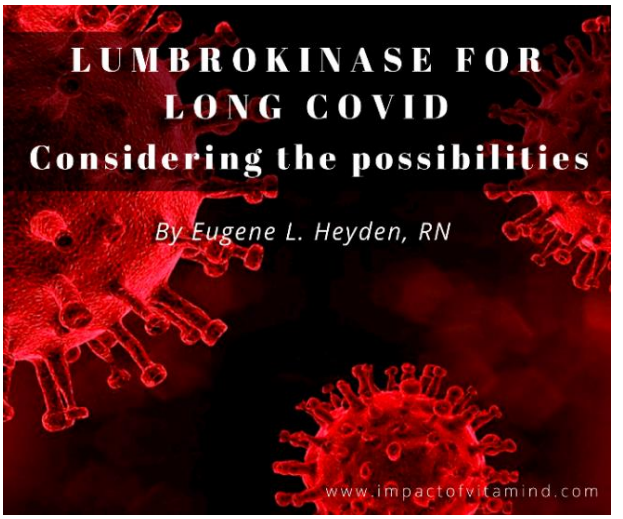


Lumbrokinase for Long COVID: Considering the possibilities

By Eugene L. Heyden, RN

Introduction

> *Studies have shown that long covid can affect the whole spectrum of people with covid-19, from those with very mild acute disease to the most severe forms.*
>
> *The symptoms of long covid include fatigue, dyspnea, cardiac abnormalities, cognitive impairment, sleep disturbances, symptoms of posttraumatic stress disorder, muscle pain, concentration problems, and headache.* **~Crook et al., 2021**

In a related article, I shared with you two procedures for Long COVID that may make it all go away, and rather quickly—namely, H.E.L.P. apheresis and Therapeutic Plasma Exchange. Unfortunately, both procedures are not generally

offered to treat Long COVID, are too expensive for the average person to afford, and are not readily available. But there may be an alternative way to approach the fundamental problem in Long covid. There is a unique dietary supplement that just might do the trick. Let's explore the possibilities. But first, there are a few things we need to get out of the way.

How you got to where you are today

> *Severe SARS-CoV-2 infection mostly presents with coagulation abnormalities, pulmonary microvascular thrombosis, and severe inflammatory response.* **∼Fard et al., 2021)**

> *Coagulopathies [coagulation abnormalities], and especially the formation of* ***extensive microclots*** *in vivo, are* ***a hallmark of both COVID and long COVID****, and we have demonstrated that these microclots too are amyloid in character.* **∼Kell et al., 2022, emphasis added**

Very briefly, a couple of years ago a novel respiratory virus, SARS-CoV-2, arose from Wuhan China, rapidly spread across the globe to eventually kill millions. Somehow it found you. Somehow it infected you. Thankfully, you did not perish. You survived. But things, they have never been the same. Something unseemly is going on behind the scenes, it is not pretty, and it is just not going away.

As it turns out, what is going on is rather simple. You have a clotting disorder, one that impairs tissue oxygenation (Kell, et al., 2022; Wang et al., 2022). And this clotting disorder exists because of the unique way the body responds to a certain portion of the virus called the spike protein—the wiggly little things that project from the body of the virus and are used to achieve attachment to a target cell. Would you like to see what the virus looks like? Check it out! https://rb.gy/golxfs

Now, back to our story.

One intriguing way the body defends against an advancing respiratory virus is to utilize a sophisticated defense mechanism known as **immunothrombosis**. Immunothrombosis defends against viral penetration into the systemic circulation by clot formation (AKA thrombus formation) at the point of entry—occurring within the capillary bed, most notably in the lungs. Apparently, the

spike protein is the trigger for such clotting to take place (Kell et al., 2022), and a heroic effort is put forth to freeze the virus in place. Although well-intentioned, this can occur extensively and lead to noticeably impaired gas exchange in the lungs and reduced oxygenation of tissues and organs. And this leads to a variety of symptoms. Did you know, immunothrombosis is one of the things you experienced as you were battling COVID? Are you following me? It's not all that hard, is it? Well, things do get a little more complex.

