

► Reliable assessment of sTCC/sC5b-9; a useful marker for assessing complement activation

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► Abstract



The complement system is a critical component of innate immunity, with terminal complement activation culminating in the formation of the membrane attack complex (MAC) or its soluble counterpart, the soluble Terminal Complement Complex (sTCC, also known as sC5b-9). Dysregulation of terminal pathway activation is implicated in a range of inflammatory and autoimmune diseases, including systemic lupus erythematosus (SLE), atypical hemolytic uremic syndrome (aHUS), and transplant rejection. As quantifying sTCC levels offers crucial insight into complement activation status, it has emerged as a potential biomarker for disease onset, severity, prognosis, and response to complement-targeted therapies. In addition, sTCC quantification enables pharmacodynamic monitoring of complement inhibitors, making it a valuable marker in the development of novel drugs targeting complement-mediated diseases. Here, we describe the development of the sTCC ELISA (cat.# [HK238](#)), a highly specific and reliable immunoassay for quantifying sTCC in human plasma and serum. The assay utilizes the neoepitope-specific monoclonal antibody aE11 to selectively capture activated C9 incorporated into sTCC, enabling precise measurement of the endpoint of terminal complement activation. Assay performance was thoroughly evaluated, demonstrating good sensitivity, specificity, recovery, linearity, and reproducibility across various matrices. Altogether, the sTCC ELISA provides a standardized, well-characterized assay for reliable assessment of terminal complement activation, supporting both mechanistic studies and clinical decision-making in the context of complement-driven diseases.

► Introduction

The complement system is a key component of innate immunity. First described in the late 19th century by Jules Bordet, it was found that blood serum contains factors enhancing antibody-mediated bacterial lysis. Later research identified terminal complement complex (TCC) formation as the final step of complement activation (1-3). The cascade involves ~50 proteins and is triggered via the classical, lectin, alternative, or indirectly via the extrinsic coagulation pathway. Beyond pathogen elimination through TCC-mediated lysis, the complement system plays roles in autoimmune and inflammatory diseases such

as SLE, AMD, and transplant rejection (4, 5), and also influences adaptive immunity. Activation leads to formation of the C3 convertase, which propagates the cascade to generate C5b via the C5 convertase. C5b marks the start of the terminal pathway (TP), leading to assembly of the C5b-9 complex (6, 7), which exists as a membrane-bound variant (MAC) or soluble form (sTCC). TCC forms a barrel-shaped pore in membranes, driving cell lysis (8).

The terminal complement complex (TCC), or MAC, is a critical effector that forms pores in target membranes. Its assembly is tightly regulated and



irreversible, involving C5b, C6, C7, C8, and multiple C9 molecules (9). Following C5 cleavage, unstable C5b binds C6 to form C5b6, which then recruits C7, triggering conformational changes that expose hydrophobic domains, enabling membrane binding. This is followed by C8, a heterotrimer (C8 α , C8 β , C8 γ), where C8 α contains a membrane attack complex/perforin (MACPF) domain responsible for insertion into the lipid bilayer (10). This stabilizes the complex and allows up to 18 C9 molecules to polymerize, forming a transmembrane β -barrel pore. Cryo-electron microscopy has shown that MAC adopts a unique “split-washer” configuration, contributing to its asymmetric structure (11, 12). MAC pore formation disrupts membrane integrity, inducing cell lysis via uncontrolled ion exchange and osmotic imbalance.

At sublytic levels, MAC also triggers intracellular signaling affecting survival, proliferation, and inflammation (13). While vital for defense against Gram-negative bacteria, dysregulated

MAC activity contributes to autoimmune and inflammatory pathology (14). Due to the inefficiency of membrane insertion, a soluble, non-lytic form of the complex, namely sTCC, is often released, representing failed MAC assembly (8, 14, 15) (Figure 1).

In recent years it became apparent that sTCC is a valuable biomarker for assessing terminal complement activation in complement-mediated diseases and for monitoring treatment response in patients receiving complement inhibitors (16-18). However, in order to do so, sensitive and reliable quantification of sTCC is essential. To quantify complement activation, various immunoassays have been developed, typically employing monoclonal antibodies that detect neo-epitopes present only in activation products. One such antibody, aE11 (HM2167), binds a C9 neo-epitope exposed only in TCC or poly-C9 (19). This ensures that the aE11 antibody specifically recognizes the assembled TCC complex, but not its individual components. To specifically measure

Box 1.

