



ESS Summer Newsletter 2016

Contents

ESS 17 th Congress: numerous “premières”	2
I would love to be a tourist in Paris!	3
An open competition to define the ESS Motto.....	5
Famous people who died of sepsis	6
Back to the historical roots II. Congresses with participation of the ESS	8
Journal Club: What is new in shock research?.....	15
Journal Club Special - Crisis of Reproducibility: Is there one? Should we care?.....	18
Welcome new ESS members	20
Invitation to publish in Shock®	21
Meeting updates.....	22
The Executive Committee of the ESS.....	23
Last words about ESS Newsletter	27

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.

Visit our WEB site: <http://www.essparis2017.org>

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).

	Amphi Jacob	CIS
13th Sept Morning	ESS Summer school Joint with WSD 2017 Welcome What is sepsis? How to model sepsis? What happens to the immune system during sepsis? What happens with the coagulation during sepsis? Why does sepsis induce multiple-organ failure? Where are innovative treatment strategies for sepsis?	Jean-Marc Cavaillon, Markus Huber-Lang 1. Markus Huber-Lang, GER 2. Marcin Osuchowski, AUT 3. Jean-Marc Cavaillon, FRA Pause 4. Tom van der Poll, NL 5. Irshad Chaudry USA 6. Chris Thiernemann, UK
13th Sept Afternoon	Session 2 Biomarkers of infection	Session 1 Revisiting sepsis definitions: impact and consequences Session 3 Immune dysregulation during sepsis and shock
13th Sept Evening		Poster session 1
14th Sept Morning	Session 4 Long term consequences of trauma and sepsis Session 5 Antibiotics and anti-microbial strategies	6. Joined Session Chinese Shock Society Physiopathology of sepsis and MODS

	Amphi Jacob	CIS
14th Sept Afternoon		7. Young Investigator Award
	Session 8 Non-bacterial sepsis	Session 9 Resolution of inflammation
14th Sept Evening		Poster session 2
15th Sept Morning	Session 10 Good or bad Gases	12. Joined Session US Shock Society New tracks for new therapies
	Session 11 Role of Comorbidities in the Danger Response to Shock	
15th Sept Afternoon	Session 13 Hemorrhagic shock	Session 15 Metabolic changes during sepsis/critical illness
	Session 14 Consequences of shock and sepsis on brain	Session 16 Animal models for human shock and sepsis

Jean-Marc Cavaillon

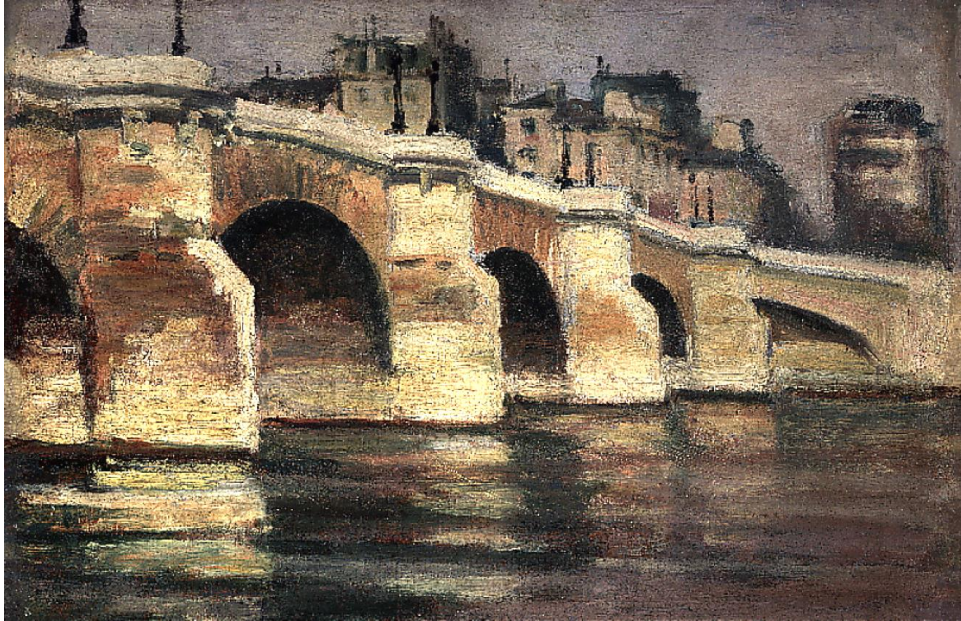
I would love to be a tourist in Paris!

