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**ESS 17th Congress: numerous “premières”**

For the first time since the founding of the European Shock Society, 33 years ago, its congress will be held in France, and Paris will host the 17th congress of the ESS from September 13th to September 15th, 2017.

An international committee has proposed 55 different topics, and 15 of them have been selected by the members of the ESS Executive committee and by the French Organizing Committee.

Three “premières” will happen:

- For the first time, based on the initiative of Markus Huber-Lang (our President-Elect), a student-oriented “Shock Summer School” will be organized on the very first morning of the congress. Its general topic will be “sepsis” and it will be fittingly held during the “World Sepsis Day”.
- For the first time ever, a joint session with the Chinese Shock Society will be organized.
- For the first time ever (yes, really!) a joint session with the US Shock Society will be organized.


The International Board and the national organizing committee have proposed numerous topics to be covered during the congress. Finally, 15 have been retained (see below).
I would love to be a tourist in Paris!

Participating in a scientific meeting offers a wonderful opportunity to discover a new place. So will be the ESS PARIS 2017. We are setting a memorable and outstanding scientific program that you should not miss. Meanwhile, do not forget to schedule a few extra days to visit Paris (and surroundings) to enjoy some of the great places it has to offer. Indeed, as a Parisian, I do not enjoy the city as I should; rather, I regularly moan about the traffic jams, the frequent strikes, the sad faces of the Parisians and their bad mood, the grey skies, the pollution and the dog feces on the sidewalks! Let me assure you though, Paris, the city of lights is worth visiting despite the moaning Parisians like myself!

I am still far away from having visited all 85 (or more) museums which exist in Paris. Thanks to an American colleague who gave me the address, I finally discovered and enjoyed the Musée Dapper, a unique place on artistic heritage of Africa! Thus, I would very much love to be a tourist in Paris as well. Let me take you with me for a virtual stroll in Paris; hopefully, you will decide to visit some of these places for real...

Just before I begin, let me provide you with a short historical background: In 250-200 BC the Parisii, a Celtic tribe make settlements on the Ile de la Cité; the city of Lutèce becomes Roman in 52 BC (by the way, do not miss the vestiges of the foundations from the Roman period in front of the Notre-Dame). In 508, Clovis makes Paris his capital. In 1190, the king Philippe August built the first royal fortress which during the next centuries will be regularly transformed, improved, enlarged by successive kings as their royal monumental palace, the Louvre. Its function as royal headquarters had lasted until the end of the XVIIth century when Louis XIVth moved to Versailles. In 1231, La Sorbonne is founded as a college of theology for underprivileged students, and in 1794, the École Polytechnique, the world's earliest technical college opens. In 1837, the first French railway connection opens with service from Paris to Saint-Germain. In 1995, the Paris mayor is elected president of France.
Paris, the city of lights.... this implies that you will have to stroll its streets during the night. Do not miss the flashing lights on the Eiffel Tower, reminiscent of the events and the fire works, which welcomed the year 2000. Do not miss the illuminations of the bridges, created to enter Paris/France into the new millennium. Altogether, thirty six bridges span the river Seine. My favorite bridge: the Pont Neuf ("new bridge"), indeed the oldest one, inaugurated in 1603 by king Henri the IVth, and the Pont Alexandre III, with its numerous sculptured decorations, the most elegant one of them all, inaugurated for the world exhibition of 1900. (To be continued...)

The Pont Neuf by an early XXth century Parisian artist

The Alexandre III bridge - a pastel by Daniel Champagne

Jean-Marc Cavaillon
An open competition to define the ESS Motto

The motto of the City of Paris is “Fluctuat nec mergitur” (Tossed but not sunk). Of course, it could be fittingly used for our society as well (!), but I invite you to propose a different (better) one!

Presently, I personally propose one uttered by Charles Richet (1850-1935), Nobel prize winner in 1913 for his discovery of anaphylaxis. In 1888, during his inaugural lecture at the Paris Medical School, he stated:

"To oppose the physician to the physiologist and the scientist to the clinician, means that one has understood neither physiology nor medicine."

Go ahead, propose a better one....

Jean-Marc Cavaillon

“There is no such thing as helplessness. It's just another word for giving up.”

