Genové očkování – quo vadis?

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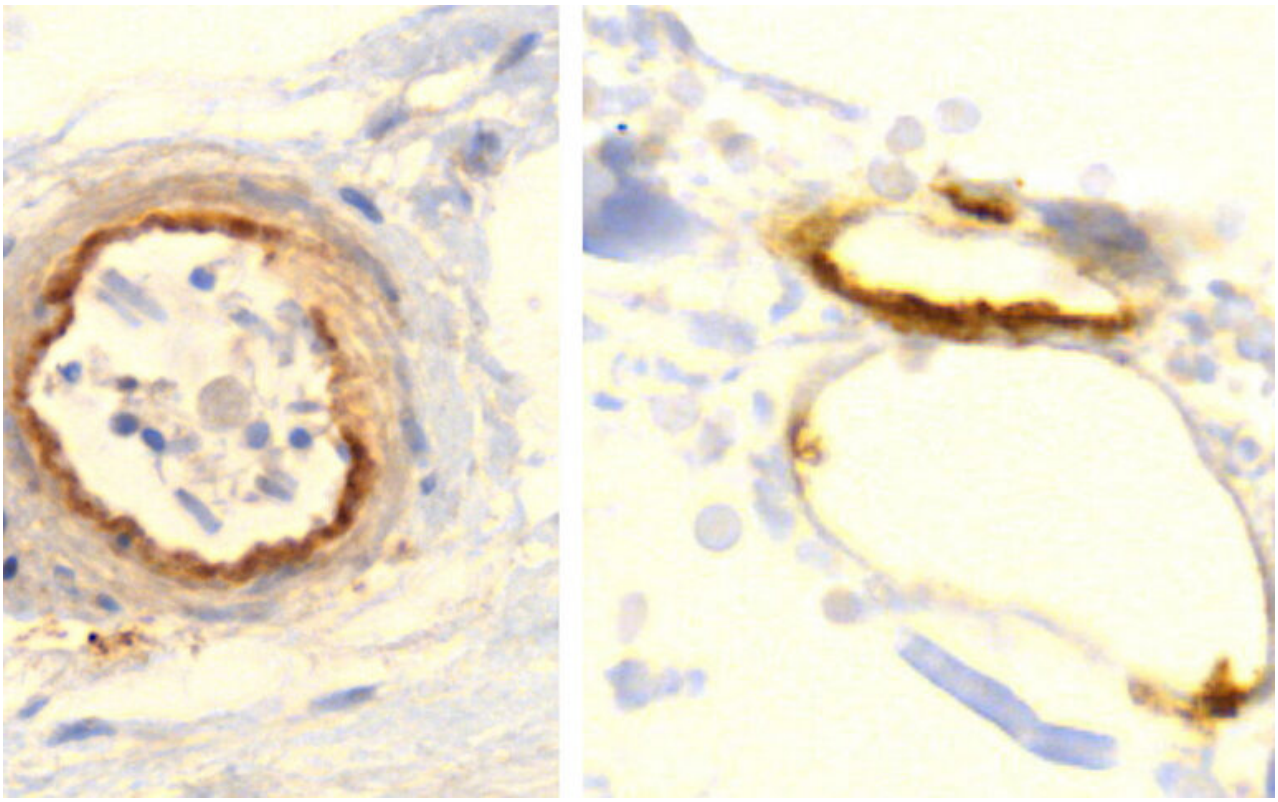

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1. Zdůvodnění vývoje vakcín

Koncepce vývoje vakcíny je přímočará: aplikace neškodného derivátu infekčního agens by měla stimulovat imunitní systém k produkci protilátek, které proti tomuto agens chrání.

Zavedení jakékoli cizorodé látky do těla však nemůže být nikdy zcela zbaveno rizik, takže primární otázkou, kterou je třeba řešit, je, zda lze očekávat, že přínos převáží rizika. Proto,

1. patogen musí být nebezpečný – infekce s ním je spojena s vysokou morbiditou a mortalitou a
2. očkování vytvoří silnou imunologickou ochranu proti závažným onemocněním.

