



**International
Complement Society**

*“Complement...we are
the Experts!”™*

9037 Ron Den Lane
Windermere
FL 34786-8328
T: 407-234-3274

ics@complement.org
www.complement.org

President

Peter Garred

Past President

Michael Holers

President-Elect

Claudia Kemper

Treasurer

Daniel Ricklin

Secretary

Trent Woodruff

Councillors

Viviana Ferreira

Claire Harris

Michael Kirschfink

Zoltán Prohászka

Joshua Thurman

Leendert Trouw

Nobutaka Wakamiya

Peter F. Zipfel

Executive Director

Sheilah Jewart

**Focus on
Complement**

Issue #62

Editor

Trent Woodruff

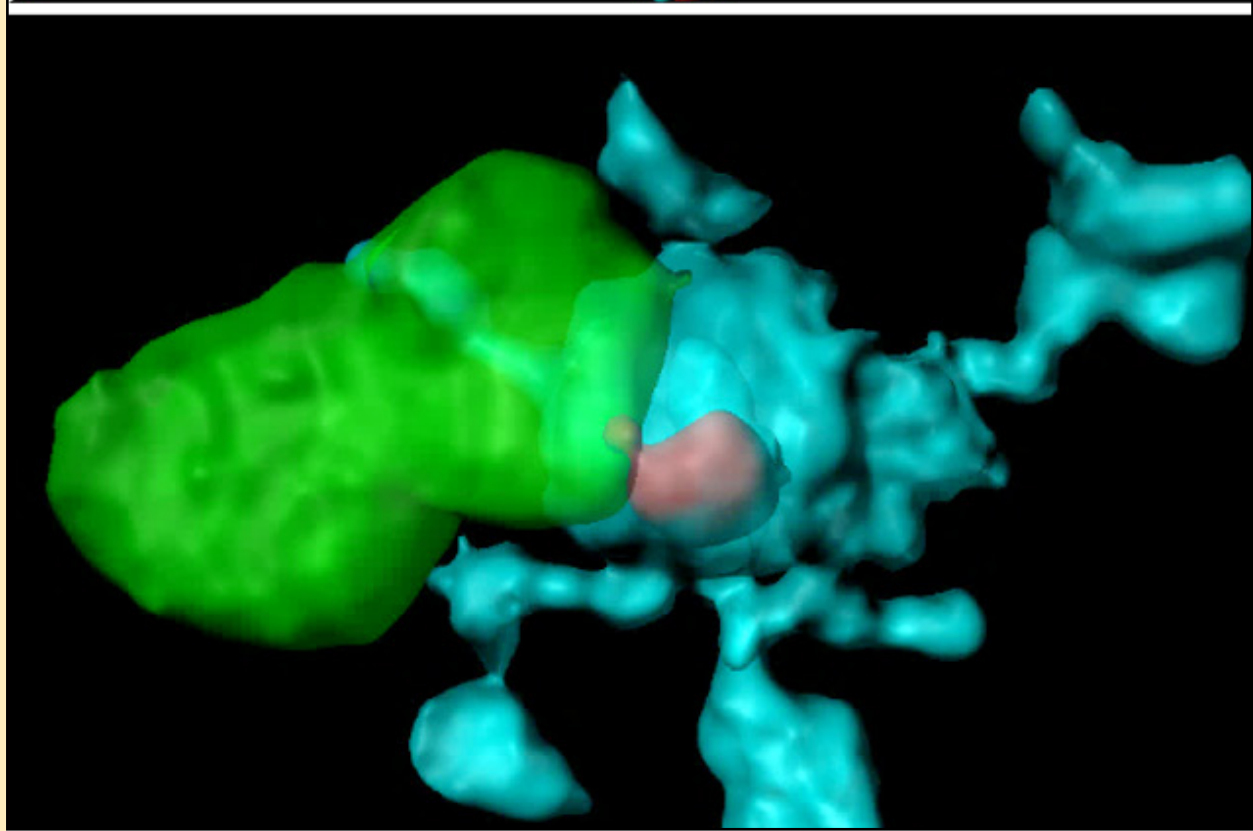
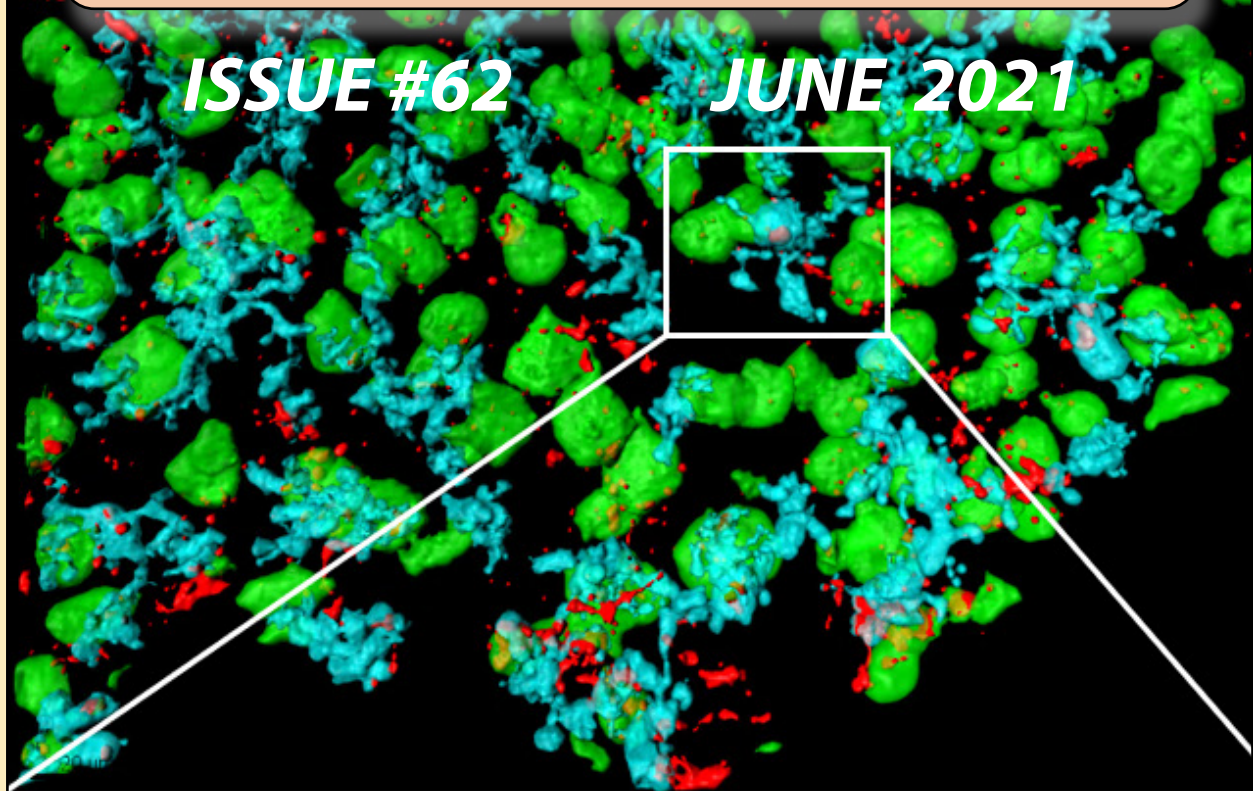
Issue Contributor

