WA COMBINED TSANZ & ANZSRS ANNUAL SCIENTIFIC MEETING

27-28 July 2018

AT THE

Australian Institute of Management
WESTERN AUSTRALIA

SPONSORED BY

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THIS MEETING HAS BEEN ORGANISED BY THE FOLLOWING BRANCH MEMBERS:

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Dr John McLachlan  
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Dr Adelaide Withers  
Ms Kelly Martinovich  
Mr Michael Beaven  
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Dr Shannon Simpson  
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WE KINDLY THANK THE FOLLOWING ORGANISATIONS FOR THEIR SUPPORT FOR THIS MEETING:

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TSANZ WA BRANCH 2018 PROGRAM

Friday 27th July
Australian Institute of Management WA, The Leadership Center
76 Birkdale St, Floreat WA 6014

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<th>Time</th>
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<tr>
<td>8.30am</td>
<td>Registration desk opens (Coffee/Tea available)</td>
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<tr>
<td>9.00am</td>
<td>Welcome and Opening of the Meeting by TSANZ WA Branch President Ingrid Laing</td>
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| 9.15am | National Keynote Speaker  
Jane Bourke - *Into the silent zone: using precision-cut lung slices to identify improved therapeutic strategies targeting small airways in chronic lung diseases* |
| 10.15am| Emerging Fields of respiratory research  
• Dino Tan - *Mechanisms of impaired anti-bacterial Th1 responses in patients with chronic obstructive pulmonary disease*  
• Thomas Iosifidis - *Translating respiratory research: From bedside to bench and back again*  
• Andrew Lucas - *Lung regeneration models* |
| 10.55am| Morning Tea                                                                                       |
| 11.25am| Neglected research areas in respiratory disease  
• Yuben Moodley - *IPF*  
• Kylie Hill - *Sedentary behaviour in people with COPD: a new lifestyle target*  
• Shannon Simpson - *Surviving preterm birth: Is BPD headed for COPD?* |
| 12.35pm| Lunch                                                                                             |
| 1.00pm | Poster viewing – 1pm onwards                                                                     |
| 1.40pm | Use of macrolides in respiratory disease session  
• Ronan Murray - *Azithromycin mechanism of action and resistance*  
• Justin Waring - *Macrolides in bronchiectasis*  
• Grant Waterer - *Macrolides in COPD and asthma*  
• Steve Stick - *Use of macrolides in children* |
| 3.00pm | Afternoon Tea                                                                                    |
| 3.40pm | Sleep research session  
• David Hillman - *Inadequate sleep in Australia: the silent epidemic*  
• Bhajan Singh - *Obstructive sleep apnoea in Western Australian*  
• Peter Eastwood - *3D Craniofacial Phenotyping and OSA* |
### CONFERENCE DINNER *(Dancing & Networking)*
Wembley Golf Course,  
200 The Boulevard, Wembley Downs WA 6019

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<td>5.30pm</td>
<td>Roaming entrée and refreshments</td>
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| 7:30pm - 8:20pm | **Master of Ceremonies:** Ingrid Laing  

**Guest Dinner Speaker:** Liz Balding - *From Little Things Big things Grow*

**Guest Dinner Speaker:** Sue Jenkins - *A Fresh Breath in the West*  
| 8.20pm        | Dessert station opens                                                |
| 9.30pm        | Close of the evening and announcement of door prize                  |

**DOOR PRIZE**  
MEMBERS  
$200 VALUED AT
### Saturday 28th July
Australian Institute of Management WA, The Leadership Center
76 Birkdale St, Floreat WA 6014

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<td>8.30am</td>
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| 9.00am  | **TSANZ President Elect Speaker**  
Bruce Thompson  
**Concurrent ANZSRS session**  
+ **ANZSRS AGM**  
- **Peter Franklin**  
  *Environment and the lung: is it safe to breathe?*  
- **Tim Whitmore**  
  *There’s just so much pus: A brief approach to Respiratory Infections and the Laboratory* |
| 9.30am  | **National Keynote Speaker**  
Jane Bourke  
*TSANZ - from the Laennac Society to Leaders in Lung Health* |
| 10.30am | **Morning Tea**                                                                                   |
| 11.00am | **New Investigator Session**  
- **Jesse Armitage**  
  *Mesenchymal stromal cell infusion alters the transcriptomic profile of peripheral blood mononuclear cells in patients with stable COPD*  
- **Samuel Montgomery**  
  *IL-1 is associated with structural lung disease in children with Cystic Fibrosis*  
- **Tylah Miles**  
  *Plasma cell and regulatory B cell-infiltration in the lungs of bleomycin-treated mice*  
- **Denby Evens**  
  *Airway epithelial cell responses to human rhinovirus infection in infants born preterm* |
| 12.00pm | **Prize giving, TSANZ AGM & Lunch**                                                              |
| 12.45pm | **Meeting Close**                                                                                 |
DINNER SPEAKERS

LIZ BALDING
Commenced working with CF in July 1987. Had spent 15 years rural nursing & wanted to work with family unit. Working with CF provided that opportunity. Completed my Bach Nursing at ECU & undertook Grad Dip Human Services (Counseling) which gave me the opportunity to provide counseling whilst working with the family unit. This provided a great fit with nursing. Made significant contributions to research over the years including presenting at national conferences & ongoing education to nursing staff at PMH. Established close links with CFWA & set up the HITH service providing links between the hospital & CFWA at home. Planning to continue working within the CF community now that I'm retired.

SUE JENKINS
Sue Jenkins has held clinical and academic positions in Perth since 1992. She has more than 130 peer reviewed papers and has supervised 33 research students. Sue is a core member of the COPD-X Guidelines Committee. In 2016 she received an OAM for her work in pulmonary rehabilitation. For the last 20 yrs Sue has shared the role of providing the pulmonary rehabilitation service at SCGH with Nola Cecins.

NATIONAL KEYNOTE SPEAKER

DR JANE BOURKE
Head, Respiratory Pharmacology Laboratory, Biomedicine Discovery Institute, Department of Pharmacology, Monash University

Dr Jane Bourke graduated with BSc (Hons) and PhD from the University of Melbourne and currently leads the Respiratory Pharmacology Group in the Biomedicine Discovery Institute at Monash University, where she combines her passions for both teaching and research. She has an international reputation as a basic scientist, and was the first Australian researcher to establish the novel precision-cut lung slice (PCLS) technique to assess small airway physiology and pharmacology in health and disease. With numerous local and international collaborators, she has applied this approach in preclinical studies in animal models of disease and more recently human tissue to study disease mechanisms and test novel therapeutic interventions for asthma and other chronic lung diseases.