Tyto předpoklady byly splněny v historických úspěších vývoje vakcín proti pravým neštovicím, tetanu, záškrtu a poliomyelitidě. Euforie vytvořená těmito vědeckými milníky však způsobila, že jeden rozhodující fakt byl přehlížen. Ve všech čtyřech případech byli agenti transportováni na místo určení v krevním řečišti.

Je nutné si uvědomit, že jde o výjimku a nikoli pravidlo. Většina virových patogenů způsobuje samoomezující infekce dýchacího nebo gastrointestinálního traktu. K vážnému poškození vnitřních orgánů způsobenému jejich šířením krevním řečištěm dochází zřídka a infekce obecně nejsou spojeny s vysokou úmrtností. Vzhledem k jejich všudypřítomnosti je vysoká úroveň základní imunity vůči takovým virům již přítomna v obecné populaci. Z těchto jednoduchých důvodů neexistuje skutečná potřeba vývoje vakcín proti většině virových patogenů.

2. Imunita vůči respiračním virům: systémová versus slizniční imunita

Nyní přejdeme k důležitému faktu týkajícímu se ochrany dýchacích cest před infekcemi: je zprostředkována buňkami imunitního systému, které sídlí uvnitř a pod našimi dýchacími sliznicemi, a tyto buňky fungují zcela nezávisle na imunitních buňkách, které chrání naše vnitřní orgány.

Klíčový aspekt tohoto funkčního oddělení mezi slizniční a systémovou imunitou se týká povahy protilátek produkovaných plazmatickými buňkami umístěnými přímo pod sliznicemi. Tyto protilátky – *sekreční imunoglobulin A* (sIgA) – jsou vylučovány přes sliznice na jejich povrch. Jsou tedy na místě, aby se setkaly s viry přenášenými vzduchem, a mohou jim být schopny zabránit navázání a infikování buněk v těchto sliznicích. Stejný způsob ochrany se týká i trávicího traktu.

Naproti tomu IgG a cirkulující IgA jsou hlavní protilátky nalezené v krevním řečišti. Nemohou zabránit vstupu virů do buněk, které vystýlají dýchací cesty nebo střevo, a mohou v nejlepším případě zabránit jejich šíření, pokud se dostanou do oběhu. Rozhodující je, *že vakcíny, které jsou injikovány do svalu – tj. do vnitřku těla – budou indukovat pouze IgG a cirkulující IgA, ale ne sekreční IgA*. Protilátky vyvolané takovými vakcínami proto nemohou a nebudou účinně chránit buňky dýchacího traktu před infekcí vzdušnými viry [1 , 2] . Toto zjištění není ani sporné, ani nové. Již před 30 lety McGhee a spol. [2] uzavřel:

Je překvapivé, že navzdory naší současné úrovni chápání běžného slizničního imunitního systému jsou téměř všechny současné vakcíny podávány lidem parenterální cestou [tj. injekčně]. Systémová imunizace je v podstatě neúčinná pro indukci slizničních imunitních odpovědí. Vzhledem k tomu, že většina infekčních mikroorganismů se vyskytuje v oblastech slizničního povrchu, je logické uvažovat o indukci ochranných protilátek a odpovědí T buněk ve slizničních tkáních.
Selhání intramuskulární injekce při indukci sekrečního IgA bylo potvrzeno ve studii Middle East Respiratory Syndrome (MERS) [3] . Stejně jako COVID-19 je toto onemocnění způsobeno koronavirem a experimentální vakcína použitá ve studii byla založena na genu, stejně jako všechny hlavní vakcíny, které jsou v současné době nasazeny proti COVID-19. Nedávno jiná studie ukázala, že mRNA COVID-vakcíny také nestimulují podstatnou produkci sekrečního IgA [4] . Z tohoto prostého důvodu nelze očekávat, že očkování zabrání infekci dýchacích cest. Naprosté selhání vakcín při prevenci infekce SARS-CoV-2 je dnes skutečně dobře zdokumentováno [5 , 6] .

