2016 Lifespan Research Day
21 July 2016
2016 Lifespan Research Day

Organising committee

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Children's Cancer Research Unit, The Children's Hospital at Westmead

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Central Clinical School and Charles Perkins Centre, The University of Sydney

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The George Institute for Global Health

Dr Kevin Yin  
National Centre for Immunisation Research and Surveillance, The Children's Hospital at Westmead and Sydney School of Public Health, The University of Sydney
Welcome message from the Lifespan Research Network co-leaders

On behalf of the Lifespan Research Network, we welcome you to the 2016 Lifespan Research Day. We especially welcome our keynote speakers Professor Joerg Eberhard, Dr Leanne Hassett and Professor David Hunter and our invited speaker Dr Francisco Schneuer, winner of the inaugural Lifespan Collaboration Award.

Lifespan Research Day is an opportunity to learn about recent research achievements and current projects that showcase the lifespan research approach. We will hear from researchers who are utilising novel methods and harnessing new and improved technologies. Our speakers will discuss research findings that not only contribute to discipline knowledge but have the potential for important health impact when translated.

Fostering collaboration and prompting new ideas are key goals of our Network. This year we have included a facilitated ‘Speed Networking’ session, providing real opportunities for researchers to meet others with similar professional interests. We will also present the 2016 Lifespan Collaboration Award. Our finalists are on our program today as oral presentations of original work. The Lifespan Collaboration Award recognises outstanding multidisciplinary, cross-faculty and/or international collaboration in lifespan research.

We hope that you enjoy the meeting, gain some new collaborators and leave with some bright ideas for future partnerships.

Professor Richard Lindley and Professor Kate Steinbeck
Co-leaders, Lifespan Research Network

About the Lifespan Research Network

The Lifespan Research Network encompasses a diverse community of researchers devoted to the study of human health and development across the stages of life. Established in 2014, the Network has an active membership from the medical and health faculties and schools as well as teaching hospitals and affiliated Centres and Institutes. It supports the four designated Strategic Priorities Area for Research Collaboration (SPARC) of The University of Sydney.

The Lifespan Research Network advocates for a ‘whole-of-life’ approach to health research. This research may have a ‘life stage relevant’ focus, explore trajectory effects from one developmental life stage to the next and/or investigate impacts on personal health and healthspan in a wider context of lifestyle, society and environment. The Network also advocates for investment in projects that address knowledge gaps in under-researched life stages and disadvantaged populations.

Throughout the year, the Lifespan Research Network hosts events and activities that champion and facilitate collaborative, cross-faculty and multidisciplinary research. Membership is open to all clinical and health services and basic science researchers from The University of Sydney and affiliated Centres and Institutes.

For further information, please contact the Lifespan Research Network Office

Phone: +61 2 9351 1915
Email: sms.lifespan@sydney.edu.au
sydney.edu.au/lifespan-research-network
# Program
## 2016 Lifespan Research Day

