



International  
Complement Society

*“Complement...we are  
the Experts!”<sup>TM</sup>”*

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**Focus on  
Complement**

*Issue #60*

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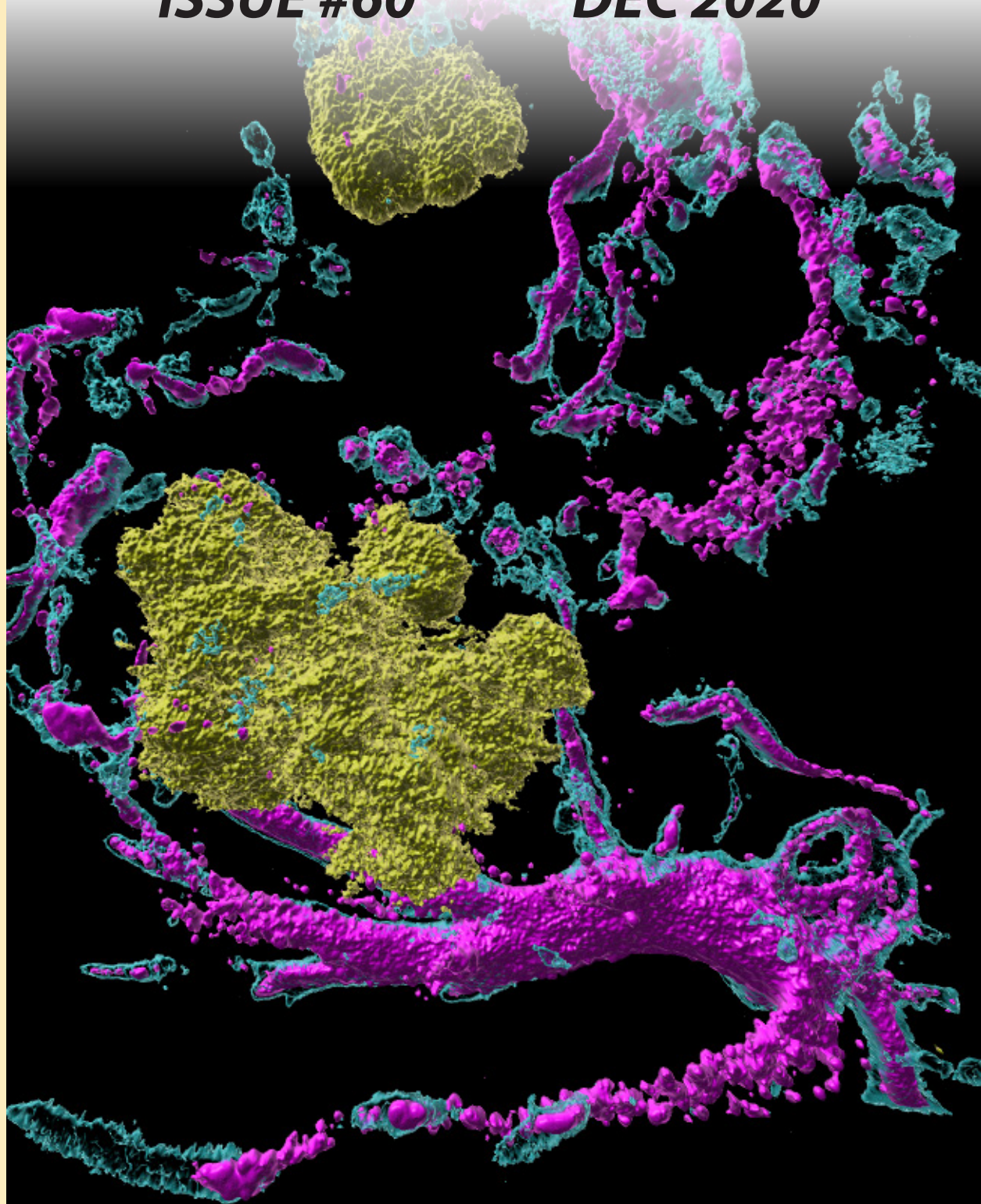
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Michael Kirschfink

# Focus on Complement

**ISSUE #60**

**DEC 2020**



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Dear Readers,

As we draw this eventful year to a close, I welcome you all to enjoy the 60th edition of *Focus on Complement* – the official newsletter of the International Complement Society (ICS).