Je všeobecně známo, že sekreční protilátky IgA (sIgA) jsou produkovány v reakci na přirozeně se vyskytující infekce dýchacích cest. Sliznice zdravých jedinců jsou následně potaženy protilátkami namířenými proti běžným respiračním virům. Schopnost těchto

protilátek předcházet infekcím je však omezená. Výsledek setkání s virem není „černý nebo bílý“ – důležitá jsou čísla. Stěna ochranných protilátek může odrazit útok malého rozsahu, ale při vyšší virové zátěži bude narušena. To je důvod, proč se infekce vzdušnými viry vyskytují opakovaně po celý život, což je skutečnost, kterou nezmění ani použití intranazálních vakcín za účelem stimulace produkce sIgA, i když intranazální aplikace vakcíny vyvolává silnější slizniční imunitní reakce než intramuskulární injekce[3 , 7] .

Podřízená úloha sekrečního IgA v boji proti systémovým virovým infekcím je zdůrazněna skutečností, že jedinci s velmi častým genetickým defektem – selektivním deficitem sIgA –, kteří nejsou schopni produkovat sIgA, netrpí dramaticky zvýšenou náchylností k těžkým respiračním infekcím. Toto pozorování lze pochopit z následujících dvou principů: za prvé, imunologická ochrana proti respiračním virům spočívá hlavně na T-buňkách; a za druhé, u pacientů s již existující imunitou jsou hladiny protilátek v krevním řečišti (cirkulující IgG a IgA) obecně dostatečné k prevenci závažného onemocnění prostřednictvím šíření viru v těle.

3. Klíčoví hráči v antivirové imunitě: T-lymfocyty

T-lymfocyty jsou klíčové pro kontrolu respiračních infekcí, a to se týká i virových infekcí obecně. Pozornost se nyní obrací k těmto buňkám, přičemž diskuse může být zpočátku zaměřena na funkci cytotoxických T-lymfocytů (CTL).

Co tyto buňky rozpoznávají a jaký je hlavní důsledek tohoto imunitního rozpoznání?

Kdykoli buňka produkuje specifický protein, vytvoří jeho více kopií. Několik z těchto kopií bude záměrně rozloženo na malé fragmenty; ty jsou pak transportovány na povrch buňky spolu se specifickou nosnou molekulou nazvanou MHC 1. Tam se fragmenty stávají přístupnými pro interakci s CTL a pro jejich rozpoznání. Různé fragmenty budou rozpoznány lymfocyty patřícími k různým „klonům“; všechny buňky daného klonu T-buněk ponesou stejné

receptory T-buněk a rozpoznávají stejné proteinové fragmenty, ale buňky patřící k různým klonům se budou lišit ve své antigenní specifitě (obrázek 1). T-buňka, která dokáže najít a vázat svůj příbuzný proteinový fragment, bude tím aktivována, aby vyvrhla smrtící toxické látky na a do cílových buněk.

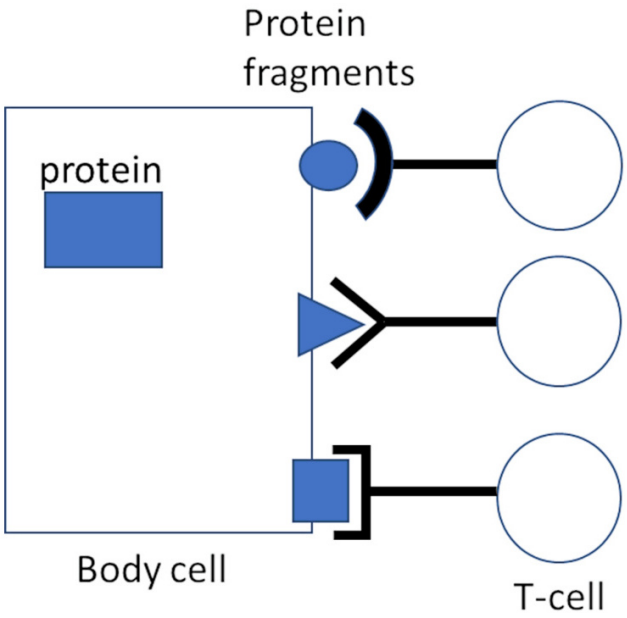

Figure 1: Lock and key interaction between protein fragments on the surface of a cell and T-cell receptors of cytotoxic T-cells. The fragments are presented to the T-cells by a specific carrier molecule, MHC 1 (not shown). The T-cell receptors on our body's T-lymphocytes can recognize, collectively, a very large spectrum of protein fragments, but all of the receptor molecules on a given T-cell are identical and will bind to the same fragments. T-cells which bind to one of the protein fragments presented by a MHC 1 molecule on a cell surface will thereby be activated.

