Preface

Editorial Compilation—Part XIV

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Welcome to the latest issue of *Seminars in Thrombosis and Hemostasis (STH)* published under the "banner" of "Editorial Compilation," this being the 14th such issue (►**Table 1**). Historically, *STH* is a theme-driven publication; however, ongoing opportunities emerge to disseminate wide-ranging contributions of current interest or controversy, and which do not straightforwardly suit an ongoing themed issue. We also require a medium for enabling publication of accepted peer-reviewed "unsolicited" manuscripts, as well as contributions from our Eberhard F. Mammen Young Investigator Award winners (see ►**Table 2** for previous editorials related to the Eberhard F. Mammen awards). As is now standard for this compilation series, the current issue contains a mixture of articles that comprise all the above elements, as well as broadly fitting within the standard themes of "thrombosis" and "bleeding."

This issue begins with a couple of reviews focused on disorders of hemostasis in the young.[1,2] First, Karapati and colleagues review the hemostatic profile of neonates with intrauterine growth restriction (IUGR). IUGR affects nearly 10 to 15% of pregnancies and is responsible for many short- and long-term adverse consequences, including hemostatic derangement. Both thrombotic and hemorrhagic events are described in the perinatal period in these neonates. The aim of this manuscript is to systematically review the literature on the laboratory studies used to evaluate the hemostatic system of the IUGR small for gestational age neonate. The authors reviewed the current literature using PubMed and Scopus until September 2022. Following defined inclusion/exclusion criteria, they finally included 60 studies in their review. Thrombocytopenia, characterized as hyporegenerative and a kinetic upshot of reduced platelet production *due* to in utero chronic hypoxia, was the main finding of most studies focusing on growth-restricted neonates. In most of cases, this was mild and usually resolved spontaneously within the first 2 weeks of life. In regard to coagulation, growth-restricted newborns present with prolonged standard coagulation tests. Data regarding coagulation factors, fibrinolytic system, and anticoagulant proteins are scarce and conflicting, mainly due to confounding factors. As thromboelastography/rotational thromboelastometry (TEG/ROTEM) is thought to provide a more precise evaluation of the in vivo coagulation process compared with standard coagulation tests, its use in transfusion guidance is growing. However, only one study regarding TEG/ROTEM was retrieved from this population, and no difference in ROTEM parameters compared with appropriate for gestational age neonates was found. Despite various laboratory aberrations, no correlation could be found with clinical manifestations of bleeding or thrombosis in the studies included in this review. The authors conclude that more studies are required to assess hemostasis in IUGR neonates and guide targeted therapeutic interventions.

Next, Radin and colleagues review pediatric presentations of the antiphospholipid syndrome (APS). The authors aimed to investigate the epidemiology, and clinical and laboratory

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Table 1 Past STH issues in the series "Editorial Compilation"

1. Favaloro EJ, Lippi G. Editorial Compilation I. Semin Thromb Hemost. 2016 Feb;42(1):5–8.
2. Favaloro EJ, Lippi G. Editorial Compilation II. Semin Thromb Hemost. 2016 Sep;42(6):599–602.
3. Favaloro EJ, Lippi G. Editorial Compilation III. Semin Thromb Hemost. 2017 Feb;43(1):4–7.
4. Favaloro EJ, Lippi G. Editorial Compilation IV. Semin Thromb Hemost. 2017 Sep;43(6):549–552.
5. Favaloro EJ, Lippi G. Editorial Compilation V. Semin Thromb Hemost. 2018 Apr;44(3):193–196.
6. Favaloro EJ, Lippi G. Editorial Compilation VI. Semin Thromb Hemost. 2019 Feb;45(1):5–9.
7. Favaloro EJ, Lippi G. Editorial Compilation VII. Semin Thromb Hemost. 2019 Jul;45(5):429–432.
8. Favaloro EJ, Lippi G. Editorial Compilation VIII. Semin Thromb Hemost. 2020 Jun;46(4):393–397.
9. Favaloro EJ, Lippi G. Editorial Compilation IX. Semin Thromb Hemost. 2021 Feb;47(1):6–10.
10. Favaloro EJ, Lippi G. Editorial Compilation X. Semin Thromb Hemost. 2021 Oct 47(7):754–758.
11. Favaloro EJ, Lippi G. Editorial Compilation XI. Semin Thromb Hemost. 2022 Mar;48(2):127–131.
12. Favaloro EJ, Pasalic L, Lippi G. Editorial Compilation XII. Semin Thromb Hemost. 2022 Jul;48(5):497–501.
13. Favaloro EJ, Pasalic L, Lippi G. Editorial Compilation XIII. Semin Thromb Hemost. 2023 Jul;49(5):427–432