**Thursday 21 July 2016**  
**New Law School Lecture Theatre 106**

<table>
<thead>
<tr>
<th>Time</th>
<th>Session</th>
<th>Title</th>
</tr>
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<tbody>
<tr>
<td>8.30am</td>
<td>Registration</td>
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<tr>
<td>9.00am</td>
<td>Welcome</td>
<td>Prof Kate Steinbeck, Lifespan Research Network co-leader</td>
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<tr>
<td>9.10am</td>
<td>Session 1</td>
<td>Lifespan and Healthspan</td>
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<tr>
<td>9.15am</td>
<td>Chair – Dr Yuyan Chen</td>
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<tr>
<td>9.15am</td>
<td>K01 Prof Joerg Eberhard, Charles Perkins Centre, The University of Sydney</td>
<td>Lifespan oral health</td>
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<td>9.50am</td>
<td>P01 Ms Hasthi Dissanayake, Boden Institute of Obesity, Nutrition, Exercise and Eating Disorders, The University of Sydney</td>
<td>Newborn body composition and markers of autonomic function: a potential tool for identification of newborns at risk of later cardiovascular disease</td>
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<td>10.05am</td>
<td>P02 Assoc Prof Devanshi Seth, Centenary Institute of Cancer Medicine &amp; Cell Biology; Drug Health Services, Royal Prince Alfred Hospital; Central Clinical School, The University of Sydney</td>
<td>Genetic risk factors for alcoholic cirrhosis: genome-wide case control study</td>
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<td>10.20am</td>
<td>P03 Dr Alexandre Stephens Clinical and Population Perinatal Health Research, Kolling Institute; Sydney Medical School Northern, The University of Sydney; Public Health Observatory, Sydney Local Health District</td>
<td>Association of gestational age and severe neonatal morbidity with mortality in early childhood</td>
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<td>10.40am</td>
<td>Morning tea</td>
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<td>11.10am</td>
<td>Session 2</td>
<td>Methods, New and Improved</td>
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<td>11.10am</td>
<td>Chair – Dr Adrienne Gordon</td>
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<tr>
<td>11.15am</td>
<td>K02 Dr Leanne Hassett, The George Institute for Global Health and The University of Sydney</td>
<td>Prescribing technology in rehabilitation: lessons learnt from the ongoing Activity and MObility UsiNg Technology (AMOUNT) rehabilitation trial</td>
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<td>11.50am</td>
<td>P04 Prof Cathie Sherrington, The George Institute for Global Health, The University of Sydney</td>
<td>AMOUNT rehabilitation trial: support and health coaching during the community program for the first 80 participants</td>
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<td>Dr Harriet Gunn, Academic Department of Adolescent Medicine, The Children’s Hospital at Westmead and The University of Sydney</td>
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<td>TransitionMate: A mobile phone application to support self-management and transition in young people with chronic illness</td>
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<td>Dr Francisco Schneuer, Menzies Centre for Health Policy, Sydney School of Public Health, The University of Sydney</td>
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<td>Prenatal origins and health outcomes of male reproductive congenital anomalies diagnosed at birth and testicular cancer in adulthood</td>
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<td>12.35pm</td>
<td>Lunch</td>
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<td>1.30pm</td>
<td>Session 3</td>
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<td>1.35pm</td>
<td>Chair</td>
<td>Dr Alison Hey-Cunningham</td>
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<td>K03</td>
<td>Prof David Hunter, The Institute of Bone and Joint Research, The University of Sydney</td>
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<td>Research solution to chronic disease burden in the community: osteoarthritis</td>
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<td>Ms An Truong, Regenerative Neuroscience Group, Brain and Mind Centre, The University of Sydney</td>
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<td>Migratory characteristics of skin-derived neural precursors: a novel stem cell therapy for Alzheimer’s disease</td>
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<td>2.25pm</td>
<td>P07</td>
<td>Ms Bridget Foley, Primary Health Care Education and Research Unit (PERU), Research and Education Network, Western Sydney Local Health District and Sydney School of Public Health, The University of Sydney</td>
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<td>Can student-led empowerment influence lifestyle behaviours in adolescents?</td>
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<td>2.40pm</td>
<td>P08</td>
<td>Dr Wendy Gold, NSW Centre for Rett Syndrome Research, Western Sydney Genetics Program, The Children’s Hospital at Westmead and Discipline of Paediatrics and Child Health, The University of Sydney</td>
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<td>HDAC6 inhibition improves the impaired phenotype in a Rett syndrome mouse model</td>
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<td>3.00pm</td>
<td>Afternoon tea</td>
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<td>4.45pm</td>
<td>Session 4</td>
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<td>4.45pm</td>
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<td>Presentation of 2016 Lifespan Collaboration Award</td>
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<td>Closing remarks, Prof Richard Lindley Lifespan Research Network co-leader</td>
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<td>5.00pm</td>
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Invited speakers

Professor Joerg Eberhard

Professor Joerg Eberhard is a dental-scientist translational researcher and the Chair of Lifespan Oral Health for the Faculty of Dentistry and the Charles Perkins Centre. After graduating from the University of Duesseldorf, Joerg went on to specialise in Periodontist, completed a postdoctoral fellowship and was awarded the Venia Legendi in 2003 at the University of Kiel. Prior to joining the Faculty of Dentistry in April 2016, Joerg was a professor and clinical-scientist at Hannover Medical School.

Professor Eberhard’s research is focused on the interactions between oral and general health through all phases of life. He is especially interested in translating the current body of knowledge in oral health to healthcare policies aimed to improve health of the whole population. This approach includes exploring the mechanistic pathways of how bacteria and inflammatory products penetrate the epithelial barrier in the oral cavity to learn more about their dissemination and adverse action to the vascular system. He was awarded the Hans-R. Mühlemann Research Prize, Swiss Society for Periodontology in 2001. In 2004, 2009 and 2011 he awarded the meridol-Award of the German Society for Periodontology. Together with a team of dental students he received the Glaxo Smith Kline Innovations Award in 2004, Dental Education Award of the Kurt-Kaltenbach Foundation, granted by the German Society for Dental Research and in the same year the DKV-Cochrane Award granted by the German Cochrane Centre.