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

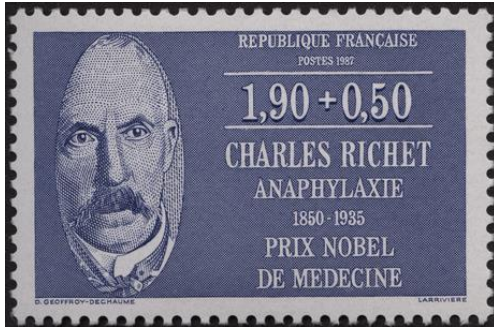


The Alexandre III bridge - a pastel by Daniel Champagne

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



Cause of Muhammad Ali's Death, Septic Shock, Targets Sick, Elderly

by ELISHA FIELDSTADT



► Tributes Pour In For Boxing's 'Greatest' Legend Muhammad Ali

<http://www.nbcnews.com/news/sports/cause-muhammad-ali-s-death-septic-shock-targets-sick-elderly-n585926>

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 αἷμα (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.

[1] Rietschel ET, Rietschel M, Beutler B. How the mighty have fallen: fatal infectious diseases of divine composers. Infect Dis Clin North Am. 2004 Jun;18(2):311-39.

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

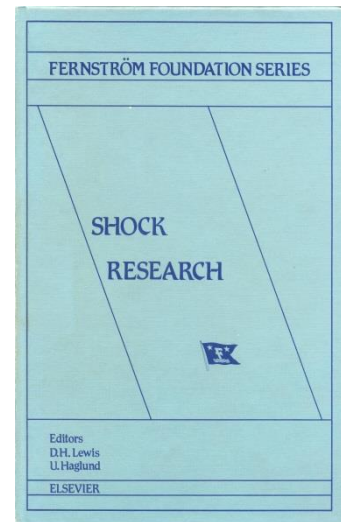
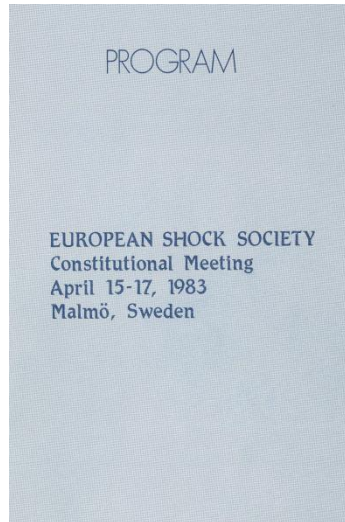
President Elect: Konrad Messmer

