PN
Physiology News

Issue 108 / Autumn 2017

Stress in the gut

It's not all in your mind

The Physiological Society

BAP
British Association for Psychopharmacology

Certificate in Non-Clinical Psychopharmacology

The Royal Cambridge Hotel, Cambridge, CB2 1PY

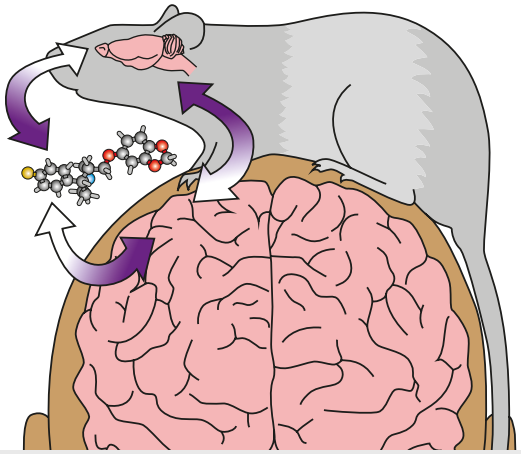
4th – 7th March 2018

This residential programme provides attendees with a unique opportunity to receive training in key aspects of experimental psychopharmacology from leading researchers in the field. The course will cover the following areas, using a combination of plenary lectures, taught and practical sections, round-table debate and a team project.

The following topics are covered:

- Clinical Neuroscience
- Pharmacokinetics in Psychiatry
- The Molecular Biology of the Mind
- Statistics and Experimental Design
- Pre-clinical Behavioural Psychopharmacology
- Combining Neurobiology and Behaviour
- In vivo-Neuroimaging and electrophysiology in Psychopharmacology

It will benefit novice and experienced psychopharmacologists, and those working in related fields, by encouraging appraisal and refinement of experimental design and training in essential skills.



Approved by the Royal Society of Biology for purposes of CPD, this event may be counted as 108 CPD credits.

For more information and to register interest go to www.bap.org.uk/nonclinical

Physiology News

We welcome feedback on our membership magazine, or letters and suggestions for articles for publication, including book reviews, from our Members.
Please email **magazine@physoc.org**

Physiology News is one of the benefits of membership, along with reduced registration rates for our high-profile events, free online access to our leading journals, *The Journal of Physiology*, *Experimental Physiology* and *Physiological Reports*, and travel grants to attend scientific meetings. Membership offers you access to the largest network of physiologists in Europe.

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Welcome to the Autumn 2017 edition of *Physiology News*

Introduction

News in brief

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Features

Membership

Cover image: Gut Feeling: Intestinal organoids provide a living 3D cellular model of the intestine in a Petri dish. Both the structural characteristics and ability to modulate the cellular state of the model enables us to mimic that seen in disease and the normal intestine whilst also reducing the extent of animal testing. This image of a mouse intestinal organoid features staining of KI-67 (green) marking the intestinal stem cells, E-cadherin (red) which enables the visualisation of cell boundaries but also the extent to their permeability, and DAPI (blue) commonly used to indicate the nucleus. My research utilises this model to test the regenerative and anti-inflammatory capabilities of stem cell secretions. By stimulating the organoids with bacterial toxins I can recreate inflammatory disease within the intestine and understand if and how stem cell secretions could be used as a therapeutic in the clinic. Ben Mellows (PhD student, University of Reading).

Back cover: Chris Wood and Nicola Fawcett, Modernising Medical Microbiology (Attribution-NonCommercial-ShareAlike 4.0 International (CC BY-NC-SA 4.0)). The image is created using the same techniques used by hospitals and laboratories to study how bacteria respond to antibiotics. Gut bacteria are stamped in decorative patterns onto nutrient jelly ('agar'), left to grow overnight, and then photographed.

Europhysiology 2018

A partnership between The Physiological Society, the Scandinavian Physiological Society, Deutsche Physiologische Gesellschaft and the Federation of European Physiological Societies

14–16 September 2018
The QEII Centre, London, UK

Symposia, keynote & plenary lecturers are online

www.europhysiology2018.org

Karen Doyle

Lecturer, Department of Physiology, NUI Galway

Once again, Roger has passed the baton to me to guest edit the Autumn edition of *Physiology News*. In this edition, we have four feature articles that touch on The Physiological Society's theme for 2017, 'Making Sense of Stress'.

David Finn talks about how stress can profoundly affect perception of pain, variously producing analgesia or exacerbation of pain under different circumstances. Lin-Hua Jiang's article highlights the role of the TRPM2 channel in oxidative stress and its possible involvement in an array of diseases ranging from diabetes to acute brain injury and chronic neurodegenerative conditions. Mark Burnley tells us about the acute and chronic responses to muscular stress during exercise – how it tries to break us, ultimately shapes us, and the sad truth of 'use it or lose it'. Kim Barrett explains the impact of stress on the friendly bacteria in the gut. She also discusses the role that the microbiota may play in maintaining a healthy brain, and in the onset and progression of brain disorders – psychiatric and neurological. In this article, we also learn about faecal microbial transplant therapy, which can be very effective in treating severe and persistent diarrhoea. It may also prove beneficial for chronic diseases such as irritable bowel syndrome. Apparently, there are do-it-yourself instructions on-line, although Kim cautions that further clinical trials are needed!

Questionable online sources of information is a strong theme in this issue. We have a commentary from Gladys Onambele-Pearson and Kostas Tsintzas on the 'alternative truths' published by self-styled gurus on social media, espousing all things human exercise physiology and nutrition. Physiologists can be a shy bunch, but perhaps we should step up and become the real gurus on social media. We probably should put ourselves out there to combat the 'fake news' as Donald Trump would describe it. There is clearly a huge appetite from the general public for information on human physiology, but we may need to brush up on our scientific communication skills to 'guru' effectively.

In this issue, we also have a feature article from Vivien Rolfe on open education, discussing widening access to educational materials in the life sciences, and in particular, open practice for laboratory practical teaching. We also learn what a Wikimedian is, in an interview with Andy Mabbett, our Wikimedian in residence. Andy talks about the Wikimedia community and their ethos of free access to all knowledge for everyone. With the explosion of information available online, the Wikimedia movement relies on the contribution of experts for accuracy and maximum impact.

Roger Thomas has provided a report on The Physiological Society's AGM recently held in London, in his own inimitable style, telling us about the purple chairs as well as other notable details from the reports from The Society's President and Officers. He also mentioned that the inaugural plenary speaker (Eric Olson) was firmly of the view that muscle is the most important tissue in the body, a sentiment no doubt shared by many, but I think I am still team brain!

Our front and back cover show stunning gut-related images. On the front, we have mouse intestinal stem-cell-containing organoids, courtesy of Ben Mellows, a PhD student at the University of Reading, and on the back cover we have a beautiful image of gut bacteria grown in culture from Nicola Fawcett and Chris Wood.

As with last year, if you spot any mistakes, please send your letter of complaint to Roger.

The Affiliate Working Group have an announcement!

We have a surprise for you! The Affiliate Working Group have been invited to edit a Special Edition of *Physiology News*. This issue (*PN* 109, December 2017) promises to focus on issues related to early career researchers from surviving an undergraduate degree to a day in the life of a newly appointed fellow. Kim Barrett, Editor-in-Chief of *The Journal of Physiology*, will provide advice on 'Getting your first paper published', whilst we'll provide our views and latest updates on Brexit (and what this means for research), REF 2020/1 (what this means for us), and a plethora of current and upcoming events. Forefront will be what to expect from our very own (and exciting) Future Physiology (Leeds, 13–14 December 2017). Furthermore, we'll have a few extra surprises, but we don't want to give everything away.

Physiology News is an excellent quarterly magazine that brings together the diversity within physiology research. This Affiliate Edition will be no exception, providing updates on the latest Society activities, including interviews with the recent AGM competition winners, whilst aiming to provide a strong collaborative platform for early career researchers within The Physiological Society. This is an exciting opportunity for all Affiliates, and we want you to get involved too. Do you have an interesting article or feature to complement our Special Edition? If so, contact: Hannah M Kirton **h.m.kirton@leeds.ac.uk**, Jo Edward Lewis **jo.lewis@nottingham.ac.uk** and Julia Turan **jturan@physoc.org**. We look forward to hearing your thoughts. Affiliates Unite!

PhD degrees

Tim Biscoe
University of Bristol, UK

I am prompted to write by the piece by Ole Petersen about his not acquiring a PhD. I went to medical school in 1950, intercalated a degree in physiology on the way through to medical qualification, registration, house jobs, and then in 1958 or so drafted into the Royal Army Medical Corps (RAMC). I ended up at Porton Down in a long line of previously drafted physiologists like Bill Douglas, Richard Adrian, John Widdecombe, Bob Torrance, Tony Taylor, and others. The head of physiology was Sir Charles Lovatt Evans, who came into the lab for tea and/or coffee on two or three days a week. In his retirement from the Jodrell Chair at University College London (UCL) he lived nearby and told many stories about UCL going back to the turn of the century.

JH Gaddum I had met briefly when an intercalating science student attending a Physiological Society meeting at The London Hospital Medical College. I was told to help Leon Bernstein with a Demonstration to do with respiration, and using a Cossor Oscilloscope. I did not understand what I was doing, and suddenly there appeared in the room Gaddum, who said to me 'My name is Gaddum, who are you?' From that Demonstration, I learned however distinguished you may think you have become, never assume anyone knows who you are; a very important lesson in life. Gaddum was certainly distinguished. I knew his work with Henry Dale, and many of us used his textbook of pharmacology. He came each year with the visiting group to Porton Down.

When I was leaving the RAMC I enquired of Gaddum, the Director of the ARC Institute of Animal Physiology, at Babraham, whether I might apply for a position. The upshot was that I was appointed and we moved into an ARC house on The Close, along with our two little daughters. The lab I had was next to the one that Gaddum used with Michael Smith. Gaddum frequently came in to see what I was doing and to chat about science. He said to me that I had talked my way into Babraham. I don't know how, but that may be a good procedure for a hungry young scientist. At that time Babraham was probably the leading place in the UK for work on the nervous system. Apart from Gaddum, the staff included Marthe Vogt, Catherine Hebb, Ann Silver, Keris Krnjevic, Victor Whitaker and many others in allied fields of physiological science like Arnold Feinstein.

There were also visiting scientists, like John Phillis from Canberra, and after Richard Keynes arrived, Murdoch Ritchie from Yale.

One day I asked Gaddum if I should work for a PhD degree. He said 'You don't want to waste your time doing that, you should do experiments and publish the results.' That would be in 1962. His reply was right for me, but also reflects the time in which we lived and worked. Nowadays, it would be usual for young scientists to study for a PhD degree regardless of a medical qualification, certainly necessary if they had no medical degree.

I have just now read the text of a discussion conducted outstandingly well by Tony Angel with Andrew Huxley on behalf of The Physiological Society. It seems to me to be an important historical document. Amongst many interesting comments made by Andrew Huxley and germane to this piece is: '... my Cambridge BA, indeed the only degree that I hold that's properly earned by examination, or thesis, I have never written a thesis, my only honest degree is a Cambridge BA based on part one of the natural sciences Tripos and a certificate of diligent study in my third year when I was dissecting the corpse' [dissection, because he had it in mind to study medicine and had to learn anatomy].

Another comment relating to Alan Hodgkin was: 'Tony Angel: What was your relationship? Was he your PhD supervisor?' To which Andrew Huxley replied: 'I never did a PhD.'

I think it follows that when Andrew Huxley was appointed as Jodrell Professor at UCL his title was 'Mr'. If I had but known it in 1962, I was in exceptional company.

Subsequently, I did put together experimental work I had done on respiratory muscles with a view to submitting the body of work for an MD degree at London University by thesis, which was possible at that time. I then enquired from the University what the fee would be and was told a large sum, £40-50 I think. Since this would represent a third of my salary, and my wife and I had two children with a third in the offing, it was clear that submission would be silly and without merit, merely a means of feeding self-esteem. There would be no benefit for my family and little or none for me, and we would have fewer resources as a family.

I was fortunate to have a wonderful wife and life, even to being proposed as a member of the Committee of The Society by our respected Editor.

Farewell!

Ken O'Halloran
University College Cork, Ireland

It may interest your readers to know that I have resigned my post as Meetings Secretary (March 2017) on principle and in objection to the strategic direction of The Society under the current leadership, principally in respect of recent restructuring decisions. I am grateful for the opportunity to have served The Society across several domains of activity, but especially in the delivery of an exciting portfolio of scientific meetings. I had the pleasure and benefit of working with committed and innovative members and staff on Meetings Committee who should continue to be congratulated for their dedication to The Society. I am grateful to Christine Carr and Sarah Bundock for their wise counsel, but especially grateful to Nick Boross-Toby who was a stalwart and great ambassador of The Society, and a wonderful collaborator enabling Meetings Committee during my tenure to think outside of the box.

I am also deeply grateful for the opportunity to have served on Council, and for the friendships and learnings of that experience over my five years as a Trustee. I wish The Society well into the future.

David Eisner, President of The Physiological Society, replies:

In my role as 'the current leadership', I am replying to Ken's email. I would first like to thank him for his service to The Society as Meetings Secretary. Our programme of meetings has benefited enormously from his infectious enthusiasm. As regards the restructuring to which he refers, this was primarily implemented to improve the effectiveness and performance of The Society as well as foster a successful office culture particularly with regards to more collaborative ways of working. Members may like to know that both publications and events (meetings) are now the responsibility of Simon Rallison in his newly created role as Director of Scientific Programmes. Simon will lead on activities aimed at promoting the dissemination and discussion of scientific research, through its high quality journals and at world-class meetings, to advance the science of physiology.

Please send your correspondence to **magazine@physoc.org**

Henry Lovett

Policy and Public Affairs Officer, The Physiological Society

Science works with the right people in the right place

Scientific research is an international pursuit, with unique facilities and local pockets of specialisation around the world. The scientific ecosystem enjoys a regular turnover of talent, spreading knowledge and skills. This ability to move around to conduct work in new locations is of great value to scientists, as shown by the results of our survey of Society members about Brexit. The key concerns raised focused on freedom of movement for researchers and students even more so than access to EU research funding.

Unfortunately, the existing UK system governing immigration of workers and students is complicated and often causes problems and delays. There is a high probability that the requirements imposed by Brexit will further complicate immigration and restrict those who can come here as researchers or students. Not to mention the legislation around immigration; the rhetoric coming from some UK sources is highly likely to discourage immigration to this country even when it would be allowed.

The Society has released a policy position statement on international mobility which addresses these and related issues, and gives recommendations to policymakers on how to improve the situation. Rather than discussing exemptions and special circumstances for particular sectors, it would be far preferable to have a flexible immigration system that works for all. The Society and other sector bodies will continue to advocate for this, including by following internal recommendations also given in the position statement. You can read it at: **bit.ly/2va7o3U**

Political changes around science responsibilities

There have recently been a number of appointments of great relevance to the science community resulting from June's general election. One key figure is the new Chair for the Commons Science and Technology Select Committee. Due to the drop in size of the Parliamentary Conservative Party at the election (and committee allocation being roughly proportional to number of MPs), this committee has passed to the Liberal Democrats, with Norman Lamb being elected Chair. The previous Chair, Conservative Stephen Metcalfe, stood unsuccessfully for the Chair of the Education Select Committee, which was won by Robert Halfon. Mr Metcalfe's predecessor, Nicola Blackwood, lost her seat at the election. The ministerial scene is much more stable for science, with Jo Johnson reappointed Minister for Universities and Science, Greg Clark remaining Secretary of State for Business, Energy and Industrial Strategy, and Justine Greening remaining Secretary of State for Education.

The House of Lords Science and Technology Committee, now Chaired by the crossbench Lord Patel, has launched an inquiry into Life Sciences in the context of the Industrial Strategy. More information can be found here: **bit.ly/2uiogSA**

Outside of the Palace of Westminster, other important appointments have been made. UK Research and Innovation (UKRI), the new umbrella body for the Research Councils and Innovate UK that was formed by the Higher Education and Research Act, now has a CEO in the august form of Professor Sir Mark Walport, currently the Government Chief Scientific Advisor. He will occupy both positions part-time until a replacement Chief Scientist is appointed. Sir John Kingman was previously appointed interim Chairman of UKRI, and will hold this position until the organisation is up and running steadily. The other body formed by the Act is the Office for Students (OfS), which takes over some responsibilities from Hefce and other higher education organisations. The CEO of OfS has been announced as Nicola Dandridge, the current Chief Executive of Universities UK. This has caused some controversy, with the appointment to the new post of the head university lobbyist being described as 'poacher turned gamekeeper'. However, Ms Dandridge undeniably has a great deal of sector experience to bring to the new body. The Chair of the OfS will be Sir Michael Barber, another long-time expert in education.

Bringing you snippets of the latest intriguing research

Alzheimer's memories may not be lost

Fear conditioned Alzheimer's mouse models forget the association between the smell of lemons and an electric shock within a week. However, optogenetic stimulation of these mice reactivates these 'lost' memories, restoring the appropriate freezing response to the smell of lemons.

DOI: 10.1002/hipo.22756

Water officially has a taste

Genetic silencing of the acid-sensing (sour) taste receptor cells blocks the tongue's taste nerve response to pure water. Furthermore, water-deprived mice with optogenetic engineered sour taste receptors would 'drink' by licking the stimulating light spout up to 2,000 times every 10 minutes in an attempt to quench their thirst.

DOI: 10.1038/nn.4575

Why our bodies reject transplant organs

Polymorphisms in signal regulatory protein alpha (SIRPα) modulate the strength of the SIRPα-CD47 interaction. CD47 gives monocytes an affinity sensing mechanism to perform 'ID checks', triggering dendritic cell maturation and activation of T-cells against foreign donor organs.

DOI: 10.1126/sciimmunol.aam6202

CRISPR fixes viable human embryo

CRISPR–Cas9 gene editing has corrected a mutation in MYBPC3, which causes hypertrophic cardiomyopathy, in dozens of viable human embryos. Safety was improved by limiting off-target edits and mosaicism via co-injection of sperm and Cas9 protein bound to guide RNA into the egg.

DOI: 10.1038/nature.2017.22382

Time doesn't mend a broken heart

Takotsubo, or Broken Heart syndrome, results in cardiomyopathy caused by a stressful event like the death of a loved one. Currently patients are thought to make a full recovery, however a new 4-month follow-up study reveals that abnormal cardiac function persists with microscopic fibrotic scarring occuring which may never heal.

DOI: 10.1016/j.echo.2017.03.016

Reports of recent Committee meetings

The purpose of these short updates is to keep you informed about the inner workings of our Committees. The following summaries detail the meetings of the past few months.

Council Committee

The main points of business at the May and July Council meetings included an update from the Property Strategy Working Group (PSWG). Chaired by President-elect Bridget Lumb, the working group has been tasked to develop a property strategy to optimise The Society's property asset, Hodgkin Huxley House (H^3), to enable The Society to achieve its charitable purposes: to support and sustain the discipline of physiology through the advancement of science and education and thereby the advancement of health. Property and facilities management consultant, Sue Cleverdon, from Cleverdon Associates Ltd, has been engaged by The Society to provide professional advice to the working group. At the May meeting, Council approved to close the conference hire business at H^3 as it wasn't financially viable. Trustees also agreed, in principle, to approve the recommendation to proceed with the preferred option for the building, which is to consolidate the staff onto the first two floors enabling the upper three floors to be rented out. This option not only increases rentals over five years but makes better use of the available H^3 space by increasing flexible use. This is subject to full feasibility analysis, detailed costings, and the building survey outcomes, and a detailed report will be taken to the September meeting of Council.

Governance was also on the Agenda at the May meeting. Council agreed that, as it delivers a new strategy for 2018-2022, it was an opportune time to review its governance. Council agreed to carry out a systematic review of the Governance Practice Framework and identify areas of weakness and causes for concern. Areas for review would then be prioritised to ensure The Society's governance is robust both now and in the future. The review would focus on four areas:

- key board functions
- improving Board processes
- board effectiveness
- defining governance roles.

An experienced Governance Manager, Rosie Waterton, has been recruited to work with the Board, Chief Executive, and Chief Operating Officer to develop, implement, and improve the governance structure and processes.

The digital communications landscape is rapidly changing, with new platforms and shifts in trends happening regularly. Expectations for where users want to find content relevant to them are shifting too; consequently The Society needs to ensure that the aims and scope of our digital channels align both with our strategic priorities and the expectations of our audiences. To ensure we are in a strong position to continue to develop and improve cross-Society communications, at the July meeting Council approved the recommendation for the creation of a Communications Officer role.

The Communications Officer's key responsibilities will include:

- Website – create new webpages for staff, write content, and prepare images.
- Social media – ensure our channels are populated with engaging content and coordinated across The Society.
- Digital content – create in-house videos and images.
- Email marketing – manage email distribution process across all teams.

