NHMRC Clinical Trials Centre

COOPERATIVE TRIALS GROUP FOR NEURO-ONCOLOGY

The achievement of better health outcomes for patients and those affected by brain tumours through clinical trials research

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7th COGNO ANNUAL SCIENTIFIC MEETING

'Translating science to patient centred trials'

FRIDAY 24th — SATURDAY 25th OCTOBER 2014

THE LANGHAM, MELBOURNE, AUSTRALIA

CONFERENCE BOOKLET

COGNO

COOPERATIVE TRIALS GROUP FOR NEURO-ONCOLOGY

CONTENTS

Program of Events	Page 1
Oral Abstract Listing	Page 3
Poster Abstract Listing	Page 4
Oral Abstracts	Page 6
Poster Abstracts	Page 9

2014 ASM ORGANISING COMMITTEE

Dr Mustafa Khasraw, Co-Convenor	Medical Oncologist, Andrew Love Cancer Centre
Dr Zarnie Lwin, Co-Convenor	Medical Oncologist, Royal Brisbane and Women's Hospital
Dr Giovanna D'Abaco	Neuroscientist, University of Melbourne/Centre for Neural Engineering
A/Prof Kate Drummond	Neurosurgeon, Royal Melbourne Hospital
Ms Marcia Fleet	Care Coordinator, Melbourne Health
Dr Cecelia Gzell	Radiation Oncologist, Genesis Cancer Care
Ms Jenny Chow	Executive Officer, COGNO
Ms Yi Feng	Administrative Assistant, COGNO
Ms Hannah O'Riley	Administrative Assistant, COGNO

INTERNET

Complimentary internet is available for all delegates in the COGNO ASM meeting rooms.

- Connect your device to the wifi option “Langham Melbourne”
- A login page will appear, select “For alternative access/payment options, please click here” (do not input your room number)
- Select the connect code option and put in COG24OCT
- You should now be connected

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COOPERATIVE TRIALS **G**ROUP FOR **N**EURO-**O**NCOLOGY
The achievement of better health outcomes for patients and those affected by brain tumours through clinical trials research

24 October 2014

Dear Colleague

On behalf of the Organising Committee, welcome to the 7th COGNO Annual Scientific Meeting!

We are delighted to announce that this year, COGNO ASM delegates had the opportunity to help sponsor a delegate from a low-income country, to attend a future COGNO meeting. We extend our sincere thanks to everyone who helped make this possible.

We are excited to host two renowned international guest speakers:

- Professor Lisa M DeAngelis MD, Memorial Sloan-Kettering Cancer Center, USA
- Professor Minesh P Mehta MB ChB, FASTRO, Maryland Proton Treatment Center, USA

The theme of this year's meeting is 'Translating science to patient centred trials'.

Highlights of our program include:

- Trial or Error: A bird's eye view of glioma research in the last decade
- Challenges in attracting clinical trials to Australia
- The new paradigm of brain mets
- Translating biology and immunity
- Keeping up with the RANOs
- When the tumour's not the target
- Unforgettable tumours

Our appreciation goes to all our sponsors: Cancer Australia, Cure Brain Cancer, Roche, MSD Oncology, Novartis Oncology, Victorian Cancer Agency (State Government Victoria), Brainlab and Cancer Institute NSW.

We hope you enjoy the ASM!

Kind regards

Dr Mustafa Khasraw
Co-Convenor
COGNO ASM 2014

Dr Zarnie Lwin
Co-Convenor
COGNO ASM 2014

Prof Mark Rosenthal
Chair
COGNO

The COGNO Scientific Program has been developed independent of sponsor involvement.

Cure Brain Cancer FOUNDATION — Gold Sponsor

NOVARTIS ONCOLOGY; State Government Victoria; MSD ONCOLOGY; Roche — Bronze Sponsors

Australian Government Cancer Australia

Roche — Lunchtime CNS Lymphoma Symposium Sponsor

BRAINLAB — Friday Morning Tea Sponsor

NSW Government; cancer institute NSW — Travel Grant Sponsor

PROGRAM OF EVENTS

Thursday 23 October: Pre-ASM Meetings (Yarra Room)		
TIME	**MEETING**	**CHAIR**
3:00-6:00pm	**COGNO Scientific Advisory Committee Meeting** (open to COGNO members only)	Dr Liz Hovey
Friday 24 October: ASM Day 1 (Ballroom)		
TIME	**SESSIONS AND SPEAKERS**	**CHAIR**
8:30-9:00am	On arrival tea/coffee	
9:00-9:10am	**Welcome and Day 1 program overview**	Dr Mustafa Khasraw & Dr Zarnie Lwin
9:10-10:30am	**Session 1** ***"Trial or Error?"*** ***A bird's eye view of glioma research in the last decade*** International Guest Speakers • Prof Lisa DeAngelis: GBM • Prof Minesh Mehta: Low grade and Grade 3	Prof Mark Rosenthal
10:30-11:00am	Morning tea	
11:00am-12:30pm	**Session 2** ***"Challenges in attracting clinical trials to Australia"*** ***A robust debate style discussion through the lens of:*** • Industry: Ms Catherine Drury (Roche) and Dr Victor Loh (Novartis) • Ethicist: Dr Giuliana Fusclado • Trial Nurse: Ms Marian Lieschke • Australian and International investigators in the audience	A/Prof Ian Kerridge
12:30-1:30pm	Lunch	
12:30-1:30pm	**CNS Lymphoma Lunch Symposium: Prof Lisa DeAngelis** **Yarra Room**	Dr Lawrence Cher
1:30-3:00pm	**Session 3** ***"COGNO Trials Update"*** • Prof Mark Rosenthal: CATNON • Dr Kathryn Field: CABARET • Dr Eng-Siew Koh: SEED • Dr Hui Gan: Antibody drug conjugate trials in glioma • Dr Mustafa Khasraw: PARP inhibitor trials in GBM	Dr Eng-Siew Koh & Dr Liz Hovey
3:00-3:30pm	Afternoon tea	
3:30-4:45pm	**Session 4** ***"The new paradigm of brain metastases"*** • Oral abstract/proferred paper Dr Brindha Shivalingam: Length of stay after craniotomy and resection of melanoma brain metastases in the modern Neurosurgical era Case based discussions on state of the art treatment for Theme 1: solitary metastasis Theme 2: oligometastases Theme 3: multiple metastases Discussants: • Prof Minesh Mehta • Dr John Fuller	A/Prof Kate Drummond
4:45-6:30pm	**Poster Walk Around/Welcome Reception**	

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Saturday 25 October: ASM Day 2 (Ballroom)		
TIME	**MEETING**	**CHAIR**
8:30-9:00am	On arrival tea/coffee	
9:00-9:10am	**Welcome and Day 2 program overview**	Dr Mustafa Khasraw & Dr Zarnie Lwin
9:10-10:30am	**Session 5** ***"Translating biology and immunity"*** • Dr Eric Hau: An update on biology of glioma • Prof Paul Waring: Personalised Therapies • Oral abstract/proferred paper Dr Kerrie McDonald: Overexpression of DNA repair genes predicts Glioblastoma sensitivity to veliparib • Oral abstract/proferred paper Dr Yi Chieh Lim: Stem-like Glioblastoma Cells Can Be Targeted With A Novel Radiomimetic Agent With Roles in DNA Double Strand Break Induction and Homologous Recombination Inhibition	Dr Giovanna D'Abaco & Dr Kerrie McDonald
10:30-11:00am	Morning tea	
11:00am-12:30pm	**Session 6** ***"Keeping up with the RANOs"*** • Dr Frank Gaillard: Imaging of GBM and use of RANO criteria • Prof Lisa DeAngelis: Pitfalls in imaging interpretation in the era of novel agents • Oral abstract/proferred paper Dr Eng-Siew Koh: Application of the RANO imaging criteria in high grade glioma in a clinical practice setting: a multi-reader concordance study • Oral abstract/proferred paper Dr Cecelia Gzell: Small increases in enhancement on MRI may predict survival post radiotherapy in patients with glioblastoma	Dr Liz Hovey
12:30-1:30pm	Lunch	
1:30-2:50pm	**Session 7** ***"When the tumour's not the target"*** • Prof Bogda Koczwara: Life after brain tumour – it is time to get our head around it • A/Prof Fary Khan: Is rehab for all? • Oral abstract/proferred paper A/Prof Jennifer Philip: Understanding the Supportive and Palliative Care Needs of People with High-Grade Malignant Glioma and their Carers	Ms Marcia Fleet & Ms Dianne Legge
2:50-3:20pm	Afternoon tea	
3:20-4:20pm	**Session 8** ***"Unforgettable tumours"*** • Prof Lisa DeAngelis: Brain stem gliomas, ependymomas • Prof Minesh Mehta: Meningiomas, vestibular schwannomas	Dr Cecelia Gzell
4:20-4:25pm	**ASM Summary**	
4:25-4:30pm	**ASM Awards and Close**	
4:30-5:00pm	**COGNO Annual General Meeting** (open to COGNO members only)	
7:00-11:00pm	**COGNO Conference Dinner (Melbourne Town Hall, catered by EPICURE)**	

RANZCR CPD POINTS AVAILABLE: 10.5 RANZCR CPD points can be claimed for attendance at the 7th COGNO Annual Scientific Meeting.

RACS CPD POINTS AVAILABLE: This educational activity has been approved in the Royal Australasian College of Surgeons' CPD Program. Fellows who participate can claim one point per hour [maximum 11 points] in Category 4: Maintenance of Knowledge and Skills towards 2014 CPD totals.

ORAL ABSTRACT LISTING

Session 4 **Length of stay after craniotomy and resection of melanoma brain metastases in the modern Neurosurgical era**
Brindha Shivalingam, John Thompson, Jonathan Stretch, Robyn Saw, Andrew Spillane, Kerwin Shannon, Georgina Long, Rick Kefford, Catriona McNeil, Alex Guminski, Angela Hong, Gerald Fogarty

Session 5 **Overexpression of DNA repair genes predicts Glioblastoma sensitivity to veliparib**
Kerrie Mcdonald, Kyoko Nozue-Okada, Motoo Nagane, Mustafa Khasraw

Session 5 **Stem-like Glioblastoma Cells Can Be Targeted With A Novel Radiomimetic Agent With Roles in DNA Double Strand Break Induction and Homologous Recombination Inhibition**
Yi Chieh Lim, Brett Stringer, Kathleen Ensbey, Zara Bruce, Paul Jamieson, Tara Roberts, Lisa Wiesmüller, Andrew Boyd, Bryan Day

Session 6 **Application of the Response Assessment in Neuro-Oncology (RANO) imaging criteria in high grade glioma in a clinical practice setting: a multi-reader concordance study**
Yehia Elhgar, Sugendran Pillay, Ramesh Cuganesan, Paul de Souza, Joseph Descallar, George Papadatos, Annette Tognela, Matthias Jaeger, Sam Hall, Eng-Siew Koh

Session 6 **Small increases in enhancement on MRI may predict survival post radiotherapy in patients with glioblastoma**
Cecelia Gzell, Helen Wheeler, Philip McCloud, Marina Kastelan, Michael Back

Session 7 **Understanding the Supportive and Palliative Care Needs of People with High-Grade Malignant Glioma and their Carers**
Anna Collins, Carrie Lethborg, Michelle Gold, Vijaya Sundararajan, Gaye Moore, Michael Murphy, Caroline Brand, Jennifer Philip

POSTER ABSTRACT LISTING

P1 **Synovial Sarcoma of L4 exit foramen and paraspinal muscles**
Anmar Al-Witri, James Dimou, Loui Rassam, John Christie