Jefferson Smith, Strange Places

Marcin Osuchowski
Famous people who died of sepsis

Cassius Clay, alias Muhammad Ali (74), was an amazing, successful, and to some the greatest boxer ever. Apart from the ring, he also fought against wars, racism and was a strong activist for peace and human rights. His recent death (June 3rd, 2016) reminds us that sepsis remains a real threat all around the world, killing around 8 million people annually including mothers at childbirth and neonates (Source: Global Sepsis Alliance). Muhammad Ali is not the first famous person to die of sepsis; below are further examples of celebrities who had gotten sick with sepsis and died because of it.

The word "septicemia" was coined in 1837 by Pierre A. Piorry (1794 -1879), a Parisian doctor very attached to the use of the right words. He created several neologisms including the word septicemia from the Greek word Σήψις, (sepsis) which means putrefaction, and αίμα (aima), which means blood.

Sepsis does not differentiate between the common people and celebrities and kills across all ranks of society and aristocracy. Let us begin with Lucrece Borgia, a daughter of cardinal Roderic Borgia (who became the pope Alexander VI). She was 39, when on June 24th, 1519, she died of puerperal sepsis when giving birth to her eighth child (from her third marriage). The child, Isabella Maria d’Este, survived but later died at the age of three.

The French composer, Jean Baptiste Lully died of sepsis in 1687, after injuring himself with a long conducting staff during Te Deum performed to celebrate recovery of the King Louis XIV from surgery. He had struck his foot (the big toe) that ended in a local infection gone systemic after he refused any treatment. Of note, quite a large number of other famous composers died of sepsis: Johann Sebastian Bach (1750), Gioacchino Rossini (1868), Georges Bizet (1875), Gustav Mahler (1911), and Aleksander Skrjabin (1915) [1].

A very sad end of Dr. Ignac Semmelweis from Budapest occurred after he had deciphered the cause of puerperal sepsis during his work in Vienna. The culprits turned out to be the hands of the medical students and doctors (or rather the pathogens they carried) who assisted at childbirths after having made corpse dissections. He had requested the students to wash their hands with a solution of calcium hypochlorite after autopsies and in effect the mortality of laboring mothers decreased dramatically. His findings were ridiculed and dismissed by the contemporaries, and Semmelweis was eventually hospitalized in an asylum where he was injured in a finger and died of sepsis in 1865 at the age of 47. Ironically, his autopsy was performed in the very hospital in which he had made his key observations.
The French painter, **Edouard Manet** had been suffering from severe syphilis that ended in a partial paralysis of his legs and a gangrene of his right foot, which required amputation. He died of sepsis eleven days after the surgery (in 1883) at the age of 51.

**Albert Neisser** (left), the famous German physician and bacteriologist who discovered the pathogen responsible of gonorrhea, (named *Neisseria gonorrhoeae*) died of sepsis in 1916 after having suffered from kidney stones. **Heinrich Hertz** (right), a German physicist who proved the existence of electromagnetic waves, died of sepsis after a tooth abscess two months before his 47th birthday.

Sepsis also killed some heads of states: **Louis XV, King of France** (1774), **Alexander I of Greece** (1920), **Rainier III, Prince of Monaco** (2005), and **pope John-Paul II** (2005). Actors were also the victims: **Rudolph Valentino** (1926), **Christopher Reeve** (2004), **Christian Brando**, (49), the eldest son of the Hollywood actor Marlon Brando (2008). Sportsmen like **Socrates**, a famous football player, former captain of the Brazilian team, died at the age of 57 (2011).

To end this terrifying list, let us mention one more unknown but famous person: **Miss Agnès Souret**. She was 26 when she died of sepsis during her tour in Argentina. In 1920, she had been elected the very first Miss France at the age of 18.


by Jean-Marc Cavaillon
Back to the historical roots II.
Congresses with participation of the ESS

1983 (April 15-17) Malmö, Sweden
European Shock Society
Constitutional Meeting
David H. Lewis
Board:
“Past-President”: David H. Lewis
President: Ian Mc. A. Ledingham
President Elect: Konrad Messmer
General Secretary: Ulf Haglund
Treasurer: Roderick A. Little
Vice Presidents: Sándor Nagy,
Gian Paolo Novelli, Jean Louis Vincent

1984 (September 8) Manchester England
The scientific basis of the care of the critically ill &
1st Meeting of the European Shock Society
Roderick A. Little
Treasurer: Roderick A. Little

