Pfizer Shot Linked to 111% Rise in Blood Clot Formation Marker and 39% Rise in Protein That Slows Blood Clot Breakdown: Journal 'Biomedicine & Pharmacotherapy'

Study also reports roughly 450% increase in inflammatory protein IL-6 and 42% rise in a blood marker of body-wide inflammation.

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A study published September 29, 2026, in *Biomedicine & Pharmacotherapy* by researchers at the University of Milan, IRCCS MultiMedica, and other Italian institutions documents physical and chemical changes in the blood of healthy adults following Pfizer COVID-19 vaccination, implicating both blood clot formation and the body's ability to dissolve blood clots.

Pfizer's own safety data reports hundreds of serious blood clot and clot-related stroke cases following vaccination, including dozens of deaths, though as voluntary reports they cannot show how often clots truly occur.

Using electron microscopy and biochemical testing, the researchers documented four notable increases (approximated from Figure 2, seen below):

- **111% increase** (**2.11-fold**) in a structural marker of platelet activation associated with blood clot formation.
- **39% increase** (**1.39-fold**) in a protein that inhibits the body's ability to dissolve blood clots.
- **450% increase** (**5.5-fold**) in an inflammatory protein (IL-6).
- **42% increase** (**1.42-fold**) in a marker of systemic inflammation.

The findings raise a two-sided blood clotting concern: the injection may simultaneously promote conditions conducive to blood clot formation while interfering with the body's mechanism for breaking down blood clots.

Did Pfizer, BioNTech, or the U.S. Food and Drug Administration (FDA) characterize these simultaneous biological changes before the injection was authorized for mass administration?

Were recipients informed of them?

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Transient ultrastructural remodeling of the open canalicular system and α-granules is associated with endothelial responses after BNT162b2 vaccination in healthy volunteers

Valentina Carlini[a], Emilio Trabucchi[b], Nadia Santo[c], Norma Lattuada[c], Stefania Saccucci[c], Raffaele Allevi[d], Francesca Rispoli[a], Barbara Bianchi[a], Ermanno Longhi[a], Maria Belen Macas Granizo[e], Marco D'Anzeo[f], Cesare Berra[a], Angelo Naselli[g], Alberto Settembrini[e], Elena Tagliabue[a], Elena Vianello[c,a], Elena Dozio[c,a], Massimiliano Corsi Romanelli[c,h], Antonio E. Pontiroli[f], Martino Bolognesi[c], Lucia La Sala[a,c,i,j,*]

[a] IRCCS MultiMedica, Via Fantoli 16/15, Milan 20138, Italy
[b] Fondazione MultiMedica ETS, Via Fantoli 16/15, Milan 20138, Italy
[c] UNITECH NOLAMITO – University Technological Platform, University of Milan, Via Gadzi 19, Milan 20133, Italy
[d] University of Milan, Dept of Biomedical Sciences and Clinic, Milan 20157, Italy
[e] University of Milan, Dept of Biomedical Sciences for Health, Milan 20133, Italy
[f] ASST della Brianza, SOD Medicine & Laboratory, Ancona 60500, Italy
[g] Experimental Laboratory for Research on Organ Damage Biomarkers, IRCCS Istituto Auxologico Italiano, Milan Italy
[h] Department of clinical and experimental pathology, IRCCS Istituto Auxologico Italiano, Milan Italy
[i] University of Milan, Dept of Health Science, Milan 20146, Italy