On 19 April 2017, John Wiley & Sons and The Physiological Society announced the renewal of their publishing partnership for a further 10-year term, building on over 13 years of collaboration. The renewed partnership will ensure The Society's journals, *The Journal of Physiology* and *Experimental Physiology*, continue to develop as world leaders of physiological research. At May Council the Trustees Council, agreed to support a number of pilot schemes aimed at broadening The Society journals' editorial coverage, increasing submissions, and attracting readers.

Finance Committee

We bid farewell to Anne King, the outgoing Treasurer, who stepped down at the 2017 Annual General Meeting (AGM) and has chaired the Committee with diligence, authority, and a dose of good humour.

Frank Sengpiel, already a committee member, starts his four-year term and will chair his first meeting in September. Recent meetings have dealt with the standard business of accounts, audit, and auditor oversight. In addition, lots of time has been spent on reviewing our investments and investment fund managers – a key obligation for charities. We move forward with a new investment policy, now approved by Council, that will continue to safeguard our most important asset as well as hold our fund managers to account. We were very pleased with the signing-off of a new 10-year publication deal with Wiley at improved terms. Finally, we liaise closely with the Property Strategy Working Group to maximise utilisation of Hodgkin Huxley House (H^3) for the benefit of The Society.

Policy & Communications Committee

The Policy and Communications Committee (PCC) held a teleconference meeting in the light of the unexpected political changes of the first half of this year. The committee was updated on the political situation and our response to developments including the new occupants of the House of Commons, Select Committee changes, and the progress of Brexit planning. Responses to these issues include targeted political engagement of new and returned parliamentarians, and our policy position statement on international mobility. The committee also started discussion of Policy and Communications plans for the upcoming year, including the possibility of taking the topic of international mobility in science post-Brexit to an event with policy-makers and politicians in the future.

Importantly the discussion confirmed changes to the committee Terms of Reference, and those of the *Physiology News* Editorial Board, meaning the relationship between PCC and *Physiology News* is now official. The Policy and Communications Committee will now oversee *Physiology News* as The Society's flagship membership communication, with editorial control of *Physiology News* continuing to rest with the *Physiology News* Editorial Board.

The team of physiologists releasing a shark back into the ocean

Be the next Member in the spotlight

Holly Shiels and a team of physiologists ventured to the Arctic to study the oldest living vertebrate, the Greenland shark. The Society's Communications team documented the trip in a series of videos and blog posts available at **bit.ly/sharkdiary**

Understanding the physiology of creatures born in Shakespeare's time is important for our sake, and theirs. Uncovering how they live for hundreds of years could shed light on human diseases associated with ageing, such as cancer and heart disease. To conserve them most effectively, we need to understand their physiology, so we can anticipate how they will respond to changes in their environment.

Readers gain insight into techniques such as calculating the heart rate of the sharks using strips of heart muscle, and estimating their age using carbon dating, as well as learning about the rocky life on a research vessel.

Holly's blogs and videos were just the beginning: we're on a mission to showcase the work of our Members! If you're interested in featuring your work in writing and in videos, get in touch at **news@physoc.org** telling us about your research written in public-friendly language.

Members awarded Fellowships

The review panel, members of the Membership & Grants Committee, were impressed with how well the applicants demonstrated wide engagement with The Society's activities. Those elected to Fellowship are as follows:

- Douglas Bovell, Weill Cornell Medical College Qatar
- Lucy Donaldson, University of Nottingham
- Frank Mojiminiyi, Usman Dan Fodio University
- Louise Robson, University of Sheffield
- Robert Unwin, University College London

The Trustees of The Society congratulate these members on their achievement.

Full details can be found here: **www.physoc.org/fellow-membership**

Vaccine for Diabetes Type 1 shows promise

Pre-clinical trials of a new enterovirus vaccine has proved successful in mice. In particular the vaccine combats coxsackievirus B1 which can induce pancreatic beta-cell autoimmunity in ~ 5% of infected children.

DOI: 10.1016/j.vaccine.2017.05.057

Men have a biological fertility clock too

Women aged under 30 with a male partner aged 40-42 saw chance of live birth after IVF fall to 46% from 73% for men aged 30-35. Women aged 35-40 also significantly benefit from having a male partner who is under age 30, in that they see a nearly 30% relative improvement in cumulative incidence of live birth when compared to women whose partner is 30-35 – from 54% to 70%.

http://bit.ly/2wFRFYk

Finishing your antibiotics might do more harm than good

A new analysis published in The BMJ highlights the lack of evidence that failing to complete a prescribed antibiotic course contributes to antibiotic resistance. Taking antibiotics for longer than necessary, however, will in fact actually increase the risk of resistance.

DOI: 10.1136/bmj.j3418

Journals accept Star Wars 'Midi-Chlorians' fake study

TIn a sting designed to detect predatory journals, four published a hoax study submitted under the names Dr Lucas McGeorge and Dr Annette Kin about 'midi-chlorians', a fictional 'microscopic life form that resides within all living cells' from the Star Wars film series that give Jedi their force powers.

http://bit.ly/2gLrSda

Science is Contagious

After factoring out differences in gender, family support, academic achievement, quality of teaching, and prior interest in STEM classes, researchers found that the level of interest from peers had a significant effect on science career choices and could improve grades. In other words, interest in STEM subjects is contagious amongst students in the classroom.

DOI: 10.1126/sciadv.1700046

Experimental Physiology

Inaugural Review prize

The 2017 prize was won by Colin N Young of The George Washington University, Washington, DC. His review article 'Endoplasmic reticulum stress in the pathogenesis of hypertension' is published in the August Issue of *Experimental Physiology*: **http://onlinelibrary.wiley.com/doi/10.1113/EP086274/full**. The award was announced at the IUPS Meeting in Brazil, where the article was made freely accessible throughout August.

Myths and Methodologies

In response to the challenge to improve transparency and reproducibility in published research results, *Experimental Physiology* is inviting a series of mini Reviews which will explore the underlying principles of selected methodologies/techniques and consider/identify appropriate and inappropriate uses. The invited authors describe a methodology, and its underlying principle of action, validity, reliability and variability, appropriate and inappropriate applications, and provide tips on effective use and the avoidance (or at least minimisation) of error. The overall aim is to give the reader a thorough understanding of a methodology, and its appropriate application, and importantly the potential limitations of the data it provides. The first Myths and Methodologies paper, Making Sense of Exercise Mass and Water Balance, is published in the September issue, along with an introduction to the series and a commentary on the paper:
http://onlinelibrary.wiley.com/doi/10.1113/EP086284/full
http://onlinelibrary.wiley.com/doi/10.1113/EP086559/full

New Consultant Editors

Experimental Physiology is pleased to welcome to the Board Nic Green and Julie Greeves. They will act as points of contact for the RAF and Army, respectively, identifying research being done that may be of interest to *Experimental Physiology* and raising awareness of article types considered for publication in *Experimental Physiology*, particularly Case Studies.

The Journal of Physiology

The Journal of Physiology team: Harold Schultz, Ole Paulsen, Sally Howells (Managing Editor), Mike Hogan, Scott Powers, Laura Bennet, Hiro Kubo, David Grundy (hiding), Priya Mistry (Editorial Assistant), Kim Barrett, Ian Forsythe, Peying Fong, Pancho Sepúlveda, Janet Taylor, Jaideep Bains.

New citation metrics released

We are pleased to announce that *The Journal of Physiology* (*JP*) and *Experimental Physiology* (*EP*) had another strong year with regard to citation metrics. We expect *Physiological Reports* to get its first Impact Factor next year.

Whilst we do not agree with the importance that is placed on the Impact Factor, and are signatories of the San Francisco Declaration on Research Assessment (DORA), the Impact Factor is still important for many authors, especially from certain countries, where they have strict rules on the minimum Impact Factor a journal should have in order for their research to be acknowledged.

JP is once again the most cited journal in the Physiology category, showing the great level of interest in all our published content. It is also ranked top in Eigenfactor Score, a rating of the influence of a scientific journal.

Both journals performed well in the cited half-life, a measure of the longevity of the articles a journal publishes.

Citation metric	*JP*	Rank in Physiology	*EP*	Rank in Physiology
Total cites in 2016	48,567	1st	5,275	27th
Cited Half-Life	>10.0	=1st	7.5	61st
Eigenfactor	0.04783	1st	0.00911	25th
Total Articles Published in 2016	447	5th	136	29th
Article Influence Score	1.748	8th	0.927	31st
Immediacy Index	1.803	5th	1.000	12th
Five-Year Impact Factor	4.898	8th	2.937	32nd
Impact Factor	4.739	9th	2.912	28th

The Journal of Physiology – editorial update

By Kim E Barrett

The leadership Team of *The Journal of Physiology* has been busy, with well-attended meetings of the Editorial Board and the team of Senior Editors taking place in London in late June.

We discussed editorial strategies and new initiatives for *The Journal*. For example, we considered how better to initiate early career researchers in the editorial process. We plan to kick off a program where junior faculty members could serve as editorial 'fellows', participating in the editorial decision making process under the mentorship of a Senior Editor. We expect to announce details of the application process for this scheme in the near future – please let us know if you are interested.

We also decided on an initiative that should increase transparency and accountability on the part of *The Journal*. Thus, starting later this year, we will publish the name of the Reviewing and Senior Editors who were responsible for handling an accepted manuscript. We are proud of the work we publish, which undergoes rigorous peer review, and therefore equally proud to have our names associated with the finished product.

We discussed ways to increase the visibility of the research in our pages, especially with those beyond the discipline.

Our social media presence is growing rapidly, with followers on Twitter, Facebook, and LinkedIn all increasing by double-digit percentages year-on-year. Press releases that highlight selected papers have also enjoyed great coverage in major national and international media outlets. We learned from our publisher, Wiley, that a very substantially higher proportion of our published papers receive 'Altmetric' scores than those in the average Wiley journal, further reflecting efforts to ensure our papers have the widest possible impact.

Finally, while I was in London I had the honour of presenting a publishing 'roadshow' at my *alma mater*, University College London. My goal was to shed light on the unwritten rules for successful publication for trainees and junior faculty. Let me know if you are interested in hosting your own roadshow!

I recently presented a workshop on publishing at the IUPS meeting, along with the Editor-in-Chief of *Experimental Physiology*, Mike Tipton, and American Physiological Society colleagues Irv Zucker (Editor-in-Chief of *American Journal of Physiology - Heart*) and Dennis Brown (Editor-in-Chief of *Physiological Reviews*).

And as always, please do not hesitate to contact me at **kbarrett@ucsd.edu**, or any member of the Leadership Team, if you have comments, compliments, or complaints about *The Journal*.

Call for papers – perinatal physiology

We are seeking research submissions that are related to the advances, challenges, and controversies in both fetal and neonatal physiology and the translation from basic biomedical physiology to the clinic.

We have invited people to write reviews in the areas of cardiorespiratory physiology; neuroprotection; inflammation and infection; fetal behaviour; developmental programming; and perinatal respiratory physiology.

We welcome research submissions from other perinatal research areas. Any enquiries regarding suitability of submissions should be directed to Managing Editor Sally Howells (**showells@physoc.org**).

We are happy to consider additional review articles. Please send a one-page outline to Managing Editor Sally Howells (**showells@physoc.org**).

We will be awarding a $500 prize for the best research paper in perinatal systems physiology and clinical translational physiology by an early career researcher in honour of the late Professor Julian Parer.

We also invite students and fellows to propose CrossTalk debates around the themes covered by the issue. These should provide readers with explicit accounts of both sides of a current controversy in physiology, allowing them to understand the arguments and arrive at an informed conclusion on the topic. Please send suggestions to Managing Editor Sally Howells (**showells@physoc.org**).

Submission deadline: 31 October 2017.

Physiological Reports

Physiological Reports at IUPS

Physiological Reports was much in evidence at the IUPS Congress in Rio. Its activities included sponsoring the symposium Dyadic and T-Tubule Remodelling in Cardiac Health and Disease, chaired by Andrew Trafford, and a session on women in science intended primarily for early career researchers. The journal also hosted Meet the Editor sessions at The Society's exhibition stand, with Sue Wray and Morten Thomsen busy fielding questions from potential authors and kissing babies.

In a world of information abundance, journals increasingly add layers of selection, to help their readers. The senior editorial team at *Physiological Reports* single out a few papers from each issue for particular recommendation. Here are the current Editors' Choice articles, all free to view (like all the journal's content) on the website: **http://physoc.onlinelibrary.wiley.com/hub/journal/10.1002/(ISSN)2051-817X/**

Chiara Ghezzi, Guillaume Calmettes, Pauline Morand, Bernard Ribalet, Scott John: **Real-time imaging of sodium glucose transporter (SGLT1) trafficking and activity in single cells**

Nerea Llamosas, Luisa Ugedo, Maria Torrecilla: **Inactivation of GIRK channels weakens the pre- and postsynaptic inhibitory activity in dorsal raphe neurons**

Debby Lee, Bridget Martinez, Daniel E Crocker, Rudy M Ortiz: **Fasting increases the phosphorylation of AMPK and expression of sirtuin1 in muscle of adult male northern elephant seals (*Mirounga angustirostris*)**

Susan T Halm, Michael A Bottomley, Mohammed M Almutairi, Maurico Di Fulvio, Dan R Halm: **Survival and growth of C57BL/6J mice lacking the BK channel, Kcnma1: lower adult body weight occurs together with higher body fat**

Future Physiology

13 – 14 December 2017
University of Leeds, UK

If you are an early career researcher, this is a must attend event in 2017

- Be inspired by the six invited speakers
- Present your research at one of the 20 oral communications
- Gain key skills at the career development workshops in publishing and also grant writing
- Network and explore new opportunities at the poster communication session

The conference is tailor made to give you the experience, renewed enthusiasm and networking opportunities to help you take the next steps in your career

Confirmed speakers

- James Brown, Aston University, UK
- Laura Corns, University of Sheffield, UK
- Deborah Henderson, Newcastle University, UK
- Jackie Hunter, CEO, Benevolent Bio, UK
- Jamie McPhee, Manchester Metropolitan University, UK

Abstract submission closes 2 October

www.physoc.org/futurephysiology

The Physiological Society

Physiology – current trends and future challenges

Henry Lovett

Policy and Public Affairs Officer,
The Physiological Society

Around this time last year, The Physiological Society released its report *Health of Physiology*, which analysed the discipline as it stands in the UK and Ireland. This has provided the inspiration to the International Union of Physiological Sciences (IUPS) to conduct a similar analysis on the strength of the discipline worldwide and the different challenges faced by physiologists and students in different countries. After the project research was carried out, the IUPS partnered with The Physiological Society to examine the responses and write up the conclusions, to produce *Physiology – Current Trends and Future Challenges*. The report, along with a series of companion essays by all the Chairs of IUPS Committees and Commissions, was released at the IUPS 2017 Congress in Rio de Janeiro, Brazil.

Details were sought from IUPS member organisations on the atmosphere around research, including funding, regulation, public perception, and links with government and industry. Twenty-seven physiological societies across six continents took part, making this project truly global in scale. Most organisations expressed concern regarding the availability of funding, with financial support from government deteriorating in recent years. Several organisations noted that funds are more likely to be disbursed to researchers doing clinical rather than basic research. Most organisations reported technical expertise in physiological sciences, but several remarked on diminishing practical skills for *in vivo* techniques and animal-based experimentation. Several responding organisations noted that animal experimentation is being discouraged.

Physiology is taught in specific undergraduate and postgraduate degrees, as well as in medical, veterinary, dental, and nursing courses. Several respondents remarked that in their country physiology is not always taught within a dedicated physiology course although there is a growing emphasis that physiology is in fact clinically relevant as the foundation of scientific medical practice and has immediate bedside applicability.

The survey also considered the career prospects of new graduates. Globally, physiologists have good opportunities in academic positions as post doctoral fellows, research associates in research laboratories, and as faculty members. Other professional opportunities are being sought by new PhDs as the struggle to obtain funding support is very onerous. Career opportunities for physiologists in non-academic institutions appear to be good in several countries. There are options in biopharmaceutical companies, biomedical-equipment-related companies, government health programmes, and in science journalism.

An exercise such as this is not merely to take stock of the state physiology is found in, but to offer a route towards improving it. With different countries experiencing very different situations it is not envisaged that these will be universally and identically implemented, but the IUPS and its new Regional Representatives will work closely with the societies to bring about these recommendations.

Recommendations:

1. Societies should advocate for continued funding of basic research and collect evidence to document its state in their country.

2. Networks and working groups should be created, domestically and internationally, by IUPS and member societies to facilitate the exchange of knowledge and best practice in teaching and research.

3. Societies should continue the efforts of the IUPS Outreach Programme to increase support among physiologists for IUPS initiatives and furthering of the World Health Organization's Health for All agenda.

4. Societies should implement outreach activities to raise awareness of and interest in physiology among the public, and encourage the uptake of physiology and related subjects by prospective undergraduate and postgraduate students.

5. Societies should develop resources to improve the teaching and learning of physiology, and to ensure graduates have a full appreciation of the complexities at all scales of physiological understanding.

6. IUPS must oversee a new Global Mentorship-Building Platform to facilitate Mentor/Mentee relationships among physiologists at various career stages, and in academic and clinical settings, to promote dialogue and aid career development.

7. Societies should explore new means to leverage funding from government and private sources, to aid the development of new initiatives designed to strengthen the discipline.

Of course, every IUPS member organisation will do its part for the furtherance of physiology. Here in the UK and Ireland it became apparent that efforts must be redoubled to keep physiology in the public consciousness and to protect the identity of the discipline to maintain a healthy pipeline of skilled students and researchers. To this end, The Society has tried to increase its outward-facing efforts and promote its work to political figures who have sway over the future of science.

While no organisation is yet in the optimum state for driving forward international physiology, there is hope in the future. This report is the first step in a unifying and momentum-raising process to bolster physiology worldwide and achieve its universal recognition as a vital and robust discipline.

Vacation Studentship Scheme: survey of 2013–2015 awardees

Angela Breslin

Education Manager,
The Physiological Society

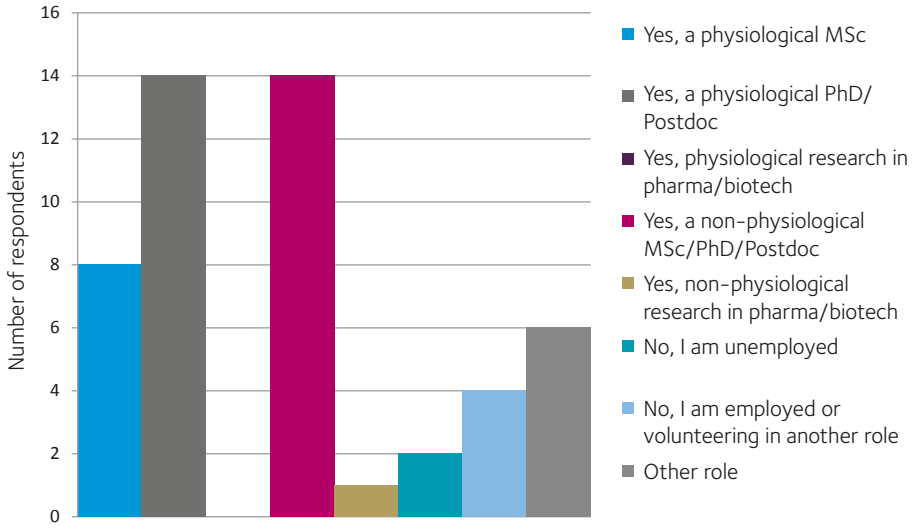
For over 20 years, The Society has been running the Vacation Studentship Scheme (VSS), offering hundreds of undergraduates the opportunity to experience real physiology research over their summer break, and we were keen to find out what impact this has had on their careers. To this end, we surveyed all the students who received a vacation studentship (VS) in 2013-2015 earlier this year, to ask them what they are doing now and whether the VSS influenced their career choices.

Of the 128 awardees contacted, we were pleased to receive 50 responses, and will engage a current undergraduate this summer to gather more feedback in the form of interviews and case studies. Further details about this work will be published in a later issue of *Physiology News*.

With regard to the survey, we were not surprised to learn that 92% of respondents have already graduated and that those who have not are currently studying medicine. Of the 92%, however, it was interesting to see that 84% are still in academia, with a majority studying a physiological MSc/PhD/postdoc (51%) over a non-physiological MSc/PhD/postdoc (33%). We were pleased to see such a significant proportion pursuing physiology after graduation.

From the variety of other routes pursued (i.e. graduate medicine, banking, event management), it's clear that awardees developed a range of transferable skills along the way too. Indeed, 94% of respondents said that the VS was either 'very helpful' or 'quite helpful' in developing their skills more broadly, frequently citing 'communication', 'data handling', and 'team working' as skills they'd developed, to name but a few.