Jane has published extensively in the disciplines of pharmacology and respiratory medicine (Thorax, ER, Nature Comm, Pharmacology and Therapeutics) and is a regular invited speaker at local and international meetings (TSANZ, Japanese Physiological Society, APSR, ATS, British Pharmacological Society). Jane holds current editorial roles (Frontiers in Physiology; Scientific Reports). Jane is a former member of the TSANZ Board (2004-10, 2012-16), where she chaired the Education and Research Subcommittee and the TSANZSRS Conference Committee. She was also Congress President of the recent Asia-Pacific Society of Respirology Congress (Sydney, 2017), attended by over 2000 delegates.
DINO TAN
Dr Dino Tan is a post-doctoral research scientist in the Stem Cell Unit, Institute for Respiratory Health and he is based at the Royal Perth Hospital Medical Research Foundation building. His current research interest is in the area of respiratory, immunology and infectious diseases. He completed his PhD in 2011 in the area of HIV immunology with a thesis that addressed the development of immune restoration disease and immune recovery in HIV patients on antiretroviral therapy. After graduating, his research focused on factors that contribute to the development and progression of chronic obstructive pulmonary disease (COPD). His research focus is on the characterization of cellular and molecular mechanisms of COPD pathogenesis while seeking new strategies through cellular and immune-based therapies to treat COPD and the associated bacterial respiratory infections.

THOMAS IOSIFIDIS
Thomas Iosifidis recently completed his PhD (Paediatrics, UWA) entitled “Defective cell migration as a mechanism of dysregulated asthmatic airway repair” with the Epithelial Research Group, Telethon Kids Institute. Thomas is currently focusing on translating his research findings into new therapeutic avenues for asthma. His research interests revolve around understanding the mechanisms involved in tissue injury, repair and remodelling in health and disease.

KYLIE HILL
Kylie Hill is a physiotherapist with 10 years clinical experience, working predominantly in the area of cardiopulmonary physiotherapy. She has a PhD and three-and-a-half years international post-doctoral experience.

Kylie is an Associate Professor at the School of Physiotherapy and Exercise Science at Curtin University where she supervises Honours, Masters and Doctoral physiotherapy students. She has published more than 100 peer-reviewed papers and has a growing interest in measuring and reducing sedentary behaviour in people with COPD.

SHANNON SIMPSON
Dr Shannon Simpson and her research team aim to understand the impact of preterm birth on lung health over the life course, conduct interventions that will improve lung health after preterm birth and to explore the mechanisms underpinning lung disease as a result of preterm birth. She has been awarded more than A$2.25 million in research funding, including 2 NHMRC project grants (CIA & CIB) and an NHMRC early career fellowship, to support this work. She has a growing international reputation in the field and has a number of high impact publications on the long term lung health in survivors of preterm birth, including in The Lancet Child and Adolescent Health earlier this year.
RONAN MURRAY

Following graduation from the University of Western Australia in 1992, Dr Murray undertook postgraduate training in Infectious Diseases and Clinical Microbiology in Australia, Ireland and the UK. He is a Fellow of the Royal College of Physicians in Ireland, the Royal Australasian College of Physicians, the Royal College of Pathologists of Australasia and the Australasian College of Tropical Medicine, and holds a Diploma in Tropical Medicine and Hygiene from the Liverpool School of Tropical Medicine. He is a Clinical Associate Professor at the University of Western Australia. He was the inaugural Head of Service for Infectious Diseases at Royal Perth Hospital and the inaugural head of the Department of Infectious Diseases at Sir Charles Gairdner Hospital, and amongst other achievements established antimicrobial stewardship and diabetic foot infection services at both of these institutions. He was awarded the E.F. Haywood Prize for the best clinical teacher at SCGH in 2010. He has published over 60 papers in the peer-reviewed literature on a wide variety of subjects, and is a regular invited speaker at local, national and international conferences.

JUSTIN WARING

Dr Waring graduated from the University of Western Australia in 1990. He completed postgraduate training in Respiratory Medicine at Royal Perth Hospital and was admitted as a Fellow of the Royal Australian College of Physicians in 1998. He did post fellowship training in London at the Homerton Hospital (Hackney) specializing in Tuberculosis (TB). In 2001 he took up the lead clinician post in the WA Tuberculosis Control Program, and in 2006 was made Medical Director. He has been the WA member on the National TB Advisory Committee (NTAC) since 2001, and was the chair from 2010 to May, 2015. In 2017 he was reappointed as deputy chair of NTAC. Dr Waring has also worked as a respiratory physician, practicing in general respiratory medicine, at Royal Perth Hospital (RPH) and in private practice since 2001. In 2014 he was appointed Head of the Respiratory Medicine Department at RPH. In 2017 he was elected Chair of the RPH Medical Advisory Committee. His research interests are in treatment of non-tuberculous mycobacterial lung infection, programmatic TB control and the immunology of latent TB infection.

GRANT WATERER

Dr Waterer is a respiratory physician at Royal Perth Hospital and is a Professor of Medicine at the University of Western Australia and Adjunct Professor of Medicine at Northwestern University, Chicago and Curtin University. His main research interests are in pulmonary infections, especially pneumonia and more recently non-tuberculous mycobacterial disease. He has over 170 peer reviewed publications and more than 60 invited international presentations. He is the section editor for infections for the ERJ and on the editorial board of 8 other journals including AJRCCM and Chest.
STEVE STICK

Dr Stick is a career clinician and clinical researcher and holds a National Health and Medical Research (NHMRC) Practitioner Fellowship. Dr Stick is a member of the Royal Australasian College of Physicians Research and Education Subcommittee, the NHMRC Scholarships Committee and the NHMRC Grant Review Panel. He is also a member of the American Thoracic Society (ATS), Pediatric Program Committee, Joint ATS/European Respiratory Society (ERS) Task Force on Infant Lung Function and USCF Foundation Data Safety Monitoring Committee.

In 2007, Dr Stick was appointed Clinical Lead of the Health Department of Western Australia, Respiratory Health Network, responsible for the translation of evidenced based models of care into state-wide health policy. Since 2005, he has been the Principal Investigator for the Australian Respiratory Early Surveillance Team for Cystic Fibrosis (AREST CF). AREST CF has developed a unique early surveillance program that has focussed attention on early manifestations of CF lung disease and has contributed to a paradigm shift from an approach based on amelioration of respiratory disease to one focussed on prevention of bronchiectasis, the major cause of morbidity/mortality in CF.