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ABSTRACT

Platelets are key coordinators of hemostatic and immune functions, rapidly responding to vascular perturbations. However, the ultrastructural signatures of platelet priming, including open canalicular system (OCS) remodeling and α-granule (αG) changes, remain poorly characterized. BNT162b2 vaccination induces a well-defined, self-limiting immune activation in healthy individuals, providing a controlled human model to investigate platelets and vascular responses. Using this model, we longitudinally investigated platelet ultrastructure, assessing alterations in OCS architecture and αG content and their association with circulating inflammatory and endothelial activation biomarkers. Healthy adults were evaluated at six time points across two BNT162b2 vaccine doses. Platelets were analyzed by TEM to quantify αG number and OCS area in a participant subcohort. Circulating levels of IL-6, CD40L, P-selectin and plasminogen activator inhibitor-1 (PAI-1) were measured by ELISA in the full cohort. Human umbilical vein endothelial cells (HUVECs) were stimulated with post-vaccination plasma to assess endothelial responses. Vaccination induced transient OCS expansion and αG depletion, accompanied by transient systemic immune activation, as reflected by changes in circulating IL-6, CD40L, and PAI-1, whereas P-selectin showed no sustained increase, consistent with a primed/intermediate activation state. Post-vaccination plasma induced dynamic changes in HUVECs IL-18s, MIP-3α, and IL-22 secretion, adhesion molecules and chemokine transcription, and 14–3–3ζ, F2YT2, PAI-1, and ADAMTS13 signaling, consistent with transient endothelial activation. BNT162b2 vaccination thus elicits coordinated, non-pathological platelet remodeling alongside transient systemic and endothelial responses. OCS and αG may represent sensitive ultrastructural indicators of transient platelet activation, defining a novel morpho-functional signature of vaccine-induced immune activation at the vascular interface.

* Correspondence to: Dept of Biomedical Sciences for Health, Via Mangiagalli 31, Milan 20133, Italy.
E-mail address: lucia.lasala@unimi.it (L. La Sala).
[1] The authors contributed equally
[2] IRCCS MultiMedica, PST Via Fantoli 20138, Milan (Italy).

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Blood Clot Formation & Dissolution

Following the first Pfizer injection, researchers documented two overlapping changes in human blood: one associated with blood clot formation and another associated with inhibition of blood clot breakdown.

Using an electron microscope, the researchers physically examined platelets (the components of human blood responsible for forming clots).

They documented an approximately **111% increase** in the relative area occupied by the platelets' internal channels, peaking two weeks after the first injection.

The researchers also observed depletion of the platelets' internal storage compartments containing proteins involved in blood clotting and inflammation.

The authors describe these physical changes as potential "indicators of transient platelet activation."

Meaning: the platelets exhibited structural changes consistent with entering an active state associated with blood clot formation.

At the same time, blood concentrations of plasminogen activator inhibitor-1 (PAI-1), a protein that inhibits the body's ability to dissolve blood clots, increased approximately **39%**, peaking one week after the first injection and remaining elevated two weeks afterward.

The researchers explicitly identify PAI-1 as a "pro-thrombotic mediator acting as a fibrinolysis inhibitor."

Meaning: a substance that favors the persistence of blood clots by inhibiting their breakdown.

Taken together, the findings suggest the Pfizer injection may simultaneously create conditions that favor blood clot formation and make blood clots harder for the body to dissolve.

How extensively did Pfizer and federal regulators investigate this combined effect before authorizing the injection for mass administration, and what were recipients told?

Fig. 1. Ultrastructural remodeling of the open canalicular system (OCS) and α-granules (αG) in circulating platelets following BNT162b2 vaccination. Representative transmission electron microscopy (TEM) images of circulating platelets collected at baseline (T0), after the first vaccine dose (T1–T3), and following the second dose (T4–T5). Platelets show a transient OCS expansion (**red asterisks**) and a concomitant reduction in αG number (**red arrows**) at early time points (T1–T3), returning to baseline morphology by T5. Legend: Arrows, alpha-granules; *, open canalicular system (OCS); m, mitochondria; scale bar = 1 μm.

Electron microscopy images of human blood platelets before and after Pfizer COVID-19 vaccination show temporary expansion of internal platelet channels (red asterisks) and depletion of protein storage compartments (red arrows)—physical changes indicative of platelet activation, a process involved in blood clot formation. (Source: *Biomedicine & Pharmacotherapy*, Figure 1, 2026.)