Leendert Trouw

Focus on Complement

ISSUE #62

JUNE 2021



In this 62nd Issue of Focus on Complement:

- [3](#) Editor's Message
- [4](#) FoC Early Career Investigator Cover Image Award
- [5](#) President's Message
- [6](#) Spotlight on Complement Teams From Around the World
- [10](#) Complement Research Reviews
- [12](#) Meeting Summaries
- [15](#) Meeting Announcements
- [19](#) Sponsor Advertisements



Dear Readers,

I welcome you all to the 62nd edition of *Focus on Complement* – the official newsletter of the International Complement Society (ICS).

In this issue, our ICS President, Professor Peter Garred, provides an overview of recent ICS activities. We congratulate Dr. Khalil Mallah who is the winner of the FoC Early Career Cover Image Award. A description of Khalil's research and cover image can be found on the next page.

We feature several research groups from Trieste, Italy, and Aarhus University in Denmark. Issue contributor Dr. Leendert Trouw reviews two articles that examine a non-traditional complement activation pathway, and mechanisms of CFHR5-mediated nephropathy.

Finally, we highlight a number of complement focussed conferences, including a summary from the ICS Guest Symposium at the recent AAI annual meeting.

I hope you all enjoy this second issue of *Focus on Complement* for 2021.

Professor Trent Woodruff
Editor, FoC
Secretary, ICS

Connect with the ICS

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) or Peter Garred (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

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

Khalil Mallah: Winner of the Focus on Complement Early Career Cover Image Award



Khalil Mallah, PhD, is a postdoctoral research fellow in the lab of Dr. Stephen Tomlinson at the Medical University of South Carolina. Khalil joined the lab of Dr. Tomlinson in February 2019 after completing his PhD in Lille University (France) while studying the spatiotemporal proteomic and lipid changes that occur in the brain after Traumatic Brain Injury (TBI). In the lab of Dr. Tomlinson, Khalil is investigating the role of complement in chronic neurodegenerative sequelae of TBI and stroke.

Cover Image Description: Confocal image of the perilesional cortical area at 6 months after a single hit TBI. The upper panel of the image shows ongoing C3 (red) deposition on neurons (green) that are surrounded by microglia (aqua). Zoomed image shows a C3 tagged neuron partially engulfed within a microglia. Data indicates an aberrant complement-dependent pruning mechanism is occurring at 6 months after TBI.

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) 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

Message from the ICS President

As you know by now, the ICS made the decision to move the ICW Berlin meeting to a virtual platform to be held December 06-10, 2021. We now have the organization in place and a fantastic meeting developing with poster awards and interesting talks. Registration is open and we invite you to submit your abstract and plan to attend. Even though we won't be in-person, you will have opportunities to interact socially with friends and colleagues throughout the meeting. If possible we also encourage you to meet locally following the online meeting to share experiences offline.

ICS Elections - It is time to remind all ICS members to send nominations to replace those ICS Board members whose term of service will have expired in 2021. The ICS Council is dedicated to identifying and recommending outstanding ICS members as candidates for election to ICS leadership positions, taking in to account the diversity reflected in members' scientific expertise and experience, research interests, geography, institution, ethnicity, and gender. ICS strives to provide an inclusive organization that is committed to diverse and equitable practices.

