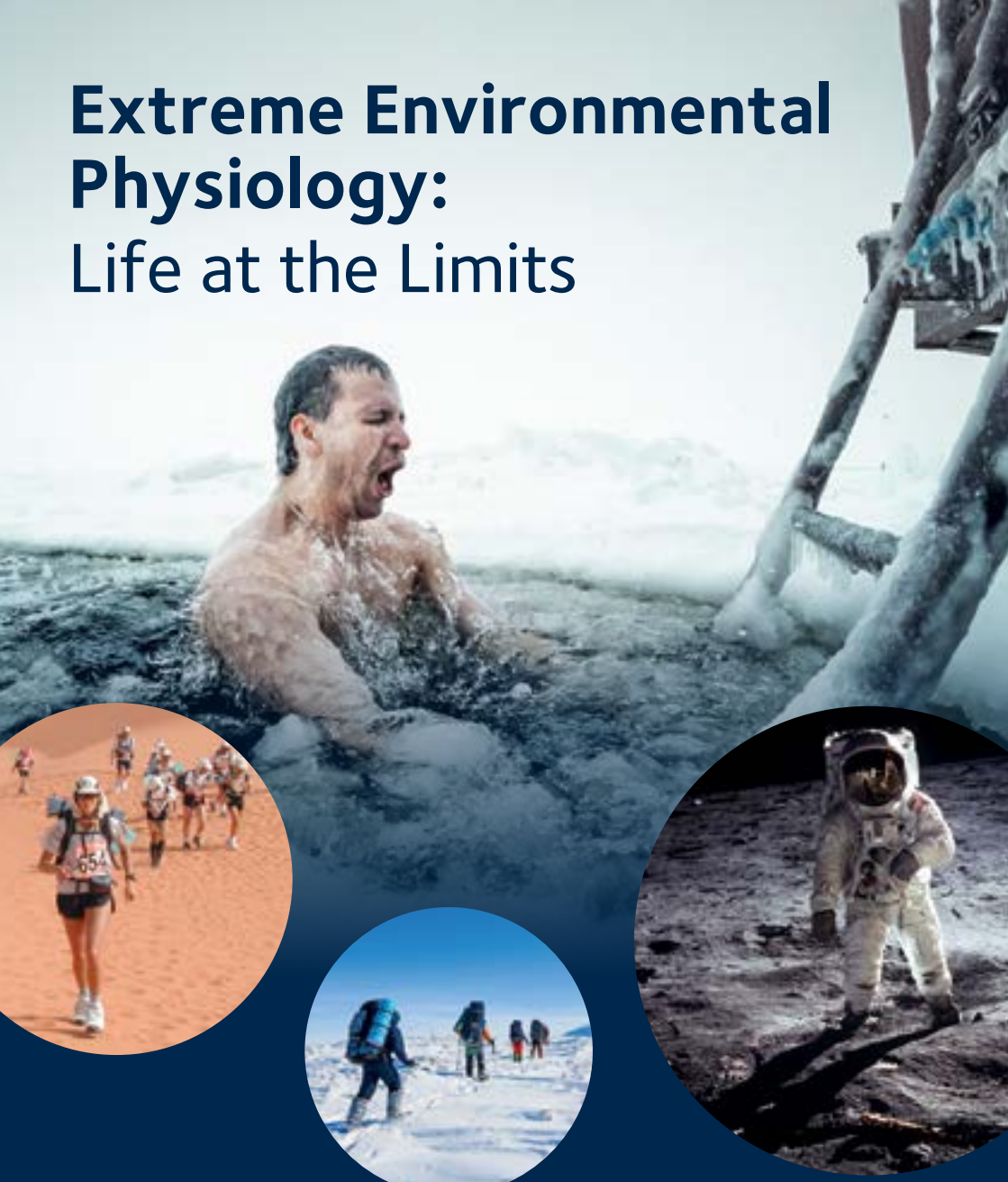
Extreme Environmental Physiology:
Life at the Limits



The Physiological Society

2-4 September 2019
Portland Building,
University of Portsmouth

Contents

The Physiological Society Topic Meeting
Extreme Environmental Physiology: Life at the Limits
2 – 4 September 2019

Portland Building, University of Portsmouth, Portland Street, Portsmouth, PO1 3AH

Organised by Mike Tipton, University of Portsmouth, UK

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Welcome

In The Physiological Society's year of Extreme Environmental Physiology it is my great pleasure to welcome you to Portsmouth University and The Society's specialist meeting on the topic.

It seems that wherever one looks extreme environmental physiology has a role to play; be that in informing the treatment of critically ill patients, as a curative intervention for a wide range of physical and mental conditions, as a major source of accidental death or injury, as a threat to elite athletes and occupational groups or as a major consequence of climate change. In this meeting we will examine a wide range of extreme environments and celebrate the wonderful breadth of physiology: we will travel from space to the ocean depths, from comparative physiology to psychophysiology to pathophysiology. I hope you will immerse yourself in the meeting and emerge wiser and enthused.

This meeting would not have been possible without the hard work and support of many individuals. I would like to thank the meeting sponsors, those who have agreed to come and give invited presentations, other national and international academics who have helped, colleagues at both the University of Portsmouth and The Physiological Society. In particular, I am grateful for the contributions of the scientific and sponsorship committee members at Portsmouth (Jo Corbett, Joe Costello, Clare Eglin, Heather Massey, Gemma Milligan, Zoe Saynor and Ant Shepherd) and, from The Physiological Society, Christine Carr and especially Rosie Hynard for her dedication, hard work and professionalism.

Mike Tipton, University of Portsmouth, UK

08:50 Welcome
Sue Deuchars, Chair, Conferences Committee, The Physiological Society, UK and Bob Nichol, Pro Vice-Chancellor (Research and Innovation), University of Portsmouth, UK

Session 1: Cold

In association with the Energy Institute

Chaired by Claire Eglin, University of Portsmouth, UK and Helen Hanstock, Mid Sweden University, Sweden

09:00 Physiology of aerobic and dexterity performance in the cold
SA01 John Castellani, US Army Research Institute of Environmental Medicine, US

09:30 Comparative effect of daylight restriction and sleep deprivation on the immune response of male Swiss mice
C01 Abayomi O. Ige, University of Ibadan, Nigeria

09:45 The pathophysiology of frostbite and other cold injuries
SA02 Chris Imray, University Hospitals Coventry and Warwickshire NHS Trust, UK

10:15 Beet the cold: Beetroot juice supplementation improves peripheral blood flow, endothelial function and anti-inflammatory status in individuals with Raynaud's phenomenon
C02 Anthony Shepherd, University of Portsmouth, UK

10:30 Break and networking

11:00 Challenges of thermoregulation in polar animals
SA03 Dominic McCafferty, University of Glasgow, UK

11:30 Winter energetics of Svalbard reindeer: Life on a tight budget
C03 Liv Monica Trondrud, Norwegian University of Life Sciences, Norway

Monday, 2 September

11:45 The seal, the bear & the super-ape: turning down the "master switch" & "fire of life" to a low, slow & cool "pilot light" – glimpses of phenomenal brain cooling at the o2-austere survival limit-of-life in a human outlier-extremophile
C04 Sebastian Murat, Bio-X Unit, Jungle Innovations, Australia

12:00 Lunch, exhibition and poster viewing

Session 2: Heat

13:25 Introduction by chairs
Jo Corbett, University of Portsmouth, UK and Polly Aylwin, Loughborough University, UK

13:30 Ultraviolet radiation exposure and human skin health
SA04 Larry Kenney, Penn State University, US

14:00 Role of bradykinin in human sweating during simulated and actual heat stress
C05 Thad E. Wilson, Marian University College of Osteopathic Medicine, US

14:15 Regional thermal hyperaemia - Evidence of a critical role of local thermosensitive mechanisms in the control of the human leg circulation during hyperthermia
C06 Nuno Koch Esteves, Brunel University London, UK

14:30 Impact of solar radiation on physical work capacity during heat stress in humans
C07 Josh Foster, Loughborough University, UK

14:45 Break and networking

15:15 Heat illness: Pathophysiology
SA05 Dan Roiz de Sa, Institute of Naval Medicine & University of Portsmouth, UK

15:45 Five days of dietary nitrate supplementation has no effect on exercise or thermoregulation in the heat
C08 Mark Waldron, Swansea University, UK

16:00 Exercise thresholds in hot environmental conditions: Is there a shift?
C09 Gil Bourgois, Ghent University, Belgium

16:15 Heat tolerance and evaporative cooling capacity in birds and small mammals
SA06 Andrew McKechnie, South African National Biodiversity Institute & University of Pretoria, South Africa

16:45 Encapsulation of carbohydrate within a pectin-alginate hydrogel does not improve blood glucose availability, whole body carbohydrate oxidation, or time trial performance during prolonged cycling in hot and humid conditions
C10 Stefano Montanari, University of Chichester, UK

17:00 The time course of adaptations to seven-weeks intermittent post-exercise sauna bathing for inducing heat acclimation in trained middle-distance runners
C11 Nathalie Kirby, University of Birmingham, UK

17:15 Poster session with refreshments

19:15 End of day one

Tuesday, 3 September

08:00 Registration, refreshments and networking

Session 3: Space

09:10 Introduction by chairs
Kevin Fong, University College London Hospitals, UK and Nejka Potocnik, University of Ljubljana, Slovenia

09:15 Physiology
Igor Mekjavic, Jožef Stefan Institute, Slovenia

09:45 Daily generation of hydrostatic gradients attenuates ocular changes associated with head-down tilt bedrest
C14 Justin Lawley, University of Innsbruck, Austria

10:00 Human research in one of the most extreme environments: Space
SA07 Thu Jennifer Ngo-Anh, European Space Agency, France

10:30 Break and networking

11:00 Pathophysiology
Kevin Fong, University College London Hospitals, UK

11:30 Compared to magnetic resonance imaging, the creatine (methyl-d3) method overestimates the loss of total skeletal muscle mass following 7 days of whole-body unloading
C15 Stephen Harridge, King's College London, UK

11:45 The influence of +Gx accelerations of relevance to suborbital spaceflight on the lung and pulmonary mechanics
C15 Ross Pollock, King's College London, UK

12:00 Lunch, exhibition and poster viewing

12:30 **Seminar:** Understanding the role of extreme physiology to improve human performance and health
Led by Mike Tipton, University of Portsmouth, UK

Session 4: Altitude

13:25 Introduction by chairs
Hugh Montgomery, University College London, UK and Lydia Simpson, Bangor University, UK

13:30 The brain at high-altitude; a radical perspective!
SA08 Damian Bailey, University of South Wales, UK

14:00 Baroreflex function in Andean high altitude natives with and without chronic mountain sickness
C15 Lydia Simpson, Bangor University, UK

14:15 Impact of oxygen supplementation on flow-mediated dilation, cerebral blood flow, and oxygen delivery during ascent to 5050m
C16 Joshua Tremblay, Queen's University, Canada

14:30 Global Reach 2018: High altitude acclimatisation improves neurovascular coupling in man
C17 Benjamin Stacey, University of South Wales, UK

14:45 Cardiorespiratory hysteresis during incremental high-altitude ascent-descent quantifies the magnitude of ventilatory acclimatization in healthy participants
C18 Jack Leacy, University College Cork, Ireland

15:00 Break and networking

15:30 The unique cardiorespiratory physiology of birds and why they excel at altitude
Lucy Hawkes, University of Exeter, UK

16:00 Hypoxia - The Good, The Bad & The Ugly?
SA09 Sundeep Dhillon, CASE Medicine, University College London, UK

16:30 Changes in cerebral oxygenation and microvascular blood volume during exercise in hypoxia and possible association with acute mountain sickness
C19 Giorgio Manferdelli, National Research Council, Italy

Tuesday, 3 September

16:45 Non-invasive assessment of pulmonary gas exchange efficiency in humans: Influence of altitude, exercise and chronic mountain sickness
C20 Connor Howe, University of British Columbia Okanagan, Canada

17:00 The effect of severe and moderate hypoxia on exercise at a fixed level of perceived exertion
C21 Owen Jeffries, Newcastle University, UK

17:15 Break and networking

17:30 Public lecture
Physiology and the extremes of humanity: Challenges for martian explorers
SA10 James Pawelczyk, Penn State University, US

18:30 End of day two

18:45 Coaches to Society Dinner

19:00 Society Dinner

Mary Rose Museum and Boathouse 4, Main Rd, Portsmouth PO1 3PY

This unique and special Society Dinner will consist of an arrival drink at the Mary Rose Museum with a talk given by museum staff.

You will then have exclusive access to the museum with staff on hand to answer questions.

This will be followed by a three-course meal, wine and soft drinks at nearby Boathouse 4.

If you don't already have a ticket, then a handful will be on sale during the conference.

08:00 Registration, refreshments and networking

Session 5: Hyperbaric and Immersion

08:55 Introduction by chairs
Igor Mekjavic, Jožef Stefan Institute, Slovenia and Heather Massey, University of Portsmouth, UK

09:00 Immersion in cold water: Sudden death and prolonged survival
SA11 Mike Tipton, University of Portsmouth, UK

09:30 Cold water face immersion in healthy subjects: How a clash of autonomic pathways might contribute to triathlon deaths
C22 Francesca McLean, University of Plymouth, UK

09:45 The effect of various breath-hold techniques on the cardiorespiratory response to facial immersion
C23 Matthew Burley, Royal Marines Commando, UK

10:00 Swimming the English Channel solo: A case study
C24 Heather Massey, University of Portsmouth, UK

10:15 Break and networking

10:45 Diving and hyperbaric physiology - what have we learned?
SA12 James Clark, King's College London, UK

11:15 High-pressure nervous syndrome and divers' wellbeing evaluation in operational setting
C26 Simin Berenji, Aarhus University, Denmark

11:30 Cardiorespiratory hypotheses how deep diving cetaceans avoid the bends
SA13 Andreas Fahlman, Oceanografic, Spain

12:00 The Sacrificial Effect: Paradoxical seal-like human empty lungs deep diving
C25 Sebastian Murat, Bio-X Unit, Jungle Innovations, Australia

Wednesday, 4 September

12:15 Lunch, exhibition and poster viewing

12:45 **Seminar:** Science in the Media
Chris Van Tulleken, University College London Hospitals, UK

Session 6: Cross adaptation and Global Warming

13:40 Introductions by chair
Ben Lee, University of Chichester, UK and Rachel Gifford, University of Birmingham, UK

13:45 A primer to cross-adaptation and cross-tolerance to novel stressors
SA14 Jim Cotter, University of Otago, New Zealand

14:15 Cross adaptation for attenuating environmental strain, and improving exercise and health physiology
SA15 Oliver Gibson, Brunel University London, UK

14:45 The impact of trait and state anxiety on physical performance in heat and hypoxia
C27 Kate O'Keeffe, Loughborough University, UK

15:00 Short-term heat acclimation enhances cold and heat endurance performance: A case study
C28 Franck Brocherie, French Institute of Sport, France

15:15 Cardiopulmonary acclimation using intermittent normobaric hypoxic exposure with and without exercise
C38 Mark Cooke, Leeds Beckett University, UK

15:30 Humans and the environment: Flipping the coin
SA16 Hugh Montgomery, University College London, UK

16:00 Prizes and thanks

16:15 End of conference

C01 Comparative effect of daylight restriction and sleep deprivation on the immune response of male Swiss mice

Abayomi O. Ige1, Deborah C. Uzuegbu1, Precious O. Adebayo1, Idara E. Emediong1, Anthony O. Odetola1, 2, Bernard Adele1, Elsie O. Adewoye1
1Department of Physiology, University of Ibadan, Ibadan, Oyo, Nigeria, 2Department of Human Physiology, Nnamdi Azikiwe University, Akwa, Anambra, Nigeria

C02 Beet the cold: Beetroot juice supplementation improves peripheral blood flow, endothelial function and anti-inflammatory status in individuals with Raynaud's phenomenon

Anthony Shepherd1, Joseph Costello1, Stephen Bailey3, Nicolette Bishop3, 5, Alex Wadley3, 5, Steven Young-Min2, Mark Gilchrist4, Harry Mayes1, Danny White1, Paul Gorczynski1, Zoe Saynor1, Heather Massey1, Clare Eglin1
1DSES, University of Portsmouth, Portsmouth, United Kingdom, 2Rheumatology Department, Portsmouth Hospitals NHS Trust, Portsmouth, Hampshire, United Kingdom, 3National Centre for Sport and Exercise Medicine, School of Sport, Exercise and Health Sciences, Loughborough University, Loughborough, United Kingdom, 4University of Exeter Medical School and NIHR Exeter Clinical Research Facility, University of Exeter, Exeter, Devon, United Kingdom, 5University Hospitals of Leicester NHS Trust, Infirmary Square, Leicester, United Kingdom

C05 Role of bradykinin in human sweating during simulated and actual heat stress

Thad E. Wilson1, Seetharam Narra1, Kristen Metzler-Wilson2, 3, 4, Artur Schneider1
1Division of Biomedical Sciences, Marian University College of Osteopathic Medicine, Indianapolis, Indiana, United States, 2Department of Physical Therapy, Indiana University, Indianapolis, Indiana, United States, 3Department of Anatomy, Cell Biology, & Physiology, Indiana University, Indianapolis, Indiana, United States, 4Department of Dermatology, Indiana University , Indianapolis, Indiana, United States

C09 Exercise thresholds in hot environmental conditions: is there a shift?

Gil Bourgois, Jan G. Bourgois, Jan Boone
Department of Movement & Sports Sciences, Ghent University, Ghent, Belgium

C10 Encapsulation of carbohydrate within a pectin-alginate hydrogel does not improve blood glucose availability, whole body carbohydrate oxidation, or time trial performance during prolonged cycling in hot and humid conditions.

Stefano Montanari1, Tessa Flood1, Holly Sharp1, Marley Wicks1, Jack Blanchard1, Matthew Kuennen2, Lee Taylor3, Ben J. Lee1

1Institute of Sport, University of Chichester, Chichester, West Sussex , United Kingdom, 2Exercise Science, High Point University, High Point, North Carolina, United States, 3Sport and Exercise Science, Loughborough University, Loughborough, United Kingdom

C15 Baroreflex function in Andean high altitude natives with and without chronic mountain sickness

Lydia L. Simpson1, Victoria L. Meah2, Andrew Steele2, Stephen A. Busch1, Sam Oliver1, Justin S. Lawley3, Michael Tymko4, Gustavo Vizcardo-Galindo5, Rómulo J. Figueroa-Mujíca5, Francisco Villafuerte5, Philip N. Ainslie4, Craig D. Steinback2, Mike Stembridge6, Jonathan P. Moore1

1School of Sport, Health and Exercise Sciences, Bangor University, Walsall, United Kingdom, 2Neurovascular Health Laboratory, Faculty of Kinesiology, Sport, and Recreation, University of Alberta, Edmonton, Alberta, Canada, 3Department of Sport Science, Division of Physiology, University of Innsbruck, Innsbruck, Austria, 4Centre for Heart, Lung, and Vascular Health, University of British Columbia Okanagan, Kelowna, British Columbia, Canada, 5Laboratorio de Fisiología Comparada, Departamento de Ciencias Biológicas y Fisiológicas, Facultad de Ciencias y Filosofía, Universidad Peruana Cayetano Heredia, Lima, Peru, 6Cardiff School of Sport and Health Sciences, Cardiff Metropolitan University, Cardiff, United Kingdom

C17 Global Reach 2018: High altitude acclimatisation improves neurovascular coupling in man

Benjamin S. Stacey3, Ryan L. Hoiland1, Hannah Caldwell1, Connor A. Howe1, Tyler Vermeulen1, Michael Tymko1, Gustavo Vizcardo-Galindo2, Daniela Bermudez2, Francisco Villafuerte2, Philip N. Ainslie1, Damian Bailey3

1Center for Heart, Lung and Vascular Health, University of British Columbia - Okanagan, Kelowna, British Columbia, Canada, 2Laboratorio de Fisiología Comparada, Departamento de Ciencias Biológicas y Fisiológicas, Facultad de Ciencias y Filosofía, Universidad Peruana Cayetano Heredia, Lima, Peru, 3Neurovascular Research Laboratory, Faculty of Life Sciences and Education, University of South Wales, Pontypridd, United Kingdom

C18 Cardiorespiratory hysteresis during incremental high-altitude ascent-descent quantifies the magnitude of ventilatory acclimatization in healthy participants

Jack K. Leacy1, Andrea M. Linares2, Shaelynn M. Zouboules2, Zahrah Rampuri2, Brittney Herrington2, Leah Mann2, Jan-Elaine Soriano2, Scott Thrall2, Jordan Bird2, Anne Kalker2, 3, Tom Brutsaert4, Ken D. O'Halloran1, Trevor A. Day2

1Department of Physiology, University College Cork, Cork, Ireland, 2Department of Biology, Mount Royal University, Calgary, Alberta, Canada, 3Radboud University, Nijmegen, Netherlands, 4School of Education, Syracuse University, New York, New York, United States

C19 Changes in cerebral oxygenation and microvascular blood volume during exercise in hypoxia and possible association with acute mountain sickness

Giorgio Manferdelli1, 2, Mauro Marzorati1, Chris Easton2, Simone Porcelli1

1National Research Council, Milan, United Kingdom, 2University of the West of Scotland, Hamilton, United Kingdom

C22 Cold water face immersion in healthy subjects: how a clash of autonomic pathways might contribute to triathlon deaths.

Mirza M. Subhan, Francesca McLean, Alfie Baldwin, Michael Pipis, Murad Rahman, Mahnur Siddiqui

University of Plymouth, Plymouth, United Kingdom

Poster Communications

C23 The effect of various breath-hold techniques on the cardiorespiratory response to facial immersion

Francis B. Stephens1, Matthew J. Burley2, 1, Bert Bond1, Craig A. Williams1
1Sport and Exercise Science, University of Exeter, Exeter, Devon, United Kingdom, 2Physical Training Department, Commando Training Centre Royal Marines (CTCRM), Exmouth, Devon, United Kingdom

C26 High-Pressure Nervous Syndrome and divers' wellbeing evaluation in operational setting

Simin Berenji Ardestani1, Costantino Balestra2, 5, Elena V Bouzinova3, Øyvind Loennechen4, Michael Pedersen1
1Clinical medicine, Aarhus University, Aarhus, Denmark, 2Environmental, Occupational, Ageing (Integrative) Physiology Laboratory, Haute Ecole Bruxelles-Brabant (HE2B), Brussels, Belgium, 3Institute for Biomedicine, Aarhus University, Aarhus, Denmark, 4TechnipFMC, Stavanger, Norway, 5Divers Alert Network Europe - Research Division, Roseto, Italy

C27 The impact of trait and state anxiety on physical performance in heat and hypoxia

Kate O'Keeffe, Simon Hodder, Alex Lloyd
Loughborough University , Loughborough , United Kingdom

C29 Does 5-days heat acclimation reduce cardiovascular drift and improve VO2max performance in hot and cool conditions?

Rachel Gifford, Sam Lucas, Rebekah Lucas
Sport science, University of Birmingham , Solihull, West Midlands, United Kingdom

C30 Psycho-physiological responses to perceptually-regulated hypoxic and normoxic interval walking in obese individuals

Liam Hobbins1, Olivier Girard2, Nadia Gaoua1, Steve Hunter1
1Sport and Exercise Science Research Centre (SESRC), London South Bank University, London, United Kingdom, 2Murdoch Applied Sport Science (MASS) Lab, Murdoch University, Perth, Western Australia, Australia

C31 Human cerebral blood flow-metabolic uncoupling during acute hypoxia: A spectroscopy study

Matthew Rogan, Alexander Friend, Gabriella Rossetti, Jamie Macdonald, Sam Oliver, Paul Mullins
Bangor University , Y Felinheli, Gwynedd, United Kingdom

C32 Effects of a heated garment on physiological responses to simulated hill walking in the cold in man

David A. Low, Ross Williams, Graeme L. Close
RISES, Liverpool John Moores University, Liverpool, United Kingdom

C33 Extremes of convection: Regulating thermal profile during downhill cycling using newspaper as a thermal insulator

Martin J. Barwood, Harry Beal
Sport, Health and Nutrition, Leeds Trinity University, Leeds, Horsforth, United Kingdom

C34 Vascular function in non-freezing cold injury patients

Matthew Maley1, Clare Eglin1, Jennifer Wright1, Sarah Hollis2, Michael Tipton1
1Sport and Exercise Science, University of Portsmouth, Portsmouth, United Kingdom, 2Regional Occupational Health Team, Catterick Garrison, United Kingdom

C35 How are you sleeping in Antarctica? One-year smartphone-based sleep monitoring pilot study at "Akademik Vernadsky" Research Base.

Oleksandr V. Shylo1, Dmytro G. Lutsenko1, Kostiantyn M. Danilenko2, Georgy O. Babiychuk1, Yevgen Moiseyenko2
1Cryophysiology, Institute for Problems of Cryobiology and Cryomedicine of the NAS of Ukraine, Kharkiv, Ukraine, Ukraine, 2National Antarctic Scientific Center, Kyiv, Ukraine

C37 Long- and short-term cold adaptations affect erythrocyte population in rats of different ages.