If the protein whose fragments had attracted and activated those CTL was encoded by a virus, then the result will be the destruction of the virus-infected cell, which is useful and necessary for eradicating a viral infection. However, note that the process of protein fragmentation and presentation is completely general—it is not limited to viral or other "non-self" proteins, but rather applies to the body's own "self" proteins as well. It is therefore vital to prevent the activation of CTL that recognize the fragments of these "self" protein-derived fragments. How is this accomplished?

Envisage the interaction between presented protein fragment and its "receptor" on the T-cell as one between lock and key. There are myriad different keys (fragments) fitting into myriad different locks (T-cell receptors). It is known that the truly incredible diversity of locks arises already during fetal development. How does this happen? Are locks molded in response to the fragments (keys) as these appear during development? Then, since the fetus is not usually exposed to any viral infections, CTL would be equipped with receptors exclusively recognizing "self" protein fragments; but these self-reactive CTL clones could hardly serve a useful purpose. If, on the other hand, the diversity of locks should arise haphazardly and by chance, without requirement for any instructing template (key), then billions of lymphocytes that recognize "non-self"—extraneous agents, including virus proteins—should be generated alongside those that recognize "self."

Intriguingly, the latter is today known to be the case [8]. Wondrously, lymphocytes recognizing "self" are silenced or held in check throughout life, preventing them from wantonly attacking healthy body cells. Mishaps occasionally occur that can lead to autoimmune disease. Come T-cells out of cover that are reactive against liver proteins, come autoimmune hepatitis. Come T-cells out of cover that are reactive against the pancreatic islets, come autoimmune diabetes.

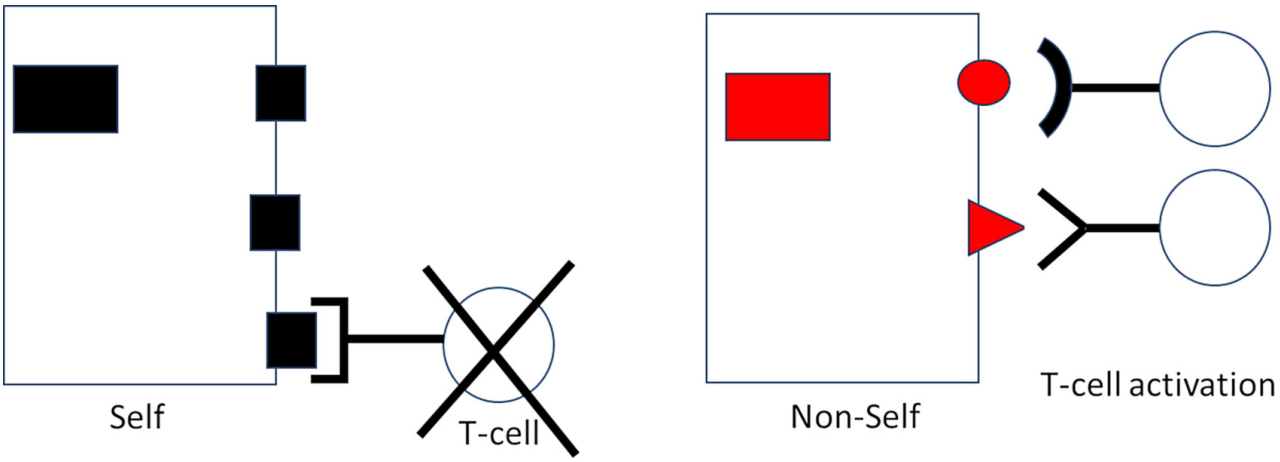

Figure 2: Clonal selection of T-lymphocytes. The diversity of T-cell receptors is initially generated at random, which means that many T-cells will carry receptors that bind to self antigens. In the thymus, such T-cells are "baited" by cells that express those antigens and then destroyed or suppressed. T-cells which do not bind self antigens will persist and may at a later time be activated and induced to multiply in response to a virus infection.