If you have already graduated, are you undertaking any further study or research?



As the VSS is intended to offer a 'taster' of what real physiology research is like, it is to be expected that some students will not pursue a research career, and other professions will benefit from the skills they can offer too. It is far better that they reach this decision after a summer project than halfway through a PhD. One respondent stated, '*It just wasn't the right style of work for me, helped me realise that research wasn't what I would like to do*'.

At the other end of the spectrum, 80% of respondents said that the VS 'improved' their perception of science research, and comments reflected very clearly how much they valued the authenticity of the experience in highlighting the practicalities but also the rewards involved with doing research.

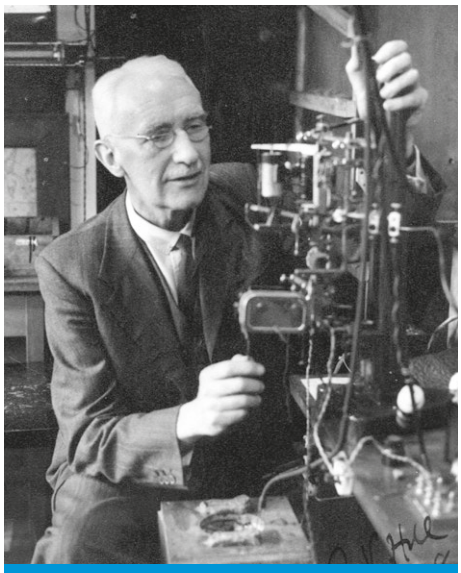
Overall, 72% of respondents indicated that the VS had a 'pivotal' or a 'high' impact on their career plans, again demonstrating how important this scheme really is in informing career choices regarding physiology. Of those who indicated a 'pivotal' impact, it is evident that some students would likely not have pursued research without the VSS: '*This vacation studentship was fundamental to me continuing a career in scientific research and allowed me to gain a PhD studentship*'. One student was even offered a PhD project by their VS supervisor.

Looking to the future, there were some really useful suggestions on how The Society could add more value to the VSS, the most popular of which were, 'opportunities to present the project', 'training and networking opportunities', and 'more awareness'. We do, in fact, offer VS awardees the opportunity to present their project through the Rob Clarke Award scheme, but it's clear from the survey that we need to raise more awareness of this and other opportunities at The Society.

With this in mind, we are making the VSS a Member-only scheme from 2018 onwards, so that VS awardees are kept informed of any opportunities through the regular Member communications. Membership is free for undergraduates, and we will explore other ways in which we can support the VSS in future.

If you'd like to find out more about this survey or discuss the VSS more generally, please contact Angela Breslin at **abreslin@physoc.org**

AV Hill's memoir, *Memories and reflections,* now available digitally



AV Hill

Austin Elliott

University of Manchester, UK

'*Being a physiologist has many advantages, not least that it saves one from believing in magic*' AV Hill, from the Preface to the memoirs

Following his retirement to Cambridge, in his ninth decade, AV Hill collected and edited much of his unpublished, or published but lesser-known, 'ephemera' into an extended memoir that he entitled *Notes and Reflections*. Hill was a prolific writer, both of papers and books describing his scientific work and on many other subjects. A fair bit on these 'other subjects' appeared during his long life, but there was yet more in the form of a mixed bag of reminiscences on his scientific friends and other acquaintances, on his work on gunnery during WW1, and his time as a government scientific adviser, envoy, and MP before and during WW2, and many other topics. Texts of speeches on many of these, and more, feature in *Memories and reflections*, which was bound into three volumes that run to a total of 800 typescript pages.

Copies of *Memories and reflections* were privately circulated, and since the mid-1970s the memoir has been a key source for scholars writing about Hill (e.g. Van Der Kloot, 2011) and for his obituarists like Bernard Katz (Katz, 1978). However, to read it required a Hill family contact or a trip to the Churchill Archives Centre in Cambridge. Given AV's long and varied career and his great importance as both a scientific and historical figure, the Hill family and The Society have been keen to see the memoir more widely available. To this end, the Churchill Archives Centre have now made it available digitally (www.chu.cam.ac.uk/archives/collections/memoirs-v-hill/), with some financial support from The Society's History and Archives Committee.

For Hill aficionados there is much first-rate material in the memoir, which also bears interesting hand-written annotations by the man himself. The material specifically 'badged' as autobiographical has mostly appeared earlier (e.g. Hill, 1970). However, a wider picture of AV and his time and milieu can be gleaned from his own choice of anecdotes about his friends, or his commentary on events he lived through and was involved in. I found much that was new to me, including Hill's comments on sharing a staircase at Cambridge before WW1 with Bertrand Russell, or delivering his brother-in-law, the economist John Maynard Keynes, to Whitehall in 1914 in a motorcycle side car. Another entertaining vignette is Hill in 1947 sending then Prime Minister Clem Attlee a framed sampler carrying the well-known WW2 motto '*Illegitimis non carborundum*' ('Don't let the b*stards grind you down').

The first of the three volumes consists entirely of 'About People', reminiscences mostly of Hill's colleagues and friends. Volume 2 contains collections dealing with Hill's political career and the role of science and scientists in society, plus accounts of his skirmishes with (among others) anti-vivisection activists and spiritualists. Volume 3 is the most eclectic, containing material on Hill's wartime work in both WW1 and WW2, a miscellany chapter, Hill's previously published autobiographical sketch (Hill, 1970), and the collected prefaces of his eight published books.

Memories and reflections should prove a treasure trove for AV fans, and fleshes out the portrait of the man given in Katz's well-known biographical memoir (Katz, 1978) and Hill's main autobiographical account (Hill, 1970). Hopefully members will enjoy reading it.

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Wikipedia Editathon

Update the world's knowledge of physiology and The Society this October

Andy Mabbett

Throughout 2017, The Physiological Society has been working with Wikimedian-in-Residence Andy Mabbett to improve the biographies of notable physiologists on Wikipedia. Find out more about how you can get involved, and our upcoming October Wikipedia Edit-a-thon.

Hello, who are you?

Hello, I'm Andy Mabbett, and I am The Physiological Society's Wikimedian in Residence.

What's a Wikimedian?

A person who contributes to one of the Wikimedia projects – of which Wikipedia is by far the biggest and best known.

What does 'in residence' mean?

I am like an artist in residence, only I can't paint or draw to save my life. I can, though, work alongside (virtually, most of the time, in this case) The Society's staff and members, to help the organisation to understand Wikipedia and its sister projects, to work with the communities who build and maintain them, and to advise on which parts of The Society's work and resources can be used to transfer knowledge to them, to support the Wikimedia ethos of making '*a world in which every single person on the planet is given free access to the sum of all human knowledge*'.

Can you tell us a bit more about Wikimedia and it's community?

Well, that's going to be a long answer. You might want to get yourself a cup of tea...

Since its inception in 2001, Wikipedia has grown to be the world's largest volunteer-led project, and its biggest on-line repository of knowledge. All the content (apart from a small proportion of its images) are freely available for use and reuse. Even if you don't know it, you use Wikipedia frequently – its content informs and is included in Google search results, the answers Siri, Alexa, and similar products give you, and on third party websites as big as the BBC's.

And its not just in English. At the time of writing, there are 299 Wikipedias (the number is growing, slowly) in different languages, some large (French, German, Russian, and so on), some small (Cornish, Punjabi), and some in endangered languages you've probably never heard of. The Welsh Wikipedia is reportedly the world's largest website in that language. Between them, these Wikipedias have over 45 *million* articles.

Wikipedia's topics cover everything from bhangra to brain surgery (and every other aspect of science), and from beer to biographies – including, of course, biographies of influential physicians and physiological scientists.

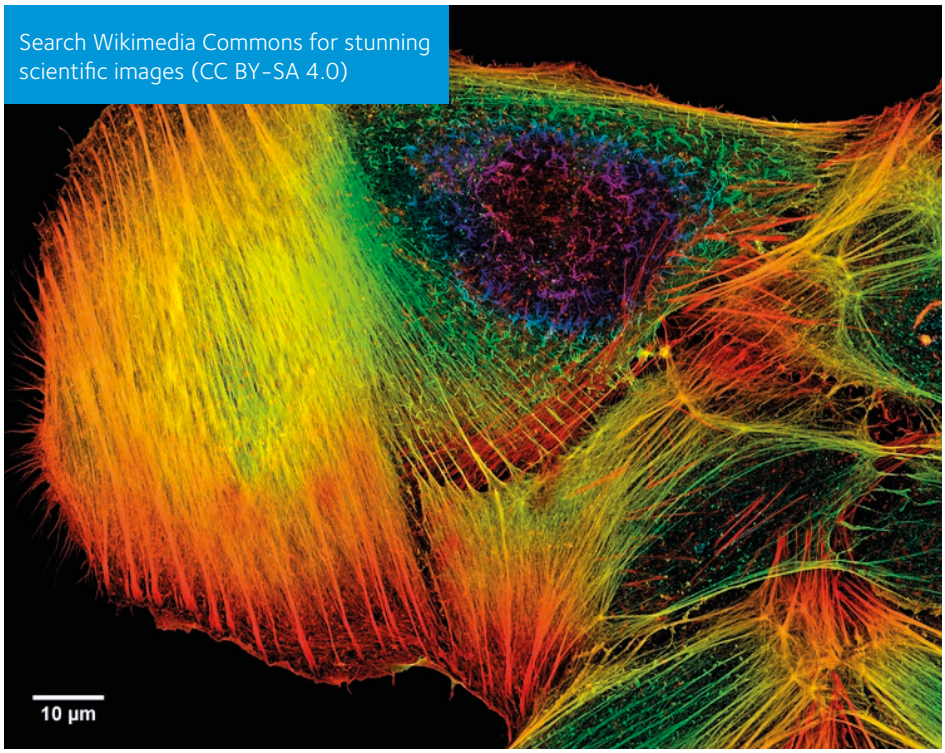
All these Wikipedias are hosted by a United States non-profit organisation called the Wikimedia Foundation. They administer computer hardware and connectivity of mind-boggling complexity, and collect and disperse funds raised through donations (if you have already given: thank you!) – Wikipedia has no advertising. The content, though, is supplied and maintained by people who write and edit content, take pictures, liaise with museums, learned societies, and other bodies, write software, and deal with administrative matters. These people collectively, are the 'Wikimedia Community'.

But it's not just Wikipedia that the Wikimedia Foundation and Community give to the world. Most of the images, video, and audio files, for instance – over 40 million of them, in fact – are gathered on a single site called **Wikimedia Commons** from where the other projects can include them on their pages – but anyone else can use them too, for free, just like Wikipedia's text, under what we call an 'open' licence.

Then there's the newest, fastest growing project, **Wikidata**, which is a repository of linked open data – a huge database of statements about the entities that Wikipedia writes about, and more, which can be displayed on Wikipedia pages, or accessed by any computer programme, for use in apps, on websites, or to answer complex queries. Want a list of all the cites in the world with female mayors, ranked by size of population, or displayed on a map? A few lines of query code can give you that, bang-up-to-date, *in under a second*.

There's **Wikisource**, a repository for out-of-copyright and openly licensed texts, whether that's a work by Chaucer, a 200-year-old poem in Indonesian, or a newly published open-access scientific paper.

Add to this **Wikiversity** (learning modules), **Wikiquote** (notable quotations), **Wikibooks** (ebooks), **Wiktionary** (multi-lingual dictionary and thesaurus), **Wikivoyage** (travel guide), and more, all with openly licensed content, and you have a vast, multi-lingual, collection of interesting, educational, historic, and useful knowledge.



Search Wikimedia Commons for stunning scientific images (CC BY-SA 4.0)

How can The Physiological Society and its members contribute?

That collection is far from complete. The Wikimedia movement relies on volunteers to find the gaps and fill them, and to improve and update the existing articles. That is where members of The Physiological Society come in. They can, of course, write articles about their favourite films, update those on the sports teams they support, or add details of the features of their home towns. But the maximum impact comes when people contribute their specialist knowledge, whether that means improving articles on drugs, surgical techniques, or how the body works, or knowing who the influential physiologists of the past are whose biographies are needed to complete the stories about the discoveries and innovations for which they were responsible.

Some Society members will be involved in higher education, and a great deal of work has been done to help people like them to use Wikipedia in their teaching. For example, last summer I taught a Wikipedia course for PhD students at the Politecnico di Milano (the Polytechnic University of Milan), who were studying a variety of science-related topics, as part of a cross-disciplinary programme. The students wrote articles related to their studies, which were marked by academics from the university, just as an essay or dissertation would be assessed. The event was so successful that it is being re-run in 2018. Feedback from academics involved in a similar course for MSc students on a part-time, three-year programme at the University of Birmingham came with a request to change from teaching Wikipedia editing in year three to doing so early in year one. Why? Because once the students knew how to add citations to Wikipedia, the referencing of their academic essays improved markedly!

As part of my work with The Society, I have been adding to Wikipedia (that is, the Wikipedia in English) lists of all the people who have been honoured with one of The Society's awards. I have listed them all on the article about The Society, which shows those who have an article in Wikipedia as blue links, and those where no such page exists as a red link. I will be working with The Society and its members on turning as many as possible of those red links into blue ones.

We'll be working on that, and on improving other articles, at an event on Friday, 13 October. At this 'editathon', assistance in editing Wikipedia and Wikidata will be available for people who have never done so before. There will be access to resources from The Society's archives, refreshments will be served, and people will be able to contribute in a supportive, friendly environment. Booking is essential, and participants will need to bring a laptop that can use the venue's Wi-Fi.

But people who do not want to write Wikipedia articles can also contribute in other ways. Copy editing, fact checking, and adding citations all help. There is also a need for photographs to illustrate articles. All the Wikipedias, in different languages, share images (and video and audio recordings) which as noted above are hosted on Wikimedia Commons. Commons only accepts files that are under an 'open' licence, allowing anyone to reuse them, and requiring no more from them than they give due credit ('attribution') to the copyright holder. Perhaps you can take a picture of the building where you work, or the equipment you use? Take a look at what's already on Commons to get a flavour for what's included. Or maybe your employer has an image bank which is gathering dust (or whatever it is that digital archives gather) and could be persuaded to share those. They could be scientific images of microscope slides or patients' visible symptoms (duly anonymised and with their permission, of course), or portraits of notable people, or they could illustrate types of equipment or procedures. Zeiss microscopes did this and you can see their superb images in Commons. Do not forget, too, that you can equally use images from Commons in your own work.

Or perhaps you'd be more at home transcribing scans of old documents for Wikisource? It's a nice, light task that can fill an odd coffee break or lunch hour, and as with Wikipedia, expert knowledge is sometimes needed, for example to understand an only partially readable drug name or other scientific term.

Whichever way you find to contribute, your input will be gratefully welcomed, and you too will be helping to build '*a world in which every single person on the planet is given free access to the sum of all human knowledge*'.

Register now to join Andy and other Society Members this October at Hodgkin Huxley House for a training and editing event. The event will focus on physiology-related topics, including both scientific and non-scientific content (the latter including biographies). We will work on Wikipedia, but also sister projects including Wikimedia Commons, Wikidata, Wikisource.

Participants will be able to access some resource material and staff will be able to provide access to papers from The Society's journals. If there is a biography of a notable physiologist or topic area you are keen to work on please bring reference and source material relating to your subject of choice.

We will also hear talks on the relationship between academics and Wikipedia from Duncan Hall, and about the efforts made at the University of Edinburgh to increase the profile on Wikipedia of the 'Edinburgh Seven', the first group of matriculated undergraduate female students at any British university, from Melissa Highton.

www.physoc.org/physiologyonwikipedia/physiology-wikipedia

2017 *Forthcoming events*

30 Nov.

H³ Symposium: Muscle Physiology and Metabolism
Hodgkin Huxley House, London, UK

www.physoc.org/musclephysiologyandmetabolism/

1 Dec.

H³ Symposium: The Integrative Physiology of Physical Inactivity Across the Lifespan
Hodgkin Huxley House, London, UK

www.physoc.org/inactivity/

8 Dec.

H³ Symposium: Sensory Transduction in Insects
Hodgkin Huxley House, London, UK

www.physoc.org/sensorytransductioninsects/

13-14 Dec.

Future Physiology
University of Leeds, Leeds, UK

www.physoc.org/futurephysiology/

Meeting Notes

H³ Symposium: Glio-vascular Coupling

12 May 2017,
Hodgkin Huxley House,
London, UK

Lucinda Craggs

Centre for Biomarker Research (CeBioR), University of Huddersfield, UK

I picked up the flier for the glio-vascular meeting whilst attending the British Neuroscience Association's Festival of Neuroscience (10–13 April, Birmingham) and immediately knew it was the kind of focused meeting that could provide me with a real value for money experience. I am so glad I found that flier, and that I made the decision to not only submit an abstract to the meeting, but also tick 'yes to oral presentation' during my submission. I recently returned to academia after a short career break working as a Medical Writer, and I have been fortunate enough to have been able to pick up my past work in neurovascular degeneration in small vessel disease (SVD) of the brain, despite moving institutions and research groups. I am currently in the early stages of developing an *in vitro* model to study the neurovascular unit, so this meeting was perfectly timed and a prime opportunity to get direct feedback on our proposed project from experts in the field.

As fate would have it, the first speaker was the ever-gracious Professor Mária A Deli (Hungarian Academy of Sciences, Hungary), a world leader in developing an *in vitro* model of the blood–brain barrier (BBB). Only two weeks prior, we had been discussing her papers in our laboratory group meetings and now I had the opportunity to hear Mária describe her techniques and the functionality of the model directly. Pure serendipity. Mária gave an excellent overview of her co-culture model of the three major cell types in the blood–brain barrier: endothelial cells, astrocytes and pericytes. Mária also spoke on some of her group's recent work investigating mutations in the MFSD2A gene, the sodium-dependent lysophosphatidylcholine (LPC) transporter. MFSD2A is expressed in the endothelium of the blood–brain barrier, where MFSD2A mutations impaired transport activity in a cell-based assay. Her work implies that the leaky BBB in MFSD2A-knockout mice could be a result of defective transport of LPC's into the BBB endothelium and raises the possibility that LPC's have a role in membrane integrity and function at the BBB.

Following Mária, the second plenary speaker of the day was France's Nathalie Rouach (College of France, INSERM, France). An expert in astrocyte physiology, Nathalie gave a detailed overview of her extensive efforts to delineate the many regulators of astrocytic function, morphology, and structure. Nathalie began with a detailed overview of the role of astroglial-expressed connexin 30 (Cx30) and its support of neuronal synapses. Her group have identified Cx30 as a key regulator of synaptic strength during hippocampus-based memory formation, revealing a critical role for Cx30 in maintaining the close relationship

Mark Dallas seeks answers from Shereen Nizari

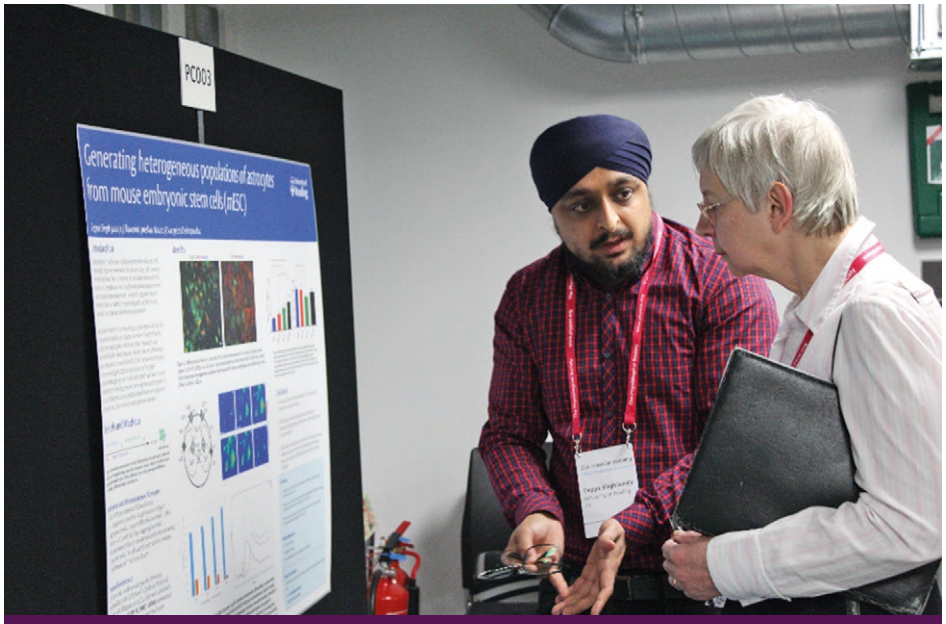
between astrocytic processes and neuronal synapses through intimate contacts made within synaptic clefts. Nathalie went on to describe the different morphologies of astrocytes and her very recent work identifying the novel role of FAT1 in the regulation of astrocytic morphology, reporting that FAT1 expression controlled elongation and ramification of astrocytic processes.

