Salvagene GROUP

Business Unit: SARS-CoV-2 Task Force
Keynote: #12

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# Salvagene Task Force clarifies the current situation regarding immunity to SARS-CoV-2

NEW!

Dear Premium Customers,

**In a video press conference, the Salvagene SARS-CoV-2 Task Force (SSCTF) answers questions from reporters on what is known about the response of the body's own defense system to SARS-CoV-2 and about how long any immunity may last – as well as what the unknown factors are.**

QUESTION: Over the past few days and weeks, a lot of younger people have been heard to say: "If I had the virus behind me, I would be immune now." And indeed, there is something reassuring about the notion that, if everyone who had been infected and survived the disease were immune afterwards, human society could begin to get back to normal. But do we actually know anything about immunity to Sars-CoV-2?

SSCTF: The short answer is No. It’s true that we’ve known about the virus for more than three months, and it has been the subject of an enormous number of studies, but so far there is very little data on how the immune system reacts to the virus.

QUESTION: Why not?

SSCTF: That would require us to study blood or tissue samples from infected people on a large scale – not only from those who are seriously ill or have even died, but also from those who display no symptoms. At the moment, doctors and laboratory staff have other things to do than collecting and preparing samples on such a scale. And in order to come to really reliable conclusions, you would also need evidence from long-term observations of survivors. But I am sure that samples are already being frozen and that the material is there – it just needs to be evaluated.

QUESTION: Nevertheless, it would be helpful to at least have some idea as we go into the next few weeks. Is it possible to make deductions from other viruses about potential immunity to Covid-19?

SSCTF: We have a good basis upon which to speculate, but of course, nothing can be said for sure. However, this new strain of coronavirus is very similar to the SARS virus of 2002/2003. So we are cautiously orienting ourselves around that.

QUESTION: Around 8,000 people fell ill with SARS, of which some 700 died. What do we know about the survivors?

SSCTF: There have been several studies in which the researchers looked for antibodies in the blood of SARS survivors. If antibodies are present, it can be assumed that the person is immune, meaning that the body’s own defense mechanisms can fend off

the pathogen. In all survivors, however, the concentration of antibodies has decreased over the years. In one study conducted six years after the illness, 21 of 23 people who were examined were found to have no antibodies remaining in their blood.

QUESTION: So this would mean that immune protection diminishes over time. With other corona viruses, which are more likely to cause only a common cold, the immune protection only lasts for a very short period of time – that is why people can get infected over and over again. Can we rule out the possibility that antibodies built up against Sars-CoV-2 offer only very short immune protection?

SSCTF: Nothing can be ruled out until it has been scientifically tested. But because SARS-CoV-2 is much more closely related to SARS-CoV and MERS-CoV than to the corona viruses triggering the common cold, the probability is rather low.

QUESTION: There is a theory that the more severe courses of disease are due to an overreaction of the immune system. Can you tell us more?

SSCTF: The immune response to some respiratory infections – for example to SARS, MERS and even influenza – sometimes goes off the rails. Then the immune system releases enormous amounts of cytokines and chemokines. These are substances that cause massive collateral damage and, in extreme cases, can even kill cells. In the next few days, we will be making further recommendations in this regard for our clients here at Salvagene.

QUESTION: But surely the immune response should actually only eliminate the cells that are infected by viruses?

SSCTF: Yes, but that is precisely what is not happening. The lung tissue also comes under severe attack, including cells that are not

infected. This exaggerated response was also the reason that so many people in the 20 - 40 age bracket died in the Spanish flu outbreak.

QUESTION: Is this due to the pathogen or to the patient?

SSCTF: Once again I have to say that we don’t know for sure. For years, scientists have been trying to isolate and replicate the H1N1 Spanish flu virus which killed up to 50 million people between 1918 and 1930. They have even used tissue from bodies exhumed in Alaska. These studies date back to the 1990s. In 2005, the virus was successfully replicated. The next step was to infect mice with the virus. This resulted in immense damage to the lung tissue. What causes the immune system to overreact so strongly is not yet understood.