General Secretary: Ulf Haglund

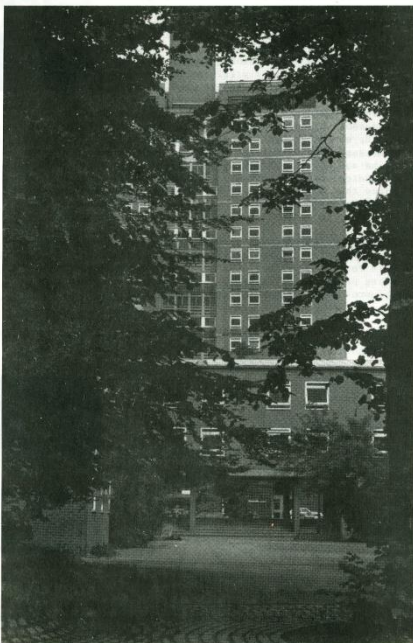
Treasurer: Roderick A. Little

Vice Presidents: Sándor Nagy,

Gian Paolo Novelli, Jean Louis Vincent



The Scientific Basis of the Care of the Critically Ill
&
European Shock Society



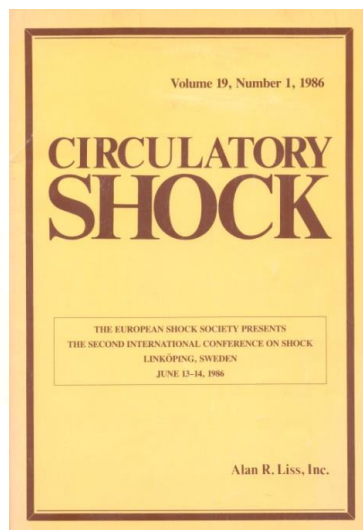
Owen's Park
University of Manchester
Manchester U.K.
5-8th September 1984

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

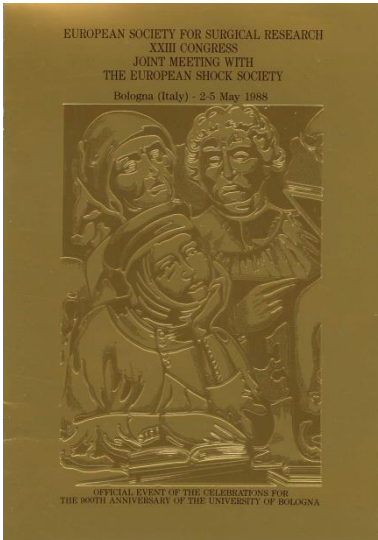
Circ Shock. 1986 19(1)

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

[Intensive Care Med. 1988;14\(3\):181-4.](#)

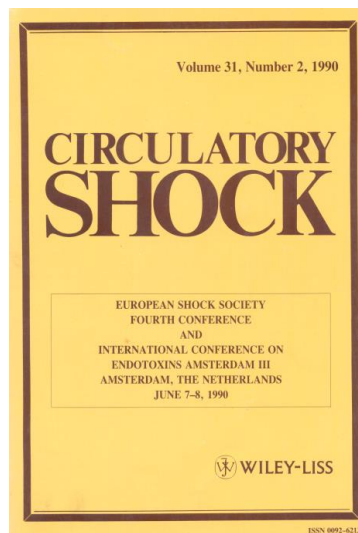


1988 (May 2-5) Bologna, Italy

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 (München, Germany) was awarded Honorary membership of the ESSR.

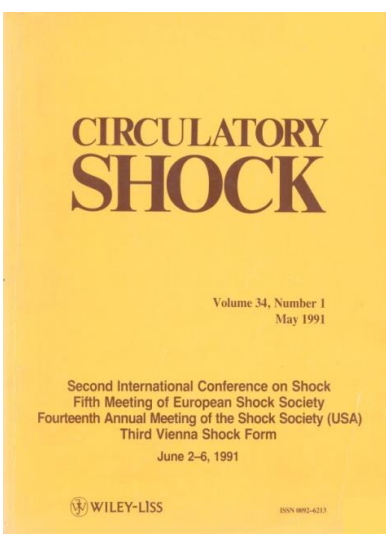


1990 (June 7-8) Amsterdam, The Netherlands

4th Meeting of the European Shock Society

Lambert G. Thijs

[Circ Shock. 1990 Jun;31\(2\):215-37.](#)



