





DAY 2: Chondroitin sulfate and exercise pressor reflex

	
Name	Mr. Norio Hotta
Country	Japan
Department	-
E-mail	-
Title	Chondroitin sulfate and exercise pressor reflex
Abstract	<p>During exercise, cardiovascular functions are augmented, contributing to the supply of oxygen to the exercising muscles. Sympathetic nervous activity is increased during exercise by the afferent inputs from muscles. This response is known as the exercise pressor reflex, and the input signals are generated by activation of muscular thin fiber afferents (group III and IV fibers). Exercise-induced tissue acidosis could augment the exercise pressor reflex. We previously reported that acid causes mechanical sensitization of thin muscle afferents. We also found that this acid-induced mechanical sensitization was attenuated by the exogenous chondroitin sulfate (chondroitin sulfate is well known as a slow-acting drug for osteoarthritis, which was officially accepted by the WHO/ International League of Associations for Rheumatology Task Force in 1994). These results suggest a novel mechanism for mechanical sensitization of thin muscle afferents that involve the extracellular matrix chondroitin sulfate proteoglycan and that daily intake of chondroitin sulfate could potentially attenuate the exaggeration in cardiovascular responses during exercise with tissue acidosis. In this presentation, we would like to discuss the clinical implications of the finding that exogenous chondroitin sulfate suppresses the acid-induced augmentation in mechanical response in thin muscle afferents.</p>




DAY 2: New applications of herbal ingestion in sports performance and health

	
Name	Ahmad Al Khatib
Country	Kuwait
Department	Dasman Diabetes Institute, Kuwait
E-mail	drahmadalkhatib@gmail.com
Title	New applications of herbal ingestion in sports performance and health
Abstract	<p>Herbal ingestions are common amongst many cultures for centuries, and have been used to enhance both health and sports performance outcomes. Numerous medicinal plants and natural herbs have been recently reviewed for their physiological mechanisms responsible for preventing chronic diseases including diabetes and cardiovascular disease. Examples include herbal tea ingestions (green and black tea, yerba maté) These mechanisms can be applied successfully to understand and optimize ergogenic herbal applications for sports performance and training outcomes, including weight and fat loss, enhanced fat metabolism, stimulated central nervous system, reduced post exercise inflammation, enhanced exercise oxidative capacity and reduced oxidative stress. Furthermore, applying a multi-component models encompassing behavioral and lifestyle components when detecting whether and how herbal ingestions enhance or augment the exercise benefits is essential to provide additional answers into their effectiveness. However, research in this area is still limited possibly due to the inter-disciplinary research requirements, complex research design, multi-level analysis and safety challenges. Well-designed human trials are needed to test the effectiveness of herbal ingestions at rest and at different exercise conditions. This includes different exercise intensity domains, different durations and different modes and modalities. Introducing novel applications of herbal ingestions can provide a bridge between the understanding of clinical and sports performance outcomes.</p>




DAY 2: Nutritional Supplementation and Blood Pressure

	
Name	Mr. Grant N. Pierce
Country	Canada
Department	-
E-mail	-
Title	Nutritional Supplementation and Blood Pressure
Abstract	<p>Nutritional supplementation and blood pressure</p> <p>Grant N. Pierce, PhD, FACC, FAHA, FAPS, FISHR, FIACS, FCAHS, FRSM (London), FRSC</p> <p>Executive Director of Research, St Boniface Hospital, and Faculty of Health Sciences, University of Manitoba, Winnipeg, Manitoba, Canada</p> <p>Hypertension (high blood pressure), known as the “silent killer”, accounts for approximately half of the nearly 17 million cardiovascular deaths worldwide making it the leading risk factor attributed to death globally. Approximately 40 per cent of adults aged 25 plus years have hypertension worldwide. If left uncontrolled, hypertension can lead to heart attacks, strokes, and many other serious complications. Current medications used to control hypertension are not always effective in controlling blood pressure and having a food that will compliment pharmaceutical approaches in the control blood pressure represents a viable strategy. We shall discuss the comparative power of dietary modification, lifestyle changes and medication for controlling hypertension. This talk will also discuss the results from both laboratory-based work and clinical trials from our lab on the effects of dietary flaxseed on cardiovascular disease with a focus on blood pressure regulation and its potential for affecting cardiovascular disease. Supported by CIHR, ARDI, Saskflax, Western Grains Research Fdn and Pizeys’ Foods.</p>