Positions open: President-Elect, Treasurer, Secretary, Councilors (2) - 6-yr terms

For information and to submit nominations by the August 06 deadline, go to this link: <https://www.complement.org/nomination-form>

Nomenclature -The ICS and ECN-endorsed, and International Union of Immunological Societies (IUIS) pre-approved, latest update on the complement nomenclature is summarized in this publication (<https://www.frontiersin.org/articles/10.3389/fimmu.2019.01308/full>). At a special meeting of the ICS Council in May 2021, all present members voted unanimously to finalize procedures with the IUIS Nomenclature Committee to officially adopt the ICS recommendations and publish them on the IUIS webpage.

As we all appreciate, the complement system is complex – as is its nomenclature. We are seeing a substantial increase in interest in complement biology now also from researchers and clinicians from 'outside' our core field. A simplified and unified complement nomenclature will facilitate the dialogue with these new colleagues and research fields.

ICS Complement and Inflammation Webinar June 30: Organizers Leendert Trouw, Josh Thurman and Trent Woodruff have put together a fantastic program for ICS members and complement community. Speakers and registration information can be found on the announcement in this FoC.

ICW 2025 Call for Bids: Where will we meet in 2025? If you are interested in hosting an ICW, please submit your letter of intent by March 01, 2022. A full proposal for the 30th Complement Workshop must be submitted by August 01, 2023, in order for the ICS Council to select the final site.

Now we see the light at the end of the tunnel and hope to see each other soon in person. We will continue to plan important topic webinars and will also focus on our early career trainees to provide them with a platform to present their research in progress. We hope to see you in December at the ICW Virtual meeting.

Best regards,
Peter Garred, President of ICS

Complement Research at Aarhus University Denmark

The groups of Drs Thiel, Vorup-Jensen, and Andersen

At Aarhus University in Denmark, a cluster of laboratories is studying different aspects of the complement system. The teams are interested to understand the role of complement in inflammatory conditions. The clinical observation that recognition of self-tissues by complement can lead to autoimmune and inflammatory diseases is a leading principle in their studies.

In the lab of **Steffen Thiel**, they have developed several unique assays for proteins of the lectin pathway and use them for studies on autoimmune diseases such as diabetes and rheumatoid arthritis and in immunodeficiency induced by viruses, chemotherapy, or in inherited immunodeficiency. The latter has proven essential to understand the function of several complement proteins. They also described several clinically relevant polymorphisms in the complement genes associated with differential circulating levels or aberrant protein function. From the very beginning of the description of the lectin pathway as a novel initiating pathway of the complement system, researchers in Steffen Thiel's lab have been studying this ensemble of complement activating molecules. It is a constant interest of the groups in Aarhus to try to unravel the ways that the pattern recognition molecules of the lectin pathway differentiate between the body's normal proteins, and the pattern of ligands on the surface of micro-organisms, and how they are taking part in the homeostatic processes in the body via recognition of apoptotic cells. Rasmus Pihl and colleagues in Steffen Thiel's lab recently uncovered a novel mechanism of inhibition of proteases, including enzymes of the complement system, exhibited by the plasma protein Inter- α -inhibitor heavy chain 4 (ITIH4). This adds to our knowledge of the network and interrelationship between extracellular proteases and their inhibitors. In a collaboration between the labs in Aarhus, they are expanding on the limited understanding of ITIH4 by structural and biochemical studies and examinations of patient cohorts where unbalanced enzyme/inhibitor ratios may exist.

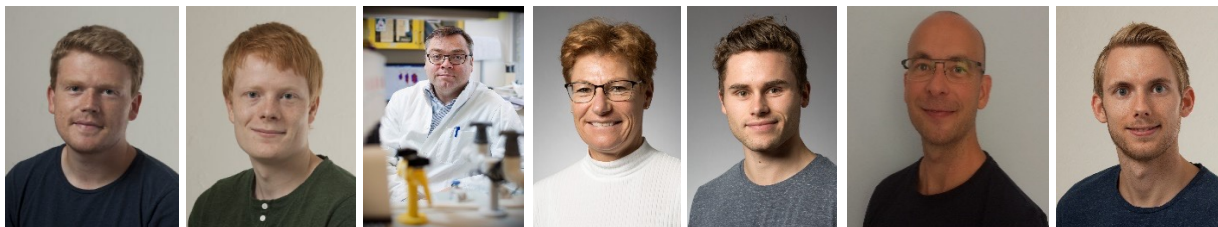
In the Biophysical Immunology lab lead by **Thomas Vorup-Jensen**, studies on MBL and IgM have linked pattern recognition in the innate immune system to fundamental thermodynamical properties of polyvalent interactions and the surface topology of microbial targets. Also, the complex ligand recognition by CR3 and CR4 is telling them about how protein structure guides functionality. During the past two decades, it has become clear how protein conformation determines the activity of these essential parts of the phagocytic machinery. There is now evidence that activated CR3 on myeloid cells may hand over complement-opsonized antigens to B lymphocytes in ways literally partnering with CR2. Also, for CR4 conformational regulation is indispensable for the activity, which furthermore is selective toward the phagocytic uptake of large particles with diameters around 600 nm. To study these phenomena, the lab uses both image stream flow cytometry as well as several tools from the nanotechnology inventory, including atomic force microscopy and nanoparticle tracking analysis.

