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Editorial

A.L. Copley Best Paper Prize 2021

Friedrich Jung[a] and Philippe Connes[b,c,d] and Christian Lehmann[e,f,g,h]
[a]*Institute of Biotechnology, Molecular Cell Biology, Brandenburg University of Technology, Senftenberg, Germany*
[b]*Laboratoire Interuniversitaire de Biologie de la Motricité (LIBM) EA7424, Equipe "Biologie vasculaire et du globule rouge", Université Claude Bernard Lyon 1, COMUE, Lyon, France*
[c]*Laboratoire d'Excellence sur le globule rouge (Labex GR-Ex), Paris, France*
[d]*Institut Universitaire de France, Paris, France*
[e]*Department of Anesthesia, Pain Management and Perioperative Medicine, Dalhousie University, Halifax, NS, Canada*
[f]*Department of Microbiology and Biophysics, Dalhousie University, Halifax, NS, Canada*
[g]*Department of Pharmacology, Dalhousie University, Halifax, NS, Canada*
[h]*Department of Physiology and Biophysics, Dalhousie University, Halifax, NS, Canada*

The Editor-in-Chief and the Editorial Board of *Clinical Hemorheology and Microcirculation* (CHM), as well as the Publisher (IOS Press) have decided to set an annual prize, named the **A.L. Copley Best Paper Prize,** to recognize the best article published every year in CHM beginning in 2016. This prize has been named in honor of the Journal's founding editor, Alfred Lewin Copley. Dr. A.L. Copley was a German American medical scientist who introduced the term "Hemorheology" and defined this area of science.

First of all, the editorial team wish to thank all authors for their valuable contributions in 2021. A group of three editors was elected by the editorial board to select the best paper in a multistep process. The criteria for Prize selection included: originality and innovation, theoretical contribution, clarity of writing and presentation, and expected impact. In the first step, each of the three editors listed the 10 best papers separately. From these 30 papers the Prize committee looked for manuscripts which have been nominated independently by more than one editor (second step). This was the case for 7 out of the 30 papers. Out of these 7 papers each editor chose what he considered the best three papers and allocated 5 points to the best of the three, 3 points to the second best and 1 point to the third (third step). The total points were added for each paper, thus allowing the papers to be ranked. The highest-ranked paper was the work from G. Hagn and colleagues which now receives the **A.L. Copley Best Paper Prize 2021**:

> Hagn G, Holbein B, Zhou J, Lehmann C. **Anti-inflammatory iron chelator, DIBI, reduces leukocyte-endothelial adhesion and clinical symptoms of LPS-induced interstitial cystitis in mice.** Clin Hemorheol Microcirc. 2021;79(3):395-406. doi: 10.3233/CH-201078.

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Three papers shared the second place:

Krüger-Genge A, Steinbrecht S, Jung CGH, Westphal S, Klöpzig S, Waldeck P, Küpper JH, Storsberg J, Jung F. **Arthrospira platensis accelerates the formation of an endothelial cell monolayer and protects against endothelial cell detachment after bacterial contamination**. Clin Hemorheol Microcirc. 2021;78(2):151-61. doi: 10.3233/CH-201096.

Wu J, Li Z, Yuan W, Zhang Q, Liang Y, Zhang M, Qin H, Li C. **Shenfu injection improves cerebral microcirculation and reduces brain injury in a porcine model of hemorrhagic shock**. Clin Hemorheol Microcirc. 2021;78(2):175-85. doi: 10.3233/CH-211100.

Junqueira CLC, Ferreira E, Junqueira ASM, Cyrino FZGA, Maranhão PA, Kraemer-Aguiar LG, Bottino DA, de Souza MDGC, Bouskela E. **Peripheral microvascular dysfunction is also present in patients with ischemia and no obstructive coronary artery disease (INOCA).** Clin Hemorheol Microcirc. 2021;79(3):381-93. doi: 10.3233/CH-201065.

G. Hagn and colleagues received the **A.L. Copley Best Paper Prize** for their study about "**Anti-inflammatory iron chelator, DIBI, reduces leukocyte-endothelial adhesion and clinical symptoms of LPS-induced interstitial cystitis in mice**". The authors evaluated the effects of DIBI on LPS induced IC in mice. Leukocyte activation, endothelial adhesion and functional capillary density were assessed by intravital microscopy of the bladder microcirculation following a single intravesical LPS administration with or without intravesical DIBI treatment. In addition, clinical IC symptoms were also assessed through behavioral and pain threshold force measurements. Four groups of female BALB/c mice were randomized into control group, IC group without therapy, IC group with DIBI therapy and control group with DIBI therapy. LPS - introduced intravesically - induced an early ($\leq$2 h) inflammation of the bladder evidenced by leukocyte activation and adhesion to bladder capillary walls. Intravesical DIBI therapy of mice 30 min following LPS administration and assessed after 1.5 h treatment showed a significant decrease in the number of adherent leukocytes compared to interstitial cystitis (IC) animals without DIBI treatment. DIBI treated mice showed a significantly lowered increase in behavioral distress scores compared to IC mice without therapy. Untreated IC mice exhibited a significantly decreased threshold force value for evoked pain response and DIBI treatment improved the threshold pain response. The study revealed that DIBI reduced inflammatory endothelial leukocyte adhesion and key indices related to pain and suffering over those observed in untreated IC mice. Our findings suggest a potential therapeutic role for DIBI for IC treatment.

The committee sincerely wishes full success to the authors in their future research and all other authors for the next **A.L. Copley Best Paper Prize** 2022.