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# Vaccine Class Effect Hypotheses and the Spike Sars-COV-2 Protein as a Procoagulant Factor

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**Abstract:**
This work is based on a real-life situation in which some covid vaccines are being investigated in depth in relation to some uncommon incidences of thrombosis. It's fascinating to look at the literature that shows a link between increased thrombosis in the most severe patients in the first and second And third waves of covid-19 illness. As a result, it is feasible to conclude that the covid-19 virus has a procoagulant characteristic. However, is this feature shared by all viral particles, or is it mediated or caused by the spike-protein? And if there is a link, isn't it a great aspect of the project vaccination based on this protein?.

**Introduction:**
Normally, it takes years to develop and test a new vaccine, but in order to combat the deadly covid-19 pandeemia, a new vaccine or gene manipulation product with a similar impact has to be introduced in a shorter period of time (like about 1 year). This methodology is not ideal for verifying the effect over a long period of time (5-10 years), and for some vaccines, additional issues appear to contradict this vaccine campaign.

Observing the media and some scientific literature, some rare incidences of thrombosis have been recorded, and some advanced countries have asked the EMA to clarify before proceeding. What's intriguing is that among the various types of thrombosis, a specific condition called cerebral venous sinus thrombosis [1] has been discovered in some cases. So, why is this particular type of pathology more common?

Thrombosis is a disease that affects the elderly in particular, and its prevalence rises with age. It could be caused by a hereditary or autoimmune condition. We must also keep in mind that neoplastic disorders are linked to an increase in TNF and procoagulant production. Infection of RNA virus cells stimulates the so-called innate immune sensor, resulting in hyperactivation of the immune response Interleukins (ILs), cytokines, TNF, and other inflammatory mediators, as well as clotting problems. Heparin is also used therapeutically in severe COVID 19 patients with coagulation problems. Heparin in COVID-19 patients is associated with lower hospital mortality: the Multicenter Italian CORIST Study, according to Augusto Di Castelnuovo et al. 2021; “In-hospital heparin treatment was associated with a lower mortality, particularly in severely ill COVID-19 patients and those with strong coagulation activation.” The findings of randomised clinical trials are anxiously sought in order to make definitive recommendations. But is the coronavirus's action tied to the SPIKE protein? [2]. If yes, was it a smart idea to develop a virus vaccine based on this protein?
Is there a vaccination class effect involved in this mechanism?

**Keywords:** : : Thrombosys • COVID-19• Spike-protein • Procoagulant factor • Epidemiology • Incidence • Vaccine • Class effect • Screening procedure pre vaccination.

**Methods:**
SARS-CoV-2 from the alveoli binds and activates platelets, promoting thrombosis and an inflammatory response in capillaries, resulting in diffuse intravascular coagulation and acute respiratory distress syndrome. The -CoV-2 spike protein binds to ACE2 and causes it to phosphorylate, causing MAPK signal transmission to be activated (Erk SARS phosphorylation, p-38, and JNK), platelet activation, and the release of coagulation factors and inflammatory cytokines. Platelet activation is caused by the interaction of SARS-CoV-2 spike protein with platelet ACE2, which is inhibited by recombinant human ACE2 protein and monoclonal anti-spike antibodies. Si Zhang et al. has been modified.
In COVID-19, SARS-CoV-2 binds platelet ACE2 to increase thrombosis. (A) Virion proteins known to bind integrins via an RGD motif (shown in space-fill) include (right) foot and mouth disease virus capsid protein (5neu—this RGD motif is highly flexible prior to integrin-binding, but structurally stabilised when bound to integrin is from a co-crystal of the capsid protein and integrin with integrin structure remov (1ahs top domain of capsid protein VP7**).**

**Result:**
“SARS-CoV-2 and its Spike protein directly enhanced platelet activation such as platelet aggregation, PAC-1 binding, CD62P expression, granule secretion, dense granule release, platelet spreading and clot retraction in vitro, and thus spike protein enhanced thrombosis formation in wild-type mice transfused with hACE2 transgenic platelets, but this was not observed in wild-type mice transfused with hACE2
We also found evidence that the MAPK pathway, which is downstream of ACE2, mediates SARSCoV-2's potentiating effect on platelet activation, and that platelet ACE2 expression reduces after SARS-COV-2 stimulation. Platelets were directly activated by SARS-CoV-2 and its Spike protein, which facilitated the release of coagulation factors, the production of inflammatory factors, and the development of leukocyte–platelet aggregates.
“These experiments reveal that TNF can deliver a significant net procoagulant stimulation to the hemostatic mechanism, and suggest that this cytokine may be a modulator of certain hypercoagulable conditions in humans,” write K A Bauer et al.

"Thrombo Embolism rates of COVID-19 are high and related with a higher risk of death," write Mahmoud B Malas et al. To establish the influence of thromboprophylaxis on COVID-19 TE and mortality risk, robust evidence from ongoing clinical trials is required.".

**Discussion:**

Literature shows that there is an increase in prothrombotic signals in covid-19 illness. This could be due to the entire virus, a portion of it, or an enhanced inflammatory response in the human body. (For example, TNF as a procoagulant agent and the effect of other variables on platelet activation.)

The presence of a coagulation abnormality in COVID-19 has led to recommendations for the use of anticoagulants to limit the risk of thrombosis-related consequences. The use of unfractionated or low molecular weight peptides has been proven in the majority of research on the therapy of this condition. Heparin

**Conclusions**:

The binding of virions to integrins on endothelial cells has the potential to activate angiogenic cell signalling pathways, dysregulate integrin-mediated developmental signalling pathways, and trigger endothelial activation to commence blood clotting. This procoagulant state, combined with increased platelet aggregation caused by virions binding to platelet integrins, could increase the creation of microthrombi, increasing the risk of pulmonary thrombosis and embolism, strokes, and other thrombotic complications. " Thrombosis caused by covid-19 and other unusual events following covid-19 vaccination have some similarities.