In July 2010, a collaboration between centres in Perth and Melbourne to investigate early childhood lung disease that Dr Stick lead was designated as a NHMRC Centre of Research Excellence. Dr Stick is also principal investigator for the 1st intervention trial to prevent bronchiectasis in newborns diagnosed with cystic fibrosis (COMBAT CF).

DAVID HILLMAN

David Hillman is a sleep physician at the Department of Pulmonary Physiology and Sleep Medicine at Sir Charles Gairdner Hospital in Perth, Western Australia, a director of the West Australian Sleep Disorders Research Institute and a research fellow at the Centre for Sleep Science the University of Western Australia.

He is a respiratory physiologist, anaesthetist and sleep physician. His clinical and research interests focus on the physiology of the respiratory system and upper airway and their relationship to respiratory disease, sleep disorders and anaesthesia. He has published extensively in these and related areas.

He is a fellow of the Australian and New Zealand College of Anaesthetists, the Royal College of Physicians of Edinburgh and an honorary fellow of the Royal Australasian College of Physicians. He is a Clinical Professor at the University of Western Australia, a past president of the Australasian Sleep Association and of the Society of Anesthesia and Sleep Medicine and past chair of Australia’s Sleep Health Foundation, a national charity devoted to improving sleep health.
BHAJAN SINGH

Bhajan obtained a Bachelor of Medicine & Surgery from the University of Queensland, Australia in 1986. As a junior medical officer he worked in north Queensland and in Aboriginal communities. Later he trained as a General Physician at the Greenslopes Hospital in Brisbane and as a Respiratory Physician at The Prince Charles Hospital in Brisbane. He became a Fellow of the Royal Australasian College of Physicians in 1995. He moved to Perth in 1996 where he trained as a Sleep Physician and completed a PhD (Distinction) in respiratory muscle physiology in 2003. He has been a recipient of the Australian Lung Foundation-Boehringer Ingelheim Chronic Airflow Limitation Fellowship and, on two occasions, the Thoracic Society of Australia & New Zealand (TSANZ) John Reid prize for physiological research. He has been a Respiratory Physiologist & Sleep Disorders Physician at Sir Charles Gairdner Hospital since 1997. He undertook a sabbatical at the University of Toronto in 2013 during which worked on the effect of fluid displacement during recumbency on upper airway narrowing. His current appointments are (1) Head of Department, Pulmonary Physiology & Sleep Medicine, Sir Charles Gairdner Hospital, (2) Clinical Professor, Faculty of Science, University of Western Australia, and (3) Director, West Australian Sleep Disorders Research Institute. He interests encompass respiratory physiology and sleep disorders, and include cardiopulmonary exercise testing, respiratory muscle physiology, ventilatory failure, non-invasive ventilation, COPD, upper airway physiology & sleep apnoea.

PETE EASTWOOD

Professor Peter Eastwood is the President of the Australasian Sleep Association, Inaugural Director of University of Western Australia’s Centre for Sleep Science, Director of the Western Australian Pregnancy Cohort (Raine) Study, and holds joint appointments as a NHMRC Senior Research Fellow at Sir Charles Gairdner Hospital and Professor at the University of Western Australia. His research investigates the pathophysiology of upper airway dysfunction in individuals with sleep-disordered breathing. More recently he has been working with the Raine Study to better understand the prevalence and risk factors for sleep disorders in young and middle-aged adults.

PETE FRANKLIN

Dr Peter Franklin is a Senior Research Fellow at the School of Population Health, UWA, as well as a Senior Scientific Officer in the Environmental Health Directorate of the WA Department of Health. Peter has a background in environmental and occupational epidemiology, with specific research interests in air pollution, asbestos, children's environmental health, childhood lung function and exhaled breath markers of respiratory disease. His PhD and early postdoctoral research was on respiratory health effects associated with indoor air pollution. Peter's current research position is with the Occupational Respiratory Epidemiology research group, which predominantly focuses on mining exposures, but continues to have a role in children's environmental health research through his affiliation with the Telethon Kids Institute.

TIM WHITMORE

Dr Whitmore completing his undergraduate training in Medicine and Medical Science at the University of Western Australia in 2010, before proceeding to finish his Basic Physician Training at Royal Perth Hospital in 2015. He is now completing his Advanced Training in combined Respiratory Medicine and Infectious Diseases, and is currently a member of the TSANZ (WA Branch) Executive Committee.
BRUCE THOMPSON

TSANZ president elect

Prof Thompson is Head of the Physiology Service within the Department of Allergy, Immunology and Respiratory Medicine, Alfred Hospital and Central Clinical School Monash University. His research interest centres on the structure and function of the small airways in a range of respiratory conditions. He is a member of the Global Lung Initiative Static Lung Volume taskforce and currently on the international committee rewriting the Spirometry Standardisation document. His contribution to respiratory research and laboratory measurement was recognised in 2011 when he was awarded the ANZSRS research medal (Fellowship).
NEW INVESTIGATORS

Jesse Armitage

Jesse completed his BSc (Biomedical science) at the University of Western Australia in 2013 and honours with the HIV immunology group at Royal Perth Hospital in 2014. In 2015, Jesse began his PhD with the Stem Cell Unit at the Institute for Respiratory Health under the supervision of A/Prof. Yuben Moodley and Dr. Dino Tan. To date his research has focused primarily on the application of mesenchymal stem cells and stem cell-derived exosomes as a novel treatment for patients with COPD. His project encompasses aspects of inflammation and immunology in context to obstructive airways disease.

Samuel T. Montgomery

Samuel is a fourth year PhD student who focuses on an important but poorly researched area in CF; early life inflammation in the absence of infection. Specifically, his research aims to investigate the IL-1R inflammatory pathway in CF and the mechanisms surrounding inflammation arising from cell death following airway hypoxia and viral infections.

Tylah Miles

Tylah Miles is a first year PhD student at the Institute for Respiratory Health (UWA). She is completing her PhD within the Tissue Repair Group under the supervision of Associate Professor Cecilia Prêle. Her PhD entitled “The Immune Regulation of Lung Fibrosis” will investigate the way immune cells communicate with epithelial cells and fibroblasts to drive lung fibrosis and their potential as therapeutic targets.