Harmonizing Bioanalytical Method Validation: The ICH M10 Guideline

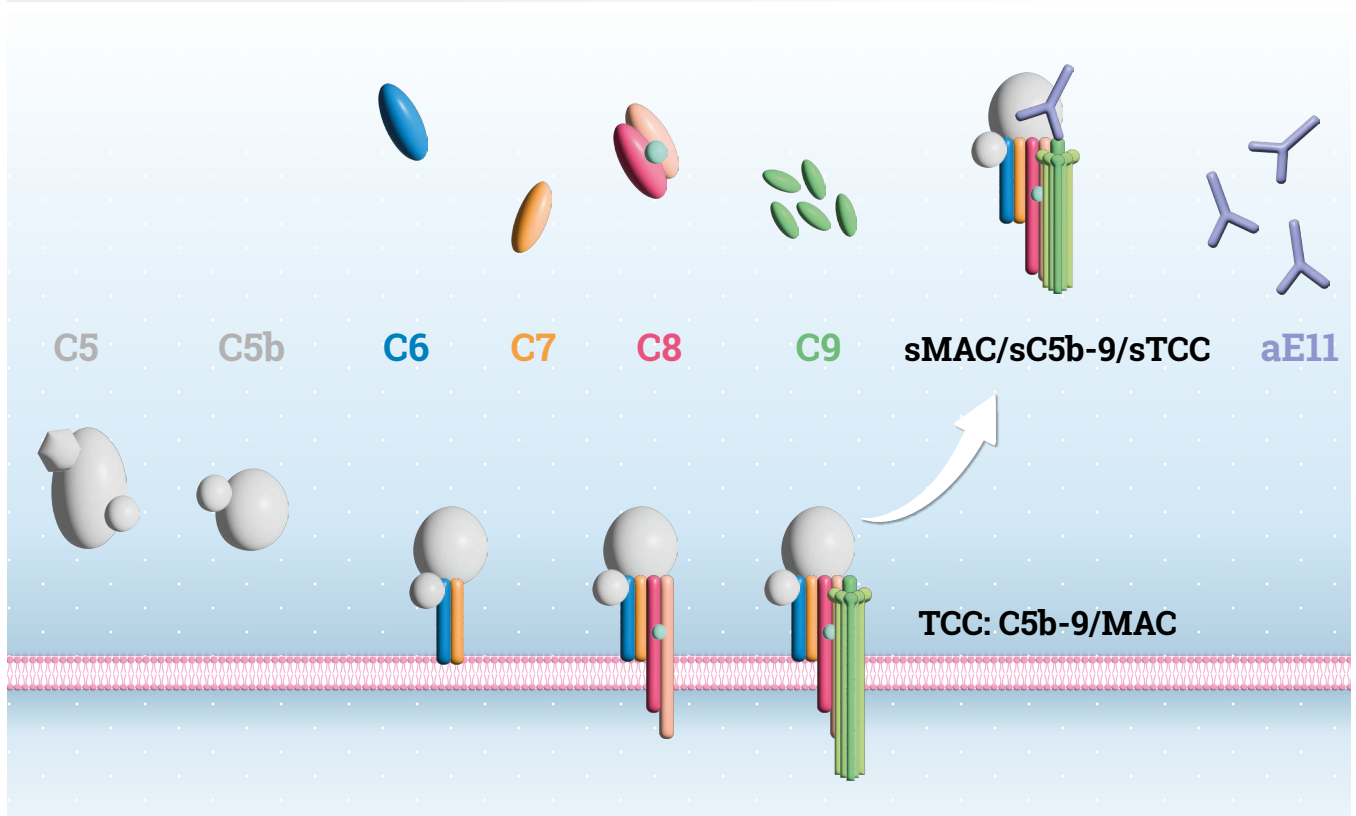
About ICH M10

The ICH M10 Guideline sets global standards for how bioanalytical methods, like ELISAs or chromatographic assays, should be validated and used in drug studies. It replaces older guidelines from the FDA and EMA to create one clear, harmonized approach. M10 covers key aspects such as calibration curves, accuracy, precision, selectivity, matrix effects, and sample stability. The goal is to ensure reliable results when measuring drugs or biomarkers in clinical and non-clinical samples. Up-to-date guidelines can be downloaded at <https://www.ema.europa.eu/en/ich-m10-bioanalytical-method-validation-scientific-guideline>. By following M10, labs around the world can work more consistently, reduce repeat work, and support faster drug development. An ELISA validated according to ICH M10 Guidelines ensures reliable, reproducible, and regulatory-compliant data on analyte concentrations in biological matrices, which is essential for drug development and decision-making. Extra documents from the EMA and FDA help with practical details like use of animal-free matrices or bioequivalence studies. In the table 1 below, we show how our assay compares to the main points of the ICH M10 guideline.