Oleksandr V. Shylo, Dmytro G. Lutsenko, Victoria Lomako
Cryophysiology, Institute for Problems of Cryobiology and Cryomedicine of the NAS of Ukraine, Kharkiv, Ukraine, Ukraine

Poster Communications

C38 Cardiopulmonary acclimation using intermittent normobaric hypoxic exposure with and without exercise.
Mark Cooke1, Christopher J. Boos1, 2, David Holdsworth3, Ade Mellor1, 4, 5, Rachael Bradley1, John O'Hara1, David Woods1, 4, 6
1Leeds Beckett University, Leeds, United Kingdom, 2Department of Cardiology, Poole Hospital NHS Foundation Trust, Poole, United Kingdom, 3Department of physiology, Anatomy and Genetics, Univeristy of Oxford, Oxford, United Kingdom, 4Royal Centre for Defence Medicine, Birmingham, United Kingdom, 5James Cook University Hospital, Middlesbrough, United Kingdom, 6Northumbria NHS Trust and Newcastle NHS Trust, Northumbria, United Kingdom

C41 Management of Heat Stress in Sport; Are Recommendations Suitable?
Polly Aylwin1, George Havenith1, Ollie Jay2, Timothy English2, Glenda Anderson2, Yorgi Mavros3, Coen Bongers2, Simon Hodder1
1Environmental Ergonomics Research Centre, Loughborough Design School, Loughborough University, Loughborough, United Kingdom, 2Thermal Ergonomics Laboratory, Faculty of Health Sciences, University of Sydney, Sydney, New South Wales, Australia, 3Physical Activity, Lifestyle, Ageing and Wellbeing Group, Faculty of Health Sciences, University of Sydney, Sydney, New South Wales, Australia

C42 Comparisons of core body temperature between an ingested telemetric pill and heart rate estimated core body temperature in firefighters
Stephen Pearson, Martyn Matthews
Health & Society, University of Salford, Manchester, United Kingdom

C43 Minimal effect of water immersion on markers of inflammation and muscle damage after intensive exercise.
Essi K. Ahokas2, Heikki Kyrolainen2, Antti A. Mero2, Simon Walker2, Helen G. Hanstock1, Johanna K. Ihalainen1, 2
1Swedish Winter Sports Research Centre, Mittuniversitetet, Östersund, Jämtland, Sweden, 2Unit of Biology of Physical Activity, University of Jyväskyla, Jyväskylä, Finland

C44 Rewarming methods following cold water swimming
Heather Massey, Michael Tipton
Department of Sport and Exercise Science, University of Portsmouth, Portsmouth, United Kingdom

C45 Follow-up of pulmonary diffusion capacity in elite swimmers before and after training sessions under indoor swimming pool conditions

Iker Garcia1, 2, Franchek Drobnic2, Gines Viscor1
1Department of Physiology and Immunology, University of Barcelona, Sant Cugat del Valles, Barcelona, Spain, 2Department of Physiology and Nutrition, Centre d'Alt Rendiment Esportiu de Sant Cugat, Sant Cugat, Barcelona, Spain

C46 Lung diffusion changes during altitude training (1.850m) in elite swimmers

Iker Garcia1, 2, Franchek Drobnic2, Teresa Valera1, Gines Viscor1
1Department of Physiology and Immunology, University of Barcelona, Sant Cugat del Valles, Barcelona, Spain, 2Department of Physiology and Nutrition, Centre d'Alt Rendiment Esportiu de Sant Cugat, Sant Cugat del Valles, Barcelona, Spain

C47 The effect of anti-gravity socks on skin microcirculation in foot

Nejka Potocnik1, Kaja Stankovic2, Polona Potocnik3
1Institute of Physiology, University of Ljubljana, Medical faculty, Ljubljana, Slovenia, 2University of Ljubljana, Faculty for health, Ljubljana, Slovenia, 3University of Ljubljana, Medical faculty, Ljubljana, Slovenia

C48 Protective effect of baker's yeast on Carbon Tetrachloride Induced Hepatotoxicity in rats

Aisha Alfituri1, Isam Busnaina1, Ahmed Bahriz2, Abobaker Bashir1
1University of Benghazi, Benghazi, Libya, 2Faculty Of Medicine, Benha University, Banha, Egypt

C49 Salivary Cytokines in Yacht Racing Athletes

Irina Shvydchenko, Anna Dubova
Department of Physiology, Kuban State University of Physical Education, Sport and Tourism, Krasnodar, Russian Federation

C50 Protective effect of co-administration of vitamins C and E on reserpine-induced motor and cognitive impairments and oxidative stress in mice

Timothy Danboyi1, Abdulwahab Alhassan2, Jimoh Abdulazeez2, Evelyn Hassan-Danboyi2
1Kaduna State University, Kaduna, Nigeria, 2Human Physiology, Ahmadu Bello University, Zaria, Zaria, Nigeria

Poster Communications

C51 Heart Rate variability as a predictive tool in the Military

Mafalda Carvalho1, Lucena Rui2
1Cardiovascular Autonomic Function Lab, Lisboa, N/A, Portugal, 2CINAMIL, Lisbon, Portugal

C52 The effect of exercise mode on exercise induced gastrointestinal damage during exercise performed at a fixed rate of metabolic heat production.

Tessa Flood1, Ella F. Walker2, 1, Sam Blacker1, Stephen Myers1, Holly Sharp3, Stefano Montanari3, Stephen McGuire1, Ben J. Lee1
1Occupational Performance Research Group, Institute of Sport, University of Chichester, Chichester, United Kingdom, 2Defence Science and Technology Laboratory, Salisbury, United Kingdom, 3Institute of Sport , University of Chichester, Chichester, United Kingdom

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The Physiological Society

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General information

The Physiological Society team can always be found at the registration desk. We will be happy to help with any queries you may have but you might be able to find an answer to your question on these pages.

Registration

The registration desk is in the Portland Building on the ground floor and will be open at the following times:

Monday, 2 September	08:00 – 19:15
Tuesday, 3 September	08:00 – 18:30
Wednesday, 4 September	08:00 – 13:40

Poster help desk

This is at the registration desk. You can find Velcro to affix your poster here. No other fixings may be used. You can also find out when you are scheduled to present.

Poster session on Monday, 2 September

Odd numbers will present from 17:15 – 18:15
Even numbers will present from 18:15 – 19:15

Internet access

There is free Wi-Fi, please ask at registration desk.

Mobile phones

Don't forget to turn off your mobile during all sessions at the meeting.

Video or audio recording of presentations

Attendees are reminded that the video and audio recording of ANY session or presentation using mobile devices or any such recording equipment is strictly prohibited.

Changes to abstracts

We cannot make changes to abstracts.

Smoking

It is illegal to smoke in enclosed public spaces. If you wish to smoke then you need to go to the designated smoking area.

Arriving late to sessions
We know it is not always possible to get to sessions on time but we do ask that if you are running late, to take your seat quietly.

Food and drink
All refreshments are included in your registration at times detailed in the programme.

Certificate of attendance
If you need a certificate of attendance and haven't already requested one, please ask at the registration desk.

Twitter
The official Twitter hashtag is **#ExtremePhys19**. Make sure you are following @ThePhySoc to keep up to date on what's happening at the meeting.

Feedback
We want you to enjoy this meeting and are always happy to hear how we can improve. You can speak to a member of the team, or fill in your comments anonymously in the feedback questionnaire that will be emailed to you after the meeting.

In case of emergency
The Physiological Society team are your first point of contact in any emergency. They will help you or find the person who can.

The Energy Institute (EI) is the chartered professional membership body bringing global energy expertise together.

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Our equipment has travelled to the Everest Base Camp and back with Science Hero, Trevor Day and his team, along with some of our staff! The team used a range of our products, such as the PowerLab data acquisition system to measure a myriad of physiological functions responding to the sudden drop in oxygen saturation in the air! Since the trip we have evolved, adapted and come a long way with some exciting developments and new product ranges, which are ideal for your physiology research in a vast range of different environments, be it the laboratory, a mountain top or the middle of the desert!

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easy setup means you can get started quickly and confidently and data integrity is guaranteed.

The Non-invasive Blood Pressure recording for Human Autonomic research gives you the ability to capture continuous blood pressure data over long sampling periods easily, with increased comfort for your subject. The system automatically corrects for movement of the finger cuff relative to the heart with the height correction unit (HCU). With our Human NIBP Nano system you can stream data directly into LabChart for easy analysis, as well as simultaneously acquire additional physiological signals by adding a PowerLab data acquisition system.

Linton Instrumentation is an independently owned company specialising in the supply, maintenance and support of laboratory instrumentation for the life sciences. We distribute innovative products and solutions from market leading manufacturers including BodyCap, Biopac Systems Inc., CWE, Philips Respironics (Actigraphy Products), Physitemp, Primetech (iPRECIO implantable drug delivery systems), and Ugo Basile, as well as our own branded products. We also have flexibility to source products from other manufacturers in order to offer our clients a complete and cost effective solution.

BodyCap is a French company developing miniature wireless electronic sensors and solutions for physiological data monitoring. Its innovative high-tech products are used in medical, research, sport & ergonomics applications. The unique features of the e-Celsius® Performance core temperature monitoring system enable researchers to monitor core body temperature in extreme environmental situations. The e-Celsius® Performance solution is based on ingestible electronic capsules which transmit data to a portable monitor and also store data internally for download after the event, thereby enabling the subject to enter any environment without the need to carry additional equipment. Animal core temperature monitoring solutions are also available.

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First-hand experience of laser Doppler research and development within Moor dates back to 1978 and with this we have the breadth of knowledge to help with your application and the enthusiasm to try and find answers to any of your questions.

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Ethical requirements

Experiments on animals or animal tissue

For work conducted in the UK all procedures must conform with current UK legislation. For work conducted elsewhere all procedures must accord with current national guidelines or, in their absence, with current local guidelines.

Experiments on humans or human tissue

All procedures must accord with the ethical standards of the relevant national, institutional or other body responsible for human research and experimentation, and with the principles of the World Medical Association's Declaration of Helsinki.

SA01

Physiology of Aerobic and Dexterity Performance in the Cold

J. Castellani

US Army Research Institute of Environmental Medicine, Natick, MA, USA

Cold exposure impacts aerobic and dexterity performance in humans. The impact of cold exposure on aerobic performance has not been thoroughly studied. The few studies that have been done suggest that aerobic performance is degraded in cold environments. Potential physiological mechanisms (e.g., decreases in deep body and muscle temperature, cardiovascular, metabolism) will be discussed. Dexterity performance is well known to decline during cold exposure and many list the loss of manual dexterity as the number one performance problem in the cold. Manual dexterity is severely degraded at skin temperatures below 15°C. Mechanisms for the decrease in dexterity include reduced hand and finger blood flow, joint mobility, muscle temperatures, and nerve conduction velocity. Potential countermeasures to improve manual dexterity during cold exposure will be discussed.

Where applicable, the authors confirm that the experiments described here conform with the Physiological Society ethical requirements.

SA02

The pathophysiology of frostbite and other cold injuries

C. Imray

University Hospitals Coventry and Warwickshire (UHCW) NHS Trust, Coventry, UK

Frostbite is a cold thermal injury which usually affects the extremities and has the potential of causing irreversible and potentially life changing tissue loss. The understanding and treatment of freezing cold injuries to the periphery has advanced substantially in the last 10 years and optimal outcomes are only likely to be achieved if a multi-disciplinary team uses the full range of diagnostic and treatment modalities that are now available. The internet and satellite phones with digital images allow immediate access by patients from remote geographical locations to hospital based specialists who can assess cold injuries and advise on early field care. The severity of frostbite injuries can now be assessed with triple phase bone scanning, allowing early prediction of likely subsequent tissue loss. Newer thrombolytic therapies have transformed treatment options when instigated at an early time point.

Non-freezing cold injuries were historically associated with military working in the field, but more recently it has been recognised the number of civilian cases has increased. Sustained exposure to cold, wet conditions often associated with immobility appear to be key risk factors. It remains an avoidable major source of longer term often neurological morbidity. The exact pathophysiology of non-freezing cold injuries remains poorly understood, but there have been some significant advances in our understanding in certain areas recently.

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SA03

Challenges of thermoregulation in polar animals

D. McCafferty

Institute of Biodiversity, Animal Health & Comparative Medicine, University of Glasgow, Glasgow, UK

Polar birds and mammals maintain their body temperature despite the extreme challenges of a cold climate. To survive, these animals have evolved a number of anatomical, physiological and behavioural adaptations to cope with low temperature and high rates of heat loss. This talk aims to provide a comparative perspective on thermoregulation in homeothermic endotherms and to highlight how thermal imaging can reveal fascinating insights into how animals cope with the challenges of thermoregulation in the cold. We examine recent studies of the thermal biology of a range of species, including penguins and seals that also demonstrate the importance of behavioural thermoregulation in allowing species to thrive in polar environments.

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SA04

Ultraviolet radiation exposure and human skin health

L. Kenney[1], L. Kenney[1,2] and S. Wolf[1]

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The UVR spectrum is categorized by wavelength as UV-A (320-400 nm), UV-B (290-320 nm), and UV-C (200-290 nm) and the biological effects of UVR vary by wavelength. UV-A constitutes ~95% of UVR that reaches the earth's surface, with the remainder UV-B. In the skin, UV-A is able to penetrate the dermis and reach the cutaneous circulation but most UV-B is absorbed in the epidermis and upper dermis due to its shorter wavelengths. Overexposure to UVR is associated with multiple health risks including DNA damage, immune suppression, premature skin aging, skin vascular dysfunction, and skin cancers. While regular physical activity is associated with a *reduced* risk of *most* cancers, skin cancer is an exception. For malignant melanoma, those in the 90th %ile for physical activity have *increased* hazard ratios compared to those in the 10th %ile. Those who participate in regular outdoor exercise or sport experience high daily doses of UVR exposure. According to ICNIRP guidelines, maximum biologically effective UVR exposure over an 8-h period should be limited to 30 J.m^{-2}, a dose equivalent to 1.0-1.3 SED for fair skin. High daily UVR exposures have been reported for hikers (2-20 SED), tennis players (2-14 SED), and runners (9-24 SED) during the summer and autumn months, exceeding the recommended UVR exposure limit by up to *8-fold*. Multiple studies demonstrate an elevated risk of skin cancer for those who regularly participate in outdoor sports or exercise, highlighting the importance of sun-protection strategies in those populations. Yet fewer than 25% of surveyed athletes report regular use of sunscreen.

Exposure to UVR not only reddens the skin but also impairs underlying vascular function. The ability of skin arterioles to dilate via nitric oxide- (NO-) is a marker of vascular health. Adequate 5-methyltetrahydrofolate (5-MTHF; the bioactive

metabolite of folate) is essential for full expression of NO-mediated vasodilatation through its role in enzymatic coupling of NO synthase. 5-MTHF is degraded by UVR *in vitro* and skin UVR exposure depletes bioavailable 5-MTHF in the exposed area, mediated by both direct photodegradation of 5-MTHF and indirectly via the production of reactive oxygen species (ROS). Acute UVR exposure may impair NO-mediated vasodilatation through either or both mechanisms. We recently demonstrated that broad spectrum UVR attenuated NO-mediated cutaneous vasodilatation, a response that was prevented by both SPF-50 sunscreen and by simulated sweat on the skin during exposure.

The *vitamin D-folate hypothesis* has been proposed to explain the evolution of human skin pigmentation. According to this hypothesis, darkened skin pigment was adapted by early human populations living in equatorial Africa to protect against photodegradation of bioavailable folate by UVR. As humans moved away from the equator to more northern latitudes and occupied regions of lower UVR exposure and greater seasonal variation, depigmentation occurred to allow for adequate biosynthesis of vitamin D. Vitamin D and folate are both recognized for their evolutionary importance in healthy pregnancy and early childhood development. Populations with darkened skin pigmentation may be at elevated risk of vascular dysfunction and cardiovascular disease in low UVR environments due to hypovitaminosis D. Conversely, lightly-pigmented populations in high UVR environments may be at risk of deleterious vascular effects of UVR-induced folate degradation. Recent evidence has emerged demonstrating the importance of both vitamin D and folate in vascular health via their effects in reducing cutaneous oxidative stress and improving NO bioavailability. The skin's ability to produce vitamin D is negatively affected by age, darker skin pigmentation, and several gene variants. Alternately, darker skin pigmentation plays a protective role against photodegradation of 5-MTHF; variations in the 5-MTHF response to UVR exposure may also be explained, at least in part, by genetic variation.

In summary, exposure to UVR is associated with both beneficial and deleterious effects on cutaneous vascular health. Both folate and vitamin D play important roles in healthy vascular function, but UVR exposure elicits opposing effects on metabolism and bioavailability of these two compounds. The effects of UVR on folate and vitamin D metabolism appears to be influenced by multiple factors, including skin pigmentation, genetics, geographical location, and age. Beyond the influence of UVR on folate and vitamin D metabolism, UVR exposure may cause oxidative stress and inflammatory responses that impair vascular health in a dose-dependent fashion. The interactions between individual characteristics and environment in modulating vitamin D and folate bioavailability and vascular health are highly complex.

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SA05

Heat illness: Pathophysiology

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The pathophysiology of heat illness and its aetiology is poorly understood and multifactorial in its presentation. Many patients appear to recover very quickly and whilst some do well others have significant morbidity. Clinicians can fail to appreciate the potential impact of this for our patients. With features of heat illness common to other conditions such as sepsis and rhabdomyolysis, this presentation will explore the condition and avenues for future research.

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SA06

Heat tolerance and evaporative cooling in birds and small mammals

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Well-hydrated humans can dissipate heat by evaporation far more rapidly than most species and the heat tolerances and evaporative cooling capacities of small endotherms are modest by comparison. Moreover, many birds and small mammals inhabit arid environments with scarce and unpredictable water resources, creating trade-offs between hyperthermia tolerance and dehydration avoidance. The potential for extreme heat events to push small endotherms beyond their physiological tolerance limits is dramatically illustrated by catastrophic mortality events involving birds and bats, the frequency of which is increasing as climate change advances. In this presentation, I review thermoregulation in the heat among birds and small mammals, and link physiology to behavioural and ecological factors that determine sensitivity to very hot conditions.

During heat exposure, small endotherms employ facultative, reversible hyperthermia. The maximum body temperatures tolerated vary widely among mammals, from 38-39 °C in marsupials and fruit bats up to ~45 °C in some rodents and small insectivorous bats. Avian maximum body temperatures are typically in the 44-46 °C range, but may be as high as ~47 °C. There is increasing evidence from studies of free-ranging populations that small endotherms often maintain very small thermal safety margins, regularly allowing body temperature to approach lethal limits during hot conditions. The maximum air temperatures small endotherms can tolerate during brief heat exposure also vary widely, from 32 °C in temperate-latitude shrews to above 60 °C in arid-zone doves and nightjars. During thermoregulation in the heat, evaporative water losses may occur via several pathways other than sweating. Panting is the primary avenue of heat dissipation for most birds and many mammals, usually accompanied by increases in resting metabolic rate. Among avian taxa in which panting is the primary

avenue of evaporative cooling, maximum ratios of evaporative heat loss (EHL) to metabolic heat production (MHP) are typically 2.0 – 2.5. Some taxa, most notably columbid birds, can dissipate substantial heat loads cutaneously, with rates of cutaneous evaporation being determined by microcirculatory adjustments over short time scales and phenotypic flexibility in *stratum corneum* lipid composition over longer time scales. Among species in which cutaneous evaporation predominates, maximum EHL/MHP values vary between 3.0 and 4.7. Another highly efficient avenue of heat dissipation found in many birds is gular flutter, which can provide the basis for EHL/MHP above 5.0. Many mammals, including marsupials, bats and rodents, spread copious amounts of saliva over their fur to enhance evaporative cooling during heat exposure. A non-evaporative avenue of heat loss that is emerging as important in birds is the beak, with the shunting of blood to the beak vasculature when air temperature is below body temperature providing the basis for rapid heat dissipation in large-beaked species such as toucans and hornbills.
Many small endotherms operate close to their physiological limits in hot environments. The increasing temperatures and more frequent heat waves associated with rapid anthropogenic climate change are predicted to cause severe declines among species inhabiting hot regions. These declines will be driven both by acute, lethal effects of extreme heat events and sublethal fitness costs associated with chronic exposure to sustained hot weather. Among desert birds, many of these sublethal fitness costs arise from trade-offs between foraging and thermoregulatory behaviours such as panting and shade-seeking; consequences include progressive loss of body condition, reduced provisioning rates to nests, lower chick growth rates and more frequent breeding failure.

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SA07

Human research in one of the most extreme environments: space

T. Ngo-Anh

European Space Agency, Paris, France

Space is one of the most extreme environments imaginable. Beyond the insulating atmosphere of the Earth, astronauts are subjected to extremes of zero grvaity, isolation and confinement and a significantly increased threat of radiation damage which pose challenges and changes on their bodies, in addition, all these factors have a direct impact on the feasibility and success of such long-duration exploration missions with a human crew. So if we want to send a human crew to the Moon, to Mars or in general into Deep Space, we need to ensure that astronauts remain fit, functional and healthy throughout the entire mission, from the very beginning to the bitter end, we need to ensure that our crew gets to their destination in deep space and then of course back, safely - ESA's Human Research Programme contributes to that endeavour through all its different research projects.
The talk will provide an overview of all activities on the very diverse research platforms that are part of ESA's Human Research Programme, the results of which will provide more insight into these challenges and changes over a longer period of time, and present a stepping stone for even longer missions such that when we send humans on a journey to Mars, we will make sure that we have conquered the unknowns of the most extreme environment of all to ensure a safe trip home back to the envinroment we know and love.

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SA08

The brain at high-altitude; a radical perspective!

D. Bailey

Neurovascular Research Laboratory, University of South Wales, Pontypridd, UK

Photosynthesising cyanobacteria breathed life into what was, until a billion years ago considered a reductive atmosphere, thus providing a selective pressure for the evolution of (oxygen) O_2-dependent micro-organisms that began with the autotrophic eukaryotes. Since these primordial times, the respiring mammalian cell has become entirely dependent on molecular O_2 since it serves as the terminal electron acceptor in mitochondrial oxidative phosphorylation and multiple enzymes require O_2 as a substrate. The human brain exemplifies this reliance on O_2 since, unlike most other tissues, an evolutionary "drive for size" means that it is now committed to a continually active state. However, this comes at a cost and corresponding high vulnerability for failure. Given that the brain's O_2 supply is so delicate, it would seem likely that evolution has favoured a feedback mechanism that senses tissue PO_2 and consequently transmits a signal to the vasculature coupling local O_2 delivery to tissue metabolic demand. The current presentation will combine the joys of laboratory-based science with the thrills (and dangers!) of extreme field testing to shed unique insight into fundamental molecular mechanisms that allows the human brain to sense O_2 and the mechanisms that regulate its delivery. Experiments with "super-human" models including high-altitude mountaineers and freedivers will be discussed, providing unique insight into how our brains can adapt and overcome extremes of O_2-lack that would otherwise be considered incompatible with ordinary human life.

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SA09

Hypoxia - The Good, The Bad & The Ugly?

S. Dhillon

CASE Medicine, University College London, London, UK

Humans evolved at sea-level and tolerate exposure to acute hypoxia poorly.
Acclimatisation to terrestrial hypobaric hypoxia is possible with slow ascent, but the mechanisms are poorly understood especially with regard to the individual variations. Some people can tolerate climbing Everest (8,848m) without supplemental oxygen, whereas others struggle to reach Everest Base Camp (5,400m) even with a gradual ascent.
This presentation will provide an overview of the latest pathophysiology underlying the common altitude illnesses (Acute Mountain Sickness, High Altitude Cerebral Oedema and High

Altitude Pulmonary Oedema) and will conclude by highlighting some knowledge gaps and areas for future research.

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SA10

Physiology and the extremes of humanity: Challenges for Martian explorers

J. Pawelczyk

College of Health and Human Development, Penn State University, State College, PA, USA

Humans will be able to travel to Mars' orbit in the late 2020's, and should be able to land on the surface of the planet sometime between 2030-2040. The 30-month mission will expose humans to reduced gravity, radiation, confinement, and environmental conditions very different to those on Earth. Can humans survive the challenges they will be faced with? Scientists are trying to answer this question by considering how we can translate the knowledge we've gained through discoveries here on Earth to the extra-terrestrial environment. Former astronaut and physiologist James Pawelczyk will highlight the unknowns that remain in the mission to get humankind to Mars, and the opportunities for biologists to help us reach the most treacherous destination humankind has ever contemplated.

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SA11

Immersion in cold water: sudden death and prolonged survival

M. Tipton

Extreme Environments Laboratory, School of Sport, Health & Exercise Science, University of Portsmouth, Portsmouth, UK

Immersion in cold water represents one of the greatest environmental stresses to which the body can be exposed. Drowning is the leading cause of death in those undertaking sport, including recreational diving, and immersion is the second most common cause of accidental death in many countries of the world. However, it is a relatively "hidden" killer with many of the 1,000+ immersion deaths that occur each day worldwide going unnoticed. Drowning is also a "disease of youth", 64% of deaths are < 30 years old; 25% are < 5years old. Drowning death, in terms of time to cardiorespiratory arrest after submersion, takes about 130 seconds; with the chance of successful resuscitation falling to nearly zero percent by 27 minutes. This time increases to around 66 minutes if water temperature is below 6 °C (Tipton & Golden, 2011).

The likelihood of both drowning and surviving is intimately related to the change in the thermal state of the body. "Change" is a powerful stimulus to homeothermic animals. The hazardous physiological responses to cold water immersion that can be precursors to pathophysiological consequences such as drowning and sudden cardiac arrest are driven by the rate of change (fall) of skin temperature and, in terms of the cardiac response, the resulting change in sympathetic and parasympathetic inputs to the heart (Tipton *et al.* 1991; Winter *et al.* 2019). The physiological responses resulting in prolonged survival underwater are also dependent on rate of change, but this time it is the rate of change (fall) of brain temperature (Tipton & Golden, 2011).

Which of these outcomes prevails on submersion: death or survival, is also influenced by a wide variety of factors that we are only just beginning to understand and, thereby, tentatively predict those at most risk.

Tipton MJ, Stubbs DA & Elliott DH (1991). Human initial responses to immersion in cold water at 3 temperatures and following hyperventilation. *J Appl Physiol* 70, 317-322

Winter J, Tipton MJ & Shattock MJ (2018). Autonomic conflict exacerbates long-QT associated ventricular arrhythmia. *J Mol & Cell Cardiol* 116,145-154

Tipton MJ & Golden FStC (2011). Decision-making guide for the search, rescue and resuscitation of submerged (head under) victims. *Resuscitation* 82, 819-824

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SA12

Diving and Hyperbaric Physiology - what have we learned?