In this issue, our ICS President, Professor Peter Garred, will provide an overview of the ICS activities in 2020, and announce upcoming events in 2021. We also congratulate Dr. Angela Gomez-Arboledas who is the winner of the FoC Early Career Cover Image Award. A description of her research and cover image can be found on the next page.

We feature the research groups of Professor Ming-hui Zhao from China, Professor Anna Blom from Sweden, and Dr. Péter Gál from Hungary. Issue contributor Professor Michael Kirschfink reviews two articles that examine complement, neutrophils and COVID-19, and guidelines for complement genetics in hereditary angioedema.

I hope you all enjoy this final issue of *Focus on Complement* for 2020, and wish everyone a safe, healthy and happy end of year.

Professor Trent Woodruff  
Editor, FoC  
Secretary, ICS

### **Connect with the ICS**

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If you would like to contribute with an article to a future issue or have suggestions for a subject theme, please contact Trent Woodruff ([t.woodruff@uq.edu.au](mailto:t.woodruff@uq.edu.au)) or Peter Garred ([Peter.Garred@regionh.dk](mailto:Peter.Garred@regionh.dk)).

Plus visit our website and follow us on Twitter to keep updated with the latest ICS and complement news.

 [www.complement.org](http://www.complement.org)

 [@complementsoc](https://twitter.com/complementsoc)

### Angela Gomez-Arboledas: Winner of the Focus on Complement Early Career Cover Image Award



Angela Gomez-Arboledas, is a postdoctoral scholar in the laboratory led by Dr. Andrea Tenner, at University of California, Irvine. Angela's research has always been focused on Alzheimer's disease (AD), and she has recently joined the complement field, investigating the role of the complement system in Alzheimer's disease using multiple AD mouse models. She is focused on unravelling the connection between C5a-C5aR1 signaling, glial cells and Alzheimer's pathogenesis, with the hope of identifying therapeutic targets for treating this devastating disease.

*Cover Image Description:* 3D reconstruction of an immunofluorescence image that shows C3+ (magenta) astrocytes (GFAP+, cyan) polarized towards an amyloid plaque (ThioS+, yellow) in the Tg2576 mouse model of Alzheimer's disease treated with a C5aR1 antagonist (PMX205). Genetic ablation or pharmacological inhibition of C5aR1 signaling in mouse models of Alzheimer's disease showed a strong reduction of A1 neurotoxic astrocytes (C3+). Fibrillar amyloid plaques and A $\beta$  fibrillary oligomers were reduced by about half and A1 neurotoxic astrocytes in the vicinity of the plaques were reduced by ~ 70% in the hippocampus in Tg2576 mice treated with PMX205. However, when a large amyloid plaque was present in these mice (such as the one in the image), A1 reactive astrocytes (C3+ and GFAP+) had their processes completely polarized towards the amyloid deposit.

The **Early Career Cover Image Award**. Each Issue the ICS board will select a scientific image to highlight on the front cover of FoC. The winning image will include a brief description of the image, and a profile of the winner within the newsletter.

**Eligibility:** graduate students, post-doctoral staff, and early career researchers (generally, but not exclusively under 40 years of age) are eligible to apply.

Interested applicants should email the FoC Editor ([t.woodruff@uq.edu.au](mailto:t.woodruff@uq.edu.au)) at least 2 weeks prior to each issue production date (1st March, 1st June, 1st September, 1st December), with one suggested image of their research. Images could include immunochemistry (tissues, cells, etc.), pathology, structures, or any other image of relevance to complement research. All images should not have any copyright that would be infringed if published in FoC (for example work already published in a journal). Submissions should also include a brief profile of the researcher and a description of the image (~100 words each).

Winners of the Award will receive a \$50 Amazon gift card, and a signed certificate from the ICS.

# President Address

Dear Colleagues, it has been a challenging year and despite the setbacks due to COVID, we have managed to move forward with ICS projects that inform and keep the members engaged during quarantine and travel restrictions.

To recap:

**ICW 2020 Berlin** – we were able to postpone the meeting and move it to 2021 without experiencing any financial loss. A special thank you to Peter Zipfel and Christine Skerka, Co-Chairs, who have worked tirelessly to re-organize and find new dates and venue for the 2021 workshop. More information about this to be announced in early 2021.

**ICS Pioneer Women** – As representatives of the complement community, it was our honor to award the first ‘Pioneering Women in Complement Research Awards’ to Professors Patricia Creveling Giclas, Irma Gigli, and the late Jarmila Janatova.