In immunothrombosis, the coagulation system, along with a cell called the platelet (which can engulf the virus to isolate and disable it)—in addition to a variety of clotting factors—all act together as a defense mechanism. Even the disturbed vascular wall lining, called the endothelium, becomes intimately involved, signaling that clot formation would be a very good idea. Surprisingly, clot formation is not exclusive to the acute COVID-19 infection, it is occurring in Long COVID as well. Indeed, we should view Long COVID is a clotting disorder.

> *Ongoing vascular endothelial damage promotes platelet adhesion and coagulation, resulting in the impairment of various organ functions. Meanwhile, thrombosis will further aggravate vasculitis contributing to further deterioration. Thus,* ***long COVID is essentially a thrombotic sequela****.* (Wang et al., 2022, emphasis added)

Contemplating its very essence, it is easy to see why Long COVID expresses itself in a variety of ways. Clotting, occurring within the capillary bed, leading to impaired tissue oxygenation, wherever it occurs, explains the persistent fatigue, the brain fog, the cognitive impairment, the shortness of breath, that are the hallmarks of Long COVID. Surprisingly, Long COVID remains long because the clots keep on forming. And they keep forming because the trigger does not easily or quickly go away (Kell et al., 2022, emphasis added).

Recall, spike protein exposure is a trigger for defensive clotting. And with respect to Long COVID, ***"There is now considerable evidence for the persistence of SARS-CoV-2."*** (Kell et al., 2022, emphasis added) It has been discovered that the SARS-CoV-2 virus, or parts thereof, can persist, circulating within the affected individual for months! Indeed, one group of investigators discovered fragments of the SARS-CoV-2 pike protein within a circulating white blood cell called the monocyte—identified **15 months** following the original COVID-19 infection (Patterson et al., 2022; Kell et al., 2022). Great! The spike protein fragment is

isolated within the body of a monocyte where it cannot evoke a regional immune response or trigger aberrant clotting. But although the life of the monocyte is somehow prolonged past its typical one-day lifespan, monocytes eventually die (by the millions each day) and spill their contents (see Patterson et al., 2022). And likely, this is one of the mechanisms whereby sequestered spike protein fragments are repeatedly released into the circulation to evoke the host response that translates into clot formation, leading to the extensive capillary blockage we see in Long COVID (Kell et al., 2022). So, although you have defeated the enemy during an initial struggle (for life), you are reacting to the body parts of the virus left behind. Consider this:

> *It is important to note that the S1 [spike] protein detected in these patients appears to be retained from prior infection or phagocytosis of infected cells undergoing apoptosis [programmed cell death] and is not the result of persistent viral replication.* (Patterson et al., 2022)

Furthermore,

> *Many pieces of research-level evidence (especially suggest strongly that fibrin amyloid microclots, driven by the presence of the SARS-CoV-2 spike protein, are an inevitable accompaniment to (and a likely cause of) Long COVID.* (Kell et al., 2022)

I'm sure you are thrilled with all this new-found knowledge and want to learn more. I am very pleased with you attitude.

Recently, it was discovered that the clots formed in Long COVID are not your ordinary clot. The clots are structurally abnormal and resist breakdown (Pretorius et al., 2021; Kell et al., 2022). One protein involved and abnormally formed is called fibrin and resembles other abnormally formed proteins collectively known as amyloids—a category of proteins that are structurally unique and notoriously resistant to enzymatic breakdown.

Normally, we easily and systematically break down clots that have served their usefulness and are no longer of value. But in Long COVID, the clots formed in response to spike protein exposure are "fibrin amyloid microclots" (Kell et al., 2022). Indeed, the fibrin amyloid clot is readily formed when whole blood is exposed to the SARS-CoV-2 spike protein, as depicted on the following page.