J.E. Clark

Centre for Human & Applied Physiological Sciences, King's College London, London, UK

The underwater environment poses a number of physical and physiological stresses on the body, not least of these is the effect of elevated ambient pressure which increases with depth. Hyperbaria is the underlying cause of a number of diving disorders mediated by gas saturation in the tissues including inert gas narcosis, oxygen toxicity and high-pressure neurological syndrome. In addition, inert (usually nitrogen) gas saturation has to be managed to avoid the clinical signs of decompression sickness.

Traditionally, gas saturation during descent and desaturation on ascent has been the focus of considerable research and has undoubtedly led to many advances in our understanding of gases under pressure in the body, the development of safer decompression algorithms for divers, and a better understanding of bubble formation upon decompression including the use of technology to ameliorate decompression illness. Accordingly, this is the primary physiological challenge considered in recreational diving training manuals, text books. However. the recognition of other pathologies and physiological challenges through better understanding of immersion physiology and dysbaria has taught us valuable lessons in understanding, diagnosing and ultimately, assessing the risks associated with diving.

This presentation will reflect on the history of diving research and take a look at current state and future perceptiveness in our understanding of the physiology of diving.

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SA13

Cardiorespiratory hypotheses how deep diving cetaceans avoid the bends

A. Fahlman

Oceanographic, Valencia, Spain

Hydrostatic lung compression in diving marine mammals, resulting in atelectasis, has been the main theoretical basis for limiting N2 uptake and avoiding gas emboli as they ascend. However, studies of beached and bycaught cetaceans and sea turtles imply that air breathing marine vertebrates may, under unusual circumstances, develop gas emboli that result in gas emboli (decompression sickness symptoms). Theoretical modelling of tissue and blood gas dynamics of breath-hold divers suggests that our current understanding of diving physiology in many species is poor, as the models predict DCS in most of their natural dive profiles. In this lecture published results from marine mammals and turtles are presented present an alternative mechanisms for how marine vertebrates control gas exchange in the lung, through management of the pulmonary distribution of alveolar ventilation () and cardiac output/lung perfusion (Q), varying the level of / (Q) mismatch in the lung. Results from studies on anatomy and physiology in animals and humans are combined to develop a novel hypothesis how marine mammals, and cetaceans in particular, could have volitional control of gas exchange during diving. This hypothesis provides an explanation for how man-made disturbances, causing stress, could alter the / Q mismatch level in the lung, resulting in an abnormally elevated uptake of N2, increasing the risk for gas emboli. In addition, this new hypothesis also explains how marine mammals are able to utilize the lung as an O2 store while minimizing N2 uptake and the risk for gas emboli. This hypothesis provides avenues for new areas of research, offers an explanation for how sonar exposure may alter physiology causing gas emboli, and provides a new mechanism for how marine vertebrates can avoid the diving related problems observed in human divers.

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SA14

A primer to cross-adaptation and cross-tolerance to novel stressors

J. Cotter

School of Physical Education, Sport and Exercise Sciences, University of Otago, Dunedin, New Zealand

Preceding sessions have focused on a specific stressor or environment, the adaptation to which can potentially be utilised to reduce strain or improve tolerance when exposed to a novel stressor. This is termed cross adaptation, while the underlying molecular basis providing cellular protection is termed cross tolerance. Cross adaptation and tolerance have great promise in occupational (esp. military), recreational, health and clinical settings, for mechanistic and practical reasons. Applying animal-based data of cross adaptation and cross tolerance to practical settings for humans at the extremes has, however, been underwhelming, e.g., for well-trained athletes using hypoxic, heat or nutritional stressors to enhance fitness per se, or patients using remote ischaemic conditioning before surgery. This presentation will therefore address the integrative context to cross adaptation and tolerance, focusing on combined-stressor contexts. Special consideration is given to exercise because it is a uniquely valuable stimulus for several reasons; (a) it contains at least six separate and self-regulating stressors, imposed endogenously, (b) it is almost universally accessible and highly dosable in time, space and intensity, and (c) cross adaptation is mediated by improved control of multiple homeostatically-regulated variables and thereby confers cross adaptation against myriad acute and life-long stressors.

Where applicable, the authors confirm that the experiments described here conform with the Physiological Society ethical requirements.

SA15

Cross adaptation for attenuating environmental strain, and improving exercise and health physiology

O. Gibson

Centre for Human Performance, Exercise and Rehabilitation (CHPER), Brunel University London, Uxbridge, UK

Cross adaptation is the process of inducing physiological adaptation utilising one environmental stressor (e.g. heat) prior to exposure in another environmental stressor (e.g. hypoxia) resulting in attenuated disruption to homeostasis relative to the unadapted state. Further to this, data has begun to examine the impact of combined stressors e.g. heat and hypoxia, on adaptation.
Cross-, and combined adaptation between environmental stressors may be induced at rest, or during exercise in both terrestrial environments (as a cross acclimatisation model) as well as artificial environments (e.g. a cross acclimation model). Irrespective of the method induction, the underpinning adaptations are derived from cellular and molecular pathways with adaptations at this level being described as “Cross Tolerance”. This presentation will outline our current understanding of mechanisms and applications pertaining to cross-, and combined adaptation and the relevant pathways of cross tolerance, in exercise and health physiology.

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SA16

Humans and the environment: flipping the coin

H. Montgomery

Medicine, UCL, London, UK

It took 3.5 billion years for multicelled life to appear on Earth, another 540 million for humans to evolve. By 1804, there were only 1 billion of us. There are now nearly 8 billion of us, and we now add another billion every 12-14 years. In the last 50 years, our use of natural resources has accelerated beyond the boundaries which sustain life on Earth, destroying the habitats in which ecosystems can prevail. The numer of vertebrates on the planet has fallen by 70% since only 1970, and 8 species become extinct each hour. We are living through the greatest and fastest mas extinction the planet has ever seen.

Now we add climate change: the greenhouse gases we add to our atmosphere retain the equivalent of 5 Hiroshima bombs of energy each second within it. Energy in an atmospheric system causes weather- and we are experiencing more frequent and more extreme weather events around the world. Polar ice is melting and sea levels rising at ever-faster rates. The threat to human health is accelerating. And this can only get worse: 1/5th of the CO2 we release today will still be warming the planet in 33,000 years time, and 70% will be doing so in 100,000 years.
Immediate human survival depends upon immediate and meaningful action- but this is not happening. Hugh will discuss the implications of this torpid state.

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C01

Comparative effect of daylight restriction and sleep deprivation on the immune response of male Swiss mice

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Circadian rhythms modulate the bodies immunes system. This study was designed to compare the effect of two circadian rhythm disruptors (daylight-restriction and sleep deprivation) on the immune response of male Swiss mice.

Animals where divided into group 1(control; n=10), which were neither daylight-restricted or sleep-deprived and groups 2,3,4 and 5 (n=20/group) respectively. Ten animals each from groups 2-5 were restricted from daylight for 12,24,42 and 72hours respectively while the remaining 10animals per group were sleep-deprived for same time interval. Post-exposure, blood samples were collected into EDTA-lined sample bottles (n=5/subgroup) for haematological indices (eosinophils, neutrophils, platelet, white blood cell (WBC), monocyte, lymphocyte) and plain sample bottles (n=5/subgroup) for serum biochemical assays (interferon-γ, superoxide dismutase (SOD) reduced glutathione (GSH), and malonaldehyde (MDA). Data were analysed with ANOVA at $p<0.05$. Compared to control, lymphocytes increased ($p<0.05$) while WBC, platelet and neutrophil reduced from 12-72hours following either sleep-deprivation or daylight-restriction. Eosinophil's also increased in the experimental groups from 24-72hours post-exposure respectively. Daylight restriction increased monocytes 48-72hours while sleep-deprivation increased monocytes at 12,24 and 72hours respectively compared to control. Daylight-restriction increased interferon-γ(pg/ml) at 12(751.8±31.8), 24(745.4±33.8), 48(890.0±30.9) and 72(773.4±53.3) hours while sleep-deprivation at 48(1078±119.1) and 72(909±94.0) hours increased respectively post-exposure compared to control (452.3±43.6). SOD (U/mg protein) in the sleep-deprived group increased at 12(2.86±0.07), 24(2.74±0.08) and 48(4.92±0.13) hours but decreased at 72hours (1.26±0.08) respectively while values in the daylight-restricted animals from 12–72hours (1.38±0.38;0.82±0.17;0.74±0.17;0.82±0.15) decreased ($p<0.05$) compared to control (2.12±0.31). Compared to controls (28.89±3.47), GSH (mg/ml) in the sleep-deprived group at 12-72hours (22.25±1.31;18.56±4.05;14.86±3.27;9.69±1.15) reduced ($p<0.05$) while values in the daylight-restricted group increased at 12(44.94±4.59) and 24(33.44±4.49) hours but reduced at 48(17.84±2.04) and 72(13.68±1.97) hours respectively. MDA (mmol/mg protein) at 12-72hours in both sleep-deprived (3.07±0.44;3.65±1.31;2.68±0.18;2.76±0.34) and daylight-restricted (2.73±0.82;3.63±0.99;3.67±0.97; 3.14±0.78) groups were increased respectively compared to control(1.77±0.22).

This study suggests that the immune system response to daylight restriction maybe faster than that of sleep deprivation. It also suggests that daylight restriction and sleep deprivation may compromise body defense mechanisms and thus predispose to infections and diseases.

Where applicable, the authors confirm that the experiments described here conform with the Physiological Society ethical requirements.

C02

Beet the cold: Beetroot juice supplementation improves peripheral blood flow, endothelial function and anti-inflammatory status in individuals with Raynaud's phenomenon.

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Raynaud's phenomenon (RP) is characterised by recurrent transient peripheral vasospasm and lower nitric oxide (NO) bioavailability in the cold. We investigated the effect of nitrate-rich beetroot juice (BJ) supplementation on *i)* NO-mediated vasodilation, *ii)* cutaneous vascular conductance (CVC) and skin temperature (T_{sk}) following local cooling and *iii)* systemic anti-inflammatory status.

Following baseline testing, twenty-three individuals with RP attended four times, in a double-blind, randomized crossover design, following acute and chronic (14 days) BJ and nitrate-depleted beetroot juice (NDBJ) supplementation. Peripheral T_{sk} and CVC were measured during and after mild hand and foot cooling, and during transdermal delivery of acetylcholine and sodium nitroprusside. Markers of anti-inflammatory status were also measured.

Plasma [nitrite] was increased in the BJ conditions ($P < 0.001$). Compared to the baseline visit thumb CVC was greater following chronic-BJ ($\Delta 2.0$ flux.mmHg^{-1}, $P = 0.02$) and chronic-NDBJ ($\Delta 1.45$ flux.mmHg^{-1}, $P = 0.01$) supplementation; however, no changes in T_{sk} was observed ($P > 0.05$). Plasma [interleukin-10] was greater whilst pan endothelin was reduced, forearm endothelial function was improved, and systolic and diastolic blood pressure (BP) were lowered by both BR and NDBJ ($P < 0.05$).

Acute and chronic BJ and NDBJ supplementation improved anti-inflammatory status, endothelial function and BP. CVC following cooling increased post chronic-BJ and chronic-NDBJ supplementation, but no effect on T_{sk} was observed.

We would like to thank the participants who volunteered to take part in this. We would also like to thank the National Institute for Health Research Clinical Research Network for adopting this study onto their portfolio and the research nurses (Rheumatology Department, Queen Alexandra Hospital, Portsmouth; Paula White and Marie White) who supported participant recruitment. We acknowledge help with data collection / analysis from Freyja Haigh. We gratefully acknowledge the funding from the University of Portsmouth and James White Drinks Ltd. MG has received funding from James White Drinks Ltd for development of the placebo. All other authors declare no conflict of interest. This research was supported by the the National Institute for Health Research (NIHR) Leicester Biomedical Research Centre. The views expressed are those of the authors and not necessarily those of the NHS, the NIHR or the Department of Health.

Where applicable, the authors confirm that the experiments described here conform with the Physiological Society ethical requirements.

C03

Winter energetics of Svalbard reindeer: life on a tight budget

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The Arctic is one of the biomes that undergoes the most dramatic seasonal changes in photoperiod, ambient temperature and primary production. Reduced winter metabolic rate as a response to food scarcity may facilitate survival through the most unfavourable conditions (1). We measured the daily energy expenditure (DEE) of adult female (age 4-8 years) Svalbard reindeer (*Rangifer tarandus platyrhynchus*) using the doubly labelled water (DLW) technique. Using telemetry data we aimed to quantify adaptations to cold and low food supply in winter by exploring drivers of individual variation in DEE. Individuals were caught in late winter (March-April) in 2017 and 2018 in Nordenskiöld Land, Svalbard, Norway. The animal handling protocol was approved by the Norwegian Food Safety Authority (permit no. 17/237024) and the Governor of Svalbard (permit no. 16/01632-9). Once captured, animals were weighed and dosed with ~0.3 mL of DLW (65 atom% ^{18}O, 35 atom% ^{2}H) per kg of body mass, and re-sampled 2 and 10-20 days later for initial and final isotope concentrations, respectively. All females were fitted with a GPS collar (weight of 0.75 kg) that recorded hourly positions and acceleration in X-Y axes every 5 min. Mean DEE of Svalbard reindeer was 6.2 MJ day^{-1} (SD = 0.7, n=21). The measured DEE was only 43% of that predicted from allometric scaling for ungulates (2). Mean body mass (BM) was 49.0 kg (SD = 3.4) and mean fat-free mass (FFM; 73% of total body water) was 42.0 kg (SD = 3.7). Using linear regression, we showed that both FFM and activity levels had positive and significant effects on DEE (r^2 = 0.51, p < 0.001). FFM and activity levels accounted for 26.6% and 24.2% of the variation in DEE, respectively. We found no effects of displacement by GPS, pregnancy or age on DEE. We then modelled DEE over winter to estimate winter energy budgets, based on activity levels and mean autumn body composition of adult females. Our estimates show that individual winter energy budgets will vary depending on body size, composition (relative amount of fat and FFM) and activity pattern throughout the winter, and that higher relative fat mass (% of BM) determines starvation buffering capacity. Energy reserves contribute only 12% of total winter energy expenditure for a light, lean and active reindeer, while it provides 35% of total winter energy expenditure for a heavy, fat and sedentary reindeer. We demonstrate that Svalbard reindeer expend less energy in the winter than previously assumed, which is likely facilitated by the insulating capacity of their fur and relatively sedentary lifestyle. Our results highlight the importance of individual variation in estimates of energy expenditure in the wild, which may provide a major advance in the assessment of species' resilience to deteriorating winter conditions in a changing climate (3).

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J. Irvine, E. Ropstad, A.Stien and V. Veiberg are core members of the research team.

Where applicable, the authors confirm that the experiments described here conform with the Physiological Society ethical requirements.

C04

The Seal, the Bear & the Super-Ape: Turning Down the *"Master Switch"* & *"Fire of Life"* to a Low, Slow & Cool *"Pilot Light"* – Glimpses of Phenomenal Brain Cooling at the O_2-Austere Survival Limit-of-Life in a Human Outlier-Extremophile

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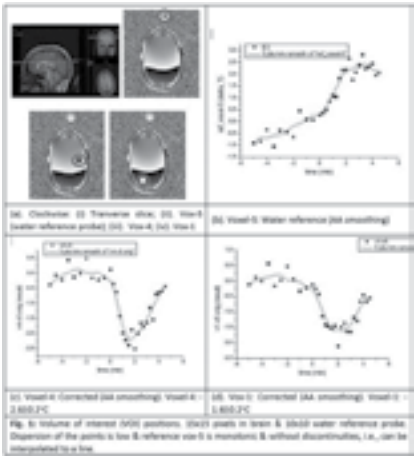
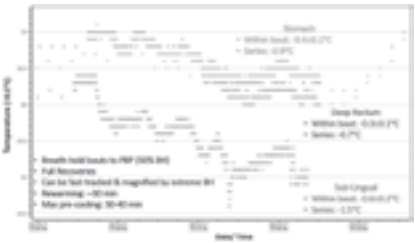
Whether humans possess a latent animalesque capability of metabolic downregulation, to subBMR-low, time-dilated-slow & anapyrexia-cool levels, ideally, fast like diving seals & sustainably like hibernating bears remains an open question & centuries-elusive, say, holy-grail, physiological & biomedical grand-challenge. Cracking the metabolic enigma code of this *dark region* of the metabolic spectrum would profoundly enlarge the scope of metabolic protection & survival in extreme & austere environments, e.g., asphyxia, hypoxia, hypo/hyperthermia, hypo/hyperphagia, hypoydration, infection, hyper/hypogravity, hypokinesia, IR radiation, scar-less wound-healing, neuro-remodeling, fast-track & cross-transplant acclimatization, biological time dilation, etc., etc., i.e., motherlode platform of biomedical *superpowers*. There exists a notion that various animal bradymetabolic states are somehow connected & may be enabled via a universal *"master switch"*,[1] e.g., diving seals & hibernating bears basically express the same physiological features.[2] Certainly, it sounds logical, neat & efficient, & would make ID this *dimmer-switch* easier. The idea of isolating the switch & trigger by turning to human divers[3] makes much sense since one might expect that any humans capability, if it exists, would most likely on-demand express in the face of the "*heroic measures to the threat of asphyxia,*"[1,3] & with that animalesque-classic & quintessential-telltale hallmark, spontaneous brain cooling. Presented here, glimpse outcomes of investigations of human brain cooling phenomena involving dozens of trials & spanning several years undertaken time ago on a human diver (the author) as part of a long-running R&D program exploring such exotic states for various *enhancement* apps. Specifically presented, best-in-class outcomes & mechanistic insights behind all-natural, prompt, ultra-rapid & profound *super-cooling* phenomena expressible from normothermia.[4] Essentially, basically, the mechanism involves animal diver & hibernator strategies, also employed by the author for deep diving, namely, long expiratory pauses between breaths.[5] The mechanism allows brain-wise preferential redistribution of cooled blood from *thermal windows* for protection against a relative hyperthermia under the threat of hypoxia. Notable, due to a greater relative metabolic demand & hypoxia sensitivity, brain

cooling expresses faster than in a seal, as evidenced by the less pronounced GI-tract cooling (Fig.2): 0.4±0.1°C (mean±s.d., n=5; *e-Celsius*, *BodyCap*, ±0.2°C). Supporting data suggests the limit in diving may be on par with that of a similarly sized seal or bear, ~32°C.



Human Expiration Breath-Holding Brain Cooling Response



Human Expiration Breath-Holding GI-Tract (& Sub-Lingual) Cooling Response

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C05

Role of bradykinin in human sweating during simulated and actual heat stress

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Bradykinin, a local dermal kallikrein-kinin system product, increases skin blood flow via a cyclic GMP mechanism but is not the active cutaneous vasodilation molecule associated with cholinergic stimulation. However, the precise role of bradykinin in sweating and thermoregulation is unclear. We tested the hypothesis that bradykinin increases eccrine sweating via increases in cutaneous capillary permeability and fluid extravasation. Protocol #1: physiological sweating was induced in 10 healthy subjects via perfusing warm (46-48°C) water through a high-density tube-lined suit to induce heat stress. During heating a bradykinin type 2 (B2) receptor antagonist (HOE-140; 40μM) and the vehicle (lactated Ringer's) were perfused intradermally via microdialysis, while sweating (capacitance hygrometry) and cutaneous vascular conductance (CVC; Doppler flux/mean arterial pressure) were obtained directly superficial to the membrane. In addition, both microdialysis membranes were then perfused with bradykinin (1mM). Protocol #2: pharmacological sweating was induced in 6 healthy subjects via intradermally perfusing a cholinergic agonist (pilocarpine; 1.67 mg/ml) to mimic heat stress and steady state sweating conditions and was followed by the same B2 antagonist and agonist approach. Increases in internal (37.1±0.1 to 37.9±0.1°C) and uncovered local skin (30.1±0.4 to 32.9±0.4°C) temperature caused increases in sweat rate (+0.79±0.12 and +0.64±0.10 mg/cm^2/min) and CVC (63±11 to 181±22 and 85±15 to 204±19 flux/mmHg for HOE-140 and vehicle, respectively), while HOE-140 and control sites were not different. Heart rate increased (62±3 to 94±6 bpm) with whole-body heating but arterial blood pressure was not significantly altered. Pilocarpine induced sweating (+0.38±0.16 and +0.32±0.12 mg/cm^2/min) and increases in CVC (88±36 to 183±55 and 73±25 to 208±66 flux/mmHg for HOE-140 and vehicle, respectively) but again, no changes between sites were noted. These data indicate that B2 receptor antagonists do not modulate physiological or pharmacological sweating. HOE-140 delivered during normothermia was also identified not to be sudorific. The addition of exogenous bradykinin also did not modulate sweating during whole-body heating or pilocarpine perfusion in either control or HOE-140 sites. These data indicate HOE-140 does not affect sweating independently and B2 agonists do not modulate absolute sweat output. Although the kallikrein-kinin system is present in eccrine sweat glands, its precise role remains to be elucidated. Current data do not support a mechanism related to absolute *in vivo* sweat output and evaporative cooling but rather its role may be more condition-specific or supportive to epithelial transport and the alteration of the interstitial milieu around the gland.

Funding Source: NIH(NIAMS) - AR069912.

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C06

Regional thermal hyperaemia—evidence of a critical role of local thermosensitive mechanisms in the control of the human leg circulation during hyperthermia

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Hyperthermia is thought to increase limb tissue blood flow (BF) through activation of thermosensitive mechanisms within the limb vasculature (1). However, the precise vascular locus in which hyperthermia causes vasodilatation and increases in BF in the different segments of the human leg is not fully characterised and understood. Specifically, the distribution of upper- and lower-leg BF during local hyperthermia—the BF responses in the major leg arteries and microcirculation as a result of whole and partial limb hyperthermia—remain unknown. This study tested the hypothesis that temperature-sensitive mechanisms alter limb haemodynamics by acting downstream from the conduit arteries, whether that be whole-leg haemodynamics in response to whole-leg heating (WLH) or leg-segmental haemodynamics in response to upper- (ULH) or lower-leg heating (LLH), respectively. METHODS: A cohort of healthy males and females (31±13 years) participated in three protocols. Leg haemodynamics of the common (CFA), superficial (SFA) and profunda (PFA) femoral arteries and popliteal artery (POA), and temperature profiles of the experimental and control leg were measured during each protocol: (1) 3h of WLH followed by 3h of passive recovery (n=5); (2) 1h of ULH followed by 30min of cooling and a subsequent 1h bout of ULH (n=8); (3) 1h of LLH (n=6). RESULTS: WLH increased mean whole-leg temperature (T_{Leg}) of the experimental leg by 4.2±1.2°C (mean±SD), whilst core and control-leg temperatures remained stable. WLH induced ≥3-fold increases in blood perfusion in CFA, SFA, PFA and POA of the experimental leg, whilst control leg haemodynamics remained stable. During WLH, upper-leg BF increased by 499±331 ml/min and lower-leg BF increased by 277±136 ml/min, in a linear response to whole T_{Leg} (R^2=.95; p<.01). When expressed in relation to limb segment tissue mass, however, upper- and lower-leg BF were similar: ~9 ml/min/100g. Following the cessation of WLH, BF remained higher in the experimental leg for the subsequent 3h. Furthermore, ULH increased upper T_{Leg} by 3.3±0.9°C and upper-leg BF by 536±243 ml/min which are comparable to WLH, without any changes to lower T_{Leg} tissue oxygenation or BF. Conversely, LLH increased lower T_{Leg} and BF—5.7±0.9°C and 287±130 ml/min—without altering upper T_{Leg}, tissue oxygenation or skin and PFA BF. DISCUSSION: The present findings demonstrate that WLH induces a sustained ≥3-fold elevation in upper- and lower-limb BF and that segmental hyperthermia matches the regional thermal hyperaemia without affecting BF, temperature or tissue oxygenation of the non-heated limb segment. These findings together with the unchanged BF in the PFA and POA during lower and upper leg heating, support the notion that local downstream thermosensitive mechanisms control human leg circulation during hyperthermia.

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Where applicable, the authors confirm that the experiments described here conform with the Physiological Society ethical requirements.

C07

Impact of Solar Radiation on Physical Work Capacity During Heat Stress in Humans

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Self-paced physical work in the heat tends to be associated with a stable working heart rate, integrating the strain of the physical work and the thermal climate. It follows that work rate decreases in-line with the severity of heat stress, in order to maintain a stable heart rate. With climate change, increased environmental heat stress will thus be met with reduced physical work capacity (PWC%), an effect which has global economic implications. Understanding the independent effect of solar radiation on human performance is critical when developing an empirical model of PWC% in future climatic scenarios.

The aim of this study was to document how the effect of solar radiation on physical work capacity changes as a function of the air temperature, humidity, and level of clothing insulation. 14 young adult males (7 semi-nude, 7 full body coveralls) walked for 1-hour at a fixed heart rate of 130 b/min, in seven air temperature (25 to 45°C) and relative humidity (20 or 80%) combinations, with and without solar radiation (800 W/m^{-2} intensity using solar spectrum lamps). A total of 172 trials (90 semi-nude, 82 with coveralls) were completed in this study. The cumulative net energy expenditure above resting metabolism was calculated based on the treadmill speed and grade. To determine PWC%, the net kilojoules of work in each heated condition was expressed relative to that achieved in a reference condition without heat stress (15°C, 50% relative humidity).