Following the two plenary talks were ten short presentations given by early career researchers (ECRs), split into two main categories: firstly vascular-focussed and then glia oriented presentations. We also had an informal poster session in the afternoon allowing even more ECRs the opportunity to present their work. Highlights from the vascular session included the *in vivo* monitoring of the neurovascular unit in mice during exercise and the finding that blood flow to visual cortices was maintained during exercise without any visual stimulation (Orla Bonnar, University of Sussex, UK), and the surprisingly understudied role of vascular factors in age-related hearing loss (presbycusis), elegantly described by Llwyd David Orton (Manchester Metropolitan University, UK).

Representing the neuronal and glia side were many more interesting talks, including Shereen Nizari (The University of Southampton) and her work investigating the link between neuronal signalling and the cerebral vasculature, AKA the neurovascular unit (NVU). Shereen reported that perivascular innervation by cholinergic neurons regulates vascular tone, highlighting the link between components of the neurovascular unit (NVU) and cholinergic loss in neurodegenerative disorders such as Alzheimer's disease. Perhaps one of the most ground breaking ECR presentations was given by Noémie Mazaré (College of France, INSERM, France), where she described her group's work to define the mechanisms by which astrocytes integrate and regulate neuronal and vascular signals through protein synthesis in their processes. They have worked to investigate the 'endfeetome' of astrocytic endfeet, where mRNA translation occurs in astroglial perivascular endfeet allowing protein delivery in a spatially controlled manner at the astrocytic–perivascular interface. Their work reveals a completely novel view of the way astrocytes communicate with the vascular bed, confirming the existence of local protein translation and maturation in astroglial perivascular processes.

The grand finale of the day was the debate 'Glia vs Vascular: who's in control'. While both Mária and Nathalie gave convincing overviews of their side of the fence, both were in agreement that with the advent of novel technologies, glio-vascular research is enjoying a revival, where old ideas and findings can be re-investigated and further analysed in ways which were not previously possible. Mária made a prediction that the new 'fashionable' research areas might include microvesicles and their role in communication between cells of the BBB. There was also some discussion of the 'gaps in the market', where Professor Joan Abbot (King's College London, UK) advised ECRs that there is much opportunity for young researchers to find a niche and develop novel specialities.

I found all the presentations to be relevant in some way to my work, and truly valued the opportunity to present my own research in a supportive and engaging setting. I highly recommend attending such small and focused meetings where world leaders in the field were more than willing to engage with and educate early career scientists. A major benefit of the day was being able to speak with several other researchers who perform similar work to myself, allowing me to broaden my research network and hopefully develop new collaborations. My personal highlight was being able to speak with Mária Deli about my project aims and her invitation to join her in the lab to learn her co-culture techniques directly from her research group. In all, I found the day incredibly valuable in terms of the direct feedback and advice I received on my work, but it was also very inspiring to be able to meet with and speak directly to key opinion leaders in the field in such an informal and friendly environment.



Joan Abbott quizzes Deppo Singh Juneja on his poster

Meeting Notes

The Physiological Society Annual General Meeting 2017

12 July 2017,
Wellcome Collection,
London, UK

Roger Thomas
Scientific Editor, *Physiology News*

Sam Scott of Liverpool John Moores University explains his poster to Holly Shiels and Mike Tipton

Since there was no UK or Irish main meeting this year, the AGM was held in the luxurious but chilly Henry Wellcome Auditorium underneath the Wellcome Collection building in London. The seats were upholstered in four different shades of purple. Jonathan Ashmore was elected as AGM Chair, and after approving the 2016 minutes and remembering deceased members he announced the names of the three new trustees (Charlotte Haigh, Elizabeth Sheader, and Stefan Trapp) elected by the 161 members who actually voted. The numbers of votes cast for each candidate were not given. He then thanked the outgoing members of Council and announced the three new Honorary Members of The Society: Peter Raven, Michael Kane, and Terje Lomo. The President, David Eisner, then drew our attention to items from the 2016 Annual Review, which had been sent to all members, and announced that the next main meeting (and AGM) will be held from the 14 to 16 September 2018, at the QEII Centre in London. He was followed by The Society's new Chief Executive Dariel Burdass, talking mainly about our future plans. These were aimed at ensuring that physiology is flourishing. There will be a focus on under-represented areas. The Massive Open Online Course (MOOC) in Physiology will be launched in September, and our policy and public engagement programmes will be strengthened.

The Hon. Treasurer Anne King then made her last presentation of the 2016 Accounts. Income had risen by 10% or £390k, but expenditure had increased too, by 11% or £440k. Assets had increased, and investments had performed well. Indeed she had enjoyed her term as treasurer and felt that all was well with The Society's finances. Finally she announced that the membership subscription rates did not need to be increased. Her successor is Frank Sengpiel.

Following changes to the articles of association, we then were asked to consider a motion proposed by David Wyllie and supported by many members from Scotland. This proposed that any future General Meetings not part of a main (scientific) meeting should be broadcast, with arrangements for members to participate remotely. Their view was that meetings were too expensive for members to attend in person. We were told that such an arrangement might cost The Society at least £8k. After a brief discussion, however, the motion was put to the vote, and passed with no votes against it. When I spoke to several Members present following the AGM they expressed regrets at the lack of a main meeting associated with the AGM, but the original decision not to have such a meeting was agreed by a majority of the members consulted.

After the rare excitement of an actual vote, the meeting heard presentations about The Society's four publications. The Editor-in-chief of *The Journal of Physiology*, Kim Barrett, reported that it registered over 3.5 million article downloads in 2016, and was available at 5390 institutions worldwide by subscription or licence. It was available also at 7711 institutions in developing countries thanks to the publisher's philanthropy. It was number one in Eigenfactor score in its subject area. In short, it was a very successful journal and still refused to impose page charges. Kim reported that she had participated in an 'Ask Me Anything' session on Reddit, and had enjoyed it much more than she expected. Mike Tipton reported that *Experimental Physiology* had recently restructured its Editorial Board and has an increasing number of full-text downloads. The Society's newest publication (jointly with the American Physiological Society) *Physiological Reports* is doing remarkably well as reported by Susan Wray, who is soon to step down as Editor-in-chief to be followed by Tom Kleyman. Most of its papers are submitted as transfers from other journals, but last year there were over 100 direct submissions. The fourth publication is *Physiology News*, in which you are reading this report.

The AGM overran its allotted time by 15 minutes and was followed by a tea break in a nearby room in which posters by selected affiliate members were displayed. There were then four oral communications by affiliates. Prizes for the best two of these and of the posters were given. Congratulations to Rebecca Neal and Sam Scott for winning the poster competition, and Victoria Meah and Annabel Taylor for the oral competition.

The meeting concluded with the inaugural 'President's Lecture' by Eric Olson, The University of Texas Southwestern Medical Center, USA, on muscle, which he claimed to be our most important tissue. To those who think that is really the CNS he pointed out that muscle provides the only output available to the nervous system and comprises almost half the average human body weight. His talk started with small regulatory peptides that modify SERCA pumps and ended with a fascinating account of his work on Duchenne Muscular Dystrophy with CRISPR methods.

Meeting Notes

IUPS 2017: The Rhythms of Life

1–5 August 2017,
Riocentro,
Rio de Janeiro, Brazil

Aline R. Bezerra Gurgel

University of Glasgow, UK

The IUPS 2017 World congress covered a four-day, first class programme of plenaries, keynote lectures, and 60 selected symposia with over 300 speakers. Held for the first time in the beautiful city of Rio de Janeiro, Brazil, this event was an exceptional opportunity to meet experts, leaders in the field of Physiology, and take part in an important scientific exchange among researchers from several international institutions.

Opening the conference, which was also the Annual Meeting of The Physiological Society, and of the Scandinavian Physiological Society, the president of IUPS, Denis Noble, gave an illuminating talk entitled 'Dance to the rhythms of life: physiology returns to centre stage', focusing on the study of biological functions. Following, Ada Yonath, from Israel, splendidly spoke on resistance to antibiotics. The first day was then closed with the celebration of genuine Brazilian rhythms, considering the cultural diversity of that country: melodies from samba to local indigenous music. The Brazilian Society of Physiology was also present and celebrating its 60th anniversary.

As a Brazilian PhD student, I was delighted to experience this unique moment in my home country. Captivated by the 'Rhythms of life', the audience was involved in a pleasant atmosphere, in a venue that comprised beautiful pieces of local fauna and flora, several comfortable seminar rooms, and a nice structure to accommodate posters - from an extensive range of topics - and delegates.

Since I am working on exercise training in heart failure, the conference provided an ideal platform to discuss my recent findings, methods, and design of study. The event contributed to promoting my critical thinking and provided space for a constructive dialogue and collaboration on relevant topics of my research. Gender equality and representativeness of women in science were also debated in a two-day session, an occasion when we could compare realities of female scientists in different countries.

Subsequently, the final day was opened with a memorable lecture by David Eisner, who spoke on calcium dynamics in the heart. After the talk, Denis Noble awarded him with the annual prize review, a moment that the delegates joyfully acclaimed. A prestigious group of other speakers also brightened the conference, contributing to remarkable sessions.

After these very productive days in Brazil, I come back to my lab with innovative and creative ideas. I would like to thank The Physiological Society for kindly awarding me with a travel grant, helping to support this fantastic opportunity.

From the Archives: reports by the Meetings Secretary JS Gillespie of the Oxford meeting of 1967

Transcribed by Roger Thomas

The Physiological Society's
Oxford Meeting,
7-8 July 1967

At the invitation of GL Brown, a meeting of The Society was held in the University Museum and in the Physiology Laboratory, Oxford, on the 7th and 8th July 1967.

The meeting is understood to have started at 11 am on Friday, with GL Brown in the chair, though at that time the Secretary and many members were the enforced guests of British Railways somewhere outside Didcot.
A leisurely beginning, however, the first communication going four slides and two rings of the warning bell beyond its allotted span, allowed everyone to catch up. Six papers were heard before lunch, and again with GL Brown in the chair, a further nine after lunch. Paper 10 was withdrawn, with the result that later authors, secure in their faith in the chairman's timing, were found wanting - or rather, could not be found. The confusion was resolved by taking paper 17 before paper 15, and postponing paper 6 until Saturday morning. Tea was followed by Demonstrations in the Physiology and Pharmacology departments. Demonstration 19 was withdrawn.

Members and guests were then entertained to sherry in Keble College, followed by a most excellent dinner there. After dinner, OF Hutter thanked GL Brown for an excellent meeting. As this was their chairman's last meeting as head of the Oxford department, he wanted, on behalf of the whole Society, to express our very best wishes to GL in his new career as Principal of Hertford College. This was enthusiastically applauded by all. In his reply, GL Brown welcomed The Society and its guests, in particular Dr H Korn from Paris, who was an official guest of The Society.

He directed The Society's thanks to those who had done most of the work, particularly his secretary Mrs Audrey Richards, and also to RH Kay for organising such a splendid lunch and dinner in Keble College. Though this would be his last opportunity of welcoming The Society as head of department, he hoped his new job would give him an opportunity of actually doing some physiology.

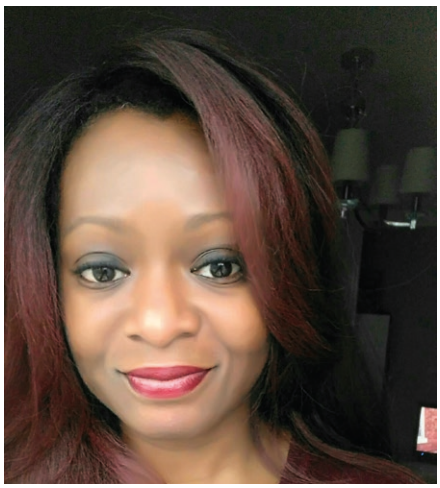
The meeting resumed at 10.00 am. on Saturday, when seven papers were heard before and the seven remaining after lunch. The meeting ended at 4 pm with tea.

Friday, 7 July
Lunch 120 – Tea 326 – Dinner 197

Saturday, 8 July
Lunch 146 – Tea 206

The rise in eminence of the pseudo 'exercise guru' in social media

A threat or an opportunity for HE researchers?



Gladys Onambele-Pearson

Manchester Metropolitan University, UK

Kostas Tsintzas

University of Nottingham, UK

In March of this year and in our capacity of co-leads for the Human and Exercise (HE) theme for The Physiological Society, we received a request from the *Physiology News* editor to write an article of current prominent importance to HE Physiologists. Admittedly, there is such a wide range of topics and interests in our HE theme that no consensus on a physiology topic per se could be had. We then settled on a topic we felt could cover the majority of the HE theme interest and decided to write a comment on our perception of, dare we say, the increasing trend for 'alternative truths' in the general area of human exercise physiology and nutrition.

Why is HE physiology particularly prone to 'self-labelled gurus/authorities/dogmas' in the subject? It may be to do with the widespread use of social media and the rise in the appreciation for an 'athletic' looking body and 'optimal' eating (to achieve this proclaimed body perfect) becoming less niche and more of a norm. Personal trainers, whatever their background (be it formally trained exercise scientists, or simply exercise and nutrition enthusiasts, including celebrities with little formal training), are establishing themselves as the voice of authority in all matters relating to exercise protocols, enhancing lean body definition, and optimising training regimes to include the type of nutrition perceived as necessary to achieve the desired goal.

Lessons from the 'exercise guru'

These gurus would tend to highlight the benefits of healthy eating over indulging in a poor diet, and of exercising in a manner that minimises boredom as opposed to relenting to being sedentary. Our observations are that these gurus are very enthusiastic, passionate even, about their message, utilising a wide range of media (Facebook, personal blogs, Twitter, Instagram) to reach their audience and clients. They will speak in terms that are easily assimilated by the majority, they will respond promptly to queries, and will sometimes (not always) use references to the scientific literature to support their claims. Of particular concern is the fact they will often simplify a large body of scientific evidence. A case in point is a quote from a 'guru' to the effect that 'Branched-Chain Amino Acids/BCAAs will improve your physical performance and reduce muscle breakdown, thus enhancing exercise recovery'. This statement is succinct (hence attractive to the general public). It is, however, not an entirely accurate summary of the effects of BCAAs in view of the lack of evidence in the literature on the ergogenic effects of BCAA supplementation before or during exercise (Bishop, 2010), and the inconsistency of their effects on delayed onset muscle soreness (DOMS) and recovery of muscle function following exercise (Jackman *et al.*, 2010; Shimomura *et al.*, 2010). Although it is true that BCAAs

affect muscle protein turnover and relevant signalling pathways after acute exercise, there is no compelling evidence coupling events observed at the cellular or molecular level after acute exercise with chronic functional adaptations (ergogenic, enhanced recovery from exercise, hypertrophy) following a prolonged period of physical training.

What is also striking is the maintenance of the rapport with the audience through addressing each member directly by their name, and often following their progress through the 'body perfect lifestyle' journey, supporting them with positive comments and encouragements via individual emails. Their reach is wide, indeed many counting their 'communities' or subscribers in the tens and even hundreds of thousands, and as such, more than many scientific papers would normally dream of directly reaching in terms of readership. What's more, the training regimes advocated often cater to one who uses a gym, or prefers outdoors exercise or equipment-free workouts in their own home, thereby, together with regular 'check-ins', arguably increasing the lifestyle protocol adherence potential.

We would argue that research physiologists in academia tend not to engage directly with the public in this way. When researching healthy eating, we are aware of and would present the pros, the cons, and the limitations of any recommendation that our published data highlights. Where we disseminate publically our findings, it will primarily be through scientific journals in a language that would tend to automatically alienate a large segment of the population through its academia-specific terminology. Where we do disseminate to lay audiences, our message is always tempered, as we are (rightly) unwilling to make conclusions appear definitive, which then has the counter-productive result of leaving the layperson unsure of what actions to take from our message. To date, there is only a minority of researchers engaging fully with social media, though the general trend is for increased recognition of the need to engage in this way to make more people (scientists and lay public alike) aware of our findings. Indeed, although an increasing number of scientists are using Twitter to disseminate their publications and build their network of colleagues, this engagement, however, is still tentative as there are no academic institutions that (yet) reward their scientists for the number of Twitter or Instagram followers, or indeed hits on their Facebook page or online Blog. On the other hand, an increasing number of journals are active on Twitter and collect relevant statistics, and some even require their authors to submit tweetable abstracts. However, there is a weak correlation between tweets and eventual citations (Haustein *et al.*, 2014), potentially explaining the social media dis-engagement of researchers. In the end, we, as research physiologists in academia, put our energies in those activities that demonstrate the type of external esteem likely to have an impact on our career development. However, we envisage a substantial number of scientists as active users of social media outlets in the near future, which will proliferate the dissemination of evidence-based messages on healthy eating and 'efficient' exercise regimes to lay audiences. It is of critical importance though that scientists develop specific communication skills to enable them to convey their messages in a way that can be easily assimilated by the majority and then applied to everyday life.

Where the 'exercise guru' may be undermining what human & exercise physiology research shows

In looking at a number of 'guru's social media postings', our other thoughts were: how do they check on the veracity of their statements? Are they happy to use anecdotal reports as evidence enough? What formal qualifications do they have to enable them to formulate these exercise programs and nutrition/supplementation protocols or is life experience adequate enough in this context? Indeed, if we are looking at the average member of the public who exercises little if at all, what good is it to them to understand the difference between training at longer muscle length versus shorter muscle length (McMahon *et al.*, 2013) when the first thing they should consider is the need to exercise on a regular basis particularly when starting from a baseline of high habitual sedentary behaviour? If another member of the public eats a poorly balanced diet, what good is it for them to distinguish between the benefits (or otherwise) of correctly timing their intake of various micronutrients when they should first understand how their habitual diet can be realistically tweaked to be more conducive to good health? These latter thoughts would probably explain why the apparent simplistic approach of the 'exercise guru' appeals to the lay audience.

Nevertheless, without wishing to vilify the 'exercise guru', here are 10 typical, arguably repressible, behaviours that we have gathered from a small sample of four 'exercise gurus'.

1. Some of the gurus could have laudable credentials. Our case studies include one with a 'Masters and Bachelors in Exercise & Nutrition Science, and over a decade of experience in physique transformation'; a 'certified exercise instructor'; a 'certified Strength and Conditioning Specialist'. Other credentials may not be justifiable for such a position of influence including being a 'fitness model', or a 'bikini model'.

'It is of critical importance though that scientists develop specific communication skills to enable them to convey their messages in a way that can be easily assimilated by the majority and then applied to everyday life'

2. Outlandish claims are not off-limits. One claims to help female clients 'manipulate their menopausal symptoms or their menstrual cycle to help you get rapid and long-lasting results (in a matter of 60-90 days) with their body image goal or dream beach body'. The program pertains to 'provide key principles on how to attack body fat...using science'.

3. Listing and directly providing links to 'the best nutrition supplements' is almost synonymous with the territory. Demonstrating the clear proof of the definitive advantage of supplementation in the first place and, further, of the chosen supplement over any other supplement brand, does not appear to be a consideration.

4. Gurus are adept at gaining the confidence of the public through not only and with almost no exception presenting with the 'goal physique themselves', but also reassuring that any financial investment in their program (through subscriptions/ purchase) would be 'completely refunded'.

5. Proving that a program works is normally through self-reports of previous clients (though the evidence is not checked), and where the program has not worked, the prospective client is told that this would only be the case where they have not in fact followed the plan to the letter.

6. Strong, definitive statements are made: 'protect your hormones'; 'carb cycling to boost your metabolism and protect your thyroid'; 'Carb cycling ... keeps the body in a fat burning state'; 'we will teach you how to make changes, what supplements you should take'; 'supplement x is specifically designed according to the macronutrient and micronutrient requirements of the female body'; 'One of the worst enemies for men and women is high levels of oestrogen'; 'we want to be in great shape ... and eliminate fat'; 'not taking fish oil makes you overweight and lazy'; 'I only recommend supplements that truly optimize your life'; 'Fish oils make you leaner'; 'Start supplementing with ~1,000mg of fish oil 3x per day. In fact, I sometimes recommend much higher'; etc. You probably see where we are getting at, with many of the claims, the statements are at best partially true and may sometimes only apply to persons with pre-existing conditions.

7. Calls to revolt against the establishment (read mainstream scientific reports) in favour of listening to the 'good guys' (read 'the guru' him/herself) are made in 'live Facebook feedback streams' by impassioned 'gurus' commanding a large audience.

8. Claims of endorsement are listed with no evidence: 'this program is used by Hollywood celebrities, professional athletes, 1000s of people'.