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

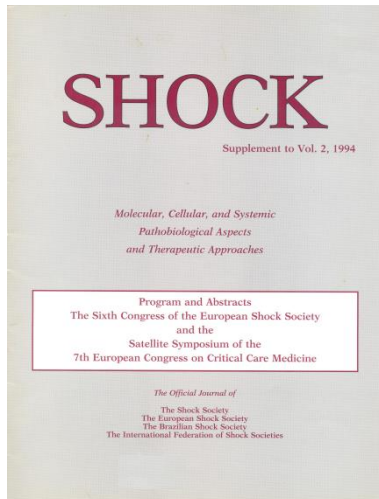
[Circ Shock. 1991 May;34\(1\):1-215.](#)

1993 (May 9-13) Vienna, Austria

4th Vienna Shock Forum in Association with the European Shock Society

Günther Schlag

[Circ Shock. 1993;Suppl 1:1-65.](#)

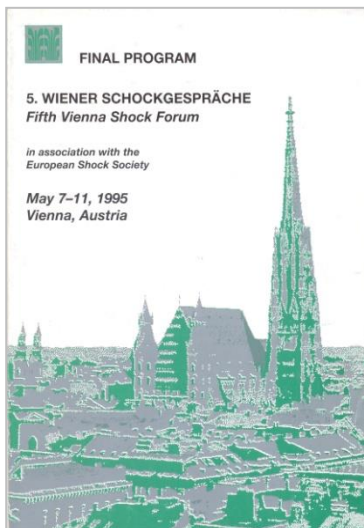


1994 (September 16-17) Årnsberg (Stockholm), Sweden

6th Congress of the European Shock Society

Ulf Haglund

[Shock. 1994;2 Suppl:1-44.](#)

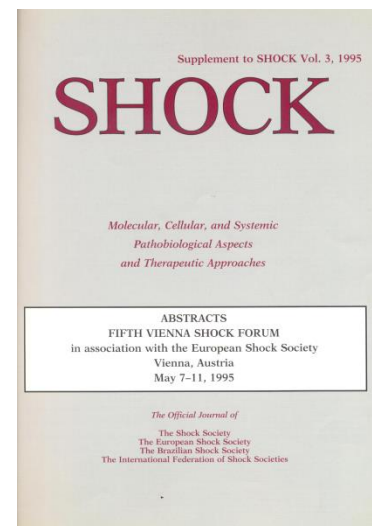


1995 (May 7-11) Vienna, Austria
5th Vienna Shock Forum in association

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

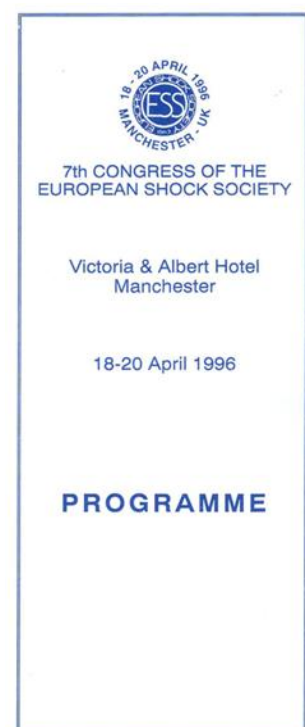
Treasurer: Uwe Brückner

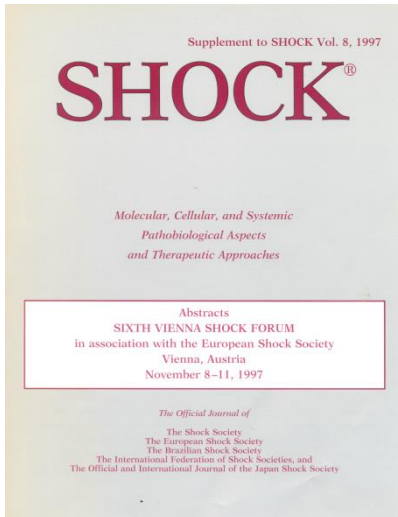
Council: Ansgar Aasen, H.R.J.A. Goris, János Hamar, Olle Ljungkqvist, Jim Parratt,

Nominating Committee: Günther Schlag, Lambert Thijs, Ulf Haglund

Honorary Members: David Lewis, Günther Schlag

[Shock. 1996;5 Suppl:1-24.](#)