The impact of solar radiation on PWC% during heat stress depended on the air temperature, humidity, and clothing. At 20% relative humidity in semi-nude, solar radiation had only a marginal impact on PWC% at air temperatures ≤40°C (< 5% PWC loss). In dry conditions but with protective coveralls, solar radiation consistently lowered PWC% at all air temperatures >25°C by 10 to 15%, indicating increased vulnerability from solar radiation when wearing protective clothing. At 80% relative humidity, solar radiation decreased PWC% similarly between semi-nude and clothed (10 to 20% PWC loss). The absolute loss in PWC% was predicted by the change in mean skin temperature, in both solar and non-solar conditions. Thermal indices which do not account for solar radiation (i.e. natural wet bulb, humidex) over-estimate PWC% in outdoor working scenarios.

In summary, solar radiation had a different effect on PWC% depending on the biophysical aspects of the environment, and if clothing was worn. Solar radiation had a marginal impact in dry conditions, unless protective clothing was used. Solar

radiation reduced PWC% in humid conditions regardless of the clothing ensemble. The study contributes to the development of a comprehensive empirical model which aims to predict PWC% in future climate scenarios.

Funding was provided by 'HEAT-SHIELD', European Union's Horizon 2020 research and innovation programme under grant agreement no. 668786.

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C08

Five days of dietary nitrate supplementation has no effect on exercise or thermoregulation in the heat

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There has been no reported effect of acute or chronic dietary nitrate supplementation on exercise in the heat, despite thermal balance depending upon a number of modifiable factors, many of which could be altered by the physiological effects elicited by the nitrate-nitrite-nitric oxide pathway (NO_3^-–NO_2^-—NO) (Amano et al., 2018; Kent et al., 2018). However, these studies have not been conducted in hot, dry environments, during exercise of higher intensities, which might provide the necessary conditions to realise these effects. Furthermore, based on previous reports (Porcelli et al. 2015), it is feasible that the potential effects of dietary NO_3^- are potentiated among participants of lower training status, who might also have greater capacity for acute heat adaptation. The aim of this study was to determine the effect of 5-days dietary nitrate consumption on exercise tolerance, thermoregulation and perceptual response during cycling in hot, dry conditions. Using a randomised, double-blind, crossover-design, 11 untrained participants (age: 23 ± 7 y; stature: 182.0 ± 5.2 cm; body mass: 78.7 ± 7.5 kg; VO_{2max}: 48 ± 12 ml/kg/min) performed two trials to their limit of tolerance (T_{lim}) at the power output associated with their thermoneutral gas exchange threshold in a hot, dry (35 °C & 30% relative humidity) environment, following ingestion of either 140 ml NO_3^- in the form of a beetroot juice beverage ~9.2 mmol (BR) or placebo (PLA), for 5 days. Breath-by-breath gas analysis was performed, alongside continuous measurements of local sweat rates, heart rate (HR), rectal (T_c) and weighted mean skin temperatures (T_{skin}). Thermal sensation and rating of perceived exertion (RPE) were also measured. Nude body mass was recorded pre- and post-exercise as an indication of whole-body sweat rate.

Plasma [NO_3^-] was increased in BR *vs.*PLA (P< 0.001) following the 5-day supplementation. There were no changes in T_{lim} between conditions (PLA: 21.6 ± 7.4 min *vs.*BR: 23.1 ± 8.3 min; P= 0.171) and there were no main effects of condition for T_c(PLA: 37.5 ± 0.04 °C *vs.*BR: 37.4 ± 0.13 °C; P= 0.629), T_{skin}(PLA: 35.4 ± 0.17 °C *vs.*BR: 35.5 ± 0.16 °C; P= 0.763), HR (PLA: 169 ± 4 beats/min *vs.*BR: 171 ± 3 beats/min; P= 0.685), or sum of local sweat rates (PLA: 557 ± 38 nL/min *vs.*BR: 565 ± 41 nL/min; P= 0.832). The RPE (PLA: 16.2 ± 0.3 *vs.*BR: 16.1 ± 0.4; P= 0.635) and thermal sensation (PLA: 2.8 ± 0.2 *vs.*BR: 2.8 ± 0.2; P= 0.858) were also not affected by condition. There were no interactions between time and condition across all variables (P< 0.05). Despite a 1.0 ± 0.5 % loss of fluid in the NO_3^- condition *vs.*PLA (0.7 ± 0.2 %), there were no significant differences (P= 0.170).

Five-days of NO_3^- supplementation had no effect on T_{lim} in dry heat, nor did it alter thermoregulation or perceptual responses of untrained participants.

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C09

Exercise thresholds in hot environmental conditions: is there a shift?

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Introduction: Exercise thresholds (e.g. ventilatory and lactate thresholds) are widely used in the field of sports science and demarcate the intensity domains of moderate, heavy and severe exercise. However, these thresholds should not be used interchangeably[1], also environmental factors (e.g. temperature) will influence the work rate at which these exercise thresholds will occur[2]. Since sports activities are often performed under hot environmental conditions, determination and interpretation of the exercise thresholds in these conditions is needed.

Methods: Twelve physically active young men performed four exercise tests in total. Two ramp incremental exercise tests (30W.min^{-1}), one in temperate conditions (18°C) (TEMP) and one in hot conditions (36°C) (HOT), were done to determine the ventilatory thresholds, i.e. gas exchange threshold (GET) and respiratory compensation point (RCP). GET was defined as the point where VCO_2 increased disproportionate to VO_2. RCP corresponded to the point where V_E increased disproportionate to VCO_2. Two step incremental exercise tests (80W+40W.3min^{-1}) were executed to define the first and second lactate threshold, both in TEMP and HOT. The first lactate threshold (LT_1) was determined as the point with the first increase in lactate concentration. The modified D_{max} method was used to define the second lactate threshold (LT_{MOD}). Exercise tests were performed in randomized order. Paired Samples T-Test was used for the statistical analysis in SPSS. Data are expressed as mean ± SD for n = 12.

Results: Work rate at LT_{MOD} in HOT is significantly different from LT_{MOD} in TEMP (233 ± 33 vs. 246 ± 38 W; p = 0.019). Work rate at thresholds LT_1, GET and RCP did not differ between HOT and TEMP. Nevertheless the heartbeat was significantly higher in HOT than in TEMP for threshold LT_1 (135 ± 9 vs. 131 ± 10 bpm; p = 0.047), GET (154 ± 8 vs. 143 ± 10 bpm; p = 0.003) and RCP (168 ± 9 vs. 162 ± 10 bpm; p = 0.047). Maximal work rate in the step protocol was significantly lower in HOT than in TEMP (297 vs. 314W; p < 0.001), but not in the ramp protocol (363 ± 50 vs. 371 ± 45 W; p = 0.164). Work rate at GET in

TEMP and HOT is significantly higher than at LT_1, respectively 188 ± 37 vs. 167 21 W (p = 0.001) and 188 ± 34 vs. 161 ± 24 W (p < 0.001). Work rate at RCP is only higher than at LT_{MOD} in HOT (243 ± 39 vs. 233 ± 33 W; p = 0.027).
Conclusion: Although there is only at one exercise threshold (LT_{MOD}) a significant lower work rate in HOT than in TEMP, the altered heart rate at the exercise thresholds must also be taken into account when analyzing and interpreting exercise tests. Therefore exercise tests must be performed in the environmental conditions as in which the peak performance has to be delivered. Additionally, ventilatory and lactate thresholds do not occur at the same work rate and thus should not be used interchangeably.

Caen et al. (2018), *Medicine and Science in Sports and Exercise* 50, 1277-1284.

de Barros et al. (2011), *Int J Sports Medicine* 32, 749-753.

Where applicable, the authors confirm that the experiments described here conform with the Physiological Society ethical requirements.

C10

Encapsulation of carbohydrate within a pectin-alginate hydrogel does not improve blood glucose availability, whole body carbohydrate oxidation, or time trial performance during prolonged cycling in hot and humid conditions.

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The Tokyo Olympics and Paralympic games in 2020 will be held in hot and humid conditions with daily wet bulb globe temperatures expected to be in excess of 28°C (1). The performance impairment associated with exercising in the heat can in part be mitigated by carbohydrate ingestion (2). Elite athletes have begun to use carbohydrate beverages encapsulated in pectin-alginate hydrogel, however at present little experimental evidence exists to support their use over traditional glucose-fructose drinks (3). Here we compare whole body substrate oxidation, plasma metabolites, and cycling time trial performance in hot and humid conditions while ingesting an encapsulated glucose-fructose drink or a nutrient matched non-encapsulated glucose-fructose drink. Eight endurance trained cyclists (6 men, age 27 ± 8 years, height 176 ± 10 cm, mass 74 ± 11 kg, VO_{2max}: 55.2 ± 9.5 ml·kg^{-1}·min^{-1}) cycled (45% VO_{2max}) for 90 minutes and completed a 15-minute time trial in hot humid conditions (32°C, 70%) on 3 occasions (water, glucose-fructose, glucose-fructose-hydrogel). Before exercise, participants drank an initial 250 mL bolus of either water, glucose-fructose (90 g carbohydrate per hour), or glucose-fructose hydrogel (90 g carbohydrate per hour). Thereafter they were provided with 145 mL of the trial drink every 15 minutes. Whole body fat and carbohydrate oxidation was determined every 10 minutes via indirect calorimetry, and capillary blood samples were obtained at 15 minute intervals to determine plasma metabolites. Data are presented as the mean values obtained throughout the 90-minute exercise period. Fat oxidation was lower during glucose-fructose (0.17 ± 0.14 g·min^{-1}) and hydrogel (0.17 ± 0.06 g·min^{-1}) trials vs. water (0.34 ± 0.14 g·min^{-1}, both p < 0.0001). Carbohydrate oxidation was higher during the glucose-fructose (1.72 ± 0.72 g·min^{-1}) and hydrogel (1.68 ± 0.62 g·min^{-1}) trials compared to water (1.32 0.52 g·min^{-1}; both p > 0.0001). There was no difference in either fat or carbohydrate oxidation between the glucose-fructose and hydrogel trials. Plasma glucose was higher throughout the glucose-fructose (5.54 ±0.34 mmol/L) and hydrogel (5.6 ± 0.43 mmol/L) trials compared to water (4.61 ± 0.34 mmol/L; trial x time interaction, F = 3.469, p = 0.012). Time trial performance was greater in the glucose-fructose (164 ± 32 kJ) and hydrogel trials (161± 31 kJ) compared to water alone (139 ± 32 kJ; both p < 0.01). The consumption of encapsulated carbohydrates does not increase blood glucose availability, alter whole body substrate oxidation, or improve time trial performance when compared to a nutrient matched glucose-fructose beverage consumed during prolonged cycling in hot and humid conditions.

Gerrett, N., et al. (2019). "Ambient Conditions Prior to Tokyo 2020 Olympic and Paralympic Games: Considerations for Acclimation or Acclimatization Strategies." Frontiers in physiology 10.

Febbraio, M. (1999). "Temperature, muscle metabolism and performance." Perspectives in exercise science and sports medicine: The metabolic basis of performance in exercise and sport: 315-353.

Sutehall, S., et al. (2018). "Sports Drinks on the Edge of a New Era." Current sports medicine reports 17(4): 112-116.

Where applicable, the authors confirm that the experiments described here conform with the Physiological Society ethical requirements.

C11

The time course of adaptations to seven-weeks intermittent post-exercise sauna bathing for inducing heat acclimation in trained middle-distance runners.

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Athletes require heat acclimation protocols that can be both flexible and integrative, whilst still being effective in inducing hallmark adaptations. We hypothesised that post-exercise sauna bathing 1) could be interspersed across a seven-week training period as a form of long-term heat acclimation, and 2) would be increasingly effective in inducing hallmark heat acclimation adaptations (i.e., reduced rectal temperature [Trec], heart rate [HR], sweating [indexed via body mass loss and sweat gland activation], perceived exertion [RPE] and thermal perception) when assessed after 3 and 7 weeks. Six trained middle-distance runners (3 female; mean±SD; age 19±1 years, VO_{2max} 60.2±10.1 ml·kg^{-1}·min^{-1}) performed a running heat tolerance test (30-minutes, 9 kph/2% gradient, 40°C/40%RH; RHTT) before ($RHTT_{PRE}$), following 3-weeks ($RHTT_{3W}$) and following 7-weeks ($RHTT_{7W}$) endurance training with 30-minutes post-exercise sauna bathing (101-108°C) 3±1 times per week. Data were analysed using a one-way ANOVA, with Bonferroni-corrected *post hoc* comparisons. To assess ordinal data, Friedman's test was performed with *post hoc* analysis by Wilcoxon sign-rank tests.
Resting Trec was lower (main effect: p=0.010) at 3 weeks (-0.2±0.1°C; p=0.036) and 7 weeks (-0.2±0.2°C; p=0.038) as compared to pre-acclimation. Peak Trec was lower (main effect: p<0.001) during $RHTT_{3W}$ (-0.3±0.2°C; p=0.043) and $RHTT_{7W}$ (-0.4±0.2°C; p=0.006), as compared to $RHTT_{PRE}$. Furthermore, peak Trec during $RHTT_{7W}$ was lower than that

during $RHTT_{3W}$ (-0.1±0.1°C; p=0.038). Peak RPE was lower (main effect: p=0.042) in $RHTT_{7W}$ only, both in comparison to $RHTT_{PRE}$ (-3±3 pts; p=0.043) and in comparison to $RHTT_{3W}$ (-1±1 pts; p=0.034). There was a main effect of time on peak HR (p=0.022; $RHTT_{PRE}$: 160±20 bpm, $RHTT_{3W}$: 149±20 bpm, $RHTT_{7W}$: 151±15 bpm), whole-body sweat loss (p=0.043; $RHTT_{PRE}$: 0.9±02 kg, $RHTT_{3W}$: 0.8±0.1 kg, $RHTT_{7W}$: 0.8±0.2 kg), and sweat gland activation on the forearm (p=0.039; $RHTT_{PRE}$: 42±15 active glands/cm^2, $RHTT_{3W}$: 69±9 active glands/cm^2, $RHTT_{7W}$: 69±13 active glands/cm^2), though these differences could not be located *post hoc*. Thermal comfort did not significantly change (p=0.072; mean ratings between "Uncomfortable" and "Slightly Uncomfortable"),
nor did ratings of thermal sensation (p=0.115; mean ratings between "Hot" and "Warm").
These data indicate that some hallmark adaptations of heat acclimation can be observed following 3-weeks intermittent post-exercise sauna bathing (i.e. resting and exercising rectal temperatures), with some further development occurring with longer (i.e. 7-weeks) exposure. Changes in perceived exertion were only observed following 7-weeks exposure. Further investigation is required to determine the effect of intermittent post-exercise sauna bathing on cardiovascular and sudomotor outcomes.

Where applicable, the authors confirm that the experiments described here conform with the Physiological Society ethical requirements.

C12

Daily Generation of Hydrostatic Gradients Attenuates Ocular Changes Associated With Head-Down Tilt Bedrest.

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Astronauts develop Space flight–associated neuro-ocular syndrome due to the persistent lack of Earth's gravity. At present, the pathophysiology is uncertain, but may be due to the absence of diurnal, postural reductions in intracranial pressure relative to intraocular pressure during periods of zero gravity (Lawley *et al.* 2017; Anderson et al. 2015). Thus, recreating gravitational gradients in space may be an effective countermeasure. We have recently shown that short durations (5 to 20 minutes) of low level (-20-30 mmHg) lower body negative pressure (LBNP) consistently reduces intracranial pressure by ~4 mmHg, and thus reintroduces about 25% of the gravitational effect of standing upright on earth (Petersen et al. 2018).
The objective of this study was to determine if prolonged (8 hrs) simulated partial hydrostatic unloading of the brain via low-level LBNP could act as a countermeasure for choroidal engorgement associated with simulated microgravity by -6 deg head down tilt bedrest.
This interventional study included five men and two women who spent 3 days in -6 deg head down tilt bedrest on two occasions in a randomized, counter-balanced, cross-over design. During one visit, participants spent 8 hours per day (10am - 6pm) inside an airtight chamber sealed at the level of the iliac crest with an internal pressure of -20 mmHg. Optical coherence tomography was performed in both trials in the supine position and after 3 days in -6 deg head down tilt. Moreover, in two participants intracranial pressure was measured directly for 9 hours in both trials to document the long-term effects of LBNP on intracranial pressure.
The primary outcome variable was the change in volume / area of the choroid (mean±sd).
Lying in the -6° head down tilt position for three days without the use of a pillow caused an increase the choroid area (Δ0.11 mm^2, p=0.05) and volume (Δ0.45 mm^3, p=0.003). If participants spent 8 hours per day under low-level LBNP, the choroid still increased in volume, but substantially (40%) less than in the control trial (Δ0.27 mm^3, p=0.05). Moreover, the increase in choroid area was completely abolished (Δ0.03 mm^2, p=0.13). LBNP caused a reduction in intracranial pressure, which remained below the -6° head down tilt value for 8 hours while the device was on and returned to normal after the device was switched off (-6 deg, 8am, 14.5±5.0; 8 hrs LBNP, 11.6±3.8; 8pm 13.6±4.4mmHg).
Eight hours per day of low-level LBNP substantially attenuates the choroid expansion associated with 3 days of strict -6 deg head down tilt bedrest. These data provide evidence that low-level LBNP maybe an effective countermeasure for Space flight–associated neuro-ocular syndrome.

Lawley JS *et al.* (2017). *J Physiol* 595, 2115-2127.

Anderson AP *et al.* (2015). J Appl Physiol 120(8), 939-46

Petersen LG *et al.* (2018). *J Physiol* 597, 237-248.

We would like to acknowledge all the volunteers, technical and medical staff for supporting this challenging experiment.

Where applicable, the authors confirm that the experiments described here conform with the Physiological Society ethical requirements.

C13

Compared to magnetic resonance imaging, the creatine (*methyl-d_3*) method overestimates the loss of total skeletal muscle mass following 7 days of whole-body unloading

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Exposure to a micro-gravity (μG) environment, particularly in the absence of counter measures, is known to induce a loss of skeletal muscle mass and function. We have recently used supine whole-body unloading on a hyper-saline filled water bed (hyper buoyancy floatation, HBF) as an analogue of microgravity (μG) and demonstrated an average ~1kg loss of total muscle mass after 7 days of HBF unloading[1] as determined by whole-body magnetic resonance imaging (MRI). Here we have compared the loss of muscle determined by the gold standard (MRI) with that predicated by the creatine dilution (D_3-creatine, D_3-cr) as described by Clarke et al. (2014)[2].

Twelve healthy male subjects aged (27.3±4.2 yrs) completed the study. Six weeks prior to unloading each subject underwent a one-week control period. Pre and post the control period and at standardised time of day subjects undertook an MRI (Siemens MAGNETOM Verio 3T, Germany). For the unloading intervention period the subjects were asked to lie supine on the HBF for 7 days Subjects were allowed a maximum of 15 mins per day when they were not on the HBF (for personal hygiene etc) and were fed a controlled diet for both the control and intervention period. One day prior to and 1.5-3hrs post-unloading, further scans were performed. To estimate muscle mass using the D_3-cr, after an overnight fast, subjects provided a baseline urine sample followed by a single 60 mg oral dose of D_3-cr (two 30 mg capsules) at ~08:00 h on day 3 of the control and day 3 of the unloading period. Total urine was collected from baseline, through to the same recorded dosage time (~08:00 h) on Day 5. Measurements of urine creatine, creatinine, D_3-cr and D_3-creatinine, were performed by liquid chromatography/mass spectrometry.
No significant changes were observed in MRI-derived muscle mass before and after the control period, and D_3-cr muscle mass was similar to mean value of the two MRI measures (31.8±5.1 v 33.3±12.7 kg (mean+ SD); p=0.309). The unloading period induced a ~1kg loss of muscle mass MRI (32.19±5.33 versus 31.25±5.33 kg; p=0.0002). However, D_3-cr predicted an 8.5kg decrease in muscle mass between the control and unloading period (31.8±5.1 v 23.3+7.4 kg; p=0.0001), which was significantly different to the post-unloading muscle measured using MRI (23.3+7.4 kg v 31.25±5.33 kg; p=0.0081). The values for D_3-cr and pre and post unloading MRI were correlated (r^2 = 0.303; p=0039 and r^2 = 0.295 ; p=0.0081, respectively), but the change in muscle mass determined by MRI was not correlated with change determined by D_3-cr (r^2 = 0.01, ns)
The results showed that whilst the D_3-cr method correlated with MRI predictions of total skeletal muscle mass, compared to the gold standard measure (MRI) the D3-cr method markedly overestimated muscle loss induced by 7 days of unloading.

Morris-Paterson T et al. (2018) Differential effects on lower and upper body muscle mass following 7 days unloading on a hyperbuoyancy floatation bed. Proc Physiol Soc 41, PCB181

Clark RV et al. (2014) Total body skeletal muscle mass: estimation by creatine (methyl-d3) dilution in humans. J Appl Physiol 116(12):1605-13.

Where applicable, the authors confirm that the experiments described here conform with the Physiological Society ethical requirements.

C14

The influence of +Gx accelerations of relevance to suborbital spaceflight on the lung and pulmonary mechanics

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Introduction: Commercial suborbital space flight will soon become a reality with members of the public potentially being exposed to acceleration loads of up to +6 Gx[1]. The clinical and physiological implications of this are poorly understood[2]. The lung is highly gravity-dependent, and gas exchange and respiratory mechanics may be impaired, both of which may have clinical implications. We therefore conducted a centrifuge study to investigate how the lungs and pulmonary mechanics are affected by 2 min exposures to acceleration of up to +6 Gx.
Method: The study was conducted on a human centrifuge with all procedures approved by King's College London and QinetiQ research ethics committees. Eleven healthy participants (3 female) were exposed to 2, 4 and 6 Gx twice, once breathing air and once breathing 15% oxygen to simulate an altitude of 8,000 ft. Regional distribution of ventilation in the lung was measured using electrical impedance tomography while ventilation and peripheral arterial oxygen saturation (SpO_2) were measured continuously. In nine participants, diaphragm electromyogram was recorded to estimate neural respiratory drive (NRD). Transdiaphragmatic pressure was measured using a dual pressure transducer tipped catheter, with the proximal transducer in the mid oesophagus and the distal transducer in the stomach to allow work of breathing to be assessed (WoB). In a subset of participants arterial blood samples were obtained during Gx exposures for determination of arterial partial pressure of oxygen.
Results: The fall in SpO_2 was greater with increasing Gx level (Figure 1), an effect which was amplified when breathing 15% oxygen (P<0.05). As Gx level increased the distribution of ventilation moved from the dorsal to ventral region of the lung (P<0.05). NRD progressively increased with Gx level from 11.6 (5.0) at 1 Gx to 45.0 (21.3) % at 6 Gx (P<0.001) as did WoB (243 ± 86 to 605 ± 258 $cmH2O.s.min^{-1}$; P = 0.0013). The lowest recorded value for PaO_2 was 41 mmHg which occurred during the 6Gx exposure while breathing 15 % oxygen.
Conclusion: The Gx levels experienced during suborbital spaceflight markedly alter the behaviour of the lung and chest wall leading to significantly increase NRD and WoB. Consequently, hypoxaemia can develop and may be exacerbated by the cabin pressure altitudes currently anticipated for some suborbital flights. While the duration of Gx exposure during suborbital flights will be relatively brief, these physiological changes will be transiently stimulated and may have clinical implications for individuals with underlying cardiovascular or respiratory conditions.

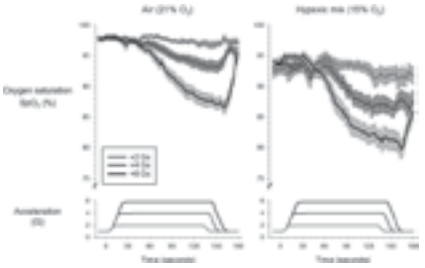

Figure 1. Oxygen saturation (SpO2) recorded during exposure to 2, 4 and 6 Gx when breathing air (Left) and 15 % oxygen (Right). A progressive decline in SpO2 occurred during all acceleration exposures. This effect was exaggerated when breathing 15 % oxygen. Values are mean ± standard error.

Blue *et al.*, (2014). *Aviat Space Environ Med 85, 721-9*

Stepanek *et al.* (2019) *N Eng J Med* 11, 1053-60

Where applicable, the authors confirm that the experiments described here conform with the Physiological Society ethical requirements.

C15

Baroreflex function in Andean high altitude natives with and without chronic mountain sickness

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High altitude native populations have adapted to the environmental stress of chronic hypoxia over generations, often demonstrating superior hypoxia tolerance. Up to a third of Andean high altitude natives, however, lose their ability to cope with chronic hypoxia and develop maladaptation syndrome chronic mountain sickness (CMS), which is associated with an increased risk of cardiovascular disease. Autonomic dysfunction has been implicated in the development and progression of many cardiovascular diseases; therefore we investigated whether autonomic function is impaired in CMS sufferers. We assessed baroreflex function in 7 Andean natives with CMS (CMS+; Hb 19.3g/dL) and 7 Andean natives without CMS (CMS-; 22.6g/dL) at their resident altitude (Cerro de Pasco, Peru; 4383m). R-R interval (RRI; Electrocardiogram), beat-by-beat arterial blood pressure (BP; photoplethysmography) and muscle sympathetic nerve activity (MSNA; microneurography) were recorded at rest and during pharmacologically induced changes in arterial blood pressure (modified Oxford method). The responsiveness (i.e gain) of the vascular-sympathetic baroreflex was determined from the slope of the linear relationship between diastolic blood pressure and MSNA burst probability, and the responsiveness of the cardiovagal baroreflex was determined from the slope of the linear relationship between RRI and systolic blood pressure. Values are presented as means (± SD) and were compared using unpaired T-tests. Resting mean arterial pressure was similar in CMS+ (83 ± 7mmHg) and CMS- (86 ± 10mmHg; $P = 0.58$). Resting RRI was higher in CMS+ (936 ± 156msec) compared with CMS – subjects (817 ± 50; $P = 0.07$). Vascular-sympathetic baroreflex gain was similar in both CMS + (-2.7 ± 1.1 %/mmHg) and CMS – subjects (-2.5 ± 1.0%/mmHg; $P = 0.72$). Cardiovagal baroreflex gain, however, was greater in CMS+ subjects (17.2 ± 6.8msec/mmHg) versus their CMS- counterparts (8.8 ± 2.6msec/mmHg; $P = 0.009$). Our data show that the responsiveness of the vascular-sympathetic baroreflex is preserved in CMS sufferers and the responsiveness of the cardiovagal baroreflex is in fact enhanced, compared to CMS- subjects. In conclusion, maladaptation to chronic hypoxia in CMS does not impair baroreflex control of blood pressure.