Dr Leanne Hassett

Dr Leanne Hassett is a Research Fellow at The George Institute for Global Health and a Lecturer in the Discipline of Physiotherapy at the University of Sydney. Leanne worked for 17 years as a clinical physiotherapist, primarily with adults after traumatic brain injury. Leanne’s current research is focused on reducing the global epidemic of physical inactivity, focusing on people with physical disabilities from neurological conditions. Her research addresses this problem through evaluating innovative delivery of healthcare, evaluating new technologies and exercise interventions, and identifying, promoting and evaluating appropriate opportunities for physical activity including disability sports. Her current research includes being a lead investigator and manager on the NHMRC funded AMOUNT rehabilitation trial, the largest trial internationally investigating the use of tailored prescription of affordable technology to increase physical activity and mobility in people receiving rehabilitation.
**Professor David Hunter**

Professor David Hunter is a rheumatology clinician researcher whose main research focus is clinical and translational research in osteoarthritis (OA). He is the Florance and Cope Chair of Rheumatology and Professor of Medicine at the University of Sydney, Chair of the Institute of Bone and Joint Research and Staff Specialist at Royal North Shore Hospital and North Sydney Orthopaedic and Sports Medicine Centre. In 2014 he was ranked as the leading expert in the world on osteoarthritis on expertscape.com. A native of New Zealand and an Australian citizen, he earned his Bachelor of Medicine, Bachelor of Surgery, and Master of Sports Medicine at the University of New South Wales. He completed a fellowship in Rheumatology at the Royal Australian College of Physicians, a Masters of Sports Medicine from UNSW, and earned a Masters of Medical Science (Clinical Epidemiology) from the University of Newcastle. He established a full-time career in medical research in 1999 and received his PhD in 2001. His research is focused on a number of key elements in OA including (but not limited to) the epidemiology of osteoarthritis, the application of imaging to better understand structure and function with application to both epidemiologic research and clinical trials, novel therapies in disease management and health service system delivery of chronic disease management. He is an editor for leading international journals in his field, has authored books on osteoarthritis and has over 350 publications in peer reviewed journals.

**Dr Francisco Schneuer**

Dr Francisco Schneuer is a Post-Doctoral Research Fellow from the Menzies Centre for Health Policy, School of Public Health. His research is focused in paediatric and perinatal epidemiology utilising administrative record-linked health data. Francisco is the recipient of the SMS 2015 Lifespan Collaboration Award together with collaborators from Children’s Hospital Westmead, the University of Western Australia and Curtin University.
Abstracts

**K01 Lifespan oral health**

**Joerg Eberhard**

Charles Perkins Centre, The University of Sydney

Oral health affects health during every phase of life. These interactions begin in the unborn, continue during childhood, adulthood and also affect the elderly. These interactions could be life-threatening or could simply add individual risk within an array of adverse lifestyle factors. Of course, individuals may also benefit from good oral health. Oral health is an example of the complexity of chronic diseases that needs further understanding provided by multi-disciplinary research collaborations. This presentation will highlight a few of the lifespan oral health interactions that may stimulate the Lifespan Research Network.

**P01 Newborn body composition and markers of autonomic function: a potential tool for identification of newborns at risk of later cardiovascular disease**

**Hasthi Dissanayake**¹,², Rowena McMullan²,³, Melinda Phang²,³, Kirsty Mckenzie²,³, Yang Kong²,³, Adrienne Gordon¹,³, Jon Hyett²,³, Camille Raynes-Greenow³, David Celermajer²,³, Jaimie Polson², Michael Skilton¹,²

¹Boden Institute of Obesity, Nutrition, Exercise and Eating Disorders, The University of Sydney
²Sydney Medical School, The University of Sydney
³Royal Prince Alfred Hospital

Background: Birth weight is associated with adult cardiovascular disease, such that those at both ends of the spectrum are at increased risk. However, body composition may be a better risk marker. Accordingly, we sought to determine whether body fat percentage is associated with alterations in autonomic function in newborns, a mechanistic contributor to hypertension, and whether this is independent of birth weight.

Method: Body fat percentage was assessed by air displacement plethysmography (PEAPOD) in term babies from RPA Hospital following birth. Blood pressure waveform (Finapres) and 3-lead ECG were recorded for 15 minutes while at rest, within 2 weeks of birth. Heart rate variability and systolic blood pressure variability, and their specific frequency components (very low, low, and high frequencies), were assessed as markers of autonomic function.

