing methylation, demonstrated here for the first time in an in vivo vertebrate model.
Recognise that smell? Investigating odour-based recognition in the banded mongoose (*mungus mungo*)

**Introduction:** Recognition is beneficial for a host of behaviours: identifying kin, avoiding or engaging competitors, and selecting a mate. Choosing the wrong animal to help, harm or reproduce with can be costly; hence, the ability to recognise individuals is intrinsic to fitness. However, deciphering mechanisms as to how recognition is possible remains a challenge. Here we discuss how odour cues are involved in genetic recognition in a population of wild banded mongooses (*mungus mungo*). Banded mongooses are small, cooperative carnivores, ideal for studying recognition as conspecifics live and breed within large closely related groups. With little dispersal or immigration groups face a real threat of inbreeding. However, mate-choice within both sexes may minimize this risk by allowing mongooses to choose the least-related individuals to mate with.

**Aims and methods:** We aim to investigate how banded mongooses use odour-communication to make important reproductive decisions. In this first step we conducted odour presentations to test whether mongooses can discriminate scents based upon relatedness and familiarity. In separate trails focal mongooses were presented with samples of anal gland secretion from both close and distant relatives within their own group and from close and distant relatives from non-neighbouring, thus totally unfamiliar groups. With little dispersal or immigration groups face a real threat of inbreeding. However, mate-choice within both sexes may minimize this risk by allowing mongooses to choose the least-related individuals to mate with.

**Results and conclusions:** We show that in opposite-sex presentations mongooses show longer and heightened behavioural responses to the odours of less-related individuals. This suggests odour cues may encode genetic information which mongooses may use to make important behavioural decisions regarding mate-choice and inbreeding avoidance.
POSTER PRESENTATIONS

Posters will be displayed throughout the day in the ground floor and first floor reception areas (key-coded yellow on the centre map, pages 3 - 4). Posters can be viewed at any time, though lunch and coffee breaks have been allocated specifically for poster viewing and marking.

Ground Floor Posters (1 - 26)

Cancer

1. Investigating the clinical pharmacology of dexamethasone in acute lymphoblastic leukaemia (ALL)
   Rosanna Jackson, Newcastle University

2. Investigation of the potential use of methylation based biomarkers for prediction of relapse in good prognostic cytogenetic subgroups of childhood ALL
   Fadhel Lafta, Newcastle University

3. The Function of RUNX1/ETO Target Genes in Leukaemic Propagation
   Yura Grabovska, Newcastle University

4. The use of next generation sequencing to extend genetic analysis in acute myeloid leukaemia
   Ruth Cranston, Newcastle University

5. Phenotype-specific Ca²⁺ signalling in differentiating neuroblastoma cells
   Claire Whitworth, Newcastle University

6. DNA-PK as a marker of non-homologous end joining in ovarian cancer
   Richard O’Sullivan, Newcastle University

Microbiology

7. Molecular characterization of the activity and requirements of a novel and promiscuous bacteriophage integrase
   Mohammed R Mohaisen, University of Liverpool

8. Stx-phage vB_EcoP 24B sculpting host bacterial function
   Giles Samuel Holt, Northumbria University
9. Seroimmunity profile, associated knowledge and concerns of hepatitis B, varicella and measles infections among new employees recruited in Saudi National Guard
   Majid Althaqafy, Newcastle University

10. Effect of two candidate factors in RSV translational coupling
    Shiney George, Newcastle University

11. Transcriptomic profiling of host-parasite interactions in the microsporidian
    *Trachipleistophora hominis*
    Andrew Keith Watson, Newcastle University

12. Cloning and functional expression of nucleoside transporters from *Leishmania major* and *Leishmania Mexicana*
    Khalid Jamaan H Alzahrani, University of Glasgow

13. Characterisation of Antimicrobial Peptides from Egyptian Elapids Snake Venoms
    Mohamed M. Tawfik, Sheffield Hallam University

    Lauren Drage, Newcastle University

**Immunology**

15. Glucocorticoid receptor function in chronic fatigue syndrome
    Megan Lynn, Newcastle University

16. Tartrazine is not an activator of the mouse oestrogen ERα and ERβ receptors
    Stephanie Meyer, Newcastle University

17. Understanding the cellular source of IL-6 and IL-23 in early rheumatoid arthritis
    Natasha Price, Newcastle University