Table 2 Past STH editorials related to Eberhard F. Mammen award announcements

1. Favaloro EJ. Welcome to a special issue of Seminars in Thrombosis and Hemostasis—the closing issue for 2008. Semin Thromb Hemost 2008;34: 693–696
2. Favaloro EJ. A tribute to Eberhard F. Mammen, M.D. (1930–2008). Semin Thromb Hemost 2008;34:703–708
3. Favaloro EJ. Welcome to the first issue of Seminars in Thrombosis and Hemostasis for 2009. Semin Thromb Hemost 2009;35:1–2.
4. Favaloro EJ. Winners of the Inaugural Eberhard F. Mammen Award for Most Popular Article. Semin Thromb Hemost 2009;35:587–590
5. Favaloro EJ. Editorial. 2009 Eberhard F. Mammen Young Investigator Award Winners. Semin Thromb Hemost 2010;36:469–470
6. Favaloro EJ. Winners of the 2010 Eberhard F. Mammen Award for Most Popular Article during 2008–2009. Semin Thromb Hemost. 2010;36(7):685–692.
7. Favaloro EJ. 2011 Eberhard F. Mammen award announcements. Semin Thromb Hemost. 2011;37(5):431–439.
8. Favaloro EJ. 2012 Eberhard F. Mammen award announcements. Semin Thromb Hemost. 2012;38:425–32.
9. Favaloro EJ. 2013 Eberhard F. Mammen award announcements. Semin Thromb Hemost 2013;39:567–574.
10. Favaloro EJ. 2014 Eberhard F. Mammen award announcements: Part I - most popular articles. Semin Thromb Hemost. 2014;40(4):407–412.
11. Favaloro EJ. 2014 Eberhard F. Mammen Award Announcements: Part II - Young Investigator Awards. Semin Thromb Hemost. 2014;40(7):718–723.
12. Favaloro EJ. 2015 Eberhard F. Mammen Award Announcements: Part I-Most Popular Articles. Semin Thromb Hemost. 2015;41(7):673–679.
13. Favaloro EJ. 2015 Eberhard F. Mammen Award Announcements: Part II-Young Investigator Awards. Semin Thromb Hemost. 2015;41(8):809–815.
14. Favaloro EJ. 2016 Eberhard F. Mammen Award Announcements: Part I - Most Popular Articles. Semin Thromb Hemost. 2016;42(4):325–330.
15. Favaloro EJ. 2016 Eberhard F. Mammen Award Announcements: Part II-Young Investigator Awards. Semin Thromb Hemost. 2017;43(3):235–241.
16. Favaloro EJ. 2017 Eberhard F. Mammen Award Announcements: Part I-Most Popular Articles. Semin Thromb Hemost. 2017;43(4):357–363.
17. Favaloro EJ. 2017 Eberhard F. Mammen Award Announcements: Part II-Young Investigator Awards. Semin Thromb Hemost. 2018;44(2):81–88.
18. Favaloro EJ. 2018 Eberhard F. Mammen Award Announcements: Part I-Most Popular Articles. Semin Thromb Hemost. 2018;44(3):185–192.

Table 2 (*Continued*)