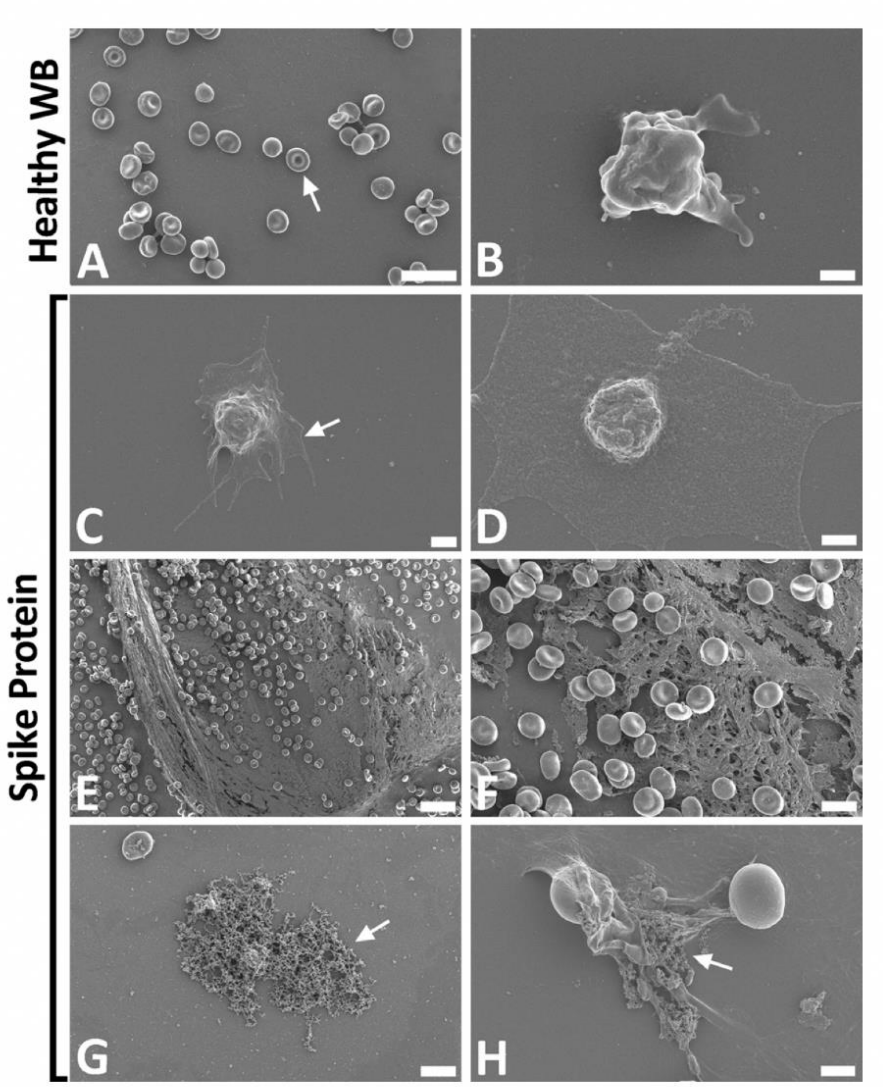


Figure 6. Whole blood sample of healthy volunteers, before and after exposure to spike protein

(**A–H**) Representative scanning electron micrographs of healthy control WB, with and without spike protein. (A,B) Healthy WB smears, with arrow indicating normal erythrocyte ultrastructure. (C–H) Healthy WB exposed to spike protein (1 ng.ml^{-1} final concentration), with (C,D) indicating the activated platelets (arrow), (E,F) showing the spontaneously formed fibrin network and (G,H) the anomalous deposits that is amyloid in nature (arrows) (scale bars: (E) 20 μm; (A) 10 μm; (F,G) 5 μm; (H) 2 μm; (C) 1 μm; (B,D) 500 nm).

Note: WB is an abbreviation for whole blood. An erythrocyte is a red blood cell. Images and description are from Grobbelaar et al., 2021. Use by author is permitted by the re-use policy of Portland Press.

The discovery of the fibrin amyloid clot phenomenon occurring in Long COVID changes everything! We now know what we are dealing with. And now we can go about the business of treating it.

Yes, we have a worm for that!

> *Lumbrokinase [LK] is a multiple-enzyme formula which can dissolve fibrin directly as well as activate plasmin, inhibit platelet aggregation and dilate capillaries to improve microcirculation and nerve conduction.* **~Gu et al., 2003**

> *The activity of LK towards amyloid dissociation was compared with the standard amyloid fibril degrading agent nattokinase (NK). Our results indicated that* ***LK can be a probable fibril degrading agent for the dissociation of amyloids.*** **~Wang et al., 2019, emphasis added**

Now this is all speculation on my part, but I have a compelling story to tell. You may want to follow along very closely.