But on the other hand, immune cells reactive against essentially all non-self proteins and present at birth are ready to spring into action whenever a challenge is issued. It is for this very reason that conventional vaccinations can successfully be performed already in early infancy. And when a Coronavirus comes around, up rises the anti-Corona CTL team. When flu comes around, up rises the anti-flu team, etc. Each bout of training strengthens the team, enabling the opponent to be more rapidly constrained and infections terminated with increasing effectiveness.

But is such acquired immunity not voided and evaded by ever new virus "variants of concern"? Not so. Here, one must note that a protein will generate many fragments that are recognized by many different CTL clones. The proteins encoded by a virus mutant may generate one or a few differing fragments, but the majority of other fragments will remain the same. For this reason, CTL-based cross-reactivity and cross-protection exists between all members of a given virus family. In connection with COVID-19 specifically, it has been noted that previously infected persons may indeed sometimes contract another infection with a new variant, but such reinfections are almost never of a serious nature [9,10]. This is just as we should

have expected; the narrative that emergence of virus mutations must be countered by development of customized vaccines has thus been fundamentally flawed right from the start.

Activation of T-lymphocytes—but in this case, *T-helper cells* rather than CTL—is also coupled to B-lymphocyte activation, and this leads to antibody production (Figure 3). While CTL recognize fragments of proteins presented on the cell surface, antibodies bind to the intact proteins themselves. Bound antibodies then trigger activation of another major arm of immune defense, the complement system, with far-reaching consequences. A plethora of inflammatory events are triggered by complement activation. Furthermore, the complement system itself will attack and destroy the cell on whose surface activation occurs.

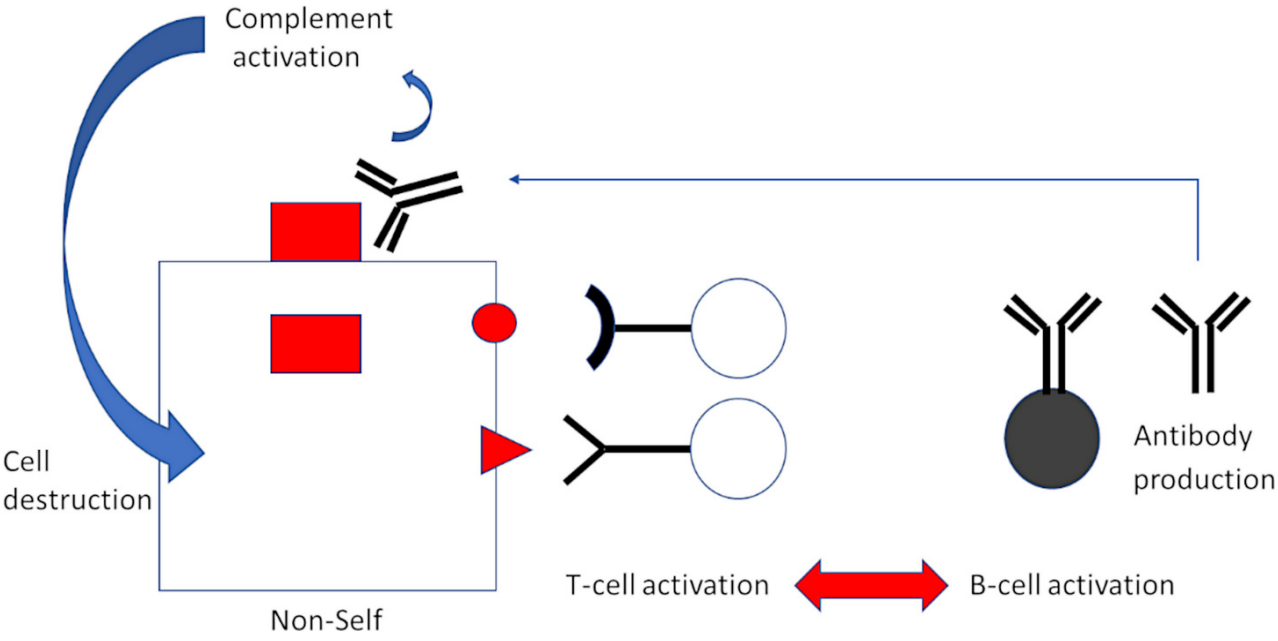

Figure 3: Cooperation of T-cells and antibodies in antiviral defense. T-helper cells are activated by the fragments of a viral "non-self" antigen in much the same way as are CTL. However, their role is not to go on the attack themselves; instead, they activate B-cells in turn, which will then start producing antibodies to the intact non-self protein. If these antibodies find their target on the surface of an infected cell, they will activate *complement*, a cascade of serum proteins which can destroy that cell and also promote inflammation generally.