1997 (November 8-11) Vienna, Austria

6th Vienna Shock Forum in association with the European Shock Society

Günther Schlag

[Shock. 1997;8 Suppl:1-72.](#)

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

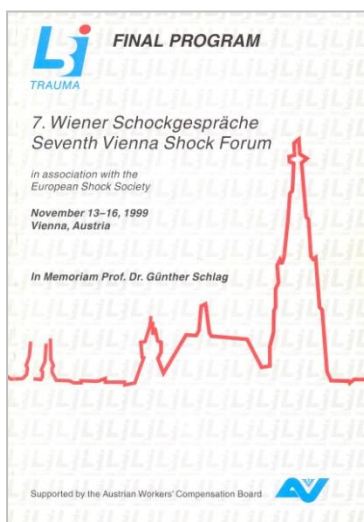
Treasurer: Uwe Brückner

Council: Ansgar Aasen, János Hamar, Olle Ljungkqvist, Jim Parratt, Edmund Neugebauer

Nominating Committee: Lambert Thijs, Ulf Haglund, Roderick A. Little

Honorary Members: David Lewis, Günther Schlag

[Shock. 1998;10 Suppl:1-19.](#)

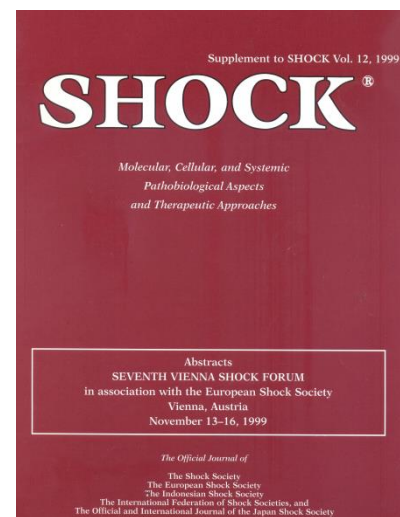


1999 (November 13-16) Vienna, Austria

7th Vienna Shock Forum in association with the European Shock Society

Günther Schlag

Shock 1999; 12 Suppl:1-60.

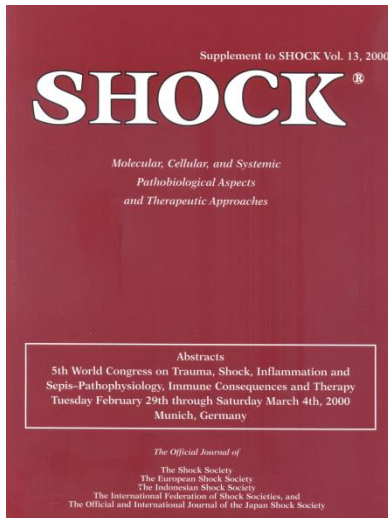


2000 (February 25-March 4) Munich, Germany

5th World Congress on Trauma, Shock, Inflammation and Sepsis – Pathophysiology, Immune Consequences and Therapy

Eugen Faist

[Shock. 2000;13 Suppl 1:1-177.](#)



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**

Board (1998-2000):

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 Ljungkqvist, Edmund Neugebauer, Didier Payen, Christoph Thiernemann, Nominating Committee: Ulf Haglund, Roderick A. Little, Jean Louis Vincent, Honorary Members: David Lewis, Günther Schlag †

Shock 2000; 13 Suppl 1: 1-177.