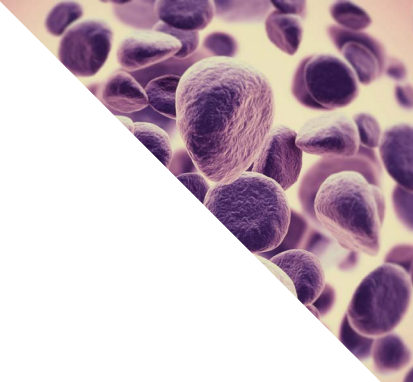
terminal pathway activation, aE11 is incorporated in several sTCC and pathway ELISAs (e.g. [HK328](#), [HK3010/3012](#)) (20). Here, we present data demonstrating that [HK328](#) enables accurate, sensitive, and reproducible quantification of sTCC

in plasma and serum, supporting its application as a well-characterized assay for assessing terminal complement activation in both research and clinical settings, including drug development and therapeutic response monitoring.

Figure 1:



Schematic representation of the membrane attack complex (MAC/C5b-9/TCC) and its soluble counterpart (sMAC/sC5b-9/sTCC). Upon complement activation, C5 is cleaved to generate C5b, which initiates the sequential, non-covalent binding of complement components C6 through C9, resulting in the assembly of the pore-forming MAC on pathogen surfaces. Concurrently, C5b can also associate with C6-C9 in the fluid phase, forming soluble intermediates that lead to the generation of sC5b-9/sTCC. These soluble complexes bind to regulatory proteins, preventing their insertion into host or microbial membranes. The monoclonal antibody aE11 recognizes a neoepitope on C9 that is present in both membrane-bound Mac and the fluid-phase sC5b-9 complexes. The Hycult Biotech human sTCC assay ([HK328](#)) recognizes the soluble part of TCC, sTCC.



► Materials and methods

Biological samples

Human CHES panels (citrate, heparin, EDTA plasma and serum samples from a single donor) from healthy donors were purchased from BioIVT (New York, USA) and stored in aliquots at -80°C. For complement-preserved serum samples, blood was collected aseptically and processed within 1 hour. After coagulation at RT for 30–45 min., samples were centrifuged at $2,000 \times g$ at 4 °C and serum was collected. Samples were stored at -80 °C until further analysis. For the calibrator, a

serum pool with a known sTCC concentration was obtained from the Department of Immunology at the University of Oslo and Oslo University Hospital Rikshospitalet (Oslo, Norway) (21). This reference material was used to calibrate an internal serum pool by parallel testing and standard curve alignment. The resulting internally calibrated pool was subsequently used as the assay standard to ensure consistency across experiments.

Development of sTCC sandwich ELISA (HK328)

To enable specific and reliable quantification of sTCC in human plasma and serum, an ELISA was developed using the C9 neo-epitope-specific monoclonal antibody aE11. The assay consists of a 96-wells plate (MaxiSorp™ 96-wells plates, Thermo Fisher Scientific, Waltham, USA) coated with mAb aE11 [HM2167](#); Hycult Biotech, Uden, The Netherlands) to specifically capture sTCC. Plasma (EDTA, citrate and heparin) and serum samples were diluted at least 20 times in assay specific dilution buffer and, next to a calibrator, incubated in the precoated wells for 1h at RT. Following a four-step wash, wells are incubated for 1 hour with a biotinylated monoclonal anti-C6 antibody (9C4), recognizing the C6 protein incorporated in the sTCC complex. After an additional wash step, streptavidin-peroxidase conjugate was added and incubated for 1h allowing to bind the biotinylated detection antibody. TMB substrate was added, and the reaction was stopped after 30 min. using 2% (w/v) oxalic acid. The optical density (OD) was measured at 450 nm using a microplate reader (BioTek Synergy HT). Unless otherwise specified, this protocol was used to perform measurements

and evaluate assay characteristics such as; specificity, sensitivity, linearity, recovery and intra- and inter-assay variability. During development, variation between measurements or conditions was assessed by calculating the coefficient of variation (%CV) over multiple determinations of a single sample.

Assay performance was evaluated according the following general requirements. For optimal dynamic range, the optical density at 450 nm for the highest standard (S1) was required to fall between 1.7 and 3.5, ensuring strong signal intensity without saturation. The blank was accepted only if $OD_{450} \leq 0.2$ to confirm minimal background. A signal-to-noise ratio (S1/blank) above 10 was required to ensure sufficient assay sensitivity and discrimination. Precision was assessed by calculating the coefficient of variation (%CV) across at least three serial dilutions of the same sample, with a threshold of <15% considered acceptable for reliable quantification. These criteria align with common immunoassay validation standards and were used throughout assay development to confirm analytical robustness.

Immunoassay specificity and cross-reactivity

Specificity and cross-reactivity of the immunoassay was investigated using native purified C5, C6, C7, C8 and C9 (Complement Technology Inc., Texas, USA). Proteins were added to the wells in a concentration of 1500 ng/mL in assay dilution buffer and obtained OD values were used to calculate concentrations. In addition, it was also investigated whether the

assays cross-react with complement proteins from species other than human. The animal plasma samples (mouse, rat, pig, non-human primate NHP, horse, rabbit) were tested up to a ten-fold higher concentration than the recommended concentration for human plasma samples.