4. Every gene-based vaccine encoding non-self is direly dangerous

It follows from the above that production of “non-self” antigens by our own body cells will invariably provoke inflammatory and cell-destructive processes. In viral infections, this is to the purpose, because it leads to elimination of befallen cells. Most viruses target a limited spectrum of tissues, and most tissues can regenerate, so wounds can heal thereafter.

Proponents of gene-based vaccines commonly argue that these agents do nothing more than mimic what happens in actual virus infections. Expression of the alien protein is thereby claimed to be short-lived and confined mainly to the site of intramuscular injection. Any cell damage should likewise be limited, and serious adverse reactions are therefore not to be expected.

Nothing could be more misleading and further from the truth.

The assertion that LNP-packaged mRNA remains at the site of injection is by now widely known to be a blatant untruth. These “vaccines” rapidly spread from the site of injection to lymph nodes and the blood circulation [11]; and long-lived expression in organs and tissues at distance from the injection site has been documented repeatedly and with range of analytical techniques [12–15]. And because the vaccine particles can enter all nucleated cells, their uptake is bound to rapidly occur in cells of the lymph nodes, in endothelial cells that line the walls of blood vessels, and in cells of every tissue they reach.

This fact immediately sets apart “mRNA-vaccination” from naturally occurring infections. Very few infectious agents systemically target lymphocytes or endothelial cells. Amongst the latter are dangerous viruses that cause hemorrhagic fevers, and bacteria that also cause life-threatening infections, e.g. typhus and Rocky Mountain spotted fever.

In striking contrast, each and every mRNA-“vaccine” will incite self-destructive processes in lymphatic organs and in blood vessels throughout the body. The immense dangers of self-attack events occurring within the immunological control network have been outlined [16]. They include the reactivation of dormant infections (e.g. Herpes simples, shingles, EBV, CMV, tuberculosis, parasites), reduced capacity to control new infections, and activation or reactivation of neoplasms [17].