19. Favaloro EJ. 2018 Eberhard F. Mammen Award Announcements: Part II-Young Investigator Awards. Semin Thromb Hemost. 2019;45(2):123–129.
20. Favaloro EJ. 2019 Eberhard F. Mammen Award Announcements: Part I-Most Popular Articles. Semin Thromb Hemost. 2019;45(3):215–224.
21. Favaloro EJ. 2019 Eberhard F. Mammen Award Announcements: Part II—Young Investigator Awards. Semin Thromb Hemost 2020;46(2):105–113
22. Favaloro EJ. 2020 Eberhard F. Mammen Award Announcements: Part I-Most Popular Articles. Semin Thromb Hemost. 2020;46(4):383–392.
23. Favaloro EJ. 2020 Eberhard F. Mammen Award Announcements: Part II—Young Investigator Awards. Semin Thromb Hemost 2021;46(3):229–237.
24. Favaloro EJ. 2021 Eberhard F. Mammen Award Announcements: Part I-Most Popular Articles. Semin Thromb Hemost. 2021 Jul;47(5):467–476.
25. Favaloro EJ. 2021 Eberhard F. Mammen Award Announcements: Part II-Young Investigator Awards. Semin Thromb Hemost. 2022 Apr;48(3):265–273.
26. Favaloro EJ. 2022 Eberhard F. Mammen Award Announcements: Part I-Most Popular Articles. Semin Thromb Hemost. 2022 Jul;48(5):502–513.
27. Favaloro EJ. 2023 Eberhard F. Mammen Award Announcements: Part I-Most Popular Articles. Semin Thromb Hemost. 2023 Jul;49(5):417–426.
28. Favaloro EJ. 2022 Eberhard F. Mammen Award Announcements: Part II-Young Investigator Awards. Semin Thromb Hemost. 2023 Apr 28. doi:10.1055/s-0043-57033 (in press)

characteristics of pediatric involvement of APS, by performing a review of the current evidence and reviewing local experience in the Northwest Italy. They performed a detailed literature search to identify articles describing clinical and laboratory characteristics of pediatric APS. In concomitance, they conducted a registry study collecting data from the Piedmont and Aosta Valley rare disease registries, including pediatric patients diagnosed with APS in the last 11 years. The literature review led to the inclusion of six articles with a total of 386 pediatric patients (65% females, 50% with systemic lupus erythematosus [SLE] as concomitant diagnosis). Rates of venous and arterial thrombosis were 57 and 35%, respectively. "Extra-criteria manifestations" included mostly hematological and neurological involvement. Almost one-fifth of patients (19%) reported recurrent events and 13% manifested as catastrophic APS. A total of 17 pediatric patients (mean age: 15.1 ± 2.8, 76% female) developed APS in the Northwest of Italy. In 29% of cases, SLE was a concomitant diagnosis. Deep vein thrombosis was the most frequent clinical manifestation (28%), followed by catastrophic APS (6%). The estimated prevalence of pediatric APS in Piedmont and Aosta Valley Region is 2.5/100,000 people, whereas the estimated annual incidence is 0.2/100,000 inhabitants. The authors conclude that clinical manifestations of pediatric APS seem to be more severe and with high prevalence of noncriteria manifestations, and that international efforts are needed to better characterize this condition and to develop new specific diagnostic criteria to avoid missed/delayed diagnosis in children with APS.

This issue of STH continues with a contribution from a 2021 Young Investigator winner, Dr. Sol Schulman,[3] and his team on tissue factor (TF)-dependent disease.[4] TF is the primary initiator of blood coagulation in humans. As improper intravascular TF expression and procoagulant activity underlie numerous thrombotic disorders, there has been longstanding interest in the contribution of heritable genetic variation in *F3,* the gene encoding TF, to human disease. This review aims to comprehensively and critically synthesize small case–control studies focused on candidate single nucleotide polymorphisms (SNPs), as well as modern genome-wide association studies (GWASs) seeking to discover novel associations between variants and clinical phenotypes. Where possible, correlative laboratory studies, expression quantitative trait loci, and protein quantitative trait loci were evaluated to glean potential mechanistic insights. Most disease associations implicated in historical case–control studies have proven difficult to replicate in large GWAS. Nevertheless, SNPs linked to *F3*, such as rs2022030, are associated with increased *F3* mRNA expression, monocyte TF expression after endotoxin exposure, and circulating levels of the prothrombotic biomarker D-dimer, consistent with the central role of TF in the initiation of blood coagulation.

Next, one of our associate editors, Akbar Dorgalaleh, provides some novel insights into heterozygous factor (F) XIII deficiency.[5] Although the prevalence and clinical significance of severe FXIII deficiency has long been established, that for heterozygous FXIII deficiency is debated, with controversial reports emerging since 1988. In the absence of large epidemiologic studies, but based on a few studies, a prevalence of 1 per 1,000 to 5,000 is estimated. In southeastern Iran, a hotspot area for the disorder, a study of more than 3,500 individuals found instead an incidence of 3.5%. Between 1988 and 2023, a total of 308 individuals were found with heterozygous FXIII deficiency, of which molecular, laboratory, and clinical presentations were available for