18. Isolation and expansion of bivirus-specific T-cells for immunotherapy
    Mairi Shepherd, Newcastle University

19. Inhibition of MAPK leads to telomerase-dependent proliferation of T lymphocytes
    Nayef Al Zhrany, Newcastle University
POSTER PRESENTATIONS

**Neuroscience**

20. The lateral geniculate nucleus in Lewy body dementia: the role of “bottom-up” pathways in hallucinogenesis  
   Daniel Erskine, Newcastle University

21. Visual cues increase saccade frequency during gait in Parkinson’s disease  
   Samuel Stuart, Newcastle University

22. Development of novel biomarkers for autoimmune related psychiatric illness  
   Sandra Mould Urias, Newcastle University

23. Investigation of gamma oscillations in the avian hippocampus  
   Pradeep Dheerendra, Newcastle University

24. Assessing emotion-related attention and processing speed in depression  
   Thomas James Walker, Newcastle University

25. Sleep and cognitive abnormalities in bipolar disorder  
   Ayana Hazu, Newcastle University

26. Exploring the relationship between task parameters and reaction time variability in the continuous performance test  
   Rachel Ann Moss, Newcastle University

**First Floor Posters (27 - 50)**

**Health and Disease**

27. “Use-it-or-lose-it” - older adults’ perceptions of brain health and its maintenance  
   Lizzie Dutton, Newcastle University

28. Artificial grammar learning and the evolution of language  
   Alice Milne, Newcastle University

29. Have socioeconomic inequalities in childhood cognitive scores changed over time? A comparison of three British cohort studies  
   Tomos Robinson, Newcastle University
POSTER PRESENTATIONS

30. A longitudinal analysis of the 2006/7 and 2012/13 National Child Measurement Programme (NCMP) data for Middlesbrough and Newcastle
Emma Mead, Teesside University

31. Exercise modalities and arterial stiffness: A systematic review and meta-analysis of randomised controlled trials
Ammar Waham Ashor, Newcastle University

32. The impact of oral health risk indicators on prevention uptake
Samridh Sharma, Newcastle University

33. Media influence on ideal body size of indigenous populations on the Mosquito Coast in Nicaragua
Tracey Thornborrow, Newcastle University

34. Association between daily air pollution levels and asthma emergency department visits in Al Jubail Industrial City, Saudi Arabia
Salem Albalawi, Newcastle University

Ageing and Mitochondrial Disease

35. Determining the nature and mechanisms of progression of mitochondrial DNA disease: why do patients get worse?
Hannah Rosa, Newcastle University

36. Adult-onset Mendelian PEO associated mitochondrial disease
Ewen Sommerville, Newcastle University

37. Characterising iPSC models of mitochondrial disease
Pavandeep Rai, Newcastle University

38. Changes in serotonin, substance P, vesicular glutamate transporter 2 and corticotropin-releasing factor immunoreactive terminals in the lumbosacral spinal cord of aged C57BL/6J male mice
Hayley Wing Sum Tsang, Northumbria University

Molecular Biology

39. Effect of Bile acid on primary cells cultured from lung transplant recipients and Human lung carcinoma cells
Adil Aldhahrani, Newcastle University
POSTER PRESENTATIONS

40. The potential role of miR-200b in development of Bronchiolitis obliteran syndrome
Shameem Sultanali Ladak, Newcastle University

41. The metabolic effects of thrombin on cultured skeletal muscle cells
Ali Al-Bayati, Newcastle University

42. Collagen XII; novel disease-causing human gene for Collagen VI-related myopathies
Golara Torabi Farsani, Newcastle University

43. Investigating the conformation of the 2A peptide in the ribosomal exit tunnel
Pippa Harvey, Newcastle University

44. TALEN-based molecular surgery for epidermolysis bullosa simplex
Magomet Aushev, Newcastle University

45. Selenoprotein H modulation of the Nrf2 signalling pathway
Anthony Moore, Newcastle University

46. Towards the fabrication of highly controlled functional DNA wires via sequence controlled enzymatic synthesis
Colette Whitfield, Newcastle University

47. Biodegradation of petroleum hydrocarbons in heavy metal-petroleum contaminated soil
Obioma Kelechi Mejeha, Newcastle University

Systems Biology

48. Validation of a systems approach for cytokine stimulation of MMP-1 and MMP-13 in chondrocytes
David Hodgson, Newcastle University

49. Design, synthesis and biological evaluation of small molecules for induction of cellular differentiation
Hesham Haffez, Durham University

50. Theoretical characterisation of redox signalling through Nrf2 during skeletal muscle contractions
Alvaro M. Guimera, Newcastle University
NEPG 2014 WORKSHOPS

Not sure whether there’s a relevant subject talk for you?