1986 (June) Linköping, Sweden
2nd Meeting of the European Shock Society
David H. Lewis
Circ Shock. 1986 19(1)
1987 (March 1-2) Brussels, Belgium
European Conference on Septic Shock of the European Society of Intensive Care Medicine and the European Shock Society
Ian Mc. A. Ledingham, Lambert G. Thijs, Konrad Messmer

1988 (May 2-5) Bologna, Italy
XXIII Congress of the European Society for Surgical Research (Giuseppe A. Ussia) / 3rd Meeting of the European Shock Society
President: Konrad Messmer (Heidelberg/FRG)
Prof. Walter Brendel (Münich, Germany) was awarded Honorary membership of the ESSR.

1990 (June 7-8) Amsterdam, The Netherlands
4th Meeting of the European Shock Society
Lambert G. Thijs

1991 (June 2-6) Vienna, Austria
“Second international Conference on Shock” / 3rd Vienna Shock Forum / 5th Meeting of the European Shock Society / 14th Annual Meeting of the Shock Society (USA)
(in association with the Japanese Shock Society and the International Endotoxin Society)
Günther Schlag

1993 (May 9-13) Vienna, Austria
4th Vienna Shock Forum in Association with the European Shock Society
Günther Schlag
1994 (September 16-17) Årensberg (Stockholm), Sweden
6th Congress of the European Shock Society
Ulf Haglund

1995 (May 7-11) Vienna, Austria
5th Vienna Shock Forum in association with the European Shock Society
Günther Schlag
Shock. 1995;3 Suppl 1

1996 (April 18-20) Manchester, England
7th Congress of the European Shock Society
Board (1994-1996):
President: Roderick A. Little
Past-President: Ulf Haglund
President-Elect: Jean Louis Vincent
Secretary: Heinz Redl
Treasurer: Uwe Brückner
Council: Ansgar Aasen, H.R.J.A. Goris, János Hamar, Olle Ljunkqvist, Jim Parratt,
Nominating Committee: Günther Schlag, Lambert Thijs, Ulf Haglund
Honorary Members: David Lewis, Günther Schlag

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1997 (November 8-11) Vienna, Austria
6th Vienna Shock Forum in association with the European Shock Society
Günther Schlag

1998 (October 15-17) La Hulpe, Belgium
8th Congress of the European Shock Society
Board (1996-1998):
President: Jean Louis Vincent
Past-President: Roderick A. Little
President-Elect: H.R.J.A. Goris
Secretary: Heinz Redl
Treasurer: Uwe Brückner
Council: Ansgar Aasen, János Hamar, Olle Ljunkqvist, Jim Parratt, Edmund Neugebauer
Nominating Committee: Lambert Thijs, Ulf Haglund, Roderick A. Little
Honorary Members: David Lewis, Günther Schlag

1999 (November 13-16) Vienna, Austria
7th Vienna Shock Forum in association with the European Shock Society
Günther Schlag

2000 (February 25-March 4) Munich, Germany
5th World Congress on Trauma, Shock, Inflammation and Sepsis – Pathophysiology, Immune Consequences and Therapy
Eugen Faist
2000 (May 25-27) Nijmegen, The Netherlands
Joint Meeting of the Surgical Infection Society of Europe (SIS-E) and the European Shock Society / 9th Congress of the European Shock Society
President: H.R. Jan A. Goris
Past-President: Jean Louis Vincent
President-Elect: Ansgar Aasen
Secretary: Heinz Redl
Treasurer: Uwe Brückner
Council: János Hamar, Olle Ljungqvist, Edmund Neugebauer, Didier Payen, Christoph Thiemermann, Nominating Committee: Ulf Haglund, Roderick A. Little, Jean Louis Vincent, Honorary Members: David Lewis, Günther Schlag

2002 (September 5-7) Oslo, Norway
10th Congress of the European Shock Society
Board (2000-2002):
President: Ansgar O. Aasen
Past-President: H.R. Jan A. Goris
President-Elect: Heinz Redl
Secretary: Michael Bauer
Treasurer: Uwe Brückner
Council: Didier Payen, Edmund Neugebauer, Christoph Thiemermann, Ishad H Chaudry, János Hamar