207 individuals. A total of 49 variants were found in the *F13A* gene, most of which were missense (61.2%), followed by nonsense (12.2%) and small deletions (12.2%), most occurring in the catalytic domain (52.1%) of the FXIII-A protein, and most frequently in exon 4 (17%) of the *F13A* gene. This pattern is relatively similar to homozygous (severe) FXIII deficiency. In general, heterozygous FXIII deficiency is an asymptomatic condition without spontaneous bleeding tendency, but it can lead to hemorrhagic complications in hemostatic challenges such as trauma, surgery, childbirth, and pregnancy. Postoperative bleeding, postpartum hemorrhage, and miscarriage are the most common clinical manifestations, while impaired wound healing has also been rarely reported. Although some of these clinical manifestations can also be observed in the general population, their frequency is seemingly higher in subjects with heterozygous FXIII deficiency. While studies of heterozygous FXIII deficiency conducted over the past 35 years have shed light on some of the ambiguities of this condition, the author concludes that further studies on a large number of heterozygotes are needed to answer the major questions related to heterozygous FXIII deficiency.

We continue the discussion of hemostasis pathways with the next contribution, from the authorship team of Tiu, Chiasakul, and Kessler, who discuss pitfalls in the use of global hemostasis assays in patients with myeloproliferative neoplasms (MPNs).[6] These MPNs mainly comprise polycythemia vera (PV), essential thrombocythemia (ET), and primary myelofibrosis (PMF). Venous and arterial thromboembolisms are major complications of these MPNs, and global hemostasis assays, including thrombin generation assay (TGA), TEG, and ROTEM, have been proposed as reliable tools to assess the hypercoagulability and thrombotic risk stratification in MPNs. The authors therefore performed a systematic literature review on the parameters of TGA, ROTEM, and TEG, and their association with thrombotic events and treatment strategies in MPNs. Thirty-two studies (all cross-sectional) were included, which collectively enrolled 1,062 controls and 1,608 MPN patients. Among the 13 studies that reported arterial or venous thrombosis, the overall thrombosis rate was 13.8%, with six splanchnic thromboses reported. Out of the 27 TGA studies, there was substantial heterogeneity in plasma preparation and trigger reagents employed in laboratory assays. There was a trend toward increased peak height among all MPN cohorts versus controls and higher endogenous thrombin potential (ETP) between ET patients and controls. There was an overall trend toward lower ETP between PV and PMF patients versus controls. There were no substantial differences in ETP between JAK2-positive versus JAK2-negative MPNs, prior history versus negative history of thrombotic events, and among different treatment strategies. Of the three ROTEM studies, there was a trend toward higher maximum clot firmness and shorter clot formation times for all MPNs versus controls. The three TEG studies had mixed results. The authors conclude that the ability of parameters from global hemostasis assays to predict for hypercoagulability events in MPN patients is inconsistent and inconclusive, and that further prospective longitudinal studies are needed to validate these tests so that thrombotic potential could be utilized as a primary endpoint of such studies.

A review on the role of CD36/GPIV in platelet biology from the writing team of Bendas and Schlesinger follows.[7] CD36 (also known as platelet glycoprotein IV) is expressed by a variety of different cell entities, where it functions as a signaling receptor, but additionally acts as a transporter for long-chain fatty acids. This dual function of CD36 has been investigated for its relevance in immune and nonimmune cells. Although CD36 was first identified on platelets, the understanding of the role of CD36 in platelet biology remained scarce for decades. In the last few years, several discoveries have shed a new light on the CD36 signaling activity in platelets. Notably, CD36 has been recognized as a sensor for oxidized low-density lipoproteins in the circulation that mitigates the threshold for platelet activation under conditions of dyslipidemia. Thus, platelet CD36 transduces atherogenic lipid stress into an increased risk for thrombosis, myocardial infarction, and stroke. The underlying pathways that are affected by CD36 include the inhibition of cyclic nucleotide signaling pathways and simultaneously the induction of activatory signaling events. Furthermore, thrombospondin-1 secreted by activated platelets binds to CD36 and furthers a paracrine platelet activation. CD36 also serves as a binding hub for different coagulation factors and, thus, contributes to the plasmatic coagulation cascade. This review provides a comprehensive overview of recent findings on platelet CD36 and presents CD36 as a relevant target for prevention of thrombotic events in dyslipidemic individuals with an elevated risk for thrombosis.