Results: Newborns with high (>90th gender and gestation-specific percentile) or low (≤10th percentile) body fat percentage had lower overall heart rate variability than those with average body fat percentage (25-75th percentile; control). Similar findings were observed for individual frequency components (all P<0.05), except for high frequency which did not differ in those with ≤10th percentile (P=0.62). There was also a non-linear association of birth weight with heart rate variability (P=0.002), although the association of high and low body fat percentage with overall heart rate variability was independent of birth weight (P=0.002).

No significant differences were found in overall blood pressure variability between groups, nor for the individual frequency components (all P ≥ 0.20).

Conclusions: Newborns with either low or high body fat percentage had altered markers of autonomic function, consistent with decreased autonomic modulation of the heart, but not the blood vessels, suggesting that body fat percentage at birth may be an independent marker of cardiovascular risk. Early risk identification may allow opportunities for early intervention strategies to retard cardiovascular disease progression into adulthood.
P02 Genetic risk factors for alcoholic cirrhosis: genome-wide case control study

John B Whitfield2, Paul S Haber1, Christopher P Day3, Steven Masson3, Ann K Daly3, Heather J Cordell1, Sebastian Mueller5, Helmut K Seitz6, Suthat Liangpunsakul6, Chi Westerhold4, Tiebing Liang6, Lawrence Lumeng8, Tatiana Foroud4, Bertrand Nalpas2, Philippe Mathurin9, Felix Stickel9, Michael Soyka10, Gregory J. Botwin11, Timothy R. Morgan11 and Devanshi Seth1 for the GenomALC Consortium

1 Centenary Institute of Cancer Medicine & Cell Biology; Drug Health Services, Royal Prince Alfred Hospital; Central Clinical School, The University of Sydney
2 Department of Genetic Epidemiology, QIMR Berghofer Medical Research Institute
3 Institute of Cellular Medicine, Newcastle University Medical School, UK
4 Institute of Genetic Medicine, International Centre for Life, Newcastle University, UK
5 Department of Internal Medicine, Salem Medical Center and Center for Alcohol Research, University of Heidelberg
6 Division of Gastroenterology and Hepatology, Department of Medicine, Indiana University
7 Liver Unit & Inserm U1016 Hospital Cochin, France
8 Le CHRU de Lille, Hôpital Claude Huriez, Lille Cedex, France
9 Department of Gastroenterology and Hepatology, University Hospital of Zurich
10 Privatklinik Meiringen (MS), Meiringen, Switzerland
11 Department of Veterans Affairs (VA) Long Beach Healthcare System, California

Background: Alcoholic Liver cirrhosis (ALC) is the major medical consequence of excessive drinking with by far the highest mortality and is the 5th leading cause of death in middle aged men. Not everyone who drinks at risky levels gets cirrhosis suggesting that apart from alcohol several risk factors (genetic and environmental) are also essential for its development. But thus far, there is no way to predict who will develop cirrhosis and a complete understanding of what these risk factors are is unknown.

Aim: The overall aim of this project is to identify genetic risk factors that predispose some drinkers to and protect others from developing cirrhosis.

Methods: Our GenomALC Consortium, funded by the National Institutes of Health (NIH), USA, is collecting worldwide (Australia, Belgium, France, Germany, Switzerland, United Kingdom, and United States) thousands of drinkers with alcoholic cirrhosis (cases) and those drinking comparable amounts over similar time, but free of significant liver disease (controls). Extensive phenotypic data are obtained using semi-structured interviews and patient records. Blood samples for DNA are collected to screen millions of gene mutations in a genome-wide association study. Genotyping will be performed once enrolment is complete (n=5000).

Results: We have successfully recruited 92% (4617/5000) of participants, assigned 2990 cases and 1213 controls so far. Description of a sub-cohort was published (Whitfield et al., Alc Clin Exp Res 2015). Both cases and controls had a high prevalence of reported parental alcohol problems, but cases were significantly more likely to report that a father with alcohol problems had died from liver diseases.

