

How the Spike Protein Causes Amyloid “Clots” (and how to stop the clot)

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What is the Mechanism?

The amyloid clots are formed when spike proteins are cleaved by the neutrophil enzyme revealing no less than 7 peptide sequences that are amyloid creating at 37degrees c. This was the result of the Swedish study.

What appears to be happening is -

1. The vaccine causes systemic wide distribution of the spike protein
2. In the presence of inflammation or infection, neutrophils pervade the infected area
3. Enzymes excreted by the neutrophils cleave the spike protein exposing the 7 amyloidogenic regions
4. These regions cause the formation of amyloid within 24 hours

See here - [Researchers discover possible connection between harmful amyloid production and COVID-19 symptoms \(news-medical.net\)](https://news-medical.net/news/2020/05/27/researchers-discover-possible-connection-between-harmful-amyloid-production-and-covid-19-symptoms/)

See here - [Amyloidogenesis of SARS-CoV-2 Spike Protein | Journal of the American Chemical Society \(acs.org\)](https://pubs.acs.org/doi/10.1021/ja0c01111)

So the Equation is ?

Spike Protein + Neutrophil enzyme → cleavage of spike → exposure of amyloidogenic sequences

Amyloidogenic sequences → formation of fibrils + amyloids

How do the Amyloid Clots Get So Big ?

These amyloid clots are often the size of a finger or even the length of an arm - so where does enough spike come from to form these huge clots?

The answer to this question may be found in the ability of amyloids to self-replicate – see <https://www.sciencealert.com/amyloid-protein-self-replication-abiogenesis-contrasts-rna-world>

According to Wikipedia -

"Amyloid fibre formation is the default state for misfolded proteins, and fibrillar aggregates found in amyloidosis result from defects in the cellular machinery that prevents protein misfolding."

In other words, the reason why the peptide regions within the spike protein are able to generate amyloids, is because these regions **cause the misfolding of proteins** - in other words, these peptide regions are **prions**.

So the answer as to how an amyloid can grow so large, lies in understanding how prions can multiply, and how amyloids can self-replicate.

Wikipedia has this to say -

"Prions are a type of intrinsically disordered protein.. more units can get added, making a sort of "fibril".[10] Prions form abnormal aggregates of proteins called amyloids, which accumulate in infected tissue and are associated with tissue damage and cell death."

Yes, prions form fibrils and abnormal aggregates of proteins calledwait for itamyloids.

So the amyloid “clots” that have started appearing in both living and deceased vaccinated are caused by prions.

And the prions themselves, numbering 7 in total, are exposed (activated) when the spike protein is cleaved by neutrophil enzyme.

Potential for Amyloid Formations in Vaccinated

The first line of defence of our innate immune system to ANY infection or injury is to send neutrophils to the site of injury or infection. It's the first thing that happens. Neutrophils are our "first-responders". They arrive at the site of infection – and excrete an enzyme designed to cut up foreign proteins into smaller pieces.

However, when this enzyme cuts up (cleaves) the spike protein, it exposes the peptide regions within the spike that act as prions (amyloid generating sequences).

So the horrifying situation is that ANYONE who has a spike protein load within their blood, and then acquires ANY infection or injury, will trigger a prion disease – amyloid will start to form. Since prions/amyloids are self-replicating, this process will proceed indefinitely.

How Long Will it Take?

Undertakers began reporting the appearance of large amyloid clots in the deceased in May of 2021 – that's five months after roll-out began. This suggests that it takes about 5 months for the amyloid clots to grow large enough to impede blood flow and cause fatalities.

However, in some people injury may occur sooner if they are engaging in vigorous exercise. Such exertion may cause an amyloid to dislodge and move to a vital artery or to the heart where such clots can cause cardiac arrest, as we are seeing in the cardiac arrests and sudden deaths of so many athletes.

And, it is not just undertakers and embalmers who have reported these clots. Dr Ryan Cole, a cardiologist, reports that he has received several samples of such clots sent to him by surgeons who have removed them from patients.

How Can We Stop the Amyloid Process ?

There is probably a way of stopping these amyloids from forming by looking at the 7 prion sequences found in the spike, and working out what would bind to these sequences thereby neutralising them - just like an antibody works - it binds to the antigen preventing it from doing harm.

We know what the 7 prion sequences are – we know the exact sequences –

Peptide	Amino acid sequence ^a	MW (Da) ^b	pI	ThT kin	Congo Red	Ultrastructure
Spike192	FVFKNIDGYFKIYSKHTPIN	2431	9.4	+	+	fibril
Spike258	WTAGAAAYYVGYLQPRTFLLK	2389	9.5	-	+	fibril
Spike365	KKKGGGYSLYNSASFSTFK	2169	10.0	-	+	amorphous
Spike532	NLVKNKCVNFNGLTGTGV	2139	9.3	+	+	amorphous
Spike601	GTNTSNQVAVLYQDVNCTEV	2155	3.7	+	+	fibril
Spike685	KKKRSVASQSHAYTMSLGA	2139	10.5	-	-	ribbons
Spike1166	LGDISGINASVNIQKEIDR	2141	4.6	+	+	fibril

^aResidues assigned in color indicate the amyloidogenic segments as predicted by WALTZ. Highlighted in gray are non-native amino acids introduced for solubility.

So all that is required is to administer a peptide that is able to bind to these sequences and block their action. Such peptides could be tested in vitro – simply by adding pure spike + neutrophil enzyme to 20 wells, then adding the blocking peptide to half of them. In the control group, the amyloid will proliferate, but in the experimental group the amyloid should be absent.