

Are Enzymes a Key to COVID Endothelial Injury?

Analysis by [Dr. Joseph Mercola](#)

✓ Fact Checked

February 10, 2023

STORY AT-A-GLANCE

- › Studies suggest damage to the endothelium, which are cells covering blood vessels, is contributing to the development of blood clots, or thrombosis, in the blood vessels of severely ill COVID-19 patients
- › Enzymes may turn out to be the missing link in helping to break up clusters of clotting proteins involved in this dangerous thrombosis, which is linked to increased mortality in COVID-19
- › Levels of Von Willebrand factor (VWF), a clotting protein released by endothelial cells, were found to be significantly elevated in COVID-19 patients in advanced stages of the disease
- › Proteolytic enzymes such as lumbrokinase, serrapeptase and nattokinase also act as natural anticoagulants by breaking down the fibrin that forms blood clots

From Dr. Joseph Mercola

Since COVID-19 first entered the scene, exchange of ideas has basically been outlawed. By sharing my views and those from various experts throughout the pandemic on COVID treatments and the experimental COVID jabs, I became a main target of the White House, the political establishment and the global cabal.

Propaganda and pervasive censorship have been deployed to seize control over every part of your life, including your health, finances and food supply. The major media are key players and have been instrumental in creating and fueling fear.

I am republishing this article in its original form so that you can see how the progression unfolded.

Originally published: November 30, 2020

Enzymes catalyze many biological reactions in your body. They regulate the rate of these chemical reactions, speeding them up so necessary functions like digestion, muscle contractions and other aspects of cellular metabolism can occur.¹

Enzymes are also emerging as key players in COVID-19, as studies suggest damage to the endothelium, which are cells covering blood vessels, is contributing to the development of blood clots, or thrombosis, in the blood vessels of severely ill COVID-19 patients.² Enzymes may turn out to be the missing link in helping to break up clusters of clotting proteins involved in this dangerous thrombosis.

Endothelium Damage Found in Critically Ill COVID-19 Cases

After noticing blackened fingers and toes – signs of what appeared to be microvascular thrombosis, or tiny blood clots in small blood vessels – in COVID-19 patients in advanced stages of the disease, physicians at the Yale School of Medicine began running clotting tests on their patients.³

Levels of Von Willebrand factor (VWF), a clotting protein released by endothelial cells, were found to be significantly elevated, which suggested to hematologist Alfred Lee that damaged endothelial cells may be releasing large quantities of VWF, leading to clots.⁴ This prompted the team to screen for additional markers of endothelial cell and platelet activation in critically and noncritically ill COVID-19 patients.

The study, which was conducted in April 2020, included 68 hospitalized patients with COVID-19 and 13 asymptomatic controls. VWF antigen was significantly elevated in COVID-19 patients admitted to the intensive care unit (ICU) compared to non-ICU COVID-19 patients,⁵ as was soluble platelet selectin (sP-selectin), which is sometimes used as a biomarker for infection and mortality.⁶

Specifically, mean VWF was 565% among ICU patients and 278% among non-ICU patients while soluble P-selectin was 15.9 ng/mL compared to 11.2 ng/mL.⁷ "Our findings show that endotheliopathy is present in COVID-19 and is likely to be associated with critical illness and death. Early identification of endotheliopathy and strategies to mitigate its progression might improve outcomes in COVID-19," the researchers concluded.⁸

Likely not coincidentally, endothelial dysfunction is also associated with insulin resistance and plays a role in the vascular complications of diabetes,⁹ as well as being involved in obesity and high blood pressure,¹⁰ conditions that raise the risk of severe COVID-19.

Even mild obesity may raise the risk of COVID-19 severity – COVID-19 patients with mild obesity had a 2.5 times greater risk of respiratory failure and a five times greater risk of being admitted to an ICU compared to nonobese patients. Those with a BMI of 35 and over were also 12 times more likely to die from COVID-19.¹¹

Another study looking into the impact of coexisting health conditions like high blood pressure, heart disease and diabetes on COVID-19 outcomes found they're linked to "poorer clinical outcomes," such as admission to an intensive care unit, a need for invasive ventilation or death.¹²

It's possible that the endothelial damage in all of these conditions plays a role in worsening COVID-19 outcomes, but it's unclear which comes first – endothelial damage or COVID-19.