2004 (March 2-6) Munich Germany
6th World Congress on Trauma, Shock, Inflammation and Sepsis / 5th International Congress on Shock (International Federation of Shock Societies
Eugen Faist
2005 (January 27-30) Vienna, Austria
11th Congress of the European Shock Society / 8th Vienna Shock Forum
Board (2002-2004):
President: Heinz Redl
Past-President: Ansgar O. Aasen
President-Elect: Uwe Brückner
Secretary: Michael Bauer
Treasurer: Ulrich Schade
Council: S. Bahrami, S. Cuzzocrea, R.J.A. Goris, A.B.J. Groenevald, J.
Hamar, M. Jochum, M. Mythen, E. Neugebauer, D. Payen, C. Christoph
Thiemermann
Prof. H.R. Jan A. Goris, (Nijmegen, The Netherlands) was awarded
Honorary membership of the ESS.

2006 (September 14–16) Ulm, Germany
12th Congress of the European Shock Society
Board (2004-2006):
President: Uwe B. Brückner
Past-President: Heinz Redl
President-Elect: Christoph Thiemermann
Secretary: Monty Mythen
Treasurer: Ulrich Schade
Council: Marianne Jochum, Salvatore Cuzzocrea, Johann Groeneveld, Edmund
Neugebauer, Soheyl Bahrami

2008 (June 28-July 2) Cologne, Germany
6th Congress of the Federation of Shock Societies / 31th Annual Conference on Shock (US Shock Society)
Edmund Neugebauer

2009 (September 24-26) Lisbon, Portugal
13th Congress of the European Shock Society
Board (2006-2009):
President: Christoph Thiemermann
Past-President: Uwe B. Brückner
President-Elect: Salvatore Cuzzocrea
Secretary: Monty Mythen
Treasurer: Ulrich Schade
Council: Soheyl Bahrami, Inge Bauer, Mihály Boros, Edmund Neugebauer,
Jacob Wang
2011 (August 31- September 2) Taormina – Giardini Naxos, Italy
14th Congress of the European Shock Society
Board (2009-2011):
President: Salvatore Cuzzocrea
Past-President: Christoph Thiemermann
President-Elect: Soheyl Bahrami
Secretary: Inge Bauer
Treasurer: Ingo Marzi
Council: Mihály Boros, Bruno Levy, Markus Huber-Lang, Yngvar Gundersen, Helder Mota-Filipe

2013 (September 25-29) Vienna, Austria
15th Congress of the European Shock Society
Board (2011-2013):
President: Soheyl Bahrami
Past-President: Salvatore Cuzzocrea
President-Elect: Edmund Neugebauer
Secretary: Inge Bauer
Treasurer: Marcin F. Osuchowski
Council: Emanuela Esposito, Andrea Szabó, Lev V. Gerasimov, Markus Huber-Lang, Andreas Spittler


2015 (September 24-26) Cologne, Germany
16th Congress of the European Shock Society / 14th International Conference on Complex Acute Illness (ICCAI)
President: Edmund Neugebauer
Past-President: Soheyl Bahrami
President-Elect: Jean-Marc Cavaillon
Secretary: Inge Bauer
Treasurer: Marcin F. Osuchowski
Council: Emanuela Esposito, Andrea Szabó, Lev V. Gerasimov, Markus Huber-Lang, Andreas Spittler

2017 (September 13 - 17) Paris, France
17th Congress of the European Shock Society
Board (2015-2017):
President: Jean-Marc Cavaillon
Past-President: Edmund Neugebauer
President-Elect: Markus Huber-Lang
Secretary: Inge Bauer
Treasurer: Marcin F. Osuchowski
Council: Emanuela Esposito, Artem N. Kuzovlev, Marc Maegle, Andrea Szabó, Andreas Spittler

by Andrea Szabó

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Journal Club: What is new in shock research?

Highlights of remarkable findings recently published in shock research


Main important messages:
The authors, using the cecal ligation and puncture model, observed that sepsis induces osteoblast ablation and rapid bone loss. They also found out that the common myeloid progenitors were increased “days post-sepsis, whereas the common lymphoid progenitors were decreased.” The authors devised an inducible deletion system that allowed them to delete osteoblasts. This deletion led to T and B lymphopenia. The osteoblast ablation was shown to be independent of the MyD88 and TRIF signaling pathways, but the process was mainly G-CSF dependent. The authors further demonstrated that the osteoblasts are a source of IL-7 and, thus, the reduction of osteoblast frequency after sepsis is associated with a reduction of IL-7; a key cytokine in lymphopoiesis. The use of osteoblast activators or IL-7 could rescue mice undergoing lethal sepsis.

