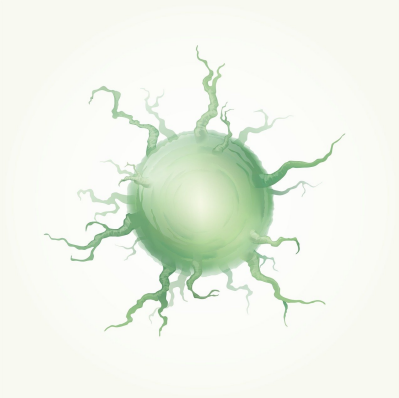

modRNA 'Vaccinations' Against SARS-CoV2 Alter the Sensitivity of the Immune System:

Description

Inflammatory Response Increases – This Could Explain the Number of Side Effects and Secondary Diseases

A new study recently made the rounds in the press: It has now been proven that the SARS-CoV2 mRNA 'vaccination' is good for the immune system. This is because it trains the immune system in the long term and enables it to respond much more quickly to all kinds of stimuli. Above all, it creates a lasting memory of this initial inflammatory response, which is very promising. I would like to take a closer look at this study from the working group of Prof. Jan Rybniker from Cologne [1] and combine this analysis in a second blog post with a workshop report of my evaluation of the side effect database of the Paul Ehrlich Institute on these very 'vaccines.' First, let's look at the study.

I am venturing into this discussion because competent colleagues from the MWGFD circles with the necessary expertise have prepared the ground for me. They have published a very informative article on the MWGFD website, to which I refer and which contains the relevant references; I recommend that anyone interested take a closer look at this article.

The study from Cologne is a good example of very clever and competent molecular biological and genetic work. The core of this work is the proof that both the spike protein of the SARS-CoV2 virus and the so-called genetic 'vaccines' cause identical activation of macrophages, i.e. the first line of our immune defence. Since the spike protein of the virus and that of the genetically modified therapeutic agents, the 'vaccine', are identical, this is not surprising. The spike protein activates a so-called 'inflammasome', i.e. a whole network of genes and associated proteins that are associated with the upregulation of inflammatory processes in macrophages. These then release pro-inflammatory cytokines, hormone-like messenger substances with which the immune system communicates within its various elements, but also with other organs, such as the brain. Interleukin (IL) 1beta in particular, but also tumour necrosis factor (TNF) alpha and IL36 were released by the activated macrophages. Interestingly, this activation is stronger after a booster, which is given after 6 months.

However, the authors were also able to show that this activation is associated with a proven increase in genetic

activity. When the blood of unvaccinated individuals is exposed to a spike protein, the activity of 50 genes changes. After one ‘vaccination,’ this number rises to 268 genes, and after a second vaccination, it rises to 2,518 genes.

Is this good or bad? That is precisely the question that divides opinion. The authors interpret and analyse this result as clearly positive. This is because they have carried out a whole series of additional provocation tests with other viruses, bacteria and signalling cascades and have seen that this sensitisation of the inflammasome is transferred to other stimuli and situations.

At the heart of this generalised inflammatory response is a central genetic regulator: the H3K27 protein. The abbreviation stands for ‘histone 3 lysine 27’ and is a marker for epigenetic changes.

Epigenetic changes

I need to back up a bit to explain this. In the picture-book understanding of genetics, gene letters are simply read and translated into proteins, which then provide the building blocks for all kinds of complex protein chains. That was how simple biological life was in the early 1960s and in simple biology textbooks. In reality, it is much more complicated. Genes, or more precisely DNA strands, are wrapped more or less tightly around protein cores like threads around spindles. These protein cores, called histones, themselves have different three-dimensional folding and shaping structures. In addition, their own protein threads protrude from them at varying distances like antennas. Methyl groups or acetyl groups can now attach themselves to these protein strands – and also to other structures. When methyl groups attach, this is called methylation. When acetyl groups attach, this is called acetylation. Basically, and very roughly speaking, methylation leads to the deactivation of the associated gene, while acetylation leads to the activation of a gene [2].

This entire mechanism serves to activate or deactivate certain genes in certain cells or for a certain period of time. This is why, for example, the muscle cells in my fingers do not produce liver enzymes while I am writing, and why my liver cells are able to metabolise the nutrients from the bread roll I ate two hours ago. It also means that some people gain weight much more quickly than others, or that Eskimos have many more capillaries in their fingers and hands than we do in Central Europe.