Next is a contribution from Sedky Adly and colleagues, who report a scoping review on laser-induced blood coagulation for surgical application.[8] It is not really clear whether "coagulation" in this context is that we usually take to mean, being coagulation of blood through various hemostasis pathways. In surgical settings, lasers are used to coagulate blood with the main aim to "cauterize" a wound and prevent excessive blood loss. Nevertheless, we felt the broader "definition" of coagulation could provide an interesting perspective to our readership. In brief, there remains a lack of evidence-based reviews on the effects of laser irradiation on blood coagulation in the literature, despite a large number of clinical trials. The authors therefore evaluated the available evidence on laser irradiation parameters used in coagulation of blood to optimize physical parameters. They performed a literature search for recent scientific studies indexed between 2017 and 2023 using the databases of PubMed and ScienceDirect. Articles were selected based on defined inclusion and exclusion criteria, and 78 publications in total were eventually included in this scoping review. They report on various parameters that were found to produce significant benefits in blood coagulation for surgical application, across a broad area of surgeries (dentistry and oral; urogenital disorders; ophthalmic disorders; embryological surgeries; dermatological disorders; gastrointestinal disorders; neurological surgeries; pulmonary disorders; cardiovascular disorders). The authors conclude, combining data from all clinically heterogeneous studies, that laser

irradiation reflects an effective method for inducing blood coagulation in several medical fields.

Another "unusual" review follows, from the team of Leerson and colleagues,[9] this on detecting oxygenator thrombosis in extracorporeal membrane oxygenation (ECMO). ECMO is a life support technique used to treat cardiac and pulmonary failure, including severe cases of COVID-19 (coronavirus disease 2019) involving acute respiratory distress syndrome. Blood clot formation in the ECMO circuit is one of the most common complications of ECMO treatment, having potentially harmful and even fatal consequences. It is therefore essential to regularly monitor for clots within the circuit and take appropriate measures to prevent or treat them. The authors present a review of the various methods used by hospital units for detecting blood clots, and discuss the benefits and limitations of each method, especially concerning detecting blood clots in the oxygenator, as it is concluded that this is the most critical and challenging ECMO component to assess. The authors also investigate the feasibility of solutions proposed in the published literature and explore two areas that hold promise for future research: the analysis of small-scale pressure fluctuations in the circuit, and real-time imaging of the oxygenator. The authors conclude that current methods for detecting blood clots cannot reliably predict clot volume, and their inability to predict clot location puts patients at risk of thromboembolism. It is further posited that a more in-depth analysis of pressure readings using machine learning could better provide this information, and that purpose-built imaging could allow for accurate, real-time clotting analysis in ECMO components.

The last review in this issue of STH is by two of the guest editors, Giuseppe Lippi and Emmanuel Favaloro, who provide a concise review on the epidemiology and predisposing factors of post-COVID venous thrombosis.[10] As previously reported, STH has previously published a series of issues around the theme of maintaining hemostasis and preventing thrombosis in COVID-19, with the final such issue published in early 2023.[11] As the international emergency for COVID-19 has been withdrawn by the World Health Organization, and we start to return to "business as usual," we are no longer publishing COVID-19 themed issues, and instead any COVID-19-related material accepted for publication will now appear in the other compilation themes in progress, such as the Editorial Compilation series (►**Table 1**). Moreover, publication of manuscripts related to acute COVID-19 will expectedly decline, and instead be replaced with manuscripts that discuss chronic pathophysiology of post-acute COVID-19 consequences. The so-called Long-COVID represents a heterogeneous clinical syndrome characterized by a pathologic continuum of signs, symptoms, and also laboratory/radiologic abnormalities that may persist for a long time after recovering from an acute SARS-CoV-2 (severe acute respiratory syndrome coronavirus disease 2) infection. Among the various components of this post-viral condition, the risk of venous thromboembolism in patients hospitalized for COVID-19 remains considerably higher after discharge, especially early after discharge (i.e., within the first 6–12 months), in older individuals, in men, in patients with longer hospital stays and more aggressive treatment (e.g., mechanical ventilation and/or intensive care), when thromboprophylaxis is not used, and in those with a persistent prothrombotic state. The authors therefore advise that patients who have these predisposing factors should be monitored more closely to intercept any thrombosis that may occur in a post-COVID time-related manner, but may also benefit from extended thromboprophylaxis and/or antiplatelet therapy.