Highlights

At the natural science faculty, **Gregers Rom Andersen** has conducted a comprehensive structural characterization of complement proteins using X-ray crystallography, small-angle X-ray scattering, and electron microscopy. Often this has involved collaborative efforts with Thiel and Vorup-Jensen. Examples include complement C3, C4, C5, FB, C2, F3, C1, MBL-MASP1/3, CR3, and ITIH4. In the past years, Andersen has, in close collaboration with Nick S. Laursen, developed a collection of complement-specific nanobodies with the aim of creating reagents for modulation of complement protein activity, diagnostics, and structural biology. The interest in therapeutic control of complement has also resulted in collaborations with industrial partners on structure determination of complement proteins bound to therapeutic molecules. Best known are the crystal structures of the C5a-spiegelmer and the C5-eculizumab complexes.

We always welcome collaborations with other research teams and industry/biotech partners, and we ask you to contact us for further discussions.

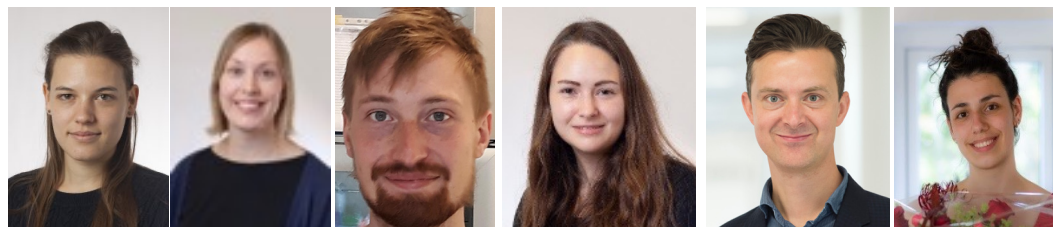
Dennis V. Pedersen
Rasmus K. Jensen
Thomas Vorup-Jensen
Bettina W. Grumsen
Kristian Juul-Madsen
Jens M.B. Jensen
Rasmus Pihl



Gregers R. Andersen
Trine A.F. Gadebjerg
Henrik Pedersen
Nick S. Laursen
Anne Trolborg
Annette G. Hansen
Steffen Thiel



Alessandra Zarantonello
Heidi G. Olsen
Martin H. Jørgensen
Josefine Lorentzen
Jakob Østergaard
Emilie Jensen



Contact: Thomas Vorup-Jensen: vorup-jensen@biomed.au.dk, Gregers Rom Andersen: gra@mbg.au.dk, Steffen Thiel: st@biomed.au.dk

Complement research in Trieste, Italy*The groups of Dr Macor and Dr Bulla*

The complement research group at the Department of Life Sciences - University of Trieste is composed of two independent but coordinated units lead by **Paolo Macor** and **Roberta Bulla**. Their activities range from diagnosis of complement deficiencies to basic research characterizing complement-mediated diseases to translational research developing new diagnostic and therapeutic approaches, moving from molecular biology studies to the characterization in animal models.

The study of the complement system in its various facets and features was founded by Francesco Tedesco, Full Professor of Immunology at the Faculty of Medicine and Surgery of the University of Trieste, awarded the European Complement Network Medal for his life time achievement in the field of complement.

The unit led by Paolo Macor is mainly focused on the development of new tailored approaches for the diagnosis and the treatment of inflammatory diseases and cancer, aiming to alternatively induce an inhibition or an enhancement of the complement activation. In the last few years, he focused on rheumatoid arthritis and antiphospholipid syndrome, as examples of inflammatory diseases, characterizing the role of the complement system in the induction of pathology. These studies allow to propose targeted recombinant antibodies and targeted nanoparticles to control its activation, demonstrating the role of the complement system in the initial phases of the diseases and the importance of its therapeutic neutralization. Similar approaches have been proposed to enhance complement activation in cancer microenvironment, in order to induce a direct killing of tumor cells or the recruitment of inflammatory cells.

The unit led by Roberta Bulla was historically focused on the study of the immunological mechanisms involved in foetal implantation in physiological and pathological pregnancy. She investigated the deposition of complement components in pathological pregnancies and she noted a strong deposition of complement components in normal human first trimester decidua. Starting from that observation, she obtained important data demonstrating a specific role played by component C1q in favoring the invasion of trophoblasts in the decidua and the remodeling of the decidual vessels. The results obtained in human placenta lead her to investigate the role of C1q in tumor growth, angiogenesis, and immune suppression. One of the recent interest of the unit is to characterize the mechanism by which C1q modulate the interaction of tumor cells with the extracellular matrix and their response to chemotherapeutic drugs. She has been investigating the interaction of complement with cells involved in host protection and inflammation with particular regard to endothelial cells obtained from normal and pathological human skin, uterus, decidua and ovary. She is collaborating with researchers and clinicians of the maternal-child hospital IRCCS Burlo Garofolo of Trieste.

