RESEARCH TO PRACTICE 2018
27-29 MARCH 2018
BRISBANE, QUEENSLAND

EARLY CAREER RESEARCHER AWARD
EXERCISE SCIENCE + HEALTH FINALISTS

Wednesday, 28 March 2018
11:00am – 12:30pm

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Presentations:

145
Suppressed osteocalcin and muscle protein signalling mediate glucocorticoid-induced basal and post-exercise insulin resistance in humans
Lewan Parker

210
Feasibility, Safety and Efficacy of High Intensity Interval Training in Patients with Chronic Kidney Disease: A Randomised Pilot Study
Kassia Beetham

211
The effects of personal, non-personal and no exercise supervision on cardiometabolic health in the workplace: a 16-week randomised controlled trial
Jayden Hunter

269
Glucose-stimulated insulin secretion is reduced in adults with prediabetes following two-weeks of exercise training independent of intensity
Monique Francois

330
Optimising exercise for cognitive health in older adults: a randomised controlled clinical trial
Mia Schaumberg

367
CD8+ T-cell depletion abolishes the anti-metastatic effects of Voluntary Running in a mouse model of Breast Cancer
Helene Rundqvist

Abstracts listed on following pages

RESEARCH TO PRACTICE 2018
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EXERCISE SCIENCE + HEALTH FINALISTS

145

Suppressed osteocalcin and muscle protein signalling mediate glucocorticoid-induced basal and post-exercise insulin resistance in humans

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Background. Glucocorticoids (GC) are used for the treatment of inflammatory and autoimmune conditions but lead to the development of insulin resistance. In mice, GC-induced insulin resistance occurs primarily through impaired osteoblast function and uncarboxylated osteocalcin (ucOC) secretion, however, this has yet to be established in humans. Furthermore, acute exercise increases ucOC secretion and insulin sensitivity, however the effects of GC on post-exercise glycaemic control and ucOC secretion are unknown. We investigated the effect of GC on basal and post-exercise insulin sensitivity, ucOC secretion, and insulin signalling.

Methods. In a randomised, crossover, double-blind study, nine healthy males (Age: 28 ± 2 years; BMI: 24 ± 1; Mean ± SEM) completed two separate cycling sessions 12 hours after ingesting either GC (20 mg prednisolone) or placebo. The homeostatic model assessment was used to assess basal insulin resistance and a 2-hour euglycaemic-hyperinsulinaemic clamp was commenced 3 hours after exercise to assess insulin sensitivity.

Results. Compared with placebo, GC decreased serum ucOC (-24%, $P<0.01$), which was associated with decreased basal (-47±5%, r=0.54, $P<0.01$) and post-exercise insulin sensitivity (-34±5%, r=0.72, $P<0.01$). GC decreased skeletal muscle protein abundance of the ucOC receptor (GPRC6A: -16%, $P<0.05$) and attenuated the post-exercise insulin-stimulated phosphorylation of glucose uptake signalling proteins $mTOR^{Ser2481}$, Akt^{Ser374} and $AS160^{Thr642}$ (-59%, -61% and -50%, respectively; $P<0.05$). Attenuated mTOR, Akt and AS160 signalling correlated with lower ucOC (r=0.61-0.71, $P<0.05$) and lower post-exercise insulin sensitivity (r=0.54-0.75; $P<0.05$).

Conclusion. GC-induced basal and post-exercise insulin resistance in humans is linked to the suppression of ucOC secretion and signalling. Targeting ucOC may be a novel approach for improving glycaemic control in populations who are insulin resistant and/or undergoing GC therapy.

RESEARCH TO PRACTICE 2018
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BRISBANE, QUEENSLAND

EARLY CAREER RESEARCHER AWARD
EXERCISE SCIENCE + HEALTH FINALISTS

210

Feasibility, Safety and Efficacy of High Intensity Interval Training in Patients with Chronic Kidney Disease: A Randomised Pilot Study