Denby Evans

Denby Evans recently completed her honours degree through the University of Western Australia. Her project involved looking at the airway epithelium of preterm infants, with a specific focus on the airway epithelial response to human rhinovirus infection. Denby continues to work in the field of preterm birth and still maintains an avid interest regarding the effect of preterm birth on the airway epithelium.
MESENCHYMAL STROMAL CELL INFUSION ALTERS THE TRANSCRIPTOMIC PROFILE OF PERIPHERAL BLOOD MONONUCLEAR CELLS IN PATIENTS WITH STABLE COPD

Jesse Armitage\textsuperscript{1,2}, Dino Tan\textsuperscript{1,2}, Yuben Moodley\textsuperscript{1,2,3}

\textsuperscript{1} School of Biomedical Sciences, University of Western Australia, WA

\textsuperscript{2} Institute for respiratory health, Sir Charles Gardiner Hospital, Perth, WA

\textsuperscript{3} Department of respiratory medicine, Fiona Stanley Hospital, Murdoch, WA

Introduction & Aims: We have previously demonstrated that mesenchymal stromal cell (MSC) infusion into patients with stable chronic obstructive pulmonary disease (COPD) elicited systemic immunological responses that target inflammatory pathways relevant to COPD, including reductions in circulating pro-inflammatory and oxidative stress biomarkers. Despite these findings, there is still a need to understand the pathways, and potential immune regulators that are targeted by MSCs in COPD.

Methods: MSCs were infused into patients with stable COPD (n=9), and peripheral blood mononuclear cells (PBMCs) were isolated pre-infusion, 1 hour, 1 day, 2 days, 7 days post-infusion, followed by collection of PBMC 1 hour after a second infusion on the 7th day. RNA was extracted from PBMC and prepared for RNA sequencing. Global transcriptomic changes in PBMC were assessed using the weighted gene co-expression network analysis (WGCNA) pipeline.

Results: MSC infusion induced a transient differential gene expression that was highest after 1 day, but returned to baseline by 7 days. WGCNA identified a gene module that was enriched in pro-inflammatory genes (IL-1, TNF and IL-8) which was significantly downregulated following MSC infusion. Further network analysis identified IL-8 as a candidate regulator of this network.

Conclusion: We provide novel data showing MSC therapy in COPD patients induces transient changes following MSC infusion at the gene expression level, and identified IL-8 as a potential target of MSC-derived paracrine factors which may have significant implications in treating COPD. Exploration of MSC-mediated mechanisms that attenuate IL-8 may lead to the improvement of clinical outcomes.

Grant Support: The project was funded by a national health and medical research council (NHMRC) grant.
IL-1 IS ASSOCIATED WITH STRUCTURAL LUNG DISEASE IN CHILDREN WITH CYSTIC FIBROSIS

Samuel T. Montgomery1, A. Susanne Dittrich2,3, Luke W. Garratt4, Lidiya Turkovic4, Dario L. Frey2,3, Stephen M. Stick1,4,5, Marcus A. Mall2,7,8, Anthony Kicic1,4,5,6 & AREST CF1,5,9,10

1: School of Paediatrics and Child Health, Univ of Western Australia, Western Australia, Australia.
2: Translational Lung Research Center Heidelberg (TLRC), Univ of Heidelberg, Heidelberg, Germany;
3: Dept of Pneumology and Critical Care Medicine, Thoraxklinik at the Univ Hospital Heidelberg, Heidelberg, Germany
4: Telethon Kids Institute, Univ of Western Australia, Western Australia, Australia.
5: Dept of Respiratory Medicine, Princess Margaret Hospital for Children, Western Australia, Australia.
6: School of Public Health, Curtin Univ., Western Australia, Australia
7: Dept of Pediatric Pulmonology and Immunology, Charité-Universitätsmedizin Berlin, Berlin, Germany
8: Berlin Institute of Health (BIH), Berlin, Germany
9: Murdoch Children’s Research Institute, Melbourne, Victoria, Australia
10: Dept of Paediatrics, Univ of Melbourne, Melbourne, Victoria, Australia

Introduction & Aims: Little is known about the role of interleukin (IL)-1 and airway epithelial cells (AEC) in the pathogenesis of cystic fibrosis (CF) lung disease. This study aimed to measure IL-1 in the CF airway, assess for associations between inflammation and structural lung changes, and assess responses of AEC exposed to anoxia.

Methods: Bronchoalveolar lavage fluid (BALf) from 102 children with CF were used to determine IL-1α, IL-1β, IL-8 levels, neutrophil counts and neutrophil elastase (NE) activity, which were then associated with structural lung changes observed on chest computed tomography (CT) scans via PRAGMA-CF. AECs (n=9) exposed to anoxia for 48 hours were assessed via flow cytometry for viability, necrosis and apoptosis, reported as a percentage of control (mean ± SD). Wilcoxon signed-rank test was used to assess significant differences (p<0.05).

Results: IL-1α and IL-1β were detectable in BALf in absence of infection, increased in the presence of bacterial infection and correlated with IL-8 (r=0.64 & r=0.64; p<0.0001), neutrophils (r=0.71 & r=0.67; p<0.0001) and NE activity (p<0.01 and p<0.001). IL-1α had the strongest association with structural lung disease (1.20 [0.33, 2.06], p<0.01) in the absence of infection (uninfected: p<0.01 vs. infected: p=0.122). AEC exposure to anoxia for 48 hours decreased viability (61%±36; p<0.05), increased necrosis (209%±97; p<0.05) and increased apoptotic events (128%±29; p<0.05).

Conclusion: Our data associates IL-1α with early structural lung damage in CF potentially exacerbated by necrosis of AEC following anoxia and suggests this pathway as a novel anti-inflammatory target.

Grant Support: CFA, CFWA, USCF, German Federal Ministry of Education and Research
PLASMA CELL AND REGULATORY B CELL-INFLTRATION IN THE LUNGS OF BLEOMYCIN-TREATED MICE

Tylah Miles¹,², David R Pearce³, Robert O’Donoghue⁴, Andrew D Lucas⁵, Mark Fear⁶, Matthias Ernst³, Geoffrey J Laurent¹,², Darryl A Knight⁶, Gerard Hoyne⁷, Robin McAnulty³ and Steven E Mutsaers¹,², Cecilia M Prêle¹,²

¹Institute for Respiratory Health and Centre for Respiratory Health, The University of Western Australia
²Centre for Cell Therapy and Regenerative Medicine, School of Medicine and Pharmacology, The University of Western Australia and Harry Perkins Institute of Medical Research, Nedlands WA;
³Centre for inflammation and Tissue Repair, Division of Medicine, University College London, London UK.
⁴Olivia Newton John Cancer Research Institute and La Trobe University School of Cancer Medicine, Heidelberg, VIC;
⁵Burn injury research unit, school of biomedical sciences, The University of Western Australia
⁶The University of Newcastle, Callaghan, NSW;
⁷The University of Notre Dame, Perth WA.

