Haemoview Diagnostics
Redefining coagulation

CUSTOMER INFORMATION BRIEF

Interpreting Low ML in ClotPro® Viscoelastometry

Summary of Smudla et al., Diagnostics 2026

Why this matters

Viscoelastometric testing (VET) is widely used to guide haemostatic therapy in trauma, sepsis, surgery, and other critical care settings. Maximum lysis (ML) is often interpreted as a measure of fibrinolysis, and low ML values have been linked to outcomes in trauma and sepsis populations. However, the biological basis of low ML in tissue factor-triggered VET assays has not been clearly established.

Smudla et al. asked whether low ML in ClotPro® TF-activated assays reflects **plasmin-mediated fibrinolysis** – a question with direct implications for how clinicians label patients with "fibrinolysis shutdown" and how they choose adjunctive therapies.

Study at a glance

Study design	Cross-sectional methodological comparison in healthy volunteers
Participants	120 healthy adults; 52.5% female; mean age 38.2 ± 14.1 years
Platform	ClotPro® system with APIRO Diagnostics assays and consumables
Comparison	Tissue factor-activated EX-test (no fibrinolysis inhibition) vs AP-test (with tranexamic acid to inhibit fibrinolysis)
Primary question	Does low maximum lysis (ML) in TF-activated viscoelastometry reflect plasmin-mediated fibrinolysis?
Source	Smudla A, Schöchl H, Calatzis A, Csíkós R G, Fazakas J. Diagnostics 2026, 16(10), 1472.

What was tested

- **EX-test** – tissue factor-activated assay without fibrinolysis inhibition; standard VET measurement of clot formation and lysis.
- **AP-test** – the same TF activation with tranexamic acid added to pharmacologically inhibit fibrinolysis. Differences between EX and AP isolate the plasmin-mediated contribution.
- CA10 (clot amplitude at 10 minutes), MCF (maximum clot firmness), and ML (maximum lysis) were compared between assays in 120 healthy adults.

Key findings

- ML values ranged from 1–13% in both assays, with nearly identical means (5.9 ± 2.6% in EX-test vs 6.0 ± 2.6% in AP-test).
- Pharmacological inhibition of fibrinolysis with tranexamic acid did not reduce ML in the TF-triggered assay.
- CA10 was similar with and without fibrinolysis inhibition; MCF showed a statistically detectable but clinically negligible difference.
- Bland–Altman analysis showed minimal bias and strong agreement across CA10, MCF, and ML.
- Authors' conclusion: low ML (≤13%) in TF-triggered viscoelastometry does not reflect plasmin-mediated fibrin degradation, and likely reflects alternative mechanisms – particularly platelet-driven clot retraction.

EX-test vs AP-test results in 120 healthy adults

Parameter	EX-test (TF-activated, no inhibitor)	AP-test (TF-activated + tranexamic acid)
CA10	Similar	Similar
MCF	Slight difference†	Slight difference†
ML range	1–13%	1–13%
ML mean ± SD	5.9 ± 2.6%	6.0 ± 2.6%
Agreement (CA10, MCF, ML)	Strong correlation; minimal Bland–Altman bias for ML	—

† Difference in MCF was statistically detectable but clinically negligible. Source: Smudla et al., Diagnostics 2026.

Practical implications for critical care, trauma, sepsis, and surgery

- Low ML values below the reference range should not, on their own, be equated with "fibrinolysis shutdown".
- Interpret low ML cautiously in the broader clinical context; consider platelet-driven clot retraction as a contributor in healthy and stable patients.
- Where fibrinolytic capacity is the clinical question, consider assays that directly probe the fibrinolytic system, such as a tPA-challenge assay.
- Decisions about antifibrinolytic therapy (e.g. tranexamic acid) should integrate clinical context, mechanism of injury, and complementary testing – not low ML alone.

Important clinical considerations

- Population was healthy volunteers; results may differ in critically ill, bleeding, or coagulopathic patients.
- Findings are restricted to the ClotPro® platform with the specific TF-triggered assays studied.
- Clot retraction and biochemical fibrinolysis markers were not measured directly; the role of clot retraction is inferential.
- Interpretation should follow the device and assay instructions for use, with attention to local reference ranges and validated clinical pathways.

