

The book: <https://www.goldegg-verlag.com/titel/corona-fehlalarm/>

The virus binds via protein projections (so-called spikes) that recognize certain molecules (receptors) on our cells. The process can be compared visually with the grasping of door handles (receptors) with violent hands (spikes). In this way, the viruses gain access to the cells, where they are multiplied. The offspring are released and can infect other cells. The immunity to coronaviruses is based on two pillars: 1) Antibodies 2) Specialized cells of the immune system, so-called helper lymphocytes and Killer lymphocytes. When a virus enters the body penetrates and causes disease, the immune system responds by mobilizing these defensive arms. Both are trained to target the invading virus specifically 4 recognize, and lymphocytes are endowed with the gift of long-term memory.

Many different antibodies are produced, each of which specifically recognizes a tiny part of the virus. Only the antibodies that bind the virus' "hands" offer protection, as they can prevent the virus from hitting the door handles. Classic virus vaccines are supposed to induce our immune system to produce such antibodies. It is often assumed that immunity against the virus is thereby achieved.

Defense strategy 2: Immune cells recognize “virus waste” on the cell surface and destroy the infected body cells

At this point, however, three points must be emphasized. 1. If you have been tested for SARS-CoV-2 antibodies and nothing has been found, it does not mean that you were not infected. The strength of the antibody formation often correlates with the severity of the disease. Infections that progress easily can be accompanied by hardly detectable antibody production. 2. If antibodies are found, it doesn't mean you are immune. Current immunological tests cannot protect anti-bodies (against the “hands” of the virus)

se defense strategy

1 Antibodies prevent “docking” Defense strategy

2 Immune cells recognize “virus waste” on the cell surface and destroy the infected body cells
VirusLymphocytesBody cellAntibodiesThe immunity against corona viruses is based on two defense strategies

6lectively prove. Other antibodies show up at the same time. The tests cannot therefore provide any reliable information about the “immune status” of an individual. The result of an encounter between “protective” antibodies and the virus is not “black or white”, not “now or never”.

Quantities are crucial. A wall of protective antibodies could ward off an attack in a favorable situation - for example, if someone coughs from a distance. The attack intensifies as the person gets closer. The scales begin to tip. Some viruses can now cross the barrier and get into the cells. When the cough comes up close, the fight becomes one-sided and ends in a quick victory for the virus.

A "successful" vaccination and production of protective antibodies does not guarantee immunity. Added to this is the fact that antibody production spontaneously declines after a relatively short period of time. Two conclusions are inevitable. 1) Trying to raise an antibody-based "immune status" makes no sense. 2) The chances of success for a vaccination are hardly available from the

start. What happens after the virus has entered the cell? The events were detected in extensive animal experiments for the original SARS virus.

7 clarified. The second arm of the immune system then comes into play. Lymphocytes arrive at the crime scene. Helper cells are activated and in turn stimulate their partner, the killer lymphocytes (1). These attack the virus-infected cells and kill them. The factory is destroyed, the fire is extinguished. Cough and fever go away. How can killer lymphocytes know which cells to attack? In simple words: Think of an infected cell as a factory that produces and assembles the parts of the virus. This creates waste products that the cell disposes of in an ingenious way: it transports them out and puts them in front of the door. The patrolling killer cells see the garbage and attack. This second arm of our immune system has hardly been talked about so far, but it is likely to be of crucial importance for the defense against coronavirus - much more so than antibodies, which form a rather shaky first line of defense. A key factor here is the fact that waste products from different coronaviruses are similar to one another. So there is a high probability that killer lymphocytes that recognize the waste of a virus will also attack cells in which other coronaviruses are produced.

Would this mean mutual immunity? In principle yes. Coronavirus mutations take place in very small steps. Protective antibodies and lymphocytes against type A are therefore also quite effective against offspring Aa. If B comes by and is not recognized so well, a new cold can result. Thereafter, the immune status expands to A, Aa, B and Bb, so the scope of immunity increases with each new infection. And lymphocytes are endowed with a long-term memory. Who does not remember the first year of their child in kindergarten? Oh no, not again, here comes the umpteenth cold to a runny nose, cough and fever. The child is sick all through the long winter! Fortunately, things will get better in the second year and maybe only one or two colds will occur in the third year. In our first years of life, a very solid immunological basis is built up, which enables a peaceful coexistence with the countless coronaviruses in the world.

What does "immunity to corona" really mean? Does "immune" mean that we are not infected at all? No, it means that we do not get seriously ill. And not getting sick is not based solely on preventing infection with antibodies, but rather The main focus is on "putting out the fire". When a new variant of the virus appears, many people can become infected, but because the fires are put out quickly, they do not get seriously ill. In relatively few cases, wildfires occur - a serious illness is the result. But as long as no other disease is involved, the immune system will usually prevail in the end. Infections with coronaviruses are therefore almost only for people with pre-existing illnesses the drop that brings the barrel to overflow. This is the reason why most corona infections take a mild course and why after an epidemic no second and even less worse wave will follow. Why do the annual corona epidemics end in summer? A speculation. Over 50% of the northern European population suffer from vitamin D deficiency in the dark winter months. Perhaps the sunshine replenishing vitamin D stores and moving outdoors are simple, important reasons.