Where applicable, the authors confirm that the experiments described here conform with the Physiological Society ethical requirements.

C16

Impact of oxygen supplementation on flow-mediated dilation, cerebral blood flow, and oxygen delivery during ascent to 5050m

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High altitude trekking provokes reductions in flow-mediated dilation (FMD) and compensatory adjustments in cerebral blood flow (CBF) to maintain cerebral oxygen delivery. However, the impact of acute restoration of oxygen on these responses is unclear. We investigated whether oxygen supplementation (O_2), to acutely relieve hypoxemia, would reverse alterations in FMD and CBF. Healthy, male participants (aged 21-42 years) were examined at 1400m (baseline), prior to and during O_2 (after a 15-minute wash-in; target oxyhaemoglobin saturation of 99%) at 3440m (day 4 at high altitude; n=7), 4371m (day 7; n=7), and 5050m (day 10; n=12). Duplex ultrasound was performed to assess blood flow and FMD in the brachial artery and derive CBF from internal carotid artery and vertebral artery blood flows. Oxygen delivery was calculated as estimated O_2 content × blood flow. Brachial artery blood flow was lower at all locations compared to baseline. FMD was reduced and CBF augmented only at 5050m compared to baseline (P=0.002 and 0.005). At 3440m, O_2 decreased brachial artery diameter by 5±5% (P=0.04), reduced blood flow by 44±15% (P<0.001), O_2 delivery by 37±16% (P<0.001) and improved FMD (P=0.04); however, the increased FMD was explained by the reductions in baseline diameter. No brachial artery parameters were affected by O_2 at 4371m. A reduction in brachial artery blood flow (-17±22%; P=0.03), but not oxygen delivery or diameter, occurred with O_2 at 5050m. There was a trend towards increased FMD with O_2 at 5050m (3.4±2.2% to 4.8±1.7%; P=0.07). CBF was unaffected by O_2 at 3440m or 4371m. At 5050m, internal carotid artery blood flow and CBF decreased by 9±13% (P=0.04) and 13±11% (P=0.009), respectively, with O_2; nevertheless, estimated cerebral oxygen delivery was elevated by 14±14% during O_2 (P=0.02). Collectively, these findings suggest that at initial modest altitude O_2 abrogates upper limb hypoxic vasodilation along the arterial tree (i.e. conduit and resistance arteries) during early ascent. With prolonged, more severe high-altitude exposure, O_2 alleviates hypoxic vasodilation in the upper limb and cerebral circulations whilst augmenting FMD, conceivably by reducing sympathetic vasomotor tone.

This study was supported by the Natural Sciences and Engineering Research Council of Canada, the Canadian Foundation for Innovation, and a Canada Research Chair (P.N.A.).

Where applicable, the authors confirm that the experiments described here conform with the Physiological Society ethical requirements.

C17

Global Reach 2018: High altitude acclimatisation improves neurovascular coupling in man

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Neurovascular coupling (NVC) is responsible for the close temporal and regional linkage of cerebral blood supply to local cerebral metabolic requirements. The present study sought to examine the influence of acute simulated high altitude (SHA) and high altitude acclimatisation (HAA) on NVC in seven healthy male lowlanders (aged 28 ± 8 years). NVC was assessed at three time points: sea level (344m); after 30 minutes of dynamic end-tidal forcing to simulate an equivalent altitude of ~4,300m (SHA) and after two weeks acclimatisation to the same altitude (HAA, Cerro de Pasco, Peru). Posterior cerebral artery blood velocity (PCAv) was assessed using transcranial Doppler ultrasound during five consecutive trials of 30s eyes open with standardised visual stimulation (flashing checkerboard), followed by 30s of eyes closed. The NVC response was characterised as the percent peak and average increase (relative to eyes closed) in PCAv during 25s of visual stimulation, averaged across the five trials. Distribution normality was confirmed by Shapiro Wilks W tests and data analysed using a repeated measures ANOVA. Significance was set at $P < 0.05$. SHA attenuated both peak (10 ± 2% *vs*. 18 ± 4%, $P = 0.021$) and average percent increases in PCAv (3 ± 2% *vs*. 8 ± 3%, $P = 0.015$) compared to sea level. Despite similar reductions in arterial oxygen saturation and partial pressures of oxygen and carbon dioxide, HAA increased both peak (25 ± 7% *vs*. 18 ± 4%, $P = 0.016$) and average (13 ± 5% *vs*. 8 ± 3%, $P = 0.045$) percent increases in PCAv during visual stimulation, when compared to sea level. The differential response of NVC to SHA and HAA (reduction and increase, respectively) may reflect influences of acid base status and nitric oxide availability considering their differences between conditions.

This work was supported by a Royal Society Wolfson Research Fellowship (#WM170007), grants from the Higher Education Funding Council for Wales (to D.M. Bailey) and a Canada Research Chair and NSERC discovery grant (to P.N. Ainslie)

Where applicable, the authors confirm that the experiments described here conform with the Physiological Society ethical requirements.

C18

Cardiorespiratory hysteresis during incremental high-altitude ascent-descent quantifies the magnitude of ventilatory acclimatization in healthy participants

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Maintenance of arterial blood gases is achieved through sophisticated regulation of ventilation, mediated by both central and peripheral chemoreceptors. Central chemoreceptors detect changes in CO_2 within the brainstem, whereas peripheral chemoreceptors are sensitive to changes in $PaCO_2$ and PaO_2. Respiratory chemoreflexes are of particular importance during exposure to high-altitude due to the competing influence and presence of both hypoxia and hypoxic ventilatory response-mediated hypocapnia on steady-state ventilatory drive. Large inter-individual variability exists in ventilatory responsiveness and acclimatization between individuals during ascent to high altitude, potentially affecting the development of acute mountain sickness (AMS). The relationship between ventilatory acclimatization to high-altitude and the development of acute mountain sickness (AMS) remains unclear, and no predictive test of AMS severity is available. We aimed to quantify ventilatory acclimatization in the context of high-altitude hypoxia by comparing differential ascent and descent values (i.e., hysteresis) in cardiorespiratory variables. We hypothesized that (a) the hysteresis area formed by cardiorespiratory variables during ascent and descent would quantify the magnitude of ventilatory acclimatization, and (b) larger hysteresis areas in ventilatory acclimatization would be associated with lower AMS symptom scores. We quantified the cardiorespiratory ascent-descent hysteresis areas in the pressure of end-tidal (P_{ET})CO_2 (Torr), peripheral oxygen saturation (SpO_2; %), ventilation (L/min), chemoreceptor stimulus index (SI; $P_{ET}CO_2/SpO_2$) and the calculated steady-state chemoreflex drive (SS-CD; V_E/SI) using portable devices (capnograph, peripheral pulse oximeter and respirometer, respectively) and assessed AMS severity symptoms using the Lake Louise Questionnaire in 25 healthy, Diamox-free trekkers ascending to and descending from 5160m in the Nepal Himalaya over 18 days. We found that (a) ascent-descent hysteresis was present in all cardiorespiratory variables, (b) large SS-CD responders (i.e., larger hysteresis in SS-CD) had lower AMS scores during ascent than low SS-CD responders, (c) AMS positive (3+) participants had lower SS-CD hysteresis areas than AMS negative (0-2), and (d) worst AMS scores during ascent were significantly, moderately and inversely-correlated to SS-CD hysteresis magnitude. We propose that ascent-descent hysteresis is a novel and feasible way to quantify cardiorespiratory acclimatization during incremental ascent to high altitude and may have broad utility given the high number of people who trek to altitude annually.

Where applicable, the authors confirm that the experiments described here conform with the Physiological Society ethical requirements.

C19

Changes in cerebral oxygenation and microvascular blood volume during exercise in hypoxia and possible association with acute mountain sickness

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INTRODUCTION

Acute mountain sickness (AMS) can occur in people who ascend above 2500m[1]. The underlying pathophysiology of AMS is not completely understood. Altered autoregulation of cerebral blood flow (CBF) and/or impaired brain oxygenation during hypoxic exposure may be involved in AMS occurrence[2]. Previous studies did not observe any difference in CBF between symptomatic (AMS+) or asymptomatic (AMS-) subjects at rest[3]. Since exercise affects CBF and oxygenation in normoxia, and hypoxia can exacerbate these modifications[4], the aim of this study was to evaluate changes in cerebral oxygenation and microvascular blood volume during exercise in normobaric hypoxia and investigate possible association with AMS occurrence at high altitude.

METHODS

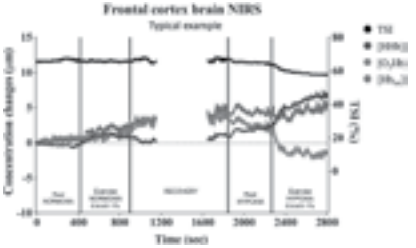
Twenty-two (15 men; 7 women) healthy young subjects (26±4yrs) were recruited for the study. Each participant completed the following exercise tests on a motorized treadmill: i) an incremental exercise to exhaustion (INCR); ii) two 8-minutes constant-speed exercises (CSE) at moderate intensity (below gas exchange threshold), one in normoxia (NORM) and the other in normobaric hypoxia (FiO_2=0.13; HYPO). Breath-by-breath $V'O_2$, $V'CO_2$, and V'E were measured by metabolic cart. HR was measured using chest band. SpO_2 was measured by finger pulse oximeter. Cerebral frontal oxygenation (HbO_2), deoxygenation (HHb), and microvascular blood volume (Hb_{tot}) were obtained by near-infrared spectroscopy (Fig.1). Occurrence of AMS, defined as a Lake Louise Scale score equal or higher than 3, was evaluated in the 24 hours following the arrival at Gnifetti hut (3647m), reached by cable car and two hours hiking.

RESULTS

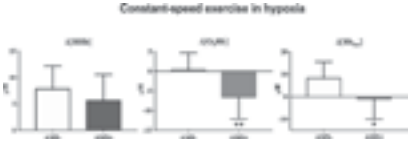
During INCR, $V'O_{2peak}$ was 3.30±0.87L/min, corresponding to 48.8±8.5ml/kg/min. During CSE, subjects exercised at about 40% of $V'O_{2peak}$. V'E and HR were significantly higher in HYPO (40.9±7.6L/min and 141±15bpm, respectively) vs. NORM (34.5±5.8L/min and 117±15bpm). SpO_2 significantly decreased during HYPO (75±5%). As for cerebral NIRS-derived parameters, HHb, HbO_2, and Hb_{tot} did not change from resting values in NORM, resulting similar between AMS+ (n=8) and AMS- (n=14) subjects. In HYPO, HHb significantly increased (by about 3μM) from resting values in both AMS+ and AMS- subjects (Fig.2). Hb_{tot} did not change from resting values in AMS+ subjects whereas it significantly increased (from 5.49±3.99 to 8.17±7.34μM) in AMS- subjects. HbO_2 (0.37±4.36μM) significantly decreased from resting values (1.44±2.14μM) only in AMS+ subjects.

CONCLUSION

Subjects presenting symptoms of AMS at altitude seem to be unable to both increase microvascular blood volume and maintain oxygenation at cerebral level during exercise in acute normobaric hypoxia, suggesting these changes may underpin later development of AMS. Future studies should confirm these findings and investigate the underlying mechanisms.



Typical example of cerebral NIRS-derived parameters obtained from the frontal cortex during constant-speed exercise in both normoxia and hypoxia.



HHb, HbO_2 and Hb_{tot} concentration changes at the end of constant-speed exercise in hypoxia for both AMS+ and AMS- subjects.

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Where applicable, the authors confirm that the experiments described here conform with the Physiological Society ethical requirements.

C20

Non-invasive assessment of pulmonary gas exchange efficiency in humans: Influence of altitude, exercise and chronic mountain sickness

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Pulmonary gas exchange efficiency, determined by the alveolar-to-arterial PO_2 difference (A-aDO_2), progressively worsens in a workload-dependent manner during exercise at sea-level. Following acclimatization to high altitude, pulmonary gas exchange efficiency is thought to improve (i.e., the A-aDO_2 difference is reduced) compared to acute hypoxia during both rest and exercise conditions. To avoid repeated arterial blood gas sampling, a new non-invasive

method to measure impairment of pulmonary gas exchange was validated during rest and exercise in acute hypoxia at sea level and implemented following acclimatization to high altitude in both lowlanders and highlanders with and without chronic mountain sickness (CMS). In study 1, 25 participants (10 female) completed an incremental maximal exercise test on an upright cycle ergometer in a normobaric hypoxia chamber (FiO_2=0.11). Simultaneous arterial blood gases via a radial arterial catheter and non-invasive gas-exchange measurements (GEM; using a MediPines Exchange Monitor AGM100®) were obtained in two-minute intervals. The traditional ideal A-aDO2 was calculated from arterial blood gases. Non-invasive gas exchange, termed the O_2 deficit, was calculated from the difference between the end-tidal and the calculated PaO_2 (via pulse oximetry and corrected for the Bohr effect by using the end-tidal PCO_2). At hypoxic rest and exercise, the results revealed strong correlations between the estimated and directly measured PaO_2 (r2=0.68; p<0.001; mean bias =1.01 mmHg) and O_2 deficit with A-aDO_2 (r2=0.70; p<0.001; mean bias =5.24 mmHg). In study 2, 11 lowlanders were tested following acclimatization at 3800m and 5100m, while 17 non-CMS, 14 mild CMS and 24 moderate/severe CMS Andean natives were tested at 5100m. Participants completed a staged steady state cycling exercise test with simultaneous GEM measurements. In study 2, elevations in O_2 deficit during exercise were reduced (P<0.05) at 5100m compared to 3800m in lowlanders. Although Andean natives with and without CMS also presented with increased O_2 deficit with exercise, there were no differences between groups. Our findings support the use of a new approach for non-invasive gas exchange during hypoxic exercise that is sensitive to acclimatization to high altitude.

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C21

The effect of severe and moderate hypoxia on exercise at a fixed level of perceived exertion

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Exercise performance during an acute exposure to hypoxia is impaired via a reduction in arterial oxygen content (Fulco et al. 1998). These decrements in performance during moderate hypoxia (FIO_2 0.13–0.15) are largely attributed to peripheral mechanisms (Amann et al. 2006) and in severe hypoxia (FIO_2 < 0.115) to a hypoxia-sensitive 'central' component via brain hypoxia (Subudhi et al. 2009). Central processing of the perception of effort is important in the determination of exercise intensity. The subjective rating of perceived exertion, termed RPE, is a psychophysiological concept (Morgan 1994) that centrally integrates perceptual, peripheral, experiential, and environmental sensory cues (Hampson et al. 2001). The purpose of this study was to determine the primary cues regulating perceived effort and exercise performance using an RPE-clamp protocol in severe and moderate hypoxia.
Eight male participants (26 ± 6 y, 76.3 ± 8.6 kg, 51.4 ± 8.0 $mL.kg^{-1}.min^{-1}$ VO_{2max}) completed three exercise trials in environmental conditions of severe hypoxia (FIO_2 0.114), moderate hypoxia (FIO_2 0.152) and normoxia (FIO_2 0.202). They were instructed to continually adjust their power output to maintain a perceived effort (RPE) of 16, exercising until power output declined to 80 % of the peak 30-s power output achieved. Expired gases were measured breath-by-breath to assess oxygen consumption (VO_2), minute ventilation, breathing frequency, tidal volume and end-tidal oxygen ($PETO_2$), and carbon dioxide ($PETCO_2$). Heart rate, oxygen saturation (SPO_2) and muscle tissue oxygenation (NIRS) were also measured.
Exercise time was reduced (severe hypoxia 428 ± 210 s; moderate hypoxia 1044 ± 384 s; normoxia 1550 ± 590 s) according to a reduction in FIO_2 (P <0.05). The rate of oxygen desaturation during the first 3-min of exercise was accelerated in severe hypoxia (-5.3 ± 2.8 $\%.min^{-1}$) relative to moderate hypoxia (-2.5 ± 1.0 $\%.min^{-1}$) and normoxia (-0.7 ± 0.3 $\%.min^{-1}$). Muscle tissue oxygenation did not differ between conditions (P >0.05). Minute ventilation increased at a faster rate according to a decrease in FIO_2 (severe hypoxia 27.6 ± 6.6; moderate hypoxia 21.8 ± 3.9; normoxia 17.3 ± 3.9 $L.min^{-1}$). $PETCO_2$ was reduced in severe hypoxia relative to normoxia (P = 0.015). Moderate to strong correlations were identified between breathing frequency (r = -0.718, P < 0.001), blood oxygen saturation (r = 0.611, P = 0.002) and exercise performance.
Performance time was diminished when exposed to decreasing FIO_2. Increases in breathing frequency and blood oxygen desaturation during the early stages of exercise were correlated with reductions in task performance. However, oxygen extraction at the muscle appeared to be tightly regulated to match the metabolic demand. Therefore, the primary cues for determining perceived effort relate to progressive arterial hypoxemia and increases in ventilation.

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Fulco CS, Rock PB, Cymerman A (1998) Maximal and submaximal exercise performance at altitude. Aviat Sp Environ Med 69:793–801

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C22

Cold water face immersion in healthy subjects: how a clash of autonomic pathways might contribute to triathlon deaths.

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Mid-competition deaths of triathletes during the swimming event are increasing and the cause of these deaths has not been fully elucidated. One hypothesis is that a conflict of autonomic signals [1] caused by the parasympathetic dive

response and sympathetic cold shock response trigger cardiac events that lead to death. The aim was to investigate the effects of cold water face immersion on heart rate variability (HRV) in healthy subjects.
This study was conducted using previously collected data. 39 healthy subjects (26 male) underwent 5 consecutive one-minute HRV measurements. The study was given ethical approval by the Research Ethics committee. All participating subjects gave informed and signed consent. HRV was recorded using an electrocardiogram. LabChart software and a PowerLab were used for data acquisition. The order of the experiments was: control 1, 26 Celsius water face immersion, control 2, 11 Celsius water face immersion and control 3. Each experiment was analysed in 6 x 10 second time bins (0-10, 10-20, 20-30, 30-40, 40-50 and 50-60 seconds). Data was also normalised by taking into account the heart rate [2].The results were analysed by repeated measures ANOVA.
Subjects' mean (± S.D.) age was 23.4 ± 7.1 years and mean BMI was 25.1 ± 5.2 kg m^{-2}. Results showed a significant sympathetic response (increased heart rate) in both face immersion tests in the initial 20 seconds following immersion with bradycardia over the subsequent 40 seconds (P<0.001 for both tests). For 11 Celsius immersion there were also significant changes in both sympathetic (increased normalised low frequency P<0.01) and parasympathetic signalling (decreased normalised high frequency P<0.001) within the first 10 seconds of the face immersion test.
The main findings indicate the first 10 seconds of the face immersion test could be a potential timeframe for autonomic conflict. The magnitude of changes in autonomic signalling were greater in the 26 Celsius water test, indicating that habituation may be occurring as this test was performed first. Further work is needed to investigate any effect of the order of tests and potentially any benefit of triathletes splashing their face with water before swimming, as a form of habituation, to possibly reduce any cardiac complications.

Shattock M & Tipton M (2012). *J Physiol* 590, 3219-30

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C23

The effect of various breath-hold techniques on the cardiorespiratory response to facial immersion

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Three to 5 maximal breath-holds have been demonstrated to increase subsequent apnoea time by up to 20%, likely via bradycardia and increased haematocrit (Hct) and haemoglobin (Hb) (Baković et al. 2003;Richardson et al. 2005). These responses are consistent with the mammalian dive reflex (MDR) on facial immersion in cold water. As anecdotal evidence suggests longer apnoea times from breath-hold techniques (BHT) used by free divers, the aim of the present study was to investigate the apnoea duration and cardiorespiratory response to facial immersion following different BHT.
Ten healthy males (34.5 ± 6.15 y(± SEM)) attended 5 randomised experimental visits where they were seated upright, underwent a 40 min BHT followed by a maximal breath hold challenge (MBH) with facial immersion, and a further 60 min of rest. On each visit, a finger plethysmograph and face mask (Human NIBP Nano, ADInstruments) measured continuously for mean arterial blood pressure (MABP), heart rate (HR), cardiac output (CO), total peripheral resistance (TPR), stroke volume (SV), and end tidal CO_2($ETCO_2$) O_2(ETO_2), and an antecubital cannula for venous blood sampling of Hb and Hct every 20 min and immediately after MBH. The BHT consisted of a quiet rest control followed by facial immersion in water at 30°C (CON) or 10°C (MDR), or facial immersion in water at 30°C following 15x1 min breath-hold sets with separated by a reducing recovery time (2:50 min -10s for each set; TOL)), 15 sets of increasing duration (30s +10s for each set; BUILD) with a 1 min recovery time, and 23 sets of increasing duration (20s +10s) and recovery time (40s * 9 sets, 50s * 3 sets, 60s * 11 sets; TV). MBH duration and cardiorespiratory values analysed using one- and two-way (group * time) ANOVAs, respectively. MDR lowered HR (P=0.032) and CO (P=0.056) during MBH compared to CON, increased Hb, but did not increase MBH duration. MBH duration was around 30% greater than CON in TOL (P=0.040), BUILD (P<0.001), and TV (P=0.001), despite similar HR responses. Hb and Hct during MBH was greater in BUILD vs. CON (P=0.027), whereas MABP was lower (P<0.001). TPR was greater than CON in TV and Hct was greater than CON in TOL. $ETCO_2$ was lower and ETO_2 was greater prior to MBH in TOL, BUILD, and TV compared to CON, whereas MDR had similar values to CON.
Consistent with the MDR, facial immersion in cold water produces a marked bradycardia and cardiorespiratory response, but these do not appear to improve apnoea duration. Some of these responses are also seen with various BHT routinely used by free divers. However, the most robust responses associated with increased apnoea time across all three BHT protocols appeared to be decreases in $ETCO_2$ and increases in ETO_2 prior to breath-hold, confirming that apnoea duration can be extended by manipulating blood gases, whilst suggesting that cardiac output and red blood cell mass are not obligatory.

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Richardson, M. *et al.*(2005) 'Increase of hemoglobin concentration after maximal apneas in divers, skiers, and untrained humans.', *Canadian journal of applied physiology = Revue canadienne de physiologie appliquee*, 30(3), pp. 276–281. doi: 10.1139/h05-120.

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C24

Swimming the English Channel Solo: A case study

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Hypothermia is a potential risk for long distance swimmers (1). This case study describes a 39 year old female masters swimmer successfully completing an early season crossing of the English Channel from England to France (starting at 0333hrs 9th July 2019, ambient temperature 15.8 [1.0] °C cloudy and overcast during the day, water temperature 16.0 [0.1]°C, wind 5.5 - 15.0 kn SE direction, sea state slight). The swim was completed in 16 hours 1 minute, 2 days after a big spring tide, covering a total distance of 32 NM. Within

this work, the thermal profile of the swim and lung function following the swim were documented.
Informed consent to test and present these data were given. Whole body DEXA scans (Horizon, Hologic, US) were performed twice, 6 months and 7 days prior to the swim. Flow volume loops were measured at these time points and 2 days following the swim. The swimmer had no ill health during the 6 month training period and was sedentary for the 24 hours prior to the swim. Eight hours before the start, a gastro-intestinal (GI) temperature pill was ingested (e-Celsius, France) and temperature logged every 5 minutes for 52 hours. Tepid food and drink were given to the swimmer following pill ingestion. An increase in body mass of 16.4 kg occurred in the 6 months prior to the swim, the majority of that increased mass was fat (12.81 kg), with a small increase in lean mass (3.59 kg) and no change in skeletal mass (2.7 kg). In the hour before the swim, GI temperature increased from 37.1 °C to 38.5 °C at the point of entry into the water. GI temperature peaked (38.7°C) 40 mins after entry and cooled at a rate of $0.2^{\circ}C.hr^{-1}$ for 11 hrs before stabilising at 36.5°C for the remainder of the swim. Recovery following the swim was unremarkable. Lung function was similar 6 months and 7 days prior to the swim (FVC; 6 months prior 4.25 L, 7 days prior 4.23 L, FEV_1; 6 months prior 3.55 L, 7 days prior 3.65 L and FEV_1/FVC; 6 months prior 83.53%, 7 days prior 86.29%), no complaints of breathing difficulties were made during or immediately post swim. However after 24 hours the swimmer complained of chronic wheezing and a tight chest, not improved with Salbutamol administration, reduced lung function was found 48 hours post swim (FVC; 3.74 L, FEV_1; 2.52 L, FEV_1/FVC; 67.11%). Symptoms were resolved with a 5 day course of oral steroids. Deep body temperature of Channel swimmers does not always reduce to levels considered hypothermic. Their tolerance likely results from their greater mass and fat percentage, having adequate fitness and fatness to generate and store heat as well as insulating against the cold. However, it is not clear why deep body temperature rose so quickly in the hour before the swim, the only explanation offered was nervousness. In addition, other factors affecting airway health need careful examination following long distance swims, even in those who are initially asymptomatic.

Pugh LGC, Edholm OG (1955) The Physiology of Channel swimmers. *Lancet* 2: 761-768

Where applicable, the authors confirm that the experiments described here conform with the Physiological Society ethical requirements.