DAY 2: Oxygen: good gas or bad gas in hyperbaria? A matter of perspective

	
Name	John R. Clarke
Country	USA
Department	Navy Experimental Diving Unit
E-mail	John.r.clarke@navy.mail
Title	Oxygen: good gas or bad gas in hyperbaria? A matter of perspective
Abstract	<p>Humans have a love-hate relationship with oxygen. We need it to survive, but when we change our environment, or do atypical things like scuba diving, or freediving while breathholding, either too much oxygen or the lack thereof becomes deleterious to our health. Under hyperbaric conditions, too much oxygen can cause pulmonary oxygen toxicity and central nervous system seizures. Underwater, CNS seizures may cause death by drowning. At altitude, hypobaria or low partial pressures of oxygen can cause high altitude pulmonary edema, and death. I will illustrate the varied ways that the availability of oxygen can cause physiological problems in diving. In both freediving and apneic swimming, oxygen can be consumed beyond levels supporting consciousness. The result can be drowning as described in at least one case of shallow water blackout and a case of loss of consciousness from apnea. Given a gradual enough exposure to altitude, people adapt to the hypoxia of high altitude, an advantage in sports competitions at high elevation cities. However, adapting to hyperoxia at depth, or hypoxia in dynamic apnea and deep freediving is far more questionable, and risky. Nevertheless, the desire to learn how to tolerate both is strong for safety reasons in military diving, freediving, and even in space. For instance, what would it take to tolerate the low oxygen environment in water when using artificial gills? How do animals survive severe hypoxia? We will explore the nature of hyperoxia and hypoxia by imagining the solutions to these and other technological and biological challenges.</p>




DAY 2: Gravity of exercise for a trip to Mars

	
Name	Alan R Hargens
Country	USA
Department	University of California, San Diego
E-mail	ahargens@ucsd.edu
Title	Gravity of exercise for a trip to Mars
Abstract	<p>During spaceflight astronauts experience musculoskeletal losses and moderate to severe back pain. There is also a significant incidence of herniated intervertebral disc in astronauts, especially in the first year after return to Earth. Our spine studies incorporate pre- and post-flight tests of International Space Station crew members to ascertain mechanisms of back pain and injury. Exercise countermeasures should include integrated cardiovascular and musculoskeletal exercises to reproduce normal, daily Earth-like stresses. Our long-term objectives are to: 1) promote crew health and well-being inflight, 2) optimize post-flight rehabilitation and 3) translate our findings for Earth Benefit. Supported by grants from NASA.</p>



DAY 2: Treat the muscles to treat the lungs: exercise training to reduce the oxidative stress

	
Name	K. Sathyavelu Reddy
Country	India
Department	Department of Zoology, S.V.University
E-mail	sathyakreddy56@gmail.com
Title	Treat the muscles to treat the lungs: exercise training to reduce the oxidative stress
Abstract	<p>Regular physical activity is an effective non-pharmacological therapy for prevention of pathologic conditions, including pulmonary and cardiovascular diseases, diabetes mellitus, metabolic syndrome and those associated with systemic inflammation. Experimental evidence indicates that regular exercise is able to increase antioxidant defenses in heart, muscle and liver of rats and to prevent the lipid peroxidation and oxidative damage to protein present in lung injury. Our studies also revealed 12 weeks period of treadmill exercise training has beneficial in preventing the age associated amendments in antioxidant machinery of different loco motor muscle fiber types. It seems exercise enhances antioxidant enzyme activity in the organs possibly mediated by adenosine which is released from ATP, when ATP is broken down during exercise. Adenosine plays a cyto-protective role via adenosine receptor against the generated Reactive Oxygen species (ROS). Glucocorticoids (GC) have anti-inflammatory activities and are used to suppress inflammation in chronic diseases such as asthma and rheumatoid arthritis. GC levels were significantly higher in the exercised old animals than in the sedentary animals. Exercise is believed to be beneficial to improve the quality of life retarding age related diseases. Regular physical exercise attenuating potentially harmful oxidative damage and suppressing inflammatory processes.</p>




DAY 2: Oxidative stress, Mitochondrial Dynamics, and Muscle Atrophy

	
Name	Yu Kitaoka
Country	Japan
Department	Department of Human Sciences, Kanagawa University
E-mail	kitaoka@kanagawa-u.ac.jp
Title	Oxidative stress, Mitochondrial Dynamics, and Muscle Atrophy
Abstract	<p>Mitochondria are dynamic organelles, continuously remodeling through the process of fusion and fission to maintain the quality and function. Previous studies demonstrated that exercise training increases the amount of reticular mitochondria, while muscle denervation and aging induce the fragmentation of mitochondrial networks, suggesting that mitochondrial morphology is likely to be coupled with muscle function. Age-related and disuse-induced skeletal muscle atrophy is associated with increased oxidative stress. The objective is to examine whether deficiency of Nrf2, a master regulator of antioxidant transcription, promotes 1) denervation-induced and 2) Age-related mitochondrial fragmentation and muscle atrophy. I will discuss our recent findings on the effects of oxidative stress on skeletal muscle mitochondria.</p>