This machinery is called the ‘epigenome’ [3-5]. It is the collective name for all processes and structures that lead to a change in the expression of gene activity. There are a whole series of them. The acetylation and methylation of genes or histone proteins is only a small part of this. But what is special about it is that the epigenome allows experience-dependent modification of genetic activity. It has also been found that such epigenetic modifications can persist for a long time, sometimes for a lifetime, and that some are even passed on to the next generation.

The Cologne researchers have now shown that repeated contact with spike protein leads to acetylation of this H3K27 protein (H3K27ac) and thus to an epigenetic change in the expression of a whole series of genes involved in the activation of inflammatory processes. This epigenetic change lasts for a relatively long time, at least six months, and can even increase after further ‘boosters.’ Meaning: they have isolated an important building block for the mechanism that explains why immunological memory exists in the first place. The reason is, that the macrophages themselves, which came into contact with the spike protein, for example, die off after a few days at most. But this epigenetic change that has been induced here is, so to speak, the substrate-free carrier of the corresponding information. This is undoubtedly an extremely important finding and a major scientific advance.

But now comes the question of interpretation. Is something like this desirable and positive for the future? The researchers (and with them the Berliner Morgenpost and others) say yes. My colleagues and I say no.

Why do some say yes and others no? It's the perspective, stupid! It depends on the perspective and, above all, on the horizon we are looking at. If we look only at infectious disease parameters and a generalisation of the immune response to all possible pathogens, then the Cologne authors led by Jan Rybniker are probably quite right. It should be borne in mind that all these parameters come from in vitro models or basic research.

We are also familiar with the phenomenon of generalisation of an immune response from epidemiology and clinical practice. Aaby, for example, has shown that large vaccination campaigns in Africa have saved significantly more lives than could be expected from the reduced mortality rate due to this one pathogen [6, 7]. This is a clinically proven generalisation. Here, however, we are only dealing with evidence from basic infectious disease research.

If we take a broader view, my colleagues are probably right. This is because the H3K27ac protein is a so-called 'super enhancer'. Enhancers are proteins or molecules that contribute to the activation of a whole variety of genes. In their text, my colleagues cite a whole series of examples where research has linked the activity of H3K27ac to corresponding diseases: primarily oncological diseases and thus also to turbo cancer, which, as I have heard from many practitioners, has only been observed in this form since the 'vaccination.'

Inflammation and mitochondrial dysfunction

In addition, the inflammasome, i.e. the cascade of inflammation triggered by the spike protein in the macrophages, leads to all the diseases already known from Covid-19 pathology, from neurological to lung, liver and heart diseases. This particular inflammasome is also associated with impaired mitochondrial activity and may explain the fatigue frequently observed both after infection and after 'vaccination.' My colleagues report that there are 1,200 articles in the scientific literature on this topic. Mitochondria are known to be the energy centres of our organism. They produce the 'fuel' adenosine triphosphate (ATP), which supplies our body with the energy it needs. Every cell has many thousands of these mitochondria. If they are damaged, we feel unwell and suffer from exhaustion. This appears to be one of the consequences of the activation of the NLRP3 inflammasome.

The difference is that with Covid-19, only a few people became truly systemically ill, i.e., the virus was able to break through the mucosal barrier and attack the body itself. With the 'vaccination,' on the other hand, there are a great many people in whom the spike protein-forming mRNA enters the bloodstream with the help of nanoparticles and thus reaches all possible organs. Pathologists Arne Burkhardt and Walter Lang have clearly proven that this happens [8]. For, I repeat, the claim that spike formation is limited to muscle cells was a fairy tale for fools from the very beginning.

What needs to be done now is to carefully monitor a large cohort of 'vaccinated' and 'unvaccinated' individuals, which do exist. They would have to be very well-balanced in terms of the most important parameters (age, gender, social status, education, pre-existing conditions) and then observed over a longer period of time: are the 'vaccinated' really less susceptible to other viral infections such as influenza, etc., or SARS-CoV2 infections? Do they experience less fatigue, cancer and other chronic diseases? Pure, unsystematic observation tells a different story here: never before, it seems to me, have so many train crew been ill and so many trains been cancelled due to illness as in the years after 2021. Never before have there been such high levels of sick leave, it seems to me. And I heard about turbo cancer for the first time in 2021, even though I have had repeated contact with oncologists and cancer therapies in other contexts. We called for such systematic observation back in 2021 [9]. I am not aware of any such observation having been carried out since then. I wonder why?

Sources and further reading

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