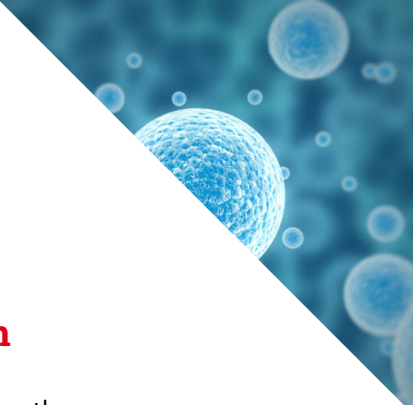
Recovery of sTCC complex in EDTA plasma samples

The recovery of sTCC in the assay was evaluated using EDTA plasma samples. Samples from different donors, exhibiting varying sTCC concentrations, were mixed in the following ratios: 100:0, 75:25, 50:50, 25:75, and 0:100. Each mixture was incubated for 30 minutes at

RT. Subsequently, sTCC concentrations in the mixed samples were measured according to the previously described protocol. Recovery was determined for each mixture by comparing the measured concentration to the expected value, calculated using the following equation:

$$\text{Recovery (\%)} = \frac{\text{Measured concentration of mixed sample}}{\text{Expected concentration } [(\% \times \text{sample A}) + (\% \times \text{sample B})]} \times 100$$

Requirements were met if recovery was between 80-120%.



Intra- and inter-assay variation and inter-batch variation

Intra-assay variation was assessed by repeated measurements of the same samples within a single ELISA plate. Inter-assay variation was determined by comparing results from the same samples tested independently by different operators in a blinded manner. Inter-batch

variation was evaluated by testing the same samples across three independently produced assay batches. A %CV of $\leq 15\%$ for intra- and inter-assay variation, and $\leq 20\%$ for inter-batch variation, was considered indicative of low variability.

Statistical analysis

For statistical analysis and data visualization GraphPad Prism 9.5 was used. Non-linear regression (Sigmoidal Four Parameter-Logistic (4PL) curve fit) was used to determine the dose-response curve and subsequently to determine

the coefficient of determination (R^2). One-way ANOVA followed by Tukey's multiple comparisons test was used to compare the effect of different matrices. A $p < 0.05$ was considered statistically significant (** $p < 0.01$, 'ns': non-significant).

Box 2.

For more information

At Hycult Biotech, we understand the challenges of immunological research. With over 30 years of expertise in innate immunity, we specialize in antibodies and immunoassays for complement and immunology research, as well as drug discovery. We develop and manufacture our products in-house under EN ISO 13485:2016 standards to ensure reliable and reproducible results. Our flexible approach allows us to offer both ready-to-use and custom solutions tailored to your study or clinical trial needs. Over 98% of our products are available for immediate delivery, supported by direct technical assistance and comprehensive documentation. Our consistent product quality helps streamline your research and save valuable time. What challenges can we help you solve? We are here to support your success. Contact us at: support@hycultbiotech.com or www.hycultbiotech.com