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Introduction & Aims Patients with chronic kidney disease (CKD) have significantly reduced cardiorespiratory fitness, which greatly impacts quality of life and may exacerbate disease progression. High intensity interval training (HIIT) increases mitochondrial biogenesis and fitness in chronic disease populations, however has not been studied in CKD. The aim of this pilot study was to compare the feasibility, safety and efficacy of HIIT with moderate intensity continuous training (MICT) in CKD.
Methods Fourteen participants with stage 3-4 CKD were randomized to either HIIT (n=9) or MICT (n=5) for three sessions of supervised exercise training per week, for 12 weeks. In each session the HIIT group completed 4x4 minute intervals at 85-95% maximum heart rate (MHR), and the MICT group completed 40 minutes of continuous training (65% MHR). Feasibility was assessed via adherence to the exercise prescription. Safety was examined by adverse event reporting. Efficacy was determined by mitochondrial biogenesis (PGC-1α protein levels from muscle tissue), cardiorespiratory fitness (VO_2peak) and exercise capacity (time on test calculated METs).
Results Participants completed a similar number of sessions (HIIT median [interquartile range]) 33[7] vs. MICT 33.5[3.3] sessions). There were no adverse events reported in either group during the training. There were no significant ($p>0.05$) time or group x time effects for cardiorespiratory fitness (HIIT pre 22.3±7.6, post 22.5±7.0; MICT pre 21.7±6.1, post 23.2±6.7 ml/kg/min) or PGC-1α (HIIT pre 1.2±0.3, post 1.4±0.4, MICT pre 1.1±0.2, post 1.2±0.4), however there was a significant time effect for exercise capacity (HIIT pre 9.8±3.4, post 10.6±2.9; MICT pre 8.8±3.4, post 10.2±3.6 METs, $p=0.01$).
Conclusion This pilot study identified that HIIT is a feasible and safe option for appropriately screened patients with CKD although it had no added benefit on mitochondrial biogenesis, cardiorespiratory fitness or exercise capacity compared with MICT.

RESEARCH TO PRACTICE 2018
27-29 MARCH 2018
BRISBANE, QUEENSLAND

EARLY CAREER RESEARCHER AWARD
EXERCISE SCIENCE + HEALTH FINALISTS

211

The effects of personal, non-personal and no exercise supervision on cardiometabolic health in the workplace: a 16-week randomised controlled trial

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Introduction & Aims: Supervised exercise has achieved greater health outcomes than unsupervised exercise in clinical exercise trials, however, comparisons between supervision types have not been made in healthy employees. This randomised controlled trial compared the effectiveness of personal, non-personal and no-exercise supervision in the workplace to improve employee cardiometabolic health.
Methods: Eighty-five Australian university employees (62 female; mean±SD 43.2±9.8 years) were randomised to either personal (1:1; SUP, N=28) supervision, non-personal (typical gym-based; NPS, N=28) supervision or unsupervised control (CON, N=29) exercise groups. Participants received a 16-week, individually tailored, moderate-to-high intensity aerobic and resistance exercise program to complete at an onsite gymnasium (SUP and NPS) or without access to a specific exercise facility (CON). Changes to cardiorespiratory fitness (CRF; $\dot{V}O_2$ peak), muscular strength (1RM bench and leg press) and body composition (body fat % measured by DXA) were analysed using repeated measures ANOVA.
Results: Mean changes to CRF were greater (p<0.01) with SUP (+10.4±11.1%) compared to CON (+3.8±8.9%), but not different to NPS (+8.6±8.2%). When compared to CON (+1.7±7.7%), mean upper body strength changes were significantly greater with both SUP (+12.8±8.4%; p<0.001) and NPS (+8.4±7.3%; p<0.05). Mean lower body strength changes were greater with SUP (+26.3±12.7%) compared to both NPS (+15.0 ± 14.6%; p<0.05) and CON (+4.1 ± 12.4%; p<0.001), and NPS compared to CON (p<0.01). Mean reductions to body fat were greater with SUP (-2.2±2.2%) compared to both NPS (-0.6±1.9%; p<0.05) and CON (-0.7±1.9%; p<0.05). **Conclusion:** Access to an onsite exercise facility and personal (1:1) exercise supervision confers greater improvements to CRF than unsupervised exercise, and greater improvements to muscular strength and body composition than non-personal (typical gym) supervision or unsupervised exercise over 16 weeks.

RESEARCH TO PRACTICE 2018
27-29 MARCH 2018
BRISBANE, QUEENSLAND

EARLY CAREER RESEARCHER AWARD
EXERCISE SCIENCE + HEALTH FINALISTS

269

Glucose-stimulated insulin secretion is reduced in adults with prediabetes following two-weeks of exercise training independent of intensity