Written by: Jean-Marc Cavaillon, Institut Pasteur Paris


The authors of this great review emphasize that (due to earlier recognition and more appropriate treatments) sepsis is regarded less of an immediate life-threatening disorder, but more of a long-term chronic critical illness which is associated with prolonged inflammation, immune suppression and tissue/organ injury. Furthermore, patients who survive sepsis have continuing risk of mortality after discharge showing also long-term cognitive and functional deficits.

The review contains excellent updates on the pathophysiology of sepsis (including inflammation, early gene activation, the C5a–C5a receptor axis, immune suppression, endothelial barrier dysfunction, coagulation and effect on organ systems and others) also depicting the diagnostic possibilities, screening and prevention as well as post-discharge management and quality of life of septic patients.

Excellent illustrations summarize main cell-surface and intracellular receptors responsible for the recognition of microbial products and endogenous danger signals (i.e. alarmins). Current conceptual model of sepsis outcome and late immunosuppressive effects of sepsis are also depicted. The review also contains updates on the changes in the vascular endothelium (in response to inflammatory stimuli during sepsis) and the coagulation – inflammation interactions occurring during sepsis. The article also demonstrates the limited efficacy of biological response modifiers, immunomodulatory agents and of the available biomarkers for diagnosis of sepsis and prediction of clinical outcome.

Written by: Andrea Szabó, University of Szeged
**Source:** Krezalek MA et al. The Shift of an Intestinal "Microbiome" to a "Pathobiome" Governs the Course and Outcome of Sepsis Following Surgical Injury. *Shock.* 2016;45(5):475-82. ([link to abstract](#))

**Short summary:** The recent publication by Krezalek MA et al. is a very interesting review article about the so far rarely considered impact of the intestinal microbiome to the pathophysiology of sepsis. Although early mortality rate after surgical injury decreased in the last ten years due to early resuscitation efforts and antibiotic therapy, we are now challenged by “late onset sepsis”, which represents the most common cause of death in modern surgical intensive care units.

The authors state that this time shift might be the result of an alteration of the commensal microbiome to a pathologic prototype due to endogenous chemosignals which are released during surgical injury. For example, mouse studies have shown that after intestinal ischemia-reperfusion injury, K-opioid peptide dynorphin is released into the intestinal tract, where it activates a series of virulence genes in *P. aeruginosa*, leading to disruption of the epithelial barrier, bacterial translocation, sepsis and death. Furthermore, as early as 6 h after major injury, lactobacilli and anaerobes as well as their cytoprotective metabolites decreased by more than 90%, suggesting that chemosignals affect both the composition and the functionality of the microbiota. Whereas the intestinal epithelium and its underlying innate and adaptive immune cells are probably capable to discriminate between symbionts and pathobionts, administered antibiotics are definitely not and therefore induce a loss of the microbiome diversity and allow predator-type pathogens to predominate.

Thus, novel therapeutic approaches are required that preserve the gut microbiome and eliminate all offending pathogens or rather their virulence tactics. In this regard, the authors developed a phosphorylated high-molecular-weight polyethylene glycol (Pi-PEG) molecule, which has the capacity to control the strength of normal microbiota to directly suppress the virulence of the pathobiota.

This review article focuses on the so far underestimated impact of the microbiome change in the pathophysiology of sepsis and might open both the researchers and clinicians a novel and promising therapeutic platform in still challenging treatment of sepsis.