Highlights

From left to right:
Mariagiulia Spazzapan,
Miriam Toffoli,
Roberta Bulla,
Chiara Agostinis,
Alessandro Mangogna,
Andrea Balduit



From left to right:
Paolo Durigutto,
Ester Desinano,
Maria Cristina Grimaldi,
Ivano Squiccimarro,
Luca De Maso,
Sara Bozzer,
Paolo Macor,
Sara Capolla

Complement inhibition at the level of C3 or C5: mechanistic reasons for ongoing terminal pathway activity

Mannes M, Dopler A, Zolk O, Lang SJ, Halbgebauer R, Höchsmann B, Skerra A, Braun CK, Huber-Lang M, Schrezenmeier H, Schmidt CQ

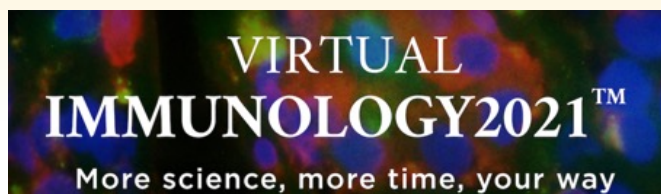
Blood; 2021 Jan 28; 137(4):443-455.

In recent years, complement inhibitors have gained increasing interest and have been successfully used in several diseases. However, residual terminal pathway activity has been described in patients treated with anti-C5 therapy. In their recent paper, Mannes et al. sought to investigate the underlying mechanisms behind this phenomenon and hypothesized that pharmacological interception leads to non-traditional complement activation. The authors show that strong activation of the classical pathway leads to C5 activation and cell lysis even when C3 is inhibited. This could be partially explained by the C3 inhibitor Cp40 not completely inhibiting C3b deposition after strong CP activation. However, classical pathway-mediated C5 activation is also observed in complete absence of C3, indicating a C3 bypass activation of C5. Indeed, dense opsonization of target surfaces with either C3b or C4b, in the absence of other complement proteins, leads to C5 priming. In this setting, membrane attack complexes are formed in the presence and, remarkably, also in the absence of biomolecular fluid-phase convertase C3bBb. This non-enzymatic activation of C5 can be explained by a conformational change of C5 to a C5b-like structure on very densely deposited C3b surfaces. Moreover, stoichiometric C5 inhibitors cannot prevent this activation, explaining the residual terminal pathway activity in patients treated with C5 inhibitors. The findings of Mannes and colleagues not only provide new mechanistic insight into C5 activation but also highlight avenues for optimization of complement inhibitors.

Gain-of-function factor H-related 5 protein impairs glomerular complement regulation resulting in kidney damage

Malik TH, Gitterman DP, Lavin DP, Lomax-Browne HJ, Hiemeyer EC, Moran LB, Boroviak K, Cook HT, Gilmore AC, Mandwie M, Ahmad A, Alexander IE, Logan GJ, Marchbank KJ, Bradley A, Pickering MC
[PNAS](#); 2021, 118(13) e2022722118

C3 glomerulopathy (C3G) is a complement-mediated kidney disease in which complement regulation is disturbed. This disease is characterized by complement deposition in the glomeruli of the kidney without deposits of immunoglobins. C3G is associated with genetic variants in the Complement Factor H-Related (CFHR) genes. The prototype C3G is CFHR5 nephropathy, in which an internal duplication of the CFHR5 gene generates FHR5 mutant proteins that lead to the accumulation of C3 within the glomeruli. To investigate how mutated CFHR5 causes C3 deposition, a CFHR5 mouse model was created. First, CRISPR/Cas9 was used to knock out the murine CFH and CFHR genes. Mice developed spontaneous C3G in the absence of both factor H (FH) and factor H related proteins (FHRs), while absence of only the FHRs did not produce an altered renal phenotype. Next, these double deficient mice were used to generate strains that transgenically co-expressed human FH and FHR5 (hFH-FHR5) or human FH and FHR5 mutant (hFH-FHR5mut). Interestingly, mice co-expressing hFH-FHR5mut developed glomerular C3 deposition, while mice co-expressing hFH-FHR5 did not. The male hFH-FHR5mut phenotype showed normal FH function, intact plasma C3 regulation, and abnormal C3 deposition in the absence of IgG, which summarizes the key features of CFHR5 nephropathy. Adeno-associated virus (AAV)-mediated expression of the FHR5mut protein in male hFH-FHR5 mice illustrates the gain-of-function effect of the mutation, as in these mice, glomerular C3 deposition developed while in the control mice it was absent. The presence of CFHR5mut is associated with renal C3 deposition despite the presence of hFH. This suggests that CFHR5mut promotes C3 deposition by impairing the ability of FH to regulate C3 deposition. To assess this, Malik et al. used AAV-mediated expression of homodimeric mini-FH, a molecule with enhanced affinity for surface C3b compared to FH. Indeed, the C3 staining intensity was reduced in homodimeric mini-FH treated hFH-FHR5mut mice. Overall, the authors created a unique model in which the animals transgenically expressing hFH and CFHR5mut develop key features of CFHR5 nephropathy. The glomerular C3 deposition could be inhibited by homodimeric mini-FH, providing future indications on how to treat CFHR-associated C3G.



Meeting report:

International Complement Society Guest Symposium at the
Annual Meeting of the American Association of Immunologists (AAI):

Complement: Alive and Kicking!