P2 **A meta-analysis of anti-angiogenic therapy for glioblastoma (GBM)**
Malaka Ameratunga, Andrew Lassman, Nick Pavlakis, Mustafa Khasraw

P3 **Infinium HM450K analysis of DNA methylation in malignant glioma**
David Ashley, Nick Wong, Andrea Muscat, Mustafa Khasraw, Kate Drummond, Mark Rosenthal, Kathryn Field, Elizabeth Algar

P4 **What are the risk factors for glioma in adults? A review of the epidemiological evidence**
Lisa Chalmers, Kerrie McDonald, Claire Vajdic

P5 **Socio-economic and lifestyle predictors of survival in high grade glioma – analysis of the Australian Genomics and Clinical Outcomes of Glioma Epidemiological Questionnaire**
Arosha Dissanayake, Julie Marsh, Kerrie McDonald, Anna Nowak

P6 **ANZMTG 01.07 WBRTMel: Whole Brain Radiotherapy Following Local Treatment of Melanoma Brain Metastases – Interim Analysis**
Kate Drummond, Gerald Fogarty, Angela Hong, Kari Dolven-Jacobsen, Claudius Reisse, Bryan Burmeister, Elizabeth Paton, Victoria Steel, Lauren Haydu, Haryana Dhillon, Brindha Shivalingam, Janette Vardy, Anna Nowak, George Hruby, Richard Scolyer, Catherine Mandel, John Thompson

P7 **Applying functional imaging in brain tumour clinical trials**
Mike Fay, Stephen Rose, Nick Dowson, Paul Thomas, Jenny Martin

P8 **Benefits of a Neuro-Oncology Nurse Program**
Emma Daly, Gabrielle O'Connor, Lauren Todorovic, Ron Freilich

P9 **Delineating the cellular hierarchy of glioblastoma and gliosarcoma for identification of therapeutic targets**
Melanie Jackson, Tracy Seymour, Kerrie McDonald, Anna Nowak, Foteini Hassiotou

P10 **Treatment related reduction in platelet count as a prognostic factor in glioblastoma multiforme**
Ben Kong, Mark Wong

P11 **Use of supportive services by adults recently diagnosed with primary brain tumours in Queensland**
Danette Langbecker, Patsy Yates

P12 **Capitalising on a unique opportunity: establishing the Brain Tumour Support Officer role at Austin Health**
Dianne Legge, Lawrence Cher

P13 **Regional support network for primary malignant brain tumours**
Dianne Legge, Kelly Mills, Lawrence Cher

P14 **Role of Focal Adhesion Kinase in Glioma Stem Cell Migration**
Kai Zheong Lim, Giovanna D'Abaco, Wayne Ng, Nicole Kountouri, Alice Pebay, Terry Johns, Kate Drummond, Andrew Kaye, Andrew Morokoff

P15 **Survival outcome of multiple resections in patients with glioblastoma multiforme (GBM) in the South Australian population**
Louisa Lisa Lo, Kimti Kumar, Thorbjorn Loch-Wilkinson, Marguerite Harding, Nimit Singhal

P16 **A novel brain cancer mouse model harbouring human-relevant PI3-kinase pathway mutations**
Theo Mantamadiotis, Michael Christie, Paul Waring, Gulay Filiz, Wayne Phillips

P17 **Low dose histone deacetylase inhibitor treatment halts rhabdoid tumour growth and induces osteogenesis and neuronal differentiation**
Andrea Muscat, Jason Cain, Melissa Ferguson, Dean Popovski, Elizabeth Algar, Fernando Rossello, Samantha Jayasekara, D.Neil Watkins, Jason Hodge, David Ashley

P18 **Low use of communication support tools in patients with primary or metastatic brain malignancy treated at two NSW metropolitan radiotherapy centres**
Diana Naehrig, Eng-Siew Koh, Monica Vogiatzis, Waka Yanagisawa, Shepherd Heather, Chris Milross, Haryana Dhillon

P19 **Identifying Patterns of Service Use and Place of Death of Patients with a High-Grade Malignant Glioma**
Jennifer Phillip, Anna Collins, Caroline Brand, Michelle Gold, Gaye Moore, Michael Murphy, Carrie Lethborg, Megan Bohensky, Vijaya Sundararajan

P20 **A novel way to remove pineal tumours using Axiem electro-magnetic navigation via endoscopy and the Nico Myriad side cutting aspiration device**
Mary-Anne Xia, Amelia Granger, Paul Nichols, Brendan Davis, Joseph Garcia-Redmond, Sarah Olson

ORAL ABSTRACTS

Length of stay after craniotomy and resection of melanoma brain metastases in the modern Neurosurgical era

Brindha Shivalingam[1,2,3], John Thompson[1,2,3], Jonathan Stretch[1,2,3], Robyn Saw[1,2,3], Andrew Spillane[1,3], Kerwin Shannon[1,2,3], Georgina Long[1,3], Rick Kefford[1,4], Catriona McNeil[1,2], Alex Guminski[1,3], Angela Hong[1,2,3], Gerald Fogarty[3]

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3 The Mater Hospital, North Sydney, Sydney, Australia
4 Westmead Hospital, Sydney, Australia

Introduction: Brain metastasis in the context of stage IV melanoma confers a grim prognosis. However survival appears to be increasing due to improved targeted systemic therapies. Aggressive management of brain disease has therefore become more worthwhile. Previously, length of stay (LOS) after craniotomy was in the order of 10-14 days. With the advent of stereotactic navigation and minimally invasive approaches in neurosurgery, length of stay has decreased in general. We present the results of a single surgeon's practice in the modern era of Neurosurgery.

Method: A retrospective analysis of medical records of all patients with metastatic melanoma was carried out between the years 2008 and 2014. All patients were referred within the context of the Melanoma Institute Australia and operated on by the affiliated Neurosurgeon.

Results: A total of 142 operations were performed on 106 patients. The average age was 60.1 years (range 16-86). A total of 92% of operations resulted in post operative LOS of 4.3 days. Only 8% of operations resulted in LOS ≥14 days. Twenty-eight (28) patients were over the age of 70 (26.4%). The vast majority of patients had operations on a single lesion (116) whilst 26 operations were on 2-3 lesions. A total of 30 (21%) operations were performed on lesions that had previously been treated with radiation.

Conclusion: Length of stay after craniotomy for metastatic disease in the brain has significantly improved due to modern techniques of surgery. Therefore the cost burden of treating melanoma metastases with surgery needs to be revisited.

Overexpression of DNA repair genes predicts Glioblastoma sensitivity to veliparib

Kerrie Mcdonald[1], Kyoko Nozue-Okada[1,2], Motoo Nagane[2], Mustafa Khasraw[3]

1 UNSW, Sydney, Australia
2 Kyorin University, Tokyo, Japan
3 Andrew Love Cancer Centre, Geelong, Australia

Background: The survival trends for glioblastoma (GBM) patients have remained largely static, reflecting a lack of improvement in the therapeutic options for patients. The development of new, active and potentially targeted drugs for the treatment of GBM represents a major unmet need. Veliparib (ABT-888; Abbvie) inhibits both PARP1 and PARP2 (poly[ADP-ribose] polymerase).

Materials and Methods: In this study, we investigated veliparib on a panel of primary and recurrent GBM cell lines derived from patients. We also examined the efficacy of veliparib on temozolomide-induced resistant cell lines to determine the suitability of veliparib as a second line chemotherapy once failure to temozolomide is evident. There are no validated markers to select GBM patients who are most likely going to benefit from this class of drugs.

Results: Consistent with previous studies, monotherapy with ABT-888 was largely ineffective on all of the cell lines. We combined a low dose of ABT-888 (5μM) with radiotherapy (3Gy). Combination treatment was highly efficacious in 4 of the 7 patient derived cell lines tested. To understand whether there were markers that might predict greater sensitivity or greater resistance to veliparib, expression of 96 candidate DNA repair pathway genes were measured using the RT2 Profiler PCR Array (Human DNA Repair; Qiagen). Sensitivity to veliparib in the patient derived lines was characterized by an overexpression of XRCC1, ATM, RAD50, MSH2, PARP1, RAD51 and MREII.

Conclusion: This novel strategy of combining ABT-888 with radiotherapy demonstrated synergistic antitumor activity for the treatment of GBM and relapsed and/or "chemo-resistant" GBM. Sensitivity to veliparib is predicted by an overexpression of DNA repair genes. Further investigation of this synergy has potential for rapid translation into the clinic.

Stem-like Glioblastoma Cells Can Be Targeted With A Novel Radiomimetic Agent With Roles in DNA Double Strand Break Induction and Homologous Recombination Inhibition

Yi Chieh Lim[1], Brett Stringer[1], Kathleen Ensbey[1], Zara Bruce[1], Paul Jamieson[1], Tara Roberts[1], Lisa Wiesmüller[2], Andrew Boyd[1], Bryan Day[1]

1 QIMR Berghofer Medical Research Institute, Brisbane, Australia
2 University of Ulm, Ulm, Germany

Background: Glioblastoma (GBM) is known to limit the efficacy of ionizing radiation (IR) and alkylating-based drug treatment. In GBM cells, the DNA damage response cascade serves as a radioresistance mechanism, promoting cell-cycle arrest and DNA repair to restore genomic integrity and ensure cell-survival. Hence patients undergoing DNA damage-based therapies are likely to relapse or even be refractory to further treatment. In this study, we introduce a new radiomimetic agent that elicits lethal DNA double strand breaks (DSB) and prevents homologous recombination (HR) repair activation.

Methods: Stem-like glioblastoma (GNS) cells were enriched using a serum-free media supplemented with essential growth factors. Surface markers (CD133, EphA3, etc) and neurosphere assay validated the multipotency of GNS cells while colony formation assay confirmed radioresistant survival after IR treatment.

Results: Salinomycin induces DNA strand breakage in which >80% of the GNS cell population undergoes apoptosis. Cell-cycle profiling showed the agent retards GNS cells at late S-phase instead of G2/M as seen with IR treatment. Further investigation indicated salinomycin destabilises Rad51 protein, which is required for DNA strand invasion during HR repair. In agreement with its specificity, salinomycin did not alter non-homologous end joining repair that represents the other major DNA DSB repair pathway. In a murine model, the median survival following receipt of GNS cells with or without IR treatment prior to intracranial engraftment was 103 days and 102 days respectively. In contrast, pre-treated salinomycin significantly inhibited tumour growth (median survival = 133 days).

Conclusion: Current treatment strategies reliance on excessive DNA DSB to achieve apoptosis can be overcome by DNA DSB repair. Our data not only demonstrate a proof-of-principle that HR is essential for GNS cells survival and consequently must be targeted to achieve DNA damage-induced apoptosis, but also identify a new radiomimetic analogue with functions in DNA DSB induction and HR inhibition.

Application of the Response Assessment in Neuro-Oncology (RANO) imaging criteria in high grade glioma in a clinical practice setting: a multi-reader concordance study

Yehia Elhgar[1 4], Sugendran Pillay[1 4], Ramesh Cuganesan[1], Paul de Souza[3 4 5 6], Joseph Descallar[8], George Papadatos[9], Annette Tognela[3 5], Matthias Jaeger[3 5 7], Sam Hall[2], Eng-Siew Koh[4 9]

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5 University of Western Sydney, Sydney, Australia
6 Ingham Institute for Applied Medical Research, Liverpool Hospital, Sydney, Australia
7 Department of Neurosurgery, Wollongong Hospital, Sydney, Australia
8 Department of Biostatistics, Ingham Institute, Liverpool Hospital, Sydney, Australia
9 Department of Radiation Oncology, Liverpool and Macarthur Cancer Therapy Centres, Sydney, Australia

Aim: The 2010 Response Assessment in Neuro-Oncology (RANO) criteria (RC) for high grade glioma was primarily designed for use in a clinical trial setting. This study aimed to 1)assess feasibility of RC application in routine practice; 2)determine whether RC use leads to greater concordance than reporting without application of response criteria; 3)determine practicality of RANO criteria use by registrar-level radiology readers.