These awards were initiated to honor retired female members of the complement community for their major and long-lasting scientific impact on our research field – and to showcase key female complementologists as role models for the younger female scientists among us.

**ICS C3 Symposium** – Complement, Clotting and COVID-19 on October 28, 2020 – we had a fantastic turnout of 600+ people from around the globe on this timely webinar and I want to thank the speakers Steven Holland, Daniel Ricklin, Ben Afzali, Tom Mollnes, Marina Noris, Eric Vivier and Claudia Kemper for moderating the symposium.

**ICS Symposium at AAI** (The American Association of Immunologists) Meeting 2021 – the ICS has been sponsoring a complement symposium at the annual AAI meeting for several years thanks to Viviana Ferreira and Andrea Tenner. The AAI has been re-scheduled for May 5-10, 2021 and will be fully online and virtual. If you are planning to attend AAI 2021, mark your calendar for May (exact date and time to be announced) to participate in this outstanding session.

**ICS 2021 Elections** – Would you like to join the leadership of ICS and help shape the direction of the society? Nominations for Officers and Council will open April 30. Each nomination must have 5 members on the petition for consideration. Nomination forms will be available on-line at the ICS website in April 2021.

I want to thank everyone for their support as we continue to navigate in the COVID storm, which hopefully will come to an end in 2021.

With best wishes for a peaceful Christmas and a Happy New Year to all of you.

Peter Garred  
President of ICS

## Complement Research at Beijing, China

### *The group of Professor Ming-hui Zhao*

Our research group, led by Professor Ming-hui Zhao, represents the hub of research in complement-associated kidney diseases in China. We focus on the understanding of complement activation in the pathogenesis of various glomerulonephritis diseases since the kidney is the most vulnerable organ in abnormal complement activation.

In thrombotic microangiopathy, we identified the genetic variants and autoimmunity of complement components (such as CFH, CFI) and coagulation (vWF) in patients with aHUS, postpartum HUS and malignant hypertension. Anti-CFH antibodies could affect both N- and C-terminal domains of CFH, leading to enhanced complement activation in both circulation and on host cells.

In lupus nephritis, we found autoantibodies against C1q, C1q-A08 (the fine epitope of C1q) and CFH are associated with disease activity. We further revealed the pathophysiological effects of autoimmunity on the regulation of complement activation. Moreover, autoantibodies against monomeric or modified CRP (mCRP) were proved to influence complement activation and clinical course in lupus nephritis; the interactions between mCRP, C1q and CFH might explain dysfunctions of complement.

In ANCA-associated vasculitis, we demonstrated that activation of the alternative complement pathway played a key role in the development of the disease. Further study found that stimulation of neutrophils with C5a and ANCA induced the neutrophil respiratory burst and degranulation, and also activated the coagulation system and generated thrombin. In other glomerular diseases, including diabetic nephropathy, membranous nephropathy and focal segmental glomerulosclerosis, we detected both serum and urinary complement activation profiles, in order to identify intervention/therapeutic targets and biomarkers to predict disease activity and disease progression.

Nowadays, our lab is a national reference laboratory for complement diagnostics, in particular in aHUS and C3G on protein levels. Our complement diagnostic packages include complement protein analyses (e.g. CFH, CFI), functional complement assays, C3NeF and autoantibodies against CFH, CFB and C3b, etc. We are always looking forward to collaborating nationally and internationally and more than happy to share resources and hear from anyone interested in our research!



Minghui Zhao



Min Chen



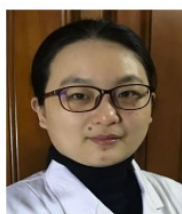
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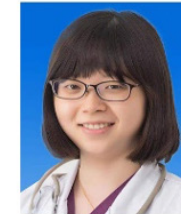
Dongyuan Chang



Zhiying Li



Jing Huang



Meng Tan

**Contact:** Dr. Ming-hui Zhao, Renal Division, Department of Medicine, Peking University First Hospital; Institute of Nephrology, Peking University; Beijing PR. China. [mhzhao@bjmu.edu.cn](mailto:mhzhao@bjmu.edu.cn)

### Complement research in Malmö, Sweden

*The group of Professor Anna Blom*

The Complement research group in Malmö has been led for 20 years by Prof Anna Blom, supported by excellent students and postdoctoral fellows, and many collaborators. The group is connected both to the hospital unit of Clinical Chemistry and to the Lund University Diabetes Centre, situated at the Clinical Research Centre at the Skåne University Hospital in Malmö.