Endothelial Cells Are the 'Main Target' of SARS-CoV-2

Imperial College London cardiologist Thomas Lüscher told The Scientist that the endothelium is the main target of SARS-CoV-2, the virus that causes COVID-19.¹³ Under healthy conditions, blood cells can pass through the endothelium lining blood vessels, but when exposed to viral infections and other inflammatory agents, the endothelium becomes sticky and releases VWF.

The end result is a cascade of clotting and inflammation, both characteristics of severe COVID-19. According to a case report published April 8, 2020, "A hallmark of severe COVID-19 is coagulopathy, with 71.4% of patients who die of COVID-19 meeting ... criteria for disseminated intravascular coagulation (DIC) while only 0.6% of patients who survive meet these criteria."¹⁴

Writing in the European Heart Journal, Lüscher argues, "COVID-19, particularly in the later complicated stages, represents an endothelial disease,"¹⁵ which may help explain why multiple organ systems, including the lungs, heart, brain, kidney and vasculature, may be affected.

An additional study by Canadian researchers, published in Critical Care Explorations in September 2020, also revealed elevated VWF and soluble P-selectin levels in COVID-19 patients, along with higher glycocalyx-degradation products,¹⁶ a sign of damage to the glycocalyx, which envelops the endothelium.¹⁷ This can also be a sign of sepsis. Taken together, the research suggests that therapies targeting the endothelium may be useful for COVID-19, which is where enzymes come in.

Enzymes Used to Treat COVID-19

With the role of coagulopathy in severe COVID-19 becoming clearer, researchers have experimented with **enzymes** in the treatment of the disease. Fibrinolytic therapy, which uses drugs or enzymes to break up blood clots, has been used in a Phase 1 clinical trial that showed the treatment reduced mortality and led to improvements in oxygenation.¹⁸ Further, researchers wrote in the Journal of Thrombosis and Haemostasis:¹⁹

"There is evidence in both animals and humans that fibrinolytic therapy in acute lung injury and acute respiratory distress syndrome (ARDS) improves survival, which also points to fibrin deposition in the pulmonary microvasculature as a contributory cause of ARDS.

This would be expected to be seen in patients with ARDS and concomitant diagnoses of DIC on their laboratory values such as what is observed in more

than 70% of those who die of COVID-19."

The researchers reported three case studies of patients with severe COVID-19 respiratory failure who were treated with tissue plasminogen activator (TPA), a serine protease enzyme found on endothelial cells that's involved in fibrinolysis, or the breakdown of blood clots.²⁰

All three patients benefited from the treatment, with partial pressure of oxygen/FiO₂ (P/F) ratios, a measure of lung function, improving from 38% to 100%.²¹ While it should be noted that several of the authors have patents pending related to both coagulation/fibrinolysis diagnostics and therapeutics, the results suggest such treatments deserve further evaluation in certain COVID-19 patients.

An evaluation of organ tissues from people who died from COVID-19 also revealed extensive lung damage, including clotting, and long-term persistence of virus cells in pneumocytes and endothelial cells.²²

The findings indicate that virus-infected cells may persist for long periods inside the lungs, contributing to scar tissue. In an interview with Reuters, study co-author Mauro Giacca, a professor at King's College London, described "really vast destruction of the architecture of the lungs," with healthy tissue "almost completely substituted by scar tissue,"²³ which could be responsible for cases of "long COVID," in which symptoms persist for months.

"It could very well be envisaged that one of the reasons why there are cases of long COVID is because there is vast destruction of lung (tissue)," he told Reuters. "Even if someone recovers from COVID, the damage that is done could be massive."²⁴ Dissolving scar tissue is another area where enzymes, particularly proteolytic enzymes, may be useful.

Three Top Enzymes Act as Natural Anticoagulants

Holistic prophylactic alternatives that might be beneficial against blood clots include proteolytic enzymes such as lumbrokinase, serrapeptase and nattokinase, all of which

act as natural anticoagulants by breaking down the fibrin that forms the blood clot. Fibrin is a clotting material that restricts blood flow, found both in your bloodstream and connective tissue such as your muscles. Fibrin accumulation is also responsible for scar tissue.