C25

The Sacrificial Effect: Paradoxical Seal-Like Human *Empty Lungs* Deep Diving

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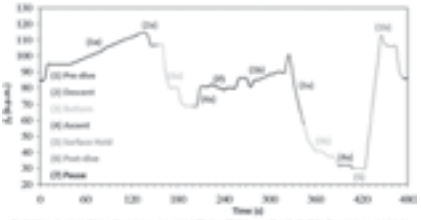
Humans dive on full lungs because the lungs contribute largely to the O_2 budget. This dependence, however, increases exposure to a range of diving risks, e.g., compression narcosis, decompression illness & hypoxia-of-ascent (HoA). Contrarian to this strategy, despite a greater-than-average lung capacity & WR-level deep diving performance capabilities, this author managed to successfully switch to the *kill-proof* animalesque expiration diving (ED) strategy without adversely affecting performance, suggesting O_2 stores are not the *be all* of successful diving & that a *less is more* sacrificial approach may potentially offer latent performance & safety benefits.[1] Here, I report on controlled trials comparing ED vs. inspiration diving (ID) on my person & involving natural (unassisted), brief (~95 sec) & shallow (15m) dives. ED involved passive expiration to functional residual capacity (FRC) & permitted freefall; ID Involved inspiration to vital capacity (VC) & required swimming to near the bottom to overcome buoyancy; swimming was only undertaken when necessary. To ensure fatigue-free performance only six dives were performed at any one time. The following physiological parameters were measured: mean±s.d. end-dive, end-expiratory O_2 gas fraction ($\%F_{EE}O2$; ±0.03%, *Aspida, Analox*), mean±s.d heart-rate (f_H; *Galileo/Polar Apnea, Uwatec,* ±1bpm) as a proxy of systemic vascular tone, blood flow distribution & O_2 consumption rate. Best-in-class outcomes are shown (Fig.1). Several major & minor mechanisms are at play to account for these paradoxical performance despite sizable differences in body O_2 stores, of which the most salient are discussed. ED result in a more prompt, pronounced & sustained DR, with bradycardia persisting throughout the ascent & for some time thereafter, fortuitously, when O_2 levels are at their lowest & need replenishing (Fig. 2). ED anapyrexia[2] results in enhanced blood O_2 extraction at the lungs at low O_2 tensions, which would slow hypoxia. Minimal lung re-inflation & inhibition of the vasodepressive pulmonary vagal inflation reflex (VPVIR), would circumvent decompression-induced hypoxia & risk of loss-of-consciousness.[3] In contrast, ID result in an anticipatory O_2-consuming work response that oppose, delays & weakens the O_2-conserving DR. Excessive buoyancy during descent requires taxing counter-swimming & is compounded by a relatively low efficiency stroke, resulting in rapid depletion of O_2 stores. In all cases, lung re-inflation unclamps the circulation & reverses the DR, commensurate in magnitude with the absolute amount & rate of lung inflation; aggravated upon nearing the surface. Blood emenating from (warm) working muscles may further restrict the use of the lungs as an O_2 source & heat-aggravate cerebral hypoxia. The ED strategy may enhance performance & safety in divers with a strong DR using a natural diving style.

Typical heart-rate trace comparing inspiration vs. expiration dive strategy series of ~90 sec & 15m depth & resorting to a natural swimming stroke.



Higher resolution heart-rate trace comparing inspiration vs. expiration dive strategy –90 sec & 15m depth & resorting to a natural swimming stroke.

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High-Pressure Nervous Syndrome and divers' wellbeing evaluation in operational setting

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Introduction: When divers are compressed to water depths greater than 150 meter sea water (msw), symptoms of high pressure neurological syndrome (HPNS) might appear due to rapid increase in pressure on the central nervous system during compression (1). The aim of this study was to first operate a new computerized tool, designed to monitor divers' wellbeing and cognitive function, and to record the results (2). The second aim was to evaluate the feasibility and validity of the Physiopad software and HPNS questionnaires as a new tool for monitoring divers wellbeing in an operational setting, including sensible visualization and presentation of results.
Methods: The Physiopad was operated onboard Deep Arctic (TechnipFMC Diving Support Vessel). The diving work was performed between 180-207 msw. The data from 46 divers were collected from the HPNS questionnaires, Hand dynamometry test, Critical Flicker Fusion Frequency test (CFFF), Adaptive Visual Analog Scale (AVAS), Simple Math Process (MathProc test), Perceptual Vigilance Task (PVT) and Time Estimation Task (time-wall).
Result: Diver's subjective evaluation revealed different symptoms, possibly also HPNS related, which lasted between 1 to 5 days in storage, with the common duration being 1 day. The results from Physiopad battery testing showed no signs of significant neurological alteration.
Conclusion: The present study showed the feasibility of using the computerized test battery to monitor saturation divers wellbeing at work. The HPNS battery and Physiopad software could be an important tool for monitoring diver's health in the future. This tool was not used during Bahr project to operationally evaluate any HPNS effect on divers as data analysis was performed post-project.
Keywords: HPNS, saturation diving, central nervous system, neuropsychology, arousal.

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The authors wish to thank Andy Butler and TechnipFMC office and project management in Aberdeen for facilitating the offshore data collection and providing the logistic. We would like to deeply thank all the Divers, Medics, Life Support personnel, Electrical Dive Technicians and crew on Deep Arctic DSV for their friendly contribution and support.

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The impact of trait and state anxiety on physical performance in heat and hypoxia.

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Anxiety incurs debilitating psychological and physiological stress which inhibits both physical and cognitive performance and can be manifested in two forms; state and trait. State anxiety is short-lived and reflects a transitionary emotional state, whereas trait anxiety is a stable characteristic reflecting an anxious predisposition. Extreme environmental conditions such as heat stress and hypoxia also induce physiological and psychological stress decreasing physical and cognitive performance. However, large inter-individual variability in response to exercise in the heat and in hypoxia have been reported. This study aimed to investigate the impact of trait anxiety independently, and in combination with state anxiety, on physical performance in the heat and in hypoxia. Following ethical approval from Loughborough University, 28 healthy males (mean ± SD; 22.1 ± 2.9 years) were recruited, in which 14 were allocated to the heat stress group (35°C, 50% relative humidity (RH)) and 14 to the hypoxic stress group (0.010 FiO_2, 21°C, 50%RH). Participants in each group completed one familiarisation session and two experimental trials. For the heat group this included: 1) heat stress with no state anxiety and 2) heat stress with state anxiety; and for the hypoxic group this included: 1) normobaric hypoxia with no state anxiety and 2) normobaric hypoxia with state anxiety. State anxiety was induced using a mental maths protocol with accompanying environmental deception. Each condition included cycling on a bike ergometer at 70% VO_{2max} until voluntary exhaustion (EXH). Objective measures included oxygen consumption and heart rate, and subjective measures included state anxiousness and rate of perceived exertion. Trait anxiety was determined using the state-trait anxiety inventory. Results indicated a significant decrease in EXH between the state anxiety and non-state anxiety conditions in both heat (-276s,

p = 0.03) and hypoxia (-367, p = 0.002). A significant negative correlation was observed in the heat where the higher trait anxious participants performed worse (r^2= 0.48, p = 0.006). The correlation was stronger when state anxiety was induced (r^2= 0.67, p < 0.001). In hypoxia however, the relationship with trait anxiety independently (r^2= 0.07, p = 0.354) and combined with state anxiety (r^2= 0.17, p = 0.142) was weak. The results from this study indicate that independently, trait anxiety inhibits performance in heat stress but not hypoxia. Further, state anxiety reduces performance in both heat and hypoxic conditions. Therefore, it is critical that interindividual trait psychological characteristics such as anxiety are considered in understanding interindividual variability in performance research. The development of psychophysiological coping mechanisms to mitigate the impact of trait and state anxiety are warranted, particularly in extreme environments.

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Short-term heat acclimation enhances cold and heat endurance performance: A case study

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Introduction: Sporting activities are increasingly taking place in hazardous and hostile thermal environments (Brocherie et al., 2015). For example, the recent "World Marathon Challenge" requires to perform 7 marathons on 7 days on 7 continents in various terrain and climate. The first stage of the race is run in Antarctica (-20°C in February) and the second one is performed few hours later in South Africa (+35°C). Research on alternative exposure to opposite thermal stimuli to investigate the effect of parallel exposure to cold and heat is very limited (Tipton et al., 2008). Therefore, this case study aimed to investigate the effect of a short-term heat acclimation (STHA) on physiological responses and physical performance in successive cold (-20°C) and hot (+35°C) environments, likely simulating the two first stage of the competition.
Methods: The subject was a 36 years old elite female ultra-endurance athlete with a background in polar expedition. During the experiment (January), she undertook approximately 8-10 h of outdoor running sessions per week, conferring a likely natural cold acclimatization. However, she had never experienced any acute exercise or acclimation/acclimatization in hot conditions (>35°C). Therefore, a STHA, consisting of 1.0-1.5 h of low-intensity aerobic exercise at 35-40°C per day for 6 days, took place during the taper phase of the training program in an environmental chamber. Before (Pre-) and after (Post-) the intervention, she completed a 110-min time-trial in the cold (-20°C) followed after 5 h 30 min of rest by a 110-min time-trial in the heat (+35°C). Performance (distance covered), peak and mean heart rate (HR_{peak} and HR_{mean}), breathing rate (BR_{peak} and BR_{mean}), rectal (T_{rec}) and skin temperatures (T_{skin}) were continuously monitored.
Results: From Pre- to Post-, HR_{peak} and HR_{mean} decreased by -4.1% and -5.3% during exercise in the cold, while $BR_{peak/mean}$ did not change. Peak and mean T_{rec} dropped by -0.8% and -1.6% in the cold, respectively, after STHA. However, while mean T_{skin} decreased (-3.6%), Peak T_{skin} increased (8.5%). Time-trial performance in the cold improved by 12.2% after intervention. After STHA, performance in the heat also improved (+12.7%) and was accompanied by the generally reported physiological adaptation: decreases in HR (-3.1% and -2.7% for HR_{peak} and HR_{mean}), BR (-24.6% and -23.3% for BR_{peak} and BR_{mean}), T_{rec} (-1.3% for both peak and mean values) and T_{skin} (-2.4% and -5.2%). Time to T_{rec} of 39°C increased from 37.5 min to 102.5 min from Pre- to Post-.
Conclusion: The universally accepted existence of heat acclimation/acclimatization was confirmed by the present findings. The novelty of this case study is that STHA (in addition to natural cold acclimatization) permits to improve performance in both cold and hot environments via hypothermic adaptation.

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Does 5-days heat acclimation reduce cardiovascular drift and improve VO_2max performance in hot and cool conditions?

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Introduction
In a hot environment, the progressive rise in heart rate (HR) and fall in stroke volume (SV) during prolonged exercise (cardiovascular drift, CVdrift) is associated with a reduction in maximal oxygen uptake (VO_2max) (Wingo et al., 2005). Ten days of heat acclimation (HA) has been shown to increase plasma volume (PV) and maximal cardiac output (Q_{max}), plus improve VO_2max performance in hot and cool conditions during cycling exercise (Lorenzo et al., 2010). This study aimed to determine the effect of 5 days HA on CVdrift and subsequent VO_2max performance during running, in hot and cool conditions. We hypothesised that 5 days HA would reduce markers of CVdrift (i.e. HR, SV and Q; thermoregulatory strain [rectal (T_{core})] and perceived exertion (RPE) during running at a standardised workload, thus enhancing VO_2max performance in hot and cool conditions compared to PRE-HA.
Methods
Ten trained middle-distance runners (VO_2max>50ml×kg×min^{-1}) will perform a standardised exercise test (running for 30-minutes, 9kph/2% gradient) in hot [40°C, 40% relative humidity (RH)] and cool (15°C, 40% RH) conditions, followed immediately by a VO_2max test PRE and POST 5 days consecutive HA (90-min controlled hyperthermia, Tre >38.5°C, in 40°C, RH 55%). Q will be estimated from VO_2 via=VO_2/arteriovenous O_2 content difference from breath-by-breath indirect calorimetry (Vmax Vyntus, Carefusion).
Results
Preliminary data (n=4) indicates that typical HA adaptations occured, with resting T_{core} and HR decreasing from day 1 to 5 of HA (37.2 ±1.1 vs 36.9 ±0.9°C and 54 ±6.2 vs 50 ±4.8 beats×min^{-1}). After 30-min standardised exercise, HR was lower POST-HA compared to PRE-HA in hot conditions but

not in cool conditions (157 ±8.01 vs 150 ±6.5 and 128 ±6.5 vs 129 ±4.8 beats×min^{-1}, respectively). HA attenuated the rise in T_{core} in hot conditions but not in cool conditions (38.9 ±1.1 vs 38.5 ±0.6°C and 38 ±1.2 vs 38 ±0.9°C, respectively). SV and RPE were reduced in cool and hot conditions POST-HA compared to PRE-HA (Q: 18.9 ±3.7 vs 17.1 ±1.5 L/min and 22.1±2.1vs21.5±2.5L/min;SV:153.2±13.7vs134±25.6mL/min and 153 ±26.7 vs 145 ±26.9 mL/min; RPE: 12 ±0.6 vs 11 ± 0.6 and 14 ±1.5 vs 11 ±0.6, respectively). 5-days HA increased VO_2max in cool conditions (3%) but not in hot conditions. At VO_2max, Q and SV were greater POST-HA compared to PRE-HÂ in cool and hot conditions (26.43 ±3.2 vs 24.88 ±3.5 L/min and 147.9 ±15.8 vs 134.9 ±22.4 mL/min and 27.17 ±4.2 vs 26.62 ±1.5L/min and 145.5 ±27.2 vs 141.3 ±2.7 mL/min, respectively).

Conclusion

These preliminary data indicate that 5-days HA reduced some markers of cardiovascular and thermoregulatory strain, whilst improving perceived exertion during exercise heat stress, but these changes had no effect on VO_2max in the hot condition. This study is ongoing and a full set of data will be presented at the conference.

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Psycho-physiological responses to perceptually-regulated hypoxic and normoxic interval walking in obese individuals

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Obese adults enjoy perceptually-regulated walking (using perceived exertion; RPE) more than a fixed-intensity. Adding hypoxia (decreased inspired oxygen fraction; FiO_2) to perceptually-regulated walking may lead to more favourable exercise-related sensations, due to potential slower velocities for a similar level of physiological stress, which may not occur in the absence of hypoxia at a matched velocity. We investigated if perceptually-regulated interval walking session in hypoxia leads to slower walking velocities vs. normoxia, matches the degree of physiological stress, and preserves exercise-related sensations. Further, we investigated if walking in normoxia at a matched walking velocity selected in hypoxia would produce similar responses in the absence of hypoxia.

Ten obese adults (BMI=32±3 kg/m^{-2}) completed a 60-min interval session (15× 2-min: 2-min walking: resting) in hypoxia (FiO_2=13%, $HYP_{self\text{-}selected}$) and normoxia ($NOR_{self\text{-}selected}$) at a perceptually-regulated velocity (RPE=14, 6–20 Borg scale), and in normoxia at the $HYP_{self\text{-}selected}$ velocity ($NOR_{imposed}$). Velocity, heart rate, arterial oxygen saturation (SpO_2), and vastus lateralis oxygenation were recorded during walking. Perceived recovery and motivation to exercise were assessed prior to each interval, while breathlessness, limb discomfort and pleasure were evaluated after. Data were averaged for each block of 3 intervals. A 2-way ANOVA analysed the main effect of condition, time and the condition × time interaction. Data are presented as mean±SD.

Compared to block 1 (6.20±0.02 km/h^{-1}), velocity was slower during block 4 (6.17±0.06 km/h^{-1}) and 5 (6.16±0.08 km/h^{-1}) and in $HYP_{self\text{-}selected}$ vs. $NOR_{self\text{-}selected}$ (6.17±0.04 vs. 6.23±0.03 km/h^{-1}, respectively, p<0.05). Compared to $NOR_{self\text{-}selected}$ and $NOR_{imposed}$, heart rate was higher in $HYP_{self\text{-}selected}$ (+6±2% and + 10±3%, respectively, p<0.05). SpO_2 was lower in $HYP_{self\text{-}selected}$ vs. $NOR_{self\text{-}selected}$ and $NOR_{imposed}$ (85±1% vs. 97±0% and 98±0%, respectively, p<0.01). Oxyhemoglobin decreased (-3±4%, p<0.01) and deoxyhemoglobin increased (+28±12%, p=0.02) from block 1 to 5, with larger changes in $HYP_{self\text{-}selected}$ vs. $NOR_{self\text{-}selected}$ (oxyhemoglobin: -4±5%, deoxyhemoglobin: + 66±10%) and $NOR_{imposed}$ (oxyhemoglobin: -18±31%, deoxyhemoglobin: +65±13%, p<0.05). Total hemoglobin decreased from block 1 to 5 (-3±1%, p=0.02). Perceived limb discomfort was lower in $HYP_{self\text{-}selected}$ (-21±4%) and $NOR_{imposed}$ (-34±6%, p<0.05) vs. $NOR_{self\text{-}selected}$. Perceived recovery decreased (-9±2%) and breathlessness increased (+9±1%, p<0.05) from block 1 to 5. Perceived motivation and pleasure were unaffected.

Perceptually-regulated interval walking in hypoxia at a lower external workload leads to larger physiological stress and lower exercise-related sensations than normoxia, which does not occur in the absence of hypoxia at a matched walking velocity.

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Human cerebral blood flow-metabolic uncoupling during acute hypoxia: A spectroscopy study

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The brain is exceptionally reliant on a pervasive supply of oxygen, which is precisely matched to neural metabolic demand, via neurovascular coupling. Hypoxic exposure (high-altitude pursuits), challenges this coupled relationship. As oxygen saturations fall, global cerebral blood flow (CBF) increases to maintain cerebral oxygen delivery, mitigating any threat of decoupling between oxygen supply and demand. However, some brain regions, including the posterior cingulate cortex (PCC) have an unexpected decrease in CBF during acute hypoxia (1) suggesting a change in regional metabolism. Investigations of the cerebral metabolic rate of oxygen in hypoxia have shown a link to an increase in the concentration of the excitatory neurotransmitter glutamate (2) which has been related to changes in neural activity and the hemodynamic response (neurovascular coupling) (3,4). We hypothesised that if neurovascular coupling is unaffected by hypoxia, the regional reductions in CBF suggesting reduced regional neural activity, should be reflected via a decrease in glutamate levels. Understanding the effects of hypoxia on neurovascular coupling, and neurometabolism is important in understanding cognitive alterations that have been reported at altitude, and in pathologic conditions involving hypoxia.

To test this, this study exposed 11 participants to a moderate hypoxic environment (Fraction of inspired oxygen [FIO_2] = 0.12) for 3.5 hoursand a procedurally matched normoxia condition

(FIO_2=.209). After 2 hours resting in an environmental chamber, participants were placed into a 3 tesla MRI scanner whilst remaining in the hypoxic or normoxic condition. Whole brain resting state microvascular perfusion was quantified by Arterial Spin Labelling and the regional resting state neurochemical environment was measured by Magnetic Resonance Spectroscopy (MRS) within the PCC. Paired samples t-tests (cluster mass FWE correction at $P<0.05$) of the ASL data confirmed a reduction in perfusion during hypoxia compared to normoxia within the PCC and right posterior temporal cortex. In contrast MRS within the PCC revealed no significant change between normoxia and hypoxia in the excitatory neurotransmitter glutamate ($p=0.9$) or any other major metabolite including creatine ($p=0.7$) and n-acetyl aspartate ($p=0.6$).
This supports our previous findings that indicate hypoxia induces a regional reduction in CBF within the PCC. Significantly, the PCC did not display a concomitant decrease in glutamate levels, or a change in other neurometabolites, that would infer a reduction in neural activity or metabolism. This challenges our present understanding of neurovascular coupling, whereby CBF is matched to demand, sustaining healthy neural functioning. Hypoxia appears to disrupt neurovascular coupling in a regionally specific manner, providing a mechanism to specific cognitive deficits experienced at altitude.

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Effects of a heated garment on physiological responses to simulated hill walking in the cold in man

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Prolonged hill walking/hiking activity places significant physical demands on participants that are influenced by the environment. Cold conditions combined with inappropriate clothing presents a serious challenge to body temperature regulation during hill walking/hiking. Advances in clothing design have enabled the development of apparel with heating apparatus to purportedly improve thermal insulation and reduce the risk of hypothermia-related events. Such investigations have not been conducted to date however. The aim of this study was to therefore examine the effect of a heated garment on the physiological responses to simulated hill walking in the cold. Four healthy males (mean±SD body mass 80±2kg, age 29±7 years) completed 2 simulated hill walks (5 km/h at 8% incline for 45 min, rest for 10 min and 4 km/h at 0% incline for 45 min; Weller et al., 1997; Ainslie et al., 2002) in 3°C in a randomised and counterbalanced manner. A fan was placed in front of participants to simulate wind. Participants wore a jacket that contained heating elements incorporated into the front and back of the jacket that were switched on (HEATED) or turned off (CONTROL) during the 2 trials. The same clothes under/in addition to the jacket were worn on both visits. Skin (lateral calf, anterior thigh, stomach, sternal, lateral upper arm and scapula) and core (intestinal) temperatures, heart rate (HR; short-range telemetry), Ratings of Perceived Exertion (RPE; 6-20) and upper and lower body thermal comfort ratings (TC; 1-9) were recorded at 5-minute intervals and were analysed using repeated measures ANOVA (main effects of time and condition).
HR increased during exercise ($P<0.05$) and was not different between HEATED and CONTROL trials (96±16 vs. 101±17 beats. min^{-1}, respectively, $P>0.05$). Lower body skin temperature was not different between HEATED and CONTROL trials (24.4±1.9 vs. 24.1±2.0 °C, respectively, $P>0.05$) but upper body skin temperature (33.5±0.5 vs. 32.1±0.4 °C, $P<0.05$) and weighted mean skin temperature were higher during HEATED (30.9±0.8 vs. 29.8±0.8 °C, $P<0.05$). The increase in core temperature was not different between trials (HEATED; 0.4±0.2 vs. CONTROL; 0.4±0.2 °C, $P>0.05$). RPE increased during exercise ($P<0.05$) and was not different between trials (9±1 vs. 9±1 AU, respectively, $P>0.05$). Lower, upper and whole-body TC decreased in the 2nd half of the protocol (all $P<0.05$). Lower body TC was not different between trials (HEATED; 4.7±0.4 vs. CONTROL; 4.6±0.5 AU, $P>0.05$), whereas upper body (5.3±0.3 vs. 4.9±0.4 AU, $P<0.05$) and whole-body (5.9±0.4 vs. 5.2±0.5 AU, $P<0.05$) TC were higher during HEATED. These findings indicate that a heated jacket increases upper body skin temperature and upper body thermal comfort, but does not affect lower body skin or core temperatures or heart rate or ratings of perceived exertion during simulated hill walking in the cold.

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Extremes of convection: Regulating thermal profile during downhill cycling using newspaper as a thermal insulator

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Introduction: Cycling uphill lead to an increase in deep body and skin temperature as higher external and metabolic workloads are required to offset gravitational resistance. Inevitably speed is reduced thereby lowering natural convection (airflow) contributing to body heat storage. Even in temperate conditions the sensation of feeling hot and uncomfortable may result which limits performance. By contrast, cycling downhill accelerates heat loss and requires lower work rates leading to cold discomfort. Historically, cyclists have behaviorally thermoregulated prior to cycling downhill by inserting newspapers up their jerseys. Yet, there is no experimental

data to support the idea that this improves thermal perception and profile; we hypothesized it would. Method: Eight trained male participants took part following ethical approval. Their mean (SD) characteristics were: age 26 (3.7) years, height 1.73 (0.1) m, mass 66.9 (10.0) kg, peak oxygen uptake (VO_{2peak}) 4.4 (0.9) L.min^{-1}, peak power output 338 (78) W). After a laboratory test of VO_{2peak} participants had two further laboratory visits completing 30-minutes simulated uphill cycling (65% VO_{2peak}188 (41) W) followed by 15-minutes of simulated downhill cycling (25% VO_{2peak} 41 (12) W) in front of an industrial fan (wind speed: 4.6 (0.1) m.s^{-1}). In one trial they inserted two standard tabloid newspapers in to their jersey (PAPER) prior to downhill cycling. The other trial was a control (NOPAPER). Whole body and torso thermal sensation (TS) and comfort (TC; both 20 cm visual analogue scale), aural temperature (T_{au}), skin temperature (T_{skin}), and newspaper mass change (ê) were measured. Data were compared using ANOVA and t-test to 0.05 alpha level. Results: After uphill cycling thermal profile was similar and participants felt *hot* (grand mean (SD) TS: 17.3 [1.4] cm), *uncomfortable* (TC: 6.2 [5.1] cm), had significantly changed T_{au}(ê1.08 [0.4] °C) and T_{sk}(31.9 [1.0] °C] °C); p<0.05 for time only. During downhill cycling the PAPER (torso) TS was higher but TC only descriptively differed (PAPER *cf* NOPAPER TS: 10.7 [1.] cm *cf* 6.9 [0.7] cm; *neutral cf slightly cool*; TC: 14.7 [0.8] cm *cf* 13.8 [1.0] cm; *comfortable cf just comfortable*). The PAPER maintained chest T_{sk}(29.5 (1.5) °C *cf* 25.6 (1.5) °C) but did not defend mean T_{sk}(p>0.05). Newspaper mass indicated some impaired sweat evaporation (êmass: 5.7 (4.9) g; p=0.01). Discussion: Downhill cycling thermal perception and local thermal profile was improved by inserting the paper. The magnitude of these effects was localized. These data support the anecdotal idea that this is an effective practice to preserve heat and thermal perception. At higher wind speeds the effects may be magnified to encompass the whole-body profile as the extent of thermal discomfort relief provided by the paper may be more salient.