DAY 2: Modulation of Gi Proteins in Hypertension: Role of Angiotensin II and Oxidative Stress

	
Name	Madhu B. Anand-Srivastava
Country	Canada
Department	University of Montreal
E-mail	madhu.anand-srivastava@umontreal.ca
Title	Modulation of Gi Proteins in Hypertension: Role of Angiotensin II and Oxidative Stress
Abstract	<p>Guanine nucleotide regulatory proteins (G proteins) are a family of GTP-binding proteins that play an important role in the regulation of a variety of physiological functions including blood pressure. Alterations in the levels of Gi proteins lead to various pathological states such as hypertension. We have previously shown an enhanced expression of Gi<math>\alpha</math> proteins in spontaneously hypertensive rats (SHR) and other models of hypertensive rats. The enhanced expression of Gi<math>\alpha</math> proteins precedes the development of hypertension in SHR. Since the levels of vasoactive peptides including angiotensin II (Ang II) are enhanced in SHR, this study examines the role of Ang II and associated signaling in enhanced expression of Gi<math>\alpha</math> proteins in SHR. Aortic vascular smooth muscle cells (VSMC) from 12 week-old SHR and Wistar-Kyoto rats (WKY) were used for the present studies. VSMC from SHR exhibited enhanced expression of Gi<math>\alpha</math> proteins as compared to age-matched WKY. The increased levels of Gi<math>\alpha</math> proteins were restored to control WKY levels by captopril; losartan; AT1 receptor siRNA as well as PD 98059; a selective inhibitor of MAP kinase. In addition, losartan also restored the enhanced levels of superoxide anion (O<sub>2</sub><sup>-</sup>) exhibited by SHR to control WKY levels. Furthermore, treatment of VSMC with antioxidants such as N-acetyl-L-cysteine (NAC) or diphenyleneiodonium (DPI) and PD98059 reversed the enhanced expression of Gi<math>\alpha</math> proteins and enhanced ERK1/2 phosphorylation to control levels. These results suggest that enhanced levels of Gi<math>\alpha</math> in SHR may be attributed to Ang II-induced enhanced oxidative stress which exerts its effects through MAP kinase signaling pathway. (Supported by grant from CIHR).</p>





DAY 2: The Role of Molecular Hydrogen Treatment in Adaptation of The Heart to Oxidative Stress

	
Name	Jan Slezak
Country	Slovakia
Department	Institute for Heart Research, Center of Experimental Medicine, Slovak Academy of Sciences
E-mail	jan.slezak@savba.sk
Title	The Role of Molecular Hydrogen Treatment in Adaptation of The Heart to Oxidative Stress
Abstract	<p>During oxidative stress highly reactive hydroxyl and nitrosyl radicals can damage critical macromolecules such as DNA, proteins or membranes and induce cell damage. Scavenging of free radicals acts preventively or therapeutically. A number of substances serve as scavengers, protecting cells and tissues against oxidative damage but most of them are either not completely effective or have side effects. A unique substance is molecular hydrogen (H<sub>2</sub>). Effects of hydrogen have been attributed to four major molecular mechanisms: 1 - a specific scavenging activity of hydroxyl radicals. 2 - scavenging activity of peroxynitrite. 3 - alterations of gene expressions. 4 - signal modulating activities (via Nrf2?)</p> <p>In experiments on Wistar rats, we studied the effect of H<sub>2</sub> on oxidative stress of the heart (Malondialdehyde and Tumor necrosis factor) and ATP production in the mitochondria of the heart after mediastinal irradiation of 10 Gy.</p> <p>Conclusion: In the early stage after irradiation H<sub>2</sub> preferentially scavenges radiation produced hydroxyl radicals. H<sub>2</sub> acts as the antioxidant protection</p>



of the coenzyme Q concentration in the cardiac tissue and in the mitochondria of the cardiac muscle and the stimulates ATP production in the mitochondria of the heart. In the later stages after irradiation there may emerge yet another mechanism of H<sub>2</sub> effects trough stimulation of Nrf2 and production of innent antioxidative enzymes. H<sub>2</sub> can protect myocardium from radiation-induced injury, decrease MDA and TNF alfa. H<sub>2</sub> application has anti-inflammatory, anti-apoptotic and antioxidant properties. H<sub>2</sub> may be novel effective clinical tool for treating radiation injury and oxidative stress-related diseases.