Introduction & Aims: STAT3 and B cells are implicated in the development of lung fibrosis. We have previously demonstrated that hyper-activated STAT3, B cell-deficient gp130⁷⁵⁷F;μMT/⁻ mice are protected from bleomycin-induced lung fibrosis, suggesting B cells are important in the regulation of STAT3-mediated fibrosis. We hypothesise that the pro-fibrotic effects of STAT3 involve B cell-mediated immune regulation.

Methods: The effect of anti-CD20-therapy in bleomycin-treated wildtype and gp130⁷⁵⁷F mice on lung fibrosis and immune cell composition was examined. Mice were given two 100μg doses of an anti-CD20 antibody or IgG2a isotype control i.p. either 7 days prior and 7 days after bleomycin, or on day 10 and day 19 post-bleomycin treatment and the extent of fibrosis measured at 28 days.

Results: FACS analysis of blood taken on days 0, 7 and 28 days post-bleomycin-treatment revealed an almost complete depletion of CD19⁺ B cells in the circulation of wildtype mice but not gp130⁷⁵⁷F. However, the extent of fibrosis, assessed using micro-CT imaging and HPLC analysis of hydroxyproline was not significantly different between groups. Histological analysis revealed an abundance of CD5⁺ cells and CD138⁺ (plasma cells) in the lungs of the anti-CD20-treated mice. FACS analysis identified an expansion of CD138⁺ and CD5⁺ cells in the lungs of bleomycin-treated mice at day 28.

Conclusion: Although antibody depletion of follicular B cells had no effect on bleomycin-induced fibrosis, residual CD138⁺ plasma cells and CD5⁺ cells are abundant in the lungs of bleomycin-treated mice. The activity of these B cell subsets may contribute to the fibrotic phenotype.

Grant Support: This work is funded by NHMRC Project Grant GNT1067511 and British Lung Foundation Grant PPRG15-10.
AIRWAY EPITHELIAL CELL RESPONSES TO HUMAN RHINOVIRUS INFECTION IN INFANTS BORN PRETERM

Denby Evans¹, Kevin Looi¹, Shannon Simpson¹, Anthony Kicic¹,²

¹Telethon Kids Institute, Subiaco, WA, Australia
²Department of Respiratory Medicine, Perth Children’s Hospital, Perth, WA, Australia; Centre for Cell Therapy and Regenerative Medicine, School of Medicine and Pharmacology, The University of Western Australia and Harry Perkins Institute of Medical Research, Nedlands, WA, Australia; School of Paediatrics and Child Health, The University of Western Australia, Nedlands, WA, Australia; School of Public Health, Curtin University, Bentley, WA, Australia

Introduction & Aims: Acute respiratory infection is one of the leading causes of hospitalisation in children who are born prematurely. Increased susceptibility to viral infections, including human rhinovirus (HRV), by these children has been postulated to be due to an altered airway, specifically the epithelium. The aim of this study was to determine if nasal epithelial cells from preterm infants would display a defective response to HRV infection compared to term infants.

Methods: Nasal epithelial cells were studied from 8 term infants (>37 weeks GA; 62.5% male, 2.09-2.60 years) and 8 very preterm infants (<32 weeks GA; 62.5% male, 1.08-1.84 corrected years). Primary cultures were infected with HRV1b over time (6-96 hours) and at various multiplicity of infections (MOI 0.025-10). Collected supernatant and RNA was then analysed via ELISA and qPCR, to characterise apoptotic, innate immune and inflammatory responses to viral infection.

Results and Conclusion: Cell viability, cell lysis, receptor expression and viral replication did not significantly differ between cohorts. Furthermore, there was no significant increase in the apoptotic associate genes, CASP8, CASP3 and CASP7. Increases in inflammatory cytokine production did not differ between term and preterm, however baseline concentrations of IL-8 and RANTES were significantly elevated in preterm samples (IL-8: 5908pg/mL vs 4242pg/mL, p=0.03; RANTES: 37.04pg/mL vs 32.00pg/mL, p=0.003). Collectively, data suggests that HRV infection elicits a similar response in both term and preterm nasal epithelial cells, though underlying inflammation exists in the preterm airways which may contribute to the more severe infection phenotype.

Grant Support: Perpetual IMPACT
INTRODUCTION & AIMS: Dysfunctional breathing is a condition common in children, characterised by an abnormal breathing pattern, particularly excessive sighing and paradoxical breathing where chest and abdominal respiratory movements are out of phase. The subjective diagnosis of this condition in children is potentially confounded by a lack of knowledge on the normal paediatric breathing pattern. We characterised breathing pattern in children and determined the changes from childhood to adulthood.

METHODS: Children (13 ± 0.67 years; n = 14) and adults (23 ± 1.41 years; n = 14) with normal lung function and no history of respiratory disorders were recruited for study. Breathing pattern was assessed for two hours by respiratory inductance plethysmography with the subject in a sitting positon at rest while watching a movie. Unless otherwise stated, data are mean ± SEM.

RESULTS: Tidal volume and minute ventilation normalised to body weight were greater in children compared with adults (p<0.05). Sigh frequency was 20.06 ± 3.40 sighs/hour in children, which was also greater than in adults; 7.66 ± 1.36 sighs/hour (p<0.01). Incidence of paradoxical breaths was 2.25 per hour [IQR 1.24-5.59] in children and was again greater than in adults; 0.00 per hour [IQR 0.00-1.34] (p<0.01).

CONCLUSION: There are substantial differences in breathing pattern between children and adults: children sigh more frequently and take more paradoxical breaths compared with adults. These features observed in healthy children could complicate the diagnosis of dysfunctional breathing if the physician is unaware of maturational effects on breathing pattern.