Initially, we characterized relationships between structure and functions for several complement inhibitors, for example C4b-binding protein and factor I. We also showed how mutations in these inhibitors impair their function and thus may lead to diseases such as age-related macular degeneration or hemolytic uremic syndrome. This is now of importance for selection of patients for treatment with emerging complement inhibitors for clinical use.

As a main antimicrobial defence, complement is a major target for evasion strategies developed by pathogens. We showed that many successful human pathogens, both bacterial and viral, use cunning strategies to avoid killing by complement and thereby establish infections. One of the mechanisms studied in detail is capturing of human complement inhibitors by bacterial pathogens such as streptococci. Using a novel transgenic mouse model developed by our long-term collaborator Sanjay Ram we proved the *in vivo* importance of these interactions and performed detailed molecular studies of the interactions involved. We also identified several other strategies such as expression of viral complement inhibitors homologous to human counterparts (such as KCP and RCP from Kaposi's sarcoma associated herpesvirus and rhesus rhadinovirus, respectively) or expression of bacterial proteins targeting the central component C3. All these findings have practical implications for vaccine development.

Complement is also crucial for removal of apoptotic cells, and malfunctions of this system lead to systemic lupus erythematosus (SLE). We detailed molecular interactions underlying complement recognition of apoptotic cells and how it is kept under control on these cells to prevent excessive inflammation. We discovered that complement inhibitor factor H is actively internalized by apoptotic cells, upon which it not only allows opsonisation of these dying cells for phagocytosis but also neutralizes pro-inflammatory effects of nucleosomes. We also showed that a large proportion of SLE patients are not able to degrade neutrophil extracellular traps (NETs), which activate complement leading to antibody production against NET components such as DNA and very high disease activity. Further, we created novel antibodies specific for detection of C4d and found that measurement of C4d in plasma of SLE patients is a good disease activity marker, in particular for nephritis.

More recently, we have been investigating roles of complement in the pancreatic islet and diabetes development, and described a role for C4BP in limiting islet amyloid-induced inflammation. We also discovered an unexpected novel function for an old complement inhibitor, CD59 – this time in insulin secretion from beta cells. We showed that this is due to interactions of CD59 with SNARE protein complexes involved in vesicular transport, and we are now characterizing isoforms of CD59 regulating insulin secretion.

### Complement research in Malmö, Sweden

*The group of Professor Anna Blom*

In continuation, the most significant publication from the group described a novel role for cytoplasmic C3 in autophagy of pancreatic beta cells. These findings, together with work from other groups, is opening a new field of studies into intracellular roles of complement, which is otherwise considered a blood component.

Further, we identified new human complement regulators such as SUSD4, COMP and CSMD1, which we also showed are involved in development of breast cancer.

Taken together, we study molecular mechanisms underlying the contribution of complement to pathogenesis of various diseases, with the aim of developing biomarkers and targets for potential therapeutic manipulation.



**Group Members:** Izabela Bednarska, Katarzyna Wozniak, Frida Mohlin, Sara Nilsson, Rebecca Trattner Evetovics, Ewelina Golec, Karolina Smolag-Klosowska, Chrysostomi Gialeli, Lucie Colineau, Damián Bierschenk, Alexander Ekström, Estefania Torres-Vega, Klaudia Kulak, Karolina Maziarz, Konstantinos Papadakos, Anna Blom, Leonie Vogt, Ben King, Myriam Martin, Alicja Nowacka, Serena Bettoni