VIRTUAL. May 10-15, 2021

Viviana Ferreira, D.V.M., Ph.D., Councilor ICS and Claudia Kemper, Ph.D., President-Elect ICS
Organizers 7th International Complement Society (ICS) Guest Symposium at AAI2021

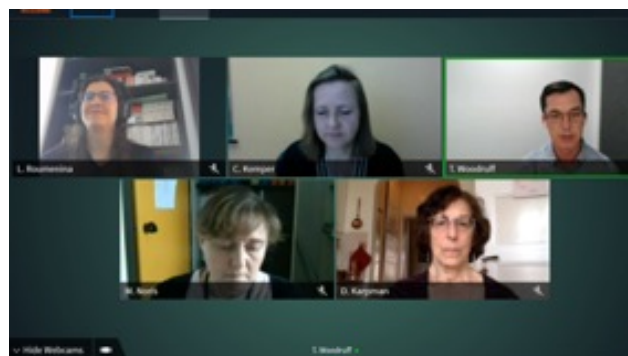
The Annual meeting of the American Association of Immunologists was held Virtually, May 10-15, 2021, after being cancelled in 2020 due to the pandemic. It was a wonderful to have these distinguished speakers represent the ICS during the 7th ICS Guest Symposium at AAI2021 by covering topics on complement and (a) neural development and degeneration, (b) cancer, (c) kidney disease, and (d) COVID-19 associated thrombosis and coagulopathy. What follows is a summary of each talk. We look forward to seeing our colleagues at the AAI ICS Guest Symposium 2022 in Portland, Oregon.

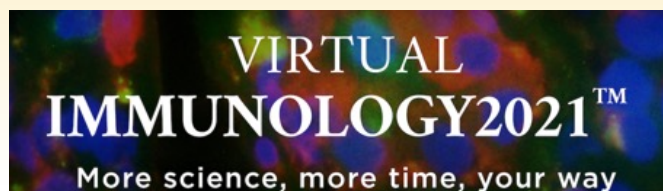
Chairs:

- Claudia Kemper, Ph.D.,
NHLBI, National Institutes of Health
- Trent Woodruff, Ph.D.,
The University of Queensland, Australia

Speakers:

Trent Woodruff, Ph.D., The University of Queensland, Australia, *From the Beginning to the End: The Complement System in Neural Development and Degeneration:* This presentation first introduced emerging roles for complement in brain development. Whilst significant attention has been focused on classical complement components, C1q and C4, and their roles in synaptic pruning, less is known about the roles of the ‘anaphylatoxins’ C3a and C5a. Data presented demonstrated essential, but opposing, physiological roles of C3a and C5a receptors during the neurogenic period of mammalian neurodevelopment. Transient inhibition of C5aR1 through in utero injection of pharmacological inhibitors to the embryonic ventricle resulted in a reduction of proliferating cells at the ventricular surface. In contrast, C3aR inhibition increased proliferation at this site. Remarkably, mice subjected to brief and transient pharmacological C5aR1 blockade during development demonstrated behavioral abnormalities that lasted into adulthood. Next data were presented highlighting a pathogenic role for C5a-C5aR1 signaling in a mouse model of amyotrophic lateral sclerosis (ALS). SOD1^{G93A} transgenic mice treated with C5aR1 inhibitor PMX205 (or C5aR1-deficient SOD1^{G93A} mice), had improved motor function and extended survival, that was paired with reductions in both microglial activation phenotypes and blood myeloid cell numbers. In ALS patients, C5aR1 expression was increased on monocytes, and higher expression was associated with respiratory decline. Together, these data demonstrate fundamental roles for complement anaphylatoxin receptors in both embryonic brain development and adult neurodegeneration.





Lubka Roumenina, Ph.D., Cordeliers Research Center, INSERM, France, *Context-Dependent Roles of Complement in Cancer*: This presentation discussed the context-dependent roles of complement in human cancers. A classification was presented of the cancers based on the prognostic impact of the co-regulated overexpression of the complement genes. This allowed to distinguish cancers, in which the overexpression of these genes confers poor (aggressive) or favorable (protective) prognosis, showing that the action of complement in tumors is context-dependent, varying according to the cancer type. Clear cell renal cell carcinoma (ccRCC) is an example of an “aggressive complement” type. Complement activation, measured by tissue or plasma C4d at the time of surgery appeared as potent negative prognostic biomarker for ccRCC progression. It was presented that surprisingly, the complement proteins are not only present as membrane deposits, but also intracellularly in the tumor cells. Intracellular C1s, C3, C4 and FH conferred poor prognosis. Exploration of the mechanism behind this prognostic impact revealed that C1s and FH have non-canonical, intracellular functions, acting in the cytoplasm and in the lysosomes respectively. Their silencing in tumor cell lines decreased proliferation and increased mortality, modifying their transcriptional program and, in the case of C1s, induced T cell activation. Based on these results it was concluded that the interplay of complement components within each cancer type is unique, governed by the properties of the tumor cells and the tumor microenvironment. The strong potential for therapeutic targeting the complement proteins in selected subgroups of cancer patients was emphasized.