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Vascular function in non-freezing cold injury patients

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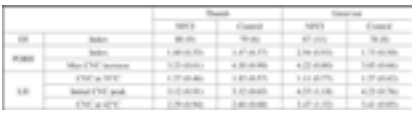
Prolonged exposure to cold and often cold/wet conditions can cause non-freezing cold injury (NFCI) in the hands and/or feet. The chronic symptoms of NFCI may last years, reducing the quality of life and limiting employability in certain occupations. The pathophysiology of NFCI is poorly understood, but may involve a combination of neural and vascular impairments. We hypothesised that the vascular responses to deep inspiration (DI), local heating (LH) and post-occlusive reactive hyperaemia (PORH) would be impaired in the Great toe and thumb of NFCI patients compared with matched Controls.
Following ethical (MODREC) approval and written informed consent, 14 NFCI patients (age [SD]: 29 [4] years; mass: 76 [7] kg; height: 1.76 [0.07] m; predicted VO_2max: 68 [8] mL.kg^{-1}.min^{-1}) and 14 matched cold-exposed Controls (29 [6] years; 78 [11] kg; 1.77 [0.08] m; 70 [11] mL.kg^{-1}.min^{-1}) undertook DI, PORH followed by LH in 24 °C ambient air. Cutaneous vascular conductance (CVC; flux/mean arterial pressure) was measured at the Great toe and thumb pad with local skin temperature clamped at 33 °C. DI protocol: participants took a rapid, deep breath to maximum inspiratory capacity and held it for 10 s followed by normal breathing, repeated three times with a 3 minute interval. The minimum blood flow during inspiration (BF_{min}) and preceding resting skin blood flow (BF_0) were used to calculate DI index: $100^*(BF_0\text{-}BF_{min})/BF_0$. PORH protocol: following a 5 minute baseline, Great toe and thumb blood flow was occluded (220 mmHg) for 3 minutes and then rapidly released. PORH index was calculated as the area under the curve during the first minute after pressure release divided by that during the last minute before cuff inflation. LH protocol: skin temperature was clamped at 33 °C for 10 minutes followed by 42 °C for 20 minutes. Between-group comparisons were conducted using independent samples *t*-tests for DI and PORH. LH was analysed using a 2-way ANOVA.
Vascular responses of the thumb and Great toe pad were similar in NFCI patients and cold-exposed Controls for each protocol (Table 1, P>0.05), thus the hypothesis is rejected. Therefore, either NFCI is not associated with vascular dysfunction, or it is possible that significant cold exposure alone alters vascular function, causing a sub-clinical condition. To investigate this, the same tests are currently being compared in a non-cold exposed Control group.

Table 1. Mean (95 % CI) index and cutaneous vascular conductance during each protocol for NFCI and Control groups



Funded by WGCC, MoD

Where applicable, the authors confirm that the experiments described here conform with the Physiological Society ethical requirements.

C35

How are you sleeping in Antarctica? One-year smartphone-based sleep monitoring pilot study at "Akademik Vernadsky" Research Base.

O.V. Shylo[1], D.G. Lutsenko[1], K.M. Danilenko[2], G.O. Babiychuk[1] and Y. Moiseyenko[2]

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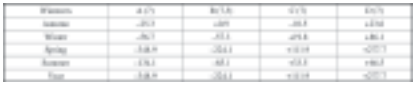
As far as the most winterers in Antarctic expeditions mentioned sleep problems its monitoring and early disturbances evaluation may be of very importance for the entire mission. There are a lot of factors disturbing sleep, but such a challenging combination that embrace the effects of fluctuations in meteorological and climatological conditions, physical inactivity, social isolation, sensory and sexual deprivation is difficult anywhere to find. Each of the factors mentioned, even separately, can influence sleep. The decline in sleep quality and quantity, in turn, may affect the performance and adaptability of the crew members. The aim of the study was to investigate the changes in sleep in winterers of 21st Ukrainian Antarctic expedition during 2016-2017 years season at the "Akademik Vernadsky" Research Base. Twelve winterers (from 22 to 63

years old, all men) participated in subjective sleep quality measures study (IDS-SR30 questionnaire first four questions were analyzed). Four of them were involved in the objective sleep measurements. Personal smartphones with installed Sleep as Android app (Urbandroid Team) were used for sleep registrations. Based on built-in accelerometer data the total sleep time (TST) and deep sleep time (DST) were calculated by the program. The differences between subjective normal and actual daily TST were defined as increments or decrements in TST. The data for the last month of stay in Antarctica (March) and data for one or two months (at winterers C and D, problems with registration) were excluded from the analysis. Data were means±SD, compared by ANOVA. Despite personal variations in subjective reports in winterers, the sum of IDS-SR30 marks increased in winter-spring time which indicates a decrease in subjective assessments of sleep quality. All 4 subjects had individual peculiarities of sleep pattern and both in TST and DST changes during the year as well. Average yearly TST were 5.96±0.5 (n=11), 6.52±0.63 (n=11), 7.73±0.85 (n=9) and 7.78±0.4 (n=10) hours in A, B, C and D winterers correspondingly. TST duration over the year decreased in A and B, but increased in C and D winterers (Table).
Thus, subjective reported sleep quality slightly declined, as well as two opposite strategies in TST changes in winterers were found in the research.

Personal decrements (-) or increments (+) in TST in winterers, hours



A-D – winterers and their subjective normal sleep time duration in hours (in brackets).

The presenting author's attendance at the Meeting is supported by IMET2000 and UNESCO Chair in Cryobiology.

Where applicable, the authors confirm that the experiments described here conform with the Physiological Society ethical requirements.

C36

Global warming: A silent threat on animal and human health

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Global warming has resulted in climate change and the rise in sea level with disastrous consequences, such as bad weather, hurricanes, wild fire, poverty, ill-health and socio-economic challenges (Olaniyi *et al*. 2013). Basically, global warming refers to the increasing average environmental (air) temperature near the earth's surface. Inter-governmental Panel on Climate Change defines climate change as a change in the state of climate that can be identified; for example, by using statistical tests, by changes in the mean and/or the variability of its properties, and that persists for an extended period, typically decades or longer. The enhanced emissions of greenhouse gases have been reported to cause this increase. Global warming has been linked with anthropogenic (human activities) and bio-geographical (natural) factors (UN's Intergovernmental Panel on Climate Change – IPCC 2007). Some alterations in practically every phase of human activity may be attributed to seasonal changes. The food, clothing, shelter, recreation, occupation, health and energy are all considerably affected by climatic surroundings (Khasnis and Nettleman, 2005). Animals, especially those reared under extensive management system and wild ones; do not have developed artificial means of self-protection, rendering them to be more adversely affected. Studies have shown that man, his livestock and plants thrive best within definite and specific condition of ambient temperature, relative humidity and sunshine; other factors affecting man are the composition and movement of atmosphere or water in which he lives. Any deviation outside the normal limit decreases efficiency, lowers the rate of production and induces potential changes in specific body parameters, which may further result in suffering or death and destruction of species (Huntington, 1978). Painfully, developing nations, such as Nigeria, have been described as ill-prepared to face the challenges. The resultant adverse effects of global warming are, apparently, serious in developing countries (Taha, 2016). These may be attributed to poor financial status and low mitigating and adaptive capacity of these nations. Adoptable preventive measures have been suggested and on-going investigations on therapeutic measure to combat vagaries in climate are crucial in order to enhance survival on the earth. Climate change involving global warming is of great global concern and collaborative efforts are prerequisite for successful combat of its negative impact on man and animals.

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I wish to acknowledge my project supervisors in persons of Prof. J. O. Ayo, Prof. M. M. Suleiman and Dr. V. O. Sinkalu for thier contributions to the success of this work. I also appreciate all the teaching and technical staff of the Department of Veterinary Physiology, Ahmadu Bello University, Zaria.

Where applicable, the authors confirm that the experiments described here conform with the Physiological Society ethical requirements.

C37

Long- and short-term cold adaptations affect erythrocyte population in rats of different ages.

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Both long- and short-term contact with the cold environment may cause stable cold adaptation/s. In spite of possible different background mechanisms of such adaptations development, the initial processes are of the same nature and involve autonomic nervous system activation to provide the growing metabolic demands of the body. The later with the corresponding generation of reactive oxygen species and hormonal changes increase the load on the red blood cells (RBCs) and may affect their mechanical stability/shape. The aim of the work was to study the effect of long- and short-term cold adaptation (LTCA and STCA, respectively) on osmotic fragility (OF) and the sphericity index (IS) of RBCs in rats of different ages. White outbred male rats of 6, 12 and 24-month-old were used in the experiments. LTCA was

achieved by keeping animals under varying ambient temperature (1-7°C) for 5 weeks under free-running light conditions (food and water *ad libitum*). For STCA development animals were subjected to -12°C or 10°C environmental temperatures (STCA-12 and STCA+10, respectively) for 2 days (totally 9 cold effects per day) as follows: the first 15 minutes of each hour during daytime the animals were exposed to cold, the next 45 minutes they were left to themselves at 26°C with free access to water and food. RBCs were subjected to hypotonic hemolysis. The resulted OF curves, obtained by the method of small-angle-scattering, were used for determination of the RBCs distribution by the IS, which in turn characterizes the shape of the cells. The shapes of the RBCs that predominated in the certain SI intervals were distributed as follows: (1...1.05) – spherocytes, (1.06...1.5) – stomatocytes, (1.5...2) – normal and (2.1...3) – flattened discocytes. Data were means±SD, compared by ANOVA. No significant changes in OF were found among control and rats after STCA+10 in all age groups. Osmotic fragility decreased (compared to control) in 6 and 24-month-old rats after STCA-12 (from 0.52±0.01 to 0.48±0.01 (p=0.05) and from 0.5±0.01 to 0.47±0.01 (p=0.04), respectively, n=5) and after LTCA in 24-month-old rats (from 0.5±0.01 to 0.46±0.01 (p=0.01), n=6). Moreover, both types of cold adaptations modify RBCs distribution by IS in rats of all ages towards the increase in the percentage of normal and flattened (highly resistant forms) discocytes, and the decrease in the number of cells the shapes of which are close to spherical one (least resistant forms).

Thus, under cold pressure the RBCs OF decreased in 6 and 24-month-old animals after STCA-12 as well as in 24-month-old animals RBCs after LTCA. Moreover, both LTCA and STCA improve the RBCs population condition by "washing" out the least resistant RBC forms from the circulation.

The presenting author's attendance at the Meeting is supported by IMET2000 and UNESCO Chair in Cryobiology.

Where applicable, the authors confirm that the experiments described here conform with the Physiological Society ethical requirements.

C38

Cardiopulmonary acclimation using intermittent normobaric hypoxic exposure with and without exercise.

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Introduction:

High altitude pulmonary oedema (HAPE) in severe cases is fatal. Altitude, ascent rate, degree of pre-acclimatisation and individual susceptibility determine overall risk of HAPE. Combining a fast ascent rate with high individual susceptibility leads to a HAPE incidence of 60% at 4559m (Bartsch et al. 2003). Elevated pulmonary artery systolic pressure (PASP) precedes the development of HAPE (Maggiorini, 2006). Normobaric hypoxia has been used as a method of identifying those individuals susceptible to HAPE using PASP as a marker of risk (Dehnert et al. 2005). However, acclimation or pre-acclimatisation techniques such as intermittent hypoxic exposure (IHE) and training (IHT) have not been evaluated as a method to reduce HAPE susceptibility.

Methods

PASP changes were assessed using IHE (n=10) and IHT (n=12) in comparison to a sea level control group (SLC, n=10) using 5 days of 5 hours.day^{-1} of normobaric hypoxia (4800m, day 1, 4, 5 and 4300m days 2 & 3). Echocardiography was used to measure PASP at four separate time points on days 1 and 5 (T1 = 0, T2 = 1.75hr, T3 = 4hrs, T4 = 5hrs). IHT and SLC walked at a gradient of 10 – 15%, carrying a 10kg load for 90 minutes from 2.5hrs - 4hrs, at relative intensities of 40 – 70% of altitude specific VO_{2max}. The Ministry of Defence Research Ethics Committee approved the study. A three way repeated measures ANOVA (RMANOVA) was used to assess group differences and interactions, with effect size assessed using partial eta squared (η^2). Independent and paired *t-tests* with Cohens *d* were conducted *post-hoc* to establish specific differences within and between groups.

Results

RMANOVA revealed a *large* significant interaction between day, time and group (P = 0,001, η^2 = 0.196) indicating each group produced a different pattern of response. SLC had consistent PASP at all time points comparing day 1 and 5. Hypoxia caused a significant increase for both IHT and IHE on day 1 (mean ± SD: 22 ± 4 mmHg, P = 0.000, *d* = 5.63; 19 ± 7 mmHg, P = 0.000, *d* = 2.82). There was no significant effect of exercise on PASP for IHT. There were no significant changes in PASP comparing day 1 and 5 for IHE (P = 0.863, *d* = 0.06). In contrast, IHT showed a significantly reduced PASP at the end of day 5 compared to day 1 (mean ± SD: 5 ± 4 mmHg, P = 0.008, *d* = 1.33).

Conclusion

Acclimation using IHT has the potential to reduce HAPE susceptibility for individuals with previous history or military personal exposed to considerable terrestrial altitudes without sufficient time to acclimatise. Further research is required to fully establish its efficacy at terrestrial altitude and understand the mechanisms responsible for differences in PASP response to IHE and IHT.

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The authors wish to thank all participants and the British Army Everest team for their time and dedication to the study.

Where applicable, the authors confirm that the experiments described here conform with the Physiological Society ethical requirements.

C39

Response to environmental stress by water truck pushers in Nigeria

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Water vending is a significantly lucrative business laden with stressful and strenuous activities making it to be implicated in oxidative damage, lipid peroxidation and the susceptibility of erythrocytes to haemolysis. The study was designed to investigate the degree of erythrocyte osmotic fragility (EOF), Malondialdehyde (MDA) concentrations, Superoxide dismutase (SOD), Catalase and reduced glutathione (GSH) activities which detoxifies the superoxide radicals produced from rigorous musculoskeletal activity such as water vending among water vendors and non-water vendors. Copies of questionnaire were randomly distributed in the study area to a total of 192 subjects of which water truck pushers (experimental group) were 96 and non-water truck pusher (Control group) were also 96 individuals. 5ml of blood was obtained from the median cubital vein via venepuncture. Ethical clearance was obtained from the health research ethics committee of A.B.U, Zaria, Nigeria. 3ml of samples stored in EDTA bottles were used for EOF whereas serum samples obtained from 2ml of centrifuged blood were assayed biochemically for MDA concentration using TBARS assay and antioxidant enzymes activities. Data obtained were analysed using independent sample T-test and cross Tab for descriptive statistics. The result showed a significant ($P < 0.05$) increase in serum MDA concentrations of the experimental group with a value of 230.33 ± 4.75 nmol/ml compared to control group with a recorded value of 215.61 ± 3.59 nmols/mL. EOF of water vendors showed significant ($P < 0.05$) increase with a value of 85.83 ± 0.37% when compared to 78.69 ± 0.53% for non-water truck pushers at 0.4% concentration of NaCl solution. Significant increase in SOD activity (15.26±0.84 IU/L) among the water vendors compared to the control (11.48±1.16 IU/L). Catalase activity shows statistically significant ($P < 0.05$) increase in water vendors with a value of 63.33 ± 1.08 IU/L compared to the non-water vendors with corresponding value of 56.19 ± 1.66 IU/L respectively and a statistically significant decrease in GSH activity (29.15±0.14 IU/L) among the water vendors compared to the control (38.93±0.95 IU/L) was recorded. In conclusion, these findings suggest that increase exists in the EOF and MDA concentration of water vendors and a redox imbalance had occurred in the antioxidant enzymes as such the need to mitigate this effect.

C

D

Plate C shows packed truck queued to get filled. D shows water vendors bargaing to sell water in bulk to a customer.

(All Plates other than C were captured using Tecno Mobile T421 model with the following specifications; Dimension 126.5*57*11.17mm, Display screen: 2.8", Camera (rear): 2MB sand 240*320 Resolution).

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The authors wish to thank Mallam Bala Mohammed of Department of Human Physiology, Faculty of Basic Medical Sciences, Ahmadu Bello University, Zaria, Nigeria and Mr Olu Ayebusi of Chemical Pathology Department, Faculty of Basic Clinical Sciences, Ahmadu Bello University, Zaria, Nigeria for their assistance throughout the period of this work

Where applicable, the authors confirm that the experiments described here conform with the Physiological Society ethical requirements.

C40

Heat exposure stress alters gastrointestinal motility and intestinal fluid accumulation in Wistar rats

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Global warming is currently a major challenge facing living and non-living things in the tropics and subtropics. The gastrointestinal (GI) tract is particularly sensitive to stressors including hyperthermia. Our study therefore, aimed at examining how gastrointestinal motility can respond to sub-chronic heat exposure (HE) stress in *Wistar* rats.

Rats (male, 110-130 g, n=5) were randomly assigned to two groups; Control in a normal room temperature at 30°C and 70-80% humidity, and a HE exposed to heat at 40°C and 70-80% humidity 3 hours daily for 14 days in a thermal controlled

room. Rectal temperatures (RT) were taken daily, before and immediately after HE with a mercury thermometer. After 14 days, rats were 18-hour fasted prior GI motility experimental procedures. Gastric emptying (GE), intestinal transit (IT) and intestinal fluid accumulation (IFA) were assessed by the methods of Droppleman *et al*., 1980, Suchitra *et al*., 2003, Sisay *et al*., 2017 respectively. Colonic motility was also, assessed by colonic bead expulsion (CBE) time, Camilleri and Linden, 2016. Values are means ± S.E.M., compared by t-test and ANOVA, as applicable to data.

Our HE resulted in hyperthermia as evidenced by increased post-exposure RT; compared to control, RT in HE rats increased throughout the HE periods (e.g. Control: 35.30±0.06 vs. HE: 38.54±0.07°C Day 1, P < 0.05). GE (Control: 40.03±4.78 vs. HE: 38.92±2.74 %, P < 0.05) and IT (Control: 27.19±2.90 vs. HE: 60.17±2.58 %, P < 0.05) was increased. Also, IFA was increased (Control: 10.73±0.60 vs. HE: 16.07±0.54 %, P < 0.05) while CBE time was decreased in the HE rats (Control: 23.24±1.89 vs. HE: 14.28±0.66 minutes, P < 0.05).

These data indicate that sub-chronic HE increased GI motility and intestinal fluid accumulation as evidenced by an increased ST and RT, GE, IT, IFA and decreased CBE time.

Table 1. Heat exposure (HE; 40 C; 70-80% humidity) induced increase in Rectal temperature after a daily 3-hour Heat exposure for 14 days.

Day 1	[illegible]	[illegible]	[illegible]
Day 2	[illegible]	[illegible]	[illegible]
Day 3	[illegible]	[illegible]	[illegible]
Day 4	[illegible]	[illegible]	[illegible]
Day 5	[illegible]	[illegible]	[illegible]
Day 6	[illegible]	[illegible]	[illegible]
Day 7	[illegible]	[illegible]	[illegible]
Day 8	[illegible]	[illegible]	[illegible]
Day 9	[illegible]	[illegible]	[illegible]
Day 10	[illegible]	[illegible]	[illegible]
Day 11	[illegible]	[illegible]	[illegible]
Day 12	[illegible]	[illegible]	[illegible]
Day 13	[illegible]	[illegible]	[illegible]
Day 14	[illegible]	[illegible]	[illegible]

Values are mean ± S.E.M of 5 rats. Where RT = Rectal temperature. a and * = p < 0.05 values differ significantly from control group.

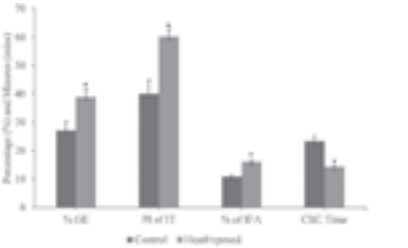

Fig. 1. Heat exposure (HE; 40 C; 70-80% humidity) induced increase in **Percentage GE, IT and IFA, and CBE** (minutes) after a daily 3-hour Heat exposure for 14 days. Values are means ± S.E.M of 5 rats. Where GE = Gastric emptying; IT = Peristaltic index; IT = Intestinal transit. IFA = Intestinal fluid accumulation and CBE = Colonic bead expulsion. * = $p < 0.05$ values differ significantly from control group.

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Where applicable, the authors confirm that the experiments described here conform with the Physiological Society ethical requirements.

C41

Management of Heat Stress in Sport; Are Recommendations Suitable?

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Increasing incidence of extreme weather events has placed the management of heat stress at sporting events under the spotlight. Guidelines to manage and mitigate heat stress for those competing in and attending outdoor sporting events are increasingly important and pertinent. Our aim was to determine the guidelines currently available internationally. The secondary aims were to assess the specific advice for minimising heat stress and to determine the suitability of guidelines for both the specialist and wider populations.

A search of government, sports medicine, and international sporting events organisations in the Anglosphere and host countries for previous and upcoming international sporting events (IOC, FIFA etc) and the six most popular outdoor sports club organisations in each Anglosphere country was conducted. Websites were systematically searched using the combined terms “weather”, “guidelines”, “physical activity” and variations of, for documents containing relevant advice. Advice was categorised into 13 components, including strategies to prevent or treat heat stress, assessing environmental parameters and scientific rationale. The occurrence of recommendations for each category was quantified using NVivo 12 software.

Websites of 198 organisations in 37 countries were searched. No guideline documents were found for 74% of the websites; 133 documents were retrieved from the remaining 26%. 87% of the 133 guidelines, listed strategies to prevent heat stress and 32% gave strategies for treatment of heat stress. Only 29% of guidelines referred to environmental conditions, such as which environmental parameters should be assessed and how, and/or the use of a thermal index. Of those guidelines highlighting environmental conditions, 74% provided advice specific to certain conditions e.g., stated cut-off temperature at which play should be suspended. References were provided on 22% of guidelines, mostly referring to primary scientific literature.

At present, the available guidelines are often poor and provide limited clear and consistent advice for protecting those involved in sport and physical activity in extreme heat events. Guidelines are inconsistent in the advice they give within the categories of preventing heat stress and assessing environmental conditions. Guidelines give greater emphasis on preventing heat stress over detailing practical methods for treating it. Measurement and interpretation of local environmental conditions is often not given. The use of environmental indices as a tool for determining cut off criteria for suspending or cancelling play is very limited. Guidelines lack strong supporting evidence and those that do cite scientific documents have often compromised the suitability of the

guidelines for use by non-specialist populations. Improved evidence based guidance is clearly needed.

Where applicable, the authors confirm that the experiments described here conform with the Physiological Society ethical requirements.

C42

Comparisons of core body temperature between an ingested telemetric pill and heart rate estimated core body temperature in firefighters

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Firefighters, may experience high environmental temperatures or carry out intensive physical tasks, or both, which leads to increased core body temperature and increased risk of fatalities. Hence, there is a need to remotely and non-invasively monitor core body temperature. The aim of the present study was to determine the suitability of a non invasive approach to determine core body temperature in firefighters.
Estimated (heart rate algorithm - Buller et al. 2013) and actual core body temperature (ingested telemetric pill) measures were collected simultaneously during firefighter training exercises (Average external temperature range 120 - 250 °C) on 44 firefighter volunteers (age 34.1 ± 8.4 years, body mass 82.8 ± 12.7 kg, height 177.4 ± 7.7 cm) whilst wearing personal protective equipment (PPE).
Prediction varied by individual, with no specific identifiable pattern between the algorithm values and directly measured body core temperatures. The group agreement value of Lin's Concordance of 0.74 (95% Upper 0.75, lower CI 0.73), was deemed poor. It could be seen from individual agreement data that the Lin's Concordance was variable (Min 0.11, 95 % CI 0.13 - 0.01; Max 0.83, 95 % CI 0.86 - 0.80).
From the observed associations between the two methods, the data indicated that the heart rate algorithm approach was not suitable for core body temperature monitoring in this population group, especially at the higher more critical core body temperatures seen.

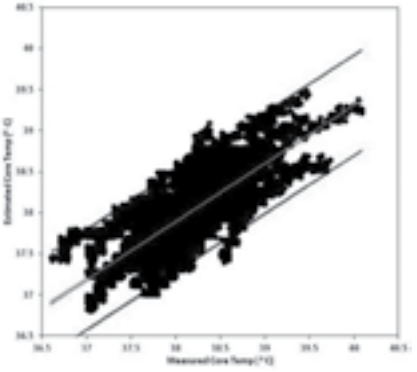

Figure 1. Group Scatter plot of CBT_{est} against CBT_m. Lin's concordance (r) = 0.742 (CI = upper = 0.749; lower = 0.734). Red lines = 95% prediction intervals; blue lines = 95% CI for line fit. Estimated Core temp = 0.708x Measured Core Temp + 10.996); r^2 = 0.584.

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C43

Minimal effect of water immersion on markers of inflammation and muscle damage after intensive exercise.