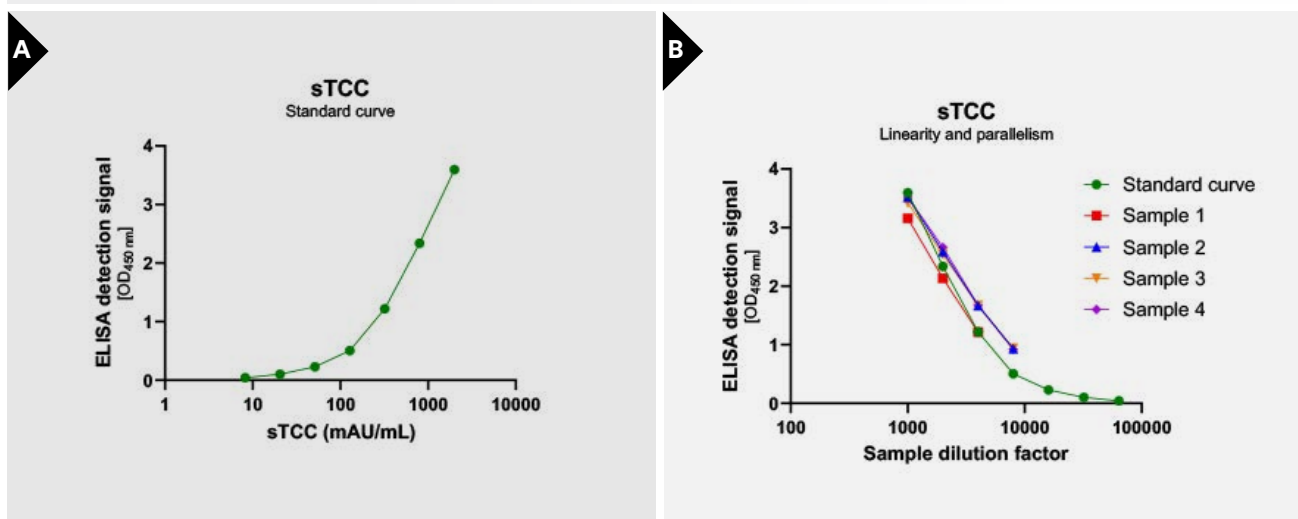
Results

Assay characteristics and performance

For enabling quantitative measurements of sTCC, a standard curve was created by calibrating a pool of zymosan-activated serum samples against a previously calibrated serum pool with a known sTCC concentration (21). Further experiments were performed using the internal produced serum pool as calibrator. Calibration was achieved using a seven-point standard curve with a dynamic range from 8.2 to 2000 mAU/mL, providing consistent and reproducible sigmoidal fits (Figure 2A). Curve progression is linear

between 2000 and 51.2 mAU/mL (upper limit of quantification; ULoQ: 2000 mAU/mL, lower limit of quantification; LLoQ: 8.2 mAU/mL) and resulted in a coefficient of determination of $R^2 = 0.9986$ between the measured OD450nm and the sTCC concentration (Figure 2A; Table 1). Linearity and parallelism were confirmed across serial dilutions of EDTA plasma samples, aligning well with the standard curve and yielding CVs <15% across tested dilutions, indicating good linearity (Figure 2B).

Figure 2:



sTCC (HK328) assay performance characteristics. **(A)** Seven-point calibration curve. **(B)** Parallelism between sTCC standard curve and EDTA-plasma samples from healthy volunteers (1000-8000 dilution range).

To assess assay accuracy and reproducibility, recovery of the target analyte and intra- and inter-assay variation were evaluated. In addition, also inter-batch variation was assessed. Recovery was analysed in EDTA plasma samples with a known sTCC concentration by mixing them in different ratios: 100:0, 75:25, 50:50, 25:75 and

0:100. Results showed adequate recovery, with results ranging between 80 and 120% (Figure 2C, Table 1). Next, reproducibility was investigated by assessing intra-assay variation (variation of multiple measurements of the same sample in a single test run) and inter-assay variation (variation of multiple measurements of the same

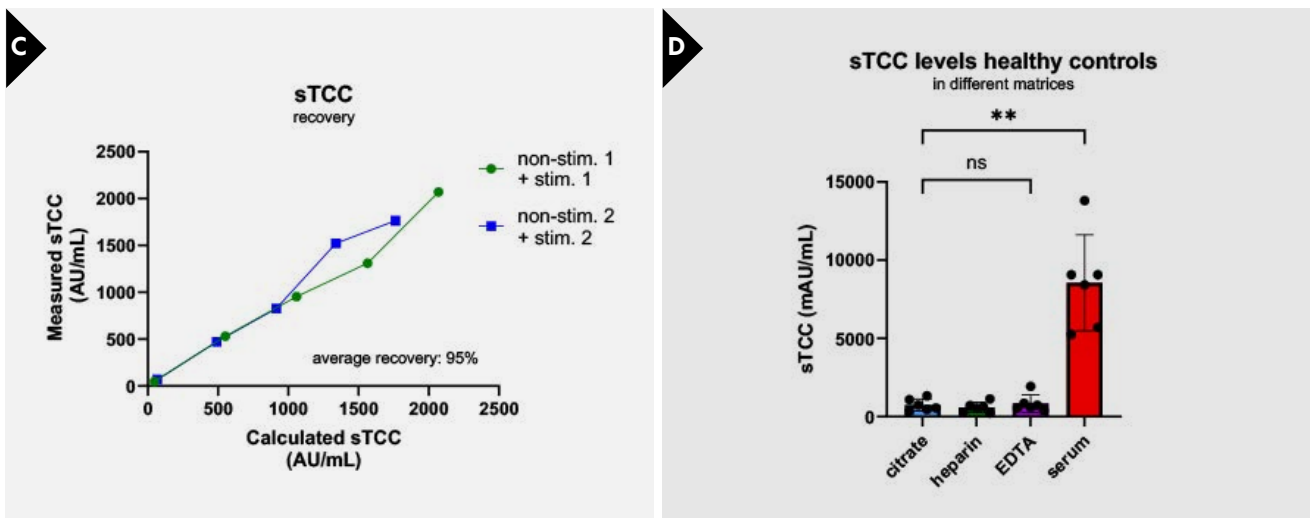
sample in several test runs performed by different operators). Results show low variability with intra-assay %CVs ranging from 3.5–8.3% and inter-assay %CVs from 8.2–13.4%, well within accepted criteria (Table 1). Lastly, batch-to-batch variation over three independently produced batches was evaluated, showing inter-batch variation ranging from 3.1–16.3% (Table 1), highlighting the assay's robustness and reproducibility over production lots.