**Written by:** Markus Huber-Lang, University of Ulm

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**Summary:** Acute kidney injury (AKI) is a common complication in hospitalized patients and is associated with short-term morbidity, long-term risk of chronic kidney disease and cardiovascular events and decreased survival. Effective strategies to prevent AKI do not exist. Since the first description of the phenomenon “remote ischemic preconditioning” (RIPC) in 1993 by Przyklenk et al., most studies concentrated on the heart. However, there is experimental and clinical evidence suggesting that RIPC has protective effects also on the kidney. This review of randomized controlled trials (based on PubMed search and review of bibliographies of relevant articles to identify additional citations) summarizes the current knowledge. A total of 17 clinical trials were included. While 9 studies report protective effects of RIPC on AKI development/severity, 8 studies have failed to show beneficial effects of RIPC on kidney function. The authors provide potential explanations for these divergent observations. Furthermore, information on RIPC mechanisms and the role of biomarkers in predicting organ protection and damage is provided.
### Main important messages:
- The overall prevalence of AKI in critically ill patients is high.
- Results of controlled clinical trials investigating RIPC as a therapeutic option to protect kidney function are controversial.
- High-risk patients undergoing cardiac surgery might benefit from RIPC.
- Further large multicenter trials are needed to elucidate the therapeutic potential of RIPC, to identify the optimal RIPC protocol and patient population and to investigate the role of (novel) biomarkers.

**Written by:** Inge Bauer, University Hospital Duesseldorf

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#### Prostaglandin E2 and systemic inflammation


### Main important messages:

→ The role of prostaglandins in systemic inflammation remains mysterious. In blood cells from septic patients, there was a downregulation of the expression of PGE2 receptor 4 (EP4) and membrane associated PGE2 synthase-2. On the contrary, there was an up-regulation of the gene for PGE2 degredating enzyme, 15-PGDH.

→ Inhibition of PGE2 synthesis by indomethacin augmented LPS-induced inflammatory cytokine production along with other signs of systemic inflammation such as splenomegaly or neutrophil infiltrates. However, co-administration of EP4 agonist prevents these effects. Moreover, inhibition of PGE2 synthesis results in increased dissemination of gut bacteria and this effect is independent of adaptive immunity.

→ The mechanism of anti-inflammatory and gut-barrier protective actions of PGE2 depends on the IL-22 production by gut-resident type 3 innate-lymphoid cells (ILCs3) which subsequently acts on epithelial cells. Interestingly, in clinical settings of sterile inflammation during acute pancreatitis, the plasma level of IL-22 is decreased. PGE2 also stimulates IL-23-driven differentiation and activation of ILCs3 what shows this mediator as the main orchestrator of this regulatory axis.

This study broadens our understanding of the role of prostaglandins in systemic inflammation. Unraveling the important role of PGE2-ILCs3-IL22 axis sheds light on the sophisticated crosstalk between innate immunity, epithelial barrier, gut microbiome during critical illness. What is more, these results provide new explanation for the failures of the trials of COXs inhibitors in systemic inflammatory related diseases as sepsis.

**Written by:** Tomasz Skirecki, Center of Postgraduate Medical Education, Warsaw
Journal Club Special

Crisis of Reproducibility:
Is there one? Should we care?

Recently, a visiting professor from Germany whom we invited to our institute’s seminar series, reminded me about the “ancient” ways of experimentation and hypothesis testing done by the scientific Giants of Old (on whose shoulders we now proverbially stand, according to Google scholar), i.e., exhaust all imaginable experimental options to prove a hypothesis is false. Only once those disproving attempts fail and reiterations of the original study demonstrate the same outcome, the posited hypothesis is taken as true. What an admirable and trustworthy way of performing science, isn’t it? Yet, when one contemplates this in the context of contemporary experimental conduct, one may realize that we may be not exactly following the footsteps of those giants (let alone standing on their shoulders).

Along the above lines, the two most recent pieces in Nature have put strong focus on the issue of reproducibility in science. The first one by Monya Baker published in May this year (1), is a survey-based study (1576 participating researchers) stressing that inability of replicate experiments (both own and those by other labs) is rife and extends beyond the 50% mark in chemistry, biology and medicine (irrespective in which category). *Nihil novi*, one may say, it happens to all of us. True, as long as such a discrepancy is successfully sorted out in the inquiring and critical confines of one’s lab – just like in the Old Times. The problems becomes serious when insufficiently (experimentally) verified information reaches the open space of World Wide Web and gets rapidly disseminated (as in the New Times?). Based on the received survey responses, the article asserts that we face too much of the latter and that the awareness of this flaw must increase in the scientific community. The top culprits named for poor reproducibility: 1) pressure to publish and 2) selective reporting. Sounds familiar, right?