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

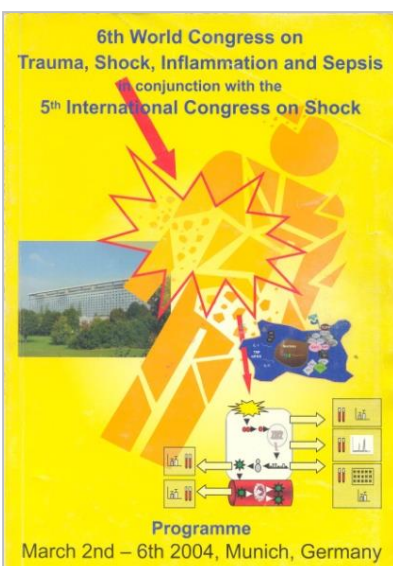
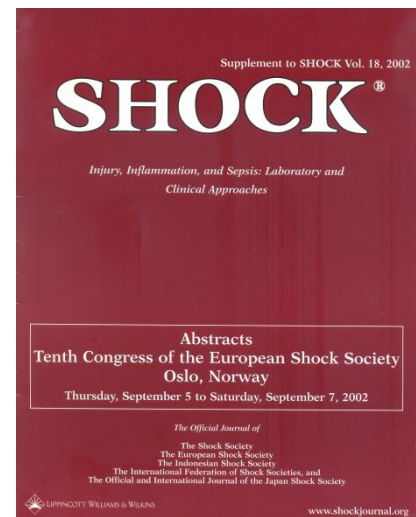
President-Elect: Heinz Redl

Secretary: Michael Bauer

Treasurer: Uwe Brückner

Council: Didier Payen, Edmund Neugebauer, Christoph Thiernemann, Ishad H Chaudry, János Hamar

[Shock. 2002;18 Suppl:1-30.](#)



2004 (March 2-6) Munich Germany

6th World Congress on Trauma, Shock 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

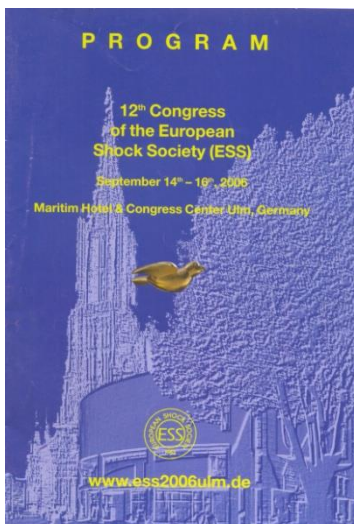
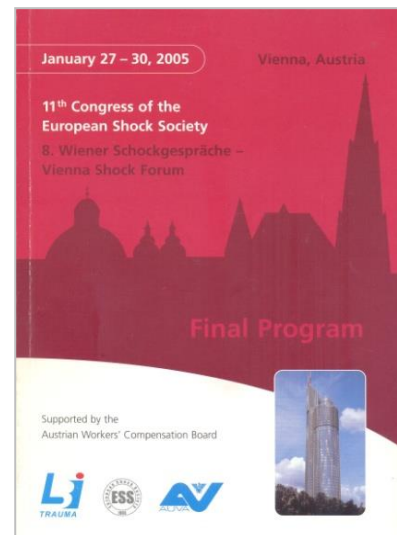
Council: S. Bahrami, S. Cuzzocrea, R.J.A. Goris, A.B.J. Groenevald, J.

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.

[Shock. 2005;23 Suppl 2:1-85.](#)

**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

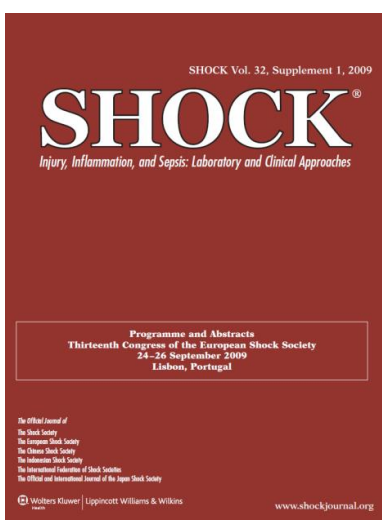
[Shock. 2006 Oct;26 Suppl 1:1-41.](#)

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

[Shock. 2008;29 Suppl 1:1-120.](#)

**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

Shock. 2009;32 Suppl 1.