**Contact:** Professor Anna Blom, [anna.blom@med.lu.se](mailto:anna.blom@med.lu.se)

## Complement Research in Budapest, Hungary

### *The group of Dr. Péter Gál*

The complement research group led by Dr. Péter Gál is located at the Institute of Enzymology of Research Centre for Natural Sciences, in Budapest, Hungary. The research group was founded by Prof. Péter Závodszky in the 1980s after his return from Prof. Rodney Porter's laboratory in Oxford. The main focus of the research group is to understand the mechanism of complement activation at molecular level, and specifically to clarify the exact roles of the early complement proteases (C1r, C1s, MASP-1, MASP-2 and MASP-3) in physiological and pathological processes. The group pioneered the recombinant expression and structure determination of complement proteases. In collaboration with Veronika Harmat at the Institute of Chemistry, Eötvös Loránd University (ELTE), Budapest, they crystallized and determined the structure of the catalytic fragment of C1r, MASP-1, MASP-2 and that of the serpin domain of C1-inhibitor. They proposed a mechanism for the autoactivation of the early complement proteases. The substrate specificity and physiological role of MASP-1 have been interesting research areas in the recent years. The Budapest group showed that MASP-1 has a broad substrate specificity by cleaving not only complement components but also the components of the blood coagulation system, high molecular kininogen and protease-activated receptor 4 (PAR4). In this way MASP-1 can modulate blood coagulation, liberate bradykinin from high molecular weight kininogen and activate endothelial cells. The relaxed substrate specificity of MASP-1 was also supported by the crystal structure of the protease domain showing a broad substrate binding groove compared to MASP-2 and C1r.

In the recent years, the group established a fruitful collaboration with the research group of Gábor Pál at the Department of Biochemistry, ELTE in Budapest. The primary research goals were to develop selective inhibitors against the complement proteases and to use these inhibitors for the clarification of the exact mechanism of each complement activation pathway. They made the first MASP-1- and MASP-2-specific inhibitors. Both inhibitors completely blocked the lectin pathway activation. Further experiments proved that MASP-1 is the exclusive activator of MASP-2 in normal human serum. Although purified, concentrated MASP-2 can autoactivate slowly in vitro, this phenomenon does not manifest in the blood where the concentration of MASP-1 far exceeds that of MASP-2. Besides activating MASP-2, MASP-1 also significantly contributes to the generation of C3 convertase (C4b2b) by cleaving the majority of C2. The researchers also developed the first MASP-3 selective inhibitor, but surprisingly it showed no effect on the lectin pathway activation in any experimental setting.

Another long-standing interest is the mechanism of alternative pathway activation. Using a human plasma-based model system and the MASP-3 selective inhibitor, they unambiguously proved that MASP-3 is responsible for activating pro-factor D in resting human blood. Although activated MASP-1, MASP-2 and thrombin efficiently activate pro-factor D, they are not present in resting blood where neither complement activation nor blood coagulation are running. MASP-3, however, is present mainly in its active form in normal human plasma ensuring the cleavage of pro-factor D even before the appearance of any PAMPs or DAMPs in the circulation. The activation status of MASP-3 raises another intriguing question; how zymogen MASP-3 is activated in the resting blood. Answering this question is one of the top research priorities of the group.

## Complement Research in Budapest, Hungary

*The group of Dr. Péter Gál*

Most recently, another interesting link between the lectin and alternative pathway has been discovered. The research group showed that MASP-1 significantly contributes to the activation of the alternative pathway on certain activation surfaces (e.g. bacterial LPS). Since LPS is the component of the outer membrane of Gram-negative bacteria, MASP-1 could have multiple roles in the defense against bacterial infection. The molecular mechanism of this phenomenon is under intensive investigation in the laboratory.

The specific inhibitors, developed against the individual proteases of the complement cascade, besides being useful research tools could also serve as lead molecules in drug development aiming at treatment complement-related diseases.



**Group:** Seen in the photo are left to right: József Dobó (Senior Research Fellow), Júlia Balczer (Technician), Ráhel Dani (PhD student), Andera Kocsis (Post doc), Dóra Varga (Masters student), Barbara Végh (Post doc), Péter Kacz (Masters student), Péter Gál (Group leader), Bence Farkas (Masters student). Not seen are: Péter Závodszy (Professor Emeritus), Judit Pataki (Technician).

**Contact:** Dr. Péter Gál, [gal.peter@ttk.mta.hu](mailto:gal.peter@ttk.mta.hu)

### **Complement and tissue factor-enriched neutrophil extracellular traps are key drivers in COVID-19 immunothrombosis**

Skendros P, Mitsios A, Chrysanthopoulou A, Mastellos DC, Metallidis S, Rafailidis P, Ntinopoulou M, Sertaridou E, Tsironidou V, Tsigalou C, Tektonidou M, Konstantinidis T, Papagoras C, Mitroulis I, Germanidis G, Lambris JD, Ritis K