To assess matrix effects, sTCC concentrations were measured in citrate plasma, heparin plasma, EDTA plasma and serum (CHES panel) from the same individual and compared to each other. In total, six CHES panels were assessed. sTCC concentrations could be accurately determined in both plasma

matrices as in serum. Plasma concentrations did not differ between citrate, heparin, and EDTA samples. However, sTCC concentrations were significantly higher in serum compared to all plasma matrices ($p=0.0011$) (Figure 2D).

Specificity of the assay was verified through cross-reactivity testing, where no measurable signal was detected for purified complement components C5b, C6, C7, C8, or C9, confirming that only the assembled terminal complex is recognized (Table 1). Moreover, species cross-reactivity analysis revealed reactivity for non-human primate, pig, and rabbit samples, while no signal was detected in mouse, rat, or horse plasma, suggesting selective interspecies applicability (Table 1).

Figure 2:



(C) Recovery of sTCC concentrations in serum samples from healthy volunteers. Recovery was investigated by mixing complement-preserved with zymosan-activated samples in different ratio's (100 + 0; 75 + 25; 50 + 50; 25 + 75; 0 + 100). After 30 min of incubation, sTCC concentrations were determined using the HK328. Recovery was calculated as difference between expected and observed.

(D) sTCC concentrations in various sample matrices (citrate, heparin, EDTA plasma, and serum) from six healthy controls.

Table 1.

Overview sTCC ELISA ([HK328](#)) assay characteristics vs. key technical requirements for Bioanalytical Method Validation and Study Sample Analysis ICH M10 guidelines.