The second piece (an editorial published on 25 August; (2)) builds upon the first one, showing already implemented effective solutions to the problem of poor reproducibility and urging fostering of a better climate for this kind of research. There is no doubt that a reproduction paper is not “sexy”; it simply re-iterates the original brilliant idea/design of the first study, further exposing its glory. Well, unless it turns out the replication attempt does not quite replicate the novel finding. Then the second-in-line paper rapidly picks up on the “coolness” factor. Until the third one proves them all wrong and “we are still totally confused but on a much higher level” in the words of the Prof. Konrad Messmer (one the ESS founders). Naturally, none of the above scenarios is wanted.
given the undesired confusion and the strong emotional factor that may enter the scene as well. We all, no doubts, wish for all the new original studies/discoveries to stand strong. But even so-called “landmark” studies or those published in the most respected journals do not automatically guarantee confidence. Biotech company Amgen was able to confirmed only 6 out 53 “landmark” papers (in haematology and oncology) (3) while Amyotrophic Lateral Sclerosis (ALS) Therapy Development Institute failed to reproduce any of the eight original (and beneficial) anti-ALS mouse studies (4). It is clear that the strength of such important discoveries does rest on the shoulders of the follow up reiterations. Without them, they remain as novel, exciting, brilliant and hopeful but unconfirmed findings if we give any heed to the words of Andreas Vesalius: “I am not accustomed to saying anything with certainty after only one or two observations.” (1546; from the Letter on the China Root).

I sincerely encourage you to read those most recent as well as the older related articles; they are worth your time. Once you do, do ask yourself the question. You know well what question; I asked it myself too. It is a good start to get back on track if we ourselves might have occasionally strayed from the footsteps of the Giants of Old.

Thus, Go forth and replicate!

Marcin Osuchowski

References:

Welcome new ESS members

We cordially welcome our new members who joined the ESS in 2016:

• Athanasios Chalkias, MD, M.Sc., PhD
  Athens, Greece

• Benedicte De Winter, MD, PhD
  Antwerp, Belgium

• James Fullerton, MD
  London, UK

• Roosmarijn E. Vandenbroucke, PhD
  Ghent, Belgium
Invitation to publish in Shock®

REMINDER

Shock is a monthly journal that publishes the results of investigations in the field of injury, inflammation and sepsis; of clinical and laboratory origin alike (current IF=3.05). It is the official Journal of all international Shock Societies, including ESS. Thanks to its efficient reviewing process, you will typically have your submitted paper reviewed within 15 days.

So do not hesitate, submit your next best results to SHOCK!

Meeting updates

**LIVES 2016**
October 1-5, 2016
Milan, Italy

[http://www.esicm.org/events/annual-congress](http://www.esicm.org/events/annual-congress)

**8th Congress of the International Federation of Shock Societies**
October 3-5, 2016
Tokyo, Japan


**26th European Organ Donation Congress**
October 28-29, 2016
Barcelona, Spain

[http://www.esot.org/events-education/events/4118/overview](http://www.esot.org/events-education/events/4118/overview)

**XVII. Congress of The European Shock Society**
September 13-15, 2017
Paris, France

The Executive Committee of the ESS

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https://research.pasteur.fr/en/team/cytokines-and-inflammation/

The Past-President:

Edmund Neugebauer, PhD
Senior Professor: Health Services Research
Former Director Institute for Research in Operative Medicine & Chair for Surgical Research
Faculty of Health- School of Medicine
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www.uni-wh.de/versorgungsforschung

The President-Elect: [elected 2015]

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The Treasurer:

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Dear present ESS member,

If you like your ESS Summer Newsletter, please feel free to share it with your colleagues in the lab, department and/or institute. Perhaps, you could use this opportunity to suggest them to join us (a registration form can be found at the end of this Newsletter). Do not forget that we need you to keep improving our society so it stands proud and strong among other international Shock Societies.

This Newsletter, put together by your peers, belongs to you! We invite you to identify with it as members of the ESS. Moreover, we ask you to help us make it even better. Accordingly, we would be delighted to publish in our next issue any input you might be wishing to share with us (e.g. discussion on a given research/popular science topic, announce available positions in your lab, a contribution to the journal club corner, historical memories, comments about sepsis 3.0 etc.)

Dear past ESS member,

Please do not forget to renew your membership. We need all colleagues, junior and senior alike, to enable the ESS to host in its ranks the best representatives of the European Shock research - at the bedside and/or at bench alike.

Jean-Marc Cavaillon
European Shock Society
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