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Water immersion methods, such as cold water immersion and contrast water therapy are popular recovery interventions after athletic training and competition. Nevertheless, post-exercise cold water immersion may actually inhibit hypertrophic signalling pathways and muscle adaptation to training (1). It is has been commonly assumed that the mechanism of impaired training adaptation is mediated by blunted inflammatory responses to muscle-damaging exercise, although this assumption has been questioned by recent data (2). A weakness of previous studies is omission of active recovery in water immersion interventions, which would arguably be utilised in addition to water immersion by athletic populations. The aim of this study was to compare the influence of three water immersion methods, performed after active recovery, on inflammatory responses to muscle-damaging exercise. Nine male participants (age 20-35 y) performed an intensive exercise protocol, consisting of maximal jumps and sprinting, on four occasions. After each trial, participants completed one of four recovery protocols in a randomised, crossover design (ACT, active recovery only, 10 min cycling; heart rate 120–140 b/min; CWI, active recovery followed by 10 min cold water immersion, 10°C; TWI, active recovery followed by 10 min temperate water immersion, 24°C and CWT, active recovery followed by contrast water therapy, 10 min alternating 10°C and 38°C in 1 min cycles). The study was conducted in accordance with the Declaration of Helsinki and approved by the local ethical review board. Venous blood samples were collected pre-exercise and 5 min, 60 min, 24 h, 48 h and 96 h post-exercise, then analysed for myocyte chemoattractant protein 1 (MCP-1) and creatine kinase (CK) using ELISA and high-sensitivity C-reactive protein (hs-CRP) using a chemiluminescence assay. Two-way repeated measures ANOVA was used to compare biomarker concentrations between groups over time. There were no differences in biomarker concentrations during exercise and recovery between groups across the six time points, however main effects of time were present for all three markers (MCP-1: $F(2.32, 18.56) = 23.1$, $p < 0.0001$; CK: $F(2.059, 16.47) = 8.74$, $p = 0.002$; hs-CRP: $F(1.07, 8.57 = 13.8$, $p = 0.005$). Tukey's post-hoc analysis of simple time effects revealed increases in MCP-1 at post-5 min versus pre in all groups except CWT. In TWI and CWI, MCP-1 was still elevated above pre at 60 min post-exercise. hs-CRP peaked at 24 h post-exercise in all groups. CK was elevated at post-60 versus pre in all groups and at post-24 except in CWT. Our findings suggest that use of cold or thermoneutral water immersion in combination with active recovery may slightly prolong the immediate post-exercise elevation in MCP-1 but

have minimal overall effect on markers of inflammation and muscle damage.

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C44

Rewarming methods following cold water swimming

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In the UK, outdoor swimming is an increasingly popular leisure activity. Rewarming techniques following swim events vary, from no rewarming provision other than changing facilities, the option of gentle exercise when changed and also the provision of warm jacuzzi baths are common. It was hypothesised that rewarming following swimming in cold water would be most rapid in the warm bath and exercise and shivering would be slower. In addition, it was hypothesized that thermal comfort and an indication to end the warm bath rewarming would be related to increases in skin temperature and blood flow rather than rectal temperature (T_{re}) rewarming.

Twelve participants (9 males, 3 females) gave informed consent to participate in this ethically approved study. Each participant performed self-paced (skins) swimming on three occasions in a swimming flume (water temperature 15 °C) and rewarmed following a balanced Latin square design using: clothed seated shivering Control (20 °C air temperature); clothed treadmill exercise (20 °C air temperature, walking at 2.5 km.hr^{-1} for 10 min increasing to 3.5 km.hr^{-1} thereafter), and a warm bath 38-40 °C. T_{re}, expired gases, heart rate and visual analog scales were measured throughout the cooling and rewarming phases. Finger skin blood flow and mean unweighted skin temperatures (upper arm, chest, thigh and shin) during the rewarming phase. Participants were asked to indicate when they felt they were warm enough to stop the rewarming protocol. Rewarming was continued until T_{re} had returned to within 0.3 °C of the baseline.

The deep body rewarming rate was faster in the warm bath (mean [SD] 1.55 [0.93] °C.hr^{-1}) compared to the Control (0.66 [0.22] °C.hr^{-1} p=0.012) and the exercise condition (1.50 [0.56] °C.hr^{-1} p=0.001). The release of vasoconstriction occurred at a significantly lower T_{re} during the warm bath (36.28 [0.31] °C), than during exercise (36.68 [0.63] °C p=0.047), or Control (36.73 [0.47] °C, p=0.036), and at a significantly higher skin temperature during the warm bath (33.24 [1.45] °C) compared to Control (30.83 [0.86°] C, p<0.001) and exercise (30.45 [1.30] °C, p=0.004). Participants indicated that they were warm enough to halt the rewarm protocol at lower T_{re} in the rewarming bath (36.30 [0.32] °C) than exercise (37.04 [0.44] °C, p=0.022) or Control (36.84 [0.44] °C p<0.001).

During bath rewarming, the release of vasoconstrictor tone occurs in response to increasing skin temperature, despite a reduced T_{re}. This may be due to the lag in T_{re}. However, in a 'field' setting, using warm baths to rewarm cold water swimmers may result in premature cessation of the rewarming protocol due to the increase in skin temperature and concomitant return of thermal comfort prior to T_{re} rewarming.

Mr Geoff Long and Mr Danny White for their technical support and all the volunteers for their participation.

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Follow-up of pulmonary diffusion capacity in elite swimmers before and after training sessions under indoor swimming pool conditions

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INTRODUCTION

The diffusion capacity for carbon monoxide (DLCO) provides a measure of gas transfer in the lungs (1). Swimmers have larger lungs and possess better diffusing capacity than other athletes and controls (2,3), but none study has measured the possible acute modification in lung diffusion of elite swimmers after training session. During the last decade, it has been described the presence of swimming-induced pulmonary oedema (SIPE), an uncommon occurrence which it is usually presented during strenuous long-distance swimming, altering the DLCO (4). The aim of this study is to evaluate the modifications in the pulmonary alveoli-capillary diffusion provoked by swim training over a period of time.

METHOD

The participants are 21 international competition swimmers (14 men and 7 women) from 13 to 19 years old with a training schedule of 25-30 hours of training per week. The lung diffusion changes have been measured before and after 11 training sessions. The water temperature of the pool is 27°C. The material used is a computerized spirometer (Gashorn, PowerCube Diffusion+, Niederlauer, Germany). The single-breath method was used to measure the DLCO(5). Respiratory parameters were analysed using a multifactorial analysis of variance (ANOVA) and the software used to facilitate the statistical analysis was StatGraphics 18.

RESULTS

The main finding of this study is that the pulmonary diffusing capacity is diminished after the swimming training in a follow-up condition (44,4±8,4 vs. 43,3±8,4 p<0,05). In addition, men have higher values than women (47,3±6,2 vs. 37,2±8,9 p<0,05) and all of them showed higher values of diffusion at the afternoon compared to the morning session (44,9±8,4 vs. 43,2±8,5 p<0,05).

DISCUSSION

The stress of exercise on the pulmonary system could reveal subtle changes in the permeability of the lungs which, normally, may not be an inconvenience in the extreme-developed lungs from elite athletes. Besides there are additional factors, not evaluated in this study, which could provoke that swimmers develop a type of alteration on diffusion capacity such as horizontal form of locomotion, high core blood flow and arterial pressure, cold water temperature (if they train outdoor), long-term exercise duration (4) and even a possibly

inflammatory collateral process related to chlorine, or swimmers asthma.
This study shows that elite swimmers could develop repeated and subclinical SIPE, not self-limiting, because of their extreme-developed lung diffusion. This could be aggravated when swimmers are exposed to strenuous swimming in cold water (4).

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The authors would like to thank the Catalonian Swimming Federation, Luis Rodriguez and Marc Tribulietx for the colaboration. Additionally, we acknowledge the High Performance Center of Sant Cugat for providing available space close to the swimming pool to do the measures.

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Lung diffusion changes during altitude training (1.850m) in elite swimmers

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INTRODUCTION
Acute exposure to moderate altitude (2.250m) increases the diffusion capacity for carbon monoxide (DLCO) (1) but after a 3 weeks altitude camp exposition (2.250m) there is a decrease in the DLCO (2) values. Swimming practice induces a marked increase in lung capacity and lung diffusion (3,4) over the training development, and elite swimmers use to train at stages located at moderate altitude. The aim of this study is to evaluate the changes in DLCO after 14 days of altitude camp in elite swimmers and the acute effect of an altitude combined session of swimming training at hypobaric altitude of 1.850m + aerobic cycling session at normobaric simulated altitude of 3.000m
METHOD
The participants are 7 international level swimmers, 3 men and 4 women, from 17 to 24 years old, with more than 3 years of experience in altitude training. The training schedule consists of 25-30 hours of training per week. The swimming session lasts 150 minutes and the distance covered is 7.500m at moderate intensity and the cycling session consists of 50 minutes at low intensity. The material used is a computerized spirometer with single-breath method to measure the changes in DLCO (5) (Easy One Pro, ndd, Zurich Switzerland).
RESULTS
There are no changes in DLCO after 14 days of altitude training camp in our sample (42,0±11,9 vs. 40,2±12,7). Likewise there are no changes after a swimming training at 1.850m + an aerobic cycling session at 3.000m in a normobaric hypoxic chamber (45,8±14,4 vs. 45,2±11,8 vs. 41,1±12,6).
DISCUSSION
Altitude exposure and exercise increase the mechanical stress on the pulmonary system, leading to subtle changes in the permeability of the lungs which, normally, may not be an inconvenience in the extreme-developed lungs from elite athletes. This new study shows that elite swimmers with experience in altitude training have not changes in DLCO during and at the end of 14 days altitude of international level training camp. Despite of there are no acute changes after a combined session of swimming (1.850m) and cycling (3.000m), a slight decrease is appreciated after cycling. Further research including more participants and with a higher heterogeneity in the bronquial ability (asthma) are required to assess this tendency.

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The authors would like to thank the Sabadell Swimming Club, the Catalonian Swimming Federation and Luis Rodriguez for the collaboration. Additionally we acknowledge the National Centre for Altitude Training of Font Romeu for providing available space to do the measures at the swimming pool.

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The effect of anti-gravity socks on skin microcirculation in foot

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Gravity causes a hydrostatic pressure gradient in fluid-filled bodily compartments. Accordingly there is a significant displacement of blood to the lower parts of the body with a reduction in the central blood volume when turning from supine to the erect position. Local and central regulatory mechanisms are activated to maintain the arterial blood pressure and to prevent edema in the limbs below the heart level (1). Consequently, the blood flow to the skin of the foot is reduced by localized vasoconstriction called veno-arteriolar reflex (VAR) (2). The aim of our study was to measure the response of skin blood flow in the foot to hypogravity conditions, simulated by wearing the anti-gravity socks (AGS) at rest

and after short lasting aerobic exercise. In nine young healthy volunteers laser-Doppler skin blood flow (LDF) was measured in two skin spots on the foot: glabrous and non-glabrous skin with different vascular anatomy (thermoregulatory and (or) nutritional microcirculatory network) and different vascular sympathetic innervation (adrenergic or cholinergic). Measurements were conducted with and without AGS at rest and after submaximal cycling. In order to avoid the influence of arterial blood pressure to cutaneous blood flow, cutaneous vascular conductance (CVC) was calculated as LDF divided with mean arterial pressure, which was measured simultaneously. At rest, statistically significant hyperperfusion was found in glabrous (LDF: 87,06±15,93PU with AGS compared to 56,51±12,48PU without AGS; P=0,04; CVC: 0,97 ± 0,17 PUmmHg^{-1} with AGS compared to 0,66 ± 0,16 PUmmHg^{-1} without; P=0,003), as well as in nonglabrous skin (LDF: 9,41±1,54PU with AGS compared to 6,76±1,15PU without AGS; P=0,04; CVC: 0,111 ± 0,003 PUmmHg^{-1} with AGS compared to 0,075 ± 0,004 PUmmHg^{-1} without; P=0,004) in hypogravity conditions. After exercise, there were no statistically significant differences in LDF and CVC in any skin sites with respect to AGS. Our results indicated that in hypogravity conditions VAR is absent in glabrous as well as in nonglabrous skin at rest. On the contrary, after physical exertion, these differences in skin perfusion disappeared in both skin spots indicating that other mechanisms and not VAR regulates the skin blood flow to the lower limb after exercise. Increased blood flow through arteriovenous anastomoses in glabrous skin and activation of vasodilatory cholinergic sympathetic nerves to the nonglabrous skin vasculature could be proposed mechanisms. Exercising in hypogravity conditions would be beneficial with respect to VAR to avoid edema in the lower limbs at rest.

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Protective Effect of baker's yeast on Carbon Tetrachloride Induced Hepatotoxicity in rats

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Carbon Tetrachloride [CCl_4] can be used as a solvent, rubber cement and insecticides. Furthermore, observation-based methods illustrated continuing emissions of 35 Gg per year of CCl_4 into the atmosphere[1]. CCl_4 produce acute and chronic liver injury. Baker's yeast is an excellent source of vitamins, minerals, and high-quality protein such as glutathione and choline. Glutathione, the most important antioxidant supporting detoxification and strengthing immunity, is present in up to 10 mM in yeast cells[2]. As well, choline is present in large quantity in Baker's yeast. Choline promotes phosphatidylcholine Synthesis; a vital for the integrity of the cell membranes[3]. Choline is also a precursor of betaine which increase the concentrations of hepatic S-adenosylmethionine that prevents CCl_4 induced DNA hypomethylation which produces cirrhosis in rats' liver[4]. In addition, Betaine as an osmolyte it improves the function of kupffer cells in rat liver macrophages and prevents the reduction of Golgi complexes and mitochondrial induced by the exposures to CCl_4[5]. Our aim was to evaluate protective effect of oral Baker's yeast against CCl_4 induced hepatotoxicity in rats' model. 30 male Sprauge Dawley rats [125-265g] were divided into three groups [n=10]. All rats were fed a normal diet for 2 weeks. Then, group1 were injected with intraperitoneal (0.1ml /100g BW) olive oil. Group2 and 3 were injected with (0.1ml /100g BW) CCl_4 dissolved on eqi-volume of olive oil. However, group3 received oral yeast (200mg) dissolved in distal water by oral tube along with normal diet for 2 weeks before CCl_4 injection. On the 16th day, the rats were humanly killed according to the national guidelines. Blood was collected to measure alanine amino transferase (ALT), aspartate amino transferase (AST) by enzymatic colorimetric method. The livers were weighed and then kept in formalin /saline 10% for histological examination. Exposure to CCl_4 significantly ($p < 0.05$) increase AST to 170±11mg/dl and ALT to 72±6mg/dl as compare to normal group 63±2mg/dl for AST and 21±2mg/dl for ALT. It was noted that yeast significantly reduced the rise in liver enzymes ($p < 0.05$) to 107±8mg/dl for AST and 35.6±3mg/dl for ALT as compare no-yeast fed CCl_4 group. Also, there was an increase in the liver weight in the no-yeast fed CCl_4 group (9.7±0.8g) and (7.8±0.1g) for yeast fed CCl_4 group as compare to control group (7.25±0.2g). Grossly the liver of the rats were damaged, swollen and yellow in CCl_4 group. Microscopically there was perivenular ballooning, hepatocyte fatty degeneration and cell necrosis with many fibrotic septa. All these finding was less evident in yeast fed group. All these data suggest that baker's yeast could be used as a food supplement to protect against xenobiotic induced hepatotoxicity which possibly due its high nutriotional values.

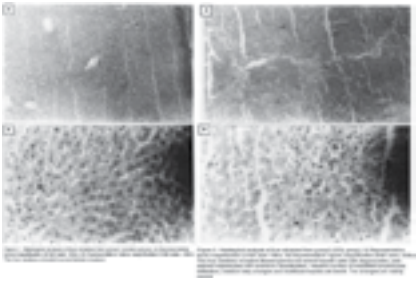

Figure 1. Histological analysis of liver obtained from group1 (control group). A) Representative lower magnification ((H&E stain 100x). B) Representative higher magnification (H&E stain, 400x). The liver sections revealed normal lobular structure.

Figure 2. Histological analysis of liver obtained from group2 (CCl_4 group). A) Representative lower magnification ((H&E stain 100x). B) Representative higher magnification (H&E stain, 400x). The liver sections revealed disarrangement of normal hepatic cells with degeneration, pale stained hepatocytes with extensive vacuolization, massive number of interstitial lymphocytes infiltration and massive fatty changes and multifocal hepatic cell death. The changes are mainly central.

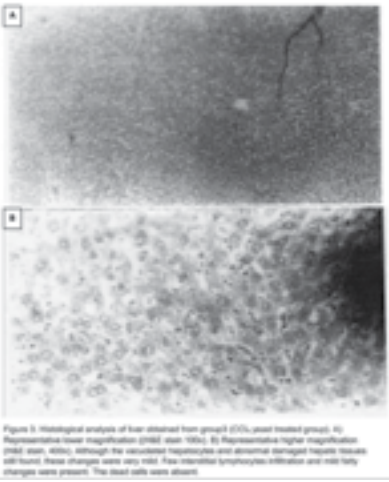

Figure 3. Histological analysis of liver obtained from group3 (CCl_4 yeast treated group). A) Representative lower magnification ((H&E stain 100x). B) Representative higher magnification (H&E stain, 400x). Although the vacuolated hepatocytes and abnormal damaged hepatic tissues were still found, these changes were very mild. The liver sections shows few interstitial lymphocytes infiltration and mild fatty changes and absence of dead cells.

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i need to acknowledge my family and my son Zakria busnaina

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Salivary Cytokines in Yacht Racing Athletes

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INTRODUCTION: It is known that in addition to intense exercise load, the body of sailors is exposed to different extreme factors of environment, such as hot weather, cold water, strong wind, and solar load (Allen, De Jong, 2006). Both physical activity and exposure to environmental stressors modify various components of the immune function (Walsh, Oliver, 2016), but few studies have examined the long-term adaptation of immune system to combined effects of adverse environment and exercise. The cytokines may be the markers of upper respiratory tract infection (URTI) risk (Gleeson et al., 2013) as well as an important predictor of overtraining in athletes (Smith, 2000) and can reflect the negative impact of physical and environmental stress on the body (Starkie et al., 2005). Therefore, the purpose was to examine the salivary cytokines in rest and cytokine responses to high intense exercise in yacht racing athletes. METHODS: Eleven highly-skilled athletes (five males and six females) and eight untrained volunteers (five males and three females) aged from 20 to 24 years were involved in the study. Sports experience of athletes ranged from 8 to 12 years. All participants signed a voluntary informed consent. None of the participants was suffered from acute or chronic diseases or reported about the intake of medication. Saliva samples were obtained before and after high intensity exercises (bicycle ergometer, 350 W, 30 sec). The concentrations of IL-8 and IL-10 were determined using ELISA. Values are medians and interquartile intervals (Me; Q1-Q3), compared by non-parametric models. Statistical significance was accepted at $P<0.05$. RESULTS: There were no significant differences in the salivary concentration of IL-8 or IL-10 before and after high intensity exercises in yacht racing athletes when compared with untrained individuals (IL-8: 1355; 1116-1959 vs. 1251; 805.5-1893.5 pg/ml in rest and 1030; 470-1944 vs. 1610.5; 1063.5-1811 pg/ml after exercises, $P>0.05$; IL-10: 37.4; 14.6-49 vs. 72.85; 14.8-140 pg/ml in rest and 47.5; 25.4-84.6 vs. 47.2; 34.15-72.25 pg/ml after exercises, $P>0.05$, respectively). Also there were no significant sex-related differences in salivary concentration of IL-8 or IL-10 in athletes and untrained individuals ($P>0.05$). CONCLUSION: Taken together, our results indicate that combined effects of adverse environment and exercise in yacht racing athletes do not lead to a change in salivary concentration of the pro-inflammatory chemokine IL-8 and anti-inflammatory cytokine IL-10. Probably, it is due to the long-term adaptation of the immune system to combined physical and environmental stressors.

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C50

Protective effect of co-administration of vitamins C and E on reserpine-induced motor and cognitive impairments and oxidative stress in mice

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Introduction: Given the central role oxidative stress (OS) played in the pathogenesis of Parkinson's disease, several antioxidants have been explored. Aim: Combining two vitamins (C and E) is aimed at conferring greater neuroprotection

against reserpine-induced OS and motor and cognitive impairments in mice. Methods: Twenty-five mice were randomly assigned into 5 groups of 5 animals each. Group I received distilled water only. Groups II-V received reserpine 0.1 mg/kg intraperitoneally on alternate days. In addition, Group III received vitamin E 200 mg/kg/day orally, group IV, vitamin C 250 mg/kg/day orally and group V, combined vitamin E 200 mg/kg/day and vitamin C 250 mg/kg/day orally. All vitamins were given concurrently one hour before reserpine injection for 28 days. Neurobehavioral assessment using novel object recognition test (NORT), Y-maze, beam walking and open field test (OFT) was carried out. Thereafter, the mice were humanely sacrificed and brain homogenate made. Values at $p<0.05$ were considered significant. Results: The negative discrimination index observed in group II (-0.35±0.23) was significantly ameliorated by the co-administration of both vitamins (0.59±0.12). In the y-maze, a significant increase in percentage alternation was recorded in group V (66.7±9.25%) compared to the other groups ($p=0.003$). In the beam walk, there was a significant decrease in number of foot slips (0.3±0.25) as well as the time to reach the safe box (3.00±0.41s) in group V compared to other groups. In the OFT, the transfer latency was significantly decreased (10.3±1.45s) while the number of lines crossed was significantly increased (56.0±13.53) in group V compared to the other groups. The malondialdehyde concentrations was significantly decreased in all vitamin-treated groups compared to reserpine-only group (42.2 ± 028 Umol/L). A significant increase was seen in superoxide dismutase and catalase levels with a non-significant decrease in GSH level across all the vitamin-treated groups compared to the reserpine-only group. Conclusion: The co-administration of vitamins C and E confers a significant neuroprotection against motor and cognitive impairments and OS induced by reserpine in mice.

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Heart Rate variability as a predictive tool in the Military

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Environmental conditions may be predictably extreme and severe, such as those in deserts, polar, alpine regions and deep ocean. However, even in normal habitat conditions, individuals may be exposed to transitory, sometime life-threatening, extreme conditions, due to their daily job activities. One such example, are the Advanced Chemical and Biological Reconnaissance Team from the Army Special Forces (ACBRT). Indeed militaries in the ACBRT are exposed to extreme working conditions, due, not only to the dangerous nature of the job (permissive, uncertain, and hostile environments), but also to the characteristic military wearable, which ultimately leads to a dangerous rise in all vital physiological parameters, within short time, sometimes in hot climates up to 45°C. Thus, real-time physiological status monitoring of these soldiers, is very important, to ensure individual and squad performance readiness. Herein, we present the preliminary evaluation of a ACBRT, during a simulation exercise (n=3). Heart rate (HR) and O2 consumption were recorded simultaneously. Heart Rate Variability was analyzed using the traditional Fast Fourier Transform (FFT). Briefly, the results show some interesting differences in the Low Frequency band (LF) profile, which varies according to the different roles. A clear sustained increase of LF can be seen in the Dirty Man (military responsible for collecting the samples), while the LF from the Clean Man (responsible for storing the samples, with no significant physical activity involved), has marked increase during the mission, with a marked decrease towards the end. On the other hand the High Frequency band (HF; parasympathetic Nervous System), revealed a similar profile amongst all the individuals throughout the mission, with a slight decrease in the beginning of the mission, followed by a steep increase half way through the mission. These changes were correlated with O2 consumption levels, indicative of a shift in the sympatho-vagal balance, towards a more pronounced sympathetic activity. Our results, even though not quite significant, considering the sample size, are a clear indicative of the possible usage of HRV, as a predictive tool for both physical and mental performance assessment, including team readiness for the mission ahead.

Where applicable, the authors confirm that the experiments described here conform with the Physiological Society ethical requirements.

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The effect of exercise mode on exercise induced gastrointestinal damage during exercise performed at a fixed rate of metabolic heat production.

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Intestinal fatty acid binding protein (IFABP), a 15 kDa protein present in the cytosol of mature enterocytes, is rapidly released into the circulation following enterocyte injury and is a sensitive measure of gastrointestinal (GI) barrier damage following exertional heat stress (1). Exertional heat stress protocols typically employ running or cycling exercise at a set percentage of VO_{2max}, which may introduce systematically different rates of metabolic heat production and differences in core temperature responses between groups differing in biophysical characteristics (e.g. sex, body mass, body surface area) (2). This in turn may affect systemic markers of GI barrier function, which are positively related to core body temperature (3). It has long been assumed that the mechanical stress associated with running provokes greater GI damage when compared to cycling, though there is little data to support or refute this notion. To determine whether exercise mode effects systemic measurements of intestinal damage, four men (height: 180.0 ± 6.1 cm,

body mass: 82.5 ± 16.6 kg, cycling VO_{2max}: 46.4 ± 8.4 mL·kg^{-1}·min^{-1}, running VO_{2max}: 53.3 ± 8.5 mL·kg^{-1}·min^{-1}) completed a cycling trial and a running trial at a matched rate of metabolic heat production (9 W·kg^{-1}) in hot, humid conditions (39.2°C, 51.0 % relative humidity). Participants exercised until either a rectal temperature of 40.0°C was obtained, or withdrew due to exhaustion. Blood samples were drawn at rest, when rectal temperature increased by 1.5°C from baseline, and at the end of exercise. IFABP was quantified in serum via enzyme linked immunosorbent assay. Metabolic heat production was similar between running and cycling (running: 9.3 ± 0.4 W·kg^{-1}, 765 ± 137 W, 374 ± 29 W·m^2; cycling: 8.7 ± 0.4 W·kg^{-1}, 715 ± 113 W, 354 ± 19 W·m^2). Running exercise time was 01:19:39 ± 00:21:20 hh:mm:ss, and cycling exercise time was 01:06:25 ± 00:14:17 hh:mm:ss. Mean exercise heart rate was similar between conditions (running: 146 ± 8 bt·min^{-1}; cycling: 148 ± 8 bt·min^{-1}), as was relative exercise intensity (running: 52 ± 7 %VO_{2max}; cycling: 56 ± 8 %VO_{2max}). Time to 1.5°C was 00:35:52 ± 00:04:31 while running, and 00:35:33 ± 00:08:48 while cycling. Peak rectal temperature (39.39 ± 0.29°C vs 39.00 ± 0.39°C) and delta change in rectal temperature (2.90 ± 0.39°C vs 2.49 ± 0.62°C) were higher in the running vs. cycling trial. IFABP concentration increased by 357 ± 484 pg·ml^{-1} and 622 ± 642 pg·ml^{-1} at +1.5°C and exhaustion during cycling, and 97 ± 67 and 378 ± 690 pg·ml^{-1} at +1.5 °C and exhaustion during running. Despite the longer exercise duration and greater peak core temperature observed during running, the release of IFABP was greater both during and after the cycling exercise when metabolic heat production was matched at 9 W·kg^{-1}.

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