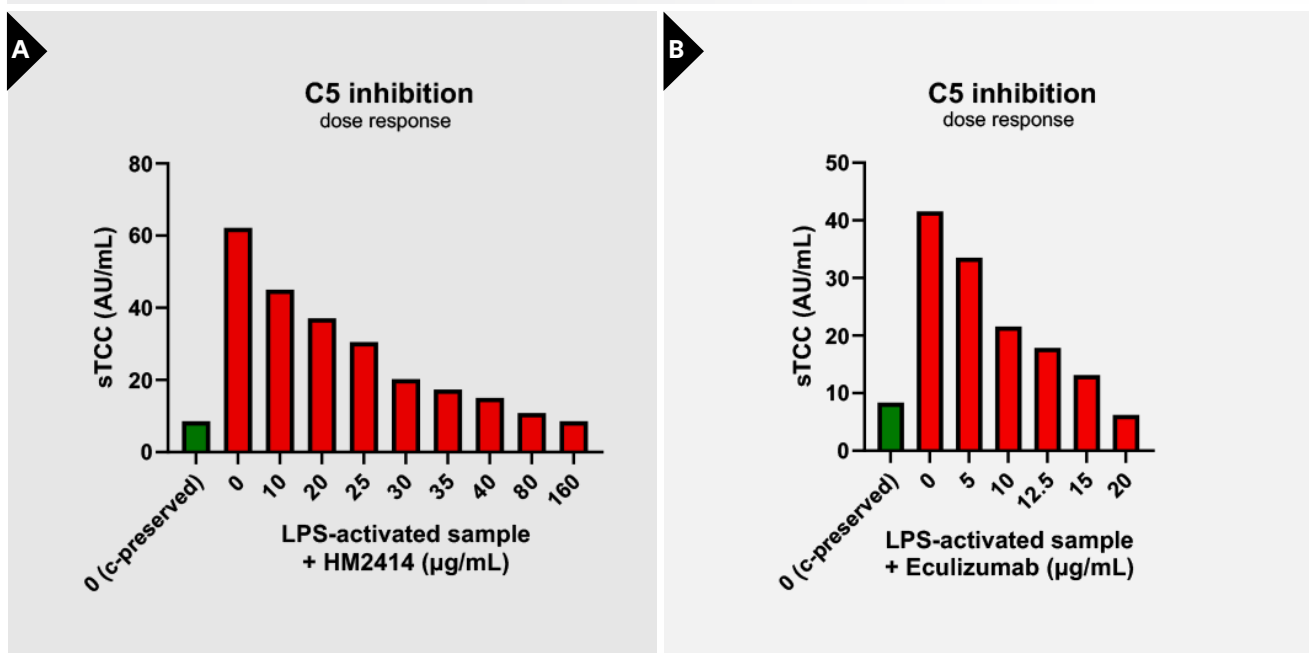
	M10 Guidelines (see also Box 1)	HK328 sTCC ELISA
Standard Curve (calibration)	▶ Minimum points: Typically 6-8 non-zero calibrator concentrations are required, spanning the full dynamic range of the assay. ▶ Replicates: Standards should be run in duplicate or triplicate. ▶ Curve fit models: Use of 4-parameter or 5-parameter logistic regression (4PL/5PL) is standard for nonlinear sigmoidal assays. ▶ Acceptance criteria: Back-calculated concentrations of standards should be within $\pm 15\%$, except at LLOQ and ULOQ ($\pm 20\%$).	▶ 7-points calibrator curve ▶ Replicates: a duplicate is advised and sufficient reagents are provided for Standards samples ▶ 4PL fit is advised and applied during Quality control ▶ Back-calculated concentrations of standards within $<10\%$
Sensitivity	▶ Lower Limit of Quantification (LLOQ): The lowest concentration that can be quantified with acceptable precision and accuracy (usually $CV \leq 20\%$ and accuracy within $\pm 20\%$).	▶ LOD: signal $\geq$ mean blank + 3 SD) ▶ Range: 2000-8.2 AU/mL ▶ LLOQ: 8.2 AU/mL ▶ ULOQ: 2000 AU/mL
Specificity and cross-reactivity	▶ Must demonstrate no significant interference from structurally similar analytes, binding proteins, or common matrix substances (e.g., hemoglobin, lipids, bilirubin). ▶ Spike-in or competitive displacement studies often used to assess this.	▶ No cross-reactivity with proteins C5, C6, C7, C8, C9 observed. ▶ Cross-reactivity other species: non-human primate, pig, rabbit ▶ No differences in sTCC levels observed between the matrices citrate, heparin and EDTA plasma
Precision	▶ Intra-assay variation (repeatability): Run multiple replicates (≥ 5) of several concentrations within a single run. ▶ Inter-assay variation (intermediate precision): Repeat over multiple days, operators, and instruments (usually at least 3 days). ▶ Acceptance criteria: Coefficient of variation (%CV) should be: ▶ $\leq 10-15\%$ for most concentrations ▶ $\leq 20\%$ near LLOQ	▶ Intra-assay (%CV): 3.5-8.3 ▶ Inter-assay (%CV): 8.2-13.4 ▶ Inter-batch (%CV): 3.1-16.3
Accuracy / recovery	▶ Assessed by spiking known amounts of analyte into the matrix and measuring recovery. ▶ Acceptance: Typically 80-120% recovery is acceptable. ▶ Recovery studies also help establish matrix effects and potential interference from serum/plasma/protein binding.	▶ Recovery: 84-118%
Linearity & dilutional linearity	▶ Measured by diluting high-concentration samples and verifying the proportional decrease in signal	▶ Linearity over multiple dilutions $\leq 15\%$ ▶ $R^2=0.9986$
Stability	▶ Evaluate stability of the analyte in the matrix under: ▶ Room temp ▶ Refrigeration ▶ Freeze-thaw cycles ▶ Also validate reagent and calibrator stability.	▶ All components are tested up to 18 months stability. ▶ Standard single use; other assay specific components up to 1 month when stored as described
Sample matrix validation	▶ For each sample type used (e.g., serum vs. plasma), performance must be validated in at least 10 independent donors. ▶ Paired-sample comparisons and spike-recovery studies are used.	▶ No differences in sTCC levels observed between the matrices citrate, heparin and EDTA plasma

Evaluation of sTCC concentrations in samples treated with complement inhibitors

To investigate the applicability of sTCC as a marker to assess complement inhibition, sTCC levels were analyzed in activated serum samples pre-incubated with two C5-targeting inhibitors: [HM2414](#) and Eculizumab. A clear dose-dependent reduction in sTCC formation was observed upon treatment with either inhibitor. Pre-incubation with [HM2414](#) resulted in a gradual and consistent decline in sTCC concentrations,

indicating effective blockade of terminal pathway activation (Figure 3A). Similarly, Eculizumab dose-dependently suppressed sTCC generation (Figure 3B). These findings demonstrate the ability of the sTCC ELISA to sensitively detect pharmacodynamic effects of complement inhibition at the level of C5, supporting its use as a readout for therapeutic efficacy in both preclinical and clinical settings.

Figure 3:



Dose-response of sTCC levels in a complement-activated sample pre-incubated with **(A)** [HM2414](#) and **(B)** Eculizumab, two complement inhibitors targeting C5.