10 What happens to the virus after an epidemic? Is it disappearing from the country? It joins its relatives and continues to circulate with them in the population. Infections are occasional, but most are barely noticed. Every now and then everyone gets their summer flu. That's life and it's always been like that.

Can a similar pattern be expected with SARS-CoV-2? The authors believe we have seen just that. 85–90% of the SARS-CoV-2 positive people did not become seriously ill. Most likely, here lymphocytes put out the fires in good time so that virus production did not go awry. To put it simply: the new virus variant could in fact infect almost anyone. However, immunity was already widespread due to the presence of lymphocytes that cross-recognized the virus.

Is there evidence that lymphocytes from non-exposed individuals cross-recognize SARS-CoV-2? Yes. In a German study, lymphocytes from 185 blood samples taken between 2007 and 2019 were examined for cross detection of SARS-CoV-2. Positive results were found in no less than 70-80%, and this was true for both helper and killer lymphocytes (2). A US study with lymphocytes from 20 unexposed donors similarly reported the presence of lymphocytes that were cross-reactive with the new virus (3). In both studies and in another from Sweden (4) it was found that all SARS-CoV-2 infections - even those with the mildest courses - caused a remarkably broad and strong stimulation of the responsible T lymphocytes. We see in this finding the clear indication that it is a booster effect - similar to a booster vaccination. This means that the cross-reactive T cells were already present and were immediately and strongly activated by the infection.

Could the idea that lymphocytes mediate cross-immunity against SARS-CoV-2 be tested? The concept of lymphocyte-mediated herd immunity that we have presented results from the integration of the latest scientific data (1–4) into the established context of immunity of the host against viral infections. The idea can actually be put to the test. In one study, *Cynomolgus* monkeys were infected with SARS-CoV-2 (5). Although all animals shed the virus, not a single one became seriously ill. Minor changes in the lungs were found in two animals, underscoring the fact that vigorous production of the virus had taken place, essentially replicating what was seen in healthy humans. It should not be difficult to check whether lymphocytes are the carriers of immunity in the animals.

To vaccinate or not to vaccinate, that is the question The development of vaccines against dreaded diseases such as smallpox, diphtheria, tetanus and poliomyelitis was an important turning point in the history of medicine. This was followed by vaccinations against a number of other diseases that are now part of the standard repertoire of preventive medicine. Vaccinations save human lives, but they do not work for all diseases and are not always useful. What about COVID-19? At the beginning of June 2020, the Federal Ministry of Finance published the key points of an economic stimulus program for the consequences of the corona Under point 53 you can read (6): “The corona pandemic will end when a vaccine is available for the population.” This sentence is astonishing in several ways

Up until now, it was actually up to the WHO to declare or end a pandemic, and not to the federal government. Actually, the definition of a pandemic was - different. One wonders what that is supposed to mean. Should we in Germany keep our distance and wear masks just because the infection numbers are perhaps rising somewhere in South America? There are also many bad infections for which, despite decades of research, no functioning vaccine is available to this day. What if that is also the case for COVID-19? Strange. But let's take a closer look at whether a global vaccination program is necessary and useful to end the corona crisis. So important is this question that a debate needs to be conducted urgently in order to reach global consensus on three

fundamental points. When is the development of a vaccine necessary? We think: When an infection in healthy people usually leads to serious illnesses and their consequences. This is not the case with SARS-CoV-2. 2. When would mass vaccination not make sense? We think that mass vaccination does not make sense if a large part of the population is already sufficiently immune from a serious illness. This is the case with SARS-CoV-2.

3. When will a vaccination be unsuccessful? We assume that a vaccination will fail if a virus is constantly changing

The authors therefore take the view that a global vaccination program for SARS-CoV-2 does not make sense and is doomed to failure from the outset. The risks are incalculable and potential benefits are not apparent. Many experts warn against the rushed development of COVID-19 vaccines (7, 8). Yet there is a hectic race to develop vaccines. There are currently no fewer than 150 COVID-19 vaccine candidates (9), some of which are already in advanced clinical trials. The main goal of vaccination is to stimulate the formation of protective antibodies against the binding protein of the virus (10, 11). Four main strategies are being pursued.

1. Inactivated or attenuated whole virus vaccines. Inactivated vaccines require the production of large quantities of the virus, which must be grown in hen's eggs or in immortalized cell lines. There is always a risk that a batch of virus could contain dangerous contaminants and cause serious side effects. In addition, there is a possibility that vaccination may paradoxically worsen the course of a subsequent infection (12), as has been seen in the past with other vaccinations (13, 14). Attenuated vaccines contain replicating viruses that have lost their ability to cause disease. The classic example was the oral polio vaccine, which was used for decades before tragic polio outbreaks occurred in Africa, caused not by wild viruses but by the oral vaccine (15)

2. Protein vaccines. These contain the virus spike protein or fragments thereof. The addition of immune stimulators (adjuvants), which can cause serious side effects, is always necessary (10).