Suggested customer conversation points

- How is low ML currently interpreted and acted on in your trauma, sepsis, or surgical protocols?
- Does your service distinguish between low ML and a directly demonstrated fibrinolytic deficit when considering antifibrinolytic therapy?
- Would access to a tPA-challenge assay change how your team evaluates suspected fibrinolysis shutdown?
- What educational or pathway updates would be useful for clinicians using ML as part of their VET interpretation?

Source

Smudla A, Schöchl H, Calatzis A, Csíkós R G, Fazakas J. *Is "physiological lysis" in viscoelastometry a plasmin-mediated process?* Diagnostics. 2026;16(10):1472.

Article: https://www.mdpi.com/2075-4418/16/10/1472 | **DOI:** https://doi.org/10.3390/diagnostics16101472

Visual summary

At-a-glance summary of the question "Is physiological lysis really plasmin-mediated fibrinolysis?" – reproduced from materials accompanying Smudla et al., Diagnostics 2026.

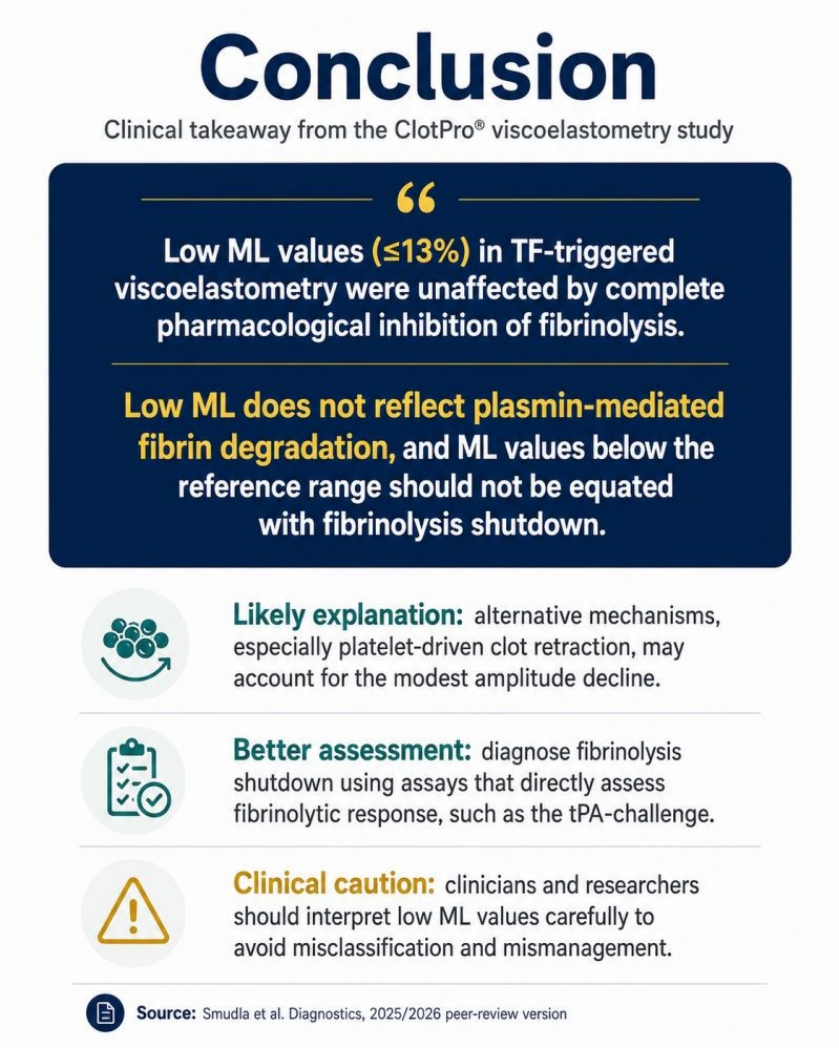

Figure: Educational visual summary. Adapted from materials accompanying the source publication.

Source and disclaimer

Smudla et al. Diagnostics 2026;16(10):1472. **Article:** https://www.mdpi.com/2075-4418/16/10/1472 | **DOI:** https://doi.org/10.3390/diagnostics16101472