Summary: We show for the first time a paternal link to this disease underscoring a genetic component to the risk and the heritability of this disease. ALC is a preventable disease if we can identify “at risk” individuals early to provide appropriate counselling and therapeutic options.
P03 Association of gestational age and severe neonatal morbidity with mortality in early childhood

Alexandre S Stephens1,2,3, Samantha J Lain1,4, Christine L Roberts1,2, Jennifer R Bowen2,5 and Natasha Nassar1,2,4

1Clinical and Population Perinatal Health Research, Kolling Institute
2Sydney Medical School Northern, University of Sydney
3Public Health Observatory, Sydney Local Health District
4Menzies Centre for Health Policy, School of Public Health, Sydney Medical School, The University of Sydney
5Department of Neonatology, Royal North Shore Hospital

Background: Although infant and child mortality rates have decreased substantially worldwide over the past two decades, efforts continue in many nations to further these declines. The identification of pertinent perinatal factors that are predictive of early childhood mortality would help with these efforts. This study investigated the association of two crucial perinatal factors, gestational age and severe neonatal morbidity at birth, with mortality during infancy (ages 29-364 days) and early childhood (ages 1-5 years).

Methods: The study population included all singleton live births, ≥ 32 weeks gestation in New South Wales, Australia in 2001-2011. Birth data were probabilistically linked to hospitalization morbidity data and deaths data (linked birth cohort N=889,160), and multivariable Cox regression models were used to assess the direct and indirect effects of gestational age on mortality with severe neonatal morbidity as a mediator.

Results: The average follow-up time per child was 4.14 years (3,687,908 total person-years), with 1,191 deaths observed. Gestational age was associated with increased mortality, and specifically from deaths attributable to infections, respiratory conditions and injuries during infancy, but not during early childhood. Severe neonatal morbidity strongly mediated the effects of gestational age during infancy, and less so during early childhood, and was associated with increased mortality from circulatory, nervous and respiratory system causes.

Conclusions: The direct effects of gestational age on mortality extended up to one year of age, whereas severe neonatal morbidity remained associated with heightened mortality into early childhood. Efforts to maximize the health and wellbeing of vulnerable infants, with emphasis on preventing infections and injuries, may help further reduce early childhood mortality.

K02 Prescribing technology in rehabilitation: lessons learnt from the ongoing Activity and MObility UsiNg Technology (AMOUNT) rehabilitation trial

Leanne Hassett

The George Institute for Global Health and The University of Sydney

People with mobility limitations can benefit from rehabilitation programs that provide a high dose of repetitive exercise. However, since providing a high dose of exercise is logistically difficult and resource intensive, people in rehabilitation spend the majority of the day inactive. New technologies potentially provide an affordable way to increase the dose of exercise and overall physical activity for people in rehabilitation.

The Activity and MObility UsiNg Technology (AMOUNT) rehabilitation trial is a currently recruiting NHMRC-funded pragmatic randomised controlled trial with an embedded economic analysis and qualitative study. The primary aim is to evaluate the effect of the addition of affordable technology-based exercise to usual care on physical activity and mobility in people with mobility limitations admitted to inpatient aged and neurological rehabilitation wards compared to usual care alone.
The experimental intervention is delivered by Physiotherapists for 6-months after randomisation and incorporates inpatient rehabilitation and post-discharge settings. The intervention is prescribed according to a protocol which matches technologies and games/exercises to mobility limitations as well as considering participant goals, technology experience and preferences. Technologies include commercially available devices and android/iOS applications (Nintendo Wii; Xbox Kinect; Fitbit; Runkeeper app), rehabilitation-specific devices (Humac; Fysiogaming), and technologies developed specifically for the trial (Stepping Tiles; exercise iPad apps; physical activity iPhone app).

We have currently recruited 260 out of the planned 300 participants and expect to complete the study in early 2017. This presentation will describe the protocol of the AMOUNT rehabilitation trial and discuss experiences of implementing technology into inpatient and post-discharge rehabilitation settings from a research and clinical perspective.

**P04 AMOUNT rehabilitation trial: support and health coaching**

_Cathie Sherrington_1, Mayken van den Berg_2, Leanne Hassett_1,3, Ashley Rabie_4, Sakina Chagpar_1,
Heather Weber_2, Siobhan Wong_1, Karl Schurr_5, Annie McCluskey_3, Richard Lindley_1, Maria Crotty_2

1 The George Institute for Global Health, The University of Sydney
2 Flinders University
3 Faculty of Health Sciences, The University of Sydney
4 Liverpool Brain Injury Rehabilitation Unit
5 Bankstown-Lidcombe Hospital

Background and aims: Technologies to enable ongoing exercise are likely to become increasingly important in the future as the proportion of older people in the population increases and resources to provide rehabilitation care become more limited. We aimed to evaluate how much, the type and mode of support provided to rehabilitation participants using technology in the community after discharge from inpatient rehabilitation as part of the NHMRC funded AMOUNT rehabilitation trial.