2011 (August 31- September 2) Taormina – Giardini Naxos, Italy

14th Congress of the European Shock Society

Board (2009-2011):

President: Salvatore Cuzzocrea

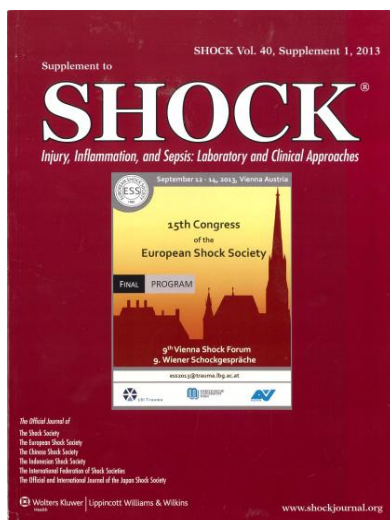
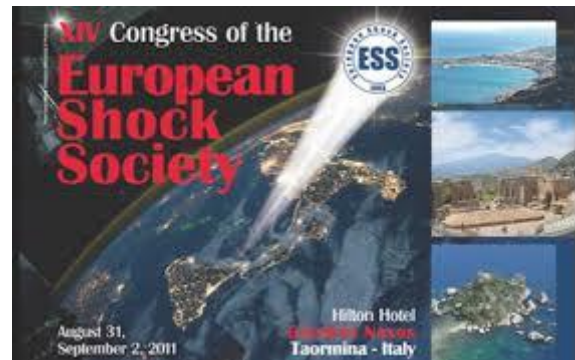
Past-President: Christoph Thiemermann

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

[Shock. 2013 Sep;40 Suppl 1:13-43.](#)



2015 (September 24-26) Cologne, Germany

16th Congress of the European Shock Society / 14th International Conference on Complex Acute Illness (ICCAI)

Board (2013-2015):

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 Maegele, Andrea Szabó, Andreas Spittler



by Andrea Szabó

[Back to Contents](#)

Journal Club: What is new in shock research?

Highlights of remarkable findings recently published in shock research

Sepsis, immunodeficiency

Source: Terashima A, Okamoto K, Nakashima T, Akira S, Ikuta K, Takayanagi H. Sepsis-Induced Osteoblast Ablation Causes Immunodeficiency. **Immunity**. 2016;44(6):1434-1443. doi: 10.1016/j.immuni.2016.05.012. Epub 2016 Jun 14. ([link to abstract](#))

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

Sepsis review

Source: Richard S. Hotchkiss, Lyle L. Moldawer, Steven M. Opal, Konrad Reinhart, Isaiah R. Turnbull, Jean-Louis Vincent. Sepsis and septic shock. **Nature Reviews Disease Primers** 2016; 1:1-21. ([link to paper](#))

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

Sepsis: when friends become foes

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

Remote ischemic preconditioning and kidney protection

Source: Zarbock A, Kellum JA. Remote Ischemic Preconditioning and Protection of the Kidney - A Novel Therapeutic Option. **Crit Care Med**. 2016 Mar;44(3):607-16. doi: 10.1097/CCM.000000000000138 ([link to abstract](#))

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.

[Back to Contents](#)

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

Prostaglandin E2 and systemic inflammation

Source: Duffin R *et al.* Prostaglandin E2 constrains systemic inflammation through an innate lymphoid cell–IL-22 axis. **Science.** 2016; 351(6279):1333-8 ([link to abstract](#))

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 degrading 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

FEATURE NEWS

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

[Back to Contents](#)

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:

- 1) [Nature, 26 May 2016; 533: 452-4.](#)
- 2) [Nature, 25 August 2016; 536: 373.](#)
- 3) [Nature, 29 March 2012; 483: 531.](#)
- 4) [Nature, 27 March 2014; 507: 381-3.](#)