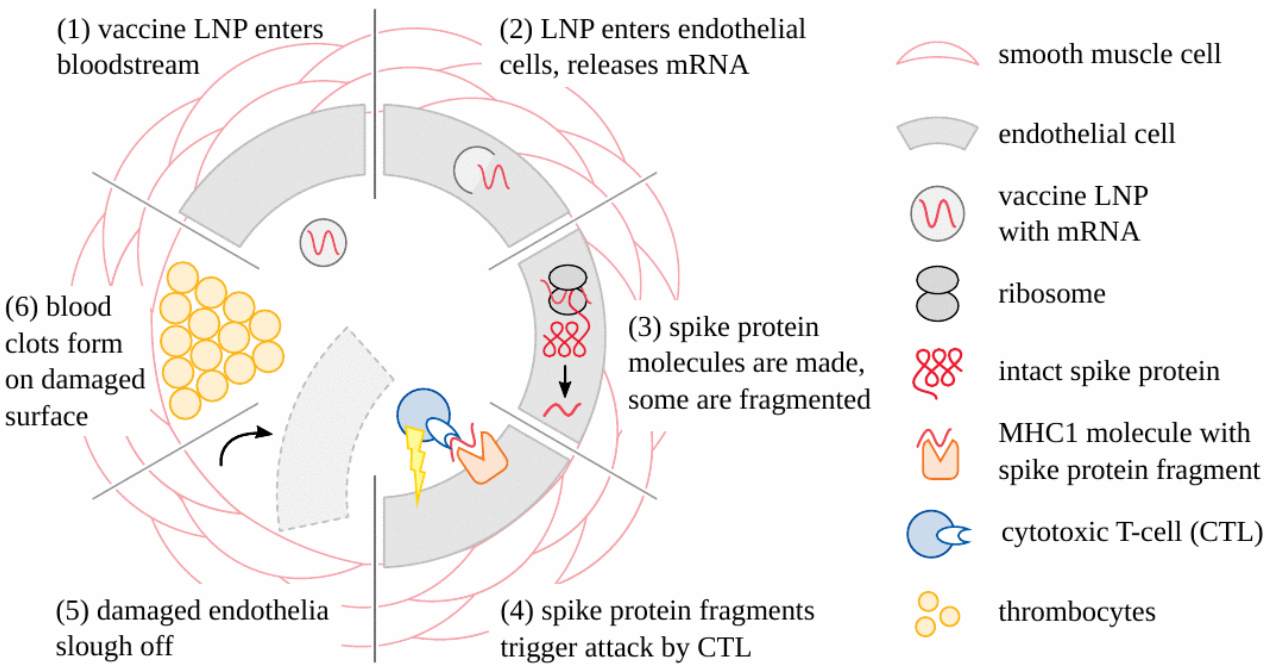

Figure 4: How COVID-19 mRNA vaccines damage blood vessels and cause clotting. After the vaccine lipid nanoparticles have entered the circulation, they are taken up by the endothelial cells, and the mRNA is released. The spike protein is then expressed; some molecules are fragmented and presented on the cell surface by a special carrier protein (MHC1). This causes the endothelial cells to be attacked by cytotoxic T-cells. Destroyed endothelial cells slough off, facilitating leakage of vaccine particles into the adjacent tissues. This also exposes the deeper layers of the vessel wall to the blood, which triggers thrombocyte aggregation and blood clotting.

Concomitantly, concerted immune attack will be mounted against the walls of vessels, whenever and wherever the endothelial cells become transfected (Figure 4). In the case of SARS-CoV-2, it is known that spike protein-specific cytotoxic T-cells are widely present in the blood of healthy individuals. This may be due to previous

infection with this virus, but alternatively also to immunological cross-reactivity with other, related Coronaviruses [18,19]. With the appearance of specific antibodies, attack on cells carrying the alien proteins will be multiplied and intensified manifold through the action of complement and phagocytic cells. Blood clots forming in the wake of endothelial injury will result in circulatory disturbances. Ischemic cell death will have irreversible consequences in the central nervous system and the heart. Damage to vessel walls will predictably cause leakage of the vaccines to and uptake by cells of the respective organs, planting the seeds for myriad autodestructive events.

Accumulating data are confirming these premonitions, and an emerging finding may prove to be distinctive and diagnostic for vaccine-mediated pathologies: vaccine-induced expression of spike protein within endothelial cells and resulting vasculitis will go hand in hand. The first illustration of this principle has been presented in a case report of a 76-year old man who died three weeks after receiving his third COVID-19 vaccination [20]. Histopathological analyses of the brain led to detection of multifocal vasculitis and necrotizing encephalitis. Small vessel vasculitis and lymphocytic myocarditis were found in the heart. Spike protein was detected within the foci of inflammation in both the brain and the heart, particularly in the endothelial cells of small blood vessels (Figure 5). Appropriate control experiments confirmed that the observed spike protein expression was indeed caused by the vaccine injections the patient had received, rather than by an undiagnosed infection with the virus itself.