**[Journal of Clinical Investigation](#); 2020 Nov 2;130(11):6151-6157.**

During SARS-CoV-2 infection a suboptimal or unrestrained immune response with the innate immune system as the first line of defence against viral infections, drives the clinical patterns, disease severity and progression of COVID-19. In SARS-CoV-2 infection a pronounced neutrophil infiltration is observed and neutrophilia appears to be predictive of poor outcomes in patients with COVID-19. Neutrophil activation and the formation of neutrophil extracellular traps (NETs) may have a prominent role in propagating the severe cytokine release observed in severe cases of COVID-19. Prior reports linked excessive NET formation with a cascade of inflammatory reactions that promotes cancer cell metastasis, destroys surrounding tissues, facilitates microthrombosis, and results in permanent organ damage to the pulmonary, cardiovascular, and renal systems. Thus, targeting NETs directly or indirectly may reduce the clinical severity of COVID-19. With respect to the well known crosstalk between complement and neutrophils Skendros and colleagues hypothesized that collaboration of these key components of the innate immune systems may also mediate early events leading to coagulopathy in COVID-19. In COVID-19 patients they found elevated levels of myeloperoxidase/DNA complexes as surrogate markers of NETosis which correlated with thrombin-antithrombin (TAT) activity. PMN tissue factor (TF) expression was increased and TF-bearing NETs were spontaneously formed. This went along with a pronounced complement activation as indicated by increased plasma levels of C5a and sC5b-9. Treatment of control neutrophils with COVID-19 platelet-rich plasma generated TF-bearing NETs that induced thrombotic activity of human aortic endothelial cells. Considering C5a as a key mediator of neutrophil TF expression C5a receptor 1 (C5aR1) inhibition not only successfully attenuated TF expression in platelet rich plasma (PRP) stimulated neutrophils, but also inhibited NET release. TF expression in control PMNs was also efficiently blocked upon inhibition of C3, the central complement component at the merging point of all three pathways, by the compstatin analogue Cp40 in a coinubation experiment with normal and COVID-19 patients' sera. With their findings, that complement activation potentiates the platelet/NETs/TF/thrombin axis during SARS-CoV-2 infection, Skendros et al. provide a mechanistic basis for a pivotal role of complement and NETs in COVID-19 immunothrombosis. Targeting complement activation or/and NET formation as therapeutic strategies may disrupt the vicious cycle of COVID-19 immunothrombosis. This is supported by recent promising clinical data from the compassionate use of the C3-targeted therapeutic AMY-101 (Cp40-based drug candidate, clinically developed by Amyndas Pharmaceuticals) in severely ill patients with COVID-19 suffering from acute respiratory distress syndrome.

### **International Consensus on the Use of Genetics in the Management of Hereditary Angioedema**

Germenis AE, Margaglione M, Pesquero JB, Farkas H, Cichon S, Csuka D, Lera AL, Rijavec M, Jolles S, Szilagyi A, Trascasa ML, Veronez CL, Drouet C, Zamanakou M; Hereditary Angioedema International Working Group

**[Journal of Allergy and Clinical Immunology in Practice](#); 2020, 8(3):901-911**

Hereditary angioedema (HAE) is a rare disease that manifests with cutaneous and/or submucosal swellings due to uncontrolled activation of the contact/kinin system. Attacks recur with unpredictable frequency and severity, laryngeal edema is potentially lethal, and the disease burden may severely disrupt patients' lives. Despite the fact that HAE is not a complement-mediated disorder, historically its biochemical and molecular analysis is still often performed in complement labs. Progress in understanding the genetic and biochemical changes in HAE has driven a continuous series of improvements in the classification and treatment of HAE. Whereas the diagnosis of HAE with C1INH deficiency (type I or type II) is readily made with standard testing, substantial challenges still exist for diagnosing angioedema patients presenting with normal C1-inhibitor (HAE-nl-C1INH). Multiple guidelines have been provided regarding the classification, diagnosis, on-demand treatment, prophylactic treatment, special considerations for women and children, development of a comprehensive management and monitoring plan, and assessment of burden of illness for both HAE with or without C1 inhibitor deficiency. With some exceptions genetics is considered not to be required in C1-INH HAE, but novel sequence variants for the Serpin1 gene but also for an increasing number of genes that are associated with HAE-nl-C1INH, such as F12 (FXII-HAE), angiopoietin (ANGPT1-HAE), plasminogen (PLG-HAE) and kininogen1 (KNG1-HAE) make genetic testing indispensable. Considering the increasing complexity of the genetic background of HAE, and lack of standardization and validation of various techniques of genotyping the Hereditary Angioedema International Working Group now presents a consensus paper, based on a meeting of experts in HAE genetics in 2018. Aiming at to define a framework which aids clinicians as well as geneticists, eleven consensus statements were generated, encompassing considerations regarding the clinical indications for genotyping patients with angioedema, the methods of detection of HAE-causative variants, the variant pathogenicity curation, the genotyping of patients with HAE in the clinic, and genetic counseling. Further uncovering the genetic basis of HAE is expected not only to facilitate a better understanding of disease pathophysiology that could drive the discovery of new therapeutic targets but also to provide useful indicators for the clinical management of the disease.