3. Viral vectors. The relevant corona gene is incorporated into the gene of a carrier virus (vector). The carrier virus then infects our cells. Replication-defective vectors cannot amplify their genome and only deliver a copy of the coronavirus gene into the cell. In order to increase the effectiveness, attempts have been made to produce replication-competent vectors. This is how the Ebola vaccine rVSV-ZEBOV was created, which was also tested on humans. Serious side effects were found in at least 20% of the vaccinees (10)

4. Gene-based vaccines. In these cases, the viral gene is either inserted in the form of circular DNA (plasmid) or the gene is brought directly into cells as mRNA. A risk of DNA-based vaccines is insertion into the cell genome (16). This so-called insertion mutagenesis is a rare occurrence. But very rare events can quickly become important if the number of opportunities reaches appropriate dimensions - as with mass vaccination.

17If it is inserted into cells of the reproductive system, the changed genetic information is transferred from mother to child. Other dangers of DNA vaccines are the production of anti-DNA antibodies and autoimmune reactions (17).

Safety concerns raised so far in connection with mRNA vaccines include systemic inflammation and possible toxic effects. The production of every virus protein will be directly or indirectly associated with the appearance of degradation products on the outside of cells, which are thereby made recognizable for an attack by killer lymphocytes. It is now clear that most healthy people already have killer lymphocytes that recognize such SARS-CoV-2 products (peptides) (4). It must therefore be assumed that autoimmune attacks are taking place on the marked cells. The

attack of killer lymphocytes on sensitive or even irreplaceable cells could have tragic consequences.

Hundreds of volunteers who were never informed of these potential risks have already received injections of DNA and mRNA vaccines. No gene-based vaccine has ever been approved for human use, and the existing corona vaccines have not undergone sufficient pre-clinical testing, as is normally required by international regulations.

18The EU decided in July that clinical studies can start without the previously mandatory environmental impact assessment for the genetically modified organisms (GMOs) used having to be completed. This also applies to the production of vaccines that contain GMOs. (19) Germany, whose population largely rejects the genetic manipulation of food, is suddenly - with broad approval from politics and society - at the forefront of the development of gene-based ones Vaccines. Laws and safety regulations have been circumvented in ways that would never have been possible under normal circumstances. The basis for this is the amended Infection Protection Act. Is this perhaps the reason why the government has declared that an epidemic situation of national scope still exists - although there has been no significant number of new cases for weeks? Because the Ministry of Health is only authorized to make exceptions to the provisions of the Drugs Act and the Medical Device Regulations in the event of an epidemic of national scope. We ask our readers to ask themselves whether the German government will even go that far may allow and even encourage genetic experiments on ignorant people. Such human experiments appear to us simply unethical and not compatible with any basic law of the civilized world.

Referenzen(1)[www.cell.com/immunity/fulltext/S1074-7613\(16\)30160-1?_returnURL=https%3A%2F%2Flinkinghub.elsevier.com%2Fretrieve%2Fpii%2FS1074761316301601%3Fshowall%3Dtrue](http://www.cell.com/immunity/fulltext/S1074-7613(16)30160-1?_returnURL=https%3A%2F%2Flinkinghub.elsevier.com%2Fretrieve%2Fpii%2FS1074761316301601%3Fshowall%3Dtrue)(2) www.researchsquare.com/article/rs-35331/v1(3) [www.cell.com/cell/fulltext/S0092-8674\(20\)30610-3\(4](http://www.cell.com/cell/fulltext/S0092-8674(20)30610-3(4)) www.biorxiv.org/content/10.1101/2020.06.29.174888v1(5)science.sciencemag.org/content/368/6494/1012.long(6) www.bundesfinanzministerium.de/Content/DE/Standardartikel/Themen/Schlaglichter/Konjunkturpaket/2020-06-03-eckpunktepapier.pdf?__blob=publicationFile&v=10(7) [www.nature.com/articles/d41586-020-00751-9\(8](http://www.nature.com/articles/d41586-020-00751-9(8)) www.sciencemag.org/news/2020/07/scientists-scoff-indian-agencys-plan-have-covid-19-vaccine-ready-use-next-month(9) www.rki.de/SharedDocs/FAQ/COVID-Impfen/COVID-19-Impfen.html(10) www.tandfonline.com/doi/full/10.1080/14760584.2018.1419067(11)www.nature.com/articles/d41586-020-01221-y(12) science.sciencemag.org/content/368/6494/945(13)jamanetwork.com/journals/jama/article-abstract/336928(14) academic.oup.com/aje/article/89/4/422/198849(15)jvi.asm.org/content/87/9/4907.long(16) www.nature.com/articles/3302213(17) link.springer.com/protocol/10.1007%2F978-1-62703-110-3_27(18) www.nature.com/articles/nrd.2017.243(19) www.europarl.europa.eu/news/de/press-room/20200706IPR82731/parlament-will-entwicklung-von-covid-19-impfstoffen-beschleunigen