

Nattokinase Dissolves Fibrinoid Microclots

Preclinical Study Shows Dose-Dependent Effect, Offers Hope

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APR 18, 2024

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Post-acute sequelae after SARS-CoV-2 infection (long COVID) and after COVID-19 vaccination are characterized by micro blood clotting. The work of [Scheim et al](#) suggests the majority of syndromes in both cases are due to Spike protein mediated hemagglutination and then the development of small clots that serve the major organs in the body. [Xi et al](#) demonstrated increased risk for microclots visualized in retinal arteries and veins in the COVID-19 vaccinated.

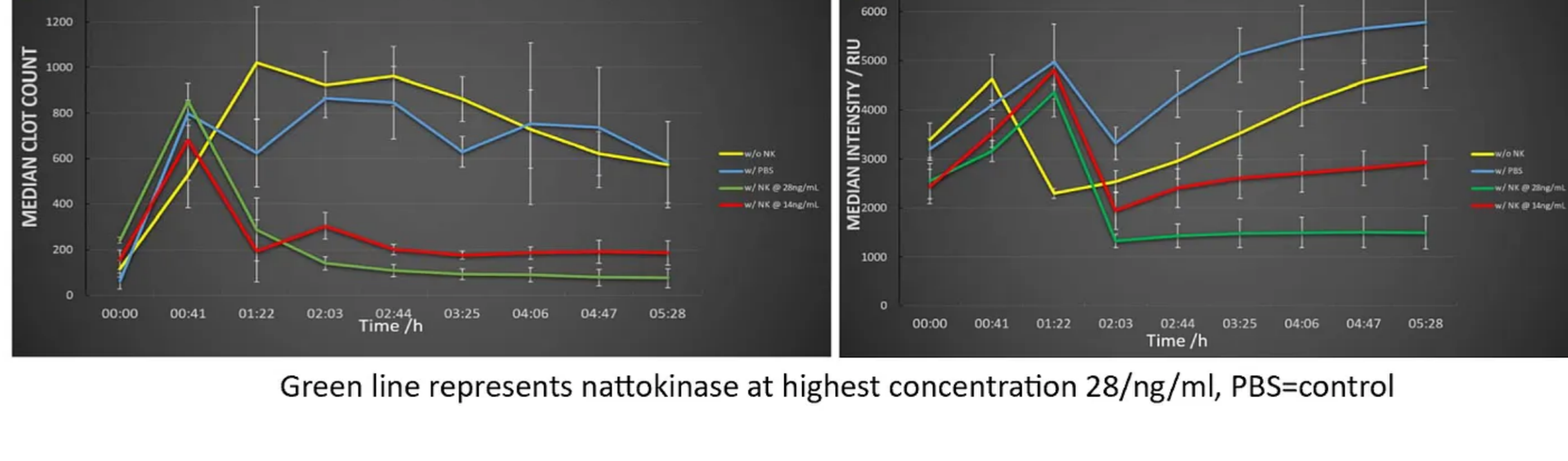
[Grixti et al](#) point out that nattokinase has quickly become part of community standard of care for post-acute sequelae as proposed in [McCullough Protocol Base Spike Detoxification](#). They went on to demonstrate that recombinant Nattokinase was fibrinolytic in a lab preparation of fibrinoid microclots, that is coagulation that is initiated with fibrinogen, thrombin, and lipopolysaccharide.

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Automated microscopic measurement of fibrinoid microclots and their degradation by nattokinase, the main natto protease

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Abstract: Nattokinase, from the Japanese fermented food natto, is a protease with fibrinolytic activity that can thus degrade conventional blood clots. In some cases, however, including in Long COVID, fibrinogen can polymerise into an anomalous amyloid form to create clots that are resistant to normal fibrinolysis and that we refer to as fibrinoid microclots. These can be detected with the fluorogenic stain thioflavin T. We describe an automated microscopic technique for the quantification of fibrinoid microdot formation, which also allows the kinetics of their formation and aggregation to be recorded. **We also here show that recombinant nattokinase is effective at degrading the fibrinoid microclots *in vitro*.** This adds to the otherwise largely anecdotal evidence, that we review, that nattokinase might be anticipated to have value as part of therapeutic treatments for individuals with Long COVID and related disorders that involve fibrinoid microclots.



doi: <https://doi.org/10.1101/2024.04.06.588397>

As you can see, the effect was dose-related. This suggests even greater biological plausibility that Nattokinase can indeed dissolve microclots. The next sets of experiments should test microclots induced by Spike protein, thrombin, and fibrinogen. The clinical community has a long way to go in translating these results from bench to bedside. There is a great need for dose-ranging studies with Nattokinase in humans to study fibrinolysis and risks of bleeding. In the meantime these data are reassuring that we are on the right track with Nattokinase broadly, empirically used in patients with post-acute sequelae after SARS-CoV-2 infection (long COVID) and after COVID-19 vaccination.

Cureus Open Access Review Article DOI: 10.7759/cureus.49204 EUIPO #018948436 Reg Feb 24 2024

Clinical Approach to Post-acute Sequelae After COVID-19 Infection and Vaccination

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Review began 11/09/2023
Review ended 11/15/2023
Published 11/21/2023
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FIGURE 1: Proposed Mechanisms Through Which S1 of Spike Protein Induced Cardiac Mitochondrial Dysfunction, Which Leads to Cardiac Injury in COVID-19 Patients.
Spike protein is the glycoprotein protein that covers the surface of SARS-CoV-2 and binds to the host ACE2 receptor to mediate the viral cell entry. It is composed of S1 and S2 subunits that are responsible for ACE2 binding and membrane fusion, respectively. S1 readily binds to ACE2 on the ACE2 membrane and is then internalized into the cytosol and localized in organelles, such as mitochondria, which induces the transitory increase in fatty acids transport and uptake for lipogenesis, fatty acid oxidation, and peroxisome biogenesis, thereby impairing mitochondrial function and promoting ROS production. In turn, ROS further exacerbates mitochondrial dysfunction and mitochondrial fragmentation. Moreover, S1 causes downregulation of TOM20; this effect might inhibit the pathways leading to mitochondrial biogenesis.

FIGURE 3: McCullough Protocol: Base Spike Detoxification (BSD)™.
A: Dissolution of spike protein-induced thrombus. Nattokinase directly degrades fibrinolytic-resistant fibrin (from spike protein) and thrombin-activated fibrinogen. B: Inhibition of spike protein via ACE2 receptors. Bromelain and curcumin block the ACE2 receptor, preventing spike protein from binding. C: Protonic degradation of spike protein. Nattokinase and bromelain degrade spike protein, rendering them inactive. D: Attenuation of spike protein-induced inflammation. Bromelain and curcumin downregulate the NF-κB signaling pathway induced by spike protein, leading to the suppression of inflammatory mediators. E: BSD treatment protocol. The full treatment regimen and the addition of other compounds based on clinical indication are illustrated.

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Hulscher N, Procter BC, Wynn C, McCullough PA. Clinical Approach to Post-acute Sequelae After COVID-19 Infection and Vaccination. *Cureus*. 2023 Nov 21;15(11):e49204. doi: 10.7759/cureus.49204. PMID: 38024037; PMCID: PMC10663976.

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Thank you for your great work and heroism
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Great article and gives hope. However, I have heard (somewhere) that our government (Durbin from Chi) is on the prowl to restrict natto, natural cures, natural cures, including vitamins - and NATTO.
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