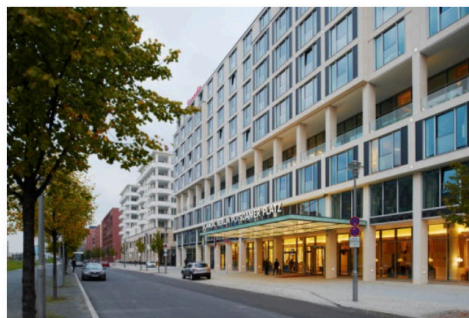
### **A CALL FOR PARTICIPANTS: THE ICS /IUIS COMMITTEE FOR THE STANDARDIZATION AND QUALITY ASSESSMENT OF COMPLEMENT MEASUREMENTS**

We invite and encourage colleagues who are actively involved in complement diagnostics and who would like to make a contribution to the work of the committee to send a short statement of interest (max 1 A4 page) introducing themselves and their work in complement diagnostics to either Prof. Kirschfink ([kirschfink@uni-hd.de](mailto:kirschfink@uni-hd.de)) or Prof. Prohászka ([prohaszka.zoltan@med.semmelweis-univ.hu](mailto:prohaszka.zoltan@med.semmelweis-univ.hu)).

The committee was founded in 2008 during the International Complement Workshop in Basel, Switzerland and is dedicated to standardization and harmonization of diagnostic complement tests among the laboratories. More informations are on the ICS (<https://www.complement.org/committee>) and IUIS (<https://iuis.org/committees/qas/>) websites. A more recent overview of the committee's activities has been given in the June issue of FoC of this year.

### 28th International Complement Workshop ICW 2021 Berlin, Germany

*September 18 – 23, 2021*



**Venue: Scandic Hotel,  
Potsdamer Platz, Berlin**



Dear Complementologists, Dear Colleagues,

Here we want to give you an update on the ICW 2021 in Berlin. In accordance with the president, Peter Garred and the executive Director Sheila Jewart of the ICS and with the help of Berrit Hedegaard from Meeting Planners, a new date and a new venue has been selected for the 28th International Complement Meeting.

It is now scheduled for the 18th to 23rd of September 2021 in Berlin, Scandic Hotel.

Please mark the date, and the latest information on abstract deadlines, hotels and housing on the new website:

<http://www.icw2021berlin.de/loc.html>

We very much hope that the vaccine situation will improve travel and that we all can move and travel around the world. However, in case those restrictions will continue we have the option of a hybrid meeting, where everyone can present her or his latest results in our field and can present talks and posters also in a virtual environment.

So far we wish you all a nice holiday season and a successful and healthy new year and we very much hope that we will meet in person in September 2021 in Berlin.

Best regards,  
Peter Zipfel and Christine Skerka Chairpersons of the ICW 2021 Berlin

### 13<sup>th</sup> International Conference on Complement Therapeutics

The field of complement-targeted drug discovery has experienced a profound transformation during the past decade. With the first complement-specific drugs on the market, clinical experience is gained and novel indications are being explored. At the same time, efforts in both academic and pharmaceutical research have produced new innovative therapeutic concepts and drug leads that interfere at different levels of the complement cascade; many of these candidates are currently undergoing clinical evaluation. Finally, genetic and molecular studies continue to reveal contributions of complement in both orphan and highly prevalent diseases. Apart from offering new hope for patients suffering from such diseases, the study of complement pathways, mutations, and deficiencies also teaches us important lessons about the role of complement in health and disease and allows us to refine our models and tools for applied and basic research. This conference aims to bring together academic and industry scientists and clinical development experts who are focused on contemporary and emerging aspects of complement-mediated disease pathogenesis and the development of therapeutics that modulate this system in a beneficial manner.