Rather than attending the student oral presentations you can visit the sponsor stalls, view the posters and attend workshops.

This year, NEPG has three diverse postgraduate workshops running in parallel to the student oral presentations.

Not sure which path to take after graduating? Our careers workshop, “How science works in a commercial environment” (10:30 -11:00) may be right up your street! Interested in science engagement but not sure where to start? We’ve also got a STEM Ambassador talk (12:00 - 12:30), where you’ll be able to find out more about the national STEM Ambassador programme - enabling people in science to engage with schoolchildren in their local area. A question and answer session on thesis and viva survival (15:00 - 15:30) is also planned for the afternoon - so why not pop along if you’re nearing the end of your PhD and are feeling a bit nervous about what’s to come? All workshops will be held in the banqueting suite on the ground floor.

10:30 - 11:00 : HOW SCIENCE WORKS IN A COMMERCIAL ENVIRONMENT
12:00 - 12:30 : STEM AMBASSADORS
15:00 - 15:30 : THESIS AND VIVA SURVIVAL
Take this opportunity to attend a session organised by Newcastle University’s Careers Service and Newcastle Science City.

Receive up to date information regarding the bio-science job scene; the opportunities and challenges.

Find out what skills and knowledge employers look for.

Discover more about the value of developing strong enterprise skills to support your research and future career opportunities including how the Rise Up team in the Careers Service could support you in developing your own business idea or explore the commercial potential of your research.**

(** applies to Newcastle University students and graduates. Participants from other universities can benefit from a one to one meeting with Business Support Managers from Newcastle Science City who are exhibiting at the conference)
STEM Ambassadors are a widely recognised and respected body of volunteers who work and study in the STEM (Science, Technology, Engineering and Maths) subjects. Their presence in a school can enlighten, enthuse and encourage young minds to pursue a path into a STEM subject.

Rhys will talk about the STEM Ambassador scheme, the past work carried out and where the Northumberland, Tyne and Wear Team are taking the programme in the coming months.

If you would like to share your passion for Science with the next generation of students, this is an opportunity not be missed!

“STEM Ambassadors”
Rhys Wilson, STEM Outreach Co-ordinator
Banqueting Suite, Ground Floor
12:00 - 12:30

“When I talk to groups of young people, there are always some that have a ‘light bulb’ moment, and realise that science is exciting and rewarding, and something that they can and want to do for a living” - Heather Williams, STEM Ambassador
Reaching the end of your Phd?

This workshop will provide a huge range of ideas and suggestions to help PhD students cope with the intellectual issues and practical difficulties of organising their work into a well-structured thesis and preparing for their viva.

You may be asking:

“How should I organise my writing time?”
“How do I stay motivated while writing up?”
“What should I expect during the viva?”

Our panel of recent PhD graduates and thesis examiners will be providing tips and strategies for starting and completing your thesis and answering any questions you may have.
NEPG 2014: CREDITS AND THANKS

NEPG2014 would not have been possible without the hard work and determination of a dedicated team of organisers. NEPG is created for postgraduates, by postgraduates: so our team have been juggling PhD studies alongside the planning and implementation of NEPG 2014! As such, we'd like to give credit and due thanks.

Chair

Sarah Billington, Newcastle University

Team Leaders

Daria Stroukova, Newcastle University (Finance)
Laura Kay, Northumbria University (Marketing)
Francesca Everest, Northumbria University (Venue)
Katie Griffiths, Newcastle University (Abstracts)
Zoltan Derzsi, Newcastle University (Website)
Michael Savage, Newcastle University (Keynote Speakers)

Zach Dixon (Abstracts) Irene del Molino del Barrio (Marketing)
Magdalena Glod (Abstracts) Marco Silipo (Marketing)
Sylvia Muller (Abstracts) Mabel Okoeki (Marketing)
Helen Gray (Venue) Rachel Challis (Finance)
Anna-Lena Dittrich (Venue)

Special Thanks to:

Richy Hetherington, for his continued support of NEPG
On-the-day volunteers
Everyone who submitted abstracts and attended!

The North East Postgraduate Conference will return in 2015 for its 10th anniversary.