Method: Serial MRI datasets were retrospectively collated from 20 newly diagnosed Glioblastoma patients from 2008-2013 in South Western Sydney undergoing surgery and adjuvant chemo-radiation (C-RT). Clinical status and corticosteroid usage to determine RC categories were supplied for each time-point and response categories assigned [complete response (CR), partial response (PR), progressive disease (PD) or stable disease (SD)]. A neuro-radiologist (R1) and radiology registrar (R2) independently scored each dataset according to RC, being blinded to actual reporting radiologist (R3) reports. R3 assessments, completed with limited clinical information and no response criteria applied, were categorized by an oncologist. Concordance between the three readers was documented.

Results: Of 65 MRI datasets reviewed, concordance between R1 and R3 was 40/65 (62%). Concordance between R1 and R2 was 55/65 (85%) and between R2 and R3 was 38/65 (58%). Notably, an imaging response category could be assigned in only 58/65 (89%) of R3 reports versus 100% when RC were applied. Scenarios with multi-focal lesions showed lower reader concordance.

Conclusion: These findings support the feasibility of RANO criteria application in routine practice, with high concordance exceeding that of routine reports, provided relevant clinical data is provided. Concordance between radiologist-level versus registrar reading was moderately high, suggesting uptake by junior staff is feasible, especially with further training, and scoring of complex lesions.

Small increases in enhancement on MRI may predict survival post radiotherapy in patients with glioblastoma

Cecelia Gzell[1,2], Helen Wheeler[1,2], Philip McCloud[1], Marina Kastelan[1], Michael Back[1,2]

1 Northern Sydney Cancer Centre, Royal North Shore Hospital, Sydney, Australia
2 Northern Sydney Clinical School, Sydney University Medical School, Sydney, Australia

Aims: To determine the rate of pseudoprogression (PsP) in glioblastoma patients (GBM) receiving chemoradiotherapy (CRT) using modified RANO and two novel volumetric analysis techniques.

Methods: GBM patients managed with CRT between 2008 and 2011 at the Northern Sydney Cancer Centre were included. Patients with incomplete MRI image sets for all study time points were excluded. Modified RANO and two novel volumetric calculations (volumetric versus rim analyses) were performed on post-operative MRI, and MRIs at 1 month (M+1), 3 months (M+3), 5 months (M+5), 7 months (M+7), and 12 months (M+12) post completion of RT. RANO definitions of response were used for all techniques. The two novel volumetric techniques utilised the ECLIPSE radiotherapy planning software system to calculate the volume of the radiological enhancement with and without the surgical cavity. Increase in enhancement or volume meeting RANO criteria within the first six months of completing RT but reducing or resolving by 12 months was considered pseudoprogression.

Results: 52 patients were included in this retrospective analysis, with median survival (MS) of 18 months (95% CI 15-23). Pseudoprogression was identified in 4 patients (8%) using RANO technique, 4 patients using volumetric, and 2 patients (4%) using rim analysis.

We examined the difference in MS for patients who had a >5% increase versus <5% at the time points M+1, M+3, and M+5 using volumetric analysis. At M+3 and M+5 there was a significant difference for those with <5% increase: M+3 MS 31 v 19 months (logrank test, p=0.005), and M+5 MS 33 v 19 months (logrank test, p=0.004).

Conclusion: This series demonstrated low rates of pseudoprogression (4-8%). An increase in the volume of cavity and enhancement of >5% at M+3 and M+5 post RT was associated with reduced survival, suggesting that increases in radiological abnormality of <25% may predict survival.

Understanding the Supportive and Palliative Care Needs of People with High-Grade Malignant Glioma and their Carers

Anna Collins[1], Carrie Lethborg[2], Michelle Gold[3], Vijaya Sundararajan[4], Gaye Moore[5], Michael Murphy[6], Caroline Brand[7], Jennifer Philip[1]

1 Centre for Palliative Care, St Vincent's Hospital, Melbourne, Australia
2 Social Work Department, St Vincent's Hospital, Melbourne, Australia
3 Palliative Care Service, Alfred Health, Melbourne, Australia
4 Department of Medicine, St Vincent's Hospital & University of Melbourne, Melbourne, Australia
5 Department of Psychiatry, St Vincent's Hospital, Melbourne, Australia
6 Department of Neurosurgery, St Vincent's Hospital, Melbourne, Australia
7 Melbourne EpiCentre, University of Melbourne & Melbourne Health, Melbourne, Australia

Aims: People diagnosed with high-grade primary malignant glioma (HGG) experience a high symptom burden, characterised by an uncertain prognosis, rapid decline of physical function and behavioural and neuro-cognitive changes. This qualitative study explored the needs of patients with HGG and their carers to inform the development of a model which seeks to improve quality of care.

Methods: Consecutive patients and their informal carers were approached through neurosurgery, oncology and palliative care units of two metropolitan hospitals. Bereaved carers were identified by health care professionals (HCPs) and invited by letter to participate. Semi-structured interviews were conducted by a single interviewer until data saturation. HCPs from two metropolitan hospitals and one community palliative care service were invited to participate in multi-disciplinary focus groups. Interviews and focus groups were recorded and transcribed, and thematically analysed by three investigators.

Results: Interviews were held with 10 patients, 23 carers (15 active, 8 bereaved), and 6 focus groups involving 35 HCPs (13 nurses, 11 doctors and 11 allied health). Carers reported difficulty managing subtle behavioural and personality changes, cognitive decline and diminished insight. Patients described difficulty accessing and navigating complex care services. Meanwhile carers had needs for information that conflicted with patients' needs for hope. HCPs reported difficulty in planning care as fluctuations in functional state were common, and they were concerned about challenging patients' hopes. Offers of support tended to be reactive and only when patients expressed need or marked disability rather than offered routinely.

Conclusions: Coordination of care across hospital and community through an identifiable, accessible and informed HCP appears critical. This would ensure proactive services and staged information provision appropriate to disease state, assist navigation through health services and ensure palliative care services are better integrated into patient and family care.

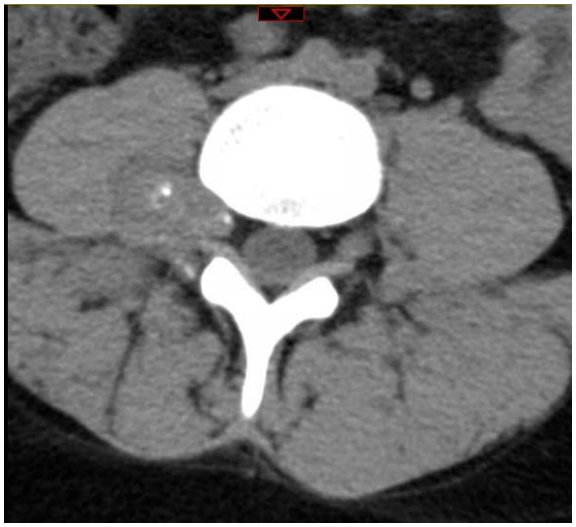
POSTER ABSTRACTS

Synovial Sarcoma of L4 exit foramen and paraspinal muscles

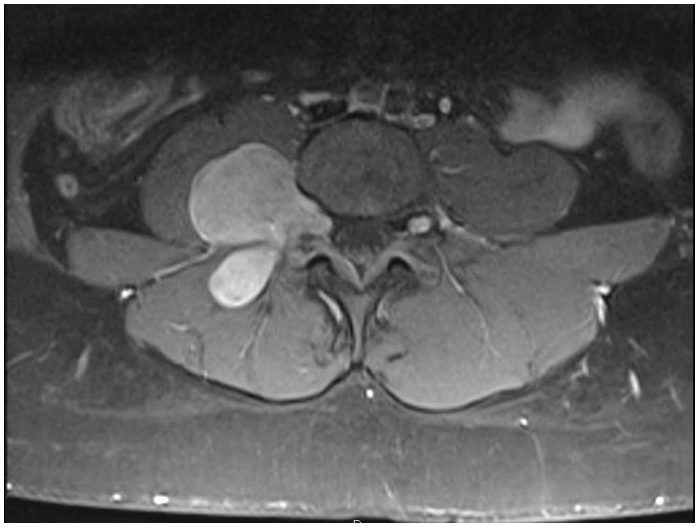
Anmar Al-Witri[1], James Dimou[1], Loui Rassam[1], John Christie[1]

1 John Hunter Hospital, Newcastle, Australia

Synovial sarcoma (SS) accounts for approximately 10% of all soft tissue sarcomas, but reports of SS affecting the axial spine are sparse. We report a 23 year old female who presented with a five year history of progressively worsening right lower limb pain, predominantly in an L4 dermatomal distribution. Imaging demonstrated a right-sided L4/5 foraminal lesion extending into the adjacent erector spinae and psoas major, initially reported radiologically as a likely schwannoma. Surgical debulking was performed and histopathological examination infact revealed classic features of SS with X;18 chromosomal translocation. Standard chemotherapy and radiotherapy were administered, followed by a second en bloc resection, and no evidence of recurrence on follow up imaging has been noted since, over the ensuing eighteen month period. Whilst our patient has experienced good tumour control, the gold standard of surgical management for SS remains en bloc resection without debulking or pre-operative needle biopsy, and unusual lesions of the axial spine seen on thorough radiological evaluation ought to prompt consideration of SS as a possible differential diagnosis, in order to optimise surgical planning and resection, and improve the efficacy of adjuvant treatments.



CT Lumbar spine Axial L4



MRI Lumbar spine axial

A meta-analysis of anti-angiogenic therapy for glioblastoma (GBM)

Malaka Ameratunga[1], Andrew Lassman[2], Nick Pavlakis[3], Mustafa Khasraw[4]

1 Olivia Newton John Cancer Centre, Austin Health, Melbourne, Australia
2 Department of Neurology and the Herbert Irving Comprehensive Cancer Center, New York, USA
3 Department of Medical Oncology, Royal North Shore Hospital, Sydney, Australia
4 Andrew Love Cancer Centre, Geelong, Australia

Aims: To evaluate the efficacy, in terms of overall survival (OS) and progression-free survival (PFS), of anti-angiogenic therapy (AAT) in glioblastoma (GBM).

Methods: A search was conducted of the Cochrane Central Register of Controlled Trials (CENTRAL), Medline and EMBASE to find randomised controlled trials (RCTs) from 2000 to April 2014 evaluating AAT in GBM versus control treatment without AAT. Pooled estimates of treatment effect on OS and PFS were calculated using the fixed-effects inverse variance weighted method.

Results: Seven RCTs were identified including 2987 patients. Significant design heterogeneity was noted in the included studies, especially in the response assessment criteria used. Four studies were only available in abstract form. The pooled hazard ratio (HR) of the seven studies for OS was not significant at 0.94 (95% confidence interval (CI) 0.86–1.02, p=0.16). There was, however, an improved PFS, with a HR of 0.74 (95% CI 0.68–0.81, p<0.00001). Bevacizumab was the AAT most likely to yield favourable results, with a pooled HR for PFS from three studies (n=1712) of 0.66 (95% CI 0.59–0.74, p<0.00001). Meta-analysis of five studies of AAT in the adjuvant setting (n=2522) did not demonstrate an improved OS, with a HR of 0.92 (95% CI 0.84–1.01, p=0.10) but showed a significantly improved PFS with a HR of 0.72 (95% CI 0.66–0.79, p<0.00001). In the recurrent setting, a meta-analysis of 2 studies (n=465) did not demonstrate an improved OS with a HR of 1.02, (95% CI 0.84–1.24). There was inadequate data to perform a meta-analysis of PFS in the recurrent setting.