Methods and design: Process evaluation of the post-hospital intervention data from a currently recruiting randomised controlled trial. Participants: Eighty participants (53% female; mean age 72±17.7; 49% neurological condition). Intervention: Additional to standard care, prescribed according to a protocol which matches games/exercises from eight rehabilitation-specific, as well as commercially available technologies, to the participant’s current mobility limitations. Outcomes: Audit of research physiotherapist intervention notes for the first 80 participants, recording frequency, duration, mode, reason for contact and topics covered.

Results: Participants received community intervention for an average of 159 (SD20.6) days. Participants and physiotherapists had on average 15 (SD5.5) contact moments (approximately every 12 days), of which 8 were phone calls (15min duration), 6 home visits (46min duration) and 1 other (20min duration). Reasons for contact were health coaching (58%) ‘quick’ contact to check up on participants (19%), data collection (10%), technology support (8%) and other (5%). Most important topics covered during health coaching sessions were objective data gathered from prescribed technologies (45%), mobility status (36%), physical activity status (41%) and adherence (40%). Technical assistance (20%), goal setting (26%) and modification of the exercise program (21%) were topics less frequently addressed.

Conclusions: Tailored physiotherapy support enables ongoing technology-based rehabilitation after discharge from hospital. The preliminary results suggest that using a tailored health coaching model to support technology use and adherence is feasible. Health coaching sessions can be provided remotely, limiting the need for frequent home visits.
P05 TransitionMate: A mobile phone application to support self-management and transition in young people with chronic illness

Harriet M Gunn1,2, Yu Zhao3, Abelardo Pardo3, Rafael A Calvo1, Katharine S Steinbeck1,2

1 Academic Department of Adolescent Medicine, The Children's Hospital at Westmead
2 Discipline of Child and Adolescent Health, The University of Sydney
3 School of Electrical and Information Engineering, The University of Sydney

Introduction: Transition from paediatric to adult healthcare requires significant adjustment and increased patient autonomy; however, commonly illness control deteriorates. The ubiquitous nature of mobile-phones facilitates novel communication channels to support chronic illness self-management and improve illness outcomes.

Aims: Develop and evaluate a smart-phone application (TransitionMate) to support self-management and transition in young people with chronic illness.

Methods: Participants were 18-25 years with diabetes, cystic fibrosis or inflammatory bowel disease who had transitioned to adult care. Participants used TransitionMate for four weeks and completed baseline and post-test questionnaires regarding application functionality. A digital footprint of participants' interactions with TransitionMate was obtained via a dedicated server. Personalised functions included reminders, mood/health-tracker, memos and contact information.

Results: To date, 16 participants have trialled TransitionMate (12 female), mean age 21 years (range 18-25). As part of their routine chronic illness management, participants performed an average of 9 daily health related tasks for example taking medications or performing a procedure. Participants requested a median 4 (range 2-6) daily personalised reminders and responded to 61% reminders (range 27-100%). Participants interacted with TransitionMate a median 7 times a day (range 2-23). Reminders were the most frequently used function (22 times weekly), the “favourite” function (82% participants) and the “most helpful” function (100% participants). TransitionMate usefulness regarding self-management and transition support was rated as 7.4 out of 10.0. Over 90% of participants would recommend TransitionMate to peers with a chronic illness.

Conclusion: This study provides promising novel data emphasising the usefulness of TransitionMate to engage and support young people with chronic illness. Results from this study have been used to guide application modifications, to develop an improved version of TransitionMate. Future studies will evaluate this new application in a larger cohort of younger participants undergoing transition.

I01 Prenatal origins and health outcomes of male reproductive congenital anomalies diagnosed at birth and testicular cancer in adulthood

Francisco Schneuer1, Andrew Holland2, Sarra Jamieson3, Elizabeth Miline3, Gavin Pereira4, Carol Bower3, Natasha Nassar1

1 Menzies Centre for Health Policy, Sydney School of Public Health, The University of Sydney
2 The Children’s Hospital at Westmead
3 Telethon Kids Institute, The University of Western Australia
4 School of Public Health, Curtin University

Background: There is growing concern about the rising rates of male reproductive congenital anomalies (hypospadias and cryptorchidism), testicular cancer and decreased male fertility. These conditions may share a common origin in fetal life through the impaired production or disrupted release of androgens in-utero, resulting in increased risk of abnormal genital development in boys and subsequent sub-fertility; and testicular cancer in adulthood.