It is important to understand that when using these enzymes for fibrinolytic therapy they need to be taken on an empty stomach, at least one hour before or two hours after meals. Otherwise these enzymes will be wasted in the digestion of your food and will be unable to serve their fibrinolytic purpose.

As noted in Scientific Reports, some of the key mechanisms by which proteolytic enzymes exert their anticoagulant effect include "defibrinogenation, inhibition of platelet aggregation, and/or interference with components of the blood coagulation cascade."²⁵ Here's a closer look at these important enzymes, all of which are available in supplement form or, in the case of nattokinase, via the food natto.

- 1. Lumbrokinase** — This enzyme is about 300 times stronger than serrapeptase and nearly 30 times stronger than nattokinase,²⁶ making it my strong personal preference and recommendation if you are using a fibrinolytic enzyme. Extracted from earthworms, lumbrokinase is a highly effective antithrombotic agent that reduces blood viscosity and platelet aggregation²⁷ while also degrading fibrin, which is a key factor in clot formation.
- 2. Serrapeptase** — Also known as serratiopeptidase, serrapeptase is produced in the gut of newborn *Bombyx mori* silkworms, allowing them to dissolve and escape from their cocoons. Research has shown it can help patients with chronic airway disease, lessening viscosity of sputum and reducing coughing.²⁸ Serrapeptase also breaks down fibrin and helps dissolve dead or damaged tissue without harming healthy tissue.²⁹
- 3. Nattokinase** — Produced by the bacteria *Bacillus subtilis* during the fermentation of soybeans to produce natto,³⁰ nattokinase is a strong thrombolytic³¹ comparable to aspirin but without the serious side effects.³²

It's been shown to break down blood clots and reduce the risk of serious clotting³³ by dissolving excess fibrin in your blood vessels,³⁴ improving circulation and decreasing blood viscosity. Interestingly, in one in vitro study, the thrombolytic activity of equivalent amounts of nattokinase and TPA were found to be identical³⁵ – TPA, remember, is the enzyme that led to improvement in the three COVID-19 case studies.³⁶

Sources and References

- ¹ Britannica, Enzyme
- ^{2, 3, 4, 13, 17} The Scientist November 3, 2020
- ^{5, 7, 8} The Lancet Haematology June 30, 2020
- ⁶ Biomark Insights. 2017; 12: 1177271916684823
- ⁹ Diabetes Obes Metab. 1999 May;1 Suppl 1:S17-22. doi: 10.1046/j.1463-1326.1999.0010s1017.x.
- ¹⁰ Circulation July 27, 2020
- ¹¹ U.S. News & World Report July 23, 2020
- ¹² medRxiv February 27, 2020
- ¹⁴ J Thromb Haemost Case Reports April 8, 2020 DOI: 10.1111/jth.14828
- ¹⁵ European Heart Journal, Volume 41, Issue 32, 21 August 2020, Pages 3038–3044
- ¹⁶ Critical Care Explorations: September 2020 - Volume 2 - Issue 9 - p e0194 doi: 10.1097/CCE.0000000000000194
- ^{18, 19, 21, 36} J Thromb Haemost. 2020 May 11 : 10.1111/jth.14828
- ²⁰ Diapharma, Tissue Plasminogen Activator
- ²² EBioMedicine November 3, 2020
- ^{23, 24} Reuters November 3, 2020
- ²⁵ Scientific Reports 2018; 8, Article Number 6210
- ²⁶ Townsend Letter May 2018
- ²⁷ BioMed Research International. Volume 2011 | Article ID 51965. 2003
- ²⁸ Respirology 2003 Sep;8(3):316-20
- ²⁹ International Journal of Surgery, 2013 Apr;11(3):209-217
- ³⁰ Int J Mol Sci. 2017 Mar; 18(3): 523
- ^{31, 34} Biol Pharm Bull. 1995 Oct;18(10):1387-91
- ³² Lab Anim Res December 2013; 29(4): 221-225
- ³³ Scientific Reports 2015; 5: 11601
- ³⁵ Article Number 6210, Lunathrombase has in vitro thrombolytic potency but is devoid of hemolytic activity or cytotoxicity against mammalian cells