Welcome new ESS members

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

WELCOME

- **Athanasios Chalkias, MD, M.Sc., PhD**
Athens, Greece



- **Benedicte De Winter, MD, PhD**
Antwerp, Belgium

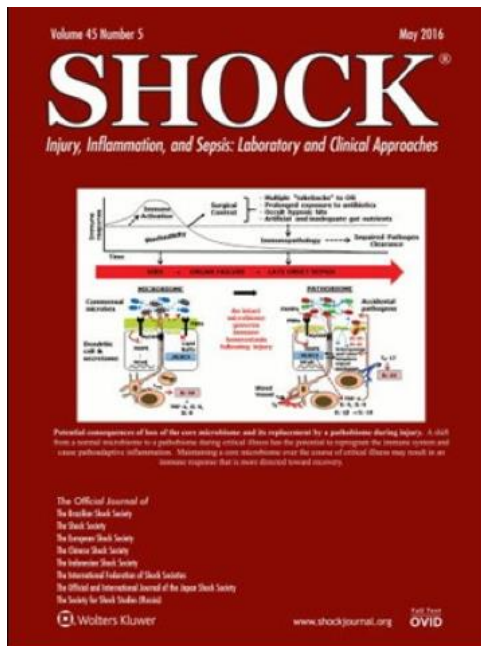
- **James Fullerton, MD**
London, UK



- **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!

<http://www.editorialmanager.com/shock>.

Meeting updates

LIVES 2016

October 1-5, 2016

Milan, Italy

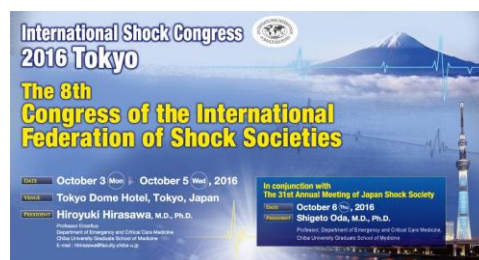


<http://www.esicm.org/events/annual-congress>

8th Congress of the International Federation of Shock Societies

October 3-5, 2016

Tokyo, Japan



<http://www.congre.co.jp/ifss2016/>

26th European Organ Donation Congress

October 28-29, 2016

Barcelona, Spain



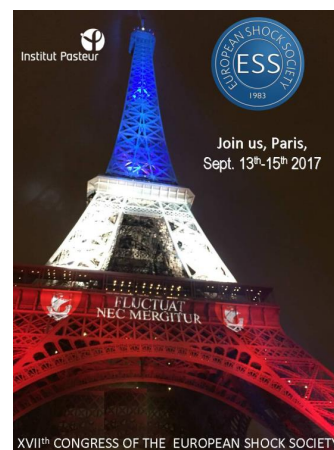
<http://www.esot.org/events-education/events/4118/overview>

XVII. Congress of The European Shock Society

September 13-15, 2017

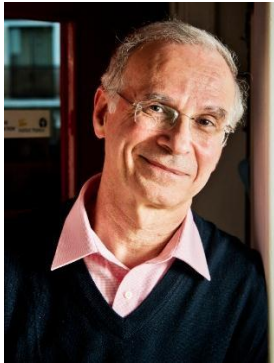
Paris, France

<http://www.essparis2017.org/>



The Executive Committee of the ESS

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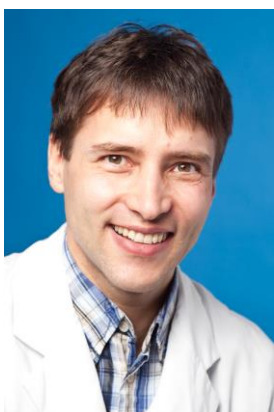
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LAST WORDS ABOUT



NEWSLETTER

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 Membership Application Form

Sign up online at www.europeanshocksociety.org/register

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or fax to:
+49 211 81 015 12053

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Email Address	<input type="text"/>
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Phone No.	<input type="text"/>

Area(s) of research interest:

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