Aims: To investigate perinatal antecedents and health outcomes of male reproductive congenital anomalies and risk of testicular cancer and sub-fertility in adulthood.

Study design and methods: Population-based cohort study of all males live born in Western Australia between 1970 and 2013. Health information will be obtained via record-linkage of birth, congenital
anomalies, hospital, cancer, family connections and assisted reproductive technology data.

Main outcomes: Male reproductive congenital anomalies: hypospadias, cryptorchidism, testicular cancer and subsequent fertility, including male infertility, fertility management and future paternal birth. Covariates will include maternal and perinatal factors, time of corrective surgery, severity, post-surgical complications, co-morbidities that may affect subsequent male fertility such as genitourinary, gastrointestinal or renal conditions, infections, chronic diseases, cancer or requiring hospital admission in childhood, adolescence and adulthood.

Multivariable analyses will be applied to assess the association between cryptorchidism, hypospadias with testicular cancer and fertility whilst taking into account family-level and individual-level factors for each outcome, potential confounding and likely causal pathways.

Significance: This study will facilitate simultaneous analysis of the links between hypospadias, cryptorchidism, sub-fertility and testicular cancer investigating potential common aetiologies. It will also increase our understanding of the long-term health and fertility outcomes of males affected by reproductive anomalies and contribute to practice guidelines and policy to improve reproductive health of future generations.

K03 Research solution to chronic disease burden in the community: osteoarthritis

David Hunter

The Institute of Bone and Joint Research, The University of Sydney

Osteoarthritis (OA) is at the forefront of an exploding epidemic of non-communicable chronic diseases. It affects over 3 million Australians and is a leading cause of disability and health service utilisation. Beyond the pharmacological and surgical advances, there are population-based interventions that have proven effective for preventing the development of OA. These measures include lifestyle management, maintaining a healthy weight, remaining active, and avoiding joint injury.

For the person with osteoarthritis, management is typically inappropriate, which has enormous downstream activity consequences for health services and contributes to poor health outcomes. Modern health care systems are typically reactive and focused upon acute care whereas the management of OA is ideally efficient, coordinated and patient centred to support integration of evidence into practice. Inflation in the cost of health care is driving changes to health systems that will not only enhance the organizational costs of OA delivery but also health outcomes.

This presentation will focus on research and implementation activity targeting both disease prevention and evidence-based health service delivery.

P06 Migratory characteristics of skin-derived neural precursors: a novel stem cell therapy for Alzheimer’s disease

An Truong1, Aileen Lowe2, Thomas Duncan1 and Michael Valenzuela1

1 Regenerative Neuroscience Group, Brain and Mind Centre, The University of Sydney

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Background: Skin-derived neural precursor (SKN) is a novel source of autologous stem cells with the potential to overcome issues impeding current cell replacement therapies for neurodegenerative diseases. While their capability for proliferation and neuronal differentiation in vitro has been well-established, and therapeutic potential to restore neuronal connectivity demonstrated in aged rodents, the migratory characteristics of SKNs have yet to be explored.

Aim: To map SKN migration in the aged rodent brain and identify molecular cues for SKN mobilization.

Methods: SKNs were isolated and expanded in vitro...
from post-mortem adult canine and human skin biopsies. Chemotaxis assays were performed using the IBDI microfluidic device to identify molecular cues of SKN migration. To study migration in vivo, fluorescently-labelled SKNs were transplanted into the rodent hippocampi. Histological analysis at days 3, 7, 10 and 140 post-transplantation (n = 3 per time point) were performed to characterise SKN displacement, phenotype and integration over time. Results: In vitro, canine SKNs exhibited random movements in the absence of chemoattractants, as indicated by p > 0.05 in the Rayleigh test for chemotactic potential and lack of moving direction. Directed migration was induced by concentration gradients of growth factors BDNF, IGF-1 and VEGF (p < 0.05). Transplanted canine SKNs migrated extensively over large distances by day 7 post-transplantation, with a predilection to the CA1 hippocampal subregion.

Conclusion: In vitro results demonstrate a strong migratory potential of SKNs, and is supported by in vivo findings that show transplanted cells migrate extensively, differentiating into mature neurons and populating all hippocampal subregions. Despite their non-neural origin, SKNs respond to chemotactic factors known to be important for migration of endogenous hippocampal stem cells, suggesting similar migratory mechanisms to hippocampal neurogenesis. Ongoing studies will identify other regulators of SKN migration and further characterize the migration of human SKNs in the aged rat hippocampus.