9. The research that is referred to (if any) in the blogs from our case studies is only that which agrees with/supports the stance of the blogger. One such example is that used by a blogger who advocates the use of high levels of fish oil and quotes a single paper in which the effects were, to say the least, statistically weak. For example, reduction in fat mass (-0.5 ± 1.3 kg in treated versus +0.2 ± 1.2 kg in the placebo group, p = 0.04), and a tendency for a decrease in body fat percentage (-0.4 ± 1.3% body fat in treated versus +0.3 ± 1.5% body fat in the placebo group, p = 0.08). Furthermore the link between fish oils and increased lean mass is often seen in clinical (usually cancer) populations exhibiting sarcopenia/cachexia but not necessarily in young/healthy populations (McDonald *et al.*, 2013). Similarly with the recommendations linked to high doses of fish oils, the balance between omega 3 and 6 is the key factor for safety here, as well as the presence of any pre-existing conditions (Ergas *et al.*, 2002).

A need to recognise the value of, and reward engagement in, social media interaction for HE physiologists?

It may be that, and we would propose, there is an inverted snobbery against social media for physiology researchers and the tide may be for the turning. In an era of 'alternative facts', is it not our responsibility that the true (scientifically evidenced) message is made public rather than allowing rampant myths to be propagated and not challenged by those with access to the wider public, who are, in the end, our target audience? We would propose that we are in a privileged position whereby a lot of our research can and does have direct application pipelines and as such should reach the end-user directly from us, as we (supposedly) understand it better than any intermediate party does. To do so, we will need to acquire the communication skills necessary for the creation of 'true science' blogs, with the type of enthusiasm, responsiveness, and application of 'results' promoted by the 'exercise gurus', which will then give the public what it deserves: nuanced recommendations based on systematic and reviewed evidence. For instance, teaching that where supplements are concerned they should be exactly that: supplements and not substitutes for other food stuff.

Is the correlation between tweets and citations around the corner? In an era where celebrities become self-styled 'exercise gurus', is it appropriate for scientists to become celebrities themselves as Brian Cox has recently proclaimed? First things first: a workload model that accounts for social media interactions anyone?

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The impact of stress on pain

Considerable overlap in the neural substrates and circuitries of stress and pain

David Finn

Professor of Pharmacology and Therapeutics, School of Medicine, Centre for Pain Research and Galway Neuroscience Centre, National University of Ireland Galway, Ireland

Pain and stress are two phenomena that most of us would rather not have to deal with throughout our lives! And yet both pain and stress have essential survival value for humans and other animals.

The ability to mount an appropriate response to stress is critical to allow us to cope with the physical and psychological challenges that life throws at us. Stress responses that are either sub-optimal or excessive can be maladaptive, leading to the development of illness, either physical or psychiatric (e.g. anxiety, depression). Pain, on the other hand, is an essential warning signal to potentially harmful or injury-producing stimuli. It facilitates learning in early life, and encourages rest and healing following injury. Its survival value is perhaps best illustrated through the very rare condition of congenital insensitivity to pain. Individuals with this condition are unable to feel pain from birth due to loss of function mutations in key proteins required for nociception (e.g. the sodium channel Nav1.7), but they typically die at a relatively young age due to an inability to detect and respond appropriately to critical illness, injury, or accumulation of injuries. However, while the ability to perceive acute pain is essential for survival, when pain persists longer than is physiologically necessary, it can become pathological and reduce quality of life. About 20% of the adult population of Europe have this type of chronic persistent pain (Breivik *et al.,* 2006). Chronic pain is a complex and diverse disease state which can be very difficult to treat (about 40% of patients report their pain medication as inadequate; Breivik *et al.,* 2006) and places a massive burden on the individual patient, on society, and on economies.

Stress-induced analgesia

Pain can be affected profoundly by stress. The impact of stress on pain usually depends on the nature, duration, and intensity of the stress, and also on the type of pain. Generally, stressors that are acute and intense, an immediate threat to homeostasis, result in a short-term suppression of pain known as stress-induced analgesia (Butler & Finn, 2009). Stress-induced analgesia has been widely studied and is part of the body's defensive 'fight or flight' response. Thus, when a person is in a potentially dangerous situation, the body temporarily dampens down pain signalling in order to allow escape or coping, regardless of injury. A lot of good work over the past 50 years has illuminated our understanding of the physiology of this important form of endogenous analgesia. We know, for example, that it is mediated by activation of the descending inhibitory pain pathway. This pathway is comprised of neurons that project from higher cortical areas and the amygdala to the midbrain periaqueductal grey, which in turn projects to the rostral ventromedial medulla, from where neurons descend to the dorsal horn of the spinal cord. Activation of this descending pathway by stress or fear inhibits ascending nociceptive transmission. We also know from the pioneering early work of Akil, Madden, Patrick, Barchas, Fanselow, Liebeskind, and co-workers that stress-induced analgesia is mediated by the endogenous opioid system.

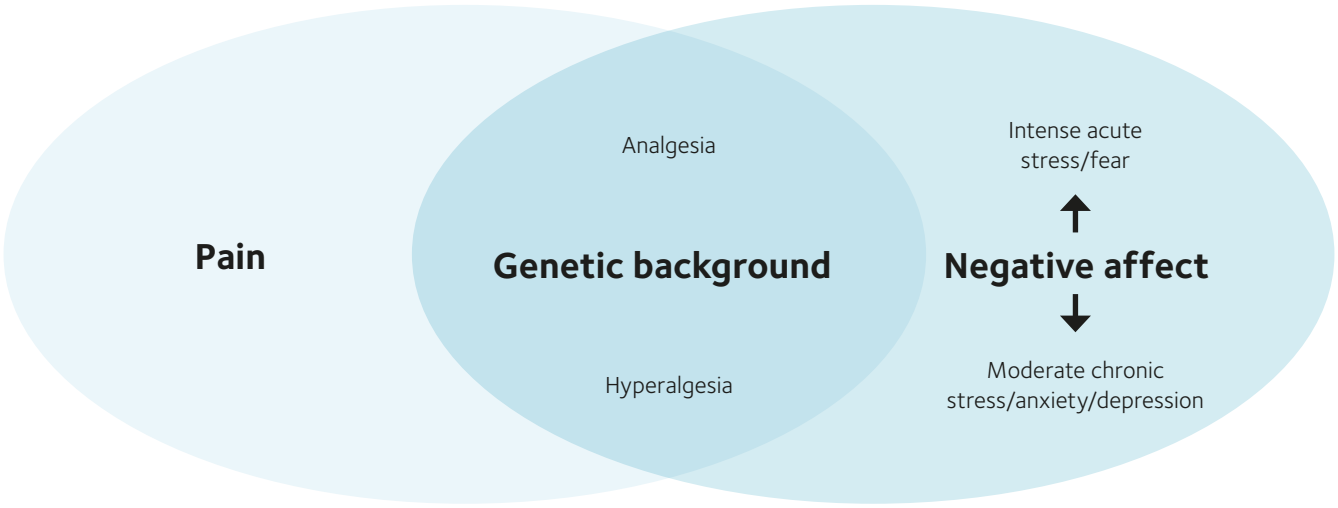

Figure 1. The impact of stress and negative affect on pain.

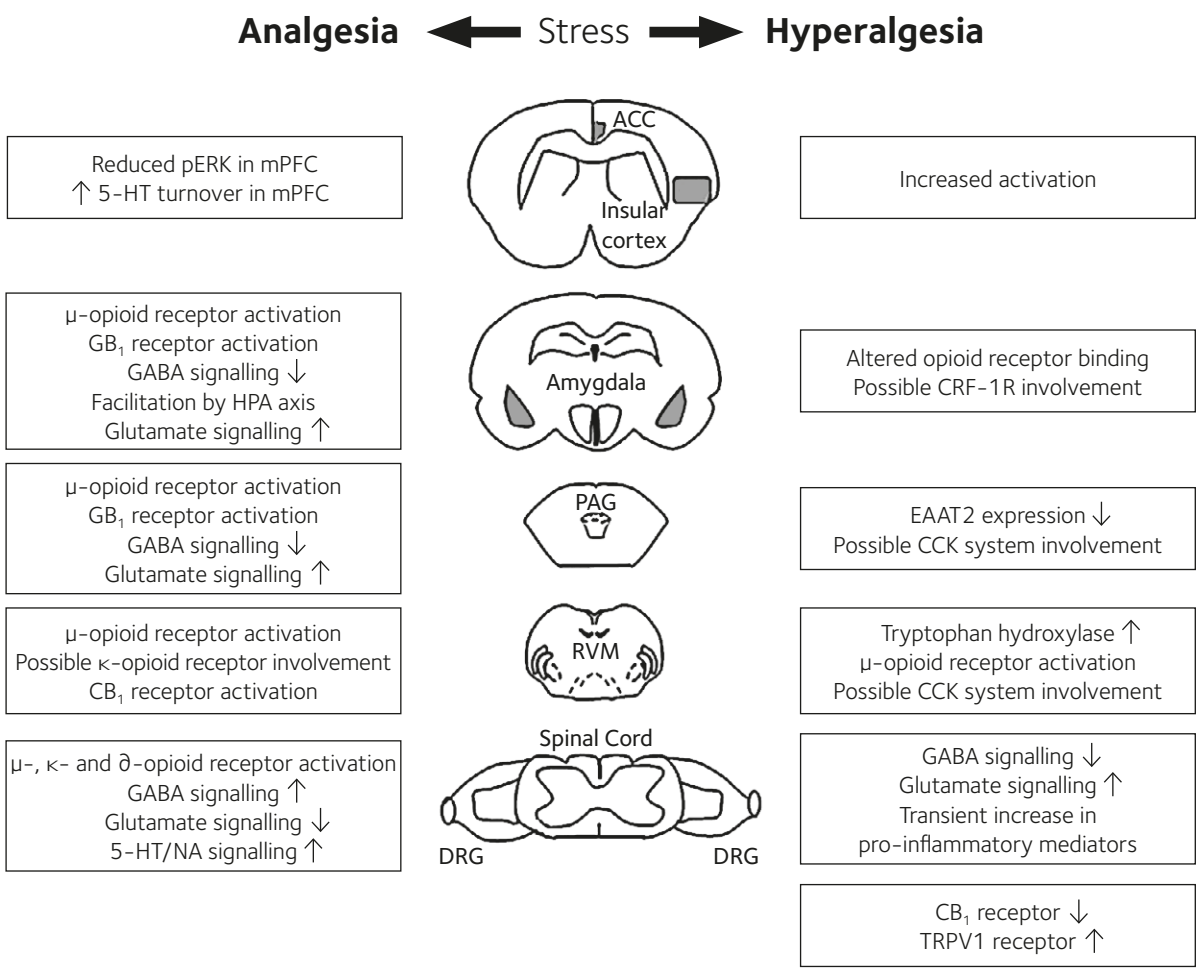

Figure 2. Summary of some of the key sites and neurobiological mechanisms thought to mediate stress-induced analgesia (SIA) and stress-induced hyperalgesia (SIH). mPFC, medial prefrontal cortex; PAG, periaqueductal grey; RVM, rostroventromedial medulla; DRG, dorsal root ganglia; pERK, phosphorylated extracellular signal regulated kinase; 5-HT, 5-hydroxytryptamine; NA, noradrenaline; GABA, gamma-aminobutyric acid; CRF-R1, corticotropin releasing factor receptor subtype 1; EAAT2, excitatory amino acid transporter 2; CCK, cholecystokinin; TRPV1, transient receptor potential vanilloid 1. Reproduced by Permission of The Royal Society of Chemistry from Olango & Finn (2013).

However, other neurotransmitter and neuropeptide systems also play a key role (e.g. GABA, glutamate, monoamines, cholecystokinin, the HPA axis, oxytocin). Indeed there are some forms of non-opioid-mediated stress-induced analgesia. Over the past 13 years or so, our group at the National University of Ireland (NUI) Galway, and other groups, have demonstrated a key role for the endogenous cannabinoid (endocannabinoid) system in mediating both conditioned (Butler *et al.*, 2008; Finn *et al.*, 2004; Olango *et al.*, 2012) and unconditioned (Hohmann *et al.*, 2005) stress-induced analgesia. In brief, stress or fear induce the on-demand synthesis and release of endogenous cannabinoid ligands (endocannabinoids) which then activate CB1 receptors in key sites throughout the descending inhibitory pain pathway, resulting in analgesia.

Stress-induced hyperalgesia

By contrast, exposure to prolonged/chronic stress or future-oriented anxiety or depression generally leads to hyperalgesia i.e. an exacerbation of pain, referred to as stress-induced hyperalgesia (Rhudy & Meagher, 2000). Stress-induced hyperalgesia is of clinical relevance, given that a high percentage of chronic pain patients have some form of stress-related psychiatric disorder. For example, patients with pain disorders are more likely to develop anxiety compared to the healthy population. Depression and chronic pain have been estimated to co-occur in up to 80% of patients, and the combination is more disabling and costlier to both patients and society than either disorder alone. In addition, experimental studies with healthy volunteers have shown that exposure to different stressors can alter how a person perceives pain (Rhudy & Meagher, 2000) and neuroimaging studies have shed light on the neural substrates involved in exacerbation of pain by stress or anxiety. Like stress-induced analgesia, animal models have played a key role in advancing our understanding of the receptor and neurochemical mechanisms that mediate stress-induced hyperalgesia (Corcoran *et al.*, 2015; Jennings *et al.*, 2014). It is clear that maladaptive changes in multiple receptors and neurotransmitter systems throughout stress- and pain-related circuitry are involved, including the opioid, GABA, glutamate, monoamine, cholecystokinin, and CRF signalling systems. More recently, we and others have also demonstrated that altered endocannabinoid system activity also underlies exacerbation of pain by stress or negative affect (Corcoran *et al.*, 2015; Rea *et al.*, 2014).

In summary, it is now very apparent that the neural basis for stress-induced modulation of pain lies in the fact that there is very considerable overlap in the neural substrates and circuitries of stress and pain. Exacerbation of pain by stress, and co-morbidity of pain with stress-related psychiatric disorders including anxiety and depression represent very significant clinical challenges. Our ever-increasing understanding of overlap and interactions in the sites and mechanisms that regulate pain and stress means that it may be possible to develop new therapies which can treat both pain and co-occurring anxiety/depression. Indeed, the current use of drugs such as pregabalin and amitriptyline for the treatment of both pain and anxiety/depression illustrates the close associations that exist between pain and psychiatric disorders and suggests that novel drugs with improved efficacy and fewer adverse effects may eventually emerge from research focused on understanding stress–pain interactions.

‘It may be possible to develop new therapies which can treat both pain and co-occurring anxiety or depression’

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Stress and the gut – it's not all in your mind

How interactions between the brain, the gastrointestinal system, and the microbial residents of the gut influence both gastrointestinal and cognitive function

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We are all intuitively aware that stress has an impact on the function of our digestive system. Whether it is the transient butterflies that accompany an acute stressor such as an exam or an interview, or the more toxic consequences of chronic stress for gut function, our cognitive experience of our world and its challenges can profoundly alter our physiological functions of digestion, absorption, and excretion. What has been less fully appreciated until recently, however, is that communication between the brain and gut is bi-directional, and gastrointestinal illnesses may be accompanied by neuropsychiatric disorders, such anxiety, depression, and memory deficits. The central nervous system and the gastrointestinal system are in constant communication, in part via the enteric nervous system or 'little brain' of the gut. We are also rapidly learning of the ways in which the microbes that reside in our gut may be important mediators of the cross-talk between gut and brain. They are, in turn, influenced by environmental conditions, such as stress and diet, in ways that modulate their impact on both digestive and cognitive function.

As humans, we like to think that we are masters of our own universe. But in fact, it has now become abundantly apparent that we, along with all other beings, are actually superorganisms. Thus, we consist not only of our own cells and genome, but also of distinctive populations of symbiotic microbes, along with their associated genetic material and repertoire of metabolic capabilities. This so-called 'microbiota' is comprised prominently of bacteria, which have been the best-studied populations, but also of fungi, bacteriophages and other viruses, and archaea, which are only now beginning to be examined. Specialised microbiota inhabit a variety of body niches, such as the intestines, oral cavity, skin, and respiratory and genital tracts, although the most extensively characterised of these is the gut-associated bacterial microbiota, consisting of thousands of unique species in a typical healthy human adult. The gut microbiota resides throughout the length of the intestine, but is most heavily concentrated in the colon. Estimates of the number of gut bacteria suggest that there are as many as 10^{14} throughout the gut, or 10 times the number of human cells in the entire body (Sekirov *et al.*, 2010). Recently, the 10:1 ratio has been disputed, with a claim that the numbers of human cells and gut bacteria are of the same order of magnitude (Sender *et al.*, 2016). But even if this is true, the microbes collectively encode significantly more genes than the cells of their host.

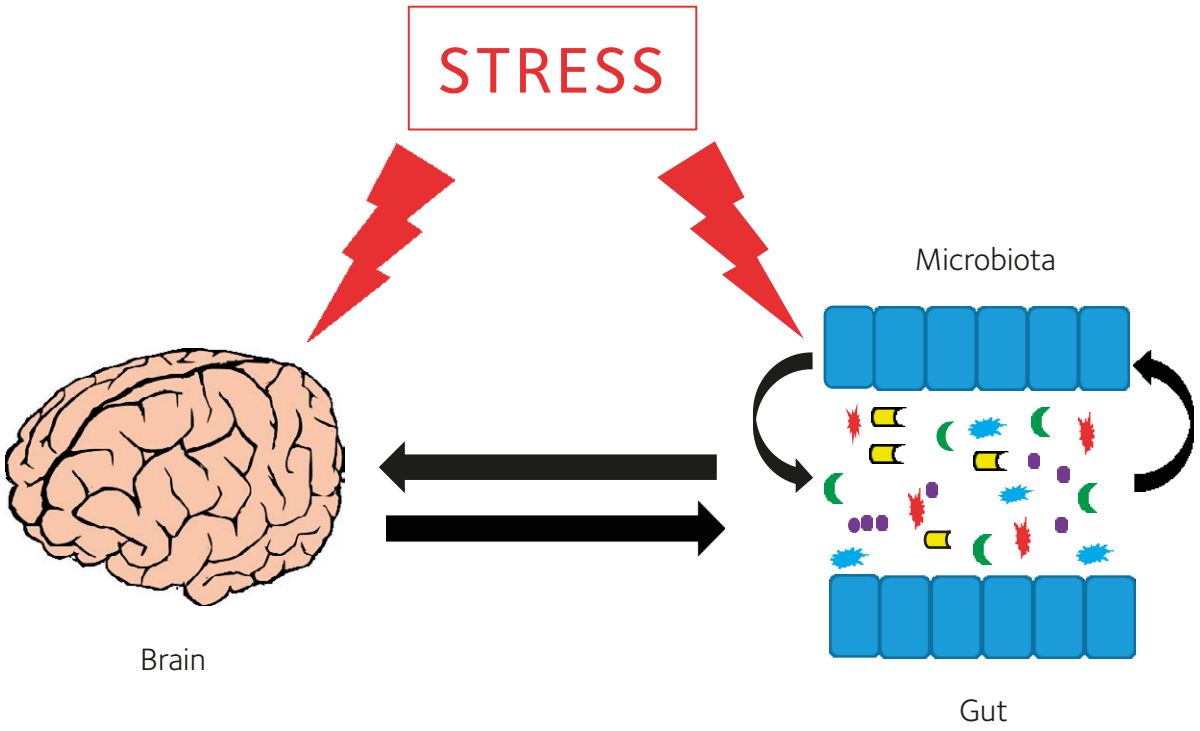

Figure 1. Model of interactions in the microbiota-gut-brain axis and the impact of stress. The gut microbiota is in constant bidirectional communication (curved black arrows) with the epithelial cells (blue boxes) that line the gut, as well as underlying cell types (not shown), via both physical interactions and the production of soluble metabolites that act as messengers. Similarly, factors from the host, including molecules displayed on the surface of the epithelium, can shape the composition of the microbiota. In turn, both bacterial messengers and those that they trigger host cells to produce can travel to the brain to influence its function (left-facing horizontal arrow). Similarly, chemical transmitters from the brain, or released from its efferent neurons, can influence gut function and thereby also regulate the microbiota (right-facing horizontal arrow). When the system is subjected to external stress, this may amplify signalling relationships by, for example, increasing the release of neurotransmitters that act on the gut, or increasing intestinal permeability, elevating levels of circulating bacterially produced metabolites that can act on the brain. The end result is a dysbiosis of the microbiota and associated dysfunction of both the gut and brain.

Our symbiotic microbes – what do they do for us?