GRANT SUPPORT: None
VARIABLE ACCURACY OF WEARABLE HEART RATE MONITORS DURING EXERCISE IN ADULTS WITH CYSTIC FIBROSIS

Madeline Gaynor¹, Abbey Sawyer¹,2,3, Sue Jenkins¹,2,3, Jamie Wood¹,2,3

¹Physiotherapy Department, Sir Charles Gairdner Hospital, Perth, Western Australia
²Institute for Respiratory Health, Western Australia
³School of Physiotherapy and Exercise Science, Curtin University, Western Australia

Introduction & Aims: In people with cystic fibrosis (CF), greater cardiopulmonary fitness is associated with increased survival and improved quality of life. Wearable activity monitors are a popular method of monitoring exercise with measures of heart rate (HR) used to indicate exercise intensity. We assessed the agreement of HR recordings obtained using the Fitbit Charge HR™, Polar® H7 HR sensor and Masimo SET® Rad-5v pulse oximeter (finger sensor) with the 3-lead electrocardiogram (ECG) during continuous and interval exercise.

Methods: Adults with CF completed 15 minutes of exercise on a cycle ergometer on two occasions whilst wearing the previously-mentioned devices. Firstly, participants cycled at 30% of estimated peak workload (Wpeak). Secondly, participants cycled at 1 minute intervals at 60% of Wpeak interspersed with 2 minutes of ‘rest’ (unloaded cycling). HR readings on all devices were recorded at minute intervals. Agreement of the HR recordings was analysed using the Bland Altman method.

Results: The Polar® H7 HR sensor and Masimo SET® Rad-5v pulse oximeter demonstrated good agreement with 3-lead ECG. The Fitbit Charge HR™, however, demonstrated poor agreement with 3-lead ECG, particularly during continuous moderate intensity exercise.

<table>
<thead>
<tr>
<th>Monitor</th>
<th>Bias (mean ± SD) compared to 3-lead ECG (bpm)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Continuous exercise</td>
</tr>
<tr>
<td>Fitbit Charge HR™</td>
<td>9.31 ± 17.30</td>
</tr>
<tr>
<td>Polar® H7</td>
<td>0.10 ± 1.17</td>
</tr>
<tr>
<td>Masimo SET® Rad-5v</td>
<td>1.00 ± 7.21</td>
</tr>
</tbody>
</table>

Conclusion: The Fitbit Charge HR™ is not recommended for assessing HR during exercise in adults with CF. Findings support the use of the Polar® H7 for accurate HR monitoring.

Grant Support: Supported by the SCGH Physiotherapy Department
CHARACTERISATION OF B CELL SUBSETS IN THE BLOOD AND LUNG OF PATIENTS WITH IDIOPATHIC PULMONARY FIBROSIS

Introduction & Aims: Idiopathic pulmonary fibrosis is the most common of the idiopathic interstitial pneumonias and is typically associated with prominent lymphoid aggregates of CD3+ T cells and CD20+ B cells within the lung tissue that are located near sites of active fibrosis. The presence of lymphoid aggregates outside of primary and secondary lymphoid tissues is a prominent feature of many autoimmune diseases and the presence of lymphoid foci can precede the onset of clinical disease.

Methods: We have examined the B cell profile of IPF patients and aged matched healthy controls using multicolour flow cytometry and analysed the serum for the presence of B cell cytokines BAFF and April, and CXCL13 a chemokine that promotes B cell migration to lung tissue.

Results: In contrast to healthy controls IPF patients show an accumulation of plasma B cells in the peripheral blood and a subset of patients show a rise in CD5+ CD23+ transitional B cells. Immunohistochemical staining of lung tissue from IPF patients revealed synchronous accumulation of CD138+ plasma cells and CD5+ B cells within the lymphoid aggregates of IPF patients. IPF patients showed a trend toward increased serum levels of BAFF, April and CXCL13.

Conclusion: The presence of plasma cells and CD5+ B cells in the blood and lung tissue of IPF patients raises the important question as to the role of B cells in IPF disease pathogenesis.

Grant Support: This work is funded by NHMRC Project Grant GNT1067511 and British Lung Foundation Grant PPRG15-10.
ONSET OF LEUKOCYTE DYSFUNCTION AND LUNG DAMAGE IN CYSTIC FIBROSIS


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Introduction & Aims: Although prior studies identified neutrophils, a leukocyte subset recruited from blood, and elastase (NE), a protease contained in their primary granules, as key contributors to chronic cystic fibrosis (CF) lung disease, their role in early disease remains unclear. We sought to determine the impact of neutrophils and NE on early lung disease in 3 CF paediatric cohorts (total n=104, age: 3 to 88 months).

Methods: We measured lung damage using chest computed tomography (CT) coupled with the PRAGMA-CF scoring system. In parallel, we phenotyped blood and bronchoalveolar lavage fluid (BALF) leukocytes by flow and image cytometry, and measured extracellular NE activity using spectrophotometric and Förster resonance energy transfer (FRET) assays. Children with aerodigestive symptoms were enrolled as controls.

Results: CF but not disease control children harbored BALF neutrophils with high exocytosis of primary granules, prior to the onset of bronchiectasis, and irrespective of age, or infection status. This cellular measure of NE exocytosis correlated with overall lung damage (Rho=0.517, p=0.0208), while the corresponding molecular measure of extracellular NE activity did not. This discrepancy is likely due to the recapture of extracellular NE by BALF neutrophils and macrophages in CF children.

Conclusion: Active NE exocytosis by airway neutrophils occurs in all CF children, and correlates with early lung damage. A complex cycle of NE exocytosis and recapture implicates CF airway fluid, neutrophils, and macrophages. These findings emphasize the need for integrated molecular and cellular approaches for biomarker development and interventions targeting neutrophilic inflammation in CF children.

Grant Support: National Institutes of Health (NIH, USA) R01HL126603 (to H.H., M.R.B., B.J.S., H.M.J., and R.T.); CF@LANTA Research Development Program Fellowship (to C.M.) as funded by the US Cystic Fibrosis Foundation (MCCART15R0); National Health and Medical Research Council (NHMRC; Australia) 111142505 (to L.W.G, R.T., A.K., and S.M.S.), and Peter Doherty Fellowship 1141479 (to L.W.G.); Western Australian Department of Health Merit Award (to L.W.G.); the German Ministry for Education and Research (FKZ 82DZL00401 and FKZ 82DZL004A1 to M.A.M.), the German Cystic Fibrosis Association Mukoviszidose e. V. (Project number 1605) and the Heidelberg Research Center for Molecular Medicine Career Development Fellowship (to A.S.D.). AREST CF is supported by several sources, including the NHMRC, NIH, US Cystic Fibrosis Foundation Therapeutics, and Cystic Fibrosis Australia. IMPEDE-CF is supported by the CF@LANTA Research Development Program Pilot Fund (to R.T. and L.G.) as funded by the US Cystic Fibrosis Foundation (MCCART15R0).
THE WESTERN AUSTRALIA THORACIC RESEARCH GROUP BIOSPECIMEN BANK