Conclusions: AAT does not appear to improve OS in the adjuvant setting, but does appear to prolong PFS. There is a need for adequately powered, randomised placebo-controlled studies in the recurrent setting. A more detailed analysis is prepared as a Cochrane systematic review.

Infinium HM450K analysis of DNA methylation in malignant glioma

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Aims: Human high grade glioma (HGG) is a heterogeneous disease. The aim of this study was to profile genome-scale DNA methylation in glioma and to determine whether specific DNA methylation biomarkers could be identified that were associated with disease progression and prognosis.

Methods: Our study cohort comprised 24 tumour specimens with extensive associated clinical annotation. We performed DNA methylation profiling on matched primary and relapse tumour DNA extracted from formalin –fixed specimens on a HM450K platform (Illumina). We used a multiple analysis pipelines to identify DNA methylation profiles associated with disease progression, prognosis and relapse. Genomic copy number analysis was performed in parallel.

Results: We identified patient-specific differential DNA methylation changes between matched primary and relapse tumour samples. Using array probe intensity to infer genomic copy number, we were able to confirm EGFR amplification in 5/24 patients analysed. However we found no recurrent DNA methylation differences associated with disease progression or prognosis.

Conclusions: While there were patient specific differences in DNA methylation between matched primary and relapse tumour samples no consistent differences were identified in the cohort. We are now examining gene networks to determine whether common pathways are affected.

What are the risk factors for glioma in adults? A review of the epidemiological evidence

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Aims: Therapeutic ionizing radiation, rare genetic syndromes, and family history are the only established risk factors for glioma. In the past five years there has been a vast increase in glioma aetiology research as cohorts have matured and consortia have pooled data. We aimed to summarise the evidence for putative risk factors for glioma in adults, with an emphasis on emerging trends.

Methods: We reviewed published observational studies examining one or more risk factors for adult glioma. Studies were identified from PubMed and Medline searches and hand-searching of reference lists.

Results: There is consistent evidence of an inverse association between glioma risk and history of atopic or allergic disease, higher immunoglobulin E levels, and SNPs associated with atopy and inflammation. Recent evidence is mixed but on balance is suggestive of a positive association with radiofrequency electromagnetic fields from wireless phones (mobile phones and cordless phones) in some subgroups. Longer follow-up is warranted to validate these trends. Potential risk factors include alcohol consumption, history of diabetes, height, and female reproductive hormones. There are suggestions of elevated rates of glioma in higher socioeconomic regions. There is no or very limited evidence for an association between risk of glioma and herpesviruses, diet, and occupation. Genetic analyses have revealed variants near TERT and TERC, which influence telomere length, are associated with high-grade glioma risk. In addition, variants residing in the DNA repair genes may also predispose to glioblastoma risk. The consistency and strength of the evidence and study limitations will be presented.

Conclusions: Epidemiological studies are now yielding some consistent findings that have the potential to inform biological investigations and ultimately preventative strategies. The interaction of epidemiological and genetic leads hold great potential.

Socio-economic and lifestyle predictors of survival in high grade glioma – analysis of the Australian Genomics and Clinical Outcomes of Glioma Epidemiological Questionnaire

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Aims: Despite advances in the treatment of high grade glioma (HGG), overall survival (OS) remains poor and heterogeneous. Pre-morbid socio-economic and lifestyle factors are associated with survival following the diagnosis of many cancers yet have been poorly explored in HGG. We

sought to clarify the role of these factors using the Australian Genomics and Clinical Outcomes of Glioma (AGOG) multicentre patient cohort.

Methods: Sixteen individual and population level socio-economic and lifestyle variables were extracted from responses to the AGOG questionnaire completed by consenting patients with HGG (Table 1). Overall survival was measured from the first available histopathological diagnosis to the date of death. Survival analysis was performed using the Kaplan-Meier method. Univariate and multivariate analyses were performed to elucidate factors associated with OS.

Results: Requisite data was available for 336 patients; their socio-economic and lifestyle characteristics are presented in Table 1. Younger age at diagnosis, histological grade III, belonging to the lowest Index of Relative Socioeconomic Advantage and Disadvantage quintile by residential post-code, highest level of education being above year 9, pre-diagnosis television/radio use >1 hour per day and pre-diagnosis alcohol consumption were associated with prolonged OS on univariate analysis. In addition to age and histological grade, only pre-diagnosis alcohol consumption was independently associated with prolonged survival ($p<0.001$, Table 2, Figure 1).

Conclusions: In this Australian cohort of HGG patients, socio-economic status and most lifestyle factors were not associated with survival. The observed independent association between pre-diagnosis alcohol consumption and prolonged survival has never before been described. It may reflect the established association between good pre-diagnosis functional status and prolonged survival, early symptomatic presentation of HGG in the context of a vulnerable brain due to chronic alcohol use or the putative pro-survival effect of a ketogenic diet in HGG given the known ketogenic effect of alcohol consumption.

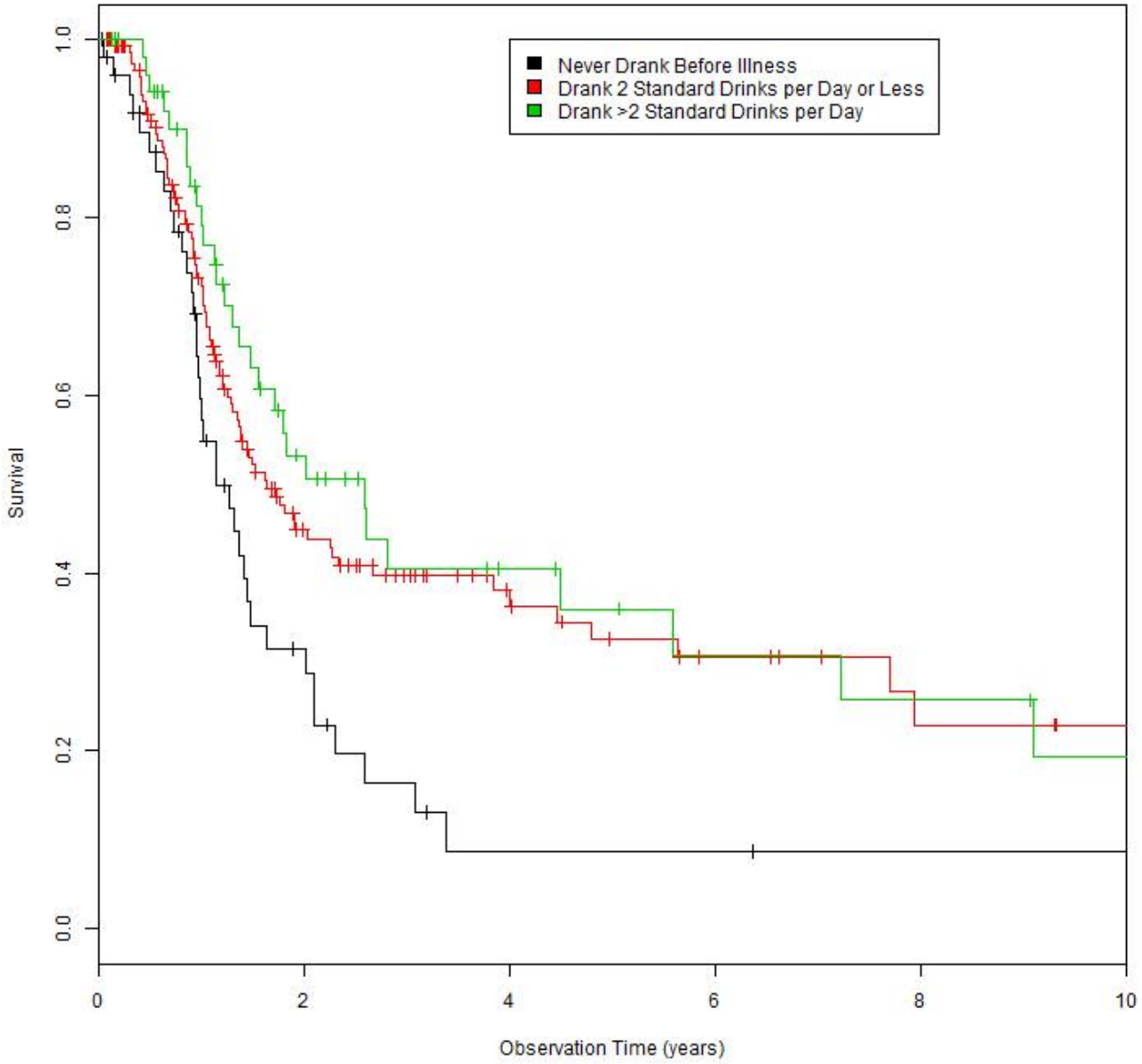

Figure 1: Kaplan-Meier plot of overall survival following a diagnosis of high grade glioma by the level of pre-diagnosis alcohol consumption.

Variable	No. of Patients (%)
Age at Diagnosis	
<40	53 (16)
40-50	57 (17)
50-60	109 (32)
60-70	78 (23)
70+	39 (12)
Missing	0 (0)
Gender	
Male	218 (65)
Female	118 (35)
Missing	0(0)
Histological Grade/Type	
Three	80 (24)
Astrocytoma	50 (15)
Oligodendroglioma	20 (6)
Oligoastrocytoma	6 (2)
Other	4 (1)
Four (GBM)	256 (76)
Missing	0(0)
IRSAD Quintile	
First (Lowest)	12 (3.6)
Second	16 (4.8)
Third	50 (15)
Fourth	65 (19)
Fifth (Highest)	113 (34)
Missing	80 (24)
Annual Household Pre-Tax Income	
<$10 000	12(3.6)
$10000-$25000	23 (6.8)
$25000-$50000	53(16)
$50000-$75000	54 (16)
$75000-$100000	43 (13)
$100000-$125000	18 (5.4)
>$125000	41 (12)
Missing	92 (27)
Remoteness Area	
Major City	196 (58)
Inner Regional	33 (9.8)
Outer Regional	18 (5.4)
Remote and Very Remote	9 (2.7)
Missing	80 (24)
Ethnicity	
Solely Caucasian	272 (81)
Not Solely Caucasian	64 (19)
Missing	0 (0)
Highest Level of Education Attained	
Year 9 or Below	35 (10)
Year 10/11/12/Vocational	204 (61)
University Enrollment	90 (27)
Missing	7 (2)
Pre-Diagnosis Television/Radio Use	
Less than 3 hours per week	11(3.3)
One hour or less per day	19 (5.7)
One to 2 hours per day	61(18)
Two to three hours per day	91(27)
More than 3 hours per day	81(24)
Missing	73(22)

Variable	No. of Patients (%)
Pre-Diagnosis Computer Use	
Never	55(16)
<3 hours/week	36(11)
<1hour/day	28(8)
1-2 hours per day	28(8)
2-3 hours per day	14(4)
>3 hours per day	102(30)
Missing	73(22)
Pre Diagnosis Reading Frequency	
Never	10 (3)
<1 per week	19 (6)
Once per week	11 (3)
2-3 per week	31 (9)
Most days	100 (30)
>Once per day	93 (28)
Missing	72 (21)
Body Mass Index - Current	
Not overweight	104 (31)
Overweight	80 (24)
Obese	131 (39)
Missing	21 (6.3)
Body Mass Index - Largest Recalled	48 (14)
Not Overweight	94 (28)
Overweight	101 (30)
Obese	93 (28)
Missing	
Waist Circumference	
Not Elevated	74 (22)
Elevated	166 (49)
Missing	96 (29)
Lifetime Smoking Status	
Never Smoked	163 (49)
Past Smoker	116 (35)
Current Smoker	52 (15)
Missing	5 (1.5)
Smoker ≥100 Lifetime Cigarettes	
No	21 (6.3)
Yes	159 (47)
Missing	156 (46)
Pre-Diagnosis Alcohol Consumption	51 (15)
Never Drank Before Illness	154 (46)
Drank ≤2 Standard Drinks per Day	55 (16)
Drank >2 Standard Drinks per Day	76 (23)
Missing	
Exercise	
Nil	10 (3.0)
Below Recommended Level	143 (42)
At or Above Recommended level	108 (32)
Missing	75 (22)