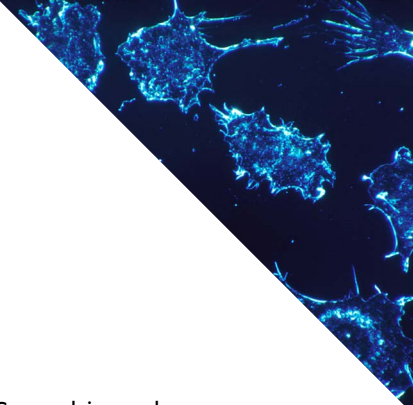


► Discussion

The aim of the current study was to develop and validate a robust and specific immunoassay for the quantification of sTCC in human plasma and serum, enabling reliable assessment of terminal complement activation under physiological conditions. In summary, these data demonstrate that the sTCC ELISA is a reliable, precise, and highly specific tool for measuring sTCC levels in human plasma and serum, thereby enabling robust detection of terminal complement activation for research and clinical support applications according to ICH M10 guidelines (see also Box 1). To facilitate comparability with international datasets and to enable inter-laboratory comparability, the assay reports sTCC levels in arbitrary units (AU) rather than absolute concentrations. This approach not only aligns with the practices of the complement standardization group but also addresses the inherent complexity of the sTCC complex, whose variable stoichiometry and lack of a defined molecular standard limit the feasibility of accurate mass-based quantification. Use of AU, calibrated against a well-characterized internal reference, ensures consistency, assay robustness, and broader applicability across laboratories and study settings. This strategy also supports the development of international reference materials and harmonized validation reporting formats, which are critical for regulatory acceptance and broader clinical implementation (see also Box 1). With regard to its potential as a biomarker, the clinical and translational relevance of sTCC quantification has been extensively explored across a broad spectrum of complement-mediated diseases and pathological conditions. Numerous studies reported that quantification of sTCC offers significant added value as a biomarker for complement activation, particularly

in the context of disease onset, severity, and therapeutic response (18, 22, 23). As sTCC levels reflect the culmination of complement activation, thereby providing an integrated readout of terminal pathway activity, it serves as sensitive marker for identifying acute complement-mediated pathology and stratifying disease severity in conditions such as neuromyelitis optica spectrum disorders (NMOSD) (23), atypical hemolytic uremic syndrome (aHUS) (17), and transplant rejection (18).

In addition, sTCC quantification also offers significant added value for assessing complement activity, in relation to therapeutic response and disease monitoring in patients receiving complement inhibitors (18, 22, 23). Dynamic changes in sTCC levels can be used to track pharmacologic effects during treatment with C5-targeting inhibitors such as eculizumab. As shown in the present study, dose-dependent reductions in sTCC concentrations upon inhibitor administration underscore its relevance for therapeutic monitoring and guiding dose adjustments (24). Beyond its role in monitoring patient response to approved complement inhibitors, sTCC also serves as a valuable marker for the assessment of novel complement-targeting therapies under development. The sTCC assay provides a robust and quantitative platform to evaluate the pharmacodynamic efficacy of novel complement inhibitors under development. By enabling sensitive monitoring of sTCC formation, the assay facilitates assessment of the extent and kinetics of terminal pathway inhibition in response to candidate compounds. This is essential for determining optimal dosing strategies, evaluating target engagement, and establishing proof-of-mechanism in early-phase studies.



In compound screening programs, the assay can be used to compare the potency of multiple inhibitors by quantifying sTCC levels in activated serum or plasma samples treated with increasing drug concentrations. Moreover, in studies using patient-derived samples, the assay supports evaluation of compound efficacy in relevant biological matrices, helping to translate in vitro findings to clinically meaningful outcomes. The assay is also well-suited for assessing adverse events (AEs) linked to unintended complement activation, either due to investigational drug mechanisms or off-target effects, thus supporting safety profiling and risk mitigation strategies early in development.

Importantly, the inclusion of sTCC as a biomarker has become increasingly common in clinical trials evaluating complement inhibitors. In some trials, sTCC serves as a critical diagnostic or stratification marker, such as in thrombotic microangiopathy (TMA; NCT03518203) and C3 glomerulopathy/aHUS (NCT05086744). In other studies, such as those on paroxysmal nocturnal hemoglobinuria (PNH; NCT03459443), systemic sclerosis (NCT03124368), and lupus nephritis, sTCC is included as an exploratory or secondary endpoint, reflecting its utility in capturing pharmacodynamic responses and supporting mechanistic insights.

► Concluding remarks

The sTCC ELISA provides a robust and specific tool for the quantification of sTCC in human plasma and serum, enabling reliable assessment of terminal complement activation. By reporting results in AU, the assay aligns with international standardization practices and accommodates the structural complexity of the sTCC complex. Beyond analytical performance, the assay demonstrates clear clinical and translational value. It supports the use of sTCC as a biomarker for disease severity and progression in

complement-mediated conditions, and enables sensitive monitoring of complement inhibitor therapies. Additionally, it offers a practical tool for evaluating drug efficacy and detecting off-target complement activation during early-phase drug development. Together, these characteristics make the sTCC ELISA a robust and well-validated tool for supporting fundamental and translational research, diagnostic applications, and complement-targeted drug development.

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