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**P07 Can student-led empowerment influence lifestyle behaviours in adolescents?**

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Smita Shah1,2,3 and the SALSA Committee

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Background: The social environment and peer influence is recognised as a strong determinant of behaviour as adolescents transition to independence. SALSA (Students As LifeStyle Activists) is a unique peer-led school-based education program that empowers high school students (13–16 year olds) in a supportive school environment to lead a healthy lifestyle.

Aim: To assess the impact of the SALSA program on modifiable adolescent lifestyle behaviours and intentions to change behaviours.

Methods: The SALSA program was implemented using a proven cascading model. In 2014/15, 22 high schools in western Sydney, participated in the pre–post- online questionnaire measuring students health behaviours and intentions. Changes in students’ reported behaviours and intentions were assessed using cluster (school) adjusted analyses. Results: The online questionnaire was completed pre- and post- SALSA by 415 Year 10 SALSA Peer Leaders (77%) and 2,056 Year 8 students (81%). SALSA Peer Leaders reported improvements in fruit intake (54% to 63% 2 serves/day, P<0.01) and intentions to increase intake (73% to 82%, P=0.01); vegetable intake (8% to 12% >5 serves/day, P<0.01) and intentions to increase intake (17% to 30%, P<0.001); intentions to consume breakfast daily (67% to 71%, P<0.01) and reductions in sugar-sweetened beverage (SSB) drinking (44% to 38% <1cup /day, P<0.01). Year 8 students also reported improvements in fruit intake (52% to 57% 2 serves/day, P<0.001) and intentions to increase intake (66% to 72%, P<0.001); intention to increase vegetable intake (19% to 22%, P<0.01); reductions in SSB drinking (53% to 48% <1cup /day, P<0.001) and intentions to reduce recreational screen-time(ST) (37% to 42% <2 hours/day, P<0.001). No improvements were seen in students’ ST and moderate-to-vigorous physical activity behaviours.

Conclusion: A student-led empowerment education approach can impact on adolescents’ lifestyle behaviours and intentions. The significant changes and high participation rates indicate the value and potential for scaling up the SALSA program.
P08 HDAC6 inhibition improves the impaired phenotype in a Rett syndrome mouse model

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Rational: Rett syndrome (RTT) is a severe paediatric neurodevelopmental disorder, predominantly caused by mutations in the Methyl-CpG-binding protein 2 (MECP2) gene. Despite the genetic cause, the pathophysiology of the neurological phenotype is still largely unknown. The microtubule network is critical to neuronal function through its role in trafficking cargo including Brain Derived Neurotrophic Factor-containing vesicles and mitochondria. The acetylation status of α-tubulin is critical to the stability of the microtubule network. Recent studies have revealed reduced α-tubulin acetylation, increased histone deacetylase 6 (HDAC6), an enzyme which removes the acetyl groups from tubulin, and concomitant microtubule instability in MeCP2-deficient cells, suggesting a potential link between these irregularities and the neurobiology in RTT.

Objective: To investigate the stability of the microtubule network and whether the inhibition of HDAC6 can restore microtubule dynamics and ameliorate the impaired motor and behavioural phenotype of the Mecp2T158A mouse model.

Methods and results: Acetylated tubulin and HDAC6 levels were measured in MeCP2-deficient cells. Microtubule stability was measured in RTT patient fibroblasts and the trafficking speed of mitochondria was measured in Mecp2T158A cultured cortical neurons. Further, we tested a highly specific HDAC6 inhibitor to determine whether HDAC6 inhibition can ameliorate the impaired motor and behavioural phenotype of the Mecp2T158A mice. Reduced acetylated tubulin and increased HDAC6 expression were observed in both patient cells and Mecp2T158A mouse cortical neurons. In addition, we found a reduction in mitochondrial velocity and increased microtubule instability in these cells. We report that HDAC6 inhibition restores tubulin acetylation levels and improves microtubule stability. Preliminary studies in our HDAC6 inhibitor drug trial reveal that treated Mecp2T158A mice show improvement in their impaired motor, behavioural and respiratory phenotype.

Conclusion: Pharmacological HDAC6 inhibition provides a novel therapeutic option for RTT, restoring neuronal trafficking deficits, potentially stabilizing the neurological phenotype associated with the disorder.