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Background: Prediabetes is characterized by defects in both insulin secretion and sensitivity that lead to impaired fasting and/or glucose tolerance. Although exercise has established effects on enhancing insulin sensitivity, the impact of short-term exercise on glucose-stimulated insulin secretion (GSIS) and incretin hormones is unclear prior to meaningful weight loss.
Purpose: To examine the effect of short-term interval and continuous exercise on GSIS in adults with prediabetes.
Methods: Thirty-one adults (Age: 61 ± 8 y; BMI 33 ± 6 kg/m^2) with prediabetes according to ADA 75g OGTT criteria and/or HbA1c were randomized to either 2-wks of energy-matched interval (INT: 60-min/d alternating 3-min at 90 and 50% HRpeak) or continuous training (CONT: 60-min/d 70% HRpeak). VO_2peak, body mass and a 120-min 75g OGTT were performed before and after training. Plasma glucose and C-peptide were collected at 30-min intervals to determine early- ($tAUC_{30min}$) and total-phase ($tAUC_{120min}$) GSIS (ΔC-peptide/Δglucose). GLP-1_{active} and GIP were also measured during 0, 30 and 60-min of the OGTT to assess incretin effects.
Results: Training reduced body mass (-0.6 ± 1.1 kg) and increased VO_2peak (+1.1 ± 2.1 mL/kg/min) independent of intensity (Time: P<0.05). INT and CONT exercise reduced glucose (by 6%, P=0.04) and C-peptide $tAUC_{120min}$ (by 17%, P<0.01). The intervention also decreased total-phase GSIS (by 12%, P<0.01) to a similar extent (Interaction: P=0.88), with no change in early-phase responses. Training tended to reduce fasting GLP-1_{active} (Time: P=0.07), whereas fasting GIP was reduced only after INT (Interaction: P=0.03). Incretin $tAUC_{60min}$ and peak stimulation responses to the OGTT were unchanged.
Conclusions: Short-term exercise training reduces GSIS independent of intensity in people with prediabetes. Further work is required to understand the mechanism by which exercise alters pancreatic insulin secretion to optimize diabetes prevention.

RESEARCH TO PRACTICE 2018
27-29 MARCH 2018
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EARLY CAREER RESEARCHER AWARD
EXERCISE SCIENCE + HEALTH FINALISTS

330

Optimising exercise for cognitive health in older adults: a randomised controlled clinical trial

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Introduction: Physical exercise is a promising strategy for combatting cognitive decline during aging. However, a systematic, multimodal approach to understanding the mechanisms by which exercise may prevent or even reverse cognitive decline is lacking. Without a thorough understanding of the optimal parameters and mechanisms by which exercise provides resilience to cognitive decline with age, the benefits of clinical and community exercise programs for cognitive decline cannot be fully realised.
Aims: We aimed to investigate optimal parameters for exercise prescription to combat age-related cognitive decline utilising novel blood-based biomarkers and high-resolution multimodal imaging.
Methods: 99 apparently healthy older adults (female n=52, age=71±4y, BMI=26±4kg.m^{-2}) met all inclusion criteria and were enrolled in the randomised, controlled clinical trial. Participants provided written informed consent, underwent a comprehensive baseline assessment of cognition, mental and physical health and fitness, and provided a fasted blood sample. 7-Tesla functional and structural MRI assessed hippocampal volumes and functional connectivity. Participants were randomised into a low-, moderate- or high-intensity exercise intervention and completed 3 supervised exercise sessions per week for 6 months. Cognition and blood biochemistry were assessed at monthly intervals and participants completed a follow-up assessment after the 6-month intervention. Recruitment and data collection is on-going.
Implications: This is the first systematic, multimodal approach to investigate the mechanistic effects of exercise on cognitive function. Understanding the neurobiological mechanisms that mediate the effects of exercise on cognitive function in health and disease will improve public health recommendations that optimise the neuroprotective and/or neuro-regenerative effects of exercise, and provide robust blood-based biomarkers to aid in the development of effective treatments for dementia.

RESEARCH TO PRACTICE 2018
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EARLY CAREER RESEARCHER AWARD
EXERCISE SCIENCE + HEALTH FINALISTS

367

CD8+ T-cell depletion abolishes the anti-metastatic effects of Voluntary Running in a mouse model of Breast Cancer

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Aim: to evaluate the effect of voluntary running on tumor progression and metastasis in the PyMT mouse model of breast cancer.
Methods: From 4 weeks of age, PyMT mice were housed with access either to wirelessly recording running wheels or locked control wheels. Tumor growth was monitored continuously, tumor stage and pulmonary metastases were determined histologically at the 12 week endpoint. In a follow up study, pre-trained mice were injected intravenously with PyMT derived tumor cells and after an additional 10 weeks of voluntary running, pulmonary metastases and immune cell infiltration was quantified. The CD8+ T-cell population was deleted using CD8 specific antibodies.
Results: PyMT mice average running distance was 6.4 km/day. No significant effects of voluntary running on tumor-initiation, volume or stage were found. However, a reduced number of metastases were observed in mice with access to running wheels. Significant reductions in pulmonary metastasis frequency were also found in runners after intravenous injections of tumor cells and running mice had a lower number of metastases with a high proliferation score. Metastatic lesions from running mice showed higher content of Granzyme B positive cells, indicating an increased infiltration of cytotoxic T-cells. Depletion of CD8+ cells abolished the reduction in metastatic burden found in running mice compared to non-running mice.
Discussion: In this highly aggressive, genetic, breast cancer model, an average of 6 km/day of voluntary running showed little effect on tumor formation and growth. However, the findings suggest that physical activity reduced outgrowth of metastatic lesions through an increased infiltration of cytotoxic immune cells.