Topics discussed during the [conference](#) include: Molecular mechanisms and targets in complement-related diseases; Novel inhibitors & pipeline compounds; Hematological disorders; Organ & cell transplantation, I/R injury and chronic rejection; Kidney diseases; Neurological & ocular diseases; Acute and chronic inflammatory disorders; Infectious diseases & sepsis; Cancer; Informative complement biomarkers in therapeutic development; Novel and unexpected indications.



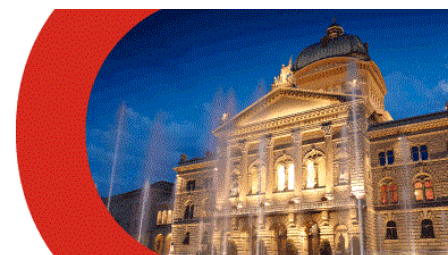
#### Organizing Committee:

John Lambris, PhD  
Dimitrios Mastellos, PhD  
Daniel Ricklin, PhD  
Antonio Risitano, MD  
Lubka Roumenina, MD

**17th - 22nd June 2021**  
**Ioannina, Greece**



**18th European Meeting  
on Complement in Human Disease**  
**Bern Switzerland • 25.-29.08.2022**



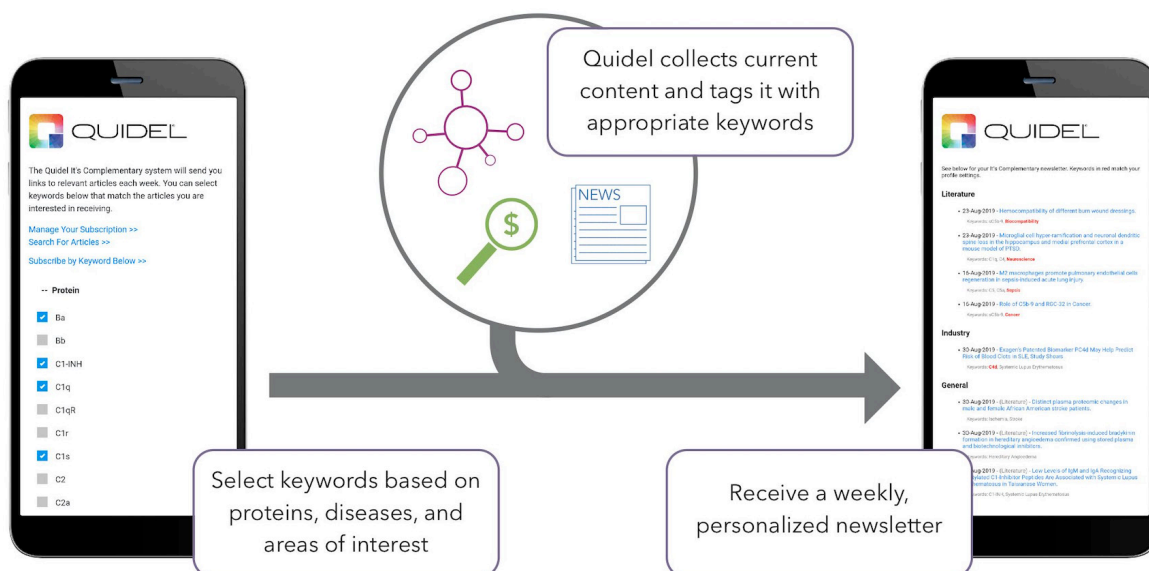


### *It's Complementary* A Personalized Complement System Newsletter

As complement system research expands, keeping up to date on the latest literature, news, and funding is difficult. Quidel Specialty Products Group (SPG) has leveraged the information it tracks to create a new tool for complement scientists: It's Complementary – a personalized newsletter that highlights the interest of each individual researcher.

How It's Complementary works:

- **Build Your Profile:** Select keywords for complement proteins and Diseases/Areas of Interest that match your research interests.
- **Subscribe:** Submit your contact information and confirm your subscription via email.
- **Relax and Enjoy:** Every Tuesday (8am PST) the It's Complementary newsletter arrives with new complement information collected over the last week, with the content such as literature, news, and funding categorized to match your profile.



Each week SPG personnel collect complement system information and tag it with the appropriate keywords. The system matches content and subscribers, building a personalized newsletter that gives researchers the information they need. In addition, use the “Search For Articles” to browse the extensive database.

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