The last full-length paper in this issue is an original study on the effects of recombinant SARS-CoV-2 spike protein variants on platelet morphology and activation, from the team of Vettori and colleagues.[12] As our readership well understands, platelets represent central elements of hemostasis and also play a pivotal role in the pathogenesis of COVID-19 thrombosis. The current study was planned to investigate the effects of different SARS-CoV-2 recombinant spike protein variants on platelet morphology and activation. Citrated whole blood collected from ostensibly healthy subjects was challenged with saline (control sample) and with 2 and 20 ng/mL final concentration of SARS-CoV-2 spike protein of Ancestral, Alpha, Delta, and Omicron variants. Platelet count was found to be decreased with all SARS-CoV-2 recombinant spike protein variants and concentrations tested, achieving the lowest values with 20 ng/mL Delta recombinant spike protein. The mean platelet volume increased in all samples irrespective of SARS-CoV-2 recombinant spike protein variants and concentrations tested, but especially using Delta and Alpha recombinant spike proteins. The values of both PFA-200 collagen-adenosine diphosphate (C-ADP) and collagen-epinephrine (C-EPI) increased in all samples irrespective of SARS-CoV-2 recombinant spike protein variants and concentrations tested, and thus reflecting platelet exhaustion, and displaying again higher increases with Delta and Alpha recombinant spike proteins. Most samples where SARS-CoV-2 recombinant spike proteins were added were flagged as containing platelet aggregates. Morphological analysis revealed the presence of a considerable number of activated platelets, platelet clumps, and platelet–monocyte and platelet–neutrophils aggregates, especially in samples spiked with Alpha and Delta spike protein variants at 20 ng/mL. Therefore, these results provide support to the evidence that SARS-CoV-2 is capable of directly activating platelets through its spike protein, though such effect varies depending on different spike protein variants.

Next, Thachil and colleagues ask the question: "Are antiphospholipid antibodies a surrogate risk factor for thrombosis in sepsis?" in a Commentary on this topic.[13] As also discussed by Radin et al in this issue,[1] APS is a hypercoagulable state caused by antiphospholipid antibodies (aPLs). APS clinically manifests with arterial or venous or microvascular thrombi and/or pregnancy complications. It is well known that the development of aPL can be a transient phenomenon, so that the current diagnostic criterion for APS requires repeat laboratory testing several weeks apart before a definitive diagnosis is made. However, transient presence of aPL may also be pathogenic, as for example been inferred for virally induced aPL, including as associated with COVID-19.[14,15] In the current Commentary, the authors attempt to give historical and clinical evidence for the importance of these antibodies, even when transient, and call for further

research into mechanisms by which they may promote thrombosis and pregnancy morbidities.

As usual for these nonthematic issues of STH, we complete this issue with some additional Commentaries and finish with some correspondence. First, Turner and colleagues point to increased levels of inflammatory and endothelial biomarkers in blood of Long COVID patients as potential markers of thrombotic endothelialitis.[16] Then, Minutti-Zanella et al discuss D-dimer levels in COVID-19 as a potential acute phase reactant.[17] Simini et al discuss the safety and efficacy of therapeutic anticoagulation with subcutaneous unfractionated heparin in patients with renal failure.[18] Marongiu and Barcellona attempt to answer the question of why rivaroxaban does not work in severe mitral stenosis.[19] Then, Monti et al describe two interesting cases of acquired von Willebrand syndrome, each requiring a different treatment path because of differing primary defects.[20] Finally, Simurda et al discuss the perioperative monitoring by ROTEM of a severe hemophilia A patient undergoing elective ankle surgery.[21]

And yet, the content described above does not end this issue of STH. As we are celebrating 50 years of publishing, each issue of 2024 will also republish a key paper from the past, and also contain invited Commentaries discussing the historical paper in the context of what we know today. In the first issue of 2024, STH republished the first paper that STH ever published, on the molecular structure of fibrinogen,[22] with an accompanying Commentary from Alessandro Casini and Marguerite Neerman-Arbez.[23] In the current issue of STH, we are republishing the third most cited manuscript from STH according to Web of Science,[24] and an accompanying Commentary from the Editor in Chief of STH.[25] The historical republished paper provides the first ever description of the PFA-100,[24] and the accompanying Commentary provides some historical context around the significance of this event, as well as taking us on a journey since that time to current times.[25]

We once again thank all the authors to this latest issue of "Editorial Compilations" for their original and comprehensive contributions, and we hope our readership enjoys this latest instalment in this series.

Conflict of Interest
None declared.

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