As illustrated by the ability to raise experimental animals in a germ-free state, the microbiota, in the gut or elsewhere, is not essential for life. Nevertheless, the gut microbiota in particular offers several advantages to the host. It embodies a large capacity for metabolic conversion of ingested nutrients that cannot otherwise be assimilated by the host, such as dietary fibre, as well as the metabolism of xenobiotics and vitamin precursors. Similarly, the microbiota is said to 'educate' the mucosal immune system, either directly or by producing immunoregulatory metabolites, ensuring (at least in health) that the gut is largely tolerant of its resident commensal organisms as well as innocuous dietary proteins, while remaining on-guard to protect against pathogens. The gut microbiota also contributes to the homeostasis of the epithelial lining as well as its barrier function, and appears to support the development of the microvasculature, acquisition of appropriate motility responses, and maturation of the enteric nervous system (Obata & Pachnis, 2016). The organisms of the microbiota also defend against colonisation with pathogens, either by direct physical exclusion, by producing bacteriostatic or bacteriocidal products, or by stimulating the host to secrete antimicrobial peptides. Indeed, a course of broad-spectrum antibiotics given for an extraintestinal disease, such as a chest infection, can render patients susceptible to the consequences of intestinal infections and/or overgrowth of injurious bacteria, such as *Clostridium difficile* (C. diff).

It is commonly held that the gut microbiota begins to establish itself immediately after birth, notwithstanding some controversial data that suggest the presence of microbes in the fetus *in utero*. Indeed, the ability to derive germ-free animals via Caesarean section (C-section) implies that if microbial DNA is in fact present in the womb, it likely does not reflect the presence of viable organisms (Perez-Munoz *et al.*, 2017).

What is well-established is that the initial gut microbiota shares many characteristics of the mother's vaginal microbiota for babies delivered vaginally, and differs substantially for those delivered by C-section. An intriguing recent study partially restored a 'normal' microbiota in the gut, oral cavity, and skin by exposing babies delivered by C-section to the mother's vaginal fluids, with the authors speculating that this might reverse the known association between C-section deliveries and an increased risk for immune and metabolic disorders (Dominguez-Bello *et al.*, 2016). After birth, the baby's microbiota is relatively simple and variable for the first year or two of life, but gradually takes on the characteristics of a mature, adult-like microbiota. In that this is also a critical period for maturation of the immune system, it is perhaps not surprising that disruptions in the normal process for parallel maturation of the microbiota are felt to predispose to autoimmune and allergic diseases. For example, the increasing tendency to protect infants from microbial exposure may set

them up for an increased later risk of asthma, metabolic disease, or obesity (the 'hygiene hypothesis'), as does excessive/indiscriminate early-life use of antibiotics (Schulfer & Blaser, 2015).

The microbiota as a mediator of responses to stress

But how does all of this relate to stress,The Society's theme for 2017 and the impetus for the invitation to write this article? The gut microbiota perhaps predictably shows alterations in the setting of a variety of intestinal disease states, such as inflammatory bowel diseases and irritable bowel syndrome, and indeed, some of the characteristics of these diseases can be transferred to naïve, previously germ-free animals with the microbiota from affected mice or humans. But it is also becoming increasingly clear that gut microbes and their products may have effects well beyond the confines of the intestine itself. Perhaps the most intriguing aspects of this area of research relate to observations that tie the composition of the gut microbiota to cognitive function and/or the cognitive response to stress. Further, the adverse effects of stressful stimuli on gut function depend on the presence of intestinal microbes. The experimental findings reported to date support the model of a bi-directional microbiota-gut-brain axis (Fig. 1) that influences the normal function of both bowel and brain alike, and may explain, for example, the comorbidity of specific digestive and psychiatric disorders (Gareau, 2016). And while the evidence in human patients is largely correlative at present, it is intriguing to observe that derangements in the microbiota have been associated with numerous neuropsychiatric conditions, including depression, autism, schizophrenia, and perhaps even Parkinson's disease (Dinan & Cryan, 2017).

Therapeutic manipulation of the microbiota – from probiotics to transplants

So if both intestinal and neuropsychiatric conditions are potentially attributable to alterations in the gut microbiota (at least in part), and if such alterations also mediate the impact of stress on the relevant organ systems, can we mitigate these outcomes by targeting the microbiota? Several approaches have been posited to have either beneficial or deleterious effects on the make-up of the gut microbiota and its intestinal and extraintestinal influences. Perhaps the most obvious of these is the diet. While the gut microbiota was at one time felt to be relatively immutable in adulthood, improved analytic approaches indicate that its make-up in fact is profoundly influenced by the composition of the diet and even by the timing of meals. For example, Western diets, high in meat and fat, decrease the diversity of the microbiota (and may even promote the emergence of pathogenic properties in commensals) whereas diets rich in plant-based fibre increase it. Another approach to targeting the microbiota is the use of antibiotics, although for the reasons discussed above these are likely to be deleterious in the main, particularly early in life. The composition of the microbiota can also be altered directly by the administration of probiotics, which are commensal micro-organisms selected for their apparent health benefits that can be taken orally. Studies in animal models demonstrate that probiotics can improve both gut and cognitive function in animals exposed to a variety of stressors, or can negate the cognitive dysfunction accompanying intestinal inflammation or infection. However, not all probiotics are created equal, and much work remains to be done both to validate animal studies in human clinical trials and to define characteristics of probiotic strains that predict efficacy in a given clinical setting.

Perhaps the approach to targeting the microbiota that has attracted the most recent public attention is the practice known as faecal microbial transplant (FMT), where faecal material is transferred from a healthy donor (often a relative) to someone suffering from a specific intestinal or extraintestinal disease. Enthusiasm for FMT derived initially from its dramatic efficacy in some patients suffering from disabling and persistent diarrheal disease as well as other symptoms associated with treatment-resistant C. diff infections. More recently, there have been encouraging data suggesting that FMT may be effective in producing remission in inflammatory bowel disease, although the long-term consequences are unknown and larger, well-controlled studies are needed. Exploratory reports even suggest beneficial effects of FMT on gastrointestinal and behavioural symptoms of autism, or in obesity and metabolic syndrome, but much further work is needed to validate these preliminary data. Ideally, FMT procedures should be conducted under carefully–controlled and physician-supervised conditions to screen for the potential presence of pathogens or toxins. Nevertheless, despite the obvious 'yuck' factor, 'do-it-yourself' instructions can readily be found online (there are even Facebook groups), and some individuals are sufficiently distressed by their condition to give it a try.

Closing thoughts – mitigating negative effects of stress

In conclusion, therefore, it is clear that our response to stress, whether manifested in our thought patterns or in our gut, is dramatically shaped by the microbiota that resides in the intestines. Particularly in humans, studies conducted to date have largely been confined to cataloguing the key players in a given setting, but animal data are provocative, and functional studies in humans will doubtless follow. No matter what, in the coming years, the explosive growth of studies aiming to target the microbiota for health benefits should give us a much better understanding of which approach, if any, is likely to be most beneficial for a given condition and even a given individual, since host factors clearly can also impact our microbial composition. This work holds the promise of ameliorating negative effects of stress, and perhaps may offer new avenues for the therapy or even prevention of the myriad of stress-related disease states that are increasing in incidence in developed countries.

The choice of citations was intended chiefly to be illustrative of selected recent contributions to the field. The author apologises to the many colleagues whose seminal works could not specifically be cited in this informal article, due to space limitations.

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The stress of exercise

How it tries to break us but ultimately shapes us

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Muscular exercise is perhaps the most common form of stress that humans experience. Exercise physiology, in turn, is the study of how the body responds to this form of stress. Exercise physiologists use exercise stress experimentally to study physiological control mechanisms. Exercise tests are also used to screen people for cardiovascular, respiratory, and muscular diseases, because a reduced ability to cope with exercise stress is a hallmark of such disorders. Specific disease states frequently produce unique physiological responses to muscular activity, aiding clinical diagnosis. At the other end of the spectrum, regular exercise testing is a common component of athletic training prescription and performance prediction. Understanding exercise stress and how to impose it is the primary mission of the exercise physiologist. Doing so allows us to assess, maintain, or enhance functional capacity in both health and athletic contexts.

Defining the stress of exercise

It would be impossible to comprehensively review all forms of 'exercise-related stress' in this article. It will instead focus on acute physiological responses to exercise and the chronic adaptations to repeated bouts of exercise (i.e. physical training). The influence of other stressors relevant to exercise (chiefly environmental stress) are mentioned only briefly.

A textbook definition of 'exercise stress' does not exist, largely because 'stress' is used as a catch-all term for anything that results in a physiological response to muscular activity. As a catch-all definition, this is not a bad start. To be a little more precise, we could define it as 'Any muscular activity that challenges homeostasis and produces a measurable physiological response'. Writing the previous sentence involved muscle activity, but it would not qualify as stress because it would not have resulted in a measurable physiological response. Rising from the chair to make a cup of tea, however, would qualify, given the contraction of the lower limbs and the changes in blood pressure that occur when standing up. This is one reason why the 'sit-to-stand' test is used in the functional assessment of older adults. To begin to understand exercise stress, we must first understand the stimulus causing it before considering the resulting physiological responses.

Muscle activity: the stimulus

Muscle activity comes in many forms, and is used for many purposes. This is one of the reasons exercise is a stressor: the neuromuscular system needs to be a jack of all trades, and few humans are phenotypic specialists. Those that are often

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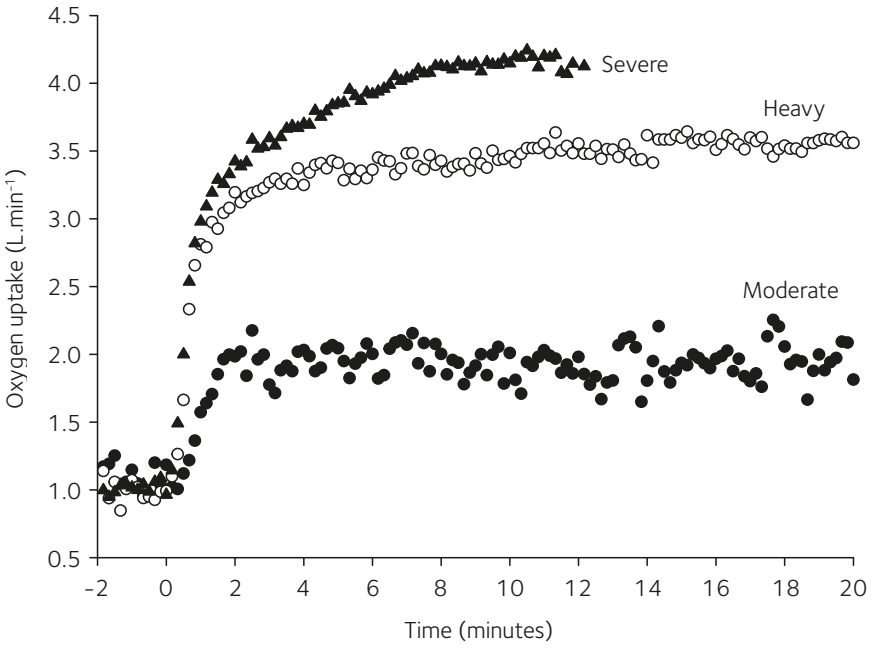

Figure 1. Oxygen uptake responses to constant-load moderate, heavy and severe cycling exercise performed on separate days. Constant-load exercise commenced at time zero, and continued for 20 minutes or until the participant failed to maintain the required pedal rate. Note the attainment of a steady state in moderate exercise, the delayed attainment of a steady state in heavy exercise and the absence of a steady state and the achievement of maximal oxygen uptake (4.20 $L.min^{-1}$) in severe exercise. See text for further details. Redrawn from Burnley and Jones (2016).

become athletes. But even the action of activating a muscle produces a myriad of different stressors which have both acute and chronic effects. Take, for example, the characterisation of muscle action as concentric, isometric, and/or eccentric. In many human activities, all three contraction types take place at various parts of a movement. Each imposes its own unique mechanical and physiological stress on the body. Isometric actions, for example, in which the muscle attempts to shorten, but does not succeed beyond the elastic nature of the muscle–tendon unit, can occlude both venous and (if sufficiently forceful) arterial blood flow, both of which will serve to amplify the metabolic and cardiovascular stress of exercise. Eccentric actions, in which the muscle is lengthened under load, can occur at considerably higher forces than those obtained isometrically, and in such cases the muscle is damaged, leading to an acute loss of maximal force-generating capacity and an inflammatory response over subsequent hours and days. This results in the soreness known colloquially as ‘DOMS’ – delayed-onset muscle soreness.

Concentric actions, in which the muscle truly contracts, are characteristic of heavy resistance exercise using free weights and other machinery. Such exercise can produce rapid fatigue but also influences respiratory and cardiovascular control as the abdominal muscles are forced to contract to brace the thorax. Holding one’s breath in conjunction with bracing the abdomen leads to the classic Valsalva manoeuvre, in which venous return and thus cardiac output are substantially reduced, resulting in light-headedness.

Locomotory activity, in which a substantial fraction of the skeletal muscle is activated (such as running, cycling, rowing, and swimming), involves low-force dynamic muscle actions which can, at various points, be concentric, eccentric, or isometric. Such exercises place a potent stress on the respiratory and cardiovascular systems, because even in untrained individuals the muscle mitochondrial capacity for oxygen utilisation far outstrips the cardiovascular system’s ability to transport oxygen from the lungs. Similarly, the muscle’s ability to produce force far outstrips the mitochondria’s ability to provide a sustainable energy supply for continued force production. These two factors underlie the direct stress which exercise produces, and both form the basis of exercise training for endurance and strength.

Absolute and relative exercise stress

Although exercise intensity is the most important component of exercise stress, a complete definition would require us to consider the duration of exercise and thus the exercise 'volume'. This is important because even mild exercise can result in significant levels of stress if continued for long enough, as anybody who has hiked anywhere can attest. A distinction also needs to be made between the absolute and relative stress that exercise produces. This is easiest to conceptualise with respect to exercise intensity. The absolute intensity can be expressed simply as the amount of force, speed, or power output the exercise requires. Intensity can also be expressed relative to an individual's physiological capabilities: as a percentage of maximal heart rate, maximal oxygen uptake, maximal power, or, in the case of maximal contractile force, as a percentage of the maximal voluntary contraction. It is then straightforward to compare individuals' capabilities in terms of the percentage requirement of an absolute intensity. For example, in cycling, a power output of 250 W might require 80% of maximal oxygen uptake in one individual but only 60% in another. Relative intensity is usually the more meaningful physiological measure of exercise stress, as detailed in 'Acute responses to exercise stress' below.

There are situations in which the absolute stress a given task places on an individual is important. Attempts to break a world record in any athletic event require the athlete to achieve a given speed for a known duration. The athlete's physiological capabilities can then be determined under laboratory conditions to determine if an attempt is feasible. In the uniformed services (police, fire service, and the various branches of the military), there are accepted minimum fitness standards required of all recruits. These standards become increasingly rigorous as the demands of the service increase. In the extreme, the demands placed on special forces soldiers are such that recruits are sometimes placed at significant physical risk. It should be noted, however, that death during vigorous exercise is extremely rare, amounting to one death per ~1.5 million episodes (Albert *et al.*, 2000). However, exercising in the heat or at altitude considerably increases the cardiovascular and/or thermal strain, and thus the acute risk, associated with exercise.

Acute responses to exercise stress

Appreciating the stress of exercise begins with an appreciation of the manner in which energy is transferred within skeletal muscle. Very small quantities of ATP are stored in the muscle. So small, in fact, that a maximal contraction lasting less than 2 seconds could exhaust the muscle's supply if no other energy sources were available to buffer its loss. One of the triumphs of 20th century biochemistry and physiology was to identify both the metabolic pathways supporting ATP homeostasis and, crucially, how they work *in vivo*. The fall in ATP that occurs during exercise is buffered by substrate-level phosphorylation (phosphorylcreatine breakdown and glycolysis leading to lactate production) and oxidative phosphorylation. These processes are interlinked via the 'phosphorylcreatine shuttle', in which the fall in phosphorylcreatine and the resultant alterations in phosphorylation potential drives mitochondrial respiration. This control mechanism can now be studied non-invasively using magnetic resonance spectroscopy and breath-by-breath measurements of oxygen uptake. Both measurements can be used to infer the stress produced by a given bout of exercise.

A muscle or muscle group that is coping with exercise-induced stress is one that can achieve a steady-state metabolic and respiratory profile (Fig. 1), in which there is no systematic change in muscle metabolites or muscle oxygen utilisation. How large the adjustment is once a steady state is reached (in this context, the rise in oxygen uptake or fall in phosphorylcreatine) gives information about the absolute magnitude of the stressor. However, it is only non-steady-state behaviour that provides information about the relative exercise stress. During 'moderate' constant-load exercise (performed below the so-called lactate threshold), a steady state is reached within 2-3 minutes, the metabolic disturbance is small, and exercise can be maintained for several hours if the participant is motivated to continue. The stress experienced in this situation is a function of the volume of exercise performed, rather than the intensity, with central fatigue (in cycling) and muscle damage (in running) being the primary outcomes of stress.

In 'heavy' exercise (exercise performed above the lactate threshold but below the maximal steady state or critical power), a steady state is delayed by 10-20 minutes, but metabolic parameters will eventually stabilise at oxygen uptake values higher than initially anticipated. In the heavy domain, volume and intensity combine to impose the stress. Together, these serve to draw down muscle fuel stores (chiefly glycogen) and provide a thermal load which may exceed the capacity to dissipate heat in hot and humid environments. The heavy domain has, therefore, been the basis of many classic studies on carbohydrate and fluid intake in the exercising human (Burnley & Jones, 2016).

Above the maximal steady state or critical power (so-called severe-intensity exercise), none of the metabolic parameters associated with exercise can stabilise: oxygen uptake, phosphorylcreatine, inorganic phosphate, and pH all change progressively until exercise is terminated at task failure. In exercise engaging a large muscle mass, task failure follows the attainment of maximal oxygen uptake. The stress of severe-intensity exercise is derived primarily from its intensity, since task failure usually occurs in considerably less than 30 minutes. Severe-intensity exercise is, therefore, representative of the stress placed on humans exercising during endurance activities of ~2-30 minutes duration.

Overall, the above scheme is relevant to stress placed on the cardiorespiratory system and muscular systems during many endurance activities, but similar intensity/duration descriptors have been developed for exercise used to express and develop strength and maximal speed. These are usually expressed as some percentage of maximal static or dynamic muscle function (e.g. the '1 repetition maximum'). Both forms of exercise intensity quantification are used to structure exercise training programmes in order to provide sufficient stress to elicit the desired effect whilst avoiding injury (see below).

Exercise stress testing

By progressively increasing exercise intensity (treadmill speed or cycling power output, for example), it is possible to evaluate the body's response to stress across the aerobic intensity spectrum described above. This is the basis of the classic incremental exercise test, variants of which are used throughout the world in cardiac or respiratory stress testing and the physiological evaluation of athletes. Of course, it is impossible to perform exercise that only stresses the heart or lungs, and so terms like 'cardiac stress testing' are something of a misnomer. Nevertheless, incremental exercise testing with electrocardiography, echocardiography, and pulmonary gas exchange measurements can reveal an array of cardiopulmonary problems, since few interventions provide the potent yet controlled stress that exercise does. That stress produces some unique signatures of cardiorespiratory dysfunction. For example, right-to-left atrial shunt caused by a patent foramen ovale in patients with pulmonary vascular disease is easily observed during incremental exercise (Wasserman *et al.*, 1999), and indeed shunting may only occur during exercise. The same testing can quantify the effects of treatment, allowing the clinician to decide whether or not lung transplantation is required.

Stress testing in athletes is required for setting training intensity zones and, where relevant, for performance prediction. In these cases, the stress placed on the body must be as specific as possible in order to yield relevant performance information.