Table 1: Baseline clinical characteristics of the cohort

Variable	Hazard Ratio	95% Confidence Interval	Multivariate p-value	Absolute Median Survival (years)	95% Confidence Interval	No. Events/No. Started
Age at Diagnosis						
<40	1	Referent	Referent	6.23	2.91-NA	23/53
40-50	1.05	0.532- 2.08	0.886	2.67	1.91-9.91	32/57
50-60	1.35	0.728- 2.51	0.339	1.82	1.46-3.08	66/109
60-70	1.83	0.959- 3.50	0.0668	1.09	0.953-1.39	55/78
70+	2.59	1.29- 5.23	<0.01*	1.19	1.01-1.40	34/39
Histological Grade						
Three	1	Referent	Referent	7.69	6.23-NA	32/80
Four (GBM)	4.84	2.84 -8.27	<0.001 *	1.28	1.15-1.48	178/256
Pre-Diagnosis Alcohol Consumption						
Never Drank Before Illness	1	Referent	Referent	1.15	0.98-1.63	36/51
Drank ≤2 Standard Drinks per Day	0.479	0.321-0.714	<0.001 *	1.64	1.36-2.67	85/154
Drank >2 Standard Drinks per Day	0.514	0.312 - 0.844	<0.001 *	2.59	1.56-7.23	29/55

Table 2: Final multivariate model of independent survival predictors using Cox proportional hazards regression analysis with stepwise backward elimination

ANZMTG 01.07 WBRTMel: Whole Brain Radiotherapy Following Local Treatment of Melanoma Brain Metastases – Interim Analysis

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7 Radium Hospital, Oslo, Norway
8 Princess Alexandra Hospital,Brisbane, Australia
9 Trans-Tasman Radiation Oncology Group (TROG), Newcastle, Australia
10 Australia and New Zealand Melanoma Trials Group (ANZMTG), North Sydney, Australia
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12 Psycho-Oncology Co-Operative Research Group (PoCoG), School of Psychology, Faculty of Science, University of Sydney, Sydney, Australia
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Aims: Whole brain radiotherapy (WBRT) following local treatment of intracranial melanoma metastases is controversial. The Australia and New Zealand Melanoma Trials Group (ANZMTG) and Trans-Tasman Radiation Oncology Group (TROG) are conducting a randomised, international, single histology trial of 200 patients comparing WBRT with observation. The primary endpoint is distant intracranial failure on magnetic resonance imaging (MRI) at 12 months post randomisation. Here we present the first planned interim analysis.

Methods: Data to 12 months from randomisation for the first 100 patients was reviewed for quality and events. Whilst trial results remain blinded to clinicians, unblinded results were presented to the independent Data Safety Monitoring Committee (DSMC), alongside the safety data for all participants.

Results: Histology review confirmed all patients had stage IV melanoma and were appropriately included. Protocol schedule compliance was high as was accuracy of reporting intracranial failure. A total of 47 distant events were correctly reported; 34 of these were distant failure alone and 13 were accompanied by local failure (Figure 1). Completion of the Quality of Life component was 90.7%, a high completion rate. For neurocognitive function (NCF) assessments 68% of expected assessments have been received. Where assessments were not performed, valid reasons were given. Minimal numbers of adverse events (AEs) were reported (Grade 3: 44, Grade 4: 3) and there have been no serious adverse events. Of 100 patients analysed, 53 died within the first 12 months with 33 reporting intracranial failure as the cause.

Conclusions: The DSMC reviewed the data, commenting on the excellent quality. No protocol specified stopping rules were met and trial treatment appears safe. The DSMC recommended the trial continue. The data provision and quality indicates a reliable outcome will be obtained when the final analysis is performed. Accrual is currently ongoing with 144 (Figure 2) patients randomised at present.

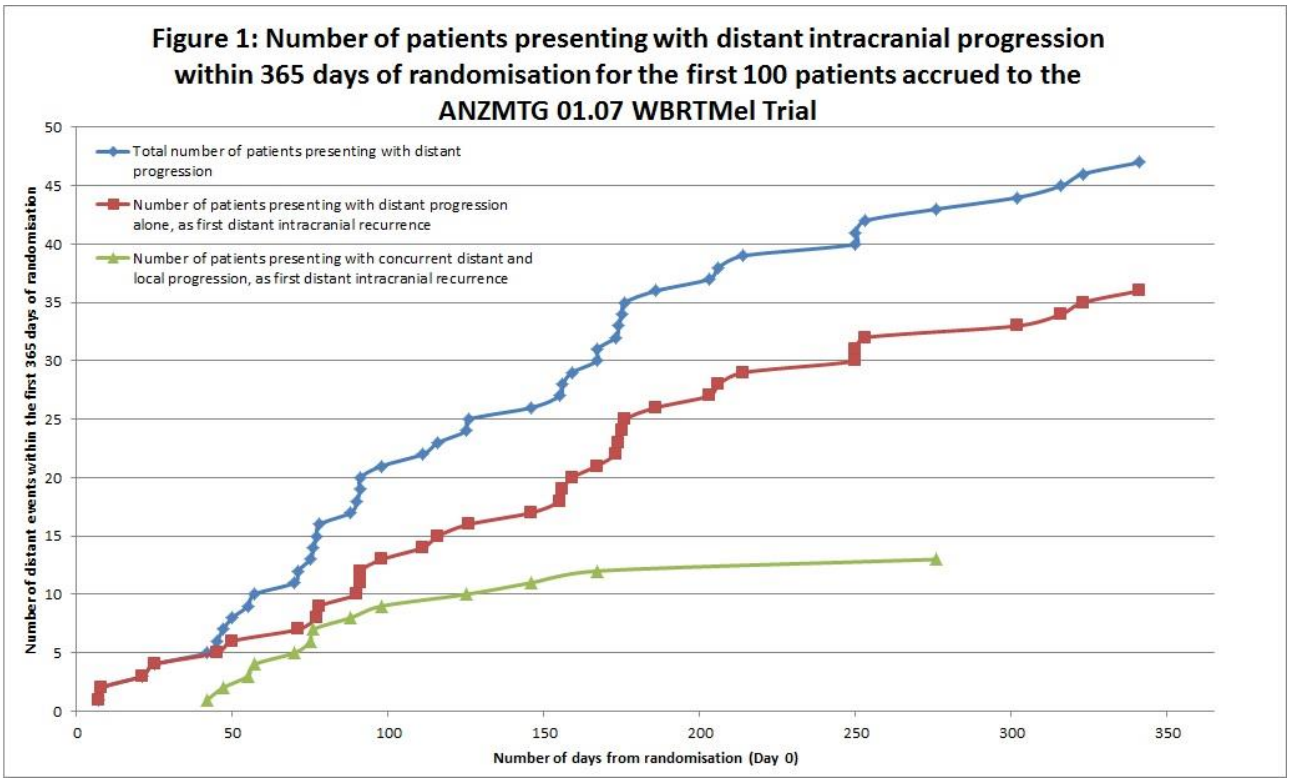

Figure 1

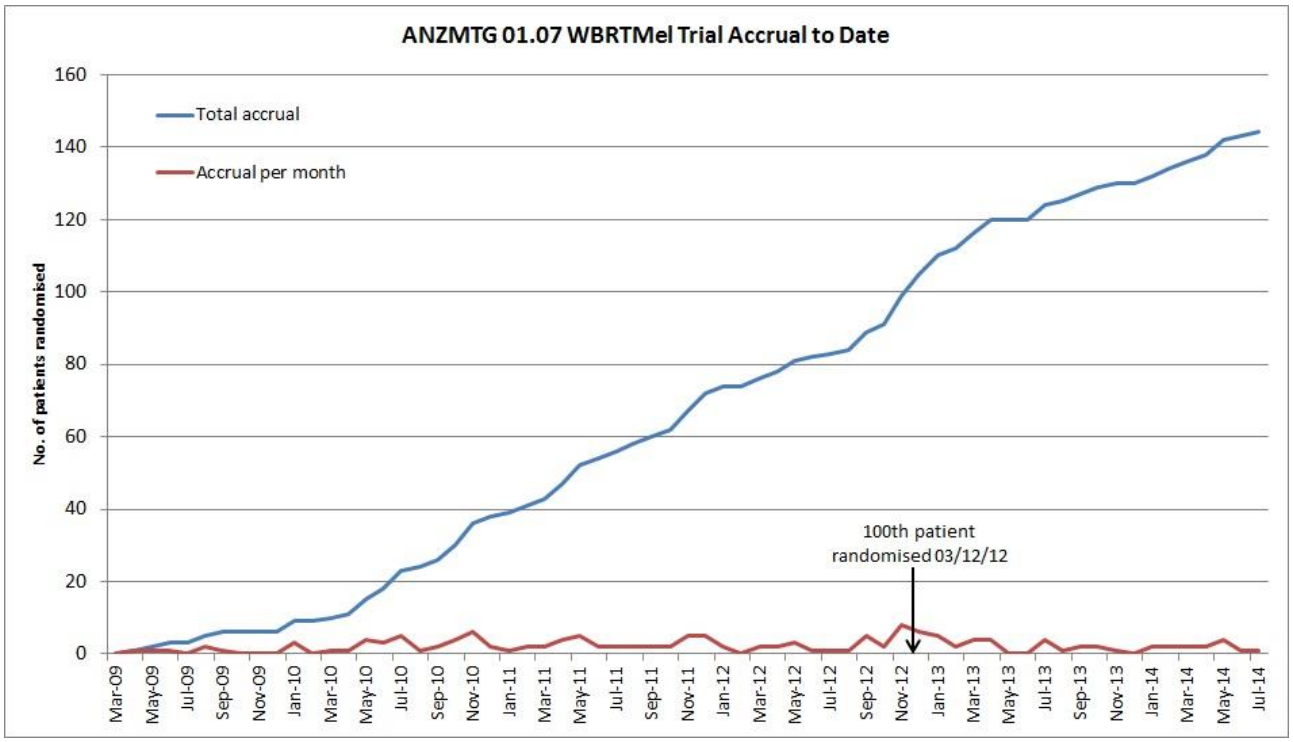

Figure 2

Applying functional imaging in brain tumour clinical trials

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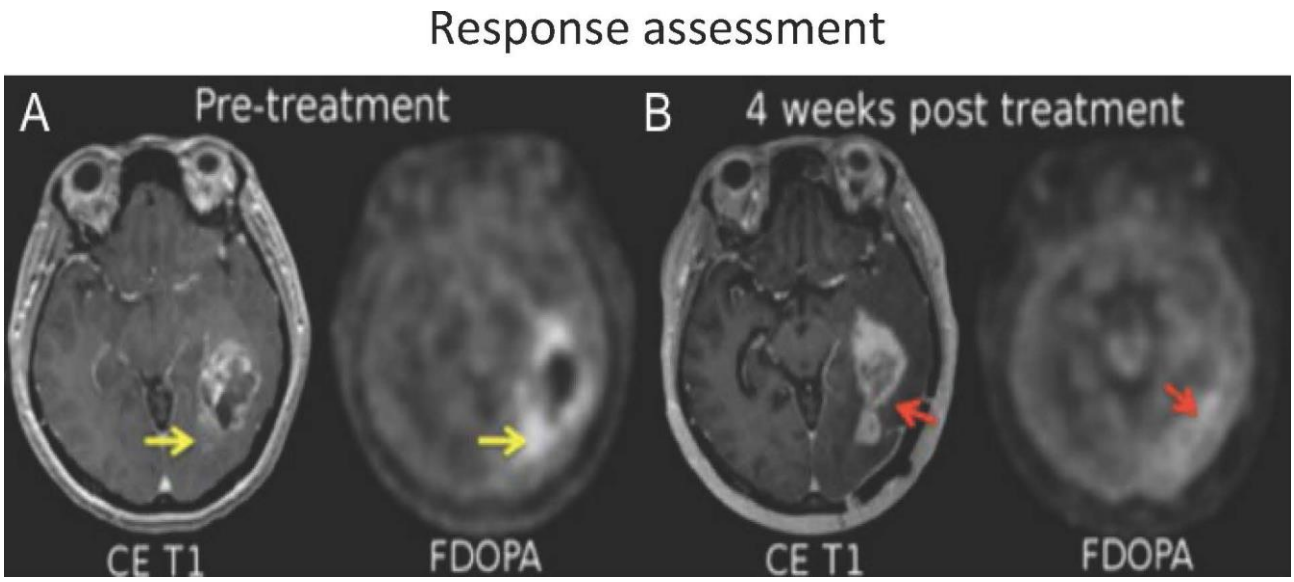
Clinical trials in high grade brain tumours are complicated by assessment of endpoints and short survival. Conventional imaging performs poorly after surgery or radiotherapy.