Diana Karpman, Ph.D., Lund University, Sweden, *Complement-Mediated Kidney Diseases*: This presentation introduced that excess, uninhibited complement activation may lead to the kidney diseases atypical hemolytic uremic syndrome (aHUS), membranoproliferative glomerulonephritis (MPGN) and C3 glomerulopathy. The diseases have been associated with complement mutations or auto-antibodies promoting uninhibited complement activation. Data was presented indicating that excess complement activation can occur on the endothelium or on platelets in aHUS, and in the fluid phase in MPGN and C3 glomerulopathy followed by complement deposition in the glomerular basement membrane. Complement can be specifically activated in the kidney by renin, an enzyme solely released in the kidney, that cleaves C3 to C3a and C3b. A renin inhibitor, aliskiren, decreased complement activation and stabilized kidney function in C3 glomerulopathy patients. Renin levels are elevated during treatment with angiotensin-converting enzyme (ACE) inhibitors which are often used in glomerulonephritis to reduce proteinuria. Altogether, this could theoretically allow renin-mediated complement activation. A clinical trial is ongoing to compare aliskiren with ACE inhibitors in patients with C3 glomerulopathy.

Marina Noris, Ph.D. Istituto di Ricerche Farmacologiche Mario Negri-IRCCS, Italy, *Complement, endothelial injury and thrombosis in COVID-19 patients*: The novel coronavirus disease COVID-19 originates in the lungs, but may extend to other organs, causing, in severe cases, multiorgan damage. COVID-19-associated tissue injury is not primarily mediated by viral infection, but rather is a result of the inflammatory host immune response, which drives hypercytokinemia and aggressive inflammation that affect lung parenchymal cells, diminishing oxygen uptake but also endothelial cells, resulting in endotheliitis and thrombotic events and intravascular coagulation. This presentation centered on presenting how the complement system represents the first response of the immune system to SARS-CoV-2 infection, and how, in addition, there is growing evidence that unrestrained activation of complement induced by the virus in the lungs and other organs plays a major role in acute and chronic inflammation, endothelial cell dysfunction, thrombus formation and intravascular coagulation, and ultimately contributes to multiple organ failure and death. Ex vivo and in vivo data was presented that implicate the terminal pathway, and particularly the C5a/C5aR axis, in the pathogenesis of COVID-19-associated thrombosis and coagulopathy.

We would like to thank our generous sponsors:

Apellis, Complement Technology (Comptech), Hycult, Quidel, Pangburn Charitable Fund

Research Topic

Complement and COVID-19 disease

**Frontiers Immunology and ICS have joined together for a
Complement Special Research Topic**

Editors: Zoltan Prohaszka and Nicolas Stephane Merle

Deadlines:

Abstract - 30 April

Manuscript - 31 August

In this Research Topic we aim to bring together researchers from the complement field to build up a wide and comprehensive landscape of the various aspects of this system in COVID-19. We welcome the submission of Review, Original Research, Perspective, Clinical Trial and Case Report covering, but not limited to, the following sub-topics:

- (1) Our current understanding of the various causes and mechanisms leading to complement activation in COVID-19 disease;
- (2) The consequences of complement activation, and dysregulation of the complement system during COVID-19 disease;
- (3) The complex relationships between complement and coagulation-, fibrinolysis-, contact systems and how these systems are affected during COVID-19 infection;
- (4) Educative or exceptional case histories or small case-series studies focused on the complement activation and clinical outcome that may help to improve clinical care-and may support the planning of further research in this field.

For full details please visit the website:

<https://www.frontiersin.org/research-topics/19894/complement-and-covid-19->

ICS Symposium: Complement and Inflammation

Wednesday, 30 June 2021 - ZOOM - 09:00-11:30 ET (3:00-5:30 CET)

Welcome & Closing Remarks

Peter Garred, MD, PhD
President ICS



Leendert Trouw
PhD
Introduction &
Moderator



Janet So-Jung Lee
MD
University of
Pittsburgh



Jörg Köhl
PhD
University of
Lübeck



Markus
Huber-Lang
MD
ICCAI



William G. Bain
MD
University of
Pittsburgh



Gestur Vidarsson
PhD
University of
Amsterdam



Betty Diamond
MD
Feinstein Institutes
for
Medical Research

Program Co-Chairs: Leendert Trouw, Josh Thurman, Trent Woodruff

Register at: www.complement.org

ICS Symposium: Complement & Inflammation
Wednesday 30 June - 9:00-11:30 ET (3:00-5:30 CET)

No registration fees but must be registered to attend:

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



28th International Complement Virtual Workshop December 06 - 10, 2021



28th International Complement Workshop ICW 2021 Virtual Meeting

December 6 – 10, 2021

Virtual meeting: December 6-10, 2021

Abstract submission closes: August 01, 2021

Early bird fee ends: August 01, 2021

Notification of abstract acceptance: End of September, 2021

Late breaking abstract deadline: TBC

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



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



13th International Conference on Complement Therapeutics

This year marks the 100th anniversary of the Nobel Prize in Physiology or Medicine awarded to the Belgian immunologist Jules Jean Baptiste Vincent Bordet for his discoveries relating to immunity. Bordet is widely recognized as the founding father of the complement field. His pioneering observations at the turn of the 20th century provided fundamental mechanistic insight on the effector functions of complement, including its bactericidal activity, complement-mediated hemolysis, the discovery of conglutinin, the binding of complement to immune complexes and the discovery of anaphylatoxins. Moreover, Bordet's research laid the foundation for complement diagnostics by developing the widely applied complement fixation test. Commemorating his Nobel Prize and in recognition of his fundamental contributions to the complement field we are dedicating this meeting to Jules Bordet. Bordet's life and research achievements will be celebrated in a keynote lecture to be delivered by Prof. Michel Goldman, of The Free University of Brussels.