Over the centuries, people have eaten earthworms (by the handfuls), not because they are surprisingly delicious, not because there was a famine throughout the land, but because that is something hidden deep inside (the worm) that has medicinal properties. No, I'm not making any of this up.

> *Interestingly, in Far East and China, earthworms have been used as a drug for thousands of years. Afterwards, Lumbrokinase (Lk) (earthworm derived fibrinolytic enzyme) was reputed to directly dissolve fibrin/fibrinogen by converting plasminogen to plasmin.* (Munawar et al., 2021)

The secret held tightly by the earthworm, even kept from the ancient Asians, is that they were in possession of a collection of digestive enzymes that can disassemble just about anything. The earthworm needs powerful enzymes, collectively called lumbrokinase, to digest the tough organic fibers they come across as they go about their business of doing the things that earthworms do. And being in possession of lumbrokinase is what turned earthworms into medicine. I don't think earthworms have a clue that the enzymes they possess can dissolve the fibrin network that holds blood clots together. They don't look all that bright. Have you noticed?

But the scientists are bright. They are inquisitive, too. And because of their diligent efforts, we now know a considerable amount about the anticoagulant properties of lumbrokinase. Here is a summary of their findings:

- Lumbrokinase stimulates the generation of tPA, a natural fibrin-reducing anticoagulant (Wang et al., 2013)

- Lumbrokinase can directly dissolve fibrin without degrading other major proteins in the bloodstream (Wang, et al., 2013)

- Lumbrokinase can activate plasmin, the principal fibrin-degrading protein in the blood (Gu et al., 2003)

- Lumbrokinase *"can dissolve fibrin directly as well as activate plasmin, inhibit platelet aggregation and dilate capillaries to improve microcirculation and nerve conduction."* (Gu et al., 2003)

- Lumbrokinase can lower fibrinogen levels in the blood stream, reducing the capacity for clot formation (Cao et al., 2013)

- Lumbrokinase can break down amyloids, various proteins that are formed in a manner as to resist normal enzymatic breakdown (Metkar et al., 2017)

In as much a Long COVID is a medical condition where amyloid fibrin clots form, it is tempting to speculate that lumbrokinase, serving as an anticoagulant and amyloid-reducing agent, may be the answer we are looking for.

And with respect to safety, lumbrokinase has safety written all over it. Indeed, the actions of lumbrokinase are so specific, they *"do not result in severe bleeding complications."* (Wang et al., 2019). Furthermore, with use of lumbrokinase, *"no internal bleeding has been reported."* (Munawar et al., 2021) *"So it is a safe drug."* (Cao et al., 2013)

Not only is lumbrokinase safe, but it is also impressive. Lumbrokinase is being considered as a viable option in the treatment of stroke, vascular dementia, myocardial infarction (heart attack), microcirculation-related diseases of the

kidney and nervous system, for hepatitis B (by degrading fibronectin), for organ fibrosis, and for amyloid clearing in Alzheimer's disease (Wang et al., 2019).

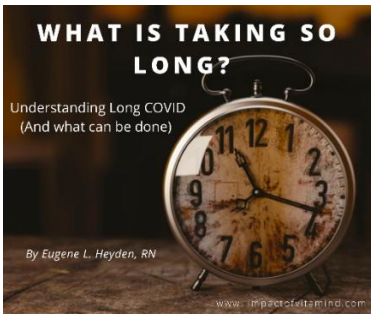
As far as the eating of earthworms go, not to worry. Lumbrokinase can be created in the lab, allowing earthworms to continue doing the things that earthworms do.

So, what do we make of it all?

In lumbrokinase, we have what appears to be an ideal therapy for Long COVID, addressing the fundamental problem of amyloid clot formation with its negative effect on the microcirculation. And on the other hand, we have basically nothing of real value to offer the patient who just can't get enough oxygen at the tissue level to feel well. H.E.L.P. Apheresis and Therapeutic Plasma Exchanges take care of the problem quickly but are not general available in the treatment of Long COVID. Therefore, I guess we're left with lumbrokinase to offer us hope.

So, the question arises, should we give lumbrokinase a try for Long COVID? Based on its anticoagulant and fibrin degrading properties, I say we should. But I also say lumbrokinase use should only be done under the guidance and watchful care of a physician. And let's see what happens.

Suggested reading



Both articles available at www.impactofvitamind.com

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