Aim: To develop methods to assess the use of new treatments in combination.

Methods: Our group has been investigating the use of combined conventional and functional imaging using non-FDG tracers and advanced computational methods.

Outcome: We believe this could become a useful way of assessing oncology treatments and combinations and will present our early results.



Benefits of a Neuro-Oncology Nurse Program

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Aims. The position of a Neuro-Oncology support co-ordinator within brain tumour units is becoming more widespread. There is no standard model of care for this position. Although it is clear that these positions are beneficial, there is little hard data to demonstrate this benefit to hospital administrations, who ultimately decide on funding for this role.

Methods. A Neuro-Oncology Nurse role was established at Monash Medical Centre in 1997, with a Palliative Care nurse taking that position. In many ways palliative care for these patients begins at diagnosis, and the palliative care background has been of great benefit with the changing needs of our patients towards the later stages of their illness. This model was reproduced at Cabrini Hospital, an acute private hospital in Melbourne, in early 2012. The Cabrini health service includes a palliative care hospital and a rehabilitation hospital, which has helped us coordinate services for our patients.

Results. We have compared the admission profile of patients in the 2 years before and the 2 years after the establishment of the Neuro-Oncology Nurse position at Cabrini. Although there were 10% more patients treated in the 2 years after the Neuro-Oncology Nurse position commenced (115 vs 105 patients), there was a 24% reduction in the total bed days in the acute private hospital (393 less bed days over 2 years), as well as more admissions to subacute care (2/3 to palliative care and 1/3 to rehabilitation), and a reduction of the average length of stay by 6% in the acute hospital and 8% in palliative care.

Conclusions. This data demonstrates the benefits of the Neuro-Oncology Nurse position to the health service. It also indicates some of the benefits to our patients, with a reduction in time spent in the acute hospital setting and improved access to continuing care services.

Delineating the cellular hierarchy of glioblastoma and gliosarcoma for identification of therapeutic targets

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Aims: We compared the cellular hierarchy of glioblastoma (GBM) and gliosarcoma (GSM) to identify molecular determinants of tumour growth and identify potential therapeutic targets. Glioma stem cells (GSC) are responsible for treatment resistance and recurrence. We postulate that there are differences in the GSC gene expression between GBM and GSM.

Methods: qRT-PCR analysis of gene expression was performed on frozen human GBM and GSM specimens, 6 human GBM and 1 GSM cell lines. Genes tested included pluripotency-controlling transcription factors (TFs) and genes associated with ectodermal, mesodermal and endodermal cell differentiation.

Results: Of the pluripotency TFs, SOX2 was the most highly expressed. OCT4 and NANOG were expressed at levels 90-180 times lower than SOX2. Downstream TF targets ESRRB, PROX1, NESTIN and PAX6 showed variable expression. We also detected high expression of the estrogen-related receptor gamma (ESRRG). Neural genes MAP2 and GAP43 and glial genes GFAP and S100B showed variable expression. Epithelial signatures (cytokeratin 18) were detected in GSM and to a lesser extent in GBM. Expression of E-cadherin was significantly higher in GBM than in GSM ($P<0.05$), suggesting greater cell motility in GSM. Endodermal signatures (GATA4) were more highly expressed in GBM. GSC-associated marker CD133 was highly expressed in both frozen GBM and GSM, but less so in the cell lines. Overall, GSM showed greater variation in gene expression than GBM.

Conclusions: Amongst others, SOX2 and ESRRG were identified as promising therapeutic targets. The presence of neural, epithelial and endodermal signatures suggests multipotential lineage of GSCs. The presence of different lineages in GSM reflects its greater heterogeneity, providing evidence that GSM is a different disease and highlights the need for patient-tailored treatments. Additionally, the differences in CD133 expression between the cell lines and patient samples suggest alterations mediated by immortalisation, and cautions against the use of cell lines for functional tests.

Treatment related reduction in platelet count as a prognostic factor in glioblastoma multiforme

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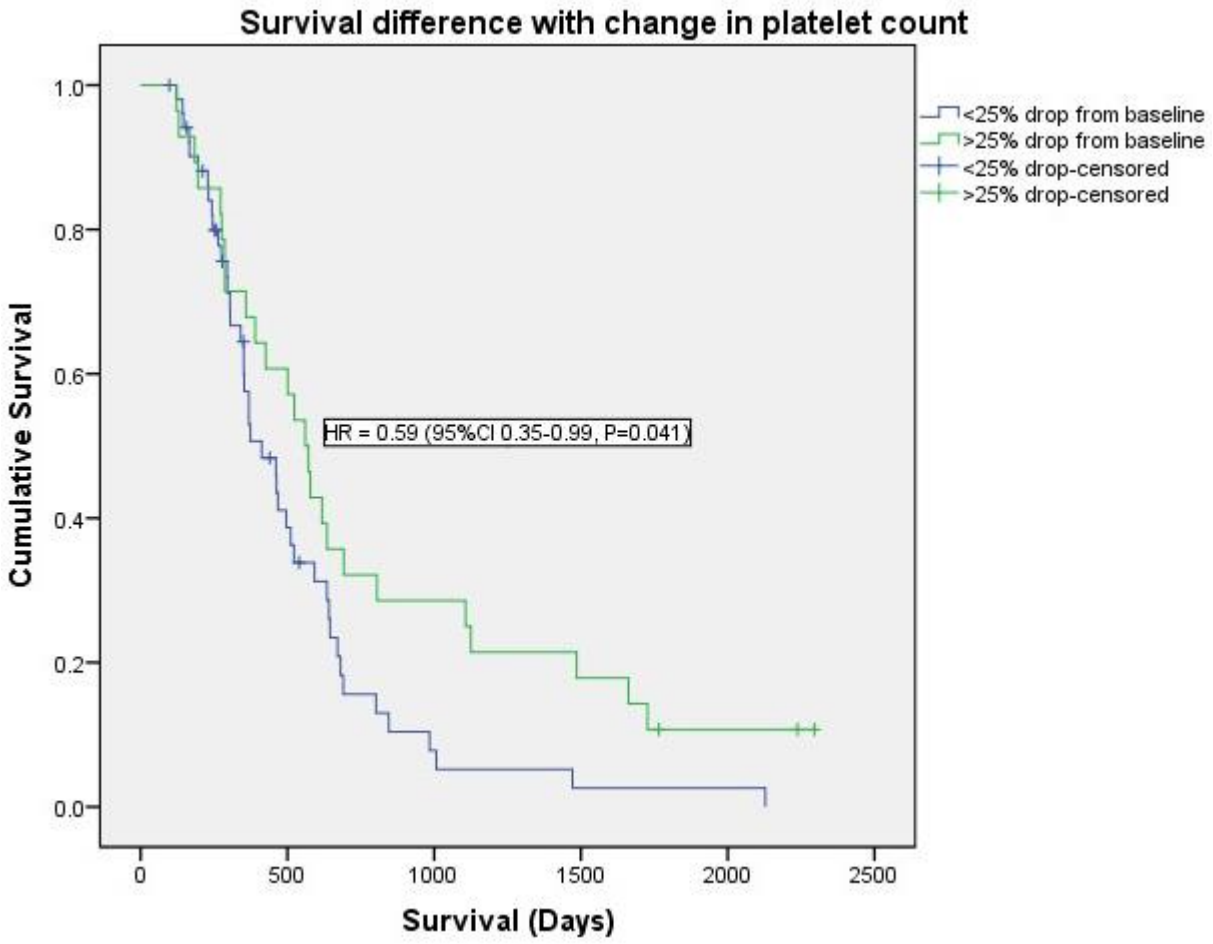
Aims: The standard of care for the treatment of glioblastoma multiforme (GBM) requires multimodal treatment including surgery, chemoradiotherapy and cyclical chemotherapy. Haematological toxicity is observed during concurrent chemoradiotherapy with temozolomide. Retrospective studies suggest that this is a possible prognostic factor for overall survival. We sought to determine whether or not haematological toxicity may be a surrogate marker for survival.

Methods: We conducted a retrospective analysis of 80 patients treated at Crown Princess Mary Cancer Centre Westmead who received concurrent chemoradiotherapy between 2007 and 2014. Haematological parameters were measured at baseline, during concurrent chemoradiotherapy and prior to adjuvant chemotherapy. Treatment groups were defined according to the degree of decrease in each parameter over the treatment period. Kaplan-Meier survival curves were calculated for each subgroup.

Results: Decreases in leucocytes, neutrophils and monocytes did not correlate with survival time. Patients who had a 10% or greater decrease in platelet counts during concurrent chemoradiotherapy had a trend towards a significant improvement in overall survival. Patients who had a 25% or greater drop in platelet count during concurrent chemoradiotherapy had a higher median survival time of 561 days (95% CI 461-660) compared with 412 days (95% CI 298-525) in those with a less than 25% drop (HR 0.59, 95% CI 0.35-0.99, p=0.041)(Fig 1). Overall survival did not correlate with the grade of haematological adverse reactions.

Fig 1 - Kaplan-Meier survival curves of patients with < 25% and >25% drop in platelet count during concurrent chemoradiation treatment.

Conclusions: This study supports the previously published literature that treatment related reduction in platelet count during concurrent chemoradiotherapy is a possible prognostic factor for patients with GBM. Prospective studies to better understand the mechanisms are warranted.



Use of supportive services by adults recently diagnosed with primary brain tumours in Queensland

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Aims: Primary brain tumours are associated with significant physical, neurological, cognitive and psychosocial morbidity. Australian treatment guidelines recommend multidisciplinary rehabilitation and psychosocial care to address patient needs, but the extent to which patients utilise services to address their needs is unclear. This study aimed to describe primary brain tumour patients' awareness of, referral to and use of services.

Methods: A representative sample of 40 adults recently (<3 months) diagnosed with primary brain tumours were recruited through the Queensland Cancer Registry. Patients or carer proxies reported their awareness of, referral to and use of 32 informational, support, health professional or practical services. Service utilisation was summarised using descriptive statistics.