QUESTION: Salvagene suggested another possible explanation for the more severe courses of disease several weeks ago, according to which the risk of overreaction increases if an individual has already had contact with a coronavirus.

SSCTF: Yes, behind this is the phenomenon of Antibody Dependent Enhancement or ADE for short. A classic example of this is dengue fever. There are different subtypes of dengue viruses. After the first infection, humans produce antibodies that protect against the virus. If these individuals then suffer an infection with a different subtype, the infection can take a more severe course than in people without antibodies.

QUESTION: Is there evidence that ADE also occurs in the latest strain of coronavirus?

SSCTF: If we look at the evidence presented by SARS, we find arguments for and arguments against. Some studies have reported an immunopathology after immunization in monkeys,

mice and ferrets, but not in hamsters. Observation of some human cell lines in the Petri dish showed that they were easier to infect in the presence of antibodies.

QUESTION: And what are the counterarguments?

SSCTF: Likewise, findings from the SARS epidemic. Then, as now, everything was done to save the lives of patients. They also took risks. In some clinics, for example, the patients were given blood serum from patients who had already recovered. The idea behind this was that the serum contained antibodies that would help the patient fight off the virus. In the worst case, however, these antibodies could have triggered ADE in the patient.

QUESTION: Fortunately, that turned out not to be the case. Was the serum treatment effective in controlling the course of the SARS disease?

SSCTF: It’s hard to say, because the hectic pace of the disease restricted the scope for conducting a controlled study. A so-called meta-analysis – essentially a summarized analysis of the individual reports – came to the conclusion that the treatment seems to have been safe and may have reduced mortality. A controlled trial would be desirable.

QUESTION: Let us return to relative lethality by age group, which is not entirely unusual for an infectious disease that affects the respiratory tract. Why is that?

SSCTF: This is because the immune system ages along with the individual. It’s a bit like with the brain: the immune system becomes forgetful over time, so it can no longer respond so effectively to pathogens that are actually known. And it cannot remember new things so well, which means that it cannot respond so quickly to new pathogens such as SARS-CoV-2. In

fact, all levels of the immune system are equally affected by aging – the innate defenses as well as the acquired defenses. Furthermore, older people tend to have stronger inflammatory reactions anyway, which makes them more susceptible overall.

QUESTION: Do you think that we will actually get a vaccine against SARS-CoV-2 in just a few months, as some companies are promising?

SSCTF: To be honest, we wouldn't want to dispense with the customary vaccine testing before something is tried on humans. We do not know the immune response to the virus well enough. If we were to accelerate the approval procedure for a vaccine now, that could backfire on us. As with ADE, there is also VDE (Vaccine Dependent Enhancement), which means that the vaccine would make the disease worse. This was the case in the 1960s, for example, when a vaccine against RS viruses was developed. This is a virus that is a common trigger for respiratory infections in small children. After children had been vaccinated, however, it was found that they became seriously ill more often than those who had not been vaccinated. The vaccine was immediately withdrawn from the market. We are therefore in favor of a very cautious approach.

QUESTION: Some experts – even proven epidemiologists or virologists – are suggesting that the strict approach adopted by governments has gone over the top. Their argument is that we do not know the number of unreported cases and can therefore assume that the lethality rate is much lower than the three percent reported from China. What is your opinion?

SSCTF: Anyone is of course free to offer their own assessment if they can provide evidence of exactly how they came to this conclusion. However, a good parameter of how bad a disease is and how seriously it has to be taken is the number of infected people who die. Worldwide this is currently about three percent, though the figure for Germany so far is around 0.5 percent. In

Germany, an above-average number of people have been tested, which explains the comparatively low mortality rate. But even a lethality rate of 0.1 percent would mean that tens of thousands of people would become gravely ill and die. It is the power of large numbers that makes this virus so dangerous. At best, the restrictions on leaving home flatten the curve and ensure that our public health system does not collapse.

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