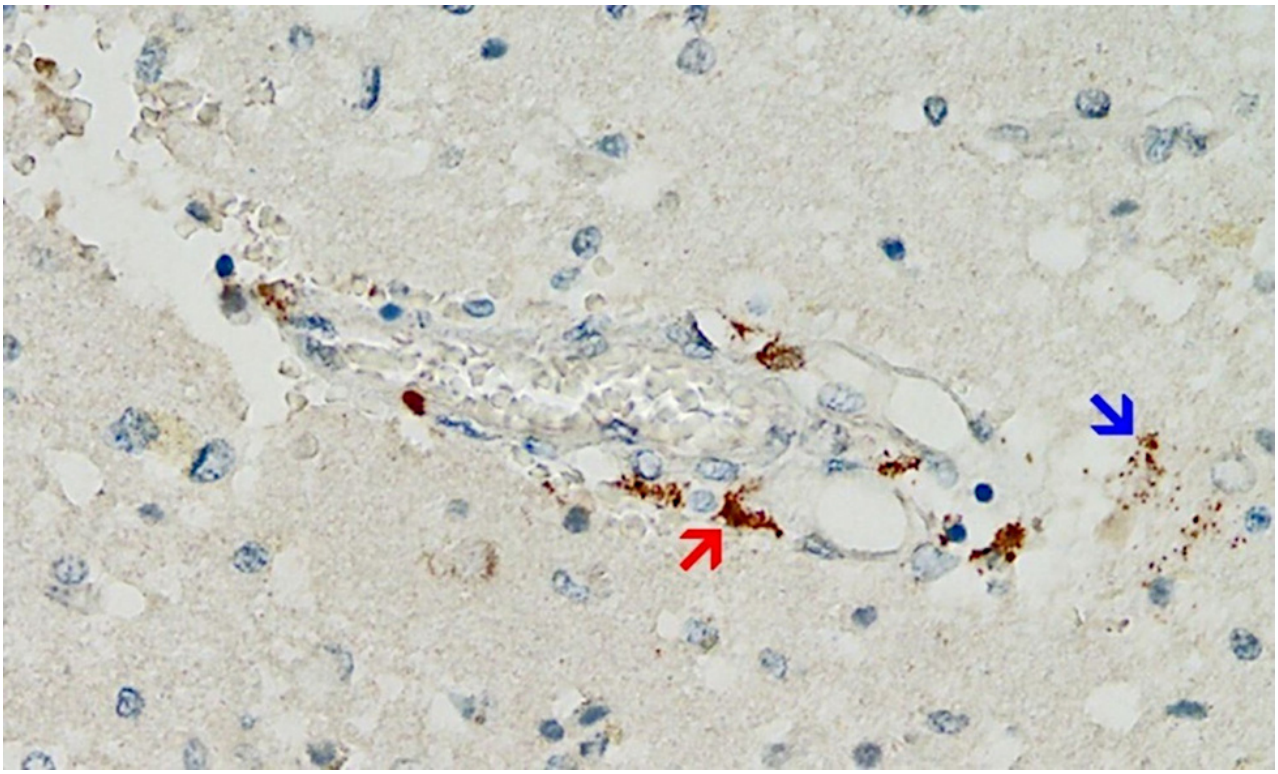

Figure 5: Expression of SARS-CoV-2 spike protein in the brain of a triple-vaccinated patient (immunohistochemistry). The brown pigment pinpoints spike protein within a small blood vessel (red arrow) as well as in glia cells of the surrounding brain tissue (blue arrow). Photograph taken from Mörz [20].

Multiorgan vasculitis, with notable involvement of small vessels, is now emerging as a common theme underlying adverse events following COVID-19 vaccination. Affliction of capillaries with thrombus formation in brain and heart are repeatedly being observed (Mörz [20]; Mörz and Burkhardt, unpublished). This pattern of disease in small and smallest vessels is novel and deemed by the authors to be characteristic for the action of gene-based vaccines.

Fulminant reactions may be expected to occur in patients who are vaccinated after recovery from genuine SARS-CoV-2 infections. Such patients will have high levels of circulating IgG antibodies against the spike protein [21], and complement attack on vaccine-transfected cells may then be immediate and massive. A case report of myocarditis-induced sudden death after first vaccination with direct evidence of complement activation on heart muscle cells has been published [22].

In our considered opinion, the outcome with future mRNA vaccines against other pathogens will be much the same as we have witnessed with the COVID-19 vaccines. It is true that the spike protein itself can promote blood clotting and inflammation without any help from the immune system [23]. Nevertheless, the already available evidence indicates that the grave, widespread and sustained injury to tissues and to blood vessels is mostly caused by the immune attack on spike protein-producing cells. This attack occurs simply because the spike protein is a non-self antigen; and since every other mRNA vaccine will encode its own non-self antigen, derived from whichever particular microbe it targets, we must expect that it will cause harm by the same mechanism and to a similar extent. These nightmarish scenarios will only get worse with every booster injection. The catastrophic events will be neither avoidable nor suppressible due to their very nature.

The disaster unfolding before our eyes could be, and was [24], predicted from first principles of immunology. The ability to distinguish between self and non-self is fundamental to life. It is already present at birth and ends only at death. It cannot be manipulated or controlled. Any attempt to do so with mRNA or other gene-based vaccines is doomed to failure.

Acknowledgment

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