Results: All or almost all participants were aware of at least one informational (100%), health professional (100%), support (97%) or practical (94%) service. Awareness of individual services was highest for speech therapists (97%), physiotherapists (94%) and diagnostic information from the internet (88%), and lowest for the Brain Tumour Alliance Australia (BTAA, 0%), exercise physiologists (19%), and neuro-psychologists (22%). Referrals were most commonly made to physiotherapists (53%), speech therapists (50%) and diagnostic information booklets (44%). In contrast, no participants reported being referred to BTAA, exercise physiologists, psychiatrists, financial counsellors or advisors, or a wig and turban service. Services most commonly used were physiotherapists (56%), diagnostic information booklets (47%), diagnostic information from the internet (47%), and speech therapists (43%).

Conclusions: Awareness, referral and utilisation of services were highest for physical support and information, and lower for psychological support and practical services. Given the significant psychosocial burden of a brain tumour diagnosis, further understanding of barriers to referral and access, and education to improve awareness of psychosocial services and reduce stigma may be needed to improve service use and consequently, reduce patients' unmet needs.

Capitalising on a unique opportunity: establishing the Brain Tumour Support Officer role at Austin Health

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Brain tumour patients and families face enormous challenges that are unmet in usual clinical practice. We describe the implementation of a cost effective support program, funded by philanthropy. With limited resources, the model was defined by drawing on the literature, identifying the critical areas of unmet need and meeting with key stakeholders within hospital and consumer communities. Service gaps were identified and strategies developed to address these elements in the most efficient way.

Direct patient service commenced in late 2008, with the Brain Tumour Support Officer (BTSO) assisting 40 families in the first 12 months. In 2013, the BTSO provides a range of support services accessed by over 130 families annually. The BTSO also plays active role in community education and awareness activities for consumers and health professionals. The BTSO model is designed to enable patients and families to be supported through their healthcare journey from diagnosis, through their treatment, and beyond. This contrasts with a nurse practitioner model, separated from but integrated with medical care. The key strategies are informing, resourcing, supporting and acknowledging the impacts at different stages. 47% of interventions are individual consultations; group interventions account for 21 % and phone-based consultations make up 23%.

Challenges along the way have included management of staff & patient expectations, avoiding the pitfall of being all things to all people. It has also been critical to engage key medical staff, peak bodies & patient advocacy organisations, to ensure all activities are relevant and endorsed.

This is a unique opportunity to work across boundaries of private/public health, in partnership with a philanthropic fund passionate about their cause. We have attempted balance between individual support and resourcing, whilst building a range of awareness and educational activities creating a natural impetus within the broader health community and beyond.

Regional support network for primary malignant brain tumours

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2 Epworth Hospital, Richmond, Australia

Background: Supportive care needs of patients and families affected by brain tumours are challenging and unique. Coordinating skilled support in rural areas is difficult, when only small numbers of patients present. This project explored ways of facilitating skill development in health practitioners, enhancing professional networks between regional and city based brain tumour support personnel.

Methods: Following a comprehensive review of educational needs, including liaising with key stakeholders, two primary strategies where implemented:

1) A clinical education day for regional and metropolitan health professionals focusing on brain tumours, treatment options, complex supportive care needs and available resources.

2)A day placement for regional health professionals with the Austin Brain Tumour Support Service.

Results: The clinical education day attracted 116 participants, with 31% travelling from regional areas. 56% of participants were nurses and 23% from allied health backgrounds. 65% of attendees were referred <10 brain tumour patients annually and 44% rated their understanding of brain tumours as fair to poor. The overall evaluation response rate was 81%. Delegates rated content on a 5 point scale, with all presentations rating greater than 4.21. 91% of respondents felt that the forum met their aims for attending and 100% would attend a similar forum if held annually. Day placement program was conducted with 2 staff from regional Victoria, both rated the experience positively. Post placement surveys indicated improved confidence in needs identification, increased ability to provide practical strategies to assist and seek support.

Conclusion: Despite the small number of people diagnosed with brain tumours in Victoria, this project demonstrated a strong need for ongoing professional development in this challenging area particularly in regional areas. Through collaboration with Cancer Council Victoria, ongoing planned initiatives include an annual brain tumour professional day, mentor program and a newsletter for health professionals focused on brain tumours.

Role of Focal Adhesion Kinase in Glioma Stem Cell Migration

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Background: Despite standard chemotherapy with temozolomide plus radiotherapy, Glioblastoma (GBM) has a poor median survival of 14 months. LPA was shown be a driver of migration and invasion of glioma. Focal adhesion kinase (FAK) is a 125kDa non-receptor kinase that mediates cell-matrix interaction as well as growth, survival, and migration in cancers. Using an in vitro glioma stem cell (GSC) model, we have previously shown that FAK and phosphorylated FAK is over-expressed and that specific inhibition of FAK by a kinase inhibitor Y15, unlike temozolomide, prevents neurosphere formation and leads

to apoptosis. Meanwhile, LPA1 has been shown in our lab to be unregulated in the GSC lines.

This study examined the involvement of LPA in FAK activation and cellular motility and subsequently the effect of FAK inhibitor Y15 in LPA-induced migration and invasion in primary glioma stem cell lines derived from GBM patients.

Methods: Three different primary patient-derived glioma stem cell lines are used. Cellular protein expression of total- and phosphorylated FAK was analysed by Western Blotting. Lactate dehydrogenase assay was employed to determine the cytotoxicity of Y15 on GSC. Cellular migration and invasion are quantified by transwell migration assay and 3D spheroid invasion assay respectively.

Results: Treatment of 1,2,4,5-benzenetetraamine-tetrahydrochloride (Y15), a FAK autophosphorylation inhibitor reduces GSC's LPA-induced motility and invasion in a dose-dependent manner, corresponding to the decrease in phosphorylation of FAK.

Conclusions: LPA-FAK axis is required in glioma stem cell migration and invasion, serving as a potential therapeutic target in controlling the invasive behavior of glioblastoma.

Survival outcome of multiple resections in patients with glioblastoma multiforme (GBM) in the South Australian population

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Objectives: Surgical reresections have been reported to prolong survival in patients with glioblastoma multiforme (GBM). This is a retrospective analysis of a single centre in Australia to compare the survival outcome of patients with GBM who underwent surgical reresections to those treated with single resection.

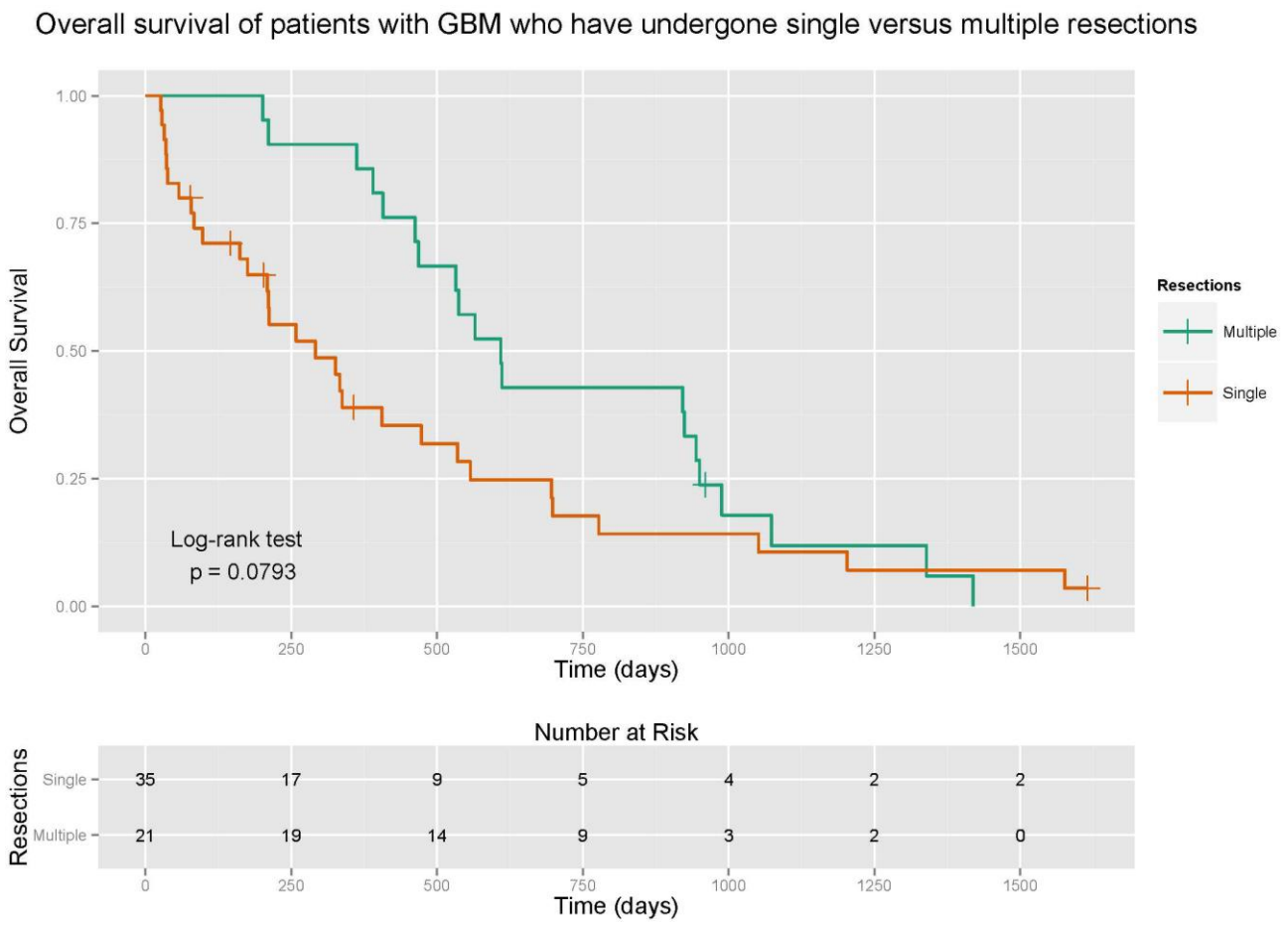
Methods: The records of adult patients who underwent surgery for GBM at a tertiary hospital in the year 2010- 2011 were retrospectively analysed. The included variables were age, date of diagnosis, functional status, tumour location and size, extent of resection, adjuvant therapy, number of resections, perioperative complications and date of last follow-up. Kaplan-Meier method and survival rates were compared using log-rank analyses.

Results: 56 patients were operated in the year 2010-2011 for glioblastoma multiforme. 35 patients had single resections (median age 66 years). 21 patients (Table 1) subsequently had reresections (median age 56 years). The commonest presenting symptoms in the reresection group were headaches (35%) and confusion (35%). 16 (80%) patients who underwent reresections had preoperative KPS 80-90. The average maximum tumour diameter was 4cm. The right frontotemporal lobes (50%) were the commonest tumour locations. The median interval time from 1st to 2nd resection was 13.5 months, 8.8months from 2nd to 3rd resections and 5 months from 3rd to 4th resection. There is a statistically insignificant trend for longer median survival in patients who underwent reresections versus single resection, 20 months and 7 months (p=0.08), respectively.

Conclusions: The median overall survival of a South Australian adult GBM patient was 13months. However, due to its small sample size, this study is unable to confirm statistically significant difference in the median survival between single versus surgical reresections in GBM patients.

Table 1. Resection numbers in patients with glioblastoma multiforme.