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.



- OPEN FOR ABSTRACT SUBMISSION UNTIL: **01/06/2021**
- OPEN FOR TRAVEL AWARD APPLICATION UNTIL: **01/06/2021**
- OPEN FOR REGISTRATIONS UNTIL: **01/09/2021**

Organizing Committee:

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

8th - 13th Sept 2021
Ioannina, Greece

FOCiS JUNE 8-11 2021 VIRTUAL ANNUAL MEETING

Link: <https://www.focisnet.org/meetings/focis-2021/program/>

Please plan to attend this Guest Symposium at the 2021 Meeting of the Federation of Clinical Immunology Societies (FOCIS). The symposium will be co-sponsored by the International Complement Society and American College of Rheumatology

Complement in Rheumatic Diseases – New Tricks for an Old Dog

Chairs:

- Joshua Thurman, University of Colorado
- Alfred Kim, Washington University

Don't let COVID19 keep you from exercising your brain! The annual FOCIS meeting covers all important areas of translational immunology and is a great opportunity to learn and to network. This seminar will review recent discoveries regarding the role of the complement system in rheumatologic disease. It will also review the current status of anti-complement therapeutics. It will be jointly sponsored by the International Complement Society and the American College of Rheumatology.

Date and time: **Tuesday, June 8, 10 AM – 1:30 PM EDT**

Speakers:

- Viviana Ferreira, DVM, PhD, University of Toledo College of Medicine
- Claudia Kemper, PhD, National Heart, Lung and Blood Institute
- Hrish Kulkarni, MD, Washington University School of Medicine
- Claire Harris, PhD, Newcastle University

We would like to thank our generous sponsors:

Alexion

Exsera BioLabs

National Jewish Health

Quidel

Contact: Joshua Thurman, joshua.thurman@cuanschutz.edu

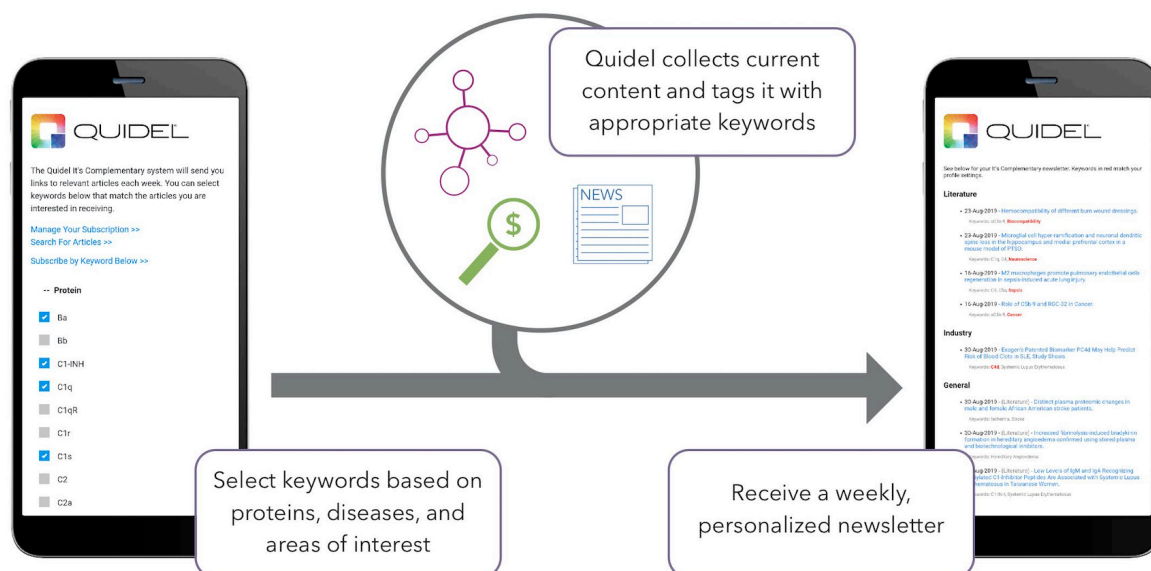


It's Complementary A Personalized Complement System Newsletter

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.

Visit <https://www.itscomplementary.com/> to learn more and subscribe.

From Our Sponsors

Focus on Complement



CompTech

Your primary source of
complement proteins and
reagents.

- Purified Complement Proteins
- Complement Cells (EA, Er) & Buffers
- Antisera to complement proteins
- Sera depleted of complement components
- Human Anaphylatoxins – C3a, C4a and desArg forms of C3a, C4a and C5a
- Now available: Human C5a (real C5a, not recombinant protein)

Complement Technology, Inc.

www.ComplementTech.com

Email: contactCTI@aol.com

Phone: 1-903-581-8284

Fax: 1-903-581-0491



Let us guide you on the Complement Path

Register to receive
your free poster
showing the
complement system
cascade



Svar Life Science AB | www.svarlifescience.com