Fig. 2. Longitudinal analysis of platelet during BNT162b2 mRNA vaccination. A–B. Quantitative analysis of OCS area (left) and αG count per platelet (right) at each time point (T0–T5). Data represent mean ± SEM; 18 platelets/donors/time point were analyzed (n = 13 donors). A transient increase in OCS area and a parallel decrease in αG number were observed after the first dose, consistent with a non-degranulating primed platelet phenotype. Differences in continuous variables between time points were evaluated by mixed models for repeated-measures, using SAS software (Version 9.4; SAS Institute Inc., Cary, NC, USA) procedures PROC MIXED. Legend: * vs T0, § vs T1, ° vs T2, # vs T3, ^ vs T4; $p < 0.05$, $p < 0.01$, $p < 0.001$, $p < 0.0001$. **C.** Platelet count ($10^3/\mu L$) was measured in 46 healthy, SARS-CoV-2–naïve individuals at six time points spanning the two-dose BNT162b2 vaccination schedule (T0–T5). Platelet count was within the range of normality (blue light area) defined by range 150–400 × $10^3/L$. In T1 and T4, corresponding at the first and second doses of vaccine, count is significantly reduced compared to pre-vaccination baseline (T0 = no vaccination). A transient decrease in platelet number was observed after the first dose (T1–T2), followed by recovery and stabilization post-second dose (T4–T5). Data were expressed as mean ± SEM. Statistical comparisons were performed using repeated-measures ANOVA with post hoc correction; symbols indicate significance between time points as specified in the figure. Differences between time points were evaluated by mixed models for repeated-measures, using SAS software (Version 9.4; SAS Institute Inc., Cary, NC, USA) procedures PROC MIXED. Sample size: n = 44 T0; n = 39 T1; n = 37 T2; n = 38 T3; n = 29 T4; n = 31 T5. Legend: * vs T0, § vs T1, ° vs T2, # vs T3, ^ vs T4. $p < 0.05$, $p < 0.01$, $p < 0.001$, $p < 0.0001$. **D-I. Systemic markers of inflammation and platelet activation following BNT162b2 vaccination.** Circulating levels of P-selectin (D), IL-6 (G), soluble CD40L (H) and PAI-1(I) were measured by ELISA at six time points during the two-dose BNT162b2 mRNA vaccination schedule (T0–T5). Data are presented as mean ± SEM. Statistical comparisons among time points were performed using repeated-measures ANOVA with post hoc testing; significance is indicated in the figure. **D.** Serum concentration of soluble P-selectin (pg/mL), a marker of platelet activation and aggregation, was evaluated during immunization time points (n = 18 T0; n = 12 T1; n = 11 T2; n = 11 T3; n = 16 T4; n = 11 T5). P-selectin levels were strongly reduced following booster administration ($p < 0.05$ vs T1). **E.** Neutrophil to lymphocyte ratio (NRL) significantly increased at the first ($p < 0.0001$) and second dose ($p < 0.0001$) compared with baseline. **F.** Platelet to lymphocyte ratio (PLR) showed a tendency to progressively increase over time, reaching a peak concomitant with the second vaccine dose ($p < 0.0001$ vs all other time points). The ratio significantly decreased 1 week after booster administration ($p < 0.0001$ vs T4). Differences among time points were evaluated by mixed-effects models for repeated-measures, using SAS software (Version 9.4; SAS Institute Inc., Cary, NC, USA) procedures PROC MIXED. Sample size: n = 44 T0; n = 39 T1; n = 37 T2; n = 38 T3; n = 29 T4; n = 31 T5. **G.** Serum concentration of the inflammatory marker interleukin-6 (IL-6, pg/dL; n = 18 T0; n = 12 T1; n = 11 T2; n = 16 T3; n = 11 T4; n = 18 T5) significantly increased after the first ($p < 0.01$) and second vaccine doses ($p < 0.0001$). After the booster, IL-6 amounts decreased significantly ($p < 0.0001$ vs T4). **H.** Serum concentration of soluble CD40 ligand (sCD40L, pg/mL), a marker of platelet activation, was evaluated during the immunization time points (n = 8 T0; n = 7 T1; n = 6 T2; n = 6 T3; n = 6 T4; n = 6 T5). **I.** Serum concentration of plasminogen-activator inhibitor-1 (PAI-1, pg/mL), a platelet-associated pro-thrombotic mediator acting as a fibrinolysis inhibitor, was measured during immunization schedule (n = 18 T0; n = 12 T1; n = 11 T2; n = 16 T3; n = 11 T4; n = 18 T5). Differences between time points were evaluated by mixed-effects models for repeated-measures, using SAS software (Version 9.4; SAS Institute Inc., Cary, NC, USA) procedures PROC MIXED. Legend: * vs T0, § vs T1, ° vs T2, # vs T3, ^ vs T4; $p < 0.05$, $p < 0.01$, $p < 0.001$, $p < 0.0001$ respectively.