Resection number	1	2	3	4
Patient number	35(62%)	15(27%)	5 (9%)	1 (2%)



A novel brain cancer mouse model harbouring human-relevant PI3-kinase pathway mutations

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We have developed a novel mouse which harbours clinically/human-relevant inducible "PI3-kinase" mutations found in many brain tumour patients. Here we show that with this model we can induce brain tumours which resemble human brain tumours. The mouse model we have developed carries two inducible mutations: 1) an oncogenic mutation of the PI3K p110-alpha subunit (p110-H1047R) and 2) inducible loss of the PI3K pathway inhibitor / tumour suppressor, PTEN. So far we have targeted these mutations to neural stem/progenitor cells. When both mutations are activated simultaneously, mice begin to exhibit ataxia and seizures at 2 months post-induction and histological analysis shows large GFAP-positive tumours around the lateral ventricles. These tumours are reminiscent of high-grade GBM. Interestingly, mice with only the p110H1047R mutation develop less aggressive, slower growing tumours. This shows that genetically modulating the levels of the PI3K pathway leads to the development of different brain tumour types. Further findings and the implications on using this mouse as a pre-clinical model will be further discussed.

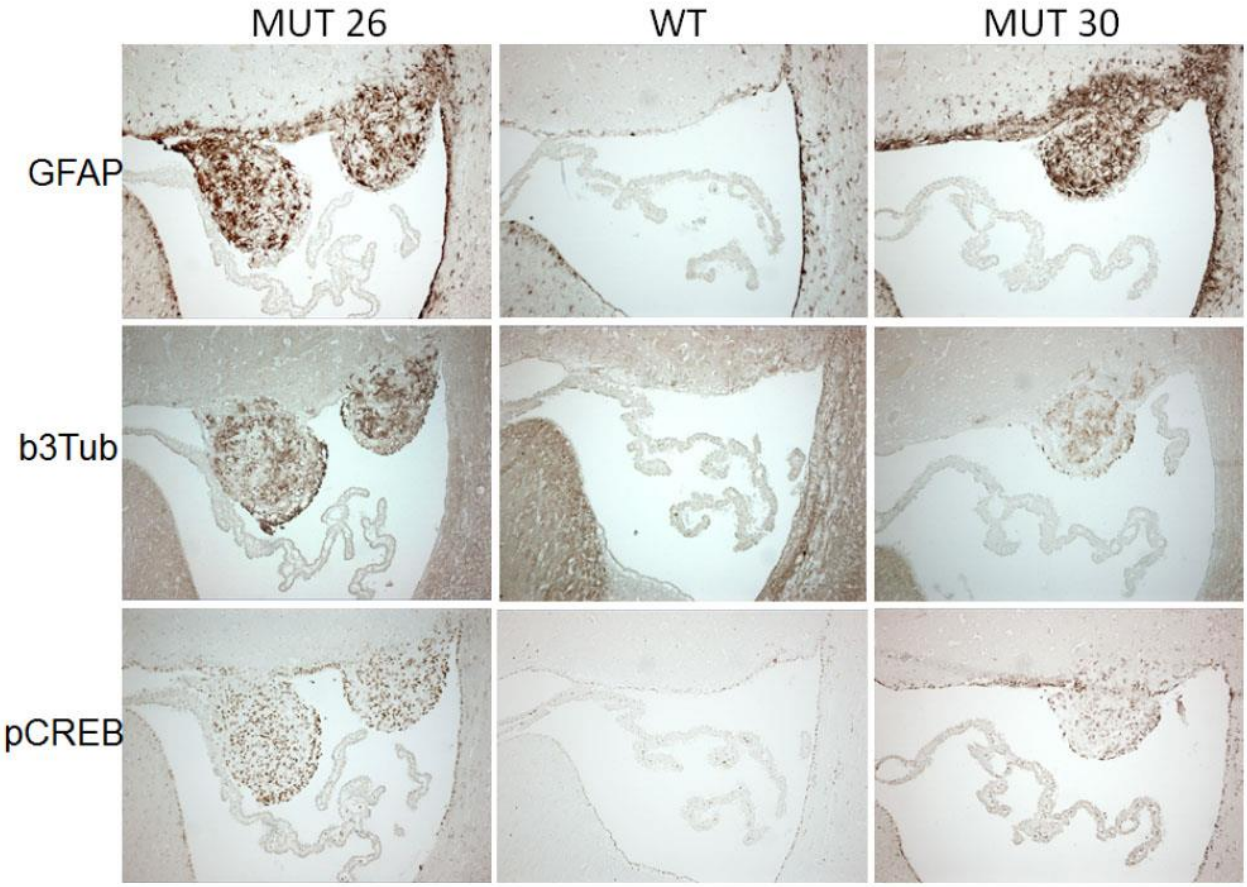

Figure 1. *Pik3ca$^{H1047R/+}$-Pten$^{lox/lox}$-Nes^{CreERT2} mouse brains show multiple tumours at only 2 months post- induction.* Tumours express glial marker GFAP, neuronal marker beta-3-tubulin; consistent with human high grade gliomas. Tumours also express pCREB, which we recently reported as a marker of glioblastoma **(Daniel et al., 2014, Oncogenesis)**.

Low dose histone deacetylase inhibitor treatment halts rhabdoid tumour growth and induces osteogenesis and neuronal differentiation

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Rhabdoid Tumour (RT) is a rare, malignant tumour of infancy arising mainly in the kidney or CNS. RTs are highly resistant to conventional treatments and outcomes remain poor despite aggressive multimodal therapy. The sole recurrent genetic abnormality in RT is homozygous deletion or inactivation of the chromatin-remodelling gene, SMARCB1, thus providing an ideal model for exploring epigenetic therapies such as the use of histone deacetylase inhibitors (HDACi).

Aims: This study investigates the effects of HDACi on cell growth and differentiation in RT cell lines and mouse xenografts and analyses resultant changes in gene expression.

Methods: In vitro based cell proliferation, cell cycle and colony forming assays were undertaken and the altered gene expression profiles upon HDACi treatment were analysed using Illumina expression beadchip arrays and quantitative real-time PCR. The effects of HDACi on RT cell differentiation, both in vitro and in a mouse xenograft tumour model, were determined and post-treatment, cells or tissue were stained with various differentiation markers.

Results: HDACi treatment inhibited RT cell growth and self-renewal in vitro and halted tumour growth in vivo. After 21 days of sustained, low dose treatment with HDACi LBH589, gene expression and qualitative differentiation marker staining of cells or tissue showed evidence of osteoblast differentiation and bone formation, as well as neural differentiation.

Conclusion: Our data suggest that low dose HDACi treatment has the potential to inhibit RT cell growth and drive differentiation. This makes differentiation therapy an exciting avenue to explore and avoids the challenges of achieving a cytotoxic response in patients. The ability of HDACi to differentiate tumour cells and reduce their ability to self-renew warrants further investigation, as it provides an appealing means of tackling the issue of tumour recurrence.

Low use of communication support tools in patients with primary or metastatic brain malignancy treated at two NSW metropolitan radiotherapy centres

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Objective: Patients with brain malignancy can experience deteriorating cognitive function with reduced capacity to process and recall information. It is unknown if health professionals (HPs) adapt communication during consultations regarding brain radiotherapy. We aimed to establish current practice, communication support tool use, and patient/caregiver perception of communication.

Methods: Study was cross-sectional in design with patients/caregivers completing 14 Likert response scales after consultations, and follow-up questionnaires 1-2 weeks later. Items included: clarity of explanation, information recall, health literacy, and surrogate questions and assessment for cognitive functions. Additional questions explored communication tool usage.

Results: Of 47 patients recruited, with median age 61 years and 42 (89%) ECOG 0-2, 36 (76%) had secondary brain metastasis and 13/44 (29%) patients had normal cognition (MoCA score ≥26/30).

At baseline, HPs reported no communication tools were used in 36/44 (81%) consultations and none were taken home. HPs reported most patients understood information with verbal explanation alone when speech, jargon, tempo were adapted.

HPs reported barriers to communication tool use in 13 consultations, including: non-availability, non-specific tools, and time constraints. Tools used included: visuals of medical equipment/processes (7), specific communication strategies (6).

Patients and caregivers reported when tools were used they helped understanding. Tools endorsed as helpful by patients; respectively caregivers included: visuals of medical equipment/processes (8;4); written information (5;8), consultation recordings (2;1). Fifteen patients and one caregiver reported no other tools were required.

At follow-up, 28/39 (71%) patients reported HP explanation as quite or extremely clear. Although patients recalled illness and treatment details, treatment-related side effects and their management were recalled by 78% and 42% respectively.

Conclusion: Few communication tools were used; when used they were helpful. Non-availability was the main barrier to use. Despite patients and HPs being content with communication, patient and caregiver recall appears impaired with likely negative impact on self-management.

Identifying Patterns of Service Use and Place of Death of Patients with a High-Grade Malignant Glioma

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Aims: The supportive and palliative care needs of patients with high-grade malignant glioma (HGG) are not well described despite patients having a high burden of symptoms, psychosocial distress and care. This study examined current patterns of hospital utilisation to provide a basis for planning appropriate care.

Methods: The Victorian Admitted Episodes Dataset, Emergency Minimum Dataset and Death Index are linked allowing patient care to be tracked over time and across sites of care. Patients admitted to hospital between January 2003 and December 2008 with an incident diagnosis of PMG

were included. The outcomes of interest were total hospital bed-days in the last 30 days of life and place of death. Variables were analysed using descriptive statistics and multivariate regression analysis.

Results: 1,997 patients were identified, of whom 1160 died during the follow-up period. Over the course of their illness, they had a mean of 4.9 hospital admissions, 2.6 emergency presentations and a mean stay of 49 bed-days. The analysis was restricted to those who died with more than 4 months of follow-up (n=678, 34%). Supportive and palliative care utilisation steadily increased, particularly at 4-6 months prior to death, with 75.5% (n=512) receiving some palliative care by their death. Just one quarter (26.0%) of patients died outside of hospital. Palliative care in the 90 days preceding the last month of life was significantly associated with low hospital utilisation (RR 0.62, 95% CI 0.46,0.82), and an increased likelihood of dying outside of hospital (RRR:2.16, 95% CI 1.16-4.04, p 0.05).

Conclusion: Palliative care referral earlier in the illness trajectory enabled patients to be cared for and die at home. The ability to link non-identifiable patient data across hospitals in Victoria gives valuable insight into service use of patients with PMG to enable supportive and palliative care systems to develop appropriate responses.

A novel way to remove pineal tumours using Axiem electro-magnetic navigation via endoscopy and the Nico Myriad side cutting aspiration device

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Background: Pineal lesions are rare and represent 0.5-1% of all intracranial neoplasms. The traditional approach is via a supracerebellar infratentorial path or a trantentorial path both of which require significant logistical efforts with positioning, surgical time and post-operative care. Here, we present a novel approach of resecting a pineal tumour though a minimally invasive technique where the outcome is comparable to the traditional way of resecting this type of tumour.

Method: Using the Medtronic AxiEM stealth system, a forehead incision would be made and a straight pathway into the frontal horn of the lateral ventricle using the Aesculap endoscope. This would then be passed through the foramen on Monroe and into the third ventricle to visualise the pineal tumour. The Nico Myriad was subsequently utilised to resect the tumour.

Results: No significant complications were seen with this method and the results of this procedure were comparable to the more traditional methods of pineal tumour resection. This was confirmed via a post-operative MRI.

Conclusion: With the advancement of technology, stealth guided minimal access surgery is now possible for removal of intraventricular and pineal tumours. This method has been shown to produce comparable outcomes with a lower risk profile when compared with traditional approaches.

Notes

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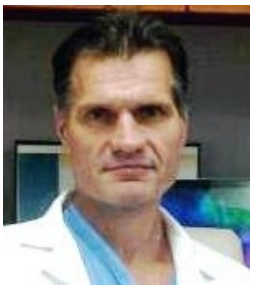

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