‘The acute physiological response to exercise stress is dominated by muscular, cardiovascular, and respiratory feedforward and feedback mechanisms, all of which attempt to limit the disruption to homeostasis produced by muscular contractions’

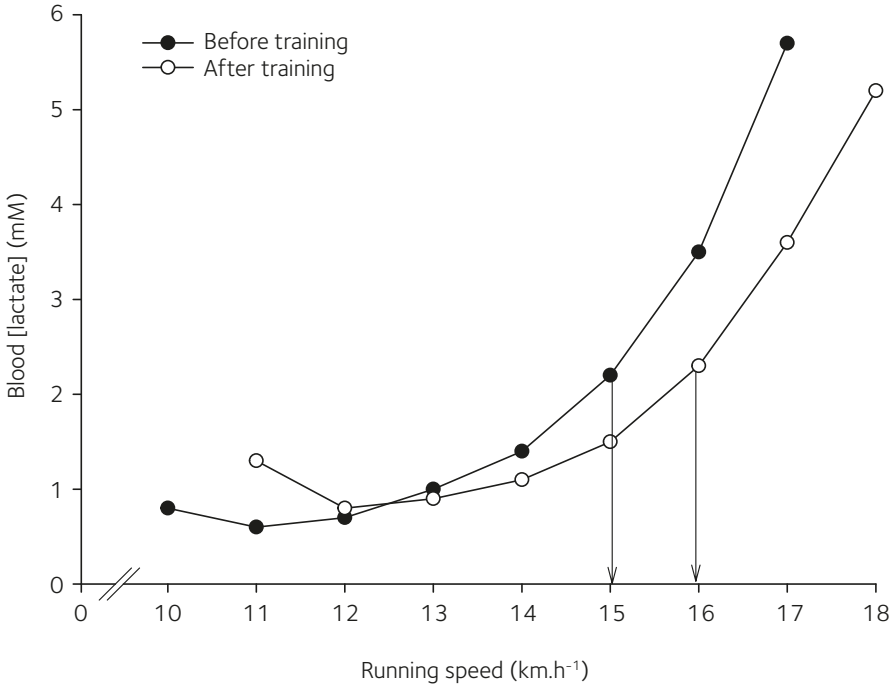

Figure 2. Blood lactate responses to incremental treadmill exercise before and after a 5 week period of endurance training. The plot displays a classic right-shift in the lactate-speed curve, indicative of a reduction in exercise-related stress at any given running speed. The downward arrows represent the speed associated with two half-marathon races, one performed in the week before (arrow at 15 km/h) and the other in the week after the training period (arrow at 16 km/h). Notice that the blood lactate concentration associated with these race speeds, and thus the relative stress of exercise experienced, is identical.

As a result, ergometers are not just mode-specific but are usually bespoke in the sense that they utilise as much of the athlete’s personal equipment as possible. The advent of highly accurate force and power measuring devices has all but eliminated the need for regular laboratory assessments of fitness in elite cycling, and the development of wearable sensors in other sports is likely to achieve the same end. Even though the modern-day sports physiologist’s laboratory is becoming increasingly portable, measurements in the field seldom match the precision and control of the laboratory environment. Translation of findings from the field to the laboratory and vice versa will remain a major area of the sports physiologist’s work for the foreseeable future.

Chronic responses to exercise stress: training

The well-documented effects of physical training are a manifestation of the chronic adaptation to exercise stress. Selye (1936) considered muscular exercise to be a form of his ‘general adaptation syndrome’, although in later writings he made a distinction between muscular adaptations to exercise and the non-specific stress reaction he was describing. A key feature of exercise stress is that adaptations to it are positive in all but the most extreme cases (overreaching or overtraining). In other words, application of training stress that does not cause injury will maintain or improve physiological function, in contrast to the so-called ‘exhaustion phase’ of the general adaptation syndrome.

Although the positive effect of training on performance capability and physiological parameters such as the maximal oxygen uptake have been known for decades, it is only relatively recently that changes in the morphological components that determine such parameters, and the molecular bases of such changes, have been characterised. Even so, only the molecular basis of ‘strength’ or ‘resistance’ training and ‘endurance’ training have so far been convincingly differentiated. Even in these cases our understanding of the processes leading to training adaptations is far from complete. To observe the clearest distinction between strength and endurance training usually requires a sample of trained participants. This is because even relatively low-intensity exercise in untrained individuals

results in both mitochondrial and myofibrillar protein synthesis (see Coffey & Hawley [2017] for review). Additionally, for logistical reasons physical training studies rarely last more than 8-12 weeks, which severely limits our ability to provide evidence-based advice to athletes who have been training for many years.

The development of an 'endurance' phenotype, as one might expect, seems to require a high energetic demand from oxidative processes, but does not require the production of high forces. As detailed above, such high-intensity aerobic exercise produces a considerable metabolic stress. This results in AMP-activated protein kinase phosphorylation and subsequent transcription of mitochondrial proteins, leading to increased mitochondrial volume and aerobic enzyme activity. At the same time, angiogenesis (initiated, in part, by the expression of vascular endothelial growth factor [VEGF]) results in a greater capillary density. There is evidence that almost every step in the O_2 conductance pathway beyond the alveoli can be enhanced by endurance training. These adaptations lead to a profound reduction in the stress experienced at a given absolute intensity. As a result, for a given level of effort, exercise performance improves (Fig.2).

Subjecting the muscle to very high forces in resistance training results in a completely different stressor and thus a completely different response to endurance exercise. Studies showing gains in maximal strength without measurable muscle hypertrophy lead to the inference that the strength gain has a neural origin. The behaviour of the motor unit pool in response to training is, however, extremely challenging to measure. It is only very recently, for example, that individual motor unit behaviour has been tracked longitudinally (Martinez-Valdes *et al.*, 2017), and these data suggest that different forms of training exert differential effects on motor unit firing rate. In the fed state, muscle hypertrophy undoubtedly occurs in response to resistance training, and a number of factors that respond to mechanical stress are likely involved in signalling cascades leading to the deposition of new contractile proteins. The 'downstream' events resulting in protein synthesis have been thoroughly investigated in the last 10-15 years. Briefly, hypertrophy appears to be the result of the upregulation of protein synthesis primarily by the mechanistic target of the rapamycin (mTOR) pathway. In addition, the effects of exercise on connective tissue and bone remodelling to support high-intensity muscular contraction should not be forgotten. These responses may play an important role in healthy ageing by the avoidance of frailty in later life. Indeed, it is because exercise affects so many organ systems that its role in primary preventative healthcare is currently of interest.

Detraining: use it or lose it

The final twist in the story of exercise-induced stress concerns the effects of reducing or removing that stress. Many of the training-induced adaptations mentioned above begin to recede when training is ceased. If exercise intensity is maintained, however, so are the training-induced gains, a fact that athletes use in preparation for competition (the so-called 'taper' phase). Complete cessation of exercise leads to a process of detraining, in which muscle size, and mitochondrial and capillary densities decrease. These changes occur within days or weeks of stopping training. Central cardiovascular adaptations, such as left ventricular volume and blood volume revert to their pre-training values over a period of months. These detraining-induced losses can explained, in part, by the fact that these adaptations (such as the gain in lean tissue mass) are energetically expensive to maintain. In this context, detraining would appear to be a cost-saving process. As noted above, however, small amounts of relatively intense exercise appear capable of maintaining the gains accrued by previous training.

Conclusions

The acute physiological response to exercise stress is dominated by muscular, cardiovascular, and respiratory feedforward and feedback mechanisms, all of which attempt to limit the disruption to homeostasis produced by muscular contractions. The disruption to homeostasis results in fatigue, and produces the metabolic and/or mechanical signals that initiate the synthesis of new proteins. Whether these new proteins build new mitochondria or new contractile elements depends upon the nature of the exercise stress. As a result, exercise stress tries to break us, but its repeated application ultimately shapes us.

'Understanding exercise stress and how to impose it is the primary mission of the exercise physiologist'

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Oxidative stress is harmful, and the TRPM2 channel bears part of the responsibility

Emerging evidence shows that oxidative stress can bring about harmful consequences via activating the TRPM2 ion channel.

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It is well known that oxidative stress, arising from generation of excessive reactive oxygen species, weak or impaired antioxidant defence, or both, is a common factor in the pathogenesis of a variety of disease states. Recent studies reveal that the TRPM2 (transient receptor potential cation channel melastatin-related subfamily member 2) ion channel bears part of the responsibility for the harmful consequences of oxidative stress such as diabetes, post-ischaemia brain damage, and neurodegenerative diseases.

Generation of ROS and oxidative stress

For all mammals like humans, life relies on continuous access to oxygen (O_2) because it is important for the efficient production of ATP, the cellular energy supplier. Molecular oxygen contains two unpaired electrons in its outer electron shell; this physiochemical property makes it readily available to generate a group of highly reactive chemicals called reactive oxygen species (ROS), such as the superoxide anion (O_2^-), hydrogen peroxide (H_2O_2), and the hydroxyl radical ($^{\cdot}OH$). A biochemist can easily describe to us how O_2^- is generated from O_2 by the electron transport chains in the process of producing ATP in mitochondria. We are also assured that cells are equipped with various enzymatic and non-enzymatic antioxidant means to eradicate ROS. For example, O_2^- can be converted to H_2O_2 by superoxide dismutases. Subsequently, H_2O_2 is changed to water by catalase or, in a bit more complicated scheme, H_2O_2 to $^{\cdot}OH$ via the Fenton reaction and then to water. No harm done! This is almost true under healthy conditions, where ROS generation is well balanced with timely elimination. ROS at very low levels can be beneficial by serving as a physiological signalling molecule in maintaining normal tissue homeostasis via regulating cell proliferation, differentiation, and programmed cell death. Immune cells such as macrophage cells generate O_2^- in large quantities mainly via NADPH oxidases and use it as a weapon to kill pathogens through the process of phagocytosis. This is because ROS at high levels can modify nucleic acids, proteins, and lipids and impair their physiological functions. By the same mechanisms of action, high levels of ROS are harmful or dangerous to mammalian cells. Thus, accumulation of ROS, resulting from excessive generation, deficiency in intrinsic antioxidant defence, or both, leads to cellular oxidative stress. It has been well documented that oxidative stress is a critical factor in the pathogenesis of a variety of disease states. Emerging evidence supports the idea that the expression of the TRPM2 channel confers susceptibility to oxidative-stress-induced cell death, thereby bringing about harmful consequences such as diabetes, post-ischaemia or reperfusion brain damage, and Alzheimer's disease.

A brief introduction: TRPM2 channel and its activation by oxidative stress

Mammalian TRPM2 (transient receptor potential melastatin-related subfamily member 2) is an integral membrane protein, belonging to the large family of transient

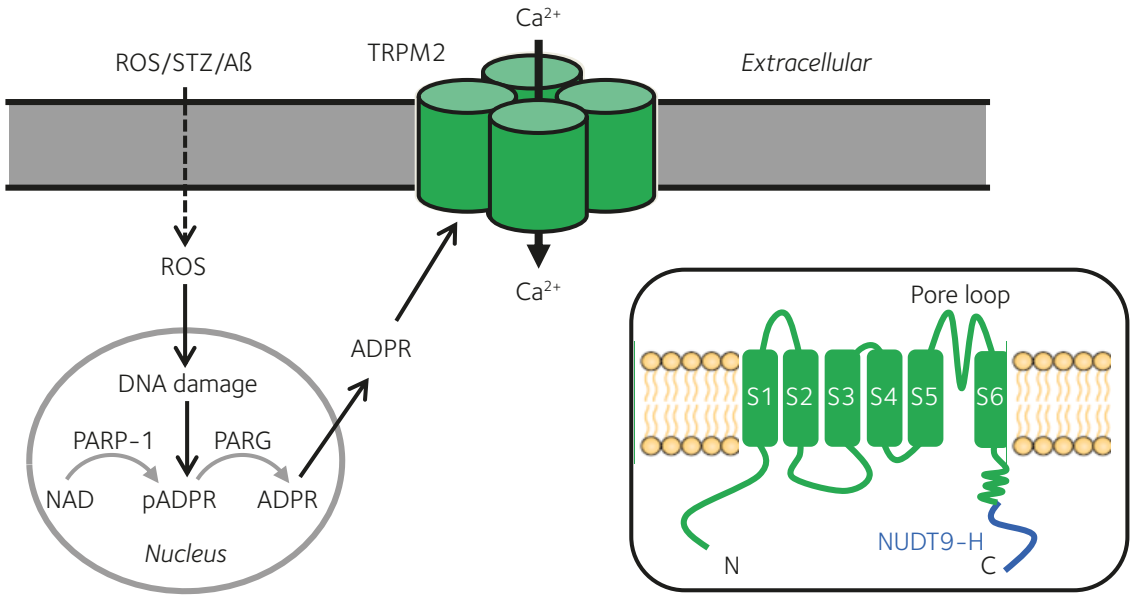

Figure 1. Schematic diagram showing TRPM2 channel activation by oxidative-stress-inducing molecules. The TRPM2 channel is a Ca^{2+}-permeable cationic channel, made of four protein subunits. As illustrated in the insert, each subunit contains cytosolic N- and C-termini and six membrane-spanning segments (S1–S6) with a pore-forming re-entrant loop (pore loop) between the fifth and sixth segments. The cytosolic C-terminal tail shows homology to NUDT-9 enzyme (NUDT9-H; in blue) and enables specific binding to and gating of the TRPM2 channel by ADPR. In TRPM2-expressing cells such as pancreatic β-cells and hippocampal pyramidal neurons, exposure to ROS or diabetogenic streptozotocin (STZ) or neurotoxic amyloid β (Aβ) peptides is known to induce oxidative stress via generation of excessive ROS. ROS damages DNA in the nucleus, and the subsequent DNA damage repair process via PARP-1 and PARG results in generation of ADPR. ADPR binds to the NUD9-H domain and opens the TRPM2 channel, leading to extracellular Ca^{2+} influx into the cell.

receptor potential proteins. As illustrated in the insert of Fig. 1, the TRPM2 protein comprises six membrane-spanning segments and intracellularly residing amino and carboxyl termini. Four TRPM2 proteins interact to form a protein complex, in which the fifth and sixth membrane-spanning segments and the re-entrant loop connecting them from each of the four proteins or subunits come together to make a central aqueous ion-conducting pore. The TRPM2 channel is permeable to Ca^{2+} and other cations (Fig. 1) (Jiang *et al.*, 2010). The C-terminal tail, highlighted in blue in the insert of Fig. 1, is similar in amino acid sequence to NUDT-9, an enzyme hydrolysing ADP-ribose (ADPR). This NUDT-9 homology domain provides a unique binding site for ADPR, and thereby allow specific gating of the TRPM2 channel by intracellular ADPR.

ADPR was long known as a by-product generated in the process of repairing ROS-induced DNA damage, engaging poly(ADPR) polymerase-1 (PARP-1) and p(ADPR) glycohydrolase (PARG) in the nucleus using nicotinamide adenine dinucleotide (NAD) as the ADPR donor (Fig. 1). ADPR is now recognised as a critical signalling molecule which selectively activates the TRPM2 channel. Excessive generation of ADPR occurs under oxidative stress. Therefore, interest has been rapidly escalating in the role that the TRPM2 channel plays in the pathogenesis of oxidative-stress-associated diseases.

TRPM2 channel mediates ROS-induced pancreatic β-cell death in diabetes

The β-cells represent the predominant type of cell in the islets of Langerhans of the pancreas. These cells are particularly important because they are responsible for making insulin and releasing it into the bloodstream in response to rising levels of glucose, and thereby help cells in the body convert sugar into energy. Loss of pancreatic β-cells leads to chronic diabetes, particularly type-1 diabetes. Type-1 diabetes arises from erroneous destruction of pancreatic β-cells by immune cells, resulting in deficient insulin production and severe hyperglycaemia, and causing life-threatening complications. Insulin deficiency due to pancreatic β-cell death also contributes to type-2 diabetes, the more common and complicated form of the disease. Pancreatic β-cells are weak in antioxidant defence and thus highly vulnerable to damage by oxidative stress. ROS-induced pancreatic β-cell death plays a critical role in the pathogenesis of diabetes.

‘The expression of the TRPM2 channel confers susceptibility to oxidative-stress-induced cell death, thereby bringing about harmful consequences such as diabetes, post-ischaemia or reperfusion brain damage, and Alzheimer’s disease’

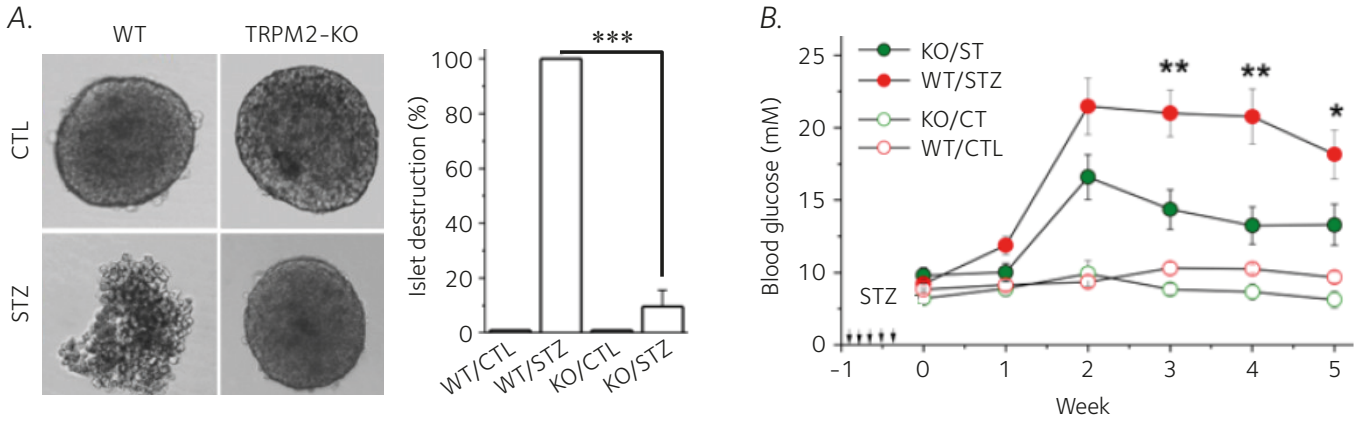

Figure 2. TRPM2 knockout mice exhibit resistance to streptozotocin-induced pancreatic islet destruction and hyperglycaemia. *A.* Left, representative images showing islets from TRPM2-KO mice, but not wild-type (WT) mice, are resistant to destruction induced by exposure to 2 mM streptozotocin (STZ) for 48 hours. Right, mean (± standard error) data from three independent experiments. ***, $p < 0.001$ comparing between STZ-treated islets from WT and TRPM2-KO mice. *B.* Mean (± standard error) blood glucose levels in TRPM2-KO and WT mice over five weeks following injection of six daily doses of STZ (40 mg/kg body weight) or vehicle (CTL), from 16 mice for STZ groups and eight mice for control groups. *, $p < 0.05$ and **, $p < 0.01$ comparing STZ-injected WT and TRPM2-KO mice. Taken and modified from Manna *et al.*, (2015).

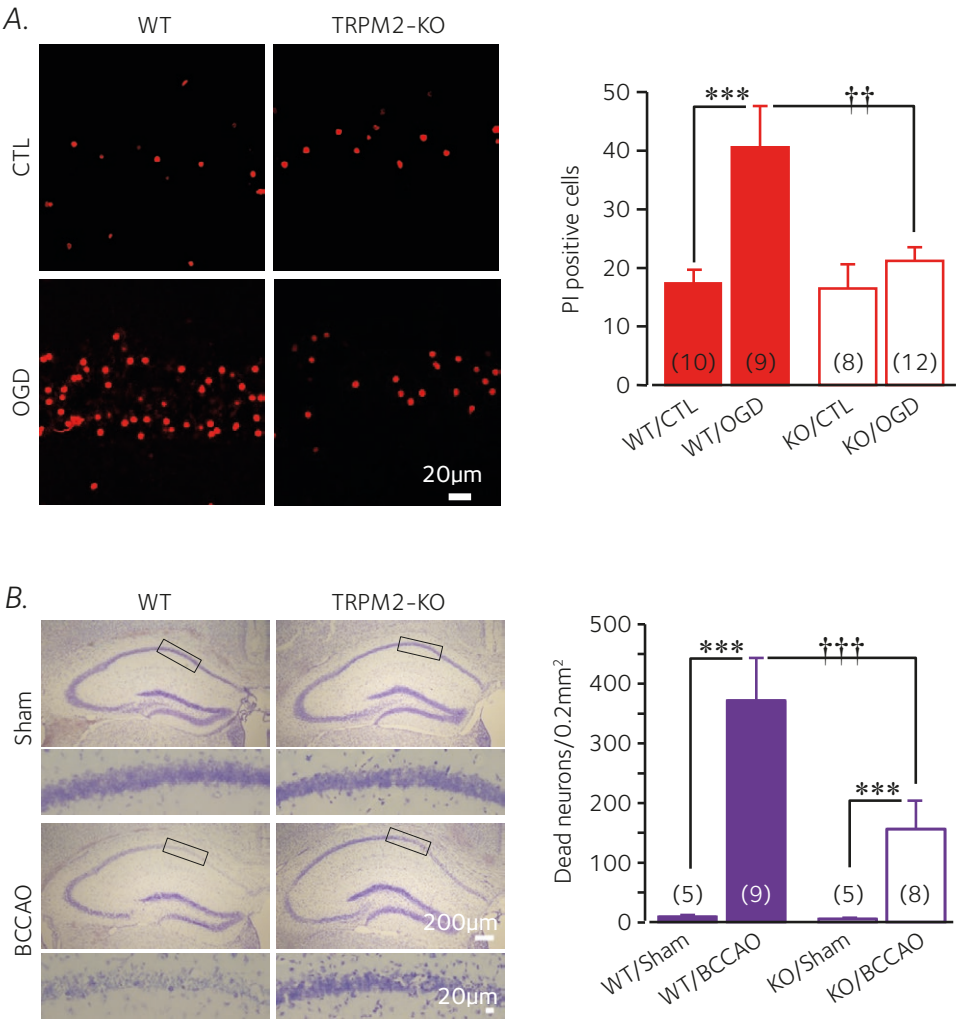

Figure 3. TRPM2 knockout protects against delayed neuronal cell death after transient ischaemia and reperfusion. (a) Left, representative images showing propidium iodide (PI, red) staining of hippocampal brain slices from WT and TRPM2-KO mice that were subjected to oxygen-glucose deprivation for 1 hour followed by reperfusion for 30 minutes (OGD-R) or control (CTL). Right, mean (± standard error) pyramidal neuronal cell death in the CA1 region, from the number of slices shown in parentheses. (b) Left, representative images showing Nissl staining of hippocampal brain slices from WT and TRPM2-KO mice after bilateral common carotid artery occlusion (BCCAO) for 15 minutes followed by reperfusion for 72 hours or sham. The rectangle boxes show pyramidal neuronal cell death in the CA1 region in enlarged images. Right, mean (± standard error) pyramidal neuronal cell death, from the number of mice shown in parentheses. ***, $p < 0.005$ comparing control and ischaemia-reperfusion; †††, $p < 0.005$ comparing between WT and TRPM2-KO mice. Taken and modified from Ye *et al.*, (2014).