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Introduction & Aims: Lung disease affects over 2.6 million people in Australia leading to approximately 19,000 deaths each year. In 2011, lung cancer was associated with the largest proportion of cancer burden. In 2014, lung cancer remained the leading cause of cancer death in Australia and it is estimated that this will persist in 2017. Ongoing research is required to understand presentations and patterns of disease, risk factors for disease, prognostic factors, and potential treatments. Biospecimen banks are a valuable resource for the collection and storage of samples and data for future research purposes. The Western Australian Thoracic Research Group (WATRG) Biospecimen Bank was established for this purpose. We wish to explore ways to make this biobank available to all patients with lung malignancy in WA.

Methods: The WATRG Biospecimen Bank was established in early 2017 within a private organisation. The biospecimen bank involves the collection and storage of plasma and leftover biopsy and tissue specimens along with clinical data for future research purposes.

Results: Since the establishment of the biospecimen bank, 68 patients have consented and over 350 plasma samples have been collected and stored for future ctDNA analysis.

Conclusion: The large number of ctDNA samples from lung cancer patients can provide the opportunity to undertake research to investigate the role of ctDNA in the diagnosis, treatment and prognosis of lung cancer patients.

Grant Support: Funded by the Bendat Respiratory Research and Development Fund and St John of God Subiaco Hospital
THE NATURAL HISTORY OF DECLINING PULMONARY FUNCTION IN CHILDREN WITH DUCHENNE MUSCULAR DYSTROPHY

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Introduction & Aims: It is unclear which factors are associated with pulmonary function decline in Duchenne Muscular Dystrophy (DMD) and which pulmonary function test (PFT) is most sensitive to disease progression. We aimed to describe changes in pulmonary function over time and the impact of disease progression on PFTs in children with DMD.

Methods: Perth and Stanford PFT data (Vital Capacity (VC), Sniff Nasal Inspiratory Pressure (SNIP), Maximal Inspiratory Pressure (MIP), Peak Cough Flow (PCF), Maximal Expiratory Pressure (MEP)) from 2012 to 2017 were pooled. Disease progression was defined as becoming non-ambulant and/or use of non-invasive ventilation (NIV). Relationships between PFTs and clinical factors were examined using t-test and Generalised Linear Model.

Results: 67 children (all male), 18 (27%) from Perth. Mean VC z-score was abnormal (-2.23, -7.96 to 2.23). Mean annual change in VC z-score -0.26 (-4.91 to 6.64), SNIP % predicted -8% (-85% to 75%), MIP % predicted 3% (-199% to 162%), PCF % predicted 10% (-64% to 96%), MEP % predicted -7% (-98% to 156%). After adjusting for age, VC z-score was significantly lower in NIV users (p=0.012), MIP % predicted was significantly lower in non-ambulant children (p=0.000).

Conclusion: Mean VC z-score was abnormal and significantly associated with some clinical parameters indicating disease progression, therefore may be the most sensitive PFT for monitoring disease progression in DMD. MIP % predicted is significantly lower in those who are non-ambulant, however the wide range in values and high within-subject variability with repeated measures means the role in monitoring disease progression requires clarification.

Grant Support: Muscular Dystrophy Association of Western Australia
BEHAVIOUR CHANGE TECHNIQUES TO OPTIMISE DAILY PHYSICAL ACTIVITY OR PARTICIPATION IN EXERCISE IN ADOLESCENTS AND ADULTS WITH CHRONIC CARDIORESPIRATORY CONDITIONS: A SYSTEMATIC REVIEW

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²Physiotherapy Department, Sir Charles Gairdner Hospital, Perth, Western Australia, Australia
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Introduction & Aims: There is strong evidence that regular physical activity (PA) decreases the risk of developing chronic health conditions. Adherence to PA recommendations remains low, particularly during transitional phases such as the entry into adulthood. Due to a high treatment burden, uncomfortable symptoms experienced during PA and ‘usual’ life demands, people with chronic cardiorespiratory conditions are at a greater risk of not meeting PA recommendations.

The aim of this review was, in people aged 15-45 years with chronic cardiorespiratory conditions, to classify interventions aimed at optimising PA as ‘promising’ or ‘not-promising’, and to categorise behaviour change techniques (BCTs) within interventions (Michie v1 Taxonomy).

Methods: Nine databases registries were searched (inception-October 2017). Studies were eligible if they reported objective measures of PA before and after an intervention. Two authors independently assessed studies for eligibility. Interventions were classified as ‘promising’ if a between-group difference on PA was demonstrated. The Michie v1 Taxonomy, incorporating 93 possible BCTs, was used to unpack the BCTs described within interventions.

Results: Across the six included studies (n=396 participants), 19 (20%) BCTs were described. The interventions of three studies were classified as ‘promising.’ In these interventions, commonly described BCTs comprised goal setting, action planning, social support and information about antecedents. A number of techniques were used solely in ‘promising’ interventions.

Conclusion: Only 20% of the possible BCTs have been reported to optimise PA in this population. Those that offer most promise comprise goal setting, action planning and social support. We detected some BCTs that were specifically used only in ‘promising’ interventions.

Grant Support: This systematic review will form part of AS’s PhD, which is supported by Curtin University, and the Institute for Respiratory Health (Conquer Cystic Fibrosis Research Program), the Australian Cystic Fibrosis Research Trust (Top-Up Scholarship) and Sir Charles Gairdner Hospital (Research Advisory Committee Grant).
ASSESSING THE WOUND REPAIR CAPACITY OF THE UPPER AIRWAY EPITHELIUM OF CHILDREN BORN PRETERM

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Introduction & Aims: Very preterm birth is associated with life-long respiratory complications. In this study we aimed to characterise the repair mechanisms of the airway epithelial cells in preterm infants and investigate if neonatal exposures predict dysregulated wound repair.

Methods: Nasal epithelial cells (NECs) obtained by cytological brushing from children born very preterm (n=22, 25-31 weeks gestation) at 1.07-1.22yrs corrected age and full-term children (n=6, 2.4-6.5yr). Cell monolayers were cultured and subsequently wounded. Time-lapse images were captured, and percentage of wound repair was assessed by tracing the wound edge and comparing to the original wound. Wound repair was then correlated with common neonatal exposures in the NICU, such as days on oxygen or respiratory support.