Measurements of human blood following Pfizer COVID-19 vaccination show an approximately 111% increase in a structural marker of platelet activation (A), depletion of platelet protein storage compartments (B), a 42% increase in a marker of systemic inflammation (F), a 450% increase in inflammatory protein IL-6 (G), and a 39% increase in a protein that inhibits blood clot dissolution (I). (Source: *Biomedicine & Pharmacotherapy*, Figure 2, 2026.)

Inflammatory Protein Surges 450%

Following the second Pfizer injection, blood concentrations of interleukin-6 (IL-6), a protein that promotes inflammation, increased approximately **450%**, reaching 5.5 times their pre-vaccination levels.

Researchers also documented an approximately **42% increase** in the platelet-to-lymphocyte ratio (a blood-based indicator of inflammation throughout the body).

In a separate laboratory experiment, the researchers exposed human cells that form the inner lining of blood vessels to plasma taken from vaccinated participants.

That plasma altered the production of substances involved in regulating inflammation and blood clotting.

In other words, the changes observed following vaccination extended beyond the blood itself: plasma collected from vaccinated individuals also altered the behavior of human blood vessel cells in laboratory experiments.

Authors Declare Changes 'Non-Pathological' After Just Five Weeks of Monitoring

Despite documenting overlapping increases in markers associated with blood clot formation, inhibition of blood clot dissolution, and systemic inflammation, the authors characterize the platelet changes as "non-pathological" and the immune activation as "self-limiting."

But the researchers explicitly acknowledge that "the follow-up was limited to only 5 weeks after the first vaccine administration and direct platelet functional assays were absent."

Meaning:

- Blood collection ended just seven days after the second injection.
- The researchers did not directly test platelet function.
- The researchers did not directly measure the body's ability to dissolve blood clots.
- The researchers did not establish the long-term clinical consequences of the observed changes.

Moreover, blood concentrations of the protein that inhibits blood clot dissolution remained numerically above pre-vaccination levels when blood collection ended (Figure 2I).

On what evidentiary basis can these changes be characterized as harmless when their functional and long-term clinical consequences were not directly established?

A biological disturbance does not have to be permanent to cause injury.

Bottom Line

Following Pfizer COVID-19 vaccination, researchers documented a **111% increase** in a structural marker associated with blood clot formation, a **39% increase** in a protein that inhibits blood clot dissolution, a **450% increase** in an inflammatory protein, and a **42% increase** in a marker of systemic inflammation.

Yet the researchers characterized the platelet changes as "non-pathological" despite ending blood collection just seven days after the second injection and acknowledging the absence of direct platelet function testing.

What did Pfizer and federal regulators know about these overlapping biological changes before mass administration, and how could recipients provide fully informed consent if their clinical consequences had not been established?

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