Many widely used diabetogenic agents such as streptozotocin bring about diabetes in rodent animals via inducing generation of excessive ROS. It was known that pancreatic β-cells and insulin-secreting insulinoma cells express a ROS-sensitive Ca^{2+}-permeable channel and its activation is critically involved in ROS-induced cell death. This channel was later on identified as a TRPM2 channel. A recent study shows that genetic ablation of TRPM2 expression in mice provides strong protection against streptozotocin-induced destruction of pancreatic islets *in vitro* (Fig. 2a) and hyperglycaemia *in vivo* (Fig. 2b) (Manna *et al.*, 2015). These findings reveal that the TRPM2 channel plays a critical role in oxidative-stress-induced pancreatic β-cell death and the pathogenesis of diabetes.

TRPM2 channel in delayed neuronal cell death and post-ischaemia brain damage

The brain constantly demands oxygen and glucose for energy production and is predisposed to damage as a result of loss of or reduction in blood supply such as ischaemic stroke. Ischaemic stroke represents one of the leading causes of adult mortality and morbidity worldwide. Ischaemia, if it is severe or long-lasting, causes brain damage and is life-threatening. The current mainstay therapeutic strategy for ischaemic stroke is to reinstate the blood circulation. Reperfusion following transient ischaemia can, however, be harmful because reperfusion provides O_2 as the substrate for generation of excessive ROS, giving arise to oxidative stress and causing brain damage. The hippocampus is a critical brain region responsible for learning and memory. In both ischaemic stroke patients and rodent models of ischaemia-reperfusion brain damage, pyramidal neurons in the CA1 region of the hippocampus are highly susceptible to post-ischaemia brain damage. Such neuronal cell death often occurs with a substantial delay, and is often referred to as delayed neuronal cell death. Delayed neuronal cell death plays a vital role in post-ischaemia brain damage and cognitive dysfunction. Currently, there is no treatment for post-ischaemia brain damage.

The TRPM2 channel is widely expressed in the brain, including in hippocampal pyramidal neuronal cells. Exposure to H_2O_2 activates the TRPM2 channel and results in hippocampal neuronal cell death. Genetic depletion of the TRPM2 expression in mice strongly prohibited delayed CA1 pyramidal neuronal cell death *in vitro* induced by oxygen-glucose deprivation and reperfusion (Fig. 3a) and brain damage *in vivo* after transient global ischaemia and reperfusion (Fig. 3b) (Ye *et al.*, 2015). TRPM2 deficiency also effectively protected against post-ischaemia impairment in learning and memory (Ye *et al.*, 2015).

These findings provide evidence to support a significant role for the TRPM2 channel in oxidative-stress-induced delayed neuronal cell death and post-ischaemia brain damage.

TRPM2 in amyloid β-induced neurotoxicity and Alzheimer's disease pathogenesis

Alzheimer's disease (AD) is a chronic neurodegenerative disease. It is the most prevalent cause of dementia among the elderly. One of the histopathological hallmarks is the formation of senile amyloid plaques containing high levels of neurotoxic amyloid β (Aβ) peptides. AD pathogenesis is highly complex, and the underlying mechanisms still remain a topic of ongoing debate. Extensive preclinical studies using AD rodent models and clinical analysis of human AD brains provide increasing evidence to support that Aβ-induced neurotoxicity and neuroinflammation are important in AD pathogenesis and progression. It is known that Aβ accumulation leads to cellular oxidative stress. Transgenic mice producing high levels of Aβ peptides exhibited greater microglial activation, neurotoxicity, synaptic loss, and age-related impairment in memory. In a recent study, it was shown that genetic deletion of the TRPM2 expression in this AD mice prevents all the above-mentioned Aβ overproduction-induced deleterious effects (Ostapchenko *et al.*, 2015). This study reveals an important role for oxidative stress activation of the TRPM2 channel in Aβ-induced neurotoxicity and AD-related cognitive dysfunction.

In summary, there is increasing evidence from recent studies to support the idea that the TRPM2 channel plays a critical role in mediating oxidative-stress-induced pancreatic β-cell and neuronal cell death. Such TRPM2-mediated cell death contributes to the pathogenesis of diabetes, post-ischaemia brain damage, and AD. Therefore, the TRPM2 channel bears a significant part of the responsibility for the harmful consequences of oxidative stress.

'Accumulation of ROS, resulting from excessive generation, deficiency in intrinsic antioxidant defence, or both, leads to cellular oxidative stress'

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Opening up science education

Open education: helping life science educators widen access to education and foster engaging and creative approaches to learning and teaching

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This article follows one written by Keith Siew in the summer edition of the magazine on 'the open science movement', which specifically talked about open access in relation to research articles and data. My article very much focuses on teaching.

I recently presented on open education at a Physiological Society H^3 symposium entitled 'Practical Innovations in Life Science Education'. This presentation followed one at the 2016 'Achieving Teaching Excellence in the Biosciences' event at Kingston University, so I thought it timely to summarise some of the ideas for a wider readership.

What is open education?

Attempting to answer this question a few years ago would give quite a different outlook from today as the range of open educational approaches and ideas have expanded. Open education is a means of widening access to education, and the accessibility of materials, and it is not a new idea. Universities and teaching institutions have been inviting the public through their doors for centuries, and in more recent times 'open' universities have further championed the widening of access to formal education. In the 1970s, open education was a dominant philosophy and practice in primary schools with the goal of having unstructured curricula to foster creativity and support diversity in learning, alongside sharing knowledge beyond the institution. In the present reiteration of open education, there are similar underpinning ideals – seeking to provide an education system that shares, and is more inclusive and equitable. The millennium saw a number of technological advances and the use of open licenses such as Creative Commons to enable scholars to share resources, data, and other outputs within the boundaries of copyright law but to global audiences simply for the first time. Wikipedia, perhaps the single most significant open resource, started to grow organically by tapping into people's desire to co-create and collaborate. Wikipedia.org is now part of the overarching WikimediaFoundation.org where collaborative working is applied to the creation and sharing of images, resources, books, data, and an expanding project list, all using Creative Commons licensing for the reuse, editing, and redistribution of the work.

Open education – from content to practice

The relationship between open education content (also called open educational resources or OERs) and emerging open practices is a hot topic of debate (**https://www.youtube.com/watch?v=gmPmZEhy3Lc**). In this webinar of practitioners, researchers, and advocates of open education, great work within schools, colleges, and universities has clearly emerged through either the generation of openly licensed content (a good starting point), or the conception of what open practice and pedagogy might be. A widely accepted framework for practice development is David Wiley's '5 R's' which sets out options for moving towards open practice (Wiley, 2014). This in my experience is a useful concept for teachers who aspire to develop their open practice; Retain (you control what happens to the resources you share) through to Reuse, Revise, Remix, and Redistribute. Open practice extends the utility of our academic work within our institution if that is what we wish – or if we have the right policies

in place, we can share beyond the walls of our universities to subject communities and learners. In the UK, some notable examples include the University of Lincoln 'Student as Producer' project where students engaged as co-creators of open content, and the open photography course #Phonar at the University of Coventry, which invited public collaboration and led to students working with professional communities as part of their learning. Much of the UK activity stemmed from the 2009–2012 HEFCE-funded Open Educational Resource programme (**www.jisc.ac.uk/guides/open-educational-resources**). Over 85 projects spanned most subject disciplines and were seminal in building the community of open practitioners that thrives today, and were brought together in an annual conference organised by the Association of Learning Technology (#OERXX). The Oxford OpenSpires project made podcasts available as open content, and a number of projects from the University of Nottingham shared open-source software for eAssessments and learning object creation. Several projects shared the creation of life science OERs, such as that within the Nottingham Health and eLearning Media Team (HELM) plus a number of my own projects at De Montfort University sharing laboratory skills OERs (Virtual Analytical Laboratory, VAL), OERs on sickle cell disease (SCOOTER project), and midwifery and biology materials (Biology Courses project). The reach and impact of these OERs across the different project platforms, and using web marketing techniques to share content via the web, has been reported (Rolfe, 2016).

Open practice for life science practicals

My recent work has explored open pedagogies in an attempt to address challenges facing laboratory practical teaching. Practicals are defined as timetabled laboratory events, and it is well documented how teaching staff and technical teams have to cope with ever-increasing student numbers and address the gaps between school and university in terms of laboratory experience (Coward & Gray, 2014). As reported elsewhere, student criticisms include no buzz, repetitive nature, and lack of social engagement (Wilson *et al.*, 2008).

So in my experience of adopting open practices in departments, what are some of the benefits and challenges?

Open education projects at De Montfort University included laboratory skills OERs. Still accessible today via the project website and YouTube, these relatively low quality materials by today's standards, were popular with students and boosted their confidence before entering the laboratory for the first time: 'VAL has been very useful in easing my nerves before lab sessions' (Biomedical Science student, Rolfe, 2009). These OERs were then embedded within the timetable with students working through workbooks prior to entering the lab. Soon, students were creating videos of their own laboratory work and sharing these either informally with each other through social media, or as part of the project website. The laboratory technical teams also created resources in areas they thought students particularly struggled with. One of the benefits cited by staff was that they needed to spend less time repeating basic instructions as students had an overview of the fundamental skills.

In other subject areas such as immunology, OERs were used in 'flipped' classroom scenarios sharing content before practicals, which provided more effective use of time once in the laboratory. In other 'circus-style' practicals, students accessed resources by QR codes at different workstations to introduce them to different techniques, which helped to cater for large student numbers in the lab in a more effective way.

Students were engaged in not just creating content but also assessment questions later shared as OERs accompanying resources on the project websites. At the end of practicals they were encouraged to reflect on their learning by producing multiple choice questions, something adapted from the work of Bevitt and Morris (2012). Our student questions were then lightly edited and used for formative tests via the Virtual Learning Environment, and also shared as OERs.

Longer term, some changes to the learning culture were observed with students taking control and implementing their own ideas, such as photographing histology images using iPhones for sharing as OERs on the Google service Picasa and later a Facebook discussion group. Some of the lasting impacts of this work are the cross-university interest it generated – for example technology and arts students becoming interested in science projects – and the OERs being available globally to support informal and formal learning, providing new insights and perspectives for students (Rolfe, 2016).

Further information?

Today, 'open' prefixes a wide range of activities from scientific communities, scholars, data, citation, access, innovation, resources, textbooks and practice and pedagogies (see #OER, #OEP, #OTB Twitter hashtags for example). A newly started project in the UK funded by the US Hewlett Foundation is exploring the use of openly licensed textbooks to support student learning, such as those produced by OpenStax and BCCampus (follow the Twitter group for more information @ UKOpenTextbooks). For community support you can join a JiscMail list as part of the Association of Learning Technology Open Education Special Interest Group (Twitter @ ALT_C, #OpenEdSig).

Resources can be found (and shared back) via a number of platforms – Jisc App store, Life Science Teaching Resource Community (**www.lifescitrc.org**), OER commons, Wikimedia group, and many others. My analysis of discovery of content via different platforms found YouTube and Flickr the most effective way of making OERs visible, in contrast to institutional repositories which were woefully inaccessible, but did serve as a permanent store for content created. However, in many of the De Montfort projects, WordPress blogs served this purpose located on low-price hosting services (Rolfe, 2016).

This article is not an extensive literature review of all open practice in physiology or science teaching. As more evidence is gathered as to the benefits and uses of OERs and open practices, a new theoretical basis for open practical pedagogies may emerge. What is important is that we continue to openly share our case studies of teaching practice to build a fuller picture. That way, larger communities of teachers can grow and benefit:

'*It has changed my practice in terms of whenever I'm doing anything I think how could this be an OER or how could it supplement what I'm doing*'. (Microbiology lecturer).

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Meet the newest members of your Council

Charlotte Haigh

University of Leeds, UK

I am sure, as for most people, the fascination of the human body, its complexities both in health and disease, is what inspired me to become a physiologist. I started my academic career studying a BSc in Biochemistry and Physiology at the University of Sheffield; this is where I became curious about biological systems at a molecular level but always had the bigger picture of how this affected the whole body in the back of my mind. This led me to study for a PhD in Molecular Endocrinology at the University of Birmingham where my project was based around the role of transforming growth factor beta in acute and chronic diabetic nephropathy. The project allowed me to work with both basic scientists as well as clinicians from the UK, Canada, and Denmark. I really enjoyed the research and applied for a postdoctoral research position back in Sheffield working on the localisation and expression of the Na^+/H^+ exchanger in the collecting duct in the kidney, then turning my attention to the isolation and localisation of K^+ channels in the proximal tubule.

During my time as a postdoc, I had the opportunity to teach some undergraduate students and took physiology into schools. Whilst gaining the skills and experience in doing this, I could see that inspiring the younger generation was really something that I valued and would shape the direction of my career.

I moved to the University of Leeds as a teaching and scholarship academic where I have been for over 13 years now. This started with teaching, within the School of Biomedical Sciences, many areas of physiology including endocrinology, gastrointestinal physiology, physiology of reproduction and fertility, and cardiovascular and respiratory physiology at all undergraduate levels. I was admissions tutor for over six years. I continued to indulge my passion for outreach and public engagement managing to secure a Wellcome Trust Public Engagement Grant entitled 'The Physiology and Pharmacology of Sporting Success'. This led me to understand the value of engaged research in much more detail and to work with senior leaders of the University to create a Public Engagement team, of which I am now academic lead, championing the benefits of public engagement with research and facilitating opportunities and ideas for the whole University!

As a trustee, I want to be part of shaping the future of The Physiological Society as it develops and establishes the new five-year strategy. I feel that I currently contribute in many ways to the charitable objectives of The Society, especially with my work around public engagement with research. I feel my knowledge, expertise, and network of colleagues in this area could contribute widely in the implementation of the new strategy. I feel my strengths in supporting The Society and being able to contribute as a Society Trustee are in the areas of education and public engagement with research, which complement The Society's objective of supporting research to advance the understanding of physiology and disease, as well as education and public engagement to develop an informed society. I look forward to being a Trustee and getting involved with the work of The Society.

Elizabeth Sheader

University of Manchester, UK

From an early age I had always wanted to know how the human body works. I loved human biology and was fascinated by how cells work together to contribute to tissue function and how this function is impaired in the diseased state. In sixth form, I was fortunate enough to gain some work experience in a physiology research lab here in Manchester. It was then I realised that human biology was in fact physiology. This experience inspired me to explore the opportunity of studying Physiology at degree level.

I started my academic career at the University of Liverpool, where I studied a BSc (Hons) in physiology. My final year research project was such a positive experience and confirmed my desire to want to do further research. So on completion of my degree, I moved to Manchester to begin my PhD in pancreatic beta cell physiology. I continued to work as a Postdoctoral Research Fellow, where I expanded my knowledge of human physiology by working on projects in renal physiology and cardiac physiology. As a PhD student and postdoc, I was always involved in

undergraduate teaching. I really enjoyed working with the students and being on the receiving end of students wanting to know how and why the body functions as it does.

I found (and still find), students enthusiasm for learning infectious. In 2001, I obtained my PGCE for Further and Higher Education from the University of Bolton. I completed this at night school whilst continuing my research career. In 2001, I became a Teaching Focused Lecturer, and now as a Senior Lecturer, I teach physiology to over 1000 students in programmes across our Faculty of Biology, Medicine and Health, including Bioscience, Medical, Dental & Allied Health related disciplines. I use innovative methods in my teaching and aim to improve student engagement in the subject matter. I also hold additional roles in student support and leadership roles as Programme Director for the Physiology programme, academic lead for Year 1 to Medical students and OSCE co-lead for Year 1 on the MB ChB.

I am dedicated to teaching and inspiring learning. I see myself as both a scientist and an educator, who aims to integrate current physiological research with the understanding of basics principles. I am very fortunate to work within a team of dedicated, supportive, and learned colleagues both in my institution and within the wider community of physiologists through The Society. I feel I have a responsibility to promote science through public engagement. This together with undergraduate education is integral to inspiring the next generation of physiologists which will help shape the future of The Society. I feel this role as a Trustee to The Society will enable me to expand my commitment to The Society and help have an input into this future. I feel it is a great privilege to take on this role.

Stefan Trapp

University College London, UK

Discovering how something works has always excited me. At school my first love was physics, but by the time I started university that had changed to the physiology of living beings. Whilst the attraction of neurophysiology is probably obvious to most, I was also captivated by the prowess of archeabacteria in utilising various sources of metabolic energy, and even intrigued by plant physiology. Discussing the role of plant chloride channels was a rather unlikely part of my PhD viva on vagal neurons. Therefore, it is the fascination with mechanisms, physical, chemical, biological, or mechanical, which got me hooked on physiology.

Whilst not lacking confidence, I am indebted to my mentors for persuading me to go for the more ambitious alternatives presented to me during my career. This started with an inspiring neurophysiologist, Professor Reto Weiler, at Oldenburg University in Germany, who sent me to Oxford University to perform experiments for my final undergraduate project and persuaded me to pursue a PhD in Goettingen to learn the patch-clamp technique. This was just as Neher and Sakmann received the Nobel Price for developing this technique. My PhD, part of which was the first description of ATP-sensitive potassium channels in autonomic neurons, led to contacts with Professor Frances Ashcroft, and her invitation to host my German Travel Fellowship. Working with her team of outstanding scientists on structure and function of the newly cloned ATP-sensitive potassium channels was an exhilarating as well as humbling experience; it was Fran who convinced me that I have what it takes to pursue a successful academic career. I then obtained a MRC Career Development Award to start my own laboratory at UCL working on glucosensing circuits in the lower brainstem. Subsequently, I became a lecturer at Imperial College and further consolidated my laboratory's work on metabolic control circuits. In 2013, I moved back to UCL to join the Department of Neuroscience, Physiology & Pharmacology, where Alexander Gourine and I co-head the Centre for Cardiovascular and Metabolic Neuroscience. We combine classic physiological experimentation with the use of optogenetic and pharmacogenetic approaches to understand the circuitry of metabolic and cardiovascular regulation and its implications for diabetes, obesity, and heart disease.

The Physiological Society has been of great importance and value for my scientific career: by providing a forum to present my data and meet my peers, by offering travel grants to attend overseas meetings, and by hosting symposia that I had proposed. It is tempting to take it for granted that this will be available for the next generation of physiologists, but the fact is that my generation of physiologists now needs to step up and take responsibility. We have to determine where The Physiological Society is heading and define how it best serves the interest of academic physiologists and physiology as a discipline in an ever-changing political and economical landscape. I am ready to play my part to ensure that The Physiological Society continues to be the success it has been for almost 150 years.

Sensory transduction in insects

8 December 2017

Hodgkin Huxley House, London, UK

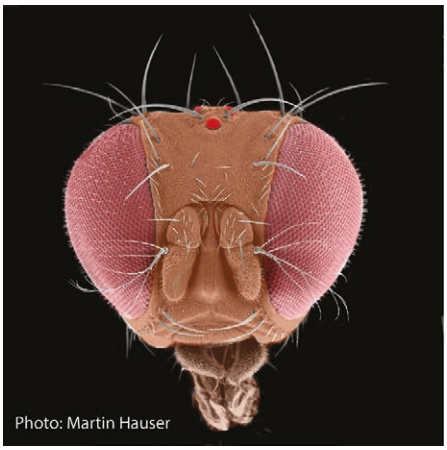
Photo: Martin Hauser


Photo: Tom Matheson


Photo: Marta Andrés

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