Results: Healthy full-term NECs were found to fully repair wounds by 36 hrs. In contrast, the preterm NECs only completed 39.5% repair. At 72 hours post injury, ten preterm NECs exhibited extremely limited repair (20-40% repair), eight achieved 50-80% wound repair, one >90 repair with three achieving full repair. The % repair was not predicted by gestational age or any of the reported neonatal exposures.

Conclusion: Our data suggest that there is an inherently delayed ability of preterm infant AEC to successfully repair, and that there are no currently identifiable neonatal predictive factors. However, their delayed response and the inability to repair may contribute to their increased susceptibility to environmental pathogens and long-term respiratory morbidity.

Grant Support: Perpetual IMPACT Philanthropy Grant: IPAP2017/1355
THE USE OF INFERIOR VENA CAVA FILTERS (IVCFs) IN SUSPECTED AND CONFIRMED MALIGNANCY: A SURVEY OF RESPIRATORY PHYSICIANS, HAEMATOLOGISTS AND MEDICAL ONCOLOGISTS

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Introduction & Aims: Cancer Associated Thrombosis (CAT) is associated with 15 -20% of all diagnoses of Venous Thromboembolism (VTE). 20% of patients with CAT receive an IVCF annually. Several international societies release practice guidelines regarding indications for IVCF insertion. Adherence to these guidelines is known to be variable. Further, these guidelines do not comment on IVCF use in malignancy. We sought to determine usage rate of IVCF in different settings including where there is suspected or confirmed malignancy.

Methods: We surveyed Respiratory Physicians, Oncologists and Haematologists about 4 theoretical cases. Case 1: Post-operative patient develops a symptomatic PE and DVT. Case 2: Patient with gynaecological malignancy develops a PE before cancer related surgery. Case 3: Patient with recurrent VTE despite anticoagulation. Case 4: Patient with metastatic malignancy with extensive VTE.

Results: There were 56 responses, 32 Respiratory Physicians (57%) and 24 Haematologists/Oncologists (43%). 77% were consultants and 23% were advanced trainees. Physicians chose to insert an IVCF in 74/224 scenarios. Case 1; 30 (21 respiratory physicians: 9 haem/oncologists), Case 2; 21 (13:8), Case 3; 11 (3:8) and Case 4; 12 (8:4). Respiratory physicians were more likely to insert an IVCF for Case 1, and less likely to do so for case 3. In Case 1, Physicians preferring to delay anticoagulation post-surgery were 8 times more likely to insert an IVCF. Compliance with guidelines was variable.

Conclusion: The heterogeneity in responses highlights the variations in VTE management especially in CAT. International Societies should consider addressing IVCF use specifically in the setting of CAT.

Grant Support: The authors would like to acknowledge the Bendat respiratory and research development fund for their support of this project.
WHEN IS A PE NOT A PE

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²Department of Cardiothoracic Surgery and Transplantation, Fiona Stanley Hospital, Western Australia
³Advanced Lung Disease and Lung Transplantation Unit, Fiona Stanley Hospital, Western Australia

Case Study: We present a case of a 46 year old female with a presumed diagnosis of multiple unprovoked pulmonary emboli. She continued to deteriorate from a cardiorespiratory point of view despite optimal medical therapy. At surgical thrombectomy she was identified to have an intimal soft tissue lesion arising from the right pulmonary artery. This lesion was subsequently diagnosed as a pulmonary artery sarcoma. At nine months post surgery she has undergone six cycles of chemotherapy and has stable disease with no metastases.

Pulmonary artery sarcoma (PAS) is a rare neoplasm which carries a poor prognosis. It typically presents at an advanced stage with pulmonary vascular obstruction. Surgical debulking is the mainstay of therapy to restore ventilation perfusion mismatching and relieve right heart strain. Median survival of these patients is 20 months with adjuvant chemo-radiotherapy.

Grant Support: Bendat Respiratory Research and Development Fund
THINKING OUTSIDE THE LUNGS - BREATHELESSNESS IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD): A FEASIBILITY STUDY

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Introduction & Aims: In people with COPD, lung hyperinflation and spinal rigidity contributes to exertional breathlessness and limits exercise capacity. Breathlessness is a debilitating symptom that impacts on daily activities. We investigated in people with stable COPD, whether thoracic mobilisations decreased breathlessness and improved lung function more than a placebo intervention.

Methods: In this double-blind cross-over trial participants received both interventions (thoracic spinal mobilisations and placebo static hands-on only) in randomised order on 2 separate occasions within a 2-week period. Following each intervention, participants performed a standardised 6-minute treadmill walking exercise task. Primary outcome measures were breathlessness (Borg scale and Multidimensional Dyspnea Profile [MDP]) and lung function (forced expiratory volume in one second [FEV₁], forced vital capacity [FVC] and inspiratory capacity [IC]).

Results: 11 participants (6 male, 69±6 years, [mean±SD], FEV₁ 45 ±25 %predicted) completed the study. Breathlessness data are shown in the table.

<table>
<thead>
<tr>
<th></th>
<th>Thoracic mobilisations</th>
<th>Placebo</th>
<th>^p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Borg scores</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre-intervention</td>
<td>0.5 (0.0-0.7)</td>
<td>0.5 (0.0-1.0)</td>
<td>0.84</td>
</tr>
<tr>
<td>Post-exercise task</td>
<td>3 (3.0-3.0)</td>
<td>3 (2.0-3.5)</td>
<td>0.78</td>
</tr>
<tr>
<td>MDP post-exercise task</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Immediate perception (0-60)</td>
<td>7 (4.5-18.0)</td>
<td>10 (5.0-17.5)</td>
<td>0.72</td>
</tr>
<tr>
<td>Emotional response (0-50)</td>
<td>1 (0.0-4.0)</td>
<td>0 (0.0-2.5)</td>
<td>0.60</td>
</tr>
</tbody>
</table>

Following the thoracic mobilisations, 6 (55%) participants reported a reduction in breathlessness (immediate perception) on the MDP. There were no significant changes in lung function between the two interventions.

Conclusion: No adverse events occurred and the protocol had high patient acceptance. Further research in a larger sample is required to determine the impact of thoracic mobilisations on breathlessness following exercise in this patient population.

Grant Support: This research was supported by Sir Charles Gairdner Hospital Physiotherapy Department.